WDDTY
WHAT DOCTORS DON'T TELL YOU

Ageing well

What you need to know about getting older – Part One

Common conditions

Women's health

Dangerous drugs

Preventing cataracts

New evidence suggests that popular prescription drugs could be causing cataracts—the leading cause of blindness worldwide

Cataracts—cloudy, opaque patches in the lens of the eye—are often considered an inevitable part of growing old, like becoming wrinkly and going grey. However, recent evidence suggests that common prescription drugs, rather than old age, may be responsible for a significant proportion of cases.

Two studies published just a few months apart have revealed that taking certain antidepressants or hormone replacement therapy (HRT)—drugs used by millions of people worldwide—can dramatically increase the risk of cataracts.

The antidepressant study—the first major investigation into such a possible link—analyzed data from nearly 19,000 elderly cataract patients and around 190,000 controls. It found that those currently taking selective serotonin reuptake inhibitors (SSRIs), the most widely prescribed antidepressants in many countries, were 15 per cent more likely to have cataracts. In the US alone, this means that 22,000 cataract cases may be due to SSRI use.

The association was especially strong for Paxil (paroxetine), Effexor (venlafaxine) and Luvox (fluvoxamine). Paxil increased cataract risk by 23 per cent, Effexor by 33 per cent and Luvox by a whopping 39 per cent. Happily, the higher risk was linked only to current drug use (Ophthalmology, 2010; 117: 1251–5).

Although more research is needed to confirm the link, previous animal studies have suggested a plausible explanation for the increased risk of cataracts among SSRI users.

"The eye's lens has serotonin receptors, and animal studies have shown that serotonin can make the lens opaque and lead to cataract formation," said study author Dr Mahyar Etminan. "If our findings are confirmed in future studies, doctors and patients should consider cataract risk when prescribing some SSRIs for seniors," he added.

The other study linking cata-racts to prescription drug use was an eight-year investigation of more than 30,000 postmenopausal Swedish women, which found that those using or who had used HRT had significantly higher rates of cataract removal than those who had never used HRT. Indeed, cataract removal risk was increased by 14 per cent in women who had ever used HRT and by 18 per cent in current HRT users—and the longer a woman used HRT, the higher was her risk.

Alcohol consumption also appeared to increase harmful HRT effects. Current HRT users who also reported having more than one alcoholic drink per day had a 42-per-cent greater risk of having to undergo cataract removal compared with women who used neither HRT nor alcohol (Ophthalmology, 2010; 117: 424–30).

As a plausible mechanism for how HRT could cause cataracts, it may be that HRT increases levels of C-reactive protein, high levels of which have been associated with cataracts in other studies. As lead author Dr Birgitta Lindblad said, "If future studies confirm the associations we found, increased risk for cataract removal should be added to the list of potential negative HRT outcomes."

These new findings suggest that, in many cases, cataracts could be prevented by avoiding certain drugs. But it's not only SSRIs and HRT that have been linked to cataracts. Other studies have found significantly increased risks with statins (BMJ, 2010; 340: c2197), oral and inhaled corticosteroids (Ophthalmology, 2009; 116: 652–7), antidiabetes drugs (Ophthalmology, 2001; 108: 1670–4) and some sun-sensitizing medications, such as the painkilling non-steroidal anti-inflammatory drug (NSAID) naproxen (Arch Ophthalmol, 2010; 128: 959–63).

The importance of diet

On the other hand, researchers are also finding that a healthy diet can offer powerful protection against cataracts.

Scientists from the University of Wisconsin's Department of Ophthalmology and Visual Sciences found that women who ate foods rich in vitamins and minerals were less likely to develop nuclear cataracts—those forming in the centre of the lens—the most common type of cataract.

The study involved over 1800 women, aged between 55 and 86 years, who took part in the Carotenoids in Age-Related Eye Disease Study (CAREDS). The daily diets of these women were assessed according to their responses to a food-frequency questionnaire (the 1995 Healthy Eating Index, HEI), while the presence of nuclear cataracts was determined four to seven years later. These women's diets were also assessed to see how closely they reflected the 1990 dietary guidelines for Americans.

A high HEI score was the strongest modifiable predictor of a low prevalence of nuclear cataracts. What contributed to the higher healthy diet scores were intakes at or above the recommended levels for vegetables, fruits, grains, milk and meat (or beans, fish or eggs), and below-recommended levels of fat, saturated fat, cholesterol and salt. Women with the highest HEI scores were found to have a 37-per-cent lower risk of developing cataracts than those with the lowest scores.

"These data add to the body of evidence suggesting that eating foods rich in a variety of vitamins and minerals may contribute to postponing the occurrence of the most common

type of cataract in the United States," the researchers concluded (Arch Ophthalmol, 2010; 128: 738–49).

There's also evidence to suggest that specific nutrients might be especially helpful for warding off cataracts. In an earlier study by the same team of researchers from the Universityof Wisconsin, women with high dietary intakes of lutein and zeaxanthin—found in dark green leafy vegetables, as well as in kiwi fruit, grapes, corn and egg yolk—had a 23-per-cent reduced prevalence of nuclear cataracts. Those with the highest intakes of these nutrients were 32-per-cent less likely to have nuclear cataracts compared with those who had the lowest intakes.

The researchers also commented that lutein and zeaxanthin, both of which accumulate in the lens of the eye, are known to protect against photodamage (caused by ultraviolet radiation), which predisposes to cataracts (Arch Ophthalmol, 2008; 126: 354–64).

A number of other nutrients, mostly antioxidants, also appear to be important for cataract prevention. A 10-year study of more than 2400 older adults in Sydney, Australia, found that higher intakes of vitamin C (from both food and supplements) or the combined intake of multiple antioxidants (vitamins C and E, beta-carotene and zinc) significantly reduced cataract risk (Am J Clin Nutr, 2008; 87: 1899–905).

Oxidative stress is thought to play a significant role in cataract formation, so it makes sense that antioxidants—which mop up free radicals that damage proteins and fibre cells in the lens—can be protective. Indeed, a study in North India showed that having higher levels of antioxidants in the blood—including vitamin C, alpha-/beta-carotene, zeaxanthin, lutein and lycopene—led to a significantly lower likelihood of having cataracts (Invest Ophthalmol Vis Sci, 2008; 49: 3328–35).

Reducing the risk

Besides avoiding certain drugs and eating a healthy, antioxidant-rich diet, there are a number of other ways to reduce your risk of cataracts.

- **Stop smoking**. Smokers are more likely to have cataracts, so kicking the habit may help to save your sight (Arch Ophthalmol, 1997; 115: 1296–303). It's thought that the cadmium in cigarettes accumulates in the lens and causes damage. Also, cigarette smoke is rich in free radicals, among other things, so it could also be that smoking depletes levels of protective antioxidants (Br J Ophthalmol, 1998; 82: 186–8).
- **Lose weight**. Researchers at Harvard University found that people with a body mass index (BMI) of 30 kg/m^2 or more (obesity) increased their risk of cataract by at least a third compared with a BMI of 23 kg/ m^2 (overweight) or less (normal) (Int J Obes Relat Metab Disord, 2002; 26: 1588–95).
- **Drink in moderation**. The 10-year Blue Mountains Eye Study in Australia (described above) found that people who drank more than two standard alcoholic drinks per day or were total abstainers were significantly more likely to undergo cataract surgery compared with moderate drinkers. Indeed, in this study, a moderate consumption of alcohol—one or two drinks a day—was associated with a 50-per-cent lower incidence of cataract surgery (Am J Ophthalmol, 2010; 150: 434–40).
- **Exercise**. A recent study has reported that vigorous exercise can protect against cataracts. The researchers found that men who ran 64 km/week or more had a 35-per-cent lower risk of developing cataracts compared with men who ran less than 16 km/week.

  Using these men's performances in 10-km races, which provide a good measure of overall and cardiorespiratory fitness, it was also found that the fittest men had only half the cataract risk of those who were the least fit (Invest Ophthalmol Vis Sci, 2009; 50: 95–100).
- **Minimize heavy-metal exposure**. Long-term, low-level exposure to toxic metals—and lead, in particular—results in their accumulation in the lens. Such a buildup increases the oxidative burden of the lens, thus leading to cataracts (JAMA, 2004; 292: 2750–4).
- **Sport those sunglasses**. When Japanese researchers reviewed studies carried out in Japan as well as in Iceland, Australia and Singapore, they found that those exposed to the highest levels of sunlight (ultraviolet light) also had the highest incidences of cataracts (Invest Ophthalmol Vis Sci, 2003; 44: 4210–4).

Joanna Evans

For more information on cataracts, consult the WDDTY Good Sight Guide.

Promising natural treatments

- Bilberry is rich in anthocyanosides, potent antioxidants with a particular affinity for the eyes. In a report of 50 patients with senile cataracts, a combination of bilberry (180 mg twice daily)—standardized to 25-per-cent anthocyanosides—and vitamin E (100 mg twice daily) for four months stopped the progression of cataracts in 96 per cent of the treated patients compared with 76 per cent of the control group (Altern Med Rev, 2001; 6: 141–66).
- Vitamin E was also tested on its own in a randomized controlled trial (RCT) of 50 patients with senile cataracts (100 mg twice daily for 30 days). Compared with a placebo, there was a significantly smaller increase in the size of cortical cataracts (those in the outer rim of the lens) in the vitamin E group (Ann Nutr Metab, 1999; 43: 286–9).
- N-acetylcarnosine-containing eye drops called Can-C have shown potential in the treatment of cataracts. An RCT of 49 patients reported that, after six months of twice-daily Can-C use, 90 per cent of the NAC-treated eyes improved in visual acuity and 89 per cent improved in glare sensitivity. The overall visual outcome in the control group showed significant worsening after two years. It should be noted, however, that this study was conducted by the company—Innovative Vision Products (IVP)—that helped to develop the eye drops (Drugs R D, 2002; 3: 87–103).

AMD: it's inflammation, not age

The latest findings suggest that macular degeneration may not be 'age-related', but due to mineral imbalances and inflammatory disease

Failing eyesight has become so closely associated with old age that the condition has become known as AMD, or 'age-related macular degeneration'. Around 15 million Americans and four million Britons suffer from the problem—where the sharpness of our central vision deteriorates—and health experts now fear that AMD will take on epidemic proportions once the baby-boomers reach their 60s and 70s.

AMD comes in two forms—'wet' and 'dry'. The dry type is by far the more common. This type occurs when photoreceptors in the central part of the eye, or 'macula', deteriorate and die. In contrast, the wet variety is caused by abnormal blood-vessel growth, which can lead to blood and protein leakages, irreversible and rapid vision loss, and even blindness. Also, the dry form can worsen and become wet.

Because medicine has associated AMD with the ageing process, it hasn't looked much beyond that for other causes of the condition. This view has also influenced its treatment. Medicine contends that it has nothing to offer the sufferer who has dry AMD, and has only recently begun to offer regular injections of anti-angiogenic drugs designed to reverse blood-vessel growth in those with the wet form. However, it's a controversial—and painful—treatment, and only one of the drugs—Lucentis (ranibizumab)—is approved in the US for wet AMD treatment.

However, doctors are now starting to recommend something called 'photodynamic therapy' (PDT), using the light-activated drug Visudyne (verteporfin), for some forms of wet AMD. The drug is injected into the patient's arm and, after a short wait, a laser beam is shone into the patient's eyes to activate the drug, which is supposed to seal up abnormal blood vessels and destroy any that are leaking. However, PDT has proved to be less effective than Lucentis injections in a study of 423 AMD patients over a two-year period (Ophthalmology, 2009; 116: 57–65.e5).

Possible causes of AMD

While macular degeneration primarily affects older people, there's little evidence to suggest that ageing is, on its own, the major cause of such failing eyesight. AMD affects 10 per cent of people aged up to 74 years and, despite its name, increases to only around a third of those between 75 and 85 years of age (www.agingeye.net/maculardegen/maculardegeninformation.php).

Medicine believes that one of its causes could be exposure over the years to direct sunlight and, especially, to blue-spectrum light. Doctors often advise older people to wear sunglasses in direct sunlight to lessen their risk of developing AMD. This advice, however, may have been based on a series of animal studies that did not properly replicate the human experience. In a series of laboratory tests, researchers shone intense ultraviolet light into animals' eyes, which were held open mechanically. Aside from the fact that these studies amounted to extreme animal cruelty, the tests also failed to take account of the blinking reflex and the way that we humans have of avoiding looking directly at the sun.

This idea has also been supported by a Cambridge, UK, study of 446 AMD sufferers that found that direct sunlight is not a cause (Br J Ophthalmology, 2006; 90: 29–32), whereas other studies suggest that, in fact, sunlight is important for good health. Blue light, in particular, helps the body to release melatonin, which protects the heart and the eyes by keeping blood pressure levels low (J Clin Endocrinol Metab, 2003; 88: 4502–5).

Another commonly held belief—that alcohol can cause AMD—may also be mistaken. A major study involving 4439 people living in Beijing and in rural areas of China could find no connection between wine- and beer-drinking, and AMD (Ophthalamology, 2009; August 25, published online ahead of print).

Instead, there's growing evidence to suggest that AMD is more likely to be the result of a mineral imbalance that is cumulative and so becomes more apparent as we get older. Low levels of zinc and copper are commonly found in people with AMD, as one study discovered when it analyzed the health profiles of 44 subjects with the condition. On average, the subjects' zinc and copper levels were 24-per-cent lower than those of the healthy controls. The researchers, from the Mayo Clinic in Rochester, Minnesota, also pointed out that, as shown by other studies where supplements were able to slow the progress of AMD, there appears to be a direct causal link between zinc and copper levels, and failing eyesight (Am J Ophthalmol, 2009; 147: 276–82.e1).

On the other hand, iron tends to cluster in the retina, and may also play a role in a broad range of ocular diseases, including AMD (along with glaucoma, cataract and conditions causing intraocular haemorrhage) because of iron-induced ocular oxidative damage (Prog Retin Eye Res, 2007; 26: 649–73).

Furthermore, too much lead can cause AMD. One study of 25 AMD

patients discovered that they all had retinal lead levels that were up to 75 per cent higher than those found in people with healthy eyes (Am J Ophthalmol, 2009; September 4, published online ahead of print).

A diet that's high in processed foods can also lead to AMD. A survey of more than 4000 people, carried out by researchers at Tufts University in Boston, MA, concluded that up to 20 per cent of all cases of AMD could have been avoided by a diet lower in processed foods such as white bread, cakes and biscuits (Am J Clin Nutr, 2007; 86: 180–8).

What's more, new breakthrough research suggests that AMD could be an inflammatory disease brought about by a polymorphism (variation) in the complement factor H (*CFH*) gene. Researchers from the US National Eye Institute (NEI) and the National Cancer Institute (NCI) reckon that the gene variation could be responsible for half of all cases of AMD. Indeed, people who carry the polymorphism are nearly six times more likely to develop the condition (Science, 2005; 308: 385–9).

Preventing AMD

Age-related macular degeneration (AMD) has features in common with heart disease, and the many ways of maintaining a healthy cardiovascular system appear to also apply to eye health.

- **Eat an organic, unprocessed diet that's low in fats, and high in fruit and vegetables**—especially lutein-rich varieties such as kale and spinach
- **Supplement with zinc and copper every day**, and take amounts that are higher than the recommended daily allowance (RDA)
- **Drink a glass of red wine a day** (Am J Ophthalmol, 1995; 120: 190–6) as, in one study, those who drank one glass a day reduced their risk of AMD by 20 per cent compared with those who either drank beer or spirits, or were teetotalers (Lancet, 1995; 351: 117)
- **Don't smoke**
- **Supplement with antioxidants A, C and E**
- **Eat plenty of tomatoes**, as they are rich in lycopene, which helps ward off AMD (Arch Ophthalmol, 1995; 113: 1518–23)
- **Take vitamins B6 and B12 together with folic acid**, as they can reduce your AMD risk (Arch Intern Med, 2009; 169: 335–41)
- **Taking the antioxidant zeaxanthin** can reduce your chances of developing AMD. Besides supplements, it's also found in foods such as mangoes, papaya, oranges, peaches, green beans, broccoli, sweet potatoes and honeydew melon (Am J Clin Nutr, 1995; 62 [6 Suppl]: 1448S–61S). In addition, the density of the macular pigment can be improved just by adding corn and spinach to the diet (Invest Ophthalmol Vis Sci, 1997; 38: 1795–801).

Possible treatments

If AMD is an inflammatory disease, then high-dose supplements could be an effective therapy. Vitamin B12 supplements have been successfully used to reduce the symptoms of other inflammatory conditions such as bursitis (Lininger SW Jr *et al., The Natural Pharmacy: Complete Home Reference to Natural Medicine*. New York, NY: Three Rivers Press, 1999), and vitamin C, at high doses, has been proven to have anti-inflammatory properties (Exp Eye Res, 1986; 42: 211–18). Vitamin E may be especially useful as it is able to suppress the symptoms of inflammation in specific parts of the body (J Vitaminol, 1972; 18: 204–9).

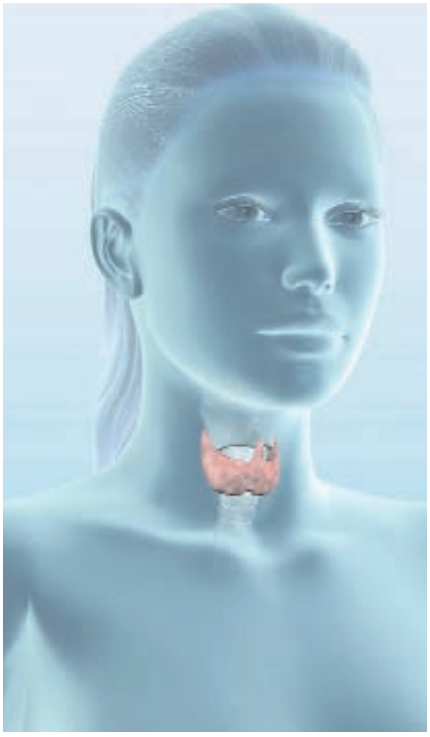
Copper and zinc are both strong anti-inflammatories, and AMD sufferers are usually deficient in both. Inflammation requires a higher copper intake to maintain levels of enzymes that are vital to the body's anti-inflammatory processes, at least in animal studies (Agents Actions, 1985; 16: 504–13). In addition, findings in animal (rat) studies showed that zinc is an important healing agent during inflammation (Int J Tissue React, 1981; 3: 73–6). In clinical studies, zinc was able to help 70 per cent of men suffering from prostate problems (Bush IM *et al.*, 'Zinc and the Prostate', presentation at the annual meeting of the American Medical Association, Chicago, 1974). Supplemental zinc, copper and manganese also reduced the risk of rheumatoid arthritis in more than 29,000 women aged 55–69 years and followed for 18 years (Am J Epidemiol, 2003; 157: 345–54).

High-dose supplements of zinc and copper, and of vitamins A, C and E—the usual antioxidants—prevented the progression of AMD in one study. The researchers, from the Age-Related Eye Disease Study Group in New Zealand, also found that most people are taking levels of vitamins that are too low to be effective. Yet, at the correct dosages, any combination of multivitamins and individual supplements can prevent AMD from worsening (N Z Med J, 2009; 122: 32–8).

Lutein/zeaxanthin, the carotenoid antioxidants found in dark-green leafy vegetables such as spinach and kale, are both highly effective in slowing the progress of AMD. This finding was made when these vegetables were added to the diets of 4519 study participants, aged 60–80 years (Arch Ophthalmol, 2007; 125: 1225–32).

Bryan Hubbard

Thyroid problems: never say forever



Doctors tell us that thyroid problems happen as we get older and that we have to live with them, but neither claim is true

Medicine just doesn't seem to be able to get it right when it comes to thyroid problems, and especially hypothyroidism, when we become sluggish and overweight because of an underactive thyroid gland.

Hypothyroidism is by far the more common of the two thyroid problems—the other is hyperthyroidism, when the body goes into overdrive—and just 10 years ago, it was considered one of the great undiagnosed diseases. But nowadays, doctors are accused of having a complete knee-jerk reaction, and are seeing thyroid problems where none exist and giving people hormone drugs unnecessarily.

"This is potentially an enormous problem, given that one in four people have their thyroid function checked," says Jayne Franklyn, president of the British Thyroid Association (BMJ, 2009; 338: b725).

As a result, patients who are wrongly being given a hormone drug such as levothyroxine could be suffering from serious effects due to an excess of thyroid hormones. Worse, the misdiagnosis could also be masking more serious conditions such as depression.

Thyroid diagnosis

The thyroid, an endocrine gland found at the base of the neck, plays an essential role in the body's functioning and metabolism. It controls the rate at which the body burns energy and makes proteins by releasing the hormones T4 (thyroxine) and T3 (triiodothyronine). The thyroid may be producing either too little of these hormones (hypothyroidism) or too much (hyperthyroidism). Hypothyroidism can also result from these hormones failing to reach the body's tissues.

Doctors have just two ways of diagnosing a thyroid problem: they can either look for symptoms, or conduct a blood test. However, both methods can be highly unreliable. Typical symptoms of hypothyroidism include tiredness, feeling the cold, dry skin, depression and weight gain, but these features can also be caused by a range of other health problems. Indeed, thyroid disease often mimics other, more serious, problems. Symptoms of Graves' disease, which can be caused by hyperthyroidism, are almost identical to many of the physiological changes that occur during pregnancy, making the diagnosis almost impossible in expectant mothers (Endocr Pract, 2009; Oct 15: 1–36; Epub ahead of print).

A more reliable diagnostic test is to carry out a standard blood test that measures thyroid-stimulating hormone (TSH) levels. But this can also produce false-positive results, especially if the patient has another health problem. If a second blood test were to be carried out after the true illness has been cleared up, TSH levels would probably show up as normal. Unfortunately, second tests are rarely ordered, and the patient will often already have started an unnecessary course of hormone drugs, which can have serious side-effects.

Causes

Doctors also tend to get it wrong when it comes to understanding the causes of thyroid disease. The standard view is that thyroid problems are either the result of people living longer or because of a genetic inheritance. It used to be caused by iodine deficiency but, at least in the West, so much of our food these days has been enriched with iodine that this cause has all but been eliminated. If anything, we are now in danger of consuming too much iodine, especially through iodine-enriched salt, which can lead to iodine-induced thyrotoxicosis, or hyperthyroidism (BMJ, 1976; 1: 372–5).

So, having eliminated iodine deficiency as a cause, medicine believes that the other two causes—ageing and genetics—are beyond the patient's control, and that only an outside agent such as a drug—levothyroxine for hypothyroidism and a beta-blocker for hyperthyroidism—is able to normalize the patient's hormone levels. In fact, levothyroxine is just as likely to cause an excess of thyroid hormones (Lancet, 1991; 337: 171–2).

But the above-mentioned two causes certainly don't account for most cases of thyroid disorders. Indeed, the growing evidence suggests that hypothyroidism and hyperthyroidism are more often caused by environmental factors and by diet—including, paradoxically, consuming too much iodized salt, which was originally enriched to combat thyroid dysfunction.

Other causes that doctors appear to overlook include:

- **food allergies**. People with multiple food allergies, such as coeliac disease, are much more likely to develop thyroid problems (J Pediatr, 2009; 155: 51–5).
- **selenium deficiency**. The thyroid gland has some of the highest concentrations of selenium in the

body, so any deficiency in this essential trace element can lead to thyroid disease (Best Pract Res Clin Endocrinol Metab, 2009; 23: 815–27).

- **pollutants**. Common environmental pollutants such as perchlorate, thiocyanate and nitrate suppress iodine absorption and have been found to cause thyroid disease in both animal and human studies (Toxicol Ind Health, 1998; 14: 121–58; Best Pract Res Clin Endocrinol Metab, 2009; 23: 801–13).
- **cancer treatment**. The healthy functioning of the thyroid can be affected by radiotherapy given for cancer (Am J Clin Oncol, 2009; 32: 150–3).
- **diabetes**, especially type 1, can cause thyroid problems (Turk J Pediatr, 2009; 51: 183–6).
- **preeclampsia**, a common complication of pregnancy, can affect thyroid functioning later in life (BMJ, 2009; 339: b4336).
- **fluoride**. This chemical in our water supply can interfere with the natural absorption of iodine (Klin Wochenschr, 1984; 62: 564–9).
- **iodine excess**. This element is considered to be essential for healthy thyroid functioning, but too much can lead to hyperthyroidism (J Endocrinol Invest, 1994; 17: 23–7). Avoiding iodine-enriched salt may not be enough, as iodine is also used in cough expectorants, antiseptics, and certain drugs and imaging contrast media (Z Kardiol, 2001; 90: 751–9).
- **prescription drugs**. Even drugs that don't contain iodine can cause thyroid problems. These include lithium, given for bipolar disorder (manic depression), which often causes an underactive thyroid (N Engl J Med, 1995; 333: 1688–94), as does the heart drug amiodarone (BMJ, 1996; 313: 539–44).
- **emotional problems**. The thyroid is especially vulnerable to emotional traumas such as bereavement and divorce (Acta Endocrinol, 1993; 128: 293–6).

Alternatives to drugs

If your thyroid function needs to be stabilized, there are plenty of proven alternatives to drugs that may do the job just as well—and without the serious side-effects.

- ***Bauhinia purpurea***. A bark extract of this flowering plant can significantly improve blood T3 and T4 levels, although the only data apparently comes from laboratory mice. In the same study, a root extract of *Withania somnifera* (Ashwagandha) was also tested, but only could only enhance T4 levels (J Ethnopharmacol, 1999; 67: 233–9).
- **Zinc**. A deficiency of this mineral can not only cause hypothyroidism, but supplementing with zinc may also reverse the condition (Int J Dermatol, 1976; 15: 757–61).
- **Bladderwrack (*Fucus vesiculosus*)**. If your thyroid problems are related to iodine insufficiency, this seaweed is one way of increasing your iodine intake (Food Add Contam, 1987; 5: 103–9).
- **Neuro Emotional Technique™ (NET)**. This mind–body technique can help to normalize thyroid dysfunction. In one study of two hypothyroid patients, NET was successful in raising their TSH and T4 levels back to normal (Complement Ther Clin Pract, 2009; 15: 67–71).
- **Armour® Thyroid replacement therapy**. This desiccated thyroid extract is derived from pigs and is an alternative to a synthetic hormone replacement drug. One study found this extract to be more effective for hypothyroidism than a drug (N Engl J Med, 1999; 340: 424–9).

It's not forever

A common belief in medicine is that a thyroid problem is forever. As a result, thyroid patients will probably have to take a drug to maintain normal hormone levels for the rest of their lives. In a few rare cases, this may indeed be necessary, but the evidence suggests that it's much more likely that thyroid problems wax and wane, assuming that the patient even had the problem in the first place.

Sadly, medicine isn't geared up for such an eventuality, and often won't even take a second, confirmatory TSH blood test a few months later, when the real cause of the imbalance may have gone away, and hormone levels may have normalized.

It's also more likely that the cause of the thyroid problem is within the control of the patient, and a little detective work can often uncover the true culprit, which then can be sorted out. Often, for example, the problem is caused by environmental pollutants, especially fluoride, and these can be reduced or removed by using air and water filters. Certainly, thyroid problems can worsen as toxins build up in the body, which may lead doctors to believe that they are the direct result of ageing.

But one thing's for sure: thyroid problems are usually not 'just one of those things'. In many cases, they can be either avoided or reversed—and without the use of powerful pharmaceuticals.

Bryan Hubbard

Hearing loss

Most hearing loss is simply seen as an inevitable consequence of growing old. Yet, the evidence suggests that it can be prevented.

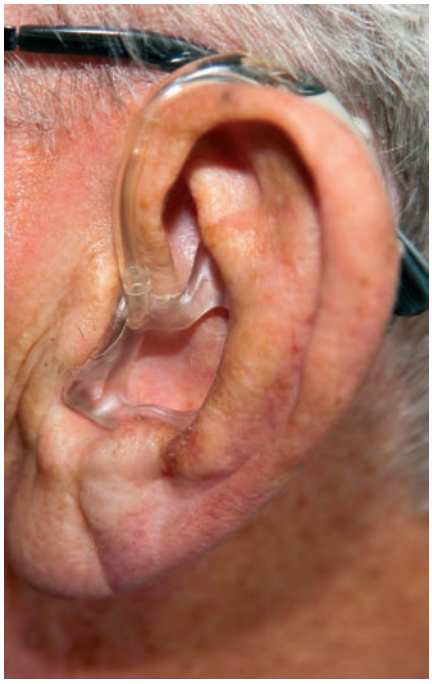
Most of us assume that hearing loss is a normal and inevitable part of ageing—and that's a viewpoint that appears to be well supported by the facts. In the US, hearing loss ranks as the third most common chronic condition in the elderly, while UK figures show that more than half the population of those aged over 60 have some degree of impaired hearing. There's even a medical term—presbycusis—to describe this gradual form of hearing loss.

However, emerging evidence now indicates that the problem may have more to do with lifestyle than simply growing old. Studies show that a number of environmental factors, including noise pollution, diet, chemical exposures and even prescription drugs, can contribute to hearing loss. Crucially, the evidence suggests that hearing loss may well be a preventable condition.

Noise pollution

Repeated exposure to loud noise is one of the most common causes of hearing loss. When noise is too loud—in general, this means above 85 decibels (dB), the level of noise made by heavy city traffic—prolonged exposure can destroy the sensitive hair cells in the inner ear. These hair cells move as sound waves travel through the ear structures, and the movement is converted to nerve impulses that are interpreted by the brain as sound.

A single loud noise, such as a gunshot blast, can permanently harm these inner-ear structures—although years of exposure to less intense sounds, such as loud music, can also cause irreversible damage (Pediatrics, 2001; 108: 40–3). As well as impaired hearing, such exposure to loud noise can lead to tinnitus (persistent ringing in the ear), hyperacusis (extreme oversensitivity to sound) and other



hearing disorders (Int J Audiol, 2003; 42: 279–88).

Traditionally, noise-induced hearing loss (NIHL) has been considered a disease of adults who worked in noisy occupations or used firearms. However, there's growing concern that children and young adults are now developing the condition as a result of overexposure to amplified music, especially through the use of personal music devices such as MP3 players.

As a recent article in the *British Medical Journal* pointed out, "The devices increasingly use earphones that insert into the ear canal which produce higher sound levels in the ear than 'over-the-ear earphones' used at the same volume. These sound levels can exceed 120 decibels, similar in intensity to a jet engine" (BMJ, 2010; 340: c1261). Indeed, several small, controlled studies have found that personal music players are associated with poorer hearing function in adolescents and young adults (Noise Health, 2009; 11: 132–40; J Otolaryngol Head Neck Surg, 2008; 37: 718–24).

Although more research is needed to clarify the role of personal music players in hearing loss, in the meantime, the advice is simple: turn down the volume and take regular breaks from using them. As a rule of thumb, if the music is uncomfortable for you to listen to, or if you can't hear any external sounds when you've got your headphones on, then the volume is too loud.

In addition, it should be borne in mind that the higher the volume of the sound, the shorter the time you should be listening to it. University of Colorado researchers have placed a safe listening limit of 4.6 hours per day on an iPod played at 70-per-cent of its volume if you're using stock iPod earphones. However, this duration falls precipitously to just five minutes if the volume is cranked up to maximum (www.colorado.edu/news/releases/2006/346.html).

Other ways to reduce the chances of developing NIHL include:

- **wearing hearing protectors**, such as earplugs or earmuffs, when you know you're going to be around dangerously loud noise. Lawn-mowers, power tools, rock concerts and motorcycles, for example, can all cause permanent damage to hearing if you don't wear ear protection;
- **turning the volume down** on your stereo, television and car radio. Also, avoid buying noisy toys, appliances or tools when there are quieter alternatives, and don't use several noisy machines at the same time. What's more, don't try to drown out unwanted noise with other sounds: for example, don't turn up the volume on your car radio or headset to drown out traffic noise, or turn up the television volume while vacuuming;
- **using sound-absorbing materials** to reduce noise at home and at work. Rubber mats can be placed under noisy kitchen appliances, computer printers and typewriters to cut down on noise. Curtains and carpeting also help to reduce indoor noise, and double-glazed windows can reduce the amount of outside noise that can penetrate into the home or workplace;
- **having your hearing checked regularly**, especially if you are frequently exposed to loud noise at either work or play.

Nutrition

Besides noise, what we eat can also have an impact on our hearing. High fat and cholesterol consumption can lead to high blood-cholesterol levels

and the production of free radicals. This, in turn, can lead to impaired hearing by reducing the flow of oxygen and nutrients to the inner ear. Indeed, a study conducted in Taiwan—the largest of its kind—found that those who had high levels of triglycerides (a form of fats made by the body) in their blood had a significantly greater risk of developing NIHL (Otolaryngol Head Neck Surg, 2007; 137: 603–6).

Similarly, researchers in Turkey reported that people with NIHL were highly likely to have hyperlipidaemia—an excess of cholesterol and fatty acids in the blood. The team also found that a low-cholesterol diet helped to improve hearing, and alleviated tinnitus in these patients (Int Tinnitus J, 2007; 13: 143–9).

On the other hand, certain fats may have an important role to play in protecting our hearing. A study recently published in the *American Journal of Clinical Nutrition* found that consumption of omega-3 fatty acids—in particular, the long-chain variety found in fish oil—reduced the risk of age-related hearing loss. Participants who consumed two or more servings of fish per week had a 42-per-cent lower risk of developing presbycusis compared with those who ate less than one serving a week. In addition, in those who already have hearing loss, fish consumption appears to prevent it from getting worse (Am J Clin Nutr, 2010; 92: 416–21).

However, much of the research on nutrition and hearing loss has focused on antioxidants. Emerging evidence shows that free-radical formation in the inner ear plays a key role in the development of NIHL. Antioxidants, which mop up free radicals, may therefore be an effective intervention.

Animal studies (so the results may not necessarily apply to humans) have demonstrated that a variety of dietary antioxidants, including vitamins A, C and E, can reduce NIHL when given prior to noise exposure. A combination of high-dose vitamin A (2.1 mg/kg), vitamin C (71.4 mg/kg) and vitamin E (26 mg/kg) plus magnesium (343 mg/kg) can prevent NIHL when taken one hour prior to noise exposure and continued once a day for five days subsequently (Free Radic Biol Med, 2007; 42: 1454–63). It appears that the vitamins work in synergy to reduce both free-radical formation and inner-ear hair-cell damage, while the magnesium preserves blood flow to the inner ear, as this is also affected by loud noise.

Remarkably, antioxidants delivered as late as three days after noise exposure were still found to be beneficial (Neuroscience, 2005; 134: 633–42)—although, again, these animal test results may not necessarily apply to humans.

Nevertheless, there is evidence to support the use of magnesium for human hearing. In a two-month, double-blind, placebo-controlled study of 300 Israeli military recruits, daily supplementation with 167 mg of magnesium significantly helped to protect their hearing against noise-induced damage (Am J Otolaryngol, 1994; 15: 26–32). Another Israeli study of 20 young men found similar results (Clin Otolaryngol Allied Sci, 2004; 29: 635–41).

Other nutrients that may be involved in ear health are folate and vitamin B12. A recent study found that, among men aged 60 years or over, those with the highest folate intakes had the lowest risk of hearing loss (Otolaryngol Head Neck Surg, 2010; 142: 231–6).

In an earlier study, which looked at a group of army personnel exposed to military noise, vitamin B12 deficiency was commonly found among those with NIHL and/or chronic tinnitus. Interestingly, 12 patients who had vitamin B12 replacement therapy saw their symptoms improve (Am J Otolaryngol, 1993; 14: 94–9).

Drugs and other toxins

A staggering range of drugs are associated with hearing loss—from painkillers to oral contraceptives. A recent study has revealed that the regular use of aspirin, acetaminophen (paracetamol) and non-steroidal anti-inflammatory drugs (NSAIDs) can significantly increase the risk of hearing loss in men (Am J Med, 2010; 123: 231–7).

Antibiotics, especially aminoglycosides such as amikacin, gentamicin and tobramycin, chemotherapy drugs, hormone replacement therapy (HRT), antimalarials and loop diuretics have also all been shown to have ototoxic (ear-damaging) effects (Braz J Otorhinolaryngol, 2006; 72: 836–44; Hear Res, 2009; 252:

Can hearing loss be treated?

Usually, a hearing aid is the only 'treatment' for age-related hearing loss, but there's now evidence to suggest that certain herbs and supplements may be able to restore hearing loss.

- **Antioxidants** are among the most promising therapies for hearing loss. In one study, alpha-lipoic acid (60 mg/ day), vitamin C (600 mg/day) and the drug rebamipide (300 mg/day), an amino-acid derivative that scavenges damaging free radicals, were given orally for at least 8 weeks to 46 elderly patients with hearing loss. At the end of the treatment, hearing levels were significantly improved at all frequencies tested (Acta Otolaryngol, 2009; 129: 36–44).

  Another study—a placebo-controlled trial this time—demonstrated that supplementing with both vitamins E (600 mg/day) and C (1200 mg/day) can be beneficial for sudden-onset sensorineural hearing loss (nerve deafness) (Acta Otolaryngol, 2008; 128: 116–21).
- ***Ginkgo biloba*** may be useful for hearing loss, according to several studies. In one, the herb was better than the conventional combined drug treatment when tested in 52 patients with acquired sensorineural hearing loss (Indian J Otolaryngol Head Neck Surg, 2000; 52: 212–9).

  Although an optimal dose has yet to be established, in general, studies have used 30–200 mg daily, with the higher doses tending to provide better results.
- **Correcting nutritional deficiencies** may help to restore hearing in some cases. Indeed, one study reported that zinc supplementation in patients who were marginally zinc-deficient improved tinnitus and sensorineural hearing loss in about one-third of elderly adults (Am J Otol, 1989; 10: 156–60).

29–36).

Apart from medication, a number of other agents have also been linked to hearing loss, including pesticides, toluene, styrene, ethylbenzene, carbon disulphide, lead and mercury (Braz J Otorhinolaryngol, 2006; 72: 836–44).

Smoking also appears to damage hearing—possibly by affecting the antioxidative mechanisms of the body or reducing the blood circulation to the hearing system. Indeed, a study of people working in noisy environments found that long-term smokers were more likely to develop permanent hearing loss than non-smokers (Clin Otolarygol, 2005; 30: 517–20).

Another found that smokers had a 69-per-cent increased risk of hearing loss compared with non-smokers, and even non-smokers living with smokers were more likely to suffer hearing loss, too (JAMA, 1998; 279: 1715–9). Indeed, infants exposed to cigarette smoke had a fivefold greater incidence of hearing loss than unexposed infants (Ir Med J, 1992; 85: 111–2).

A sound solution

So far, the evidence suggests that hearing loss is not something that should simply be accepted as an inevitable part of ageing. It is a condition that can be prevented by paying attention to what we eat, to our noise exposure and to the toxins that surround us on a regular basis.

Ultimately, if you follow a healthy, balanced diet that includes plenty of antioxidants and good fats, and focus on turning the volume down in your life, you should be well on the way to ensuring the sweet sounds of good hearing for years to come.

Joanna Evans

Parkinson's disease

Clean up your diet and lifestyle to avoid this debilitating disease

Parkinson's disease (PD) is a debilitating neurological disorder thought to affect one or two people in every 1000. It's named after Dr James Parkinson, the Scottish physician who first described it as the 'shaking palsy' in an 1817 essay.

Although it's been around for some time, modern medicine is still baffled by the condition. The symptoms—including tremors, muscle rigidity and slowed movements—can so closely resemble a number of other disorders that PD is, in fact, notoriously difficult to diagnose.

Indeed, a recent study has reported that at least one in 20 Parkinson's cases is a misdiagnosis (Mov Disord, 2009; 24: 2379–85). This means that over 6000 people in the UK could be taking dangerous anti-Parkinson's drugs unnecessarily. Alarmingly, many of these drugs can even cause the symptoms they're meant to treat—such as confusion and involuntary movements—and such side-effects are not always reversible (Am Fam Physician, 2007; 75: 1045–8).

Possible causes

Medicine is also confused as to what exactly causes PD. The symptoms are thought to arise from a lack of the chemical messenger dopamine in the brain, which happens when the specific brain cells that produce it die or become impaired. However, researchers still don't know what triggers such a chain of events in the first place.

Also, recent evidence suggests that ageing is less important than has been previously believed (Environ Health Perspect, 2005; 113: 1234–8). Indeed, what's emerging is that environmental factors throughout life—such as what we eat, where we live and what we do for a living—are crucial pieces of the puzzle.

So far, it appears that exposures to particular toxins or foods can increase the risk of PD.

- **Pesticides**. Numerous animal and human studies link pesticides to PD. People living within 500 metres of land sprayed with pesticides have a 75-per-cent increased chance of developing the disease. In addition, those exposed to agricultural pesticides as a child or young adult have an even greater risk (Am J Epidemiol, 2009; 169: 919–26).

  Household pesticide use is also associated with PD. US researchers reported that using insecticides and herbicides in your home or garden can double your risk of developing the disease (Lancet, 2000; 335: 1701; http://news.bbc.co.uk/2/hi/health/738020.stm).
- **Polychlorinated biphenyls (PCBs)**. These man-made organic compounds, formerly widely used in electrical and hydraulic equipment, were linked to PD in two US occupational studies, which found that female workers exposed to PCBs were around two to three times more likely to die with PD compared with the general population (Environ Health, 2006; 5: 13; Epidemiology, 2006; 17: 8–13). There's also considerable animal and laboratory evidence to back this up. In fact, even low levels of PCB exposure—as seen in the general population—may have the potential to disrupt normal dopamine functioning in the brain, which could eventually lead to PD (Toxicol Sci, 2006; 92: 490–9).
- **Heavy metals**. One study found that those with the highest lifetime lead exposures are twice as likely to have PD as those with the lowest exposures (Environ Health Perspect, 2006; 114: 1872–6). Other metals, including aluminium, mercury, copper, manganese and iron, may also be risk factors (Neuroepidemiol-ogy, 1999; 18: 303–8).
- **Dairy**. Harvard researchers found that high intakes of dairy foods nearly doubled the risk of PD in men (Ann Neurol, 2002; 52: 793–801), and there's a slightly increased risk in women, too (Am J Epidemiol, 2007; 165: 998–1006).
- **Fruit**. Researchers at the University of Hawaii found a connection between high intakes of fruit and fruit juice, and PD in middle-aged men. Those who ate more than three servings a day had a 70-per-cent higher risk, while those eating only one or more servings a day increased

Alternative and complementary therapies

Although there's no cure for Parkinson's disease (PD), its symptoms and quality of life can be improved; however, only attempt the following under medical supervision.

- **Diet**. The standard therapy for PD is levodopa, or L-dopa, but its effectiveness can vary throughout the day. These fluctuations can be reduced when almost all of the day's protein is eaten in the evening meal (Neurology, 1989; 39: 552–6). In one trial of 43 patients, 70 per cent were successfully using such a protein redistribution diet after more than a year (Arch Neurol, 1992; 49: 149–51).
- **Supplements.** These appear to be beneficial for PD.
  - **Coenzyme Q10**. PD patients have reduced levels of this antioxidant in their blood and brain (Neurosci Lett, 2008; 447: 17–9). In those with early PD, 1200 mg/day of CoQ10 for 16 months significantly slowed the course of the disease compared with a placebo (Arch Neurol, 2002; 59: 1541–50).
  - **B vitamins**. It's been observed that those who have a high intake of vitamin B6 have a lower risk of PD (Neurology, 2006; 67: 315–8). B6 supplements, therefore, may be useful for those in the early stages of the disease (although this vitamin should not be taken by people who have heart conditions).
  - **Vitamins C and E**. In patients with early PD, high doses of these antioxidants (750 mg of ascorbate and 800 IU of alpha-tocopherol, four times a day) delayed the need for L-dopa by an average of around 2.5 years (Ann Neurol, 1992; 32 Suppl: S128–32).
  - **Amino acids**. In one small trial, three weeks of treatment with the amino acid L-methionine improved rigidity, tremor and other symptoms in previously untreated PD patients. "Therapeutic effects were similar to those observed with L-dopa treatment," the researchers said (Rev Neurol [Paris], 1982; 138: 297–303). Other possibly helpful amino acids are L-tryptophan (in conjunction with L-dopa) and L-tyrosine (Int J Neurosci, 1989; 45: 215–9; C R Acad Sci III, 1989; 309: 43–7).
- **Herbs**. An herbal remedy, called HP-200, containing an extract of the tropical legume *Mucuna pruriens*, was found to considerably reduce PD symptoms, with only mild, mainly gastrointestinal, side-effects (J Altern Complement Med, 1995; 1: 249–55).

More recently, *Mucuna* was tested against the standard PD drugs L-dopa and carbidopa by the UK's Institute of Neurology. Patients were randomly given single doses of either 15 g or 30 g of *Mucuna* for one week, then single doses of 200 mg and 50 mg of standard L-dopa and carbidopa, respectively, the next week. The trial was randomized, so no one knew who was taking what. *Mucuna* worked faster and had longer-lasting benefits than the standard drug treatment—with far fewer side-effects (J Neurol Neurosurg Psychiatry, 2004; 75: 1672–7).

- **Acupuncture**. A US study found that two sessions a week for an average of around six weeks led to improvements in tremor, walking, handwriting, slowness, pain, sleep, depression and anxiety in 85 per cent of the study participants. What's more, there were no adverse effects from the treatment (Mov Disord, 2002; 17: 799–802).
- **Magnetic therapy**. Repetitive transcranial magnetic stimulation (rTMS), a non-invasive technique that stimulates neurons in the brain, is showing promise for PD. A recent meta-analysis, which combined the results of 10 randomized trials in PD patients, reported a significant improvement in motor function with high-frequency rTMS treatment (Mov Disord, 2009; 24: 357–63).
- **Exercise**. Physical activity appears to be beneficial for PD patients by improving their physical performance and activities of daily living (Clin J Sport Med, 2006; 16: 422–5). In particular, it can help with balance, thereby potentially reducing the risk of falls (J Neurol Phys Ther, 2009; 33: 14–26).
- **The Alexander Technique**. This form of physical re-education of the body can help PD patients both physically and mentally, according to one study. Patients who were given lessons in the technique were significantly less depressed, and reported greater improvement in their disability, compared with those not given any such lessons (Clin Rehabil, 2002; 16: 695–708).
- **Detox**. As toxins such as pesticides and heavy metals are thought to be involved in the development of PD, encouraging their removal from the body may be helpful. See our special report in *WDDTY* vol 20 no 10 (page 15) for what works for detoxification.

their risk by 55 per cent. The researchers noted that it is unlikely that fruit itself is the problem but, rather, the increased exposure to plant-borne toxins, pesticides or herbicides (Presentation at the American Academy of Neurology Annual Meeting in Honolulu, 2003).

Preventing Parkinson's

These data suggest that lifestyle changes, such as choosing organic fruit, limiting dairy, and avoiding pesticides and other toxic chemicals, may help to prevent PD. Also, drinking tea (CNS Neurosci Ther, 2008; 14: 352–65), taking regular exercise (Mov Disord, 2008; 23: 69–74) and maintaining a healthy weight (Neurology, 2006; 67: 1955–9) all appear to reduce PD risk.

Interestingly, experts now reckon that genes alone are responsible for less than 10 per cent of PD cases—and even then, the environment may play a part (Environ Health Perspect, 2009; 117: 117–21). It's likely that environmental factors, such as diet and pesticide exposure, interact with genes to bring about the disease, suggesting that, with more research, the vast majority of cases may be preventable.

Joanna Evans

Osteoporosis: the brain–bone connection

New research has revealed an intriguing link between osteoporosis and depression

Osteoporosis, a leading cause of bone fractures, is the most widespread degenerative disease in the West, affecting around three million people in the UK and 10 million in the US. Women are most at risk, with one in six Western women expected to suffer a hip fracture at some point in their lives.

Our genes, age, diet and lifestyle are all known to have an influence on our bone health. Now, it appears that our mental health may also have a significant part to play.

Indeed, intriguing new research has found that depression, a condition that afflicts more than 120 million people worldwide, could be an important risk factor for osteoporosis—and especially in women.

The brain–bone link

Two recent meta-analyses—pooling the results of several previous studies—have found a clear connection between depression and low bone mineral density (BMD), an indicator of osteoporosis and fracture risk. The latest, an international study carried out by scientists at the Hebrew University of Jerusalem, assessed data from 23 research projects in eight countries, comparing bone density in 2327 people suffering from depression with that of 21,141 non-depressed individuals.

The researchers found that the depressed had substantially lower BMD than the non-depressed, and that depression was also associated with markedly greater activity of osteoclasts, the cells that break down bone.

What's more, the connection between depression and bone loss was particularly strong in premenopausal women with clinical depression diagnosed by a psychiatrist.

Bone-building alternatives

A variety of drugs can prevent and treat osteoporosis, but they all come with a raft of undesirable side-effects. Bisphosphonates, for instance, stop bone breakdown, but are linked with severe bone pain, oesophageal cancer, heart-rhythm irregularities and osteonecrosis of the jaw (ONJ), where bone tissue fails to heal after even minor trauma, such as tooth extraction (Curr Opin Rheumatol, 2009; 21: 363–8). Happily, a combination of vitamins and minerals such as strontium and magnesium offer an alternative.

- **Calcium** is important for bone health although, says Annemarie Colbin, too much emphasis is placed on this mineral alone. Indeed, there's evidence that too much calcium can even increase the risk of broken bones (Am J Epidemiol, 1997; 145: 926–34). Healthy bones require many nutrients—magnesium, phosphorus, boron, copper, manganese and zinc, plus vitamins C, D, K, B6 and folic acid—all working together. We also need sufficient amounts of protein for healthy collagen, and healthy fats for vitamin D uptake and protection against bone-destroying free radicals (for more information on calcium, see *WDDTY* vol 20 no 3).
- **Vitamin D** is essential for strong bones and calcium absorption, and not having enough D has been linked to osteoporosis and low bone mineral density (BMD) in women aged over 50 (Joint Bone Spine, 2008; 75: 567–72). You can get D from food (fish, liver, eggs), but the best source is the sun. Just 15 minutes of sunshine on your skin every day should produce all the vitamin D you need. In winter, you may need to take supplements. Studies show a 30-per-cent decrease in non-spinal fractures among seniors taking 800 IU/day of vitamin D (Ann Med, 2005; 37: 278–85).
- **Vitamin K** is not just important for blood-clotting, but also plays a major role in bone metabolism. In a three-year study of nearly 200 women aged 50–60 years, those taking vitamin K1 (phylloquinone; 1 mg/day) with a mineral + vitamin D supplement lost less bone than those taking a placebo or the mineral + vitamin D alone (Calcif Tissue Int, 2003; 73: 21–6). Vitamin K2 (menatetrenone; 45 mg/day) boosted BMD and reduced spinal fractures in osteoporosis sufferers almost as effectively as etidronate, but without the side-effects of the bisphosphonate (J Orthop Sci, 2001; 6: 487–92). However, due to its blood-coagulating properties, vitamin K should never be taken with warfarin.

  Good food sources of vitamin K include leafy green vegetables and fermented products such as natto.
- **Strontium** appears to have the dual effect of increasing bone formation while decreasing bone breakdown (Bone, 2008; 42: 129–38). In a three-year study of 1649 postmenopausal women with osteoporosis, 2 g/day of oral strontium ranelate (a prescription drug) increased BMD and reduced fracture risk by more than 40 per cent *vs* a placebo (N Engl J Med, 2004; 350: 459–68).

  However, side-effects included diarrhoea, nausea and even memory loss with long-term treatment (Drugs, 2010; 70: 733–59). Natural sources of strontium include wholegrains, parsley, fish, Brazil nuts and lettuce.

The scientists concluded that "all individuals psychiatrically diagnosed with major depression are at risk for developing osteoporosis, with depressed young women showing the highest risk" (Biol Psychiatry, 2009; 66: 423–32).

The other meta-analysis came to much the same conclusion. On reviewing data from more than 10,000 people from 14 separate studies, US researchers from the Mayo Clinic in Arizona reported that depression was associated with a significant decrease in BMD of the spine and hip, especially in depressed women and in those with clinical depression. "Depression should be considered as an important risk factor for osteoporosis," the researchers concluded (Osteoporos Int, 2009; 20: 1309–20).

So, is depression the cause of the bone loss or does some other factor explain this novel link?

According to one study, it may not be depression *per se* that's the problem, but the drugs used to treat it. In 5000 people aged 50 years and over, Canadian researchers found that those regularly taking the antidepressants known as 'selective serotonin-reuptake inhibitors', or SSRIs, had a twofold increased risk of bone fractures. SSRI use was also dose-dependently associated with greater odds of falling, and lower BMD of the hip and spine (Arch Intern Med, 2007; 167: 188–94). A review of the scientific literature reached a similar conclusion (Eur Neuro-psychopharmacol, 2009; 19: 683–92).

Nevertheless, there's also evidence that depression itself could be causing bone loss. Hebrew University of Jerusalem researchers have found that depression sets off the sympathetic nervous system—connecting the brain to the internal organs and skeleton—which is primarily aroused by stress. Its activation causes secretion within the bone of 'noradrenaline' ('norepinephrine'), which has a detrimental effect on bone-building cells. The Israeli researchers were able to show that chronic treatment with a drug that blocked noradrenaline in the bone also blocked the effects of depression on bone (Proc Natl Acad Sci USA, 2006; 103: 16876–81; Ann NY Acad Sci, 2010; 1192: 170–5).

Although more studies are needed in this field—dubbed 'neuro-psycho-osteology'—some scientists are already calling for depression to be officially recognized as a risk factor for osteoporosis. Indeed, the findings suggest that dealing with depression may be an important means of keeping bones strong and preventing fractures (see *WDDTY* vol 18 no 12, or www.wddty.com/how-you-beat-depression, for more information on how to treat depression naturally).

Other risk factors

Besides depression, other factors can increase the risk of osteoporosis. Some, such as being female, getting older and having a family history of the condition, can't be changed. But there are other things we can do something about.

◆ **Sedentary lifestyle**. People who spend a lot of time sitting are more likely to develop osteoporosis. As Marilyn Glenville explains in *The Natural Health Bible for Women* (Duncan Baird, 2010), bone strength depends on supply and demand. "If you demand lots from it, it will supply the bone density to accommodate your demands; if you make few demands on it, your bone density will reduce proportionately."

Studies show that regular weight-bearing exercise is important for building and maintaining bone strength. Low-impact activities such as walking and gentle aerobics can prevent bone loss, while high-impact exercise like running and weight-training can increase bone density (Postgrad Med J, 2003; 79: 320–3). Football is a helpful activity. A 14-week study of women, aged 20–47 years, who played football twice a week showed significantly increased bone density in the shins. Surprisingly, similar female runners who trained for the same amount of time didn't see such dramatic effects (Scand J Med Sci Sports, 2010; Mar 4; Epub ahead of print).

For the less mobile, whole-body vibration training (WBVT), which involves standing, sitting or lying on a vibrating platform to stimulate muscle contractions, is a good option. Postmenopausal women having WBVT three times a week for six months saw significant increases in hip BMD, as well as in strength and balance (J Bone Miner Res, 2004; 19: 352–9). WBVT also prevented bone loss in the spine and femur (thigh bone) compared with a placebo (J Bone Miner Res, 2004; 19: 343–51).

If you already have osteoporosis, exercise that improves balance and coordination, such as Tai Chi or step aerobics, can help to prevent falls and fractures (BMC Geriatr, 2006; 6: 6).

◆ **Acidic diet**. While some foods supply us with essential bone-building nutrients such as calcium, others can be detrimental to bone health. Says Annemarie Colbin, author of *The Whole-Food Guide to Strong Bones: A Holistic Approach* (Oakland, CA: New Harbinger Publications, 2009), watch out for acid-forming foods—such as meat and sugar—as excess intakes drain calcium and other minerals from the bones. Studies in mice show that acidosis—a tilt towards an acidic blood pH—encourages bone loss and inhibits bone formation (Curr Opin Nephrol Hypertens, 2004; 13: 423–36). This may explain why older women who eat chocolate (high in sugar) every day have less bone density and strength (Am J Clin Nutr, 2008; 87: 175–80), and why certain drinks are linked to osteoporosis (see below).

The best diet for better bones includes acid-forming and alkalizing foods such as leafy green vegetables (kale, collard and mustard greens, watercress, arugula), roots (carrots, turnips, parsnips, radishes), broccoli and squash (for more information, see *WDDTY* vol 19 no 12).

◆ **Coffee and alcohol**. Caffeine increases urinary loss of calcium and magnesium (J Nutr, 1993; 123: 1611–4), and four or more cups of

coffee a day is linked to a higher risk of fractures, particularly in women with low calcium intakes (Osteoporos Int, 2006; 17: 1055–64). In contrast, tea appears to be bone-protective (Am J Clin Nutr, 2007; 86: 1243–7), as it contains other healthful ingredients such as flavonoids.

Heavy alcohol consumption is also implicated in osteoporosis (Alcohol Clin Exp Res, 2010; Feb 24; Epub ahead of print), but a moderate consumption (1–2 drinks/day) may be beneficial to bone in men and in postmenopausal women (Am J Clin Nutr, 2009; 89: 1188–96).

◆ **Smoking**. Cigarettes are a known risk factor for low BMD and osteoporosis (J Cross Cult Gerontol, 2005; 20: 109–25). Smoking more than a pack a day is associated with a 60-per-cent greater risk of osteoporosis (Bone, 2010; Mar 31; Epublication ahead of print). Also, Japanese researchers have discovered changes such as fewer marrow cells and osteoblasts (bone-making cells) in the bones of smoke-exposed rats. Although these findings may not apply to humans, they suggest that even passive smoking may adversely affect bones (Orthopedics, 2010; 33: 90–5).

◆ **Changes in weight**. Studies have shown that those who have successfully lost weight also had greater bone loss compared with those who didn't lose weight (J Clin Endocrinol Metab, 2007; 92: 3809–15). So, if you're on a weight-loss diet, be sure to increase your physical activity (preferably with high-impact exercise) and ensure that your intake of bone-building nutrients is adequate (see box, page 13).

◆ **Certain medications**. Long-term use of corticosteroid medications such as prednisone and cortisone can lead to osteoporosis (Presse Med, 2006; 35: 1571–7).

Other drugs that have been linked to brittle bones include aromatase inhibitors, used to treat breast cancer (Bratisl Lek Listy, 2010; 111: 27–32), and acid-blocking proton pump inhibitors (Curr Gastroenterol Rep, 2010; Apr 24; Epublication ahead of print).

Prevention is the key

Ultimately, as osteoporosis is a 'silent' condition—it's usually asymptomatic until a bone fracture occurs—understanding what causes it is our best weapon against it. Indeed, research suggests that making simple changes to our diet and lifestyle now can have a big impact on our bones for years to come.

Joanna Evans

Male pattern baldness

'Miracle' drug cures for hair loss come at a price, but natural remedies like vitamin E may work wonders

Medical experts have recently warned that a top-selling anti-baldness drug can cause serious sex-related side-effects and, yet, there's no adequate warning on the label.

Finasteride, sold in the UK and US as Propecia, is used to treat androgenetic alopecia (AGA) in men—or 'male pattern baldness'. It inhibits 5-alpha-reductase, an enzyme that converts testosterone into another male hormone, 'dihydrotestosterone' (DHT), a key cause of hair loss.

Clinical trials show that finasteride can halt hair loss in up to 90 per cent of users, and can even lead to hair regrowth in roughly half of them (Eur J Dermatol, 2002; 12: 38–49). But doctors in the US and in Ireland claim that the drug can cause impotence and other sex-related side-effects, and are calling for more detailed information to be included in the packaging.

Drugs manufacturer Merck, which makes Propecia, acknowledges on its website that certain sex-related side-effects, such as a reduced desire for sex, difficulty in achieving an erection and a decrease in semen, can occur with the drug, although the company says that less than 2 per cent of men experience such side-effects. They also say that, according to their research, these side-effects will go away once the drug is stopped.

However, there are reports of these sex-related side-effects continuing well after men have stopped the treatment. A recent BBC Radio 1 news report featured the case of 26-year-old James, who stopped taking the drug after noticing a loss of libido. He expected the problem to go away, but things only got worse. "After about three weeks . . . I more or less became completely impotent," he said.

James was then put on testosterone therapy, a lifelong commitment. Sadly, that didn't work, so he has now been offered a penile implant.

"Every day, I wish I could turn back the clock," said James. "It did work well for my hair, but the cost is ridiculous—losing my sex life."

Merck claims that such cases are extremely rare and could be caused by something other than finasteride. However, a recent study of the drug noted that "[p]rolonged adverse effects on sexual function such as erectile dysfunction and diminished libido are reported by a subset of men, raising the possibility of a causal relationship" (J Sex Med, 2010 Dec 22; Epub ahead of print).

Also, a safety investigation by the Swedish Medical Products Agency has advised that Propecia may result in irreversible sexual dysfunction. The Agency's updated safety information lists difficulty in obtaining an erection that persists indefinitely, even after stopping the drug, as a possible side-effect. A public assessment report in 2009 by the UK's Medical and Healthcare Products Regulatory Agency (MHRA) also noted persistent erectile dysfunction after stopping treatment with Propecia as a possible undesirable effect.

Other side-effects associated with finasteride include allergic reactions such as rash, itching and hives, swelling of the lips and face; breast tenderness and enlargement; and testicular pain. Finasteride has even been linked to depression and male breast cancer (J Cosmet Dermatol, 2010; 9: 331–2; Drug Safety Update, 2009; 3: 3).

Other hair-loss treatments

Another drug used to treat AGA is minoxidil (Rogaine/Regaine), a topical treatment that has to be applied at least twice a day for four months before any results are seen. However, in addition to possible allergic skin reactions, there's also the risk of systemic effects due to absorption of the drug through the scalp. Such reactions include blurred vision, chest pain, very low blood pressure, fast or irregular heartbeat, headache and weight gain (*WDDTY* vol 16 no 3).

Fortunately, there is a growing number of promising non-pharmaceutical options for dealing with baldness.

Saw palmetto (*Serenoa repens*) can inhibit 5-alpha-reductase levels by 32 per cent with no effect on testosterone in men. Extracts of the plant have also demonstrated an antagonistic effect on testosterone receptors. Both these actions—together with its impressive safety profile—make saw palmetto a useful and safe herbal hair-loss remedy (Cutis, 2004; 73: 107–14).

In 2002, US researchers tested the efficacy of saw palmetto for mild-to-moderate male pattern baldness in a double-blind, placebo-controlled study. Altogether, 26 men were given, twice daily, either a softgel containing 200 mg of saw palmetto extract with 50 mg of beta-sitosterol, a plant sterol found in saw palmetto, or a placebo. The result was that 60 per cent of those taking the active softgel rated their hair growth as improved compared

with only 10 per cent of those taking the placebo (J Altern Complement Med, 2002; 8: 143–52). Beta-sitosterol and saw palmetto are both components used in the commercial hair-loss product HairGenesis (www.hairgenesis.com).

Other herbs may also be beneficial when applied topically. When the 7.5-per-cent herbal hair cream HairPrime (www.hairprime.com) was tested in 24 men with moderate-to-severe AGA, after 40 weeks, the hair counts of those using HairPrime increased by 77 per cent compared with 3 per cent in the placebo group. But it didn't work for everyone; nearly one-third of users saw only a modest response (J Dermatol Treat, 1996; 7: 159–62).

The exact formula of this herbal hair cream is a proprietary secret. However, the *European Handbook of Dermatological Treatment* by A.D. Katsambas and T.M. Lotti (Springer, 2003) states that the product includes extracts of fennel, buckwheat, mint, chamomile, *Thuja* and hibiscus in a water-based cream.

Green tea shows promise as a hair-loss remedy. One Korean review noted that the polyphenol epigallocatechin-3-gallate (EGCG) in green tea might be able to prevent or treat AGA by inhibiting 5-alpha-reductase activity. They reported the results of test-tube studies showing that EGCG can stimulate the growth of human hair (Phytomedicine, 2007; 14: 551–5). However, much more research is needed before any recommendations can be made.

Vitamin E, a potent antioxidant, may also have a role to play in treating male pattern baldness. A study by Malaysia-based company Carotech, presented at the Vitafoods International Conference in Geneva in 2009, revealed that a patented tocotrienol (a form of vitamin E) complex might increase hair growth in people with AGA by 42 per cent.

The eight-month, randomized placebo-controlled trial gave 28 volunteers with AGA either the tocotrienol complex (total tocotrienol, 100 mg) or a placebo (a softgel capsule with 600 mg of soybean oil). Hair coverage, measured by counting the number of hairs in a preselected 2 x 2-cm area, was significantly increased by an average of 41.8 per cent with tocotrienol, eight of whom had more than 50-per-cent new hair growth. The placebo group, on the other hand, saw no significant improvement in hair coverage, and only one volunteer showed a more than 20-per-cent increase in hair count (www.carotech.net/index/news/219.html). These results, however, have yet to be officially published.

Other antioxidants such as beta-carotene, lycopene, lutein, zeaxanthin, zinc and vitamin C may also help hair loss. As oxidative stress could be playing a role in AGA, antioxidants—known to scavenge free radicals—may help (J Cutan Aesthet Surg, 2010; 3: 82–6).

Zinc may be especially useful, as it's an essential cofactor for a number of enzymes and is also involved in important functional activities in the hair follicle (Ann Dermatol, 2009; 21: 142–6). Zinc can also reduce 5-alpha-reductase activity in rat prostate tissue, although such animal results may not apply to humans (Andrologia, 1993; 25: 369–75). Although clinical studies in AGA patients are lacking, one study in men with alopecia areata (patchy hair loss) reported that zinc supplementation had positive therapeutic effects in nine of the 15 patients studied (Ann Dermatol, 2009; 21: 142–6).

Aromatherapy may be worth a try, according to the evidence so far. In a randomized controlled trial of 86 patients with alopecia areata, half of them massaged their scalp daily with essential oils (thyme, rosemary, lavender, and cedarwood in a mixture of carrier oils), while the other half massaged their scalp with only carrier oils, also daily. The researchers found that 44 per cent of the essential-oil group showed improvement compared with only 15 per cent of the control group (Arch Dermatol, 1998; 134: 1349–52).

Another trial, this time in men and women with male pattern baldness and lasting 26 weeks, showed that essential oils used in combination with pulsed electromagnetic field therapy (PEMF; a technology more commonly used for healing bone fractures) can significantly reduce hair loss and improve hair counts compared with a placebo in more than half of cases (Adv Ther, 2003; 20: 220–9).

Low-level light therapy (LLLT) has recently been approved by the US Food and Drug Administration (FDA) for the treatment of hair loss (South Med J, 2010; 103: 917–21). This form of non-invasive therapy uses low-energy laser beams to stimulate hair growth.

Previously, this sort of treatment was only available at hair-loss clinics, but the technology has now been adapted for home use. The HairMax LaserComb is one such home-use device, and has recently been tested in a randomized, sham-device-controlled, double-blind trial of 110 men with

What about hair transplants?

Hair transplantation has come a long way, and the latest techniques are able to achieve very natural-looking results. One such technique is called 'follicular unit extraction' (FUE). This is a sutureless method of hair restoration in which hair follicles are extracted from the back of head under local anaesthesia—using special tools called 'micropunches'—and implanted in the bald areas. This is an extremely labour-intensive method, however, which means it's the most expensive type of hair transplant procedure (J Cutan Aesthet Surg, 2010; 3: 76–81). A typical session can set you back around £7000, and you may require multiple sessions. Although the more traditional hair-transplant techniques are cheaper, you could still end up spending up to £10,000 in total—depending on the extent of your hair loss.

Besides the hefty pricetag, the other drawback with hair transplantation is that it's not risk-free. Common side-effects include pain, swelling and scabbing, while more unusual problems include bleeding and infection.

Yet another aspect to consider is that some techniques can leave obvious scarring where the donor hair has been removed.

Finally, even when the surgical procedure goes perfectly and no scarring is visible, hair transplantation is not a cure for baldness. This means that you may well continue to lose hair around the grafts.

male pattern baldness.

Compared with the group using the sham device, the men who used the HairMax LaserComb saw significant improvements in hair density and overall hair growth (Clin Drug Investig, 2009; 29: 283–92).

However, one downside of this hair-loss treatment is that the device is expensive, costing between £290 and £460, depending on which model you choose (see www.hairmax.com for more information).

Lifestyle changes may have some benefit for hair loss. According to one report, fried foods and red meat should be avoided to reduce the over-all activity of the oil- and sebum-producing glands, which are the sites of 5-alpha-reductase activity, as hyper-active glands can lead to higher levels of conversion to the DHT that causes male pattern baldness.

The report also recommends avoiding sugary foods such as chocolates, sweets and cakes, as well as foods that contain artificial flavourings, additives and preservatives. On the other hand, sprouts, green leafy vegetables, pulses and nuts, along with plenty of water every day, can provide the nutrients required for healthy, glowing hair (J Cutan Aesthet Surg, 2010; 3: 82–6).

Smoking is another lifestyle habit to avoid, as nicotine is known to reduce blood flow to the hair follicles and can also lead to the buildup of damaging free radicals in hair roots. One study found a strong connection between smoking and the incidence of male pattern baldness in men (Arch Dermatol, 2007; 143: 1401–6).

Stem cells are now being studied as a future cure for baldness. When a team of researchers compared bald and hairy patches in scalp samples from men with AGA, they discovered that, although both bald and hairy patches had similar numbers of stem cells, most of the cells in the bald patches had failed to develop to the next stage. Stem cells that had matured into so-called 'progenitor cells' were 10 to 100 times more abundant in the hairy patches than in the bald ones, thus suggesting that they are essential for new hair growth.

Currently, the research team—from the University of Pennsylvania School of Medicine in Philadelphia—is trying to work out why some stem cells become dormant while others remain active, in the hope of developing a new hair-loss treatment. Preliminary studies in mice had suggested that stem-cell transplants might be able to regenerate hair growth (J Clin Invest, 2011 Jan 4, pii: 44478; Epub ahead of print).

Joanna Evans

Chronic back pain

There is now a multitude of effective drug-free alternative treatments for this common, often disabling, condition

More than 80 per cent of us will suffer from disabling low back pain (LBP) at some point in our lives. Although most of us will recover within a few months, some will go on to develop chronic LBP—pain that persists for three months or longer. In most cases, it's not possible to identify a specific cause of the pain, and the treatment usually involves course after course of dangerous drugs (BMC Musculoskelet Disord, 2010; 11: 163).

Happily, there's a vast array of complementary and alternative therapies now available for chronic LBP, many of which are supported by good evidence of success (see box, page 20). But perhaps the best way to tackle LBP is to prevent it from becoming a problem in the first place.

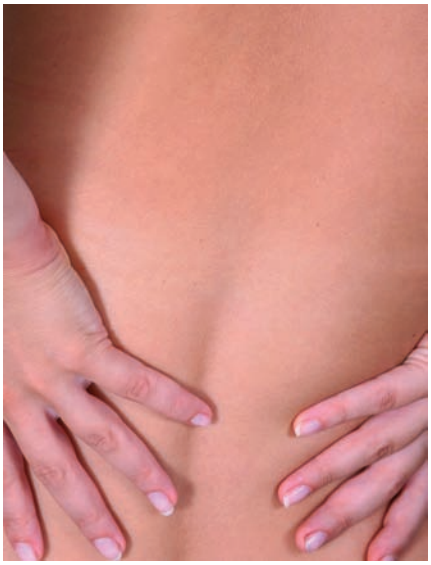
Preventing back pain

According to the latest evidence, there are a number of ways you can reduce your risk of LBP.

- **Stay active.** Lower levels of physical activity are linked to LBP (Aust J Physiother, 2009; 55: 53–8), while regular exercise appears to prevent the condition (Joint Bone Spine, 2008; 75: 533–9). This makes sense as having strong, flexible muscles are essential for a healthy back.

  However, overly strenuous or excessive exercise can be bad for the back (Pain, 2009; 143: 21–5), so be sure to choose the right form of activity for you.

  According to Wisconsin-based spine expert Dr Peter Ullrich, an ideal back workout includes a combination of stretching, strengthening and low-impact aerobic conditioning.
- **Watch your weight**. Being overweight places an additional burden on the spine and strain on the back muscles. In one study of more than 60,000 men and women, a high body mass index (BMI) was significantly associated with an increased prevalence of LBP, particularly in women (Spine [Phila Pa 1976], 2010; 35: 764–8).



- **Stop smoking**. Cigarette-smoking appears to be linked to LBP, according to several studies. In a recent meta-analysis that pooled the results of 40 separate studies, current smokers were 80-per-cent more likely to suffer from chronic LBP and also had more than twice the risk of disabling LBP (Am J Med, 2010; 123: 87.e7–35).

  More worrying, exposure to secondhand smoke during childhood can increase the risk of developing back problems later in life. Researchers have postulated that this might be because tobacco smoke has detrimental effects on the developing spine (Eur J Public Health, 2004; 14: 296–300).
- **Get enough sunshine**. Mounting research suggests that a lack of vitamin D, produced naturally by the body in response to sunlight, could be contributing to chronic musculoskeletal pain, including LBP (BMJ, 2005; 331: 109).

  In one study of patients with chronic, non-specific (no obvious cause) LBP attending spinal and internal medicine clinics in Saudi Arabia for six years, 83 per cent were found to have abnormally low levels of vitamin D. After supplementing with the vitamin, however, symptomatic clinical improvement was seen in all those who had low initial concentrations of the vitamin (Spine [Phila Pa 1976], 2003; 28: 177–9).

  According to Dr Stewart Leavitt, a member of the American Academy of Pain Management and editor-in-chief of the online journal *Pain Treatment Topics*, vitamin D deficiency can cause musculoskeletal pain by causing hypocalcaemia—abnormally low levels of circulating calcium—which "sets in motion a cascade of biochemical reactions negatively affecting bone metabolism and health". One of these reactions is increased parathyroid hormone (PTH), causing a spongy bone matrix to form. This leads to fluid absorption, resulting in an expansive pressure that triggers the abundant pain fibres in the tissues overlying the bones.

  This suggests that anyone who has non-specific LBP should be tested for vitamin D deficiency.
- **Manage stress**. Psychological factors such as stress and depression are also thought to play a role in LBP. In one UK study that followed 4500 adults (aged 18 to 75 years) for 12 months, the likelihood of having a new episode of LBP was greater among those who scored in the upper-third of a questionnaire for psychological distress (Spine [Phila Pa 1976], 1995; 20: 2731–7). In another study, psychological distress was a better predictor of back pain than the standard diagnostic techniques (Spine [Phila Pa 1976], 2004; 29: 1112–7). Psychological factors are also implicated in the transition from acute to chronic LBP (Spine [Phila Pa 1976], 2002; 27: E109–20).
- **Avoid poor posture**. In particular, sitting in a chair for long periods of time creates imbalances in the musculoskeletal system that can increase the risk of pain and injury. According to one study, workers who sit for more than half a day in awkward postures are significantly more likely to suffer from LBP (Eur Spine J, 2007; 16: 283–98). (See *WDDTY* vol 20 no 3 for tips on keeping a healthy back while sitting.)

  Poor lifting technique can also be the cause of back pain. You should always push, rather than pull, when you need to move heavy objects and, if you have to lift, let your legs do the work by holding the

Drug-free treatments for back pain

- **Exercise**. Not just good for preventing back pain, exercise can treat it, too. One review concluded that exercise can reduce pain and improve physical function in chronic or recurrent LBP (Joint Bone Spine, 2008; 75: 533–9). The most effective strategy appears to be an individually designed exercise programme that includes stretching and strengthening, and is carried out under limited supervision—for example, home-based exercises with regular follow-ups by a therapist (Ann Intern Med, 2005; 142: 776–85). However, group exercise can also help, especially if you join a class that focuses on the mind as well as the body.
  - *Yoga*. A two-year study showed that an Iyengar yoga class twice a week can improve functional disability, pain intensity and depression in adults with chronic LBP, and also allowed the use of less pain medication (Spine [Phila Pa 1976], 2009; 34: 2066–76).
  - *Pilates Method*. Increasingly used to treat chronic LBP, this form of exercise can be effective for reducing pain and improving general physical function (J Bodyw Mov Ther, 2008; 12: 364–70).
  - *Qigong*. The main posture used in this form of exercise is similar to the posture recommended by healthcare professionals dealing with back pain (Wien Med Wochenschr, 2004; 154: 564–7). Also, when used as an adjunct to drug treatment, this meditative form of movement and breathing techniques can successfully relieve chronic pain (Am J Chin Med, 2010; 38: 695–703).
- **Acupuncture**. In one major review, acupuncture was found to be superior to the usual care for treating chronic LBP, thereby justifying the recent recognition of this traditional Chinese medicine technique as a therapeutic option for LBP by the UK's National Institute for Health and Clinical Excellence (NICE) (Ann R Coll Surg Engl, 2010 Jun 7; Epub ahead of print).
- **Massage**. Several studies suggest that therapeutic massage is useful against chronic LBP, especially when combined with exercise and patients' self-care education (Spine [Phila Pa 1976], 2009; 34: 1669–84). In one trial, the benefits of massage were still evident 9–10 months after the therapy had ended (Trials, 2009; 10: 96).
- **Alexander Technique**. This discipline, which emphasizes the self-perception of body movement, was found to be more effective than conventional care or massage as a treatment for chronic or recurrent back pain. What's more, just six lessons followed by prescribed exercises were nearly as successful as 24 lessons of Alexander Technique on its own (Br J Sports Med, 2008; 42: 965–8).
- **Spinal manipulation**. Performed by chiropractors as well as some osteopaths and physical therapists, spinal manipulation was recently pitted against back school (consisting of group exercise and education) and individual physiotherapy (exercise, passive mobilization and soft-tissue therapy) for the treatment of chronic LBP. The results showed that spinal manipulation provided better short- and long-term functional improvements, and more pain relief, than either back school or individual physiotherapy (Clin Rehabil, 2010; 24: 26–36).
- **Biofeedback**. This mind–body technique was found to be more effective than behavioural therapy or conservative medical treatment for sufferers of chronic back pain. The researchers also reported that biofeedback was the only method to significantly reduce pain over the two-year follow-up (J Consult Clin Psychol, 1993; 61: 653–8).
- **Hypnotherapy**. Numerous studies show that hypnosis is an effective treatment for a range of chronic pain conditions. In a small preliminary study to assess its success in chronic LBP, a brief, four-session standardized self-hypnosis protocol, combined with psychoeducation, dramatically reduced pain intensity and pain interference (Int J Clin Exp Hypn, 2010; 58: 53–68).
- **Supplements**. In addition to a balanced diet, a number of supplements may be useful for beating back pain.
  - *Vitamin D* could be the key to curing LBP if you are deficient in this nutrient (see main story). In a study of 360 patients with chronic LBP, vitamin D eased symptoms in virtually all those with the most severe D deficiency (Spine [Phila Pa 1976], 2003; 28: 177–9). Although sunshine is our best source of this vitamin, most of us don't get enough of it that way. Pain expert Stewart Leavitt recommends (with the supervision of a qualified practitioner) a daily supplement of 2000 IU of vitamin D3 (cholecalciferol), along with a daily multivitamin that includes calcium and 400–800 IU of vitamin D. Be patient, as it may take up to nine months to experience the maximum effects of this regimen.
  - *B-complex vitamins* may also help. A combination of vitamins B1, B6 and B12, taken twice a day at 50 mg, 50 mg and 1 mg, respectively, together with the popular non-steroidal anti-inflammatory drug (NSAID) diclofenac (50 mg twice daily), was better at relieving back pain than the NSAID alone (Curr Med Res Opin, 2009; 25: 2589–99).
  - *Proteolytic enzymes,* such as trypsin and serrapeptase, may be useful as they are known to have anti-inflammatory properties (Indian J Pharm Sci, 2008; 70: 114–7).
- **Herbs**. The following may help to relieve back pain.
  - *Capsaicin*, found in all hot peppers, can ease many types of chronic pain when applied regularly to the skin. In one study, a capsaicin plaster was significantly better than a placebo in patients with chronic back pain (Arzneimittelforschung, 2001; 51: 896–903).
  - *Devil's claw* (*Harpagophytum procumbens*) is effective for LBP when the daily dose provides at least 50 mg of the active ingredient harpagoside. One trial found it to be just as effective as the NSAID rofecoxib (Spine [Phila Pa 1976], 2007; 32: 82–92).
  - *White willow bark* (*Salix alba*) is chemically related to aspirin and appears to provide short-term relief of LBP. Studies used daily doses standardized to 120 mg or 240 mg of salicin, which has anti-inflammatory actions (Spine [Phila Pa 1976], 2007; 32: 82–92).

load close to your body, keeping your back straight and bending only at the knees. Avoid lifting and twisting simultaneously.

- **Treat childhood back pain**. Contrary to popular belief, non-specific LBP is a serious problem among children and teenagers. Indeed, a review of the relevant studies suggests that rates are almost as high as in adults (Ugeskr Laeger, 2002; 164: 755–8).

There are also clear correlations between experiencing LBP as a child/adolescent and suffering from LBP—especially chronic LBP—as an adult (Arch Pediatr Adolesc Med, 2009; 163: 65–71). It is vital, therefore, that the condition, when it arises, be dealt with promptly.

The possible causes of childhood LBP include intensive sports activities and carrying a too-heavy backpack, as well as the factors mentioned above (Rev Chir Orthop Reparatrice Appar Mot, 2004; 90: 207–14). For backpack safety tips, see http://orthoinfo.aaos.org/topic.cfm?topic= A00043.

Joanna Evans

Stroke

Anyone can suffer a stroke—not just the elderly. But some little-known health tips, like keeping your gums healthy, can help slash your risk

Stroke is a dreaded disorder that affects some 150,000 people in the UK each year. It occurs when blood circulation to the brain fails due to either blockage (ischaemic stroke) or bleeding (haemorrhagic stroke), causing brain cells to die, with symptoms such as paralysis, speech difficulties and vision problems.

According to the Stroke Association, around 1000 people aged under 30 suffer a stroke every year. Often, it's secondary to other health issues such as diabetes and heart conditions. The good news is that there are a number of ways to reduce the risk.

Stroke prevention

For years, millions of people worldwide, including the 'worried well', have been taking aspirin to protect against heart attack and stroke. Yet, the latest evidence is that this 'wonder drug' may be doing more harm than good. Not only can aspirin cause serious and fatal gastrointestinal injury and bleeding (Drug Ther Bull, 2009; 47: 122–5), but it can also increase stroke risk, especially in the over-75s. Over the past 25 years, the number of strokes associated with such blood-thinners has increased sevenfold (Lancet Neurol, 2007; 6: 487–93).

Happily, there are safer ways to cut your risk of suffering a stroke.

- **Eat plenty of fruit and vegetables**. Having more than the recommended five servings of fruit and veg per day (one serving = half a cup) can reduce the risk of stroke by 26 per cent (Lancet, 2006; 367: 320–6).
- **Take regular exercise**. Inactivity can almost double your risk of stroke (Am J Epidemiol, 1996; 143: 860–9), while vigorous exercise early in life protects against later stroke, despite other risk factors such as social class, smoking, alcohol intake, family history, hypertension and diet. Continued vigorous exercise later in life reduces the risk even further. Of more than 11,000 men, aged 58 years on average, those who exercised moderately (one hour of brisk walking, five days a week) had a 46-per-cent lower stroke risk than those who exercised little or not at all (Stroke, 1998; 29: 2049–54).
- **Maintain a healthy weight**. Obesity is a risk factor for stroke, and also contributes to other risks such as high blood pressure, diabetes, high cholesterol and obstructive sleep apnoea (where the sufferer stops breathing temporarily throughout the night). As well as keeping an eye on your BMI (body mass index), it is also worth watching your waistline, as this has been independently linked to stroke (Arch Intern Med, 2007; 167: 1420–7; Stroke, 2003; 34: 1586–92).
- **Limit alcohol**. Moderate alcohol consumption—no more than two drinks per day—can protect against ischaemic stroke, while heavy drinking has the opposite effect (JAMA, 1999; 281: 53–60).
- **Eat more wholegrains**. Compared with eating no wholegrains, women who ate more than one wholegrain food a day reduced their stroke risk by around 35 per cent (JAMA, 2000; 284: 1534–40).
- **Increase your intake of 'good' fats**. Fish, rich in omega-3 fatty acids, can prevent stroke (Prev Med, 1999; 28: 520–9; JAMA, 2002; 288: 3130–6) but, if you're worried about mercury, just eat a few walnuts a day. These, along with flaxseed, soybean and canola oils, are an excellent source of essential omega-6 alpha-linolenic acid (ALA), high blood levels of which reduced stroke risk even in men at high risk of cardiovascular disease (Stroke, 1995; 26: 778–82).
- **Take supplements**. B vitamins can help to reduce homocysteine, which has been linked to stroke (Stroke, 2002; 33: 2351–6).

Supplementing with even low levels of B vitamins (1 mg of folic acid (B9), 10 mg of B6 and 400 mcg of B12) significantly lowered homocysteine levels within six weeks (Am J Clin Nutr, 1993; 57: 47–53). In another study, folic acid reduced the risk of stroke by 18 per cent (Lancet, 2007; 369: 1876–82), while combining beta-carotene, selenium, vitamins E and A, and zinc reduced stroke deaths by 29 per cent (Ann Intern Med, 2006; 145: 372–85).

- **Try herbs**. Garlic and *Ginkgo biloba* are powerful blood-thinners (Arzneim Forsch, 1993; 43: 119–22; Haemostasis, 1989; 19: 219–23), and ginger can also help: 5 g/day of raw ginger for just one week led to a 37-per-cent drop in the blood-clotting agent thromboxane (Prostaglandins Leukot Essent Fatty Acids, 1989; 35: 183–5).
- **De-stress**. Stress is a major contributor to hypertensive disease and stroke. *Qi gong*, an ancient Chinese form of exercise that emphasizes a tranquil mind, relaxed body and smooth breathing, lowers blood pressure (Zhongguo Zhong Xi Yi Jie He Za Zhi, 1993; 13: 413–4, 388–9) and slashes stroke risk. In one long-term study, only 19 per cent of those who regularly practised *Qi gong* died of stroke compared with 42 per cent of the controls (J Tradit Chin Med, 1986; 6: 235–8). Meditation also appears to be beneficial (Stroke, 2000; 31: 568–73).
- **Keep your gums healthy**. Those with severe gum disease have a two-fold higher risk of stroke (Arch Intern Med, 2000; 160: 2749–55).
- **Get enough sunshine**. Vitamin D

deficiency may play a role in heart disease and stroke (Curr Vasc Pharmacol, 2009; 7: 414–22). Exposing your skin to sunlight for 10–15 minutes a day (with no sunscreen) is the best way to get your dose of vitamin D (BMJ, 2003; 327: 1228–9). Alternatively, supplementing with 600–1000 IU of vitamin D3 (the natural form) is a good idea for those who don't get much sun.

- **Look after your lungs**. The health of your lungs appears to be a good indicator of the risk of stroke (Stroke, 2009; 40: 1986–90). In fact, smoking, passive smoking and outdoor air pollution have all been linked to stroke (Tob Induc Dis, 2004; 2: 7; Stroke, 2003; 34: 2776–80). In addition to not smoking, there are a number of ways to protect your lungs from harmful pollutants:
  - ❖ **install ionizers in your home and car**, as they emit negatively charged particles—or 'negative ions'—into the air; these attach to pollutants, causing them to 'drop out' of the atmosphere;
  - ❖ **take antioxidants**, such as vitamins A, C and E, which scavenge free radicals; and
  - ❖ **keep tabs on the levels of pollution in your area** by contacting your local air-quality monitoring service. (Readers in the UK can visit the UK National Air Quality Archive at www.airquality. co.uk, while those in the US can check out the US Environmental Protection Agency website at www.airnow.gov.) When levels are high, don't exercise or exert yourself outdoors, as the faster you breathe, the more pollution you will then draw into your lungs.

Joanna Evans

Promising post-stroke therapies

- **Music therapy**. Listening to music for a few hours every day can boost cognitive and emotional recovery in the early stages following a stroke. Finnish researchers compared the recovery of 60 stroke patients who listened daily to either music of their choice, audio books or nothing at all (controls) for two months. At this time, all patients also received the standard medical care and rehabilitation. The results showed that the recovery of verbal memory and focused attention (ability to control and perform mental operations, and resolve conflicts) improved significantly more with music than with audio books and nothing at all. The music-listeners also felt less depressed and confused than the control group, and these differences were still evident six months later (Brain, 2008; 131: 866–76).
- **Guided imagery, or visualization**. Now used as part of stroke rehabilitation, this appears to be especially effective for helping patients to relearn their motor skills and how to perform everyday tasks (Brain Inj, 2004; 18: 1163–72; Stroke, 2006; 37: 1941–52).
- **Transcranial magnetic stimulation (TMS).** This non-invasive brain-stimulation technique appears to promote functional recovery in stroke patients. In a randomized controlled trial of 52 stroke patients who received either TMS or sham (control) brain stimulation in addition to the usual therapy, the TMS group showed greater improvement, as assessed by three disability scales (Neurology, 2005; 65: 466–8).
- **Vitamin E**. Research in mice has revealed that a form of vitamin E called 'tocotrienol', found in supplements, may be able to prevent brain cells from dying after a stroke. Tocotrienol appears to block the activity of a brain-damaging enzyme that is produced following a stroke—even when taken at very low levels (J Neurochem, 2010; 112: 1249–60). These results, however, may not necessarily apply to humans.
- **Biofeedback**—getting back information on bodily functioning to learn how to actively control these functions—has been applied to many aspects of stroke rehabilitation, but the results are mixed. A review of 13 trials, involving only 269 patients, found no treatment benefits with electromyographic biofeedback (Cochrane Database Syst Rev, 2007; 2: CD004585). Nevertheless, researchers have concluded that "biofeedback in general can have a very positive impact" by boosting patients' self-confidence (Top Stroke Rehabil, 2007; 14: 59–66).
- **Traditional Chinese medicine**. A preparation of 14 herbal and plant extracts known as *Danqi Jiaonang*, or NeuroAid, taken as capsules, is currently being studied as a therapy for stroke. In a recent clinical trial, although the results did not reach statistical significance, the NeuroAid-treated post-stroke patients showed improvement over controls after 4 weeks of treatment (Cerebrovasc Dis, 2009; 28: 514–21); a longer and larger multicentre study of the remedy is currently ongoing (Int J Stroke, 2009; 4: 54–60).
- **Acupuncture** is another traditional Chinese technique that's proving useful. The method known as 'Jin three-needle therapy' appears to be especially effective at improving cognitive function and in helping post-stroke patients to get along in their day-to-day life (Zhongguo Zhen Jiu, 2009; 29: 689–94).

Beating the change

We now know that the risks of HRT far outweigh the benefits, so what alternatives are there to help women through the menopause?

Now that hormone replacement therapy (HRT) has been largely discredited, millions of women embarking on the menopause are turning to alternative medicine to manage their symptoms. In fact, only 10–25 per cent of women experiencing menopausal symptoms seek treatment from a traditional healthcare provider, according to a recent report, and many of them are dissatisfied with the conventional medical recommendations (J Womens Health [Larchmt], 2005; 14: 634–49).

However, finding a safe and effective alternative treatment is no easy task. One of the favourites is so-called 'natural' progesterone, championed by the late Dr John Lee. But, as *WDDTY* reported in 2006, it is far from natural. There are even some concerns that it might cause cancer, just like HRT (see *WDDTY* vol 17 no 2). Similar concerns have been raised about soy and isoflavone supplements (see *WDDTY* vol 16 no 8), which are also among the more popular alternative remedies for menopause. So what's a girl to do?

Happily, research shows that there are a number of simple, safe steps you can take to help make the transition into menopause a smooth one.

- **Exercise regularly**. Growing evidence suggests that being physically active is one of the best things you can do to keep menopausal symptoms under control. A recent study of 336 menopausal women found that those who were the most physically active had less severe symptoms and generally felt better than their less-active counterparts (Climacteric, 2010; 13: 355–61).

In another study, 36 postmenopausal women were split into two groups: an aerobic-exercise group and a resistance-exercise group. Both groups exercised three times a week for eight weeks under the supervision of a physiotherapist. At the end of the study, both types of exercise were found to have positive effects on the symptoms of menopause, although aerobic exercise had a slightly greater impact. In addition, both exercise groups saw improvements in their psychological health, depression and quality of life (Obstet Gynecol Int, 2010; 2010. pii: 274261).

Regular exercise can also reduce the risk of postmenopausal breast cancer, osteoporosis and cardiovascular disease—an important consideration, as the risk of developing these diseases increases after the menopause (Arch Intern Med, 2010; 170: 1758–64; Aust J Public Health, 1993; 17: 23–6). Aim for at least 30 minutes of moderate-intensity physical activity on most days.

- **Boost your intake of omega-3 fats**. A new study has found that omega-3 fatty-acid supplements can help fight depression and hot flushes in women going through the menopause. The study involved 20 women with major depressive disorder given 2 g/day of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) for eight weeks. The results showed that 70 per cent of the women saw a reduction of at least 50 per cent in their Montgomery–Asberg Depression Rating Scale (MADRS) scores. Moreover, hot flushes decreased in both number and severity (Menopause, 2010 Oct 27; Epub ahead of print).

In addition, like exercise, omega-3 supplements can also help to prevent postmenopausal osteoporosis, breast cancer and heart disease (Obstet Gynecol Surv, 2004; 59: 722–30).

- **Take vitamin E**. A randomized controlled trial found that daily doses of vitamin E (400 IU) significantly reduced the frequency and severity of hot flushes in postmenopausal women (Gynecol Obstet Invest, 2007; 64: 204–70). Previous research carried out more than 50 years ago also suggests that vitamin E may help mood swings and vaginal dryness (NY State Med; 1952; 52: 1289).
- **Consider aromatherapy**. This form of treatment uses essential oils extracted from the flowers, leaves, fruit, bark and roots of medicinal plants. Aromatherapy

Signs of menopause

Officially, you're not in the menopause until it's been a whole year since your last menstrual period. However, the signs and symptoms of menopause may well appear long before that. These include:

- hot flushes
- night sweats
- irritability
- mood swings
- depression
- insomnia
- impaired memory or concentration
- loss of libido
- vaginal dryness
- increased abdominal fat.

massage works on the nervous system through the senses of both smell and touch.

In a small study carried out in Japan, 15 women with menopausal symptoms were examined by a gynaecologist before receiving a 30-minute aromatherapy session, involving a consultation, massage and home-massage guidance. After one month of at-home care and a second aromatherapy session, all reported significant improvements in their symptoms (J Altern Complement Med, 2005; 11: 491–4).

Intriguingly, another study discovered that aromatherapy massage, with an additional focus on the abdomen, can reduce abdominal fat and improve body image in post-menopausal women (Taehan Kanho Hakhoe Chi, 2007; 37: 603–12).

- **Take up yoga**. A systematic review of 18 clinical trials from six countries found that yoga-based and certain other mind–body therapies can be beneficial by alleviating a range of menopausal symptoms. Eight out of nine studies of yoga, tai chi and meditation-based programmes reported improvement in overall menopausal and vasomotor symptoms (such as hot flushes and night sweats); six of seven trials using yoga-based programmes indicated improvement in mood and sleep, and four studies reported reduced musculoskeletal pain.

  Results from the other trials suggest that breath-control-based and other relaxation therapies are also promising for relieving menopausal symptoms (Maturitas, 2010; 66: 135–49).
- **Consider acupuncture**. One small Swiss study showed that acupuncture was able to significantly reduce vasomotor symptoms and physical disturbances in menopausal women. What's more, the effects were still evident three months after the last session (J Altern Complement Med, 2001; 7: 651–8).

  More recently, a randomized controlled trial of 175 women reported that acupuncture, in addition to the usual care, was associated with marked clinical improvement in hot flushes and other menopause-related symptoms (Menopause, 2010; 17: 269–80).
- **Try the herbal supplement Femal**. This "unique blend of pollen extracts from seven organically cultivated flowers grown in . . . Sweden" also contains vitamin E. In one Danish study, 54 menopausal women were randomly given either two Femal tablets each morning, or two identical placebo tablets, for three months. The results showed that 65 per cent of the Femal group enjoyed a reduction in hot flushes compared with 38 per cent in the placebo group.

  On looking at 15 other menopausal "quality-of-life" symptoms, Femal again came out on top (Climacteric, 2005; 8: 162–70).

  Femal is available from Boots and independent chemists.
- **Lose weight**. If you're overweight or obese, losing weight can help to keep hot flushes under control, according to a recent study. University of California at San Francisco researchers studied 338 overweight or obese women and found that those who followed an intensive weight-loss programme showed greater improvements in bothersome hot flushes compared with those following a structured health-education programme (controls) (Arch Intern Med, 2010; 170: 1161–7).
- **Stop smoking**. The results of one study suggest that women who suffer from hot flushes are more likely to be smokers (J Womens Health, 1998; 7: 1149–55). Another study found that women taking HRT for relief of menopausal symptoms were more likely to be current smokers.

  "This relation between HRT use and smoking could result from an anti-oestrogen effect of smoking, intensifying menopausal symptoms," said researchers (J Epidemiol Community Health, 1987; 41: 26–9).
- **Investigate allergies**. According to Dr Ellen Grant, author of *The Bitter Pill and Sexual Chemistry*, hot flushes could be due to an allergic reaction to common foods or chemicals. In her study of 60 migraine sufferers, a variety of foods and drinks, including wheat, eggs, sugar, tea and coffee, commonly caused vascular reactions such as flushing, headaches, and pulse or blood pressure increases. The most common chemical allergens were cigarette smoke and domestic gas (Lancet, 1979; 313: 966–9; *WDDTY* vol 17 no 2). So, if you suffer from hot flushes, keep a diary to try to pinpoint any possible triggers.
- **Consider black cohosh**. This herb has a long history of traditional use for a variety of 'female complaints', including menstrual problems and childbirth. Scientific studies have found it to be a safe and effective remedy for a number of menopausal symptoms, but mainly hot flushes and mood swings. Indeed, Germany's governmental regulatory agency Commission E has approved the use of 40 mg/day of black cohosh (sold as Remifemin) for six months for the relief of such symptoms.

  Although early studies suggested that the herb has oestrogenic effects, more recent evidence shows that black cohosh does not have an oestrogenic mechanism of action but, instead, acts on serotonin receptors to relieve menopausal symptoms (J Womens Health [Larchmt], 2005; 14: 634–49).

  Nevertheless, there have been concerns (from animal studies, which may not apply to humans) as to its potential to increase the spread breast cancer, so it's generally not recommended for patients or women with a family history of the disease (Cancer Res, 2008; 68: 8377–83).

  If you do wish to try black cohosh, it's best to do so only under the supervision of a registered naturopath or medical herbalist.
- **Give *Ginkgo* a go**. Several studies show that *Ginkgo biloba* can help to boost memory and concentration, which can suffer during the menopause. One study found that 120 mg/day for just one week

significantly improved memory and attention compared with a placebo in postmenopausal women (Pharmacol Biochem Behav, 2003; 75: 711–20).

When combined with other herbs, *Ginkgo* may also boost the libido. A herbal formulation (Herbal vX) of *Ginkgo* and *Muira puama* was tested in 202 healthy pre- and postmenopausal women complaining of low sex drive. After one month of treatment, statistically significant improvement was found in frequency of sexual desire, sexual intercourse and sexual fantasies, as well as in satisfaction with sex life in general, intensity of sexual desires, excitement of fantasies, ability to reach orgasm and intensity of orgasm (Adv Ther, 2000; 17: 255–62). However, as the trial was uncontrolled, we don't know to what extent the results are down to the 'placebo effect'.

In one double-blind, placebo-controlled trial, the supplement ArginMax (from www.arginmax.com and www.virilityhealth.co.uk), which contains *Ginkgo,* plus L-arginine, ginseng, damiana, multivitamins and minerals, was tested in just over 100 women, aged 22–73 years, who reported a lack of sexual desire.

In those going through the menopause, the supplement increased the frequency of intercourse, satisfaction with the sexual relationship and vaginal dryness.

In postmenopausal women, half reported increased sexual desire compared with only 8 per cent with the placebo. Moreover, the formulation has been shown to exhibit no oestrogenic activity (J Sex Marital Ther, 2006; 32: 369–78).

- **Eat healthily**. Keeping to a balanced diet that includes a variety of fruit and vegetables can protect against bone loss, cancer and premature death (Am J Clin Nutr, 2000; 71: 142–51; Cancer Epidemiol Biomarkers Prev, 2004; 13: 1485–94; JAMA, 2000; 283: 2109–15).

The nutrients that are especially important for menopausal women are calcium, vitamins D, C and the Bs, and potassium, zinc, copper and magnesium. Aim to get these from your diet, and supplement when necessary.

Joanna Evans

Herbal helpers for women over 50

Here are some of the helpful herbs that women can take as they go through the menopause and after

As women grow older and reach the menopause and beyond, the inevitable changes that occur in their bodies due to age and hormones may require lifestyle changes, including a new look at nutritional requirements. The following herbs have all been proven to help.

Black cohosh (Actaea racemosa)

This is good for menopausal symptoms, but needs a prescription from a registered naturopath or medical herbalist (McKenna DJ *et al. Botanical Medicines: The Desk Reference for Major Herbal Supplements, 2nd edn*. London, Oxford & NY: The Haworth Press, 2002: 41–3). The herb can also help with postoperative functional deficits following ovariectomy or a full hysterectomy.

Contraindications: Pregnancy; lactation; oestrogen-dependent tumours, including some breast cancers due to its potential oestrogenic actions; and salicylate hypersensitivity, as this is a salicylate-containing plant.

Drug interactions: Hormone replacement therapy (HRT), as this herb may lead to oestrogen excess.

In studies using a fluid extract of this herb, up to 890 mg/day has been given—with no toxic effects (Brinker F. *Herb Contraindications & Drug Interactions, 3rd edn*. Sandy, OR: Eclectic Medical Publications, 2001: 40–1; also www.eclecticherb. com/emp), although the usual dose of an ethanolic extract is only 8 mg/day.

Evening primrose oil (EPO)

Postmenopausal women often suffer from dry skin (eczema-like), hair loss, breast pain and poor wound-healing due to depleted oestrogen. However, EPO (*Oenothera biennis*) can help to reverse these conditions (McKenna op.cit., 327–35).

Contraindications: Epilepsy; and mania.

Drug interactions: Phenothiazines: induces seizures and hallucinations; tamoxifen: speeds up clinical response; cyclosporine: less kidney damage; and anticoagulants: may enhance blood-thinning effects.

The highest dose generally recommended is 0.1 mL/kg/day, so someone weighing 55 kg (8.5 stone) should take 5.5 mL/day (Brinker op. cit., 92–3; see also www.eclecticherb.com/emp).

Garlic (Allium sativum)

It is well established that garlic lowers blood pressure, blood glucose, blood lipids and cholesterol, but it also prevents blood clots, and is mildly antibiotic, antifungal, anticancer, antioxidant, immunomodulatory and liver-protective (McKenna op. cit., pp 375–96).

Contraindications: Gastritis; early pregnancy; hypothyroidism; and oesophageal reflux.

Drug interactions: Warfarin or other blood-thinners: because it enhances anticoagulation; indomethacin; dypiridamole; paracetamol/ acetaminophen: it may prevent liver toxicity; and insulin (as it lowers blood glucose, insulin doses may need adjusting).

A dose of 4 g/day of fresh garlic, or 8 mg of essential oil of garlic as support therapy, is typically prescribed to reduce blood lipids, and to prevent age-dependent vascular changes (Brinker, op. cit., pp 99–101).

Ginkgo biloba (maidenhair tree)

Ginkgo helps to prevent memory loss, loss of mental alertness, dementia, stroke, free-radical damage in traumatic brain injury

and Alzheimer's (Arch Phys Med Rehabil, 2000; 81: 668–78).

Contraindications: Bleeding disorders, especially those with reduced blood-clotting, so it may contribute to haemorrhage if used for a lengthy period prior to surgery.

Drug interactions: Aspirin (when used chronically to prevent blood clots), anticoagulants, heparin, other non-steroidal anti-inflammatory drugs (NSAIDs), ticlopidine, thiazide, trazodone, cyclosporine and papaverine; it may enhance meclofenoxate, used to treat senile dementia and Alzheimer's, and the effects of monoamine oxidase inhibitors (MAOIs), a class of antidepressant drugs.

In a study of 33 women taking an average dose of 209 mg/day, *Ginkgo* leaf extract counteracted the sexual dysfunction associated with antidepressants, mostly SSRIs (selective serotonin reuptake inhibitors), but also phenelzine (an MAOI), vivactil (protriptyline, a tricyclic), and venlafaxine, nefazodone and bupropion (serotonin–norepinephrine reuptake inhibitors, SNRIs) .

It is also thought that *Ginkgo* may reduce the efficacy of anticonvulsants such as carbamazepine (Brinker op. cit., pp 103–7).

Ginkgo is usually given as a total daily dose of 120–240 mg of the standardized extract [containing at least 24-per-cent ginkgo flavone

glycosides and 6-per-cent terpene lactones (ginkgolides and bilobalides)] for at least eight weeks to treat chronic conditions, and a review of its benefits is typically carried out before it is taken for more than three months.

Arnica montana (wolfsbane)

Never use *Arnica* internally unless it's dispensed in a homeopathic potency, as it has toxic effects on the liver and kidneys. It can also cause gastrointestinal irritation if taken undiluted (Brinker F. *Herb Contraindications & Drug Interactions, 3rd edn*. Sandy, OR: Eclectic Medical Publications, 2001: 31–2; see also www.eclecticherb. com/emp). The *Arnica* tablets sold over-the-counter (OTC) at many pharmacies come from manufacturing pharmacies such as Nelsons or Weleda, and have undergone processes to have such toxicity removed, so they can safely be taken as advised by the packaging, without the guidance of a homeopath. There should be no side-effects or contraindications.

Arnica tablets can reduce bruises and contusions, as can suitably diluted *Arnica* creams [Schilcher H, Kammerer S. *Leitfaden Phytotherapie, 3 aufl (Guide to Phytotherapy, 3rd edn)*. Munich, Jena: Urban & Fischer Verlag, 2010: 30–2]. Indeed, virtually every cosmetic and plastic surgeon in London's Harley Street recommends taking *Arnica* before and after surgery by their middle-aged patients to minimize the severe bruising that is inevitable after such surgery.

Hawthorn (Crataegus oxyacantha)

With the reduced oestrogen in postmenopausal women comes an increased risk of heart problems. Women aged over 50 then suddenly run the same risks of atherosclerosis, hypertension and congestive heart failure as do men.

However, hawthorn has proved to have numerous beneficial actions on the heart and cardiovascular system. It can regulate heart rhythm through its anti-arrhythmia actions, and dilate the coronary arteries, thereby reducing the chances of heart attack. It can also increase the blood supply to the heart. Furthermore, hawthorn can lower systolic pressure (the first number in a blood pressure reading), which is especially beneficial in people over 50, who have the highest risk of isolated systolic hypertension (ISH). If left untreated, ISH can lead to heart disease, stroke and dementia. The herb also improves low blood pressure, which can lead to dangerous sudden fainting episodes (McKenna DJ *et al. Botanical Medicines: The Desk Reference for Major Herbal Supplements, 2nd edn*. London, Oxford & NY: The Haworth Press, 2002: 659–70). However, those taking beta-blockers for blood pressure should know that hawthorn may cause a slight rise in diastolic blood pressure (the second number in a blood pressure reading).

Hawthorn used with garlic powder protects the heart, liver and pancreas against damage from isoprenaline, a bronchodilator known to have a powerful stimulant effect on the heart and cardiovascular system. Taken with adenylic acid, the herb enhances the action of digoxin, prescribed for cardiac and coronary insufficiencies. When used as an alcoholic extract (4:1 ratio), it inhibits the arterial contractions induced by phenylephrine (Brinker, op. cit., pp 118).

St John's wort (Hypericum perforatum)

This herb has been in continuous recorded use as a natural medicine since the days before Hippocrates (ca. 460–ca. 370 BC).

Although best known nowadays for its antidepressant effects, it's also known to promote wound-healing, and can inhibit protein kinase C, which has the beneficial effect of preventing the usual diabetic vascular complications of nephropathy, retinopathy and neuropathy. In addition, St John's wort has antiviral activity, coronary vasodilatory effects, painkilling (analgesic) and liver-protective effects, and can help against sleep disturbances by boosting melatonin synthesis (McKenna, op. cit., pp 923–71).

However, this herb should not be used in pregnancy, prior to sun exposure or solarium therapy, or ultraviolet light therapy, or in those with endogenous (genetic) depression—especially in those cases with psychotic symptoms or high suicidal risk.

St John's wort can have interactions with nefazodone, sertraline, trazodone, fluoxetine, paroxetine, indinavir, warfarin, phenoprocoumon, reserpine, protease inhibitors, theophylline (requires a 250-per-cent increase in the daily dose of St John's wort to maintain effectiveness), monoamine oxidase inhibitors (MAOIs), midazolam and naloxone, as well as with photo-sensitizers (causing light sensitivity) such as piroxicam and tetracycline. Prior to surgery, it may interact adversely with anaesthetics (so the anaesthetist should always be informed when St John's wort has been taken) (Brinker, op. cit., pp 178–84).

For mild-to-moderate depression, therapeutic doses of the herb extract are 300–900 mg/day, and up to 1800 mg/day for more severely depressed patients. The extract should be standardized at 0.3-per-cent hypericin.

Harald Gaier

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Hair loss

As oestrogen levels fall during the menopause, you may have increasingly regular bad-hair days. Here's how to keep your locks full and healthy.

Hair problems of every variety are more common during the menopause, and can worsen postmenopausally. Although the change may be imperceptible to others, when running your hands through your hair, you may find that it feels thinner all over. Alternatively, your hair may look noticeably thinner at the front. Other menopausal hair problems include dullness, dryness, split ends, poor hair growth and dandruff. You may even find that your body hair, including pubic hair, is thinner or completely gone, while some women will find unwanted hair growing on the face.

Causes of hair changes

As you go through the menopause, levels of oestrogen in your body fall, such that your body 'believes' that it has more male hormones (such as the androgen testosterone) in circulation. What has happened is that counter-balancing oestrogen is no longer present. As androgens dominate your system, you may experience masculine symptoms such as male-pattern bald-ness, acne and increased facial hair (particularly on the upper lip).

You may even be genetically predisposed to hair loss. In 'androgenic alopecia', a tendency towards hormonal imbalance is inherited, and shows up particularly in later life. According to research, up to 13 per cent of women have this sort of hair loss prior to the menopause. After the menopause, the condition is even more common, with one study showing that as much as 75 per cent of women over the age of 65 are affected.

If you notice a lot of hair on your pillow in the morning or your hairdresser mentions that your hair seems thinner, you should see a doctor for a check-up. Although it might simply be related to the menopause, other medical conditions can result in hair loss, including anaemia (low iron) and thyroid problems. Stress, too, can cause hair loss—at any age.

Conventional treatments

If you've entered the menopause and your hair is thinning, your doctor is likely to offer one of these options:

- **Hormone replacement therapy (HRT).** Most women find the thought of going bald terrifying. Some have told me that they've considered taking HRT just to stop hair loss in its tracks. However, although HRT raises your levels of oestrogen, it doesn't always solve the hair-loss problem and, ironically, one of the listed side-effects of HRT itself is hair loss. Unfortunately, there's no way to predict how your body will respond until you try it.
- **Minoxidil**. First developed for high blood pressure, this vasodilator can also thicken the hair. It's taken as either a pill or as a lotion applied directly to the hair and scalp. As with any drug, there are side-effects, the most common one of which is an itchy scalp. Other side-effects include acne, headache, very low blood pressure, irregular heartbeat, chest pain and blurred vision. Most important, however, is that this medication does not address the cause of the problem, so as soon as you stop using it, your hair loss will resume.
- **Spironolactone.** This synthetic corticosteroid has mild diuretic properties, and acts by interfering with the binding of male hormones in the hair follicles to prevent hair loss. However, it has been linked with an increased risk of stomach bleeding, as well as rashes, irregular periods and drowsiness.

Your diet

I think of hair as a barometer of overall health (along with the skin and nails). If you consider a cat or dog, when such an animal is unwell, its glossy, sleek fur coat becomes dull, limp, lifeless and thin. This offers a clue as to how you might be able to slow the deterioration of your hair—namely, by looking after your entire state of health by eating well, and making sure to have your full quota of essential vitamins and minerals.

As well as making the choice to eat healthy foods, make sure you eat regularly throughout the day, including mid-morning and mid-afternoon

Four-step scalp massage

Massage your scalp, using the technique below, for a few minutes every day, twice a day. You can also use this technique when you want to massage essential oils into your scalp (see box, page 30).

1. Using your fingertips, make small circular movements all along your hairline, beginning in the middle of the forehead, then working down the sides, around the ears and over to the back.
2. Using the tips of the thumbs and fingers of both hands, pinch your scalp all over your head—but be careful not to pull too hard at your hair as you do this. Make the movements quick and fluid. Your scalp should tingle when you've finished.
3. Make your hands into claws and place your fingertips at your hairline on your forehead (palms over your head). Use your fingertips to 'comb' your whole scalp from front to back.
4. Finally, beginning at the centre of your head, work outwards and around, using your fingertips to make small circular movements that gently manipulate the scalp. Use a firm pressure, and do this over the whole of your head.

snacks, and don't skip meals. This will help to keep your blood-sugar levels in balance and, in turn, control your hormone levels by helping to prevent an excess of the male hormone testosterone in your system.

- **Protein**. Your hair follicles need good-quality protein in different forms in order to grow, so stock up on pulses, nuts, seeds and fish. In the body, protein is broken down into its constituent parts, which are called 'amino acids'. The most important of these for preventing hair loss are arginine, cysteine, lysine and tyrosine, all of which are found in protein-rich foods.
- **Essential fats**. If you have dry hair that breaks easily and lacks shine, you may lack the necessary levels of essential fatty acids. Boost your intake by eating plenty of oily fish (such as salmon, tuna and sardines), nuts and seeds.
- **Biotin**. Egg yolk, brown rice, lentils, oats, soybeans, sunflower seeds, walnuts and green peas are rich in biotin. This important B vitamin helps to metabolize essential fats, and is crucial for healthy hair, as well as for the overall health of your skin and nails.
- **Iron**. Eat plenty of iron-rich foods such as dark-green vegetables, and stock up on vitamin-C-rich foods, too, which will help your body to better absorb iron.

Supplements

- **B-complex vitamins** (containing 25 mg of each B vitamin) should be taken daily, and are essential for your nervous system so, if your hair loss is stress-related, supplementing with this will help.
- **Vitamin C with bioflavonoids** (500 mg twice daily as magnesium ascorbate) will help in the manufacture of collagen, which holds hair tissue together, thus preventing splitting. Vitamin C also aids iron absorption.
- **Vitamin E** (600 IU daily) is thought to help reduce testosterone levels in women.
- **Zinc** (50 mg daily, as zinc citrate) helps the oil glands on hair follicles to prevent shedding, whereas a zinc deficiency can weaken hair, causing it to break, while preventing it from growing back at its normal rate.

Other natural treatments

- **Homeopathy**. See a homeopath for a constitutional remedy or try the following, taking a 30 C potency of the appropriate remedy, twice daily:
  - *Nat Mur* (*Natrum Muriaticum*) is the ideal remedy if your hair falls out when it's brushed, combed or even touched
  - *Phos* (*Phosphorus*) is suitable if you have bald spots and your hair comes out in clumps
  - *Sepia* is the one to use when your hair loss is accompanied by fatigue and chronic headaches.
- **Acupuncture**. In traditional Chinese medicine, hair loss (and premature greying) is linked to a deficiency in the kidney meridian. You may wish to see an acupuncturist to have the relevant meridians stimulated to promote hair growth.
- **Aromatherapy**. Massaging essential oils into your scalp can increase the circulation to your head while reducing stress. Certain oils are beneficial to hair health. *Rosemary essential oil* is thought to stimulate hair follicles. Dilute 3–6 drops in 3 tsp of a carrier oil such as jojoba or grapeseed, and massage the blend into your scalp. *Clove oil* (at the same dilution), which contains eugenol, is known to stimulate hair growth, and *cedar of Lebanon oil* can also help. Do this two to three times per week, using the four-step technique (see box, page 29). If you can bear to, leave the diluted essential oil in your hair overnight and wash it out in the morning. (Wear a shower cap to protect your bed linen.)

- **Omega-3 fatty acids** (1000 mg of fish oil daily, containing 700 mg EPA and 500 mg DHA) should be taken for three months (or take linseed/flaxseed oil if you're a vegetarian).

Herbs

- **Horsetail** (*Equisetum arvense*). The outer skin of the stems of this herb contains large amounts of silica, a chemical compound that improves the formation of connective tissue in the body and, so, improving the health of the hair (and skin and nails, too). Take 300 mg in capsule form, twice daily.
- **Siberian ginseng** (*Eleutherococcus senticosus*). If stress is contributing to your hair loss, this will help to support your adrenal glands. Take 1 tsp of tincture in a little water, or 250–300 mg in capsule form, twice daily.
- **Herbal hair rinse**. Make your own rosemary hair rinse by steeping 30 g (1 oz) of rosemary leaves and stems in 570 mL (1 pint) of water for 20 minutes. Wash and rinse your hair as you would normally, then do a final rinse using your rosemary 'tea'. Do this every time you wash your hair, as rosemary is believed to encourage hair growth from follicles. For extra shine, add a cup of nettle tea.

Self-help

- **Be gentle with your hair**. Use a soft brush and avoid blow-drying, curling or straightening your hair as much as possible. If you must use hair straighteners or curling tongs, then make sure to use a heat-resistant hair protector spray, too, made from natural products (together with a natural shampoo and conditioner). To prevent breakages, comb your hair carefully when wet, making sure to tease out tangles rather than pulling.
- **Avoid stress**. Stress can worsen hair loss, so endeavour to keep your stress levels to a minimum. Find a relaxation routine that suits you, be it meditation, visualization or a breathing exercise. Make sure you spend at least part of each day—even if it's only 30 minutes—doing something you enjoy for yourself, such as reading a book or listening to your favourite music.

Marilyn Glenville

Adapted from The Natural Health Bible for Women *by Marilyn Glenville, London: Duncan Baird Publishers, 2010. Dr Glenville is a nutritional therapist, psychologist, author and broadcaster specializing in female hormone problems.*

Saving face

Research shows that you can keep wrinkles at bay without resorting to botox or surgery

Skin changes such as wrinkles and sagging are among the most visible signs of ageing. To an extent, such changes are an inevitable part of growing old—the result of 'intrinsic' (internal) factors beyond our control that include genes and changes in hormone levels over time.

However, research shows that the vast majority of skin ageing is due to 'extrinsic' (external) factors, such as sun exposure, smoking, poor nutrition, environmental pollution and stress. These are factors that we can do something about and, ultimately, our understanding of them is the key to keeping our skin young- and healthy-looking for longer.

Sun exposure

Although sunlight is undoubtedly good for us, when it comes to our skin, overexposure to the sun is its number-one enemy. Ultraviolet (UV) radiation from the sun is by far the most important factor in skin ageing, especially its premature ageing. In fact, 'photoageing', as it's known, is estimated to account for up to 90 per cent of all visible skin-ageing (Int J Cosmet Sci, 2008; 30: 87–95).

Both ultraviolet A (UVA) and B (UVB) radiation are responsible, but UVA penetrates more deeply into the skin and, thus, is able to damage both the epidermis—the outermost layer, made up of skin cells, pigment and proteins—and the dermis—the next layer in, made up of collagen and elastin fibres (Acta Dermatovenerol Alp Panonica Adriat, 2008; 17: 47–54).

The tell-tale signs of photoageing include wrinkles, sagging, dry or rough skin, age spots, spider veins and actinic keratoses—thickened wart-like, rough, reddish-brown to blackish patches of skin (Coll Antropol, 2008; 32 Suppl 2: 177–80). Most at risk are those of us who live in sunny climates, spend a lot of time outdoors and have fair skin (Arch Dermatol, 2002; 138: 1462–70).

The most obvious plan of action is to protect the skin with a sunscreen. Indeed, UV filters are now found in numerous everyday cosmetic products such as makeup, moisturizers and hand creams. However, several sunscreen chemicals are linked to adverse effects, such as hormone disruption and allergic reactions (see *WDDTY* vol 19 no 3).

A safer option is to use products that contain physical filters, such as zinc oxide and titanium dioxide, rather that chemical filters. These agents are capable of reflecting both UVA and UVB rays and, as they don't penetrate into the skin, they are unlikely to have toxic/allergic effects (Acta Dermatovenerol Alp Panonica Adriat, 2008; 17: 47–54).

Even better, look for products that contain antioxidants such as vitamins C and E, and coenzyme Q10, as these natural free-radical fighters play an important role in protecting the skin against UV-induced damage (Curr Probl Dermatol, 2001; 29: 157–64). One 16-week study found that twice-daily application of a cream containing antioxidants provided protection against UVB-induced oxidative stress in the epidermis, a crucial factor in skin ageing (Photodermatol Photoimmunol Photomed, 1999; 15: 115–9). Another revealed that the topical application of coenzyme Q10 was effective against UVA-induced oxidative stress in the

Natural wrinkle fighters

- **Coenzyme Q10**. Topical application of this antioxidant not only protects against sun damage, but also appears to improve the appearance of wrinkles. A clinical trial showed that the use of a 1-per-cent CoQ10 cream for five months significantly reduced wrinkles, as rated by a dermatologist (Biofactors, 2008; 32: 237–43).
- **Vitamin C**. In one placebo-controlled trial, a topically applied cream containing 5-per-cent vitamin C led to a "clinically apparent improvement" of sun-damaged skin after six months. In particular, deep furrows and skin elasticity were improved (Exp Dermatol, 2003; 12: 237–44).
- **Niacinamide**. According to clinical testing, applying 5-per-cent niacinamide (vitamin B3) in a moisturizer to the face can improve various signs of ageing, including hyperpigmentation, fine lines and wrinkles, redness/ blotchiness, yellowing (sallowness) and elasticity (Int J Cosmet Sci, 2004; 26: 231–8; Dermatol Surg, 2005; 31: 860–5).
- **Alpha-lipoic acid (ALA)**. In a trial of 33 women, aged 40–75 years, each treated half her face with a cream containing 5-per-cent ALA, and the other half of her face with a placebo cream, twice daily for 12 weeks.

  The results showed significant improvement in the signs of photoageing on the ALA-treated side of the face. Specifically, laser profilometry—which measures the surface of the skin with extreme accuracy—revealed an average decrease in skin roughness of 51 per cent compared with 41 per cent on the placebo-treated side (Br J Dermatol, 2003; 149: 841–9).

skin, concluding that "CoQ10 has the efficacy to prevent many of the detrimental effects of photoaging" (Biofactors, 1999; 9: 371–8).

Antioxidants taken internally may also be beneficial. Taking supplements, or including plenty of antioxidant-rich foods in your diet, can bolster the skin's natural defences against sunlight (see below).

Other ways to protect yourself against the sun include avoiding exposure between 11am and 3pm, when the sun's rays are strongest, and wearing protective clothing, such as a wide-brimmed hat and long sleeves, when you're outdoors during the day. Also, be sure to avoid indoor tanning devices, which are another source of UV radiation.

Hope in a jar?

Of the countless creams on the market claiming to erase wrinkles and stop sagging, few have undergone rigorous scientific trials to prove their effectiveness. One exception is No 7 Protect & Perfect Intense Beauty Serum, made by Boots, which was tested last year in a double-blind, randomized controlled trial. After one year, the researchers found "significant clinical improvement in facial wrinkles", which they believed was due to the increased production of fibrillin-1 in the skin, a protein that promotes skin elasticity. Bear in mind, however, that the study was funded by Boots (Br J Dermatol, 2009; 161: 419–26).

Nevertheless, for those who might be looking for a more natural solution (the Boots serum contains a raft of harsh chemicals), your best bet is to look for products with high levels of antioxidants such as vitamin C and E, and co-enzyme Q10.

Weleda's new Pomegranate Firming Face Serum, for example, contains antioxidant-rich pomegranate juice and, according to the website, dermatological tests have shown that, after 28 days of use, the depth of wrinkles was reduced by 29 per cent while skin moisture levels increased by 39 per cent (www.weleda.co.uk).

Dietary factors

Nutrition is one of the most important factors involved in skin health and its condition. If the skin is not sufficiently nourished, it won't be able to defend itself against external insults, such as the sun's rays, that contribute to the signs of ageing (Dermatoendocrinol, 2009; 1: 271–4).

Indeed, an international study conducted in 2001 confirmed that the foods we eat can have a significant impact on the state of our skin (J Am Coll Nutr, 2001; 20: 71–80). Researchers at Melbourne's Monash University studied 453 people living in Australia, Greece and Sweden to determine whether or not food and nutrient intakes were related to wrinkling of the skin in sun-exposed areas.

What they discovered was that a high intake of vegetables, legumes and olive oil appeared to be protective against developing wrinkles, while a high intake of meat, dairy and butter had the opposite effect. In particular, full-fat milk (rather than skimmed milk, cheese and yoghurt), red meat (especially processed meat), potatoes, soft drinks/cordials and cakes/pastries were all associated with extensive skin-wrinkling. Protective foods included leafy green vegetables, broad beans, lima beans, nuts, olives, dried fruit/prunes, cherries, grapes, apples and tea.

According to the researchers, these protective foods may have contributed to less skin-wrinkling because of their high contents of antioxidant vitamins and phytochemicals. Indeed, previous studies found that certain antioxidants and plant ingredients can protect the skin against UV radiation when taken as supplements, so high intakes of these nutrients would tend to lead to less wrinkling at sun-exposed sites. The antioxidant vitamins C and E, the carotenoids beta-carotene and lycopene, and the proanthocyanidins, a group of flavonoids found in grape seeds and other plant sources, are just some of the nutrients that appear to have photoprotective effects (see *WDDTY* vol 19 no 3).

The Monash researchers also noted that sugar and sugar products can contribute to poor skin health through the 'glycosylation of proteins'—when a sugar molecule attaches itself to a protein molecule—in the skin. This, in turn, can contribute to skin-wrinkling and photoageing, and might explain why foods such as soft drinks and cakes/pastries are associated with more wrinkling of the skin (J Am Coll Nutr, 2001; 20: 71–80).

A more recent study also reported a link between diet and skin-ageing. Using data from the first National Health and Nutrition Examination Survey, UK researchers examined the associations between nutrient intakes and 'skin-ageing appearance' in more than 4000 American women aged 40–74 years. They found that having higher dietary intakes of vitamin C (from orange juice, citrus fruits, fruit juices and tomatoes) and linoleic acid (found in oils such as rapeseed and soybean oils, and in foods such as green leafy vegetables and nuts), and lower intakes of fats and carbohydrates, were associated with better skin-ageing appearances.

In fact, higher vitamin C intakes were associated with fewer wrinkles and less senile dryness (dry skin as a result of ageing); higher linoleic-acid intakes were associated with less of a likelihood of senile dryness and thinning skin; and higher fat and carbohydrate intakes were linked with a more wrinkled and thinning skin. These associations were independent of age, race, education, sunlight exposure, income, menopausal status, body mass index, supplement use, physical activity and energy intake (Am J Clin Nutr, 2007; 86: 1225–31).

In general, the evidence suggests that including plenty of antioxidant-rich fruit and vegetables in your diet is one of the best things you can do to save your skin.

Smoking and pollution

Cigarette-smoking is another important causal factor in skin ageing; so, if you need yet another reason to quit the habit, think of your skin. Smoking increases the quantities of matrix metalloproteinases (MMPs)—enzymes that break down the collagen, elastin fibres and connective tissue in the skin (J Dermatol Sci, 2007; 48: 169–75). These

enzymes also cause oxidative stress, which impairs collagen production (J Investig Dermatol Symp Proc, 2009; 14: 53–5). The result of this double whammy is skin that's old before it's time. Indeed, studies show that the longer you continue to smoke, the older you'll look (Plast Reconstr Surg, 2009; 123: 1321–31).

Fortunately, just stopping smoking can have a rejuvenating effect on the skin. In a study of 64 women who took part in a nine-month stop-smoking programme, the results suggested that kicking the habit can dramatically reduce the biological age of your skin.

The women's skin was assessed before and after the programme in terms of lines, pigmentation, elasticity, brightness and texture to establish its biological age. At the end of the programme, an average reduction of about 13 years in the biological age of the women's skin was found whereas, at the beginning of the study, their skin had an average biological age of nine years older than their actual, chronological age (Skinmed, 2010; 8: 23–9). These results suggest that stopping smoking is an important way to prevent and even reverse the signs of skin-ageing.

Besides tobacco smoke, the air pollution from road traffic and other sources can also have a negative impact on the skin.

A recent German study of 400 elderly women found that air-pollution exposure was significantly linked to extrinsic signs of skin ageing and, in particular, pigment spots. An increased exposure to soot and particles from traffic was associated with 20-per-cent more pigment spots on the forehead and cheeks. Back-ground particle pollution that was not directly attributable to traffic was also positively associated with pigment spots on the face (J Invest Dermatol, 2010 Jul 22; Epub ahead of print).

While it's impossible to completely avoid air pollution, increasing antioxidant intake can boost the skin's natural defences. Also, keep tabs on the air pollution in your area by contacting your local air-quality monitoring service (in the UK: www.airquality.co.uk/index.php; in the US: www.epa.gov/oar/airpolldata.html). When pollution levels are high, avoid spending a lot of time outdoors if possible. For further advice on how to minimize your exposure to air pollution, see *WDDTY* vol 18 no 5.

Other face-saving factors

Clearly, wrinkles, sagging and the other signs of skin ageing are not simply the inevitable consequence of getting old. The sun, tobacco smoke and outdoor air pollution can all have a negative impact on our skin, and what we eat can determine how well our skin responds to these insults.

In addition, severe physical and psychological stress can both take their toll on the appearance of your skin (Acta Dermatovenerol Alp Panonica Adriat, 2008; 17: 47–54), so consider stress management techniques such as meditation and yoga as part of your anti-ageing regime.

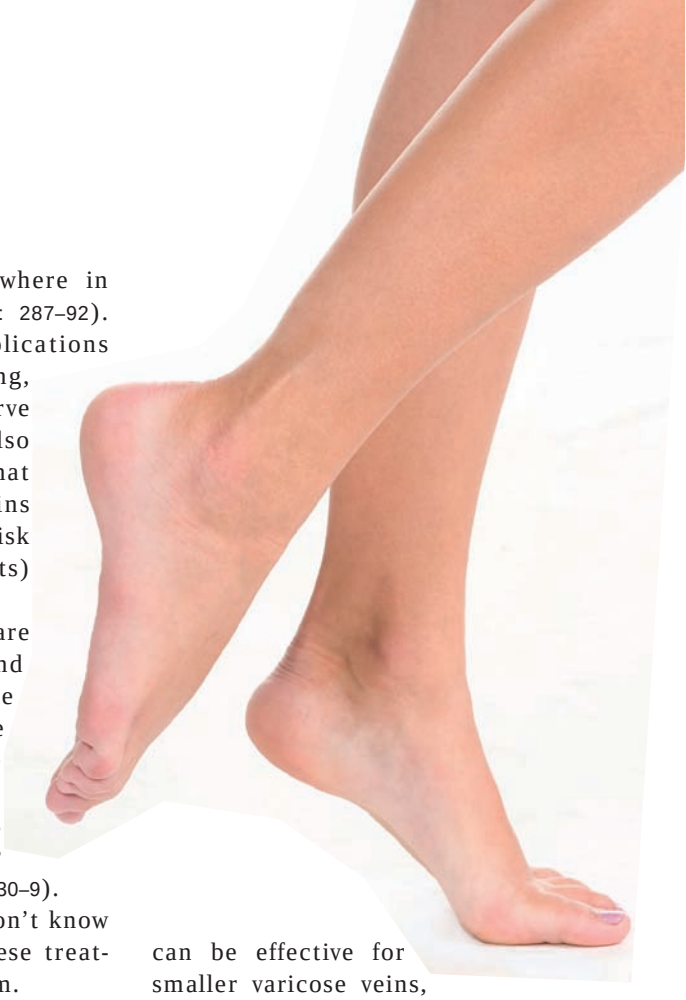
Also, it may help to get into the habit of sleeping on your back rather than on your side, as years of resting your face on the pillow in the same way every night can lead to wrinkles known as 'sleep lines' (Scand J Plast Reconstr Surg Hand Surg, 2004; 38: 244–7).

Finally, don't forget to drink plenty of water to keep your skin hydrated and smooth-looking.

Joanna Evans

Varicose veins

Varicose veins affect nearly a third of us and rarely go away on their own so, thankfully, there's a range of herbs and supplements that can help



Varicose veins are veins that have become twisted and swollen because blood isn't flowing through them properly. They can develop anywhere in the body, but the veins in the legs are the most commonly affected, as they have to move blood along against gravity. Increased blood pressure and hormonal changes during pregnancy can also trigger the problem, which helps to explain the higher incidence of varicose veins in women than in men (Cochrane Database Syst Rev, 2007; 1: CD001066).

For many sufferers, varicose veins are simply a cosmetic concern. Others, however, may experience aching, throbbing, itching or burning sensations, which usually worsen with prolonged standing.

Rarely, varicose veins can lead to more serious problems such as infection, leg ulcers and thrombosis (Am Fam Physician, 2008; 78: 1289–94).

Treating varicose veins

Conventional treatment of varicose veins includes a range of options—from compression stockings to surgery and laser treatments. Compression stockings, which steadily squeeze the legs to improve blood flow, are usually the first course of action, but studies conclude that they are no more effective than simply resting (Cochrane Database Syst Rev, 2007; 1: CD001066).

Surgery, on the other hand, can effectively get rid of these unsightly veins and help to relieve symptoms. However, varicose veins may gradually recur through a process of 'neovascularization'—regrowth and enlargement of veins—even after successful surgery, or they may simply develop elsewhere in the legs (BMJ, 2006; 333: 287–92). Other possible complications include bleeding, bruising, infection, scarring and nerve damage. There is also evidence that suggests that surgery for varicose veins might even increase the risk of thrombosis (blood clots) (BMJ, 1996; 312: 1158).

Two newer treatments are radiofrequency ablation and laser treatment, which use heat to seal off varicose veins. These less invasive techniques appear to be at least as effective as surgery, but with fewer risks (J Vasc Surg, 2009; 49: 230–9).

Nevertheless, we still don't know how safe and effective these treatments are in the long term.

Yet another option is sclerotherapy, in which chemicals are injected into varicose veins to cause their walls to collapse. This can be effective for smaller varicose veins, but the risk of neovascularization remains high. There are also concerns over the development of deep vein thrombosis (DVT), visual

Tips for prevention

The following self-care methods may help to prevent varicose veins or, at least, stop them from getting worse.

- **Exercise regularly** to aid your circulation. Walking or anything that gets the legs moving is ideal.
- **Reduce leg strain** by avoiding prolonged standing and excessive heavy lifting.
- **Elevate the legs**. To improve leg circulation, take a few short breaks daily to raise your legs to above the level of your heart by, for example, lying down with your legs propped up on three or four pillows.
- **Don't cross your legs when sitting**, as this can slow the upward flow of blood back to the heart and increase pressure on the leg veins.
- **Try massage** and other therapies that increase circulation.
- **Watch your weight**, as overweight and obesity are known to contribute to the development of varicose veins (Am Fam Physician, 2008; 78: 1289–94).
- **Avoid high heels**. Low-heeled shoes work the calf muscles more, which is better for venous blood flow.
- **Avoid wearing tight clothing** around your waist, legs and groin, as this can reduce blood flow.
- **Consider your diet**. Hard stools and constipation can lead to varicose veins as straining increases pressure in the abdomen that, in turn, increases pressure on the lower legs. So, it's important to consume good amounts of water-soluble fibre and liquids. Foods that are especially helpful for varicose veins include berries, cherries, grapes, buckwheat and onions.

disturbances and stroke (Am Fam Physician, 2008; 78: 1289–94; J Neurol Neurosurg Psychiatry, 2010; 81: 582–3).

Happily, there are a number of natural ways to treat varicose veins.

Natural remedies

- **Supplements**. Among the most useful supplements are those that contain flavonoids, some of which have anti-inflammatory effects and can help to strengthen the blood vessels.
  - ❖ *Hydroxyethylrutoside* (HR), a type of flavonoid derived from rutin (found in buckwheat and asparagus), improved varicose veins in a controlled trial of pregnant women (Zentralbl Gynakol, 1995; 117: 190–7). Typically, the HR dose is 1000 mg/day, but its efficacy may be increased by combining the supplement with topical HR gel, applied two to three times a day (Angiology, 2008; 59 Suppl 1: 7S–13S).
  - ❖ *Proanthocyanidins*, flavonoids found naturally in apples, pine bark, grapeseed, bilberry and some red wines, have also been successfully used to treat varicose veins. A single dose of proanthocyanidins (150 mg) improved leg-vein function in people with widespread varicose veins (Sem Hop, 1981; 57: 2009–13).
  - ❖ *Daflon*, a combination of the flavonoids diosmin and hesperidin, has been shown to be effective for chronic venous insufficiency (CVI; pooling of blood in the legs), a condition commonly associated with varicose veins. According to one review, its comprehensive mode of action on the veins, lymphatic vessels and microcirculation makes it the treatment of choice not only for the early stages of CVI, but also for the more severe stages, too (Angiology, 2001; 52 Suppl 1: S49–56).
  - ❖ *Pycnogenol* (maritime pine-bark extract) may be even more effective than Daflon, according to one study. Italian researchers found that, after eight weeks, CVI patients taking 150 or 300 mg/day of Pycnogenol improved more than those taking 1000 mg/day of Daflon (Clin Appl Thromb Hemost, 2006; 12: 205–12).
- **Herbs**. Plant remedies are a popular treatment for varicose veins, and the following appear to be especially useful.
  - ❖ *Horse chestnut (Aesculus hippocastanum)*. Extracts from the seeds of this plant (HCSE) have traditionally been used to treat patients with CVI and to alleviate its associated symptoms. Four clinical trials in patients with CVI and one study in patients with varicose veins showed that HCSE—taken either as an oral tincture or tablets (20 mg or 50 mg), or applied as a topical gel—can reduce leg pain and swelling as well as heaviness and itching (Adv Ther, 2006; 23: 179–90). For best results, take it as early in the condition as possible (BMC Cardiovasc Disord, 2001; 1: 5; doi: 10.1186/1471-2261-1-5).
  - ❖ *Gotu kola (Centella asiatica)*. This appears to have a beneficial effect on the connective tissues in varicose veins (Int J Clin Pharmacol Res, 1990; 10: 229–33). In a randomized placebo-controlled trial—the 'gold standard' of scientific evaluation—a titrated extract of gotu kola (60 or 120 mg/day) was effective for reducing leg swelling and leg heaviness in patients with CVI (Angiology, 1987; 38: 46–50).
  - ❖ *Butcher's broom (Ruscus aculeatus)*. Taken orally, this herb can improve venous tone

Spider veins

Also known as 'telangiectasias' or 'sunburst varicosities', so-called spider veins are a smaller, milder form of varicose veins. Although they are harmless and generally cause no pain or discomfort, many people hate the way they look.

Sclerotherapy is the usual treatment for spider veins but, as already mentioned (see main text), it's been associated with serious side-effects such as stroke and deep vein thrombosis (DVT). More commonly, sclerotherapy causes hyperpigmentation, or darkening of the skin, along the course of the treated vein. This side-effect is reported in up to 30 per cent of patients treated for spider/varicose veins. In most cases, it disappears within 6–24 months but, for some, the discoloration may persist for five or more years (J Dermatol Surg Oncol, 1987; 13: 547–50).

Fortunately, the natural remedies and preventative tips for varicose veins can benefit spider veins, too. In addition, vitamin K cream is claimed to be an effective natural treatment. Topical vitamin K was able to reduce laser-induced bruising and purpura (red/purple discolorations due to bleeding under the skin) (J Am Acad Dermatol, 2002; 47: 241–4; Dermatol Surg, 1999; 25: 942–4). However, *WDDTY* could find no published studies showing that vitamin K can reduce or eliminate spider veins themselves.

Nevertheless, an unpublished study reported in *WDDTY*'s sister publication *PROOF!* (vol 4 no 3) tested the efficacy of a 5-per-cent pharmaceutical-grade vitamin K product (Dermal-K) on 50 women. Among these women after six months, three saw significant reduction or complete healing of their spider veins within 8–14 days, 17 saw significant reduction or complete healing in 15–30 days, 26 achieved significant reduction within 31–46 days, two had a significant reduction within 47–120 days and two showed no effects at all. There was no improvement in 50 women who used an aloe vera skin cream as a comparison.

So, vitamin K cream may be worth a try, but proceed with caution as the long-term safety of this treatment remains unknown.

Another option is to mask spider veins with makeup. Indeed, there are long-lasting (and some waterproof) body makeups specially formulated to camouflage spider veins and other skin blemishes. In the UK, the British Red Cross runs a free treatment service at centres across the country, used by patients with birthmarks, burns, scars and spider veins (see www.redcross.org.uk).

and poor circulation (Fortschr Med, 1989; 107: 52, 55–8). In a placebo-controlled trial of 148 women with CVI, those taking butcher's broom extract saw significant improvements in leg swelling as well as in heaviness, tension and tingling (Arzneimittelforschung, 2002; 52: 243–50). Dosage for capsules standardized for ruscogenins is 7–11 mg, although some experts recommend higher dosages of 16.5–33 mg of total ruscogenins three times a day (Altern Med Rev, 2001; 6: 608–12).

❖ *Witch hazel (Hamamelis virginiana)*. This herb is famous for its astringent and anti-inflammatory properties (Altern Med Rev, 2001; 6: 608–12), and, in the form of an ointment, it is recommended by the German Commission E for varicose veins.

Note, however, that the ointment may need to be applied three or more times a day for several weeks before there is any noticeable improvement.

❖ *Red vine leaf (Folia vitis viniferae) extract*. A source of flavonoids, this herb was able to reduce calf swelling in patients with CVI (Arzneimittelforschung, 2000; 50: 109–17). Another study concluded that red vine leaf extract shows "a fast onset of action and an excellent efficacy" in the treatment of CVI (Praxis [Bern 1994], 2006; 95: 187–90). The usual dose is 360–720 mg/day.

◆ **Hydrotherapy**. The use of water to treat illness appears to work for varicose veins. In a study of 61 patients, three and a half weeks of hydrotherapy reduced leg swelling and other symptoms of varicose veins (Vasa, 1991; 20: 147–52). In another study, thermal hydromassage therapy was also effective for patients with CVI (Minerva Cardioangiol, 2008; 56: 401–8).

◆ **Reflexology**. This form of foot massage reduced symptoms in a study of 55 women with varicose veins and leg swelling (Cochrane Database Syst Rev, 2007; 1: CD001066).

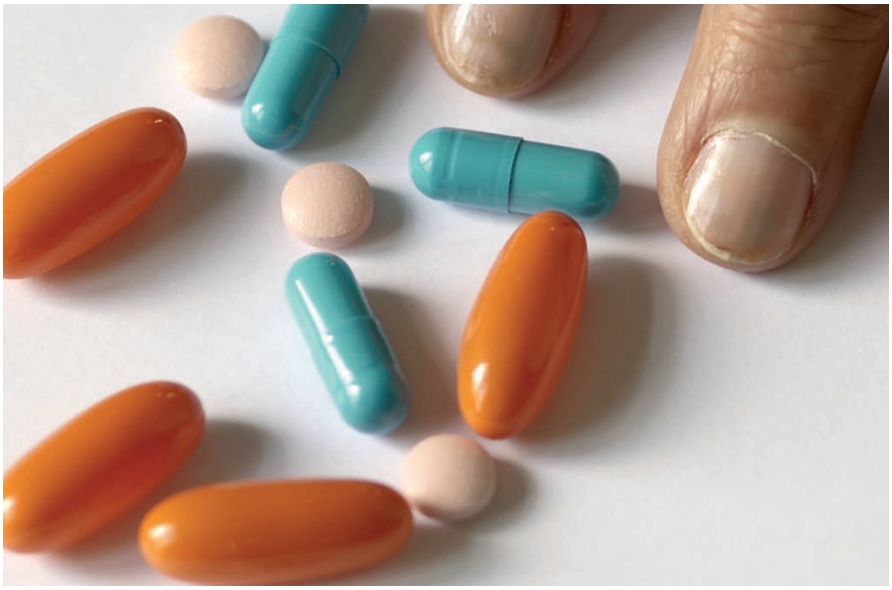
◆ **Homeopathy**. According to *WDDTY* columnist Dr Harald Gaier, there are several effective homeopathic remedies for varicose veins, including *H. virginiana* (witch hazel), *Aristolochia clematis*, *Paeonia officinalis*, *Viburnum opulus* and *Ruta graveolens*.

For best results, see a qualified homeopath, who can prescribe a remedy based on your individual symptoms.

Joanna Evans

Medicine and the elderly: Age shall not weary them—but drugs might

For the elderly, the cure is very likely worse than the disease



Poets may exhort us not to grow old, but they could have added the rider that, when we do, stay away from the doctor for as long as possible. Growing evidence suggests that aggressive interventions by doctors are accelerating the decline in the general health and mental capacities of the elderly, and may even be a direct contributor to their death.

The over-65s take one-third of all pharmaceuticals that are prescribed by doctors every year, despite the fact that they represent just 13 per cent of the total population. On average, an elderly person is taking around six drugs at any given time (Johnston CB. *UCSF Division of Geriatrics Primary Care Lecture Series May 2001. Geriatric Assessment in a Time Dependent Practice: Practical Approaches for Primary Care Practitioners*).

Polypharmacy—when more than one drug is prescribed at a time—is an even bigger problem in hospitals and care homes, where the average patient is given at least seven different drugs every day. In addition, powerful dementia drugs are being prescribed for most patients as a 'chemical cosh' to keep them quiet, evidently mainly for the convenience of the medical staff. An official UK government review has revealed that the drugs are being inappropriately prescribed in around 80 per cent of cases. In the UK alone, this works out to around 150,000 people who are being given anti-psychotic drugs just to keep them pacified. Worse, the drugs are directly responsible for about 1800 deaths every year (http://news.bbc.co.uk/2/hi/8356423.stm).

This may perhaps explain why 11 per cent of elderly patients, admitted to hospital for other medical conditions, will suffer a heart attack while they are there. In addition, they are also twice as likely as a younger patient to die within 30 days from such an attack. Researchers made the discovery when they examined the health records of 7054 patients who were admitted to hospital as part of the US Veterans' Health Administration between 2003 and 2004. Of those patients, 792—or 11.2 per cent—suffered a heart attack while under hospital care (Arch Intern Med, 2006; 166: 1410–6).

Tick-box medicine

Most patients are over 65 and, yet, they rarely see a doctor who specializes in geriatric medicine. This might be because, in almost every country except the UK, there is a serious shortage of geriatricians. In Canada, for example, there are fewer than 200 qualified geriatricians serving the entire country. In an attempt to attract more medical graduates into geriatric medicine, Dr Laura Diachum, at the University of Western Ontario, has gone as far as to describe the speciality as "totally sexy" (J Am Geriatr Soc, 2006; 54: 1453–62).

As a result of the lack of geriatric specialists, an elderly patient is invariably seen by a general practitioner, whose almost instinctive tendency will be to start reaching for the prescription pad. Sadly, as discovered by a doctoral thesis defended by Sandra Pennbrant, at the Sahlgrenska Academy in Sweden, elderly patients tend to become passive when faced by the doctor and feel intimidated by the practitioner's power and, so, fail to participate in the consultation by almost never challenging the decision to start taking a drug or even asking any questions (http://gupea.ub.gu.se/dspace/handle/2077/21198).

Doctors routinely hand out prescription drugs simply because the patient is old, not because he or she needs them, says Michael Oliver, an emeritus professor of cardiology at Edinburgh University. This 'tick-box medicine', as he calls it, means that elderly people are not only taking drugs they don't need, but they are also being exposed to side-effects that can seriously endanger their health. "Nowadays, few elderly people are allowed to enjoy being healthy," he says (BMJ, 2009; 338: b873).

One example of this so-called tick-box medicine is bringing down levels of blood cholesterol, especially in the elderly. But this is, in fact, a failure to understand the changing metabolism of older patients, who appear to need higher levels of cholesterol for their

general wellbeing and, especially, to support mental acuity. One study that involved 3572 men, aged 71–93 years, discovered that those who had the lowest cholesterol levels—from 2.09 to 4.32 mmol/L (80.0 to 167.0 mg/dL)—were up to 40 per cent more likely to die than those whose cholesterol levels were higher. Indeed, this study, which monitored the health of the participants for 20 years, questioned whether there was any "scientific justification for lowering cholesterol to very low concentrations (below 4.65 mmol/L [179.8 mg/dL]) in elderly people" (Lancet, 2001; 358: 351–5).

Not only may higher cholesterol levels be health-giving in the elderly, but the cholesterol-lowering drugs themselves may also be doing more harm than good, according to a study from the Yale University School of Medicine. There, the researchers found that, while there was a marginal benefit from the drugs in reducing heart fatalities, they also found that the patients were dying of other causes as a result of taking the drugs (JAMA, 1994; 272: 1335–40).

Worse, most general practitioners are unaware of the dangers of the drugs they are prescribing to their elderly patients, despite the frequent drug alerts and warnings they are sent by drug-monitoring agencies.

In fact, researchers have discovered that around one in five elderly patients is being given drugs that are dangerous, and could be the cause of debilitating side-effects. In a study of 760,000 elderly people taking a prescription drug, it was revealed that 21 per cent were taking one or more drugs that were on the Beers list, a compilation of all the prescription drugs that geriatric patients, in particular, should avoid (Arch Intern Med, 2004; 164: 1621–5).

In another study, researchers found that 20 drugs had been identified as being too dangerous for use by the elderly and, yet, most of these patients (79.6 per cent) were being prescribed one of these drugs and 20.4 per cent were taking two or more, including the beta-blocker propranolol, the antihypertensive agents methyldopa and reserpine, the painkiller dextropropoxyphene and the anticoagulant dipyridamole (JAMA, 1994; 272: 292–6).

Many elderly people regularly take a common NSAID (non-steroidal anti-inflammatory drug) such as aspirin and ibuprofen to help ease their aches and pains. Yet, according to a survey of 4099 people aged 70 years and over, these drugs increase the risk of kidney dysfunction. Those who took an NSAID at least once a day had the highest levels of blood urea nitrogen and serum creatinine, both of which are markers of kidney problems (J Am Geriatr Soc, 1999; 47: 507–11).

Prescribed drugs may also be responsible for dry eyes and dry mouth, assumed to be a natural consequence of growing old. However, common painkillers such as aspirin could be responsible, according to a study of 2481 patients aged 65–84 years. Antidepressants and antipsychotics may also be the cause of similar side-effects (Arch Intern Med, 1999; 159: 1359–63).

What's more, doctors are more than

Living a long and healthy life

Aside from avoiding drugs and medicine, there are three keys to a healthy life into old age: diet; exercise; and an active social life.

- **Diet**. Vitamin E—found in nuts, seeds, and corn and olive oil—is arguably the single most important nutrient in determining whether or not we will live a long and healthy life. In a study of 698 men and women, aged over 65 and living in Tuscany, Italy, those who had low levels of the vitamin in their circulation suffered a decline in their physical capacity over a three-year period compared with those who had higher levels of the vitamin. This was also the only vitamin that had a direct impact on the physical wellbeing of the study participants. Their levels of folate, vitamin D, iron and B vitamins apparently made no difference to the physical decline. The researchers believe that vitamin E, which is an antioxidant, prevents damage to DNA, muscles and neurons (JAMA, 2008; 299: 308–15).

  The Mediterranean diet, primarily consisting of fresh fruit, vegetables and olive oil, has been proven in countless studies to help maintain health into older age. One study, involving 1393 participants, discovered that the diet protects against cognitive decline as we age. Those who stayed close to the diet nearly halved their chances of developing mild cognitive impairment compared with those who ate low amounts of fruits and vegetables (Arch Neurol, 2009; 65: 216–25).
- **Exercise**. Even moderate exercise, such as a brisk walk every day, can help to keep you healthy into older age, and even elderly patients who already have conditions such as heart disease and arthritis can see improvement. In heart patients aged 50 years and over, moderate exercise that leaves you a little out of breath increased life expectancy by 3.7 years in men and 3.5 years in women (Arch Intern Med, 2005; 165: 2355–60). Older people with osteoarthritis of the knee also noticed improvements in their knee pain and mobility after 18 months of brisk walking for 40 minutes, three times a week (JAMA, 1997; 277: 25–31). Overall, older people who take regular exercise have stronger hearts, better circulation, stronger bones, better balance, less pain, better sleep quality, sharper minds and a lower risk of developing most cancers (Am Fam Physician, 2002; 65: 419–26).
- **Social networks**. An active social life, including volunteer work, attending sports and social events, and visiting friends and relatives, is as important as exercise for helping older people to maintain good health. In a US study of 906 retired people, it was found that physical decline was up to a third more rapid in those who were socially inactive, even when they took regular exercise (Arch Intern Med, 2009; 169: 1139–46). Curiosity and maintaining an interest in the world also play important roles in determining how long—and how well—we live. One study of 1118 men, with an average age of 70 years, found that having this attitude in life was the single most important factor in determining longevity.

  In ancillary analyses in 1035 older women (mean age: 69 years), curiosity appeared to be equally important in women as well (Psychol Aging, 1996; 11: 449–53).

ready to prescribe 'off-label'—handing out drugs for health problems for which they have been neither tested nor licensed to treat. The problem has become so prevalent, and dangerous, that America's drugs watchdog, the Food and Drug Administration (FDA), has warned doctors to stop using atypical antipsychotic drugs to treat general behavioural problems in elderly patients. Indeed, agents such as olanzapine, aripiprazole, risperidone and quetiapine are so dangerous that they double the risk of death—and they are supposed to be prescribed only to those with schizophrenia (www.fda.gov/cder/drug/advisory/antipsychotics.htm).

The four giants

In 1965, when geriatric medicine was still in its infancy, Bernard Isaacs, a professor of geriatric medicine at Birmingham University in the UK, said the elderly were faced with four 'giants' that would determine their health: immobility; instability; incontinence; and impaired intellect (Isaacs B. *An Introduction to Geriatrics*. London: Ballière, Tindall and Cassell, 1965). Every health concern in the elderly could be traced back to one of those four conditions, he said. However, neither Isaacs nor the other pioneers of geriatric medicine could have foreseen that, in many older patients, these 'giants' would be caused by the very medicines that were supposed to help them, and not by the ageing process itself.

- **Immobility and instability**. Around 30 per cent of all over-65s fall each year, and this proportion rises to half of all those in hospital or in nursing care, where multiple drugs are an essential part of their daily regimen. Also, 25 per cent of these patients die within six months of falling. While there may be a number of reasons why an elderly person falls, prescription drugs are among the biggest causes, responsible for around 18 per cent of all cases (Johnston CB. *UCSF Division of Geriatrics Primary Care Lecture Series May 2001. Geriatric Assessment in a Time Dependent Practice: Practical Approaches for Primary Care Practitioners*).

  Tranquillizers and sedatives, such as benzodiazepine, can increase the risk of a fall in an elderly patient by nearly threefold. Flurazepam and triazolam (both of which are benzodiazepines) are the most dangerous, according to Canadian researchers, and the elderly patient was most likely to suffer a fall within the first two weeks of starting such drug therapy (Age Ageing, 1996; 25: 273–8).

  In a separate small US study a year later, the geriatric researchers in Durham, NC, found that benzodiazepines acted directly on the central nervous system, and affect neuromuscular processing and balance control, thereby causing falls, disorientation and slower responses in the elderly (J Am Geriatr Soc, 1997; 45: 435–40).

  Powerful antidepressants of the class known as 'SSRIs' (selective serotonin-reuptake inhibitors) as well as the older tricyclic drugs, can also significantly increase the risk of falls in the elderly (N Engl J Med, 1998; 339: 875–82). As a consequence, elderly people taking an SSRI are 2.4 times more likely to suffer hip fractures compared with those not taking these drugs, researchers from the University of Toronto, Ontario, have found. However, the earlier types of antidepressant are not much safer for elderly patients. In this study, which involved 8239 patients, aged 66 years and over, who had been treated in hospital for hip fractures, it was found that those taking a tricyclic antidepressant such as desipramine or nortriptyline were 2.2 times more likely to suffer from hip fractures as a result of falls (Lancet, 1998; 351: 1303–7).

  Insulin can also cause falls, and is among the three drugs that are also most likely to cause an adverse reaction in the elderly; the other two are warfarin, a blood thinner, and digoxin, a heart drug. The three drugs alone accounted for 59,108 of the 177,504 cases of adverse drug reactions reported by US emergency services in 2004–2005. Insulin can cause sudden hypoglycaemia (low blood sugar), which may result in a seizures or unconsciousness (Ann Intern Med, 2007; 147: 755–65).

  In fact, most prescription and over-the-counter drugs appear to increase the risk of falling. In a meta-analysis of studies published from 1996 to 2007, involving more than 79,000 participants, aged over 60, who were taking some sort of pharmaceutical, it was found that many drugs "significantly" raised the risk of falls. The biggest culprits included sedatives, SSRIs, antihypertensives, diuretics, beta-blockers, and even NSAID painkillers such as aspirin and ibuprofen.

  "Elderly people may be more sensitive to drugs' effects and less effective at metabolizing medications, leading to adverse events which, in turn, lead to falls," said researcher Carlo Marra, at the

Staying sharp

Mental decline isn't an unavoidable consequence of ageing. There's lots we can do to stay sharp right up to the end.

People who exercise at least once a week, don't smoke and maintain an active social life are more likely to retain their cognitive abilities throughout their 70s and 80s, according to a study of 2500 participants aged 70–79. However, an eight-year follow-up showed major cognitive decline in 16 per cent of this population, whereas 53 per cent displayed normal, age-related decline. Yet, the research team saw a unique profile of activity and social engagement that appeared to have protective effects in around 30 per cent of participants who displayed no cognitive decline whatsoever. A good education, not smoking and a good level of literacy were found to be important factors, and those who carried out volunteer work and were not living on their own also appeared to be less likely to suffer from cognitive decline (Neurology, 2009; 72: 2029–35).

Maintaining an interest in intellectual pursuits, such as reading and doing crossword puzzles, can also help to ward off dementia—and the more you do, the better. The study participants who engaged in 11 intellectual activities a week, such as reading every day and doing the crosswords four times a week, delayed the onset of dementia by 1.29 years compared with those who read or did the crosswords

University of British Columbia in Vancouver, Canada (Arch Intern Med, 2009; 169: 1952–60).

- **Incontinence**. This is often viewed as an unfortunate consequence of ageing, but this is not so. The US Agency for Health Care Policy and Research reports that eight out of 10 cases of incontinence can b either resolved or greatly improved by medical interventions (Agency for Health Care Policy and Research. *Overview: Urinary Incontinence in Adults, Clinical Practice Guideline Update*. Rockville, MD. March 1996; www.ahrq.gov/clinic/uiovervw.htm). The one exception is age-related prostate enlargement, which can cause urinary incontinence.

In fact, incontinence can arise for many reasons and in people of all ages, although one in 10 of those aged over 65, and three in 10 aged over 80, have some loss of bladder control, and half of all elderly patients in nursing homes have incontinence. Many prescription drugs cause temporary incontinence and, as the elderly take one-third of all drugs prescribed, it's not unreasonable to conclude that they are a primary cause of the condition in the over-65s. Anticholinergic agents, which block the passage of neural impulses, include drugs such as antihistamines, antidepressants, opiates, antispasmodics and Parkinson's drugs, and can all lead to incontinence (Cochrane Database Syst Rev, 2006; 4: CD003781), as can heart drugs such as the calcium-channel blockers.

Diuretics, or 'water pills', increase the body's loss of fluid by promoting the production of urine and, as a result, can frequently cause acute incontinence. They are among the most regularly prescribed drugs among the elderly—and often unnecessarily so, as one study has identified (BMJ, 1994; 308: 511–3).

Alpha-adrenergic blockers, which include drugs for hypertension (high blood pressure) such as doxazosin (Cardura), prazosin (Minipress) and terazosin (Hytrin), can also cause incontinence (Drug Saf, 1994; 11: 12–20), as can the angiotensin-converting enzyme (ACE) inhibitors such as benazepril (Merck Manuals; www.merck.com/mmhe/sec11/ch147/ch147a.html).

- **Impaired intellect**. Around 22 per cent of those aged 71 years and over have some degree of cognitive impairment, which is often seen as a forerunner of dementia and Alzheimer's disease (Ann Intern Med, 2008; 148: 427–34). However, researchers at the Mayo Clinic reckon that this figure could be lower, with only 12 per cent of individuals aged between 70 and 89 years displaying such symptoms, according to a study funded by the National Institute on Aging (www.mayoclinic.org/news2006-rst/3306.html).

The Mayo researchers define cognitive impairment as having problems with remembering words, or placing things in time and space, or finding it more difficult to make decisions, or suffering from short-term memory loss. The problem doubles in those aged between 80 and 89, and it also appears to affect more people who have received only basic levels of education.

However, although ageing is a genuine factor in cognitive decline, pharmaceuticals also play a role in accelerating the problem. Scientists from the University of Florida have demonstrated that any drug that has anticholinergic (nerve-blocking) qualities can speed cognitive decline in the elderly patient. Although this includes drugs known to have anticholinergic actions, such as those used to treat an overactive bladder, many other drugs have similar effects, but have not been listed as such in the literature (Presentation at the American Academy of Neurology 60th Annual Meeting, Chicago, IL, Abstract S51.001, 17 April 2008).

Indeed, it appears that almost every commonly prescribed or over-the-counter drug can produce symptoms that resemble dementia in the elderly. A pair of researchers from the Medical University of South Carolina have reported that many drugs cause side-effects that include confusion and memory loss, two signs of cognitive impairment. In addition, the problem can be magnified when the patient is taking more than one drug at the same time (J R Soc Med, 2000; 93: 457–62).

Polypharmacy

It's evident that the dangers to the elderly of taking any one drug are bad enough, but it's almost impossible to measure the negative impact of taking many drugs at the same time, as most elderly patients are doing.

Even young Hollywood celebrities, such as Heath Ledger and Brittany Murphy, may have died as a result of taking several pharmaceuticals at the same time. As Dr Bruce Goldberger, a professor of toxicology at the University of Florida's College of Medicine, said of the latter's death, "Mixing a number of these drugs could have resulted in her death".

Every drug is tested for its safety and efficacy on its own, but no one is testing the lethal cocktails that can result from taking several powerful chemical compounds in combination.

Nevertheless, what is clear, as pharmacologists at the University of Wales College of Medicine, Cardiff, have pointed out, is that drugs—and especially polypharmacy—may be responsible for many of the common health problems seen among the elderly, such as confusion, weakness, incontinence, depression and falls, all of which have been blamed on growing old (Drugs Aging, 1998; 12: 485–94).

Without drugs and medicine, perhaps getting older wouldn't be such an unhealthy rite of passage.

Bryan Hubbard

The damaged brain: How drugs cause dementia

New evidence shows that all forms of dementia may largely be caused by most of the drugs we take as we get older

We may be living longer and better than ever but, by the time we reach our golden years, the chances of us having any awareness of it are rapidly fading. Dementia of all varieties, including Alzheimer's disease, is now epidemic, approaching an incidence similar to that of the major killers: for instance, seven million Americans aged over 65 are currently diagnosed with dementia compared with 10 million of all ages with cancer.

Furthermore, the incidence rates of dementia are sharply on the increase. Predictions suggest that the disorder will increase fourfold among the elderly within the next 40 years, and escalate across all age groups.

Nevertheless, the raw statistics fail to reveal the most insidious aspect of the disease—namely, that the chances that any one of us will suffer from dementia sharply increases with every decade. According to one team of researchers, who tracked the incidence of the disorder over time, after the age of 60, your risk of developing dementia doubles every five years. This means that, by the time you reach your mid-80s, you face a one-in-four risk of having dementia and, by the time you reach 90, the odds increase to one chance in three (Alzheimer Dis Assoc Dis, 2003; 17: 63–7).

One reason for the prevalence is simply due to the ambiguous nature of the term. Virtually every form of cognitive decline is now classified as dementia, including memory loss, and impairment in planning, judgment, reasoning and ordinary thought processes.

However, modern medicine must take the greatest blame for the mental decline seen among the elderly. Even though they represent only one-seventh of the population, the over-65s take one-third of all prescription drugs—and usually a cocktail of them. The average senior is taking six drugs at a time, many of which affect the brain.

Evidence is emerging that a large coterie of drugs given for other conditions, such as high cholesterol, depression, inflammation, insomnia, anxiety, heart disease and arthritis—in short, most of the drugs given to us as we grow older—can all bring on dementia.

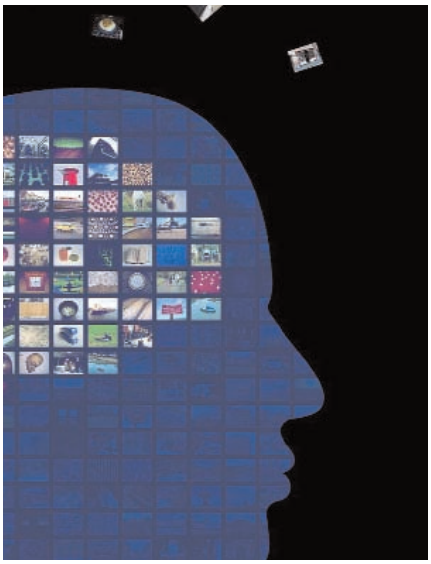
Many of these drugs cause actual damage to the structure of the brain, including shrinking brain volume and destroying the crucial fatty structures of brain cells, thus leading to the abnormal accumulation of tissue in vital brain structures.

Given the fact that some 90 per cent of Americans from their mid-50s onwards are taking at least one drug regularly and nearly one-third are taking five or more drugs, it may well be—as American psychiatrist Grace E. Jackson claims in her brilliant and damning self-published study—that dementia is, in many cases, a drug-induced disease (Jackson GE. *Drug-Induced Dementia*. AuthorHouse, 2009). Of the 36 million Americans who now take statins, for instance, an estimated 162,000 people could be severely cognitively impaired because of these drugs in the US alone.

The chicken or egg dilemma

Although used as an all-purpose catch phrase, dementia could be defined as any condition in which there is an observable abnormality involving neurons or glial cells. On the basis of such a description, it could be said that there are four types of true dementia:

- **Lewy body dementia**, in which patients have movement disorders much like those seen in Parkinson's disease, with abnormal deposits of 'Lewy body' proteins, named after the German neurologist who first observed them, throughout the neurons of the brain;



- **vascular dementia**, where the brain's blood supply has become cut off or interrupted, usually as a result of large or small strokes, causing the death of neurons;
- **frontotemporal dementia**, usually diagnosed in patients aged under 65, where the frontal or temporal lobes (including the hippocampus) of the brain shrink; and
- **Alzheimer's disease**.

According to Jackson, Alzheimer's victims all share three specific abnormalities:

- **senile plaques**, abnormal clumps of amyloid and other sorts of proteins that form outside of cells in the gray matter of the brain;
- **neurofibrillary tangles**, abnormal, twisted bundles of fibres within brain neurons mostly made up of tau proteins that impair the formation of tubulin, a protein necessary for healthy connective nerve tissue, the result of which is that messages in the brain aren't transmitted properly; and
- **granulovacuolar degeneration** (GVD), where neurons in the brain have abnormal 'holes' (vacuoles), each of which contains a small, dense protein.

The problem is that researchers cannot agree on whether these characteristic findings are the cause or the effect of an abnormal process in the body. The

Why the brain needs fats

Although medicine used to believe that the master conductor of brain activity was the neurons, or nerve cells, that create and release neurotransmitters and electrical signals, this view has been revised by a more holistic view of the brain. This concept appreciates that the brain works in its entirety through a web of activity between neurons and four varieties of glial cells. Glial cells, which surround the neurons, keep them in place, provide them with nutrients, and destroy and mop up pathogens. They also insulate one neuron from another and modulate the transmission of signals. One major function of glial cells is to form myelin—the insulating sheath that covers every tentacle of a nerve cell—which is largely made up of lipid (fatty) tissue.

Yeon-Kyun Shin, a professor of biophysics at Iowa State University, recently went on record to say that high cholesterol is vital for good brain function, and a lack of cholesterol impairs the brain's thinking ability and memory.

"If you deprive the brain of cholesterol, then you directly affect the machinery that triggers the release of neurotransmitters. Neurotransmitters affect the data-processing and memory functions; in other words, how smart you are and how well you remember things," he said (Proc Natl Acad Sci USA, 2009; 106: 5141–6).

evidence from studies of the effects of aluminium and mercury on the brain suggest that they are the result of toxicity rather than being a true cause of dementia. In test-tube studies using human brain cells, for instance, minute doses of mercury produced changes identical to those seen in Alzheimer's disease (J Neurochem, 2000; 74: 231–6).

In other, animal studies, professor of medical biochemistry Boyd Haley and colleagues at the University of Kentucky fed rats aluminium, but observed no changes in tubulin levels, whereas mercury-fed rats displayed diminished tubulin levels similar to those seen in typical Alzheimer's patients. Furthermore, some researchers suggest that the vacuoles seen in GVD are filled with toxic materials, such as aluminium, which the neuron has 'fenced in' to keep the rest of the brain safe (Am J Pathol, 1999; 155: 1163–72).

Research at the University of Pittsburgh has discovered that a fat-binding agent known as 'apolipo-protein D' (or apoD in medico-speak) is present in the brain plaques seen in patients with Alzheimer's disease.

However, apoD is also found in other kinds of pathology, and many animal and human studies have shown that antipsychotic drugs induce the production of apoD. Furthermore, scientists have found this type of lipoprotein in fat and brain cells following injury, which suggests that it plays a role in cell renewal and repair.

Nevertheless, the evidence in general suggests that toxicity—a form of insult to the brain—rather than natural cellular degeneration is the cause of dementia.

Antidepressants

There is no doubt that certain drugs cause damage to the structure of the brain, thereby impairing its function. Among the chief offenders are antidepressants, which appear to target the white matter of the brain.

The white matter is the part of the brain that contains bundles of nerve fibres covered with myelin, a white fatty substance that forms an insu-lating sheath around each fibre. It is through these neural bundles that messages are passed between different areas of gray matter, which is made up of unmyelinated nerve cells, within the nervous system.

This means that the white matter is rather like a telephone network, responsible for the rapid transmission of nerve impulses and cell-to-cell communication.

One natural aspect of ageing is losing neural connections. Each of us begins adulthood with some 176,000 km of white matter, but we all should expect to lose around 10 per cent of these connections with every decade of life.

However, antidepressants clearly hasten this process. In 2008, a US study carried out by Duke University Medical Center in Durham, NC, published the results of the 10-year study, which examined the magnetic resonance imaging (MRI) scans of more than 1800 patients performed over two periods of time—first, from 1991 to 1994, and then, from 1997 to 1999. The authors compared the findings for the 163 patients who had begun taking antidepressants between the first and second scans with those for patients who were not using any such drugs.

They found similar incidences between the two groups of virtually every condition looked at—diabetes, stroke, heart attacks, hypertension—save one. Those taking the anti-depressant drugs experienced more 'bright spots' in the white matter on MRI. This sort of appearance on a scan is thought to indicate damage to blood vessels, impaired blood flow, demyelination (degeneration of the myelin sheath of nerve cells), disintegration of the blood–brain barrier and even damage to the nerve cells in the gray matter of the brain (Stroke, 2008; 39: 857–62).

Indeed, those taking the drugs suffered a 36-per-cent incidence of white-matter damage compared with 27 per cent in those who had been drug-free over the decade. In addition, all types of antidepressants —be they old or new—hastened the decline. What's more, although the worst offenders were the old-style tricyclic antidepressants, adverse effects were observed with all of the newer types of drugs that inhibit the uptake of serotonin. Overall, 60 per cent of the patients who used either type of antidepressants showed increased damage to white matter that was above the norm.

Antidepressants also appear to shrink the hippocampus, part of the limbic system of the brain that is involved in long-term memory, spatial navigation, learning and mood. There is solid scientific evidence that patients who are chronic users of antidepressants, particularly SSRIs (selective serotonin reuptake inhibitors) such as Prozac (fluoxetine),

have smaller hippocampal structures compared with controls.

In one fascinating study, the hippocampi of long-time depressed patients who were taking long-term medication were compared with those of two other patient groups: those who'd just been diagnosed with the illness and had not yet begun taking any drugs; and a group of non-depressed controls. There was no difference in brain size between the just-diagnosed depressed patients and the controls, whereas the long-time medicated showed a hippocampus that was 12 per cent smaller, on average, than those of the others (Proc Natl Acad Sci USA, 2003; 100: 1387–92).

The importance of this study is that it demonstrates that it's the drugs themselves, and not the depression on its own, that causes the brain to shrink.

Autopsies of cavaders have also revealed brain damage in those who'd been long-term users of anti-depressants. In one Dutch study, of patients who'd been taking anti-depressants, 73 per cent showed evidence of brain cell death (apoptosis) compared with 33 per cent in patients using long-term steroids and 6 per cent of their matched controls (Am J Pathol, 2001; 158: 453–68).

In addition, epidemiological studies have also shown a greater incidence of dementia among populations using antidepressants. Researchers in Copenhagen carried out a sweeping study, involving nearly a third of the entire Danish population, focused on patients aged over 40 who'd taken antidepressants, even if just one single time. The risk of developing dementia was two to five times higher in those who'd used antidepressants compared with non-users (J Affect Disord, 2009: 117: 24–9).

However, Jackson believes that this population study may have underestimated the risk, as one-fifth of those using antidepressants died during the study follow-up period.

Statins

Statins, those 'miracle' cholesterol-lowering drugs, also appear to lead to progressive cognitive decline. This is particularly ironic, as medicine has been under the illusion that cutting down on cholesterol in the elderly is desirable particularly for the brain (see box, page 42) and, consequently, that statins can keep Alzheimer's at bay.

It is true that statins can cross the blood–brain barrier and alter cholesterol metabolism in the brain, but this has no bearing on Alzheimer's disease. Earlier this year, Harvard University researchers, in conjunction with a number of other centres in the US, Germany and Spain, gave statins for 12 weeks to patients with mild Alzheimer's disease or mild memory loss.

During the course of this study, they found a "modest but significant" inhibition of brain cholesterol biosynthesis. Nevertheless, although the drugs lowered cholesterol in the brain, this had no alleviating effects on Alzheimer's disease whatsoever (Alzheimer Dis Assoc Disord, 2010 May 13 doi: 10.1097/WAD.0b013e3181d61fea).

In fact, the lack of effectiveness of statins as a treatment for Alzheimer's disease was finally established last year, when two reviewers independently analyzed two large-scale randomized controlled trials, involving a total of 26,340 patients as part of the HPS 2002 and PROSPER 2002 studies, and reached an agreement after discussing their results. Their conclusion was that statins given late in life to individuals at risk of vascular disease have no effect in preventing either Alzheimer's disease or dementia (Cochrane Database Syst Rev, 2009; 2: CD003160).

Indeed, far from helping memory and cognitive function, statins can cause sudden and complete memory loss.

This evidence emerged when flight surgeon Duane Graveline suffered global amnesia when taking atorva-statin (Lipitor) for the first time. When his family doctor Jay S. Cohen took up his case with Pfizer, the drug's manufacturer, he was sent clinical evidence gathered before the drug's release showing that there were 4.5 cases of severe cognitive disturbance out of every 100 patients given the drug.

This included cases of impaired, worsening or general lapses of memory, general forgetfulness and short-term memory loss. Pfizer's own studies had also discovered instances in which patients had difficulty concentrating, or suffered abnormally

The role of coenzyme Q10

It is well known that patients taking statins lose coenzyme Q10 (CoQ10) in a dose-related manner. The drugs block production of both cholesterol and CoQ10 by inhibiting the enzyme precursor of not only cholesterol, but also that of CoQ10.

CoQ10 participates in chemical reactions, particularly those that involve cellular energy production, and helps to make cell membranes more resistant to oxygen damage. It is found abundantly in the heart mostly because of the huge energy requirements of cardiac cells.

In fact, studies have shown that a deficiency of CoQ10 is linked with heart failure and impaired heart function. Of 15 published studies in the literature, nine have confirmed that statins can significantly lower CoQ10 levels (Arzneim Forsch, 1999; 49: 324–9).

Critics of statins believe that the widespread use of these drugs has caused an increase in 'statin cardiomyopathy', where the heart loses its ability to pump blood or heart rhythm is disturbed, leading to irregular heartbeats. Drug manufacturers tacitly acknowledge this effect by offering several drug formulations that combine a statin with CoQ10.

But a less well-known problem with blocking CoQ10 is that it interferes with cognitive performance, resulting in memory loss and muddled thinking. In an elderly person, this sort of side-effect is almost invariably passed off as age-related dementia, requiring yet another coterie of 'wonder drugs'.

Other researchers have been discovering that statins also inhibit and cause mutations in mitrochondria cells, the energy power packs of the body. Scientists now suspect that an array of neurodegenerative diseases are due to mutated or altered mitochondria.

slow or difficulty in thinking, slowed or decreased mental activity, impaired intellect or judgment and even irrational thinking.

Graveline and Cohen then conducted an online literature search of MedWatch, the US Food and Drug Association (FDA) database of reported drug side-effects, for reports of severe cognitive impairment or serious amnesia associated with Lipitor. They found 662 such reports, including 399 cases of amnesia and 236 cases of memory impairment. The investigating pair also found that, over time, the complaints had become more frequent (Townsend Lett Docs, 2009; 311: 64–70).

MedWatch, which is thought to be notified of only 2.5 to 5 per cent of all drug side-effects, therefore suffers from vast underreporting, so the true incidence of memory problems from statins is probably closer to 66,000 or more. In fact, Graveline and Cohen believe that virtually every patient taking the drug suffers from cognitive damage in one form or another that may be too mild to be initially detected. This is perhaps because all statins lower levels of coenzyme Q10, known to be vital for brain function (see box, page 43).

Graveline and Cohen's detective work has also been vindicated by a meta-analysis carried out by Duke University in Durham, NC, which uncovered 60 patients with memory loss attributable to statins. Half the patients had noticed cognitive decline within just two months of starting the drugs; of these, more than half found that their memory improved as soon as they stopped taking the drugs. Furthermore, all of the four patients who started taking the drugs again suffered a recurrence of their memory problems.

As a further nail in the coffin, not one single experimental study could find any evidence to support any benefit with statins in delaying cognitive decline (Pharmacotherapy, 2003; 23: 871–80).

Antipsychotic agents

Another major culprit causing dementia is that broad class of drugs called 'antipsychotics'. The first so-called 'neuroleptic' medications were developed in the 1950s to relieve patients suffering from hallucinations, paranoid schizophrenia and other psychoses. Unfortunately, these drugs brought with them unwanted extrapyramidal (brain motor system) side-effects such as tardive dyskinesia, characterized by muscle stiffness, tics, tremors and other awkward movements.

The arrival of clozapine (Clozaril) introduced the next generation of neuroleptics—which also includes olanzapine (Zyprexa), quetiapine (Seroquel) and risperidone (Risperdal)—that were dubbed 'atypical antipsychotics' to distinguish them from their older and supposedly more dangerous cousins. These drugs are supposed to suppress the psychotic and antisocial aspects of schizophrenia without all the antipyramidal effects (World J Biol Psychiatry, 2000; 1: 204–14).

However, this newer generation of drugs comes with its own laundry list of dangerous, even life-threatening, side-effects, including serious mental deterioration.

There is no doubt that the so-called 'antipsychotic drugs' can cause or speed up the development of dementia (J Neurol Neurosurg Psychiatry, 2007; 78: 233–9). This is ironic because these drugs are often given to sedate or calm patients with dementia, whereas it appears that they are also speeding up and worsening the process of cognitive decline.

Nevertheless, the most compelling evidence comes from autopsy studies that have compared patients using antipsychotic drugs with those who did not. In one study by the Wolfson Centre for Age-Related Diseases at King's College London, those who'd been given neuroleptics had a 30-per-cent greater density of amyloid plaques and 65- to 367-per-cent more neurofibrillary tangles than those who were free of neuroleptic drugs (Int J Geriatr Psychiatry, 2005; 20: 872–5).

Moreover, in a similar US study, 102 patients with schizophrenia showed evidence on autopsy of brain

Other drugs to avoid

Besides the agents already mentioned (see main story), there is mounting evidence that you should also avoid the following types of drugs.

- **Stimulants**, such as methylphenidate (Ritalin). A vast number of animal studies show that all amphetamines cause parts of the brain to shrink, trigger degeneration of dopamine cells in the brain and reduce the survival of neurons in the hippocampus, among many other effects (Jackson GE. *Drug-Induced Dementia*. AuthorHouse, 2009).
- **Beta-blockers, calcium-channel blockers and ACE inhibitors**, and other drugs that aggressively lower blood pressure. These antihypertensives can lower blood flow to the brain and produce all the hallmarks of Alzheimer's disease (Drugs Aging, 1999; 15: 15–28).
- **Antiarrhythmic drugs**, given to correct abnormal heart rhythms, can also cause dementia.
- **Anticholinergic drugs**, a giant category that includes drugs for gastro-intestinal problems such as diverticulitis and ulcerative colitis, respiratory ailments such as asthma and genitourinary disorders such as cystitis or prostatitis, are associated with cognitive decline in the elderly (Presentation at the American Academy of Neurology 60th Anniversary Annual Meeting in Chicago, IL, 17 April 2008).
- **Opioids**, many of which can cause delirium (Drugs Aging, 1993; 3: 349–57; Postgrad Med J, 2004; 80: 388–93).
- **Non-steroidal anti-inflammatory drugs** (NSAIDs). Initially thought to offer protection against Alzheimer's, different types of these common painkillers can cause a variety of cognitive changes, ranging from delirium (indomethacin and sulindac) to disturbances in memory and concentration (naproxen and ibuprofen) (Drugs Aging, 1999; 15: 15–28).
- **Levodopa**. This antiparkinsonian medication causes cognitive symptoms in up to 60 per cent of users (Clin Geriatr Med, 1998; 14: 101–27).

deterioration that was suggestive of Alzheimer's or some other form of dementia. The signs were present in 74 per cent of those who'd been given antipsychotic drugs, but in only 36 per cent of those who'd died prior to the advent of these drugs (Alzheimer Dis Assoc Disord, 1994; 8: 211–27).

In other words, taking antipsychotics more than doubled the patients' chances of developing dementia.

The worst combination of all is taking an antipsychotic together with an antidepressant, which appears to quadruple the speed at which the disease develops (J Neurol Neurosurg Psychiatry, 2007; 78: 233–9).

What's more, Alzheimer's patients who are given an antipsychotic—a common practice, usually to sedate them—have double the usual risk of death. In a major study involving various centres across the UK—the first independent study of its kind not paid for by a drug company—less than half the patients (46 per cent) taking an antipsychotic were still alive at the two-year follow-up. After three years, only 30 per cent of those taking antipsychotics were alive compared with 59 per cent taking a placebo (Lancet Neurol, 2009; 8: 151–7; doi: 10.1016/S1474-4422(08)70295-3).

In another UK study that was focused on care facilities in the North East of England, the London-based researchers compared the efficacy of the antipsychotic quetiapine, the cholinesterase inhibitor rivastigmine and a placebo in calming institutionalized patients with dementia. Neither of the patients in the active-treatment groups were any calmer than those who were simply taking sugar pills, although there was one significant difference with quetiapine—it was associated with significantly greater cognitive decline (BMJ, 2005; 330: 874).

Antipsychotics also appear to shrink the volume of the frontal lobes of the brain by 0.2 per cent per year, according to a University of Iowa study (Arch Gen Psychiatry, 2003; 60: 585–94).

Other studies have shown size reductions in a variety of areas of the brain with both older (haloperidol) and newer (olanzapine) antipsychotic medications (Arch Gen Psychiatry, 2003; 60: 585–94).

What is coming to light is a clear association between antipsychotic drugs and cognitive decline. In a painstaking English survey, every case of dementia recorded on a dementia register during 1993–1994 was examined. Researchers studied the patients' diagnoses and treatments from their various medical carers and interviewed their next of kin, then matched these patients to a similar group of elderly people living in Southeast London.

Of the patients on the register, 13 per cent had a past history of psychiatric treatment, and the use of psychiatric drugs was three to four times higher among those who'd gone on to develop dementia (Age Ageing, 1998; 27: 181–8).

Benzodiazepines

In addition to the major antipsychotic drugs, benzodiazepine tranquillizers and sleeping pills are also responsible for cognitive decline. One Argentinian study noted evidence that sleeping pills, which are often handed out without a prescription in that country, led to severe memory and cognitive impairment, and delirium (Vertex, 2001; 12: 272–5).

Newer studies now show that this effect has to do with an effect on gangliosides in the brain. These molecules, which contain fat and sugar, are present to a large extent in brain lipids and on the surface of every neuron. They are essential for regulating cell growth, maintaining the integrity of the material contained within cells, and responding to foreign invasion by toxins and bacteria. Without these gangliosides, we lose myelin and entire neurons, and may even die.

A series of studies by the Institute for Medical Research in Belgrade, Yugoslavia, has demonstrated that, at least in rats—so it may not apply to humans—chronic doses of Valium (diazepam) led to the loss of 46 per cent of gangliosides in the cerebellum within six months. After a short period of total drug withdrawal, the rat brain still had not fully recovered (Physiol Res, 1999; 48: 143–8).

When the researchers repeated the study in 2002, focusing especially on the effects of the drug on various regions of the brain, they found significant reductions of gangliosides in the hippocampus, cerebral cortex and cerebellum, as well as increases of simple gangliosides in other areas (Neurol Sci, 2002; 23: 69–74). These findings are consistent with those of many human neurological diseases, including Alzheimer's.

Indeed, in a 1993 British study, researchers took computed tomography (CT) brain scans of long-term benzodiazepine users and compared them with those of drug-free controls. The scans revealed that the drug users had a reduction of brain tissue in their frontal and occipital lobes, as well as in the left caudate nucleus—areas that are crucial for cognitive function (Psychiatry Res, 1993; 48: 135–44).

At present, in the US alone, six million patients are being treated for dementia at a cost of $90 billion, or one-third of all Medicare bills. This means that 1 per cent of the entire gross domestic product of the US is being spent on a mostly iatrogenic (doctor-induced) condition.

Medicine has reached the point where it is chasing its own tail, attempting to mop up with yet more drugs and treatments a vast and costly problem that it has itself caused in the first place.

Evidence is mounting that one of the major toxic insults to the brain is the mercury from amalgam fillings, but this effect may be eclipsed by the dangers we face from the modern medical response to ageing.

Keeping bright and alert in old age requires a few simple practices: regular exercise; eating an antioxidant-rich wholefood diet along with good fats; minimizing toxic exposure to heavy metals; engaging in regular brain workouts (crossword puzzles or reading); and staying connected through a social network. However, now there is one more simple homily to add to the list: avoid as many prescription drugs as you can.

Lynne McTaggart

HRT: the latest cancer risk

Just when it looks as if it couldn't get any worse for HRT, a new study shows that it causes lung cancer

Anyone with shares in hormone replacement therapy (HRT) is in the doldrums. HRT was touted as the modern-day equivalent of the fountain of youth—until 2002, when the US Women's Health Initiative (WHI) study, one of the largest-ever studies of these drugs, discovered that women taking HRT were more likely to have breast cancer, ovarian cancer, stroke and heart disease.

And now, new evidence from the WHI data shows that the use of HRT during the menopause increases lung cancer by 60 per cent after five years.

Dr Rowan Chlebowski, a medical oncologist at Harbor-UCLA Medical Center in Los Angeles, CA, analyzed the WHI statistics, and found a link between prolonged HRT use and non-small cell (NSC) lung cancer.

Although most of the adverse publicity after the WHI study focused on the breast cancer risk, this is the first time that a link between HRT and lung cancer has been identified. It also happens that NSC lung cancer is the leading cause of death in women.

The most vulnerable women were smokers using HRT, who accounted for slightly more than half of all cases of NSC lung cancer. In the WHI study, the researchers found one extra death from this for every 100 women using Prempro, Wyeth's combination oestrogen–progestin HRT drug.

Ovarian cancer risk

This announcement, made at the annual meeting of the American Society of Clinical Oncology in May, coincided with the release of the findings from a large-scale, long-term Danish study showing that women who take HRT increase their risk of ovarian cancer by 38 per cent (JAMA, 2009; 302: 298–305).

The study, which included more than 900,000 women aged 50–79, found that the current use of hormones accounted for a 38-per-cent greater chance of ovarian cancer. This translates to one extra case every year for every 8300 women using HRT, or 140 extra cases during the eight-year follow-up, accounting for 5 per cent of all cases of the cancer.

The risk of having the disease were the same regardless of how long the drug was taken, which formulation was used, how much oestrogen was included in the mix and what sort of delivery method was chosen.

As ovarian cancer is difficult to detect and is often fatal, such a risk should not be brushed aside, the Copenhagen researchers noted.

HRT to blame

Up to now, medicine has tended to blame a familial 'predisposition'—that is, a family history of breast cancer and the presence of certain predisposing genes—on many incidences of breast cancer, particularly in women using HRT. Nevertheless, the latest trawl through the WHI data shows that HRT shoulders much of the blame.

Epidemiologists from the University of Rochester Medical Center, in New York, followed-up the more than 16,000 postmenopausal women given either HRT or a placebo during the five-year WHI study, which was abruptly abandoned when the health risks emerged. When they evaluated the 349 women who'd developed breast cancer, they could find no link with a family history.

The researchers concluded that a family history and hormones have "independent and non-interacting effects"—in other words, the cases of cancer were most likely entirely caused by HRT (Epidemiology, 2009; 15 May: doi: 10.1097/EDE. 0b013e3181a71279).

Fewer cases after the facts

Following the disclosures of the WHI trial, breast cancer rates fell by 13 per cent among more affluent women in California, but only by 7 per cent in rural areas. When researchers from the Northern California Cancer Center investigated the discrepancy, they found that women in the more rural areas had not heard about the link between HRT and cancer, and so had continued taking the drugs—which would again tend to point the finger at HRT (BMC Med, 2009; in press).

After the WHI results were reported, Wyeth's products took a hammering, and their use declined by 50 per cent. Nevertheless, sales of the oestrogen-only drug Premarin and various cream formulations still constitute a business totalling up to one billion dollars per year.

Last month, the US Food and Drug Administration added a final nail in the HRT coffin with a boxed warning for Prometrium, a progesterone drug, warning of the WHI's results showing increased risks of heart attack, stroke, invasive breast cancer, blood clots and deep vein thrombosis, and a greater risk of dementia, among those taking combined oestrogens and progestogens of any variety.

Lynne McTaggart

The current risks of HRT

According to the US Women's Health Initiative study, hormone therapy increases your chances of developing the following diseases by these percentages:

- Coronary heart disease-related events, 29 per cent
- Stroke, 41 per cent
- Deep vein thrombosis, 200 per cent
- Blood clot in lungs, more than 200 per cent
- Invasive breast cancer, 24 per cent
- All cancers among previous users, 86 per cent
- Ovarian cancer, 38 per cent
- Lung cancer, 60 per cent.

The big 'cure-all' unmasked

The great all-purpose preventative for cholesterol has been found to cause prostate cancer

Statins, the gold standard of cholesterol treatment, have become the liver tonic of the modern age. Doctors hand them out for everything from osteoporosis to senile dementia, as early studies suggest that they may have preventative effects.

When early laboratory evidence showed that statins induce apoptosis (cell death) and reduce prostate cancer cell growth and spreading (Cancer Epidemiol Biomarkers Prev, 2008; 17: 88–94), medicine concluded that statins were a potent cancer preventative (J Natl Cancer Inst, 2006; 98: 1819–25). Early studies bolstered this view, offering preliminary evidence that long-term statin use could prevent cancers of the breast, prostate, blood and colon.

Studies of populations with prostate cancer found links between statins and the prevention of more advanced forms of the disease (Curr Opin Urol, 2008; 18: 333–9). Doctors were particularly enthusiastic when a case-control study found a cancer risk reduction of 20 per cent with statins (J Clin Oncol, 2004; 22: 2388–94).

Now, that reputation—that statins are a powerful cancer preventative—has been sullied with the latest findings that statins may actually cause prostate cancer in overweight men. Researchers at the Fred Hutchinson Cancer Research Center in Seattle, WA, discovered the link while studying more than 1000 cases of prostate cancer, diagnosed between 2002–2005, compared with a similar number of age-matched controls.

Although there was no risk of developing the disease in normal-weight statin users, including those who had taken the drugs for more than 10 years, overweight men— those with a body mass index of more than 30 kg/m^2—increased their risk by 1.5 times over overweight non-users. Also, the risk increased to 1.8 times with statin use for five or more years (Am J Epidemiol, 2008; 168: 250–60).

But the Seattle evidence is only the latest to challenge the myth of cancer prevention. A University of Athens review of 19 epidemiological studies found no evidence of a protective effect with statins and even suggested that the earlier evidence of lowered risk was just coincidental (Int J Cancer, 2008; 123: 899–904).

More detailed analyses found a lower incidence of advanced cancer, but no reduction in the risk of overall prostate cancer (Curr Opin Urol, 2008; 18: 333–9). An even larger meta-analysis of 35 randomized controlled trials showed a link between the drug and developing cancer, depending on the patient's age: the older the man, the more likely he was to develop cancer (J Clin Oncol, 2006; 24: 4808–17).

Another meta-analysis of pravastatin in elderly patients confirmed an association between the drug and an increased risk of cancer with increasing age (CMAJ, 2007; 176: 649–54).

As for other forms of cancer, the Department of Pharmacology team at the University of Athens School of Medicine has systematically examined and combined all the evidence of statin use and cancer incidence. Their review of 14 studies found no evidence to support claims that statin use can protect against malignancies of the blood, such as leukaemia (Br J Clin Pharmacol, 2007; 64: 255–62).

They also found no evidence that statins can significantly reduce the risk of colorectal cancer, although they concluded that there might be some effect with higher doses (J Clin Oncol, 2007; 25: 3462–8). The same methodology also found no evidence that statins can reduce the risk of either pancreatic or breast cancers (Am J Gastroenterol, 2008; 103: 2646–51; J Clin Oncol, 2005; 23: 8606–12).

Yet another research team, from the Department of Epidemiology and Survellance Research at the American Cancer Society, found that neither short- or long-term (five years or more) use of the drug prevented colorectal cancer (J Natl Cancer Inst, 2006; 98: 69–72).

Similarly, a Boston University study of more than 3600 patients in Massachusetts could also find no protective effect other than a lower risk of stage IV (advanced, metastasizing) cancer among statin users, an association that the authors believe requires confirmation (J Natl Cancer Inst, 2007; 99: 32–40).

The final blow was dealt by a University of Connecticut School of Pharmacy meta-analyses of nearly 90,000 participants involved in all studies claiming a protective effect against all cancers. Again, the data indicate that no type of cancer is affected by statin use (JAMA, 2006; 295: 74–80).

Aside from the damning evidence against its role as a cancer preventative, the latest evidence also shows that statins don't prevent fractures or type 2 diabetes either (Pharmacoepidemiol Drug Saf, 2007; 16: 627–40; Curr Med Res Opin, 2008; 24: 1359–62). This kicks away several platforms on which the drug's reputation as a preventative treatment has been based.

Lynne McTaggart

Take as little as possible

If you must take a statin drug, take the smallest dose possible. New evidence shows that even the minimum daily dose of 20 mg/day causes potentially fatal muscle pain and weakness—and may even lead to complete breakdown of muscle tissue (N Engl J Med, 2008; 359: 789–99). Higher doses or taking the drug with other drugs had a magnifying effect, causing a far higher incidence of myopathy than the official figures claim. The latest evidence shows that as much as 1.6 per cent of patients develop muscle weakness—that's 128,000 patients in the US alone.

Building brittle bones

Atrial fibrillation is the latest in a long line of side-effects caused by Fosamax and its relatives

The world's most popular drugs for osteoporosis cause heart problems—and we're only just finding out 10 years after the manufacturers, who may have known it all along.

A recent study with the tortuous acronym HORIZON (Health Outcomes and Reduced Incidence With Zoledronic Acid Once Yearly) found that postmenopausal women taking zoledronic acid (Acasta) by injection just once a year reported higher bone density, but also higher rates of serious atrial fibrillation (AF), than those taking a placebo (N Engl J Med, 2007; 356: 1809–22). Acasta is a bisphosphonate like Fosamax, which reduces the turnover of bone.

That discovery was followed this year by a University of Washington study looking at the use of aledronate sodium (Fosamax) among female patients at an integrated healthcare delivery system between 1 October 2001 and 31 December 2004. When 719 women with confirmed AF were compared with 966 controls without AF, a significantly higher number of those with AF had used alendronate. In fact, using alendronate at any point in their lives nearly doubled their risk of AF. The researchers estimated that this class of drugs may cause up to 3 per cent of the AF in postmenopausal women with osteoporosis (Arch Intern Med, 2008; 168: 826–31).

But not all studies agree. A Danish epidemiological study examined 13,586 patients with AF and atrial flutter, and found that etidronate and alendronate were both used to almost the same degree by those with and without AF. Their conclusion: "No evidence was found that use of bisphosphonates increases the risk of atrial fibrillation and flutter" (BMJ, 2008; 336: 813–6).

Nevertheless, according to some law firms, the association between bisphosphonates and heart problems emerged 10 years ago, when evidence of AF with Fosamax was spotted on reviewing the evidence of a five-year study (1992–1997) called the Fracture Intervention Trial. Its findings showed a 50-percent higher risk of AF for those taking Fosamax compared with taking a placebo (JAMA, 1998; 280: 2077–82).

However, this is only the latest in a litany of problems with what was first touted to be the solution to osteoporosis and other bone diseases such as Paget's disease of the bone. Fosamax and the other bisphosphonates work by limiting the normal tearing down of old bone, part of the dynamic process of constant renewal in bone growth.

Worse bone problems

In the 12 years since its launch on the world market, Fosamax and its relatives have been the target of a number of lawsuits for causing osteonecrosis of the jaw, or 'dead jaw'. This condition is characterized by a permanent loss of blood to bone tissue.

This severe condition causes extreme pain, numbness, loosening of the teeth and exposure of bone in the oral cavity. When allowed to go undetected and untreated, this situation can lead to death of bone tissue and even collapse of the bone within the jaw—a condition that is irreversible. In this case, the exposed bone can eventually lead to infection and fracture, and may require long-term antibiotic therapy or surgery to remove the dead and dying bone tissue.

In fact, Merck & Co, the manufacturers of Fosamax, have even been the subject of a number of personal-injury lawsuits filed by women who have developed osteonecrosis of the jaw while taking the drug.

Other side-effects

Before these latest disclosures, the biggest concern with this class of drugs was the potential for gastrointestinal damage. Soon after the launch of Fosamax, patients com-

plained of stomach pain, heartburn and irritation of the oesophagus. In severe instances, the drug caused fatal oesophageal perforation (Am J Gastroenterol, 2001; 96: 3212–3).

Patients were advised to take the drug with a large glass of water while standing up—and had to remain standing for at least half an hour to reduce the time that the drug is in contact with the lining of the oesophagus.

In addition to damaging the oesophagus, these drugs also cause stomach ulcers—and permanently in some cases (Dig Dis Sci, 2002; 47: 1665–78; MedGenMed, 2002; 4: 3). They can also cause severe hepatitis, skin 'poisoning' and a painful inflammation of the eye known as 'anterior uveitis' (Gastroenterol Clin Biol, 2002; 26: 179–80; Aten Prim, 2002; 29: 61–2; Invest Clin, 2002; 43: 49–52).

The most astonishing aspect of the osteoporosis drug saga is that, despite all of the serious and even fatal side-effects, the drug companies have been given license to use these drugs as a long-term preventative—before a woman has developed osteoporosis. Also, doctors routinely suggest that they be taken for a decade after the menopause (Dan Med Bull, 2002; 49: 1–18), despite the lack of any evidence that they do any good.

In fact, women who take the drug for at least seven years suffer three times more fractures of the spine in the last two years of taking the drug than in the first three years. So, although the bones have greater density, they are more brittle (J Clin Endocrinol Metab, 2001; 86: 1835).

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