- **The recent discovery of how brain cells die in Alzheimer's provides a real chance of avoiding, slowing and even reversing some aspects of the disease**
- **Powerful natural anti-oxidants have a key role to play, as do ...**
- **Phospholipids, from soy and other sources, which rebuild brain cell membranes. Mineral supplements may also help**
- **Recent research indicates how nutrition plays a vital role in building intelligence in the early years**

Chapter 17

Building and maintaining a healthy brain

Good nutrition builds better brains. And nutritional depletion threatens those brains in later life. This chapter looks at both aspects – but we'll start with the older brain.

Alzheimer's

Alzheimer's is one of our most feared diseases – and in an ageing society, it is becoming ever more prevalent. It affects two in every 100 people over the age of 65. In those of us who reach our eighth decade, it will hit an alarming one in five.

The victim is, in a way, the least affected. Consciousness of the growing darkness soon fades as self-awareness recedes, but the gradual loss of faculties and the slow disintegration of the personality impose terrible burdens on the carers (typically daughters or younger sisters), and generate huge social and economic costs.

The disease is even more tragic when it occurs in otherwise healthy individuals, robbing them of their retirement, grandchildren, wisdom and memories.

Until very recently there has been no light in this darkness; no therapeutic strategem or device that could stop or slow the descent into unreason. But that is changing.

New evidence shows that the condition, though age-related, is not caused by the ageing process per se[65].

Use it or lose it

This chapter outlines a nutritional approach to defending yourself against brain degeneration.

Equally importantly, brain scientists have found that keeping mentally active – through work, hobbies and even activities like crossword puzzles – is a key element in retaining mental acuity.

As long as we keep making new **connections** between brain cells we can maintain our alertness – provided there is no underlying physical deterioration. That is where nutrition comes in.

Nutritional and preventative approaches may be able to halt the disease process – and even put it into reverse, if the illness is diagnosed and treated early enough.

Aluminium – one of the usual suspects

It's been known for over 100 years that aluminium is extremely neurotoxic. In the early days of renal dialysis, kidney patients died of brain damage caused by aluminium in the dialysing water getting into the brain.

In 1991, an MRC research group in Newcastle, England, found aluminium in the 'plaques' and 'tangles' in the brain that characterise Alzheimer's disease. And in 1994, a study of patients exposed to aluminium during renal dialysis found Alzheimer-like changes in their brains[35].

In 1992, Professor McLachlan's group in Toronto, Canada published the first account of a treatment which stabilised Alzheimer's, and slowed the otherwise rapid and irreversible decline into dementia and death. This treatment involves removing aluminium from the bodies and brains of their patients. It's a difficult and expensive chemical leaching process, unsuitable for extensive clinical use.

Aluminium and acid rain

Acid rain kills fish in rivers and lakes. Adding lime to neutralise the acidity doesn't help – because it is the aluminium washed out of the soil by the acid rain that kills the fish[116, 117].

Silicic acid poured onto the water binds to the aluminium to form sand – and prevents any further loss of life[118, 119].

The aluminium-silicon link

In nature, aluminium and silicon nearly always occur together, welded into some of the most stable, insoluble and long-lived compounds known. Clays, quartz, sand, mica and sandstone are major components of the Earth's crust. And on a building site, you'll find aluminium silicate in cement, concrete, bricks and glass. These are all safe, neutral and, importantly, **in**soluble compounds.

But although aluminium's public image is efficient, clean, cheap, safe – think of baking foil, cooking pans and double glazing - aluminium is ***not*** intrinsically safe.

Soluble forms of aluminium are highly toxic. They chew up cell membranes, degrade DNA, poison most of the enzymes

Where does your water come from?

Surface water (ie from rivers) tends to have more aluminium than silicon.

Deep water (ie from wells) tends to have more silicon.

Intake of aluminium and silicon in foods also contributes to your overall silicon/ aluminium ratio. If this is too low, consider a silicic acid supplement.

essential to cellular function; and destroy the proteins which give the cell structure and organisation.

Babies' brains at risk?

Some excellent work has been done at a UK university* with pregnant mice which were given aluminium in the drinking water[128, 129].

The newborn mice didn't thrive quite as well as a group not given aluminium. As they grew up, they scored consistently poorly on memory tests, balance and coordination – in short, they were brain damaged. And, when analysed, their brains showed specific neurochemical changes.

They were less social. Their interactions with their mothers and their litter mates were impoverished. They showed more aggression.

Aluminium – the 'lead' of the 21st century

Some eminent scientists believe that chronic accumulation of aluminium in the brain may contribute to, or accelerate, damage in the brain such as is found in Alzheimer's[120-122].

Early studies suggested that areas where the water had the highest content of soluble aluminium had the highest incidence of Alzheimer's Disease[123, 125]. Further investigation indicated that it was in areas where water was high in aluminium and low in silicon that the incidence of Alzheimer's was high. In areas where the water was silicon-rich, and aluminium-poor, Alzheimer's was relatively uncommon[124].

The evidence linking aluminium to Alzheimer's has been disputed, and it is fair to say that even if aluminium is a contributory factor, it is clearly not the only one.

But what is indisputable is that aluminium in the body causes cell damage, dysfunction and death. Aluminium in the brain causes brain damage. Furthermore, aluminium in the brain is concentrated in the senile 'plaques' and 'tangles' that are characteristic signs of Alzheimer's[126, 127].

Care of the foetus

Exposure to aluminium while still in the womb may be responsible for the type of brain damage in babies that leads to impaired intelligence and behavioural problems in later life.

Where does it come from?

Drinking water is one source of aluminium. In many areas it's added to drinking water as a clearing agent. But there are other sources. Aluminium is widely used by the food processing industry, in medicine and in cosmetics.

Although industrialists knew how potentially dangerous aluminium compounds were, they felt it was safe to use them in our food and water because they believed our defences against aluminium were good enough. They thought that aluminium compounds could not be absorbed from the gut. Unfortunately, we now know that some aluminium compounds are absorbed.

Our defences are fine at dealing with the aluminium compounds

which occur in nature, but they can't cope with some of the new aluminium compounds which industry is now producing.

Some of these are highly soluble, such as aluminium maltolate, an additive used in America by the food processing industry. So are the aluminium salts of various fatty acids, which are used in the EC. (The E number for this latter group is E470b.)

Then there is aluminium hydroxide, an ingredient used in many antacids. It's insoluble, but unfortunately the passage of aluminium through tissue barriers, such as the gut wall, is dramatically increased by the presence of other compounds such as citrates or sugars. And these are present in many foods and drinks[130].

Citrates are also used by the pharmaceutical industry to produce dispersible and effervescent medications, such as aspirin or paracetamol.

The combination of aluminium-containing antacids and citrate-containing medicines produces levels of aluminium in the blood which if sustained, lead to brain damage, dementia and death[131, 132].

The British Royal Society of Medicine responded in 1993 by recommending that aluminium-containing antacids should not be taken by pregnant women, who have a tendency to suffer from heartburn[134]. (They may have been influenced by unpublished work which found aluminium in the brains of aborted human foetuses.)

The silicon solution

At this point, you may be beginning to wonder what can be done to reduce exposure to this hidden hazard. There are several possible strategies. You could filter all your water, wash your food in filtered water, check the E numbers on all processed foods, throw out your aluminium cookware, stop using pharmaceutical products and cosmetics.

Alternatively, you could increase your zinc intake, together with Vitamin B6 which improves zinc absorption.

Some of the signs of aluminium toxicity are very similar to signs of zinc depletion. This isn't surprising because aluminium competes for zinc in the body (as well as iron, magnesium,

Daily aluminium intake

Our daily intake of aluminium is estimated to be between 10 and 30mg. It is ingested in so many forms that contact with the metal is unavoidable.

Increased aluminium intake

In 1993, Professor James Edwardson's group published the results of a series of crucial experiments[133].

Radioactively labelled aluminium was given to five healthy volunteers, in orange juice which contained citrates and sugars. Edwardson measured the amount of aluminium that was absorbed into their blood.

Then he gave the volunteers the same drink, but with added silicic acid. This time the amount of aluminium absorbed into the bloodstream was reduced by a massive 85 per cent.

He concluded: "A long-term increase in the dietary intake of silicic acid could prove to be of therapeutic value by reducing the gastro-intestinal absorption of aluminium in those at risk".

manganese and other metal ions), and blocks its physiological actions[135, 142]. But even this would not offer complete protection.

Another answer is to take supplements of the essential trace element silicon in the form of silicic acid. The silicic acid binds with the dietary aluminum to produce aluminium silicate (sand!), which passes harmlessly through the body[133].

The richest food sources of silicon are the cereals, with oats in particular containing very high amounts[136]. We eat less cereals, especially oats, than ever before. Could this be contributing to the current increase in neurodegenerative disorders such as Alzheimer's or Parkinsonism?

The positive effects of silicon

Animals fed a silicon rich diet have lower levels of aluminium in their brains[137].

High levels of brain aluminium correlate with mental impairment.

The conclusion seems to be that a silicon rich diet should be one of the protective strategies against aluminium intoxication and mental deterioration.

You find silicon in oats, cereals and hops.

INCLUDE

More oats and cereals in your diet.

If you are especially concerned use a silicic acid supplement. Tablets are relatively ineffective; colloidal silicic acid is preferable.

Also ensure your diet is rich in Vitamin B6 and zinc.

ESSENTIAL TRACE ELEMENTS

Trace elements like silicon have been largely ignored by medicine, due to the common belief that trace element depletion is rare. Evidence to the contrary was never taken seriously, because it comes from the humble vet.

Veterinarians have known for years that trace element depletion is extremely common in domestic and farm animals, and that mineral supplementation can often result in improved health.

Humans are unlikely to be exempt and recent trends in food processing and eating habits increase the probability of trace element depletion.

	Average Intake	Optimum Intake
Fe (iron)	13.2mg	15mg*
Mg (manganese)	4.6mg	10mg
Zn (zinc)	11.1mg	20mg
Cu (copper)	1.5mg	3.5mg
Se (selenium)	35mcg	100-200mcg
Cr (chromium)	30mcg	100-150mcg
Si (silicon)	10-100mg	100-1000mg

*In women of child-bearing age. Men and post-menopausal women require rather less.

One disease – many causes

All of the above research suggests that aluminium may be a contributory factor to Alzheimer's; but it is not the only one. In fact, there seem to be several different ways of getting Alzheimer's.

Genetic susceptibility has been demonstrated in a small proportion (about 5 per cent) of cases. Head injury, depressive

illness and late first pregnancy are also associated with a higher risk of the disease.

However, none of these factors – and certainly not the genetic factor[66] – are enough to explain the 15 or more millions of people worldwide who have been diagnosed as having Alzheimer's disease. When a (non-infectious) disease occurs on this enormous scale, it is often more fruitful to look for an environmental and/or nutritional cause.

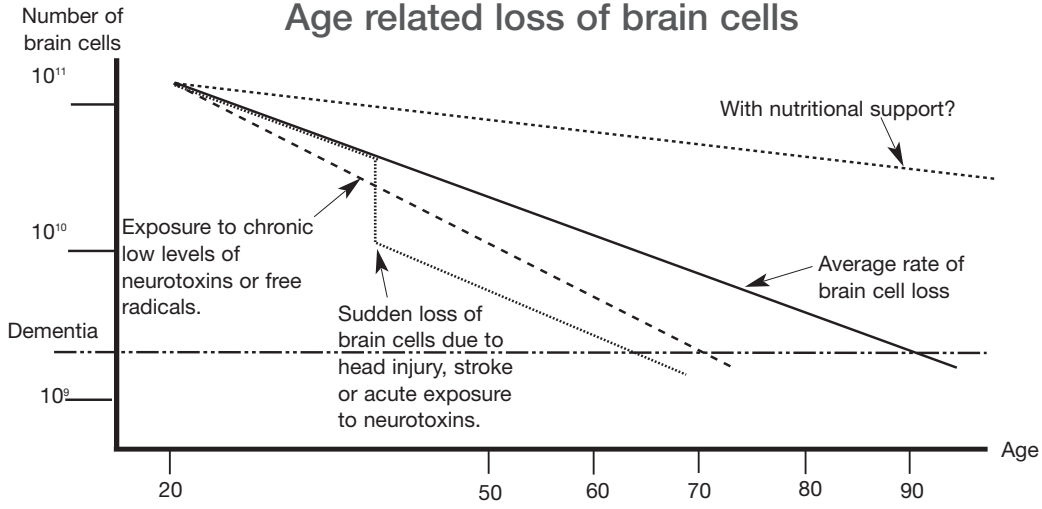
There is evidence that some degenerative brain diseases of old age, such as Alzheimer's and Parkinson's disease, which are both on the increase, may be caused by long-term exposure to low levels of environmental toxins. Aluminium compounds could conceivably fit the bill, but there are other potential neurotoxins such as organic solvents and agrochemicals.

Even more persuasive is the evidence that depletion of micro-nutrients – vitamins, minerals and trace elements – leading to excessive free radical damage, is the most important cause of most cases of Alzheimer's.

Different causes – common mechanism?

Head injury, depressive illness and late first pregnancy may seem unrelated, but all are thought to cause brain cell damage; and anything that causes brain cell loss brings dementia closer.

Age related loss of brain cells



- **Many of us lose brain cells as we age. Eventually our intelligence diminishes and we may become demented.**
- **Any additional factor causing loss of brain cells will speed the onset of symptoms.**
- **The underlying rate of brain cell loss is not uniform or inevitable – many people retain all their faculties into old age.**
- **Nutritional support is the *only* way to reduce the rate of brain cell loss to a minimum.**

A free radical disease

Brain protection

Lipids account for 60% of the brain's dry weight, and the bulk of these are poly-unsaturates.

Poly-unsaturated fats are very vulnerable to free radical attack.

Any brain fitness programme must therefore include anti-oxidants.

Most medical scientists accept that oxidative stress, an excess of free radicals, is a prime cause of coronary artery disease; and they are happy to recommend anti-oxidants to reduce the risk of heart attacks. But they have not yet made the glaringly obvious connection to diseases of the ageing central nervous system, including Alzheimer's. Even more than the heart, the brain and related tissues are a prime target for oxidative damage because …

1. Brain nerve cell membranes contain high levels of poly-unsaturated fatty acids (PUFAs), which are extremely vulnerable to free radical attack.
2. There is a lot of oxygen used in the brain. (The brain receives 25% of the cardiac output even though it is only 3% of the body's weight.) The more oxygen, the more likely that oxidation may take place.
3. The brain's anti-oxidant defences are poor. Levels of glutathione, catalase, Vitamin E, etc. are low[101, 109-112]. Levels of iron (a potential pro-oxidant) are high.
4. At least one compound found in the brain (dopamine) forms breakdown products which create free radicals[108].
5. The elderly's eating habits leave them progressively more at risk of being depleted in micro-nutrients and especially the anti-oxidants.

More anti-oxidant data

Free radical damage is strongly implicated in loss of memory and cognitive function during ageing.

Anti-oxidant supplements (Vitamins C and E) given to elderly rats improved some cognitive parameters[83].

Anti-oxidant intake is important in reducing the risk of age-related cognitive decline in elderly humans[78-80, 105, 106, 113]; and in Alzheimer's patients[81, 82, 114].

In a very recent study it was found that elderly subjects who took Vitamins C and E were largely protected from developing Alzheimer's[104].

It's hardly surprising therefore that the incidence of the neuro-degenerative diseases is high in the elderly, and increasing as the population ages. Older brains have suffered more cell loss due to chronic oxidative damage; and this could be enough to explain why we lose brain cells as we get older. It's probably little more than cumulative oxidative damage leading to progressive nerve cell loss.

This may show up as age-related cognitive decline (ARCD), a gradual loss of mental processing power which occurs commonly with age; or its end stage, which is Alzheimer's disease. Oxidative

neurone loss may, if exacerbated by specific metabolic, genetic or other environmental factors, manifest itself as Parkinsonism, Huntingdon's disease or Amyotrophic Lateral Sclerosis.

These diseases have much in common. They are all characterised by slowly progressive brain cell death. Their onset is often subtle, beginning in mid-life and slowly becoming symptomatic in later life. And there is a great deal of evidence that oxidative damage, leading to brain cell suicide, is involved in all these conditions[37, 106-108].

Anti-oxidant supplements should therefore have a role to play in reducing the risk of neuro-degenerative illness, as they do in heart disease[85, 113]..

The choice of anti-oxidant is determined by the types of molecules we wish to protect. In the brain, the main targets of free radical attack are the Poly-unsaturated Fatty Acids (PUFAs) in the brain cell membranes. And here the pioneering work of Dr Stanley Deans at the SARC Institute in Auchinruive, Scotland, shows the way forward.

Dr Deans found that as lab animals age, levels of PUFAs in their brains gradually fall[96]. The numbers of viable brain cells and the animals' 'brain power', ie their ability to learn and run through a maze, fell away more or less in parallel[29].

But was the decline in PUFAs the cause of the loss of brain cells, or was it merely a consequence?

In Dr Dean's next study, the animals were given anti-oxidants in the form of thyme oil which entered the brain, and stopped the oxidation of lipids. As these animals aged, there was no reduction in brain PUFA levels; no loss of viable brain cells; and no age-related fall-off in memory[29-32].

The implication is clear. Oxidative damage to brain cells causes cumulative brain cell loss, and is likely to be a prime cause of the age-related loss of brain cells which manifests itself as age-related cognitive decline, and other conditions such as Alzheimer's disease.

The logical route to reducing the risk of these conditions is, therefore, via appropriate anti-oxidant supplementation.

LOOK FOR ...

a supplement that includes anti-oxidants like Vitamins C and E, flavonoids and thyme oil.

Alpha lipoic acid is an interesting alternative. And as glutathione peroxidase is important in protecting lipids from oxidation, its co-factor selenium should also be included; together with riboflavin, the co-factor for glutathione

Spice of life

PUFA levels in elderly brains are almost equally well preserved with clove, nutmeg and pepper oil[29-32, 207-211].

Add these to your diet!

FREE RADICALS, AGE SPOTS AND IQ

As cells age they accumulate the 'ageing pigment' lipofucsin[91, 97, 102], which is the end-product of PUFA oxidation[168, 169]. Lipofucsin build-up in the skin forms age spots, which are unsightly but harmless. In nerve cells, which are more vulnerable due to their high PUFA content and poor anti-oxidant defences, lipofucsin build-up first stuns and then kills the cell. Increased levels of brain lipofucsin are linked to reduced intelligence, and vice versa[97].

If the rate of PUFA oxidation and lipofucsin formation is slowed with anti-oxidants such as Vitamin E[85], lipofucsin deposits gradually fade away. Centrophenoxine, a powerful anti-oxidant drug[171] widely used to improve mental performance, removes lipofucsin from brain[173, 174], skin[175] and red blood cells[170]. In animal studies centrophenoxine improved mental performance and extended life span by up to 30%[172]; in a human study it increased cerebral blood flow significantly[170].

Similar studies have shown that, when elderly patients were given long-term anti-oxidant vitamins, their general condition and mood improved[203, 204].

More anti-oxidant evidence

Beta amyloid, which is found in other brain disorders[33-35] is not the only source of the destructive free radicals which contribute to Alzheimer's disease.

Aluminium (in the form of aluminium silicate) found in the tangles in Alzheimer's brain tissue, is another generator of free radicals[46].

This is another very good reason to take a comprehensive anti-oxidant supplement.

Anti-oxidants block a part (perhaps the most significant part) of the whole disease process.

More anti-oxidant evidence

Some laboratories have concentrated on the problem of plaque, a characteristic micro-lesion found in the brains of subjects with ARCD and Alzheimer's.

The main component of plaque is a peptide called beta amyloid. There are good reasons to think that this peptide may be involved in causing Alzheimer's.

In Down's Syndrome, for example, an excess of amyloid is produced and the incidence of early onset Alzheimer's is very high[24]. More recently, beta amyloid has been shown to be toxic to nerve cells[25, 26]. Interestingly enough, it kills them by producing free radicals, which oxidise PUFAs in the nerve cell membranes and more or less tears them apart[27, 28, 43, 68-72].

The neurotoxic effects of beta amyloid can be blocked by anti-oxidants such as Vitamin E[26]. So if beta amyloid is a cause of Alzheimer's, then, once again, lipid-soluble anti-oxidants such as Vitamin E, thyme oil or the herb rosemary should be useful in slowing the progression of the disease.

In support of the anti-oxidant hypothesis, a recent study showed that high-dose Vitamin E (2,000IU/day) slowed the

progress of Alzheimer's very significantly[81]. But Vitamin E is not the optimal anti-oxidant to treat brain hyper-oxidation, as it takes months to enter the brain at high levels[84-87].

It would be more logical to examine the potentially therapeutic role of thyme oil[29, 32] and other natural anti-oxidants which enter the brain more rapidly such as the soy isoflavones: labs in the UK and elsewhere are already doing this.

Additions to anti-oxidants

The basic anti-oxidant strategy attempts to protect the PUFAs that are such important components in brain cell membranes. But there are other, complementary strategies.

The cell membrane is a dynamic entity; PUFAs are constantly being lost, via oxidation and other routes, and being replaced by new PUFAs derived ultimately from the diet. Combining the appropriate anti-oxidants with appropriate PUFA supplements should therefore reduce the deficit, enhance the replacement rate, and give supra-additive effects.

There is one proviso. For optimal replacement, it is very unlikely that the usual PUFA supplements will be sufficient. Free PUFAs hardly exist in the brain; they are largely incorporated into molecules called phospholipids. Phospholipids are simply fatty acids (lipids) combined with phosphates and other groups. It is these phospholipids which form the basis of all cell membranes, and which are the main target of free radical attack in the brain.

Membrane ageing

I don't underestimate the importance of nuclear and mitochondrial ageing.

However, I think that phospholipid-related ageing of the membranes is also very likely to be a major contributor to the overall rundown in cellular and organ function that constitutes the ageing process (see Chapter 21).

Phospholipids – a key to brain function

Phospholipids, because of their molecular structure, are surfactants; a category of compounds which includes soaps and detergents. If you add soap to water, and mix it, bubbles form; and if you add phospholipids to water, they naturally form into membranes which in turn become micro-bubbles. These are the prototype cell membranes.

Like all tissues, the cell membranes are dynamic; they are constantly being damaged and repaired. Phospholipids in the

membranes are broken down to form neurotransmitters and other messenger substances that transfer information between brain cells. In addition they may simply be oxidised.

If these phospholipids are not replaced, membranes deteriorate and the cell becomes progressively more dysfunctional and eventually dies. So, the rate of phospholipid replacement is critical in keeping the brain cells and brain functioning.

We obtain phospholipids from our diet, and we can synthesise them in the liver. But there are problems. Our dietary intake of phospholipids is at an historic low, thanks to trends in food processing[90], and specifically to our increased use of refined oils. Levels of phospholipids may reach 3 per cent in virgin oils, but in refined oils they are virtually undetectable.

Production of phospholipids in the liver may be sub-optimal too because it is complex, slow and energy intensive. It is slowed even further by multiple micro-nutrient depletion, which surveys show is extremely common in the elderly. Anti-oxidant depletion simultaneously increases the rate of phospholipid oxidation. This combination of reduced production and accelerated breakdown leaves the elderly doubly vulnerable to phospholipid depletion.

This is why levels of phospholipids in cell membranes, such as in brain cell membranes, decline with age. This is a major component in the ageing process in many tissues[30-32, 91, 96, 102], where increased oxidation of phospholipids, and the resulting accumulation of the oxidation end-product, lipofucsin[30-32, 97, 102], leads to a progressive loss of membrane and other cellular functions.

The fact that the elderly are doubly at risk of phospholipid depletion – their poor micro-nutritional state leading to reduced rates of phospholipid production **and** increased rates of phospholipid oxidation – means they are logical candidates for anti-oxidants **plus** phospholipid supplements. But which phospholipid?

Balance

Every one of your cells is surrounded by a membrane. This is largely made up of phospholipids.

These vital compounds are constantly being broken down and repaired.

If the rate of loss outstrips the rate of replacement, this leads to progressive disrepair of the membrane, progressive dysfunction and death.

Anti-oxidant enzymes

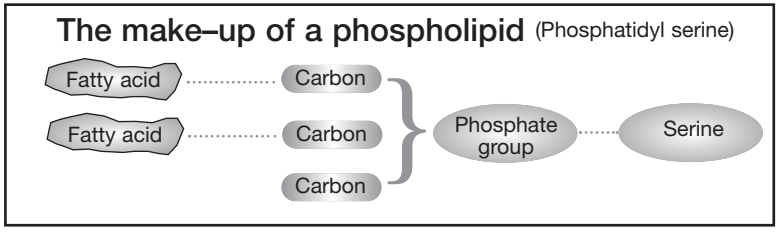
The key anti-oxidant enzyme that protects phospholipids in cell membranes is glutathione peroxidase. This requires selenium to function – and many people are dangerously low in selenium.

The enzyme cycle also requires riboflavin. Selenium and riboflavin are essential ingredients in anti-Alzheimer's nutrition.

An important new brain supplement

Here is where it pays to know a little biochemistry.

A phospholipid typically consists of two fatty acids, linked to a 3-carbon 'collar', which is linked to a phosphate group and one other group. This last group may be choline, making phosphatidyl choline (PC); or serine, making phosphatidyl serine (PS).



Although phosphatidyl serine is much scarcer than phosphatidyl choline, it is particularly important in keeping the brain cells' membranes healthy. Because of its surface charge, PS has the unique ability to bind to protein molecules such as the ion pumps. (These pumps, which sit in the cell membranes, are essential for keeping the cells alive, and for maintaining their electrical functions, which are especially important in nerve cells.)

If PS is in short supply, the binding action weakens. The ion pumps slow down, and then drop out of the cell membranes altogether[88]. The nerves become dysfunctional, swell up and burst. But if levels of PS can be restored to normal or near-normal values, nerve cell death may be averted and function restored.

PS supplements have been used on their own to improve membrane, and particularly nerve cell membrane function. In elderly animals, many of the symptoms of an ageing brain (including failing circadian and estral rhythms, memory loss, and the loss of nerve cell connections in the brain), can be prevented or reversed with PS[88, 98, 99] – but **not** with other phospholipids.

Does this apply to humans? There are 42 clinical studies of PS used to treat Alzheimer's, and its pre-clinical precursor Age-Related Cognitive Decline (ARCD). These trials, although most are small and/or poorly designed, show improved learning and memorising of information; enhanced recall of frequently misplaced objects; better recall of telephone numbers; recognition of names and faces; and maintenance of concentration while reading, conversing and performing various tasks[93, 94, 95, 100].

The phospholipid story in a nutshell

The brain needs a constant supply of phospholipids.

Our dietary intake of phospholipids (found in unrefined oils and animal brains) has declined.

We can make phospholipids in the liver. Unfortunately, as we get older and in cases of low micro-nutrient intake (common in older people), the rate of phospholipid production declines. (Another example of a bottleneck.)

In addition older people generally have a lower intake of anti-oxidants, and anti-oxidants are vital in protecting phospholipids in the brain from free radical damage.

So maintaining brain function demands good anti-oxidant intake.

In cases where Alzheimer's is a *real risk*, consider circumventing the bottleneck and supplementing with the key phospholipid – phosphatidyl serine.

Sadly, the fundamental flaw in all these trials is that phosphatidyl serine was used alone; and as we can now see, it should ideally have been combined with choline, anti-oxidants and other co-factors. I believe such a combination would be a major step forward in the prevention and treatment of Alzheimer's.

Note: The role of phosphatidyl serine (PS) may help to explain why cholinergic nerves, which use acetyl choline as their neurotransmitter, are the first to die in Alzheimer's. All the nerve cells in the elderly brain are depleted of phospholipids, including phosphatidyl choline (PC) as well as PS; but in cholinergic nerves, there is an additional loss of PC molecules from the membranes as the cell uses them to make acetyl choline.

When levels of phosphatidyl choline in the membrane are critically low, the cell attempts to replenish them from wherever it can; and if there is insufficient PC in the diet, or being made in the liver, the nerve steals from its much smaller store of PS molecules. If levels of PS are already low, this additional drain may be enough to dislodge the ion pumps; leaving the cholinergic nerve exposed to a uniquely lethal combination of oxidative, osmotic and electrical stresses.

Some cholinergic nerve cells also contain high levels of iron. When these cells die the iron is released and causes more free radical damage. This also may contribute to the fact that cholinergenic nerves are more likely to be lost in Alzheimer's.

Low B vitamins and nerve cell damage

Depletion of the B vitamins folic acid, B6 and B12 has been linked to nerve cell damage via excessive homocysteine levels.

Homocysteine is a pro-oxidant.

B depletion is common in the elderly, and a B supplement should therefore be combined with the anti-oxidant/PS regime for additional protection[195].

In recent studies, Vitamins B6, B12 and folic acid supplements improved some aspects of intelligence in the elderly[167, 205].

Alternatively, a betaine supplement is recommended.

When should prevention begin?

It is not universally agreed that age-related cognitive decline (ARCD) is the pre-clinical form of Alzheimer's, but it seems increasingly likely that it is. The behavioural changes, neurochemical changes, and even the histological signs of Alzheimer's (the plaques and tangles thought to be the hallmark of the disease), are all found to a lesser extent in ARCD. And, although some doctors still maintain that ARCD is a middle-aged condition, Professor Tom Crook at NIMH Bethesda has shown that if sufficiently sensitive instruments are used, the first signs of mental decline appear in the 40s and become progressively worse with each subsequent decade; a decline which starts well before the routine tests notice anything wrong[94].

Nutritionally, prophylaxis should therefore ideally begin in the 40s, but should still help even if started later in life.

Other strategies for reducing risk

Nerve cells may be killed off by causes other than oxidative stress. Accordingly, other nutritional factors may have something to offer. Cell suicide in the brain may, for example, be triggered by hormonal changes which occur with age.

Normally, nerve cells are sustained by a range of nerve growth factors. If the growth factors dry up, the nerve cells switch on their suicide programme. This could be why women who take HRT have a lower risk of developing Alzheimer's[75, 76]. It's reckoned that the oestrogen in HRT may function as a nerve growth factor[77] and, like a cellular Samaritan, encourage the neurone not to switch on its suicide (apoptotic) programme.

If true, a high intake of soy products (which contain oestrogen-like compounds) should also be protective. Resveratrol in red wine is another potential protector.

Another factor which can trigger apoptotic cell death is a sustained reduction in the oxygen supply. The sudden and complete lack of oxygen caused by a stroke or strangulation leads to uncontrolled cell death (necrosis). But a long-drawn-out reduction in the oxygen supply to the cell, which is what happens when the cerebral arteries gradually fur up, is known to switch on the apoptosis sequence.

This means that bad circulation is another possible cause of Alzheimer's, which is probably why hypertension increases the risk of the disease[67]. In this case, and particularly if there is evidence of arterial disease in other organs such as the heart or legs, a crash course of anti-atheroma nutrition is in order. At the very least this approach will reduce the likelihood of cerebral infarcts, or mini-strokes, which can add to and exacerbate the underlying Alzheimer's; and reduce the loss of brain tissue if a stroke does occur[103].

Stroke

Only one third of dementia is due to Alzheimer's, another third is damage from multiple strokes, and the rest are mixed. Anti-stroke nutrition will, therefore, greatly reduce the risk of dementia, as well as the damage caused by the stroke itself.

Oestrogen anti-oxidant

Oestrogen may be a nerve growth factor; but it is also certainly an anti-oxidant, and a lipid-soluble one at that[77].

This is just what is needed to protect brain cell membranes – so oestrogen may be acting primarily as a membrane protector.

The isoflavones in soy, which not only mimic oestrogen but also act as strong anti-oxidants, and are known to enter the brain quite rapidly, are almost certainly protective.

Stroke prevention

Stroke is a loss of blood supply to an area of the brain, caused either by an artery rupturing (generally due to high blood pressure), or becoming blocked from atheroma or blood clots.

Blood pressure is lowered by changing from salt to a potassium/magnesium substitute and by taking flavonoids (see page 329). Atheroma and platelet stickiness are reduced by flavonoids [91,115], which thus decreases stroke risk[212].

The high speed anti-atheroma programme

This would include the Vitamins C and E, Q10 and lycopene. It could also initially contain the amino acids lysine and proline, switching to grapeseed, bilberry or hawthorn flavonoids after about a week, plus a good fish oil.

It should also contain ginkgo, which has been shown to increase the resistance of nerve cells to hypoxia[12, 13], and reduce oxidative damage to the nerve cells[17].

The Tau theory in a nutshell

If axonal flow is blocked, the brain cell dies. This could be another route to Alzheimer's, so preventing such blockage is vital.

Blockages are caused when proteins called Tau proteins stick together in the tubelets that carry the axonal flow.

Manganese appears to return the Tau protein to normal and thus may help to restore or maintain axonal flow and hence brain cell function[7-10].

Go with the (axonal) flow

A relatively new idea is that Alzheimer's may be triggered in certain cases by depletion of the trace element manganese. This concept was introduced by Dr Iqbal at the New Institute for Basic Research in Developmental Difficulties, New York, and hinges on the concept of 'axonal flow'.

The next page shows how axonal flow in the brain works. Essentially, the constant activity involved in the transmission of information between brain cells creates wear and tear at the dendrites – the end of the cell containing the synapses where information is transferred from one brain cell to another.

To repair this damage involves transporting 'broken parts' from the dendrites along an axon to the nucleus. Then 'replacement parts' are sent from the nucleus back out to the dendrites. If this transport loop is blocked the brain cell dies. Preventing such disruption may be another way to defend against Alzheimer's.

You can see that the internal transport system ('the axonal flow conveyor belt') for each brain cell is built of Tau proteins.

In a healthy cell, the Tau proteins remain separate. Dr Iqbal's work suggests that, under certain circumstances, Tau proteins stick together, and build up into the helical filaments found in the brain 'tangles', in a runaway reaction. But what makes those first few Tau proteins stick together?

Some studies have shown that in Alzheimer's, Tau proteins in the filaments have too many phosphate groups attached to them[1-4, 21] – known as hyper-phosphorylated Tau, or Hyper-T.

Dr Iqbal's group believe that Hyper-T is what makes the Tau proteins stick together. They found that Hyper-T disrupts axonal flow[5]. They also showed that in Alzheimer's disease, the nerves die back in a pattern of retrograde degeneration[6], which is exactly what happens when axonal flow is blocked.

So if the phosphorylation of Tau proteins could be reduced, this might restore axonal flow and help to slow the course of Alzheimer's disease.

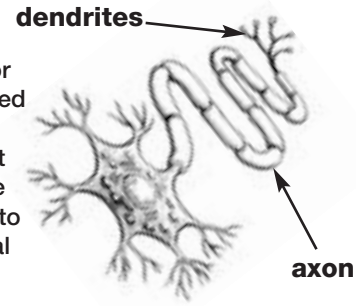
Dr Iqbal's lab has discovered two groups of enzymes: the Tau kinases, which attach phosphate groups onto Tau protein, and the Tau phosphatases, which strip them off again.

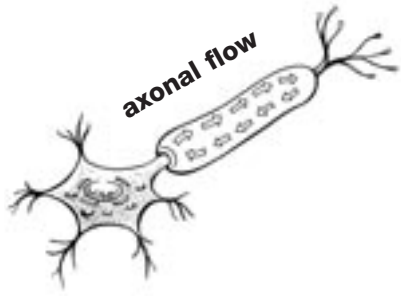
AXONAL FLOW

A blockage in 'axonal flow' leads to brain cell death and may be involved in Alzheimer's disease. Restoring axonal flow could help stop brain nerve cells from dying back.

1 The structure of a brain cell

A brain nerve cell typically consists of a rounded cell body or nucleus, and a long trunk reaching out from the nucleus called an axon. The axon ends in a spray of nerve endings called dendrites. These dendrites are highly active sites because at the end of each dendrite is a synapse. Neurotransmitters are the chemicals that allow the electrical impulses of thoughts to 'jump' across the synapse. There appears to be a substantial degree of tissue wear and tear at these dendrites.



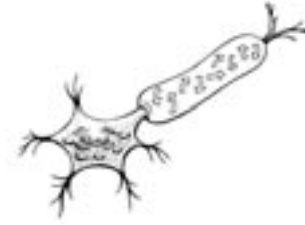


2 Axonal flow

The workshop where replacement parts for dendrites are made is back in the cell nucleus. To get the broken-down parts from the dendrite nerve endings back to the nucleus, and to send replacements back out again to the dendrites, involves 'axonal flow'.
This is a sort of conveyor belt which runs the length of the axon. One lane ferries new components from the nucleus to the nerve endings, and the return loop brings debris back from the nerve endings to the nucleus, where they can be recycled.

3 Retrograde degeneration

If the flow of materials along the conveyor belt is blocked the nerve endings die first, and then the axon starts to die back in a slow process of decay, called retrograde degeneration, which eventually reaches back to the cell body. Up until this moment the decay is reversible, but once the degeneration reaches the cell body the nerve cell is irretrievably dead.



4 Cross section of axon conveyor belt

The axon's conveyor belt is built out of micro-tubelets, which in turn are made from smaller components, including one called Tau protein.

5 Plaques and tangles

There are two characteristic lesions in the brains of Alzheimer victims: plaques and tangles. The tangles consist of Tau proteins stuck together in long, paired helical filaments. In this form they can no longer contribute to axonal flow.

This suggests two ways of restoring axonal flow, and therefore the health of the brain's neurons.

1 Slow down the Tau kinase enzymes, which attack the phosphate groups. However, there are several enzyme groups involved and inhibiting all of them would almost certainly cause severe toxicity problems.

2 Encourage the Tau phosphatases, and increase the rate at which excess phosphate groups are stripped off the Tau proteins. This seems a more likely proposition, because these enzymes are underactive in Alzheimer's brain tissue[7]. Furthermore they can be activated by the trace element manganese[8, 9, 10].

This leads to two tentative conclusions. Firstly, chronic manganese depletion might increase the risk of Alzheimer's. This would also explain why the Tau phosphatase enzymes are underactive in the disease. Secondly, a manganese supplement might be able to stop or slow the disease process in some cases, although it would not confer anti-oxidant protection, which remains essential.

A small manganese supplement might help to normalise Tau protein, which could restore axonal flow, stop further degeneration of the affected neurones, and perhaps even permit the regrowth of damaged axons and nerve terminals.

NB Manganese **must** always be combined with copper, selenium and zinc, and with antioxidant and membrane support.

Preventative manganese?

Although the manganese hypothesis is not proven, it may be worth adding a small amount (1-2mg/day) to the anti-Alzheimer's nutritional programme. High doses of manganese are not recommended, and have been linked to an increased risk of new variant CJD.

Smart drug

The so-called 'smart drug', hydergine, which may mimic nerve growth factors[38, 39] may be helpful in the early stage of Alzheimer's[40, 41].

Unsurprisingly, it's less effective in the later stages[42].

TOP MANGANESE FOODS

Tea, spinach, broccoli, orange juice, nuts, beans, wholegrains and blueberries.

MANGANESE LEVELS

Manganese is essential, but, as is common with trace minerals, too much may be toxic. A level of 50mg manganese a day gives maximal activation of the Tau phosphatases[11]. However, this high dose is not recommended as it may increase the risk of nvCJD. At this time, we do not recommend intakes higher than the current RDA of 4mg. Patients with liver disease should be particularly cautious, as the resulting build-up of manganese in the body could cause toxicity problems[62-64, 73, 74].

For those on a normal diet, a supplement of 1-2mg a day should be sufficient.

Aluminium, manganese and zinc

The manganese story may prove to be an object lesson in how science develops. Many non-scientists believe in a confrontational model of science, where old theories hold sway until they are destroyed by new evidence, and replaced by different and better ideas.

This sort of conceptual coup d'état does occur, but is not as common as evolutionary change, where an old idea which previously seemed complete is revealed by new knowledge to be part of a larger whole.

Although there were always gaps in the evidence for aluminium being the sole cause of Alzheimer's, there is too much evidence linking aluminium to brain damage to allow the hypothesis to die away altogether.

Manganese depletion may seem to be a better candidate now than aluminium excess, but rather than replace the aluminium theory it may actually extend it. Aluminium is not only toxic itself, it also competes with manganese – so if someone was low in manganese, aluminium exposure could tip them into manganese deficiency, which might also lead to Alzheimer's. (Zinc also competes with manganese; extra zinc given to Alzheimer's patients makes manganese depletion, and their condition, worse.)

This suggests that a manganese-rich diet might give some protection against Alzheimer's. However, do not take high doses of manganese; high doses, especially if taken without copper, zinc and selenium, may be a cause of a different type of brain damage; new variant CJD.

This demonstrates that nutrients do not act like and should not be used like drugs. Large doses of single nutrients are seldom useful and may be counter-productive. The route to health maintenance is via comprehensive micro-nutritional support.

Finally, it is only fair to say that not all scientists agree with Dr Iqbal's work. Some say that most Tau proteins in the paired helical filaments are not over-phosphorylated[22, 23]. And some say that hyper-phosphorylated Tau might discourage, rather than encourage filament formation[23]. So the debate continues.

Dangerous skins?

If you eat potatoes or aubergines, be cautious. Potato and aubergine skins contain the tropane alkaloids, calystegine A3 and B2[12].

This group of compounds causes degenerative neurological diseases in cattle[13, 14, 18], and may be implicated in causing anencephaly and spina bifida in humans[19].

They may not be a significant cause of Alzheimer's, but anything which kills large numbers of brain cells will exacerbate the disease, and speed its onset.

Tropane alkaloids are soluble in water, so if the potatoes or aubergines are to be boiled or stewed the toxins will leach safely into the cooking water. If, however, the potatoes are roasted or baked traces of the alkaloids may remain in the skins.

Additional help for Alzheimer's

Q10

Another factor which may contribute to Alzheimer's disease is mitochondrial ageing. Mitochondria become less efficient with age, and old, burned-out mitochondria can't make enough ATP (the energy compound) to fuel the body cells[44]. This is another well-established cause of cellular suicide.

In cases of Alzheimer's caused largely by mitochondrial inefficiency, high-dose Co-enzyme Q10, which can improve mitochondrial output, is the theoretical remedy of choice[57].

The scientific reports, however, are confusing; some find sub-normal levels of Q10 in the brains of Alzheimer's victims[14], while others find increased Q10[45]. This may be yet another indication that you can end up with Alzheimer's via a number of different routes.

Flavonoids

There is good evidence that Alzheimer's is, at least in part, an inflammatory condition. The disease was classically described as having some of the appearances of inflammation, and it has been described by some contemporary researchers as 'arthritis of the brain'.

Other experts disagree, but it is an interesting fact that in people with rheumatoid arthritis, and who were treated with anti-inflammatory drugs, the incidence of Alzheimer's is rather lower than in the general population. When this was first discovered, further work revealed that the reduction of risk occurred mostly in the sub-group of arthritic patients who were treated with indomethacin, one of the few drugs of its type which penetrates the brain in large amounts.

Indomethacin is a powerful drug with potentially serious side effects, and is not recommended for long-term prophylactic use except under medical supervision. However, there are various flavonoids, derived from food crops, which do much the same thing as indomethacin.

The oligomeric procyanidins (OPCs), found in bilberry extract and grapeseed extract, for example, enter the brain, are potent

LOOK FOR ...

a supplement that includes anti-oxidants, carotenoids, a flaxseed oil or grapeseed oil and also OPC flavonoids (oligo-proanthocyanidins) like grapeseed/bilberry extract – at about 100mg.

To that add isoflavones at about 30-40mg. These all have anti-oxidant and anti-inflammatory properties and make a good preventative

Oxidation – Inflammation

The processes of oxidation and inflammation are very closely intertwined. Significant oxidation cannot exist without inflammation and vice versa.

Flavonoids

The flavonoids' abilities to reduce platelet stickiness and improve vascular health should protect against strokes[91, 115].

anti-inflammatory agents and, remarkably, have the additional ability of preventing the hyper-phosphorylation of proteins such as the Tau proteins[15, 16].

This combination of therapeutic actions, and their well-attested safety, makes the oligomeric procyanidins excellent candidates for long-term prophylaxis against Alzheimer's (see Chapter 6, Flavonoids & isoflavones).

Rosemary

Rosemary contains flavonoids which are anti-inflammatory and powerful lipid-soluble anti-oxidants. They have another valuable property: they bind to iron, preventing it from generating free radicals[54]. This herb, combined perhaps with thyme, would be a very good bet in the treatment not only of Alzheimer's, but also of Parkinson's disease and Amyotrophic Lateral Sclerosis.

'Rosemary for remembrance'

An alternative to rosemary is hawthorn.

This plant also contains flavonoids which are anti-oxidant, bind iron, and enter the brain where they are needed.

The only side effect is mild sedation. Herbal practitioners sometimes use hawthorn to help sleep (see Chapter 6, Flavonoids & isoflavones).

Light at the end of the tunnel?

Screening for a lethal disease that cannot be cured raises difficult ethical dilemmas. At the time of writing, conventional wisdom says, "There is no drug which can prevent Alzheimer's, and there is very little that can be done, other than to slightly delay the progress of the disease."

I am no longer convinced that we have to be so pessimistic, or as passive. In all the neuro-degenerative diseases (Alzheimer's, Parkinson's and Amyotrophic Lateral Sclerosis) free radicals have been strongly implicated in causing brain damage[55].

As we get older, and as our diet becomes depleted in micro-nutrients, more and more of us are developing the so-called neuro-degenerative diseases, and these are eating up an ever-growing share of the health-care budget.

Anti-oxidant therapy would appear to be a logical response. Vitamin E is probably an important anti-oxidant in the brain under normal circumstances[56], but is not necessarily the best form of therapy, as it takes a very long time to get into the brain and may become pro-oxidant in existing disease states.

I prefer to combine Vitamins C and E with OPC flavonoids, and isoflavones as a preventative strategy. I would add a thyme oil

and phosphadityl serine supplement in cases where actual mental decline is suspected, with B vitamins or betaine and optional manganese.

AN ALZHEIMER'S TEST?

When a solution of tropicamide (a drug used by opthalmologists) is applied to the eye, the pupils of Alzheimer's patients dilate by 13 per cent or more; whereas the pupils of healthy subjects don't.

This finding is being checked, but it may provide us with a relatively cheap and hence accessible early warning system.

Increased levels of Tau proteins in spinal fluid, although more difficult to measure, may be an alternative. Preliminary findings indicate that these are not only raised in Alzheimer's patients, but also in people with genes predisposing them to Alzheimer's, before they develop the illness[36].

Stress and Alzheimer's

It may be possible to reduce the risk of Alzheimer's with a life-style change. There is evidence that depressive illness is a risk factor for Alzheimer's[47-50]. This may be due to the fact that during depression, levels of the stress hormones (glucocorticoids) increase in the body to the point where they become neurotoxic, and cause brain damage[51], a process which tends to worsen with age[52, 53].

Chronic stress leads to the production in the body of the same neurotoxic hormones and this could be one of the reasons why under-educated people have a higher risk of developing Alzheimer's. It has been suggested that this is because their brains are 'less exercised', but it could equally well be because the under-educated are likely to be lower down the social ladder, and living more stressful lives. And it may be another reason to take one of the ginsengs which reduce the stress response.

A more potent and more specific antidote, however, could well be the hormone dehydroepiandrosterone (DHEA). This has been described as an anti-ageing hormone. Levels of DHEA in the blood peak by the end of the second decade and then go into a long decline, falling to a third or less of peak values by the age of 60[58, 139].

DHEA has many interesting properties, but the one which appears particularly relevant here is its powerful anti-glucocorticoid action[59, 138-140].

The glucocorticoid blocking effect means that DHEA could protect brain cells from stress or depression-induced injury. The fact that DHEA levels are very low in the elderly could be a significant factor in predisposing to Alzheimer's disease.

For this reason, a DHEA supplement is also worth considering as part of a risk reduction programme. The side effects of DHEA appear to be entirely positive: they include enhanced immune function[60, 61], a marked increase in feelings of wellbeing[20], and probable improvements in weight control and diabetes[20]. (See Chapter 15, Bones).

Food for thought?

So far we have been looking at ways to prevent mental deterioration in later life. But what about building brains at the *beginning* of the life cycle? Can micro-nutrients make children more intelligent?

This apparently simple question has been fiercely debated ever since 1988 when Gwilym Roberts, a science teacher in Wrexham, Wales, found that vitamin and mineral supplements increased the (non-verbal) intelligence of children in his school.

They also became better able to concentrate and less disruptive. The teaching staff were delighted, as were Larkhall, the nutrition company that supplied Mr Roberts with the supplements.

It didn't take long, however, for the opposition to muster. The seemingly innocent Wrexham study sparked off a period of fierce infighting, industry lobbying, lawsuits and dirty tricks which led, ultimately, to the end of Mr Roberts's career in Britain.

Initially academics and doctors rejected Roberts's findings out of hand, probably because he was a 'mere' schoolteacher.

Dr David Benton, however, could not be so easily dismissed. A psychologist at Cardiff University in Wales, he repeated Roberts's trial with equally positive results[157]. In the USA, Dr Stephen Schoenthaler reported similar findings[158, 159].

These studies, together with Gwilym Roberts's school trial, were included in QED, a BBC television programme, which generated an enormous amount of publicity.

The 'lost' generation

Professor John Garrow, Head of Nutrition at London's Bart's Hospital and former Chairman of HealthWatch, was one of the scientists who initially argued against Mr Roberts' findings. Subsequently he was prepared to admit the possibility that he may have been wrong. But then again, the evidence against him was overwhelming.

Professor Garrow is famous in nutritional circles for claiming that the only thing supplements do is create expensive urine. Not long after he said this, research carried out in his own and other

33% of children risk nutrient deficiency

As many as one in three schoolchildren are nutritionally depleted. Even the anti-supplement lobby now admits that giving these children extra nutrients means their behaviour and school performance improves.

Poor diet – poor marks

The unhealthier the child's diet, the worse his or her behaviour and achievement levels.

Research shows that a good supplement can help improve matters.

Sceptics confounded

Only one study attempted to refute Benton and Roberts's work[160]. This study was subsequently revealed to be so fatally flawed as to be effectively worthless[161].

Poly-unsaturates and birth defects

A lack of the right poly-unsaturates in pregnancy is linked to an increased risk of babies with cerebral palsy, sight problems and learning difficulties.

Pre-term babies appear to be most vulnerable – and most responsive to Omega 3 supplements[242-244].

hospitals proved that supplementing with folic acid and other micro-nutrients during pregnancy reduced the risk of low birth weight[209] and disorders such as spina bifida[177, 178], cleft palate, hare lip, and urinary tract malformations[189, 190, 192].

The real victims in this hotly debated case, however, are the many schoolchildren (as many as one in three, even in the 'developed countries'!) who are considered to be eating such deficient diets that their non-verbal IQ, and school performance, could be improved by supplements.

A generation of these children could have been helped if 10 years of medical infighting had not squandered that opportunity.

Admittedly, the children of the academics and doctors who opposed vitamins may not have needed them, because they were probably eating a relatively good diet. But if the establishment had read Gwilym Roberts's report more closely, they would have found that their children were not the most at risk.

Roberts had noticed that it was the children who ate the unhealthiest diets, and who were often underachievers, who seemed to benefit most from the supplements.

Autism

Many autistic children are intolerant of citrus and other foods containing flavonoids, due to an under-active enzyme (phenol sulphur transferase). Fish oil boosts this enzyme; medium chain triglycerides have even more effect[210].

Cautious in advocating vitamins, he concluded that, although it would be better to improve their general diet, supplementation could have an important part to play in enabling them to perform better at school. And this is where, for the moment, the issue rests.

The science suggests that for parents concerned about their children's diet (and how many children do you know who eat a sensible diet?), a properly designed vitamin and mineral supplement can help in IQ tests and in the classroom[162-166].

It's hardly a new concept. The idea that eating fish is somehow good for the brain has been around for many years. The medical profession dismissed it as an old wives' tale – and yet, the emerging science of nutrition and IQ has proved that this old tale is largely correct.

Bringing up baby

It's important for children and adults to eat the right nutrients to optimise their mental functions. But it's even more critical for infants.

The growing brain has a high requirement for certain poly-unsaturated fatty acids (PUFAs). Large amounts of these were present in our diet as we evolved as a species[187], and are secreted in mother's milk. But some formula milks still don't contain the right PUFAs. As a result, they can impair the normal growth and maturation of the bottle-fed baby's brain.

This has been demonstrated in a number of studies. In one typical trial, formula-fed infants scored significantly worse in tests of brain and nerve function than breast-fed babies. The authors considered this was because the bottle babies were PUFA-deficient[219]. Bottle-fed babies tend to grow more slowly, and suffer more illnesses. At one year they weigh less[143], their IQ is lower[144, 229], and their coordination is not as good as their breast-fed siblings[145], these differences appear to be life-long[230].

As a result of studies like these, the influential European Society for Paediatric Nutrition decided in '94 that all infant foods should contain the PUFAs essential for brain function.

It's surprising that the formula manufacturers didn't think of this first, because logic tells you that any formula should resemble mother's milk as closely as possible[132].

We don't know how much damage has been done to the brains of how many children, but at least this unnecessary nutritional depletion is being stopped. But, although it's a good idea to supplement baby foods, this is like bolting the stable door after the horse (or in this case the baby) has left.

ESSENTIAL FATTY ACIDS IN PREGNANCY

PUFAs critical to brain growth and function include DHA (docosahexanoic acid) and AA (arachidonic acid)[152, 201]. The best source of DHA is a good fish oil, and AA is formed in the body from vegetable oils. So eat oily fish two or three times a week, plus a helping or two of walnuts or brazil nuts (see Chapter 8, Essential fatty acids).

A good multi-vitamin and mineral supplement including Omega 3 and 6 (or flax or hemp oil) is also strongly recommended; the PUFAs and other nutrients essential to your baby's growth are absorbed into your blood stream, cross the placenta, and supply the baby with everything it needs to develop its potential to the full[192, 202].

Folic acid

Folic acid supplementation is now a universal recommendation in pregnancy to reduce the risks of low birth weight, spina bifida, hare lip and malformation of the urinary tract.

To this add a well-designed pregnancy supplement and a 1000mg capsule of a good Omega 3 fish oil – or a 1000mg flaxseed or hemp oil capsule.

Avoid high doses of Vitamin A in pregnancy

Although most nutritional supplements are very safe, high doses of Vitamin A should be avoided by women of child-bearing age.

Doses as low as 10,000IU (four times the RDA) have been linked to an increased incidence of birth defects[197].

Beta carotene is a safe substitute but must always be combined with Vitamin C and never with smoking. (Pregnant women shouldn't be smoking anyway!)

Asthma and infant feeds

A recent study has revealed that breast-fed infants are considerably less likely to develop asthma and other allergic illnesses later in life, than infants who are weaned onto formula feeds at an early age[194].

It seems that the infant formula manufacturers may have a degree of responsibility for the current epidemic of asthma that is sweeping the globe (see Chapter 19, Asthma).

Cerebral palsy

Difficult births have also been linked to cerebral palsy.

Breast is best

Some formula baby milks still don't have the essential PUFAs that an infant's developing brain needs.

Bottle-fed babies grow more slowly, weigh less and have lower IQs than breast-fed infants.

Manufacturers are now changing their formulas to prevent this.

Delicate balance

Throughout pregnancy, the developing foetal brain is highly dependent on a good supply of a number of key nutrients. At the pioneering Institute of Human Nutrition in East London, Professor Michael Crawford, Wendy Doyle and co-workers have been publishing papers linking maternal dietary deficiencies (including PUFAs and a range of vitamins and minerals) to birth problems such as cerebal palsy since the mid-'80s[147, 148].

Studies at other labs have shown similar results. PUFA intake is absolutely crucial for the normal growth of the retina and the brain, which contains very high levels of PUFAs in the membranes of brain cells[149, 240]. If the mother eats a grossly deficient diet in the three to six months before the pregnancy, or during it, the lack of PUFAs means that the baby's growing brain will take up the wrong fatty acids, incorporating saturated fatty acids instead of the poly-unsaturated ones[150, 151].

This is the best that nature can do to compensate, but it is not good enough. It may lead to premature, low birth-weight babies with brain damage such as cerebal palsy[152, 153, 219], or in lesser cases, visual problems and learning deficits[153, 154].

As a result, Crawford's team (and most of their international colleagues) now recommend that all pregnant women, and indeed any woman planning to become pregnant, should eat a diet containing portions of oily fish, as well as green leafy vegetables.

However, for the many women who can't or won't eat such foods, supplementation is an obvious option.

Breast-feeding and the infant brain

The human brain continues to grow after birth, as does its need for the essential PUFAs, so nursing mothers should continue taking the supplements until weaning.

But although fish oil improves infants' brain development[179, 190] and learning ability[176]; resist the temptation to feed supplements directly to your baby. Fish oil, for example, contains another PUFA called EPA, which does not occur in breast milk. There is a good reason for this: EPA competes with DHA and makes it less

available for the baby's brain. This means that while fish oils are fine for adults, and especially pregnant and nursing mothers, they should not be given to infants.

The best formula manufacturers now enrich their formulae with DHA not from fish oil but eggs, which is a step in the right direction; but the best food for babies remains mother's milk.

In fact, mother's milk is a beautiful example of human adaptability. Because PUFA intake is so crucial for the normal development of the growing brain, the breast tries to maintain a constant ratio of Omega 3 and Omega 6 PUFAs in the milk, despite large fluctuations in the mother's diet.

The PUFA content of breast milk in women living in fish or vegetable eating communities is very similar, despite a very different dietary intake of Omega 3 and Omega 6 PUFAs[146].

If the mother is not eating enough of one or the other type of PUFAs for her baby, her body will cannibalise her own resources to give the baby what it needs. It is only in extremis, or when an abnormal diet is eaten, that safety mechanisms fail and foetal brain damage ensues.

The evidence that common micro-nutrient deficiencies before conception and during pregnancy can adversely affect the developing foetal brain, is increasing. And it's not just the exotic micro-nutrients. The most common depletion, that of iron, is now also suspected of contributing to impaired intelligence in childhood and in later life also[192, 193].

Keeping in mind that many pregnancies are not confirmed until after the first three months, this is another potent argument for improved nutrition for all women of child-bearing age. Widespread food fortification programmes, and/or supplementation are the only answers.

Not just a problem for the poor

Infant brain damage caused by foetal malnutrition, which is caused in turn by maternal malnutrition, is a well-known pattern in areas of the world where food shortages and starvation are common.

Premature birth risks

Fish oils can prolong pregnancy without any detrimental effects. So Omega 3 PUFA supplements can be tried if you are prone to premature labour[155].

Another good reason for pregnant women to take fish oil supplements during pregnancy is that they may reduce the risk of the dangerous rise in blood pressure which can complicate the last three months and birth[156].

Fish oil should always be taken with anti-oxidants, which, especially Vitamins C and E, may reduce the risk further[204-207].

Iron and intelligence

Iron levels in pregnancy are often depleted and can lead to lower intelligence in children[192, 193].

TOP IRON FOODS

Liver, meat, chicken, whole grain, peas, beans, spinach, nuts.

Iron – caution

Iron supplements are not advised for men or post-menopausal women unless iron-depletion anaemia has been diagnosed.

In the West, too, low birth-weight, cerebral palsy and related problems are strongly associated with social deprivation. And even in wealthy and well-nourished communities, many pregnant women are deficient in a range of vital micro-nutrients.

This has been shown by recent studies where multi-vitamin and mineral supplements reduced the risk not only of babies with congenital neural tube defects such as spina bifida and oro-facial clefts[177, 178], but also congenital disorders of the urinary tract[188, 189, 190, 192].

Are the roots of schizophrenia in the womb?

The developing brain is uniquely sensitive to malnutrition. The psychoanalytical and viral theories of schizophrenia have been discredited. Some forms of schizophrenia have now been linked to faulty development of the brain during growth in the womb, probably during the second trimester[193-197].

Studies have suggested that maternal dietary deficiency, leading to foetal deficiency, is a cause of schizophrenia which may not manifest itself until 20 or more years later in the affected child's life[198, 199].

It is not known which micro-nutrients are involved, but folic acid and Vitamin B12 are likely candidates. Both of these vitamins are important for nerve function. B12 deficiency in adults leads to nerve damage, and a deficiency in either or both of these vitamins during pregnancy has been linked to neural tube defects such as spina bifida[177, 178, 213].

It may be no coincidence that in certain communities (such as Ireland) where there are specific problems with B vitamins, a high incidence of both spina bifida and juvenile onset schizophrenia have been reported.

However, there is probably more than one way of contracting schizophrenia. There is evidence for a genetic causative factor in some cases of schizophrenia[190, 191, 199] which may interact with the nutritional factors.

Adolescence

Hostility and aggression in adolescents were reduced by fish oils[247-249] and by vitamin and mineral supplements.

Food and mood

The brain communicates with the body – and vice versa – through electrical signals, through neuro-transmitters (chemicals) and via hormones. The proper function of neuro-transmitters and hormones depends upon correct nutrition. So we shouldn't be surprised if mental function improves with optimum nutrition (or that the immune system suffers through stress and grief).

In some cases patients with mental problems have responded as well, if not better, to nutraceutical treatment than to pharmaceutical treatment.

For example, some patients with depression have responded well to Vitamin B complex, magnesium, the amino acid tryptophan and DMAE (see box below).

Generally, the nutritional supplement programme, recommended on page 348, should not only help maintain a good state of mental health, but act preventatively against the risk of degenerative disease.

Depression – some answers

Depression is the most common psychiatric illness. The World Health Organisation estimates that as many as five per cent of the world's population suffer from depression, and numbers could be on the increase.

Temporary depressive states are even more common and affect as many as one in five of us.

Prozac is the drug of choice to treat depression, but a herbal equivalent has been around for centuries – St John's Wort (*Hypericum perforatum*).

This herb was known in the Middle Ages as 'Fuga daemonum', or 'flight of demons'. Its common name, Walpurgis Herb tells a similar tale: this was a herb which could drive out the devils of melancholy.

It is only in the past few years that the herb has been systematically studied. The results indicate that the medieval herbalists were right. St John's Wort contains compounds with a powerful anti-depressant action – and fewer side effects than most anti-depressant drugs.

In two fairly typical trials Hypericum significantly reduced the symptoms of depression, and effected a complete recovery in many cases[180, 181]. Symptoms of insomnia and fatigue responded particularly well. Unlike some anti-depressants, Hypericum doesn't cause drowsiness[105, 182]. Dependence and addiction do not develop.

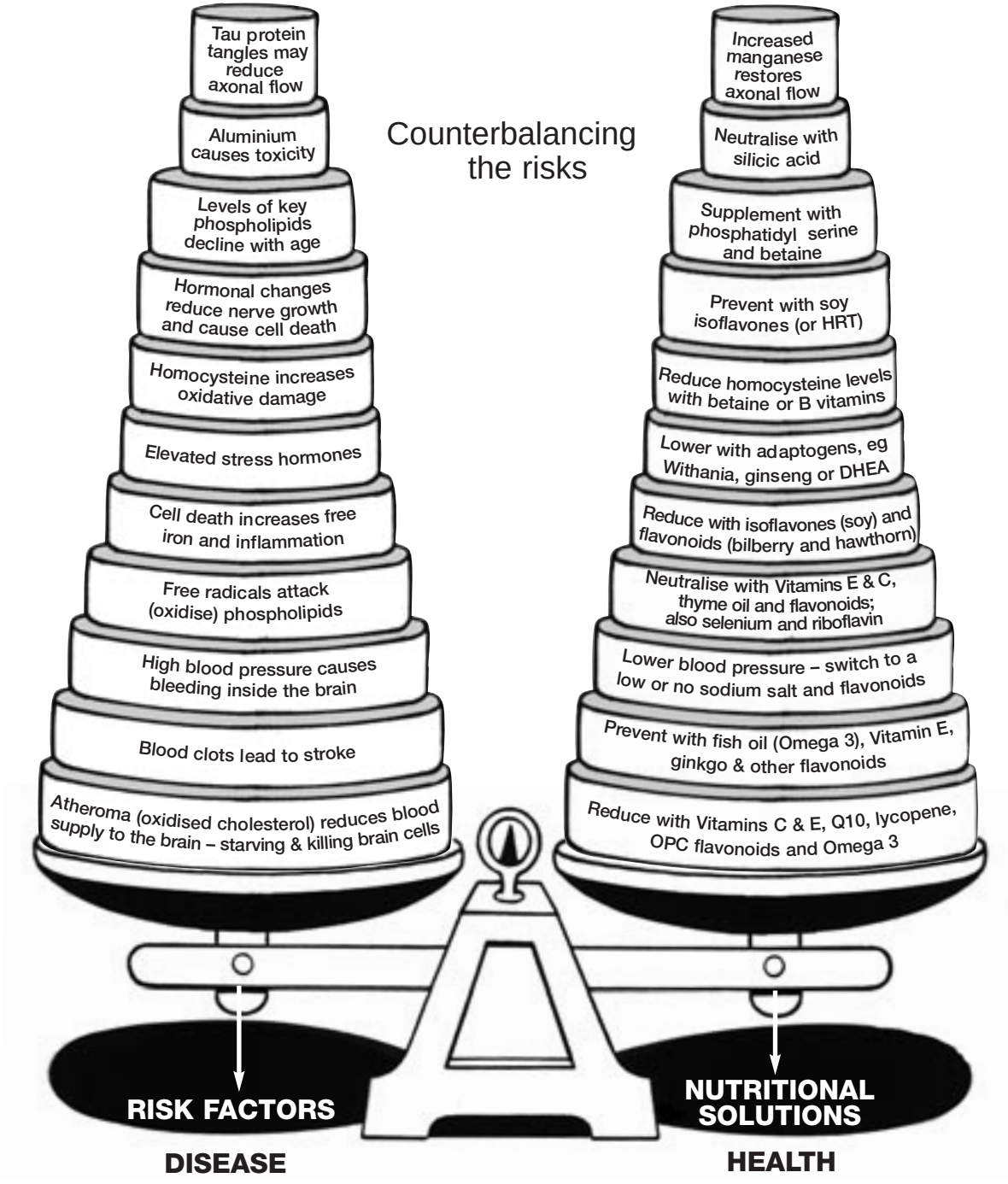
Nutrients are just as important, and may be more effective. Methyl groups are needed to make the crucial (monoamine) neurotransmitters involved in mood, so methyl depletion (surprisingly common) predisposes to depression[241]. This explains trials which show that strong Vitamin B complex[196], S-adenosyl methionine[231-236] and DMAE (di-methyl-aminoethanol) [183-186], all have anti-depressant activity. However, the clinicians clearly didn't understand the biochemistry; because the best source of methyl groups is betaine (see Chapter 11).

Logic dictates that betaine must be as or more effective than standard anti-depressant drugs in cases where methyl depletion is involved – and this is reflected in my personal experience.

Betaine is contra-indicated in bipolar illness (manic-depression), as it may trigger episodes of mania[235]. For these patients, fish oil is the nutrient of choice. When Omega 3 fatty acids are built into nerve cell membranes they act as damping or modulating agents[237] and have been shown in four studies to lead to marked improvement in symptoms[238,239,245,246].

My preferred anti-depressant regime begins with betaine, and then adds Hypericum and the amino acid tryptophan, if needed.

Preventing mental deterioration



SUMMARY

Alzheimer's aid box

Protecting yourself against Alzheimer's is another example of a multi-layered defence strategy

Level 1

- Anti-oxidant nutrients (at the levels recommended on page 348) to prevent free radical damage to the fatty acids in the brain – especially the phospholipids;
- Flavonoids (eg bilberry/grapeseed extract) to help prevent inflammation;
- B vitamins and betaine to prevent nerve damage caused by homocysteine.

Level 2

- A thyme oil supplement.

Level 3

- Supplement with phosphatidyl serine to boost levels of this key phospholipid in the nerve cell membranes.

Level 4

- A manganese supplement which may help prevent Tau protein tangles and hence disruptions of axonal flow.

Additional action

- A Q10 supplement to help delay mitochondrial ageing.
- Silicic acid in a colloidal form.

Level 1 defence action would be an appropriate strategy for anyone over 40.

Level 2 action for anyone over 55.

Levels 3 and 4 strategies would be appropriate in cases where the onset of Alzheimer's is actually suspected.

Caution with alumininium

- Don't use aluminium based antacids, especially if you're pregnant.
- If you think you are at risk, use a silicic acid supplement.
- Don't cook acid foods (ie fruit) in aluminium cookware.
- Avoid foods containing E470b.

Brain food in childhood

- If your child has problems at school, look at his or her diet. A good, all-round supplement could help.
- Breast is best for many reasons, but if you are bottle feeding, check the formula contains the right poly-unsaturated fatty acids. (Often referred to as LCPs.)
- Pregnant and breast-feeding women should take folic acid, B12, Omega 3 as fish oil, evening primrose oil, plus a good multi-vitamin and mineral supplement.
- Be careful with Vitamin A if pregnant – mixed carotenoids are safer.