

Overcome Your Cholesterolphobia

Cholesterol is a word that strikes fear into the hearts of the middle-aged. Like rust, cholesterol never sleeps; a gluggy buttery substance constantly seeping through artery walls, causing a build-up of plaques that any day now will block the blood supply to heart or brain causing a heart attack or stroke. It's that grim vision that's in the back of our minds as we tuck into bacon and eggs 'as a treat' or reach for a slice of cheesecake. It's that vision that chills us as the doctor says our LDL (low-density lipoprotein) is 'a bit on the high side', and our HDL (high-density lipoprotein) is too low. From then on, our life becomes a numbers game, a personal Dow Jones index of mortality.

Such is the demonisation of cholesterol that it's sometimes easy to forget that the link between cholesterol and heart disease is still only a hypothesis. No one actually knows what causes arteries to become blocked, and there are a growing number of researchers who are becoming sceptical about the value of lowering cholesterol. Leading the critical charge is Dr Uffe Ravnskov, a meticulous, independent Danish researcher and author of *The Cholesterol Myths*. 'People with high cholesterol live the longest,' asserts Ravnskov. 'This

statement seems so incredible that it takes a long time to clear one's brainwashed mind to fully understand its importance. Yet the fact that people with high cholesterol live the longest emerges clearly from many scientific papers.' Nevertheless, a multi-billion-dollar industry is built on exactly the opposite hypothesis. In 2007, the global market for lipid-lowering drugs such as statins is expected to top \$30 billion.

To a non-scientist, the cholesterol hypothesis does not seem very plausible. Cholesterol is not some toxic waste that builds up in the body. It's a vital substance required in every cell. It plays an important role in repairing and protecting the body. Without cholesterol our brains would be unable to function well: it helps us form memories and facilitates the uptake of serotonin, the neurotransmitter that modulates mood, emotion, sleep and appetite. It is a precursor of the steroid hormones that determine sexuality, and regulate sugar levels and mineral metabolism. Our liver manufactures about 80 per cent of the cholesterol we use, and diet accounts for the rest. The brain manufactures its own cholesterol because the lipoproteins that transport cholesterol are too big to pass through the blood-brain barrier. Cholesterol is also responsible for the production of bile acids, without which we could not digest or absorb fats. So is such a vital substance also an agent of disease and death?

There are many other questions that cast doubt on the cholesterol hypothesis. Why do coronary heart disease-related deaths occur when cholesterol levels are low, average or high? And why do men and women with the same cholesterol levels have very different rates of heart disease? It's also puzzling that some people who die of heart disease are found to have no plaques in their arteries and low levels of blood cholesterol.

The late Professor William Stehbens of the Wellington College of Medicine spent 50 years studying atherosclerosis, the hardening of the arteries, which is the underlying cause of coronary heart disease. He was a trenchant critic of the cholesterol hypothesis. In 2001 he published two papers, 'Coronary Heart Disease, Hypercholesterolemia' and 'Atherosclerosis: I. False Premises, II Misrepresented Data', which put a large dent in the cholesterol hypothesis. As Stehbens has pointed out, atherosclerosis is a disease that affects us all: 'During the years from foetus to maturity atherosclerosis progresses when cholesterol levels are well below those allegedly preventing or inducing regression.' Cholesterol does accumulate in our arteries, but it is a manifestation that occurs as a result of the disease, not a cause of it, he argued.

Stehbens believed that atherosclerosis is caused by haemodynamics (the turbulent flow of blood). In the laboratory he was able to induce severe atherosclerosis in sheep by altering the flow of blood to mimic that in humans. The fact that as many as 50 per cent of people who have heart bypass operations go on to develop severe atherosclerosis again within 10 years seems to support this haemodynamic hypothesis. The recurrence of the disease obviously has nothing to do with cholesterol, Stehbens believed; it recurs because the grafted vein does not have the arterial architecture to support the increased volume of blood.

Other theories put forward for atherosclerosis progression include infection from a virus or bacteria that causes inflammation of the artery wall, high blood pressure on the artery lining, or high levels of the amino acid homocysteine, due to vitamin deficiency. More than 300 risk factors have been identified for coronary heart disease, including everything from obesity and smoking to baldness and earlobe creases.

It's cholesterol, however, that is endlessly promoted as the major risk factor of heart disease because there's big money in enrolling people in a lifetime drug regimen. Food manufacturers have also been quick to jump on the cholesterol bandwagon. Cholesterol-lowering margarines sell for up to three times the cost of standard margarines. Fat-free foods have proved a bonanza for supermarkets because they encourage consumers to overeat without the anxiety of increasing waistlines or cholesterol levels. Reducing saturated fat or cholesterol in your diet may do little to prevent fatal or non-fatal heart attacks. 'In fact, no clinical trial on reducing saturated fat intake has ever shown a reduction in heart disease,' says Dr Malcolm Kendrick in a Spiked (website) essay, 'The Great Cholesterol Myth'. Nor is reducing dietary cholesterol likely to be helpful. As the Framingham Heart Study concluded: 'There is no indication of a relationship between dietary cholesterol and serum cholesterol level.' Ancel Keys, the American scientist most closely associated with the cholesterol hypothesis, put it even more bluntly in 1997: 'There's no connection whatsoever between cholesterol in food and cholesterol in blood. And we've known that all along. Cholesterol in the diet doesn't matter unless you happen to be a chicken or a rabbit.'

Drugs will lower cholesterol effectively, but the benefits of taking cholesterol-lowering drugs are not as clear-cut as drug companies like to claim. For example, in the 2002 ALLHAT (Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial) involving 10,000 participants, the rates of death, heart attack and heart disease were identical among those who took cholesterol-lowering drugs, statins and those in the control group.

Statins have been lauded by some doctors almost as magic

bullets, reducing the risk of coronary death by as much as 41 per cent. It sounds impressive, but when you look at the absolute figures, it is less so; some patients may well have second thoughts about embarking on a lifetime drug regime. The 41 per cent is taken from the 4S five-year study, which trialled the drug simvastatin (marketed under various names such as Lipex and Zocor). In the trial, 5 per cent or 111 individuals died from a heart attack; in the control group, 8.5 per cent or 189 died – which makes a risk reduction of 3.5 per cent. So where does the 41 per cent come from? By dividing the 3.5 per cent difference in risk by the control group's 8.5 per cent. To put it another way, if you have had a heart attack, the chance of avoiding death from another one over five years is 91.5 per cent; if you take simvastatin, the chance increases to 95 per cent.

When you look at studies of individuals with no heart disease but high cholesterol levels, the protective benefits of statins are even smaller. If you are about 55 and your cholesterol level is around 7.00 mmol/l, your chances of avoiding death from a heart attack if you take pravastatin for five years is 98.8 per cent. If you take nothing at all, the chance drops by less than one per cent to 98.2 per cent. Since statins have the same protective effect even when cholesterol levels are normal or low, it's hard to argue that the effectiveness of statins for people with heart disease is due to cholesterol-lowering. Some have suggested that statins' success may be due to anti-inflammatory, plaque-stabilising or anti-coagulant properties.

Taking drugs is always taking a risk. Statins are considered safe drugs, but since none of the studies have been longer than five years no one knows what the long-term effects may be. Some statins have been found to stimulate cancer growth

in rodents, though the doses used in animal experiments were much higher than those recommended for clinical use. Of possibly more concern are reports from US cardiologist Dr Peter Langsjoen and others that statins can cause heart failure. Statins reduce the amount of cholesterol produced by the liver, but they also at higher doses deplete supplies of another chemical coenzyme Q₁₀, which keeps the heart healthy. Deficiencies in coenzyme Q₁₀ can lead to heart failure. The elderly are particularly susceptible.

In 2001, Bayer was forced to withdraw cerivastatin because of increasing cases of rhabdomyolysis, a rare disorder that causes muscle damage. Over 100 deaths and 1600 injuries have been linked to the drug according to Bayer. Statins can cause liver and nerve damage, mental disturbances, memory and cognition problems and a number of other side effects in a small number of people.

The US National Institutes of Health is conducting a study into the side effects of statins. While acknowledging the benefit of statins to men at high risk of heart disease, Dr Beatrice Golomb, who heads the study, says, 'However, benefit to survival with statins or other cholesterol-lowering agents has never been demonstrated in women (even those at high cardiac risk), in the older elderly, or in men at lower cardiac risk, and there are reasons to be concerned that the risk–benefit ratio may be less favourable in those groups.' As Golomb points out, after age 75 higher cholesterol is actually associated with living longer.

Many of the side effects may go unreported because some symptoms, such as memory loss and confusion, may be written off simply as old age. When Dr Duane Graveline, a former astronaut and aerospace research scientist, was prescribed a statin he found that after six weeks he began to

have memory blackouts or transient global amnesia. In his book, *Lipitor: Thief of Memory*, he recalls one weird episode, 'In this extremely sobering experience with Lipitor-associated transient global amnesia, I regressed back to my teens for twelve hours.' A fate, surely, worse than death.