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CHAPTER 3: THE TRUTH ABOUT SATURATED FAT

The Prudent Diet

In reality there has been a lot of proper experimental research carried out to discover the truth about saturated fat, even if the Secretary of State for Health has never read any of it. For example, in 1957 a group of 1,113 New York businessmen were put on what was called a 'Prudent Diet'.[29] They were aged from 40 to 59 years old, because men in that age group, especially overweight businessmen, are most at risk of heart attacks. They replaced butter with cooking oil and margarine, eggs with cold cereal and skimmed milk, and beef with chicken and fish. A second group of 467 similar men ate whatever they had been eating before. After 9 years the number of men who had developed some symptom of coronary heart disease was much higher in the second group than those in the first group who had kept to the diet.[30] So it did appear that a low-fat diet was beneficial in preventing *symptoms* of heart disease. However there were nine *deaths* from heart disease among the Prudent Dieters and *no deaths* from heart disease in the control group. I would have preferred to be in the test group of men who ate saturated fat and stayed alive.

The Multiple Risk Factor Intervention Trial

Since the Prudent Diet experiment was inconclusive, a much bigger study was organized in the 1970s. This involved 12,866 middle-aged men. They were all deliberately chosen as men who were thought to be at risk of heart failure because of their weight, blood pressure or cholesterol level. It was called the Multiple Risk Factor Intervention Trial.[31] Over an average period of 7 years half the men were encouraged, through regular visits to a clinic, to reduce their saturated fat and cholesterol consumption and increase their consumption of polyunsaturated fats (the fats that come from plants), to give up smoking cigarettes (which many of them did), and, where necessary, to lower their blood pressure by means of prescribed drugs. The other half were left to more conventional medical care.

And what was the result? The researchers wrote, '*The overall results do not show a beneficial effect on coronary heart disease or total mortality from the multifactor intervention.*' In fact in half of the 22 test centres the number of deaths from coronary heart disease was actually greater in the intervention group which had cut their saturated fat consumption, in spite of the fact that a significant number of men in this group also stopped smoking during the trial period.

The Helsinki Study

A similar study started in Helsinki in 1974, involving 1,222 businessmen who were at risk of cardiovascular disease. Half of them went on a strict low-fat diet for 5 years, reduced or cut out smoking, and were treated with drugs for high blood pressure where necessary. But in

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[29] Cristakis G. *Effect of the Anti-Coronary Club Program on Coronary Heart Disease Risk-Factor Status.* Journal of the American Medical Association, 1966; 198: 129-35.

[30] 69.0% higher for men who were 40 to 49 years old at the start of the trial, and 53.6% higher for men who were 50 to 59 years old when it started.

[31] *Multiple Risk Factor Intervention Trial*. Journal of the American Medical Association, 1982; 248:1465-77.

this experiment they also took more exercise. And guess what? The results were as bad as before for the low-fat diet supporters. After 15 years 65 men in the group who continued life as normal had died, but in the group who had been on a low-fat diet and adopted a healthier lifestyle 95 had died![32]

The Coronary Prevention Study

Again the results of the World Health Organization's European Coronary Prevention Study published in 1983 were called 'depressing' because once more no correlation between fats and heart disease was found. The researchers had cut saturated fats down to only eight per cent of the subjects' calorie intake, yet in the U.K. section there were once again more deaths in the intervention group than in the control group.[33] To be honest the researchers ought to have expected this, knowing that coronary heart disease had become an issue only since people switched from eating things like butter, lard and full-cream milk to margarine, vegetable oil and low-fat diets. One really wonders why they went on spending money trying to prove the opposite.

The Caerphilly Project

Finally I have another paper,[34] which was published in the British Journal of Nutrition in 1993. It is based on a very comprehensive 18-year study of the health of nearly all the men who were aged 45 to 59 when it first began and who lived in and around Caerphilly in Wales between 1979 and 1997 – around 2,500 of them. I say it was a very comprehensive study because it even recorded how frequently the men shaved and had sexual intercourse. One dependent study actually examined whether these factors might contribute to heart disease! The project was funded by the Medical Research Council and I believe it was managed by my old university at Bristol. Some 200 papers have been published on its findings, a remarkable indication of its value and importance. The authors of my paper were interested in the relationship between diet and IHD. IHD stands for ischaemic heart disease or reduced blood supply to the heart. This is usually caused by coronary heart disease, so the two terms are very similar. After 13 years the authors reported, '*There was some evidence suggesting a positive association between total fat intake and IHD risk, but the trend was not consistent and not statistically significant. There was no association for animal fat.*' So they found *no evidence whatsoever* that eating animal fat caused heart disease, although they did find a slight indication that other kinds of fat might be linked to heart disease. What is especially interesting is that the authors then stated that their findings were consistent with other studies, and they cited a list of them in support of this assertion.[35]

The verdict

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[32] Strandberg T E et al. *Mortality in participants and non-participants of a multifactorial prevention study of cardio-vascular diseases: a 28 year follow up of the Helsinki Businessmen Study.* British Heart Journal, 1995; 74: 449-454.

[33] World Health Organization. European Collaborative Group. *Multi-factorial trial in the prevention of coronary heart disease: 3. Incidence and mortality results. European Heart Journal*, 1983; 4:141.

[34] Fehily A M et al. *Diet and incident ischaemic heart disease: the Caerphilly Study*. British Journal of Nutrition, 1993; 69:303-314.

[35] Fehily A M et al., cited above – *Table 10: Relationship between intake of total fat and of saturated fatty acids (or animal fat) and incidence of ischaemic heart disease in major prospective studies.*

All this means it is absolutely certain that eating animal fats is not responsible for the heart disease epidemic which began around the time I was born. It is really extraordinary that the government and so many semi-official organizations continue to this day to ignore all this authoritative research. The saturated fat in butter and meat and lard and dripping and full-cream milk and full-fat cheese will not block your arteries or cause heart failure, whatever the government or the popular press or health charities or the food manufacturers may tell you.

Of course the popular view, as promulgated by the Food Standards Agency in the film I mentioned earlier, is that saturated fats cause a build-up of plaque in the arteries that eventually restricts the flow of blood to the heart causing a heart attack. The truth is that the plaque that causes our arteries to narrow doesn't build up in the arteries themselves, the channels through which the blood flows, but in the actual *wall* of the arteries. It is produced when the endothelium, the thin slippery lining of our arteries, is damaged in some way. As to what happens next, there seem to be two different stories. One is that when the damage is repaired scar tissue is formed, and if this happens repeatedly it narrows the arterial channels until a blood clot comes along and blocks the channel altogether resulting in a heart attack. The other story is that because the damaged endothelium is no longer slippery, small blood clots get stuck in it, where they turn into plaque, which again narrows the channels as before. Whichever it is, or perhaps both, it is the initial damage that produces the blood clots which block our arteries and cut off the supply of blood to the heart. Rather than blame blocked arteries on pork lard or dripping, as the film did, we have to discover what is causing the initial damage to the cell walls.

In any case meat fat doesn't go into the arteries, it goes into the stomach and intestines where it is changed into other things. And even if it did go into the arteries it would remain liquid at body temperature: it wouldn't solidify as it would if you poured it down a waste pipe, which is what the film claimed. And if fat entered our arteries directly as the film implied then so would popcorn and peanuts, and I can't imagine anything that would block our arteries more effectively than popcorn and peanuts would!

Finally, as I said before, the main component of arterial plaque, measured in autopsies of people who have died of coronary heart disease and as reported in the Lancet,[36] is not saturated fat but *polyunsaturated* fat, the very kind of fat that the film told us we *should* be eating.

So it was a silly, ignorant film, but at least it taught us something. It taught us how unscientific and misleading the government's propaganda on healthy eating can be, including that of the Food Standards Agency.

[36] Felton C V et al. *Dietary polyunsaturated fatty acids and composition of human aortic plaques*. Wynn Institute for Metabolic Research, London, U.K. Lancet, October 29, 1994; 344(8931):1195-6. (Also cited earlier.)

CHAPTER 7: BATTLE OF THE OMEGAS

Mega trouble

There are some special kinds of fat that we need in very small quantities. Nearly everyone agrees that eating the wrong amounts of them is causing some very big problems. What is not agreed is how to get the right amounts of them into our diet.

Both omega-3 and omega-6 fatty acids are essential parts of a human's diet, but consuming too much omega-6 and too little omega-3 spells mega trouble. You may have seen an advertisement for a 'healthy' spread telling you that it has 'added omega-3'. The reason that some margarine manufacturers add omega-3 fat to their products is that the seed oils from which the spreads are made contain too much omega-6 fat. Omega-3 has to be added to compensate for this. Butter is naturally healthy and doesn't *need* added omega-3, because it already contains enough and has far less omega-6.

Back in 1982 it was discovered that people who ate plenty of omega-3 fatty acids in their diets generally lived longer. In the same year, three scientists called Bergstrom, Samuelsson and Vane won a Nobel Prize for discovering that too little omega-3 produces a whole range of diseases and the reason for this. Both omega-6 and omega-3 fatty acids make some important things called signalling molecules. Signalling molecules tell our cells how to grow and repair themselves and defend themselves against infection, and they send messages around our nervous system. So you can understand that they are essential for keeping us healthy. But omega-3 and omega-6 make two different kinds of signalling molecules, which each do very different jobs. And the problem is that these two kinds of omega fats compete for the same enzyme 'food' in our bodies, rather like red and grey squirrels used to compete for similar food until the little red ones died out in most parts of Britain. So if we eat too many grey squirrel omega-6 molecules there isn't enough food in us for the little red omega-3 ones, and then they can't do their job properly.

Bergstrom and his colleagues discovered that too much omega-6 and too little omega-3 results in painful and chronic inflammation within our bodies, and it is this which causes many of the diseases people suffer from, particularly in Western nations where our diet contains far more omega-6 than omega-3. Inflammation is a major cause of coronary heart disease, many forms of cancer, asthma,[91] and various autoimmunity and neurodegenerative diseases such as coeliac disease, rheumatoid arthritis, Parkinson's and Alzheimer's diseases.[92] Doctors often treat it with nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen and naproxen, because these block the production of signalling molecules by omega-6. You've probably seen advertisements for ibuprofen on television. NSAIDs produce substantial profits for their manufacturers, costing from £2.50 to £185 a month per patient.[93] The NHS could save millions of pounds if they simply told sufferers to reduce their intake of

[91] Okuyama H et al. *Dietary Fatty Acids - The N6/N3 Balance and Chronic Elderly Diseases. Excess Linoleic Acid and Relative N-3 Deficiency Syndrome Seen in Japan*. Progress in Lipid Research, 1997; 35:4:409-457.

[92] www.drweil.com/drw/u/QAA400149/balancing-omega-3-and-omega-6.html. Accessed February 2013.

[93] US$4 to US$300 a month. Consumer reports.org. www.consumerreports.org/health/resources/pdf/best-buy-drugs/Nsaids2.pdf

omega-6 fatty acids, but of course NSAID manufacturers aren't going to mention that in their advertisements.

Too much omega-6 can also result in blood clots, high blood pressure, irritation of the digestive tract, depressed immune function, sterility, cell proliferation and weight gain.[94] It is especially likely to produce insulin resistance, which is a major cause of type 2 diabetes.[95] Margarine is a huge source of omega-6, and even bread has eight times as much omega-6 as omega-3.

Both omega-6 and omega-3 fats are polyunsaturated fats, for there are at least 40 different ones. So, while earlier research identified polyunsaturated fats in general as the causes of obesity and ill health, in more recent years it has been discovered that it is in these two particular groups of omega polyunsaturated fats where a major problem lies. So when I talk about consuming too much omega-6 I am still talking about eating too much food or oil that comes from seeds and nuts, such as polyunsaturated cooking oils, rice, flour, maize and soya products.

Too little omega-3 brings problems of its own. Omega-3 is essential for the proper development of the eye retina and hence for sight. Children whose mothers ate oily fish (the major source of omega-3) during their pregnancy tend to have better eyesight than other children.[96] Omega-3 fats are essential for the proper development and functioning of the brain, so a lack of them can cause mental problems. A lack of omega-3, especially before and after birth, has been associated with learning difficulties.[97] It can result in depression,[98,99,100,101] dyslexia, hyperactivity and even violent and criminal behaviour.[102] In several studies it was found that giving omega-3 supplements to violent young men in prison reduced rule breaking, aggressive behaviour and violent incidents.[103,104] When the

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[94] Horrobin D F. *The regulation of prostaglandin biosynthesis by the manipulation of essential fatty acid metabolism.* Reviews in Pure and Applied Pharmacological Sciences, 1983; 4: 339-383;
Devlin T M, ed. Textbook of Biochemistry, 2nd Edition, 1982, Wiley Medical, 429-430;
Fallon S & Enig M G. *Tripping Lightly Down the Prostaglandin Pathway*s. Price-Pottenger Nutrition Foundation Health Journal, 1996; 20:3:5-8.

[95] Berry E M. *Are diets high in omega-6 polyunsaturated fatty acids unhealthy?* European Heart Journal Supplements, 2001, 3 (Supplement D); D37–D41.

[96] Williams C at al. (Longitudinal Study of Pregnancy and Childhood Study Team.) *Stereoacuity at age 3.5 years in children born full term is associated with prenatal and postnatal dietary factors: a report from a population-based cohort study*. American Journal of Clinical Nutrition, 2001; 73:316-322.

[97] Okuyama H et al. *Dietary Fatty Acids - the N-6/N-3 Balance and Chronic Diseases. Excess Linoleic Acid and the Relative N-3 Deficiency Syndrome Seen in Japan*. Progress in Lipid Research, 1997; 35:4:409-457. (Also cited earlier.)

[98] Maes M et al. *Fatty acid composition in major depression: decreased omega-3 fractions in cholesteryl esters and increased C20: 4 omega-6/C20:5 omega-3 ratio in cholesteryl esters and phospholipids.* Journal of Affective Disorders, 1996; 38:35–46.

[99] Edwards R et al. *Omega-3 polyunsaturated fatty acid levels in the diet and in red blood cell membranes of depressed patients*. Journal of Affective Disorders, 1998; 48:149–55.

[100] Peet M et al. *Depletion of omega-3 fatty acid levels in red blood cell membranes of depressive patients*. Biological Psychiatry, 1998; 43:315–9.

[101] Maes M et al. *Lowered omega-3 polyunsaturated fatty acids in serum phospholipids and cholesteryl esters of depressed patients.* Psychiatry Research, 1999; 85:275–91.

[102] www.drweil.com/drw/u/QAA400149/balancing-omega-3-and-omega-6.html. Accessed February 2013.

[103] Gesch CB at al. *Influence of supplementary vitamins, minerals and essential fatty acids on the antisocial behavior of young adult prisoners. Randomised, placebo-controlled trial*. British Journal of Psychiatry 2002; 181;22–28.

supplements stopped the violent behaviour resumed.[105] Before going to a psychologist, any parent with a depressed, dyslexic, hyperactive or violent child should consider what he is eating. Mega problems may have an o-mega cause.

One other effect of a lack of omega-3, which is especially interesting to me as an oldie and has only recently been discovered, is that people who consume more omega-3 fatty acids do not age so quickly and therefore live longer.[106] Remember you'll be an oldie one day, if you remember your Highway Code and the world doesn't end first!

It is not the amount of omega-3 we consume that matters so much as the relative amounts of omega-6 and omega-3, in other words how badly the grey squirrels outnumber the red ones. Let's call the ratio between the two kinds the O-6-3 ratio. In the diet of Greenland Eskimos (more correctly the Inuits) the O-6-3 ratio has been estimated at 1:1, i.e. equal quantities of omega-6 and omega-3. In the traditional Japanese diet it has been estimated at around 4:1, or four times as much omega-6 as omega-3. Typical European diets however provide O-6-3 ratios of between 10:1 and 14:1;[107] in the U.S.A. the ratio is higher, and in Israel, where there is a particularly high prevalence of cardiovascular diseases, hypertension, type 2 diabetes, obesity and cancer, the ratio is as high as 26:1.[108] Contrast these O-6-3 ratios in the Western world with values of between 1:1 and 4:1 on which humans are thought to have evolved,[109] and you can see how much our Western diet has been altered by commercial food manufacturers.

Look at the extraordinary chart below in Figure 16. It shows an almost exact association between the death rates from heart attacks and the percentage of omega-6 fats in the highly unsaturated fatty acids (HUFA[110]) found in the body tissues of various population groups. As the percentage of omega-6 fats in a population group increases, its death rate increases in exactly the same proportion. This is the chart that Ancel Keyes should have shown the world back in 1953. Then we wouldn't have had all the nonsense about saturated fat causing heart attacks.

Figure 16: Association between percentage of omega-6 in highly unsaturated fatty acids and coronary heart disease death rates[111]

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[104] Hibbeln J R et al. *Omega-3 fatty acid deficiencies in neurodevelopment, aggression and autonomic dysregulation: Opportunities for intervention.* International Review of Psychiatry, April 2006; 18(2): 107–118.

[105] Zaalbergl A et al. *Effects of Nutritional Supplements on Aggression, Rule-Breaking, and Psychopathology Among Young Adult Prisoners.* Aggressive Behaviour, Volume 36, pages 117–126, 2010.

[106] Farzaneh-Far R et al. *Association of marine omega-3 fatty acid levels with telomeric aging in patients with coronary heart disease.* Journal of American Medical Association, Vol. 303, No. 3, January 20, 2010.

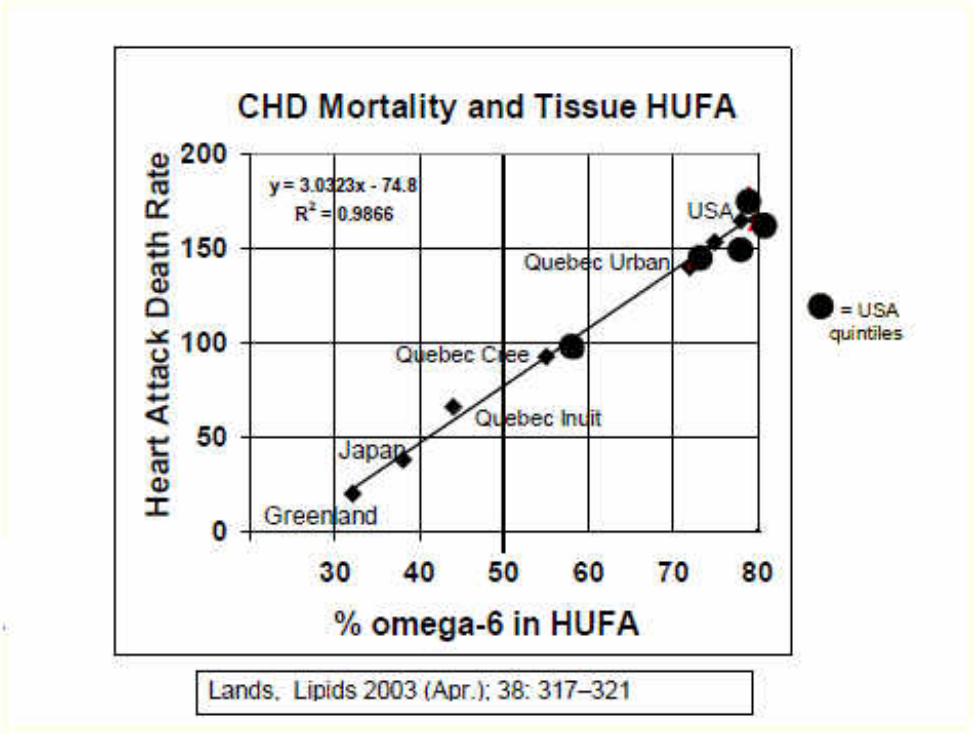
[107] *Fats and oils in human nutrition: Report of a Joint Expert Consultation.* FAO Food and Nutrition Paper No. 57, 1994.

[108] Berry E M. *Are diets high in omega-6 polyunsaturated fatty acids unhealthy?* European Heart Journal Supplements, 2001, 3 (Supplement D); D37–D41. (Also cited earlier.)

[109] Budowski P & Crawford M A. *Alpha linolenic acid as a regulator of the metabolism of arachidonic acid: dietary implications of the ratio, n-6:n-3 fatty acids*. Proceedings of the Nutrition Society, 1985; 44: 221–9.

[110] Highly unsaturated fatty acids are polyunsaturated fats that contain several double chemical bonds in each molecule and are therefore easily oxidized.

[111] Lands W E M. *Diets could prevent many diseases*. Lipids, 38:317-21, 2003.



More recently Dr. Bill Lands, who produced the chart in Figure 16, has shown that the *difference* between the amounts of omega-6 and omega-3 in our diet can give a good estimation of the percentage of omega-6 that will end up in our tissues, and hence a good estimation of the risk of a heart attack.[112] In other words, what matters is both the ratio of omega-6 to omega-3 in a particular food and how much of it we eat. If some food has a bad, high ratio but we eat only a tiny amount of it then our bodies will cope; but if the ratio in a particular food is only moderately high and we virtually live on it, then we probably won't live on it much longer!

"That's all very well," you might say, "but how can one possibly create a family menu that provides an O-6-3 ratio of less than four? It's complicated enough trying to count calories." Certainly most parents wouldn't have the time, inclination or ability to add up the omega contents of food while they are doing their shopping, and even if they wanted to they would find that very few products display the amounts of omega-3 and omega-6 in their nutritional information.

There are three possible ways to correct an imbalance in the omega ratio:

(i) Eat more food that's high in omega-3
(ii) Take omega-3 supplements, e.g. fish oil capsules
(iii)Eat less food that's high in omega-6

Let's look at these three options in turn.

Solution 1: Eat more food that's high in omega-3

[112] Lands B & Lamoreaux E. *Using 3–6 differences in essential fatty acids rather than 3/6 ratios gives useful food balance scores*. Nutrition & Metabolism, 2012; 9:46.

It's extraordinary, but the only substantial source of omega-3 fatty acids in the form our body needs them comes from the sea. Some omega-3 can be obtained from grass-fed meat, poultry fed on grass and insects, and eggs from such poultry, and the body can obtain a certain amount in the forms that it needs from plants, but by far the best source of omega-3 is fish, and in particular oily fish. Oily fish also contain vitamins A and D, so it is doubly good for you.

Oily fish are fish that store their fat in their flesh. Some common types are:

- herrings (which include kippers and bloaters)
- sardines and pilchards (pilchards are large sardines)
- mackerel
- salmon
- fresh or frozen tuna
- anchovies
- swordfish
- halibut
- trout
- sprats
- whitebait
- carp

There is a much fuller list of oily fish as well as non-oily fish in Annexe 5. Herrings have the most omega-3 by weight, with the others in approximately descending order. Fish roe, weight for weight, is even better than herrings. Crabs and shellfish are other good sources of omega-3, but the best source of all is caviar. Caviar has three times as much omega-3 as the same weight of herrings, but you'll probably need a much better paid job before you can afford to buy Royal Beluga caviar for your family at £2,570 per kilogram!

In general the omega-3 content of any particular species of fish is similar whether it is fresh, frozen or canned. Fish canned in oil loses some omega-3 because fatty acids dissolve in oil, so when you pour the oil away you pour away some of the omega-3 too.[113] You could drink the oil as well as eat the fish if you wanted to, but whatever you do don't drink it if it is sunflower oil because its omega content is 100% omega-6 so it will completely undo the good of the omega-3 in the fish.[114] Even olive oil has more omega-6 than is really good for you. The healthiest canning mediums for fish are spring water and brine (salt water). Canned tuna however contains very little omega-3 whatever it is canned in, so you can't count canned tuna towards your oily fish consumption.

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[113] Tesco states that the omega-3 content of its canned fish is the same whether it is canned in oil or water. I find this hard to understand, especially since Glenryck Ltd states that its pilchards canned in oil contain only half the amount of EPA + DHA that its pilchards in water contain. EPA and DHA are the kinds of omega-3 found in fish which our bodies need. Perhaps the loss of these in Tesco fish canned in oil is made up for by linoleic acid or LA, a kind of omega-3 found in oils such as olive oil and sunflower oil, but which is of far less nutritional value.

[114] A 125gm tin of typical mackerel fillets in sunflower oil contains 90gm of mackerel fillets and 35gm of oil. The mackerel contains 2.8 x 90 / 100 = 2.52gm of omega-3. The sunflower oil contains 22.7 x 35 / 100 = 7.95gm of omega-6. The combined O-6-3 ratio is therefore 7.95/2.52 = 3.15:1. This is within the healthy range of 4:1 and 1:1, but it will not help to bring a high dietary O-6-3 ratio down to a healthy level, which eating only the mackerel would do.

Non-oily white fish such as cod, haddock and plaice also contains omega-3 and vitamins, but not nearly so much as oily fish does. However cod liver oil is an excellent source of omega-3, because non-oily fish store their fat in their liver. Of course cod liver oil on its own doesn't provide the protein that eating the fish itself does.

So, eating oily fish will increase our omega-3 consumption, but the question is, how much fish do we need to eat before the O-6-3 balance in our diet is at a healthy level? Naturally the answer to that question depends on how much omega-6 we are consuming. Unfortunately all the research into this subject, so far as I know, has been carried out on people who are on a typical Western diet, so the resulting recommendations really apply only to people who are eating high amounts of omega-6, but at least this can give us a start.

The British Food Standards Agency (FSA) says that everyone should eat 'at least' two 140gm portions of fish a week, including one portion of oily fish. However, women who are likely to have a child, and pregnant and nursing women, should eat no more than two portions a week of oily fish, and other people should eat no more than four.[115] The upper limit is set because of concerns about harmful contaminants in sea water such as mercury and dioxins that may get into some fish.

The Scientific Advisory Committee on Nutrition (SACN), which advises the FSA, makes the same recommendations, but adds that everyone should consume a minimum of 450mg per day or 3,150mg per week of 'long-chain omega-3 fatty acids'.[116] For children the International Cod Liver Omega-3 Foundation suggests a minimum intake of 200mg per day or 1,400mg a week.

The two most important long-chain omega-3 fatty acids so far as our diet is concerned are eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), and those are the two main kinds of omega-3 found in fish. A third one, docosapentaenoic acid or DPA, is found in fish in smaller quantities. Another kind of omega-3 is alpha-linolenic acid or ALA. This is found in plants, and the body can convert it to EPA and DHA, so it is an essential source of omega-3 for vegetarians. However, some studies have indicated that ALA is ineffective in reducing the likelihood of a heart attack or the recurrence of one.[117]

In terms of fish, a 140gm portion of the oily fish salmon contains about 2,800mg of EPA and DHA and a similar portion of the non-oily fish plaice contains about 350mg, so a weekly portion of oily fish and one of non-oily fish would provide the 3,150mg weekly ration recommended by SACN for adults. For children two 60gm portions a week would provide the recommended 1,400mg of omega-3.

While at least two portions of fish a week is the official advice given in the U.K., the words 'at least' cover up the fact that for people on a typical Western diet two portions are not nearly enough to keep us healthy, as many other authorities show. Table 2 summarizes the advice from the FSA and other bodies.

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[115] www.food.gov.uk/multimedia/faq/oilyfishfaq/#.URpmq5Zs9aY June 2004. Accessed March 2013.

[116] Scientific Advisory Committee on Nutrition, FICS/04/02, dated 14/04/04. www.sacn.gov.uk/pdfs/fics_sacn_04_02.pdf. Accessed March 2013.

[117] Wang C at al. *n-3 Fatty acids from fish or fish-oil supplements, but not alpha-linolenic acid, benefit cardiovascular disease outcomes in primary- and secondary-prevention studies: a systematic review*. American Journal of Clinical Nutrition, July 2006; 84(1):5-17.

Table 2: Recommended mixed oily and non-oily fish consumption necessary for cardiac and other health on a Western diet[118]

Source	Recommended number of 140gm portions of mixed fish per week	Comments
UK Food Standards Agency	2 at least	With limitations on maximum consumption for women who may have a child and for pregnant and nursing mothers.
UK Scientific Advisory Committee on Nutrition	2 or 3	To reduce the risk of heart trouble, from various sources.
	4	For people over 65 or those who have survived some form of heart attack.
	6 at least	To significantly reduce the risk of heart trouble.
American Heart Association	2	'Especially oily fish'. With limitations on certain species for pregnant and nursing mothers and young children.
	4 at least	People who have had a heart attack.
	9 to 18	Equivalent in capsule form for people who need to lower their triglyceride level.
Holland & Barrett	Up to 7 portions of fish, or the equivalent in fish oil.	From various cited research sources, for protection from heart trouble, lupus and rheumatoid arthritis.
US National Institutes of Health	9 or the equivalent in fish oil.	For someone on a 2,000Kcal a day diet, or in proportion.
It may take from 2 to 6 months for the benefits of increasing one's intake of fish oil to be measurable.		

So there you go. Table 2 shows that to be absolutely certain that our diet isn't contributing to a heart attack or other health problems we should be eating six to nine portions of uncontaminated fish a week! This is completely unrealistic, unless one is an Eskimo or a shark. Bearing in mind that on average British people eat only a little over one portion of fish a week, if we all ate only the FSA's minimum recommendation of two portions a week, twice as many fish would have to be taken out of the sea, and it is doubtful if the already depleted fish stocks could survive this. That may be why the government recommends only two portions a week, even though its own Scientific Advisory Committee says we ought to eat at least six portions a week to reduce the risk of heart disease significantly. So let's look at the second solution.

Solution 2: Take omega-3 supplements

Omega-3 supplements come in several forms:

- fish oil and cod liver oil capsules

[118] See Annexe 6 for detailed references and the derivation of the quoted numbers.

- krill oil capsules
- flaxseed oil capsules
- omega-3 enriched foods

In general, governments don't recommend the use of such supplements, although the NHS in Britain does provide vouchers for free vitamin drops that contain fish oil for children up to their fourth birthday, if you can manage to get hold of them. While the US Food and Drug Administration (FDA) hasn't published any recommendations for omega-3 supplementation, the American Heart Association (AHA) suggests that people who have been diagnosed as having coronary heart disease should consume 1gm a day of omega-3 (EPA + DHA), either from oily fish or, in consultation with a doctor, from fish oil capsules. The AHA also says that people who need to lower their triglyceride level should take 2gm to 4gm a day of EPA + DHA in fish oil capsules.

There is no apparent reason why governments should not recommend omega-3 supplementation, unless it is that they don't want to admit their existing dietary advice is inadequate, so many people do buy omega-3 supplements in order to keep themselves healthy. However a lot of this is needed in order to provide a significant benefit, as I shall now explain.

(i) Fish oil and cod liver oil capsules

Some Norwegian researchers discovered that over an 8-week period salmon raised the level of EPA + DHA in the blood more than cod liver oil did, even though the amount of cod liver oil they provided contained two and a half times as much EPA + DHA as the salmon did.[119] Another study demonstrated that to obtain the same increase in EPA and DHA in the blood one needs twice as much EPA from fish oil as from salmon, and *nine times as much* DHA.[120] Therefore in this section I am going to assume that to supplement our omega-3 intake from cod liver oil or fish oil we need to consume about three times as much EPA + DHA as we would if we obtained it from fish.

(ii) Krill oil

Other researchers in Norway found that krill oil was more beneficial than fish oil. Krill are small shrimp-like crustaceans on which many fish feed. Krill oil is an increasingly popular source of omega-3: for instance the British eBay website lists well over 200 products and the American version some 450. However there are concerns that unrestricted krill trawling in the south Atlantic and other places will in time deplete the fish stocks. The researchers discovered that both krill oil and fish oil raised the level of EPA + DHA in the subjects' blood by a similar amount, but the krill oil dosage contained only 62.8% as much EPA + DHA as the fish oil dosage did.[121] Assuming that the fish oil used in the research was similar in its effects to pure cod liver oil this means that to supplement omega-3 from krill oil one has to consume about twice as much EPA + DHA as one would if one obtained it from fish.

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[119] Elvevoll EO et al. *Enhanced incorporation of n-3 fatty acids from fish compared with fish oils.* Lipids, December 2006; 41(12):1109-14.

[120] Visioli F et al. *Dietary intake of fish vs. formulations leads to higher plasma concentrations of n-3 fatty acids*. Lipids, 38(4):415-8, April 2003.

[121] Ulven S M et al. *Metabolic Effects of Krill Oil are Essentially Similar to Those of Fish Oil but at Lower Dose of EPA and DHA, in Healthy Volunteers*. Lipids, 46 (1): 37–46. January 2011.

(iii) Flaxseed oil

Flaxseed oil is an excellent source of omega-3 in the form of ALA, but only a small proportion of ALA is converted by our bodies into the EPA + DHA that they need, at least by men on a typical Western diet. The proportion is higher for women and for people who do not ingest a lot of omega-6 fatty acids. The measured conversion rate varies from 0.2%[122] to 15%,[123] with 5% being the most commonly accepted figure.[124] All researchers report that the conversion rate for DHA, the kind of omega-3 needed by our nerve and brain cells, is much lower than for EPA.

Some researchers in North Dakota compared the effectiveness of flax oil and fish oil.[125] A company of firefighters, a group of men considered to be at risk of heart trouble due to stress and relatively poor diet, were given daily capsules of fish oil or flax oil over a period of 12 weeks. Both were effective in raising the levels of EPA, DHA and DPA in the blood, but the fish oil was much more effective than the flax oil, as shown in Table 3.

Table 3: Percentage increase in base level of EPA, DHA and DPA in the blood after 12 weeks of omega-3 supplementation among four experimental groups of men

Oil	No. of capsules per day	Omega-3 dose per day in milligrams	Percentage increase in blood EPA + DHA + DPA after 12 weeks
Fish	1	268mg EPA + DHA + DPA	123%
Fish	2	536mg EPA + DHA + DPA	187%
Flaxseed (linseed)	4	2,392mg ALA	54%
Flaxseed (linseed)	6	3,588mg ALA	71%

Using average values of the figures above it appears to obtain the same benefit from flax oil as from fish oil one needs 18.4 times as much ALA from flax seed oil as EPA + DHA + DPA from fish oil. This means that in order to produce a similar benefit from flax oil as from fish itself one needs 3 x 18.4 = 55 times as much ALA from flax oil as EPA + DHA + DPA from fish!

(iv) Milled flaxseed

Flax seeds have a hard shell, so in order to digest them they must either be toasted for 5 to 10 minutes in a skillet or in the oven at 190^{o}C, or else they must be ground or milled. In the latter case they must be kept in an airtight container to protect them from oxidation.

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[122] Pawlosky R J et al. *Physiological compartmental analysis of alpha-linolenic acid metabolism in adult humans.* Journal of Lipid Research, 2001; 42:1257–1265.

[123] Emken E A et al. *Dietary linoleic acid influences desaturation and acylation of deuterium-labeled linoleic and linolenic acids in young adult males.* Biochemica et Biophysica Acta, 1994; 1213:277–288.

[124] Gerster H. *Can adults adequately convert alpha-linolenic acid (18:3n-3) to eicosapentaenoic acid (20:5n-3) and docosahexaenoic acid (22:6n-3)?* International Journal for Vitamin and Nutrition Research, 1998(2); 68 (3):159–173.

[125] Barceló-Coblijn G et al. *Flaxseed oil and fish-oil capsule consumption alters human red blood cell n-3 fatty acid composition: a multiple-dosing trial comparing 2 sources of n-3 fatty acid.* American Journal of Clinical Nutrition, September 2008; 88(3):801-9.

(v) Required quantities of omega-3 supplements

Earlier, I said that for people on a typical British diet who have not experienced heart trouble SACN recommends a minimum consumption of 3,150mg of omega-3 a week from oily and non-oily fish, which is equivalent to a 140gm serving of salmon and a 140gm serving of cod. So to obtain a similar benefit from supplements you would need to consume two times as much omega-3 from krill oil, three times as much from fish oil, or 55 times as much from flaxseed oil. Table 4 shows the resulting minimum recommended amounts of each supplement for adults, together with the corresponding costs of typical products in the U.K. at the time of writing. As previously mentioned, it has been suggested that children should obtain a minimum of 1,400mg of omega-3 a week, so for children the quantities and costs shown in the table should be reduced by a factor of 1,400/3,150 = 0.44. The cost of some items may be considerably less in North America.

Table 4: Methods of obtaining a week's minimum recommended intake of 3,150mg EPA + DHA from fish or the equivalent benefit from oil, and some representative costs in the U.K.

Source	Description	Conversion factor	Amount required per week	Cost [1]
Oily fish + non-oily fish, fillets	Frozen salmon fillet + frozen cod fillet (Tesco)	1	One 140gm portion of each	£2.50
Oily fish + non-oily fish, canned	Sardines canned in brine + tuna canned in brine (Tesco)	1	One 120gm tin of sardines + one 185gm tin of tuna	£1.17
Cod liver oil	1,000ml costs £13.48. 10ml contains 1,400mg EPA + DHA. (Holland & Barrett (H & B))	3	67.5ml	£0.91
Cod liver oil capsules	240 Seven Seas high strength cod liver oil capsules cost £15.37. (H & B) 1 capsule contains 180mg EPA + DHA.	3	52 capsules	£3.33
Krill oil capsules	60 omega-3 krill oil capsules cost £20.23. (H & B) 1 capsule contains 120mg EPA + DHA.	2	52 capsules	£17.70
Flaxseed oil	946ml flaxseed oil costs £19.57. (H & B) 10ml contain 4,660mg ALA.	55	372ml	£7.69
Flaxseed oil capsules	240 flaxseed oil capsules cost £40.04. (H & B) 2 capsules contain 1,872mg ALA.	55	185 capsules	£30.86
Milled flaxseed powder	600gm costs £6.73 (H & B). 10gm contains 2,100mg ALA.	55	825gm	£9.25

Hemp oil	520ml of GranoVita hemp oil made from organically grown hemp seed costs £10.78 (H & B). 10ml contains 1,660mg ALA.	55 (Assumed the same as for flax oil.)	1,044ml	£21.64
[1] Based on some advertised prices in March 2013				

The costs shown in Table 4 are per adult per week, so it's obvious that, apart from free vitamin drops, anything other than fish or bottled cod liver oil would be too expensive for the average family. For many people, even fish fillets are a luxury. And drinking 372ml a week of flaxseed (linseed) oil is unthinkable.

(vi) Omega-3 enriched foods

One other way of increasing one's intake of omega-3 in a small way is to eat foods that have omega-3 added to them. As previously mentioned, margarine manufacturers add omega-3 to make up for the excessive omega-6 in the margarine itself, and omega-3 is also added to some juice drinks, breakfast cereals, milk, cheese, eggs and even pet food! In most cases the words 'with added omega-3' are little more than an advertising gimmick, for the tiny amount added makes no significant difference whatsoever.

Flora Omega-3 Plus spread, for example, contains fish oil; hence it is advertised as containing two kinds of omega-3, EPA and DHA. But only 1.8% of it is fish oil. Nearly all the rest comprises vegetable oils in which the omega fatty acids are mainly omega-6. Therefore although it does contain omega-3 this hardly helps to redress the imbalance between the two kinds of omega fats.

Omega-3 is 'added' to some eggs by feeding the hens with canola (rapeseed) oil. This oil has a healthy O-6-3 ratio of 2:1, but because rapeseed is a plant, the omega-3 part is mainly ALA, and the amount of ALA in an egg that is converted to EPA and DHA in our bodies is so small that again it is of minimal benefit.

Some cows have fish oil added to their diet, so that dairy producers can advertise 'omega-3 enhanced' milk, butter or cheese and sell them at a premium. Doing this does increase the omega-3 content, but not by very much, and whether this is a good use of the world's dwindling stocks of fish is open to question. In any case natural milk from cows fed on grass has an excellent O-6-3 ratio of 1.5:1 so its omega-3 content needs no enhancing.

Birds Eye 'Omega-3 fish fingers' also contain a little added fish oil, and they are made from pollock, which contains twice as much omega-3 as cod does. However, pollock still contains only a quarter as much omega-3 as mackerel and herring, and even in these good quality fish fingers only 58% is actually fish. The remaining 42% is the surrounding crumb, and this contains omega-6 fats that offset the benefit of the omega-3 in the fish.

(vii) Omega-3 supplements: conclusions

Clearly the cheapest effective way to supplement your omega-3 supplement is not to pay extra for omega-3 enhanced foods, but to drink bottled cod liver oil or fish oil.

But is even that such a good idea? Fish itself contains protein (essential for growth and for the repair and regular replacement of nerves, tissues and bones, as well as combating infection), vitamin A (important for night vision and other things) and vitamin D (important for masses of reasons). Fish also contains selenium and other important minerals. Apart from a little vitamin D in cod liver oil, omega-3 capsules of all kinds contain almost none of these essential nutrients.

For only a little more than the cost of fish oil or cod liver oil you can buy the equivalent amount of canned fish and have it as part of a meal. A breakfast of half a tin of sardines on buttered toast would actually be cheaper than an equivalent dose of cod liver oil with a bowl of cornflakes and milk, and it would provide a better O-6-3 balance.

So don't take omega-3 supplements, eat fish instead! Cod liver oil capsules and krill and flaxseed oil are too expensive in the quantities required to be effective, and they are all nutritionally inferior to fish, whether fresh, frozen or canned. But much more than this, there is one reason why no responsible person should try to supplement his omega-3 intake from fish oil.

Fish *oil* isn't made from fresh air: it comes from fish. And as we have seen, in order to obtain the same amount of EPA and DHA in the blood from fish oil as from fish one has to consume *three times as much EPA and DHA*. That's why there is a factor of three against cod liver oil in Table 4. Now let's say that eating just the minimum recommended two portions of fish a week means killing two whole fish a month. If instead we obtain our minimum recommended omega-3 supplement from fish oil instead of fish, *six* fish will have to be killed every month! Currently, as I said, people in the U.K. eat on average only a little over one portion of fish a week, and even with that small amount the fish stocks in the North Sea are depleted. If we all supplemented our omega-3 by means of fish oil to obtain the same benefit as eating two portions of fish a week, then we would have to kill *six times* as many fish as at present, and obviously there would be none left within a year. Can it possibly be called responsible and unselfish to practise something that would create an environmental disaster if everybody did it?

So what on earth is the solution? How can we tip the omega scales in the other direction, if it can't be done by eating fish or by dietary supplements? The solution is very simple, but it is one our governments continually shy away from. It is this: we have to reduce our intake of omega-6.[126]

Solution 3: Eat less high omega-6 food

The only sensible way to reach a healthy balance of omega-3 and omega-6 fatty acids is to reduce our intake of omega-6: to get rid of some of those grey squirrels that are gobbling up all the red squirrels' food inside us. You would think this is obvious, but far from encouraging us to do it, the government tells us to use high omega-6 vegetable-based spreads and cooking oils instead of the traditional butter, lard and dripping in which omega-6 and omega-3 fats are in a healthy balance. On top of this they tell us to obtain most of our calories from starchy foods such as bread and breakfast cereals and potatoes and rice. Potatoes are

[126] Simopoulos A P et al. *Workshop on the Essentiality of and Recommended Dietary Intakes for Omega-6 and Omega-3 Fatty Acids.* Journal of the American College of Nutrition, Vol. 18, No. 5, 487–489, 1999.

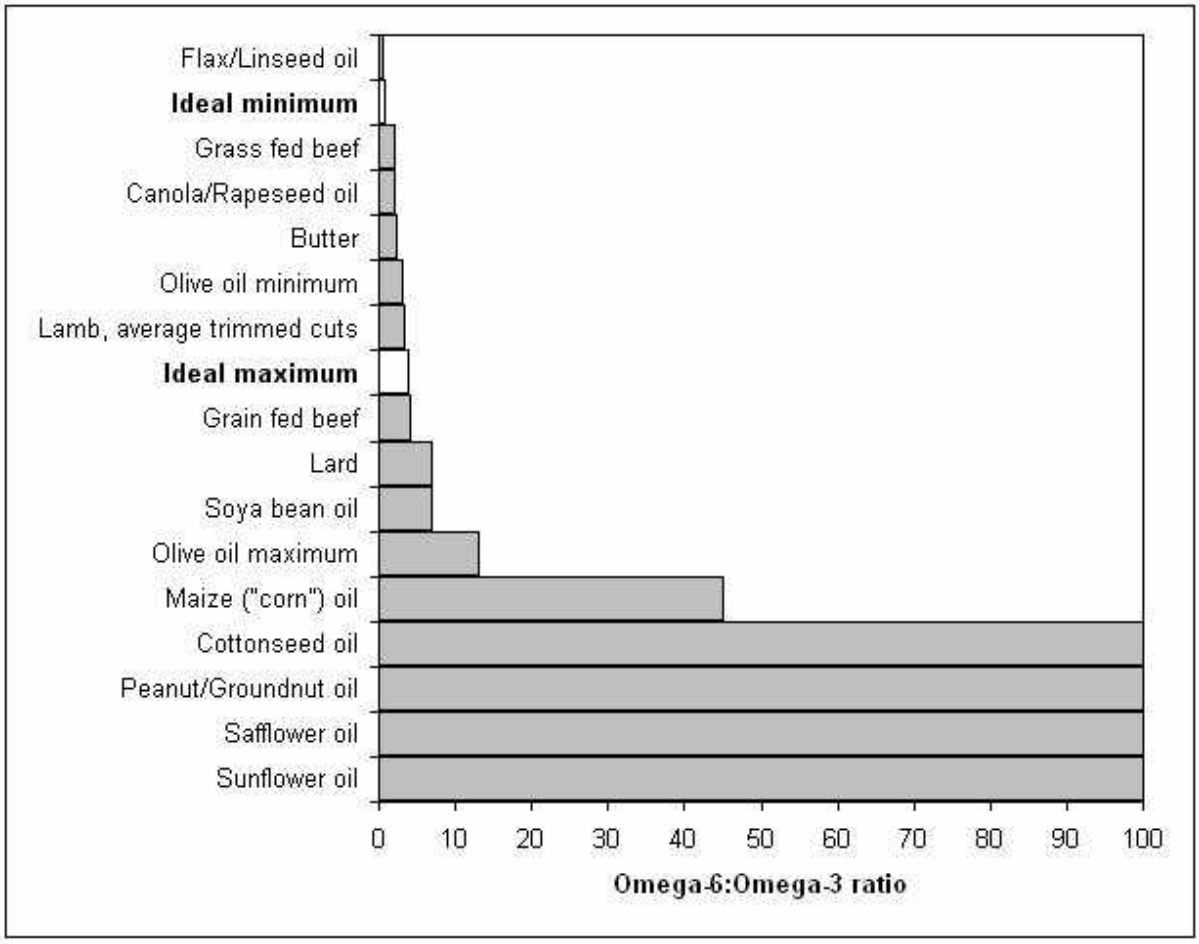
fine and rice isn't too bad, but wheat flour, which is the basis of most bread and pastry and cakes, has a dreadful O-6-3 ratio of 17:1.

Food manufacturers introduce this same problem very early on in life. A popular toddler milk sold in the U.K. as 'nutritionally superior to cow's milk' has an O-6-3 ratio of 10:1, a rather strange claim if you recall that the ratio for natural cow's milk is a very healthy 1.5:1. Some toddler cereals that are widely used in the U.S.A. have ratios as high as 137:1![127] It is hardly surprising that diet-related health problems are affecting even children.

Bearing in mind that a healthy O-6-3 ratio lies between 1:1 and 4:1, Figures 17 and 18 clearly demonstrate why the ratios in our current Western diets are so unhealthily high.

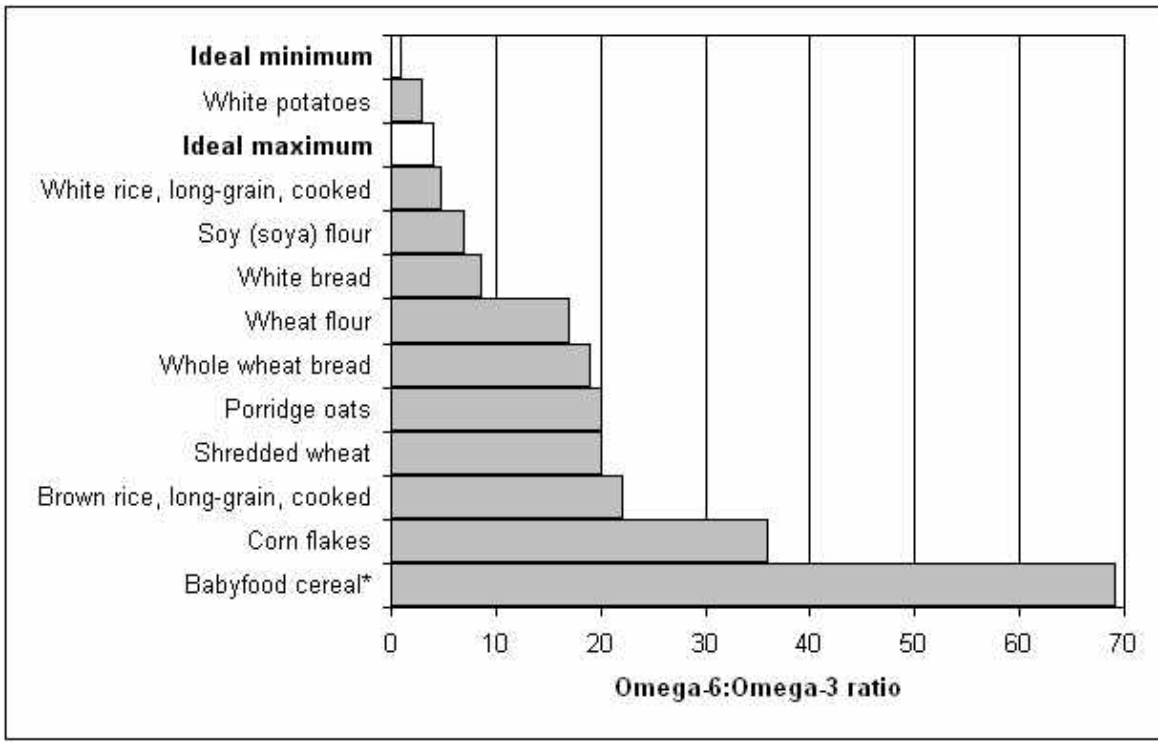
[127] *Babyfood cereal. Junior oatmeal cereal with apple sauce and banana.* Nutrition Tables, United States Department of Agriculture.

Figure 17: Ratios of omega-6 to omega-3 for some common sources of fat[128]



[128] Various sources

Figure 18: Ratios of omega-6 to omega-3 for potatoes and some common cereals[129]



* Babyfood cereal. Junior oatmeal cereal with apple sauce and banana: O-6-3 ratio = 69:1 or 137:1. (USDA nutrition database. Two values are given.)

As you can see in Figures 17 and 18, some foods like beef and butter and potatoes do have omega-6 and omega-3 fats in a healthy ratio between 1:1 and 4:1. But let's look at a few others.

Flax oil, otherwise known as linseed oil, has an O-6-3 ratio of only 0.25:1 or 0.5:1, depending on whom you believe. Whichever is right, it has *more* omega-3 than omega-6, so it is a useful source of omega-3 for vegetarians. Weight for weight it has six times as much omega-3 per gm as most fish oils,[130] but as it is plant-based the kind of omega-3 it contains (alpha linolenic acid or ALA) is not nearly so nutritious as the kinds of omega-3 found in fish, meat and eggs. Assuming, as previously discussed, that only 5% of the ALA is converted to EPA and DHA, we would still have to drink three and a half times as much flax oil as fish oil to obtain a similar result. Added to that, ALA is probably not as effective as fish oil in preventing heart attacks or their recurrence, as previously mentioned, and the high proportion of polyunsaturated fatty acids in it makes it unsuitable for cooking purposes, because heating polyunsaturated fats makes them oxidize and produce harmful free radicals. It would however be fine used cold in a salad dressing.

Canola or rapeseed oil is generally labelled as 'vegetable oil' in the U.K. It is actually a modified form of rapeseed oil and, as shown on the first chart, it has a healthy O-6-3 ratio of 2:1. However 28% of the fat in rapeseed oil is polyunsaturated fat, as compared with 11% in lard and only 3% in grass-fed dripping (beef suet), so once again it isn't nearly so healthy for

[129] Source: U.S. Department of Agriculture Food Database

[130] Bartram T. *Bartram's Encyclopedia of Herbal Medicine: The Definitive Guide to the Herbal Treatments of Diseases*. Da Capo Press, September 2002, p. 271.

cooking purposes. And since the extraction process involves heating it to temperatures up to 260°C much of the polyunsaturated fat may be oxidized before you even purchase it, at least according to Mark Sissons, author of '*The Primal Blueprint*'.[131] It is obvious that the healthiest fats for frying and roasting are dripping and lard, because they consist primarily of saturated and monounsaturated fats, which do not easily oxidize and go rancid.

The contribution of beef to the omega balance depends very much on how the cattle are raised. In 2006-2009 the O-6-3 ratio for grass-fed Angus cross steers in the U.S.A. was measured as 1.65:1, while for grain-finished beef (beef fattened with grain prior to slaughter) it was 4.84:1.[132] The grass-fed beef had the same amount of protein in it, but more beta-carotene, B-vitamins and minerals; twice as much conjugated linoleic acid (which fights cancer); three times as much total omega-3; and nearly four times as much vitamin E!

For lard, the rather high 6:1 ratio shown in the first chart is very much an average value. The actual ratio again depends on what the poor old pig ate while it was alive. Lard from free-range pigs fed naturally on grass and roots, etc., has a healthy O-6-3 ratio of around 3:1. But lard from pigs kept in pens and fed on maize, soya and other industrially grown crops and offscourings can have a ratio as high as 33:1![133] However, only 11% of lard consists of polyunsaturated fat (the kind that contains omega fatty acids), whereas almost 70% of normal sunflower oil consists of polyunsaturated fat, mostly omega-6, so even the worst kind of lard contains far less omega-6 than sunflower and similar vegetable oils do. While most British supermarkets sell dripping made from British beef without any additives, supermarket lard in Britain generally comes from unspecified countries and its purity is not quite so certain. In Europe, the 2013 horsemeat saga demonstrated how hard it is to know for certain what ingredients supermarket food really contains. So why not ask your local butcher for advice on obtaining pure dripping, lard and other cooking fats from pasture-fed animals?

The last three oils shown on the fats bar chart are cottonseed, peanut and sunflower oil. These really have ratios of over 100:1 for they contain almost no omega-3 at all. There is a special type of sunflower oil called high oleic sunflower oil which is much healthier, but it is not widely available and in Britain, at least, it is very expensive.[134]

Although nuts are a useful source of protein for vegetarians, with the exception of walnuts they have very high O-6-3 ratios and should therefore be eaten sparingly. Walnuts provide a fairly good balance of omega-6 and omega-3 fatty acids in a ratio of 5:1.

It is not only the widespread use of seed oils in spreads and cooked foods that has upset the balance in our diet. Dr. Mary Enig, author of '*Know Your Fats: The Complete Primer for Understanding the Nutrition of Fats, Oils and Cholesterol*', says that modern agricultural and industrial practices have reduced the amount of omega-3 fatty acids in commercially available vegetables, eggs, fish and meat. For example, organic eggs from hens allowed to feed on insects and green plants can contain omega-6 and omega-3 fatty acids in approximately equal quantities, but ordinary supermarket eggs can contain up to nineteen times more omega-6 than omega-3! (When I read this I was very glad that we've been buying

[131] www.marksdailyapple.com/healthy-oils/#axzz2MZckHjSf. Accessed March 2013.

[132] Duckett S K et al. *Effects of winter stocker growth rate and finishing system on tissue proximate, fatty acid, vitamin and cholesterol content*. Journal of Animal Science, 2009; 87 (9): 2961–70.

[133] Jaminet P and Jaminet S C. *Perfect Health Diet.* Scribner, 2012.

[134] At the time of writing high oleic sunflower oil was being sold on the U.K. eBay website for £41.75 per litre.

organic free-range eggs for the last 3 years.) Nevertheless, apart from non free-range eggs I don't believe there is much of a problem with vegetables, fish and meat in the U.K. at least. Cattle here are mostly still fed on grass rather than maize and other cereals, and I find it hard to see why the O-6-3 ratio in vegetables and fish should have changed significantly, except perhaps in the case of farmed salmon and trout. So buy free-range eggs and keep eating fresh vegetables, fish (preferably wild) and good quality meat.

Reducing our consumption of omega-6 fatty acids

So now we come to the all-important question: how can we reduce our consumption of omega-6 fatty acids?

- Apart from high oleic sunflower oil (which you probably couldn't afford), avoid altogether corn/maize/sweetcorn, cottonseeds, sunflower seeds and peanuts/groundnuts, plus all oils and products made from them, including popcorn and peanut butter.
- Use flax/linseed oil or extra virgin olive oil in salads, or if you can't afford them use rapeseed/canola/vegetable oil.
- Cook with butter, dripping, lard or goose fat, preferably from pasture-fed animals or birds. Vegetarians should use butter, extra virgin cold pressed olive oil, or else organic virgin coconut oil, which has only about 4% omega-6[135]. Palm oil is also stable at high temperatures, but should be obtained from a sustainable source – e.g. West African red palm oil.[136]
- Strictly limit your consumption of food made from seeds – including bread, pastry, pasta, breakfast cereals and rice.

Clearly if you buy any prepared foods such as cakes, biscuits, burgers, chips or ready meals there is a good chance that soya, cottonseed or other high omega-6 oils have been used to make them. That's why home-cooked food is so much healthier, and why it's so important to teach your children to cook and make home cooking a part of their lifestyle.

The website 180degreehealth.com says there are ten high omega-6 foods we really should avoid. Actually eleven foods are listed, not ten, so maybe the author was the same person who invented a baker's dozen. Anyway, here is the blacklist in detail:

- commercial peanut butter
- common oil-roasted snack nuts
- commercially fried potato fries (chips and crisps)
- other fried foods from restaurants
- commercially made salad dressings, including most vinaigrettes, Caesar dressing and coleslaw
- commercial mayonnaise and mayonnaise-based sauces
- doughnuts
- high-fat desserts
- margarine and foods often made with margarine, like cookies and brownies (cakes)
- vegetable oils, especially corn/maize/sweetcorn oil
- vegetable shortening (i.e. lard substitutes such as 'Trex', 'Cookeen' and 'Crisco') and foods made with it, such as biscuits, pie crusts and other pastries

[135] Mark Sissons, author of *The Primal Blueprint*. www.marksdailyapple.com/coconut-oil-health-benefits/#refined. Accessed March 2013.

[136] Mark Sissons. www.marksdailyapple.com/healthy-oils/#axzz2MZckHjSf. Accessed March 2013.

So there we are. To defuse the obesity bomb ticking beneath every home in our nation we have to stop consuming so much omega-6. We have to stop stuffing our children with it. We have to make a radical change in our families' diets, and take a firm decision to banish the hoards of grey squirrels which enter our stomachs in seed oils from soya beans, sunflower seeds, maize and peanuts, and from all the products made with them – margarines and spreads, cooking oils, ready meals, fast foods, pastries and snacks of all kinds. We must revert instead to the traditional foods our grandparents and ancestors lived on, when there was no epidemic of obesity, coronary heart trouble and type 2 diabetes. We should use animal fats for cooking, drink full-cream milk, eat meat and fresh vegetables, and prepare our food at home without the preservatives and additives and other ingredients added by manufacturers to make money at the expense of a nation's health. We must ignore the alluring and deceptive advertisements of a multimillion dollar food manufacturing industry, stop poisoning our kids with its profits, and nourish ourselves and our families with the food our bodies were designed for. That is how we and our children can remain healthy into a long old age. There is no other way.

It might sound as though that brings us to the end of dietary-related health problems, but I'm afraid it doesn't. In Victorian days, two wooden giants called Corineus and Gogmagog stood in Guildhall in the city of London. One held a mace and the other an axe: clearly they would have been formidable foes had they been alive. If we think of the mace-bearing giant as killing people with the wrong kinds of fat, the axe-bearing one is killing us equally effectively with something else. That something else is *sugar*.

CHAPTER 10: CHOLESTEROL – NOT THE BIG BAD WOLF

Forget cholesterol, it's not a problem

Any doctor who believes that cholesterol *causes* heart disease is at least 10 years out of date. I have a paper on my desk called '*A hypothesis out of date: the diet-heart idea*'.[176] It was published in the Journal of Epidemiology in 2002 by a Swedish doctor called Uffe Ravnskov, who published a similar paper in the *British Medical Journal* that same year.[177] Ravnskov spent over 10 years studying the evidence that linked saturated fatty acids and cholesterol to heart disease. He was particularly interested in why so many scientists were claiming that there was a link, and he found that in nearly every case they either ignored evidence that there is no causal link or else were claiming a link when their own research didn't really justify it. In his paper he cites 71 research papers on the subject, and concludes from them that high levels of cholesterol are not the cause of heart disease.

Cholesterol is good for oldies

At least one of the papers cited by Ravnskov[178] describes some research which discovered that old people die twice as often from a heart attack if they have a *low* cholesterol level rather than a high level of it. Another study[179] of more than a thousand elderly patients with severe heart failure found that within 5 years about 30% of those with high cholesterol levels (above 223mg/l of blood) had died, but 62% of those with low cholesterol levels (below 129mg/l) had died. The investigators ensured that everyone involved was adequately fed to make sure that a low cholesterol level was not simply the result of undernourishment. The study made it clear that an old person with heart trouble and a low cholesterol level is twice as likely to die of heart failure in the next 5 years as one with a high cholesterol level. And Ravnskov found *eleven* different studies that came up with similar findings.[180] So at my age I am certainly not going to worry about keeping my cholesterol level down. I would rather have a high cholesterol level so that I can live as long as possible.

Cholesterol is good for ladies

For ladies there is even better news. A high cholesterol level means you are less likely to suffer a heart attack whatever your age may be. One of the most famous studies of cholesterol and heart disease is the Framingham Heart Study, which began in 1948 with 5,209 men and women between the ages of 30 and 62 living in the town of Framingham, Massachusetts.

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[176] Ravnskov U. *A hypothesis out-of-date: the diet-heart idea.* Journal of Clinical Epidemiology 55 (2002); 057-1063.

[177] Ravnskov U. *Diet-heart disease hypothesis is wishful thinking.* British Medical Journal 2002; 324:238.

[178] Krumholz H M et al. *Lack of association between cholesterol and coronary heart disease mortality and morbidity and all-cause mortality in persons older than 70 years. Journal of the American Medical Association, 1990;* 272, 1335-1340.

[179] Horwich T et al. *Low serum total cholesterol is associated with marked increase in mortality in advanced heart failure. Journal of Cardiac Failure, August* 2002; 8(4):216-24.

[180] Ravnskov U. *High cholesterol may protect against infections and atherosclerosis. Quarterly Journal of Medicine* 2003; 96, 927-934.

During the first 20 years of the study an association between high cholesterol levels and heart disease was indeed found in many of the subjects, but the researchers reported that in women over 50 '*cholesterol had no predictive value*'.[181]

Much more recently in Norway there was a huge study of over 52,000 Norwegians of both sexes aged 20 to 74. Over a period of 10 years their blood cholesterol levels were monitored, and if they died within that period the cause of their death was recorded. The results were published in the *Journal of Evaluation of Clinical Practice* in 2012.[182] The following amazing result was reported: '*Among women, cholesterol had an inverse association with all-cause mortality as well as CVD mortality.*' This means that as a woman, the *higher* your cholesterol level is, whatever your age, the *less likely* it is that you will die within the next 10 years. And those figures were corrected for smoking and high blood pressure, so they take into account the effects of cholesterol only. CVD stands for Coronary Vascular Disease and it includes such things as stroke and heart failure as well as coronary heart disease. They found that for women the chance that you will die of CVD if your cholesterol level is above 7.0mmol/l is only three-quarters of your chance of dying of CVD if the level is below 5.0mmol/l. So a *high* cholesterol level is good news for women of all ages!

It is true that, in the Norwegian study, the death rate from coronary heart disease alone was slightly higher for the highest concentrations of cholesterol (above 7.0mmol/l) than for the mid range. However the authors of the paper concluded, '*If our findings are generalizable, clinical and public health recommendations regarding the 'dangers' of cholesterol should be revised. This is especially true for women, for whom moderately elevated cholesterol (by current standards) may prove to be not only harmless but even beneficial*'. Clearly they were using some scientific caution in the wording of their conclusion, but their meaning is obvious. If you are a woman forget about your cholesterol – it's not a problem.

Part of the reason that the medical world still believes high cholesterol levels to be associated with heart trouble is that much of the earlier research was carried out on middle-aged men who are very prone to heart attacks, and in their case there does seem to be an association between the two. However, just as Ancel Keys did, they forget that an association between two things doesn't necessarily mean that one causes another. Most deaths occur in bed, but that doesn't mean that it is safer to sleep on the floor! When a water company van is parked at the roadside there is nearly always a damaged water main in the vicinity. But does that mean that water company vans drive around the countryside bursting our water mains? Of course it doesn't. The vans bring workmen along to repair the damage, not to cause it. And that's the reason that cholesterol turns up when there is a problem in our arteries.

One of cholesterol's jobs is to help reduce inflammation and repair damaged cell walls, problems that are caused by stress, smoking, or diet. So when our arteries become damaged the liver generates cholesterol to repair them. And that's almost certainly why people's cholesterol levels rise as they get older. They need more of it. In other words, cholesterol doesn't produce heart disease: it's heart disease that produces cholesterol!

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[181] Kannel W B & Gordon T (editors). Framingham Monograph, Section 24. *An Epidemiological Investigation of Cardiovascular Disease*. U.S. Department of Health, Education and Welfare, National Institutes of Health, 1968.

[182] Petursson H et al. *Is the use of cholesterol in mortality risk algorithms in clinical guidelines valid? Ten years prospective data from the Norwegian HUNT 2 study.* Journal of Evaluation in Clinical Practice, February 2012; 18(1):159-68.

Cholesterol is good for everyone

Cholesterol is a very important type of fat which is needed for a range of totally essential bodily functions. There is some cholesterol in food, but 80% to 85% of the cholesterol in our bodies is produced by our liver in whatever quantities it is needed. If we avoided foods with cholesterol in them then our liver would simply produce more of it to make up the shortfall, and vice versa.[183] One discovery in the Framingham Heart Study was that when subjects who had very high cholesterol levels (over 300mg/dl) were compared with those who had very low levels (under 170mg/dl) there was no difference in the overall amount of fat that they consumed in their diets. In other words, the amount of fat we eat has nothing at all to do with our cholesterol levels. Dr. Uffe Ravnskov once ate 59 eggs is 9 days to see if a cholesterol-rich diet would increase his blood cholesterol. It didn't: his cholesterol level actually fell by 11%.[184]

So our bodies make as much cholesterol as they need for various essential tasks. Here are some of those tasks that cholesterol carries out:

- Our bodies consist of trillions of cells. Cholesterol is an essential component of the cell membrane, the protective skin around a cell that allows nutrients, hormones and other substances into the cell and lets waste products come out. It is needed to build and rebuild these cells.
- Many essential bodily activities are controlled by messengers called hormones. Cholesterol is required to produce the hormones that regulate sexual functions, the body's response to stress, infection and inflammation, the digestion of protein and carbohydrates, and even some aspects of behaviour. Hormone deficiency makes our bodies susceptible to major disease and various kinds of malfunction.
- Cholesterol acts as an antioxidant, protecting us from heart disease and cancer.[185]
- Cholesterol is used in the production of bile, which the body needs in order to digest other fats.
- Cholesterol in conjunction with sunlight produces vitamin D (which is a hormone rather than a vitamin). Vitamin D is needed for the absorption of the calcium and phosphorus that our bones are made of. Insufficient vitamin D causes rickets and brittle bones.
- Cholesterol coats our nerve fibres and is one component of synapses, the connections between cells that enable them to pass messages from one to another. So it is an essential part of our nervous system and our brain.
- About a quarter of the brain's total weight consists of cholesterol. Mother's milk is especially rich in cholesterol and contains a special enzyme that helps the baby utilize this nutrient.[186] Babies and children need cholesterol-rich foods throughout their growing years for the proper development of their brains and nervous systems.[187]

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[183] Lecerf J M & de Lorgeril M. *Dietary cholesterol: from physiology to cardiovascular risk*. British Journal of Nutrition, 2011; 106 (1): 6–14.

[184] Ravnskov U. *The Cholesterol Myths*, p. 109. New Trends Publishing Inc, Washington DC, 2000.

[185] Cranton E M & Frackelton J P. *Free radical pathology in age-associated diseases: Treatment with EDTA chelation, nutrition and antioxidants*. Journal of Holistic Medicine, Spring/Summer 1984; 6(1):6-37.

[186] Jensen R et al. *Lipids of human milk and infant formulas: a review*. American Journal of Clinical Nutrition, 1 June 1978; 31 (6):990–1016.

[187] Alfin-Slater R B & Aftergood L. *Lipids.* Modern Nutrition in Health and Disease, 6th ed, p.131. R S Goodhart and M E Shils, eds, Lea and Febiger, Philadelphia, 1980.

The cholesterol delivery service

Cholesterol is cholesterol: there's no such thing as good or bad cholesterol. When people use these terms they are really talking about are the two different chemicals that carry cholesterol around the bloodstream. Cholesterol can't dissolve in blood because it is a fat, so it has to be carried around in special chemicals that can dissolve in blood. These chemicals are called lipoproteins, or fat-carrying proteins. The low density ones are thought to be bad for us, again because there always seem to be a lot of them around in people who have heart trouble, and the high density ones are thought to be good for the opposite reason.

When we eat carbohydrates in the form of starch or sugar, they are converted to glucose, which is either used immediately for energy purposes or is sent to the liver. The liver uses it for various things, including the manufacture of the all-important cholesterol, and any excess glucose it turns into fat that our body can store for use later. It does this by building fat molecules called triglycerides. It then has to transport these insoluble triglycerides and the cholesterol through the blood to the parts of our body that need them. In order to do this the liver does something else as well: it assembles some 'delivery lorries' called lipoproteins.

Low-density lipoproteins, or LDLs as they are usually called, are what I call the Low-loader Delivery Lorries. They transport cholesterol and triglycerides from the liver to the building sites and warehouses of our cells. High-density lipoproteins, or HDLs, are the High-sided Delivery Lorries, which are believed to carry back any used or spare cholesterol from the building sites to their factory in Liverpool for recycling or waste disposal.[188] It's LDL which is confusingly called 'bad cholesterol', and HDL which is confusingly called 'good cholesterol'. As I said, the reason people use those terms is that high levels of LDL in the bloodstream are associated with a high incidence of heart disease, whereas high levels of HDL are associated with a low incidence of it.

Incidentally, dietary fats, as distinct from carbohydrates, are also converted into triglycerides and cholesterol, but dietary fats are processed in the intestines and the resulting triglycerides and cholesterol are distributed, not by LDLs, but by great big container lorries called chylomicrons. 'Chylomicron' is Greek for 'small milky one'. Chylomicrons are very tiny milky-looking globules, which may be small compared with a raindrop, for example, but they are around twenty times bigger than LDL particles. So you can see that LDLs, which are associated with heart disease, have nothing to do with any fat that we eat. LDLs are created only as a result of eating carbohydrates!

Repairs and breakdowns

So if the body creates LDL, and if high levels of LDL are associated with heart disease, does that mean that our bodies are making something that is bad for us? No, it doesn't. One reason that people with heart trouble usually have large concentrations of LDLs in their blood is that when our cells are damaged they have to be repaired, and cholesterol is one of the main materials used to repair them. So if our arteries have suffered a lot of damage then the liver has to generate large amounts of cholesterol to repair them, which means making lots of LDLs to carry the cholesterol to the damaged cells.

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[188] Lewis G F & Rader D J. *New insights into the regulation of HDL metabolism and reverse cholesterol transport*. Circulation Research, June 2005; 96 (12): 1221–32.

However, it may be true that the LDL particles themselves contribute to arterial damage, at least when there are too many of them in our blood. The belief is that when an LDL lorry breaks down on an arterial roadway, a breakdown vehicle called a macrophage comes along and gobbles it up before driving off with it to the rubbish tip. Macrophages are the original Pac-Men, because 'macrophage' comes from two Greek words meaning 'big eater'! They are one kind of white blood cell. The trouble is that eating a whole lorry makes the macrophage so fat it gets stuck in the walls of a blood vessel, or it can do. And that can lead to the formation of plaque. When enough plaque has formed the blood vessel becomes so narrow that a blood clot can block it completely, resulting in a heart attack or a stroke. You won't find the process described in quite those terms in other literature, but in broad outline that is the story commonly presented.

An LDL hypothesis

I don't think anyone knows for certain why the LDL lorries break down, but perhaps this is the explanation.

The triglycerides that the liver makes are produced, as I said, from sugars and carbohydrates. These are transported along with cholesterol by the LDL lorries. But when we eat more sugar and carbohydrates than we need, the liver has to make so many triglycerides that some of the lorries leave the factory in Liverpool with only triglycerides in them, or at least only a little cholesterol. And since carbohydrates come mostly from plant sources, it may be that the triglycerides our liver makes contain mostly polyunsaturated fats, which are mainly the kind that come from plants. So the LDL lorries carry lots of polyunsaturated fat but relatively little cholesterol. However it is thought that cholesterol can protect polyunsaturated fat from oxidation. So if LDLs loaded with polyunsaturated fat and not much cholesterol hang around for a long time in the bloodstream because there are more of them than our bodies really need, then eventually the polyunsaturated fats oxidize. Once that happens white blood cells in the form of macrophages identify the LDL particles as carrying damaged goods and swallow them up.[189]

An HDL hypothesis

Heart disease is associated with low levels of HDL as well as with high levels of LDL. This may be because HDL molecules are used to make LDL molecules. The liver doesn't actually make LDL directly to carry the triglycerides: it makes VLDL, or very low-density lipoprotein. When the VLDLs have delivered their triglycerides then HDL molecules turn them into LDLs, which are then carrying only cholesterol.

If a lot of VLDLs are made because there are a lot of triglycerides then a lot of HDL molecules will be needed to convert them to LDLs. Hence the supply of HDLs gets used up and the level of them in our blood falls. So the 'bad' LDL uses up the 'good' HDL, and heart trouble is then associated with low levels of HDL.

Whether that is the true explanation or not, your guess is as good as mine!

It's carbohydrates, stupid!

[189] Steinberg D. *Low density lipoprotein oxidation and its pathobiological significance.* Journal of Biological Chemistry, 1997; 272:20963-20966.

So LDL carrying oxidized polyunsaturated fats fur up our arteries because we eat too many carbohydrates. The LDL problem is nothing to do with eating saturated fat in foods like bacon and butter.

In reality there is an even closer association between triglycerides and heart disease than there is between LDL and heart disease. It's true that dietary fats also produce triglycerides, but the chylomicrons distribute these to our muscles and fat storing cells within about 5 hours of a meal, after which there is no trace of them in the blood,[190] whereas the triglycerides our liver produces from carbohydrates keep trickling out for many more hours after we have eaten. And, since fasting blood tests are usually carried out 12 hours after a meal, the triglyceride levels measured relate mainly to the carbohydrates we have eaten, not to fat.[191] Once again, since high triglyceride levels are associated with a higher risk of heart disease, it seems to be carbohydrates that are implicated.

Experiments with high fat/low carbohydrate diets

So here's a little test. If a man went on a diet in which most of his calories came from fats and only a very few from carbohydrates, what would happen to his levels of LDL, HDL, triglycerides and cholesterol? According to most doctors his LDL, triglycerides and cholesterol would all increase, being associated in most doctors' minds with heart disease and dietary fat, and his HDL levels would decrease. However, according to what I have been saying, the level of LDL and triglycerides in the blood would decrease on a high fat diet, and eventually cholesterol levels would decrease too because there would not be so much cell damage to repair if the fat was mostly saturated or monounsaturated fat. So what would actually happen?

In 2002 some scientists at the University of Connecticut got hold of twenty men of normal weight and with normal blood cholesterol levels.[192] (I am tempted to say that they had to search the whole of the U.S.A. to find twenty such men, but that would be cheeky!) They put twelve of these men on a very low carbohydrate, high fat diet for 6 weeks,[193] and allowed the other eight who didn't fancy the idea of such a diet to continue with their normal one as a 'control group'. The diet of the twelve high fat diet volunteers included moderate amounts of vegetables and salads as well as a daily vitamin and mineral supplement. What they found in the men on the high fat diet was that after 6 weeks their triglycerides had fallen by 33% and their 'good' HDL had risen by more than 11%. Surprisingly, there was no significant change in their LDL, although the kind of LDL measured (there are various kinds) did show an improvement. What most doctors and certainly most people would find even more surprising was that there was on average no change in their cholesterol levels at the end of the test period. A diet in which 61% of their energy was obtained from fat did not increase their cholesterol levels at all!

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[190] Steinberg D. *Low density lipoprotein oxidation and its pathobiological significance.* Journal of Biological Chemistry, 1997; 272:20963-20966. (Also cited earlier.)

[191] Chen Y D et al. *Why do low-fat high-carbohydrate diets accentuate postprandial lipemia in patients with NIDDM?* Diabetes Care, 1995; 18:10-16.

[192] Sharman M J et al. *A ketogenic diet favorably affects serum biomarkers for cardiovascular disease in normal-weight men. Journal of Nutrition* 2002; 132:1879-1885.

[193] *The energy distribution in the normal diet was* 17% protein, 47% carbohydrate and 32% fat. The distribution in the low carbohydrate diet was 30% protein, 8% carbohydrate and 61% fat. There were no restrictions on the type of fat.

Not surprisingly the fasting insulin level of the men on the high fat, low carbohydrate diet fell by an average of 34%, which is a very healthy sign. They also lost on average just over 2kg in weight. Yes, that's right. *The men on a high fat diet lost weight.* In the control group the only significant change was that at the end of the investigation period they were all 6 weeks older!

The scientists who conducted this research rather cautiously concluded that in the short term a high fat, low carbohydrate diet is not harmful and may improve one's chances of avoiding 'atherogenic dyslipidemia', i.e. arterial damage caused by high fat levels in the blood. Why they didn't they have the courage to conclude that such a diet is healthy, I don't know!

All right, you might say, but 6 weeks isn't very long. Quite true, but listen to this. Following that study in Connecticut, doctors at Duke University, North Carolina, decided to conduct a study lasting 6 months to determine the longer-term effects of a low carbohydrate diet.[194] This was an amazingly brave piece of research, because they allowed 41 overweight or obese volunteers to eat as much fat as they liked, so long as they restricted their carbohydrate intake to 25 grams a day! Actually the project started with 51 volunteers, but ten had to drop out for work or other reasons, including two who couldn't keep off their doughnuts and bagels and pizzas. The remaining 41 volunteers, aged between 35 and 53, comprised both men and women, with the majority being white women. They all wanted to lose weight.

They attended regular meetings at which they were encouraged to take at least 20 minutes of aerobic exercise like walking, cycling or swimming three times a week. Blood samples were taken at these meetings. After they had lost 40% of whatever weight they were trying to lose they were allowed to increase their carbohydrate intake to 50 grams a day. That still isn't much – one thick slice of bread! The rest of their diet consisted of unlimited amounts of meat, fish and seafood (e.g. beef, pork, chicken, turkey, fish, shellfish), unlimited eggs, 4 ounces of cheese per day, 2 cups of salad vegetables per day, and 1 cup of low-carbohydrate vegetables per day. The subjects were told to eat as much meat and eggs as they liked until their hunger was relieved. How about that for a weight-loss diet?

So what happened? On average the dieters lost 9kg in weight. This time their LDL decreased on average by 7%, a small improvement, and their cholesterol level fell by 5%. Their average HDL increased by 19%, a substantial improvement, and finally their triglyceride level, a major warning sign for the development of heart disease, fell by an enormous 43%! The effect on their insulin levels was not reported.

Hence after 6 months' eating unlimited amounts of fat and protein, but strictly limited amounts of carbohydrate, these participants lost weight, lowered their LDL, cholesterol and triglyceride levels, and substantially improved their HDL. These were similar changes to those found in the shorter study, but were even greater in magnitude over the longer period! All these changes were in accordance with what I was predicting, and they were all the complete opposite of what generally accepted wisdom would predict. But these are facts. They prove that fat is good for you, and that the amounts of carbohydrates we are currently encouraged to eat are bad for us.

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[194] Westman E C et al. *Effect of 6-month adherence to a very low carbohydrate diet program. American Journal of Medicine,* 2002; 113: 30-36.

I'm not recommending a diet as extreme as either of those described above, partly because of the cost and partly because they do seem to be somewhat unnatural. However it's very hard to argue against the following conclusions:

- Eating more fat does not increase your cholesterol level if you keep your carbohydrate consumption down and include vegetables and salads in your diet.
- Eating more fat and less carbohydrate decreases the risk factors for a heart attack.
- It is possible to lose weight by restricting one's carbohydrate intake without restricting one's intake of fat.

From what I have said earlier, it seems most likely that it was decreasing their carbohydrate intake rather than eating more fat that decreased the risk factors for the project participants. So if you are already eating enough fat don't get the idea that simply adding more will improve your health, especially if you are already overweight!

The final proof

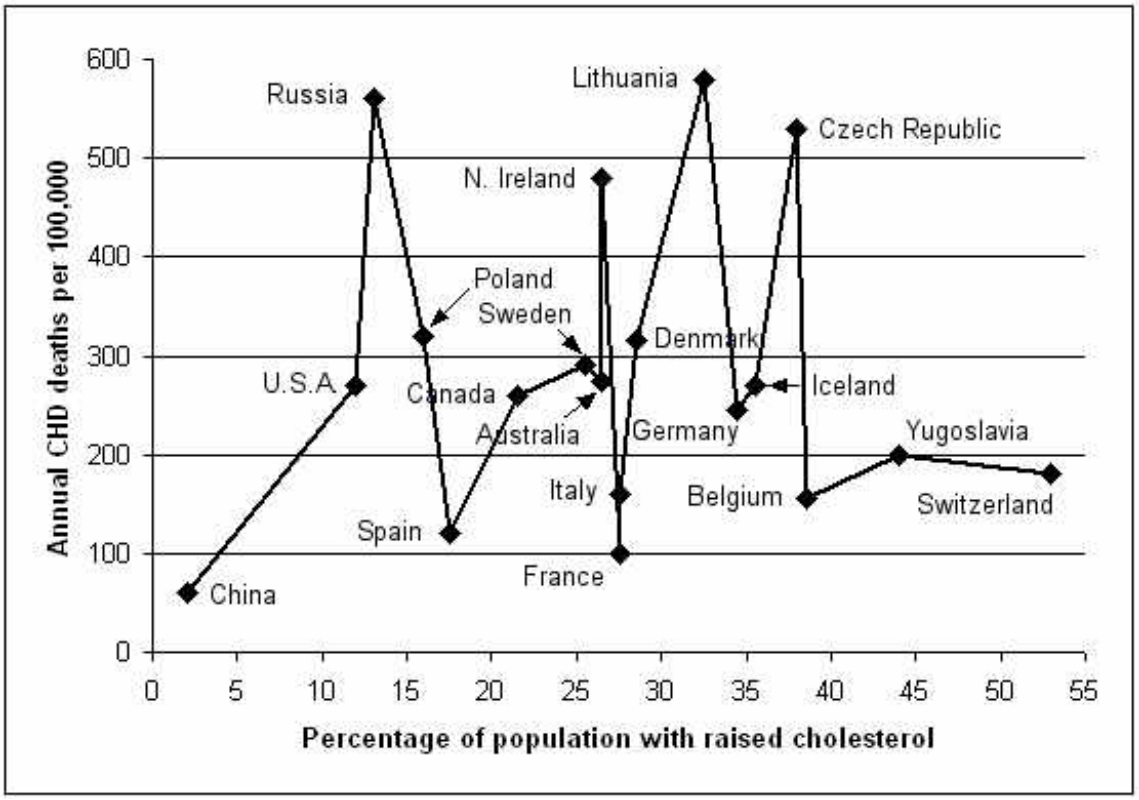
Now here's my final shot at anyone who claims that high cholesterol levels cause heart disease or are even associated with it. The world's largest ever study to monitor trends in heart disease was started by the World Health Organization in the early 1980s and ended in 2003.[195] Some 10 million people in 21 countries in Europe, Australia, North America, China and Russia participated in 'MONICA' (Multinational ***MONI***toring of trends and determinants in ***CA***rdiovascular disease). When the data were examined in 2003 no overall relationship whatever was found between national blood cholesterol levels and incidences of cardiovascular disease or death.[196] Figure 19 illustrates this very clearly!

Figure 19: Relationships between percentage of national population with high cholesterol levels and death rates from coronary heart disease[197]

[195] Tunstall-Pedoe H et al. *World's largest study of heart disease, stroke, risk factors, and population trends 1979-2002.* World Health Organization, MONICA Project.

[196] Tunstall-Pedoe H. *MONICA Monograph and Multimedia Sourcebook.* World Health Organization, Geneva, Switzerland, 2003.

[197] High cholesterol levels were defined in the Monica study as greater than 6.5mmol/l (260mg/dl.) Data for the figure was extracted from the previous reference by Dr. Malcolm Kendrick, author of *The Great Cholesterol Con*.


Annual CHD deaths per 100,000
600
500
400
300
200
100
0
0
5
10
15
20
25
30
35
40
45
50
55
Percentage of population with raised cholesterol
Russia
Lithuania
Czech Republic
N. Ireland
Poland
Sweden
Denmark
U.S.A.
Canada
Iceland
Australia
Germany
Yugoslavia
Italy
Belgium
Switzerland
Spain
France
China