THE CHOLESTEROL MYTH

Part 1: Introduction

The tragedy of science is the slaying of a beautiful hypothesis by an ugly fact. T H Huxley

Over the past couple of decades there has been a growing concern about fats and cholesterol. Dieticians, nutritionists and doctors have been telling us that fat is a killer. Governments have introduced national policies based around its reduction. Eat less cholesterol, saturated fat and salt, eat more fibre-rich foods we are all told. The evidence is incontrovertible that if we do not, we are doomed to the West's greatest killer - heart disease.

But is the evidence so clear? Despite the certainty implied by the propaganda, the debate continues in the medical journals, behind the scenes. Is diet a killer? Apart from those with a very rare disease, has cholesterol got anything to do with heart disease - or any other disease? And even if it has, will a change of diet be beneficial?

Like all debates, this one about cholesterol has two sides. *The Cholesterol Myth* explores the evidence on which present healthy eating' dietary recommendations are based.

Much of the evidence used in the cholesterol debate is complex. Nevertheless, with so much of only one side of the debate having been published and your having been subjected to so much that is misleading, I will try to explain the other side in as much detail as this paper allows.

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THE B. M. A. AND THE GOVERNMENT RECOMMEND THAT THE BRITISH PEOPLE SHOULD DRINK EIGHTY PERCENT MORE MILK, EAT FIFTY-FIVE PERCENT MORE EGGS, FORTY PERCENT MORE BUTTER AND THIRTY PERCENT MORE MEAT.

On the basis of research in the 1920s and 1930s by Sir John Boyd Orr and others, that was the advice given to the British people in 1938. The Government introduced free school milk - full cream, that is - and later we 'went to work on an egg'. As a consequence, child deaths from diphtheria, measles, scarlet fever and whooping cough fell dramatically - well before the introduction of antibiotics and widespread immunisation. Rickets, called 'the English Disease' because it was so wide-spread, and other deficiency diseases were relegated to the past. Other factors helped, but most important of all was the better nutrition that gave children a higher resistance. The recommendations above shaped our diet for nearly fifty years and helped to give us a mean life expectancy that is now among the highest in the world. Sixty years in 1930, our mean life expectancy had climbed to seventy years by 1960 and to seventy-five years by 1990. Now we are told they are shortening our lives - killing us with coronary heart disease. Why the sudden change? To discover that, we need to know something of the history of coronary heart disease and how the strategy to combat it evolved.

Coronary heart disease

There are many diseases that affect the heart but the one that the 'healthy eating' strategies seek to prevent is Coronary Heart Disease (CHD), more correctly called ischaemic heart disease (IHD). CHD is a condition where the coronary arteries that supply blood to nourish the heart muscle are narrowed by a build-up of material on their walls (an atheroma) to such an extent that they become blocked. This cuts off the blood supply to part of the heart muscle, and we have a heart attack. The narrowing also encourages the clotting of blood and, in consequence, it is possible for a clot to cause a heart attack long before the atheroma is large enough to do so. The material generally blamed for the build-up is cholesterol and the 'healthy eating' advice given to the public to reduce the incidence of CHD is aimed simply at reducing the levels of cholesterol in the blood.

Cholesterol

Because of the propaganda, you can be forgiven for thinking that cholesterol is a harmful alien substance that should be avoided at all costs. In fact, nothing could be further from the truth. Cholesterol is an essential component in the

body. It is found in all the cells of the body, particularly in the brain and nerve cells. Body cells are continually dying and new ones being made. Cholesterol is a major building block from which cell walls are made. Cholesterol is also used to make a number of other important substances: hormones (including the sex hormones), bile acids and, in conjunction with sunlight on the skin, vitamin D_3. The body uses large quantities of cholesterol every day and the substance is so important that, with the exception of brain cells, every body cell has the ability to make it.

Cholesterol may be ingested in animal products, but less than twenty percent of your body's cholesterol needs will be supplied in this way. Your body then makes up the difference. If you eat less cholesterol, your body merely compensates by making more. Although the media and food companies still warn against cholesterol in diet, it has been repeatedly demonstrated that the level of cholesterol in your blood is affected very little by the amount of cholesterol you eat.

Cholesterol and CHD

For reasons still unknown, coronary heart disease suddenly took off during the 1920s throughout the industrialised world. By the 1940s it was becoming the major cause of premature death. And nobody knew why.

In 1950 an American doctor, John Gofman, hypothesised that blood cholesterol was to blame. This was supported in 1951 when pathologists were sent to Korea to learn about war wounds by dissecting the bodies of dead soldiers. To their surprise they discovered unexpected evidence of coronary heart disease: unexpected for they knew that death from heart disease was extremely rare under middle age and these men averaged only twenty-two years of age. So the pathologists performed detailed dissections on the hearts of the next 300 corpses. In thirty-five percent they found deposits of fibrous, fatty material sticking to the artery walls. A further forty-one percent had fully formed lesions, and in three percent of the soldiers these lesions were sufficiently large that they blocked at least one coronary artery. Thus, over three-quarters of all the men examined showed evidence of serious coronary heart disease - and they were barely out of their teens.

Doctors now had a problem. As there are no symptoms with the partial blockage of the coronary arteries, how could they tell, without resorting to surgery, who was in danger? They had to find what was different in those with the disease and those free of it.

To cut a long story short, they found cholesterol in the material that builds up on artery walls and causes them to become blocked; people who died of heart disease often had high levels of cholesterol in their blood; and those who suffered the rare hereditary disease, *familial hypercholesterolaemia* (hereditary high blood cholesterol), also suffered a higher incidence of CHD. And so, not unnaturally perhaps, cholesterol and heart disease became linked.

But there are a number of significant points that the cholesterol theory overlooks. For example, there is a marked difference between the build-up found in those with familial hypercholesterolaemia and those with coronary heart disease: hypercholesterolaemia causes large deposits at the mouths of the coronary arteries, often leaving the arteries themselves unblocked, and so does not reproduce the type of obstruction found in coronary heart disease. People with myxoedema or nephrosis also have high blood cholesterol levels - yet in them, there is no increase in the incidence of CHD. Neither is raised blood cholesterol a predictor of CHD in people over sixty. It has also long been known that simple events, such as putting a cuff around the arm prior to taking a blood sample, or fear of the needle, can result in raised cholesterol values. And, even where these are avoided, large fluctuations are known with peak to nadir variations of as much as twenty-three percent. Lastly, cholesterol is only one of the constituents of an atheroma and, if you think about it, cholesterol is so necessary and so widespread in the body, it would have been surprising if it had not been found. Nevertheless the lowering of blood cholesterol became the sole objective in the fight against CHD; and the two principal methods used to achieve this are with diet and drugs.

References:

J W Gofman, *et al*. The role of lipids and lipoproteins in atherosclerosis . *Science*. 1950; 111: 166.

J P Strong, H C McGill jr. The natural history of coronary atherosclerosis. *Am J Pathol* . 1962; 40: 37.

W F Enos, R H Holmes, J Beyer. Coronary disease among United States soldiers killed in action in Korea. Preliminary report. *JAMA* 1953; 152: 1090.

Part 2: Dietary Fats and Heart Disease

For what a man would like to be true, that he more readily believes. Francis Bacon

That diet might play a part as a cause of CHD was hypothesised by another American doctor, Ancel Keys, in 1953. Using data from seven countries in his 'Seven Countries Study', Keys compared the death rates from CHD and the amounts of fats eaten in those countries to demonstrate that heart disease mortality was higher in the countries that consumed more fat than it was in those countries that consumed less. (At that time, data from many more countries were available. It seems that Keys ignored the data from those that did not support his hypothesis.) And so the 'diet/heart' hypothesis was born.

But how do we know it is true? It is all very well having a theory, what you have to do then is prove it. In medicine, the usual way is to select two groups of people, as identical for sex, age, and lifestyle as possible. One group called the *control group* , carries on as normal while the other, called the *intervention group* , tries the new diet, drug or whatever. After a suitable time, the two groups are compared and differences noted.

Keys' fat-diet/heart disease hypothesis was persuasive so, to test it, several large-scale, long-term, human intervention studies were set up in many parts of the world. These involved hundreds of thousands of subjects and hundreds of doctors and scientists and cost billions of dollars in an attempt to prove that a fatty diet caused heart disease.

Framingham Heart Study

The most influential and respected investigation of the causes of heart disease is the Framingham Heart Study. This study was set up in the town of Framingham, Massachusetts, by Harvard University Medical School in 1948 and is still going on today. It was this study that gave rise to the dietary 'risk factors' with which we all are so familiar today. The Framingham researchers thought that they knew exactly why some people had more cholesterol than others - they ate more in their diet. To prove the link, they measured cholesterol intake and compared it with blood cholesterol. As Table I shows, although subjects consumed cholesterol over a wide range, there was little or no difference in the levels of cholesterol in their blood and, thus, no relationship between the amount of cholesterol eaten and levels of blood cholesterol was found. (Although it is interesting that women who had the highest levels of cholesterol in their blood were ones who had eaten the least cholesterol.)

Table I: *Cholesterol intake - The Framingham Heart Study*

		Blood Cholesterol in Those	
	Cholesterol Intake	**Below Median Intake**	**Above Median Intake**
	mg/day	**mmol/l**	**mmol/l**
Men	704 ± 220.9	6.16	6.16
Women	492 ± 170.0	6.37	6.26

Next, the scientists studied intakes of saturated fats but again they could find no relation. There was still no relation when they studied total calorie intake. They then considered the possibility that something was masking the effects of diet, but no other factor made the slightest difference.

After twenty-two years of research, the researchers concluded:

"There is, in short, no suggestion of any relation between diet and the subsequent development of CHD in the study group."

On Christmas Eve, 1997, after a further twenty-seven years, the *Journal of the American Medical Association* (JAMA) carried a follow-up report that showed that dietary saturated fat reduced strokes. As these tend to affect older men than CHD, they wondered if a fatty diet was causing those in the trial to die of CHD before they had a stroke. But the researchers discount this, saying:

"This hypothesis, however, depends on the presence of a strong direct association of fat intake with coronary heart disease. Since we found no such association, competing mortality from coronary heart disease is very unlikely to explain our results."

In other words, after forty-nine years of research, they are still saying that they can find no relation between a fatty diet and heart disease.

Multiple Risk Factor Intervention Trial

One of the largest and most demanding medical studies ever performed on humans, The Multiple Risk Factor Intervention Trial (known in the medical world, by its initials, as MR. FIT) involved 28 medical centres and 250 researchers and cost $115,000,000. The researchers screened 361,662 men and deliberately chose subjects who were at very high risk to ensure that they achieved a statistically significant result. They cut cholesterol consumption by forty-two percent, saturated fat consumption by twenty-eight percent and total calories by twenty-one percent. Yet even then they didn't succeed. Blood cholesterol levels did fall, but by only a modest amount and, more importantly, coronary heart disease was unaffected. Its originators refer to the results as "disappointing" and say in their conclusions:

"The overall results do not show a beneficial effect on Coronary Heart Disease or total mortality from this multifactor intervention."

The Tecumseh Study

The Tecumseh Study attempted to correlate blood cholesterol levels measured one day with the amounts of fats eaten the previous day - but found none. Interestingly, Table II demonstrates that the people who ate the *least* cholesterol had the *highest* levels of blood cholesterol. Although not looking for it, this study also found that blood cholesterol levels were quite independent of whether the dietary fats were saturated or unsaturated. Thus another 'diet-heart' hypothesis, that only saturated fats are to blame, was invalidated.

Table II : *Fat intake and blood lipids - The Tecumseh Study*

	Blood Cholesterol in Thirds		
Daily Intake:	**Lower**	**Middle**	**Upper**
Fat - total (g)	128	134	133
Fat Saturated (g)	52	54	54
Polyunsat/Sat ratio	0.51	0.51	0.51
Cholesterol (mg)	554	566	533

WHO European Coronary Prevention Study

The results of the World Health Organisation's European Coronary Prevention Study were called "depressing" because once again no correlation between fats and heart disease was found. They had cut saturated fats down to only eight percent of calorie intake daily, yet in the UK section there were more deaths in the intervention group than in the control group.

The North Karelia Project

North Karelia, which had Finland's highest rates of heart disease, was compared with neighbouring Kuopio in The North Karelia Project. In North Karelia, risk factors were cut by seventeen percent over the period of the study. As Table III shows, in North Karelia there was a reduction in both CHD mortality and total mortality. Table III also shows, however, that in Kuopio, the control group, where there were no restrictions, there was an even bigger decline in both CHD and total mortality.

Table III: *Age Adjusted Rates/1,000. Age Group 30-64 years - The North Karelia Project.*

		1970/1	1976/7	Decline
Total Mortality				
Men	N Karelia	13.8	11.6	16%
	Kuopio	13.6	11.4	16%
Women	N Karelia	4.8	3.9	19%
	Kuopio	5.0	3.8	24%
CHD Mortality				
Men	N Karelia	7.7	6.3	18%
	Kuopio	7.7	5.8	25%
Women	N Karelia	2.5	1.7	32%
	Kuopio	2.5	1.6	36%

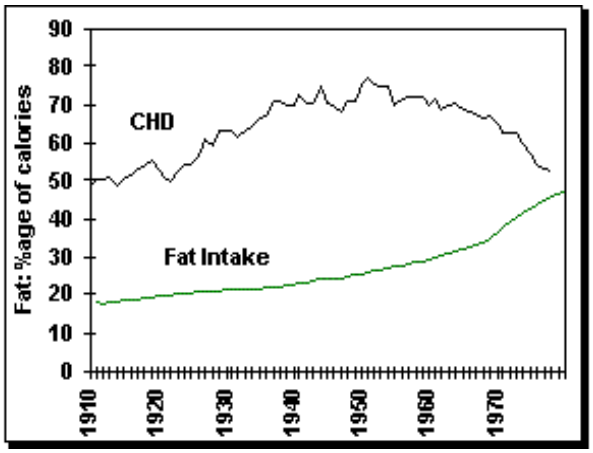
These figures suggest that adopting a 'healthy' lifestyle may actually have inhibited the decline in heart disease. They certainly give it no support.

This paper does not allow me to go through the more minor studies but they all show little convincing correlation between either the amount of fat eaten and heart disease or the type of fat eaten and heart disease. A review of twenty-six studies published in 1992 concluded that:

"Lowering serum cholesterol concentrations does not reduce mortality and is unlikely to prevent coronary heart disease. Claims of the opposite are based on preferential citation of supportive trials."

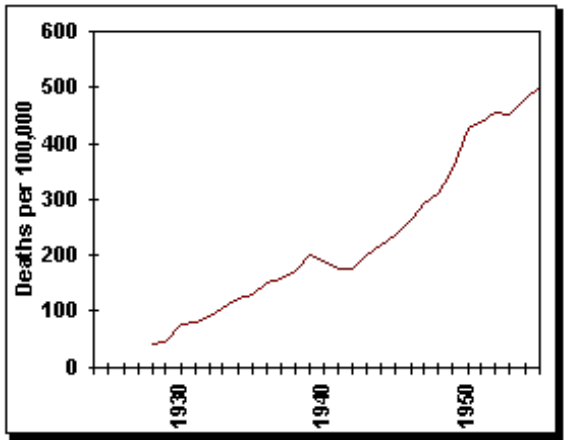
One that seemed to support the 'healthy' recommendations was a Finnish trial published in 1975. In the five years that the trial ran, cholesterol levels were lowered significantly, and the study was hailed as a success. But in December 1991 the results of a 10-year follow-up to that trial found that those people who continued to follow the carefully controlled, cholesterol-lowering diet were *twice* as likely to die of heart disease as those who didn't - some success! Professor Michael Oliver, writing in the *British Medical Journal* commenting on the results, writes

"As multiple intervention against risk factors for coronary heart disease in middle aged men at only moderate risk seem to have failed to reduce both morbidity and mortality such interventions become increasingly difficult to justify. This runs counter to the recommendations of many national and international advisory bodies which must now take the recent findings from Finland into consideration. Not to do so may be ethically unacceptable."



Despite this wealth of evidence, nutritionists and the media continue to mislead us. They tell us, for example, that the recent fall in the numbers of heart deaths in the USA is because Americans are eating less fat. The graph below, however, shows clearly that while CHD in the USA peaked in the 1950s and has fallen consistently since, this is against a background of *rising* fat intake.

I find difficulty understanding how the fat hypothesis gained such credibility in the USA as its history more than most does not support it. The North American continent had been opened up by explorers and trappers who lived, very healthily, as did the Amerindians, almost entirely on fresh meat and pemmican. As real pemmican is half dried lean meat and half rendered animal fat, and as fat has over twice the calorific value of protein, more than seventy percent of the energy in their diet came from fat.



Dieticians also say that the British had less CHD in the 1940s when fat was rationed. However, the decade of rationing went on into the early 1950s with fat being the last food to come off ration in 1954. Again the graph shows clearly that the most rapid *rise* in CHD occurred during that period.

Also, during the period of rationing, British farmers had a very low incidence of heart disease when one would have expected their intake of fats, particularly animal fats, to have been higher than most.

Experience in other countries

Keys based his fat-causes-heart disease hypothesis on a comparison between countries. When we are told that we are 'the sick man of Europe', we are also compared to other countries. So let me do a similar comparison.

1. In Japan, intakes of animal fat have more than doubled since the end of the Second World War. Over the same period their incidence of coronary heart disease has fallen consistently. In Israel too an increased consumption of saturated fats was followed by a fall in coronary deaths.

2. The dietary changes in Sweden parallel those in the USA, yet heart disease mortality in Sweden was rising while American rates were falling.
3. There is also a threefold variation in rates of heart disease between France and Finland even though fat intake in those two countries is very similar.
4. Among south Asians in Britain there is an unusually high incidence of heart disease, yet living on largely vegetarian diets, they have low levels of blood cholesterol and eat diets that are low in saturated fat.
5. Indians in South Africa have probably the highest rates of coronary disease in the world yet there is no apparent reason why they should based on the current dietary hypotheses.
6. Until recently, Indians in India had a very low incidence of heart disease while using ghee (clarified butter), coconut oil and mustard seed oil - all of which are highly saturated. The epidemic of heart disease in India began only after these were replaced with peanut, safflower, sunflower, sesame and soybean oils, all of which are high in polyunsaturated oils.
7. Lastly, the World Health Organisation is apparently in ignorance of epidemiological data that do not support its recommendation to reduce dietary saturated fat. While it talks of coronary heart disease being responsible for most deaths in Caribbean countries, fat intake there is remarkably low.

Polyunsaturated fats

The arguments for the polyunsaturated fat hypothesis are no more convincing than those for the cholesterol theory. The claim is that unsaturated fats have a protective or preventative effect on CHD. But in Israel, when consumption of polyunsaturated fats was about twice that of most Western countries, there was a very high incidence of CHD. Those given high polyunsaturated diets in a trial in New South Wales fared significantly worse than those on a free diet. And this is the finding in most trials that have increased the ratio of polyunsaturated fats.

From as early as 1971, an excess of cancer deaths has been reported in trials using diets that were high in polyunsaturated fats. Polyunsaturated fats are also blamed for a doubling in the incidence of gallstones in the general public.

One of the pioneers of the polyunsaturated-fat-prevents-CHD hypothesis was the American cardiologist E. H. Ahrens Jr.. After twenty-five years of further research, however, he concluded that it was "irresponsible" to continue to press the polyunsaturated fat recommendations on the general public. He went on:

"If the public's diet is going to be decided by popularity polls and with diminishing regard for the scientific evidence, I fear that future generations will be left in ignorance of the real merits, as well as the possible faults in any dietary regimen aimed at prevention of coronary heart disease."

Another of the original proponents of the low-fat, low-cholesterol hypothesis, and a member of the Norwegian Council for Diseases of the Heart and Arteries, Professor Jens Dedichen of Oslo, also changed his mind. In the 1950s Norway launched a cholesterol-lowering regimen in which soy margarine, that is high in polyunsaturated fatty acids, replaced butter, and soy oil was used extensively. During the subsequent 20 years the increase in the use of soy-based products was accompanied by a steep and continuing rise in deaths from coronary thrombosis. Professor Dedichen drew attention to the failure of the programme - and received a very hostile reaction from his colleagues.

Also castigated were members of the National Academy of Sciences and the National Research Council of America when in a report of May 1980, they stated that prevention of heart disease could not be achieved by reducing blood cholesterol using either diet or drugs, and said that such measures should be abandoned.

Margarine - a natural food?

The polyunsaturated fats used to make margarine are generally obtained from vegetable sources such as sunflower seed, cottonseed, and soybean. As such they might be thought of as natural foods. Usually, however, they are pressed on the public in the form of highly processed margarines, spreads and oils and, as such, they are anything but natural.

In 1989, the petroleum-based solvent, benzene, that is known to cause cancer, was found in Perrier mineral water at a mean concentration of fourteen parts per billion. This was enough to cause Perrier to be removed from supermarket shelves. The first process in the manufacture of margarine is the extraction of the oils from the seeds, and this is usually done using similar petroleum-based solvents. Although these are then boiled off, this stage of the

process still leaves about ten parts per million of the solvents in the product. That is 700 times as much as fourteen parts per billion.

The oils then go through more than ten other processes: degumming, bleaching, hydrogenation, neutralization, fractionation, deodorisation, emulsification, interesterification, . . . that include heat treatment at 140 ^{o}-160 ^{o} with a solution of caustic soda; the use of nickel, a metal that is known to cause cancer, as a catalyst, with up to fifty parts per million of the nickel left in the product; the addition of antioxidants such as butylated hydroxyanisol (E320). These antioxidants are again usually petroleum based and are widely believed to cause cancer.

The hydrogenation process, that solidifies the oils so that they are spreadable, produces *trans* -fatty acids that rarely occur in nature.

The heat treatment alone is enough to render these margarines nutritionally inadequate. When the massive chemical treatment and unnatural fats are added, the end product can hardly be called either natural or healthy.

Recent United States studies showed that heart disease worsened in those who switched from butter to polyunsaturate-rich margarine. Research published in March 1993, confirmed this. In a study that involved 85,000 nurses, women who ate just four teaspoons of polyunsaturated margarine a day had a sixty-six percent *increased* risk of CHD compared to those who ate none. A review of men's experience in the Framingham Study published in 1995 also found that 6 teaspoons a day (mean of lowest intake vs mean of highest), increased risk by nearly a third. The authors conclude:

"Intake of margarine may predispose to development of CHD in men".
- and CHD is the one disease eating this sort of margarine was supposed to reduce!

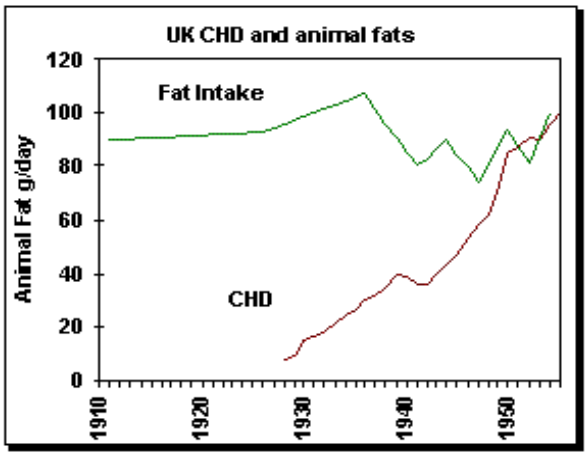
You may be interested in a list of the ingredients that may be present in butter and margarine:

Butter:	**Margarine:**
milk fat (cream), a little salt	Edible oils, edible fats, salt or potassium chloride, ascorbyl palmitate, butylated hydroxyanisole, phospholipids, tert-butylhydroquinone, mono- and di-glycerides of fat-forming fatty acids, disodium guanylate, diacetyltartaric and fatty acid esters of glycerol, Propyl, octyl or dodecyl gallate (or mixtures thereof), tocopherols, propylene glycol mono- and di-esters, sucrose esters of fatty acids, curcumin, annatto extracts, tartaric acid, 3,5,trimethylhexanal, ß-apo-carotenoic acid methyl or ethyl ester, skim milk powder, xanthophylls, canthaxanthin, vitamins A and D.

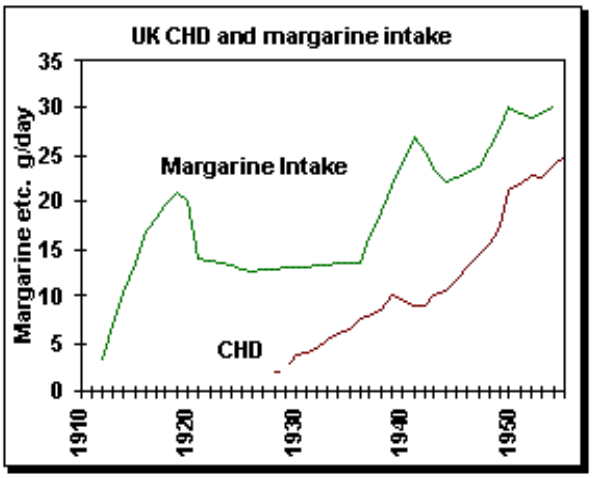
Dietary fat patterns

The total amount of fats in our diet today, according to the MAFF National Food Survey, is almost the same as it was at the beginning of this century. What has changed, to some extent, is the types of fats eaten. At the turn of the century we ate mainly animal fats that are largely saturated and monounsaturated. Now we are tending to eat more polyunsaturated fats - it's what we are advised to do.

It is interesting to compare the growth of heart disease in this country with intakes of different fats. The next graph illustrates the birth of CHD in Britain together with the intake of animal fat since the beginning of the century. When compared with the CHD curve, it is clear that there is no obvious relationship



If we plot CHD together with intakes of margarines and vegetable shortenings, however, we find a different curve.



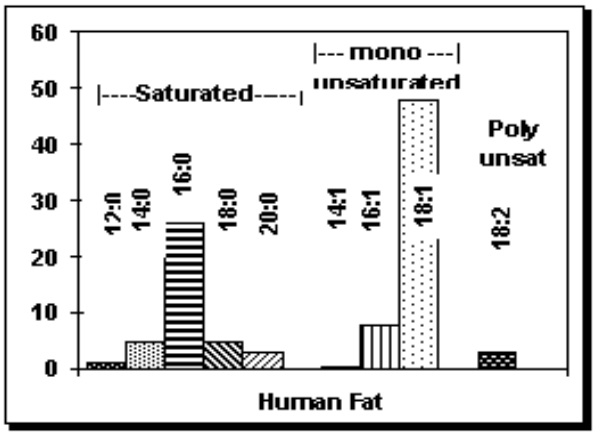
Margarine use began around the turn of the century. Butter was expensive. The poor bought margarine as a substitute for butter and sales were brisk. The rapid rise in margarine consumption was followed a couple of decades later by that dramatic rise in heart disease deaths.

If there is a causal relationship between fat intake and heart disease, these two graphs suggest to me that it is the margarines that are the more likely candidates for suspicion.

Polyunsaturated fats and Cancer

Body cell walls are made of cholesterol. The graph below demonstrates that the human body's fat make-up is largely of saturated and monounsaturated fatty acids. We contain very little polyunsaturated fat. Cell walls have to allow the

various nutrients that body cells need from the blood, but stop harmful pathogens. They must be stable. An intake of large quantities of polyunsaturated fatty acids changes the constituency of cholesterol and body fat. Cell walls become softer and more unstable.



Many laboratories have shown that diets high in polyunsaturates promote tumours. It has been known since the early 1970s that it is linoleic acid that is the major culprit. As Professor Raymond Kearney of Sydney University put it in 1987:

"Vegetable oils (eg Corn oil and sunflower oil) which are rich in linoleic acid are potent promoters of tumour growth."

Carcinogens - background radiation, ultraviolet radiation from the sun, particles in the air we breathe and the food we eat - continually attack us all. Normally, the immune system deals with any small focus of cancer cells so formed and that is the end of it. But linoleic acid suppresses the immune system. Indeed it is so good at this that in the 1970s sunflower oil was given to kidney transplant patients to prevent kidneys being rejected - until an excess of cancer deaths was reported. With a high intake of margarine, therefore, a tumour may grow too rapidly for the weakened immune system to cope thus increasing our risk of a cancer.

Since 1974, the increase of polyunsaturated fats has been blamed for the alarming increase in malignant melanoma (skin cancer) in Australia. We are all told that the sun causes it. Are Australians going out in the sun any more now than they were fifty years ago? They are certainly eating more polyunsaturated oils: even milk has its cream removed and replaced with vegetable oil. Victims of the disease have been found to have polyunsaturated oils in their skin cells. Polyunsaturated oils are oxidised readily by ultra-violet radiation from the sun and form harmful 'free radicals'. These are known to damage the cell's DNA and this can lead to the deregulation we call cancer. Saturated fats are stable. They do not oxidise and form free radicals.

Malignant melanoma is also said to be increasing in this country. Does the sun cause this? In Britain the number of sufferers is so small as to be relatively insignificant. Even so, it is not likely that the sun is to blame since all the significant increase is in the over-seventy-five-year-olds. People in this age group tend to get very little sun.

Melanoma occurs ten times as often in Orkney and Shetland than it does on Mediterranean islands. It also occurs more frequently on areas that are *not* exposed to the sun. In Scotland, for example, there are five times as many melanomas on the feet as on the hands; and in Japan, forty per cent of pedal melanomas are on the soles of the feet.

In 1991, two studies, from USA and Canada, found that linoleic acid, the major polyunsaturated fatty acid found in vegetable oils, increased the risk of breast tumours. This, it seems, was responsible for the rise in the cancers noted in previous studies. Experiments with a variety of fats showed that saturated fats did not cause tumours but, when small amounts of polyunsaturated vegetable oil or linoleic acid itself was added, this greatly increased the promotion of breast cancer.

A study of 61,471 women aged forty to seventy-six, conducted in Sweden, looked into the relation of different fats and breast cancer. The results were published in January 1998. This study found an inverse association with monounsaturated fat and a positive association with polyunsaturated fat. In other words, monounsaturated fats protected against breast cancer and polyunsaturated fats increased the risk. Saturated fats were neutral.

All polyunsaturated margarines, from the brand leader to shops' 'own brands' are around thirty-nine percent linoleic acid. Of cooking oils, sunflower oil is fifty percent and safflower oil seventy-two percent linoleic acid. Butter, on the other hand, has only a mere two percent and lard is just nine percent linoleic acid. Linoleic acid is one of the essential fatty acids. We must eat some to live, but we do not need much. The amount found in animal fats is quite sufficient.

Because of the heart disease risk, in 1994 the manufacturers of Flora changed its formula to cut out the trans fats and other manufacturers have since followed. But that still leaves the linoleic acid.

The anti-cancer fat

Linoleic acid is one of the essential fatty acids that our bodies need but cannot synthesise. We must eat some to survive. Fortunately there is one form of linoleic acid that is beneficial. Conjugated linoleic acid (CLA) differs from the normal form of linoleic acid only in the position of two of the bonds that join its atoms. But this small difference has been shown to give it powerful anti-cancer properties. Scientists at the Department of Surgical Oncology, Roswell Park Cancer Institute, New York and the Department of Biochemistry and Molecular Biology, New Jersey Medical School, showed that even at concentrations of less than one percent, CLA in the diet is protective against several cancers including breast cancer, colorectal cancer and malignant melanoma.

Conjugated linoleic acid has one other difference from the usual form - it is not found in vegetables but in the fat of ruminant animals. The best sources are dairy products and the fat on red meat, principally beef. It is another good reason not to give up eating red meat or to cut the fat off.

Scientists at the University of Wisconsin also believe that CLA has a slimming action. They put the dramatic increase in obesity in the USA down to Americans not eating beef fat.

Monounsaturated fats

Several populations in the world, Eskimos and those in the Mediterranean countries for example, eat high-fat diets yet have very low incidences of heart disease. This realisation has led to research scientists switching their attentions to monounsaturated fats found in fish oils and olive oil.

Although the supposed virtues of monounsaturated fats are being talked of in the press as possible saviours of Western man, the monounsaturated theory is not new. It was first demonstrated over thirty years ago that giving people more unsaturated fats could lower blood cholesterol. However, surveys of countries with different tastes in fats and oils have failed to show that this protects against heart disease. For example, Norwegians, who eat a lot of saturated fats, have lower rates of the disease than New Zealanders who eat a similar amount. But if, as has been suggested, the Norwegians are protected by the monounsaturated oils in the fish that they eat, then why is it that in Aberdeen, where a lot of fish is also consumed, the heart disease rate is double that of Oslo? Proponents also forget that many other people, such as the Maasai tribes of Africa, who don't eat either fish or olive oil, also have a low incidence of heart disease.

There is also no evidence that either mono- or polyunsaturated oils are of benefit to those who have already suffered a heart attack. As long ago as 1965 survival rates were studied in patients eating different oils. Splitting patients into three groups, who were given polyunsaturated corn oil, monounsaturated olive oil and saturated animal fats respectively, it was found that only the corn oil lowered blood cholesterol levels. At first sight, therefore, it seemed that men in the polyunsaturated group had the best chance of survival. However, at the end of the two-year trial only fifty-two percent of the polyunsaturated corn oil group were still alive and free of a fresh heart attack. Those on the monounsaturated olive oil fared little better: fifty-seven percent survived and had no further attack. Those eating the saturated animal fats, however, fared much better with seventy-five percent surviving and without a further attack.

Breast Cancer. The Swedish study by Alicia Wolk and colleagues mentioned above did find, however, that monounsaturated fats were protective against breast cancer.

Animal fats such as lard are around 43% Saturated, 47% Mono-unsaturated and 10% Polyunsaturated - which the evidence suggests is just about ideal.

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Part 3: The Bran Wagon

The tragedy of science is the slaying of a beautiful hypothesis by an ugly fact. T H Huxley

The belief that regular bowel movement is important for health is very ancient. But the present theory is based on Dr. Dennis Burkitt's discovery that relatively few rural black Africans suffer from cancer of the colon. He attributed this to their relatively crude diet.

The theory was that, as fibre made food travel through the gut faster, it allowed less time for cancer-inducing agents to form. This, of course, presupposed that food became carcinogenic in the gut and there was no evidence that it did. Neither was there any evidence that moving food through the intestine at a faster rate decreased the risk of colon cancer. Moreover, the rural Africans' lifestyle was far from that of the Western city dweller: their diet is different, but also they were not exposed to so many pollutants, toxins or mental stresses. Indeed, there were many factors that could have been responsible for a difference in disease patterns. Other communities - the Mormons of Utah, for example - also enjoyed a low incidence of colon cancer yet they ate a low-fibre diet.

So the theory was unsubstantiated at the time and it was to be disproved in practice later as the rural Africans moved into towns and adopted a Western style low fibre diet. Their incidence of colon cancer has remained low and this has continued with the second generation. Nevertheless, these later findings were not publicised. Burkitt's theories caught the attention of the media. Always ready to exploit a good story, they expanded what was at best a very weak hypothesis into a treatment dogma that teaches that fibre is a panacea for all manner of illnesses.

Commercial interests were quick to see the potential in the recommendation and jump on the bran wagon. Burkitt's recommendation was based on vegetable fibre, but bran (cereal fibre) has a far higher fibre content and bran was a practically worthless by-product of the milling process that, until then, had been thrown away. Almost overnight, it became a highly priced profit maker. Although totally inedible, backed by Burkitt's fibre hypothesis, bran could now be promoted as a valuable food. But Dr. Hugh Trowell, Burkitt's partner and another strong advocate of dietary fibre, stated in 1974 that:

"A serious confusion of thought is produced by referring to the dietary fibre hypothesis as the bran hypothesis, for many Africans do not consume cereal or bran"

Fibre and coronary heart disease

The idea that fibre could protect against heart attacks was hypothesised by Trowell in 1972, again based on research on rural Africans. The dietary intervention trials mentioned earlier, however, concluded that increasing dietary fibre had no beneficial effect on heart disease.

Fibre and other diseases.

It may be useful at this stage to consider the claims for fibre in curing or preventing other diseases. For example, bran has been a popular way to manage irritable bowel syndrome (IBS) for about thirty years, despite the fact that no placebo-controlled study of bran in IBS has yet shown any convincing beneficial effect. A study, published in 1994, found that while fruit fibre was effective, bran only made the situation worse. Far from being a cure for IBS, they found that it was the bran that was causing it! Bran also caused bowel disturbances, abdominal distension and pain.

Moreover, there is no direct evidence that an increase of fibre by itself will prevent or cure the other diseases. With respect to colon cancer, Burkitt's theory was questioned with the suggestion that the low cancer rates in rural Africans may be due to their high early death rates from other causes, so that they do not reach the age at which cancer peaks in Europeans. As the Africans' life expectancy was only forty years at the time Burkitt did his research and Western cancers don't peak until the age of sixty-five, one wonders why this wasn't noticed before.

There is also a growing scepticism in the USA that lack of fibre causes cancer; some studies even suggesting that a fibre-enhanced diet *increases* the risk of colon cancer.

Other adverse effects

Tests into the supposed benefits of dietary fibre soon showed that there could be other harmful side effects. All the nutrients in food are absorbed through the gut wall and this takes time. Fibre, by speeding food through the gut faster so that less nutrients are absorbed, inhibits the absorption of iron, calcium, phosphorus, magnesium, energy, proteins, fats and vitamins A, D, E and K. This happens with all types of fibre although with a normal Western-style, nutrient-rich diet, the loss caused by vegetable fibre intake is unimportant. More importantly, phytate found in cereal fibre (bran) also binds with calcium, iron and zinc making them indigestible, which in turn causes malabsorption. One study, for example, showed that subjects absorbed more iron from white bread than from wholemeal bread even though their intakes of iron were fifty percent higher with the wholemeal bread. Bran has also been shown to cause faecal losses of calcium, iron, zinc, phosphorus, nitrogen, fats, fatty acids and sterols, thus depleting the body of these materials.

These findings are a cause for concern in several sections of the population who are at considerable risk from eating too much fibre - and bran fibre in particular:

1. The incidence of **osteoporosis** (brittle bone disease) is increasing and now affects one in two post-menopausal women, one in five of whom will die as a direct result. Osteoporosis is also increasingly affecting men. Osteoporosis is caused by several factors, but lack of calcium is the basic problem. Bran both inhibits the absorption of calcium from food and depletes the body of the calcium it has. Moreover, zinc, which bones need to heal, is another mineral whose absorption is adversely affected by bran.
2. Sufferers from **Alzheimer's Disease** (senile dementia) are found to have abnormal amounts of aluminium in their brains. Tests on the people of Guam and parts of New Guinea and Japan, who get Alzheimer's disease at a much younger age, suggest that it is lack of calcium, causing a hormonal imbalance that permits the aluminium to penetrate the brain.
3. Infants may suffer similar **brain damage** if fed soy-based baby milk as this too has a high phytate content, inhibiting the absorption of zinc, which is essential for proper brain development.
4. Vitamin deficiency diseases such as **rickets** that were common in Britain until a diet high in dairy products and meat was advocated are on the increase again. The situation is getting so bad here that doctors suggest that vegetarian-based fad diets should be classified a form of child abuse.
5. In the UK, USA, Canada and South Africa the intake of 'anti-nutrients' such as dietary fibre that impair the absorption of iron, accompanied by a low intake of meat (another result of the diet-heart recommendations), is producing a real risk of **iron deficiency anaemia.**
6. **Depression, anorexia, low birth weight, slow growth, mental retardation** , and **amenorrhoea** are associated with deficiencies of zinc and the first five of these are also associated with a deficiency of iron.
7. Lastly, excess fibre affects the **onset of menstruation, retards uterine growth** and, later, is associated with **menstrual dysfunction** .

Because of the phytate, Professor David Southgate, arguably the world's leading authority on the effects of fibre, concludes that infants, children, young adolescents and pregnant women whose mineral needs are greater should be protected from excessive consumption of fibre.

Writing of the **colon cancer** risk, Drs. H. S. Wasan and R. A. Goodlad of the Imperial Cancer Research Fund stated in 1996:

"Until individual constituents of fibre have been shown to have, at the very least, a non-detrimental effect in prospective human trials, we urge that restraint should be shown in adding fibre supplements to foods, and that unsubstantiated health claims be restricted." . . . "Specific dietary fibre supplements, embraced as nutriceuticals or functional foods, are an unknown and potentially damaging way to influence modern dietary habits of the general population."

Until fibre can be shown not to be detrimental they suggest that

"restraint should be shown in adding fibre supplements to foods, and that unsubstantiated health claims should be restricted".

January 1999 saw the publication of the largest trial into the effects on fibre on colon cancer ever conducted. After studying 88,757 women for sixteen years, doctors at the Brigham and Women's Hospital and Harvard Medical School say that

"No significant association between fiber intake and the risk of colorectal adenoma was found." . . . "Our data do not support the existence of an important protective effect of dietary fiber against colorectal cancer or adenoma."

Summary

Bran is bad news. While there is not too much harm from fruit fibre, the usual bran that is pushed at us - wheat bran- should be avoided like the plague it is.

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Part 4: The Dangers of Low Blood Cholesterol

Nature has taken good care that theory should have little effect on practice. Samuel Johnson

Low blood cholesterol and cancer

So far advertisers and news media have concentrated on the supposed danger from high levels of blood cholesterol. The dangers of low blood cholesterol levels have largely been ignored.

Countries with diets high in saturated fats also tend to have high levels of colon cancer. In 1974 a review of the Framingham data and those from Keys' 'Seven Countries' study was carried out. It was expected to show that the cancer could also be blamed on high blood cholesterol. However, the baffled researchers found the opposite: those with the cancer had cholesterol levels that were *lower* than average.

Reports of more than twenty studies into the relation between blood cholesterol and cancer have been published since 1972. Most have reported an association between low blood cholesterol and cancer. The authors of the Renfrew and Paisley Study conclude:

"it may be a mistake to assume that dietary advice given to the general population to reduce the intake of saturated fat will necessarily reduce overall mortality."

In a study from the USA published in 1990, changes in blood cholesterol over time were studied in patients with colon cancer. The doctors found that there had been an average thirteen percent decline in blood cholesterol levels in

the ten years prior to diagnosis of the cancer compared with an average increase of two percent in the control group. Both those with the cancer and those free from it had similar blood cholesterol levels initially. It is possible that the decline in blood cholesterol levels was a result of the cancer, not the cause of it, but this is ruled out by the investigators. They compare cholesterol studies with apparently contrary findings and show that in reality they are consistent. Comparing those that reported normal or high cholesterol readings several years prior to diagnosis with others where, at the time of diagnosis, levels were low, they conclude that it was a long term lowering of blood cholesterol levels that gave rise to the cancers. Interestingly, the average blood cholesterol level of those who developed the cancers declined to an average 5.56 mmol/l and yet the British government's *Health of the Nation* strategy still aims to reduce everyone's levels to below 5.2 mmol/l.

Low cholesterol means more strokes

Published at about the same time was a very large study in Japan, covering two decades, which concluded that low levels of blood cholesterol also increase the incidence of stroke.

Over the past few decades, Japan has experienced a rapid change in its living and eating patterns. The Japanese are eating more total fat, saturated fatty acids and cholesterol, animal fats and protein, and less rice and vegetables. This has provided a unique opportunity for a large-scale, natural experiment into the effects of those changes.

Investigators have shown that this change to Western and urban eating patterns, departing as it does from centuries old traditions, has been accompanied by a general lowering of blood pressure and a large decline in the incidence of stroke deaths and cerebral haemorrhage between the 1960s and the 1980s. They attribute this decline to an increase in blood cholesterol levels over the period. Supporting their findings were the results of a follow-up of 350,000 men screened for the MRFIT in the United States that showed that the risk of death from cerebral haemorrhage in middle-aged men was six times greater if they had low blood cholesterol levels.

On Christmas Eve, 1997, yet one more study's results were headlined in the press. The Framingham researchers said that *"Serum cholesterol level is not related to incidence of stroke . . ."* and showed that for every three percent more energy from fat eaten, strokes would be cut by fifteen percent. They conclude:

"Intakes of fat and type of fat were not related to the incidence of the combined outcome of all cardiovascular diseases or to total or cardiovascular mortality."

So, after forty-nine years of research, they are still saying that there is no relation between a fatty diet and heart disease. The evidence now is clear and unequivocal: animal fats are not harmful.

Two more studies, which considered total blood cholesterol levels and mortality in the elderly, were published in the *Lancet* almost simultaneously in 1997. In the first, scientists working at the Leiden University Medical Centre found that

"each 1 mmol/l increase in total cholesterol corresponded to a 15% decrease in mortality".

Similarly, doctors at Reykjavik Hospital and Heart Preventive Clinic in Iceland noted that the major epidemiological studies had not included the elderly. They too studied total mortality and blood cholesterol in the over 80s to show that men with blood cholesterol levels over 6.5 had less than half the mortality of those whose cholesterol level was around the 5.2 we are told is "healthy".

Low cholesterol and Alzheimer's Disease

Approximately half of the brain is made up of fats. Dr. F. M.Corrigan and colleagues, writing in 1991 about the relief of Alzheimer's Disease, ask that *"strategies for **increasing** the delivery of cholesterol to the brain should be identified"*. In the fight against Alzheimer's disease, they recommend increasing fat intake.

And at the other end of life

In 1991 the US National Cholesterol Education Programme recommended that children over two years old should adopt a low-fat, low-cholesterol diet to prevent CHD in later life. A table showing a good correlation between fat and cholesterol intakes and blood cholesterol in seven to nine-year-old boys from six countries supported this advice. What it did not show, however, was the strong correlation between blood cholesterol and childhood deaths

in those countries. These are at Table V. As is clearly demonstrated, the death rate rises dramatically as blood cholesterol levels fall. So for children too, low blood cholesterol is unhealthy.

Table V: *Blood cholesterol and mortality in under-5s in six countries*

	Blood cholesterol	Childhood deaths
Finland	4.9	7
Netherlands	4.5	9
USA	4.3	12
Italy	4.1	12
Philippines	3.8	72
Ghana	3.3	145

Low blood cholesterol, aggressive behaviour and suicide

Lastly, since 1992, several observers have noted increases in suicides among those undertaking cholesterol-lowering dietary regimes. Decreases in blood cholesterol cause decreases in serotonin receptors leading to increased microviscosity and affecting the balance of cerebral lipid metabolism which could have profound effects on brain function.

In institutions, aggressive people and those with antisocial personality have been found to have lower blood cholesterol levels than normal: Typically 5.04mmol/l vs 6.02mmol/l. Mental patients with high blood cholesterol (7.55mmol/l) were less regressed and withdrawn than those with lower (4.80mmol/l).

Dr Matthew G Dunnigan of Stobhill General Hospital, Glasgow, concludes that:

"Without definite data on all-cause mortality and with current unresolved concerns about excess deaths from non-cardiac causes in RCTs, decisions to embark on lifelong lipid lowering drug treatment in most patients with primary hypercholesterolaemia depend on the doctor's interpretation of available evidence. As in other situations in which certainty is illusory, this varies from evangelical enthusiasm for lowering lipid concentrations to therapeutic nihilism."

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Part 5: Cholesterol-Lowering Drugs

For every problem there is a solution, neat, plausible and wrong. H L Mencken

Although it became clear that a change in diet had little effect on CHD, that did not end the scientists' efforts to demonstrate that CHD could be prevented. If diet couldn't do it, then intervention with drugs would provide the evidence. And since drugs could be controlled much more strictly, and used in conjunction with placebos, the findings would be more demonstrable. But the drugs used to reduce blood cholesterol have all proved to be something of a disaster.

Launched on the public in 1961, **Triparanol** causes the levels of blood cholesterol to fall by inhibiting the liver's ability to make cholesterol. Two years later it was with- drawn because of serious side effects. Luckily for

triparanol's manufacturers, a public scandal was avoided as the media's attentions were focussed on another drug marketed at the same time and by the same company - thalidomide.

More recently, a number of other drugs have been the subject of extensive and expensive trials. First was **Cholestyramine** (Questran) which reduces cholesterol by interfering with digestion. The gall bladder manufactures bile acid from cholesterol, and the bile acid is used in the intestine to digest fats. But when the drug is present in the gut, it binds with the bile acid, removing it from its normal function. Because the drug is indigestible, it, together with the bile acid, is excreted and the gall bladder has to make more by drawing cholesterol from the bloodstream.

As the trial would be very expensive, the scientists examined 480,000 men over a period of three years to find suitable subjects. They had to be men in the coronary age group and with extremely high blood cholesterol levels. As such men are in the most vulnerable group, their chances of success were greatly increased.

The investigators confidently announced in advance that blood cholesterol levels would be lowered by an average of 28% and, after seven years, coronary heart disease would be reduced by 50% in the treatment group.

At the end of the trial, however, cholesterol levels had fallen by less than a quarter of that called for at the start and heart disease rates were hardly affected. The $142 million trial was a total flop. Even if it had proved a success, however, those participating were so unrepresentative of the population that the question of its efficacy for the typical adult would still have remained. Another flaw that became apparent was an increase in the incidence of oral-gastro-intestinal cancers which could not be dismissed as a random chance. In the Lipid Research Clinics trial there were 21 cases and 8 deaths from gastrointestinal cancer in those taking the drug, compared to 11 cases and just 1 death in the control group.

Other organisations tested other drugs. The World Health Organisation sponsored its own trial with **Clofibrate** (Atromid). This too was targeted against cholesterol and was confidently expected to lower blood cholesterol levels by 30%.

As with cholestyramine, the levels were lowered by much less than the expected amount and at the end of the trial it became clear that there had been many more deaths in the group taking clofibrate than in the control group - notably from gallstones, and cancer of the liver and digestive system. In the WHO clofibrate trial, as Table IV demonstrates, the drug killed more than it saved.

Table IV: *WHO European Primary Prevention Trial with Clofibrate. 9.6-year follow up*

	Clofibrate	**Placebo**
Cause of death	**5,331 men**	**5,296 men**
CHD	157	138
Stroke	30	19
Other cardiovascular diseases	21	16
Cancers	125	99
Other medical	30	13
Accidents	31	30
Unknown	2	2
All causes (Total)	**396**	**317**

Among other drugs to be tested were:

a. The female hormone **Oestrogen** on the theory that if premenopausal women did not get heart disease, perhaps oestrogen would protect men. But the hormone caused heart attacks rather than preventing them.

b. The hormone **Dextrothyroxine** , which lowers cholesterol levels, abandoned quickly when an increase in mortality was noticed in the treatment group.

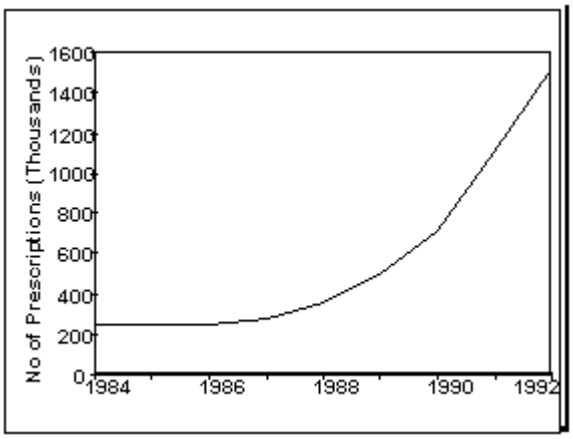
c. The vitamin **Niacin,** which looked promising, but although there appeared to be a reduction in non-fatal heart attacks, there were marked side effects: skin disorders such as darkening, itches and rashes, as well as digestive problems and gout.

d. **Gemfibrozil** (Lopid) was tested and again an increase in deaths was noticed in the treatment group although this time the numbers did not reach statistical significance.

e. **Compactin** which worked in a similar way to triparanol was withdrawn hurriedly and in some secrecy. The reason this time appears to be connected with cancer in dogs.

f. Lastly, despite the previous experiences with triparanol and compactin, yet another inhibitor, **Lovastatin,** has been approved for lifetime use on the general public after tests of very short duration only. (Derivatives pravastatin and simvastatin are marketed as Lipostat and Zocor.)

A study of all trials into cholesterol lowering by drugs up to 1987 showed an *increase* in mortality in those treated with drugs of 13.6%.



In 1993 a meta-analysis of all randomised controlled trials of cholesterol-lowering treatments showed that only those with very high risk showed evidence of benefit. In all others mortality was increased. Its authors conclude that:

any

"Currently evaluated cholesterol-lowering drugs seem to produce mortality benefits in only a small proportion of patients at very high risk of death from coronary heart disease . . . a cautious approach to the use of cholesterol lowering drugs should be advocated".

Despite this, nearly eight times as many prescriptions for cholesterol-lowering drugs were being issued just 6 years later!

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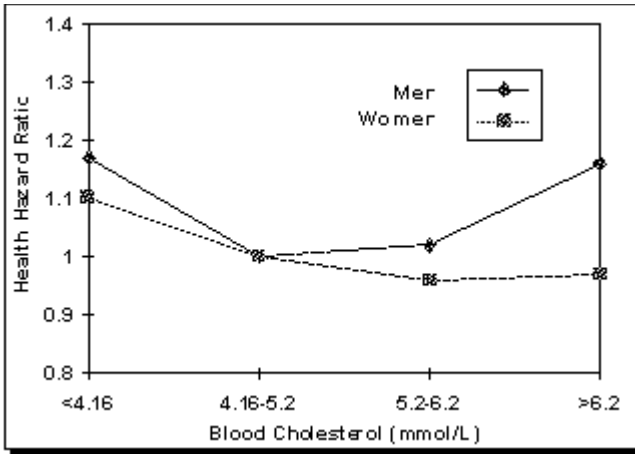
Part 6: Has Anyone Gained?

So far we have been looking at cholesterol lowering in terms of numbers of deaths, but the trials have shown impressive results in the reduction of non-fatal heart attacks and a consequent improvement in the quality of life. In the case of drugs, the reduction was in the order of twenty-three percent. Many see this as proof that lowering cholesterol in the total population, by whatever means, is worth fighting for.

But those trials were conducted on men rather than women. They were also conducted on those who had hypercholesterolaemia or, at least, very high blood cholesterol levels - not people with normal levels. They totally overlook the now well-established, non-linear relation between blood cholesterol and heart disease that indicates that lowering blood cholesterol in the general population is not economically worthwhile. The widespread agreement that the mainstay of the campaign should be a change in diet and lifestyle for all also overlooks the complete lack of evidence that such a course would have any significant beneficial effect. It even overlooks the fact that the trials involving cholesterol lowering by dietary means did not show any significant reductions in blood cholesterol.

In 1992 a report of 19 major studies published over the past twenty years suggested that public policy for reducing blood cholesterol should be reviewed. The graph below plots the relative mortality risk from all causes associated with levels of blood cholesterol in men and women. In the case of women, you can see clearly that risk rises as blood cholesterol falls. The report's author, Dr. Hulley, states:

"We are coming to realize that the resulting cardiovascular research, which represents the great majority of the effort so far, may not apply to women".



With men, the situation is more complicated as the curve is U-shaped. However, it is still noticeable that the risk with low cholesterol is similar to the risk with high cholesterol. Dr. Hulley concludes:
"the findings call into question policies built over several decades on evidence that focussed only on CHD as an outcome . . . it may be time to review national policies aimed at shifting the entire population distribution of blood cholesterol to the left."

Another analysis based on a number of American studies estimated that on a lifelong programme of cholesterol reduction by diet, the gain in life expectancy for those at very high risk (that is the 1 in 500 with hypercholesterolaemia) would be between eighteen days and twelve months, and for those at low risk (that is the other 499) between three days and three months. That is not very much with which to tempt people to endure a lifetime of unpalatable diets. And these figures assumed that cholesterol lowering was both effective and safe: they didn't take into account the increased risk of other debilitating and fatal diseases. Once these are added to the equation, it becomes quite evident that the current campaign is certain to do more harm than good. A study of Maori in New Zealand showed that those with the *lowest* levels of blood cholesterol had the *highest* mortality. Findings also borne out by the Framingham Study.

What we have then is a number of very large-scale, long-term human intervention studies showing that lowering blood cholesterol is possible but that it has no beneficial effect on coronary heart disease in the general population, and other studies showing that a low blood cholesterol level, or the methods used to attain it, are increasing the incidence of other serious killer diseases.

Thirty years ago it was said that

"current medical thinking . . . is that while cholesterol may be involved in some way with arteriosclerosis and heart disease, it is no longer held to be the main factor." . . . "A recent survey of cholesterol findings in geriatric cases involving arteriosclerosis showed a significant number of patients to have normal or low cholesterol."

Those remarks have been confirmed by all the major studies published to date. Forty years after the Framingham Heart Study began, its researchers looked at total mortality and cholesterol. The evidence was that for those with low cholesterol levels, deaths from non-cardiac causes offset any reduced incidence of heart disease. There was *"no increased overall mortality with either high or low serum cholesterol levels"* among men over forty-seven years of age. There was no relationship with women older than forty-seven or younger than forty. The researchers also concluded that people whose cholesterol levels are falling may be at *increased* risk.

And ten years later the Framingham researchers say: **"Intakes of fat and type of fat were not related to the incidence of the combined outcome of all cardiovascular diseases or to total or cardiovascular mortality."** Thus we now have fifty years of studies all demonstrating that animal fat is harmless.

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Part 7: So Where Does That Leave Heart Disease?

All published efforts to help by drug or dietary reduction of blood cholesterol have uniformly failed. Sir John McMichael, Professor Emeritus of Medicine, University of London

Is coronary heart disease really the major killer it's made out to be? It is true that a large percentage of deaths in Britain are attributed to CHD. The question is: Is this a cause for concern? As you can see in Table VI, CHD deaths have increased in people over seventy-five years of age. But does this illustrate a problem?

Table VI: - *CHD Mortality in UK Over Age 70 by Sex and Age*

Ages	70-74	75-79	80-84	Over 85
Men				
1975	16297	12561	8666	6270
1995	13379	12975	12223	10254
Women				
1975	10598	12868	12589	14617
1995	7695	9915	13717	21263

It is a fallacy to believe that if these people had modified their diet or lifestyle, they would still be alive. Despite what the health industry tells us, we are not an immortal species and cannot expect to live forever. I suggest that these figures merely show that people tended to live longer in 1995 than in 1975. This is true of both sexes and that, surely, is a good thing.

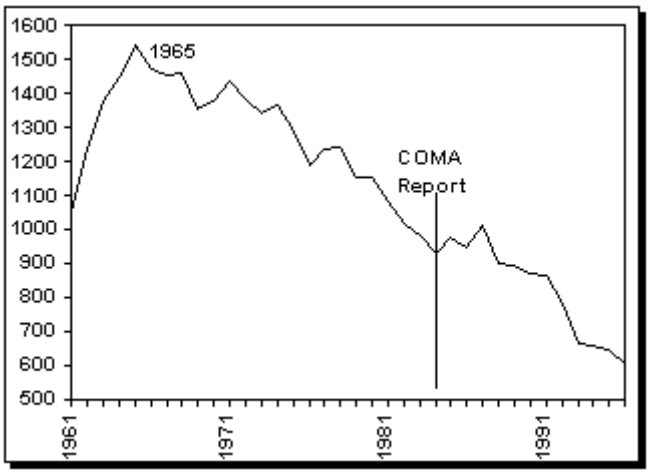
Premature death from CHD is a legitimate concern. If dietary change can reduce premature deaths it is arguably to their advantage that people be urged to change their ways. However, Table VII illustrates clearly that CHD deaths have fallen considerably in all under-seventy age groups and both sexes over the past two decades.

Table VII: – *CHD Mortality in UK Under Age 70 by Sex and Age*

Ages	40-44	45-49	50-54	55-59	60-64	65-69
Men						
1975	1290	2914	5783	7214	11678	15448
1995	643	1473	2261	3766	6170	9591
Women						
1975	202	473	1072	1902	3950	7104
1995	124	262	480	979	2028	4188

Some say that this is evidence that 'healthy eating' is working. Do not be misled. This reduction cannot be the result of the 'healthy diet' recommendations - they only began with the COMA report of 1984, but *premature* CHD deaths had started to decline nearly twenty years before in 1965, as is graphically illustrated below in men aged 40-44.

This was a time when people were brought up or spent the greater part of their lives with the recommendations with which this paper began. They had free, full-cream milk at school, ate bread and dripping and fried breakfasts. During the period after World War II when deaths from CHD peaked and started to fall, rationing had ended and a diet that was relatively high in fat was the vogue.



Not that this will come as any surprise to the Medical Research Council. In its report on the Caerphilly Study published in 1993, the MRC's Epidemiology Unit at Cardiff showed that men who drank more than a pint of full-cream milk a day had only one tenth the incidence of heart disease as those who drank none. They also demonstrated that those who ate a high-energy diet lived longer than those who cut dietary fats. Their findings indicate that far from being a killer, the diet we are told to avoid by the nutritionists may actually protect us against heart disease!

These findings confirmed a Japanese study of 1992. Japan has low levels of death from coronary heart disease but Okinawa has the lowest of all. While blood cholesterol levels are generally low in Japan, Okinawa's levels are much higher: similar to those in Scotland. In 1994 a paper examined the relationship of nutritional status to further life expectancy and health in the Japanese elderly based on three epidemiological studies. It found that Japanese who lived to the age of one hundred were those who got their protein from meat rather than from rice and pulses. The centenarians also had higher intakes of animal foods such as eggs, milk, meat and fish. Significantly, their carbohydrate intake was lower than that of their fellow countrymen who died younger.

An example of increasing risk

I have noticed, as I preach my gospel, that many women say "I'd rather drink skimmed milk. I don't like the taste of full-cream milk now, it's too rich". This is a trend that worries me.

We all need calcium but women need a good supply to prevent osteoporosis in later life. Milk is the best dietary source of calcium. As all the calcium in milk is in the milk, not in the cream, skimmed milk contains slightly more calcium than full-cream milk. On the face of it, therefore, it looks like a good idea to drink skimmed milk. BUT for calcium to be absorbed from the gut, it has to be there in the presence of fat and vitamin D - and skimmed milk contains neither. As a result, while just over fifty percent of the calcium in full-cream milk is absorbed, only about five percent is absorbed from skimmed milk. AND if you drink your skimmed milk with bran muesli for breakfast, you probably won't absorb even that five percent.

In 1979 the late Professor Sir John McMichael performed an inquest on the diet/heart hypothesis. Pointing out that

"All published efforts to help by drug or dietary reduction of blood cholesterol have uniformly and convincingly failed" . . . "we need a fresh approach to the problem at scientific level and should avoid further public speculation and confusion by repeated propaganda through the media until we have clarified our own professional minds and shaken off what most critical doctors are likely to regard as an untenable hypothesis of causation."

It is a pity that no-one seems to have taken any notice of him.

Fat has over twice the energy value of either carbohydrates or proteins, and other essential nutrients: lipids used in the brain and central nervous system without which we become irritable and aggressive; sterols, precursors of the bile acids and a number of hormones (including the sex hormones); and the fat-soluble vitamins A, D, E and K. The

late Dr. John Yudkin, when Professor of Nutrition and Dietetics at London University, called fat the most valuable food known to man. It is both stupid and wasteful to throw it away.

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Part 8: A Question of Ethics

Is it ethical to impose a regime on people in the hope that heart disease will be reduced? Surely prevention is better than cure, you may say. But is it? Such an attitude ignores the real possibility that such intervention may do more harm than good. 'Preventative' medicine as practised in the case of heart disease, takes two forms. Firstly we are to change our lifestyles, and secondly we are screened by our doctors on an opportunity basis.

But this screening is not prevention of the disease, it is merely the early detection of it. For such procedures to be of use a number of criteria are well established. One important one is that the disease should be both common and serious, as screening for an uncommon disease will throw up many false results. These will inevitably incur the cost of further testing, and cause unnecessary anxiety which itself is harmful.

The first problem with screening in CHD, is deciding what to test for. As a predictor of coronary risk, total blood cholesterol turns out to be irrelevant, and merely testing for that is regarded by many experts as misguided. Far more reliable, they claim, is measurement of HDL (the 'good' cholesterol). However, in a test of the accuracy of checking for HDL at various laboratories, values differed by as much as 40% in 95% of the samples tested. In another study, 16 instruments manufactured by nine companies were tested in 44 laboratories. In this test, although the inaccuracies of the machines were lower at 3.6-4.4%, biases attributed to the methods used ranged from -6.8% to +25%. The accuracy of desktop machines is even more suspect.

A third study to evaluate the ability of cholesterol screening to detect individuals with blood cholesterol abnormalities concluded that 41% of those with abnormal levels would not be detected using present guidelines.

Another criterion is that an effective treatment for the disease is available, as there is little point in early diagnosis or detection of a disease for which there is no effective remedy.

Some will say that we do know the cause of coronary heart disease; it is high cholesterol, or too much fat in our diets, or not enough exercise. Or it could be something else. In 1981, two hundred and forty six 'risk factors' for heart disease were listed. That number is now well over three hundred. These so called risk factors include having English as a mother tongue, having a diagonal crease in the left earlobe, not taking siestas, not eating mackerel, snoring and wearing tight underpants. What a list of this size really tells us is that we have little idea what causes coronary heart disease. And it is certain that if all the 300 plus do play a part, we have no chance of defeating the disease.

A director of the Health Education Programme of the American Medical Association denounced the lifestyle changes with their false promise of benefit as a quasi- religious crusade when in 1984 he wrote: " *Constant lifestyle self-scrutiny in search of risk factors, denial of pleasure, rejection of the old evil lifestyle and embracing a rigorous new one are followed by periodical affirmations of faith at revival meetings. . . the self-righteous intolerance of some wellness zealots borders on health fascism. Historically, humans have been at greatest risk while being improved in the best image of their possibilities as seen by somebody else."*

Telling people who feel fit and well that they are not and, that if they do not make major changes to their lives, they could drop dead at any moment, not only worries them unnecessarily, it can have a profound effect on their attitudes to life. The benefits of mass screening are doubtful and the risk of harm is high. Such intervention, therefore, can only be justified ethically when either the patient has requested it or symptoms are such as to make it desirable.

If we go to our doctor with a complaint and he treats us with the best medical knowledge, he should not be held responsible for defects in that knowledge. If, however, the doctor initiates treatment without being consulted by the patient, then he is in a very different situation. Cochrane and Holland write that before advocating a course of action in such circumstances, " *He should, in our view, have conclusive evidence that screening can alter the natural history of disease in a significant proportion of those screened."* If he does not, he may be held responsible for any harm done.

But in the case of heart disease, recognised medical standard tests and ethics have been thrown out the window. The recommendations were forced on the public even before they had been tested, and now the perpetrators are afraid to admit that they could have been wrong. But until they do, whole populations are suffering unnecessarily.

In the United States blood cholesterol level testing for all is routine and that nation is becoming a nation of 'cholesterophobics'. More concerned with death than with life, many interviewed said that their lives were ruined as, if they had a treat, it was accompanied by feelings of guilt. One of COMA's principles is that the measures should " *afford a reasonable prospect of improvement in life expectancy overall, and in the quality of life for the population as a whole."* Experience around the world, and particularly from the United States, makes it certain that neither of those principles will be met.

In Britain, general practitioners, practice nurses and health visitors are starting to use desk-top cholesterol testing machines, the majority of which have been loaned by drug companies. A suggestion in the *Lancet* is that this is designed merely to enhance the drug companies' profits by increasing sales of cholesterol-lowering drugs, and questions their ethics. There is also the question of the psychological harm that could be done to people in view of the United States experience of the inaccuracy of such machines.

Medical bias towards illness

There is evidence that the medical profession is biassed in favour of diagnosing illness rather than health. A classic example was a test run in New York on 1,000 11-year-old children and their tonsils. On first examination 61% were found to have had their tonsils already removed. The other 39% were re-examined by a group of doctors who recommended tonsillectomy operations for 46% of them. The rest were again examined by yet another team and, again, nearly half were recommended for operations to remove their tonsils. After three examinations, only 65 of the original 1,000 had not been recommended for the operation. The test ended there as they ran out of physicians to perform the examinations. However, if the physicians had had their way, it is obvious that a great many unnecessary operations would have been performed.

High error rate in diagnoses

The bias towards illness may also combine with a high diagnostic error rate. Post- mortem examinations in a British university hospital showed that of the patients who had died of a diagnosed specific heart disease, over half had actually died of something else. And when the same sample was tested in different laboratories, different results were given in 25% of cases. Diagnostic machines, it seems, are no better. In a competition between doctors and computers in 83 cases recommended for pelvic surgery, pathology showed that both the doctors and computers were right in only 22 cases. In 37 the computers proved the doctors' diagnoses wrong, in 11 the doctors showed the machines to be in error and in 10 cases they were both wrong.

Variations between countries

There also exists a large variation between diagnostic and prescribing practices between countries which makes comparisons between them of little use. For example, in Britain, a patient is 7 times more likely to be prescribed a course of vitamins than in Sweden, and in Sweden, 8 times more likely to get gamma globulin medication than in Britain. United States surgeons perform operations twice as readily as in Britain and the French will amputate almost anything. Appendicitis and deaths attributed to it is diagnosed in Germany 3 times more frequently than in any other country.

And so to the cost

In the late 1980s, intervention alone in the United States was estimated at $14 billion a year. The cost of cholestyramine for an estimated five million people at 1990's prices was $10 billion to which up to another $10 billion must be added for laboratory tests and doctors' services. In Britain, if we also undertook a mass screening and cholesterol reducing programme, it has been suggested that drug treatment would be recommended for 10% of men aged 40-69 and, as a result, the NHS drug bill in England and Wales, £2.3 billion in 1992, would be increased by 20%. To put it in terms more familiar to the average person, the cost of the drugs alone would be between £80 and £120 per person per month. The evidence suggests that for that money we might be able to delay a fatal heart attack in the average person by between 3 days and 3 months - but shorten that person's life by a larger amount as he or she died of cancer, osteoporosis or stroke.

The effect on the NHS

Sir William Beveridge set up the National Health Service on the assumption that *"there exists in any population a strictly limited amount of illness which, if treated under conditions of equity, will eventually decline."* It was calculated that the cost of the service would fall as the rates of illness went down. No-one considered that the NHS would redefine and broaden its service to such an extent that only budgetary restrictions would keep it from expanding indefinitely.

The increasing sophistication of treatments available and demanded of the National Health Service are putting it under a tremendous strain. To spend scarce money and resources on any unnecessary treatment is waste, but to waste billions of pounds on such unproven and dubious hypotheses as the present, so-called 'healthy eating' recommendations is quite irresponsible and, in the long term, can only be harmful.

Dr Halfdan Mahler, Director General, the World Health Organisation recognised such waste when he said in 1984:

"The major - and most expensive - part of medical technology as applied today appears to be far more for the satisfaction of the health professions than for the benefit of the consumers of health care."

Side effects

The current 'diet-heart' strictures and media pressure aimed at ever lower blood cholesterol levels, have driven more people towards unnatural and unhealthy cult diets. Consequently, there has been a rapid rise in the incidence of infant malnutrition, deficiency diseases and other killer or debilitating diseases. Without sufficient dietary fat, the body is unable to use the fat soluble vitamins. Without vitamin D the body cannot utilise calcium. In conjunction with an increase of bran in the diet, this is another possible factor in the growing incidences of diseases such as osteoporosis and rickets.

Vegetarian traits are increasing. As animal products are the only natural source of vitamin B-12, Vegans, who eat no such animal products, run a real risk of pernicious anaemia. Bottles of pills are not a good substitute as they are generally poorly absorbed. Fermented soy products, such as tempeh, and spirulinas found in health-food shops, which are supposed to contain vitamin B-12, for the most part contain only analogues of the vitamin which are not active for humans and which, in some cases, actually block vitamin B-12 metabolism. Children of Vegans also usually have a lower body weight and height and suffer other health problems.

Doctors in Britain are reporting cases in 'the muesli belt' of severe nutritional disorders which include kwashiorkor, marasmus and rickets which are due solely to their parents' food faddism. Until recently, these diseases were only found among severely malnourished children in Africa. In Britain it is becoming so serious that they suggest that such cases should be regarded as forms of child abuse. But are the parents to blame? Could not some of the blame for this deplorable situation be fairly laid at the doors of the nutritionists?

Doctors in the USA also are reporting ever increasing numbers of children suffering from nutritional dwarfing and other deficiency problems attributable entirely to pressures to eat nutrient-poor, low-calorie foods because they are 'healthy'. These children are destined to have far-reaching problems beyond just being smaller than their peers. It has been shown that adults whose birth-weights early rates of growth were low have a much higher incidence of CHD. Brain growth and intelligence are also found to be much lower in such undernourished children.

We really seem incapable of learning from previous experiences. During World War II, when we are supposed to have been so healthy, protein-calorie deficiency was so pronounced that in many people pathology showed there was as much as 25% loss of muscle from their hearts - and similar patterns of protein deficiency are found today.

And it is not just humans who suffer side effects. In the constant quest for ever leaner meat, food animals are being engineered which could not survive naturally. Belgian Blue cattle, for example, bred to provide lean meat, have double muscles. This makes the calves too large to pass along the birth canal and they have to be delivered by Caesarean section. Other cattle and pigs are fed hormones to make them grow with less fat. As yet it is anyone's guess what the long-term consequences of this will be on both the animals and humans.

The strictures against red meat also mean that fewer cattle and sheep are being reared and more fields are used to grow cereals, rape and other vegetable crops. Unlike the animals, which on the whole produce natural fertiliser for the pastures, the vegetable and cereal crops require large amounts of manufactured nitrogen fertilisers to be spread. As we know, these leach in ever-increasing quantities out of the soil to pollute our water supplies. Grass, the food of the cattle and sheep, on the other hand, locks the nitrates in the soil, thus preventing pollution.

The Mediterranean diet

The 'Mediterranean' diet is healthier than ours, we are told. We should eat what the French, Italians and Spanish eat. That could be right - but not for the reasons usually given.

The Mediterranean diet is what the health fanatics advocate because, they say, it is low in fat. This is nonsense! Obviously, they have never been there. They don't seem to know that northern Italians love butter, that bowls of pork dripping are sold on Spanish markets or that the Spanish spread it thickly on their toast for breakfast. They don't know that goose fat is used to make *cassoulet* in the south of France, or that throughout the Mediterranean the sausages, salamis and pâtés all contain up to fifty percent fat.

The Mediterranean diet may be healthier than the British but, contrary to popular belief, it is very far from being a low-fat diet!

However, there are a number of major differences between the Mediterranean countries and Britain that may play a significant part in their effects on health. Not only is the food eaten by the average working family in southern Europe very different from that eaten by a typical family in Britain, more importantly, the way it is bought, presented and eaten is also different. A brief list of the principal differences is tabled below.

Mediterranean Eating Pattern	British Eating Pattern
The average Mediterranean diet comprises natural, unprocessed meat, vegetables and fruit that are usually bought fresh daily.	The average British diet is composed of packaged, highly processed foods with chemical additives.
Meat plays an important part in the diet	We are told to eat less meat
Fats eaten are butter, olive oil and unprocessed animal fats	Fats eaten are highly processed margarines, low-fat fat substitutes, and vegetable oils.
Meals are taken slowly, without hurrying. Lunch usually takes up to two hours - and is followed by a siesta .	Food is rushed. Lunches are eaten on the run or combined with work. Often, they are junk-food snacks.
Over sixty percent of energy intake is before 2.00 pm.	The largest meal is eaten in the evening
Wine (believed to be protective against heart disease), is drunk during meals as part of that meal.	Beer, wines and spirits are drunk in the evening after the evening meal.

Cholesterol testing

Imagine it is 2.00 a.m., you are lying in bed when you hear a noise downstairs that you know is caused by a burglar. You know how quickly your heart starts to race. Well, that is how quickly your cholesterol level can rise - and for the same reason. One of the effects of the 'fight or flight' reflex is to raise blood cholesterol. Any form of physical or mental stress has this effect. So if you run to your doctor's, your cholesterol level will be higher than if you walked; if you have been standing it will be higher than if you sat. If you are anxious, or your doctor looks worried, it will be higher. If your blood cholesterol were tested hourly throughout a day, or daily over a month, it would not be unusual to find a wide variation in values.

Blood cholesterol levels also rise naturally as you get older so that while a reading of 9 mmol/l is high at the age of twenty, it is perfectly normal if you are fifty.

Cholesterol measurements are not very accurate - less than eighty percent - even when conducted in a laboratory. A survey showed that on the same sample, laboratories could differ by as much as 1.3 mmol/l. When it is tested with a doctor's desktop machine the accuracy will inevitably be lower.

To put it in perspective, let us assume that you are around thirty years old and your cholesterol level is a perfectly respectable 6.0 mmol/l. You hurry to the surgery and are anxious about the result. This could raise it by twenty-five percent to 7.5. If it is sent to a laboratory giving the high readings it could be raised by a further 1.3. Your perfectly normal 6.0 is now a high 8.8!

In fact, so many variables affect cholesterol levels that a one-off test is a waste of time, and an unnecessary worry for the patient that can do more harm than good. Bear that in mind if you are subjected to a cholesterol test.

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Part 9: The Dangers of a "Healthy" Diet

'Healthy eating' tells us to eat low-fat, high-carbohydrate diets but in the last few years of the Twentieth Century several papers demonstrated the harm this could do.

Obesity

Back in 1932 obese patients on different diets lost weight thus:

- Average daily losses on high carbohydrate/low fat diet - 49g
- Average daily losses on low carbohydrate/high fat diet - 205g

Drs Lyon and Dunlop say:

"The most striking feature of the table is that the losses appear to be inversely proportionate to the carbohydrate content of the food. Where the carbohydrate intake is low the rate of loss in weight is greater and conversely."

It's no coincidence that the numbers of people getting fat has risen dramatically since 'healthy eating' was advocated. As long ago as 1863 it was shown that low-fat, high-carbohydrate diets make people fat. The medical world is at last waking up to this fact. In 1994 Professor Susan Wooley of the University of Cincinnati's College of Medicine and David M Garner, Director of Research at the Beck Institute for Cognitive Therapy and Research wrote that:

"The failure of fat people to achieve a goal they seem to want and to want almost above all else must now be admitted for what it is: a failure not of those people but of the methods of treatment that are used."

In other words, blaming the overweight for their problem and telling them they are eating too much and must cut down, is simply not good enough. It is the dieticians' advice and the treatment offered that are wrong. Wooley and Garner conclude:

"We should stop offering ineffective treatments aimed at weight loss. Researchers who think they have invented a better mousetrap should test it in controlled research before setting out their bait for the entire population. Only by admitting that our treatments do not work and showing that we mean it by refraining from offering them can we undo a century of recruiting fat people for failure."

In 1997 two more Americans, Drs AF Heini and RL Weinsier noticed the trend and blamed it on low-fat diets saying:

"Reduced fat and calorie intake and frequent use of low-calorie food products have been associated with a paradoxical increase in the prevalence of obesity".

Heart disease and diabetics

Obese people tend to go on to suffer type II diabetes (NIDDM) and diabetics are more prone to heart disease. For this reason patients with NIDDM are counselled to eat a 'healthy' low-fat, high-carb diet. But as a paper in the medical journal, *Diabetes Care* , pointed out

"Low-fat, high-carbohydrate diets eaten by patients with diabetes (NIDDM) have been shown to lead to higher day-long plasma glucose, insulin, triglycerides, and VLDL-TG, among other negative effects. In general, study has demonstrated that multiple risk factors for coronary heart disease are worsened for diabetics who consume the low-fat, high-carbohydrate diet so often recommended to reduce these risks."

In June 1999 the 81st Annual Meeting of The Endocrine Society was told:

" **A very high-fat, low-carbohydrate diet has been shown to have astounding effects in helping type 2 diabetics lose weight and improve their blood lipid profiles. The thing many diabetics coming into the office don't realize is that other forms of carbohydrates will increase their sugar, too. Dieticians will point toward complex carbohydrates . . . oatmeal and whole wheat bread, but we have to deliver the message that these are carbohydrates that increase blood sugars, too** ."

. . .and postmenopausal women

In 1997 it was discovered that

"Low-fat, high-carbohydrate diets [15% protein, 60% carbohydrate, 25% fat] increase the risk of heart disease in post-menopausal women."

. . . in fact everyone

Dr. Gerald M. Reaven, of Stanford University School of Medicine in California, and colleagues compared the effects of a low-fat, high-carbohydrate diet [25% fat, 60% carb, 15% protein] with a high-fat, low-carbohydrate diet [45% fat, 40% carb, 15% protein], on blood fats and cholesterol. They found their subjects had significantly higher fasting plasma triglyceride concentrations, remnant lipoprotein cholesterol concentrations, and remnant triglyceride concentrations when they were on the high-carbohydrate, low-fat diet, both after fasting and after breakfast and lunch. The study participants also had significantly lower HDL (the 'good' cholesterol) concentrations on this diet. The authors conclude:

"Given the atherogenic potential of these changes in lipoprotein metabolism, it seems appropriate to question the wisdom of recommending that all Americans should replace dietary saturated fat with [carbohydrate]."

But then, in 1992, from the Framingham study again came:

"In Framingham, Mass, the more saturated fat one ate, the more cholesterol one ate, the more calories one ate, the lower the person's serum cholesterol" . . . "we found that the people who ate the most cholesterol, ate the most saturated fat, ate the most calories, weighed the least and were the most physically active."

Low-fat, high-carb diet and breast cancer

And that's not all:

The largest and most comprehensive study on diet and breast cancer to date found that:

- women with the lowest intake of fat had a significantly higher incidence of breast cancer and
- women with the highest intake of starch also had a significantly higher incidence of breast cancer.
- Saturated fats were not implicated in breast cancer.

The biggest study so far into the relation between breast cancer and fat intake is the Nurses' Health Study, conducted by Harvard University Medical School. A total of 88,795 women free of cancer in 1980 were followed up for 14 years. Comparing breast cancer rates in women who derived more than thirty percent of their calorie intake from fat with women who derived less than twenty percent of calories from fat, they show that those on low-fat diets had a higher rate of breast cancer than those who ate more. They went on to look at the various different types of fats and found that breast cancer rates were lower for all types except one: omega-3 fish oils, which are touted as 'healthy', were the only ones that **increased** cancer rates. However, the increase was small. Dr Michelle Holmes and colleagues conclude:

"We found no evidence that lower intake of total fat or specific major types of fat was associated with a decreased risk of breast cancer" .

Carbohydrates are not healthy

As we have seen so far, the emphasis on increasing carbohydrates at the expense of fats has not been an unqualified success. And there are good reasons for this.

We have known since 1863 that carbohydrates cause obesity; since 1935 that they cause diabetes; since 1941 that they increase aggressiveness and criminality in children; for almost 30 years promote coronary heart disease; and more recently that they increase the risk of cancers. So is it merely coincidence that diseases in whose aetiology carbohydrates are implicated have risen so dramatically since we have eaten more carbohydrates?

No. Healthy eating is becoming something of a disaster. The best advice appears to be that we should:

- **reduce** carbohydrate intake and
- **increase** our intake of animal fats.

To sum up, what emerges from this discussion is:

Fats

The totality of evidence suggests that we should eat animal fats in preference to vegetable oils because:

1. Polyunsaturated fats found in margarines and cooking oils may lower cholesterol levels but they increase cancer risk.
2. Trans-fats found in highly processed margarines and oils also increase CHD risk.
3. 'Healthy' omega-3 oils may increase cancer risk.
4. Monounsaturated fats are no better as far as heart disease is concerned but they may reduce cancer risk.
5. Saturated fats are healthier in CHD, particularly if you have already had a heart attack. They are not implicated as a cause of cancer.
6. Conjugated linoleic acid found only in animal fats is a powerful anti-cancer agent.
7. Animal fats are just under half saturated and just under half monounsaturated, with a small, but sufficient proportion of polyunsaturated fats.

Carbohydrates

- Carbohydrate intake from sugars and starches in breakfast cereals, bread, pasta, rice, et cetera, should be reduced because they increase diseases including obesity, cancer, diabetes and CHD.

Bran

- Bran (cereal fibre) should be avoided like the plague.

Conclusion

An assessment of all the cholesterol-lowering dietary trials published in 1987 showed an aggregate six percent **more** deaths in those who adopted a cholesterol-lowering diet over those on a free diet. A similar review of drug trials showed an aggregate of over thirteen percent more deaths in those taking cholesterol-lowering drugs.

More resources, time and money have been spent over the last fifty years on coronary heart disease than any other disease in medical history and all it has proved is that doctors don't know as much as they thought they did. If half a century of serious research has failed to find a causal link between a fatty diet and heart disease, it can only be because there is no link.

To make intelligent decisions you must be given advice that is based on proven facts rather than unfounded assumptions. And the facts at present seem to be that milk, cream, butter, meat and fresh fruit and vegetables are the healthy foods whilst high-in-polyunsaturates spreads and oils, bran flakes and packaged foods are not.

Seventy years after it began we still do not know what caused the dramatic rise in coronary heart disease deaths in the 1920s or why coronary mortality is now falling. But one thing that the last fifty years of studies has demonstrated is that cholesterol has had very little to do with it.

The research has also demonstrated no evidence of a need to endure an unpalatable, fatless, bran-laden diet. Apart from being less pleasurable to eat, it is now clear that 'healthy eating' is not so healthy after all.

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THE CHOLESTEROL MYTH

by T.J. Moore

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Diet has hardly any effect on your cholesterol level; the drugs that can lower it often have serious or **fatal side effects**; and there is **no evidence at all that lowering your cholesterol level will lengthen your life.** In fact, recent JAMA article revealed that cholesterol-lowering drugs actually INCREASE your risk of heart attack by 241%

An article drawn from Thomas J. Moore's book, 'Heart Failure', published by Random House, Inc.

Quote from the article:
"THE RISKS OF LOW CHOLESTEROL might have remained a mostly hypothetical question had not the Food and Drug Administration in September of 1987 approved the cholesterol-lowering drug lovastatin. .....

Lovastatin, or Mevacor, was approved and is now moving into widespread use without having completed any trials to measure its effect on coronary heart disease and systematically monitor potential side effects."