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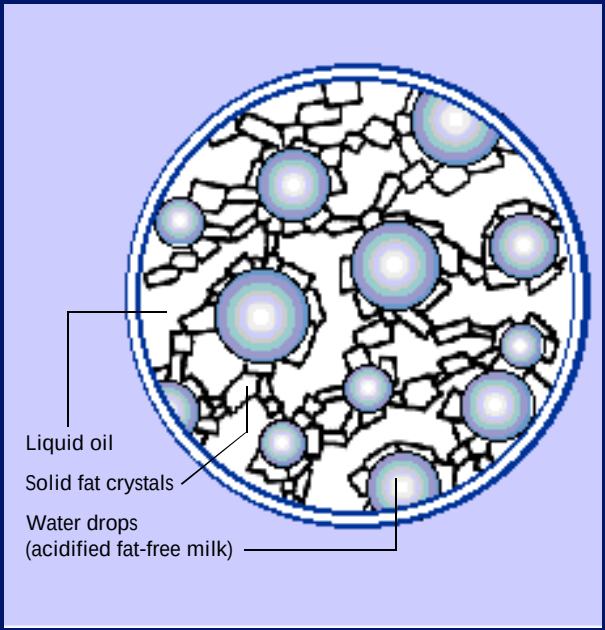

DIETARY FAT

SOME ASPECTS OF NUTRITION AND HEALTH AND PRODUCT DEVELOPMENT

ILSI
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DIETARY FAT

SOME ASPECTS OF NUTRITION AND HEALTH AND PRODUCT DEVELOPMENT

by A. Stewart Truswell

ILSI Europe

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Printed in Belgium

ISBN 0-944398-74-X

FOREWORD

Fats are attractive dietary constituents, but excess consumption may lead to a range of problems. An IFTEC (International Food Technology Exposition and Conference) symposium held at The Hague, The Netherlands, on 18 November 1992 addressed some of the problems and opportunities associated with fats. The speakers were Dr. O. Korver (NL), Dr. J.L. Zevenbergen (NL), Dr. E. Riboli (F), Dr. D. Kromhout (NL), Prof. J.E. Blundell (UK), Dr. D.J. Mela (UK), Dr. A.C. Juriaanse (NL), Dr. A.T. Grosch (USA) and Prof. B.G. Swanson (USA).

This short, easy-to-read booklet, written by Prof. A.S. Truswell, is based on the symposium. It deals with some nutritional aspects of fats (effects on plasma cholesterol, cancer — energy intake relationship) and food technologists' problems and achievements in producing reduced-fat and reduced-saturated-fat foods using fat mimetics and substitutes.

Author: A. Stewart Truswell
Series Editor: Nicholas J. Jardine

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INTRODUCTION

Edible fats are important food components that enhance palatability by providing texture and enhancing flavour. They also provide essential fatty acids and fat-soluble vitamins. We enjoy eating foods containing fat, but there is a negative side: excessive consumption may not be good for health. The question is, What are the right amounts and types of fat we should use and eat?

The average consumer is somewhat aware of the relationship between dietary fat and health. Despite this, there is still a great deal of confusion. Scientists use technical jargon, formulate hypotheses and express themselves in terms of probabilities. Journalists look for news, which is not always balanced. Governments and industry translate complex research findings into information that can be used to make policies and to educate the consumer. A balance must be achieved between complicated scientific communication and information that can be more easily communicated and understood by the public. In this booklet, we have tried to present information about dietary fat and health in a straightforward manner.

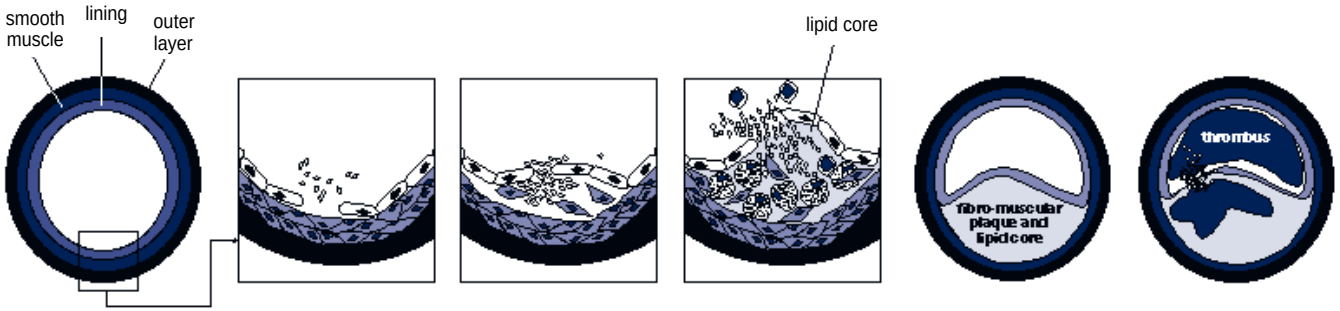
INFLUENCE OF FAT ON BLOOD LIPID LEVELS AND OTHER RISK FACTORS FOR CORONARY DISEASE

The basic facts

- Coronary heart disease (CHD) is the leading cause of death in the industrial countries except Japan and perhaps France.
- Genetic potential is key to determining whether CHD disease is expressed in any particular environment.
- The incidence of CHD is influenced by environment, because immigrant groups often take on the incidence of their new host country and because there have been large changes during this century in deaths from CHD. It was uncommon before 1925 and then rose steadily except for a dip in Europe during the Second World War. However, since the mid-1960s CHD death rates have decreased considerably in the United States, Australia and, to a lesser extent, some other western European countries. Meanwhile they have risen in the countries of eastern Europe.
- CHD is a multifactorial disease, and diet is one of the fundamental environmental parameters, operating through blood cholesterol and other factors. Other major risk factors are smoking, high blood pressure and a family history of premature CHD. Additional risk factors are diabetes mellitus and physical inactivity and obesity. However, these do not account for all of the risk, so there must be other, unknown factors.
- The above risk factors have been established by large prospective (or cohort) studies in which healthy middle-aged people are examined for life-style and medical characteristics and then followed for years to see who develops the manifestations of CHD and who does not. Characteristics at the initial examination are then compared between individuals who develop CHD and those who did not. Prospective CHD studies have been conducted in at least 14 countries.
- The clinical manifestations of CHD are sudden death, myocardial infarction (i.e. heart attack) and angina pectoris (chest pain).
- The structural default inside the body — the pathology in CHD — is narrowing of the coronary arteries by excessive atherosclerosis which occurs underneath the inner lining (endothelium) and encroaches on the diameter of and the flow through the artery (Figure 1). Under the microscope atherosclerotic patches (called plaques) are seen to contain accumulations of cholesterol inside and between cells (including cells swollen from macrophage activity, so-called foam cells). Plaques may rupture and activate clotting of the blood and thereby impede or block blood flow through the narrowed artery. This sudden reduction of blood supply to part of the heart is often what produces symptoms.
- Most middle-aged and older people in Western countries gradually develop some degree of coronary atherosclerosis. But as a rule, disease symptoms occur only when there is severe narrowing of a coronary artery or when it is suddenly ("acutely") narrowed by thrombosis.
- Atherosclerosis tends to occur in most arteries in the body. If it is severe in an artery to the brain, it can lead to stroke. If it is severe in an artery to the leg, it can lead to circulatory trouble in the foot and pain on walking.
- The long-term risk of CHD increases with increasing blood plasma total cholesterol (Figure 2). Cholesterol is a fatty (lipid) substance that is completely insoluble

FIGURE 1.

The response-to-injury hypothesis of atherosclerosis



A normal artery, which has a convoluted lining. Damage to the lining, which occurs as a result of daily wear and tear from turbulent blood flow, can be repaired quite easily.

Minor injuries to the artery lining may become so frequent that normal repair mechanisms cannot keep up. Platelets gather to repair the excess damage which often occurs where blood flow is most turbulent.

The platelets aggregate to form a fibrous mass. Smooth muscle cells from the wall of the artery migrate into the lining. These are the first events in the build up of the atherosclerotic plaque.

Macrophages gather to repair the lesion. While doing so, they also engulf any LDL particles in the vicinity which have been damaged by free radical action. The macrophages swell to become foam cells and are deposited as part of the plague. The atherosclerotic plaque protrudes into the lumen of the artery, where it is exposed to turbulent blood flow, making second and subsequent injuries more likely.

After many years, the plaque may grow to a size where it restricts normal blood flow in the artery, and may cause symptoms such as angina (chest pain).

A plaque wall may eventually rupture, thereby triggering the formation of a blood clot (thrombus). A large thrombus can completely block an artery already narrowed by plaque, causing a heart attack by cutting off the blood supply to a section of heart muscle.

macrophage *scavenger white blood cell*

foam cell *macrophage after engulfing LDL*

smooth muscle cell

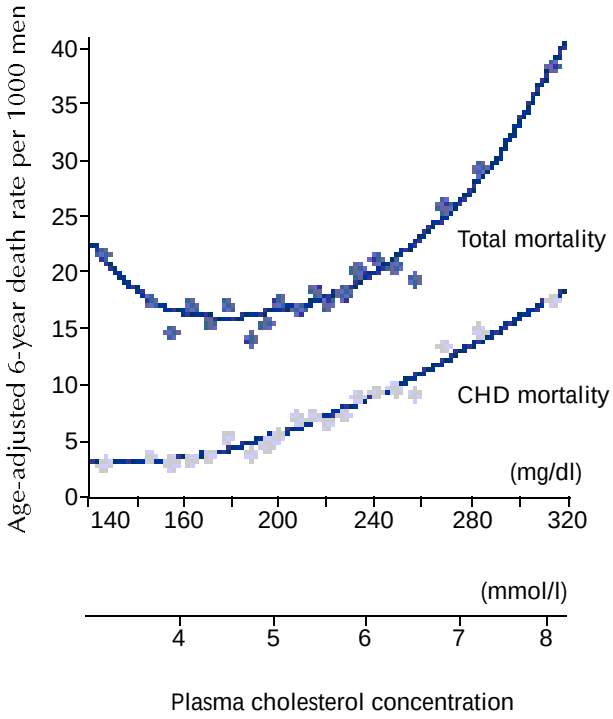
platelets *circulating particles involved in blood clotting*

Source: Bender AE, The Evidence for a Relationship Between Diet and Some Major Diseases in Europe (October 1989 Symposium). Brussels: ILSI Europe, 1994

in plasma. It is solubilized in specialised carrier proteins called lipoproteins. About 70% of total plasma cholesterol is carried in low-density lipoproteins (LDL). This form of cholesterol has been found in prospective studies to be a strong risk factor for CHD: the higher the plasma LDLcholesterol level, the greater the risk. The remainder of the plasma cholesterol — about 30% — is carried in high-density lipoprotein (HDL), which, by contrast, is protective against CHD. HDL cholesterol concentration is normally higher in women than in men, probably one reason why women tend to experience CHD some years later than men. Simply stated, the higher the LDL cholesterol level, the more cholesterol tends to accumulate in arteries, whereas HDL removes accumulated cholesterol in the tissues and carries it back to the liver for disposal.

FIGURE 2.

Relationship between blood plasma total cholesterol level and coronary heart disease in the MRFIT study



Source: Martin M et al, Serum cholesterol, blood pressure and mortality: Implications from a cohort of 361 662 men, et al, Lancet 2,(1986):933-936

- Although some of the cholesterol in our bodies is absorbed from the diet, most is made in the liver and other tissues in a series of steps starting with acetate. Cholesterol has many essential functions in the body. It is part of all cell membranes; in the brain it is a major component of myelin. It is also the precursor substance for the sex hormones, adrenocortical hormones and bile acids.
- In affluent societies on high-fat diets, levels of plasma total and LDL cholesterol in adults are markedly higher than in human populations that have lower rates of CHD.
- Cholesterol is lost from the body by excretion from the liver into the bile, some chemically altered as bile acids, some as unchanged cholesterol. These subsequently pass into the faeces as faecal bile acids and faecal neutral steroids, respectively.
- Our ability to predict an individual's risk of developing CHD is still incomplete. CHD can develop in people who have none of the known major risk factors such as raised plasma LDL cholesterol, high blood pressure, tobacco smoking and a strong family history of CHD.
- Careful trials aimed at preventing or reducing CHD have been carried out beginning in the 1960s. They are difficult to control and expensive to conduct, and interpretation has been controversial. The results have been encouraging in some but not all trials. Taken together they show that certain dietary measures resulted in a significant reduction in CHD deaths but that deaths from all causes were often reduced only in populations with a high incidence of CHD.

EFFECT OF DIETARY FATS ON PLASMA CHOLESTEROL

The classical findings

More than 20 years ago the research groups of Keys and Hegsted in the United States independently published the results of their many experiments on healthy men eating a wide variety of dietary fats. They found that saturated fats raised total blood cholesterol levels compared with a nutritionally equivalent carbohydrate intake. It had been thought since 1956 that polyunsaturated fats (rich in linoleic acid) reduced blood cholesterol, whereas monounsaturated fats (rich in oleic acid) had no effect. Although techniques to measure the levels of LDL cholesterol and HDL cholesterol were not well developed at the time, it was concluded that these changes in total cholesterol appeared to be the result of changes in LDL cholesterol. These findings formed the basis for dietary guidelines and recommendations in the Western industrialised countries in the 1970s and 1980s (see also Table 1):

- Total fat should be 30% or less of total energy intake.
- Saturated fat should be reduced to less than 10% of total energy intake.
- Polyunsaturated fats and oils should be 6-10% of total energy intake.
- Some expert committees also recommended low intakes of dietary cholesterol (less than 300 mg / day).

TABLE 1

Dietary principles aimed at reducing the risk of coronary heart disease

- Total fat intake should be equal to or less than 30% of total energy intake.
- Saturated fat intake should be less than 10% of total energy intake.
- Polyunsaturated fat should constitute up to 10% of total energy intake (of this, 10-20%, i.e., 1-2% of total energy, should be n-3 and the rest n-6), and it should contain adequate vitamin E, at least 0.6 mg / g polyunsaturated fat. The balance of dietary fat should be monounsaturated.
- Dietary cholesterol intake should be less than 300 mg / day.
- Dietary fibre (including soluble fibre) intake should be 25-30 g / day.
- Sodium intake should be less than 100 mmol (6 g salt) / day.
- Energy intake should be such as to achieve and maintain desirable body weight.
- Eat plenty of fish (including fatty fish), vegetables, fruits and whole-grain cereal.

PLUS, EXERCISE REGULARLY AND DO NOT SMOKE

NEWER DEVELOPMENTS

The polyunsaturated and monounsaturated fatty acid question

Between 1965 and 1985 controlled experiments on the effect of dietary fats and oils containing different fatty acids on plasma lipids were seldom done. It was thought that the different effects had been established, so researchers focused on other components, such as dietary fibre, proteins and (available) carbohydrates. However, with the regular quantification of HDL cholesterol it was noticed that some diets with high polyunsaturated fatty acid (PUFA) content (for example, linoleic acid) lowered HDL cholesterol somewhat (which would be undesirable) along with the desired lowering of LDL cholesterol.

In one meta-analysis — a way of assessing separatly undertaken clinical trials together — a PUFA: saturated fatty acid (SFA) ratio (P:S) under 1.5 was associated with no change of HDL cholesterol in 20 trials, a decrease in 6 and an increase in 4. Only when the P:S ratio exceeded 1.5 did HDL cholesterol usually fall. Official guidelines do not aim for a P:S above 1.5, so recommended diets should have little or no effect on HDL. High intakes of monounsaturated fatty acids (MUFA) have less tendency to lower HDL cholesterol.

Since 1985 the effect of MUFA, mostly oleic acid, has been reexamined. There have been some reports that MUFA were almost as effective in reducing plasma total cholesterol and LDL cholesterol as PUFA in the form of linoleic acid. When MUFA replaces the same amount of SFA, the total cholesterol level is somewhat lower than if carbohydrates replace the SFA. A meta-analysis using the 27 best-controlled clinical trials that compared PUFA and MUFA since 1970 produced a new equation for total cholesterol (see the box on the Keys equations).

These newer trials show slightly less elevation of total cholesterol by SFA, less reduction by PUFA and a very small but not statistically significant lowering effect by MUFA.

Why the difference between the earlier and the more recent results with PUFA? One possible reason is that the habitual diet in Western countries now contains twice the linoleic acid that it did in the 1950s and 1960s. (For example, this can be seen in a doubling of the linoleic acid concentration in samples taken from body fat in the United States from around 8% to 16%.) The effects of both MUFA and PUFA may be influenced by habitual PUFA intakes.

An issue gaining attention in recent years is the possible effect of linoleic acid on the risk of cancer. In some animal experiments, linoleic acid has been shown to promote the growth of chemically induced tumours. Not all animal studies have confirmed this finding, however, and some have even shown that increasing linoleic acid may inhibit induced tumors. The relevance of animal experiments, however, is uncertain, and epidemiologic studies do not support a link between PUFA intake and increased cancer risk. Nevertheless, these results have influenced opinion on PUFA, so that recent dietary recommendations place less emphasis on an increase in PUFA. Rather, the public is advised to maintain PUFA intakes at 6-8% of dietary energy. An intake of up to 10% of energy is still advised by some as a population average; by others it is accepted as a limit for individuals.

Other polyunsaturated fatty acids: Fish oil and n-3 fatty acids

Recent years have witnessed great interest in the potential health effects of fatty fish or (extracted) fish oil. This research was stimulated by epidemiologic reports that Greenland Eskimos have a low incidence of CHD

Keys equations

The pioneering work of the research teams led by Keys and Hegsted were summarized in equations incorporating their findings that saturated fatty acids were twice as effective in raising plasma cholesterol as polyunsaturated fatty acids were in reducing it.

The original Keys equation (1957) was:

Change in plasma cholesterol (mg / dl) = 2.7 x (change in Sat) - 1.3 x (change in Poly),

where Sat and Poly are the percentages of total energy supplied by saturated and polyunsaturated fatty acids, respectively. At first, Keys considered that dietary cholesterol had no effect on plasma cholesterol and that all saturated fatty acids were equally cholesterolemic. In a later equation (1965), it was recognized that stearic acid (C18:0) was a "neutral" saturate, and an additional term for dietary cholesterol was included:

Change in plasma cholesterol = 2.4 x (change in Sat') - 1.2 x (change in Poly) + 1.5 x (change in DC),

where Sat' includes only three saturates - C12:0, C14:0 and C16:0 - and DC is the change in the square root of dietary cholesterol (as mg / 4.28 MJ). Monounsaturated fatty acids were considered neutral.

A 1992 meta-analysis of 27 well-controlled trials by Mensink and Katan also suggests that the cholesterol-lowering effect of monounsaturated fatty acids, if any, is very small:

Change in plasma cholesterol (mg / dl) = 1.5 x (change in Sat') - 0.12 x (change in Mono) - 0.6 (change in Poly),

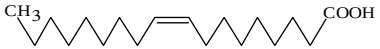
Where, Mono is the percentage of total energy supplied by monounsaturated fatty acids and Sat' again includes only 12:0, 14:0 and 16:0 saturated fatty acids.

FIGURE 3.

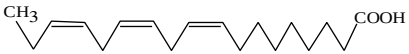
The structure of some fatty acids and their nomenclature

palmitic acid
16 carbon, saturated

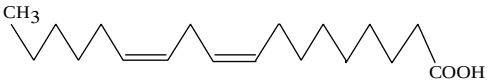
stearic acid
18 carbon, saturated



oleic acid
18 carbon, monounsaturated

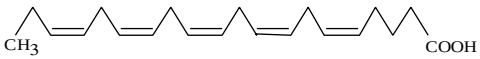


linolenic acid
18 carbon, polyunsaturated
(n–3)

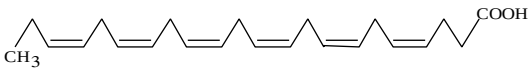


linoleic acid

Polyunsaturated fatty acids are often classified into 'families' depending on the position of the first double bond counting back from the methyl end of the molecule. For example, linoleic acid is referred to as an n–6 fatty acid.



eicosapentaenoic acid
20 carbon, polyunsaturated
(n–3)



docosahexaenoic acid
22 carbon, polyunsaturated
(n–3)

Source: Bender AE, The Evidence for a Relationship Between Diet and Some Major Diseases in Europe (October 1989 Symposium). Brussels: ILSI Europe, 1994

despite a diet rich in fat and cholesterol. It was hypothesized that their very high consumption of the n-3 fatty acids of marine origin (also termed ω-3 or omega-3 fatty acids) may be protective against CHD, particularly eicosapentaenoic acid (20:5 n-3), which is the precursor of a series of prostaglandins, the 3-series, different from the more abundant 2-series derived from arachidonic acid (20:4 n-6) (see Figure 3).

The prostaglandins derived from eicosapentaenoic acid have been shown not to promote thrombosis. For example, regular consumption of even quite modest quantities of fish in Zutphen in The Netherlands has been associated epidemiologically with lower indices of CHD. It is not yet settled whether this is due to the n-3 fatty acids in fish or to some other effect of fish.

Fish oils reduce plasma triacylglycerols (triglycerides) considerably by reducing synthesis of VLDL (very low density lipoprotein). They tend to raise HDLcholesterol but have little effect on LDL cholesterol or total cholesterol; indeed, high doses of fish oil may actually raise LDLand total cholesterol. Because of this, it seems prudent to recommend the consumption of fish oil only under medical supervision to people with very high plasma triaclylglycerol levels (hypertriglyceridaemia). General dietary recommendations encourage healthy people to eat more fish (especially fatty fish).

Because of increased intakes of linoleic acid (an n-6 PUFA) in the last 30 years, the ratio of n-3 to n-6 fatty acids in Western diets has fallen to about 1:20 or even 1:30. Experimental evidence indicates that these two PUFA series compete for metabolism into prostaglandins and other biologically active products. Both linoleic (an n-6) and α-linolenic (an n-3) acid are essential fatty acids. The n-3 PUFA are obtainable from plants as α-linolenic acid or from animal sources, particularly fish, as eicosapentaenoic and docosahexaenoic acids (Table 2). Ratios of total n-3 to n-6 PUFA are about 1:7 in human milk, and there are theoretical grounds for aiming for similar ratios in the general diet. This is especially important for infant formulas because docosahexaenoic acid (22:6 n-3) is one of the principal fatty acids in brain lipids.

Individual saturated fatty acids

SFA, though often considered as a group in public health advice, do not all have the same effect on plasma cholesterol. SFA with fewer than 12 carbon atoms are thought to have no significant effect. Very long chain SFA with 20 carbons or more are not abundant in our diet and are thought to be poorly absorbed and to have no appreciable cholesterol-raising effect. The SFA referred to in the cardiac health context are those with chain lengths of 12-18, i.e., lauric, myristic, palmitic and stearic acids (Table 2). In the 1970s Keys and Hegsted reported that stearic acid (18:0) has little or no cholesterol-raising effect. This was confirmed in the late 1980s. Possible thrombotic effects of stearic acid have been suggested, but firm evidence is lacking. Thus it seems that the other three acids — lauric, myristic and palmitic — are largely involved in raising LDL cholesterol levels. It has been difficult to separate their effects because they appear together in fats and oils and there is no natural oil that contains more myristic (14:0) than palmitic (16:0) or lauric (12:0) acid. Of the three, myristic acid, though seldom present in high proportions in food, appears to be the most actively hypercholesterolaemic, with lauric acid being moderately active. Results with palmitic acid have varied, but well-controlled dietary studies show that the cholesterol-raising effect of palmitic acid is somewhere between those of myristic and lauric acid (Figure 4).

Trans fatty acids

Unsaturated fatty acids can be in the cis (bent) or trans (straight) form; those in food and body tissues are

TABLE 2

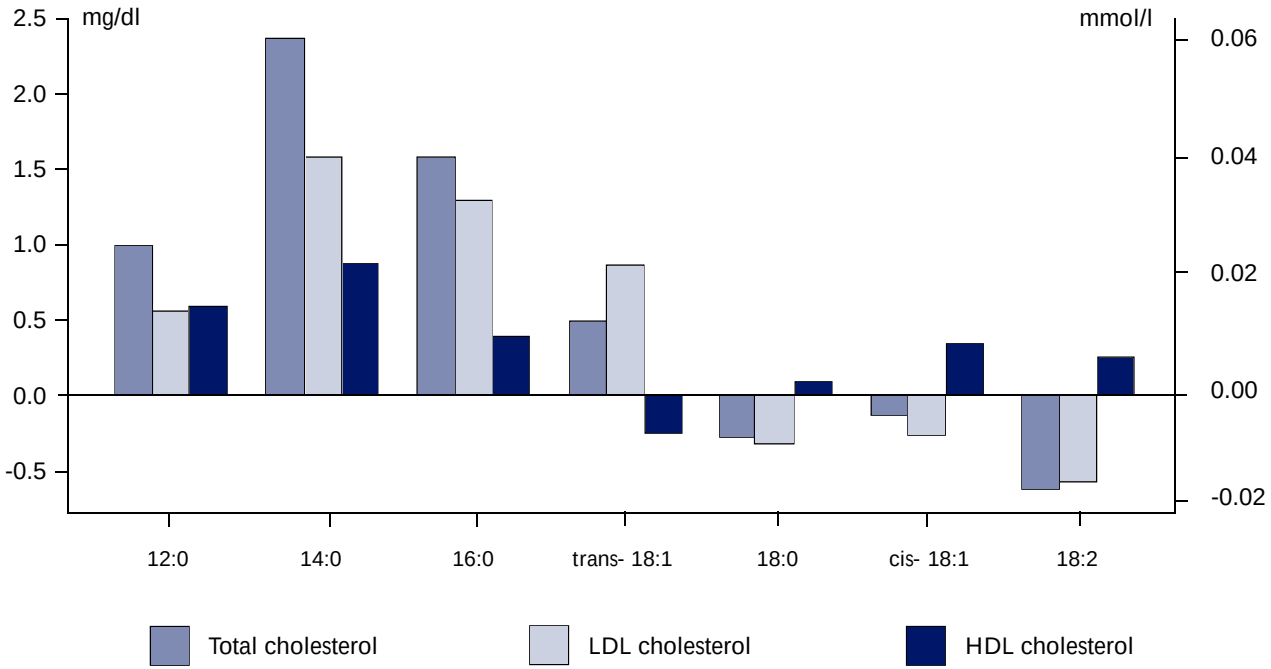
The major fatty acids

Symbol [a]	Common name	Typical fat source
Saturated fatty acids		
2:0	Acetic	Vinegar
4:0	Butyric	Butterfat
8:0	Caprylic	Palm kernel oil
10:0	Capric	Coconut oil
12:0	Lauric	Coconut oil
14:0	Myristic	Butterfat, coconut oil
16:0	Palmitic	Most fats and oils
18:0	Stearic	Most fats and oils
20:0	Arachidic	Lard, peanut oil
22:0	Behenic	"Caprenin"
Unsaturated fatty acids		
16:1 n-7	Palmitoleic	Fish oils
18:1 n-9 (cis)	Oleic	Most fats and oils
18:1 n-9 (trans)	Elaidic	Hydrogenated vegetable oils, butterfat, beef fat
18:2 n-6	Linoleic acid	Most vegetable oils
18:3 n-3	α-Linolenic	Soybean, canola oils
20:1 n-11	Gadoleic	Fish oils
20:3 n-9	Eicosatrienoic	Essential fatty acid-deficient animals
20:3 n-6	Dihomo-gamma-linolenic	
20:4 n-6	Arachidonic	Lard
20:5 n-3	Eicosapentaenoic	Fish oils
22:1 n-9	Erucic	Rapeseed oil
22:6 n-3	Docosahexaenoic	Fish oils

[a] The figure before the colon indicates the number of carbon atoms which the fatty acid molecule contains, and the figure after the colon indicates the total number of double bonds. The n- designation gives the position of the first double bond counting from the methyl end of the molecule. See Figure 3 for the chemical structure of some fatty acids.

FIGURE 4.

Effects of individual fatty acids when they replace 1% of energy as carbohydrates



Source: Zock PL, Katan MB, Impact of Myristic Acid Versus Palmitic Acid on Serum Lipid and Lipoprotein Levels in Healthy Women and Men. Arteriosclerosis and Thrombosis 14, (1994):567-575

overwhelmingly cis. Trans fatty acids are formed in small amounts in the rumen of cows and sheep and so can appear in red meat and milk. They can be produced in larger proportions during the manufacturing of margarine and shortenings when unsaturated oils are hydrogenated. The most abundant trans fatty acid in foods is elaidic acid (trans 18:1 n-9), whose cis counterpart is oleic acid (Table 2).

Our information about the effects of trans fatty acids comes from a number of earlier conflicting reports and several recent dietary trials in which the trans fatty acid given was well defined and LDL cholesterol and HDL cholesterol were separated. It now appears that at relatively high intakes, exchange of trans 18:1 for the usual cis 18:1 (oleic) fatty acid leads to a significant increase of LDL cholesterol and a small decrease of HDL. Based on these and subsequent trials including trials employing realistic intake levels, it is now generally accepted (see, for example, The Report of the British Nutrition Foundation on Trans Fatty Acids, 1995) that trans fatty acids should be grouped with saturated fats from the cardiac health point of view. The effect of longer-chain trans fatty acids (20 and 22 carbons), as

present in partially hydrogenated fish oil, has not been well studied.

An important question is whether relatively high intakes of trans fatty acids actually promote CHD. In the absence of controlled intervention trials, one must rely on epidemiologic observations for an answer. In a cohort of 85 000 nurses, consumption of trans fatty acids was an independent predictor of CHD. Some case-control studies also showed higher intake of trans fatty acids or margarine in patients with CHD. However, two recent studies did not find a higher consumption of trans fatty acids, as measured by their content in body fat, in CHD cases than in control subjects. Thus, a causal link between trans fatty acid intake and CHD is not established. Nevertheless, until more evidence becomes available, it seems prudent to consider high intakes of trans fatty acids as a potential CHD risk factor. At the same time, it must be kept in mind that intakes of saturated fatty acids are much higher than those of trans fatty acids, and reduction of saturated fat intake remains a primary dietary goal in the prevention of CHD.

Thrombosis and reduced blood flow

The effect on blood cholesterol is the best-known way that dietary fats may predispose to atherosclerosis and hence CHD, but there may be other mechanisms. The early fall of CHD mortality with food rationing in the Second World War was too rapid to result from reduced atheroma formation and was probably due to a reduced tendency to thrombosis. There is evidence that dietary fatty acids have different effects on the tendency to thrombosis, most probably through changes in the lipids of the membranes of platelets and hence changes in platelet function. Early studies suggested that SFA increase the tendency to thrombosis. The tendency to thrombosis is reduced by n-6 PUFA, but n-3 PUFA do this at lower intakes (most diets contain smaller amounts of n-3 than n-6 PUFA). Fish oils prolong bleeding time, and their 20:5 fatty acid forms prostaglandins that are not thrombogenic. It is of potential importance to remember that our understanding of the effect of different fatty acids on the tendency to thrombosis in humans comes from indirect evidence: epidemiology, animal experiments and in vitro tests.

Oxidation of low-density lipoprotein

The early lesions of atherosclerosis are characterized by accumulation of fat-laden cells, macrophages just under the inner lining (endothelium) of an artery (see Figure 1). These cells, called foam cells, derive from one of the series of white blood cells, the monocytes, and the lipid comes from the LDL of circulating blood. It has been found in in vitro model experiments that monocytes and macrophages take up very little LDL unless it has been chemically modified by oxidation. The oxidation of LDLnear or in the artery makes it more atherogenic. The oxidation presumably results from free radicals, but the LDL can be protected by antioxidant substances in or near the artery. Such antioxidants include vitamin E, which is fat-soluble and can dissolve in the LDL, and vitamin C, which is water-soluble and can be carried in the plasma. The World Health Organization — MONICA study of 12 European subpopulations with about the same average plasma cholesterol levels but quite different incidences of CHD found a significant inverse relationship between plasma tocopherol (vitamin E) levels and CHD incidence. However, the epidemiologic data on vitamin E and CHD are not consistent. Some but not all follow-up studies suggest that vitamin E might protect against CHD, although a large multicentre European case-control study did not find an association between vitamin E in body fat (a marker for long-term intake) and CHD. In several animal models administration of vitamin E or some other antioxidant attenuated

experimental atherosclerosis. In tests in vitro, diets very rich in PUFA produced LDL that was more susceptible to oxidation than did diets rich in MUFA. The relevance of these experiments to the whole-body situation is not clear because they involved only plasma LDL (not arterial tissue) and high doses of single test substances. Furthermore, animal experiments and epidemiologic studies show a lower risk of CHD when PUFA are substituted for (part of) the SFA to lower blood cholesterol.

It is important to note that most vegetable oils rich in PUFA naturally contain relatively high levels of vitamin E. The suggestion has been made that generous intakes of vitamin E above official nutrient recommendations may protect against lipid oxidation and hence lower the risk of CHD. Whether vitamin E protects against CHD should become clear when several current prevention trials with vitamin E supplements are completed.

Dietary fatty acids and cardiac arrhythmias

Yet another way in which different fatty acids could affect the risk of CHD has been investigated by workers in Adelaide, Australia. In rats and marmosets it has been found that the incidence of dangerous heart rhythm disturbance after tying and releasing a coronary artery was lower in animals that had previously been fed for a long time high-PUFA diets rich in fish oil or linoleic acid than in animals fed saturated fat or olive oil diets. These experiments may be relevant to sudden cardiac death.

INFLUENCE OF FAT ON THE RISK OF CANCER

Early animal experiments

From the late 1930s to the 1950s, Tannenbaum, a Chicago pathologist, carried out hundreds of experiments on mice with chemically induced or spontaneous tumours and compared the effects of different diets on the progress of the tumours. He found that the higher the fat intake, the higher the proportion of mice that developed tumours. He designed experiments to study the effect of fat intake keeping energy intake constant, and the effects of energy intake keeping fat intake constant. These are not easy experiments because fat is such a rich source of energy.

Tannenbaum came to the conclusion early in the 1950s that energy was a more powerful promoter of rodents' tumours than fat. At a given level of energy restriction, however, a high-fat diet permitted the formation of more tumours than its low-fat counterpart. This work is difficult to interpret because other changes in the diet are inevitable when fat and energy content are widely varied.

Epidemiologists enter the field

After this there was little research interest in diet and cancer until the early 1970s, when Doll and coworkers in Oxford published data for mortality from different types of cancer around the world and noted associated dietary variations. This has stimulated a great deal of epidemiologic and other research on dietary factors in the aetiology of cancer. Table 3 comes from a 1981 book by Doll and Peto.

Among their important conclusions were:

- Habitual diet is probably the most important controllable complex contributing in the aetiology of all cancers.

TABLE 3

Proportion of cancer deaths attributed to avoidable factors

Factor or class of factors	Percentage of all cancer deaths	
	Best estimates	Range of acceptable estimates
Tobacco	30	25-40
Alcohol	3	2-4
Diet	35	10-70
Food additives	<1	-5[a]-2
Reproductive and sexual behaviour	7	1-13
Occupation	4	2-8
Pollution	2	<1-5
Industrial products	<1	<1-2
Medicines and medicinal procedures	1	0.5-3
Geophysical factors	3	2-4
Infection	10	1-?

[a] This value allows for a possible protective effect of antioxidants and other preservatives.

Source: Doll R., Peto R., The Causes of Cancer - Quantitative Estimates of Avoidable Risks in the United States Today. Oxford: Oxford University Press, 1981; also published in The Journal of the National Cancer Institute (1981):66; 1191-1308

- Dietary fat appears to be an important contributor to cancers of the breast and of the large intestine.
- Food additives are not major causes of cancer.
- ß-Carotene, a common lipid pigment in foods, may be a protective agent against some cancers.

Doll and Peto's graphs comparing estimates of average national intakes of a food component (for example, fat) against age-standardized national death rates from a particular cancer were criticized on the grounds that correlations could also be demonstrated with numbers of cars or television sets (i.e., indices of economic development). However, low rates of colorectal and breast cancer in Japan suggest that the rates correlate more consistently with fat intake than with economic development.

DIET AND CANCER OF THE COLON AND RECTUM

In the 1970s a number of case-control studies of people with recently diagnosed cancer of the colon and rectum were conducted. In 21 different studies there was a consistent negative association between such cancers and vegetable consumption, even though the consumed vegetables varied. The change in risk was usually considerable, i.e., 60-70%. Dietary fibre is one of the best candidates for the protective factor, but to distinguish fibre from other vegetable components is difficult. Clinical prevention trials are needed in people with precancerous lesions such as polyps in the colon.

Mortality rates for these cancers in various countries are highly correlated with estimated average total fat intakes. For some years the results of case-control studies for total fat intake were unimpressive, but recently eight good new case-control studies in the United States, United Kingdom, Singapore, Australia, China and Majorca implicate total fat or saturated fat or meat. It is difficult to disentangle these three factors because saturated fat intake is usually correlated with meat intake.

Prospective studies give much more reliable information but take longer and cost more. Willett and Hennekens in Boston began a prospective study of 122 000 female nurse volunteers at the end of the 1970s, and of these, 90 000 completed a food frequency dietary questionnaire. After 8 years of follow-up, 150 nurses had developed cancer of the colon. When the original cohort was grouped into quintiles (fifths) of the distribution of estimated animal fat intake, a progressive increase in the proportion of cancer cases was found. In the uppermost quintile of animal fat intake, the relative risk of developing colon cancer was 1.8 (80% higher than in the lowest quintile of original fat intake).

DIET AND BREAST CANCER

Countries with a high mortality from breast cancer also have a high estimated average fat intake, but it does not necessarily follow that fat causes breast cancer. The results from case-control studies of diet and breast cancer are much less clear than those for cancer of the colon and rectum. Some studies found an increased risk of breast cancer with higher intakes of milk, dairy products and total fat, but other studies did not. A few indicated that fruit and vegetable intakes were protective, possibly owing to their vitamin C content.

Until the results of the large prospective study of United Staes nurses became available, it had been generally assumed that a high fat intake was a risk factor for breast cancer. But there are a number of discordant findings. Plasma cholesterol levels (a general indicator of fat intake) do not predict breast cancer. It takes more than a single generation for women migrating from a low-incidence country to acquire the breast cancer rates of a high-incidence host country. Tallness and obesity are both associated with breast cancer. The social gradients of breast cancer and CHD are different. Associations are different for the less common premenopausal breast cancer than the more common postmenopausal breast cancer.

As the results of the United States nurses study accumulate, they show an impressive association between animal fat intakes and colon cancer (150 cases). However, they show no association between breast cancer and fat intake despite a larger number of cases (1 500 reported up to 1992). Two smaller studies in the United States have also failed to show an association with fat intake.

There are several possible explanations for these inconsistent findings. One is that any effect of diet on breast cancer is exerted in early life, before menarche.

Another is that there is a threshold fat intake (between 20% and 30% of total energy) above which any increase in fat intake makes no difference. Perhaps not enough United States nurses were eating a really low fat diet below the threshold. A case control study from a more heterogeneous population in northern Italy tends to support this idea. Women consuming less than 28% of their total energy as fat (and less than 9% as saturated fat) had only 50% of the average risk of breast cancer. As intakes rose above 36% of energy from fat, there was no further increase in risk. In this study, animal protein and fat intake were strongly correlated. Because it was not possible to disentangle their possible effects, no firm conclusions can be drawn.

Progress in this research will probably come from large prospective studies combined with laboratory research. A prospective study has been undertaken in seven countries in Europe which will have a total of 400 000 middle-aged men and women participants. In addition to questionnaire data, blood samples will be taken and stored in liquid nitrogen for subsequent measurement of biochemical markers and even DNA analysis. For all subjects there will be a dietary questionnaire based on usual consumption during the preceding year, and a 24-hour recall will be collected from a subsample of 5% of the whole cohort for calibration adjustment between countries.

Conclusions

Cancers of the colon and rectum and the breast are the most likely of the common cancers to be associated in some way with fat intake, although the evidence for breast cancer is less convincing. In general, the rates of these cancers and of CHD follow similar trends in European countries. At present, therefore, provisional broad dietary advice for reducing the risk of each of these conditions is the same: Eat less fat (particularly animal fat), avoid overweight, and eat more vegetables, fruits and cereals.

FAT AND SATIETY

In affluent societies in Europe, North America and Australasia more adults are overweight than underweight. Overweight is the result of an imbalance between energy intake (eating) and energy expenditure (physiologic maintenance of the body, physical activity). Even very small differences between intake and expenditure can result in large changes in body weight over time: the chronic consumption of only a few excess kilocalories per day leads to an increase in body weight of several kilograms over a decade. Whatever forces are at work, whether genetic or environmental, people find it easier to become overweight than underweight. Nearly all overweight people are in this state involuntarily; something is happening to them passively. This suggests that whatever the mechanisms of appetite are, there is weaker control of overconsumption than under-consumption. In a number of affluent countries the percentage of overweight adults has increased in recent years despite intense interest in diet and weight control.

It is known that if food intake is decreased for whatever reason, physiologic and metabolic signals will inform the brain of the diminished nutrient supply and there will be an increase in biological drive to compensate. Any energy/calorie deficit produces a very strong defence of the nutritional state: it is difficult to undereat. We know from clinical experience that only about 5-10% of obese people ever achieve long-term control of their body weight.

If energy intake is increased, however, there is little if any evidence for a compensatory decrease in the biological drive to eat. The system appears to work asymmetrically, with a very strong defence against undereating but very weak (if any) defence against overeating. In the evolutionary context, we can visualize that there were no biological penalties in survival terms when our hunter-gatherer ancestors overate.

Over the course of the last 100 or so years, the intake of (total) protein and its proportion in the diet have remained more or less constant in Western countries. However, there have been large reciprocal changes in the amounts of carbohydrates and fats. At the turn of the 20th century people were consuming about 70-75% of their energy as (total) carbohydrates and only 14-20% energy as fats. Nowadays many people are eating equal amounts of energy from carbohydrates and from fats.

It is generally found that dietary protein has a potent and enduring effect on satiety, probably through both preabsorptive and postabsorptive actions. It is very difficult to overeat protein. The important question is whether there are differences in the satiating effect of equal amounts of energy derived from carbohydrate and fat.

If a food or meal contains more or less fat in place of carbohydrate, how will this affect satiation and, indirectly, obesity? There are several ways of comparing the satiety produced by fat and carbohydrate.

Medium-term study

In one 2-week study by Lissner, 24 women were allowed limited ranges of food. They were given low-fat, high-carbohydrate foods, or medium-fat, medium-carbohydrate foods, or high-fat, low-carbohydrate foods. In all three groups of foods, the protein content was the same. Those on the medium-fat, medium-carbohydrate diet consumed an average of 9.62 MJ (2 300 kcal) daily, whereas those on the low-fat, high-carbohydrate diet ate 11% less energy. However, those on the high-fat, low-carbohydrate diet overate and there was no sign of adaptation or compensatory reduction of food intake over the period of the study.

Short-term studies

In a trial in a specially adapted laboratory, 24 adults spent a day with minimally distracting stimuli and ate food from plates that were continuously weighed. Three breakfasts were given: a basal meal of 1.84 MJ (440 kcal), the basal meal plus 1.67 MJ (400 kcal) as carbohydrate, or the basal meal plus 1.67 MJ as fat. Hunger was monitored for the next 4 hours, and was found to return more slowly after the high-carbohydrate breakfast than after the others. Moreover, when a snack was offered 2 hours after breakfast, subjects ate smaller amounts after the high-carbohydrate breakfast but the intake was no lower after the high-fat breakfast than after the basal, lower-energy meal. However, if the next meal was offered after 4.5 hours (i.e., lunch), there was no significant difference in intake between the three groups.

In a subsequent trial, food intake was measured for 24 hours after these three breakfasts. Subjects who had the high-fat breakfast consumed significantly more energy over the course of 24 hours, but those given the high-carbohydrate breakfast ate no more than those given the basal, lower-energy meal.

From these and other carefully controlled experiments, it seems that carbohydrate has a strong satiating effect, which probably varies with the type of carbohydrate. But fat has only a weak effect on satiety; appetite and subsequent energy intake are not suppressed after extra fat consumption. It is possible that fat weakens satiation so that overconsumption of energy is likely on high-fat diets.

Utilization of different meals

It also appears that for equivalent energy intakes, high-fat diets are less efficiently oxidized as fuel than high-carbohydrate diets. After a high-carbohydrate meal, carbohydrate oxidation increases during the next 24 hours, whereas adding fat to a meal does not increase fat oxidation. Although it is possible for dietary protein, carbohydrate and alcohol to be converted to fat in the body, it does not normally happen when people eat mixed diets that include fat. It follows that body fat is largely derived from fat in food.

NUTRITIONAL IMPLICATIONS OF FAT SUBSTITUTES

The concept of replacing some or all of the fats in foods with nutritionally inert substitutes has received considerable attention from the food industry, nutritionists and dietitians, the news media and consumer organizations. Development of these materials (Table 4) is largely aimed at the objective of reducing people's excessive fat and energy consumption. Yet, although the safety and uses in foods have been scrutinized by industry scientists and official food safety committees, little research has been done on the nutritional implications of fat substitutes (Table 5).

Carbohydrate-based and protein-based fat substitutes

These present few toxicological concerns, being biologically well understood, but they have limited uses in foods. They are not well suited to baking and cannot be used as frying media or for the development of lipid-associated flavours. They are suitable for such foods as ice cream and cold sauces and dressings. Most of the carbohydrate- and protein-based materials are partly or fully digested and absorbed. They can reduce fat intake by replacement and may reduce energy intake because their energy density is only about half that of fats.

Nonabsorbable lipids

These products have functional and sensory properties in foods similar to the traditional fat ingredients they are intended to replace. Their potential applicability is much wider than that of the first group and includes most areas where processed fats and oils are used at present. But toxicological uncertainties about these compounds have limited their approval and use in foods up to now. Only one, sucrose polyester, has

TABLE 4

Examples of current and proposed ingredients for fat substitutes

Carbohydrate-based and protein-based materials

Modified glucose polymers
Modified tapioca, corn, potato and rice starches
Gums and algins
Cellulose derivatives
Microparticulated proteins

Nonabsorbable Lipids

Fatty acid esters of sugars and sugar alcohols
Polycarboxylic acid and propoxylated glyceryl esters
Alkylglyceryl ethers
Substituted siloxane polymers
Branched (sterically hindered) triacylglycerol esters
Specific naturally occurring lipids

Polyglycerol esters

TABLE 5

Potential advantages and disadvantages of fat substitutes

Possible advantages

Reduced total fat intake
Reduced intake of cholesterol
Reduced absorption of cholesterol
Reduced energy intake
Increase in intake of complex carbohydrate (with some materials)

Possible disadvantages

Reduced essential fatty acid intake
Reduced intake of fat-soluble vitamins
Increased cost
Compensatory energy intake
Changes in intestinal transit (for high intakes of non-absorbable materials)

reached the stage where proposals for food use are being realistically considered. Since these materials pass through the gut more or less unchanged, they function as a slow stream of "solvent" to take up other lipid substances, such as fat-soluble vitamins or drugs, and reduce their absorption. They may hasten intestinal transit and might cause some diarrhoea.

Polyglycerol esters

These form an intermediate group. Their functional properties in foods and metabolism in the body are determined by their actual structure, depending on whether they behave more as carbohydrate or as fats.

How much dietary fat could fat substitutes replace, and at what level could they be used?

The answers to these questions will be a major consideration in assessing potential population intake and implications for nutritional status. The present carbohydrate- and protein-based fat substitutes are generally approved for a limited number of applications: pourable dressings and some dairy-based products. It should be pointed out that local regulations vary: products subject to regulation in the United States are sometimes regarded as foods in Europe and do not

require approval. If lipid-based materials such as sucrose polyester were to be widely approved, this would extend the range of the potential uses of fat substitutes to margarines and domestic and commercial cooking oils. Nonetheless, there would still be a major proportion of dietary fat not replaced: that contributed by meat, fish, cheese and eggs and by minimally processed cereal products (which, although low in fat content, make a sizeable contribution to total intake because of the large amount consumed). Consumption of some specific food products could change as consumers become aware of lower fat contents.

Possible implications for requirements of essential fatty acids and fat- soluble vitamins

The adult requirement for n-6 fatty acids (see Table 2 and Figure 3) such as linoleic acid is thought to be 2-7 g/day (1-3% of total energy intake). That for n-3 fatty acids has been estimated as 0.5% of total energy in the form of eicosapentaenoic plus docosahexaenoic acids (0.3-0.4 g/day) and α-linolenic acid (0.8-1.1 g/day). However, it has been suggested that an intake of n-3 fatty acids above requirements may be beneficial.

Essential fatty acid deficiency is almost unknown in otherwise healthy people. Essential fatty acids are contained in foods such as meat, fish, cereals and other plant foods that are unlikely to be replaced through the use of fat substitutes. Extensive replacement of edible oils by lipid-based substitutes could conceivably lead to the loss of unsaturated fatty acids including linoleic acid. If a particular subgroup of the population were to become heavy users of fat substitutes, for example, adolescent girls, it should be possible to incorporate linoleic acid in the fat replacer.

Of the fat-soluble vitamins, vitamin D is derived largely through the action of sunlight on the skin. The requirements for vitamin K are small; it is widely available in leafy vegetables and partly supplied by synthesis in the intestine. Only vitamin A (and the carotenoids) and vitamin E are of concern. Decreased plasma concentrations of these have been observed in several reported studies with sucrose polyester, but it is not certain whether this reflects reduced labile pools or is due to decreased levels of plasma lipoproteins (including cholesterol) or take-up in "solvent".

Effects on plasma lipids

Consumption of nonabsorbable lipids may reduce the intestinal absorption of cholesterol, both that from the diet and that in the enterohepatic cycle (cholesterol is excreted in the bile and then partly reabsorbed). A number of studies have shown that sucrose polyester can reduce plasma total cholesterol, LDLcholesterol and triglycerides without lowering the level of the beneficial HDL cholesterol. LDL cholesterol, can fall by 20% or more and faecal neutral steroids can increase significantly, indicating increased excretion of cholesterol. Whether or not lipid-based fat substitutes gain approval and acceptance for use by the general population, foods containing these ingredients could be effective as dietetic products available on medical prescription for people with hyperlipidaemia.

What would likely happen to energy intake?

In two short-term studies, subjects ate on different days a high-fat breakfast or the same breakfast with the fat largely replaced by sucrose polyester. During the rest of the day, the fat replacement group compensated almost completely for the energy deficit of the sucrose polyester breakfast. In a pilot study of food intakes by 32 subjects in their own homes, half were provided with fat-substituted foods and the other half had foods of similar appearance with normal fat. The fat-substituted

group did not consume less energy overall, although their initial intake was lower. They did, however, decrease their fat intake as a percentage of total energy from 38% to 30%.

There is an obvious need for more studies like these. As well as answering the question about energy intake when people are unaware of whether fat has been replaced in foods, there is the equally important question of whether people will modulate their total energy intakes if labels indicate that some of their foods contain low-energy fat substitutes.

Possible impact on cost

Wide introduction of fat substitutes would likely increase costs to consumers, since the costs of development and technology and the small initial market would presumably mean that the ingredients would cost more than traditional fat ingredients. Moreover, it is entirely possible that people eating fat-substituted foods would, either from physiological or psychological causes, eat more of other foods for energy compensation, and thus further increase food costs.

Future needs

The bulk of research on fat substitutes has been derived from proprietary, industrial efforts to establish the function and safety of these ingredients. Greater effort should now be directed toward examining or ensuring their nutritional efficacy, for example, their influence on micronutrient absorption by the general population, and particularly their benefits under conditions of normal consumer use.

RESPONSE OF INDUSTRY TO RECOMMENDED CHANGES IN FAT INTAKE

Dietary fat intake

In the 1960s, people in most Western industrialized countries obtained about 40% of their energy from fat, of which saturated fat contributed around 20% and polyunsaturated fat 4-5% (Table 6). Since then, total fat intake has not changed much, but the proportion of polyunsaturated fat has increased at the expense of saturated fat.

There are many pitfalls in estimating national dietary intakes, and methods vary among countries. Apparent consumption (national food and drink disappearance data) must be distinguished from individual intake data, but the latter can be obtained only from samples of people who (it is hoped) represent the population as a whole. Different procedures are used to estimate individual nutrient intakes — 24-hour recall, food records and food frequency questionnaires — and each procedure has its strengths and weaknesses. Note, too, that total fat appears lower if alcohol is included in the total energy denominator and that saturated fat appears higher if trans fatty acids are included with the saturated fat (Table 6).

Nutrient intake values depend on the composition of the foods eaten, and these may vary markedly from the average values quoted in food composition tables. In one study in The Netherlands, linoleic acid provided 7% of total energy by a 24-hour recall method using food table values but only 4% by the duplicate-plate method (where subjects prepare a duplicate of everything eaten for analysis).

TABLE 6

Contribution of fat intake as a percentage of total energy intake for different countries

	Total fat	Saturated fatty acids	Mono-unsaturated fatty acids	Poly-unsaturated fatty acids
United States[a] (women): USDA Nationwide Food Consumption Survey, 1985	37	13	13	7
United Kingdom: Office of Population Census and Surveys, 1990				
• Including alcohol	38.5	16 (18)[b]	11.5	6
• Excluding alcohol	40.5	17 (19)[b]	12	6
Germany: Ernährungsbericht, 1988	40	15	14	6
Netherlands: WVC National Food Survey, 1988	40	16	15	7
Belgium: National Food Survey, 1988				
• North	42	17	17	9
• South	42	19	17	6
France (males): MONICA Nutritional Survey, 1985-1987	36	15	13	6

[a] New data from the NHANES III study indicate that total fat consumption in the United States has declined over the past decade to 34% of total energy intake.

[b] Values in parentheses include trans fatty acids.

The role of the food industry

The role of the food industry in meeting dietary guidelines is to provide good quality products that help consumers modify their diets to meet these recommendations without disturbing their habitual eating patterns to an unacceptable degree. The response to advice on dietary fat has been to introduce products with reduced fat content and with unsaturated fats replacing at least some saturated fat. The first of these fat-modified products involved the "visible fats", for nutritional, legal and technical reasons.

Working with visible fats it is possible to increase polyunsaturates to around 10% of energy intake, but lowering saturates to 10% or less is a greater challenge, since more than half of saturated fat is "invisible" in other foods (Figure 5). In 1982, for example, Germans consumed 12 kg of saturated fats in visible fats but 14.4 kg in invisible fats.

The next phase clearly is to reduce saturated fat in the invisible part of the diet. Industry is doing this by replacing fat with nonfat ingredients (for example, water or dietary fibre) or saturated fats with unsaturated fats. Gradually, the legal obstacles are disappearing and product quality is improving.

Product quality

Yellow fat spreads. In some countries sales have increased for these good quality reduced-fat (40%) spreads as well as for the more traditional unsaturated margarines.

Cheese. Quality is complicated by the large variety of low-fat cheeses made. Good quality is easier to achieve with vegetable-fat cheese than with low-fat dairy cheese.

Bakery products. These products present technical problems, and in many cases the required functions are incompatible with achieving improved nutritional profiles. For instance, saturated fat still has to be used for puff pastry.

Communicating nutrition information

Health claims. For fats, the major health messages are:

- A diet low in saturated fat and cholesterol is associated with a reduced risk of CHD.
- Diets low in total fat may reduce the risks of some types of cancer and are helpful for body weight control.

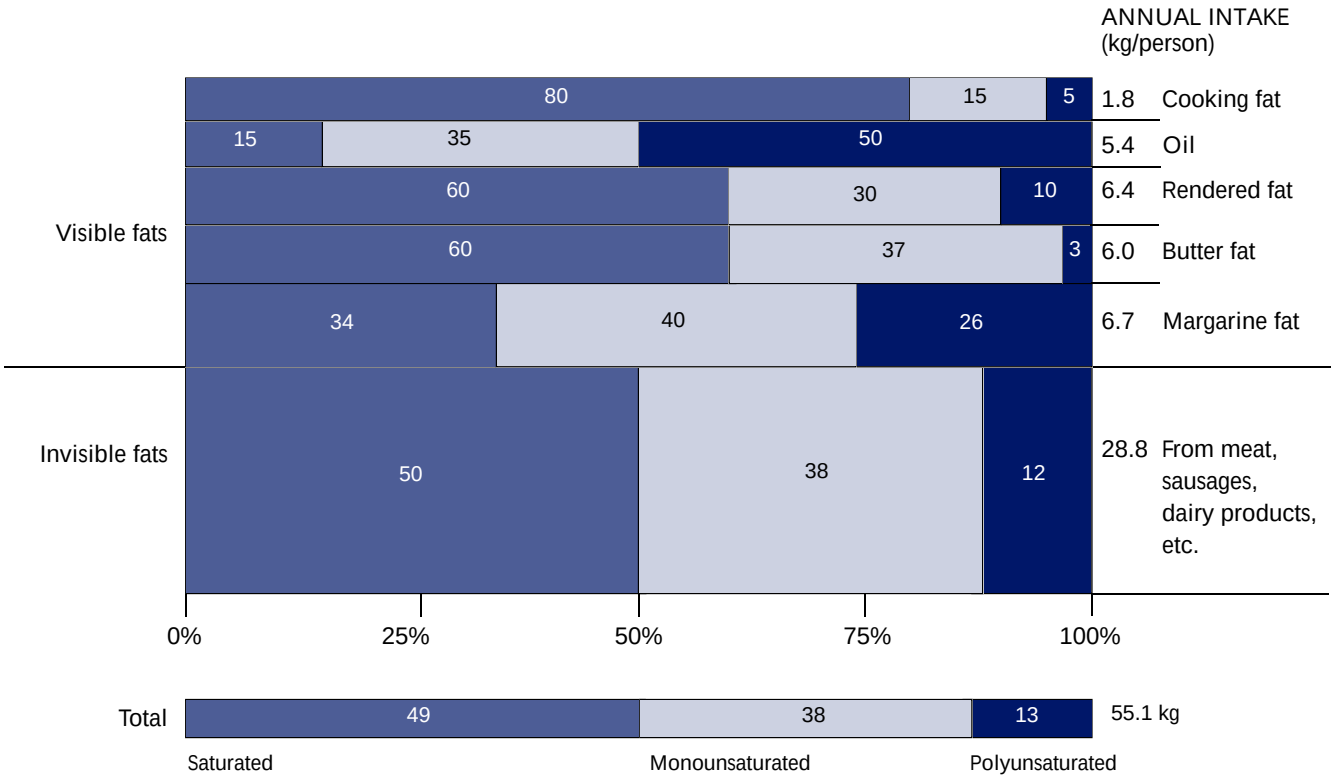
Authorities in the United States have drawn up a list of health claims based on current scientific consensus that will be permitted on food labels. This issue is still under discussion in the European Union. The U.S. Food and Drug Administration has approved carefully worded versions of the two claims above.

New scientific developments. Long after the original work by the research groups of Keys and Hegsted (see above), new information continues to be published on the relative importance of individual fatty acids in influencing plasma cholesterol. It is difficult to communicate details such as the contents of stearic or myristic acid to the average consumer. It may be worthwhile to give the contents of fatty acids considered to be cholesterol raising, cholesterol lowering or cholesterol neutral, and the terms would not need to be altered if scientific consensus changed.

"No cholesterol" claims. These can be misleading. Because the potential health effects of dietary cholesterol are considerably less serious than those of dietary saturated fat, removing cholesterol from a high-

FIGURE 5.

Fat composition in Germany, 1982



Total individual fat intake is 55.1 kg/year, or 151.0 g/day, representing 39% of energy intake. Visible fats are fatty foods that the consumer can recognize as fatty, such as butter, margarine, cream, lard, frying fats, salad oils and peanut butter. Invisible fats are contained in such foods as lean meat, fish, milk, cheese, cakes, biscuits, pastries, snack foods and chocolate. It is difficult for consumers to estimate how much fat they are taking in from these foods.

saturated-fat product tackles the less serious problem while leaving the more serious problem unsolved. Most proposals for such claims therefore require the reduction of both. EU proposals are that for a "no cholesterol" claim a product should contain (per 100 g) less than 3 mg cholesterol, 2 g saturated fatty acids, 10 g total fat and 15% of its energy as total fat.

Consequences of changing fat content and/or composition

When margarine consumption increased during the 1930s, it was realized that it should contain the same levels of vitamins A and D as the butter it was replacing. Because margarines are made from vegetable oils, they are also important sources of vitamin E (Table 7).

For most countries, recommended dietary allowances (RDAs) of vitamin E are around 10 mg/day. If low-fat and very-low-fat products are consumed, vitamin E intakes can fall, so it would be reasonable to double vitamin E contents of fat spreads. This is particularly important as because more vitamin E is needed when the diet is high in polyunsaturated fats. Moreover, an increasing number of scientists are recommending higher intakes of vitamin E than recommended by present RDAs because of research that suggests it may be protective against cancer and/or oxidation of LDL (see above).

Thorough discussions among industrial, academic and regulatory scientists are essential to debate the consequences of product changes such as these and decide on appropriate measures.

TABLE 7

Percentage of vitamin E intakes provided by different food groups in Germany, 1986

Beverages	0
Sugars and confectionery	1
Eggs	4
Nuts, seeds, legumes	1
Milk and other dairy products	3
Meat and other dairy products	8
Fat and oils	39
Fruits and vegetables	32
Grain and products	12

Source: Ernährungsbericht, 1988.

TABLE 8

Potential for reducing fat consumption with low-fat products

	Fat consumption (g/day)	
	Netherlands food consumption survey, 1987-1988	If replaced by available low-fat analogs
Visible fats		
Spreads	19.9	10.0
Butter	4.3	2.0
Kitchen fats	6.6	6.6
Invisible fats		
Sauces	2.6	0
Dairy products	18.7	9.0
Eggs / meat / fish	23.6	20.0
Baked goods	11.0	6.0
Snacks	7.2	7.2
Other	10.7	10.7
Total	104.6	71.5

FAT SUBSTITUTION ON THE BASIS OF FAT FUNCTIONALITY

Consumers are interested in "light", reduced-energy, reduced-fat foods, but it is not easy to replace all of the functions of fat in foods. In the U.S. market in 1990, the proportion of low-fat products as a percentage of all foods in a given category were: spreads 40%, dairy foods 36%, frozen desserts 35% (yogurt 62%, ice cream 3%), processed meats 7%, baked goods 3%; overall 17%.

If full-fat foods, whether they contain visible or invisible fats, could be satisfactorily replaced by low-fat products, an individual's fat intake could be reduced to 70% of its present level (Table 8), achieving the nutritional goal of 30% of total energy from fat without changing food habits.

TABLE 9

Functions of fat in different categories of products

Fat-continuous foods	Water-continuous emulsified foods	Fat in (biopolymer) matrix foods
Flavour carrier	Flavour carrier	Flavour carrier
Structure builder	Structure builder	Structure interruptor
	Creaminess	
Reflectance	Reflectance	
Aeration	Aeration	Aeration
Heat transfer		Heat transfer
Melting		Melting

FUNCTIONS OF FAT IN FOOD PRODUCTS

The technological functions of fat differ in three classes of foods: fat-continuous foods, water-continuous emulsified products and (biopolymer) matrix foods (Table 9).

In fat-continuous foods

Examples are butter and margarine. These foods consist of an emulsion of water in edible oil in which fat crystals separate the aqueous phase (Figure 6). A successful range of low-fat spreads has been developed over the last few years in which fat functionality is maintained.

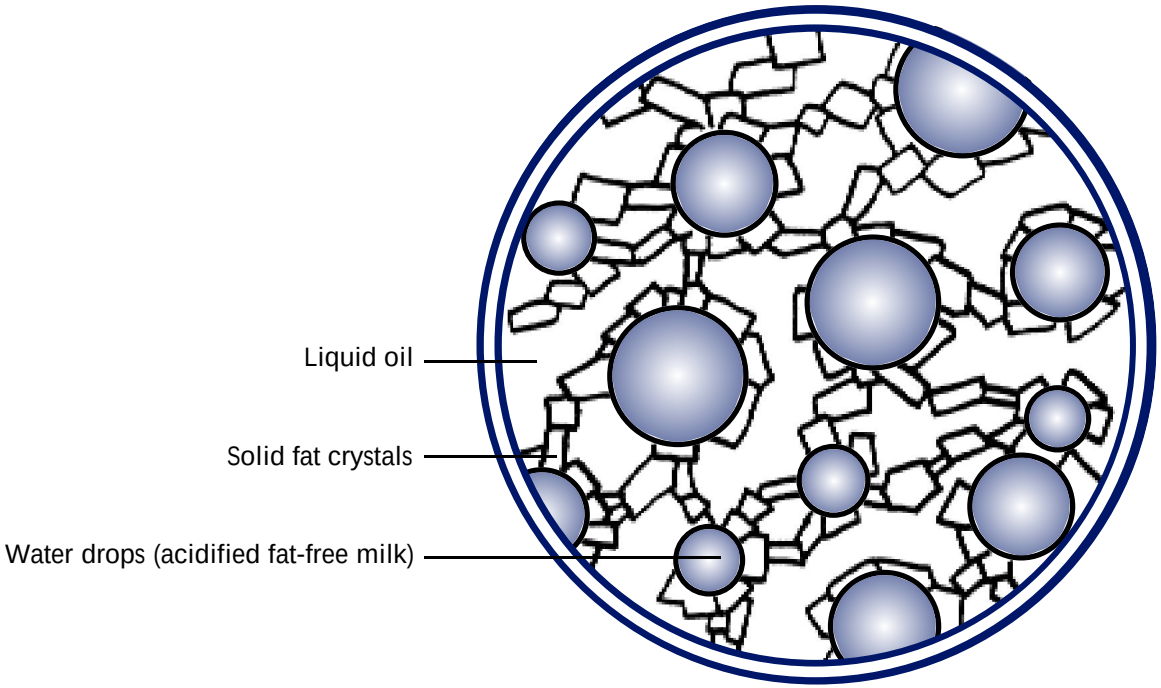
Appearance. Translucency is determined by the depth to which light can penetrate a product. This depends on the presence of fat crystals and their surface structure and refractive index. In spreads, the fat crystals scatter light and make the product opaque.

Texture. Hardness depends on the amount of fat crystals and their shape and interactions (the water phase contributes very little).

Mouthfeel. The proportion of solids in a margarine is determined by temperature. In the mouth (temperature 30-35°C) fat crystals melt, the fat phase softens and the water droplets can coalesce with saliva. This gives the good mouthfeel of full-fat spreads and butter.

Flavour. Fat acts as a source of flavour and as a reservoir for fat-soluble flavour compounds. Flavour perception is determined by the ratio of the flavour compound in the gas above the product and in the product itself, which in turn is determined by the volatility of the flavour compound. Most flavours are more soluble in fat than in water, so any reduction of the proportion of fat will change flavour release and perception.

FIGURE 6.
Structure of a typical fat-continuous food such as butter or margarine



Heat transfer. This is a very important function of pure oils.

In water-continuous emulsified products

Typical examples of these products are milk, dressings, soups and creams. Here the fat is present as dispersed fat or oil droplets.

Appearance. Light scattering gives an opaque, white appearance.

Texture. Mayonnaise generally has a fat level of 70-80% with the oil drops tightly packed together. Only at very high fat levels is hardness influenced by the fat phase. Reduction of fat and / or its replacement by hydrocolloid thickeners alters the viscoelastic properties.

For whipping products such as creams, fat is important to stabilize the air. Air bubbles in whipped cream are surrounded by flocculated fat droplets that maintain the air bubble and give consistency to the cream.

Mouthfeel. High-fat products are perceived as more "creamy" than low-fat products, and creaminess is associated with high quality and the feeling of fullness. Creaminess is correlated with the flow behaviour of materials.

Flavour. Fat influences flavor in a manner similar to that for fat-continuous products.

In (biopolymer) matrix foods

In these products the surrounding matrix of structured biopolymer material, either protein or carbohydrate, plays a dominant role in determining the products' properties. Examples include bakery products, meat, cheese, some snack foods and even frozen desserts such as ice cream. The functions differ with product type.

In baked goods fat plays important roles in preparation and during baking. In cakes, for example, fat determines the volume by stabilizing the air in the batter.

In meat fat affects texture, taste and to some extent tenderness. Fat also enhances flavour in several ways.

In cheese fat is present in small globules. If fat is removed, the cheese becomes more elastic and firmer. Fat also contains flavour components.

In ice cream fat plays an important role in aeration of the product.

ROUTES FOR FAT REPLACEMENT

There are three major approaches for replacing fat in the diet. First, emulsification techniques can be used in low-fat spreads. Second, biopolymer ingredients can be used, and this approach is the most common. Third, fat can be replaced by synthetic fatlike materials.

Fatlike molecules with emulsifier activity

In fat-continuous spreads the fat can be considerably (but not completely) reduced using emulsification technology. Emulsifiers can also help stabilize the air cells in ice cream. Flavour and mouthfeel are retained by the residual fat. Emulsifiers display their fatlike properties while "hiding" the hydrophilic properties of the reduced-fat food.

Biopolymer-based fat mimetics in water

Many products now marketed as "fat replacers" were formerly sold with the more realistic and modest claim of "thickener". These substances contribute energy, though not fat. Because they are not "fatlike" they cannot replace all functions of fat in foods. Carbohydrate or protein polymers cannot replace frying oil because they will not melt and are likely to denature and burn. Carbohydrate and protein biopolymers cannot provide the same flavour profile as fat.

In products such as low-fat dressings, fat is replaced by water in combination with a thickener (giving a liquid product) or gelling agent (giving an elastic product). But some fat is still required to maintain the plastic characteristics of a spread.

Meat products with less fat can be perceived as dry after preparation. Hydrocolloids such as carrageenan are therefore added to increase juiciness.

Mouthfeel is difficult to mimic. In margarines, fat melts at mouth temperature, but very few polymers exist that melt at similar temperatures. Gelatin can melt at this temperature, but unlike fat it does so quickly and cannot produce the same plastic rheology.

Fatlike materials as fat substitutes

Sucrose polyesters are the best-known example of these materials. In sucrose polyesters, the hydroxyl groups of sucrose are replaced with fatty acids in the same way that the hydroxyl groups of glycerol are replaced with fatty acids in natural fats and oils, the triacylglycerols (also called triglycerides). Sucrose polyesters resemble fats in their physical properties but cannot be digested or absorbed, and consequently do not contribute energy to the diet. Because they do not occur in nature and are synthetic, regulatory clearance is needed before they can be used in foods.

Other synthetic products have been proposed. One has a glycerol-like backbone to which fatty acids are linked in such a way that they are poorly digestible, but these compounds have not been approved for use in foods either.

Another suggested way to reduce the available energy in foods that contain fat is to use products such as synthetic triacylglycerols, described in detail below.

FAT REPLACERS

Ingredients to replace some or all of the fat in foods fall into two categories: fat substitutes and fat mimetics.

Fat substitutes resemble fats chemically, have similar physical and thermal properties, and can replace an equal amount of fat. In theory, they can replace fats for frying and function similarly in food products. These are synthetic compounds that contain fatty acids, are immiscible with water and cannot be digested and/or absorbed or are only partly absorbed. Thus, fat substitutes have properties closely similar to fat, but provide less or no available energy when eaten.

Fat mimetics are food ingredients based on starch, cellulose or protein, generally polymers that have been modified physically, chemically or enzymatically to provide fatlike properties in the water phase. They help replace the mouthfeel of fats, but do not have their other functions. Their energy content is 16 kJ (4 kcal) per gram. However, when mixed with water (in which they are soluble or at least dispersible) they provide only about 4 kJ (1 kcal) per gram, compared with 37 kJ (9 kcal) per gram for fat. Thus, fat mimetics give the mouthfeel but not the other properties of fat, and they provide less energy than fat.

TABLE 10

Fat mimetics based on starch or protein

Water-soluble	Water-dispersible
Maltodextrins	Microcrystalline cellulose
Gums	Protein
ß-Glucan	Starch
Modified starches	

FAT MIMETICS BASED ON PROTEIN OR STARCH

There has been tremendous growth in activity in this field. The long-polymer-chain starting material can be reduced in size by controlled acid or enzymic treatment, for example, in making maltodextrins from starch. Enzymic hydrolysis of starch can be used to yield oligomers (smaller chains) of carboxymethyl cellulose derivatives. Microspheres of proteins can be prepared that will mimic fat. Two large molecules can be reacted under controlled conditions to produce materials such as a protein-gum complex with unique fatlike characteristics.

Water binding

The major change in reduced-calorie products is an increase in the water phase and a reduction in the oil phase. It is desirable to maintain a reduced but still continuous oil phase. Fat mimetics are soluble or dispersible in the water phase in a way that will mimic the texture of the original emulsion (Table 10). One of their main functions is to hold the necessary added water when the oil phase is reduced or replaced.

The first-generation fat mimetics were essentially good water-binding agents that converted added free water to bound water, such as hydrocolloids. But water binding capacity tends to depend on temperature, particle size and pH. The fact that a substance is a good water-binding agent does not mean it can deliver all the other attributes of fat mimetics.

Maltodextrins are prepared from corn, potato or tapioca starches and can be used in various food applications, from liquid systems to baked goods. Gums are usually used as conjugates with other insoluble fat mimetics such as microcrystalline cellulose.

The most widely used water-dispersed fat mimetic is microcrystalline cellulose. It forms a colloid with rheology similar to that of fat, setting up an insoluble network that immobilizes water and imparts a creamy mouthfeel.

Acid-modified maize starch functions in a way similar to that of a network of starch crystalloids that form a tight gel, consisting of 2.5-15-µ micron aggregates of submicron particles.

Mouthfeel

It is especially difficult to mimic the mouthfeel of fat with a fat-free, as distinct from a low-fat, product, since a little fat can go a long way. Fat-free products can lack lubricity and an oily mouthfeel. Starch based mimetics can give a starchy or pasty texture.

The water-dispersed fat mimetics mimic fat in water-continuous emulsions, with protein or starch particles in place of oil and the product stabilized by anti-aggregating agents, additives that maintain a stable colloidal dispersion of the nonfat microspheres. The mouthfeel of the fat mimetic system depends on particle size, size distribution, surface characteristics of the microspheres and the stability of the colloidal suspension.

Stability

Stability is a key issue in the usefulness of fat mimetics. In the dispersed-water category, the main determinant of ingredient stability is whether the microparticles will aggregate to form clusters. Several fat mimetics do not withstand common operating conditions well. Some cannot stand aseptic processing, few will stand retort conditions and some do not even stand pasteurization. This alters texture and sometimes off-flavours form. A related issue is the compatibility of the fat mimetic with particular food-processing conditions, such as dehydration under heat or shear.

Appearance

The fat replacer should provide opacity to compensate for the lack of fat emulsion.

Flavour

This is one of the most critical issues for fat mimetics for four reasons:

- Fat mimetics are used in relatively large amounts, so any off-flavour will come through to the final product.
- The large surface area may bind flavours.
- When the fat level is considerably reduced, the flavour system has to be adjusted. Flavours are increased or decreased, depending on whether they are hydrophobic or water-soluble.
- In fat-free products, flavour molecules may be exposed to faster degradation.

Regulation

Most of the fat mimetics provide clear labels and are generally recognized as safe; many do not have E numbers.

The future of fat mimetics

It will take time for fat substitutes to gain regulatory approval. Meanwhile there will be more fat mimetics tailored for specific food applications. Blends of ingredients will be used, probably in conjunction with flavour delivery systems. Consumers will be looking for products with less total fat and less saturated fat.

Another concern is for reduction of trans fatty acids, which are formed when unsaturated oils are hydrogenated during the production of margarine.

To enable the food technologist to use vegetable oils without hydrogenation, oil-phase texturizing agents are being developed. Unlike fat mimetics, which are incorporated in the water phase, these are added to vegetable oil. Some can be used for spreads and in shortenings for baked goods. These oil-texturizing agents will serve as important complements to the fat mimetics, to further enable the food-processing industry to formulate healthy food products with reduced fat and energy.

FAT SUBSTITUTES

Development of fat substitutes

Developing a new fat substitute that will have the functions and properties of fat but contribute little or no energy to the diet is a lengthy process. There are six stages:

- research leading to the synthesis of fatlike molecules with the ester linkages altered or protected
- toxicological studies in laboratory animals
- human studies
- petitions submitted to food regulatory authorities for approval
- with regulatory approval, development of food products using the new ingredient
- postmarketing surveillance.

At present, most of the potential fat substitutes are between the second and fourth stages. Examples of some of the more promising are given below.

Synthetic triacylglycerols

Chemically triacylglycerols (also called triglycerides) are fats, so they provide all the functions of fat in foods. In one product the three fatty acids, all saturated, are caprylic (8:0), capric (10:0) and behenic (22:0). The two shorter-chain fatty acids, derived from palm kernel and coconut oils, are not thought to present significant risks to health (see above) and may stimulate metabolism. The very long chain behenic acid, prepared by hydrogenation of erucic acid from rapeseed oil, is only poorly absorbed in the body, and the medium-chain fatty acids tend to stimulate metabolism rather than go into storage. As a result, the available energy in synthetic triacylglycerol is only about 21 kJ (5 kcal) per gram, about half that for most fats.

The components of this synthetic triacylglycerol occur naturally, and in some countries it has been approved for use in confectionery. Some concerns remain because knowledge of the metabolism of behenic acid is incomplete. Its use as a frying fat is limited by price and thermal stability.

Polyesters

The chemical name sucrose polyester is not strictly accurate because it is not a long-polymer-chain product. Sucrose serves as the backbone of sucrose polyester, but other sugars such as methyl glucose, raffinose and sorbitol can also be used. The fatty acids can be saturated or unsaturated, and the particular combination used will affect the functional properties, melting point, consistency and stability (less-esterified sugars can be used as emulsifiers).

Fat substitutes of this type are not hydrolysed by digestive enzymes and hence contribute no energy to the diet. The enzymes are unable to penetrate the bulky nonpolar region around the sucrose centre. The esterified natural cis (i.e., bent) unsaturated fatty acids are more protective in this respect than esterified trans unsaturated or saturated fatty acids.

The fat substitutes offer a way of adopting a healthier diet without radically changing the foods we enjoy. They may also reduce cholesterol absorption and reabsorption and lower blood cholesterol concentrations.

The two side effects that have been reported can be countered. Diarrhoea or anal drip can be prevented by incorporating saturated fatty acids so that the compound melts above body temperature. Reduced vitamin A and E absorption can be dealt with by supplementation or dietary selection.

Other fat substitutes

Esterified propoxylated glycerols (EPG) resemble natural triacylglycerols, except that oxypropylene is incorporated between the glycerol backbone and the fatty acid. They are thermally stable and thus suitable for frying and baking. Short-term animal studies suggest they are safe and resistant to hydrolysis.

Dialkyl dihexadecylmalonate (DDM), consisting of a monomer and a dimer, has a melting point below body temperature but higher than that of conventional frying oils. Because it is stable when heated and not absorbed, DDM has the potential for use in a blend with conventional frying oils to make reduced-calorie frying oil. Rat studies have found no toxic effects.

Trialkoxytricarballylate (TATCA) has a structure similar to that of a natural triacylglycerol, with the glycerol replaced by tricarballylic acid and the fatty acids replaced by saturated or unsaturated alcohols. TATCAwas not digested in animal studies.

Polycarboxylic acids designed as fat substitutes have two to four carboxylic acid groups in a polycarboxylic acid backbone esterified with saturated or unsaturated straight or branched-long-chain alcohols. They have physical and functional properties similar to those of the triacylglycerol vegetable oils, but cannot be digested and contribute no energy to the diet. However, one example, trioleyltricarballylate, appears to be toxic for rats at high intakes.

Conclusions

It is clear that fat replacement is here to stay, and the challenge for food technologists is to try to replace the functions of fat in food. Claims for new fat replacers should specify the functions they can replace.

The use of synthetic fatlike products could lead to the most complete maintenance of all of the functions of fat, even in hot frying oils. The major problems are likely to involve safety and thus regulatory approval.

Partial fat replacement is possible in fat-continuous products using emulsifiers. Provided that a significant amount of fat remains in the product, specific functions such as mouthfeel and flavour can be retained relatively easily.

Biopolymers, carbohydrates and proteins can replace several of the functions of fat in a wide range of food products. It is unlikely that any single ingredient of this type could replace all of the functions of fat, but as knowledge of the ingredients increases and manufacturing technology develops, it should become more possible to balance these properties.

GLOSSARY

Atherosclerosis: A degenerative disease of arteries in which there is thickening caused by accumulation of material beneath the inner lining (plaque), eventually restricting blood flow. The material characteristically contains cholesterol and macrophage cells.

Case-control study: Type of epidemiologic study in which the exposure of patients to factors that may cause their disease is compared retrospectively with the exposure of control subjects without the disease.

Cholesterol: A lipid (sterol) made in the body from acetyl CoAand present in the diet; a constituent of cell membranes (especially in nerve tissues), blood and atherosclerotic plaques.

Cis fatty acid: Natural form of unsaturated fatty acid, with a bend in the hydrocarbon chain at the double bond(s).

Cohort or follow-up study: Type of epidemiologic study which measures exposure to factors that may affect health in a group of people (cohort), and relates these factors to the subsequent disease experience (during follow-up).

Coronary heart disease (CHD): A condition resulting from impaired blood supply from the heart's own arteries (coronary arteries) to the heart muscle.

Energy intake: The chemical energy in foods which can be metabolized to produce energy available to the body; usually used to mean the day's total energy (calories) supplied by all the food (and drink) consumed.

Enzymes: Proteins that catalyse specific reactions in the body.

Epidemiology: The study of the distribution and determinants of disease in human populations.

Fat: Triacylglycerol either solid (e.g., margarine, butter, shortening, lard) or liquid (e.g., oil) at room temperature (narrow definition).

Fat mimetics: Compounds that carry out only some of the functions of fat in foods.

Fat-soluble vitamins: Vitamins — organic substances that cannot be produced in the body but are essential for cellular functions and must be obtained from the diet — carried in fat and usually acquired from fatty foods. Vitamins A, D, E and K are the fat-soluble vitamins.

Fat substitutes: Compounds that can replace conventional fat in foods and carry out all of the same functions.

Fatty acids: Organic acids with a hydrocarbon chain of varying length; constituent of triacylglycerols and related compounds.

Fatty streaks: Slightly raised fatty deposits in arteries that do not cause obstruction; possibly but not definitely precursors of atherosclerotic plaque.

High-density lipoproteins: Plasma lipoproteins containing relatively low concentrations of cholesterol and other lipids; thought to be beneficial because they cycle cholesterol out of the tissues.

Hypercholesterolaemia: Concentrations of cholesterol in the blood higher than normal (or reference), values. Causes include dietary and genetic.

Hypertriglyceridaemia: Concentrations of fat (triglyceride or triacyglycerol) in the blood higher than normal, or reference, values.

Intervention trials: Trials in which one or more factors that may affect health are altered, with the aim of demonstrating beneficial effects compared with a control group not receiving the intervention.

Invisible fats: Fats contained in foods and not recognizable as such, e.g., in biscuits, cakes.

In vitro: A reaction or study carried out in the test tube or other laboratory device rather than in an animal's (or human's) body.

Lipid: General name for fatty material insoluble in water (nonpolar), including fats (solid at room temperature), oils (liquid at room temperature), phospholipids and cholesterol.

Lipoproteins: Particles composed of specialized protein and lipids (triacylglycerol, cholesterol and phospholipid) which enable (water-insoluble) lipids to be carried in blood plasma.

Low-density lipoproteins: Plasma lipoproteins containing high concentrations of lipids (so called because their density is low compared with that of water), including cholesterol; increased concentrations are a risk factor for coronary heart disease.

Meta-analysis: Quantitative literature review that combines the results from published studies. In a good meta-analysis, quality criteria for the studies to be included are applied.

Metabolism: Complex, interacting network of biochemical reactions within a living organism.

Oil: Triacylglycerol liquid at room temperature.

Phospholipid: Compounds of glycerol with two fatty acids and one phosphate group.

Plaque (atherosclerotic): Term used for the area on the inside of an artery affected by atherosclerosis. Until its late stages atherosclerosis is not uniform but occurs in small patches, or plaques.

Risk factor: Physical condition or life-style that in epidemiologic studies appears to increase susceptibility to a particular disease.

Satiation: The term for the physiologic processes that bring eating a meal to an end and control meal size.

Satiety: The feeling of having eaten enough.

Saturated fat: Triacylglycerol with a preponderance of saturated fatty acids.

Saturated fatty acid: Fatty acid whose hydrocarbon chain contains no double bonds.

Thrombosis: Blockage of artery resulting from aggregation of platelets and fibrin, a fibrous protein.

Trans fatty acid: A form of unsaturated fatty acid that is straight (rather than bent i.e., cis) at a double bond; not common in nature but formed during some manufacturing processes.

Triacylglycerols: Compounds of glycerol and three fatty acids also called triglycerides; the main constituent of dietary and body fat.

Unsaturated fat: Triacylglycerol with a preponderance of unsaturated fatty acids.

Unsaturated fatty acid: Fatty acid whose hydrocarbon chain contains at least one double bond. Monounsaturated fatty acids contain one double bond; polyunsaturated fatty acids contain two or more.

Visible fats: Fats recognizable as such, e.g., butter, margarine, cream and fat in meat.

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