TABLE 2. THE EFFECT OF TRANS FATTY ACID ON SERUM LIPIDS

Serum Lipids	Effect	
	Increased	Decreased
Total Cholesterol	↑	
LDL-Cholesterol	↑	
HDL-Cholesterol		↓
LDL/HDL ratio	↑	
Lipoprotein Lp(a)	↑	

1.4 for women in the highest quartile of *trans* fatty acids in adipose tissue. The report by Hu *et al.* (1997) showed an association between *trans* fats and coronary heart disease. They studied 80 082 women who were 34 to 59 years of age and had no known coronary heart disease, stroke, cancer, hypercholesterolemia or diabetes in 1980. During 14 years of follow up, they documented 939 cases of non fatal myocardial infarction or death from coronary heart disease.

Total fat intake was not significantly related to the risk of coronary heart disease (for a 5% increase in energy from fat, the relative risk was 1.02). As compared with an equivalent energy from carbohydrates, the relative risk for a 2% increment in energy intake from *trans* unsaturated fat was 1.93; that for a 5% increment in energy from monounsaturated fat was 0.81; and that for a 5% increment in energy from polyunsaturated fat 0.62. Each increase of 5% energy intake from saturated fat, as compared with equivalent energy intake from carbohydrates, was associated with a 17% increase in the risk of coronary heart disease and a relative risk of 1.17 (*Table 3*). The relative risks for women in the upper quintile were estimated as 1.35 and 1.27 in comparable models. They concluded that the quality of fat rather than the total amount of fat is important in modifying the risk profiles for CHD.

TABLE 3. RELATIVE RISK OF CHD ASSOCIATED WITH INCREASES IN THE PERCENTAGE OF ENERGY FROM SPECIFIC TYPES OF FAT AND DIETARY CHOLESTEROL

Variable	Relative risk
Saturated fat (each increase of 5% energy)	1.17
Monounsaturated fat (each increase of 5% energy)	0.81
Polyunsaturated fat (each increase of 5% energy)	0.62
Trans unsaturated (each increase of 2% energy)	1.93
Cholesterol (each increase of 200mg/1000 keal)	1.12

Source : Hu *et al.* (1997)

Trans fatty acids can deleteriously affect lipoproteins by increasing TC, LDL-C, lipoprotein (a) and decreasing HDL-C relative to their cis isomers. This has raised the need to replace hydrogenated fats with natural solid fats in a large number of food formulations. The nutritional efficacy of the solid fats replacing hydrogenated fats should be such that they do not adversely affect plasma lipids and other CHD risk factors. In this context, palm oil is perceived as a suitable alternative.

Nestel *et al.* (1992) compared a *trans* elaidic rich fat with a 16:0-rich blend (16:0 contributed mainly by palm oil). Both test blends resulted in higher TC and LDL-C than an oleic-rich control diet. There was essentially no difference in TC and LDL-C between the elaidic-rich and palm oil-rich diets. HDL-C was however significantly raised on the 16:0-rich diet and the resulting LDL/HDL-C ratio was more favourable on the palm oil diet than the *trans* diet. This led the authors to conclude that there is little benefit from avoiding the use of palm oil by substituting *trans* fatty acids in food formulations. Sundram *et al.* (1997) undertook a direct comparison between *trans* elaidic fat designed to replace the saturates (16:0, 12:0 + 14:0) in foods and food processing. Feeding of elaidic acid at 5.5% en significantly elevated TC and LDL-C relative to the 16:0- (palm olein) and 18:1-rich fats, uniquely depressed HDL-C and

increased lipoprotein (Lp(a) relative to all the fats tested (including 12:0 + 14:0). The 16:0 and cis 18:1-rich diets elicited identical effects on lipoproteins. The authors concluded that the impact of *trans* elaidic acid on the lipoprotein profile of humans appears to be worse than the saturates occurring in natural oils and fats.

While there seems to be wide agreement that *trans* fats have adverse effects on cholesterol profiles and CHD risks, yet there is little agreement about either the magnitude of the problem or what actions should be taken. The reluctance to recommend measures remains an enigma especially when reducing the intake of *trans* fats may not be particularly difficult (Byers, 1997).

THE MEDITERRANEAN DIET

For a long time, the low rates of coronary heart disease in populations in the Mediterranean was specifically attributed to their diets being low in saturated fat and high in monounsaturates. This was based on the classic study of Ancel Keys in the 1950s (Keys, 1980). The argument of several scientists from the Mediterranean countries that the diet of their region was more than a low-saturated fat diet and had implications for diseases other than CHD was lost to the wider scientific community.

It is recognized now that the overall Mediterranean dietary pattern is more important than individual nutrition components. The common components identified in the Mediterranean diet include: the energy intake from fat is very high (40%), a large fraction of dietary fat, close to 50%, is monounsaturated and mainly derived from olive oil with a high monounsaturated/saturated fatty acid ratio; ethanol consumption at moderate levels and mainly in the form of wine; high consumption of vegetables, fruits, legumes, and grains; moderate consumption of milk and dairy products, mostly in the form of cheese; and low consumption of meat and meat products (*Table 4*).

Recognition is growing that several aspects of the Mediterranean diet, and in particular consumption of olive oil in conjunction with vegetables and legumes, can convey a substantial degree of protection against a wide range of chronic diseases (Trichopoulou and Lagiou, 1997a). Nevertheless, it is to be appreciated that substitution of traditional edible oils with olive oil would not transfer all the benefits of the Mediterranean diet and so one should not get the wrong message and consume monounsaturated fats with impunity. Studies conducted in Malaysia have shown that palm oil and olive oil incorporated into diets resulted in similar serum lipid profiles in healthy individuals.

TABLE 4. SOME CHARACTERISTICS OF THE MEDITERRANEAN DIET

High monounsaturated/saturated fat ratio
Ethanol consumption in the form of wine at moderate levels
High consumption of vegetables, fruits, legumes and grains
Moderate consumption of milk and dairy products (cheese)
Low consumption of meat and meat products
Physical activity
Relaxing psychosocial environment
Afternoon siesta habit
Favourable climate

Source : Trichopoulou and Lagiou (1997a)

Ng *et al.* (1992) evaluated the effects of palm olein and olive oil on serum lipids and lipoproteins in comparison to a coconut oil diet. Each test oil was served as the sole cooking oil and contributed two thirds of the total fat intake. The coconut oil diet significantly raised all the serum lipid and lipoprotein parameters, *i.e.* TC, LDL-C and HDL-C. However, the one-to-one exchange between palm olein (rich in 16:0) and olive oil (rich in 18:1) resulted in similar TC, LDL-C and HDL-C values. This showed that in healthy normocholesterolaemic humans, palm olein can be exchanged for olive oil (high oleic) without adversely affecting the serum lipids and lipoprotein levels. Choudhury *et al.* (1995) managed a 5% en exchange between palm oil (16:0-rich) and olive oil (18:1-rich) in 21 healthy normocholesterolaemic Australian men and women consuming a low fat (30% en) and low dietary cholesterol (<200 mg/day) diet. Under these conditions, TC and LDL-C were not significantly different between the two oils, so that when 16:0 in palm oil was replaced with 18:1 in olive oil, the expected

increases in TC and LDL-C were not evident. In a previous human study, Truswell *et al.* (1993) also reported a similar effect between palm olein and canola oil.

Sundram *et al.* (1995) fed 23 healthy normocholesterolaemic male volunteers carefully designed whole food diets containing canola oil (18:1-rich), palm olein (16:0-rich) or an American Heart Association Step 1 diet (AHA), all contributing approximately 31% en fat and <200 mg dietary cholesterol/day. These diets represented the direct exchange of 7% en derived from 18:1 + 18:2 between canola oil and palm olein whereas the main difference between palm olein and AHA was <4% en exchanged between 16:0 and 18:2. Serum TC, VLDL-C and LDL-C were not significantly affected by the high 18:1 canola and the high 16:0 palm olein diets. Only HDL-C after the AHA diet was significantly raised compared with the other two diets.

In contrast to the above studies, Zock *et al.* (1994) reported that replacing 10% en from 16:0 with 18:1 in normocholesterolemic subjects significantly lowered TC and LDL-C. This Dutch study did not use natural fat sources. The 18:1-rich diet was prepared by blending high 18:1 sunflower oil, fully hydrogenated sunflower oil and high 18:2 sunflower oil and interesterified palm oil mixed with other edible oils. The 16:0-rich diet was formulated by blending fractionated palm oil, cottonseed oil, and fully hydrogenated sunflower oil. The feeding of fat blends containing atypical triglyceride moieties may have been partially responsible for the observed increase in TC and LDL-C. By contrast, when Sundram *et al.* (1990) maximally replaced the habitual Dutch diet with palm oil, TC and LDL-C were unaffected. The palm oil diet however resulted in significant improvements in HDL2-C and the apolipoprotein A1/B ratio signaling some cardiovascular benefits rather than the reverse to be true for palm oil.

GENETICS AND DISEASE

There is evidence now that many common diseases such as atherosclerosis, diabetes and cancer involve not only changes in the gene or a genetic predisposition to the illness but also dietary factors that may enhance or inhibit expression of the genetic predisposition. Genetics determines the susceptibility to disease, but whether or not a predisposed individual develops the disease may depend on environment factors such as nutrition (Simopoulos, 1997).

Since genetic variants are expressed in a specific environment, populations should not copy each other's dietary recommendations for the prevention of coronary heart disease. The varying needs of people have always made mass diet prescriptions scientifically, culturally and sociologically incorrect. It has a tendency to encourage people to follow advice that is either unnecessary or inappropriate for them, seek alternative solutions, or ignore the problem (Dodd, 1997).

VERY LOW FAT DIETS MAY HARM SOME PEOPLE

The genetic make up of a person influences how he or she responds to a particular diet, and although very low fat diets may benefit some people, they could be harmful to others, (BMJ News, 1998). Individual responses to food cannot be predicted reliably on the basis of studies of large populations of people.

Studies in healthy people show that there are genetic differences in the response to a low fat diet (*Table 5*). Those who from their metabolic profiles, are at highest risk of heart disease show the greatest benefit from very low fat diets, but the remaining two thirds of the population show only minimal benefit, and for some they would be harmful. Dietary approaches to reducing CHD do not benefit all individuals to the same degree. The lipid lowering response to a low saturated cholesterol lowering diet designed to cut risk for CHD varied widely among 120 individuals studied (Schaefer *et al.*, 1997).

TABLE 5. GENES ASSOCIATED WITH LIPID METABOLISM

Apolipoproteins: Apo A-1V-1/1, Apo E polymorphism, Lipoprotein a
Enzymes: HMG CoA reductase, Lipoprotein lipase, Triglyceride lipase, LCAT
Transfer proteins: Lipid transfer proteins, CETP
Receptors: Apo E, HDL, LDL-receptor activity

People with apo E 4 genetic variant of the apoprotein E tend to have a higher blood cholesterol concentration, increased risk of heart disease and increased risk for Alzheimer's disease. Another genetically influenced condition, LDL-subclass pattern B, influences the blood cholesterol response to a low fat diet. This condition is characterized by small dense forms of LDL, lower blood levels of high density lipoprotein (HDL), increased blood concentration of triglycerides, and a predisposition to the most common form of diabetes mellitus. People with this condition have a three fold higher risk of coronary heart disease than those with larger forms of LDL. People with the small dense forms of LDL (pattern B) respond to a low fat diet better than people with larger forms of LDL (pattern A). Unless a person knows that he has pattern B LDL, he should avoid extremely low fat diets, as he could develop adverse effects (Krauss, 1997).

Methods to predict dietary responsiveness should be developed to target those likely to receive the greatest benefit. Research on the interaction of genes and diet should, in the future, lead to dietary plans and possibly drug regimes that are tailored to an individual's predisposition for heart disease and stroke.

OBESITY: THE FATTENING OF SOCIETY

The prevalence of obesity appears to be increasing globally and is a relatively recent public health concern, particularly in developed nations. It is a multifaceted problem with wide reaching medical, social and economic consequences (*Table 6*). Current data suggests that genetic influences, in the form of susceptible genes, and gene environment interactions play important roles in the etiology of obesity (Jeffcoate, 1998). Both genetic and environmental factors modify body weight. The appreciation that the current epidemic of obesity is largely environmental, because of a mismatch between man's ancient genes and his modern environment, will help direct preventive and therapeutic strategies (Bray, 1998).

Body weights in the North American population have slowly crept up over the past century. Obesity rates in Britain have more than doubled since 1980. Currently, 54.4% of adult Americans are overweight and 22.5% are obese. For this national expansion there can only be two explanations: Americans are eating more, and/or exercising less. Obesity presents two challenges: treating people who are currently obese and preventing obesity in people who are still lean or moderately overweight. Neither of these challenges are currently being addressed adequately. Diet and exercise programmes remain the cornerstone approaches. There is good evidence that reducing total calories intake is the most important factor in weight loss. Increasing physical activity of all individuals who are obese should be an important public health priority (Foreyt and Poston, 1997).

TABLE 6. OBESITY ASSOCIATED SERUM LIPID CHANGES

Serum lipids	Effect
Triglycerides	↑
Free Fatty Acids	↑
Total Cholesterol	↑
LDL-Cholesterol	↑
LDL/HDL ratio	↑

A recent report in the Lancet (Bray, 1998) evaluates the treatment of obesity using a pharmaceutical agent 'orlisat' that is not an appetite suppressant. It acts by blocking the pancreatic lipase, thereby reducing triglyceride digestion and the amount of fat available for metabolism and thus induces weight loss.

THE ROLE OF SATURATED FATS

Saturated fats have been much maligned because of their association with animal fats and the earlier concerns about hypercholesterolemia and heart disease. It

is now well established that all saturated fatty acids are not equivalent in terms of their hypercholesterolemic effects. Stearic acid, for example, is neutral in its cholesterolèmic action. What is important is the quantity and quality of fat in the diet as is becoming evident from recent studies (Khosla and Sundram, 1997).

Saturated fatty acids have important biological roles (Rogers, 1995). The best weight gain in experimental animals was obtained with fat mixtures containing 30% saturated fatty acids in diets. Low levels of dietary saturated fatty acids are incompatible with maximum weight gain in weanling rats. In newborn piglets fed a milk replacer, a low platelet count occurred with vegetable oils having a low level of saturated fatty acids. A 1990 WHO report points out that 'epidemiological data suggest that as the intake of saturated fatty acids decreases to about 10% energy there is a progressive fall in mortality due to cardiovascular disease'. The evidence for lowering the level of saturates further in the total diet is still missing.

The role of different saturated fatty acids in regulating lipoprotein metabolism continues to be under investigation. The effect of palmitic acid on plasma lipoprotein cholesterol remains debatable. The human study of Cook *et al.* (1997) investigated the relationship between endogenous synthesis of cholesterol and the content of palmitic acid in a diet contributed by palm oil. The high levels of palmitic acid in the diet did not significantly affect serum total and LDL-cholesterol levels. Fractional synthetic rate of cholesterol was not different between dietary treatments (high versus low palmitic acid contents). This suggested that there was no relation between endogenous synthesis of cholesterol and the palmitic acid content in the diet.

DIETARY FATS AND CANCER

A recently released report from the Department of Health (UK) in a rigorous review of the literature found little evidence for a strong association between the intake of total fat or of individual fatty acids and any type of cancer (Gurr, 1998).

TRENDS IN CONSUMPTION OF OILS AND FATS

The importance of oils and fats in meeting the ever increasing population demand for food and non food products continues to grow. Of the visible dietary lipids, vegetable oils have gradually been assuming greater importance. An analysis of the relationship between the percentage of energy from fat and GNP per capita for the period 1962-1990 reveals that the structure of the income-diet relationship has undergone a significant change. The world average per capita consumption of oils and fats has increased substantially from 14.57kg in 1987 to 17.12kg in 1997, with developed nations having a higher consumption than the world average. The proportion of energy from vegetable fats in 1990 was much higher than in 1962, accounting for up to 15% of the total energy. The data suggest that the percentage of energy from fat was less dependent on income than previously. The dramatic difference was largely accounted for by a major increase in the consumption of vegetable oils and fats (Drewnowski and Popkin, 1997). The growth in demand for vegetable oils is strongest in the developing nations of Asia. In this regard oil palm cultivation and palm oil production have contributed significantly to the edible oil needs of the world. During the past decade, palm oil's share of total vegetable oil production increased from 15% to about 21% (*Table 7*). Animal fats were replaced by a greater proportion of vegetable oils and products. Because of the growing concern over consumption trends and their adverse health effects, especially cardiovascular risks, there was a worldwide demand for vegetable oils and fats. It is envisaged that oils and fats consumption will undergo gradual changes due to economic factors, nutritional concerns and environmental considerations (Trichopoulou and Lagiou, 1997b).

CONCLUSION

The implications of some of the above observations as well as their consequence on diet and disease will be profound. The quantity and quality of dietary fat may influence lipid metabolism and the health

effects of specific fatty acids continue to be the subject of much research, discussion and debate. Fats have a place in the total diet and the lowest fat diets are not necessarily the most healthy. Disease risk is associated with the total diet over time and we have to take cognizance of public health nutrition principles (*Table 8*). It is important to recognize the impact of fats on overall health. Nutritionists recognize that focussing on fat alone will not achieve a better overall nutrition, or even an improved fat intake. Dietary recommendations have to change with time and the evidence available.

TABLE 7. WORLD PRODUCTION OF 17 MAJOR OILS FATS*

Total vegetable oils	79 425 000 tonnes
(Palm oil)	(15 518 000) tonnes
Total animal fats/oils	20 487 000 tonnes
Grand total	99 912 000 tonnes

*PORLA - 1997

TABLE 8. PUBLIC HEALTH NUTRITION PRINCIPLES

The reduction in risk for disease is affected by the total diet and life-style pattern, not by use of an individual food;

Individual foods by themselves do not prevent or cause a disease;

All the chronic diseases in which diet has been implicated to play a causative and/or preventive role, are multi factorial in nature as to aetiology and progression;

The precise role of diet for many such diseases remains to be determined; and

The role of diet for each individual cannot be predicted because of marked individual variability resulting mainly from hereditary and life-style factors.

Source: DA Wenck, Banen, M, Dewan, S.P. (Eds). Nutrition, Boston Publishing Co., Boston, Virginia, 1983.

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