Changing Perceptions on the Role of Saturated Fats in Human Nutrition

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INTRODUCTION

Dietary fat continues to be a major research priority because of its association with heart disease, cancer and other chronic diseases (Chandrasekharan, 1999). Misconceptions about fats abound. Saturated fats have been much maligned because of their association with animal fats and the earlier concerns about hypercholesterolemia and heart disease (Uffe Ravnskov, 1995). Frequent and conflicting reports in the media about their health effects often create confusion in the minds of the public (Chandrasekharan and Yusof, 1998). There is a tendency for the media to focus on the negative health implications of dietary fats and to overlook its nutritional importance. This situation has no doubt influenced consumers' perceptions of fats and oils. However, there has been a better understanding of oils and fats and their effects on health and disease over the years. It is now well established that all saturated fatty acids are not equivalent in terms of their hypercholesterolemic effects and that it is the total dietary fatty acids that are important in nutrition (Chandrasekharan et al., 2000).

THE BENEFITS OF FATS AND OILS

Dietary fat and nutrients associated with it play a critical role in the health and functioning of the human body (*Table 1*). Fat is a concentrated energy source relative to carbohydrate and protein and is an efficient form of stored energy. About 80% of the ingested fat and the saturated and unsaturated fatty acids it contains is stored in special cells and burnt up as required. The various fats also contribute to the physical and functional properties of most food products, affecting the sensory as well as nutritional aspects of foods to make the foods more palatable. Dietary fat including saturated fats supply fatty acids which become constituents of the structural lipids of the cells, particularly the cell membranes and neural tissue. Some fats have specific physiological roles to play (Rogers, 1988).

TABLE 1. FUNCTIONS OF FAT

Supply energy
Store energy
Give satiety value
Carry fat soluble vitamins
Supply essential fatty acids
Perform metabolic functions
Cushion body organs
Keep body warm

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DIETARY RECOMMENDATIONS AND REQUIREMENTS FOR FATS

Fats and oils are components of normal diets and consumed by the vast majority of the population on a regular basis. Like other micronutrients, fat comprises a number of different components (*Table 2).* Dietary fat contains an average 96% by weight as fatty acids, the other 4% is glycerol and other lipids. In mixed natural diets, the share of C:12 - C:16 in total saturates is fairly constant at 60%-70% by weight. Data from the United States show that 50% of the fat and about 59% of the saturated fatty acids (SFA) come from meat, poultry, fish, egg and dairy products (Nelson, 1991). Again like other micronutrients, it is almost never eaten in isolation but in combination with other nutrients and in a variety of forms.

TABLE 2. COMPONENTS OF DIETARY FATS

Triacylglycerols
Phosphatides
Sterols
Mono and di-glycerides
Free fatty acids
Fatty alcohols
Fat soluble vitamins and antioxidants

The requirement for total fat - the most concentrated source of energy may vary greatly depending on the physiological needs that are to be met. The desirable intake of

fats and essential fatty acids change from infancy to old age but quantification of the requirements for each stage of life has still to be accomplished. The determination of the optimal range of fatty acids in the human diet for development and health will keep us challenged for sometime. According to the FAO/WHO, the minimum recommended level of fat intake is 20% of the dietary energy (Scrimshaw, 1998). On an average, fat contributed 42% of the energy in the diet of people in the United Kingdom. National dietary guidelines in most industrialized countries recommend that intakes of total fat and SFA be reduced. The recommendation that a diet contain equal proportions of SFA, monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) was intended for a population with a high intake of fats.

TRENDS IN CONSUMPTION OF OILS AND FATS

Population growth, economic progress and urbanization lead to an increase in the consumption of oils and fats as well as greater dietary diversity in both developing and developed nations. The importance of oils and fats in meeting the ever increasing population demand for food and non-food products continues to grow (*Table 3*). The growth in demand for vegetable oils is strongest in the developing nations of Asia (Drewnowski and Popkin, 1997). During the past decade, palm oil's share of total vegetable oil production increased from 15% to about 21% (Chandrasekharan and Yusof, 2000).

Fat consumption is usually greater than the recommended levels in developed countries. In the United States, data suggest a steady annual increase in the average consumption of grammes of fat during the last 50 years. While the average amount of saturated fat has stabilized since 1960s (16 en %), the rates of increase in PUFA have accelerated fast enough to maintain an upward trend. This is reflective of western countries. The average amounts of fats consumed are also increasing in countries that are improving their general socio-economic conditions. The change from lower to higher levels of fat consumption by adding steadily increasing proportion of saturated fat is probably representative of many rapidly developing countries. The average annual per capita consumption of fat is around 8 kg in the most populous nations of Asia as compared with 16 kg for the world and more than 40 kg for the developed countries.

In health conscious individuals, there is a growing awareness of the adverse effects of excessive dietary fat intake. Concurrently, there are public health concerns about the quantity and quality of fat in the diet and one message that is given is to *choose a diet that is low in saturated fat and cholesterol and moderate in total fat*.

TABLE 3. WORLD POPULATION AND CONSUMPTION PATTERN OF OILS AND FATS

Year	1995	1996	1997	1998	1999	2000
Population (in billion)	5.659	5.744	5.823	5.902	5.980	6.057
Consumption of fats (kg caput^{-1} yr^{-1})	16.16	16.86	17.28	17.46	18.10	18.80
World production (million tonnes)	94.00	96.94	101.15	102.68	109.74	114.51
Palm oil's share (%)	16.17	16.80	17.73	16.41	18.75	19.04

Source: Oil World Annual (2001).

THE ROLE OF SATURATED FATS

The body has a requirement for all types of fatty acids - saturated, monounsaturated and polyunsaturated. For the total fatty acids in an adult diet, SFAs could be 10%-25%, linoleic acid could be 10%-20% and linolenic acid could be about 2% (PUFAs) and the rest could be oleic acid (Rogers, 1995). Saturated fats can be derived both from animal and vegetable sources and their fatty acid content is variable (*Table 4*).

SFAs are also major components of phospholipids in the membrane lipid bilayer and can be regarded as an essential component of the membrane structure. For proper functioning of membranes, a good combination of these three classes of fatty acids is essential. The phospholipids in the gray matter of the brain are rich in saturated fatty acids. The phospholipids of the lining lung surfactant lipids are esterified entirely with palmitic acid, without which the lungs would collapse during expiration. Palmitic acid is also an important structural storage lipid in man and is preferentially oxidized as a source of energy by the body. Palmitic acid is the second most abundant fatty acid (after oleic acid) and the most abundant SFA in the United States and United Kingdom diets, accounting for approximately two-third to three-quarter of all SFAs consumed (8%-10% of total calories). It is present in fish oils, in milk, body fat and virtually all vegetable oils.

If we are to understand the health benefits of SFAs and lipids, it is necessary to specify the effects of each saturated sub-group. The different biological effects of the three saturated sub-groups tend to be ignored. The recognition of saturated fats as either short, medium or long and not merely saturated has been advocated for sometime now (Kabara, 2000a). It has been known for decades that sub-groups exist for unsaturated fats, *i.e.* monounsaturated fats (n-9) and polyunsaturated fats (n-6 - vegetable oils, and n-3 - fish

TABLE 4. SATURATED FATTY ACIDS IN THE DIET

Fatty acid	Abbreviation	Common sources	Remarks
Butyric	C4:0	Butter fat.	All these fatty acid can also be synthesized by the body from carbohydrate sources when the diet is low in fats.
Caproic	C6:0		
Caprylic	C8:0	Butter fat, coconut.	
Capric	C10:0	Coconut.	
Lauric	C12:0	Coconut and palm kernel oil (PKO).	
Myristic	C14:0	Butter, PKO, coconut, tallow, lard, chicken fat.	
Palmitic	C16:0	Vegetable oils: palm, corn, peanut, rapeseed, coconut, soyabean, cocoa. Animal fats: tallow, lard, butter, chicken fat.	
Stearic	C18:0	Cocoa butter, palm oil, soyabean oil, beef tallow, lard, butter, chicken fat.	Can be converted to oleic acid (MUFA) by desaturases in the human body.
Arachidic	C20:0	Lard, peanut oil.	There are also referred to as the long chain fatty acids (LCF).
Behenic	C22:0	Peanut oil.	
Lignoceric	C24:0	-	
Trans	*t*	Hydrogenated fats - usually from hydrogenation of polyunsaturated oils.	Adverse health effects including a higher risk for CHD have been reported.

oils), but little recognition is given even today to the subgroups of saturated fats (Kabara, 2000b). Each sub-group has different metabolic, biological and pharmacological functions. Not only is the composition of the saturated fat important but the position of fatty acid attachment to the glycerol structure also is significant.

FAT INTAKE AND CORONARY HEART DISEASE (CHD) RISK

Many inherited and environmental factors interact to affect predisposition to development of CHD (*Table 5*). For many years, a fat-rich diet has been considered a major cause of atherosclerosis and cardiovascular disease, in particular CHD. The nature of the so called atherogenic diet has been disputed but most health authorities consider SFA harmful and PUFA beneficial (Uffe Ravnskow, 1998). The atherogenic diet is thought to operate by raising serum low density lipoprotein cholesterol (LDL-C), as a high LDL-C is thought to stimulate atherosclerosis and thus CHD.

There has been an attempt to measure the atherogenecity of a food by taking into account its content of cholesterol, SFA and PUFA. This may lead to some interesting exercises but the characteristics of the total diet rather than those of the individual foods are of critical importance. Among the ingredients in the diet, the type of fatty acids, especially SFAs are considered to be the major contributors to hypercholesterolemia, an established risk factor for CHD. This explains the emphasis on the reduction of SFAs by most health agencies (Grundy, 1991). Uffe Ravnoskov (1995;1998) in reviewing longitudinal studies within populations, found no differences between the diet of coronary patients and others (*Figure 1*).

Both epidemiological and controlled clinical trials suggest that

TABLE 5. PUBLIC HEALTH NUTRITION PRINCIPLES

- The reduction in risk for disease is affected by the total diet and lifestyle pattern, not by 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/preventive role are multi factorial in nature as to etiology 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 heredity and life style factors.

Source: Wenck et al. (1983).

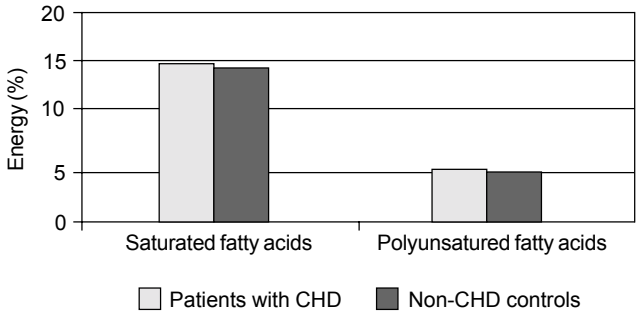

Figure 1. Intake of saturated and polyunsatured fatty acids in patients with CHD and non-CHD controls.

each 1 mg dl^{-1} (0.026 mmol $litre^{-1}$) increment in LDL-C causes an increase in coronary risk of 1%. Epidemiological observations also show an increase of 2%-3% in risk for each 1 mg dl^{-1} (0.026 mmol $litre^{-1}$) decrease in high density lipoprotein cholesterol (HDL-C). A causal relationship between changes in HDL-C and changes in risk is credible but not proven (Mensink and Katan, 1992).

The effects of diet on other risk factors for CHD such as blood pressure, platelet function and HDL-oxidability are important. Unfortunately, the extent of these effects in humans is not well defined. Questions about diet and CHD risk cannot be settled by drawing theoretical inferences from short-term dietary studies (Mensink and Katan, 1992).

Mortality from CHD has decreased in most countries for many years. Better treatment may be another explanation than better prevention because the 40% decrease in CVD mortality seen in Framingham between 1950-70 was followed by a 40% increase in its prevalence (Uffe Ravnskov, 1998). High intake of fat and low cardiovascular death rates were characteristic of countries with a high per capita GNP. The intake of SFA correlated with prevalence and five-year incidence of CHD mortality, but not with major ECG findings at entry (Uffe Ravnskov, 1998).

ALL SATURATED FATTY ACIDS ARE NOT EQUIVALENT IN THEIR CHOLESTEROLEMIC EFFECTS

The formulae of Keys *et al*. and Hegstedt *et al*. (Khosla and Sundram, 1996) on the relationship between dietary fats and serum cholesterol level formed the basis of policies for the dietary prevention of ischaemic heart disease (IHD). According to their regression equation, SFAs strongly elevate LDL-C levels. The predicted effect on total serum cholesterol level largely mirrored that on LDL-C. An important and practical question is whether all SFAs raise the LDL-C level. If not, then it is inappropriate to lump all saturates together when making dietary recommendations.

These formulae have been superceded by others because they were considered inadequate. Values predicted using the formulae tend to be higher than the laboratory values (*Table 6*). Wide discrepancies between observed and predicted values have been reported by Prior *et al*. (1981) and Ahrens (1986). Further, they do not differentiate between the effects of diet on LDL and HDL cholesterol. The distinction is important because LDL and HDL cholesterol may have opposite effects on the risk for IHD and some studies have suggested that the cholesterol lowering effect of (n-6) PUFAs is not limited to LDL but extends to HDL-C. The equations for HDL-C show that under isocaloric conditions (metabolic ward type), all three classes of fatty acids will elevate HDL-C when they replace carbohydrates in the diet. The effect diminishes with increasing unsaturation (Mensink and Katan, 1992). Several lines of evidence suggest that all SFAs do not have the same effect on LDL-C concentration. Some may not raise the level at all, and even among those that do increase the LDL-C levels, the degree of rise may not be identical.

The medium and short chain fatty acids with less than 10 carbon atoms (caprylic and caproic) are handled by the body more like carbohydrates than fats and have no effect on plasma cholesterol concentrations. Fatty acids with less than 12 carbon atoms have no cholesterolemic effect (Khosla and Sundram, 1996). Some believe that lauric acid (C12:0) has seemingly little cholesterol raising action. Keys equated lauric acid with palmitic acid in its cholesterol raising action, whereas Hegsted postulated that it is only mildly hypercholesterolemic (Khosla and Sundram, 1996). The diet normally contains much less myristic acid (C14:0) than palmitic acid. Current evidence suggests that myristic acid (C14:0) has the highest potency

TABLE 6. LACK OF CONGRUENCE BETWEEN THEORETICAL AND OBSERVED VALUES IN SERUM CHOLESTEROL LEVELS IN POLYNESIANS*

Subjects/place (n)	Serum cholesterol levels mg /100 ml		
	Observed	Theoretical	Difference (%)
Pukapuka			
Males (87)	170	238	68 (40)
Females (78)	176	257	75 (43)
Tokelau			
Males (26)	208	290	82 (39)
Females (51)	216	296	80 (37)

Source: *adapted from Prior *et al*. (1981).

for raising total cholesterol and LDL-C levels. Myristic acid is four to six times as hypercholesterolemic as the other two cholesterol raising saturates - lauric and palmitic acids. This is directly related to its ability to down regulate LDL-receptor activity in humans (Grundy, 1991).

Recent evidence suggests that palmitic acid (C16:0) is neutral. This neutrality is however subject to the make up of the host and to a large extent on the total amount of fat calories consumed (Sundram *et al*., 1995). 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-C levels. The fractional synthetic rate of cholesterol was not different between the 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.

Stearic acid (C18:0) has long been postulated to be a neutral fatty acid. Several lines of evidence suggest that stearic acid does not raise the serum cholesterol level. Stearic acid is rapidly converted to oleic acid by desaturation - which could account for its lack of hypercholesterolemic effect. If stearic acid should not be included with the SFA, the whole field will have to be reevaluated. For example, the original equation developed by Keys *et al*. was: SC=2.74S-1.31P. The size of S is lowered by removing stearic acid, the coefficient of S will have to increase. The cholesterol elevating effects of S would have to be accounted by a smaller value of S. It is possible that the whole equation might change (Khosla and Sundram, 1996). Even though stearic acid does not have a cholesterol raising effect compared with oleic acid, it may lower HDL-C and increase lipoprotein(a) Lp(a) concentrations (Khosla and Sundram, 1996). In FDA labelling, stearic acid has not been removed from the list of cholesterol raising fatty acids.

It is obvious that different saturated fatty acids are by no means equivalent in terms of their influence on blood cholesterol levels. Metabolic studies suggest that saturated fatty acids differ in their effects on blood lipids and lipoprotein concentrations. Medium chain saturated fats fail to raise cholesterol levels when supplied with sufficient polyunsaturated fatty acids to avoid essential fatty acid (EFA) deficiency. Further, different hypercholesterolemic fatty acids have different thresholds at which they exert an effect on plasma cholesterol, which are dependent on the concentration of linoleic acid and cholesterol in the diet and the initial plasma LDL-cholesterol concentration (Sundram *et al*., 1995). Studies showing harmful effects of so-called *tropical oils* generally were carried out in the absence of EFA in the diet (Kabara, 2000b). Palm oil was a victim of misperceptions based on misinformation. However, many well controlled studies have vindicated the health and nutritional benefits of palm oil (Sundram and Chandrasekharan, 2000), as summarized in *Table* 7.

The different effects of specific saturated fats on plasma lipids and lipoproteins imply that that these fats may have different effects on the risk of CHD. The role of different SFAs in regulating lipoprotein metabolism continues to be under investigation.

HOW DO SFAs RAISE SERUM CHOLESTEROL LEVELS?

The action of SFAs to increase cholesterol levels occurs independently of dietary cholesterol. The mechanism whereby SFAs raise serum cholesterol has yet to be determined with certainty. Their major effect is however on LDL-C. One possibility is that they enhance hepatic synthesis of apolipoprotein (apo) B containing lipoproteins. Another is that they *down regulate* the synthesis of LDL-receptors. Dietary cholesterol raises plasma LDL concentration by exactly the same mechanism (Grundy, 1991).

Fatty Acid Distribution

The way in which fatty acids are distributed in a triacylglycerol may also influence plasma cholesterol, irrespective of the overall composition of the fatty acids. Triacylglycerols in all naturally occurring fats and oils contain mixtures of different fatty acids, saturated and unsaturated, within the same molecules (*Table 8*). The terms *saturated* and *unsaturated* fats are therefore imprecise and misleading and we should speak of fats or oils containing a high or low proportion of saturated or unsaturated fatty acids (Kabara, 2000a). Further, the different fatty

TABLE 7. NUTRITIONAL BENEFITS OF PALM OIL

Palm oil and palm olein have many nutritional benefits which have been confirmed in both metabolic and epidemiological studies. They:

- are easily digested, absorbed and metabolized;
- play a useful role in meeting the energy and EFA needs in many regions of the world;
- are cholesterol free;
- are naturally saturated. This means that hydrogenation is not required;
- are rich in protective antioxidants, beta carotene and vitamin E;
- are composed of a balanced mixture of fatty acids;
- contain a moderate amount of linoleic acid; and
- have not been shown to elevate blood cholesterol levels in comparison with olive, canola or groundnut oil.

TABLE 8. FATTY ACID COMPOSITION (%) OF COMMON DIETARY OILS

	SATS				MONOS		POLYS	
	Capric/ caprylic <10:0	Lauric 12:0	Myristic 14:0	Palmitic 16:0	Stearic 18:0	Oleic 18:1	Linoleic 18:2	Linolenic 18:3
Lauric and myristic-rich								
Palm kernel oil	8	48	16	8	2	15	3	-
Coconut oil	16	48	18	9	2	5	2	-
Butter fat	9	3	10	26	15	30	5	1
Palmitic and oleic-rich								
Palm olein	-	0.3	1.0	38	4	44	12	0.3
Oleic-rich								
Olive	-	-	1	11	3	77	7	0.6
Canola	-	-	-	5	3	61	22	8
Linoleic-rich								
Soya	-	-	-	11	1	22	54	7
Sunflower	-	-	-	7	5	21	66	-

acids vary in their effects and what is significant is the collective and cumulative actions of the fatty acids in the diet (*Table 9*). In this connection, both epidemiological and metabolic studies have suggested that a diet in which palm oil forms a major source of dietary fat does not lead to hypercholesterolemia (Sundram and Chandrasekharan, 2000).

The acyl groups located at the sn-1 and sn-3 positions are absorbed as free fatty acids while the acyl group at the sn-2 position is absorbed as a monoglyceride. Short and medium chain fatty acids are solubilized in the aqueous phase of the intestinal content, where they are absorbed, bound to albumin and transported directly to the liver via the portal vein. Long chain fatty acids (LCFA), however, are transported via the lymphatics and systemic circulation as chylomicrons before finally ending up in the liver. However, the location of LCFA on the glycerol molecule also can influence its metabolic destiny. Free palmitic and stearic acids in the sn-1 and sn-3 positions of their glycerol have low coefficients of absorption because of their melting points above body temperature and their ability to form calcium salts. Therefore, fats that have saturated LCFA at the sn-1 and sn-3 positions of triglycerides can exhibit different absorption patterns and metabolic effects compared to fats with palmitic acid or stearic acid located at the sn-2 position, which are absorbed more efficiently as monoglycerides (Rogers, 1988).

FACTORS AFFECTING SERUM CHOLESTEROL LEVELS

Those not familiar with recent developments in lipid nutrition generally think of saturated fatty acids in terms of their alleged capacity to raise blood cholesterol. Unfortunately, they have been conditioned to the fact that the major factor in raising cholesterol is saturated fats and so become oblivious to the reality that there are many factors which can affect cholesterol levels (*Table 10*).

SFAs if they are consumed in excess in conjunction with other dietary constituents will lead to a state of over nutrition and obesity. There is increasing evidence that obesity raises the LDL-C level in many people by over production of lipoprotein by the liver.

The effects of dietary fats/oils on serum lipids and lipoproteins generally reflect the collective influence of multiple fatty acids in the diet or food. A normal mixed diet contains many components that influence the blood cholesterol level in either direction. Variations in the ratio of polyunsaturated to saturated fatty acids (P:S) is well established as a mediator of serum cholesterol concentrations.

TABLE 9. THE FATTY ACIDS IN PALM OIL AND THEIR EFFECTS ON BLOOD CHOLESTEROL

Fatty acid	Effect on blood cholesterol	Amount in palm oil (%)
12:0 Lauric	↑	< 0.5
14:0 Myristic	↑	< 1
16:0 Palmitic	Now regarded as neutral	44
18:0 Stearic	Neutral	5
18:1 Oleic	↓	40
18:2 Linoleic	↓	10
18:3 Linolenic	↓	< 1

TABLE 10. FACTORS AFFECTING PLASMA LIPIDS/LIPOPROTEINS

1.	Diet	- Content and compositions of dietary fat; and - Carbon chain length, degree of saturation, position of first double bond, *cis-trans* configuration. Dietary cholesterol Dietary fibre Alcohol Energy balance, obesity
2.	Lifestyle factors.	
3.	Environment and genetics.	

The specific effect of PUFAs on serum cholesterol level is less than previously thought. Recent studies have failed to show any effect of PUFAs on serum total and LDL cholesterol levels beyond that which could be accounted for by the displacement of saturates from the diet. Isocaloric replacement of saturates by polyunsaturated fatty acids is predicted to lead to a fall in HDL cholesterol that is statistically though not biologically significant (Rogers, 1988).

The level of cholesterol in the diet may modify the extent of the change in serum cholesterol induced by the type of dietary fat. There is variability between individuals in the response of serum cholesterol levels to diet. The relation between fatty acid intake and serum lipoprotein levels may not be truly linear. Individuals vary in their responsiveness to saturated fatty acids. The relation between diet and serum lipoprotein may be influenced by genetic or environmental factors. These differences include other dietary components, age, degree of obesity and genetic differences in lipid metabolism.

Trans-Fatty Acids

Trans-fatty acids are unnatural isomers formed during the hydrogenation of polyunsaturated oils. During hydrogenation, there is a decrease in total unsaturation and an increase in the concentration of SFAs and some *trans*-fatty acids are usually produced. *Trans*-fatty acids have been associated with adverse changes in serum lipoproteins and increased risk for CHD. Epidemiological studies have indicated that increased consumption of *trans*-fatty acids increases the risk of CHD (Chandrasekharan and Yusof, 2000).

Dietary Fats and Cancer

Medium chain triglycerides, as opposed to polyunsaturated fats, have no growth promoting effects in tumor bearing animals. Many studies since the early 1920s have shown an association between consumption of unsaturated oils and the incidence of cancer.

Dietary Fats and Stroke

Gillman and colleagues (Gillman *et al*., 1997), using data from the Framingham heart study, have reported that lower levels of dietary fat are associated with an increased risk of ischaemic stroke. The authors reported a substantial trend in protection against stroke as intakes of total fat, monounsaturates and saturates increased. The impact of monounsaturates and saturates was similar in magnitude. No association was seen with polyunsaturates.

Dietary Fats and Hemostasis

The hemostatic system involves not only interacting processes for the formation of a stable clot (thrombus), platelet aggregation and blood clotting, but also a mechanism to dissolve the thrombus. Fatty acids have been reported to affect some of the processes. In recent times, there has been much interest in postprandial lipemia and thrombosis. Fats and oils of varying fatty acid composition do not differ with regard to the acute effects on plasma triacylglycerol levels and factor seven activity (Chandrasekharan, 2000).

Fatty Acids in Milk

The fat content in human milk is 3.5% on a volume basis, but it supplies 45% - 55% of the total energy (Rogers, 1988). Palmitic acid is the predominant SFA of human milkfat (27.3%). Despite this large proportion of a long chain poorly absorbed fatty acid, human milkfat is well absorbed. The high absorption has been attributed to the position of palmitic acid on the triglycerides of human milk.

Human milk (contains 15 mg dl^{-1} cholesterol) has the characteristics of an adult diet that is conducive to hypercholesterolemia and subsequently coronary heart disease (Rogers, 1988). The high fat content of human milk and infant formulae, exceeds that which is deemed prudent in diets for healthy individuals later in life.

CONCLUSION

Fats play an important role in the health of the body and the key is what kind and how much you eat. Further, the role of oils and fats must be seen in perspective with other dietary components and diet in turn with other environmental factors. Many concepts and studies published in journals and promoted vigorously often do not stand the test of time and are eventually rendered irrelevant by new studies, whereas some are flatly contradicted. By focusing our attention on the total nutrition picture, choosing a variety of food everyday from the different food groups in moderation, there is a good chance that we can meet the personal nutrition challenge and be well nourished. It makes sense that

dietary recommendations and perceptions about oils and fats have to change with time and the emerging evidence.

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