Original Article

Key role of dietary fats in coronary heart disease under progressive urbanization and nutritional transition

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The increased vulnerability to non-communicable diseases (NCD) of developing populations experiencing a demographic and epidemiological transitions to increased risk of NCD at a time when the battle against infectious diseases, is ongoing. Apart from population growth, the major attributes of developmental transition are confined to changes in occupational pattern in family structure, lifestyle, dietary practices and progressive ageing of population. The emergence of the NCD is significantly associated with changes in dietary pattern, in most of the countries. Coronary heart disease (CHD) is the leading cause of death in developed countries and the incidence is increasing in developing countries, including India. The disease needs awareness of the risk factors responsible for prevention. The purpose of this review is to present an overview of the role of dietary fats in growth and development and in health and disease. Although the causation of CHD is multifaceted and the risk factors associated in general are several, there are specific and important elements, such as dietary fats and lifestyle. Dietary fats are an important component as they serve a number of functions in the body. The minimum desirable and upper limits of fat intake have been given, based on recommendations of expert groups. Sources of different fats are made available worldwide and the production, consumption, storage, oxidation and nomenclature are being discussed in the light of health and disease. The relative essentiality of the omega-6 and omega-3 fatty acids is recognized in terms of pharmacologically active eicosanoid metabolism. Nevertheless, epidemiological, physiological and clinical studies have demonstrated that long-chain omega-3 fatty acids present in fish oils have quite diverse health benefits. Appropriate guidelines need to be recommended at a national level consistent with dietary habits. The ratios of balanced fatty acids, namely omega-11, omega-9, omega-6 and omega-3, should be worked out appropriately in ameliorating nutrition-related disease states. Any simple dietary modification that can lead to a substantial reduction in morbidity and mortality from CHD would be of great medical, social and economic benefit.

Key words: coronary heart disease, developmental transitions, dietary fats, dietary fish/fish oils, essential fatty acids, India, Orissa.

Introduction

Cardiovascular disease (CVD), particularly coronary heart disease (CHD), is one of the greatest epidemics that humankind has ever faced globally.[1] In the developed world, CHD accounted for 5.4 million deaths, which constituted 47% of all deaths, compared to 4.2 million deaths, which constituted 11% of all deaths in the developing world.[2] For the first time in history, analyses show that non-communicable diseases (NCD) now constitute a more significant contribution to ill-health throughout the world than do infectious diseases.[3] According to World Health Organization (WHO) estimates, major NCD today are responsible for at least 40% of all deaths in developing countries and 75% in industrialized countries, where CHD is the first cause of mortality. South Asian migrants (India, Bangladesh, Pakistan and Sri Lanka) have the highest incidence of CHD among all ethnic groups in the world.[4,5] By 2020, up to three-quarters of deaths in developing countries would result from NCD, and CHD would be the number one cause.[1,3]

Over the past two decades, there has been a noticeable awareness of NCD, particularly with CHD, as a major public health problem in India.[6] It is estimated that more than 40 million people in India are suffering from CHD, causing immense national loss in terms of productivity, morbidity and mortality.[7] South India shows higher rates of CHD, with a prevalence of 14%, than north India (10%), which is similar with the rates prevailing in the affluent south Asian to countries overseas.[8] It has been shown that urban Indian populations have a threefold greater prevalence. This situation is similar to the Europeans, in that positive correlation between dietary fat and CHD, more fat is consumed in urban areas than in the rural population.[9] Prevalence of CHD in rural India shows increasing trend from 1.7% in 1974 to 5% in recent years.[10] It is difficult to predict the lifestyle risk factors and, unfortunately, detailed studies that consider diverse diets and habits in India have not been done. This review aims to provide the association between dietary fats and NCD and their role in reducing CHD and other diet-related diseases.

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Developmental transitions

The global health transition, also referred to as the epidemiological transition, can be defined as the complex changes in patterns of health, disease and mortality that result from demographic and associated economic and social changes in a world population that is getting older.[11] In this transition, NCD replaces infectious diseases as the primary cause of morbidity and mortality. Nutritional transition is a sequence of characteristic changes in dietary pattern and nutrition intake associated with social, cultural and economic changes during the demographic transition.[12] Traditionally, the nutrition transition, although not named as such, is described as a change from traditional, indigenous, rural, high-fibre, low-fat diet that is eaten by poorer people to a more affluent, western type of diet, rich in animal fats and low in fibre, which is eaten by those who are better off economically.[13]

India is the second largest developing country in the world after China, with a population of 1 billion in 2000. It is an agricultural country with rapid population growth and the majority of the population (74%) live in rural areas. Rising population densities in the countryside push people towards the cities and urban growth is well ahead of the rural rates. The urban population was around 17% in 1950; it has now reached over 35% and is expected to exceed 40% within the next decade.[14] This process of urbanization is expected to continue in the decades to follow. It involves changes in occupation patterns, lifestyle, family structure and value systems. These changes are reflected in alterations to dietary practices and in levels of physical activity. In recent years, India is going through nutritional and epidemiological transition of shift in focus from the problems of malnutrition to those of overnutrition or affluent malnutrition.[15] Sizeable sections of populations, once poor, are now ascending the socioeconomic scale and are adding to the growing numbers of affluent urban middle class which may account for 200 million of the Indian population. The changes in dietary practices, physical activity levels and lifestyles associated with rising affluence induced by developmental transition contribute to the increasing prevalence of overweight/obesity.[16,17] The total food grain production in India had increased from 50.9 million tonnes in 1950 to 199.3 million tonnes in 1997 and the per capita food grain availability was maintained, despite population growth.[18] The changes in socioeconomic development over the years in India have brought about an improvement in the overall nutritional status of the country. The intake of dietary fat and animal food is increasing.[10]

Early diet

When *Homo erectus* emerged 1.7 million years ago, early humans were hominid seedeaters, diggers and grubbers, scavengers and hunter-gatherers.[19] Their eating patterns frequently ranged from feast to famine, consisting of half plant-based foods and the other half foods of animal origin. The fat content of Palaeolithic wild game was significantly lower than that in meat currently available.[20] The important features of changes during evolution were an increase in the proportion of dietary calories derived from fat and a shift from fats relatively high in unsaturated fatty acids, to those high in saturated fatty acids.[21] It is important to remember that humans make food choices based on disposable income, occupation, education, ethnicity, rural-urban residence, religious beliefs, nutrition knowledge and physiologic characteristics.[22] The development of farming resulted in major changes in food availability. The link between national income and the nutritional composition of diet has been superseded in recent years by increasing levels of fat in the diets of the poor. In today's affluent societies, the virtually limitless availability of dietary fat and calories and the ever-decreasing rates of physical activity predispose present-day humans to increased risk of CHD, diabetes, cancer, hypertension, obesity and other risks to health.[10,23]

Dietary fats

In the context of diet and nutrition, adequate amounts of fat are essential for normal health; they provide a concentrated source of metabolized energy and act as a vehicle for dietary supply and fat-soluble vitamins (A, D, E and K). Fats are hydrophobic and require less water for storage than either protein or carbohydrate. They impart palatability to food, as well as retarding the emptying of the stomach. Based on biological functions, fats can be separated into storage fats: mainly triglycerides accumulated in specific depots in the tissues and structural fats consisting of phospholipids and cholesterol. During digestion, absorption and transport, the fatty acids are delivered either as combined esterified form to triglycerides, phospholipids and cholesterol or as free non-esterified form stored in adipose tissue. The type of fat incorporated in cell membranes is determined to a significant degree by the type of fats consumed in the diet.[24] Fat consumption in excess of normal physiological requirement has been linked to an increase in the risk of obesity and CHD. According to present knowledge, the most important single dietary component responsible for CHD in human beings is dietary fat.[25] Several prospective studies provide consistent evidence of relation between fat intake and the risk of CHD.[26–29]

Nomenclature of fatty acids

Dietary fats are solids or liquids at room temperature composed of fatty acids of varying chain lengths and double bonds grouped as saturated (SFA), monounsaturated (MUFA) and polyunsaturated (PUFA). The principal fatty acids are mostly straight-chain aliphatic monocarboxylic acids with an even number of carbon atoms. Common names are mostly used for fatty acids.[30] For example, stearic acid (18 : 0) $CH_3(CH_2)_{16}COOH$ is systematically called octadeconoic acid, and erucic acid (22 : 1 omega-11) $CH_3(CH_2)_9 CH = CH(CH_2)_9COOH$ is docosenoic acid. In the nomenclature, the figure before the colon indicates the number of carbon atoms and that after the colon the number of double bonds in the molecule. The final number refers to the position of the first double bond counting from the methyl (CH_3 or Omega/W/n) end of the carbon chain.

Saturated fatty acids

In SFA, all carbon atoms are joined by single bonds with the exception of the carboxyl (COOH) group and all other valence positions are occupied by hydrogen. They are not essential nutrients and are readily synthesized in the body from acetate. In addition, SFA can stand oxidant stress, even at frying temperature, better than unsaturated fats. Dietary

SFA decreases the fluidity of cell membranes, thereby imparting the membranes greater physical stability. Fatty acids of short and medium chain lengths (C_4-C_{10}) are readily absorbed and rapidly transported to the liver and tissues. They circumvent the need for chylomicron formation, thereby saving energy. Fatty acids with a chain length above C_{12} are absorbed and acylated into glycerolipids in the intestinal mucosa, which are then assembled into chylomicrons and secreted into the blood. The type of fatty acids consumed has considerable effects on lipoprotein and apolipoprotein concentrations. In this regard, lauric (12 : 0), myristic (14 : 0) and palmitic (16 : 0) acids are hyperlipidemic, whereas stearic acid (18 : 0) has a negligible impact, perhaps because it is poorly digested and quickly desaturated to oleic (18 : 1 omega-9) acid.[31] These fatty acids increase plasma cholesterol by impairing low-density lipoprotein cholesterol (LDLC) uptake by the liver receptor mechanisms by impairing low-density lipoprotein (LDL) removal from circulation.[32] A high SFA diet has been reported to increase hepatic apoprotein A-1 levels.[33] This is the major apoprotein of high-density lipoprotein cholesterol (HDLC) and is cardioprotective. This rise has a negligible impact on risk association with the increase of LDL. Animal foods in India, such as meat, milk, ghee and butter, contain more SFA than MUFA and PUFA (Table 1).[34] American consumption of SFA declined by just 4% between 1960 and 1980, and this was associated with an almost 25% age-adjusted decline in CHD mortality.[28] The CHD rates tripled and doubled in Japanese migrants to the west coast of the United States and Hawaii, respectively. This change was paralleled by changes in the average levels of fat in the diet.[28,35]

Monounsaturated fatty acids

The fatty acids contained in most foods are predominantly MUFA having one double bond produced commercially in large quantities by heating vegetable oils. Here, stereochemical isomerization can occur because the sections of the molecule on any side of the double bond can lie either on the same (*cis*) or the opposite side (*trans*). These include palmitoleic acid (*cis* 16 : 1 omega-7), oleic acid (*cis* 18 : 1 omega-9), elaidic acid (*trans* 18 : 1 omega-9), erucic acid (*cis* 22 : 1 omega-9), brassidic acid (*trans* 22 : 1 omega-9) and cetoleic acid (*cis* 22 : 1 omega-11) having different biological properties. The major fatty acids, oleic, elaidic and stearic acids, are produced by hydrogenation process of linoleic and alpha-linolenic acids (ALA). Elaidic acid is a rigid, synthetically produced molecule that solidifies fats as it contains atherogenic properties such as saturation.[35] Generally most of the Indian cooking fats and oils are a rich source of MUFA (Table 2).[34,36,37] Conventional Indian diets are rich in ghee, a complete mixture of SFA and MUFA made by churning yoghurt, topping off the butter and evaporating water.[38] By the 1990s, it became apparent that the consumption of trans-fatty acids had unequally adverse effects on blood lipid levels in metabolic and epidemiologic investigations.[39,40] High intake of MUFA in Mediterranean countries is associated with a low incidence of CHD.[28,40–44] Monounsaturated fatty acids probably have a neutral effect on plasma lipids, but increase high-density lipoprotein (HDL) levels and are less prone to LDL oxidation than PUFA.[29,38,41] However, the strongest epidemiological evidence relating trans-fatty acids to the risk of CHD has been provided by many large prospective studies.[45–47]

Polyunsaturated fatty acids

A high proportion of PUFA in the diet is beneficial from the point of view of CHD. The naturally occurring PUFA in animal and man are all cis-configurations.They are heterogeneous, both chemically and physiologically, and are vital cellular membrane constituents. It has been found that substituting PUFA for SFA resulted in lipid lowering, due to the large space occupied within lipoprotein particles and fewer cholesterol esters in the lipoproteins.[48,49] Esters with PUFA seem to be more soluble and more easily mobilized than esters with SFA. The nonesterified PUFA stored in adipose tissue are selectively incorporated into the cells of tissues and are involved in four separate metabolic oxidative pathways: (a) the acylCoA or beta-oxidation leading to the oxidation of fatty acids into carbon dioxide and energy; (b) the monooxygenase pathway metabolized by omega-1 and omega-2 hydroxylation, and by omega-6, omega-9, omega-12 and omega-15 epoxidation; (c) the cyclo-oxygenase pathway produces prostaglandins and thromboxanes, and (d) the lipoxygenase pathway producing hydroxy-, hydroperoxy-fatty acids and leukotrienes.[50]

Desirable and upper limits of fats

Recommended dietary allowances (RDA) or intakes (RDI) are the intake of nutrients derived from diet which keeps nearly all people in good health, depending on age and physiological state. The Food and Agriculture Organization (FAO) and WHO are the expert groups on the minimum desirable and upper limits of dietary fat intakes. Their recommendations need to address chronic disease prevention.[30] An active individual with an energy balance may take up to 35 energy % (en%) from dietary fat, but SFA should not exceed 10 en%. Similarly, a sedentary individual should not consume >30 en% from fat, particularly SFA and from animal sources.[51] Diet comprising about 40-en% or more tends to lead to a positive energy balance in adipose tissue and obesity, which increases the risk of CHD. Fat energy ratio is the proportion of dietary energy derived from total fat and it ranges from 7 to 46% in different countries. According to FAO food balanced sheets (1988–90), Indian diets have a fat energy ratio of 15%, total availability of fat at 38 g/capita/day, with 27% of total fat derived from animal sources.[52]

Nutrient allowances for Indians were recommended by an Expert Committee of the Indian Council of Medical Research in 1988.[34,53] In designating the fat requirements, the total invisible fat content of cereal-based diets eaten in India and the minimum essential fatty acids (EFA) requirement of various groups were considered. The requirement of fat in the form of invisible (10 en%) and visible (20 g/day or 9 en%) has been recommended. For normal adults, dietary fat should supply at least 15 en%, and for pregnant and lactating women it should be at least 20 en%. The average ideal daily intake of EFA of a normal adult individual is placed at 3 en%, while the requirements during pregnancy and lactation are 4.5 en% and 5.7 en%, respectively. To obtain this level of EFA in the diet, the visible fat should be 15–25 g/day in terms of oils, which provide the required amount of EFA. However, upper

Table 1. Fatty acid content of animal products in India (g/100g edible portion)[58]

Animal products	Fat g/100g	Saturated fatty acid	Mono-unsaturated	Polyunsaturated fats	
				Linoleic	α-linolenic*
Beef	25.1	10.4	10.2	0.6	2.5
Mutton	14.8	5.8	6.5	1.0	0.9
Pork	35.0	11.4	16.2	3.8	1.9
Chicken	15.1	6.0	6.4	1.8	0.5
Egg (hen)	11.3	3.4	4.2	1.3	0.5
Fish(herring)	16.4	2.9	2.3	0.2	10.1
Buffalo milk	8.7	5.4	2.3	0.1	0.5
Cow milk	3.5	2.2	0.9	0.1	0.1
Goat milk	3.8	2.4	1.0	0.2	0.1
Human milk	3.2	1.5	1.0	0.3	0.2
Butter	80.0	50.0	20.1	1.8	4.2
Ghee	100.0	65.0	32.0	2.0	1.0

Values vary depending on the feed of the animals. *Includes alpha-linolenic acid and long-chain fatty acids.

Table 2. Fatty acid composition of Indian common cooking oils (g/100g)[58]

Fat/oil	Fatty acids				
	Saturated	Mono-unsaturated	Polyunsaturated		Predominant
			Linoleic	α-linolenic*	
Coconut	90	7	2	< 0.5	Saturated
Palm kernel	82	15	2	< 0.5	Saturated
Ghee	65	32	2	< 1.0	Saturated
Vanaspati	24	19	3	< 0.5	Saturated
Red palm oil	50	40	9	< 0.5	Saturated and Monounsaturated
Palm oil	45	41	0	< 0.5	Saturated and Monounsaturated
Olive oil	13	76	10	< 0.5	Monounsaturated
Groundnut	24	50	25	< 0.5	Monounsaturated
Rapeseed/mustard	8	70	12	10.0	Monounsaturated
Sesame	15	42	42	1.0	Monounsaturated and Polyunsaturated
Rice bran	22	41	35	1.5	Monounsaturated and Polyunsaturated
Cotton seed	22	25	52	1.0	Polyunsaturated
Corn	12	32	55	1.0	Polyunsaturated
Sunflower	13	27	60	< 0.5	Polyunsaturated
Safflower	13	17	70	< 0.5	Polyunsaturated
Soybean	15	27	53	5.0	Polyunsaturated

* Includes alpha-linolenic acid and long chain fatty acids.

limits of fat in the diets should not exceed 30 en% (>80 g/day) particularly in sedentary individuals. The desirable amount of linoleic acid (LA) to be consumed by children has also been placed at 3 en%. The ratio of LA to ALA in the diet of a normal adult person should be between 5:1 and 10:1. Since in India 10–15 en% of fat comes from invisible fat, visible fat should be kept below 20 en%. Thus, daily fat should be kept below 50 g/day. However, no RDA has been recommended for other long-chain omega-6 and omega-3 PUFA intake in a well balanced diet.[34]

Current production and consumption of fats

The amount of total dietary fat available worldwide was estimated to be 68 g/capita/day. In developed and developing countries, the average total available fat was 143 g/capita/day and 50 g/capita/day, respectively. The amount of fat consumed varies from one country to another. In Asia, dietary fat is predominantly of vegetable origin, elsewhere animal fats may constitute one-half to two-thirds of dietary fat intake. Since the production of edible oils in India is not sufficient to meet the growing demands of our population, a significant amount is imported from other countries.[54] The National Nutrition Monitoring Bureau in India carried out surveys during 1996–97 and found that the average consumption of fats and oils in different states of India varied in adults from a low of 13 g/capita/day in Orissa, to a high of 77 g/capita/day in Punjab. However, the average consumption of fats and oils was reported to be only 18 g/day in preschool children.[55,56] The structure of fats or oils is determined by the degree of their unsaturation; the greater the saturation, the harder and more solid the fat is. The fat present in cereals, pulses and vegetables (invisible fats) provides seven en% from fat.[53] The minimum visible fat requirement (fat derived through oils, ghee, butter and vanaspati) has been estimated to be 20 g/person/day.[37] An average Indian consumes about 13 g/day of visible fat (79%), ghee (3%) and vanaspati (18%). In rural India, average diet contributes about 22 g/day visible fat and accounts for 13 en%.[37] The

diet of poor people consists predominantly of cereals and even they contain a significant quantity of invisible fat and have a little visible fat. The invisible fat from cereals and pulses alone contributes as much as 6.0 en% in an adult diet that provides 8400kJ.[56] Vanaspati is used by Indian people as a substitute for ghee and has about 50% of trans-fatty acids. It is produced by hydrogenation of vegetable oils and has a long shelf-life without affecting the aroma and taste. The mean intake of trans-fatty acids in India is around 2.0 g/person/day.[56] Nearly 57% of the vanaspati produced is consumed in the four northern states of Punjab, Uttar Pradesh, Madhya Pradesh and Bihar with a daily consumption of 20 g/adult/day which provides 11 g/day of trans-fatty acids and amounts to an intake of 4 en%.[57]

Indian edible oils are a better source of EFA, their levels ranging from 10% in palmolein to 25% in groundnut oil, 50–70% in cottonseed, corn, sunflower, soyabean and safflower oils, while fats like ghee, coconut and hydrogenated oils are comparatively saturated and have a poor content of EFA (Table 2).[34,58] The content of invisible fat in many food materials have a relatively high LA (rice 50%, wheat 55%, bengalgram 65% and redgram 55%) and a low content (about 3%) of ALA, while spices have a particularly high content of ALA (5.4%). As a result, the average Indian diet is predominantly based on staple cereals (rice and wheat) accounting for 75–80% of dietary energy and some pulses, including the diets of poor income groups which carry sufficient LA levels and a proportion of (ALA) as well (Table 3). Therefore, the incidence of EFA deficiency is almost absent in the Inadian subcontinent. Several studies of such populations indicate that the EFA status reflected in the fatty acid components present in their blood is satisfactory.[24,59]

Table 3. Essential fatty acids in Indian plant foods (g/100g edible portion)[58]

Food source	Fat g/100g	Linoleic acid	α-linolenic* aicd
Cereals			
Rice	1.7	0.5	0.01
Wheat	2.9	1.1	0.17
Millets			
Blackgram	2.0	0.1	0.70
Ragi	1.5	0.3	0.05
Jowar	3.3	1.5	0.05
Maize	4.8	2.2	0.05
Bajra	5.5	2.2	0.13
Legumes and Pulses			
Bengalgram	1.7	0.1	0.70
Rajmah	2.2	0.4	0.70
Cowpea (Lobia)	2.8	0.8	0.50
Greengram dhal	1.7	0.6	0.22
Redgram	2.2	1.0	0.11
Lentil	2.0	0.8	0.16
Bengalgram dhal	6.9	3.5	0.20
Soya	2.0	8.0	1.00
Condiments and Spices			
Dry chillies	1.7	9.1	0.26
Cumin seeds	9.0	3.0	0.02
Coriander seeds	2.0	2.1	0.50
Fenugreek seeds	10.0	3.4	1.90
Vegetables†			
Green leaf	0.2	0.06	0.03
Other vegetables‡	0.7	0.04	0.20

*Includes alpha-linolenic acid and long-chain fatty acids; † amaranth, spinach, cabbage, ambat chukka; ‡ tomatoes, beans, lady finger, brinjals, bottle gourds, ridge gourds.

Processing and preservation of fats

Refining is a process of purifying fats and oils, which remove suspended toxic substances, free fatty acids, odour and colour. The fatty acid composition of the oil is not altered, but lose their characteristic taste. Heating of fats or oils during cooking oxidizes the fatty acids, with a variety of peroxides, aldehydes, ketones, monomers and polymers being formed. Trans-unsaturated fats are formed from cis-configuration during the heat treatment of oils, particularly de-odorization and deep frying process. These trans-fatty acids are found in ruminant fats because of bacterial actions in the rumen. The susceptibility of oil is proportional to the degree of unsaturation and inhibited by anti-oxidants. Foods prepared in these fats tend to become rancid quickly and prolonged or repeated heating of fatty acids produces toxic substances.[38] Another important, but often overlooked component of diet is the hydrogenation of liquid vegetable oils to semisolid fats which is used to increase the stability, making possible the long-term storage and wider distribution of a more palatable fat. This is done to protect fats from oxidation to diminish the problems of objectionable odours, flavours and adds texture to foods. These isomers are subsequently found in frying oils and deep-fried foods.[57,60]

Essential fatty acids

The essentiality of PUFA was first recognized in 1930; LA (18 : 2 omega-6) and ALA (18 : 3 omega-3) are EFA required for the normal growth and function of all tissues.[61] All the double bonds must be in the cis-configuration, the presence of even one double bond in the trans-position causes loss of EFA activity. Neither of these acids can be synthesised in the body and must therefore be obtained from the diet. Animals, including man, cannot insert double bonds in between the 3rd and 4th carbon atoms in the omega-3 series, and in between the 6th and 7th carbon atoms in the omega-6 series.

Within the body, the parent EFA (LA and ALA) chain length is extended by the addition of two carbon atoms at the carboxyl end. Linoleic acid is converted by delta-6-desaturation (removing two hydrogen atoms) and elongation (inserting an extra double bond), delta-5-desaturation and elongation, and desaturation. This leads to form a series of long-chain PUFA; gamma linolenic acid (18 : 3 omega-6), dihomo-gamma linolenic acid (DGLA, 20 : 3 omega-6) and arachidonic acid (AA, 20 : 4 omega-6) all having the omega-6 structure. Similarly, ALA is converted through the same enzymes to constitute a series of omega-3 family.[62] The predominant omega-6 PUFA is LA and AA, easily obtained from plant foods and vegetable oils. Green and leafy vegetables supply the ALA of omega-3 series. The ALA derived PUFA, eicosapentaenoic acid (EPA, 20 : 5 omega-3), docosapentaenoic acid (DPA, 22 : 5 omega-3) and docosahexaenoic acid (DHA, 22 : 6 omega-3) make up a large part of fish oils including even freshwater fish.[24,63,64] A diet containing fish and fish oils provides much more EPA and more DHA than would be formed from ALA by vegetables.[65] Red blood cell membranes deficient in EFA become stiffer than normal, do

not pass through capillaries so easily and are likely to lead to reduced tissue oxygenation. The differences in efficiency of elongation of LA and ALA in different individuals could be the basis of many pathophysiological conditions.[50]

Eicosanoids, the oxygenated derivatives of EFA containing 20-carbon atoms, notably DGLA, AA and EPA, are important intercellular signalling agents. They represent a heterogeneous family of compounds produced locally as and when required and then are rapidly destroyed. The conversion of AA to biologically active eicosanoids clarify many of the essential functions and their remarkable biopotency (at nanomolar to picomolar levels) helped to clarify many of the disease events.[50] Thus, it expands our understanding of the etiology of many chronic degenerative diseases to know how dietary PUFA may affect, accelerate or retard these disease states. They are involved particularly in cardiovascular, renal, inflammatory and pulmonary functions and in the protective roles of blood cells such as platelets, monocytes, macrophages and neutrophils. Eicosanoids modulate secretary smooth muscle (contraction or relaxation), and cascade-type reactions.[65]

Relative significance of omega-6 and omega-3 PUFA

Much attention has been focused recently on the omega-3 PUFA, which have metabolic effects that differ substantially from those of omega-6 PUFA and are quite effective in decreasing the levels of serum lipids.[66] The net effect of eicosanoids that are formed from omega-3 fatty acids (trienoic prostanoids and pentaenoic leukotrienes) is anti-atherogenic compared to that of omega-6 fatty acids (dienoic prostanoids and tetraenoic leukotrienes). They are less biologically active than corresponding products of AA and poor substrates for cyclo-oxygenase and lipoxygenase enzymes. The desaturase enzymes involved in membrane lipid incorporation and turnover have affinity for the omega-3 PUFA than the omega-6 fatty acids.[50] The presence of ALA in the diet can inhibit the conversion of the large amounts of LA. Due to the potent effects of omega-3 PUFA, the synthesis of eicosanoids by tissue is rigorously controlled and AA levels must therefore be kept low, so to avoid undesirable physiological effects that will not result from excess eicosanoids.[67] No mention has been made of the long-chain omega-3 PUFA (EPA and DHA) multifaceted actions. The overall evidence indicates that the omega-3 PUFA are overwhelmingly more important than the omega-6 PUFA.[66] A balance between omega-6 and omega-3 PUFA in the diet is important since each competes for the same enzymes and have different biological functions.

The possible protective relationship of omega-3 PUFA-enriched diet on human health and disease has been represented diagramatically (Fig. 1). Epidemiological and clinical studies have shown that consumption of fish rich in omega-3 PUFA reduces the incidence of CHD mortality rates.[68,71] Dietary fish oils have been investigated in term of their hypolipidaemic,[66,72] antithrombotic,[73–75] anti-arrhythmic,[76] antihypertensive,[77,78] anti-inflammatory[79] and anticarcinogenic[80] aspects. Fish consumed more than once a week is likely to provide a reduced risk of CHD.[81,82] It is unclear whether all the benefits seen in most, but not all, epidemiological studies are explained by the content of omega-3

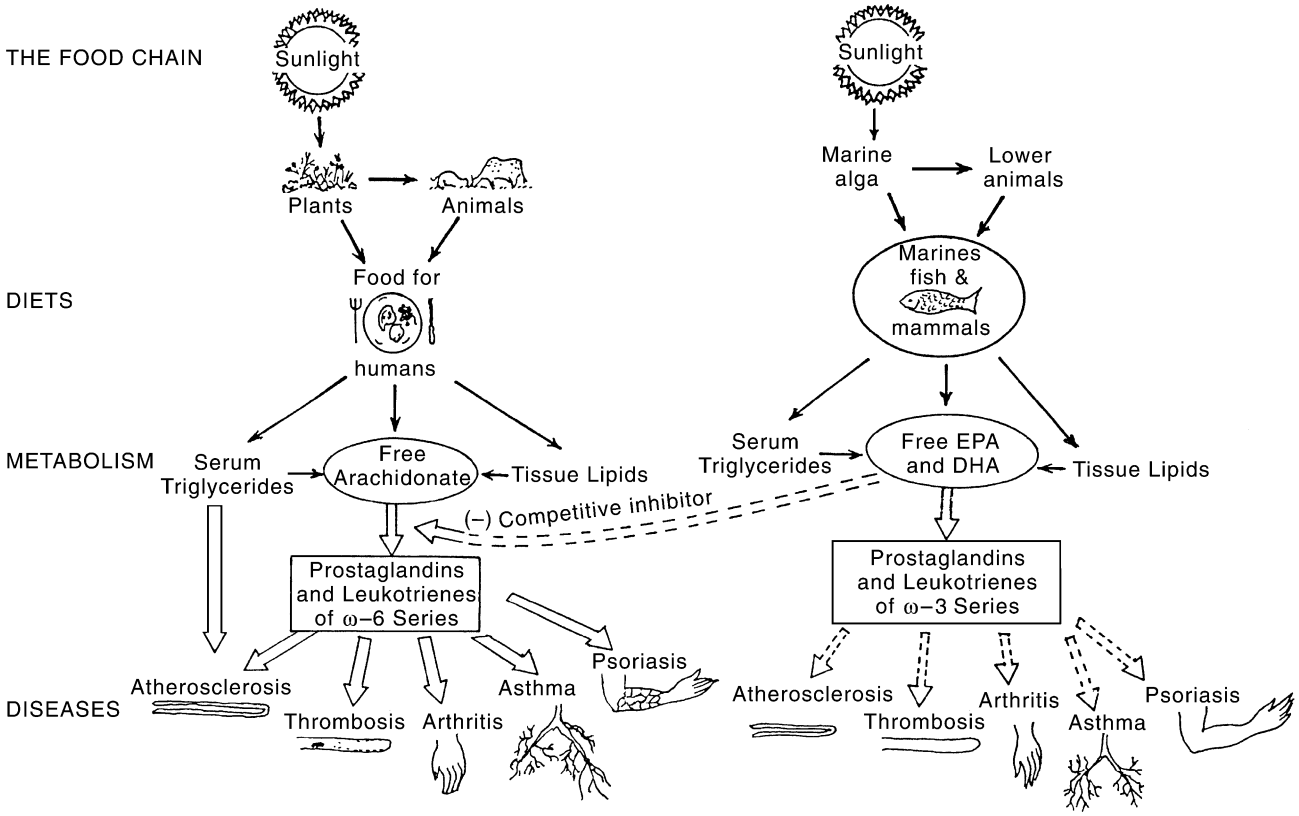

Figure 1. Relationships with human diseases of polyunsaturated fatty acids of w-6 and w-3 series in the food chain.
Less potent (dashed). ⟹ Highly potent.

PUFA.[83,84] In recent years, the potentiality of PUFA has gained considerable recognition to enrich various food sources using omega-3 PUFA.[85]

There is also a need for advocating the beneficial effects of unsaturated fats and their domestic consumption, which can prevent a number of diseases. The relation between dietary intake of specific types of unsaturated fatty acids and the risk of various degenerative diseases needs to be explored. As the years pass, it seems reasonable to place the quality of PUFA in a prominent position for dietary modifications. The RDI for omega-6 and omega-3 PUFA in place of RDA was recommended more recently at the National Institute of Health, Maryland, USA.[86] The adequate intake for a normal healthy adult is expected to meet the amount needed to maintain a defined nutritional state of a specific healthy population. Based on a 8400 kJ diet or 30 en%, the daily recommended normal and upper limits for LA are 4.44 g (2 en%) and 6.6 g (3 en%), respectively. Similarly, adequate daily intakes of ALA (2.22 g or 1 en%), long-chain omega-3 PUFA, EPA and DHA combined (0.65 g or 0.3 en%), individual EPA and DHA (<0.22 g or 0.1 en%) and trans-fatty acids (>2.0 g or 1.0 en%) are suggested. The saturated fatty acids should not exceed 8 en%, however, there is no recommended RDI for MUFA. The recommendations during pregnancy and lactating women are the same as those for adults, but ensure additional DHA intake of 300 mg/day. The composition of the infant formula/diet is also suggested based on both the growth and neural development of formula-fed infants in a manner similar to those of breast-fed infants.

Indian populations are comprised of about 4000 endogamous groups, stratified into different castes and tribes based on sociocultural and traditional practices. Each ethnic group has its own lifestyle and food habits that are inseparable. As populations grow older and the ratio of deaths due to chronic diseases versus infectious diseases increases, it is important to determine the extent to which adults are experiencing increased risk from NCD.[87] The challenges for developing countries like India are the need to design appropriate, multifaceted and multisectorial programmes that address under- and overnutrition jointly. There are prominent features of nutritional transition associated with increase in dietary intake of fat and decrease in the intake of complex carbohydrates and fibres accompanied by a significant decrease in physical activity. The replacement of SFA must be encouraged with other protective nutrients, since Indian diets are rich in LA particularly derived from invisible fat. It is very difficult to determine the accurate intake of fatty acids in a general population, and the cooking habits practised by these cultural groups vary. However, projections made by diet surveys in India show an alarming increase in cholesterol-raising SFA consumption and is a cause of concern for diet-related diseases particularly CHD in the next millennium.

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