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# Polyunsaturated Fatty Acids In Health And Nutrition

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Polyunsaturated fatty acids (PUFA) comprise the parent essential fatty acids (EFAs) and their long chain more unsaturated derivatives (LCPUFA). PUFA are indispensable for human development and health. EFAs cannot be synthesised denovo in humans and therefore they need to be consumed as part of the diet.

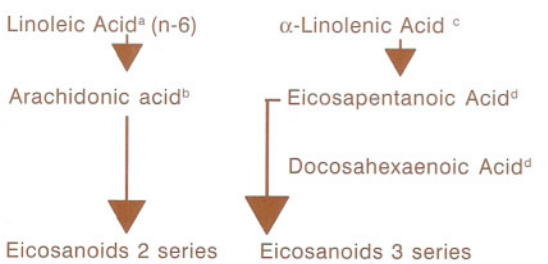
There are two classes of PUFA namely n-6 and n-3, synthesised from linoleic (LA) and a-linolenic (ALNA) acids, respectively. Since the time the essentiality of EFAs was established in 1929, while studies on nutritional and health effects of n-6 PUFA received wide attention, studies on the effect of n-3 fatty acids attracted relatively little notice. However, observations on Eskimos[1] in 1970s sparked great interest in n-3 PUFA research and today we know that both n-6 and n-3 PUFA have important roles in growth and development and in prevention of diet-related chronic diseases.

**Metabolism and biological effects of PUFA:** LA and ALNA are desaturated and elongated in the human body cells to LC n-6 PUFA and LC n-3 PUFAs, respectively (Figure 1). In this conversion LA and ALNA compete for the same enzymes, and ALNA has higher affinity than LA. The most important LC n-6 PUFA are dihomogammalinolenic acid (DHGLA) and arachidonic acid (AA); these are the precursors of eicosanoids of '1' and '2' series, respectively[2]. The important LC n-3 PUFA are eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), the latter can be retroconverted to EPA. EPA is the precursor of eicosanoids of '3' series. The primary EFAs, LC n-6 and LC n-3 PUFA, are important structural and functional membrane components and therefore essential for the formation of new tissues.

The levels of n-6 PUFA and n-3 PUFA in membranes and their ratio affect a range of biochemical processes such as membrane fluidity, nutrient transport, activity of membrane bound enzymes, receptor function, either directly or via production of eicosanoids and leukotrienes. In response to various stimuli, AA and EPA of membrane phospholipids compete for the cyclo-oxygenases and lipoxygenases and are converted to eicosanoids (for example, prostaglandins, thromboxanes and leukotrienes) of '2' and '3' series, respectively (Figure 1). These compounds have important bio-regulatory functions. Eicosanoids derived from AA have opposing metabolic properties to those derived from EPA and therefore a balanced intake of n-6 and n-3 PUFA is essential for health[2].

**PUFA and coronary heart disease (CHD):** In the development of CHD both atherosclerosis and thrombosis are known to play a part. In the past, the major focus for preventing CHD by diet was on lowering serum total cholesterol and its principal carrier LDL cholesterol by reduction of total fat, saturated fat and cholesterol, and increase in PUFA; dietary advice simply consisted in substituting vegetable oils (safflower, corn, etc) for animal fats. While vegetable oils are good sources of LA, not all of them provide ALNA (except rapeseed and soyabean). In the pathogenesis Of CHD several metabolic risk factors

**FIGURE 1**
**Metabolism of PUFA**




[a] *plan foods and vegetable oils;*
[b] *animal foods;*
[c] *mustard/rapeseed, soyabean, pulses, green leafy vegetables, walnuts, fenugreek and wheat;*
[d] *fish, fish oils and algae*

## CONTENTS

such as hypertension, low serum HDL cholesterol, high oxidative susceptibility of LDL, poor antioxidant status and insulin resistance (syndrome X) are involved. LD, by itself, will not be able to address all these factors[3].

The pioneering studies of Bang and Dyerberg[1] showed that the low rates of mortality from CHD despite high fat intake in Eskimos could be due to relatively large intakes of LC n-3 PUFA from marine foods in their diets. Since then other epidemiological studies confirmed that regular consumption of fish reduces the risk of CHD. When humans consume fish or fish oils the ingested EPA and DHA partially replace the n-6 PUFA (specially AA) in cell membranes and lead to:

- decreased production of thromboxane $A_2$, a potent platelet aggregator and vasoconstrictor, prostaglandin $E_2$ and leukotriene $B_4$, inducers of inflammation and leukocyte chemotaxis and adherence, and
- increased concentration of prostacyclin $I_3$ (both $PGI_2$ and $PGI_3$ are equally active vasodilators and inhibitors of platelet aggregation) and leukotriene $B_5$, a weak inducer of inflammation and chemotactic agent.

Therefore, increasing n-3 in membranes produces anti-inflammatory, antiaggregatory and vasodilatory effects[2,3]. n-3 fatty acids have also been shown to have hypotriglyceridemic effects, decrease postprandial lipemia and prevent ventricular arrhythmias (which lead to sudden death). Interestingly, dietary PUFA have been shown to regulate lipolysis and lipogenesis by modulating the expression of genes encoding for key metabolic enzymes and also play a central role in the regulation of adipocyte related genes[4]. Further, it has been shown that insulin action in skeletal muscle and adipose tissue can be modified by PUFA composition of membranes[5]. Since the n-3 acids from fish and fish oils down-regulate several of the factors which have atherogenic potential and enhance factors which have antiatherogenic potential, these contribute to cardiovascular health. The current emphasis, thus, is that human diets should provide:

- low SFA, trans fatty acids (present in hydrogenated oils) and cholesterol,
- adequate absolute levels as well as an optimal balance of n-6 and n-3 fatty acids, and
- adequate antioxidants.

The low SFA and cholesterol would lower LDL cholesterol; antioxidants would prevent oxidation of LDL cholesterol; and balance of n-6 and n-3 acids in membranes would ensure optimal function of major defense mechanisms of the human body (blood coagulation, inflammation, immunological) and also ensure efficient insulin action. The current recommended range for PUFA intakes in humans are: LA 3-8 en per cent, ALNA 0.5-2.5 en per cent – ratio of LA/ALNA (n-6/n-3) being 5:1-10:1[2].

**PUFA in foetal growth and development:** The supply of AA and DHA is crucial during foetal development and after birth until biochemical development in the brain and retina is completed[6]. During pregnancy, the foetus depends completely on the maternal source of PUFA (diet and stored lipids) and after birth, AA and DHA are obtained from breast milk. Biochemical PUFA nutritional status, particularly of DHA decreases after pregnancy but return to the normal status is slow. Therefore, in women with less birth spacing, the maternal neonatal DHA status is poor and this may lead to lower neonatal PUFA status. Depletion of maternal DHA reserves during pregnancy and lactation has been suggested as one possible cause of postpartum depression. Further, neonatal DHA status correlates positively with birth weight. Some workers therefore recommend DHA supplementation during pregnancy.

DHA is one of the abundant fatty acids present in the brain, in the other neural tissues and in the retina. It has been shown that deficiency of n-3 fatty acids in infant monkeys resulted in impairment of visual activity, abnormal electroretinograms and disturbances of cognition. The observation that the impairment of visual activity continued even after repletion of DHA suggests that the damage can be permanent. Some of these functional changes have been observed in infants fed milk formulae devoid of n-3 fatty acids.

Human milk provides significant amounts of EFAs AA and DHA. However, the contents vary with dietary PUFA intake during pregnancy and lactation. Although most infant formulae contain LA and ALNA, they are devoid of AA and DHA. However, the capacity of newborns, particularly of the premature, to form AA and DHA from precursors is very limited. It is therefore important to ensure adequate intake of n-6 and n-3 fatty acids, both during pregnancy and lactation.

In infant formulae, apart from ensuring adequate levels of LA and ALNA and their ratio, the addition of both AA and DHA in amounts similar to the levels present in breast milk is strongly advocated. A recent recommendation for infant formulae/diet is as follows: (percentage of total fatty acids): LA 10, ALNA 1.5, AA 0.5, DHA 0.35, and EPA <0.1 (since higher levels of EPA may antagonise AA and interfere with growth)[6]. Recent evidences indicates that low birth-weight babies may be at a greater risk of developing CHD (particularly insulin resistance and its sequalae) in later life as compared to those born with normal birth weight[7]. Thus, optimal PUFA intake is essential at all stages of life – conception, growth and development – in prevention for chronic diseases in adults.

## THE INDIAN CONTEXT

A major nutrition-related problem in India is chronic undernutrition associated with low fat intake. Undernutrition starts even at the time of conception (because of maternal undernutrition) and a large number of infants are born with low birth weight. Alongside, with industrial and economic development, and urbanisation, there is increase in diet-related chronic diseases.

The data presented in Table 1 give PUFA contents of some commonly consumed plant foods. It is found that in all plant foods, LA is the major PUFA. Among cereal and millets, wheat and bajra have higher amounts of ALNA. On an average, legumes and pulses provide more ALNA than cereal and millets. In fact, a few of these (black gram, *rajmah,* soyabean and cowpea) furnish LA/ALNA ratio below 2.0 and can therefore contribute significantly to increasing dietary ALNA. Depending on the choice and amount of cereal/millet and legumes/pulses consumed, Indian diets provide ~5 g LA (2 en per cent) and ~0.6 g ALNA from these food items.

On an average, green leafy veg-

**TABLE 1**
**Fat and PUFA Contents in Plant Foods (g/100 g)**

| Name | Fat | LA | ALNA |
|---|---|---|---|
| ***Cereals*** | | | |
| Wheat | 2.9 | 1.10 | 0.17 |
| Rice | 1.7 | 0.50 | 0.01 |
| ***Millets*** | | | |
| Bajra | 5.5 | 2.2 | 0.13 |
| Jowar | 3.3 | 1.5 | 0.05 |
| Ragi | 1.5 | 0.3 | 0.05 |
| Maize | 4.8 | 2.2 | 0.05 |
| ***Legumes*** | | | |
| Rajmah (black) | 2.2 | 0.4 | 0.73 |
| Rajmah | 2.2 | 0.5 | 0.70 |
| Soyabean | 20.0 | 10.4 | 1.40 |
| Cowpea | 2.8 | 0.8 | 0.48 |
| Bengal gram | 6.0 | 2.7 | 0.18 |
| Peas | 2.1 | 0.8 | 0.15 |
| ***Pulses*** | | | |
| Black gram | 1.7 | 0.1 | 0.70 |
| Green gram | 1.7 | 0.6 | 0.20 |
| Lentil | 2.0 | 0.8 | 0.20 |
| Bengal gram | 6.9 | 3.5 | 0.20 |
| Red gram | 2.2 | 0.9 | 0.10 |
| ***Green leafy vegetables*** | | | |
| a* (≥0.2 g ALNA) | 0.8 | 0.09 | 0.35 |
| b* (<0.2 g ALNA) | 0.3 | 0.04 | 0.08 |
| ***Vegetables*** | | | |
| c* (Beans) | 0.2 | 0.06 | 0.03 |
| d* (Other vegetables) | 0.2 | 0.06 | 0.02 |
| ***Fresh fruits*** | | | |
| e* (≥0.03 g ALNA) | 0.5 | 0.08 | 0.04 |
| f* (<0.03 g ALNA) | 0.3 | 0.04 | 0.01 |
| ***Spices*** | | | |
| Fenugreek seeds | 10 | 3.4 | 1.87 |
| Cumin seeds | 9.4 | 2.1 | 0.48 |
| Dry chillies | 17.2 | 9.0 | 0.26 |
| Turmeric | 0.7 | 0.2 | 0.05 |
| Coriander seeds | 20.3 | 3.0 | 0.02 |
| ***Oilseeds*** | | | |
| Mustard | 40 | 5 | 2.5 |
| Sesame | 40 | 16 | 0.4 |
| Groundnut | 40 | 10 | 0.2 |
| Coconut | 40 | 0.6 | - |
| ***Nuts*** | | | |
| Walnuts | 68 | 40 | 7.6 |
| Cashew nuts | 50 | 9 | 0.3 |
| Almonds | 56 | 8 | 0.3 |
| Pistachio | 30 | 9 | 0.4 |

*Values are mean of three market samples of each food items; a* Agathi, colocasia, drumstick leaves and fenugreek leaves; b* Amaranth, onion leaves, gogu, ambat chuka, paruppu keerai, cauliflower, malayu, ceylon pasali, spinach, cabbage, pudina and curry leaves; c* Scarlet runner, cluster beans and French beans; d* Lady's fingers, ridge gourd, bottle gourd, tomatoes and brinjal; e* Mango two varieties (Alphanso, Rasalu), plum, papaya and grapes*
*Source: Ref 8 and Ghafoorunissa (unpublished).*

etables furnish more ALNA than other vegetables and fresh fruits. Four green leafy vegetables (agathi, colocasia, drumsticks and fenugreek) provide 10 times more ALNA than vegetables and fruits and two times more than most of the legumes and pulses. Fenugreek, and oilseeds such as mustard, perilla, flaxseeds and walnuts are rich sources of ALNA.

Among animal foods, milk (cow and buffalo) is a poor source of PUFA, eggs provide both LA and ALNA, mutton and chicken are good sources of AA. However, the n-3 PUFA content in animal foods can be increased by inclusion of seeds/oils such as perilla, canola, soyabean (ALNA), algae and fish meat, or fish oils (n-3 fatty acids) in animal feeds. Fish is a good source of n-3 fatty acids, fish containing low fat (<2 per cent), medium fat (2-5 per cent) and high fat (>5 per cent), furnishes about 0.2, 0.9 and 1.2 g n-3 fatty acids/100 g muscle, respectively.

Common edible oils and fats vary considerably in their percentage of LA (Table 2) and only mustard/rapeseed, soyabean have appreciable levels of ALNA. Depending on the amount and choice of cooking oils (groundnut, sesame, safflower, sunflower) consumed in the total diet, LA levels (visible fat + invisible fat from all foods) range between 9-17 en per cent (recommended = 3-8 en per cent) and ALNA ranges between 0.1 - 0.3 en per cent (recommended = 0.5-2.5 en per cent), the ratio of LA/ALNA (n-6/n-3) in Indian diets varies between ~30:1-70:1 (recommended 5:1 - 10:1)[8]. However, when oils such as ricebran, soyabean and rapeseed/mustard are consumed, the levels range between LA, 4-12 en per cent, ALNA, 0.6-1.8 en per cent and LA/ALNA ratio = 2:1-12:1[8]. In the US[9] and Europe[10], the dietary n-6/n-3 ratio is ~20:1 – 30:1 but in Japan[11] it is ~4:1. The higher n-3 PUFA intake in Japan is because of high consumption of fish, soyabean and canola oils. In human studies Emken *et al*[12] showed that the conversion of deuterated ALNA to LC n-3 PUFA was reduced by ~50 per cent when dietary intake of LA was increased from 4.7 to 9.3 per cent of energy. Data on plasma and platelet fatty acid compositions of apparently normal men suggested low n-3 PUFA nutritional status as compared to populations in Japan, the US and Europe. The ratio of AA/EPA is high in Indians as compared to other populations[8,13]. However, Indians who regularly consume fish have a good n-3 PUFA status.

An important question that needs to be addressed then is, can low intakes of ALNA and high ratio of LA/ALNA be a yet another nutritional factor contributing to undernutrition *in utero* and during early growth and development (resulting in low birth-

**TABLE 2**
**Approximate PUFA Composition of Fats and Oils (% of Total Fatty Acids)**

| Name | LA | ALNA | LA/ALNA (n-6/n3) |
|---|---|---|---|
| ***Oils having high SFA*** | | | |
| Coconut, Ghee, Vanaspati, Palm kernel | 2-3 | < 0.5 | 4 |
| ***Oils having low LA*** | | | |
| Palmoil, Olive | 10 | < 0.5 | 20 |
| ***Oils having moderate LA*** | | | |
| Groundnut | 25 | < 0.5 | 50 |
| Rice bran | 35 | 1.5 | 23 |
| ***Oils having high LA*** | | | |
| Sesame , Cottonseed, Corn | 40-55 | 1 | 40-55 |
| Sunflower, Safflower | 60-70 | < 0.5 | 120-140 |
| ***Oils having ALNA*** | | | |
| Mustard/rapeseed | 12 | 10 | 1.2 |
| Soyabean | 50 | 5 | 10 |
| Canola | 20 | 8 | 2.5 |
| ***Unconventional oils*** | | | |
| Flax seed (linseed) | 13 | 53 | 0.2 |
| Perilla | 16 | 59 | 0.3 |

weight) and consequently increase the susceptibility to the widely prevalent insulin resistance syndrome and its metabolic sequelae which lead to CHD in Indians? Studies in this direction should receive top priority.

From the foregoing discussion, it is clear that there is a need to increase n-3 PUFA intake in Indians. However, the levels required for optimal health benefits would depend on whether ALNA, the precursor (plant) or LC n-3 PUFA, the biologically active end product (fish and fish oils) is the source of n-3 PUFA and also on the level of LA (plant foods) and AA (animal foods) in the diet.

Metabolic studies were conducted in Indian men consuming cereal-based vegetarian diets to investigate the efficacy of using ALNA in comparison to LC n-3 PUFA for increasing LC n-3 PUFA and producing antithrombotic effects. It was found that at a level of 16 g (6 en per cent) LA, 3.7 g (1.4 en per cent) ALNA could increase LC n-3 PUFA to the same extent as 1 g LC n-3 PUFA. This works out to an equivalence of 11 g ALNA for 1g LC n-3 PUFA[14]. Therefore, to ensure optimal intakes of LA, ALNA and their balanced ratio, decrease in LA and increase in ALNA from oils has been suggested. The approximate combination of oils has also been worked out so that the consumer has the option to select oils according to their pattern of purchase and culinary practices[13]. Currently, 'in home' study is in progress to evaluate the long term health benefits of increasing ALNA from the recommended oil combinations in comparison to single oils. Further, regular consumption of foods containing high levels of ALNA is advocated. In fact, use of green leafy vegetables every day will not only increase ALNA intake but also provide several other nutritional benefits. However, non-vegetarians have the choice of eating fish to accomplish a good n-3 PUFA status – fish also provides good quality proteins and micronutrients.

A specific recommendation to eat fish once or twice a week, before conception, during pregnancy and lactation and breast feeding may significantly contribute to growth and development of babies and ensure good health of both mother and baby. Since vegetarians do not consume fish, it is essential to increase ALNA in the diets of pregnant and lactating women. Studies are needed to determine the desirable levels of ALNA or LC n-3 PUFA to be included in diets of pregnant and lactating women to prevent low birth weight and ensure adequate DHA content in breast milk. The use of value added products of fish and use of flaxseed and perilla seeds in processed foods, fast foods and in the fortification of cereals (food-food fortification) will provide good source of n-3 PUFA and several nutrients which are limiting in cereal-based low fat diets contribute to improving the nutritional and health status of Indians.

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