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1.3 Nutritional Needs

1.3.5 Fats

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Key Words

Lipids · Essential fatty acids · Linoleic acid · α-Linolenic acid · Long-chain polyunsaturated fatty acids · Arachidonic acid · Docosahexaenoic acid · Saturated fatty acid · *Trans* fatty acid

Key Messages

- Optimal lipid nutrition begins in fetal life with adequate n–3 to n–6 fatty acid and preformed long-chain polyunsaturated fatty acid (LCPUFA) supply through the maternal diet and PUFA metabolism
- Breast milk from mothers consuming a balanced diet provides the best source of bioavailable lipids for term neonates
- Linoleic and α-linolenic acids are essential fatty acids; in addition, LCPUFA are important for lifelong health
- LCPUFA in the diet and the mother's genetic control of metabolism are important for visual and cognitive development in the first months of life, after which they contribute to lifelong health
- *Trans* fatty acids interfere with LCPUFA metabolism, affect lipoprotein cholesterol regulation and promote cardiovascular disease
- The balance between dietary n–3 and n–6 fatty acids is important to promote lifelong health, reducing the disease risk linked to allergic and inflammatory responses

Introduction

Fats are the main source of energy for infants and young children, and n–6 and n–3 fatty acids are essential for normal growth and development. Fat-soluble vitamins (A, D, E and K) require dietary lipids for absorption. Fats provide flavor and texture to foods, and thus affect taste and acceptability of diets as well as gastric emptying and satiety. Membrane lipid composition in part defines the functional properties of membranes (fluidity, transport properties, receptor activity, uptake and release of substances, signal transduction and conduction, and ion flows). Fatty acids can also affect gene expression directly or by regulating transcription factors that affect the expression of multiple other genes (i.e. peroxisome proliferator-activated receptors). Dietary lipids provide structural components for brain and retinal structures, cell membranes and transport of lipid components in plasma, and they form the only true energy store of the body (adipose tissue). Fats and oils are key dietary factors affecting cardiovascular risk, obesity and diabetes. Linoleic acid (LA; C18:2n–6) and α-linolenic acid (LNA; C18:3n–3) are essential; they serve as precursors of the long-chain polyunsaturated fatty acids (LCPUFA) such

as arachidonic acid (AA; C20:4n–6) and docosahexaenoic acid (DHA; C22:6n–3). Dietary lipids and mother and child genetic variation in fatty acid desaturase and elongase enzymes determine the balance between n–3 and n–6 effects. Neural cell phospholipids in the retina and cerebral cortex are rich in DHA, while vascular endothelia are rich in AA. LCPUFA are precursors of eicosanoids (C20) and docosanoids (C22), which act as local and systemic mediators for clotting, immune, allergic and inflammatory responses; they also affect blood pressure as well as vessel and bronchial relaxation and constriction. The dietary balance of n–6 and n–3 fatty acids can have profound influences on these responses, modulating the onset and severity of multiple disease conditions (allergy, atherosclerosis, hypertension and diabetes).

Lipids have long been considered as part of the exchangeable energy supply for infants and young children; thus, of primary concern has been the degree to which dietary fat is absorbed as an important contribution to the energy supply during early life.

Fats in the First Year of Life

High-fat formulas (40–60% of energy), characteristic of infant feeding, contribute to the energy density of the diet required to support rapid weight gain, and especially to the fat accumulation observed over the first year of life. This has been traditionally considered a desirable trait, considering the increased risk of infection and potential dietary inadequacy after 6 months of life. However, the need for this fat gain for survival has been reexamined as we presently face an environment that promotes energy excess and thus increases the risk of obesity and chronic diseases later in life [1, 2]. The 2006 WHO Growth Standards, based on predominant breastfeeding for the first 6 months of life, suggest a leaner model of growth for the 2nd semester of life (see Chapter 4.1). In addition, the 2010 Food and Agriculture Organization/WHO recommendation on fat has reduced amounts of total fat after 6 months and even more after 2 years of life [3].

Essentiality of PUFA and LCPUFA

The essentiality of LA for human nutrition was identified about 70 years ago. In the 1980s, n–3 fatty acids were found to be essential for humans, considering the altered visual function in children receiving parenteral lipids high in n–6, which was reversed by provision of LNA, the n–3 precursor found in soy oil. Studies on preterm infants postnatally fed LCPUFA revealed that those receiving no DHA had altered electric responses to light and significant delays in maturation of visual acuity, which were only partially improved by LNA [4, 5]. These studies served to establish a need for LNA and suggested that, at least in preterm infants, DHA was also needed. Further studies have established a need for n–3 fatty acids in term infants, with some but not all studies demonstrating a benefit from receiving preformed DHA. Several stable isotope studies using labeled LA and LNA have demonstrated a limited and highly variable capacity to convert these precursors into the corresponding LCPUFA, i.e. AA and DHA, supporting the view that the latter may be considered conditionally essential during early life [2]. Preterm and term formulas are now supplemented with AA and DHA. Higher levels of DHA in formulas and breast milk should be needed for extremely preterm infants [6, 7].

LCPUFA can affect adipogenesis, but findings on their short- and long-term effects on body composition among trials using varied supplemented n–3 LCPUFA formulas are contradictory [8]. DHA should be considered essential for the treatment of certain chronic diseases, such as aminoacidopathies, and other inborn metabolic disorders because of dietary restrictions in some diseases, or because metabolism of LCPUFA is affected, as in peroxisomal diseases [9].

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Table 1. Composition of commonly used vegetable oils

Source of oil	Fat, g	Saturates	Mono-unsaturates	Poly-unsaturates	n–6 PUFA	n–3 PUFA	Cholesterol, mg
Canola	100.0	7	59	30	20	9.3	0
Corn	100.0	13	24	59	58	0	0
Sunflower	100.0	10	19	66	66	0	0
Rapeseed	100.0	7	56	33	22	11.1	0
Soya	100.0	15	43	38	35	2.6	0
Olive	100.0	14	74	8	8	0.6	0
Vegetable solid fat	100.0	25	45	26	3	1.6	0
Animal fat lard	100.0	39	45	11	10	1	95
Milk fat	81	50	23	3	21	1.2	219

Table 2. Recommended fish as a source of eicosapentaenoic acid and DHA

High levels of eicosapentaenoic acid and DHA (>1,000 mg per 100 g fish)	Herring Mackerel Salmon Tuna – bluefin Greenland halibut
Medium level (500–1,000 mg per 100 g fish)	Flounder Halibut Tuna – canned white
Low level (≤300 mg per 100 g fish)	Tuna – skipjack Tuna – canned light Cod Catfish Haddock

Table 3. Contribution of various foods to *trans* fats consumed

Food group	% of total
Cakes, cookies, crackers, pies, bread, doughnuts, fast-fried chicken, etc.[a, b]	40
Animal products	21
Stick margarine	17
Fried potatoes	8
Potato chips, corn chips, popcorn	5
Household shortening	4
Breakfast cereals, candy	5
Soy oil	2

United States Department of Agriculture analysis reported 0 g of *trans* fats in salad dressing.
[a] Includes breakfast cereals and candy.
[b] Unless specifically modified and labeled.

Artificial infant formulas based on mixes of vegetable oils (coconut, palm, corn, soy, sunflower and safflower) provide LA- or oleic acid-rich contents, and some LNA from soy oil, attempting to mimic the composition of human milk (table 1). Coconut oil fractions rich in medium-chain triglycerides are used in an effort to promote absorption, especially in the feeding of preterm infants and those with fat malabsorption syndromes, since C8–10 fatty acids are absorbed directly from the intestinal mucosa passing to the portal vein [9]. Over recent years DHA or DHA + AA have been added to many infant formulas. However, it is nearly impossible to fully replicate the unique fat composition and structure of human milk lipids. Human milk lipase activity further contributes to the improved fat digestibility of human milk. After 6 months, with the introduction of solid complementary foods, egg yolk, liver and fish can provide preformed DHA and AA (table 2) [2].

Lipids in Human Milk

Breast milk provides a ready source of both precursors and long-chain n–6 and n–3 derivatives, and is considered sufficient in these nutrients, provided mothers consume a nonrestrictive diet. The ac-

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Table 4. Fat supply for children older than 2 years for the prevention of nutrition-related chronic diseases (based on Food and Agriculture Organization references)

Dietary component	Amount
Total dietary fat intake	25–35% of energy, depending on activity
Saturated fatty acids	<8% of energy (mainly C12, C14 and C16)
PUFA	5–15% of energy
n–6 PUFA	4–11% of energy
n–3 PUFA	<3% of energy
Eicosapentaenoic acid + DHA	100–300 mg, depending on age
n–6:n–3 ratio	5:1 to 10:1
Monounsaturated fatty acids	No restriction within limits of total fat
Cholesterol	<300 mg/day
Antioxidant vitamins	Generous intake desirable
Potentially toxic factors[1]	
Trans fatty acids	<1% of total energy
Erucic acid[2]	<1% of total fat
Lauric and myristic acids	<8% of total fat
Cyclopropenoids	Traces
Hydroperoxides	Traces

[1] Limit processed foods, hard fats and hard margarine as a practical way to limit intake of saturated and *trans* fatty acids.
[2] Use only rapeseed oil derived from genetic varieties low in erucic acid (canola).

tual amount of essential fatty acids and LCPUFA present in human milk varies depending on the maternal diet, being low in occidental diets, and also on maternal genetic variants in the desaturase-encoding genes [10]. Recently, an intake of at least 300 mg/day of eicosapentaenoic acid plus DHA, of which 200 mg/day are DHA, has been recommended during pregnancy and lactation [3].

Human milk provides close to 50% of the energy as lipids. Oleic acid is the predominant fatty acid, while palmitic acid is provided in the sn-2 position of the triglyceride, enhancing its absorption. Preformed cholesterol in breast milk (100–150 mg/dl) provides most of what is needed for tissue synthesis, thus downregulating endogenous cholesterol synthesis in the initial months of life.

Trans fatty acids are the product of hydrogenation of vegetable oils (soy) with the object of making these less susceptible to peroxidation (rancidity); thus the processed foods prepared with *trans* fatty acids have a longer shelf life, which is in the interest of producers and retailers. However, the effect of these fats on lipoprotein metabolism is indeed more harmful than that of saturated fats (C14, C16), since they not only increase LDL cholesterol (the cholesterol-rich atherogenic lipoprotein) but also lower HDL cholesterol (the protective lipoprotein responsible for reverse cholesterol transport). The net effect is that these fats contribute substantially to raising the risk of cardiovascular disease, as seen in table 3. *Trans* fatty acids during pregnancy and lactation have been associated with several negative outcomes related to conception, fetal loss and growth. The vulnerability of the mother-fetus/infant pair suggests that the diet of pregnant and lactating women should be as low in industrially derived *trans* fatty acids as practical [3].

Fats in the Second Year of Life and Beyond

After 2 years of life, recommendations on fat intake need to consider the level of habitual physical activity, since the need for energy-dense food

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sources such as fat should be adjusted to the energy required to promote healthy weight and active living; the energy needs for growth after 2 years represents 2–3% of the daily needs. Sedentary children will meet their energy needs easily with fat energy of around 30% of the total, while active children may benefit from higher fat energy (see table 4 for full details). In terms of cardiovascular disease prevention, the key aspect is the quality of the fat; decreasing saturated fats (especially C14 myristic and C16 palmitic acids) is crucial, even if C18 stearic acid is neutral in terms of cholesterol, since most of it is converted to oleic acid by the liver. Thus, a mild elevation in LDL cholesterol is offset by a rise in HDL. The key issue in the prevention of obesity is keeping energy intake and expenditure in balance at a healthy weight. Reducing fat intake is one way of achieving this, but it may not be the most sustainable way [3, 11].

DHA supply in children shows no evidence of an effect on cognitive function. There is some evidence of a benefit to behavioral changes in attention deficit syndrome, but not enough evidence for an effect on cystic fibrosis, asthma or modifying body composition [12].

Conclusions

- According to the breast milk model, the intake of lipids in the first 6 months of life should provide 40–60% of total energy, have an n–6: n–3 ratio of 5–10:1 and <1% *trans* fats, and should be free from erucic acid
- Total fat should be gradually reduced to 35% at 24 months
- After the age of 2 years, dietary fat should provide 25–35% energy; n–6 PUFA should provide 4–10% energy, n–3 1–2% energy, saturated fat <8% energy and *trans* fats <1% energy
- n–6 fatty acids should be limited to <8% and total PUFA to <11% of total energy; n–9 oleic acid can bridge the difference
- The quality of the fat, more than its quantity, is important for lifelong health

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