The Role of Dietary Fat in Child Nutrition and Development: Summary of an ASNS Workshop[1]

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The role of dietary fat in the growth, development and long-term health of children was explored from metabolic, genetic, dietary and behavioral perspectives at a workshop held September 17–18, 1997, at the Federation of American Societies for Experimental Biology, Bethesda, MD. The workshop focused on metabolic, genetic, behavioral and food intake studies that might be useful in evaluating the effects of level and type of fat in the diets of children on their risks of developing cardiovascular disease (CVD).[3] Participants sought to identify evidence dealing with the following: *1*) current intake and utilization of lipids in children, *2*) factors affecting that intake, and *3*) the consequences of that intake on the pathogenesis of atherosclerosis.

METABOLIC AND GENETIC CONSIDERATIONS

The need for fat. The disparagement of dietary fat sometimes obscures the fact that children and adults need fat in their diets. It supplies essential fatty acids (EFA) and aids in the absorption of fat-soluble vitamins A, D, E and K. It is a substrate for the production of hormones and mediators. Fat, especially in infancy and early childhood, is essential for neurological development and brain function. Mother's milk and infant formula supply 40–50% of their energy as fat (Fidler et al. 1998).

Body fat can arise from endogenous metabolism and from ingested carbohydrate and protein. The metabolism of these three macronutrients and the consequences of dietary changes are interrelated and complex, involving many enzyme systems. Globally, populations obtain energy from widely ranging intakes of carbohydrates and fat (Stephen et al. 1995).

The implications of carbohydrate metabolism are complex and deal with the transport of monosaccharides, their regulation by insulin and the insulin receptor signaling system (Longo and Elsas 1998). Inherited disruptions of the insulin receptor system can result in decreases in subcutaneous fat, growth retardation and chronic ketonuria. For example, galactosemia is a disease requiring nutritional intervention to save the child's life (Elsas et al. 1995). Clinical guidelines during the newborn period identify and remove lactose from the diet (and hence mother's milk) to prevent a very serious hepatotoxic syndrome that leads to death. There are ~90 mutations known in the three enzymes required for the conversion of galactose to glucose. Genetic variations in the six known glucose transporters may also reduce their ability to handle monosaccharides and to respond to insulin, which may make the patient intolerant to low fat, high carbohydrate diets. This type of diet, often prescribed for children with hypercholesterolemia, may also induce hyperglycemia and insulin resistance in children with familial combined hypercholesterolemia (Cortner et al. 1998). Such diets, particularly those with a high monosaccharide content, may also reduce HDL concentrations, which is antithetical to the goal of this specific diet intervention (Starc et al. 1998). If dietary intervention for hypercholesterolemia is attempted in childhood, all changes in the diet —not only the fat content—must be evaluated to ensure that the overall risk to the health of the child is minimized.

EFA from foods. The essentiality of the (n-6) fatty acid linoleic acid (LA) has been known for decades. For example, infants drinking skim milk had mild diarrhea and dry, inflamed skin (Wiese et al. 1958); results of a controlled low fat feeding study showed that the rapidly developed dermal symptoms could be reversed with very small amounts of dietary LA (Hansen et al. 1958). Summarizing results obtained with 428 infants, Hansen et al. (1963) concluded that the unwanted dermal signs in all infants receiving 0.04% of energy intake as linoleate could be reduced to 40% by increasing linoleate intake to 0.07% and prevented by giving 1.3%. These results, plus those for diarrhea, suggest that most unwanted symptoms would be prevented by two- to threefold greater amounts of dietary LA (i.e., ~0.2%). Similarly, Combes and Stakelum (1962) noted that thousands of well-grown infants were raised with diets containing from 0.4 to 0.9% LA; Cuthbertson (1976) recalculated those values to show that the amount needed to prevent deficiency symptoms in infants was probably <0.5%. Estimates of the amount needed for the optimal growth and function of healthy infants range from <0.5 to 5% of energy intake (Crawford et al. 1978).

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[3] Abbreviations used: AA, arachidonic acid; ALA, α-linolenic acid; CSFII, Continuing Survey of Food Intakes by Individuals; CVD, cardiovascular disease; DGFA, Dietary Guidelines for Americans; DHA, docosahexaenoic acid; EFA, essential fatty acids; HEI, Healthy Eating Index; LA, linoleic acid; LCPUFA, long-chain polyunsaturated fatty acids; NHANES, National Health and Nutrition Examination Survey; PUFA, polyunsaturated fatty acids; RDA, recommended dietary allowances.

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Substantial data support the dietary essentiality of the (n-3) fatty acid α-linolenic acid (ALA). As parent fatty acids of the (n-6) and (n-3) series, LA and ALA are converted by a series of desaturations and elongations into their respective principal products, the 20- and 22-carbon long-chain polyunsaturated fatty acids (LCPUFA), i.e., arachidonic acid (AA) and docosahexaenoic acid (DHA). Because the same enzymes are utilized, metabolic competition exists between LA and ALA. Biomarkers reflecting this competition clearly indicate nutrient status (reviewed recently by Lands 1995), and many reports describe how the relative abundance of specific fatty acids in plasma lipids indicate dietary adequacy. For example, Collins et al. (1971) noted that dermal symptoms occurred only several weeks after the level of 20:3(n-9) in plasma phospholipids became greater than that of 20:4(n-6). Similarly, infants showing signs of deficiency had triene/tetraene ratios of ~1.4, whereas those free of deficiency symptoms had an average ratio of ~0.3 (Hansen et al. 1963).

Long-term dietary deprivation of (n-3) fatty acids results in measurable changes in the visual and neurological function of primates (Neuringer and Connor1986), which may be explained by the fact that DHA is the major (n-3) fatty acid in brain and retinal phospholipids. Both DHA and AA accumulate in the developing brain and retina during gestation and early infancy. These LCPUFA are present in human milk but not in infant formulas. However, not all human milk has the same amounts of (n-3) and (n-6) LCPUFA; this amount always depends on the mother's ingestion of appropriate proportions of the EFA. Mothers should be counseled on this aspect of maternal nutrition. Several groups have established that infants, even those who are preterm, can convert LA to AA and ALA to DHA, but some investigators believe that these conversions are insufficient to support optimal growth and development. This area is being actively debated [British Nutrition Foundation Task Force on Unsaturated Fatty Acids 1992, European Society of Pediatrics, Gastroenterology and Nutrition (ESPGAN), Committee on Nutrition 1991, FAO/WHO 1994, International Society for the Study of Fatty Acids and Lipids (ISSFAL) 1994, Raiten et al. 1998, Salem et al. 1996, Sauerwald et al. 1997]. The rationale for adding LCPUFA to preterm infant formula is more compelling than that for adding them to formulas for full-term infants.

Less emphasis has been placed on the LCPUFA requirements of healthy children beyond infancy. Further research is required before any recommendations can be made regarding the optimal intakes of (n-6) and (n-3) fatty acids for healthy children beyond the current recommendations for LA intake. However, the ratio of (n-6) to (n-3) fatty acids in the American diet has increased dramatically during this century. It is not unreasonable to expect that a change in the (n-6) to (n-3) ratio of the diet could have significant physiologic effects. The cell membrane phospholipid fatty acid composition can affect membrane fluidity, membrane permeability and perhaps receptor function. Furthermore, several LCPUFA of both the (n-6) and (n-3) series are precursors of eicosanoids (such as prostaglandins, thromboxanes and leukotrienes), many of which have potent biologic actions. Given the competition between the (n-6) and (n-3) series of fatty acids for enzymes responsible for their elongation and desaturation as well as the different types of eicosanoids they produce, we should attend to the (n-6) to (n-3) ratio when recommending intakes of EFA. A useful biomarker of (n-6)/(n-3) intakes is the proportion of (n-6) LCPUFA in plasma phospholipids, i.e., ~75% for people eating diets characteristic of the U.S. and ~50% for those eating diets typical for Japan. For Japanese who moved to Brazil, the value shifted from 54% to 73% and the incidence of heart disease increased several-fold (Mizushima et al. 1992). A possible indicator of current excessive intake of LA in the U.S. is the near absence of 20:3(n-9) in human plasma phospholipids (Lands 1992).

Ratios [(n-6) to (n-3)] from ~4 to 16 have been recommended (ESPGAN Committee on Nutrition 1991, British Nutrition Foundation Task Force on Unsaturated Fatty Acids 1992, FAO/WHO 1994, International Society for the Study of Fatty Acids and Lipids 1994). Neuringer and Connor (1986) and Holman (1998) suggest that the (n-6)/(n-3) ratio should not exceed 10 and may be optimum at 4.

Children's diets and the risk of cardiovascular disease in adulthood. No direct evidence links childhood nutrition to CVD in adulthood. Ecological studies show substantial variation in CVD among countries, but studies of the relationship between migration and changes in CVD risk have not been sufficiently detailed to separate the effects of migration in adulthood from those in early childhood. These data include cross-sectional associations among risk factors studied in both childhood and adulthood, and longitudinal associations based on tracking these factors over time. Many of the correlations among these factors, both cross-sectionally and longitudinally, are weak, i.e., on the order of 0.2. Such weak correlations provide little support for drawing firm causal inferences.

Overall, the major determinants of plasma cholesterol levels in children and adolescents are similar to those in adults. The key determinant is an individual's genetic background, but major modifying factors include diet and behavior. In most populations, children respond like adults to higher intakes of saturated fat (and to a lesser degree, cholesterol) with variable increases in plasma cholesterol levels.

The relationships between dietary fat and CVD are real but complicated. For example, saturated fat is said to raise blood cholesterol levels, but not all saturated fatty acids have this effect. Stearic acid, the main fat in chocolate, is neutral with respect to cholesterol (Kris-Etherton and Yu 1997). Further, debate continues over whether concerns about fat should focus on total fat intake or on the relative proportions of saturated, monounsaturated and polyunsaturated fatty acids (PUFA) consumed (Grundy 1997). There are also questions about *trans* fatty acids (Lichtenstein 1997) and oxidized fats and their potential relationship to CVD. Even among polyunsaturates, the ratio of (n-6) to (n-3) fatty acids in the American diet has increased dramatically, a development that might be contributing to the high incidence of CVD.

In addition to relationships to dietary fats, scientists have uncovered other dietary links to CVD (Dwyer 1995, Halliwell 1997, Rimm and Colditz 1993, Stampfer et al. 1993). These include effects of antioxidants such as vitamin E, fruits and vegetables, and fiber levels, which appear to reduce the risk of CVD among adults. We do not know the extent to which all f these dietary components increase or decrease the risks of CVD in children.

The belief that diet plays a major role in regulating serum cholesterol levels in children and adults (and, by inference, the prevalence of coronary heart disease) emerges from population studies in counties that are rapidly "Westernizing" dietary intakes and habits (NCEP 1991). In Spain and Japan, for example, there have been substantial recent increases in childhood plasma cholesterol levels so that current mean levels in these children exceed that of the U.S. 75th percentile. Metabolic programming might help explain why Japanese children have average higher blood cholesterol levels compared with American children even though they eat lower amounts of cholesterol-raising foods. Increasing total and saturated fat intakes in populations not previously exposed to them in large

quantities may lead to greater and more adverse changes in their blood-lipid profiles compared with populations long adapted to Western diets.

Numerous studies have shown that the early stages of atherogenesis begin in infancy and childhood and occur independently of gender, race, diet or national origin. These early lesions are fatty streaks that occur first in the aorta and, after age 12 y, in the coronary arteries (Robbins and Cotran 1979, Stary 1990). More advanced stages of atherogenesis, which cause raised lesions (plaques), appear to be inhibited in childhood and do not occur until after puberty in boys and much later in girls.

Risk factors for coronary artery disease in adults include genetics (revealed by family history), hypertension, hypercholesterolemia (as it reflects increases in LDL), homocystinemia, cigarette smoking, diabetes and obesity. The physiologic factors that promote atherogenesis after puberty in boys are plasma androgens (Berenson et al. 1981). The increase in estrogens in girls at puberty does not appear to affect their childlike lipid profile. The pattern of relatively low plasma LDL and high HDL persists in women until just before menopause when the loss of estrogen increases the risk of plaque formation and coronary artery disease.

Clinical atherosclerosis is unknown in healthy children who have low blood pressure, low levels of LDL, higher levels of HDL and are nonsmokers. Only in cases of embolic, inflammatory or anomalous structural disease of the coronary arteries (Bor 1969) or in rare cases (1 per 1,000,000 births) of homozygous familial hypercholesterolemia (Bilheimer et al. 1985) homocystinuria (McCully 1969) or progeria (Talbot et al. 1945) does coronary artery disease occur in children. Hypercholesterolemia, hypertension and diabetes, however, do appear in children. The question is whether these risk factors promote atherosclerosis beyond the fatty streak in children. Serum cholesterol levels in children rise from ~75 mg/dL at birth with consumption of breast milk or formulas to ~140 mg/dL at 4–6 mo of age without major differences in their arteries. Serum cholesterol values remain at ~155 mg/dL without any significant plaque formation until beyond puberty (Olson 1995).

The earliest demonstration of a delay in plaque formation in boys until puberty was shown in the work of Henry McGill and co-workers at Louisiana State University in the 1950s. These investigations compared the extent and degree of atherosclerosis of the aorta in a total of 941 persons who came to autopsy in New Orleans, San Juan, Costa Rica and Guatemala City (Tejada et al. 1958). The specimens were about equally divided among the three cities and represented persons from birth to 80^{+} y. The amount and grade of atherosclerosis was quantitated beginning with fatty streaks and ending with calcified plaques. Data showed that only fatty streaks were seen in children up to age 25 y in all three countries. For those older than 25 y, there was a more rapid increase in plaque concentration in New Orleans compared with that in the countries in Central America. The rise in significant atherosclerosis after age 25 y was proportional to the average serum cholesterol in the three countries.

More recently, investigators performed autopsies as part of the Pathological Determinants of Atherosclerosis in Youth Study (PDAY) on 2876 accident victims from 15 to 34 y old of whom one half were black and one fourth were women (McGill et al. 1997, PDAY Research Group 1990, Strong et al. 1999). In a group of adolescent boys and girls 15–19 y of age, fatty streaks made up ~20% of the area of the aorta and the plaques were 0.35% of the area of the aorta. In the right coronary artery, fatty streaks involved 1.8% of the area and the plaques occupied 0.5% of the area for women and 0.7% for men. In a group of men and women 15–24 y old, increased LDL cholesterol level correlated with increases in the number of fatty streaks in the aorta in both men and women, but had no effect on plaques. In the right coronary arteries, hypercholesterolemia had no effect on streaks or plaques. Strong et al. (1999) concluded that both fatty streaks and plaques increased in prevalence and extent from ages 15 to 34 and that primary prevention of atherosclerosis as contrasted with the primary prevention of clinically manifest atherosclerosis disease must begin in childhood or adolescence. Similar results were observed by Berenson et al. (1992) and Tracy et al. (1995) in 150 persons ages 6–30 y in the Bogalusa Heart Study. They found that although fatty streaks were visible in the coronaries after age 10 y, no plaques were observed until after age 15 y. No correlation between intimal thickness in the coronaries and total serum cholesterol or LDL cholesterol was noted.

In the study of battle casualties in World War II and the conflicts in Korea and Vietnam, the average age of fatalities among soldiers was ~25 y. Soldiers were studied in World War II (n = 140), Korea (n = 200) and Vietnam (n = 105). The prevalence of coronary artery plaques was noted in ~45% of the autopsies in all three studies, which is higher than that observed in this same age group in the PDAY study, but without clinical sequelae. A report from Strong et al. (1999) on the PDAY study has expanded the number of subjects from 1443 to 2876. They observed fatty streaks and raised lesions that increased in prevalence and extent between ages 15 and 34 y. They concluded that, "primary prevention of atherosclerosis, as contrasted with primary prevention of clinically manifest atherosclerotic disease, must begin in childhood or adolescence."

In examining the effectiveness of diet in modifying serum cholesterol in hypercholesterolemic children aged 8–10 y, the Dietary Intervention in Children Study (DISC Collaborative Study Group 1995) showed that lowering dietary fat to 29% of energy and saturated fat to 10% had a negligible effect on total (−3 mg/dL) and LDL cholesterol (−4 mg/dL) over 3 y compared with a control group. Tracking serum lipids from childhood to adulthood involves determining the rank of each individual for total cholesterol and LDL cholesterol over time.

Lauer and Clarke (1990) studied 2367 school children in Muscatine, IA; the children were examined at ages 8–18 y and then reexamined as adults 12 y later. Of 249 children >75th percentile (175 mg/dL) at baseline, only 42 (17%) had serum cholesterol above the 75th percentile (>240 mg/dL) as adults. Of hypercholesterolemic children, 47% became normocholesterolemic (<200 mg/dL) as adults.

Orchard et al. (1983) studied 611 subjects as children (11–14 y of age) and later as adults 20–30 y of age in Beaver County, PA. They found that of 116 children in the highest quintile (>80th percentile) with respect to serum cholesterol, 56 (48%) had serum cholesterol values in the highest quintile as adults.

Webber et al. (1991) studied 1586 children in 1973–1974 and again between 1984 and 1986 in Bogalusa, LA. Of the 158 children who had serum cholesterol >75th percentile, only 35 (22%) were above the 75th percentile (>240 mg/dL) as adults. Tracking for HDL cholesterol was better after age 9 y for white males. Approximately 50% of those children who had total cholesterol levels or LDL cholesterol levels >75th percentile at baseline remained elevated 12 y later. These results for tracking agree well with the findings of Lauer and Clarke (1990) from the Muscatine Study. Thus, the tracking of hypercholesterolemia from childhood to adulthood is imperfect, with a range of 13, 17 and 48% in the three studies.

Genetic and dietary interactions. Most diseases have some combination of genetic and environmental roots. Among adults, for example, genetic factors contribute to the wide differences seen in the response of serum lipoproteins to consumption of both total and saturated fat in the diet (Ordovas et al. 1995). The genetic-environmental interactions can be very complex. The most well-defined genetic trait affecting response of LDL cholesterol to diet is the apoE4 variant of apoprotein E (the normal form, apoE3), which affects ~1 in 7 Americans. Compared with those with apoE3, individuals with apoE4 have a tendency toward higher blood cholesterol levels and increased risk of CVD. Most studies show that LDL reductions from consumption of low fat, low cholesterol diets are greater in subjects with apoE4 than in those with apoE3 (Lopez-Miranda et al. 1994) Another, less common gene variant, apoA-IV-2, appears to prevent the rise in LDL induced by an increased intake of dietary cholesterol (McCombs et al. 1994).

A much more common genetic variant, LDL subclass pattern B, produces especially small and compact LDL (Austin et al. 1990). This trait is found in ~33% of adult men and 15–20% of postmenopausal women, but in only 5% of children and premenopausal women. Pattern B is also characterized by metabolic disturbances, including lower blood levels of HDL cholesterol, increased levels of triglyceride and apolipoprotein B (the major LDL protein) and predisposition to diabetes mellitus. As a result, those with pattern B have an overall threefold higher risk of CVD compared with those whose blood carries larger LDL particles (pattern A). Although blood levels of LDL are not elevated in pattern B individuals, the small size of their LDL particles may be particularly harmful compared with larger LDL particles found in pattern A individuals because they are more likely to be retained in the artery wall and are more susceptible to oxidation (an event that appears to be critically important in the development of atherosclerosis and CVD). It seems that pattern B can result from alterations in one of several genes, but the specific mutations responsible for the trait have not been identified (Rotter et al. 1996). As suggested by the low prevalence of pattern B in children, age and other factors such as hormonal status, body weight and diet might also be major determinants of the presence and severity of the pattern B trait in genetically predisposed individuals.

Initial studies indicate that the reduction in LDL cholesterol induced by a low fat, high carbohydrate diet was twice as great in the 18 men with pattern B compared with the 87 subjects with pattern A (Dreon et al. 1994). But in about one third of the pattern A men, the low fat diets shifted them to a pattern B profile without reducing LDL levels, increasing rather than decreasing the ratio of total to HDL cholesterol (an index of CVD risk). These findings raise the possibility that individuals with this response may actually have adverse rather than beneficial metabolic consequences from eating low fat diets. In a second study of 133 men, there were stepwise improvements in blood LDL levels in pattern B individuals, but not pattern A, as dietary fat was reduced from 40 to 30 to 20% of energy. Replacing saturated fat with either carbohydrates or monounsaturated fats showed that both dietary shifts lowered the LDL cholesterol in the pattern B men, but only the diet with higher amounts of monounsaturated fat reduced levels of triglycerides and apoprotein B. This effect was not seen in the pattern A individuals; thus efforts to reduce the incidence of heart disease by modifying fat intake may be more effective in high risk pattern B individuals than in pattern A subjects who have a normal blood cholesterol profile.

Strong evidence suggests associations between specific genotypes or haplotypes and plasma lipid levels and postprandial lipid responses, but the expression or response to these genotypes in children, both individually and as populations, remains largely unstudied. That 95% of children have pattern A may explain why they are resistant to reduction of LDL by dietary fat.

The growing knowledge of gene-diet interactions as they affect the risk of CVD and other disease offers compelling evidence that individual responses to dietary interventions cannot be predicted reliably from typical effects observed in large population studies. Both genes and individual variability at the metabolic level may have a tremendous influence on the net response to a dietary manipulation. As new tools for genetic analysis become available, they will undoubtedly allow more individualized dietary recommendations for CVD prevention.

Screening children for CVD risk. Participants examined the question whether available evidence justifies childhood cholesterol screening. Recommendations for screening should be based on evidence that the costs and risks are justified by projected benefits (Toronto Working Group on Cholesterol Policy 1990). Some investigators concluded that no cholesterol-lowering intervention has been shown to be safe and effective in children, and the benefits of intervention in childhood (as opposed to later) are likely to be trivial in relation to costs (Newman et al. 1992).

In the DISC (1995) study, the effect of an intervention that included 29 visits and 36 telephone calls over 3 y was a 2% (4 mg/dL) decrease in LDL cholesterol levels. These results are consistent with the disappointing results from studies of free-living adults. More intensive ("Step Two") diets applied in high risk populations can reduce cholesterol by ~5%, but they reduce HDL cholesterol by at least as much as they reduce LDL cholesterol and have not been shown to be safe in children (Newman and Hulley 1996).

Cholesterol screening in children is not necessary for the following reasons: *1*) there are no safe and effective treatment options; *2*) childhood serum cholesterol values do not predict adult levels very well; *3*) there are psychological consequences to children being labeled hypercholesterolemic (Rosenberg et al. 1997); and *4*) significant atherosclerosis (raised lesions) does not occur until adolescence in boys and later in girls.

Several expert groups, such as the American College of Physicians and the U.S. Preventive Health Services Task Force, recommend that routine cholesterol screening begin at around age 35 in men and 45 in women (American College of Physicians 1996, Canadian Task Force on the Periodic Health Examination 1993, Garber et al. 1996, Garber 1997, Garber and Browner 1997). Perhaps earlier screening might be warranted for those with diabetes, those who smoke or those with other CVD risk factors.

Questions remain about the importance of observations that lower cholesterol levels in adults are associated with adverse effects unrelated to CVD, particularly violent deaths (Muldoon et al. 1990, Newman et al. 1992, Smith et al. 1993, Strandberg et al. 1991). The significance of such observations to children appears questionable. Nonetheless, violence is a much more common cause of death in children than in adults.

The genetics and energetics of childhood obesity. It is well known that obesity runs in families. With the possible exception of some rare genetic disorders such as Bardet-Beidle syndrome, however, obesity does not exhibit a clear pattern of Mendelian inheritance. The risk of becoming obese when a first-degree relative is overweight or obese is estimated to be two to three times higher than for the general public (Allison et al. 1996, Lee et al. 1997). Moreover, the risk increases with

the severity of obesity and is about eight times higher in families of extremely obese [body mass index (BMI) >45] subjects. The heritability is highest with twin studies (50–80%), intermediate with nuclear family data (30–50%), lowest in adoption studies (10–30%), and clusters around 25–40% in the age- and gender-adjusted phenotype when numerous relations are used (Chagnon et al. 1997a, Maes et al. 1997). The common familial environmental effect tends to be marginal, and there is no consistent evidence of sex and age differences (Fabsitz et al. 1992, Korkeila et al. 1991).

Energy intake and expenditure are influenced by genetic factors (Bouchard et al. 1994, Pérusse and Bouchard 1994). In the Quebec Family Study, a 3-d dietary record was obtained in 1597 individuals from 375 families. Pérusse and Chagnon (1997) found a significant transmission effect (cultural + genetic) between parents and offspring for fat intake, with a genetic effect reaching 19% for the percentage of energy derived from fat.

Molecular epidemiology studies have identified several candidate genes potentially involved in the etiology of obesity (Pérusse and Chagnon 1997). The most recent obesity gene map reveals the presence of >100 putative loci related to obesity located on chromosome Y in humans (Chagnon et al. 1997b). Much of the recent progress in the genetics of obesity has occurred by identifying new genes and molecules such as leptin and uncoupling protein 2 that are involved in the regulation of energy balance. However, most attempts to relate mutations in these or other genes to human obesity have failed (Comuzzie and Allison 1998). A limited number of cases have shown that mutations clearly lead to obesity in humans (Pérusse et al. 1999). Examples of genes containing these mutations are the leptin (LEP) (Comuzzie et al. 1997, Montague et al. 1997), prohormone convertase (PC1) (Jackson et al. 1997), LEP-R, POMC, MC4R and PPARγ2. These new discoveries show that single-gene mutations in the metabolic pathways of animals could be responsible for obesity in humans, but these effects do not apply to the majority of obese humans. Pérusse and Bouchard (1999) recently reviewed data related to obesity in childhood and the role of genetic factors in phenotypes related to obesity. They concluded that genetic factors influence body fat content, energy balance and the responsiveness to dietary intervention.

The prevalence of obesity has continued to increase in children despite a general increased awareness in health, nutrition and fitness. In addition, the induction of obesity-related disease is dramatically increasing in children (e.g., the incidence of noninsulin-dependent diabetes mellitus in children and adolescents has increased 10-fold over the last decade). The etiology of the development of childhood obesity and subsequent disease is poorly understood, but is likely to be explained by alterations in the regulation of balance between energy expenditure and energy intake related to genetic control and available food. It is not known whether obesity is due to changes in the magnitude of one or both of these variables. In addition, the negative health aspects of obesity in children may, as in adults, be related more specifically to body fat distribution rather than to total body fat (Goran 1997).

Energy requirements are generally defined as the amount of dietary energy for maintenance of health, growth and an "appropriate" level of physical activity, and can be based objectively on a measurement of total energy expenditure by using doubly labeled water. This method enables an integrated measurement of total, resting, growth- and physical activity–related energy expenditure and has been used in a wide array of different subgroups of the population. These studies are unanimous in showing that current estimates of energy needs in children and infants are too high by ~25% and, if adhered to, would promote obesity (Goran 1997). Energy expenditure is the most variable component in children, but whether reduced energy expenditure is the key to obesity is controversial. Further studies are required to quantitate the changes in energy balance in obese children, particularly because body changes may be small but cumulative. It has been shown that low levels of physical activity in preschool children (derived from doubly labeled water measurements) are associated with increased levels of body fat (Davies et al. 1995)

Few studies, if any, have examined dietary fat requirements based on objective measurements. By applying the same principle that has been described for dietary energy needs, dietary fat requirements can be estimated on the basis of fat utilization. A hypothetical 5-y-old child, for example, has a free-living energy expenditure of ~1450 kcal/d, based on measurements using doubly labeled water. Given a measured respiratory quotient of 0.89, 1450 kcal/d translates to a daily fat oxidation of 49 g. An additional 6 g fat/d are stored for growth, according to longitudinal studies of change in body composition at this age. Thus, the dietary fat usage in this case can be estimated at ~54 g/d, accounting for about 34% of energy. Great individual variation (especially in the level of physical activity) makes it difficult to translate this type of empirical data into recommendations for the general population.

DIETARY INTAKE AND EATING BEHAVIOR OF CHILDREN

Dietary intake. Data on the eating habits of U.S. children came from nationally representative dietary surveys conducted by the federal government. The USDA periodically conducts the Nationwide Food Consumption Survey (NFCS) and, on a regular basis, smaller surveys—the Continuing Survey of Food Intakes by Individuals (CSFII). The U.S. Department of Health and Human Services conducted the second National Health and Nutrition Examination Survey (NHANES II) in 1976–1980 and the third NHANES survey in 1988–1994. Survey data must be interpreted carefully because reported nutrient intakes are likely underestimates of actual intakes, a situation probably caused by underreporting (Black et al. 1993). In particular, there seems to be bias in underreporting "sin" foods. Underreporting of intake is a greater problem as children reach adolescence (Livingstone et al. 1992), particularly among the obese (Bandini et al. 1990). A relatively new methodology called the multiple-pass 24-h recall (used by USDA since 1994) holds promise for more accurate reporting of food intake (Johnson et al. 1996).

USDA surveys show an upward trend in energy intake across age-gender groups per day, perhaps due in part to children now eating about seven times a day compared with three to four times in the early 1970s. Calcium and iron intakes are below recommended levels, particularly among adolescent females. Specifically, the surveys from 1987 to 1995 show that fat and saturated fat intake as a percentage of energy has declined steadily in children's diets over this 8-y period (Johnson et al. 1994a and 1994b; Kennedy and Goldberg 1995, Wilson et al. 1997). A result that stratifies along income shows that children aged 2–17 y decreased their fat intake from an average of 36% of energy to 33% and their saturated fat intake from 14% to 12% of energy. Total fat and saturated fat intake has been declining since 1987 among children in middle- and upper-income households. But it has been increasing among low-income children who have been drinking

whole milk rather than lowfat or skim milk, which may be a major factor in this trend.

In contrast to fat intake as a percentage of energy consumed, actual fat intake in grams has not declined between 1987 and 1995 and, in fact, has increased in some age-gender groups such as adolescent boys (Johnson et al. 1994a and 1994b; Kennedy and Goldberg 1995, Morton and Guthrie 1998, Wilson et al. 1997). Data from NHANES II and III confirm this temporal trend of slightly less fat intake as a percentage of energy but higher intake by weight measured in grams, in part because energy intake has increased over time. Furthermore, fat intake does not decline with age; the share of energy from total and saturated fat differs by less than 1% across all age and gender groups from ages 1 to 19 y. Morton and Guthrie (1998) found that the total grams of fat consumed daily by children 2–17 y old increased slightly when they compared data from the 1994–1995 USDA CSFII with that of the 1989–1991 survey. The percentage of energy from fat decreased due to higher levels of carbohydrate, particularly from beverages (soft drinks) in adolescent males.

Various studies show that fat intake can vary by sociodemographic characteristics. Factors associated with higher fat intake include the following: *1*) being black or Hispanic; *2*) living in a rural area; *3*) being from a lower-income household or having a greater risk of household food insecurity; *4*) having an unemployed father or mother employed in a clerical or service or farming occupation; and *5*) eating foods away from home (Johnson and Wang 1997, Kennedy and Goldberg 1995, Lin et al. 1996).

An association between total fat intake and diet quality was demonstrated after investigators examined weighted data from a representative sample of 45,752 children 5–17 y of age from the CSFII 1989–1991 survey (Johnson and Wang 1997 and 1998). They focused on the intake of energy from saturated fat, cholesterol, sodium, fiber and four "problem" nutrients—vitamin A, vitamin E, calcium and zinc; for these nutrients, 30% or more of the sample had intakes <77% of the respective recommended dietary allowances (RDA). With the sample divided into five quintiles by the percentage of fat intake and adjusted for age, race and sex, the percentage of fat intake was positively associated with the intake of energy, saturated fat, cholesterol, sodium, vitamin E, calcium and zinc and negatively associated with vitamin A; there was little association with fiber intake.

The decline in calcium intakes as fat intake decreased is a special concern, i.e., it suggests that the parallel public health messages to decrease fat and increase calcium have not been consistently presented (or interpreted) as mutually compatible. The message "eat less fat" cannot be translated to mean "drink less milk." Dairy products account for ~75% of calcium in the U.S. food supply, and milk consumption has dropped markedly among children, especially adolescents. Currently, only 14% of girls and 35% of boys between 12 and 19 y meet the RDA of 1200 mg calcium/d (Wilson et al. 1997). Even fewer would meet the new Adequate Intake of 1300 mg for calcium established by the Food and Nutrition Board.

The dietary patterns of children in a biracial (black and white) Louisiana community were studied from 1973 to 1994 in the longitudinal Bogalusa Health Study (Nicklas 1988). Dietary data on children as early in life as 6 mo of age compared favorably in terms of nutrient intake and secular trends in food intake to data from national USDA surveys between 1987 and 1994. Over the 21 y of the study, total energy intake was unchanged, but the percentage of energy from fat decreased significantly from 38.4 to 36.0%. The percentage of energy from saturated fat decreased as well, reflecting less consumption of palmitic, stearic and oleic acids. Consumption of monounsaturated fat decreased, whereas polyunsaturated fat intake increased. However, three of four children still exceeded the dietary recommendations for fat and saturated fat, and they had dietary cholesterol intake >100 mg/1000 kcal. There is a decline in consumption of milk, vegetables/soups, breads/grains and eggs with increased intakes of fruit/fruit juices, carbonated beverages, seafood, poultry, cheese and beef. Fat intake from milk, beef, pork and desserts has decreased, whereas it has increased from mixed meats, poultry, breads and grains. Data on micronutrient intakes have to be interpreted with caution because it is difficult to obtain accurate information on dietary supplement use and because of incomplete data on the nutrient content of some foods. There has been a positive trend over time in the intake of phosphorus and calcium, but a negative trend in intake of iron, thiamin, niacin, folate and vitamin E. These data indicate that the diets of the Bogalusa children have improved since 1973 and that the nutrient density of the diet has improved as intakes of total fat and saturated fat have decreased (Nicklas et al. 1992 and1993). However, the children weighed more for height, and <25% took part in any vigorous physical activity for at least 20 min/d. Inadequate intakes (< two thirds of the RDA) of nutrients such as calcium, iron and zinc become more of a problem after age 10 and into adolescence.

More recently, the Child and Adolescent Trial for Cardiovascular Health (CATCH) Study showed success in maintaining mean vitamin and mineral intake of intervention group children when total dietary fat was reduced 2.4% (Nicklas et al. 1996). Also, with the exceptions of iron and vitamin D, the STRIP study showed that the micronutrient intakes of infants and toddlers on fat-reduced diet (27–29% of calories) met standards (Lagström et al. 1997).

In examining the dietary patterns of preschool children, USDA data show that ~98% of preschool children meet or exceed the RDA for energy. For preschool children, the USDA food pyramid uses a 1600 kcal intake, which is found in the CSFII 1995 data. More than 50% of 3- to 5-y-olds meet 100% of the RDA except for calcium, zinc and vitamin E. Preschool children are a little more vulnerable to dietary deficiencies under restrictive conditions; they are less able to help themselves to food and have smaller stomach capacity. In addition to the need to maintain adequate caloric density to ensure growth and development, there is a rationale for a transition period beyond age two in reaching a dietary recommendation such as "no >30% of energy from fat." Perhaps there should be a more individual approach for some children who are in the bottom quintile for height and weight. However, the Pediatric Nutrition Surveillance System shows us that among 2- to 5-y-olds, the prevalence of overweight is about double that of being underweight.

The total fat intake of preschool children is currently ~32%, and unsaturated fat intake is ~12% of energy. About 33% of preschoolers meet the 30% guideline for total fat consumption; only ~23% meet the saturated fat guideline, and 88% meet the cholesterol guideline. In the Bogalusa Heart Study, scientists compared the ante-mortem cholesterol levels with the degree of fatty streaks in young individuals and showed correlation between LDL levels and atherosclerosis in children and adolescents. Children in the highest quintile for cholesterol have a 70% chance over a period of 12 y of being in the upper two quintiles(Nicklas et al. 1992). In one Head Start project, ~38% of 3- to 5-y-olds are above the guidelines and 11% of those in the highest category have cholesterol >200 mg/dL (Williams, unpublished results).

Fifty percent of children between ages 3 and 5y are at day

care or preschool centers and therefore eat outside the home. Children <6 y of age are among the fastest-growing group of patrons in sit-down restaurants. CSFII 1989–1991 data show that preschool children consume ~3.6 servings of fruits and vegetables per day, compared with the recommended 5 servings. This result is irrespective of race but is affected positively by household income. Fiber intake tends to be adequate up to age 10, then begins to fall short of recommendations.

The history of numerical recommendations in guidelines for fat intake for all Americans >2 y old includes the following: 1985 NIH National Consensus Development Panel; 1992 National Cholesterol Education Program including the American Academy of Pediatrics, which had declined to recommend numerical guidelines in 1983 and 1986; and the Healthy People 2000 recommendations. Now the focus is on the transition period from the higher fat diets of infancy to the recommended 30% level by about ~5 y of age. This avoids an abrupt drop in energy density of the diet and safeguards the more vulnerable nature of preschool children to dietary deficiencies under restrictive conditions. These guidelines were derived from data in the following four areas: dietary fat and obesity; dietary fat and cancer; dietary fat and atherosclerosis; and dietary behavior and habits.

Determinants of eating behaviors. Eating is a psychosocial event, which in children and in most adults is not based on the science of nutrition; food is central to life in many more ways than simply its ability to nourish. It provides pleasure, helps to define people culturally and socially, can be a form of art, and is often used as metaphor (e.g., a nice person is "sweet"). Food also conveys correctness or incorrectness, the latter being increasingly visible in the United States regarding the eating of fat. If nutritionists do not recognize and acknowledge the multiple meanings of food, their effectiveness as nutrition educators and agents of dietary change will be reduced.

Children's food acceptance patterns are shaped by their early experiences with food and eating; however, this does not mean that early experience has an especially important role in later acceptance. The dietary pattern of the first 6 y of life is not predictive of the pattern during the second 6 y (Rozin 1990). Humans progress from eating only one food at birth to eating almost anything edible as adults. Yet, except for an innate preference for sweet tastes and a dislike of bitter ones, they have little biological equipment for discriminating between what can be eaten and should and should not be eaten (Rozin 1990, Rozin and Vollmecke 1986). During the long transition to an adult diet, children's early experience involves familiarization with foods by repeated exposure and learning about where foods come from and the nutritional and physiologic consequences of eating. Humans also have an ambivalent response to new foods—both an interest and a fear of them—so they tend to be neophobic, avoiding unfamiliar foods because they expect not to like them (Pliner 1994, Pliner and Hobden 1992).

Neophobia and a preference for sweet-tasting foods may have played a protective role in a potentially hazardous food environment, but culture has taken over much of their role by removing dangerous ingestibles from the immediate environment or by labeling them as unsafe. Studies of food neophobia in children show that children tend to be more fussy about trying novel foods if they are temperamental, shy, fearful and emotional, but become much more willing to try unfamiliar foods as they become older (Pelchat and Pliner 1995, Pliner and Loewen 1997). However, toddlers (ages 1–2 y) have relatively little neophobia and will ingest almost anything (Rozin et al. 1985). Other research has shown that the type of exposure to food is important (Birch and Marlin 1982, Birch et al. 1987). Some children show a greater willingness to try unfamiliar foods under stimulating situations, such as away from home in the presence of nonparental adults, at parties, and when the food has an unusual name. Others are more willing to try unfamiliar foods under familiar, safe situations such as at home with parents, at regular meals, and when the food does not have an unusual name.

Obviously, a food must be available or present to be consumed, but accessibility influences the probability of it being consumed. For example, carrots stored somewhere in the refrigerator are accessible to a child, but become more accessible—and more likely to be consumed—if they are cleaned, sliced, and displayed at the front of a child-accessible shelf in the refrigerator. To understand behavior, the environment in which it occurs and the characteristics of the people engaging in the behavior must be analyzed. The behavior, in turn, affects both the environment and the person. This is the principle of reciprocal determinism (Baranowski et al. 1996).

Research on fruit and vegetable consumption among children in the 3rd through 5th grades showed that personal preference (foods liked are eaten, whereas foods disliked are avoided) was the only factor that significantly predicted consumption, although it accounted for only 7–13% of the variance (Domel et al. 1996, Resnicow et al. 1997). Most children liked common fruits such as bananas and apples; preferred vegetables were corn and carrots, perhaps because they are relatively sweet. Because most elementary-school children probably have little control over the foods they consume, it seemed that availability would promote consumption. Focus group research revealed that produce availability was often limited because the parents did not like these foods and did not make them accessible (e.g., because they take time to clean and prepare, and there are issues of cost, spoilage, storage and waste) (Baranowski et al. 1993, Kirby et al. 1995). However, dietary patterns of parents have a low correlation (0.15) with the patterns of their adult children (Rozin 1990 and 1991).

Some parents disliked the fact that fruits purchased with the intent of lasting a week were consumed within a day or two by their children. Research to date suggests that if produce is available in the home or at school, children were more likely to eat it if they like it (especially true for fruits and juices), that consumption is positively correlated with social class, and that produce availability in school lunch programs is positively correlated to the social class of the student body (Hearn et al. 1996). It is counterproductive, however, to restrict children's access to high fat foods and enforce intake of "good" foods. Coercion promotes revolt and may cause resentment among children, leading them to reject "good" foods.

Some workshop participants cautioned that Americans and health-care professionals should have a more positive and relaxed attitude toward food and its relationship to health. How important is food to health, especially compared with other things such as loving parent-child relationships? Many people maintain distorted and simplistic views about nutrition (e.g., that fat and salt are toxins), which can lead to unnecessary confrontations between parents and children. It is also important to remember that the conventional wisdom in nutrition has been known to change radically over time and might happen in the future; thus, health-care professionals should be humble about projecting their current "wisdom" onto people's lives. There is also nothing magic about nutritional recommendations. The secret is not in one single food or one single food component. There are no good foods or bad foods; no good or bad fats. The seal of approval logos by

organizations such as the American Heart Association on certain foods encourage the idea that there are good and bad foods; a more sensible label would advise consumers to eat particular food products in limited, moderate or unlimited amounts.

An important reason to have a more relaxed attitude toward food is that dietary patterns are certainly not the only, and perhaps not even the major determinant of health over the life span. Today's children, for example, need to become more active, spend less time on sedentary pursuits such as watching television, and avoid smoking—all factors that affect the risk of CVD, obesity and other diseases. The percentage of high-school students enrolled in physical-education classes is decreasing and television viewing is increasing.

Considerations about dietary fat intake levels. Should children between the ages of 2 and 5 y develop dietary patterns that supply ≤30% of energy as fat, <10% of energy as saturated fat and ≤300 mg of dietary cholesterol per day? Low fat diets can vary greatly in their composition. Lower fat diets can be made palatable and do not impair growth under the intensive surveillance of clinical investigators such as in the DISC (1995) or CATCH studies (Luepker et al. 1996, Lytle 1998). Nevertheless, the effect of a diet containing 29% of energy from fat in the DISC study had little effect on serum total and LDL cholesterol in prepubertal children >3 y of age.

Windhalm and Lifshitz (1992) and Fall et al. (1992) were unable to demonstrate beneficial effects from lower fat, lower cholesterol diets that started in childhood for all children, including those with normal blood cholesterol levels. In some studies, lower fat diets reduced HDL cholesterol levels and not LDL levels (Clark et al. 1997, Hunnighake et al. 1993).

Lower fat diets that avoid or severely limit animal products such as meat and milk, for example, may not supply enough energy or micronutrients such as iron, zinc and calcium to support normal growth and development in children (Lifshitz and Moses 1989). Lifshitz and Moses (1988 and 1989) studied children with nutritional growth retardation and found that parents unintentionally fed their children inadequate diets in an attempt to prevent them from developing obesity or hypercholesterolemia. In 1994, McCann et al. (1994) speculated that half of the infants and children coming to failure-to-thrive clinics in the United States are receiving so-called healthy diets that are very low in energy and fat and contain no low nutrition snacks. Some evidence suggests that nutritional deprivation in childhood could pose greater risks for CVD risk in adulthood than nutritional overload (Barker 1996).

Guidelines concerning children's dietary fat intake. The Dietary Guidelines for Americans (DGFA) published by the USDA and the U.S. Department of Health and Human Services (USDHHS) form the cornerstone of nutrition policy in the United States (USDA and USDHHS 1995). All federal nutrition programs, both service delivery and education/promotion, are required to be consistent with them. About 20% of Americans participate in at least one of these nutrition programs (i.e., Food Stamps or School Lunch); thus, 50–60 million Americans are directly affected by the dietary guidelines. The USDA and USDHHS issue a new edition of the DGFA every 5 y; the content changes as the science base evolves (Groziak and Miller 1998).

Canada has parted ways with the United States on the issue of fat intake by children. Canadian dietary guidelines state: "From the age of two until the end of linear growth, there should be a transition from the high-fat diet of infancy to a diet that includes no > 30% of energy as fat, and no > 10% of energy as saturated fat" (Joint Working Group of the Canadian Paediatric Society and Health Canada 1993). This recommendation was based on the judgment that providing energy and nutrients to ensure adequate growth and development is the more important consideration in the nutrition of children. The reduction of fat in children's diets has been shown also to reduce the intake of energy and several nutrients including calcium, iron, zinc and B-complex vitamins. Furthermore, benefit from reduced-fat diets in children was not established. By recommending a tapered decrease in the fat percentage of energy intake from age two to adolescence, the Canadian group affirmed support for conventional fat guidelines for adults.

The 1995 DGFA recommended that children at 2 y of age gradually adopt a diet that, by ~ 5 y of age, provides no > 30% of energy from fat (Dietary Guidelines Advisory Committee 1995). The 1995 edition of the DGFA also states that "major attempts to change a child's diet should be accompanied by monitoring of growth by a health professional at regular intervals." The American Academy of Pediatrics, Committee on Nutrition (1998) updated its statement on Cholesterol in Children. The recommendation now is that after 2 y of age, children and adolescents should gradually adopt a diet that, by ~5 y of age, contains no >30% of energy and no <20% from fat. This lower limit addresses concerns that some parents and their children may overinterpret the need to restrict their fat intakes.

Assessment of dietary quality. The USDA has developed the Healthy Eating Index (HEI) to quantitate dietary quality using a single score that ranges from 0 to 100 (Kennedy et al. 1995). The HEI consists of 10 components, each contributing one tenth of the total score. The first five are based on the recommended servings from the five major food groups in the Food Guide Pyramid; four are related to the quantitative limits of the dietary guidelines regarding total fat, saturated fat, cholesterol and sodium; and one is a measure of variety in the diet.

A recent study evaluated the food intakes of 3307 children ages 2–19 y, using data from CSFII 1989–1991, and compared these data against the recommended number of servings from each food group in the Food Guide Pyramid (Muñoz et al. 1997). Only 1% met all the recommendations; 16% met none of them. Scores tended to be similar on each half of the HEI, i.e., those components related to the Food Guide Pyramid and those related to specific dietary components and variety. Preschoolers tended to have the highest scores among all children because parents or other caretakers provide their meals. These scores decreased with age. The HEI score was positively associated with household income, regardless of age. In general, children were closer to eating the recommended number of servings of dairy and meat products than they were for fruits and vegetables. Many of the grain products consumed were in the form of snacks, which have more sugar, salt and fat than standard breads and cereals. Only 33% scored a perfect 10 for total fat, saturated fat and/or sodium intake. The majority had high scores pertaining to cholesterol. Scoring well on total fat consumption (i.e., ≤30% of energy from fat) did not ensure a good total score on the HEI.

SUMMARY

EFA. Lipids are an essential component of a child's diet. They provide EFA, food energy and enhance absorption of certain nutrients. Both the (n-6) (linoleic acid) and (n-3) (linolenic acid) fatty acids are essential components of human diets in low quantities. Estimates of the dietary level of linoleic acid that will meet needs range from 0.5 to 5% of energy. At

TABLE 1

Workshop speakers[1]

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Tim Byers, M.D.
University of Colorado School of Medicine
Richard J. Deckelbaum, M.D.
Columbia University
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Emory University
Michael I. Goran, Ph.D.
University of Alabama at Birmingham
Craig Jensen, M.D.
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University of Vermont
Eileen Kennedy, D.Sc., R.D.
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Theresa A. Nicklas, Dr.P.H., L.D.N.
North Dakota State University
Robert E. Olson, M.D., Ph.D.
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Louis Pérusse, Ph.D.
Lavel University
Patricia L. Pliner, Ph.D.
University of Toronto
Paul Rozin, Ph.D.
University of Pennsylvania
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American Health Foundation

[1] Publication of this list does not constitute agreement by the speakers with the summary statements and research recommendations.

present, the ratio of intake of linoleic to linolenic acid is on the order of 100-fold in the United States. Many experts believe that the (n-3) fatty acids derived from certain vegetable oils and fish oils should be more balanced with the (n-6) fatty acids to reduce the ratio to <10.

Obesity. Body stores of fat depend on dietary intake, energy balance, needs and metabolic status. Genetic factors affect the incidence of obesity and response to fat and total energy intake. The prevalence of obesity/overweight in children is increasing despite a general awareness by children and their parents of health, nutrition and fitness. Survey data indicate that average energy intakes are increasing due to higher carbohydrate intakes. Higher carbohydrate diets may increase the risks of hypertriglyceridemia, hyperglycemia and insulin resistance in children as they become obese. Energy imbalance, consumption of excess energy and insufficient physical activity are more responsible for causing obesity than is consumption of excess dietary fat per se.

Atherosclerosis. Evidence that children develop clinically significant atherosclerosis (plaque formation) before puberty is limited to rare (1 in a million) homozygous familiar hyperlipidemia or homocystinuria. Significant CHD does not develop before the third decade of life, but experts recommend that high risk individuals begin preventive measures in adolescence. Clinical benefit from lowering the level of fat in children's diets to <30% of energy has not been established. Nevertheless, lipid indicators respond in the same direction as those of adults. For example, lowering dietary fat levels in children with hypercholesterolemia causes a small change in LDL cholesterol (−4 mg/dL).

Dietary patterns and fat intake. The availability and accessibility of foods that are prospective choices for a healthy diet must be present in the household to achieve a change in dietary habits. High fat (compared with low fat) foods and sweet foods often are preferred by children. Coercion of children by parents to avoid fatty foods may bias children away from healthy dietary habits. Although teleological, dietary patterns in childhood do not always predict those occurring in adulthood.

Expression of dietary composition goals in terms of fat as a percentage of energy is inadequate in the absence of specification of total energy intake. Survey data of the dietary intake of U.S. children over the last decade demonstrate a decrease from ~36 to 33% of energy from fat. A decrease of similar magnitude was observed over two decades in the Bogalusa Heart Study. National survey data show that fat intake expressed in grams has not declined and has increased in some age-gender groups such as adolescent boys. Cross-sectional survey data show little variation in the level of fat intake as percentage of energy from age 1 y through adolescence. Lower fat diets are associated with a lower intake of calcium, a nutrient consumed by many children at below recommended levels.

Public health messages. An excessive focus on fat can lead to undesirable behaviors by children and parents as well as to misdirected efforts by health-promotion organizations and the private sector food industry. Negative messages using terms such as *avoid* and *limit* and messages using terms that require integration across different foods such as *percentage* or *total fat* are more apt to be ineffective and counterproductive. Positive messages designed to assist consumers select foods for an enjoyable, varied diet appropriate to their lifestyle could result in significant benefit to public health. Given the current level of knowledge, such messages must be empirically designed and adequately tested.

RESEARCH NEEDS

The continuing debate concerning a recommendation of separate dietary guidelines on fat for children makes a strong case for more research on this general topic. Workshop participants offered specific recommendations for research.

1. Research is needed to determine in more detail what children eat, how much they eat and why they eat what they do. There is a paucity of scientific literature on how children respond to nutrition education.
2. New methodologies for assessing dietary intakes are needed. Energy intake is poorly measured as studies using doubly labeled water have demonstrated. Evidence is also accumulating that there is differential reporting of foods and macronutrients in the usual history taking.
3. Better genetic tools are needed to search for particular genes that control responsiveness to specific dietary interventions.
4. Continued funding for longitudinal studies that characterize dietary patterns and the health status of children is needed. Long-term studies of effects of children's diets on their adult dietary habits and health status are also sorely needed.

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