1982). The brain reaches 50 percent of its adult size by age 2, 75 percent by age 4, and 100 percent by age 6 to 10. Body growth is measured by increases in length and weight; skin-fold measurements may be used to evaluate body fat. The heights and weights for representative samples of boys and girls have been used to establish growth reference curves, developed by the National Center for Health Statistics (NCHS) (Hamill and Moore 1976; Hamill et al. 1977; Hamill et al. 1979). As a general rule, growth patterns follow percentile lines on these growth charts.

When growth performance for a given child is measured at regular intervals and plotted on such charts, unusual changes in weight and height percentile can identify children who are at nutritional risk from either under- or overnutrition. Growth patterns are highly individual, however, and average growth rates do not always apply to individual children. This observation is especially applicable in adolescence.

The velocity of physical growth in adolescence is second only to infancy. The most rapid period of growth in adolescence is known as the growth spurt. It precedes menarche in females and spermatogenesis in males. The age at which peak growth occurs is later and the growth spurt more intense in males than in females. There are also differences in its timing and length. The growth spurt usually begins between ages 8 to 12 in females and ages 10 to 14 in males; peak growth velocity occurs at ages 10 to 14 in females and 12 to 16 in males (Lucas, Rees, and Mahan 1985). Linear growth stops toward the end of adolescence, usually between the ages of 14 to 16 in females and 16 to 18 in males (Tanner 1962). The magnitude and duration of growth vary widely among adolescents, making chronologic age an unsatisfactory index of nutritional needs. Instead, individual body size, stage of sexual maturation, and current rate of growth must be considered.

Key Scientific Issues

- Role of Dietary Factors in Maternal Health
- Role of Dietary Factors in Fetal Health
- Role of Dietary Factors in Infant Health
- Role of Dietary Factors in the Health of Children
- Role of Dietary Factors in the Health of Adolescents
- Role of Dietary Factors in Chronic Disease in Childhood

Role of Dietary Factors in Maternal Health

Nutritional Needs of Pregnant and Lactating Women

Extra energy and nutrients are needed to support the growth of maternal tissues, such as the uterus and breast, and the increased metabolic demands of pregnancy as well as the growth of the fetus and placenta. During lactation, the energy and nutrients provided in the milk, and those required for its production, must be replaced.

Weight Gain. Research has demonstrated that both maternal prepregnancy weight and weight gained during pregnancy are important determinants of infant birth weight. Inadequate weight gain during pregnancy and low prepregnancy weight combined with low weight gain are associated with lower-than-average infant birth weights and greater risks for fetal or neonatal death and neonatal disease; these problems decline as weight gain increases.

Such associations do not necessarily prove causality, however; healthy infants of average birth weight have been born to women whose weight change during pregnancy ranged from loss to high gain. For this reason, and because genetic and behavioral factors other than weight gain also influence infant birth weight, it has been difficult to define the optimal weight gain during pregnancy for women of varying prepregnancy weights. Most studies that have correlated average weight gain with birth outcome in normal weight adult women with normal pregnancies have found the optimal weight gain to fall within the range of 22 to 27 lb (Naeye 1979; Rosso 1985), yet one-fifth of white adult women and one-fourth of black adult women did not gain this much weight during normal pregnancies of 40 or more weeks' duration in 1980 (Taffel 1986). When all pregnancies in this study, regardless of length of gestation, were considered, one-third of black mothers gained no more than 20 lb, and black mothers were nearly twice as likely as white mothers to gain less than 16 lb (20 percent as compared with 11 percent). Nevertheless, most of these women gave birth to healthy babies.

The influence of prepregnancy weight on birth outcome also makes it difficult to define an optimal weight gain; a low weight gain by women with a low prepregnancy weight is associated with the highest incidence of LBW, but higher weight gains during pregnancy reduce the risk (Taffel 1986). This relationship may not hold for adolescents, however. Although the mean weight gain of adolescent women during normal pregnancy has been shown in several studies to average about 35 lb, their risk of having a

LBW infant was still higher than that for adult women (Meserole et al. 1984; Frisancho, Matos, and Flegel 1983; Garn et al. 1984; Loris, Dewey, and Poirier-Brode 1985).

The effect on infant birth weight of differing levels of weight gain by obese women during pregnancy is also uncertain. Obesity during pregnancy is linked to an increased risk of maternal complications such as hypertensive disorders, gestational diabetes, infections, and surgical deliveries (Abrams and Laros 1986; Kliegman and Gross 1985; King 1986; Garbaciak et al. 1985). Obesity is also associated with higher rates of infant mortality (Naeye 1979). These risks are reduced when obese women do not gain more than 24 lb during pregnancy (Naeye 1979; Kliegman et al. 1984; Abrams and Laros 1986; Garbaciak et al. 1985), but the lower limit of weight gain that can produce optimal birth outcome has not been defined. This issue is important because caloric restriction and diet-induced weight loss in pregnant obese women has been associated with reduced infant birth weight (Naeye 1981; Campbell and MacGillivray 1975; Grieve, Campbell-Brown, and Johnstone 1979), suggesting that obese women should gain at least some weight during pregnancy (Campbell 1983).

Energy. The recommended intake of energy during pregnancy includes an increment of 300 kcal/day over the energy allowances for nonpregnant women (NRC 1980). This amount was obtained by calculating the total energy cost of synthesizing maternal and fetal tissues and dividing this figure by the number of days of pregnancy (Hytten and Leitch 1971). Although the 300 kcal/day increment has been widely accepted, actual measurements of the energy balances of pregnant women in several international populations have demonstrated that they may consume as few as 50 kcal/day over prepregnancy energy intake levels yet gain weight normally and produce infants of normal birth weight (Durnin 1987). Although the reason for this discrepancy is not yet known, preliminary observations suggest that the metabolic needs of pregnant women do not increase significantly (Durnin et al. 1985; McNeil and Payne 1985; Saha 1986). Nevertheless, caloric supplementation of pregnant women in a poor community where the average caloric intake was 1,500 kcal/day increased the average birth weight and reduced the incidence of LBW infants (Lechtig et al. 1975).

Recent investigations show that lactating women also require fewer calories than the 500 kcal/day calculated as needed above prepregnant energy levels (Butte, Garza, Stuff, et al. 1984). Present knowledge thus does not permit the precise definition of caloric requirements during pregnancy and lactation (Worthington-Roberts, Vermeersch, and Williams 1985).

Protein. The RDA for protein during pregnancy includes an additional 30 g/day beyond the 44 g/day recommended for nonpregnant women (NRC 1980). Protein is abundant in the American diet, and inadequate intake during pregnancy is reported infrequently. Studies that have associated diets containing 20 percent of total calories from protein (as compared with the 12 to 14 percent usually recommended) with a higher risk of premature deliveries and neonatal mortality suggest that protein intakes significantly higher than those recommended may be harmful (Rush, Stein, and Susser 1980a, 1980b).

Vitamins and Minerals. The RDA for vitamins and minerals for pregnant or lactating women include increased levels above those for nonpregnant women (NRC 1980). In healthy women with normal pregnancies, vitamin and mineral needs can usually be met by consuming an adequate diet. With the possible exceptions discussed below, supplements, although usually recommended, have not been associated with measurable health improvements in this population (Hemmiki and Starfield 1978).

American women may have a low intake of dietary folate. Some European studies have suggested an association of folate deficiency (along with deficiencies of other vitamins) with the development of neural tube defects. These observations stimulated clinical trials in which folate and multivitamin supplements appeared to reduce the risk of neural tube defects in subsequent children born to women who had previously given birth to infants with these defects (Smithells et al. 1981; Smithells et al. 1983; Laurence et al. 1981). These studies, however, were poorly controlled and flawed methodologically (Dobbing 1983). A recent report has found no difference in folate or vitamin B_{12} concentrations in blood samples from early pregnancy taken from mothers of infants with or without neural tube defects (Molloy et al. 1985).

The need for iron is increased during pregnancy, with about 500 mg needed to increase the number of maternal red blood cells, about 600 mg needed for the fetus and placenta, about 200 mg needed to replace normal maternal losses, and another 200 mg needed to replace the red blood cells lost during delivery. Because menstruation ceases for 9 months, about 300 mg of iron are saved, and the total additional iron needed for a pregnancy with a normal delivery is about 1,200 mg (King 1986), or about 4 mg/day. As discussed in the anemia chapter, iron deficiency occurs infrequently in the general population. Nevertheless, the National Research Council recommends an iron supplement of 30 to 60 mg/day to prevent depletion of iron stores during pregnancy and lactation (NRC 1980). Even when women are not anemic, they may complete pregnancy with low iron reserves (Taylor et

al. 1982). Clinical studies indicate that iron absorption increases progressively throughout pregnancy (Lind 1982). Although as much as 90 percent may be absorbed under these defined experimental conditions, the true efficiency of dietary iron absorption under normal situations of food intake is unknown (King 1986). For these reasons, pregnant women should be evaluated periodically to determine their level of iron stores and should receive supplements when stores are low (Romslo et al. 1983).

The RDA for zinc includes a 5 mg/day increment during pregnancy over the 15 mg/day recommended for nonpregnant women (NRC 1980). Zinc deficiency is teratogenic in rats (Hurley 1980, 1981), and abnormal brain development and behavior have been described in the offspring of zinc-deficient monkeys (Sandstead et al. 1977, Sandstead et al. 1978). Evidence from human populations suggests that the rate of fetal malformations and other poor outcomes of pregnancy may be higher in populations where zinc deficiency has been recognized (Sever and Emanuel 1973; Cavdar et al. 1980; Bergmann, Makosch, and Tews 1980; Jameson 1976; Soltan and Jenkins 1982; McMichael et al. 1982; Kiilholma et al. 1984; Buamah, Russell, and Bates 1984). However, conflicting reports have also been published (Mukherjee et al. 1984; Hunt et al. 1984; Ghosh et al. 1985), and questions remain about satisfactory measures of zinc status. The role of zinc deficiency in the adverse outcome of human pregnancy remains uncertain at present (Swanson and King 1987), and supplements are not recommended.

Adolescent Pregnancy

Maternal age influences fetal and infant mortality rates and birth weight. Mothers 15 years of age or younger (approximately 60,000 per year) have increased rates of pregnancy-induced hypertension and premature delivery, are more likely to deliver infants of LBW, and have higher rates of fetal loss and infant mortality (AAP Committee on Nutrition 1985b). In 1985, among live-born infants in the United States with birth weights under 2,500 g, 0.5 percent were born to mothers under 15 years of age and 6.7 percent to those 15 to 17 years of age (NCHS 1987c). A high proportion of adolescent mothers are nonwhite and of low socioeconomic status (IOM 1985). While nutrition is by no means the only issue in caring for the pregnant adolescent, it is a controllable risk factor that can be reduced by programs that provide support, prenatal care, and extra food (Heald and Jacobson 1980).

Disorders During Pregnancy

Pica. Pica, a persistent compulsion to eat unsuitable substances of little or no nutritional value, is a recognized complication of pregnancy. Although

the pica of pregnancy most often involves consumption of dirt or clay (geophagia) or of starch (amylophagia), compulsive ingestion of many other nonfood substances has been noted. Examples include ice, burnt matches, hair, stone or gravel, charcoal, soot, cigarette ashes, mothballs, antacid tablets, milk of magnesia, baking soda, coffee grounds, and the inner tubes of tires. The practice of pica is not new, nor is it limited to any geographical area, race, or culture. The medical implications of pica are not well understood. Inadequate absorption of iron is the hazard most commonly attributed to pica. Other less commonly reported complications include congenital lead poisoning resulting from the consumption of wall plaster (Pearl and Boxt 1980) and small bowel obstruction from the ingestion of laundry starch (Worthington-Roberts, Vermeersch, and Williams 1985). These issues are discussed in more detail in the chapter on behavior.

Hypertension. Preeclampsia and eclampsia are hypertensive conditions induced by pregnancy. Preeclampsia is characterized by a rise in blood pressure, generalized edema that may cause sudden, large weight gain from retained water, and loss of protein in the urine. Eclampsia is the most severe form of the disorder, characterized by convulsions that may lead to coma. Both present serious health risks to mother and fetus (Lindheimer and Katz 1985). Pregnancy-induced hypertension occurs among young women (especially those under 15 years), primiparas, especially older primiparas, and those who are underweight or of low socioeconomic status (Pritchard and MacDonald 1980). The cause of these conditions is uncertain. Whether dietary variables may be part of the cause cannot be determined from available data. Hypertension existing before pregnancy may also adversely affect pregnancy.

Diabetes. Infants born to women with diabetes are at greatly increased risk for prematurity, congenital defects, excessively high birth weight, respiratory distress syndrome, and other conditions that increase overall mortality, especially when the mother's blood glucose levels remain high during pregnancy (see chapter on diabetes). Risks of maternal complications are also increased by established diabetes. These problems are reduced by maintaining strict control of blood glucose levels before and throughout pregnancy (Freinkel, Metzger, and Potter 1983); dietary counseling is important in helping patients to achieve such control (Fuhrmann et al. 1983).

In some women who are not otherwise diagnosed as diabetic, pregnancy is associated with significant alterations in carbohydrate metabolism such that overnight fasting leads to lower-than-normal blood sugar and insulin levels and carbohydrate consumption leads to higher-than-normal levels

(Kalhan and Hertz 1985). These changes, which are most marked near the end of pregnancy, are referred to as gestational diabetes (Second International Workshop 1985). Because current diagnostic methods cannot easily distinguish between established and gestational diabetes, it is uncertain whether gestational diabetes itself—in the absence of preexisting undiagnosed diabetes—increases risks to mothers and infants (Scherger and Hudson 1985.)

All authorities agree that diabetes screening is indicated for pregnant women with risk factors for overt diabetes: a previous history of gestational diabetes, a large-for-gestational-age infant, excess amniotic fluid during pregnancy, excretion of sugar in urine, increased thirst or urination, or recurrent vaginal or urinary tract infections. Because diabetes screening can identify diabetes in mothers who may not have been diagnosed previously and who might benefit from preventive services, various authorities recommend screening for all pregnant women (Second International Workshop 1985; CDC 1986), including administration of an oral glucose tolerance test between the 24th and 28th weeks of pregnancy, or earlier for women with risk factors. Others, however, believe that current understanding of gestational diabetes is insufficient to justify universal screening at this time (Scherger and Hudson 1985). Dietary components of prenatal care of women found to have diabetes or gestational diabetes are discussed in the chapter on diabetes.

Role of Dietary Factors in Fetal Health

Nutritional Needs of the Fetus

Because no one animal model is appropriate for all studies of the human fetus, and because direct investigation of the fetus is limited by ethical and technological considerations, knowledge of the nutrient needs of the human fetus is fragmentary. This section reviews information about fetal and placental nutrient requirements and their response to maternal nutrition.

Energy. Current evidence indicates that glucose and amino acids, rather than fatty acids, are the primary metabolic fuels for the fetus. Fetal energy requirements increase during pregnancy to a maximum estimated from 110 kcal/day (Widdowson 1981) to 150 kcal/day (Rosso 1983) near term. These are modest values when compared with the mother's estimated energy requirement of 2,000 to 2,800 kcal/day (Widdowson 1981).

The glucose concentration in the maternal circulation is always higher than that in the umbilical vein, and maternal glucose levels greatly influence the

rate of glucose transfer across the placenta. For example, in a woman with diabetes and chronic hyperglycemia, larger amounts of glucose than normal are transported to the fetus. Conversely, during maternal caloric restriction, the rate of transfer may be markedly reduced. The placenta uses about half of the oxygen and glucose that are removed from maternal circulation (Nichols and Nichols 1983), and there is some evidence that the glucose needs of the placenta are met preferentially to those of the fetus (Jones and Rolph 1985).

Protein. A fetus near term requires about 6 g (Widdowson 1981) to 8 g (Rosso 1983) of protein per day. Most of this comes as small amounts of essential and nonessential amino acids received continuously from the placental circulation. The free amino acid concentrations in the umbilical artery and vein are higher than those in the mother's blood, indicating that amino acid transport across the placenta is an active metabolic process. Presumably, active transport of amino acids protects the fetus against inadequate maternal protein intake. Another source of protein occurs in amniotic fluid the fetus swallows. This could amount to as much as 750 mg/day. Animal studies indicate that glucose and amino acids can be taken up from amniotic fluid more rapidly than can be accounted for by swallowing alone (Charlton 1984). Administration of nutrients directly into the fetal stomach can normalize the birth weight and length of fetuses born to nutritionally deprived dams (Charlton and Johenger 1985).

Vitamins. Vitamin requirements of the human fetus have not been established. Fat-soluble vitamins cross the placenta by simple diffusion, so that maternal dietary intake would be expected to influence fetal levels. Most water-soluble vitamins are transported from the placenta to fetus by active uptake processes, so that fetal blood vitamin concentrations are higher than those in maternal blood. Although specific vitamin deficiencies have been shown to induce reproductive loss and developmental defects in experimental animals, similar data for human fetuses are not available.

Fetal Risk From Vitamin A. Isotretinoin is a vitamin A acid that is known to cause birth defects or developmental disability in 25 percent of children exposed to it in the first trimester of pregnancy. There are case reports of birth defects associated with large doses of vitamin A. Moreover, vitamin A is well known to cause birth defects in laboratory animals. It has been suggested that women who are pregnant or likely to become pregnant should avoid taking supplements containing more than the RDA level (see introduction and background chapter) for pregnant women (CDC 1987).

Minerals. Mineral requirements of the fetus, estimated from studies on fetal body composition, seem to be higher during the last few weeks of pregnancy than at any other time during prenatal or postnatal development (Widdowson 1981). Little is known, however, about the specific needs for individual minerals. In experimental animals, prenatal calcium deficiency causes rickets in the infant. In animals and humans, iron is stored in fetal red blood cells, and term infants are born with ample reserves. Because premature infants have a smaller red cell mass, they are at increased risk for iron deficiency after birth.

Effects of Other Dietary Factors

Alcohol. During the past 15 years, health researchers have become aware that excessive alcohol consumption adversely affects fetal development. Fetal alcohol syndrome (FAS) was first described among infants born to women who were chronic alcoholics (Jones 1973). These infants exhibited specific abnormalities of the eyes, nose, heart, and central nervous system; irritability and hyperactivity after birth as a result of alcohol withdrawal; and impaired physical and mental development despite nutritional rehabilitation. The minimum level of alcohol consumed during pregnancy that causes FAS has not been established. Some studies show that even one or two alcoholic drinks per day are associated with higher rates of spontaneous abortion, premature detachment of the placenta, and LBW infants (Council on Scientific Affairs 1983). Other studies, however, find that this level of alcohol intake during pregnancy does not increase congenital malformations (Mills and Graubard 1987). Because it is difficult to obtain accurate information about alcohol intake (see chapter on alcohol), this issue is not easy to resolve. Thus, as a prudent measure, the Surgeon General has suggested that women avoid alcohol use during pregnancy (FDA 1981).

The mechanism by which alcohol injures the fetus has not yet been defined. Alcohol can cross the placenta and produce direct toxic effects. Another possible mechanism may be the induction of nutrient deficiencies similar to those that have been shown to produce fetal abnormalities in experimental animals (Hurley 1980).

Although the adverse effects of maternal alcohol consumption on fetal development have been known for more than a decade, a significant number of women still consume alcohol while they are pregnant. For example, one study of women delivering in a large urban hospital reported that 82 percent consumed alcohol during pregnancy (Lillien, Huber, and

Rajala 1982). Because pregnancy course and outcome can be significantly improved if problem drinkers change their habits after conception (Rosett 1983; Rosett, Weiner, and Edelin 1983), routine health and prenatal care should include counseling about the hazards of alcohol use.

Caffeine. Caffeine, which is contained in many widely consumed beverages, does cross the placenta and reach the fetus. Caffeine is an animal teratogen, but there is no clear epidemiologic evidence of a similar effect in humans. Exposure of the pregnant rat to bolus doses of caffeine by intubation, approximately 80 mg/kg or above, results in signs of maternal toxicity, increased resorptions, decreased fetal weight and size, teratogenic effects involving limbs, and skeletal ossification deficiencies (Collins et al. 1981). At doses as low as 6 mg/kg, there is little evidence of maternal or fetal toxicity, although the offspring do display delayed skeletal ossification, a condition that has been shown to be reversible (Collins et al. 1987). The importance of the dosing method in producing teratogenic and other effects is reflected in that caffeine given to pregnant rats at doses of 204 mg/kg in the drinking water results in no teratogenic effects, although signs of maternal and fetal toxicity occur at doses of 87 mg/kg/day, and delayed skeletal ossification occurs at doses as low as 27 mg/kg/day (Collins et al. 1983).

Other studies in rodents have also been conducted to determine the effects of perinatal exposure to caffeine on the neurobehavioral development of offspring. Evidence indicates that gestational exposure of rodents to caffeine either in drinking water or by intubation may affect neurofunctional development at doses of approximately 25 mg/kg/day or greater (Glavin and Krueger 1985; Butcher, Vorhees, and Wootten 1984; Holloway and Thor 1982; West et al. 1986; Peruzzi et al. 1983).

Human studies have also produced inconsistent results. A study of 5,200 maternity cases in Germany reported lower birth weights in women with "high" coffee consumption, regardless of cigarette smoking, socioeconomic class, age, or number of children (May and Netter 1974). A survey of 800 Mormon households found that women who drank coffee had a higher rate of spontaneous abortions, but this effect was not related to dose (Weathersbee, Olsen, and Lodge 1977). Japanese researchers studied the effects of caffeine ingestion in a sample of 9,921 healthy women pregnant for 6 months or longer and reported that women who drank more than five cups of coffee per day had a higher incidence of premature labor, fetuses small for gestational age, and infants with birth defects, but this study did not control for socioeconomic variables (Furuhashi 1985). Other recent reports have found caffeine intake to be associated with increased risk of late first

and second trimester spontaneous abortion (Srisuphan and Bracken 1986) and reduced infant birth weight (Martin and Bracken 1987).

Other studies, however, have suggested that coffee drinking has little or no effect on fetal health. A study of over 20,000 pregnancies absolved coffee drinking from contributing to fetal defects or other untoward outcomes of pregnancy (Van den Berg 1977). An analysis of interview and medical record data for over 12,000 pregnant women evaluated the relation between coffee consumption and adverse outcomes of pregnancy, controlled for smoking and other relevant variables, and found no effect on birth weight, gestation period, or birth defects (Linn et al. 1982). Similarly, a case-control study of over 200 infants with selected birth defects found that the caffeine consumption patterns of their mothers did not differ significantly from those of control mothers (Rosenberg et al. 1982). Finally, an examination of almost 500 cases of birth defects recorded in Finland showed no difference in coffee consumption between the mothers of these infants and control mothers (Kurppa et al. 1982). Although data from human populations do not provide significant evidence that caffeine affects pregnancy outcome, the Food and Drug Administration advises pregnant women to eliminate or limit consumption of caffeine-containing products as a precautionary measure (FDA 1980).

Role of Dietary Factors in Infant Health

Nutritional Needs of Normal Infants

The nutritional requirements of normal infants have been investigated to the extent possible with this population, and RDA's have been established (NRC 1980). These recommendations are largely based on analyses of human milk and on the nutrient content of foods that healthy, thriving infants consumed. A number of studies have measured nutrient needs directly. The best measure of the adequacy of an infant's diet is the growth rate in length and weight. This is usually evaluated by plotting the data from repeated measurements over time on standard NCHS charts (Roche and Hines 1980; Baumgartner, Roche, and Hines 1986).

Energy. Infants require three to four times greater amounts of energy per kilogram of body weight (90 to 120 kcal/kg/day) than do adults to support their relatively high metabolic rate and needs for growth (Heird and Cooper 1988). Energy requirements of individual infants are determined by body size and composition, rates of physical growth, and activity patterns. The RDA of 115 kcal/kg/day in the first 6 months and 105 kcal/kg/day for the second 6 months meets the needs of most normal, well-nourished infants, but infants' needs vary considerably, and the energy intake of any one

infant may vary from day to day. Normal infants appear to adjust intake to needs, provided the mother is sensitive to cues of satiation: loss of interest in food, releasing the nipple from the mouth, or turning the head from the nipple or pushing the bottle or cup away. Many infants who are growing normally consume less energy than RDA levels. One study, for example, found the average intakes of breastfed infants to decline from 110 kcal/kg/day at 1 month to 71 kcal/kg/day at 4 months (Butte, Garza, Smith, et al. 1984).

Water. The evaporative, fecal, and urinary water losses determine water requirements for infants. Evaporative losses in healthy, full-term normal infants range from 30 to 70 ml/kg/day. Fecal losses average 1 to 4 ml/kg/day (up to 10 ml/kg/day in breastfed infants) but can increase suddenly and vary greatly in infants with diarrhea. Because of their large surface area relative to mass, infants lose proportionately more water by skin evaporation than do adults. Infants also have less renal concentrating capacity than adults. Thus, infants are vulnerable to water imbalance. Nevertheless, breast milk and infant formulas provide adequate water, and healthy infants rarely require supplemental water except in very hot weather (AAP Committee on Nutrition 1985b). Instead, dehydration problems are most likely to occur during episodes of vomiting, diarrhea, or fever, when dehydration can occur so suddenly as to constitute a medical emergency (Fomon 1974).

Protein. Protein requirements are also proportionately greater in infants than in adults. Dietary protein must be sufficient to support increases in body protein that range on average from 3.7 g/day for the first month in males and 3.3 g/day for the first month in females down to 1.8 and 1.7 g/day, respectively, for months 9 to 12. These needs generally can be met by protein intakes of about 1.8 g/100 kcal for infants during the first month of life, decreasing to 1.2 g/100 kcal for infants 4 to 6 months of age (Fomon et al. 1986). The Infant Formula Act of 1980 established a minimum standard for the protein content of infant formulas, 1.8 g/100 kcal, which is based on a protein equivalent in nutritive quality to casein (AAP Committee on Nutrition 1976). The RDA of 2.2 g/kg for the first 6 months and 2.0 g/kg for months 7 to 12 also considers protein quality (NRC 1980).

Fat and Carbohydrate. Because dietary fat is a concentrated source of the calories needed to meet infants' high energy needs, infants should consume 3.8 to 6.0 g of fat per 100 kcal, or 34 to 54 percent of total calories (Fomon 1974). Dietary fat is required for normal brain development. Three percent of total calories should be provided by linoleic acid, an essential fatty acid (AAP Committee on Nutrition 1976). Carbohydrate should supply 30 to 60 percent of the total daily energy intake in infancy.

Vitamins and Minerals. Although infant requirements for micronutrients are not as well defined as those for energy and protein (Heird and Cooper 1988), RDA's have been established for many vitamins and minerals (NRC 1980). Full-term infants who obtain breast milk from a well-nourished mother will receive most necessary vitamins (AAP Committee on Nutrition 1985b), although, under some circumstances, vitamins D and K are exceptions. If breastfed infants have limited exposure to sunlight, vitamin D supplementation may be required to prevent rickets (Reeves, Chesney, and DeLuca 1982). Human milk is low in vitamin K, and this vitamin should be routinely administered parenterally to all infants at birth to prevent bleeding disorders (AAP Committee on Nutrition 1985b). Infants fed a commercially available formula that is properly prepared should receive an adequate intake of vitamins (AAP Committee on Nutrition 1976).

The bioavailability of minerals generally is greater from human milk than it is from formulas. The RDA for calcium is designed to meet the need of formula-fed infants, who retain 25 to 30 percent of the calcium in cow milk-based formula. Breastfed infants retain about 65 percent of the calcium consumed. Studies suggest that the bioavailability of zinc from human milk is 41 percent, compared with 28 percent from cow milk, 31 percent from cow milk formulas, and 14 percent from soy formula (Sandstrom, Cederbald, and Lonnerdal 1983).

Iron deficiency is the most common nutrient deficiency in infancy (see chapter on anemia). Rapidly growing infants absorb 49 percent of the iron in human milk, 10 percent of the iron in cow milk, and 3 percent of the iron in iron-fortified formulas (Saarinen, Siimes, and Dallman 1977). For this reason, some authorities believe that breastfed infants do not need additional iron until the age of about 6 months (Saarinen 1978). Current recommendations are that infants begin consuming iron-fortified cereals at 4 to 6 months of age to prevent anemia (Garry et al. 1981; AAP Committee on Nutrition 1985b). Because human milk is low in fluoride and because enamel development in permanent teeth is significant during the first year of life, a fluoride supplement may be desirable for children who do not have access to adequately fluoridated drinking water (see chapter on dental diseases). Requirements for other minerals have been reviewed (AAP Committee on Nutrition 1985b).

Infant Feeding

The available information on infant feeding has been reviewed extensively and was the subject of a 1984 report commissioned by the Assistant Secretary for Health (Task Force 1984).

Human Milk. Human milk is the food of choice for infants. It provides appropriate amounts of energy and nutrients, it contains factors that provide protection against infections, and it rarely causes allergic responses.

Breastfeeding of the newborn in the hospital increased from 24.7 percent in 1971 to 62.5 percent in 1984, but only 27.5 percent of infants are still breastfed by 6 months of age (Martinez and Kreiger 1985). Rates of breastfeeding are highest among mothers who live in the western part of the United States, have had at least some college education, are from upper income families, and are white. Numerous investigations and reports have discussed the unique values of human milk, trends in rates of breastfeeding in the United States, and methods for promoting breastfeeding (DHHS 1984).

Table 15-2 compares the average content of selected nutrients in human milk, infant formulas, and other kinds of milk used to feed full-term infants (AAP Committee on Nutrition 1985b). The nutrient content of human milk reflects the maternal diet, the time of day the milk is expressed, and the length of time the mother has been breastfeeding. Protein concentrations, for example, decrease from the first week through the sixth to ninth month (Anderson, Atkinson, and Bryan 1981), and the concentrations of iron, copper, and zinc also decrease during that period (Vuori and Kuitunen 1978; Siimes, Vuori, and Kuitunen 1979).

Fat provides about 50 percent of the calories in human milk, most in the form of triglyceride, with the fatty acid pattern reflecting the maternal diet. Linoleic acid provides an average of 4 percent of the calories in human milk. The cholesterol content averages 20 mg/100 ml but varies considerably, and values up to 47 mg/100 ml have been reported (Jensen, Hagerty, and McMahon 1978). Lactose is the major carbohydrate. Human milk has a lower content of the amino acids tyrosine and phenylalanine and a higher content of taurine and cystine than cow milk.

The concentrations of water-soluble vitamins in human milk generally reflect the maternal dietary intake and nutritional status. Providing folate supplementation to a woman deficient in this vitamin increases milk folate levels (Cooperman et al. 1982). Vitamin B_{12} deficiency has been reported in breastfed infants whose mothers are strict vegetarians (Johnson and Roloff 1982). As noted above, breastfed infants require supplemental vitamin K at birth and may require vitamin D supplementation if exposure to the sun is inadequate.

Table 15-2
Content of Selected Nutrients in Human Milk, Commercial Formulas, and Other Milks Used for Feeding Normal Full-Term Infants

Nutrient	Per Liter	Mature Human Milk[a] (21.6 ± 1.5 kcal/oz)	Milk-based Formulas[b] (20 kcal/oz)	Soy Protein-based Formulas (20 kcal/oz)	Whole Cow Milk (20 kcal/oz)	Skimmed Milk (11 kcal/oz)	Goat Milk (21 kcal/oz)
Protein	g	10.5 ± 0.2	15	18–21	34	35	37
Fat	g	39.0 ± 0.4	36–38	36–39	37	2	43
Carbohydrate	g	72.0 ± 0.25	69–72.3	66–69	48	50	46
Calcium	mg	280 ± 26	400–510	630–700	1,219	1,270	1,380
Phosphorus	mg	140 ± 22	300–390	420–500	959	1,050	1,140
Sodium	mEq	7.8 ± 1.7	7–10	9–15	22	23	23
Potassium	mEq	13.4 ± 0.9	14–21	19–24	38	44	54
Chloride	mEq	11.8 ± 1.7	11–14	11–15	27	31	44
Iron	mg	0.3 ± 0.1	1.1–1.5 (12–12.7)[c]	12–12.7	0.4	0.4	0.5
Estimated renal solute load	mOsm	73	92–105	122–138	226	240	269

[a] Average values with standard deviations for comparison.
[b] Values listed are subject to change. Refer to product label or packaging for current information. Milk-based formulas contain lactose, and soy protein-based formulas do not.
[c] Iron content of iron-fortified formulas.

Source: Adapted from the American Academy of Pediatrics Committee on Nutrition 1985b.

In addition to nutrients, human milk contains antibodies and other anti-infective factors that are thought to protect infants against gastrointestinal infections. For example, lactoferrin may slow bacterial growth by depriving infective organisms of necessary iron. Lysozymes may destroy bacterial cell membranes after they have been inactivated by peroxidases and ascorbic acid present in human milk. Secretory immunoglobulins in milk protect against organisms that infect the gastrointestinal tract (Welsh and May 1979). Perhaps most important, breast milk fosters colonization of the infant digestive tract with protective *Lactobacilli*. In addition, a number of other cellular and soluble factors may provide specific and nonspecific defenses against infectious agents (Ogra and Greene 1982).

Infant Formulas. The Food and Drug Administration specifies the nutrient composition of commercial infant formulas (CFR, Title 21, Section 107.100—Nutrient Specifications). As suggested by Table 15-2, infant formulas are designed to simulate human milk. Manufacturers modify cow milk by replacing its fat with vegetable oils that are well absorbed, diluting it to a more appropriate concentration of minerals and other solutes, heating it to improve protein digestibility, and adding vitamins and minerals. Soy-based substitutes are available for infants who develop allergic or other sensitivities to substances in cow milk-based formulas. Soy formulas contain isolated soy protein, methionine, corn syrup or sucrose, vegetable oils, vitamins, minerals, carnitine, taurine, and stabilizers. Other products are available for infants who cannot tolerate either soy or cow milk. When properly prepared, commercial formulas support normal growth and development. Errors in preparation, however, have resulted in medical problems. Inadequately diluted formula increases the concentrations of calories, protein, and solutes circulated to the kidney for excretion and can increase levels of sodium and other substances in blood, resulting in disturbances of acid-base balance and toxic symptoms (Chambers and Steel 1975). Overdiluting the formula reduces the level of sodium and other salts in the blood, thereby causing adverse reactions (Partridge et al. 1981), and it does not provide adequate energy and nutrients for growth.

Imitation Milks. Substitute or imitation milks inadequate in calories and nutrients are not suitable for feeding to infants (AAP Committee on Nutrition 1984). Malnutrition has been observed in infants fed a formula made of barley water, corn syrup, and whole milk (Fabius et al. 1981) and in those fed nondairy creamer (Sinatra and Merritt 1981).

Cow Milk. Unmodified whole cow milk is inappropriate to feed to young infants because it causes occult bleeding from the gastrointestinal tract in some infants, leading to anemia and, occasionally, to allergies (see chapter

on infections and immunity). Its lipids are less digestible than the lipids of human milk or most vegetable oils, and its concentrations of minerals and other solutes nearly exceed the excretory capacity of the immature kidney (AAP Committee on Nutrition 1983b). Intestinal blood loss has been noted in younger infants and those who have consumed large volumes of milk, although this usually presents no problem after the age of 6 months or so (Fomon et al. 1981). As indicated in Table 15-2, cow milk has a higher renal solute load than either human milk or infant formulas. By 6 months of age, the kidneys of most normal infants have matured and can excrete excess solutes sufficiently. If infants receive at least one-third of their calories from foods and consume no more than 1 liter of milk per day, there is little disadvantage to the use of whole cow milk after 6 months of age (AAP Committee on Nutrition 1983b). Sometimes, however, infants consuming whole cow milk may not receive adequate dietary iron to meet their nutritional requirements (Tunnessen and Oski 1987).

For infants, 2-percent and nonfat milks are deficient in energy, essential fatty acids, and certain vitamins, and they contain excessive protein and minerals per calorie provided. They are not recommended during the first year of life (AAP Committee on Nutrition 1985b; Fomon et al. 1977). When supplemented with vitamin C and iron, evaporated milk formula (3 oz of evaporated milk, 4.5 oz of water, 2 tsp of corn syrup) is an acceptable, low-cost substitute for infants less than 6 months of age (Fomon et al. 1979).

Goat Milk. Goat milk should be used carefully during infancy because it is low in iron, folate, and vitamins B, C, and D (AAP Committee on Nutrition 1985b). It requires supplementation with these nutrients. As shown in Table 15-2, solute concentration is even higher than in cow milk (Harrison et al. 1979).

Solid Foods. By the age of 4 to 6 months, infants have usually matured enough to sit and to control movements of the head, tongue, lips, and jaw. They can indicate when they do and do not want to eat. At this point, feeding pureed solid foods becomes appropriate. The recommended routine is to introduce single-ingredient foods to the diet, one at a time, at weekly intervals (AAP Committee on Nutrition 1980a, 1985b). Iron-supplemented cereals are usually the first foods added. If properly prepared and stored, pureed foods made at home are nutritionally equivalent to those prepared commercially. By the age of 1 year, foods should provide more than 50 percent of the energy intake of infants (Montalto, Benson, and Martinez 1985). Salt need not be added to foods prepared for normal infants, and sugar should be added sparingly, or not at all. Infants should not be fed hot dogs, nuts, grapes, popcorn, uncooked carrots, round

candies, and similar food items that can cause choking (AAP Committee on Nutrition 1985b).

Low Birth Weight Infants

Infants born prematurely or after intrauterine growth retardation are at high risk for malnutrition and may require special feedings. Before 26 weeks of gestation, the fetus's gastrointestinal system is too immature to digest proteins, fats, or lactose. Fully competent digestive processes do not develop until about 32 to 36 weeks of gestation. Infants born prior to 32 to 34 weeks of gestation may not be able to suck effectively. LBW infants may have medical problems such as necrotizing enterocolitis (a potentially lethal bacterial infection of the intestine), fluid and electrolyte imbalances, and respiratory distress syndrome (difficulty exchanging oxygen and carbon dioxide due to immature lungs) that increase needs for energy and nutrients (AAP Committee on Nutrition 1977). Whether the goal of nutritional support for such infants should be to maintain normal (AAP Committee on Nutrition 1985a) or slower-than-normal weight gain (Keen and Pearse 1985; Steen 1985) is as yet undecided. The challenge is to provide adequate calories and nutrients in a form that the immature digestive and excretory systems can handle and that does not cause complications.

Providing 95 to 160 kcal and about 3 g of protein per kg per day (maintained at about 10 percent of calories ingested as the infant grows), 2 to 4 percent of calories as linoleic acid, 40 to 50 percent of calories as carbohydrate, and sufficient water to compensate for the unusually high losses from skin helps achieve adequate nutrition for LBW infants (Heird and Cooper 1988). Mineral and electrolyte intake may also need to be adjusted to compensate for these infants' unusual requirements (AAP Committee on Nutrition 1985a). Human milk and formulas designed for full-term infants contain insufficient calcium and phosphorus to meet the needs of LBW infants and must be supplemented to permit adequate bone growth and mineralization (Greer, Steichen, and Tsang 1982). Because iron supplements increase susceptibility to vitamin E deficiency, recommendations for iron supplementation in the LBW infant are cautious, advising 1 mg of iron per 100 kcal (Heird and Cooper 1988). Recommendations for vitamins are generally the same as those for full-term infants, although fat-soluble vitamins may pose special problems due to poor fat absorption. Preventing bone disease in LBW infants depends not only on adequate calcium and phosphorus, but also on an intake of at least 500 IU of vitamin D per day (AAP Committee on Nutrition 1985a). Vitamin E requirements may be higher for LBW infants than for term infants. Although 15 to 30 IU of supplementary vitamin E per day has been suggested for LBW infants

(AAP Committee on Nutrition 1985a), the data are not sufficient to support firm recommendations (CDC 1984). The recommendation for folate in LBW infants is 50 μg/100 kcal (Heird and Cooper 1988). This vitamin must be added separately to liquid multivitamin preparations because of its instability (AAP Committee on Nutrition 1985a).

Methods for meeting nutritional goals for LBW infants usually include a combination of human milk (from the mother or pooled from other sources); other special supplements, formulas, or products fed by mouth or tube (enteral nutrition); or intravenous feeding (parenteral nutrition). Milk from mothers of LBW infants contains more energy; has higher average concentrations of protein, fat, and sodium; has lower concentrations of lactose and phosphorus; and contributes to a more rapid growth rate than does milk produced by term mothers (Anderson, Atkinson, and Bryan 1981; Atkinson et al. 1980; Atkinson, Bryan, and Anderson 1981; Chessex et al. 1983; Gross et al. 1981; Schanler and Oh 1980). Human milk also has the advantage of passive transfer of antibodies. Some LBW infants can be nourished adequately on their mother's milk, whereas others thrive better when provided with additional supplements. One such supplemental product, a human milk fortifier that contains protein, carbohydrate, calcium, phosphorus, trace minerals, and vitamins, has been associated with improved growth and prevention of bone demineralization in LBW infants (Ronnholm, Spila, and Siimes 1982).

Special formulas for LBW infants contain more protein, calcium, and phosphorus than formulas for term infants (Atkinson 1983). Although the composition of these formulas varies, all can support growth (AAP Committee on Nutrition 1977; Gross 1983). The composition of several formulas for premature infants are provided in a publication of the American Academy of Pediatrics Committee on Nutrition (AAP Committee on Nutrition 1985a).

Parenteral nutrition is often used in the first few days or weeks of life to support anabolism in LBW infants who cannot tolerate full enteral nutrition. Amino acids are usually used as the nitrogen source, and concentrations administered are limited to those that do not produce elevated plasma levels or the accumulation of ammonia in the blood (Heird and Cooper 1988). Glucose concentration must be adjusted to prevent hyperglycemia and local irritation of peripheral veins. Excessive lipids must be avoided to mitigate hyperlipidemia. Vitamins, minerals, and electrolytes must be added to meet individual requirements (AAP Committee on Nutrition 1983a, 1985b).

Cholestasis, or a suppression of the flow of bile, is a common complication of prolonged parenteral nutrition in LBW infants. While the etiology of this condition is unknown, the degree of prematurity and length of time on parenteral nutrition are important correlates (Gibbs 1980). Lack of oral feedings may also play a role. Although this complication is often reversible when parenteral administration is discontinued, it may progress to hepatic toxicity, cirrhosis, and liver failure (Reynolds 1985).

Role of Dietary Factors in Child Health

Energy

The energy requirements of children are determined by their individual basal metabolic rates, rates of growth, and activity patterns. Therefore, appropriate intakes for children of the same age, sex, and size vary. The RDA's recommend a range of energy allowances that average 105 kcal/kg/day for children 1 year of age to 80 kcal/kg/day for children 2 to 10 years of age (NRC 1980). Higher levels of energy intake are required to compensate for inadequate body weight due to low birth weight, growth retardation, or other factors (Ashworth and Millward 1986).

Nutrients

Children need protein for the maintenance of body tissues, changes in body composition, and synthesis of new muscles. During growth, the protein content of the body increases from about 15 percent at 1 year to 18 to 19 percent by 4 years, which is also the value for adults (Fomon et al. 1982). Estimates of protein needs for growth range from 1 to 4 g/kg/day (Ashworth and Millward 1986). The RDA decreases from 1.8 g/kg/day at 1 year to 0.8 g/kg/day at 18 years (NRC 1980).

Inadequate intakes of vitamins and minerals will be reflected in slow growth rates, inadequate mineralization of bones, and very low body reserves of the micronutrients. Clinical signs of vitamin deficiency in children are reported infrequently. The most common mineral deficiency, iron, appears to be declining, although children from low-income families are at greater risk (see anemia chapter). With the relatively low prevalence of clinical signs of vitamin and mineral deficiency in the general population of children, there is no evidence that supplementation is necessary for this group (AAP Committee on Nutrition 1980b). Although vitamin and mineral supplements increase the quantity of these nutrients in the diet (Cook and Payne 1979), they have not been shown to improve biochemical indices of nutrient status in children who are already well nourished (Breskin et al. 1985). For this reason, recommendations on vitamin and mineral supple-

mentation for children target those at high risk, those from socioeconomically deprived families, and those who have poor appetites or eating habits (AAP Committee on Nutrition 1980b).

Eating Patterns

Preschool children are a nutritionally vulnerable group. Their growth rate is slower than it was in infancy and their nutritional needs in relation to body size proportionately reduced. Thus, they often want and eat relatively little food. Food intake can be reduced even further by the increasing independence (expressed as refusals to eat) and immature feeding skills that are characteristic of very young children. Despite these problems, surveys have indicated that, with the exception of a small subgroup, American preschool children are in relatively good nutritional health. Children of lower socioeconomic status are at higher risk of inadequate nutrient intakes (especially iron deficiency) and poorer growth. Although parents have the main responsibility for providing adequate and appropriate food for preschool children, day care providers supply an increasing proportion of the food that children consume.

Parents continue to be the main influence on the food intake of school-aged children, although an increasing proportion of the diet is consumed in schools, day care centers, and fast food restaurants (Select Panel 1981). Between the ages of 4 and 6, children increase the varieties of foods they are willing to eat (see behavior chapter). Snacks become an important source of calories and nutrients (Crawford, Hankin, and Huenemann 1978) and may contribute as much as one-third of calories and fat, one-fifth of the protein, and nearly one-half of the carbohydrate 10-year-old children consume (Farris et al. 1986). These patterns emphasize the need for parents and schools to provide appropriate meals and snacks and guidance in food choices. Of special concern is the need to encourage appropriate levels of daily physical activity (Select Panel 1981) and choice of nutritious snacks that do not promote tooth decay.

Role of Dietary Factors in Adolescent Health

Energy and Nutrient Requirements

Energy and nutrient requirements are directly related to the stage and rate of growth, and demands are greatest during the peak velocity of growth. Because individuals enter adolescence at different ages and have different rates of growth for different time periods, it is difficult to establish specific nutrient requirements for individuals. Few studies have been conducted on the nutritional needs of adolescents, and the RDA's are established mainly

by extrapolation from adult age groups (NRC 1980). For most nutrients, RDA's are similar to those for adults. The RDA's provide a wide range of energy intakes for two age groups, 11 to 14 years of age and 15 to 18 years of age (NRC 1980). However, many girls experience their peak growth velocity before the age of 11.

The RDA for calcium, 1,200 mg/day, is higher for adolescents than for adults and is designed to meet the needs of the adolescent who is growing at the fastest rate. Achieving maximum bone mass during the teens and twenties can reduce the risk of developing osteoporosis later in life (Lucas, Rees, and Mahan 1985; see chapter on skeletal diseases). The higher RDA for iron for adolescent males is also related to rapid growth, which is accompanied by increases in blood volume, muscle mass, and iron-containing enzymes. Although there are few data on the zinc requirements of adolescents, this population may be at risk for marginal intakes (Mahan and Rosebrough 1984). Vitamin requirements are correlated with growth demands rather than age. For example, males have higher needs for folate than females, and the values for both sexes increase with physical maturity (Daniel, Gaines, and Bennett 1975).

Eating Patterns

The growth spurt of adolescence demands significant increases in calories and nutrient intake to support the rapid growth rate and increased body size. In early adolescence, children still depend on their parents for food, but by the end of adolescence they are largely independent (Select Panel 1981). Irregular eating patterns are common in adolescence, reflecting this growing independence from the family and the teenager's increasingly busy social life and athletic, academic, and vocational activities. Breakfast and lunch are often skipped or eaten on the run. Snacking is characteristic of this age group and contributes significantly to nutrient intake; these snack foods are often higher in calories, fat, and sugar—and lower in vitamins, minerals, and fiber—than foods consumed at family meals (Hampton et al. 1967; Pao 1980). Because lifetime dietary patterns are established during these years, adolescents should be encouraged to choose nutritious foods, to develop good eating habits, and to maintain appropriate levels of physical activity (Select Panel 1981).

Role of Dietary Factors in Chronic Disease in Childhood

Several chronic diseases have special implications in the nutrition of infants, children, and adolescents. Some of these are discussed elsewhere in this Report. Childhood hyperactivity (or attention deficit disorders) and eating disorders such as anorexia nervosa and bulimia, for example, are

reviewed in the chapter on behavior, and juvenile diabetes is reviewed in the diabetes chapter. Children with chronic disease and other handicapping conditions frequently require therapeutic diets accompanied by intensive nutrition counseling and support (see, for example, Baer 1982). Discussion of issues related to most of these conditions is beyond the scope of this Report, but a few examples follow.

Coronary Heart Disease

The relationship between diet in infancy, childhood, and adolescence and the development of adult atherosclerosis and coronary heart disease (see chapter on coronary heart disease) is of great current interest. Within the past 5 years, cholesterol-lowering diets for children with elevated blood cholesterol levels (Consensus Development Panel 1985), as well as for those with normal levels (Weidman et al. 1983), have been recommended to prevent onset of the adult disease. These recommendations are that all children older than 2 years adopt a diet that reduces dietary fat intake to 30 percent or less of calories, saturated fat to less than 10 percent of calories, and daily cholesterol intake to 250 to 300 mg or less. However, other groups have advised against specific recommendations because they find insufficient evidence for the safety and efficacy of such diets in children (AAP Committee on Nutrition 1983c, 1986).

Increasing evidence suggests that atherosclerosis begins in childhood. Cholesterol concentrations, which are lower in cord blood than in maternal blood, rise after infants begin to be fed. Infants fed human milk or cow milk have higher blood cholesterol levels at age 6 months than do those fed formulas containing vegetable oils, but these differences are reduced once cholesterol-containing foods are added to the diet (Farris et al. 1982). By the age of 1 year, blood cholesterol levels correlate with dietary intake of saturated fat and cholesterol; they rise rapidly during the first 2 years of life (Berenson et al. 1979). Blood cholesterol levels at age 6 months are correlated with levels at age 7 years (Freedman et al. 1987). Childhood blood cholesterol levels have a strong genetic component, and children whose parents have high levels are two to three times more likely to be in the 95th percentile for blood cholesterol than children of parents with low to normal levels (Berenson et al. 1979).

Atherosclerotic plaques have been identified in the coronary arteries of young soldiers killed in the Korean and Vietnam wars (Strong 1986) and in adolescents and even younger children who suffered accidental death (Velican and Velican 1980). Children in the United States exhibit higher blood cholesterol levels and have a higher dietary intake of fat and cholesterol than children in populations with lower heart disease rates (Weidman

et al. 1983). Measurements of other heart disease risk factors such as high blood pressure (Burke et al. 1987) or obesity (Harsha et al. 1987) made in early childhood are highly correlated with those made in the same children at age 7. Thus, pediatricians have been urged to identify and to treat children with elevated blood cholesterol levels (Wynder et al. 1983; Consensus Development Panel 1985).

On the other hand, no data are available from prospective studies to demonstrate that feeding cholesterol-lowering diets to children can either support normal growth and development or reduce later heart disease rates. Thus, the American Academy of Pediatrics (AAP) recommends that children's diets follow current dietary trends with moderation, including decreased consumption of saturated fats, cholesterol, and salt and an increased intake of polyunsaturated fats (AAP Committee on Nutrition 1986). It also stresses that current data indicate that no changes in the current diet for infants and children under the age of 2 are necessary. Whether a diet consisting of 30 percent of calories from fat should be advised for the general population of children over age 2 remains controversial, but a fat intake over 40 percent of calories is excessive (La Rosa and Finberg 1988). Until better data are available, the AAP recommends screening high-risk children for early detection and treatment for heart disease risk factors such as high blood cholesterol, high blood pressure, and obesity (AAP Committee on Nutrition 1986).

Obesity

The increased rate of pediatric obesity in the United States is an important public health issue (Gortmaker et al. 1987). Childhood obesity can lead to adult obesity and all its complications; currently, insufficient information is available about the causes and prevention of early onset of obesity (see chapter on obesity).

The cause of obesity in infants is poorly understood. Speculations that formula feeding and early introduction of solid foods might be responsible are not supported by research studies (Ferris et al. 1980; Dubois et al. 1979), and no differences in subsequent fatness have been observed among children who were breast or bottle fed (Fomon et al. 1984). Both genetic and environmental factors are involved (see obesity chapter). A recent study of adopted children, for example, found a strong correlation of body weights to the weight of the biologic rather than the adoptive parents (Stunkard et al. 1986). Lower-than-normal activity levels are also related to childhood obesity (Berkowitz et al. 1985). One study observed a direct relationship between body weight and number of hours spent watching television (Dietz and Gortmaker 1985).

Studies examining the role of early infant obesity as a risk factor for later obesity are also inconsistent. Some studies found that most obese infants become thinner as they grow older and concluded that early weight gain does not predict later obesity (Shapiro et al. 1984). Others have found that body weights at the age of 1 year strongly predict body weights at age 7 (Harsha et al. 1987). Nearly all studies agree, however, that the correlation between childhood obesity and later obesity increases as children grow older (AAP Committee on Nutrition 1981). These and other issues related to obesity are reviewed in the chapter devoted to that topic.

Cognitive Performance

LBW infants are at greater risk for developmental disorders than are normal-weight infants (Parkinson, Wallis, and Harvey 1981). Infants with early intrauterine growth retardation (prior to 26 weeks' gestation) are at greater risk than infants whose growth retardation begins after 26 weeks' gestation (Fancourt et al. 1976). Early intrauterine growth retardation is associated with less successful school performance, lower intelligence, and more behavioral and other handicaps (Harvey et al. 1982).

The role of maternal and early infant malnutrition, as distinguished from other causes of fetal and infant growth retardation, is uncertain. Animal and human studies have shown that severe malnutrition during fetal growth and early infancy retards brain cell division and alters nerve myelination, but cognitive and behavioral effects of less severe nutritional deprivation cannot easily be distinguished from other environmental effects.

Data from populations in which malnutrition is endemic indicate a relationship between growth retardation of infants and young children and low performance in mental development tests (Lasky et al. 1981). Children with protein-energy malnutrition in infancy who were tested at ages 5 to 11 years had poorer academic performance than children who were well nourished in infancy, which is reflected in classroom behavior problems such as lack of attention, poor memory, poor motivation, and easy distractibility (Galler, Ramsey, and Solimano 1984). However, socioeconomic disadvantages as well as poor nutritional status could cause these problems. Children subjected to prenatal malnutrition because of war-induced famine or to acute periods of malnutrition during infancy can overcome nutritionally induced growth deficits and can exhibit cognitive function levels that correlate most strongly with the parents' educational status (Stein et al. 1972; Beardslee et al. 1982). Malnourished children adopted by socioeconomically advantaged families have been able to catch up in mental development (Graham and Adrianzen 1972).