Dietary Supplement Use in Women: Current Status and Future Directions

Pregnancy and Lactation: Physiological Adjustments, Nutritional Requirements and the Role of Dietary Supplements¹

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ABSTRACT Nutritional needs are increased during pregnancy and lactation for support of fetal and infant growth and development along with alterations in maternal tissues and metabolism. Total nutrient needs are not necessarily the sum of those accumulated in maternal tissues, products of pregnancy and lactation and those attributable to the maintenance of nonreproducing women. Maternal metabolism is adjusted through the elaboration of hormones that serve as mediators, redirecting nutrients to highly specialized maternal tissues specific to reproduction (i.e., placenta and mammary gland). It is most unlikely that the heightened nutrient needs for successful reproduction can always be met from the maternal diet. Requirements for energy-yielding macronutrients increase modestly compared with several micronutrients that are unevenly distributed among foods. Altered nutrient utilization and mobilization of reserves often offset enhanced needs but sometimes nutrient deficiencies are precipitated by reproduction. There are only limited data from well-controlled intervention studies with dietary supplements and with few exceptions (iron during pregnancy and folate during the periconceptional period), the evidence is not strong that nutrient supplements confer measurable benefit. More research is needed and in future studies attention must be given to subject characteristics that may influence ability to meet maternal and infant demands (genetic and environmental), nutrient-nutrient interactions, sensitivity and selectivity of measured outcomes and proper use of proxy measures. Consideration of these factors in future studies of pregnancy and lactation are necessary to provide an understanding of the links among maternal diet; nutritional supplementation; and fetal, infant and maternal health. J. Nutr. 133: 1997S-2002S, 2003.

KEY WORDS: • pregnancy • lactation • nutritional requirements • dietary supplements

The health of mothers and their infants is a priority in the United States, and *Healthy People 2010*, our nationwide health promotion and disease prevention agenda, identifies measurable objectives for improvement (1). Many of these objectives are based on nutrition research that offers promise for enhancing reproductive outcomes. Accumulating evidence from evaluation of public health nutrition programs and nutrient-specific intervention trials indicates that maternal nutritional

modifications can and do produce desirable health advantages (2-4).

During pregnancy and lactation, nutritional requirements increase to support fetal and infant growth and development as 112 well as maternal metabolism and tissue development specific to reproduction. Total nutrient requirements are not necessarily the simple sum of those accumulated in maternal tissues, g products of pregnancy and lactation and those attributable to maintenance of nonreproducing women even though this prot on 0 cess of summation is sometimes used to derive estimates of recommended nutrient intakes. Pregnancy and lactation are anabolic states that are orchestrated via hormones to produce a redirection of nutrients to highly specialized maternal tissues $\stackrel{\circ}{\overset{\circ}{\underset{N}{\sim}}}$ characteristic of reproduction (i.e., placenta and mammary N gland) and their transfer to the developing fetus or infant. In this article the physiological adjustments and nutritional requirements of pregnant and lactating women and the possible role of dietary supplementation in meeting requirements for nutrients likely to be limiting in the diet are discussed.

Physiological adjustments of pregnancy

Hormonal changes during pregnancy. Plasma levels of human chorionic gonadotropin increase immediately upon implantation of the ovum; the hormone is detectable in urine

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within 2 wk of implantation. It reaches a peak at ≈ 8 wk of gestation and then declines to a stable plateau until birth. Human chorionic gonadotropin maintains corpus luteum function for 8–10 wk. Human placental lactogen (also called human chorionic somatomammotropin) has a structure that closely resembles growth hormone, and its rate of secretion appears to parallel placental growth and may be used as a measure of placental function. At its peak, the rate of secretion of placental lactogen is 1–2 g/d, far in excess of the production of any other hormones. Placental lactogen stimulates lipolysis, antagonizes insulin actions and may be important in maintaining a flow of energy-yielding substrates to the fetus. Placental lactogen along with prolactin from the pituitary may promote mammary gland growth. After delivery, placental lactogen rapidly disappears from the circulation.

The placenta becomes the main source of steroid hormones at weeks 8–10 of gestation. Before then, progesterone and estrogens are synthesized in the maternal corpus luteum. These hormones play essential roles in maintaining the early uterine environment and development of the placenta. The placenta takes over progesterone production, which increases throughout pregnancy. Progesterone, known as the hormone of pregnancy, stimulates maternal respiration; relaxes smooth muscle, notably in the uterus and gastrointestinal tract; and may act as an immunosuppressant in the placenta, where its concentration can be 50 times greater than in plasma. Progesterone may promote lobular development in the breast and is responsible for the inhibition of milk secretion during pregnancy.

The secretion of estrogens from the placenta is complex (5). Estradiol and estrone are synthesized from the precursor dehydroepiandrosterone sulfate (DHEA-S), which is derived from both maternal and fetal blood. The synthesis of estriol is from fetal 16- α -hydroxy-dehydroepiandrosterone sulfate (16-OH-DHEA-S). The fetus is unable to synthesize pregnenolone, the precursor of DHEA-S and 16-OH-DHEA-S, and must get the precursor from the placenta. The placental secretion of estrogens also increases manyfold with the progression of pregnancy. The functions of high estrogen levels in pregnancy include stimulation of uterine growth, enhancement of uterine blood flow and possibly promotion of breast development. Because estrogen precursors originate in the fetus, maternal estrogen levels can be used as a measure of fetal viability.

The increased amount of estrogens during pregnancy also stimulates a population of cells (somatotrophs) in the maternal pituitary to become mammotrophs, or prolactin-secreting cells. The increased prolactin secretion probably helps promote mammary development. In addition, the increased number of pituitary mammotrophs at the end of pregnancy provides the large amounts of prolactin necessary to initiate and maintain lactation.

Blood volume and composition. During pregnancy there is an increase in blood volume of \approx 35-40%, expressed as a percentage of the nonpregnant value, that results principally from the expansion of plasma volume by \approx 45–50% and of red cell mass by $\approx 15-20\%$ as measured in the third trimester. Because the expansion of red cell mass is proportionally less than the expansion of plasma, hemoglobin concentration and hematocrit values fall in parallel with red cell volume. Hemoglobin and hematocrit values are at their lowest in the second trimester of pregnancy and rise again in the third trimester. For these reasons, trimester-specific values for hemoglobin and hematocrit are proposed for screening for anemia in pregnant women (6).

Total plasma protein concentration falls from \approx 70 to 60 g/L largely because of a fall in albumin concentration from ≈ 4 to 2.5 g/l00 mL near term. Plasma concentrations of $\alpha 1$ -, $\alpha 2$ - and β -globulins increase by $\approx 60\%$, 50% and 35%, respectively, whereas the γ -globulin fraction decreases by 13% (7). Estrogens are responsible for these changes in plasma proteins, which can be reproduced by administration of estradiol to nonpregnant women. Plasma levels of most lipid fractions, including triacylglycerol, VLDL, LDL and HDL, increase during pregnancy.

Recommended weight gain. The average weight gained by healthy primigravidae eating without restriction is 12.5 kg (27.5 lb) (5). This weight gain represents two major components: 1) the products of conception: fetus, amniotic fluid and the placenta and 2) maternal accretion of tissues: expansion of blood and extracellular fluid, enlargement of uterus and mammary glands and maternal stores (adipose tissue).

Low weight gain is associated with increased risk of intrauterine growth retardation and perinatal mortality. High weight gain is associated with high birth weight and secondarily with increased risk of complications related to fetopelvic disproportion. A large body of epidemiologic evidence now shows convincingly that maternal prepregnancy weight-forheight is a determinant of fetal growth above and beyond gestational weight gain. At the same gestational weight gain, it women give birth to infants smaller than those born to heavier women. Because higher birth weights present lower risk for infants, current recommendations for weight gain during pregnancy are higher for thin women than for women of normal weight and lower for short overweight and obese women (7). These recommendations are summarized below <u>2</u> (Table 1).

Recommendations for weight gain during pregnancy were formulated in recognition of the need to balance the benefits and of increased fetal growth against the risks of labor and delivery are complications and of postpartum maternal weight retention. The target range for desirable weight gain in each prepregnancy weight-for-height category is that associated with delivery of a full-term infant weighing between 3 and 4 kg. Recent evidence indicates that <50% of 622 women sampled in upstate New York gained weight within the ranges recom-mended and that weight gain greater than these recommended amounts placed them at risk for major weight gain 1 y post-delivery (8). **Nutritional needs during pregnancy** Determination of nutrient needs during pregnancy is comlivery of a full-term infant weighing between 3 and 4 kg.

Determination of nutrient needs during pregnancy is complicated because nutrient levels in tissues and fluids available for evaluation and interpretation are normally altered by hormone-induced changes in metabolism, shifts in plasma volume and changes in renal function and patterns of urinary excretion. Nutrient concentrations in blood and plasma are often \aleph decreased because of expanding plasma volume, although total circulating quantities can be greatly increased. Individual profiles vary widely, but in general, water-soluble nutrients and metabolites are present in lower concentrations in pregnant

TABLE 1

Current recommendations for weight gain during pregnancy*

Weight-for-height category	Recommended total gain, kg (lb)
Low (BMI < 19.8)	12.5–18 (28–40)
Normal (BMI 19.8–26.0)	11.5–16 (25–35)
High (BMI > 26–29)	7–11.5 (15–25)

* Data from Institute of Medicine (7). BMI, body mass index.

than in nonpregnant women whereas fat-soluble nutrients and metabolites are present in similar or higher concentrations. Homeostatic control mechanisms are not well understood and abnormal alterations are ill-defined.

Dietary Reference Intakes for pregnant and lactating women in comparison with those of adult, nonreproducing women are presented in **Table 2**. Also presented in Table 2 are comparative cumulative energy and nutrient expenditures of adult, pregnant and lactating women. The recommended intakes for pregnant adolescents generally would be increased by an amount proportional to the incomplete maternal growth at conception. The percentage increase in estimated energy requirement is small relative to the estimated increased need for most other nutrients. Accordingly, pregnant women must select foods with enhanced nutrient density or risk nutritional inadequacy.

Energy. Energy needs during pregnancy are currently estimated to be the sum of total energy expenditure of a nonpregnant woman plus the median change in total energy expenditure of 8 kcal/gestational week plus the energy deposition during pregnancy of 180 kcal/d (13). Because total energy expenditure does not change greatly and weight gain is minimal in the first trimester, additional energy intake is recommended only in the second and third trimesters. Approxi-

mately an additional 340 and 450 kcal are recommended during the second and third trimesters, respectively.

Protein. Additional protein is needed during pregnancy to cover the estimated 21 g/d deposited in fetal, placental and maternal tissues during the second and third trimesters (13). Women of reproductive age select diets containing average protein intakes of \approx 70 g/d (14), a value very close to the theoretical need of 71 g during pregnancy.

Vitamins and minerals. The assessment of vitamin and mineral status during pregnancy is difficult because there is a general lack of pregnancy-specific laboratory indexes for nutritional evaluation. Plasma concentrations of many vitamins and minerals show a slow, steady decrease with the advance of gestation, which may be due to hemodilution; however, other vitamins and minerals can be unaffected or increased because of pregnancy-induced changes in levels of carrier molecules (15). When these patterns are unaltered by elevated maternal intakes, it is easy to conclude that they represent a normal physiological adjustment to pregnancy rather than increased needs or deficient intakes. Even when enhanced maternal intake does induce a change in an observed pattern, interpretation of such a change is difficult unless it can be related to some functional consequence (15). For these reasons, much of our knowledge is based on observational studies and interven-

TABLE 2

Comparison of recommended	daily energy	and nutrient	intakes and	l cumulative	expenditures	of adult,
	pregnai	nt and lactatii	ng women			

Nutrient	Dietary Reference Intakes (DRI) ¹			Calculated cumulative expenditure (9 mo)			Percentage increase over nonreproducing adult women	
	Adult women	Pregnancy	Lactation	Adult women	Pregnancy	Lactation	Pregnancy %	Lactation %
Energy, ² kcal	19–50 y	↑ 340 kcal/d 2nd trimester ↑ 452 kcal/d 3rd trimester	↑ 500 kcal/d 0–6 mo ↑ 400 kcal/d 7–9 mo	variable	75,000– 80,000	126,000	Ŷ	ſ
Protein, ³ g	46	71	71	12,420	19,170	19,170	54.35	54.35
Vitamin C,3 mg	75	85	120	20,250	22,950	32,400	13.33	60.00
Thiamin, ³ mg	1.1	1.4	1.4	297	378	378	27.27	27.27
Riboflavin, ³ mg	1.1	1.4	1.6	297	378	432	27.27	45.45
Niacin, ³ ng NĚ	14	18	17	3,780	4,860	4,590	28.57	21.43
Vitamin B-6,3 mg	1.3	1.9	2	351	513	540	46.15	53.85
Folate, 3 µg DFE	400	600	500	108,000	162,000	135,000	50.00	25.00
Vitamin B-12,3 µg	2.4	2.6	2.8	648	702	756	8.33	16.67
Pantothenic acid, ⁴ mg	5	6	7	1,350	1,620	1,890	20.00	40.00
Biotin, ⁴ μg	30	30	35	8,100	8,100	9,450	0.00	16.67
Choline, ⁴ mg	425	450	550	114,750	121,500	148,500	5.88	29.41
Vitamin A, ³ $\mu g RE$	700	770	1300	189,000	207,900	351,000	10.00	85.71
Vitamin D, ⁴ μq	5	5	5	1,350	1,350	1,350	0.00	0.00
Vitamin E, ³ mg α -TE	15	15	19	4,050	4,050	5,130	0.00	26.67
Vitamin K, ⁴ μg	90	90	90	24,300	24,300	24,300	0.00	0.00
Calcium, ⁴ mg	1000	1000	1000	270,000	270,000	270,000	0.00	0.00
Phosphorus, 4 mg	700	700	700	189,000	189,000	189,000	0.00	0.00
Magnesium, ³ mg	310	350	310	83,700	94,500	83,700	12.90	0.00
Iron, ³ mg	18	27	9	4,860	7,290	2,430	50.00	-50.00
Zinc, ³ mg	8	11	12	2,160	2,970	3,240	37.50	50.00
lodine, ³ µg	150	220	290	40,500	59,400	78,300	46.67	93.33
Selenium, ³ μg	55	60	70	14,850	16,200	18,900	9.09	27.27
Fluoride, ⁴ mg	3	3	3	810	810	810	0.00	0.00

¹ Values are from the Institute of Medicine (9–13).

² Calculations are based on recommended intakes per day, assuming 9 months is equivalent to 270. Abbreviations NE, niacin equivalents; DFE, dietary folate equivalents; RE, retinol equivalents; TE, tocopherol equivalents.

³ and ⁴ are, respectively, Recommended Dietary Allowance (RDA), the average daily dietary intake level that is sufficient to meet the nutrient requirements of nearly all (97 to 98 percent) individuals in a life stage and gender group and based on the Estimated Average Requirement (EAR); and Adequate Intake (AI), the value used instead of an RDA if sufficient scientific evidence is not available to calculate an EAR.

tion trials in which low or high maternal intakes are associated with adverse or favorable pregnancy outcomes. Available data on vitamin and mineral metabolism and requirements during pregnancy are fragmentary at best, and it is exceedingly difficult to determine consequences of seemingly deficient or excessive intakes in human populations. However, animal data show convincingly that maternal vitamin and mineral deficiencies can cause fetal growth retardation and congenital anomalies. Similar associations in humans are rare. Selected vitamins and minerals that are likely to be limiting or excessive in the diets of pregnant women and their association with pregnancy outcome are briefly discussed.

Placental transport of vitamin A between mother and fetus is substantial, and recommended intakes are increased by \approx 10% (12). Low maternal vitamin A status is inconsistently associated with intrauterine growth retardation in communities at risk for vitamin A deficiency. Dietary supplementation with vitamin A or β -carotene is reported to reduce maternal mortality by 40% but to not affect fetal loss or infant mortality rates (16,17). Overt vitamin A deficiency is not apparent in the United States; instead, the concern during pregnancy is about excess (18).

The main circulating form of vitamin D in plasma, 25hydroxycholecalciferol, is responsive to increased maternal intake and falls with maternal deficiency. The biologically active form of the vitamin, 1,25-dihydroxycholecalciferol, circulates in bound and free forms and both are elevated in pregnancy (19). All forms of vitamin D are transported across the placenta to the fetus. Vitamin D deficiency during pregnancy is associated with several disorders of calcium metabolism in both the mother and her infant, including neonatal hypocalcemia and tetany, infant hypoplasia of tooth enamel and maternal osteomalacia (20). Supplementation of 10 μ g (400 IU)/d in affected women lowered the incidence of neonatal hypocalcemia and tetany and maternal osteomalacia whereas higher amounts (25 μ g/d) increased weight and length gains in infants postnatally (21). The prevalence of vitamin D deficiency is high in pregnant Asian women in England and in pregnant women in other European countries at northern latitudes, where the amount of ultraviolet light reaching the earth's surface is not sufficient for synthesis of vitamin D in the skin during winter months. Food sources of vitamin D are few and no increase in vitamin D intake during pregnancy is recommended (9). However, recent data from the third National Health and Nutrition Examination Survey indicate that ≈42% of African American women and 4% of white women show biochemical evidence of vitamin D insufficiency (22). Research is needed to assess vitamin D requirements of women of reproductive age, the extent to which the diet or light exposure can furnish needed amounts and the possible benefit of supplemental quantities before and during pregnancy.

Compromised maternal folate intake or status is associated with several negative pregnancy outcomes including low birth weight, abruptio placentae, risk for spontaneous abortions and neural tube defects (23). Folic acid supplementation prevents both the occurrence and recurrence of neural tube defects (24) and significantly reduces the incidence of low birth weight (25). Previously, folic acid supplementation was started relatively late in pregnancy but now in the United States, the Food and Drug Administration requires folic acid fortification of most grain products, and intakes have dramatically increased. The recommended intake for folate during pregnancy is 600 μ g/d (10). It will be important to evaluate the extent to which folic acid fortification increases intake of reproducing women, decreases neural tube defects and affects growth and development of the fetus.

The total iron cost of pregnancy is estimated at 1040 mg, of which 200 mg are retained by the woman when blood volume decreases after delivery and 840 mg are permanently lost. Iron is transferred to the fetus (\approx 300 mg) and used for the formation of the placenta (50-75 mg), expansion of red cell mass (\approx 450 mg) and blood loss during delivery (\approx 200 mg). Hemoglobin concentration declines during pregnancy along with serum iron, percentage saturation of transferrin and serum ferritin. Although these decreases reflect hemodilution to a large extent, transferrin levels actually increase from mean nonpregnant values of 3 mg/L to 5 mg/L in the last trimester of pregnancy, perhaps to facilitate iron transfer to the fetus. Enhanced intestinal iron absorption (two- to threefold) is an important physiological adjustment that assists pregnant women in meeting the requirement for absorbed iron, which is estimated to be ≈ 3 mg/d. Maternal anemia is associated with perinatal maternal and infant mortality and premature delivery. To preserve maternal stores and to prevent the development of iron deficiency, the recommended iron intake during pregnancy is increased by 9 mg to a total of 27 mg/d (12). This level cannot normally be obtained from foods, and supplementation is required to achieve recommended intakes. The routine use of iron supplements during pregnancy, however, is not universally endorsed. Another paper in this publication pro-vides recent evidence supporting iron supplementation during pregnancy (26).

Maternal iodine deficiency leading to fetal hypothyroidism results in cretinism, characterized by severe mental retardation (3). Thyroid hormones are critical for normal brain develop-ment and maturation. Manifestation of other features of cre-tinism (deafmutism, short stature and spasticity) depends on the stage of pregnancy when hypothyroidism develops. When the stage of pregnancy when hypothyroidism develops. When $\frac{1}{3}$ it develops late in pregnancy, the neurological damage is not as severe as when it exists early in pregnancy. Cretinism is as severe as when it exists early in pregnancy. Cretinism is prevented by correcting maternal iodine deficiency before or during the first 3 mo of pregnancy. The World Health Organization estimates that 20 million people worldwide have brain damage resulting from maternal iodine deficiency that brain damage resulting from maternal iodine deficiency that could be prevented by iodine supplementation (27). The rec- \vec{N} ommended iodine intake is 220 μ g/d during pregnancy (12). The mean intake of U.S. women of childbearing age is \approx 170 μ g/d, excluding iodine from iodized salt (5).

Endocrine regulation of lactation

The establishment and maintenance of human lactation tin and withdrawal of estrogens and progesterone results in the onset of milk secretion (lactogenesis). The breasts must have undergone appropriate growth and development beginning in puberty and completed during pregnancy for milk secretion to occur. The initiation of lactogenesis does not require infant sucking but lactation cannot be maintained unless the infant is put to the breast by 3 or 4 d postpartum. For the first 3–5 d postpartum the mammary secretion is termed "colostrum." This early milk is thick and straw-colored, rich in minerals and immune factors (i.e., lactoferrin and secretory immunoglobulin A) and low in lactose and total protein. The concentration of lactose increases and that of sodium and chloride decrease as milk secretion is enhanced. The characteristics of mature milk are evident by day 10 of lactation.

With established lactation, prolactin is required for maintenance of milk production. Prolactin release into the circu-

lation from mammotrophs in the anterior pituitary is in response to sucking. Prolactin secretion is mediated by a transient decline in the secretion of dopamine from the hypothalamus, which normally inhibits its secretion. Milk secretion continues as long as the infant continues to nurse more than once a day. The daily milk volume transferred to the infant increases from \approx 50 mL on day 1 to 500 mL by day 5, \approx 650 mL by 1 mo and 750 mL at 3 mo of lactation. Most women can secrete considerably more milk than needed by a single infant. Milk secretion is continuous and the quantity produced is principally regulated by infant demand. Oxytocin release from the posterior pituitary results from neural impulses reaching the hypothalamus caused by sucking of the nursing infant. Circulating oxytocin causes contraction of myoepithelial cells that surround mammary alveoli and ducts, forcing milk into ducts of the nipple so that it can be removed by the infant. This response is termed "milk ejection" or "let-down" and can be initiated by the mere sight of the infant or by hearing the infant cry. Continuation of lactation and associated hyperprolactinemia inhibit ovarian activity by suppressing the pulsatile release of luteinizing hormone and by interfering with the secretion of gonadotropin-releasing hormone. This provides 98% protection from pregnancy during the first 6 mo of lactation if the nursing mother continues to be amenorrheic (29). Milk secretion ceases in 1 or 2 d when infant sucking or milk removal is terminated.

Nutritional needs during lactation

The nutritive demands of lactation are considerably greater than those of pregnancy. In the first 4-6 mo of the postpartum period, infants double their birth weight accumulated during the 9 mo of pregnancy. The milk secreted in 4 mo represents an amount of energy roughly equivalent to the total energy cost of pregnancy. However, some of the energy and many of the nutrients stored during pregnancy are available to support milk production. The recommended intakes for energy and specific nutrients during lactation are summarized in Table 2. Most of these recommended intakes are based on our knowledge of the amount of milk produced during lactation, its energy and nutrient contents and the amounts of maternal energy and nutrient reserves. Recommended intakes during lactation are based on even less quantitative data than recommendations during pregnancy. Lactation is viewed as successful when the fully breast-fed infant is growing well and maintaining appropriate biochemical indexes of nutritional status. The quantity of milk consumed by the infant and the nutrient content of human milk under these circumstances are often used as proxies to assess maternal nutritional adequacy during lactation. In very few studies have specific measures of nutritional status been applied to the lactating mother.

Human milk feeding is adequate as the sole source of nutrition for up to age 6 mo providing that the maternal diet and reserves are adequate and a sufficient quantity is transferred to the infant. The composition of human milk is exceedingly variable; nonetheless, such variance is compatible with successful lactation. The *Handbook of Milk Composition* provides comprehensive data on human milk composition and factors capable of altering it (30). During lactation the mammary gland exhibits metabolic priority for nutrients, often at the expense of maternal reserves (31). Measurable differences in milk nutrient content due to dietary intake can and do occur, most notably in the vitamin constituents (32).

The recommended energy intake during the first 6 mo of lactation is an additional 500 kcal under the assumption that 170 kcal/d will be mobilized from energy stores accumulated in

pregnancy. The energy demands of comparable periods of full lactation (780 mL/d) greatly exceed those of pregnancy. The recommended energy intake after 6 mo is reduced to an additional 400 kcal/d because milk production rates decrease to 600 mL/d. Few studies have evaluated maternal nutrient adequacy, milk content and infant nutrient indicators in the second half of the first year of infancy and lactation.

As with energy, recommended intakes for several vitamins and minerals are similarly higher in lactation than in pregnancy (Table 2) with the notable exception of iron (12). The recommended iron intake for women of reproductive age is 18 mg/d. Recommended iron intakes for nonreproducing women were estimated based on basal losses and menstrual losses. For lactating women, estimations were based on basal losses, with the assumption that menstruation resumes at 6 mo, plus the quantity secreted in milk. It is difficult to reconcile that iron § needs during lactation would be less than those of the nonreproducing women considering that 16% of women of childbearing age enter pregnancy with biochemical evidence of iron deficiency (ferritin concentration $<15 \ \mu g/L$) (12) and that in 1996, 29% of low income women were anemic (hemoglobin concentration < 110 g/L), a prevalence rate that has not $\frac{10}{10}$ changed since 1979 (32). Moreover, national data indicate that one-fourth of all females of childbearing age failed to meet the previous recommended intake of 15 mg/d, 25% less than the current recommended amount (33). It may be prudent to factor in recovery of iron stores and mitigation of iron deficiency after pregnancy in formulating recommended iron intakes during lactation.

Available information on nutrients in milk that can be influenced by maternal nutrition as well as nutrients associated with recognizable deficiencies in breast-fed infants are summarized elsewhere (30,34). At present our information about the role of dietary supplementation in lactation is limited. The available information, in large part, is from studies conducted in early lactation. The nutritional demands of lactation are directly proportional to intensity and duration, and evaluation in early lactation may not bear on circumstances in late lactation (>6 mo). The need for continued study is paramount now that evidence exists that the initiation of breast-feeding and breast-feeding to 6 mo in the United States have reached their highest levels to date, 69.5% and 32.5%, respectively (35).

As is the case during pregnancy, nutrient density of the 2 maternal diet assumes great importance during lactation because the estimated increase in energy needs is less than $\overline{\circ}$ estimated increases in needs for other nutrients. At energy intakes less than recommended, maternal intakes of calcium, magnesium, zinc, vitamin B-6 and folate may be correspondingly low (30). The extent to which low intakes of these and other nutrients affect the success of lactation and long-term maternal and infant health has not been examined except when a distinct nutritional deficiency is evident in the nursing infant, for example, in vitamins D and B-12. A supplement of vitamin D (10 μ g/d) is recommended for women who avoid milk and other foods fortified with vitamin D. Similarly, a supplement of vitamin B-12 (2.6 μ g/d) is recommended for lactating women who are complete vegetarians (30). In addition, most studies have considered a single nutrient in isolation. It is possible that limitations in one nutrient may be a marker for other nutrient inadequacies (e.g., iron and folate deficiencies often coexist) and focusing on one nutrient may limit our understanding of nutrient-nutrient interaction.

Summary

Our knowledge of the effect of maternal dietary adequacy on the success of reproduction is far from complete as is the role that dietary supplement may play. Although few scientific studies furnish clear links between maternal nutrient intakes from foods and supplements and reproductive outcomes, there are indications that maternal nutritional adequacy does influence performance indexes both in pregnancy and lactation. Birth weight and infant growth measures are the principal indicators of reproductive success used in scientific studies and these markers may not provide the needed sensitivity to assess the influence of maternal nutrition. Moreover, the health and nutritional status of the mother should be evaluated. Research is needed to identify sensitive, noninvasive and specific biomarkers of functional reproductive outcomes. This understanding is essential for the development of meaningful public heath policies and recommendations directed at reproducing women for ensuring appropriate nutrient intakes from food and the safe and effective use of dietary supplements for nutrients that are limited in the maternal diet.

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