B Vitamins in Breast Milk: Relative Importance of Maternal Status and Intake, and Effects on Infant Status and Function1,2

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ABSTRACT

Infants should be exclusively breastfed for the first 6 mo of life. However, maternal deficiency of some micronutrients, conveniently classified as Group I micronutrients during lactation, can result in low concentrations in breast milk and subsequent infant deficiency preventable by improving maternal status. This article uses thiamin, riboflavin, vitamin B-6, vitamin B-12, and choline as examples and reviews the evidence for risk of inadequate intakes by infants in the first 6 mo of life. Folate, a Group II micronutrient, is included for comparison. Information is presented on forms and concentrations in human milk, analytical methods, the basis of current recommended intakes for infants and lactating women, and effects of maternal supplementation. From reports of maternal and/or infant deficiency, concentrations in milk were noted as well as any consequences for infant function. These milk values were used to estimate the percent of recommended daily intake that infants fed by a deficient mother could obtain from her milk. Estimates were 60% for thiamin, 53% for riboflavin, 80% for vitamin B-6, 16% for vitamin B-12, and 56% for choline. Lack of data limits the accuracy and generalizability of these conclusions, but the overall picture that emerges is consistent across nutrients and points to an urgent need to improve the information available on breast milk quality.

Introduction

In 2001, after a debate ongoing for some years, the WHO concluded that infants should be exclusively breastfed for the first 6 mo of life rather than the previously recommended 4–6 mo (1). This recommendation was supported by evidence that extending exclusive breastfeeding would provide more protection against gastrointestinal infections. A review prepared as part of the background documentation evaluated the nutrient adequacy of exclusive breastfeeding for 6 mo (2). The authors identified maternal vitamins A and B-6 as the only maternal deficiencies that would lead to suboptimal concentrations in milk and adversely affect the infant. However, that review did not include deficiencies of other nutrients that can adversely affect the quality of breast milk, and the overall consultation concluded that apart from a possible negative effect of exclusive breastfeeding on iron status, “the available evidence is grossly inadequate to assess risks of deficiency in other micronutrients.”

There has been relatively little research on the extent to which the nutrient content of human milk is affected by maternal status and intake and how low milk concentrations affect infant status. One reason for this may be fear of discouraging breastfeeding if milk is found to be lacking in nutrients. Also, it is generally assumed that poor breast milk quality does not play a major causal role in the widespread growth faltering that occurs in developing countries early in the first year of life, because the breast milk content of nutrients usually associated with growth, such as zinc and phosphorus, is unaffected by maternal status. This assumption remains to be tested, however. Although infant micronutrient status at 6 mo of age is often poor (3,4), this tends to be attributed to, without study, substitution of some breast milk by nutrient-poor fluids and foods, low birthweight and/or preterm delivery resulting in poor infant stores at birth, and/or infant morbidity. Investigation has also been limited by other uncertainties, including the range of maternal intakes.

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of acceptable concentrations of nutrients in breast milk, the
efficacy of maternal supplementation for increasing micro-
nutrients in milk, and blood values that denote micronu-
trient deficiencies in the infant.

It is useful to categorize nutrients into 2 groups during
lactation (5). Group I nutrients (thiamin, riboflavin, vitamin
B-6, vitamin B-12, choline, retinol, vitamin A, vitamin D, se-
lenium, and iodine) are of the most interest in public health
nutrition, because their secretion into milk is rapidly and/or
substantially reduced by maternal depletion. Conversely,
maternal supplementation with these nutrients can increase
breast milk concentrations and improve infant status. In
contrast, the concentration of Group II nutrients (folate, cal-
cium, iron, copper, and zinc) in breast milk is relatively un-
affected by maternal intake or status; the mother gradually
becomes more depleted when intake is less than the amount
secreted in milk, and maternal supplementation benefits the
mother rather than the infant.

This article focuses on the example of B vitamins, all of
which except folate are Group I micronutrients. A literature
review was conducted to summarize relevant information
on B vitamins during lactation, including forms and con-
centrations in breast milk, methods of analysis, and values
used when setting current recommended intakes in infants
and lactating women. Articles were identified using the
PubMed database and the terms “lactation,” “human milk,”
“breast milk,” “milk,” “milk composition,” and the name
of each vitamin. Additional references were obtained from
those cited in these articles. The search years were not re-
stricted. Data from measurements made early in lactation
were avoided, because concentrations of thiamin, vitamin
B-6, and folate are lower and vitamin B-12 are higher
in the first postpartum weeks. However, at least in well-
nourished women, concentrations of all B vitamins are
relatively stable after about wk 3 of lactation (6). Especially
useful were reports of maternal and/or infant deficiency,
where values were noted for concentrations of the vitamins
in maternal blood, breast milk, and infant blood as well as
any effects on infant function. Where breast milk concen-
trations were clearly inadequate based on poor maternal and/or
infant status, these concentrations were used to estimate the
gap between the infant’s possible daily intake of the vitamin
from breast milk and their recommended intake. Any re-
ported effects of maternal supplementation on the mother,
breast milk, and infant were also noted. This review updates
a similar approach that the authors published nearly a de-
cade ago (7) and expands the information on B vitamins.

Current status of knowledge
Thiamin
Thiamin is present in human milk as thiamin (~30%) and
thiamin monophosphate (~70%) (8). The phosphorylated
forms of thiamin cannot cross membranes, but the free thia-
imin is released by phosphatase in the upper small intestine.
A common method of analysis is to oxidize the thiamin in
milk to the thiochrome, which is extracted and measured by
fluorescence spectrometry (9). In milk from well-nourished
women, the concentration used in setting infant and maternal
intake recommendations in the US and Canada and by the
FAO is 0.21 ± 0.04 mg/L (0.62 mmol/L) based on a 1985 re-
port, so the Adequate Intake (AI) recommended for infants is
0.2 mg/d in the first 6 mo, assuming that breast-fed infants
consume 780 mL milk/d (10). Contrary to the expected effect
for a Group I nutrient, thiamin supplementation (1.7 mg/d)
did not increase milk concentration to a higher level (0.24
mg/L) than in a nonsupplemented group (0.22 mg/L) of
U.S. women, but sample size was small (n = 12) and all had
received supplemental thiamin during pregnancy. A range of
thiamin in milk from ~0.02 to 0.35 mg/L was reported in
well-nourished Russian women, with concentration strongly
associated with maternal intake (from food and in some cases
supplements) over the range of ~0.5–5 mg/d (11).

The global prevalence of thiamin depletion is uncertain
but likely to be higher where diets are high in refined or pol-
ished unfortified grains and antithiamine factors or thiam-
nases and low in animal source foods and legumes (12).
HIV-AIDS is associated with poor thiamin status (13), and
in 1979 deficiency was prevalent in Ghanaian children
aged 6 mo to 6 y (14). Certainly, severe deficiency is much
less common than it was some 50 y ago. At that time, infantile
beri-beri was prevalent in countries such as Malaysia where
the staple diet was based on polished rice and animal source
food consumption was low. In 1942, Clements (15) reported
that when the breast milk thiamin concentration fell to <0.12
mg/L, infant growth was poor and <0.06 mg/L, growth was
completely arrested. The infants became pale, screamed,
and died rapidly from heart disease. He proved that the low
concentrations in human milk produced these symptoms
by feeding milk from thiamin-deficient women to dogs.

Maternal thiamin deficiency rapidly can result in very low
concentrations of the vitamin in milk, which are improved
with supplementation. Studies in The Gambia (16), India
(17,18), and Karen women in Thailand (19) between 1964
and 2001 reported concentrations of 0.16 mg/L, 0.22 and
0.11 mg/L, and 0.12 mg/L, respectively, substantially lower
than those used to set the AI in the US and Canada. Maternal
deficiency was common in all locations. In India, with sup-
plements of 0.2–20 mg/d (dosage was increased over an
8-mo period), milk thiamin increased from 0.11 to 0.27
mg/L. In The Gambia, maternal supplements of 2 mg/d im-
proved maternal status within 3 wk and infant status in 1–9
d, with breast milk content increasing to 0.22 mg/L (16). In-
fants developed symptoms of deficiency earlier if their
mothers were deficient during pregnancy. The functional
consequences of mild infant depletion have scarcely been in-
vestigated, but in Thai Karen, in whom deficiency results
from their dependence on polished rice and high intakes of
antithiamine factors in betel nut, fermented fish, and
tea leaves, maternal supplementation in pregnancy im-
proved the visual alertness of infants at birth. Recently,
Karen women in a refugee camp in Northern Thailand
were assessed for thiamin adequacy. In light of the earlier
observations of deficiency in Karen, which had been recog-
nized as a major cause of infant mortality, the women had
been given thiamin supplements during pregnancy and lactation (92 mg/d, which is far higher than the recommended intake of 1.5 mg/d) as well as food rations with and without thiamin-fortified flour. Total breast milk thiamin did not differ before (0.27 ± 0.13 mg/L) or after (0.29 ± 0.83 mg/L) fortification (n = 180).

In summary for thiamin, breast milk concentrations and infant status are strongly dependent on maternal intake and status (with maternal deficiency during pregnancy likely an additional risk factor for infant deficiency). Maternal supplementation rapidly improves the vitamin concentration in milk and infant status. An older report of severe deficiency suggests it may reduce infant growth (15).

**Riboflavin**

Riboflavin is found in human milk as flavin adenine dinucleotide (54%) which is 48% riboflavin by weight, riboflavin (39%), and small amounts of other flavins (20). The range in concentration of 5 well-nourished women was reported to be 180–800 µg/L, values 50% higher than those found when using older methods that did not correct for fluorescence quenching (20). The Institute of Medicine assumes a mean of 0.35 mg/L (10) based on data from the 5 American women (average 0.39 mg/L) and samples measured by a valid microbiological assay from 2 participants and reported by WHO (average 0.31 mg/L); values from prior studies were not be used due to the underestimation caused by inappropriate analysis. The daily secretion in breast milk of 43 well-nourished Russian women was ~0.010–0.55 mg and strongly correlated with maternal intake from diet and supplements over the range of ~1–8 mg/d (11).

The global prevalence of riboflavin deficiency is uncertain but likely very high, because intake depends primarily on animal source food and green vegetable consumption. Reported prevalences include 77% in lactating urban Guatemalan women (LH Allen and MT Ruel, unpublished data), 85% in night-blind pregnant Nepali women (21), 14% in Ghanaian infants (4), 30% in Kenyan school children (22), and in 1982, nearly all pregnant and lactating women and their infants in The Gambia (23). Recently, it was reported that 66% of adults in the UK, if they are low milk-consumers, have marginal riboflavin status correctable by supplementation (24).

Maternal riboflavin deficiency rapidly results in low milk concentrations of the vitamin. Values in The Gambia and India (5 studies between 1964 and 1982) revealed concentrations of 0.16–0.22 mg/L (18,23,25). Maternal supplementation postdelivery in The Gambia (2 mg/d) increased the milk concentration to 0.22–0.28 mg/L compared to 0.12 mg/L in a placebo group, with the effect of supplementation similar in milk samples taken between 10 and 84 d postpartum (25). In this population where almost all mothers and infants were deficient at baseline, status (assessed from erythrocyte glutathione reductase activation coefficients) and clinical symptoms of deficiency improved as did the iron status of the women. A riboflavin-fortified weaning food also rapidly normalized the status of the infants (23).

Thus, there is no doubt that milk riboflavin concentration was inadequate in this study.

**Vitamin B-6**

The predominant form of vitamin B-6 in breast milk is pyridoxal (75%), with smaller amounts of pyridoxal phosphate (9%), pyridoxamine, and pyridoxine, and mostly has been measured by HPLC for many years (26,27). The average concentration assumed when setting the AI for infants and lactation was 0.13 mg/L, based on a 1976 publication on a small group of U.S. women with intakes < 2.5 mg/d (28). The vitamin B-6 intake of mothers is a strong predictor of infant status (29).

The global prevalence of vitamin B-6 deficiency is very uncertain, because status is rarely measured. Estimates in a 2001 Indonesian study were 10% of children in urban areas and 40% of those in rural areas (30). In 1990 it was reported that ~40% of peri-urban Egyptian mothers had low concentrations of the vitamin in their milk [<415 nmol/L (0.10 mg/L), mean 435 (0.11 mg/L) nmol/L], although their status was not assessed. In the group with values < 415 nmol/L, birthweight of the infant was much lower. Lower milk concentrations were associated with abnormal behaviors in the women (less response to their infants’ vocalizations and less effective intervention when their infant was distressed) and their infants displayed less consolability, inappropriate build-up to a crying state, and poorer response to aversive stimuli (31). Convulsive seizures were observed in infants fed breast milk containing <0.10 mg thiamin/L (32). Some other evidence suggested suboptimal perinatal vitamin B-6 status in industrialized countries. In the US, low maternal intakes were related to poorer maternal status in pregnancy and lactation and lower levels in breast milk at 14 d postpartum (32). Infant pyridoxal-5′-phosphate concentrations predicted infant growth among 20 mother-infant pairs in the US (29). Of 44 Finnish infants, 7 had low vitamin B-6 status and also slower growth (33). Lactating women in the US (n = 25) whose vitamin B-6 intake was greater than the group median value had higher vitamin B-6 concentrations in their milk and their infants’ habituation and autonomic stability scores on the Brazelton Neonatal Assessment Scale were very strongly correlated with milk content of the vitamin (34). However, there is no evidence that any of the women were B-6 deficient and most took daily multivitamins during pregnancy; associations between the milk vitamin and infant behavior could have been due to other factors, including milk levels of other nutrients that were not measured.

Maternal supplementation with vitamin B-6 produces a rapid increase in milk concentrations. In U.S. women given a 15-mg dose of pyridoxine hydrochloride, there was a doubling of milk pyridoxal, pyridoxamine, and pyridoxal phosphate from 3 to 8 h later (26). All B-6 vitamers increased, although the proportional increase in pyridoxal was largest. A 2.5-mg dose increased milk concentration by ~50%. In a follow-up experiment, 47 mothers were supplemented throughout the first 6 mo of lactation with 2.5, 4.0, 7.5, or...
10.0 mg of vitamin B-6/d, given as multi-vitamin/mineral supplements (35). Only those infants whose mother took the 10-mg/d supplement met their AI of 0.3 mg/d. Their plasma pyridoxal phosphate concentration paralleled that of maternal intake. However, the 2.5-mg/d supplement supported a milk concentration ranging from 0.89 to 1.31 nmol/L (0.22–0.32 mg/L) and normal infant growth and vitamin B-6 status. An older study in India is the only maternal supplementation trial that could be identified in a developing country (18). In this small study (n = 10), 0.4–40 mg/d (the dose was increased daily during the first 8 mo of lactation) in a multiple micronutrient supplement increased milk concentrations to 0.16 mg/L compared to 0.08 mg/L in placebo controls (18).

For vitamin B-6, we conclude that the limited evidence available suggests that breast milk concentrations and infant status definitely reflect maternal intake and status and that maternal supplementation rapidly increases the amount in milk. Associations between milk vitamin B-6 and infant size at birth or growth, and with infant behavior, may be important, but more data on this question are needed.

Vitamin B-12

Deficiency and depletion of vitamin B-12 is prevalent where the intake of animal source foods is low, because these foods contain almost all of the vitamin B-12 in the food supply, except for fortified foods. Reported prevalences of deficient plus low serum B-12 concentrations reported in Latin America are typically around 40% and reach 60 to 80% in some populations in Africa and Asia, affect all ages, and are associated with low animal source food consumption (36). Clearly, risk of deficiency is not limited to strictly vegetarian diets; intakes (37) and status (38) of lacto-ovo vegetarians are substantially lower than those of omnivores.

The neonatal period is thought to be a special period of vulnerability to vitamin B-12 deficiency (39). Maternal status prior to and during pregnancy is strongly associated with cord blood concentrations of vitamin B-12 and total homocysteine (40) as well as infant stores of the vitamin at birth (39). Severe maternal deficiency, caused by vegetarianism or undiagnosed pernicious anemia, is well recognized to result in clinical symptoms of deficiency within a few months of delivering an infant. A summary of case studies indicates that symptoms appear around 4–7 mo of age and include severe growth stunting (length, weight, and head circumference) and cerebral atrophy and a large number of muscular, behavioral, and other developmental problems, some of which are not reversed by treatment in ~40–50% of cases (41). Importantly, in all cases, the infants were exclusively breastfed, suggesting that the low vitamin B-12 concentration of breast milk was a contributory factor.

Vitamin B-12 in breast milk is tightly bound to haptocorrin, which is present in much higher amounts than in serum or plasma and has to be released from this binding to be measured accurately. Investigators have used a variety of approaches to remove the vitamin from haptocorrin, including pretreatment with papain or other enzymes. However, a method developed in 2009 using a cobinamide-sepharose column to remove apo-haptocorrin revealed that previous methods would have under- or overreported the amount of vitamin B-12 in breast milk depending on the method subsequently used for measuring the vitamin (42). The Institute of Medicine used a value of 0.43 µg/L (310 pmol/L) for the concentration in milk based on samples from 9 unsupplemented Brazilian women and an older assay method (43). However, using the new method, the median value in a group of 25 well-nourished Californian women, most of whom consumed supplements containing 5 µg/d during pregnancy, was 1.2 µg/L, whereas in 183 low socioeconomic status women in peri-urban Guatemala City, concentrations fell below the detectable limit of 50 pmol/L in 65% of the participants and <100 pmol/L in 77% (44). More data are needed to establish normal values in unsupplemented women.

There has been relatively little research on the effect of supplementing lactating women with oral doses of the vitamin; the usual therapeutic approach is to give high (500–1000 µg) i.m. doses of vitamin B-12 to the infant and mother once clinical symptoms are apparent. In a small pilot study, we supplemented Guatemalan lactating women with vitamin B-12 depletion (serum vitamin B-12, 150–221 pmol/L) with 3–1000 µg/d of the vitamin for 2 mo (K.L. Deegan, et al., unpublished data). Even at the highest doses, the concentration in breast milk increased from 67 to only 180 pmol/L, <1% of any dose was transferred to milk, and no dose reduced infant serum or urinary methylmalonic acid. Thus, maternal supplementation with vitamin B-12 in lactation may be too late to restore adequate milk concentrations and infant status. Further studies of this important question are needed.

Choline

In human milk, the majority of choline is found (using proton NMR spectroscopy) as phospho- and glycerophosphocholine, with smaller amounts of free choline, phosphatidylcholine, and sphingomyelin (45). HPLC and LC-MS analysis produce similar values. The total choline concentration doubles in the first week postpartum. Most formulas contain free choline, with a total concentration less than that of mature milk.

The AI for lactation and the infant assumes a milk choline concentration of 160 mg/L (1.5 mmol/L). Although choline is both consumed in the diet and endogenously synthesized, based on limited human data the choline concentration in milk is related to maternal intake. In 46 healthy women in the US, total dietary choline intake was significantly but weakly correlated with breast milk phosphatidylcholine but not other choline metabolites (46). In the same study, 48 women were randomly assigned to supplementation with 750 mg choline/d from mid-pregnancy to 45 d postpartum. Their milk concentrations of free choline, betaine, and phosphocholine were significantly higher. For the placebo and supplemented groups combined, correlations between total choline intake and milk concentrations of...
free choline, phosphatidylcholine, betaine, and phospho-
choline were all significant, and plasma choline was corre-
lated with milk choline. A novel aspect of this study was
the observation that common single nucleotide polymor-
phisms in genes for methylene tetrahydrofolate reductase
and phosphatidylethanolamine N-methyltransferase, enzymes
required for the endogenous synthesis of choline, altered the
slope of the relationships between choline intake and the con-
centrations in milk and maternal plasma.

A relatively large study of Turkish women in 2005 mea-
sured choline components in the serum of lactating and
nonlactating women and infants, and in breast milk, at
stages from soon after delivery through >1 y postpartum, al-
though samples were not collected longitudinally in the
same participants (47). The total choline concentration in
milk ranged from 1.35 to 1.60 µmol/L (144–170 mg/L) in
samples from 142 women between 12 and 180 d of lactation.
Maternal serum free choline, phosphocholine, and glycer-
ophosphocholine were correlated with the free choline in
milk and the free choline in infant serum was correlated
with the milk concentration of free choline, phosphocho-
line, glycerophosphocholine, and total choline (r values
0.21–0.47). It was noted that lactating women had much
higher serum free and phospholipid-bound choline than
nonlactating women and that these values fell gradually dur-
ing the first 6 mo postpartum.

In the only study identified from a developing country
(48), the milk choline concentration was lower in Ecuador
than in Boston. In 1982, 40% of a group of 55 women in
Ecuador had concentrations < 1 nmol/L and 6% had values
> 3 nmol/L. Comparative concentrations in a group of 11
women in the US were 0% < 1 mol/L and 55% > 3 nmol/L.
To date, there are no supplementation data from developing
countries and a lack of information on whether low choline
in breast milk affects infant development.

Folate
5-Methyl tetrahydrofolate is the predominant form of folate
in human milk. The vitamin is bound to folate binding pro-
tein and a tri-enzyme procedure increased measured con-
centrations by 85% compared to previous methods (49).
The AI for lactation and pregnancy assumes that the average
milk folate concentration is 85 µg/L (193 nmol/L), based on
similar values among 4 reports between 1986 and 1991 (10).
A value of 81 µg/L was reported in 2009 from 53 U.S.
women who were not supplemented during lactation (50).
Supplementation with 5-methyl tetrahydrofolate or folic
acid did not affect milk concentrations of milk folate. Matern-
al folic acid supplements increased the amount of unme-
tabolized folic acid in milk, although this represented only
8% of total milk folate.

The global prevalence of folate deficiency and depletion is
not well understood, but it appears that it may be a condi-
tion that is at least as common in industrialized countries
as in poorer regions of the world (51,52). Folate is particu-
larly lacking in refined cereals and intakes may be higher
where the usual diet is higher in legumes, vegetables, and
fruits (51). Unlike those B vitamins classified as Group I nu-
trients for lactation, folate is a Group II nutrient, because
concentrations in human milk are maintained even when
the mother is deficient in the vitamin and are unaffected
by maternal supplementation (53). In rural Mexican Otomi,
milk concentrations were ~100 nmol/L, unrelated to mater-
nal status and unaffected by maternal supplementation with
400 µg folic acid provided from 22 to 138 d postpartum
(54). As a consequence of maintaining the level of folate se-
cretion in milk, women with low intakes will become more
depleted as lactation progresses. Earlier reports that mater-
nal iron deficiency may lower milk folate were not con-
firmed in a subsequent study in Mexico (54).

Serum folate was measured in a longitudinal study of 361
healthy Norwegian infants from birth through 2 y of age
(55). Neither folic acid fortification of flour nor periconcep-
tional folic acid supplementation was officially recommen-
ded at the time of the study. Serum folate concentrations
averaged ~ 47–55 nmol/L in the first 6 mo of life, were sub-
stantially lower at age 12 mo, and fell to one-half the birth
concentrations by 24 mo. There were no differences in se-
rum folate between breast-fed and nonbreast-fed infants at
any age. However, serum folate at 6 mo was positively corre-
lated with the duration that the infant had been exclusively
breastfed. Thus, exclusively breast-fed infants have good fo-
late status when fed by higher income (56) or more poorly
nourished (54,57) mothers, but the folate status of the
mothers themselves may deteriorate. Maternal supplemen-
tation in lactation may therefore be important for preserving
maternal stores, which are especially important for sub-
sequent conception and pregnancy (58).

Inadequacy of intakes
From this review, studies were identified in which the milk
concentration of the vitamin was insufficient to support ad-
equate infant status and/or their mother was clearly defi-
cient. Using the reported concentration of the nutrient in
milk in the most appropriate studies and assuming intake
was 780 mL/d during the 1st 6 mo of life (as was assumed
when setting the AI for the US and Canada), the amount of
the vitamin that the exclusively breast-fed infant could ob-
tain from breast milk was calculated and compared to the
AI recommendation (Table 1). Assuming that the AI recom-
mandations are correct, it is clear that intakes of exclusively
breast-fed infants fed by deficient mothers can be very inad-
equate. In our previous publication on this topic, we also
found that estimated intakes compared to the AI could be
56% for vitamin A, 50% for vitamin C, 6–23% for iodine,
and 52% for selenium (7).

Conclusions
Poor maternal status of Group I nutrients, including thia-
emin, riboflavin, vitamin B-6, vitamin B-12, and choline
used as examples in this review, causes the concentrations
of these nutrients to be low in breast milk and the infant
to become deficient. In contrast, in maternal folate deple-
tion, breast milk folate concentrations are maintained; if
the mother’s intake is inadequate, she will become more deple-
pitated in the vitamin as lactation progresses. The prevalence
of maternal deficiency of these micronutrients is uncertain
but likely to be high, especially for riboflavin and vitamin
B-12 in the many poorer populations of the world that
have a low intake of animal source foods. Based on the growth
retardation that can accompany inadequate intakes of thia-
min and vitamins B-6 and B-12 in breast milk, low milk con-
centrations of these nutrients could contribute to the
widespread growth stunting that occurs in the first year of
life. Maternal supplementation during lactation rapidly
increases the concentrations of thiamin, riboflavin, and vitamin
B-6 in milk, but increases in vitamin B-12 were small even
when high doses were given to the mother for 2 mo.

The estimates of inadequate intake in Table 1 have de-
tine limitations. The AI values are based on a very small
number of samples in most cases and often collected from
women whose intake from foods and supplements was not
clear. In the case of vitamin B-12 milk, the AI values used
were probably measured using invalid methods. There are
also a very limited number of studies of breast milk concen-
ctrations in different locations and methods used for ribofla-
vin and vitamin B-12 were not valid in older studies. Infant
deficiency could have resulted in part from breastfeeding not
being exclusive. Nevertheless, the general picture is consist-
ten across all the Group I nutrients.

Clearly, much more needs to be known about the micro-
nutrient composition of breast milk in different population
groups with a range of nutritional status. Sample collection
methods need to account for any diurnal and intra-individ-
ual variation and data are still limited concerning this ques-
tion. Reference values need to be developed based on concen-
trations in the milk of well-nourished but unsupple-
mented women. Accurate analytical methods are now avail-
able for all vitamins in human milk, but for some nutrients
these have been used in the analysis of very few samples. MS
and other methods can simultaneously analyze concen-
trations of several vitamins, including most of the B vitamins,
which should enable additional data to be more efficiently
obtained. Much more information is also needed on the mi-
nutrient status of exclusively breast-fed infants, including
normal reference values for blood measures, and functional
consequences of deficiency or depletion. Taken together,
such data could enable the development of estimated ade-
quate requirements for infants that provide a more valid ba-
sis for calculating the prevalence of inadequate intakes and
requirements in this age group. The limited available data
suggest that poor maternal vitamin B-12 status during preg-
nancy affects infant stores of the nutrient at birth. Inade-
quate maternal intake during lactation is most likely to
lead to further maternal depletion, especially in the case of
the Group II nutrients such as folate.

Taken together, these gaps in our information about the
micronutrient content of breast milk, especially in popula-
tions with poor nutritional status, indicate that this should
be a research priority. This does not imply that exclusive
breastfeeding should not be recommended for the first 6
mo of life. Rather, the purpose of this review is to raise
the issue that more attention needs to be paid in various lo-
cations concerning whether women with low stores or in-
takes of nutrients should be provided with supplements
during lactation, the doses effective for increasing milk con-
centrations sufficiently to improve infant status, and the ma-
ternal and infant health benefits of this strategy.

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soluble and water-soluble vitamin contents of breast milk from Japa-

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**TABLE 1** Milk concentrations assumed when setting the AI for the first 6 mo of life compared to value in milk of deficient infants and percent of AI this represents

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Milk concentration/L assumed for setting AI</th>
<th>Milk concentration reported for deficient infants (reference)</th>
<th>AI consumed by deficient infants, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiamin, mg</td>
<td>0.21</td>
<td>0.16 (16)</td>
<td>60</td>
</tr>
<tr>
<td>Riboflavin, mg</td>
<td>0.35</td>
<td>0.21 (16)</td>
<td>53</td>
</tr>
<tr>
<td>Vitamin B-6, mg</td>
<td>0.13</td>
<td>0.10 (16,18,59)</td>
<td>80</td>
</tr>
<tr>
<td>Vitamin B-12, µg</td>
<td>0.42</td>
<td>&lt;0.05</td>
<td>16</td>
</tr>
<tr>
<td>Choline, mg</td>
<td>160</td>
<td>90 (48)</td>
<td>56</td>
</tr>
</tbody>
</table>

1 Milk intake was assumed to be 780 mL/d.

2 Study selected because of larger sample size. Note that breast milk concentration was higher than in India (18) and Thailand (19).

3 Selected based on largest sample size and clear evidence of deficiency in mothers and infants.

4 Similar values from these studies in India, The Gambia and Egypt.

5 Selected because of large sample size and evidence of deficiency in mothers and infants.

6 Based on data from the only available low income country (Ecuador). Forty percent of samples were lower than this value. Infant choline status was not assessed.


