Models linking nutritional deficiencies to maternal and child mental health1–3

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ABSTRACT
The primary goal of this article was to illustrate how nutritional deficiencies can translate into adult or child mental health problems. Whereas brain development and function play an essential role in the etiology and maintenance of mental health problems, what is required are models that go beyond nutrition-brain relations and integrate the contributions of nutritionally related contextual and behavioral characteristics. Four such models are presented. The multiple risks model derives from evidence showing covariance between nutritional deficiencies and other life stressors. Given that poorly nourished adults may be less able to actively cope with stressors, nutritional deficiencies may accentuate the negative impact of stress exposure on mental health. The cross-generational model is based on evidence showing less adequate patterns of mother-child interactions when mothers are poorly nourished. Impairments in mother-child interactions increase the likelihood of child mental health problems and the risk of subsequent child nutritional deficiencies. The attachment model derives from evidence showing that poorly nourished infants may be less likely to elicit the types of maternal child-rearing patterns that translate into secure infant-mother attachments. Insecure attachments in infancy are associated with an increased risk of both short-term and long-term child mental health problems. The temperament model is based on evidence documenting that certain patterns of infant temperaments are related to an increased risk of later behavioral problems. Infant nutritional deficiencies can influence the development of temperament, and certain temperament patterns can contribute to an increased risk of infant nutritional deficiencies.

MODELS LINKING NUTRITIONAL DEFICIENCIES TO MATERNAL AND CHILD MENTAL HEALTH
Historically, research on the behavioral-developmental consequences of nutritional deficiencies initially focused on cognitive development and function (1). The prevailing hypothesis guiding much of this initial research was that nutritional deficiencies adversely influenced central nervous system (CNS) development, which directly led to cognitive deficits (2). A consistent body of evidence documents the detrimental influence of both protein-calorie malnutrition (3, 4) and micronutrient deficiencies on CNS development and function (5, 6). Evidence further documents that the nutrients related to CNS development and function were also those that influence individual differences in cognitive development and cognitive performance (7, 8). However, as research findings continued to accumulate, it became increasingly clear that understanding the impact of nutritional deficiencies on cognitive development and function required going beyond just direct nutrition-brain pathways (9–11).

As a means of more accurately conceptualizing the variety of pathways through which nutritional deficiencies translate into cognitive deficits, Levitsky and Barnes (12) proposed the functional isolation hypothesis. The core premise of the functional isolation hypothesis is that the cognitive and behavioral deficits found in malnourished organisms are the result of both direct influences of malnutrition on CNS development and function, as well as indirect nutritionally driven reductions in young organisms’ involvement with their environment, and alterations in the nature of parenting behaviors directed toward the poorly nourished infant (11, 13).

Similar to what is known about nutrition-cognition pathways, brain development and brain function play an essential role in the etiology and maintenance of mental health problems (14, 15). Many of the nutrients documented as influencing brain development are also associated with mental health problems, for example, folate (16), essential fatty acids (17), and iron (18). However, on the basis of what is known about the role of functional isolation processes and the complex nature of pathways linking the CNS to mental illness (19, 20), understanding how nutritional deficiencies translate into mental health problems will again require models that go beyond nutrition-brain relations and incorporate contextual and behavioral characteristics that are linked to nutritional deficiencies. Four multidimensional models linking nutritional deficiencies to adult and child mental health outcomes are described in the following sections.

Nutrition and adult mental health
Recent reviews have documented both the relatively high prevalence of maternal depression and the adverse consequences of maternal depression for maternal and family functioning and

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for children's health and development (21, 22). For these reasons, our discussion of models relating nutrition to adult mental illness will focus on maternal depression.

**Multiple risk model**

Models relating nutrition to maternal depression should include a nutrition-CNS pathway. One such pathway could involve changes in serotonin, which is a mood regulator that is linked to depression (23). Synthesis of serotonin depends, in part, on intake of foods containing the amino acid tryptophan (24) and vitamin B-6 (25). However, direct nutrition-brain relations are not the only possible pathway leading to maternal depression.

Nutritional deficiencies rarely occur in isolation from other bioecologic and psychosocial risks factors such as economic stress, chronic illness, low social support, or unstable life circumstances (11). Women at increased risk of maternal depression are those who are dealing with exposure to multiple cumulative bioecologic and psychosocial stressors, including nutritional deficiencies (22). However, the detrimental impact of exposure to multiple risks can be attenuated by environmental protective factors, such as higher levels of education (26, 27) or greater social support (28, 29). Thus, risk of maternal depression increases as the number of encountered risk factors, including inadequate nutrition, increases and decreases as the number of protective factors increases.

In a standard risk model, all risk factors are equally weighted. However, within a risk-protective framework, greater weighting of nutritional deficiencies should be considered, given that poorly nourished adults have less energy available for active coping with daily stresses (30). By reducing a women’s ability to actively cope with multiple stresses, nutritional deficiencies may accentuate the impact of multiple risk factors on maternal depression.

**Cross-generational model**

An alternative way of conceptualizing nutrition-maternal depression pathways is to look across generations. Children of depressed mothers are also at risk of behavioral problems throughout childhood, including symptoms of depression (31, 32). In part, this increased risk may reflect the inheritance of maternal genes, predisposing to an increased risk of depression (31). However, the increased risk of depression for children of depressed mothers may also reflect impairments in mother-child interaction patterns that occur when mothers are depressed (33, 34).

Furthermore, maternal depression is not the only factor that can impair the quality of mother-child interactions. Evidence also suggests that the quality of mother-child interactions can vary depending on the level and quality of maternal dietary intake (35–37). Thus, to the extent that maternal nutrition covaries with maternal depression, children of depressed, poorly nourished women may be at even greater risk of receiving developmentally inappropriate rearing.

In addition to being at increased risk of impairments in parent-child interactions, children of depressed women may also be at increased risk of inadequate nutrition. A number of studies have shown an increased risk of breastfeeding problems or shorter durations of breastfeeding (27, 38) and slower physical growth for infants of depressed mothers (32, 39, 40). Evidence from longitudinal studies suggests that maternal depression undermines infant growth, rather than the reverse order of maternal depression occurring as a reaction to infants’ growth faltering (39, 40). These findings suggest that maternal depression and maternal nutritional deficiencies can increase the risk of inadequate or inappropriate parenting, which can increase a child’s risk of both nutritional deficiencies and mental health problems.

**Summary: models involving multiple risk and cross-generational effects**

Pathways between maternal nutritional deficiencies and maternal depression involve CNS function, exposure to multiple covarying risk factors, accentuation of the impact of risk exposure by inadequate nutrition, and cross-generational transmission of the nutrition-driven risk of depression. As shown in Figure 1, the multiple risk and cross-generational models are not mutually exclusive and may in fact co-occur.

![FIGURE 1](https://academic.oup.com/ajcn/article-abstract/89/3/935S/4596784)
disorders. Nutritional deficiencies in infancy and childhood can influence not only CNS function but also CNS structural development (3, 42) and the development of neurotransmitter systems (43). However, alterations in brain development are not the only pathways linking nutrition to child mental health problems.

Attachment model

The development of a secure attachment relation provides a context that allows infants to explore their world, learn how to regulate their emotions, and learn how to interact with others (44). Infants with insecure attachments to their mothers are at risk of later impaired social relationships (45, 46) and an increased risk of preschool and childhood behavioral problems (47, 48). Availability, sensitivity, and responsivity of the primary caregiver are essential for the formation of a secure attachment. A variety of factors can adversely influence the quality of mother-infant interactions and, thus, result in the development of an insecure attachment. For example, infants of depressed mothers are at significantly increased risk of developing an insecure attachment (51, 52).

To the extent that inadequate maternal nutrition impairs the quality of mother-infant interactions, we would expect infants of poorly nourished mothers to be at risk of insecure attachments. At present, virtually no evidence exists that directly relates the quality of maternal nutritional status to offspring attachment. However, inadequate maternal nutrition is not the only pathway through which nutrition can influence a child’s risk of insecure attachment. The quality of caregiver-infant interactions is bidirectional and reflects reciprocal interactions as infants and caregivers respond to one another. Poorly nourished infants may be less likely to elicit the types of maternal interactions that can translate into secure attachments. Studies have documented variability in the quality of parent-child interactions as a function of the adequacy of child nutrition (35, 37, 53), even after the adequacy of maternal nutrition was accounted for (9, 54).

Although very little evidence is available, results from a few studies conducted in developing countries have reported that infants with moderate-to-severe growth retardation are significantly more likely to be insecurely attached than are infants with more adequate growth (55–58). Whether these findings can be generalized to populations that are not severely malnourished, or to other measures of nutritional status besides physical growth, is a critical question. Initial results from a collaborative project done with colleagues at the Institute for Nutritional Investigation in Peru suggest generalizability; infants who display mild levels of growth retardation or have a poorer iron status are more likely to show patterns of behavior reflecting insecure attachments (TD Wachs, G Posada, O Carbonell, H Creed-Kanashiro, P Gurkas, unpublished observations, 2008).

The available evidence lends tentative support to a pathway wherein poorly nourished infants are less able to elicit developmentally appropriate rearing patterns from their primary caregivers, which, in turn, results in poorly nourished infants being at a significantly greater risk of insecure attachment. Infants with insecure attachments are at a significantly greater risk of later behavioral and mental health problems than are securely attached infants. To the extent that maternal and infant nutritional status covary, the path from nutritional deficiencies to subsequent child behavior problems should be even stronger.

Temperament model

A growing body of evidence links nutritional deficiencies in infancy to characteristic patterns of infant temperament. Much of this evidence is focused on iron deficiency with a consistent pattern of findings, from newborns through 5 y of age, which indicates that children with iron deficiency have a pattern of temperament characterized by increased inhibition or wariness, increased irritability or negative emotionality, and lower levels of positive affect, sociability, and reactivity (9, 59, 60). Other studies have reported lower activity levels, reactivity, alertness, self-regulation, sociability, and more negative emotionality for children with mild-to-moderate deficiencies in protein-energy or B vitamin intake (61).

The link between nutrition, temperament, and children’s behavioral problems is based on a consistent body of evidence documenting that individual variability in infant temperament is related to later increased risk of behavioral problems in children (62, 63). For example, infants with inhibited temperaments are at greater risk of later internalizing problems such as anxiety disorders, whereas infants high in negative emotionality are at greater risk of externalizing problems such as aggression (64, 65). Given that individual differences in temperament are related to brain development and brain function (66, 67), there is likely to be a pathway from nutritional deficiencies to areas of brain development and function that mediate individual differences in temperament and from individual differences in temperament on to risk of internalizing and externalizing disorders. However, there are likely to be alternative nutrition-related explanatory pathways that also predict later behavior disorders. For example, depressed mothers are more likely to perceive their infants as being more fussy-difficult to manage than are nondepressed mothers (68, 69).

In addition, relations between nutrition and temperament are also likely to be bidirectional. For example, infants with fussy-difficult unmanageable temperaments are at increased risk of reduced levels of breastfeeding and of higher levels of postweaning feeding problems (70). Infants with these temperament patterns are at particular risk of feeding problems if their mothers are depressed and having problems interacting with their infants (71).

Summary: pathways involving infant nutrition and behavioral problems

Available evidence documents that early nutritional deficiencies can result in insecure attachments and inhibited or “difficult” temperament patterns in infancy and childhood. Both insecure attachments and inhibited or difficult temperament patterns, in turn, significantly increase the risk of later adjustment disorders. In addition, having an inhibited or difficult temperament may increase an infant’s risk of less adequate nutrition. The attachment and temperament models are complementary and not mutually exclusive (Figure 2). Furthermore, the adult and child models shown in Figures 1 and 2 are also complementary.

CONCLUSIONS

Models based exclusively on nutrition and CNS pathways do not provide sufficient explanations of how nutritional deficiencies translate into maternal and child mental health problems.
Sufficient explanations need to incorporate nutrition-CNS pathways as well as pathways involving maternal characteristics such as depression, contextual characteristics such as child rearing patterns, and individual characteristics such as child attachment and temperament. Whereas models including brain, context and maternal and child characteristics are more complex, they also are a more accurate representation of the processes involved in nutrition-mental health relations. Evidence involving interventions designed to promote early cognitive development has shown stronger and more lasting outcomes when intervention strategies are multidimensional in nature, integrating both nutritional and nonnutritional factors (72). It is likely that the same result would occur when multidimensional pathways linking nutrition to mental health outcomes are used to formulate intervention strategies designed to prevent or treat adverse mental health consequences in poorly nourished mothers and children. (Other articles in this supplement to the Journal include references 73–78.)

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REFERENCES


