Maternal thiamine deficiency: still a problem in some world communities\textsuperscript{1,2}

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Thiamine deficiency remains an important health care issue in many world populations. Causes of thiamine deficiency include inadequate diets, consumption of foods containing thiaminases or antithiamine compounds, and prolonged cooking of foods. In addition, clinical disorders such as chronic alcoholism, HIV-AIDS (1), and gastrointestinal disorders are associated with a high incidence of thiamine deficiency. Populations at particularly high risk include victims of political trade embargos (2) and displaced persons in refugee camps. In this latter regard, an important study by McGready et al (3) in this issue of the Journal describes a high incidence of postpartum thiamine deficiency (assessed by using the erythrocyte transketolase activation assay) in a group of Karen women from a refugee camp on the Thailand-Burma border. Up to 58% of these women were thiamine deficient at 3 mo postpartum despite the distribution in their rations of what appeared to be adequate dietary thiamine supplements. Thiamine supplementation was limited during pregnancy to women with peripheral neuropathy and other clinical signs of beriberi. This nutritional policy was started when it was recognized that infantile beriberi is a major cause of infant mortality in this population.

It is well established that thiamine requirements are increased during pregnancy and lactation. Increased thiamine requirements during the third trimester of pregnancy are generally thought to result from sequestration of the vitamin by the fetus and placenta. For example, concentrations of thiamine and other water-soluble vitamins are 2-fold higher in umbilical cord blood than in maternal blood (4). Additionally, McGready et al (3) observed that, despite the high incidence of thiamine deficiency in the Karen mothers, breast-milk thiamine concentrations remained within normal limits, suggesting preferential delivery of thiamine to the milk at the expense of the mother.

It is clear that a major cause of thiamine deficiency in many parts of the world is a staple diet of milled or polished grains, despite >50 y of accumulated evidence showing that such a diet leads to serious thiamine malnutrition. In the Karen study, polished rice rather than the traditional brown rice was the staple diet of most of the population. The population’s choice of polished over brown rice was the result of 2 considerations: the prolonged cooking time and additional fuel required to cook brown rice and the cultural stigma associated with consumption of brown rice, which is considered to be a food of the poor.

Thiamine deficiency in the Karen population was exacerbated by inclusion in the diet of foods such as betel nut, fermented tea leaves, and fish paste, all of which contain high concentrations of thiaminases or antithiamine compounds. In other studies, the presence of thiaminases in the diet was shown to result in the need for additional dietary supplements. For example, in a study from northeastern Thailand, thiamine supplementation of 100 mg/d was found to be sufficient to counteract the lowering of thiamine status resulting from the consumption of fermented fish paste but not adequate to restore thiamine status in individuals chewing betel nut (5). Betel nut chewing is common in many parts of Southeast Asia and in some African countries, and it is likely that the thiamine supplements to the ration in the Karen population were insufficient given the widespread practice (86%) of betel nut chewing in this population. Reports of a high incidence of thiamine deficiency during pregnancy and lactation were previously reported in India, Malaysia, and Ghana, where, in some cases, the consumption of foods rich in thiaminases was also implicated (6).

Studies in experimental animals have shown that thiamine deficiency leads to delayed maturation profiles for enzymes involved in brain energy metabolism (7), delayed lipogenesis (8), and intrauterine growth retardation in the offspring. Transfer of antithiamine compounds to the fetus has not been adequately studied. It has been suggested that thiamine deficiency is also a cause of intrauterine growth retardation in humans. For example, in a study from Germany, it was observed that mothers with pregnancies complicated by intrauterine growth retardation had significantly lower erythrocyte thiamine concentrations than did mothers with a normal pregnancy (9). In the study by McGready et al, visual alertness was found to be significantly better in infants of thiamine-supplemented mothers than in those of unsupplemented mothers, but the sample size was considered to be too small to assess the association between thiamine deficiency and the presence of intrauterine growth retardation in these cases.

There is an urgent need for adequate thiamine supplementation in high-risk populations. McGready et al suggest fortification of refugee rice or a return to traditional brown rice. In addition, the consumption of foods containing antithiamine compounds should be discouraged in these populations, particularly during pregnancy and lactation. Further studies of the long-term effects of...
maternal thiamine deficiency on human brain development in these high-risk populations are clearly warranted.

REFERENCES