THIAMIN DEFICIENCY: A DIRECTED CASE STUDY

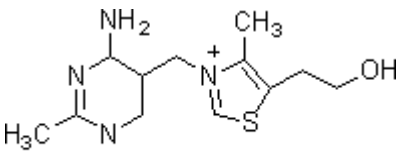
by
Preeti Dhar
Department of Chemistry
SUNY New Paltz

The people of Malaysia belong to three communities: the local people called Malays, immigrants from South India called Tamils, and immigrants from China. During the early 20th century, the Malays lived mainly in coastal villages, the Tamils worked on rubber estates, and the Chinese worked in tin mines. In general, the people were very poor and they consumed a limited variety of foods.[1]

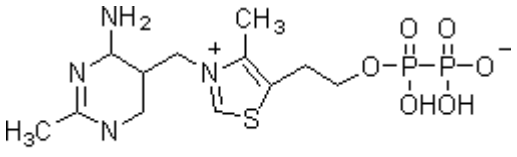
Rice was the staple diet of all three communities and therefore the key source of caloric and nutrient content. Malays typically grew their own rice and dehulled it before consuming it; dehulling partially removes the aleurone layer of the rice seed. Following their South Indian food habits, the Tamils ate parboiled rice, and the Chinese ate polished white rice imported from Thailand. It was found that among the three Malaysian communities, the Chinese community was much more prone to suffer from some degree of sub-optimal thiamin intake. Out of every 1,000 Chinese inhabitants, about 120 had mild thiamin deficiency, about 80 had severe thiamin deficiency, and about 16 people had died of it. Sub-optimal intake of thiamin was rare in Malays and was not reported at all in Tamils.[1]

Thiamin, also called Vitamin B1, is required for all tissues, and its deficiency causes the disease Beri-beri. Less severe deficiency results in non-specific symptoms such as weight loss, malaise, irritability, and confusion. Although the origin of the word Beri-beri is disputed, it is believed to be a Philippine word meaning "I can't, I can't" and was probably coined to refer to the lack of neuromotor coordination in people afflicted with this disease.[2]

Thiamin is water-soluble and is stable in the acidic medium. It is unstable to heat, oxygen, and UV light in neutral and alkaline medium. The active form of thiamin is thiamin pyrophosphate (TPP). This active form is a cofactor for pyruvate dehydrogenase and transketolase enzymes that are involved in the metabolism of carbohydrates. These enzymes play an important role in the biosynthesis of a number of cell constituents such as neurotransmitters, pentoses, and reduced equivalents, used in oxidant stress defenses.[3]



Thiamine



Thiamine Pyrophosphate

The human body cannot store thiamin and therefore requires a continuous supply of the vitamin. The thiamin in an individual on a thiamin-free diet typically depletes in fewer than two weeks. Thiamin absorption is affected by compounds, called thiamin antagonists or anti-thiamin factors, that alter the structure of thiamin. These compounds include alcohols, polyphenols, flavonoids, and thiaminase, a heat labile enzyme found in certain foods.[2]

In affluent countries, where the diet is rich in thiamin, the most common cause of thiamin deficiency is excessive alcohol consumption. Alcohol intake affects thiamin status in two ways. First, alcoholics get a significant percentage of their daily energy intake from alcoholic beverages, which are nutrient deficient. In other words, alcoholics replace their consumption of nutrient dense foods that are part of their normal diet with the consumption of nutrient deficient alcohol. Second, alcohol inhibits the intestinal ATPase involved in the absorption of thiamin.[4]

Sub-clinical thiamin insufficiency associated with excessive alcohol consumption produces an encephalopathy called Wernicke-Korsafoff syndrome. Patients with this syndrome have a higher than normal incidence of abnormal transketolase, which has a very low binding affinity to TPP. This problem can be overcome by putting patients on high-dose thiamin therapy. Some patients, however, might have a form of abnormal transketolase that does not bind at all with TPP. Such people do not respond to high-dose thiamin therapy.[4]

Thiamin deficiency is observed in individuals who have low thiamin intake and a diet rich in polished rice, especially if their diet also consists of anti-thiamin factors such as tea, coffee, raw fermented fish, and betel nuts. The minimum amount of thiamin needed by the body can increase because of increased physiological or metabolic demands arising from pregnancy and lactation, heavy physical exertion, illnesses like cancer, liver diseases, hyperthyroidism, and surgery.

Since TPP is required for carbohydrate metabolism, the recommended dietary allowances for thiamin are based on the assumption that its intake is related to total energy intake. The recommended dietary allowance in the United States is 0.5 mg/1000 Kcal.[5]

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[1] World Health Organization. 1999. *Thiamine deficiency and its prevention and control in major emergencies* (WHO Monograph Series No. 13). Geneva: World Health Organization.

[2] Guthrie, H.A. and M.F. Picciano. 1995. *Human Nutrition*. St. Louis: Mosby Publishing. pp. 476-483.

[3] Singleton, C.K., and P.R. Martin. 2001. Molecular mechanisms of thiamine utilization. *Current Molecular Medicine* 1(2): 197-207.

[4] Coombs, G. 1992. Chapter 10 in: *The Vitamins: Fundamental Roles in Nutrition and Health*. New York: Academic Press.

[5] Food and Nutrition Board, National Research Council. 1989. *Recommended Dietary Allowances*, 10th edition. Washington: National Academy Press.

Questions

1. Thiamin deficiency was observed only in the Chinese community even though all three communities had the same staple (rice) diet. Explain.

2. On packages of enriched rice sold in supermarkets, the rice preparation directions state that the rice should not be washed before cooking. Explain.

3. Severe metabolic acidosis caused by high levels of lactate is often associated with thiamin deficiency. Explain.

4. Provide two plausible explanations for what might cause above normal levels of pyruvate in blood and urine. How can one determine which of the two explanations is correct?

5. Why does thiamin utilization increase among cancer patients?

6. A negative correlation exists between the need for thiamin and the amount of fat in the diet. Explain.

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