

Thiamine, Number One For A Reason

Back in the late nineteenth century, particularly in the Eastern countries like China, Japan, India and the South Pacific region, there was an endemic disease that was the target of much research. Beriberi is a debilitating condition defined by impaired glucose metabolism that leads to complications affecting the cardiovascular, nervous, muscular, and gastrointestinal systems.

No one knew exactly what caused Beriberi. The best theory at the time was that it was caused by a germ of unknown origin, most likely found in contaminated or decayed food. It wasn't until 1884 when a Japanese doctor in the employ of the navy named Takaki Kanehiro made an important observation that Beriberi was particularly common among the lower-ranked crews who were fed a diet that consisted of rice almost exclusively.

In 1883, he learned of a mission that had a severe outbreak of Beriberi on board during a nine-month voyage from Japan to Hawaii where over half the crew had become ill with symptoms of beriberi, particularly edema, emotional disturbances, tingling in the arms and legs, and twenty-five of them had died, mostly from heart failure. In cooperation with the Japanese navy, he conducted an experiment where an identical voyage would be made with all the same destinations, the only difference being the diet. Instead of rice, the crew was fed meat, fish, barely, beans, as well as rice. This time instead of over 150 men out of 375 developing beriberi, only 14 did. This was a major breakthrough since it showed that beriberi was related to the diet rather than the germ theory.

In 1897, the next major breakthrough occurred. A Dutch scientist named Christiaan Eijkman was conducting Beriberi research in Indonesia. At the

time, he was conducting experiments on chickens and out of the blue they began developing paralysis and other symptoms similar to beriberi. The only change was in their feed. Previously, the chickens had been fed leftover rice from military rations, but then the cook refused to allow the rice to be fed to animals. Rice then had to be purchased elsewhere. This new rice was the source of the chickens resulting symptoms. The difference between the old and new rice was that the new rice was polished. When the chickens were returned to the un-polished rice, the symptoms disappeared.

Eijkmann had discovered that beriberi was a *deficiency* and it was an essential nutrient contained in the un-polished portion of whole rice. This observation won him the Nobel Prize for Medicine in 1929, which was shared with Sir Frederick Hopkins because this observation contributed to the discovery of vitamins.

What was that missing nutrient? The essential vitamin that if it had been discovered sooner could have saved the lives of thousands? Thiamine, better known as vitamin B1.

The fact that a deficiency in this vitamin can cause death in as little as nine months means that understanding both dietary sources and proper supplementation levels is crucial to maintaining optimum health. Even though death is an extreme, deficiency in B1 has been proven to compromise nearly every organ system in the body because of vitamin B1's prolific necessity.

Thiamine performs several important functions in the body, the most crucial of which is the metabolizing of carbohydrates in order to provide sufficient energy to the cells of the body. To a lesser degree, it is also involved in the processing of amino acids and fats.

Thiamine is also helps maintain the quality and tone of the smooth muscle in the intestinal tract, which thereby aids digestion, and excretion of wastes.

It's easy to see how a limited diet of polished white rice can lead to all sorts of trouble, mainly a compromised energy production system which means that all the subsequent dependent systems are inhibited from functioning. This is the reason why B1 deficiency is such a problem in developing countries where the primary dietary staple food is rice. But what about those of us that live in the Western countries where we eat whole grains, meat, vegetables, fruits? Such a dietary variety should offer plentiful sources of vitamin B1 and make B1 deficiency practically non-existent right? Right?

As with most things in life, it may look good on paper, but the real world is where it counts and what actually happens is often very different from what is theorized to be true.

The most abundant, but not necessarily the best, source of B1 is from whole unprocessed grains. It is unfortunate that the great majority of the grains in the United States are processed and then the lost nutrients are replaced in a process called "enrichment" or "fortification". If you read the labels on many of the common cereals in the supermarket, it will be a rare occasion when you find one that doesn't contain un-bleached, un-enriched, or un-processed flour and grains. For those of you who eat sushi, Indian, or other Asian foods, how often is brown rice the norm rather than the requested exception?

Assuming that enrichment solves the problem of insufficient B1 from grains, what about protein sources of B1 like meats, poultry and fish? While meats do contain thiamine in various concentrations, B1 is extremely vulnerable to heat. Boiling vegetables,

grilling or frying meats, baking poultry destroy the thiamine inside.

If one were to attempt to acquire all of their necessary thiamine from their diet, it would be important to know which foods contain the greatest and best quality of thiamine in order to incorporate them into their daily meals.

The top five food sources of thiamine, in order of density, are:

- Romaine lettuce
- Asparagus
- Crimini mushrooms
- Spinach
- Sunflower seeds

Other high-ranking foods include members of the legume family, eggplant, summer and winter squash, as well as green leafy vegetables like celery, cabbage, brussels sprouts, and kale.

A commonly held belief is that beriberi is still very rare in the United States and therefore even with these issues, B1 deficiency is a not a major concern for Western populations. One has to remember that beriberi is an *extreme* deficiency. The gas tank of a car doesn't just go from full to empty instantly. It is a gradual process with various degrees of consumption. B1 deficiency is no different. Mild deficiencies are quite common in the US population.

The symptoms of a mild deficiency are also associated with other medical conditions and a diagnosis is sometimes difficult. Through a process of elimination, and where other remedies have been ineffective in relieving symptoms, a B1 deficiency may be the underlying problem.

Mild B1 deficiency symptoms include:

- Fatigue
- Insomnia

- Headaches
- Numbness
- Neuritis, pain and inflammation of nerves
- Aching
- Burning sensations in hands and feet
- Indigestion
- Constipation
- Diarrhea
- Loss of appetite
- Weight-loss
- Weakness

These are just the physical symptoms associated with mild B1 deficiency. There are also a number of documented mental symptoms that accompany deficiency. These include:

- Apathy
- Confusion
- Emotional instability
- Irritability
- Depression
- Fear of impending disaster

These mental symptoms typically manifest after a few days of B1 deprivation. Another interesting result of continued research into B1 deprivation has shown that B1 can reverse the symptoms of neurological impairment such as manual speed and coordination, complex body-reaction time, hand-eye coordination, manual steadiness and body sway.

There is a group in the US who have a higher propensity towards B1 deficiency of greater severity, and that is the chronic alcoholic. Alcohol requires greater amounts of B1 to process, and since an alcoholic more often than not has a poor diet that is greatly lacking in sufficient B1, the body's reserves are quickly depleted. To further compound the problem, the alcohol in the blood blocks the absorption and utilization of whatever B1 they may have in their body, and thus beriberi is not an uncommon condition amongst

alcoholics. In addition to alcohol, smoking and consuming sugar also increase the rate of thiamine depletion in the body.

Because thiamine is water-soluble and not lipid-soluble, there is difficulty in getting sufficient B1 into the brain where deficiency-related neural damage can lead to some of the mental symptoms mentioned earlier, and if left unhandled, could also lead to more serious nervous system complications. In the early 1960s, Japanese scientists developed a substance that navigated around this problem.

Benfotiamine is a lipid-soluble thiamine precursor that can cross the blood-brain barrier and when metabolized by the body becomes thiamine pyrophosphate (the active form of thiamine). It is the most effective B1 metabolic precursor available and has no known toxicity, though there are rare instances of B1 hypersensitivity, which would require a reduced dosage.

Benfotiamine has been used by German medical professional in the successful treatment of a number of nervous systems disorders including multiple sclerosis, and nerve damage and pain conditions like sciatica. Benfotiamine has been proven effective for diabetic retinopathy, neuropathy, and nephropathy when taken in high doses.



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