

BENFOTIAMINE

There is an incredibly effective way to supplement with Vitamin B1. That is with the lipid-soluble Benfotiamine. Truly, you can think of Benfotiamine as “Vitamin B1 on steroids.” It is exponentially more metabolically powerful than ordinary Thiamine.

Humans do not synthesize Thiamine, and neither is it stored significantly in tissues. The Thiamine Diphosphate form (also known as Thiamine Pyrophosphate (TPP)) is the primary active form of the vitamin. TPP is a necessary cofactor for enzymes controlling carbohydrate metabolism, broadly including the intermediates of the Glycolysis energy pathway, the Krebs Cycle energy pathway, the Pentose Phosphate energy pathway, as well as metabolism of many amino acids. (1)

Since TPP is the limiting factor for energy production throughout the body (particularly in the liver, heart, and brain, and is particularly essential in diabetes and all the cardiovascular and neurodegenerative sequela of diabetes), TPP is thus a core requirement for health maintenance and longevity. (2)

Many pathologies, including neurodegenerative diseases, diabetes, and cardiovascular pathology, create extraordinary nutrition needs for this vitamin. Supplementing with TPP is essential for patients as they begin down the road to any of these pathologies --- but please understand --- vitamin supplementation (or any supplementation, as per the Nutri-Spec paradigm of “treat the patient not the disease”) is in no way a “treatment” of these diseases. It is simply the means to supply the extraordinary nutrition needs of individuals suffering certain pathologies. (3)

Poor Vitamin B1 Absorption

The clinical dilemma in supplementation is the limited absorption and assimilation of Vitamin B1 in any of its common supplement forms, Thiamine HCl, Thiamine Monophosphate, or even TPP. Consider a study by C M Tallaksen, et al ...

After an oral dose of 50 mg (!!!) of Thiamine HCl (--- Keep in mind that the Recommended Daily Intake of Vitamin B1 is only 1.1 to 1.4 mg --- So --- that 50 mg is a super-mega dose of more than 40 times the physiological need for Vitamin B1), peak concentration in plasma is reached after 53 minutes, and represents an 80% increase over its presupplementary level. But the elimination half-life is 154 minutes, and only 2.5% of the 50 mg dose is recovered in the urine. The bioavailability of the 50 mg dose is only 5.3%. Those numbers indicate that more than 90% of a 50 mg dose passes right through the intestinal tract without being absorbed. (4)

Thiamine is intestinally absorbed by active transport through the intestinal lining, and that transport mechanism is very quickly saturated. With extreme pharmacological doses of oral Thiamine HCl, a passive absorption pathway comes into play, which requires as much as a 1500 mg dose (nearly 1000 times the Recommended Daily Intake!) to spike the Thiamine plasma level significantly after 4 hours. But, that absorption represents a miniscule percentage of the supplemented dose, and that high amount of B1 absorbed, is not retained well --- falling by nearly 70% over the ensuing 6 hours. (5)

[The maximum absorbable dose of vitamin B1 in any of its common forms is 4 mg. When people supplement with 1 mg of vitamin B1, they generally absorb almost all of it; when supplementing with 2 mg they get most of it; when supplementing with 3 mg they absorb barely more than 2 mg; and when they supplement with 10, 50, or even 100 mg of vitamin B1, the most they can possibly absorb is something less than 4 mg. ----- What does that say about all the silly health food store and "Alternative" doctors selling B complex garbage with as many as 50 to 100 mg of vitamin B1? They are either ignorant of nutrition physiology, or they are being blatantly dishonest, selling a product they know is almost entirely a waste of money.]

To overcome the poor absorption and assimilation of vitamin B1, lipid-soluble Benfotiamine has been developed. Since Thiamine, particularly TPP, is a coenzyme for enzymes essential in all the pathways of cellular energy production, its inadequacy has deleterious effects on the tissues that are the major energy consumers --- particularly the brain, heart, and of course the liver, which directs and integrates energy production from all macronutrient sources. (8)

One research study shows that Benfotiamine absorption is 5 times as high as Thiamine HCl supplementation, and cellular utilization (as shown by cellular transketolase activity in red blood cells) is 3.6 times higher than with Thiamine HCl supplement. Another research study shows that, compared to Thiamine HCl supplementation, Benfotiamine absorption is 11 times as high as the equivalent Thiamine HCl dose, and the level of TPP in red blood cells is 5 times higher. (6,7)

Given that active transport is required for intestinal absorption of Thiamine from our foods, and additional transporters are required for Vitamin B1 to enter cells, the big advantage of Benfotiamine becomes quite obvious. It freely passes from the intestine into the circulation, then on to the liver, and requires no carriers to cross cell membranes throughout the body. Benfotiamine is quickly converted to TPP, and so the beauty of Benfotiamine supplementation is that you will get more TPP from supplementing with Benfotiamine than you do supplementing with TPP itself. (3)

It's All About Metabolic ENERGY

No efficient metabolism of either fats, carbohydrates, or amino acids can exist with a suboptimal Thiamine level.

By facilitating energy production via modulating Glycolysis of glucose, Beta-Oxidation of Fatty Acids, Krebs Cycle energy production, the Pentose Phosphate Pathway of energy production, and the entire Electron Transport Chain producing ATP through oxidative phosphorylation, Thiamine prevents the formation of Reactive Oxygen Species (ROS) in the mitochondria. It thus exerts huge protection against oxidative stress in all body systems. It is critical in defense against Dysaerobic Imbalance and Exogenous INFLAM-AGING.

Metabolic AGING --- Advanced Glycation End-Products

Benfotiamine facilitates the action of TPP the cofactor for the enzyme transketolase. Transketolase enzyme moves the precursors of Advanced Glycation End-Products (AGEs) towards the pentose phosphate pathway in the liver, thereby reducing the production of AGEs and their associated catabolic oxidative damage. Of course, AGEs are the major source of damage and accelerated aging in diabetics, but they contribute to some degree to premature aging in most patients. (3,8)

The reduction of AGEs by Benfotiamine subsequently decreases metabolic stress, which benefits vascular complications seen in diabetes. The many effects of benfotiamine on the AGE-dependent pathway are well-established.

Advanced Glycation Endproducts (AGE) are a well-established complication of both Type I and Type II Diabetes. Even in non-diabetics, glycated hemoglobin (as indicated by the blood analyte HbA1c) is one of the major indications of our Nutri-Spec model of Endogenous INFLAM-AGING. Excess AGE and altered AGE metabolism are typical of Metabolic Syndrome even without diabetes, and is also found in neurodegenerative diseases as well as Metabolic-Associated Fatty Liver Disease (MAFLD). Elevated AGE (as indicated by your patients with elevated HbA1c) is a perfect indication of the need for Vitamin B1 supplementation as Benfotiamine. (9)

Eicosanoid/Prostaglandin INFLAM-AGING

Several studies have shown that Benfotiamine also modulates pathways other than AGE. The anti-inflammatory benefits of Benfotiamine supplementation go beyond the control of inflammatory cytokines and CRP, and extend to the eicosanoid prostaglandin inflammatory pathway. Benfotiamine supplementation reduces the inflammatory markers inducible nitric oxide synthase and cyclooxygenase-2, as well as their products, Nitric Oxide, Prostaglandin E2, Leukotrienes and Thromboxane. (10)

This arachidonic acid pathway we can also call the omega 6 fatty acid pathway, or more appropriately in NUTRI-SPEC terms, the inflammatory prostaglandin pathway. What this means is that Benfotiamine protects against the oxidative (Dysaerobic) free radical damage from consuming polyunsaturated vegetable oils (HOHUM PUFAs --- Heated, Oxidized, Hydrogenated and otherwise Un-Metabolizable Polyunsaturated Fatty Acids).

A Broad Array of Anti-Inflammatory Benefits

Benfotiamine also helps control other major inflammatory metabolites, including NF-k-Beta, protein kinase B, mitogen activated protein kinases, and vascular endothelial growth factor receptor 2 (VEGF2) signaling pathways. (11)

Thiamine is an important cofactor in the immune system, with multiple functions in regulating and controlling function of a broad array of immune cells --- including T-cells, B-cells, Natural Killer cells, and macrophages. In autoimmune inflammatory conditions such as rheumatoid arthritis, Thiamine reduces inflammation and pain by decreasing the proinflammatory cytokines TNF-alpha, Interleukin-1-Beta, Interleukin 6, along with C-reactive protein, while at the same time increasing the anti-inflammatory IL-10. (11)

In summary --- benfotiamine is a major part of your NUTRI-SPEC arsenal to protect your patients from ImmunoNeuroEndocrine Stress and INFLAM-AGING.

Special Needs for Vitamin B1

Some examples of extraordinary need for Vitamin B1 include the increased Thiamine excretion typical of diabetes. Here we have a vicious cycle in which Thiamine deficiency feeds back into exacerbating the causative factors. (2)

Alcohol intake also creates a special need for Thiamine.

There are more than 30 drugs that inhibit Thiamine utilization.

The active transport of Thiamine absorption significantly decreases with age --- and here again is a vicious cycle in which inadequate Thiamine accelerates the aging process, but age inhibits Thiamine absorption. (3)

Metabolic-Associated Fatty Liver Disease demonstrates another vicious cycle. Insufficient Vitamin B1 is created by the pathology, but that deficiency exacerbates the pathological process. (8)

Foods that particularly destroy Thiamine include coffee, tea, certain fish (particularly raw or fermented fish), shellfish, and obviously, alcohol.

Brain Health

Benfotiamine also shows promise in geriatric patients with cognitive decline. It has even been studied in association with Alzheimer's disease.

Studies in subjects with neurodegenerative diseases show deficient uptake and utilization of Vitamin B1 resulting from the pathology. Furthermore, this deficiency found in brain and nerve levels, can exist even in the presence of a normal plasma level, and seemingly adequate dietary intake.

Elevated AGE and their receptor occur in the brain and in the periphery of Alzheimer's patients, and are found in both plaques and tangles. The level of AGE correlates with cognitive function (even in healthy individuals). Yet even in extreme neurodegenerative disease, the level of AGE can be controlled by Thiamine supplementation. (12)

Point of emphasis: We are not suggesting that you "treat" Alzheimer's disease and other neurodegenerative conditions with Benfotiamine (or with any nutrition supplement). However, we strongly suggest you treat individuals with neurodegenerative disease, because they have extraordinary nutrition needs that result from the pathological process.

In the brain and nervous system, not only is Vitamin B1 essential to sustain high brain energy production, it is also involved in the production of neurotransmitters essential for brain and nerve function. Thiamine also plays a role in the transmission of nerve impulses and in the maintenance of the myelin sheath. Aberrant Thiamine metabolism in the brain resulting from neurodegenerative diseases leads to secondary oxidative stress, lactic acidosis, exacerbation of neuroinflammation, and excitotoxicity of brain cells. Inadequate level of TPP is a major factor in neuropathy that occurs secondary to diabetes. (11,12,13)

Autophagy

Autophagy is the cellular catabolic process essential for degradation of cellular waste products plus the renewal and recycling of intracellular proteins. Insufficient Thiamine inhibits the autophagy essential for the maintenance of homeostasis, leading to damaged and dysfunctional cellular organelles and deficiency of the nutrients that should be recycled.

The autophagy deficiency associated with Vitamin B1 parallels the process of Endogenous INFLAM-AGING, the aging pathway driven by metabolic clocks that activate in early adulthood, and accelerate the aging process via mTOR and the development Senescent Cells. Brain/nerve cells are particularly vulnerable to deficient autophagy in the buildup of metabolic waste products. These can result in cognitive decline and motor impairments. (14)

Benefits Beyond B1 Actions

Interestingly, while Benfotiamine protects against Oxidative Stress, and in general confers benefits for the extraordinary nutrition needs created by many pathologies, Benfotiamine yields benefits not shown by supplementation with Thiamine, and which are independent of Thiamine status. For example, when investigating the effect of Benfotiamine and Thiamine on the master regulator of cellular antioxidative response (Nrf2/ARE pathway) researchers find that Benfotiamine and its metabolites activate this pathway, while Thiamine does not. So again, we see that benefits from Benfotiamine are not all exerted through its conversion to Thiamine. (11)

Another example is the production of the Master Anti-Oxidant Glutathione, which increases dramatically with Benfotiamine supplementation, but only minimally by Thiamine supplementation. Research shows that many of the benefits of Benfotiamine occur after the Benfotiamine enters the cell, but before conversion to Thiamine. It is the Benfotiamine intermediates that have the greatest anti-inflammatory, antioxidant, neuroprotective, and hepatoprotective properties.

So, with Benfotiamine supplementation you are getting all the benefits of maximizing Thiamine activity, while at the same time the huge bonus derived from Benfotiamine as an Adaptogen and Rejuvenin before it is converted to TPP. (11)

Early signs of insufficient Thiamine activity are non-specific, and are virtually always overlooked or blamed on some other cause by traditional medical evaluation. These early signs include: (9)

- Fatigue
- Weakness
- Memory problems
- Sleep disturbances
- Lack of appetite --- Anorexia
- Abdominal pain
- Nausea

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