

NUTRI-SPEC



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THE NUTRI-SPEC LETTER

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MAXIMIZE YOUR HEALTH SPAN

Dear Doctor,

Dysaerobic Imbalance defines oxidative free radical damage --- the fullest expression of what we Nutri-Spec practitioners call "Exogenous INFLAM-AGING."

DYSEROBIC = Catabolic Oxidative Stress =

Relentless Tissue Destruction =

Exogenous INFLAM-AGING =

Devastation of Your Health Span.

Every symptom you have ever suffered, and every condition your patients present, is to some degree a manifestation of Dysaerobic/Catabolic stress. Dysaerobic forces begin attacking you from all directions, beginning the moment you are conceived --- and never let up.

LIVE STRONGER LONGER?

Your Health Span --- how well you feel and how many years you feel well --- is largely determined by your capacity to defend against, then rebuild from ---

DYSAEROBIC DESTRUCTION.

In last month's Letter, you learned your newly formulated OXYGENIC D features ---

4 WEAPONS OF MASS CONSTRUCTION ---

assuring you will continuously regenerate tissue structure & function in defense against oxidative stress. We singled out one of those 4, the anti-catabolic amino acid GLUTAMINE. Now, you need to discover the anti-Dysaerobic power OXY D gives you with ...

BENFOTIAMINE.

You can think of Benfotiamine as ---

“VITAMIN B1 ON STEROIDS.”

It is exponentially more powerful than ordinary Thiamine.

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 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, far more efficiently than ordinary Thiamine, decreases the production of Advanced Glycation End-products (AGE) and 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. (1,2)

The protection against 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.

AGE are a devastating complication of both Type I and Type II Diabetes. But 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 are also found in neurodegenerative diseases as well as Metabolic-Associated Fatty Liver Disease (MAFLD). Elevated AGE (as indicated by your patients with rising HbA1c), is a perfect indication of the need for Vitamin B1 supplementation as Benfotiamine. (3)

Eicosanoid/Prostaglandin INFLAM-AGING

Several studies show 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. (4)

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 Poly-Unsaturated 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. (5)

Thiamine is an important cofactor in the immune system, with multiple roles in regulating and controlling the function of many 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. (5)

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

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 Thiamine 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. (7)

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 leads to secondary oxidative stress, lactic acidosis, exacerbation of neuroinflammation, and excitotoxicity of brain cells. An inadequate level of Thiamine is a major factor in neuropathy that occurs secondary to diabetes. (5,7,8)

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 a 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 of Senescent Cells. Brain/nerve cells are particularly

vulnerable to deficient autophagy and the buildup of metabolic waste products. These can result in cognitive decline and motor impairments. (9)

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 (the Nrf2/ARE pathway), researchers find that Benfotiamine and its metabolites activate this pathway, while Thiamine does not. So again, we see that the benefits from Benfotiamine are not all exerted through its conversion to Thiamine. (5)

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 metabolites that have the greatest anti-inflammatory, antioxidant, neuroprotective, and hepatoprotective properties.

So, with Benfotiamine supplementation in Oxy D (--- and Activator) you are getting all the benefits of maximizing Thiamine activity, while at the same time a huge bonus derived from Benfotiamine as an Adaptogen and Rejuvenin, before it is converted to Thiamine. (5)

Poor Absorption of Thiamine

The clinical dilemma in supplementation is the limited intestinal absorption and cellular assimilation of Vitamin B1 in any of its common supplement forms, Thiamine HCl, Thiamine Monophosphate, or even the coenzyme Thiamine Pyrophosphate (TPP).

The maximum absorbable dose of vitamin B1 in any of its 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 stores 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. (3)

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 supplementation. 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 Thiamine in red blood cells is 5 times higher. (6,10)

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 TTP, and so the beauty of Benfotiamine supplementation is that you will get more TPP from supplementing with Benfotiamine than you do from supplementing with Thiamine itself. (1)

Dysaerobic Destruction? Catabolic Oxidative Damage?

Exogenous INFLAM-AGING? Endogenous INFLAM-AGING?

YOUR NEW OXYGENIC D ---

protects you from, and rebuilds tissue structure & function from, all forms of Oxidative Stress.

HEALTH SPAN? LIFE SPAN?

LIVE STRONGER LONGER!

Special for May: 1 **FREE** of Oxygenic D and of Oxy-Max/Diphasic PM for every 6 you buy

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