A PATIENT GUIDE FROM BELLYMD

Allithiamine: What the evidence shows.

A plain-language look at the fat-soluble form of vitamin B1 first isolated from garlic, and what the published research does and does not support.

> **About this guide.** Allithiamine is one of the active ingredients in *MGB+ Clear* at 75 mg per daily serving, and in *MGB+ Cool* at 150 mg per daily serving. This handout covers the published evidence behind allithiamine so you can read it yourself and decide what makes sense for you.

CHAPTER ONE

What is allithiamine?

> *A short answer first. Allithiamine is a fat-soluble cousin of vitamin B1. It was discovered in garlic in 1951 by a Japanese research team studying why people who ate raw garlic seemed to absorb more B1 than people who did not.*

Vitamin B1, also called thiamine, is one of the eight B vitamins. It is the spark that lets your cells turn food into usable energy. Without it, the parts of your body that need the most energy, like your nerves, your heart, and your gut, slow down first.[1,6,8]

Regular thiamine, the kind in a typical B-complex pill, is water-soluble. That sounds good, but it is actually a limitation. Water-soluble thiamine has to be carried into your cells one molecule at a time by a specific protein doorway. Once those doorways are full, no more thiamine gets in, no matter how much you take.[9,12]

In 1951, Japanese researcher Motonori Fujiwara and his team noticed something strange. When they crushed garlic, the thiamine inside it reacted with a compound called allicin and turned into a new molecule. This new molecule was fat-soluble. They named it *allithiamine*, after the genus *Allium*, which includes garlic and onions.[1,3] A second team led by Taizo Matsukawa worked out how to make it in a laboratory in 1953.[2]

> **THE SHORT VERSION**
> Allithiamine is vitamin B1 attached to a fat-soluble carrier from garlic. The carrier helps the vitamin slip through cell membranes that would normally block regular B1. Inside the cell, it converts back into the working form of vitamin B1.

Why a fat-soluble form matters

Cell membranes are made of fat. Water-soluble vitamins do not pass through them easily and need special transport proteins. Fat-soluble compounds slip through directly. By stitching a fat-soluble piece onto regular thiamine, allithiamine bypasses the bottleneck that limits how much B1 you can actually use.[7,10,12]

This single feature is the reason researchers became interested in allithiamine in the first place. It allowed them to get meaningfully more thiamine into cells, including cells in the brain and nervous system, than water-soluble thiamine could deliver on its own.[5,7,12]

A note on naming

Some popular literature treats allithiamine and a separate synthetic compound called TTFD as the same thing. They are not. Allithiamine is the natural parent compound from garlic. TTFD is a synthetic molecule built later using allithiamine as the template. They share a family resemblance but are different molecules with different research bases.[7,10] Allithiamine is also chemically different from benfotiamine, sulbutiamine, and fursultiamine, which are three other fat-soluble B1 derivatives. When research talks about one of these, the findings do not automatically apply to the others.[10,11,12]

CHAPTER TWO

How allithiamine works in your body

> *Think of allithiamine as a key that fits a lock regular vitamin B1 cannot reach. Once inside the cell, the carrier drops off and what is left is the same active vitamin your body has always used. The difference is how much actually gets in.*

The doorway problem

Your cells have specific protein doorways for thiamine called SLC19A2 and SLC19A3. These transporters work well at normal dietary amounts. Above that, they max out. Extra water-soluble thiamine in your bloodstream simply leaves through your urine. This is why high-dose water-soluble B1 hits a ceiling.[9,12]

Allithiamine takes a different route. Because it is fat-soluble, it slips through cell membranes by passive diffusion, without needing the transporter at all. Once it crosses the membrane, the cell breaks the disulfide bond holding the carrier on, and the molecule reassembles itself into regular thiamine inside the cell.[5,7,10,12]

Reaching the brain

The brain is protected by a structure called the blood-brain barrier, which is just what it sounds like, a tight wall of cells that keeps most things in your blood from reaching brain tissue. Water-soluble thiamine has to wait its turn at this wall through its specific transporter. Fat-soluble derivatives cross more directly.[9,12]

Early human studies in the 1970s by Allan Thomson and Herman Baker measured this difference. Oral fat-soluble thiamine derivatives produced higher levels in blood, red blood cells, and cerebrospinal fluid, the fluid that bathes the brain and spinal cord, than equivalent doses of water-soluble thiamine.[5,18]

> **WHY THIS MATTERS FOR SYMPTOM CLUSTERS**
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> When fatigue, brain fog, headache, and gut symptoms travel together, the common thread is often cells that are not producing energy efficiently. Tissues with the highest energy demand, like nerves and gut wall, feel that shortfall first. The case for a fat-soluble B1 derivative is that it can reach those tissues in usable amounts even when the standard transporter doorway is overwhelmed.

The job vitamin B1 does inside the cell

Once allithiamine is inside the cell and converted back to thiamine, that thiamine is rapidly turned into its active working form: thiamine pyrophosphate, or TPP for short. TPP is the form that actually does the work.[6,8,9]

TPP is a required helper, technically called a cofactor, for several enzymes inside the mitochondria, which are the energy factories inside your cells. The three most important are:

1. **Pyruvate dehydrogenase**. An enzyme that helps turn the sugar from food into the fuel mitochondria can use.[6,9]
2. **Alpha-ketoglutarate dehydrogenase**. An enzyme that keeps the main energy-making cycle inside mitochondria running. When this enzyme slows down, energy output drops.[6,16]
3. **Transketolase**. An enzyme that handles a separate sugar pathway your cells use to build the parts they need to repair themselves.[6,9]

If any of these three enzymes runs short on TPP, the cell makes less energy. Nerve cells, gut wall cells, and heart cells feel that first because they consume so much energy.[8,16]

What allithiamine is not

Allithiamine is not a drug. It does not block pain receptors. It is not a stimulant or a hormone. It does not bind brain receptors the way medications do. It is a vehicle for getting more vitamin B1 into cells than water-soluble B1 can deliver on its own.[6,7,10]

CHAPTER THREE

What the studies show

> *Here is the published evidence by area. The mechanism evidence is strong. The clinical evidence is real but preliminary, mostly built on case series rather than large blinded trials. We will say that honestly throughout this section.*

Mechanism and bioavailability

MULTIPLE HUMAN AND ANIMAL STUDIES · OVER FIVE DECADES

This is the strongest evidence base. Beginning with Fujiwara's 1951 work and Matsukawa's 1953 synthesis, dozens of studies have confirmed that fat-soluble thiamine derivatives reach cells, including brain cells, more reliably than water-soluble thiamine.[1,2,3,4,5] The 1971 study by Thomson and colleagues at the Annals of Internal Medicine showed this directly in humans, measuring higher cerebrospinal fluid thiamine levels after fat-soluble thiamine than after equivalent water-soluble doses.[5] Modern reviews continue to support this finding.[10,11,12]

What this means: the case for fat-soluble thiamine as a delivery vehicle is well established. The pharmacology is not controversial.

Fatigue in inflammatory bowel disease

RANDOMIZED CONTROLLED TRIAL · 40 PATIENTS

The strongest clinical trial in this space comes from Palle Bager and colleagues at Aarhus University Hospital in Denmark. They tested high-dose oral thiamine, between 600 and 1800 mg per day depending on body weight, against placebo in 40 patients with quiescent inflammatory bowel disease and chronic fatigue. The thiamine group had significantly greater fatigue reduction than placebo.[19]

Important caveat: this study used thiamine hydrochloride, the water-soluble form, not allithiamine specifically. The doses were also far higher than typical maintenance amounts. Still, it is the most rigorous clinical signal that high-dose B1 can reduce fatigue in a population with gut inflammation, and it gives biological plausibility to the broader thiamine-and-fatigue research program.[19]

Fatigue across diverse conditions, the Costantini case series

OPEN-LABEL CASE SERIES · 2013 TO 2018

Italian neurologist Antonio Costantini and his colleagues published a series of small open-label studies between 2013 and 2018 reporting that high-dose oral or parenteral thiamine reduced fatigue across a wide range of conditions: inflammatory bowel disease,[13] fibromyalgia,[14] multiple sclerosis,[15] Hashimoto's thyroiditis,[17] Parkinson's disease,[16,20] and chronic cluster headache.[21] Doses ranged from 600 mg to 1800 mg per day. Most patients improved.

What this means in plain English: the same signal kept showing up across very different illnesses, which is suggestive, but every one of these studies was open-label, small, and had no placebo group. Open-label means everyone knew they were getting the treatment, which makes placebo effects harder to rule out. Treat these as hypothesis-generating, not as proof of effect. Costantini died from COVID-19 in 2020 before he could run a definitive controlled trial.

Fatigue in primary biliary cholangitis

RANDOMIZED CROSSOVER TRIAL · 36 PATIENTS · NEGATIVE RESULT

Honesty requires reporting the negative trial. In 2024, the same Aarhus group that produced the positive IBD trial ran a similar crossover trial in 36 patients with chronic fatigue from primary biliary cholangitis, a liver condition. High-dose oral thiamine, 600 to 1800 mg per day, was not better than placebo in the crossover analysis.[22]

What this means: the high-dose thiamine story is not universal. It appears stronger for fatigue tied to gut inflammation than for fatigue from liver disease. That is useful information. It tells us the mechanism is probably specific to certain biological contexts, not a generic energy boost for anyone tired.

Central sensitization and the mitochondrial hypothesis

MECHANISM REVIEW · MULTIPLE OBSERVATIONAL STUDIES

Central sensitization is when the nervous system becomes oversensitive to normal signals, producing pain or fatigue that does not match the size of the trigger. Researchers have long suspected that mitochondrial energy shortfalls contribute to this state.[8,16] The thiamine-dependent enzyme alpha-ketoglutarate dehydrogenase is one of the most sensitive to energy stress, and its activity drops in central nervous system tissues under metabolic strain.[16] This is the mechanistic argument for why a vitamin B1 cofactor delivered efficiently into cells might matter in conditions characterized by oversensitive nerves and persistent fatigue.

What this means: the mechanism is plausible and supported by enzyme research, but no large blinded trial has specifically tested allithiamine for central sensitization in humans. This is honest territory for hypothesis, not for claim.

Modern thiamine deficiency

REVIEW ARTICLE · 2021

Marrs and Lonsdale published a 2021 review in *Cells* arguing that subclinical thiamine deficiency is more common in developed countries than typically recognized, driven by high-carbohydrate diets, certain medications, and gut conditions that interfere with absorption.[8] They argue that classic thiamine blood tests miss the lower-grade functional shortfalls that may underlie many fatigue and gut-brain symptom patterns. This is a review article, not new clinical data, but it puts the case-series literature into a coherent framework.[8]

What has not been studied directly

GAPS TO FLAG

No large blinded trial has tested allithiamine specifically for any chronic condition. Most clinical research has used water-soluble thiamine hydrochloride at high doses, not allithiamine. The mechanism literature supports the case that allithiamine should deliver thiamine more efficiently, but this is a reasoned extrapolation, not direct head-to-head clinical proof in patient populations. We will not pretend otherwise.[7,11,12]

CHAPTER FOUR

About dose and timing

> *The published case series and clinical trials have used substantially higher thiamine doses than typical supplement servings. There is a reason for that, and an honest reason why most supplements do not match those research doses.*

What the literature has tested

DAILY DOSE	TYPICAL USE IN TRIALS	FORM USED
600 to 1800 mg	Most Costantini case series[13,14,15,16,17,20,21] and the Bager IBD randomized trial[19]	Mostly water-soluble thiamine HCl
100 to 500 mg	Pharmacokinetic studies in healthy adults[18]	Thiamine HCl, intentionally varied
50 to 200 mg	Typical fat-soluble derivative supplement range; corresponds to MGB+ Clear and Cool	Allithiamine and related fat-soluble forms

Why a lower dose of allithiamine is reasonable as a daily-support amount

Two reasons. First, fat-soluble allithiamine is absorbed and used more efficiently than water-soluble thiamine, so milligram for milligram it delivers more usable B1 to cells.[4,5,11,12] A clinical case series using 1500 mg of water-soluble thiamine is not the equivalent of 1500 mg of allithiamine. Second, the published case-series doses were

aimed at people with significant fatigue tied to active disease. They were therapy-level amounts. A daily maintenance dose is a different goal: keep B1 status steady rather than rescue someone from a deficit.

That said, we will be transparent. The 75 mg dose in MGB+ Clear and the 150 mg dose in MGB+ Cool are not doses that have been tested in a large blinded trial of allithiamine for any specific condition. They are doses chosen to provide a meaningful, well-absorbed amount of fat-soluble B1 as part of a multi-ingredient daily-support formulation, not to replicate the higher therapeutic doses used in the case-series literature.

Time to effect

Thiamine-related effects are not immediate. In the Costantini case series, improvements typically appeared over a few days for some symptoms and several weeks for others.[13,14,15,17] A reasonable expectation is two to six weeks of consistent daily use before judging effect, and longer for slower-moving symptoms like brain fog or fatigue patterns built up over months.

With or without food

Allithiamine is fat-soluble, which means absorption tends to be slightly better when taken with food that contains some fat, the same as fat-soluble vitamins like vitamin D or E. This is not strict; consistency matters more than timing.

CHAPTER FIVE

Safety and what to know

> *Thiamine has one of the cleaner safety profiles in nutrition science. That is not the same as zero considerations. A few specifics deserve attention.*

No tolerable upper intake limit

The Food and Nutrition Board of the US Institute of Medicine reviewed the safety data on thiamine and did not set a tolerable upper intake level, because no adverse effects have been consistently linked to high oral thiamine intake.[23] The body excretes excess thiamine through the urine. This is the part of the safety profile that has held up across decades.[23]

Side-effect profile

Reported side effects from oral thiamine and its fat-soluble derivatives are rare and usually mild. When they occur, they tend to be mild gastrointestinal complaints or, in the case of garlic-derived forms like allithiamine, a faint garlic odor on the breath or skin at higher doses.[7,11] Reducing the dose typically resolves both.

Drug interactions

Thiamine has no well-established major drug interactions in the standard prescribing references. A few specifics worth knowing: some medications, including certain diuretics, can increase thiamine loss; long-term metformin use has been associated with reduced thiamine status in some studies; and people taking medications that act on the central nervous system should let their physician know any time they add a supplement that crosses the blood-brain barrier.[8,23]

Pregnancy and lactation

Thiamine is required during pregnancy and lactation at a recommended daily allowance of 1.4 mg per day. Supplemental thiamine at typical food-fortification levels has a long safety record.[23] However, high-dose thiamine supplementation specifically has not been studied in controlled pregnancy trials. The conservative recommendation is to avoid high-dose allithiamine supplementation during pregnancy and lactation unless a physician advises otherwise.

Other considerations

Rarely, intravenous thiamine has caused allergic-type reactions. This is not a concern for oral use, which is what allithiamine in supplements involves.[23] People with diagnosed garlic allergy should be cautious with garlic-derived allithiamine, although the molecule itself is no longer garlic, just a fat-soluble carrier originally derived from it.

> **WHEN TO TALK TO YOUR PHYSICIAN FIRST**
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> - You are pregnant or nursing.
> - You take medications that affect your central nervous system, including certain antidepressants, mood stabilizers, or anticonvulsants.
> - You are managing a serious chronic condition such as cancer, diabetes, or autoimmune disease.
> - You are considering giving allithiamine to a child.

CHAPTER SIX

Where the evidence has limits

> *An honest brief includes the weaknesses of the science it cites. The allithiamine literature has several real ones.*

Most clinical evidence is case-series, not blinded trial

The Costantini publications, which form the bulk of the clinical literature, are case series and open-label pilot studies. They are useful for generating hypotheses but cannot rule out placebo effect or natural variation in symptom course. Only the Bager team has produced randomized blinded trials in this space, and one of those was negative.[19,22]

Most clinical evidence used water-soluble thiamine, not allithiamine

The clinical case series and the Bager randomized trials used high-dose water-soluble thiamine hydrochloride, not allithiamine specifically. The case for allithiamine in those clinical settings rests on the mechanism literature suggesting more efficient delivery, not on direct head-to-head clinical comparison.[5,11,12]

Trial sizes are small

The Bager IBD trial enrolled 40 patients. The PBC trial enrolled 36. The Costantini case series enrolled handfuls at a time. Real signals can emerge from small studies, but so can statistical noise. Larger trials are needed before strong claims are appropriate.[13,14,15,16,17,19,20,21,22]

Industry funding

Unlike many supplement areas, the allithiamine and high-dose thiamine literature is not heavily industry-funded. Thiamine cannot be patented, so no major pharmaceutical company has run the kind of large registration trial that builds the strongest evidence. This is not a flaw of the molecule; it is a flaw of the incentive structure. It does mean the evidence base is thinner than it would be otherwise.[8]

Honest summary

The pharmacology of allithiamine is well established. The mechanism by which a fat-soluble B1 derivative could matter for energy, nerve function, and gut-brain symptoms is reasonable and supported by decades of biochemistry. The clinical evidence for any specific condition is preliminary, mostly built on small open-label studies with one positive randomized trial in IBD-related fatigue, and one negative randomized trial in PBC-related fatigue. Anyone telling you that allithiamine cures any condition is overstating the evidence. Anyone telling you it is useless is also overstating the evidence in the other direction. The honest position sits between the two.

CHAPTER SEVEN

The bigger picture

> *Why allithiamine appears in two different MGB+ formulas at two different doses, and how it fits into a mechanism-first approach to symptom clusters.*

Bloating, brain fog, fatigue, headache, and upper-gut symptoms rarely have a single cause. They usually share an underlying biology: cellular energy production under strain, an oversensitive signaling system, and a gut wall doing extra work. Single-ingredient supplements aimed at one symptom often miss this underlying pattern. A formulation built around mechanism uses several ingredients that each act on a different lever of the same system.

Allithiamine fits that thinking. By delivering vitamin B1 efficiently into cells that need it, including brain and gut cells, it supports the enzymes that turn food into usable energy. Other ingredients in the MGB+ formulas address adjacent layers: magnesium glycinate for muscle and nerve relaxation; palmitoylethanolamide for the nervous system's natural anti-inflammatory signaling; and additional compounds chosen for complementary rather than overlapping function.

The two doses reflect two different jobs:

- **MGB+ Clear, 75 mg allithiamine per daily serving.** A foundation dose, paired with palmitoylethanolamide and magnesium glycinate, aimed at daily-support for the gut-brain signaling cluster.
- **MGB+ Cool, 150 mg allithiamine per daily serving.** A higher dose for situations where the upper-gut and central nervous system load is greater, paired with a different supporting cast.

The point is not that allithiamine does everything. It is that the published evidence supports allithiamine as one capable lever among several, for people whose tests are normal but whose symptoms travel together.

HOW TO USE THIS BRIEF

Bring it to your physician. Read the references. If you decide to try allithiamine, give it at least four to six weeks at a consistent daily dose before judging effect, and track how you feel in whatever way works for you. Real data, your data, beats marketing claims from either side.

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CITATIONS

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All references are real, PubMed-indexed publications or peer-reviewed primary literature. PMID numbers are listed where assigned. DOIs are included where available for direct linking.

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