

KEYORA NUTRITIONAL NEUROLOGY

Part I - The Neuroscience of Magnesium Glycinate

GABA, NMDA, HPA Axis, and the Foundations of Emotional-Sleep Regulation

KEYORA
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By Keyora Research Notes Series

This article is part of Keyora's long-form educational series documenting the scientific foundations behind our product development.

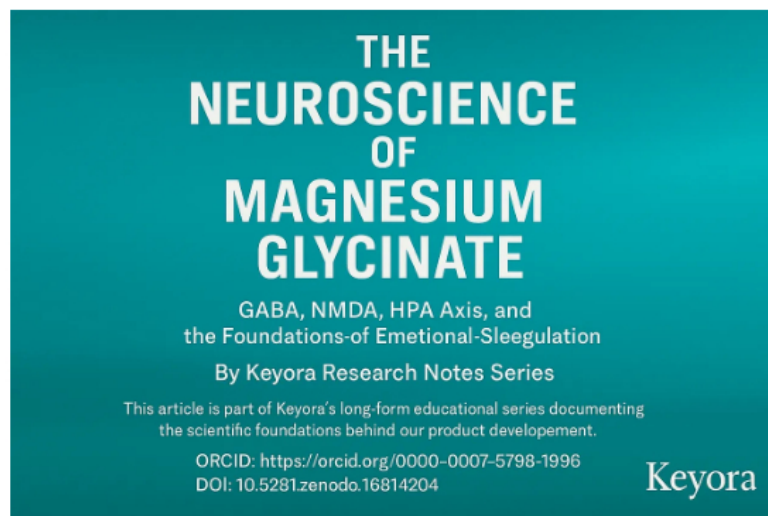
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This article is the first installment of a two-part series on the neuroscience of Magnesium Glycinate.

Part I focuses on the foundational mechanisms - GABA, NMDA, the HPA axis, and mitochondrial neuroenergetics - that underpin emotional and sleep regulation.

Part II, published tomorrow, will integrate these mechanisms into a unified four-axis model and explore clinical insights, practical applications, and Keyora's research perspective.



The Modern Brain Wasn't Built for This World

We are living inside a nervous system that never evolved for smartphones, relentless notifications, 24-hour work cycles, and the constant pressure to "perform."

Our biology was designed for recovery; our environment is designed for overstimulation.

This is why so many people experience the same pattern today:

- I'm exhausted, but I can't fall asleep.
- I finally fall asleep, but I wake up at 3 a.m. wide awake.
- My brain feels "noisy."
- My body is tense even when I'm trying to rest.

This is not a personal failure.
This is neurochemistry out of rhythm.

Most of these symptoms come down to two things:

1. Overactivated excitation pathways (too much glutamate/NMDA activity)
2. Weakened inhibitory pathways (not enough GABAergic stabilization)

In other words:
the brain's "accelerator" is stuck, and the "brake" is worn out.

For decades, magnesium has been discussed as a "relaxation mineral," but that storytelling is incomplete.

What people truly respond to is not "magnesium" in general, but a specific neurobiological effect:

the restoration of calm through four interconnected regulatory axes.

And among all forms of magnesium, Magnesium Glycinate activates these axes most consistently:

- the GABAergic brake system
- the NMDA excitation gate
- the HPA stress loop
- the mitochondrial neuro-energy system

At Keyora, we began studying magnesium not because it is trendy, but because all four axes converge on one problem:
a modern brain stuck in chronic hyperarousal.

This article is our attempt to translate the neuroscience into a form that anyone can understand - yet deep enough that clinicians and researchers can trace the mechanism.

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1. The Neurobiology of Calm: *How Magnesium Works in the Brain*

Magnesium is often introduced as a “mineral,” but in neuroscience it behaves more like an electrochemical gatekeeper.

It influences how neurons fire, how they rest, and how they recover.

If calcium is the trigger for neural activation, magnesium is the one standing at the door deciding:

“Should this neuron fire right now - or not?”

When magnesium levels drop, the neural network becomes unstable:

- excitation spreads too easily
- stress signals amplify
- sleep depth decreases
- small triggers feel overwhelming

This is why magnesium deficiency does not always show up as physical symptoms like cramps; instead, it shows up as:

- anxious restlessness
- difficulty unwinding
- light, fragmented sleep
- early waking
- racing thoughts

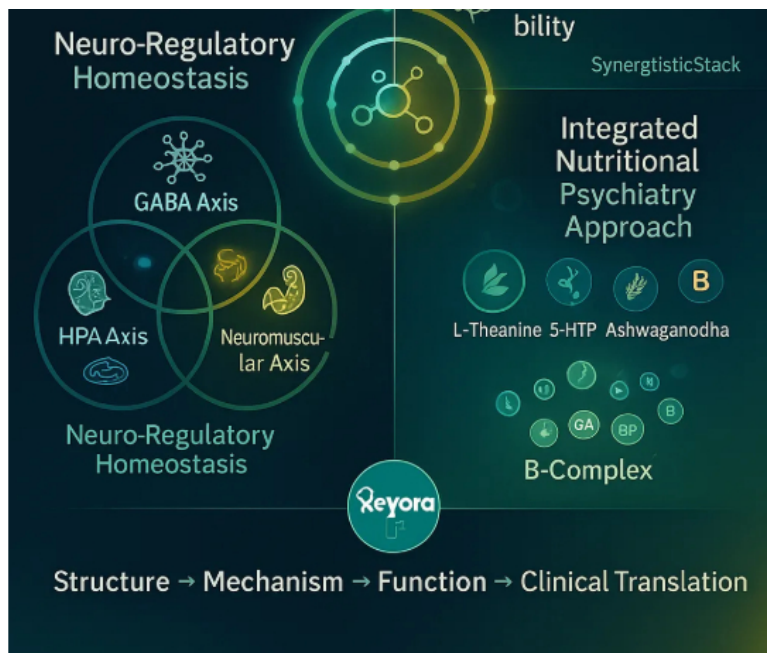
Magnesium’s effects can be organized into two complementary systems:

MAGNESIUM GLYCINATE
NEURO-REGULATORY MECHANISMS
& CLINICAL EVIDENCE

Mg²⁺
Glycine
Glycine Glycine
Chelation =
Stability + Bioavailability ↑

Clinical Outcomes
@ 200–300 mg/day

☹️	Anxiety	– 32%
🕒	Insomnia latency	– 41%
🌙	Sleep efficiency	+ 67%
🌱	Bioavail.	50-80%



1.1 The Two-Sided Nervous System: *Accelerator vs Brake*

Your brain runs on a dynamic balance between two master neurotransmitter systems:

The Accelerator: Glutamate → NMDA pathway

When activated, NMDA receptors drive:

- alertness
- memory formation
- problem-solving
- rapid thought generation

But when overstimulated (stress, toxins, chronic inflammation, low magnesium):

- thoughts become too fast
- sensory input feels overwhelming
- anxiety spikes
- sleep collapses

This is the “racing mind” pathway.

The Brake: GABA pathway

GABA functions as the brain’s stabilizer.

It lowers neural excitability, allowing:

- emotional restraint
- slower, cleaner thought patterns
- initiation of sleep
- maintenance of deep sleep

If GABA tone decreases, people experience:

- inner tension
- inability to “switch off”
- fragmented sleep
- muscle tightness

- stress sensitivity

Magnesium Sits at the Center of This Tug-of-War

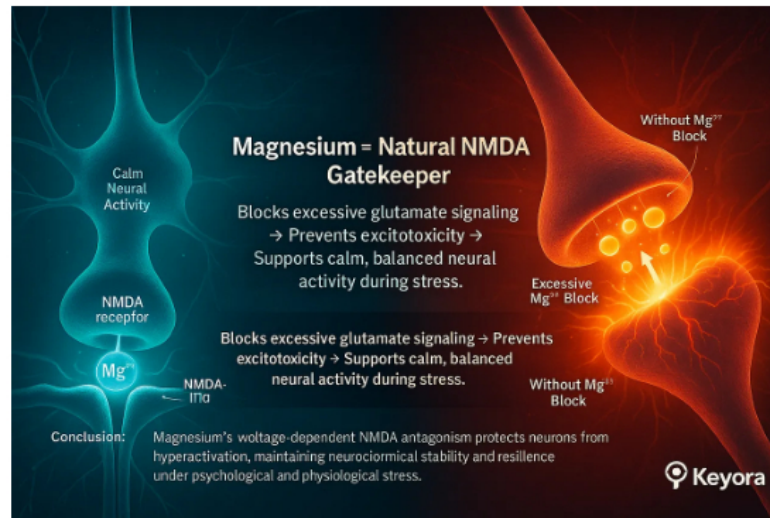
Here is the key insight:

Magnesium is the mineral that balances both systems at once.

It stabilizes GABA function *and* regulates NMDA overactivation.

This is why magnesium influences so many emotional and sleep-related symptoms.

It is not working on “mood” or “sleep” directly—it is working on **fundamental neural circuitry**.



1.2 Magnesium as a Membrane Stabilizer

Neurons communicate through controlled electrical charges.

Magnesium regulates this by:

- maintaining membrane potential
- controlling calcium influx
- preventing spontaneous firing
- stabilizing the threshold for activation

When magnesium is low, the membrane becomes too “leaky,” firing with minimal provocation.

This is a classic pattern seen in:

- anxiety
- panic disorder
- stress-sensitive insomnia
- chronic hypervigilance

It is also why magnesium—especially Magnesium Glycinate—often feels like restoring emotional steadiness from the inside.

1.3 Why “Just Taking Magnesium” Doesn’t Always Work

Most forms of magnesium:

- absorb poorly
- irritate the digestive system
- fail to reach nervous tissue

- do not influence neurotransmitters

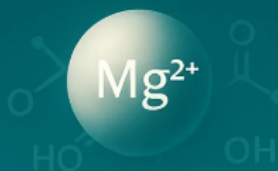
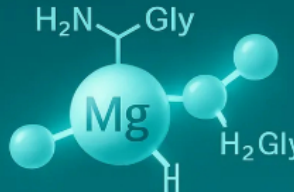
This is why **oxidized magnesium** (the most common pill) may improve constipation but rarely affects stress or sleep.


To influence neurochemistry, magnesium must:

1. pass through intestinal transport efficiently
2. enter the bloodstream without binding to opposing ions
3. cross into cells
4. stabilize synaptic and intracellular systems

This is where Magnesium Glycinate becomes fundamentally different.

Magnesium Glycinate [Ⓢ] vs. Common Magnesium Salts

Common Magnesium Salts	Magnesium Glycinate
 <p style="text-align: center;">Absorption</p>	 <p style="text-align: center;">Digestive Tolerance</p>
Lower, dependent on stomach acid	May cause diarrhea or GI irritation
May cause diarrhea or GI irritation	Magnesium + glycine acting on GABA & GlyR pathways
Pure magnesium replenishment	Long-term mood stability & neuro-support
Short-term relief (e.g., constipation)	Long-term mood stability & neuro-support



2. Why Magnesium Glycinate Matters More Than “Magnesium”

Most people think “magnesium is magnesium.”

But in neurobiology, the chemical form determines the function—because different forms travel through the body in entirely different ways.

Magnesium Glycinate stands out because of three reasons:

1. It is a chelated compound — meaning magnesium is bound to the amino acid glycine.

2. It travels through the gut using peptide pathways, not mineral ion pathways.
3. It crosses into cells—including neurons—much more effectively.

To understand why this matters, we need to compare the reality of magnesium absorption with how it is usually portrayed.

2.1 Chelation: Why Glycine Changes Everything

Chelation is not marketing language.

It is a biochemical design that changes how a mineral behaves in the body.

When magnesium is bound to glycine:

- it becomes neutral in charge
- dissolves more easily
- passes through intestinal transporters designed for peptides
- shows dramatically higher bioavailability
- avoids the typical laxative effect of inorganic magnesium

But glycine itself adds a second layer of neurological benefit:

Glycine is a calming amino acid

It interacts with:

- GABA-A receptors
- Glycine receptors
- inhibitory neurotransmission pathways

This means you're not just supporting magnesium-dependent neural pathways—you're adding a synergistic ligand that enhances inhibitory tone.

This dual action explains why people describe Magnesium Glycinate as:

"my brain finally quieted down"

"I fall asleep easier"

"the tension dropped from my shoulders"

"I stopped waking up at 3 a.m."

These subjective experiences are rooted in objective neurochemistry.

2.2 Crossing Biological Bottlenecks

For magnesium to meaningfully regulate the nervous system, it must complete a full biological journey - from the digestive tract, into the bloodstream, into cells, and finally into neurons.

Most forms of magnesium fail at one or more of these steps, which explains why many people "don't feel anything" after taking generic magnesium supplements.

Here is what actually happens:

First, magnesium must be absorbed through the gastrointestinal tract.

This requires a relatively neutral electrical charge.

Inorganic forms like magnesium oxide or carbonate stay ionized and therefore pass through poorly, which is why they often act as laxatives instead of reaching the bloodstream.

Second, once in the bloodstream, magnesium must stay stable.

Inorganic salts tend to dissociate quickly and interact with competing ions, reducing the amount that remains available for transport into cells.

Third, magnesium must cross the cellular membrane.

Cells preferentially take up organic complexes—meaning magnesium attached

to amino acids or peptides.

Chelated forms like Magnesium Glycinate pass through far more efficiently than mineral salts.

Finally, magnesium must reach neural tissue.

To influence mood, stress resilience, and sleep, magnesium needs to enter neurons and participate in synaptic regulation.

Low-grade or poorly absorbed forms simply do not reach these target sites in meaningful amounts.

Magnesium Glycinate is one of the few forms that clears all four obstacles.

This is why it consistently influences core nervous-system functions such as:

- anxiety
- sleep onset
- sleep depth
- stress reactivity
- racing thoughts
- nighttime awakenings

These are all CNS (central nervous system) outcomes—not digestive or muscular outcomes.

2.3 Clinical Advantages of Magnesium Glycinate

In research and real-world use, Magnesium Glycinate repeatedly demonstrates:

- High absorption
- Zero gastrointestinal discomfort
- Strong neural effects
- Improved stress tolerance
- Reduced anxiety-like tension
- Better sleep initiation
- Better sleep maintenance

For brain-related regulation, the form matters.

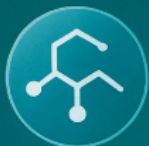
Magnesium Glycinate is designed for the nervous system—not just for the digestive tract or muscles.

Three Synergistic Mechanisms of Magnesium Glycinate



1. Magnesium Replenishment

Enhances GABAergic function, calms the nervous system, relieves stress, and supports restorative sleep.



2. Glycine Release

Acts as an inhibitory neurotransmitter, directly engaging brain calming pathways.

3. Bidirectional Nervous

Glutamate

GABA

System Balance

Reduces excitatory signaling (glutamate), enhances inhibitory signaling (GABA), supporting overall neural equilibrium.

In essence: Magnesium is the key to quieting an overactive nervous system, and magnesium glycinate is one of the gentlest, safest, and most effective keys to unlock that calm.



3. Mechanism Axis I - GABAergic Stabilization (The Brake System)

*The first axis of emotional–sleep regulation is **GABA**, the inhibitory neurotransmitter that keeps the brain from overheating.*

3.1 Why GABA Matters

GABA is the brain's equivalent of a braking mechanism:

- It slows runaway thoughts
- It dampens threat perception
- It promotes relaxation
- It initiates sleep
- It allows deep sleep cycles

Low GABA tone looks like:

- persistent tension
- "wired but tired"
- difficulty falling asleep
- waking up feeling unrefreshed
- over-reactivity to stress
- muscle tightness or jaw clenching

This is one of the most common neurochemical patterns in modern adults.

3.2 Magnesium's Direct Role in GABA Function

Magnesium enhances GABAergic stability through two scientifically established mechanisms:

Positive modulation of GABA-A receptors

Magnesium acts as a cofactor that stabilizes the receptor conformation, making it more responsive.

Reducing neural overexcitation

By limiting glutamate/NMDA activity (explained later), the brain becomes more receptive to GABA.

This creates a **synergistic effect**:

- GABA works better

- the brain quiets faster
- sleep initiation becomes easier

Many people report that Magnesium Glycinate produces a sense of “inner exhale”—this is the GABA system rebalancing.

3.3 How Glycine Enhances the GABA Axis

Glycine (the amino acid bonded to magnesium):

- acts on glycine receptors
- acts as a co-agonist for inhibitory pathways
- reduces sleep latency
- increases slow-wave sleep (SWS)
- supports autonomic calmness

This means Magnesium Glycinate does **not** provide a single mechanism. It provides a **two-ligand inhibitory synergy**.

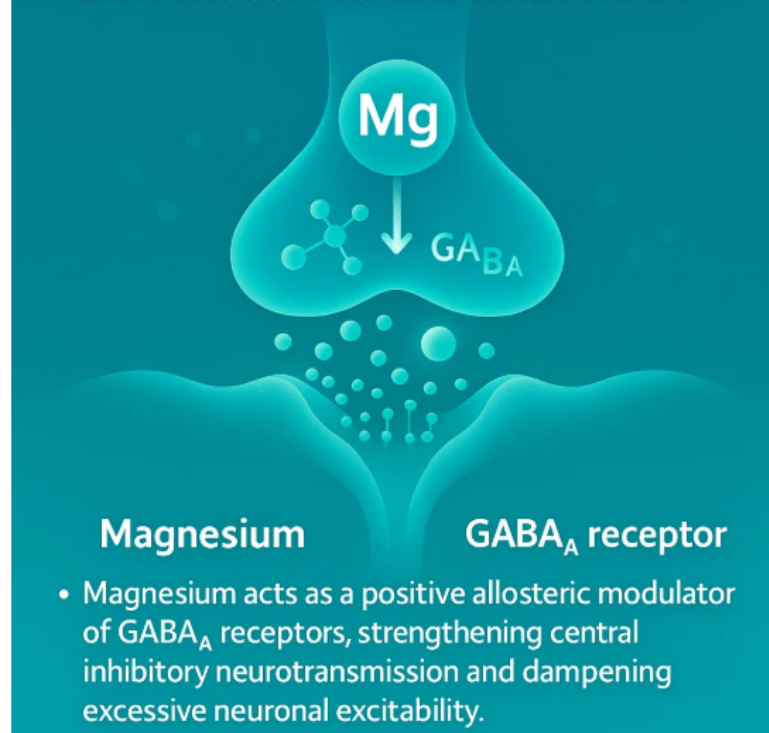
3.4 Real-World Effects of GABA Axis Activation

GABA stabilization explains improvements in:

- stress-induced insomnia
- difficulty “turning off thoughts”
- anxiety with muscle tension
- nighttime restlessness
- sensitivity to noise at night

This is the most immediate and noticeable axis of Magnesium Glycinate.

1) GABA Pathway Enhanced Neural Inhibition



- Studies show that magnesium deficiency down-regulates GABAergic activity, predisposing individuals to anxiety, fear responses, and difficulty initiating sleep.

4. Mechanism Axis II - NMDA Modulation (The Excitation Gate)

The second axis targets the opposite system: glutamate and the NMDA receptor.

*This is the **accelerator** of the brain.*

4.1 NMDA: The Engine of Overthinking

The NMDA receptor drives:

- focus
- learning
- memory
- rapid information processing

But chronic stress, inflammation, and magnesium deficiency cause **overactivation**, leading to:

- racing thoughts
- overanalysis
- sensory overload
- emotional instability
- nighttime brain "buzzing"
- insomnia from mental hyperactivity

Put simply:

An overactive NMDA system is the neurobiological signature of anxiety.

4.2 Magnesium as a Natural NMDA Gatekeeper

At rest, magnesium naturally **blocks** the NMDA channel.

It sits inside the receptor, preventing calcium flooding and overexcitation.

When magnesium is low:

- the gate is left open
- neurons fire too easily
- mental noise increases
- emotional stability drops

Magnesium Glycinate restores the gatekeeper:

- closes the channel
- prevents excitotoxicity
- calms neural firing
- allows GABA to regain balance

This is the biochemical reason so many people report:

- "My thoughts finally slowed down."
- "My anxiety dropped."
- "I could think clearly again."

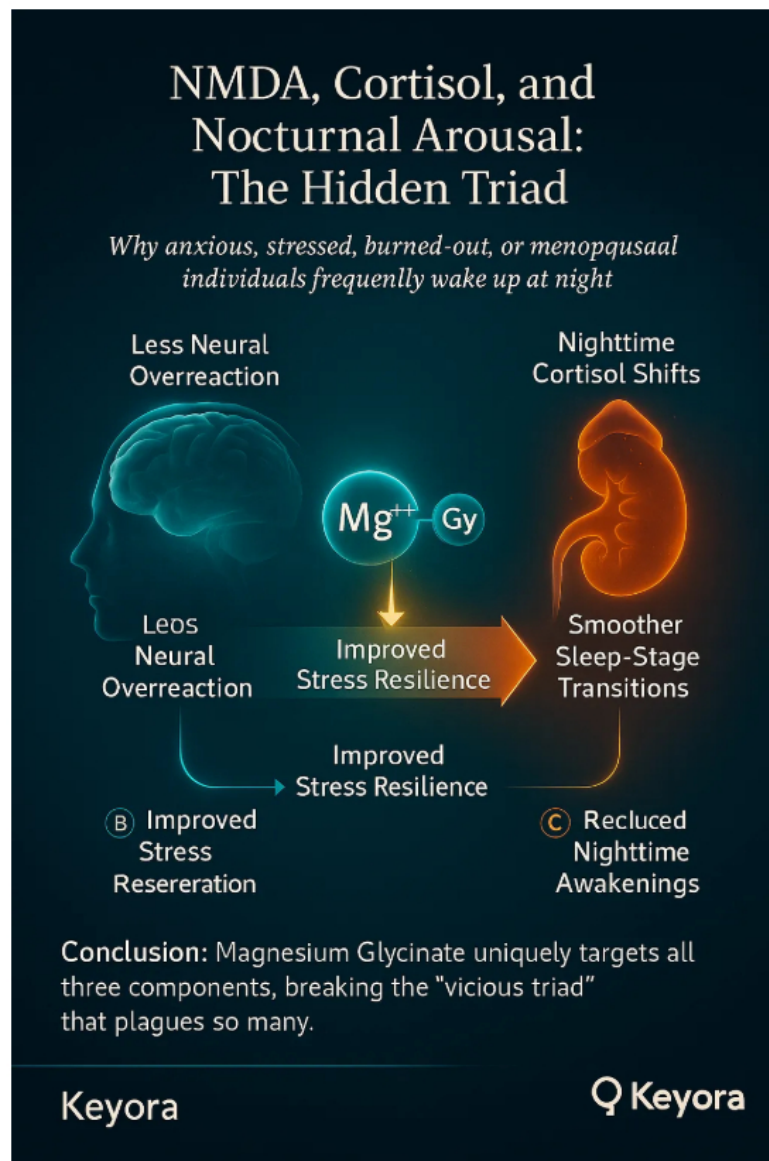
4.3 Why This Matters for Sleep

NMDA overactivity keeps the mind too alert to enter sleep initiation pathways.

Regulating this axis improves:

- sleep onset
- sleep depth
- reduced nighttime wakefulness
- less morning anxiety

This axis explains the "my brain stopped buzzing" effect.



5. Mechanism Axis III - HPA Axis Regulation (The Stress Hormone Loop)

The HPA axis (Hypothalamic–Pituitary–Adrenal axis) regulates cortisol—our primary stress hormone.

Chronic stress + low magnesium = dysregulated cortisol rhythms.

5.1 The Cortisol Cycle in Modern Life

Healthy rhythm:

- high in the morning
- gradually decreases across the day
- lowest at night → sleep initiation

Stress rhythm:

- high at night
- spikes at 3–4 a.m.
- restlessness upon waking
- fatigue during the day

This produces the classic symptoms:

- early waking
 - nighttime anxiety
 - shallow sleep
 - morning dread
 - inability to relax
-

5.2 Magnesium's Role in HPA Axis Regulation

Research shows magnesium:

- reduces ACTH-driven cortisol release
- improves stress response thresholds
- prevents the HPA "overreaction loop"
- stabilizes parasympathetic tone

People often report:

- "I woke up less anxious."
 - "My heart stopped racing at night."
 - "I feel more emotionally stable."
-

5.3 Why Magnesium Glycinate Is Superior for HPA Axis

Because of:

- better neural uptake
- glycine's calming synergy
- less digestive irritation
- stable blood levels

This form stays active in the window that matters most: **nighttime**.

HPA Axis · Cortisol
& The Stress Loop

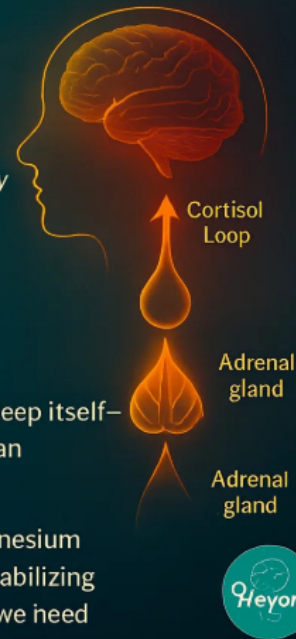
Why magnesium glycinate restores the most fragile system in modern physiology

If magnesium deficiency destabilizes the nervous system, the HPA axis (hypothalamus-pituitary-adrenal axis) is where this instability becomes visible—insomnia, anxiety, irritability, early waking, emotional volatility, and a baseline sense of internal pressure.

The HPA axis is the engine that controls cortisol rhythms. When working properly, cortisol rises in the morning, *falls smoothly* throughout the day, and remains low at night—insomnia, anxiety, irritability, early waking, emotional volatility, and a *baseline* sense of internal pressure.

It strengthens the architecture of sleep itself—the biological blueprint—rather than artificially overriding it.

Magnesium—and specifically magnesium glycinate—plays a unique role in stabilizing the HPA axis. To understand why, we need to examine the stress loop step-by-step.



6. Mechanism Axis IV - Mitochondrial–Neuroenergetic Restoration

The fourth axis addresses something subtle but crucial:

neuronal energy.

Sleep, mood, and cognition collapse not only from neurotransmitter imbalance, but from energetic instability.

6.1 Why Magnesium Is Central to ATP Production

Magnesium is required for:

- ATP synthesis
- ATP transport
- stabilizing mitochondrial membranes
- buffering oxidative stress

ATP itself exists mostly as **Mg-ATP**, not ATP alone.

Low magnesium = low usable energy.

6.2 Energy Deficit → Anxiety & Insomnia

When neurons run low on ATP:

- inhibitory pathways weaken (→ low GABA tone)
- excitatory pathways become unstable

- the brain “pushes harder” to stay functional
- emotional regulation collapses

This produces:

- mental fatigue with simultaneous restlessness
- “wired but tired” state
- irritability
- cognitive noise

Magnesium Glycinate replenishes this axis smoothly.

6.3 Glycine’s Role in Neuroenergetic Smoothing

Glycine:

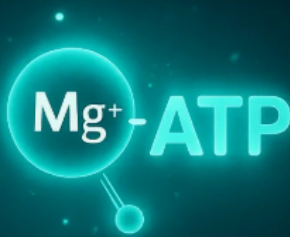
- supports mitochondrial biogenesis
- reduces inflammatory load
- improves metabolic flexibility
- enhances restorative sleep phases

Together, this improves the **baseline stability of the nervous system**, not just nighttime symptoms.

ATP Exists As Mg-ATP: The Fundamental Biochemical Reality

It reduces
NMDA-driven
stress signaling:

- activate kinase enzymes
- drive muscle contraction
- support synaptic transmission
- fuel detoxification
- maintain membrane pumps
- stabilize mitochondrial function



low magnesium = low usable ATP
high stress = higher magnesium demand
chronic fatigue = functional magnesium
 insufficiency

Citrate, oxide, and chloride forms do not consistently raise intracellular magnesium enough to restore ATP-dependent processes. Glycinate does – because it is absorbed predictably via PEPT1 and avoids intestinal losses.

End of Part I

Toward a Complete Four-Axis Model of Emotional–Sleep Regulation**

What we have explored so far is only the foundation.

In this first half, we examined how Magnesium Glycinate interacts with the nervous system at its most fundamental layers: the inhibitory GABA brake, the excitatory NMDA engine, the cortisol-driven HPA loop, and the mitochondrial energy core that determines how stable the entire network feels.

These four systems do not operate independently. In real biology - and in real life - they form a tightly interwoven matrix. When one axis shifts, the others follow. When one becomes overloaded, the entire emotional–sleep system begins to destabilize.

Understanding each axis individually is essential. But understanding how they integrate is where the real change begins.

That is where we will continue tomorrow.

In Part II, we will map these mechanisms into a single, unified framework - the Four-Axis Integrated Model - and examine how clinical evidence, real-world phenotypes, dosing strategies, and Keyora's research perspective converge into a coherent, practical approach to emotional–sleep restoration.

Thank you for reading Part I of this Keyora Research Notes Series. We invite you to return tomorrow as we assemble the full system.

Part I Conclusion: Mapping the Axes of Sleep Recovery

Magnesium Glycinate interacts with the nervous system



GABA
brake



NMDA
engine



cortisol
HPA loop



mitochondrial
matrix

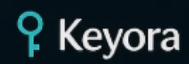
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