SelfDecode™

GABA & Glutamate Pathway

Sample Client

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Table of Contents

Personal information

NAME

Sample Client

SEX AT BIRTH

Male

HEIGHT

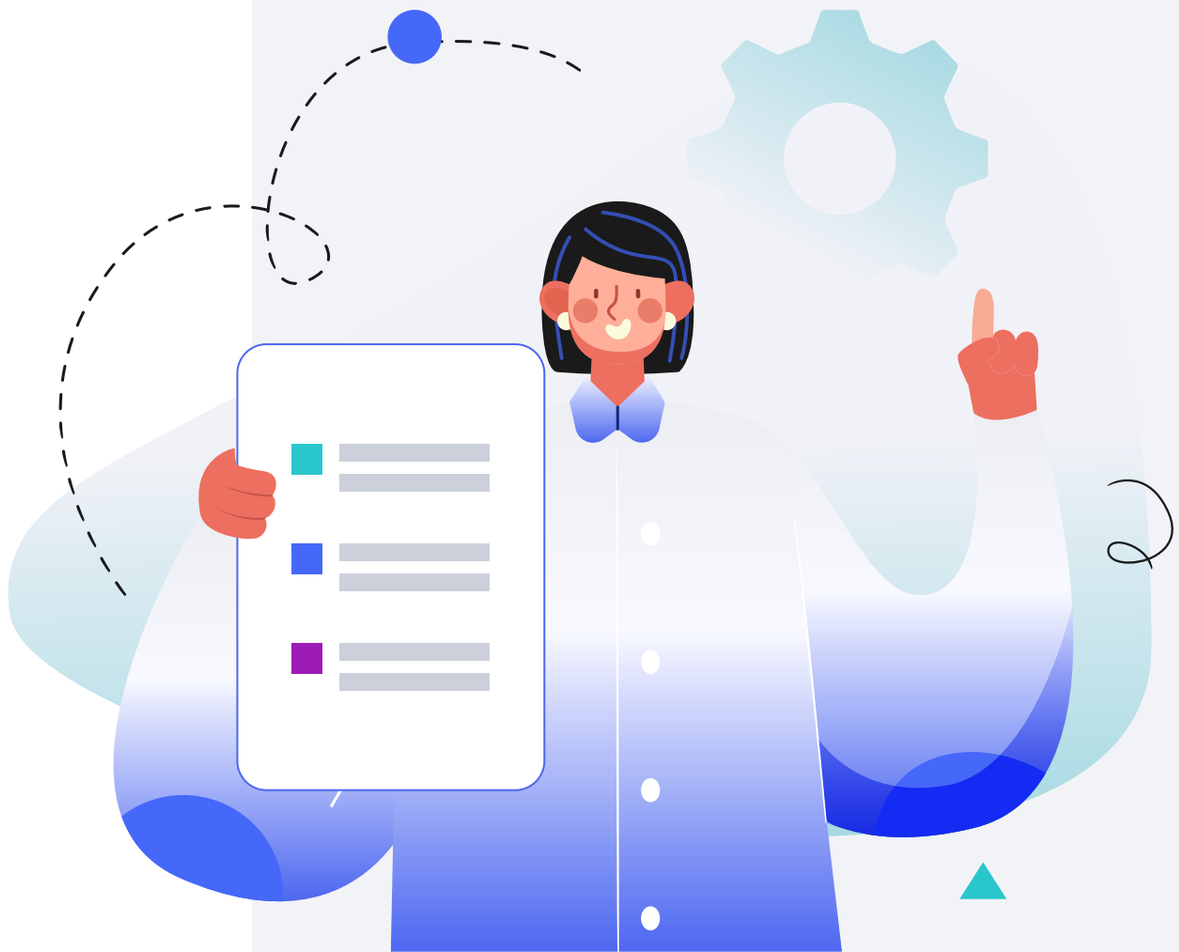
5ft 10" 178cm

WEIGHT

215lb 97.5kg

DISCLAIMER

This report does not diagnose this or any other health conditions. Please talk to a healthcare professional if this condition runs in your family, you think you might have this condition, or you have any concerns about your results.



How this works

Why do some people feel chronically anxious, mentally foggy, or wired yet exhausted — even when nothing in their life obviously explains it? For many, the answer lies in a fundamental imbalance between two of the brain's most important chemical messengers.

Glutamate and GABA are the brain's primary accelerator and brake. Glutamate drives neuronal activity — powering learning, focus, and fast thinking. GABA quiets it down — promoting calm, sleep, and emotional stability. When these two systems are in balance, the brain is resilient and clear. When they're not, the effects ripple through mood, cognition, stress tolerance, and physical health.

This report examines the genes that govern every major step of this system — and where your personal variants may be tipping the balance.

It begins with how the brain acquires and processes glutamate, its primary excitatory fuel — including key players like **GLS and SLC1A2**. From there, it looks at the critical conversion step where glutamate is transformed into GABA — a process entirely dependent on the **GAD1 and GAD2** enzymes and the nutrients that power them. It then examines the receptors that receive glutamate's excitatory signals, including **GRIN2B and GRIA1,** and how variants here can mean your brain runs hotter than it should.

On the calming side, the report covers the GABA-A receptor genes — including **GABRA6 and GABRG2** — responsible for translating inhibitory signals into genuine relaxation and mental quiet, and the **SLC6A1** transporter that controls how quickly GABA is cleared from the synapse. Finally, it looks at the metabolic pathways governed by **ALDH5A1 and ABAT**, where impairment can allow toxic byproducts to accumulate.

Together, these pathways tell a precise and personal story about your brain's chemical balance — and where targeted support can make the most meaningful difference.

How to Interpret Your GABA & Glutamate Pathway Report

The opening visual diagram is the core of the report. It shows the step-by-step biochemical flow of GABA & glutamate metabolism and effects, with each gene and its associated SNP result placed at the exact step where it acts. Reading these diagrams helps you understand not just how individual genes behave, but how they interact as a system.

Gene result labels tell you whether your variant is associated with:

- **Typical activity (blue)** — your gene functions within the normal expected range
- **Altered activity (orange or red)** — your variant differs from the most common form; this may mean higher or lower enzyme activity depending on the specific gene
- **Better activity (green)** — your variant is associated with a more favorable functional outcome for that step

Directional arrows on genes indicate whether a variant tends to push that step up (upregulating) or down (downregulating) relative to typical activity.

Biological compounds shown in the diagrams represent the metabolites produced at each stage. Where lab result slots appear alongside these compounds, you can enter your own bloodwork values to see how your measured hormone levels align with what your genetics might predict.

If a lab result is available for a specific compound, the badge color next to its name will indicate whether the result falls within the optimal range.

The **legend** in the bottom left corner explains the meaning of each element on the pathway, so please read it carefully.

Understanding the Pathway as a System

Because these genes operate as an interconnected network, your results should be read holistically rather than gene by gene. A few principles that help with interpretation:

- **The glutamine-glutamate cycle sets the brain's baseline level of excitation.** GLS converts glutamine into glutamate (the brain's main excitatory chemical) inside neurons, while GLUL converts it back in support cells for safe storage and recycling. The balance between these two steps

determines how much free glutamate is available before any other gene has a chance to act — making them important context for everything else in the report.

- **Glutamate clearance genes determine whether excess is safely removed or allowed to build up.** SLC1A2 and SLC1A3 are the brain's main transporters for reabsorbing glutamate after it has been used. Reduced activity in either means glutamate lingers longer, increasing the risk of overstimulation. These genes should be read alongside GLS and GLUL — a tendency toward high glutamate production combined with poor clearance is more significant than either alone.
- **GAD1 and GAD2 are where the brain converts excitatory signals into calming ones.** Both enzymes convert glutamate into GABA, which is the brain's main calming chemical. GAD1 maintains a steady background level; GAD2 responds to immediate demand. Variants reducing either enzyme directly reduce the calming signal available to the brain. Both depend on vitamin B6, so nutrient status at this step matters.
- **Glutamate receptor genes shape how strongly excitatory signals land.** GRIN2A, GRIN2B, GRIA1, GRIA3, GRM3, and GRM7 determine how the brain receives and regulates glutamate signals. GRM3 and GRM7 act as a natural brake — reducing glutamate release when levels get too high. Variants that weaken this self-regulating mechanism are particularly worth noting when other genes already point toward glutamate excess.
- **GABA receptor genes determine how well the calming signal is received.** GABRA1, GABRA2, GABRA6, GABRB2, GABRB3, and GABRG2 each make up part of the receptor that GABA binds to. Different combinations of these subunits dominate in different brain areas — GABRA1 is the most widespread and affects sleep and seizure threshold; GABRA2 is concentrated in areas involved in anxiety and impulse control; GABRG2 is the site where medications like benzodiazepines work. Having multiple receptor variants does not simply add up — the impact depends on which brain circuits are most affected.
- **SLC6A1 controls how long GABA stays active after it is released.** This transporter recycles GABA out of the synapse, ending its calming effect. Variants that speed up this recycling shorten the window in which GABA can act — effectively reducing its functional impact even when production and receptors are working normally.
- **ALDH5A1 and ABAT clear the waste products left over when GABA is broken down.** After GABA has done its job, ABAT begins breaking it down and ALDH5A1 clears a toxic byproduct called succinic semialdehyde. If ALDH5A1 activity is reduced, this byproduct can accumulate and be converted into a substance that further disrupts the brain's excitatory-inhibitory balance. These genes sit at the end of the pathway but can make upstream imbalances worse, especially under stress.
- **CACNA1C connects calcium channel activity to the overall balance between excitation and calm.** This gene controls a calcium channel that affects how easily neurons fire and release neurotransmitters across both the excitatory and inhibitory systems. Its variants are particularly relevant when read alongside the NMDA receptor and GABA receptor gene results.

Other Result Details

The **Results Overview** provides a structured table of every gene and SNP analyzed in the report, grouped by sub-pathway. Use this section to quickly locate your genotype for any specific gene. Each gene appears with its relevant SNP identifiers and your genotype. Alongside the genetic results, suggested lab markers are listed for each sub-pathway — these are the blood, urine, or saliva tests most relevant to validating or complementing your genetic findings.

The detailed **Gene–SNP Breakdown** section explains each gene and variant in depth. For every gene you will find:

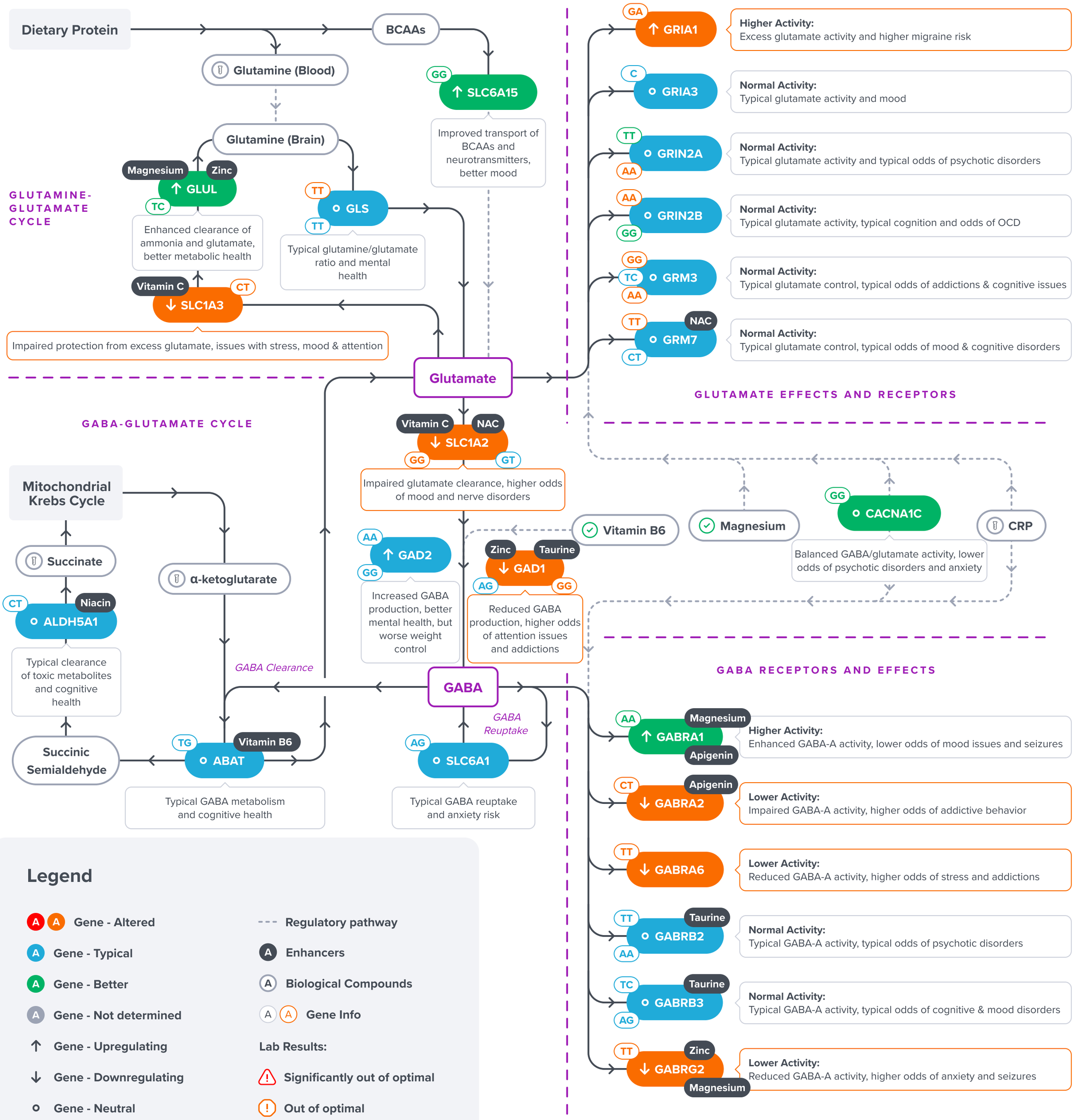
- **What the gene does** — its biological role in hormone production, transport, signaling, or detoxification
- **What your specific genotype means** — whether your variant is associated with higher, lower, or typical activity at that step, and what health outcomes have been linked to it in research
- **Enhancers** — nutrients or compounds that support the function of that enzyme, listed where relevant

The **Recommendations** section lists personalized dietary, lifestyle, and supplement strategies, ranked by likely impact based on your specific gene variants. Each recommendation includes the genes it is relevant to and explains the biological mechanism behind it. The recommendations are intended as a starting point for discussion with a healthcare provider, not as a prescriptive protocol.

Lab Markers to Check list the laboratory tests most relevant to your genetic profile, organized by the gene(s) that make them informative for you. Running these tests allows you to see whether the functional tendencies suggested by your genetics are actually showing up in your measured physiology. Genetics describes predisposition; labs confirm whether that predisposition is being expressed under your current diet, lifestyle, and environment.

GABA - Glutamate Pathway

GABA - Glutamate Pathway



Legend

- Gene - Altered
- Gene - Typical
- Gene - Better
- Gene - Not determined
- Gene - Upregulating
- Gene - Downregulating
- Gene - Neutral
- Genotype
- Genotype not determined
- Main pathway
- Regulatory pathway
- Enhancers
- Biological Compounds
- Gene Info

Lab Results:

- Significantly out of optimal
- Out of optimal
- Normal
- Optimal
- Missing lab result

Results Overview

Glutamine-Glutamate Cycle

Gene - SNP Summary

GRIA1	rs2195450	↑ GA
GLS	rs12185688	o TT
	rs13000464	o TT
GRM3	rs12704290	↓ GG
	rs6465084	↓ AA
	rs274622	o TC
SLC6A15	rs1545843	↑ GG
SLC1A2	rs4354668	↓ GG
	rs3794087	o GT
GRIN2A	rs11644461	o TT
	rs7206256	o AA
GRM7	rs3749380	↓ TT
	rs779867	o CT
SLC1A3	rs2269272	↓ CT
GRIA3	rs687577	o C
GRIN2B	rs1019385	↑ AA
	rs1805476	↓ GG
CACNA1C	rs1006737	o GG
GLUL	rs10911021	↑ TC

Labs Summary

Homocysteine · Magnesium · Magnesium, RBC · Zinc · C-Reactive Protein (CRP) · Coenzyme Q10 · Cortisol · Glycine, Plasma · IL-6 · Serine, Plasma · TNF-alpha

GABA-Glutamate Cycle

Gene - SNP Summary

GABRA2	rs279858	↓ CT
GAD1	rs1978340	o AG
	rs3749034	↓ GG
GAD2	rs992990	↑ AA
	rs2236418	↑ GG
GABRA6	rs3219151	↓ TT
ABAT	rs2270288	o TG
GABRB2	rs1816071	o TT
	rs1816072	o AA
SLC6A1	rs2697153	o AG
GABRG2	rs211037	↓ TT
ALDH5A1	rs2760118	o CT
GABRB3	rs2081648	o TC
	rs4906902	o AG
GABRA1	rs2279020	↑ AA

Labs Summary

Magnesium · Magnesium, RBC · Vitamin B6 · Zinc · Cortisol · Gamma-Aminobutyric Acid (GABA), Plasma

Glutamine, Plasma

Your recommendations

Your recommendations are prioritized according to the likelihood of it having an impact for you based on your lab results, along with the amount of scientific evidence supporting the recommendation.

You'll likely find common healthy recommendations at the top of the list because they are often the most impactful and most researched.

		DOSAGE			DOSAGE
1	L-Theanine	100 mg	2	Magnesium L-Threonate	1500 mg
3	Taurine	500 mg	4	Magnesium Glycinate	200 mg
5	N-acetylcysteine (NAC)	1200 mg	6	Zinc	15 mg
7	Limit Caffeine Intake		8	Myo-inositol	4 g
9	Potassium BHB	3 g	10	Ashwagandha KSM-66	
11	Coenzyme Q10 (CoQ10)	100 mg	12	GABA Supplement	1500 mg
13	Glutamine	5 g	14	Glycine	3 g
15	Lemon Balm	500 mg	16	Lithium Orotate	5 mg
17	Low-Carbohydrate Diet		18	Low-Glutamate Diet	
19	Omega-3 (Fish Oil)	2000 mg	20	Pyridoxal-5-Phosphate (PLP)	20 mg
21	Riboflavin (Vitamin B2)	25 mg	22	Sarcosine	2 g

L-Theanine

How to implement

Take 100-400 mg of L-theanine supplement daily. It can be consumed at any time of the day, with or without food.

TYPICAL STARTING DOSE

100 mg

How it helps

L-theanine is an amino acid found in green tea that crosses the blood-brain barrier and exerts gentle, broad effects on neurotransmitter balance. It increases GABA activity, promotes alpha brain wave states associated with calm alertness, and acts as a partial NMDA receptor antagonist — directly reducing glutamate's excitatory impact. Unlike sedatives, it produces relaxation without drowsiness. For those with overactive glutamate signalling or underactive GABA systems, L-theanine provides meaningful and well-tolerated daily support with an excellent safety profile and consistent clinical backing.

Personalized to Your Genes

↓ GABRA2	Positively modulates GABA-A receptors, particularly relevant for anxiety-related α2 subunit pathways
↓ GABRA6	Positive GABA-A modulator that broadly compensates for reduced α6 subunit-mediated inhibition
↓ GABRG2	Supports GABA-A activity broadly and promotes alpha-wave states impaired by γ2 dysfunction
○ GRIN2A	Partial NMDA antagonist effect that gently dampens dysregulated receptor activity
○ GRIN2B	Crosses the BBB and exerts a partial NMDA antagonist effect, dampening overactivation
○ GRM3	Modulates glutamate tone and promotes calming neurotransmitter balance as a compensatory measure
○ GRM7	Modulates glutamate/GABA balance, providing compensatory inhibitory tone

Magnesium L-Threonate

How to implement

Take magnesium L-threonate in a dosage of 1,500-2,000 mg per day, divided into two or three doses, with or without food. This supplement regimen should be followed consistently for at least one month to evaluate its benefits on cognitive function and sleep quality.

TYPICAL STARTING DOSE

1500 mg

How it helps

Magnesium L-threonate is the only form of magnesium demonstrated to meaningfully raise magnesium levels inside the brain. This is critical because the brain's primary defence against glutamate overactivity — NMDA receptor blockade — is magnesium-dependent. Standard magnesium forms don't cross the blood-brain barrier well, making threonate uniquely suited for neurological applications. It has shown improvements in cognitive function, synaptic density, and anxiety in clinical research. For those with glutamate excess or GABA deficiency affecting mental health, this is the most targeted magnesium option.

Personalized to Your Genes

↓ GABRG2	Crosses the BBB effectively — critical when γ2-containing receptors that govern synaptic clustering are underactive
↑ GRIA1	Magnesium modulates AMPA receptor sensitivity — directly relevant to this gene's overactivity
↓ SLC1A2	NMDA receptor blockade is critical when synaptic glutamate overflows due to impaired SLC1A2 clearance
↓ SLC1A3	Crosses the blood-brain barrier to block NMDA receptors when synaptic glutamate goes unchecked
○ GRIN2A	Magnesium is the primary endogenous NMDA channel blocker — essential when receptor regulation is altered
○ GRIN2B	The most direct intervention — magnesium physically blocks the NMDA channel this gene overactivates
○ GRM3	Buffers excess synaptic glutamate that goes uncontrolled when GRM3 metabotropic braking is impaired

Taurine

How to implement

Take 1-4 g of taurine supplement daily, divided into two or three doses with meals for optimal absorption. It can be taken continuously, with periodic evaluations of its effects and benefits.

TYPICAL STARTING DOSE

500 mg

How it helps

Taurine is an amino acid with direct inhibitory activity in the brain — it binds to both GABA-A and glycine receptors, producing calming effects that closely mimic GABA itself. This makes it especially valuable when GABA production is low or GABA receptors are underperforming. Taurine also modulates calcium signaling, protecting neurons from overstimulation driven by excess glutamate. It's naturally concentrated in the brain and declines with age and stress, making supplementation broadly relevant for neurological balance and resilience.

Personalized to Your Genes

↓ GABRA6	Direct GABA-A agonist — particularly relevant as α6 is highly expressed in the cerebellum where taurine is active
↓ GABRG2	GABA-A agonist that partially compensates for reduced synaptic receptor clustering from γ2 underactivity
↓ GAD1	Acts on GABA receptors as a partial agonist, compensating for reduced GABA output from this variant
↓ SLC1A2	Provides inhibitory buffering against excess synaptic glutamate that SLC1A2 fails to clear
↓ SLC1A3	Inhibitory neuromodulator that helps buffer excess synaptic glutamate this transporter fails to clear
○ GRM7	Inhibitory neuromodulator that helps offset excess glutamate release when GRM7 autoreceptors are underactive

Magnesium Glycinate

How to implement

Take 200-400 mg of magnesium glycinate daily, preferably in the evening or divided into two doses with meals to enhance absorption. Continue this supplementation routine daily for at least one month to evaluate its benefits on your health.

TYPICAL STARTING DOSE

200 mg

How it helps

Magnesium is one of the brain's most important natural regulators of glutamate activity. It physically blocks NMDA receptors — the main entry point for glutamate's excitatory signals — preventing overstimulation and excitotoxicity. Low magnesium is extremely common and directly worsens anxiety, poor sleep, and glutamate excess. The glycinate form is highly absorbable and also delivers glycine, itself a calming neurotransmitter. This makes magnesium glycinate a foundational supplement for anyone with glutamate overactivity or insufficient GABA signaling.

Personalized to Your Genes

↓ GABRA2	Broadly supports GABAergic signaling when α2-mediated anxiolytic function is reduced
↓ GABRA6	Supports inhibitory tone in cerebellar circuits where α6 subunit function is most prominent
↓ GAD1	Supports GAD enzyme activity and independently promotes GABAergic tone
○ GRM7	Compensates for lost presynaptic glutamate braking by dampening NMDA receptor excitability downstream

N-acetylcysteine (NAC)

How to implement

Take 600 mg of N-Acetylcysteine (NAC) supplement daily with water. It can be taken at any time of the day, but try to take it at the same time each day for best results.

TYPICAL STARTING DOSE

1200 mg

How it helps

NAC is a precursor to glutathione, the brain's master antioxidant, but its most relevant action here is modulating the cystine-glutamate antiporter — a key transporter that controls how much glutamate is released into the extracellular space. By upregulating this system, NAC effectively reduces excess glutamate outside of cells, lowering the risk of excitotoxicity. It also protects neurons from oxidative damage caused by accumulated metabolic byproducts. NAC is one of the most versatile and well-researched tools for glutamate management.

Personalized to Your Genes

↓ SLC1A2	Reduces extracellular glutamate load that accumulates when this astrocytic transporter is underactive

↓ SLC1A3	Boosts glutathione and modulates cystine-glutamate exchange, reducing extracellular glutamate
GRIN2B	Reduces extracellular glutamate via cystine-glutamate antiporter, lowering NMDA stimulation upstream
GRM3	GRM3 regulates cystine-glutamate exchange; NAC directly supports this pathway when the receptor is underactive

Zinc

How to implement

Take a 15 mg zinc supplement daily, ideally with a meal to enhance absorption.

TYPICAL STARTING DOSE

15 mg

How it helps

Zinc is a key endogenous modulator of both NMDA and GABA-A receptors. At NMDA receptors, zinc acts as a natural brake — reducing their sensitivity to glutamate and preventing overstimulation. At GABA-A receptors, zinc helps regulate activity depending on the subunit composition involved. Zinc deficiency is common and can significantly worsen symptoms of glutamate excess or GABA insufficiency. Because zinc directly interacts with the receptor proteins encoded by several of the genes in this report, maintaining adequate zinc status is an important and targeted priority.

Personalized to Your Genes

↓ GABRG2	γ2 subunit normally reduces zinc sensitivity; with reduced γ2, zinc balance becomes especially important to optimize
GRIN2A	Zinc is a co-modulator of NMDA receptors; deficiency destabilizes altered receptor function further
GRIN2B	Zinc is an endogenous NMDA inhibitor — directly reduces overactivity of the receptor this gene encodes

Limit Caffeine Intake

How to implement

Limit your caffeine consumption to less than 200 milligrams per day, equivalent to about two 6-ounce cups of coffee. Aim to avoid caffeine-containing foods and beverages such as tea, chocolate, and some soft drinks, especially in the late afternoon and evening to minimize sleep disturbances.

How it helps

Caffeine works primarily by blocking adenosine receptors, but it also stimulates glutamate release throughout the brain. For most people this is manageable, but for those with already-elevated glutamate tone or impaired GABA signaling, excess caffeine can tip the excitatory/inhibitory balance toward anxiety, overstimulation, and poor sleep. High caffeine also suppresses glutamate transporter activity. Moderating intake — rather than eliminating it — is the practical goal, as caffeine sensitivity varies significantly by genetics and current neurochemical status.

Personalized to Your Genes

↓ SLC1A3	Caffeine acutely impairs glutamate transporter activity, worsening the effect of this variant
GRM7	Caffeine increases glutamate release — particularly problematic when the presynaptic brake (GRM7) is already weak

Myo-inositol

How to implement

Take 2 grams of myo-inositol supplement orally twice a day, preferably with meals to enhance absorption. This regimen should be followed daily for at least 6 months to observe significant benefits.

TYPICAL STARTING DOSE
4 g

How it helps

Myo-inositol is a naturally occurring compound that acts as a second messenger in signalling pathways downstream of GABA-A and glutamate receptors. It enhances receptor sensitivity — making existing GABA more effective even when production or receptor density is low. Clinical trials support its use for anxiety, panic disorder, and OCD at doses of 4–12g/day, with an excellent safety profile. For those with multiple GABA-A receptor gene variants, inositol offers a way to amplify the effect of whatever GABA signalling capacity remains, without directly sedating the brain.

Personalized to Your Genes

↓ GABRA2	Modulates GABA-A receptor sensitivity and has strong evidence for anxiety and panic — α2 is the key anxiolytic subunit
↓ GABRA6	Enhances GABA-A receptor sensitivity, making reduced receptor populations more responsive

Potassium BHB

How to implement

Follow the dosage instructions provided on the supplement label or as advised by a healthcare professional. Common doses range from 3 to 6 grams per serving. Potassium BHB supplements typically come in powder or capsule form. If using a powder, mix it with water or a low-carb beverage according to the package instructions. Ensure it is well-dissolved before consuming. For optimal results, take Potassium BHB in the morning or before workouts. This timing can help boost energy levels, enhance mental clarity, and improve exercise performance. Maintain adequate hydration, as BHB can have a diuretic effect. Drinking plenty of water helps prevent dehydration and supports electrolyte balance.

TYPICAL STARTING DOSE

3 g

How it helps

Beta-hydroxybutyrate (BHB) is a ketone body produced during fasting or ketogenic eating, and it has direct effects on brain neurotransmistry. BHB upregulates the expression of glutamate transporters in astrocytes — the brain's cleanup cells — helping clear excess synaptic glutamate more efficiently. It also enhances GABA synthesis relative to glutamate and reduces neuronal excitability. For those with impaired glutamate clearance, exogenous BHB provides a fast route to these benefits without requiring strict dietary changes.

Personalized to Your Genes

↓ SLC1A2	Ketones upregulate glutamate transporter expression in astrocytes, partially compensating for this variant
↓ SLC1A3	Ketone bodies upregulate glutamate transporter expression, partially compensating for low SLC1A3

Ashwagandha KSM-66

How it helps

Ashwagandha is an adaptogenic herb with well-documented anxiety-reducing and neuroprotective effects. Its active compounds, withanolides, interact with GABA-A receptors and help modulate the stress response, reducing the cortisol-driven glutamate surges that occur during chronic stress. High cortisol is a major trigger for excess glutamate release, so dampening the stress axis indirectly protects GABA/glutamate balance. The KSM-66 extract is the most clinically studied form, with consistent evidence for reducing anxiety and improving cognitive resilience.

Personalized to Your Genes

GRM7

Stress worsens dysregulated glutamate release; adaptogenic support is especially relevant with impaired GRM7

Coenzyme Q10 (CoQ10)

How to implement

Take a 100 mg Coenzyme Q10 (CoQ10) supplement once daily with a meal that contains fat for better absorption.

TYPICAL STARTING DOSE

100 mg

How it helps

CoQ10 is a critical component of the mitochondrial electron transport chain, responsible for generating the cellular energy that neurons need to manage glutamate signaling safely. When neuronal energy metabolism falters — as it does under conditions of glutamate overactivation — cells become more vulnerable to excitotoxic damage. CoQ10 also acts as a fat-soluble antioxidant, neutralizing the oxidative stress generated by excess glutamate activity. It has specific evidence for reducing migraine frequency, a condition closely linked to glutamate dysregulation.

Personalized to Your Genes

↑ GRIA1

Supports neuronal energy metabolism that becomes strained under AMPA receptor overactivation

GABA Supplement

How to implement

Take a GABA supplement in a dose up to 500 mg, three times daily. This supplementation should be taken with water, preferably with food to minimize gastrointestinal discomfort. The duration of supplementation can vary, but many users may start to see benefits within a few weeks of consistent use.

TYPICAL STARTING DOSE

1500 mg

How it helps

GABA supplements provide the inhibitory neurotransmitter directly, though their ability to cross the blood-brain barrier has historically been debated. More recent evidence, particularly for PharmaGABA (a naturally fermented form), supports genuine central effects — likely via the gut-brain axis and vagal nerve signalling. For those with genetically reduced GABA production or receptor activity, supplemental GABA can provide a meaningful boost to inhibitory tone, reducing anxiety, supporting sleep, and offsetting the effects of glutamate excess. It works best alongside cofactor support (B6, magnesium) that optimises endogenous production as well.

Personalized to Your Genes

↓ GAD1 — Provides direct GABA precursor support when endogenous production via GAD1 is genetically reduced

Glutamine

How to implement

Take 5 to 10 grams of glutamine powder, mixed with water or another beverage, daily. It can be divided into two servings, one in the morning and the other in the evening. This supplementation is generally considered safe for long-term use, but it's best to consult with a healthcare provider for personalized advice.

TYPICAL STARTING DOSE

5 g

How it helps

Glutamine is the brain's most abundant amino acid and the direct precursor to both glutamate and GABA. It serves as the primary nitrogen shuttle between neurons and astrocytes, playing a central role in the glutamate-glutamine cycle that recycles neurotransmitters after each synaptic signal. When GABA production is low, increasing glutamine availability provides more raw material for the GAD enzyme to work with. Glutamine also supports gut integrity and immune function, reducing systemic inflammation that indirectly disrupts brain neurotransmitter balance.

Personalized to Your Genes

↓ GAD1

Primary substrate for GABA synthesis; increasing availability partially compensates for reduced GAD1 activity

Glycine

How to implement

Take 3 to 5 grams of glycine supplement per day with water, preferably before bedtime for sleep improvement or spread throughout the day for general health benefits. Continue indefinitely as needed, but consult a healthcare provider for long-term use beyond three months.

TYPICAL STARTING DOSE

3 g

How it helps

Glycine is a dual-function neurotransmitter — inhibitory in the spinal cord and brainstem, but also a required co-agonist at NMDA glutamate receptors in the brain. This means glycine availability directly shapes how active NMDA receptors are. Supplementing glycine before bed helps optimize this co-agonist tone, supporting healthy receptor function rather than driving excess activation. Glycine also promotes deeper, more restorative sleep — an essential window for glutamate clearance and synaptic recalibration. It is safe, well-tolerated, and broadly supportive of brain chemistry.

Personalized to Your Genes

GRIN2A

NMDA receptors require glycine as a co-agonist — optimizing glycine tone helps normalize altered signaling

Lemon Balm

How to implement

Take a 500 mg lemon balm supplement daily. This dosage can be consumed at once or divided into smaller doses throughout the day, according to personal preference or the guidance of a healthcare provider.

TYPICAL STARTING DOSE

500 mg

How it helps

Lemon balm (Melissa officinalis) is a well-studied herb for anxiety and sleep, with mechanisms directly relevant to GABA balance. Its active compound rosmarinic acid inhibits GABA transaminase — the enzyme that breaks GABA down — effectively raising GABA levels in the brain. This mechanism makes lemon balm especially valuable for those with excessive GABA reuptake or degradation. It also has mild anti-glutamate effects and reduces cortisol-driven excitatory signalling. Standardised extracts have clinical evidence for reducing anxiety and improving sleep quality with minimal side effects.

Personalized to Your Genes

↓ GABRA2 — Inhibits GABA reuptake and supports GABA-A activity, compensating for reduced α2 function

Lithium Orotate

How to implement

Take 5 mg of lithium orotate supplement daily, preferably with a meal to enhance absorption. It is recommended to consult with a healthcare provider prior to starting the supplement, particularly for long-term use.

TYPICAL STARTING DOSE

5 mg

How it helps

Low-dose lithium orotate (distinct from prescription lithium) has neuroprotective and neuromodulatory properties relevant to glutamate balance. It reduces glutamate-driven excitotoxicity by modulating downstream signalling pathways, supports BDNF (a key neuronal growth factor), and stabilises neuronal firing patterns disrupted by glutamate or calcium channel dysregulation. Research suggests it also supports glutamate recycling and reduces neuroinflammation. At the low doses used in orotate form, it is considered safe and is increasingly used in functional medicine for mood stabilisation and brain resilience.

Personalized to Your Genes

GRM3 — Supports glutamate recycling and neuroprotection when metabotropic receptor control is compromised

Low-Carbohydrate Diet

How to implement

Limit your daily intake of carbohydrates to less than 26% of your total daily calories. For a standard 2000-calorie diet, this means consuming no more than 130 grams of carbohydrates per day. Focus on including non-starchy vegetables, lean proteins, and healthy fats in your meals while minimizing the intake of sugars, bread, pasta, and other high-carb foods.

How it helps

A low-carbohydrate diet reduces blood glucose and insulin fluctuations that can drive neuroinflammation and glutamate excess. More directly, carbohydrate restriction promotes ketone production, which shifts brain energy metabolism in ways that favour GABA over glutamate — the brain actually synthesizes more GABA relative to glutamate in a ketotic state. Low-carb eating also reduces excitatory signalling through AMPA and NMDA receptors. For those with overactive glutamate pathways, dietary carbohydrate reduction is one of the more impactful non-supplement interventions available.

Personalized to Your Genes

↑ GRIA1 Ketones reduce AMPA-mediated excitability and have evidence for migraine reduction

Low-Glutamate Diet

How to implement

To follow a low-glutamate diet, avoid foods high in glutamate such as processed meats (e.g., sausages, ham), certain cheeses (e.g., Parmesan), soy products, and foods containing MSG. Opt for fresh fruits, vegetables, whole grains, and lean proteins. Implement this diet daily over an extended period for potential benefits.

How it helps

A low-glutamate diet involves reducing foods that are naturally high in free glutamate or that contain added MSG and glutamate-based flavour enhancers. High dietary glutamate can raise extracellular glutamate levels in the brain, particularly in those with impaired clearance or overactive glutamate receptors — compounding the effects of genetic vulnerabilities in this report. Key foods to limit include aged cheeses, cured meats, soy sauce, fish sauce, bone broth concentrates, yeast extract, and ultra-processed foods. For those with multiple glutamate-related gene variants, dietary glutamate reduction is one of the most direct and immediately actionable interventions available.

Personalized to Your Genes

↑ GRIA1 Reduces the substrate load that overstimulates already hyperactive AMPA receptors

Omega-3 (Fish Oil)

How to implement

Take 1-2 g of omega-3 (fish oil) supplement daily, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

2000 mg

How it helps

Omega-3 fatty acids (EPA and DHA) are structural components of neuronal cell membranes and directly influence how receptors — including NMDA and GABA-A — are embedded and function. DHA in particular affects membrane fluidity in ways that modulate metabotropic glutamate receptor sensitivity. EPA has potent anti-neuroinflammatory effects, reducing the inflammatory signalling that dysregulates GABA/glutamate balance. Omega-3 deficiency is widespread and worsens receptor dysfunction across multiple pathways implicated in this report. Regular intake supports the foundational architecture of healthy brain signalling.

Personalized to Your Genes

GRM3	Improve neuronal membrane fluidity, which affects metabotropic receptor sensitivity and signaling efficiency

Pyridoxal-5-Phosphate (PLP)

How to implement

Take 20-50 mg of pyridoxal-5-phosphate daily, with or without food, preferably at the same time each day. Swallow with water, and if you experience stomach discomfort, take it with a meal. Consult your healthcare provider for personalized dosing, especially if pregnant, breastfeeding, or on medications, and avoid exceeding the recommended dose unless advised.

TYPICAL STARTING DOSE

20 mg

How it helps

Pyridoxal-5-phosphate (PLP) is the active form of Vitamin B6 and the essential cofactor for GAD — the enzyme that converts glutamate into GABA. Without adequate PLP, this conversion slows dramatically, leading to excess glutamate and insufficient GABA. Many people have impaired conversion of standard B6 to PLP, making the active form the only reliable option.

Correcting PLP deficiency is arguably the most targeted nutritional intervention for low GABA production, directly addressing the enzyme's rate-limiting requirement.

Personalized to Your Genes

↓ GAD1 P5P is the essential cofactor for GAD1 — directly supports the enzyme this gene encodes

Riboflavin (Vitamin B2)

How to implement

Take a riboflavin (vitamin B2) supplement daily, with a dose ranging from 5mg to 400mg, depending on the specific health concern or advice from a healthcare provider. Swallow the supplement with water, preferably with a meal to enhance absorption. This regimen can be continued long-term or as directed by a healthcare professional.

TYPICAL STARTING DOSE

25 mg

How it helps

Riboflavin (Vitamin B2) is essential for mitochondrial energy production in neurons. Brain cells with high glutamate activity have enormous energy demands — when mitochondria underperform, neurons become more vulnerable to excitotoxic damage. Riboflavin also supports the enzymes involved in GABA metabolism, including those that clear toxic downstream metabolites. It has well-established evidence for reducing migraine frequency, which is closely tied to glutamate dysregulation. Deficiency quietly worsens neurological resilience across multiple pathways.

Personalized to Your Genes

↑ GRIA1 Well-established support for mitochondrial function in neurons — reduces migraine frequency specifically

Sarcosine

How to implement

Take sarcosine as an oral supplement, typically in powder form. The common dosage ranges from 2 grams to 3 grams per day, usually divided into two equal doses taken in the morning and evening. It is recommended to continue this regimen for at least 4 to 6 weeks to observe effects.

TYPICAL STARTING DOSE

2 g

How it helps

Sarcosine (N-methylglycine) inhibits the glycine transporter GlyT1, which normally recycles glycine away from the synapse. By blocking this reuptake, sarcosine increases the availability of glycine at NMDA receptors — fine-tuning their activity rather than suppressing or over-stimulating them. This is particularly relevant for genes like GRIN2A where NMDA receptor activity is dysregulated rather than simply too high or too low. Sarcosine has been studied in clinical settings for cognitive and mood support, with a mechanism that is uniquely precise for NMDA modulation.

Personalized to Your Genes

GRIN2A — Glycine reuptake inhibitor that fine-tunes NMDA co-agonist availability at the synapse

GABRA2

GABRA2 Report

The *GABRA2* gene encodes the alpha-2 subunit of the GABA-A receptor, the brain's primary fast-acting inhibitory receptor. This receptor is made up of different combinations of five protein subunits. These subunits form a chloride channel that opens upon binding to the neurotransmitter GABA (gamma-aminobutyric acid) to calm neuronal activity [R, R].

GABRA1 is found in roughly 60% of all GABA-A receptors in the brain and is particularly important in the regulation of sleep, anxiety, and seizure threshold. Unlike the more broadly distributed alpha-1 subunit, alpha-2 plays an especially prominent role in the prefrontal cortex and limbic system — areas central to anxiety, impulse control, and addiction [R, R].

Enhancers:

Apigenin

SNP	Your Genotype
rs279858 Alleles **C:** Reduced GABRA2 activity **T:** Increased GABRA2 activity	↓CT **Your genotype is linked to reduced GABRA2 activity and higher odds of addictive behavior**

Intro and Health Effects

By far, the best-characterized *GABRA2* variant is **rs279858**. Its minor 'C' allele may decrease *GABRA2* expression [R].

This variant has been associated with an increased risk of alcohol use disorder. Carriers may also experience more stimulating and enjoyable effects of heavy drinking [R, R, R, R].

It has also been associated with:

- Elevated externalizing problems and rule-breaking behavior during adolescence [R, R]
- Increased susceptibility to parental monitoring [R]

GABRA6

GABRA6 Report

The GABA-A receptor is the primary target of GABA (gamma-aminobutyric acid) in the brain. Genes encoding different parts of that receptor, such as *GABRA6* and *GABRG2*, can influence its function [R].

GABA is the main inhibitory neurotransmitter that prevents the over-stimulation of brain cells. An abnormal GABA activity can produce mental illness and symptoms such as anxiety, depression, insomnia, convulsions, and epilepsy [R, R].

SNP	Your Genotype
rs3219151 Alleles **C:** Increased GABRA6 activity **T:** Reduced GABRA6 activity	↓TT **Your genotype is linked to reduced GABRA6 activity and higher odds of stress and addictions**

Intro and Health Effects

GABRA6 encodes the alpha6 subunit of the GABA-A receptor. The 'T' allele of its rs3219151 polymorphisms, which may impair GABA-A activity, has been associated with an increased risk of [R]:

- Anxiety and impaired stress response [R, R, R]
- Alcohol dependence [R]
- Stress-associated suicide [R]

However, this variant has been associated with a reduced risk of schizophrenia, and the studies testing its effects on epilepsy produced mixed results [R, R, R].

GABRG2

GABRG2 Report

The *GABRG2* gene encodes the gamma-2 subunit of the GABA-A receptor, the brain's primary fast-acting inhibitory receptor. This receptor is made up of different combinations of five protein subunits. These subunits form a chloride channel that opens upon binding to the neurotransmitter GABA (gamma-aminobutyric acid) to calm neuronal activity [R, R].

Truncating mutations in the *GABRG2* gene prevent GABA-A receptors from properly embedding themselves in the neuronal membrane, significantly reducing the brain's inhibitory capacity [R, R].

Importantly, GABRG2 is the subunit through which benzodiazepines bind to the GABA-A receptor to exert their calming effects. This makes the *GABRG2* gene particularly relevant to anxiety treatment [R, R].

Enhancers:

Zinc Magnesium

SNP

rs211037

Alleles

C: Typical GABRG2 activity

T: Reduced GABRG2 activity

Your Genotype

↓TT

Your genotype is linked to reduced GABRG2 activity and higher odds of anxiety and seizures

Intro and Health Effects

The *GABRG2* gene codes for the gamma-aminobutyric acid type A subunit gamma2 (GABRG2) of the GABA-A receptor. The 'T' allele of its rs211037 polymorphism likely impairs GABA-A receptor function and has been associated with an increased risk of [R]:

- Anxiety and impaired stress response [R]
- Seizures [R]
- Benzodiazepine-associated liver encephalopathy [R]

GAD1

GAD1 Report

The *GAD1* gene encodes the 67 kDa isoform of glutamic acid decarboxylase, an enzyme that breaks down the anxiety-inducing neurotransmitter glutamate into GABA (gamma-aminobutyric acid) in the brain [R, R].

GABA is the main inhibitory neurotransmitter that prevents the overstimulation of brain cells. An abnormal GABA activity can produce mental illness and symptoms such as anxiety, depression, insomnia, convulsions, and epilepsy [R, R].

Enhancers:

Zinc

SNP	Your Genotype
rs1978340	AG
Alleles **A:** Reduced GAD1 activity **G:** Increased GAD1 activity	**Your genotype is linked to typical GAD1 activity and GABA production, and typical odds of attention issues and addictions**

Intro and Health Effects

Another variant associated with schizophrenia is the minor 'A' allele at rs1978340. This variant has been shown to reduce GABA levels [R, R, R, R].

This variant has also been linked to:

- Cocaine dependence [R]
- Panic disorder and respiratory symptoms in this condition [R, R]
- Earlier onset of alcohol dependence [R]

SNP

rs3749034

Alleles

A: Increased GAD1 activity

G: Reduced GAD1 activity

Your Genotype

↓GG

Your genotype is linked to reduced GAD1 activity, reduced GABA production, and higher odds of attention issues and addictions

Intro and Health Effects

The most well-researched *GAD1* polymorphism is rs3749034. Its major 'G' allele has been shown to reduce GAD1 activity, resulting in lower GABA levels in the brain. In addition, this variant has been associated with an increased risk of [R]:

- Schizophrenia [R, R, R]
- Hyperactive and impulsive symptoms in ADHD [R]

However, this variant has also been linked to lower odds of bipolar disorder and fewer respiratory symptoms in people with panic disorder [R, R].

GRIA1

GRIA1 Report

The GRIA1 gene encodes the subunit 1 of the AMPA receptor, one of the brain's main receptors for glutamate. This receptor allows signals from this neurotransmitter to pass between brain cells [R].

GRIA1 plays a key role in synaptic plasticity and cognitive flexibility, as well as in memory and learning. In the frontal cortex, GRIA1 expression gradually decreases with age. However, its excessive activity may play a role in glutamate nerve damage (excitotoxicity) [R].

SNP	Your Genotype
rs2195450 Alleles **A:** Increased GRIA1 activity **G:** Reduced GRIA1 activity	↑GA **Your genotype is linked to increased GRIA1 activity, excess glutamate activity, and higher migraine risk**

Intro and Health Effects

One of the main GRIA1 variants is rs2195450. Its minor **'A' allele** may increase GRIA1 levels and has been associated with an **increased risk of migraine**. However, not all studies found this association [R, R, R, R, R, R].

This variant has also been associated with an increased risk of **diabetic retinopathy** in people with type 2 diabetes [R].

SLC1A2

SLC1A2 Report

Enhancers:

Vitamin C

N-acetylcysteine (NAC)

SNP	Your Genotype
rs4354668 Alleles **G:** Reduced SLC1A2 activity **T:** Increased SLC1A2 activity	↓ GG **Your genotype is linked to reduced SLC1A2 activity, and higher odds of mood and nerve disorders**

Intro and Health Effects

One of the main *SLC1A2* variants is **rs4354668**. Its minor 'G' allele may decrease *SLC1A2* expression by ~30% and has been associated with an increased risk of [R]:

- Major depressive disorder [R]
- Schizophrenia [R, R]
- Harmful drug use [R]
- Bipolar disorder, especially in response to adverse childhood experiences [R, R, R]
- Cerebral palsy [R]

Moreover, carriers of this allele with schizophrenia may have worse cognitive ability and respond worse to cognitive remediation therapy [R, R, R].

SNP	Your Genotype
rs3794087 Alleles **G:** Increased SLC1A2 activity **T:** Reduced SLC1A2 activity	○ GT **Your genotype is linked to typical SLC1A2 activity, and typical odds of mood and nerve disorders**

Intro and Health Effects

Another well-researched variant is **rs3794087**. Its minor ‘T’ allele has been associated with an approximately 40% increased risk of essential tremor in Chinese, Taiwanese, and German studies. However, this association couldn’t be replicated in other studies [R, R, R, R, R, R, R, R].

SLC1A3

SLC1A3 Report

The SLC1A3 gene encodes the 'excitatory amino acid transporter 1' (EAAT1), also known as 'solute carrier family 1 member 3'. EAAT1 is a close partner to EAAT2 (encoded by SLC1A2) in the brain's glutamate clearance system. EAAT1 plays a particularly important role in the cerebellum and in early brain development, where precise glutamate regulation is critical for circuit formation [R, R].

EAAT1 is found mainly on the astrocytes and helps protect neurons from excessive glutamate exposure (excitotoxicity) [R, R].

Enhancers:

Vitamin C

SNP	Your Genotype
rs2269272 Alleles **C:** Increased SLC1A3 activity **T:** Reduced SLC1A3 activity	↓CT **Your genotype is linked to reduced SLC1A3 activity, impaired protection from excess glutamate, and issues with stress, mood & attention**

Intro and Health Effects

One of the main SLC1A3 variants is rs2269272. Its minor 'T' allele may decrease gene expression and has been associated with an increased risk of:

- Stress [R]
- Depression [R]
- ADHD [R]

On the bright side, this allele has been associated with decreased suicidal behavior [R].

ABAT

ABAT Report

The *ABAT* gene encodes an enzyme called '*4-aminobutyrate aminotransferase*' or 'GABA-transaminase'. This enzyme breaks down the neurotransmitter gamma-aminobutyric acid (GABA) when it's no longer needed [R, R].

Common variants decreasing gene expression have been associated with Alzheimer's disease [R].

Enhancers:

Vitamin B6 (Pyridoxin)

SNP	Your Genotype
rs2270288	TG
Alleles	
G: Typical ABAT activity	
T: Reduced ABAT activity	

Your genotype is linked to typical ABAT activity, typical GABA metabolism and cognitive health

Intro and Health Effects

A study of over 400k participants identified several *ABAT* variants associated with an increased risk of Alzheimer's disease, the main ones being 'T' of **rs2270288** and 'A' of **rs3743798**. These variants were shown to reduce *ABAT* expression [R].

The association of the first variant with Alzheimer's disease was replicated in another study of almost 500k participants [R]. These variants are usually inherited together, so you will either have both or neither of them.

ALDH5A1

ALDH5A1 Report

The ALDH5A1 gene encodes succinic semialdehyde dehydrogenase (SSADH), a mitochondrial enzyme that plays a critical role in the breakdown of GABA — the brain's primary inhibitory neurotransmitter. While glutamate accelerates neuronal activity, GABA does the opposite: it quiets signals down and prevents the brain from becoming overloaded.

After GABA is released into a synapse and recaptured by nearby cells, it is degraded through a two-step process. The first step produces a toxic intermediate called succinic semialdehyde (SSA). SSADH handles the second step, converting SSA into succinate — a harmless molecule fed into the cell's energy-producing Krebs cycle. Without working SSADH, SSA accumulates and is diverted into gamma-hydroxybutyrate (GHB), a neuroactive substance that further disrupts the brain's excitatory-inhibitory balance.

Beyond GABA metabolism, emerging research suggests SSADH also contributes to antioxidant defence in neuronal mitochondria, making it relevant to both neurotransmission and cellular stress resilience.

Enhancers:

Vitamin B3 (Niacin)

SNP	Your Genotype
rs2760118	CT
Alleles	
C: Typical ALDH5A1 activity	
T: Reduced ALDH5A1 activity	

Your genotype is linked to typical ALDH5A1 activity, typical clearance of toxic metabolites, and cognitive health

Intro and Health Effects

An ALDH5A1 variant **rs2760118-T** swaps a single amino acid in the SSADH enzyme. On its own, this causes a modest **~17% reduction in enzyme activity**, generally considered subclinical. However, research shows it becomes significantly more consequential in two contexts:

- When it co-occurs on the same chromosome **with other ALDH5A1 variants**, it can compound their effects and push enzyme activity into a problematic range
- **When the brain is under oxidative stress**, cells carrying this variant show worse mitochondrial resilience than those with the normal version.

A study in brain cells found that cells expressing a combination of common ALDH5A1 SNPs, including rs2760118, had significantly reduced SSADH activity, greater **lipid peroxidation, and disrupted mitochondrial membrane potential when**

exposed to oxidative stress — effects not seen in cells with the normal enzyme. This points to a vulnerability in the brain's GABA-processing machinery under neurological stress conditions.

Additionally, rs2760118 may be a **modifier variant in confirmed SSADH-deficiency cases** — amplifying the effects of nearby mutations that would otherwise be silent. Its relatively high population frequency (~35%) means a significant portion of the general population carries at least one copy, potentially with subtle effects on GABA clearance and mitochondrial stress response.

GABRB2

GABRB2 Report

The *GABRB2* gene encodes the beta-2 subunit of the heteropentameric receptor for the neurotransmitter GABA (gamma-aminobutyric acid). The beta subunit forms part of the core channel through which inhibitory signals flow, making it indispensable for GABA-A receptor function [R, R].

GABRB2 is widely expressed throughout the brain and plays important roles in maintaining the excitation-inhibition balance underlying cognition and emotional stability [R].

SNP

rs1816071

Alleles

C: Reduced GABRB2 activity

T: Typical GABRB2 activity

Your Genotype

TT

Your genotype is linked to typical GABRB2 activity and typical odds of psychotic disorders

Intro and Health Effects

Another major variant is **rs1816071.** Its 'C' allele has been associated with an increased risk of schizophrenia, but less severe positive symptoms in people with this disease [R, R, R].

SNP

rs1816072

Alleles

A: Typical GABRB2 activity

G: Reduced GABRB2 activity

Your Genotype

AA

Your genotype is linked to typical GABRB2 activity and typical odds of psychotic disorders

Intro and Health Effects

One of the main *GABRB2* variants is **rs1816072**. Its minor 'G' allele may reduce *GABRB2* expression [R, R].

This variant has been associated with an increased risk of schizophrenia in Asians, but a decreased risk in Caucasians. [R, R].

GABRB3

GABRB3 Report

The *GABRB3* gene encodes the beta-3 subunit of the heteropentameric receptor for the neurotransmitter GABA (gamma-aminobutyric acid) [R].

The beta-3 subunit is expressed at different levels within the cerebral cortex, hippocampus, cerebellum, thalamus, olivary body, and piriform cortex of the brain at different points of development. During brain development, the function of the beta-3 subunit in the GABAA receptor allows for the proliferation, migration, and differentiation of precursor cells [R, R].

SNP	Your Genotype
rs2081648 Alleles **C:** Increased GABRB3 activity **T:** Reduced GABRB3 activity	TC **Your genotype is linked to typical GABRB3 activity and typical odds of cognitive & mood disorders**

Intro and Health Effects

Another variant, the major ‘T’ allele of **rs2081648**, has been associated with:

- Increased risk and severity of autism spectrum disorder [R, R, R]
- Increased risk of ADHD [R]

However, not all studies found an association between this variant and autism spectrum disorders [R, R].

Based on the phenotypes, the ‘T’ allele of rs2081648 may reduce GABRB3 activity.

SNP	Your Genotype
rs4906902 Alleles **A:** Increased GABRB3 activity **G:** Reduced GABRB3 activity	AG **Your genotype is linked to typical GABRB3 activity and typical odds of cognitive & mood disorders**

Intro and Health Effects

The best-researched *GABRB3* SNP is **rs4906902**. Its minor 'G' allele reduces *GABRB3* gene expression and has been associated with [R, R]:

- Increased risk of autism spectrum disorders [R]
- Increased risk of heroin dependence [R]
- Increased risk of depression in patients with epilepsy [R]
- Increased risk of schizophrenia (as part of a haplotype) [R]
- Decreased risk of fibromyalgia [R]

GAD2

GAD2 Report

The *GAD2* gene encodes the GAD65 form of the glutamate decarboxylase enzyme, which converts the amino acid and excitatory neurotransmitter glutamate into the calming neurotransmitter gamma-aminobutyric acid (GABA) using pyridoxine (vitamin B6) as a cofactor [R, R].

Because it simultaneously reduces glutamate levels while increasing GABA production, GAD2 is a critical junction point in the regulation of GABA/glutamate balance. While GAD67 (encoded by *GAD1*) maintains baseline GABA levels, GAD65 is responsible for on-demand, synaptic GABA production [R].

SNP

rs992990

Alleles

A: Increased GAD2 activity

C: Reduced GAD2 activity

Your Genotype

↑AA

Your genotype is linked to increased GAD2 activity and better mental health, but worse weight control

Intro and Health Effects

Another variant, the 'A' allele of **rs992990**, has been associated with a **decreased risk of OCD**. Interestingly, this allele has been associated with increased hunger, weight gain, and obesity risk. [R, R, R].

SNP

rs2236418

Alleles

A: Reduced GAD2 activity

G: Increased GAD2 activity

Your Genotype

↑GG

Your genotype is linked to increased GAD2 activity and better mental health, but worse weight control

Intro and Health Effects

One of the main *GAD2* variants is **rs2236418.** Its minor 'G' allele may increase gene expression by 6-fold. This variant has been associated with an increased risk of obesity. However, carriers may have decreased risk of type 2 diabetes and lower blood glucose levels [R, R, R, R, R, R].

GLS

GLS Report

The GLS gene encodes glutaminase, a mitochondrial enzyme with a central role in brain chemistry. Its main job is deceptively simple: converting glutamine into glutamate, the brain's principal excitatory neurotransmitter. Without this conversion, neurons could not maintain the constant supply of glutamate needed to fire and communicate.

GLS sits at the heart of the glutamate–glutamine cycle — a continuous loop between neurons and their neighbouring support cells (astrocytes). After a neuron releases glutamate into a synapse, astrocytes capture it and convert it back to glutamine for safe storage. That glutamine is then shuttled back to the neuron, where GLS converts it to glutamate once more, ready for the next signal.

Because glutamate is both essential for cognition and toxic in excess, keeping this cycle balanced is critical.

SNP

rs12185688

Alleles

C: Typical GLS activity

T: Altered GLS activity

Your Genotype

TT

Your genotype is linked to altered GLS activity, altered glutamine/glutamate ratio, and unknown effects on mental health

Intro and Health Effects

Two GLS variants, **rs13000464-C and rs12185688-T**, form part of a haplotype — a group of variants — associated with a **higher glutamine-to-glutamate ratio (Gln/Glu)** in the brain region involved in sensory integration and attention.

In the glutamate–glutamine cycle, a higher Gln/Glu ratio is a sign that the cycle is running harder than usual — more glutamine is accumulating than neurons are converting back to glutamate, which reflects underlying glutamatergic overactivity. This pattern has been observed in people with bipolar disorder during manic episodes and, to a lesser degree, in schizophrenia.

Importantly, this association was seen across healthy controls as well as people with psychiatric diagnoses, suggesting these variants influence a general neurochemical trait — a baseline difference in how the glutamate cycle operates — rather than directly causing illness.

SNP

rs13000464

Alleles

C: Altered GLS activity

T: Typical GLS activity

Your Genotype

TT

Your genotype is linked to typical GLS activity, typical glutamine/glutamate ratio, and mental health

Intro and Health Effects

Two GLS variants, **rs13000464-C and rs12185688-T**, form part of a haplotype — a group of variants — associated with a **higher glutamine-to-glutamate ratio (Gln/Glu)** in the brain region involved in sensory integration and attention.

In the glutamate–glutamine cycle, a higher Gln/Glu ratio is a sign that the cycle is running harder than usual — more glutamine is accumulating than neurons are converting back to glutamate, which reflects underlying glutamatergic overactivity. This pattern has been observed in people with bipolar disorder during manic episodes and, to a lesser degree, in schizophrenia.

Importantly, this association was seen across healthy controls as well as people with psychiatric diagnoses, suggesting these variants influence a general neurochemical trait — a baseline difference in how the glutamate cycle operates — rather than directly causing illness.

GRIA3

GRIA3 Report

The GRIA3 gene encodes a receptor for the neurotransmitter glutamate called ‘glutamate ionotropic receptor AMPA type subunit 3’ [R].

Glutamate is an important excitatory neurotransmitter in the brain. Moreover, it helps make another chemical messenger, GABA, with calming effects. Glutamate also helps make the main body’s antioxidant glutathione [R, R, R].

Glutamate receptors are heteromeric protein complexes composed of multiple subunits. The subunit encoded by this gene belongs to a family of AMPA (alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionate)-sensitive glutamate receptors, which are crucial for synaptic transmission and brain plasticity [R].

SNP	Your Genotype
rs687577	C
Alleles **A:** Reduced GRIA3 activity **C:** Typical GRIA3 activity	**Your genotype is linked to typical GRIA3 activity, typical glutamate activity, and mood**

Intro and Health Effects

The minor **‘A’ allele** of the rs687577 polymorphism has been associated with a **decreased risk of depression** in women and with longer sleep duration. Given the role of glutamate as an excitatory neurotransmitter, this variant may reduce GRIA3 activity [R, R].

GRIN2A

GRIN2A Report

The GRIN2A gene encodes a protein called GluN2A, which is a subunit of the glutamate receptor N-methyl-D-aspartate (NMDA). Upon binding to glutamate, the NMDA receptor opens and allows the flow of calcium ion currents to activate brain cells. The NMDA receptor is involved in brain development, brain plasticity, learning, and memory, among others [R, R].

The GluN2A subunit determines where in the brain the NMDA receptor is located and how it functions. Moreover, it's the subunit to which glutamate binds [R].

SNP

rs11644461

Alleles

C: Altered GRIN2A activity

T: Normal GRIN2A activity

Your Genotype

TT

Your type is linked to normal GRIN2A activity, balanced glutamate activity and lower odds of psychotic disorders

Intro and Health Effects

One of the main GRIN2A variants is rs11644461. Its minor **'C' allele** has been associated with an **increased risk** of early-onset and continuous-type schizophrenia [R, R, R].

SNP

rs7206256

Alleles

A: Altered GRIN2A activity

G: Normal GRIN2A activity

Your Genotype

AA

Your genotype is linked to altered GRIN2A activity, altered glutamate activity, and higher odds of psychotic disorders

Intro and Health Effects

The minor **'G' allele** of rs7206256 has been associated with a **reduced risk** of early-onset schizophrenia [R, R].

GRIN2B

GRIN2B Report

The GRIN2B gene encodes a protein called GluN2B, which is a subunit of the glutamate receptor N-methyl-D-aspartate (NMDA). Upon binding to glutamate, the NMDA receptor opens and allows the flow of calcium ion currents to activate brain cells. The NMDA receptor is involved in brain development, brain plasticity, learning, and memory, among others [R, R].

While GRIN2A dominates in the adult brain, GRIN2B is particularly important during early brain development, where it helps shape how neural circuits are formed and refined. It plays a central role in synaptic plasticity, learning, and emotional regulation [R].

SNP	Your Genotype
rs1019385 Alleles **A:** Increased GRIN2B activity **C:** Reduced GRIN2B activity	↑AA **Your genotype is linked to increased GRIN2B activity, worse cognition, and higher odds of OCD**

Intro and Health Effects

One of the main GRIN2B variants is rs1019385. Its 'C' allele may reduce gene expression and glutamate levels in the anterior cingulate cortex [R, R].

This allele has been associated with:

- Reduced risk of OCD [R]
- Better response to rTMS and antidepressants (SSRIs) in OCD patients [R]
- Higher educational attainment [R]

SNP	Your Genotype
rs1805476 Alleles **G:** Reduced GRIN2B activity **T:** Increased GRIN2B activity	↓GG **Your genotype is linked to reduced GRIN2B activity, better cognition, and lower odds of OCD**

Intro and Health Effects

The **'T' allele** of rs1805476 has been associated with higher glutamate levels in the anterior cingulate cortex [R].

This allele has been associated with an **increased risk** of:

- Tourette syndrome [R]
- Contamination obsessions and cleaning compulsions in male OCD patients [R]

GRM3

GRM3 Report

The *GRM3* gene encodes a glutamate receptor called '*glutamate metabotropic receptor 3*' or mGluR3. This receptor acts as a fine-tuner of glutamate signaling to control how much glutamate is released and cleared from sending neurons and astrocytes [R, R].

GluR3 is expressed in the prefrontal cortex — the brain region responsible for superior cognitive functions such as decision-making, working memory, and emotional regulation [R, R].

SNP

rs12704290

Alleles

A: Increased GRM3 activity

G: Reduced GRM3 activity

Your Genotype

↓ GG

Your genotype is linked to reduced GRM3 activity, and higher odds of addictions and cognitive issues

Intro and Health Effects

Finally, the 'G' allele of **rs12704290** has been associated with an increased risk of schizophrenia in several studies [R, R, R, R, R, R].

SNP

rs6465084

Alleles

A: Reduced GRM3 activity

G: Increased GRM3 activity

Your Genotype

↓ AA

Your genotype is linked to reduced GRM3 activity, and higher odds of addictions and cognitive issues

Intro and Health Effects

The 'A' allele of **rs6465084** has been associated with reduced prefrontal cortex efficiency, suggesting impaired glutamate signaling regulation. This variant has been associated with an increased risk and severity of [R]:

- Schizophrenia [R, R]
- Alcohol dependence (and worse executive function in people with this condition) [R, R]

- Psychosis in people with bipolar disorder [R, R]

On the bright side, this variant has been associated with:

- Better response to antipsychotics in schizophrenia [R]
- Decreased risk of major depressive disorder [R]

SNP

rs274622

Alleles

C: Increased GRM3 activity

T: Reduced GRM3 activity

Your Genotype

TC

Your genotype is linked to typical GRM3 activity, and typical odds of addictions and cognitive issues

Intro and Health Effects

The ‘T’ allele of **rs274622** has also been associated with reduced prefrontal cortex efficiency, as well as with [R]:

- Increased risk of schizophrenia (in non-Asians) [R, R]
- Worse executive function in alcoholics [R]
- Greater loss of working memory in response to antipsychotics [R]

GRM7

GRM7 Report

The *GRM7* gene encodes a glutamate receptor called '*glutamate metabotropic receptor 7*' or mGluR7. This receptor, found at the junction of neurons, has a lower affinity for glutamate than others and acts primarily as a brake on glutamate release. When the levels of this neurotransmitter get too high, mGluR7 is activated to prevent neuron overstimulation — a process called excitotoxicity [R, R].

GRM7 is highly expressed in brain regions involved in fear, anxiety, and stress responses, such as the amygdala and hippocampus. Mice lacking this gene show alterations in behavior, learning, movement, and sleep [R, R].

Enhancers:

N-acetylcysteine (NAC)

SNP	Your Genotype
rs3749380 Alleles **C:** Increased GRM7 activity **T:** Reduced GRM7 activity	↓ TT **Your genotype is linked to reduced GRM7 activity, and higher odds of mood and cognitive disorders**

Intro and Health Effects

One of the main *GRM7* variants is **rs3749380**. Its minor 'T' allele has been associated with decreased *GRM7* expression and higher glutamate levels, as well as with an increased risk of [R, R]:

- Schizophrenia [R]
- ADHD [R]
- Alcohol consumption [R].

SNP	Your Genotype
rs779867 Alleles **C:** Reduced GRM7 activity **T:** Increased GRM7 activity	o CT **Your genotype is linked to typical GRM7 activity, and typical odds of mood and cognitive disorders**

Intro and Health Effects

Another well-researched variant, the major ‘C’ allele of **rs779867**, has been associated with an increased risk of:

- Schizophrenia [R]
- Autism spectrum disorder [R]
- Multiple sclerosis [R]
- ADHD [R]
- Major depressive disorder [R]

SLC6A1

SLC6A1 Report

The *SLC6A1* gene encodes GAT-1, the brain's primary transporter for the reuptake of the neurotransmitter gamma-aminobutyric acid (GABA). By doing so, GAT-1 terminates the inhibitory signals in response to GABA and recycles this neurotransmitter for future use [R, R].

SNP

rs2697153

Alleles

A: Reduced SLC6A1 activity

G: Increased SLC6A1 activity

Your Genotype

AG

Your genotype is linked to typical SLC6A1 activity, typical GABA reuptake and anxiety risk

Intro and Health Effects

One of the main SLC6A1 variants is rs2697153. Its minor 'G' allele has been associated with an increased risk of anxiety disorders, but also with a better response to kava. Based on these effects, the allele may increase GAT-1 activity [R, R].

CACNA1C

CACNA1C Report

CACNA1C codes for part of a calcium channel (Cav1.2) in the brain. These calcium channels play a role in plasticity -- your brain's ability to change and adapt [R].

This signaling plays an essential role in synaptic plasticity, neurotransmitter release, and neuronal excitability—all processes linked to cognitive function and attention regulation [R].

Because these functions are so critical, even small changes in how this gene works can have a big impact on brain health and mental well-being.

SNP	Your Genotype
rs1006737 Alleles **A:** Altered CACNA1C activity **G:** Normal CACNA1C activity	GG **Your genotype is linked to normal CACNA1C activity, good GABA/glutamate balance, and lower odds of psychotic disorders and anxiety**

Intro and Health Effects

The main CACNA1C variant is **rs1006737**. Its **"A" allele** is linked to [R, R, R]:

- Schizophrenia
- Bipolar disorder
- Tense, worried, and anxious feelings

This variant generally **increases CACNA1C expression** and may enhance calcium channel activity. However, the downstream effects on brain function depend on the balance between beneficial and harmful consequences of altered calcium signaling.

GABRA1

GABRA1 Report

The *GABRA1* gene encodes the alpha-1 subunit of the GABA-A receptor, the brain's primary fast-acting inhibitory receptor. This receptor is made up of different combinations of five protein subunits. These subunits form a chloride channel that opens upon binding to the neurotransmitter GABA (gamma-aminobutyric acid) to calm neuronal activity [R, R].

GABRA1 is found in roughly 60% of all GABA-A receptors in the brain and is particularly important in the regulation of sleep, anxiety, and seizure threshold [R, R].

Enhancers:

Magnesium Apigenin

SNP

rs2279020

Alleles

A: Increased GABRA1 activity

G: Reduced GABRA1 activity

Your Genotype

↑AA

Your genotype is linked to increased GABRA1 activity, and lower odds of mood issues and seizures

Intro and Health Effects

By far, the main *GABRA1* variant is **rs2279020.** Its minor 'G' allele has been associated with an increased risk of idiopathic generalized epilepsy and treatment resistance. However, the variant may not be associated with epilepsy in all ethnicities [R, R, R, R, R, R, R, R].

This allele has also been associated with an increased risk of depression [R].

Based on its effects, this variant may reduce *GABRA1* expression on the surface of brain cells [R].

GLUL

GLUL Report

The GLUL gene encodes an enzyme called ‘glutamate-ammonia ligase’ or ‘glutamine synthetase’ that catalyzes the synthesis of the amino acid glutamine by combining glutamate and ammonia [R].

By doing so, it plays key roles in [R]:

- Ammonia and glutamate detoxification
- Acid-base homeostasis
- Cell signaling
- Cell proliferation

Enhancers:

Magnesium Zinc

SNP

rs10911021

Alleles

C: Reduced GLUL activity

T: Increased GLUL activity

Your Genotype

↑TC

Your genotype is linked to increased GLUL activity, enhanced clearance of ammonia and glutamate, and better metabolic health

Intro and Health Effects

The main GLUL variant is rs10911021. Its minor ‘T’ allele has been associated with a **decreased risk** and mortality of coronary heart disease in patients with type 2 diabetes. Moreover, this variant has been associated with [R, R, R, R, R, R]:

- Better blood lipid profile (lower LDL and lipoprotein(a), but higher HDL cholesterol levels)
- Better antioxidant status (lower MDA and GSSG and higher GSH levels)

This variant has been associated with **higher GLUL expression**, resulting in enhanced ammonia and glutamate detoxification. The authors of the study suggested that supplementation with glutamine may help prevent coronary heart disease in carriers of the ‘C’ allele [R].

SLC6A15

SLC6A15 Report

The SLC6A15 gene encodes a member of the solute carrier family 6 protein family that transports branched-chain amino acids and neurotransmitters into brain cells, which aids their healthy development and function [R, R, R].

By affecting glutamate neurotransmission, this transporter regulates emotional behavior. Variants in this gene have been associated with psychiatric disorders such as major depression [R, R, R].

Research in animals suggests this transporter may also influence food intake and weight gain [R].

SNP	Your Genotype
rs1545843 Alleles **A:** Reduced SLC6A15 activity **G:** Increased SLC6A15 activity	↑GG **Your genotype is linked to increased SLC6A15 activity, improved transport of BCAAs and neurotransmitters, and better mood**

Intro and Health Effects

The main SLC6A15 variant is rs1545843. Its **'A' allele** has been associated with reduced SLC6A15 levels and impaired nerve connections in the brain. This variant has been associated with an **increased risk of depression**. Patients with depression may also have impaired memory and sustained attention performance, and enhanced responses to ACTH and cortisol is they carry the "AA" variant [R, R, R, R, R].

Lab markers to check

Glucose, Fasting — 97 mg/dL — 9 Nov 2025

Homocysteine — 12.84 umol/L — 9 Nov 2025

Personalized to Your Genes

↑ GRIA1	Elevated homocysteine potentiates glutamate receptor excitotoxicity and migraine risk
↓ SLC1A2	Glutamate excitotoxicity and vascular inflammation interact — both worsen mental health outcomes here
↓ SLC1A3	Glutamate excitotoxicity and neuroinflammation are tightly linked when this transporter is underactive
○ GRM3	Elevated homocysteine disrupts glutamate receptor signaling and worsens mental health outcomes

Insulin, Fasting — 8.7 uIU/mL — 13 Jan 2026

TSH — 2.64 mIU/L — 4 May 2026

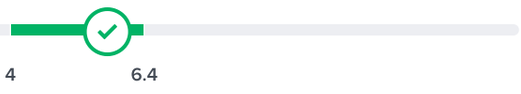
Magnesium — 2.17 mg/dL — 9 Nov 2025

Personalized to Your Genes

↓ GABRA2	Deficiency amplifies anxiety and excitability when the primary anxiolytic GABA-A subunit is underperforming

↓ GABRA6	Deficiency compounds inhibitory signaling deficits when α6-mediated cerebellar GABA-A activity is already low
↓ GABRG2	γ2 subunit is critical for receptor synaptic localization — magnesium deficiency worsens the functional gap
↑ GRIA1	Magnesium deficiency directly amplifies AMPA overactivity from this variant
↓ SLC1A2	Determines how effectively NMDA receptors can be buffered when clearance is compromised
↓ SLC1A3	Intracellular magnesium status determines how well NMDA receptors can be buffered
○ GRIN2A	Direct NMDA modulator — deficiency will worsen dysregulated signaling from this variant
○ GRIN2B	The primary buffer for this variant's overactive NMDA signaling — must be optimized

Magnesium, RBC 5.7 mg/dL 13 Jan 2026



Personalized to Your Genes

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T3, Free (FT3)	3.2 pg/mL	2.3 – 3 – 4.7	13 Jan 2026
T4 Free (FT4)	11.4 pmol/L	10.3 – 20.6	4 May 2026
Uric Acid	5.3 mg/dL	2 – 3.7 – 4 – 6 – 9	13 Jan 2026
Vitamin B6	55 ng/mL	5 – 8.15 – 115.4	13 Jan 2026

Personalized to Your Genes

↓ GAD1	The rate-limiting cofactor for GAD1 — deficiency drastically worsens the impact of this variant

Vitamin D, 25-Hydroxy, Total	55 ng/mL	10 – 20 – 30 – 75 – 150	13 Jan 2026
Zinc	17.12 umol/L	7.65 – 9.18 – 12.24 – 19.89	9 Nov 2025

Personalized to Your Genes

↓ GABRG2	With γ2 reduced, zinc modulation of remaining GABA-A receptors shifts — monitoring levels is essential
GRIN2A	Direct NMDA modulator — deficiency will worsen dysregulated signaling from this variant
GRIN2B	Zinc deficiency removes a key endogenous brake on the overactive receptor

- 4-Hydroxybutyric (Mosaic Diagnostics)
- 5-HIAA, Plasma
- 8-Hydroxy-2-deoxyguanosine, Urine
- a-Ketoglutarate (Genova)
- Ammonia
- C-Reactive Protein (CRP)

Personalized to Your Genes

↓ SLC1A2	Glutamate excitotoxicity and vascular inflammation interact — both worsen mental health outcomes here
↓ SLC1A3	Glutamate excitotoxicity and neuroinflammation are tightly linked when this transporter is underactive
GRIN2A	Inflammation shifts NMDA receptor tone and amplifies mental health effects of this variant
GRM3	Neuroinflammation and impaired GRM3 signaling mutually worsen each other
GRM7	GRM7 has anti-inflammatory roles; its loss compounds inflammatory signaling in the brain

- Coenzyme Q10

Personalized to Your Genes

↑ GRIA1	Deficiency worsens neuronal energy crisis triggered by receptor overactivation

Cortisol

Personalized to Your Genes

↓ GABRA2	α2 subunit mediates anxiolytic effects — its underactivity makes stress hormone dysregulation more likely
↓ GABRA6	Stress-driven excitability is poorly buffered when cerebellar inhibitory circuits are underactive
○ GRM7	Stress-driven cortisol spikes amplify glutamate dysregulation when GRM7 control is impaired

Gamma-Aminobutyric Acid (GABA), Plasma

Personalized to Your Genes

↓ GABRA2	Baseline GABA availability determines how much functional signaling is possible through already reduced receptors
↓ GABRA6	Baseline GABA levels determine how much the reduced receptor population can be meaningfully activated
↓ GABRG2	Reflects whether low receptor activity is compounded by low substrate availability
↓ GAD1	Direct readout of whether production is functionally impaired by this variant

Glutamine, Plasma

Personalized to Your Genes

↓ GAD1	Imbalance here reflects impaired conversion toward GABA and is a functional marker of GAD1 underactivity

Glycine, Plasma

Personalized to Your Genes

GRIN2A	NMDA co-agonist availability — critical to assess when receptor function is genetically altered

HOMA-IR

IL-6

Personalized to Your Genes

GRIN2A	Inflammation shifts NMDA receptor tone and amplifies mental health effects of this variant
GRM3	Neuroinflammation and impaired GRM3 signaling mutually worsen each other
GRM7	GRM7 has anti-inflammatory roles; its loss compounds inflammatory signaling in the brain

Isoleucine, Plasma

Leucine, Plasma

Malondialdehyde

Serine, Plasma

Personalized to Your Genes

GRIN2A — NMDA co-agonist availability — critical to assess when receptor function is genetically altered

Succinic Acid

TNF-alpha

Personalized to Your Genes

GRIN2A — Inflammation shifts NMDA receptor tone and amplifies mental health effects of this variant

GRM7 — GRM7 has anti-inflammatory roles; its loss compounds inflammatory signaling in the brain

Valine, Plasma

Glossary

α-ketoglutarate
A key compound in the Krebs cycle that connects energy production with glutamate metabolism.

Apigenin
A plant compound found in herbs like chamomile that may support calmness by influencing GABA activity.

BCAAs (Branched-Chain Amino Acids)
Essential amino acids that can influence brain chemistry and compete with other amino acids like tryptophan and glutamine.

CRP (C-Reactive Protein)
A marker of inflammation in the body that can influence neurotransmitter balance, including glutamate activity.

GABA (Gamma-Aminobutyric Acid)
The brain's main calming neurotransmitter, helping reduce stress, anxiety, and excessive neural activity.

Glutamate
The brain's main excitatory neurotransmitter, important for learning and memory but harmful in excess.

Glutamine (Blood)
An amino acid circulating in the bloodstream that can be transported into the brain and converted into glutamate.

Glutamine (Brain)
A storage and transport form of glutamate in the brain, helping regulate neurotransmitter balance.

Mitochondrial Krebs Cycle
A series of energy-producing reactions in mitochondria that also support neurotransmitter metabolism.

NAC (N-Acetylcysteine)
A compound that supports antioxidant defenses and helps regulate glutamate levels in the brain.

Neurotransmitters
Chemical messengers that allow neurons to communicate, such as GABA and glutamate.

Niacin (Vitamin B3)
A vitamin that supports energy production and helps regulate neurotransmitter balance.

Succinate
A metabolite in the Krebs cycle formed during GABA breakdown, contributing to cellular energy production.

Succinic Semialdehyde
An intermediate compound formed when GABA is broken down before entering the energy cycle.

Taurine
An amino acid that supports calming neurotransmission and may enhance GABA activity.

Toxic Metabolites
Harmful byproducts that can build up when neurotransmitter breakdown is impaired, potentially damaging brain cells.