MOSAIC DIAGNOSTICS

DNA Signature Spectrum Map

GENETICS REPORT

1	7	137
Report	Report Panels	Gene Variants

Example Client

Prepared for

Order ID: _____

DOB: _____

August 26, 2026

Report Date

Practitioner: Erika Gray

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Result Scores Summary

Overview of all 29 engine results across this section.

2	9	18
NEEDS ATTENTION	MODERATE	OPTIMAL

Needs Attention (2)

OMEGA-3 NEED	Significantly increased	Nutrition
MAGNESIUM DEFICIENCY RISK	Significantly increased	Nutrition

Moderate (9)

ADHD PROPENSITY	Increased	Neurodevelopment
METHYLATION ABILITY	Undermethylation potential	Methylation
MTHFR ACTIVITY	60-70% enzyme activity	Methylation
MTR	Typical activity	Methylation
CYP2C19	Intermediate metabolism	Detoxification
GLUCURONIDATION	Reduced function	Detoxification
GENERAL HISTAMINE SENSITIVITY	Increased	Immune & Inflammation
FOLIC ACID SUPPLEMENTATION TOLERANCE	Decreased	Nutrition
REPORTED SLEEP QUALITY	Below average	Sleep

Optimal (18)

ATTENTION	Typical	Neurodevelopment
MTRR	Increased activity	Methylation
COMT	Typical activity	Methylation

CYP1A1	Rapid metabolism	Detoxification
CYP1A2	Increased	Detoxification
CYP2B6	Normal	Detoxification
CYP2C9	Normal metabolism	Detoxification
PON1	Typical activity	Detoxification
CYP2D6	Increased	Detoxification
CYP2E1	Normal metabolism	Detoxification
IGE LEVELS	Decreased	Immune & Inflammation
FOOD-BASED HISTAMINE SENSITIVITY	Typical	Immune & Inflammation
VITAMIN B6 LEVEL PROPENSITY	Typical	Nutrition
VITAMIN B9 NEED	Typical	Nutrition
VITAMIN B12 LEVEL	Typical	Nutrition
GLUCOSE METABOLISM	Typical	Nutrition
OMEGA-6 RISK	Typical	Nutrition
SLEEP DEPRIVATION SENSITIVITY	Typical	Sleep

Our genetic test provides insights into how your unique genetic profile influences health areas. By understanding these genetic tendencies, you and your healthcare provider can make more informed lifestyle and wellness choices to support long-term health goals.

THESE GENE RESULTS MIGHT TELL YOU

- ✓ How your body may respond to certain nutrients
- ✓ Dietary patterns and environmental factors
- ✓ Potential predispositions in detoxification, metabolism, and physical performance
- ✓ Why some wellness strategies may work differently for you

THESE GENE RESULTS WILL NOT TELL YOU

- ✕ Any medical diagnoses
- ✕ Cancer risk assessments
- ✕ False positives
- ✕ Predictions about future health conditions
- ✕ How lifestyle factors outside of genetics impact health outcomes

This commentary is designed solely to support the practitioner's clinical evaluation, as all diagnostic interpretations, treatment plans, and medical management decisions remain entirely under the practitioner's independent authority. The findings in this report outline genetic predispositions for informational purposes only and do not constitute diagnostic or treatment recommendations, guarantee health outcomes, or replace direct clinical consultation. These statements have not been evaluated by the Food and Drug Administration, and our tests are not intended to diagnose, treat, cure, or prevent any disease. Patients must be advised not to initiate or modify any diet, exercise, medication, or lifestyle regimen without consultation with a licensed healthcare provider.

1 Neurodevelopment

9 variants analyzed

This section delves into the complex interplay of genetic factors that contribute to the risk of developing Attention-Deficit/Hyperactivity Disorder (ADHD), a neurodevelopmental condition characterized by patterns of inattention, hyperactivity, and impulsivity. This analysis is aimed at uncovering the genetic predispositions that may influence the likelihood of an individual being diagnosed with ADHD. Research has identified multiple genes associated with an increased risk of ADHD, such as variants in the ANKK1 (dopamine receptor) gene, the COMT gene, and other genes that affect neurotransmitter synthesis, transport, and signaling. These genes are crucial for regulating attention, behavior, and emotional response.

ADHD PROPENSITY

YOUR RESULTS

INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals identified with an increased ADHD genetic risk have a higher likelihood of developing ADHD. Their genetic profile suggests a greater susceptibility to ADHD symptoms, including difficulties with focus, hyperactivity, and impulsiveness, compared to those with a typical risk

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Phosphatidylserine (PS)

200-300 mg/day

PS is the most evidence-based supplement for ADHD in children, with 2 double-blind RCTs showing significant improvement in ADHD rating scales. Supports prefrontal dopaminergic signaling and reduces the cortisol reactivity that competes with PFC-driven attention. Particularly well-tolerated, no taste, can be added to food for texture-sensitive individuals.

SUPPLEMENT

Omega-3 (EPA:DHA 2:1 or EPA-dominant)

EPA 500-1,500 mg/day depending on age and weight

EPA-dominant omega-3 is the most replicated supplement for ADHD. A 2012 Cochrane-level meta-analysis confirms significant improvement in inattention, hyperactivity, and cognitive performance. Effect size is modest vs. stimulant medication but meaningful for genetic risk contexts and as adjunct support.

DIET

Eliminate Artificial Food Colors (AFCs) and Preservatives

Strict elimination of Red 40, Yellow 5/6, Blue 1/2, sodium benzoate

AFC + sodium benzoate combination produces measurable ADHD symptom worsening in general population children (McCann et al. Lancet 2007) and dramatically more in ADHD-susceptible genetics. Elimination produces results within days.

DIET

Protein-First Breakfast (20g+ protein within 30 min of waking)

Eggs, Greek yogurt, meat, protein smoothie

Morning protein provides tyrosine and phenylalanine for dopamine synthesis, establishing the catecholamine foundation for the day's attention demands. Children with ADHD propensity who eat high-carbohydrate breakfasts start the day with sub-optimal prefrontal dopamine.

LIFESTYLE

Outdoor Exercise Before Focused Tasks ('Green Exercise')

20-30 min outdoor physical activity before school or work

Green exercise (outdoor nature-based movement) produces the largest acute ADHD symptom improvement of any non-pharmacological intervention. A meta-analysis shows nature walks reduce ADHD symptoms more than urban walks or indoor exercise.

LIFESTYLE

Reduce Screen Time and Digital Interruptions

<1 hour recreational screen/day for children; no phones during focused tasks for adults

High-speed digital media trains the dopamine system toward rapid reward cycles that compete with sustained attention. In ADHD propensity genetics, the dopamine reward threshold is already dysregulated; fast-paced media worsens this chronically.

FURTHER TESTING

Serum Ferritin (target >50 ng/mL for ADHD)

Annual until stable

Ferritin is the highest-value single test for ADHD because iron deficiency independently causes ADHD-identical symptoms and is highly correctable. A ferritin below 30 ng/mL identifies a correctable biological driver; below 50 ng/mL warrants supplementation in ADHD propensity contexts.

FURTHER TESTING

Comprehensive Micronutrient Panel (zinc, magnesium RBC, copper, B6/P5P)

Zinc, magnesium, and copper deficiencies are each independently associated with ADHD symptoms and are common in neurodiverse children due to restricted eating patterns. A single panel identifies the specific deficiency driving symptoms.

ATTENTION

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Those with a typical predisposition for attention are likely to exhibit focus and concentration abilities that align with the average population. Their capability to stay engaged with tasks, avoid distractions, and manage multiple tasks simultaneously should match general expectations. Engaging in activities that require sustained focus, along with maintaining a healthy lifestyle and practicing mindfulness, can support and potentially improve attentional skills

2 Methylation

22 variants analyzed

Methylation is a vital biochemical process that occurs in every cell of our body. It plays a crucial role in various biological functions, including DNA synthesis, gene expression, detoxification, neurotransmitter synthesis, and more. This section explores genetics related to methylation and provides insights into methylation capacity and potential risks. Genetic variations in key genes involved in the methylation pathway, such as MTHFR, COMT, and others, can influence your methylation capacity. These variations can impact the efficiency of methylation reactions, potentially leading to imbalances in various metabolic processes. Understanding genetic predispositions for methylation can help shed light on how the body may process and utilize essential nutrients like folate, vitamin B12, and other cofactors involved in the methylation cycle. It can also provide insights into potential risks associated with impaired methylation, such as elevated homocysteine levels or reduced detoxification capacity. This section also includes related micronutrients which are relevant to methylation activity. Optimal methylation is important for overall health and well-being. Lifestyle factors, diet, and targeted supplementation can assist in supporting methylation capacity. This can include ensuring adequate intake of methyl-donating nutrients, adopting a healthy diet rich in whole foods, managing stress, and optimizing lifestyle choices that promote overall methylation balance.

METHYLATION ABILITY

YOUR RESULTS

UNDERMETHYLATION POTENTIAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with Undermethylation Potential methylation propensity genetics have a potential for undermethylation. Their genetic profile suggests their body may have a tendency towards reduced methylation capacity, but it may not necessarily lead to undermethylation in all cases. It is still important for Individuals with Undermethylation Potential methylation propensity genetics to be mindful of factors that can influence methylation and take proactive steps to support optimal methylation through a well-balanced diet, adequate nutrient intake, and lifestyle practices that promote overall well-being. Foundational micronutrients which benefit from support include B vitamins, magnesium, choline and zinc.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Methylcobalamin B12 - high-dose sublingual

Sublingual: 1,000-5,000 mcg/day

The most significant functional medicine finding in autism methylation research. James et al. 2004 showed that a specific subset of children with autism have measurably reduced methylation capacity (low SAM:SAH ratio, low plasma glutathione) that responds to methylcobalamin supplementation.

SUPPLEMENT

Folinic Acid (not folic acid, not methylfolate as first choice)

400 mcg-2 mg/day (children); 2-5 mg/day (adults)

In autism, folinic acid is preferred over methylfolate as first-line because folate receptor antibodies are common (approximately 75% of autism families in some studies) and block methylfolate transport but not folinic acid. Folinic acid provides active folate through the RFC pathway, bypassing both barriers.

DIET

Methyl-Donor-Rich Foods (eggs, liver, beets, leafy greens, sardines)

Daily rotation of 2-3 sources

Choline (eggs), betaine (beets, quinoa), folate (leafy greens), and B12 (sardines, liver) are the primary dietary methyl donors. For children who are selective eaters, establishing egg and leafy green acceptance through food chaining provides consistent nutritional methylation support alongside supplements.

DIET

Reduce Processed Foods with Synthetic Folic Acid

Synthetic folic acid competes with folinic acid at the folate receptor and cannot be converted by MTHFR-impaired enzymes. In autism where folate receptor antibodies are prevalent, excess unmetabolized folic acid worsens the receptor blockade.

LIFESTYLE

Reduce Environmental Toxin Exposure (pesticides, BPA, heavy metals)

Organic produce where possible; BPA-free containers; water filter

Environmental toxins consume methylation capacity through Phase II detoxification. In autism populations where methylation is already impaired, additional toxin load depletes SAM faster. Reducing the detox burden preserves methyl groups for neurological methylation functions.

FURTHER TESTING

Plasma Methylation Panel (SAM, SAH, SAM:SAH ratio, homocysteine, GSH:GSSG)

The specific biomarker panel that identified methylation impairment in autism research. SAM:SAH ratio below 3.0 and reduced glutathione with elevated GSSG confirms the dual methylation + oxidative stress pattern documented in autism subgroups.

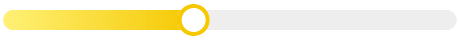
FURTHER TESTING

Folate Receptor Alpha Antibody Test (FRAAT)

FRAAT identifies antibodies blocking folate transport into the brain. In autism, FRAAT positivity (approximately 75% in some cohorts) explains why folate supplementation fails to improve neurological outcomes. Positive FRAAT mandates switching to folinic acid and potentially high-dose folinic acid therapy under physician guidance.

MTHFR ACTIVITY

YOUR RESULTS



60-70% ENZYME ACTIVITY

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with 60-70% MTHFR enzyme activity have a moderate reduction in enzyme activity and may be at an increased risk for elevated homocysteine levels and associated health problems. However, the risk is generally not as high as for individuals with further reduced enzyme activity. Individuals with this enzyme activity level may still benefit from folate and other b vitamins to support overall health.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Folinic Acid (Leucovorin) - First Line Over Methylfolate in Autism

2-5 mg/day (Rx) or OTC folinic acid equivalent

In autism, MTHFR impairment is compounded by frequent folate receptor antibodies that block methylfolate from entering the brain. Folinic acid uses the Reduced Folate Carrier (RFC-1) pathway that bypasses this block. RCT evidence: Frye et al. high-dose folinic acid showed significant improvement in verbal communication in autism children.

SUPPLEMENT

Methylcobalamin B12 (paired with folinic acid)

1,000-5,000 mcg sublingual daily or SC every 3 days

Folinic acid and methylcobalamin work synergistically. Folinic acid requires B12 for the MTR reaction that regenerates methionine. Without adequate B12, folinic acid supplementation can cause folate trapping. Use methylcobalamin (not cyanocobalamin) for optimal brain delivery.

DIET

Avoid All Synthetic Folic Acid (especially in autism context)

Strict: no fortified cereals, breads, or supplements containing 'folic acid'

The autism-specific concern is greater than in the general population: unmetabolized folic acid competes with folinic acid at the folate receptor, worsening neurological folate delivery in FRAAT-positive individuals. Particularly critical for children consuming high quantities of fortified cereals and formula.

DIET

Natural Folate Foods (liver, lentils, asparagus, dark leafy greens)

Daily integration

Natural polyglutamate folates from food do not compete with folinic acid at the folate receptor and support methylation through different absorption pathways. Chicken liver (536 mcg/3oz) and lentils (358 mcg/cup) are the most concentrated natural sources.

LIFESTYLE

Nitrous Oxide - Caution

Alert all dentists and medical providers

Nitrous oxide permanently inactivates methylcobalamin. In a child with MTHFR impairment + low methylation capacity, exposure to nitrous oxide exposure could cause a B12 and folate deficiency.

FURTHER TESTING

Homocysteine (plasma)

Target <7 µmol/L

Homocysteine is the most accessible functional marker of MTHFR activity in the clinical setting. Elevated homocysteine in the context of autism + MTHFR variant confirms functional methylation impairment. The autism-specific target is <7 µmol/L, tighter than cardiovascular targets.

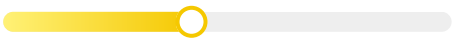
FURTHER TESTING

Folate Receptor Alpha Antibody Test (FRAAT)

FRAAT testing determines whether methylfolate vs. folinic acid is the appropriate supplement choice. The two have fundamentally different CNS delivery mechanisms. A positive FRAAT changes the clinical approach entirely and should be tested before committing to a long-term folate protocol.

MTR

YOUR RESULTS



TYPICAL ACTIVITY

UNDERSTANDING WHAT THIS MAY MEAN

MTR encodes methionine synthase, an enzyme that converts homocysteine back to methionine using vitamin B12 as a cofactor. This reaction is central to the methylation cycle and plays a key role in maintaining healthy homocysteine levels and supporting DNA synthesis, neurotransmitter production, and overall methylation capacity. This is the most common result, occurring in approximately 56% of individuals. Despite being the typical result, typical MTR activity corresponds with the greatest potential for undermethylation and elevated homocysteine levels among the three possible outcomes. Adequate intake of vitamin B12, folate, and other methyl-donor nutrients is particularly important for supporting this enzyme. Regular monitoring of homocysteine levels may be beneficial, especially in the context of other methylation variants.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Methylcobalamin B12

Per methylation propensity protocol (see Methylation Propensity results)

MTR (methionine synthase) uses methylcobalamin as its direct cofactor to convert homocysteine to methionine. MTR variants impair this step. Follow the broader methylation protocol already established for this individual. This is the primary rate-limiting factor for MTR enzyme function.

SUPPLEMENT

Riboflavin (B2)

25-50 mg/day as riboflavin 5-phosphate

MTRR regenerates the methylcobalamin cofactor that MTR depends on. MTRR variants reduce this regeneration capacity, making B2 specifically rate-limiting for the entire MTR-MTRR cycle. Active form (riboflavin 5-phosphate) preferred. Riboflavin deficiency is common in autism due to restricted diets.

FURTHER TESTING

Plasma Homocysteine

Both MTR and MTRR variants are primary drivers of homocysteine elevation. Plasma homocysteine is the functional readout for both enzymes and guides supplementation intensity. Target <7 µmol/L in pediatric populations.

MTRR

YOUR RESULTS

INCREASED ACTIVITY

UNDERSTANDING WHAT THIS MAY MEAN

MTRR encodes methionine synthase reductase, an enzyme responsible for reactivating methionine synthase (MTR) when it becomes inactive. MTR requires vitamin B12 in its active form to function; MTRR recycles this cofactor so the methylation cycle can continue running. Reduced MTRR activity can impair MTR function even when B12 intake is sufficient, making micronutrient status particularly important in individuals with MTRR variants. This result reflects a homozygous variant associated with increased MTRR activity, providing greater-than-average support for MTR reactivation and methylation cycle function. This is generally a favorable finding, suggesting efficient recycling of the B12 cofactor needed to sustain healthy homocysteine metabolism. Maintaining a nutrient-dense diet with adequate B12 and folate continues to support optimal methylation outcomes.

COMT

YOUR RESULTS

TYPICAL ACTIVITY

UNDERSTANDING WHAT THIS MAY MEAN

Those with typical or average COMT activity exhibit average methylation and neurotransmitter breakdown, mirroring the general population. Maintaining a balanced diet rich in nutrients that support methylation, such as folate, vitamin B12, and betaine, is recommended. Regular physical and mental health check-ups can help ensure that methylation processes are functioning optimally

3 Detoxification

42 variants analyzed

The Phase 1 detoxification system in our bodies plays a crucial role in metabolizing and eliminating various substances, including toxins, medications, and environmental chemicals. This section explores genetic predispositions for specific aspects of Phase 1 detoxification, providing insights into how the body may process substances. Once exposed to toxicants, the first phase of detoxification involves chemically transforming the compounds to make them easier to remove from the body. The effect of this transformation is that the molecules often become more toxic. Yet it is typically more desirable to have more effective phase 1 metabolism as long as the phase 2 system is working effectively. Two aspects covered in this section are mold and phthalate metabolism. Genetics play a role is determining predispositions toward the breakdown of these compounds. Understanding these predispositions can assist in providing more precise recommendations. Additionally, the metabolism of medications is also influenced by a number of genetic factors. While many enzymes are involved in medication metabolism, typically the most impactful is the CYP2D6 enzyme. This enzyme is responsible for metabolizing a wide range of medications. In addition to general CYP2D6 medications, genetic results for over-the-counter medications such as NSAIDs and acetaminophen are also included. Metabolism genetics can vary greatly and may influence outcomes. Understanding genetic tendencies related to Phase 1 detoxification can assist in informed decisions about environmental exposures, medication choices, and potential areas of support for detoxification pathways. It's important to note that genetics is just one factor in detoxification, and lifestyle choices, environmental exposures, and overall health are also factors.

CYP1A1

YOUR RESULTS

RAPID METABOLISM

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with this genetic result have a rapid metabolism of environmental toxins and harmful substances, including mold-related compounds. Genetic variations associated with this result suggest that their bodies have an enhanced ability to metabolize and eliminate toxins, such as mold, pesticides, and other environmental chemicals, efficiently. This may provide a better ability to handle environmental exposures and reduce susceptibility to adverse health effects. However, it is still important for individuals with this result to be cautious of potential toxin-related risks and take appropriate preventive measures to minimize exposure. Additionally, it is important to ensure Phase II detoxification systems are working effectively to neutralize and eliminate the byproducts produced during this process.

CYP1A2

YOUR RESULTS

INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

CYP1A2 is responsible for metabolizing caffeine, acetaminophen (Tylenol), several antipsychotic and antidepressant medications, and environmental compounds including mycotoxins from mold exposure. Individuals with Increased CYP1A2 activity process these substances faster than average. Caffeine may be cleared more quickly, reducing its stimulant effect, and certain medications may reach lower-than-expected therapeutic levels before being metabolized. A comprehensive pharmacogenomic panel is recommended to evaluate whether this activity level is affecting medication efficacy and to guide treatment decisions.

CYP2B6

YOUR RESULTS

NORMAL

UNDERSTANDING WHAT THIS MAY MEAN

CYP2B6 is involved in metabolizing bupropion, ketamine, methadone, certain HIV antiretrovirals, and cyclophosphamide, a chemotherapy agent. It also plays a role in processing environmental exposures including phthalates, a class of plasticizers found in common household products, and some organophosphate pesticides. A Normal CYP2B6 result indicates average processing capacity for these medications and environmental compounds. Standard medication dosing is generally appropriate, though phthalate exposure reduction remains a practical wellness consideration regardless of genetic result.

CYP2C9

YOUR RESULTS

NORMAL METABOLISM

UNDERSTANDING WHAT THIS MAY MEAN

CYP2C9 is a key enzyme in the metabolism of blood thinners such as warfarin, NSAIDs including ibuprofen and naproxen, certain antidiabetic drugs, and some blood pressure medications. A Normal CYP2C9 result indicates average metabolic activity for these substances. Standard dosing of CYP2C9-metabolized medications is generally appropriate, though drug interactions and individual health factors should still be reviewed with a prescribing provider.

CYP2C19

YOUR RESULTS

INTERMEDIATE METABOLISM

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with an intermediate CYP2C19 metabolism may process certain medications at a slower rate than normal, which can increase the risk of side effects or drug toxicity. Your healthcare provider may need to adjust your medication dosage or switch to a different medication altogether if taking medications that are affected by CYP2C19.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

NAC (N-Acetylcysteine)

600-1,200 mg/day

Replenishes hepatic glutathione, the primary conjugation substrate for Phase I-activated toxin intermediates. When CYP enzymes generate reactive intermediates faster than Phase II can conjugate them, NAC provides the cysteine needed to restore glutathione. NAC has the additional benefit of modulating glutamate in autism.

SUPPLEMENT

Broccoli Seed Extract (Sulforaphane)

400-600 mcg sulforaphane/day (standardized extract)

Sulforaphane from broccoli seed extract induces Phase I CYP enzyme expression via NRF2 activation, supporting the liver's initial oxidation of environmental toxins, drugs, and metabolic byproducts. Significantly more potent than mature broccoli. Standardized seed extract delivers 10 to 100x more sulforaphane per serving than general cruciferous vegetables.

DIET

Cruciferous Vegetables (broccoli, cauliflower, Brussels sprouts)

5+ servings/week or broccoli sprout extract if vegetable acceptance is limited

Indole-3-carbinol and DIM from cruciferous vegetables modulate CYP1A1, CYP1B1, and CYP3A4 activity, important for detoxifying environmental estrogens, polycyclic aromatic hydrocarbons, and xenobiotics. Broccoli sprout extract provides a more concentrated, food-acceptance-friendly format.

LIFESTYLE

Reduce Environmental Chemical Exposure

Organic produce, fragrance-free personal care, air purifier, water filter

Pesticides (organophosphates, glyphosate), phthalates, parabens, and VOCs all require CYP-mediated Phase I detoxification. In autism where Phase I capacity may be reduced, reducing the input is more impactful than trying to increase output.

LIFESTYLE

Sweat Regularly (infrared sauna or vigorous exercise)

3-5x/week; infrared sauna 20 min at 140 degrees F for those who tolerate it

Sweat is a meaningful elimination route for fat-soluble environmental toxins (pesticides, flame retardants, phthalates, BPA) that bypass hepatic Phase I processing via lipid partitioning. Infrared sauna penetrates deeper and is better tolerated by sensory-sensitive individuals due to lower humidity.

FURTHER TESTING

Organic Acids Test (OAT) - Detox Markers (pyroglutamic acid, 2-hydroxyhippuric acid)

MosaicDX

OAT provides indirect evidence of Phase I/II detox capacity. Elevated pyroglutamic acid confirms glutathione depletion; elevated 2-hydroxyhippuric acid indicates Phase II glucuronidation is under load. Practical first-line assessment of overall detox capacity without requiring CYP genotyping.

PON1

YOUR RESULTS

TYPICAL ACTIVITY

UNDERSTANDING WHAT THIS MAY MEAN

PON1 (paraoxonase 1) is an enzyme primarily associated with detoxification of organophosphate pesticides and environmental neurotoxins, and it also plays a role in reducing oxidative stress by protecting against lipid oxidation. A typical PON1 result indicates average enzyme activity, meaning the body processes organophosphate compounds and manages lipid oxidation at a typical rate. While this represents a lower-risk result, reducing dietary and environmental pesticide exposure remains a sound general practice, as PON1 activity can be influenced by nutritional status and overall toxic burden.

CYP2D6

YOUR RESULTS

INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

CYP2D6 is a critical enzyme for metabolizing a large class of psychiatric and neurological medications, including many antidepressants, antipsychotics, ADHD medications such as atomoxetine and amphetamines, and opioid pain relievers including codeine and tramadol. Individuals with Increased CYP2D6 activity process these medications faster than average, which can reduce their effectiveness at standard doses. For prodrugs like codeine, increased activity can lead to faster-than-expected conversion to active compounds, raising safety considerations. A comprehensive pharmacogenomic panel is strongly recommended to guide safe and effective medication management.

CYP2E1

YOUR RESULTS

NORMAL METABOLISM

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with this genetic result have a normal CYP2E1 metabolism. Their genetic profile suggests that their bodies can effectively metabolize and eliminate acetaminophen and other CYP2E1 metabolites within the average range. It is still important for individuals with this result to use acetaminophen responsibly and follow recommended dosing guidelines to avoid potential risks associated with excessive use or overdose.

GLUCURONIDATION

YOUR RESULTS

REDUCED FUNCTION

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with this genetic result may have reduced glucuronidation function. Genetic variations associated with this result suggest that their bodies may have a decreased capacity to perform glucuronidation efficiently. It is important for individuals with this result to be mindful of their toxicant exposure and consider lifestyle practices that support overall detoxification and elimination processes. This may include consuming a nutrient-rich diet, staying hydrated, engaging in regular physical activity, and avoiding excessive exposure to environmental toxins.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Calcium D-Glucarate

500-1,000 mg/day

Inhibits beta-glucuronidase, the gut enzyme that reverses glucuronidation and allows toxins and hormones to re-enter circulation. Directly supports Phase II detox capacity. Well-tolerated and particularly relevant for children with impaired detox SNPs where reducing enterohepatic recirculation reduces overall detox demand.

DIET

Cruciferous Vegetables (Broccoli Sprouts preferred)

Daily serving; sprouts most potent

Sulforaphane activates NRF2, upregulating UGT enzymes responsible for glucuronidation. Broccoli sprouts contain 20-50x the sulforaphane of mature broccoli. Can be blended into smoothies for texture-sensitive children. The most potent food-based induction of Phase II glucuronidation available.

DIET

Quercetin-Rich Foods (apples, onions, berries)

Daily in diet

Quercetin and polyphenols support glucuronidation enzyme activity and reduce gut beta-glucuronidase. Also has mast cell-stabilizing properties relevant to the autism/immune overlap, providing dual benefit for conditions frequently co-occurring in this population.

LIFESTYLE

Reduce Gut Dysbiosis (probiotic support + low sugar)

Ongoing

Dysbiotic gut bacteria are a primary source of beta-glucuronidase. A high-sugar diet fuels these bacteria. Probiotic support (Lactobacillus and Bifidobacterium strains) reduces beta-glucuronidase activity measurably and supports overall Phase II glucuronidation capacity.

FURTHER TESTING

Organic Acids Test (OAT) - Detox Markers

MosaicDX

Markers such as pyroglutamic acid and 2-hydroxyhippuric acid on the OAT reflect Phase II conjugation capacity. Useful baseline before and after glucuronidation support protocol to confirm whether the intervention is producing measurable improvement in detox pathway function.

4 Immune & Inflammation

16 variants analyzed

Mast cells play a pivotal role in the body's immune response, particularly in allergic reactions, where they release histamine and other mediators in response to allergen exposure. Immunoglobulin E (IgE) is a type of antibody that binds to allergens and triggers mast cell activation. Variations in the genetic regulation of mast cells and IgE can significantly influence the severity of allergic responses and susceptibility to related conditions, such as asthma, eczema, and anaphylaxis. This section explores the genetic factors contributing to mast cell activation and IgE production, providing insights into personalized approaches for managing allergies and related disorders. The regulation of mast cell activation involves numerous genes, including those coding for mast cell receptors, signaling molecules, and the enzymes involved in mediator synthesis. Genetic variations in these components can alter mast cell reactivity and the subsequent release of histamine and other substances, affecting the severity and threshold of allergic reactions. For example, mutations in the KIT gene, which encodes a receptor tyrosine kinase essential for mast cell development and function, have been associated with increased mast cell proliferation and activity. The production of IgE is tightly regulated by both genetic and environmental factors. Variations in genes involved in the immune response, such as those encoding cytokines and their receptors (e.g., IL-4, IL-13, and their receptors), can influence IgE levels and the propensity for allergic sensitization. Furthermore, polymorphisms in the FCER1A gene, encoding the high-affinity IgE receptor, can affect IgE binding to mast cells and basophils, modulating the allergic response. While genetic predispositions play a crucial role in mast cell activation and IgE-mediated responses, environmental exposures, diet, and lifestyle factors also significantly impact allergy development and management. A holistic approach that combines genetic insights with environmental and lifestyle modifications can offer comprehensive management strategies to improve quality of life for individuals with allergic and mast cell-related disorders.

IGE LEVELS

YOUR RESULTS

DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with a decreased risk for elevated immunoglobulin E (IgE) levels and mast cell activation possess genetic factors suggesting they are less likely to experience conditions associated with high IgE levels, such as allergies, asthma, and other atopic disorders. This reduced genetic susceptibility implies a lower likelihood of excessive mast cell activation, which can lead to hypersensitivity reactions. Maintaining a healthy lifestyle and environment can further minimize the risk of developing related conditions.

FOOD-BASED HISTAMINE SENSITIVITY

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with a typical sensitivity to food-based histamine exhibit an average response to histamine, similar to the general population. This indicates that they have a typical likelihood of experiencing symptoms related to histamine intolerance, such as headaches, nasal congestion, or skin irritation, when consuming histamine-rich foods. Awareness and moderation are key; individuals should monitor their responses to certain foods and adjust their diet accordingly to manage symptoms effectively

GENERAL HISTAMINE SENSITIVITY

YOUR RESULTS

INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals identified with increased histamine sensitivity carry genetic markers that heighten their reaction to histamine, affecting their response not just to foods but also to environmental factors. This increased sensitivity can exacerbate symptoms related to asthma, sinusitis, and allergies, such as more frequent or severe nasal congestion, skin reactions, and respiratory distress. For those with an increased sensitivity, managing environmental exposures, consulting with healthcare professionals for appropriate treatments, and possibly following a low-histamine diet can be key strategies in reducing symptoms and improving quality of life.

SUPPLEMENT

DAO (Diamine Oxidase) Enzyme

1-2 caps with high-histamine meals

DAO is the primary enzyme that breaks down dietary histamine in the gut. Many individuals with mast cell issues have reduced DAO activity; supplementing before high-histamine meals prevents dietary histamine from triggering systemic mast cell symptoms (GI distress, flushing, behavioral changes, sleep disruption).

SUPPLEMENT

Quercetin (mast cell stabilizer)

500-1,000 mg/day, 20 min before meals (cap at 250 mg if slow COMT)

Quercetin is the most potent natural mast cell stabilizer, inhibiting histamine release from mast cells and blocking IgE-mediated degranulation. It also inhibits histamine-N-methyltransferase, reducing histamine production rather than just blocking symptoms.

DIET

Low-Histamine Diet (elimination trial 4-6 weeks)

Avoid: fermented foods, aged cheeses, cured meats, wine, vinegar, leftover proteins, certain fish

Reduces the histamine load arriving from the gut, relieving the DAO/HNMT clearance burden and reducing mast cell trigger exposure. Strict elimination for 4-6 weeks establishes whether dietary histamine is a significant contributor before permanent restrictions.

DIET

Fresh-Cooked Protein Only (no leftovers for high-sensitivity individuals)

Cook and eat within 2 hours; freeze immediately if storing

Histamine accumulates in protein foods during storage. Even refrigerated leftovers accumulate significant histamine within 24 hours. For histamine-sensitive individuals, the difference between fresh-cooked and 24-hour refrigerated leftovers can be clinically significant.

LIFESTYLE

Identify and Reduce Mast Cell Triggers (heat, stress, vibration, infection, chemical exposure)

Environmental audit + symptom diary

Mast cell activation in autism is triggered not only by food but by: temperature changes, emotional stress, vibrational stimuli (heavy bass music), fragrance exposure, infections, and exercise. A symptom diary identifying non-dietary triggers allows environmental modifications that reduce total mast cell burden.

LIFESTYLE

Air Quality Control (HEPA + fragrance elimination)

HEPA air purifier in bedroom + fragrance-free household products

Airborne allergens, mold spores, and synthetic fragrances are potent mast cell triggers. Fragrance elimination (switching to fragrance-free detergents, personal care products, and cleaning supplies) is among the highest-yield environmental changes for mast cell-driven behavioral and physiological reactivity.

FURTHER TESTING

Serum Tryptase + Plasma Histamine

Collect at peak symptoms if possible

Elevated serum tryptase (>11.4 ng/mL) confirms systemic mast cell activation; elevated plasma histamine confirms histamine as the active mediator. Both inform whether this trait is genetically propensity only or actively expressed.

FURTHER TESTING

Comprehensive Allergy / Sensitivity Panel (IgE food panel)

IgE identifies immediate (mast cell-driven) allergic reactions; IgG identifies delayed immune reactions to specific foods that amplify the overall mast cell activation burden. Combined testing creates an individualized dietary guide rather than relying on generic low-histamine lists.

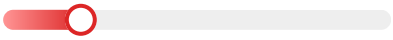
5 Nutrition

32 variants analyzed

Omega fatty acids, including omega-6 and omega-3, are essential polyunsaturated fats that play vital roles in the body's overall health. This section explores genetic propensities for omega-6 risk and omega-3 need, providing insights into how your body may respond to these fatty acids. Omega-6 fatty acids are important for many biological processes, but an imbalance between omega-6 and omega-3 intake may have implications for health. Some individuals may have a genetic predisposition for a higher risk of imbalanced omega-6 levels, which may lead to inflammation and other health concerns. These individuals may benefit from a dietary approach that focuses on achieving a balanced ratio of omega-6 to omega-3 fatty acids. Incorporating more sources of omega-3 such as fatty fish can help restore the balance. Omega-3 fatty acids are known for their anti-inflammatory properties and various health benefits. Some individuals may have a genetic propensity for increased omega-3 needs, indicating a higher requirement to maintain optimal health. These individuals may benefit from consuming higher amounts of omega-3-rich foods or considering omega-3 supplementation, especially if dietary intake is limited. Understanding your genetic tendencies related to omega-6 risk and omega-3 need can provide valuable insights for tailoring dietary choices to optimize overall health. It's important to note that genetics is just one aspect, and a well-rounded approach to nutrition, including a diverse and balanced diet, regular physical activity, and other healthy lifestyle habits, is crucial for overall well-being. Examples of Omega-6 food sources include: various vegetable oils, nuts, and seeds. Examples of DHA and EPA (Omega-3) food sources include: mackerel, salmon, cod liver, herring, oysters, and sardines.

OMEGA-3 NEED

YOUR RESULTS



SIGNIFICANTLY INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with significantly increased omega-3 need genetics have a notably elevated requirement for EPA and DHA omega-3s, compounded by reduced conversion of plant-based ALA to EPA and DHA. Prioritize marine-sourced omega-3s through fatty fish at several meals per week and consider higher-dose EPA/DHA supplementation under healthcare guidance.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

EPA + DHA Fish Oil (or algae oil)

EPA 500-1,500 mg + DHA 300-1,000 mg/day (dose by weight for children)

Omega-3 fatty acid status is consistently lower in autism and ADHD populations vs. neurotypical controls; supplementation trials show benefits in attention, communication, and behavioral symptoms. Dose by weight for children: EPA approximately 20 mg/kg/day is a common clinical starting dose.

SUPPLEMENT

Phospholipid-Bound Omega-3 (krill oil or herring roe)

500-1,000 mg krill oil/day

Phospholipid-bound omega-3 reaches the brain more efficiently than triglyceride-form fish oil because phospholipid fatty acids cross the blood-brain barrier via a specialized transport system (LPC-DHA transport). For neurodevelopmental applications where brain DHA delivery is the goal, phospholipid form may provide superior CNS delivery.

DIET

Fatty Fish 3x/Week (sardines, mackerel, salmon - lowest mercury)

Canned sardines or mackerel as most cost-effective + low mercury options

Low-mercury fatty fish provide omega-3 in highly bioavailable forms alongside vitamin D, selenium, and protein. Sardines (packed in water or olive oil) are the most nutritionally dense, lowest-mercury, and most affordable option, addressing multiple autism nutritional concerns in one food.

DIET

Reduce Omega-6 Vegetable Oils (soybean, corn, sunflower, canola oils)

Replace cooking oils with olive oil, avocado oil, or butter/ghee

The omega-6:omega-3 ratio in Western diets (approximately 15:1 or worse) actively inhibits omega-3 conversion and metabolism. Excess omega-6 from vegetable oils competes with omega-3 at the same elongase/desaturase enzymes. Simply reducing omega-6 intake can improve omega-3 utilization without increasing omega-3 dose.

LIFESTYLE

Vitamin D Optimization (required for omega-3 metabolism)

Maintain 25-OH vitamin D at 60-80 ng/mL

Vitamin D regulates the expression of fatty acid desaturase enzymes (FADS1, FADS2) that convert dietary ALA to EPA and DHA. Deficiency, extremely common in autism, impairs omega-3 conversion capacity. Vitamin D optimization is a prerequisite to omega-3 metabolic efficiency.

FURTHER TESTING

Omega-3 Index (red blood cell EPA + DHA)

RBC omega-3 index reflects 3-month average omega-3 status in cell membranes, the most functionally relevant compartment. Target >8% for neurodevelopmental optimization. Low index despite supplementation may indicate FADS gene variants reducing conversion or absorption issues.

FOLIC ACID SUPPLEMENTATION TOLERANCE

YOUR RESULTS

DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Likely to have reduced conversion of folic acid to folate within the body; increased potential for folic acid buildup and increased homocysteine levels. Folic acid supplementation should be limited in favor of folate or folinic acid.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Methylcobalamin B12 - high-dose sublingual

Sublingual: 1,000-5,000 mcg/day

The most significant functional medicine finding in autism methylation research. James et al. 2004 showed that a specific subset of children with autism have measurably reduced methylation capacity (low SAM:SAH ratio, low plasma glutathione) that responds to methylcobalamin supplementation.

SUPPLEMENT

Folinic Acid (not folic acid)

2 mg/kg/day (Frye et al. RCT protocol); typical range 1-5 mg/day

The Frye 2018 RCT, the first double-blind RCT of folinic acid in autism, showed significant improvement in verbal communication in children with autism. Folinic acid is preferred over folic acid and over methylfolate (bypasses folate receptor antibodies via RFC pathway). The most evidence-based folate form for autism.

DIET

Liver (chicken, beef) 1x/week - nutrient density per ounce unmatched

3 oz chicken liver: 536 mcg folate, 17 mcg B12, 0.9 mg B6 + iron, zinc, choline

Liver provides the entire B12/B6/folate triad in one food with exceptional bioavailability. For selective eaters, chicken liver in small amounts can be disguised in ground meat dishes (bolognese, meatballs) at 10-20% of meat volume without detectable flavor or texture change.

DIET

Animal Protein Daily (ensures B12 sufficiency)

2+ servings/day: eggs, meat, fish, dairy

B12 is found exclusively in animal foods. In autism, restricted eating patterns that exclude animal protein are a primary cause of B12 deficiency. Identifying accepted animal proteins, even if limited to eggs, chicken nuggets, or specific processed meats, and building them consistently into the daily pattern is more sustainable than relying solely on supplements.

LIFESTYLE

Comprehensive Feeding Therapy (if food selectivity is severe)

Feeding therapy with SLP or OT specializing in pediatric feeding

Nutritional deficiency in autism is often driven by feeding restrictions that no supplement can fully compensate for long-term. Feeding therapy using the Sequential Oral Sensory approach or DIR/Floortime feeding integration addresses the root cause, expanding the diet to include nutrient-dense foods.

FURTHER TESTING

Plasma Methylation Panel (SAM, SAH, homocysteine, folate, B12)

The complete methylation picture, not just individual B-vitamin levels. In autism where multiple methylation cycle enzymes may be impaired simultaneously, measuring the functional output (SAM:SAH ratio, homocysteine) alongside substrate levels identifies whether the supplementation protocol is achieving actual methylation normalization.

FURTHER TESTING

Folate Receptor Alpha Antibody Test (FRAAT)

Determines whether folinic acid or methylfolate is the appropriate folate supplement. FRAAT positive means folinic acid only (methylfolate receptor is blocked). FRAAT negative means either form is appropriate. This single test determines the entire folate supplementation strategy and should be done before any folate protocol is committed to.

VITAMIN B6 LEVEL PROPENSITY

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with Typical B6 genetics are likely to maintain sufficient vitamin B6 levels with a balanced diet. B6 supports mood regulation and sleep quality.

VITAMIN B9 NEED

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical folate (B9) genetics have average requirements for this nutrient. Consume folate-rich foods, leafy green vegetables, legumes, and fortified cereals, to meet daily needs.

VITAMIN B12 LEVEL

YOUR RESULTS

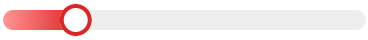
TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with Typical B12 genetics are likely to maintain healthy vitamin B12 levels with a balanced diet. Supplementation may still be beneficial, particularly for those with limited animal product intake.

MAGNESIUM DEFICIENCY RISK

YOUR RESULTS



SIGNIFICANTLY INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with significantly increased risk magnesium deficiency risk genetics may have a high risk of experiencing magnesium deficiency. It is crucial for these individuals to consume a balanced diet that includes magnesium-rich foods and/or consider magnesium supplementation to prevent deficiency and associated health risks.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Magnesium Glycinate (primary form in autism)

Children: 3-5 mg/kg/day in divided doses; Adults: 300-400 mg/day

Magnesium deficiency is the most common micronutrient finding in autism and the most impactful single correction. Research consistently shows that magnesium repletion in children with autism produces improvements in hyperactivity, sleep, attention, and sensory tolerance. Glycinate form provides the best GI tolerance.

SUPPLEMENT

Magnesium + B6 (P5P) Combined

Magnesium glycinate 200 mg + P5P 25 mg/day

The magnesium-B6 combination has specific clinical evidence in autism: Mousain-Bosc et al. RCT showed significant improvements in the autism quotient, social interaction, and communication scores. P5P (active B6) assists cellular magnesium uptake and both cofactors support GABA synthesis.

DIET

Magnesium-Rich Foods Accessible to Selective Eaters (pumpkin seeds, dark chocolate, banana, avocado)

Daily integration

Pumpkin seeds (156 mg/oz) can be blended into smoothies or nut butter. Dark chocolate (64 mg/oz) is frequently accepted. Bananas (approximately 32 mg each) are among the most commonly accepted fruits in selective eaters. Building these foods into accepted routines provides consistent dietary magnesium support.

LIFESTYLE

Reduce Stress and Sensory Overload (primary magnesium depleter in autism)

Proactive sensory regulation and stress prevention strategies

In autism, the stress response is chronically activated. Cortisol and adrenaline release from sensory overload, transitions, and social demands continuously depletes magnesium. Proactive stress reduction (sensory diet, predictable routines, transition warnings) reduces magnesium consumption, making supplementation more effective.

FURTHER TESTING

RBC Magnesium (not serum)

Target >5.5 mg/dL

Serum magnesium is maintained in normal range at the expense of intracellular stores. Serum normal with RBC low is the common finding in autism. RBC magnesium reflects the intracellular depletion that produces symptoms. Annual testing guides dose adjustments and confirms repletion adequacy.

GLUCOSE METABOLISM

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Those with typical glucose metabolism exhibit genetic markers that reflect the average population's ability to metabolize glucose. This indicates a standard efficiency in processing blood sugar, with lifestyle and dietary habits playing a significant role in maintaining healthy glucose levels. Individuals with this predisposition are advised to follow a healthy lifestyle, including regular exercise, a balanced diet, and regular medical check-ups, to support optimal glucose metabolism and prevent metabolic disorders

OMEGA-6 RISK

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with Typical omega-6 risk genetics process omega-6 fatty acids in a manner consistent with the general population. Consuming omega-6s in moderation while ensuring adequate omega-3 intake supports the best balance for cardiovascular and inflammatory health. Blood testing can assist in measuring omega levels.

6 Sleep

16 variants analyzed

Sleep deprivation can have significant impacts on physical and mental well-being, affecting cognitive function, mood, performance, and overall health. However, not everyone experiences the same effects of sleep deprivation, in fact, there are individuals who seem to function relatively well even with less sleep. It is important to note that while genetics play a role, other factors such as lifestyle, stress levels, and sleep hygiene practices also influence sleep deprivation sensitivity. Maintaining a healthy sleep routine, creating a conducive sleep environment, and managing stress can all contribute to better sleep quality and minimize the impact of sleep deprivation. This page explores the genetic predisposition for sleep deprivation sensitivity, suggesting how different individuals may respond to sleep loss.

SLEEP DEPRIVATION SENSITIVITY

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical sleep deprivation sensitivity respond to sleep loss in a manner similar to the general population, experiencing standard impairments to cognition, mood, and performance. Prioritizing sufficient sleep remains important for sustained health and function.

REPORTED SLEEP QUALITY

YOUR RESULTS

BELOW AVERAGE

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with Below Average subjective sleep quality genetics may experience lower-than-average self-reported sleep satisfaction. Subjective and objective sleep quality do not always align. A biometric tracker can help determine whether interventions are working. Consultation with a provider or coach may be beneficial if poor sleep quality is persistent.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Melatonin (immediate and extended-release)

0.5-1 mg immediate release for sleep onset; 2-5 mg XR for sleep maintenance; start at lowest effective dose

Melatonin is the most evidence-based sleep intervention in autism. Multiple RCTs confirm it reduces sleep onset latency by 30-60 min and increases total sleep time. Melatonin synthesis is impaired in many autism individuals (reduced ASMT enzyme activity). Start at 0.5 mg and increase by 0.5 mg weekly.

SUPPLEMENT

Magnesium Glycinate (evening)

100-200 mg (children); 300-400 mg (adults) 30-60 min before bed

Evening magnesium reduces sleep onset latency through NMDA antagonism and GABA-A potentiation. In autism where sensory hypervigilance delays sleep onset, magnesium's calming effect on the nervous system acts synergistically with melatonin. Glycine (in glycinate) additionally promotes sleep onset through thermoregulatory mechanisms.

DIET

No Screen Light After Dark (primary melatonin disruptor)

Sunset-time screens off for all devices; amber lighting in evening

Autism individuals show particular sensitivity to light-induced melatonin suppression. Even brief evening blue light exposure delays an already-impaired melatonin onset further. Amber-spectrum glasses or f.lux/Night Shift screen filters are practical accommodations that do not require removing devices.

DIET

High-Carbohydrate Bedtime Snack (tryptophan + carb for melatonin pathway)

Banana and a small amount of nut butter for example

Carbohydrate at bedtime enhances tryptophan transport into the brain for serotonin and melatonin synthesis. In autism where the melatonin pathway is already impaired, optimizing dietary substrate delivery addresses the downstream deficiency. Banana provides tryptophan + carbohydrate simultaneously and is frequently accepted by selective eaters.

LIFESTYLE

Consistent Visual Sleep Schedule (visual schedule for bedtime routine)

Picture-based routine posted at child's eye level; same sequence nightly

Visual bedtime routines reduce the transition anxiety that delays sleep in autism. A predictable, visually represented sequence of 5-8 steps (bath, pajamas, teeth, book, lights off) removes the uncertainty that drives protest behaviors and hypervigilance at bedtime.

LIFESTYLE

Weighted Blanket (deep pressure for sleep onset)

10% of body weight + 1 lb; silk or jersey material for tactile sensitivity

A 2020 RCT showed significant improvement in both sleep onset and subjective sleep quality in children with autism using weighted blankets vs. standard blankets. Deep pressure stimulation of the proprioceptive system produces calming parasympathetic activation. One of the most accessible evidence-based autism sleep interventions.

FURTHER TESTING

Salivary Melatonin Profile (evening + nighttime)

Measures the timing and peak of melatonin secretion; identifies whether the sleep problem is delayed onset (late melatonin peak), insufficient total melatonin (low peak), or early morning termination. The result determines whether immediate-release vs. extended-release melatonin, timed light therapy, or precursor support is the appropriate intervention.

This report uses a color-coded system to indicate the clinical significance of each genetic result and the level of recommended action. Each result is assigned one of three status levels based on your genetic variants.

No Action Required

Your genetic result in this area falls within a typical range. No specific intervention is indicated at this time. General wellness practices apply.

Practitioner Review / Action

Your genetic result suggests a moderate predisposition that warrants attention. Review with your practitioner and consider targeted lifestyle or supplementation support.

Practitioner Action

Your genetic result indicates a significant predisposition. Practitioner-guided intervention including targeted supplementation, dietary modification, and follow-up testing is recommended.

HOW TO READ YOUR RESULTS

- Each module page shows one or more gauges representing specific genetic results within that category.
- The gauge position and color indicate where your result falls relative to the general population.
- Personalized recommendations appear only for Moderate and Needs Attention results — Optimal results require no specific action.

DNA Signature - Spectrum Map Genetic Variants Summary

All 83 genotyped variants across this section, grouped by report panel.

- Homozygous Variant (+/+) — two copies of the variant
- Heterozygous (-/+) — one copy of the variant
- Homozygous Normal (-/-) — no copies of the variant

Neurodevelopment 9

HTR1B1 C861G rs6296	CG	**CLOCK A*213G** rs1801260	AA
ANKK1 G2137A rs1800497	GG	**HNMT A*60G** rs1050891	AG
COMT G472A rs4680	AG	**ADA C22T** rs73598374	CC
SNAP-25 G*239T rs3746544	TG	**KL T1054G** rs9536314	TT
MAO-A T891G rs6323	GG		

Detoxification 42

GSTP1 A313G rs1695	AA	**CYP2D6 C181-1G** rs201377835	CC
COMT G472A rs4680	AG	**CYP2D6 C506-1T** rs3892097	CC
CYP3A4 T-392C rs2740574	TT	**CYP2D6 C985+39T** rs28371725	TC
CYP2C9 C430T rs1799853	CC	**CYP2D6 G100A** rs1065852	GG
CYP2C19 G681A rs4244285	AG	**CYP2D6 G63-2604A** rs5758550	AA
CYP1A2 C1291T rs28399424	CC	**CYP2D6 T971G** rs5030867	TT
CYP1A2 C-729T rs12720461	CC	**GPX1 G599A** rs1050450	AG
CYP1A2 T739G rs2069526	TT	**GSTA1 A-135G** rs3957357	AA
CYP1A2 G-3860A rs2069514	GG	**GSTM1 (A>G)** rs366631	AA
CYP1A2 C-163A rs762551	AC	**GSTO1 C419A** rs4925	CC
CYP2B6 T983C rs28399499	TT	**GSTO1 g.G4494A** rs2164624	AG
CYP2B6 T-82C rs34223104	TT	**GSTO2 A424G** rs156697	AA
CYP2B6 G516T rs3745274	TG	**GSTP1 C341T** rs1138272	CC
CYP1A1 C-27+606A rs2606345	AC	**UGT1A1 C-364T** rs887829	TC
CYP1A1 T1384C rs1048943	TT	**UGT1A6 A541G** rs2070959	AG
CYP2E1 C1263T rs2515641	CC	**UGT1A6 G627T** rs17863783	GG

CYP2C9 A1075C rs1057910	AA	**UGT1A9 T-275A** rs6714486	TT
CYP2C9 C1003T rs28371685	CC	**UGT2B15 A253C** rs1902023	AC
CYP2C9 C1080G rs28371686	CC	**PON1 T575C** rs662	TT
CYP2D6 C1012T rs59421388	CC	**CYP1A1 G2452T** rs1799814	GG
CYP2D6 C124T rs5030862	CC	**CYP1A1 A3798G** rs4646903	AA

Immune & Inflammation

16

IL-13 C-220T rs1800925	CC	**AOC1 C1990G** rs1049793	GG
IL-13 A431G rs20541	GG	**AOC1 C47T** rs10156191	CC
IL-33 G3239A rs928413	AA	**AOC1 C995T** rs1049742	CC
IL-33 (T>C) rs3939286	CC	**AOC1 G-92-231T** rs2052129	GG
IL-33 (A>C) rs1342326	AA	**AOC1 G1329A** rs2071514	AA
FCER1A T-59-285C rs2427827	CC	**HDC T1932G** rs2073440	TT
FCER1A T-59-36C rs2251746	TC	**HNMT A*60G** rs1050891	AG
FCER1A G302A rs2298805	GG	**HNMT C314T** rs11558538	TC

Sleep

16

ADA C22T rs73598374	CC	**BTBD9** rs3923809	AA
DEC2 G1151C rs121912617	GG	**CLOCK A793-1130G** rs11932595	AG
COMT G472A rs4680	AG	**CYP1A2 C-163A** rs762551	AC
ADORA2A rs3761422	TC	**FABP7 C182T** rs2279381	CC
ADORA2A C333-527T rs2236624	CC	**MEIS1 A889-10767G** rs12469063	AG
ADORA2A rs5751862	AG	**MEIS1 T965+6302G** rs2300478	TG
PRIMA1 A230-1629G rs6575353	AA	**ADORA2A T1083C** rs5751876	TC
PTPRD G103-113009A rs1975197	AG	**ADORA2A-AS1 (C>G)** rs4822492	CG

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