Nutricode

Genomics Driven Personalised Approach to Nutrition and Fitness

GenWell

SCIENCE FOR BETTER LIVING

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REPORT

Nutricode

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Sample ID	Sample Type	Sample Collected
Sample Received	Report Generated	

NutriDNA: Advanced Genetic Insights for Smarter Nutrition and Fitness

Nutrition plays a vital role in maintaining overall health, managing body weight, and enhancing physical fitness. While traditional dietary advice has been largely generic, nutrigenetics—a cutting-edge branch of science—introduces a personalized approach by linking nutrition with genetics. This scientific field examines how genetic variations influence the body's response to nutrients, paving the way for tailored dietary and fitness strategies.

Understanding Nutrigenetics

Nutrigenetics focuses on the interplay between genes and nutrition. Each individual's genome, with approximately ~20,000 genes, contains variations that not only define physical characteristics but also affect how the body metabolizes and absorbs nutrients. These genetic differences can influence how we process carbohydrates, fats, proteins, vitamins, and minerals, making each person's nutritional needs unique. For example, individuals with certain variations in the FTO gene may be predisposed to higher odds of obesity, but targeted interventions—such as customized dietary plans and exercise routines—can help them achieve effective weight management.

Key Advantages of Nutrigenetics

Genetically Tailored Weight Management

Genetic variant-based insights help customize diets, leading to more effective weight loss & maintenance.

DNA-Driven Fitness Optimization

Genetic variants shape training plans, aligning workouts with natural strengths & endurance to maximize performance & reduce injury risks.

Proactive Wellness Through Genetics

By tailoring diets to an individual's genetic profile, nutrigenetics supports proactive health management & disease prevention

Precision Nutrition for Better Health

Personalized nutrient intake optimizes vitamin absorption, energy levels, sleep quality, & overall well-being.

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Summary of Results

Trait	Genes	Result
Vitamin And Mineral Metabolism		
Folate Requirement	*MTHFR*	Typical
Vitamin A Requirement	*BCMO1*	High risk
Vitamin B12 Requirement	*FUT2*	High risk
Vitamin C Requirement	*SLC21A*	Typical
Vitamin D Requirement	*CYP2R1, GC*	Typical
Vitamin E Requirement	*Intergenic (APOA5)*	High risk
Choline Requirement	*MTHFD1*	Typical
Magnesium Requirement	*TRPM6*	High risk
Zinc Requirement	*SLC30A3*	High risk
Calcium Requirement	*GC*	Typical
Low Iron Status	*TMPRSS6, TFR2, TF*	Typical
Iron Overload	*SLC17A1, HFE*	Typical
Food Reactions, Intolerances, And Sensitivities		
Lactose Intolerance	*MCM6*	High risk
Salt Sensitivity	*ACE*	Typical
Caffeine Metabolism	*CYP1A2*	High risk
Optimizing Weight Loss And Long Term Weight Management		
Adiponectin Levels	*ADIPOQ*	Typical
Weightloss Response To Physical Activity	*FTO, ADRB2*	Slightly Enhanced
Energy Balance (RMR)	*UCP1*	Typical
Protein Intake And Weightloss	*FTO*	Typical
Saturated And Unsaturated Fats Intake And Weightloss	*FTO*	Typical

Summary of Results

Trait	Genes	Result
Total Fats Intake And Weightloss	*TCF7L2*	Typical
Saturated Fats Intake And Obesity Risk	*APOA2*	High risk
Monounsaturated Fats Intake And Weightloss	*PPARγ2*	Typical
Starch Intake And Weight-Gain Risk	*AMY1*	Typical
Weight Regain Risk - Maintenance Of Long-Term Weight Loss	*ADIPOQ*	Typical

Eating Behavior And Inherited Food/Taste Preferences

Satiety	*FTO*	Typical
Fat Taste Perception	*CD36*	Enhanced
Sugar Preference (Sweet Tooth)	*GLUT2*	Typical
Snacking (Eating Between Meals)	*MC4R*	Typical
Hunger	*NMB*	Typical

Factors Influencing Metabolic Health And Associated Risk Levels

Response To Whole Grain Intake	*TCF7L2*	Typical
Response To MUFA Intake	*PPARG2*	Typical
Total Cholesterol Levels	*APOA5*	High risk
LDL Cholesterol Levels	*ABCG8*	High risk
HDL Cholesterol Levels	*ABCA1*	Typical
Triglycerides Levels	*ANGPTL3*	High risk
Fasting Glucose Levels	*ADCY5*	High risk
Insulin Levels	*IRS1*	Typical

Summary of Results

Trait	Genes	Result
Response To PUFA (Omega3-Omega6 Ratio) Intake	*FADS1*	Typical
Exercise Response To Cholesterol Levels	*LIPC*	Typical
Caffeine Intake And Cardiometabolic Health Risks	*CYP1A2*	High risk
Caffeine Intake - Anxiety And Sleep Disturbance Risk	*ADORA2A*	High risk
Optimizing Exercise Performance, Fitness, And Managing Injury Risk		
Motivation To Exercise	*BDNF*	Typical
Exercise Behavior	*CYP19A1, LEPR*	Enhanced
Response To Power And Strength Training	*ACTN3*	Enhanced
Risk For Muscle Damage	*ACTN3*	Typical
Response To Endurance Training	*PGC1A, NRF2, NFIA_AS2, ADRB3, GSTP1*	Enhanced
Pain Intolerance	*COMT*	Enhanced
Risk For ACL (Achilles Tendon) Injury	*COL5A1*	Typical
Risk For Rotator Cuff Injury	*MMP1*	High risk
Inflammation Risk, Antioxidant Defense, And Long-Term Wellness		
Interleukin 6 - Inflammation Risk	*IL6*	Typical
Risk For Oxidative Stress And Damage - Superoxide Dismutase 2 (SOD2)	*SOD2*	High risk
Nitric Oxide - Role In Vasodilation	*NOS3*	High risk

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Summary of Results

Trait	Genes	Result
Sleep Quality And Lifestyle		
Short Sleep Duration	*CLOCK*	Typical
Alcohol Sensitivity	*ALDH2*	Typical

High Risk Typical Enhanced Moderate Slightly Enhanced

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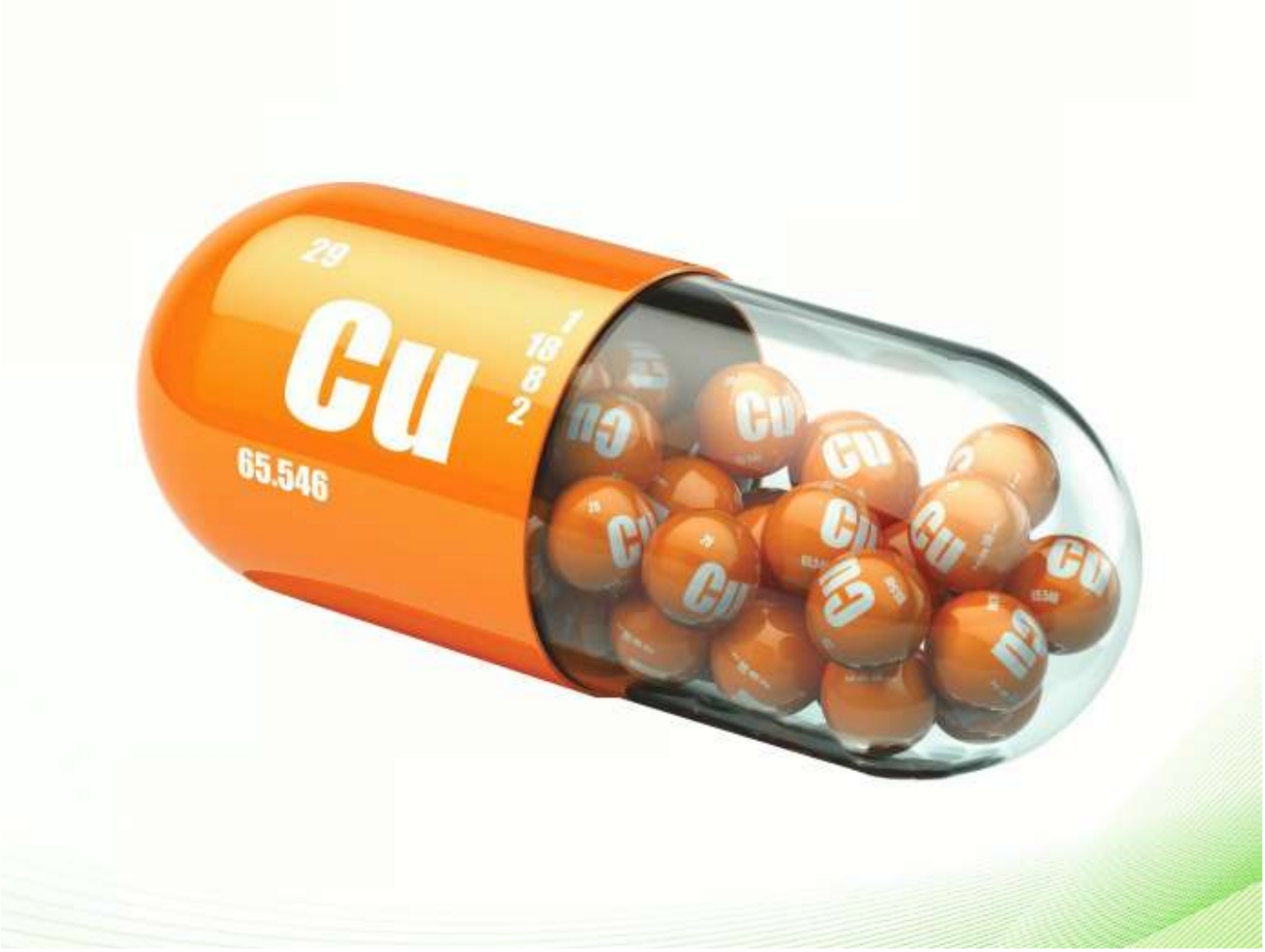
Vitamin And Mineral Metabolism

Ensuring optimal levels of vitamins and minerals is foundational to every aspect of health, from cellular energy production and DNA repair to immune competence and neurological function. This category of the NutriDNA panel that assesses variants in key transporters, enzymes, and receptors allows you to anticipate how your body handles nutrients like folate, vitamin D, and iron. Rather than relying on one-size-fits-all recommendations, you gain clarity on which micronutrients you may absorb poorly, convert inefficiently, or risk accumulating in excess.

With these insights, you can proactively tailor dietary choices and supplementation strategies to your unique needs. Monitor your B12 levels; if they fall below the normal range, consult a nutritionist or healthcare provider. Likewise, identifying a genetic predisposition to iron overload helps you track serum ferritin closely and adjust dietary iron intake before tissue damage occurs.



Folate Requirement

Folate, also known as vitamin B9, is a vital water-soluble nutrient required for cell growth, DNA synthesis, red blood cell production, and proper fetal development during pregnancy. A deficiency in folate has been linked to a higher risk of cardiovascular diseases, such as heart attacks and strokes. Research indicates that the absorption of dietary folate varies among individuals, even with identical intake levels. This variation is often influenced by genetic factors, particularly mutations in the *MTHFR* gene, which is crucial for metabolizing folate and other B vitamins. Inefficient folate processing can increase the likelihood of deficiency and associated health problems. Folate functions as a coenzyme or cosubstrate in single-carbon transfers in the synthesis of nucleic acids, metabolism of amino acids, conversion of homocysteine to methionine which is an important methyl donor in methylation cycle.

Functions

Helps form our genetic material (DNA/RNA)

Supports brain development during pregnancy and in infants

Helps our cell grow and multiply

Helps form the red blood cells

Keeps our brain and nervous system healthy

Balanced levels of homocysteine in the blood

MTHFR

The *MTHFR* gene product is an enzyme that helps in and is necessary for utilizing folate. This *MTHFR* enzyme converts dietary folate into a bioavailable intermediate. People with the CT, TT variant are known to have reduced enzyme activity and hence are at risk for folate deficiency and associated symptoms. List of symptoms (anemia, fatigue, gastrointestinal issues, loss of appetite, Mouth ulcers, Sore tongue, weakness, difficulty concentrating, Irritability, shortness of breath, changes in skin, hair, or fingernail pigmentation). High levels of blood homocysteine is an important indicator of this deficiency.

Sources Of Folate

Papaya

Lentils

Spinach

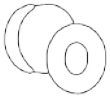
Asparagus

Broccoli

Chickpea

Folate Requirement

 Edamame

 Kiwi

YOUR GENES

Gene	rsID	Your Genotype
MTHFR	rs1801133	GG
MTHFR	rs1801131	TT

Genetic Impact

Based on the status of your *MTHFR* gene variants (both non-risk alleles), your risk of folate deficiency is similar to that of the general population.

Interpretation

Genetically, your enzyme activity for converting natural folate and folic acid to its active form (5-MTHF) remains unaffected. Therefore, your risk of folate deficiency is similar to that of the general population and is influenced more by dietary intake than genetics.

Recommendation

- Maintain a balanced diet with folate-rich foods (leafy greens, legumes, citrus fruits).
- No special supplementation needed unless dietary intake is inadequate.
- Regular health check-ups to monitor folate levels if necessary.

Vitamin A Requirement

Vitamin A refers to a group of fat-soluble retinoids, including retinol and retinyl esters, essential for vision, immune function, reproduction, and organ development. It supports cell growth and differentiation, playing a vital role in the eyes, heart, lungs, and other organs. Dietary sources include preformed vitamin A from animal products and provitamin A carotenoids from plants, which are converted into active form of vitamin A (retinol) by the enzyme *BCMO1*. Genetic variations in the *BCMO1* gene can affect this conversion and impact immune response pathways, cell growth, visual cycle pathway etc.

Functions

Supports our immune system

Supports healthy vision

Supports brain development during pregnancy and in infants

Helps our cell grow and multiply

BCMO1

Beta-carotene monooxygenase 1 (*BCMO1*) is a key enzyme involved in converting beta-carotene, the plant-derived precursor of vitamin A, into its active forms—retinal and retinoic acid. *BCMO1* is expressed in various human tissues and functions as a soluble protein. It cleaves beta-carotene retinal, which can then be further metabolized into retinoic acid, a crucial regulator of cellular activities. However, individuals carrying the GG genotype of the *BCMO1* gene exhibit a reduced ability to convert beta-carotene efficiently. As a result, they may need to ensure adequate intake of preformed vitamin A from animal-based sources or supplements to meet their nutritional needs.

Symptoms of Vitamin A Deficiency include Night blindness, Dry eyes, Increased susceptibility to infections, Delayed wound healing, Dry or rough skin, Impaired growth and development.

Sources Of Vitamin A

Cod liver oil

Cantaloupe

sweet potato

Bell peppers

Carrot

Butternut squash

Chicken liver

Tuna

Grapes

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Vitamin A Requirement

YOUR GENES

Gene	rsID	Your Genotype
BCMO1	rs11645428	GG

Genetic Impact

Based on your gene status (both risk alleles) for the *BCMO1* gene mutation, you are genetically at a high risk for Vitamin A deficiency.

Interpretation

Genetically, your *BCMO1* enzyme activity is reduced to 51%, which significantly impacts the body's ability to convert provitamin A carotenoids from fruits and vegetables into active Vitamin A. This reduced conversion efficiency may increase your risk of Vitamin A deficiency compared to the general population.

Recommendation

- Focus on increasing intake of preformed Vitamin A from animal sources like liver, fish, eggs, and dairy products.
- If vegetarian focus on carotenoid-rich colorful vegetables and fruits (carrots, spinach, kale, oranges, mango and butternut squash).
- Include fortified plant-based foods (e.g., fortified milk or cereals) to support Vitamin A intake.
- Monitor Vitamin A levels to ensure adequacy, as conversion from carotenoids may be less efficient especially if you have a diet low in animal-based foods.

Food Reactions, Intolerances, And Sensitivities

Undetected food intolerances and sensitivities can drive chronic inflammation, gastrointestinal distress, and nutrient malabsorption, eroding quality of life over time. This category of NutriDNA panel that reveals your genetic capacity to produce digestive enzymes (like lactase in adulthood), metabolize stimulants (such as caffeine), or handle dietary sodium gives you a roadmap for precision elimination or moderation. By understanding these predispositions in advance, you avoid the guesswork—and the unnecessary dietary restrictions—that often accompany an undiagnosed intolerance.
This proactive knowledge empowers you to maintain both comfort and nutritional adequacy. For instance, rather than broadly avoiding dairy, you can focus on fermented dairy sources or enzyme supplementation if you're genetically predisposed to lactose intolerance. Similarly, if your panel indicates slower caffeine clearance, you can time your intake to minimize sleep disruption and anxiety. In each case, you preserve dietary variety while safeguarding digestive and metabolic health.

Lactose Intolerance

Lactose intolerance occurs when the body produces insufficient amounts of lactase, the enzyme responsible for breaking down lactose, a sugar found in dairy products. Without enough lactase disruption of the lactose metabolic pathway occurs and undigested lactose ferments in the gut, leading to symptoms such as bloating, gas, diarrhea, and abdominal pain.Additionally, gut motility pathways may be affected, as the presence of undigested lactose increases osmolality in the gut, drawing water into the intestines and potentially causing diarrhea. Inflammatory pathways may also be triggered by the fermentation process, exacerbating the symptoms.This condition can significantly affect an individual's quality of life, requiring dietary modifications to avoid lactose-containing foods. Common strategies include the use of lactase supplements and careful label reading for processed foods containing lactose. Additionally, prebiotic and probiotic supplements may improve gut health and alleviate symptoms by promoting beneficial bacteria in the intestines.

MCM6

The *MCM6* gene contains regulatory elements that control the expression of the LCT gene, which encodes lactase, the enzyme responsible for breaking down lactose, the sugar found in milk and dairy products. The SNP (LCT-13910 G>A), located upstream of the LCT gene, determines whether lactase production is sustained after childhood. Individuals with the GG genotype typically exhibit lactase non-persistence, meaning lactase production declines after weaning, leading to lactose intolerance. Those with the AA genotype maintain lactase production into adulthood and can typically digest lactose without discomfort. Carriers of the GA genotype often have intermediate lactase activity and may tolerate small amounts of lactose, though sensitivity can vary.

Symptoms of lactose intolerance include Bloating, Diarrhea, Gas, Nausea, Pain in your abdomen, Stomach “growling” or rumbling sounds, and Vomiting.

Top Sources Of Lactose-Free And Low-Lactose foods

Fortified Yogurt

Almond milk

Cashew milk

Soymilk

Low fat cheese

Coconut milk

Paneer

Lactose Intolerance

YOUR GENES

Gene	rsID	Your Genotype
MCM6	rs4988235	GG

Genetic Impact

Based on your *MCM6* gene variant status (two risk alleles), you are genetically at high risk for lactose intolerance due to reduced lactase enzyme activity. You may experience digestive discomfort after consuming lactose-containing foods.

Interpretation

Genetically, you are at high risk for lactose intolerance due to reduced lactase production. You may experience digestive discomfort after consuming dairy products.

Recommendation

- Avoid or minimize lactose-containing foods (milk, ice cream, soft cheeses).
- Opt for lactose-free dairy, plant-based alternatives (almond, oat, soy milk), or hard cheeses and yogurt, which are naturally lower in lactose.
- Consider lactase enzyme supplements when consuming dairy based on advice from a health expert..
- Include other sources of calcium-rich foods as part of a balanced diet.

Salt Sensitivity

Sodium is an essential micronutrient that regulates blood pressure, blood volume, plasma volume, and cellular transport. While naturally present in foods like fresh meat, fish, and vegetables, excessive sodium intake, common with processed foods, can elevate blood pressure and increase the risk of hypertension, heart disease, strokes, and kidney failure. Hypertension(blood pressure above 140/90 mm Hg) is linked to sodium sensitivity where high sodium intake quickly raises blood pressure. Conversely, salt-resistant individuals show minimal blood pressure changes despite high sodium consumption.The relationship between sodium and blood pressure involves genetic factors, notably variations in the *ACE* (angiotensin-converting enzyme) gene, which modulates sodium sensitivity by influencing pathways like the renin-angiotensin-aldosterone system (RAAS).This system regulates fluid balance, vascular resistance, and sodium retention, all critical to blood pressure control.

Functions

Helps our cells take up oxygen and nutrition

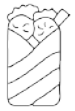
ACE

The *ACE* gene encodes the angiotensin-converting enzyme (*ACE*), which plays a central role in the renin-angiotensin system (RAS) - a hormonal pathway that regulates blood pressure, fluid balance, and vascular tone. The *ACE* SNP (G>A) affects *ACE* activity, where the A allele is associated with higher *ACE* enzyme levels, leading to increased vasoconstriction and sodium retention. Individuals with the GA or AA genotypes are more likely to be salt-sensitive, meaning that high sodium intake significantly increases their risk of developing elevated blood pressure. In contrast, GG genotype carriers generally have normal salt handling and a lower genetic predisposition to salt-induced hypertension.

High sodium levels in the body can cause symptoms like Excessive thirst, Confusion, Muscle twitching or Spasms, Bloating, Swelling in extremities, and severe cases, seizures.

Salt-Heavy food sources you should reduce

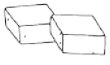
Fast food



Burritos

French fries

Ketchup



Soup cubes

Salt Sensitivity

YOUR GENES

Gene	rsID	Your Genotype
ACE	rs4343	GG

Genetic Impact

Based on your *ACE* gene variant status(non-risk allele), your response to dietary salt is typical. Standard sodium intake guidelines are appropriate for you.

Interpretation

Genetically, your *ACE* activity is typical, and your salt sensitivity is likely normal. Your risk for salt-induced hypertension is similar to that of the general population.

Recommendation

- Follow general sodium guidelines (≤2300 mg/day).
- Maintain regular physical activity and a balanced diet rich in potassium (bananas, spinach, beans).

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Optimizing Weight Loss And Long Term Weight Management

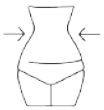
Weight management is not purely a matter of willpower or general calorie counting—it's intricately tied to your genetic profile, which shapes how you respond to macronutrient ratios, exercise modalities, and behavioral cues. This category of NutriDNA panel assessment reveals whether you may thrive on higher-protein diets, benefit most from particular fat types, or require specific exercise prescriptions to maximize fat oxidation. This precision approach sidesteps trial-and-error dieting and helps you adopt strategies that align with your innate metabolic wiring.

Over the long term, these insights guide sustainable habits rather than short-lived fixes. By aligning meal timing, training intensity, and nutrient composition with your genetic tendencies, you reduce frustration, minimize plateaus, and lower the risk of weight gain. The result is a personalized roadmap that supports not just initial weight loss but the maintenance of a healthy weight for years to come.

Adiponectin Levels

Adiponectin is a hormone produced by fat cells (adipocytes) that plays a key role in regulating metabolism, insulin sensitivity, and inflammation. Unlike many other hormones produced by fat cells, adiponectin has beneficial effects on the body. It helps increase the body's sensitivity to insulin, improve fat breakdown, and reduce inflammation, which is important for overall metabolic health. Higher levels of adiponectin are typically associated with a lower risk of developing conditions like type 2 diabetes, heart disease, and obesity-related complications. However, low levels of adiponectin are often found in individuals with obesity and metabolic disorders. Adiponectin's role in regulating various aspects of metabolism makes it a significant focus of research in understanding and managing metabolic diseases.

Functions



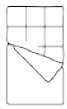
Helps with better metabolism and weight loss

Improve mood

ADIPOQ

The *ADIPOQ* gene, located on chromosome 3, encodes adiponectin, a hormone primarily secreted by adipocytes that regulates insulin sensitivity, lipid metabolism, and inflammation. It is also known as the fat-burning hormone. Genetic variations within the *ADIPOQ* gene have been shown to significantly affect the circulating levels of adiponectin. The *ADIPOQ* SNP (G>A) is located in the promoter region of the *ADIPOQ* gene and is associated with reduced adiponectin expression. The risk allele affects transcription factor binding in the promoter region of the *ADIPOQ* gene, resulting in decreased gene expression. This lowers adiponectin levels, impairing its beneficial metabolic effects, including enhancing insulin sensitivity, promoting fat oxidation, and suppressing inflammation. As a result, risk allele carriers may be more prone to fat accumulation, insulin resistance, and low-grade systemic inflammation. Understanding these genetic variants is critical, as they influence individual responses to diet and lifestyle interventions aimed at improving metabolic health.

Dietary Sources That Enhance Adiponectin



Dark chocolate

Apple cider vinegar

Kale

Bell peppers

Cinnamon

Ginger

Avocados

Adiponectin Levels

YOUR GENES

Gene	rsID	Your Genotype
ADIPOQ	rs17366568	GG

Genetic Impact

Based on your *ADIPOQ* gene variant status (non-risk allele), your adiponectin levels are likely within the normal range. Therefore, your risk for adiponectin-related metabolic issues is similar to that of the general population.

Interpretation

Genetically, your *ADIPOQ* gene expression is likely normal, supporting healthy adiponectin levels and metabolic balance. Your risk for adiponectin-related metabolic issues is similar to that of the general population.

Recommendation

- Maintain a balanced diet and regular physical activity to support glucose and lipid metabolism.
- Routine health checks; no specific dietary intervention needed beyond general wellness.

Weightloss Response To Physical Activity

Physical activity is essential for weight loss and overall health, improving mental well-being, fitness, and preventing chronic diseases. It activates key metabolic pathways that boost energy expenditure, fat oxidation, and muscle growth. Cardiovascular exercises like running or cycling trigger the AMP-activated protein kinase (AMPK) pathway, which breaks down fat and enhances glucose uptake in muscles, improving insulin sensitivity. Resistance training stimulates the mTOR pathway, boosting muscle protein synthesis and increasing lean muscle mass, which raises basal metabolic rate. Together, these pathways enhance fat metabolism and muscle development, promoting more effective weight loss. Regular exercise is crucial for reducing body fat, improving metabolic health, and supporting sustainable weight management.

FTO, ADRB2

FTO is a key gene linked to body weight regulation. Variants in this gene can influence appetite and metabolic efficiency. However, individuals with higher-risk *FTO* profiles can significantly benefit from regular physical activity, which has been shown to greatly reduce genetic predisposition to weight gain. These individuals often show a strong response to well-structured exercise programs.

The *ADRB2* gene encodes the beta-2 adrenergic receptor, which is the dominant adrenergic receptor in adipose tissue and plays a key role in lipolysis, mobilizing stored fat in response to catecholamines like epinephrine. The Arg16Gly polymorphism results in an amino acid change at position 16, impacting receptor regulation. Functional studies have shown that the Gly16 variant leads to enhanced downregulation of the receptor in response to agonist stimulation, potentially reducing lipolytic efficiency during exercise. In contrast, individuals with the Arg16 genotype retain greater receptor availability and signaling, resulting in more effective fat breakdown during physical activity.

The combination of *FTO* and *ADRB2* profiles helps predict how effectively someone may respond to exercise for weight management.

Weightloss Response To Physical Activity

YOUR GENES

Gene	rsID	Your Genotype
FTO	rs9939609	TT
ADRB2	rs1042713	GG

Genetic Impact

Based on your *FTO* (TT) and *ADRB2* (GG) variant status, you have a modestly enhanced weight-loss response to exercise; slightly upping workout duration or intensity can help you tap into this advantage.

Interpretation

Genetically, you carry the *ADRB2* Gly (G) allele in homozygosity, which boosts exercise-induced fat breakdown. You can expect a slightly enhanced weight-loss response to physical activity.

Recommendation

- Target 30minutes/day/week of moderate aerobic exercise (brisk walking, cycling) plus 2 resistance sessions.
- Include steady-state cardio (30–45 min/session) and occasional HIIT to leverage A*DRB2* Gly-driven fat mobilization.
- Pair workouts with balanced meals (low-GI carbs, lean protein, healthy fats) to stabilize appetite.
- Reassess every month and gradually increase workout intensity to sustain progress.

Satiety

Satiety is the feeling of fullness and satisfaction after eating that signals the body to stop consuming more food. It plays a key role in regulating energy intake and preventing overeating. Satiety is influenced by various factors including the type of food consumed (protein and fiber-rich foods enhance satiety), hormonal signals (like leptin and ghrelin), and genetic variations that affect appetite control pathways in the brain.Disruptions in satiety signaling can lead to increased hunger, frequent cravings, and weight gain — especially when combined with high-calorie diets or sedentary lifestyles. Understanding your genetic predisposition to satiety can help tailor dietary strategies for better appetite management and long-term weight control.

FTO

The *FTO* gene (Fat Mass and Obesity-Associated gene) is one of the most well-studied genes linked to body weight regulation. It plays a role in energy balance, appetite control, and satiety signaling, particularly through effects on the hypothalamus, the brain region that regulates hunger. Presence of the *FTO* risk allele is studied to be associated with increased appetite, reduced satiety after meals, Higher preference for calorie-dense foods, greater risk of weight gain and obesity, especially in environments with high food availability. This is believed to happen as it influences the expression of hunger-regulating neuropeptides such as ghrelin and leptin.

Satiety

YOUR GENES

Gene	rsID	Your Genotype
FTO	rs9939609	TT

Genetic Impact

Based on your *FTO* gene variant status (non-risk allele), your satiety and appetite regulation are likely typical. Your risk for overeating and hunger-driven weight gain is similar to that of the general population.

Interpretation

Genetically, your *FTO* gene function is likely typical, supporting normal appetite regulation and satiety signaling. Your risk for excessive hunger and obesity is similar to that of the general population.

Recommendation

- Standard dietary habits are usually sufficient for maintaining energy balance.
- Continue regular physical activity and portion control for overall wellness.

Fat Taste Perception

Fat is universally palatable due to its desirable properties in smell, texture, and taste. It dissolves flavor chemicals, releasing them when heated, which enhances the sensory experience of food. Fatty foods also provide a unique mouthfeel, with emulsions contributing to the creamy texture of items like ice cream, peanut butter, and chocolate. Food intake is largely determined by our taste perceptions, with fat taste perception playing a key role. Fat is essential for absorbing vitamins A, D, E, and K and provides more than double the energy from carbohydrates or proteins. Our preference for fatty foods can be influenced by genetics, affecting how we perceive fat and make dietary choices. However, excessive fat consumption, especially unhealthy fats, can lead to heart health issues and altered body composition. Balancing fat intake with healthier options like monounsaturated fats is important for weight management and overall health.

CD36

The *CD36* gene codes for a receptor involved in detecting fatty acids in food. When fatty acids enter the mouth, they bind to *CD36* receptors on the taste receptor cells in the tongue. This binding activates a signaling pathway that sends a message to the brain and digestive system, triggering the release of digestive enzymes (e.g., lipases) and satiety hormones (such as PYY) that help process the fat and regulate food intake.

Carriers of the 'A' allele (AA genotype) of the *CD36* gene exhibit reduced expression of these receptors on their tongues, making it more difficult to detect fatty acids. As a result, they may compensate by consuming more fat to achieve the same sensation of fullness. Additionally, impaired fat detection in these individuals leads to a weakened satiety response, characterized by lower levels of the hormone PYY, which normally signals fullness after fat consumption. This combination of reduced sensitivity to fatty acids and impaired satiety could contribute to overeating, higher fat intake, and potentially increased body mass index (BMI) and obesity risk in 'AA' carriers.

Foods that enhance Fat Taste Perception

Olive oil

Salad dressing

Fat Taste Perception

YOUR GENES

Gene	rsID	Your Genotype
CD36	rs1761667	GG

Genetic Impact

Based on your gene status (two favourable alleles), you are likely a "super-taster" for fat. Your heightened sensitivity to fatty flavors naturally discourages overindulgence in high-fat foods, helping you maintain a balanced fat intake.

Interpretation

Genetically, your fat taste receptors are highly sensitive, allowing you to detect even small amounts of fat in foods more acutely than average.

Recommendation

- Aim for 20–35% of calories from fats, prioritizing monounsaturated and polyunsaturated sources.
- Use small amounts of avocado, nuts, and olive oil to meet essential fatty-acid needs without overwhelming your palate.
- Since you detect richness easily, be mindful of dressings, sauces, and spreads—opt for lighter or oil-free versions.
- Pair fatty foods with high-fiber vegetables and lean proteins to create satisfying, nutrient-dense meals.
- Include diverse fat sources (seeds, fatty fish, plant oils) to ensure adequate omega-3 and omega-6 intake.

Nutricode

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Factors Influencing Metabolic Health And Associated Risk Levels

Cardiometabolic conditions such as dyslipidemia, insulin resistance, and hypertension emerge from complex interactions between genes and the environment. This category of NutriDNA panel is evaluating variants in cholesterol regulators, glucose-metabolism enzymes, and inflammatory mediators that empower you to stratify your risk well before clinical symptoms appear. Armed with this foresight, you can prioritize the most impactful lifestyle adjustments—whether that's refining fat quality, moderating carbohydrate load, or enhancing dietary fiber—to counteract your specific genetic susceptibilities.

Rather than applying generic "heart-healthy" or "low-glycemic" labels, you learn exactly which dietary and exercise modifications will yield measurable improvements in your lipid panels, glycemic control, and inflammatory markers. Regular follow-up testing then confirms efficacy and allows dynamic fine-tuning, turning a once-reactive healthcare journey into a proactive, data-driven strategy.

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Response To Whole Grain Intake

Whole grains, such as oats, brown rice, and whole wheat, are low-glycemic, high-fiber carbohydrates that help regulate blood sugar more effectively than refined grains. Rich in essential micronutrients like magnesium, folic acid, and vitamin E, they support metabolic health. The fiber in whole grains activates AMP-activated protein kinase (AMPK), improving insulin sensitivity and glucose uptake. It also promotes beneficial gut bacteria that produce short-chain fatty acids (SCFAs), enhancing insulin sensitivity and reducing inflammation. Whole grains reduce chronic inflammation, a key factor in insulin resistance, and support healthy weight management. Additionally, magnesium enhances insulin signaling, improving glucose regulation. Consuming whole grains has been linked to improved blood glucose control and a reduced risk of type 2 diabetes, particularly in high-risk individuals.

Functions

Regulates blood sugar

Healthy teeth and oral health

Reduces cancer risk

TCF7L2

The *TCF7L2* gene encodes the transcription factor 7-like 2 protein factor which is a member of the TCF/LEF family and is involved in Wnt signaling, which influences insulin secretion and beta-cell function. In the C>T variant T allele impairs insulin production and raises type 2 diabetes risk, particularly when dietary carbohydrates are predominantly high glycemic index (GI). Consuming whole grains, rich in fiber and low GI, improves glycemic control by slowing glucose absorption and reducing the insulin demand that T-allele carriers struggle to meet.

Whole Grain Food Options

Brown rice

Barley

Buckwheat

Response To Whole Grain Intake

YOUR GENES

Gene	rsID	Your Genotype
TCF7L2	rs7903146	CC

Genetic Impact

Based on your *TCF7L2* variant status (no risk allele), your insulin function is typical, and standard whole-grain intake can be helpful.

Interpretation

Genetically, you carry no risk allele for this variant and thus, have typical insulin secretion. Your risk for type 2 diabetes aligns with standard population levels.

Recommendation

- Follow general advice: 45–50% of calories from carbohydrates, favoring whole grains over refined sources.
- Maintain regular exercise to support insulin sensitivity.

Response To MUFA Intake

Monounsaturated fats are a type of healthy unsaturated fat found in foods like olive oil, avocados, nuts, and seeds. These fats are liquid at room temperature and have been shown to offer numerous health benefits, particularly for heart health. They help reduce levels of LDL cholesterol (the "bad" cholesterol) while increasing HDL cholesterol (the "good" cholesterol), thereby supporting cardiovascular health. Additionally, monounsaturated fats are rich in antioxidants that help reduce inflammation in the body. For weight loss and body fat reduction, incorporating monounsaturated fats into the diet can be beneficial, as they promote satiety and help regulate appetite, making it easier to maintain a calorie-controlled diet. These fats also support healthier fat metabolism, contributing to improved body composition when combined with regular physical activity. As part of a balanced diet, monounsaturated fats can aid in weight management by reducing overall body fat and promoting fat loss without sacrificing nutrition.

Functions

Reduces join pain

PPARG2

The PPARG2 isoform of the PPARG gene encodes peroxisome proliferator-activated receptor gamma 2, a nuclear receptor that senses fatty acids and regulates genes involved in adipocyte differentiation, insulin sensitivity, and lipid metabolism. The (Pro12Ala) C>G variant enhances PPARG2's ability to bind monounsaturated fatty acids (MUFAs) as ligands. Individuals carrying at least one G allele (CG or GG) demonstrate greater activation of PPARG2 in response to a MUFA-rich diet, promoting improved insulin action, more efficient fat oxidation, and better weight-loss or maintenance outcomes. CC homozygotes display the standard receptor responsiveness and derive typical benefits from MUFA intake.

Sources of Monounsaturated Fats

Almond butter

Walnut

Olive oil

Avocados

Response To MUFA Intake

YOUR GENES

Gene	rsID	Your Genotype
PPARG2	rs1801282	CC

Genetic Impact

Based on your PPARG2 variant status (CC), your response to dietary MUFAs is typical; you should follow general healthy-fat guidelines to support metabolic health.

Interpretation

Genetically, your PPARG2 activity is normal; incorporating MUFAs supports health similarly to the general population.

Recommendation

- Include MUFA sources (olive oil, avocado, nuts) as part of a balanced diet (20–35% of calories from fat, with ≤10% saturated fats).
- Combine MUFA-rich meals with whole grains, lean proteins, and fiber for overall metabolic health.
- Maintain regular physical activity (150 min/week moderate exercise).

Nutricode
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Optimizing Exercise Performance, Fitness, And Managing Injury Risk

Performance potential and injury susceptibility are deeply influenced by genetic variation in muscle fiber composition, connective-tissue integrity, and inflammatory response. By assessing markers for power versus endurance aptitude, collagen strength, and cytokine regulation, a nutrigenomic panel guides both the intensity and type of training most beneficial for you. This precision ensures you capitalize on your strengths—whether explosive lifting or long-distance running—while minimizing the risk of overuse injuries and chronic inflammation.

Moreover, knowing your genetic recovery profile enables smarter periodization and recovery strategies. Those with slower antioxidant enzyme activity may benefit from additional rest days or targeted nutritional support, whereas individuals with robust healing genotypes can maintain a higher training frequency. Such data-driven planning transforms generic workout plans into individualized performance blueprints.

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Motivation To Exercise

Exercise motivation varies significantly between individuals due to a combination of physiological, psychological, and genetic factors. While some people are naturally inclined to stay active, others struggle to get started or maintain consistency.Key factors influencing exercise initiation include self-perception, social support, and positive feedback. Once a person starts exercising, continued motivation is driven by biological characteristics, social influences, psychological factors like self-confidence, and behavioral habits. Additionally, genetic factors play a role in determining energy levels and mood, impacting long-term exercise adherence.

BDNF

BDNF encodes brain-derived neurotrophic factor, a peptide that supports neuronal growth, synaptic plasticity, long-term memory, and the "reward" circuitry that makes physical activity feel good. The variant ValMet (C > T) causes changes in the protein structure, leading to reduced secretion of the *BDNF* protein. Carriers of the T allele (CT or TT) release *BDNF* more efficiently during exercise and typically report a stronger post-exercise mood boost and greater drive to be active. Individuals with the CC genotype produce functional *BDNF* but do not receive the same motivational uplift, so they may need extra behavioural cues to stay consistent with exercise.

Motivation To Exercise

YOUR GENES

Gene	rsID	Your Genotype
BDNF	rs6265	CC

Genetic Impact

Based on your *BDNF* gene variant status(normal allele), your exercise motivation is average, as the normal population — setting reminders, training with partners, and tracking progress can help you stay consistent.

Interpretation

Genetically, you may have normal *BDNF* secretion and no additional motivation to exercise, and a higher likelihood of weight regain without structured activity plans.

Recommendation

- Exercise may feel harder—schedule workouts, add variety, and lean on support systems to stay engaged
- Use accountability partners, group classes, or training apps to stay on track.
- Choose music, gamified fitness, or varied sessions (e.g., dance, circuits) to raise enjoyment.
- Start with moderate intensity and build gradually to keep perceived effort manageable.
- Celebrate small wins; visible progress reinforces habit.

Exercise Behavior

Exercise behavior includes various factors such as intention, attitude, duration, and frequency of physical activity. While athletes and fitness enthusiasts often maintain consistent habits, many individuals struggle to stay committed. Despite the well-established physical and mental health benefits of regular exercise, such as disease prevention, improved mood, and better sleep, reports show that over 70% of Indian adults do not engage in adequate physical activity, falling short of global recommendations. Exercise behavior is shaped by genetics, motivation, personal preferences, and external factors like time, finances, and access to resources. Barriers like lack of motivation, tiredness, soreness, and health issues can hinder consistency, making it crucial to address these challenges for long-term commitment to physical activity.

CYP19A1, LEPR

The *CYP19A1* gene encodes aromatase, which converts androgens into estrogens; A-allele carriers for the variant(G>A) have higher aromatase activity, modestly boosting estrogen-mediated reward from physical activity. The *LEPR* gene encodes the leptin receptor, which regulates appetite and energy balance; T-allele carriers (G>T variant) exhibit slightly reduced leptin sensitivity, which can paradoxically increase the drive to move as the body seeks energy balance. Together, favorable alleles in both genes correlate with greater intrinsic drive to exercise.

Exercise Behavior

YOUR GENES

Gene	rsID	Your Genotype
CYP19A1	rs2470158	AA
LEPR	rs12405556	TT

Genetic Impact

Based on your gene status (both favourable alleles) (AA)*CYP19A1* and (TT)*LEPR*, you inherit both enhancers of exercise drive and are very likely to enjoy and stick with physical activity.

Interpretation

Genetically, your combined high-activity aromatase and leptin-receptor variants amplify reward and enhanced motivation during exercise.

Recommendation

- Capitalize on your natural motivation: schedule regular workouts (5×/wk) and set progressive challenges.
- Mix resistance, HIIT, and endurance to keep reward pathways engaged.
- Use goal setting: leverage fitness trackers and milestones to reinforce activity habits.
- Monitor recovery: don't overtrain—rest days and sleep support sustained engagement.

Inflammation Risk, Antioxidant Defense, And Long-Term Wellness

Chronic inflammation and oxidative stress drive aging, neurodegeneration, and a host of chronic diseases. This category of NutriDNA panel examines variants in pro- and anti-inflammatory cytokines (such as IL-6) and antioxidant enzymes (like SOD2), pinpointing where your internal defense systems may falter. This knowledge allows you to deploy precise nutritional and lifestyle countermeasures, such as increasing specific phytonutrients or tailoring exercise intensity, to counteract genetic vulnerabilities.

Rather than generic antioxidant supplementation, you learn which cofactors (e.g., zinc, manganese) your body requires most. You also adopt targeted anti-inflammatory dietary patterns (like enhanced omega-3 intake) when your genes predispose you to heightened inflammatory signaling. Over time, this personalized approach supports resilience against oxidative damage and fosters healthier aging trajectories.

Interleukin 6 - Inflammation Risk

Interleukin-6 (IL-6) is a protein that helps the body respond to stress, injury, or infection. It acts as a messenger, signaling the immune system to fight infections or repair damage. IL-6 is often called an inflammation marker because its levels increase when there's inflammation in the body, such as during infections, injuries, or certain chronic conditions like arthritis or obesity. While it helps in healing and defense, too much IL-6 over time can contribute to long-term inflammation, which is linked to diseases like diabetes and heart problems. Interestingly, during exercise, IL-6 also plays a helpful role, supporting energy use and muscle repair. Its ability to both help and harm makes IL-6 a key marker for understanding and managing health and inflammation.

IL6

The *IL6* gene plays a central role in regulating inflammatory pathways by encoding the cytokine interleukin-6 (IL-6), a key mediator in immune response and inflammation. The gene variant (–174 G>C) is located in the promoter region of the *IL6* gene and is known to influence gene expression. The G allele has been associated with increased promoter activity, leading to higher IL-6 production. Elevated IL-6 levels contribute to chronic low-grade inflammation, which is linked to an increased risk of cardiovascular disease, obesity, insulin resistance, and other inflammatory conditions.

High levels of Interleukin-6 (IL-6), a key inflammatory cytokine, can cause a range of symptoms, including fever, joint pain, fatigue, and mood changes.

Foods That Combat Inflammation


Kale


Cinnamon


Ginger


Chia seeds


Blueberries

Interleukin 6 - Inflammation Risk

YOUR GENES

Gene	rsID	Your Genotype
IL6	rs1800795	CC

Genetic Impact

Based on your *IL6* gene variant status (non-risk allele), your baseline inflammatory response is likely typical. Your risk for chronic inflammation is similar to that of the general population.

Interpretation

Genetically, your *IL6* gene promoter activity is not enhanced. Therefore, your IL-6 production and inflammation levels are likely within normal range, and your risk for chronic inflammation is similar to that of the general population.

Recommendation

- No specific intervention required beyond general health maintenance.
- General anti-inflammatory lifestyle is sufficient: regular exercise, antioxidant-rich diet, and stress management.

Risk For Oxidative Stress And Damage - Superoxide Dismutase 2 (SOD2)

Superoxide Dismutase 2 (*SOD2*) is an important antioxidant enzyme found in the mitochondria, the energy-producing centers of cells. Its primary role is to protect cells from damage caused by oxidative stress, a process where harmful molecules called reactive oxygen species (ROS) build up and can harm cells and tissues. *SOD2* specifically targets superoxide radicals, a type of ROS, by converting them into less harmful substances like hydrogen peroxide, which are further broken down by other enzymes. *SOD2* is essential for maintaining healthy cellular function and plays a protective role in conditions involving oxidative stress, such as aging, cardiovascular diseases, neurodegenerative disorders, and certain cancers. Its activity is influenced by both genetic and environmental factors, making it a key focus in research on oxidative damage and chronic diseases.

Functions

Reduces cancer risk

SOD2

The *SOD2* gene encodes the mitochondrial enzyme superoxide dismutase 2, which plays a key role in the body's defense against oxidative stress by converting harmful superoxide radicals into hydrogen peroxide. The Val16Ala variant results in a substitution in the enzyme's mitochondrial targeting sequence, potentially altering enzyme structure and import efficiency. The Val variant is believed to impair the proper transport of the *SOD2* enzyme into mitochondria, where it is needed to neutralize superoxide radicals. This mislocalization or reduced import efficiency results in lower mitochondrial *SOD2* activity, leading to a buildup of reactive oxygen species (ROS). Over time, this can cause increased oxidative damage, impaired cellular function, and heightened vulnerability to inflammation, aging, and chronic diseases due to reduced antioxidant defense at the mitochondrial level.

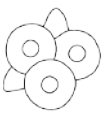
This excess oxidative stress may contribute towards the development or exacerbation of conditions like Polycystic Ovary Syndrome (PCOS), Coronary Heart Disease (CHD), etc.

Sources Of Antioxidant-Rich Foods

Ginger

Chia seeds

Green Tea



Blueberries

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Risk For Oxidative Stress And Damage - Superoxide Dismutase 2 (SOD2)

YOUR GENES

Gene	rsID	Your Genotype
SOD2	rs4880	AA

Genetic Impact

Based on your *SOD2* gene variant status (two risk alleles), you are genetically at high risk for impaired antioxidant activity in mitochondria. This may increase your vulnerability to oxidative stress and cellular damage, especially during aging, intense exercise, or chronic inflammation.

Interpretation

Genetically, your *SOD2* antioxidant activity in mitochondria is low. This may increase your vulnerability to oxidative stress and cellular damage, especially during aging, intense exercise, or chronic inflammation.

Recommendation

- Strongly prioritize antioxidant support: polyphenols, flavonoids, CoQ10, selenium, vitamin C/E.
- Avoid environmental toxins (pollution, smoking); moderate alcohol intake.
- Consider antioxidant supplementation in consultation with a nutrition specialist.
- Consider lifestyle habits that minimize inflammation (sleep, hydration, stress management).

Sleep Quality And Lifestyle

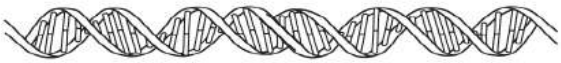
Restorative sleep underpins cognitive performance, metabolic regulation, and emotional resilience, yet genetic differences in circadian rhythm genes (CLOCK) and alcohol-metabolism enzymes (like ALDH2) can make conventional sleep hygiene tips insufficient. This category of NutriDNA panel reveals whether you naturally lean toward shorter or fragmented sleep, and how substances such as caffeine or alcohol will impact your rest. Armed with this insight, you can craft a sleep environment and routine that truly aligns with your biology.

For instance, evening light exposure, meal timing, and beverage choices can be optimized based on your genetic predispositions, transforming trial-and-error approaches into data-backed sleep strategies. By personalizing your nightly routine down to these molecular determinants, you ensure each night's rest is as restorative as possible, fueling better mood, sharper focus, and stronger metabolic health.

Short Sleep Duration

Short sleep duration, typically defined as getting less than 7 hours of sleep per night, is associated with a range of negative health outcomes. Chronic sleep deprivation can impair cognitive function, mood, and decision-making, while also increasing the risk of developing conditions like obesity, diabetes, and cardiovascular disease. It disrupts metabolic processes, including the regulation of hunger hormones like ghrelin and leptin, leading to increased appetite and cravings for high-calorie foods. Additionally, short sleep duration has been linked to decreased insulin sensitivity, weight gain, and impaired immune function, making it an important factor to address for overall health and well-being.

CLOCK



The *CLOCK* gene (among other genes) plays a central role in regulating the body's circadian rhythm — the internal biological clock that controls sleep-wake cycles, hormone secretion, and metabolism. The 3111T>C gene variant can influence gene expression and circadian timing, affecting duration, chronotype, and risk of sleep disorders, fatigue, and metabolic dysregulation. Mechanistically, this variant may delay the expression of circadian genes, pushing the body's natural sleep phase later and reducing total sleep duration, particularly in environments with irregular schedules or artificial light exposure. Short sleep, or insufficient sleep, can manifest in various symptoms, affecting cognitive and behavioral outcomes like Difficulty focusing and concentrating, slowed reaction times, Trouble with learning and memory, Poor judgment, Increased errors and accidents, and extreme Fatigue.

Short Sleep Duration

YOUR GENES

Gene	rsID	Your Genotype
CLOCK	rs1801260	AA

Genetic Impact

Based on your *CLOCK* gene variant status (non-risk allele), your risk for short sleep duration is similar to that of the general population.

Interpretation

Genetically, your *CLOCK* gene does not carry the risk variant associated with reduced sleep duration. Your circadian rhythm regulation is likely unaffected, and your risk for short sleep duration is similar to that of the general population.

Recommendation

- Likely to maintain regular sleep duration (7–8 hours).
- General sleep hygiene is sufficient: consistent bedtime, limited screen exposure before bed.

Alcohol Sensitivity

Alcohol sensitivity, often known as the "alcohol flush effect," is a reaction some individuals experience after consuming alcohol. This effect is marked by redness and warmth, particularly on the face and neck, accompanied by symptoms like nausea, dizziness, and a rapid heartbeat. The flush occurs due to an inability to properly metabolize alcohol, often due to genetic differences in enzymes involved in alcohol breakdown. One of the key enzymes, aldehyde dehydrogenase (ALDH), plays a critical role in breaking down acetaldehyde, a toxic byproduct of alcohol metabolism. When this enzyme is less active or absent, acetaldehyde builds up in the body, causing the flush and other associated symptoms. This genetic variation can lead to discomfort and, in some cases, an increased risk of certain health issues.

ALDH2

The *ALDH2* gene encodes the enzyme aldehyde dehydrogenase 2, which is essential for breaking down acetaldehyde, a toxic intermediate produced during alcohol metabolism. A change in this gene (Glu504Lys) results in a functional variant known as *ALDH2**2. This variant dramatically reduces enzyme activity and can cause anywhere between 30% to complete loss of enzyme function. Studies also report that risk carriers may have 6–19 times higher blood levels of acetaldehyde after drinking compared to non-carriers. This buildup is not only uncomfortable but also carcinogenic, increasing the risk of esophageal cancer, gastric irritation, and liver damage even at low alcohol intake. As a result of acetaldehyde accumulates rapidly after alcohol consumption, leading to unpleasant symptoms such as facial flushing, nausea, rapid heartbeat, and dizziness — commonly referred to as "alcohol flush reaction".Alcohol sensitivity, often known as the "alcohol flush effect," is a reaction some individuals experience after consuming alcohol. This effect is marked by redness and warmth, particularly on the face and neck, accompanied by symptoms like nausea, dizziness, and a rapid heartbeat. The flush occurs due to an inability to properly metabolize alcohol, often due to genetic differences in enzymes involved in alcohol breakdown. One of the key enzymes, aldehyde dehydrogenase (ALDH), plays a critical role in breaking down acetaldehyde, a toxic byproduct of alcohol metabolism. When this enzyme is less active or absent, acetaldehyde builds up in the body, causing the flush and other associated symptoms. This genetic variation can lead to discomfort and, in some cases, an increased risk of certain health issues.

Alcohol Sensitivity

YOUR GENES

Gene	rsID	Your Genotype
ALDH2	rs671	GG

Genetic Impact

Based on the status of your *ALDH2* gene variants (non-risk allele), your risk for alcohol sensitivity (alcohol flush reaction) is similar to that of the general population.

Interpretation

Genetically, your *ALDH2* enzyme activity for breaking down acetaldehyde remains unaffected. Therefore, your risk for the buildup of acetaldehyde is normal, and your risk for alcohol sensitivity (alcohol flush reaction) is similar to that of the general population.

Recommendation

- You are likely to tolerates alcohol at usual levels (within recommended limits).
- Long-term health risks of alcohol (e.g., liver disease) still apply with chronic use. Hence, best is to avoid.

LIMITATION DETAILS

As with any laboratory test, there is a small chance that this result may be inaccurate for a procedural reason, such as an error during specimen collection and labeling (incorrect patient identification), an error in processing, data collection, or interpretation. Currently available data indicates that technical error rate for analysis involving DNA tests is anywhere between 1-3%. Variants that have not been confirmed by an independent analysis could represent technical artifacts. However, our validation study (Document No. VR-017v1) showed 100% concordance between the results obtained by NGS data and Sanger sequencing (confirmation of the variant), when the supporting read fraction of the variant with at least 20 reads was >30%.

Large insertions, deletions, duplications, inversions, repeat expansions and complex rearrangements cannot be characterized accurately by NGS as it uses short-read sequencing data. Such structural variants have a much higher false- positive and false-negative rate than seen for SNVs (single nucleotide variant). It is possible that the genomic region where a disease causing variant exists in the person being tested (which may impact the phenotype) was not captured using the current technologies and therefore was not detected.

For an autosomal dominant condition, if the variant does not seem to be inherited from parents, it could be due to a de novo (new) event or due to a germline mosaicism in an unaffected parent. In case of germline mosaicism, there is a risk of the disease recurrence in the family. However, due to technical limitations of this test, germline mosaicism cannot be determined by this test. Additionally, it is possible that a particular genetic abnormality may not be recognized as the underlying cause of the genetic disorder due to incomplete scientific knowledge about the function of all genes in the human genome and the impact of variants on those genes.

Not all variants detected may be listed in the report. Inclusion of variants is dependent upon our assessment of their significance. The quality of sequencing and coverage varies between regions. Specific genomic regions, such as homopolymers, tandem repeat sequence, GCrich regions, high sequence homology, etc. influence the quality of sequencing and coverage. This may result in an occasional error in sequence reads or lack of detection of a particular genetic lesion. Accurate interpretation of this report is dependent on detailed clinical history of the patient. In the event of unavailability of detailed clinical history, the lab cannot guarantee the accuracy of the interpretation. This report is strictly not a medical diagnostic report and shall not be construed as the medical certificate or medical laboratory report.