



Genomics Driven Personalised
Approach to Nutrition and Fitness



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Male

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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.

Summary of Results

Trait	Genes	Result
Vitamin And Mineral Metabolism		
Folate Requirement	MTHFR	● Typical
Vitamin A Requirement	BCM01	● 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	PPARy2	● 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



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

ABRIDGED SAMPLE

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.

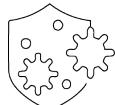
With these insights, you can proactively tailor dietary choices and supplementation strategies to your unique needs. Monitor your B12 levels; if they fall below the normal range, consult a nutritionist or healthcare provider. Likewise, identifying a genetic predisposition to iron overload helps you track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



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.

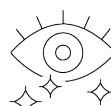
Functions



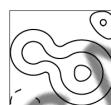
Supports our immune system



Supports brain development during pregnancy and in infants



Supports healthy vision



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



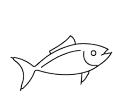
Carrot



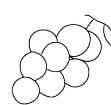
Butternut squash



Chicken liver



Tuna



Grapes

Vitamin A Requirement

YOUR GENES

Gene	rsID	Your Genotype
BCMO1	XXXXXX	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



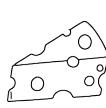
Almond milk



Cashew milk



Soymilk



Low fat cheese



Coconut milk



Paneer

Lactose Intolerance

YOUR GENES

Gene	rsID	Your Genotype
MCM6	XXXXXX	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.

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.



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	XXXXXX	TT
ADRB2	XXXXXX	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.

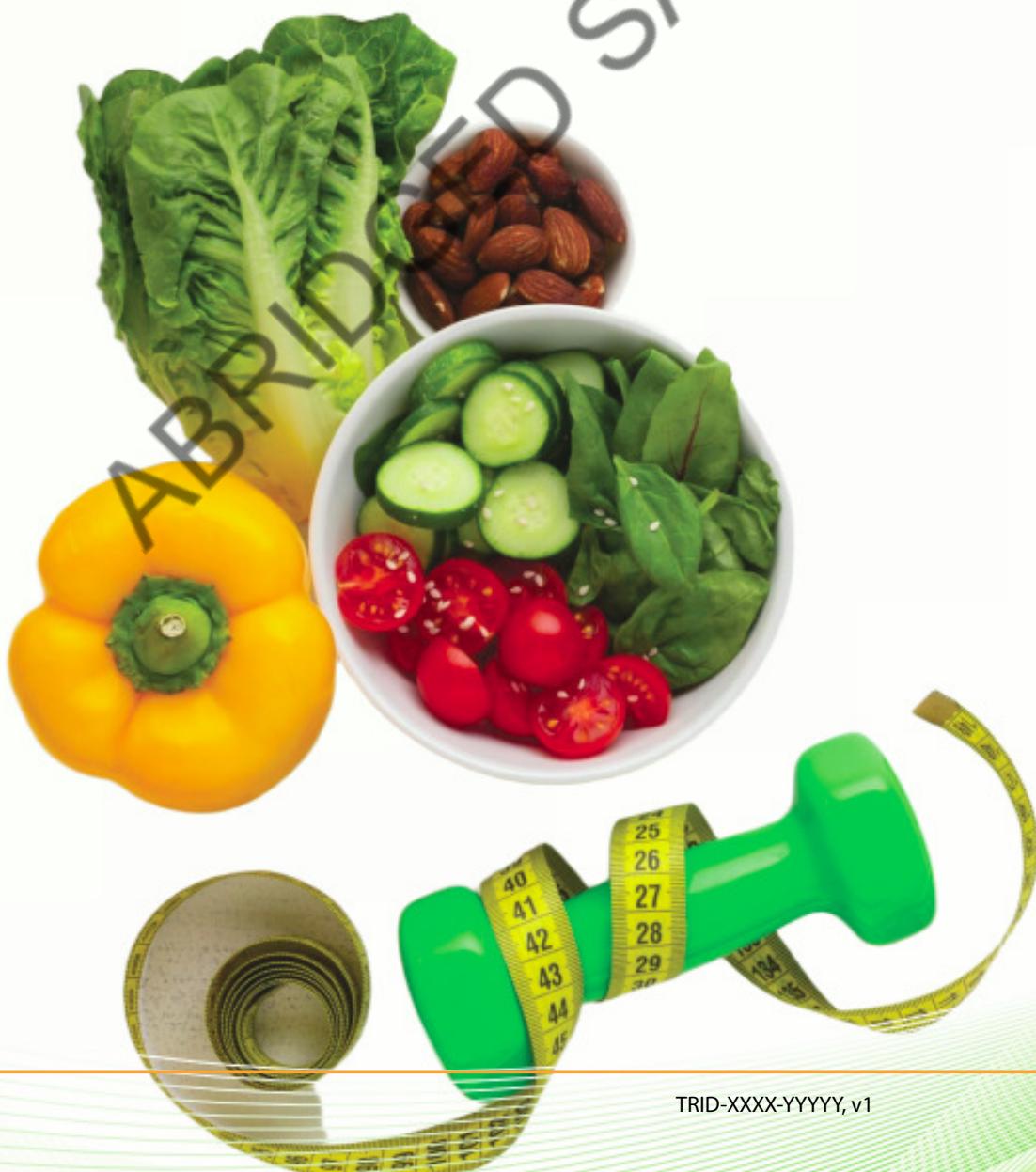
Recommendation

- 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 *ADRB2* 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.

Eating Behavior And Inherited Food/Taste Preferences

Appetite regulation, satiety signaling, and taste perception are partly hardwired, influencing everything from meal frequency to food choices. In this category of NutriDNA panel, analysis of genes involved in dopamine signaling, sweet-taste receptors, and hunger hormones reveals your predisposition toward behaviors such as snacking, craving sweets, or preferring fatty foods. Recognizing these innate tendencies early on enables you to structure your environment—and your meal planning—in ways that harness rather than fight against your genetic inclinations.

With this tailored understanding, you can adopt subtle interventions to support healthier defaults: for example, swapping in naturally sweet fruits for processed sugars if your gene insight indicates a strong sweet tooth, or timing high-protein snacks to curb urges if you carry alleles linked to rapid hunger rebound. The result is not deprivation but a personalized eating pattern that aligns with how your brain and body were wired to seek satisfaction.



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.

ABRIDGED SAMPLE

Satiety

YOUR GENES

Gene	rsID	Your Genotype
FTO	XXXXXX	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.

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.



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	XXXXXX	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.

ABRIDGED SAMPLE

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.



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.

ABRIDGED SAMPLE

Motivation To Exercise

YOUR GENES

Gene	rsID	Your Genotype
BDNF	XXXXXX	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.

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



Chia seeds



Cinnamon



Blueberries



Ginger

Interleukin 6 - Inflammation Risk

YOUR GENES

Gene	rsID	Your Genotype
<i>IL6</i>	XXXXXX	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.

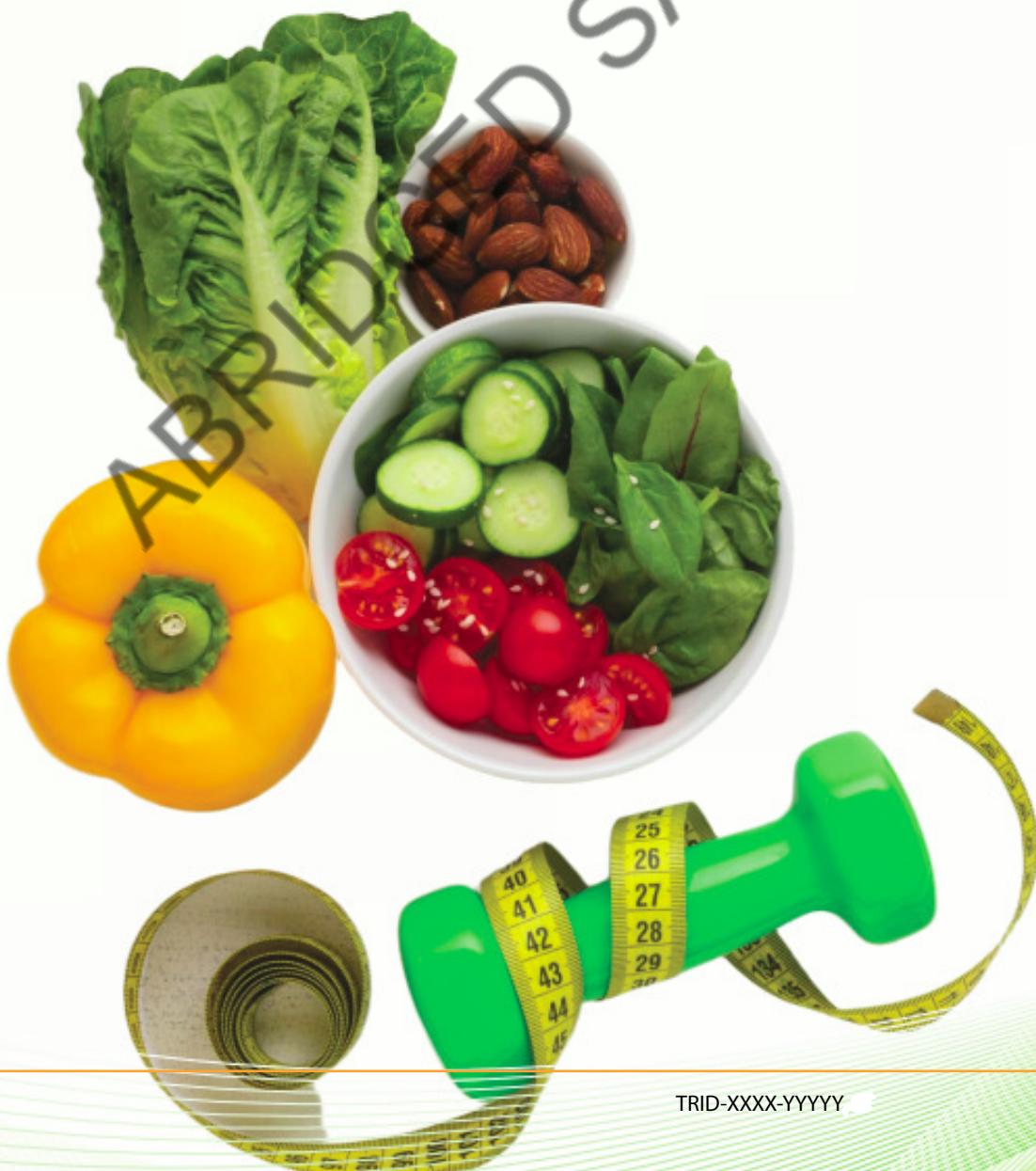
Recommendation

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

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.



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	XXXXXX	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 tolerate 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.