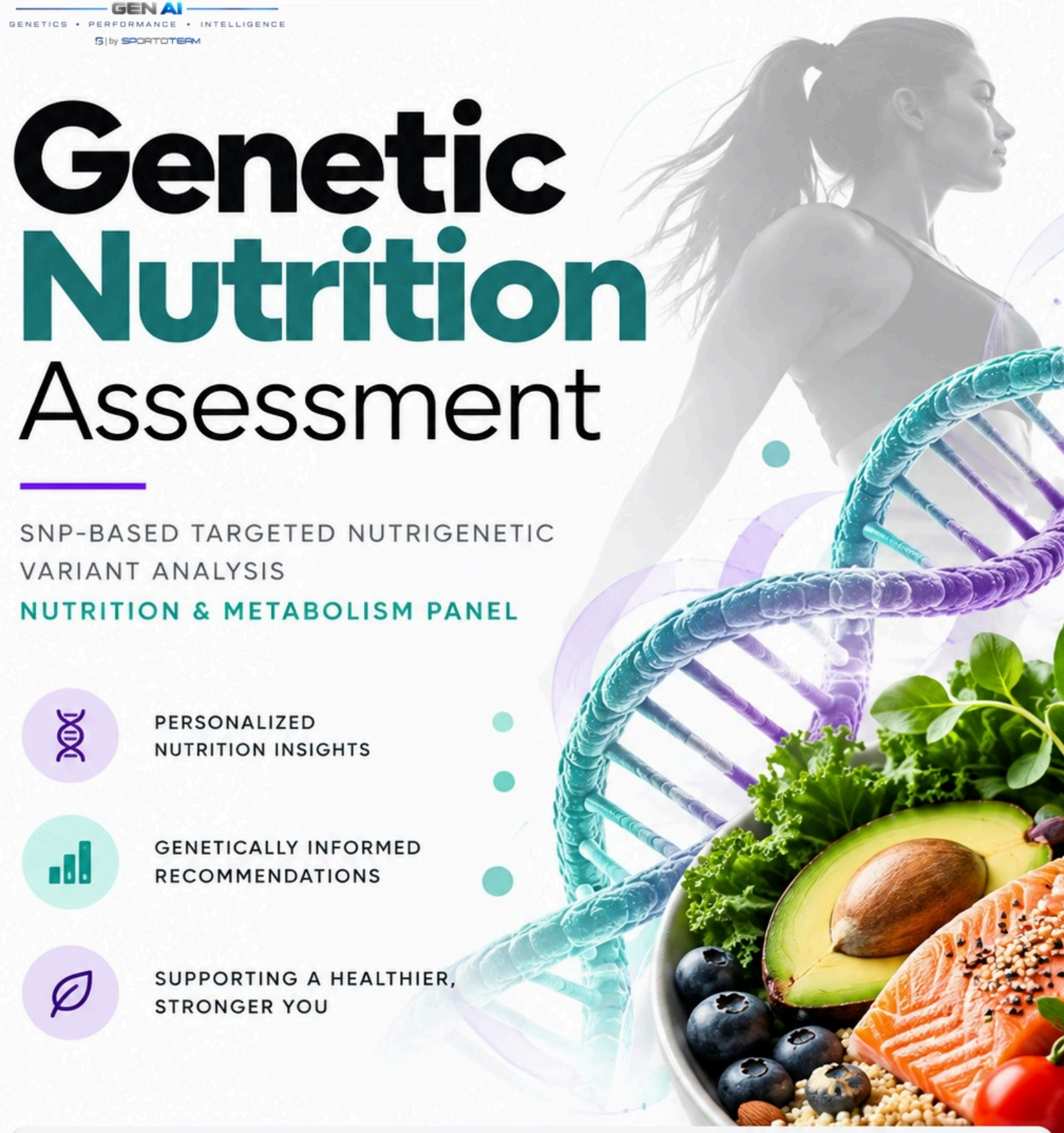
SPV GEN AI
GENETICS • PERFORMANCE • INTELLIGENCE
by SPORTOTEAM

Genetic Nutrition Assessment

SNP-BASED TARGETED NUTRIGENETIC VARIANT ANALYSIS

NUTRITION & METABOLISM PANEL



- PERSONALIZED NUTRITION INSIGHTS
- GENETICALLY INFORMED RECOMMENDATIONS
- SUPPORTING A HEALTHIER, STRONGER YOU

PATIENT PROFILE
29 years • Female
ID · NUT-2026-D1326

SAMPLE / REPORT DATE
14.08.2026
BUCCAL SWAB · SNP ARRAY

Contents

01 Client and Sample Information

02 Genetic Analysis — 12 Areas

03 Summary Assessment

04 Area-by-Area Interpretation

05 For the Dietitian and Coach

06 Recommendations

07 References and Disclaimer

METHOD

Genetic Nutrition — Method

This report is based on targeted SNP genotyping using a high-density SNP array. Selected genetic variants related to nutrition, metabolism, nutrient use, dietary response and lifestyle were analysed. Genotypes were interpreted using current scientific literature and population-based genetic evidence.

Genetic Analysis Results

Genetic Analysis Results (1)

GENE	POLYMORPHISM (RS NO.)	GENOTYPE	ASSOCIATED TRAIT	INTERPRETATION	EVIDENCE LEVEL
FTO	rs9939609 (T>A)	AT	Appetite, satiety and body weight tendency	You carry one copy of the variant most consistently linked to higher body weight; it mainly affects your appetite and how quickly you feel full, rather than how fast you burn energy. The key finding: regular physical activity reduces this effect by about a third, and it does not stop any diet from working.	STRONG
MC4R	rs17782313 (T>C)	TC	Appetite signalling and portion size	You carry one copy of a variant linked to slightly larger portions and a stronger hunger signal. In practice this shows up as portion size rather than food choice, so plate structure matters more for you than the type of diet you follow.	MEDIUM
TCF7L2	rs7903146 (C>T)	CT	Blood sugar regulation and diet quality	You carry one copy of the strongest known blood sugar variant. Your insulin response may be less tolerant than average after refined-carbohydrate meals; it is the only variant for which a Mediterranean diet was shown in a randomised trial to cancel the extra risk.	STRONG
PPARG	rs1801282 (Pro12Ala)	CG (Pro/Ala)	Insulin sensitivity and dietary fat response	You carry the protective Ala variant linked to better insulin sensitivity, but the benefit depends on the *type* of fat you eat and is strongest when unsaturated fats outweigh saturated fats. This is a true gene-diet interaction, not a fixed advantage.	STRONG

Genetic Analysis Results (2)

GENE	POLYMORPHISM (RS NO.)	GENOTYPE	ASSOCIATED TRAIT	INTERPRETATION	EVIDENCE LEVEL
GCKR	rs1260326 (Pro446Leu)	CT	Blood triglycerides after meals	You carry one copy of a variant linked to higher circulating triglycerides and a larger fat rise after a fatty meal. Carriers also show a better triglyceride response to omega-3 intake, so oily fish is more valuable for you than for most people.	STRONG
FADS1	rs174547 (T>C)	TC	Converting plant omega-3 to active forms	This gene controls how well you convert plant omega-3 (walnuts, flax, chia) into EPA and DHA, the forms your body actually uses. You are an intermediate converter: conversion works but is not efficient, so direct sources (oily fish or algal oil) do the job far more reliably.	STRONG
APOA5	rs662799 (-1131T>C)	AG	Triglyceride response to dietary fat	You carry one copy of a variant in which total fat intake tracks triglycerides and waist circumference more closely than in non-carriers. Both the quality and total amount of fat matter more for you than average.	MEDIUM
APOA2	rs5082 (-265T>C)	CC	Body weight response to saturated fat	You have the genotype in which high saturated fat intake has repeatedly been linked to higher body weight and BMI, confirmed in Mediterranean, US and Asian populations. Keeping saturated fat moderate is a more valuable action for you than for most people.	STRONG

Genetic Analysis Results (3)

GENE	POLYMORPHISM (RS NO.)	GENOTYPE	ASSOCIATED TRAIT	INTERPRETATION	EVIDENCE LEVEL
MTHFR	rs1801133 (C677T)	CT	Folate processing and homocysteine	You carry one copy of the reduced-activity variant. As long as your folate intake is adequate (leafy greens, legumes, citrus), a single copy has little practical effect. It is not a disease marker and does not require a special "methylated" supplement.	STRONG
GC	rs2282679 (A>C)	AC	Vitamin D transport in the blood	You carry one copy of a variant linked to lower measured vitamin D, because less carrier protein is available to transport it. This means your blood level tends to run lower for the same sun exposure and intake.	STRONG
CYP2R1	rs10741657 (A>G)	AG	Activating vitamin D in the liver	You carry one copy of a variant linked to lower vitamin D status. Together with your GC result, two independent markers point the same way: have your vitamin D measured rather than assuming it is normal.	MEDIUM
FUT2	rs602662 (G>A)	GA	Vitamin B12 availability	You carry one copy of a variant affecting how B12 is absorbed and transported. It does not cause deficiency on a normal mixed diet, but if you reduce or cut out animal foods, B12 is the first thing to check and supplement.	MEDIUM

Genetic Analysis Results (4)

GENE	POLYMORPHISM (RS NO.)	GENOTYPE	ASSOCIATED TRAIT	INTERPRETATION	EVIDENCE LEVEL
TMPRSS6	rs855791 (Val736Ala)	AG	Iron status and haemoglobin	You carry one copy of a variant linked to lower iron and haemoglobin levels. In a menstruating endurance runner already losing iron through sweat, foot strike and monthly bleeding, this is one of the most practically useful results in the report.	STRONG
BCO1	rs12934922 (A>T)	AT	Converting plant vitamin A to active form	You convert beta- carotene from carrots, pumpkin and sweet potato into retinol, the active vitamin A, less efficiently. The fix is simple: include some preformed vitamin A (eggs, dairy, oily fish) rather than relying only on plant sources.	MEDIUM
MCM6 / LCT	rs4988235 (-13910C>T)	CT	Lactose digestion in adulthood	You carry one lactase persistence copy, so you keep producing the lactose enzyme in adulthood. Milk and dairy should be well tolerated; occasional symptoms are more likely due to quantity or something other than lactose.	STRONG
CYP1A2	rs762551 (-163C>A)	AA	How fast caffeine is broken down	You clear caffeine quickly. Fast metabolisers are the group showing the clearest performance benefit from caffeine and in whom coffee is not linked to increased cardiovascular risk.	STRONG

Genetic Analysis Results (5)

GENE	POLYMORPHISM (RS NO.)	GENOTYPE	ASSOCIATED TRAIT	INTERPRETATION	EVIDENCE LEVEL
ADORA2A	rs5751876 (T>C)	CT	Caffeine sensitivity, alertness and sleep	This gene controls the receptor caffeine actually acts on, explaining why two people who clear caffeine at the same rate can feel it so differently. You are intermediate: you should tolerate caffeine well, but late-afternoon doses may still cost you sleep quality.	STRONG
ALDH2	rs671 (Glu504Lys)	GG	Clearing alcohol's toxic breakdown product	You carry the standard version, so you break down acetaldehyde, the compound behind flushing, nausea and headache, normally. No alcohol flush reaction is expected. This is not permission to drink more; standard health advice applies unchanged.	STRONG
ADH1B	rs1229984 (Arg48His)	CC	Alcohol breakdown rate	You carry the common version that breaks down alcohol at a standard rate. Together with your ALDH2 result, alcohol behaves ordinarily in your body, meaning the usual limits apply, not higher ones.	STRONG
TAS2R38	rs713598 (Pro49Ala)	CG	Bitter taste perception	You are an intermediate bitter taster. Crucifers (broccoli, cabbage, Brussels sprouts, rocket) taste more bitter to you than to a non-taster; this is a real physiological explanation, not pickiness. Roasting, olive oil, salt and acid reliably reduce bitterness.	MEDIUM

Genetic Analysis Results (6)

GENE	POLYMORPHISM (RS NO.)	GENOTYPE	ASSOCIATED TRAIT	INTERPRETATION	EVIDENCE LEVEL
PPARGC1A	rs8192678 (Gly482Ser)	GG (Gly/Gly)	Building muscle's energy factories	Your muscles efficiently build mitochondria, the cells' energy factories, in response to endurance training. This is the machinery that burns fat and carbohydrate during exercise, and it responds well in you. (Interpreted for European ancestry.)	STRONG
PPARA	rs4253778 (G>C)	GC	Fat vs carbohydrate as exercise fuel	You carry one copy of each allele: the G allele, linked to endurance profiles and greater reliance on fat as fuel, and the C allele, linked to power profiles and carbohydrate use. You have a truly flexible, not specialised, fuel system.	MEDIUM
MCT1 / SLC16A1	rs1049434 (Glu490Asp)	AT (heterozygous)	Using lactate as fuel	You carry one copy of the allele linked to moving lactate into muscle to burn as fuel and to a higher lactate threshold. In practice, carbohydrate is used well rather than wasted in long or hard sessions.	MEDIUM
SOD2	rs4880 (Ala16Val)	CC (Val/Val)	Handling exercise stress within muscle	You clear exercise-induced oxidative stress within muscle more slowly, so hard sessions leave a bigger mark. The practical advice is the opposite of what people expect: get antioxidants from food and avoid high-dose vitamin C and E supplements, which have blunted training adaptation.	MEDIUM

Genetic Analysis Results (7)

GENE	POLYMORPHISM (RS NO.)	GENOTYPE	ASSOCIATED TRAIT	INTERPRETATION	EVIDENCE LEVEL
IL6	rs1800795 (-174G/C)	GC	Muscle damage and recovery response	After the same hard session, your muscle damage blood marker (CK) may read much higher than a training partner's, while everything about you is completely normal. Recovery nutrition — timing protein and carbohydrate after hard sessions — is worth more for you than average.	MEDIUM

Summary Assessment

This profile *describes a metabolically competent, training-responsive body, three specific nutrient priorities — iron, vitamin D and omega-3 — and whole-food carbohydrate quality, rather than carbohydrate restriction, as a clear preference*. Four findings define this person.

1. Three separate micronutrients need to be measured, not guessed. Two independent vitamin D markers point low, an iron marker points low in someone whose sport and physiology already deplete iron, and a B12 marker indicates that animal food intake matters. Meaning: a single annual blood panel — ferritin, vitamin D, B12, full blood count — turns three genetic tendencies into three known numbers.

2. Carbohydrate quality matters more than quantity. A blood-sugar variant is present, but it is also the only variant proven in a randomized trial to be neutralized by a Mediterranean-style diet. Meaning: you don't need a low-carb diet. You need slow, whole, fiber-rich carbohydrate instead of refined — and carbohydrate around training is an asset.

3. Fat quality is a high-value lever. Three separate markers — saturated-fat sensitivity, triglyceride response and poor plant omega-3 conversion — all point to the fat side of the plate. Meaning: replacing saturated fat with olive oil, nuts and oily fish is the single highest-return change in this profile.

4. Your body registers hard training strongly, and the answer is food, not pills. Two markers point to a pronounced muscle-damage and oxidative-stress response. Meaning: prioritize recovery nutrition and sleep; especially avoid high-dose antioxidant supplements, shown to blunt the adaptation you train to gain.

Overall: a profile that rewards ordinary good nutrition done right, with three blood markers to monitor and one clear instruction — fuel the training, don't supplement around it.

Nutrition Domain Profile

★★★☆☆	**Appetite and body-weight tendency**	STRONG
★★★☆☆	**Carbohydrate and blood-sugar handling**	STRONG
★★☆☆☆	**Omega-3 status**	STRONG
★★☆☆☆	**Iron status**	STRONG
★★★☆☆	**Vitamin A from plant sources**	MEDIUM
★★★★☆	**Caffeine and alcohol handling**	STRONG
★★★★★	**Response to physical activity**	STRONG
★★★☆☆	**Fat handling and blood lipids**	STRONG
★★☆☆☆	**Vitamin D status**	STRONG
★★★☆☆	**B-vitamin status (B12, folate)**	STRONG
★★★★★	**Food tolerance (lactose, gluten)**	STRONG
★★★★☆	**Exercise fuel use and recovery needs**	MEDIUM

PRIORITY NUTRIENTS

Three nutrients stand out, and all three can be measured with a single blood draw.

Area-by-Area Interpretation

Appetite and Body-Weight Tendency

RESULT	**FTO AT · MC4R TC**
EXPLANATION	Both of your appetite-related markers point the same way, and both act on the appetite side rather than on metabolism. You probably feel full a little later and serve yourself a little more.
PRACTICAL	Work on structure, not willpower. Protein and fiber at every meal, water before eating, portions served on a plate rather than eaten from the pack, and consistent meal timing. Keep training frequency high — this is the specific, evidence-based countermeasure to this genotype. And be assured: this variant does not make weight loss harder once you start.

Carbohydrate and Blood-Sugar Handling

RESULT	**TCF7L2 CT · PPARG Pro/Ala**
EXPLANATION	You carry one copy of the strongest known blood-sugar variant, together with a protective insulin-sensitivity variant. Net effect: your system handles good carbohydrate well and refined carbohydrate less well.
PRACTICAL	Keep carbohydrate, upgrade its quality. Whole grains, legumes, whole fruit, and carbohydrate eaten with protein, fat or fiber rather than alone. Carbohydrate around training is an asset and should not be cut. Have your fasting glucose and HbA1c measured with your annual panel.

Dietary Fat and Blood Lipids

RESULT	**APOA2 CC · APOA5 AG · GCKR CT · APOE E3/E3**
EXPLANATION	Three separate markers say the same thing: the type of fat you eat has a larger-than-average effect on your body weight and blood fats. Your APOE result means your cholesterol responds predictably to these changes.
PRACTICAL	This is your highest-return change. Make olive oil your default cooking and salad oil, eat nuts and seeds regularly, keep butter, cream, fatty processed meat and pastries occasional, and eat oily fish twice a week. Have a full lipid panel checked annually.

Omega-3 Status

RESULT	**FADS1 TC · GCKR CT**
EXPLANATION	Your body can convert plant omega-3 (ALA) into the active forms EPA and DHA, but not efficiently. Flaxseed and walnuts are good for you, but they are not a reliable way to raise EPA and DHA.
PRACTICAL	Get EPA and DHA directly — two servings of oily fish a week (salmon, sardines, mackerel, herring) or an algae-based supplement if you don't eat fish. The range used in sports nutrition guidelines is about 2 g combined EPA+DHA per day; go by the label's EPA+DHA content, not capsule weight.

Vitamin D Status

RESULT	**GC AC · CYP2R1 AG**
EXPLANATION	Two independent markers, one affecting transport and one activation, point to a lower measured vitamin D level for the same sun exposure and intake.
PRACTICAL	Have serum 25(OH)D measured once, ideally in late winter when it is lowest. If low, correct it with a dose under medical supervision and re-test; if normal, reasonable sun exposure and food sources (oily fish, eggs, fortified foods) maintain it. Don't take high-dose vitamin D indefinitely without testing — more is not better.

Iron Status

RESULT	**TMPRSS6 AG · HFE negative**
EXPLANATION	You carry one copy of a variant linked to lower iron and hemoglobin, and no iron-overload variant. Your genetics push iron down, not up.
PRACTICAL	Have ferritin, hemoglobin and transferrin saturation checked annually — and straight away if you notice unusual fatigue or a drop in training tolerance. Eat iron-rich foods with a vitamin C source (lentils with peppers, spinach with lemon) and keep tea and coffee an hour away from iron-rich meals. Supplement only on the basis of a blood result, at the dose and schedule set by your physician.

B Vitamins: B12 and Folate

RESULT	**FUT2 GA · MTHFR CT**
EXPLANATION	One copy of a B12-related variant and one copy of the reduced-activity folate variant. Neither causes problems on a normal mixed diet. If your diet narrows, B12 is the first thing to monitor.
PRACTICAL	Keep folate-rich foods regular — leafy greens, legumes, citrus, fortified grains. Include B12 sources (fish, meat, eggs, dairy, or fortified foods and a supplement if you eat plant-based). Ordinary folic acid works normally with your genotype; expensive "methylated" supplements are not needed for this result. If planning a pregnancy, follow your physician's standard folic acid advice regardless of genotype.

Vitamin A from Plant Sources

RESULT	**BCO1 AT**
EXPLANATION	You convert beta-carotene from orange and dark-green vegetables into active vitamin A less efficiently than average. The vegetables are still worth eating; they just do less vitamin A work than the label suggests.
PRACTICAL	Include some preformed vitamin A in your diet — eggs, dairy, oily fish or occasional liver. Keep eating carotenoid-rich vegetables with a little fat (olive oil on roasted vegetables); this improves absorption. High-dose vitamin A supplements are not indicated, and high-dose retinol can be harmful.

Food Tolerance: Lactose and Gluten

RESULT	**LCT CT · HLA-DQ2.5 and DQ8 negative**
EXPLANATION	You keep producing the lactose-digesting enzyme into adulthood, and you lack both haplotypes required for celiac disease. Neither dairy nor gluten needs restricting for genetic reasons.
PRACTICAL	Keep dairy in your diet — it is a practical source of protein, calcium and iodine. Keep whole grains in your diet; they support your blood-sugar result. If you have bloating or gut symptoms despite these results, the cause is something else (portion size, FODMAPs, fiber changes, caffeine, training-related gut stress) and is worth investigating properly with a dietitian, not by cutting food groups.

Caffeine and Alcohol

RESULT	**CYP1A2 AA · ADORA2A CT · ALDH2 GG · ADH1B CC**
EXPLANATION	You clear caffeine quickly but have intermediate sensitivity at the receptor level. On the alcohol side, two standard variants mean alcohol behaves ordinarily in your body.
PRACTICAL	Use caffeine where it helps — 3–6 mg/kg about 60 minutes before a session — but stop about 8 hours before bed to protect sleep. For alcohol, standard national guidance applies; a normal-metabolizer genotype does not raise the sensible limit, and alcohol impairs sleep and recovery regardless.

Exercise Fuel, Recovery and Training Adaptation

RESULT	**PPARGC1A Gly/Gly · PPARA GC · MCT1 AT · SOD2 Val/Val · IL6 GC**
EXPLANATION	Your metabolism is flexible in fuel use and you build mitochondria well; this favors endurance. However, two markers point to a pronounced muscle-damage and oxidative-stress response after hard training.
PRACTICAL	Fuel the training: use carbohydrate as an asset in long or hard sessions, pay attention to protein and carbohydrate timing after hard sessions, and prioritize sleep. Key point: avoid high-dose vitamin C and E supplements, shown to blunt training adaptation; get antioxidants from food.

For the Dietitian and Coach

▸IN DAILY LIFE

- Nothing here is a diagnosis. These are tendencies, and each is smaller than the effect of what you actually eat, how much you move and how well you sleep.
- The single most effective change is on the fat side of the plate: olive oil instead of butter, nuts instead of pastries, oily fish twice a week. Three separate genes in your report point to this.
- You don't need to cut carbohydrate. You need better carbohydrate. Whole grains, legumes and fruit instead of refined and sugary foods — and carbohydrate around training helps, not harms.
- Get one blood test. Ferritin, vitamin D, B12, full blood count, lipids, glucose. Your three genetic findings become real numbers with a single draw, and numbers always beat estimates.
- You don't need to avoid dairy or gluten. Both results are clear, and cutting food groups without reason costs you nutrients and enjoyment.

> **IN SUMMARY** This profile rewards ordinary good nutrition applied consistently, with three nutrients worth measuring and one clear food-first instruction. Nothing exotic is required.

▸FOR THE DIETITIAN — ONE PAGE

PRIORITY	ACTION	RATIONALE IN PROFILE
1	Baseline bloods: ferritin, transferrin saturation, full blood count, 25(OH)D, B12, lipids, HbA1c	TMPRSS6, GC + CYP2R1, FUT2 all point to nutrients that should be measured, not assumed
2	Shift fat quality: unsaturated by default, saturated fat in moderation	APOA2 CC, APOA5 AG and GCKR CT converge on fat quality as the highest-return lever
3	Mediterranean-style carbohydrate quality; don't restrict total carbohydrate	TCF7L2 CT, with randomized trial evidence that this diet neutralizes the effect
4	Direct EPA/DHA — oily fish twice a week or algal oil	FADS1 TC reduced conversion; GCKR carriers show greater omega-3 benefit
5	Structured recovery nutrition; ~1.6 g/kg/day protein spread across meals	IL6 GC and SOD2 Val/Val point to a pronounced damage and oxidative-stress response
6	Explicitly stop high-dose vitamin C and E supplements (deprescribe)	Controlled trials show blunted training adaptation; the SOD2 result makes this more relevant
7	Maintain high training frequency as a weight-management tool	Physical activity reduces the FTO effect by about one third
8	No dairy or gluten restriction; investigate gut symptoms in their own context	LCT persistence positive, celiac haplotypes negative

Recommendations

✦ Dietary pattern

A Mediterranean-style pattern fits this profile more closely than any other, and is the only one with randomized trial evidence of neutralizing a variant carried here. Build meals around vegetables, legumes, whole grains, fish, olive oil, nuts and moderate dairy.

✦ Fat

Olive oil as the default fat. Nuts and seeds daily. Oily fish twice a week. Keep butter, cream, fatty processed meat and pastries occasional.

✦ Carbohydrate

Keep it, upgrade its quality. Whole, intact sources, paired with protein or fiber. Carbohydrate before and during long or hard training sessions.

✦ Protein

About 1.6 g/kg per day, spread over three to four meals, with 20–40 g at the post-training meal. This supports the training, not the genotype — good advice in any case.

✦ Micronutrient monitoring

Annual panel including ferritin, 25(OH)D, B12 and full blood count. Supplement only in response to a result.

✦ Supplements this profile points to

Vitamin D if the blood level is low; omega-3 (fish or algal oil) if oily fish intake is below two servings a week; iron only if ferritin shows a need. If strength or repeated-sprint performance is a goal, creatine is a well-evidenced option regardless of genotype.

✦ Supplements to avoid

High-dose vitamin C and E, especially during training blocks. High-dose vitamin A. Routine iron without a blood test. "Methylation" supplement combinations marketed on the basis of an MTHFR result.

✦ Caffeine

Where useful, 3–6 mg/kg about 60 minutes before a session; try it in training first, and stop about 8 hours before bed.

✦ Alcohol

Standard national guidance applies. A normal-metabolizer genotype does not raise the sensible limit, and alcohol impairs sleep and recovery regardless.

✦ Follow-up

Genetic findings don't change; your blood results, training load, body composition and life stage do. Review annually, or sooner if diet, training or health status changes markedly.

Gene Book

This appendix gives short, actionable notes on each key gene examined in the nutrition panel. The notes are not diagnostic and should be evaluated by a physician or dietitian.

FTO	Fat-mass and appetite-regulation gene. Some variants slightly reduce satiety and may encourage overeating; physical activity meaningfully reduces this effect.
TCF7L2	Affects insulin secretion and blood-sugar handling. Some variants raise type 2 diabetes risk; a Mediterranean-style diet largely neutralizes this.
APOA2	Governs the metabolic response to saturated fat. The CC variant increases the tendency to gain weight on a high saturated-fat intake.
FADS1 / FADS2	Controls conversion of plant omega-3 to EPA/DHA. With reduced-converter variants, direct fish or algal oil is recommended.
CYP2R1	Mediates vitamin D activation in the liver. Some variants lower circulating 25(OH)D levels.
HFE	Hereditary iron overload gene. Some variants (C282Y, H63D) can cause iron accumulation and require clinical evaluation.
FUT2	Affects intestinal B12 absorption and microbiome composition. The non-secretor variant is linked to lower B12 status.
LCT / MCM6	Determines lactase persistence in adults. Persistent variants digest lactose easily; non-persistent variants should add dairy gradually.
CYP1A2	Primary caffeine-metabolising enzyme. Fast metabolisers tolerate caffeine well; slow metabolisers should limit intake.
TAS2R38	Governs bitter taste perception. Supertasters find vegetables (especially crucifers) more bitter; preparation methods ease acceptance.
MC4R	Involved in central appetite control. Some variants affect hunger signaling and portion-size preference.
GCKR	Regulates liver glucokinase; linked to triglycerides and fasting glucose. One variant shows a strong omega-3 response.
APOA5	Regulates triglyceride metabolism. Some variants are linked to high fasting triglycerides; omega-3 provides a clear benefit.
GC	Encodes vitamin D-binding protein. Some variants are linked to lower measured 25(OH)D levels.
TMPRSS6	Regulates iron absorption via hepcidin. Some variants raise the predisposition to iron deficiency, especially in menstruating athletes.
MTHFR	Plays a role in folate metabolism. Some variants increase folate needs and may affect homocysteine levels.
BCO1 / BCO1	Controls conversion of plant beta-carotene to vitamin A. Some variants markedly reduce conversion; preformed vitamin A sources are recommended.
HLA-DQ2 / DQ8	Primary genetic determinant of coeliac disease. Its absence largely rules out coeliac disease; its presence may warrant testing or follow-up.
ADH1B / ALDH2	Plays a role in alcohol metabolism. Some variants slow alcohol clearance and produce marked effects even at low intake.
PROP1 / TAS1R2	Genes involved in sweet and umami perception. May influence sugar preference and food choices.
SOD2	Encodes a mitochondrial antioxidant enzyme. The Val/Val variant identifies the group in whom high-dose antioxidant supplements blunt training gains.

References

[1] Kilpeläinen TO, Qi L, Brage S, et al. Physical activity attenuates the influence of FTO variants on obesity risk: a meta-analysis of 218,166 adults and 19,268 children. PLoS Med. 2011;8(11):e1001116. PMID 22069379. https://pubmed.ncbi.nlm.nih.gov/22069379/

[2] Livingstone KM, Celis-Morales C, Papandonatos GD, et al. FTO genotype and weight loss: systematic review and meta-analysis of 9563 individual participant data from eight randomised controlled trials. BMJ. 2016;354:i4707. PMID 27650503. https://pubmed.ncbi.nlm.nih.gov/27650503/

[3] Rahati S, Qorbani M, Naghavi A, Pishva H. Association and interaction of the MC4R rs17782313 polymorphism with plasma ghrelin, GLP-1, cortisol, food intake and eating behaviors. BMC Endocr Disord. 2022;22:234. PMID 36123585. https://pubmed.ncbi.nlm.nih.gov/36123585/

[4] Alizadeh S, et al. Interaction of MC4R rs17782313 variants and dietary carbohydrate quantity and quality on basal metabolic rate and general and central obesity. BMC Endocr Disord. 2022. PMID 35538513. https://pubmed.ncbi.nlm.nih.gov/35538513/

[5] Guan Y, et al. Correlation of the TCF7L2 (rs7903146) polymorphism with an enhanced risk of type 2 diabetes mellitus: a meta-analysis. Genet Mol Res. 2016. PMID 27706609. https://pubmed.ncbi.nlm.nih.gov/27706609/

[6] Corella D, Carrasco P, Sorlí JV, et al. Mediterranean diet reduces the adverse effect of the TCF7L2-rs7903146 polymorphism on cardiovascular risk factors and stroke incidence: a randomized controlled trial. Diabetes Care. 2013;36(11):3803-3811. PMID 23942586. https://pubmed.ncbi.nlm.nih.gov/23942586/

[7] Lamri A, Abi Khalil C, Jaziri R, et al. Dietary fat intake and polymorphisms at the PPARG locus modulate BMI and type 2 diabetes risk in the D.E.S.I.R. prospective study. Int J Obes (Lond). 2012;36(2):218-224. PMID 21540831. https://pubmed.ncbi.nlm.nih.gov/21540831/

[8] Ylönen SK, Salminen I, Lyssenko V, et al. The Pro12Ala polymorphism of the PPAR-gamma2 gene affects associations of fish intake and marine n-3 fatty acids with glucose metabolism. Eur J Clin Nutr. 2008;62(12):1432-1439. PMID 17700648. https://pubmed.ncbi.nlm.nih.gov/17700648/

[9] Orho-Melander M, Melander O, Guiducci C, et al. Common missense variant in the glucokinase regulatory protein gene is associated with increased plasma triglyceride and C-reactive protein but lower fasting glucose concentrations. Diabetes. 2008;57(11):3112-3121. PMID 18678614. https://pubmed.ncbi.nlm.nih.gov/18678614/

[10] Shen H, Pollin TI, Damcott CM, et al. Glucokinase regulatory protein gene polymorphism affects postprandial lipemic response in a dietary intervention study. Hum Genet. 2009;126(4):567-574. PMID 19526250. https://pubmed.ncbi.nlm.nih.gov/19526250/

[11] Perez-Martinez P, Delgado-Lista J, Garcia-Rios A, et al. Glucokinase regulatory protein genetic variant interacts with omega-3 PUFA to influence insulin resistance and inflammation in metabolic syndrome. PLoS One. 2011;6(6):e20555. PMID 21674002. https://pubmed.ncbi.nlm.nih.gov/21674002/

[12] Tanaka T, Shen J, Abecasis GR, et al. Genome-wide association study of plasma polyunsaturated fatty acids in the InCHIANTI Study. PLoS Genet. 2009;5(1):e1000338. PMID 19148276. https://pubmed.ncbi.nlm.nih.gov/19148276/

[13] Lattka E, Illig T, Koletzko B, Heinrich J. Genetic variants of the FADS1 FADS2 gene cluster as related to essential fatty acid metabolism. Curr Opin Lipidol. 2010;21(1):64-69. PMID 19809313. https://pubmed.ncbi.nlm.nih.gov/19809313/

[14] Cormier H, Rudkowska I, Paradis AM, et al. Association between polymorphisms in the fatty acid desaturase gene cluster and the plasma triacylglycerol response to an n-3 PUFA supplementation. Nutrients. 2012;4(8):1026-1041. PMID 23016130. https://pubmed.ncbi.nlm.nih.gov/23016130/

[15] Lai CQ, Corella D, Demissie S, et al. Dietary intake of n-6 fatty acids modulates effect of apolipoprotein A5 gene on plasma fasting triglycerides, remnant lipoprotein concentrations, and lipoprotein particle size. Circulation. 2006;113(17):2062-2070. PMID 16636175. https://pubmed.ncbi.nlm.nih.gov/16636175/

[16] Sánchez-Moreno C, Ordovás JM, Smith CE, et al. APOA5 gene variation interacts with dietary fat intake to modulate obesity and circulating triglycerides in a Mediterranean population. J Nutr. 2011;141(3):380-385. PMID 21209257. https://pubmed.ncbi.nlm.nih.gov/21209257/

[17] Mattei J, Demissie S, Tucker KL, Ordovas JM. Apolipoprotein A5 polymorphisms interact with total dietary fat intake in association with markers of metabolic syndrome in Puerto Rican older adults. J Nutr. 2009;139(12):2301-2308. PMID 19828688. https://pubmed.ncbi.nlm.nih.gov/19828688/

[18] Corella D, Tai ES, Sorlí JV, et al. Association between the APOA2 promoter polymorphism and body weight in Mediterranean and Asian populations: replication of a gene-saturated fat interaction. Int J Obes (Lond). 2011;35(5):666-675. PMID 20975728. https://pubmed.ncbi.nlm.nih.gov/20975728/

[19] Lai CQ, Smith CE, Parnell LD, et al. Epigenomics and metabolomics reveal the mechanism of the APOA2-saturated fat intake interaction affecting obesity. Am J Clin Nutr. 2018;108(1):188-200. PMID 29901700. https://pubmed.ncbi.nlm.nih.gov/29901700/

[20] Colson NJ, Naug HL, Nikbakht E, et al. The impact of MTHFR 677 C/T genotypes on folate status markers: a meta-analysis of folic acid intervention studies. Eur J Nutr. 2017;56(1):247-260. PMID 26497154. https://pubmed.ncbi.nlm.nih.gov/26497154/

References

e-wide association study of eine blood concentrations. Am J Hum Genet. 2009;84(4):477-482. PMID 19303062. https://pubmed.ncbi.nlm.nih.gov/19303062/

[22] Hickey SE, Curry CJ, Toriello HV. ACMG Practice Guideline: lack of evidence for MTHFR polymorphism testing. Genet Med. 2013;15(2):153-156. PMID 23288205. https://pubmed.ncbi.nlm.nih.gov/23288205/

[23] Wang TJ, Zhang F, Richards JB, et al. Common genetic determinants of vitamin D insufficiency: a genome-wide association study. Lancet. 2010;376(9736):180-188. PMID 20541252. https://pubmed.ncbi.nlm.nih.gov/20541252/

[24] Ahn J, Yu K, Stolzenberg-Solomon R, et al. Genome-wide association study of circulating vitamin D levels. Hum Mol Genet. 2010;19(13):2739-2745. PMID 20418485. https://pubmed.ncbi.nlm.nih.gov/20418485/

[25] Hazra A, Kraft P, Lazarus R, et al. Genome-wide significant predictors of metabolites in the one-carbon metabolism pathway. Hum Mol Genet. 2009;18(23):4677-4687. PMID 19744961. https://pubmed.ncbi.nlm.nih.gov/19744961/

[26] Chambers JC, Zhang W, Li Y, et al. Genome-wide association study identifies variants in TMPRSS6 associated with hemoglobin levels. Nat Genet. 2009;41(11):1170-1172. PMID 19820698. https://pubmed.ncbi.nlm.nih.gov/19820698/

[27] Bösch ES, et al. Genetic variants affecting iron metabolism in healthy adults: a systematic review to support personalized nutrition strategies. Nutrients. 2024;16:4159. PMID 39599580. https://pubmed.ncbi.nlm.nih.gov/39599580/

[28] Lietz G, Oxley A, Leung W, Hesketh J. Single nucleotide polymorphisms upstream from the β-carotene 15,15'-monooxygenase gene influence provitamin A conversion efficiency in female volunteers. J Nutr. 2012;142(1):161S-165S. PMID 22113863. https://pubmed.ncbi.nlm.nih.gov/22113863/

[29] Borel P. Genetic variations involved in interindividual variability in carotenoid status. Mol Nutr Food Res. 2012;56(2):228-240. PMID 21957063. https://pubmed.ncbi.nlm.nih.gov/21957063/

[30] Enattah NS, Sahi T, Savilahti E, et al. Identification of a variant associated with adult-type hypolactasia. Nat Genet. 2002;30(2):233-237. PMID 11788828. https://pubmed.ncbi.nlm.nih.gov/11788828/

[31] Guest N, Corey P, Vescovi J, El-Sohemy A. Caffeine, CYP1A2 genotype, and endurance performance in athletes. Med Sci Sports Exerc. 2018;50(8):1570-1578. PMID 29509641. https://pubmed.ncbi.nlm.nih.gov/29509641/

[32] Barreto G, Loureiro LMR, Reis CEG, Saunders B. Caffeine, CYP1A2 genotype, and exercise performance: a systematic review and meta-analysis. Med Sci Sports Exerc. 2024;56(2):328-339. PMID 37844569. https://pubmed.ncbi.nlm.nih.gov/37844569/

[33] Wang J, et al. Does the ergogenic effect of caffeine supplementation depend on CYP1A2 genotypes? A systematic review with meta-analysis. J Sport Health Sci. 2024. PMID 38158179. https://pubmed.ncbi.nlm.nih.gov/38158179/

[34] Guest NS, VanDusseldorp TA, Nelson MT, et al. International Society of Sports Nutrition position stand: caffeine and exercise performance. J Int Soc Sports Nutr. 2021;18(1):1. PMID 33388079. https://pubmed.ncbi.nlm.nih.gov/33388079/

[35] Cornelis MC, El-Sohemy A, Kabagambe EK, Campos H. Coffee, CYP1A2 genotype, and risk of myocardial infarction. JAMA. 2006;295(10):1135-1141. PMID 16522833. https://pubmed.ncbi.nlm.nih.gov/16522833/

[36] Rétey JV, Adam M, Khatami R, et al. A genetic variation in the adenosine A2A receptor gene (ADORA2A) contributes to individual sensitivity to caffeine effects on sleep. Clin Pharmacol Ther. 2007;81(5):692-698. PMID 17329997. https://pubmed.ncbi.nlm.nih.gov/17329997/

[37] Landolt HP. "No thanks, coffee keeps me awake": individual caffeine sensitivity depends on ADORA2A genotype. Sleep. 2012;35(7):899-900. PMID 22754033. https://pubmed.ncbi.nlm.nih.gov/22754033/

[38] Erblang M, Drogou C, Gomez-Merino D, et al. The impact of genetic variations in ADORA2A in the association between caffeine consumption and sleep. Genes (Basel). 2019;10(12):1021. PMID 31817803. https://pubmed.ncbi.nlm.nih.gov/31817803/

[39] Cho Y, et al. Genetic influences on alcohol flushing in East Asian populations. BMC Genomics. 2023;24:638. PMID 37875790. https://pubmed.ncbi.nlm.nih.gov/37875790/

[40] Luczak SE, Glatt SJ, Wall TL. Meta-analyses of ALDH2 and ADH1B with alcohol dependence in Asians. Psychol Bull. 2006;132(4):607-621. PMID 16822169. https://pubmed.ncbi.nlm.nih.gov/16822169/

References

egetable intake in college-aged s and TAS2R38 genotype. Chemosens Percept. 2010;3(3-4):137-148. PMID 21157576. https://pubmed.ncbi.nlm.nih.gov/21157576/

[42] Keller KL, Adise S. Variation in the ability to taste bitter thiourea compounds: implications for food acceptance, dietary intake, and obesity risk in children. Annu Rev Nutr. 2016;36:157-182. PMID 27070900. https://pubmed.ncbi.nlm.nih.gov/27070900/

[43] Mikołajczyk-Stecyna J, Malinowska AM, Chmurzynska A. TAS2R38 and CA6 genetic polymorphisms, frequency of bitter food intake, and blood biomarkers among elderly woman. Appetite. 2017;116:57-64. PMID 28455260. https://pubmed.ncbi.nlm.nih.gov/28455260/

[44] Chen Y, et al. Meta-analyses of the association between the PPARGC1A Gly482Ser polymorphism and athletic performance. Biol Sport. 2019;36(4):301-309. PMID 31938000. https://pubmed.ncbi.nlm.nih.gov/31938000/

[45] Vimaleswaran KS, Luan J, Andersen G, et al. The Gly482Ser genotype at the PPARGC1A gene and elevated blood pressure: a meta-analysis involving 13,949 individuals. J Appl Physiol. 2008;105(4):1352-1358. PMID 18467552. https://pubmed.ncbi.nlm.nih.gov/18467552/

[46] Ahmetov II, Williams AG, Popov DV, et al. The combined impact of metabolic gene polymorphisms on elite endurance athlete status and related phenotypes. Hum Genet. 2009;126(6):751-761. PMID 19653005. https://pubmed.ncbi.nlm.nih.gov/19653005/

[47] Petr M, Stastny P, Zajac A, et al. Association of elite sports status with gene variants of peroxisome proliferator activated receptors and their transcriptional coactivator. Int J Mol Sci. 2019;21(1):162. PMID 31881714. https://pubmed.ncbi.nlm.nih.gov/31881714/

[48] Guilherme JPLF, et al. The MCT1 gene Glu490Asp polymorphism (rs1049434) is associated with endurance athlete status and lower blood lactate accumulation. Biol Sport. 2021;38(4):673-681. PMID 34475628. https://pubmed.ncbi.nlm.nih.gov/34475628/

[49] Massidda M, et al. Influence of the MCT1-T1470A polymorphism (rs1049434) on repeated sprint ability and blood lactate accumulation in elite football players. Eur J Appl Physiol. 2021;121(11):3037-3045. PMID 34480633. https://pubmed.ncbi.nlm.nih.gov/34480633/

[50] Seki S, et al. Association among MCT1 rs1049434 polymorphism, athlete status, and physiological parameters in Japanese long-distance runners. Genes (Basel). 2024;15:1627. PMID 39766893. https://pubmed.ncbi.nlm.nih.gov/39766893/

[51] Ahmetov II, Naumov VA, Donnikov AE, et al. SOD2 gene polymorphism and muscle damage markers in elite athletes. Free Radic Res. 2014;48(8):948-955. PMID 24865797. https://pubmed.ncbi.nlm.nih.gov/24865797/

[52] Hernando B, Gil-Barrachina M, Tomás-Bort E, et al. The effect of long-term ultra-endurance exercise and SOD2 genotype on telomere shortening with age. J Appl Physiol. 2020;129(2):208-213. PMID 32881625. https://pubmed.ncbi.nlm.nih.gov/32881625/

[53] Yamin C, Duarte JAR, Oliveira JMF, et al. IL6 (-174) and TNFA (-308) promoter polymorphisms are associated with systemic creatine kinase response to eccentric exercise. Eur J Appl Physiol. 2008;104(4):579-586. PMID 18758806. https://pubmed.ncbi.nlm.nih.gov/18758806/

[54] Baumert P, Lake MJ, Stewart CE, et al. Genetic variation and exercise-induced muscle damage: implications for athletic performance, injury and ageing. Eur J Appl Physiol. 2016;116(9):1595-1625. PMID 27294501. https://pubmed.ncbi.nlm.nih.gov/27294501/

[55] Girelli D, Busti F. Diagnosis and management of hereditary hemochromatosis: lifestyle modification, phlebotomy, and blood donation. Hematology Am Soc Hematol Educ Program. 2024. PMID 39644049. https://pubmed.ncbi.nlm.nih.gov/39644049/

[56] Grosse SD, Gurrin LC, Bertalli NA, Allen KJ. Clinical penetrance in hereditary hemochromatosis: estimates of the cumulative incidence of severe liver disease among HFE C282Y homozygotes. Genet Med. 2018;20(4):383-389. PMID 28771247. https://pubmed.ncbi.nlm.nih.gov/28771247/

[57] Porto G, Brissot P, Swinkels DW, et al. EMQN best practice guidelines for the molecular genetic diagnosis of hereditary hemochromatosis (HH). Eur J Hum Genet. 2016;24(4):479-495. PMID 26153218. https://pubmed.ncbi.nlm.nih.gov/26153218/

[58] Griffin BA, Walker CG, Jebb SA, et al. APOE4 genotype exerts greater benefit in lowering plasma cholesterol and apolipoprotein B than wild type (E3/E3), after replacement of dietary saturated fats with low glycaemic index carbohydrates. Nutrients. 2018;10(10):1524. PMID 30336580. https://pubmed.ncbi.nlm.nih.gov/30336580/

[59] Weggemans RM, Zock PL, Ordovas JM, et al. Apoprotein E genotype and the response of serum cholesterol to dietary fat, cholesterol and cafestol. Atherosclerosis. 2001;154(3):547-555. PMID 11257255. https://pubmed.ncbi.nlm.nih.gov/11257255/

[60] Brown NK, Guandalini S, Semrad C, Kupfer SS. A clinician's guide to celiac disease HLA genetics. Am J Gastroenterol. 2019;114(10):1587-1592. PMID 31274511. https://pubmed.ncbi.nlm.nih.gov/31274511/

References

utility of celiac disease-):2199-2206. PMID 24705698. https://pubmed.ncbi.nlm.nih.gov/24705698/

[62] Jäger R, Kerksick CM, Campbell BI, et al. International Society of Sports Nutrition position stand: protein and exercise. J Int Soc Sports Nutr. 2017;14:20. PMID 28642676. https://pubmed.ncbi.nlm.nih.gov/28642676/

[63] Morton RW, Murphy KT, McKellar SR, et al. A systematic review, meta-analysis and meta-regression of the effect of protein supplementation on resistance training-induced gains in muscle mass and strength. Br J Sports Med. 2018;52(6):376-384. PMID 28698222. https://pubmed.ncbi.nlm.nih.gov/28698222/

[64] Bjørnsen T, Salvesen S, Berntsen S, et al. Vitamin C and E supplementation blunts increases in total lean body mass in elderly men after strength training. Scand J Med Sci Sports. 2016;26(7):755-763. PMID 26129928. https://pubmed.ncbi.nlm.nih.gov/26129928/

[65] Martínez-Ferrán M, Cuadrado-Peñafiel V, Sánchez-Andreo JM, et al. Antioxidant vitamin supplementation on muscle adaptations to resistance training: a double-blind, randomized controlled trial. Nutrition. 2023;105:111848. PMID 36283241. https://pubmed.ncbi.nlm.nih.gov/36283241/

[66] Peeling P, Castell LM, Derave W, et al. Considerations for the consumption of vitamin and mineral supplements in athlete populations. Sports Med. 2023;53(Suppl 1):15-24. PMID 37358750. https://pubmed.ncbi.nlm.nih.gov/37358750/

[67] Zhang L, Quan M, Cao ZB. Effect of vitamin D supplementation on upper and lower limb muscle strength and muscle power in athletes: a meta-analysis. PLoS One. 2019;14(4):e0215826. PMID 31039170. https://pubmed.ncbi.nlm.nih.gov/31039170/

[68] Sist M, Zou L, Galloway SDR, et al. Effects of vitamin D supplementation on maximal strength and power in athletes: a systematic review and meta-analysis of randomised controlled trials. Front Nutr. 2023;10:1163313. PMID 37841405. https://pubmed.ncbi.nlm.nih.gov/37841405/

[69] Pengelly M, Pumpa K, Pyne DB, Etxebarria N. Iron deficiency, supplementation, and sports performance in female athletes: a systematic review. J Sport Health Sci. 2025. PMID 39536912. https://pubmed.ncbi.nlm.nih.gov/39536912/

[70] Sim M, Garvican-Lewis LA, Cox GR, et al. Iron considerations for the athlete: a narrative review. Eur J Appl Physiol. 2019;119(7):1463-1478. PMID 31055680. https://pubmed.ncbi.nlm.nih.gov/31055680/

[71] Sims ST, Kerksick CM, Smith-Ryan AE, et al. International Society of Sports Nutrition position stand: nutritional concerns of the female athlete. J Int Soc Sports Nutr. 2023;20(1):2204066. PMID 37221858. https://pubmed.ncbi.nlm.nih.gov/37221858/

[72] Jäger R, Heileson JL, Abou Sawan S, et al. International Society of Sports Nutrition position stand: long-chain omega-3 polyunsaturated fatty acids. J Int Soc Sports Nutr. 2025;22(1):2441775. PMID 39810703. https://pubmed.ncbi.nlm.nih.gov/39810703/

[73] Kreider RB, Kalman DS, Antonio J, et al. International Society of Sports Nutrition position stand: safety and efficacy of creatine supplementation in exercise, sport, and medicine. J Int Soc Sports Nutr. 2017;14:18. PMID 28615996. https://pubmed.ncbi.nlm.nih.gov/28615996/

[74] Celis-Morales C, Livingstone KM, Marsaux CFM, et al. Effect of personalized nutrition on health-related behaviour change: evidence from the Food4Me European randomized controlled trial. Int J Epidemiol. 2017;46(2):578-588. PMID 27524815. https://pubmed.ncbi.nlm.nih.gov/27524815/

[75] Filippini T, Malavolti M, Whelton PK, et al. Blood pressure effects of sodium reduction: dose-response meta-analysis of experimental studies. Circulation. 2021;143(16):1542-1567. PMID 33586450. https://pubmed.ncbi.nlm.nih.gov/33586450/

[76] Agrawal P, et al. Genetics, nutrition, and health: a new frontier in disease prevention. J Am Nutr Assoc. 2024;43(4):326-338. PMID 38015713. https://pubmed.ncbi.nlm.nih.gov/38015713/

[77] Semenova EA, Hall ECR, Ahmetov II. Genes and athletic performance: the 2023 update. Genes. 2023;14(6):1235. PMID 37372415. https://pubmed.ncbi.nlm.nih.gov/37372415/

[78] Webborn N, Williams A, McNamee M, et al. Direct-to-consumer genetic testing for predicting sports performance and talent identification: consensus statement. Br J Sports Med. 2015;49(23):1486-1491. PMID 26582191. https://pubmed.ncbi.nlm.nih.gov/26582191/

Where a PMID could not be verified at the time of writing, a PubMed search link is provided. Laboratory quality procedure requires every reference to be verified against PubMed before a report is released.

LEGAL NOTICE

The results in this report describe **genetic predispositions and statistical associations, not deterministic predictions.** Nutritional status and metabolic health are the product of genetics acting together with total diet, physical activity, sleep, body composition, medication, the gut microbiome, life stage, environment and chance.

This test does not diagnose any disease and does not predict future health outcomes. It must not be used to diagnose or exclude diabetes, cardiovascular disease, coeliac disease, anaemia, food allergy or intolerance, or any other condition, nor as a substitute for medical assessment, blood testing or professional dietetic advice.

Where this report suggests a practical action, it is because that action is beneficial and safe regardless of genotype. **Measured blood results and an accurate record of what you actually eat always outrank genetic data.** Current evidence shows that adding genotype information to good personalised dietary advice produces limited additional benefit; this report is therefore a tool for prioritising sound nutrition, not for replacing it.

IL6	Involved in post-exercise inflammation and recovery signalling. Some variants show a stronger repair response; protein timing matters.
PPARGC1A	Master regulator of mitochondrial biogenesis. Involved in exercise nutrition and endurance adaptations.
ACE	Affects vascular tone and blood pressure. Some variants are linked to salt sensitivity and haemodynamic exercise response.