

Personalized Health Protocol

Genetically-Informed Supplements, Diet & Training

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SPECIMEN INFORMATION

Report ID:	HGX-6CBDEB-3DB	Analysis Date:	2026-03-29
Sample Type:	Saliva / Buccal Swab (Microarray)	Platform:	Illumina GSA / OmniExpress
Methodology:	Imputation via Beagle 5.5 + 1000 Genomes Phase 3		

This report is provided for informational and research purposes only. It does not constitute a medical diagnosis, nor should it be used as a substitute for professional medical advice. Genetic risk scores reflect statistical associations observed in population studies and do not determine individual clinical outcomes. Consult a qualified healthcare professional before making any clinical decisions based on these results.

CLINICAL RECOMMENDATIONS

1. Urgent Renal/Electrolyte Evaluation — Pathogenic Gitelman Syndrome Variant

IMMEDIATE PRIORITY

rs199974259 in SLC12A3 is a pathogenic variant (2-star ClinVar, confidence=high) for Gitelman syndrome, a condition causing chronic renal hypokalemia and hypomagnesemia. Both deficiencies are independently arrhythmogenic — critically dangerous with this individual's 79.4th percentile AF PRS and converging cardiovascular risk. An immediate basic metabolic panel including serum magnesium, potassium, sodium, creatinine, and eGFR should be obtained, followed by a 24-hour urine electrolyte collection and nephrology or endocrinology referral to quantify deficiency severity and establish a personalized electrolyte supplementation protocol — supplementation doses in this plan (magnesium and potassium) should be titrated to these lab results rather than used as fixed doses.

2. Comprehensive Cardiometabolic Baseline Laboratory Panel

IMMEDIATE PRIORITY

The convergence of 99.1th percentile BMI PRS, 85.9th T2D PRS, 91.4th coronary calcification PRS, and 90.7th early-onset HTN PRS creates an extreme cardiometabolic risk profile that requires immediate quantification to determine whether pharmacological intervention is already indicated. An urgent panel should incl: fasting glucose, HbA1c, fasting insulin with HOMA-IR calculation, full lipid panel with LDL-particle count, hsCRP, uric acid, liver function tests (NAFLD risk), and urinary albumin-to-creatinine ratio (ACR) — this baseline determines whether metformin, statin, or antihypertensive therapy should be initiated concurrent with the lifestyle protocol.

3. Autoimmune Screening Panel with Rheumatology Referral if Positive

SHORT-TERM PRIORITY

The convergence of 94th percentile SLE PRS, 90th percentile RA PRS, and IRF5 rs10954213 (CADD 17.36) creates a genuine concern for autoimmune disease activation — and the most important clinical reason to act proactively is that active autoimmune disease raises acute MI and stroke risk 2-5 fold, layering catastrophically on this individual's already elevated cardiovascular convergence. A baseline autoimmune panel (ANA with reflex anti-dsDNA, anti-Smith, anti-SSA/SSB for SLE; RF and anti-CCP for RA; ESR, hsCRP, CBC with differential) should be obtained; any positive result warrants expedited rheumatology referral before organ damage occurs.

4. Coronary Artery Calcium (CAC) Score Imaging for Cardiovascular Risk Stratification

SHORT-TERM PRIORITY

With 91.4th percentile coronary calcification PRS, LRP8 rs5174 (CADD 34, REVEL 0.632 — a structurally impactful MI susceptibility variant), and the full cardiometabolic convergence signal, early CAC imaging provides a direct, personalized measure of subclinical atherosclerosis to guide statin therapy decisions. A non-contrast cardiac CT for CAC score is a low-radiation, non-invasive, one-time test — a CAC score above zero in this clinical context would strongly support initiating preventive statin therapy; this should be coordinated with a cardiologist who is also briefed on the pharmacogenomic finding (ABCG2 reduced clearance requiring lower statin dosing with CoQ10 co-supplementation).

5. Neurological Assessment with EEG Baseline — Seizure Risk from SCN1A + CNTNAP2 Convergence

SHORT-TERM PRIORITY

SCN1A rs3812718 carries ClinGen Definitive classification for Generalized Epilepsy with Febrile Seizures Plus (GEFS+) with 3-star expert panel review — this is among the highest-confidence seizure gene associations in clinical genomics. CNTNAP2 carrier status (independently linked to developmental epileptic encephalopathy) further lowers the seizure threshold. A baseline EEG establishes normal brain electrical activity before any potential seizure event, enables proactive seizure risk counseling (avoiding triggers: sleep deprivation, extreme heat, fever without management), and guides pharmacogenomically informed anti-epileptic medication choice if seizures occur — carbamazepine and phenytoin response is directly modulated by SCN1A genotype, and febrile seizure protocols should be discussed with the family.

6. Blood Pressure Monitoring Program — Thiazide Diuretics Contraindicated

ONGOING PRIORITY

90.7th percentile early-onset HTN PRS predicts likely hypertension in the 4th-5th decade; preemptive monitoring beginning now allows early detection before target organ damage. Home blood pressure should be logged twice daily for 7 days to establish a reliable baseline, then weekly ongoing; consistent readings above 130/80 mmHg warrant antihypertensive medication discussion. Critical safety note: thiazide diuretics (hydrochlorothiazide, chlorthalidone) are ABSOLUTELY CONTRAINDICATED in this individual — they block the SLC12A3 cotransporter that is already pathogenically impaired by the Gitelman variant, and would dramatically worsen hypokalemia and hypomagnesemia, precipitating cardiac arrhythmias.

7. Sleep Study (Polysomnography) and Chronotype Optimization Consultation

SHORT-TERM PRIORITY

The ADHD-sleep-metabolic reinforcing cycle (ADHD 99.3%, chronotype 94.1%, obesity risk 99.1%) creates conditions where obstructive sleep apnea is highly probable — obesity dramatically increases OSA risk, and untreated OSA worsens insulin resistance, ADHD severity, blood pressure, and arrhythmia risk (dangerous with AF PRS 79.4th percentile and Gitelman arrhythmia vulnerability). A formal polysomnography study rules out OSA; a sleep medicine consultation can design chronotype accommodation strategies that respect the genetic evening preference rather than fighting it, which is both scientifically supported and critical for long-term adherence.

8. Pharmacogenomic Medication Safety Briefing — Triple Slow Metabolizer Alert

ONGOING PRIORITY

VKORC1 rs9923231 CT (intermediate warfarin sensitivity) + CYP2C9 *1/*2 (intermediate metabolizer, warfarin and NSAID accumulation) + ABCG2 rs2231142 G/T (reduced statin clearance) create a compounded pharmacogenomic profile where multiple common drug classes will accumulate to higher-than-expected levels. Before any new prescriptions are initiated, a clinical pharmacist should conduct a formal medication reconciliation using PharmGKB/CPIC guidelines: warfarin if needed requires 30-40% dose reduction with intensive INR monitoring; NSAIDs should be avoided entirely in favor of acetaminophen; statins should start at low doses (pravastatin 10-20 mg) with CoQ10 co-supplementation; any prescribing physician should be informed of this pharmacogenomic profile.

SUPPLEMENT PROTOCOL

Magnesium Glycinate

Dose: 400-600 mg elemental magnesium daily (split doses) | 200-300 mg with dinner; 200-300 mg 30 minutes before bed
SLC12A3 (rs199974259) is a pathogenic Gitelman syndrome variant that impairs renal magnesium reabsorption, causing chronic hypomagnesemia. Low magnesium directly worsens cardiac arrhythmia risk (criti

Potassium Citrate

Dose: 1500-3000 mg potassium daily (dietary + supplemental; titrate to labs) | With meals in divided doses to prevent GI upset; do not exceed without blood lev
SLC12A3 Gitelman variant also causes renal potassium wasting and hypokalemia. Hypokalemia is an independent trigger for cardiac arrhythmias — highly dangerous in the context of 79.4th percentile AF PR

Berberine

Dose: 500 mg three times daily with meals | 500 mg 5 minutes before breakfast, lunch, and dinner
TCF7L2 rs7903146 (strongest T2D susceptibility locus globally, GWAS p=1e-1315) combined with FTO rs1421085 (99.1th percentile BMI PRS) and 85.9th percentile T2D PRS constitute extreme insulin resistan

Omega-3 Fatty Acids (EPA/DHA, triglyceride-form)

Dose: 3-4 g combined EPA/DHA daily (pharmaceutical-grade recommended) | With the largest meal of the day for best absorption
Addresses three converging high-priority pathways in a single intervention: (1) Cardiovascular — triglyceride reduction and anti-platelet effects counteract F5 rs6025 thrombotic risk and LRP8 CAD susc

Vitamin D3 + MK-7 Vitamin K2

Dose: 4000-5000 IU D3 + 100-200 mcg MK-7 K2 daily | With breakfast or the first fat-containing meal (fat-soluble vitamins)
Vitamin D deficiency amplifies autoimmune risk (SLE 94th percentile, RA 90th — D3 is a critical immune system modulator downstream of IRF5); improves insulin sensitivity (T2D PRS 85.9th percentile); r

CoQ10 (Ubiquinol form)

Dose: 200-400 mg daily | With fatty meal; split dose if using 400 mg
ABCG2 rs2231142 G/T reduces statin clearance, which both increases statin drug exposure and amplifies CoQ10 depletion when statins are prescribed (statins block the mevalonate pathway that produces Co

L-Theanine

Dose: 200-400 mg daily | 200 mg in morning paired with coffee (synergistic for focus); 200 mg 60-90 minut
ADHD PRS 99.3rd percentile combined with evening chronotype 94.1th percentile creates a destructive cycle: ADHD impulse dyscontrol delays sleep onset, sleep deprivation worsens ADHD symptoms 4-fold, a

Quercetin (with Bromelain for absorption)

Dose: 500-1000 mg quercetin + 250 mg bromelain daily | With meals; bromelain enhances quercetin bioavailability
96th percentile gout PRS represents one of the highest-penetrance risk signals in this genome — chronic hyperuricemia damages kidneys (73rd CKD PRS), predisposes to painful gout flares, and acts as a

Alpha-Lipoic Acid (R-ALA form)

Dose: 600 mg daily | On empty stomach 30-45 minutes before a meal (best absorption kinetics)
R-ALA is both a potent insulin sensitizer (activates AMPK and GLUT4 translocation — directly addressing TCF7L2 + FTO T2D convergence) and a universal antioxidant operating in both aqueous and lipid co

N-Acetyl Cysteine (NAC)

Dose: 600-1200 mg daily (600 mg twice daily with meals) | 600 mg with breakfast and 600 mg with dinner
Glutathione precursor supporting two key pathways: (1) SLE (94th percentile) + RA (90th percentile) autoimmune convergence — oxidative stress is a key trigger for autoimmune flares via IRF5 pathway, a

TRAINING PROTOCOL

Training Type: Hybrid

99.1th percentile BMI PRS + 85.9th T2D PRS + 90.7th HTN PRS demand aggressive metabolic conditioning — Zone 2 cardiovascular training is the gold standard intervention for mitochondrial fat oxidation and insulin sensitivity, while resistance training is essential to build the lean muscle mass that serves as the primary glucose sink (every 5 kg of lean mass reduces T2D risk approximately 15-20%). T

1. Zone 2 Cardiovascular Training (cycling, swimming, brisk walking, rowing)**4-5 days per week, 45-60 minutes per session**

Zone 2 (60-70% maximum heart rate, conversational pace) is the most potent metabolic medicine for this genome — it maximally activates mitochondrial biogenesis, fat oxidation pathways, and GLUT4 glucose transporters, directly counteracting the FTO-TCF7L2 insulin resistance convergence and attacking

2. Resistance Training (full-body compound movements: squats, deadlifts, rows, press, lunges)**2-3 days per week, 45-60 minutes, non-consecutive days**

Compound resistance training is metabolically irreplaceable for this genome: building lean mass increases the insulin-independent glucose uptake capacity of skeletal muscle, which is the single largest driver of improving insulin sensitivity beyond Zone 2 cardio. Resistance training also dramatically

3. Post-meal Walking (light, 10-20 minutes after main meals)**Daily after lunch and dinner**

Post-prandial walking reduces acute glucose excursions by 25-30% — highly targeted at TCF7L2 beta-cell dysfunction where impaired insulin response amplifies glucose spikes. This low-intensity activity has no seizure risk, requires no planning, and works as a 'metabolic interrupt' that directly address

4. Yoga or Mindfulness-Based Movement (flexibility, breathwork, body scan)**2 times per week, 30-45 minutes (evening sessions preferred)**

Chronic stress activates cortisol which directly worsens insulin resistance, triggers autoimmune flares (SLE/RA IRF5 pathway), and dramatically amplifies ADHD symptom severity. BDNF rs6265 Val/Val genotype shows heightened stress-induced memory consolidation and anxiety under threat — structured par

DIETARY PROTOCOL

- Anti-inflammatory Mediterranean foundation: extra virgin olive oil, fatty fish, colorful vegetables, legumes, nuts, and whole grains form the backbone — simultaneously addresses the SLE (94th percentile), RA (90th percentile), and cardiovascular convergence with a single dietary framework
- Low-glycemic-index strategy is mandatory: TCF7L2 rs7903146 impairs beta-cell insulin response to glucose — choosing low-GI carbohydrates (whole grains over refined, fiber-rich vegetables, legumes) blunts the glucose spikes that TCF7L2 dysfunction cannot adequately buffer
- Electrolyte-conscious eating — do NOT aggressively restrict sodium: SLC12A3 Gitelman variant causes renal salt wasting, meaning overly aggressive sodium restriction can precipitate dangerous hyponatremia, fatigue, and worsen electrolyte imbalances; aim for moderate sodium from whole food sources, not low-sodium processed alternatives
- Weight management is the highest-leverage single intervention in this genome: 5% body weight reduction reduces cardiovascular event risk 20-30% and T2D incidence 58%; a sustainable 500-750 kcal/day deficit through caloric awareness (not starvation) should be the overarching goal until a healthy BMI is reached
- Low-purine protein sourcing: 96th percentile gout PRS demands avoiding organ meats and shellfish while prioritizing plant proteins (lentils, beans), chicken, fish, and low-fat dairy — purines from vegetables (spinach, mushrooms) are well-tolerated and do not raise uric acid clinically
- Protein adequacy at 1.4-1.6 g/kg body weight: sufficient protein preserves lean muscle mass during caloric restriction, supports BDNF synthesis (critical for ADHD cognition via rs6265 pathway), and maintains satiety (important for ADHD impulse control around food)
- Chrono-nutrition alignment: the 94.1th percentile evening chronotype means metabolic tolerance and insulin sensitivity peak later in the day — a moderate dinner is metabolically appropriate; avoid very large meals after 9 PM when circadian insulin signaling declines
- Hydration: 2-3 liters water daily; Gitelman-related electrolyte wasting is amplified by dehydration, and adequate hydration reduces gout flare risk (uric acid dilution and renal clearance)

LIFESPAN IMPACT ESTIMATES**Sustained 10% body weight reduction and maintenance****+8-12 yrs**

At 99.1th percentile BMI PRS with a multiplicative cardiometabolic risk stack (T2D 86%, HTN 91%, CAC 91%, stroke 88%), obesity acts as the master amplifier — each 5 BMI unit increase raises MI risk 25-30%. Sustained 10% weight reduction reduces all-cause mortality 20-30%, cardiovascular mortality 40%, and T2D incidence 58% (Diabetes Prevention Program). Combined with the bottom-15th percentile longevity PRS, aggressive weight management could recover the longevity deficit by removing the primary

Blood pressure control to below 120/80 mmHg (preemptive)

+4-7 yrs

90.7th percentile early-onset HTN PRS predicts hypertension developing in the 4th-5th decade or earlier. SPRINT trial: systolic target below 120 reduced major cardiovascular events 25% and all-cause mortality 27% vs standard 140 target. The critical insight is early intervention — controlling HTN before vascular damage occurs has a disproportionately larger longevity benefit than treating established hypertension with existing organ injury.

Type 2 diabetes prevention (maintain HbA1c below 5.7%)

+4-6 yrs

TCF7L2 rs7903146 (GWAS $p=1e-1315$) + FTO + 85.9th T2D PRS create near-certain T2D trajectory without aggressive prevention. Diabetes Prevention Program: intensive lifestyle reduces T2D incidence 58%, metformin reduces by 31%. Developing T2D adds approximately 6-7 cardiovascular years of risk age and dramatically accelerates the coronary calcification trajectory (already at 91.4th percentile). Preventing onset rather than treating T2D removes a major multiplier from the entire convergence signal.

ADHD formal diagnosis and evidence-based treatment

+3-5 yrs

Barkley meta-analysis: untreated ADHD is associated with 13-14 year reduction in life expectancy through accidents, cardiovascular comorbidity, metabolic dysfunction, and medical non-adherence. At 99.3rd percentile genetic liability, ADHD expression is near-certain; effective treatment (stimulant medication + behavioral interventions) is the infrastructure that enables adherence to every other protocol in this plan — diet, exercise, sleep, and medication compliance all improve dramatically with

Zone 2 aerobic training (150+ minutes per week, sustained lifelong)

+3-5 yrs

JAMA meta-analysis (n=116,221): 150-300 minutes/week moderate exercise associated with 17% lower all-cause mortality. For this genome specifically, exercise simultaneously improves insulin sensitivity (T2D prevention), lowers blood pressure, reduces uric acid production (gout), upregulates BDNF at the rs6265 pathway (ADHD cognitive protection), and produces potent anti-inflammatory effects counteracting the SLE/RA autoimmune convergence — making it the only single intervention with documented be

Sleep optimization aligned to chronotype (7-8 hours on evening schedule)

+2-4 yrs

Evening chronotype (94.1th percentile) forced into early social schedules creates chronic social jetlag — equivalent metabolically to repeated transmeridian travel. Sleep deprivation independently raises T2D risk 30%, cardiovascular disease 20%, and amplifies ADHD symptoms 4-fold. Meta-analysis: below 6 hours/night associated with 12% higher all-cause mortality. Aligning sleep schedule to the genetic chronotype (sleeping 12-8 AM rather than 11-7 AM, if possible) dramatically improves sleep quali

Autoimmune disease early detection and proactive management

+2-4 yrs

SLE (94th percentile) + RA (90th percentile) genetic risk creates a meaningful probability of autoimmune disease activation. Active autoimmune disease raises acute MI risk 2-5 fold and stroke risk 2-3 fold through systemic vascular inflammation — layering catastrophically on top of the existing cardiovascular convergence. Early biological DMARDs (if SLE/RA diagnosed) prevent the autoimmune-cardiovascular cascade that could accelerate the already elevated coronary calcification and stroke PRS tra

Statin therapy initiation guided by CAC score (pravastatin low-dose with CoQ10)

+2-3 yrs

91.4th percentile coronary calcification PRS + LRP8 rs5174 (CADD 34, MI susceptibility, REVEL 0.632) indicate a high early CAD trajectory. CTT meta-analysis: statin therapy reduces major cardiovascular events 25-35% in high-risk individuals. Given ABCG2 G/T reduced statin clearance, use pravastatin 20-40 mg (not rosuvastatin high-dose) with CoQ10 200-400 mg co-supplementation. CAC score confirmation prior to initiation avoids unnecessary treatment in those with zero calcification despite elevate

IMPORTANT NOTICE

This health protocol is generated from genetic data analysis and is for informational purposes only. It does not constitute medical advice or a clinical prescription. All supplement, dietary, and exercise recommendations should be discussed with your healthcare provider before implementation. Individual responses may vary.