Personalised *Genomic Analysis.*

133 genetic markers · 12 biological domains · Written personally

Prepared for	Report Date	Platform
A.M.	**August 2026**	**23andMe v5**

Sample report — anonymised, not a real client. Educational purposes only. Does not constitute medical advice or treatment.

CLINICAL CONTEXT

Primary complaints: Chronic fatigue, anxiety, difficulty concentrating, mood instability, poor sleep despite adequate hours. Morning energy consistently below baseline. Sensitivity to caffeine — feels worse not better. Has tried multiple B-complex vitamins with no improvement and occasional worsening.

Current supplementation: B-complex (standard folic acid), magnesium oxide, zinc, omega-3. No current medications.

What this report works through: Standard B-complex is likely counterproductive for this profile. Folic acid cannot be converted by an impaired MTHFR enzyme and actively competes at the receptor. The worsening on B-vitamins is a COMT-mediated catecholamine accumulation effect — not a detox response.

PRIORITY ACTION SUMMARY

PRIORITY	INTERVENTION	GENETIC RATIONALE	TIMING
URGENT	Stop folic acid. Switch to 5-MTHF (Quatrefolic or Metafolin only)	MTHFR TT — folic acid cannot be processed; active receptor blocker	Now
HIGH	Switch to methylcobalamin (sublingual preferred)	FUT2 AA — structural gut B12 absorption impairment	Week 1
HIGH	Add magnesium glycinate (concurrent COMT buffer)	COMT Met/Met — prevents catecholamine accumulation during methylation support	Week 1
MODERATE	Caffeine — morning only, cut before noon	CYP1A2 slow + COMT Met/Met — caffeine compounds catecholamine load	Now

Methylation is the biochemical foundation for neurotransmitter synthesis, gene expression, hormone clearance, and cellular energy. The variants below interact as a system.

MTHFR C677T

rs1801133 | **TT · Homozygous** | SIGNIFICANT

The C677T substitution reduces MTHFR enzyme activity by approximately 70% in TT homozygous individuals — the rate-limiting step for converting dietary folate to 5-methyltetrahydrofolate. Downstream consequences include reduced SAM-e availability, impaired homocysteine recycling, and reduced serotonin and dopamine synthesis substrate.

Standard folic acid competes with active folate at the receptor and worsens the functional deficit. The reported worsening on standard B-complex supplementation is consistent with this mechanism.

Frosst P et al. Nat Genet 1995;10:111. Schneider JA et al. Neurology 2000;54:1265.

COMT Val158Met

rs4680 | **Met/Met · Slow clearance** | SIGNIFICANT

COMT clears dopamine, adrenaline, and noradrenaline from the prefrontal cortex. Met/Met produces an enzyme 3-4x slower than Val/Val. Catecholamines accumulate during stress, focused work, or stimulant use — producing anxiety and exhaustion after cognitive effort.

Critical interaction: active folate and methylcobalamin increase catecholamine synthesis. When COMT cannot clear the downstream load, anxiety and insomnia result. The protocol addresses this with staged introduction and concurrent magnesium as a buffer.

Lachman HM et al. Pharmacogenetics 1996;6:243. Chen J et al. Science 1997;278:1311.

MAOA Promoter VNTR

rs6323 | **Low-activity allele** | MONITOR

MAOA clears serotonin, dopamine, and noradrenaline across the limbic system. Low-activity MAOA means emotional reactions run more intensely and persist longer — not psychologically but because neurochemical clearance operates slowly.

Combined with slow COMT: dual monoamine clearance deficit. Tyramine-rich foods — aged cheese, fermented foods, cured meats — may produce more pronounced responses in this profile.

Caspi A et al. Science 2003;301:386. Brunner HG et al. Science 1993;262:578.

PROTOCOL PREVIEW — ONE SUPPLEMENT CARD

Each supplement card documents the research form, timing, food context, and the specific reason it exists for this genetic profile. Research doses from published literature. Final dose decision rests with your clinician.

5-MTHF Folate — Active Form

Research dose: 400–800 mcg · Daily

FORM	**Quatrefolic (Gnosis) or Metafolin (L-5-MTHF)** — not folic acid in any form. Pre-converted molecule that bypasses the impaired MTHFR enzyme. Standard B-complex folic acid is contraindicated for TT carriers.
TIMING	Morning with your largest meal. Consistent daily timing matters more than the exact hour.
WITH FOOD	**Required.** Protein and fat slow absorption and reduce overstimulation risk for COMT Met/Met. Not optional for this genetic combination.
RESEARCH DOSE	400–800 mcg in published literature. Titration schedule and ceiling in the full protocol.

Why this for you specifically: MTHFR TT genotype means the enzyme converting dietary folate to active form operates at 30-40% efficiency. Folic acid competes at the receptor. Switching to 5-MTHF removes this block. Worsening on standard B-vitamins is mechanistically consistent with this finding.

COMT Sensitivity Note: Met/Met genotype — start at 400 mcg. Assess for 2 weeks before increasing. Symptoms of accumulation: anxiety, wired feeling, disrupted sleep. These mean reduce, not stop. Concurrent magnesium glycinate is documented as catecholamine buffer in the full protocol.

Your complete protocol includes 8–12 supplement cards, current supplement stack review, drug-supplement interaction notes, food-as-amplifier guidance, and a 90-day implementation plan.

Your results came back normal.
You still don't feel normal.

This sample showed one genetic section and one supplement card. Your full DNA Blueprint covers 133 markers across 12 biological domains — interpreted as a system. Written personally. Not generated by software.

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$397	**$697**	**$1,197**
133 markers · 12 domains Full protocol + drug interactions Written personally	Everything in Tier 1 + Bloodwork integration + Written Q&A; session	Everything in Tier 2 + 3 monthly updates + 3 written Q&A; sessions