This document was generated by EverMint Health and reviewed by a clinical professional. It is intended for clinical use only and should not replace direct medical advice.

Pathway to Longevity

Alex



85	60	12.3	7.5
Quality of Life	MLI Score	Lifespan	Healthspan
wellbeing / 100	provisional	of 41 yrs at risk	of 25 working yrs

Primary Focus

Cardiometabolic & Lipid Optimization

Alex's recent baseline labs reveal a clinically significant lipid profile requiring priority attention: total cholesterol 245 mg/dL (ref <200), LDL-C 144 mg/dL (ref <100), LDL particle number 2,136 nmol/L (ref <1,138 nmol/L optimal), LDL Pattern B with peak size 217.4 Å (ref >222.9 Å optimal), Apolipoprotein B 119 mg/dL (in the moderate range, ref optimal <90), Lipoprotein(a) 76 nmol/L (just above the moderate threshold of 75 nmol/L), and Non-HDL cholesterol 168 mg/dL (ref <130 mg/dL). These findings converge with accepted genetic signals for elevated cholesterol risk, chronic inflammation risk, pro-inflammatory fat, vascular health susceptibility, and blood pressure dysregulation risk, making cardiometabolic and lipid optimization the most data-supported primary focus. A positive ANA at 1:80 titer with dual patterns (Nuclear Homogeneous and Cytoplasmic Rods and Rings) and a family history notable for diabetes, stroke, and autoimmune disorders further reinforce the need for a proactive, comprehensive approach.

Top 3 Immediate Priorities

1. Clinician review of the markedly elevated Vitamin B12 (>2,000 pg/mL), the positive ANA (1:80, dual pattern), and the awaiting-review pharmacogenomic CYP2D6 anxiety drug interaction note — these findings require clinical interpretation before the plan can be fully activated and before any supplement or medication adjustments are made

2. Establish a confirmed medication list by clinician review of reported Rx A , Rx B , ibuprofen, and Midol — these have direct interactions with the pharmacogenomic results on record (CYP2D6 ultrarapid, CYP2C19 rapid, SLCO1B1 decreased) and must be reconciled before any lipid or mood-related interventions are added

3. Begin capturing the four missing lifestyle inputs (sleep quality, stress load, weekly activity, blood-work recency) to convert the provisional MLI score to a complete baseline, and initiate the anti-inflammatory, lipid-supportive dietary and exercise foundation outlined in the Activate phase

Health Influencers

Lipid & Particle Profile — 30%

Multiple markers out of optimal range: Total cholesterol 245 mg/dL, LDL-C 144 mg/dL, LDL-P 2,136 nmol/L, LDL Pattern B, ApoB 119 mg/dL, Lp(a) 76 nmol/L, Non-HDL 168 mg/dL; HDL-C 77 mg/dL and Chol/HDL ratio 3.2 are favorable.

Genetic predisposition for elevated cholesterol and pro-inflammatory fat compound the lab findings. HDL-C and Chol/HDL ratio are protective elements. Lp(a) elevation is largely genetically determined and not modifiable by lifestyle alone — clinician should note for long-term cardiovascular risk stratification.

Genetic Cardiometabolic Risk Profile — 20%

Accepted genetic signals include elevated cholesterol risk, vascular health susceptibility, blood pressure dysregulation risk, chronic inflammation risk, pro-inflammatory fat, blood sugar dysregulation risk, and hormone balance impairment risk.

These are genetic predispositions from an accepted document, not clinical diagnoses. They inform the direction of the plan and heighten the importance of modifiable lifestyle and nutritional factors. A hereditary heart health panel identified at least one variant of uncertain significance (VUS) — no pathogenic or likely pathogenic variants were identified; VUS follow-up note is awaiting clinician review.

Inflammation & Immune Markers — 20%

hs-CRP 1.4 mg/L (in the average-risk range per lab reference: optimal <1.0, average risk 1.0–3.0); ESR 9 mm/h (normal); ANA positive at 1:80 titer with dual patterns (Nuclear Homogeneous and Cytoplasmic Rods and Rings); standard CRP <3.0 mg/L (normal); family history includes autoimmune disorders and rheumatoid arthritis.

The ANA positivity at 1:80 with dual patterns is an abnormal result requiring clinician interpretation and potential rheumatology follow-up. It does not establish an autoimmune diagnosis on this record. Genetic signals for chronic inflammation risk and oxidative stress risk amplify the importance of monitoring this domain.

Metabolic & Body Composition — 15%

BMI 24.5 (recorded). Fasting glucose 86 mg/dL (normal), HbA1c 5.5% (normal, ref <5.7%). Accepted genetic signals include blood sugar dysregulation risk, slower resting metabolism, high fat storage potential, reduced exercise fat burning response, and increased appetite/reduced satiety.

Current glucose and HbA1c are within reference ranges. Genetic predispositions toward adipogenesis, weight gain/loss resistance, and reduced fatty acid metabolism warrant proactive nutritional and exercise strategies even in the absence of a metabolic diagnosis.

Nutritional & Methylation Status — 10%

Vitamin B12 >2,000 pg/mL (above lab reference range of 200–1,100 pg/mL — markedly elevated; clinical note awaiting clinician review). Folate serum >24.0 ng/mL (normal). Vitamin D 25-OH 30 ng/mL (at the lower boundary of the lab's optimal range of 30–100 ng/mL). Vitamin B6 17.3 ng/mL (normal). Accepted genetic signals include methylation inefficiency, increased choline requirement, reduced folate utilization, and increased vitamin D requirement — some of these are in the awaiting-clinician-review queue.

The markedly elevated Vitamin B12 is flagged in the awaiting-clinician-review queue and must not be interpreted as a finding until reviewed. Vitamin D at exactly the lower boundary of optimal is notable, especially given a genetic signal for increased vitamin D requirement. Methylation and folate pathway genetics (MTHFR compound heterozygous variants are listed as awaiting clinician review from the Blueprint document and cannot be stated as a confirmed finding) should inform supplement recommendations once reviewed.

All reproductive hormones within reference range for menstruating female at time of draw (baseline) : Estradiol 122 pg/mL, Progesterone 15.4 ng/mL (consistent with

Hormonal Status	luteal phase), FSH 5.5 mIU/mL, LH 9.5 mIU/mL, Total Testosterone 28 ng/dL, Free Testosterone 2.8 pg/mL, Prolactin 5.4 ng/mL, Total Estrogens 236 pg/mL, DHEA-S 44 mcg/dL (low-normal within ref range 19–237 mcg/dL), Cortisol 14.4 mcg/dL (normal), TSH 2.71 mIU/L (normal), Free T4 1.2 ng/dL (normal), Free T3 2.9 pg/mL (normal).	**5%**

Accepted genetic signal for hormone balance impairment risk noted. Current hormone labs are reassuring within reference ranges. DHEA-S is within range but on the lower side; clinician may wish to monitor longitudinally given age 40 and the genetic signal.

Critical Lab Findings

LDL Particle Number
2,136 nmol/L — concern

LDL particle number is substantially elevated above the lab's optimal reference of <1,138 nmol/L. Particle number is considered a stronger predictor of atherosclerotic risk than LDL-C alone, particularly when paired with Pattern B and elevated small and medium LDL fractions.

LDL-Cholesterol (calc)
144 mg/dL — concern

LDL-C is above the lab reference of <100 mg/dL. Combined with elevated LDL particle number and Apolipoprotein B, this represents a consistent signal of atherogenic lipid burden.

Apolipoprotein B
119 mg/dL — attention

ApoB falls in the moderate range per lab reference (optimal <90, moderate 90–129, high ≥130). ApoB reflects the total burden of atherogenic particles and aligns with the elevated LDL-P and Non-HDL findings.

Non-HDL Cholesterol
168 mg/dL — concern

Non-HDL cholesterol is above the lab reference of <130 mg/dL, capturing all atherogenic lipoprotein fractions including LDL, VLDL, IDL, and Lp(a).

Cholesterol, Total
245 mg/dL — concern

Total cholesterol exceeds the lab reference of <200 mg/dL. While HDL-C is favorably elevated at 77 mg/dL (and the Chol/HDL ratio is within optimal range at 3.2), the absolute total cholesterol elevation reflects the LDL and non-HDL burden.

LDL Pattern
Pattern B — concern

LDL Pattern B (peak size 217.4 Å, ref >222.9 Å optimal) indicates a predominance of small, dense LDL particles, which are associated with greater atherogenic potential than large, buoyant LDL.

LDL Small
379 nmol/L — concern

Small LDL particles are substantially elevated above the lab's optimal reference of <142 nmol/L, consistent with Pattern B and increased cardiovascular risk.

LDL Medium
430 nmol/L — concern

Medium LDL particles are elevated above the lab's optimal reference of <215 nmol/L, further contributing to the atherogenic particle burden.

Lipoprotein (a)
76 nmol/L — attenti

Lp(a) sits just above the lab's moderate threshold of 75 nmol/L (optimal <75, moderate 75–125, high >125). Lp(a) elevation is largely genetically determined and independently increases cardiovascular risk. It should factor into long-term risk stratification.

ANA Screen, IFA — POSITIVE (titer 1:80, dual pattern: Nuclear Homogeneous and Cytoplasmic Rods and Rings) — concern

A positive ANA at 1:80 titer with two distinct patterns detected on reflex testing is an abnormal result. It does not establish an autoimmune diagnosis but warrants clinician interpretation, clinical correlation, and consideration of rheumatology referral, particularly given the family history of autoimmune disorders and rheumatoid arthritis.

High-Sensitivity CRP (hs-CRP) — 1.4 mg/L — attention

hs-CRP falls in the average cardiovascular risk range (lab reference: optimal <1.0, average 1.0–3.0, higher risk 3.1–10.0). While not in the high-risk range, it is above optimal and is relevant in the context of the lipid findings and genetic inflammation risk signals.

Vitamin B12 — >2,000 pg/mL — concern

Vitamin B12 exceeds the upper limit of the lab reference range (200–1,100 pg/mL) and is reported as markedly elevated. A clinical note regarding this finding is listed as awaiting clinician review. The cause (supplementation, dietary, or other) has not been established on this record and requires clinician assessment before any B12 supplementation recommendations are made.

Vitamin D, 25-OH, Total — 30 ng/mL — attention

Vitamin D sits exactly at the lower boundary of the lab's optimal range (30–100 ng/mL). Given the accepted genetic signal for increased vitamin D requirement, maintaining and ideally improving this level is warranted. A clinical note about this finding is awaiting clinician review.

HDL Cholesterol — 77 mg/dL — optimal

HDL-C is well above the lab's reference threshold of ≥50 mg/dL, and HDL Large particles are at 12,855 nmol/L (above the optimal reference of >6,729 nmol/L). This is a favorable protective element within an otherwise elevated lipid profile.

Hemoglobin A1c — 5.5% — optimal

HbA1c is within the normal range (<5.7%), indicating good average glucose control at the time of testing. This is reassuring in the context of a genetic signal for blood sugar dysregulation risk.

Glucose, Fasting — 86 mg/dL — optimal

Fasting glucose is within the normal reference range (65–99 mg/dL).

eGFR — 115 mL/min/1.73m² — optimal

Kidney filtration is robust and well above the threshold of ≥60 mL/min/1.73m².

Albumin/Creatinine Ratio, Random Urine — 3 mg/g creat — optim

No evidence of microalbuminuria; urine ACR is well within the normal range (<30 mg/g creat).

BNP (B-Type Natriuretic Peptide)

5 pg/mL **optimal**

BNP is well within the normal range (<100 pg/mL), providing reassurance regarding cardiac wall stress at this time.

Homocysteine

6.0 umol/L **optimal**

Homocysteine is within the normal reference range (≤11.0 umol/L), a favorable finding given the genetic signal for methylation inefficiency.

Triglycerides

127 mg/dL **optimal**

Triglycerides are within the normal reference range (<150 mg/dL). Maintaining this level through dietary and lifestyle measures is important given the metabolic and fat storage genetic risk profile.

Genetic Pathway Considerations

Lipid Metabolism & Cardiovascular Risk

Accepted genetic signals for elevated cholesterol risk, pro-inflammatory fat, vascular health susceptibility, and blood pressure dysregulation risk align with and compound the observed lab findings. These predispositions suggest that lipid management will require sustained, multi-modal strategies rather than single-point interventions.

- Prioritize dietary saturated fat reduction and increase of fiber-rich, plant-based foods to support LDL and non-HDL reduction
- Incorporate omega-3-rich foods (clinician to assess whether omega-3 supplementation is appropriate given reported ibuprofen use and unconfirmed medications)
- Monitor blood pressure regularly given genetic blood pressure dysregulation risk and salt sensitivity signal (salt sensitivity is in the awaiting-clinician-review queue)
- Clinician to consider whether statin suitability assessment is warranted — note SLCO1B1 decreased function (pharmacogenomic result on record) which may affect statin transport and myopathy risk
- Long-term Lp(a) monitoring given genetic elevation likelihood

Inflammation & Oxidative Stress

Accepted genetic signals for chronic inflammation risk and oxidative stress risk, combined with an hs-CRP in the average-risk range and a positive ANA, suggest an inflammatory milieu that may contribute to both cardiometabolic and immune system burden.

- Anti-inflammatory dietary pattern (noted in awaiting-clinician-review queue as a follow-up recommendation from the Blueprint document)
- Emphasize antioxidant-rich foods (colorful vegetables, berries, cruciferous vegetables)
- Clinician to assess adequacy of Vitamin C intake given accepted genetic signal for increased Vitamin C requirement
- Reduce pro-inflammatory dietary contributors (refined carbohydrates, ultra-processed foods)
- Monitor hs-CRP and ESR longitudinally

Methylation & Folate Utilization

Accepted genetic signals for methylation inefficiency and reduced folate utilization are present. Homocysteine is currently normal at 6.0 umol/L, which is reassuring, though this pathway warrants ongoing monitoring. MTHFR compound heterozygous variants are listed as awaiting clinician review from the Blueprint document and cannot be stated as a confirmed finding at this time.

- Clinician to review MTHFR variant status from awaiting-review items before making specific methylation supplement recommendations
- Prioritize dietary methylation support: dark leafy greens, legumes, eggs (choline source), and B-vitamin-rich whole foods
- Do not add B12 supplementation until the markedly elevated serum B12 (>2,000 pg/mL) has been clinician-reviewed and explained
- Recheck homocysteine at next lab draw to monitor methylation status longitudinally
- Increased choline requirement genetic signal supports emphasis on choline-rich food source

Metabolic Rate, Fat Storage & Weight Regulation

Multiple accepted genetic signals converge on metabolic disadvantage: slower resting metabolism, high fat storage potential, increased appetite/reduced satiety, reduced exercise fat burning response, weight gain and weight loss resistance, and reduced fatty acid metabolism. These predispositions mean Alex may need to work harder with lifestyle and nutrition strategies to achieve and maintain body composition goals relative to the general population.

- Structured resistance and strength training to support resting metabolic rate (genetic signal for above-average power potential is favorable for strength training responsiveness)
- Protein-forward eating pattern to support satiety given increased appetite/reduced satiety genetic signal
- Consistent meal timing and avoidance of prolonged fasting or severe caloric restriction, which may further slow metabolism
- Exercise programming should account for genetic signal of reduced exercise fat mobilization — duration and consistency may matter more than intensity alone
- Clinician and exercise specialist collaboration recommended (noted in awaiting-review follow-up recommendations)

Bone Health

Accepted genetic signal for bone health imbalance risk is present. Current PTH, calcium, and phosphorus are within normal ranges. Vitamin D is at the lower boundary of optimal, and a genetic signal for increased vitamin D requirement is accepted.

- Support vitamin D level above the lower boundary; clinician to determine appropriate supplementation dose based on 30 ng/mL baseline and increased requirement genetic signal
- Ensure adequate dietary calcium from whole food sources
- Weight-bearing and resistance exercise supports bone density
- Monitor vitamin D 25-OH longitudinally, particularly given both genetic signal and baseline value at the lower edge of optimal

Detoxification & Liver Health

Accepted genetic signals for detoxification impairment risk are present. Liver enzymes (AST 19 U/L, ALT 15 U/L, GGT 18 U/L, ALP 44 U/L, bilirubin 0.4 mg/dL) are all within normal reference ranges, which is reassuring. UGT1A1 intermediate metabolizer status is on record (pharmacogenomic result) and may affect bilirubin and drug metabolism.

- Support detoxification pathways with cruciferous vegetables, adequate hydration, and fiber
- Minimize unnecessary hepatic burden: limit alcohol and unnecessary over-the-counter NSAIDs (relevant to reported ibuprofen and Midol use — clinician to advise)
- Monitor liver enzymes at subsequent lab draws
- UGT1A1 intermediate metabolizer status should be communicated to any prescribing providers

Mood & Cognitive Health

Accepted genetic signals for mood and behavior susceptibility, mood imbalance susceptibility, and cognitive decline risk are present. Anxiety is reported on the chart as imported from an outside record and is not yet confirmed. Reported medications include Rx A and Rx B (neither confirmed by a clinician).

- Clinician to confirm or clarify the reported anxiety history and reported use of Rx A and Rx B before making any mood-related supplement or intervention recommendations — CYP2D6 ultrarapid metabolizer status on record has pharmacogenomic implications for some psychiatric medications (clinical note awaiting review)
- Anti-inflammatory dietary pattern and omega-3-rich foods support both mood and cognitive health
- Sleep quality and stress load are missing inputs for the provisional MLI score — capturing these is a priority for completing the risk picture
- Cardiovascular health optimization (the primary focus of this plan) directly supports cognitive health outcomes long-term

Collagen, Joint & Connective Tissue

Accepted genetic signals for collagen and joint tissue damage susceptibility are present. Family history includes chronic back pain and rheumatoid arthritis. The positive ANA result requires clinical correlation.

- Clinician to assess joint symptoms in the context of positive ANA and family history of rheumatoid arthritis
- Adequate dietary protein supports collagen synthesis
- Vitamin C intake supports collagen formation — relevant also to the increased Vitamin C requirement genetic signal
- Weight-bearing exercise supports connective tissue health when performed with appropriate load management
- Rheumatology referral may be appropriate pending clinician review of ANA result

Pharmacogenomics

Multiple actionable pharmacogenomic results are on record from the Color Genetics Medication Response panel (on record) : CYP2D6 ultrarapid metabolizer, CYP2C19 rapid metabolizer, CYP1A2 ultrarapid metabolizer, CYP3A5 poor metabolizer, SLCO1B1 decreased function, VKORC1 intermediate function, UGT1A1 intermediate metabolizer, IFNL3 unfavorable response. These results have direct implications for drug dosing and selection across multiple drug classes.

- CYP2D6 ultrarapid metabolizer status: many antidepressants, analgesics, and other medications are substrates — a clinical note in the awaiting-review queue specifically flags a CYP2D6 anxiety drug interaction risk relevant to the reported psychiatric medications (Rx A , Rx B); clinician must review before confirming or adjusting these
- CYP2C19 rapid metabolizer: affects metabolism of PPIs, some antidepressants, antiplatelets (clopidogrel) — relevant if any such agents are considered
- SLCO1B1 decreased function: impairs hepatic uptake of statins (particularly simvastatin and pravastatin), increasing myopathy risk — critical consideration if statin therapy is considered for lipid management
- VKORC1 intermediate function: affects warfarin sensitivity — relevant if anticoagulation is ever considered
- CYP1A2 ultrarapid metabolizer: affects caffeine, some antidepressants, and clozapine — consistent with accepted genetic signal for fast caffeine metabolism
- All pharmacogenomic results should be shared with all prescribing providers as noted in the awaiting-review follow-up recommendation

Phases — Activate → Progress → Optimize

Nutrition

Activate **Foundation: Anti-Inflammatory, Lipid-Supportive Eating Pattern**

Establish a whole-food, anti-inflammatory dietary foundation that directly addresses the elevated lipid and inflammation findings while respecting genetic metabolic predispositions toward fat storage and reduced satiety.

- Increase soluble fiber intake through oats, legumes, flaxseed, and vegetables to support LDL and non-HDL reduction
- Replace saturated fats with unsaturated fat sources (avocado, olive oil, nuts, seeds) aligned with lipid-lowering goals
- Prioritize lean protein at every meal to support satiety given accepted genetic signal for increased appetite and reduced satiety
- Reduce refined carbohydrates and added sugars to protect triglycerides and support blood sugar stability given the blood sugar dysregulation genetic signal
- Increase dietary choline-rich foods (eggs, legumes, cruciferous vegetables) to address increased choline requirement genetic signal
- Include colorful, antioxidant-rich vegetables and berries daily to address oxidative stress and inflammation genetic signals
- Note: omega-3 fatty acid food sources and any supplement recommendations should be reviewed with clinician given reported ibuprofen and Midol use (potential additive antiplatelet effect)

Progress **Refinement: Targeted Nutrient Density & Metabolic Support**

Deepen dietary quality with targeted attention to nutrients relevant to Alex's genetic profile and lab findings, and begin tracking dietary patterns for clinician review.

- Incorporate regular servings of fatty fish (e.g., salmon, sardines, mackerel) as a whole-food omega-3 source for cardiovascular support — clinician to confirm safety given reported NSAID use
- Emphasize Vitamin C-rich foods (bell peppers, citrus, kiwi, strawberries) to address increased Vitamin C requirement genetic signal and collagen support
- Support methylation pathways with dietary folate (dark leafy greens, lentils, asparagus) and B-vitamin-rich foods, particularly as MTHFR variant status awaits clinician review
- If salt sensitivity genetic signal is confirmed after clinician review of awaiting items, begin mindful sodium reduction strategy
- Track dietary pattern over a 2–4 week period and share with care team to inform further personalization
- Introduce more plant-based meals weekly to reduce saturated fat load and increase polyphenol intake

Optimize **Precision: Personalized Nutrient Timing & Functional Foods**

Fine-tune dietary strategy based on follow-up lab results and clinician-reviewed genetic findings, optimizing for sustained lipid improvement and long-term cardiometabolic resilience.

- Reassess and adjust macronutrient distribution based on follow-up lipid panel, HbA1c, and body composition data
- Strategically time higher-carbohydrate intake around exercise bouts to support fat mobilization given reduced exercise fat burning response genetic signal
- Explore functional foods with evidence for lipid support (plant sterols, beta-glucan) after clinician review of full medication and supplement picture
- Maintain dietary choline, folate, and B-vitamin support with ongoing homocysteine monitoring to guide adequacy
- Establish a sustainable long-term dietary pattern that accounts for genetic predisposition toward weight regain — avoid cyclical restrictive dieting

Lifestyle

Activate **Foundation: Structured Movement & Baseline Tracking**

Establish consistent physical activity and begin capturing the missing lifestyle inputs (sleep quality, stress load, weekly activity) that are preventing a complete risk score.

- Begin tracking weekly physical activity, sleep hours/quality, and perceived stress using a method the member can sustain (wearable, app, or journal) — these are the missing inputs for Alex's provisional MLI score
- Introduce 2–3 sessions per week of moderate-intensity cardiovascular exercise to support lipid profile improvement
- Add 2 sessions per week of resistance/strength training — genetic signal for above-average power potential suggests good responsiveness to strength work
- Aim for consistent sleep and wake times to support cortisol regulation and metabolic health
- Clarify surgical history context (appendicitis) with clinician to ensure no activity restrictions apply

Progress

Build: Consistent Routine & Stress Reduction Integration

Increase exercise consistency and volume, and implement targeted stress management practices, with particular attention to the reported anxiety history and the missing stress load input for the longevity index.

- Progress to 150+ minutes per week of moderate cardiovascular activity, with attention to duration over intensity given reduced exercise fat mobilization genetic signal
- Increase resistance training frequency to 3 sessions per week with progressive overload for bone density and metabolic rate support
- Introduce a consistent stress reduction practice (e.g., breathwork, mindfulness, yoga) — especially relevant given reported history of anxiety (not yet confirmed) and the mood/behavior susceptibility genetic signal
- Clinician to assess whether reported Rx A and Rx B use (not yet confirmed) requires coordination with mental health provider before intensifying stress management programming
- Prioritize 7–9 hours of sleep per night; address any sleep barriers with the care team

Optimize

Optimize: Advanced Recovery & Longevity Habits

Sustain and refine lifestyle habits for long-term cardiovascular and metabolic health, and revisit the MLI score once missing inputs are captured to establish a complete baseline.

- Complete the missing lifestyle inputs (sleep quality, stress load, weekly activity, and blood-work recency) to move the MLI from provisional to fully computed
- Implement recovery strategies appropriate to genetic signal for moderate exercise recovery support (adequate protein post-exercise, sleep prioritization, active recovery days)
- Establish cardiovascular health monitoring routine: regular blood pressure checks given blood pressure dysregulation risk genetic signal
- Consider cardiac CT / calcium score (reported as not yet done) — clinician to determine appropriateness given lipid findings, age 40, and family history of stroke
- Maintain annual lab review cadence to track lipid trends, ANA, inflammatory markers, and vitamin D over time

Supplements

Activate **Foundation: Clinician-Reviewed Essentials Only**

No supplement recommendations should be initiated until the clinician has reviewed the markedly elevated Vitamin B12, the awaiting-review pharmacogenomic and genetic items, and confirmed the unconfirmed medication list. The following represents the framework for clinician consideration only.

- HOLD all B12 supplementation pending clinician review of markedly elevated serum B12 (>2,000 pg/mL) — source must be identified before any addition
- HOLD omega-3 / fish oil supplementation pending clinician assessment of reported ibuprofen and Midol use for potential additive antiplatelet effect
- HOLD any psychiatric or mood-related supplements (e.g., 5-HTP, St. John's Wort) pending clinician review of CYP2D6 ultrarapid metabolizer status and reported Rx A /Rx B use
- Clinician to assess whether Vitamin D supplementation is appropriate to maintain/improve the 30 ng/mL baseline, given the increased requirement genetic signal — dose to be determined by clinician
- Clinician to assess magnesium supplementation as supportive for cardiovascular, blood pressure, and bone health domains — no confirmed contraindication on record, but unconfirmed medications must be reviewed first
- Member records no known drug allergies (NKDA) per self-report

Progress **Targeted: Lipid, Methylation & Inflammation Support**

Once clinician has reviewed pending items and confirmed the medication picture, introduce targeted supplements aligned with recorded lab and genetic findings.

- If clinician confirms omega-3 safety: consider pharmaceutical-grade fish oil for lipid and inflammation support — dose to be clinician-determined
- If MTHFR variant status is confirmed after clinician review: consider methylated folate (5-MTHF) and methylated B vitamins as appropriate — only after B12 elevation is explained and managed
- Vitamin D: re-check 25-OH D after 8–12 weeks of any supplementation to confirm adequate response given increased requirement genetic signal
- Clinician to assess Vitamin C supplementation appropriateness given increased Vitamin C requirement genetic signal
- Do not add statin-adjunct supplements (e.g., red yeast rice, which contains monacolin K) without full clinician review — SLCO1B1 decreased function has implications for any statin-like compound

Optimize **Precision: Sustained, Evidence-Aligned Support**

After confirmed medication reconciliation and follow-up labs, refine the supplement regimen to the smallest effective set aligned with Alex's personal data.

- Re-evaluate all supplements against follow-up lipid panel, B12, Vitamin D, and homocysteine results at 3–6 months
- Clinician to assess collagen peptide or Vitamin C supplementation for collagen/joint susceptibility genetic signal, particularly if joint concerns arise clinically
- Bone health support (Vitamin D, magnesium, potentially Vitamin K2) to be formalized based on follow-up labs and DEXA scan if indicated
- Maintain minimal effective supplement regimen — avoid polypharmacy burden given pharmacogenomic complexity (multiple non-standard metabolizer statuses on record)

$ Financial & Life Impact

Total Years at Risk

12.3 yrs

Working Years at Risk

7.5 yrs

Alex's provisional longevity index (MLI 60, computed with defaults for sleep quality, stress load, weekly activity, and blood-work recency) estimates 12.3 years at risk over her lifetime and 7.5 working years at risk — meaning that unaddressed risk factors could affect both her productive working life and her healthspan in later decades. Because this score is provisional due to four missing inputs, the true risk figure may shift meaningfully once complete data are captured, making filling those data gaps a concrete financial and health priority.

Follow-Up Labs

- Repeat comprehensive lipid panel with LDL particle number, ApoB, and Lp(a) in 3–6 months to assess response to dietary and lifestyle interventions
- ANA follow-up: clinician to determine whether anti-dsDNA, anti-Sm, anti-SSA/SSB, anti-Scl-70, or other ANA-reflex antibody testing is indicated given the 1:80 titer with dual patterns and family history of autoimmune disorders — rheumatology referral to be considered
- Vitamin B12 repeat (timed appropriately per clinician) with clinical investigation of the source of markedly elevated level (>2,000 pg/mL)
- Vitamin D 25-OH re-check in 8–12 weeks after any supplementation is initiated, to confirm adequacy given increased requirement genetic signal and current level at the lower boundary of optimal (30 ng/mL)
- Homocysteine: recheck at next lab draw to monitor methylation pathway longitudinally, particularly once MTHFR variant status from awaiting-review items is confirmed
- Fasting insulin and HOMA-IR: not currently on record — clinician to consider adding given genetic signals for blood sugar dysregulation risk and metabolic predispositions
- DEXA scan: not currently on record — clinician to consider for bone density baseline given accepted bone health imbalance risk genetic signal, age 40, and Vitamin D at lower boundary of optimal
- Cardiac CT / coronary artery calcium score: member reports this has not been done — clinician to assess appropriateness given elevated lipid burden (Total cholesterol 245, LDL-C 144, LDL-P 2,136, ApoB 119), age 40, and family history of stroke
- HbA1c and fasting glucose: continue monitoring annually given blood sugar dysregulation genetic risk signal
- Complete blood count and iron studies: current values are within normal ranges — continue monitoring annually
- Thyroid panel (TSH, Free T4, Free T3): currently normal — continue monitoring given autoimmune family history and positive ANA

Care Team Notes

This PTL draft is based on a rich but partially incomplete data set. Several items require clinician action before the plan can be fully activated: (1) The markedly elevated Vitamin B12 (>2,000 pg/mL) has a clinical note in the awaiting-review queue — the source must be identified before any B12 or methylation supplement recommendations can be made. (2) The positive ANA at 1:80 with dual reflex patterns (Nuclear Homogeneous and Cytoplasmic Rods and Rings) requires clinical correlation and likely rheumatology consultation, particularly given family history of autoimmune disorders and rheumatoid arthritis — no autoimmune diagnosis has been established on this record. (3) The reported medications (ibuprofen, Midol, Rx A , Rx B) are unconfirmed by a clinician; the CYP2D6 ultrarapid metabolizer pharmacogenomic result has a specific clinical note in the awaiting-review queue flagging an anxiety drug interaction risk — this must be reviewed before any psychiatric or mood-related supplement or medication changes are made. (4) SLCO1B1 decreased function is on record and is critical context if statin therapy is being considered for the elevated lipid burden. (5) Multiple items from the Blueprint genetic document remain in the awaiting-clinician-review queue including MTHFR compound heterozygous variants, APOE genotype, salt sensitivity, increased vitamin D requirement, reduced folate utilization, and reduced fatty acid metabolism — these should be reviewed and accepted or rejected to complete the genetic picture. (6) The MLI score of 60 is provisional due to four missing inputs (sleep quality, stress load, weekly activity, blood-work recency); capturing these inputs is a concrete early action that will allow a complete risk assessment. (7) The history of anxiety is imported from an outside record and has not been confirmed — it is referenced as reported and awaiting confirmation throughout this plan. (8) The cardiovascular history includes member-reported EKG, Holter monitor, echocardiogram, and chest X-ray — results of these studies are not on file; clinician may wish to obtain records for longitudinal context given the lipid findings.