● PATHWAY APEX · MODE A · LABORATORY VERIFIED · 05/12/2025

Amir Andrei Vakili · Age 28 · Male · Founder, Pathway Apex

49

years old — metabolically

+21 years above chronological age

Your biology is ageing faster than you are. At your current trajectory a private physician would be having a very different conversation with you within 2–3 years. Every marker in today's panel has a direct, addressable root cause.

Lab verified · Week 0	5 primary markers	Mode A · Metabolic Age	30% guarantee
05/12/2025	HOMA · HbA1c · CRP · TG:HDL · T	49 years · Gap +21	HOMA-IR · Week 12 verified

SECTION 1 — OPERATING CAPACITY

You are currently operating at *59% of your biological capacity*

Based on your metabolic state — State 3 · Normal. Every percentage point below 100 represents cognitive output, physical energy, and decision quality your biology is withholding. This is not a motivation problem. It is a measurable metabolic one.

YOUR METABOLIC STATE

State 3 · Normal

HOMA-IR 2.31 · 1.5–2.5 band

Current capacity	59%
Week 12 target	87%
Biological maximum	97–100%

The gap between 59% and 87% is not motivation or effort. It is measurable metabolic dysfunction — and it reverses in 12 weeks.

METABOLIC SPECTRUM — YOUR POSITION

STATE 1 ELITE	STATE 2 OPTIMISED	STATE 3 NORMAL	STATE 4 UNDER	STATE 5 SEVERE	STATE 6 T2D

WHAT EACH STATE ACTUALLY FEELS LIKE

97 % CAP

STATE 1 · ELITE

HOMA-IR <1.0

All-day stable energy. No crashes. Razor-sharp cognition. Wakes refreshed. Fast recovery.

87 % CAP

WEEK 12 TARGET

STATE 2 · OPTIMISED

HOMA-IR 1.0–1.5

Consistently high energy. Rare dips. Clear focus most of the day. Good sleep. Reliable performance.

59 % CAP

← YOU ARE HERE

STATE 3 · NORMAL

HOMA-IR 1.5–2.5

Functional but inconsistent energy. Afternoon crash most days. Occasional fog. Sleep adequate not restorative.

34 % CAP

STATE 4 · UNDER-OPTIMISED

HOMA-IR 2.5–3.8

Significant consistent fatigue. Daily brain fog. Severe crashes. Poor sleep. Recovery impaired.

16 % CAP

STATE 5 · SEVERE

HOMA-IR 3.8–6.0

Exhausted most of the day. Multiple coffees to function. Severe brain fog. Very low libido.

7
% CAP

STATE 6 · T2D THRESHOLD

HOMA-IR 6.0+

Chronic extreme fatigue. Significant cognitive impairment. Multiple medications. Cardiovascular risk 3× elevated.

SECTION 2 — WHAT THIS FEELS LIKE

You have normalised symptoms *that are not normal*

Before the data. How many of these feel familiar right now?

YOUR DAILY EXPERIENCE — RIGHT NOW

- The 2pm meeting where the thread of the conversation keeps slipping.

  You put it down to a bad night. It happens most days. HOMA-IR at 2.31 means every meal drives a larger insulin response than necessary — and a steeper energy crash two hours later.

- You arrive at 8am already running a deficit. First two hours are caffeine and momentum.

  You have accepted this as the price of the job. It is not. It is the signature of a cortisol-disrupted morning combined with systemic inflammation suppressing morning energy systems.

- The evening with the people you care about where you are physically present but not really there.

  The cortisol that fuels professional performance also suppresses the parasympathetic state that makes genuine presence possible. Testosterone at 430 ng/dL compounds this.

- You train five times a week and the markers still look like this.

  At sustained stress 6/10, training above recovery capacity adds cortisol to an already dysregulated system. The effort is real. The adaptation is not arriving. This is the most common metabolic trap in high performers.

None of these are personality traits. None are inevitable. Every one is a measurable downstream consequence of what your metabolic panel shows below — and every one reverses when the root cause is addressed.

SECTION 3 — METABOLIC SPECTRUM

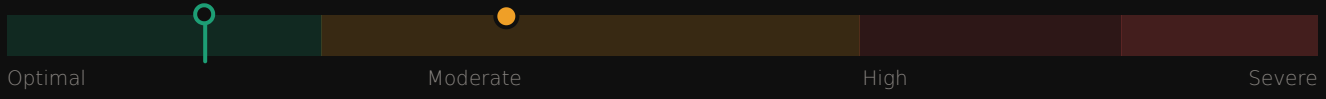
The biological evidence *of what you just recognised*

Five markers from your baseline panel. Each plotted against the full clinical range with a Week 12 target. These five markers determine your metabolic age.

Your baseline position · Week 12 target · Optimal zone

HOMA-IR — Insulin Resistance Index

2.31 → <1.62 (guaranteed) Optimal <1.0



Glucose 104 × Insulin 9 ÷ 405 = 2.31. Primary guarantee metric. Pancreas working 2× harder than required. A 30% improvement to below 1.62 is guaranteed at Week 12. Reference age: 45.

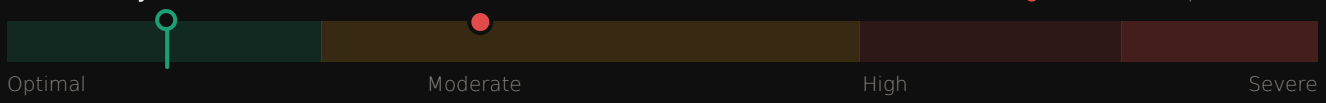
HbA1c — 3-Month Glucose Average

5.6% → <5.4% Optimal <5.0%

At the ceiling of the normal range. Reflects 90 days of sustained glucose elevation — cannot be manipulated by fasting. Three months of insulin resistance crystallised in this number. Reference age: 45.

hsCRP — Systemic Inflammation

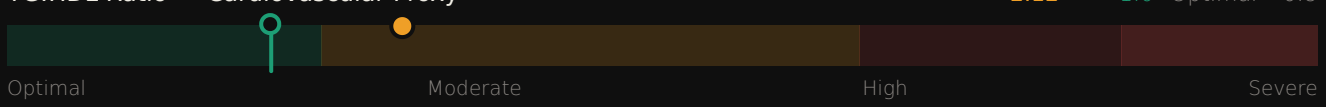
1.8 mg/L → <0.6 Optimal <0.3



Flagged — moderate cardiovascular risk zone (1–3 mg/L). Root cause: Vitamin D at 16.2 ng/mL driving inflammatory cytokine production. D3 + K2 + Omega-3 targets this directly. Reference age: 55.

TG:HDL Ratio — Cardiovascular Proxy

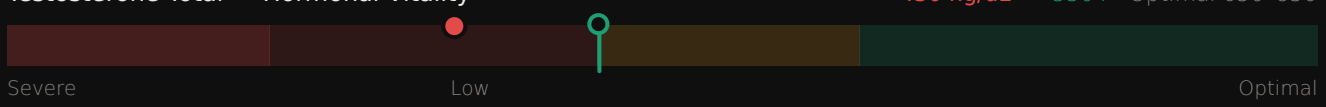
2.12 → <1.0 Optimal <0.8



Triglycerides 108 ÷ HDL 51 = 2.12. Above 2.0 correlates strongly with insulin resistance. Zone 2 cardio is the primary intervention. Reference age: 45.

Testosterone Total — Hormonal Vitality

430 ng/dL → 550+ Optimal 650–850



14.9 nmol/L. A 28-year-old should be 650–900 ng/dL. Root cause: Vitamin D deficiency suppressing Leydig cell testosterone synthesis. No hormonal supplementation required. Reference age: 55.

A metabolically healthy 28-year-old would show

HOMA-IR Below 1.0	HbA1c Below 5.0%	hsCRP Below 0.3 mg/L
TG:HDL Below 0.8	Testosterone 650–900 ng/dL	Vitamin D 40–55 ng/mL

SECTION 4 — METABOLIC LIFESTYLE INDEX

Why your markers *look the way they do*

Six lifestyle domains — each with a direct causal mechanism connecting it to the biomarker profile above. Each score maps to a specific biochemical pathway.

MLI COMPOSITE SCORE

6.2

out of 10

High Acceleration

> *Stress and cortisol — the highest-weighted domain — score 6.0/10. This single domain is estimated to account for 35–40% of your insulin resistance independently of diet. The movement domain is also*

flagged — not because of insufficient effort, but because 5 sessions per week at this stress level was adding cortisol load rather than producing metabolic adaptation.

Sleep Architecture — High weight

5.4

6–7 hrs · Groggy on waking · Slow-wave sleep deficit accumulating nightly. GLUT4 receptor regeneration in skeletal muscle incomplete every morning.

Stress & Cortisol — Highest weight

6.0

Sustained stress 6/10 · Primary independent glucose driver. Cortisol converting stored substrate into circulating glucose every morning before eating — independently of diet.

Alcohol Burden — Moderate weight

0.0

Never or rarely · Zero alcohol burden. A significant protective factor pulling the MLI composite down and providing meaningful headroom.

Movement & Glucose — High weight

5.0

5 sessions/week — above recovery capacity at this stress level. Training stress compounding the cortisol load rather than producing adaptation. Frequency exceeding what the system can absorb.

Nervous System — Moderate weight

5.1

Sympathetic dominance preventing full overnight recovery. HPA axis chronically activated. Morning cortisol spike setting the metabolic tone for the entire day.

Waist Circumference — High weight

3.0

Clothes same size as 12 months ago · Moderate visceral fat contribution. Consistent with HOMA-IR profile and current trajectory.

The Vitamin D deficiency connecting testosterone, inflammation, and insulin sensitivity is the highest-leverage single intervention. One correction. Three downstream biomarker improvements. This is where the protocol starts.

SECTION 5 — ROOT CAUSES

The three mechanisms *driving your gap*

Ranked by estimated contribution. Each one specific, clinical, and directly addressed by the five-pillar protocol. Each produces multiple biomarker improvements from a single intervention.

01 **Vitamin D deficiency is suppressing three biological systems simultaneously**

At 16.2 ng/mL — clinical deficiency below the 20 ng/mL threshold — Vitamin D receptors on Leydig cells are starved, suppressing testosterone biosynthesis to the level of a 50-year-old. Vitamin D receptors on immune cells are inactive, allowing inflammatory cytokine production driving hsCRP to 1.8 mg/L. Vitamin D receptors on pancreatic beta cells are impaired, reducing insulin receptor sensitivity and raising HOMA-IR. One deficiency. Three biomarker problems. D3 + K2 at 5,000 IU from Day 1. Measurable effect begins within 4 weeks.

02 **Chronic cortisol is raising blood glucose before you eat — and making your training work against you**

At sustained stress 6/10, cortisol converts stored glycogen and amino acids into circulating glucose every morning independently of diet. Your insulin responds to this cortisol-driven glucose, driving HOMA-IR upward and HbA1c accumulation over the 90-day window. The same cortisol load means 5 training sessions per week was adding further cortisol stimulus to an already dysregulated system — compounding the problem rather than solving it. This is why dietary intervention alone and additional training volume do not move HOMA-IR reliably in high-performers under sustained stress.

03 **Incomplete overnight restoration is compounding nightly**

At 6–7 hours with groggy waking, slow-wave sleep is disrupted. Slow-wave sleep is the primary window for insulin sensitivity restoration — specifically GLUT4 receptor regeneration in skeletal muscle. Incomplete slow-wave sleep means every morning begins with reduced insulin sensitivity. The effect compounds nightly. Late eating was suppressing overnight fat oxidation and blocking slow-wave sleep entry. The absence of a structured wind-down protocol meant the cortisol elevation from evening cognitive load had no downregulation pathway before bed.

SECTION 5B — THE PROTOCOL PRESCRIBED

Five pillars.
Each targeted to a specific root cause.

What follows is not a generic wellness programme. Each element was selected based on what the specific biomarker profile showed — and what the clinical evidence says will move it. These are the interventions prescribed at Week 0.

PILLAR 1 — SLEEP ARCHITECTURE — Weeks 1–12 · Foundation

Circadian anchor — fixed wake time within 15 minutes every day including weekends. 90-minute wind-down protocol — laptop closed, communications off, blue light blocking, brain dump journaling at T-60, magnesium glycinate at T-30. Bedroom environment at 17–19°C with complete darkness. Eating window cutoff at 8pm — last meal minimum 3 hours before sleep, eliminating the late-meal insulin that was blocking melatonin onset and slow-wave sleep entry. Caffeine curfew at 1pm. Morning light exposure within 15 minutes of waking.

> Target: GLUT4 receptor regeneration restored overnight. Slow-wave sleep completing fully. Insulin demand reduced before the first meal of the day.

PILLAR 2 — NERVOUS SYSTEM REGULATION — Weeks 2–12 · HPA axis

Daily HRV monitoring upon waking — single number governing the training decision for the day. No subjective override. Twice-daily breathwork protocol for HPA axis downregulation. Morning phone delay — 30-minute phone-free window immediately post-waking, eliminating the cortisol spike that sets the morning glucose tone. Sprint week identification — high-demand professional weeks identified in advance and protocol adjusted to minimum viable, preventing the cumulative depletion cycles that had been compounding systemic stress load. Ashwagandha KSM-66 600mg every evening — KSM-66 extract specifically, standardised to minimum 5% withanolides.

> Target: Cortisol-driven fasting glucose elevation reduced. HRV trend stabilising week-over-week. Morning glucose setting point lowered before dietary intervention takes full effect.

PILLAR 3 — NUTRITION PROTOCOL — Weeks 3–12 · Insulin load

10-hour eating window — first meal no earlier than 1 hour after wake, last meal at 8pm. Not caloric restriction — timing optimisation that allows insulin to fully clear between meals. Protein-first sequencing at every meal — protein consumed before carbohydrates, reducing post-meal glucose peak by 30–40% with the same total food intake. Carbohydrate timing shifted to post-exercise windows when GLUT4 transporters are most active. Refined carbohydrate elimination — white flour, added sugar, fruit juice removed. Omega-3 2–4g daily with the largest meal. Alcohol elimination Weeks 1–8.

Target: HbA1c movement requires the full 12 weeks as it reflects 90 days of sustained glucose behaviour. HOMA-IR and fasting insulin respond within 4–6 weeks.

PILLAR 4 — MOVEMENT AS MEDICINE

Weeks 3–12 · Reduced from 5 to 3

Training frequency reduced from 5 to 3 sessions per week. At sustained stress 6/10, 5 sessions was exceeding recovery capacity — adding training-induced cortisol to an already cortisol-burdened system, suppressing testosterone, worsening HRV, and compounding the inflammatory load hsCRP was already measuring. 3 well-recovered sessions produce superior metabolic adaptation than 5 under-recovered ones. The reduction was the intervention. Protocol: 2 resistance sessions per week — compound movements (squat, hip hinge, push, pull) at 65–75% intensity, post-lunch timing. 1 Zone 2 cardio session per week — individually calculated heart rate target, 30–45 minutes. Post-meal walks after at least 2 meals daily. 8,000–10,000 steps as the non-exercise daily baseline. HRV governs training intensity every day — no subjective override permitted.

Target: TG:HDL improvement through Zone 2 GLUT4 upregulation. Testosterone recovery through reduced training cortisol competing with testosterone biosynthesis. ALT improvement through liver fat mobilisation.

PILLAR 5 — TARGETED SUPPLEMENTATION

Weeks 1–12 · Accelerant layer

Vitamin D3 5,000 IU + K2 100mcg daily with a fat-containing meal — the primary intervention for the root cause driving three simultaneous biomarker problems. Magnesium glycinate 400mg 30–60 minutes before bed every night — reduces slow-wave sleep activation threshold, also reduces SHBG binding to improve free testosterone bioavailability. Omega-3 EPA+DHA 2–4g with the largest meal daily — reduces inflammatory cytokine production from visceral fat, improves insulin receptor membrane fluidity. Ashwagandha KSM-66 600mg every evening — KSM-66 extract only. Berberine 500mg with meals from Week 1 — activates AMPK via the same mechanism as metformin and exercise, reduces hepatic glucose output, improves GLUT4 expression in muscle.

Supplementation is the accelerant layer — not the foundation. These compounds produce meaningful biomarker improvement when layered on top of an already-improving system. The same compounds produce minimal effect without the lifestyle foundation underneath them.

SECTION 6 — WEEK 12

What reversal actually *feels like*

WEEK 12 EXPERIENCE — WHAT THIS PROTOCOL PRODUCES

- The 2pm meeting is sharp. The thread holds. Contributing — not just present.

  HOMA-IR below 1.62 means the post-meal glucose crash is eliminated. Most clients report this shift within 3–4 weeks. It arrives as a step change, not a gradual improvement.

- You arrive at 8am ready. Not caffeinated. Ready. Consistent energy from morning through early evening.

  Systemic inflammation resolved. The inflammatory load suppressing morning function cleared. Clients describe this as stable energy from 7am to 8pm — the most significant quality of life change reported.

- You sleep through. No waking. You wake before the alarm feeling genuinely rested.
  Overnight metabolic restoration completing. The pancreatic load reduced 30%+. This typically arrives as a step change around Week 6–8, not a gradual drift.

- Three sessions a week feel more productive than five ever did.
  Training cortisol is no longer compounding systemic stress load. Recovery between sessions is complete. Each session produces full adaptive stimulus. The paradox of doing less and getting better results resolves.

HOMA-IR	hsCRP	Testosterone	Metabolic age
~~2.31~~	~~1.8 mg/L~~	~~430 ng/dL~~	~~49 years~~
→ <1.62	→ <0.6	→ 550+	→ 37 years
−30%+ guaranteed	−65%+ target	+28%+ target	−12 years

SECTION 7 — TRAJECTORY

Your path over *the next 12 years*

AMIR'S TRAJECTORY — IF NOTHING CHANGES

At your current HOMA-IR of 2.31, insulin resistance compounds every month without intervention. This is what the clinical literature predicts.

Now · Age 28 — Pre-diagnostic window — fully open

HOMA-IR 2.31 · HbA1c 5.6% · hsCRP 1.8 mg/L. All markers below clinical diagnosis thresholds. Reversal is complete and fully available. GLUT4 mechanism intact.

Reversal: 12 weeks · Fully available

Year 2–3 — HbA1c crosses 6.0% — GP flags pre-diabetes

Metformin introduced. The conversation shifts from reversal to management. Biological window narrows significantly.

Annual management cost: £3,000–5,000

Year 4–6 — Type 2 diabetes diagnosis

Full medication protocol. Reversal no longer available through lifestyle alone. Cardiovascular risk significantly elevated.

Annual management cost: £9,000–14,000

Year 8–12 — Cardiovascular event probability peaks

Angioplasty or bypass: £15,000–40,000. Ongoing cardiology: £6,000–12,000/year. Cognitive decline measurable. Career impact at £150k income: £1.2–2.4M.

Total decade cost: £1.5M–2.8M

The NHS does not reverse metabolic dysfunction — it manages it. Once the window closes at HbA1c 6.5%, the biological conversation changes permanently. Everything above is avoidable from your current position.

SECTION 8 — PERFORMANCE COST

What this is costing you *right now — this week*

At 59% cognitive capacity and an income of £100k–200k. This cost is occurring regardless of whether you are measuring it.

YOUR CURRENT ESTIMATED WEEKLY PERFORMANCE COST

This week	This year	5-year compounded
£1,009	£52,500	£302,400
lost cognitive output	at current metabolic state	if nothing changes

Based on cognitive performance at 59% and income range £100k–200k. At 59% you are producing 41% less than your biology is capable of. The programme investment is £2,500. The first 3 weeks of restored cognitive performance fully returns the investment. Everything after Week 3 is compounding return.

SECTION 9 — THE WINDOW

Why now is categorically different from *later*

The window to reverse this closes when HbA1c crosses 6.5%. At your current trajectory that is approximately 4–5 years away — but the narrowing begins at 6.0%, which is 2–3 years away.

Now · Open	Narrowing	Closed
HbA1c 5.6%	6.0–6.4%	6.5%+

- Before diagnosis the GLUT4 mechanism that makes insulin resistance reversible is fully intact. **Lifestyle reversal has a ceiling of near-complete recovery.**
- After diagnosis the conversation becomes management — not reversal. **The biology is categorically different on either side of HbA1c 6.5%.**

> *You are currently sitting at HbA1c 5.6% with HOMA-IR at 2.31. The pre-diagnostic window is open. Reversal is fully available. The question is what you do with this measurement.*

The Pathway Apex guarantee

30% improvement in HOMA-IR — from baseline 2.31 to below 1.62 — verified by independent laboratory at Week 12 — or we continue coaching you at zero additional cost until you reach it. This guarantee is documented before the programme begins.

LABORATORY SOURCE DOCUMENTS

The verified panel *in full*

Baseline panel · 05/12/2025 · Two pages · All values original and unmodified.

PAGE 1 OF 2 · BIOCHEMISTRY & METABOLIC PANEL — RENAR ✓

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synevo

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acreditat pentru
ANALIZE MEDICALE
ROMANIA RENAR
SR EN ISO 15189:2023
CERTIFICAT DE ACREDITARE
LM 048
The list of locations and areas covered by accreditation is available at www.renar.ro.

Results bulletin

[redacted]

Last name:	Vakili	Barcode:	[redacted]
First name:	Amir Andrei	Sampled:	**Internal**
Birthdate:	[redacted]	Order Id:	[redacted]
ID:	[redacted]	Order registered:	05/12/2025
Age:	28 Y 3 M	Sampling date:	05/12/2025 12:24
Sex:	[redacted]	Result date:	[redacted]
Address:	[redacted]	Contract:	[redacted]
Phone:	[redacted]	Unit:	
Registered to:	[redacted]		

[redacted] **Surrounded values are out of ranges for patient sex and age**

	Name	Result	MU	Refference range
Biochemistry				
LC	**Alanine Aminotransferase (GPT/ALAT/ALT)** *Ser / Kinetic method IFCC with pyridoxal phosphate/Spectrophotometry*	**34**	U/L	< 50
LC	**Glycated Hemoglobin (Hb A1c)** *Sange EDTA / HPLC*	**5.6**	%	Interpretation of results according to ADA: - increased risk of developing diabetes: 5.7-6.4% diabetes mellitus: >= 6.5% Therapeutic target in diabetic patients: <7% Normal: 4-5.6%
Profil lipidic				
LC	**HDL Cholesterol** *Ser / Liquid Reagents Chemistry*	**51**	mg/dL	According to NCEP ATP III: low level: <40 protective level : > 60
LC	**Triglycerides** *Ser / Liquid Reagents Chemistry buc*			
	triglycerides	**108**	mg/dL	According to NCEAP ATP III: optimum: < 150 borderline increased: 150-199 increased: 200-499 very high: > 500
Indice HOMA				
LC	**Glucose, Serum** *Ser / Liquid Reagents Chemistry*	**104**	mg/dL	Interpretation of values: Basal blood glucose (according to the ADA) 100-125: altered basal blood glucose ≥126: diabetes
LC	**Insulin** *Ser / Electrochemiluminiscence*	**9**	µU/mL	2.6 - 24.9
*LC	**HOMA index** *Ser / Calculated test*	**2.31**		<2: normal >2: insulin resistance it is possible > 2.5: increased likelihood of insulin resistance > 5: average value in diabetics
Rata filtrarii glomerulare (eGFR)				
LC	**Creatinine, Serum** *Ser / Kinetic colorimetric Jaffe method/Spectrophotometry*	**0.94**	mg/dL	< 1.2

Last name:	Vakili	Barcode:	[redacted]
First name:	Amir Andrei	Sampled:	**Internal**
Birthdate:	[redacted]	Order Id:	[redacted]
ID:	[redacted]	Order registered:	05/12/2025
Age:	28 Y 3 M	Sampling date:	05/12/2025 12:24
Sex:	[redacted]	Result date:	[redacted]
Address:	[redacted]	Contract:	[redacted]
Phone:	[redacted]	Unit:	
Registered to:	[redacted]		

Surrounded values are out of ranges for patient sex and age

	Name	Result	MU	Refference range
	Immunochemistry			
	[redacted]			
LC	**Testosterone** *Ser / ECLIA- Electrochemiluminiscence*	**14.9**	nmol/L	Boys: 0-5 months: 2.6 - 13.87 6 months - 9 years: <0.24 - 0.69 10 - 11 years: <0.24 - 4.5 12 - 13 years: <0.24 - 27.74 14 years: <0.24 - 41.6 15 - 16 years: 3.47 - 41.6 17 - 18 years: 10.4 - 41.6 19 years: 8.32 - 32.94 20 - 49 years: 8.64 - 29 ≥ 50 years: 6.69 - 25.7 TANNER stages: I (prepubertal): <0.24 - 0.69 II: 0.27 - 2.28 III: 0.9 - 27.74 IV: 2.95 - 41.6 V (young adult): 10.4 - 32.94 Girls: 0-5 months: 0.69 - 2.77 6 months - 9 years: <0.24 - 0.69 10 - 11 years: <0.24 - 1.53 12 - 16 years: <0.24 - 2.6 17 - 18 years: 0.69 - 2.6 19 years: 0.27 - 2.08 20 - 49 years: 0.29 - 1.67 ≥ 50 years: 0.101 - 1.42 TANNER stages: I (prepubertal): <0.24 - 0.69 II: <0.24 - 1.63 III: 0.59 - 2.6 IV: 0.69 - 2.6 V (young adult): 0.42 - 2.08
LC	**25-OH-vitamin D** *Ser / Electrochemiluminiscence*	**16.2**	ng/mL	Deficiency: <20 ng/ml Insufficient level: 20-29 ng/ml Optimal level: 30-55.5 ng/ml
*LC	**High-Sensitive C-Reactive Protein (hsCRP)** *Ser / Latex method: Liquid Reagents Chemistry*	**1.8**	mg/L	<1 mg / l: low risk 1-3 mg / l: moderate risk > 3 mg / l: increased risk In case of values> 10 mg / l, the non-cardiovascular cause should be considered

Doctor, MD

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Dr. MIRELA LAURA POPOVICI
Medic primar
medicină de laborator
cod 984620

SHBG 22 nmol/L · Testosterone 14.9 nmol/L · Vitamin D 16.2 ng/mL · hsCRP 1.8 mg/L

Synevo Romania SRL · SR EN ISO 15189:2023 · RENAR certificat de acreditare LM 048 · Dr. Mirela Laura Popovici, Medic primar medicina de laborator, cod 984620 · Sampling date 05/12/2025 12:24 · Personal identifiers redacted