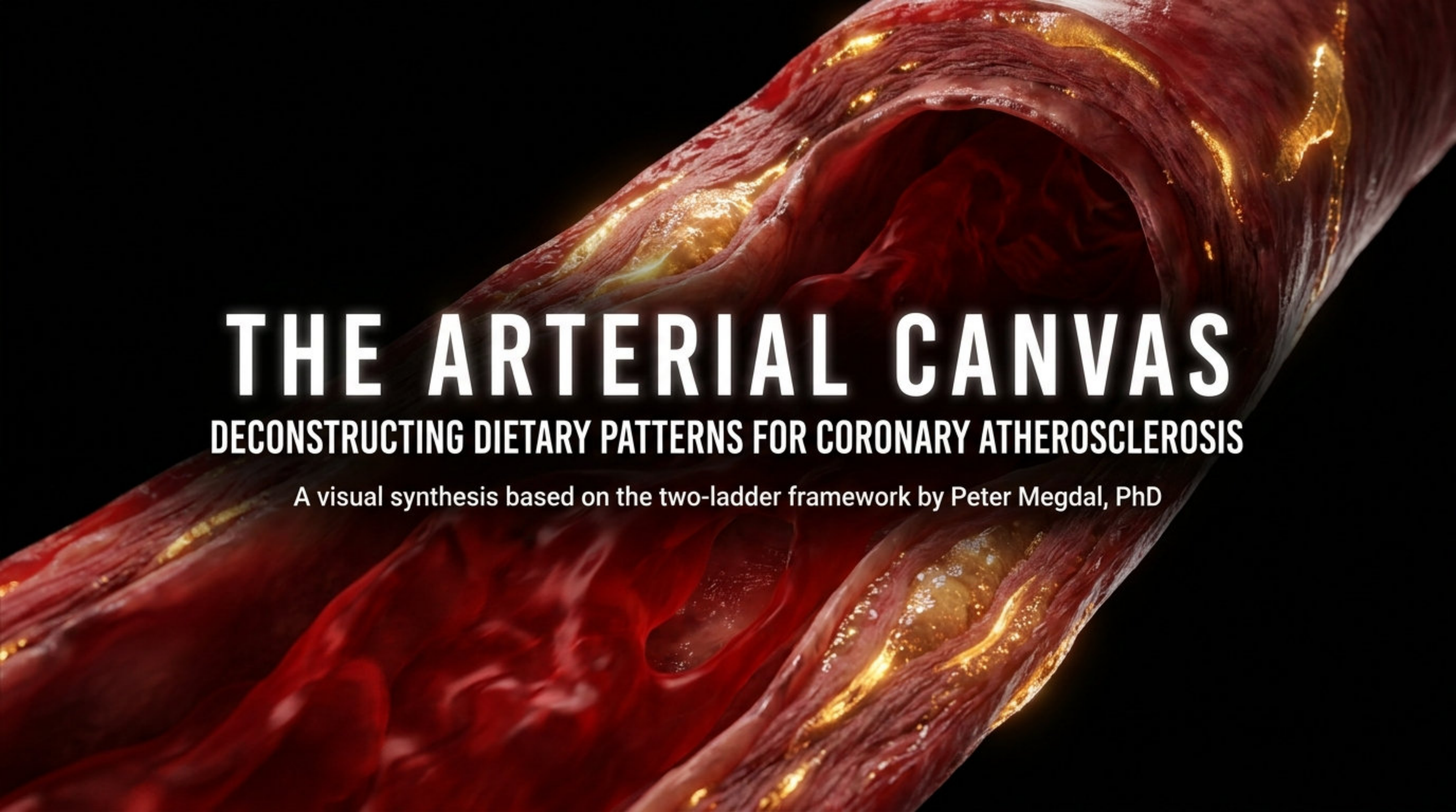

THE ARTERIAL CANVAS
DECONSTRUCTING DIETARY PATTERNS FOR CORONARY ATHEROSCLEROSIS
A visual synthesis based on the two-ladder framework by Peter Megdal, PhD

CORONARY DISEASE IS NOT INEVITABLE AGING; IT IS A LIFELONG EXPOSURE PROBLEM.

THE OLD FRAMING

Management was palliative—relieve angina, treat infarction, slow an inexorable decline.

THE NEW PARADIGM

Atherosclerosis is driven by cumulative exposure to Apolipoprotein B (ApoB). Because the process is dynamic, it is highly modifiable over a lifetime.

THE INITIATING CRIME: APOB RETENTION IN THE ARTERIAL INTIMA

THE PARTICLE

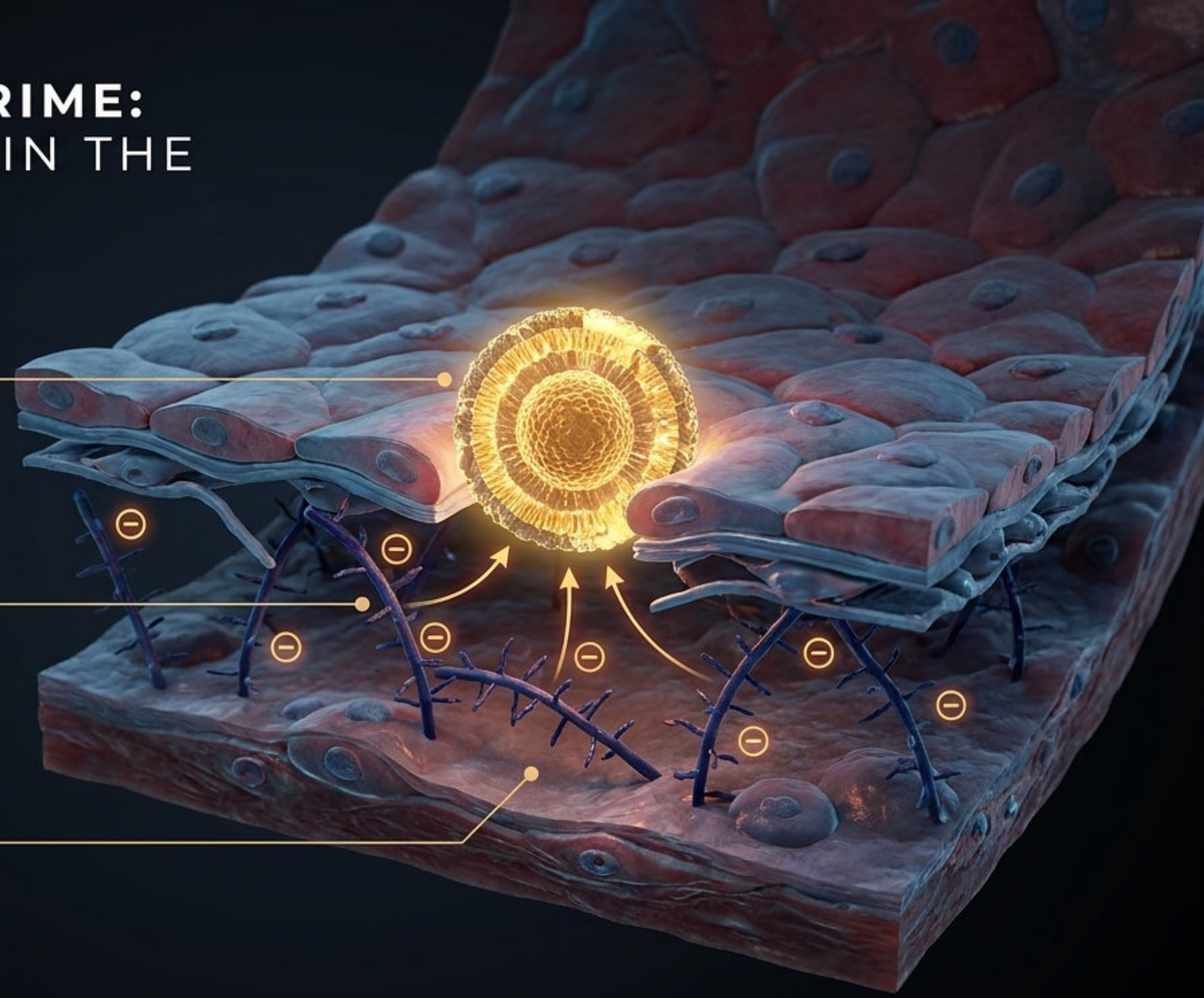
Plasma ApoB approximates the concentration of all circulating atherogenic lipoproteins (LDL, IDL, VLDL, Lp(a)).

THE RETENTION

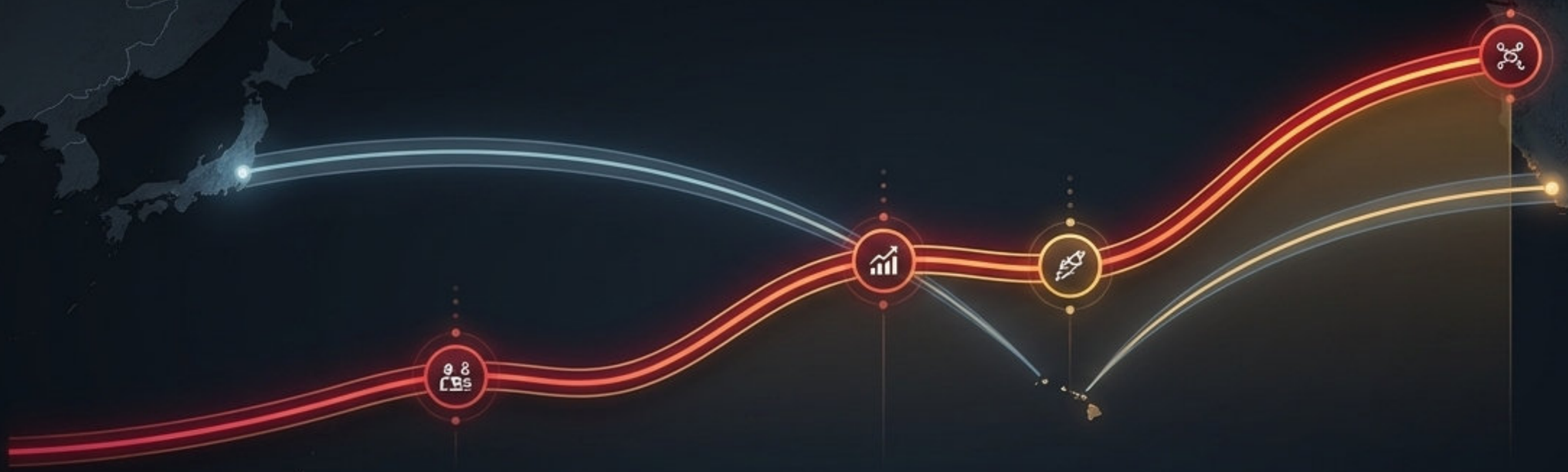
Atherosclerosis initiates when ApoB particles are trapped via electrostatic binding to negatively charged proteoglycans in the arterial wall.

THE CONSEQUENCE

Retained particles provoke inflammation and macrophage foam-cell formation, eventually seeding a dangerous, lipid-rich necrotic core.



MIGRANT STUDIES PROVE THE DISEASE TRACKS WITH DIETARY EXPOSURE, NOT JUST GENETICS



THE NI-HON-SAN EXPERIMENT

Followed ~11,900 men of Japanese ancestry. Genetics were held constant; environment and diet changed.

THE GRADIENT

Age-adjusted definite CHD prevalence doubled (from ~5.3 per 1,000 in Japan to 10.8 in California).

THE MECHANISM

The risk tracked perfectly with a parallel rise in serum cholesterol, dietary saturated fat, and westernization.

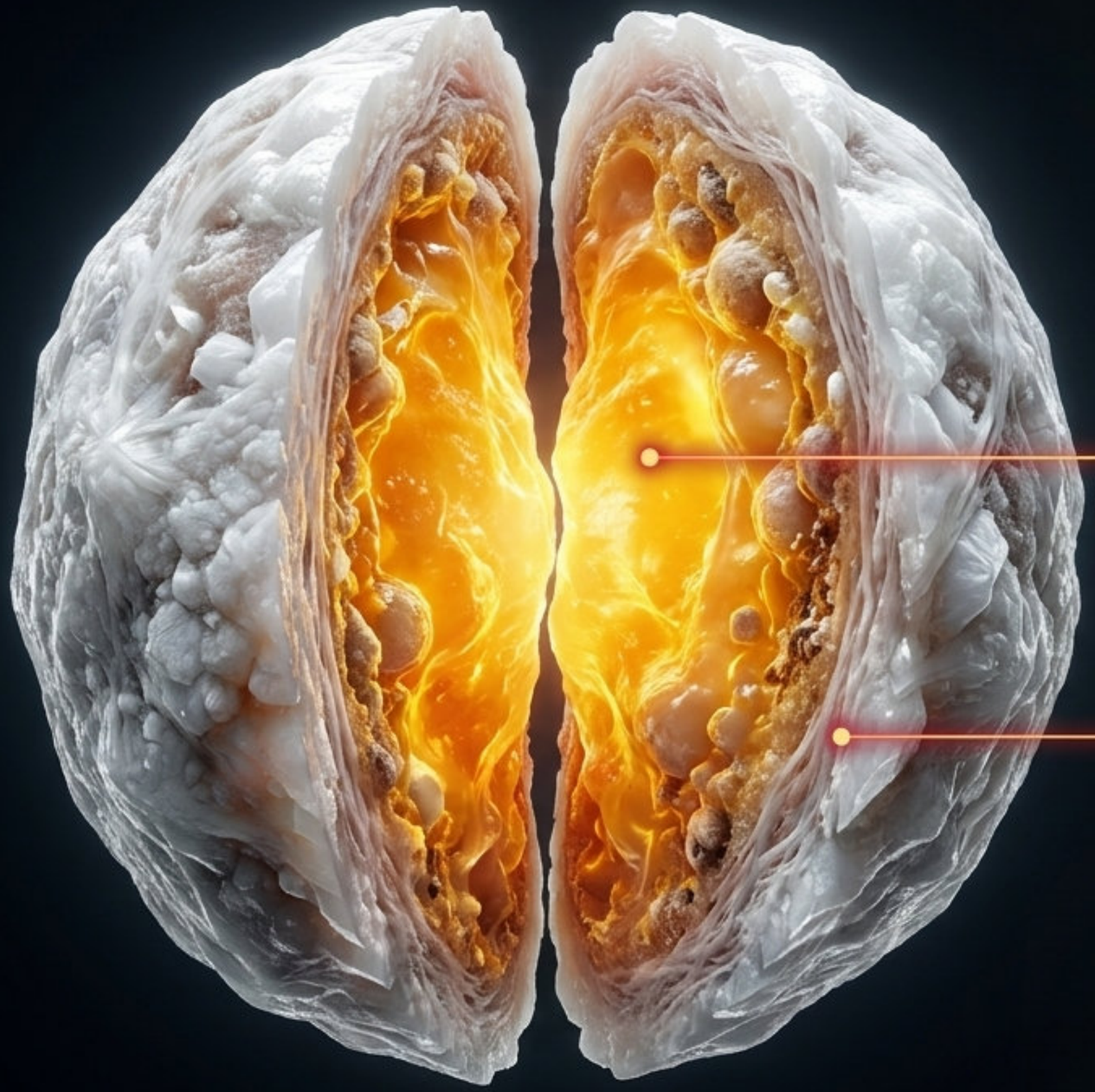
THE RED HERRING: WHY ANGIOGRAMS DECEIVE US ABOUT PLAQUE REGRESSION.

1. INITIAL STATE
Clean artery wall, wide central lumen.

2. OUTWARD REMODELING
Outward Remodeling: A massive, dangerous plaque burden hides behind a perfectly normal-looking lumen silhouette.

3. CONSTRICTIVE REMODELING
Constrictive Remodeling: A healing, de-lipidated plaque shows an unchanged or smaller lumen.

THE TAKEAWAY: LUMINAL STENOSIS IS A FLAWED ENDPOINT. WE MUST MEASURE PLAQUE COMPOSITION, NOT JUST THE CHANNEL.



The anatomy of reversal: Regressible lipids vs. stubborn scaffolds.

The Lipid-Rich Core (Regressible)

Foam cells and necrotic debris. This is the dangerous, rupture-prone compartment. Primate studies show normalizing cholesterol (lowering extreme highs to ~200 mg/dL) depletes this core rapidly.

The Fibrocalcific Scaffold (Stubborn)

Dense calcification and collagen. This is structural and slow to remodel. It persists even when the lipid leaves.

The Verdict: We do not want to just shrink the plaque; we want to drain the volatile lipid and stabilize the cap.

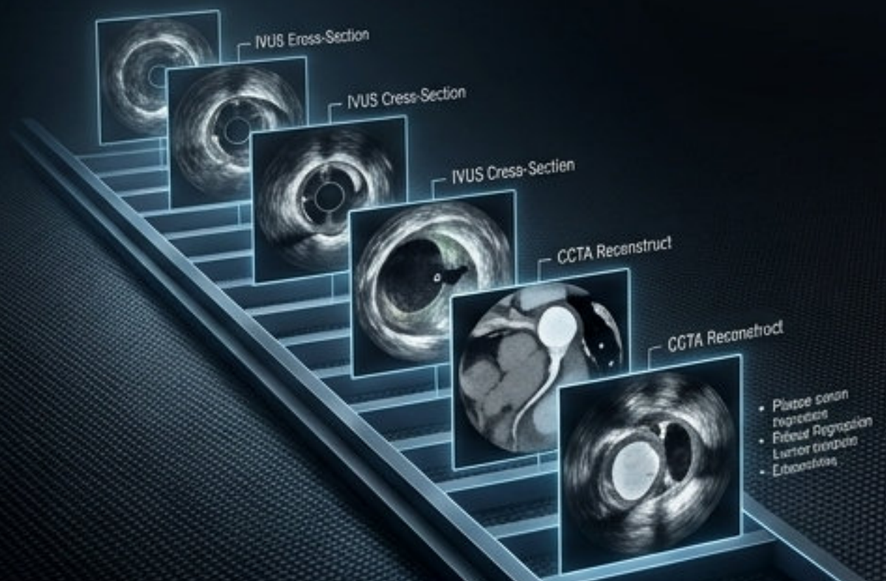
THE ‘TWO LADDERS’ FRAMEWORK: SEPARATING IMAGES FROM SURVIVAL

The Conflation Error: A diet that changes a surrogate imaging marker is not proven to prevent death.
A diet that prevents death has not necessarily shrunk a coronary lesion.

The Solution: We must rank diets on two independent ladders,
grading the certainty of evidence for each distinct outcome.

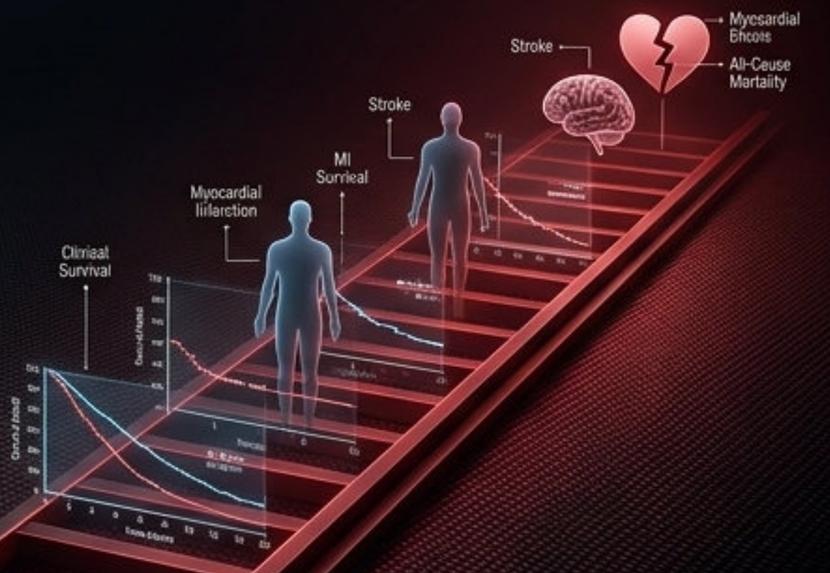
LADDER A (IMAGING)

Evidence for
Plaque Regression



LADDER B (HARD EVENTS)

Evidence for Hard Event Reduction
(MI, Stroke, Death)



LADDER A (IMAGING): THE DIETARY SIGNALS AND THEIR SEVERE LIMITATIONS.

ORNISH LIFESTYLE TRIAL (HISTORIC)

- Intensive very-low-fat vegetative diet + exercise + stress management.
- Showed QCA angiographic stenosis improvement.

FLAW: Lumen-based surrogate; diet inseparable from bundled lifestyle changes (Certainty: Very Low for diet alone).

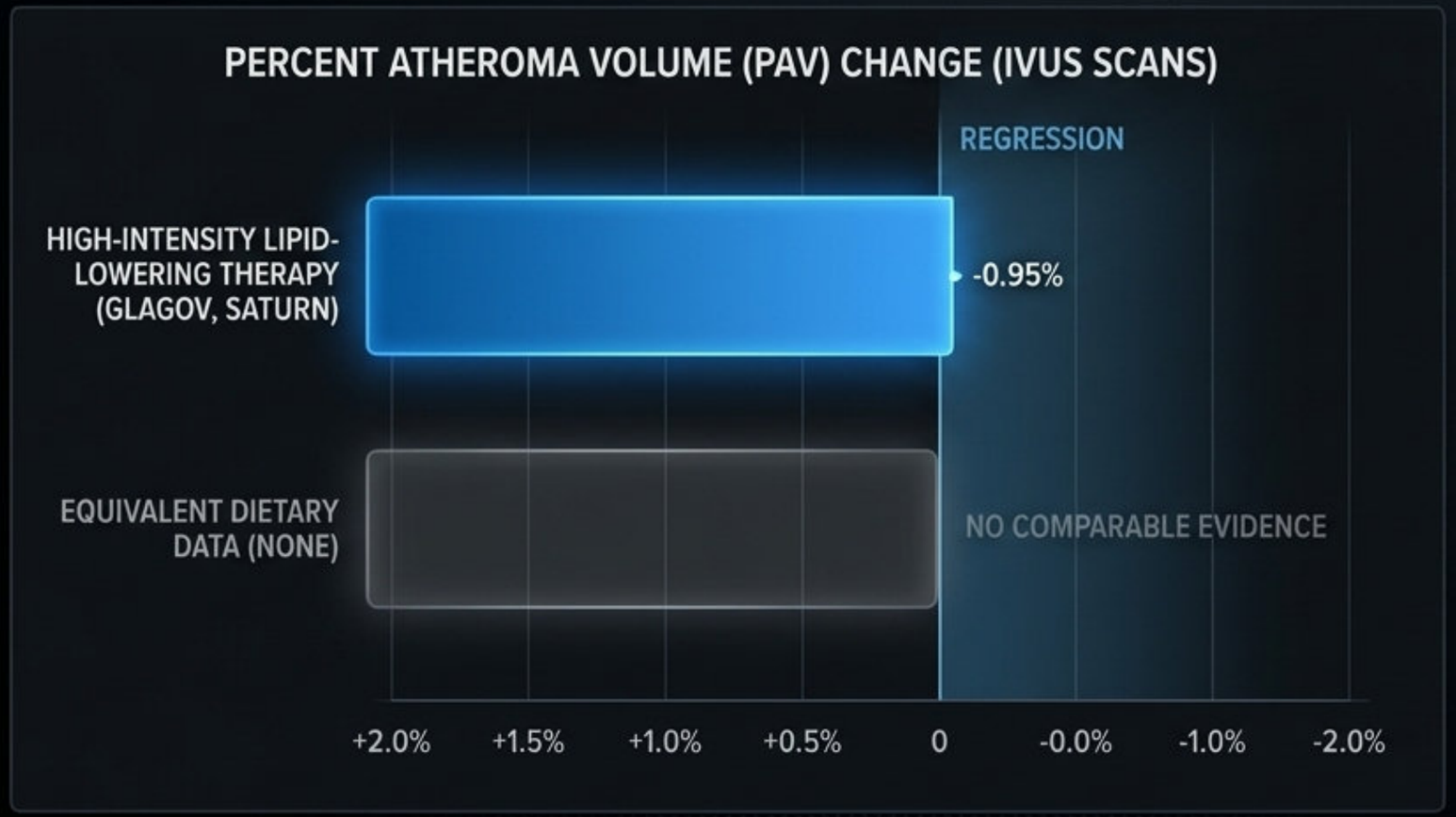
DISCO-CT (CONTEMPORARY)

- DASH diet + activity.
- Showed reduction in CCTA-measured noncalcified plaque volume (-51.3 mm^3 vs -21.3 mm^3).

FLAW: Modest sample (n=92), bundled intervention (Certainty: Low).

SUMMARY: DIETARY PLAQUE-REGRESSION EVIDENCE REMAINS VERY-LOW-TO-LOW CERTAINTY.

THE PHARMACOLOGIC BENCHMARK: THE STANDARD THAT DIETS MUST BE JUDGED AGAINST.



THE GOLD STANDARD

High-intensity lipid-lowering therapy (GLAGOV, SATURN trials) provides consistent, moderate-to-high certainty for coronary atheroma regression.

GLAGOV TRIAL

Adding evolocumab to a statin drove LDL-C to 36.6 mg/dL, causing regression in 64.3% of patients (PAV change of -0.95%).

THE REALITY CHECK

No dietary trial has produced coronary plaque regression matching the magnitude and rigor of high-intensity lipid-lowering therapy.

LADDER B (HARD EVENTS): THE MEDITERRANEAN HEAVYWEIGHT

The Track Record: The Mediterranean pattern carries the strongest randomized hard-event evidence among named diets (Low-to-Moderate certainty).

CORDIOPREV (SECONDARY PREVENTION)

7-year trial against low-fat diet. Reduced major adverse cardiovascular events by ~25-28%.

PREDIMED (PRIMARY PREVENTION)

Diet supplemented with EVOO or nuts reduced composite of MI, stroke, or CV death by ~28-30%.

Mechanism: Broad benefits across lipids, BP, endothelial function, and inflammation.

PLANT-BASED, PORTFOLIO, AND DASH: COHORT SIGNALS AND BLOOD PRESSURE DROPS.

PLANT-BASED & PORTFOLIO (COHORTS)

The Adventist Health Study-2 found the lowest mortality among pesco-vegetarians (HR 0.81). The Portfolio diet lowers LDL-C substantially and links to lower CV disease (HR ~0.86).

Caveat: Observational data subject to healthy-user bias.

DASH (FEEDING TRIALS)

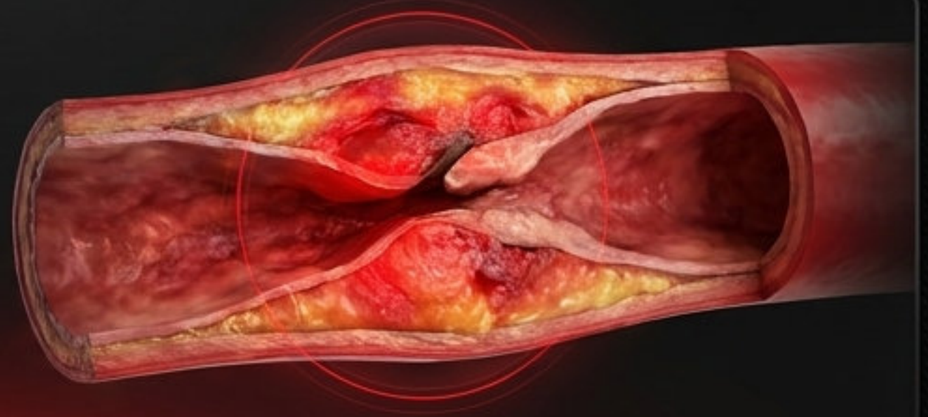
Unmatched for blood pressure control. Lowered BP by 11.4/5.5 mmHg in hypertensives.

THE SHARED THREAD: Food quality is decisive. An unhealthful plant-based diet (refined grains, sweets) increases risk (HR 1.32).

The Failures and the Unproven: Generic low-fat and Ketogenic patterns.

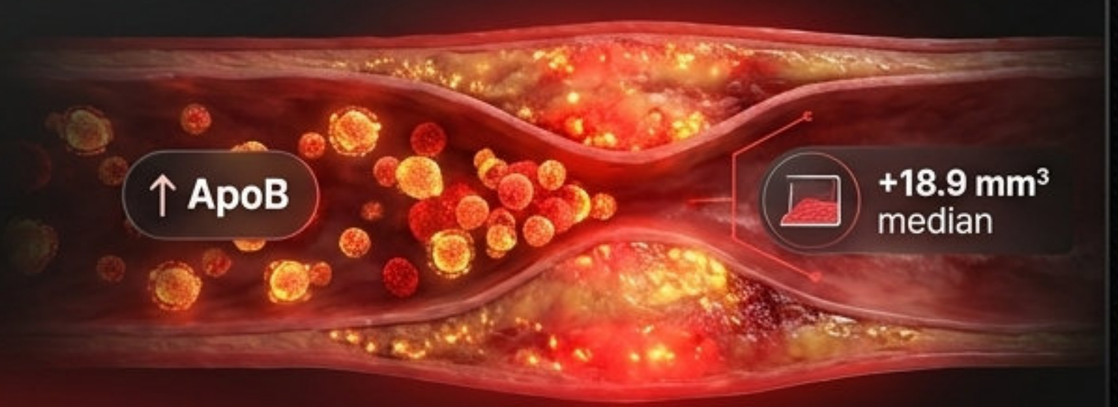
Generic Low-Fat (The Failure)

The massive Women's Health Initiative (WHI) trial (~49,000 women, 8 years) found that a generic low-fat prescription did not significantly reduce coronary heart disease or stroke.



Ketogenic (The Unproven/Risk)

In lean-mass hyper-responders, ApoB spikes aggressively. The KETO-CTA cohort signaled an increase in noncalcified plaque volume (+18.9 mm^3 median). Marked ApoB elevation is inconsistent with evidence-based lipid management.



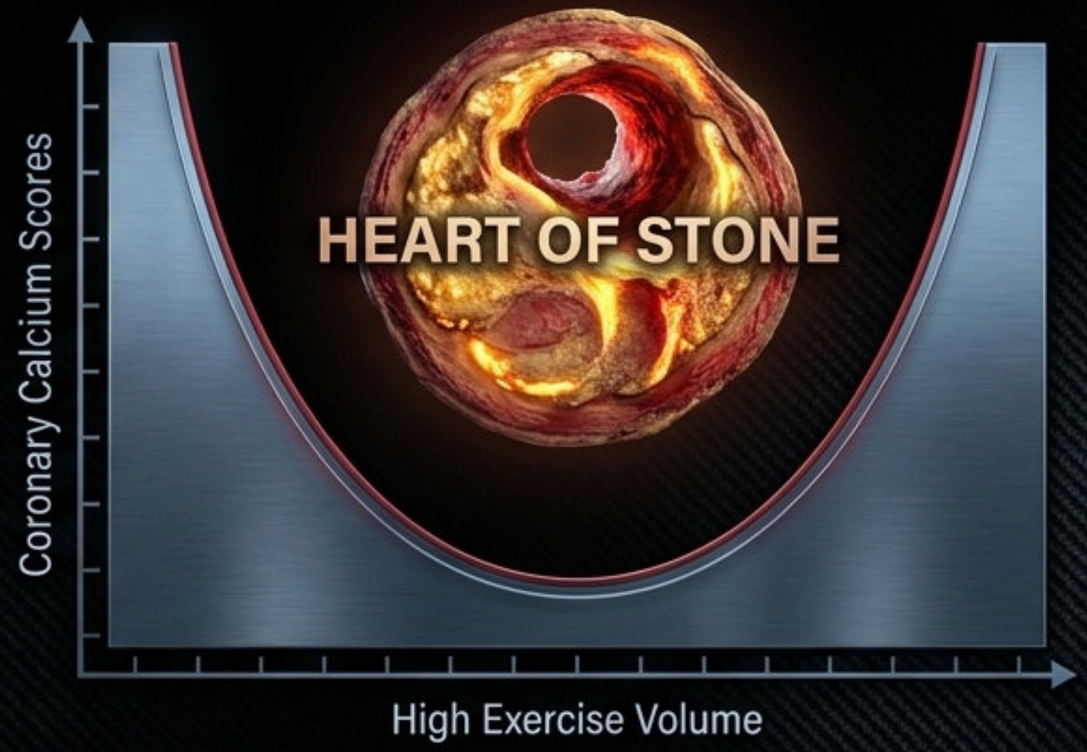
THE DIETARY COMPARISON MATRIX:
INTEGRATING BOTH LADDERS.

DIET	LADDER A: IMAGING	LADDER B: EVENTS	ROLE
Mediterranean-style	Favorable carotid; coronary limited	Strongest RCT evidence: Low-Mod	Broadest evidence base
Very-low-fat (Ornish)	Historical angiographic signal: Very Low diet alone Very Low	Suggestive / confounded	Historical proof-of-concept
Fully Plant-Based (WFPB)	No direct trial	Cohort support only	Therapeutic option
DASH	Coronary NCP drop in DISCO-CT: Low Low	Strong BP proxy	BP-focused building block
Drugs (Benchmark)	Mod-High	High	The gold standard

The Athlete Paradox: Why more calcium can mean more stability

THE PARADOX

Lifelong high-volume endurance training is associated with higher coronary-artery calcium scores than matched controls.



THE MORPHOLOGY

The athletes' plaques are predominantly calcified, not lipid-rich. This creates hearts of stone—a denser, stabilized, rupture-resistant phenotype.

THE LESSON

Exercise modestly lowers ApoB but heavily drives plaque stabilization. Rising calcium in an athlete often reflects healing, proving again why calcium-only endpoints are poor guides to risk.

COMPLEMENTARY LEVERS: DIET AND DRUGS ARE NOT COMPETITORS.

DIET (THE LIFELONG BASELINE)

Addresses the atherogenic-lipoprotein burden continuously across a lifetime, preventing accumulation.

DRUGS (THE POTENT CORRECTOR)

Guideline lipid-lowering therapy provides the highest-certainty regression from mid-life onward when targets aren't met.

Early to Lifelong

Mid-life Onward

SYNTHESIS:

The best outcomes come from combining an ApoB-lowering dietary pattern with guideline pharmacotherapy.

EXERCISE (THE METABOLIC ENGINE)

Sustains a lean phenotype and drives plaque stabilization.

THE CONVERGENT CORE:
THE UNDENIABLE DIETARY VERDICT.

THE CONVERGENCE

Every winning pattern—whether Mediterranean, DASH, or Whole-Food Plant-Based—converges on the exact same core.

THE PRESCRIPTION

Shift the diet toward a plant-predominant, minimally processed pattern.

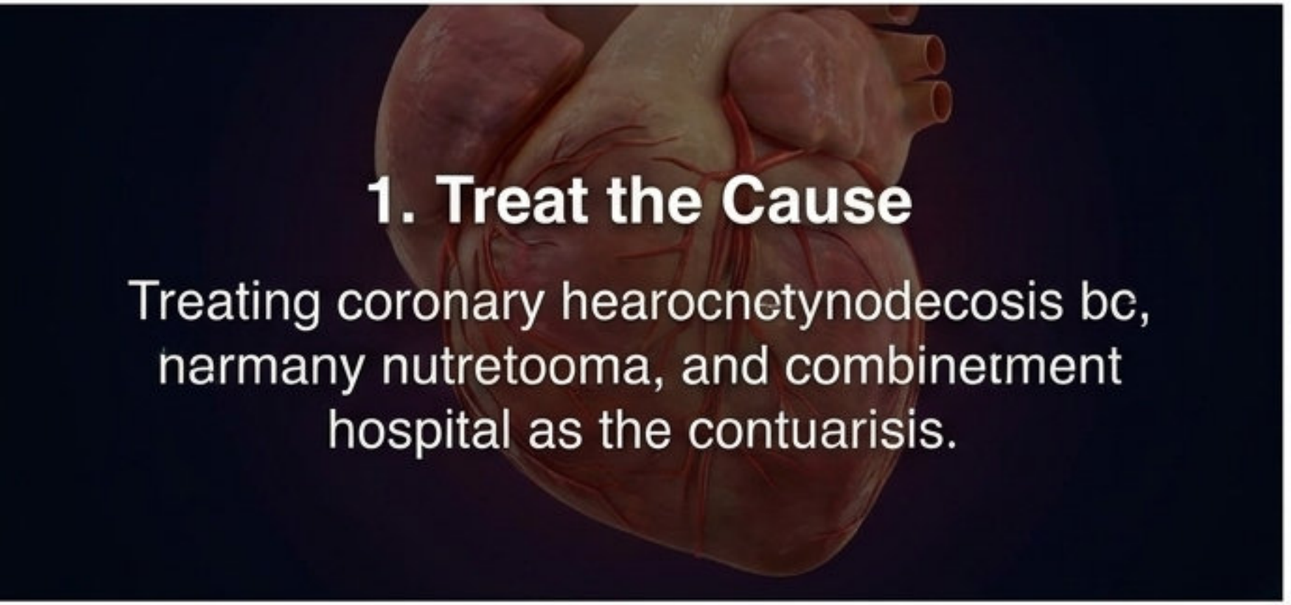
Include:
More vegetables, legumes, whole grains, nuts, and unsaturated fats.

Exclude:
Less red and processed meat, refined carbohydrate, and sodium excess.

THE REALITY

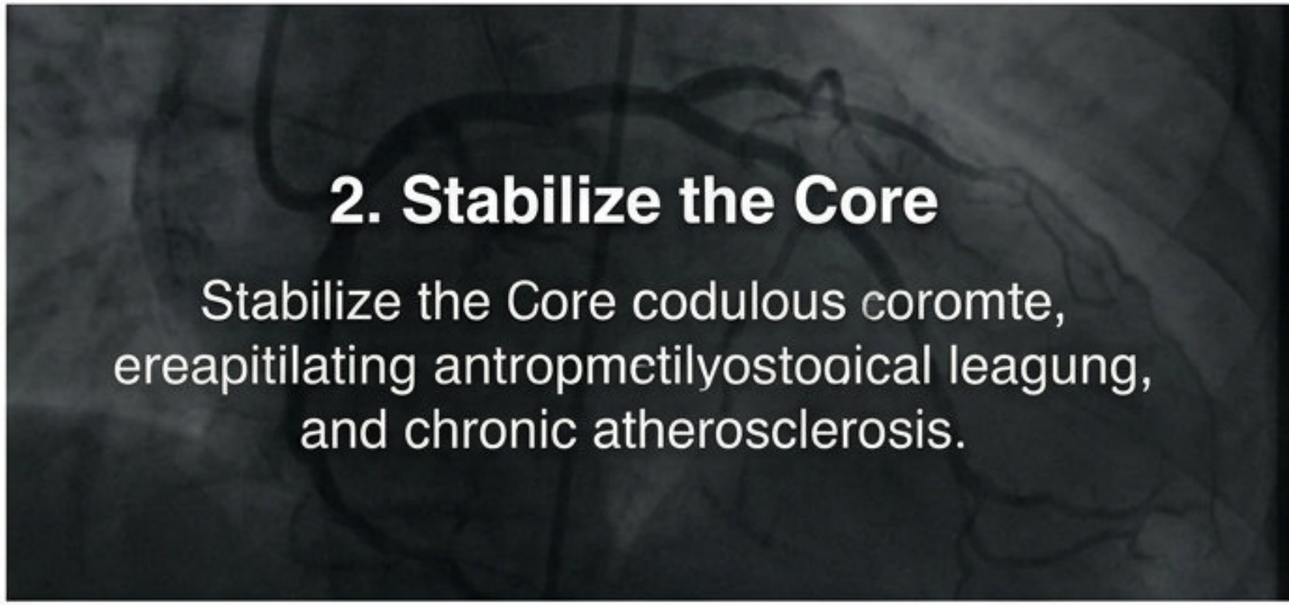
Food quality matters vastly more than the plant-versus-animal label alone.

Clinical Synthesis: The framework for treating coronary atherosclerosis.



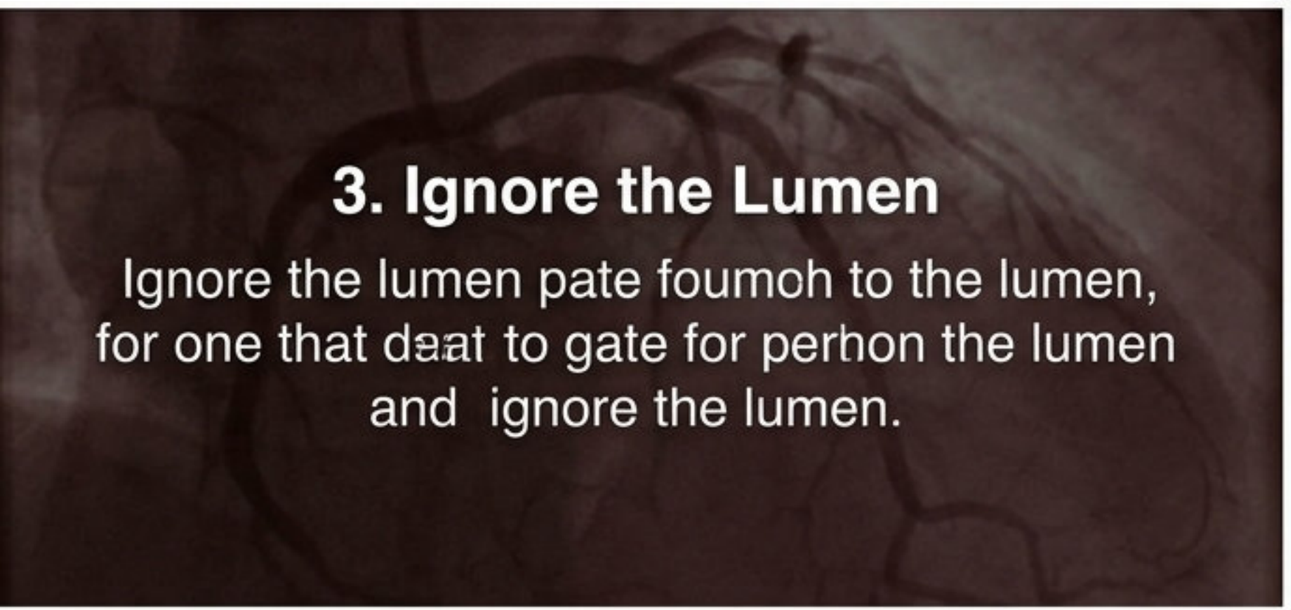
1. Treat the Cause

Treating coronary hearocnetynodecosis bc, narmany nutretooma, and combinetment hospital as the contuarisis.



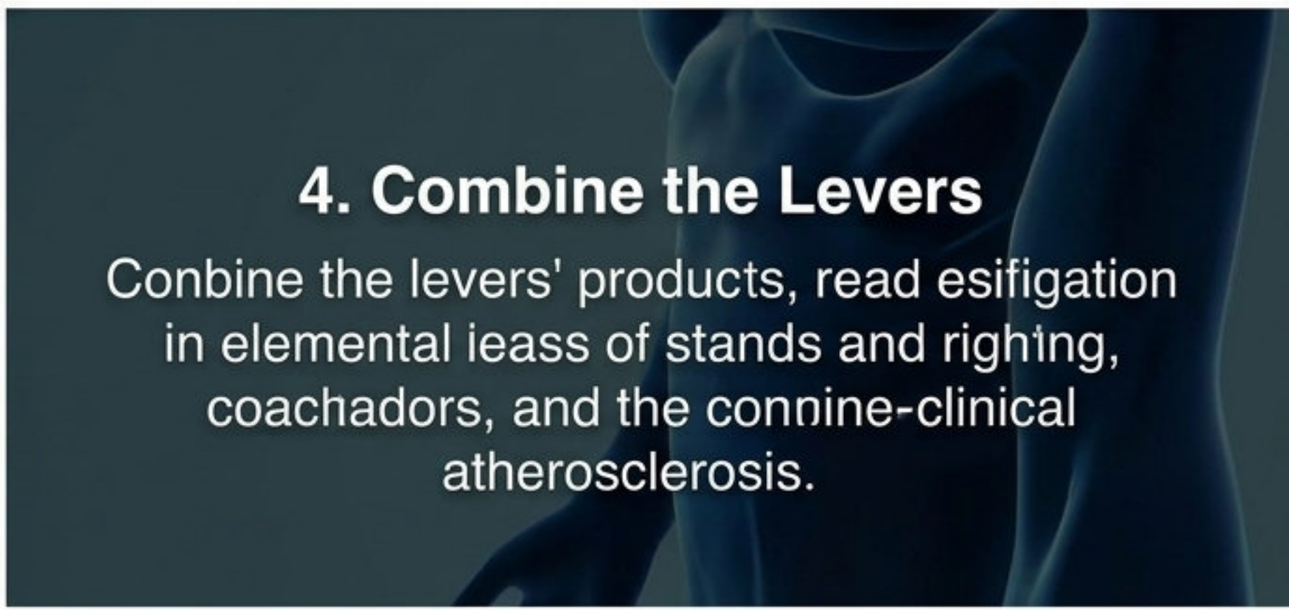
2. Stabilize the Core

Stabilize the Core codulous coromte, ereapitilating antropmetilyostooical leagung, and chronic atherosclerosis.



3. Ignore the Lumen

Ignore the lumen pate foumoh to the lumen, for one that daat to gate for perhon the lumen and ignore the lumen.



4. Combine the Levers

Conbine the levers' products, read esifigation in elemental ieass of stands and righing, coachadors, and the connine-clinical atherosclerosis.

Created with AI assistance. Reviewed by Peter Megdal, PhD.