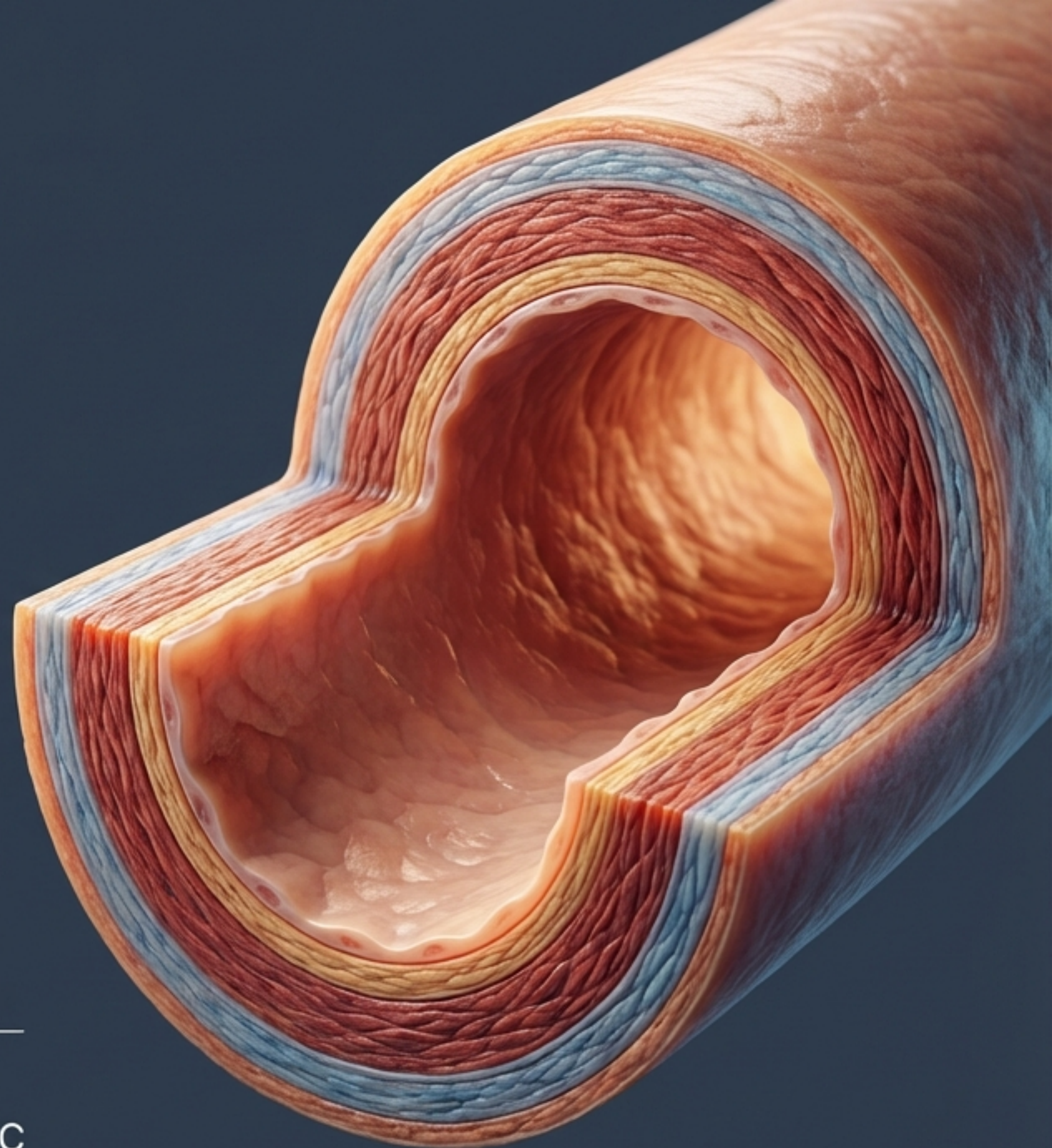


Reversing Coronary Artery Disease

*The Biological Case for the 10%
Ultra-Low-Fat Protocol*

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Challenging the Paradigm of Permanent Progression



Risk Management

Approach: Downstream Interventions

Tools: Statins, Percutaneous Coronary Intervention (PCI), Bypass Surgery (CABG)

Outcome: **Slowing** the expected **progression** of the disease.



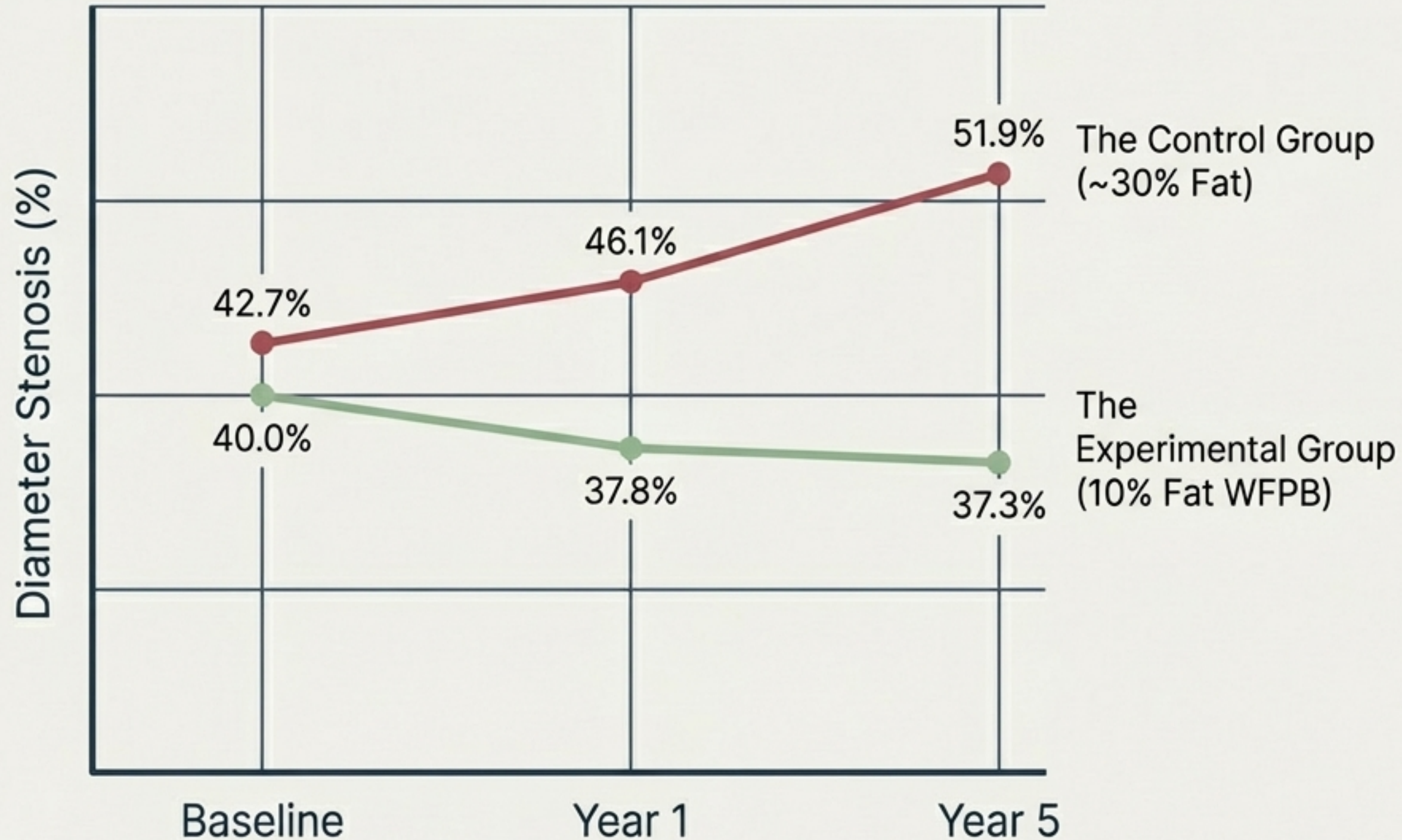
Disease Reversal

Approach: Upstream Cellular Drivers

Tools: Ultra-Low-Fat (10%), Whole-Food Plant-Based (WFPB) Nutrition

Outcome: **Halting** and **reversing** arterial plaque **formation**.

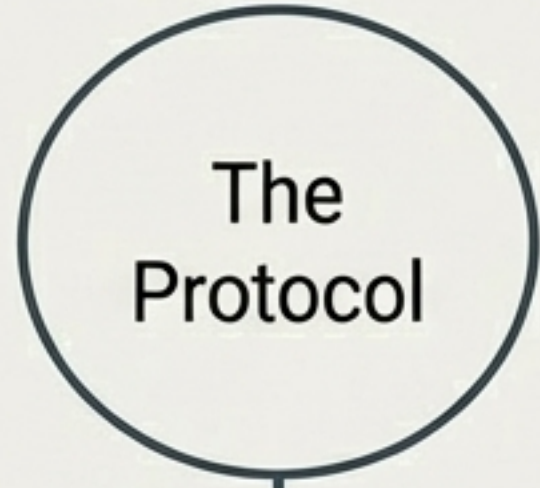
Moderate Changes Yield No Regression



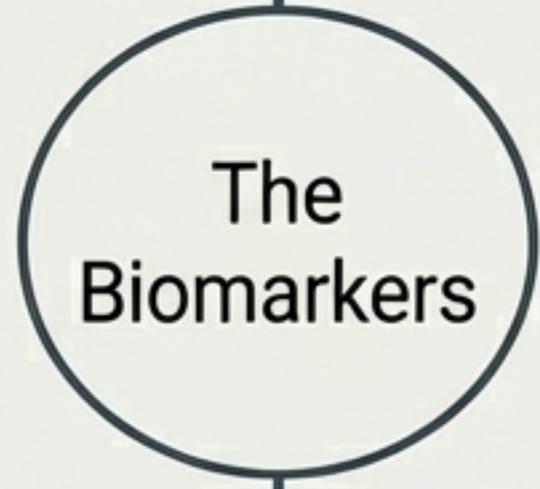
Baseline: Advanced CAD patients.

Key Takeaway: In advanced disease, standard dietary recommendations do not reverse stenosis.

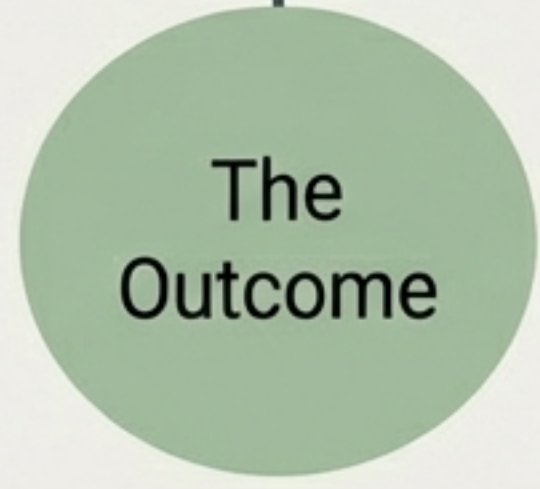
The 12-Year Longitudinal Reversal Protocol



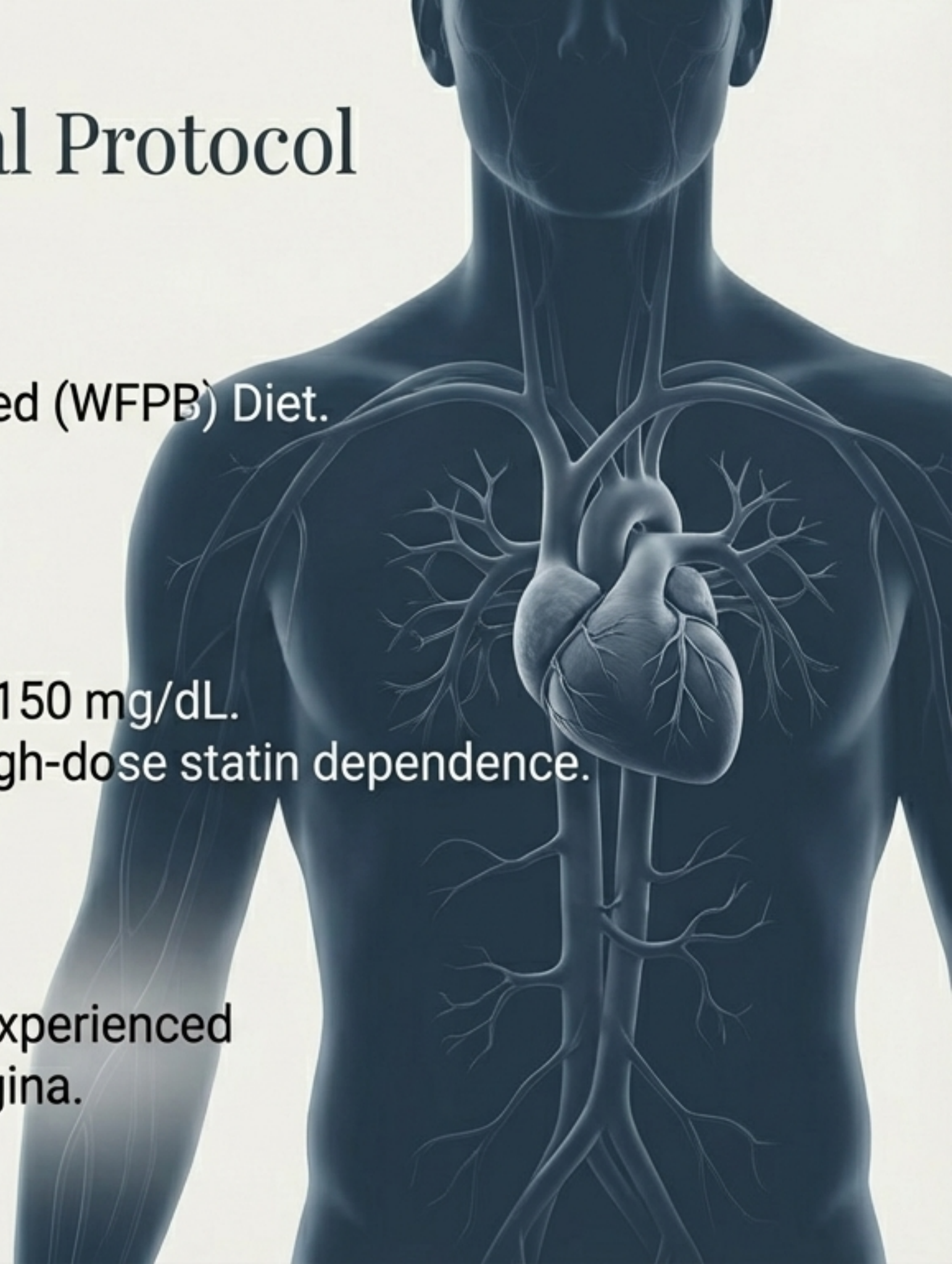
100% Oil-Free, Whole-Food Plant-Based (WFPB) Diet.



Total Serum Cholesterol maintained <150 mg/dL.
LDL maintained <80 mg/dL without high-dose statin dependence.



73% of adherent end-stage patients experienced **disease reversal** and cessation of angina.



The Lipid-Immune Interface and the Concentration Gradient

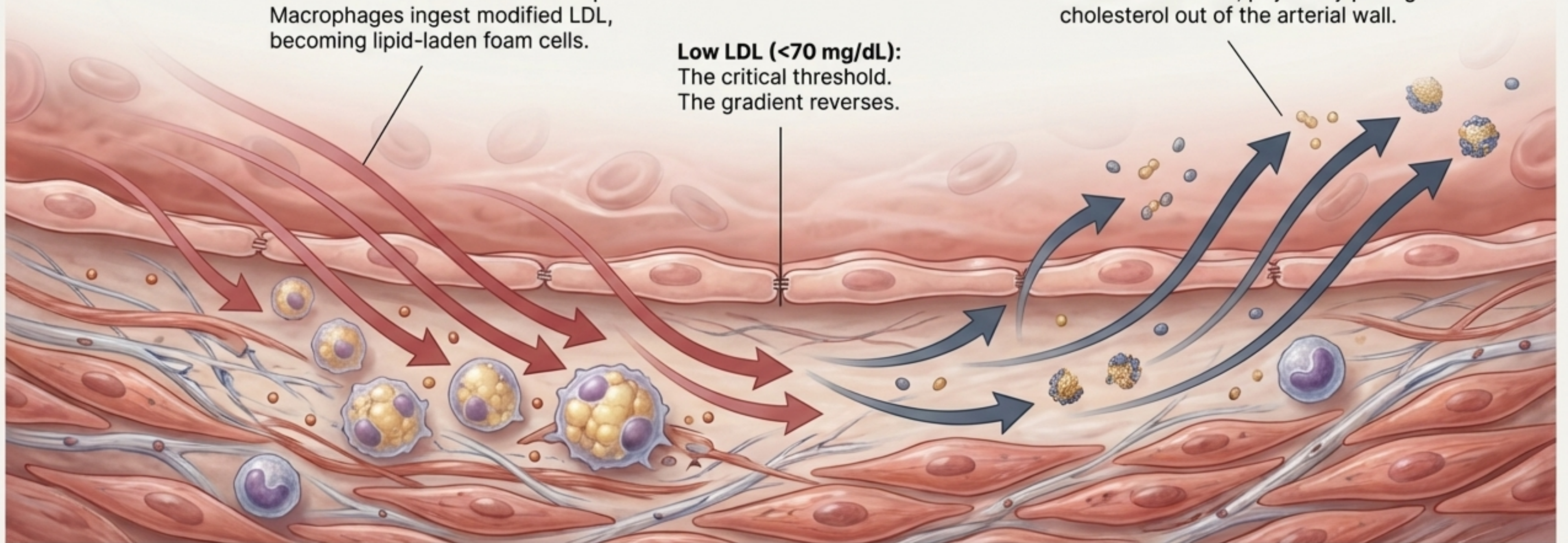
High LDL:

Gradient favors the movement and retention of LDL into the sub-endothelial space. Macrophages ingest modified LDL, becoming lipid-laden foam cells.

Low LDL (<70 mg/dL):
The critical threshold.
The gradient reverses.

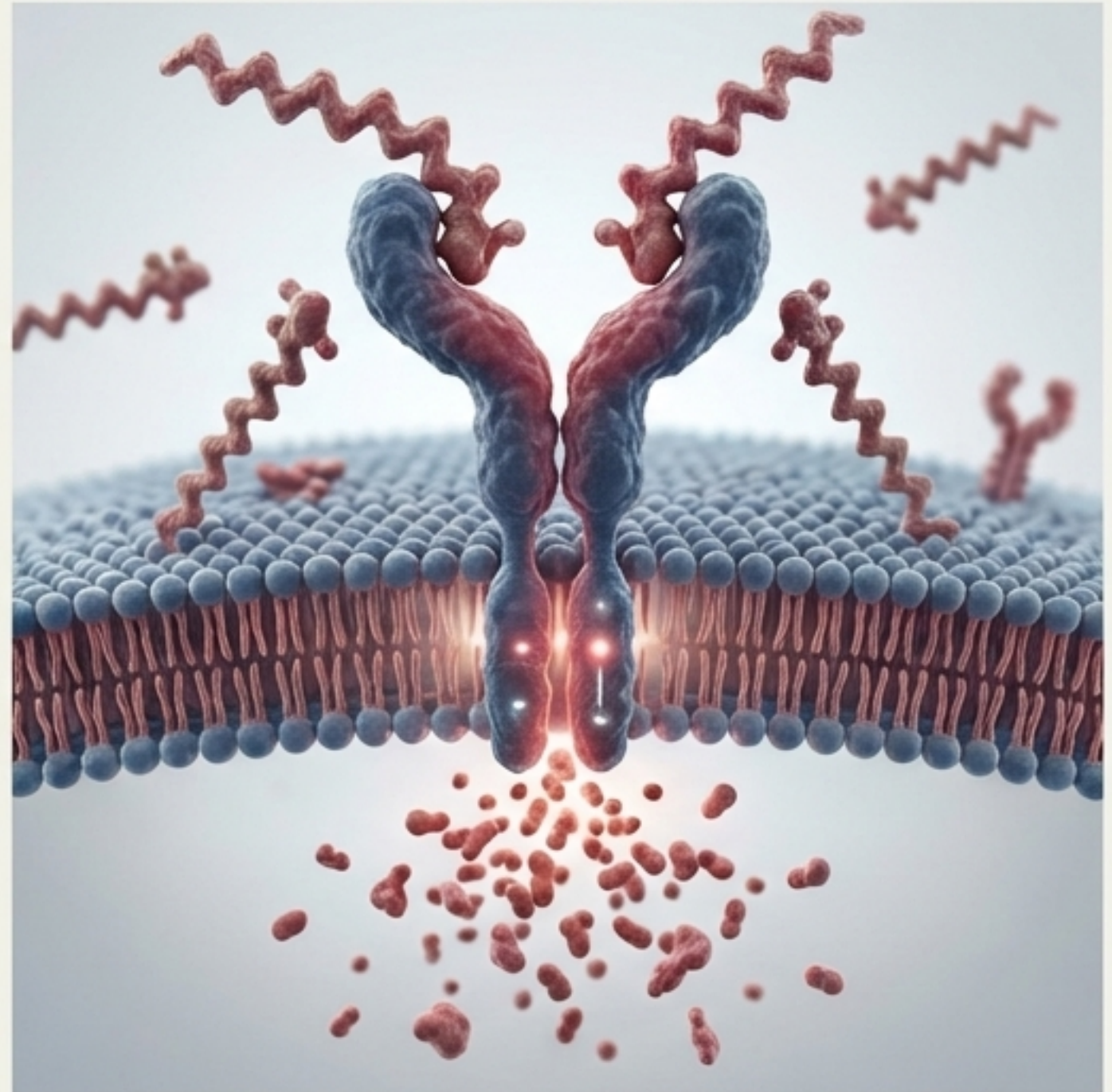
Reverse Cholesterol Transport:

At ultra-low circulating levels, the body initiates net efflux, physically pulling cholesterol out of the arterial wall.

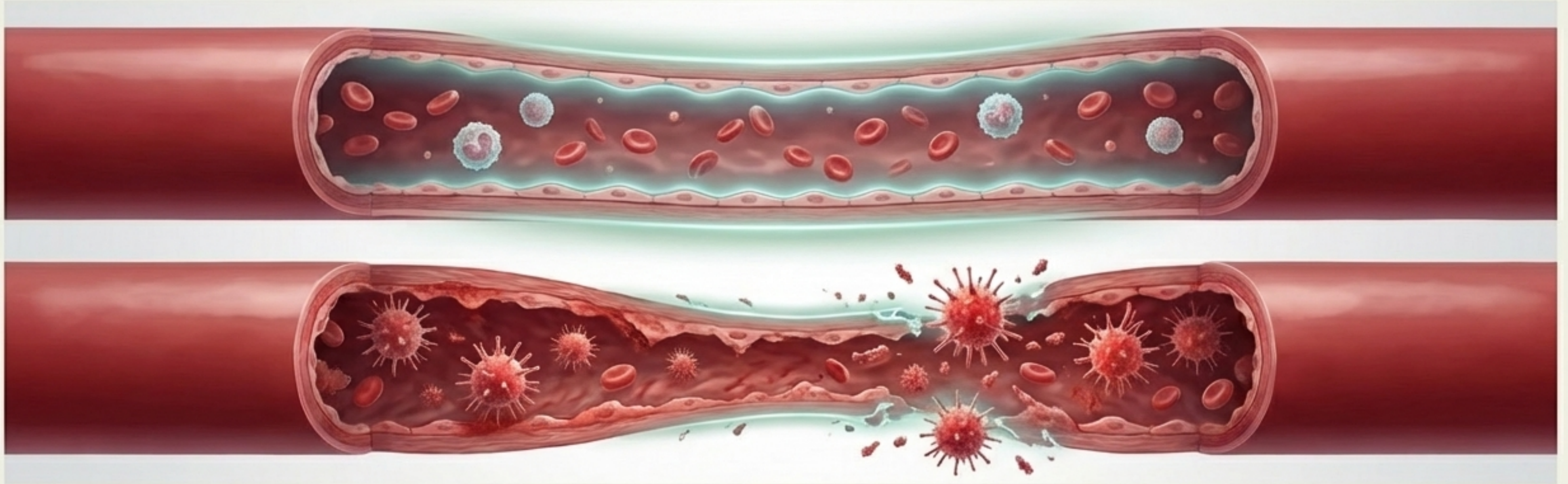


Metaflammation and Plaque Instability

- **The Trigger:** Saturated fatty acids (SFAs) act as danger signals.
- **The Alarm:** SFAs activate Toll-like receptor 4 (TLR4), a pattern-recognition receptor typically used to detect microbial toxins.
- **The Cascade:** Promotes NF- κ B signaling, unleashing inflammatory cytokines (IL-6, TNF- α).
- **The Result:** This immune response actively weakens the fibrous cap, increasing vulnerability to rupture and thrombosis.



The “Irritant” Effect and Nitric Oxide Destruction



The Protective Mechanism: The single-cell endothelial layer produces Nitric Oxide (NO), vital for vasodilation and keeping the vessel non-stick.

The Postprandial Insult: Meals high in animal fat or refined plant oils flood the circulation with triglyceride-rich lipoproteins.

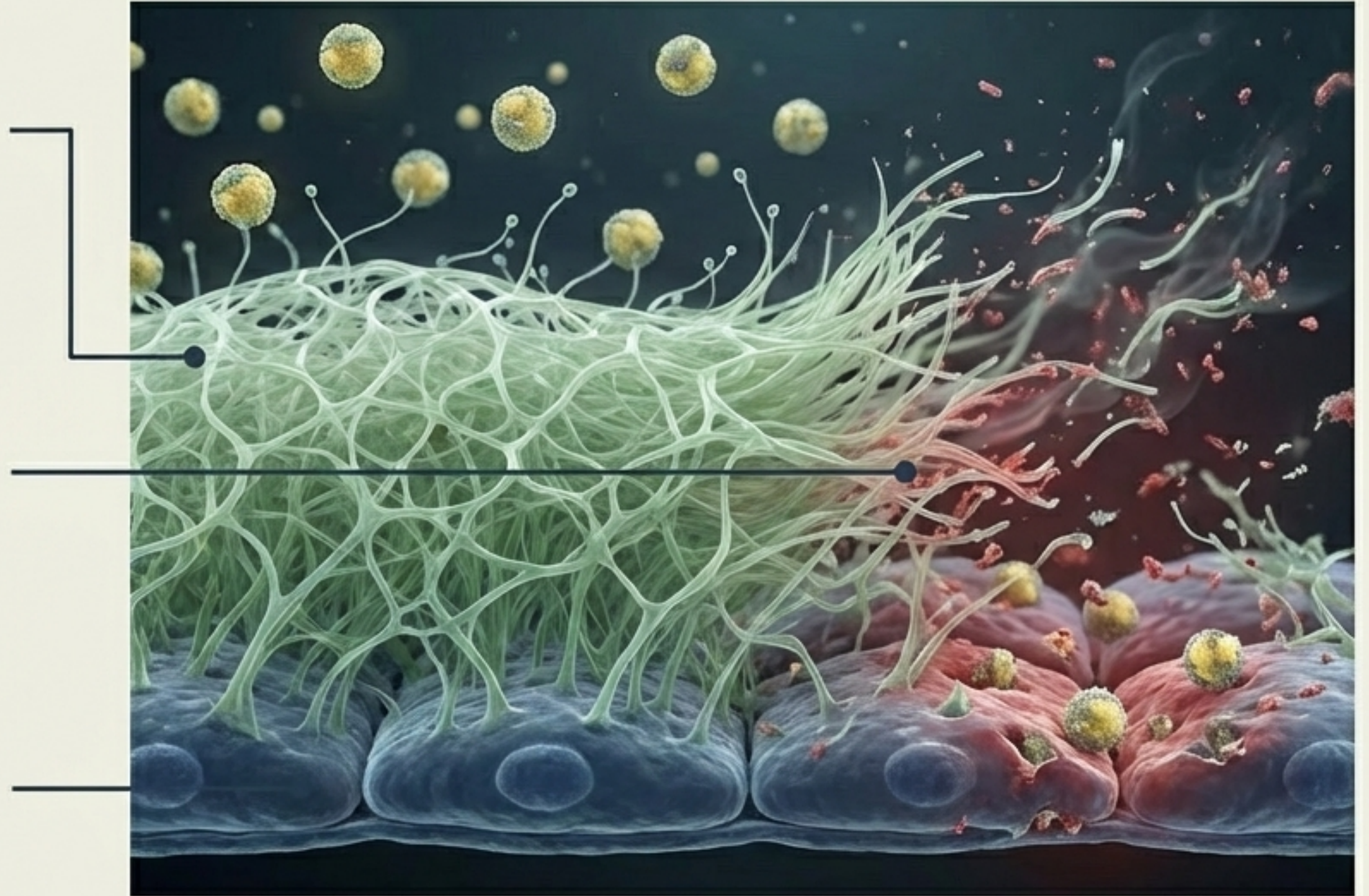
The Destruction: The resulting oxidative stress neutralizes NO, converting it into peroxynitrite. The vessel loses its vasodilatory signaling and becomes irritated.

The Biological Barrier: The Endothelial Glycocalyx

The Defense: A mechanotransducer and physical gel barrier that reduces leukocyte adhesion and prevents LDL infiltration.

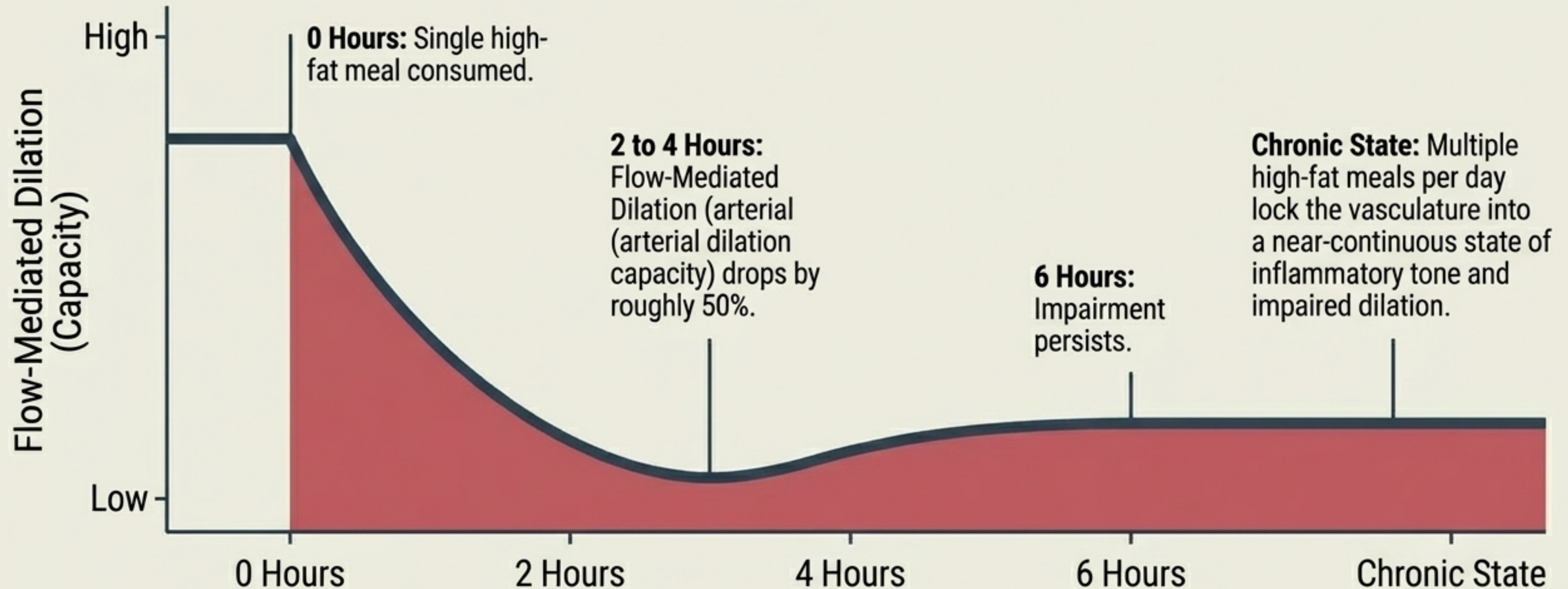
The Vulnerability: Metabolic stressors (hyperglycemia, oxidative stress, oxidized LDL) cause this delicate layer to shed directly into circulation.

The Consequence: Stripped of the glycocalyx, the vessel surface becomes highly adhesive and vulnerable to injury.



The Immediate Impact: A 50% Drop in Function

The Postprandial State



Epidemiological Evidence: Societies Without Heart Disease



Population	Fat Intake (%)	Primary Carb	CAD Mortality
Rural China (1970s/80s)	<10% Fat	Grains/Legumes	1/17th the rate of the US
Traditional Okinawa (Pre-1960)	6% Fat	Purple Sweet Potato	Extremely Low
Modern USA	35%+ Fat	Refined Sugars	High

Note: Rural Chinese populations achieved average serum cholesterol of ~127 mg/dL—levels considered low in the West, but associated with an absence of chronic disease.

The Crossover Limit for Advanced CAD

For individuals with severe coronary artery disease, the crossover limit is exceptionally low.

Disease regression demands a highly stable biochemical environment. Moderate diets (25–30% fat) may support primary prevention, but they lack the biochemical intensity required to clear the lipid burden from established, advanced lesions.

Frequent dietary cheats prevent sustained low LDL and maintain chronic inflammatory pressure.

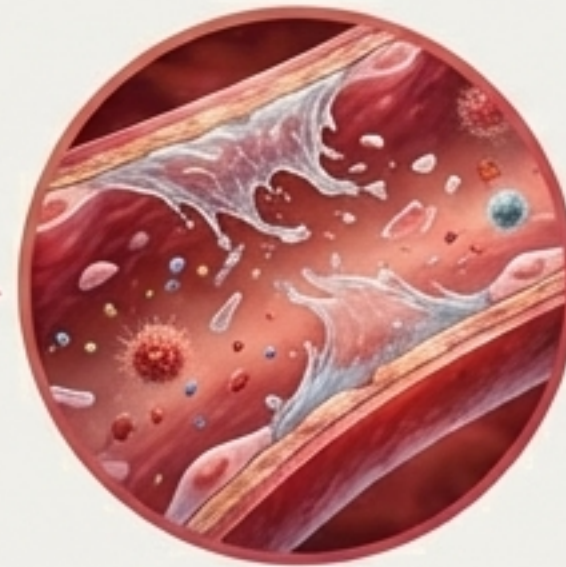


The Anatomy of Nutritional Indiscretion



1. The 6-Hour Window

Endothelial responsiveness and vasodilation are immediately impaired.



2. Glycocalyx Damage

Hyperglycemia and oxidative stress promote the physical shedding of the protective endothelial surface layer.



3. Immune Re-Activation

For a plaque currently in a cooling stabilization phase, the sudden influx of saturated fats re-activates TLR4 signaling, amplifying inflammatory cascades and risking cap rupture.

Achieving Sustained Atherosclerotic Regression

Vascular irritation reflects real postprandial physiology. By addressing the root drivers—cholesterol retention and immune activation—rather than managing symptoms alone, an ultra-low-fat WFPB protocol provides a credible, non-surgical pathway toward comprehensive cardiovascular health.

The Required Environment:

- ✓ Sustained ultra-low LDL (<70 mg/dL)
- ✓ Reduced TLR4 inflammatory signaling
- ✓ Minimal postprandial lipid burden
- ✓ Strict adherence to <10% total fat, free of refined oils



Boston Heart Disease Consultation

An evidence-based organization utilizing the best available science to reverse and cure heart disease.

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