The Prescription Every Cardiologist Should Give

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Agenda

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Patient Case Study

A missed opportunity story

02

Social Media Wars

Misinformation and its impact

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Cholesterol Basics

Structure, function, atherosclerosis

04

Risk Assessment & Testing

Standard panels and advanced markers

05

Treatment Approaches

Medications, supplements, diet

Opening Case

Meet a 55-year-old patient with a 20-year history of **untreated hypercholesterolemia**.

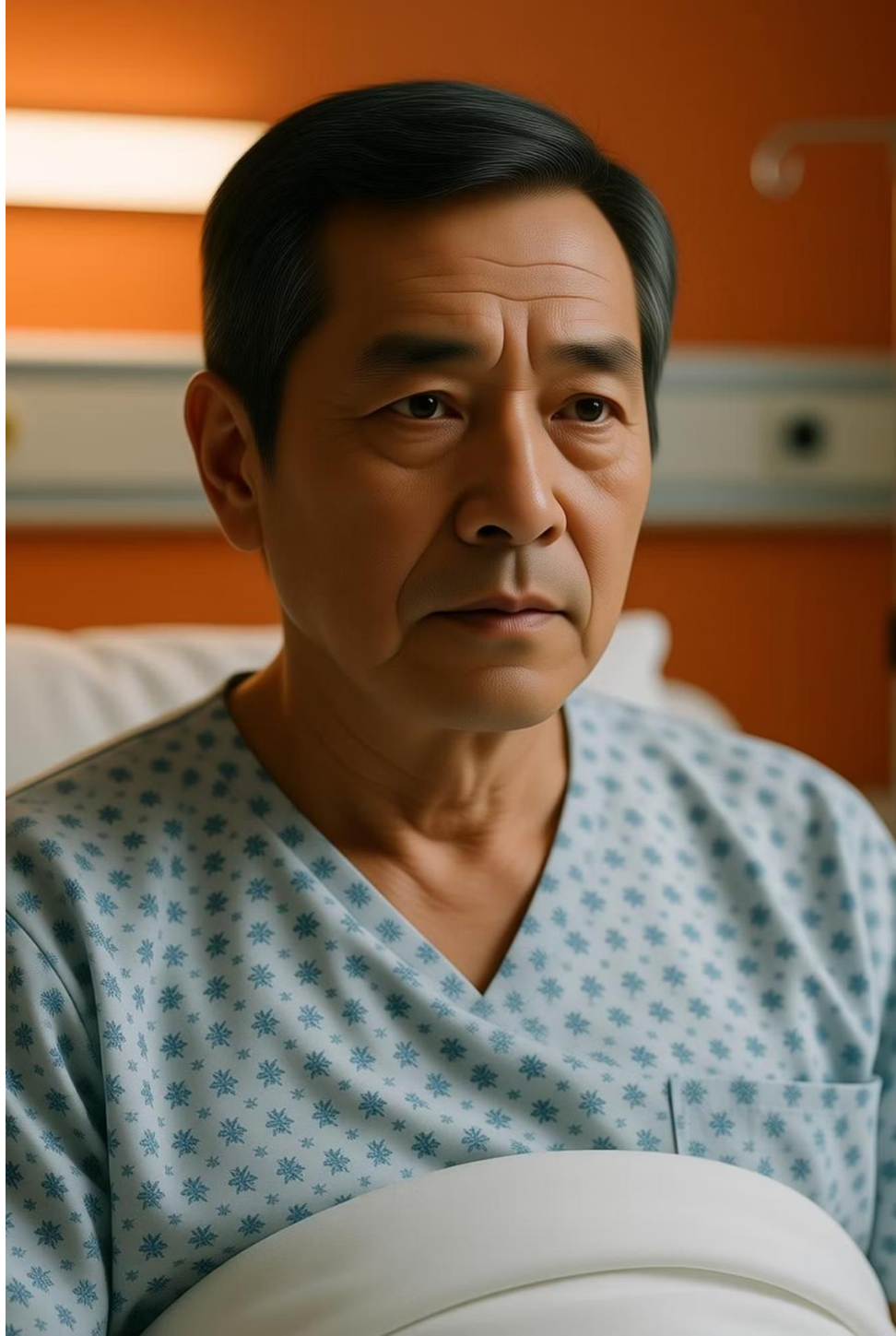
Despite multiple prior medical encounters, their lipid profile consistently showed concerning numbers:

- Total Chol: 268 mg/dL
- Triglycerides: 215 mg/dL
- LDL-C: 206 mg/dL
- HDL-C: 47 mg/dL

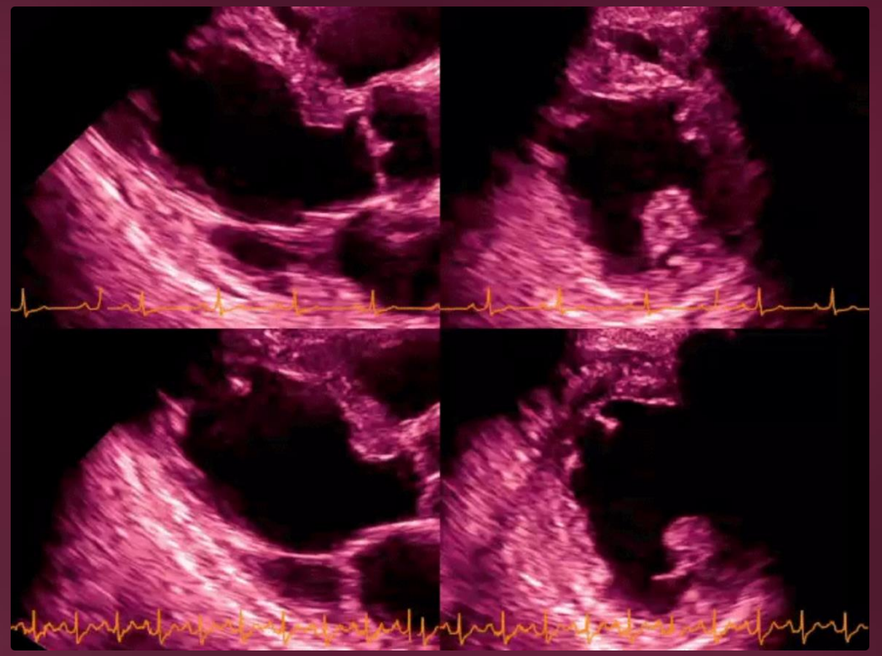
Hospitalization

Hospitalized for progressive chest pain

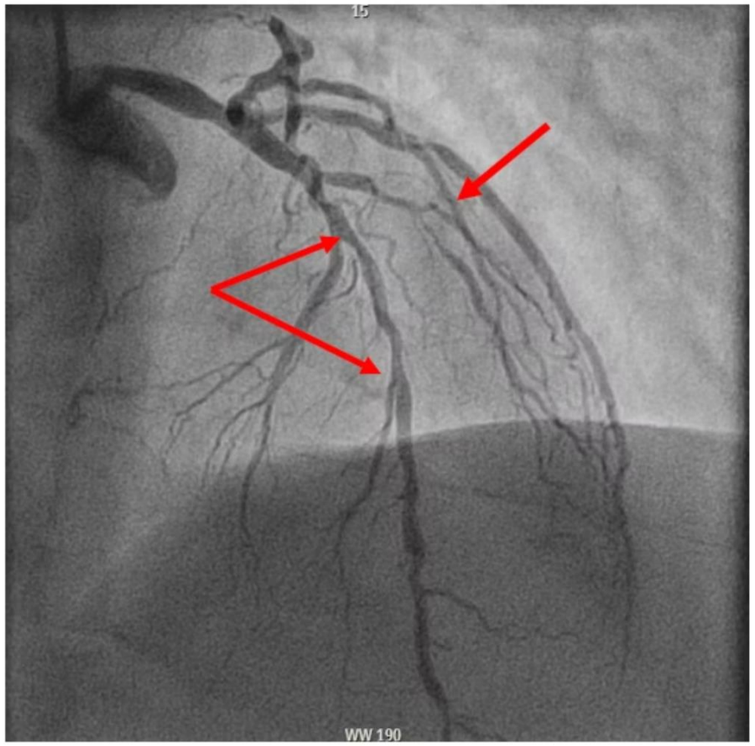
Medical record review reveals missed intervention opportunities

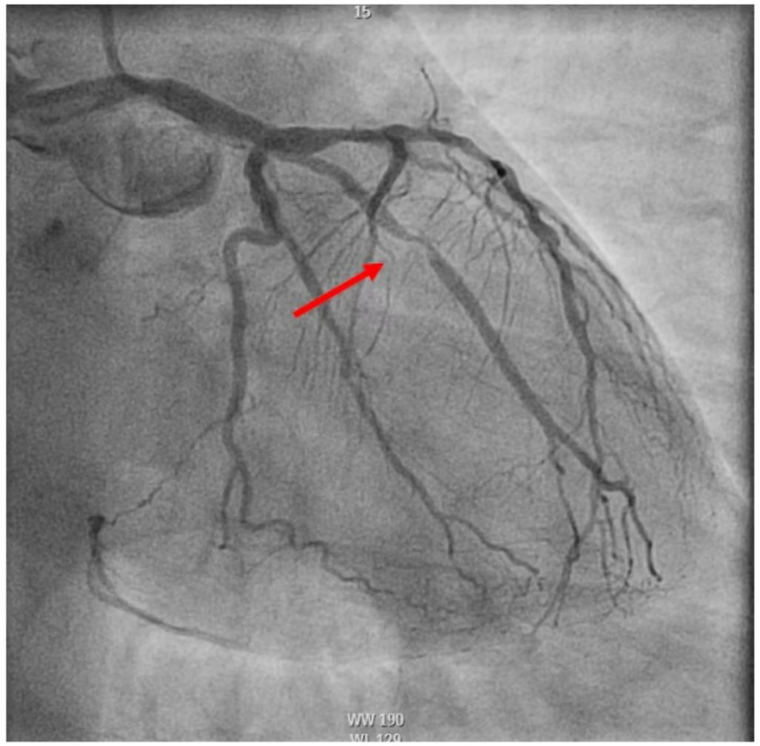


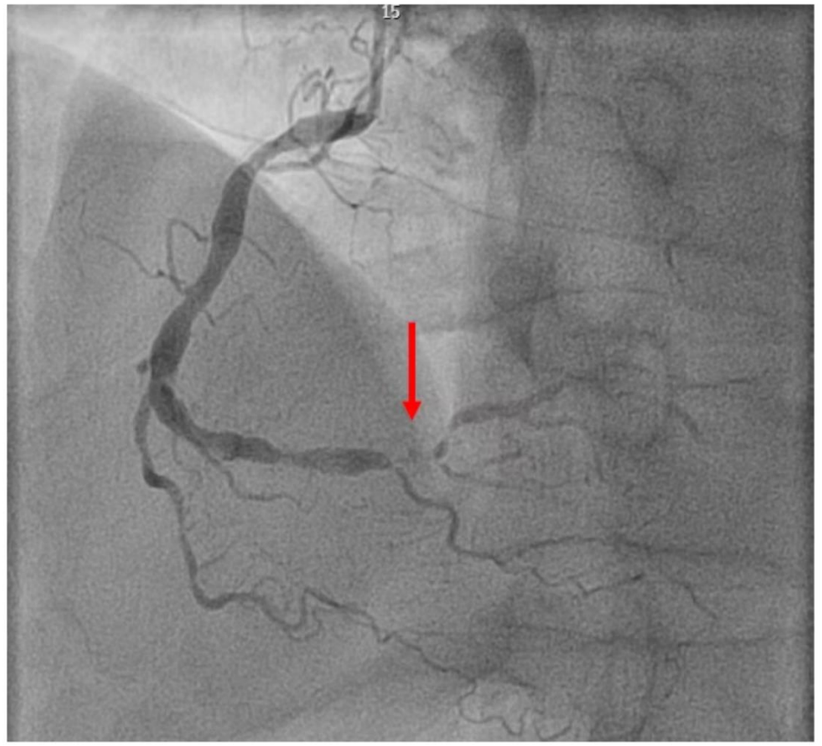
THE UNEXPECTED DISCOVERY

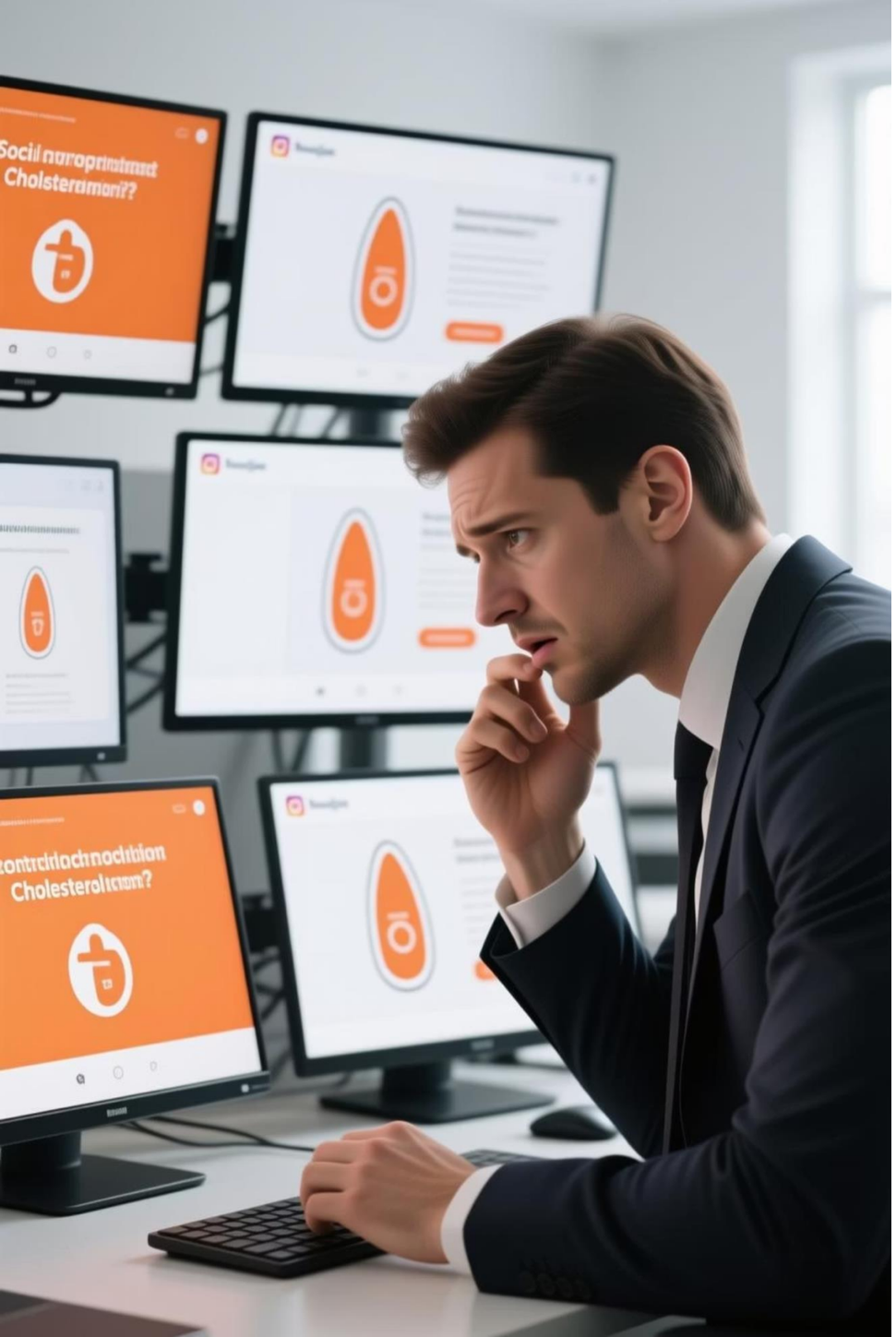


Recommendations For Cardiac Catheterization









The Social Media Cholesterol Wars

Common Misconceptions from Reddit

> "LDL is pretty much irrelevant"

> "Pharmaceutical industry just wants to sell drugs"

> "Statin side effects are worse than high cholesterol"

Analysis found widespread misinformation across platforms [4][5]

Impact on medication adherence and cardiovascular outcomes [6]

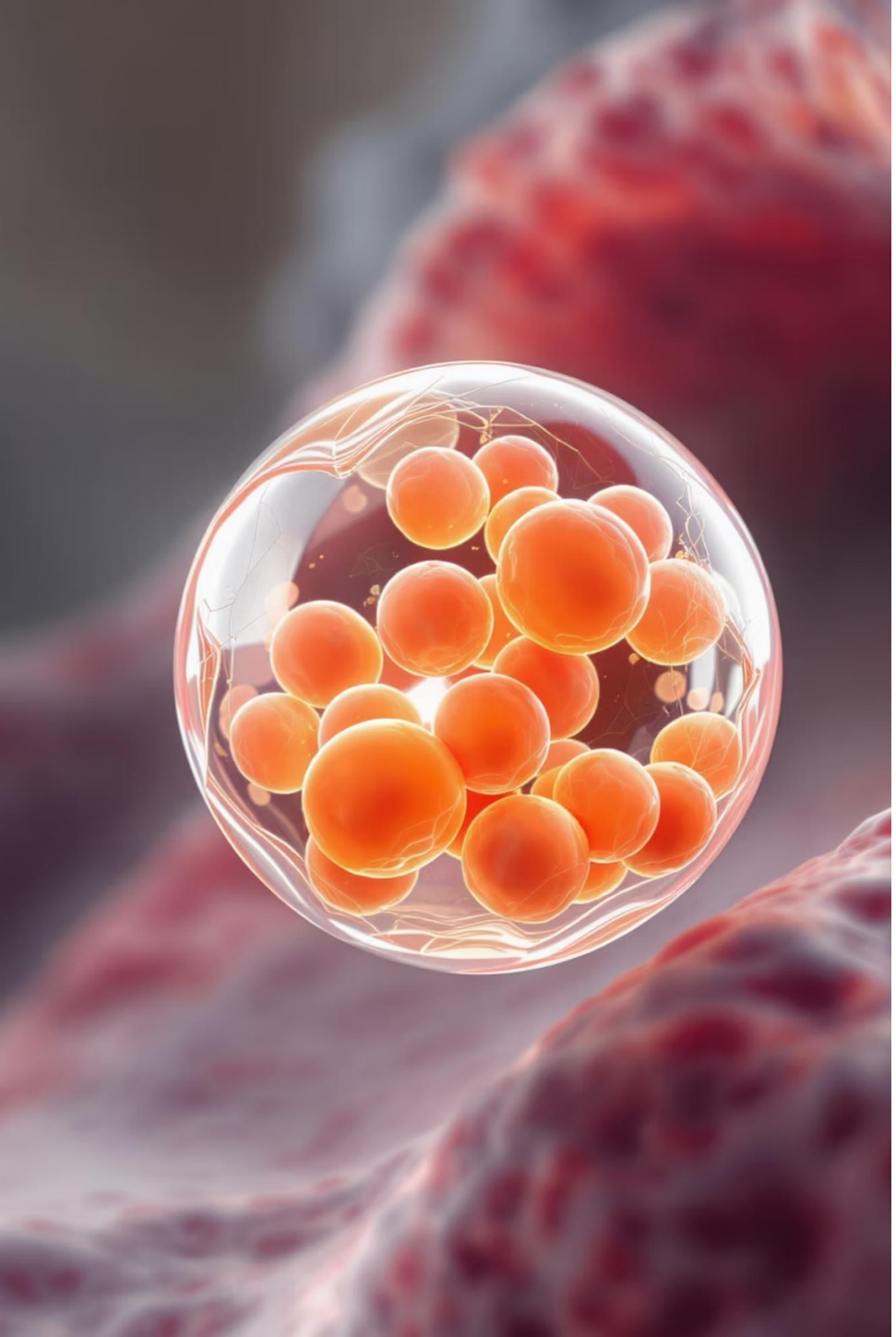
AI flagging tools being developed to combat misinformation [79]

References: [4] PMC 10126874, [5] Medscape 991585, [6] PMC 11179751, [79] Community.the-hospitalist.org

#1 reason there is confusion about ***cholesterol***- "not understanding what it is"

Cholesterol 101

Cholesterol is a waxy, fat-like substance ***vital for healthy cells, hormone production, and vitamin D synthesis.*** While essential and naturally produced by your body, an excess can accumulate in arteries.



Lipoprotein Transport: Why Cholesterol Needs a Ride

Cholesterol, being a lipid, is not soluble in blood, which is primarily water. To travel throughout the body, it **must be packaged into specialized particles called lipoproteins**. Think of lipoproteins as tiny, spherical delivery trucks that ferry cholesterol and other fats to and from cells.

Lipoprotein Transport System

Low-Density Lipoprotein (LDL)

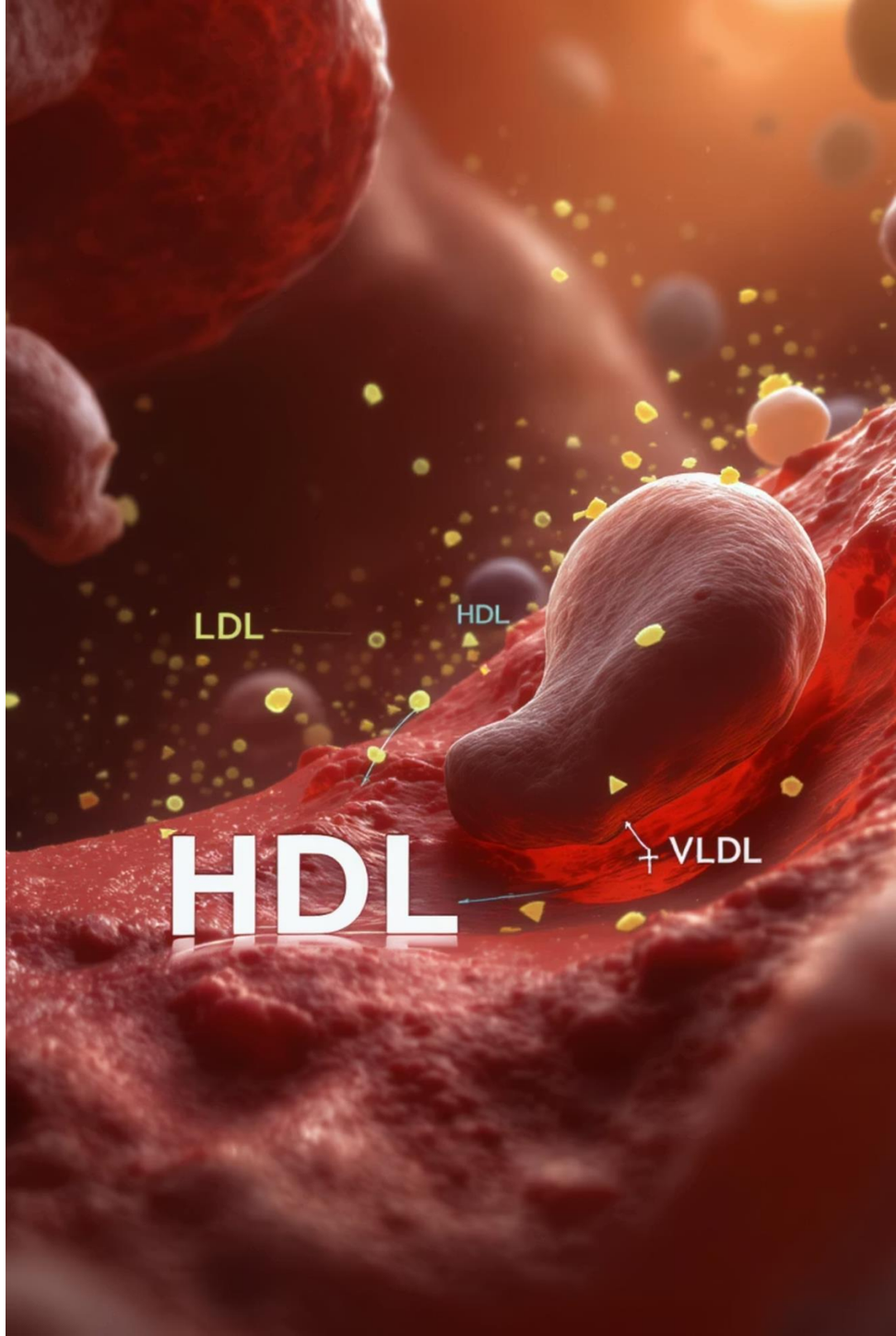
LDL transports cholesterol from the liver to cells.

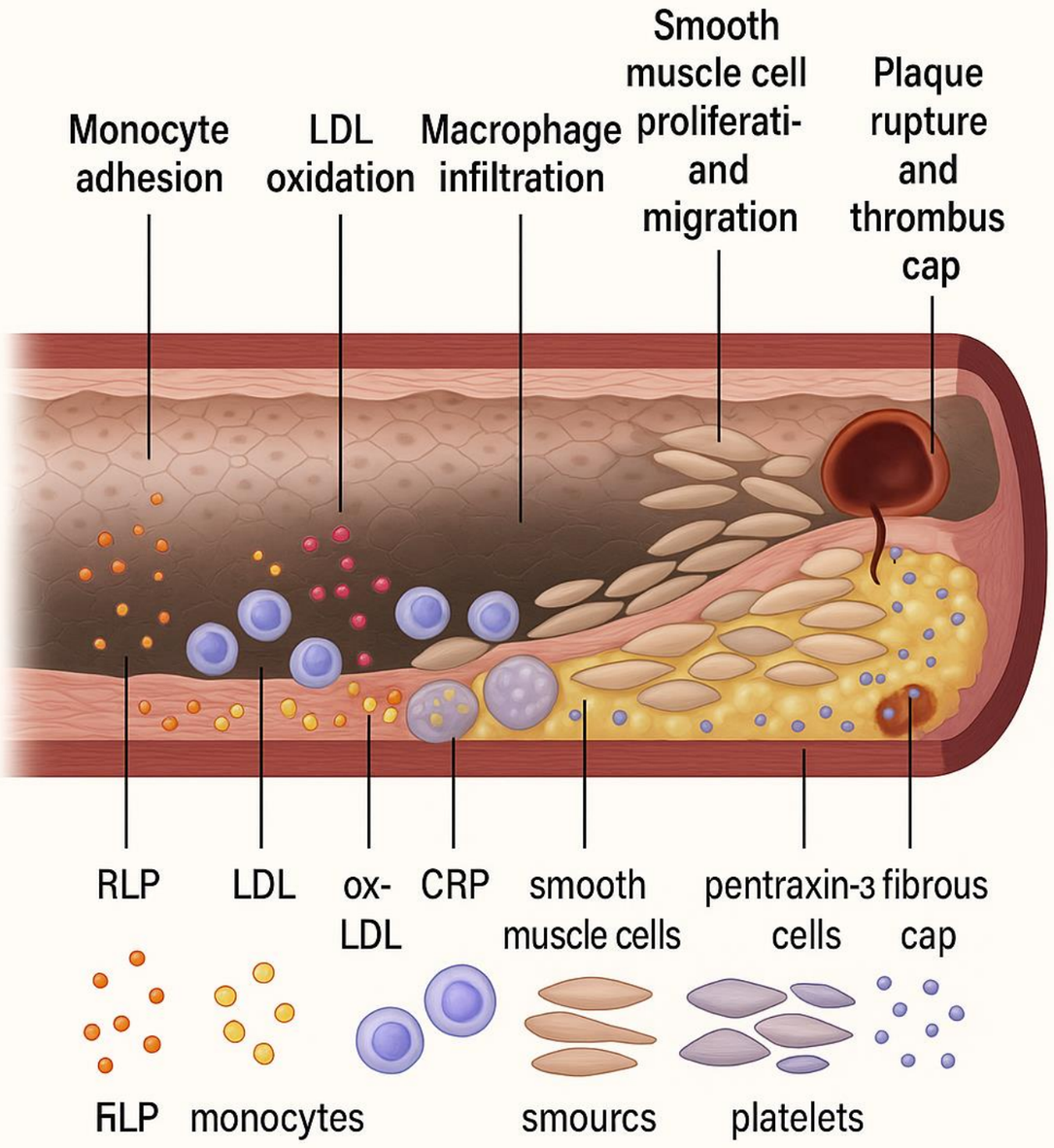
High-Density Lipoprotein (HDL)

HDL removes excess cholesterol from arteries and returns it to the liver.

Very Low-Density Lipoprotein (VLDL)

VLDL transports triglycerides to tissues; its remnants can contribute to arterial plaque.





EPA reported to exert beneficial effects at multiple steps in the atherogenic pathway

The Atherosclerotic Process

Endothelial Dysfunction: The Initial Spark

Atherosclerosis begins with endothelial dysfunction, where the normally smooth and protective inner lining of blood vessels becomes damaged. This ***damage increases its permeability and stickiness***.

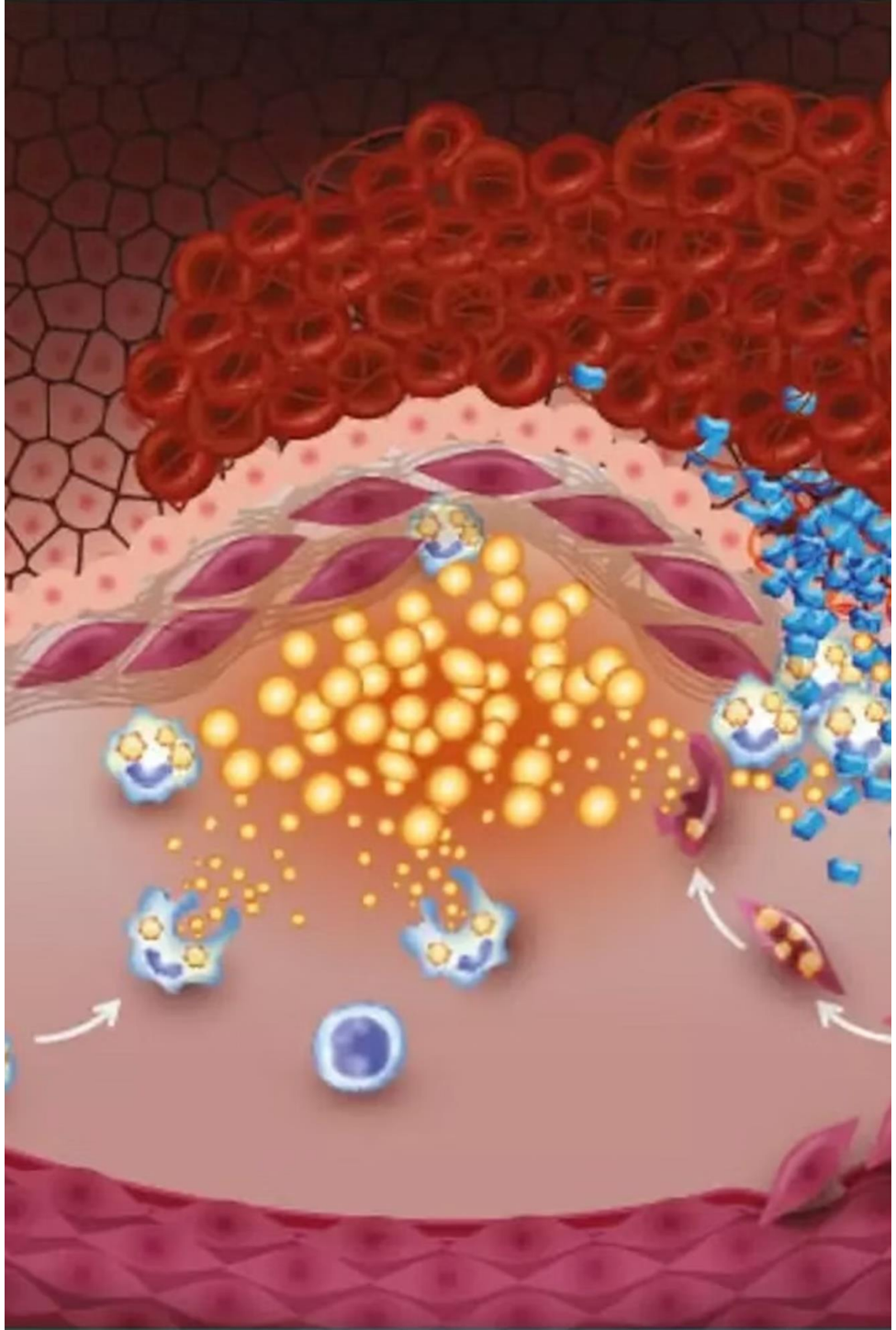
LDL Infiltration and Plaque Formation

Following initial endothelial damage, LDL particles penetrate the arterial wall and undergo oxidation. This modification triggers an immune response, attracting monocytes which transform into macrophages.

Macrophages then avidly engulf the oxidized LDL, transforming into lipid-laden "foam cells." The accumulation of these foam cells forms a visible fatty streak, the earliest pathological lesion of atherosclerosis.

Plaque Rupture: What Increases Vulnerability

Plaque rupture, not just size, primarily triggers acute cardiovascular events like heart attacks and strokes. Key instability factors include a thin fibrous cap, a large necrotic core, and critically, the presence of **cholesterol crystals**. These sharp structures mechanically stress the plaque and trigger inflammation, significantly increasing rupture risk.



Stenosis Severity and Myocardial Infarction Risk

Early studies tied most heart attacks to mild stenoses (<50%), but newer data show acute heart attacks usually ***arise from ~66% lesions*** that thrombose—***not 70–90% blockages.***

Standard Lipid Panel

- Total cholesterol
- LDL-C (calculated)
- HDL-C
- Triglycerides

UNDERSTANDING TRIGLYCERIDES

What Are Triglycerides?

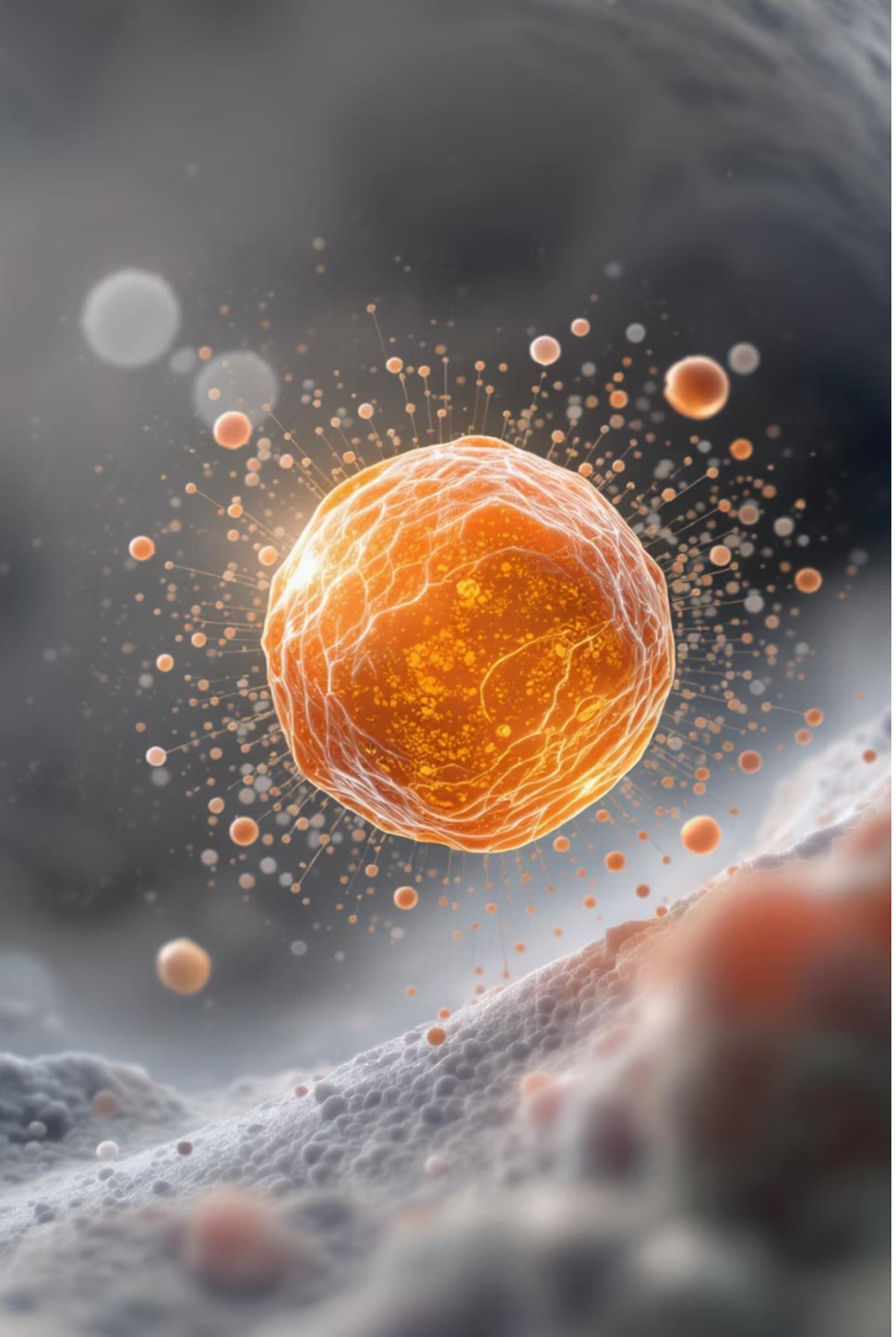
- ***Main form of fat storage*** in the body
- Composed of glycerol and three fatty acids
- Transported in the blood by lipoproteins (chylomicrons, VLDL, remnants)

Elevated levels often indicate metabolic issues

Triglyceride Levels

Normal	<150 mg/dL
Borderline High	150–199 mg/dL
High	200–499 mg/dL
Very High	≥500 mg/dL

Important insight: High triglycerides are independently associated with cardiovascular risk, even when LDL cholesterol is controlled.



HDL CHOLESTEROL: THE "GOOD" CHOLESTEROL

Cholesterol Scavenger

HDL performs **reverse cholesterol transport.**

Artery Protector

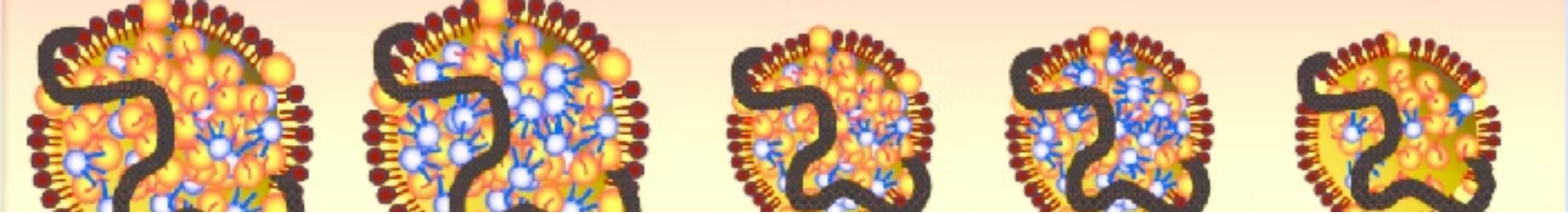
Prevent plaque buildup (atherosclerosis)

Beyond Cholesterol

Possesses **anti-inflammatory and antioxidant properties**,.

Optimal HDL Levels:

Optimal	**>60 mg/dL**
Good	**40–59 mg/dL**
Poor (Increased Risk)	**<40 mg/dL**



LDL CHOLESTEROL

Calculation & Limitation

LDL-C is typically calculated using the Friedewald equation:

> LDL-C = Total Cholesterol - HDL-C - (Triglycerides/5)

Note: This calculation is inaccurate if triglycerides are >400 mg/dL.

LDL Level Classifications

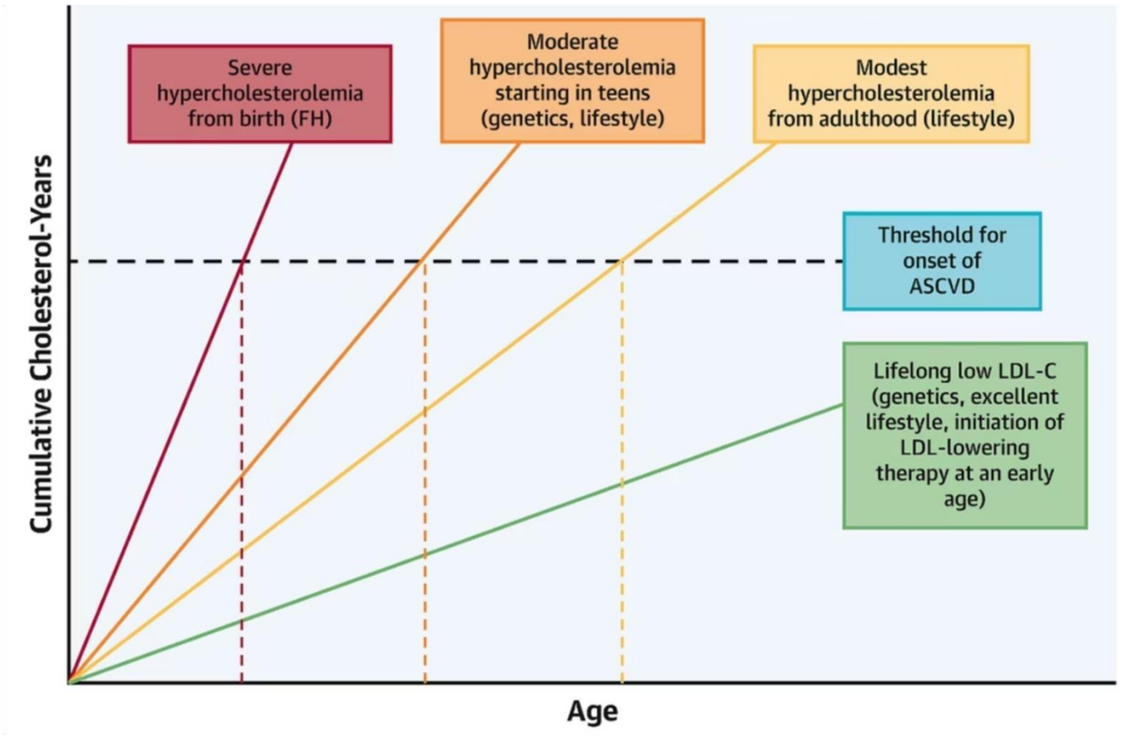
Category	LDL (mg/dL)
Optimal	<100
Near optimal	100–129
Borderline high	130–159
High	160–189
Very high	≥190

High-risk individuals may target <70 mg/dL.

#2 reason there is confusion about cholesterol- not understanding ***risk***

The Cumulative Risk Paradigm

Research shows that cardiovascular risk is determined not just by your current cholesterol levels, but by your lifetime exposure to atherogenic lipoproteins.



References: [13] PMC 8459309, [14] JAMA Cardiology 2784038

Cumulative LDL-C Exposure and Cardiovascular Risk

57%

Increased Risk

For cumulative exposure vs. single time point measurement [14]

20-29

Age Range

Early-life cholesterol levels predict future events [15]

Time-weighted average LDL-C superior to single measurement for risk prediction

References: [14] JAMA Cardiology 2784038, [15] PACE-CME

Primary vs. Secondary Prevention

Primary Prevention

Before cardiovascular disease develops

The 2022 USPSTF review of 22 RCTs (90,624 participants) found statins in ***primary prevention reduced all-cause mortality by 8%, MI by 33%, and stroke by 22%.***

Secondary Prevention

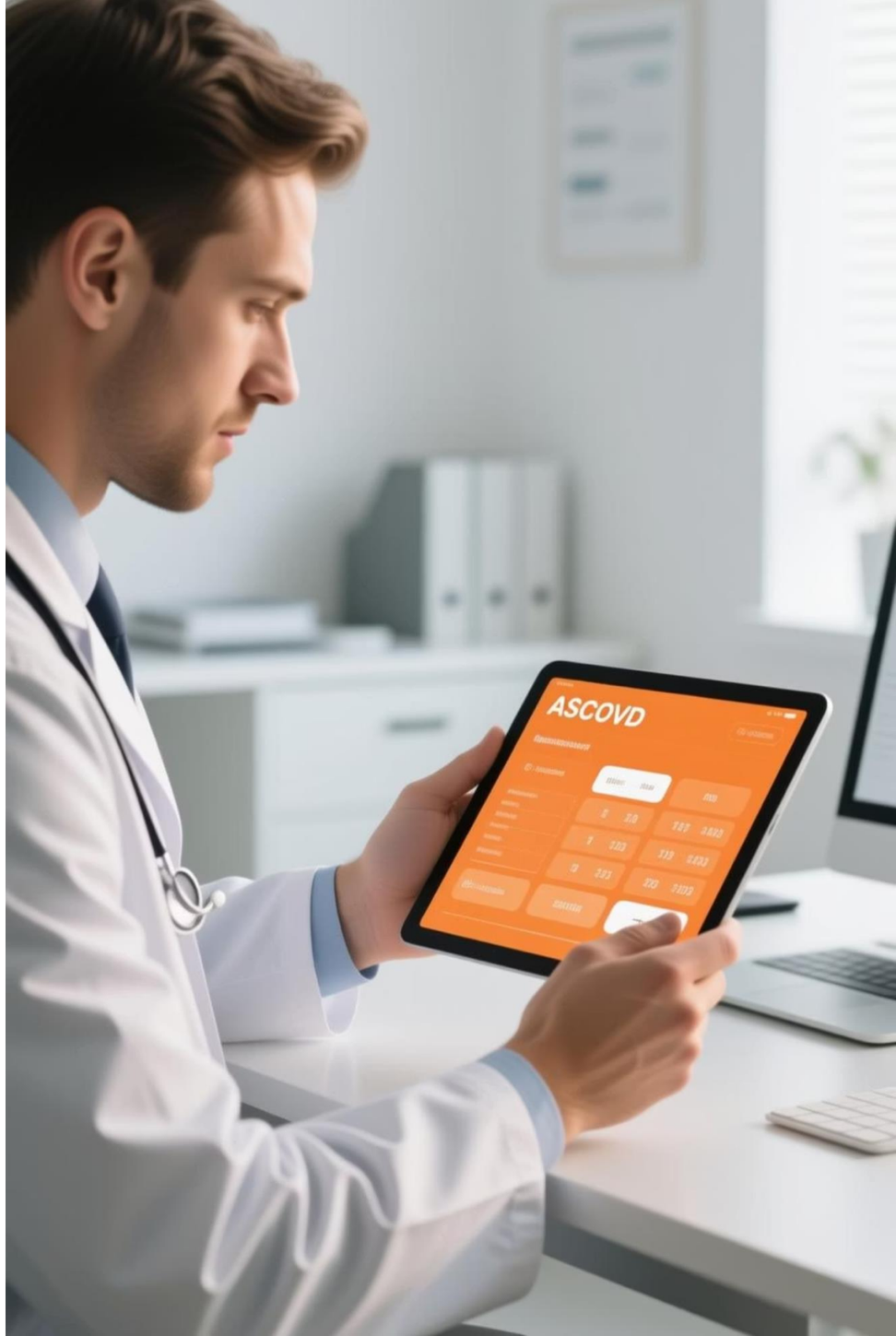
After cardiovascular disease is established

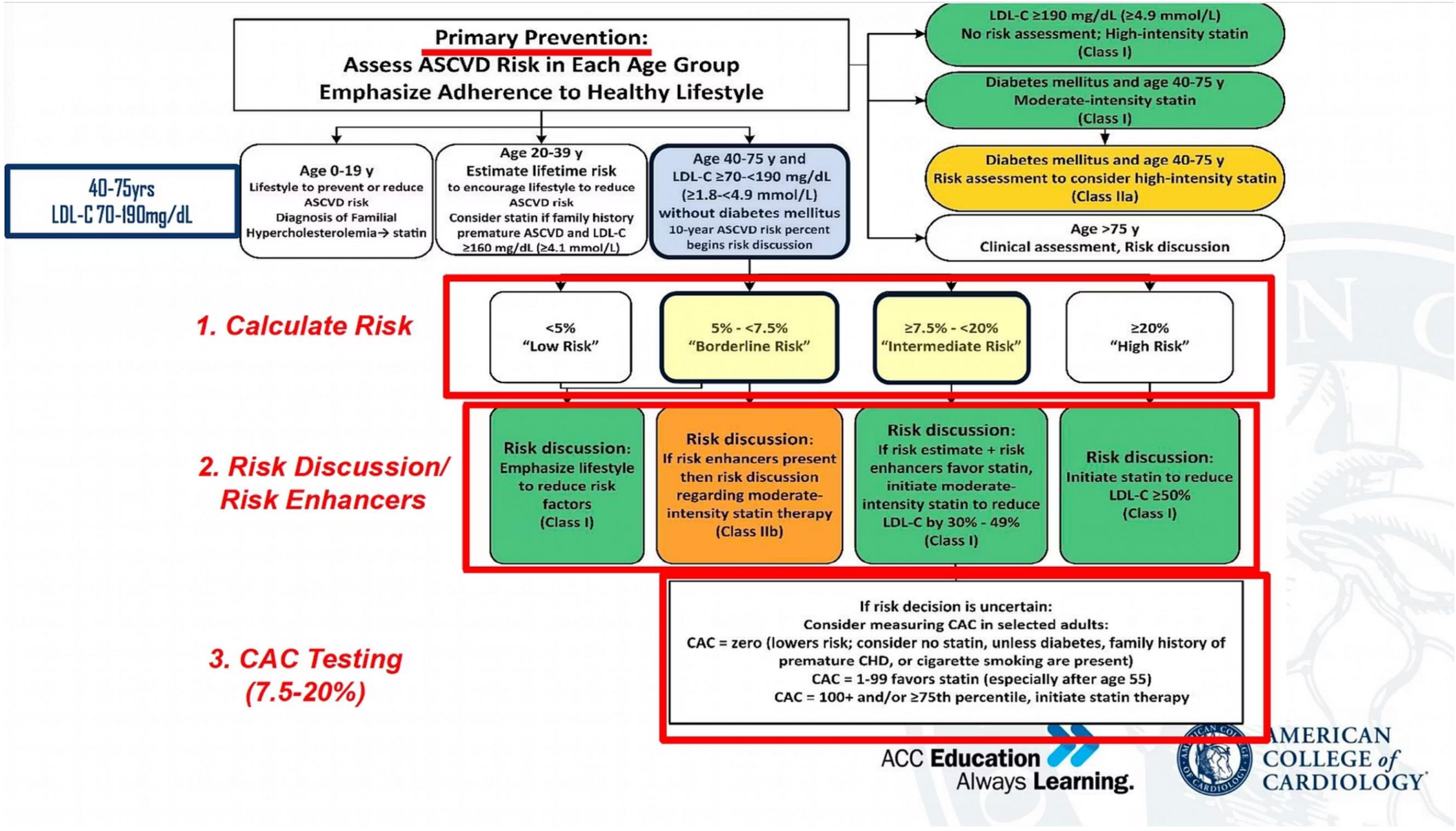
25% risk reduction per 38.6 mg/dL LDL-C reduction [16]

Greater absolute benefit in higher-risk patients

References: [16] PMC 3709236, [17] Statins in Primary and Secondary Prevention. PMC. 2012

Risk Assessment Tools





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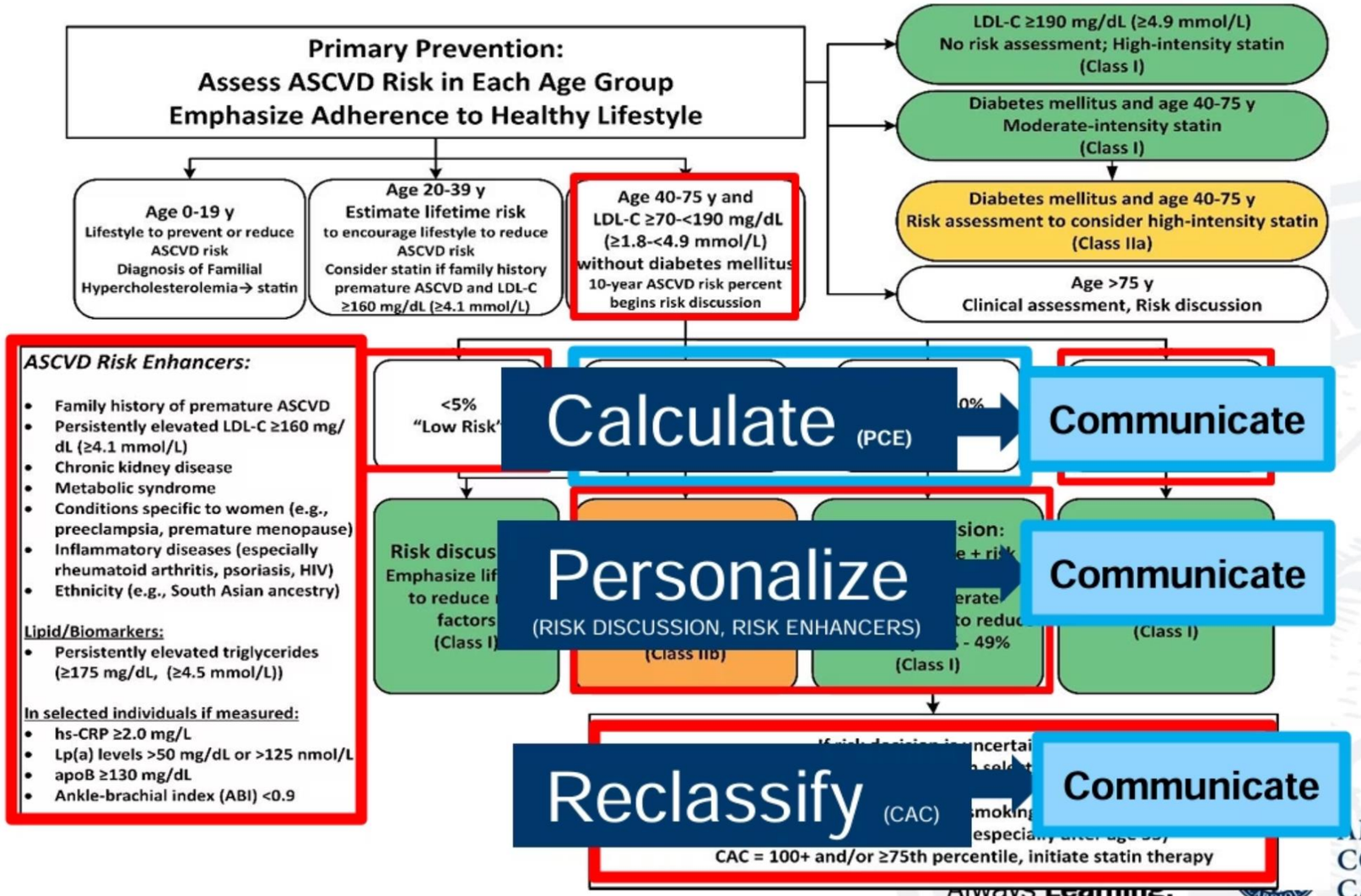
3 Steps in Risk Assessment



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Grundy SM, et al. *J Am Coll Cardiol.* 2018 Nov 8. pii: S0735-1097(18)39034-X.; Grundy SM et al. *Circulation.* 2018 Nov 10:CIR0000000000000625.

2018 Pooled Cohort Equations

10-year ASCVD Risk

Estimates probability of heart attack or stroke in next decade

Risk Factors

Age, sex, race, cholesterol, BP, diabetes, smoking status

Risk Categories

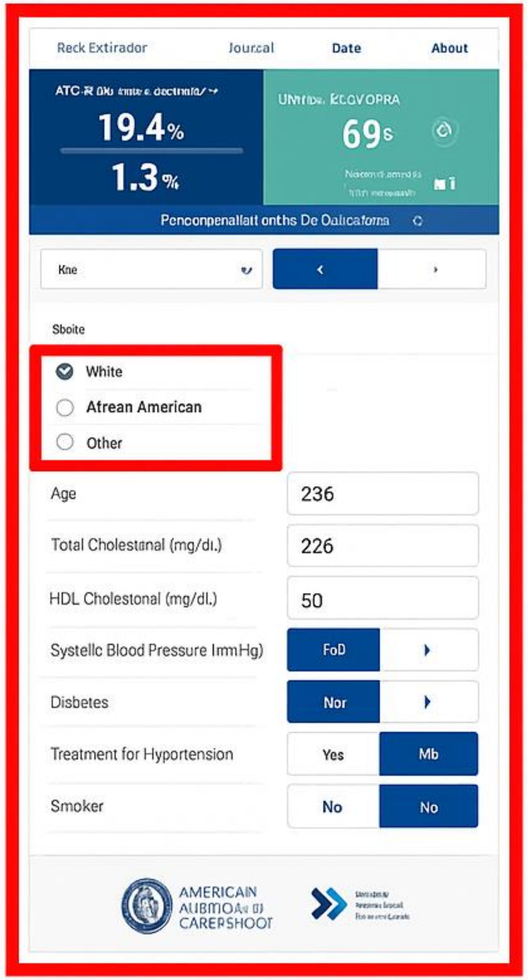
Low (<5%), Borderline (5–7.5%), Intermediate (7.5–20%), High (>20%)

Reference: [18] ACC/AHA Prevention Guidelines. 2018

ACC/AHA ASCVD Risk Estimator

Optimal risk factors

- TC < 170 mg/dL
- HOL-C ≥ 50 mg/dL
- Systolic BP < 110 mnHg
- Not taking medications for HTN
- Not a diabetic
- Not a smoker



10-year risk of non-fatal MI, coronary heart disease death, and fatal and non-fatal stroke

Intended for use if no ASCVD and LDL-C is <190 mg/dL

Goff DC, et al. *J Am Coll Cardiol* 2014,$3.2835-59

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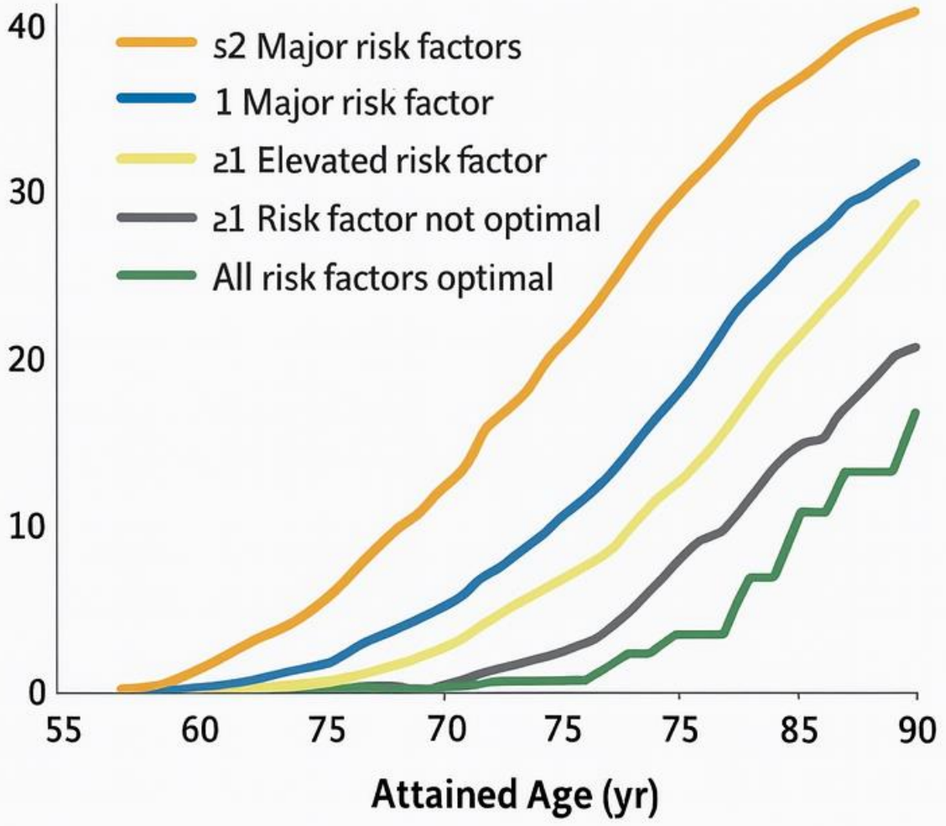
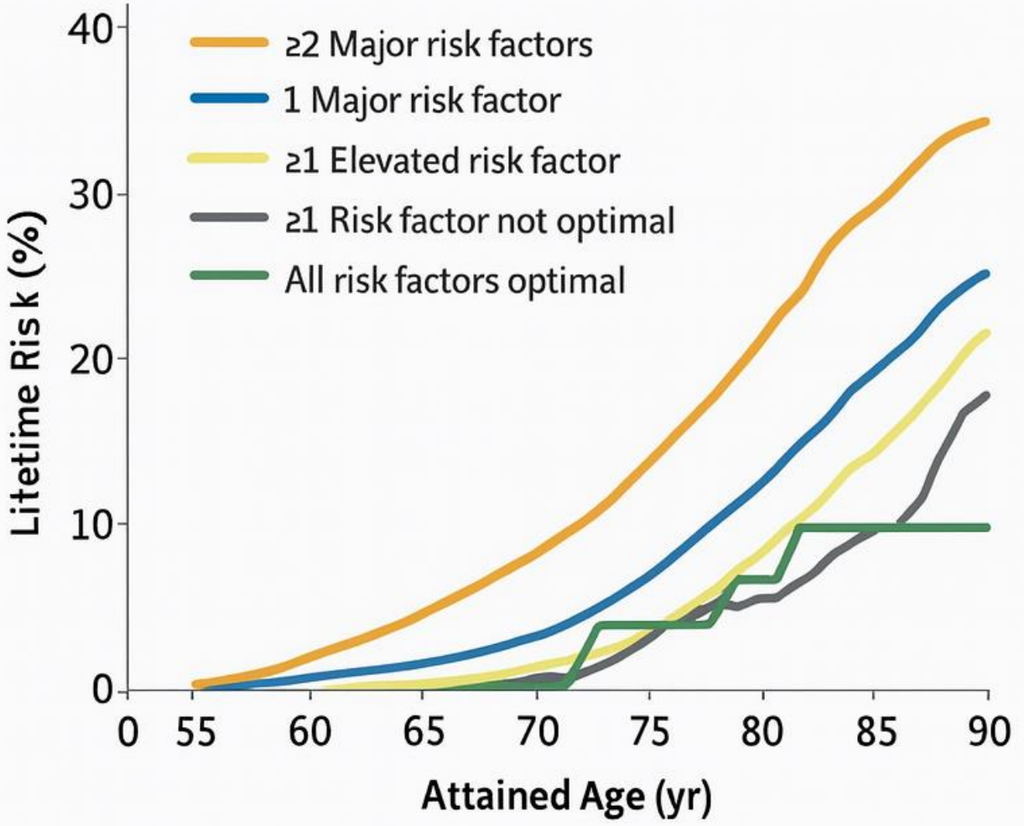
Assessment of Lifetime Risk

MEN



WOMEN



Berry JD et al. N Engl J Med 2012;321–329

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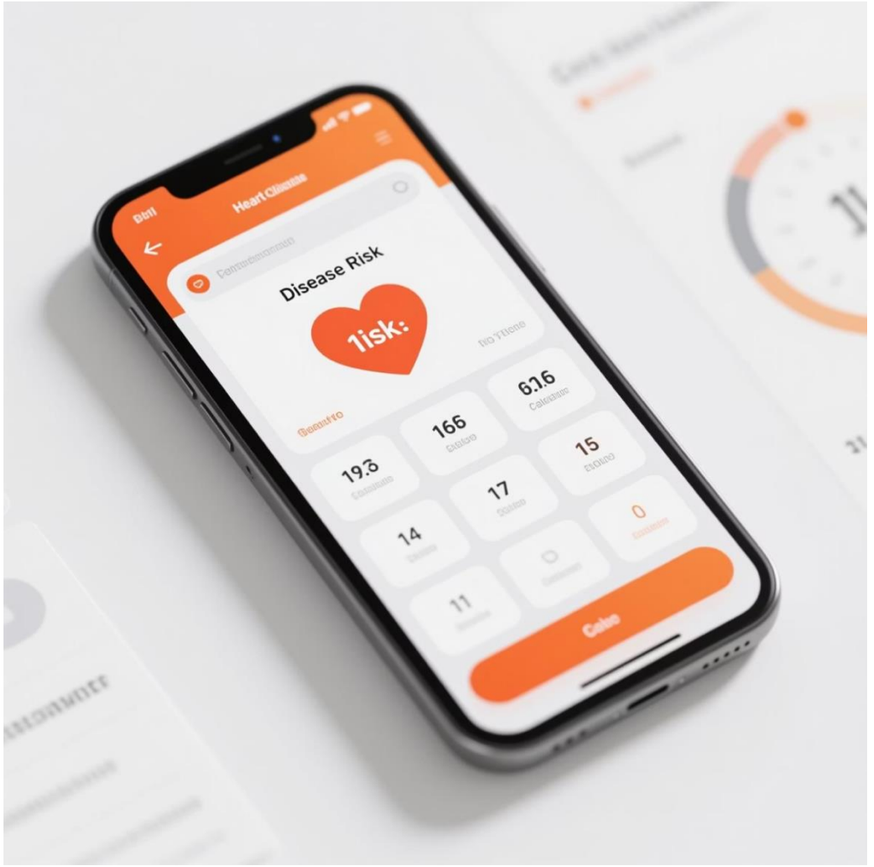
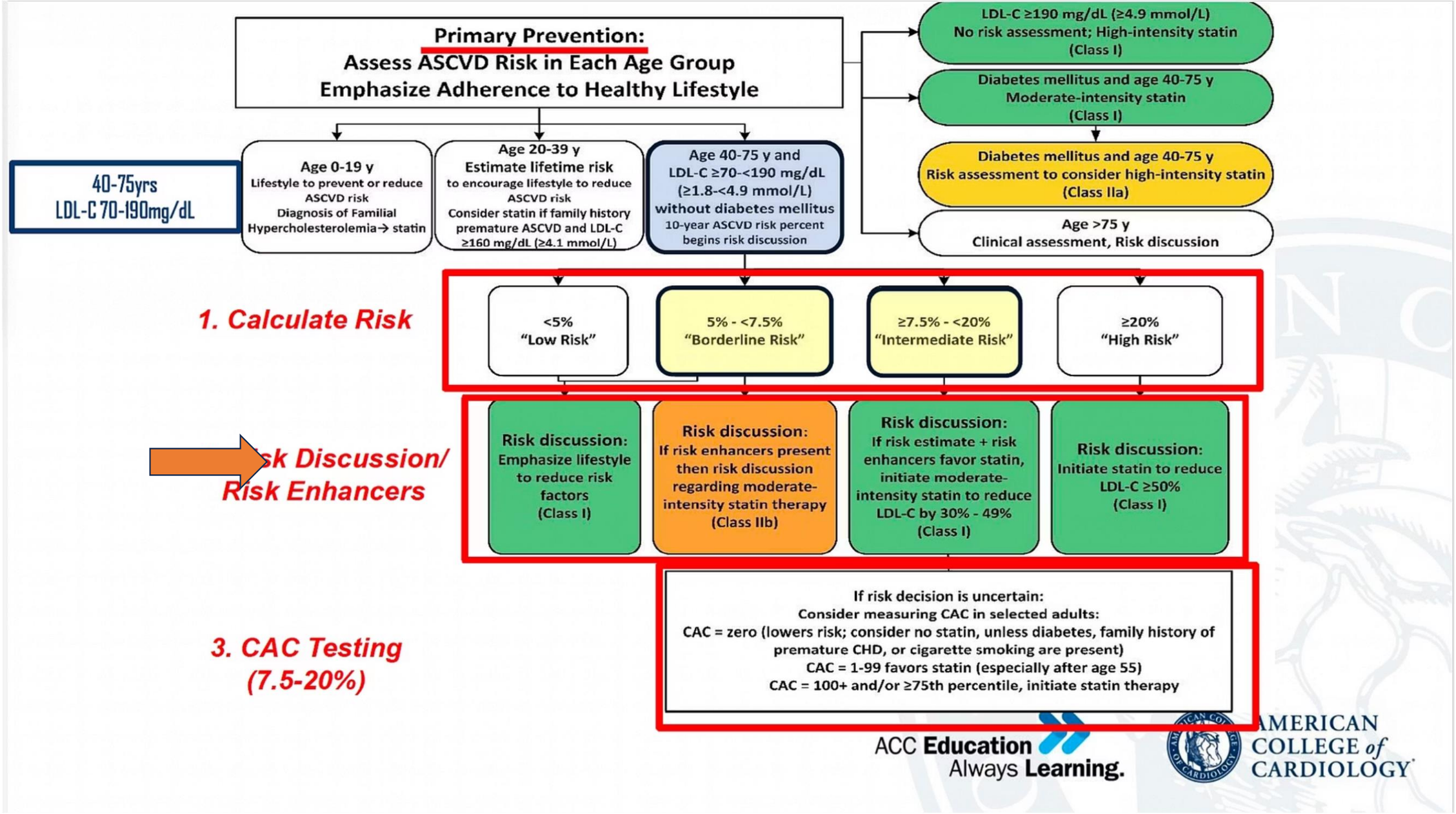
2023 PREVENT Calculator

- Total CVD, ASCVD, and heart failure risk
- Additional parameters:
  - Kidney function
  - HbA1c
  - Social determinants
- Risk enhancers for intermediate-risk patients



Reference: [19] AHA PREVENT Calculator. 2023


Primary Prevention:
Assess ASCVD Risk in Each Age Group
Emphasize Adherence to Healthy Lifestyle
40-75yrs
LDL-C 70-190mg/dL
Age 0-19 y
Lifestyle to prevent or reduce ASCVD risk
Diagnosis of Familial Hypercholesterolemia→ statin
Age 20-39 y
Estimate lifetime risk
to encourage lifestyle to reduce ASCVD risk
Consider statin if family history premature ASCVD and LDL-C ≥160 mg/dL (≥4.1 mmol/L)
Age 40-75 y and LDL-C ≥70-<190 mg/dL (≥1.8-<4.9 mmol/L) without diabetes mellitus
10-year ASCVD risk percent begins risk discussion
LDL-C ≥190 mg/dL (≥4.9 mmol/L)
No risk assessment; High-intensity statin
(Class I)
Diabetes mellitus and age 40-75 y
Moderate-intensity statin
(Class I)
Diabetes mellitus and age 40-75 y
Risk assessment to consider high-intensity statin
(Class IIa)
Age >75 y
Clinical assessment, Risk discussion
1. Calculate Risk
<5%
"Low Risk"
5% - <7.5%
"Borderline Risk"
≥7.5% - <20%
"Intermediate Risk"
≥20%
"High Risk"
sk Discussion/
Risk Enhancers
Risk discussion:
Emphasize lifestyle to reduce risk factors
(Class I)
Risk discussion:
If risk enhancers present then risk discussion regarding moderate-intensity statin therapy
(Class IIb)
Risk discussion:
If risk estimate + risk enhancers favor statin, initiate moderate-intensity statin to reduce LDL-C by 30% - 49%
(Class I)
Risk discussion:
Initiate statin to reduce LDL-C ≥50%
(Class I)
3. CAC Testing
(7.5-20%)
If risk decision is uncertain:
Consider measuring CAC in selected adults:
CAC = zero (lowers risk; consider no statin, unless diabetes, family history of premature CHD, or cigarette smoking are present)
CAC = 1-99 favors statin (especially after age 55)
CAC = 100+ and/or ≥75th percentile, initiate statin therapy
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Risk Enhancing Factors

Family History

Premature ASCVD (male <55, female <65) in a first-degree relative.

Metabolic Syndrome

A cluster of conditions including abdominal obesity, elevated triglycerides, low HDL, hypertension, and elevated glucose.

Chronic Inflammatory Conditions

Conditions like rheumatoid arthritis, psoriasis, or HIV increase systemic inflammation, contributing to ASCVD risk.

Elevated Lp(a)

Lipoprotein(a) is a genetically determined lipid particle, an independent risk factor for ASCVD and aortic stenosis.

References: [20] AHA. Risk-Enhancing Factors for ASCVD.

A Frustrated Patient

A PATIENT'S DILEMMA

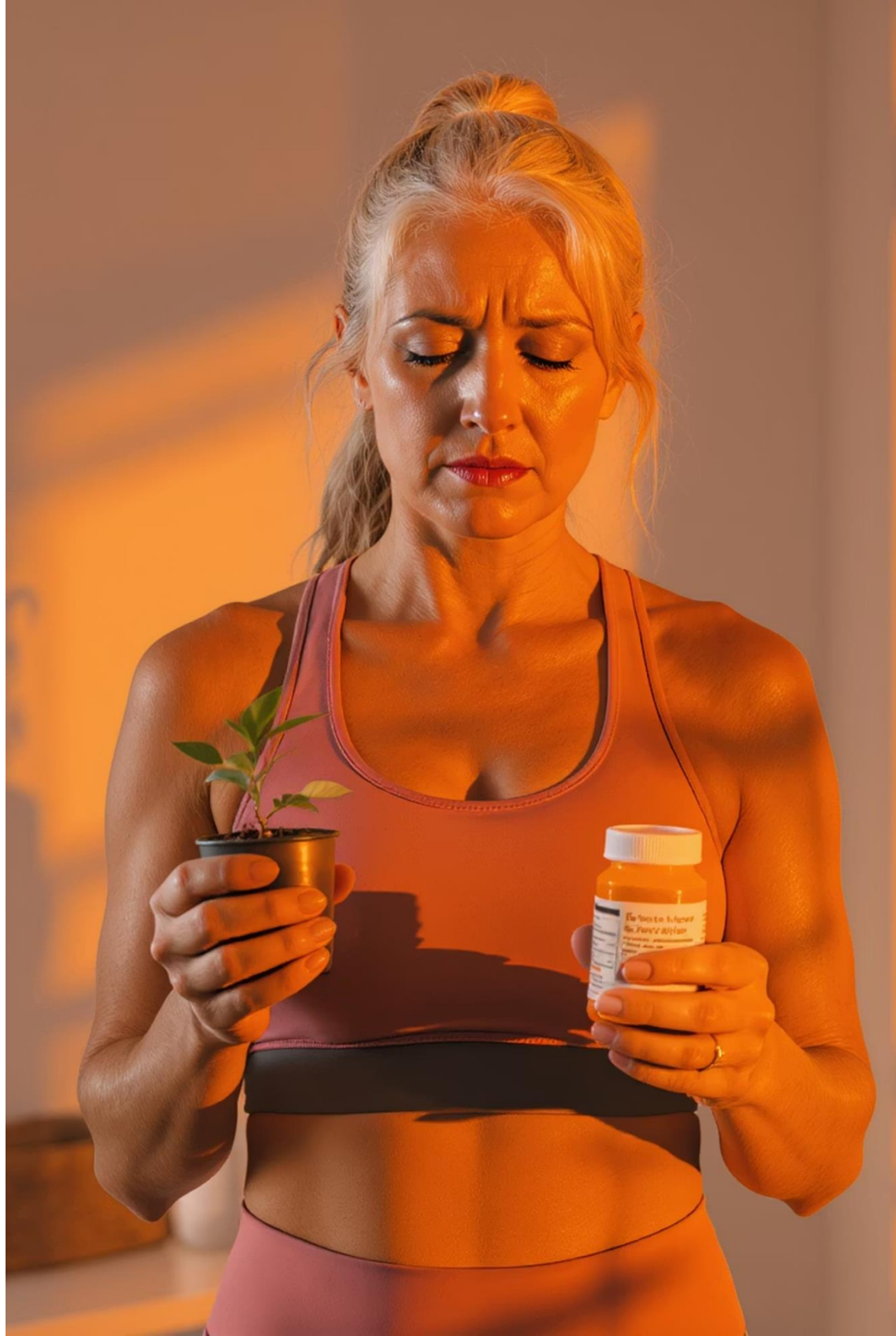
Patient Profile

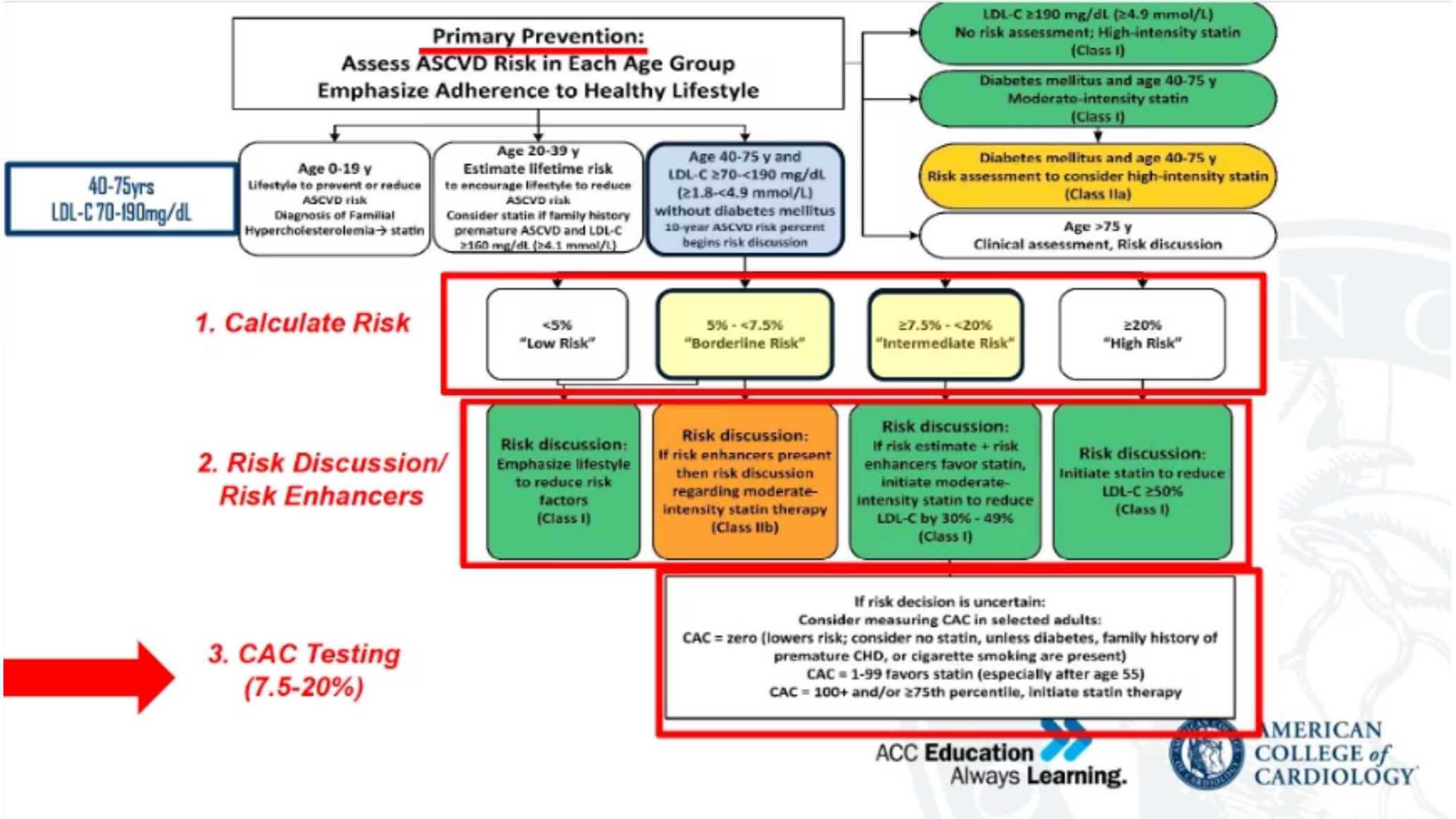
- 63-year-old female
- Yoga enthusiast
- Whole food plant-based eater

Key Lab Results

- Total Cholesterol: 267 mg/dL
- Triglycerides: 107 mg/dL
- HDL: 61 mg/dL
- LDL Calc: 185 mg/dL
- CRP: <2 mg/

Despite a seemingly healthy lifestyle faced with significantly elevated LDL-C and was hesitant about starting medication.





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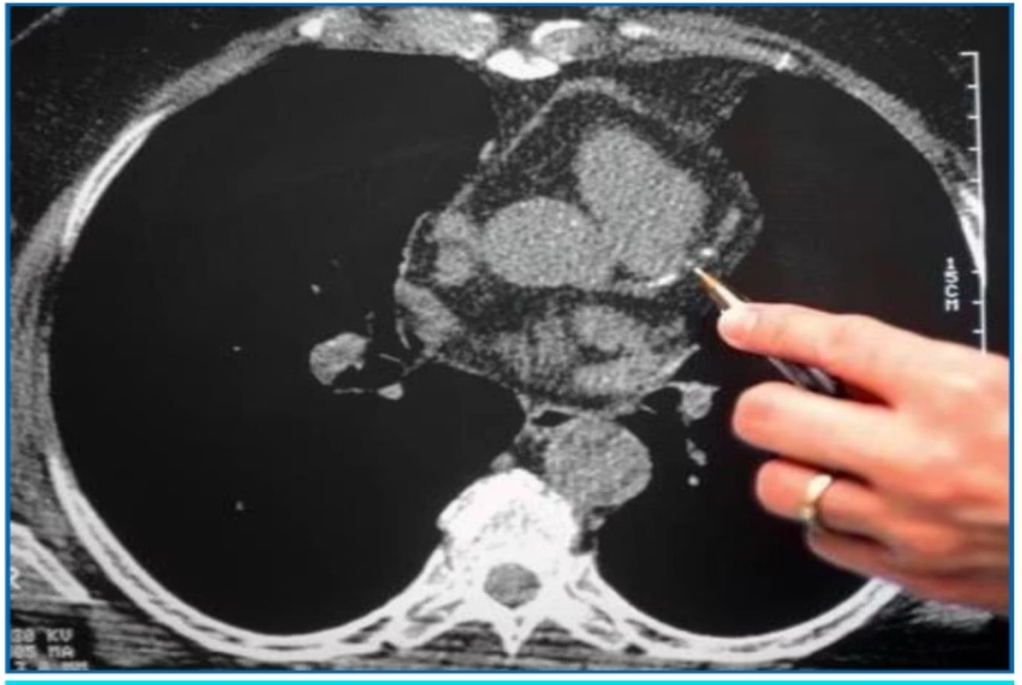
CAC Testing



Imaging of coronary artery calcification as a specific sign of atherosclerosis

- Rapid CT scan of heart
- Does not require contrast
- Inexpensive
- Widely Accessible
- 3-5 minutes
- Low radiation dose

- Disease-based imaging
- Risk integrator

Coronary Artery Calcium (CAC) Scoring

Agatston Score Interpretation

- 0: No identifiable plaque
- 1-99: Mild plaque
- 100-399: Moderate plaque
- 400+: Severe plaque

Clinical Utility

- Risk reclassification tool
- Especially useful for intermediate risk
- Zero score has excellent negative predictive value

Why use Coronary Artery Calcium Score

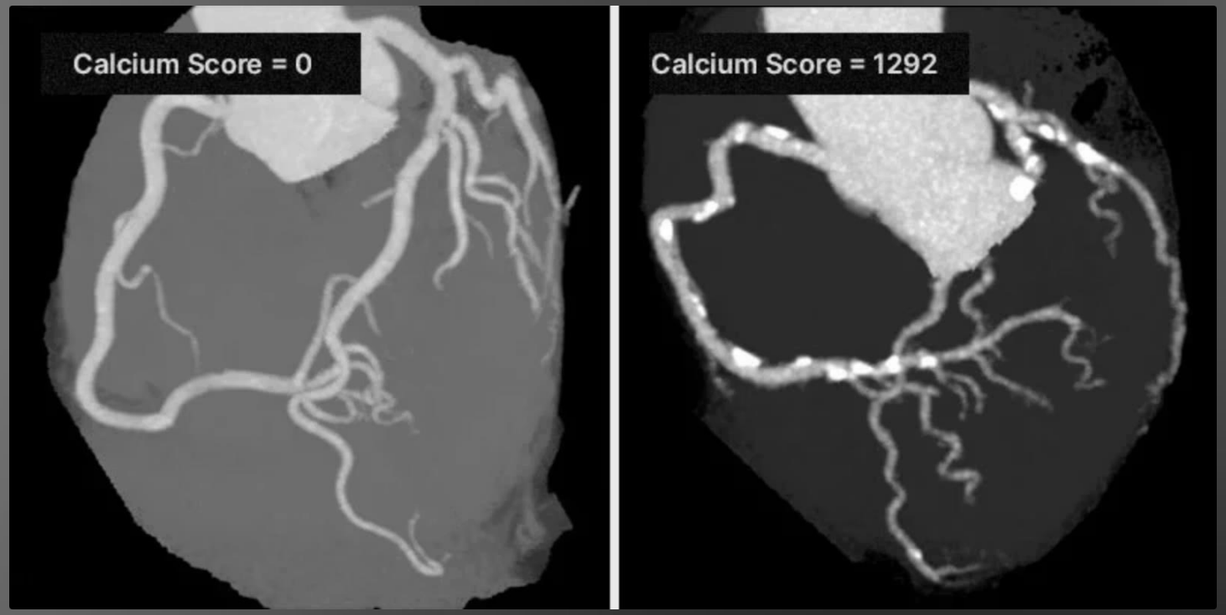
Unveiling Hidden Risk

Refining Risk Prediction

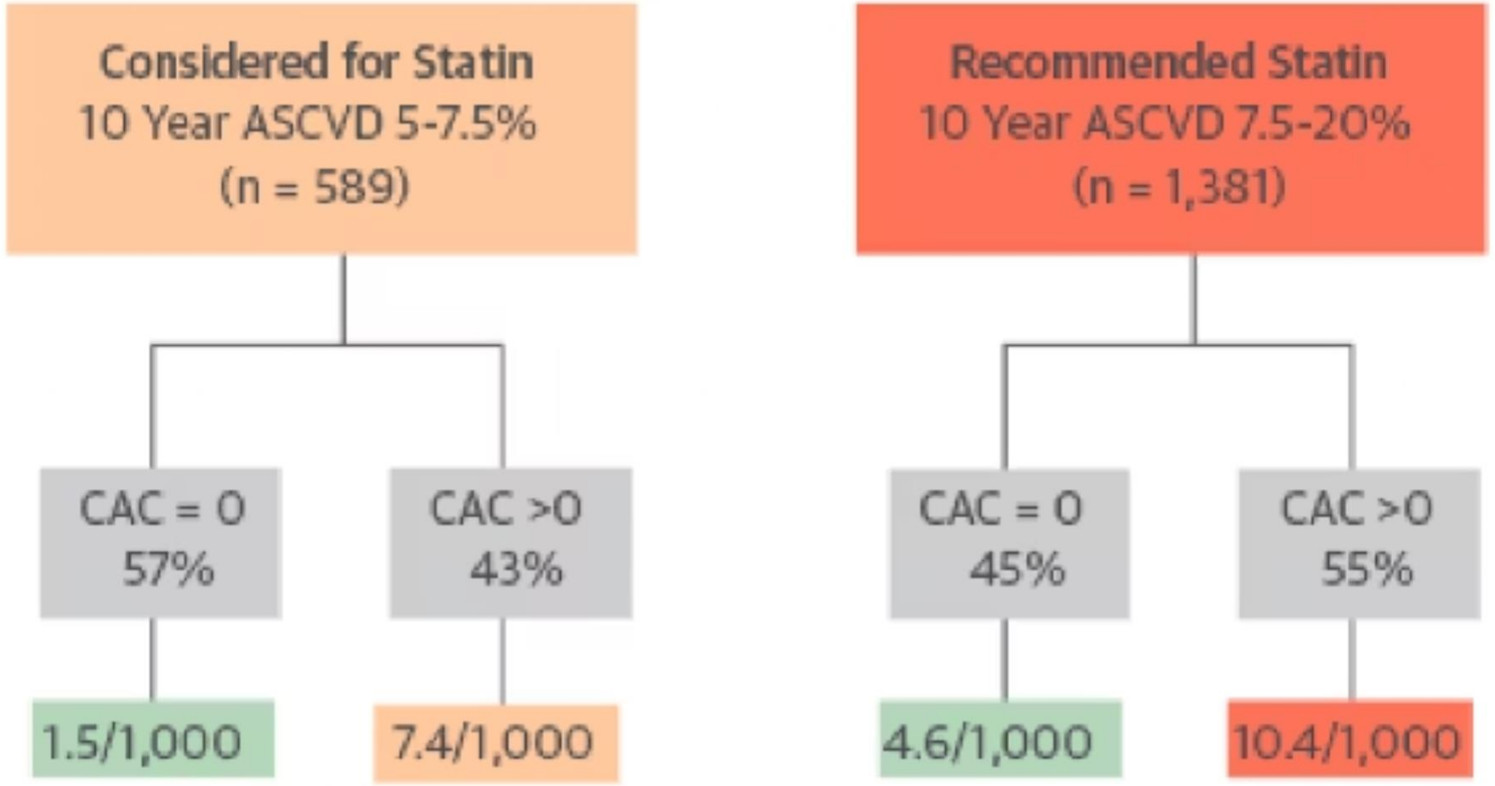
Guiding Treatment Decisions

Empowering Patient Engagement

A CAC score of zero (CAC=0) suggests a very low risk of ASCVD events for the next 5-10 years, potentially allowing for de-escalation of therapy in some cases.

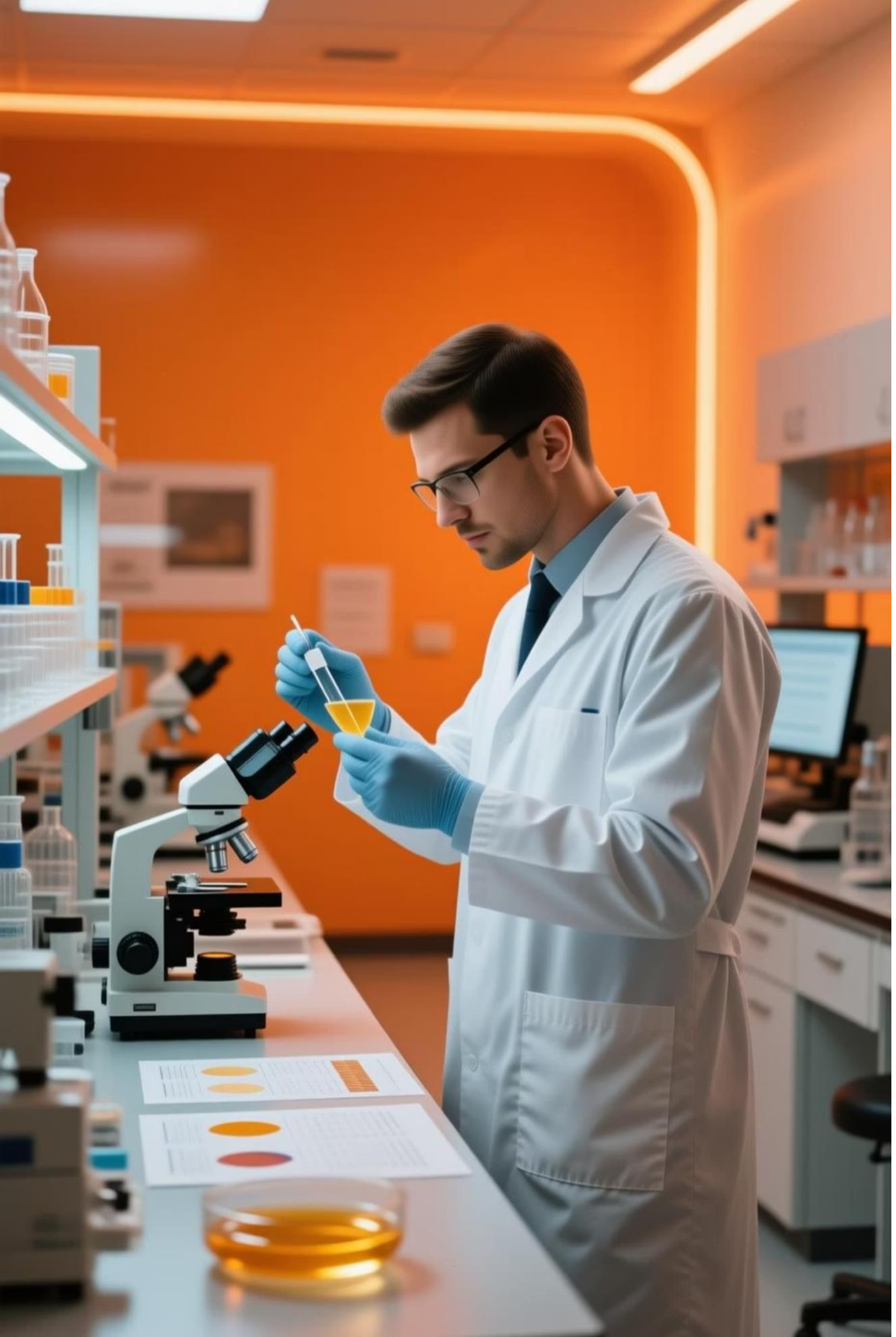


CAC 0 and Statin Eligibility: MESA Study



~50% of statin candidates have CAC=0; risk less than treatment threshold

#3 reason there is confusion about cholesterol lab values don't tell the whole story



Advanced Lipid Testing

The Hidden Risks

What Your Standard Lab Tests Didn't Check

LDL Particle Number (LDL-P)

- Superior cardiovascular risk prediction vs. LDL-C
- Discordance analysis: when LDL-C and LDL-P disagree
- Better reflects actual atherogenic burden

References: [22] Dovepress RRCC, [23] PubMed 23386699

LDL-C vs. LDL-P: A Critical Distinction

LDL-C (Cholesterol Content)

Measures the amount of cholesterol contained within LDL particles—

LDL-P (Particle Number)

Measures the total number of LDL particles present

Apolipoprotein B (ApoB)

Direct Measure

One ApoB molecule per atherogenic particle

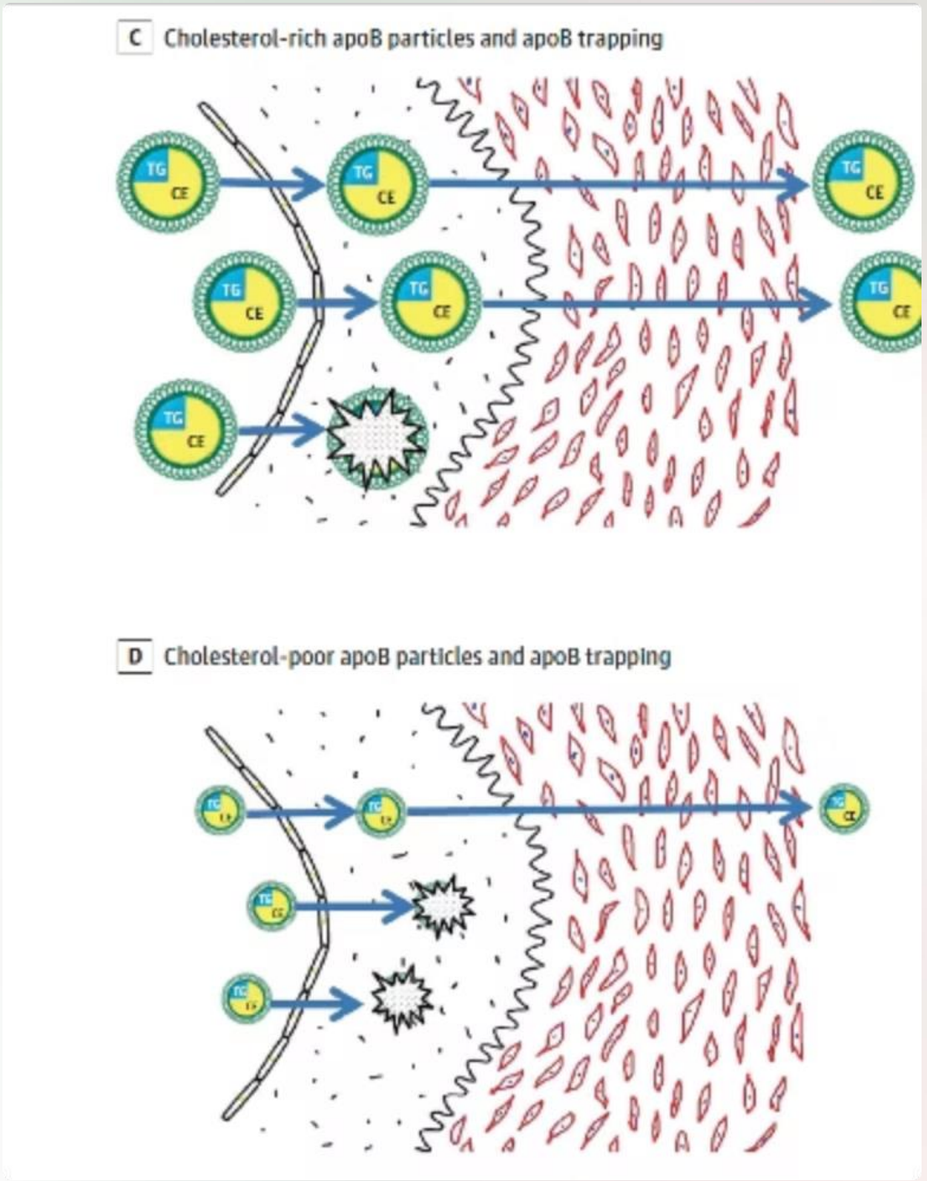
Superior Predictor

Better than LDL-C in clinical trials

Clinical Advantage

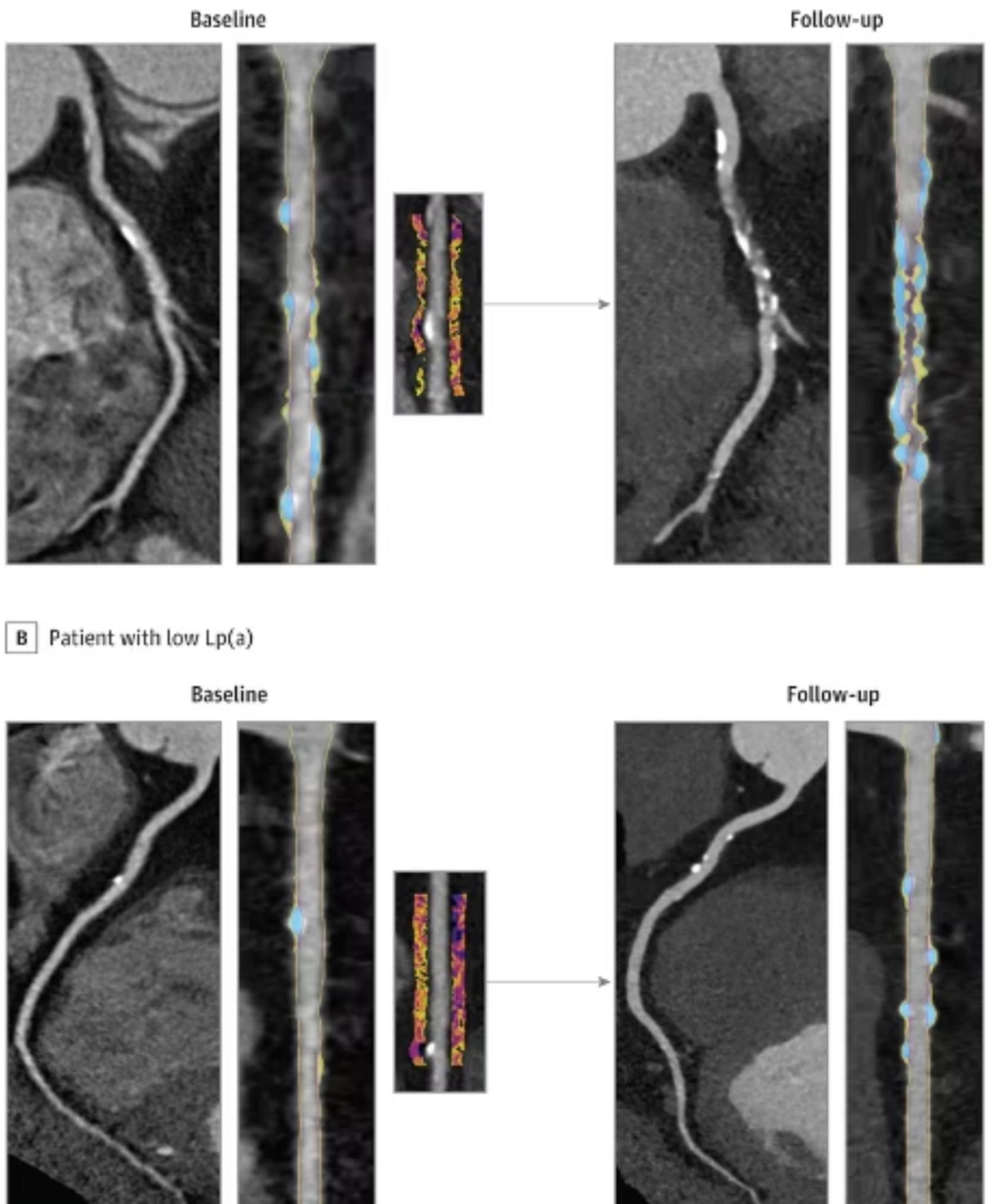
Accurate in non-fasting state

References: [24] JAMA Cardiology 2786334, [25] JAMA Cardiology 2786333



ApoB is associated dose-dependently with an increased risk of MI and ASCVD in women and men. This finding demonstrates that apoB provides important predictive value beyond LDL-C across the entire LDL-C spectrum.

Higher Lp(a) levels are associated with increased progression of coronary plaque burden and increased presence of low-density noncalcified plaque and peri-coronary adipose tissue inflammation.



Lipoprotein(a) [Lp(a)]

6-7x

Atherogenic Potential

More atherogenic than LDL per particle

20%

Population With Elevated Levels

Genetically determined, largely treatment-resistant

50 mg/dL

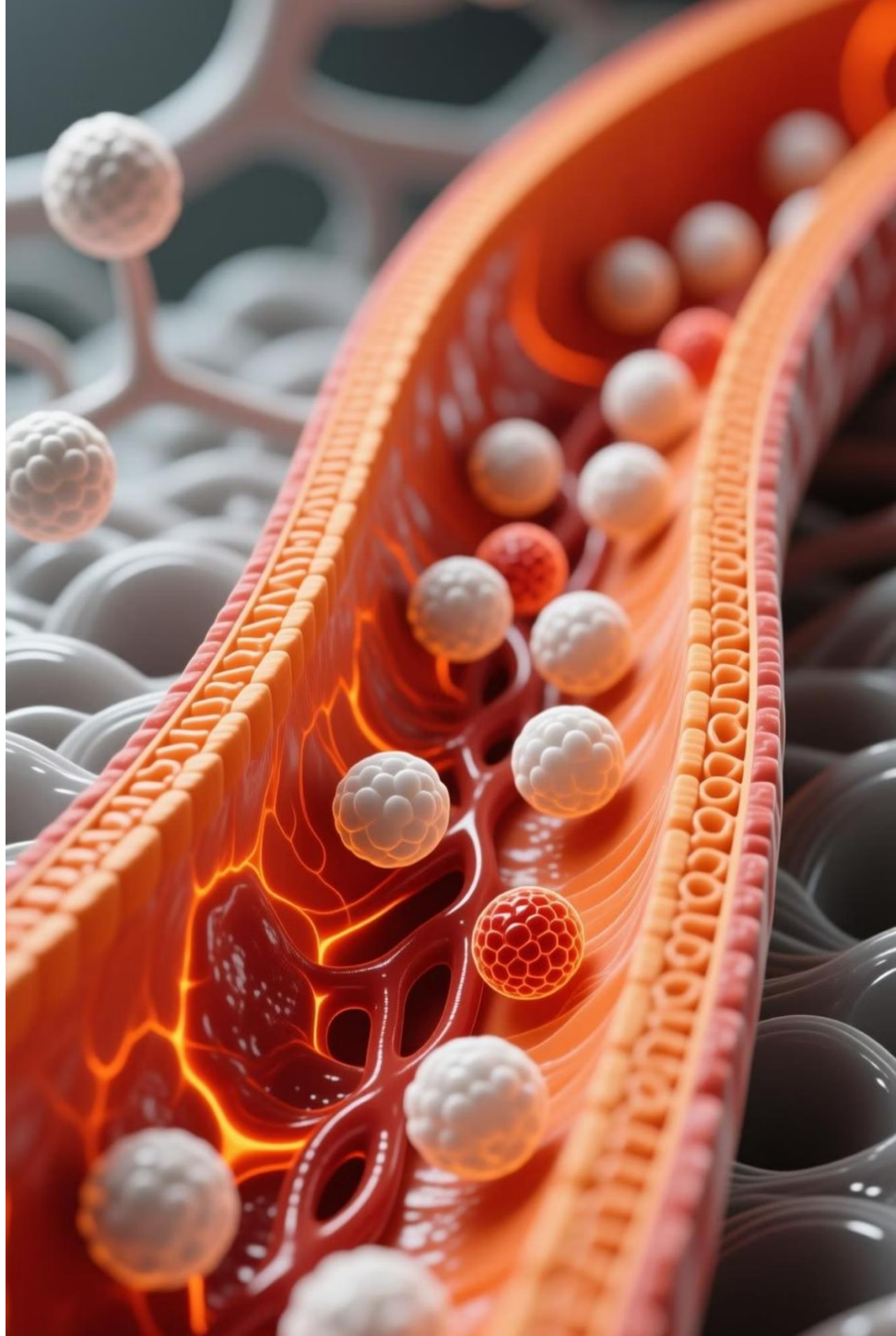
Risk Threshold

Consider screening in patients with premature ASCVD

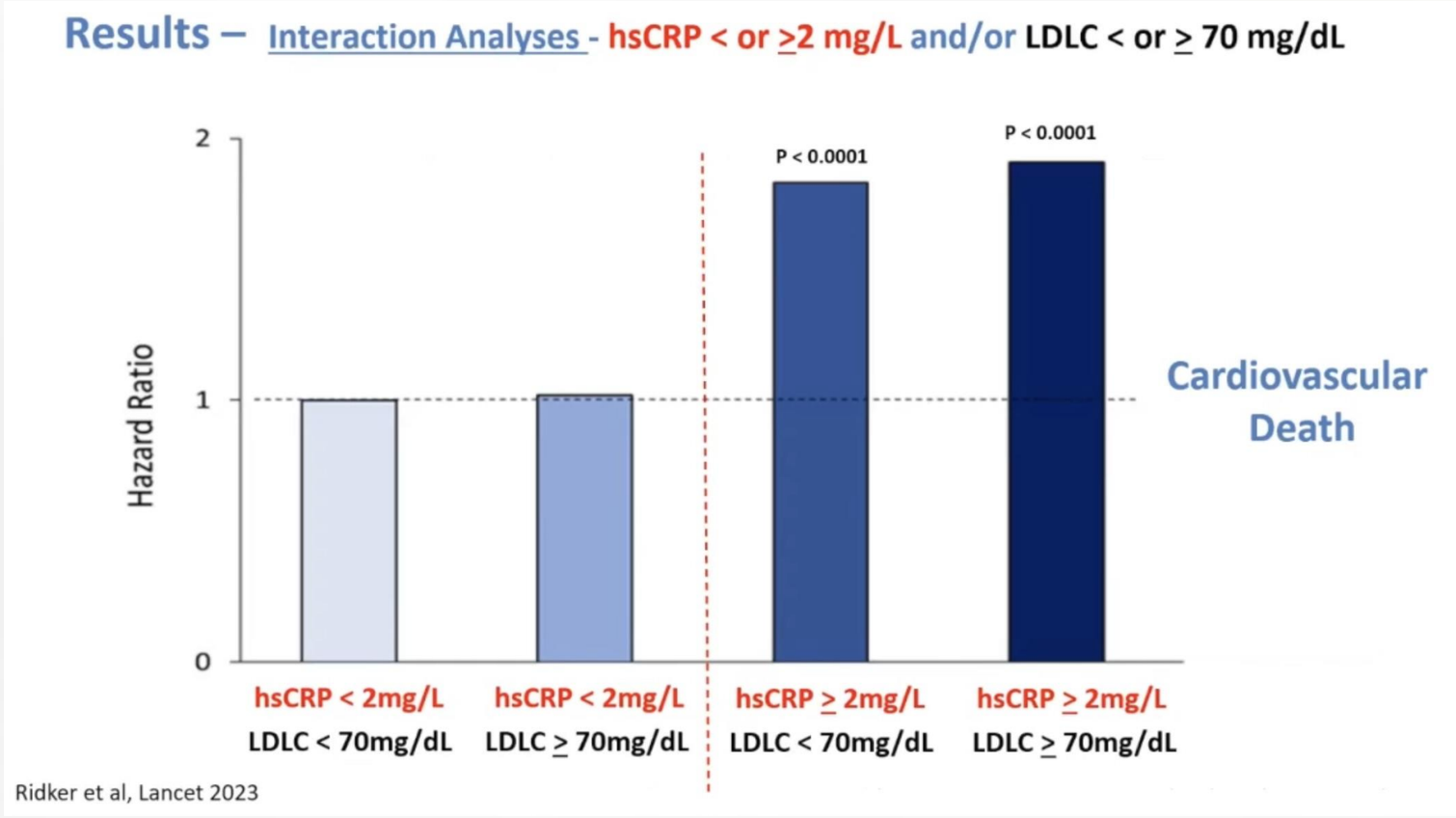
References: [26] Lipid World Biomedcentral, [27] PubMed 38233012, [28] JACC 2023

Inflammatory Markers

The Fire Within



Inflammation, as measured by markers like C-reactive protein (CRP), also contributes to residual cardiovascular risk.



4th reason cholesterol hesitancy exists: Statins

Social Media's Impact on Statin Attitudes

76%

Misleading Tweets

Percentage of statin-related tweets containing misleading information [1]

43.4%

Bot Negativity

Increase in negative sentiment from 27.8% to 43.4% in bot accounts [3]

38.4%

Human Negativity

Increase in negative sentiment from 30.9% to 38.4% in human accounts [3]

References: [1] Rodriguez F, et al. JAMA Network Open. 2023, [3] Slavin SJ, et al. J Am Heart Assoc. 2024

Statin Therapy

Evidence-Based Statin Use in Primary Prevention

20-25%

Relative Risk Reduction

For major coronary events [32][17]

Absolute benefit varies by baseline risk

22%

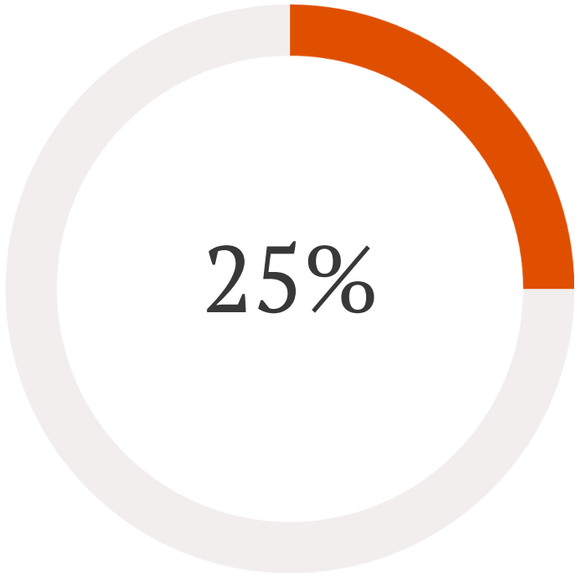
CHD Event Reduction

In women with appropriate statin therapy [17]

Similar relative risk reduction in men and women

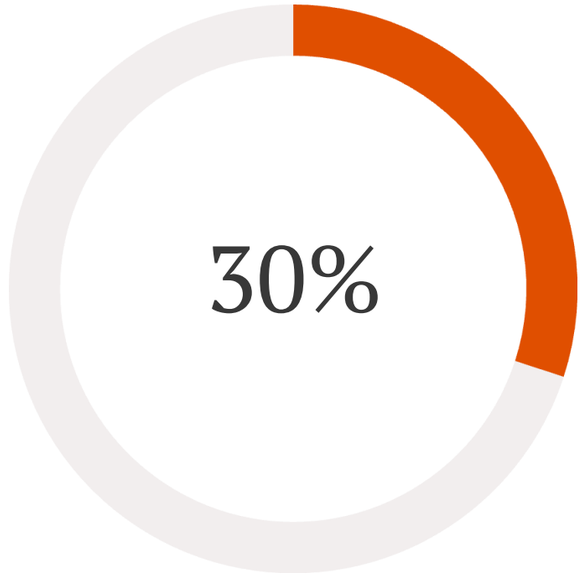
References: [17] PMC. 2012, [32] PubMed. 2013

Evidence-Based Statin Use in Secondary Prevention



Risk Reduction

Per 38.6 mg/dL LDL-C reduction [16]



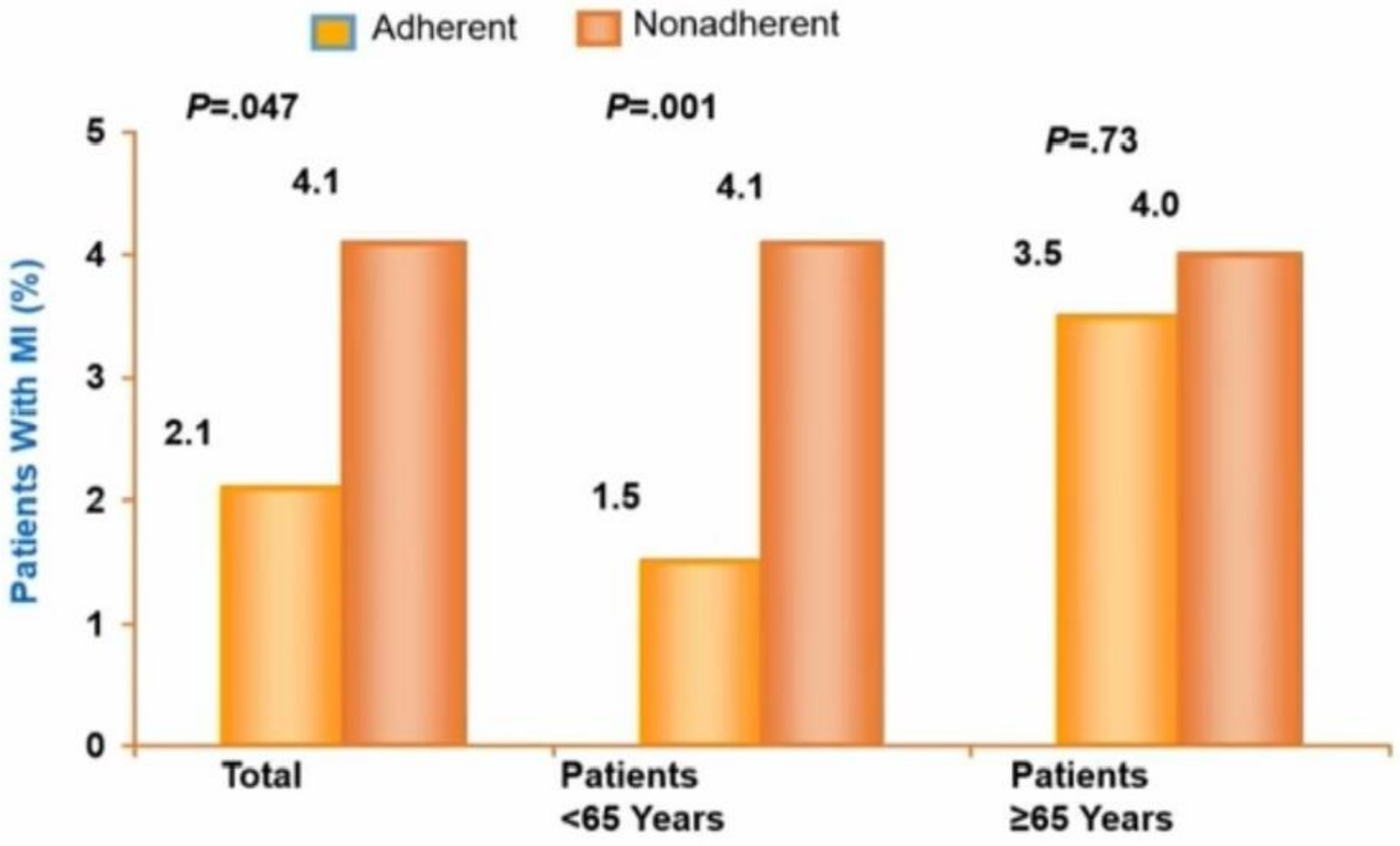
Mortality Benefit

Clear survival advantage in high-risk patients

Intensive vs. moderate-intensity therapy beneficial in established ASCVD

References: [16] PMC 3709236, [32] PubMed. 2013

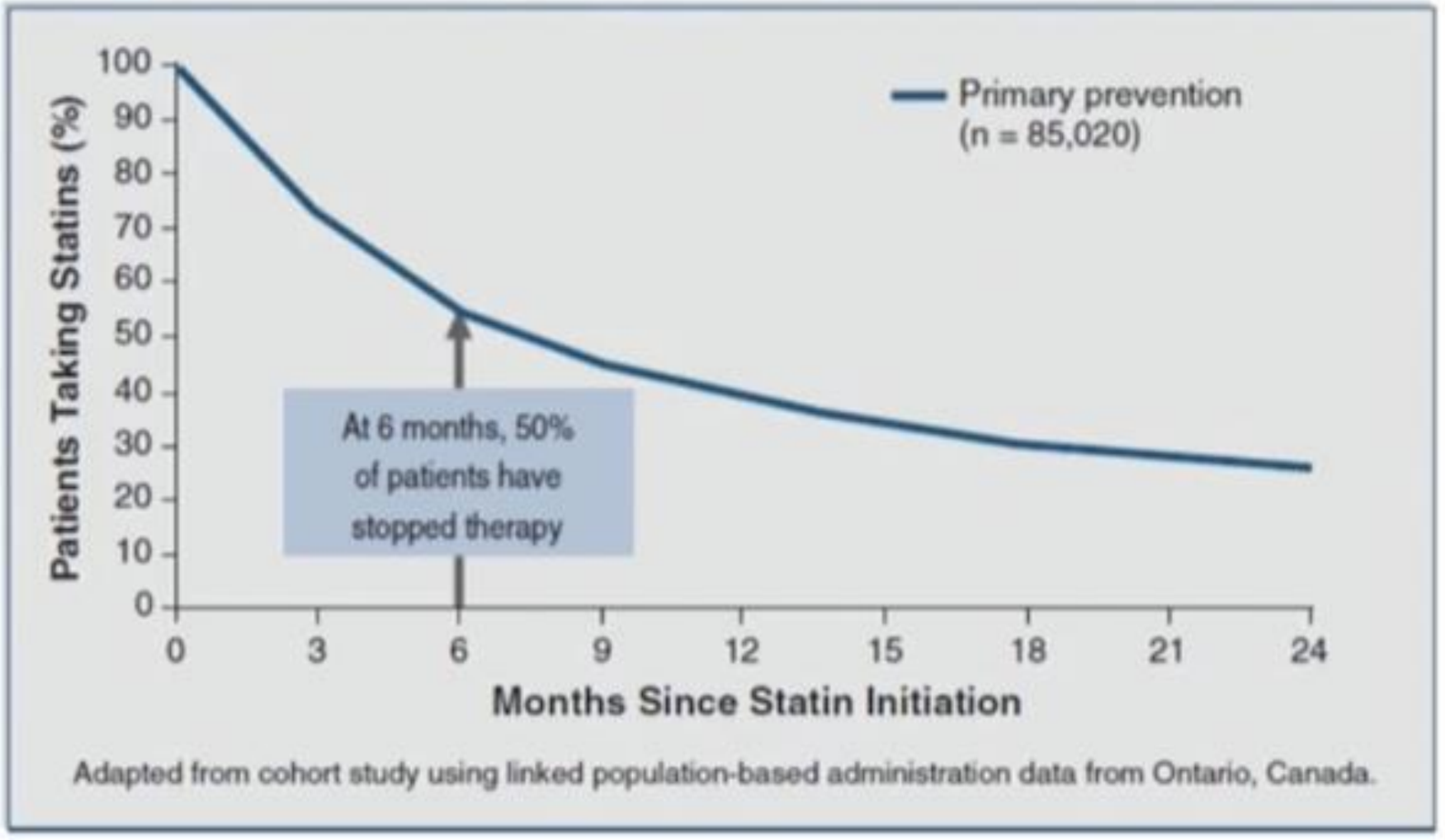
Patients Non-adherent to Statin Therapy Are Twice as Likely to Experience Subsequent MI



Adherence defined as fill frequency ≥80% (n=661).
Nonadherence defined as fill frequency ≤60% (n=395).

Non adherence to Statin Treatment begins early

Adherence continues to drop over time, particularly when treating the asymptomatic patient



Adapted from cohort study using linked population-based administration data from Ontario, Canada (N=85,020). Jackevicius et al.

Statin Intolerance: Myth vs. Reality

The Truth About Statin Side Effects

- True intolerance rates: 5-10%
- Perceived rates much higher due to nocebo effect
- Rechallenge strategies often successful
- Non-statin options: ezetimibe, PCSK9 inhibitors

In search of other options

The Supplement Secret

Evidence-Based “Natural Options”

Red Yeast Rice (RYR)

Background

Ancient Chinese medicine dating to 800 AD4

Contains multiple monacolins, including monacolin K (identical to lovastatin)

Mechanism

Inhibits HMG-CoA reductase (statin-like action)

Efficacy

LDL-C ↓ 15-25% (comparable to low-dose statin)6

China Coronary Secondary Prevention Study: 45% ↓ in CV events

Dose

1,200-2,400 mg daily

Red Yeast Rice: Safety & Considerations

Side Effects

Myalgia (5-10%)

Elevated liver enzymes (rare)

Similar to low-dose statins but potentially fewer

Precautions

Contraindicated with statins

Avoid in pregnancy/lactation

Drug interactions similar to statins

Quality Concerns

Monacolin content varies widely between products9

Some contain citrinin (nephrotoxic)

Cost: $20-40/month (not covered by insurance)

Niacin (Vitamin B3)

Mechanism

Inhibits hepatic DGAT2 enzyme10

Decreases VLDL synthesis

Affects ApoA1 & Lp(a)

Lipid Effects

LDL-C ↓ 15-20%

HDL-C ↑ 15-30%

TG ↓ 20-40%

Lp(a) ↓ 20-30%11

⊗ **Key Point:** Niacinamide does NOT affect lipid levels despite being a form of vitamin B3

Dose

500-1,000 mg twice daily (extended-release)

Niacin: Evidence & Limitations

1

Historical Success

Coronary Drug Project (1975): 27% ↓ in MI

2

Disappointments

AIM-HIGH & HPS2-THRIVE: No CV benefit when added to statins

3

Current Position

Consider for statin-intolerant patients or for Lp(a) reduction

Safety Concerns

Flushing (75-90%), hepatotoxicity, hyperglycemia, gout

Monitor: LFTs, uric acid, glucose

Cost: $10-200/month depending on formulation

Berberine

Background

Alkaloid from plants used in traditional Chinese medicine

Mechanism

PCSK9 inhibition

AMPK activation

Intestinal microbiome modulation

Efficacy

LDL-C ↓ 15-30%

TG ↓ 20-35%

No CV outcome studies

Dose & Safety

500 mg 2-3 times daily

Side effects: GI issues, constipation

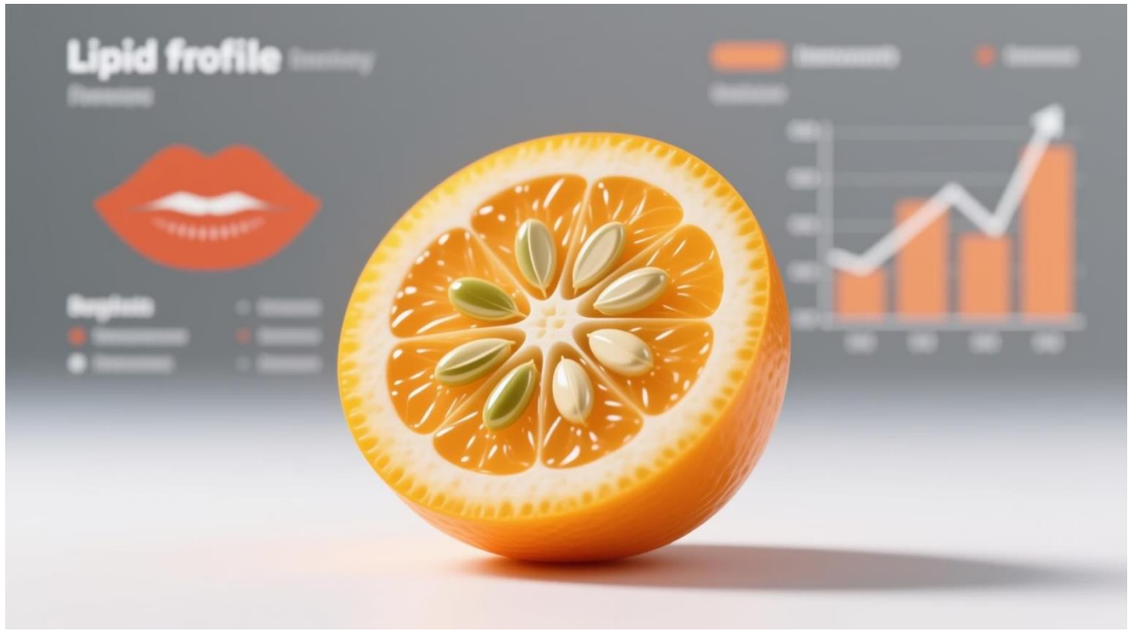
Interaction risk with CYP3A4 medications20

Cost: $15-30/month

Bergamot

Background

Citrus fruit extract from Calabria, Italy



Mechanism

Contains flavonoids

AMPK activation

HMG-CoA reductase inhibition

Evidence & Considerations

Small studies show LDL-C ↓ 15-40%

No CV outcome studies

Dose: 500-1500 mg daily

Few side effects reported

Cost: $25-45/month

Amla (Indian Gooseberry)

Clinical Results

- 17% total cholesterol reduction
- 21% LDL-C reduction
- Comparable efficacy to 20mg simvastatin

Key Benefits

- Rich in galloellagitannins
- Anti-inflammatory properties
- Antioxidant effects
- Generally well-tolerated

Mechanism of Action

- Inhibits HMG-CoA reductase, similar to statins, reducing cholesterol synthesis.
- Enhances the activity of lipoprotein lipase, improving triglyceride clearance.
- Increases bile acid excretion, promoting cholesterol elimination from the body.
- Provides antioxidant effects, protecting LDL from oxidation.

Reference: [36] Nutritional Outlook

Garlic

Active Compounds

Allicin and organosulfur compounds

Lipid Effects

LDL-C ↓ 5–15%

Inconsistent results between studies

Vascular Benefits

↓ Coronary calcium progression

Improved endothelial function

Dose: 600–1200 mg aged garlic extract daily

Side effects: Odor, GI upset, bleeding risk

Cost: $10–20/month

Clinical Recommendations

Verify Quality

Look for **USP, NSF, or ConsumerLab** verification

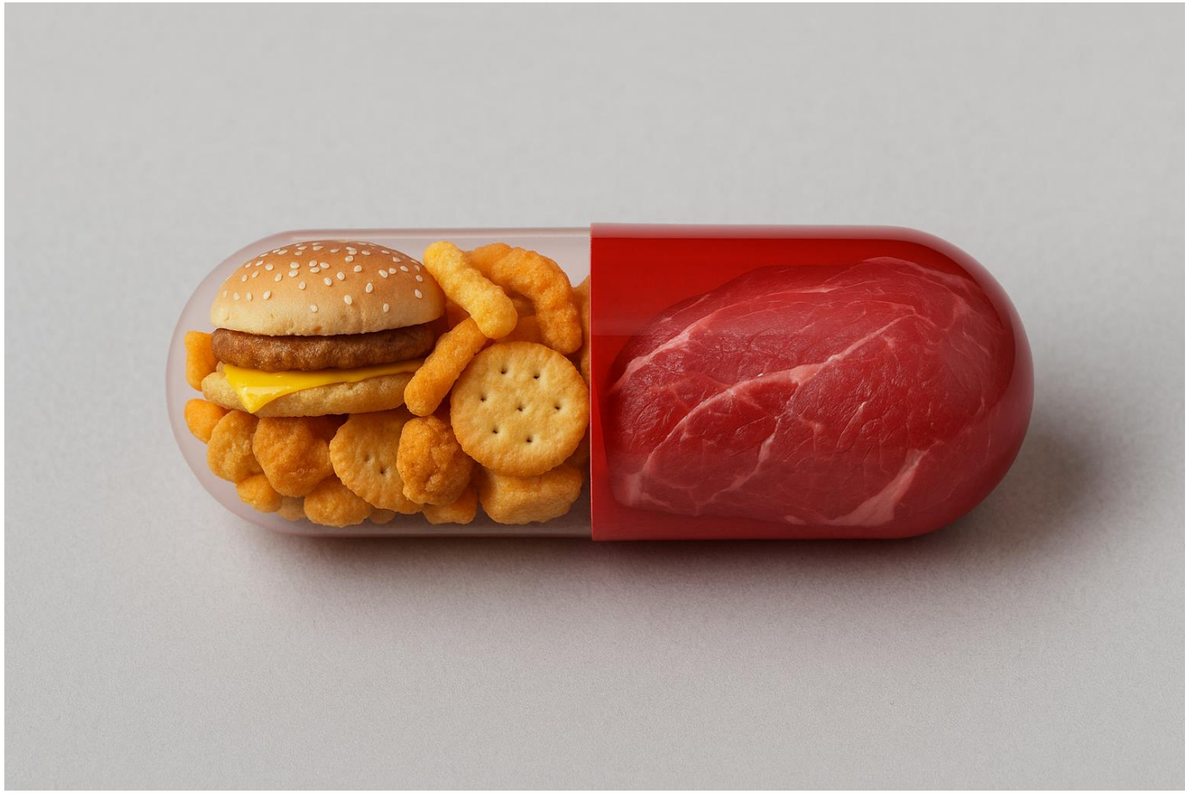
Monitor Effects

Check lipids 8-12 weeks after starting

Consider Evidence Hierarchy

1. Red yeast rice
2. Niacin (selected patients)
3. Berberine

Full citations available at: clinicalreference.org/lipid-supplements

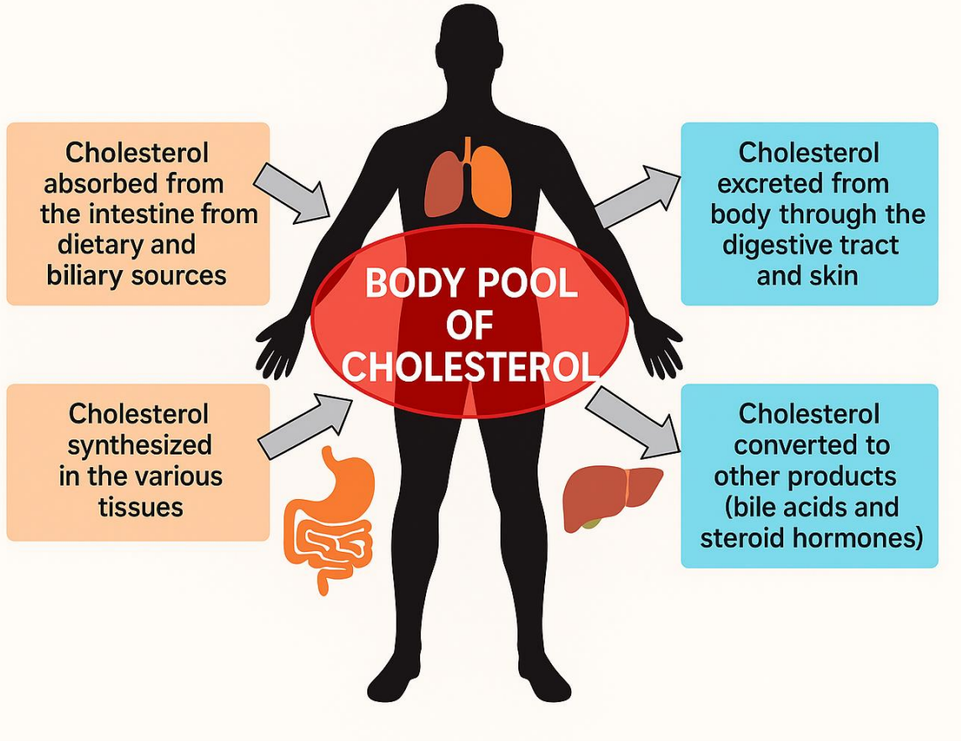


“The food you eat can be
……the slowest
form of poison”

THE DIETARY CHOLESTEROL CONTROVERSY

How Much Does Dietary Cholesterol Matter?

- Humans typically absorb only approximately 50% of dietary cholesterol ***(range: 25-75%)***
- Individual ***responses to dietary cholesterol vary*** significantly based on genetics
- ***Saturated fat*** ***has a more significant impact on blood cholesterol than dietary cholesterol***



THE SATURATED FAT DEBATE

Saturated fatty acids (SFAs) raise LDL cholesterol through multiple pathways:

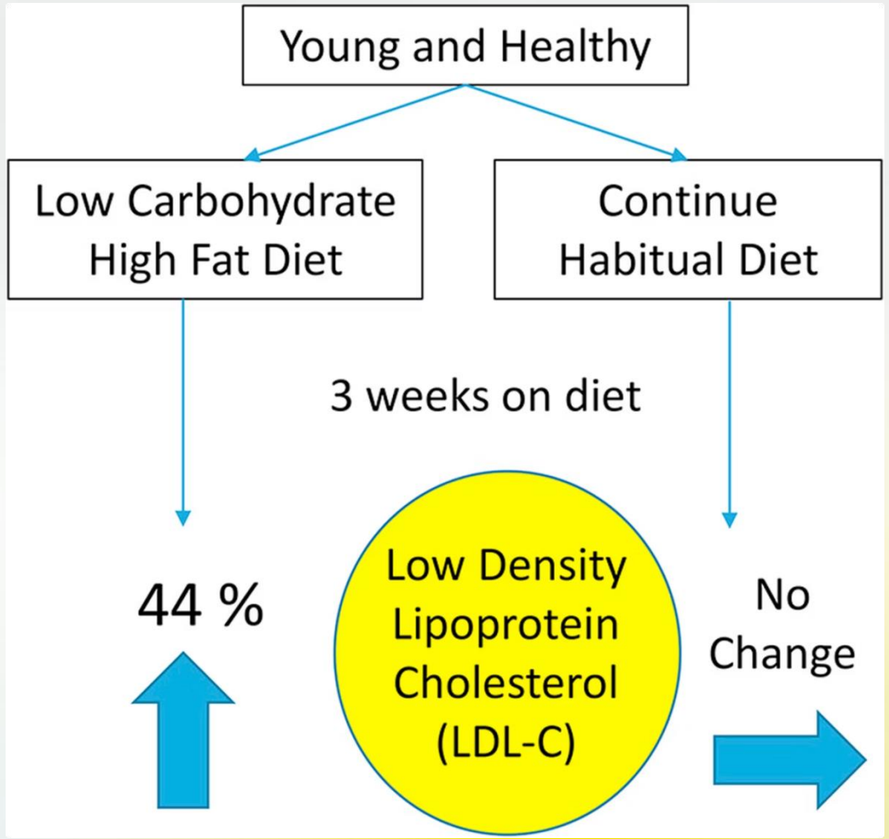
- ***Suppressing LDL receptor*** activity in the liver
- ***Reducing LDL clearance*** from the bloodstream
- ***Promoting LDL production***
- Altering LDL particle composition

These effects are particularly pronounced with longer-chain saturated fats found in animal products.

Evidence from Controlled Studies

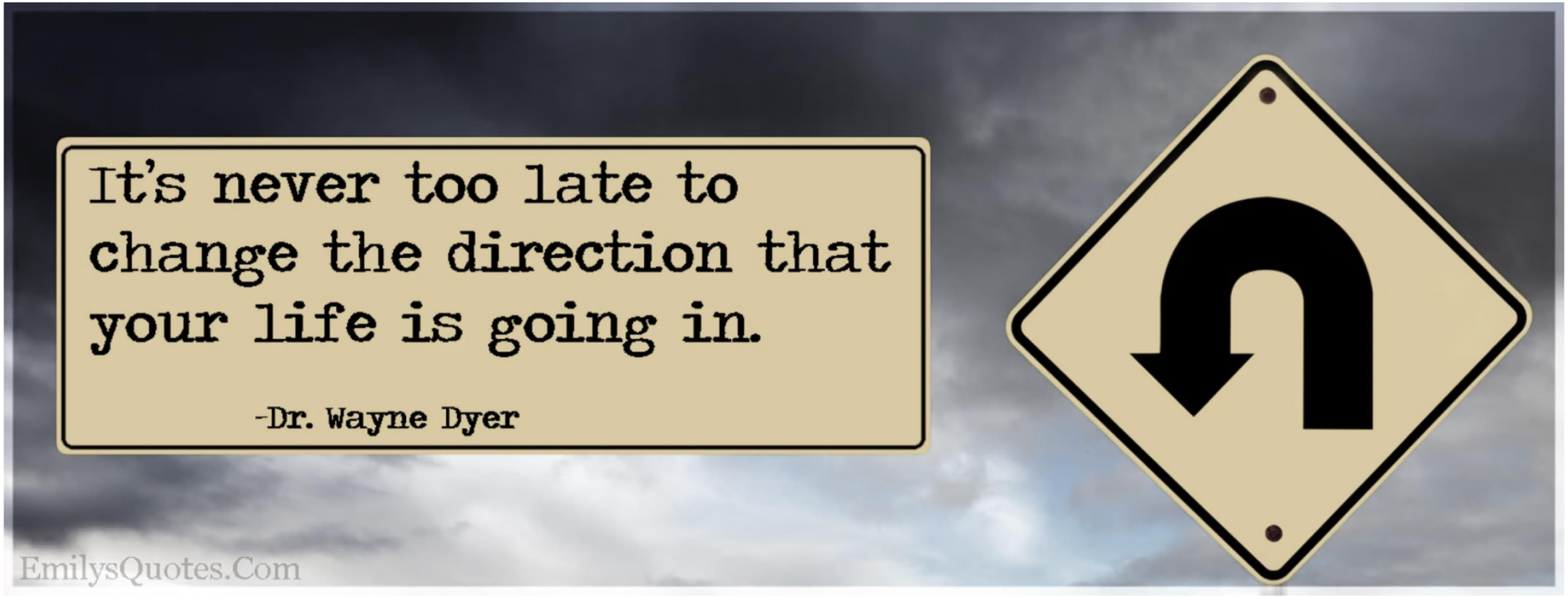
- A 2015 JAMA study found replacing butter with plant-based oils cut premature death risk by 17%
- Clinical trials consistently show that replacing saturated fat with polyunsaturated fat ***reduces LDL-C by approximately 10 mg/dL for each 5% energy substitution***
- Low-carb, high-fat diets have been shown to ***increase apolipoprotein B levels,*** even when weight loss occurs
- Meta-analyses show each ***1% energy replacement of SFA with PUFA reduces CHD risk by approximately 2-3%***

THE LOW-CARB DIET EFFECT ON CHOLESTEROL



A randomized controlled study explored the impact of a strict Low-Carbohydrate/High-Fat (LCHF) diet (less than 20g carbs/day) on healthy adults over three weeks.

- LDL-C levels significantly **increased by 44%** in the LCHF group compared to controls.
- Other markers like **apolipoprotein B, total cholesterol, and HDL-C** also rose in the LCHF group.


It's never too late to change the direction that your life is going in.
-Dr. Wayne Dyer
EmilysQuotes.Com

Food as Medicine

The Class I Recommendation

COLUMBUS BATISTE, MD
Interventional Lifestyle Cardiologist

HEALTHY HEART NATION

Patient's name: _____ Age: _____

Address: _____ Date: _____

RX Get Selfish

S - Spiritual
E - Exercise
L - Love
F - Food
I - Intimacy
S - Sleep
H - Humor

Dr. Columbus Batiste

THE HEALTHY HEART DOC

It’s ***not just what you don’t eat***,
It’s about ***what you are eating***
for your Health!

Global Burden of Cardiovascular Disease from Dietary Factors

A comprehensive analysis revealed the significant global impact of dietary choices on cardiovascular health, varying across different socioeconomic regions.

Significant Burden

Approximately **40% of CVD mortality and Disability-Adjusted Life Years (DALYs)** are directly attributable to dietary risk factors.

Top Global Risks

Globally, the leading dietary contributors to CVD are **high-sodium intake, low whole grain intake, and insufficient legume consumption.**

Regional Variations

Specific risk factors vary by region:

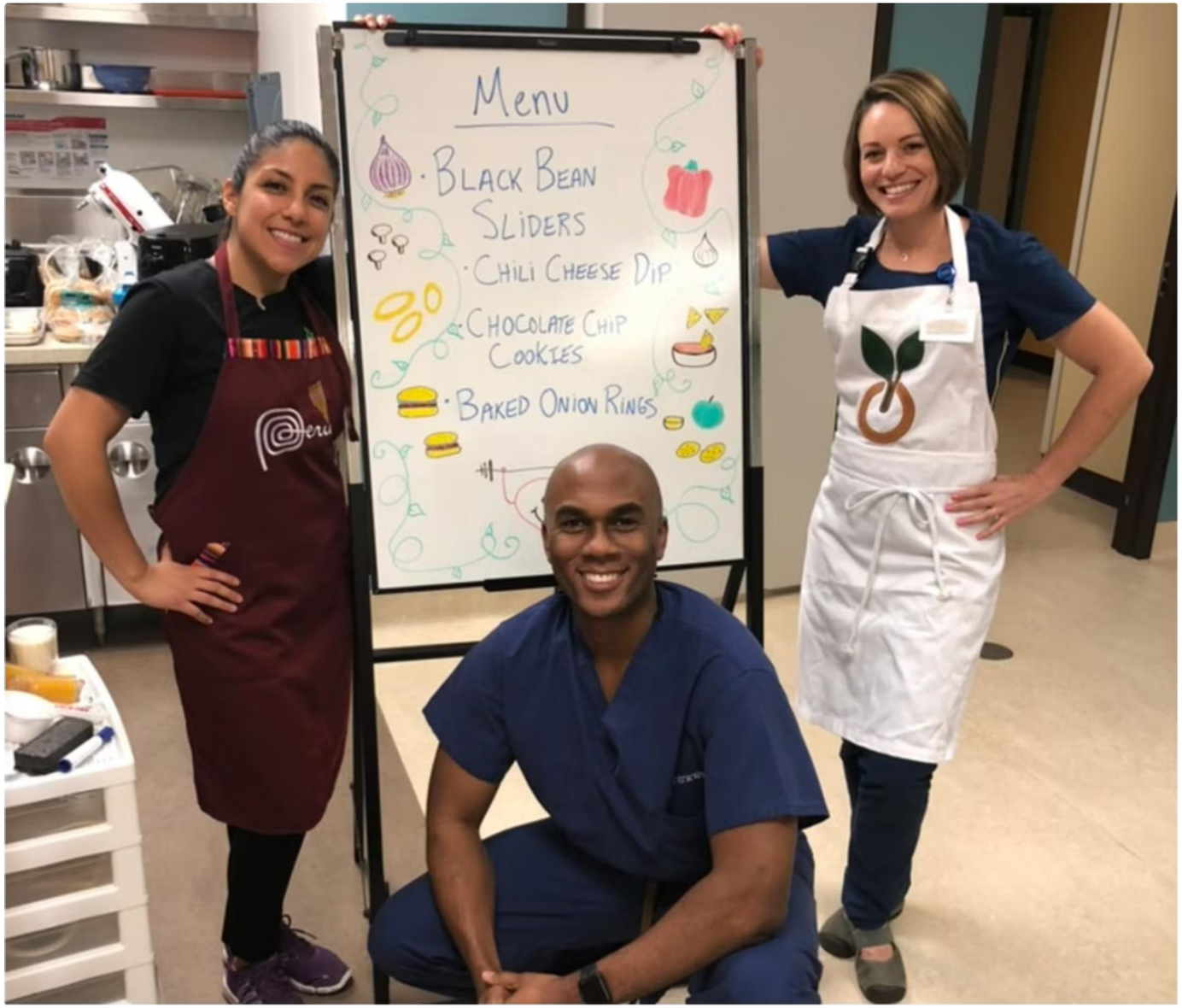
- **High SDI:** High red and processed meat intake.
- **Middle SDI:** High-sodium intake.
- **Low SDI:** Low fruit and vegetable intake.

Key Priorities

Global efforts should prioritize **reducing sodium intake** and **increasing whole grain and legume consumption** to effectively lower the CVD burden.

Reference: J Nutr. 2023 Jun;153(6):1730-1741.

CATH LAB: (***COOKING ALTERNATIVE TO HEALTH)***



Focus on What One *Is* Eating For Their Health

The Power of Plant Proteins

Impact on Mortality & Cardiovascular Health

10%

Overall Mortality

Reduction when replacing 3% of energy from animal protein with plant protein.

11-12%

Cardiovascular Mortality

Lower risk for men (11%) and women (12%) by substituting animal protein with plant protein.

21-24%

Egg Protein Replacement

Significant reduction in overall mortality risk by replacing egg protein with plant protein.

13-15%

Red Meat Replacement

Lower overall mortality risk when substituting red meat protein with plant protein.

Breakfast Bowl

TOP 3 WAYS OATMEAL LOWERS CHOLESTEROL

1. Beta-Glucan Fiber

2. Reduction in ApoB Levels

3. Antioxidant and Anti-inflammatory Effects

Oats contain unique antioxidants called avenanthramides

Clinical evidence: Meta-analyses show that consuming 3g of beta-glucan daily (about 1.5 cups of oatmeal) can reduce LDL-C by 5-10% and significantly reduce non-HDL-C and apoB.

TOP 3 WAYS BERRIES LOWER CHOLESTEROL

1. **Polyphenol Power**

inhibit cholesterol synthesis and absorption, and improve LDL receptor function.

2. **Anti-inflammatory Effects**

Berry antioxidants combat inflammation by reducing oxidative stress, decreasing inflammatory markers, and **preventing LDL oxidation.**

3. Soluble Fiber Content

Berry soluble fiber binds cholesterol and bile acids, promoting their excretion and **improving gut microbiome health**.

TOP 3 WAYS NUTS AND SEEDS LOWER CHOLESTEROL

The Heart-Healthy Power of Nuts and Seeds

1. Beneficial Fat Profile

Nuts and seeds are rich in heart-healthy fats (MUFAs, Omega-3s, ALA) that improve LDL:HDL ratios and reduce inflammation. **Plant sterols also block cholesterol absorption.**

2. Fiber and Antioxidants

Soluble fiber binds cholesterol, while antioxidants (polyphenols, Vitamin E) **prevent LDL oxidation and improve arterial health.**

3. Direct Effects on ApoB

Studies show specific benefits: walnuts significantly reduce ApoB, almonds help maintain HDL-C while lowering LDL-C, and various nuts improve LDL particle characteristics.

Clinical recommendation: Consuming 1-2 ounces (about a handful) of nuts daily can significantly improve lipid profiles without causing weight gain.

Tofu Bowl

TOP 3 WAYS SOY LOWERS CHOLESTEROL

The Benefits of Soy Protein

1. Direct Effects on Metabolism

Soy protein i**ncreases LDL receptor activity, decreases cholesterol synthesis, and promotes bile acid excretion.** Its phytosterols also compete with cholesterol for absorption.

2. Isoflavone Benefits

Soy isoflavones like genistein and daidzein improve arterial elasticity, **reduce inflammation, and may boost nitric oxide production, contributing to better blood vessel health**.

3. Displacement Effect

Replacing animal protein with soy often reduces saturated fat and cholesterol intake while increasing fiber, leading to an overall healthier dietary pattern.

> "Soy protein… decreased LDL cholesterol by 4.76 mg/dL and total cholesterol by 6.41 mg/dL compared with non-soy protein controls."
>
> — Journal of Nutrition, 2019

TOP 3 WAYS GREEN LEAFY VEGETABLES LOWER CHOLESTEROL

Green leafy vegetables like kale and spinach offer key compounds for cardiovascular health.

1. Bile Acid Binding

They bind bile acids in the digestive tract, forcing the liver to use more cholesterol for new bile acids and reducing cholesterol absorption. Steam cooking significantly enhances this effect.

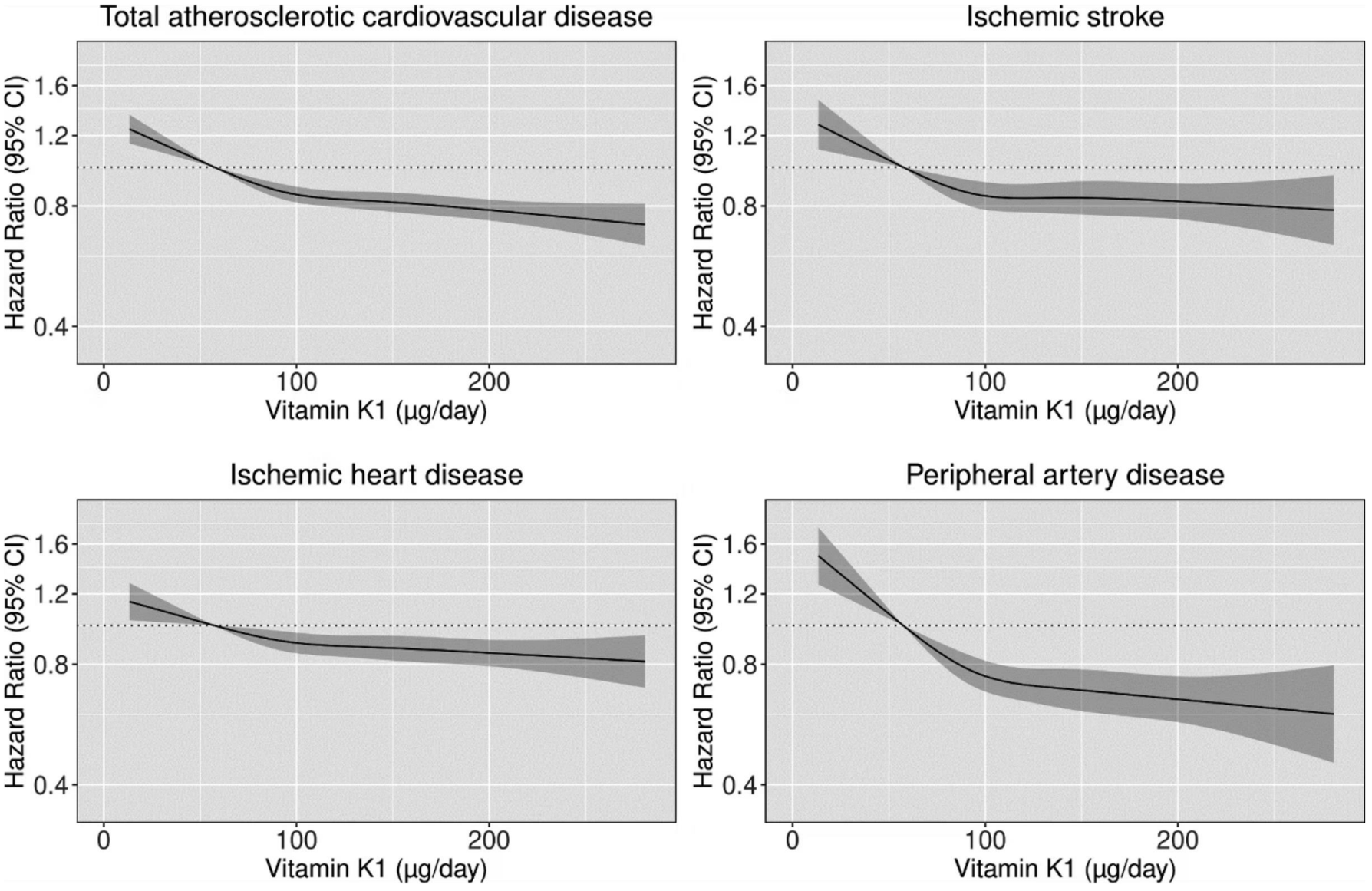
2. Lutein and Zeaxanthin

These powerful carotenoids provide antioxidant and anti-inflammatory benefits, protecting arterial tissue, preventing LDL oxidation, and improving endothelial function.

3. Nitrate Content

Rich in dietary nitrates, they convert to nitric oxide, which improves blood vessel dilation, reduces blood pressure, enhances endothelial function, and decreases blood clotting.

Clinical perspective: Incorporating these vegetables, especially steamed, is a simple way to support cholesterol management.


Total atherosclerotic cardiovascular disease
Ischemic stroke
Ischemic heart disease
Peripheral artery disease
Hazard Ratio (95% CI)
1.6
1.2
0.8
0.4
0
100
200
Vitamin K1 (µg/day)

TOP 3 WAYS BROWN RICE LOWERS CHOLESTEROL

The Whole Grain Advantage

Brown rice, retaining its bran and germ, provides essential nutrients and beneficial compounds for cholesterol management.

1. Fiber Content

Its soluble and insoluble fiber binds bile acids and reduces cholesterol absorption, also supporting gut health and weight management.

2. Unique Compounds

Brown rice offers γ-oryzanol, tocotrienols, and phytosterols that actively lower cholesterol and provide antioxidant benefits.

3. Pre-Germinated Benefits

Pre-germinated brown rice (PGBR) enhances nutrient bioavailability, showing improvements in fasting blood glucose, total cholesterol, and triglyceride levels.

> "Blood concentrations of fasting blood glucose, fructosamine, serum total cholesterol and triacylglycerol levels were favorably improved on the pre-germinated brown rice diet ($p<0.01$)."
>
> — Journal of Nutritional Science and Vitaminology, 2008

TOP 3 WAYS BEANS LOWER CHOLESTEROL

The Powerful Effects of Legumes

Legumes consistently lower cholesterol through several key mechanisms.

1. Soluble Fiber Power

Soluble fiber **binds cholesterol and bile acids**, preventing reabsorption and prompting the liver to use more cholesterol.

2. Plant Protein Effects

Legume protein reduces hepatic cholesterol synthesis, i**ncreases LDL receptor activity**, and displaces animal proteins.

3. Phytochemicals & Resistant Starch

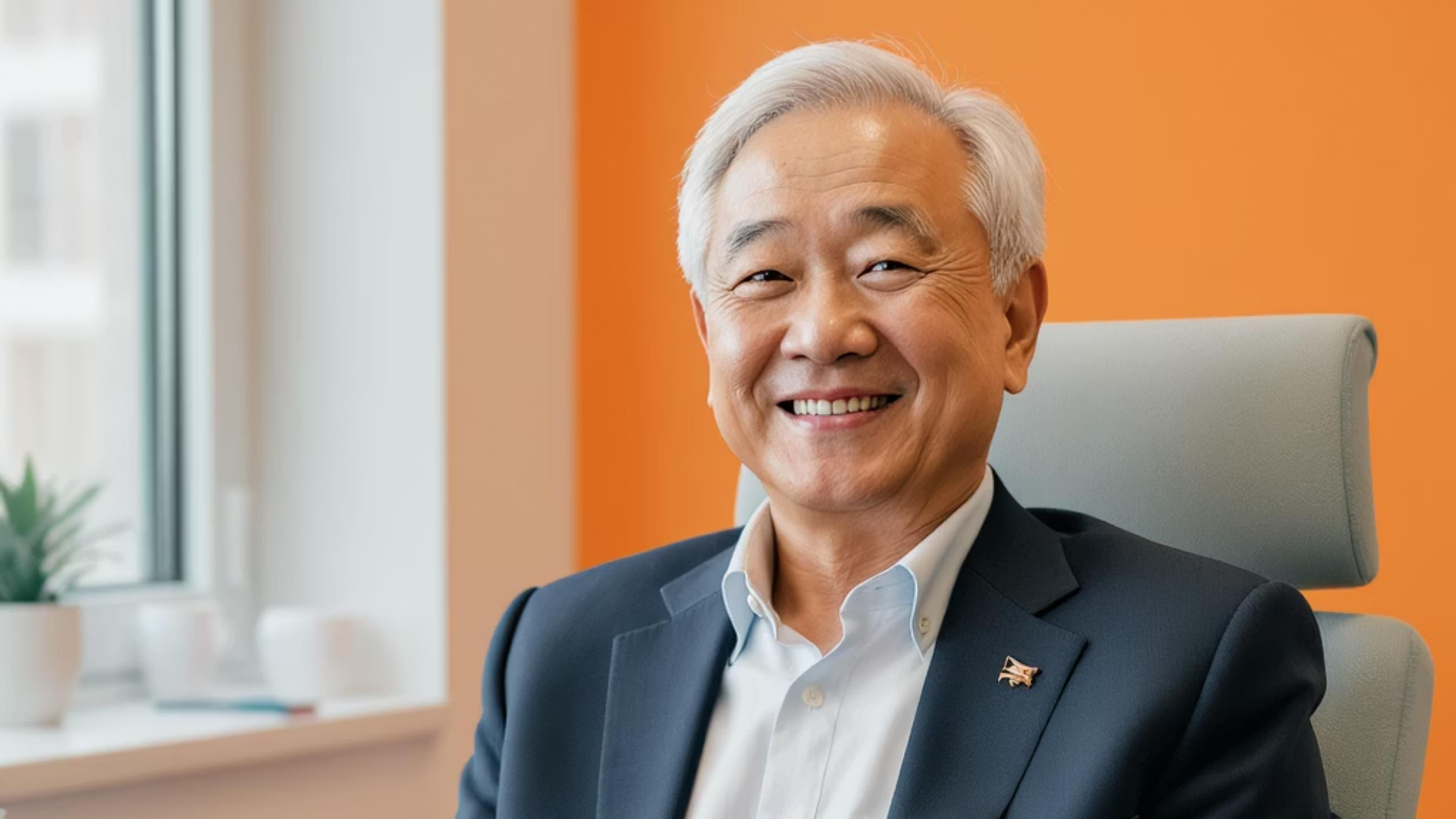
Phytosterols and saponins b**lock cholesterol absorption**, while resistant starch and antioxidants support gut health and reduce inflammation.

Allium Vegetables

Cholesterol-Lowering Effects

- Sulfur compounds inhibit cholesterol synthesis
- Organosulfur compounds reduce inflammation
- Allicin in garlic has numerous cardiovascular benefits
- Associated with reduced plaque formation

References: [44] UCLA Health 2024, [45] Brown Health 2023



Optimizing Your Dietary Approach

Good: Reduce Harmful Foods

Focus on eliminating ultra-processed items, sugary beverages, and trans fats to lay a basic foundation for health.

Better: Embrace Whole Foods

Increase intake of fruits, vegetables, whole grains, and lean proteins, moving towards a more balanced and nutritious diet.

Best: Personalized Therapeutic Diets

Adopt evidence-based patterns like the Mediterranean or Portfolio diet, tailored to individual needs and health goals for optimal outcomes.

Component	Ref Range & Units	7/15/23 7:35 AM	10/5/21 11:25 AM	7/21/20 11:53 AM	8/7/17 2:06 PM	5/31/16 7:44 AM	2/13/14 12:19 PM	3/10/08 9:12 AM
CHOLESTEROL	<=199 mg/dL	160	284 ^	333 ^	330 ^	277 ^	333 ^	302 ^ R
TRIGLYCERIDE	<=149 mg/dL	78	189 ^ CM	161 ^ CM	144 CM	439 ^ CM		76 R
Comment: Note that if triglycerides (TG) are sufficiently high, then direct LDL (if TG>1300 mg/dL) or HDL (if TG>2000 mg/dL) measurements may not be valid.								
HDL	>=40 mg/dL	66	81	78	66	47	61	72 R
LDL CALCULATED	<=99 mg/dL	79	165 ^	223 ^	235 ^	See Direct LDL		215 ^ R
CHOLESTEROL/HIGH DENSITY LIPOPROTEIN	<=3.9	2.4	3.5 CM	4.3 ^ CM	5.0 ^ R, CM	5.9 ^ R, CM		4.2 R, CM
Comment: See LabNet for more information.								
CHOLESTEROL, NON-HDL	mg/dL	94	203 CM	255 CM	264 CM	230 CM	272 R, CM	
Comment: NonHDL targets are 30 mg/dL higher than LDL targets.								

Results of Aggressive Lifestyle

KEY TAKEAWAYS FOR YOUR PATIENTS HEART HEALTH JOURNEY

1. Understand The Real Risk

2. Calculate Personal Risk

3. Re-assess and Adapt

4. Prioritize Lifestyle Interventions

5. Inspect What Is Expected

The prescription every physician should give is food as medicine

www.DrBatiste.com