



Brigham and Women's Hospital

Founding Member, Mass General Brigham

Inflammation and Atherothrombosis: Update for 2026

Paul M Ridker, MD, MPH

Director, Center for Cardiovascular Disease Prevention

Divisions of Cardiovascular Medicine and Preventive Medicine

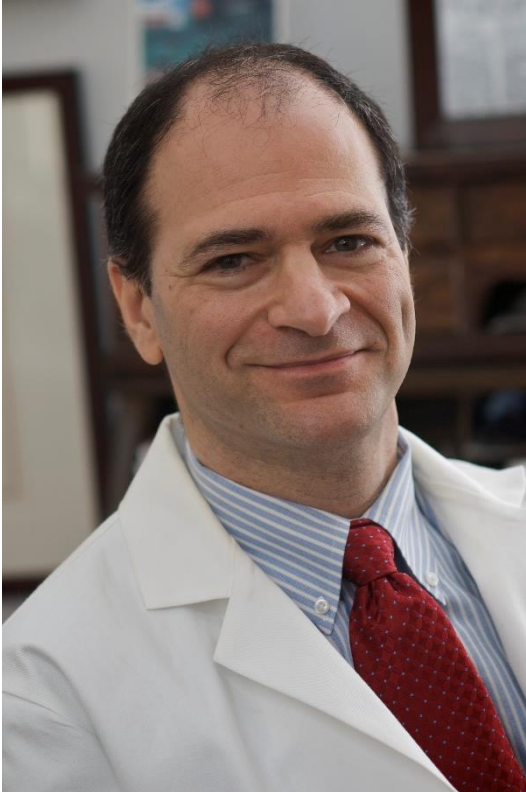
Brigham and Women's Hospital

Eugene Braunwald Professor of Medicine

Harvard Medical School



Paul M Ridker, MD, MPH



Harvard Medical School
Harvard TC Chan School of Public Health
Residency and Cardiology Fellowship at Brigham and
Women's Hospital

Eugene Braunwald Professor of Medicine at HMS

Research and Clinical Focus : Inflammation and
Atherosclerosis



DISCLOSURES

Please state any/all financial disclosures for past 24 months.

Dr. Ridker has received investigator-initiated research grants from Kowa, Pfizer, AstraZeneca, Novo Nordisk, NHLBI, NCI

Dr. Ridker has served as a consultant to Agepha, Novartis, Arrowhead, AstraZeneca, CSL Behring, SOCAR, Novo Nordisk, Eli Lilly, New Amsterdam, Cardio Therapeutics, Caristo, Merck, Nodthera, Pfizer, NHLBI, Angiowave, Bio-Age, Tourmaline Bio.

Dr Ridker serves on the medical advisory board of the Leducq Foundation (Paris FR).



OBJECTIVES

To understand the role of inflammation in atherosclerotic cardiovascular disease.

To understand how and why to screen for high sensitivity C-reactive protein (hsCRP) in both primary and secondary prevention.

To understand the emerging role of targeted anti-inflammatory therapy as an adjunct to lipid lowering in the prevention and treatment of atherosclerotic disease (IL-1 inhibition, IL-6 inhibition, colchicine, etc).



October 2025

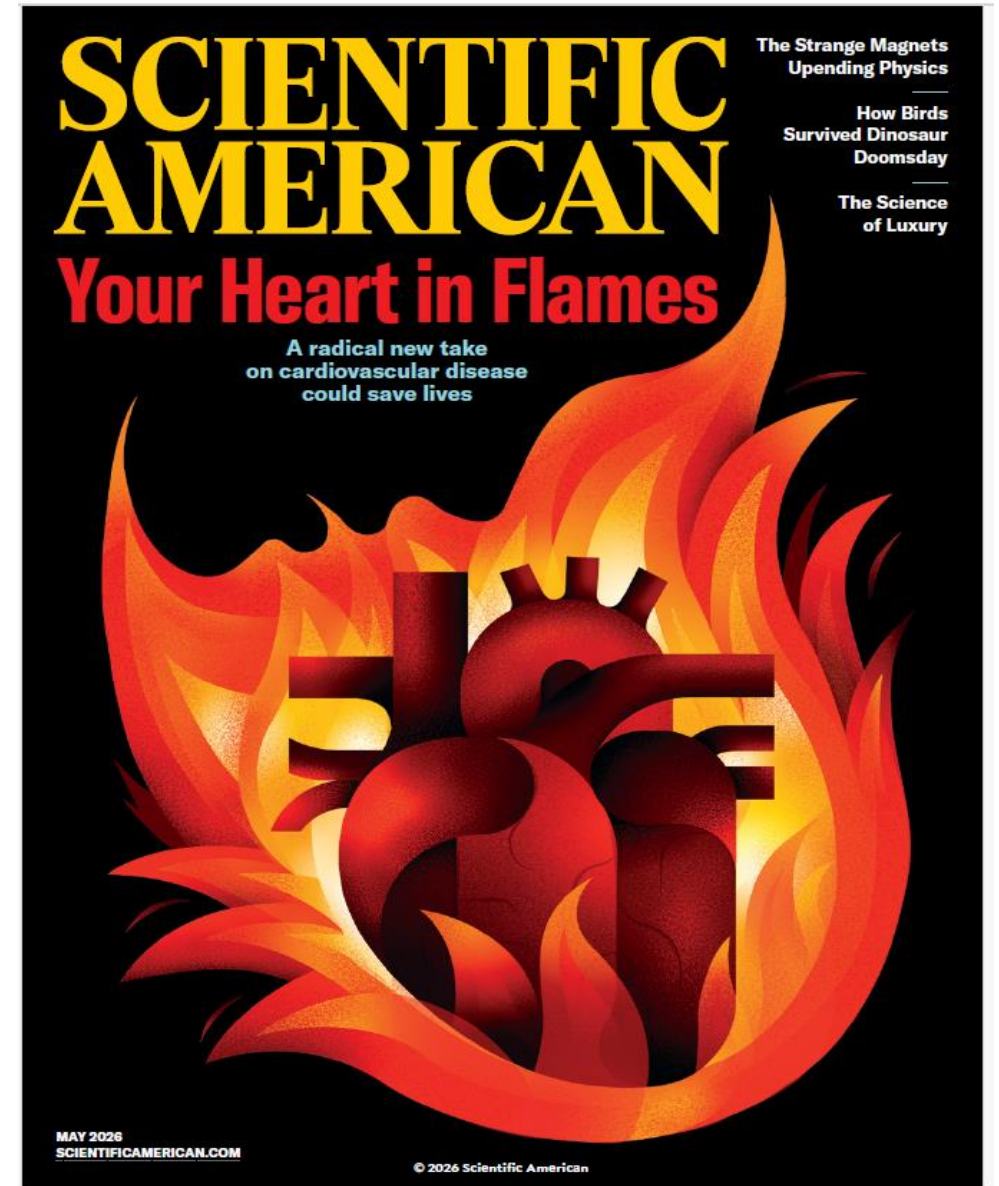
ACC SCIENTIFIC STATEMENT

Inflammation and Cardiovascular Disease: 2025 ACC Scientific Statement

A Report of the American College of Cardiology

Mensah GA et al. *J Am Coll Cardiol* 2025;S0735-1097(25)07555-2.

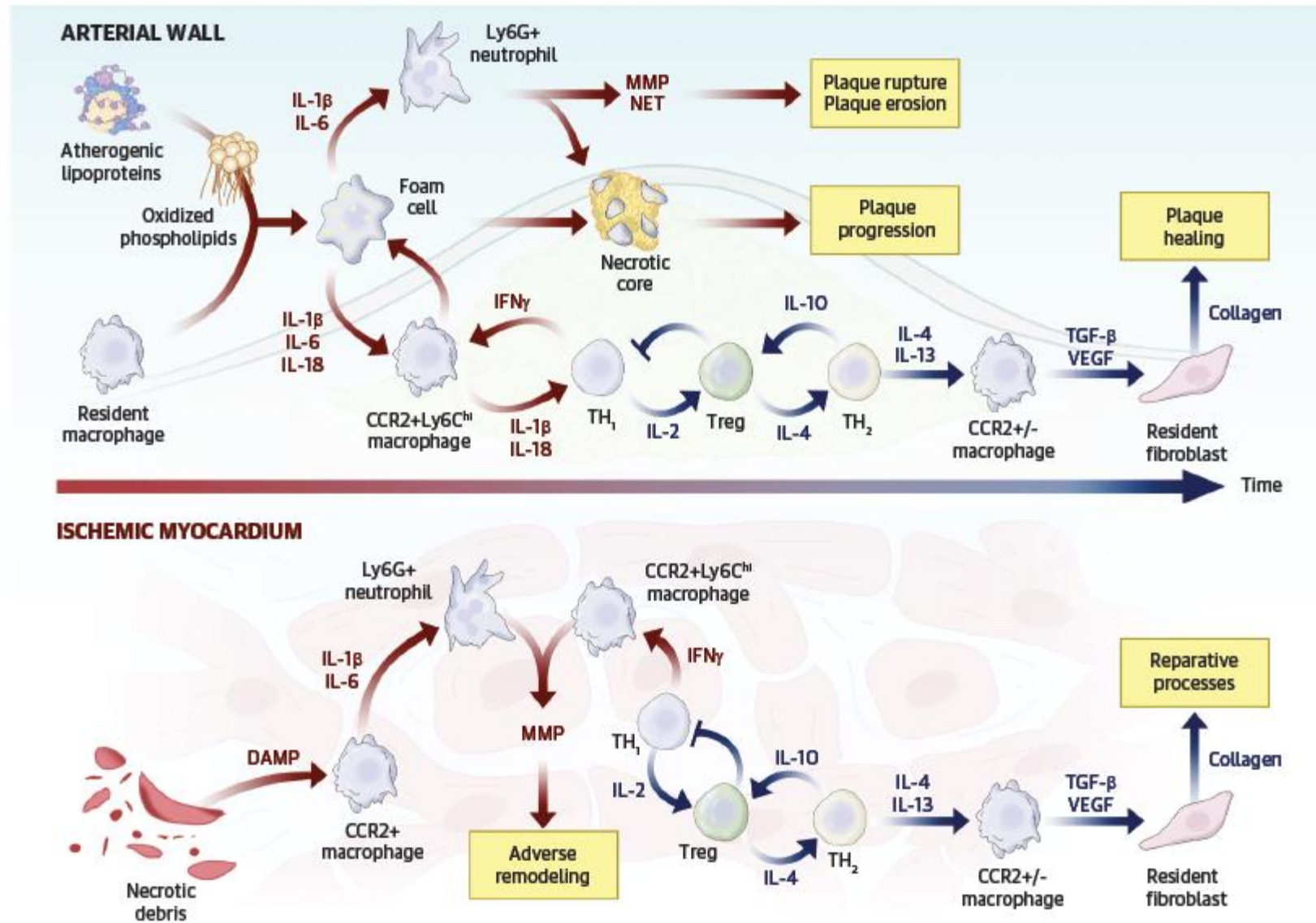
May 2026



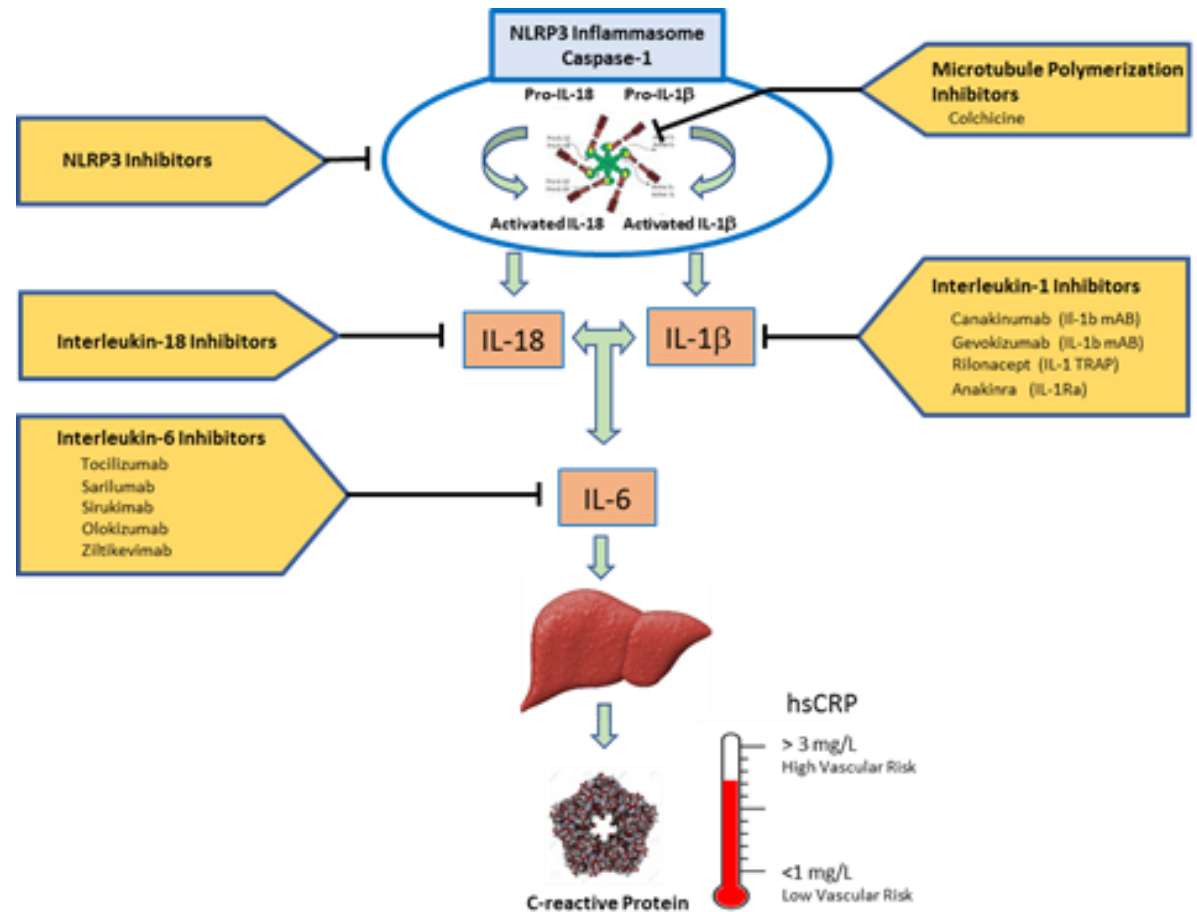
Inflammation and Cardiovascular Disease: 2025 ACC Scientific Statement

- 1. The evidence linking inflammation with atherosclerotic CVD is no longer exploratory but is compelling and clinically actionable. The time for taking action has now arrived.**
- 2. Doctors will not treat what they do not measure. Universal screening of hsCRP in both primary and secondary prevention patients represents a major clinical opportunity and is therefore recommended.**
- 3. In primary prevention, the finding of a persistently elevated hsCRP level should lead to the initiation or intensification of statin therapy, irrespective of LDL cholesterol.**
- 4. CANTOS proved that targeting inflammation lowers CV risk. In addition to low dose colchicine which is FDA approved, several novel anti-inflammatory agents, including IL-6 inhibitors, are now being evaluated in ongoing randomized trials in the settings of chronic kidney disease, dialysis, HFpEF, and acute coronary syndrome.**

Immune Cell Dynamics Driving Plaque Modification and Myocardial Remodeling

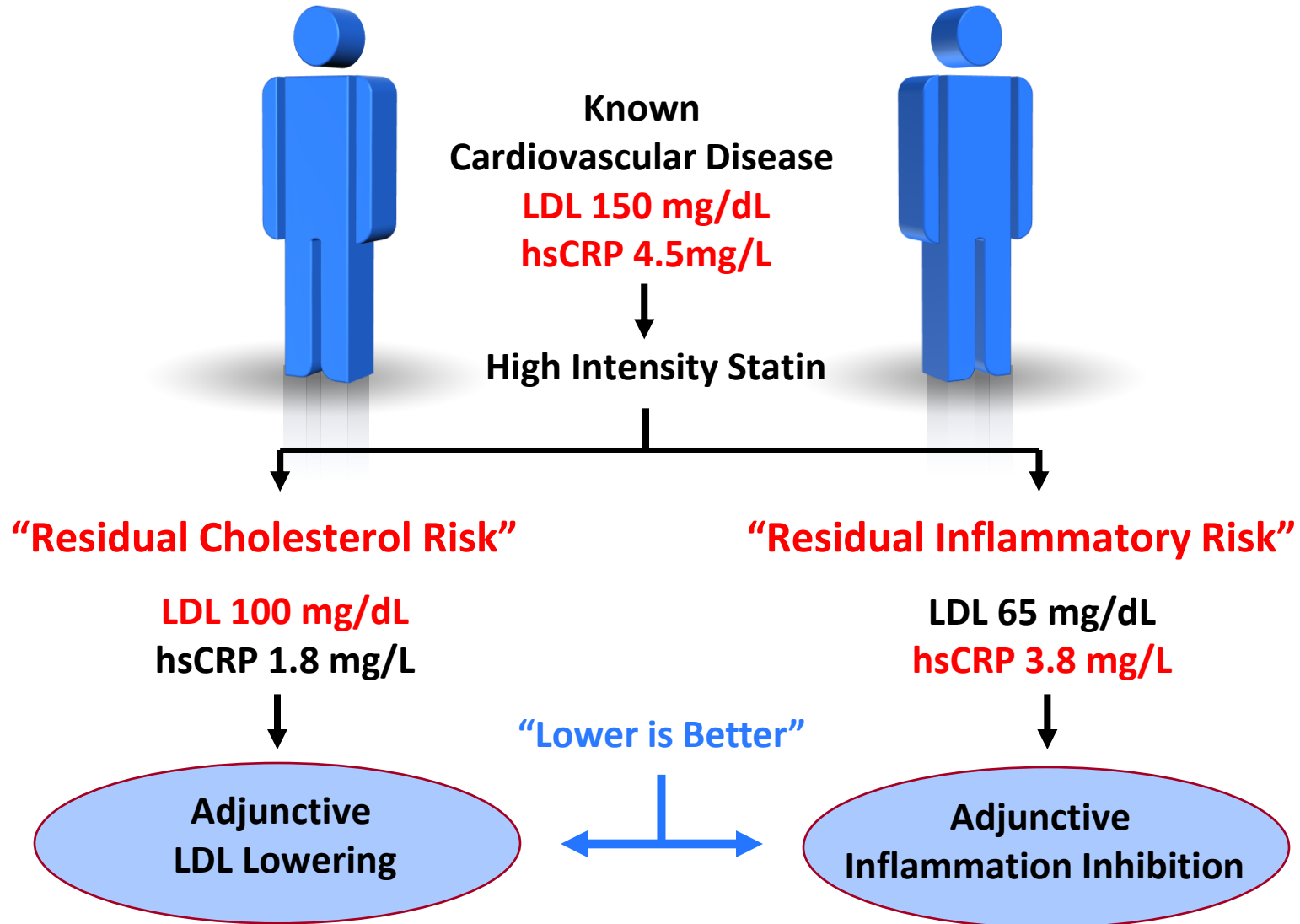


Can Targeted Anti-Cytokine Therapy Reduce Cardiovascular Event Rates and Prolong Life?



Residual Inflammatory Risk: Addressing the Obverse Side of the Atherosclerosis Prevention Coin

Eur Heart J 2016;37:1720-22



Universal screening for hsCRP in patients with atherosclerotic disease: a Major therapeutic opportunity

Giovanna Liuzzo ^{1,2} and Paul M Ridker ^{3*}

- hsCRP, a biomarker of silent vascular inflammation, is at least as strong a predictor of future cardiovascular events as is LDL-C in both primary prevention and secondary prevention.
- Doctors will not treat what they do not measure. We need universal guideline recommendations to screen all patients for hsCRP just as we screen for cholesterol.
- Review contemporary clinical data in secondary and primary prevention

Low Grade Systemic Inflammation Precedes By Many Years the Onset of Vascular Events

The New England Journal of Medicine

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VOLUME 336

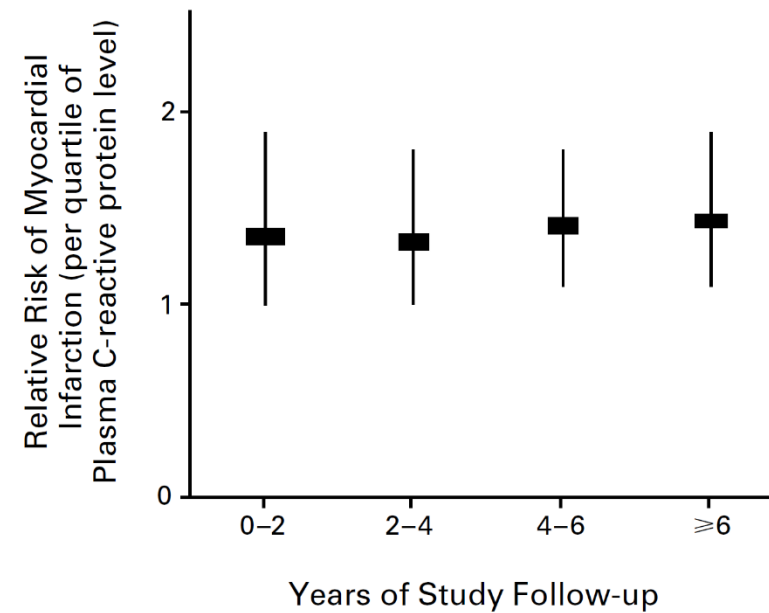
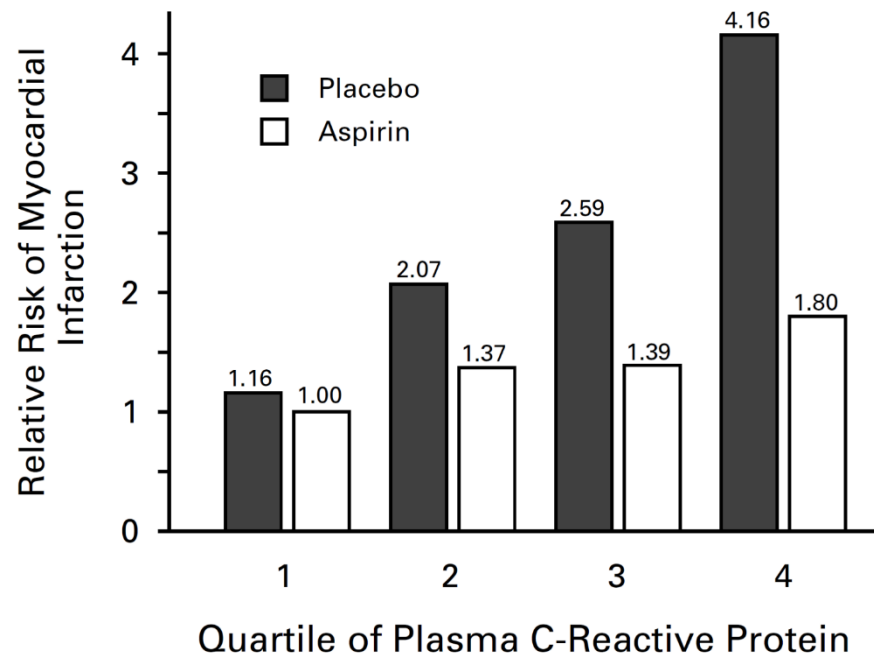
APRIL 3, 1997

NUMBER 14



INFLAMMATION, ASPIRIN, AND THE RISK OF CARDIOVASCULAR DISEASE IN APPARENTLY HEALTHY MEN

PAUL M. RIDKER, M.D., MARY CUSHMAN, M.D., MEIR J. STAMPFER, M.D., RUSSELL P. TRACY, PH.D.,
AND CHARLES H. HENNEKENS, M.D.



C-Reactive Protein, Interleukin 6, and Risk of Developing Type 2 Diabetes Mellitus

Aruna D. Pradhan, MD, MPH

JoAnn E. Manson, MD, DrPH

Nader Rifai, PhD

Julie E. Buring, ScD

Paul M Ridker, MD, MPH

Context Inflammation is hypothesized to play a role in development of type 2 diabetes mellitus (DM); however, clinical data addressing this issue are limited.

Objective To determine whether elevated levels of the inflammatory markers interleukin 6 (IL-6) and C-reactive protein (CRP) are associated with development of type 2 DM in healthy middle-aged women.

Design Prospective, nested case-control study.

Pradhan et al,
JAMA 2001;286:327-334

C-Reactive Protein and the Risk of Developing Hypertension

Howard D. Sesso, ScD, MPH

Julie E. Buring, ScD

Nader Rifai, PhD

Gavin J. Blake, MD, MPH

J. Michael Gaziano, MD, MPH

Paul M Ridker, MD, MPH

Context Although it has been hypothesized that hypertension is in part an inflammatory disorder, clinical data linking inflammation with incident hypertension are scarce.

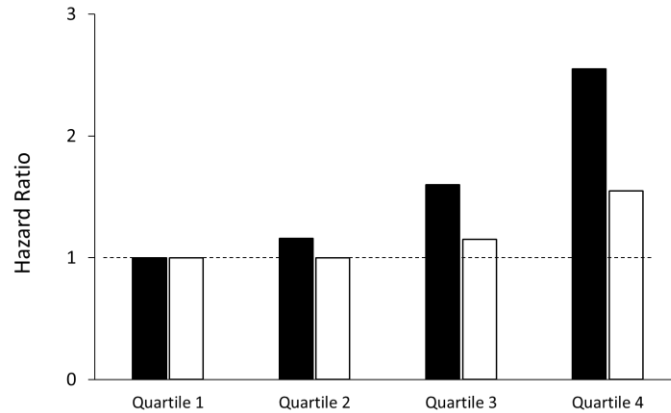
Objective To examine whether C-reactive protein levels, a marker of systemic inflammation, are associated with incident hypertension.

Design, Setting, and Participants A prospective cohort study that began in 1992 of 20525 female US health professionals aged 45 years or older who provided baseline blood samples with initially normal levels of blood pressure (BP) (systolic BP <140 mm Hg and diastolic BP <90 mm Hg. and no history of hypertension or antihyper-

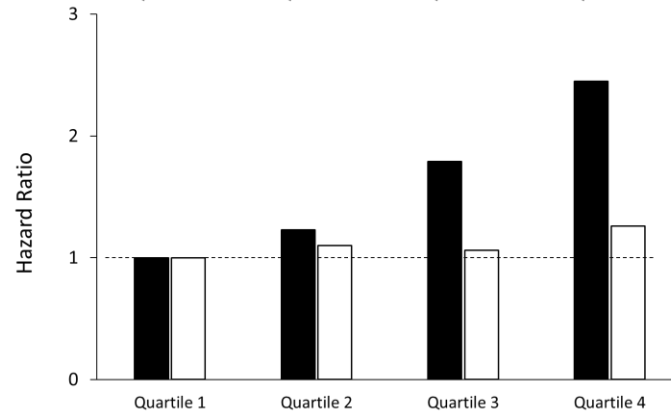
Sesso et al,
JAMA 2003;290:2945-2951

Secondary Prevention: Direct Comparison of hsCRP and LDLC as Predictors of Cardiovascular Death Among Statin Treated Patients

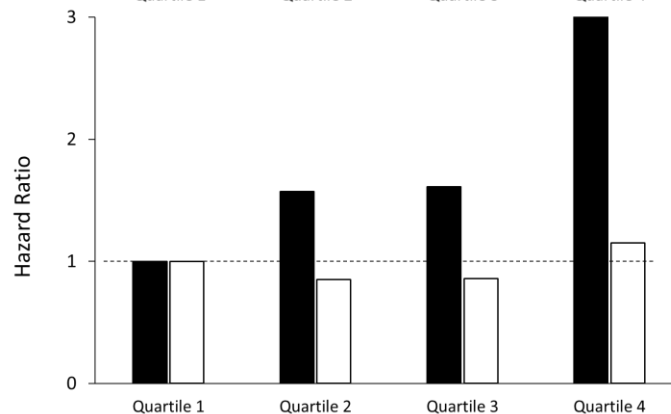
PROMINENT
(N = 9,988)



REDUCE-IT
(N = 8,179)



STRENGTH
(N = 13,078)

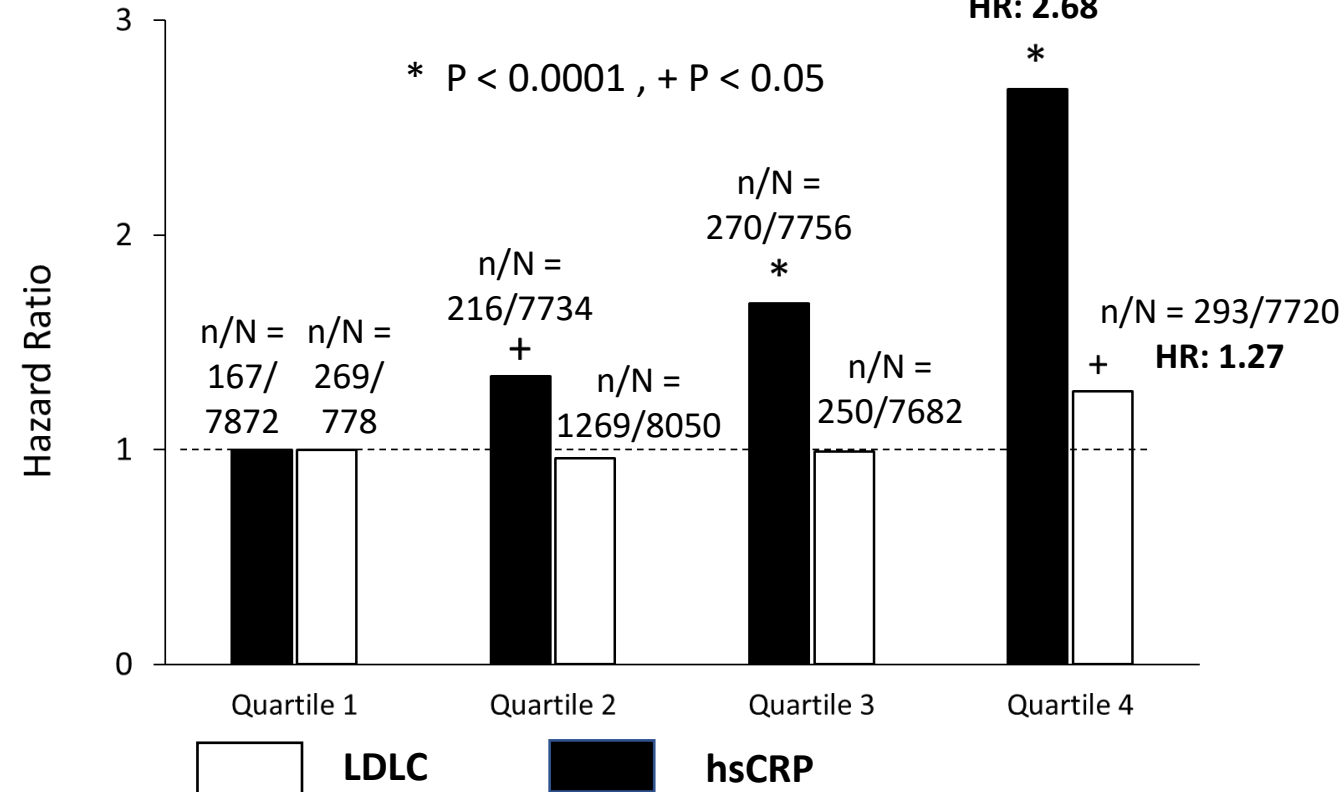


Pooled Data (N = 31,245)

Cardiovascular Death

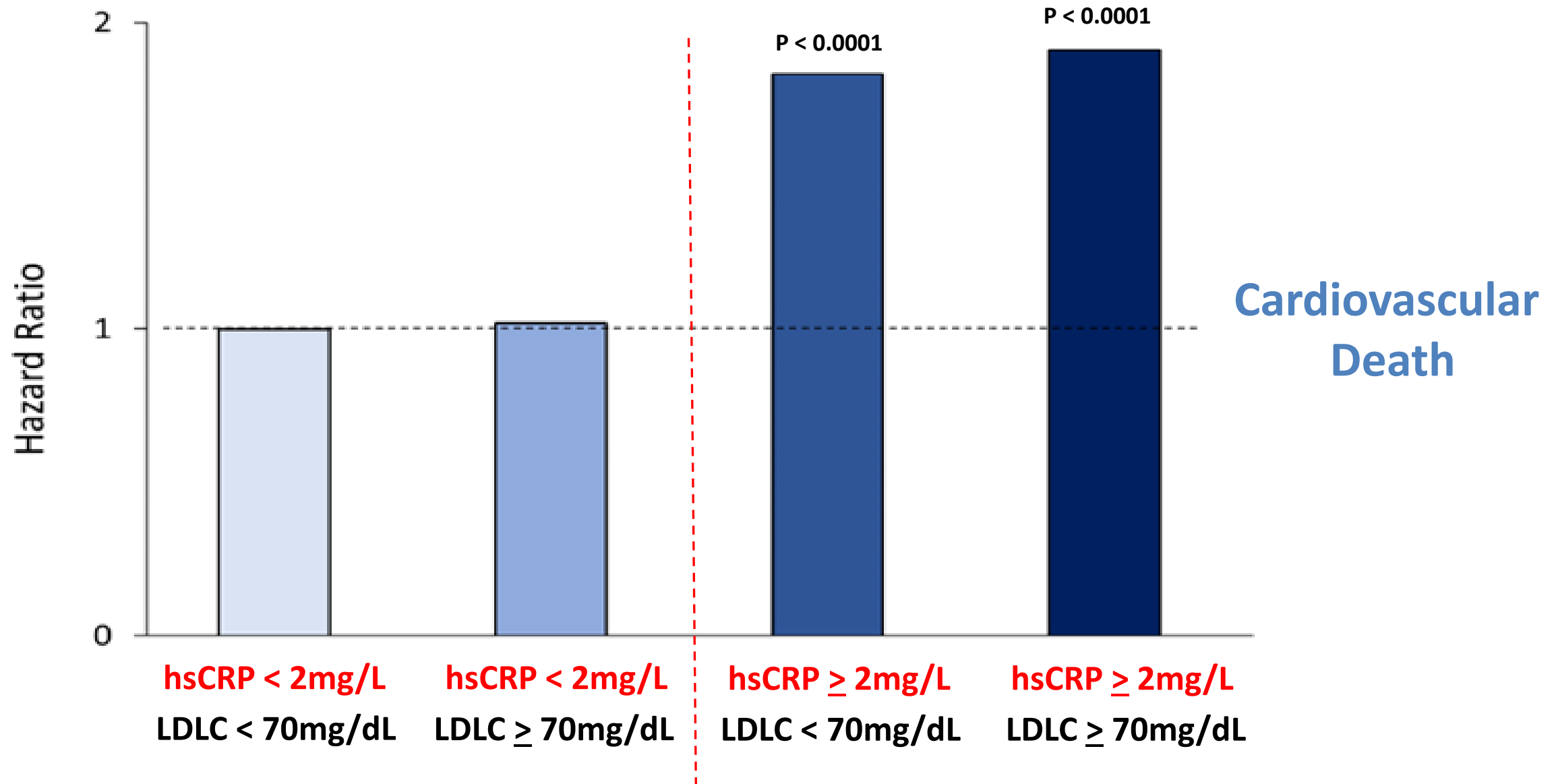
n/N = 423/7806
HR: 2.68

* P < 0.0001 , + P < 0.05



Lancet 2023;401:1293–1301

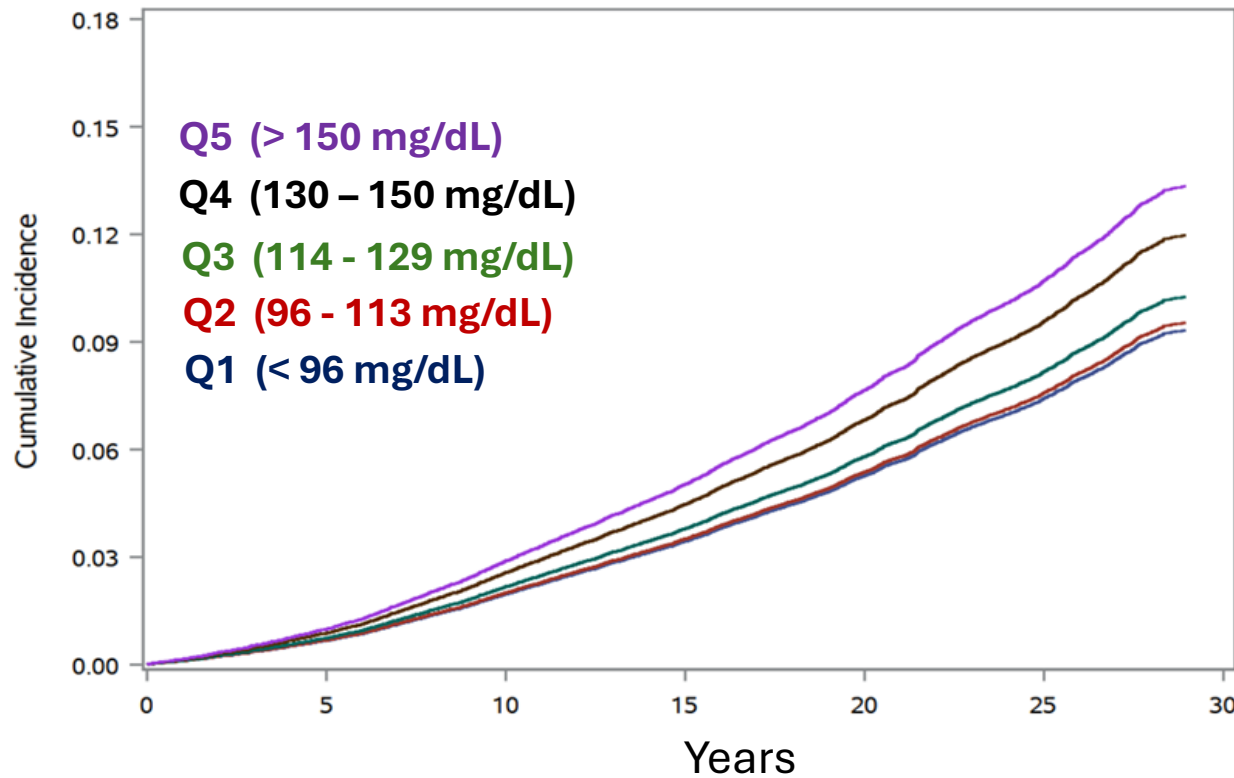
hsCRP is a Powerful Determinant of Cardiovascular Death Irrespective of LDLC Among 31,245 Contemporary Statin Treated Secondary Prevention Patients



**30-Year Covariable Adjusted* Hazard Ratios (HR) and Cumulative Incidence for
First Major Adverse Cardiovascular Events in 27,939 Initially Healthy American Women
According to Baseline Quintile of LDL-C and hsCRP**

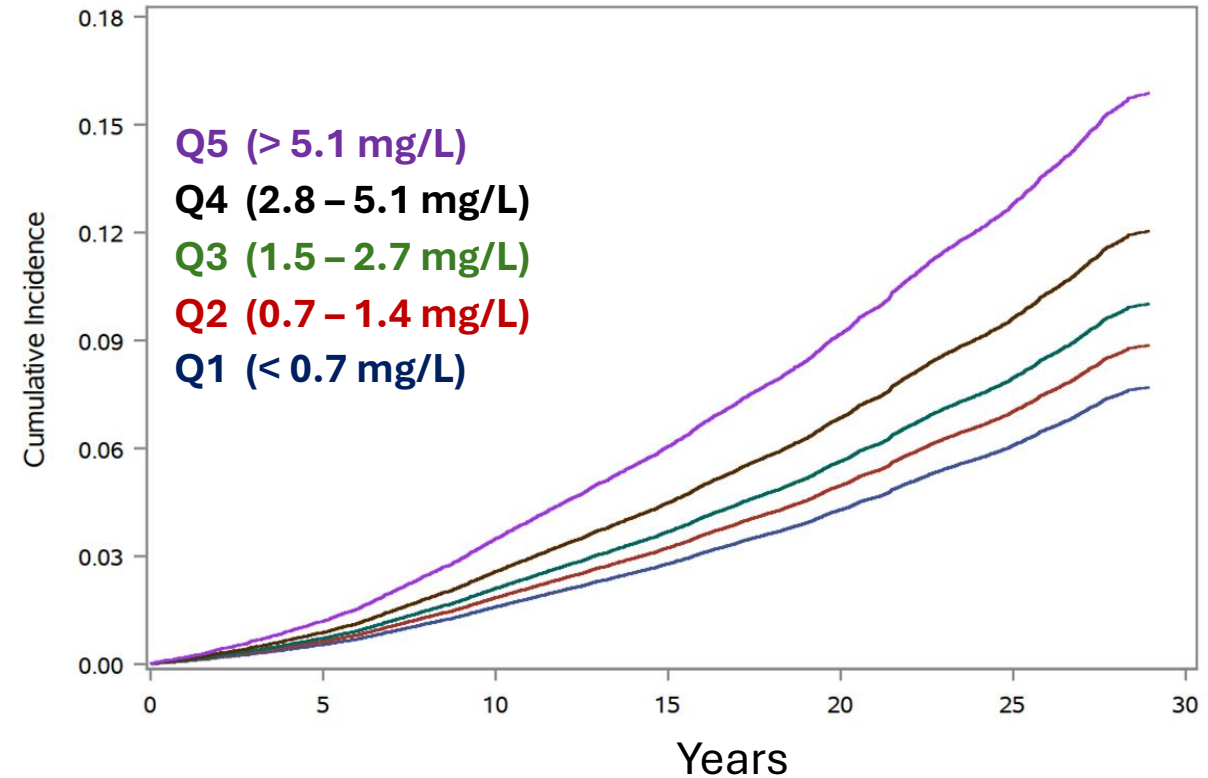
LDL-C

HR* per SD 1.10 (95%CI 1.06-1.14)



hsCRP

HR* per SD 1.23 (95%CI 1.18-1.27)



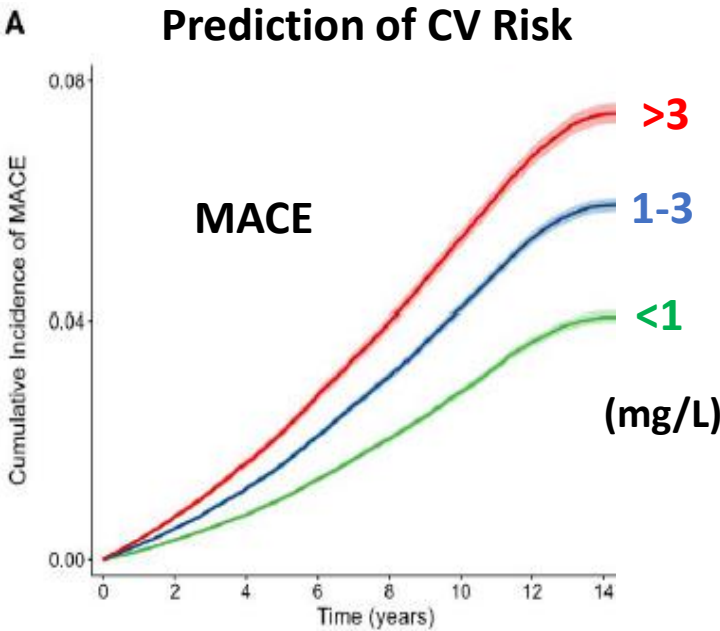
*HR adjusted for age, treatment assignment, smoking status, diabetes, blood pressure, eGFR, HRT use, and BMI



UK Biobank

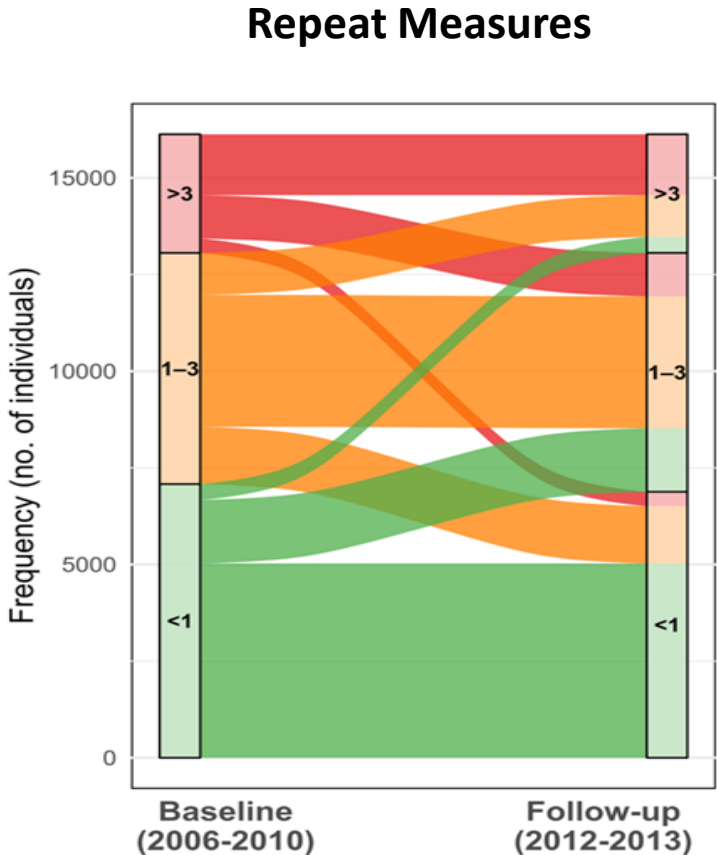
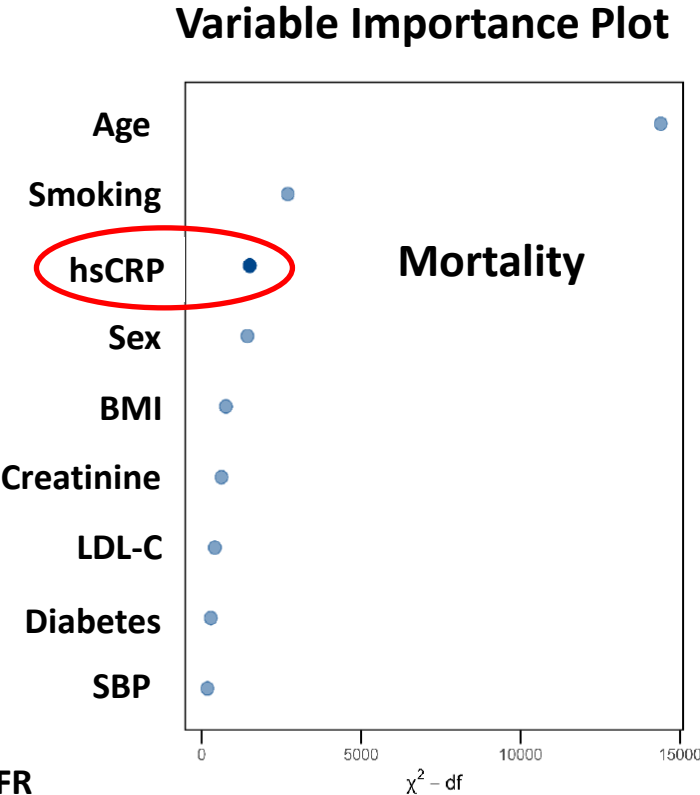
N = 448,653

C-reactive protein and cardiovascular risk in the general population



$HR_{adj} = 1.34 (1.29-1.39)$

Adjusted for age, sex, BMI, DM, smoking, BP, LDL-C, eGFR

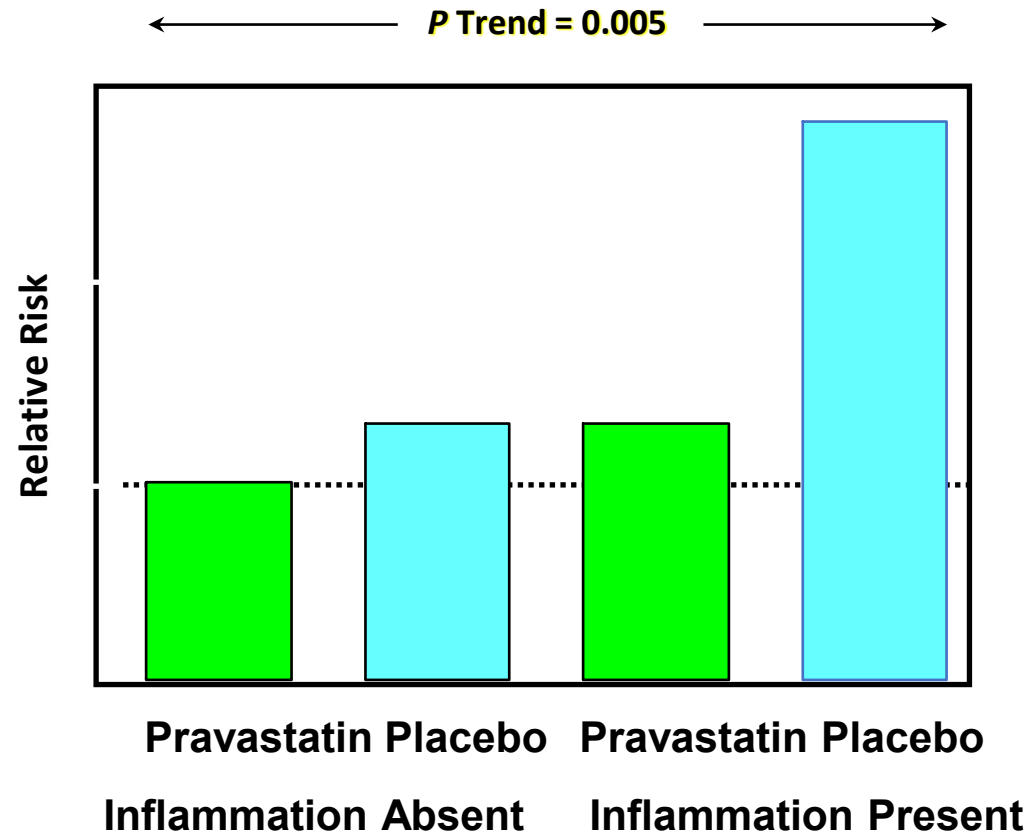


Repeat confirmation that
hsCRP strongly and
independently predicts
future CV events

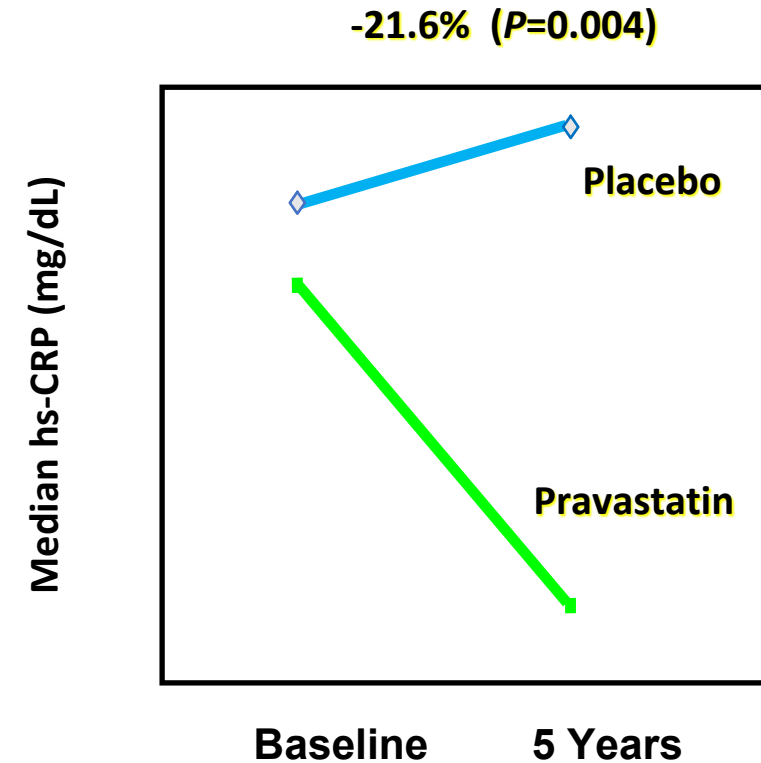
The relative importance of
hsCRP on mortality risk is larger
than that of all other factors
Except age and smoking

hsCRP levels <1, 1-3, and >3 mg/L
are stable over time.

Inflammation, Statin Therapy, and hsCRP: Initial Observations



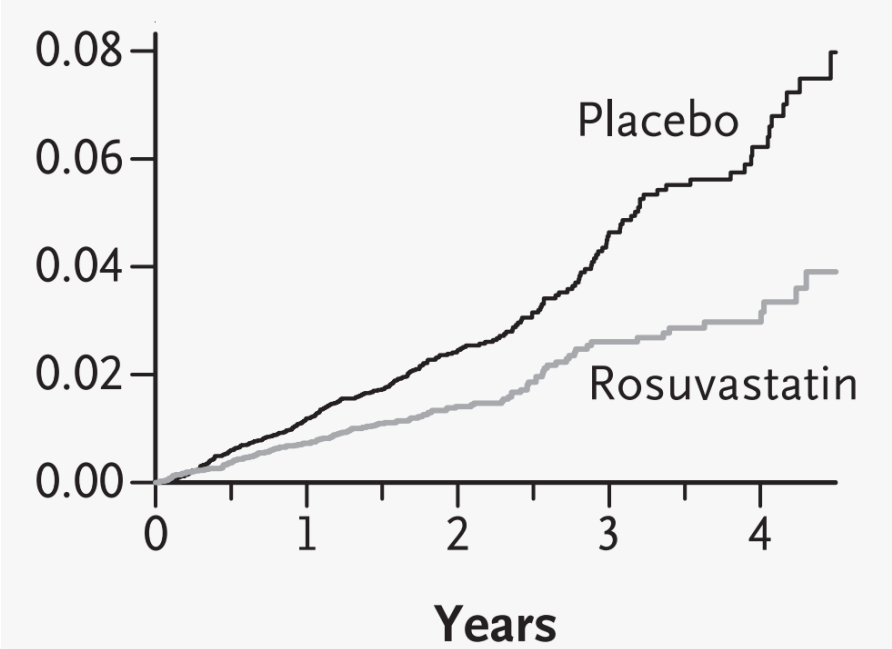
Circulation 1998;98:839–844.



Circulation 1999;100:230-235.

JAMA 2001;286:64-70.

Effectiveness of Statin Therapy in Primary Prevention When LDL is Low but hsCRP is High: JUPITER

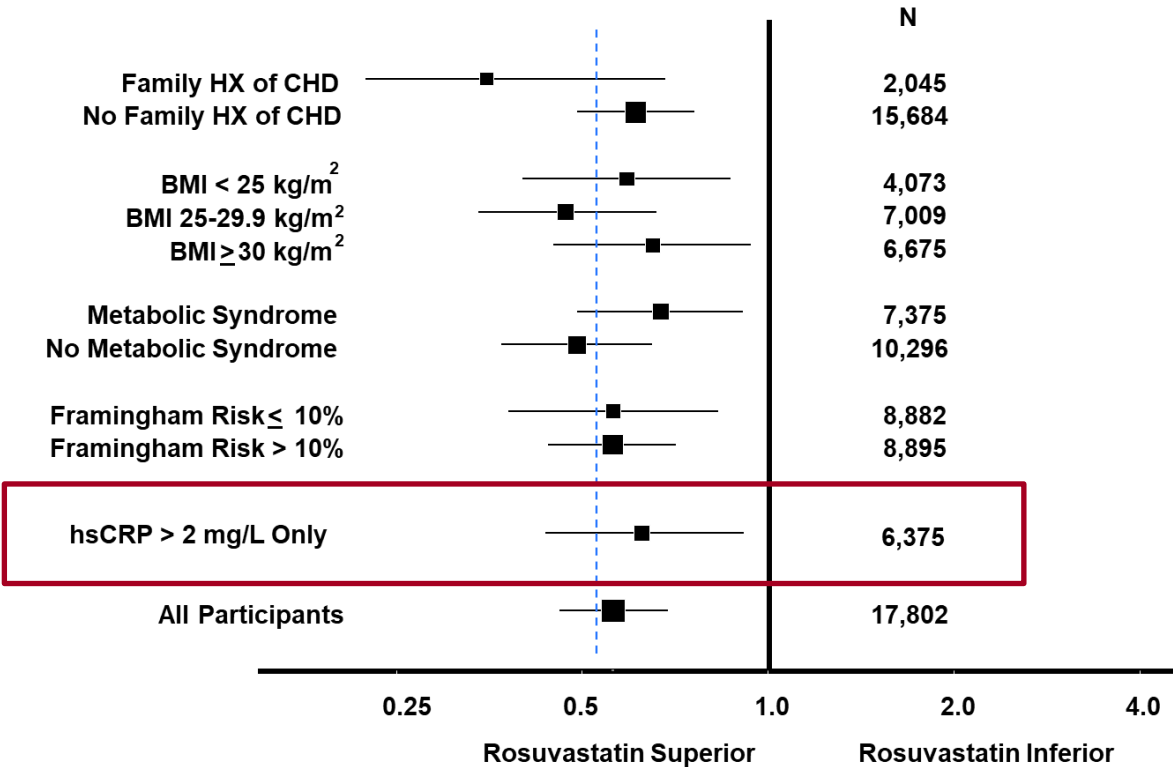


Endpoint	Relative Risk Reduction (%)	HR (95% CI)	P
Primary Endpoint	44	0.56 (0.46-0.69)	<0.00001
MI	63	0.37 (0.22-0.58)	<0.00001
Stroke	48	0.52 (0.34-0.79)	0.002
Revascularization	46	0.54 (0.41-0.72)	<0.0001
Death	20	0.80 (0.67-0.97)	0.02

NEJM 2008;359:2195-2207



Rosuvastatin to Prevent Vascular Events in Men and Women with Elevated C-Reactive Protein



Introducing a New Clinical Syndrome “SMuRF-Less but Inflamed”

“SMuRFs” = Standard Modifiable Risk Factors

“SMuRF-Less” = No Standard Modifiable Risk Factors

“SMuRF-Less but Inflamed” = SMuRF-Less + Elevated hsCRP



“SMuRFs”



“SMuRF-Less”

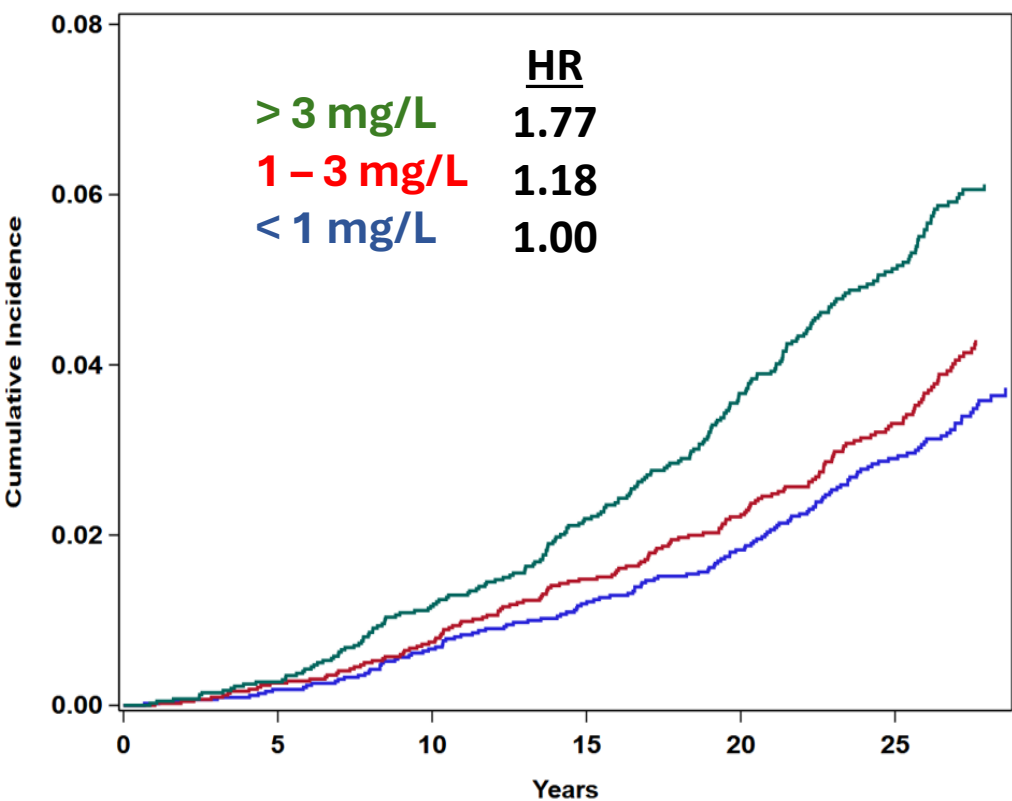
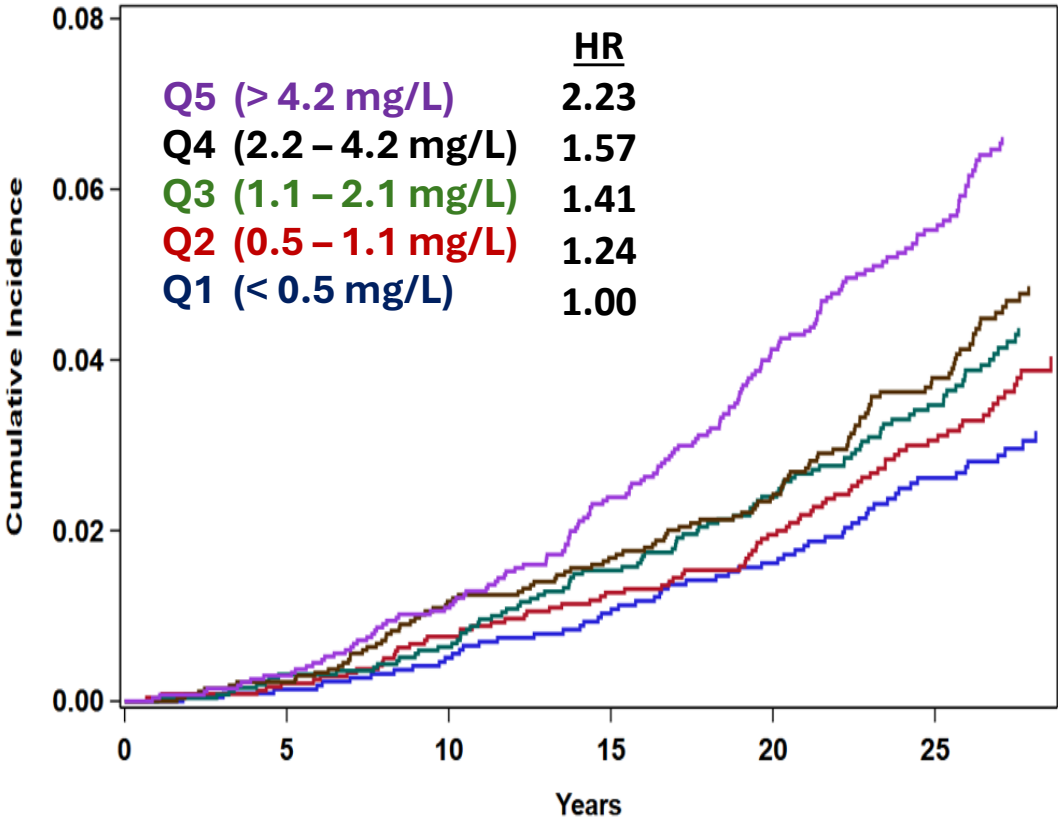
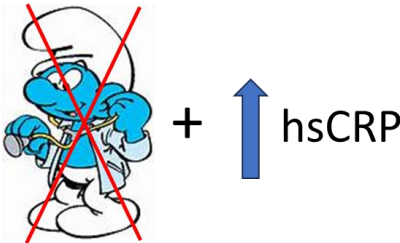


+ ↑ hsCRP

“SMuRF-Less but Inflamed”

30-Year Age Adjusted Cumulative Incidence of Coronary Heart Disease Events Among 12,530 Initially Healthy SMuRF-Less Women According to Baseline Level of hsCRP

“SMuRF-Less but Inflamed”

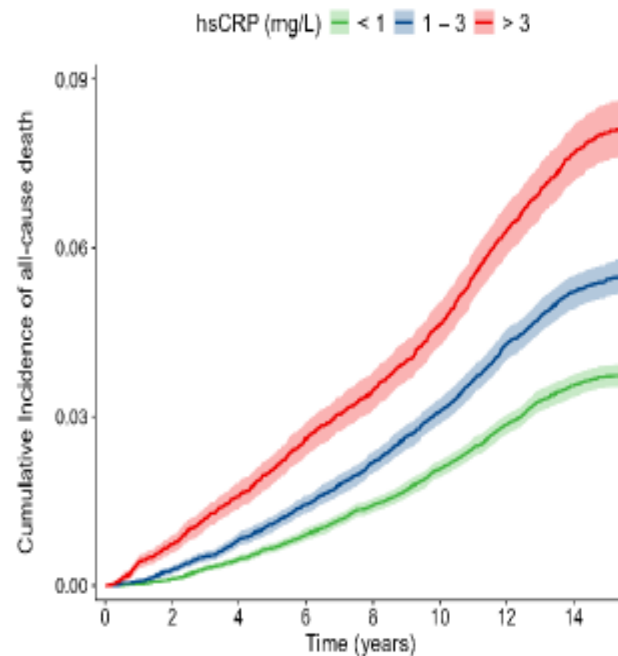


C-reactive protein and cardiovascular risk in the general population

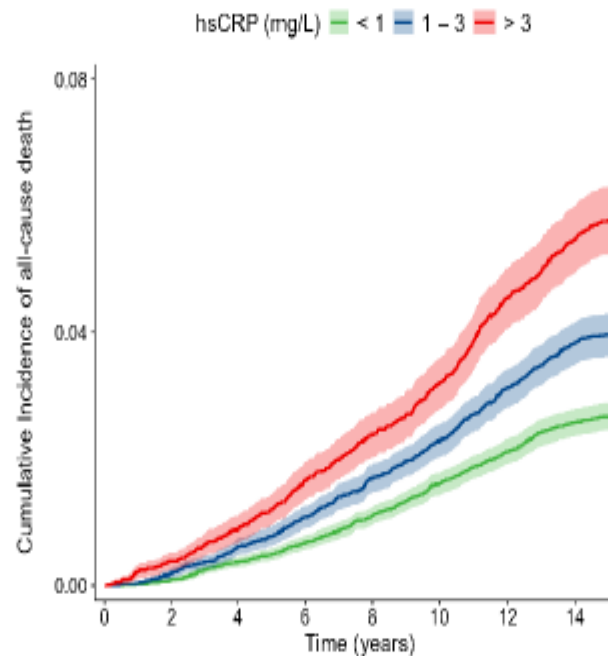
**Confirmation of the
“SMuRF-Less but Inflamed”
Clinical Syndrome in Men as
Well as Women**

hsCRP as a predictor of all-cause mortality among “SMuRF-Less but Inflamed” UK Biobank Participants

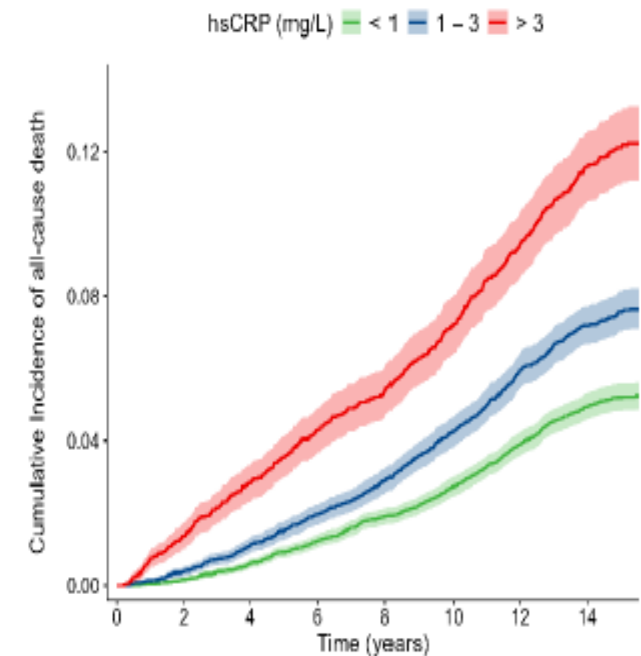
Total Cohort



Women



Men



Statin Therapy for the “SMuRF-Less but Inflamed”

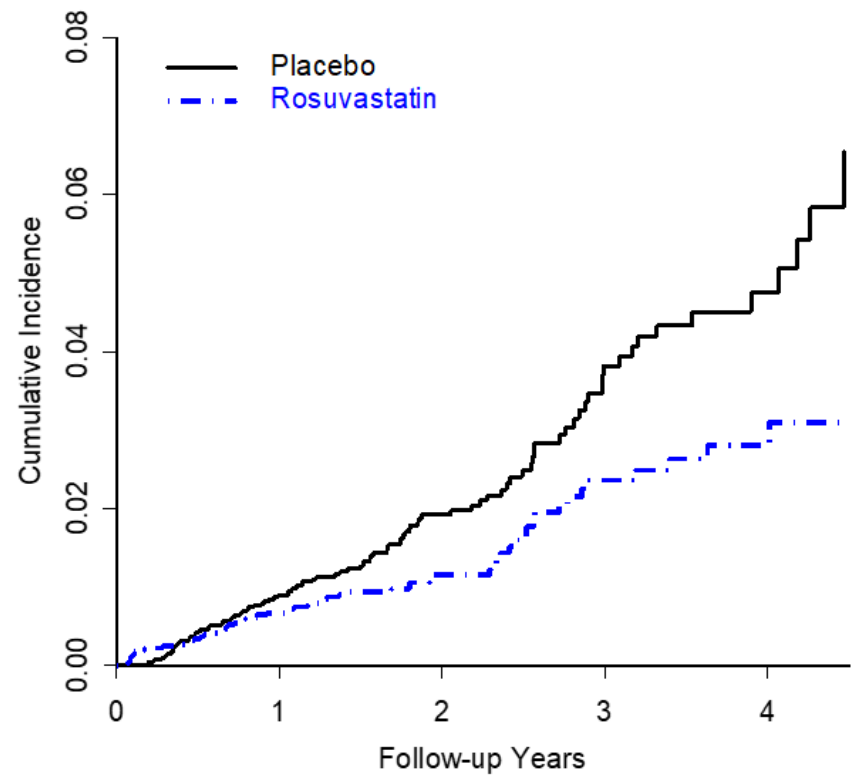
Cardiovascular outcomes in the primary prevention JUPITER trial among 8,278 individuals with none of the Standard Modifiable Risk Factors (SMuRFs), adjusted for body mass index and eGFR.

Primary Endpoint: SMuRFs-less + High hsCRP

“SMuRF-Less
but Inflamed”



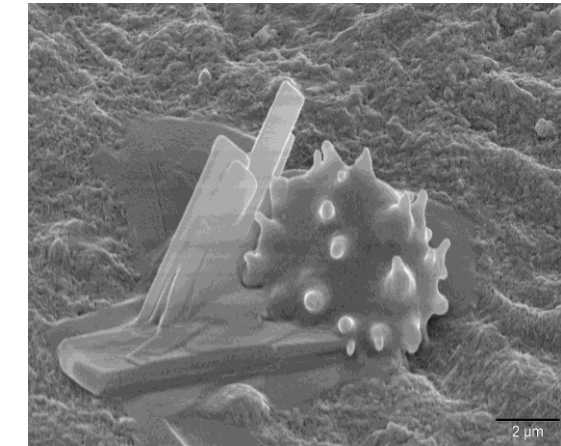
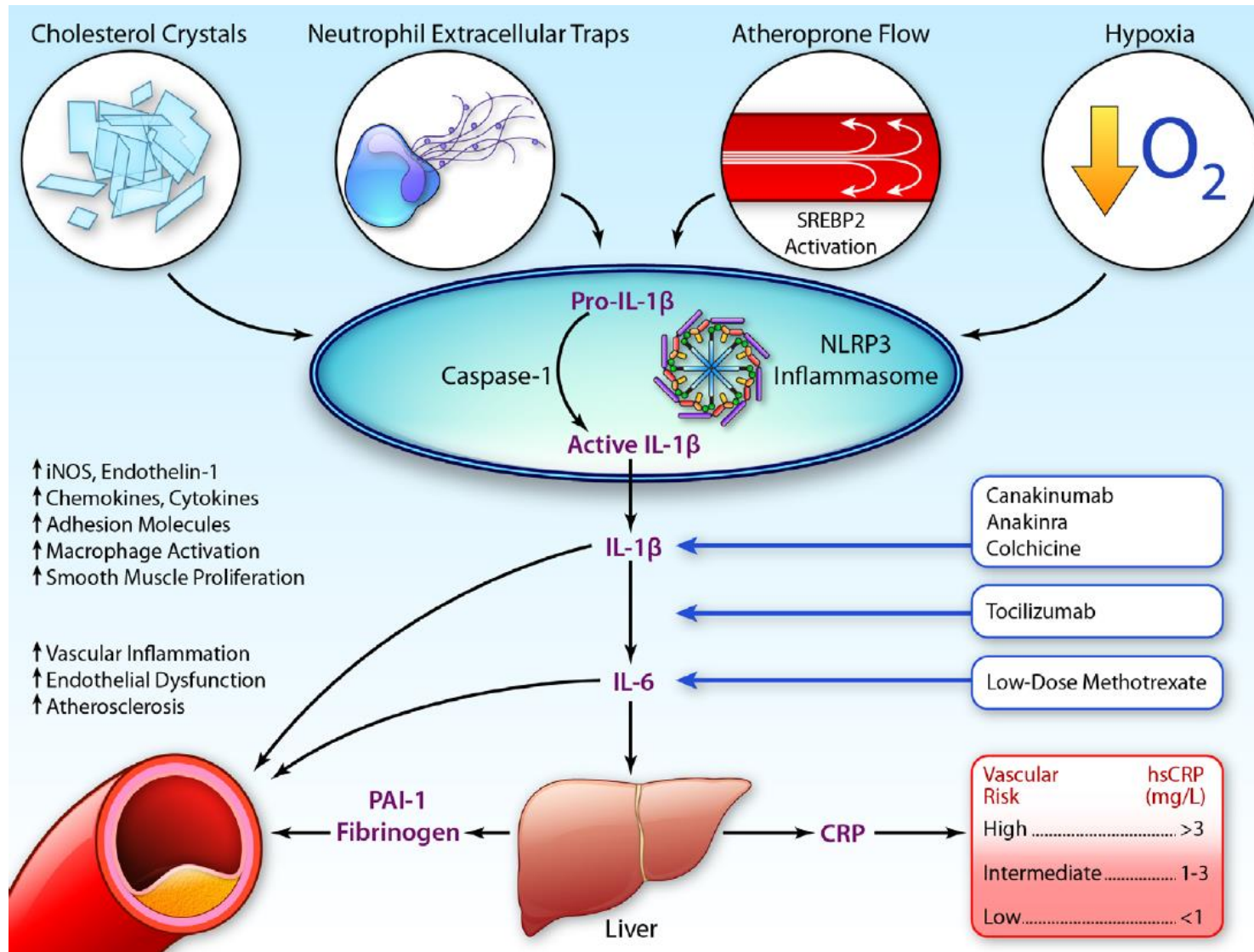
N = 8,278



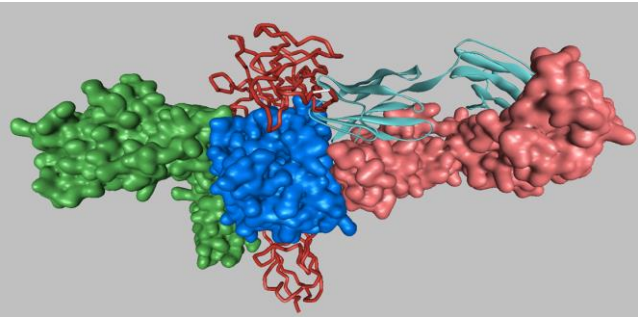
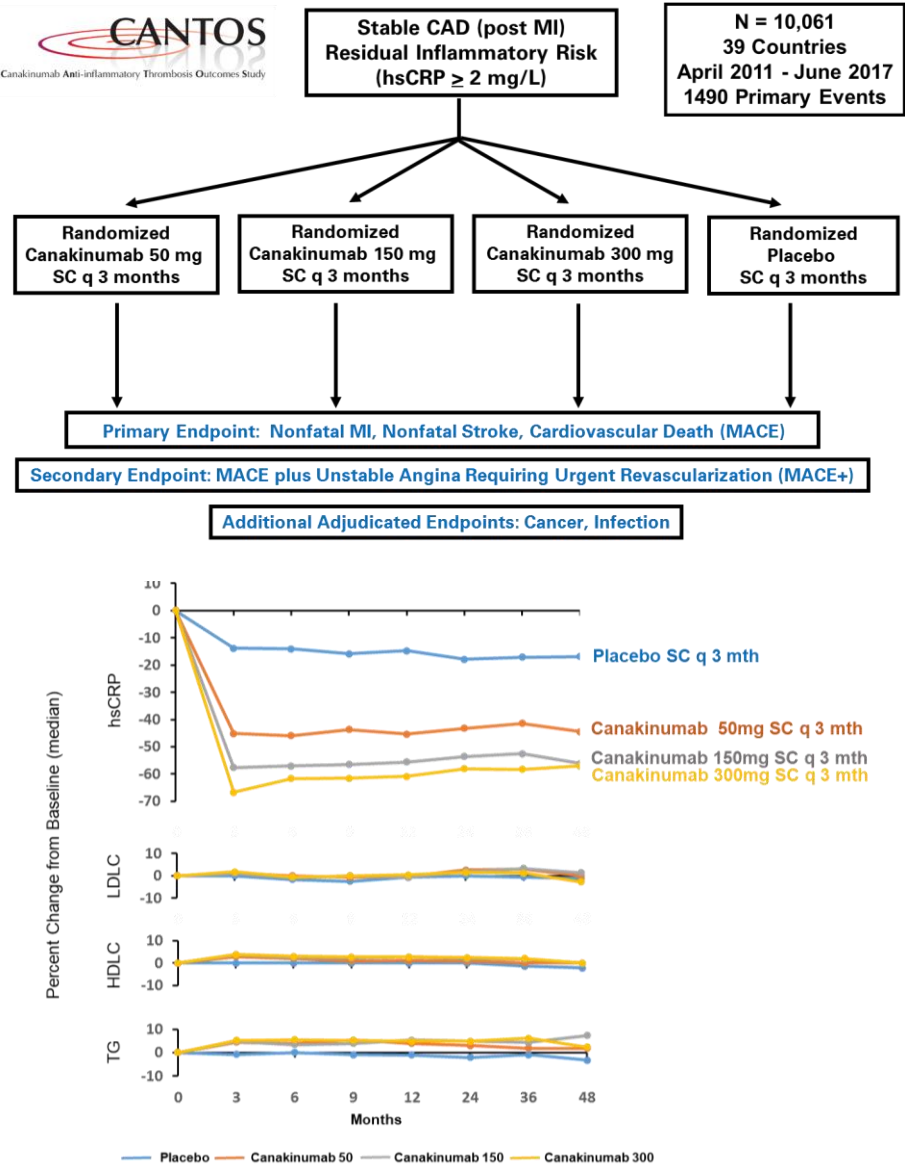
Endpoint	Relative Risk Reduction (%)*	HR *	(95%CI)*
Primary Endpoint	38 %	0.62	(0.45 – 0.85)
MI	69 %	0.31	(0.15 – 0.66)
Stroke	47 %	0.53	(0.25 – 1.09)
Revascularization	39 %	0.61	(0.40 – 0.93)

*Adjusted for BMI and eGFR

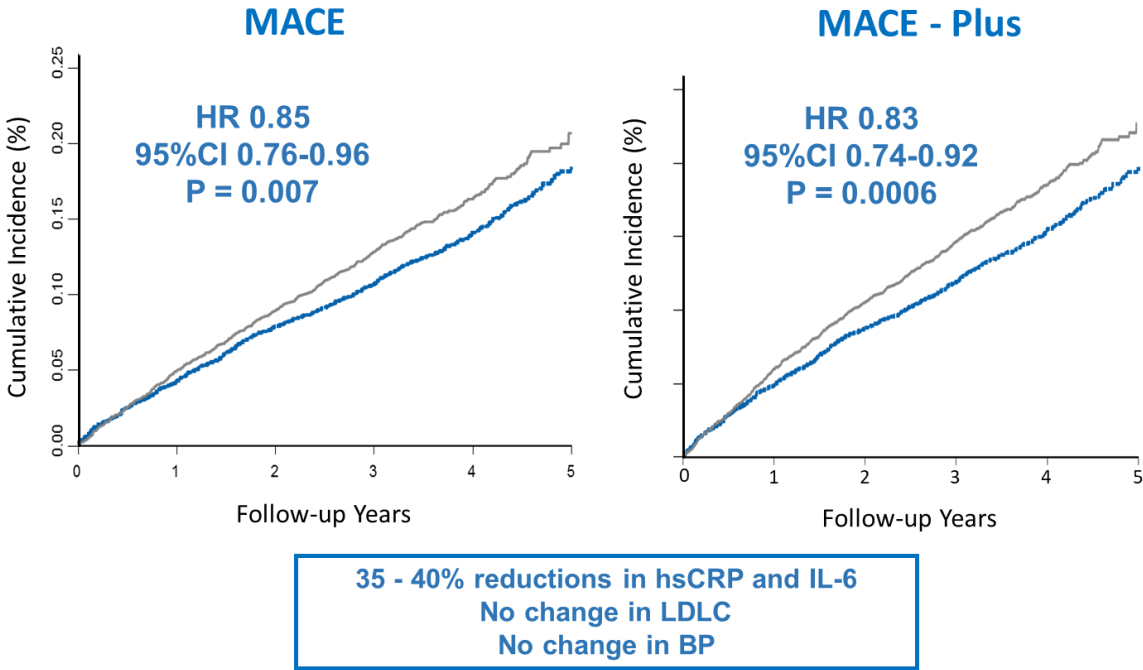
From CRP to IL-6 to IL-1 to NLRP3: Moving Upstream to Identify Novel Targets for Atheroprotection



Canakinumab, a Human Monoclonal Antibody Neutralizing IL-1 β

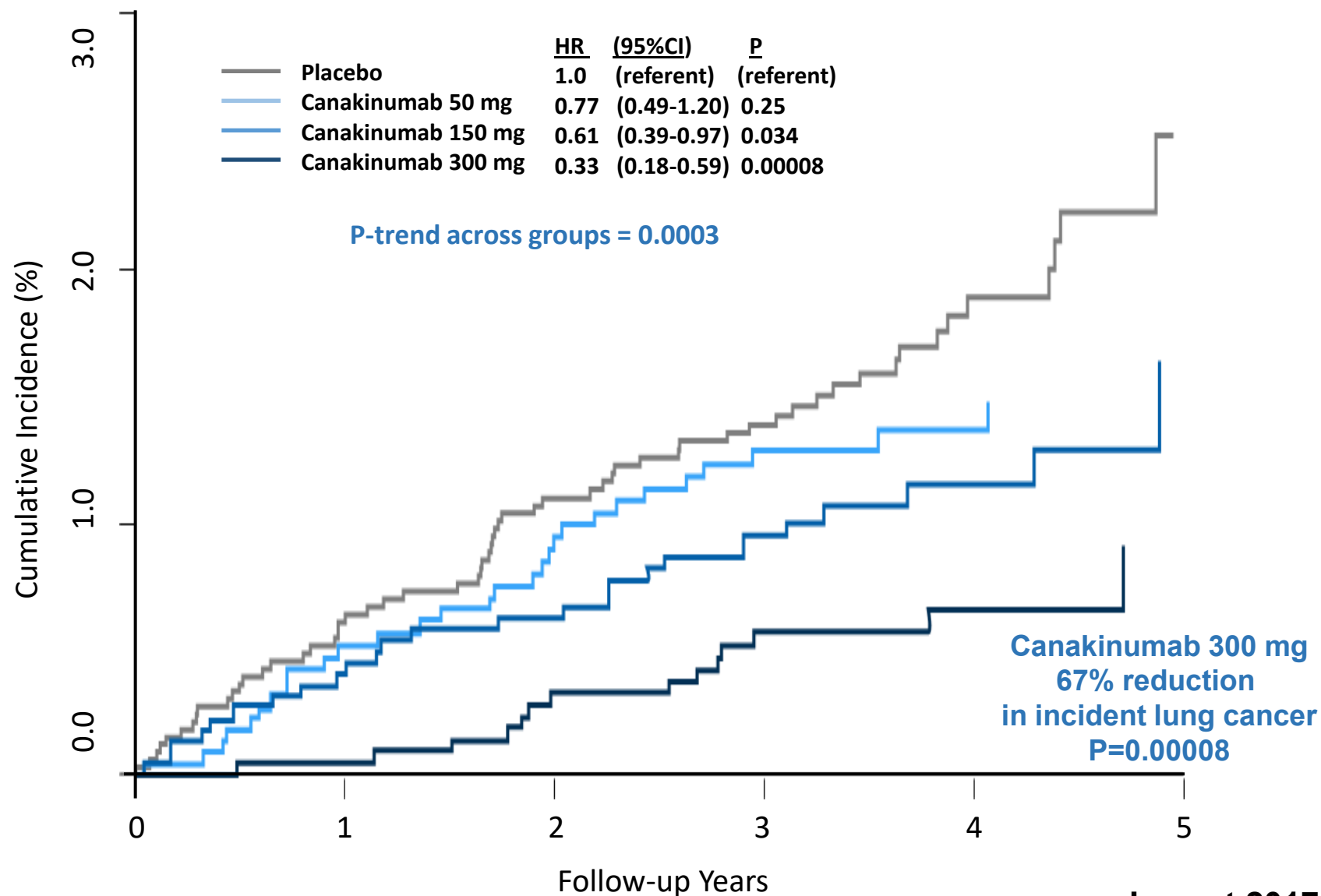


— Placebo SC q 3 months
— Canakinumab 150/300 mg SC q 3 months



CANTOS: Additional Non-Cardiovascular Clinical Benefits

Incident Lung Cancer



Low-Dose Colchicine for Secondary Prevention of Cardiovascular Disease

Stefan M. Nidorf, MD, MBBS,* John W. Eikelboom, MBBS,† Charley A. Budgeon, BSc (HONS),‡
Peter L. Thompson, MD§
Perth, Australia; and Hamilton, Ontario, Canada

LoDoCo1
2013

ORIGINAL ARTICLE

Efficacy and Safety of Low-Dose Colchicine after Myocardial Infarction

Jean-Claude Tardif, M.D., Simon Kouz, M.D., David D. Waters, M.D.,
Olivier F. Bertrand, M.D., Ph.D., Rafael Diaz, M.D., Aldo P. Maggioni, M.D.,
Fausto J. Pinto, M.D., Ph.D., Reda Ibrahim, M.D., Habib Gamra, M.D.,
Ghassan S. Kiwan, M.D., Colin Berry, M.D., Ph.D., José López-Sendón, M.D.,
Petr Ostadal, M.D., Ph.D., Wolfgang Koenig, M.D., Denis Angoulvant, M.D.,
Jean C. Grégoire, M.D., Marc-André Lavoie, M.D., Marie-Pierre Dubé, Ph.D.,
David Rhainds, Ph.D., Mylène Provencher, Ph.D., Lucie Blondeau, M.Sc.,
Andreas Orfanos, M.B., B.Ch., Philippe L. L'Allier, M.D., Marie-Claude Guertin, Ph.D.,
and François Roubille, M.D., Ph.D.

COLCOT
2019

Long-term colchicine for the prevention of vascular recurrent events in non-cardioembolic stroke (CONVINCE): a randomised controlled trial

Peter Kelly, Robin Lemmens, Christian Weimar, Cathal Walsh, Francisco Purroy, Mark Barber, Ronan Collins, Simon Cronin, Anna Czlonkowska, Philippe Desfontaines, Adinda De Pauw, Nicholas Richard Evans, Urs Fischer, Catarina Fonseca, John Forbes, Michael D Hill, Darius Jatuzis, Janika Körv, Peter Kraft, Christina Kruuse, Catherine Lynch, Dominick McCabe, Robert Mikulik, Sean Murphy, Paul Nederkoorn, Martin O'Donnell, Peter Sandercock, Bernadette Schroeder, Gek Shim, Katrina Tobin, David J Williams, Christopher Price

CONVINCE
2024

ORIGINAL ARTICLE

Colchicine in Patients with Chronic Coronary Disease

S.M. Nidorf, A.T.L. Fiolet, A. Mosterd, J.W. Eikelboom, A. Schut, T.S.J. Opstal,
S.H.K. The, X.-F. Xu, M.A. Ireland, T. Lenderink, D. Latchem, P. Hoogslag,
A. Jerzewski, P. Nierop, A. Whelan, R. Hendriks, H. Swart, J. Schaap, A.F.M. Kuijper,
M.W.J. van Hessen, P. Saklani, I. Tan, A.G. Thompson, A. Morton, C. Judkins,
W.A. Bax, M. Dirksen, M.M.W. Alings, G.J. Hankey, C.A. Budgeon, J.G.P. Tijssen,
J.H. Cornel, and P.L. Thompson, for the LoDoCo2 Trial Investigators*

LoDoCo2
2020

ORIGINAL ARTICLE

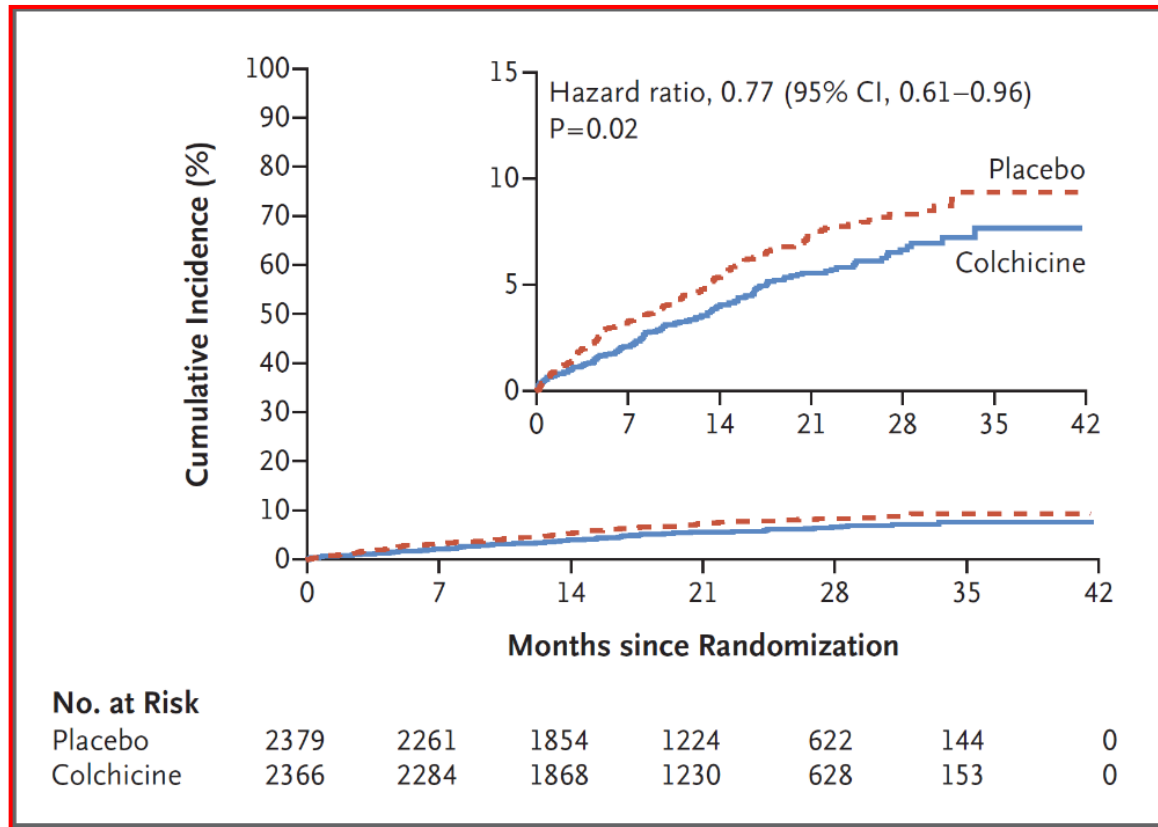
Colchicine in Acute Myocardial Infarction

S.S. Jolly, M.-A. d'Entremont, S.F. Lee, R. Mian, J. Tyrwhitt, S. Kedev,
G. Montalescot, J.H. Cornel, G. Stanković, R. Moreno, R.F. Storey, T.D. Henry,
S.R. Mehta, M. Bossard, P. Kala, J. Layland, B. Zafirovska, P.J. Devereaux,
J. Eikelboom, J.A. Cairns, B. Shah, T. Sheth, S.K. Sharma, W. Tarhuni, D. Conen,
S. Tawadros, S. Lavi, and S. Yusuf, for the CLEAR Investigators*

CLEAR
2024

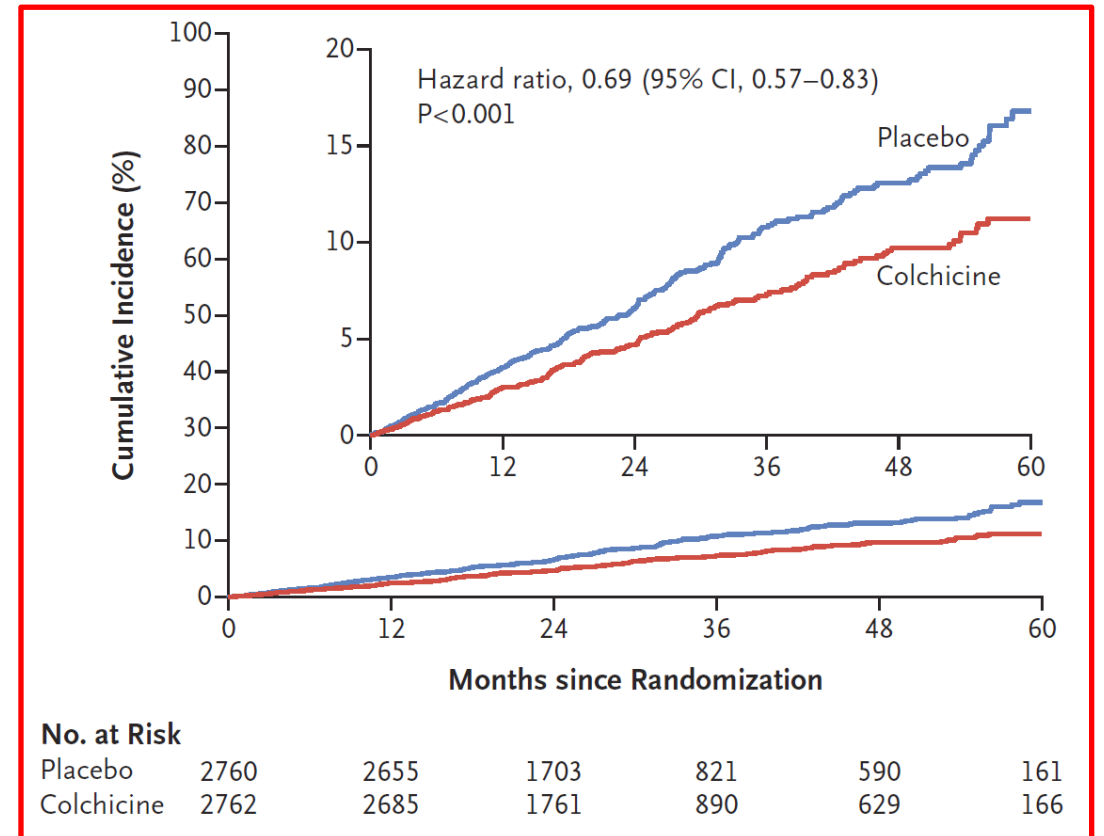
Low Dose Colchicine 0.5 mg po qd for CVD Risk Reduction

COLCOT (N = 4,745 recent MI)



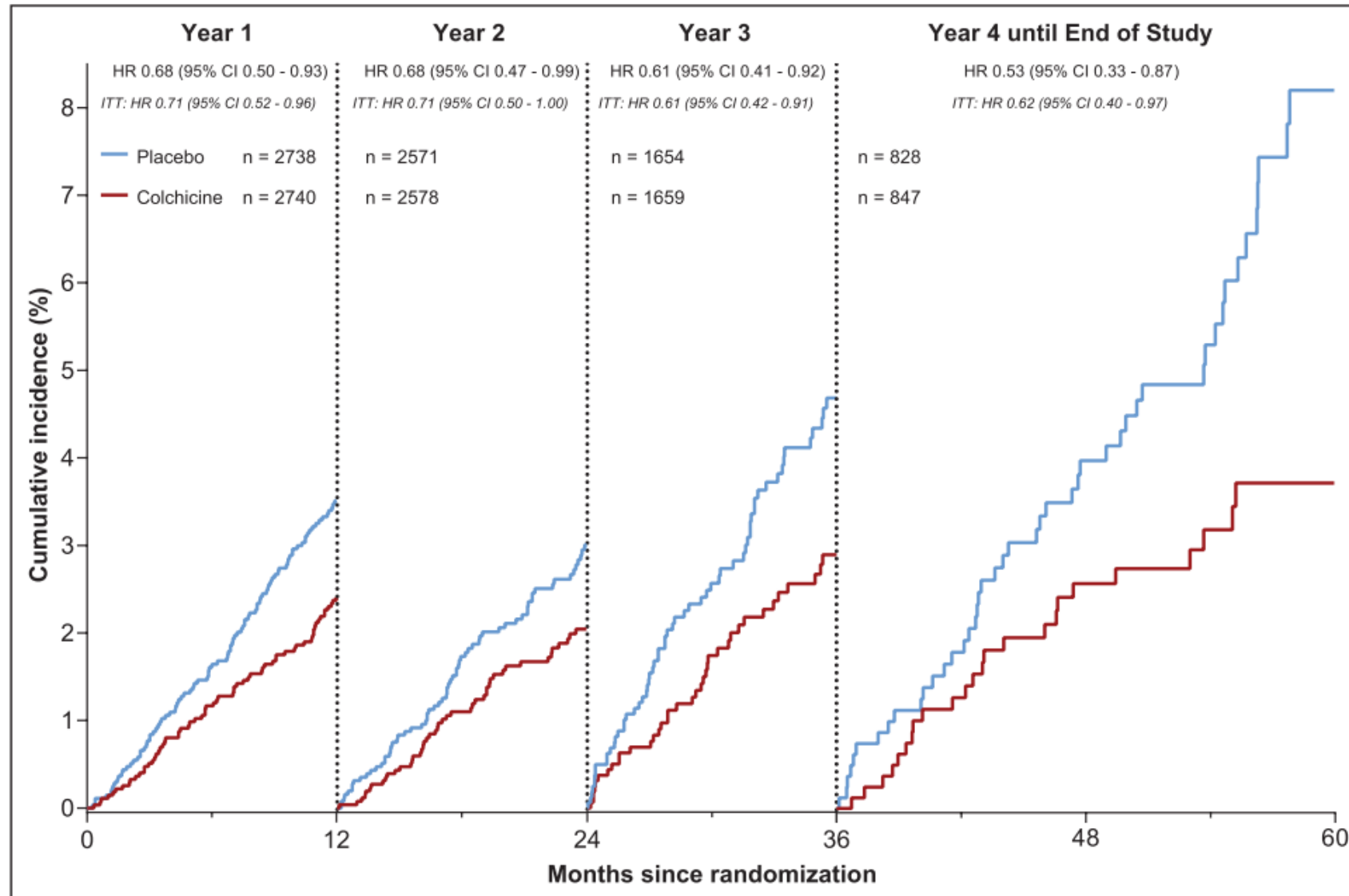
Tardif JC et al NEJM 2019;381:2497-2505

LoDoCo2 (N = 5,522 stable CAD)



Nidorf SM et al NEJM 2021;384:776-779

Long-Term Efficacy of Low Dose Colchicine in Patient with Chronic Coronary Disease : LODOCO2



No increase risk of infection

No increase risk of neutropenia

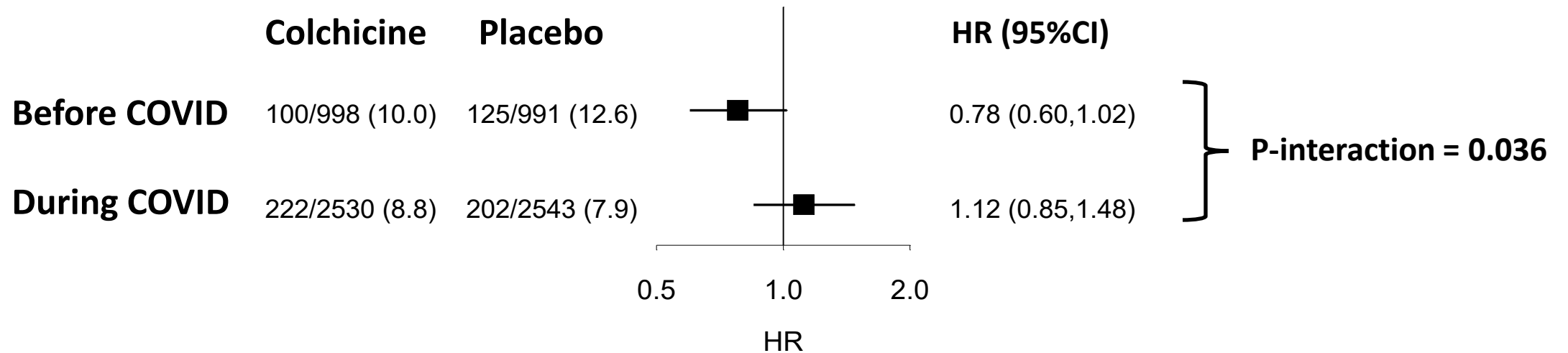
No increase risk of cancer

No increase risk of myopathy

Infrequent short term GI
Intolerance

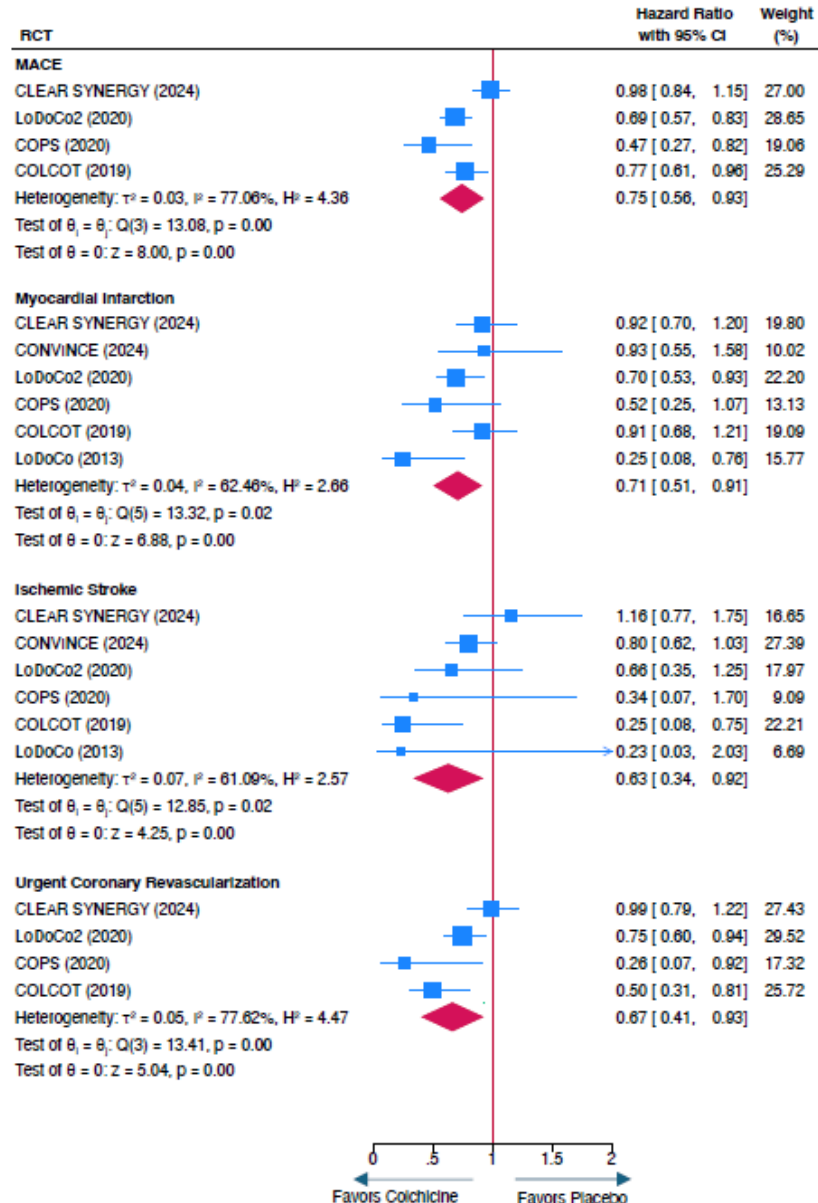
Neutral for all-cause mortality

Potential Impact of the COVID Pandemic on Outcomes in CLEAR-SYNERGY Low Dose Colchicine Trial in Acute STEMI



Adapted from Jolly et al, NEJM 2025;392:633-642 using WHO Definitions of COVID Onset and Completion

Updated Meta-Analysis of Low Dose Colchicine and Atherosclerotic Events 2025



MACE

HR

0.75 (0.56 – 0.93)

MI

HR

0.71 (0.51 – 0.91)

Ischemic CVA

HR

0.63 (0.34 – 0.92)

Urgent Revasc

HR

0.67 (0.41 – 0.93)



ESC

European Society
of Cardiology

European Heart Journal (2024) 45, 1596–1601

<https://doi.org/10.1093/eurheartj/ehae208>

STATE OF THE ART REVIEW

Ischaemic heart disease

Low-dose colchicine for atherosclerosis: long-term safety

Safe use of long-term low-dose (0.5 mg daily) colchicine

Very safe

Normal renal and
liver function

Temporary hold

Avoid concomitant
use with
Clarithromycin
Ketoconazole
Fluconazole
Cyclosporine
Ritonavir

Avoid or cease

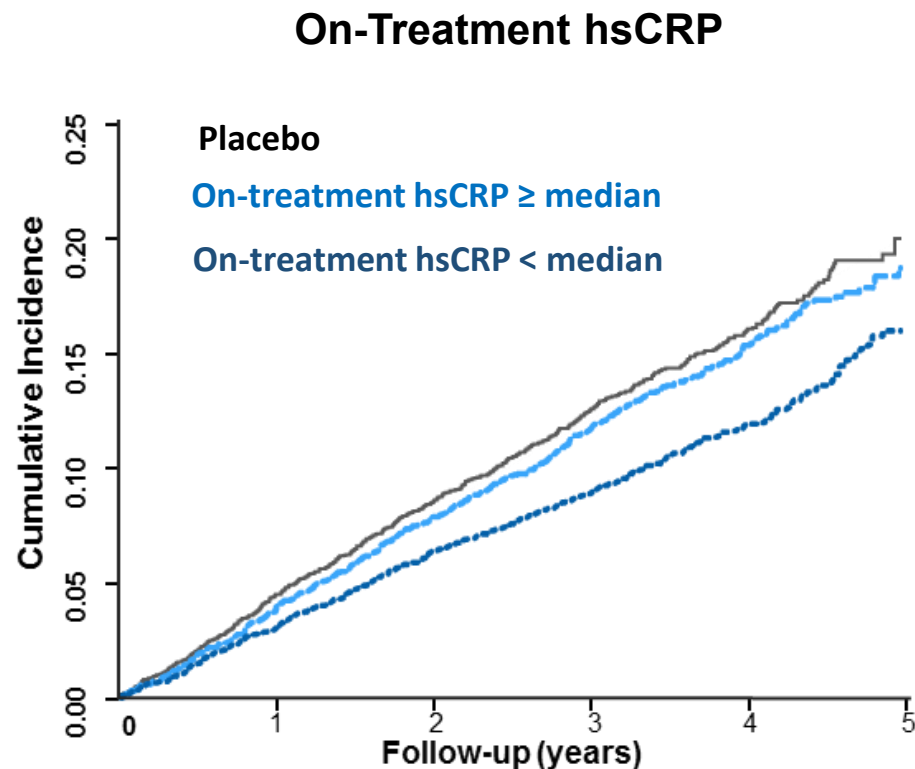
eGFR < 45 mL/min
Cytopenia
Advanced liver dysfunction
CK or ALT > 3xULN

No effect on renal or liver function, risk of bleeding, wound healing, fertility, pregnancy, neonatal health, cognition; no increase in cancer or serious infection

Risk of bone marrow suppression and myotoxicity increases if used with Clarithromycin, Ketoconazole, Fluconazole, Cyclosporine or Ritonavir especially if eGFR falls < 45 mL/min

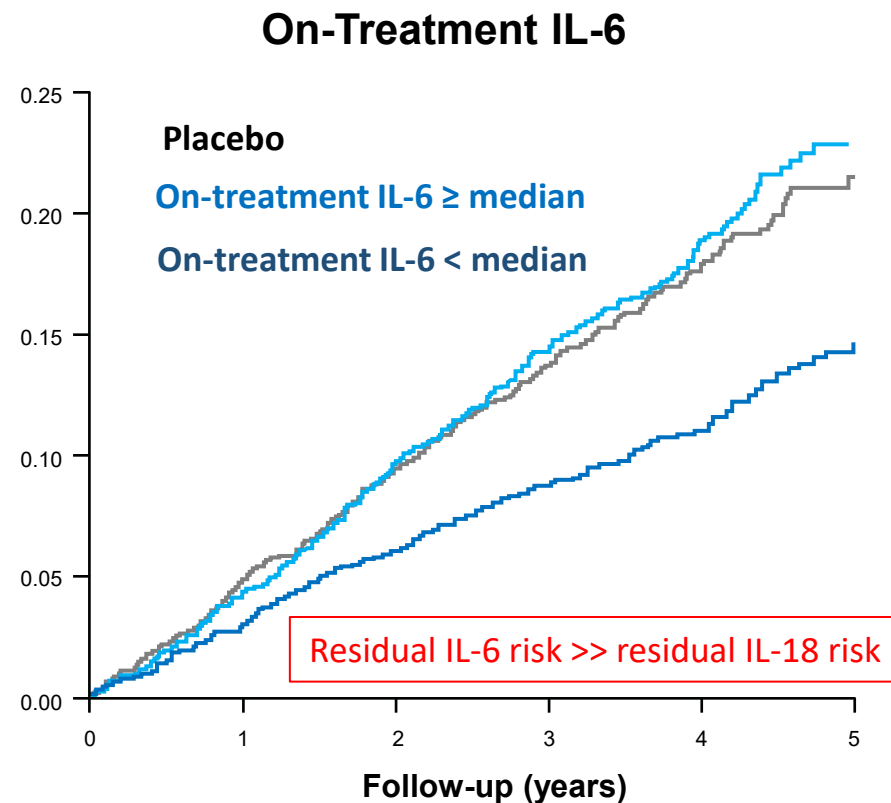
Why So Much Focus on IL-6?

CANTOS: Greater Risk Reduction With Greater Cytokine Inhibition



MACE
25% reduction in risk for those achieving hsCRP below median
5 % reduction in risk for those achieving hsCRP above median
(No change in LDL cholesterol)

Lancet 2018;391:319-328



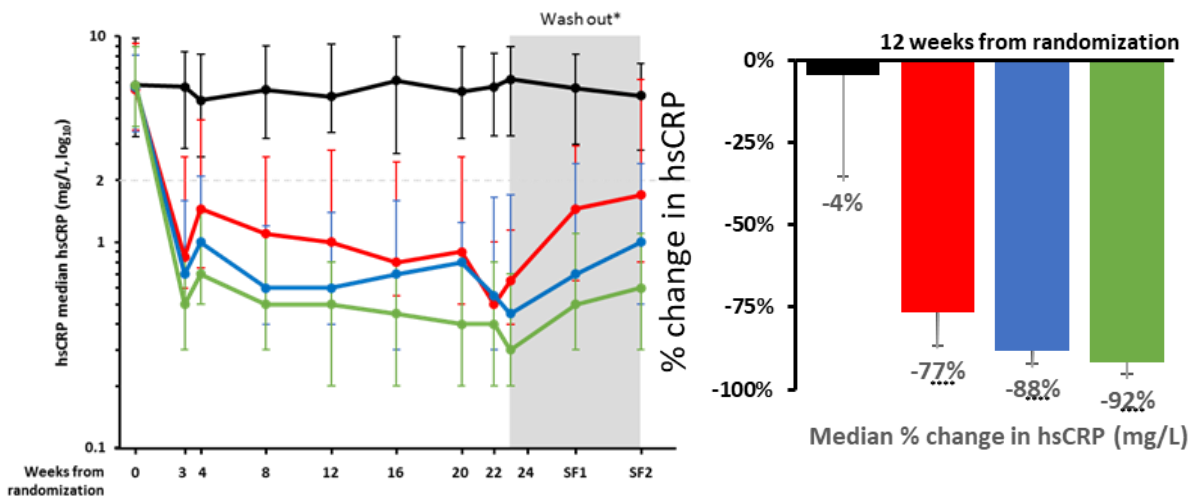
MACE
36% reduction for those achieving IL-6 below median
No benefit for those achieving IL-6 above median
(No change in LDL cholesterol)

Eur Heart J 2018;39:3499-3507

Addressing IL-6 Inhibition in ASCVD and CKD

IL-6 inhibition with ziltivekimab in patients at high atherosclerotic risk (RESCUE): a double-blind, randomised, placebo-controlled, phase 2 trial

THE LANCET



Lancet 2021;397:2060-2069

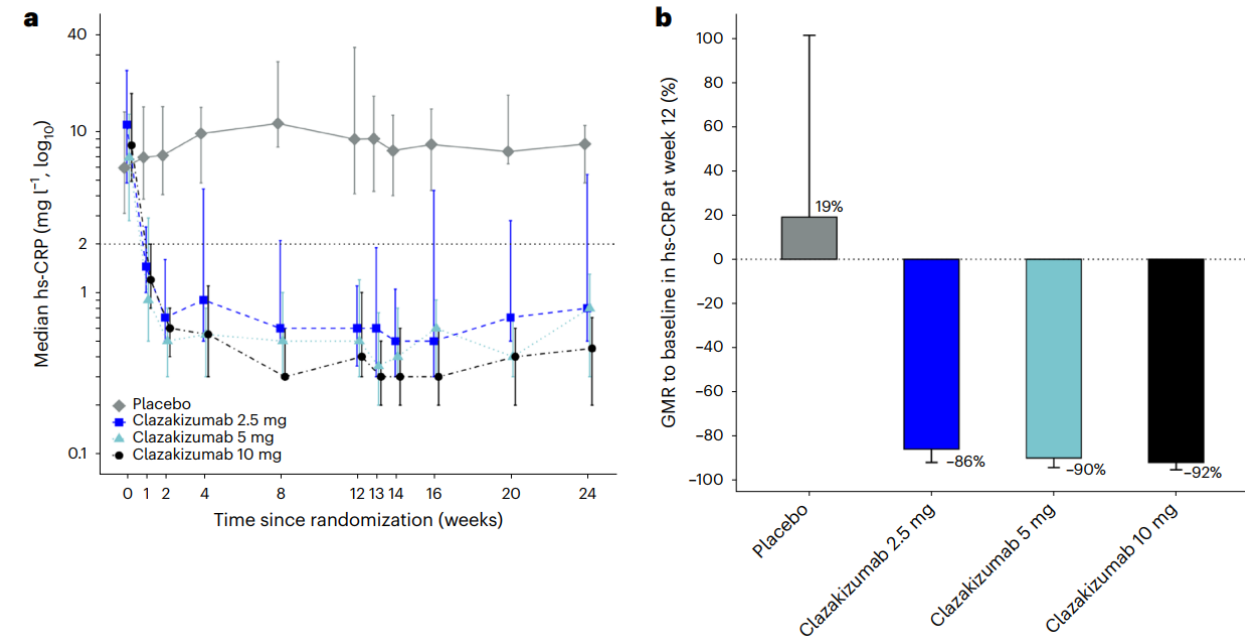
nature medicine



Article

<https://doi.org/10.1038/s41591-024-03043-1>

IL-6 inhibition with clazakizumab in patients receiving maintenance dialysis: a randomized phase 2b trial



Chertow et al, Nat Med 2024;30:2328-2336

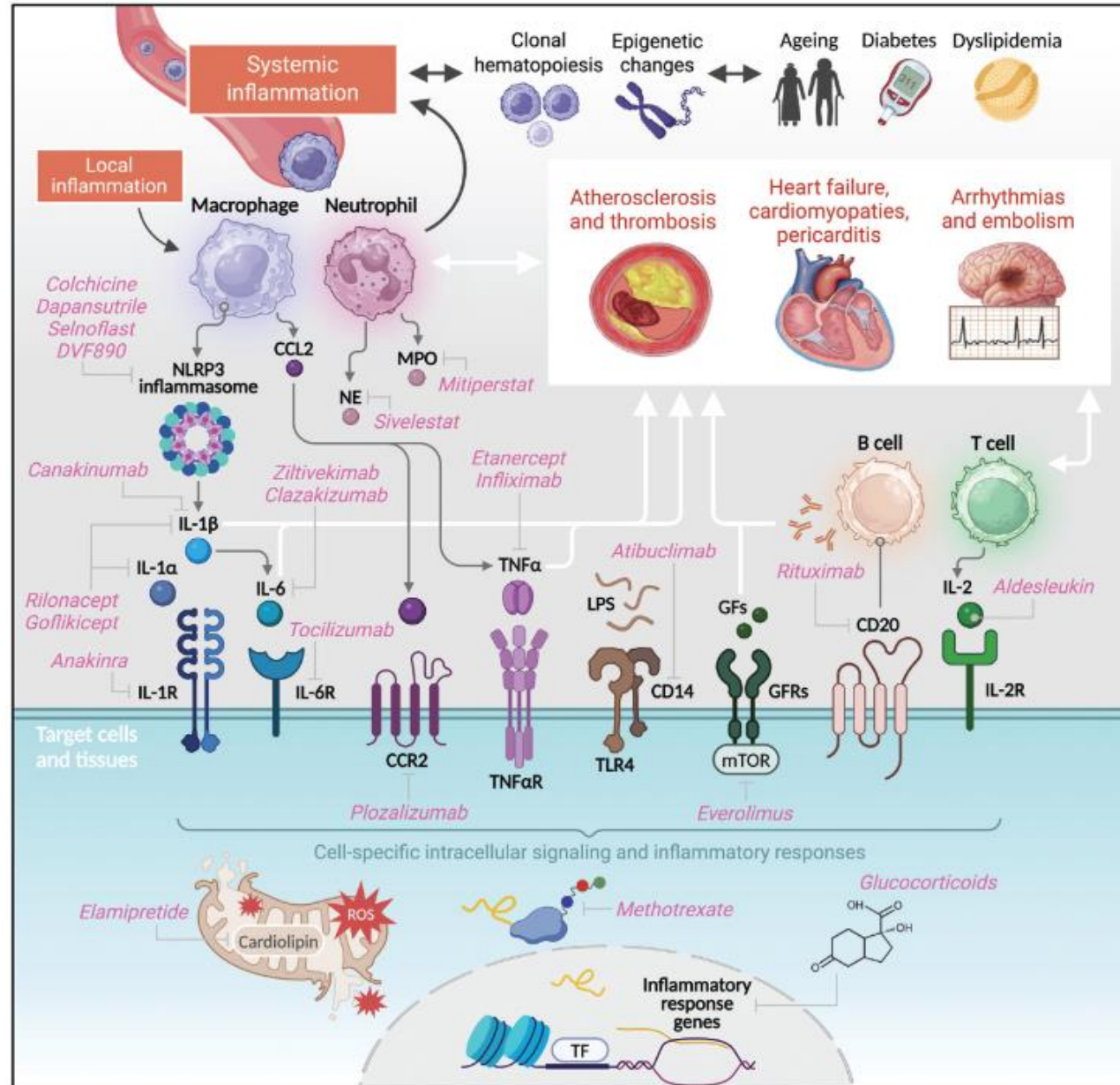
Ongoing and anticipated randomized, double-blind, placebo-controlled trials of Interleukin-6 inhibition with incident major adverse cardiovascular events as the primary endpoint.

IL-6 Inhibitor	Trial	Population	Sample Size
Ziltivekimab Novo Nordisk	ZEUS NCT05021835	ASCVD, CKD, hsCRP > 2 mg/L	6376 Fully enrolled
	HERMES NCT05636176	HFpEF/HFmrEF, hsCRP > 2 mg/L	4900 Enrolling
	ARTEMIS NCT06118281	Acute STEMI and non-STEMI	8344 Fully Enrolled
Clazakizumab CSL Behring	POSIBIL₆ESKD NCT05485961	Dialysis, diabetes or ASCVD, hsCRP > 2 mg/L	2190 Enrolling
Pacibekitug Tourmaline / Novartis	In Development		

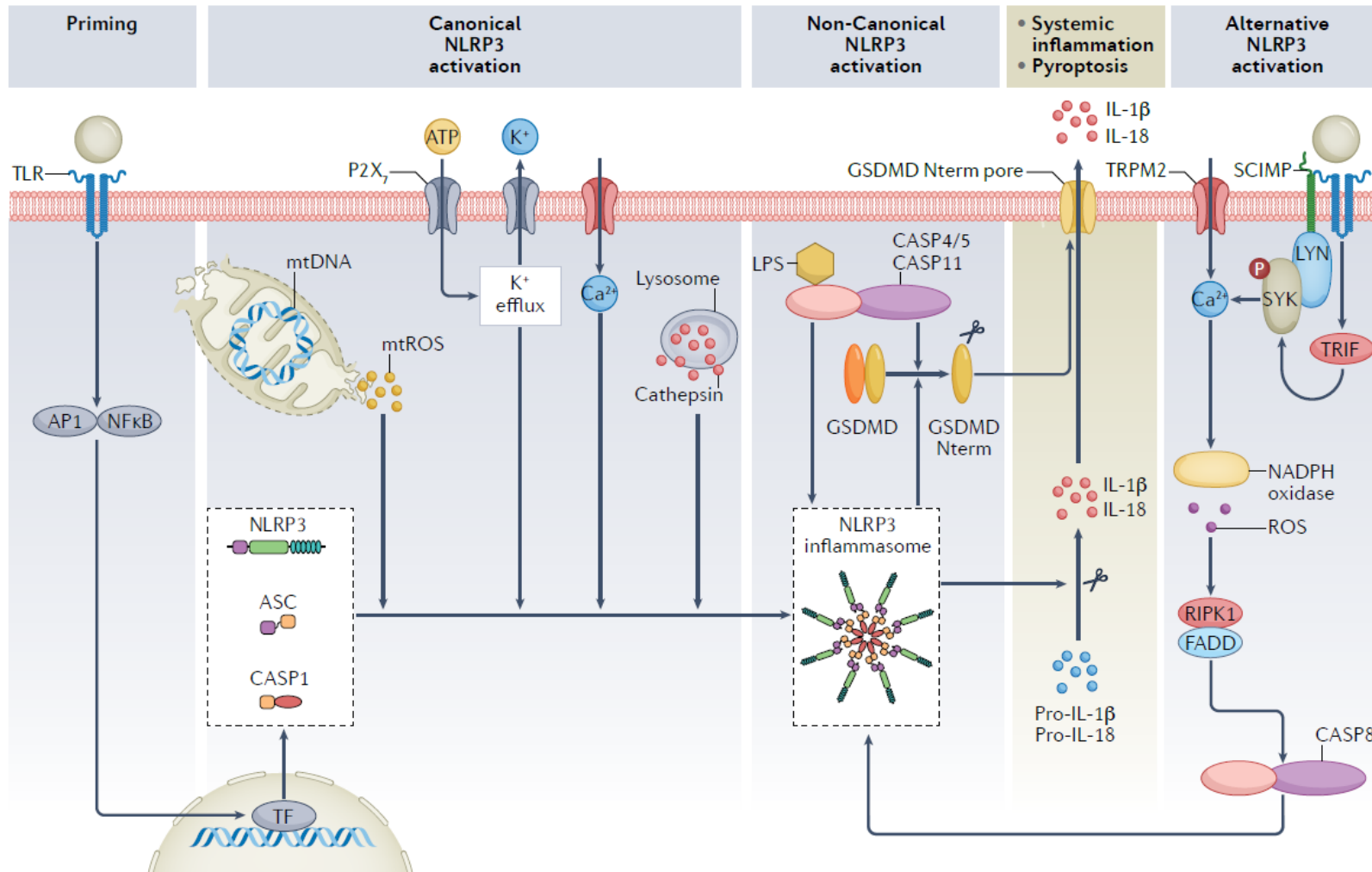
IL-6 = interleukin-6; ASCVD = atherosclerotic cardiovascular disease; CKD = chronic kidney disease; hsCRP = high sensitivity C-reactive protein; HFpEF = heart failure with preserved ejection fraction; HFmrEF = heart failure with mid-range ejection fraction; STEMI = ST elevation myocardial infarction

The growing anti-inflammatory drug armamentarium to combat cardiovascular diseases

Canakinumab
Ziltivekimab
Colchicine
MTX
Aldesleukin
Anakinra
NLRP3 inhibition
Inflammation Resolution



The NLRP3 Inflammasome: Potential Role of NLRP3 Inhibition in Atherosclerosis



Name (chemical name)	Chemical structure
16673-34-0 5-Chloro-2-methoxy-N-[2-(4-sulfamoylphenyl)ethyl]benzamide	
BAY 11-7082 3-[(4-Methylphenyl)sulfonyl]-(2E)-propenenitrile	
Colchicine N-[(7S)-1,2,3,10-tetramethoxy-9-oxo-6,7-dihydro-5H-benzo[a]heptalen-7-yl]acetamide	
INF4E Ethyl 2-[(2-chlorophenyl)(hydroxymethyl)acrylate	
MCC950 N-[(1,2,3,5,6,7-hexahydro-s-indacen-4-yl)carbamoyl]-4-(2-hydroxypropan-2-yl)furan-2-sulfonamide; N-[(1,2,3,5,6,7-hexahydro-s-indacen-4-yl)amino]carbonyl]-4-(1-hydroxy-1-methylethyl)-2-furansulfonamide	
OLT1177 (dapansutritile) 3-(methanesulfonyl)propanenitrile	

“The Western diet appears to be mistakenly recognized by the immune system as a threat to the organism”

Anette Christ, Eicke Latz, Nature Rev Immunology 2019

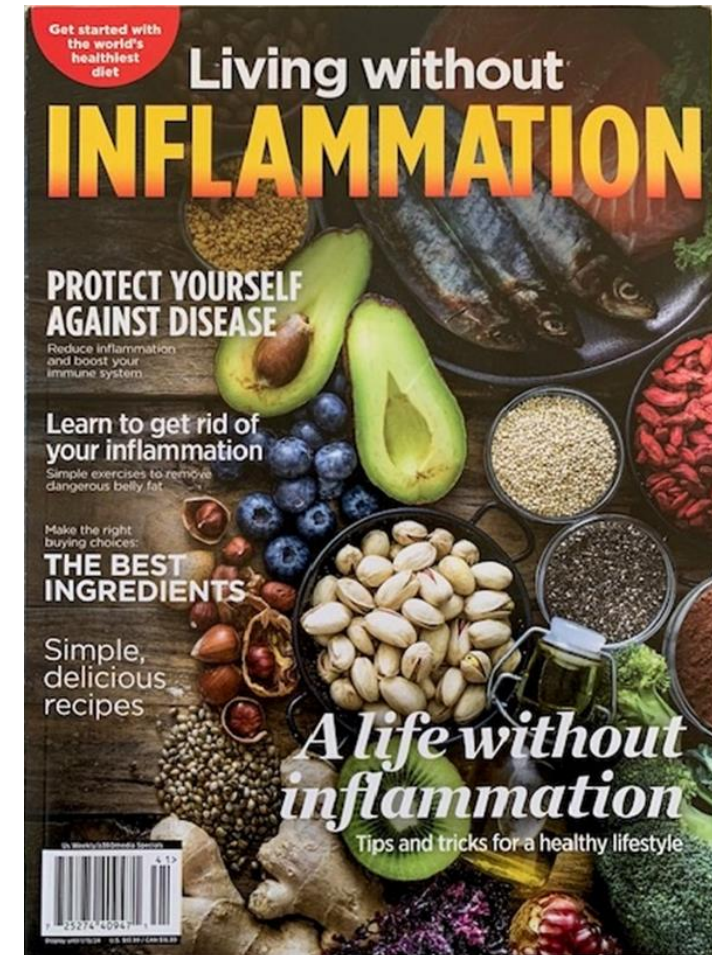
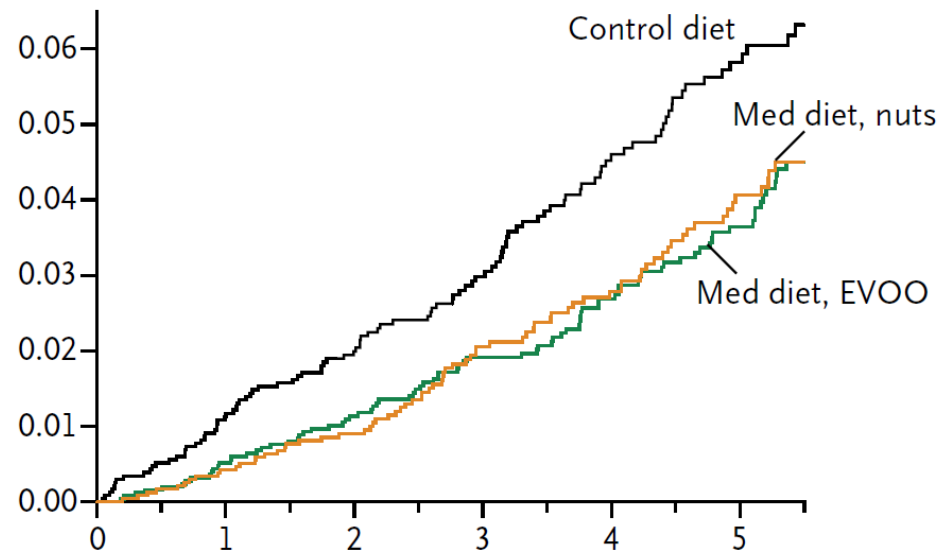
THE NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

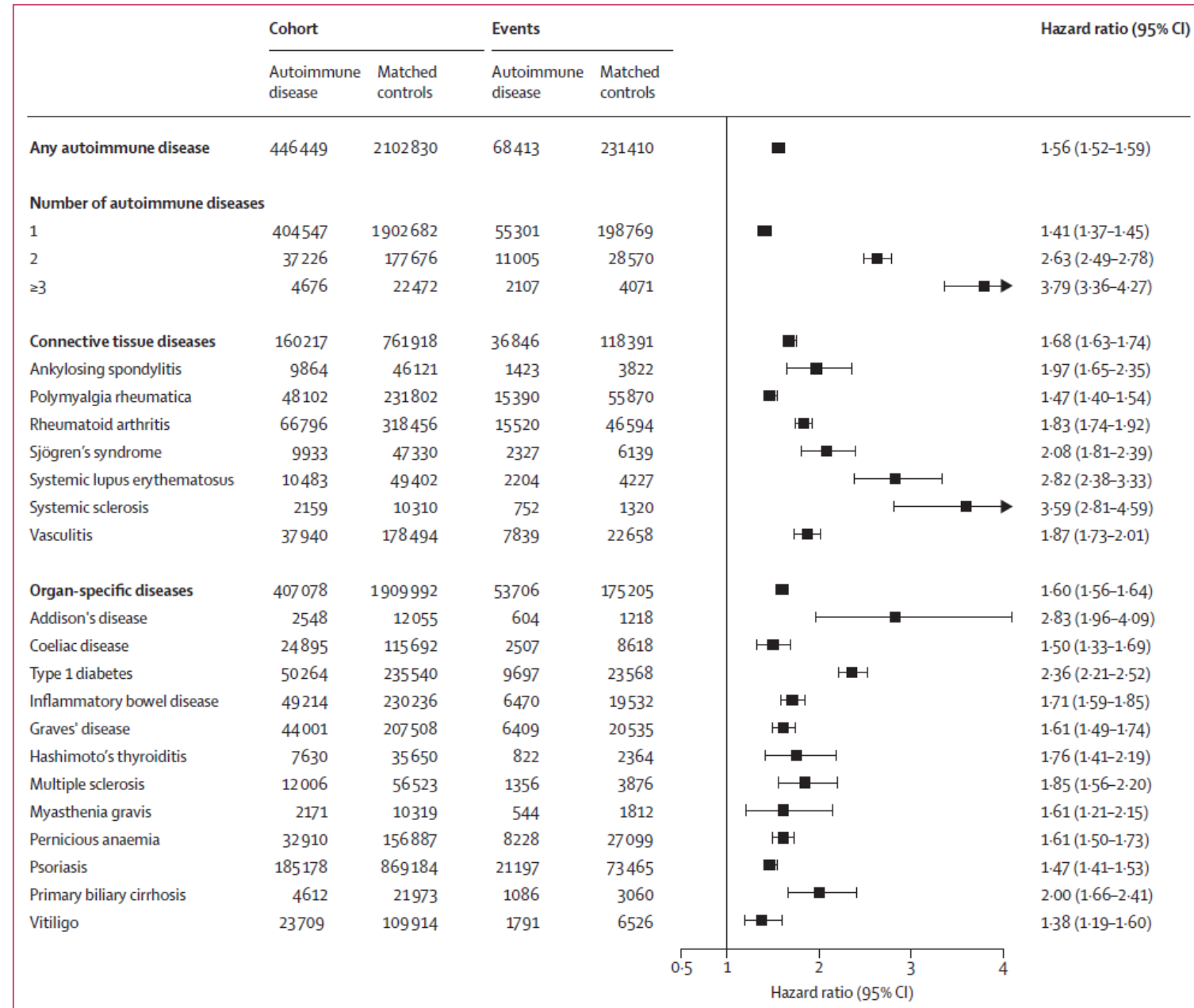
Primary Prevention of Cardiovascular Disease with a Mediterranean Diet

Ramón Estruch, M.D., Ph.D., Emilio Ros, M.D., Ph.D., Jordi Salas-Salvadó, M.D., Ph.D., Maria-Isabel Covas, D.Pharm., Ph.D., Dolores Corella, D.Pharm., Ph.D., Fernando Arós, M.D., Ph.D., Enrique Gómez-Gracia, M.D., Ph.D., Valentina Ruiz-Gutiérrez, Ph.D., Miquel Fiol, M.D., Ph.D., José Lapetra, M.D., Ph.D., Rosa Maria Lamuela-Raventós, D.Pharm., Ph.D., Lluís Serra-Majem, M.D., Ph.D., Xavier Pintó, M.D., Ph.D., Juan Rivera, M.D., Ph.D., Miquel Angel Muñoz, M.D., Ph.D., José V. Díez, M.D., Ph.D., Miguel Ángel Martínez-González, M.D., Ph.D.

Estruch et al,
NEJM 2013;368:1279-90



Autoimmune Diseases and Cardiovascular Risk Among 22 Million Individuals In the United Kingdom



Conrad et al,
Lancet 2022;400:733-43

Residual Inflammatory Risk and Residual Cholesterol Risk are Not in Competition

- It is not an either/or situation: Lipid lowering and inflammation inhibition are not in conflict but are synergistic. In the future, the combined use of aggressive LDL-lowering and inflammation inhibiting therapies may well become standard of care for almost all atherosclerosis patients.
- In addition to increasing the use of targeted anti-inflammatory therapies for atherosclerosis (low dose colchicine, IL-1 and IL-6 inhibition), evidence regarding residual inflammatory risk and residual cholesterol risk strongly support ongoing ACC/AHA prevention efforts directed at diet, weight loss, exercise, and smoking cessation, all of which lower vascular inflammation and lower cardiovascular event rates.

MOC REFLECTIVE STATEMENT (BRIEF TAKE HOME NOTES FOR REFERENCE)

Levels of the inflammatory biomarker hsCRP < 1, 1 to 3, and > 3 mg/L represent low, moderate, and high risk of future atherosclerotic disease, independent of cholesterol and blood pressure.

Individuals with elevated hsCRP should be strongly considered for statin therapy even if their LDL cholesterol levels are low. The same is true for individuals with elevated hsCRP and no other standard modifiable risk factors, a clinical syndrome known as “SMuRF-Less but Inflamed”.

Among statin treated patients with known atherosclerotic disease, hsCRP is a more powerful predictor of recurrent cardiovascular events than is LDL cholesterol.

Such individuals are candidates for systemic anti-inflammatory therapy which targets the IL-1 to IL-6 pathway of innate immunity.

While several major trials are ongoing, low dose colchicine (0.5 mg po qd) is the only current FDA approved anti-inflammatory agent for atherosclerosis treatment and prevention.



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