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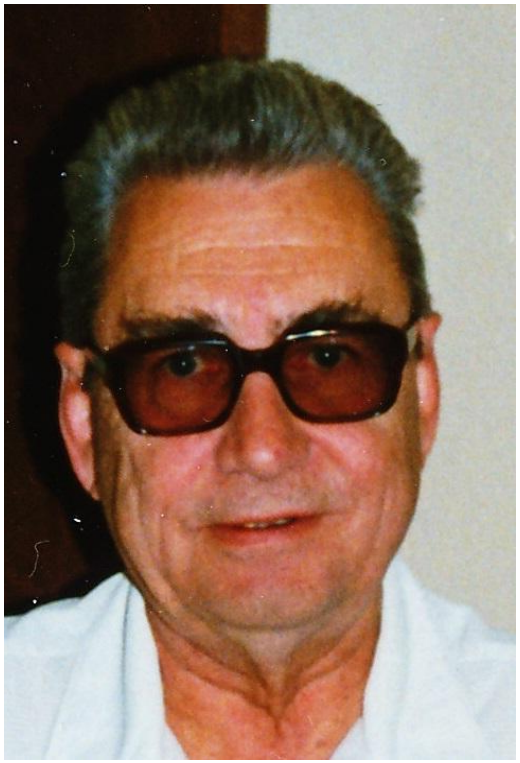

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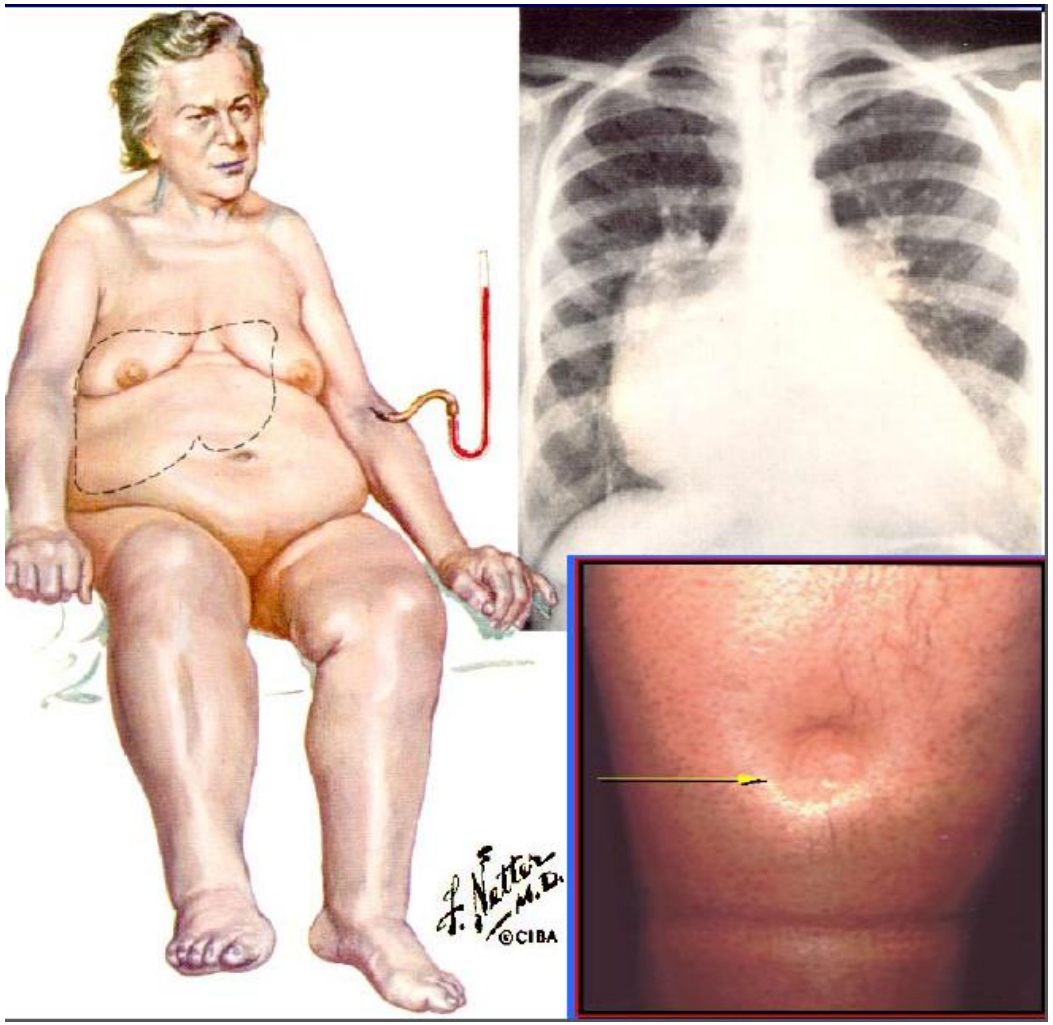
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American Journal of Clinical Nutrition

Volume 30, Issue 5, 1977, Pages 664-673

The influence of egg consumption on the serum cholesterol level in human subjects (Article)

Kummerow, F.A., Kim, Y., Hull, J., Pollard, J., Ilinov, P., Drossiev, D.L., Valek, J.

Bursides Res. Lab., Univ. Illinois, Urbana, Ill. 61801, United States

Abstract

The influence of whole fresh eggs on the serum cholesterol level in men and women was studied independently in hospitalized patients in Sofia, Prague and Urbana-Champaign. The patients were fed two eggs, or the equivalent of 2 eggs, in a custard base or milk shake in addition to the foods that were consumed in their diet pattern. The serum cholesterol level was determined before and at periods varying from 5 hr to 54 days after the consumption of the eggs. The mixed fatty acid composition of the total lipids in the serum and the erythrocytes was also determined. In the majority of patients, the serum cholesterol level did not change significantly 5 hr after the consumption of 465 mg of cholesterol in an egg custard base or milk shake or after up to 54 days of continued consumption of two whole eggs per day. The serum cholesterol level of some subjects increased and others decreased at all three experimental sites. A comparison of the mixed fatty acid composition of the total serum lipids obtained from men and women who had received treatment for other reasons than cardiovascular disease with those that had been treated for cardiovascular disease indicated that the serum from both groups contained a substantial amount of polyunsaturated fatty acids. The lipids extracted from the red blood cells obtained from patients in Urbana-Champaign and Sofia did not differ significantly in linoleic and arachidonic acid content.

Indexed keywords

EMTREE drug terms: fatty acid; lipid

Cited by 15 documents since 1996

Impact of a traditional dietary supplement with coconut milk and soya milk on the lipid profile in normal free living subjects
Ekanayaka, R.A.I. , Ekanayaka, N.K. , Perera, B.
(2013) Journal of Nutrition and Metabolism

Egg yolk fatty acid profile of avian species - influen on human nutrition
Golzar Adabi, S.H. , Ahbab, M. , Fani, A.R.
(2013) Journal of Animal Physiology and Animal Nutrition

Egg yolk consumption and carotid plaque
Spence, J.D. , Jenkins, D.J.A. , Davignon, J.
(2012) Atherosclerosis

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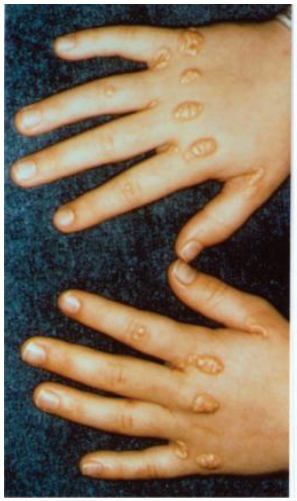
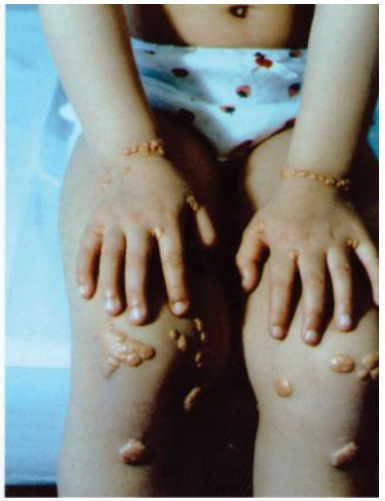
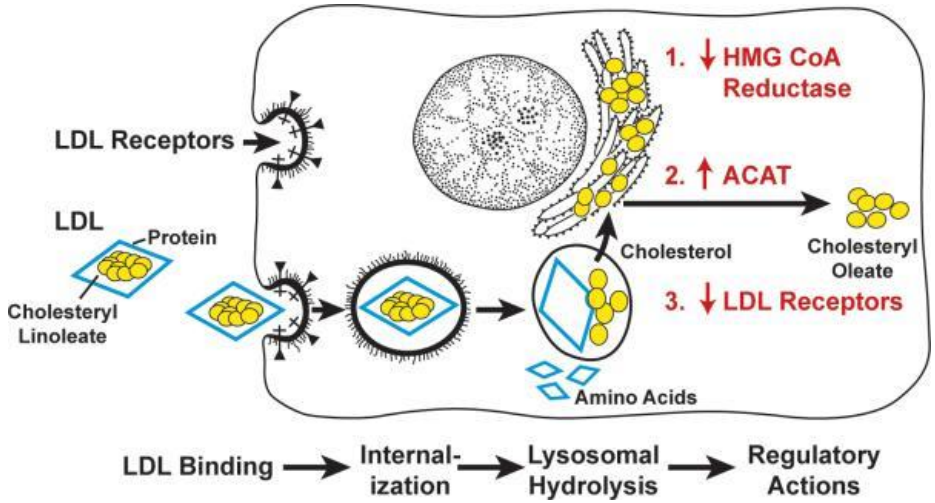





LDL Receptors
LDL
Protein
Cholesteryl Linoleate
1. ↓ HMG CoA Reductase
2. ↑ ACAT
Cholesterol
Cholesteryl Oleate
3. ↓ LDL Receptors
Amino Acids
LDL Binding
Internal-ization
Lysosomal Hydrolysis
Regulatory Actions

Randomised trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S)

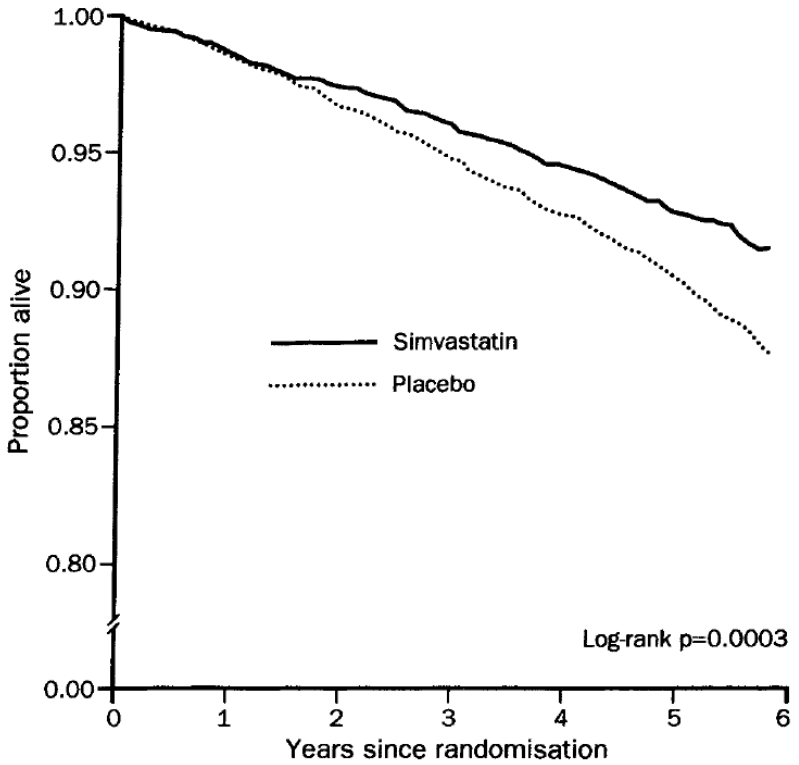
*Scandinavian Simvastatin Survival Study Group**

Summary

Drug therapy for hypercholesterolaemia has remained controversial mainly because of insufficient clinical trial evidence for improved survival. The present trial was

Introduction

High serum cholesterol is regarded by many as the main cause of coronary atherosclerosis.[1] Several cholesterol-lowering interventions have reduced coronary heart



Recommendations on management of hyperlipidaemia

Recommendations	Class[a]	Level[b]	GRADE	Ref[c]
The recommended target levels are <5 mmol/L (less than ~190 mg/dL) for total plasma cholesterol and <3 mmol/L (less than ~115 mg/dL) for LDL cholesterol for subjects at low or moderate risk.	I	A	Strong	457,458
In patients at high CVD risk, an LDL cholesterol goal <2.5 mmol/L (less than ~100 mg/dL) is recommended.	I	A	Strong	459–461
In patients at very high CVD risk, the recommended LDL cholesterol target is <1.8 mmol/L (less than ~70 mg/dL) or a ≥50% LDL cholesterol reduction when the target level cannot be reached.	I	A	Strong	459, 462, 463
All patients with familial hypercholesterolaemia must be recognized as high-risk patients and be treated with lipid-lowering therapy.	I	A	Strong	464, 465
In patients with an ACS, statin treatment in high doses has to be initiated while the patients are in hospital.	I	A	Strong	466–468
Prevention of non-haemorrhagic stroke: treatment with statins must be started in all patients with established atherosclerotic disease and in patients at high risk for developing CVD. Treatment with statins must be started in patients with a history of non-cardioembolic ischaemic stroke.	I	A	Strong	469, 470
Occlusive arterial disease of the lower limbs and carotid artery disease are CHD risk-equivalent conditions and lipid-lowering therapy is recommended.	I	A	Strong	471, 472
Statins should be considered as the first-line drugs in transplant patients with dyslipidaemia.	IIa	B	Strong	473
Chronic kidney disease (stages 2–5, i.e. GFR <90 mL/min/1.73 m^2) is acknowledged as a CHD risk-equivalent and the LDL cholesterol target in these patients should be adapted to the degree of renal failure.	IIa	C	Strong	474

ACS = acute coronary syndrome; CHD = coronary heart disease; CVD, cardiovasular disease; GFR = glomerular filtration rate; LDL = low-density lipoprotein.
[a]Class of recommendation.

European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). European Heart Journal 2012:1-77

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EDITORIAL

Heart Attacks: Gone with the Century?

This issue of *Science* highlights the progress and promise of research in cardiovascular disease, the most frequent cause of death in men over age 35 and women over age 65 in the United States. Heart attacks were recognized as a public health problem only in this century. They are likely to lose this notoriety early in the next. The reason? Four decades of progress in understanding cholesterol and the lipoproteins that carry it in blood plasma.

Michael S. Brown and Joseph L. Goldstein

The authors are professors of molecular genetics at the University of Texas Southwestern Medical Center in Dallas, TX, USA.

BIOMEDICINE

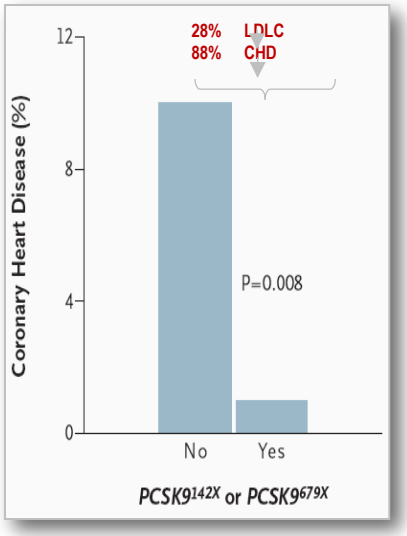
Lowering LDL—Not Only How Low, But How Long?

People with a mutation in a proteolytic enzyme are at a substantially lower risk for coronary heart disease because of their lifelong reduction of plasma low-density lipoprotein.

Michael S. Brown and Joseph L. Goldstein

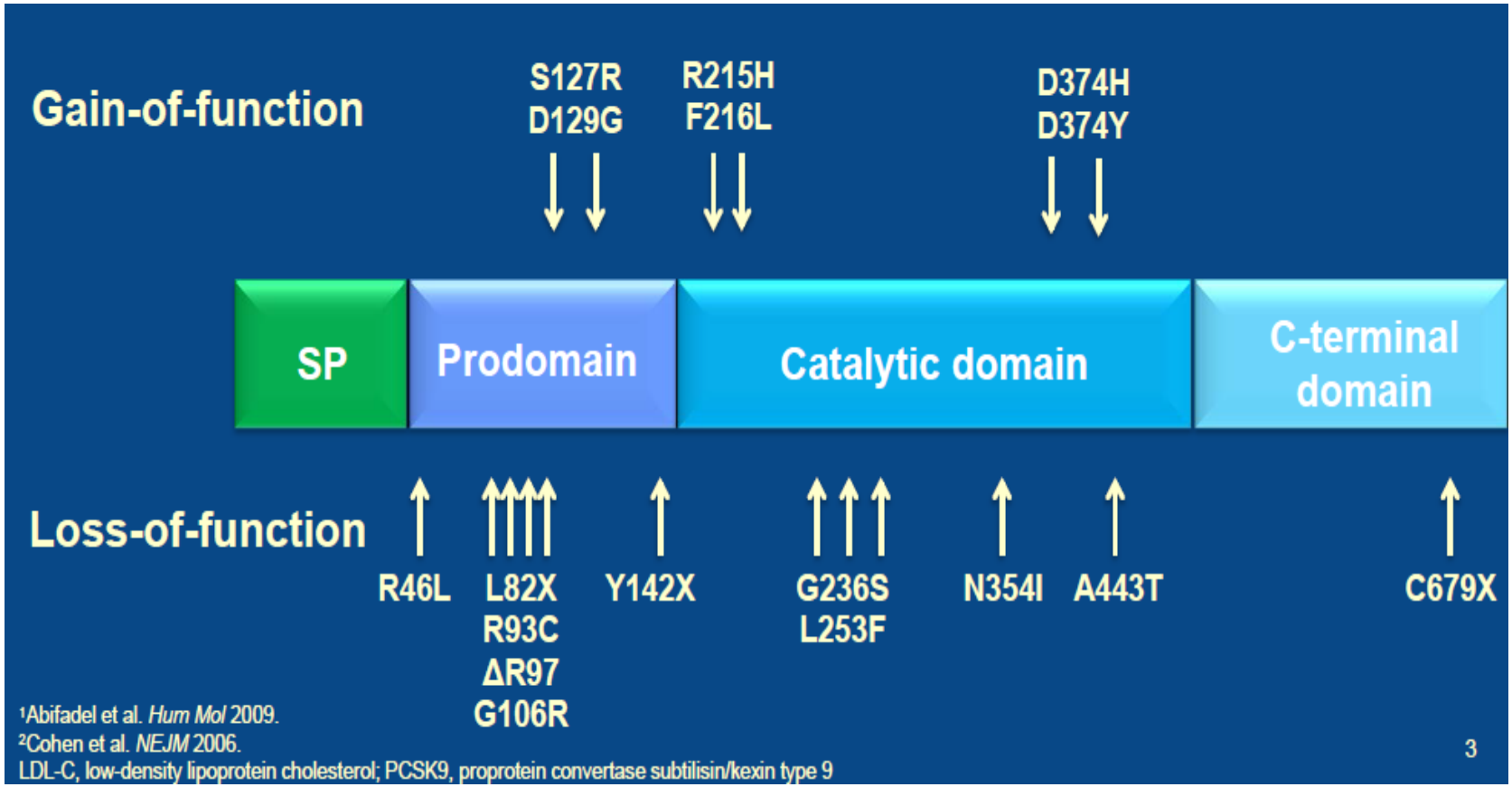
Key Events: PCSK9 Biology and Its Impact on LDL Lowering

2003	2004	2005	2006	2007	2008	2009	2010	2011
Mutations in PCSK9 associated with high serum cholesterol levels in human subjects[1,2] PCSK9 identified as a cholesterol-regulated gene[3]	Overexpression of PCSK9 in mice increases serum cholesterol and reduces liver LDL-R[4]	Nonsense mutations in PCSK9 associated with lower serum cholesterol in human subjects[5] PCSK9 project initiated at Amgen[6]	Loss-of-function PCSK9 mutations associated with lower serum cholesterol and reduced risk of CHD in human subjects[7] PCSK9 and LDL-R interaction identified[6] XenoMouse antibody campaign initiated[6]	Crystal structure of PCSK9 published[8,9]	Clinical development of AMG 145 initiated[6]	AMG 145 enters first-in-human clinical testing[6] Cholesterol-lowering effect of anti-PCSK9 mAb in mice and NHP published in *PNAS*[10]	AMG 145 vs. placebo Phase I safety study initiated[11]	Effect of single dose of AMG 145 on serum LDL-C tested[11] Phase II clinical trials initiated[6]



PCSK9=proprotein convertase subtilisin/kexin type 9; LDL-R=low-density lipoprotein receptor; FH=familial hypercholesterolaemia; NHP=nonhuman primate; mAb=monoclonal antibody

1. Abifadel M et al. *Nat Genet* 2003;34:154–6; 2. Seidah NG & Prat A. *J Mol Med (Berl)* 2007;85:685–96; 3. Horton JD et al. *Proc Natl Acad Sci USA* 2003;100:12027–32; 4. Maxwell KN & Breslow JL. *Proc Natl Acad Sci USA* 2004;101:7100–5; 5. Cohen J et al. *Nat Genet* 2005;37:161–5; 6. Data on file, Amgen; 7. Cohen JC et al. *N Engl J Med* 2006;354:1264–72; 8. Piper DE et al. *Structure* 2007;15:545–52; 9. Cunningham D et al. *Nat Struct Mol Biol* 2007;14:413–9; 10. Chan JC et al. *Proc Natl Acad Sci USA* 2009;106:9820–5; 11. Dias C et al. *Circulation* 2011;124:A10701



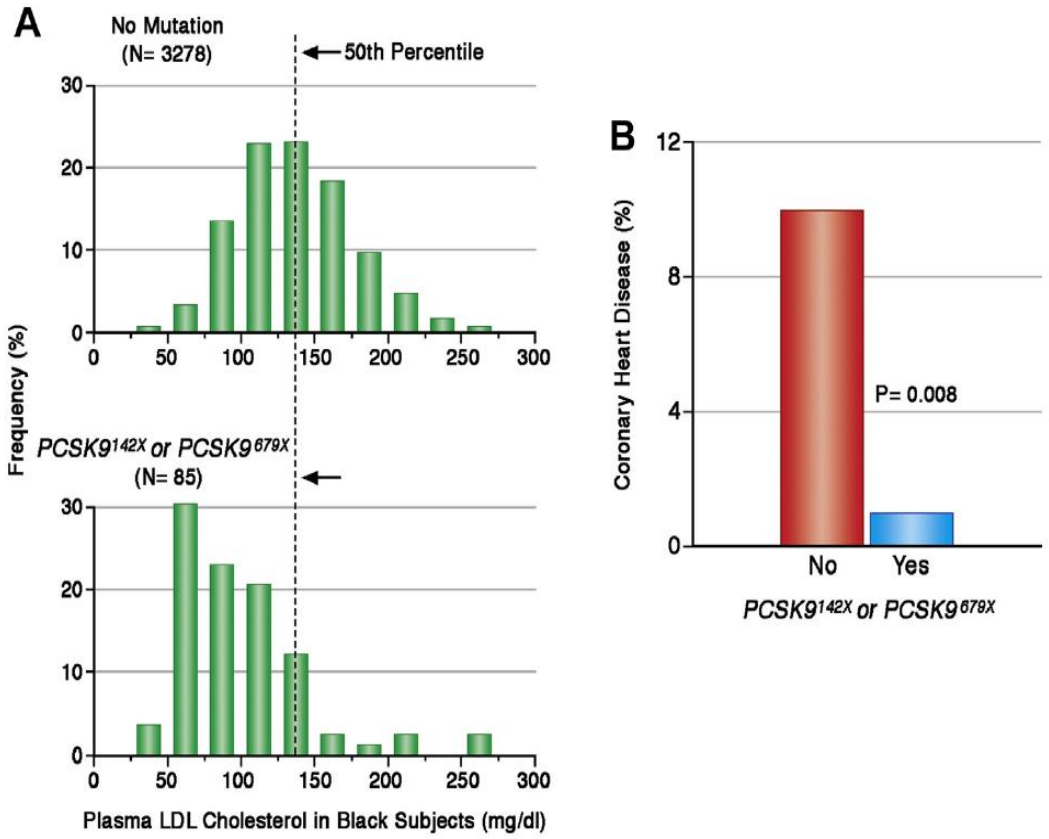
[1]Abifadel et al. *Hum Mol* 2009.
[2]Cohen et al. *NEJM* 2006.
LDL-C, low-density lipoprotein cholesterol; PCSK9, proprotein convertase subtilisin/kexin type 9

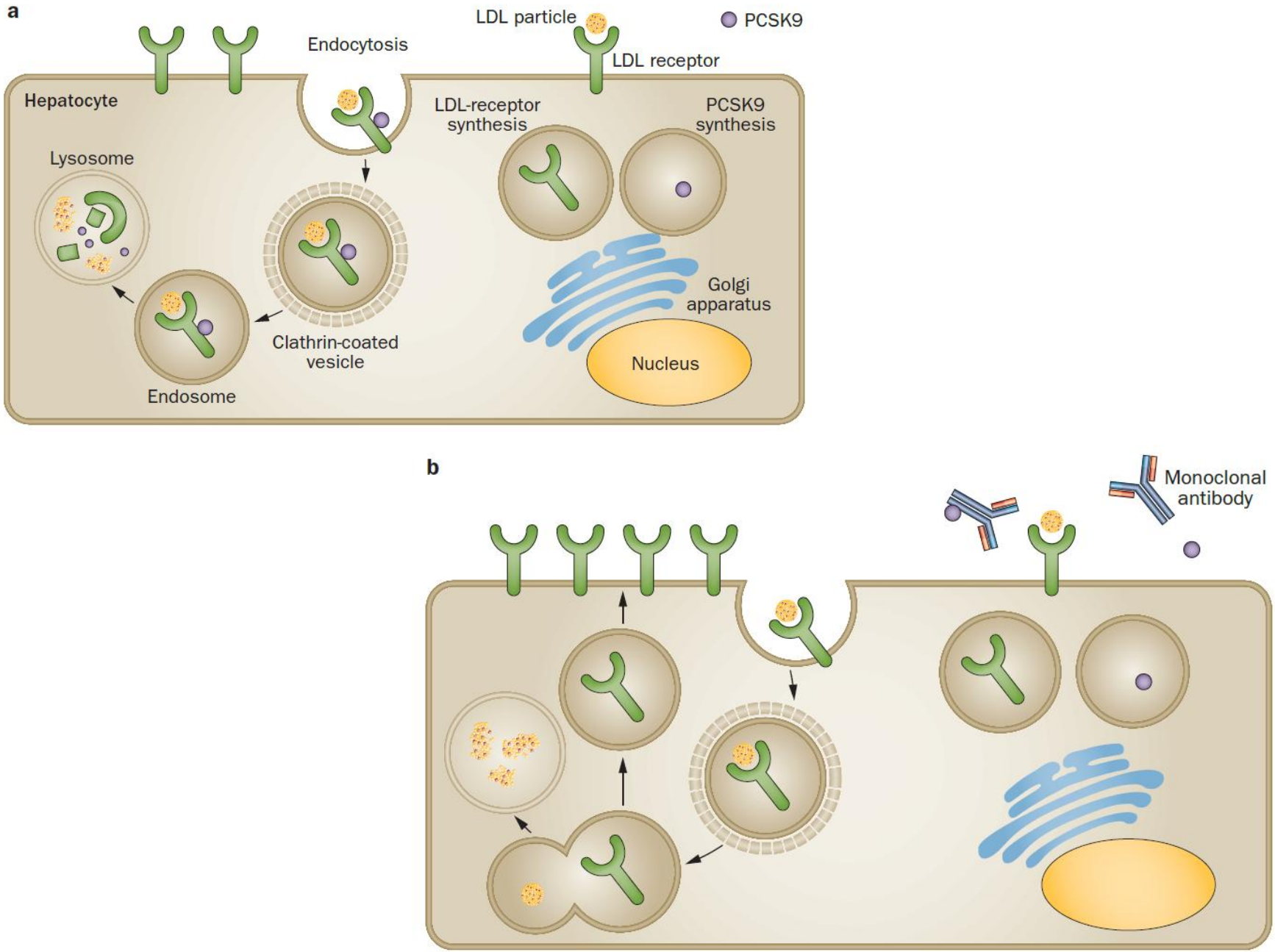
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Sequence Variations in *PCSK9*, Low LDL, and Protection against Coronary Heart Disease

Jonathan C. Cohen, Ph.D., Eric Boerwinkle, Ph.D., Thomas H. Mosley, Jr., Ph.D., and Helen H. Hobbs, M.D.




a
LDL particle
PCSK9
Endocytosis
LDL receptor
Hepatocyte
LDL-receptor synthesis
PCSK9 synthesis
Lysosome
Golgi apparatus
Clathrin-coated vesicle
Nucleus
Endosome
b
Monoclonal antibody

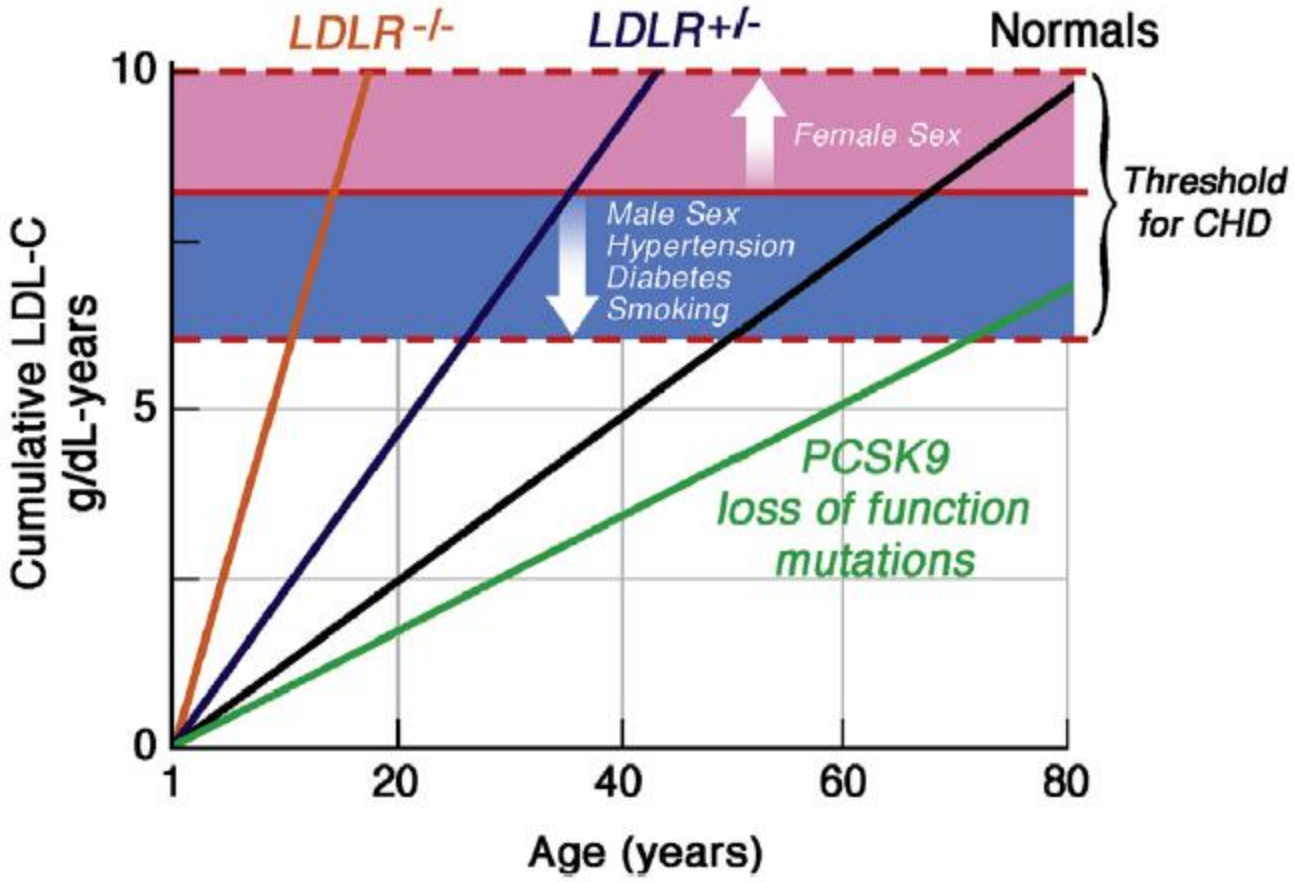
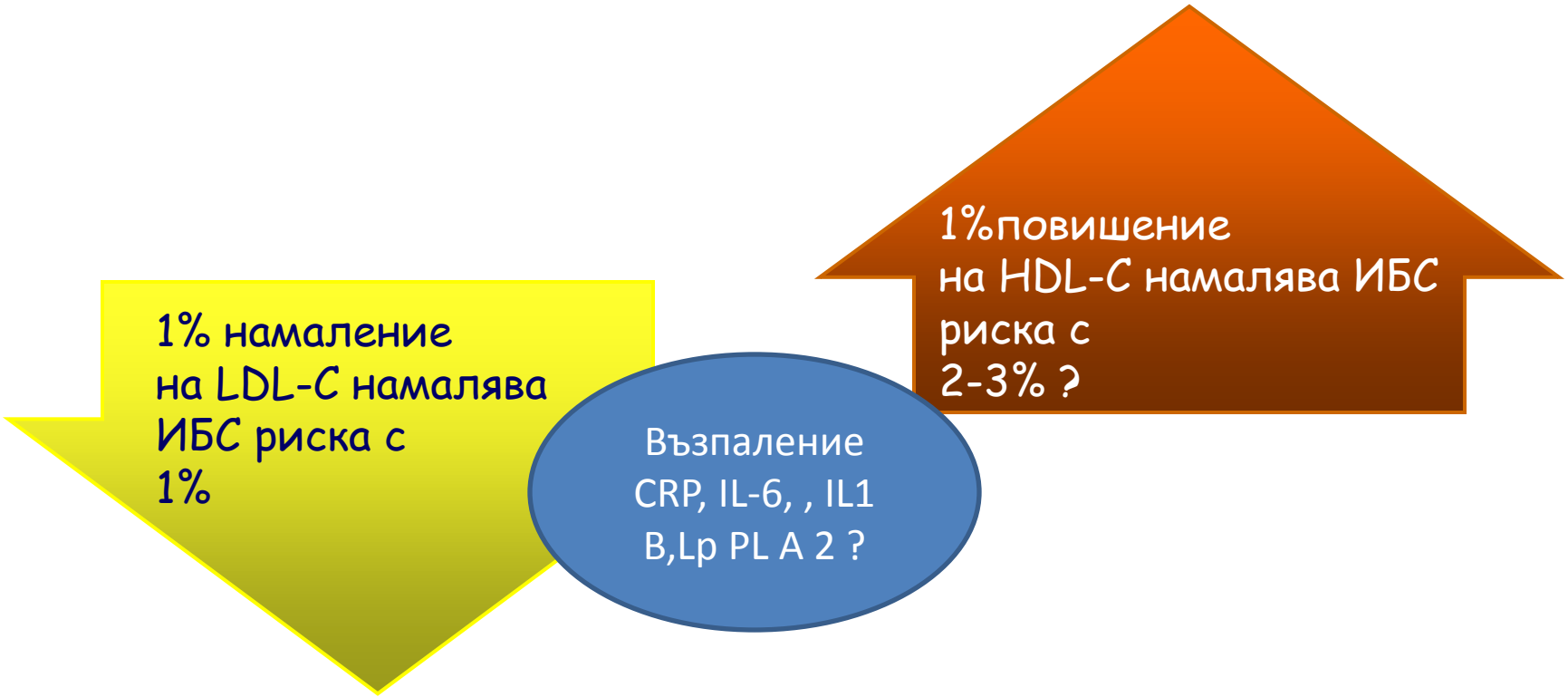


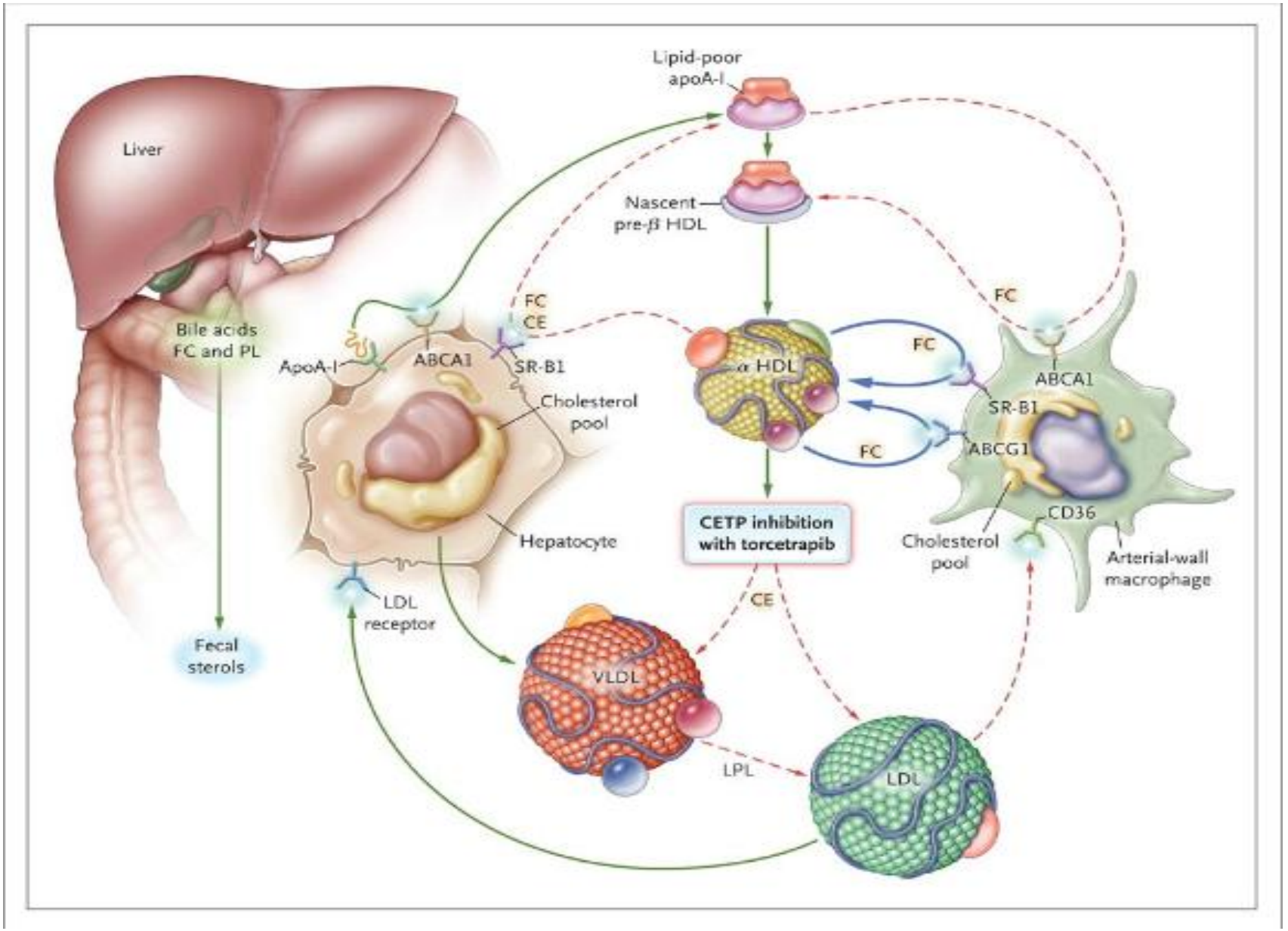
Figure 1 **Relationship Between Age and Cumulative LDL-C Exposure**

Връзка между промяната на нивата на LDL-C и HDL-C и риска за сърдечно заболяване



Third Report of the NCEP Expert Panel. NIH Publication No. 01-3670 2001. http://hin.nhlbi.nih.gov/ncep_slds/menu.htm

Schematic Representation of the Metabolism of HDL Cholesterol



Nissen S E. et al. N Eng J Med 2007; 356 : 1304-16

Effects of Combination Lipid Therapy in Type 2 Diabetes Mellitus

The ACCORD Study Group*

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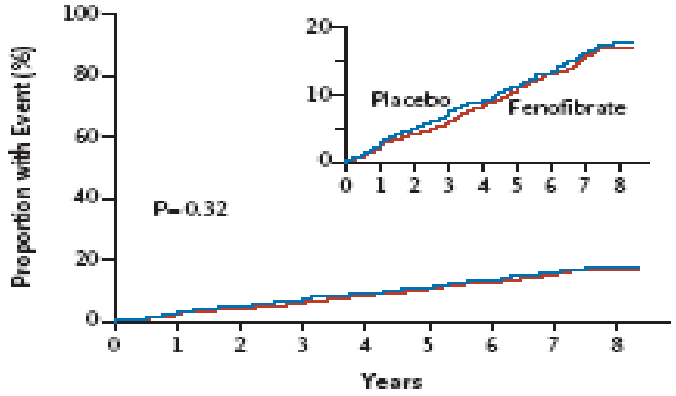
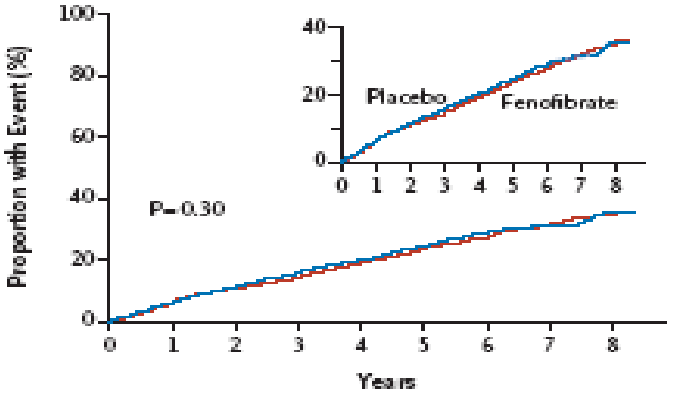
A Primary Outcome



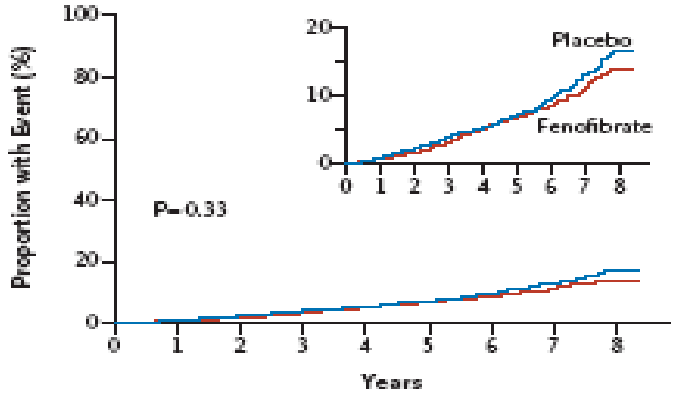
No. at Risk									
Fenofibrate	2765	2644	2565	2485	1981	1160	412	249	137
Placebo	2753	2634	2528	2442	1979	1161	395	245	131

B Expanded Macrovascular Outcome



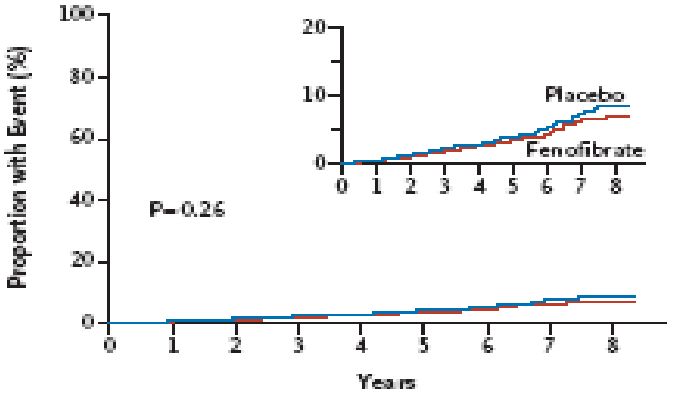
No. at Risk									
Fenofibrate	2765	2538	2390	2262	1751	999	354	211	112
Placebo	2753	2531	2357	2207	1732	992	316	201	104

C Death from Any Cause



No. at Risk									
Fenofibrate	2765	2737	2704	2646	2147	1271	469	285	157
Placebo	2753	2723	2680	2615	2164	1293	450	274	157

D Death from Cardiovascular Causes



No. at Risk									
Fenofibrate	2765	2700	2660	2606	2114	1255	457	285	155
Placebo	2753	2689	2633	2574	2128	1270	437	271	153

Action to control Cardiovasculr Risk in Diabetes (ACCORD) study group. N Eng J Med 2007; 356 : 1304-16

Niacin in Patients with Low HDL Cholesterol Levels Receiving Intensive Statin Therapy

The AIM-HIGH Investigators*

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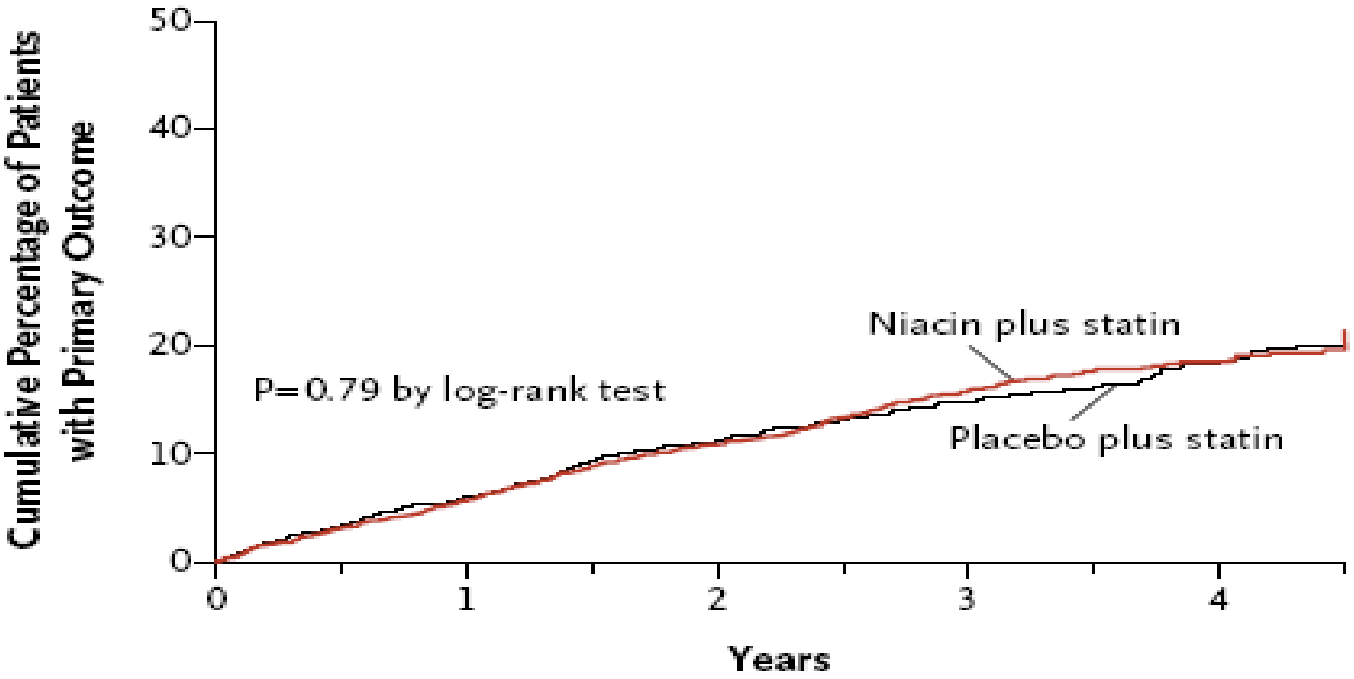

No. at Risk

Placebo plus statin	1696	1581	1381	910	436
Niacin plus statin	1718	1606	1366	903	428

Figure 1. Kaplan–Meier Curve for the Primary End Point.

Atherothrombosis Intervention in Metabolic Syndrome with Low HDL/High Triglycerides: Impact on Global Health Outcomes (AIM-HIGH) trial. N Eng J Med 2011; 365 : 2255-67

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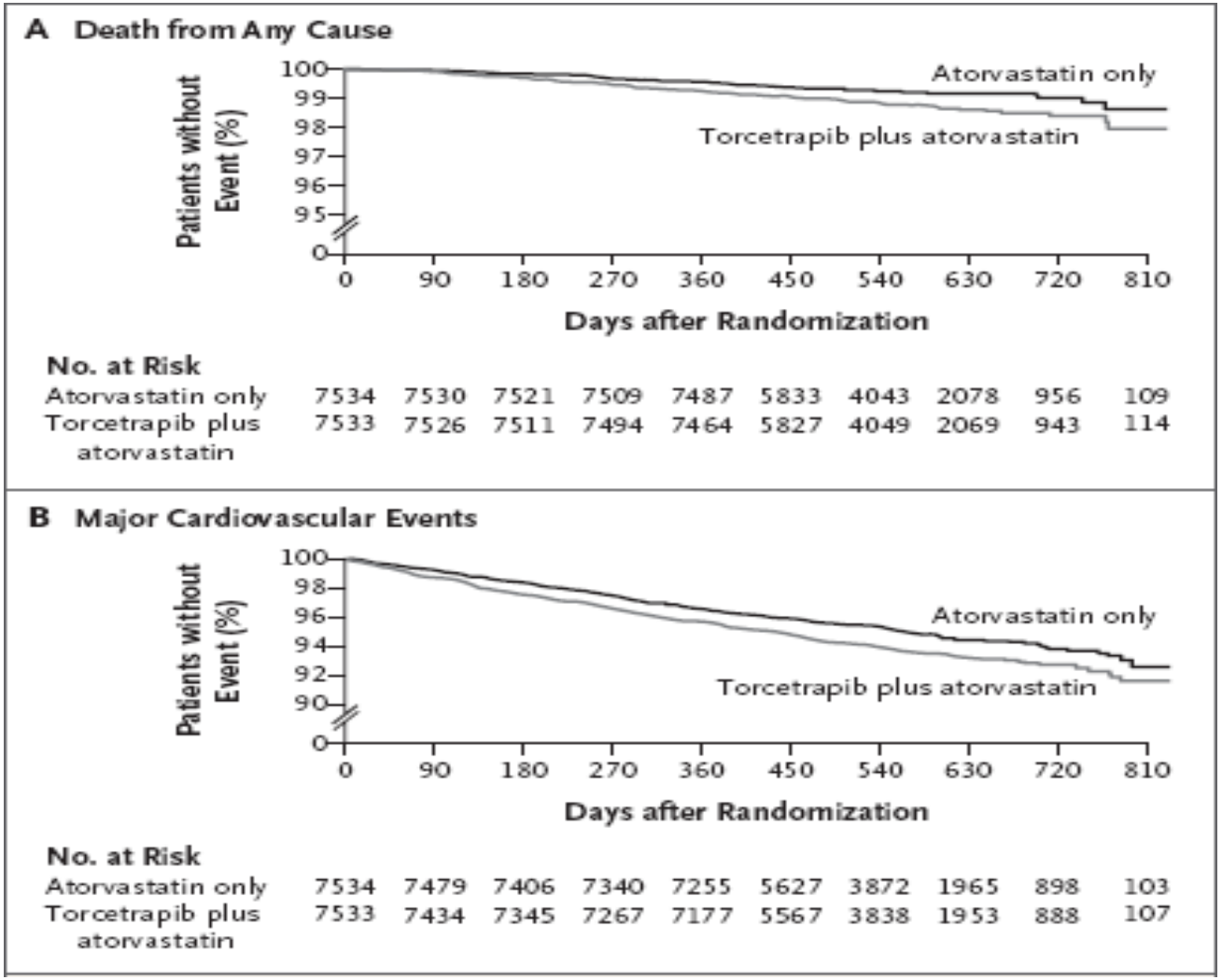
Effects of Torcetrapib in Patients at High Risk for Coronary Events

ILLUMINATE TRIAL



Barter P J et al. N Eng J Med 2007; 357 : 2109-22

dal-OUTCOMES trial: efficacy and safety of dalcetrapib in patients with recent acute coronary syndrome.

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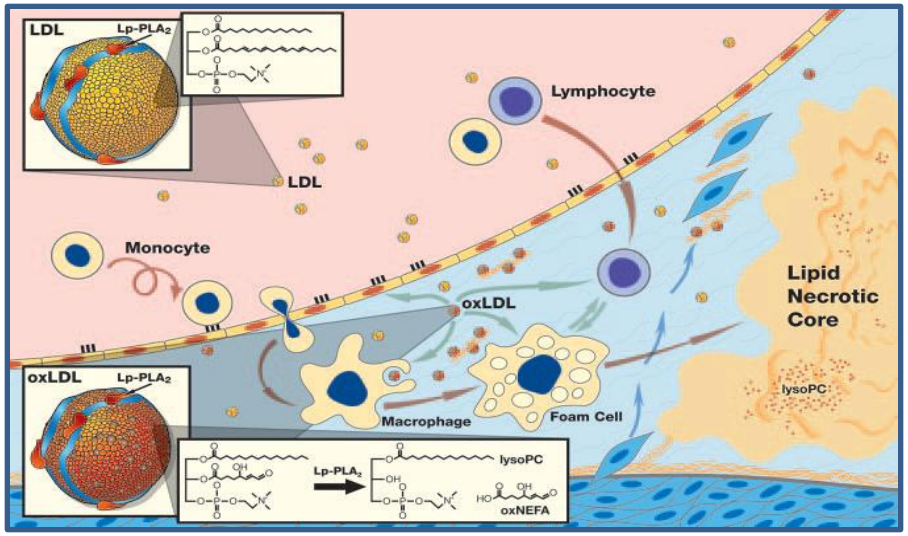
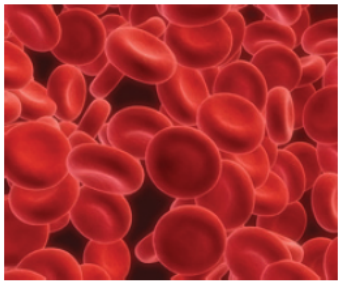
Roche Terminates Development Of CETP Inhibitor Dalcetrapib

Roche stops dalcetrapib trial for lack of benefit

Lp-PLA 2 – Възможности за повлияване



VISTA-16 Trial Stopped Early



In March 2012, it was announced that the VISTA-16 trial was being stopped early for futility. This announcement does not have any impact on the darapladib program. The VISTA-16 trial was studying a drug called varespladib that inhibits an enzyme called $sPLA_2$. The SOLID-TIMI 52 trial is studying darapladib that inhibits the $Lp\text{-}PLA_2$ enzyme. The two enzymes are distinct and are only weakly correlated. There are also many differences between the studies in design.

STABILITY

SOLID
TIMI 52

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STATE-OF-THE-ART PAPERS

microRNAs in Cardiovascular Diseases

Current Knowledge and the Road Ahead

Gianluigi Condorelli, MD, PhD,*†‡ Michael V. G. Latronico,* Elena Cavarretta, MD, PhD§

Rozzano, Rome, and Latina, Italy

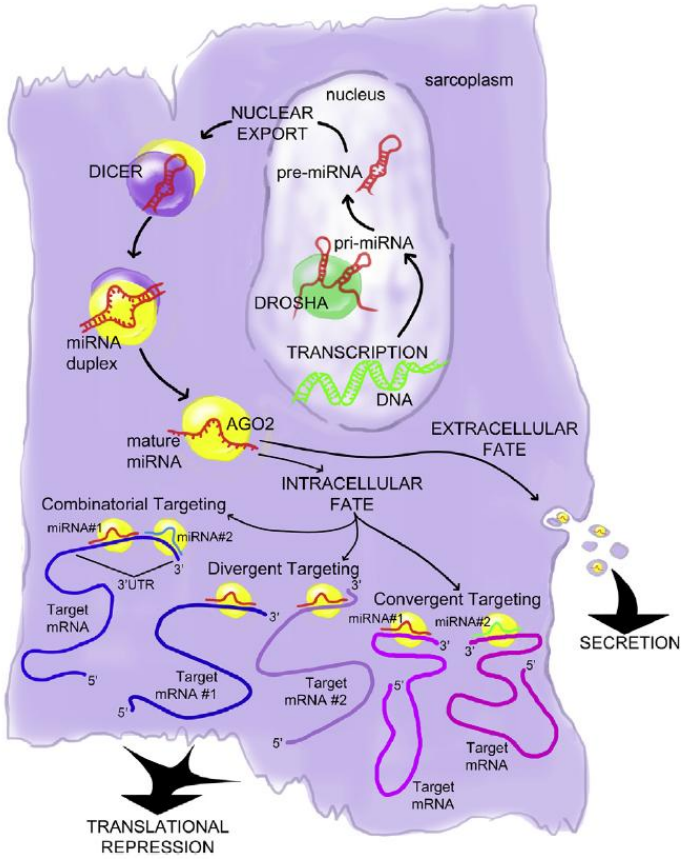

Figure 1 Schematic of miRNA Biogenesis and Function

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Curing Atherosclerosis Should Be the Next Major Cardiovascular Prevention Goal

Jennifer G. Robinson, MD, MPH,* Samuel S. Gidding, MD†‡

Iowa City, Iowa; Wilmington, Delaware; and Philadelphia, Pennsylvania