Lipids 201 : Beyond atorvastatin 40

This webinar will start shortly.

phn TASMANIA
An Australian Government Initiative

primary health TASMANIA

Cardiology at the interface of primary and secondary care - session four

Lipids 201 : Beyond atorvastatin 40

Zoom webinar – Wednesday 3 September 2025

6.30 - 8pm

Acknowledgement of traditional owners

We acknowledge the Tasmanian Aboriginal people as the traditional owners and ongoing custodians of the land on which we are meeting today. We pay our respects to Elders past and present.

We would also like to acknowledge Aboriginal people who are joining us today.

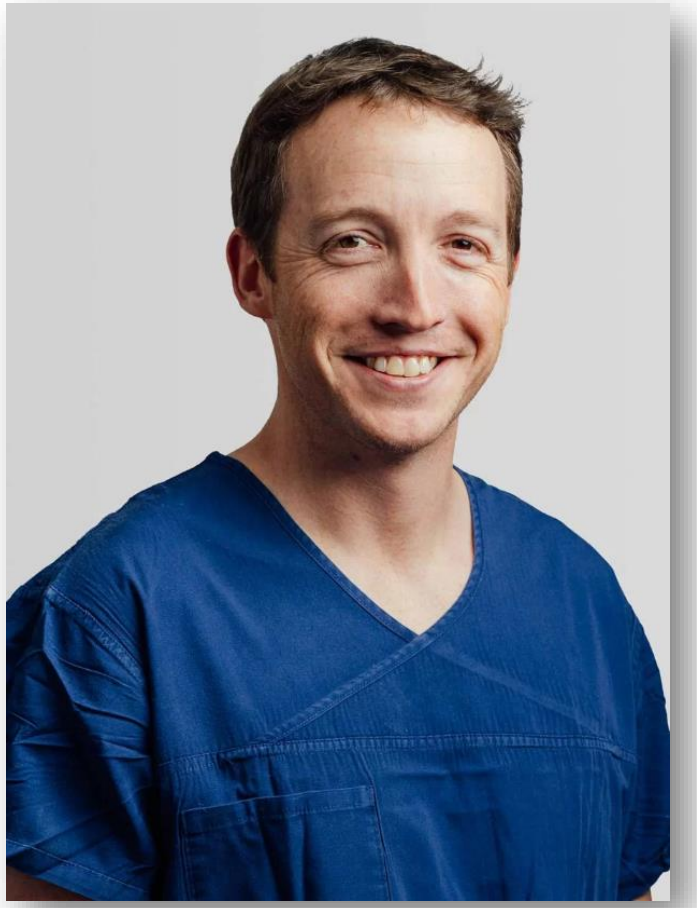
Learning outcomes

- Explain the role of dyslipidemia in the development of atherosclerotic cardiovascular disease.
- Evaluate the role of absolute risk assessment in the management of cardiovascular risk.
- Identify contemporary LDL targets in high risk populations
- Describe the treatment options for high-risk dyslipidemia.

Some housekeeping

- Tonight's webinar is being recorded
- Please use the Zoom Q&A feature to ask questions
- At the end of the webinar your browser will automatically open an evaluation survey. We appreciate you taking the time to complete this to help us improve our events programme
- Please don't forget to register for your next webinar at: https://www.primaryhealthtas.com.au/for-health-professionals/events/

Presenter



Dr Andrew Black

Panel

Dr Paul MacIntyre

Dr Graeme Bleach

Lipids 201: Beyond atorvastatin 40

ESC European Society of Cardiology

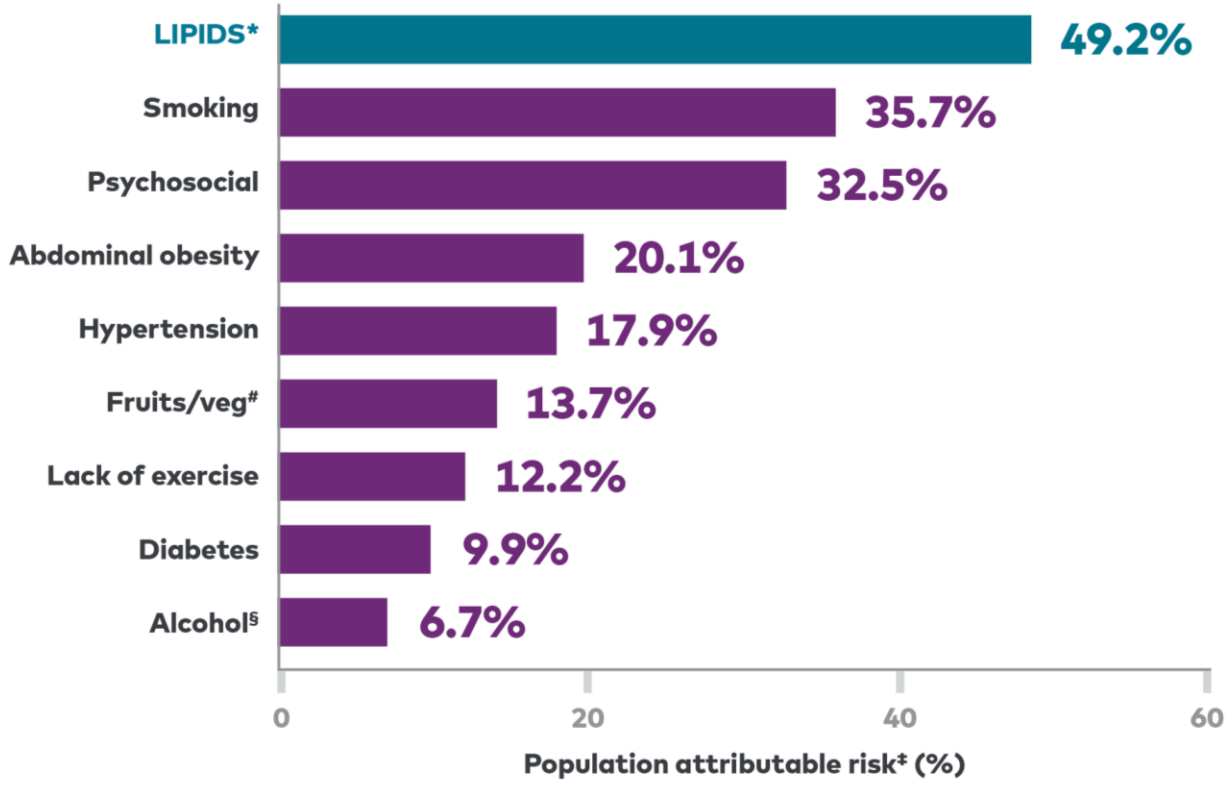
European Heart Journal (2020) **41**, 111–188
doi:10.1093/eurheartj/ehz455

ESC/EAS GUIDELINES

2019 ESC/EAS Guidelines for the management of dyslipidaemias: *lipid modification to reduce cardiovascular risk*

The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS)

Why does it matter?

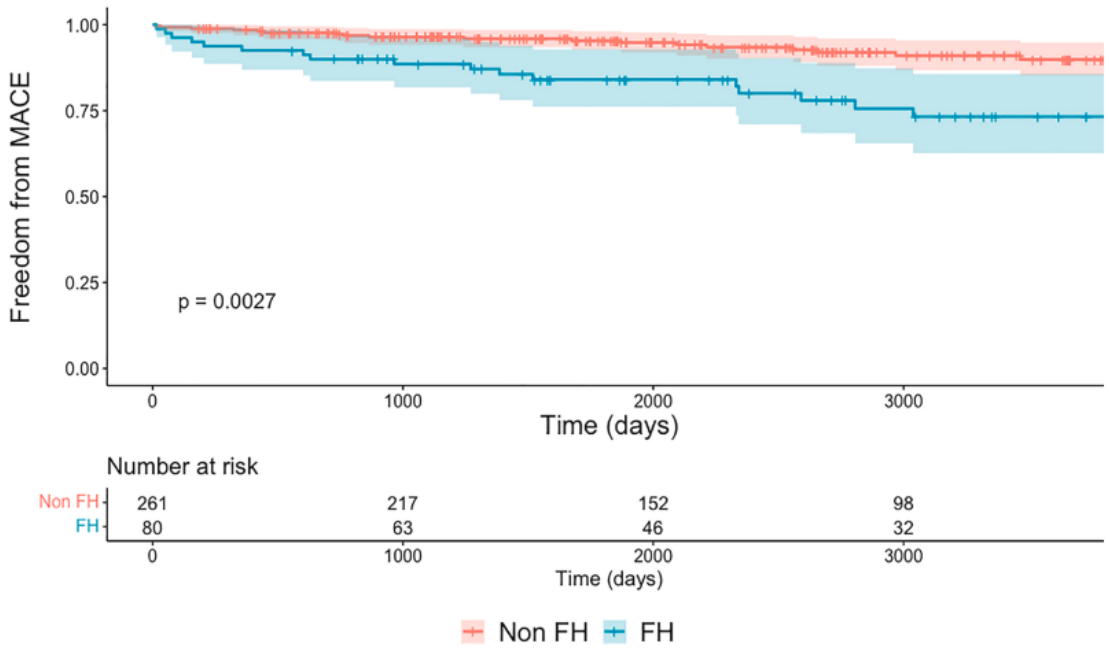


1. Yusuf S et al. Lancet 2004; 364: 937–952.
2. Rockhill B et al. Am J Public Health 1998; 88: 15–19.

Biochemical and clinical outcomes by FH status.

XXX	Non-FH (n=261)	Possible FH (n=249)	FH (n=80)	P-value (FH vs Non-FH)
LDL-C <1.8 mmol/L achieved	119 (45.6)	85 (34.1)	15 (18.8)	<0.001
LDL-C <1.4 mmol/L achieved	44 (16.9)	27 (10.8)	7 (8.8)	0.11
MACE	23 (8.8)	37 (14.9)	17 (21.3)	0.0047
Acute coronary syndrome	11 (4.2)	19 (7.6)	8 (10.0)	0.09
Coronary revascularisation	5 (1.9)	11 (4.4)	5 (6.3)	0.1
Stroke	5 (1.9)	7 (2.8)	1 (1.3)	1
Cardiac death	4 (1.5)	6 (2.4)	5 (6.3)	0.06

Abbreviations: FH, familial hypercholesterolemia; LDL-C, low-density lipoprotein cholesterol; MACE, major adverse cardiovascular event.



- Deconinck O, et al. Heart, Lung, Circ 2025

Absolute cardiovascular risk

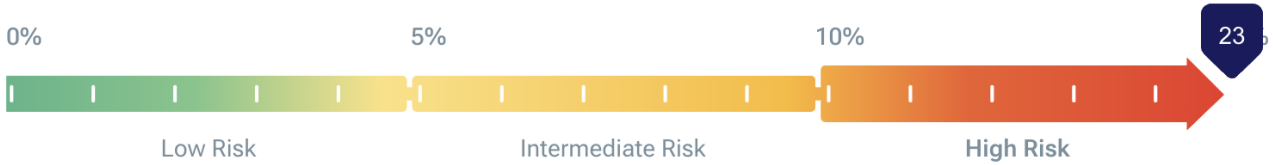
- Risk factors synergistic
- Higher risk → Greater benefit
- Physicians and patients under-estimate risk
- AusCVDRisk: cvdcheck.org.au

Australian guideline and calculator for assessing and managing cardiovascular disease risk

- Target population
  - All people 45 – 79 years
  - People with diabetes aged 35 – 79
  - First nations people aged 30 – 79
  - Without known CVD
  - Exclusions
    - Moderate to severe CKD: eGFR < 45
    - Familial hypercholesterolemia
    - (Calcium score > 100; extreme Lp(a) elevation)

23%

High risk



Consider reclassifying down a category if ?

- Coronary artery calcium score of 0 ? ☐
- East Asian ethnicity (Chinese, Japanese, Korean, Taiwanese, or Mongolian ethnicities) ? ☐

Consider reclassifying up a category if ?

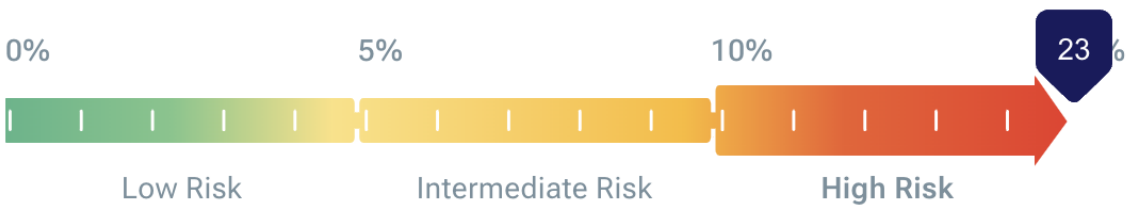
- Coronary artery calcium score > 99 units, or ≥ 75th percentile for age and sex ? ☐
- First Nations people ? ☐
- Māori, Pacific Islander or South Asian ethnicity (Indian, Pakistani, Bangladeshi, Sri Lankan, Nepali, Bhutanese or Maldivian ethnicities) ? ☐
- Family history of premature CVD ? ☐
- Chronic kidney disease ? ☐
- People living with severe mental illness ? ☐

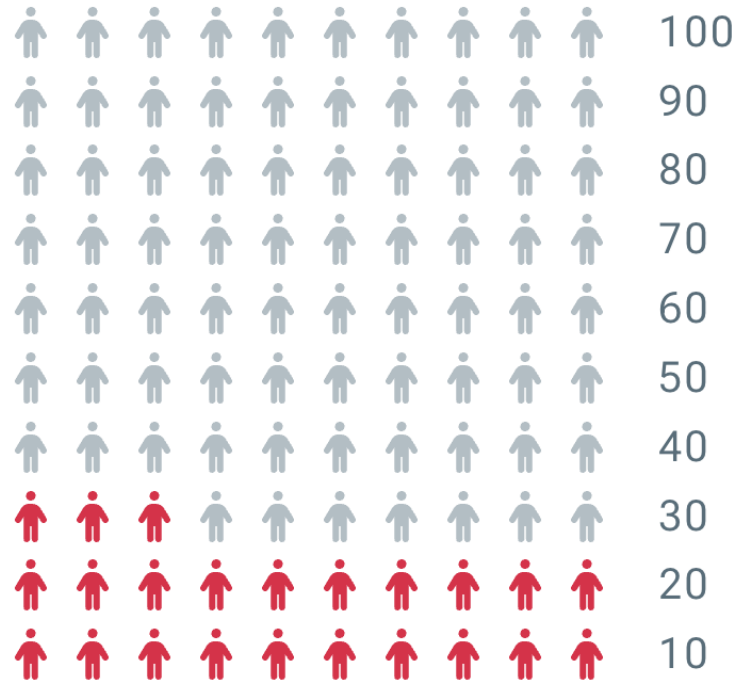
Select to proceed to the results page

Reclassify down to low risk | Reclassify down to intermediate risk | Continue without reclassifying

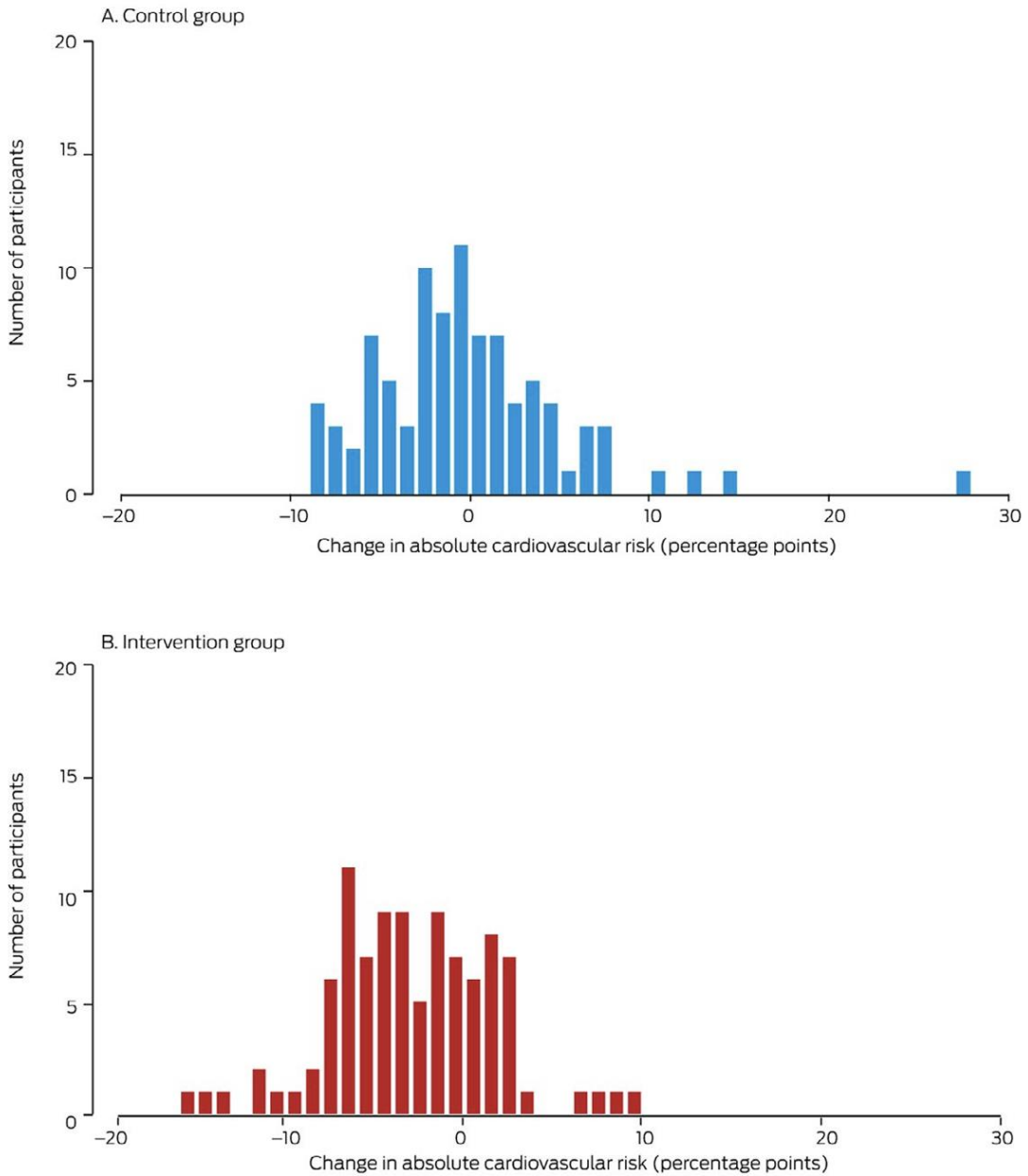
23% High risk

Your current risk of having a heart attack or stroke in the next 5 years is 23 out of 100, which is considered high. Imagine 100 people like you. 23 of those people will have a heart attack or stroke in the next 5 years if they don't take action.





3 Changes in individual absolute cardiovascular risk scores between baseline and follow-up assessments, by study arm



Black JA, et al. *Med J* Aust 202021:214(6)266-271

Young people

- Low absolute risk may conceal high relative risk
- Lifetime risk
- Risk age (www.HeartScore.org)
- Careful with calcium scores
  - ⚠️ Symptomatic

Women

- Risk is *deferred*, not lower

Other risk modifiers

- AF
- Physical inactivity
- Obesity
- Major psychiatric disorders
- Chronic inflammatory conditions

Overall optimal risk control

ESC risk groups

Very high risk

- ASCVD
  - Event, intervention, >50% plaque (multiple)
- DM2 with end-organ damage
- DM1 > 20 years duration
- Severe CKD (eGFR < 30)
- SCORE > 10% 10-year risk

High risk

- TC > 8, LDL > 4.9, BP > 180/110
- FH
- DM > 10 years
- Moderate CKD (eGFR 30 – 59)
- SCORE 5 - 10%

Moderate risk

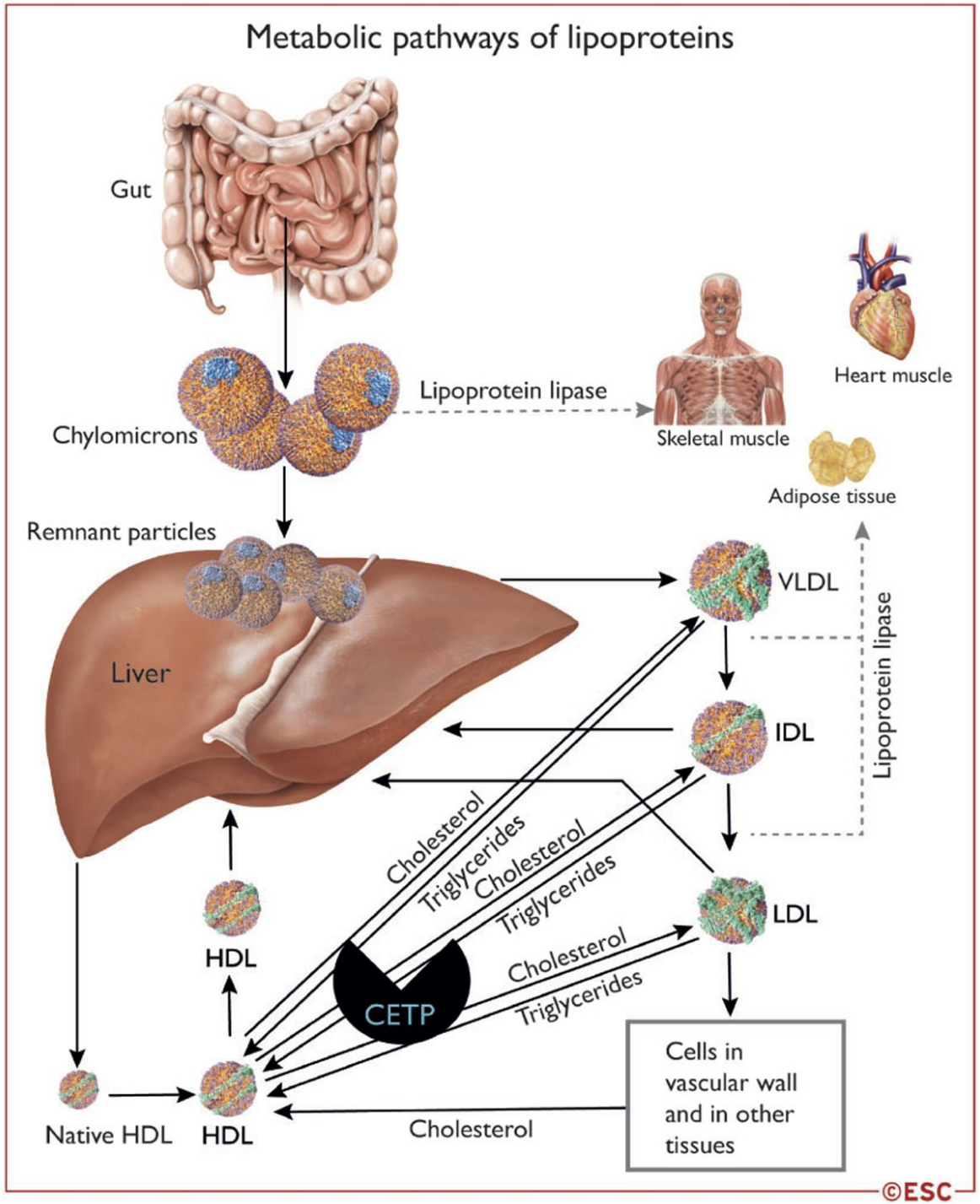
- DM < 10 years
- SCORE 1 – 5%

Low risk

- SCORE < 1%

	Total CV risk (SCORE) %	Untreated LDL-C levels					
		<1.4 mmol/L (55 mg/dL)	1.4 to <1.8 mmol/L (55 to <70 mg/dL)	1.8 to <2.6 mmol/L (70 to <100 mg/dL)	2.6 to <3.0 mmol/L (100 to <116 mg/dL)	3.0 to <4.9 mmol/L (116 to <190 mg/dL)	≥4.9 mmol/L (≥190 mg/dL)
Primary prevention	<1, low-risk	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention
	Class[a]/Level[b]	I/C	I/C	I/C	I/C	IIa/A	IIa/A
	≥1 to <5, or moderate risk (see *Table 4*)	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention
	Class[a]/Level[b]	I/C	I/C	IIa/A	IIa/A	IIa/A	IIa/A
	≥5 to <10, or high-risk (see *Table 4*)	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
	Class[a]/Level[b]	IIa/A	IIa/A	IIa/A	I/A	I/A	I/A
	≥10, or at very-high risk due to a risk condition (see *Table 4*)	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
	Class[a]/Level[b]	IIa/B	IIa/A	I/A	I/A	I/A	I/A
Secondary prevention	Very-high-risk	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
	Class[a]/Level[b]	IIa/A	I/A	I/A	I/A	I/A	I/A

Lipoproteins

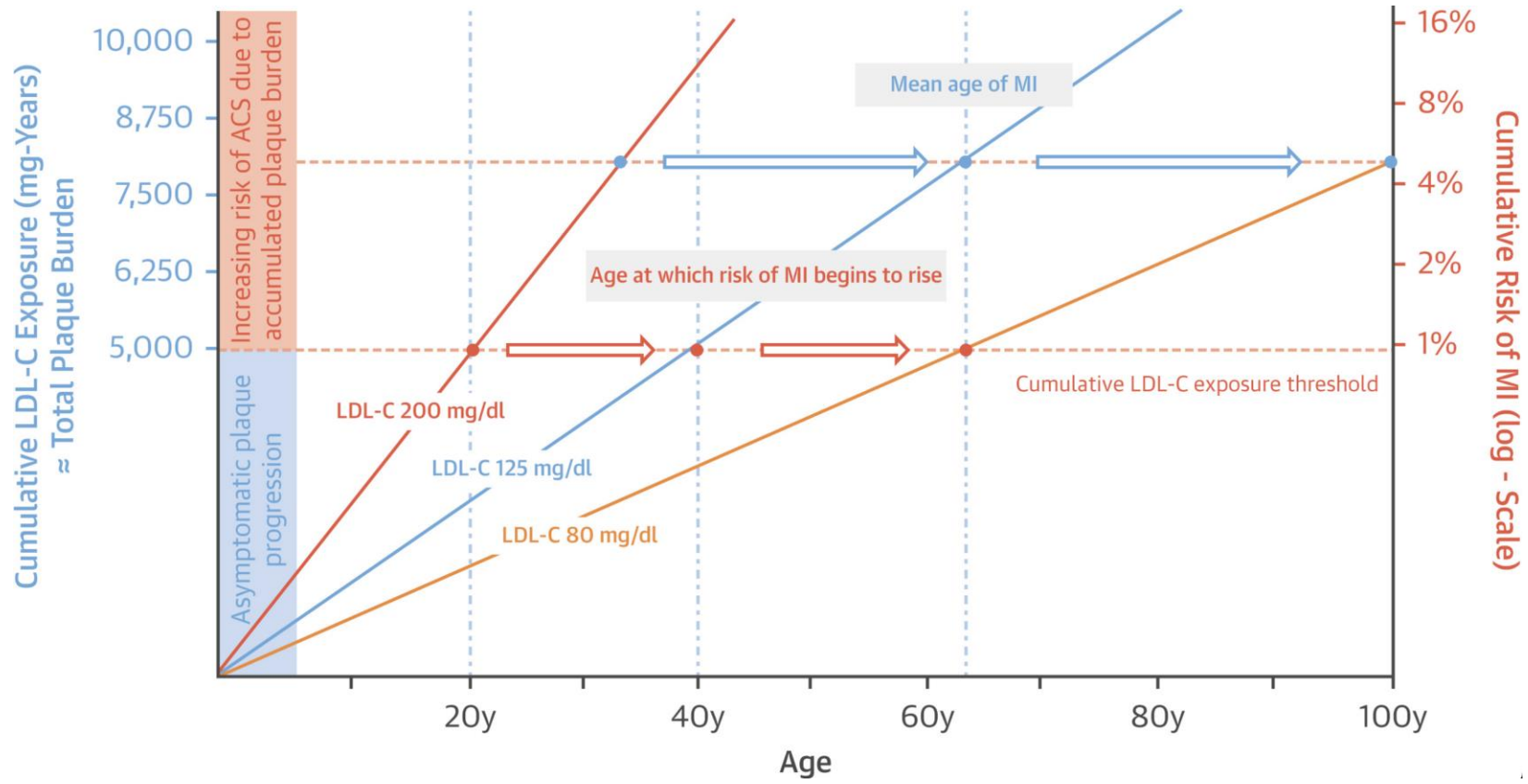


Mach F, et al. *Eur Heart* J 2019;00:1-18

	Diameter (nm)	Major apolipoprotein
Chylomicrons	80 – 100	ApoB-48
VLDL	30 – 80	ApoB-100
IDL	25 – 30	ApoB-100
LDL	20 – 25	ApoB-100
HDL	8 – 13	ApoA-1
Lp(a)	25 – 30	Apo(a)

- Pro-thrombotic, pro-inflammatory, pro-atherogenic
- Strongly and causally associated with ASCVD

LDL particles have a causal and cumulative effect on ASCVD risk



Ference B et al. J Am Coll Cardiol 2018;72(10):1141-1156

Lipid measurements

- Standard panel: TC, HDL, TG
  - LDL calculated: LDL = TC - HDL - (TG/2.2)
- Direct LDL measurement
- ApoB measurement
- Non-fasting generally adequate
- Lp(a) measured once
  - Family history

Treatment targets

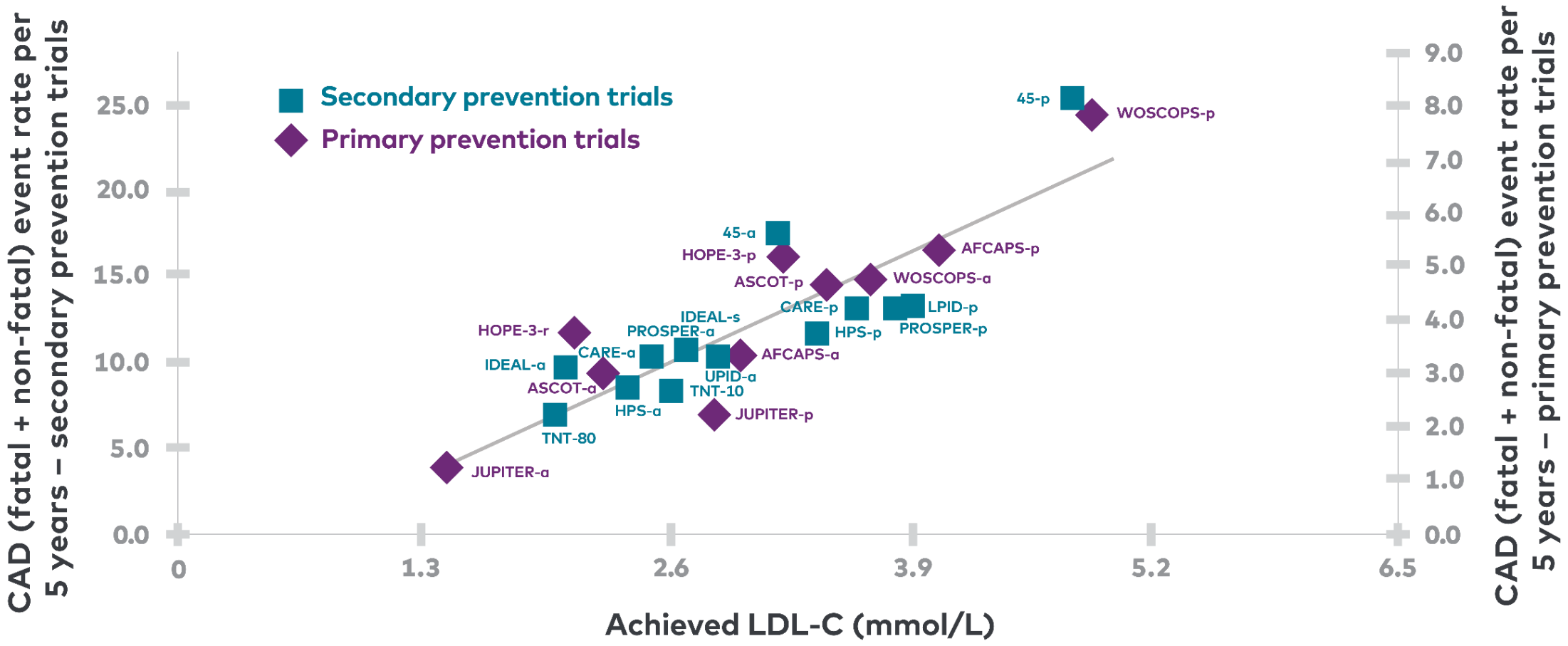
- Risk reduction directly related to reduction in LDL
- No lower threshold
- Benefits not restricted to statin therapy

- ESC guidelines
  - Reduce LDL to as low as possible
  - Minimum 50% reduction, plus
  - Target goal
    - Secondary prevention
      - < 1.4mmol/L secondary prevention (class I)
      - < 1.0mmol/L if recurrent events (class IIb)
    - Primary prevention
      - < 1.8mmol/L if high risk (class I)
      - < 2.6mmol/L if moderate risk (class IIa)
      - < 3.0mmol/L if low risk (class IIb)

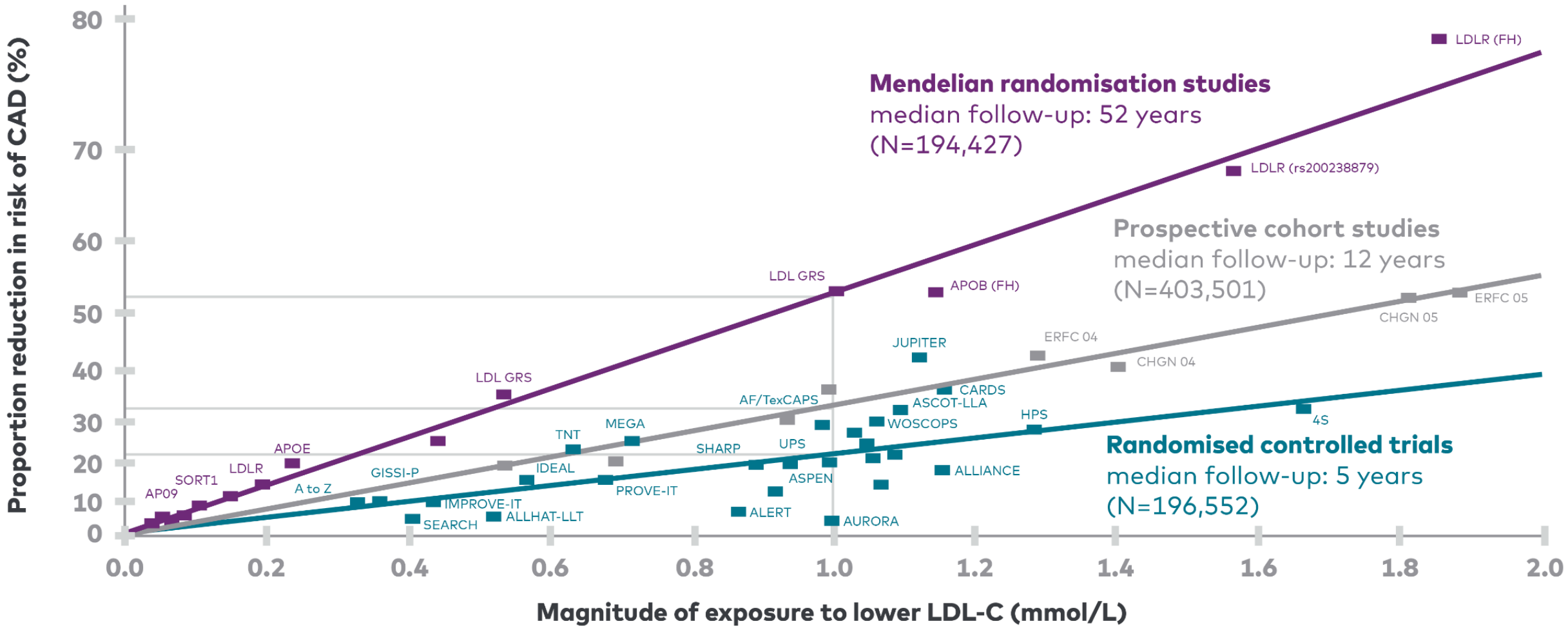
Management - lifestyle

- Dietary measures
  - Saturated fats increase LDL
    - Reduced intake ~ 10% reduction
  - Trans fats increase LDL and lower HDL
  - Plant sterols compete with cholesterol for intestinal absorption (olive & canola oil, veg, fruit, nuts, grains, legumes)
    - High in Mediterranean diet
      - Event rates ↓ (1 ° and 2° prevention)
      - LDL-C ↓ (~ 0.2mmol/L)
  - Portfolio diet
    - Low saturated fat, increased fiber, plant sterols, soy protein, nuts
    - LDL reduction ? 20 – 30% ? 15%; no CV outcome trials
- Weight loss: LDL ↓ 0.2mmol/L per 10kg loss
- Exercise

Management - pharmacotherapy

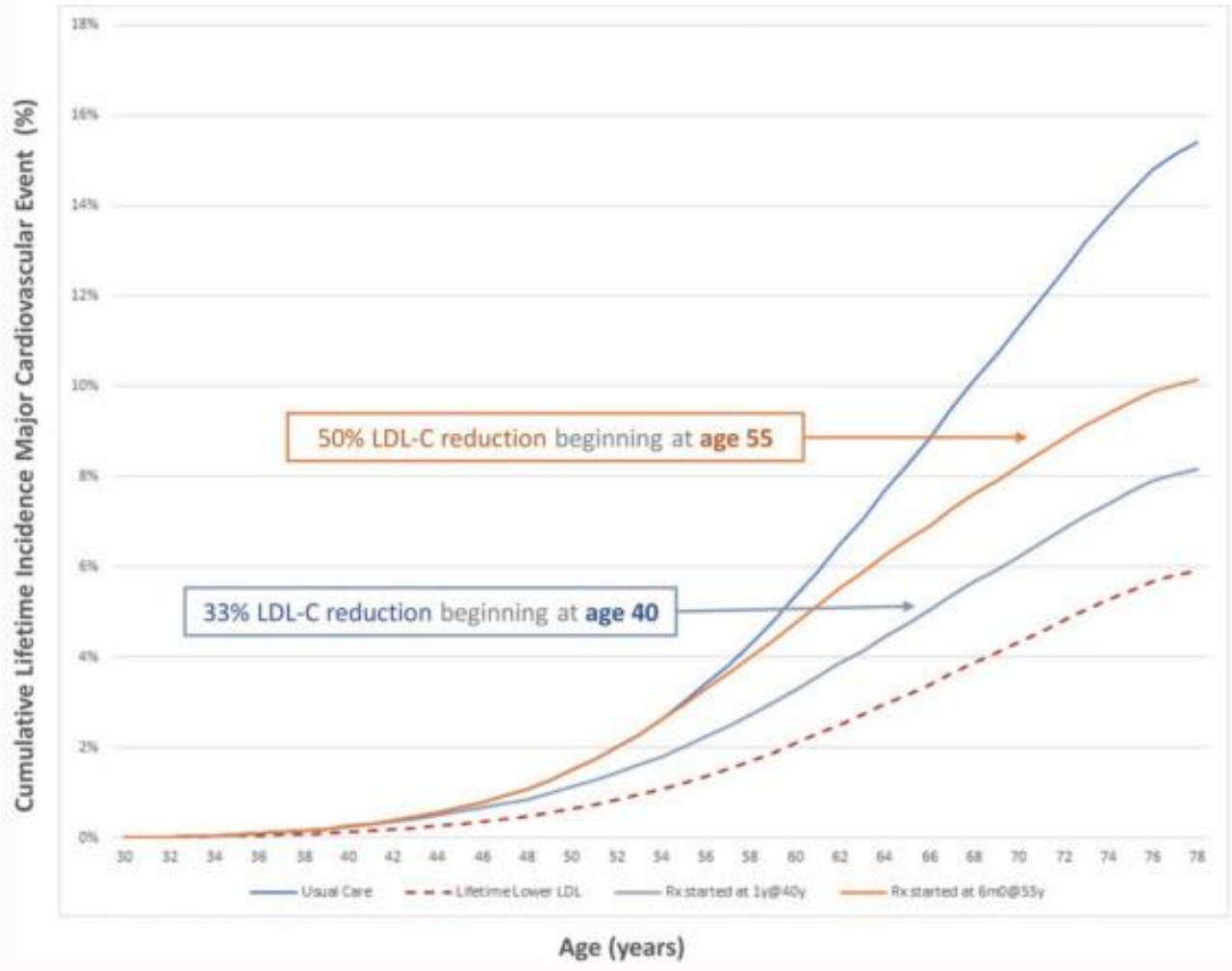


Ference BA et al. Eur Heart J 2017; 38: 2459–2472



Ference BA et al. Eur Heart J 2017; 38: 2459–2472

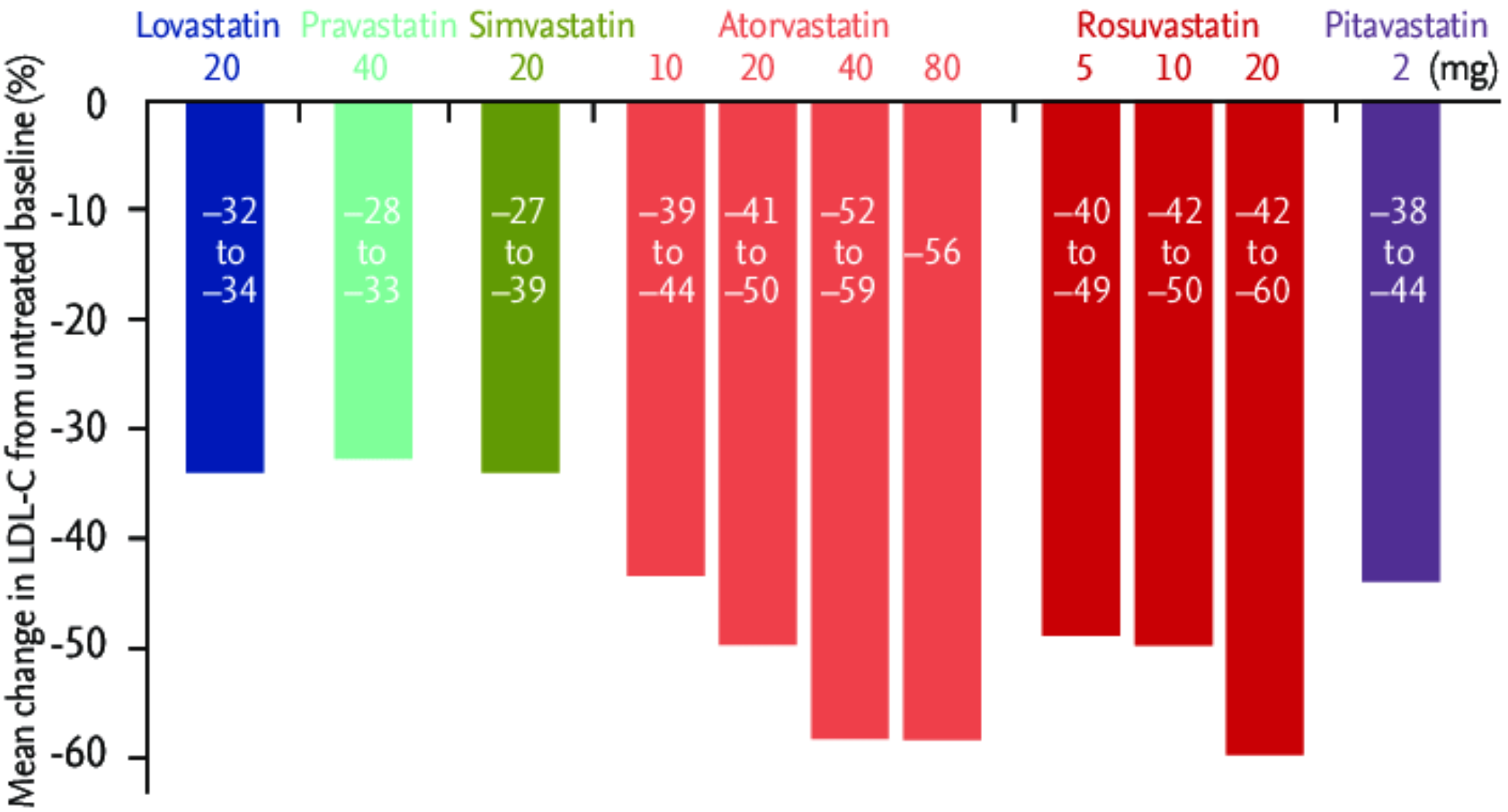
Treat high risk early



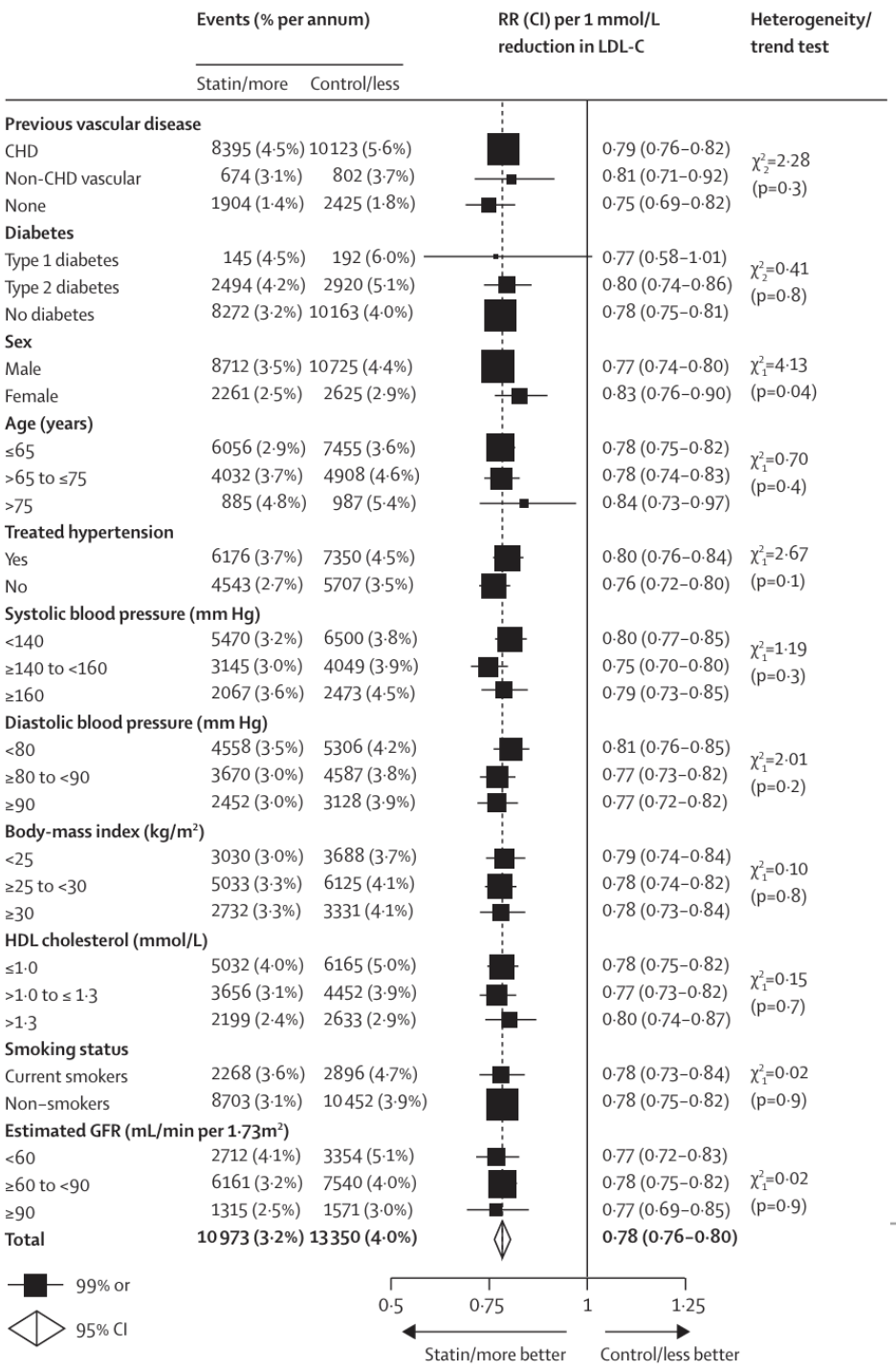
Ray KK et al, Cholesterol Road Map 2022

Statins

- Inhibit HMG-CoA-reductase
- Reduce LDL synthesis



Rhee E & HC Kim, *The Korean Journal of Internal Medicine* 2019;34(4):723-771

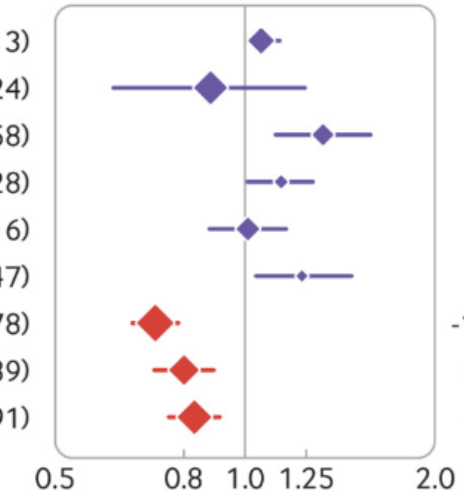


Cholesterol Treatment Trialists' (CTT) Collaboration Meta-analysis

- 26 RCTs, n > 170,000
- More vs less intensive statin *or* statin vs placebo
- 1mmol/L reduction in LDL
  - → 20% ↓ vascular events at 5 years
  - → 10% ↓ all-cause mortality
  - Consistent primary vs secondary prevention

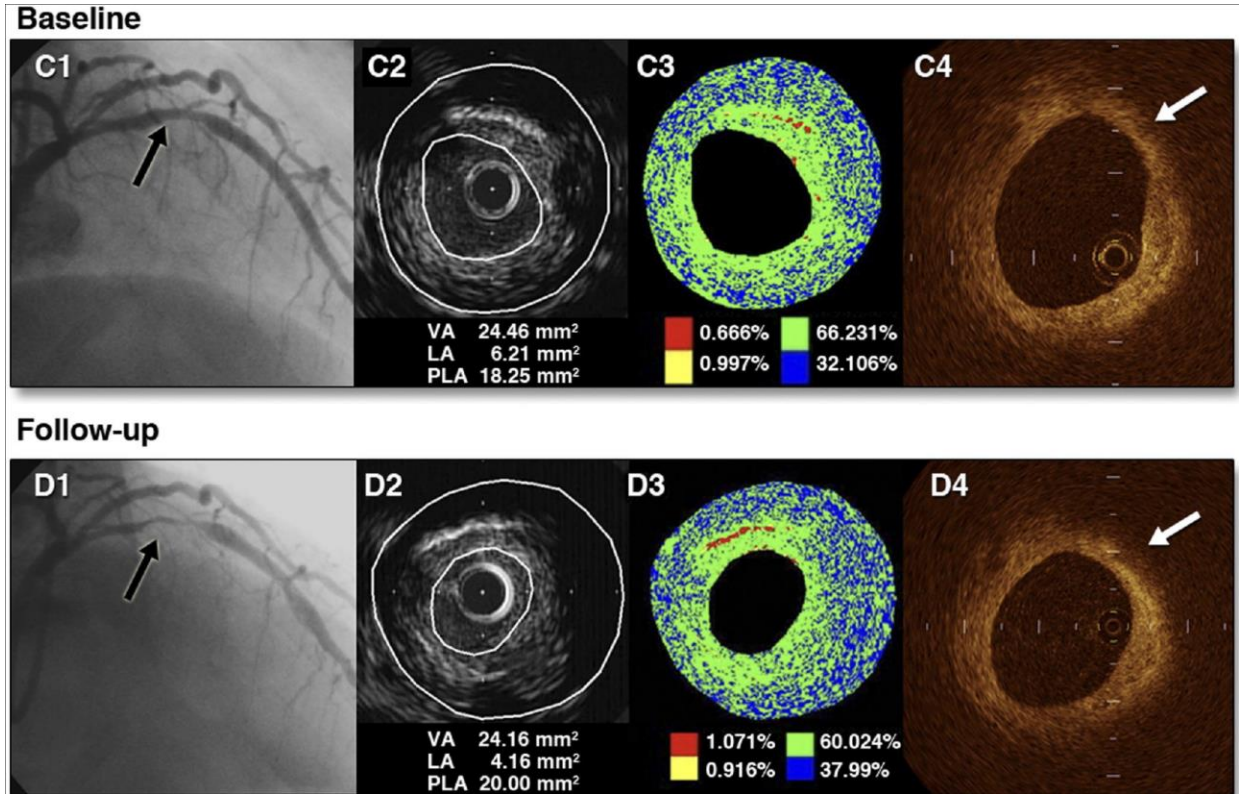
Lancet 2010;376:1670-81

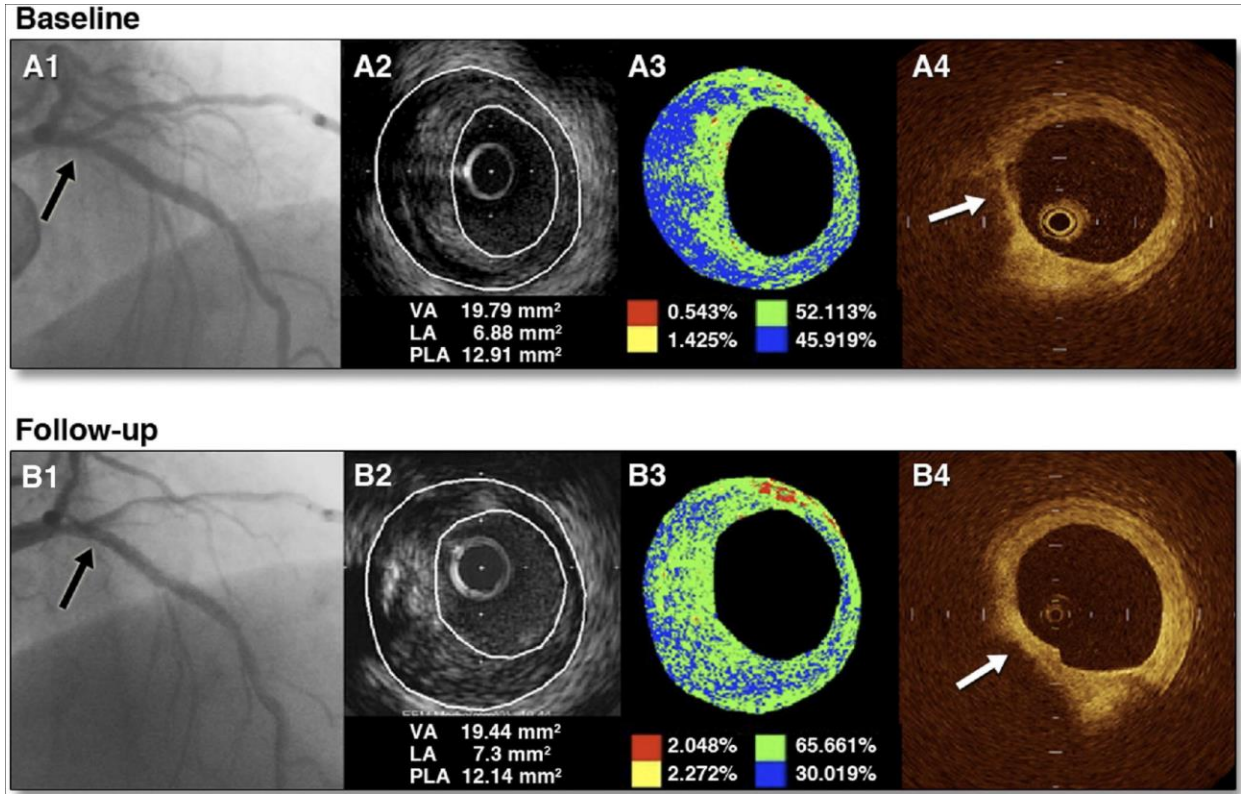
Outcome	No of studies	No of events/total: Statin group	No of events/total: Control group	Odds ratio (95% CI)	Odds ratio (95% CI)	Absolute risk difference (95% CI)	Test of effect (P value)	I^2 (%) (95% CI)	Q test (P value)
Muscle symptoms	21	3459/36 026	2785/29 278	1.06 (1.01 to 1.13)		15 (1 to 29)	0.030	1 (0 to 47)	0.45
Muscle disorders	25	70/46 746	55/38 994	0.88 (0.62 to 1.24)		0 (-1 to 1)	0.461	0 (0 to 0)	0.99
Liver dysfunction	21	406/31 305	217/23 498	1.33 (1.12 to 1.58)		8 (3 to 14)	0.001	0 (0 to 23)	0.84
Renal insufficiency	8	597/16 858	520/15 143	1.14 (1.01 to 1.28)		12 (1 to 24)	0.037	0 (0 to 23)	0.89
Diabetes	9	1190/29 318	1161/29 311	1.01 (0.88 to 1.16)		1 (-10 to 13)	0.882	50 (0 to 77)	0.04
Eye conditions	6	321/15 282	234/10 046	1.23 (1.04 to 1.47)		14 (2 to 29)	0.019	0 (0 to 36)	0.85
Myocardial infarction	22	996/50 093	1316/45 055	0.72 (0.66 to 0.78)		-19 (-23 to -15)	<0.001	33 (0 to 60)	0.07
Stroke	17	634/39 340	786/39 133	0.80 (0.72 to 0.89)		-9 (-12 to -5)	<0.001	20 (0 to 55)	0.22
Death from CVD	22	836/51 005	979/44 954	0.83 (0.76 to 0.91)		-8 (-12 to -4)	<0.001	27 (0 to 57)	0.12



- Systematic review and meta-analysis
- 62 primary prevention trials
- n > 120,000
- 4-year follow-up

Cai T, et al. *BMJ* 2021;374:n1537





Hattori K, et al. *JACC Cardiovasc Imaging* 2012;5(2):169-177

- Statin adverse effects
  - Statin-associated muscle symptoms (SAMS)
    - 10 - 15% in observational studies
  - Myositis
    - CK > ULN
  - Rhabdomyolysis
    - 1 – 3 cases / 100,000 person-years
    - Consider if CK > 10x ULN (often >40x ULN)

- CK > 10 ULN: Stop, check UEC, monitor
- CK 4 - 10 ULN no Sx: Repeat, 2 – 6 weeks
- CK 4 - 10 ULN + Sx: Stop and monitor CK +/- rechallenge
- CK < 4x ULN – ok / monitor

- Managing SAMS
  - ? Hypothyroidism
  - Drug interactions
  - Cautious dosing in elderly
  - ‘Statin holiday’
  - Alternative statin
  - Low dose
  - Alternate-daily dose

- Hepatic adverse effects
  - Mild ALT elevation in 0.5 – 2.0%
  - Generally, check at baseline and once at 8/52
  - > 3x ULN → cease or reduce dose & re-check 4/52

- Diabetes

Meta-Analysis > Lancet. 2010 Feb 27;375(9716):735-42.
doi: 10.1016/S0140-6736(09)61965-6. Epub 2010 Feb 16.

Statins and risk of incident diabetes: a collaborative meta-analysis of randomised statin trials

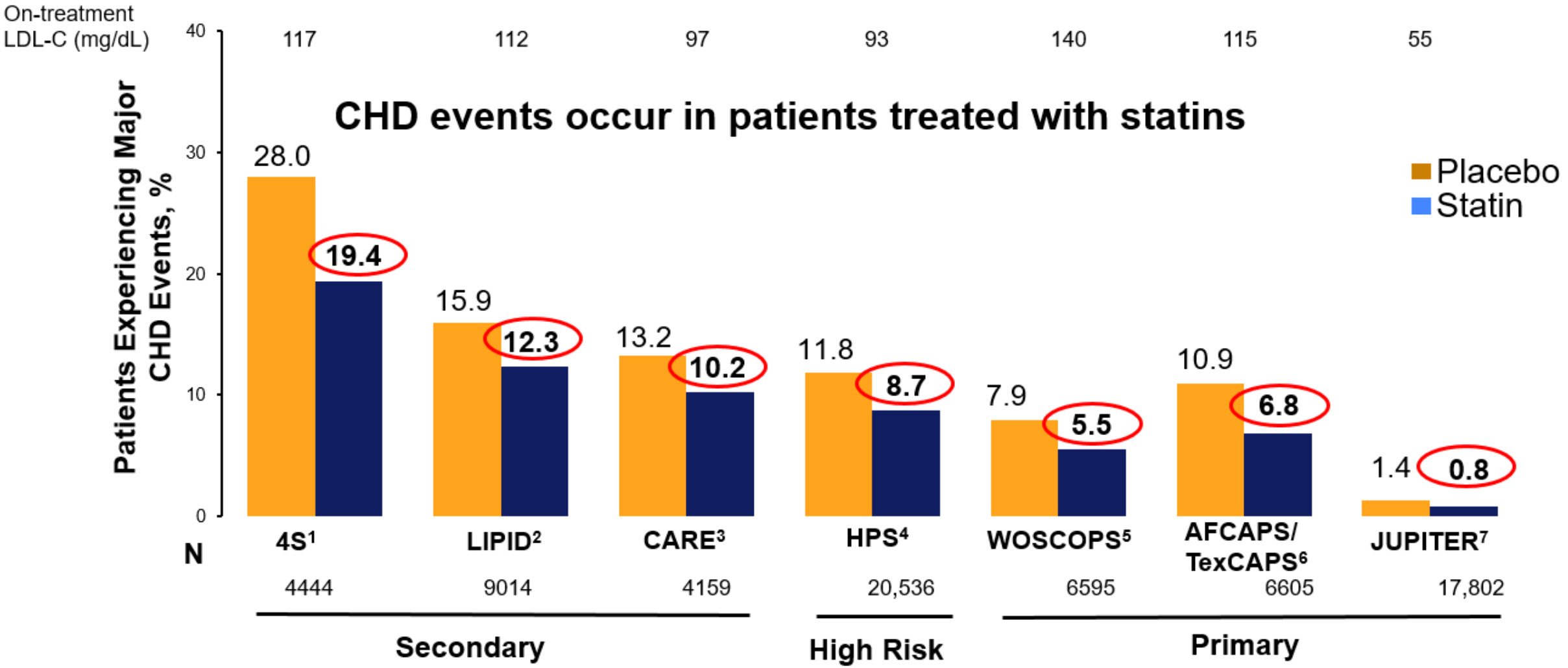
Naveed Sattar [1], David Preiss, Heather M Murray, Paul Welsh, Brendan M Buckley, Anton J M de Craen, Sreenivasa Rao Kondapally Seshasai, John J McMurray, Dilys J Freeman, J Wouter Jukema, Peter W Macfarlane, Chris J Packard, David J Stott, Rudi G Westendorp, James Shepherd, Barry R Davis, Sara L Pressel, Roberto Marchioli, Rosa Maria Marfisi, Aldo P Maggioni, Luigi Tavazzi, Gianni Tognoni, John Kjekshus, Terje R Pedersen, Thomas J Cook, Antonio M Gotto, Michael B Clearfield, John R Downs, Haruo Nakamura, Yasuo Ohashi, Kyoichi Mizuno, Kausik K Ray, Ian Ford

- 13 statin trials
- Secondary prevention
- n=91,000
- Diabetes OR 1.09 (95% CI 1.02 – 1.17)
- NNH 255 over 4 years

- Haemorrhagic stroke
  - ↑ in CTT meta-analysis
  - Not consistently replicated in other analyses

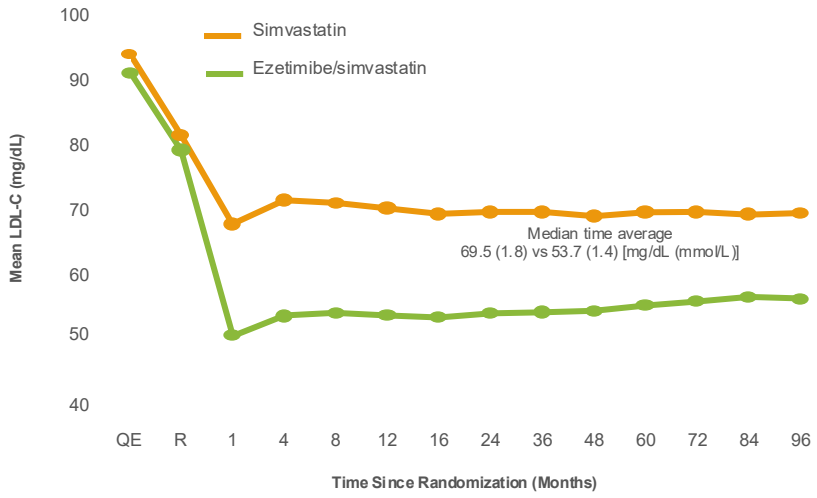
- Drug interactions (myopathy / rhabdo)
  - Most statins undergo metabolism via CYP3A4 (not prava / rosuvastatin)
  - Potential interactions:
    - Azole antifungals
    - Macrolide antibiotics
    - HIV protease inhibitors
    - Calcium channel blockers
    - Amiodarone
    - Grapefruit juice
    - Ciclosporin
    - Gemfibrozil (fenofibrate ok)

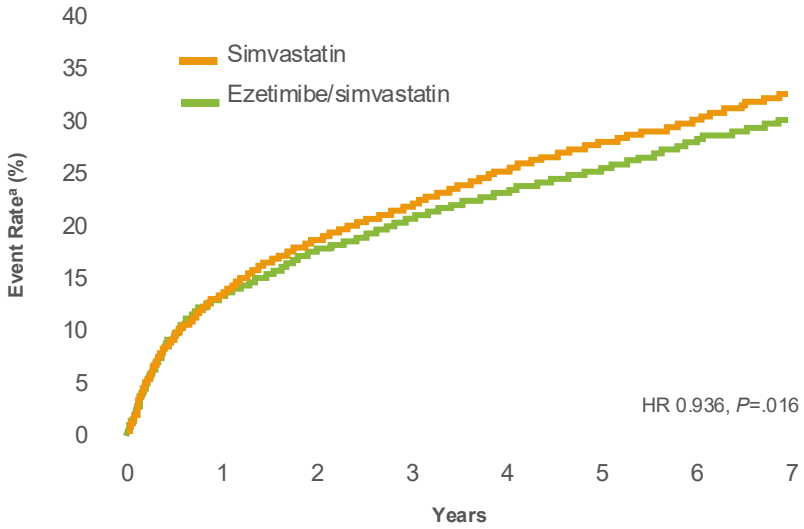
- Pregnancy and breastfeeding


On-treatment LDL-C (mg/dL)
117
112
97
93
140
115
55
CHD events occur in patients treated with statins
Patients Experiencing Major CHD Events, %
40
30
20
10
0
Placebo
Statin
28.0
19.4
15.9
12.3
13.2
10.2
11.8
8.7
7.9
5.5
10.9
6.8
1.4
0.8
N
4S[1]
LIPID[2]
CARE[3]
HPS[4]
WOSCOPS[5]
AFCAPS/ TexCAPS[6]
JUPITER[7]
4444
9014
4159
20,536
6595
6605
17,802
Secondary
High Risk
Primary

Ezetimibe

- Decreases intestinal uptake of dietary and biliary cholesterol
- LDL-C down ~ 20% +/- statin





- Cannon CP, et al. *N Engl J Med*. 2015;372(25):2387-97.

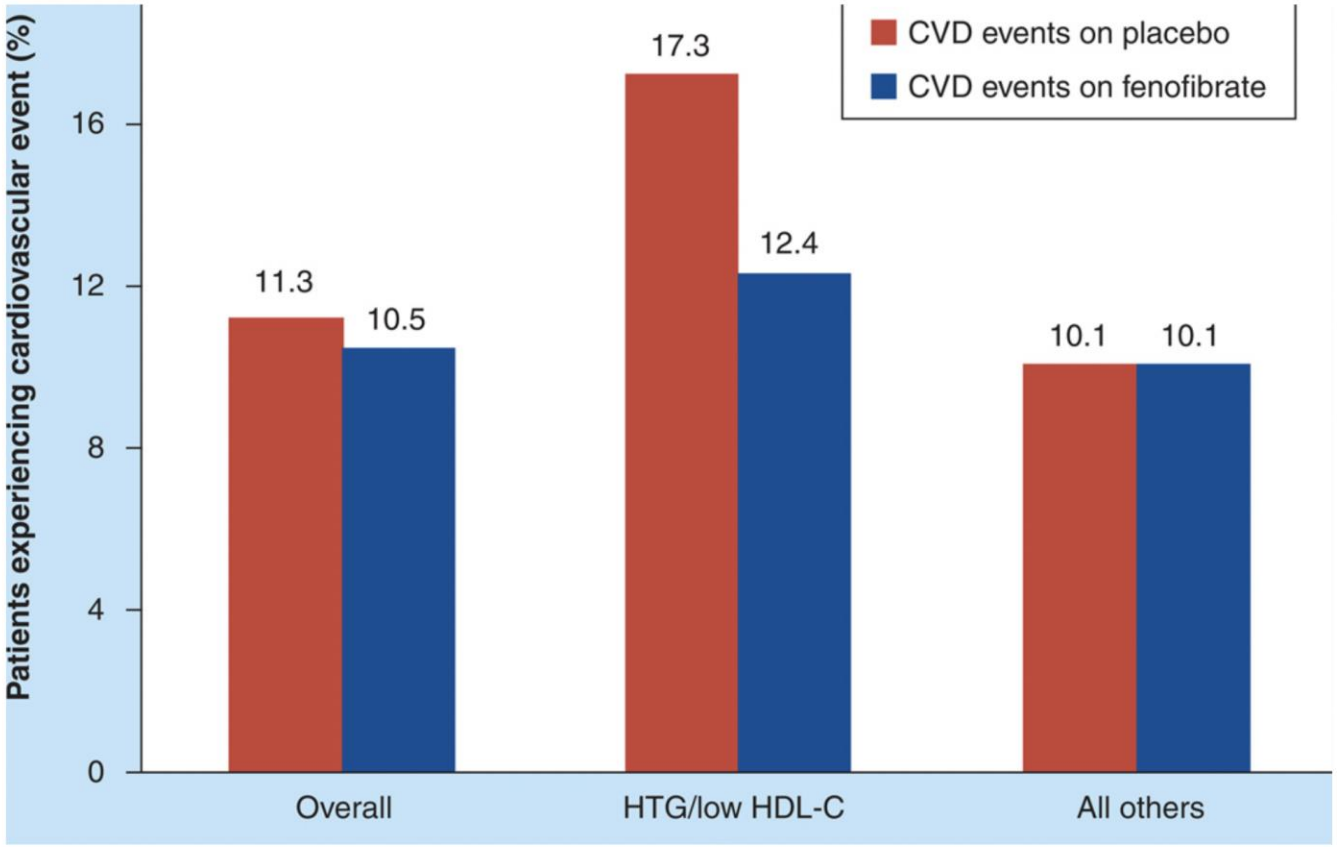
- PRECISE-IVUS trial
  - Atorvastatin vs atorvastatin + ezetimibe
  - Prospective, randomized, controlled trial
  - n=202 post PCI
  - Plaque volume (IVUS) at baseline and 9-12 months

	Absolute Change				
	LZ Group (n = 100)	p Value Compared With Baseline	L Group (n = 102)	p Value Compared With Baseline	p Value Between Groups
Plaque volume, mm^3	-3.9 (-10.6 to 0.0)	<0.001	-1.0 (-6.8 to 5.7)	0.4	0.001
Percent atheroma volume, %	-1.4 (-3.4 to -0.1)	<0.001	-0.3 (-1.9 to 0.9)	0.03	0.001
ACS cohort	-2.3 (-3.7 to -0.5)	<0.001	-0.2 (-1.3 to 0.5)	0.2	<0.001
SAP cohort	-1.2 (-2.2 to -0.1)	0.001	-0.7 (-2.3 to 1.1)	0.08	0.2

- Tsujita K, et al. *J Am Coll* Cardiol 2015;66:495-507

Fibrates

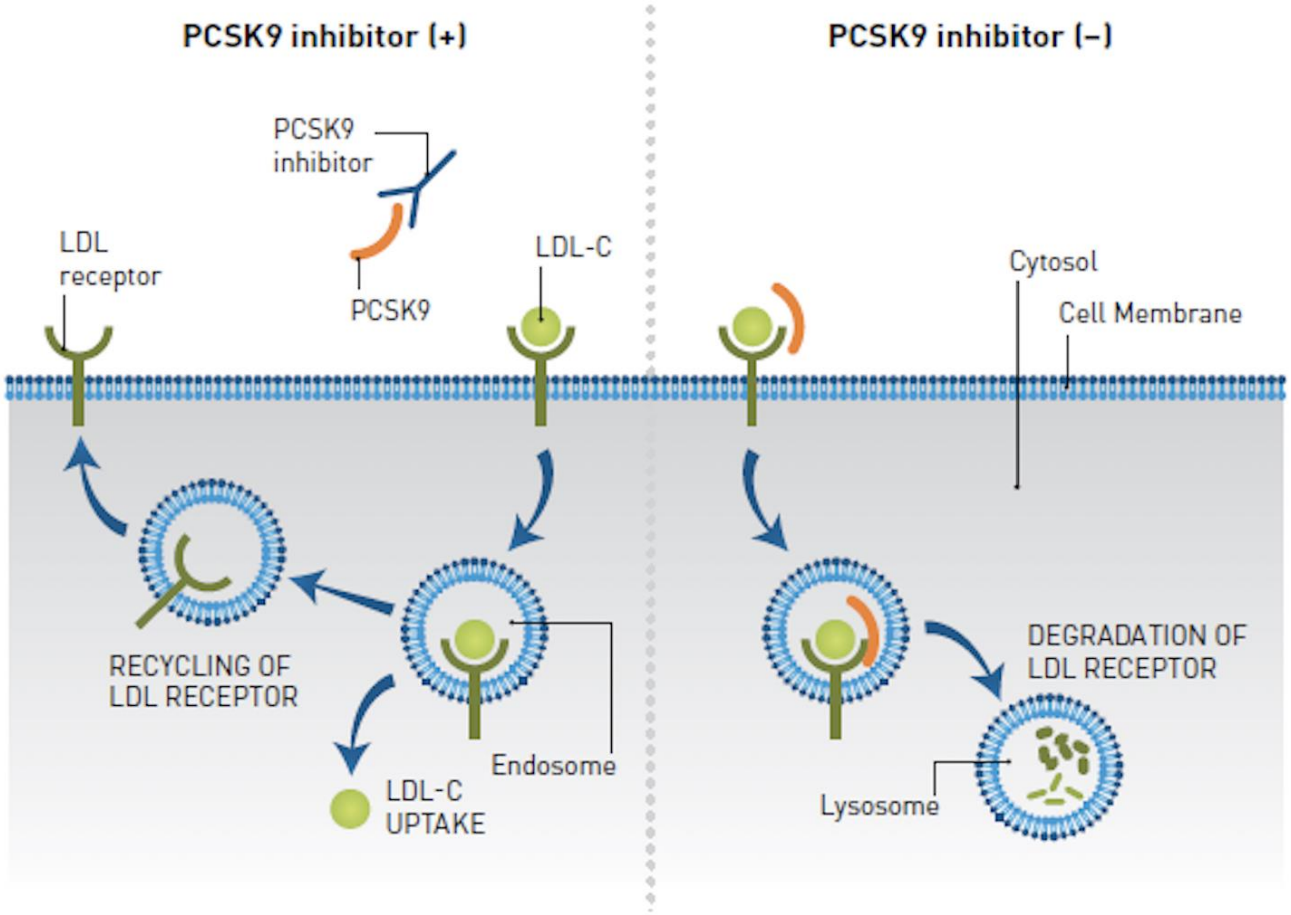
- Agonists of PPAR-α
- Affect lipid metabolism
  - 50% reduction in TG
  - < 20% reduction in LDL
  - < 20% increase in HDL
- CV events
  - Mostly -



Elam M, et al. *Clin Lipidol* 2011;6(1):9-20

PCSK9 inhibitors

- Monoclonal antibodies
  - Evolocumab
  - Alirocumab
- siRNA
  - Inclisiran



Ahn CH, Choi SH. *Diabetes Metab J* 2015;39(2):87-94

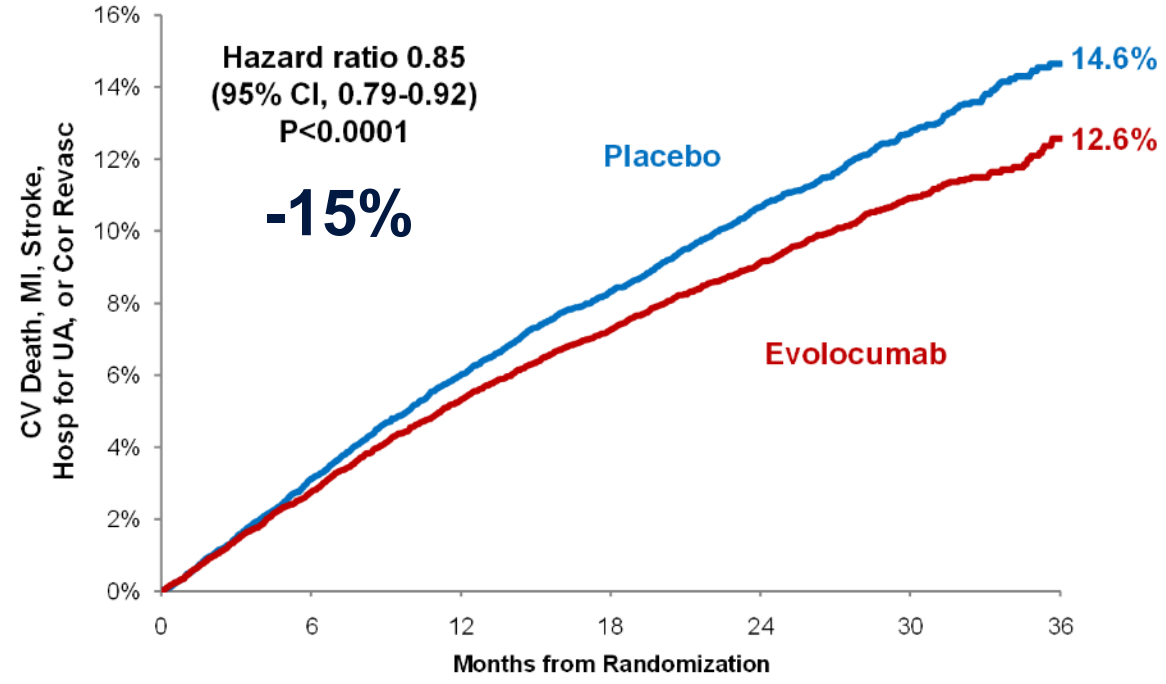
- LDL down 50 - 60%
- TG down 25%
- HDL up 10%
- Lp(a) down 30 – 40%

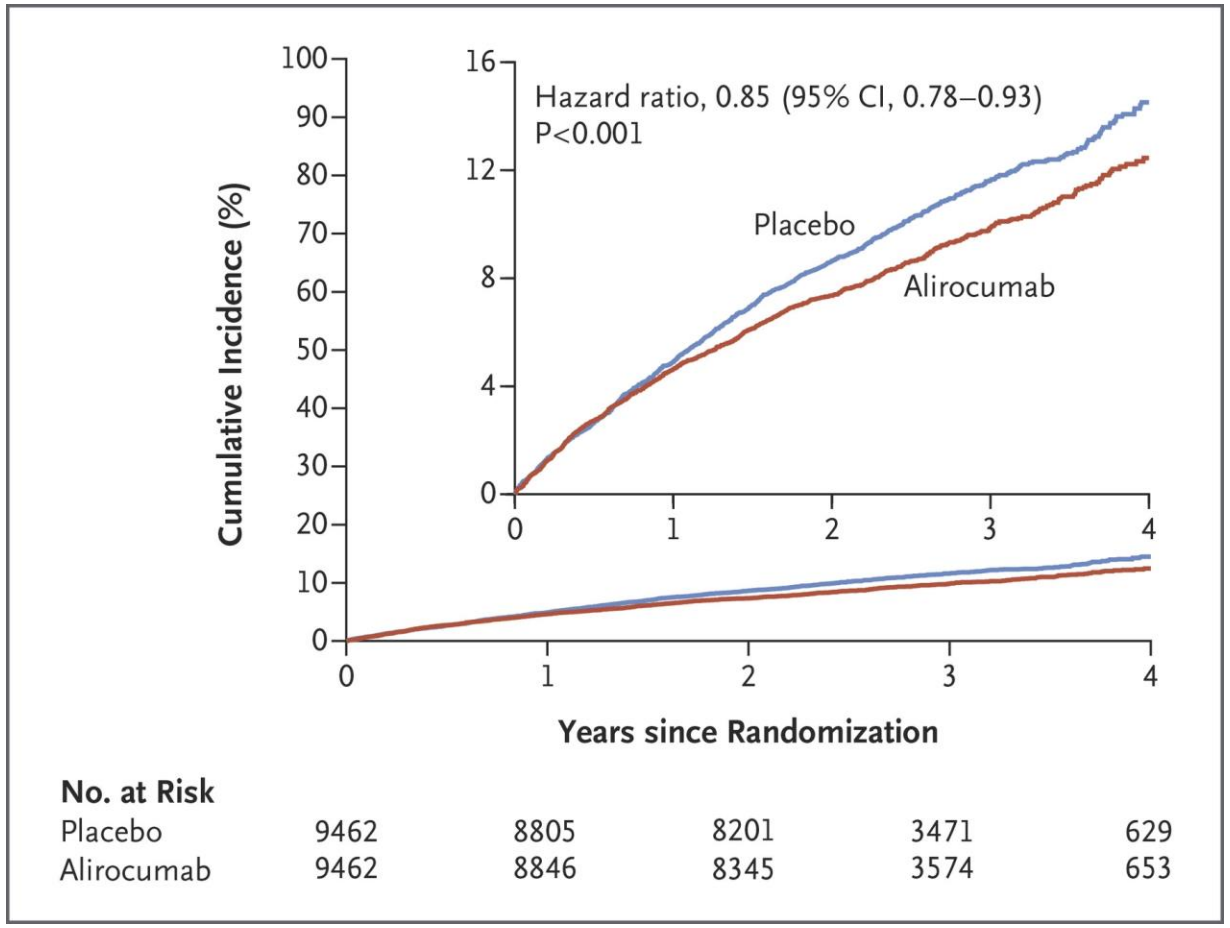
	Evolocumab (AMG 145)	Alirocumab (SAR236553 / REGN727)
Trial	**FOURIER**	**ODYSSEY Outcomes**
Sample size	**27,564**	**18,924**
Patients	**Stable ASCVD (MI, stroke, or PAD) with high-risk features**	**4-52 weeks post-ACS**
Age	63	58
Statin **High-intensity statin** **No statin**	Atorvastatin ≥20 mg or equivalent 69% 0.2%	Evidence-based medical Rx 89% 2.5%
LDL-C mg/dL (mmol/L): inclusion **Baseline LDL-C mg/dL (mmol/L)**	≥70 (≥1.8) 92 (2.4)	≥70 (>1.8) 87 (2.3)
PCSK9 inhibitor dosing	Q2W or Q4W	Q2W
Endpoint	1°: CV death, MI, stroke, revascularization, or hospitalization for UA Key 2°: CV death, MI, or stroke	CHD death, MI, ischemic stroke, or hospitalization for UA
Follow-up	26 months	34 months

CHD = coronary heart disease; CV = cardiovascular; PAD = peripheral arterial disease; Q2W/Q4W = every 2 weeks/every 4 weeks; UA = unstable angina

www.clinicaltrials.gov

Primary Endpoint





No. at Risk

Placebo	9462	8805	8201	3471	629
Alirocumab	9462	8846	8345	3574	653

- Safety and adverse events
  - No interactions
  - Itching at injection site
  - Flu-like symptoms
  - No diabetes signal

FOURIER: Efficacy and safety of very low LDL-C

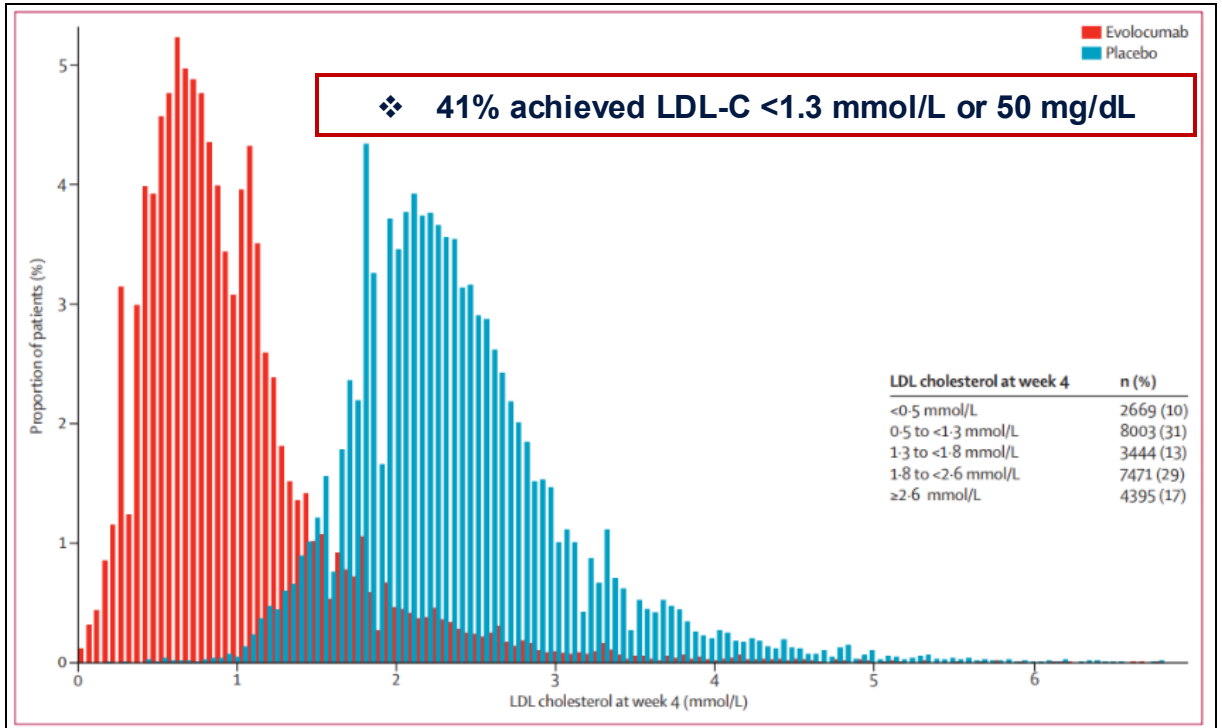
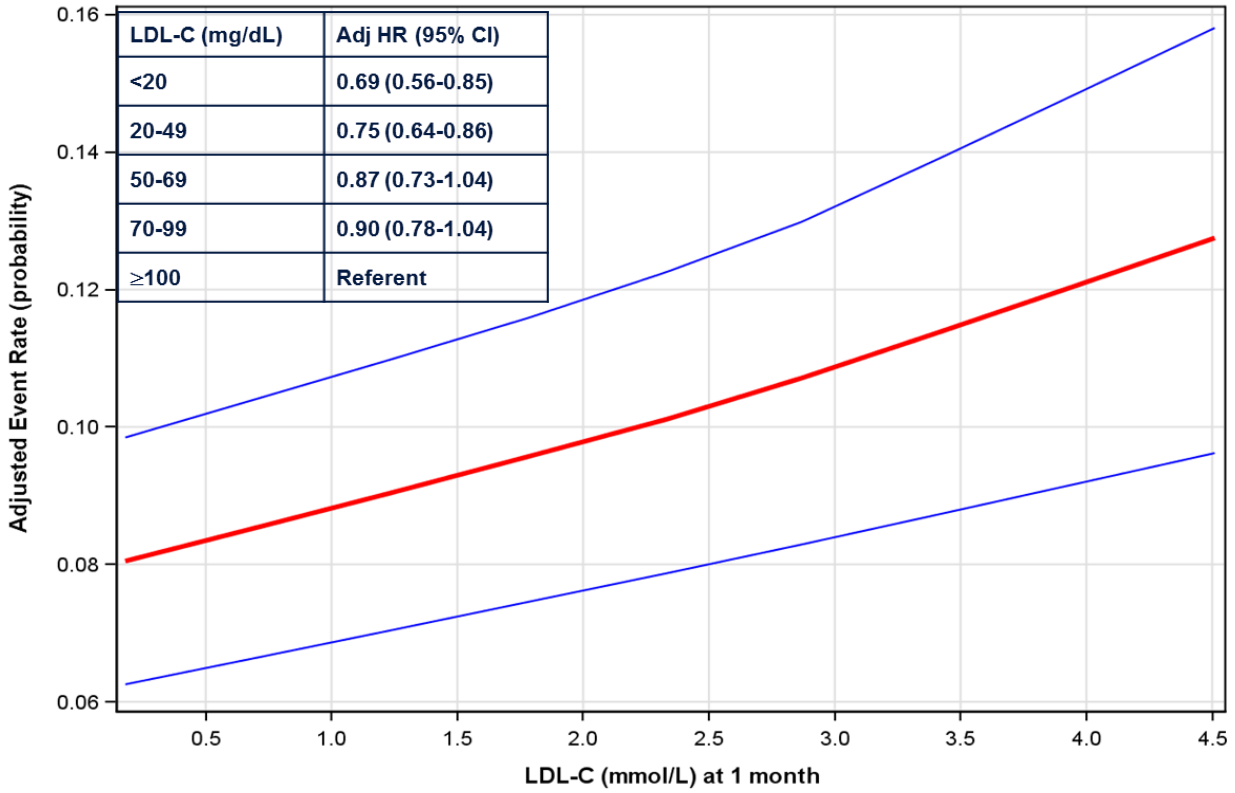
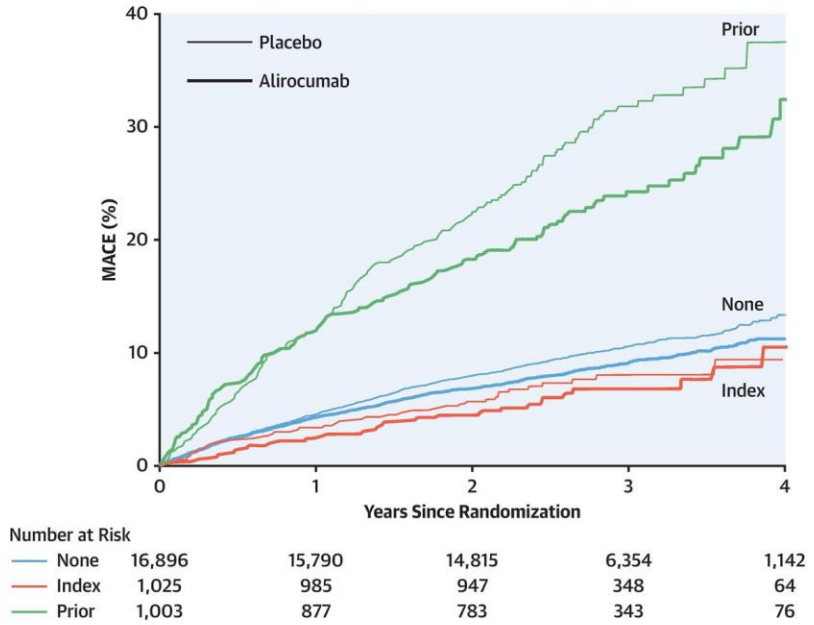


Figure 1: Distribution of achieved LDL-cholesterol concentrations at 4 weeks in patients who did not have a primary efficacy or prespecified safety event before the study
Red bars are evolocumab (median 0·8 mmol/L, IQR 0·5–1·2). Blue bars are placebo (median 2·2 mmol/L, IQR 1·9–2·7).



ODYSSEY: Prior CABG and polyvascular disease

CENTRAL ILLUSTRATION: Prior Coronary Artery Bypass Graft Status and Alirocumab Versus Placebo: Primary Endpoint

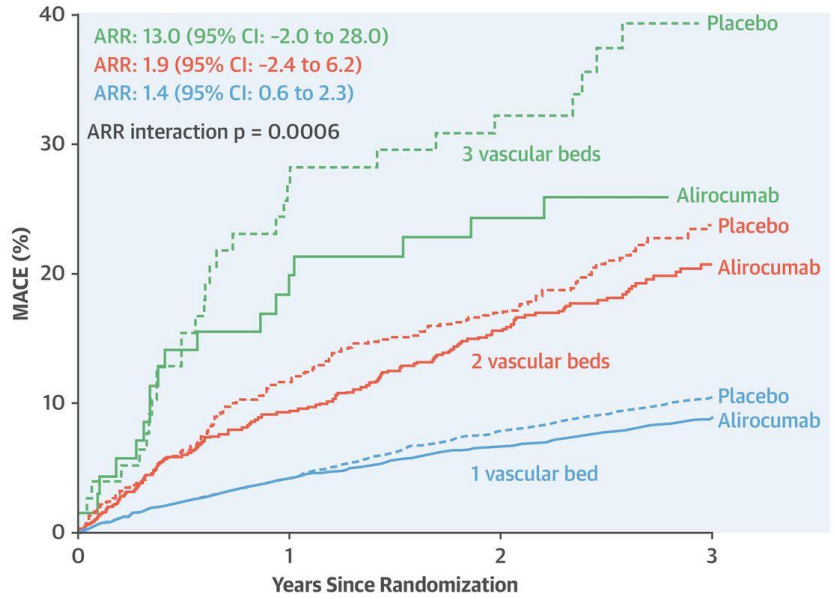


Number at Risk

None	16,896	15,790	14,815	6,354	1,142
Index	1,025	985	947	348	64
Prior	1,003	877	783	343	76

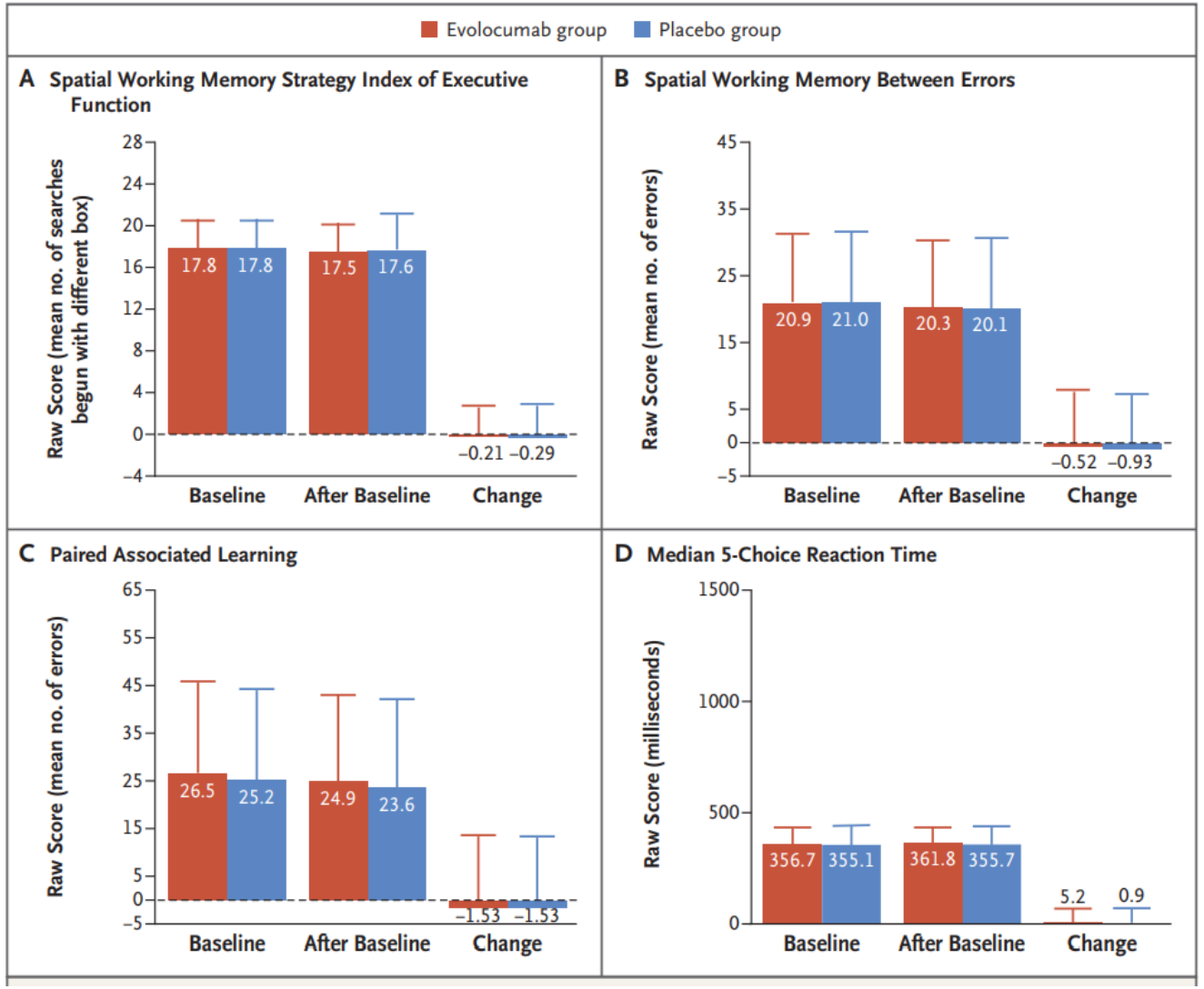
Goodman, S.G. et al. J Am Coll Cardiol. 2019;74(9):1177-86.

CENTRAL ILLUSTRATION: Alirocumab and Vascular Disease: Primary Major Adverse Cardiovascular Event Endpoint



Jukema, J.W. et al. J Am Coll Cardiol. 2019;74(9):1167-76.

EBBINGHAUS Study of cognitive function during treatment with evolocumab

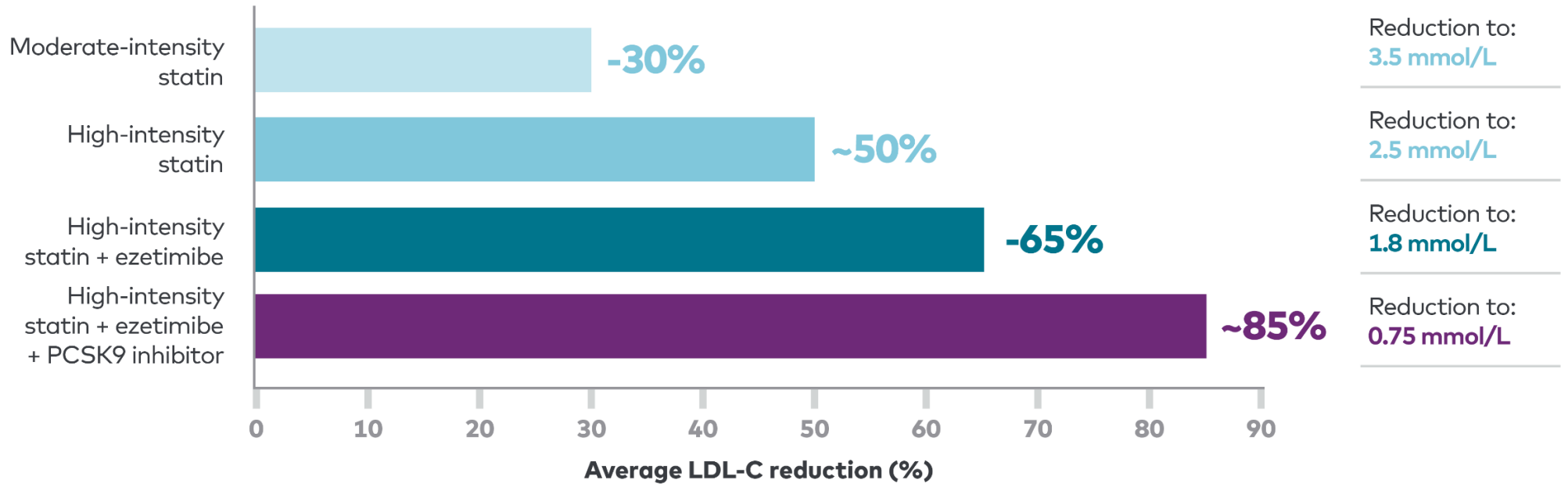


N Engl J Med 2017;377:633-43.

Inclisiran

- ORION series of clinical trials
  - ORION-9, 10, 11, 8 (OLE)
    - LDL down ~ 50 - 55%
  - CV outcomes trials pending
    - ORION-4
    - VICTORION-2 PREVENT

  - Compliance



Example
Baseline LDL-C: 5.0 mmol/L

Reduction to: **3.5 mmol/L**

Reduction to: **2.5 mmol/L**

Reduction to: **1.8 mmol/L**

Reduction to: **0.75 mmol/L**

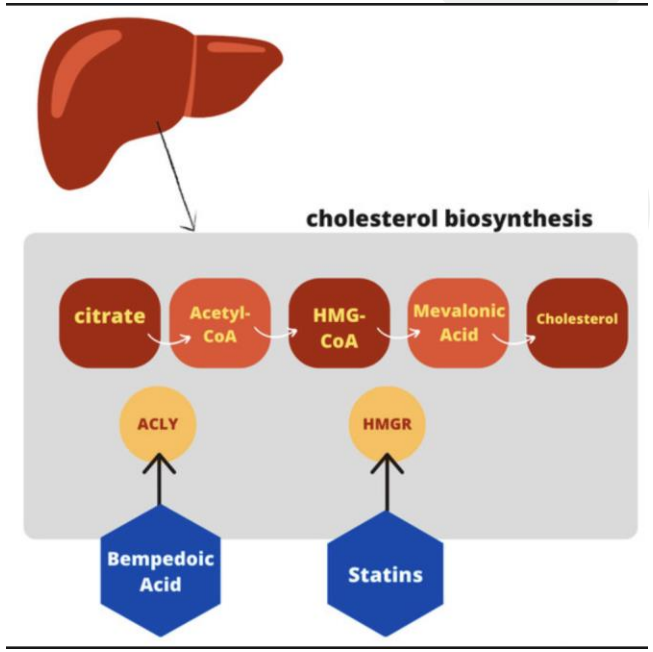
Mach F et al. Eur Heart J 2020; 41: 111–188

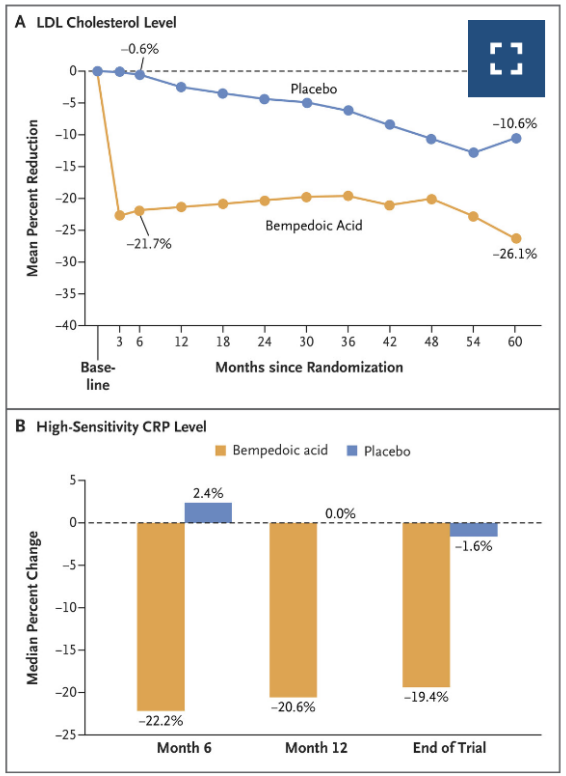
PCSK9 inhibitor eligibility (Aus)

- Max tolerated statin (atorva or rosuva) + ezetimibe
- FH criteria
  - DLCNS >=6 (or molecular diagnosis)
  - LDL > 1.8mmol/L if symptomatic ASCVD, or LDL > 5.0mmol/L
- Non-FH criteria
  - Symptomatic ASCVD and LDL > 1.8mmol/L *plus*
    - ASCVD in >=2 vascular territories
    - Multi-vessel coronary disease
    - Diabetes with microalbuminuria or age >=60 or first nations people
    - TIMI risk score secondary prevention >=4

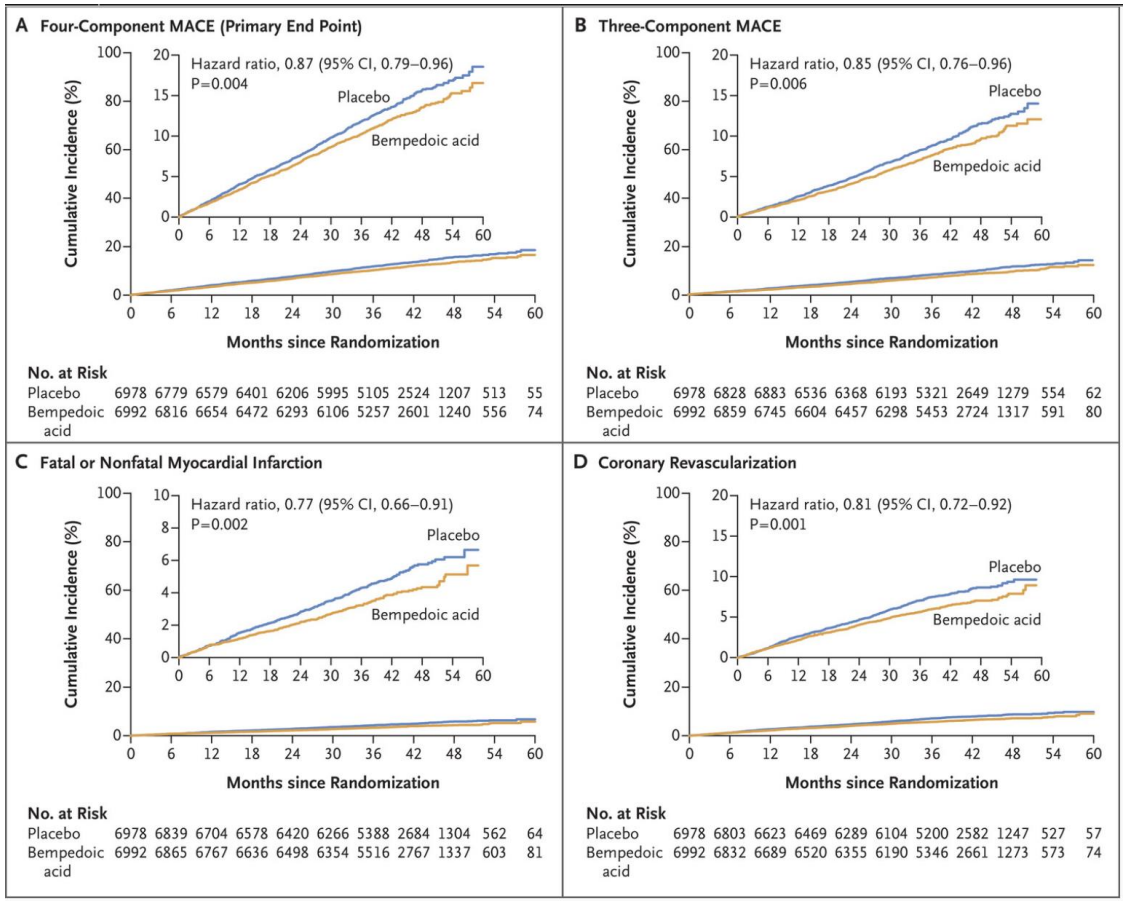
Bempedoic acid

- ATP citrate lyase inhibitor
- Prodrug, only activated in hepatocytes
  - Low incidence of muscle related side effects
- Reduced LDL synthesis
  - Upregulation of LDLR
  - ↓ LDL-C ~ 20- 30%
- Statin intolerance and PCSK9i ineligible
  - Ezetimibe + bempedoic acid ↓ LDL 50%





Changes in LDL Cholesterol and High-Sensitivity CRP Levels over Time.

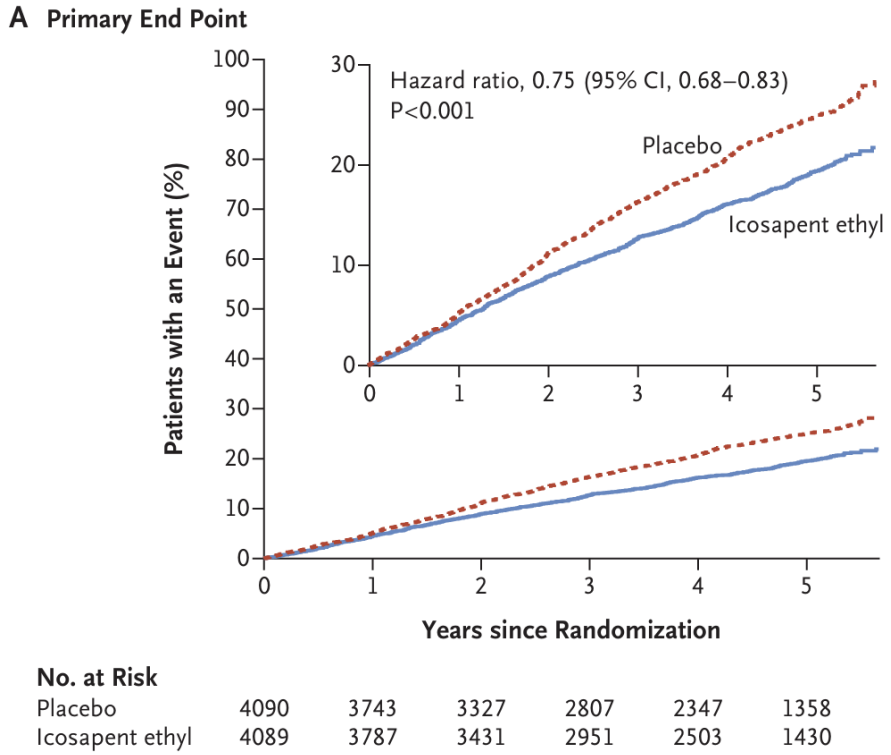


Nissen S, et al. *N Engl J Med* 2023;388:1353-1364

Omega-3 fatty acids

- Eicosapentaenoic acid (EPA)
- Docosahexaenoic acid (DHA)
- EPA + DHA combination not effective
- Over the counter preparations not effective

- EPA
  - JELIS
    - Statin + 2gm/day EPA or placebo
    - Primary and secondary prevention, high TC
    - MACE 2.8 vs 3.5%, HR 0.81 (p=0.011)
  - REDUCE-IT
    - Icosapent ethyl – 2gm bd purified EPA vs placebo
    - Established CVD or high-risk primary prevention
      - TG 1.5 – 5.6
      - LDL 1.06 – 2.59
    - MACE 17.2 vs 22.0% (HR 0.75; 95% CI: 0.68 – 0.83; p<0.0001)
    - Adverse events
      - Hospitalisation for AF 3.1 vs 2.1% (p=0.004)
      - Serious bleeding events 2.7% vs 2.1% (p=0.06)



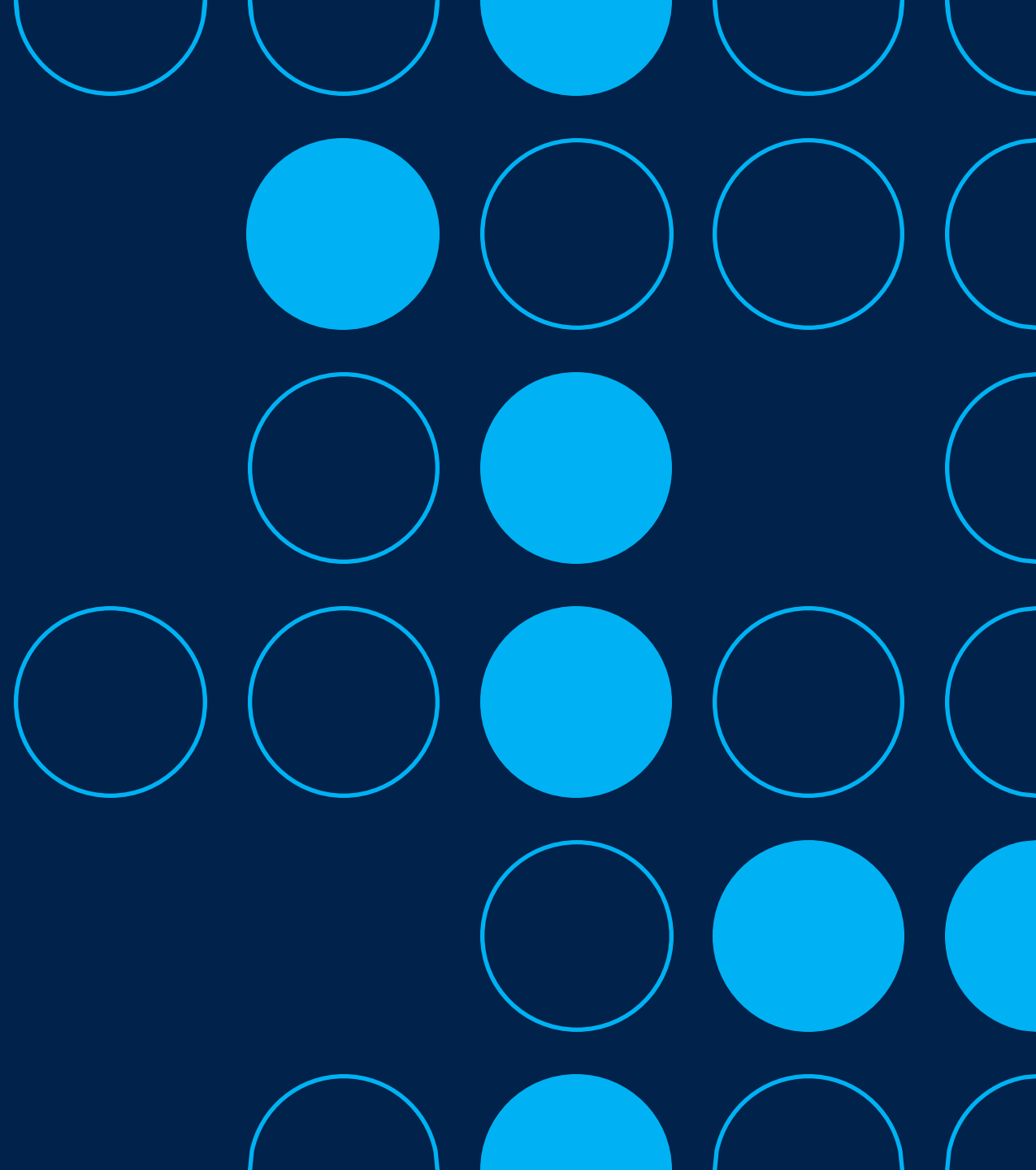
Yokoyama M, et al. *Lancet* 2007;369:1090-1098
Bhatt DL, et al. *NEJM* 2019;380:11-22

- Icosapent ethyl – PBS
  - “Established ASCVD with hypertriglyceridaemia”
  - On statin (or intolerant)
  - LDL 1.0 – 2.6mmol/L
  - fTG 1.7 – 5.6mmol/L

  - ii bd
  - Bleeding (? timing), AF

Nicotinic acid

- Raises HDL, maybe reduces LDL
- 2 RCTs – no benefit; ↑ SAE



Familial hypercholesterolemia

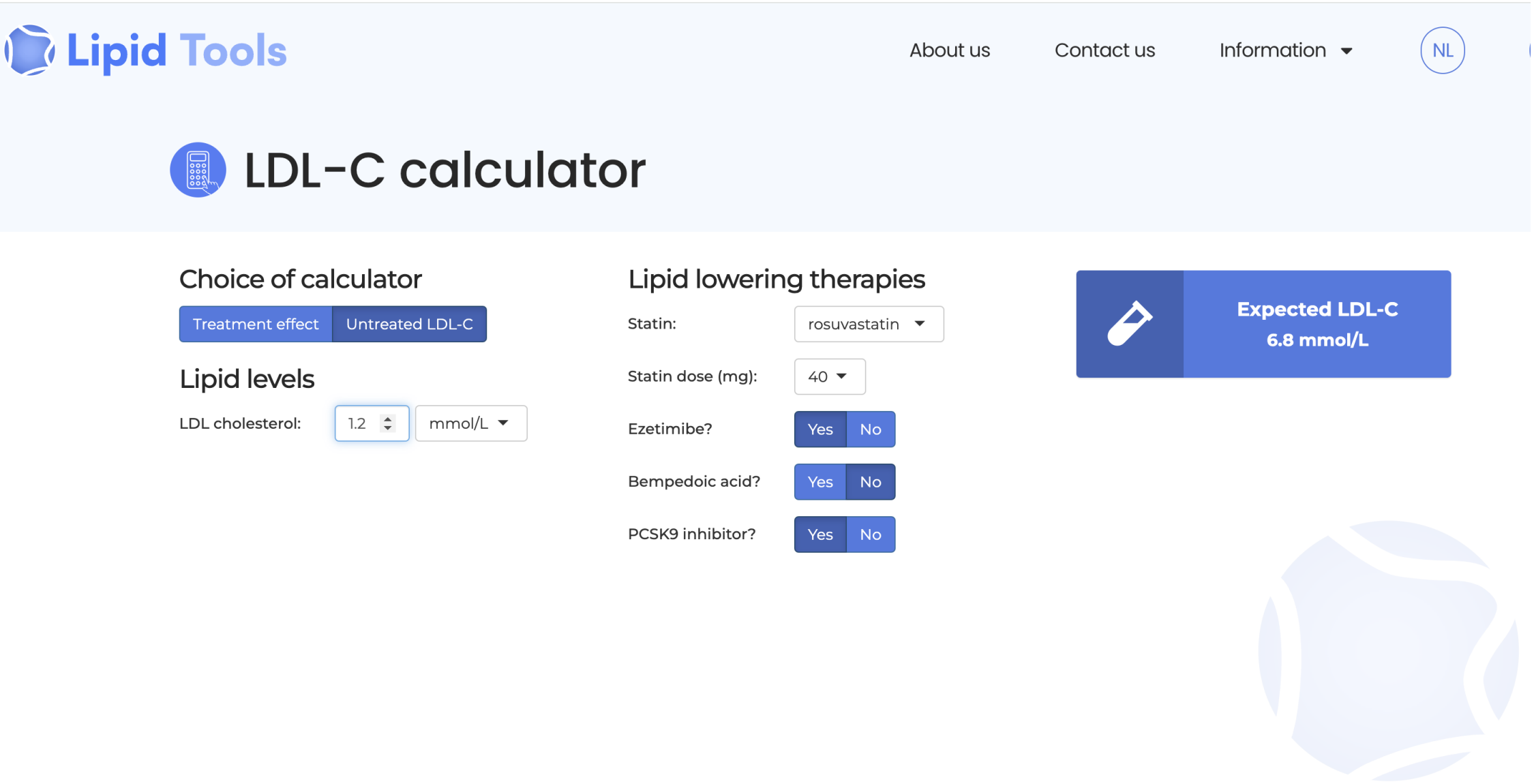
- Autosomal dominant; prevalence ~ 1:200
- Under-recognised, under-treated
- Dutch Lipid Score >=6
  - LDL >=5 mmol/L
- Vascular event risk high
- Treat early
- Genetic testing (DLS 6+) +/- cascade

Criteria		Score	Patient Score
Family history			
First degree relative with known premature coronary and/or vascular disease (men aged <55 years, women aged <60 years) OR First degree relative with known LDL-cholesterol above the 95th percentile for age and gender		1	
First degree relative with tendinous xanthomata and/or arcus cornealis OR Children aged <18 years with LDL-cholesterol above the 95th percentile for age and gender		2	
Clinical history			
Patients with premature coronary artery disease (men aged <55 years, women aged <60 years)		2	
Patients with premature cerebral or peripheral vascular disease (men aged <55 years, women aged <60 years)		1	
Physical examination			
Tendinous xanthomata		6	
Arcus cornealis before 45 years of age		4	
Investigation			
LDL-cholesterol (mmol/L)	LDL-C ≥8.5	8	
NB. This is the **untreated** LDL-cholesterol concentration. See supporting documentation for method of calculation.	LDL-C 6.5–8.4	5	
	LDL-C 5.0–6.4	3	
	LDL-C 4.0–4.9	1	
		Patient total	

Diagnosis	Total
Definite FH	**>8**
Probable FH	**6-8**
Possible FH	**3-5**
Unlikely FH	**<3**

fH Australasia Network

athero.org.au


Lipid Tools
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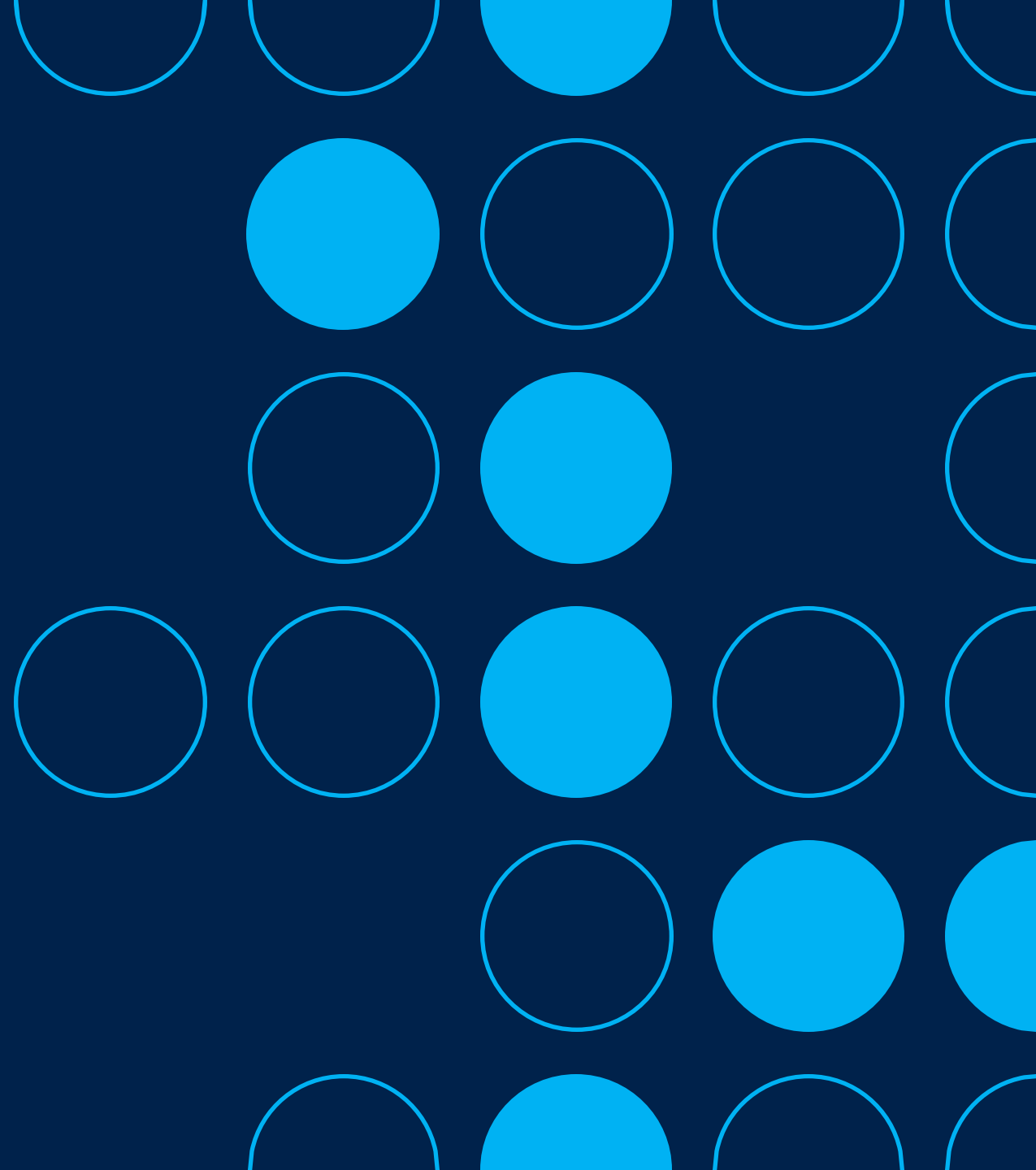
LDL-C calculator
Choice of calculator
Treatment effect
Untreated LDL-C
Lipid levels
LDL cholesterol:
1.2
mmol/L
Lipid lowering therapies
Statin:
rosuvastatin
Statin dose (mg):
40
Ezetimibe?
Yes
No
Bempedoic acid?
Yes
No
PCSK9 inhibitor?
Yes
No
Expected LDL-C
6.8 mmol/L

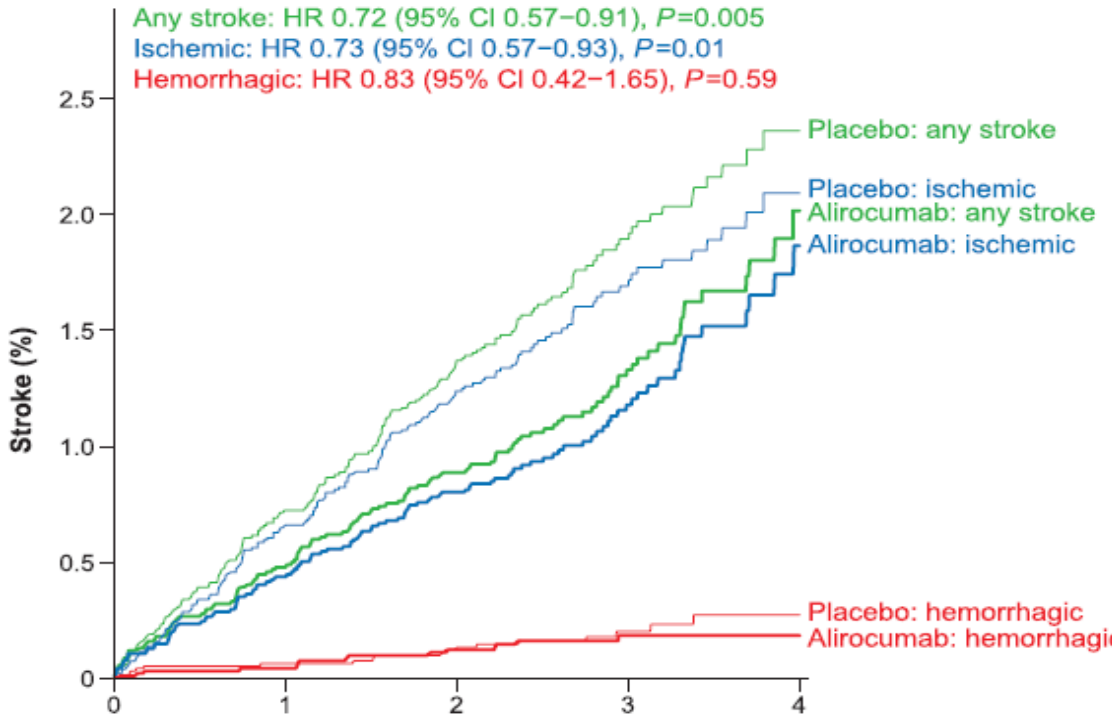


fourier

End point	Placebo, n (%) N=13780	Evolocumab, n (%) N=13784	95% CI Hazard ratio	*P* Value
All stroke	262 (1.9)	207 (1.5)	0.79 (0.66–0.95)	0.01
Ischemic	226 (1.6)	171 (1.2)	0.75 (0.62–0.92)	0.005
Hemorrhagic	25 (0.18)	29 (0.21)	1.16 (0.68–1.98)	0.59
Unknown	14 (0.10)	13 (0.09)	0.93 (0.44–1.97)	0.84
Ischemic stroke or TIA	295 (2.1)	229 (1.7)	0.77 (0.65–0.92)	0.003
TIA	76 (0.55)	61 (0.44)	0.80 (0.57–1.12)	0.20

Giugliano et al. *Stroke* 2020;51:1546-54

ODYSSEY OUTCOMES



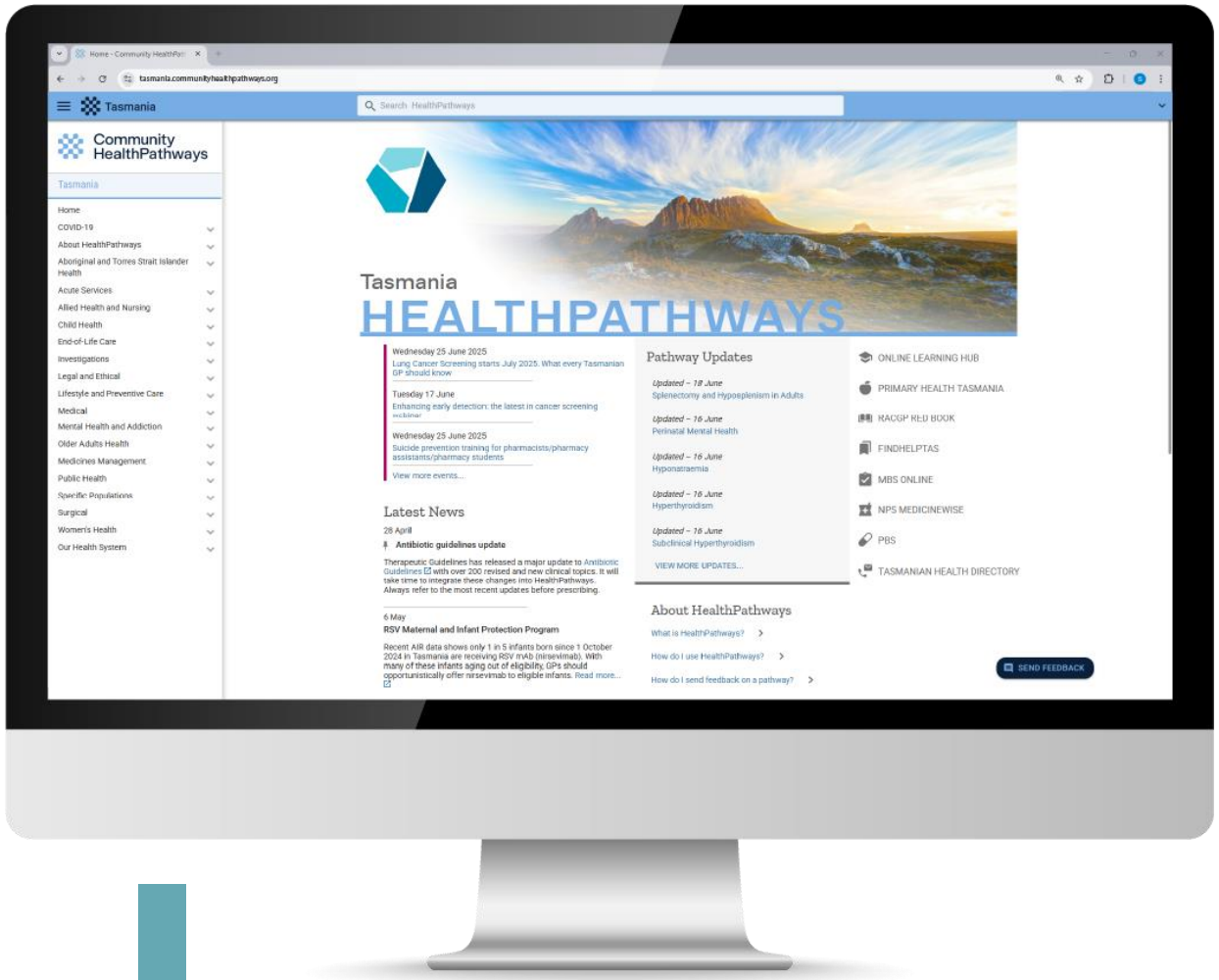
Jukema et al. *Circ* 2019;140:2054-62

Tasmanian HealthPathways

Tasmanian HealthPathways

is a web-based information portal developed by Primary Health Tasmania. It is designed to help primary care clinicians plan local patient care through primary, community and secondary healthcare systems.

tasmania.communityhealthpathways.org



Tasmanian HealthPathways

Cardiology

In This Section

- Acute Coronary Syndrome
- Anticoagulation
- Atrial Fibrillation (AF)
- Cardiac Catheterisation Complications
- Cardiovascular Drugs and Monitoring
- Cardiovascular Risk Assessment (CVRA)
- Chest Pain
- Presyncope and Syncope
- Heart Failure
- Heart Murmurs in Adults
- Heart Valve Disease
- Hyperlipidaemia and Familial Hypercholesterolaemia
- Infective Endocarditis Prophylaxis
- Long QT Syndrome
- Palpitations
- Postoperative Care of Cardiac Patients
- Cardiology Requests

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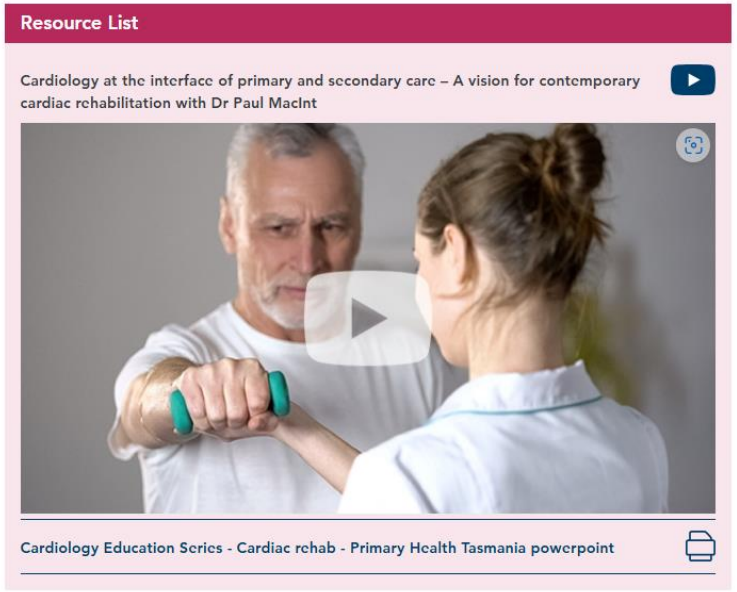
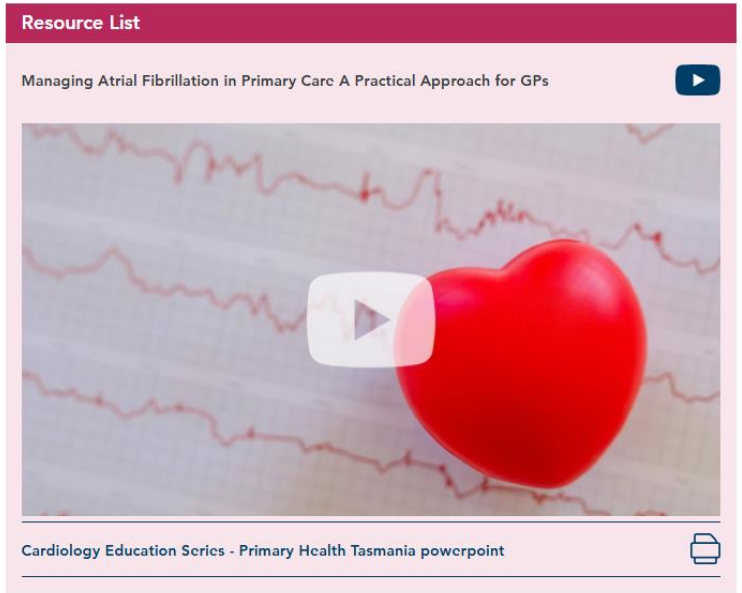
Cardiology Series

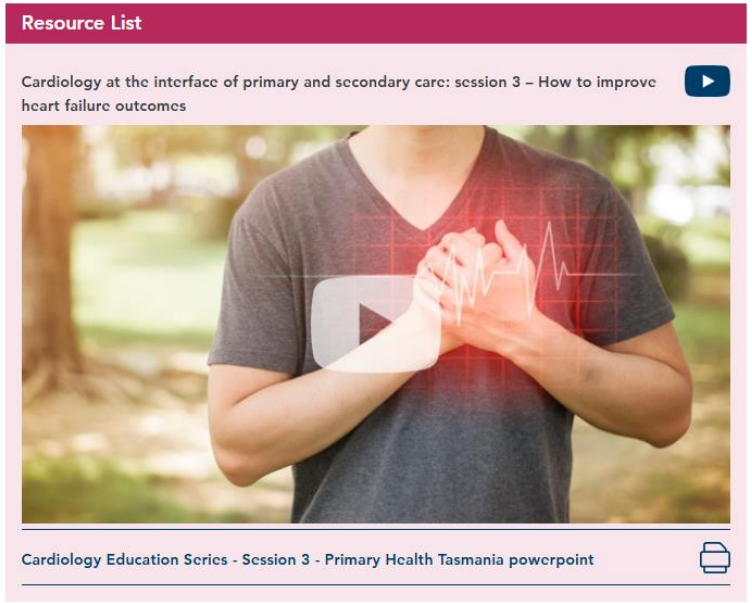
Cardiology at the interface of primary and secondary care – A vision for contemporary cardiac rehabilitation with Dr Paul MacIntyre



Cardiology at the interface of primary and secondary care – Managing Atrial Fibrillation in Primary Care: A Practical Approach for GPs



Cardiology at the interface of primary and secondary care – How to improve heart failure outcomes



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