Cardiovasculaire casuïstiek en innovatie

Een interactief avondprogramma

Gepersonaliseerde lipidenverlaging in de praktijk

Woensdag 26 juni 2024

NLA
Nederlandse Lipiden Academie

PANORAMA
Personalized Algorithms, based on Novel Outcomes Reshaping cArdiovascular Management Approaches

CVGK

Agenda




19:00 uur
Introductie
Prof. dr. Erik Stroes, internist-vasculair geneeskundige, Amsterdam UMC

19:10 uur
De plaats van PCSK9-inhibitie bij atherosclerotische stenose
Dr. Bimmer Claessen, interventiecardioloog, Amsterdam UMC

19:40 uur
Klinische inertie en het belang van therapietrouw
Daan van den Bersselaar, verpleegkundig specialist, Catharina Ziekenhuis, Eindhoven

20:10 uur
Residueel cardiovasculair risico na acuut coronair syndroom
Dr. Sanne van Wissen, internist-vasculair geneeskundige, OLVG, Amsterdam

20:45 uur
Einde webinar

Nederlandse Lipiden Academie

Accreditatie

Nederlandse Vereniging voor Cardiologie

Om in aanmerking te komen voor accreditatiepunten dient u mee te doen met de interactieve polls

Interactie met de sprekers tijdens het programma

Via de livestream kunt u:

- Vragen stellen aan de sprekers
- Meedoen met interactieve polls

Dit webinar is financieel mogelijk gemaakt door:

AMARIN

AMGEN

NOVARTIS

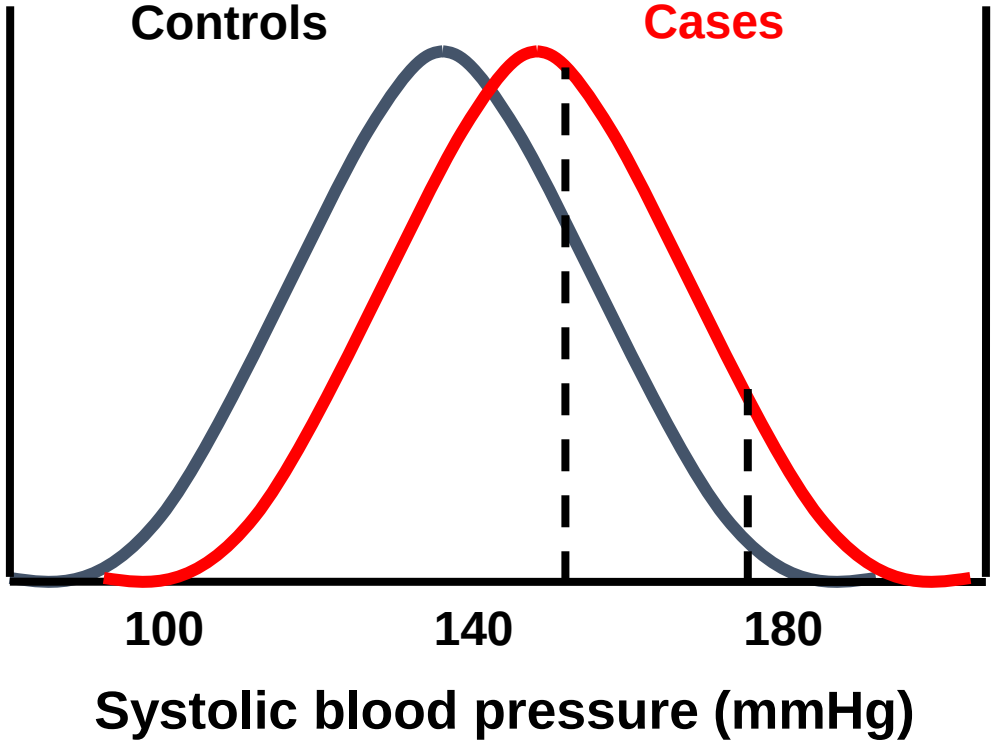
Introductie

Prof. dr. Erik Stroes

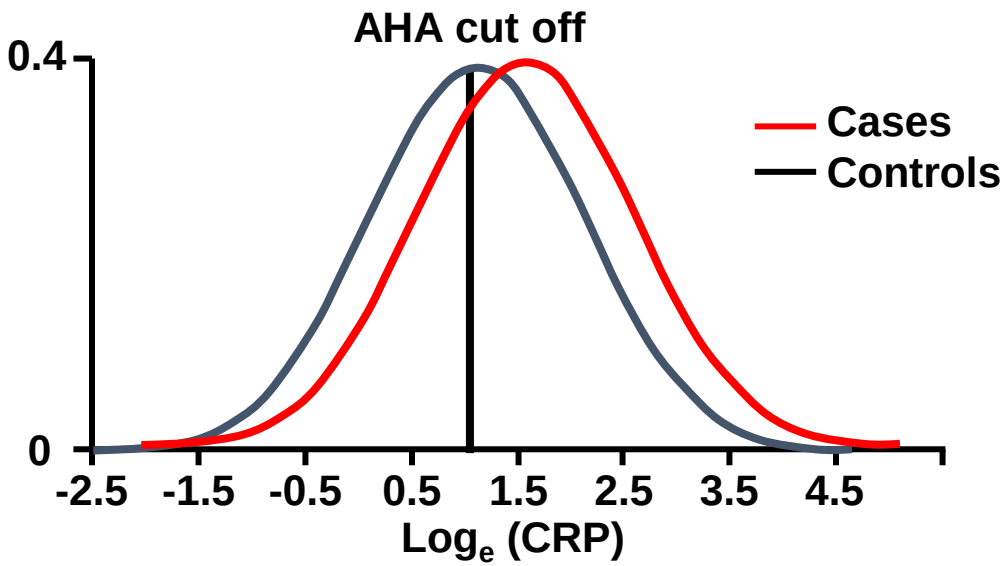
Internist-vasculair geneeskundige, Amsterdam UMC

For optimized tailored therapy, we need to correctly determine (future) CV-risk

Impact of RR on CV-event



Impact of CRP on CV-event



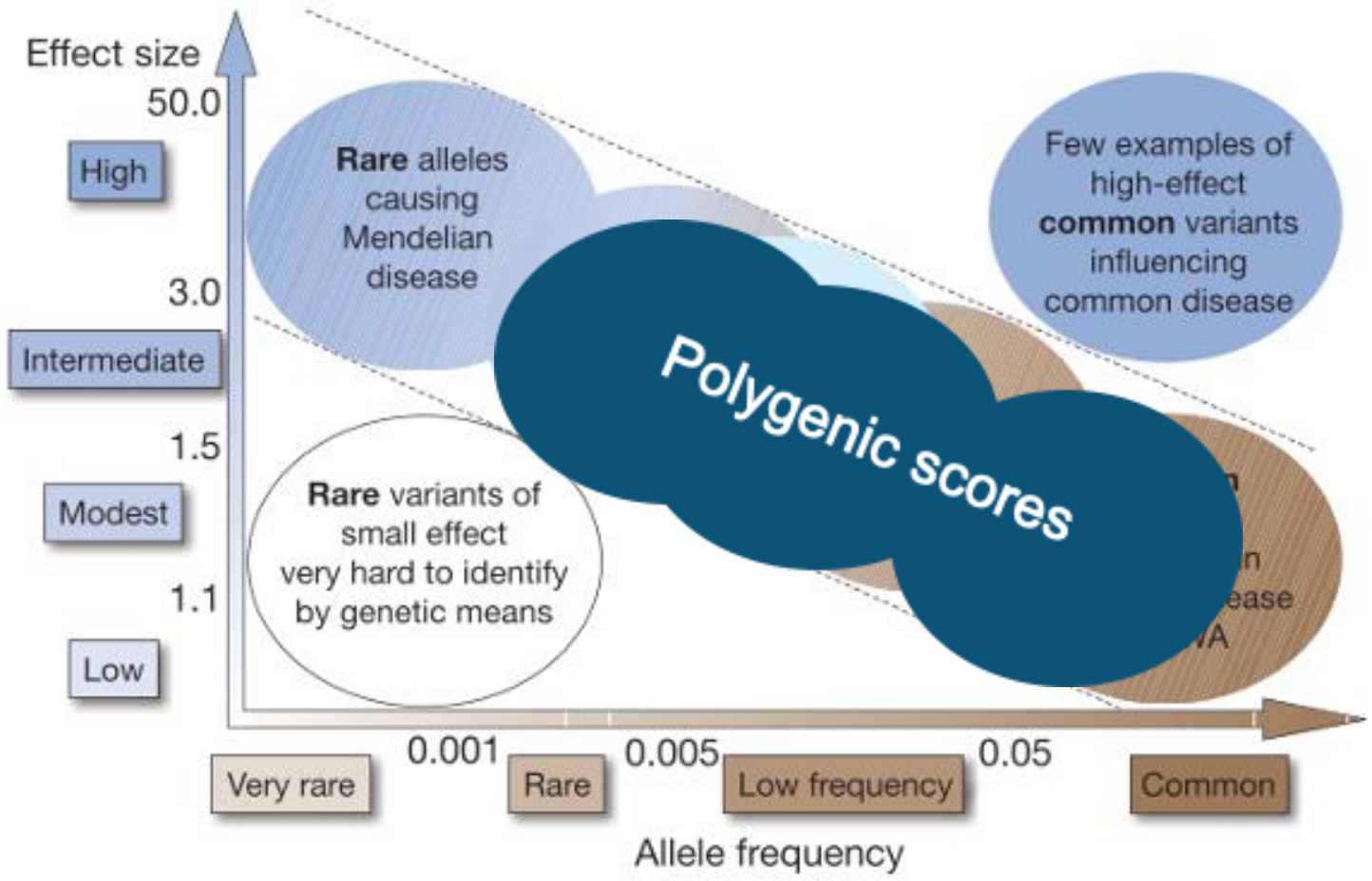
Rose G. Int J Epidemiol 1985;14:32-38. Wald NJ. BMJ 1999;319:1562-65. Law J Med Screen 2004;11:3-7

Why do risk algorithms perform so poor ?

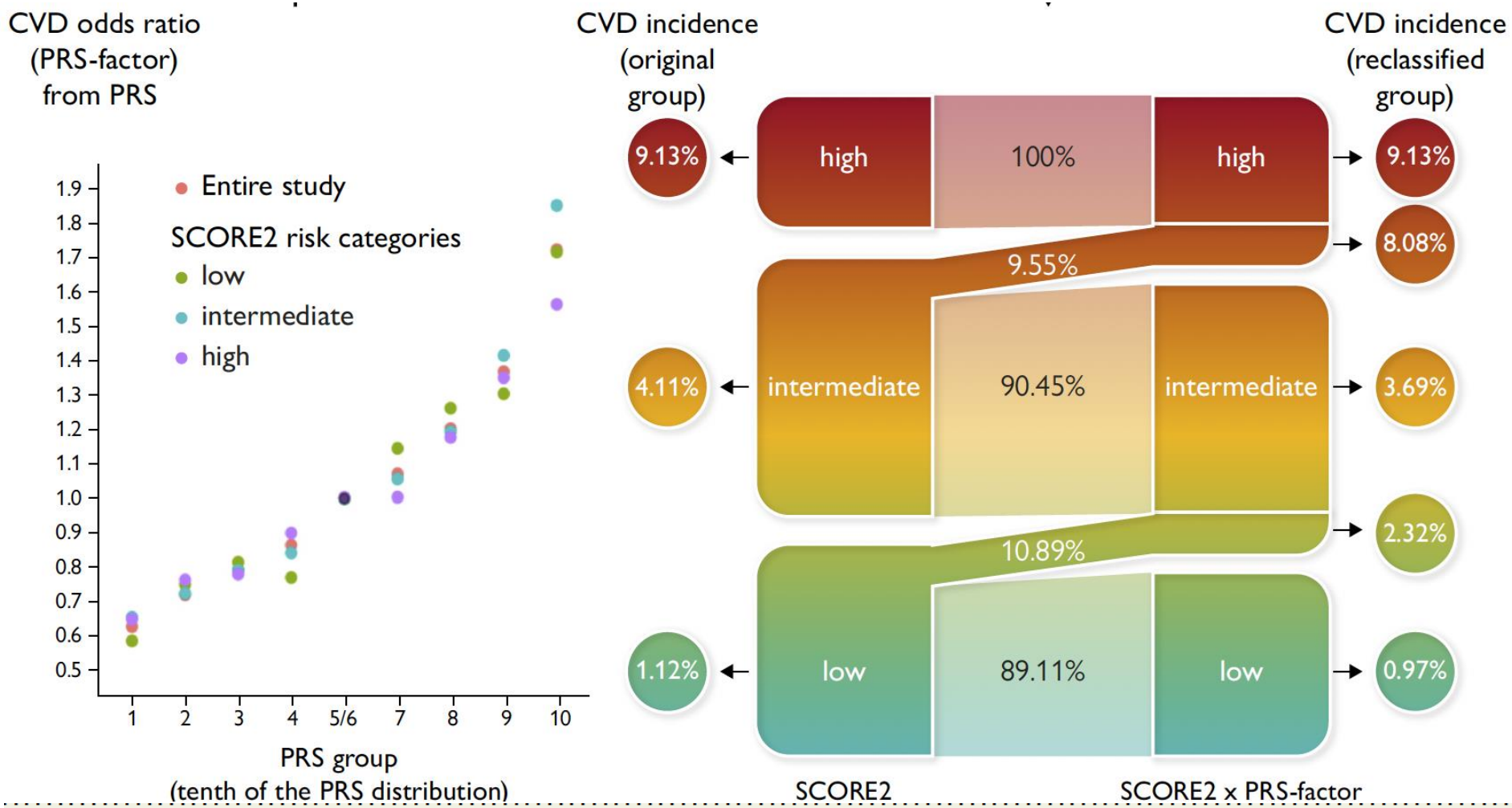
- they assume all risk factors have equal detrimental effect in all subjects
  denying the huge variation in 'athero-protective' factors between subjects
- they do not incorporate the impact of other 'established' risk factors
  MASLD, renal, pulmonary, chronic inflammatory, etc
- they do not account for novel 'not-yet fully characterized' risk factors
  clinical, psychological, environmental, genetic, etc

Can we do better?
assessing atherogenic vulnerability by genetic phenotyping

Rationale:
CV-disease encompasses a substantial genetic component



Use of GPS_{CAD} reclassifies guideline-recommended prediction of CVD

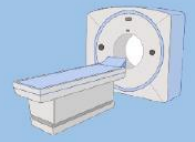


This study demonstrates that absolute CVD risk, determined by a clinical risk score, and relative genetic risk, determined by a PRS, provide independent information. The two components may form a simple multiplicative model improving precision of guideline-recommended tools in predicting incident CVD.

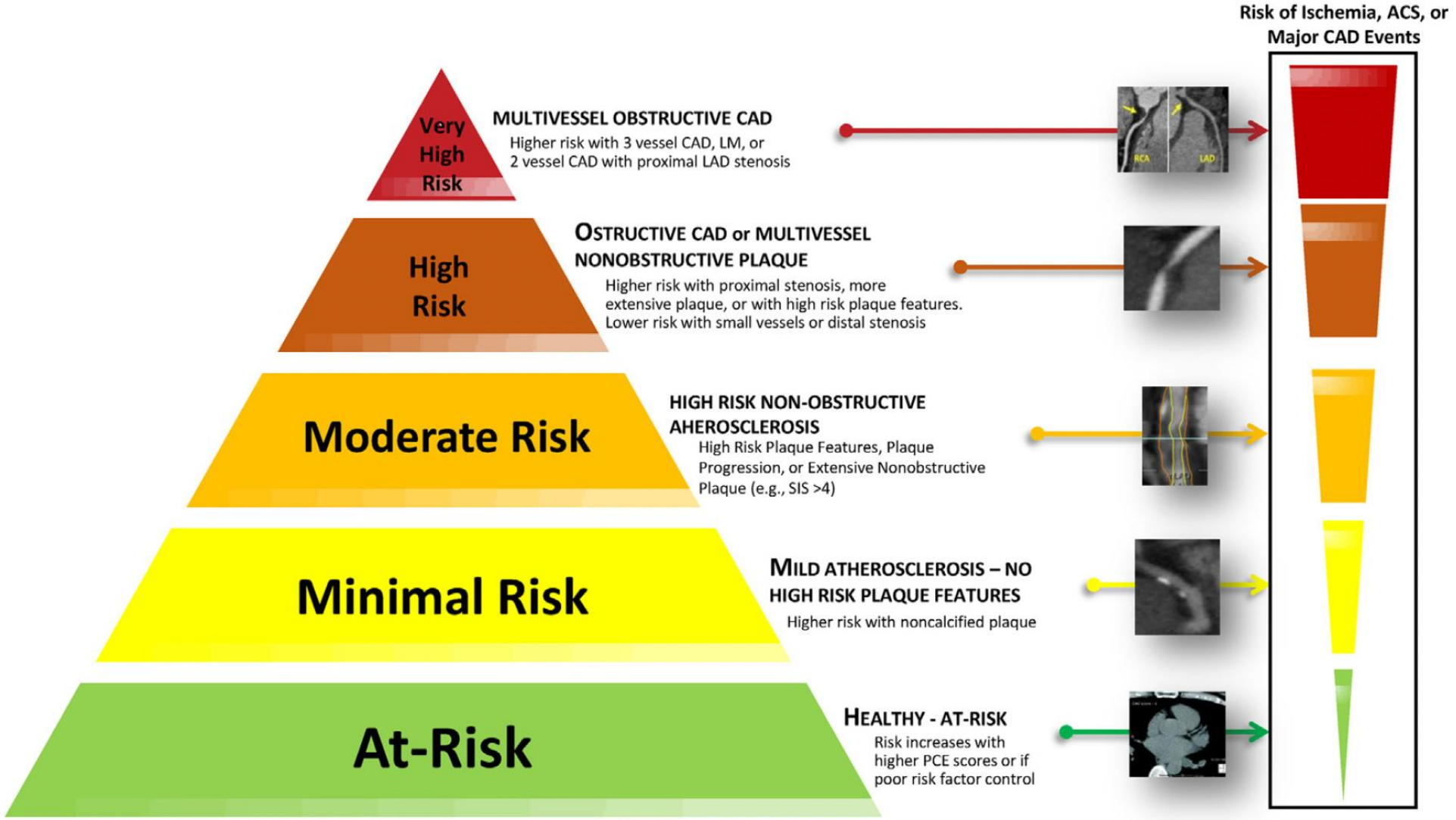
Li, Schunkert, Eur H J 2023

Can we do better?
assessing atherogenic vulnerability by imaging

Biological Aging Approach to Risk Assessment

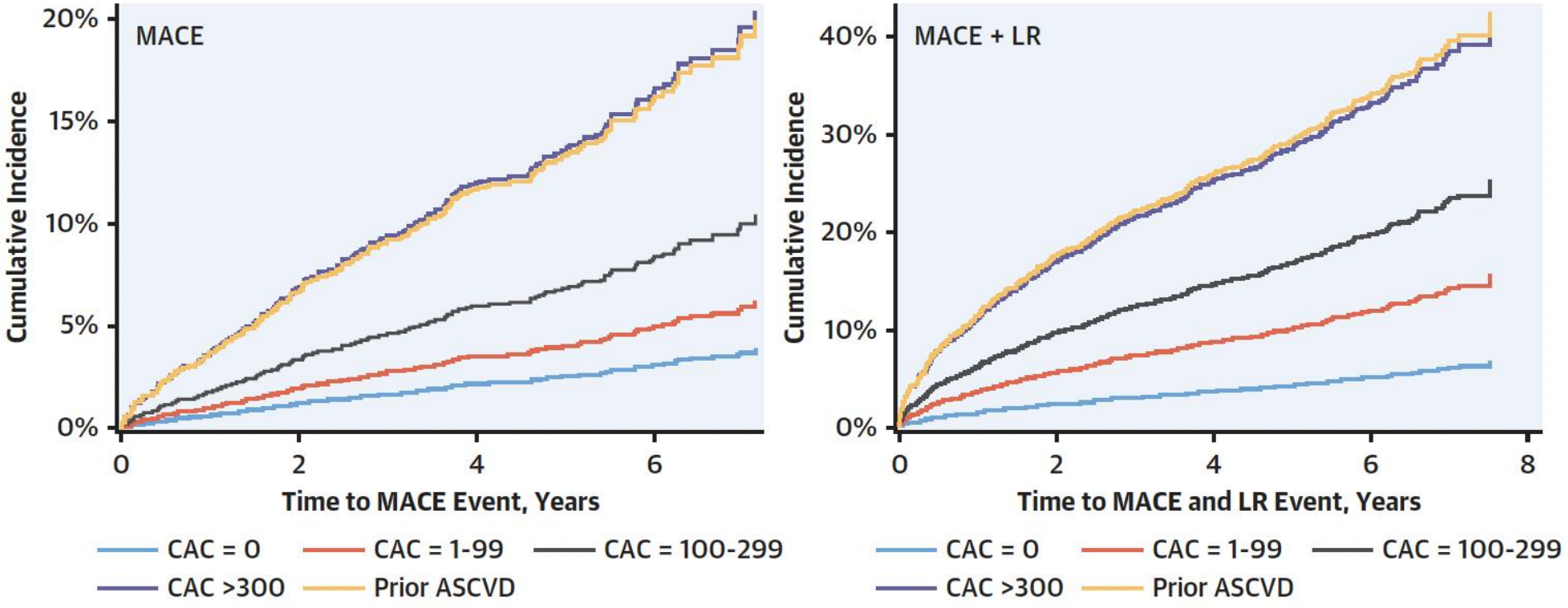


CAC scan



Significant coronary calcification (CAC > 300) equals risk in secondary prevention

CONFIRM registry



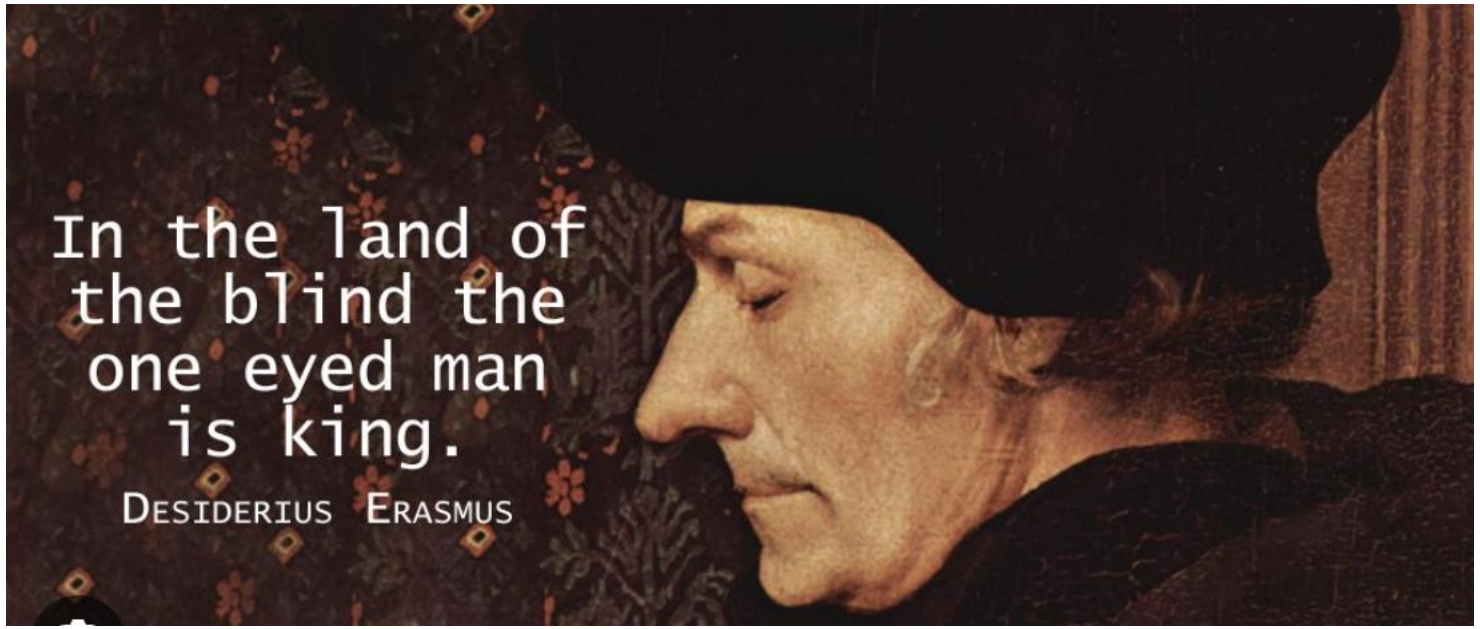
CONCLUSIONS Patients with CAC scores >300 are at an equivalent risk of MACE and its components as those treated for established ASCVD. This observation, that those with CAC >300 have event rates comparable to those with established ASCVD, supplies important background for further study related to secondary prevention treatment targets

Budoff M, JACC CV Imaging 2023

Having correctly identified '(very) high risk' subjects, we need to optimally 'treat' the (causal) risk factors

the case for LDL-cholesterol

1995-97	**Statins**
2002	Ezetimibe
2013	Lomitapide
2016	PCKS9 inhibition
2019	Cholestagel
2021	Inclisiran
2022	Bempedoic acid
2023	Evinacumab



If you ***CAN'T*** do better, statin-only is '***acceptable***'
If you ***CAN*** do better, lack of uptitration is ethically '***unacceptable***'

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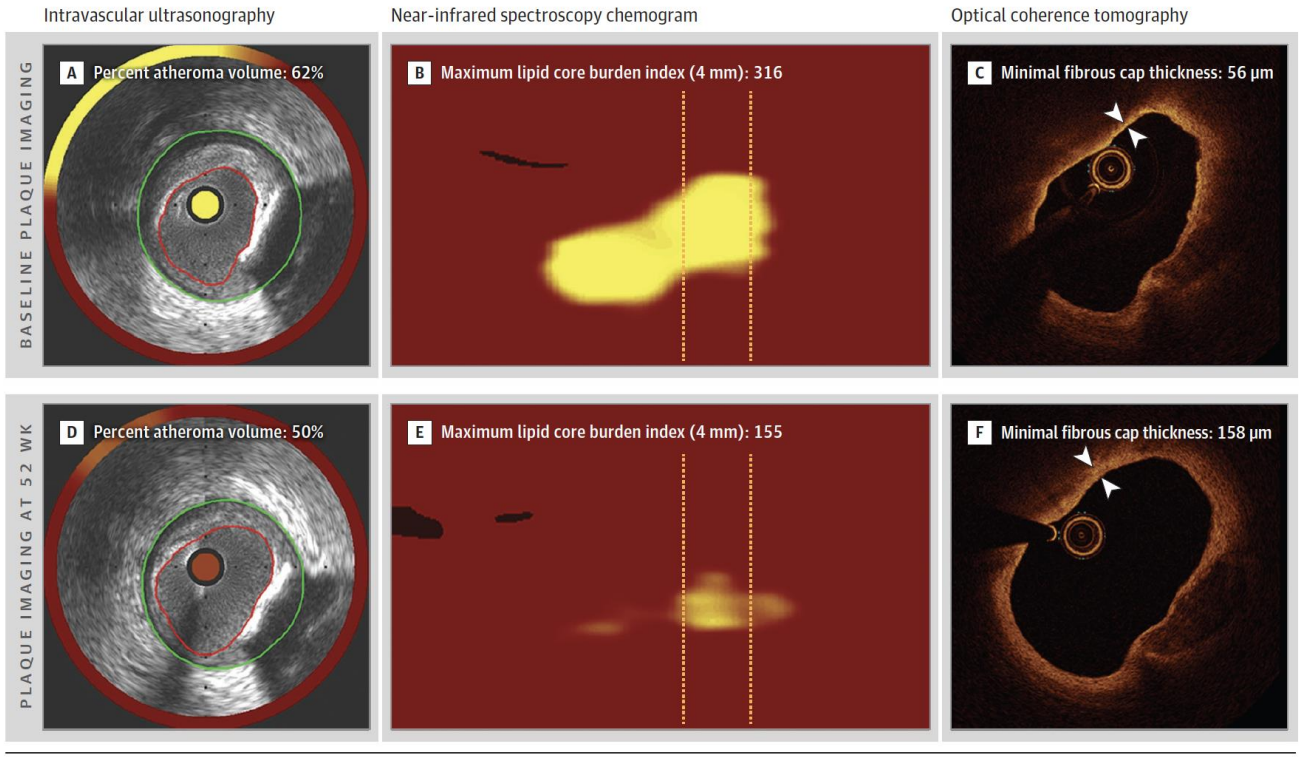
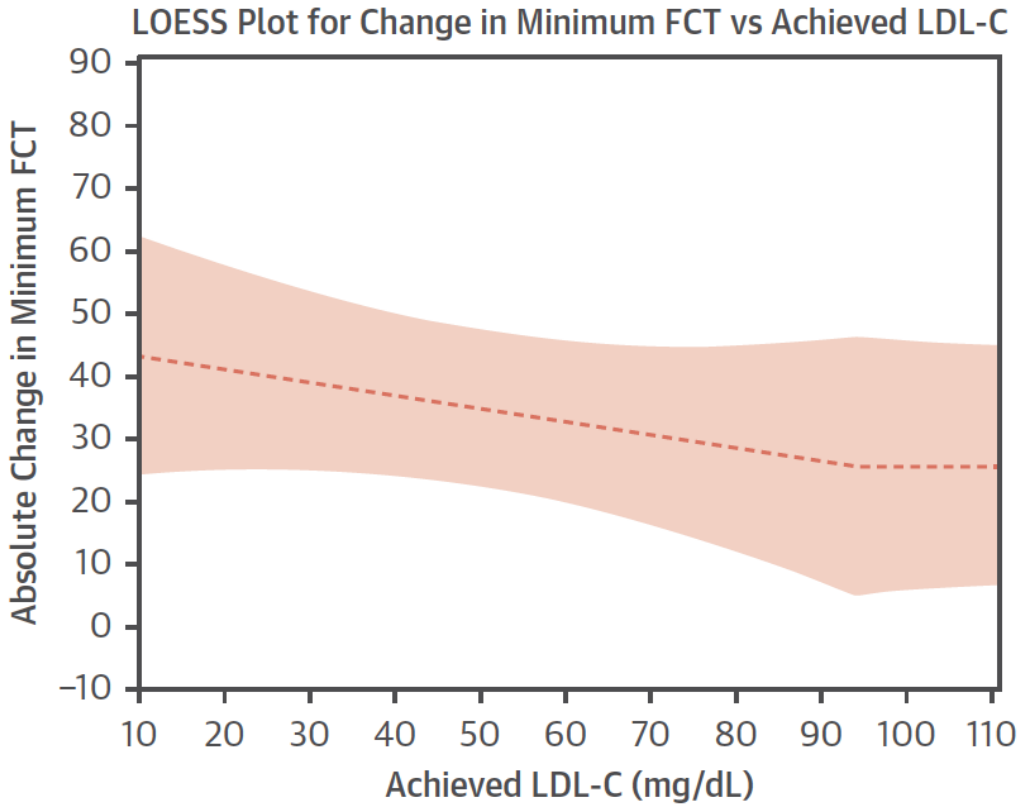
Imaging studies:
At very low LDL-C: plaque stabilisation

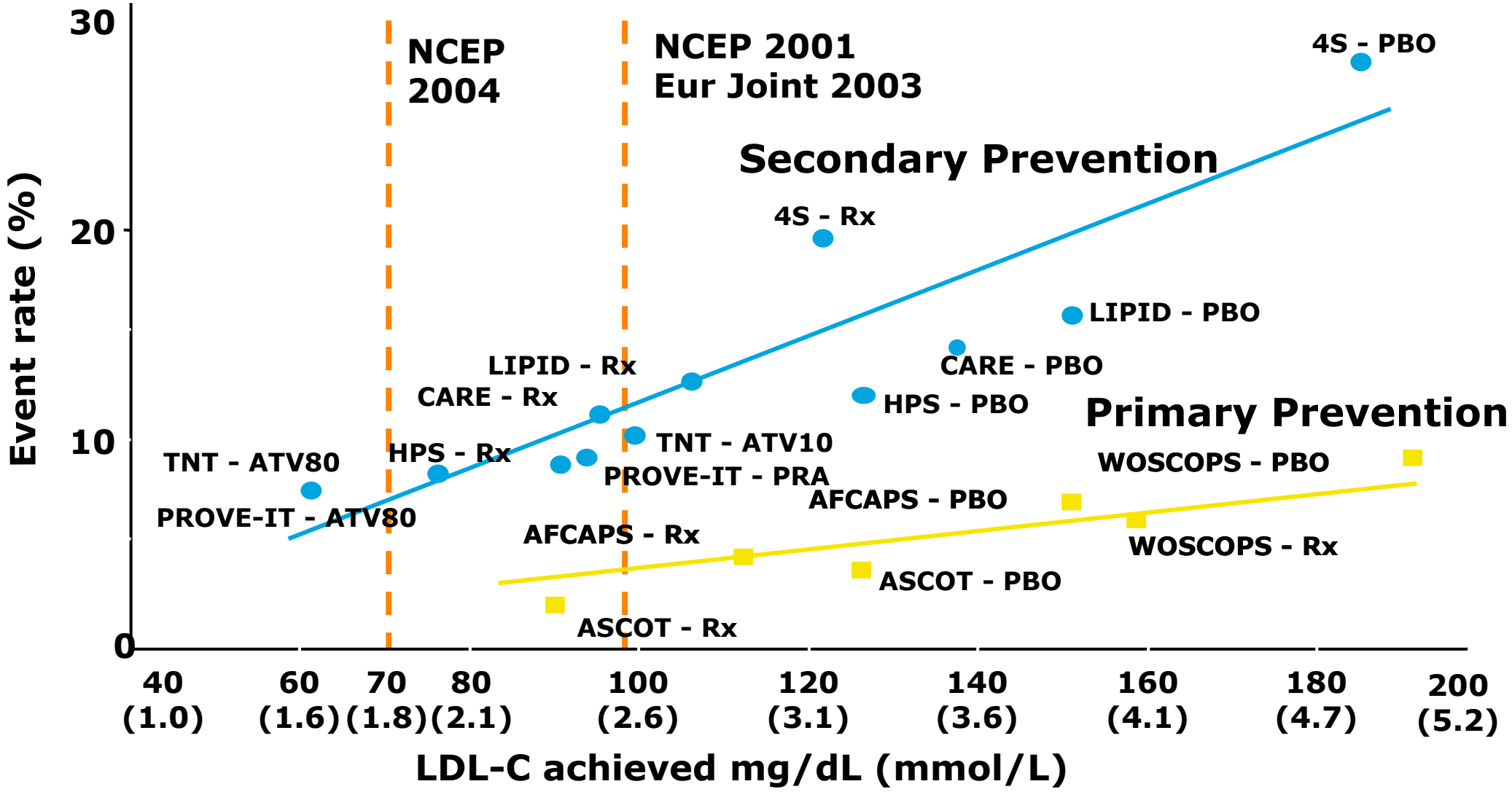
PACMAN-AMI & *HUIJGENS trials*





Intensive LDL-C lowering induces plaque stabilisation

CV-outcome studies:
Lower achieved LDL-C is Lower CV-risk



Adapted from Rosensen, Exp Opin Emerg Drugs 2004;9:269; LaRosa J et al, N Engl J Med,2005;352:1425

CV-outcome studies:
Non-statin LDL-C reduction equally beneficial

	3-Component MACE	**(Non)fatal MI**
IMPROVE-IT *Ezetimibe*	0.90	0.87
FOURIER *Evolocumab*	0.80	0.73
ODYSSEY Outcomes *Alirocumab*	0.86	0.86
CLEAR OUTCOMES *Bempedoic acid*	0.85	0.77

Casus 'Bimmer Claessen'

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Cannon *NEJM*2015; Sabatine *NEJM* 2017; Schwartz *NEJM* 2017; Nissen S, NEJM 2023

Current guidelines: Dynamic LDL-C target levels: *higher* risk requires *lower* LDLc target level

Risk category	LDL goals (starting with untreated LDL-c)	
	2016	2019
Very high risk	<70 mg/dl (1.8 mmol/l) or >50% ↓ if LDL-c 70-135 mg/dl (1.8–3.5 mmol/l)	**<55 mg/dl (1.4 mmol/l) and >50% ↓**
High risk	<100 mg/dl (2.6 mmol/l) or >50% ↓ if LDL-c 100-200 mg/dl (2.6–5.2 mmol/l)	**<70 mg/dl (1.8 mmol/l) and >50% ↓**
Moderate risk	<116 mg/dl (3 mmol/l)	<100 mg/dl (2.6 mmol/l)
Low risk	<116 mg/dl (3 mmol/l)	<116 mg/dl (3 mmol/l)

For patients with ASCVD experiencing a second vascular event within 2 years while taking maximally tolerated statin therapy, an LDL-c goal of <40 mg/dl (1.0 mmol/l) may be considered

IIb | B

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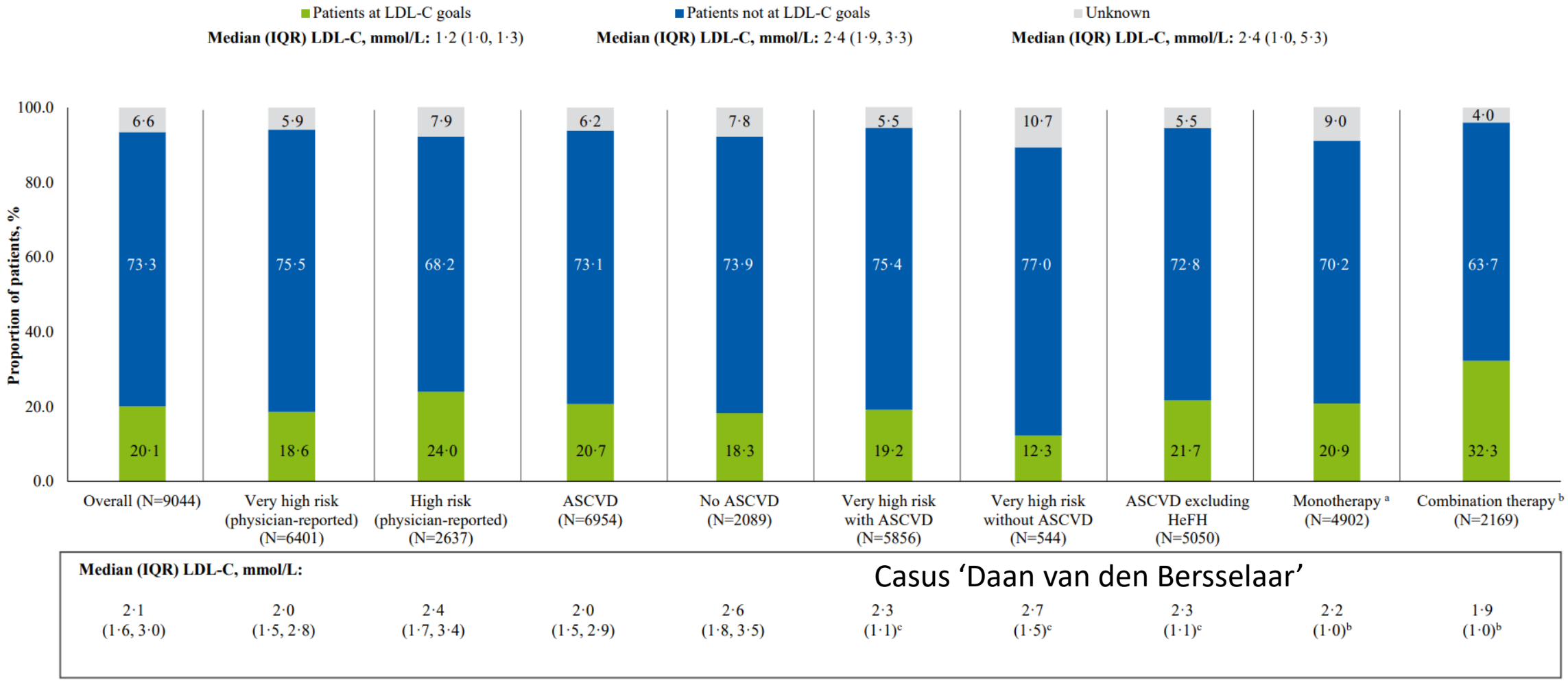
Adapted from: Catapano AL, et al. Eur Heart J 2016;37:2999-3058. Mach F, et al. Eur Heart J 2020; 41: 111-188

LDL-C target achievement is achievable:
The LDL-C lowering toolbox is full

	Expected LDL-C reduction			
	Statin tolerant		Statin intolerance*	
	>60%	>80%	>35%	>60%
Current options	Rosuvastatin 20-40 + Ezetimibe 10	Rosuvastatin 20-40 + Alirocumab/Evolocumab (+ ezetimibe 10)	Ezetimibe 10 plus bile acid abs	
	Atorvastatin 40-80 + Ezetimibe 10	Atorvastatin 40-80 + Alirocumab/Evolocumab (+ ezetimibe 10)		Ezetimibe 10 + Alirocumab/Evolocumab*
	Rosuvastatin 5-10 + Alirocumab/Evolocumab			
	Atorvastatin 10-20 + Alirocumab/Evolocumab			
Emerging options	Rosuvastatin 5-10 + inclisiran	Atorvastatin 40-80 + inclisiran (+ ezetimibe 10)	Bempedoic acid 180 + Ezetimibe 10	Bempedoic acid 180 + Ezetimibe 10 + PCSK9 targeted therapy**
	Atorvastatin 10-20 + inclisiran	Rosuvastatin 20-40 + inclisiran (+ ezetimibe 10)		Ezetimibe 10 + Inclisiran
	Atorvastatin 20 + Ezetimibe 10 + Bempedoic acid 180	High intensity statin + oral PCSK9 inhibitor		High intensity statin + oral PCSK9 inhibitor

How are we performing on LDL-target achievement in the various CV-risk categories?

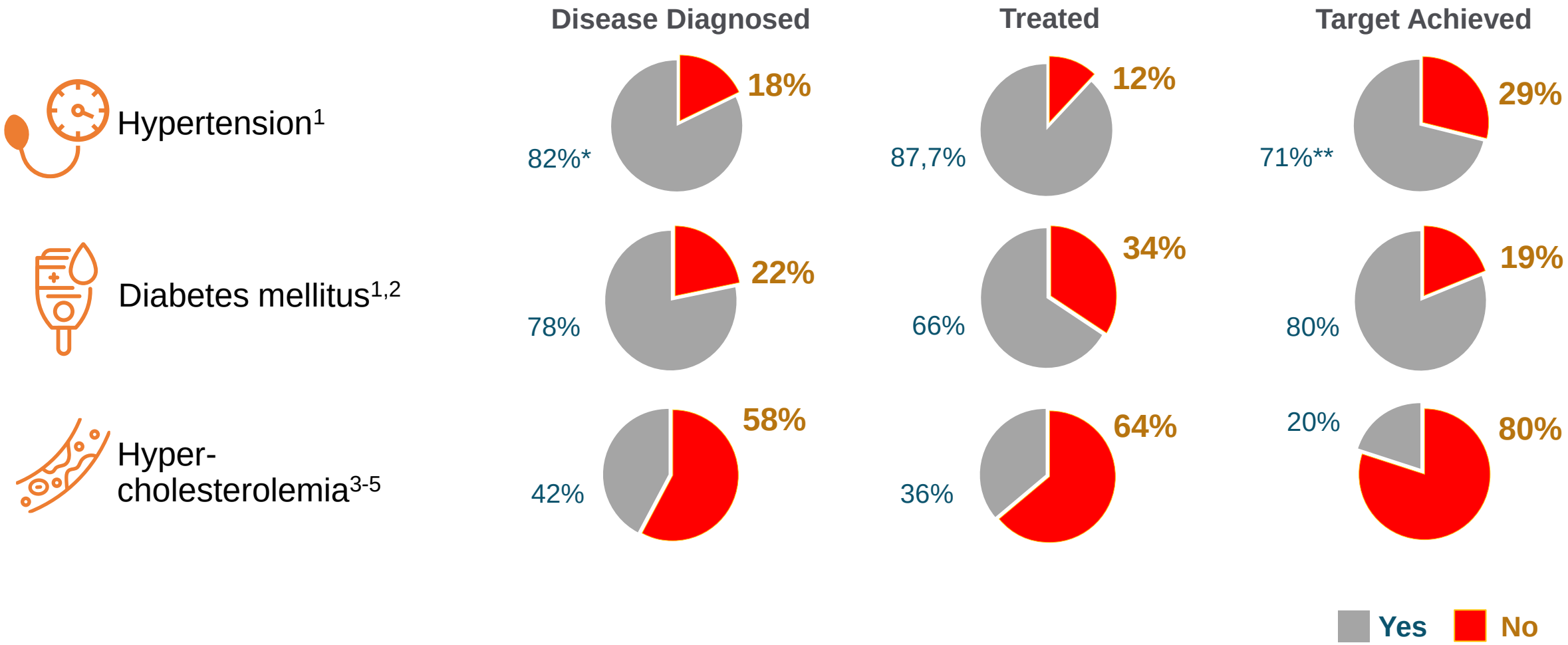
LDL-C target achievement in SANTORINI 2020-21



Casus ‘Daan van den Bersselaar’

Ray KK, Lancet Reg Health 2023

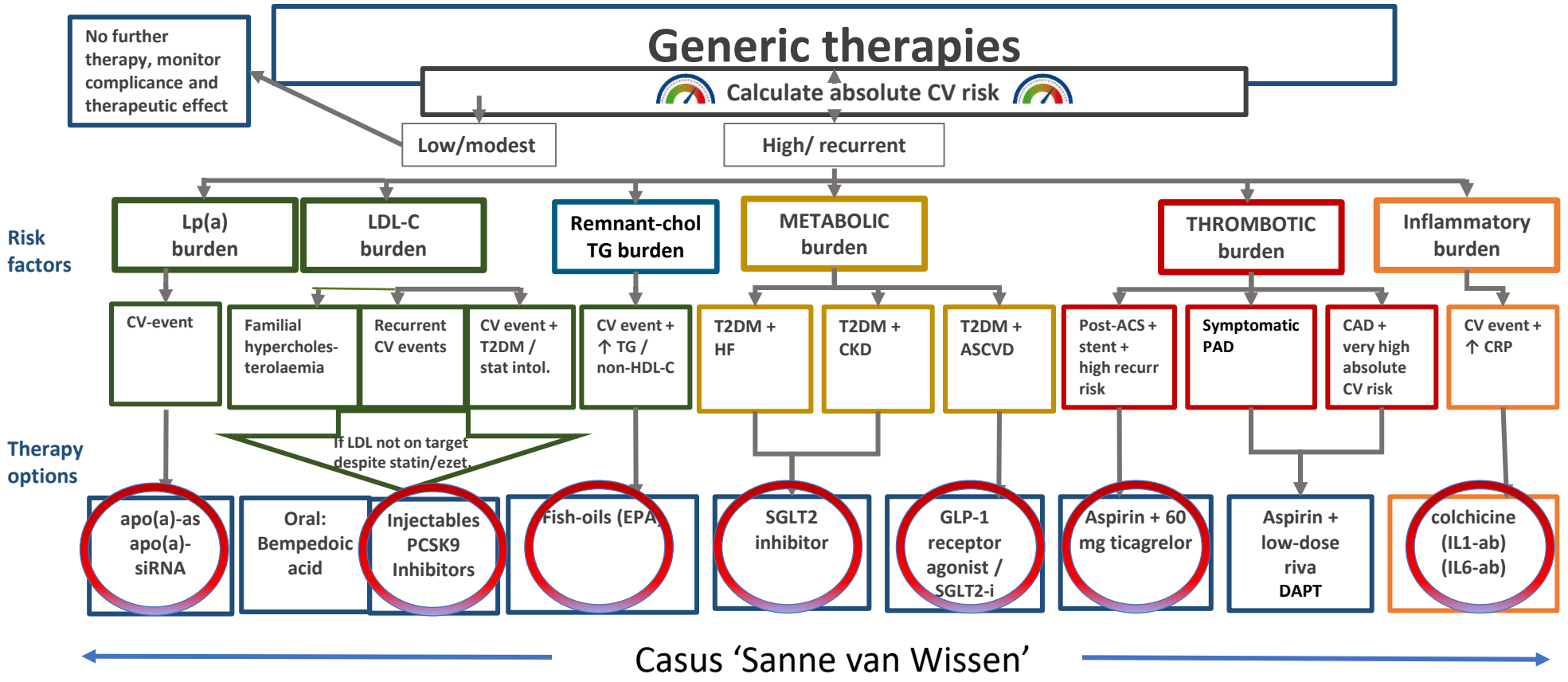
How are we performing on LDL-target achievement compared to other ‘risk’ domains?



**<140/90 mmHg percentage

1. Neuhauser HK, et al. *J Hum Hypertens*. 2015 **2.** Du Y, et al. *BMJ Open Diabetes Res Care*. 2015 **3.** Scheidt-Nave C, et al. *Bundesgesundheitsblatt* 2013.
4. März W, et al. *Atherosclerosis*. 2018. **5.** Fox KM, et al. *Clin Res Cardiol*. 2018.

In pursuit of residual CV-risk in secondary prevention patients:
Target and Treat all major factors contributing to CV-risk



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