

Kardiometabolische Prävention mit besonderem Blick auf Lipidmanagement und Hypertonie



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Disclosures

Mit freundlicher Unterstützung von:

Bezahlte Fortbildungen:

Böhringer Ingelheim, Cardiac Dimensions, Daiichi Sankyo, Amgen

Vortragstätigkeit:

Novartis, Kwizda, Boehringer Ingelheim, Bayer, Amgen

The logo for Amgen, consisting of the word "AMGEN" in a bold, blue, sans-serif font. The logo is centered within a white rectangular box.

Gliederung

- Lipidmanagement
- Art. Hypertonie



Gesamtcholesterin 5,18 mmol/L (200 mg/dL), LDL-C bei 3,68 mmol/L (142 mg/dL), HDL-C bei 1,17 mmol/L (45 mg/dL).

Reduzierte intestinale Cholesterinresorption: Der Mann nahm nur etwa 18 % des zugeführten Cholesterins tatsächlich auf – im Vergleich zu 46–55 % bei gesunden Vergleichspersonen unter cholesterinreicher bzw. -armer Diät.

Gesteigerte Gallensäuresynthese: Die Umwandlung von Cholesterin zu Gallensäuren war bei dem Mann gegenüber den Vergleichspersonen nahezu verdoppelt. Er synthetisierte täglich rund 1.513 μmol Gallensäuren, die Vergleichsprobanden bei cholesterinarmer Ernährung 766 μmol pro Tag und bei cholesterinreicher Ernährung 812 μmol pro Tag.

Kern F Jr N Engl J Med. 1991 Mar 28;324(13):896-9.

Machen wir alles richtig?

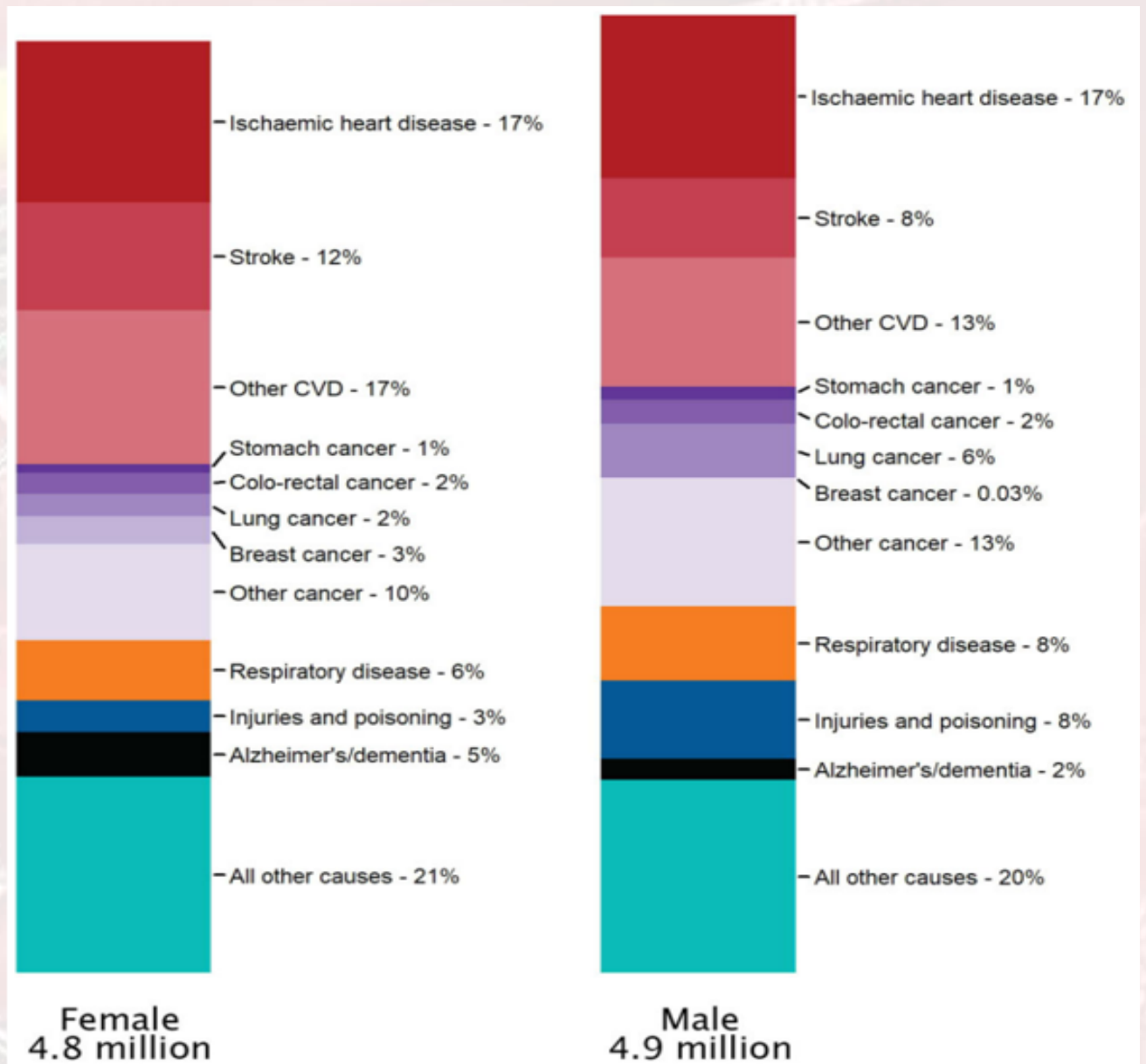


Quelle: Georg Parsché

30.09.2025

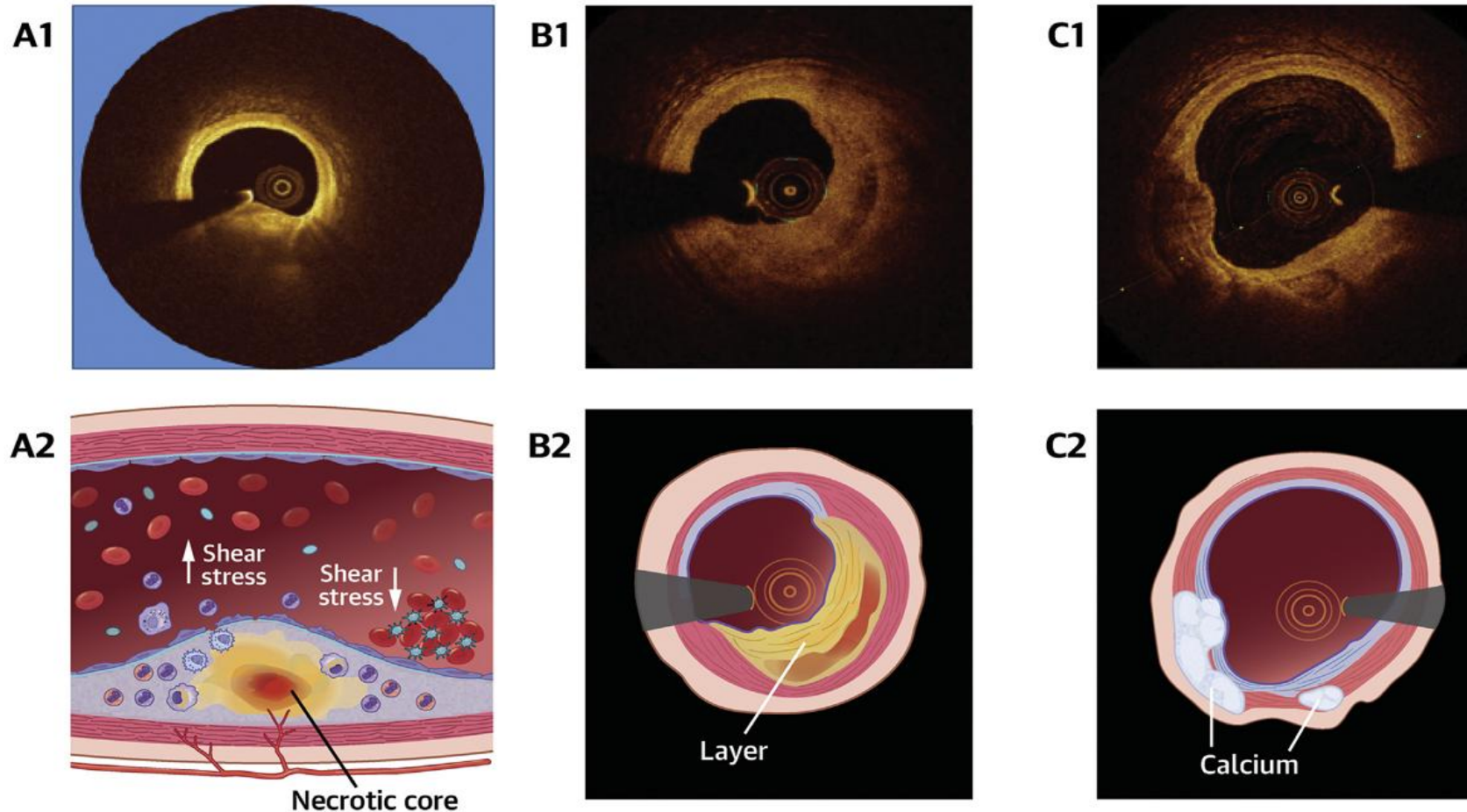
Kardiometabolische Prävention mit besonderem Blick auf
Lipidmanagement und Hypertonie

Todesursachen in Europa



Timmis A et al. Eur Heart J. 2022;Feb;04; PMID:35016208.

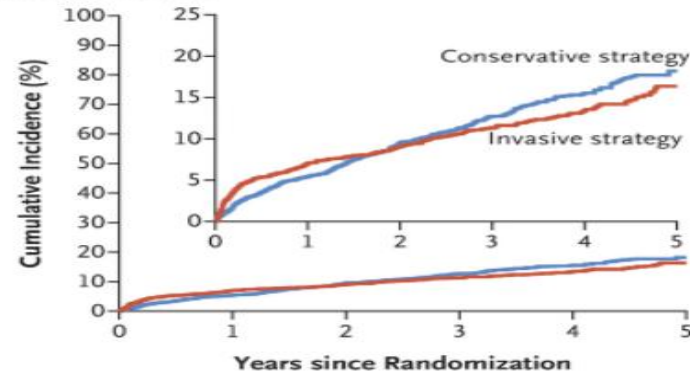
OCT- Plaquebogen/Fibröse Kappe



Adriaenssens, T. et al. J Am Coll Cardiol. 2021;78(12):1275-1287.

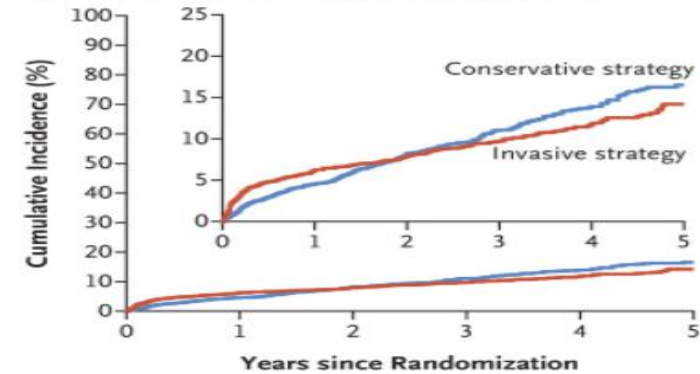
Ischemia-Studie

A Primary Composite Outcome



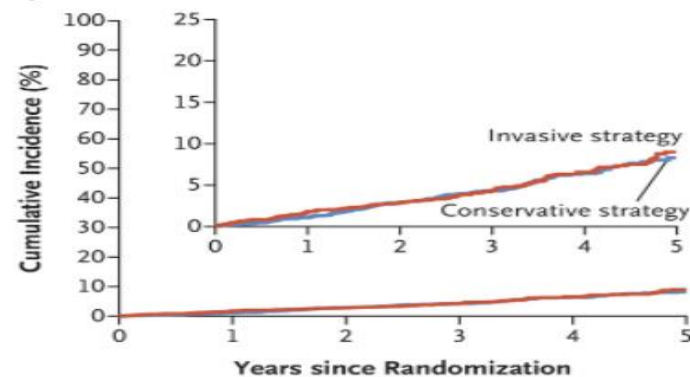
No. at Risk						
Conservative strategy	2591	2431	1907	1300	730	271
Invasive strategy	2588	2364	1908	1291	730	271

B Death from Cardiovascular Causes or Myocardial Infarction



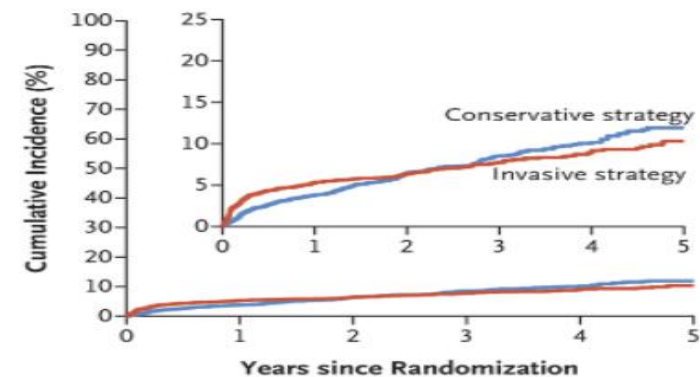
No. at Risk						
Conservative strategy	2591	2453	1933	1325	746	298
Invasive strategy	2588	2383	1933	1314	742	282

C Death from Any Cause



No. at Risk						
Conservative strategy	2591	2548	2065	1445	844	349
Invasive strategy	2588	2518	2061	1431	827	317

D Myocardial Infarction



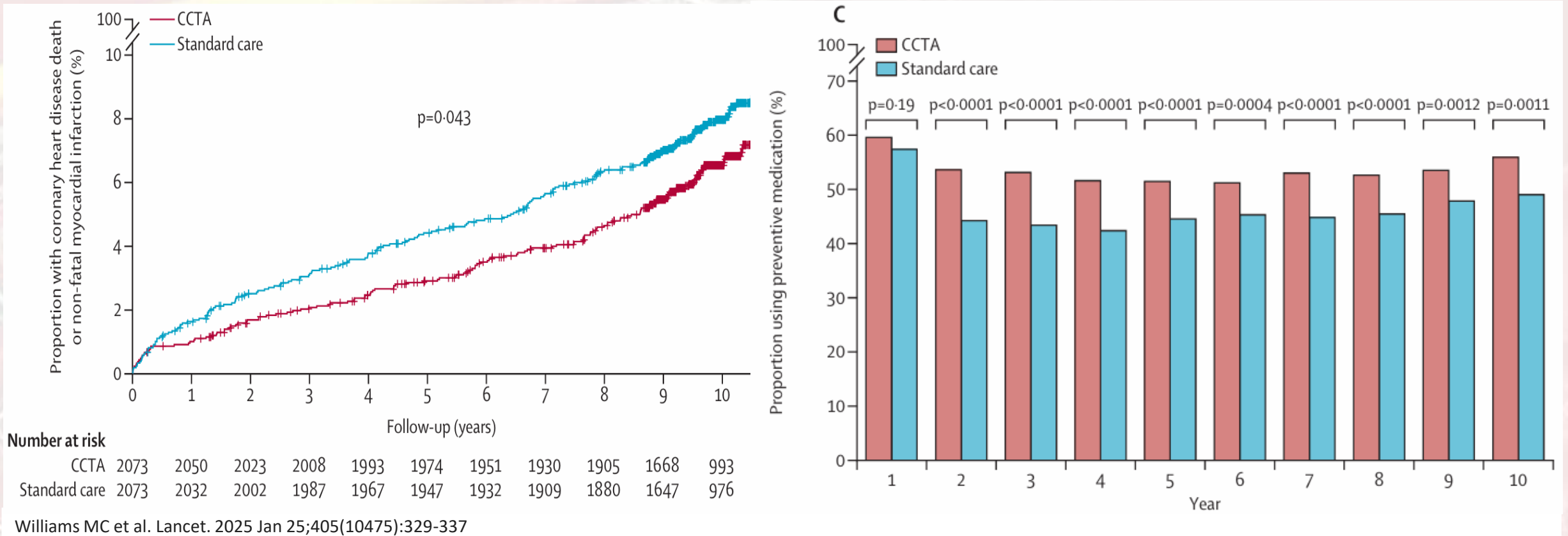
No. at Risk						
Conservative strategy	2591	2452	1931	1321	747	298
Invasive strategy	2588	2379	1931	1313	742	283

Maron DJ N Engl J Med. 2020 Apr 9;382(15):1395-1407.

Stellenwert der LDL-Senkung/ individuelles Ziel-LDL

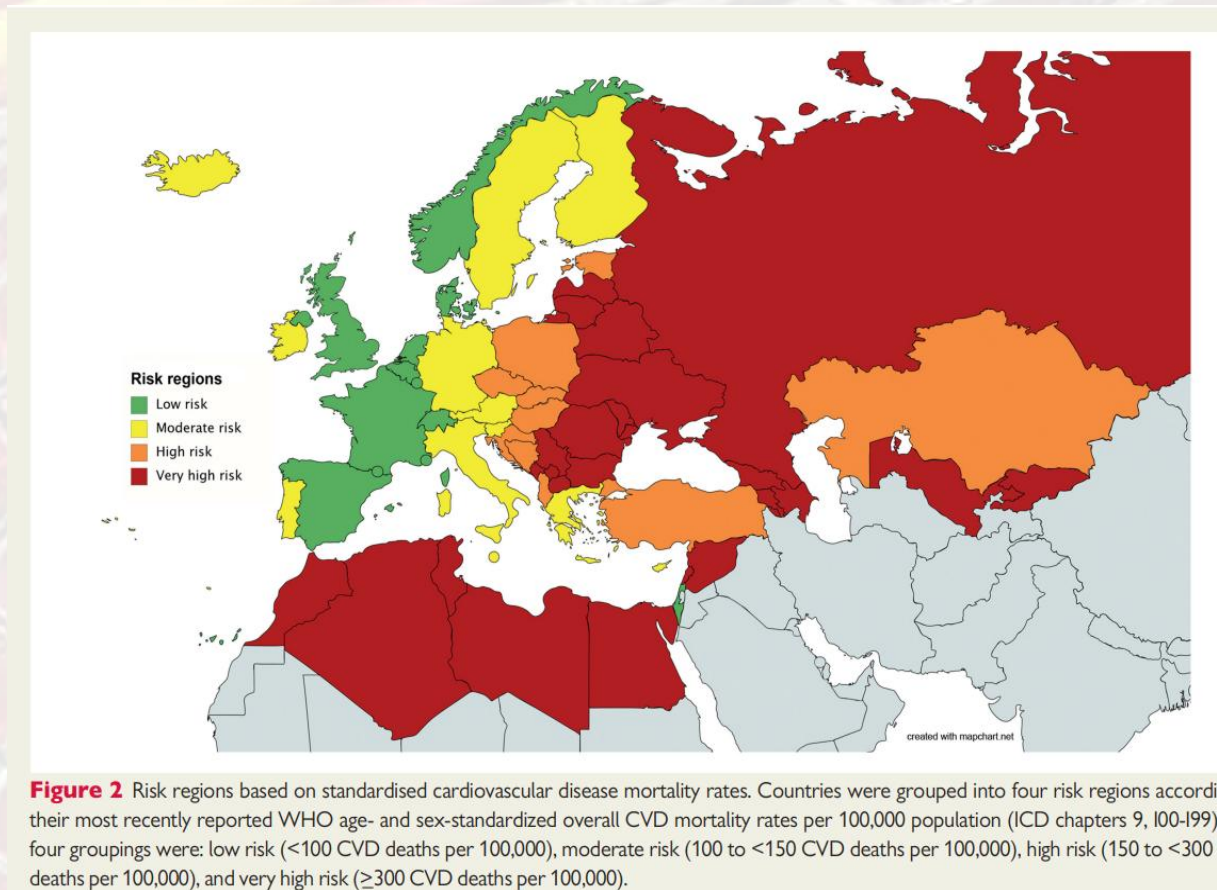
- Eine entscheidende Rolle spielt hierbei die **individuelle** Senkung des LDL-Cholesterins. Je nach Kollektiv Beginn und Ziel-LDL in Abhängigkeit von:
- SCORE 2, SCORE 2-Diabetes , SCORE 2 -DM
- zusätzl. RF: Stress, Bewegungsmangel, Adipositas, Familienanamn. etc.
- vorhandene ASCVD
- GFR, Proteinurie
- Mikrovaskulopathie (bei DM, Endorganschäden)

Coronar-CT reduziert Infarkte

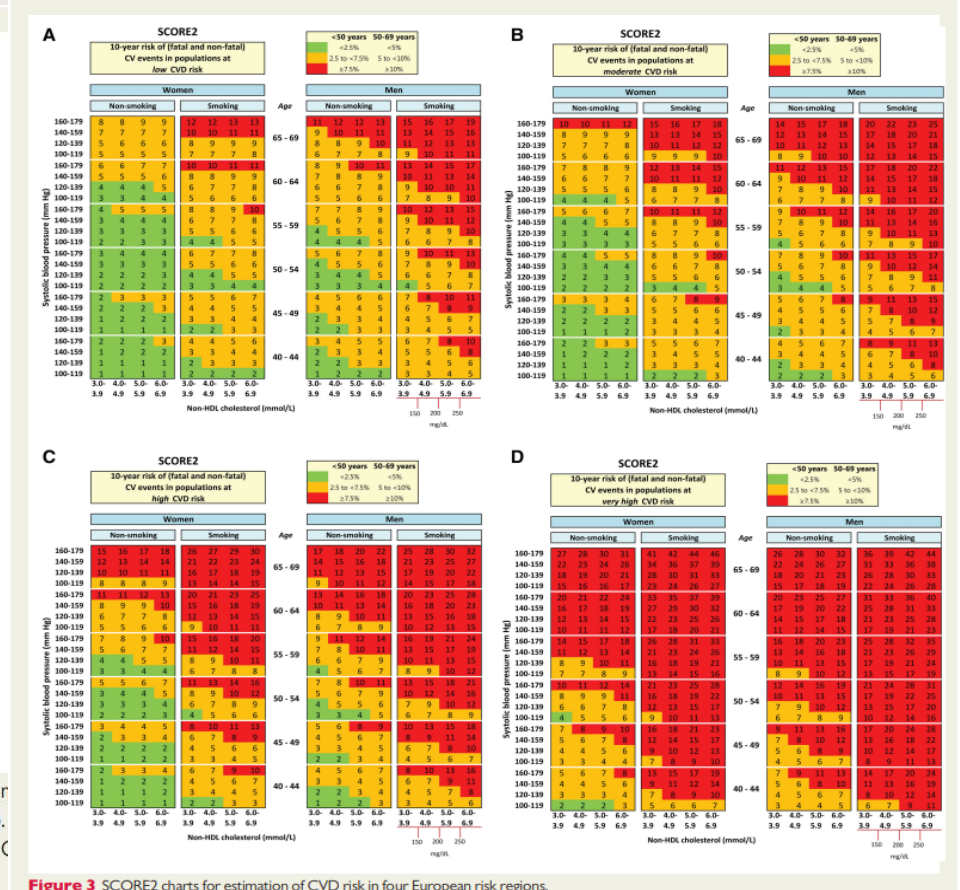


CT-Gruppe mit weniger Infarkten als ohne CT wegen besserer Prävention bei hämodynamisch nicht relevanter KHK, Revaskularisation in beiden Gruppen gleich (initial stabile AP und V.a. KHK)

Primärprävention bei vermeintlich Gesunden: SCORE 2* = Systematic Coronary Risk Evaluation



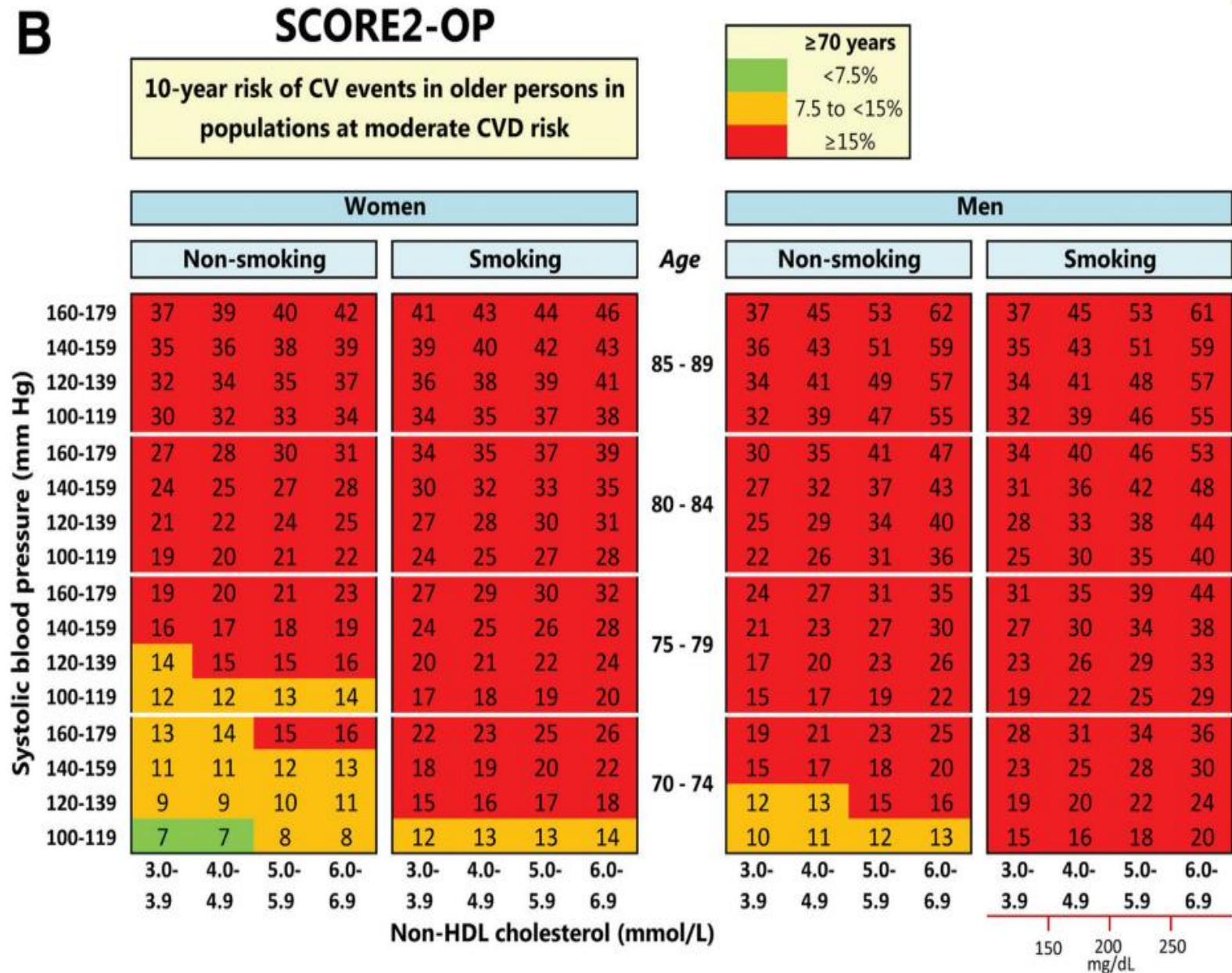
SCORE2 working group et al. Eur Heart J. 2021 Jul 1;42(25):2439-2454.



* ab einem Alter von 40 Jahren

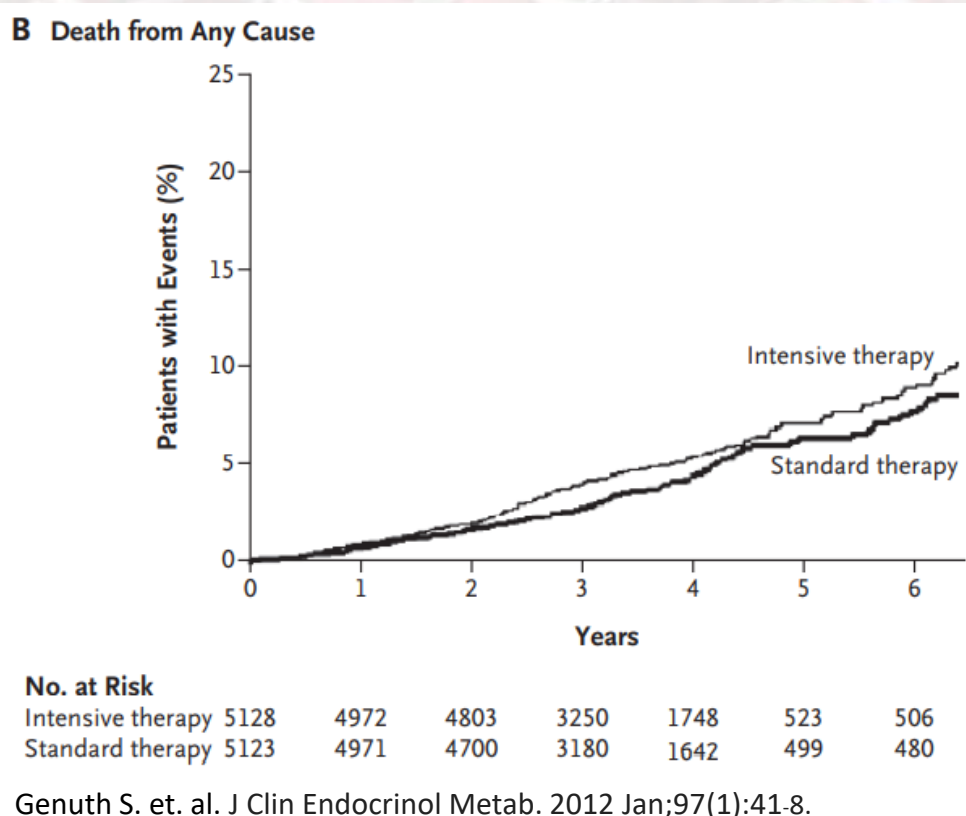
Non-HDL :
CHOL_{ges.} minus HDL-Chol.

Visseren FLJ et al. Eur Heart J. 2021 Sep 7;42(34):3227-3337.



Diabetes mellitus

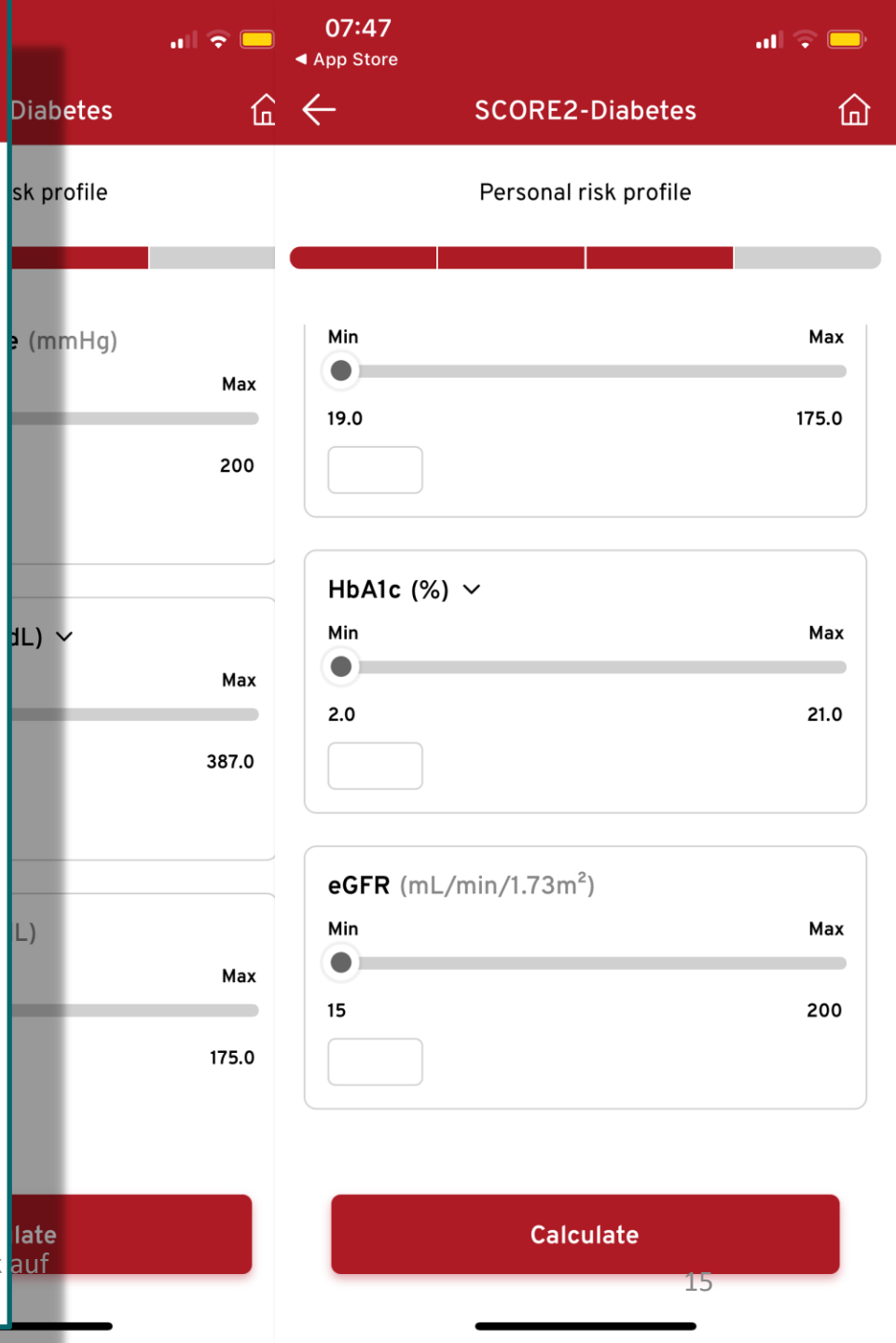
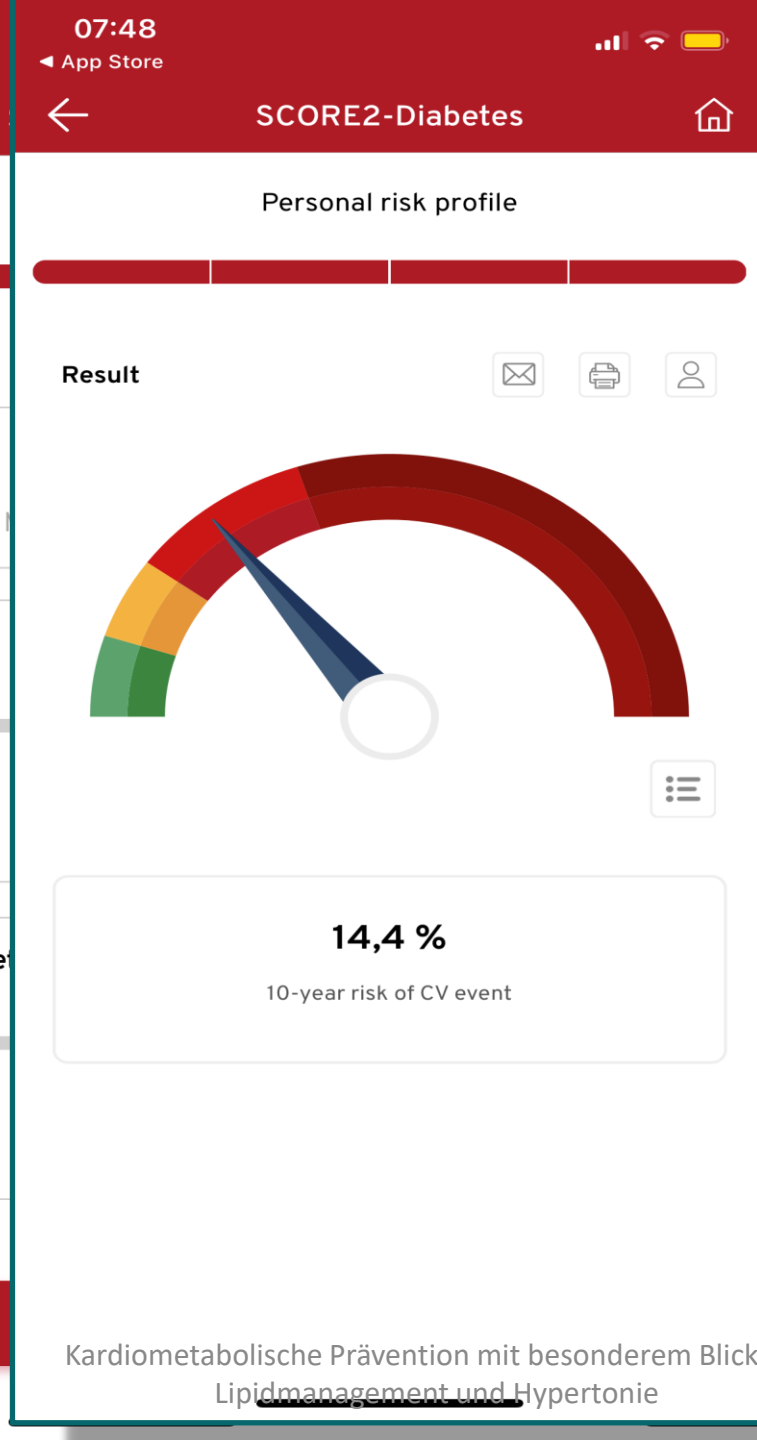
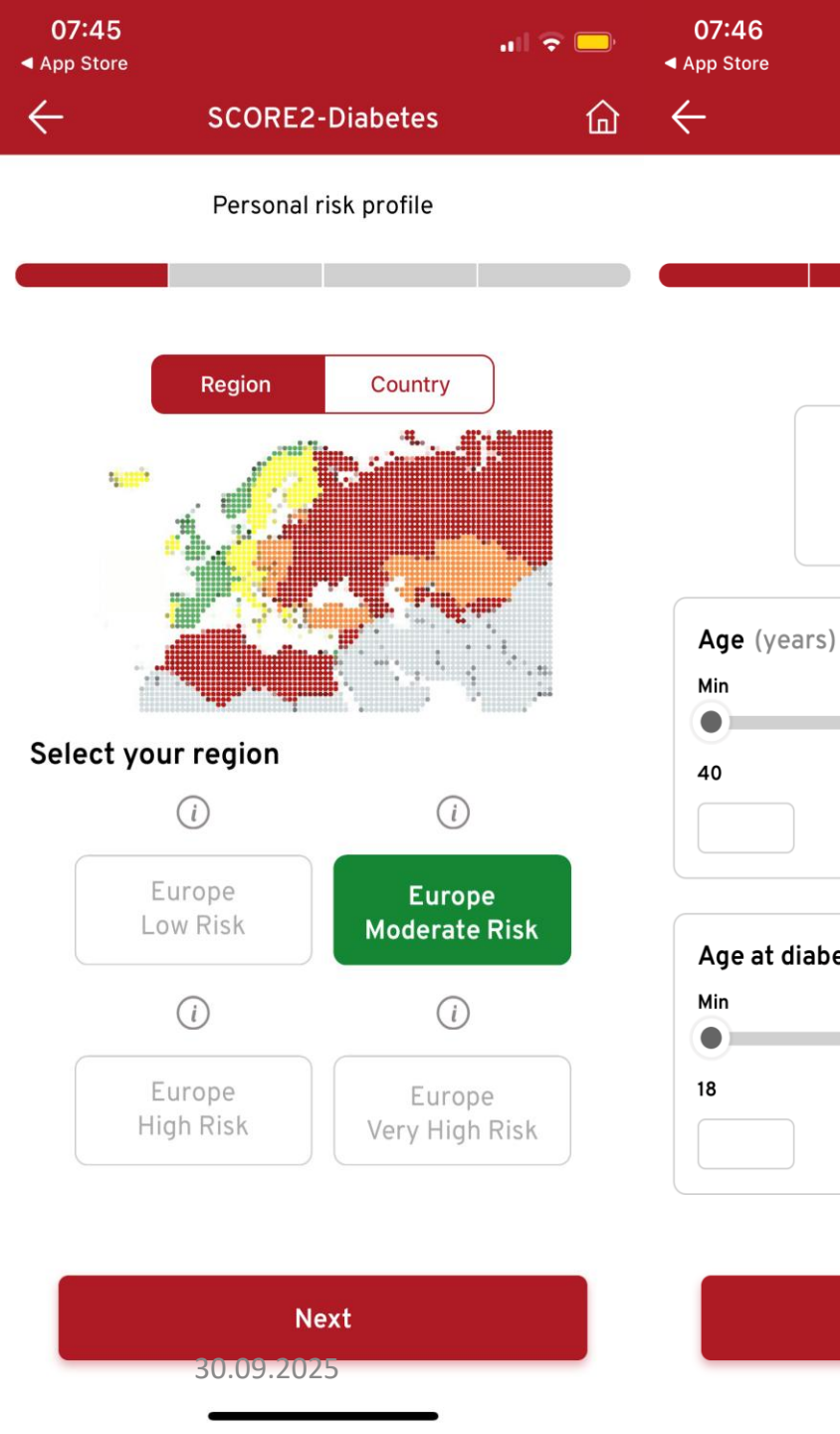
Accord-Studie



Studiendesign

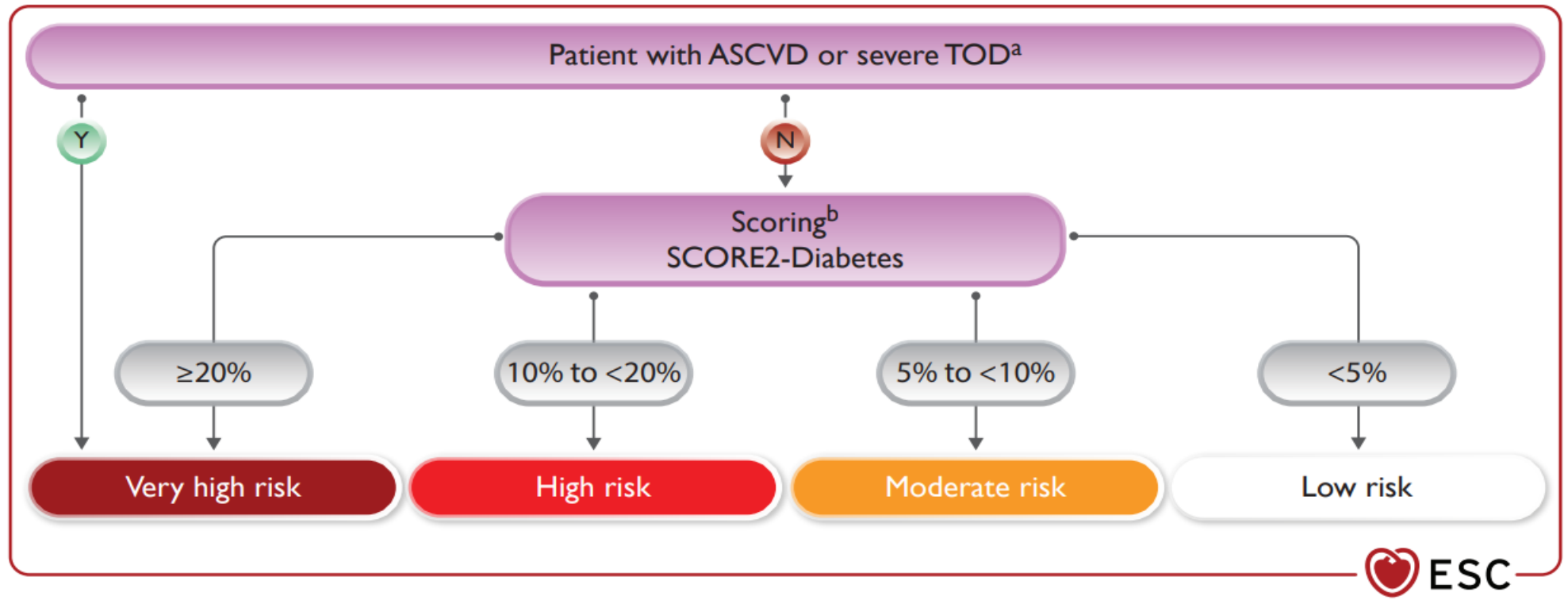
- Über 10000 Patienten
- Intensivierte Therapie: HbA1c <6%
- Standardtherapie: HbA1c 7,0-7,9%

-> wir sind mittlerweile von einer Behandlung des HbA1c zu einer suffizienten Organprotektion übergegangen.



Diabetes ohne ASCVD und Endorganschäden → SCORE 2 Diabetes*

Marx N et al. Eur Heart J. 2023 Aug 25;ehad192.



* 10-Jahres-Risiko für CVD in Abh. vom SCORE 2 + HbA1c, Alter bei Diagnose DM und der GFR (40- 69 Jahre)

Primär

Box 1 Risk modifiers for consideration beyond the risk estimation based on the SCORE2 and SCORE2-OP algorithms

DM– Ziel-

Demographic/clinical conditions

- Family history of premature CVD (men: <55 years; women: <60 years)
- High-risk ethnicity (e.g. Southern Asian)
- Stress symptoms and psychosocial stressors
- Social deprivation
- Obesity
- Physical inactivity
- Chronic immune-mediated/inflammatory disorders
- Major psychiatric disorders
- History of premature menopause
- Pre-eclampsia or other hypertensive disorders of pregnancy
- Human immunodeficiency virus infection
- Obstructive sleep apnoea syndrome

Biomarkers

- Persistently elevated hs-CRP (>2 mg/L)
- Elevated Lp(a) [>50 mg/dL (>105 nmol/L)].

Risikopr

Geringes Risiko

Mittleres Risiko

Hohes Risiko(I)

Sehr hohes Risiko

Sekundärpräven

Extremes Risiko

Ziel-LDL

5 mg/dl

0 mg/dl

mg/dl + 50% v. BL

mg/dl + 50% v. BL

mg/dl + 50% v. BL

mg/dl

Mach F et al. Eur Heart J. 2020 Jan 1;41(1):1-10.

Mach F et al. Eur Heart J. 2025 Aug 29:e

Risikoeinteilung

Very high risk

People with any of the following:

- Documented ASCVD, either clinical or unequivocal on imaging. Documented ASCVD includes previous ACS (MI or unstable angina), chronic coronary syndromes, coronary revascularization (PCI, CABG, and other arterial revascularization procedures), stroke and TIA, and peripheral arterial disease. Unequivocally documented ASCVD on imaging includes those findings that are known to be predictive of clinical events, such as significant plaque^a on coronary angiography or CT scan or on carotid or femoral ultrasound or markedly elevated CAC score by CT^b
- DM with target organ damage,^c or at least three major risk factors, or early onset of T1DM of long duration (>20 years)
- Severe CKD (eGFR <30 mL/min/1.73 m²)
- A calculated SCORE2 or SCORE2-OP $\geq 20\%$ for 10 year risk of fatal or non-fatal CVD
- FH with ASCVD or with another major risk factor

High risk

People with any of the following:

- Markedly elevated single risk factors, in particular **TC >8 mmol/L (>310 mg/dL), LDL-C >4.9 mmol/L (>190 mg/dL)** or BP $\geq 180/110$ mmHg
- Patients with FH without other major risk factors
- Patients with DM without target organ damage,^c with DM duration ≥ 10 years or another additional risk factor
- Moderate CKD (eGFR 30–59 mL/min/1.73 m²)
- A calculated SCORE2 or SCORE2-OP $\geq 10\%$ and <20% for 10 year risk of fatal or non-fatal CVD

Moderate risk

People with any of the following:

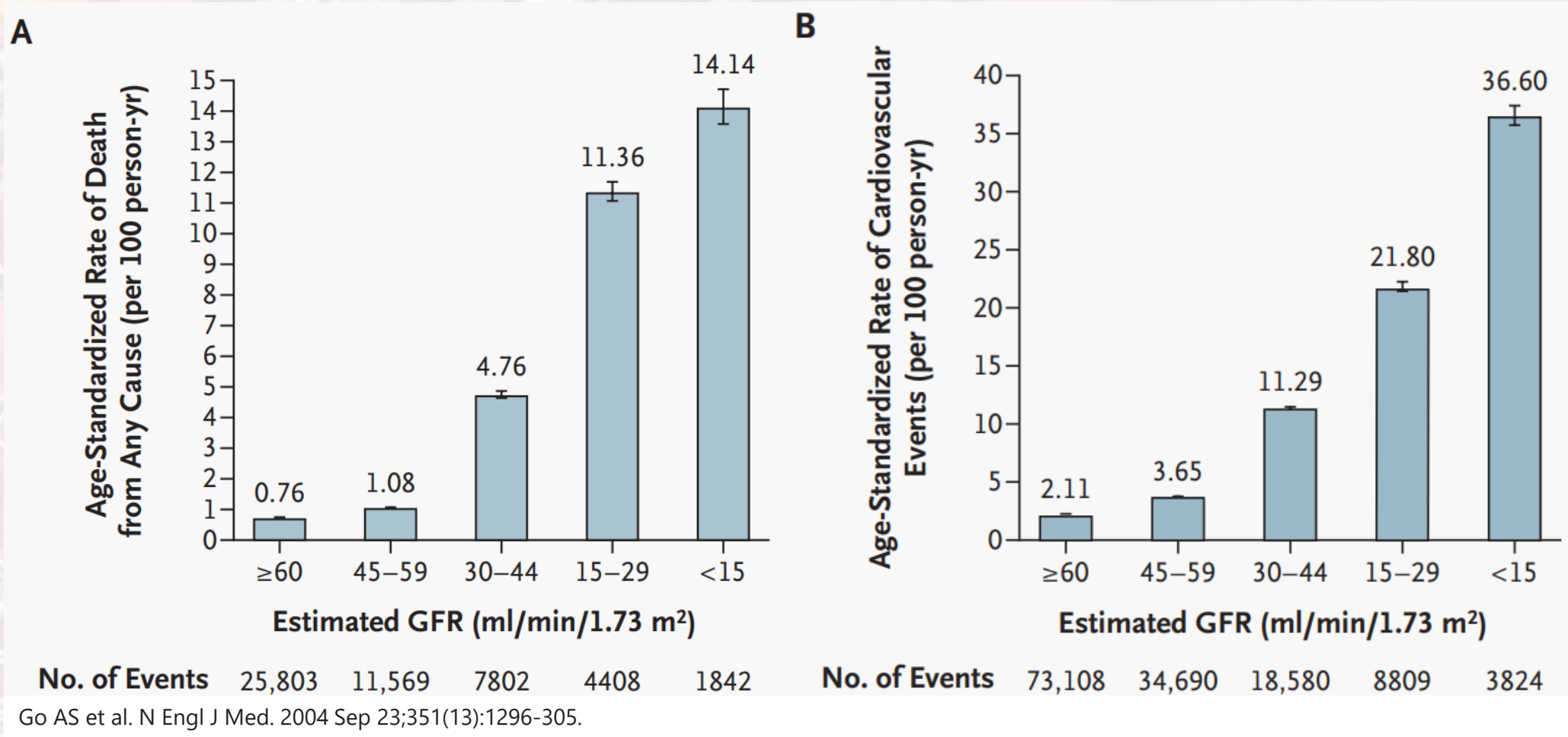
- Young patients (T1DM <35 years; T2DM <50 years) with DM duration <10 years, without other risk factors
- Calculated SCORE2 or SCORE2-OP $\geq 2\%$ and <10% for 10 year risk of fatal or non-fatal CVD

Low risk

- Calculated SCORE2 or SCORE2-OP <2% for 10 year risk of fatal or non-fatal CVD

Mach F et al. Eur Heart J. 2025 Aug 29;ehaf190. doi: 10.1093/eurheartj/ehaf190. Epub ahead of print. PMID: 40878289.

Niereninsuffizienz: ein unabhängiger CV-Risikofaktor



UACR als früher Marker für CKD



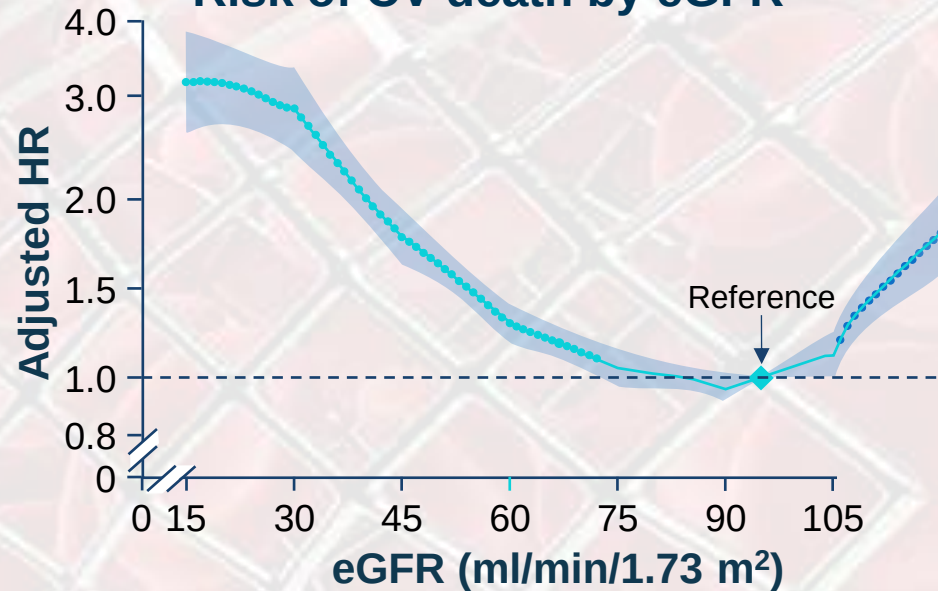
CKD = eGFR <60 ml/min/1.73 m² for >3 months¹

and
OR

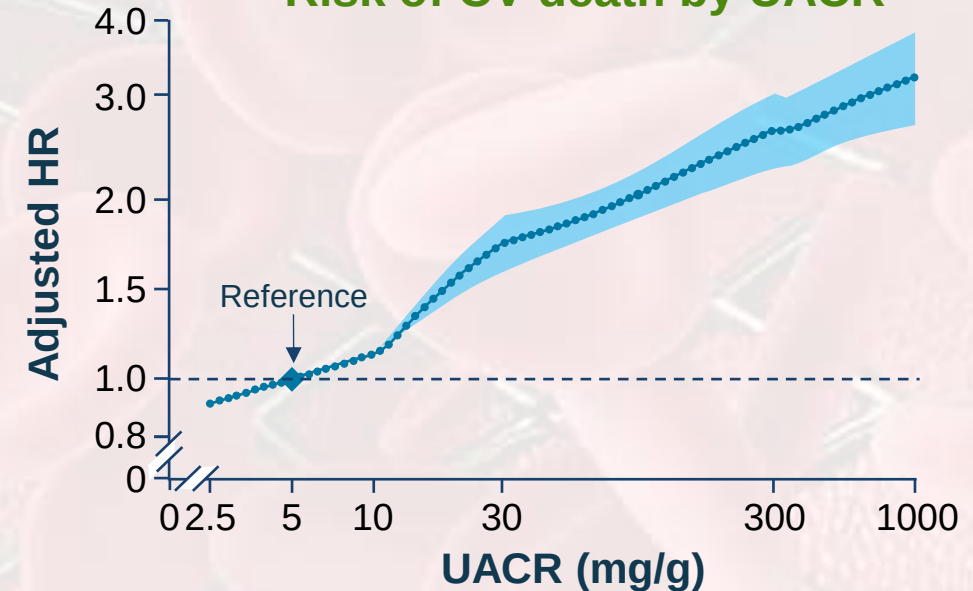


CKD = albuminuria
UACR >30 mg/g for >3 months¹

Risk of CV death by eGFR*²



Risk of CV death by UACR*²



Adapted from Matsushita K, *et al.* 2015

*Adjusted for age, sex, race or ethnic origin, smoking, SBP, antihypertensive drugs, diabetes, total and HDL cholesterol concentrations, and albuminuria (UACR or dipstick) or eGFR, as appropriate; dots represent statistical significance (p<0.05); all axes are log scales except for the eGFR axis

CKD=Chronic kidney disease; CV=Cardiovascular; eGFR=estimated glomerular filtration rate; HR=hazard ratio; SBP=systolic blood pressure; T2D=type-2-diabetes; UACR =Urine albumin-to-creatinine ratio

KDIGO-Klassifikation

Grams ME et al. JAMA. 2023 Oct 3;330(13):1266-1277.

CKD is classified based on: • Cause (C) • GFR (G) • Albuminuria (A)				Albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–299 mg/g 3–29 mg/mmol	≥300 mg/g ≥30 mg/mmol
GFR categories (ml/min/1.73 m²) Description and range	G1	Normal or high	≥90	Screen 1	Treat 1	Treat 3
	G2	Mildly decreased	60–89	Screen 1	Treat 1	Treat 3
	G3a	Mildly to moderately decreased	45–59	Treat 1	Treat 2	Treat 3
	G3b	Moderately to severely decreased	30–44	Treat 2	Treat 3	Treat 3
	G4	Severely decreased	15–29	Treat* 3	Treat* 3	Treat 4+
	G5	Kidney failure	<15	Treat 4+	Treat 4+	Treat 4+

Low risk (if no other markers of kidney disease, no CKD)
High risk

Moderately increased risk
Very high risk

CKD: Ziel-LDL in Abh. von GFR und UACR ohne DM

	A1 (AKR: <30 mg/g)	A2 (AKR30-300mg/g)	A3 (AKR300-3000 mg/g)
G1 >90 ml/min	Score 2	Score 2	
G2 60 – 90 ml/min	Score 2	Score 2	
G3a 45-59 ml/min			
G3b 30-44 ml/min			
G4 15-29 ml/min			
G5 <15 ml/min			

Nach Visseren FLJ et al. . Eur Heart J. 2021 Sep 7;42(34):3227-3337 und Mach F et al. Eur Heart J. 2020 Jan 1;41(1):111-188.

rot = hohes Risiko (Ziel-LDL < 70 mg/dl); hellrot = sehr hohes Risiko (Ziel-LDL < 55 mg/dl)

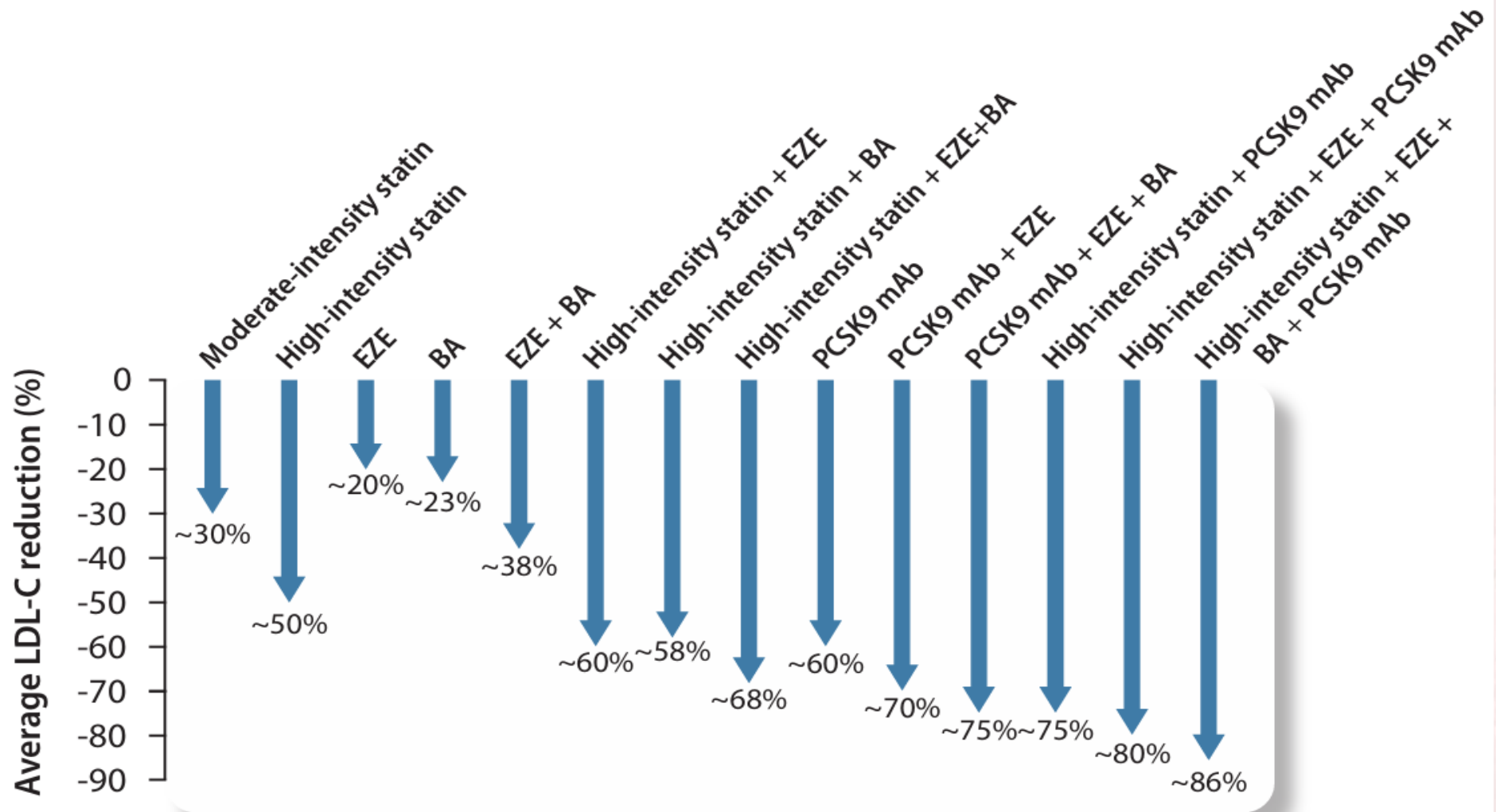
Ziel LDL bei CKD +in Abh. v. der UACR und der GFR bei DM

	A1 (AKR: <30 mg/g)	A2 (30-300mg/g)	A3 (300-3000 mg/g)	Endorgansch.
G1 >90 ml/min	Score 2-DM	Score 2-DM		
G2 60 – 90 ml/min	Score 2-DM	Score 2-DM		
G3a 45-59 ml/min				
G3b 30-44 ml/min				
G4 15-29 ml/min				
G5 <15 ml/min				

Nach Visseren FLJ et al. . Eur Heart J. 2021 Sep 7;42(34):3227-3337 und Mach F et al. Eur Heart J. 2020 Jan 1;41(1):111-188.

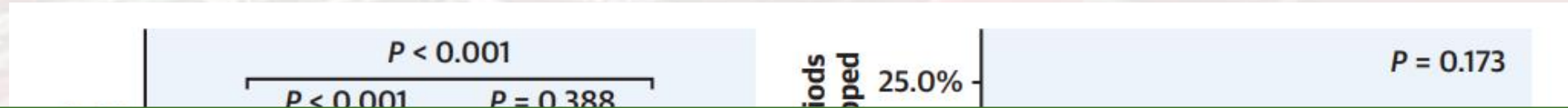
rot = hohes Risiko (Ziel-LDL < 70 mg/dl); hellrot = sehr hohes Risiko (Ziel-LDL < 55 mg/dl)

LDL- senkende Therapie



Mach F et al. Eur Heart J. 2025 Aug 29;ehaf190. doi: 10.1093/eurheartj/ehaf190. Epub ahead of print. PMID: 40878289.

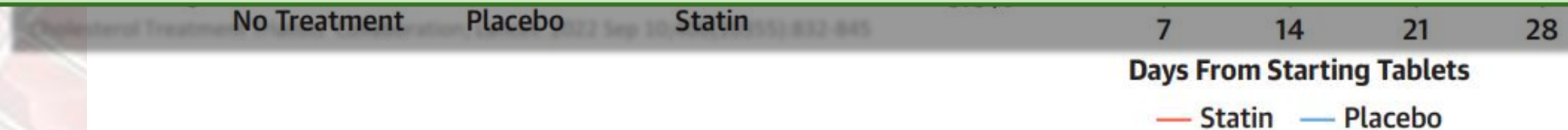
Ist eine Statinmyopathie häufig?



9% der PatientInnen unter Statintherapie klagen über Muskelschmerzen

Bei 90% dieser PatientInnen sind diese Schmerzen nicht Statin-assoziiert

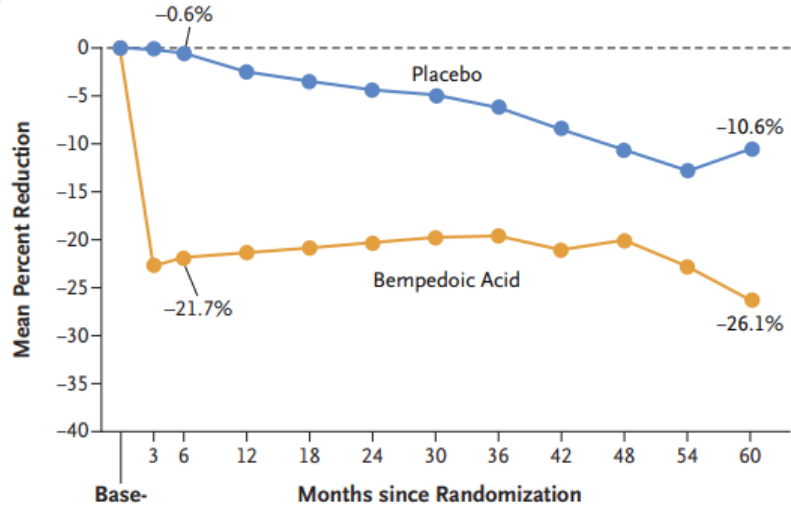
Cholesterol Treatment Trialists' Collaboration; Lancet. 2022 Sep 10;400(10355):832-845



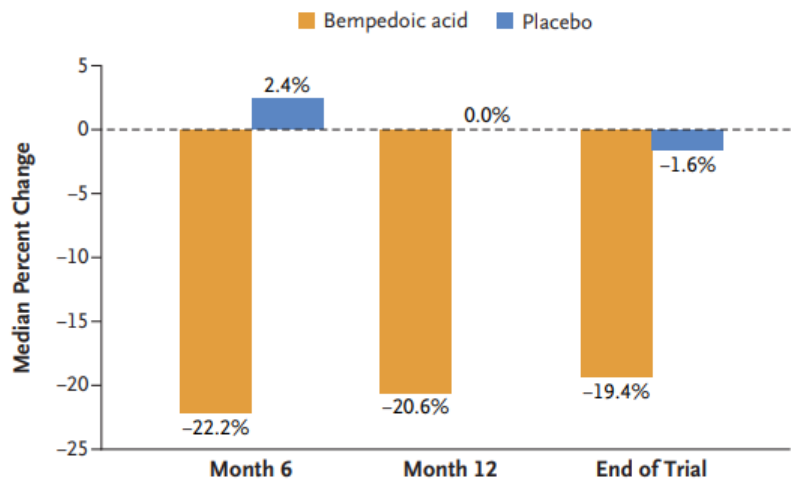
Howard, J.P. et al. J Am Coll Cardiol. 2021;78(12):1210-1222.

Bempedoinsäure-Clear-Studie

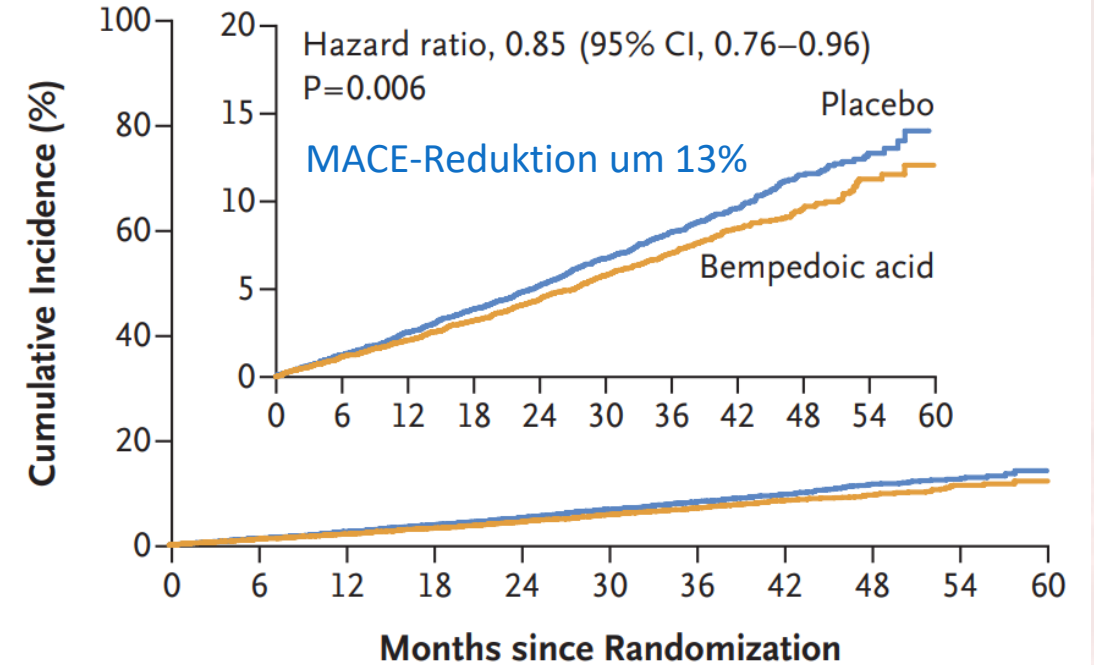
Nissen SE et al. N Engl J Med. 2023 Apr 13;388(15):1353-1364.



High-Sensitivity CRP Level



B Three-Component MACE

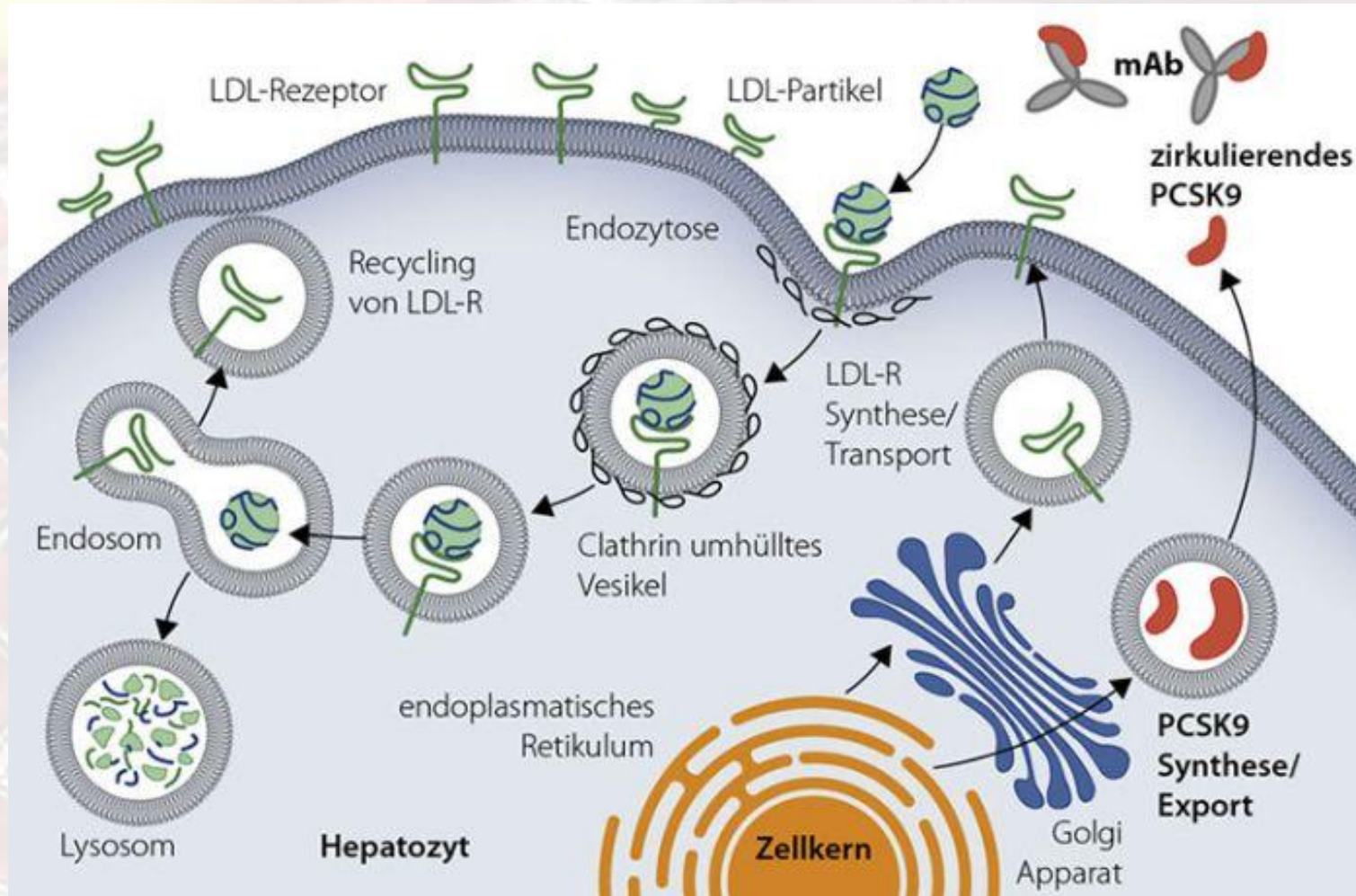


No. at Risk

Placebo	6978	6828	6883	6536	6368	6193	5321	2649	1279	554	62
Bempedoic acid	6992	6859	6745	6604	6457	6298	5453	2724	1317	591	80

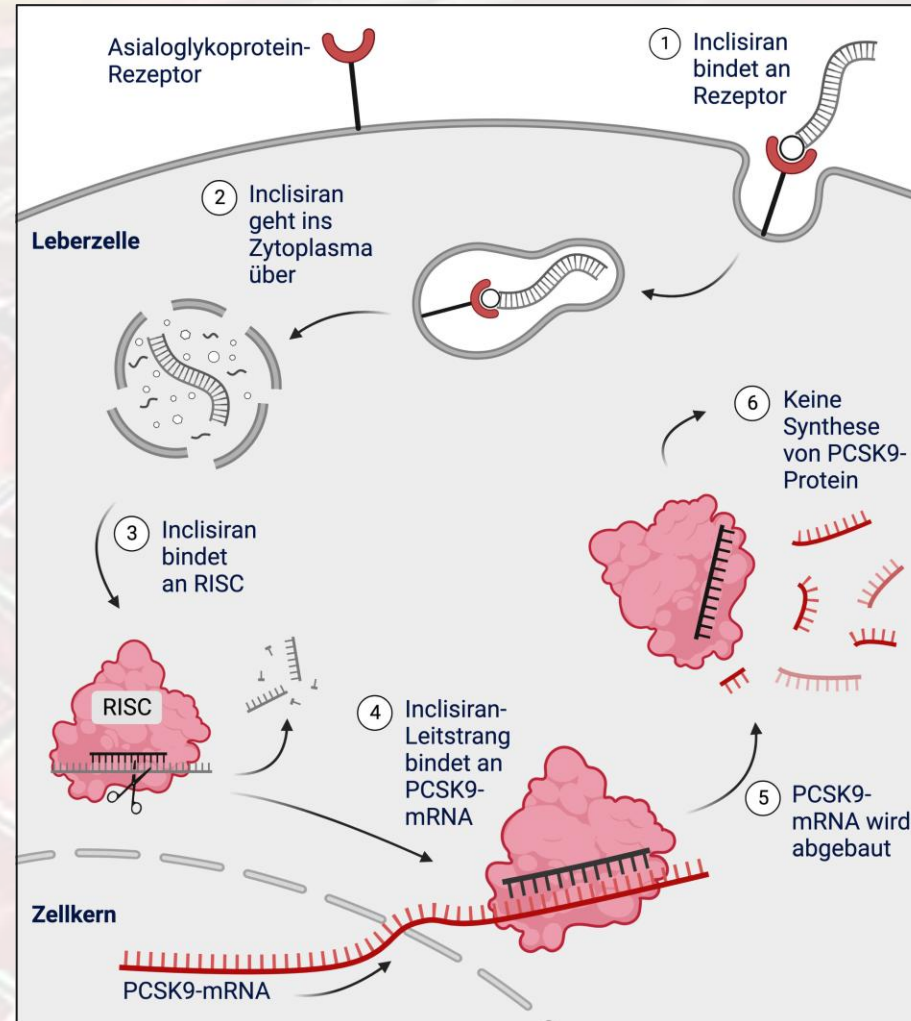
Nissen SE et al. N Engl J Med. 2023 Apr 13;388(15):1353-1364.

PCSK 9-Inhibitoren (Evolocuman, Alirocumab) bzw. siRNA (Inclisiran)



Quelle: www.cmpus.sanofi
Quelle: doccheck.com

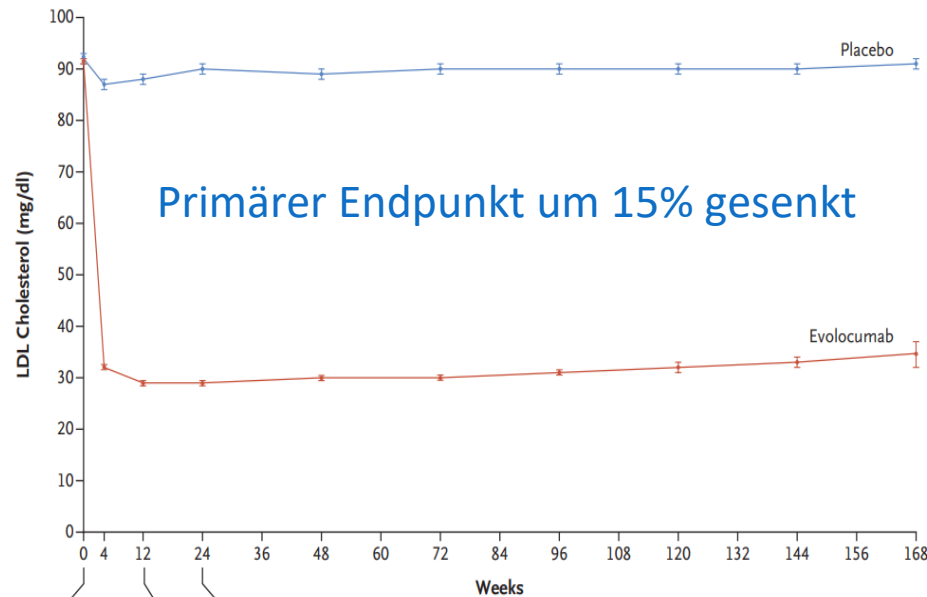
PCSK 9-Inhibitoren: siRNA (Inclisiran)



Quelle: www.cmpus.sanofi

PCSK 9-Fourier (Evolocumab)

Verabreichung alle 2-4 Wochen s. c.

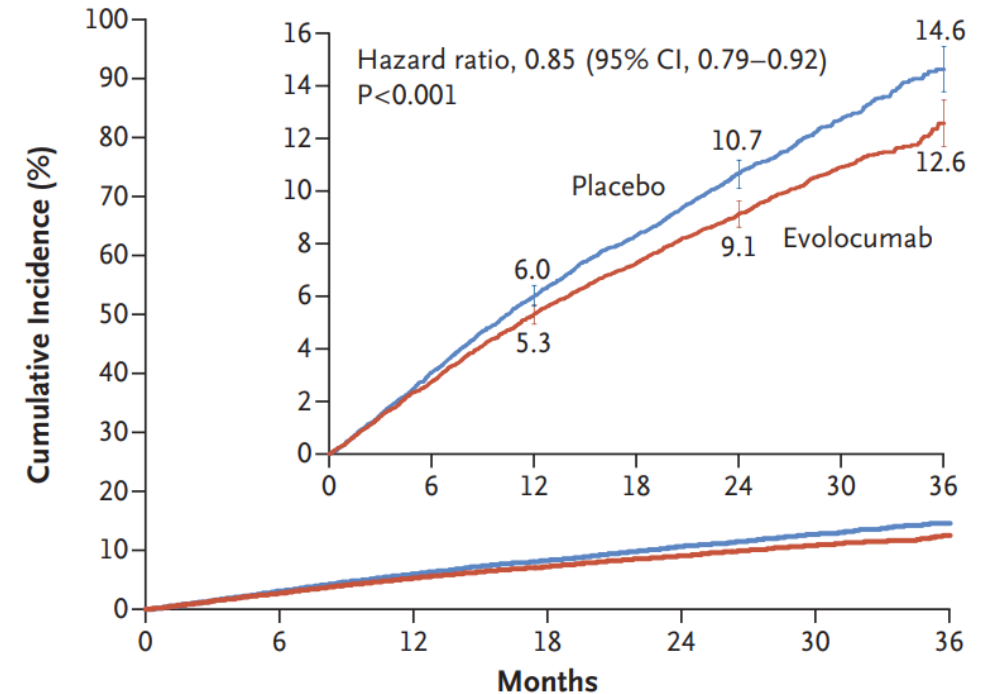


No. at Risk	0	4	12	24	36	48	60	72	84	96	108	120	132	144	156	168
Placebo	13,779	13,251	13,151	12,954	12,596	12,311	10,812	6926	3352	790						
Evolocumab	13,784	13,288	13,144	12,964	12,645	12,359	10,902	6958	3323	768						

Absolute difference (mg/dl)	54	58	57	56	55	54	52	53	50
Percentage difference	57	61	61	59	58	57	55	56	54
P value	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

Giugliano RP et al. Lancet. 2017 Oct 28;390(10106):1962-1971.

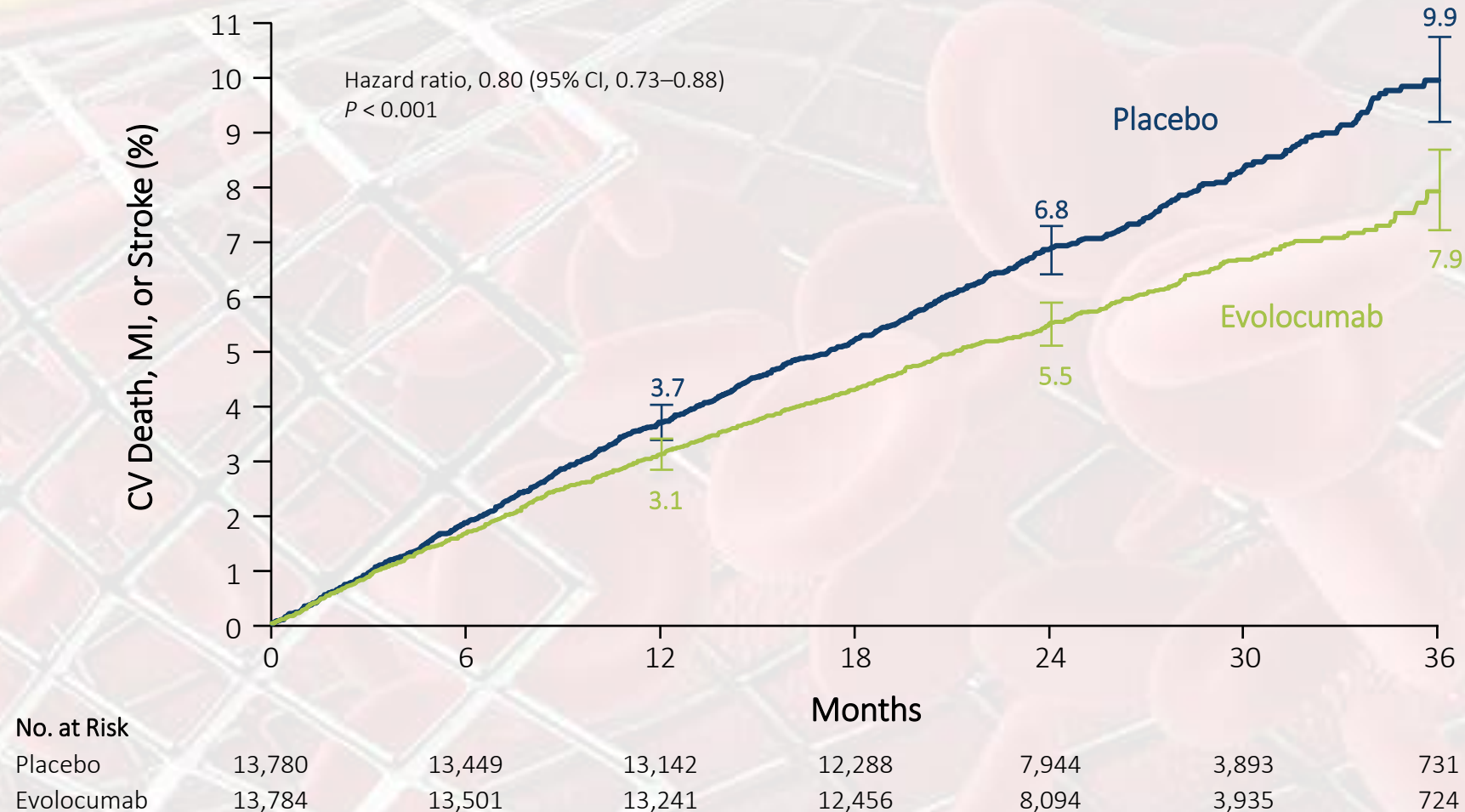
A Primary Efficacy End Point (CV-Tod, MI, Insult, Hosp., Revasc.)



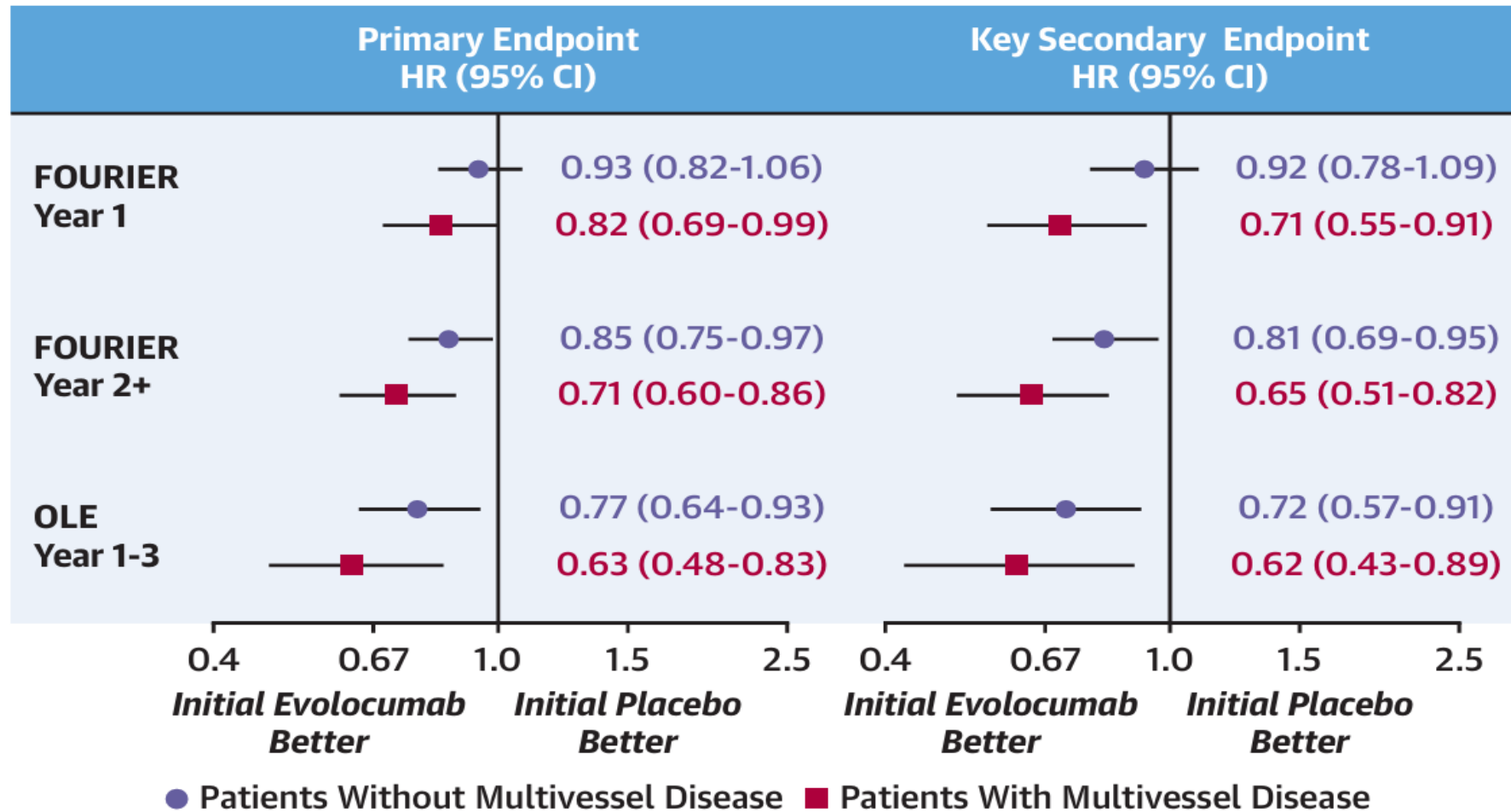
No. at Risk	0	6	12	18	24	30	36
Placebo	13,780	13,278	12,825	11,871	7610	3690	686
Evolocumab	13,784	13,351	12,939	12,070	7771	3746	689



Evolocumab Reduced 3-Point MACE by 20% vs Placebo



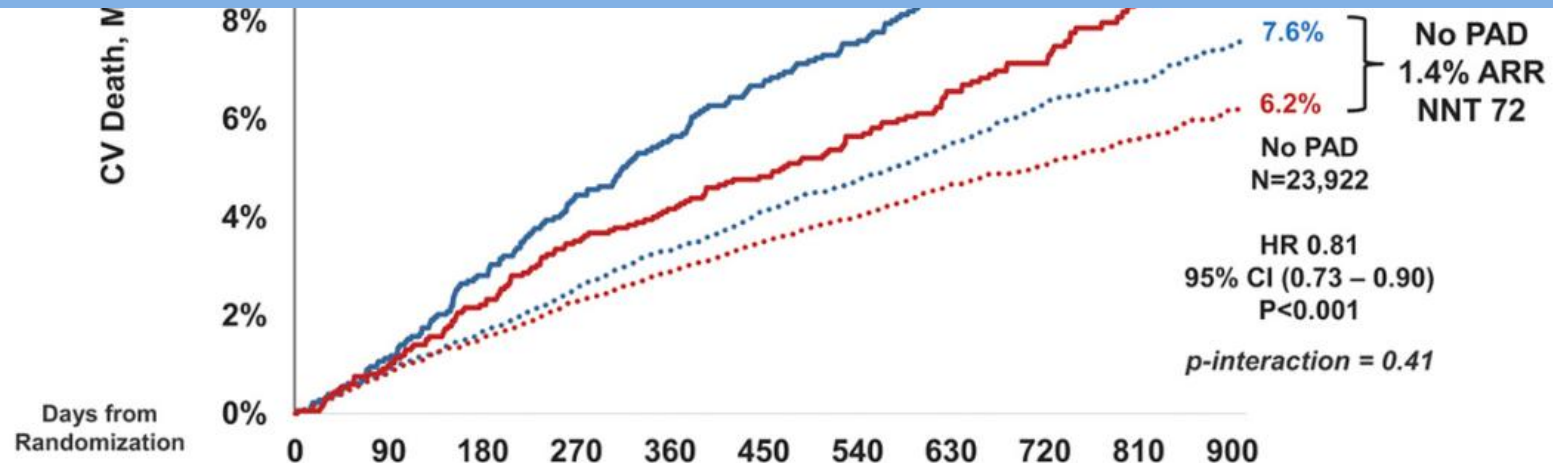
Evolocumab-Langzeitdaten



McClintick DJ, et al. J Am Coll Cardiol. 2024;83(6):652-664.

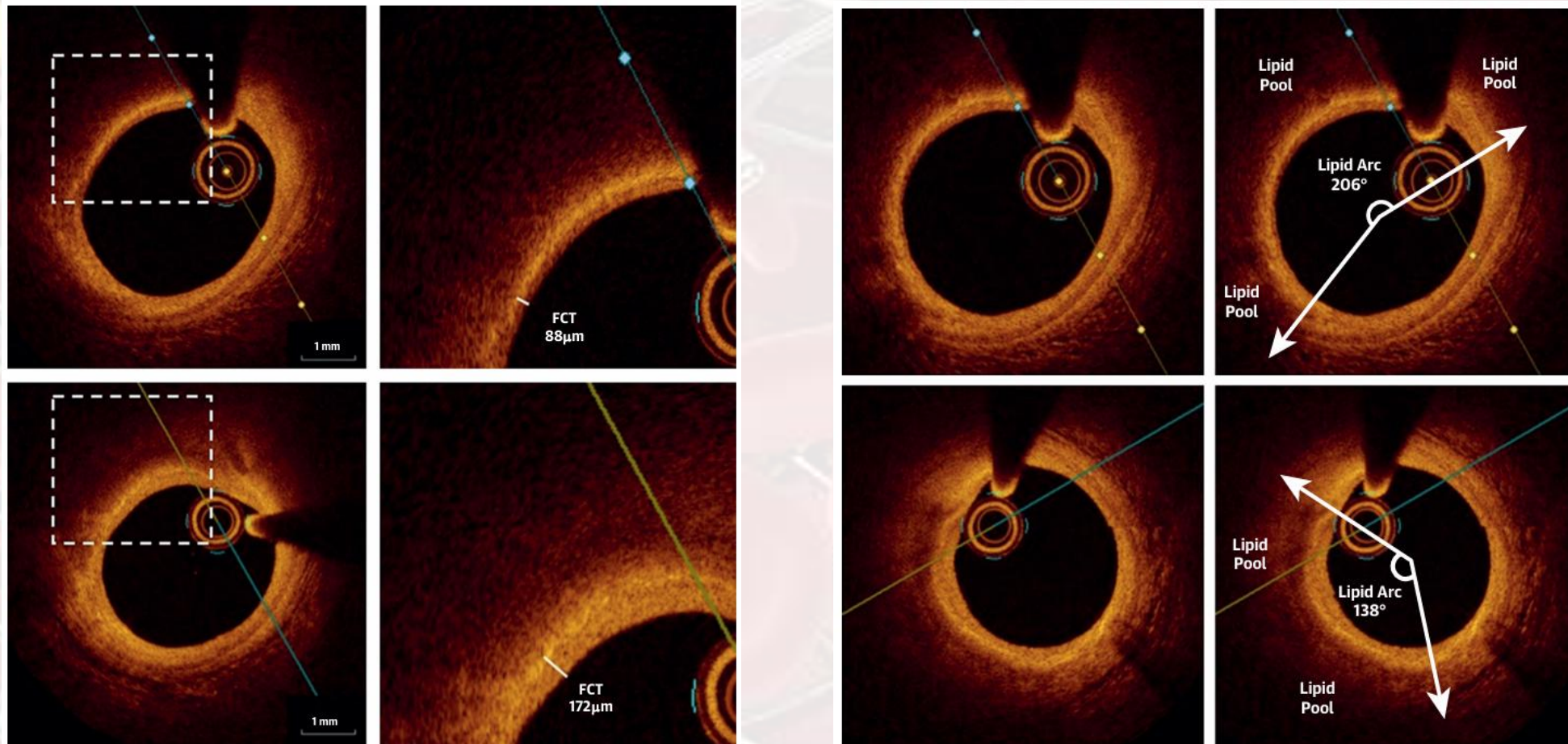
Evolucomab: MACE mit und ohne PAVK

Evolocumab senkt 3-P-MACE bei PAVK ohne MI oder Insult in der Anamnese um 43%



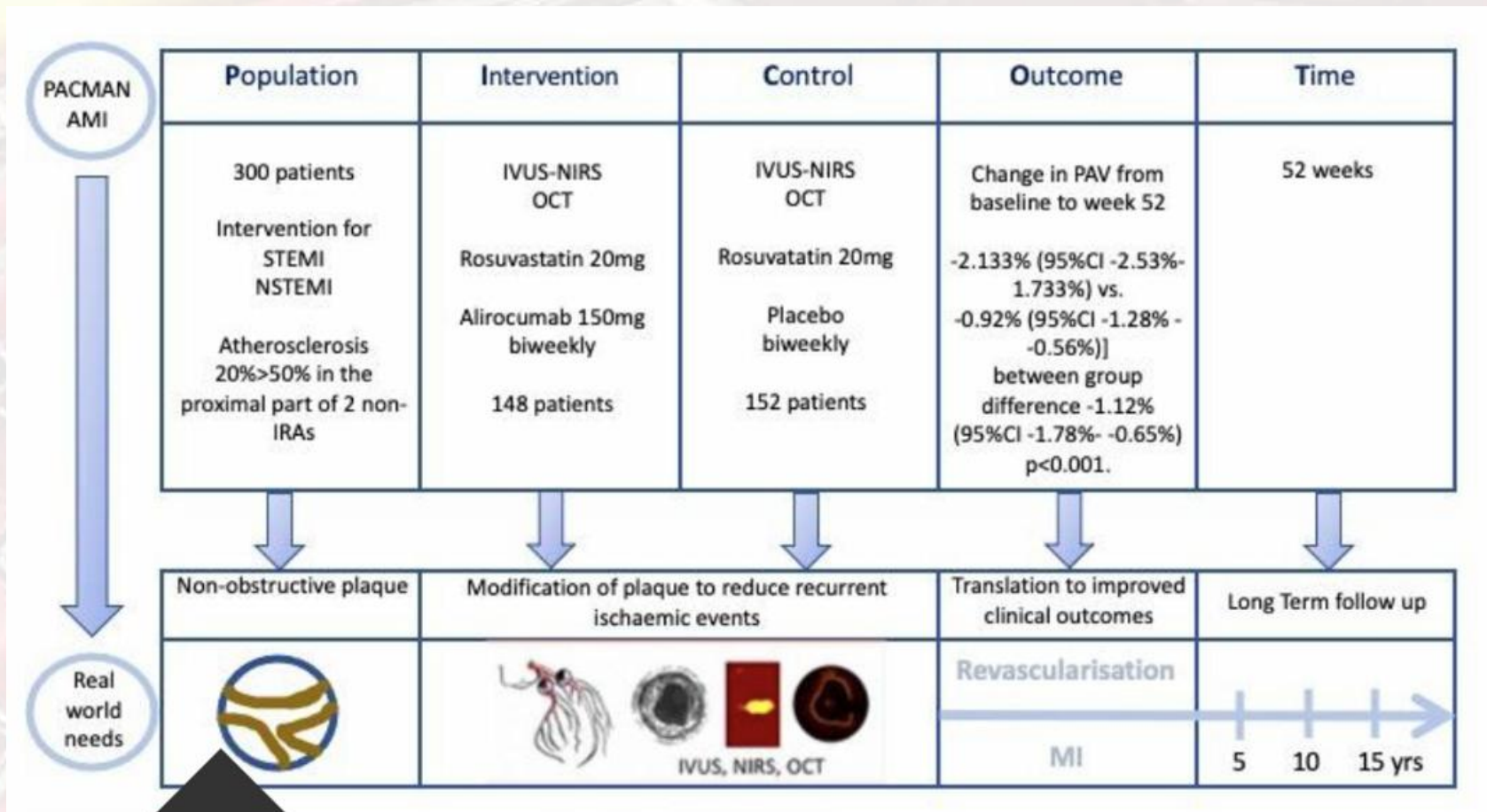
Bonaca MP et al. Circulation. 2018 Jan 23;137(4):338-350.

Evolocumab: Lipidbogen/Fibröse Kappe



Nicholls SJ et al. JACC Cardiovasc Imaging. 2022 Jul;15(7):1308-1321

PCSK-9-Inhibitoren



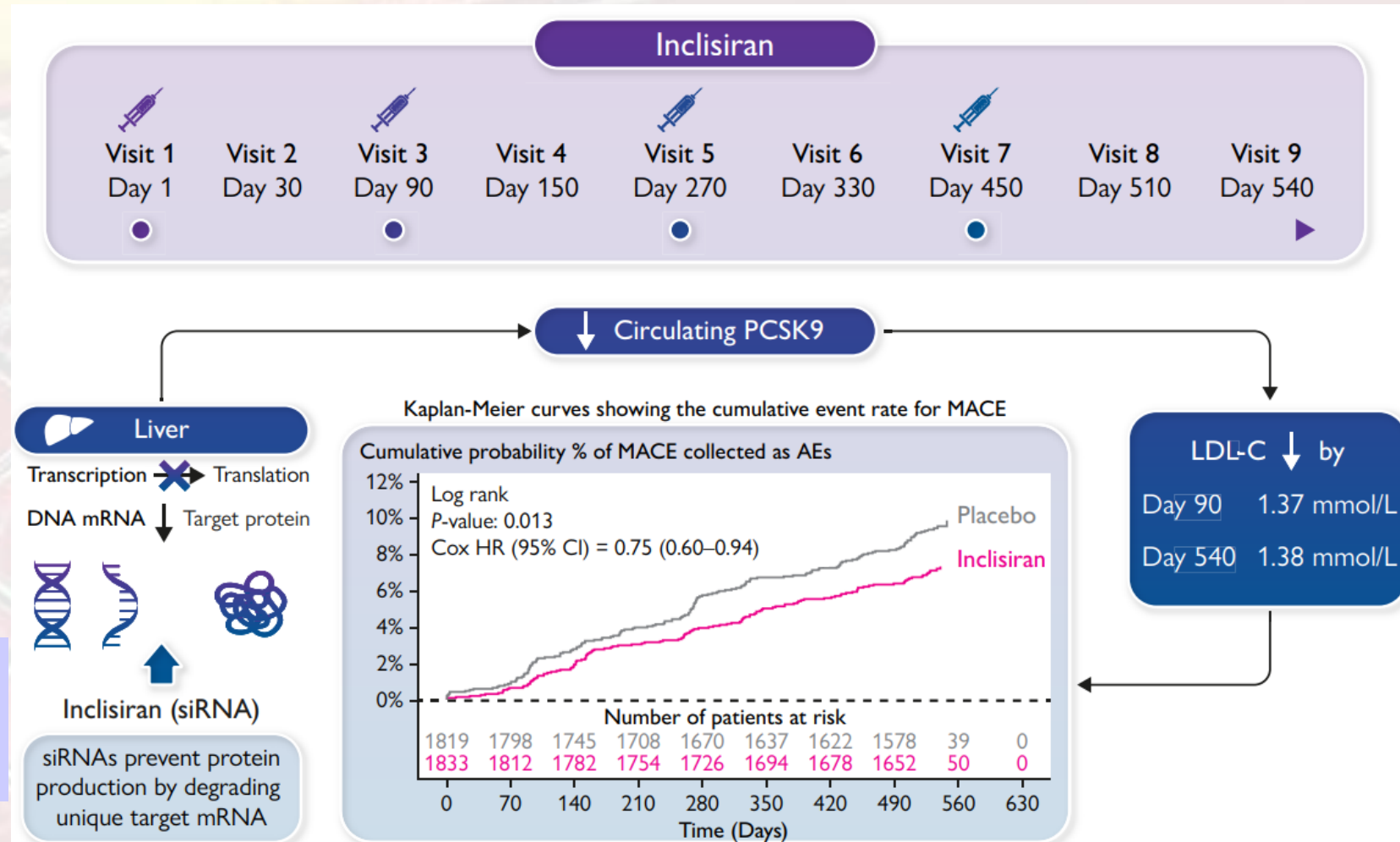
JAMA. Published online April 3, 2022. doi:10.1001/jama.2022.5218

PCSK-9-Inhibitoren/Plaquemorphologie

- Primärer Endpunkt: Veränderung des Plaquevolumens nach 52 Wochen
 - Abnahme mittleres Plaquevolumen um 2,3% (Placebo 0,92%)
 - Zunahme der Dicke der fibrösen Kappe (Plaquestabilisierung)
 - Signifikante Abnahme des Lipidgehaltes in der Plaque
- Somit deutliche Verbesserung des morphologischen Bildes

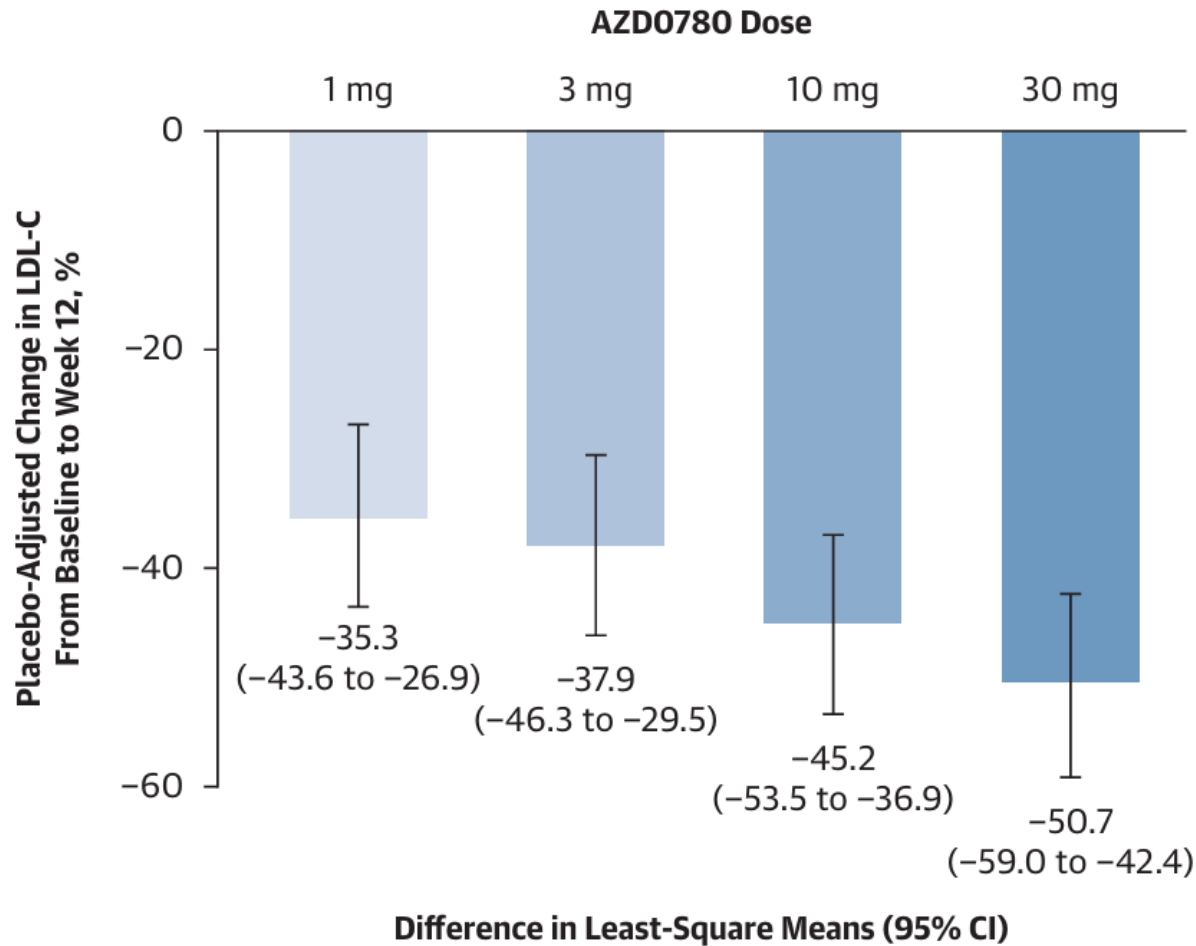
Gepoolte retrospektive Analyse aus ORION 9-11.
Hinweise auf Reduktion des kombinierten Endpunktes

Endpunktdaten aus prospektiv randomisierten Doppelblindstudien fehlen



Ray KK et. al. Eur Heart J. 2023 Jan 7;44(2):129-138.

Oraler PCSK 9 Inhibitor



Key Points

- All AZD0780 doses demonstrated statistically significant reductions in LDL-C vs placebo ($P < 0.001$)
- AZD0780 was well tolerated at all doses

Koren MJ, et al. JACC. 2025;■(■):■-■.

Empfehlungen zum Einsatz von PCSK 9-I.

Diabetes mellitus

Lipid-lowering treatment in patients with diabetes

Statins are recommended as the first-choice LDL-C-lowering treatment in patients with diabetes and above-target LDL-C levels. Administration of statins is defined based on the CV risk profile of the patients and the recommended LDL-C (or non-HDL-C) target levels.

A PCSK9 inhibitor is recommended in patients at very high CV risk, with persistently high LDL-C levels above target despite treatment with a maximum tolerated statin dose, in combination with ezetimibe, or in patients with statin intolerance.

If the target LDL-C is not reached with statins, combination therapy with ezetimibe is recommended.

including a PCSK9 inhibitor is

recommended.^{516,517}

added to ezetimibe may be considered.^{523,524,526}

Mach F et al. Eur Heart J. 2020 Jan 1;41(1):111-188.

Visseren FLJ et al. Eur Heart J. 2021 Sep 7;42(34):3227-3337.

Marx N et al. Eur Heart J. 2023 Aug 25;ehad192.

Niereninsuffizienz

The use of statins or statin/ezetimibe combination is recommended in patients with non-dialysis-dependent stage 3–5 CKD.^{214,222,495,496}



More evidence is needed for PCSK9 inhibitors in specific populations, including patients with severe CKD and on dialysis.

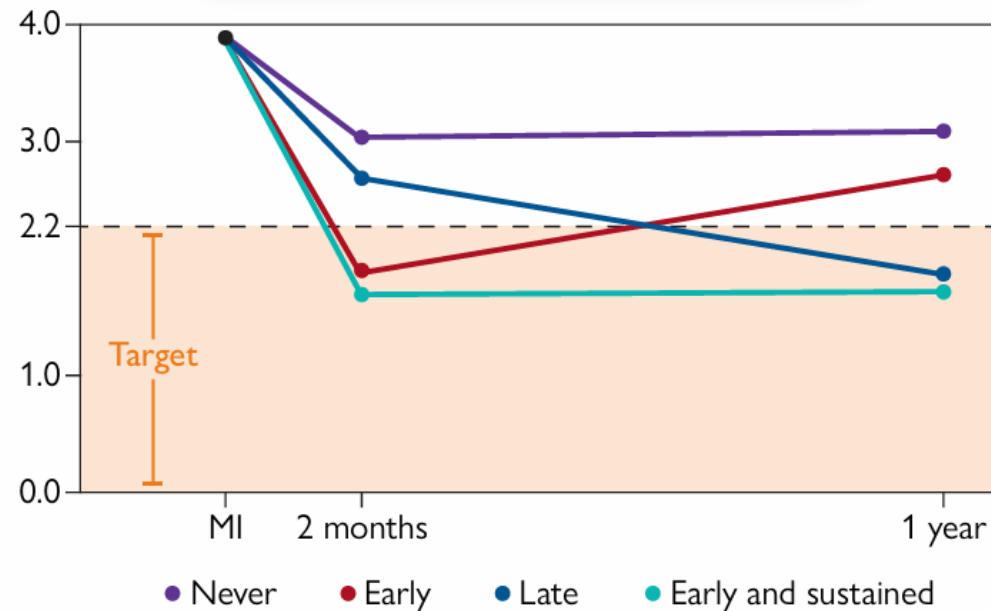
Mach F et al. Eur Heart J. 2020 Jan 1;41(1):111-188.

Myokardinfarkt-möglichst früh in Zielbereich

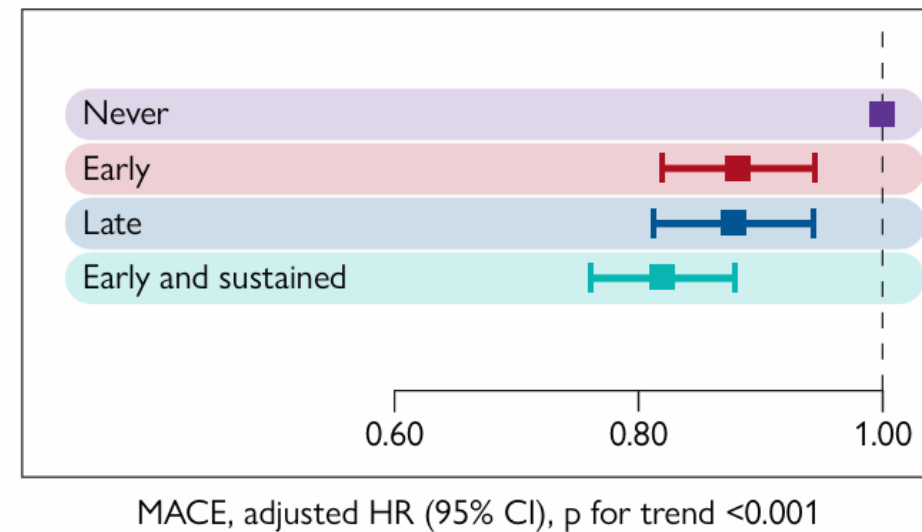
Timing of reaching and duration of staying at non-HDL-C target

46 518 patients with MI and 7407 MACE (all-cause mortality, MI, or stroke)

Median non-HDL-C (mmol/L)



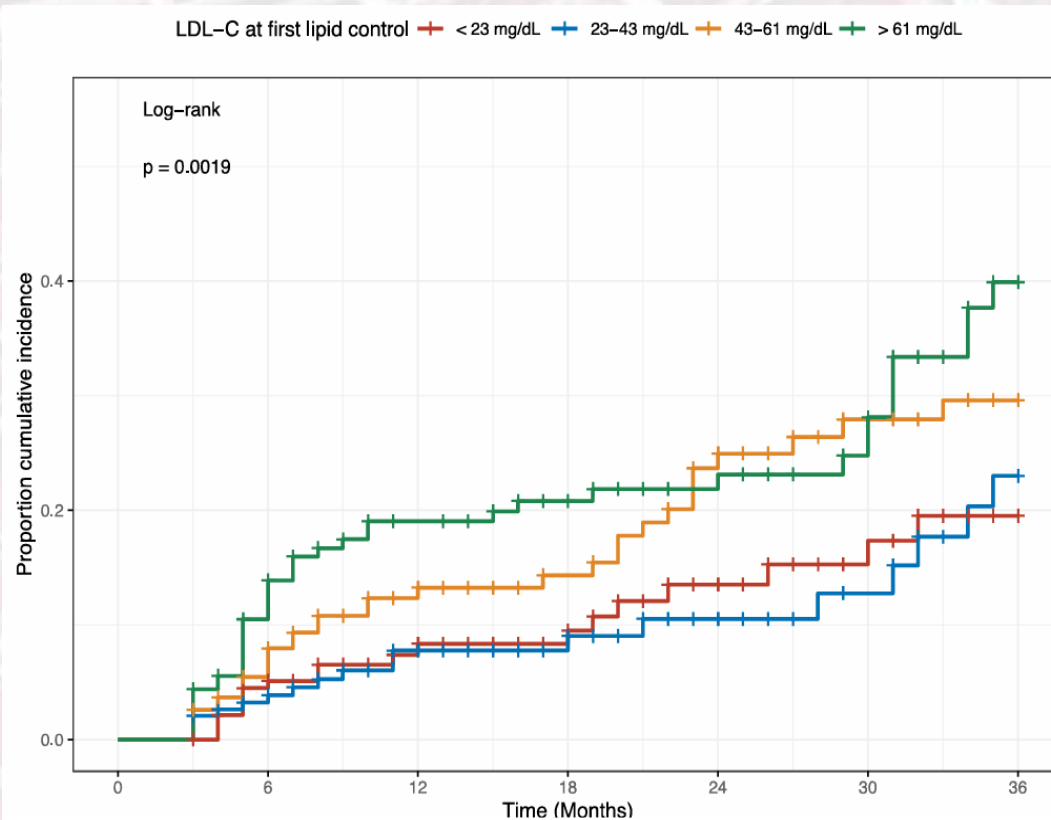
Timing



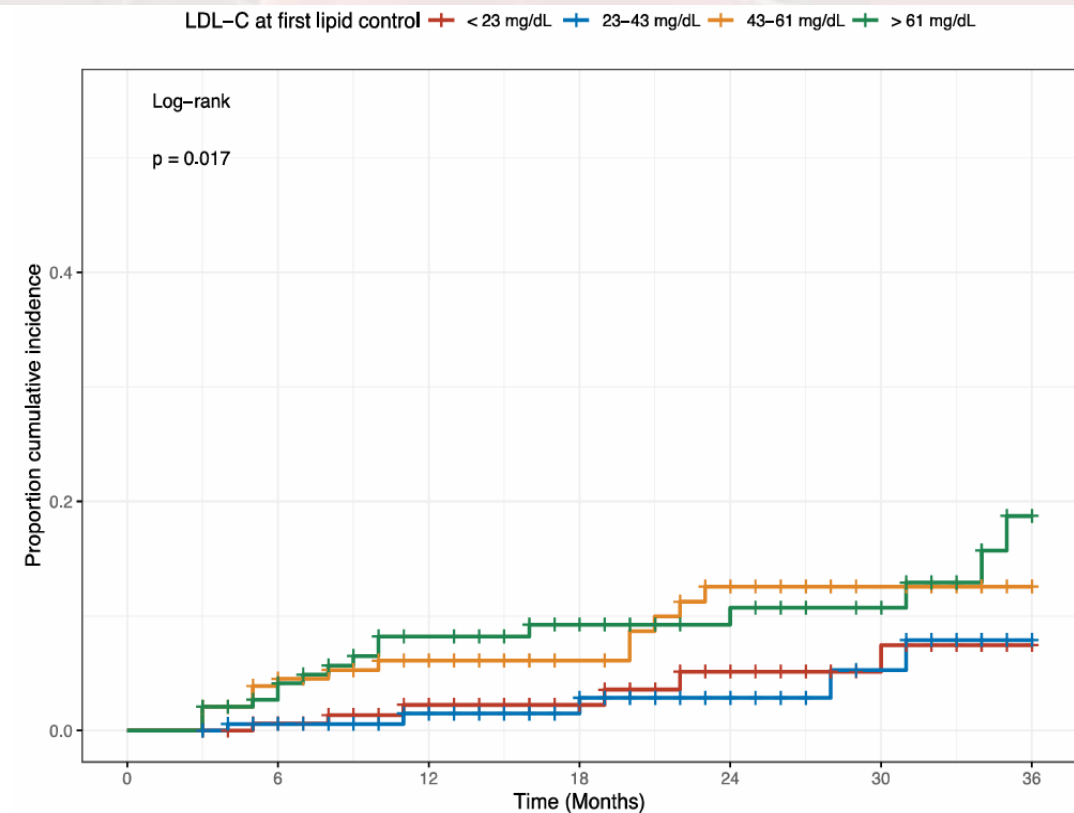
Schubert J et al. Eur Heart J. 2024 Oct 14;45(39).

PCSK 9- strike early, strike strong- MI

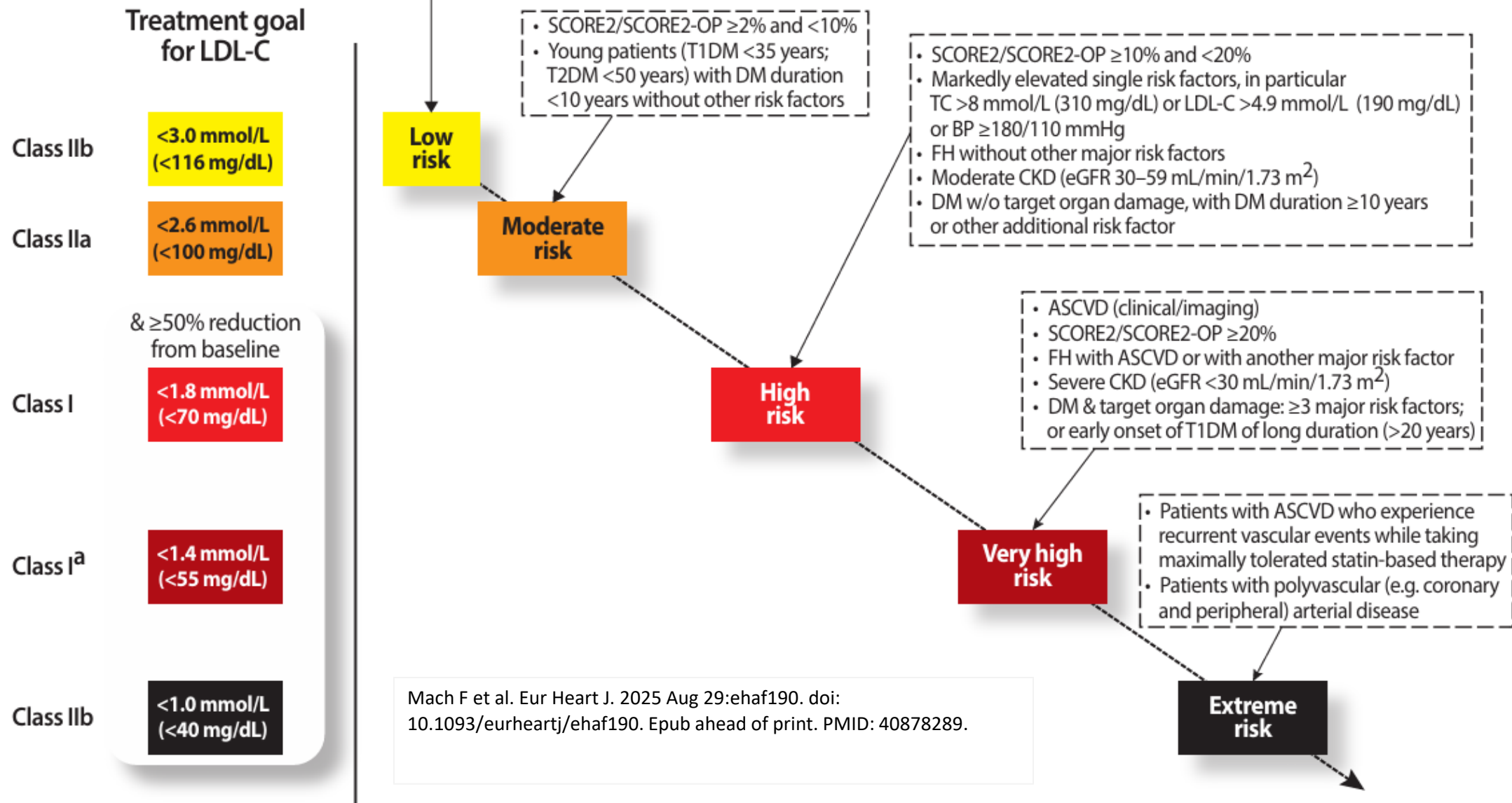
4 Punkt MACE



Gesamtmortalität



Gargiulo P. et al. Eur J Prev Cardiol. 2024 Nov 11;31(15):1806-1816.



^aClass IIa for individuals in primary prevention with FH at very high risk

Mach F et al. Eur Heart J. 2025 Aug 29;ehaf190. doi: 10.1093/eurheartj/ehaf190. Epub ahead of print. PMID: 40878289.

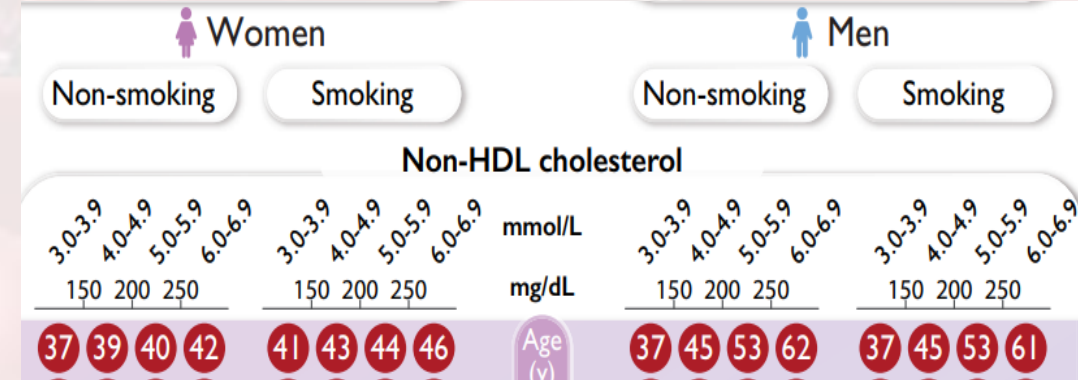
Gesundheit

Blutfettstoff als Gefahr: „Lp(a) muss bei jedem gemessen werden“



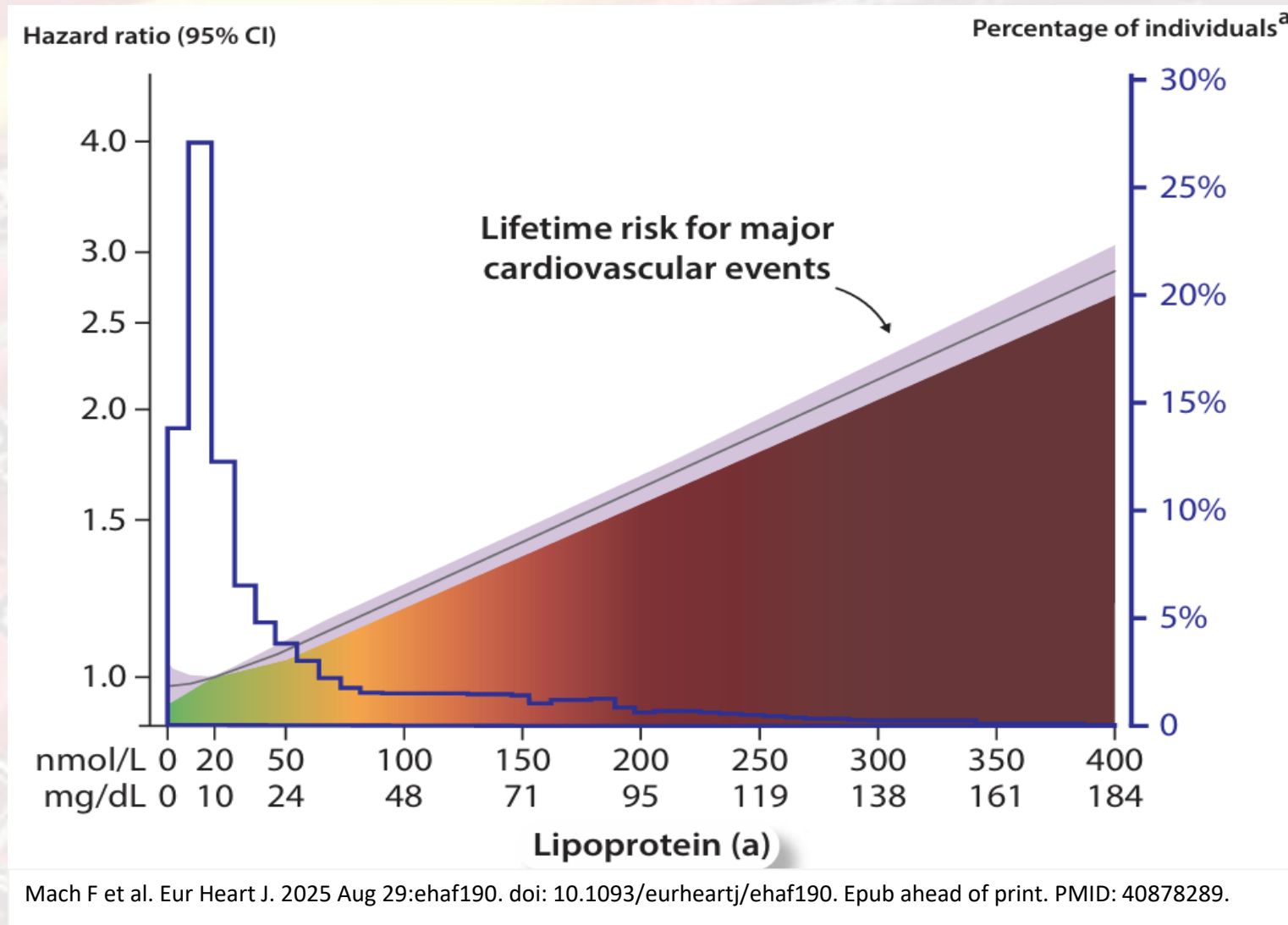
Non-HDL = CHOLges-HDL

Non-HDL =
(LP(a)+VLDL+LDL+IDL+Chylomikrone
n)-HDL

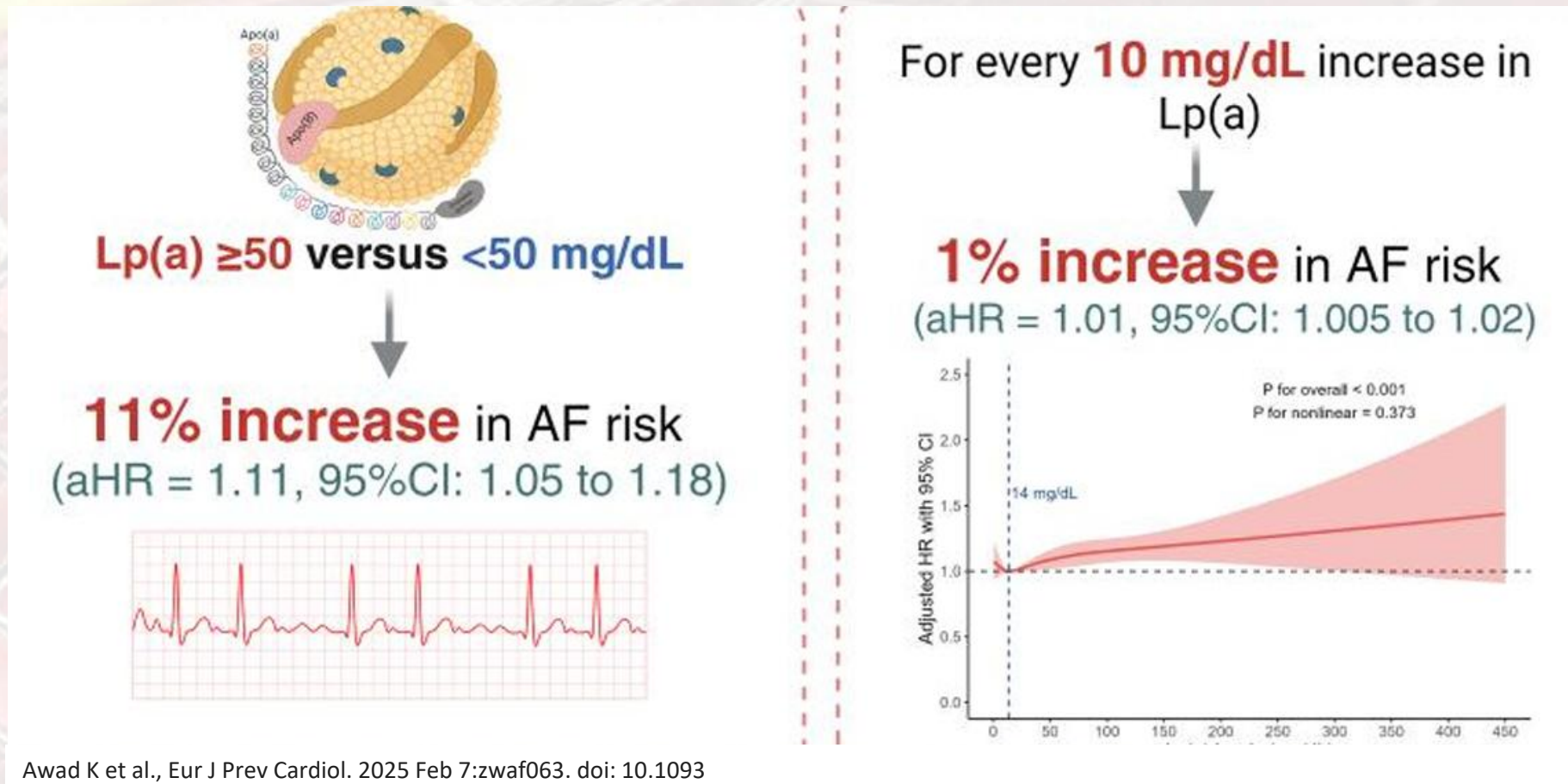


Lp(a): einmal im Leben bestimmen

Lp (a)



Lp (a)-Vorhofflimmern

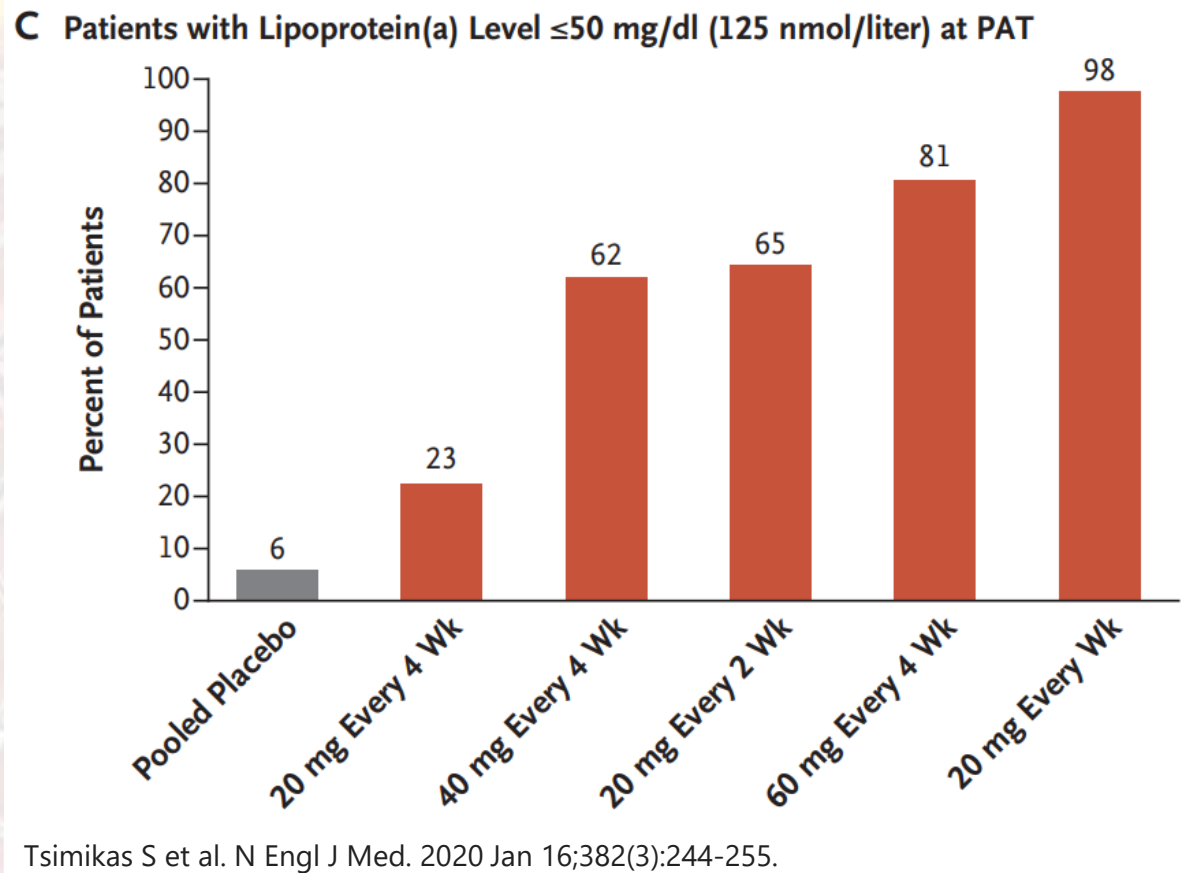


Lp(a)-Risiko

<75 nmol/L (<30 mg/dL)	kein erhöhtes Risiko
75–125 nmol/L (30–50 mg/dL)	moderat erhöhtes Risiko
125–430 nmol/L (50–180 mg/dL)	deutlich erhöhtes Risiko
>430 nmol/L (>180 mg/dL)	sehr stark erhöhtes Risiko

Quelle: cholesterinallianz.at

Ausblick Lp(a)-Plasma-Levels-Pelacarsen



Pelacarsen-Antisense-Oligonucleotid → Hemmung der mRNA von Lp(a)

Je nach Dosierung und Verabreichungsintervall senkt Pelacarsen Lp(a) in bis zu 98% der Fälle unter 50 mg/dl im Vergleich zu Placebo

Senkung von bis zu 80%

Endpunktdaten 2026 erwartet (HORIZON)

Warum Non-HDL?

- Alle Lipoproteine berücksichtigt: VLDL, Lp (a), IDL, LDL, Chylomikronen
- NON-HDL = $CHOL_{ges} - HDL$
- Friedewal-Formel: $LDL = CHOL_{ges} - HDL - (TRI/5)$
—> Hohe TRI → falsch niedriges LDL

Score 2 ist unabhängig von den Triglyceriden, Fibrat erst wenn TRI >500 mg/dl (Pankreas) oder um Ausgangs-LDL zu bestimmen

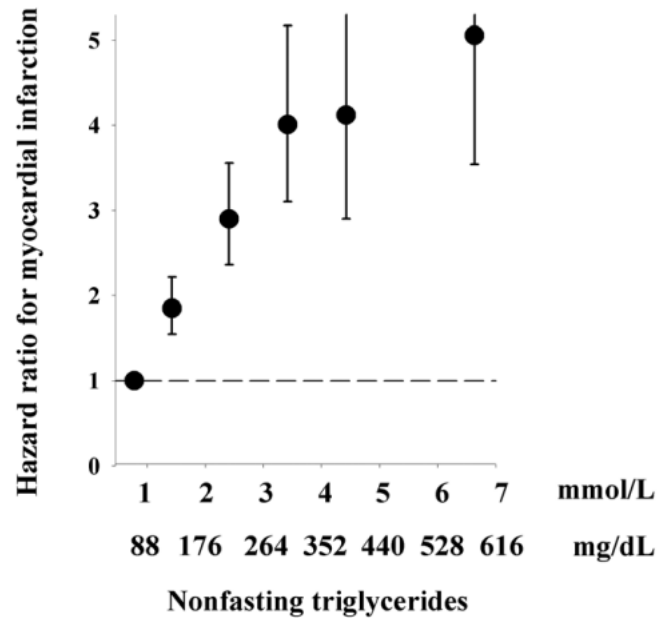
Triaglyceride

Copenhagen City Heart Study and Copenhagen General Population Study

Myocardial infarction

N=96,394 (Events=3,287)

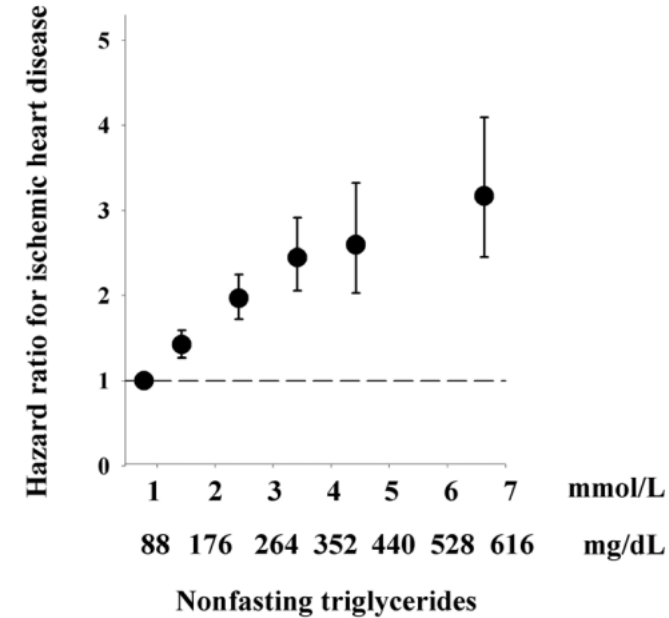
Median follow-up 6 years



Ischemic (=coronary) heart disease

N=93,410 (Events=7,183)

Median follow-up 6 years



Nordestgaard BG, Varbo A. Lancet. 2014 Aug 16;384(9943):626-635.

Icosapent ethyl (=Omega 3 Fettsäure) → IIb

End Point	Icosapent Ethyl (N=4089) <i>no. of patients with event (%)</i>	Placebo (N=4090) <i>no. of patients with event (%)</i>	Hazard Ratio (95% CI)	P Value
Primary composite	705 (17.2)	901 (22.0)	0.75 (0.68–0.83)	<0.001

Recommendations

High-dose icosapent ethyl (2 × 2 g/day) should be considered in combination with a statin in high-risk or very high-risk patients with elevated triglyceride levels (fasting triglyceride level 135–499 mg/dL or 1.52–5.63 mmol/L) to reduce the risk of cardiovascular events.^{8,111}

Class^a

Level^b

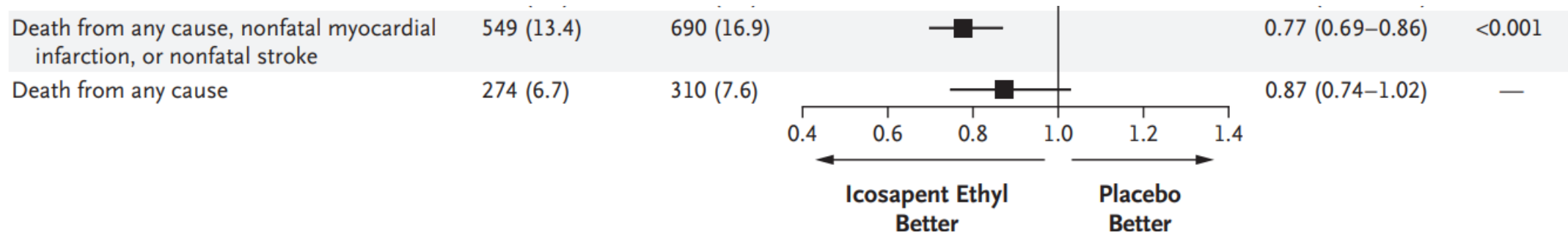
IIa

B

Volanesorsen (300 mg/week) should be considered in patients with severe hypertriglyceridaemia (>750 mg/dL or >8.5 mmol/L) due to familial chylomicronaemia syndrome, to lower triglyceride levels and reduce the risk of pancreatitis.^{6,117}

IIa

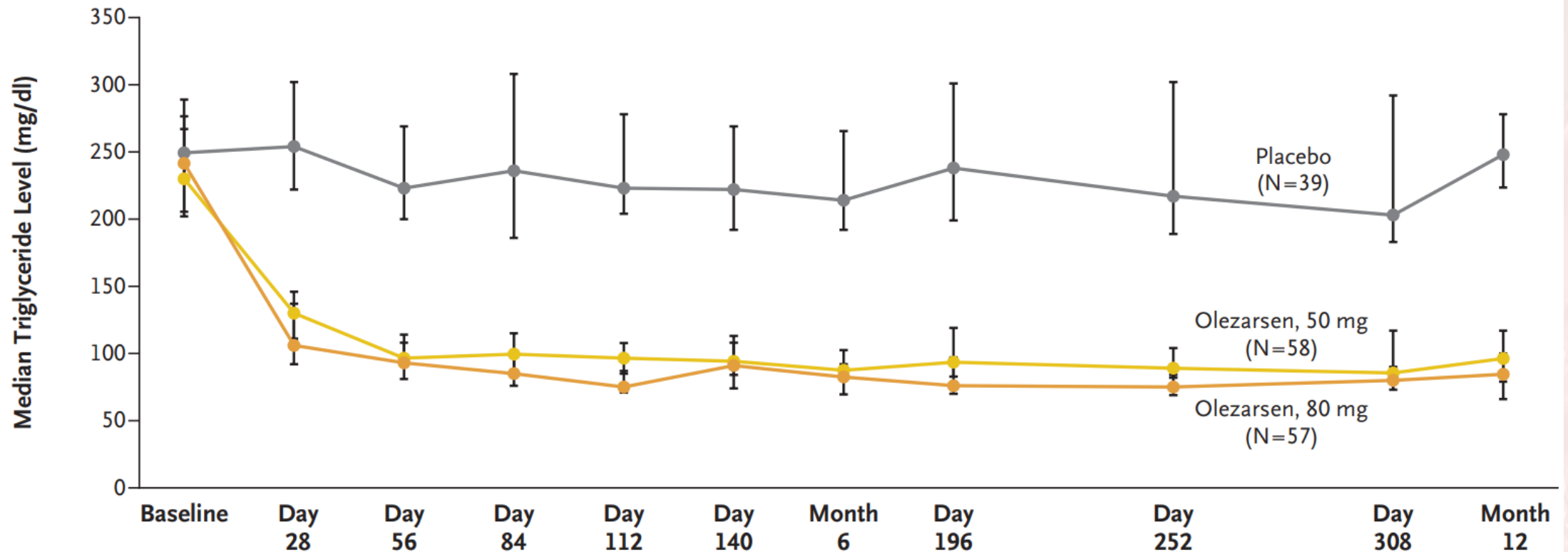
B



Bhatt DL et.al, N Engl J Med. 2019 Jan 3;380(1):11-22.

Olezarsen- noch keine MACE-Daten

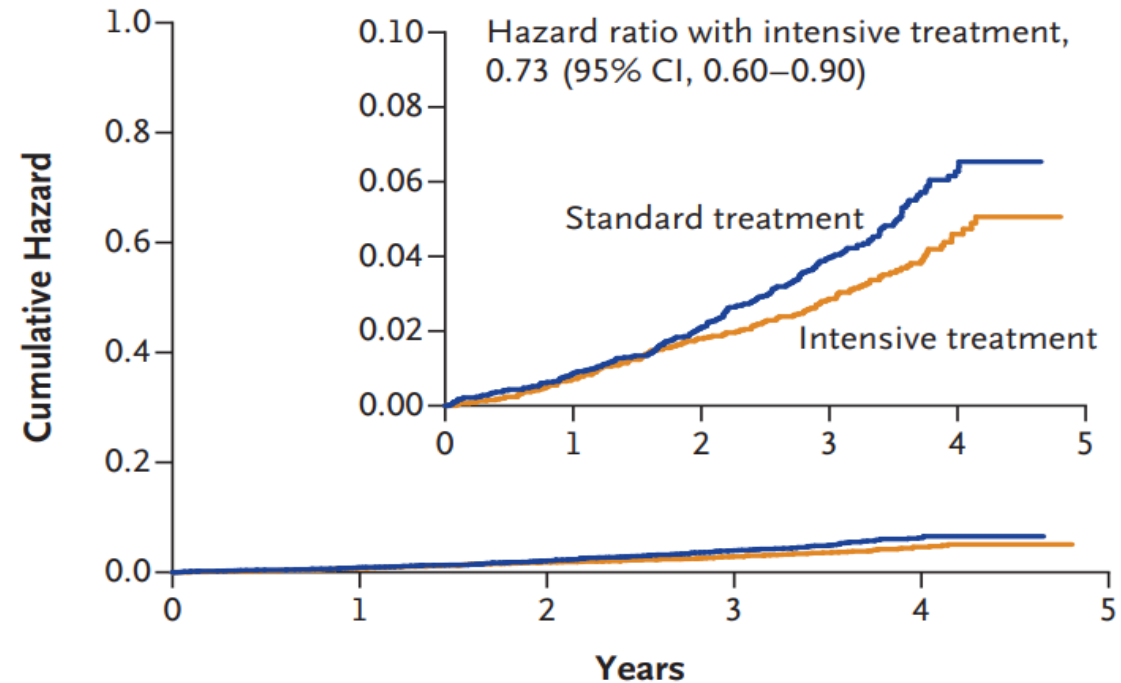
B Triglyceride Levels through 12 Months



Bergmark BA et al. N Engl J Med. 2024 Apr 7. doi: 10.1056/NEJMoa2402309.

Blutdruck: Sprint-Studie

B Death from Any Cause



No. at Risk

Standard treatment	4683	4528	4383	2998	789
Intensive treatment	4678	4516	4390	3016	807

Wright JT Jr et al. N Engl J Med. 2017 Dec 21;377(25):2506.

Non-elevated blood pressure	Elevated blood pressure	Hypertension
Office BP SBP <120 mmHg and DBP <70 mmHg	Office BP SBP 120–139 mmHg or DBP 70–89 mmHg	Office BP SBP ≥140 mmHg or DBP ≥90 mmHg
HBPM SBP <120 mmHg and DBP <70 mmHg	HBPM SBP 120–134 mmHg or DBP 70–84 mmHg	HBPM SBP ≥135 mmHg or DBP ≥85 mmHg
ABPM Daytime SBP <120 mmHg and Daytime DBP <70 mmHg	ABPM Daytime SBP 120–134 mmHg or Daytime DBP 70–84 mmHg	ABPM Daytime SBP ≥135 mmHg or Daytime DBP ≥85 mmHg
Insufficient evidence confirming the efficacy and safety of BP pharmacological treatment	Risk stratify to identify individuals with high cardiovascular risk for BP pharmacological treatment	Cardiovascular risk is sufficiently high to merit BP pharmacological treatment initiation

McEvoy Jwet.al. Eur Heart J. 2024 Oct 7;45(38):3912-4018.

Elevated Blood Pressure + Kardiovaskuläre RF: Ziel-RR: **immer** 120-129/70-85 mmHG

	Established clinical cardiovascular disease	Atherosclerotic cardiovascular disease ^a Heart failure
	Moderate or severe CKD	eGFR <60 mL/min/1.73 m ² or albuminuria ≥30 mg/g (≥3 mg/mmol)
	Other forms of hypertension-mediated organ damage	Cardiac ^b Vascular ^b
	Diabetes mellitus	Type 1 and type 2 diabetes mellitus ^c
	Familial hypercholesterolaemia	Probable or definite familial hypercholesterolaemia

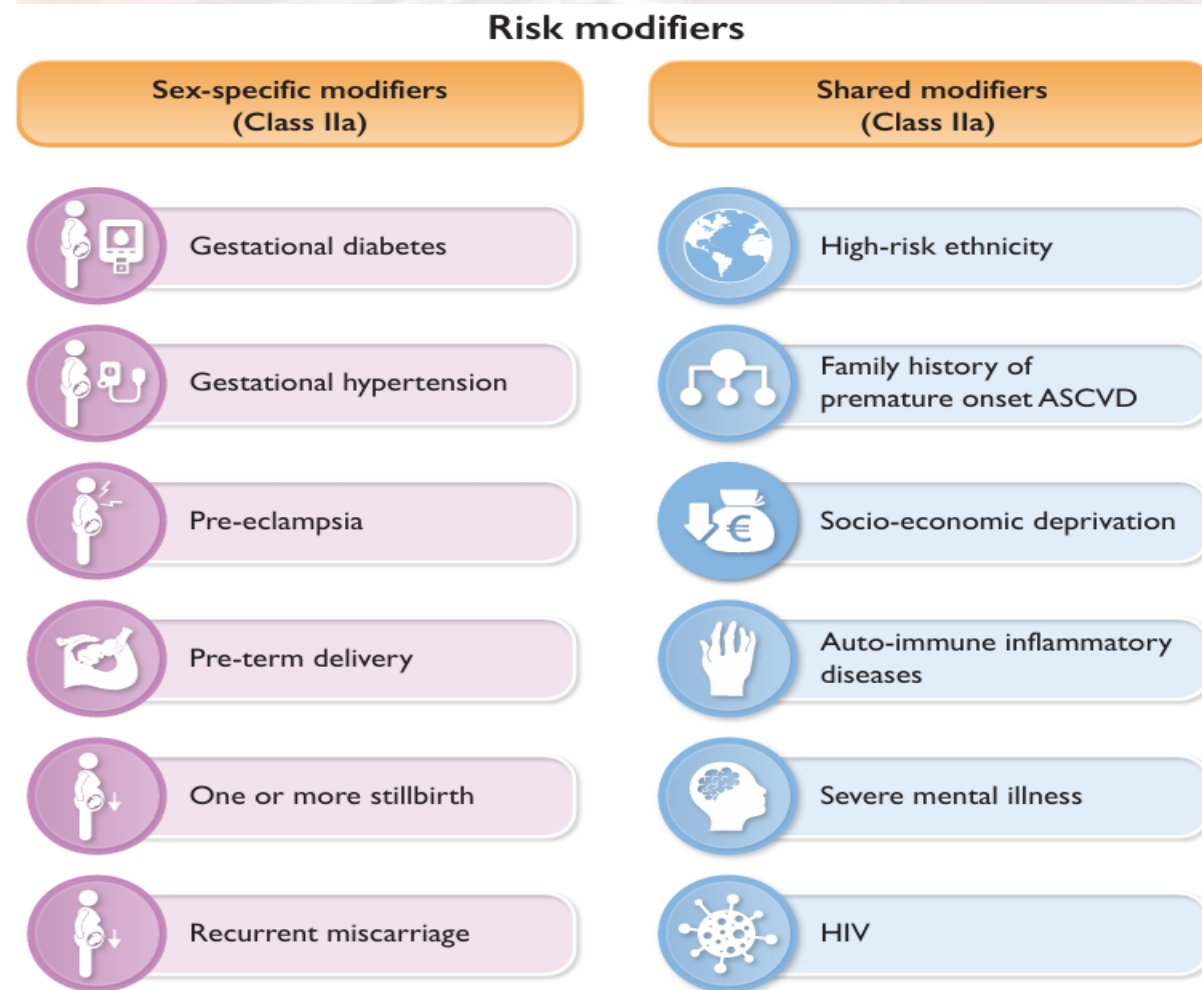
 ESC

McEvoy Jwet.al. Eur Heart J. 2024 Oct 7;45(38):3912-4018.

Kardiometabolische Prävention mit besonderem Blick auf
Lipidmanagement und Hypertonie

Elevated Blood Pressure + SCORE 2/OP/DM(<60 a ohne Endorganschäden) 5-10% +
Modified Risk Factors: Ziel-RR: 120-129/70-85 mmHg

McEvoy Jwet.al. Eur Heart J. 2024 Oct 7;45(38):3912-4018.



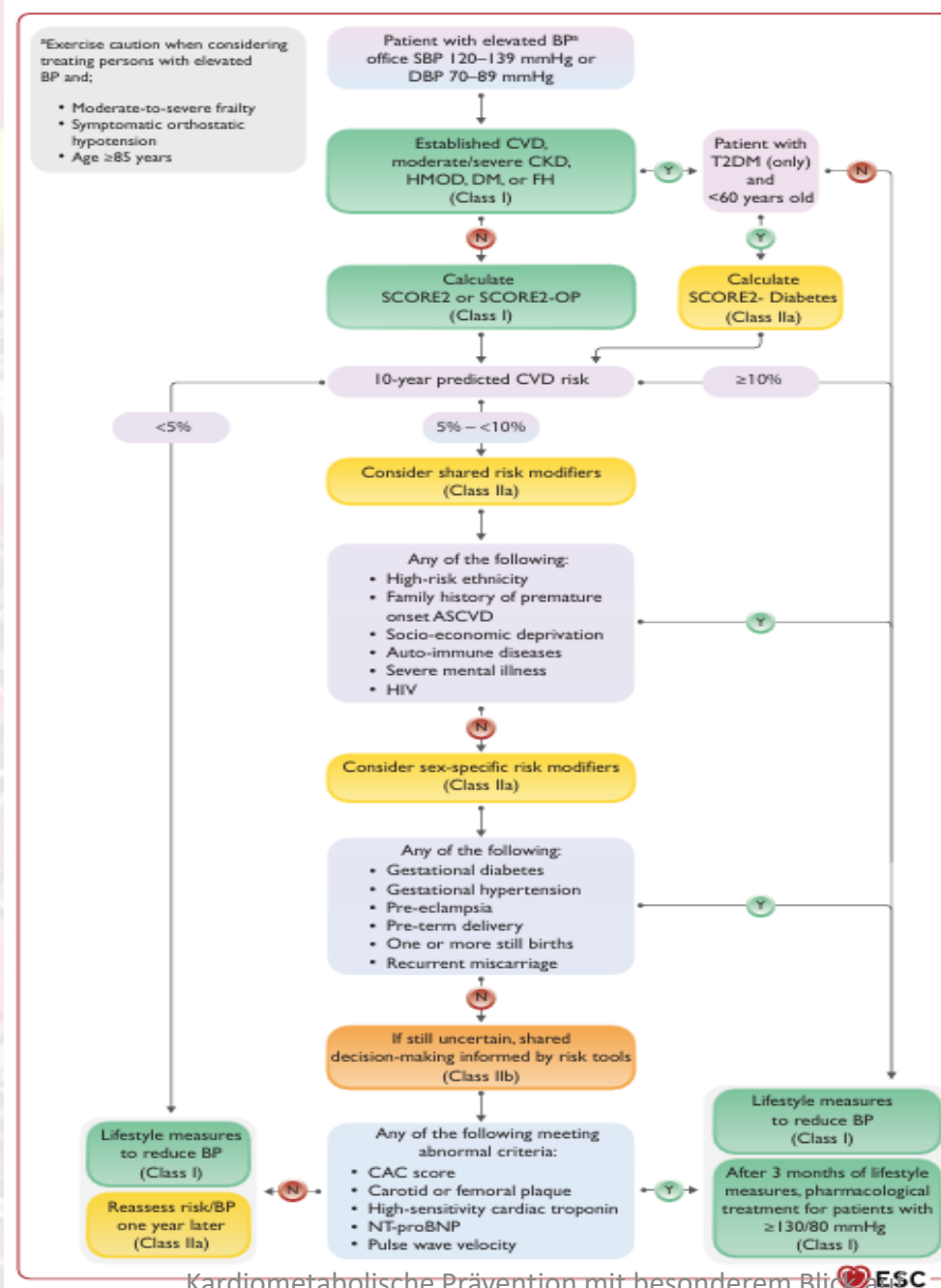
Elevated Blood Pressure ohne kardiovaskuläre RF +/- Modified Risk Factors: Ziel-RR: 120-129/70-85 mmHg in Abh. von SCORES

- SCOR 2/SCORE 2OP/SCORE 2 DM (<60 a ohne Organschäden) >10%
→ sofort medikamentöse Th. wenn SBP 130-139 mmHg
- SCOR 2/SCORE 2OP/SCORE 2 DM (<60 a ohne Organschäden) 5-10%
→ medikamentöse Therapie nach 3 Monaten Lebensstilmodifikation, wenn RR > 130/80 mmHG

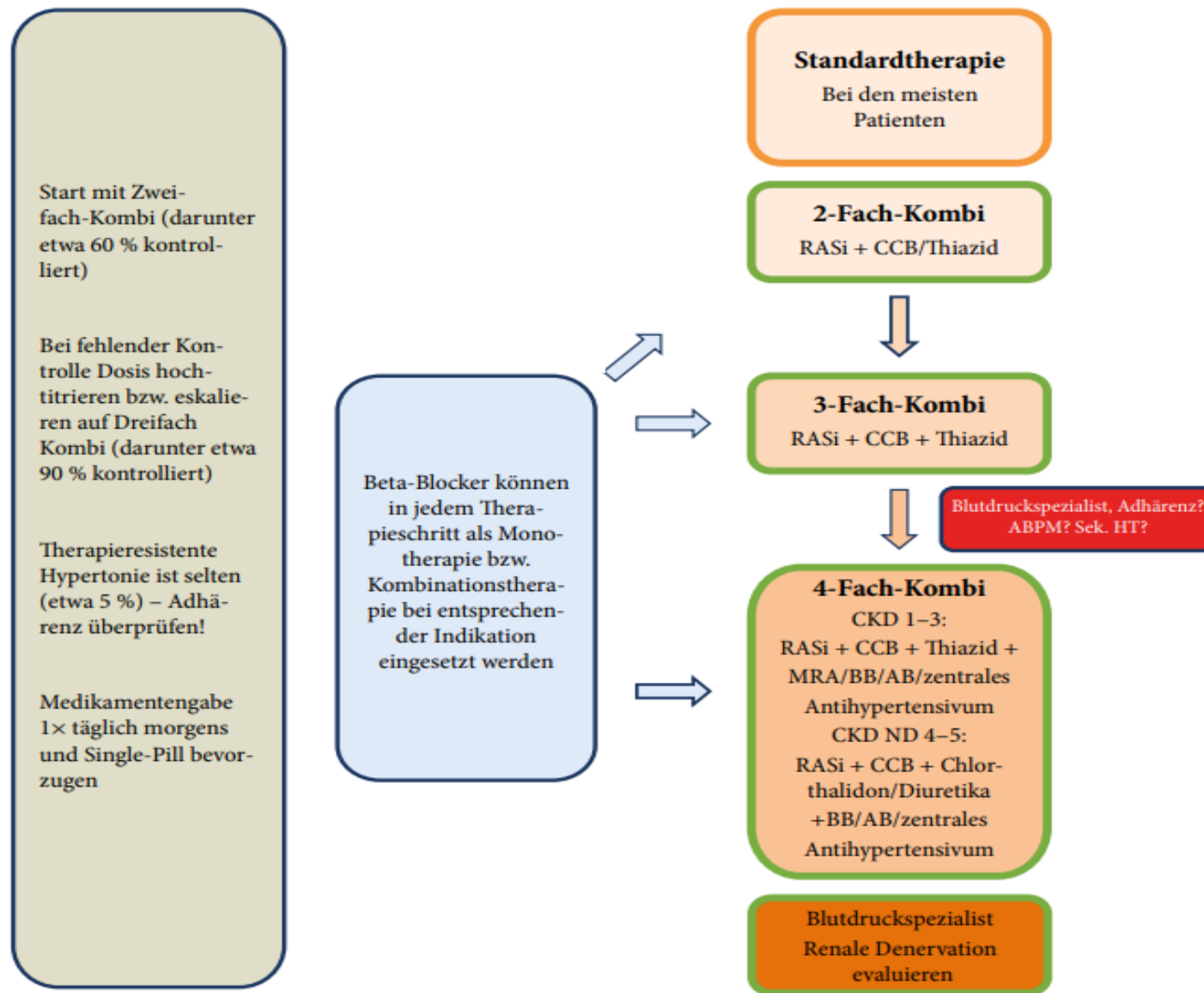
Ohne Modified Risk Faktoren: SCOR 2/SCORE 2OP /SCORE 2 DM (<60 a ohne Organschäden) <10% → Therapie erst wenn RR > 140/90 mmHG

Ohne Modified Risk Faktoren: SCOR 2/SCORE 2OP /SCORE 2 DM (<60 a ohne Organschäden) >10% → medikamentöse Th. wenn SBP 130-139 mmHg

Gilt nur für Selbstmessung zu Hause, für alle gilt: wenn Elevated Blood Pressure vor medikamentöser Therapie **Lebensstilmodifikation für 3 Monate.**

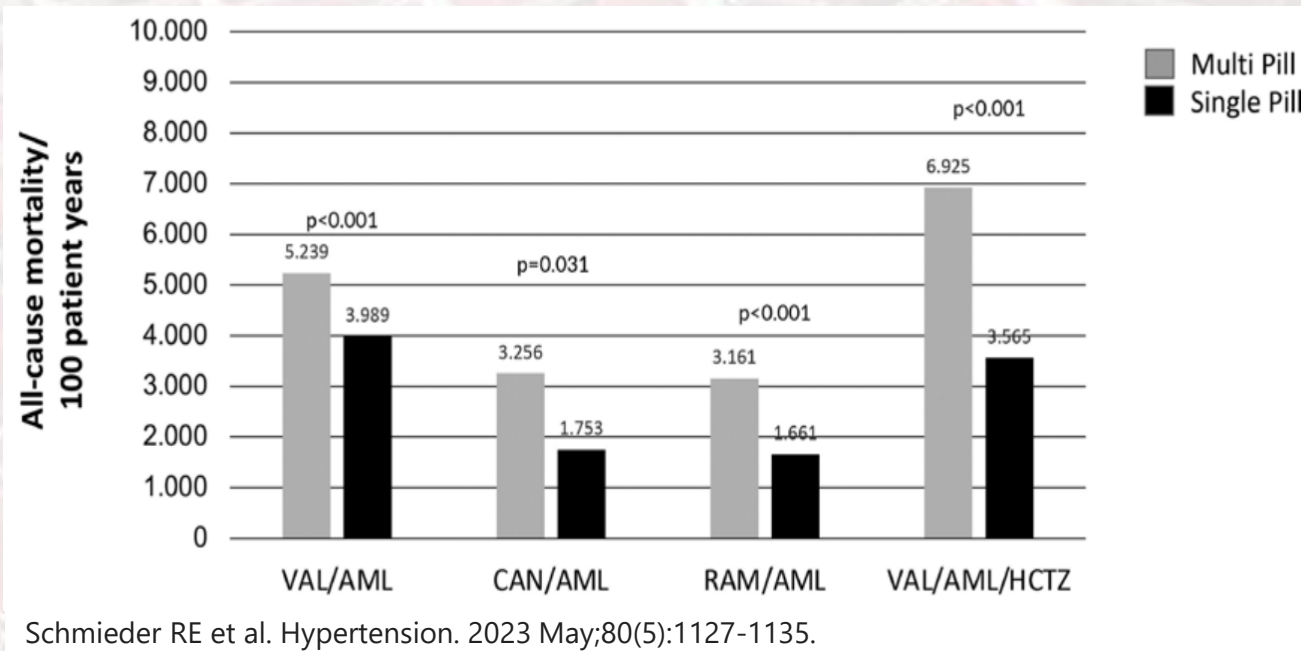


Screening sekundäre Hypertonie: Bei PatientInnen <40 a bei denen die voll ausdosierte Dreifachtherapie keine zufriedenstellenden RR-Werte liefert

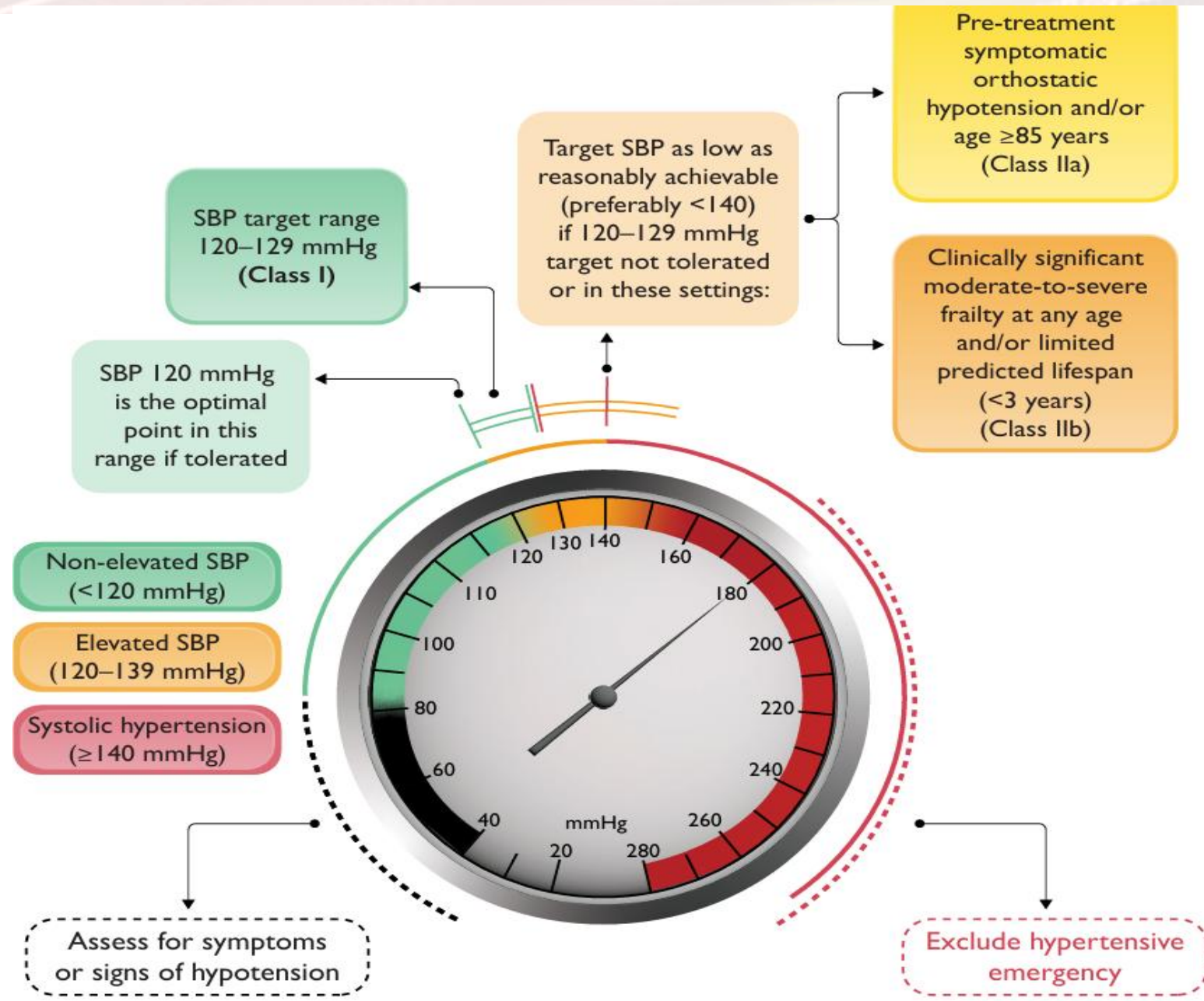


Weber T Hohenstein-Scheibenecker K et al. Austrian Journal of Hypertension 2023; 27 (2), 20-28

Immer nach dem „Single-Pill-Prinzip“ vorgehen: START-Studie



- Kardiovaskuläre Ereignisse um 20% reduziert
- Sterblichkeit um 13% reduziert



Zusammenfassung

Eine evidenzbasierte Behandlung des RR und der Hyperlipidämie ist nur durch eine suffiziente Risikostratifizierung möglich

Hierzu sollten die Scores und Checklisten verwendet werden

Hauptziel der Lipidtherapie ist die Verhinderung von Plaquerupturen durch medikamentöse Plaquestabilisierung, bzw. überhaupt die Verhinderung, dass sie sich bilden.

Lp(a): behandeln wir indirekt bei erhöhten Scores (erhöhtes Lp(a) → Non-HDL → höhere Scores

Triglyceride: errechnetes LDL falsch niedrig, behandeln mit Icosapent-Ethyl

RR: Unterscheidung Normotension, Hypertension und „erhöhter Blutdruck“

Erhöhter Blutdruck: 3 Monate Lebensstilmodifikation, dann individuelle Risikostratifizierung

Immer Kombipräparate mit Wirkstoffen mit erwiesenem CV-Benefit verabreichen

Wir behandeln primär Risiko mit dem Ziel der Organprotektion und nicht rein Zahlen

Und wo kann man
das alles
nachlesen?

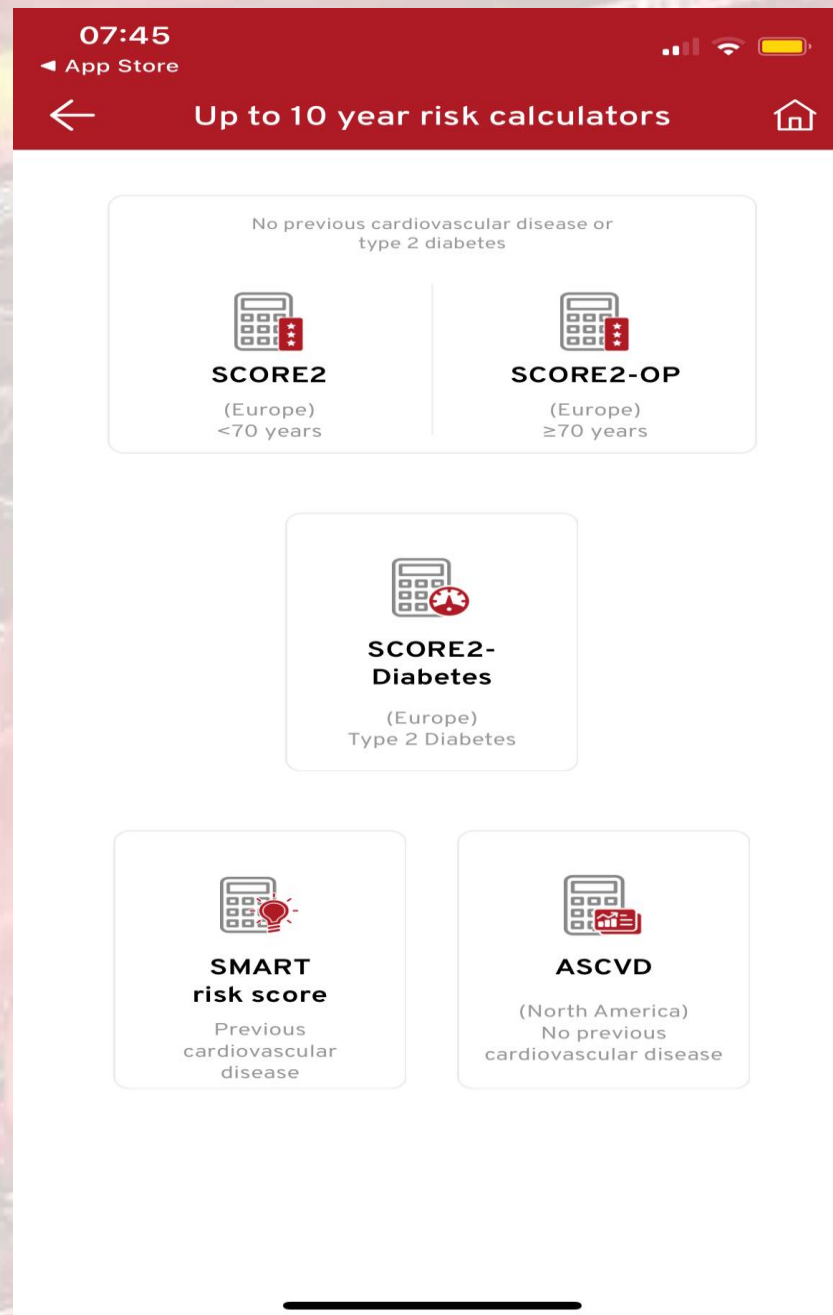
escardio.org/guidelines

30.09.2025

ESC Pocket Guidelines App – Clinical Decision Support System

Clinical Practice
Guidelines Committee

ESC-Scores APP



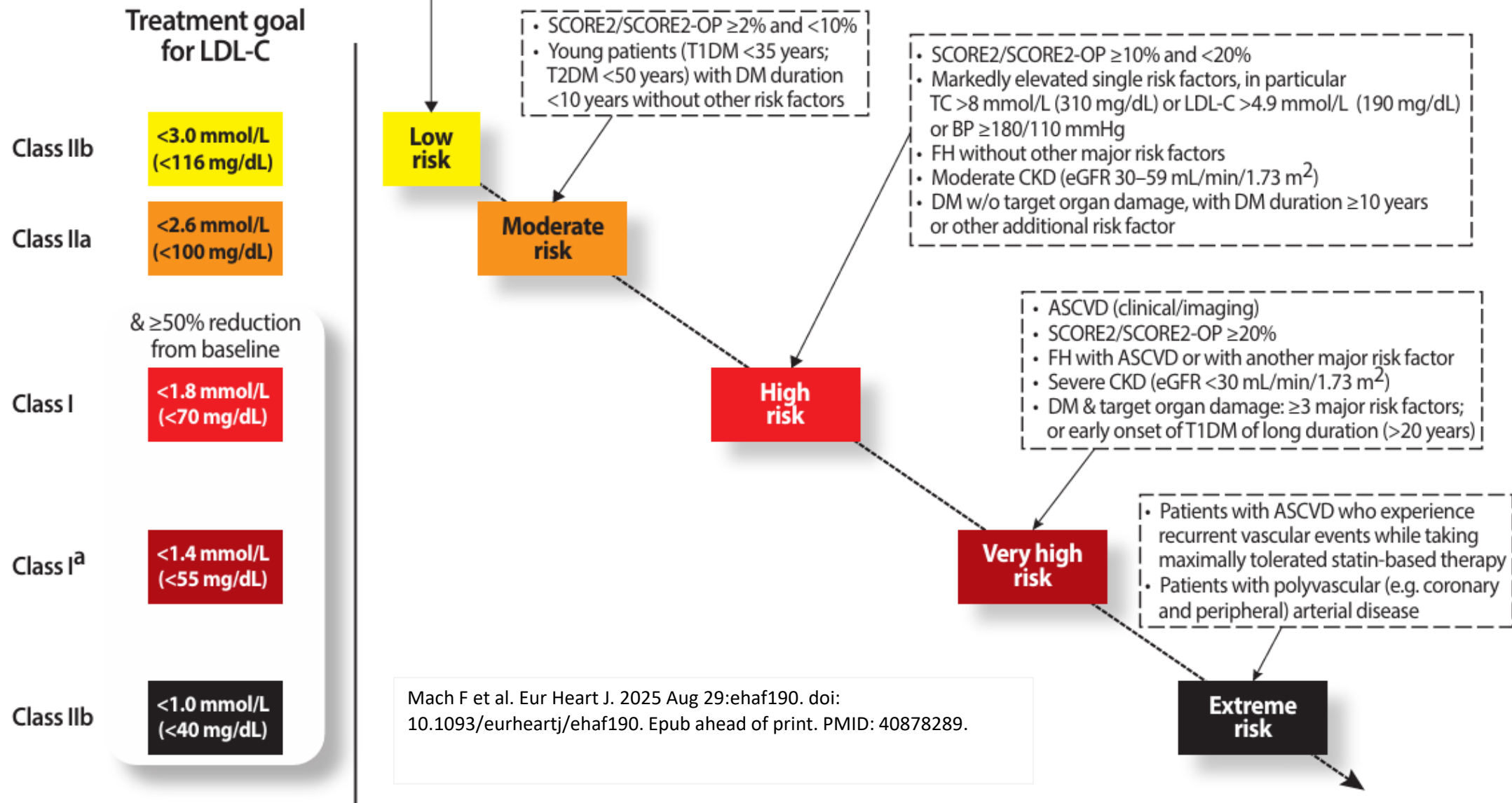
Box 1 Risk modifiers for consideration beyond the risk estimation based on the SCORE2 and SCORE2-OP algorithms

Demographic/clinical conditions

- Family history of premature CVD (men: <55 years; women: <60 years)
- High-risk ethnicity (e.g. Southern Asian)
- Stress symptoms and psychosocial stressors
- Social deprivation
- Obesity
- Physical inactivity
- Chronic immune-mediated/inflammatory disorders
- Major psychiatric disorders
- History of premature menopause
- Pre-eclampsia or other hypertensive disorders of pregnancy
- Human immunodeficiency virus infection
- Obstructive sleep apnoea syndrome

Biomarkers

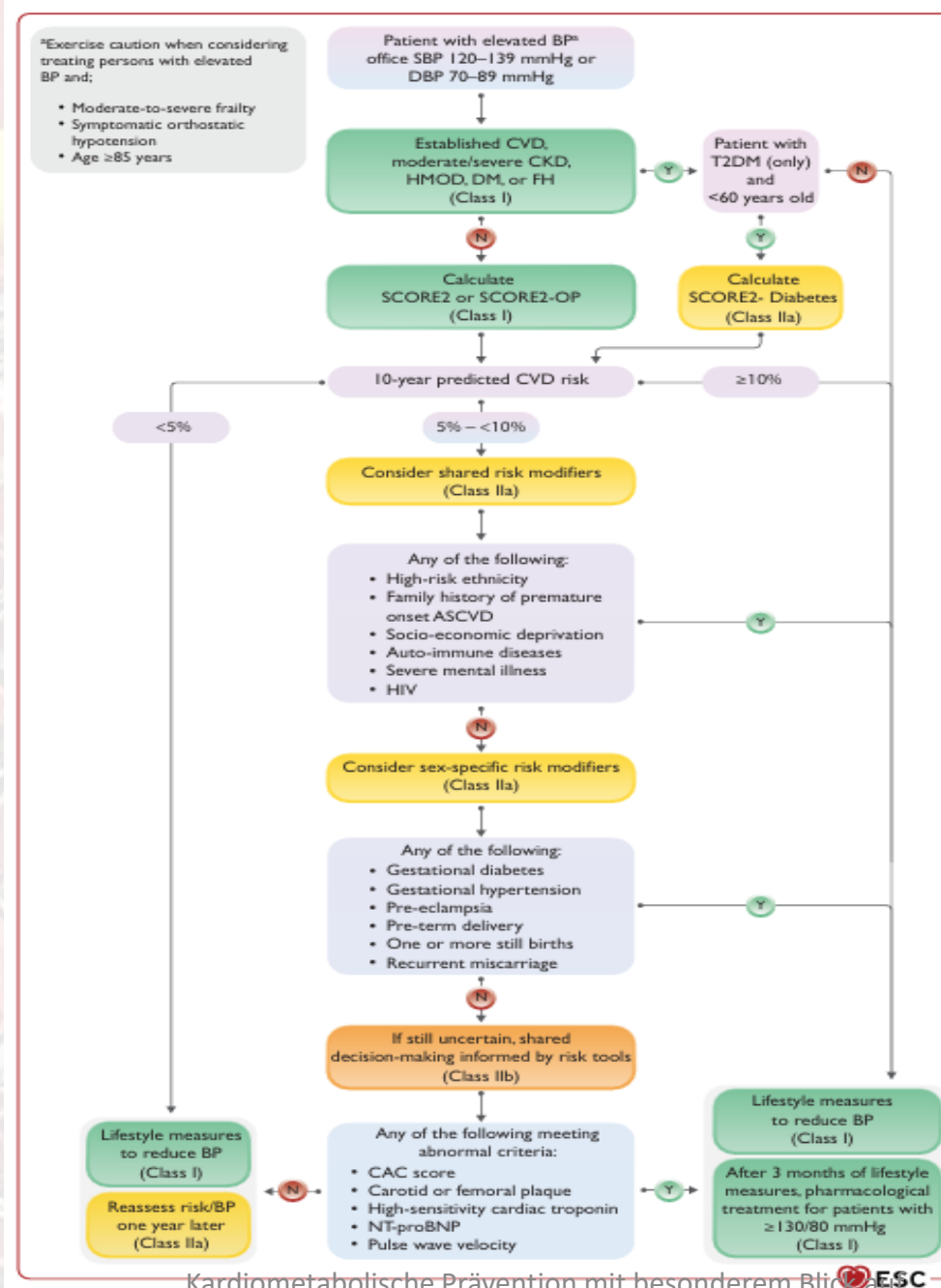
- Persistently elevated hs-CRP (>2 mg/L)
- Elevated Lp(a) [>50 mg/dL (>105 nmol/L)].



Mach F et al. Eur Heart J. 2025 Aug 29;ehaf190. doi: 10.1093/eurheartj/ehaf190. Epub ahead of print. PMID: 40878289.

^aClass IIa for individuals in primary prevention with FH at very high risk

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Insufficient evidence confirming the efficacy and safety of BP pharmacological treatment	Risk stratify to identify individuals with high cardiovascular risk for BP pharmacological treatment	Cardiovascular risk is sufficiently high to merit BP pharmacological treatment initiation



The light at the end of the tunnel

