Medizinische Universität Graz | Forschungseinheit Interdisziplinäre Metabolische Medizin

LIPIDTHERAPIE IM WANDEL

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Conflict of Interest

- **Vortragstätigkeit und Beraterhonorare:**
  Amgen, AstraZeneca, Bayer, Böhringer Ingelheim, Daiichi Sankyo, Eli Lilly, Kapsch, Novartis, NovoNordisk, Sanofi-Aventis

- **Investigator Initiated Study Grants:**
  Böhringer Ingelheim, Eli Lilly, NovoNordisk, Sanofi-Aventis

Medizinische Vorgeschichte:

Adipositas

Bluthochdruck seit 7 Jahren

Diabetes mellitus Typ 2 seit 2 Jahren

Hypercholesterinämie

Medikation:

Ramipril 10 mg 1-0-0

Metformin 1000 mg 1-0-1

Dulaglutide 1,5 mg /Woche

Atorvastatin 80 mg 0-0-1

Ezetimibe 10 mg 0-0-1

ASS 100 mg 0-1-0

Ticagrelor 90 mg 1-0-1

Bisoprolol 2,5 mg 1-0-0

91 kg Körpergewicht

1,72 m Körpergröße

BMI: 30.8 kg/m^2

HbA1c 83 mmol/mol (9.7%)

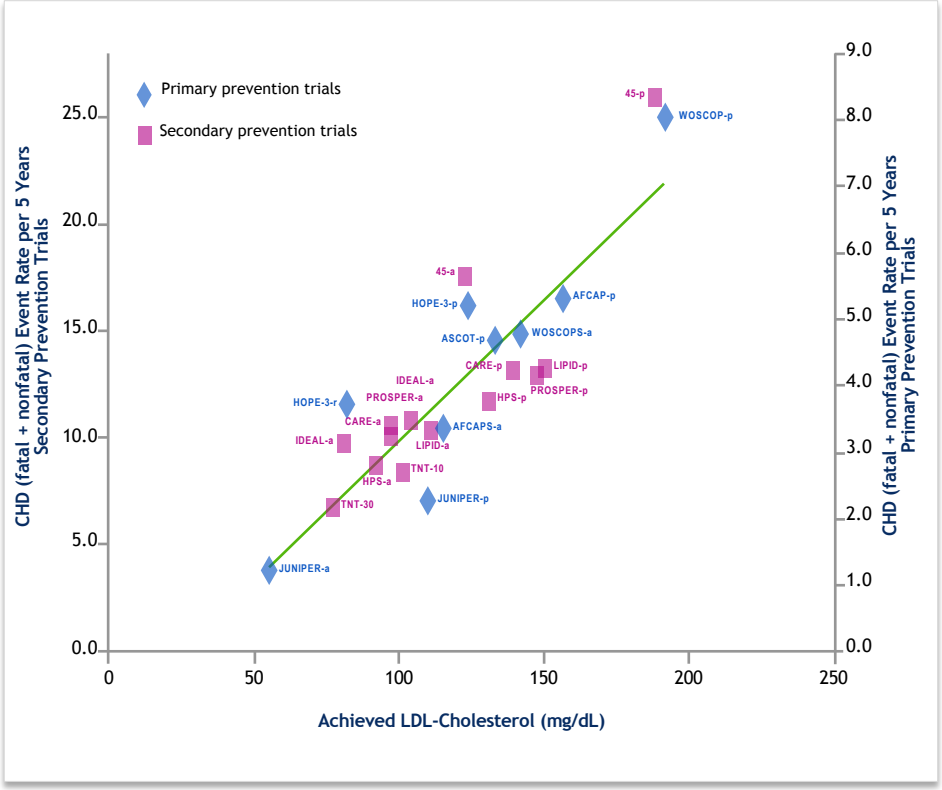
LDL-C 203 mg/dl

eGFR 72 ml/min/1,73 m2

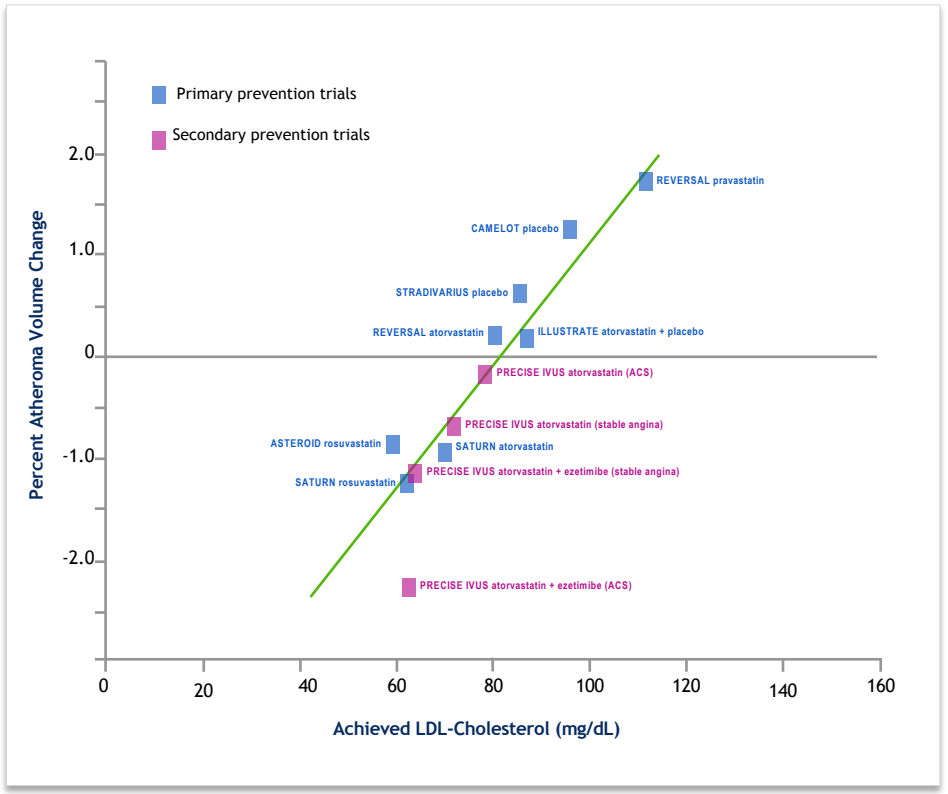
RR 142/92 mmHg

LDL-C Senkung in klinischen Studien

CV event rates reduced



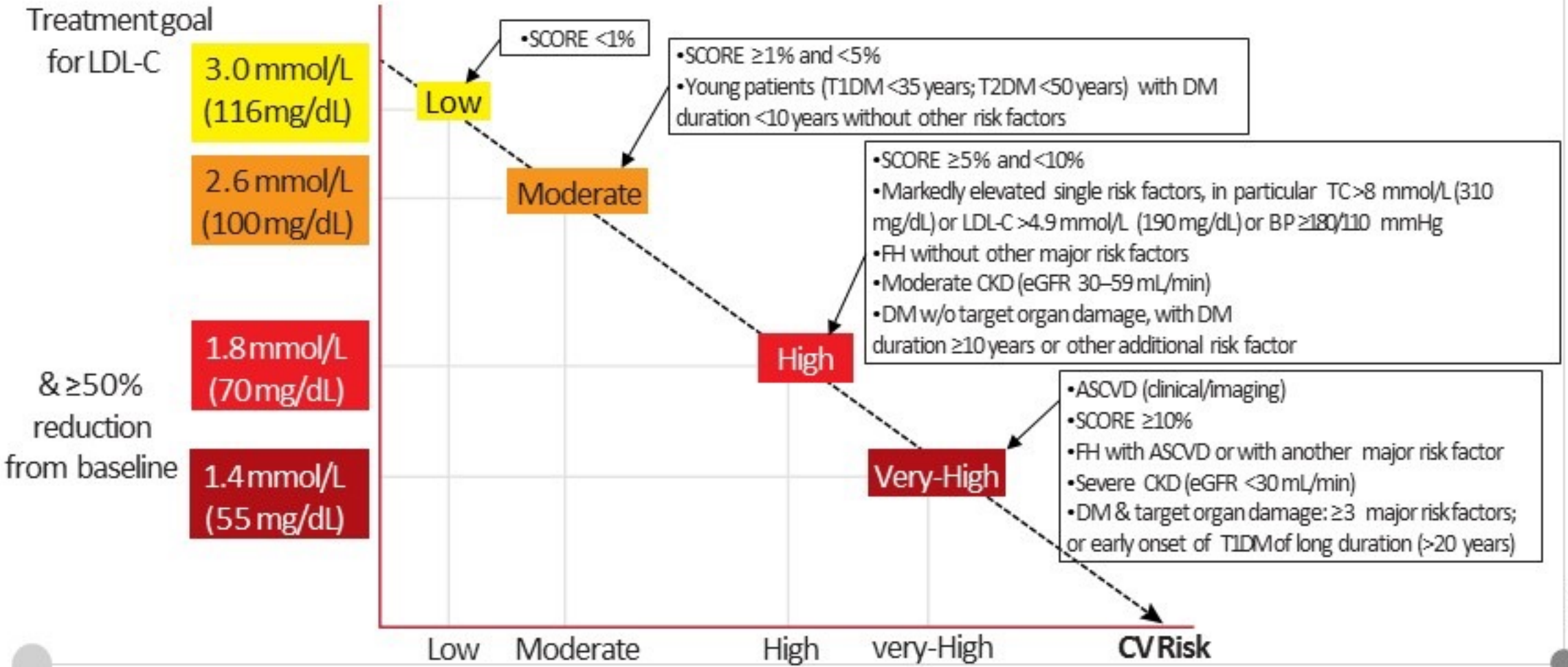
Progression of atherosclerosis slowed



ASCVD, atherosclerotic cardiovascular disease; CHD, coronary heart disease; Ference BA et al. *Eur Heart J*. 2017;38(32):2459-2472.

Central Illustration Upper panel Treatment goals for low-density lipoprotein cholesterol (LDL-C) across categories of total cardiovascular disease risk

EAS ESC European Society of Cardiology



www.escardio.org/guidelines

2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk (European Heart Journal 2019 -doi: 10.1093/eurheartj/ehz455)

Intervention strategies as a function of total cardiovascular risk and untreated low-density lipoprotein cholesterol levels

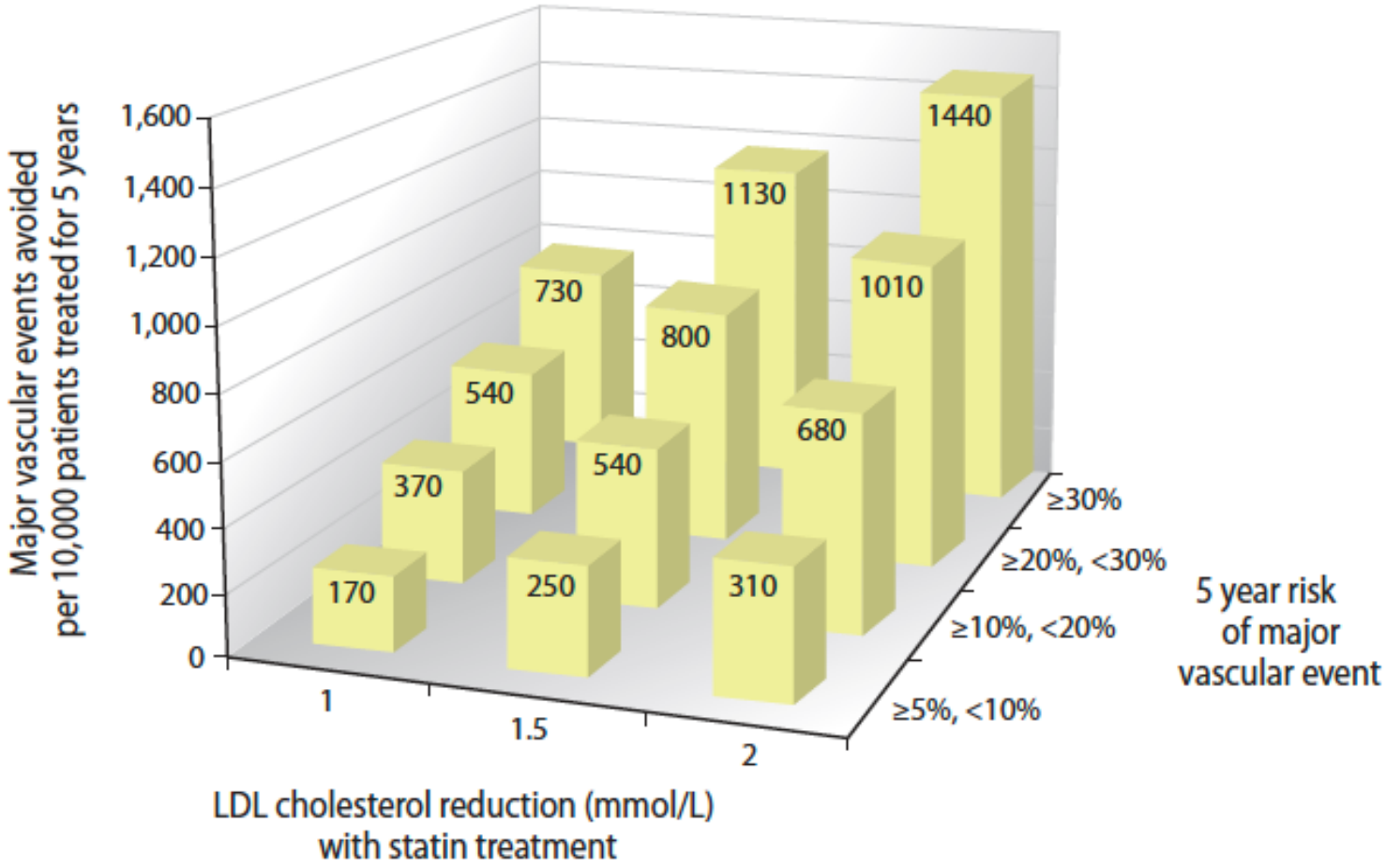
EAS ESC European Society of Cardiology

	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	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	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	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

www.escardio.org/guidelines

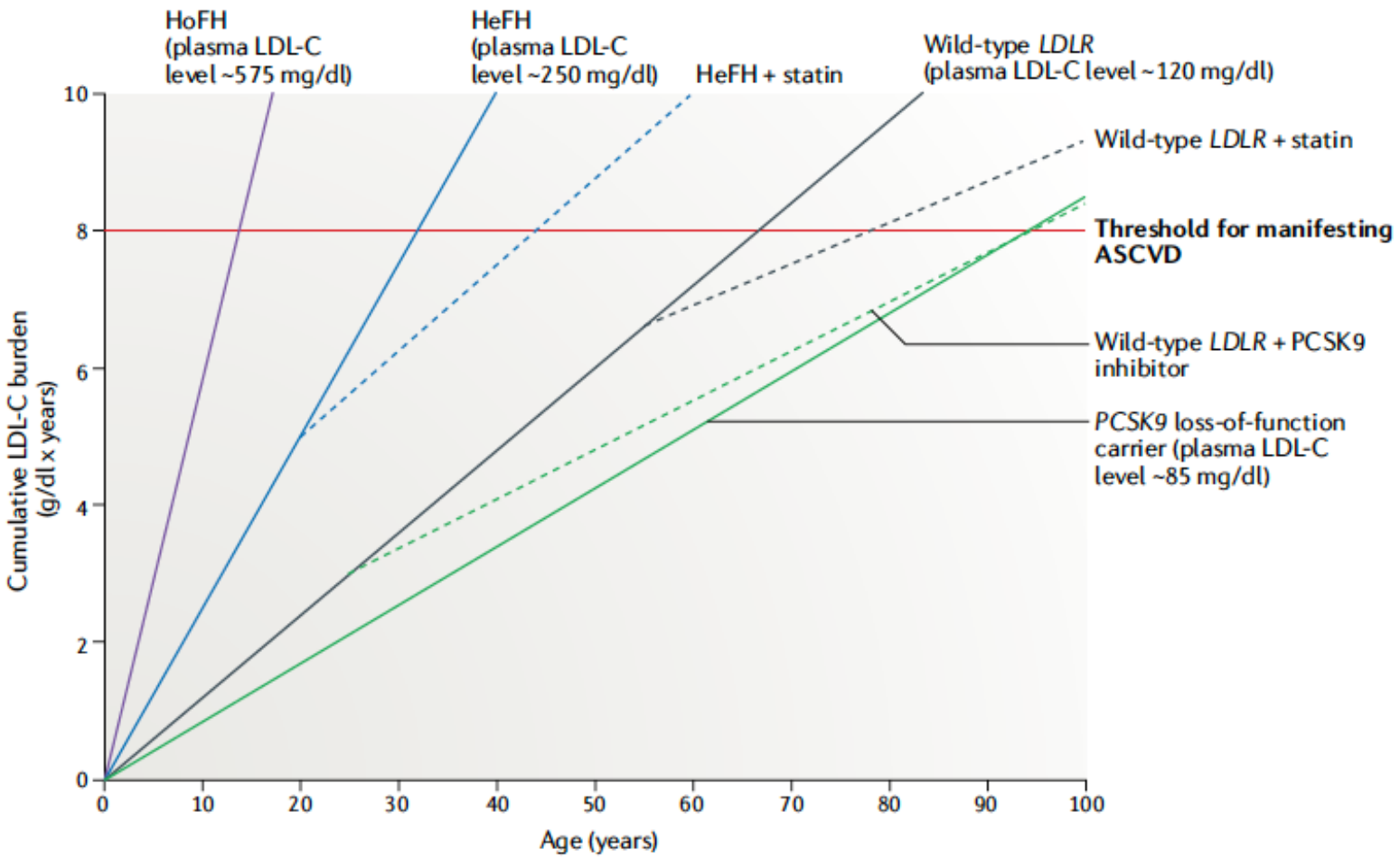
2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk (European Heart Journal 2019 -doi: 10.1093/eurheartj/ehz455)

ESC/EAS Leitlinien 2019

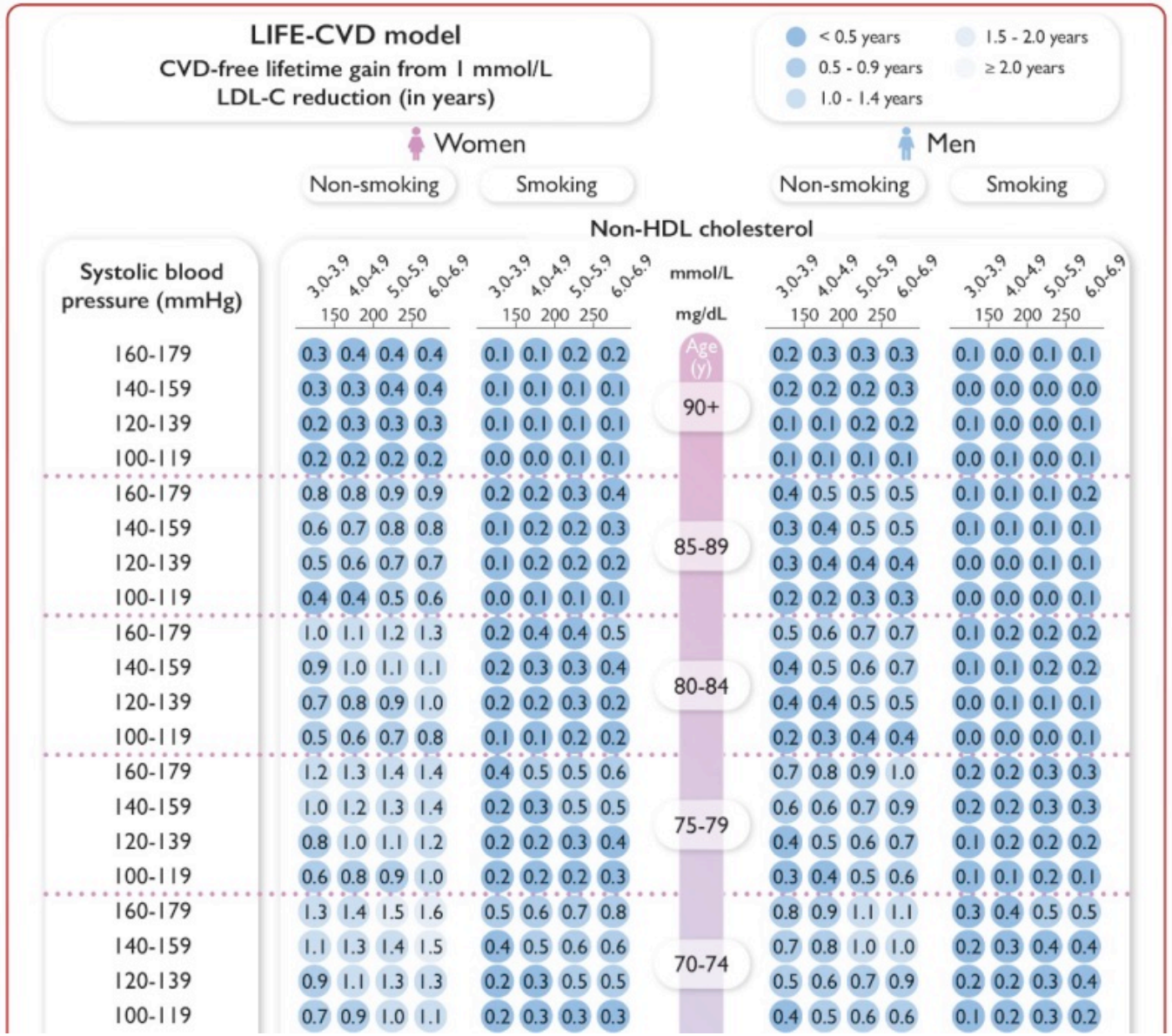


2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk, EHJ 2019; Rory Collins, Interpretation of the evidence for the efficacy and safety of statin therapy, Review, Lancet 08. September 2016; EASD 2016 Statins for All? N. Sattar and R. Kausik, 14. September 2016

”Time of exposure”

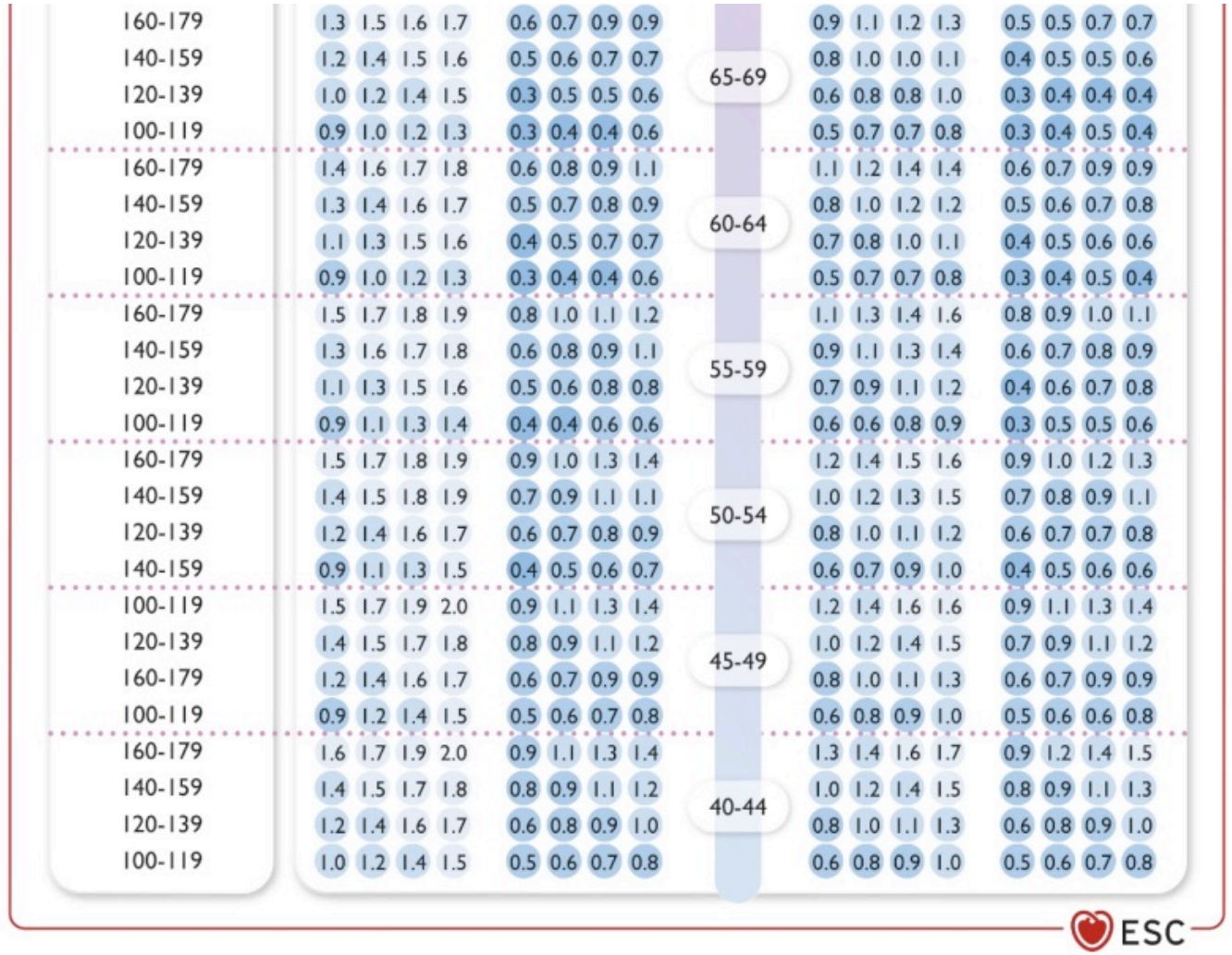


Sabatine M. Nature Reviews Cardiology 2019



Average years-free-of-cardiovascular disease gained per 1 mmol/L (40 mg/dL) LDL-C reduction in apparently healthy persons (1)

2021 ESC Guidelines on cardiovascular disease prevention in clinical practice
(European Heart Journal 2021 – doi:10.1093/eurheartj/ehab484)



160-179	1.3	1.5	1.6	1.7	0.6	0.7	0.9	0.9	65-69	0.9	1.1	1.2	1.3	0.5	0.5	0.7	0.7
140-159	1.2	1.4	1.5	1.6	0.5	0.6	0.7	0.7		0.8	1.0	1.0	1.1	0.4	0.5	0.5	0.6
120-139	1.0	1.2	1.4	1.5	0.3	0.5	0.5	0.6		0.6	0.8	0.8	1.0	0.3	0.4	0.4	0.4
100-119	0.9	1.0	1.2	1.3	0.3	0.4	0.4	0.6		0.5	0.7	0.7	0.8	0.3	0.4	0.5	0.4
160-179	1.4	1.6	1.7	1.8	0.6	0.8	0.9	1.1	60-64	1.1	1.2	1.4	1.4	0.6	0.7	0.9	0.9
140-159	1.3	1.4	1.6	1.7	0.5	0.7	0.8	0.9		0.8	1.0	1.2	1.2	0.5	0.6	0.7	0.8
120-139	1.1	1.3	1.5	1.6	0.4	0.5	0.7	0.7		0.7	0.8	1.0	1.1	0.4	0.5	0.6	0.6
100-119	0.9	1.0	1.2	1.3	0.3	0.4	0.4	0.6		0.5	0.7	0.7	0.8	0.3	0.4	0.5	0.4
160-179	1.5	1.7	1.8	1.9	0.8	1.0	1.1	1.2	55-59	1.1	1.3	1.4	1.6	0.8	0.9	1.0	1.1
140-159	1.3	1.6	1.7	1.8	0.6	0.8	0.9	1.1		0.9	1.1	1.3	1.4	0.6	0.7	0.8	0.9
120-139	1.1	1.3	1.5	1.6	0.5	0.6	0.8	0.8		0.7	0.9	1.1	1.2	0.4	0.6	0.7	0.8
100-119	0.9	1.1	1.3	1.4	0.4	0.4	0.6	0.6		0.6	0.6	0.8	0.9	0.3	0.5	0.5	0.6
160-179	1.5	1.7	1.8	1.9	0.9	1.0	1.3	1.4	50-54	1.2	1.4	1.5	1.6	0.9	1.0	1.2	1.3
140-159	1.4	1.5	1.8	1.9	0.7	0.9	1.1	1.1		1.0	1.2	1.3	1.5	0.7	0.8	0.9	1.1
120-139	1.2	1.4	1.6	1.7	0.6	0.7	0.8	0.9		0.8	1.0	1.1	1.2	0.6	0.7	0.7	0.8
140-159	0.9	1.1	1.3	1.5	0.4	0.5	0.6	0.7		0.6	0.7	0.9	1.0	0.4	0.5	0.6	0.6
100-119	1.5	1.7	1.9	2.0	0.9	1.1	1.3	1.4	45-49	1.2	1.4	1.6	1.6	0.9	1.1	1.3	1.4
120-139	1.4	1.5	1.7	1.8	0.8	0.9	1.1	1.2		1.0	1.2	1.4	1.5	0.7	0.9	1.1	1.2
160-179	1.2	1.4	1.6	1.7	0.6	0.7	0.9	0.9		0.8	1.0	1.1	1.3	0.6	0.7	0.9	0.9
100-119	0.9	1.2	1.4	1.5	0.5	0.6	0.7	0.8		0.6	0.8	0.9	1.0	0.5	0.6	0.6	0.8
160-179	1.6	1.7	1.9	2.0	0.9	1.1	1.3	1.4	40-44	1.3	1.4	1.6	1.7	0.9	1.2	1.4	1.5
140-159	1.4	1.5	1.7	1.8	0.8	0.9	1.1	1.2		1.0	1.2	1.4	1.5	0.8	0.9	1.1	1.3
120-139	1.2	1.4	1.6	1.7	0.6	0.8	0.9	1.0		0.8	1.0	1.1	1.3	0.6	0.8	0.9	1.0
100-119	1.0	1.2	1.4	1.5	0.5	0.6	0.7	0.8		0.6	0.8	0.9	1.0	0.5	0.6	0.7	0.8

ESC

Average years-free-of-cardiovascular disease gained per 1 mmol/L (40 mg/dL) LDL-C reduction in apparently healthy persons (2)

2021 ESC Guidelines on cardiovascular disease prevention in clinical practice
(European Heart Journal 2021 – doi:10.1093/eurheartj/ehab484)

Medizinische Vorgeschichte:

Adipositas

Bluthochdruck seit 7 Jahren

Diabetes mellitus Typ 2 seit 2 Jahren

Hypercholesterinämie

Medikation:

Ramipril 10 mg 1-0-0

Metformin 1000 mg 1-0-1

Dulaglutide 1,5 mg /Woche

Atorvastatin 80 mg 0-0-1

Ezetimibe 10 mg 0-0-1

ASS 100 mg 0-1-0

Ticagrelor 90 mg 1-0-1

Bisoprolol 2,5 mg 1-0-0

91 kg Körpergewicht

1,72 m Körpergröße

BMI: 30.8 kg/m^2

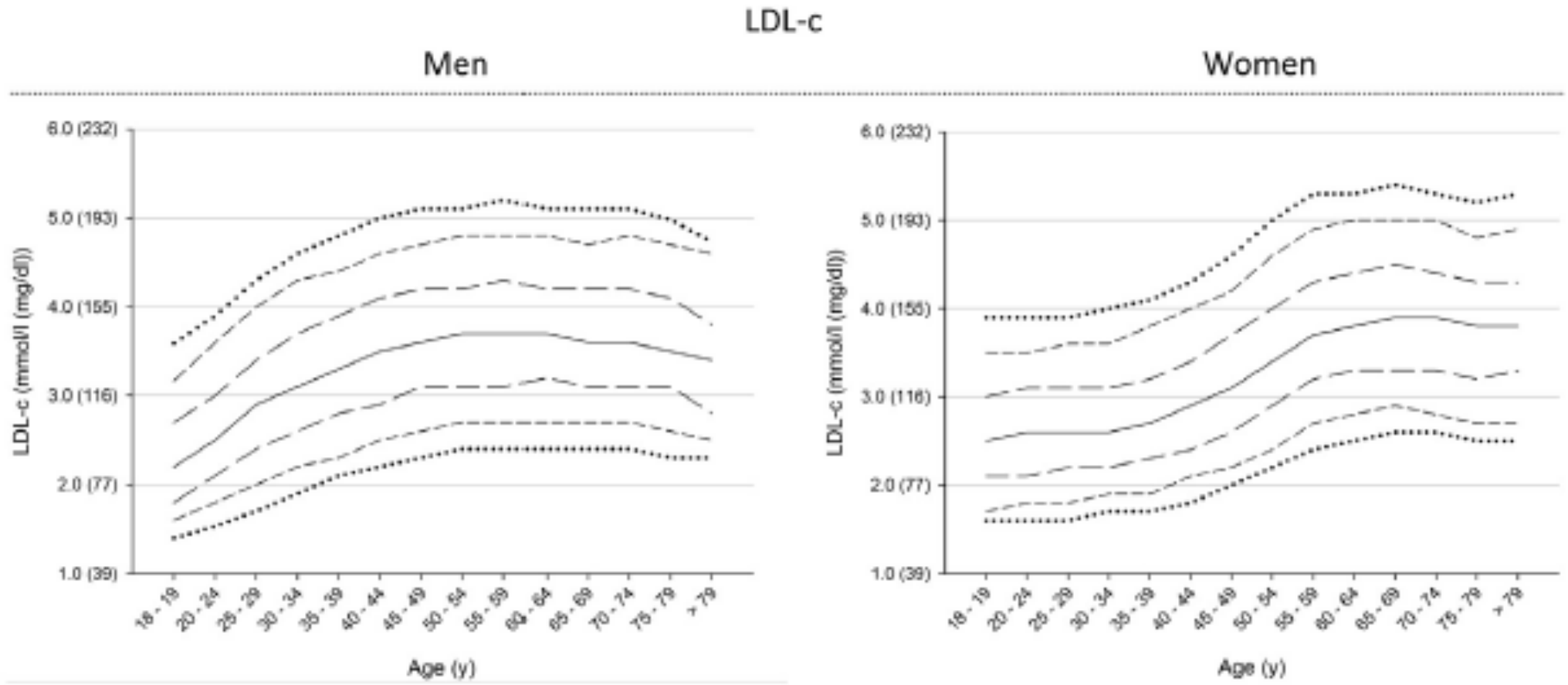
HbA1c 83 mmol/mol (9.7%)

LDL-C 203 mg/dl

eGFR 72 ml/min/1,73 m2

RR 142/92 mmHg

LDL-C Werte in der Bevölkerung



Balder JW. et al. J Clin Lipidol 2017

Dutch Lipid Clinic Network for the Diagnosis of familial hypercholesterolemia

Familienanamnese	Max. 2 Punkte
Verwandter 1. Grades mit vorzeitiger KHK (Männer < 55a, Frauen < 60a)	1
Verwandter 1. Grades mit LDL-C > 95. Perzentile (nach Alter/Geschlecht)	1
Verwandter 1. Grades mit tendinösem Xanthom und/oder Arcus corneae	2
Kind < 18 Jahre mit LDL >95. Perzentile (nach Alter/Geschlecht)	2
Klinische Anamnese	**Max. 2 Punkte**
Patient mit vorzeitiger KHK (Männer < 55a, Frauen < 60a)	2
Patient mit vorzeitiger CAVK/PAVK (Männer < 55a, Frauen < 60a)	1

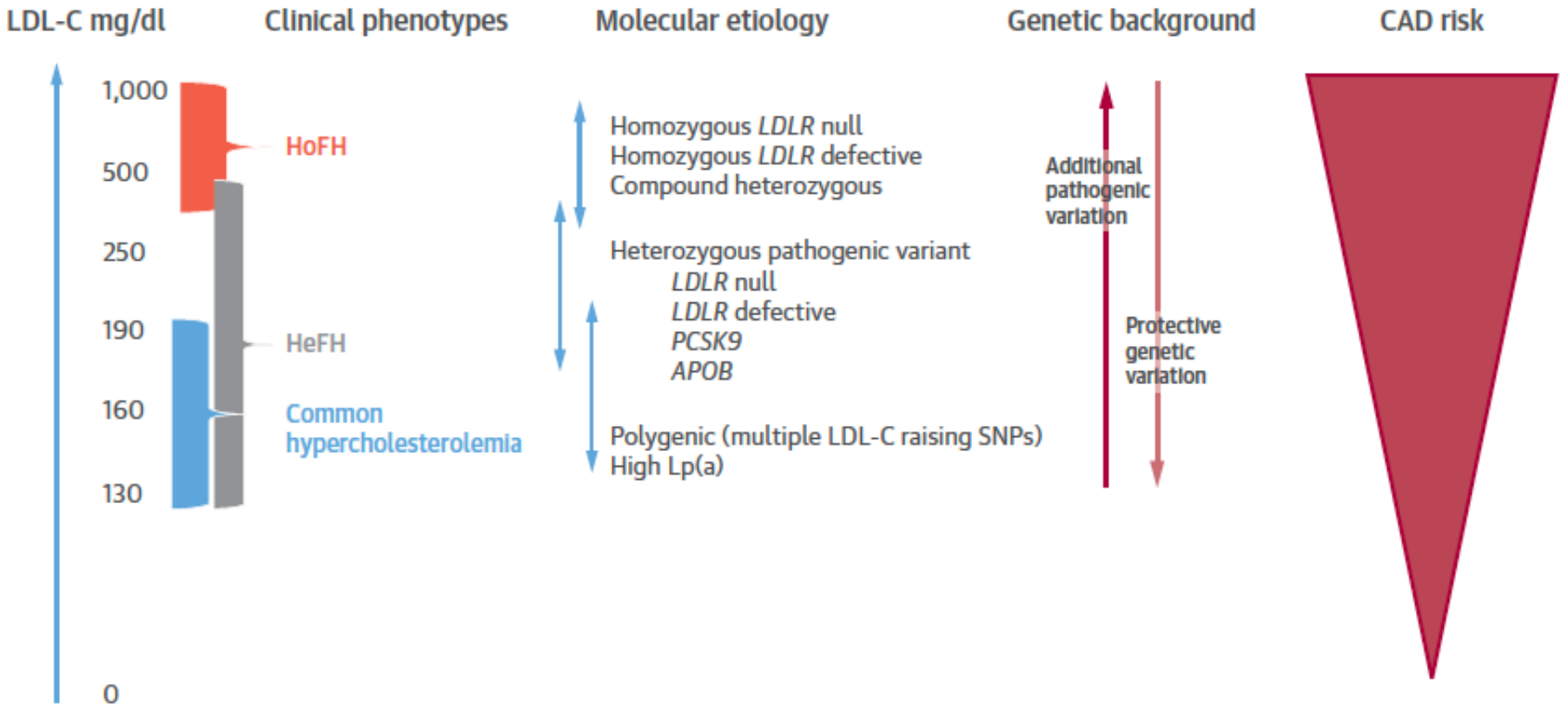
Klinische Untersuchung	Max. 6 Punkte
Patient mit tendinösem Xanthom	6
Patient mit Arcus corneae < 45a	4
LDL-C	**Max. 8 Punkte**
> 325 mg/dL	8
251–325 mg/dL	5
191–250 mg/dL	3
155–190 mg/dL	1
Molekulargenetische Untersuchung	**Max. 8 Punkte**
Krankheitsverursachende Mutation in LDL-R, ApoB, PCSK9	8

Score:

9-26 familiäre Hypercholesterinämie
6-8 wahrscheinliche fam. Hypercholesterinämie
3-8 mögliche fam. Hypercholesterinämie
0-2. unwahrscheinliche fam. Hypercholesterinämie

Nordestgaard BD et al. EHJ 2013

Familiäre Hypercholesterinämie



JACC Scientific Expert Panel, JACC 2018

Therapieoptionen

- Statine
- Ezetimibe

- PCSK9-Hemmer
- Inclisiran
- Bempedoinsäure

Intensity of lipid lowering treatment

Treatment	Average LDL-C reduction
Moderate intensity statin	≈ 30%
High intensity statin	≈ 50%
High intensity statin plus ezetimibe	≈ 65%
PCSK9 inhibitor	≈ 60%
PCSK9 inhibitor plus high intensity statin	≈ 75%
PCSK9 inhibitor plus high intensity statin plus ezetimibe	≈ 85%

ESC/EAS Guidelines 2019

Muskelsymptome unter Statinen

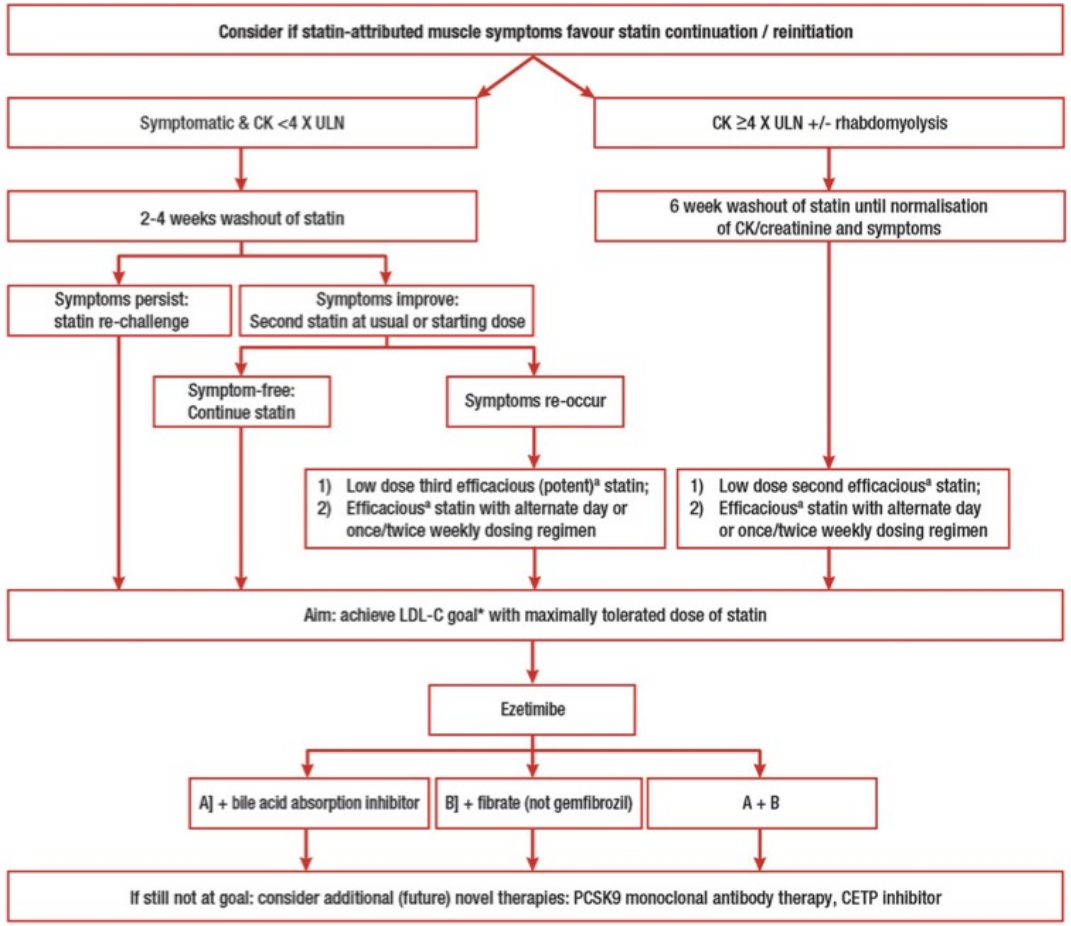
7 – 29% der PatientInnen klagen über Statin-assoziierte Muskelsymptome

1/1.000 -1/10.000 PatientInnen haben eine CK-Erhöhung > 10xULN

Zusätzliches Risiko für Rhabodmyolysen (CK >40 x ULN) unter Statinen:
1 per 10 000 (21 Studien, Standard Statin Dosis versus Kontrollen)
(14 *vs* 9 Fälle)

European Atherosclerosis Society Consensus Panel, EHJ 2015; CTT Lancet 2010

Vorgehen bei Muskelsymptomen unter Statinen

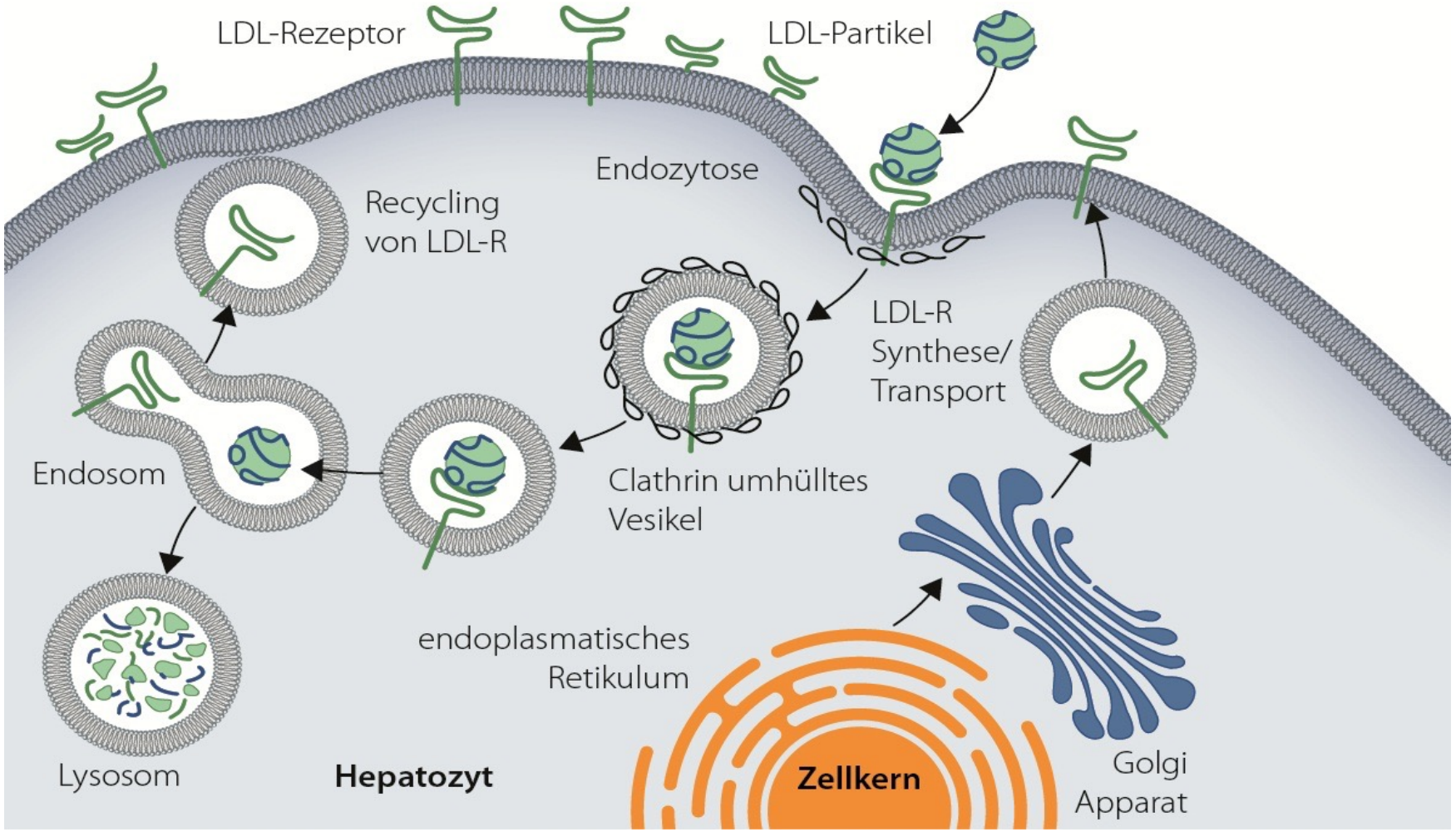


Key:

CETP = cholesteryl ester transfer protein; CK = creatine kinase; LDL-C = low-density lipoprotein cholesterol; PCSK9 = Proprotein convertase subtilisin/kexin type 9; ULN = upper limit of the normal range;

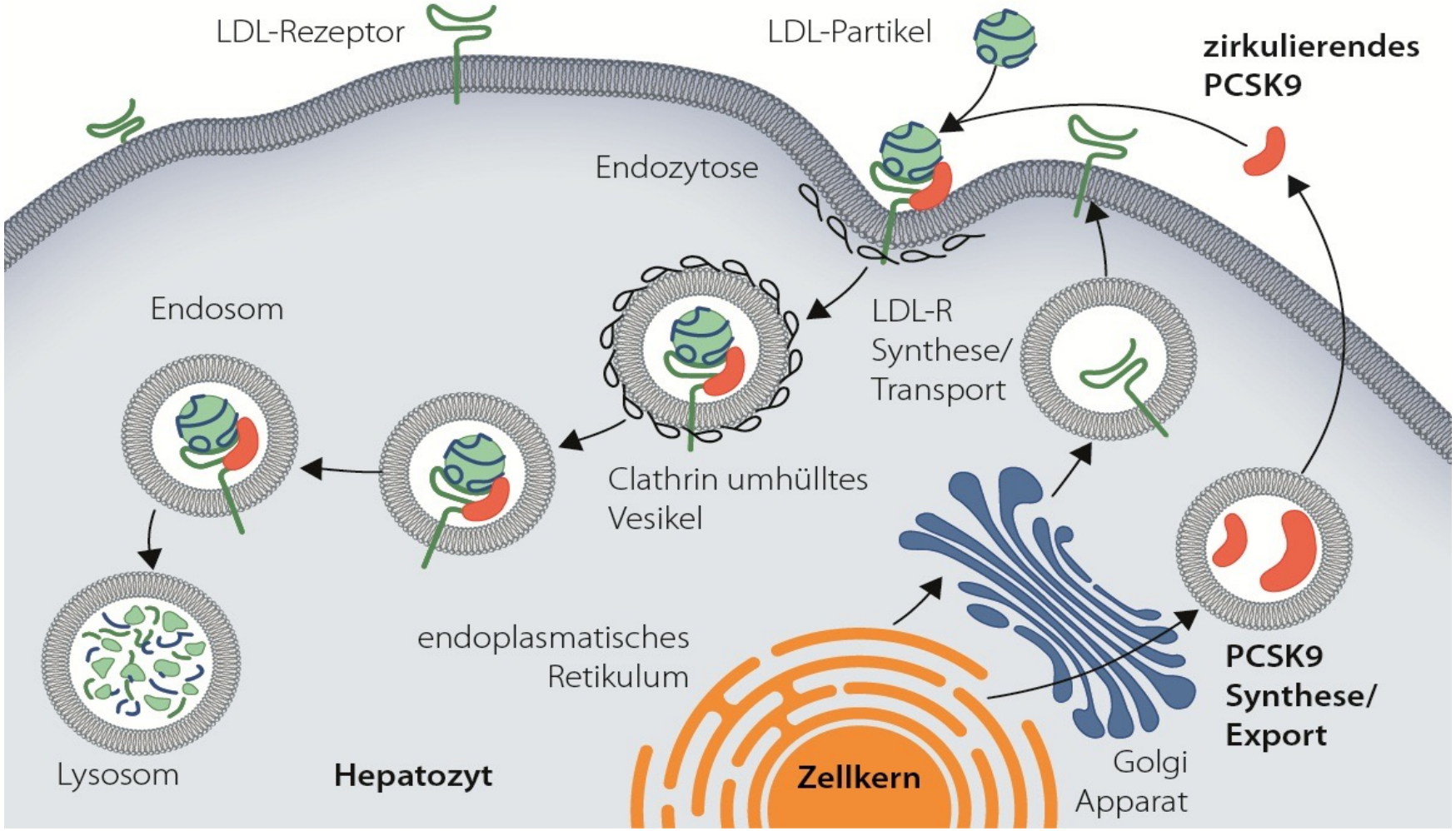
European Atherosclerosis Society Consensus Panel, EHJ 2015

LDL-Rezeptor Zyklus



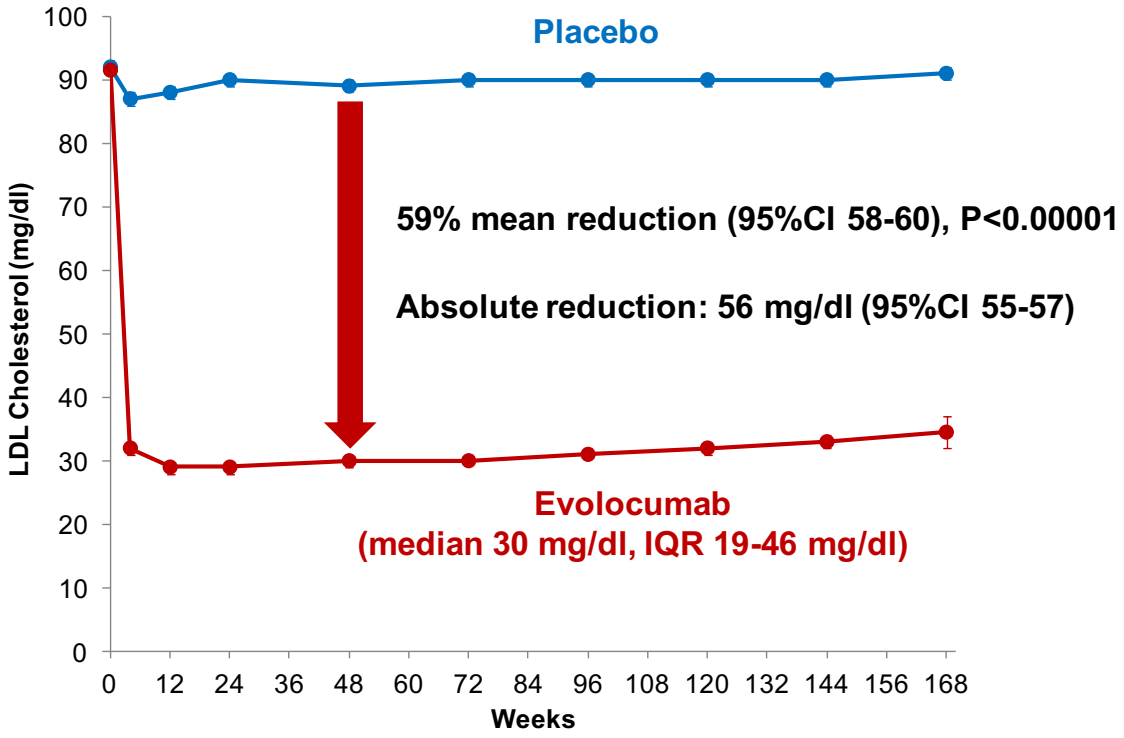
modifiziert nach: Lambert G, et al. The PCSK9 decade. J Lipid Res 2012 Dec;53(12):2515-24.

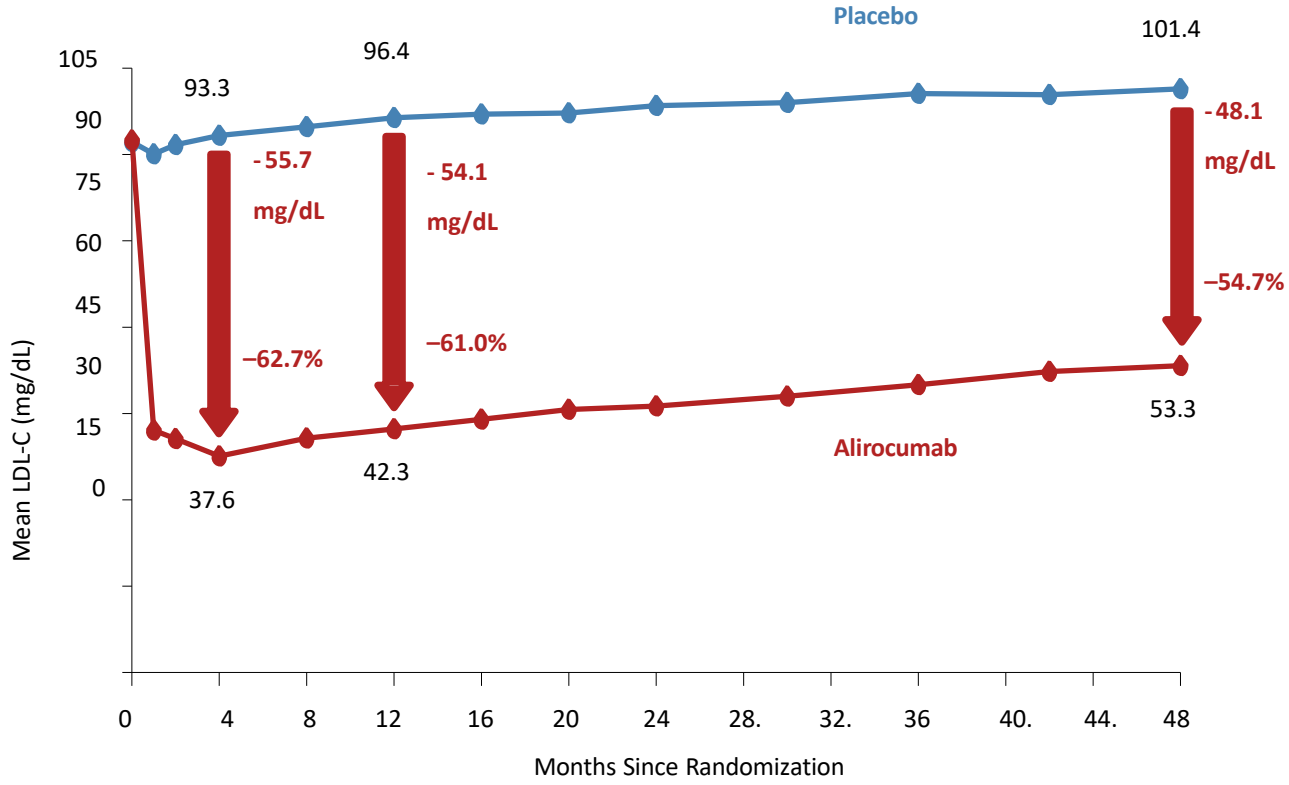
PCSK9 verhindert das Recycling des Rezeptors



modifiziert nach: Lambert G, et al. The PCSK9 decade. J Lipid Res 2012 Dec;53(12):2515-24.

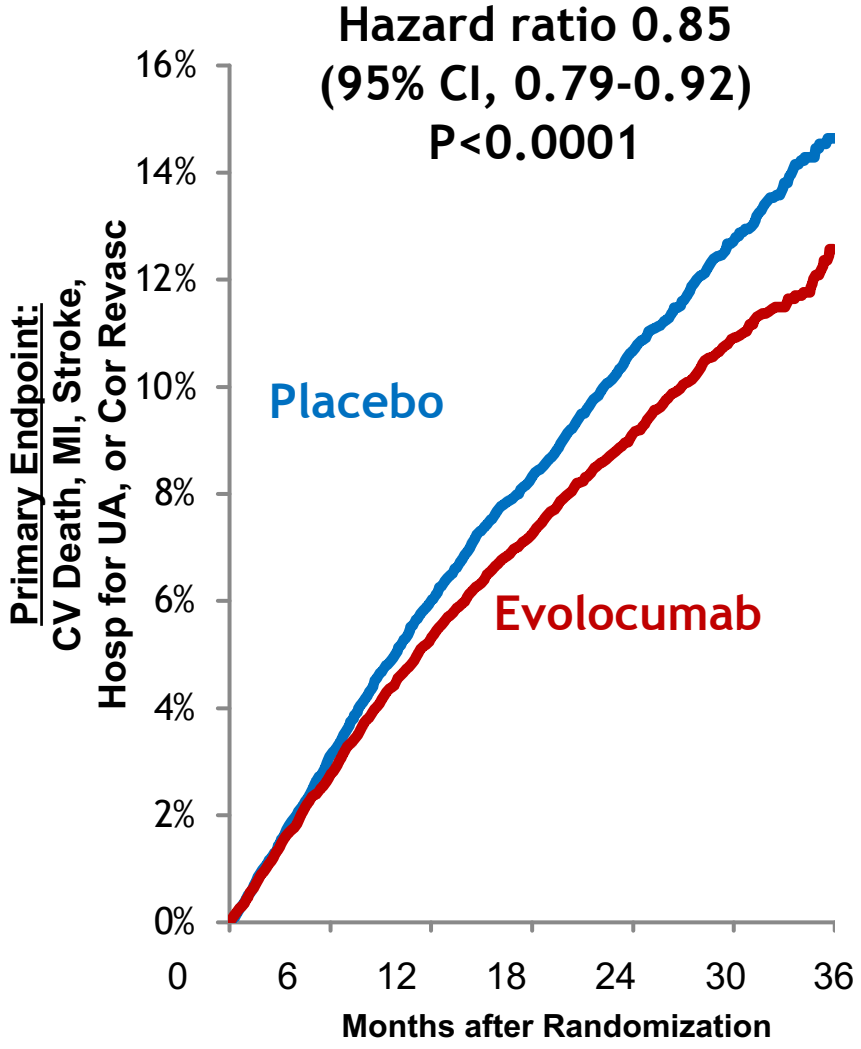
LDL-Cholesterinsenkung mit PCSK9-Hemmern

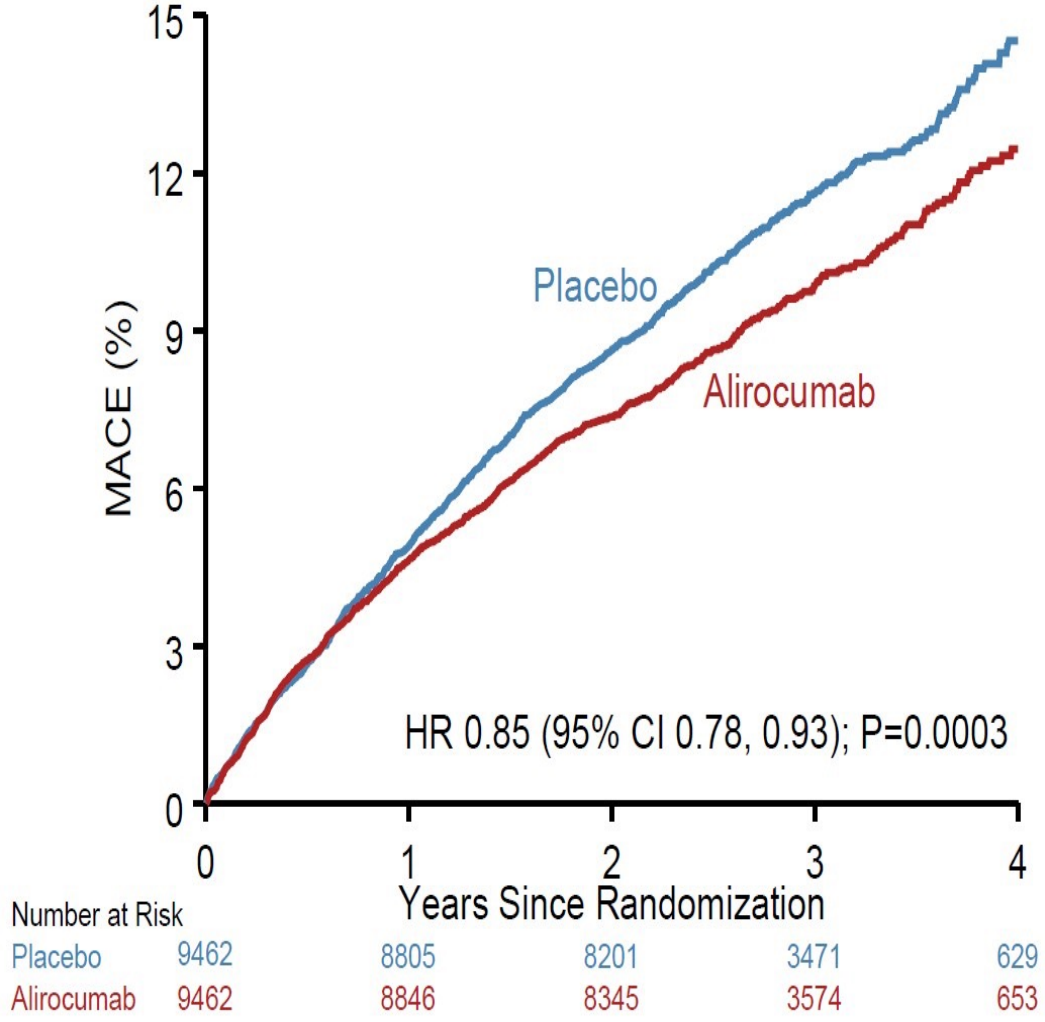




Sabatine MS et al. NEJM 2017;376:1713-1722

Primäre Endpunkte in FOURIER und ODYSSEY-OUTCOME



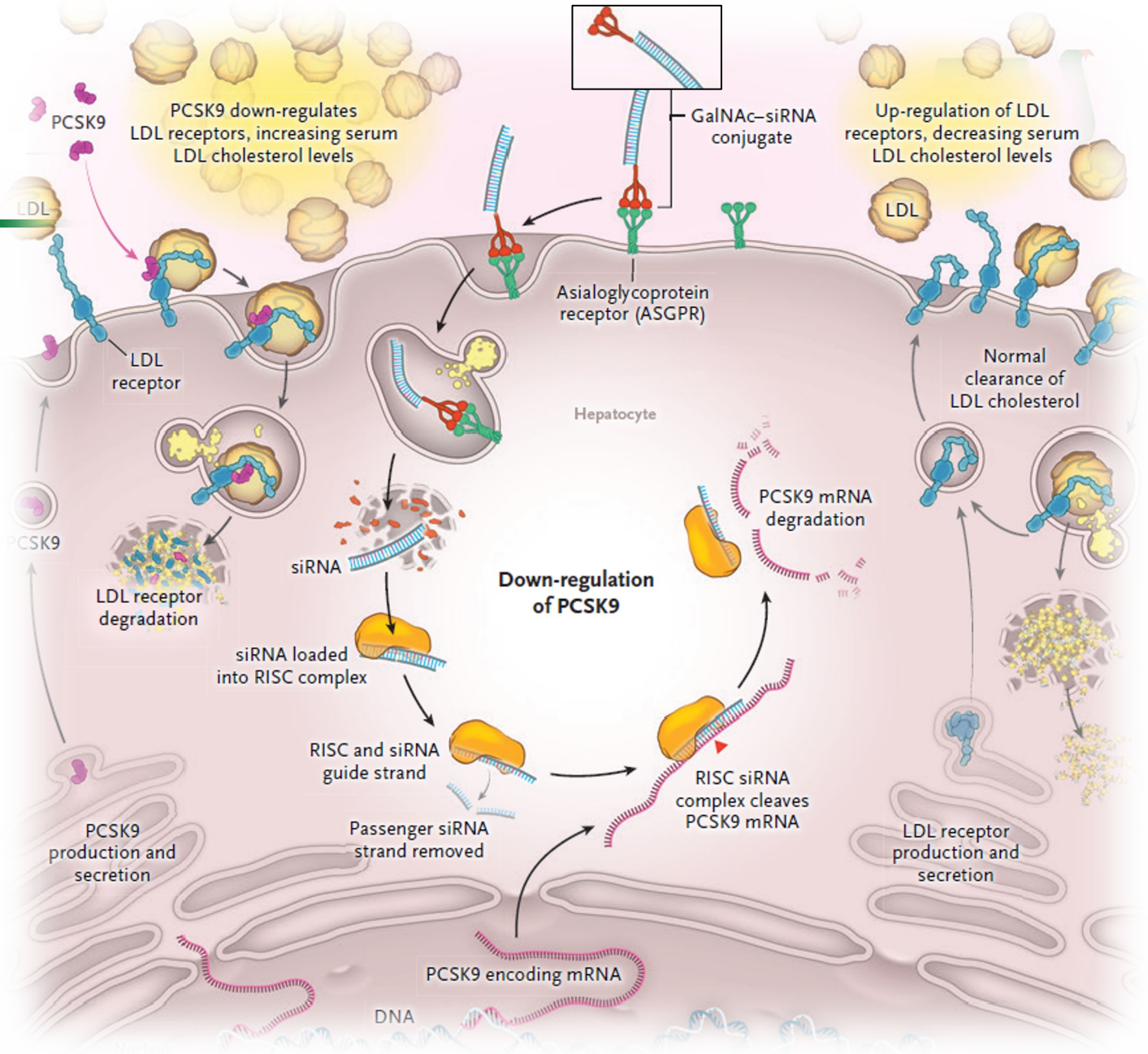


MACE: CHD death, non-fatal MI, ischemic stroke, or unstable angina requiring hospitalization

Sabatine MS et al. *NEJM* 2017;376:1713-1722; Schwartz GG et al. NEJM 2018

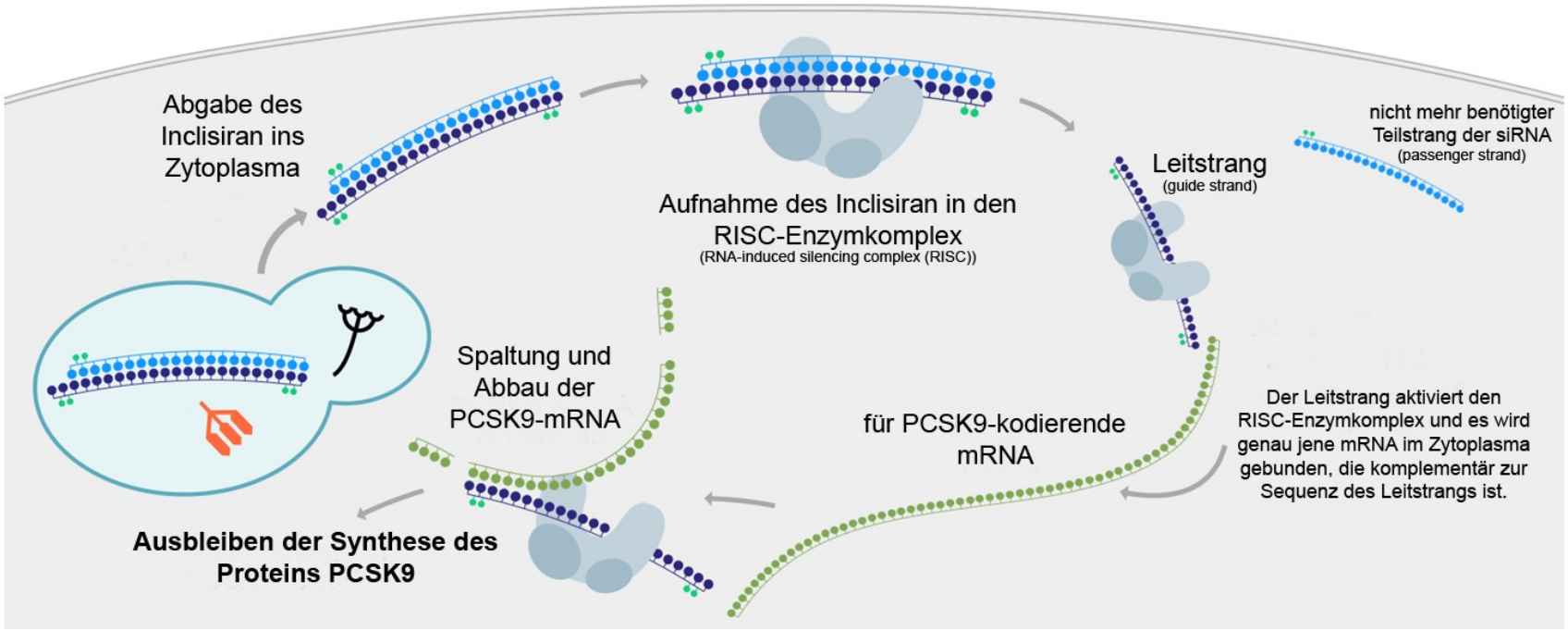
Inclisiran

- Die small interfering RNA (siRNA) Inclisiran wird durch seine GalNac-Modifikation spezifisch nur in Leberzellen aufgenommen
- In der Leber bindet Inclisiran als siRNA spezifisch die mRNA von PCSK9 und verhindert in diesem körpereignen, natürlichen Prozess die Synthese des PCSK9-Proteins
- Dadurch kommt es zu einer Hoch-Regulation des LDL-C-Rezeptors, wodurch wiederum der LDL-C-Spiegel im Blut gesenkt wird.



Khvorova (2017), NEJM, DOI: 10.1056/NEJMp1614154

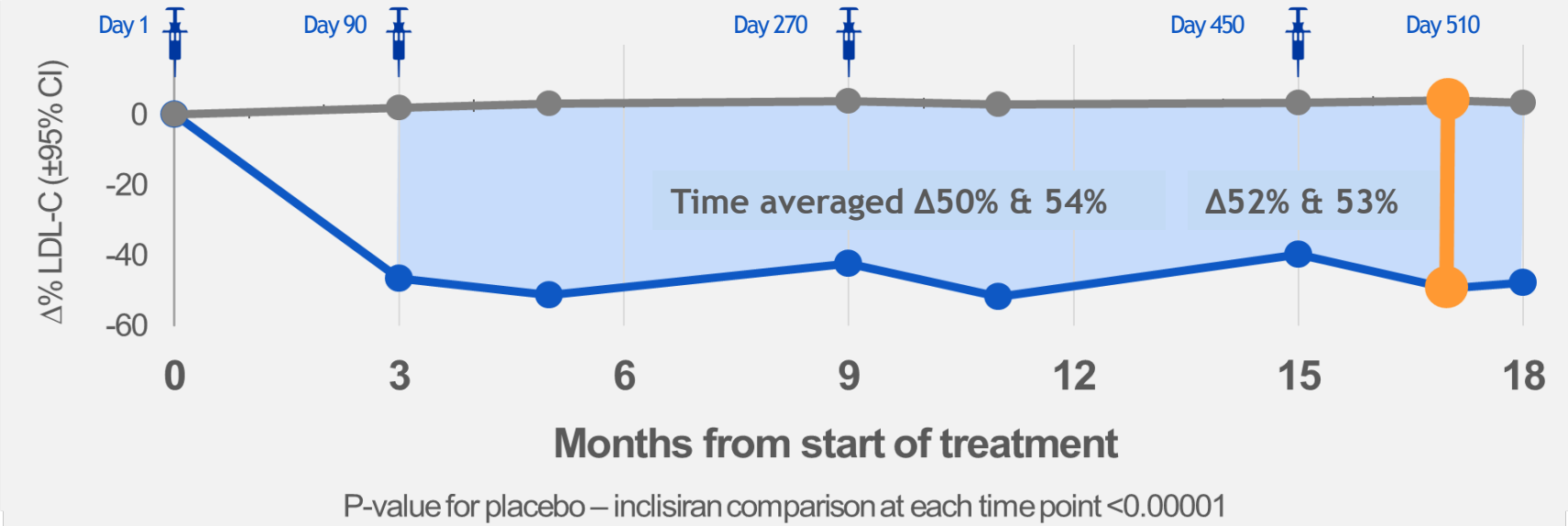
Wirkmechanismus



1. Wang N, et al. *Circ Res*. 2017;120:1063-1065. **2.** Springer AD, et al. *Nucleic Acid Ther*. 2018;28:109-118.
3. Khvorova A, et al. *N Engl J Med*. 2017;376:4-7. **4.** Tsouka AN, et al. *Curr Pharm Des*. 2018;24:3622-363

LDL-C Senkung mit Inclisiran

Gemittelte **LDL-C Senkung** (ORION 10 bzw. 11) über die Dauer von 18 Monaten:



All 95% confidence intervals are less than ±2% and therefore are not visible outside data points

Ray et al (2020), NEJM, DOI: 10.1056/NEJMoa1912387

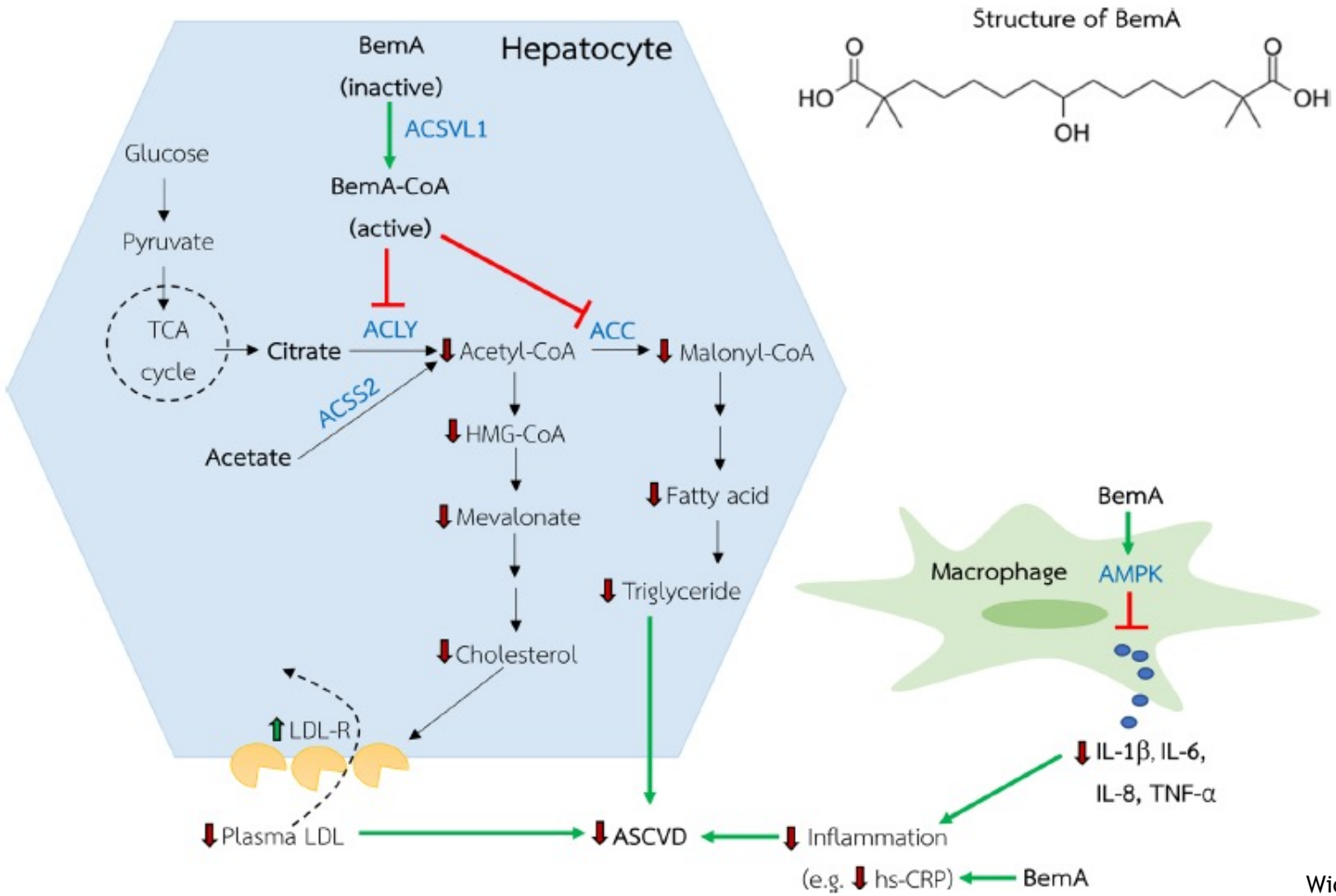
Sicherheitsprofil

Table 2. Adverse Events and Key Safety Laboratory Findings.*

Variable	ORION-10 Trial			ORION-11 Trial		
	Inclisiran (N=781)	Placebo (N=778)	Risk Ratio (95% CI)	Inclisiran (N=811)	Placebo (N=804)	Risk Ratio (95% CI)
	no. of patients (%)			*no. of patients (%)*		
Injection-site adverse events‡						
Any reaction	20 (2.6)	7 (0.9)	2.9 (1.2–6.7)	38 (4.7)	4 (0.5)	9.4 (3.4–26.3)
Mild	13 (1.7)	7 (0.9)	1.9 (0.7–4.6)	23 (2.8)	3 (0.4)	7.6 (2.3–25.2)
Moderate	7 (0.9)	0	—	15 (1.8)	1 (0.1)	14.9 (2.0–112.3)
Severe	0	0	—	0	0	—
Persistent	0	0	—	0	0	—
Laboratory results						
Liver function						
Alanine aminotransferase >3× ULN	2 (0.3)	2 (0.3)	1.0 (0.1–7.1)	4 (0.5)	4 (0.5)	1.0 (0.2–4.0)
Aspartate aminotransferase >3× ULN	4 (0.5)	5 (0.6)	0.8 (0.2–3.0)	2 (0.2)	4 (0.5)	0.5 (0.1–2.7)
Alkaline phosphatase >3× ULN	5 (0.6)	3 (0.4)	1.7(0.4–6.9)	1 (0.1)	2 (0.2)	0.5 (0.0–5.5)
Bilirubin >2× ULN	4 (0.5)	3 (0.4)	1.3 (0.3–5.9)	6 (0.7)	8 (1.0)	0.7 (0.3–2.1)
Kidney function: creatinine >2 mg/dl	30 (3.8)	30 (3.9)	1.0 (0.6–1.6)	5 (0.6)	11 (1.4)	0.5 (0.2–1.3)
Muscle: creatine kinase >5× ULN	10 (1.3)	8 (1.0)	1.2 (0.5–3.1)	10 (1.2)	9 (1.1)	1.1 (0.5–2.7)
Hematology: platelet count <75×10^9/liter	1 (0.1)	0	—	0	1 (0.1)	—

Ray et al (2020), NEJM, DOI: 10.1056/NEJMoa1912387

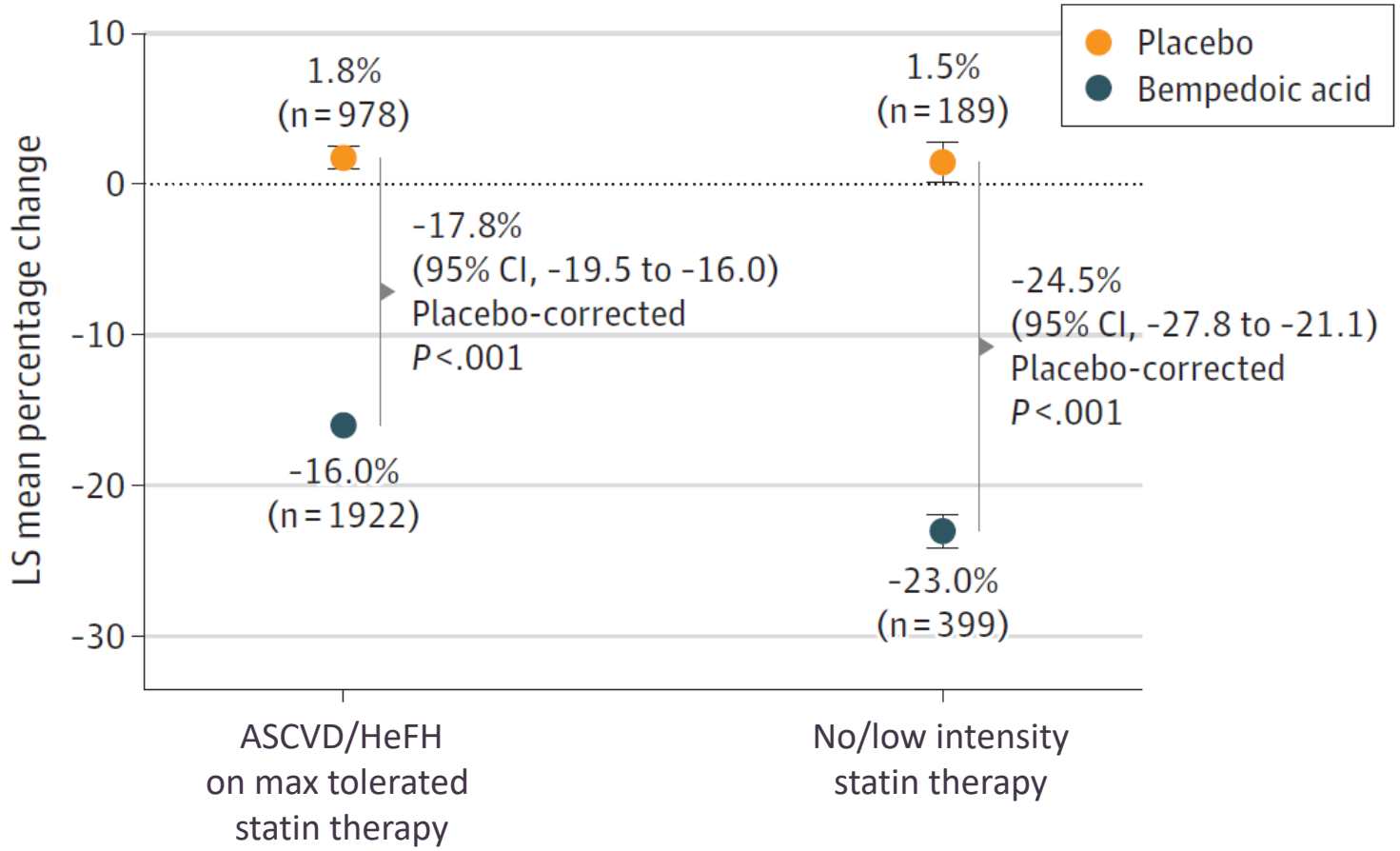
Bempedoinsäure



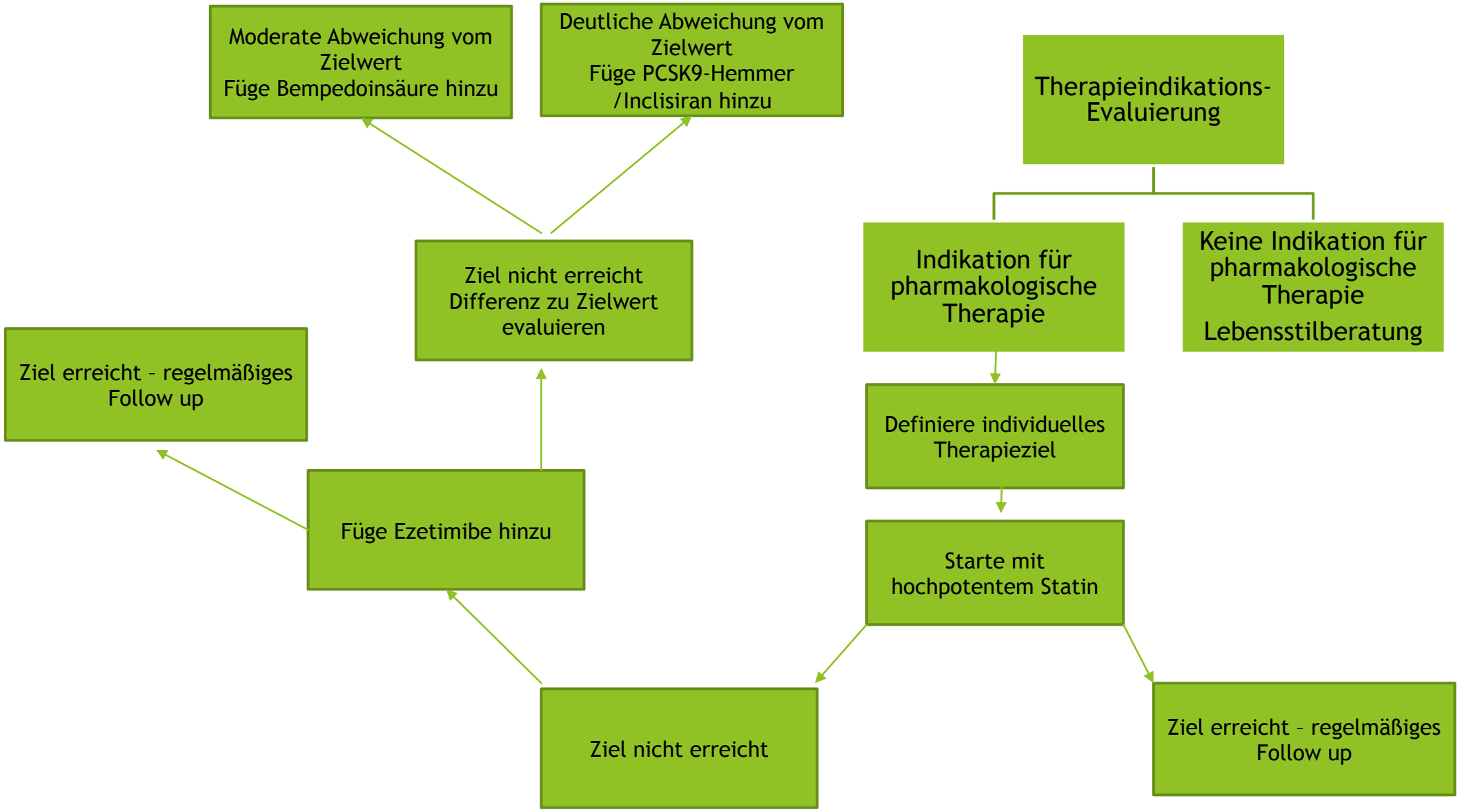
Wichaiyo S. & Supharattanasitthi W. Clinical Drug Investig 2021

Bempedoinsäure gepoolte Analyse

Reduktion nach 12 Wochen



Banach et al. JAMA Cardiology 2020 doi: 10.1001/jamacardio.2020.2314. Online ahead of print

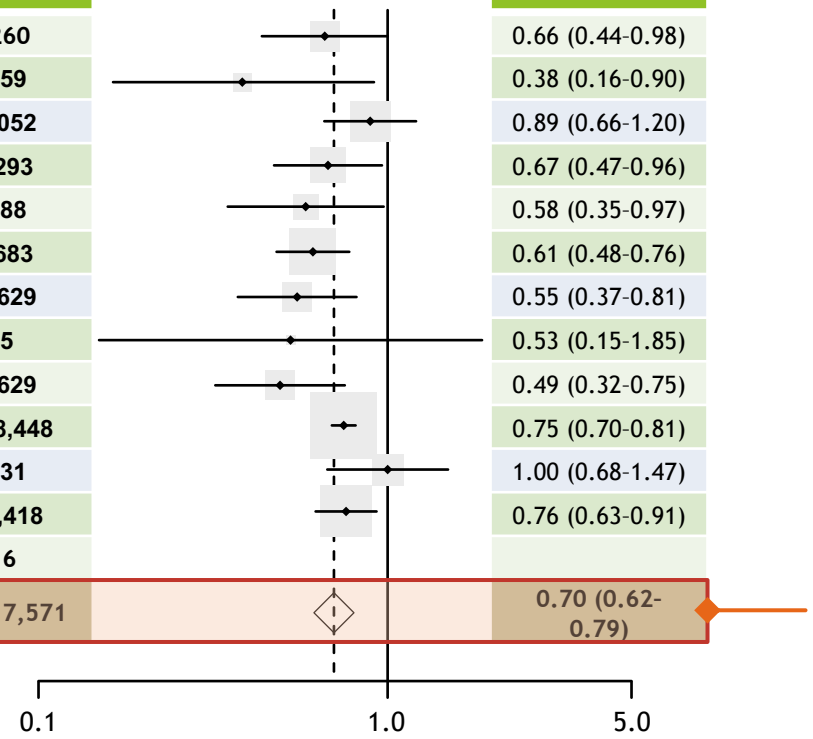

Therapieindikations-Evaluierung
Indikation für pharmakologische Therapie
Keine Indikation für pharmakologische Therapie
Lebensstilberatung
Definiere individuelles Therapieziel
Starte mit hochpotentem Statin
Ziel erreicht – regelmäßiges Follow up
Ziel nicht erreicht
Füge Ezetimibe hinzu
Ziel erreicht – regelmäßiges Follow up
Ziel nicht erreicht Differenz zu Zielwert evaluieren
Moderate Abweichung vom Zielwert Füge Bempedoinsäure hinzu
Deutliche Abweichung vom Zielwert Füge PCSK9-Hemmer /Inclisiran hinzu

Spezielle Populationen

Meta-Analyse: CKD ohne Dialyse

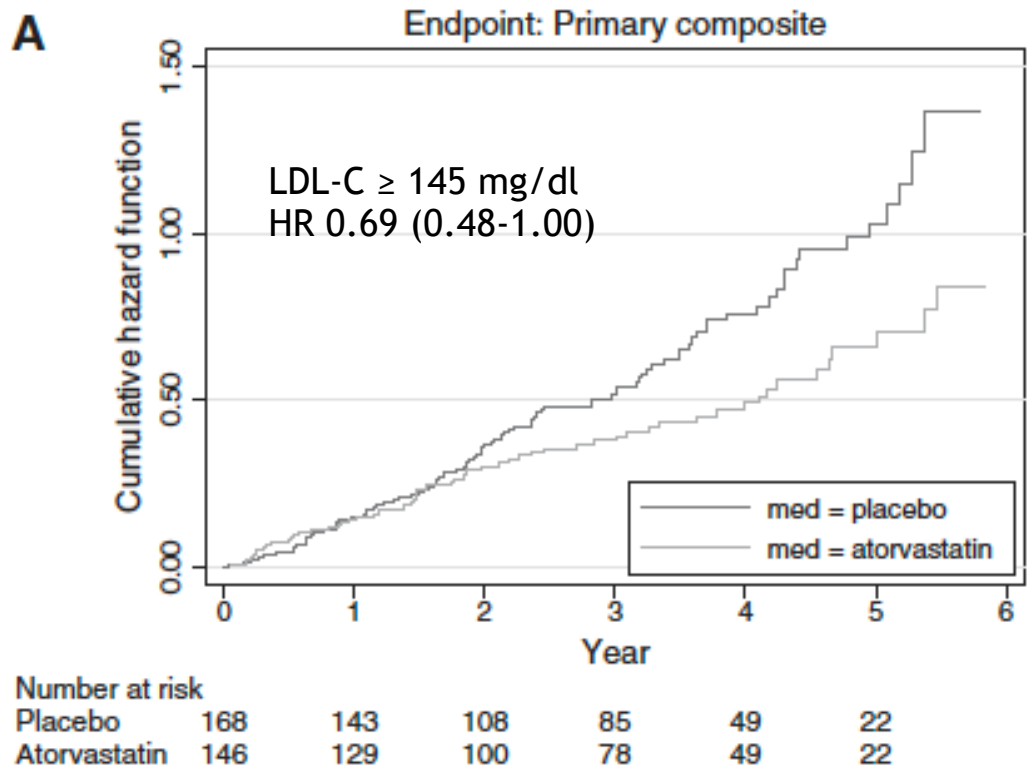
Major CV events in patients with CKD

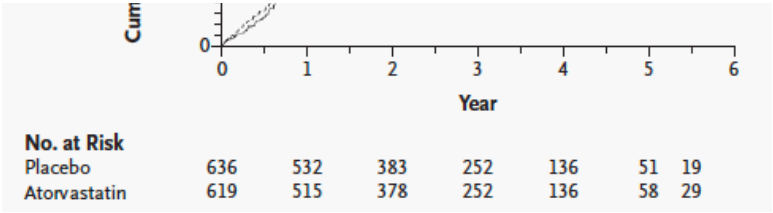
Study, year	Events/patients		Odds ratio (95% CI)
	Statin	Control	
4S 2009	53 / 245	77 / 260	0.66 (0.44-0.98)
AFCAPS/TexCAPS 2010	8 / 145	21 / 159	0.38 (0.16-0.90)
ALERT 2003	88 / 1,050	98 / 1,052	0.89 (0.66-1.20)
ALLIANCE 2009	78 / 286	105 / 293	0.67 (0.47-0.96)
CARDS 2009	25 / 482	42 / 488	0.58 (0.35-0.97)
HPS2003	182 / 646	268 / 683	0.61 (0.48-0.76)
JUPITER 2011	40 / 1,638	71 / 1,629	0.55 (0.37-0.81)
LORD 2010	4 / 58	8 / 65	0.53 (0.15-1.85)
MEGA 2006	33 / 1,507	71 / 1,629	0.49 (0.32-0.75)
PPP 2005	1,590 / 8,376	2,011 / 8,448	0.75 (0.70-0.81)
PREVEND IT 2006	60 / 431	60 / 431	1.00 (0.68-1.47)
SHARP 2014	217 / 2,396	281 / 2,418	0.76 (0.63-0.91)
Sawara 2008	0 / 22	0 / 16	
Overall (I^2 = 33.3%)	2,378 / 17,282	3,113 / 17,571	0.70 (0.62-0.79)



Su X, et al. Am J Kidney Dis. 2016;67:881-892.

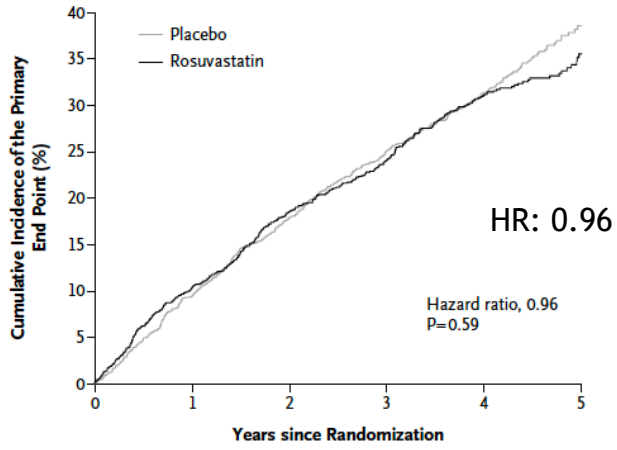
Lipidstudien bei ESRD - RCTs





AURORA-Studie

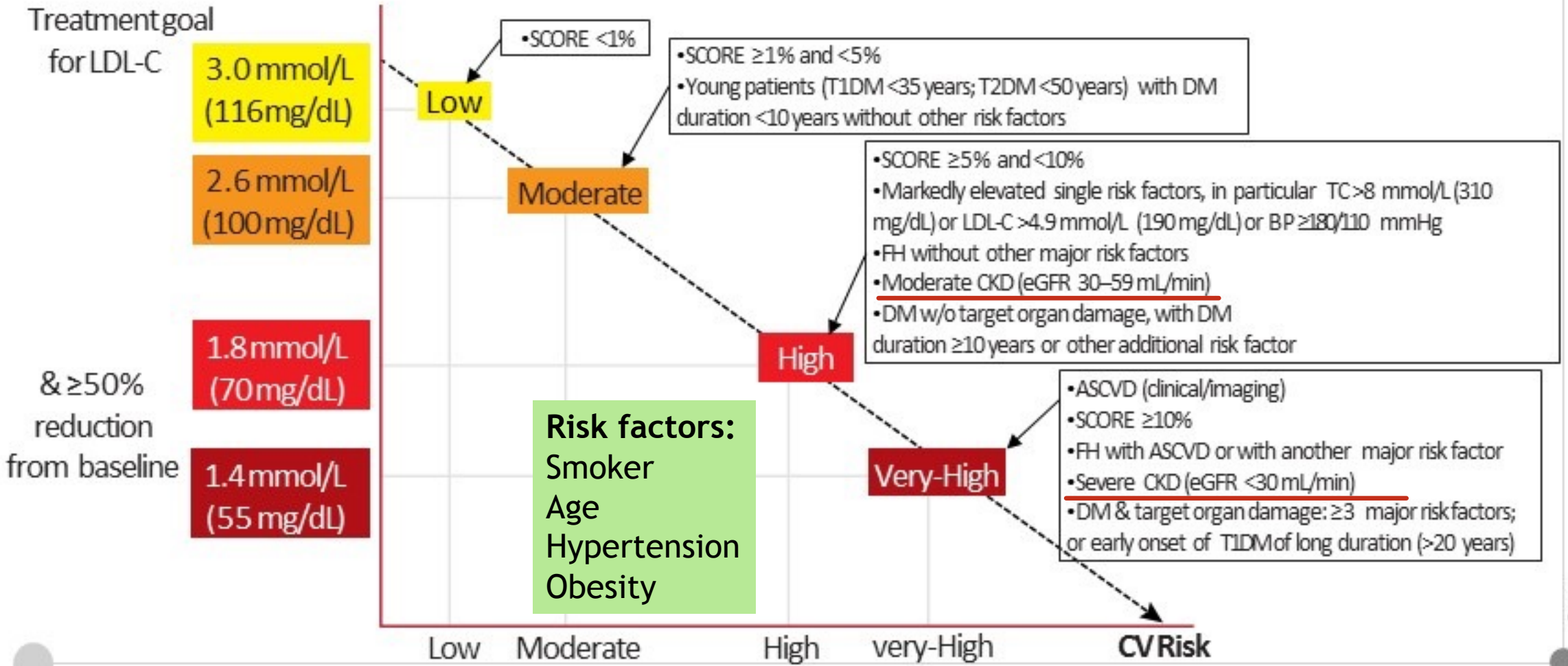
- 2776 Patienten mit Hämodialyse
- Rosuvastatin 10 mg vs. Placebo
- Ausgangs-LDL-C: 100 ± 35 vs. 99 ± 34 mg/dl
- Primärer Endpunkt:
  CV Tod, Schlaganfall, Myokardinfarkt



HR: 0.96 (95%-CI: 0.84-1.11)

Wanner C. et al. NEJM 2005; Fellstrom BC et al. NEJM 2009; März W. et al. Clin J Am Soc Nephrol 2011

Central Illustration Upper panel Treatment goals for low-density lipoprotein cholesterol (LDL-C) across categories of total cardiovascular disease risk



www.escardio.org/guidelines

2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk (European Heart Journal 2019 -doi: 10.1093/eurheartj/ehz455)

KIDIGO – Empfehlungen 2013

In adults with newly identified CKD (including those treated with chronic dialysis or kidney transplantation), we recommend evaluation with a lipid profile (total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides). (1C)

In adults with CKD (including those treated with chronic dialysis or kidney transplantation), follow-up measurement of lipid levels is not required for the majority of patients. (Not Graded)

In adults aged ≥50 years with eGFR <60 ml/min/1.73 m2 but not treated with chronic dialysis or kidney transplantation (GFR categories G3a-G5), we recommend treatment with a statin or statin/ ezetimibe combination. (1A)

In adults aged ≥ 50 years with CKD and eGFR >60 ml/min/1.73 m2 (GFR categories G1-G2) we recommend treatment with a statin. (1B)

In adults with dialysis-dependent CKD, we suggest that statins or statin/ezetimibe combination not be initiated. (2A)
In patients already receiving statins or statin/ezetimibe combination at the time of dialysis initiation, we suggest that these agents be continued. (2C)

Patient

Ambulanz für Diabetes und Stoffwechselkrankheiten
Leiter: Assoz.Prof. PD Dr. Harald Sourij

Tel. +43 (316) 385-13270
Fax +43 (316) 385-14332
diabetes.ambulanz@klinikum-graz.at

JA	NEIN	ZUM ANKREUZEN MIT BESCHREIBUNG ZUR VERVOLLSTÄNDIGUNG				
☐	☐	Primäre Hypercholesterinämie; Sekundärprävention nach akutem kardiovaskulären Ereignis				
		☐ **KHK:**	☐ Myokardinfarkt Datum_____	☐ PTA/Stent/CABG Datum_____	☐ andere _____	
		☐ **zAVK:**	☐ Insult Datum_____	☐ PTA/Stent/PTA Datum_____	☐ andere _____	
		☐ **pAVK:**	☐ Amputation Datum_____	☐ PTA/Stent/Bypass Datum_____	☐ andere _____	
☐	☐	Sehr hohes kardiovaskuläres Risiko (≥10% gemäß ESC-Leitlinie)				
☐	☐	LDL Cholesterin ≥ 100 mg/dl unter maximal tolerierbarer Kombinationstherapie				
		LDL-C_____mg/dl am _____				
		Unter folgender Therapie: Substanz(en)_____, Dosis_____				
☐	☐	Ezetrol wurde versucht				
		Es bestehen Unverträglichkeiten gegen:				
		Substanz	Max. Dosis (mg)	Myalgien	Hepatopathie	Max. CK/ALT-Wert (U/l)
		Atorvastatin		☐	☐	
		Rosuvastatin		☐	☐	
		Simvastatin		☐	☐	
		Pravastatin		☐	☐	
		_____		☐	☐	
Zusätzliche Information:						

Die/der Patientin wird von unserer Ambulanz bezüglich eines Termines verständigt werden.

LKH-Univ.Klinikum Graz

Universitätsklinik für Innere Medizin
Klinikvorstand: Univ.-Prof. Dr. Alexander Rosenkranz
A-8036 Graz, Auenbruggerplatz 15, Tel.: +43(0)316 385 - 12383, Fax: +43(0)316 385 - 13428

Steiermärkische Krankenanstaltengesellschaft m.b.H. *Medizinische Universität Graz*

Patient

Klinische Abteilung für Kardiologie
Leiter: Univ.-Prof. Dr. Andreas Zirlik

Klinische Abteilung für Endokrinologie und Diabetologie
Leiter: Univ.-Prof. Dr. Thomas Pieber

Zuweisung metabolisch-kardiologische Hochrisikoambulanz

JA	NEIN	ZUM ANKREUZEN MIT BESCHREIBUNG ZUR VERVOLLSTÄNDIGUNG			
☐	☐	Atherosklerotische Erkrankung vorliegend			
		☐ **KHK:**	☐ Myokardinfarkt Datum_____	☐ PCI/CABG Datum_____	☐ andere _____
		☐ **zAVK:**	☐ Insult/TIA Datum_____	☐ PTA/Stent/Carotis-OP Datum_____	☐ andere _____
		☐ **pAVK:**	☐ Amputation Datum_____	☐ PTA/Stent/Bypass Datum_____	☐ andere _____
		☐ **Herzinsuffizienz**		Weitere Details, wenn vorhanden: _____	
☐	☐	LDL Cholesterin über dem individuellen Ziel (Zielwert: _____) trotz maximal tolerierbarer Kombinationstherapie			
		LDL-C_____mg/dl am _____			
		Unter folgender Therapie: Substanz(en)_____, Dosis_____			
☐	☐	Intoleranz gegenüber Statinen: Wenn ja, welche(s) Präparat(e)? _____ Welche Intoleranz? _____			
☐	☐	Blutdruckkontrolle unzureichend (trotz antihypertensiver Kombinationstherapie)			
☐	☐	Diabetes mellitus vorliegend und Blutzuckerkontrolle unzureichend			
☐	☐	Weitere kardiovaskuläre Risikofaktoren ☐ Lp(a)-Erhöhung ☐ Albuminurie ☐ Rauchen ☐ GFR _____			
Zusätzliche Information zum Grund der Vorstellung:					

Med Uni Graz

Danke

Medical
University of Graz

Interdisciplinary Metabolic Medicine
Trials Unit