Lowering Cholesterol.

Saving Lives.

Tackling Cholesterol Together

TheAHSNNetwork

ACCELERATED ACCESS COLLABORATIVE

HEART UK THE CHOLESTEROL CHARITY

NHS

UNIVERSITY OF LEEDS

The Leeds Teaching Hospitals NHS Trust

Management of lipids after a cardiovascular event

Welcome to the sixth in a series of webinars as part of the national education programme Tackling Cholesterol Together.

Delivered in partnership by The NHS Accelerated Access Collaborative (AAC), The AHSN Network and the cholesterol charity, HEART UK

The webinar will start at 12 noon

November 2021

All programme content, recordings and next webinar and clinic bookings will be housed in the HEART UK pages. Visit the site for the **new** e-Learning modules on diet, launching late November https://www.heartuk.org.uk/tackling-cholesterol-together/home

Housekeeping

- **This meeting will be recorded** and will be made available in the HEART UK Tackling Cholesterol Together pages
- **There will be time** to stop and ask questions at the end of the webinar
- **Feel free to ask questions** or upvote questions in the chat function when it becomes available
- **Any questions that we are not able to cover in the Q&A** sections today will be addressed following the event
- **Any questions you provided** during registration will be covered during the session

Agenda

	Topic	Presenter
01	**Welcome**	Sue Critchley
02	**Influence of LDL-C on risk after a cardiovascular event**	Professor Stephen Wheatcroft
03	**Considerations for Optimising Post CVD Lipids Management**	Dr Rani Khatib
04	**Lipid Optimisation in Peripheral Arterial Disease**	Mr Marc Bailey
05	**Q&A. Close and next steps**	Sue Critchley

Objectives of today's webinar

01

Understand the scale of CVD in the UK. **Review** evidence on the influence of LDL-C on CVD risk and the impact of intensive lipid lowering therapy.

02

After a cardiovascular event, **identify** the increased risk for another cardiovascular event over time and according to individual age and risk category.

03

Consider the NICE CG181 and ESC targets approach to managing cholesterol in secondary prevention. Review the barriers and **find** opportunities to optimise, including a review of statin reluctance and intolerance.

04

Referring to NICE CG147, **appreciate** the clinical presentation of Peripheral Artery Disease as a high risk for cardiovascular events (and the indication for a secondary prevention approach).

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02

Influence of LDL-C on risk after a cardiovascular event

Stephen Wheatcroft

Professor of Cardiometabolic Medicine / Consultant Cardiologist

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NHS Trust

Cardiovascular disease in the UK - the scale of the problem

Cardiovascular diseases cause a quarter of all deaths in the UK;

>160 000 deaths each year;

~460 deaths each day; one every three minutes

7.6 million people in the UK are living with cardiovascular disease

>100 000 hospital admissions each year with heart attacks

Every 5 minutes, one person has a stroke

>100 000 people in the UK with stroke each year

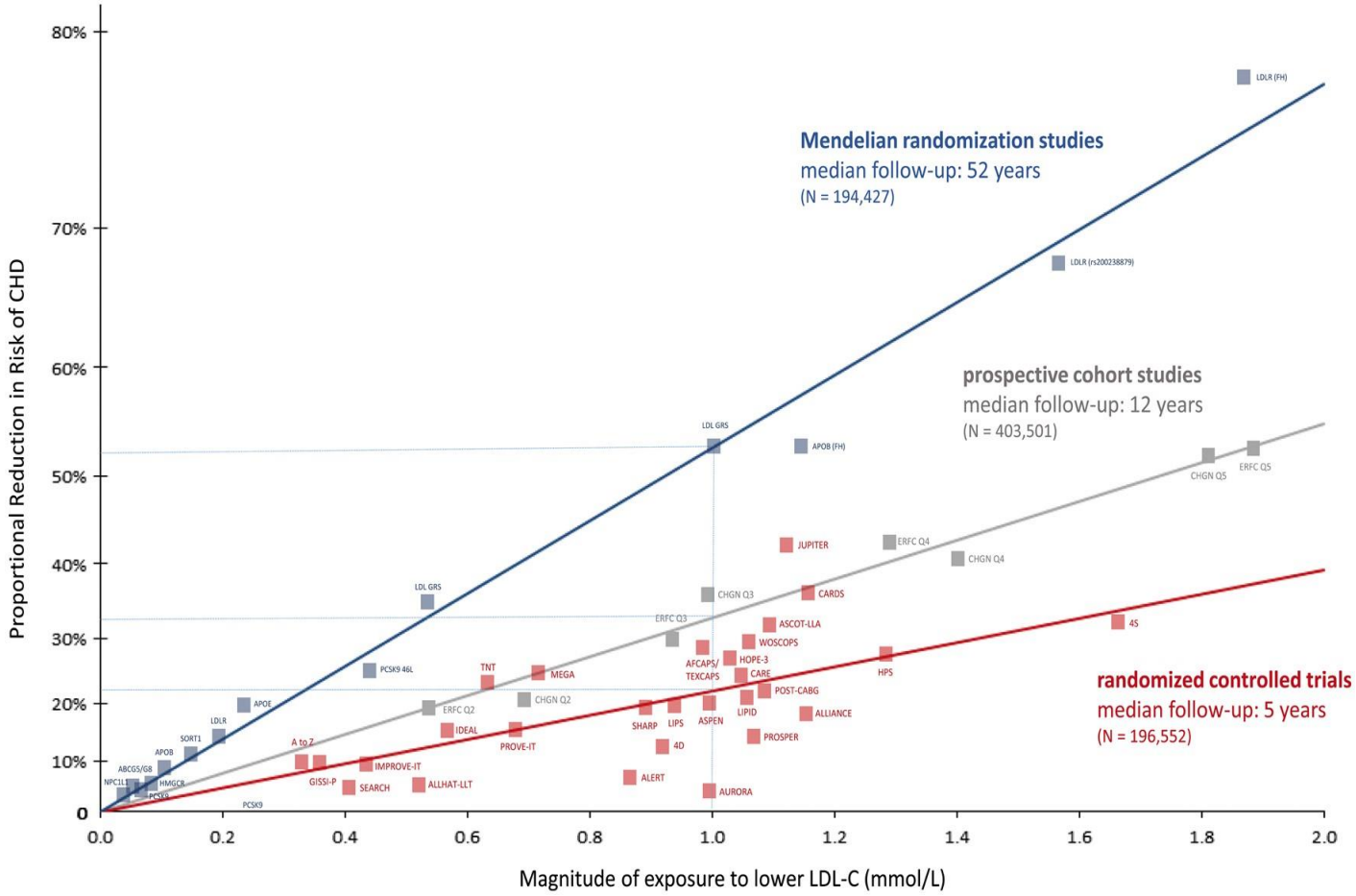
Every 5 minutes, one person has a stroke

Around **1 in 5** people over the age of **60** have peripheral arterial disease

https://www.bhf.org.uk/what-we-do/our-research/heart-statistics
https://www.stroke.org.uk/what-is-stroke/stroke-statistics

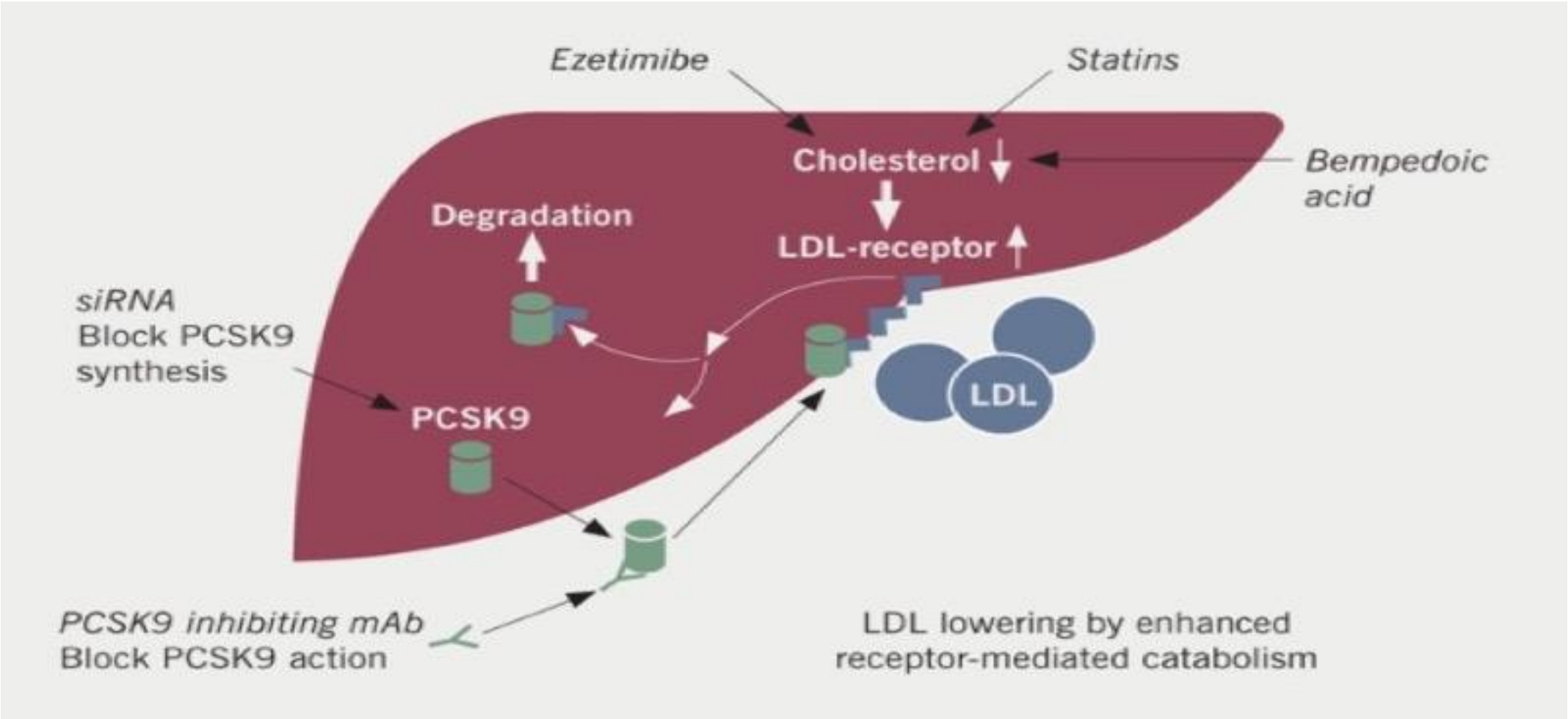
Cardiovascular disease – influence of LDL-cholesterol on CVD risk

Log-linear association per unit change in low-density lipoprotein cholesterol (LDL-C) and the risk of coronary heart disease



Eur Heart J, Volume 38, Issue 32, 21 August 2017, Pages 2459–2472, https://doi.org/10.1093/eurheartj/ehx144

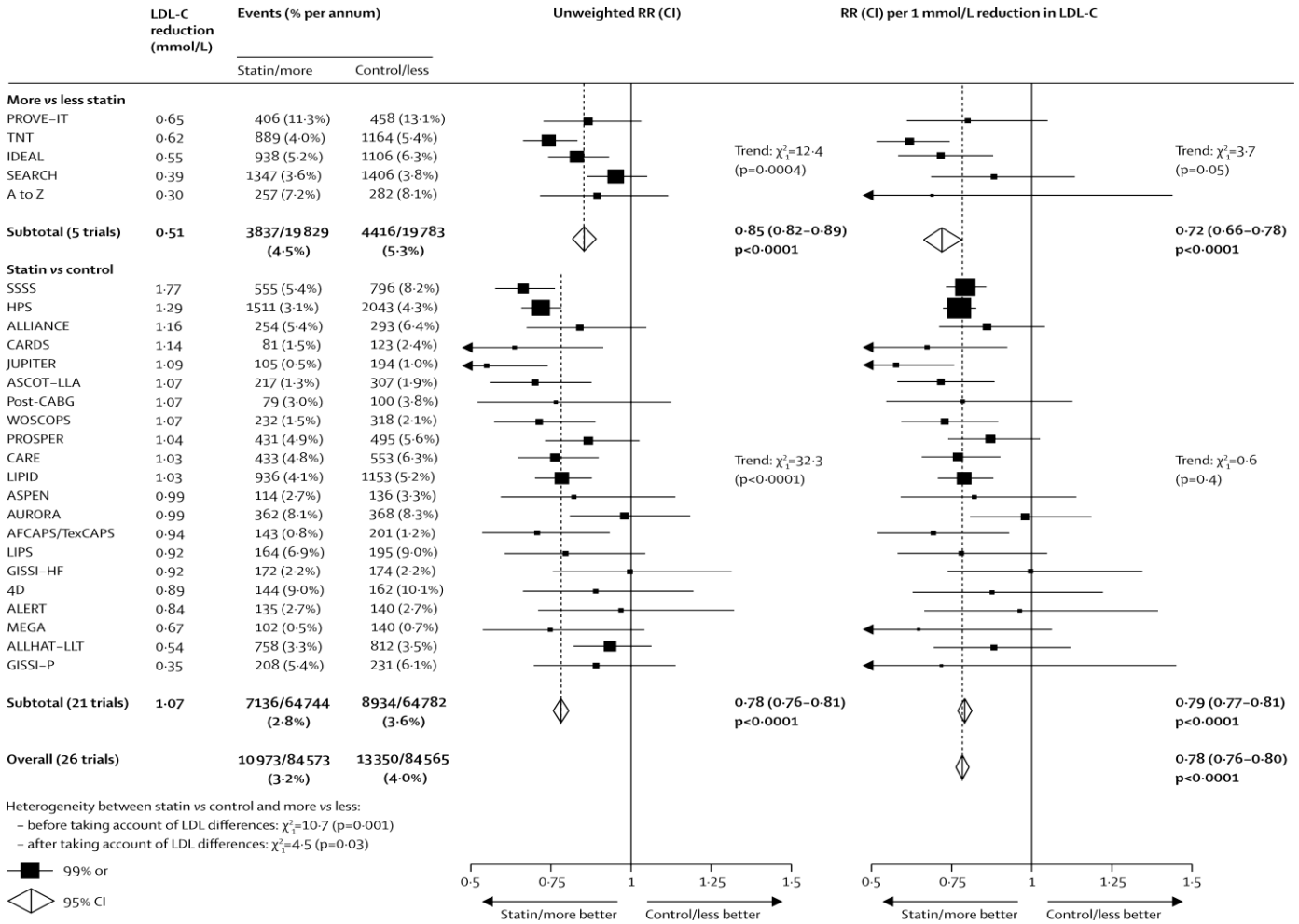
Currently available options for lipid lowering



Packard CJ Br J Cardiol 2021;28(suppl 2):S7–S12doi:10.5837/bjc.2021.s07

Intensive statin therapy reduces CV events – clinical trial evidence

Effect on major vascular events of intensive lowering of LDL cholesterol: a meta-analysis of data from 170 000 participants in 26 randomised trials

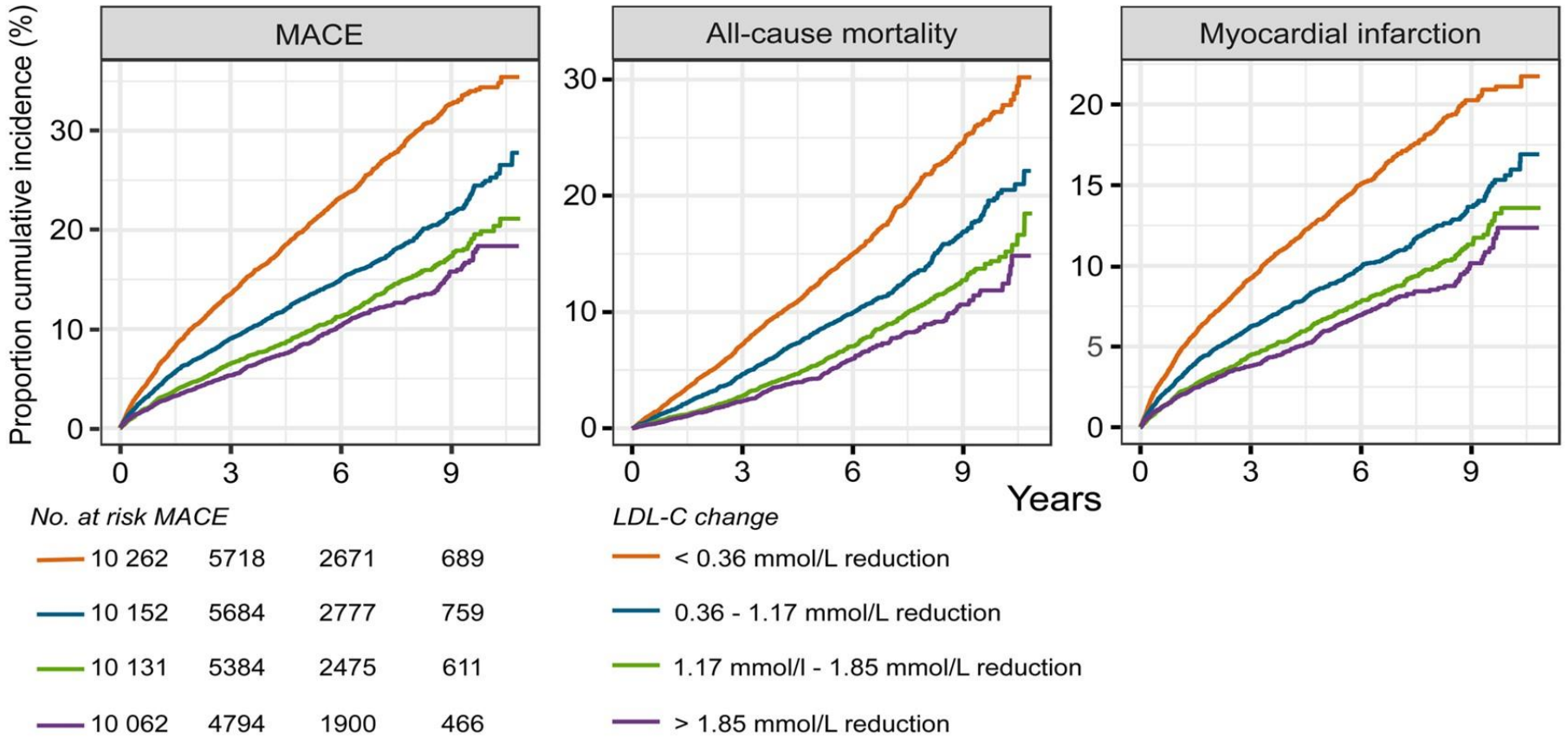


~ 20% reduction in risk of vascular events per 1mmol/L reduction in LDL-C

The Lancet 2010 3761670-1681DOI: (10.1016/S0140-6736(10)61350-5)

Effect of intensive lipid lowering on CV events after myocardial infarction – real world evidence

Data from 40 607 MI patients in the SWEDEHEART Registry Effects of change in LDL-cholesterol between index event and 6-10 week follow up



Eur Heart J, Volume 42, Issue 3, 14 January 2021, Pages 243–252, https://doi.org/10.1093/eurheartj/ehaa1011

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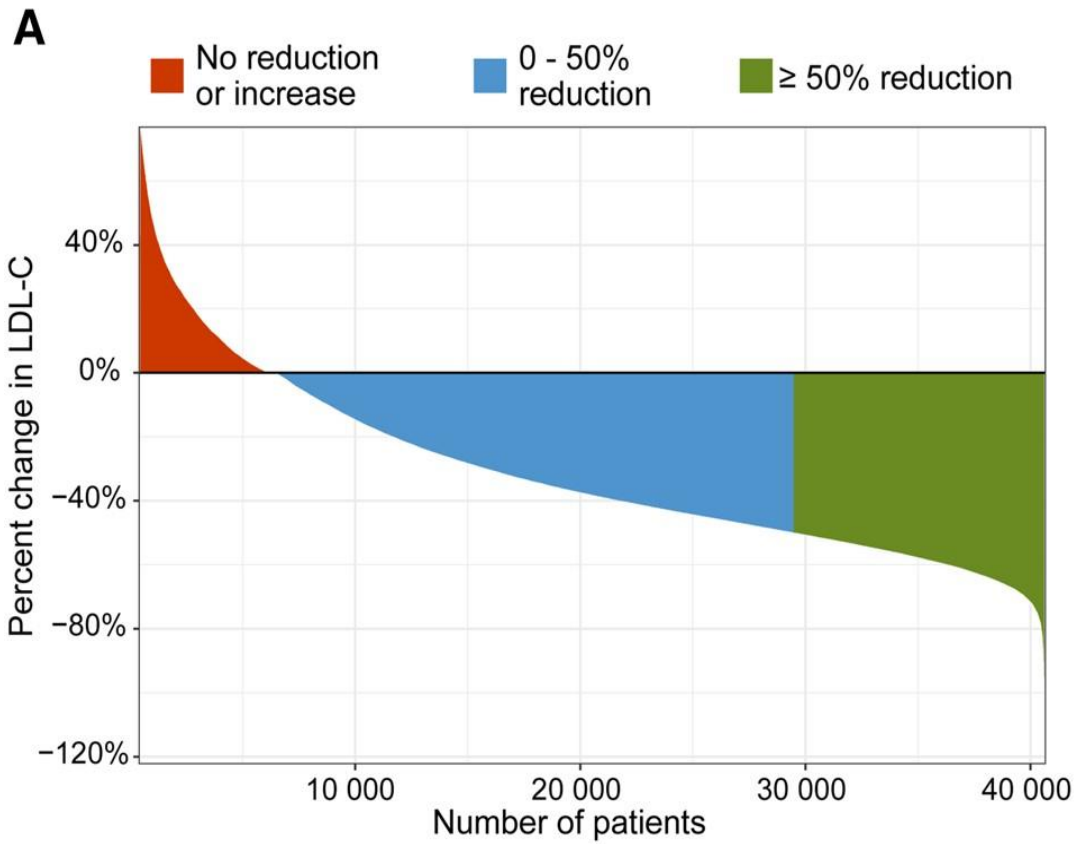
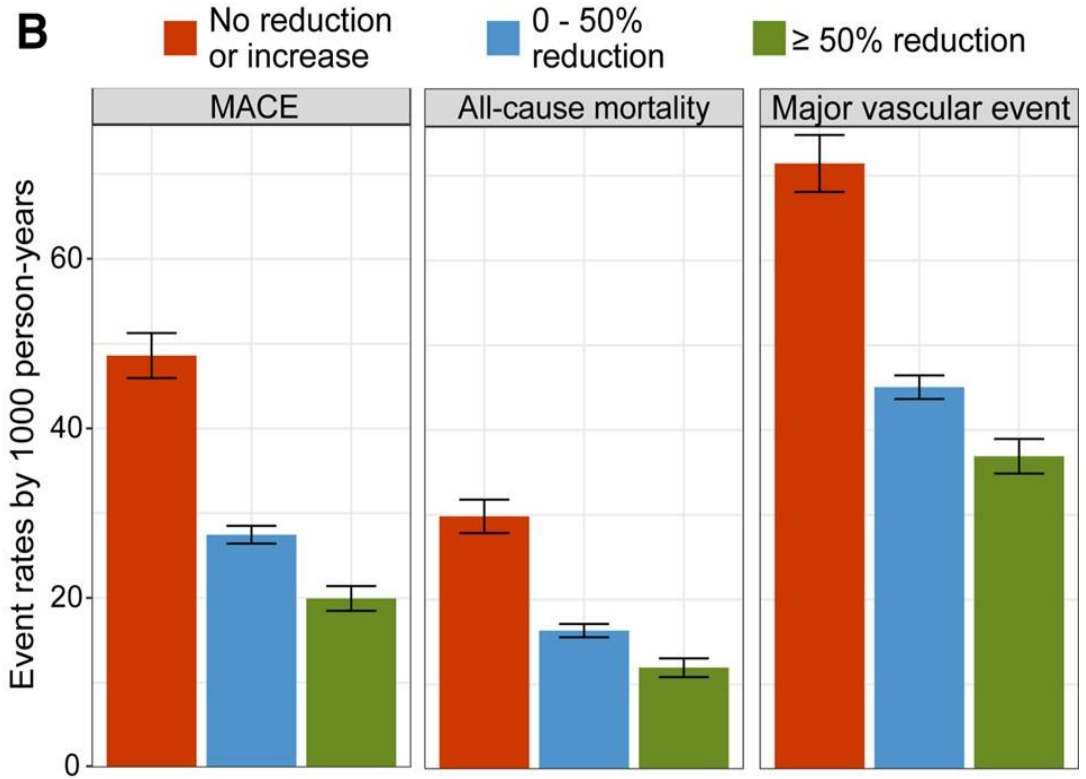
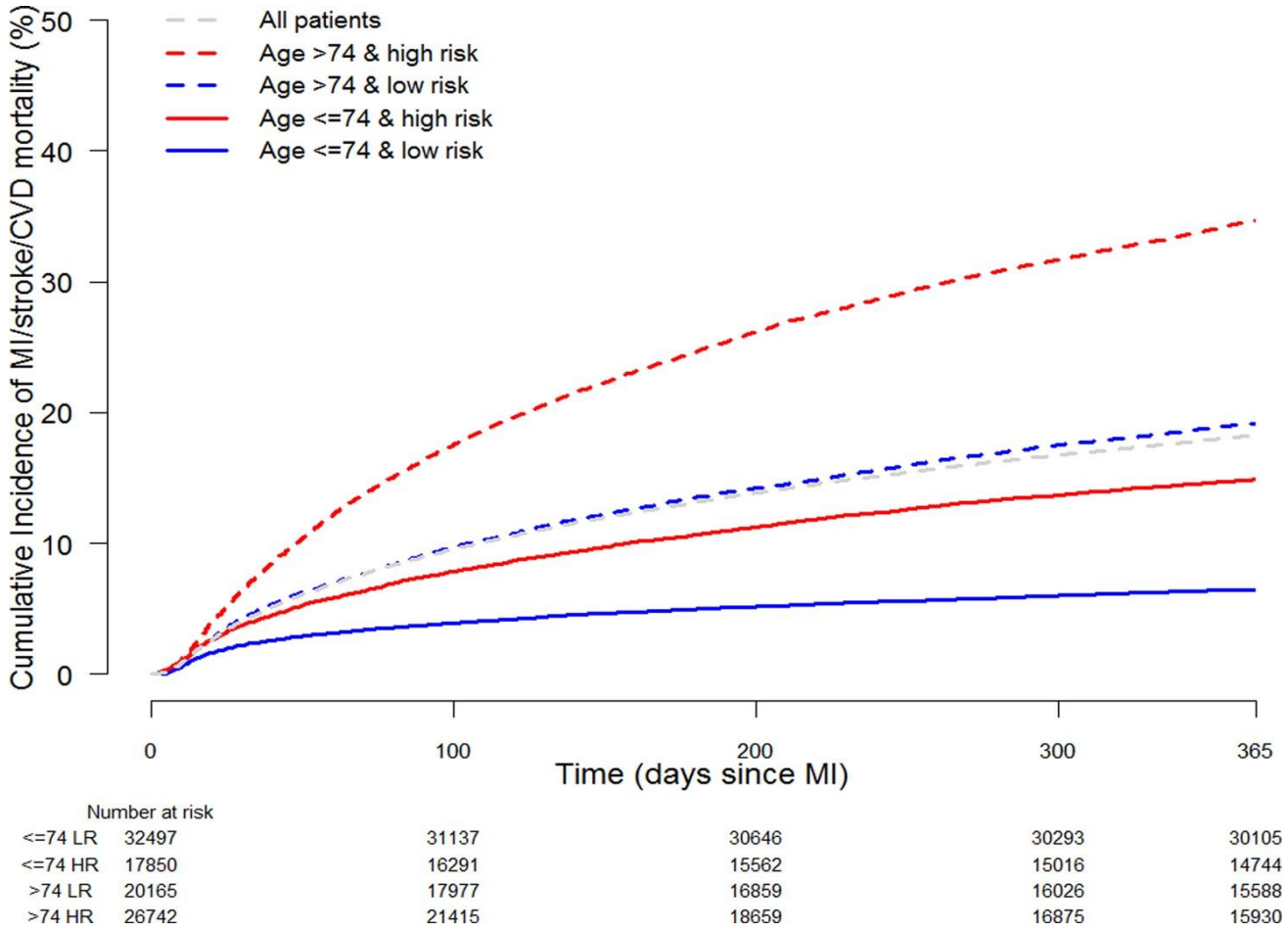




Eur Heart J, Volume 42, Issue 3, 14 January 2021, Pages 243–252, https://doi.org/10.1093/eurheartj/ehaa1011

We know that CV risk is high in the first year after an index CV event

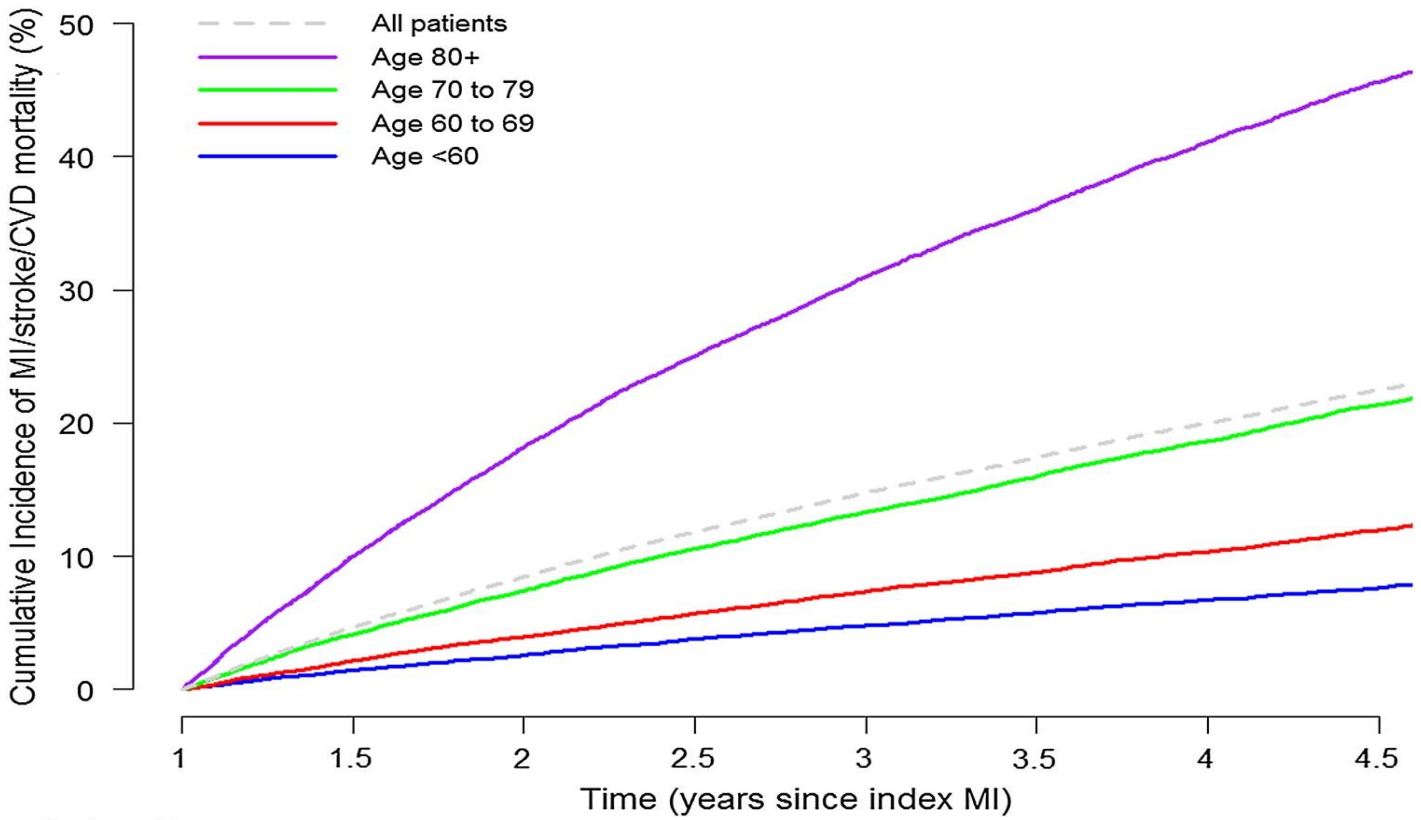
108 315 patients admitted to hospitals in Sweden with a primary MI



Eur Heart J, Volume 36, Issue 19, 14 May 2015, Pages 1163–1170, https://doi.org/10.1093/eurheartj/ehu505

But remember that CV risk remains high after an index CV event

108 315 patients admitted to hospitals in Sweden with a primary MI – Showing risk of events between 1 year and 4.5 years after event



Number at risk

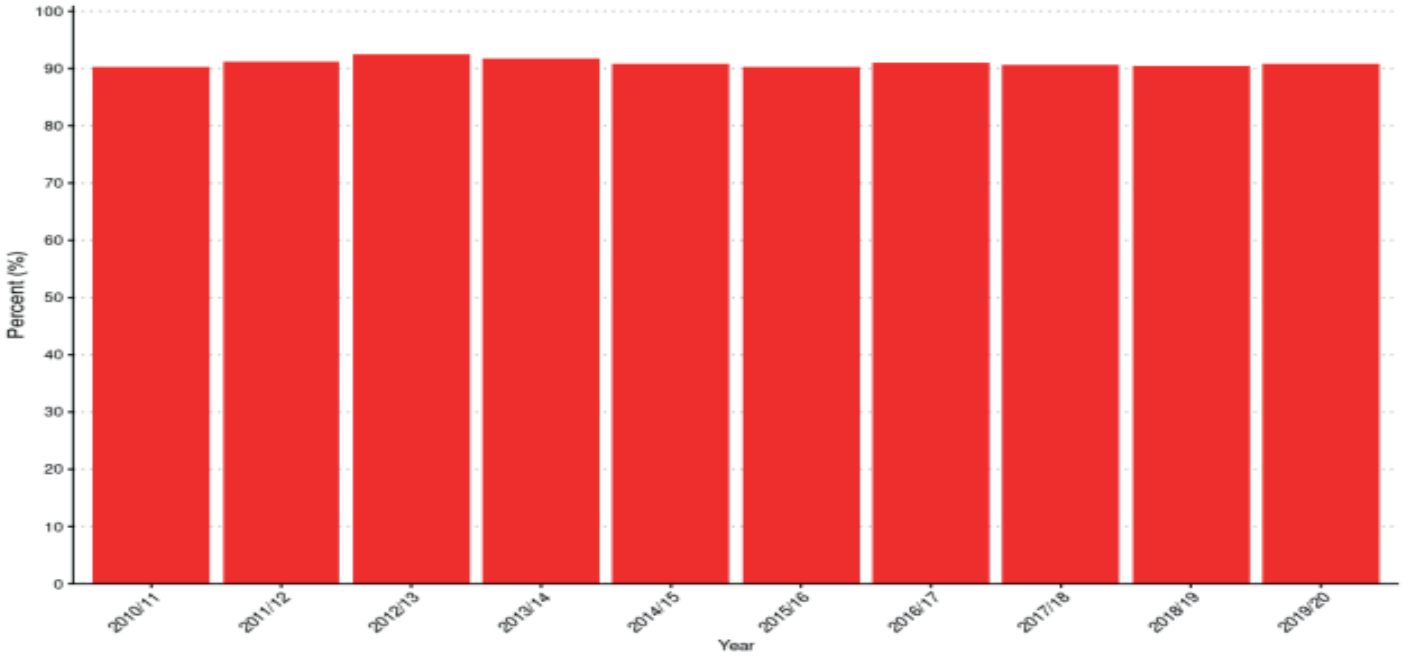
<60	15359	15102	13433	11862	10249	8794	7356	5882
60-69	19667	19108	16796	14656	12545	10636	8688	6815
70-79	20501	19357	16755	14300	12023	10030	8055	6260
80+	21160	18414	14793	11770	9375	7345	5480	3949

Eur Heart J, Volume 36, Issue 19, 14 May 2015, Pages 1163–1170, https://doi.org/10.1093/eurheartj/ehu505

We are good at initiating therapy after myocardial infarction

MINAP Report 2021
Proportion (%) of patients discharged on all secondary prevention medication for which they are eligible, 2010/11 - 2019/20

ACEi/ARB + aspirin + P2Y12 inhibitor + beta-blocker + statin



https://www.nicor.org.uk/wp-content/uploads/2021/10/MINAP-Domain-Report_2021_FINAL.pdf

Repeating lipid profile and reviewing therapy after statin initiation for an acute event is essential

Secondary prevention:

- Start statin treatment in people with CVD with atorvastatin 80 mg. Use a lower dose of atorvastatin if any of the following apply: potential drug interactions, high risk of adverse effects, patient preference.
- Do not delay statin treatment in secondary prevention to manage modifiable risk factors.
- If a person has acute coronary syndrome, do not delay statin treatment. Take a lipid sample on admission and about 3 months after the start of treatment.

Monitoring:

- Measure total cholesterol, HDL cholesterol and non-HDL cholesterol in all people who have been started on high-intensity statin treatment at 3 months of treatment and aim for a greater than 40% reduction in non-HDL cholesterol.
- Provide annual medication reviews for people taking statins. Use these reviews to discuss medicines adherence and lifestyle modification and address CVD risk factors. Consider an annual non-fasting blood test to inform the discussion.

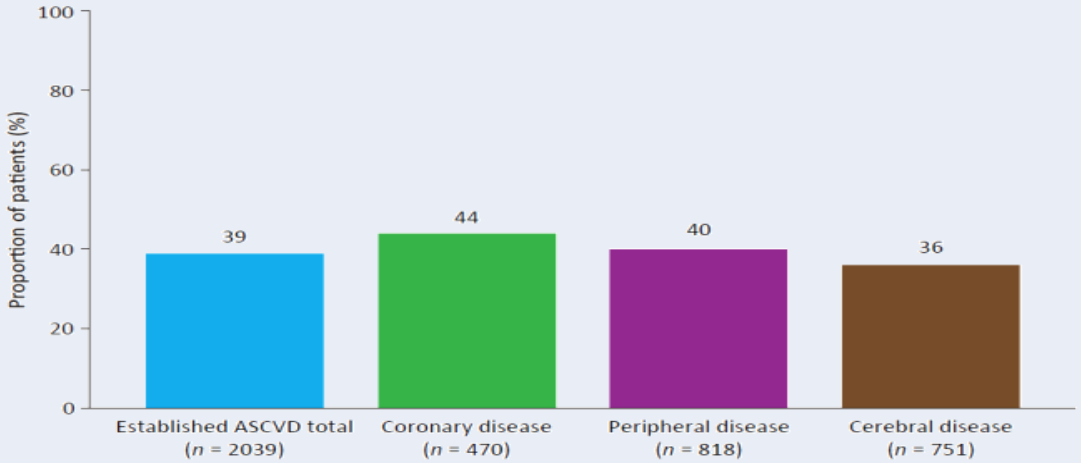
‘FIRE AND FORGET’ IS NOT APPROPRIATE

NICE. Cardiovascular disease: risk assessment and reduction, including lipid modification, Clinical guideline [CG181]

We are not good at treating to target after myocardial infarction

Da Vinci Study
5888 people prescribe lipid-lowering therapy across Europe

Attainment of ESC/EAS 2016 LDL-C targets



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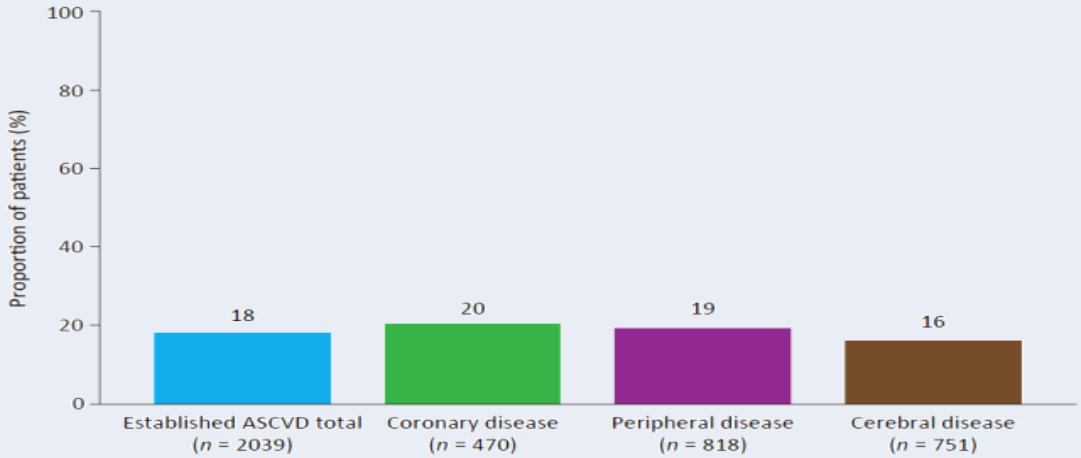


Eur J Prev Cardiol. 2021 Sep 20;28(11):1279-1289. doi: 10.1093/eurjpc/zwaa047.

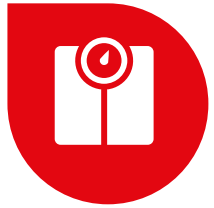
We are not good at treating to target after myocardial infarction

Profile and treatment of chronic coronary syndromes in European Society of Cardiology member countries: The ESC EORP CICD-LT registry

9174 patients with previous ST-elevation myocardial infarction (STEMI), non-STEMI or coronary revascularisation, or other CCS

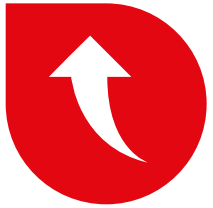
"Poorly controlled cardiovascular risk factors were common across all cohorts"

Current smoking (18.5%)

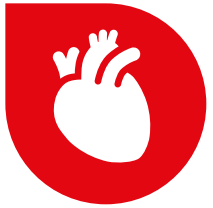


Obesity (33.9%)

Diabetes (25.8%)



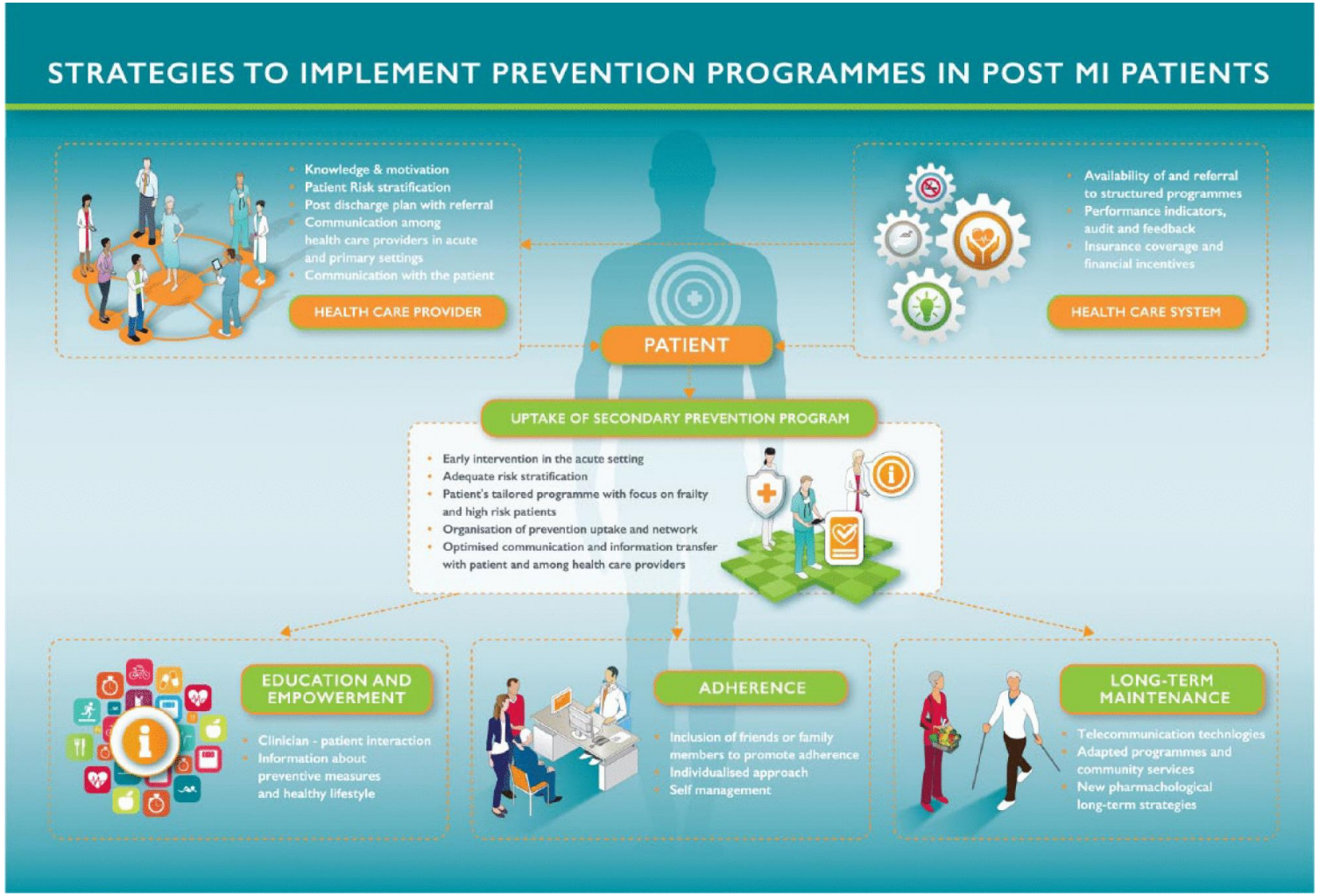
Raised low-density lipoprotein cholesterol (73.3%)



Persistent hypertension (24.7%)

European Journal of Preventive Cardiology, Volume 28, Issue 4, April 2021, Pages 432 445, https://doi.org/10.1177/2047487320912491

We are not good at treating to target after myocardial infarction



Eur Heart J Acute Cardiovasc Care, Volume 6, Issue 4, 1 June 2017, Pages 299–310, https://doi.org/10.1177/2048872616689773

Opportunities to optimise lipid management after a CV event – the UK perspective

Cardiovascular event

1 2 3 months 12 Every 12 months

Secondary Care

In hospital
lipid profile

discharge

cardiac rehab

OP appt
lipid profile
assess adherence

PCSK9 inhibitor clinics

PCSK9i initiation

Statin initiation

Statin optimisation

Management of statin intolerance

Ezetimibe initiation

Ezetimibe/bempedoic acid initiation

Primary Care

repeat lipid profile assess adherence

lipid profile assess adherence

lipid profile assess adherence

Management of statin intolerance

LDL-C >1.8mmol/L

Ezetimibe / Bempedoic acid

LDL-C >4.0mmol/L (3.5mmol/L if very high risk)

Refer for PCSK9i initiation

LDL-C 2.6mmol/L or greater

Inclisiran initiation

03

Considerations for Optimising Post CVD Lipids Management

Dr Rani Khatib

Consultant Cardiology Pharmacist

Leeds Teaching Hospitals NHS Trust

Honorary Senior Lecturer, University of Leeds
National Clinical Champion for PCSK9i & Lipid Optimisation
Accelerated Access Collaborative, NHS England
Co-Chair Cardiology Group, UKCPA
Member of Science Committee European Society of Cardiology, ACNAP

T: @DrRaniKhatib | E: r.khatib@leeds.ac.uk
W: https://medicinehealth.leeds.ac.uk/medicine/staff/507/rani-khatib

The Patient with MI - profile

Non-modifiable risk factors for MI

- Increasing age
- Being male
- Family history of premature CHD
- Premature menopause
- 40-60% higher risk in South Asian patients compared with other populations

Modifiable risk factors for MI

- Smoking
- Diabetes mellitus (and impaired glucose tolerance)
- Metabolic syndrome
- Hypertension
- Hyperlipidaemia
- Obesity
- Physical inactivity

CHD, coronary heart disease; MI, myocardial infarction.

1. Acute myocardial infarction. Available from: https://patient.info/doctor/acute-myocardial-infarction. Last accessed August 2020.

Barriers to prevention of secondary events [1–3]

Patients receive sub-optimal Secondary Prevention Medications (SPM) [1,2]

Cholesterol and blood pressure targets are not achieved[1,2]

Poor medication adherence[1,3]

Post-MI patients who are adherent to SPM are significantly less likely to be readmitted for a cardiovascular-related issue than non-adherent individuals[4]
This applies across all classes of post-MI SPM[5]

MI, myocardial infarction; SPM, secondary prevention medications.

1. Khatib T, *et al. Open Heart* 2018;5:e000921; 2. Rathod KS, *et al. Br J Cardiol* 2012;19:167–169. 3. Naderi S, *et al. Am J Med* 2012;125:882–887. 4. Choudhry NK, *et al*. *Am Heart J* 2014;167:51–58. 5. Ho PM, *et al. Am Heart J* 2008;155:772–779.

Adherence issues

Among 500 patients with coronary artery disease in West Yorkshire, 43.5% were found to be non-adherent with at least one SPM

Statins contributed to **66.7%**

Aspirin to **61.7%**

of overall non-adherence

identified by the Single Question tool.

A number of modifiable barriers to adherence were identified in the 219 non-adherent individuals, including:

- Forgetfulness **(84.9%)**
- Worry that medicines will do more harm than good **(33.8%)**
- Feeling hassled about taking medicines **(18.7%)**
- Feeling worse when taking medicines **(14.2%)**
- Not being convinced of the benefit of medicines **(9.1%)**

SPM, secondary prevention medications.

1. Khatib R, *et al. Open Heart* 2019;6:e000997.

Meds & Risk optimisation needs 12m post MI

- 12-month review was conducted with 201 post-MI patients
  - Patients had ≥10 months of follow up since inpatient treatment for MI between April 2016 and September 2017
- Review was in clinic by a cardiologist, consultant pharmacist or advanced clinical pharmacist
- Mean time from admission to data extraction was 384 days
(range: 308–794 days)

MI, myocardial infarction.

1. Khatib et. al. The post-MI medicines optimisation project Phase 2. Project outcomes report. December 2019. Unpublished

Patient baseline characteristics

Variable	Baseline value (N=210)
Sex, n (%)	
Male	141 (70.1)
Female	60 (29.9)
Age, years, mean (range)	66.9 (34–95)
History of prior MI, n (%)	
Yes	43 (20.9)
No	158 (78.6)
History of prior coronary intervention n, (%)	
Yes (any type)	42 (20.9)
PCI alone	27 (13.4)
CABG alone	5 (2.49)
PCI and CABG	10 (5.0)
No	159 (79.1)
Risk factors	
Type 1 diabetes	3 (1.5)
Type 2 diabetes	48 (23.9)
Current or ex-smoker	99 (49.3)
eGFR <60 mL/min/1.73 m^2 on admission	42 (20.9)

Variable	Baseline value (N=210)
Multi-vessel disease, n (%)	
Yes	104 (51.8)
No	76 (37.8)
Unknown	21 (10.4)
Intervention, n (%)	
PCI	151 (75.1)
CABG	1 (0.5)
Medical management	49 (24.4)
BP recorded at clinic appointment, n (%)	
Yes	121 (60.2)
No	80 (39.8)
SBP at clinic appointment, mmHg, mean (range)	125 (88–189)
DBP at clinic appointment, mmHg, mean (range)	71 (45–99)

BP, blood pressure; CABG, coronary artery bypass graft; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; MI, myocardial infarction; PCI, percutaneous coronary intervention; SBP, systolic blood pressure.

1. Khatib et. al. The post-MI medicines optimisation project Phase 2. Project outcomes report. December 2019. Unpublished.

Are we Measuring Lipid Profile ?

On Admission

- LTHT Protocols - all patients with MI to have baseline total cholesterol (TC) and low-density lipoprotein (LDL) -cholesterol measured within the first 24 hours.
- 167/201 patients (83.1%) had their baseline TC
- 137/201 (68.2%) had their baseline LDL-cholesterol measured.

Repeat (>3 months post MI)

- 148 patients (73.6%) had TC rechecked
- 93 (46.3%) required LDL-cholesterol testing.

Khatib et. al. The post-MI medicines optimisation project Phase 2. Project outcomes report. December 2019. Unpublished.

Achieving Targets - Lipid levels

- **Target levels:**
  - Total cholesterol <4.0 mmol/L
  - LDL-cholesterol <1.8 mmol/L

	Patients (at admission) n/N (%)	Patients (at 12 months) n/N (%)	Absolute change, %
TC < 4.0 mmol/L	45/167 (26.9)	99/148 (66.9)	40.0
LDL-C < 1.8 mmol/L	18/137 (13.1)	60/108 (55.6)	42.5

- At 12 months
  - **40% more** patients were achieving the total cholesterol target compared with baseline (admission)
  - **42.5% more** patients were achieving the LDL-cholesterol target at 12 months compared with baseline (admission)
- The majority of patients, whether they achieved targets or not, were on high-intensity statin therapy

LDL, low density lipoprotein.

1. Khatib et. al. The post-MI medicines optimisation project Phase 2. Project outcomes report. December 2019. Unpublished.

What do you know about targets for LDL-C ?

ESC/EAS recommendations across CV risk categories[1]

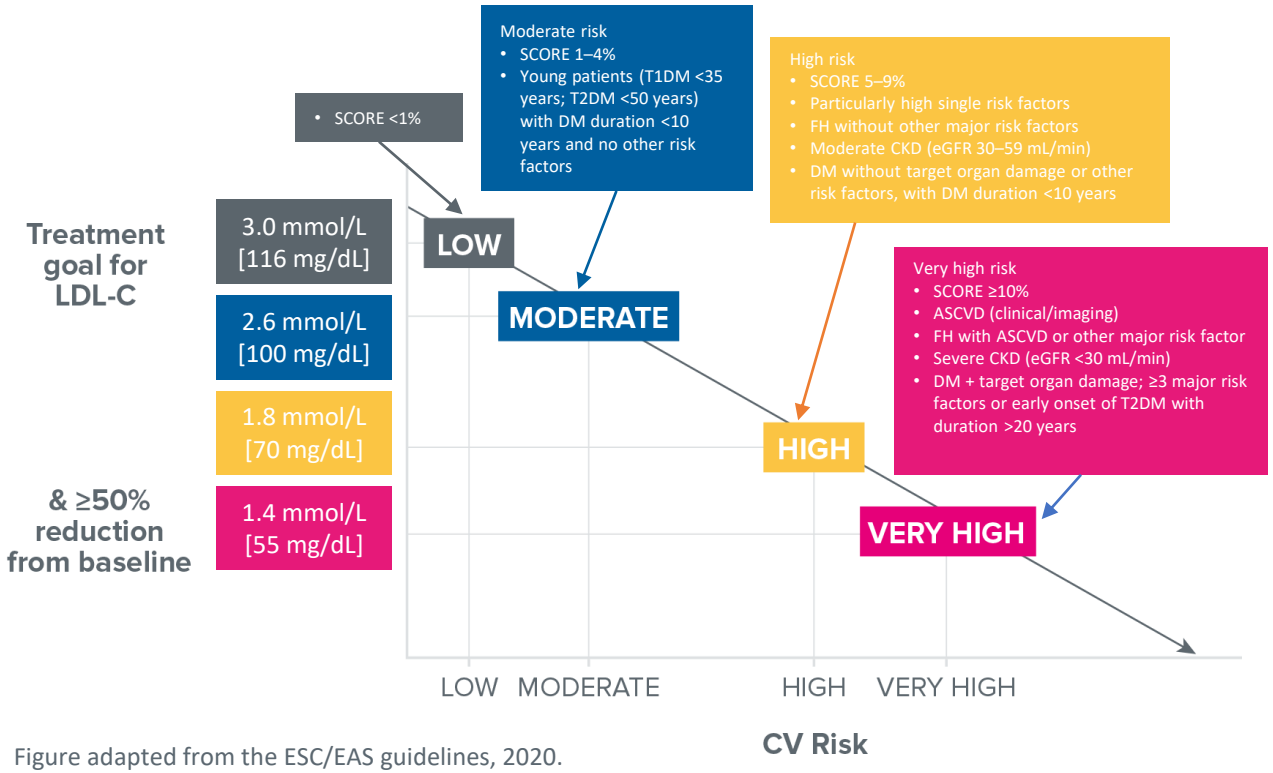


Figure adapted from the ESC/EAS guidelines, 2020.

TITRATION THRESHOLD / TARGET[2]		
	NICE titration threshold	**JBS3**
Primary prevention	Intensify LLT if non-HDL-C reduction from baseline is <40%	Non-HDL-C <2.5 mmol/L (LDL-C <1.8 mmol/L)
Secondary prevention		
FH	Optimise LLT to achieve ≥50% reduction in LDL-C (or Non-HDL-C)	

If baseline cholesterol is unknown in the setting of secondary prevention, use the JBS3 consensus recommendation. Non-HDL-C = TC – HDL-C; LDL-C = non-HDL-C minus (fasting TG* / 2.2).
*valid only when fasting TG <4.5 mmol/L.

- AAC: Accelerated Access Collaborative; ASCVD: atherosclerotic cardiovascular disease; CKD: chronic kidney disease; CV: cardiovascular; DM: diabetes mellitus; EAS: European Atherosclerosis Society; eGFR: estimated glomerular filtration rate; ESC: European Society of Cardiology; HDL-C: high-density lipoprotein cholesterol; FH: familial hypercholesterolaemia; JBS: Joint British Societies; LDL-C: low-density lipoprotein cholesterol; LLT: lipid-lowering therapy; NICE: National Institute for Health and Care Excellence; T1/2DM: type 1/2 diabetes mellitus; TG: triglycerides.
- 1. ESC/EAS Guidelines for the management of dyslipidaemias. Mach F, et al. Eur Heart J 2020;41:111–188; 2. Khatib R & Neely D on behalf of the AAC Clinical Subgroup. June 2021. Pathway approved by NICE July 2021. Available at: https://www.england.nhs.uk/aac/publication/summary-of-national-guidance-for-lipid-management/ (accessed September 2021).

Prescribing Statin and ezetimibe therapy

- The majority of patients were prescribed a statin and a small number of patients were prescribed ezetimibe therapy

Statin and ezetimibe prescription at discharge and 12 months

Drug	Discharge, n (%) N=201	12 months, n (%) N=201
Statin	193 (96.0)	188 (93.5)
Ezetimibe	4 (2.0)	13 (6.5)
Neither	5 (2.5)	9 (4.5)

- Atorvastatin was the most frequently prescribed statin, both at discharge and at 12 months

- The addition of ezetimibe to statin therapy can help in lipid lowering
- At discharge, 4 patients were prescribed ezetimibe, 3 of whom were not receiving statins
- At 12 months, 13/201 patients were receiving ezetimibe
  - 2 of those had LDL-cholesterol < 1.8 mmol/L, 4 had LDL-cholesterol ≥ 1.8 mmol/L, and 7 had no data.

1. Khatib et. al. The post-MI medicines optimisation project Phase 2. Project outcomes report. December 2019. Unpublished.

Escalation of Lipid Lowering Therapy

Patients were not achieving targets despite being on high intensity statins!

Statin therapy	TC ≥ 4.0 mmol/L N=49	LDL-cholesterol ≥ 1.8 mmol/L N=47
High intensity	32 (65.3)	35 (74.5)
Low intensity	11 (22.4)	9 (19.1)
None	6 (12.2)	3 (6.4)

Why?

What can be done?

1. Khatib et. al. The post-MI medicines optimisation project Phase 2. Project outcomes report. December 2019. Unpublished.

Combination Lipid Lowering Therapy

STATIN INTENSITY TABLE

Approximate reduction in LDL-C					
Statin dose mg/day	5	10	20	**40**	**80**
Fluvastatin			21%	27%	33%
Pravastatin		20%	24%	29%	
Simvastatin		27%	32%	37%	42%
Atorvastatin		37%	43%	49%	55%
Rosuvastatin	38%	43%	48%	53%	
Atorvastatin + Ezetimibe 10mg		52%	54%	57%	61%

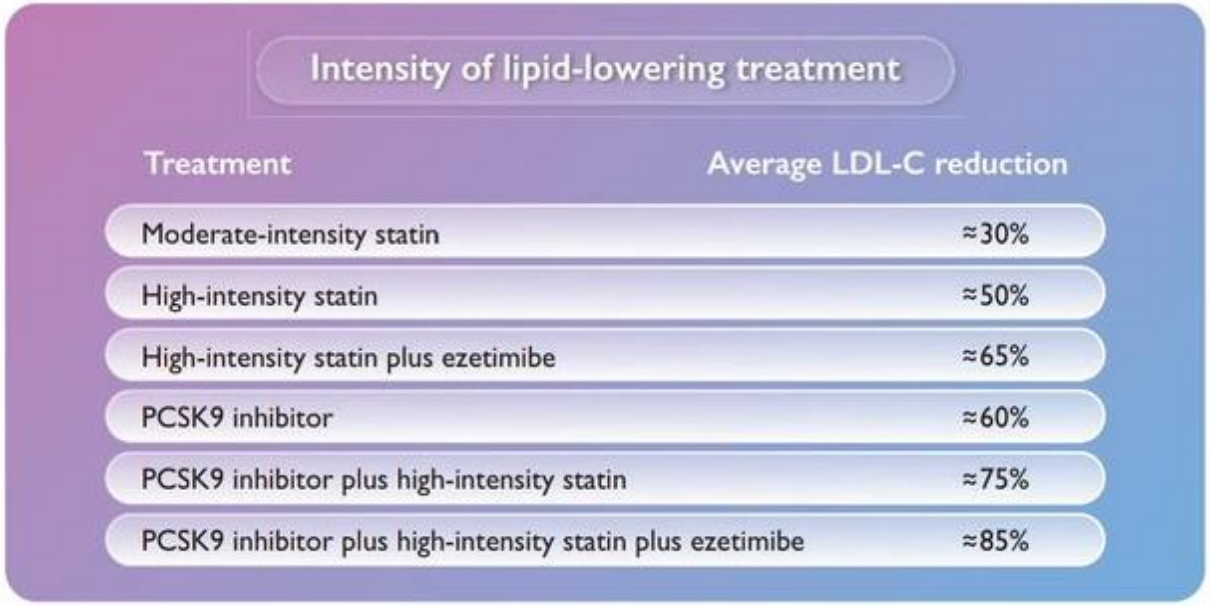
Low intensity statins will produce an LDL-C reduction of 20-30%

Medium intensity statins will produce an LDL-C reduction of 31-40%

High intensity statins will produce an LDL-C reduction above 40%

Simvastatin 80mg is not recommended due to risk of muscle toxicity

- **Rosuvastatin** may be used as an alternative to Atorvastatin if compatible with other drug therapy. Lower starting dose maybe needed in some. See BNF.
- Low/medium intensity statins and should only be used in intolerance or drug interactions.
- **Ezetimibe** when combined with any statin is likely to give greater reduction in non-HDL-C/LDL-C than doubling the dose of the statin.
- **PCSK9i** (NICE TA393,394) alone or in combination with statins or Ezetimibe produce an additional LDL-C reduction of approximately 50% (range 25-70%).
- **Bempedoic acid** when combined with ezetimibe (TA694) produces an additional LDL-C reduction of approximately 28% (range 22-33%) but the long-term treatment effect of bempedoic acid is uncertain.



European Heart Journal, Volume 42, Issue 34, 7 September 2021, Pages 3227–3337, https://doi.org/10.1093/eurheartj/ehab484

Khatib R & Neely D. 2021. National Lipid Management Pathway. Available from https://www.england.nhs.uk/aac/publication/summary-of-national-guidance-for-lipid-management/. Accessed 17th Sept 2021

NICE recommendation for PCSK9i + Innovation score card

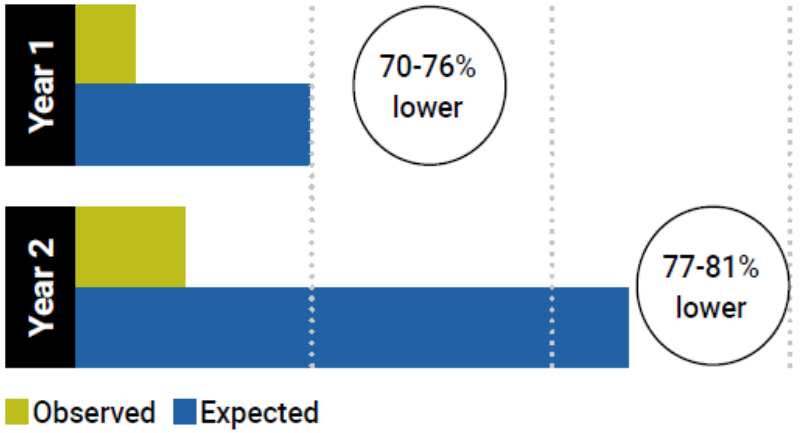
Evolocumab and alirocumab are recommended as options for treating primary hypercholesterolaemia or mixed dyslipidaemia, only if LDL-C concentrations are persistently above the thresholds specified below despite maximal tolerated lipid lowering therapy

Patient populations	Without CVD	With CVD	
		At high risk of CVD*	At very high risk of CVD†
Primary non-FH or mixed dyslipidaemia	Not recommended at any LDL-C concentration	LDL-C >4.0 mmol/L	LDL-C >3.5 mmol/L
Primary HeFH	LDL-C > 5.0 mmol/L	LDL-C >3.5 mmol/L	

* High risk of CVD is defined as a history of any of the following: ACS (such as MI or UA requiring hospitalisation), coronary or other arterial revascularisation procedures, coronary heart disease, ischaemic stroke or peripheral artery disease.
† Very high risk of CVD is defined as recurrent CV events or CV events in more than one vascular bed (that is, polyvascular disease).

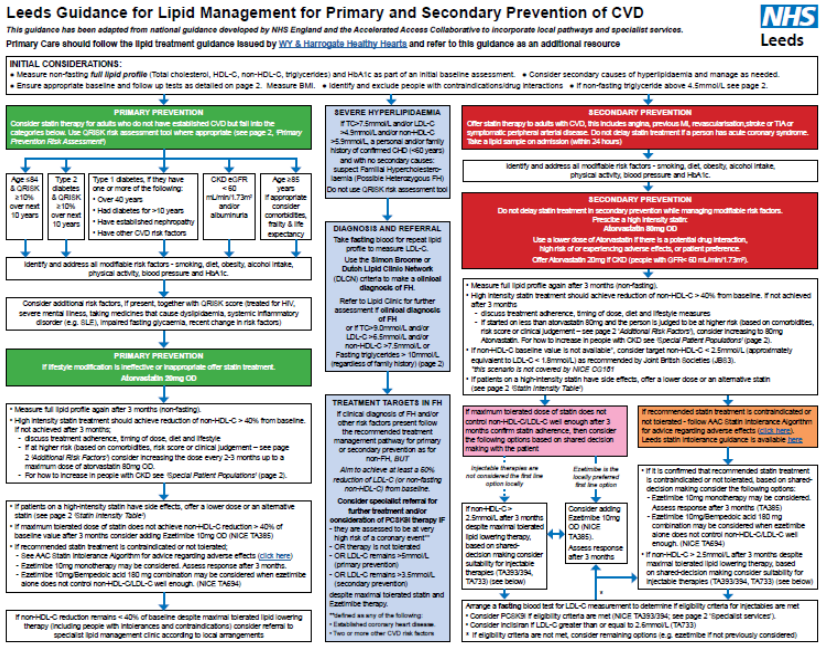
Alirocumab for treating primary hypercholesterolaemia and mixed dyslipidaemia Technology appraisal guidance [TA393]Published date: 22 June 2016 Evolocumab for treating primary hypercholesterolaemia and mixed dyslipidaemia Technology appraisal guidance [TA394]Published date: 22 June 2016

The card reports on medicines in NHS in England which have been positively appraised by NICE



https://digital.nhs.uk/data-and-information/publications/statistical/nice-technology-appraisals-in-the-nhs-in-england-innovation-scorecard/to-march-2019/2.-estimates-report#primary-hypercholesterolaemia-and-mixed-dyslipidaemia

What about Inclisiran? The Leeds Lipids Management Pathway



Leeds Guidance for Lipid Management for Primary and Secondary Prevention of CVD

This guidance has been adapted from national guidance developed by NHS England and the Accelerated Access Collaborative to incorporate local pathways and specialist services.

Primary Care should follow the lipid treatment guidance issued by WY & Harrogate Healthy Hearts and refer to this guidance as an additional resource

NHS Leeds

INITIAL CONSIDERATIONS:

- Measure non-fasting *full lipid profile* (Total cholesterol, HDL-C, non-HDL-C, triglycerides) and HbA1c as part of an initial baseline assessment. • Consider secondary causes of hyperlipidaemia and manage as needed.
- Ensure appropriate baseline and follow up tests as detailed on page 2. Measure BMI. • Identify and exclude people with contraindications/drug interactions • If non-fasting triglyceride above 4.5mmol/L see page 2.

PRIMARY PREVENTION

Consider statin therapy for adults who do not have established CVD but fall into the categories below. Use QRISK risk assessment tool where appropriate (see page 2, *Primary Prevention Risk Assessment*)

- Age ≤84 & QRISK ≥10% over next 10 years
- Type 2 diabetes & QRISK ≥10% over next 10 years
- Type 1 diabetes, if they have one or more of the following: • Over 40 years • Had diabetes for >10 years • Have established nephropathy • Have other CVD risk factors
- CKD eGFR <60 mL/min/1.73m² and/or albuminuria
- Age ≥85 years If appropriate consider comorbidities, frailty & life expectancy

Identify and address all modifiable risk factors - smoking, diet, obesity, alcohol intake, physical activity, blood pressure and HbA1c.

Consider additional risk factors, if present, together with QRISK score (treated for HIV, severe mental illness, taking medicines that cause dyslipidaemia, systemic inflammatory disorder (e.g. SLE), impaired fasting glycaemia, recent change in risk factors)

PRIMARY PREVENTION

If lifestyle modification is ineffective or inappropriate offer statin treatment.
Atorvastatin 20mg OD

- Measure full lipid profile again after 3 months (non-fasting).
- High intensity statin treatment should achieve reduction of non-HDL-C > 40% from baseline. If not achieved after 3 months:
  - discuss treatment adherence, timing of dose, diet and lifestyle
  - If at higher risk (based on comorbidities, risk score or clinical judgement – see page 2 *'Additional Risk Factors'*) consider increasing the dose every 2-3 months up to a maximum dose of atorvastatin 80mg OD.
  - For how to increase in people with CKD see *'Special Patient Populations'* (page 2).
- If patients on a high-intensity statin have side effects, offer a lower dose or an alternative statin (see page 2 *'Statin Intensity Table'*)
- If maximum tolerated dose of statin does not achieve non-HDL-C reduction > 40% of baseline value after 3 months consider adding Ezetimibe 10mg OD (NICE TA385)
- If recommended statin treatment is contraindicated or not tolerated;
  - See AAC Statin Intolerance Algorithm for advice regarding adverse effects (click here)
  - Ezetimibe 10mg monotherapy may be considered. Assess response after 3 months.
  - Ezetimibe 10mg/Bempedoic acid 180 mg combination may be considered when ezetimibe alone does not control non-HDL-C/LDL-C well enough. (NICE TA694)

If non-HDL-C reduction remains < 40% of baseline despite maximal tolerated lipid lowering therapy (including people with intolerances and contraindications) consider referral to specialist lipid management clinic according to local arrangements

SEVERE HYPERLIPIDAEMIA

If TC>7.5mmol/L and/or LDL-C >4.9mmol/L and/or non-HDL-C >5.6mmol/L, a personal and/or family history of confirmed CHD (<60 years) and with no secondary causes: suspect Familial Hypercholesterolaemia (Possible Heterozygous FH)
Do not use QRISK risk assessment tool

DIAGNOSIS AND REFERRAL

Take fasting blood for repeat lipid profile to measure LDL-C
Use the Simon Broome or Dutch Lipid Clinic Network (DLCN) criteria to make a clinical diagnosis of FH

Refer to Lipid Clinic for further assessment if clinical diagnosis of FH
or if TC>9.0mmol/L and/or LDL-C >6.5mmol/L and/or non-HDL-C >7.5mmol/L or Fasting triglycerides > 10mmol/L regardless of family history) (page 2)

TREATMENT TARGETS IN FH

If clinical diagnosis of FH and/or other risk factors present follow the recommended treatment management pathway for primary or secondary prevention as for non-FH, *BUT*

Aim to achieve at least a 60% reduction of LDL-C (or non-fasting non-HDL-C) from baseline.

Consider specialist referral for further treatment and/or consideration of PCSK9i therapy IF

- they are assessed to be at very high risk of a coronary event*
- OR therapy is not tolerated
- OR LDL-C remains >5mmol/L (primary prevention)
- OR LDL-C remains >3.5mmol/L (secondary prevention)

despite maximal tolerated statin and Ezetimibe therapy.

*Defined as any of the following:
- Established coronary heart disease.
- Two or more other CVD risk factors

SECONDARY PREVENTION

Offer statin therapy to adults with CVD, this includes angina, previous MI, revascularisation,stroke or TIA or symptomatic peripheral arterial disease. Do not delay statin treatment if a person has acute coronary syndrome. Take a lipid sample on admission (within 24 hours)

Identify and address all modifiable risk factors - smoking, diet, obesity, alcohol intake, physical activity, blood pressure and HbA1c.

SECONDARY PREVENTION

Do not delay statin treatment in secondary prevention while managing modifiable risk factors.
Prescribe a high intensity statin:
Atorvastatin 80mg OD
Use a lower dose of Atorvastatin if there is a potential drug interaction, high risk of or experiencing adverse effects, or patient preference.
Offer Atorvastatin 20mg if CKD (people with GFR= 60 mL/min/1.73m²).

- Measure full lipid profile again after 3 months (non-fasting).
- High intensity statin treatment should achieve reduction of non-HDL-C > 40% from baseline. If not achieved after 3 months
  - discuss treatment adherence, timing of dose, diet and lifestyle measures
  - If started on less than atorvastatin 80mg and the person is judged to be at higher risk (based on comorbidities, risk score or clinical judgement – see page 2 *'Additional Risk Factors'*), consider increasing to 80mg Atorvastatin. For how to increase in people with CKD see *'Special Patient Populations'* (page 2).
- If non-HDL-C baseline value is not available*, consider target non-HDL-C < 2.6mmol/L (approximately equivalent to LDL-C < 1.8mmol/L) as recommended by Joint British Societies (JBS3).
  *this scenario is not covered by NICE DG181
- If patients on a high-intensity statin have side effects, offer a lower dose or an alternative statin (see page 2 *'Statin Intensity Table'*)

If maximum tolerated dose of statin does not control non-HDL-C/LDL-C well enough after 3 months confirm statin adherence, then consider the following options based on shared decision making with the patient

If recommended statin treatment is contraindicated or not tolerated - follow AAC Statin Intolerance Algorithm for advice regarding adverse effects (click here). Leeds statin intolerance guidance is available here

Injectable therapies are not considered the first line option locally

Ezetimibe is the locally preferred first line option

If non-HDL-C > 2.5mmol/L after 3 months despite maximal tolerated lipid lowering therapy, based on shared-decision making consider suitability for injectable therapies (TA393/394, TA733) (see below)

Consider adding Ezetimibe 10mg OD (NICE TA385).
Assess response after 3 months

- If it is confirmed that recommended statin treatment is contraindicated or not tolerated, based on shared-decision making consider the following options:
  - Ezetimibe 10mg monotherapy may be considered. Assess response after 3 months (TA385)
  - Ezetimibe 10mg/Bempedoic acid 180 mg combination may be considered when ezetimibe alone does not control non-HDL-C/LDL-C well enough. (NICE TA694)
- If non-HDL-C > 2.5mmol/L after 3 months despite maximal tolerated lipid lowering therapy, based on shared-decision making consider suitability for injectable therapies (TA393/394, TA733) (see below)

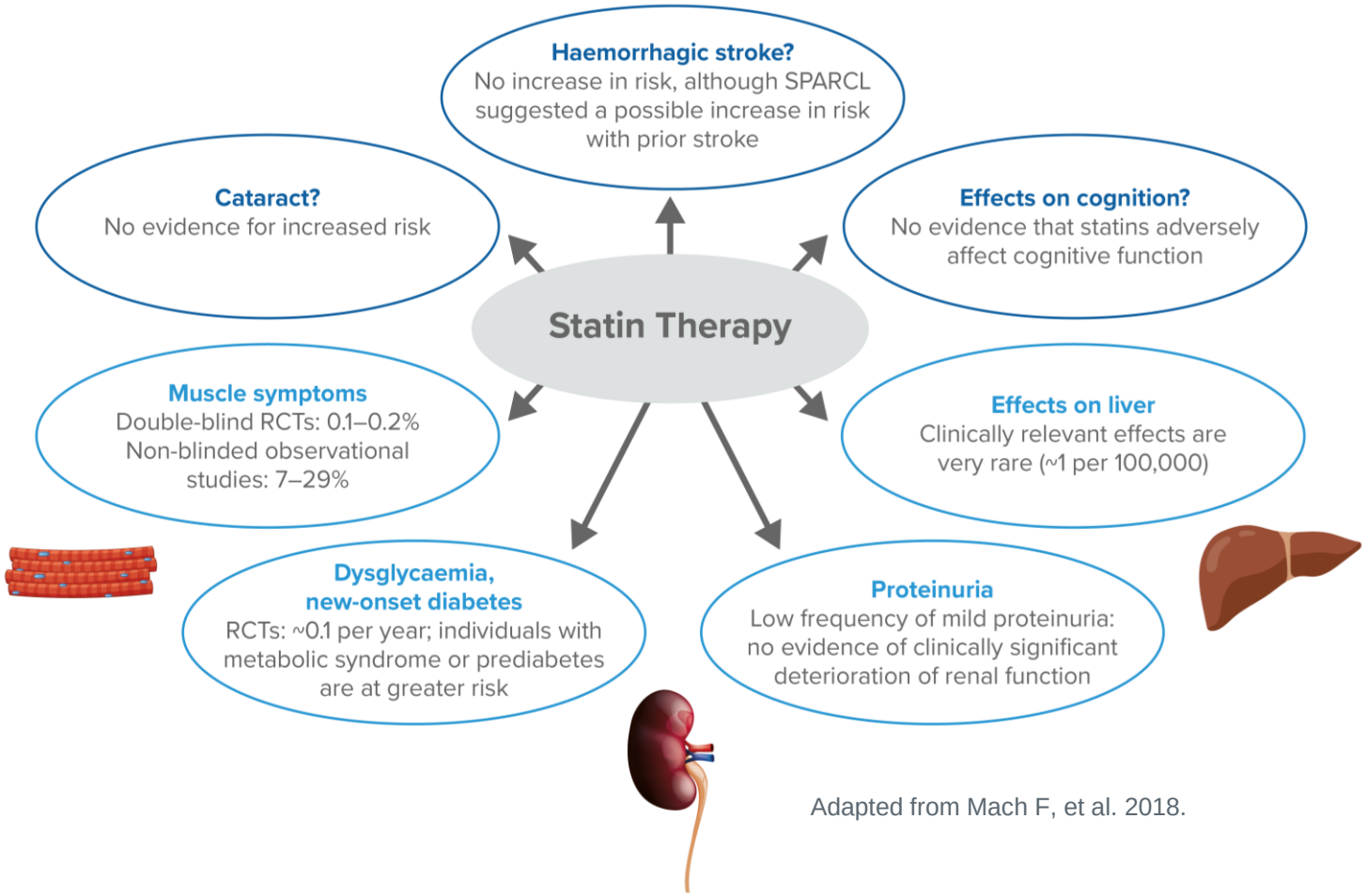
Arrange a **fasting** blood test for LDL-C measurement to determine if eligibility criteria for injectables are met

- Consider PCSK9i if eligibility criteria are met (NICE TA393/394; see page 2 'Specialist services').
- Consider inclisiran if LDL-C greater than or equal to 2.6mmol/L (TA733)

* If eligibility criteria are not met, consider remaining options (e.g. ezetimibe if not previously considered)

Statin intolerance and concerns[1]

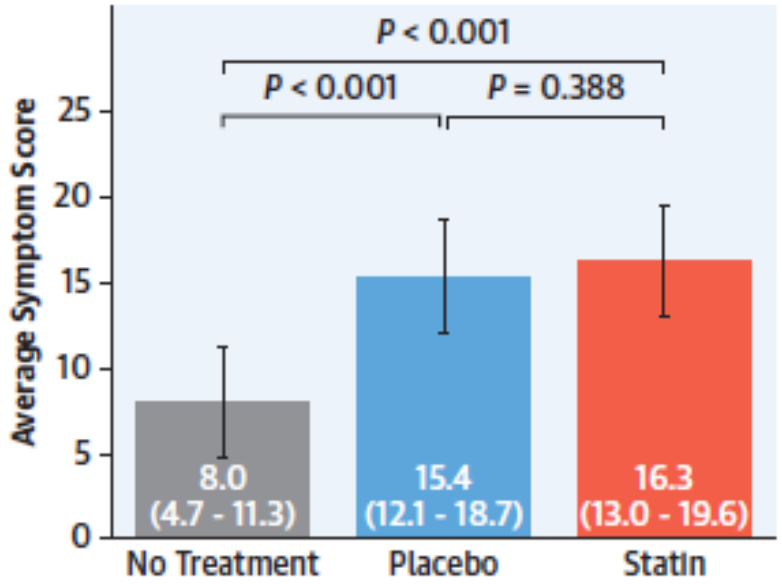
Overview of the relative prevalence of the main types of adverse effects reported with statin therapy

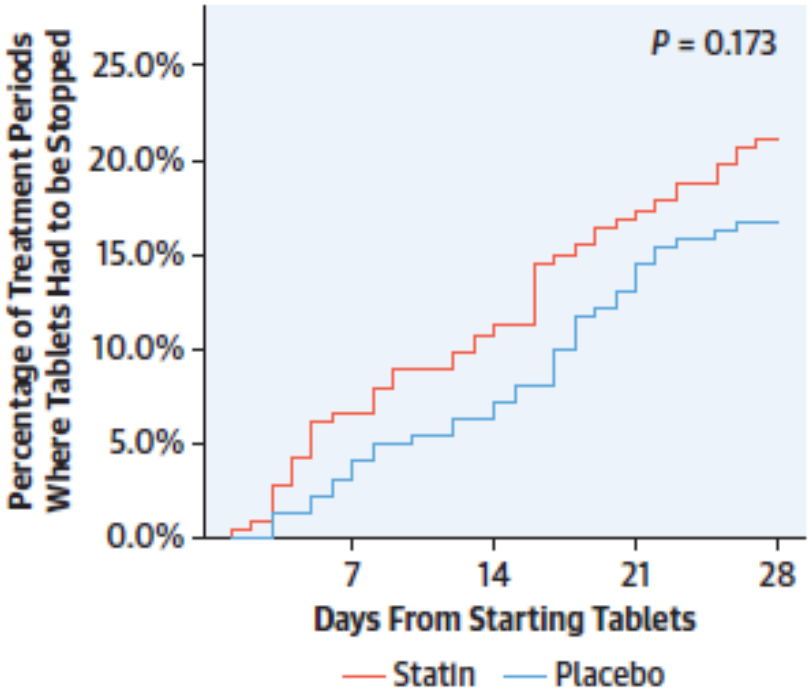


Adapted from Mach F, et al. 2018.

- RCT: randomised controlled trial; SPARCL: Stroke Prevention by Aggressive Reduction in Cholesterol Levels trial.
- 1. Mach F, et al. Eur Heart J 2018;39:2526–2539.

CENTRAL ILLUSTRATION Symptom Scores and Cumulative Early Tablet Stopping Rates by Treatment





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

(Left) The mean symptom scores across the 3 treatment types (statin, placebo, and no treatment). **Whiskers** indicate the associated 95% CIs. **(Right)** The cumulative rate of stopping tablets for patients starting a statin **(red)** or placebo **(blue)** after a no-tablet month. *P* value derived from a mixed-effects logistic regression model.

The NECEBO effect

Statin Intolerance Pathway

Person at high CVD risk reports potential intolerance to recommended high intensity statin treatment

This resource relates to NICE guidance:
CG181, CG71, TA385, TA393/394, QS100

New onset or worsening of muscle symptoms since starting statins?
(pain, tenderness or weakness)

NO → Consider other potential side effects for statins
- Be aware of **Statin Reluctance** and **Nocebo Effect**
- See **'Person Centred Care'** box at page 2

YES

Symptoms typical for Statin Related Muscle toxicity (SRM)*?

NO → Muscular symptoms not related to statins

*Symmetrical pain and/or weakness in large proximal muscle groups, worsened by exercise

YES

Measure Creatinine Kinase (CK)
Assess severity of symptoms
+/- repeat baseline assessment**

**Consider other causes if new onset of muscle symptoms of > 2 weeks duration in a person previously tolerant of statin therapy for > 3 months

Non SRM: Consider other causes e.g. PMR, Vit D deficiency.
Check bone profile, Vit D, CRP.

Tolerable symptoms
No clinical concern
CK < 4x ULN

→ Improvement within 2 weeks
Resolved within 6 weeks
Patient happy to continue

NO → Wait for 2 weeks before **rechallenge**

Intolerable symptoms
and/or clinical concern and/or CK > 4x and < 10x ULN

CK > 10x and < 50x ULN

CK > 50x ULN

Stop statin for 4-6 weeks
Document time to symptom onset and time to resolution

YES → **Renal function**
stable/normal eGFR

NO → Stop statin and consider **Rhabdomyolysis**

Has CK normalised?

→ Consider **Statin induced necrotising autoimmune myopathy** (SINAM)

YES

Have symptoms resolved?

NO → Non-SRM. **Consider other causes**

YES

Has the patient been **symptom free for at least 2 weeks?**

NO → Wait for 2 weeks before **rechallenge**

YES

Reassess and **restart** with lower dose/alternative statin (see page 2 - **'Statin-based Approaches'**)
Offer low or moderate dose of a higher intensity statin (Atorvastatin 10 or 20 mg OD, or Rosuvastatin 5 or 10mg OD)

No recurrence of muscle symptoms
Titrate at 8 weeks intervals to achieve appropriate targets

Recurrence of muscle symptoms
Short time to onset
Symptoms intolerable

Symptoms tolerable
Treatment goals achieved
Patient happy to continue

NO → **Consider further options**
(For example co-administering ezetimibe or as monotherapy)
see page 2 - **'Statin-based Approaches'**

IF NOT EFFECTIVE → **Seek specialist advice and consider** PCSK9i
(NICE TA 393, 394)

Urgently
seek specialist advice and inpatient assessment

Abbreviations

CK = Creatinine Kinase
CRP = C-Reactive Protein
eGFR = Estimated Glomerular Filtration Rate
PMR = Polymyalgia Rheumatica
SINAM = Statin Induced Necrotising Autoimmune Myopathy
SRM = Statin Related Muscle Toxicity
ULN = Upper Limit of Normal Range
Vit D = Vitamin D

Please refer to page 2 for more details

Introduction

- Statins are the cornerstone for prevention and treatment of cardiovascular (CV)disease with a substantial evidence of reduction of morbidity and mortality. Refer to Lipid Management Pathway and related NICE guidelines (CG181,CG71) for guidance on initiation, titration and monitoring of statin therapy.
- In clinical trials, statins were found to be largely well tolerated (often with a similar adverse effect ((AE) profile to placebo), however this is not reflected in clinical practice where up to 75% of people started on a statin will discontinue treatment within 2 years.
- Stopping statin therapy is associated with an increased risk of major CV events and there is growing concern that clinicians are labelling patients as 'statin intolerant' too quickly. Indeed, statin discontinuation is significantly associated with negative media coverage.

Definition of Statin Intolerance

- Intolerance to initial statin therapy is defined by NICE as the presence of clinically significant adverse effects that represent an unacceptable risk to the patient or that may reduce compliance with therapy.
- Other definition: any adverse event (AEs) considered unacceptable by the patient, and/or some laboratory abnormalities, both attributed to statin treatment and leading to its discontinuation.

Statin-associated muscle symptoms (SAMS)

- SAMS are one of the principal reasons for statin non-adherence and/or discontinuation. However, not all such symptoms should lead to a label of 'statin intolerance' as they may not be truly statin related muscle toxicity(SRM) as demonstrated by resolution on de-challenge and recurrence with re-challenge.

Non-Statin related musculoskeletal symptoms (Non SRM)

- If patients report symptoms that are not typical of SRM (e.g. asymmetric distribution, failure to resolve with de-challenge despite normal CK) consider other musculoskeletal disorders, metabolic, degenerative or inflammatory e.g. Vitamin D deficiency, polymyalgia rheumatica. Check Bone profile, Vit D, CRP.

Considerations when starting a statin to reduce risk of SRM

- Check baseline thyroid, liver and renal function, any potential drug interactions, and avoid the highest doses in at risk groups (See "Risk Factors" below).
- Ask the person if they have had persistent generalised unexplained muscle pain, whether associated or not with previous lipid-lowering therapy. If they have, measure CK. If CK levels are > 4x ULN do not start statin -investigation required. **Do not measure CK if person is asymptomatic.**
- Warn patients about AEs, specifically muscle symptoms. Advise people who are being treated with a statin to seek medical advice if they develop muscle symptoms (pain, tenderness or weakness). If this occurs, measure CK (see page 1).

Risk factors for SRM and statin intolerance

Endogenous factors

- Female gender
- Advanced age (> 75yrs)
- Frailty (reduced lean body mass)
- History of muscle disorder or high CK
- Impaired renal or hepatic function
- Personal or Family history of intolerance to lipid-lowering therapies
- Hypothyroidism

Exogenous Factors

- Excessive alcohol intake
- High intensity exercise
- Dehydration
- Drug interactions with statins (including herbal medicines)
- Vitamin D deficiency

Classification of statin related muscle toxicity (SRM)

SRM	Phenotype	Incidence	Definition
SRM 0	CK elevation < 4x ULN	1.5-26%	No muscle symptoms
SRM 1	No muscle symptoms	190/100,000 Patient-years; 0.3-33%	Muscle symptoms without CK elevation
SRM 2	Myalgia, intolerable	0.2-2/1,000	Muscle symptoms, CK < 4x ULN, complete resolution on de-challenge
SRM 3	Myopathy	5/100,000 Patient-years	CK elevation > 4x ULN < 10x ULN ± muscle symptoms, complete resolution on de-challenge
SRM 4	Severe myopathy	0.11%	CK elevation > 10x ULN < 50x ULN, muscle symptoms, complete resolution on de-challenge
SRM 5	Rhabdomyolysis	0.1-8.4/100,000	CK elevation > 10x ULN with evidence of renal impairment + muscle symptoms or CK > 50x ULN
SRM 6	Autoimmune-mediated necrotising myositis (SINAM)	~2/million per year	Detection of HMGCR antibodies, HMGCR expression in muscle biopsy showing autoimmune myositis, incomplete resolution on de-challenge

HMGCR = 3-hydroxy-3-methylglutaryl coenzyme A reductase ULN = upper limit of normal

- SRM is a spectrum from myalgia to severe myopathy
- SRM 0 - does not preclude statin therapy, consider reducing starting dose
- SRM 1-3 manage according to pathway
- When SRM4 is suspected, without evidence of impaired renal function, discontinue statin therapy immediately and refer for outpatient assessment. Assess and treat possible contributory factors and re-assess the need for a statin. Intensify lifestyle modifications and consider alternative lipid lowering regimens.
- If rhabdomyolysis (SRM5) is suspected, immediately stop statins, urgently refer to inpatient assessment and management including intravenous rehydration as required to preserve renal function. Do not wait for measurement of urinary myoglobin. Post recovery, manage as for SRM4.
- Statin induced necrotising autoimmune myositis (SINAM) (SRM6) should be suspected in patients with progressive muscle weakness and ongoing CK elevation despite statin withdrawal. Requires immunosuppressive treatment and avoidance of re-exposure to statins. Re-assess the need for lipid lowering therapy - may be eligible for treatment with PCSK9 inhibitor (NICE TA 393, 394).

Person-centred approach to address statin intolerance

Initial Consultation

- Be aware of "nocebo effect"1 and "statin reluctance"2
- Reinforce healthy lifestyle habits(e.g. exercise, reducing weight)
- Listen to the concerns of each patient
- Explain LDL-C targets and strategies to lower LDL-C/non-HDL-C
- Discuss options to reduce LDL-C/non-HDL-C with pros and cons
- Explain the benefits of statins
- Evaluate & identify any risk factors and address (e.g. drug interactions)
- Work with patients to identify and agree best options and next steps

Follow up

- Follow up on agreed plan and address any issues/concern.
- Advise patients to contact you if they experience muscle symptoms
- Ongoing patient education and regular review helps addressing concerns around medicine safety and underline the importance of adherence.

(1) Nocebo effect is negative expectations of the patient regarding a treatment leading to reporting more negative effects even if they are prescribed a placebo.

(2)Statin reluctance is an attitudinal state of aversion to taking statins (often without prior exposure).

Statin-based approaches to manage muscle symptoms

- Adopt person-centred approach as described above
- Therapy with a lower dose statin is preferred to no statin
- Apply a repetitive "De-Challenge" - "Re-Challenge" approach to establish if symptoms are caused by a statin(s) and the best statin regimen for each patient
- Switch to a different statin or re-challenge with the same statin using a lower dose or frequency (intermittent dosages)
- Patients who do not tolerate statins on a daily basis, alternate day or twice-weekly dosing is a good option.
- Rosuvastatin and atorvastatin have longer half-lives, permitting their use on anon-daily regime
- Adding ezetimibe to a lower dose statin may be better tolerated with robust reduction of LDL-C/non-HDL-C
- Once a new regime is tolerated, dose/frequency can be up-titrated slowly to achieve LDL-C/non-HDL-C goals with minimal or no muscle complaints

It is important to note that cardiovascular benefits have not been proven for all the above approaches but any reduction of LDL-C/non-HDL-C is beneficial.

LDL-C lowering options for patients with genuine statin intolerance

- Refer to the AAC Lipid Management Algorithm. (click here)
- Consider ezetimibe, (NICE TA 385) therapy as per algorithm
- Consider PCSK9i if eligible for treatment according to NICE TA 393, 394

Non-muscle related statin side effects

May vary between different statins. In clinical trials some side effects often associated with statins are not statistically different from placebo.

Most commonly reported: gastrointestinal disturbance and asymptomatic increases in hepatic transaminases (ALT or AST). May affect up to 1 in 10 statin users.

Rarer side effects include: Hepatotoxicity, new onset Type 2 Diabetes (benefits outweigh risk, do not stop statin), Renal insufficiency, proteinuria, Neurocognitive and neurological impairments (no apparent link from RCTs), Intracranial haemorrhage (conflicting evidence, benefit outweigh possible harm), Interstitial lung disease, Pancreatitis, Skin disorders including alopecia, Lupus-like reaction, Sleep disturbance, headache, dizziness, fatigue, depression, sexual dysfunction.

Management: If symptoms appear statin related, consider de-challenge and re-challenge or change to a different statin (e.g. hydrophilic instead of lipophilic).
Liver enzyme abnormalities - minor increases in liver enzymes (< 2x ULN) may be seen within the first three months of statin therapy; temporary discontinuation and further assessment is warranted if levels exceed 3x ULN. Several studies have confirmed that the cardiovascular benefits of statin treatment in high-risk populations outweigh the rare adverse effects, such as rhabdomyolysis.

Authors: Dr Rani Khatib & Dr Dermot Neely on behalf of the AAC Clinical Subgroup. June 2020. Review date: June 2021. Pathway endorsed by NICE July 2020. Please refer to the Lipid Management Pathway and Full List of References (click here).

ACCELERATED ACCESS COLLABORATIVE

NHS

04

Lipid Optimisation in Peripheral Arterial Disease

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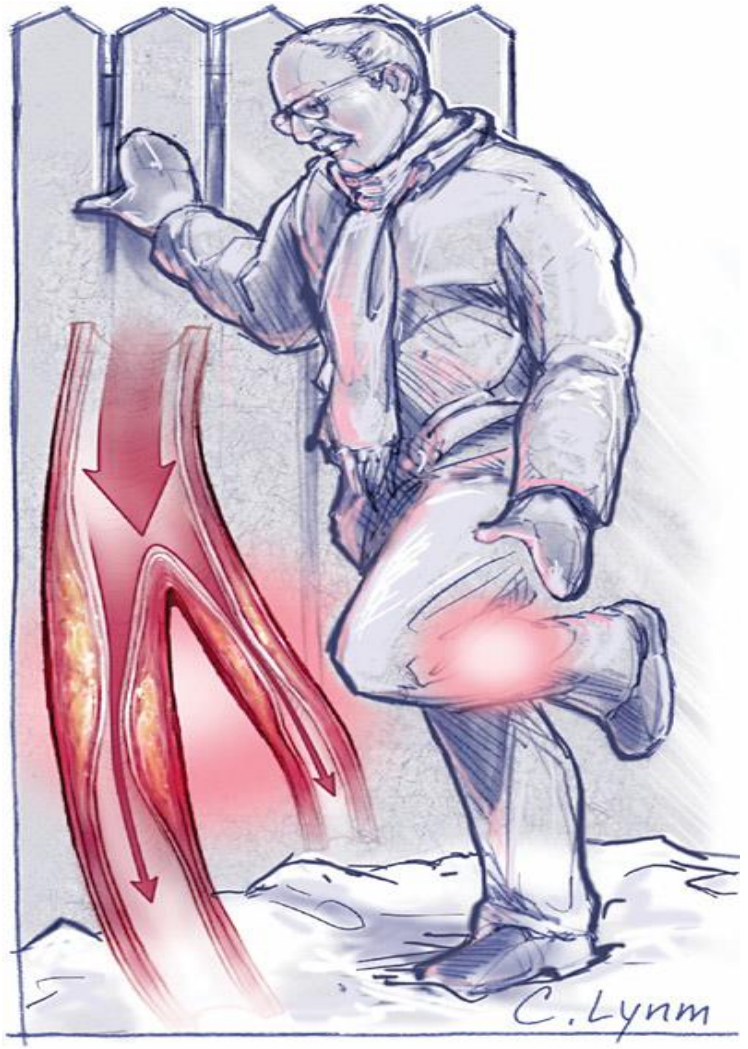
Peripheral Arterial Disease

- **Reduction in blood flow through large peripheral arteries (typically legs)**
- Atherosclerosis
- Thrombo-embolus
- Vasculitis
- External compression
- Rare vascular wall abnormalities (e.g. CAD)

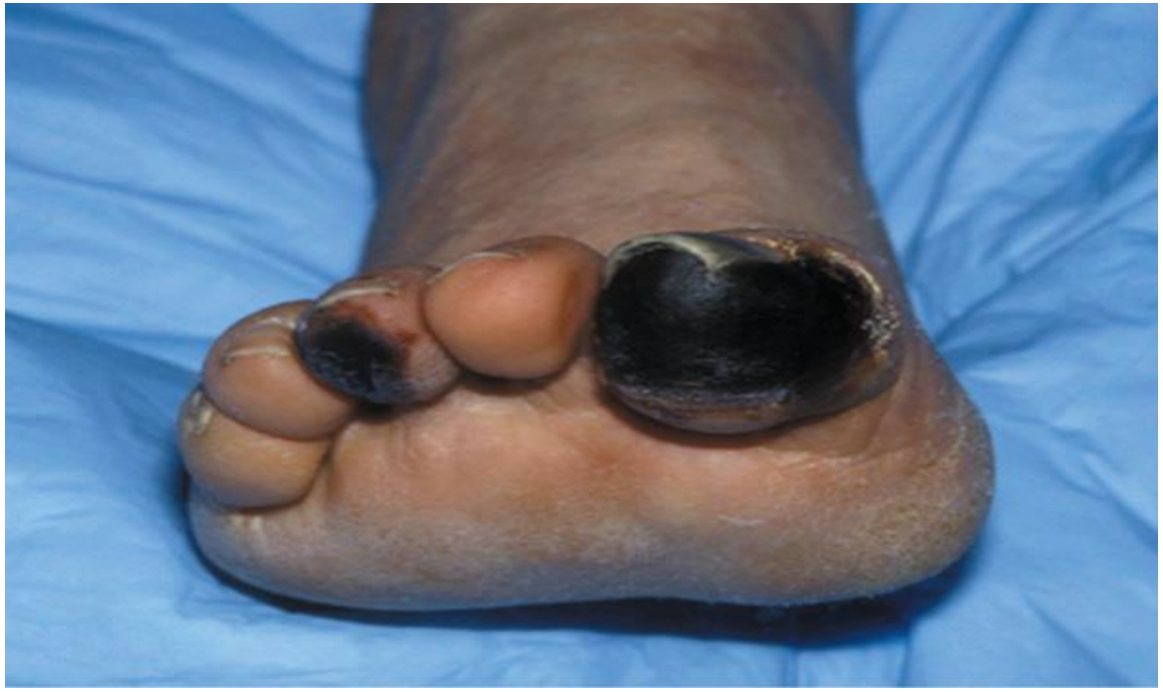
Intermittent Claudication

- Intermittent pain in muscles
- Induced by exercise
- Predictable distance
- Rapidly relived by rest
- Worse up hill / cold weather
- Limits walking distance / ADLs
- Not limb threatening
- **98%** will retain their leg
- **80%** stable over 5



Chronic Limb Threatening Ischaemia

- Vascular rest pain
- Typically at night in feet
- Tissue loss (>2/52)
  - Ulceration
  - Gangrene
  - Superadded Infection
- WIFI Score
- High risk of limb loss
- High benefit of Revascularisation
- High mortality (25% at 1 year of diagnosis)



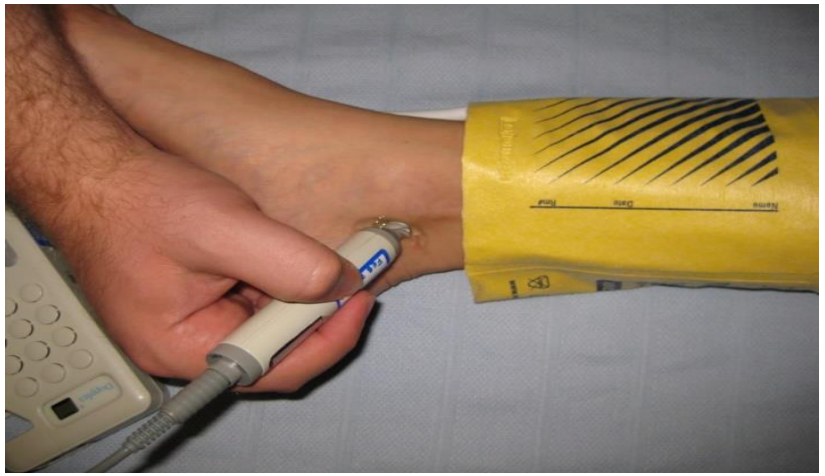
Diagnosis

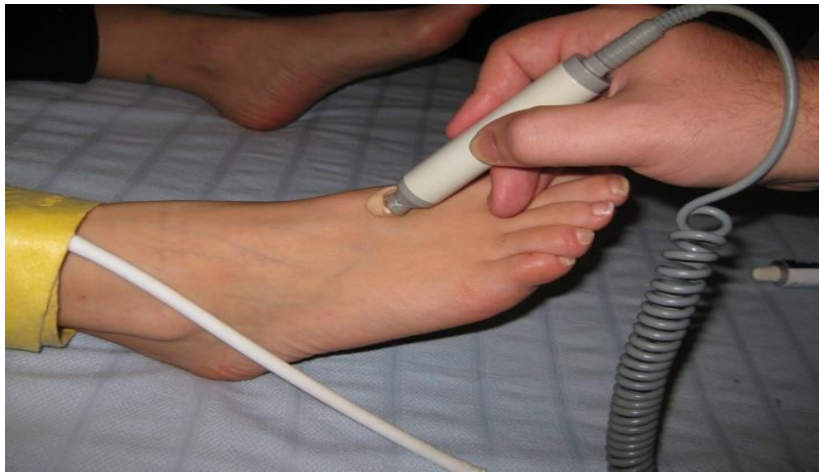
- Vascular examination
- Physiological bedside tests

ABPI*

>1.2	Calcified
0.9-1.2	**Normal**
0.5-0.9	PAD
<0.5	CLI

Toe pressure <60mmHg

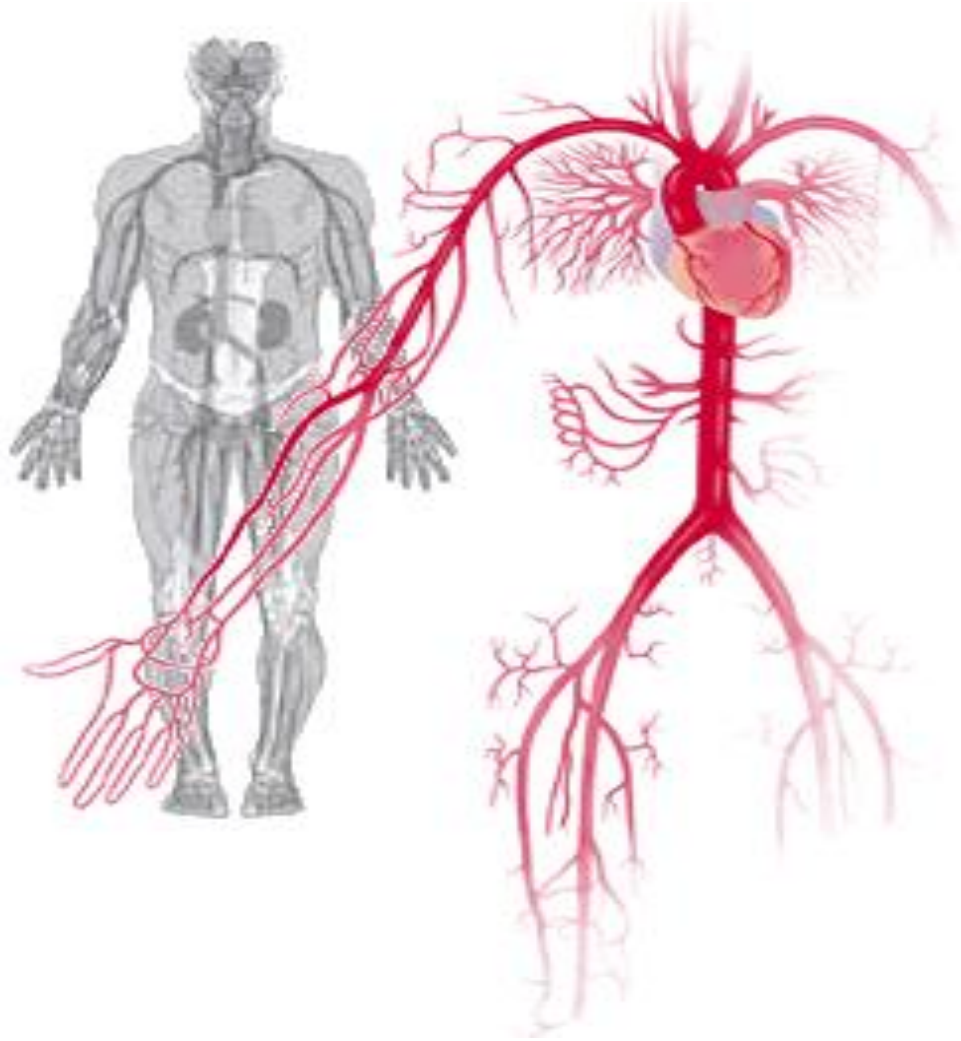




* Caution in diabetes or CKD

Peripheral Arterial Disease

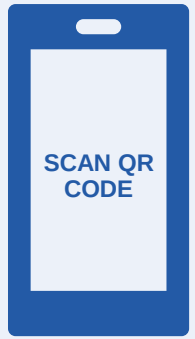
- **~237million** cases worldwide
- ¼ of the global burden of CVD
- Majority are claudicants
- **1-2%** risk of MALE/yr1,2
- **5-10%** risk of MACE/yr1,2
- Highest risk of future MACE
- High proportion of polyvascular disease
- **75%** of claudicants die from MACE
- Massively medically undertreated3



1. EUCLID: Hiatt et al. NEJM 2017; 376: 32-40; 2. FOURIER: Sabatine et al. NEJM 2017; 376: 1713-1722; 3. VERN: Saratzis et al. Br J Surg 2019; 106: 168-77.

NICE PAD Guidelines CG147

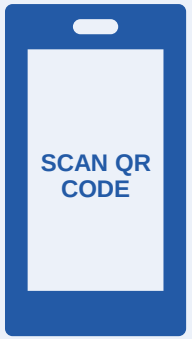
- Intermittent Claudication
  - Supervised exercise
  - Smoking cessation
  - Optimal medical therapy
  - Vasodilators
  - Angioplasty if severely disabling
- CLTI
  - Urgent assessment by vascular surgery
  - Imaging with a view to revascularisation
  - MDT review of all patients



Peripheral arterial disease: diagnosis and management; Clinical Guideline 147, Aug 2012; updated Dec 2020

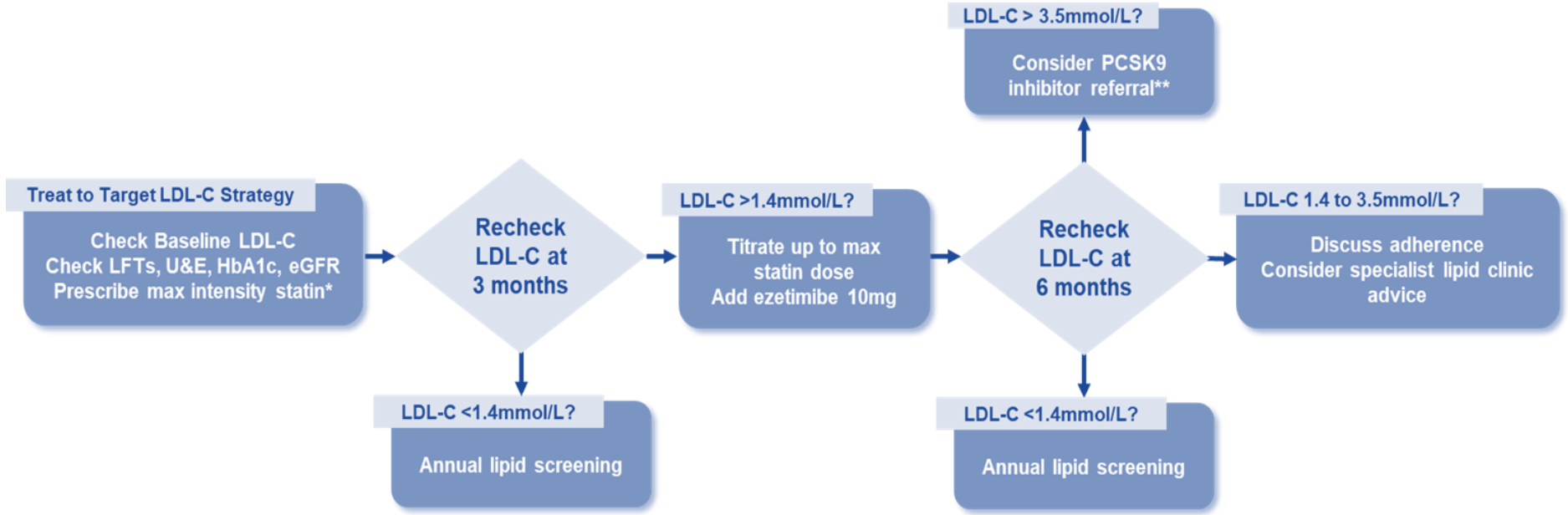
NICE PAD Guidelines CG147

- Secondary prevention of CVD
  - Smoking cessation
  - **Lipid modification / statins**
  - Anti-platelet therapy
  - Prevention, diagnosis, management
    - Diabetes
    - Hypertension
- Lipid targets:
  - LDL cholesterol <1.8mmol/L or 40% reduction (NICE)
  - LDL cholesterol <1.4mmol/L or 50% reduction (ESC)
  - LDL cholesterol >3.5mmol/L on maximal therapy, PCSK9i



Peripheral arterial disease: diagnosis and management; Clinical Guideline 147, Aug 2012; updated Dec 2020

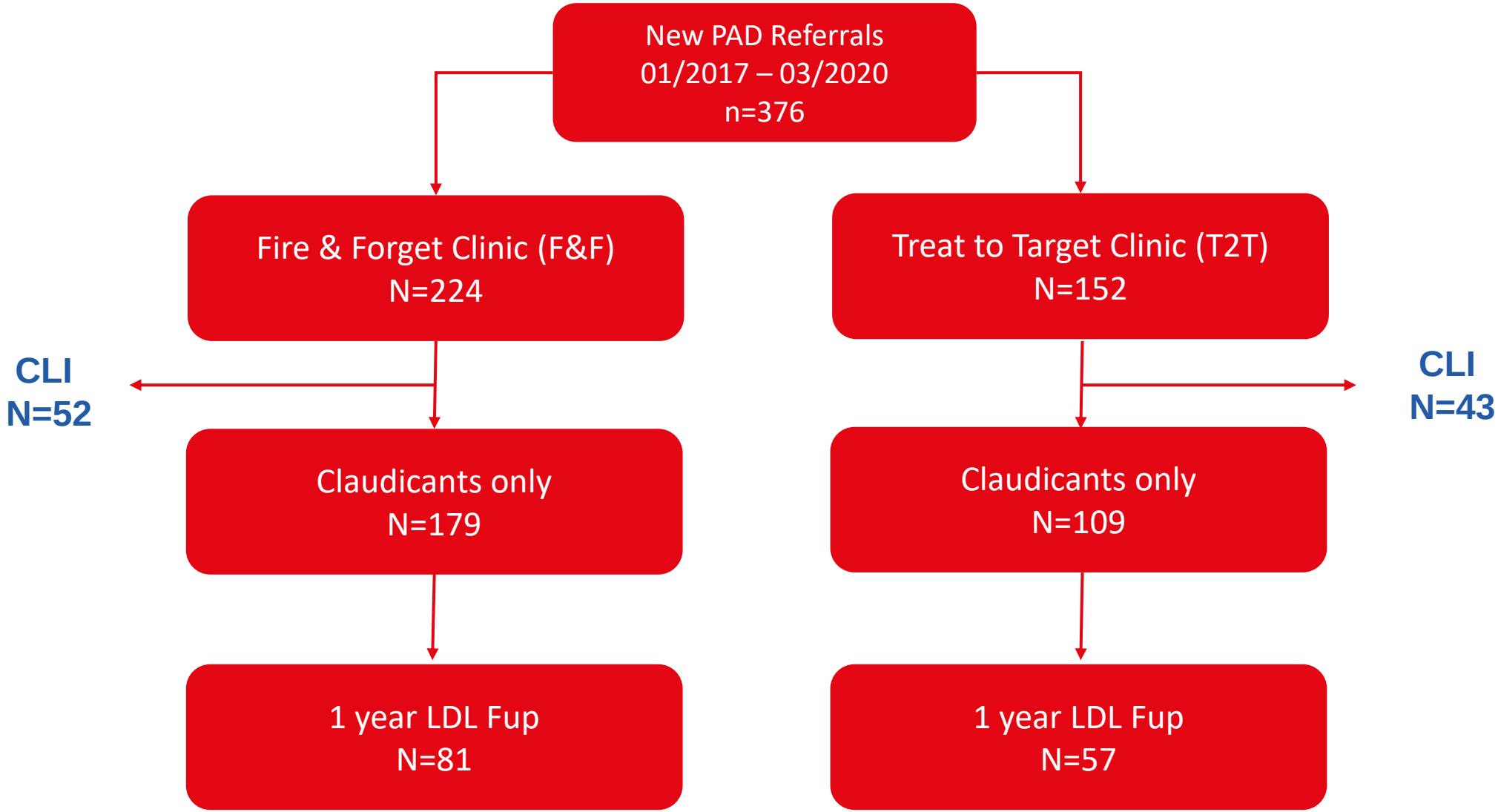
Treat to target LDL approach



Sucharitkul et al 2021, Annals of Vascular Surgery

NICE statin intolerance algorithm

NICE statin intensities

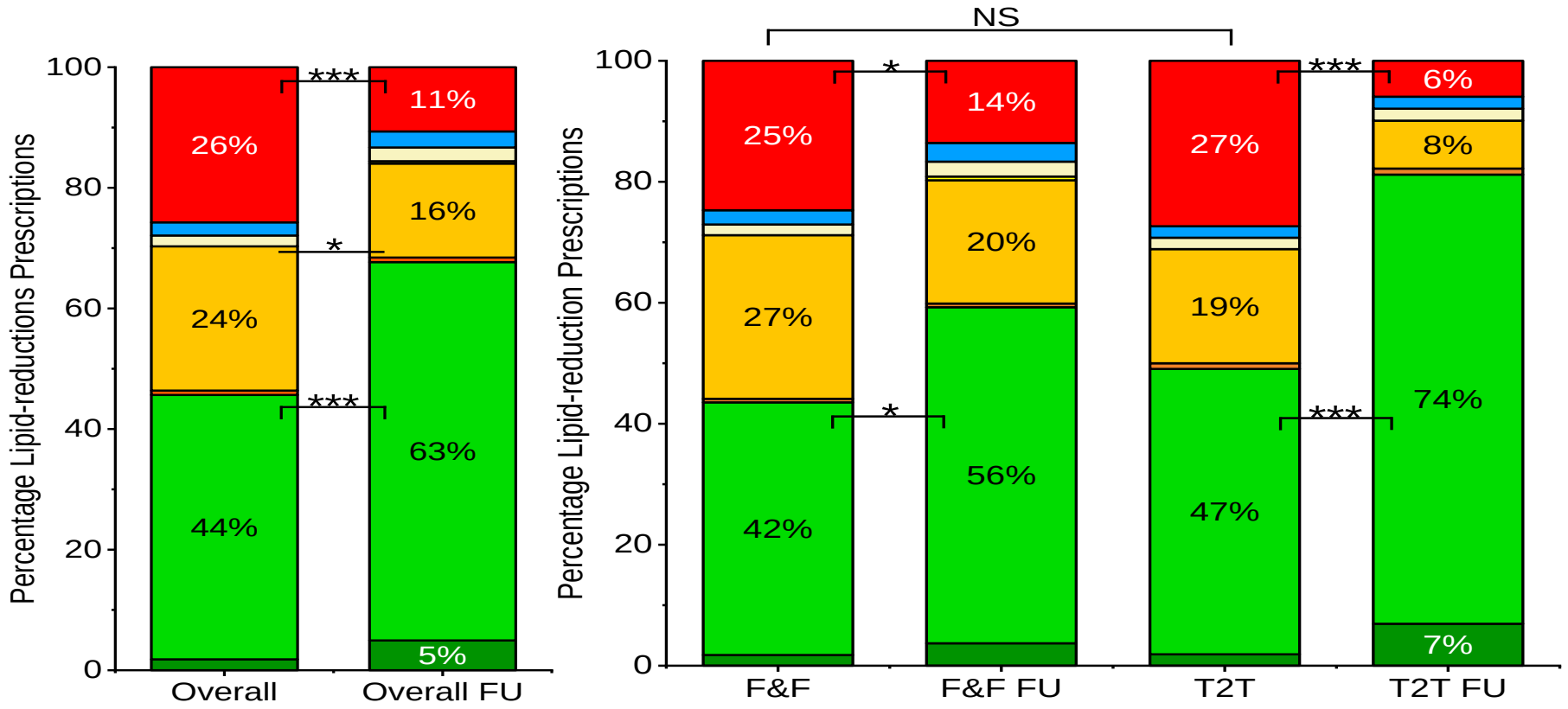


Sucharitkul et al. unpublished

Baseline Demographics				
Characteristics no. (%)	**F&F I/II n= 179**	**T2T I/II n=109**	**p**	**adj. p**
Age yrs mean (SD)	66.8 (±9.8)	68.3 (±10.9)	0.2522	0.7100
Female	46 (26.7)	32 (29.4)	0.6824	1.0000
White ethnicity (of given)	148 (86.0)	77 (70.6)	0.7539	1.0000
BMI kg/m² mean (SD)	27.2 (±4.9)	27.2 (±4.6)	0.8214	1.0000
Fontaine Staging no. (%)				
I	5 (2.9)	1 (0.9)	0.4102	0.9600
II a/b	167 (97.1)	108 (99.1)	0.4102	0.8200
Comorbidities no. (%)				
Diabetes T1/2	56 (32.6)	37 (33.9)	0.7952	1.0000
IHD	52 (30.2)	33 (30.3)	1.0000	1.0000
COPD	43 (25.0)	17 (15.6)	0.0730	0.2600
Hypertension	98 (57.0)	76 (69.7)	0.0331	0.2300
CKD	96 (55.8)	73 (67.0)	0.0598	0.2800
Cerebrovascular Hx	20 (11.6)	25 (22.9)	0.0186	0.2600
Mono PAD	108 (62.8)	67 (61.5)	0.8996	1.0000
Poly PAD	64 (37.2)	42 (38.5)	0.8996	0.9700

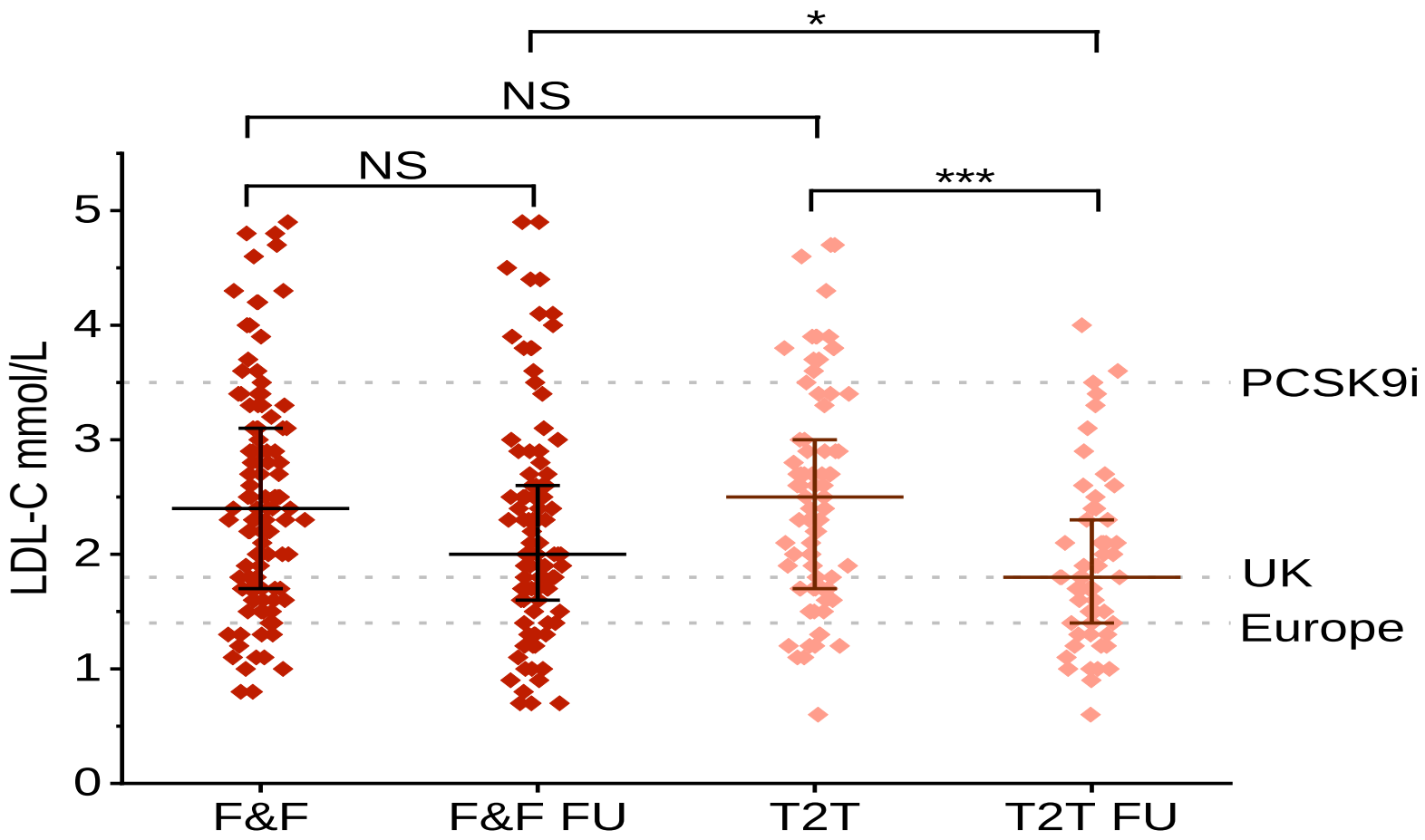
Sucharitkul et al. unpublished

Results: Lipid therapy



Sucharitkul et al. unpublished

Results: LDL-c



Sucharitkul et al. unpublished

Summary

- PAD means high cardiovascular risk
- The diagnosis of PAD should be sought out
- All PAD patients need intensive lipid treatment
- A T2T approach to an LDL <1.8mmol/L is advised
- Rapid commencement of highest intensity statin
- Knowledge of the de-challenge/re-challenge statin intolerance approach
- Re-check LDL at 3 months and use Ezetimibe
- Refer to lipid clinic if LDL >3.5mmol/L on maximal treatment or intolerance problems

Conclusion

- People with CV events or with PAD are at high risk of further events
- Effective therapies to reduce risk through intensive lipid lowering are available but are currently under-deployed
- Measure and re-measure full lipids profile appropriately.
- Address statin reluctance & intolerance.
- Optimise Lipid Lowering Therapy and use Combinations.
- Identify and address non-adherence.

05 Q&A

Next steps:

.Join us for the next webinar:

Weds 8th December 11am-12 noon: ‘Novel Therapies - Now and Future’.

Dr Ameet Bakhai, Professor Kosh Ray and Dr Yassir Javaid will integrate primary and secondary care perspectives, walk us through the novel medicines in the lipid pathway and discuss what is on the horizon.

Dr Ameet Bakhai
Consultant Cardiologist and Research Director. Royal Free London NHS Foundation Trust

Professor Kosh Ray
Professor of Public Heath in the Department of Public Health and Primary Care at Imperial College London as well as Honorary Consultant Cardiologist at the Imperial College NHS Trust.

Dr Yassir Javaid
Cardiovascular Lead Northamptonshire CCG 2013 - present
Primary Care CVD lead East Midlands Clinical Network (2013-2020)

Join us for an informal case based interactive clinic on post CVD event management: Date 1st December 11am-12 noon

All programme content, recordings and next webinar bookings will be housed in the HEART UK pages. Visit the site for the new e-Learning modules on Identifying FH in primary care, Statin Intolerance, and the Lipid Management Pathway

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Thank you

This webinar has now finished.

Today's slides and recording will be available after the webinar on the HEART UK pages. Visit the site for the **new** e-Learning modules on diet launching in November. Identifying FH in primary care, Statin Intolerance, and the Lipid Management Pathway modules are also available.

All programme content, recordings and next webinar bookings will be housed here: https://www.heartuk.org.uk/tackling-cholesterol-together/home