Updates in Lipid Management

Sadeer Fhadil MFRPSI MRPharmS IPresc

Lead Cardiac Pharmacist

Barts Health NHS Trust

Objectives

1. Why the focus on lipids?
2. QOF indicators
3. How? Case study

CVD is responsible for 24% of all deaths in the UK[1]

CVD leads to 1 death every 3 minutes[1]

CVD healthcare costs total £9 billion per year[1]

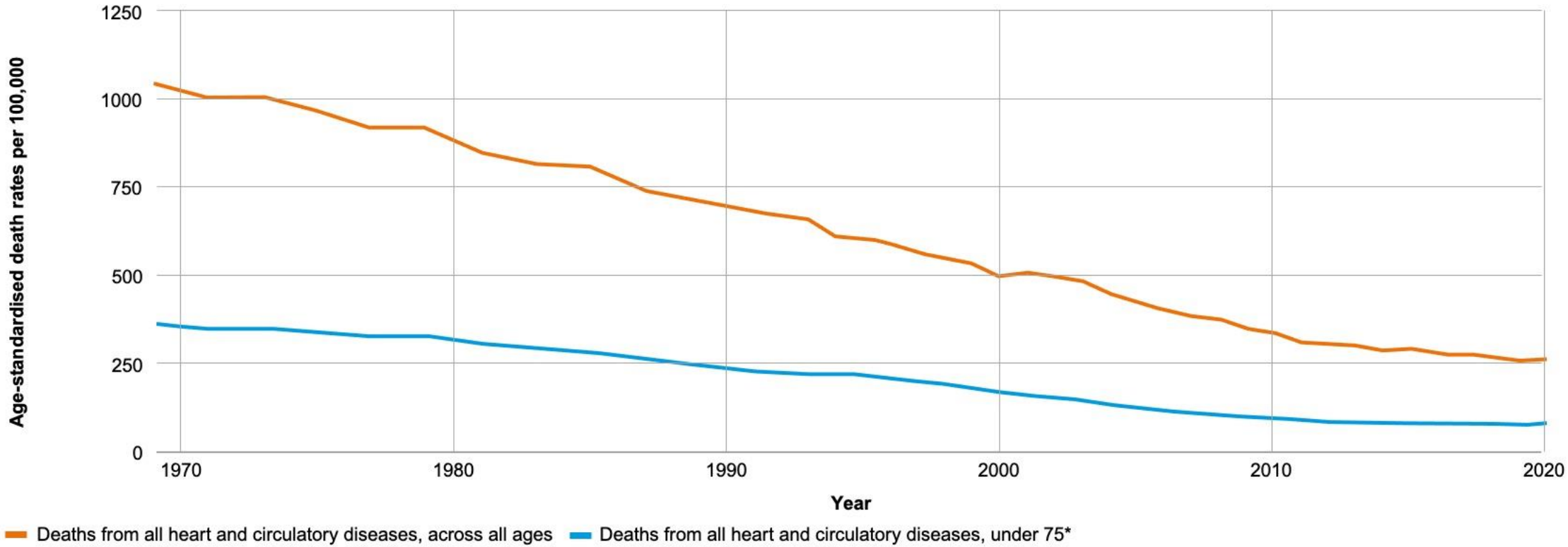
NHS Long Term Plan names CVD as "The single biggest area where the NHS can save lives over the next 10 years"[2]

CVD, cardiovascular disease; NHS, National Health Service.

1. British Heart Foundation. UK Factsheet January 2022. Available at: https://www.bhf.org.uk/-/media/files/research/heart-statistics/bhf-cvd-statistics---uk-factsheet.pdf. Accessed August 2022. **2.** NHS. The NHS Long Term Plan. Available at: https://www.longtermplan.nhs.uk/wp-content/uploads/2019/08/nhs-long-term-plan-version-1.2.pdf. Accessed August 2022.

After four decades of steady decline in CVD deaths, improvement has recently slowed[1]

Deaths from all heart and circulatory diseases, in the UK[1]



*Premature deaths.
CVD, cardiovascular disease.
1. British Heart Foundation. Heart and Circulatory Disease Statistics 2022. Available at: https://www.bhf.org.uk/what-we-do/our-research/heart-statistics/heart-statistics-publications/cardiovascular-disease-statistics-2022. Accessed August 2022.

ASCVD risk is impacted by a range of modifiable and non-modifiable factors[1]

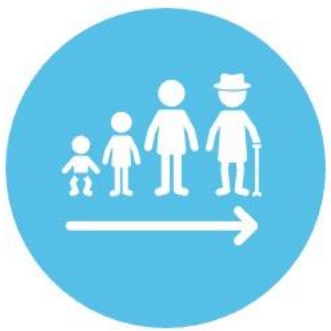
ApoB-containing lipoproteins (inc. LDL-C)

High blood pressure

Smoking

Diabetes

Adiposity



Age

Ethnicity

Psychosocial stress

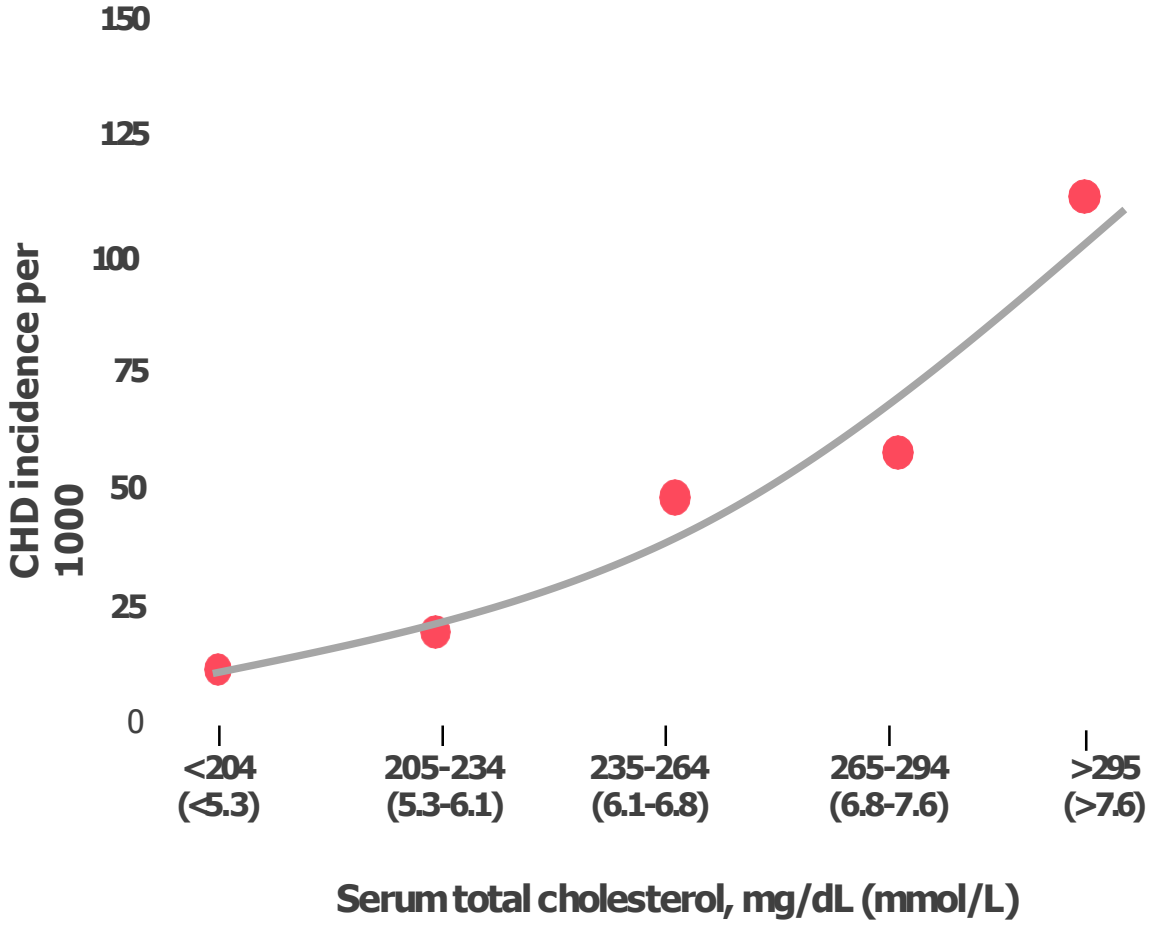
Physical activity & exercise

Nutrition and alcohol

ApoB, apolipoprotein B; ASCVD, atherosclerotic cardiovascular disease; LDL-C, low-density lipoprotein cholesterol.

1. Visseren FLJ, *et al*. *Eur Heart J*. 2021;42(34):3227–3337.

The Framingham Study: Relationship Between Cholesterol and CHD Risk

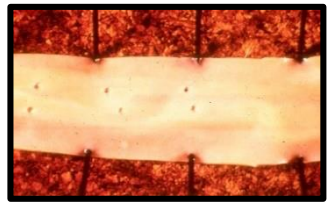


Castelli WP. *Am J Med*. 1984;**76**:4-12
https://pubmed.ncbi.nlm.nih.gov/6702862/

Nikolai Nikolaevich Anitschkow (1885–1964)

Lots of Egg Yolk

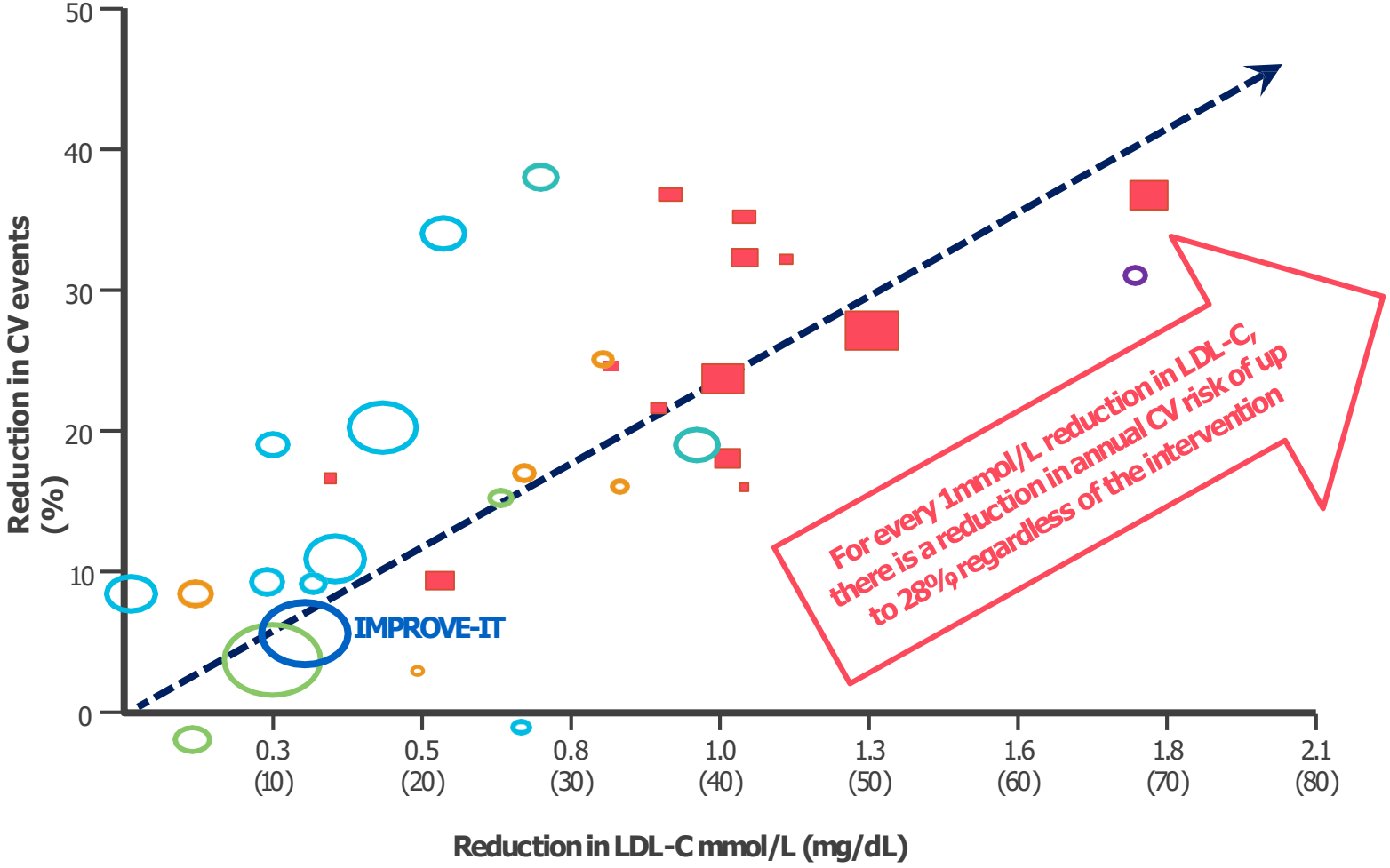
Vegetables



"Without cholesterol there can be no atherosclerosis"

Stehbens WE. Anitschkow and the cholesterol over-fed rabbit. Cardiovasc Pathol 1999;8:177-8.
Finking G, Hanke H. Nikolaj Nikolajewitsch Anitschkow (1885-1964) established the cholesterol-fed rabbit as a model.
Igor E. Konstantinov, Nicolai Mejevoi, and Nikolai M. Anichkov. Nikolai N. Anichkov and His Theory of Atherosclerosis.
Tex Heart Inst J. 2006; 33(4): 417–423
.https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1764970/
https://pubmed.ncbi.nlm.nih.gov/10722241/

Lower achieved LDL-C correlates to lower risk of CV events, regardless of intervention



- Ezetimibe
- Fibrate
- Bile acid resin
- Niacin
- Diet/unsaturated fatty acid
- Ileal bypass
- CTTC trials (statin)

For illustrative purposes only; individual trials should not be directly compared.

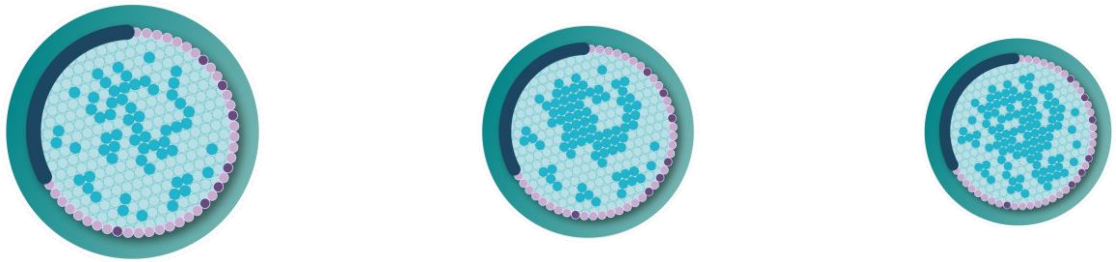
Data from studies of non-statin lipid-lowering medications superimposed upon data from the CTTC 2005 meta-analysis. The IMPROVE-IT trial was adequately powered to show the efficacy on incremental LDL-C lowering on CV outcomes. CV, cardiovascular; CTTC, Cholesterol Treatment Trialists' Collaboration; LDL-C Low-density lipoprotein cholesterol Baigent, et al. *Lancet*. 2005:366;1267–1278; CTT Collaboration. *Lancet*. 2010;376:1670–1681; Cannon, et al. *N Engl J Med.* 2015;372:2387–239
https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(05)67394-1/fulltext

The key initiating event in atherogenesis is the retention of LDL-C and other cholesterol-rich apo B-containing lipoproteins within the arterial wall[1]

Very-low-density lipoprotein (VLDL)[2]

Intermediate-density lipoprotein (IDL)[2]

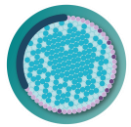
Low-density lipoprotein (LDL)[2]



Atherogenic lipoprotein[2,3]

- Can be taken up and retained by arterial cell walls
- Associated with the development of atherosclerosis

High-density lipoprotein (HDL)[2]



Anti-atherogenic lipoprotein[3]

- Plays an important role in reversing cholesterol transport

LDL is the **most abundant atherogenic lipoprotein in plasma** and the key deliverer of cholesterol to the artery wall[4]

apo B: apolipoprotein B; LDL-C: low-density lipoprotein cholesterol.

1. ESC/EAS Guidelines for the management of dyslipidaemias. Mach F, et al. Eur Heart J 2020;41:111–188; 2. Holmes MV, Ala-Korpela M. Nat Rev Cardiol. 2019;16:197–98; 3. Feingold KR. Introduction to Lipids and Lipoproteins. [Updated 2021 Jan 19]. In: Feingold KR, et al. Endotext [Internet]: MDText.com, Inc. Available from: https://www.ncbi.nlm.nih.gov/books/NBK305896/ (accessed September 2021); 4. Borén J, et al. Eur Heart J 2020;41:2313–2330.

As LDL-C levels increase, the development of atherosclerotic plaques increases in a dose-dependent manner[1,2]

Mechanisms underlying the entry, retention, and accumulation of LDL in arterial walls[3]

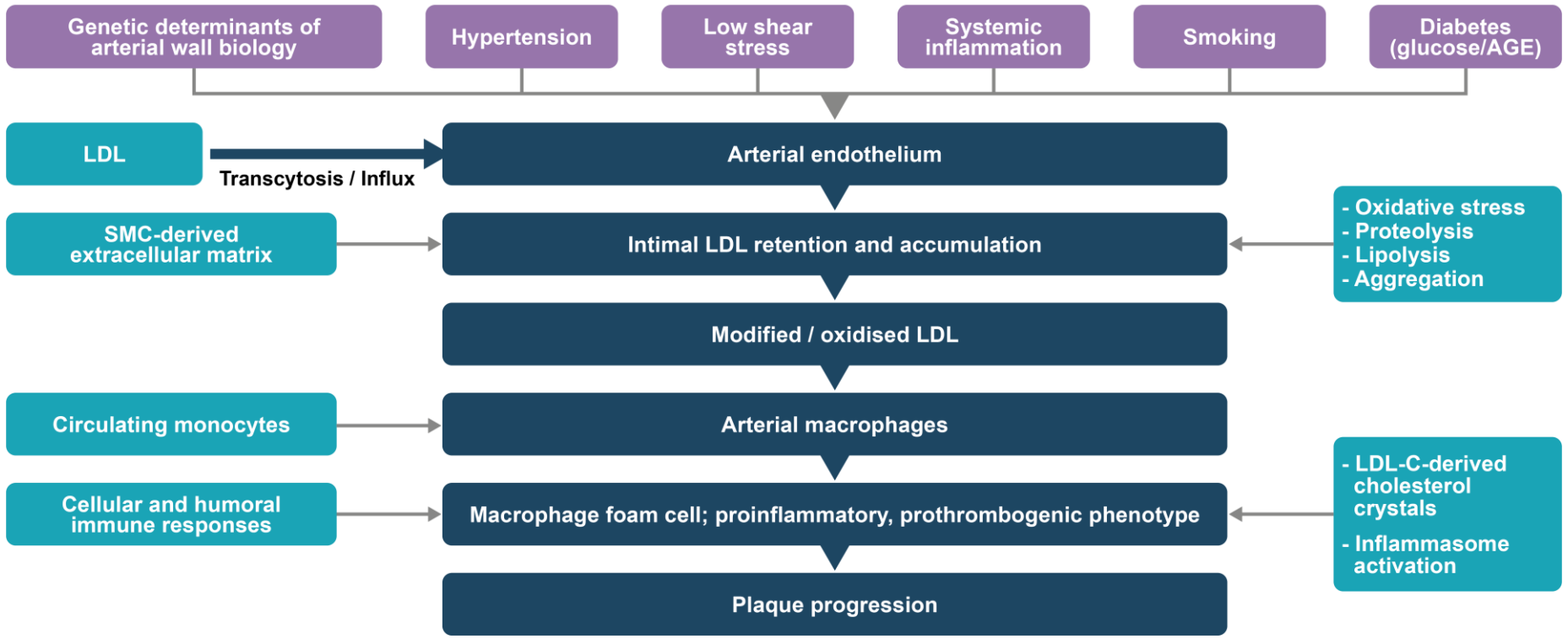


Figure adapted from Borén J, et al. 2020.

In general, **the higher the LDL-C level the faster the plaques evolve[4]**

AGE: advanced glycation end-product; LDL: low-density lipoprotein; LDL-C: low density lipoprotein-cholesterol; SMC: smooth muscle cells.

1. ESC/EAS Guidelines for the management of dyslipidaemias. Mach F, et al. Eur Heart J 2020;41:111–188; 2. Ference BA et al. Eur Heart J 2017;38:2459–2472; 3. Borén J, et al. Eur Heart J 2020;41:2313–2330; 4. Goldstein J, Brown M. Cell 2015;161(1):161–172..

Rupturing of atherosclerotic plaques can lead to CV events, such as stroke, myocardial infarction and ultimately cardiac death[1,2]

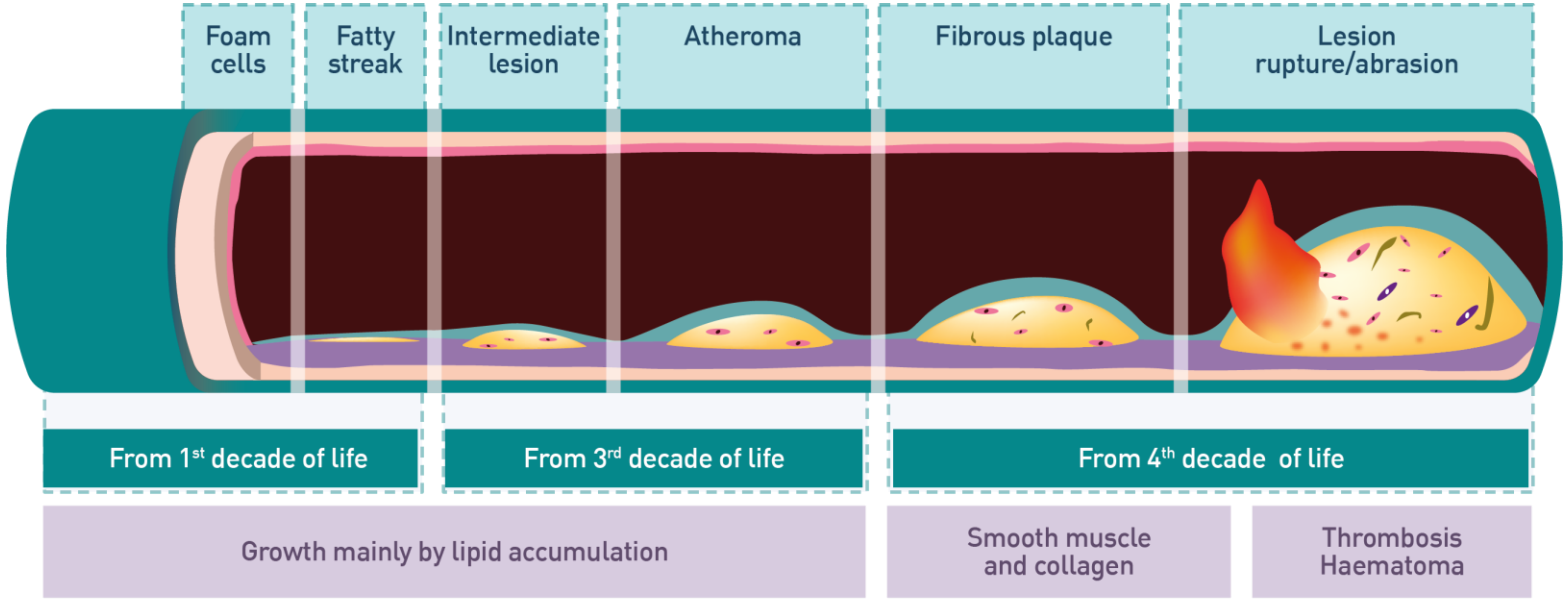


Figure adapted from Pepine CJ, et al. 1998.

Unstable or ruptured plaques block blood flow and increase the risk of CV events; early and robust treatment may reduce the risk of CV events[2]

CV: cardiovascular.

1. Fleg JL, et al. JACC Cardiovasc Imaging 2012;5:941–955; 2. ESC/EAS Guidelines for the management of dyslipidaemias. Mach F, et al. Eur Heart J 2020;41:111–188; 3. Pepine CJ. Am J Cardiol. 1998;82:23S-27S

Treatment targets for LDL-C

ESC/EAS recommendations across CV risk categories[1]

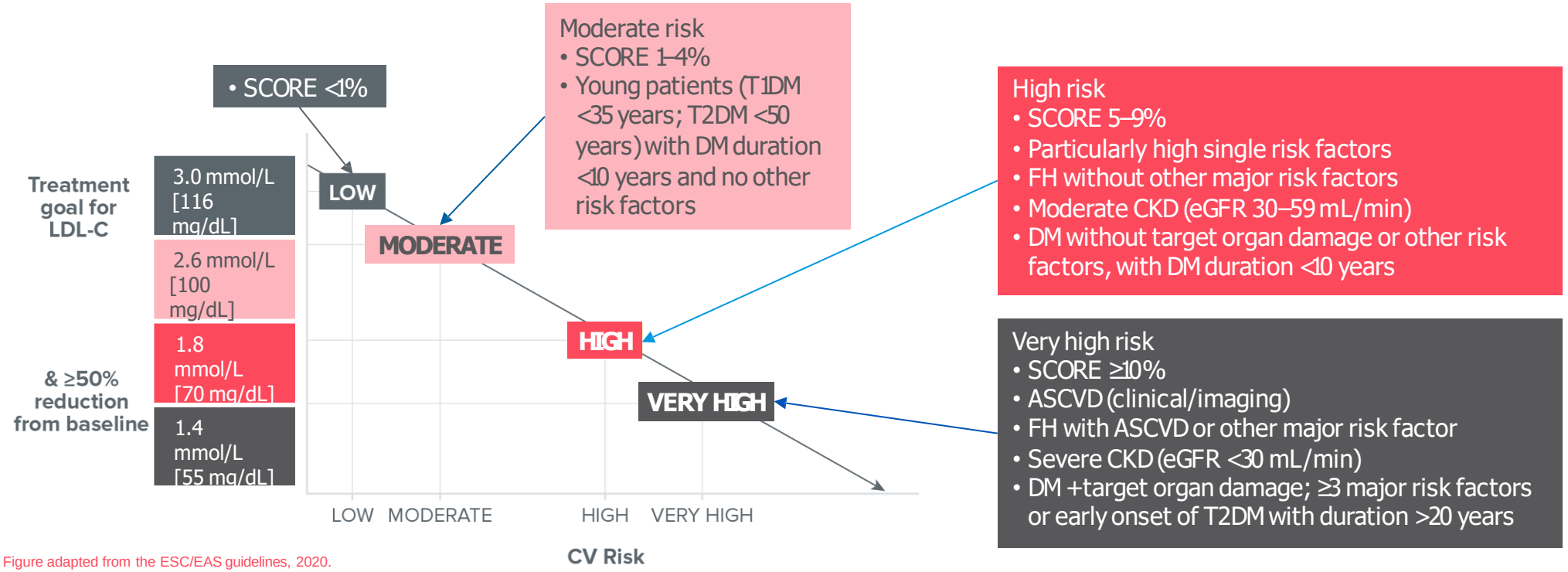


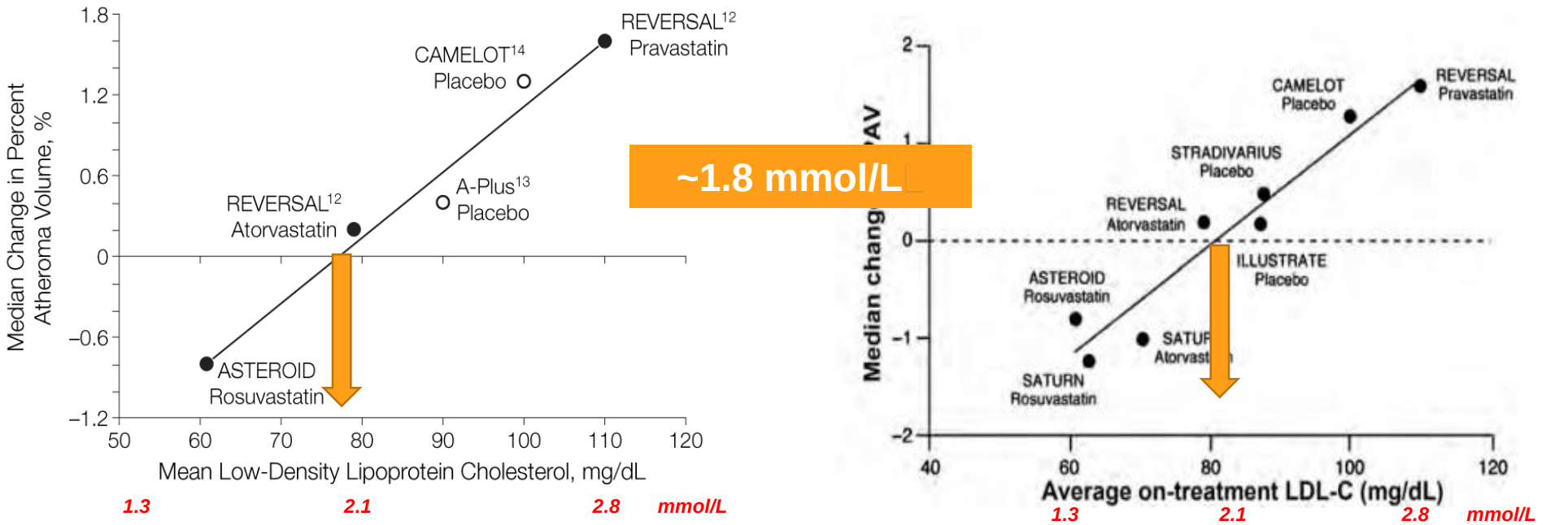
Figure adapted from the ESC/EAS guidelines, 2020.

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

2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk | European Heart Journal | Oxford Academic (oup.com)

Mach F, et al. ESC/EAS Guidelines for the management of dyslipidaemias. 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).

LDL-C and plaque regression



Adapted from Puri *et al.* 2013.

SATURN: The Study of Coronary Atheroma by Intravascular Ultrasound: Effect of Rosuvastatin vs. Atorvastatin; ASTEROID: A Study to Evaluate the Effect of Rosuvastatin on Intravascular Ultrasound-Derived Coronary Atheroma Burden; ILLUSTRATE: Investigation of Lipid Level Management Using Coronary Ultrasound To Assess Reduction of Atherosclerosis by CETP Inhibition and HDL Elevation; REVERSAL: Reversal of Atherosclerosis with Aggressive Lipid Lowering; STRADIVARIUS: Strategy To Reduce Atherosclerosis Development InVolving Administration of Rimonabant—the Intravascular Ultrasound Study; CAMELOT: Comparison of Amlodipine vs. Enalapril to Limit Occurrences of Thrombosis.

Puri *et al.* Factors underlying regression of coronary atheroma with potent statin therapy. *EHJ* 2013;34(**24**):1818-1825

Objectives

1. Why the focus on lipids?
2. QOF indicators
3. How? Case study

QOF Indicators 2023/4

Table 1. Indicators related to lipid modification in the Quality and Outcomes Framework (QOF) of the General Medical Services (GMS) contract.

CHOL001 Percentage of patients on the QOF Coronary Heart Disease, Peripheral Arterial Disease, Stroke/TIA or Chronic Kidney Disease Register who are currently prescribed a statin, or where a statin is declined or clinically unsuitable, another lipid lowering therapy	14	70-95%
CHOL002 Percentage of patients on the QOF Coronary Heart Disease, Peripheral Arterial Disease, or Stroke/TIA Register, who have a recording of non HDL cholesterol in the preceding 12 months that is lower than 2.5 mmol/L, or where non HDL cholesterol is not recorded a recording of LDL cholesterol in the preceding 12 months that is lower than 1.8 mmol/L	16	20-35%
Data from: [NHS England, 2023]		

ceg

QOF Cholesterol Control and Lipid Management

'Cholesterol Control and Lipid Management' was introduced to QOF this year, designed to support high-risk CVD patients who could benefit from further combination therapies.

Practices should be aware: At the point data is collected to assess QOF achievement, patients will need to have a prescription for a statin/lipid lowering therapy on their records (CHOL001) and have a non-HDLC or LDLC reading at or below targets within the 12-month QOF Period (CHOL002). UK guidance indicates a 12-week wait from initiation of therapy to re-testing of lipids, so practices will need to have initiated the above activity by 31 December 2023 to get the appropriate follow-up test results in the system in time for the QOF data collection.

Steps to take now to maximise your achievement

We suggest reviewing your QOF indicators now to identify the patients who either:

1. Need a statin or alternative lipid lowering therapy to be prescribed (CHOL001)
2. Have not had a Full Lipid Profile in the last 12 months (CHOL002)
3. Have not had a non-HDL cholesterol below 2.5 (or LDL below 1.8)

For those in **Group 1**, the statin/lipid lowering therapy (LLT) needs to be prescribed after 1 October – please check they have a medication on their repeat list in advance of this date. If not, consider inviting them to discuss starting one. You may want to block out time a month after for your pharmacist to ensure patients have been prescribed their statin/LLT to meet the indicator.

In **Group 2**, there may be large numbers of patients who have not had a Full Lipid Profile blood test for over 12 months. To avoid overloading phlebotomy clinics, please consider staggering the recall. To aid the booking into practice clinics, consider using Accubook.

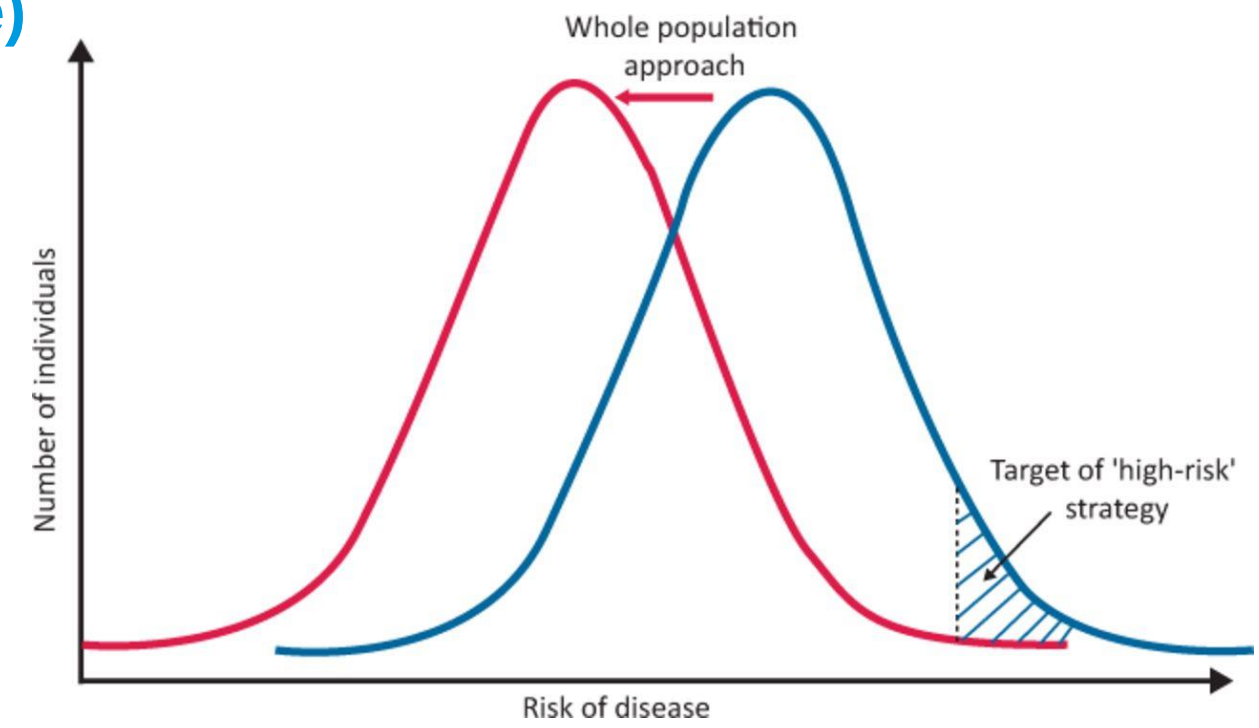
For patients in **Group 3**, review the medication, check adherence and lifestyle measures, and consider increasing statin dose, adding ezetimibe or injectable therapies (inclisiran/PCSK9). Add a reminder and explain to the patient that they need a repeat blood test in 3 months' time. Doing this earlier will give you enough time to work through this process and ensure the cholesterol blood

Essentially:

Aim to put everyone with CVD onto a high dose statin (or an alternative)

Get as many patients as possible down to an LDL <1.8mmol/L

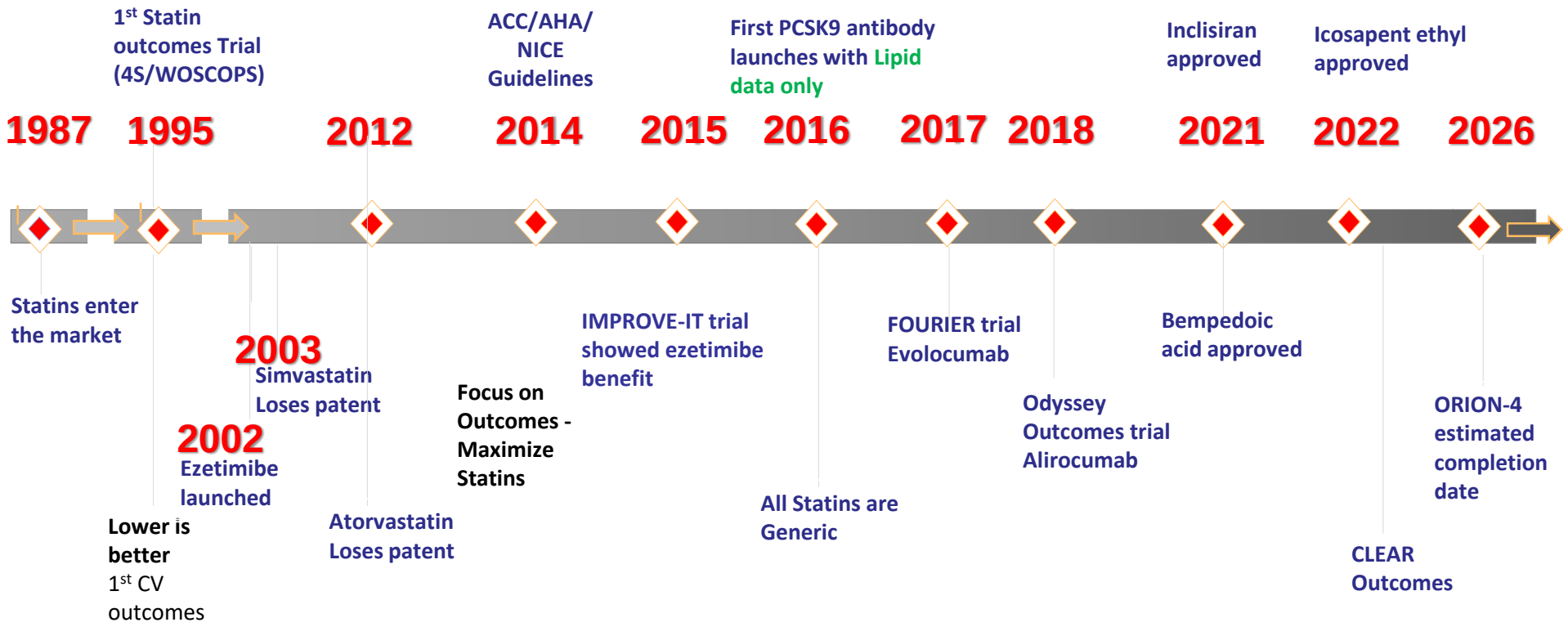
If you lower the LDL, you will lower the CVD risk, regardless of the starting level



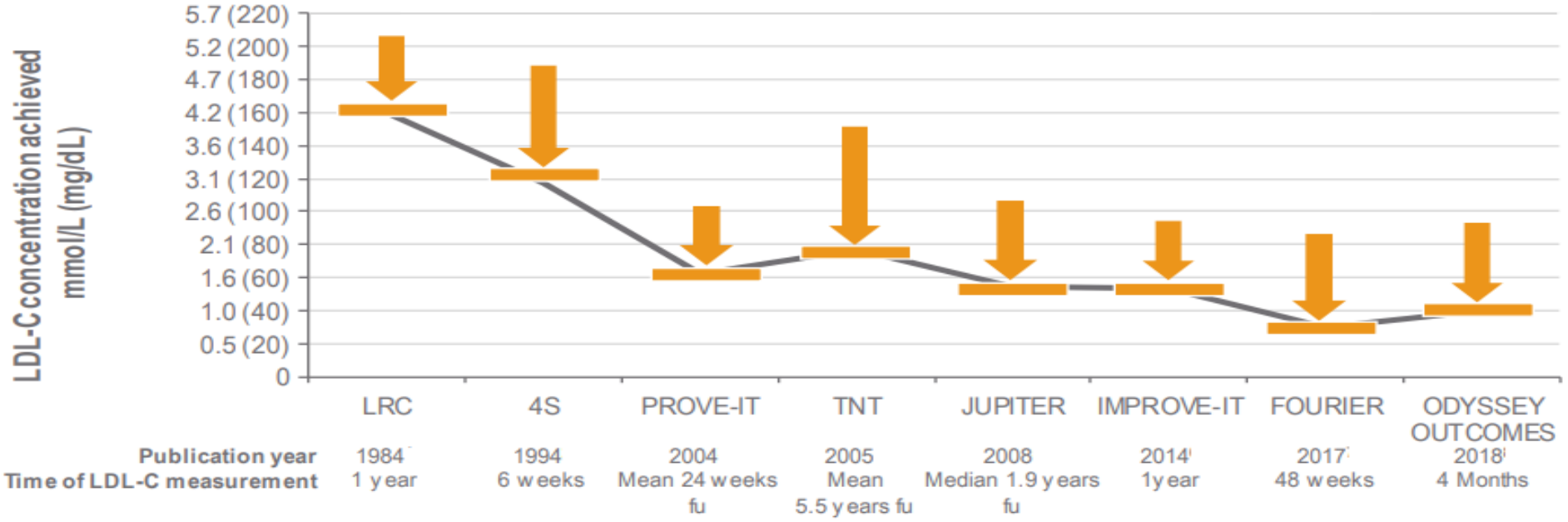
Objectives

1. Why the focus on lipids?
2. QOF indicators
3. How? Case study

Landscape & Timeline



Historical perspective of LDL-C levels achieved in major randomised controlled trials of lipid-lowering therapies[1,2]



Adapted from Masana, *et al. J Clin Lipidol*. 2010 and Schwartz, *et al. N Eng J Med*. 2018.

Arrows indicate the mean LDL decrease obtained in the study.

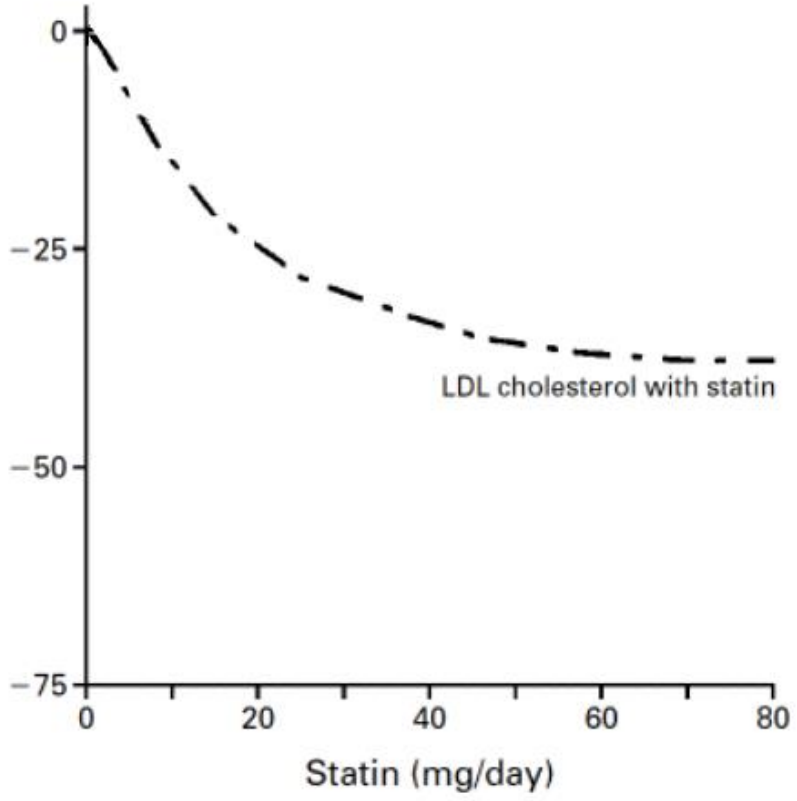
LDL-C, low-density lipoprotein-cholesterol.

1. Masana *et al.* Clinical and pathophysiological evidence supporting the safety of extremely low LDL levels - The zero-LDL hypothesis. *J Clin Lipidol.* 2018:12(2):292-299. 2. Schwartz GG, *et al.* Alirocumab and Cardiovascular Outcomes after Acute Coronary Syndrome. *N Eng J Med.* 2018;379(22):2097–2107 https://pubmed.ncbi.nlm.nih.gov/29398429/

Doubling the dose with statins does not double the effect on LDL-C reduction[1]

- The dose required to reduce serum LDL-C concentrations to a similar degree varies substantially among statins
- The response to dose increases is not proportional
- In general, a doubling of the dose above the minimal effective dose, decreases serum LDL-C concentrations by an additional 6%
- The maximal reduction in serum LDL-C concentrations induced by statin treatment ranges from 24–60%

Effect of treatment with statin on LDL-C concentrations



Adapted from Knopp. *N Engl J Med*, 1999.

LDL-C, low-density lipoprotein cholesterol.

1. Knopp RH. Drug treatment of lipid disorders. *New Engl J Med.* 1999;341:498–511.

Lowering LDL-C correlates with reduced CV risk[1]

Up to **80%** of patients with ASCVD* who use moderate or high intensity statins do not achieve the recommended LDL-C goal† of <1.8 mmol/L[2,3,4]

Additional LLTs are needed to complement current therapies and help uncontrolled patients achieve their LDL-C goals[2,3]

*Documented ASCVD included a history of acute MI, silent MI, unstable angina, coronary revascularisation procedures, clinically significant CHD, symptomatic peripheral arterial disease or cerebrovascular atherosclerotic disease.[3]

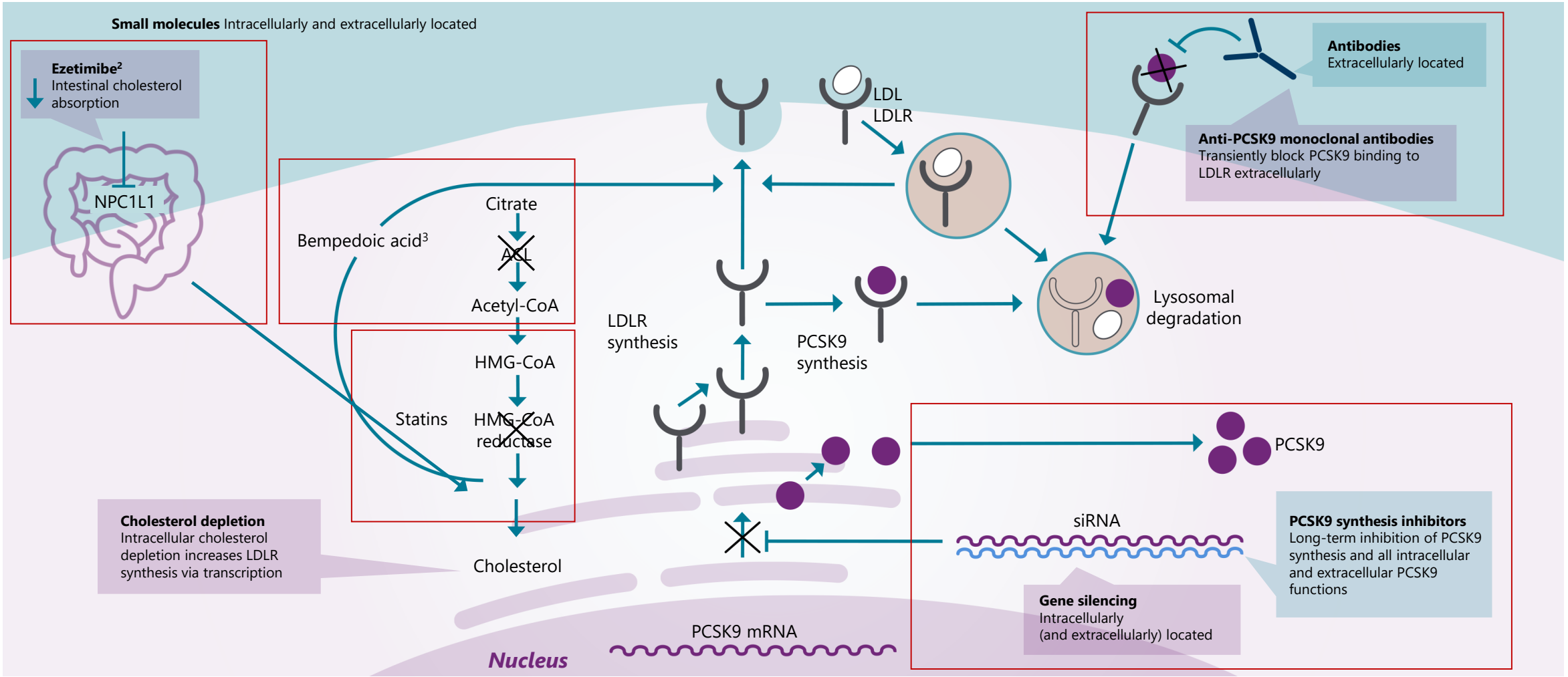
†The 2019 ESC/EAS Guidelines recommend an LDL-C reduction of ≥50% from baseline and LDL-C goals of 1.8 mmol/L and 1.4 mmol/L in high risk and very high risk patients, respectively.[3]

CI, confidence interval; CV, cardiovascular; CV, cardiovascular; LDL-C, low-density lipoprotein cholesterol; ASCVD, atherosclerotic cardiovascular disease; LLT, lipid lowering therapy.

1. Ference BA, *et al.* Reduction of low density lipoprotein-cholesterol and cardiovascular events with proprotein convertase subtilisin-kexin type 9 (PCSK9) inhibitors and statins: an analysis of FOURIER, SPIRE, and the Cholesterol Treatment Trialists Collaboration. *Eur Heart J* 2018;39:2540–2545; 2. Fox KM, *et al.* Treatment patterns and low-density lipoprotein cholesterol (LDL-C) goal attainment among patients receiving high- or moderate-intensity statins. *Clin Res Cardiol* 2018;107:380–388; 3. Mach, *et al.* 2019 ESC/EAS Guidelines for the management of dyslipidaemias: *lipid modification to reduce cardiovascular risk. Eur Heart J*. 2020;41:111–188. 4.Kotseva K, *et al.* Lifestyle and impact on cardiovascular risk factor control in coronary patients across 27 countries: Results from the European Society of Cardiology ESC-EORP EUROASPIRE V registry. *Eur J Res Prev Cardio* 2019;26:824–835.

THERAPEUTIC APPROACHES TO REDUCING LDL-C VIA THE LDL RECEPTOR

small molecules, monoclonal antibodies, siRNA



Adapted from Nordestgaard BG et al. Nat Rev Cardiol 2018.[1]

ACL – ATP-citrase lyase; CoA – coenzyme A; HMG – 3-hydroxy-3-methylglutaryl; LDL – low-density lipoprotein; LDL-C – low-density lipoprotein cholesterol; LDLR – low-density lipoprotein receptor; mRNA – messenger ribonucleic acid; NPC1L1 – Niemann Pick C1-like 1 protein; PCSK9 – proprotein convertase subtilisin/kexin type 9; siRNA – small interfering ribonucleic acid

References: 1. Nordestgaard BG et al. Nat Rev Cardiol 2018;15(5):261-272. **2.** Landmesser U et al. Eur Heart J 2018;39(14):1131-1143. **3.** Agarwala A and Goldberg AC. Future Cardiol 2020;16(5):361-371.

Intensity of lipid-lowering treatment

Treatment	Average LDL-C reduction
Moderate intensity statin	30%
High intensity statin	50%
Ezetimibe	20%
High intensity statin + Ezetimibe	65%
Bempedoic acid	20%
Bempedoic acid + Ezetimibe	40%
Inclisiran	50%
PCSK9i	60%
PCSK9i + High intensity statin + Ezetimibe	85%

Summary of National G
Primary and Secondary

INITIAL CONSIDERATIONS:
- Measure non-fasting ***full lipid profile*** (total choleste
- Ensure appropriate baseline and follow up tests as c

PRIMARY PREVENTI

Consider statin therapy for adults who do not have establis
below. Use QRISK risk assessment tool where appropriate
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 or or more of the following:
- Over 40 years
- Had diabetes for >10 years
- Have established nephropathy
- Have other CVD risk factors

Identify and address all modifiable risk factors - smoki
physical activity, blood pressure a

Consider additional risk factors, if present, together wi
severe mental illness, taking medicines that cause dyslipida
(e.g. SLE), impaired fasting glycaemia, recen

PRIMARY PREVENTI

If lifestyle modification is ineffective or inappropr
Atorvastatin 20mg dai

- Measure full lipid profile again after 3 months (non-fasting)
- High intensity statin treatment should achieve reduction of not achieved after 3 months;
  - discuss treatment adherence, timing of dose, diet and l
  - If at higher risk (based on comorbidities, risk score or c *'Additional Risk Factors'*) consider increasing the dose dose of atorvastatin 80mg daily.
  - For how to increase in people with CKD see *'Special P*

- If patients on a high-intensity statin have side effects, offer (see page 2 *'Extent of lipid lowering with available therapi*
- If maximum tolerated dose of statin does not achieve non- value after 3 months consider adding Ezetimibe 10mg dail
- If statin treatment is contraindicated or not tolerated;
  - See AAC Statin Intolerance Algorithm for advice regardi
  - Ezetimibe 10mg monotherapy may be considered. Asse
  - Ezetimibe 10mg/bempedoic acid 180 mg combination m alone does not control non-HDL-C/LDL-C well enough (

If non-HDL-C reduction remains < 40% of baseline desp
therapy (including people with intolerances and contraindic
lipid management clinic according to lo

SECONDARY PREVENTION

Offer statin therapy to adults with CVD, this includes CHD, angina, Acute Coronary Syndrome (MI or unstable angina), 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 daily

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 < 2.5mmol/L (approximately equivalent to LDL-C < 1.8mmol/L) as recommended by Joint British Societies (JBS3).
  **this scenario is not currently covered by NICE CG181. NICE will consider this as part of the guideline update with publication currently expected December 2023*
- If patients on a high-intensity statin have side effects, offer a lower dose or an alternative statin (see page 2 *'Extent of lipid lowering with available therapies'*)

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).

If statin intolerance is confirmed, consider:
- **Ezetimibe 10mg** monotherapy. Assess response after 3 months (TA385)
- **Ezetimibe 10mg/bempedoic acid 180 mg** combination when ezetimibe alone does not control non-HDL-C sufficiently. (NICE TA694)

If non HDL-C remains > 2.5mmol/L despite other lipid lowering therapies consider Injectable therapies - arrange a fasting blood test and assess eligibility criteria (TA393/394, TA733)

Ezetimibe 10mg daily (NICE TA385). Reassess after three months. If non-HDL-C remains > 2.5mmol/L; consider **injectable therapies** arrange a fasting blood test and assess eligibility

* See overleaf for information to support shared decision making

** Inclisiran and PCSK9i should not be prescribed concurrently

Injectable therapies**
If non-HDL-C > 2.5mmol/L; Arrange fasting blood test to measure LDL-C to assess eligibility:
- **Inclisiran** - if fasting LDL-C ≥ 2.6mmol/L despite maximum tolerated lipid lowering therapy (TA733)

OR

- **PCSK9i** - see overleaf for LDL-C thresholds. (TA393/4)

If eligibility criteria not met, consider **ezetimibe 10mg daily** (if not previously considered)

Additional CV risk reduction considerations - check fasting triglycerides levels and consider icosapent ethyl. See triglycerides section overleaf.

ACCELERATED ACCESS COLLABORATIVE

NHS

s of hyperlipidaemia and manage as needed.
g triglyceride above 4.5mmol/L see page 2.

DARY PREVENTION
ludes CHD, angina, Acute Coronary Syndrome (MI or unstable
nptomatic peripheral arterial disease. Do not delay statin
ome. Take a lipid sample on admission (within 24 hours).

risk factors - smoking, diet, obesity, alcohol intake,
ty, blood pressure and HbA1c.

DARY PREVENTION
dary prevention while managing modifiable risk factors.
e a high intensity statin:
vastatin 80mg daily
s a potential drug interaction, high risk of or experiencing
ects, or patient preference.
KD (people with GFR< 60 mL/min/1.73m²).

s (non-fasting).
reduction of non-HDL-C > 40% from baseline. If not achieved

se, diet and lifestyle measures
d the person is judged to be at higher risk (based on
nt - see page 2 *'Additional Risk Factors'*), consider increasing
n people with CKD see *'Special Patient Populations'* (page 2).
*, consider target non-HDL-C < 2.5mmol/L (approximately
mended by Joint British Societies (JBS3).
CE CG181. NICE will consider this as part of the guideline
ecember 2023
e effects, offer a lower dose or an alternative statin
ilable therapies')

ontrol non-HDL-C/LDL-C well enough after 3 months confirm
ptions based on shared decision making* with the patient

Ezetimibe 10mg daily (NICE TA385). Reassess after three months. If non-HDL-C remains > 2.5mmol/L; consider **injectable therapies** arrange a fasting blood test and assess eligibility

* See overleaf for information to support shared decision making

** Inclisiran and PCSK9i should not be prescribed concurrently

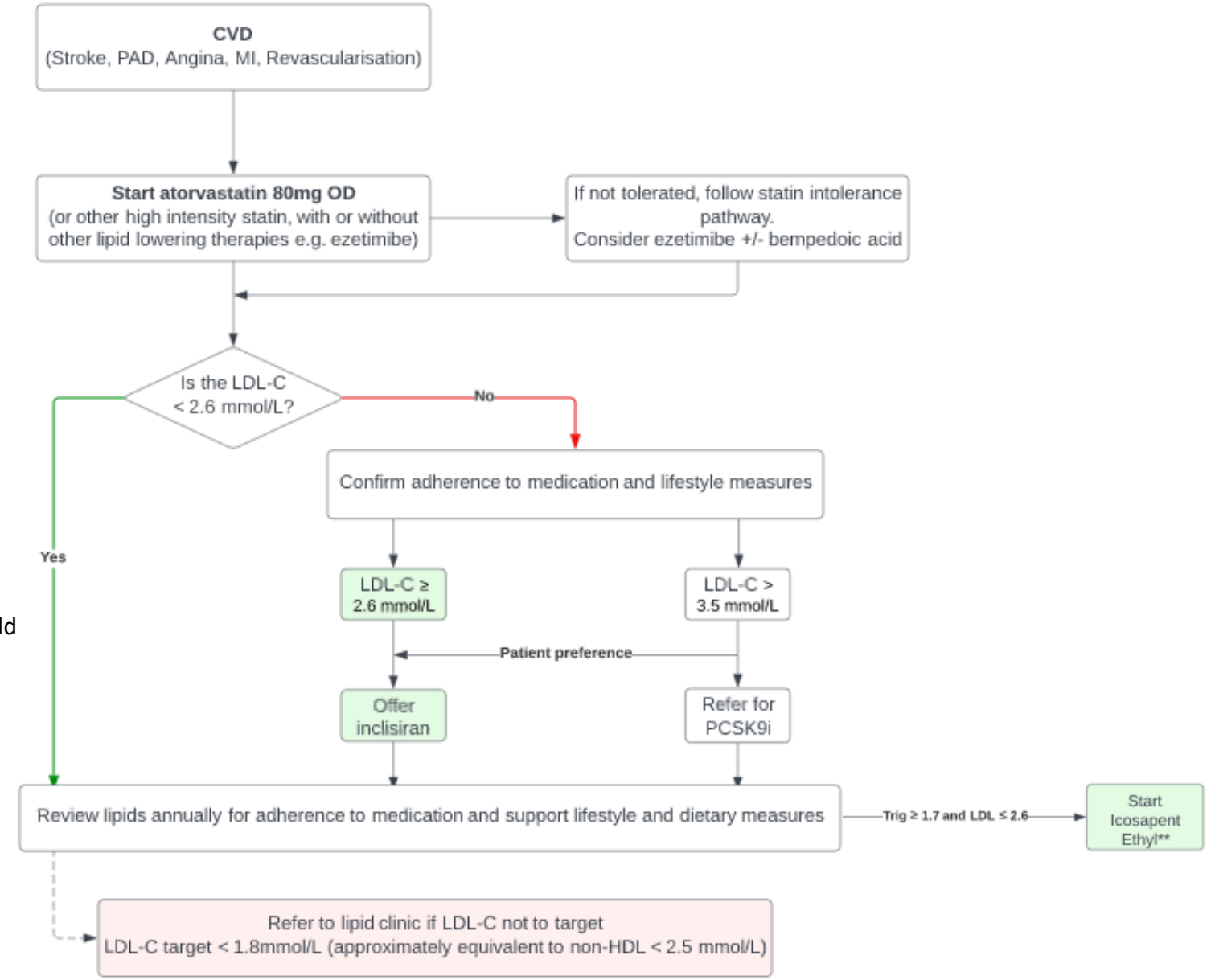
Injectable therapies**
If non-HDL-C > 2.5mmol/L; Arrange fasting blood test to measure LDL-C to assess eligibility:
- **Inclisiran** - if fasting LDL-C ≥ 2.6mmol/L despite maximum tolerated lipid lowering therapy (TA733)

OR

- **PCSK9i** - see overleaf for LDL-C thresholds. (TA393/4)

If eligibility criteria not met, consider **ezetimibe 10mg daily** (if not previously considered)

s - check fasting triglycerides levels and consider icosapent



If >1.8 mmol/L add ezetimibe

A&G to CVD risk and lipid clinic

Case study

64 year old male, presents with inferior STEMI, managed with PCI to RCA

- Nil significant PMH or FHx of CVD
- BP 132/74 mmHg; Wt 83kg; BMI 27kg/m^2; current smoker
- TFTs / LFTs / U&Es / HbA1c – unremarkable

Lipid profile on admission	
TC	6.7 mmol/L
Non-HDL-C	5.5 mmol/L
LDL-C	4.6 mmol/L
TG	1.86 mmol/L
HDL-C	1.2 mmol/L

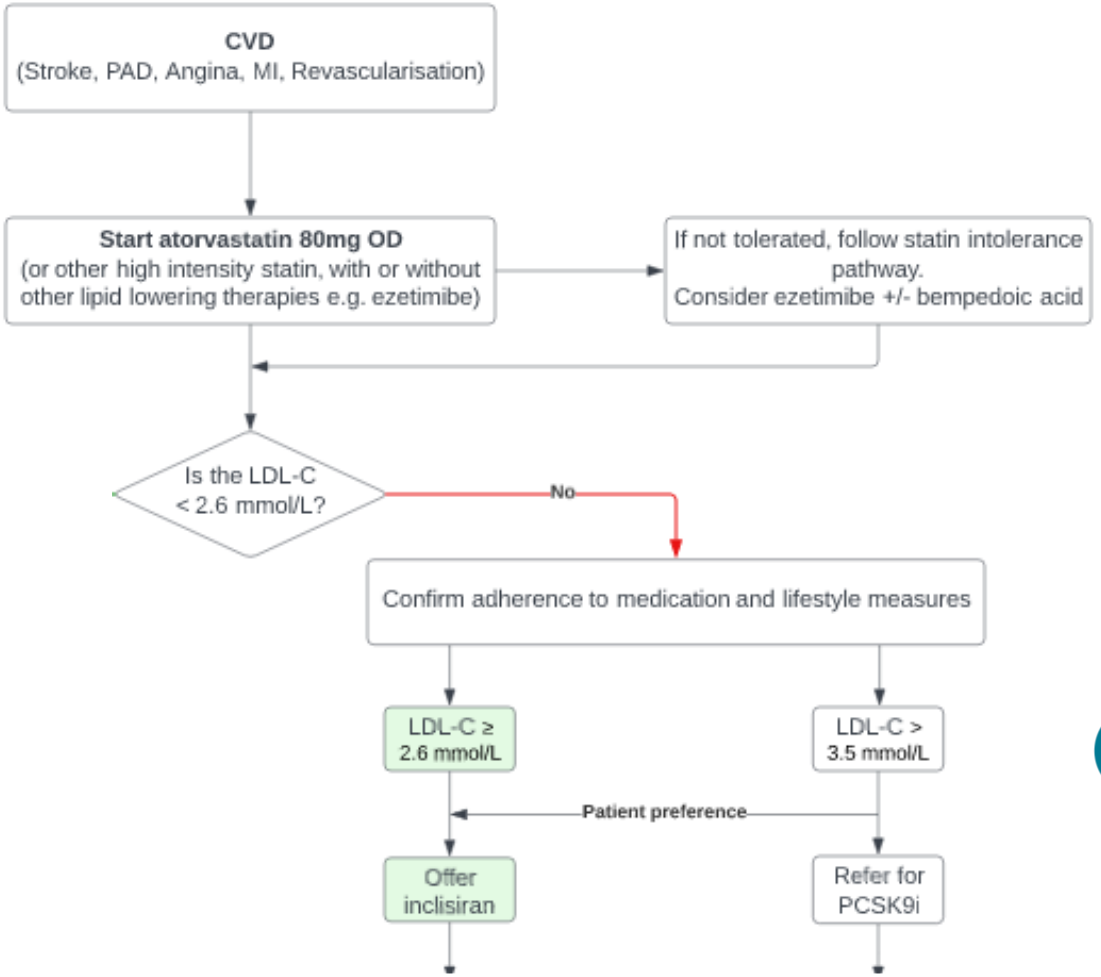
- Post-PCI echo: LV 55-60%; Nil RWMA or valvular disease
- CRUSADE score moderate

Medicines on discharge

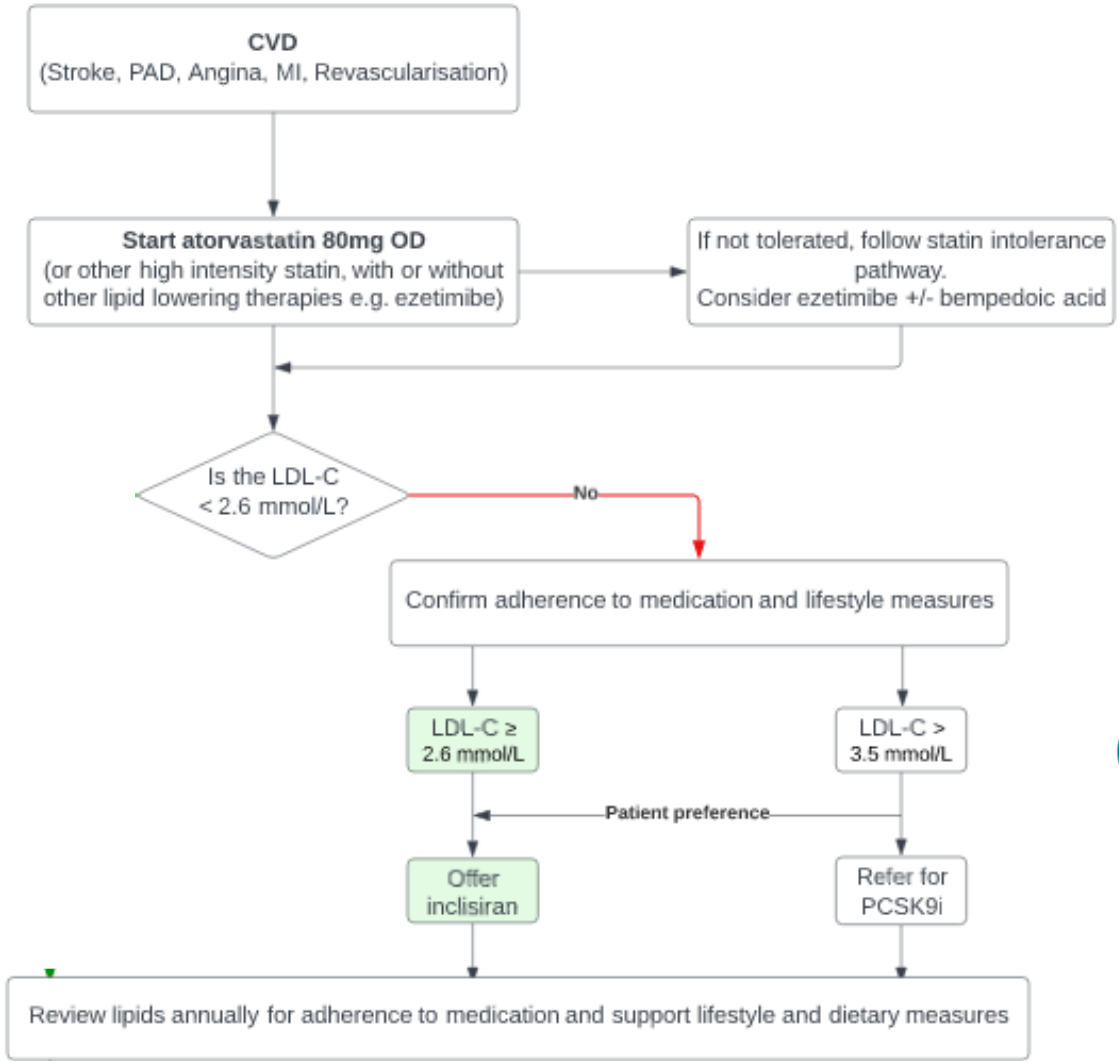
- Aspirin 75mg OD
- Ticagrelor 90mg BD for 12 months
- **Atorvastatin 80mg OD**
- Bisoprolol 2.5mg OD
- Ramipril 2.5mg OD
- Lansoprazole 30mg OD
- GTN 400mcg spray 1-2 prn

HDL: High density lipoproteins LDL: Low-density lipoproteins; TC: Total cholesterol; TG: Triglycerides.

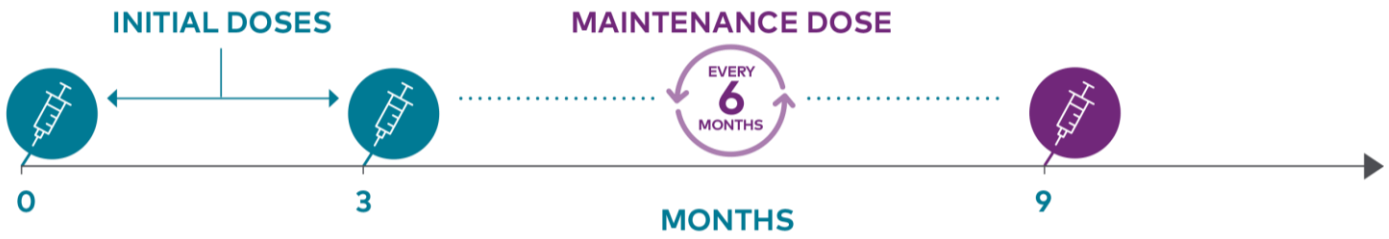
This is not a real patient.



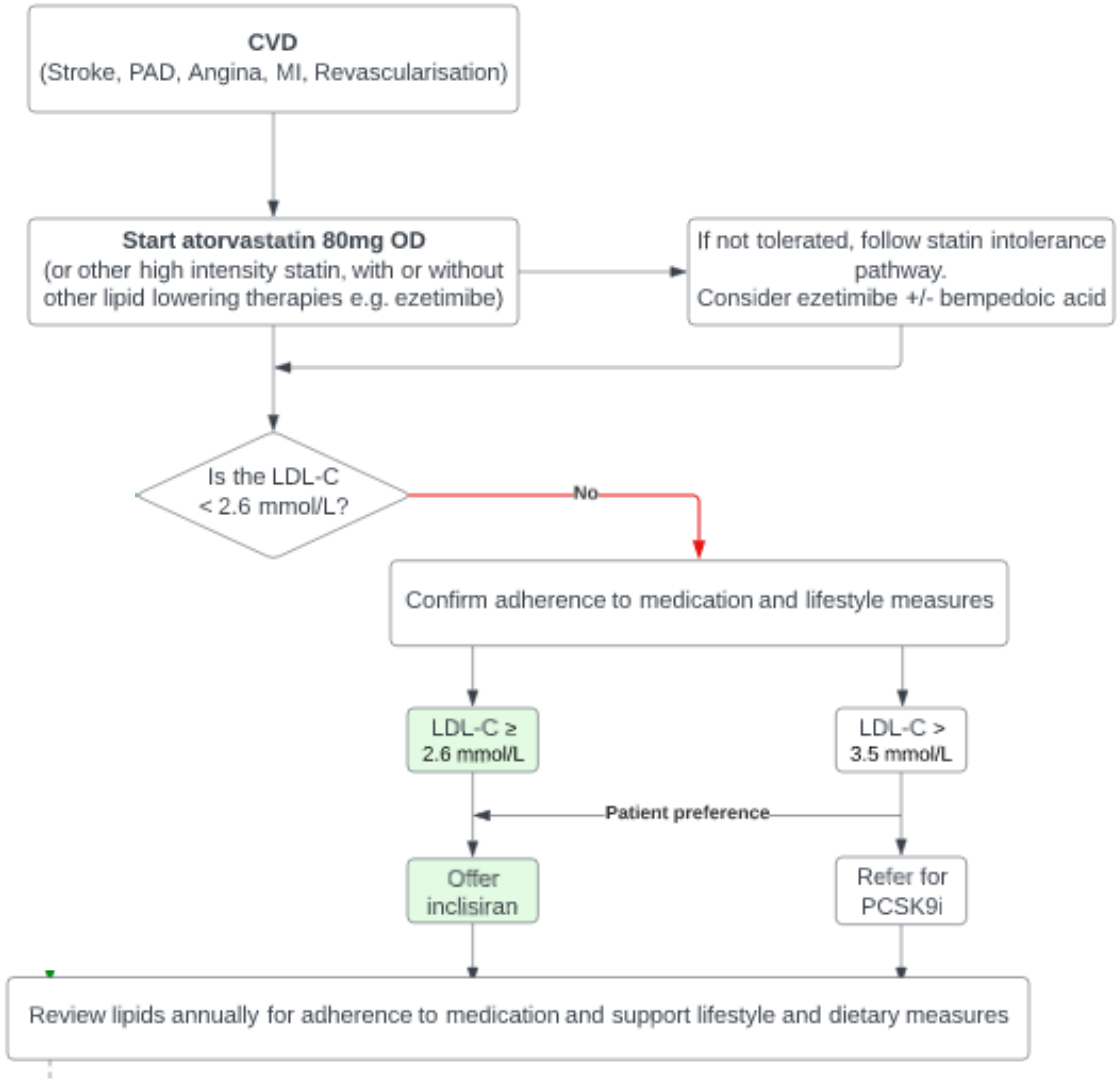
Lipid profile at 3 months	
TC	4.9 mmol/L
Non-HDL-C	3.6 mmol/L
LDL-C	2.7 mmol/L
TG	1.98 mmol/L
HDL-C	1.3 mmol/L



Lipid profile at 3 months	
TC	4.9 mmol/L
Non-HDL-C	3.6 mmol/L
LDL-C	2.7 mmol/L
TG	1.98 mmol/L
HDL-C	1.3 mmol/L



Lipid profile at 12 months	
TC	3.3 mmol/L
Non-HDL-C	2.1 mmol/L
LDL-C	1.3 mmol/L
TG	1.83 mmol/L
HDL-C	1.2 mmol/L



Lipid profile at 3 months	
TC	4.9 mmol/L
Non-HDL-C	3.6 mmol/L
LDL-C	2.7 mmol/L
TG	1.98 mmol/L
HDL-C	1.3 mmol/L

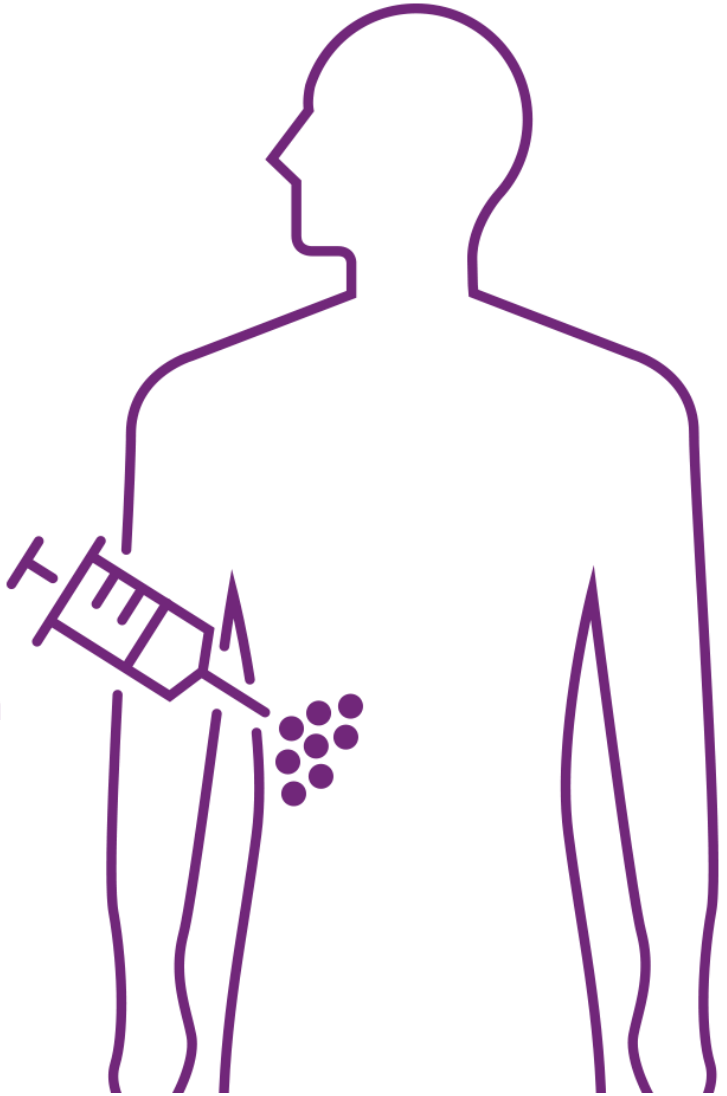
Lipid profile at 12 months	
TC	4.2 mmol/L
Non-HDL-C	3.0 mmol/L
LDL-C	2.2 mmol/L
TG	1.75 mmol/L
HDL-C	1.2 mmol/L

Inclisiran – approved for NEL

- **2 Maintenance doses a year**
- HCP-administered
- Subcutaneous injection
- No refrigeration required
- Fixed dose, no adjustments required
- Prescribed on FP10 or purchase direct from AAH (FP34D form)

Preferred site:
Abdomen

Alternative sites:
Upper arm or thigh



Areas to avoid:
Active skin disease or injury (e.g., sunburns, skin rashes, inflammation, skin infections)

MANAGING MISSED DOSES

PLANNED DOSE MISSED BY **<3 MONTHS**	Administer inclisiran and continue dosing as per patient's original schedule[1]
PLANNED DOSE MISSED BY **>3 MONTHS**	Start new dosing schedule: initial dose, second dose at 3 months, followed by a dose every 6 months[1]

Thank you

CVD Risk and Lipids Clinic @ Barts

Advice and Refer via ERS

“To prevent a heart attack, take one statin every day. Take it out for a run, then take it to the gym, then take it for a bike ride…”