Heart Foundation

FOR HEALTH PROFESSIONALS

Practical guide to pharmacological lipid management

Managing LDL-C in patients with high CVD risk+

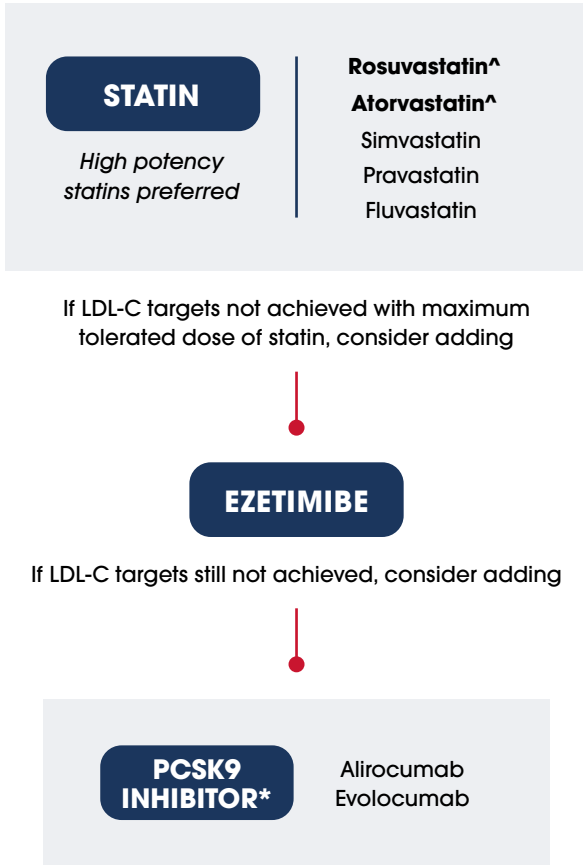

Figure 1. Practical guide to pharmacological lipid management – flowchart[1]

+Defined as patients with established CVD, high absolute CVD risk score >15% or who are at clinically determined high risk

^High potency statins

*View Product Information and visit the PBS website for more details on PCSK9 inhibitor clinical indications and PBS subsidies

Practice considerations

- Strongly recommend healthy lifestyle changes (diet, physical activity, smoking cessation and weight management) to all patients, regardless of medicine initiation.[2,3] View the Heart Foundation's nutrition position statements.
- Encourage adherence to medicines by explaining the benefits on overall CVD risk. Explain serious side effects are rare.[2]
- Initiate the highest tolerated dose of statin therapy for patients following hospitalisation for acute coronary syndrome.[4] Allow at least four weeks between statin dose increases to optimise effects from current dose.[5]
- For patients unable to tolerate a prescribed statin, consider a lower dose or switching to an alternative statin. Statin intolerance is often overestimated (true prevalence 8–10%).[6]
- If LDL-C targets are still not met with a combination of statin, ezetimibe and PCSK9 inhibitor, bile acid binding resins may be added. Side effects often limit their use.[7]
- Bile acid binding resins, fibrates and nicotinic acid have been shown to improve lipid levels but evidence to support their addition to statin therapy to improve cardiovascular outcomes is limited.[1]
- Note: Pharmacological management of familial hypercholesterolaemia (FH) may differ from this algorithm, see 2020 FH Guidelines.[8]
- If triglycerides are persistently elevated with maximum tolerated statin and ezetimibe, recommend healthy lifestyle changes and consider adding a fibrate and/or a high dose omega 3 fatty acid.[9]

CVD: cardiovascular disease; LDL-C: low-density lipoprotein-cholesterol; PCSK9: proprotein convertase subtilisin/kexin type 9

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Comparison of LDL-C lowering potential of lipid-lowering medicines

Drug classes

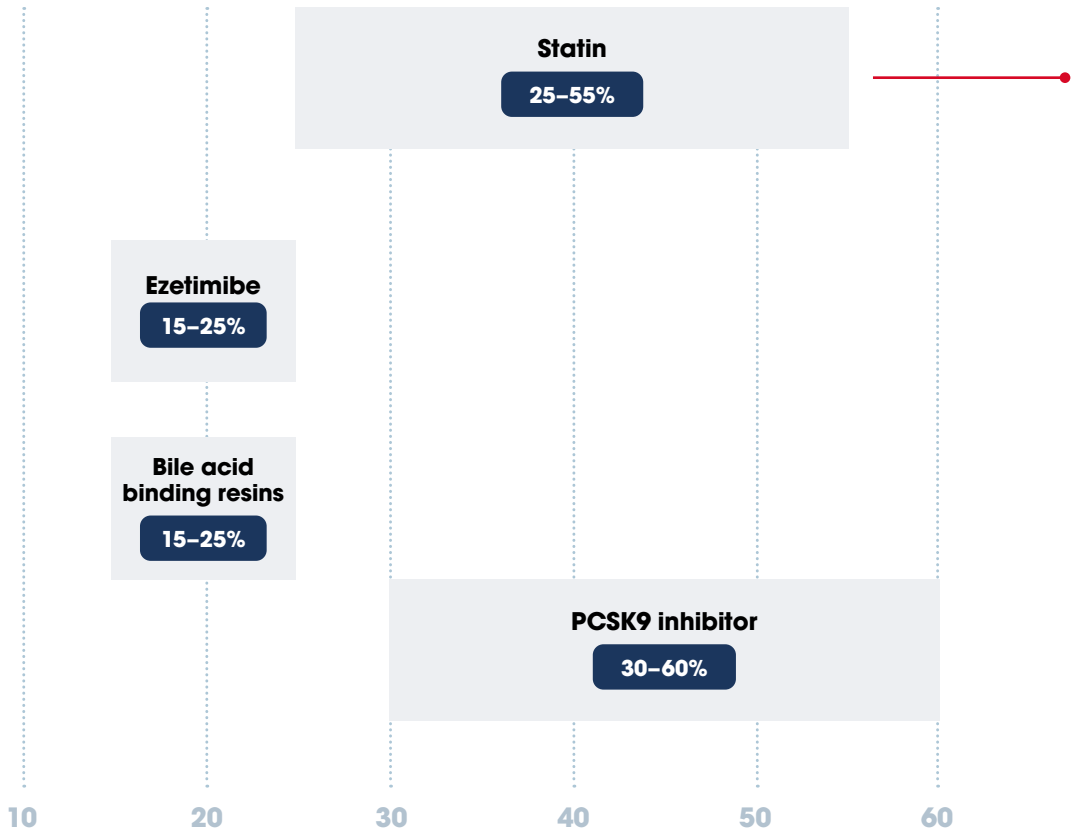

Figure 2. Potential reduction in LDL-C of different drug classes (%)[5]

Statin LDL-C lowering intensity[5,10]

STATIN	Low intensity (<30% reduction in LDL-C)	Moderate intensity (30–49% reduction in LDL-C)	High intensity (>50% reduction in LDL-C)
Atorvastatin	N/A	10–20mg	40–80mg
Fluvastatin	20–40mg	80 mg	N/A
Pravastatin	10–20mg	40–80mg	N/A
Rosuvastatin	N/A	5–10mg	20–40mg
Simvastatin	5–10mg	20–80mg	N/A

Practice considerations

- Combination therapy starting with a first line agent (statin) plus ezetimibe and a PCSK9 inhibitor may help to lower LDL-C by more than 80%.[11]
- Australian guidelines currently recommend an LDL-C target of <2 mmol/L for primary prevention and <1.8 mmol/L for secondary prevention.[3,4] More recently, some international guidelines recommend a lower LDL-C target (<1.4 mmol/L) in the secondary prevention setting.[7]

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