

Statins

Competitive inhibitors of HMG-CoA reductase that upregulate hepatic LDL receptors, first-line for lowering LDL cholesterol, and dominated in practice by muscle symptoms and interaction risk.

Online guide: <https://www.medguideglobal.com/pharmacology/cardiovascular/statins>

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Quick revision

One Liner: Statins competitively inhibit HMG-CoA reductase, so the hepatocyte makes less cholesterol, upregulates its LDL receptors and pulls more LDL out of the blood - and almost every safety issue in the class comes from muscle or from interactions that raise statin exposure.

1. Mechanism: selective competitive inhibition of HMG-CoA reductase, which converts HMG-CoA to mevalonate in the cholesterol synthesis pathway. [2]
2. Reduced hepatic cholesterol synthesis upregulates LDL receptors and increases hepatic uptake of LDL cholesterol. [2]
3. Seven agents are FDA-approved: atorvastatin, rosuvastatin, simvastatin, pravastatin, fluvastatin, lovastatin and pitavastatin. [2]
4. Therapy is graded by intensity: high-intensity lowers LDL cholesterol by more than half, moderate-intensity by roughly a third to a half, and low-intensity by less than a third. [2]
5. Atorvastatin, simvastatin, fluvastatin, pitavastatin and lovastatin are lipophilic, while rosuvastatin and pravastatin are hydrophilic. [2]
6. Myopathy with diffuse myalgia or muscle tenderness is the characteristic adverse effect; rhabdomyolysis is the most serious but is rare. [2]
7. Myopathy risk rises sharply with CYP3A4 inhibitors for atorvastatin, lovastatin and simvastatin, and with gemfibrozil. [2]
8. Transaminase elevations are usually transient and may resolve even if therapy continues, and serious hepatotoxicity is rare. [2][4]
9. New-onset diabetes mellitus and, rarely, immune-mediated necrotising myopathy are recognised class adverse effects. [2]

Overview

1. Statins are selective, competitive inhibitors of HMG-CoA reductase, the enzyme that converts HMG-CoA to mevalonate in the cholesterol synthesis pathway. Reducing hepatic cholesterol synthesis triggers upregulation of LDL receptors and increased hepatic uptake of LDL cholesterol, and it is that receptor effect rather than the synthesis block itself that accounts for the fall in circulating LDL. [2]
2. Seven agents are approved - atorvastatin, rosuvastatin, simvastatin, pravastatin, fluvastatin, lovastatin and pitavastatin - and they are grouped in practice by the intensity of LDL lowering achieved rather than by chemistry. Approved uses span hyperlipidaemia and mixed dyslipidaemia, primary dysbetalipoproteinaemia, hypertriglyceridaemia, atherosclerosis, familial hypercholesterolaemia, and both primary and secondary prevention of atherosclerotic cardiovascular disease. Statins are the first-line lipid-lowering drugs for hypercholesterolaemia. [2][3]
3. Beyond lipids, the class shows anti-inflammatory, antioxidant, antiproliferative and immunomodulatory properties and promotes plaque stability. The practical limits on use are muscular and pharmacokinetic: myalgia and myopathy are the characteristic complaints, rhabdomyolysis is the rare severe end of that spectrum, and the interactions that raise statin exposure are what convert an ordinary prescription into a hazardous one. [2]

Classification and drug examples

Intro: Two groupings are used in parallel. Intensity of LDL lowering guides therapeutic choice, while lipophilicity and metabolic route determine tissue distribution and interaction risk. Both are needed, because the agent that gives the required LDL reduction may also be the one a patient's other medicines make unsafe.

1. Name: CYP3A4-metabolised statins

1. Note: The members whose exposure rises when a CYP3A4 inhibitor is co-prescribed, which is where most clinically important statin interactions arise.

1. Name: Atorvastatin

1. Route: Oral

1. Note: Lipophilic, with a long half-life allowing administration at any time of day; capable of high-intensity LDL lowering. [2]

2. Name: Simvastatin

2. Route: Oral

2. Note: Lipophilic with low oral bioavailability and a short half-life; the member most often named in interaction warnings. [2]

3. Name: Lovastatin

3. Route: Oral

3. Note: Lipophilic and short-acting; combination with ciclosporin, tacrolimus, everolimus or sirolimus is contraindicated. [2] [2]

2. Name: Statins handled outside CYP3A4

2. Note: Members metabolised by other routes, which changes but does not abolish their interaction profile; transporters such as OATP1B1, OATP1B3 and P-glycoprotein remain relevant across the class.

1. Name: Rosuvastatin

1. Route: Oral

1. Note: Hydrophilic, capable of high-intensity LDL lowering, with a long half-life and only limited CYP2C9 involvement. [2]

2. Name: Pravastatin

2. Route: Oral

2. Note: Hydrophilic and the least protein-bound member of the class, at around half; still carries myopathy risk with gemfibrozil. [2]

3. Name: Fluvastatin

3. Route: Oral

3. Note: Lipophilic and metabolised by CYP2C9 rather than CYP3A4; short-acting. [2]

4. Name: Pitavastatin

4. Route: Oral

4. Note: Lipophilic with the highest oral bioavailability of the class; contraindicated with ciclosporin-type immunosuppressants. [2] [2]

Mechanism of action

Summary: Statins competitively inhibit HMG-CoA reductase, reducing hepatic cholesterol synthesis; the hepatocyte responds by upregulating LDL receptors, which increases clearance of LDL cholesterol from the circulation.

Target: HMG-CoA reductase, the enzyme converting HMG-CoA to mevalonate

Cidal Static: not applicable

Kill Kinetics: not applicable

1. Step: 1

1. Title: The drug reaches the hepatocyte

1. Detail: Lipophilic members enter tissues by passive diffusion, while hydrophilic members depend on transporter proteins to gain entry - a difference that also shapes their interaction profiles. [2]
2. Step: 2
2. Title: HMG-CoA reductase is competitively inhibited
2. Detail: The drug competes with the natural substrate at the enzyme that converts HMG-CoA to mevalonate, the committed step of cholesterol biosynthesis. [2]
3. Step: 3
3. Title: Hepatic cholesterol synthesis falls
3. Detail: With mevalonate production reduced, the hepatocyte's own cholesterol content declines. [2]
4. Step: 4
4. Title: LDL receptors are upregulated
4. Detail: The cell compensates by increasing LDL receptor expression on its surface, which increases hepatic uptake of LDL cholesterol from plasma. [2]
5. Step: 5
5. Title: Additional vascular effects follow
5. Detail: Beyond the lipid change, the class shows anti-inflammatory, antioxidant, antiproliferative and immunomodulatory effects and promotes plaque stability. [2]

Major clinical uses

1. Drug: Atorvastatin
1. Organism: Not applicable
1. Indication: Secondary prevention of atherosclerotic cardiovascular disease
1. Line: first-line
1. Note: High-intensity therapy lowers LDL cholesterol by more than half; the choice of agent and intensity rests with the treating clinician. [2]
2. Drug: Statins as a class
2. Organism: Not applicable
2. Indication: Primary hyperlipidaemia and mixed dyslipidaemia
2. Line: first-line
2. Note: Statins are described as the first-line lipid-lowering drugs for hypercholesterolaemia. [2][3]
3. Drug: Rosuvastatin
3. Organism: Not applicable
3. Indication: Familial hypercholesterolaemia, including paediatric and homozygous forms within the class's approvals
3. Line: first-line
3. Note: One of the two agents able to deliver high-intensity LDL reduction. [2]
4. Drug: Statins as a class
4. Organism: Not applicable
4. Indication: Primary dysbetalipoproteinaemia, hypertriglyceridaemia and atherosclerosis
4. Line: targeted
4. Note: All are within the approved indications for the class, though fibrates are the agents primarily used to lower triglycerides. [2][3]
5. Drug: Pravastatin
5. Organism: Not applicable
5. Indication: Lipid lowering where a non-CYP3A4 route is preferred
5. Line: targeted
5. Note: Being outside the CYP3A4 pathway removes one common interaction, but gemfibrozil still raises myopathy risk with this agent. [2]

- 6. Drug: Statins as a class
- 6. Organism: Not applicable
- 6. Indication: Off-label use in non-alcoholic fatty liver disease, cardiac allograft vasculopathy and prevention of contrast-induced acute kidney injury
- 6. Line: adjunct
- 6. Note: Recorded off-label uses; each remains a specialist decision rather than routine practice. [2]

Pharmacokinetics

- 1. Drug: Atorvastatin
 - 1. Route: Oral
 - 1. Absorption: Bioavailability around 12% because of extensive first-pass metabolism
 - 1. Csf Penetration: Not clinically relevant
 - 1. Metabolism: CYP3A4
 - 1. Elimination: See prescribing reference
 - 1. Half Life: Long, permitting administration at any time of day
 - 1. Dose Adjust: Review concurrent CYP3A4 inhibitors before use [2]
- 2. Drug: Simvastatin
 - 2. Route: Oral
 - 2. Absorption: Bioavailability under 5%
 - 2. Csf Penetration: Not clinically relevant
 - 2. Metabolism: CYP3A4
 - 2. Elimination: See prescribing reference
 - 2. Half Life: Short, which is why evening administration or a modified-release form is used
 - 2. Dose Adjust: Avoid with gemfibrozil; exposure rises several-fold with diltiazem or verapamil [2]
- 3. Drug: Rosuvastatin
 - 3. Route: Oral
 - 3. Absorption: Bioavailability around 20%
 - 3. Csf Penetration: Not clinically relevant
 - 3. Metabolism: CYP2C9 to a lesser degree; largely handled by transporters
 - 3. Elimination: See prescribing reference
 - 3. Half Life: Long, permitting administration at any time of day
 - 3. Dose Adjust: See prescribing reference [2]
- 4. Drug: Pravastatin
 - 4. Route: Oral
 - 4. Absorption: Hydrophilic; tissue entry depends on transporter proteins
 - 4. Csf Penetration: Not clinically relevant
 - 4. Metabolism: Outside the CYP3A4 pathway
 - 4. Elimination: See prescribing reference
 - 4. Half Life: Short
 - 4. Dose Adjust: Myopathy risk still rises with gemfibrozil [2]
- 5. Drug: Pitavastatin
 - 5. Route: Oral
 - 5. Absorption: Bioavailability above 60%, the highest in the class
 - 5. Csf Penetration: Not clinically relevant
 - 5. Metabolism: See prescribing reference
 - 5. Elimination: See prescribing reference
 - 5. Half Life: See prescribing reference

5. Dose Adjust: Contraindicated with ciclosporin, tacrolimus, everolimus or sirolimus [2]

1. Oral bioavailability varies enormously across the class, from under 5% to over 60%, largely because of differences in first-pass metabolism. Plasma protein binding is high for every member except pravastatin, which is around half bound. [2]

2. Transporters matter as much as enzymes: OATP1B1, OATP1B3 and P-glycoprotein are all involved in statin handling, which is why interactions are not confined to CYP inhibitors. [2]

3. Monitoring centres on the lipid panel at baseline and again a few weeks after starting or changing therapy, then at intervals thereafter; creatine kinase is checked when colchicine is co-prescribed, and renal function where impairment exists. [2]

4. This page gives no dose regimens by design. Intensity, agent choice and dose depend on cardiovascular risk, the LDL target, renal and hepatic function and concurrent therapy, and belong in a prescribing reference used by the treating clinician.

Adverse effects

1. Effect: Myopathy and muscle symptoms

1. Detail: Diffuse myalgia or muscle tenderness is the characteristic complaint and the commonest reason therapy is interrupted. [2]

2. Effect: Transaminase elevation

2. Detail: Mild to moderate elevations occur during therapy, are typically transient and asymptomatic, and may resolve with continued treatment without any dose change. [2][4]

3. Effect: New-onset diabetes mellitus

3. Detail: A recognised adverse effect of statin therapy, weighed against the cardiovascular benefit rather than treated as an automatic reason to stop. [2]

1. Effect: Rhabdomyolysis

1. Detail: The most serious complication of statin use, though its occurrence is rare; risk rises with interactions that increase statin exposure.

1. Action: Muscle symptoms with dark urine or marked weakness prompt urgent assessment and review of the whole medication list. [2]

2. Effect: Statin-associated immune-mediated necrotising myopathy

2. Detail: A distinct and rare autoimmune muscle disease that, unlike simple myalgia, does not settle promptly when the drug is withdrawn.

2. Action: Persistent or progressive weakness after stopping the drug warrants specialist evaluation. [2]

3. Effect: Clinically apparent liver injury

3. Detail: Rare and unpredictable, at a rate of about one case per million person-years of use, although statins account for roughly 5% of cases of clinically apparent drug-induced liver injury overall because so many people take them.

3. Action: Baseline liver tests before starting, with retesting only if symptoms develop. [4]

1. Drug: Simvastatin and lovastatin

1. Effect: Exposure increases three- to eightfold with diltiazem or verapamil, which is the largest of the commonly encountered statin interactions. [2]

2. Drug: Lovastatin and pitavastatin

2. Effect: Ciclosporin is a labelled contraindication for pitavastatin and a combination to avoid with lovastatin; tacrolimus, everolimus and sirolimus are combinations to avoid with either agent rather than labelled contraindications. [2][1]

3. Drug: Statins as a class

3. Effect: Confirmed ALT elevation beyond three times the upper limit of normal occurs in about 0.1% of users compared with 0.04% on placebo. [4]

Contraindications, precautions and interactions

1. Active hepatic disease, or unexplained persistent elevation of aminotransferases. [2]
2. Breastfeeding, and pregnancy - the latter with an exception recognised in 2021 regulatory guidance for patients at high cardiovascular risk. [2]
3. Concomitant ciclosporin in a patient taking pitavastatin. Ciclosporin with lovastatin, and tacrolimus, everolimus or sirolimus with either agent, are combinations to avoid rather than labelled contraindications - each raises statin exposure and the risk of myopathy and rhabdomyolysis. Lovastatin's own labelled contraindication in this area is concomitant use of a strong CYP3A4 inhibitor. [2][1]
1. Existing muscle symptoms or a previous episode of statin-associated myopathy, which change both the choice of agent and the threshold for investigation. [2]
2. Renal impairment, where creatinine and creatinine clearance are monitored during therapy. [2]
3. Chronic liver disease, where the class is nonetheless considered usable - it is described as safe in non-alcoholic fatty liver disease and probably safe in other chronic liver disease and after transplantation. [4]
1. With: CYP3A4 inhibitors - macrolides, azole antifungals, protease inhibitors, amiodarone, diltiazem, verapamil
1. Effect: Raise the exposure of atorvastatin, lovastatin and simvastatin and therefore the risk of myopathy. [2]
2. With: Gemfibrozil
2. Effect: Reduces simvastatin metabolism, raising its concentration and myopathy risk; the combination is one to avoid, and a similar risk applies with pravastatin. [2]
3. With: Colchicine
3. Effect: Co-prescription is a reason to check creatine kinase during therapy. [2]
4. With: Digoxin
4. Effect: Monitoring for digoxin toxicity is advised alongside high-intensity atorvastatin. [2]

Comparison tables

Title: Statin properties that change prescribing

Caption: Solubility, metabolic route and duration of action are what separate the members of the class. Agent and intensity selection belong to the treating clinician working from a current prescribing reference.

1. Key: drug
1. Label: Drug
1. Is Row Header: true
2. Key: solubility
2. Label: Solubility
3. Key: metabolism
3. Label: Main metabolic route
4. Key: duration
4. Label: Duration
5. Key: point
5. Label: Key point
1. Drug: Atorvastatin
1. Solubility: Lipophilic
1. Metabolism: CYP3A4
1. Duration: Long half-life
1. Point: High-intensity option; CYP3A4 interactions apply [2]
2. Drug: Rosuvastatin
2. Solubility: Hydrophilic
2. Metabolism: CYP2C9 to a lesser degree

- 2. Duration: Long half-life
- 2. Point: The other high-intensity option, outside CYP3A4 [2]
- 3. Drug: Simvastatin
- 3. Solubility: Lipophilic
- 3. Metabolism: CYP3A4
- 3. Duration: Short half-life
- 3. Point: Avoid with gemfibrozil; large interaction with diltiazem and verapamil [2]
- 4. Drug: Pravastatin
- 4. Solubility: Hydrophilic
- 4. Metabolism: Non-CYP3A4
- 4. Duration: Short half-life
- 4. Point: Least protein-bound member; gemfibrozil still raises myopathy risk [2]
- 5. Drug: Pitavastatin
- 5. Solubility: Lipophilic
- 5. Metabolism: See prescribing reference
- 5. Duration: See prescribing reference
- 5. Point: Highest oral bioavailability; contraindicated with ciclosporin-type agents [2]

High-yield exam pearls

- 1. Pearl: The LDL fall is a receptor effect, not a synthesis effect.
- 1. Why: Blocking synthesis lowers the hepatocyte's own cholesterol, which upregulates LDL receptors on its surface; it is the extra receptors clearing LDL from plasma that produce the measured fall. [2]
- 2. Pearl: Which statin, matters for which interaction.
- 2. Why: Atorvastatin, lovastatin and simvastatin are CYP3A4 substrates, whereas fluvastatin is handled by CYP2C9 with rosuvastatin involved to a lesser degree, so a CYP3A4 inhibitor is a problem for some members of the class and not others. [2]
- 3. Pearl: Gemfibrozil with simvastatin is a named combination to avoid.
- 3. Why: Reduced metabolism of simvastatin raises its concentration and the risk of myopathy, and the combination is described as one to avoid; gemfibrozil with pravastatin poses a similar risk. [2]
- 4. Pearl: Calcium channel blockers are an underrated statin interaction.
- 4. Why: Diltiazem and verapamil raise the exposure of simvastatin and lovastatin by three- to eightfold, which is a far larger effect than most people expect from a cardiology co-prescription. [2]
- 5. Pearl: Routine liver monitoring is not part of statin care.
- 5. Why: Guidance is to test liver enzymes before starting and to retest only if symptoms develop, because serious hepatotoxicity is rare - of the order of one case per million person-years of use. [4]
- 6. Pearl: Chronic liver disease is not automatically a barrier.
- 6. Why: Statins are described as usable safely in non-alcoholic fatty liver disease and as probably safe in other forms of chronic liver disease and after liver transplantation, which is the opposite of the common assumption. [4]

Common exam traps

- 1. Trap: "Statins work by blocking cholesterol absorption from the gut."
- 1. Correction: That is ezetimibe. Statins competitively inhibit HMG-CoA reductase inside the hepatocyte and lower LDL cholesterol chiefly by upregulating hepatic LDL receptors. [2][3]
- 2. Trap: "Any rise in transaminases means the statin must be stopped."
- 2. Correction: Elevations are usually transient and asymptomatic and may resolve while therapy continues without a change in dose. Confirmed elevations beyond three times the upper limit of normal are uncommon. [2][4]
- 3. Trap: "All statins carry the same interaction profile."

3. Correction: They do not. The CYP3A4 substrates are the members endangered by macrolides, azole antifungals, protease inhibitors and rate-limiting calcium channel blockers, and ciclosporin-type immunosuppressants are contraindicated specifically with lovastatin and pitavastatin. [2]
4. Trap: "Rhabdomyolysis is the usual muscle problem with statins."
4. Correction: Diffuse myalgia and muscle tenderness are the usual presentation. Rhabdomyolysis is the most serious complication but is described as rare, and immune-mediated necrotising myopathy is rarer still. [2]

Self-test questions

1. Question: What is the immediate biochemical action of a statin?
 1. Competitive inhibition of HMG-CoA reductase, which converts HMG-CoA to mevalonate
 2. Inhibition of the NPC1L1 sterol transporter in the intestinal brush border
 3. Activation of peroxisome proliferator-activated receptor alpha
 4. Binding of bile acids in the intestinal lumen
1. Explanation: Statins are selective, competitive inhibitors of HMG-CoA reductase, the rate-limiting enzyme of the cholesterol synthesis pathway. The other three options describe ezetimibe, the fibrates and the bile acid sequestrants respectively. [2][3]
2. Question: Why does inhibiting hepatic cholesterol synthesis lower circulating LDL cholesterol?
 1. Because the drug binds LDL particles directly in plasma
 2. Because lower hepatocyte cholesterol upregulates LDL receptors, increasing hepatic uptake of LDL
 3. Because cholesterol is diverted into bile acid synthesis and excreted
 4. Because the drug blocks intestinal absorption of dietary cholesterol
2. Explanation: The fall in intracellular cholesterol triggers upregulation of LDL receptors on the hepatocyte surface, and it is the increased receptor-mediated uptake that clears LDL from the circulation. [2]
3. Question: A patient on simvastatin is started on clarithromycin. Why is this a concern?
 1. Clarithromycin displaces simvastatin from albumin
 2. Clarithromycin inhibits CYP3A4, and simvastatin is a CYP3A4 substrate, so exposure and myopathy risk rise
 3. Clarithromycin independently causes rhabdomyolysis in all patients
 4. Clarithromycin blocks LDL receptor recycling
3. Explanation: Simvastatin, together with atorvastatin and lovastatin, is metabolised by CYP3A4. Inhibitors of that enzyme - including macrolides, azole antifungals, protease inhibitors, amiodarone, diltiazem and verapamil - increase statin exposure and therefore myopathy risk. [2]
4. Question: Which lipid-lowering drug is specifically named as one to avoid combining with simvastatin?
 1. Ezetimibe
 2. Evolocumab
 3. Gemfibrozil
 4. Colesevelam
4. Explanation: Gemfibrozil reduces the metabolism of simvastatin, raising its concentration and the risk of myopathy, and the pairing is described as a combination to avoid. Gemfibrozil with pravastatin carries a similar myopathy risk. [2]
5. Question: Which statement about statins and the liver is correct?
 1. Routine periodic liver testing is required for all patients on a statin
 2. Serious hepatotoxicity is common and dose-related
 3. Liver tests are checked before starting, with retesting only if symptoms arise, because serious injury is rare
 4. Statins are contraindicated in all forms of chronic liver disease
5. Explanation: Multiple academic societies and the regulator recommend baseline testing with retesting only if symptoms appear. Serious hepatotoxicity is rare and unpredictable, and statins are considered usable in non-alcoholic fatty liver disease. [4]
6. Question: Which two statins are hydrophilic rather than lipophilic?

1. Atorvastatin and simvastatin
2. Rosuvastatin and pravastatin
3. Lovastatin and fluvastatin
4. Pitavastatin and simvastatin
6. Explanation: Rosuvastatin and pravastatin are the hydrophilic members; atorvastatin, simvastatin, fluvastatin, pitavastatin and lovastatin are lipophilic. Lipophilic agents penetrate tissues by passive diffusion, whereas hydrophilic ones depend on transporter proteins. [2]
7. Question: Which of the following is a recorded contraindication to statin therapy?
 1. Active hepatic disease or unexplained persistent aminotransferase elevation
 2. Hypertension
 3. Chronic kidney disease at any stage
 4. A family history of familial hypercholesterolaemia
7. Explanation: Active hepatic disease and unexplained persistent aminotransferase elevations are contraindications, alongside breastfeeding and pregnancy, the latter with an exception recognised in 2021 regulatory guidance for patients at high cardiovascular risk. Familial hypercholesterolaemia is an indication, not a contraindication. [2]

Frequently asked questions

1. Question: Do statins have to be taken at night?
 1. Answer: It depends on the agent. Fluvastatin, lovastatin, pravastatin and simvastatin have shorter half-lives and are given in the evening or as extended-release preparations, whereas atorvastatin and rosuvastatin have longer half-lives and can be taken at any time. [2]
2. Question: How common is serious muscle damage on a statin?
 2. Answer: Diffuse myalgia and muscle tenderness are the usual muscle complaints. Rhabdomyolysis is the most serious complication but is described as rare, and the risk rises when another drug increases statin exposure. [2]
3. Question: Why does the choice of statin matter when other medicines are involved?
 3. Answer: Because the metabolic routes differ. Atorvastatin, lovastatin and simvastatin depend on CYP3A4, so inhibitors of that enzyme raise their concentrations, while fluvastatin runs through CYP2C9 and rosuvastatin uses it only to a lesser degree. [2]
4. Question: Are liver tests needed regularly during statin treatment?
 4. Answer: No. Guidance is to obtain liver tests before starting and to repeat them only if symptoms arise, since clinically serious hepatotoxicity is rare and unpredictable. [4]
5. Question: What do statins do besides lowering LDL cholesterol?
 5. Answer: They show anti-inflammatory, antioxidant, antiproliferative and immunomodulatory effects and promote plaque stability. These pleiotropic properties are part of why the cardiovascular benefit is not entirely explained by the lipid change alone. [2]

References

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