
Major updates in Hyperlipidemia: 2026

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Learning Objectives

1. Identify at least two new changes to hyperlipidemia management in 2026
2. Differentiate patient goals and targets using new guideline recommendations
3. Develop at least two new ways to support patient care using available literature

Which cardiovascular disease studies have you heard of?

- A. Bogalusa Heart Study
- B. Copenhagen City Heart Study
- C. Dallas Heart Study
- D. Framingham Heart Study
- C. Jackson Heart Study

Which cardiovascular disease studies have you heard of?

A. Bogalusa Heart Study:

- Began in 1973 to monitor factors leading to heart disease in pediatric patients from birth to mid-life in a biracial community in Louisiana

B. Copenhagen City Heart Study:

- Began in 1975 as a prospective study of 20,000 men and women in Denmark, initially focused on CVD and stroke but since expanded to include many other cardiac, mental health, neurologic and pulmonary conditions

C. Dallas Hearts and Minds Study:

- Began in 2000 focused on a range of cardiac conditions including CVD, hypertension, and heart failure in TX.

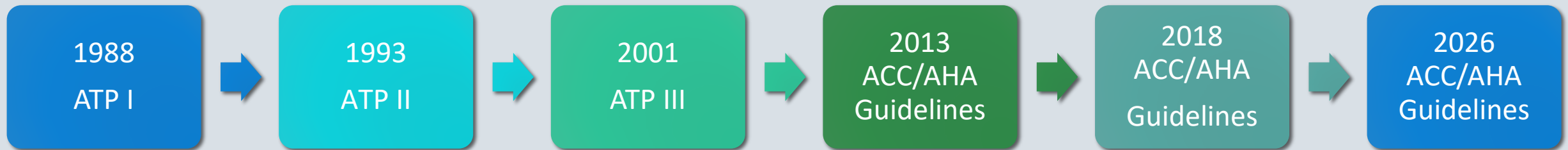
D. Framingham Heart Study:

- Began in 1948 and has tracked CVD development over 3 generations of participants in MA.

C. Jackson Heart Study:

- Began in 1997 in Mississippi and is the largest epidemiologic study of CVD in Black and African-American patients.

Clinical Practice Guidelines



Burden of CVD

Global mortality estimated at 19.2– 20.5 million/year (2021-23) and represents approximately 32% of all global deaths

- Rates vary geographically with the highest rates in Eastern European countries (553 deaths/100,000) compared to Australia/Asian countries (122.5 deaths/100,000)
- Approximately 125 million (50%) of all US adults had one or more CVD conditions
 - One person dies every 33 seconds from CVD
 - Cost estimates range between \$240-250 billion/year for healthcare, medications, and lost productivity

Estimated average years of life lost due to MI: **10 (women); 12 (men)**

Recent updates and key highlights



AHA PREVENT risk calculator replaces pooled cohorts equations as primary method to estimate 10-year ASCVD risk / guide lipid lowering therapy beginning at age 30

Established CPR model (Calculate 10-year risk; Personalize with factors not included in equation; Reclassify with Coronary artery calcium and Reassess treatment)



LDL therapy may be considered for those w/ASCVD risk of 3-5% and should be considered for those w/5-10% risk, using shared medical care



Re-established LDL-C and HDL-C goals to guide treatment

Percent reduction in LDL-C remains the priority



Adults aged 40-75 years with diabetes, CKD stage 3 or higher, or HIV should receive lipid lowering therapy regardless of LDL-C level

For those >75 years, should be considered in combination w/lifestyle modifications



LDL-C goal <55 mg/dL and HDL-C <85 mg/dL is recommended for secondary prevention for those at very high risk of recurrence



Clinical Practice Guidelines

Purpose: Early and aggressive hyperlipidemia detection/treatment

Primary CHD prevention emphasized

LDL: primary target but HDL is brought back into focus (out as of 2018)

- Lower the LDL, lower the CHD risk over the lifespan, even for those with low risk

Introduction of “CPR” approach: Calculate 10-year risk, Personalize/Patient factors, Reclassify and reassess with labs and treatment outcomes

[AHA PREVENT \(Predict Risk of CV Events\) calculator:](#)

- Provides 10- and 30-year risk estimates for CVD, ASCVD, and HF in ages 30 - 79

AHA ASCVD risk calculator

Sex
☒ Male ☐ Female

Age (years)
30-79

SBP (mmHg)
90-200

Total Cholesterol (mg/dL)
130-320

HDL Cholesterol (mg/dL)
20-100

eGFR (mL/min/1.73m²)
15-140

BMI (kg/m²)
18.5-39.9

Diabetes
Any history of diabetes.
☒ No ☐ Yes

Current Smoking
Any cigarette use within the last 30 days
☒ No ☐ Yes

Lipid-lowering medication
Current use of statin medication to lower cholesterol
☒ No ☐ Yes

Anti-hypertensive medication
Current use of any medication for hypertension
☒ No ☐ Yes

Comparison of Demographic Factors, Predictors, and Outcomes in the newly developed 2023 PREVENT equations and the ACC/AHA 2013 Pooled Cohort Equations

	PREVENT	PCEs
Demographic factors		
Age	Modeled as the time scale	Predictor
Sex	Sex-specific equations	Sex-specific equations
Race	Race free	Race specific
Clinical predictors		
Systolic blood pressure	X	X
Antihypertensive treatment	X	X
Total cholesterol		X
Non-HDL cholesterol	X	
HDL cholesterol		X
Statin treatment	X	
Diabetes	X	X
HbA1C	X	
Tobacco use	X	X
eGFR	X	
UACR	X	
Social Determinants of Health		
Education		
Income		
SDI	X	
Outcomes		
CHD	X	X
Stroke	X	X
HF	X	



Lipid Panel Monitoring

LDL-C: primary target for lipid lowering therapy

- Typically calculated by the lab, rather than measured directly

AHA guidelines recommends either [Martin equation](#) or the [Sampson equation](#)

Non-HDL: LDL + VLDL

- Encompasses all known potential atherogenic particles
- Proven a stronger predictor of CVD death than LDL in ↑ TG pts, DM pts, and women

Apolipoprotein B: measurement of plaque-forming particles in the blood

Lipoprotein(a): genetic test measuring presence of this cholesterol-carrying protein

Biomarkers and non-invasive testing

Biomarkers that may serve as risk enhancers:

- $\text{hs-CRP} \geq 2$; Lipoprotein(a) > 50 ; apolipoprotein B ≥ 130 ; ankle-brachial index < 0.9

High sensitivity C-reactive protein: non-specific marker of inflammation

Lipoprotein A (LpA): genetic marker of cholesterol particles

Apolipoprotein B (ApoB): primary protein found in LDL, VLDL, and IDL, transports cholesterol

Coronary artery calcium (CAC): measurement of calcified plaque in the arteries

Summary of major guideline updates discussed thus far

1. New approach to calculating 10-year risk of ASCVD using PREVENT
2. CPR approach: Calculate, Personalize/Patient-specific, Recalculate/Reassess
3. Lipid-lowering therapy may be indicated with a 10-year ASCVD risk $<3\%$
4. Biomarkers such as hs-CRP or lipoproteins serve as “risk enhancers” or accelerators for border-line patients

Patient case

Any volunteers willing to share their medical information?

Needed information: Age, Ht/Wt/BMI, SBP, eGFR, Hx of diabetes, tobacco use (30 days), current use of anti-hypertensive or lipid-lowering medications

Patient JL

45 y.o male, 5'10", 70.3 kg / BMI: 22.67

PMH: Hx of DVT

Medications: flaxseed, MVI, vitamin D 2000 IU

SH: No history of tobacco use

Vitals: P 122/68 (BP Location (NBP): Right arm, Patient Position: Sitting, | Pulse 71 | Temp 36.2 °C (97.1 °F) (Temporal) | Ht 177.8 cm (5' 10") | Wt 71.7 kg (158 lb) | SpO2 100% | BMI 22.67 kg/m²

Labs

Component	Value	Flag	Ref Range	Units	Status
Cholesterol, Total	183			mg/dL	Final
Comment:					
Desirable: < 200 mg/dL Borderline High: 200 - 239 mg/dL High: > or = 240 mg/dL					
Triglyceride	45			mg/dL	Final
Comment:					
Normal: <150 mg/dL Borderline High: 150-199 mg/dL High: 200-499 mg/dL Very High: > or =500 mg/dL					
HDL Cholesterol	77			mg/dL	Final
Comment:					
Males: High Risk: <40 mg/dL					
LDL Cholesterol	97			mg/dL	Final
Comment:					
Desirable: <100 mg/dL Above Desirable: 100-129 mg/dL Borderline High: 130-159 mg/dL High: 160-189 mg/dL Very High: > or =190 mg/dL LDL calculation was calculated using the NIH LDL Formula.					
Non-HDL Cholesterol	106			mg/dL	Final
Comment:					
Desirable: <130 mg/dL Above Desirable: 130-159 mg/dL Borderline High: 160-189 mg/dL High: 190-219 mg/dL Very High: > or = 220 mg/dL					
Est GFR by Creatinine - Male					
Normal Range: >=90 mL/min/1.73 m²					108 mL/min/1.73 m²

Total cholesterol: 183
Triglycerides: 45
HDL: 77
LDL: 97
Non-HDL: 106
eGFR: 108

Using the PREVENT calculator



American
Heart
Association

Results for CVD

Estimated **10-year**
risk of CVD

1.3%

Estimated
Prevent-Age
44
Years

Estimated **30-year**
risk of CVD

8.9%

Estimated **30-years**
Prevent-CVD risk
percentile
7th
percentile

CPR Framework for Risk Evaluation

LEGEND	
	COR 1
	COR 2a
	COR 2b
	COR 3-No Benefit
	COR 3-Harm
(Class of Recommendation)	

Calculate and Classify

Personalize

Reclassify and Reassess

Decide and Treat

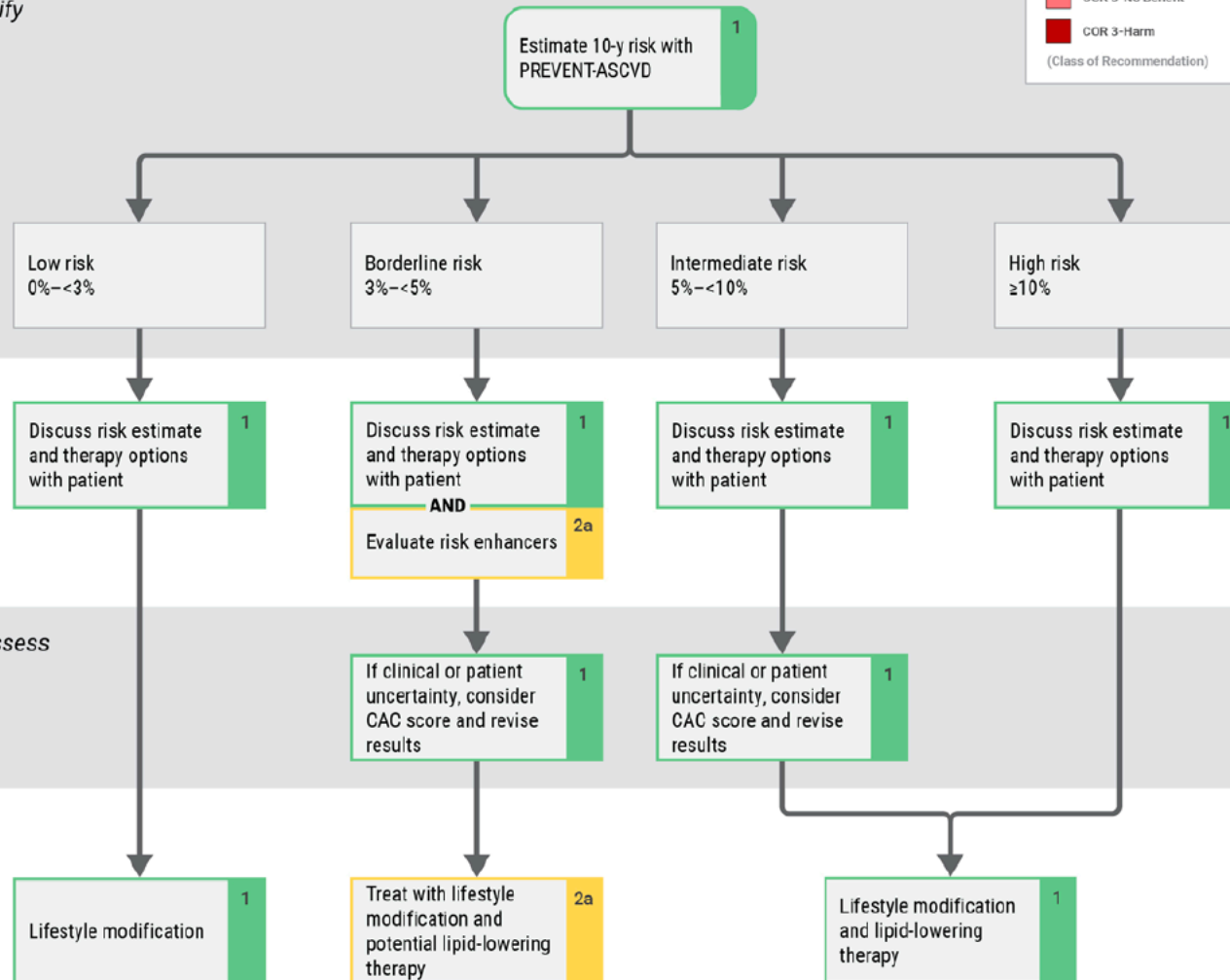


Table 13. Risk Enhancers

Risk Enhancers
History of premature ASCVD in a parent or sibling (onset age <55 y for men, <65 y for women)
Higher risk ancestry (eg, South Asian, Filipino)
High polygenic risk (if measured) (Section 4.2.3.5, "Polygenic Risk Scores")
Chronic inflammatory diseases (eg, systemic lupus, rheumatoid arthritis, advanced psoriasis, inflammatory arthritis)
Lp(a) ≥125 nmol/L or ≥50 mg/dL
hsCRP ≥2 mg/L on >1 occasion (if measured)
TG persistently ≥175 mg/dL (2 mmol/L) (if nonfasting) and ≥150 mg/dL (1.7 mmol/L) (if fasting)
CKM syndrome
LDL-C persistently ≥160–189 mg/dL (4.1–4.9 mmol/L), non-HDL-C ≥190–219 mg/dL or apoB ≥120 mg/dL*
Reproductive risk markers (premature menopause, preeclampsia, gestational diabetes, gestational hypertension, preterm delivery; Section 4.2.3.4, "Reproductive Risk Marker")

Lifestyle management (no longer modification)



Health Behavior Interventions in Patients With Hypertriglyceridemia

Implemented shared decision-making intervention	TG ≥150 to 499 mg/dL* (1.7 to <5.7 mmol/L)	TG ≥500 to 999 mg/dL* (5.7 to <11.3 mmol/L)	TG ≥1000 mg/dL [†] (≥11.3 mmol/L)
Added sugars (percent calories)	<6%	<5%	Eliminate
Total fat (percent calories)	30%–35%	20%–25% [‡]	10%–15% ^{§¶}
Alcohol	Avoid	Abstain completely	Abstain completely
Physical activity	At least 150 minutes/week of accumulated moderate-intensity or 75 minutes/week of vigorous intensity aerobic activity (or equivalent combination of both) and 2 days/week of upper and lower body resistance exercise		
Weight loss (percent body weight)	Recommended weight loss goal of 5%–10% for patients who are overweight or obese with elevated TG		



2026 Dyslipidemia

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How to approach targeted pharmacotherapy interventions

Recall, this guideline brought “back” laboratory targets

- 30-50% reduction from baseline (most common approach)
- LDL-C < 100 mg/dL for most, consider <70 or <55 in high-risk patient populations
- Non-HDL < 130 mg/dL for most, consider <100 or <85 in high-risk patient populations
- For specific patient populations, targets for apoB and CAC also exist

Primary Prevention in Adults 30–79 Y Without ASCVD

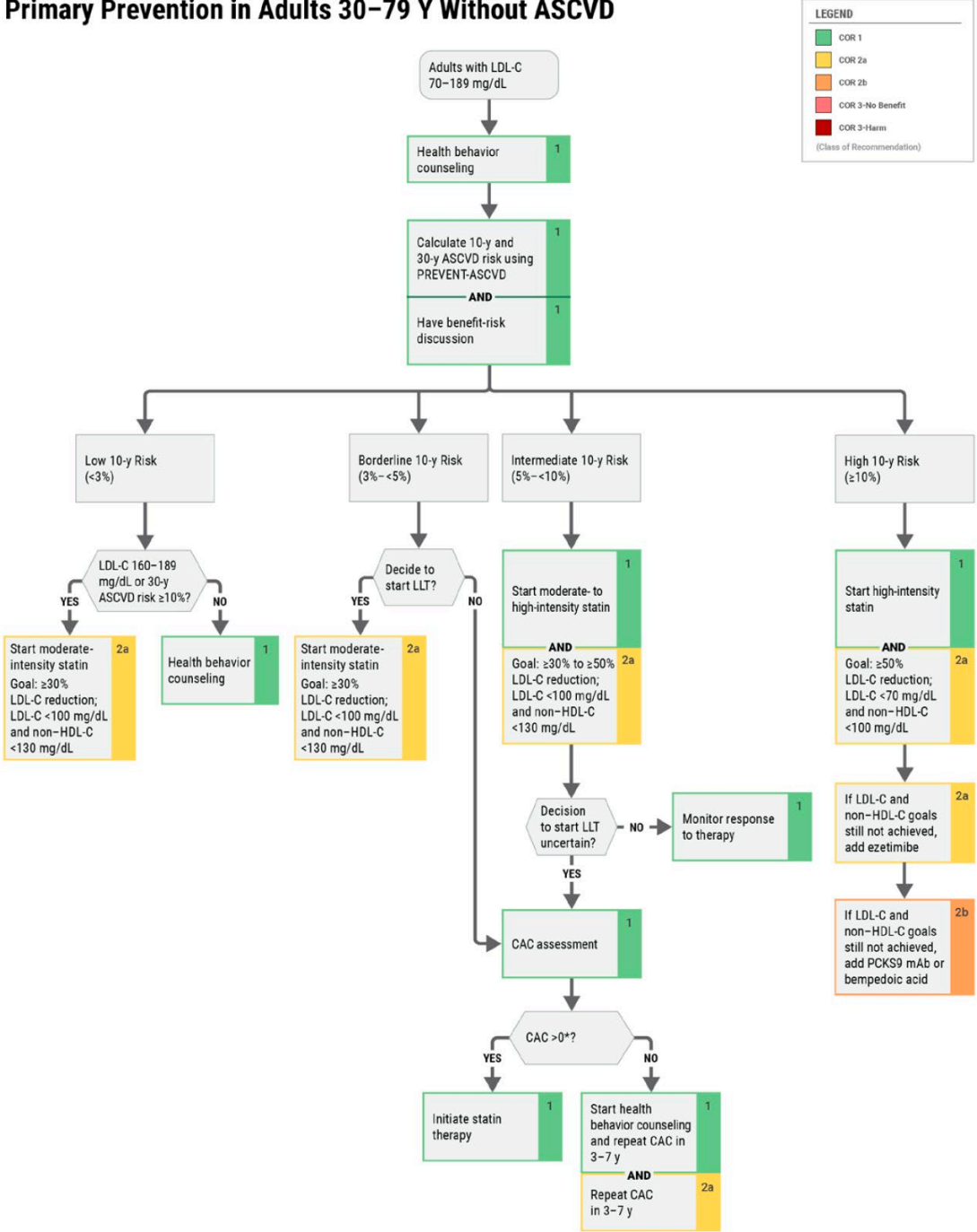


Table 6. High-, Moderate-, and Low-Intensity Statin Therapy*

	High-Intensity	Moderate-Intensity	Low-Intensity
Expected % LDL-C Reduction†	≥50%	30%–49%	<30%
Preferred Statins	Atorvastatin (40 mg) 80 mg Rosuvastatin 20 mg (40 mg)	Atorvastatin 10 mg (20 mg) Rosuvastatin (5 mg) 10 mg	
Other Statins	–	Fluvastatin XL 80 mg Fluvastatin 40 mg BID Lovastatin 40 mg (80 mg) Pitavastatin 1, 2, 4 mg Pravastatin 40 mg (80 mg) Simvastatin 20, 40 mg‡	Fluvastatin 20, 40 mg Lovastatin 20 mg Pravastatin 10, 20 mg Simvastatin 10 mg

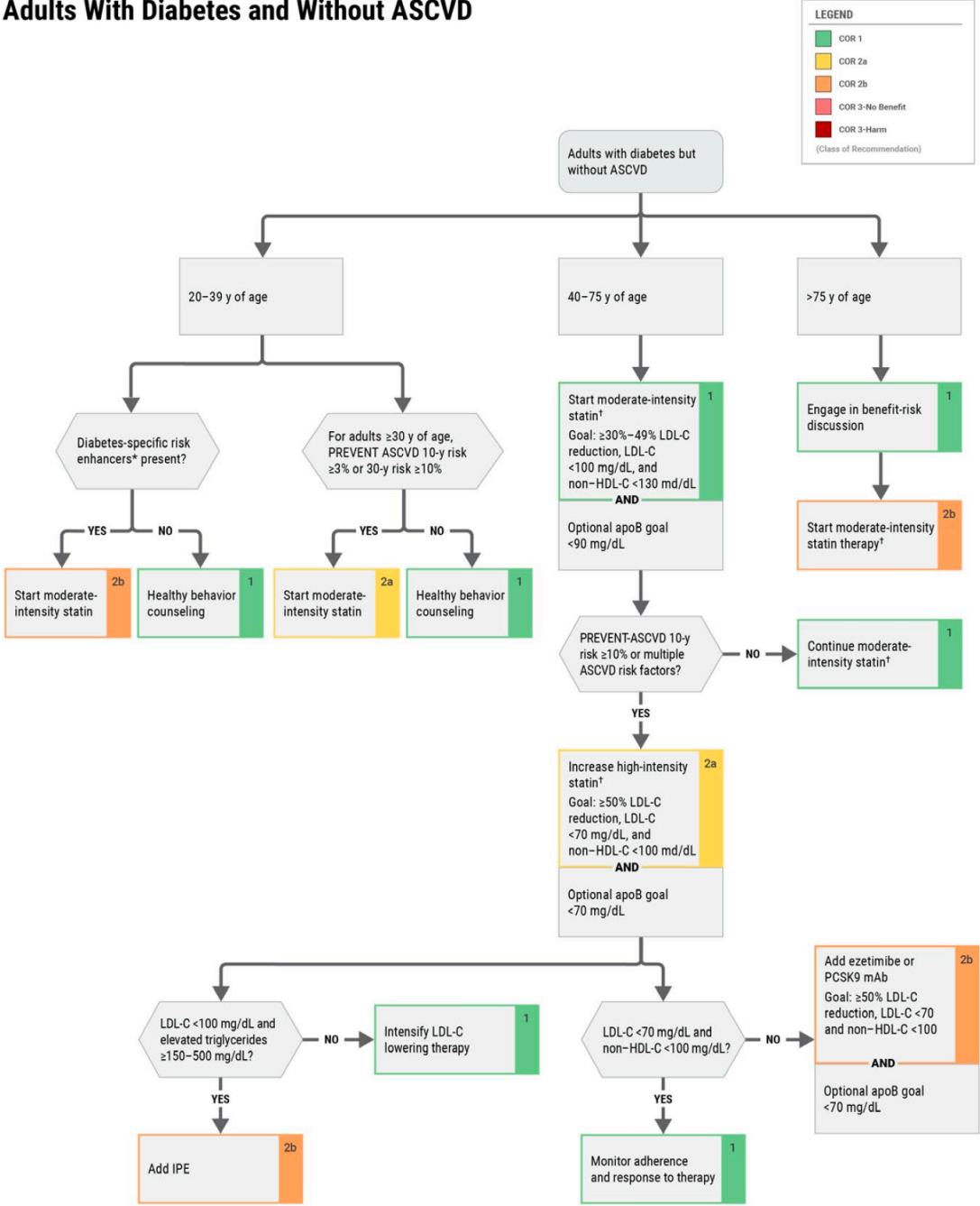


Table 17. Diabetes-Specific Risk Enhancers Independent of Other Diabetes-Related Risk Factors

Risk Enhancers
Long duration (≥10 y for type 2 diabetes or ≥20 y for type 1 diabetes)
Albuminuria ≥30 µg of albumin/mg creatinine
eGFR <60 mL/min/1.73 m ²
Retinopathy
Neuropathy
ABI <0.9

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ABI indicates ankle-brachial index; and eGFR, estimated glomerular filtration rate.

Lipoprotein Goals for ASCVD Risk Reduction

Patient population	LDL-C <100 mg/dL (2.6 mmol/L) Non-HDL-C <130 mg/dL (3.4 mmol/L)	LDL-C <70 mg/dL (1.8 mmol/L) Non-HDL-C <100 mg/dL (2.6 mmol/L)	LDL-C <55 mg/dL (1.4 mmol/L) Non-HDL-C <85 mg/dL (2.2 mmol/L)
Primary prevention	PREVENT-ASCVD <10% • If TG ≥150 mg/dL to 499 mg/dL, apoB goal: <90 mg/dL	PREVENT-ASCVD ≥10% • If TG ≥150 mg/dL to 499 mg/dL, apoB goal: <70 mg/dL	N/A
Severe hypercholesterolemia	Without FH, ASCVD risk factors, and subclinical atherosclerosis	With FH, ASCVD risk factors, or subclinical atherosclerosis	Severe hypercholesterolemia or HeFH with clinical ASCVD
Diabetes	Without ASCVD risk factors or diabetes-specific risk modifiers • apoB goal: <90 mg/dL	With ASCVD risk factors or diabetes-specific risk factors • apoB goal: <70 mg/dL	N/A
Subclinical atherosclerosis	CAC = 1–99 AU and <75th percentile for age, sex, and race	• CAC ≥100 to 299 AU or ≥75th percentile for age, sex, race • CAC ≥300 to 999 AU ◦ Optional goal: LDL-C <55 mg/dL, non-HDL-C <85 mg/dL and consider apoB goal <55 mg/dL	CAC ≥1000 AU
Hypertriglyceridemia	<50 y old with no additional risk enhancers	• With clinical ASCVD not at very high risk ◦ apoB goal: <70 mg/dL • Age 40–75 y with ≥1 ASCVD risk factor ◦ apoB goal: <70 mg/dL	With clinical ASCVD at very high risk • apoB goal: <55 mg/dL
Clinical ASCVD	N/A	Not at very high risk • Optional goal: LDL-C <55 mg/dL, non-HDL-C <85 mg/dL and consider apoB goal <55 mg/dL	• At very high risk ◦ apoB goal: <55 mg/dL • With CKD



Hyperlipidemia Agents

First line agents:

- HMG CoA reductase inhibitors (Statins)
- Selective cholesterol absorption inhibitors
- Proprotein convertase subtilisin kexin (PCSK) 9 inhibitors

Add-on in specific situations:

- Adenosine triphosphate-citrate lyase inhibitor
- Small interfering RNA treatment
- Bile acid sequestrants
- Niacin (Nicotinic acid)
- Fibric acid derivatives
- Omega-3-fatty acids

Table 5. Characteristics of Common Lipid-Lowering Medications to Treat Dyslipidemia*

Class	Mechanism of Action	Medications	Typical Dose Range	Dosing Frequency	Comments†
LDL-C–Lowering Medications					
HMG-CoA reductase inhibitors (aka statins)	Competitively inhibits HMG-CoA reductase (rate-limiting step of endogenous cholesterol production); increases LDL receptor expression on the surface of hepatocytes	Atorvastatin	10–80 mg	Once daily	Oral agents First-line therapy for ASCVD risk reduction in nearly all patients Extensive CVOT evidence demonstrates reductions in events over wide range of LDL-C levels in primary and secondary prevention Expected LDL-C reduction: 18%–55%‡ Atorvastatin and rosuvastatin can achieve high-intensity LDL-C reductions and are preferred for patients at high/very high ASCVD risk Atorvastatin, pitavastatin, and rosuvastatin have long half-lives that enable dosing any time of the day
		Fluvastatin	20–80 mg	Once or twice daily	
		Lovastatin	10–80 mg	Once or twice daily	
		Pitavastatin	1–4 mg	Once daily	
		Pravastatin	10–80 mg	Once daily	
		Rosuvastatin	5–40 mg	Once daily	
		Simvastatin	5–40 mg	Once daily	
Cholesterol absorption inhibitor	Blocks the sterol transporter protein, NPC1L1, to inhibit intestinal and biliary sterol absorption; increases LDL receptor expression on the surface of hepatocytes secondary to reduced hepatic sterol levels	Ezetimibe	10 mg	Once daily	Oral agent CVOT evidence demonstrates reductions in cardiovascular events in very high-risk secondary prevention in combination with a moderate-intensity statin therapy Expected LDL-C reduction: monotherapy, 18%; combination with a statin, 25% incremental reduction‡ Drug of choice in sitosterolemia to reduce elevated sitosterol and campesterol

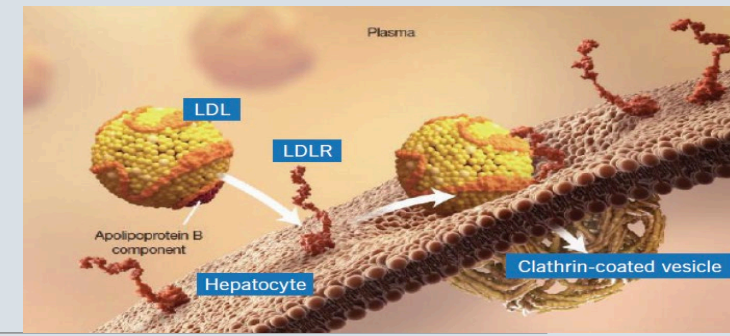
Table 5. Characteristics of Common Lipid-Lowering Medications to Treat Dyslipidemia*

Class	Mechanism of Action	Medications	Typical Dose Range	Dosing Frequency	Comments†
LDL-C-Lowering Medications					
PCSK9 inhibitor: monoclonal antibodies	Fully human monoclonal antibodies: binds to PCSK9 in the circulation and decreases degradation of LDL receptors	Alirocumab	75–150 mg, or 300 mg	Every 2 wk Every 4 wk	Subcutaneous agents CVOT evidence demonstrates reductions in cardiovascular events in very high-risk secondary prevention in combination with a maximally tolerated statin therapy Expected LDL-C reduction: 45%–64%‡ Lower mean LDL-C reduction (21%–31%) in homozygous FH due to <i>LDLR</i> gene variants ^{4,5}
		Evolocumab	140 mg	Every 2 wk	
ATP citrate lyase inhibitor	Inhibits ATP citrate lyase in the liver to decrease cholesterol production upstream of HMG-Co reductase in the cholesterol synthesis pathway; increases LDL receptor expression on the surface of hepatocytes	Bempedoic acid	180 mg	Once daily	Oral agent CVOT evidence demonstrating reductions in cardiovascular events in individuals treated for high-risk primary and secondary prevention with statin-attributed side effects Prodrug that is activated by very long-chain acyl-CoA synthetase, found primarily in the liver Expected LDL-C reduction: monotherapy in patients with statin-attributed side effects, 21%–24% ^{6,7} combination with a statin, 17%–18%‡

Table 5. Characteristics of Common Lipid-Lowering Medications to Treat Dyslipidemia*

Class	Mechanism of Action	Medications	Typical Dose Range	Dosing Frequency	Comments†
LDL-C–Lowering Medications					
PCSK9 inhibitor: small interfering RNA	Utilizes endogenous RNA interference mechanism and directs catalytic breakdown of mRNA for PCSK9 and decreases degradation of the LDL receptor	Inclisiran	284 mg	Initial dose followed by second dose in 3 mo, then every 6 mo thereafter	Subcutaneous agent administered by a health care professional Expected LDL-C reduction: 48%–52%‡ CVOT in progress
Bile acid sequestrants	Bind bile acids in the gut, interrupt enterohepatic recirculation of bile acids and impede their reabsorption, decrease bile acid pooling in the liver, increase conversion of cholesterol to bile acids; increase LDL receptor expression on the surface of hepatocytes	Cholestyramine	8–16 g	Once or twice daily	Oral agents CVOT evidence from 1 trial demonstrating cardiovascular event reduction in primary prevention men as monotherapy Nonsystemic, add-on to statin therapy or in patients with statin-intolerance Expected LDL-C reduction: 10%–27%‡ Gastrointestinal side effects may limit use Both LDL-C lowering and incidence of side effects are dose related May increase serum TG levels; avoid if TG ≥300 mg/dL
		Colesevelam	3.75 mg	Once or twice daily	
		Colestipol	2–16 g	Once or multiple times daily	

Oral PCSK9 Inhibitor



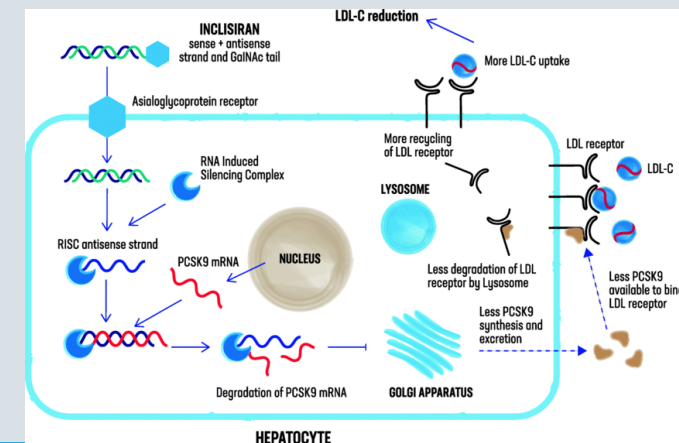
Enlicitide (Lipfendra™)

- MOA: act to increase LDLR (LDL-receptors) available to bind to circulating LDL, previously blocked by PCSK9, reducing circulating LDL
- FDA approved July 2026
- Dosed at 20 mg/day with similar LDL-C lowering compared to injectable agents (55-60% reduction) at a similar cost, and reductions may be seen as early as within 30 days

Small interfering RNA treatment

Inclisiran (Leqvio™): Targets the PCSK9 enzyme but rather than specifically inhibiting its functionality, it is uptaken into the PCSK9 DNA and acts as a DNA terminating agent

- Approved in late 2022 as an adjunctive LDL-lowering agent in patients unable to tolerate PCSK9 inhibitors, unable to self-inject, or those with poor adherence, who have not yet achieved LDL goals, despite maximally tolerated statin therapy
- Administered as a twice yearly after initial titration at months 0 and 3
- Trial data supports a reduction in LDL by up to 60%
- ORION 1-3 trials have demonstrated sustained LDL levels of approximately 50% at 4 year with minimal adverse effects (injection site reactions being most common)

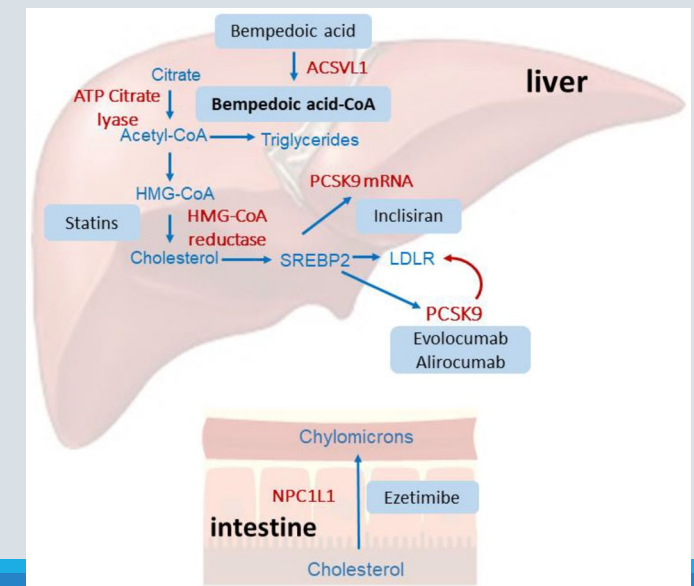


Adenosine triphosphate-citrate lyase inhibitor

Bempedoic acid (NexletolTM): inhibits hepatic cholesterol synthesis (ACL is upstream of HMG CoA reductase) and upregulates LDL receptors (lowering LDL-C)

Dosing: 180 mg once daily (recommended with maximally tolerated statin dose)

- Dose limitations exist for pravastatin (40 mg) and simvastatin (20 mg) due to drug-drug interactions with OATP1B1/B3 (organic anion-transporting polypeptide) which is responsible for hepatic uptake of medications, which may cause over-exposure increasing risk of myalgia/ALT increases



Class	Mechanism of Action	Medications	Typical Dose Range	Dosing Frequency	Comments†
Triglyceride-Lowering Medications					
Fibrates	Stimulates PPAR-alpha, which activates lipoprotein lipase and reduces apolipoprotein C-III production; increases lipolysis and elimination of TG-rich particles	Fenofibrate	40–200 mg	Once daily	Oral agents CVOTs show no reduction in cardiovascular events when fenofibrate is added to statin therapy in primary prevention patients with diabetes; CVOT evidence demonstrating reductions in cardiovascular events in primary and secondary prevention patients with gemfibrozil monotherapy First-line option for severe hypertriglyceridemia (≥ 500 mg/dL, especially when ≥ 1000 mg/dL) Expected TG reduction: 30%–50%‡ Many different formulations of fenofibrate and fenofibric acid are available with varied dosages Dose may need to be reduced in decreased kidney function; avoid in severe kidney dysfunction Blunted to no TG reduction in patients with familial chylomicronemia syndrome due to reduced lipoprotein lipase activity^a Gemfibrozil should not be combined with statin therapy because of serious potential drug interaction
		Fenofibric acid	35–135 mg	Once daily	
		Gemfibrozil	600 mg	Twice daily	
Omega-3 fatty acids	Reduces hepatic VLDL TG synthesis and/or secretion; enhances TG clearance from circulating VLDL; other mechanisms are possible	Icosapent ethyl	4 g	Twice daily with food	Oral agents First-line option for severe (≥ 500 mg/dL) hypertriglyceridemia CVOT evidence demonstrating reductions in cardiovascular events with icosapent ethyl in individuals being treated for primary prevention of ASCVD patients with type 2 diabetes or secondary prevention if TG between 150–499 mg/dL and LDL-C is 41–100 mg/dL despite statin therapy Expected TG reduction: 15%–61%‡ Icosapent ethyl contains EPA only Omega-3 acid ethyl esters contain DHA and EPA, which can modestly raise LDL-C (but not apoB) Nonprescription fish oil supplements are not equivalent to, or considered prescription medications, and have variable fatty acid content Give with a fat-containing meal to ensure absorption (pancreatic lipase stimulation breaks ethyl bonds to facilitate absorption)
		Omega-3 acid ethyl esters	4 g	Once or twice daily with food	

Table 5. Continued

Class	Mechanism of Action	Medications	Typical Dose Range	Dosing Frequency	Comments†
Niacin	Reduces esterification of hepatic TG, decreases release of free fatty acids from adipose tissue and increases activity of lipoprotein lipase to enhance removal of TG-rich lipoprotein fatty acids	Extended-release niacin	500–2000 mg	Once daily	Oral agents CVOTs show no reduction in cardiovascular events when added to statin therapy in individuals being treated for secondary prevention of ASCVD Last-line agent for severe hypertriglyceridemia due to relatively high risk of intolerable side effects; must initiate low dose to minimize side effects and slowly titrate up to an effective dose Expected TG reduction: sustained-release, 10%–30%; immediate-release 20%–50%‡ Increases insulin resistance, high rates of skin flushing, and significant hepatotoxicity
		Immediate-release niacin	250–6000 mg	Twice to 3 times daily	

Other clinical areas with a lack of clear data

Pregnancy / breastfeeding:

- FDA and other guidelines list as contraindicated due to risk of teratogenicity secondary to pleotropic effects in developing fetus/neonates
- 2021 systematic review and meta-analysis reviewed previously published data of 1.3 million subjects, found no increased risk of statins causing birth defects, cardiac, or other, congenital abnormalities (all $p > 0.1$)

Hemodialysis:

- 2020 retrospective study of 65,000 patients in South Korea followed for median of 3.5 years
- Use of statin therapy demonstrated lower risk of all-cause mortality compared to non-users. Use of combination of statin and ezetimibe demonstrated even higher rates of survival

Heart failure:

- Several studies demonstrated lower rates of cholesterol is associated with higher mortality in patients with HF
 - Theorized to be due to increased infection risk secondary to lipoprotein reduction and/or reduction of ubiquinone leading to cardiac toxicity.
- 2011 meta-analysis of 45,000 subjects (stages II-IV) followed for median of 4 years demonstrated reduction in all cause mortality and cardiovascular readmission, but not in cardiac mortality.
 - Similar findings reiterated in 2025 where LDL < 100 was associated with higher all-cause mortality but not ischemic events in HFrEF

Table 20. Lipid-Lowering Therapies During Pregnancy and Lactation

	Pregnancy	Lactation
Statins	Should be discontinued in most pregnancies Can be considered in high-risk individuals (ASCVD or FH ²⁹)	Avoid use ³⁰
Ezetimibe	Avoid use due to insufficient data regarding risk to fetus ³¹	Avoid use ³¹
Bile acid sequestrants	Safe to use. No evidence of risk in humans due to lack of systemic absorption ¹² Known to interfere with absorption of fat-soluble vitamins High rate of gastrointestinal side effects	Not excreted in human milk ¹² Caution if used—associated with malabsorption of fat-soluble vitamins (A, D, E, and K). Prenatal vitamins may not be sufficient
Niacin	Avoid use due to insufficient data regarding risk to fetus or clinical utility for the mother ¹²	Avoid use ¹²
Fibric acid derivatives	Can be considered (after the first trimester) for severe hypertriglyceridemia only if the potential benefit justifies the potential risk to the fetus ¹²	Avoid use during lactation. If taken during pregnancy, lactation can be resumed 5 days after last dose of fibric acid derivatives ¹²
Omega-3 fatty acids	Can be considered for severe hypertriglyceridemia ¹²	Known to be excreted in human milk. Effects on infants are unknown Caution if used during lactation ¹²
Bempedoic acid	Avoid use due to insufficient data regarding risk to fetus ¹²	Avoid use ¹²
PCSK9 mAb	Avoid use due to insufficient data regarding risk to fetus ¹²	Avoid use ¹²
Inclisiran	Avoid use due to insufficient data regarding risk to fetus ²⁹	Avoid use ²⁹
Evinacumab	Avoid use due to insufficient data regarding risk to fetus ³²	Avoid use ³²
Lomitapide	Avoid use due to risk of embryo-fetal toxicity ³¹	Avoid use ³³

Table 19. Lipid-Lowering Agents in Childhood and Adolescents (eg, <Age 18 Years)

Medication	FDA Approved Dosing for Use in Childhood	FDA Indicated Age	Expected LDL-C Reduction at Maximum Pediatric Dose	Potential Adverse Effects
Statins ⁵				Myalgias (rare), abdominal pain (rare), accelerated risk of diabetes type 2 (unknown, reported in adults), hepatotoxicity (very rare), HMG-CoA antibody myopathy (very rare) rhabdomyolysis (extremely rare)
Atorvastatin	5 mg, 10 mg, 20 mg, 40 mg, 80 mg PO/d	≥10 y	≥50%	
Pitavastatin	1 mg, 2 mg, 4 mg PO/d	≥8 y	30–49%	
Pravastatin	10 mg, 20 mg, 40 mg PO/d	≥8 y	30–49%	
Simvastatin	5 mg, 10 mg, 20 mg, 40 mg* PO/d	≥10 y	30–49%	
Rosuvastatin	5 mg, 10 mg, 20 mg PO/d	≥8 y for 5 and 10 mg doses and >10 y for 20 mg dose	≥50%	
Cholesterol absorption inhibitors				
Ezetimibe	10 mg PO/d	≥10 y	13–20% ^{9,43}	Myalgias, muscle weakness, fatigue, diarrhea (all very rare)
Bile acid binding resin	Colesevelam: 3.75 g PO/d as 1 packet or six 625 mg tablets divided twice daily	≥10 y	15% ^{33,34}	Significant drug interactions exist. Contraindicated if TG are elevated (will raise TG). Abdominal pain, constipation (rare), pancreatitis (very rare)
PCSK9 monoclonal antibodies				
Evolocumab	140 mg SQ every 2 wk or 420 mg SQ every 2 wkt	≥10 y	38% (HeFH) 14% (HoFH) ⁸	Hypersensitivity (rare), nasopharyngitis (common), headache, sore throat (common)
Alirocumab	Pediatric dosing by weight in 2- or 4-wk intervals SQ	≥8 y	34–44% (HeFH) 13% (HoFH) ^{30,44}	Nasopharyngitis (common), tonsilitis (common), headache (common), injection site reaction (common). Attention and memory problems, syncope were rarely reported ⁹⁰
Other cholesterol-lowering medications				
Evinacumab	15 mg/kg IV q 4 wk	≥5 y old HoFH only	48% (HoFH) ⁴⁵	Fatigue, pyrexia, headache, oropharyngeal pain, nasopharyngitis, abdominal pain
Supplements				
Psyllium fiber	10 g PO/d		6–24% ⁴⁶	GI effects, although these tend to resolve with regular use
Plant Stanol/ Sterol	2 g PO/d (available as capsules or as food additives)		6–12% ⁴⁷	Diarrhea, steatorrhea

How might this information be used in
your daily lives?

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Performing medication and adherence reviews

- Identify optimization opportunities and refer to prescriber when appropriate
 - Oral PCSK-9, twice yearly injection treatment, escalation of statin doses
 - Drug-drug interactions
 - Use of non-lipid-lowering therapies for comorbid conditions (GLP-1 agonist, SGLT-2i)

Lifestyle counseling, including awareness and education over updated guidance

Meeting patients and their families where they're at

- Identify opportunities for community outreach and engagement, if appropriate to your workplace, that include non-professional settings

Objective #1: Identify at least two new changes to hyperlipidemia management in 2026



AHA PREVENT risk calculator replaces pooled cohorts equations as primary method to estimate 10-year ASCVD risk / guide lipid lowering therapy beginning at age 30



LDL therapy may be considered for those w/ASCVD risk of 3-5% and should be considered for those w/5-10% risk, using shared medical care



Re-established LDL-C and HDL-C goals to guide treatment

Assessment question 1

Which one change in approach to managing CVD is new for 2026?

- A. Continued use of the Pooled Cohorts equation to calculate 10-year ASCVD risk
- B. Implementation of the Cardiopulmonary Resuscitation (CPR) approach to customize treatment
- C. Initiation of lipid-lowering therapy with ASCVD risk less than 3%

Assessment question 1

Which one change in approach to managing CVD is new for 2026?

A. Continued use of the Pooled Cohorts equation to calculate 10-year ASCVD risk

- Incorrect: pooled cohorts was replaced by PREVENT

B. Implementation of the Cardiopulmonary Resuscitation (CPR) approach to customize treatment

- Incorrect: new “CPR” designation is Calculate 10-year risk, Personalize/Patient factors, Reclassify and Reassess

C. Initiation of lipid-lowering therapy with ASCVD risk less than 3%

- **Correct: Most patients will be indicated starting with ASCVD risk >3%, but discussions can occur at any risk level**

Objective #2: Differentiate patient goals and targets using new guideline recommendations



LDL therapy may be considered for those w/ASCVD risk of 3-5% and should be considered for those w/5-10% risk, using shared medical care



Adults aged 40-75 years with diabetes, CKD stage 3 or higher, or HIV should receive lipid lowering therapy regardless of LDL-C level



LDL-C goal <55 mg/dL and HDL-C <85 mg/dL is recommended for secondary prevention for those at very high risk of recurrence

Assessment question 2

Those, ages 40-75, with this medical condition are recommended to receive lipid-lowering therapy regardless of ASCVD risk/LDL-C levels?

- A. Gestational diabetes
- B. Heart failure (preserved or reduced)
- C. Hemodialysis dependency

Assessment question 2

Those, ages 40-75, with this medical condition are recommended to receive lipid-lowering therapy regardless of ASCVD risk/LDL-C levels?

A. Gestational diabetes

- Incorrect: Presence of diabetes (type 1 or 2) is indicated for treatment regardless of level

B. Heart failure (preserved or reduced)

- Incorrect: conflicting evidence exists over the role LDL-C <100 has on mortality in this population, but current guidelines recommend following ASCVD-risk guidance

C. Hemodialysis dependency

- **Correct: Starting at stage 3 CKD and above, treatment is indicated regardless of level**

Objective #3: Develop at least two new ways to support patient care using available literature



Identify opportunities to education over newly approved lipid-lowering agents (enlicitide & inclisiran), or newly indicated patient populations (<18 years, 30-40 years, or those with specific medication conditions e.g. CKD, Diabetes)



Monitor for drug-drug interactions, optimization and/or general educational opportunities around medication therapy or lifestyle management

Assessment question 3

Which agents are appropriate to initiate in patients as young as 8 years of age:

- A. Atorvastatin at any dose
- B. Ezetimibe at any dose
- C. Rosuvastatin at any dose

Assessment question 3

Which agents are appropriate to initiate in patients as young as 8 years of age:

A. Atorvastatin at any dose

- **Correct, all doses are indicated as safe for patients ages 8+ years**

B. Ezetimibe at any dose

- Incorrect: indicated as safe for patients ages 10+ years

C. Rosuvastatin at any dose

- Incorrect: 5 and 10 mg doses approved for ages 8-9 years, and 20 mg dose approved 10+ years

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