

2008 GUIDELINE FOR THE DIAGNOSIS AND MANAGEMENT OF DYSLIPIDEMIA FOR ADULTS ≥ 18 YEARS OF AGE

This clinical guideline is designed to assist clinicians by providing a framework for the evaluation and treatment of patients with or at risk for dyslipidemia. It is not intended to replace a clinician's judgement or to establish a protocol for the treatment of a particular condition. A guideline rarely will establish the only approach to a problem.

CONTENTS

1. Treatment Overview of Dyslipidemia.....	2
2. Major CHD Risk Factors (Exclusive of LDL-C).....	2
3. Screening Guidelines	3
4. Lipid Treatment Goals and Cutpoints.....	3
5. General Treatment Sequence Recommendations.....	4
6. Drug-Specific Treatment Recommendations	6
7. Dose Equivalence Approximations.....	7
8. Profiles of Major Drug Classes	7
9. Follow-up Evaluation and Maintenance Therapy.....	8
10. Dyslipidemia Medications	9
11. Resources.....	9
12. References.....	10
13. Acknowledgements & Disclosures	10

Treatment Overview of Dyslipidemia

Dyslipidemia is a powerful risk factor for coronary heart disease (CHD). Clinical trials conclusively have demonstrated that treatment of lipid disorders can reduce CHD morbidity and mortality. Treatment of lipid disorders also helps prevent stroke and peripheral arterial disease.

- 1. Basic principle.** The intensity of risk-reduction therapy should be based on the patient's risk of a CHD event. Patients with established CHD have a 10-year risk of having a CHD event (cardiac death or myocardial infarction) that is > 20% (> 2%/year). Accordingly, primary prevention patients with a similar risk ("**CHD Risk Equivalent**") should be treated as aggressively. Such patients include those with non-coronary atherosclerosis (cerebrovascular disease, peripheral vascular disease, abdominal aortic aneurysm), type 2 diabetes mellitus, or an estimated 10-year CHD risk > 20% using the Framingham risk factor equation. Similarly, the CHD risk of patients with extreme lipid abnormalities such as low-density lipoprotein cholesterol (LDL-C) > 190, high-density lipoprotein cholesterol (HDL-C) < 40, or triglycerides (TG) >500 mg/dL qualify for aggressive treatment. These patients often have genetic disorders and their CHD risk is not reflected adequately by standard risk prediction techniques.
- 2. Methods of risk assessment.** Risk assessment is accomplished by a 2-step procedure. Count the number of major risk factors³ (not including LDL-C). For persons with 2 or more risk factors, refine risk assessment using the Framingham risk factor equation. Easy to use, computerized and chart-based scoring tools to estimate 10-year risks of CHD events are available online at <http://www.nhlbi.nih.gov/guidelines/cholesterol> and at <http://hin.nhlbi.nih.gov/atpiiii/calculator.asp?usertype=prof>.

For screening and treatment, individuals can be categorized into five general risk categories:

- 1. Very High** –
 - Recent acute coronary syndrome¹⁹
 - CHD or non-coronary atherosclerotic vascular disease¹ and one of the following:
 - Diabetes mellitus
 - Metabolic Syndrome⁸
 - Current smoking
 - Chronic kidney disease
- 2. High** – CHD or CHD risk equivalent. Patients with extensive subclinical atherosclerosis, chronic kidney disease and recipients of solid organ transplant are also at high risk.
- 3. Moderately High** – No CHD with ≥ 2 risk factors **and** 10 yr CHD risk 10-20%
- 4. Moderate** – No CHD with ≥ 2 risk factors **and** 10 yr CHD risk < 10%
- 5. Lower** – No CHD with ≤ 1 risk factor

Major CHD Risk Factors (Exclusive of LDL-C)

- 3. In addition to high LDL-C, several factors increase the risk of CHD.**
 - Current cigarette smoking
 - Hypertension (blood pressure $\geq 140/90$ mmHg or current treatment with antihypertensive medication)
 - Low HDL-C (< 40 mg/dL)
 - Family history of premature CHD (1st-degree relative: male < 55 or female < 65 years old)
 - Age (male ≥ 45 , female ≥ 55 years old)

Note: HDL-C > 60 mg/dL can be considered a "negative" risk factor. Its presence may remove one risk factor from the count.

Screening Guidelines

Screening Risk Group	Begin Screening	Frequency	Test
CHD, CHD risk equivalent, or ≥ 2 risk factors	Age 20 years or at onset	Yearly	Fasting lipid panel
Familial dyslipidemia or family history of premature CHD	Age 20 years	2 years	Fasting lipid panel
None of the above	Age 20 years	5 years	Fasting lipid panel or non-fasting total cholesterol and HDL-C ⁴

- Screening to determine the presence of atherosclerosis or risk factors can occur at any routine visit. If non-fasting lipids are measured, obtain a fasting lipid profile if total cholesterol is > 200 mg/dL or HDL-C is < 40 mg/dL in a male or < 50 mg/dL in a female.
- For rare patients with CHD or a CHD risk equivalent but no evidence of a dyslipidemia, re-screen every year depending on presence of associated risk factors.

Lipid Treatment Goals and Cutpoints

Revised ATP III LDL-C goals based on recent clinical trial evidence

Treatment Risk Group	Primary Target = LDL-C	Consider Drug Therapy*	Secondary Target= Non-HDL-C ⁷ (if TG ≥ 200)
Very High ²	LDL-C < 70	≥ 70	> 100
High —CHD or CHD risk equivalent	LDL-C < 100 (< 70 optional ⁶)	≥ 100	Non-HDL-C < 130
Moderately High —No CHD with ≥ 2 risk factors and 10 yr CHD risk 10-20%	LDL-C < 130	≥ 130	Non-HDL-C < 160
Moderate —No CHD with ≥ 2 risk factors and 10 yr CHD risk $< 10\%$	LDL-C < 130	≥ 160	Non-HDL-C < 160
Lower —No CHD with ≤ 1 risk factor	LDL-C < 160	≥ 190	Non-HDL-C < 190

**Based on health care providers judgement, it is optional to treat patients to the lipid targets at the next higher level of RISK. For example, an LDL-C target of 70mg/dL is optional in all patients with CHD (see below). An LDL-C of 100 mg/dl is an option in patients at moderately high risk.*

- Optional target LDL-C < 70 mg/dL.** Option for anyone with CHD, but recommended for the following groups of CHD patients:
 - Baseline LDL-C < 100 mg/dL at time of diagnosis
 - Acute coronary syndrome
 - Diabetes mellitus
 - Severe, poorly controlled risk factors, especially smoking
 - Multiple risk factors of Metabolic Syndrome⁸, especially TG ≥ 200 and HDL-C < 40 mg/dL
 - Chronic kidney disease
- Non-HDL-C = total cholesterol minus HDL-C.** When triglycerides are ≥ 200 mg/dL, LDL-C levels are measured less accurately and may underestimate CHD risk. Therefore, for patients with elevated TG levels, treatment of the non-HDL-C level is a secondary target after the LDL-C target has been reached. The non-HDL-C level includes cholesterol carried in several atherogenic lipoproteins, such as LDL, very low-density lipoprotein (VLDL), intermediate density lipoprotein (IDL), and lipoprotein (a).

- 8. Metabolic Syndrome.** This is a common condition associated with increased CHD risk. Metabolic abnormalities include: hyperinsulinemia often accompanied by glucose intolerance, abdominal obesity, hypertension, hyperuricemia, prothrombotic state, proinflammatory state, and the atherogenic lipoprotein phenotype (low HDL-C, hypertriglyceridemia, small dense LDL particles). Treatment of insulin resistance with exercise, weight loss and medications is a secondary target of therapy, in addition to lipid goals. The diagnosis of metabolic syndrome depends on the presence of three (3) or more of the following factors:
- waist > 40" for males, 35" for females (lower for Asian Americans: >35" for males, 31" for females; and for patients with genetic disposition: >37" in males and 31" in females)
 - fasting glucose > 100 mg/dL
 - TG \geq 150 mg/dL
 - HDL-C < 40 mg/dL males, < 50 mg/dL females
 - blood pressure \geq 130/85 mmHg or current treatment with antihypertensive medication
- 9.** Type 2 diabetes mellitus and non-coronary atherosclerosis are CHD risk equivalents. Optimization of glycemic control may reduce TG. Use of metformin and pioglitazone may simplify the management of diabetic dyslipidemia because they raise HDL-C, lower TG levels, and reduce the number of small LDL particles.
- 10.** Secondary causes of hyperlipidemia should be identified and treated:
- type 2 diabetes mellitus⁹
 - obstructive liver disease
 - progestin use
 - Metabolic Syndrome⁸
 - renal disease
 - anabolic steroid use
 - hypothyroidism
 - corticosteroid use
 - alcohol use
- 11. Therapeutic Lifestyle Changes (TLC).** These should be instituted in all patients if LDL-C or non HDL-C are above goal. The TLC diet involves reduced intake of cholesterol-raising nutrients such as saturated and trans-fats, which should be restricted to < 7% of calories. Dietary cholesterol is restricted to < 200 mg per day. Additional LDL-C lowering options include 2 grams of plant stanol or sterols per day (fortified margines, yogurts, juices, etc.) and soluble fiber (3–6 g daily). Additional TG-lowering options include use of fish oil (omega-3 fatty acid) supplements (3-6 g daily). Weight reduction and increased physical activity are critical for the management of all dyslipidemias and to improve cardiovascular fitness. More information on TLC can be found within the NHBLI website at: <http://rover2.nhlbi.nih.gov/chd/lifestyles.htm>.

General Treatment Sequence Recommendations

Medication Sequence	Lipid Pattern		
	TG < 200 mg/dL	TG 200 – 499 mg/dL	TG \geq 500 mg/dL
1st	statin ¹³	statin ¹³ or niacin ¹⁴	niacin ¹⁴ , fish oils or fibrate
2nd	add niacin ¹⁴ , BAS ¹⁷ , or ezetimibe ¹⁶	statin + niacin ¹⁴	combination of niacin ¹⁴ , fish oils or fibrate
3rd	add niacin ¹⁴ , BAS ¹⁷ , or ezetimibe ¹⁶	add ezetimibe ¹⁶	add third TG-lowering drug, consider a statin

- 12. General Principles of drug therapy.** Drug therapy should be started if LDL-C or non-HDL-C are above target values. For patients with CHD or CHD risk equivalent, drug therapy may be considered in combination with TLC at any time. Patients at high risk and very high risk should start drug therapy immediately. The decision to initiate drug treatment usually commits patients to lifelong therapy. In general, single drug therapy is initiated; however, the lipid-lowering effect is not directly proportional to the dose. A 30-40% LDL-C reduction should be targeted. Doubling a statin dose may decrease LDL-C by an additional 5% to 7%. Doses reaching or exceeding maximum daily doses have little or no benefit and increase the risk of side-effects. Combination therapy frequently will increase the likelihood of reaching lipid goals. TLC should be instituted prior to or concurrent with drug therapy. All patients with dyslipidemia should be referred for dietary counseling, preferably with a trained provider.
- 13.** The TG-lowering effects of statins are dose- and potency-dependent. As TG levels increase, higher doses of statins are required. For moderate elevations of LDL-C in patients with elevated TG, prescription niacin (Niaspan)¹⁴ is an alternative. In general, statins should not be used alone in patients with TG >500 mg/dL.
- 14.** The American Heart Association recommends use of a prescription niacin (Niaspan) rather than niacin supplements.

Supplements are not regulated and often contain more or less than the labeled amount of niacin, vary from lot to lot, and some are more hepatotoxic.

15. In general, the combination of statins and fibrates is discouraged. If this combination must be used, fenofibrate is safer than gemfibrozil. If gemfibrozil is used, statin doses should be kept low. Be very cautious in older patients, those with kidney disease, and those on drugs that affect statin metabolism.
16. Ezetimibe inhibits intestinal cholesterol absorption. Statins are considered first line therapy for patients requiring LDL-C reduction. Use ezetimibe in combination with statins for those patients unable to reach goal on highest tolerated dose of a statin. BAS and niacin also may be used. Ezetimibe reduces LDL-C by 17-23%. Ezetimibe is a first line agent only for patients with contraindications or drug intolerances to statins. This medicine has not been proven to reduce CHD events.
17. Bile acid sequestrants (BAS) can raise TG levels. If TG levels are elevated at follow-up, consider switching to Niaspan¹⁴.
18. Omega-3 polyunsaturated fats (fish oils) at doses of 3-6 g/day lower TG levels by 30-50%. They should be administered with meals. Prescription fish oil (LOVAZA) is available. Most fish oil preparations that are available as dietary supplements are effective and also appear to be free of contaminants, but tend to be less potent. A non-prescription alternative that can be used is SUPER-EPA 2000. Fish oils are safe when used in combination with other lipid-lowering medications.
19. **Acute MI patients.** LDL-C and HDL-C levels may be artificially low due to inflammation during acute illnesses, such as acute coronary or cerebral syndromes. Heparin also can decrease TG levels. If lipid levels are checked in hospitalized patients it should be recognized that they tend to underestimate ambulatory, usual lipid levels. For patients with acute syndromes, high-dose statin therapy (e.g., atorvastatin 80 mg/dL) should be initiated to target LDL-C (70 mg/dL). Rapid and aggressive reduction of LDL-C after an acute coronary syndrome improves clinical outcomes.
20. **Following LDL-C after an Acute Cardiac Event.** For patients with an inpatient hospital stay for an acute cardiac event, LDL-C should be checked at least one time between 42 and 365 days after discharge to assure the patient is at target.
21. **Low HDL-C.** Low HDL-C is very common in persons with premature CHD, particularly males < 45 and females < 55 years old. A goal is to raise HDL-C as much as possible, by treating associated causes (hypertriglyceridemia, Metabolic Syndrome, physical inactivity, cigarette smoking, excessively high carbohydrate diets, etc). Achieving LDL-C and non-HDL-C targets and treatment of the Metabolic Syndrome are the dominant strategies. An isocaloric increase in dietary monounsaturated fats may help. For patients with isolated low HDL-C in whom drug therapy is appropriate, consider Niaspan 1000-2000 mg QHS, fish oils or fibrates in patients with elevated TG. Additional LDL-C lowering with a statin may be considered.
22. **Men <35 years old and pre-menopausal women.** The 10-year risk of CHD events is low unless they have severe risk factors, such as familial hypercholesterolemia, cigarette smoking, or diabetes mellitus. Even though clinical CHD is uncommon, subclinical atherosclerosis can progress rapidly. Furthermore, CHD is a lifelong disease and risk factors in youth predict premature CHD. TLC is the dominant strategy for young patients; however, drug therapy should be considered. Patients with extreme lipid abnormalities also should be treated aggressively.

23. Drug-Specific Treatment Recommendations*

Very High²			
TG Level	RX Goal	Start and maximize	Then add or switch to
< 499	LDL-C < 70	atorvastatin 40-80 mg, simvastatin 40-80 mg or Vytorin 20-80/10 mg**	Add Niaspan 1000-2000 mg daily or BAS
≥ 500	LDL-C < 70 and non-HDL-C < 100	Niaspan 2000 mg, omega-3 fish oils 4 g or fenofibrate 200 mg	Add Niaspan, omega-3 fish oils, fenofibrate, or statin
High – CHD OR CHD Equivalent			
TG < 200	LDL-C < 100 (optional < 70 ⁶)	If LDL-C ≥ 100, simvastatin 20-40 mg or lovastatin 40 mg QHS. If LDL-C ≥ 130, simvastatin 40 mg	If patients don't tolerate or reach goal on a high dose statin alone, add Niaspan 1000 mg-2000 mg QHS, ezetimibe, or BAS.
TG 200-499	LDL-C < 100 and Non-HDL-C < 130	simvastatin 20-40 mg QHS	Add Niaspan 1000-2000 mg QHS
TG ≥ 500	LDL-C < 100 and Non-HDL-C < 130	Niaspan 2000 mg QHS, omega-3 fish oils, or gemfibrozil 600 mg BID or fenofibrate 200 mg QD	Add Niaspan or omega-3 fish oils or fibrate
Moderately High/Moderate – Primary Prevention ≥ 2 Risk Factors and 10-year CHD Risk < 20%			
TG < 200	LDL-C < 130	If LDL-C ≥ 130, simvastatin 20 mg or lovastatin 40 mg QHS If LDL-C ≥ 160, simvastatin 20 – 40 mg or lovastatin 40-80 mg QHS If LDL-C ≥ 190, simvastatin 40 mg QHS	If patients don't tolerate or reach goal on a high dose statin alone, add Niaspan 1000 mg-2000 mg QHS, ezetimibe, or BAS.
TG 200-499	LDL-C < 130 and Non-HDL-C < 160	simvastatin 20-40 mg	Add Niaspan 1000-2000 mg QHS
TG ≥ 500	LDL-C < 130 and Non-HDL-C < 160	Niaspan 2000 mg QHS or omega-3 fish oils, gemfibrozil 600 mg BID or fenofibrate 200 mg QD	Add Niaspan or omega-3 fish oils or fibrate
Low – Primary Prevention ≤ 1 Risk Factor			
TG < 200	LDL-C < 160	If LDL-C ≥ 160, simvastatin 20 mg or lovastatin 40 mg QHS If LDL-C ≥ 190 simvastatin 20-40 mg or lovastatin 40-80 mg QHS If LDL-C ≥ 220, simvastatin 40 mg QHS	If patients don't tolerate or reach goal on a high dose statin alone, add Niaspan 1000 mg-2000 mg QHS, ezetimibe, or BAS.
TG 200-499	LDL-C < 160 and Non-HDL-C < 190	simvastatin 20-40 mg or lovastatin 40-80 mg QHS	Add Niaspan 1000-2000 mg QHS
TG ≥ 500	LDL-C < 160 and Non-HDL-C < 190	Niaspan 2000 mg QHS, omega-3 fish oils or gemfibrozil 600 mg BID or fenofibrate 200 mg QD	Add Niaspan, omega-3 fish oils or fibrate

* Specific treatment recommendations were made based on local HMO formulary coverages. These are subject to change at any time.

If switching to an alternative statin, use Table 24, below, for approximate equipotent dosing.

**This combination increases the chances of getting LDL-C to goal, but has not been proven to reduce cardiac events.

Consider referral to UW Health Preventative Cardiology Clinic for complicated patients with refractory dyslipidemia (requiring more than 2 drugs to reach target), suspected genetic dyslipidemias, drug intolerances or patients with secondary causes.

24. Dose Equivalence Approximations (for available strengths)

	<i>atorvastatin</i>	<i>fluvastatin</i>	<i>lovastatin</i>	<i>pravastatin</i>	<i>simvastatin</i>	<i>rosuvastatin</i>	<i>simvastatin/ ezetimibe</i>
Approximate Equipotent Dose	----	20 mg	10 mg	10 mg	5 mg	----	----
	5 mg	40 mg	20 mg	20 mg	10 mg	----	----
	10 mg	80 mg	40 mg	40 mg	20 mg	5 mg	----
	20 mg	----	80 mg	80 mg	40 mg	10 mg	10 mg/10 mg
	40 mg	----	----	----	80 mg	20 mg	10 mg/20 mg
	80 mg	----	----	----	----	40 mg	10 mg/40 mg
	----	----	----	----	----	----	10 mg/80 mg

If changing statins, start on equivalent dose and recheck LDL-C levels and transaminases after 6-8 weeks.

Simvastatin, lovastatin, and atorvastatin are metabolized by the CYP3A4 system, a common metabolic pathway for many medications including, but not limited to, protease inhibitors, azole antifungals, macrolide antibiotics and cyclosporine. Consider using other statins in patients with renal insufficiency or patients on multiple medications with other drugs metabolized by this isoenzyme system (such as verapamil, diltiazem, or amiodarone).

25. Profiles of Major Drug Classes

	Statins	Azetidinone	Nicotinic Acid	Omega-3 Fish Oils	Fibrates	Bile Acid Sequestrants
Medications	atorvastatin (Lipitor) fluvastatin lovastatin pravastatin rosuvastatin* (Crestor) simvastatin	ezetimibe (Zetia) ¹⁶	prescription niacin (Niaspan) ¹⁴ extended-release niacin/lovastatin (Advicor) ³⁰ extended release niacin/simvastatin (Simcor) ³⁰	LOVAZA	gemfibrozil fenofibrate (Lofibra, Tricor, Antara)	cholestyramine cholestyramine light colesevelam (Welchol)
Lipid Effects						
LDL-C	lower 18-60%	lower 17-23%	lower 10-25%	may raise	lower 5-20% (may raise)	lower 15-30%
HDL-C	raise 5-15%	raise 2%	raise 15-35%	raise	raise 10-20%	raise 3-5%
TG	lower 7-30%	lower 4-11%	lower 20-50%	lower 30-50%	lower 20-50%	none or raise
Major Use	elevated LDL-C, mild to moderately elevated TG, mildly low HDL-C	elevated LDL -C	moderately elevated LDL-C, elevated TG, low HDL-C	TG ≥ 500 mg/dl	TG > 400 mg/dL	moderately elevated LDL-C with normal TG
Contraindication - Absolute - Relative	liver disease ²⁸ pregnancy ²⁸ concomitant use of fibric acid derivatives ²⁷	severe liver disease	liver disease ²⁸ pregnancy ²⁸ gout, hyperuricemia	none	pregnancy ²⁸ liver or severe renal disease, gallstones	bowel obstruction TG > 200 mg/dL
Long Term Safety	extensive clinical use, 10-yr safety data		3 years safety data for Niaspan	10 years	yes	yes
Major Side Effects	myopathy, myalgia, abdominal pain, hepatotoxicity	well tolerated by most	flushing ²⁹ , abdominal pain, gout, hyperglycemia, hepatotoxicity, ulcers	flatulence indigestion burping	dyspepsia, gallstone pain, hepatotoxicity	constipation, flatulence, decreased drug absorption
Usual Daily Dose	see drug-specific treatment table ²³	10 mg QD	Niaspan ²⁹ 1000-2000 mg QHS Advicor 500/20 mg-2000/40 mg Simcor 1000/20 mg-2000/40 mg	4g QD	G [¶] = 600 mg BID L = 67-200 mg QD T = 54-160 mg QD	cholestyramine 4-16 g daily colesevelam 3750 mg daily
Maximum Daily Dose	see drug-specific treatment table ²³	10 mg QD	Niaspan 1000-2000 mg QHS Advicor 2000/40 mg Simcor 2000/40 mg	4g QD	G = 600 mg BID L = 200 mg QD T = 160 mg QD	cholestyramine 24 g daily colesevelam 4375 mg daily
LFT Monitoring ²⁷	0,2 then q 12 mos.	0,2, then q 12 mos	0,2,6 then q 12 mos.	none	0,2,6 then q 12 mos.	none
CPK monitoring ²⁷	muscle aches, pains, tenderness	none	muscle aches, pains, tenderness	none	muscle aches, pains, tenderness	none

[¶]G=gemfibrozil, L=Lofibra, T=Trico

Follow up Evaluation and Maintenance Therapy

26. Fasting lipid levels should be obtained 6-8 weeks following the initiation or alteration of therapy. For patients at goal, lipid profiles should be performed annually.
27. Patients should be monitored for side effects of antilipidemic therapy as outlined in the drug profiles above. All medications can cause transaminase elevation (incidence approximately 1%) and myopathy (< 0.1%). The risk is higher at the higher statin doses. If ALT or AST > 3x normal or if the patient develops myopathy with an elevated CPK-MM fraction, the antilipidemic agent dose should be reduced, the agent should be changed, or the patient referred for further evaluation by a lipid management specialist. Transaminase should be repeated to assess progression or resolution.
28. Pregnancy is an absolute contraindication for the use of lipid-lowering agents because the risks to the fetus outweigh the short-term benefits of cholesterol reduction.
29. To minimize Niaspan-associated flushing and headaches introduce with 500 mg QHS and titrate upwards every 4 weeks. Take with a low-fat snack and aspirin (325 mg). The maximum nightly dose of Niaspan is 2000 mg. Niaspan is associated with a lower incidence of LFT elevation than previous sustained-release niacin products; however, LFTs still need periodic monitoring. The increase in HDL-C with Niaspan continues after 4 weeks. Transaminases should be checked after 6 weeks of therapy then every 6-12 weeks for 6 months. Rechecking lipids may be deferred to 12 or even 16 weeks to see the maximum HDL-C effect, before titrating above 1000 mg QHS. Serum glucose and uric acid should be monitored periodically if patient is at risk for worsened glycemic control or gout.
30. Advicor and Simcor are fixed combinations of extended-release niacin and lovastatin (Advicor) or simvastatin (Simcor). These products may be useful for patients on a stable statin dose in whom niacin is going to be added, or for patients on a stable niacin dose for whom a statin dose for whom a statin is going to be added.
31. The most common reasons for drug failure include: 1) the wrong drug, 2) the wrong dose, 3) failure to use combination therapy, and 4) patient non-compliance. If the patient is compliant yet has failed to reach goal, referral to a specialty clinic is strongly recommended. Referral should also be considered for patients with familial dyslipidemia and patients requiring multiple risk factor management in a multidisciplinary setting.
32. **Advanced lipoprotein testing.** In some patients with premature heart disease or Metabolic Syndrome, LDL-C and non-HDL-C may underestimate the severity of the lipoprotein abnormality, leading to increased but unrecognized risk. *It should not be used in patients who have LDL-C or non-HDL-C above target values.* Advanced lipoprotein testing using the NMR Spectroscopy or measurement of apo B-100 can be considered in patients with:
 - strong family history of premature CHD
 - Metabolic Syndrome or type II diabetes mellitus
 - recurrent CHD events who are at lipid goal
 - patients with TG > 150 but without other components of Metabolic Syndrome
 - combined lipid disorders which are difficult to treat to target
 - an unexpected response to therapy

Dyslipidemia Medications - See plan-specific formularies for coverage determinations

Class	Medication	Dosage Strengths
Statins* <i>(Most HMOs include the statin class in their tablet splitting programs.)</i>	atorvastatin (Lipitor) fluvastatin (Lescol, XL) lovastatin lovastatin ER (Altoprev) pravastatin (Pravachol) rosuvastatin (Crestor) simvastatin (Zocor)	10, 20, 40, 80 mg 20, 40, 80(XL) mg 10, 20 40 mg 10, 20, 40, 60 mg SR 10, 20, 40, 80 mg 5. 10, 20, 40 mg 5, 10, 20, 40, 80 mg
Azetidione	ezetimibe (Zetia)	10 mg
Nicotinic Acid	Niaspan	500, 750, 1000 mg
Omega-3 fish oils	LOVAZA Super EPA 2000	1 mg 1000 mg
Fibrates	gemfibrozil fenofibrate (Tricor) (Triglide) (Antara) (Lipofen)	600 mg 67, 134, 200 mg 45, 145 mg 50, 160 mg 43, 87, 130 mg 50, 100, 150 mg
Bile Acid sequestrants	cholestyramine cholestyramine/light colesevelam (Welchol) colestipol (Colestid)	powder in packets or cans powder in packets or cans 625 mg tablets 1G tabs, 5G granule packets
Combinations	ezetimibe/simvastatin (Vytorin) niacin/lovastatin (Advicor) niacin extended release/simvastatin (Simcor) amlodipine/atorvastatin (Caduet) aspirin buffered/pravastatin (Pravigard PAC)	10/10, 10/20, 10/40, 10/80 mg 500/20, 1000/10 mg SR 500/20 mg, 750/20 mg, 1000/20 mg 2.5/10 2.5/20, 2.5/40, 5/10, 5/20, 5/40, 5/80 10/10, 10/20, 10/40, 10/80 mg 81/20, 81/40, 81/80, 325/20, 325/40, 325/80 mg

Resources

Most medical groups have a provider experienced at treating patients with difficult to manage dyslipidemia. Please consult regional facilities and providers for additional resources in your area. For specialty consultations or additional assistance, contact the UW Hospital and Clinics Preventive Cardiology program.

University of Wisconsin Hospital and Clinics Phone 608-263-1530
Preventive Cardiology Program 608-263-7420
600 N. Highland Avenue
Madison, WI 53792

For additional nutrition information contact the UW Health Nutrition Center at 608-287-2780, or one of the UW Health Outpatient Nutrition Clinics: UW Hospital 608-263-4360, West Clinic at 608-262-9181, East Clinic at 608-265-7405 or University Station at 608-263-7772. Consult local facilities and providers for additional resources in your area.

National Cholesterol Education Program <http://www.nhlbi.nih.gov/guidelines/cholesterol/>

Useful patient and physician materials including on-line and downloadable risk calculators² for desktop computers and palm computers.

American Heart Association <http://www.americanheart.org>

Resources for patients and professionals.

References

- Grundy SM, et al. Implications of Recent Clinical Trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. *Circulation* 2004; 110: 227-239
- Executive Summary of the 3rd Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA* 2001;285:2486-2497.
- Grundy SM. Role of therapy with “statins” in patients with hypertriglyceridemia. *Am J Cardiol* 1998;81:1B-6B.
- Stone NJ, Blum CB, Winslow E. *Management of lipids in clinical practice*. 5th ed. Caddo, OK, Professional Communications, Inc., 2005.
- Cannon CP, et al. Intensive versus Moderate Lipid Lowering with Statins after Acute Coronary Syndromes. *NEJM* 2004; 350: 1495-1504.
- Ballantyne CM, et al. Dose-comparison study of the combination of ezetimibe and simvastatin (Vytorin) versus atorvastatin in patients with hypercholesterolemia: The Vytorin Versus Atorvastatin (VYVA) Study. *American Heart Journal* 2005; 149: 464-473.
- McBride PE, Stein JH. Contemporary Diagnosis and management in Preventative Cardiology. *Handbooks in Health Care*, Newton, PA, 2006.

Acknowledgements & Disclosures

University of Wisconsin Medical Foundation, University of Wisconsin Hospitals and Clinics, Meriter Hospital, Physicians Plus Insurance Corporation, Unity Health Insurance and Group Health Cooperative have cooperated in the revision of these lipid management guidelines.

James Stein, MD and Patrick McBride, MD, MPH, both from the UW Preventive Cardiology Program and the University of Wisconsin School of Medicine and Public Health, led the revision efforts. Additional contributing task force members include: Jessica Bartell, MD; Fred Brodsky, MD; Maggie Dugan, NP; Lawrence Fleming, MD, MBA; Derek Hubbard, MD; Pam Kittleson, RPh; and Jennifer Schauer, PharmD.

Please direct questions, comments and suggestions regarding these guidelines to Pam Kittleson, RPh, UWMF provider education pharmacist, at pamela.kittleson@uwmf.wisc.edu.

Reviewed and updated February 2008

Scheduled for reconsideration second quarter 2010

Disclosures

These faculty have disclosed the following:

- James Stein, MD, Professor of Medicine, Division of Cardiovascular Medicine, and Director of the Preventive Cardiology Program, University of Wisconsin School of Medicine and Public Health
Scientific Consultant: Merck, Pfizer, Schering-Plough
Research/training grants: Bristol-Myers Squibb, Kos, Sanofi-Aventis, Takeda
Speaker's bureau: All honoraria donated directly to charity
- Patrick McBride, MD, Professor of Medicine and Family Medicine, Division of Cardiovascular Medicine, University of Wisconsin School of Medicine and Public Health
Consultant: Johnson & Johnson, Reliant