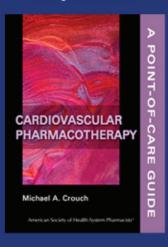


# Dyslipidemia

Contemporary Evaluation and Management

Evan M. Sisson and McKenzie Calhoun Michael A. Crouch, Guest Editor

adapted from





## Dyslipidemia

### Contemporary Evaluation and Management

#### EVAN M. SISSON, PHARMD, MHA, CDE, FAADE

Associate Professor Virginia Commonwealth University School of Pharmacy Richmond, Virginia

#### McKenzie CALHOUN, PHARMD

Assistant Professor Bill Gatton College of Pharmacy East Tennessee State University Johnson City, Tennessee

Guest Editor

#### MICHAEL A. CROUCH, PHARMD, FASHP, BCPS

Fred E. McWhorter Dean and Professor McWhorter School of Pharmacy Samford University Birmingham, Alabama



Any correspondence regarding this publication should be sent to the publisher, American Society of Health-System Pharmacists, 7272 Wisconsin Avenue, Bethesda, MD 20814, attention: Special Publishing.

The information presented herein reflects the opinions of the contributors and advisors. It should not be interpreted as an official policy of ASHP or as an endorsement of any product.

Because of ongoing research and improvements in technology, the information and its applications contained in this text are constantly evolving and are subject to the professional judgment and interpretation of the practitioner due to the uniqueness of a clinical situation. The editors and ASHP have made reasonable efforts to ensure the accuracy and appropriateness of the information presented in this document. However, any user of this information is advised that the editors and ASHP are not responsible for the continued currency of the information, for any errors or omissions, and/or for any consequences arising from the use of the information in the document in any and all practice settings. Any reader of this document is cautioned that ASHP makes no representation, guarantee, or warranty, express or implied, as to the accuracy and appropriateness of the information contained in this document and specifically disclaims any liability to any party for the accuracy and/or completeness of the material or for any damages arising out of the use or non-use of any of the information contained in this document.

Director, Special Publishing: Jack Bruggeman

Acquisitions Editor: Robin Coleman Editorial Project Manager: Ruth Bloom Production Manager: Amberly Hyden Cover and Page Design: Carol Barrer

© 2015, American Society of Health-System Pharmacists, Inc. All rights reserved.

No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, microfilming, and recording, or by any information storage and retrieval system, without written permission from the American Society of Health-System Pharmacists.

ASHP is a service mark of the American Society of Health-System Pharmacists, Inc.; registered in the U.S. Patent and Trademark Office.

ISBN: 978-1-58528-471-9

10987654321

#### CONTENTS

#### INTRODUCTION

- Table 1-1. Cholesterol concentrations
- Table 1-2. Epidemiologic data for dyslipidemia
- Figure 1-1. Approach to Management of Dyslipidemia
- Table 1-3. Comparison of ATP-III guidelines, ACC/AHA 2013 guidelines, and NLA recommendations

#### **PATHOPHYSIOLOGY**

#### Clinical Presentation, Diagnosis, and Disease Classification

- Table 1-4. Major risk factors for ASCVD
- Table 1-5. Criteria for classification of ASCVD
- Table 1-6. National Lipid Association criteria for ASCVD risk assessment, treatment goals for atherogenic cholesterol, and levels to consider drug therapy

#### **GENERAL TREATMENT PRINCIPLES**

Table 1-7. Components of dietary recommendations

#### American College of Cardiology/American Heart Association Guidelines

Table 1-8. Statin intensity

#### **National Lipid Association Recommendations**

Table 1-9. Risk indicators (other than major ASCVD risk factors) that might be considered for risk refinement

Table 1-10. Drugs that can lead to or worsen dyslipidemias

#### **PHARMACOTHERAPY**

**Statins** 

**Cholesterol Absorption Inhibitors** 

**Bile Acid Sequestrants** 

**Fibric Acid Derivatives** 

**Nicotinic Acid** 

**Omega-3 Fatty Acids** 

Table 1-11. Relative LDL-lowering efficacy of statin and statin-based therapies

#### **MONITORING**

Table 1-12. Lipid effects of nonstatin agents

Table 1-13. Clinical conditions that can worsen lipid profiles

#### **CLINICAL QUESTIONS AND CONTROVERSIES**

What Impact Do Statins Have on Cognitive Function?

Do Statins Cause or Worsen Type 2 Diabetes?

Are Statins Safe to Use in Patients with Chronic Liver Disease?

Adapted and updated from Barbara S. Wiggins, Dyslipidemia. In: Michael A. Crouch, Cardiovascular Pharmacotherapy: A Point-of-Care Guide, Bethesda, MD: American Society of Health-System Pharmacists © 2010.

What Drug-Drug Interactions Impact Statin Therapy?

Can Fibrates Be Safely Added to a Statin for the Management of Hypertriglyceridemia?

What Is the Best Way to Manage Suspected Statin-Induced Myalgia?

Should Co-enzyme Q10 Be Recommended for the Treatment and or Prevention of Statin-Associated Muscle Aches?

Which Risk Calculator Is Best?

What Is the Best Strategy to Manage Residual Cardiovascular Risk?

What LDL-C Level Is Too Low?

Should All Patients with an Acute Coronary Event or at High CHD Risk Be Started on High-Dose Statin Therapy?

What Is the Role of CRP in Determining CHD Risk and LDL-C Goals?

#### **FUTURE TREATMENTS**

#### **REFERENCES**

#### INTRODUCTION

Cholesterol is a naturally occurring (fat-like) substance that can be found throughout the body, and it serves as a building block for the production of cell membranes, various hormones, vitamin D, and bile acids. Cholesterol is derived from two sources: diet and synthesis by the liver. Transportation of cholesterol occurs via the blood stream in the form of lipoproteins (lipid plus protein). These lipoproteins are formed from specific apolipoproteins, which are synthesized in the liver and intestines. The amount of lipoproteins formed is determined by dietary fat intake, hormones, drugs, and alcohol consumption.<sup>1</sup>

There are six classes of apolipoproteins (four of which are discussed in this eReport) as well as several subclasses. The different lipoproteins vary in size and density with regard to fat and protein content. The major lipoproteins, in order of largest to smallest, include chylomicrons, very low density lipoproteins (VLDL), low density lipoproteins (LDL), and high density lipoproteins (HDL).

Chylomicrons are triglyceride-rich lipoproteins that are formed following the consumption of dietary fat. These lipoproteins assist in the transportation of fat from the intestines to the blood stream in the form of triglycerides. VLDL are also triglyceride rich and comprise approximately 10% to 15% of total serum cholesterol.<sup>1</sup>

VLDL, which transport 60% to 70% of total serum cholesterol, also serve as a precursor of low density lipoprotein cholesterol (LDL-C). LDL transports cholesterol to cells throughout the body and is considered the most atherogenic of the lipoproteins.

High density lipoprotein cholesterol (HDL-C) makes up approximately 20% to 30% of total serum cholesterol. It is considered the "good cholesterol." This lipoprotein is secreted from the liver, acquires cholesterol from the body's tissues, and transports it back to the liver. The high and optimal ranges for cholesterol concentrations are listed in Table 1-1. Atherogenic cholesterol levels (also known as non-HDL-C) can be estimated by subtracting HDL-C levels from the total measured amount of cholesterol. Non-HDL-C generally approximates the amount of cholesterol transported by lipoproteins containing apolipoprotein B (apoB) but does not require any additional testing beyond a standard lipid profile.

Dyslipidemia is defined as values for lipoproteins that are elevated (e.g., total cholesterol, LDL-C, and triglycerides) or reduced (HDL-C). Table 1-2 provides general epidemiologic data of dyslipidemia in the  $U.S.\ population.^2$