Updates to the 2016 NLA Annual Summary: Secrets Revealed!
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Disclosures

• Dr. Harold Bays is owns no pharmaceutical stocks or patents. In the past 12 months, Dr. Harold Bays’ research site has received research grants from Amarin, Amgen, Ardea, Arisaph, AstraZeneca, Bristol Meyers Squibb, Catabasis, Cymabay, Eisai, Elcelyx, Eli Lilly, Esperion, Ferrer/Chiltern, Gilead, GSK, Hanmi, Hisun, Hoffman LaRoche, Home Access, Janssen, Johnson and Johnson, Kowa, Merck, Necktar, Novartis, NovoNordisk, Omthera, Orexigen, Pfizer, Pronova, Regeneron, Sanofi, Takeda, and TIMI. In the past 12 months, Dr. Harold Bays has served as a consultant/advisor for Alnylam, Akcea, Amgen, AstraZeneca, Eli Lilly, Ionis (ISIS), Merck, Novartis, Pronova, Regeneron, Sanofi and Takeda. In the past 12 months, Dr. Harold Bays has served as a speaker for Amarin, Amgen, Astra Zeneca, Eisai, Regeneron, Sanofi and Takeda.
Introduction:

What is the NLA Annual Summary?
Original Contribution

National Lipid Association Annual Summary of Clinical Lipidology 2016

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2016 NLA Annual Summary Outline:

- Topics
- Reviewers
- Principles
- Adaptation to Information Age
- Practical navigation examples
- Integration of Social Media
- Planned 2017 updates
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Who were the reviewers of the 2016 NLA Annual Summary?
Clinical Lipidology: The Review Board was comprised of NLA members, NLA national officers, the Editor of the Journal of Clinical Lipidology, Guest Editor of this document, and other invited reviewers. The NLA Review Board was constituted to allow for a broad perspective and diversity regarding the science and clinical considerations in the evaluation and treatment of patients with dyslipidemia. The NLA Annual Summary of Clinical Lipidology Review Board was instructed to incorporate evidence-based medicine as well as expert opinion.

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II. NLA Recommendations for Patient-Centered Management of Dyslipidemia

Lipid evaluation and management principles

- Basic principles in the evaluation and management of dyslipidemia for the purpose of reducing atherosclerotic cardiovascular disease (ASCVD) risk include:
  - An elevated level of triglyceride cholesterol is reflective of an increase in circulating apolipoprotein B (apo B) containing lipoproteins, and is most often clinically assessed by measuring non high density lipoprotein cholesterol.

www.lipid.org
What are the principles of the NLA Annual Summary?
Principles

The 2016 NLA Annual Summary of Clinical Lipidology was founded on evidence-based medicine and is generally consistent with established national and international lipid guidelines. Where definitive evidence was lacking, the best available evidence was applied. This summary should not be interpreted as rules or directives with regard to the most appropriate care of any single patient with dyslipidemia, because no set of recommendations or guidelines can have 100% applicability to an individual patient. Thus, evaluation and treatment decisions should be based on patient-centered, individual circumstances. As such, this document should be used in conjunction with, and not a replacement for the preferences of patients with dyslipidemia and the judgment of their treating clinicians.
How has the NLA Annual Summary adapted to the Information Age?
Appendix A and B Table and Figure Hyperlink Format

Highlighted hyperlinks within the document lead to Appendix A and B. When viewed online, hyperlinks in Appendix A and B, as well as hyperlinks in the E link section lead to applicable publications, tables, figures, and charts. In an age of wide-scale availability of Internet access, computers, smartphones, and tablets, the intent is to provide a central directory of information applicable for both medical science, as well as for the day-to-day management of patients with dyslipidemia. Providing electronic links to tables, figures, charts, and publications allows for better maintaining a summary document format, easier access to more in-depth information, and greater comprehensiveness of material important in the evaluation and management of patients with dyslipidemia.
What are some practical examples of how the NLA Annual Summary can be navigated?
“How do I find a quick summary of the latest information on Familial Hypercholesterolemia?”
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PCSK9 activity via gain-of-function genetic variant, circulating LDL-C levels are increased.

• Example #4: Betasitostatemia is a rare inherited plant sterol storage disease that can phenotypically mimic FH.
  
  □ Clinical findings of tendon xanthomas and increased ASCVD risk may be out of proportion to the patient’s lipid profile, which may demonstrate modest to no increase in LDL-C levels.
  
  □ Betasitostatemia is an autosomal recessive condition that occurs as a result of mutations in adenosine triphosphate binding cassette transporters (ABC) G5 or ABCG5, which are sterol transporters that facilitate plant sterols and cholesterol efflux from intestinal and hepatic cells into the intestinal and biliary lumen.

□ A lack of gastrointestinal plant sterol secretion back into the gastrointestinal lumen increases circulating physiosterol levels.

□ The diagnosis of betasitostatemia is typically made by measuring plant sterol levels, not by genotyping of ABCG5/G8.

□ Betasitostatemia is an example of a genetic condition that requires an accurate diagnosis because ezetimibe is the only lipid-altering drug with a specific Food and Drug Administration (FDA)-indicated use for treating patients with betasitostatemia.

□ Ezetimibe impairs intestinal plant sterol (and cholesterol) absorption and therefore reduces circulating plant sterol levels.

□ Examples of other genetic abnormalities related to dyslipidemia include disorders of lipoprotein (a), apolipoprotein E, apo CII, Apo-AV, and ABC transporter (ie, Tangier disease). Future genotyping may help identify mutations in these lipid-related parameters, and may also help help identify patients most likely to have adverse experiences with certain medications, such as myopathy to statins.

□ Some genetic contributors to dyslipidemia involve a combination of gene variants. For example, many genetic causes of moderate hypertriglyceridemia are likely polygenic, in nature, requiring a secondary factor for expression.13

EVALUATION AND MANAGEMENT OF FAMILIAL HYPERCHOLESTEROLEMIA14-17

Genetics

• The FHs represent a group of genetic defects that result in an extreme elevation of LDL-C levels starting in utero, and increased risk of premature atherosclerotic CHD, as much as 20-fold in untreated FH patients.

• Although homozygous FH occurs in approximately 1 out of every 250,000 to 1 million individuals, heterozygous FH is among the most common congenital metabolic disorders, occurring in approximately 1,200 to 1,500 individuals, with an increased rate (1:100) among those of Lebanese, French Canadian, Ashkenazi Jewish, and several South African backgrounds resulting from founder effects.

• FH is most commonly (> 90%) an autosomal dominant lack of LDL receptor activity, usually from LDL receptor mutation (with more than 1200 described mutations).

• Less commonly, FH may be due to an apo B-100 gene mutation (eg, Arg3500Gln), which accounts for about 5% of genetically identified FH cases, or PCSK9 gain-of-function mutations (overexpression), leading to increased degradation of the LDL receptor and accounting for about 1% of cases of FH.20

□ Other potential mechanisms may contribute to the phenotypic presentation of FH.

□ Elevation of serum cholesterol and triglycerides can be expected in patients with FH.

□ Patients with homozygous FH (the same genetic defect inherited from each parent) or compound heterozygous FH (different genetic defects inherited from both parents) typically have LDL-C levels >500 mg/dL.

□ Patients with heterozygous FH (single genetic defect inherited from either parent) typically have LDL-C levels >160 mg/dL in pediatric patients and >190 mg/dL in adult patients.

□ Patients with FH may occasionally have elevated triglyceride levels; thus, high triglyceride levels do not exclude the diagnosis of FH.

Diagnosis13,20-22

• Several groups have offered diagnostic criteria for FH, including Simon Broome, Dutch Lipid Clinic Network, and MedPed: Dutch Lipid Clinic criteria apply to adults; Simon Broome and MedPed can also apply to children.

□ Diagnostic criteria for FH depend upon measured findings of very high LDL-C levels as well as family history of marked elevated LDL-C levels and early-onset ASCVD. Given this clinical presentation, tendon xanthomas are pathognomonic for FH, with genetic testing often, but not always, confirmatory.

Screening and genetic testing for familial hypercholesterolemia13,14

• Cascade (family) screening for FH is recommended in individuals and families with very high LDL-C levels.

• Genetic testing is generally not required for diagnosis or clinical management of FH; however, a characteristic clinical presentation, coupled with DNA testing by a reliable testing laboratory that confirms an applicable mutation in an individual, can provide unequivocal diagnosis.

□ The possibility of FH is not excluded by negative DNA testing because genetic testing fails to reveal a specified mutation in approximately 30% of clinically defined FH patients.

□ Cascade family screening for FH is recommended in individuals and families with very high LDL-C levels.

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Treatment priorities

• Maximize reduction in other ASCVD risk factors.

□ Maximize nutrition and physical activity interventions.

□ Lower LDL-C levels by at least 50% or more, to <100 mg/dL, if feasible.

□ Cascade testing of first-degree relatives should be offered to all individuals with FH.

□ The 10-year CHD risk in the FH patient is not adequately predicted by any conventional risk assessment tools; assessment of 10-year risk is not recommended.

Lipid-altering pharmacotherapies for FH: general principles

• High-intensity statins are the pharmacotherapy of first choice for patients with FH, but should be avoided in women who are breastfeeding, who may potentially become pregnant, or who are pregnant because statins have not been adequately studied in pregnant women.

□ Other lipid-altering agents that may be useful in combination with statins, or in combination with each other for patients who cannot take statins include ezetimibe, bile acid sequestrants, PCSK9 inhibitors, and niacin.

□ In addition to high-intensity statins, other lipid-altering pharmacotherapies have an approved indication to treat patients with homozygous FH.

□ Mipomersen is an antisense oligonucleotide that targets the messenger RNA for apo B.

□ Mipomersen is an antisense inhibitor of apo B synthesis that when administered in combination with maximum tolerated doses of lipid-lowering therapy can reduce LDL-C levels by an additional 25% in homozygous FH patients.

□ Mipomersen is an inactivating product that may cause injection site reactions.

□ Mipomersen may increase hepatic fat.

□ Mipomersen may increase liver transaminase levels; however, clinical trial data have not reported permanent liver failure.

□ Lomitapide is a microsomal triglyceride transfer protein inhibitor, which impairs VLDL secretion and reduces circulating apo B-containing lipoproteins.

□ Lomitapide may reduce LDL-C levels by up to 50% in patients with homozygous FH on maximum tolerated lipid-lowering therapy and LDL apheresis.

□ Common adverse experiences with lomitapide include fat malabsorption, diarrhea, increased liver fat, and elevated liver transaminases.

□ Substantially due to the alterations in liver transaminases and increase in hepatic fat, mipomersen and lomitapide are available through Risk Evaluation and Mitigation Strategy programs.

Evolocumab is a human monoclonal PCSK9 inhibitor that among its indications is treatment for homozygous familial hypercholesterolemia. Compared with placebo in patients with homozygous hypercholesterolemia, evolocumab significantly reduced LDL cholesterol at 12 weeks by 31%, suggesting residual LDL receptor activity in a subset of patients with homozygous FH.37

□ Other treatment options for lowering cholesterol in patients with FH include LDL apheresis, and in the most severe and resistant cases, portacaval anastomosis and liver transplantation.

IV. SECONDARY CAUSES OF DYSLIPIDEMIA1,24

• Beyond genetic considerations, dyslipidemia can also be due to secondary causes.

□ A “two-hit phenomenon”24 is commonly encountered in the clinical evaluation and management of patients with primary hyperlipidemia (eg, the relatively common familial combined hyperlipidemia or familial hypertriacylglycerolemia; the more rare lipoprotein lipase deficiency; apo C-III deficiency, familial dysbetalipoproteinemia).

□ “First hit” = Genetic predisposition.

□ “Second hit” = Exacerbation by secondary factors that worsen lipid levels, often resulting in profound hyperlipidemia.

□ This “second hit” can be the result of underlying disorders of metabolism or disease (eg, untreated hypothyroidism, inadequately controlled diabetes mellitus or from drugs that unproportionally alter lipid metabolism.

□ Secondary causes of hyperlipidemia are listed in the Appendix, and many elevate triglycerides either alone, or elevate both triglycerides and LDL cholesterol.

□ Secondary causes of hypertriacylglycerolemia are often associated with decreases in HDL-C levels. However, alcohol consumption, oral estrogen, and bile acid sequestrants are examples of agents that can increase both triglyceride and HDL-C levels.

V. ADDITIONAL LIPID PARAMETERS25-31

High-density lipoprotein cholesterol

□ Epidemiologically, HDL-C has an inverse relationship with ASCVD risk, irrespective of sex, race, or ethnicity.

□ Increased HDL-C levels are often associated with decreased risk of ASCVD.

□ Decreased HDL-C levels are often associated with increased ASCVD risk.

□ HDL-C may not be causally related to atherosclerosis and cardiovascular events; however, it is a biomarker of ASCVD risk.

□ HDL particles include many proteins and lipids that influence the function of HDL, and may provide atheroprotection via favorable effects upon atherosclerotic
The appearance of the serum can provide clues to diagnosis of genetic dyslipidemia.
## APPENDIX A: National Lipid Association (NLA) Annual Summary of Clinical Lipidology 2016: Tables, Figures, Charts, and Hyperlinks

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<td>Table 5. MEDPED diagnostic criteria for heterozygous familial hypercholesterolemia</td>
<td>*</td>
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<tr>
<td></td>
<td>Table Summary Recommendations from the National Lipid Association Expert Panel on Familial Hypercholesterolemia</td>
<td>16</td>
</tr>
</tbody>
</table>
Hypertriglyceridemia: its etiology, effects and treatment

George Yuan, Khalid Z. Al-Shali, Robert A. Hegele

Fig. 1. Clinical manifestations of primary hypertriglyceridemia. A: Lipidic cutaneous xanthomas (thick on the patient's knee) are filled with foam cells that appear as yellow or orange papules or plaques. Most often associated with markedly elevated plasma triglyceride concentrations. In patients with familial chylomicronemia (hyperchylomiconemia type III), they usually occur on the skin of the trunk, buttocks, or extremities. B: Lipemic plasma. Whole blood has been allowed to stand at 4°C overnight. The sample on the left comes from a patient whose fasting total cholesterol concentration was 142 mg/dL and triglyceride concentration was 418 mg/dL. The sample on the right comes from a normolipemic subject. C: Lipemia retinalis. A milky appearance of the retinal vessels and pink retina can be seen when plasma triglyceride concentration exceeds 35 mg/dL. D: Tuberous xanthomas, filled with foam cells, appear as reddish or orange, often shiny nodules, up to 3 cm in diameter. They are usually inoperable and are not removable. In patients with familial dysbetalipoproteinemia (hyperbetalipoproteinemia type V), they usually appear on extensor surfaces, these are on a patient's elbows. E: Palmar crease xanthomas are filled with foam cells and appear as yellowish deposits within palmar creases. These skin lesions are pathognomonic for familial dysbetalipoproteinemia (hyperbetalipoproteinemia type V).
Diagnosis\textsuperscript{18,20–22}

- Several groups have offered diagnostic criteria for FH, including Simon Broome, Dutch Lipid Clinic Network, and MedPed: Dutch Lipid Clinic criteria apply to adults; Simon Broome and MEDPED can also apply to children.
| Evaluation and Management of Familial Hypercholesterolemia | Table 3: Simon Broome diagnostic criteria for familial hypercholesterolemia | * |
| | Table 4. Dutch Lipid Clinic Network diagnostic criteria for familial hypercholesterolemia | * |
| | Table 5. MEDPED diagnostic criteria for heterozygous familial hypercholesterolemia | * |
| | Table Summary Recommendations from the National Lipid Association Expert Panel on Familial Hypercholesterolemia | 16 |
| Tabla 1: Simon Brown diagnostic criteria for Familial Hypercholesterolemia*

**Definite Familial Hypercholesterolemia:**

Required laboratory = high cholesterol levels:

- Adult = Total cholesterol levels > 290 mg/dL (7.5 mmol/L) or LDL-C > 150 mg/dL (4.0 mmol/L)
- Child less than 16 years of age = Total cholesterol levels > 260 mg/dL (6.7 mmol/L) or LDL-C > 155 mg/dL (4.0 mmol/L)

**Plus at least one of the two:**

1. Plus physical finding - tendon xanthomas, or tendon xanthomas in first or second degree relative
2. DNA-based evidence of an LDL-receptor mutation, familial defective apo B:100, or a PCSK9 mutation.

**Possible Familial Hypercholesterolemia**

Laboratory = high cholesterol levels:

- Adult = Total cholesterol levels > 290 mg/dL (7.5 mmol/L) or LDL-C > 150 mg/dL (4.0 mmol/L)
- Child less than 16 years of age = Total cholesterol levels > 260 mg/dL (6.7 mmol/L) or LDL-C > 155 mg/dL (4.0 mmol/L)

**Plus at least one of the two:**

1. Family history of at least one of the following.
   1. Family history of myocardial infarction at:
      1. Age 60 years or younger in first degree relative
      2. Age 50 years or younger in second-degree relative
2. Family history of elevated total cholesterol
   1. Greater than 290 mg/dL (7.5 mmol/L) in adult first- or second-degree relative
   2. Greater than 260 mg/dL (6.7 mmol/L) in child, brother or sister aged younger than 16 years.

---


www.lipid.org
“How do I find a quick summary of the latest on lipid-altering drugs in development?”
## XVII. INVESTIGATIONAL LIPID-ALTERING AGENTS IN DEVELOPMENT 2016 *(Table 1)*

<table>
<thead>
<tr>
<th>Class of agent and mechanism of action</th>
<th>Name</th>
<th>Manufacturer</th>
<th>Sample references or Clinical Trials.gov Identifiers</th>
<th>Sentinel, reported safety/ tolerability findings</th>
<th>Sentinel lipid effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors</td>
<td>Bococizumab</td>
<td>Pfizer (RN316)</td>
<td>195</td>
<td>Rare injection site reactions, with most cases being mild</td>
<td>&gt;50% reduction in LDL-C and non-HDL-C levels</td>
</tr>
<tr>
<td></td>
<td>ALN-PCS</td>
<td>Alnylam and the Medicines Company</td>
<td>180,196</td>
<td>Mild localized injection site reactions</td>
<td>Mean LDL cholesterol reduction 64%, with some degree of LDL cholesterol lowering maintained over 140 days, potentially supportive of a once-quarterly and possibly biannual subcutaneous administration</td>
</tr>
</tbody>
</table>

*(continued on next page)*
“How do I quickly find hyperlinks to prescribing information for lipid-altering drugs?”
### APPENDIX A  (continued)

<table>
<thead>
<tr>
<th>Section of this NLA Annual Summary</th>
<th>Title and links to applicable tables/figures/charts</th>
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<td><strong>Fluvastatin</strong>: <a href="https://www.pharma.us.novartis.com/product/pi/pdf/Lescol.pdf">https://www.pharma.us.novartis.com/product/pi/pdf/Lescol.pdf</a></td>
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<td><strong>Lovastatin</strong>: <a href="http://www.merck.com/product/usa/pi_circulars/m/mevacor/mevacor_pi.pdf">http://www.merck.com/product/usa/pi_circulars/m/mevacor/mevacor_pi.pdf</a></td>
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<td><strong>Pravastatin</strong>: <a href="http://packageinserts.bms.com/pi/pi_pravachol.pdf">http://packageinserts.bms.com/pi/pi_pravachol.pdf</a></td>
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<td><strong>Rosuvastatin</strong>: <a href="http://www1.astrazeneca-us.com/pi/crestor.pdf">http://www1.astrazeneca-us.com/pi/crestor.pdf</a></td>
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<td><strong>Omega-3-acid ethyl esters (EPA and DHA)</strong>: <a href="https://www.gsksource.com/gskpnm/htdocs/documents/LOVAZA-PI-PIL.PDF">https://www.gsksource.com/gskpnm/htdocs/documents/LOVAZA-PI-PIL.PDF</a></td>
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<td></td>
<td><strong>Icosapent ethyl (EPA only)</strong>: <a href="http://www.vascepa.com/full-prescribing-information.pdf">http://www.vascepa.com/full-prescribing-information.pdf</a></td>
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<td><strong>Omega-3-carboxylic acids (EPA and DHA free fatty acid formulation)</strong>: <a href="http://www1.astrazeneca-us.com/pi/epanova.pdf">http://www1.astrazeneca-us.com/pi/epanova.pdf</a></td>
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<td><strong>Alirocumab</strong>: <a href="http://products.sanofi.us/praluent/praluent.pdf">http://products.sanofi.us/praluent/praluent.pdf</a></td>
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<td><strong>Evolocumab</strong>: <a href="http://pi.amgen.com/united_states/repatha/repatha_pi_hcp_english.pdf">http://pi.amgen.com/united_states/repatha/repatha_pi_hcp_english.pdf</a></td>
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<td><strong>Fenofibric acid</strong>: <a href="http://www.rxabbvie.com/pdf/trilipix_pi.pdf">http://www.rxabbvie.com/pdf/trilipix_pi.pdf</a></td>
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<td><strong>Lomitapide</strong>: <a href="http://www.juxtapidremssprogram.com/_pdf/012187_JuxtapidPI_8.5x11_FIN.PDF">http://www.juxtapidremssprogram.com/_pdf/012187_JuxtapidPI_8.5x11_FIN.PDF</a></td>
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<td><strong>Mipomersen</strong>: <a href="http://www.kynamro.com/~media/Kynamro/Files/KYNAMRO_PI.pdf">http://www.kynamro.com/~media/Kynamro/Files/KYNAMRO_PI.pdf</a></td>
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“How do I quickly find hyperlinks to Tables and Charts of Parts 1 & 2 of the NLA Recommendations?”
## APPENDIX A: National Lipid Association (NLA) Annual Summary of Clinical Lipidology 2016: Tables, Figures, Charts, and Hyperlinks

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<tr>
<th>Section of this NLA Annual Summary</th>
<th>Title and links to applicable tables/figures/charts</th>
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<td>NLA Executive Summary</td>
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<td><a href="#">Table 2. Treatment goals for non-HDL-C, LDL-C, and Apo B in mg/dL</a></td>
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<td><a href="#">Table 3. Criteria for ASCVD risk assessment, treatment goals for atherogenic cholesterol, and levels at which to consider drug therapy</a></td>
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<td><a href="#">Table 7. Major risk factors for ASCVD</a></td>
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<td><a href="#">Table 8. Criteria for classification of ASCVD</a></td>
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<td><a href="#">Table 9. High- or very high-risk patient groups</a></td>
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<td><a href="#">Table 10. Sequential steps in ASCVD risk assessment</a></td>
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<td><a href="#">Table 11. Risk indicators (other than major ASCVD risk factors) that might be considered for risk refinement</a></td>
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<td>Section of this NLA Annual Summary</td>
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<td>Table 14. Phases of drug interaction</td>
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<td>Table 15. Transporter classes</td>
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<td>Table 23. Interactions between antiretroviral therapy and statins (page S81)</td>
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<td><strong>Dyslipidemia in patients with inflammation</strong></td>
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</table>

NA, not applicable

*Online NLA Resource Center
“How do I quickly find hyperlinks to Worldwide Lipid Guidelines and Recommendations?”
Lipid Guidelines, Recommendations, and Position Statements

- National lipid association recommendations for patient-centered management of dyslipidemia: Part 1, full report.
- National lipid association recommendations for patient-centered management of dyslipidemia: Part 2
- Key Aspects of the NLA Recommendations for the Patient-Centered Management of Dyslipidemia
- A lipologist perspective of global lipid guidelines and recommendations, part 2: Lipid treatment goals
- An International Atherosclerosis Society position paper: global recommendations for the management of dyslipidemia: Executive Summary.
- 2012 Update of the Canadian Cardiovascular Society Guidelines for the Diagnosis and Treatment of Dyslipidemia for the Prevention of Cardiovascular Disease in the Adult
- ESC/EAS Guidelines for the management of dyslipidaemias: The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS)
- National Cholesterol Education Program Adult Treatment Panel III Guidelines: Implications of recent clinical trials
“How do I find hyperlinks to online ASCVD Risk Calculators?”
Atherosclerotic Cardiovascular Disease Risk Assessment Tools and Calculators

- American College of Cardiology/American Heart Association atherosclerotic cardiovascular disease Risk Estimator
- United States National Heart, Lung, and Blood Institute Framingham Risk Score
- Reynolds Risk Score
- QRISK Risk Calculator
- Lloyd-Jones Framingham Algorithm
- Systemic Coronary Risk Estimation (SCORE)
- Prospective Cardiovascular Munster Study (PROCAM) Risk Scores (Quick Check and Health Check)
- A lipidologist perspective on global lipid guidelines and recommendations, Part 1: Lipid treatment targets and risk assessment
## ASCVD Risk Estimator*

All fields are required to compute ASCVD risk.

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>Race</th>
<th>Systolic Blood Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>20-79</td>
<td>White</td>
<td>90-200</td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td>African American</td>
<td></td>
</tr>
<tr>
<td>HDL - Cholesterol (mg/dL)</td>
<td>Total Cholesterol (mg/dL)</td>
<td>Other</td>
<td></td>
</tr>
<tr>
<td>20-100</td>
<td>130-320</td>
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</tr>
<tr>
<td>Diabetes</td>
<td>Treatment for Hypertension</td>
<td>Smoker</td>
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</tr>
<tr>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>No</td>
<td>No</td>
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</tbody>
</table>

*Intended for use if there is not ASCVD and the LDL cholesterol is <190 mg/dL.

**Optimal risk factors include: Total cholesterol of 170 mg/dL, HDL cholesterol of 50 mg/dL, Systolic BP of 110 mm Hg, Not taking medications for hypertension, Not a diabetic, Not a smoker.
“How do I find hyperlinks to NLA Position Statements and Recommendations?”
E1 National Lipid Association Position Statements and Hyperlinks

- 2015 National Lipid Association recommendations for patient-centered management of dyslipidemia: Part 2: (Lifestyle therapies, groups with special considerations, older patients, patients with human immune deficiency, patients with inflammation, patients with residual risk, strategies to assist with adherence, team-based collaborative care).
- 2015 Lipids and bariatric procedures part 1 of 2: Scientific statement from the National Lipid Association, American Society for Metabolic and Bariatric Surgery, and Obesity Medicine Association: FULL REPORT
- 2014 Statin Safety Update.
- 2013 Obesity, adiposity, and dyslipidemia: A consensus statement from the NLA.
- 2011 Familial Hypercholesterolemia: Screening, Diagnosis and Management of Pediatric and Adult Patients.
- 2008 National Lipid Association Statement Regarding Reporting of Non-HDL on Standard Laboratory Reports.
“But does the NLA Annual Summary really take full advantage of the Information Age? What about social media?”
E2 Other National Lipid Association documents

Lipid Clinic and CMR operations manual/course

(https://www.lipid.org/education/courses/coding)

Coding and reimbursement

(https://www.lipid.org/practicetools/reimbursement)

E3 Links to:

Podcasts: https://www.lipid.org/aggregator/audio

Webcasts: https://www.lipid.org/aggregator/webcasts

Slide shows: https://www.lipid.org/aggregator/slideshows

Websites: https://www.lipid.org/aggregator/websites

Applications: https://www.lipid.org/aggregator/application

CME: https://www.lipid.org/aggregator/CME

Patient information: https://www.lipid.org/aggregator/patients
2016 NLA Annual Summary:

- Topics
- Reviewers
- Principles
- Adaptation to Information Age
- Practical navigation examples
- Integration of Social Media
- Planned 2017 updates
“But what have you done for me lately . . . what updates are planned for 2017?”
Accepted Manuscript

2016 ACC Expert Consensus Decision Pathway on the Role of Non-Statin Therapies for LDL-Cholesterol Lowering in the Management of Atherosclerotic Cardiovascular Disease Risk

Donald M. Lloyd-Jones, MD, FACC, Chair, Writing Committee, Pamela B. Morris, MD, FACC, Vice Chair, Writing Committee, Christie M. Ballantyne, MD, FACC, Writing Committee, Kim K. Bircher, PharmD, AACC, Writing Committee, David D. Daly, Jr., MD, Writing Committee, Sondra M. DePalma, MHS, PA-C, CLS AACC, Writing Committee, Margo B. Minissian, PhD, ACNP, AACC, Writing Committee, Carl E. Orringer, MD, FACC, FNLA, Writing Committee, Sidney C. Smith, Jr., MD, FACC, Writing Committee

PII: S0735-1097(16)32398-1
DOI: 10.1016/j.jacc.2016.03.519
Reference: JAC 22437

To appear in: Journal of the American College of Cardiology
Updates to the 2016 NLA Annual Summary: Secrets Revealed!