Clinical Utility of LDL Particle Number to Optimize Management of LDL-Related Cardiovascular Risk

William C. Cromwell, MD; Douglas W. Triffon, MD

PREFACE
Managing low-density lipoprotein (LDL) cholesterol is an integral part of clinical practice. Recently, recommendations have shifted from targeting specific LDL levels to emphasizing the use of therapies shown to reduce atherosclerotic cardiovascular disease (ASCVD) events. As a result, moderate- and high-dose statin therapy is now emphasized for use in several defined patient groups. What remains controversial is how physicians should evaluate individual LDL response to statin therapy and whether LDL-guided adjustments in treatment can lead to further reduction in ASCVD events.

This supplement reviews:
- Data demonstrating a more reliable measure of LDL quantity—LDL particle number (LDL-P)—that can identify statin-treated individuals with continued LDL-related ASCVD risk and guide therapy adjustment likely to result in additional reduction in ASCVD events
- Expert society recommendations endorsing LDL-P measures for management of high-risk populations
- Outcome data demonstrating attainment of low LDL-P vs low measure of cholesterol (LDL-C) in the management of high-risk populations resulting in a number needed to treat (NNT) of 23
- An integrated, step-wise model used to put these data and recommendations into practice.

LDL MANAGEMENT IN CLINICAL PRACTICE: CURRENT STATE OF AFFAIRS
The causal role of LDL particles in the development and progression of ASCVD is well known. LDL particles move into the arterial wall via a gradient-driven process—the greater the circulating concentration of LDL particles, the greater the rate of movement into the arterial wall. Once inside the intima, LDL particles that bind to arterial wall proteoglycans are retained, oxidized, and subsequently taken up by macrophages to form foam cells. The greater the circulating levels of LDL over time, the greater the acceleration of this process and the higher the risk for ASCVD events.

Due to its ubiquitous accessibility, LDL-C (the measurement of cholesterol carried in LDL particles) has
become the customary measure used to estimate LDL quantity in clinical practice. However, there are 2 widely available, cost-effective, FDA-cleared measures of LDL quantity that do not rely on cholesterol carried in LDL particles: (1) LDL-P by nuclear magnetic resonance (NMR)—a direct measurement of the LDL particle number (NMR LDL-P),\(^5\) and (2) apolipoprotein B (Apo B)—an estimate of the LDL-P.\(^6\) Neither method relies on the variable cholesterol content in LDL particles.

Similar to the accepted use of LDL-C measurement in clinical practice is the prescribed treatment of patients with HMG Co-A reductase inhibitor (statin) therapy and the resulting improvement in ASCVD events. As a consequence of decreased de novo cholesterol synthesis caused by statins, LDL receptors are up-regulated resulting in increased clearance of circulating LDL particles.\(^7\) A meta-analysis of statin intervention trials demonstrates, at a population level, the greater the LDL reduction, the greater the reduction in ASCVD risk among statin-treated groups.\(^8\)

Over the past 3 decades successive national and international guidelines have advocated strategies to lower LDL levels. Historically, the guiding principle adopted by groups such as the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) has been to link LDL-C goals with ASCVD risk; the higher the patient’s ASCVD risk, the lower the LDL-C goals advocated to mitigate that risk.\(^9,10\)

In 2013 the American College of Cardiology (ACC) and American Heart Association (AHA) jointly issued the \textit{ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Disease in Adults} which advocated a different approach to managing LDL-related ASCVD risk.\(^1\) In contrast to prior guidelines that focused on attaining discrete LDL targets, the ACC/AHA guideline focused on randomized controlled trial (RCT) data to determine treatment strategies most consistent with cardiovascular outcome improvement. From this perspective, initiation of moderate- or high-dose statin therapy was advocated for defined groups of patients that demonstrated significant outcome improvement following statin therapy.

Important limitations may exist for individual care if management is viewed as complete following the initiation of statin therapy. Although RCT data allow therapies to be prioritized by virtue of proven benefit observed in treated populations, individual response is variable. Some patients receiving statins experience fewer ASCVD events while others fail to benefit despite treatment. Optimizing individual care requires the ability to identify statin-treated patients who continue to harbor increased ASCVD risk, as well as identify the incremental reduction in ASCVD risk following adjustment of therapy.

The degree to which on-treatment LDL measures can guide adjustment in care leading to improved ASCVD outcomes has become controversial. Because RCTs were not designed to assess optimal LDL levels associated with ASCVD risk reduction, the 2013 ACC/AHA guideline made no recommendations regarding LDL levels as lipid-lowering treatment targets. Although silent on LDL targets, the ACC/AHA guideline did advise measuring on-treatment LDL values to assess adherence, judge individual response to therapy, and serve as part of a conversation between physician and patient regarding further adjustments in care, including statin therapy intensification or statin combination therapy.

In contrast to the ACC/AHA position, numerous guidelines and expert panel recommendations endorse various on-treatment LDL targets (LDL-C, non-high-density lipoprotein cholesterol [HDL-C], or LDL-P measures) to adjudicate individual response and guide therapy adjustment.\(^11-22\) We believe these varying recommendations represent a two-step approach to patient care. First, based on RCT data, physicians should use outcome-proven therapy in groups with established benefit. Second, LDL-P should be used to evaluate individual response to therapy, guide therapy adjustment, and optimize opportunities for outcome improvement.

**LDL MEASUREMENTS: LDL-C VERSUS LDL-P**

LDL-C has been used for decades to estimate circulating LDL concentration. However, the cholesterol content of LDL varies widely among individuals and is often dependent on existing metabolic conditions (eg, insulin resistance, metabolic syndrome, type 2 diabetes mellitus), as well as the presence of lipid-altering medications.\(^6,23-27\)

Due to varying amounts of cholesterol carried in LDL, frequent disagreement (discordance) is noted between measures of cholesterol (LDL-C) and particle number quantity (NMR LDL-P, Apo B).\(^23,24,28-30\) In the Quebec Cardiovascular Study—a community study of 2103 men ages 45 to 76 years without ischemic heart disease—51% of subjects showed discordance (> ± 10% difference) between Apo B and LDL-C.\(^29\) Similarly, in the Multi-Ethnic Study of Atherosclerosis (MESA), discordance (> ± 12% difference) between LDL-P and LDL-C was noted in 50% of 6814 healthy, ethnically diverse men and women ages 45 to 84 years not
on lipid-lowering medication. Additionally, split-sample measurements of LDL-C vs NMR LDL-P obtained from 2355 subjects with type 2 diabetes and LDL-C <100 mg/dl (<20th percentile value) showed only 25% of subjects had concordantly low LDL-P <1000 nmol/L (<20th percentile value).

**LET OUTCOMES BE OUR GUIDE: RELATIONSHIP OF LDL-P AND ASCVD EVENTS**

To determine the potential utility of alternate LDL measures (LDL-P, Apo B) in guiding management of CV risk, outcomes must be evaluated in 2 specific ways. First, differences in CV events associated with the traditional measure (LDL-C) and alternate measures (LDL-P, Apo B) must be determined when these measures are discordant. When traditional and alternate LDL measures are discordant, CV events track with measures of LDL-P rather than LDL-C. When LDL-C and LDL-P measures agree (are concordant), CV outcomes are similarly associated with each measure. Since populations are composed of patients for whom alternate LDL measures may or may not be discordant, it is now understood that discordance analysis is essential to assess meaningful outcome differences between alternate and traditional measures of an actionable risk factor.

Second, improvement in CV events should be evaluated in patients who are managed to similarly low values of the alternate measure (LDL-P) vs those of the traditional measure (LDL-C). Toth et al analyzed data from the HealthCore Integrated Research Database to assess the impact of attaining low LDL-P vs low LDL-C on incident CV events among individuals at high ASCVD risk (eg, established coronary heart disease, stroke, transient ischemic attack, peripheral arterial disease, diabetes mellitus). In response to more intensive therapy (eg, higher potency statin, greater use of statin combinations with ezetimibe, colesevelam, niacin), patients achieving LDL-P <1000 nmol/L (mean 860 nmol/L) during the course of their normal medical care experienced a significant 22% to 25% reduction in risk of CV events (eg, myocardial infarction, revascularization, angina, stroke) vs patients managed to LDL-C <100 mg/dL (mean 79 mg/dL) at 12, 24, and 36 months of follow up. Importantly, due to significant CVD event reduction at each time point, only 23 individuals needed to be treated to LDL-P <1000 nmol/L to prevent one CVD event at 36 months of follow up compared to patients attaining a mean LDL-C of 79 mg/dL (70% on statin therapy).

**MAKING SENSE OF DIFFERENT TREATMENT RECOMMENDATIONS: IS USE OF LDL-P CONSISTENT WITH CURRENT GUIDELINES?**

A variety of approaches are advocated by various guidelines and expert panels to evaluate individual response to therapy following initiation of statin therapy. Because the RCTs which served as a basis for the 2013 ACC/AHA cholesterol treatment guideline did not incorporate an assessment of optimal LDL-C levels associated with ASCVD risk reduction, the guideline made no recommendations regarding LDL-C targets for lipid-lowering therapy. Additionally, ACC/AHA resource limitations precluded the review of Apo B and other lipid or lipoprotein measures for guiding lipid therapy.

In contrast, the American Association of Clinical Endocrinologists (AACE), the National Lipid Association (NLA), the American Diabetes Association (ADA) in conjunction with the American College of Cardiology (ACC), and the American Association for Clinical Chemistry (AACC) have endorsed the use of LDL-P to evaluate individual LDL response and guide adjustment of therapy in high-risk patients with acceptable LDL-C and non-HDL-C values. A summary of expert society recommendations is shown in Table 1.

**PUTTING IT TOGETHER: INTEGRATION OF LDL-P IN CLINICAL PRACTICE**

In an effort to harmonize the aforementioned outcome data, guidelines, and expert recommendations, we developed an algorithm utilized at Scripps Green Hospital and the Lipoprotein and Metabolic Disorders Institute, respectively (Table 2).

**STEP 1: Assess ASCVD risk (10-year and lifetime)**

ASCVD risk status can be established by clinical history of ASCVD, presence of subclinical ASCVD, presence of comorbid conditions with high ASCVD risk (eg, stage III-IV chronic kidney disease, type 1 or type 2 diabetes with known ASCVD or the presence of >1 major risk factor, metabolic syndrome, organ transplant, coronary calcium score >300, abdominal aortic aneurysm), or use of validated 10-year or lifetime ASCVD risk calculators. Given these multiple points of reference, we assign a patient's risk category based on the highest risk level identified by any of these approaches (Table 2).
STEP 2: Institute appropriate course of therapy

After evaluating secondary causes of dyslipoproteinemia (e.g., hypothyroidism, diabetes mellitus, kidney disease, medications), initial therapy consists of therapeutic lifestyle management and treatment of comorbid conditions identified. As outlined in the 2013 ACC/AHA cholesterol treatment guideline, use of moderate- or high-dose statins is preferred as initial therapy for patients identified as candidates for therapy. These agents include:

- **Moderate Dose Statins**: atorvastatin 10 to 20 mg, fluvastatin 80 mg, lovastatin 40 mg, pitavastatin 2 to 4 mg, pravastatin 40 mg, rosuvastatin 5 to 10 mg, simvastatin 20 to 40 mg

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**TABLE 1. Recommendations for Using LDL-P Measures as Targets of Therapy**

<table>
<thead>
<tr>
<th>Population</th>
<th>Biomarker</th>
<th>Percentile equivalent cutpoint</th>
<th>5th</th>
<th>20th</th>
<th>50th</th>
<th>80th</th>
</tr>
</thead>
<tbody>
<tr>
<td>Framingham</td>
<td>LDL-C (mg/dL)</td>
<td>&lt;75</td>
<td>100</td>
<td>130</td>
<td>160</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Apo B (mg/dL)</td>
<td>&lt;60</td>
<td>80</td>
<td>100</td>
<td>120</td>
<td></td>
</tr>
<tr>
<td></td>
<td>NMR LDL-P (nmol/L)</td>
<td>&lt;850</td>
<td>1100</td>
<td>1400</td>
<td>1800</td>
<td></td>
</tr>
<tr>
<td>MESA</td>
<td>LDL-C (mg/dL)</td>
<td>&lt;70</td>
<td>95</td>
<td>120</td>
<td>140</td>
<td></td>
</tr>
<tr>
<td></td>
<td>NMR LDL-P (nmol/L)</td>
<td>&lt;800</td>
<td>1000</td>
<td>1300</td>
<td>1600</td>
<td></td>
</tr>
</tbody>
</table>

**Guideline/expert panel recommendation**

<table>
<thead>
<tr>
<th>Very high risk</th>
<th>High risk</th>
<th>Moderate risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>2008 ADA / ACCF Consensus Statement</td>
<td>Apo B &lt;80 mg/dL</td>
<td>Apo B &lt;90 mg/dL</td>
</tr>
<tr>
<td>2009 AACC Lipoproteins &amp; Vascular Diseases Division Working Group Recommendations</td>
<td>“Data are needed”</td>
<td>Apo B, LDL-P &lt;20th percentile</td>
</tr>
<tr>
<td>2011 NLA Expert Recommendations Inflammatory Biomarkers and Advanced Lipid Testing</td>
<td>Apo B &lt;70 mg/dL, or LDL-P &lt;20th percentile</td>
<td>Apo B, LDL-P &lt;20th percentile</td>
</tr>
<tr>
<td>2012 AACE Guidelines for Management of Dyslipidemia</td>
<td>Apo B &lt;80 mg/dL</td>
<td>Apo B &lt;90 mg/dL</td>
</tr>
<tr>
<td>2013 AACE Comprehensive Type 2 Diabetes Management Algorithm Consensus Statement</td>
<td>“Even more intensive therapy might be warranted”</td>
<td>Apo B &lt;80 mg/dL or LDL-P &lt;1000 nmol/L</td>
</tr>
<tr>
<td>2015 NLA Recommendations for Patient-Centered Management of Dyslipidemia</td>
<td>Apo B &lt;80 mg/dL</td>
<td>Apo B &lt;90 mg/dL</td>
</tr>
<tr>
<td>2015 AACE and American College of Endocrinology Clinical Practice Guidelines for Developing a Diabetes Mellitus Comprehensive Care Plan</td>
<td>N/A</td>
<td>Apo B &lt;80 mg/dL or LDL-P &lt;1000 nmol/L</td>
</tr>
<tr>
<td>2016 AACE and American College of Endocrinology Comprehensive Type 2 Diabetes Management Algorithm Consensus Statement</td>
<td>Apo B &lt;80 mg/dL or LDL-P &lt;1000 nmol/L</td>
<td>Apo B &lt;90 mg/dL or LDL-P &lt;1200 nmol/L</td>
</tr>
<tr>
<td>2016 NLA Annual Summary of Clinical Lipidology</td>
<td>Apo B &lt;80 mg/dL</td>
<td>Apo B &lt;90 mg/dL</td>
</tr>
</tbody>
</table>

**Abbreviations**: AACC, American Association of Clinical Chemistry; AACE, American Association of Clinical Endocrinologists; ACCF, American College of Cardiology Foundation; ADA, American Diabetes Association; Apo B, apolipoprotein B; LDL-C, low-density lipoprotein cholesterol; LDL-P, low-density lipoprotein particle number; MESA, Multi-Ethnic Study of Atherosclerosis; N/A, not applicable; NLA, National Lipid Association; NMR LDL-P, nuclear magnetic resonance low-density lipoprotein particle number.
Step 1: Assess ASCVD risk (10-year and lifetime)

High risk
- Presence of any one of the following:
  - Known clinical or subclinical ASCVD (coronary, carotid, or peripheral vascular disease)
  - LDL >95th percentile of the adult population (any one of the following):
    • LDL-C >190 mg/dL, non-HDL-C >220 mg/dL, NMR LDL-P >220 nmol/L, Apo B >140 mg/dL
  - Type I or type 2 diabetes mellitus and one of the following:
    • Known ASCVD or ≥1 risk factor; lifetime risk >39%
  - Primary prevention, age 40-79 years, with 10-year estimated risk ≥7.5% (2013 ACC/AHA risk calculator)
  - Primary prevention, age 20-59 years, with lifetime risk ≥39% (2013 ACC/AHA risk calculator)
  - Comorbid conditions conferring high ASCVD risk (any one of the following):
    • Familial hypercholesterolemia; Stage II-IV chronic kidney disease; organ transplant; abdominal aortic aneurysm; metabolic syndrome; coronary calcium score >300

Moderate risk
- Primary prevention, age 40-79 years, 10-year estimated risk 5% to 7.5% (2013 ACC/AHA risk calculator)

Step 2: Institute appropriate course of therapy

For all patients
- Evaluate for secondary etiologies of dyslipoproteinemia
- Institute therapeutic lifestyle and comorbidity management for all patients

If triglycerides <500 mg/dL:
- For high-risk ASCVD patients: consider using moderate or high-dose statin therapy
- For moderate-risk ASCVD patients: use clinical judgment to consider instituting statins
  - Moderate-dose statins: atorvastatin 10–20 mg, fluvastatin 80 mg, lovastatin 40 mg, pravastatin 20 mg, rosuvastatin 5–10 mg, simvastatin 20–40 mg
  - High-dose statins: atorvastatin 40–80 mg, rosuvastatin 20–40 mg
- If statin-intolerant, alternatives include: ezetimibe, resins, or niacin (based on clinical judgment)

If triglycerides >500 mg/dL:
- Use clinical judgment to consider marine omega-3, fibrate or niacin as initial therapy

Step 3: Assess LDL-P response with an outcome-proven measure of LDL-P 12 weeks after starting therapy

<table>
<thead>
<tr>
<th>ASCVD risk category</th>
<th>NMR LDL-P (nmol/L) target</th>
<th>Apo B (mg/dL) target</th>
</tr>
</thead>
<tbody>
<tr>
<td>High risk</td>
<td>&lt;1000 (may consider &lt;800 based on clinical judgment)</td>
<td>&lt;90 (may consider &lt;80 based on clinical judgment)</td>
</tr>
<tr>
<td></td>
<td>Alternatively, achieve at least a 50% reduction</td>
<td></td>
</tr>
<tr>
<td>Moderate risk</td>
<td>&lt;1300 (may consider &lt;1000 based on clinical judgment)</td>
<td>&lt;100 (may consider &lt;90 based on clinical judgment)</td>
</tr>
<tr>
<td></td>
<td>Alternatively, achieve a 40% to 50% LDL-P reduction</td>
<td></td>
</tr>
</tbody>
</table>

Note: Factors such as family history of premature CHD, high hs-CRP, high Lp(a), low HDL-P may prompt more aggressive LDL-P targets.

Step 4: Use clinical judgment and adjust therapy as indicated (eg, suboptimal LDL-P response)

For patients not at LDL-P goal, use clinical judgment and consider the following:
- Intensification of therapeutic lifestyle measures
- Intensification of statin therapy
- Use of statin combinations including statin + ezetimibe, statin + resin, statin + niacin, statin + EPA omega-3

Step 5: Assess response with an outcome-proven measure of LDL-P and modify therapy as needed to achieve LDL-P goal

- Recheck LDL-P approximately 12 weeks following each adjustment of therapy
- Once at goal, recheck LDL-P every 6–12 months
- Lipid consultation may be helpful in difficult-to-manage cases

Abbreviations: ACC, American College of Cardiology; AHA, American Heart Association; Apo B, apolipoprotein B; ASCVD, atherosclerotic cardiovascular disease; CHD, coronary heart disease; EPA, eicosapentaenoic acid; HDL-P, high-density lipoprotein particle number; hs-CRP, high sensitivity C-reactive protein; Lp(a), lipoprotein (a); LDL, low-density lipoprotein; LDL-C, low-density lipoprotein cholesterol; LDL-P, LDL particle number; NMR, nuclear magnetic resonance; non-HDLC, non-high-density lipoprotein cholesterol.
• **High Dose Statins:** atorvastatin 40 to 80 mg, rosuvastatin 20 to 40 mg.

If patients are statin-intolerant, alternative therapy may include ezetimibe, bile acid resins, or niacin, based on clinical judgment.\(^{18,20}\) For patients with triglyceride (TG) levels >500 mg/dL, clinical judgment should be used to consider marine omega-3, fibrates or niacin as initial therapy.\(^{20}\)

**STEP 3: Assess LDL-P response with an outcome-proven measure of LDL-P 12 weeks after starting therapy**

To evaluate individual response to statin therapy, a measure of LDL-P should be performed approximately 12 weeks after treatment initiation or adjustment.\(^{13-19}\) While both NMR LDL-P and Apo B are supported outcome-proven measures of LDL-P, it should be noted that better assay precision has been documented for NMR LDL-P than Apo B. Additionally, available data demonstrate NMR LDL-P is significantly more predictive of ASCVD risk than Apo B in instances where these 2 measures report differences in outcome association.\(^{44}\) Target values for LDL-P are listed for high- and moderate-risk patients.\(^{11,13-18,20-22}\) Based on clinical judgment, physicians may feel more intensive therapy is needed, especially for patients with progressive AS- CVD. Factors independently predictive of increased residual ASCVD risk despite statin therapy include high-sensitivity C-reactive protein (hs-CRP),\(^{55,46}\) lipoprotein (a) (Lp[a]),\(^{47,48}\) and NMR HDL particle number (HDL-P).\(^{49,50}\) When clinical or laboratory factors indicate increased residual risk, physicians should use clinical judgment in determining the value of more intensive LDL-P-lowering therapy.\(^{1,20,42}\)

**STEP 4: Use clinical judgment and adjust therapy as indicated (eg, suboptimal LDL-P response)**

If the patient is above target for LDL-P, use clinical judgment to modify therapy to further lower LDL-P. Options include increased efforts at therapeutic lifestyle changes (weight loss and dietary modification), statin therapy intensification, and/or addition of combination LDL-P-lowering agents to statins (eg, ezetimibe, colesvelem, niacin).\(^{13,18,19,35}\) The effect of various lipid-lowering agents on LDL-P is shown in **TABLE 3.**\(^{42}\) LDL particle excess is more frequently encountered among patients with type 2 diabetes and when one or more criteria for metabolic syndrome are present (eg, increased waist circumference, elevated blood sugar, elevated blood pressure, elevated TG, low HDL-C).\(^{13,26,27,51}\) Population data show the greater the number of criteria for metabolic syndrome noted, the greater the increase in LDL-P.\(^{52}\)

**STEP 5: Assess response with an outcome-proven measure of LDL-P and modify therapy as needed to achieve LDL-P goal**

If therapeutic adjustments are made, LDL-P response should be followed (tested) approximately 12 weeks after change of therapy and annually thereafter once the patient has achieved the desired LDL-P response.

**CONCLUSION**

In light of outcome data and recommendations discussed above, measures of LDL particle number (Apo B, NMR LDL-P) occupy a unique position among CV biomarkers. These measures serve as analytic improvements in quantifying LDL—a causal risk factor for development and promotion of atherosclerosis. This is particularly im-

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**TABLE 3. Effect of Lipid-Lowering Therapies on LDL-P**

<table>
<thead>
<tr>
<th>Lipid-altering agent</th>
<th>Reduction in LDL-P (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate- to high-dose statins (simvastatin, pitavastatin, atorvastatin, rosuvastatin)</td>
<td>35 – 55</td>
</tr>
<tr>
<td>Ezetimibe</td>
<td>15 – 25</td>
</tr>
<tr>
<td>Bile acid sequestrants</td>
<td>15 – 30</td>
</tr>
<tr>
<td>Niacin extended release</td>
<td>10 – 25</td>
</tr>
<tr>
<td>Statins + ezetimibe or bile acid sequestrants</td>
<td>50 – 70</td>
</tr>
<tr>
<td>Statins + niacin</td>
<td>50 – 70</td>
</tr>
<tr>
<td>Statins + ezetimibe or bile acid sequestrants + niacin</td>
<td>&gt;60</td>
</tr>
</tbody>
</table>

Adapted from: Cromwell W, Dayspring T. Lipid and Lipoprotein disorders: Current clinical solutions. Baltimore: International Guideline Center; 2012.\(^{42}\)
important for patients with type 2 diabetes mellitus, metabolic syndrome, CVD risk-equivalents, and those on statins: patients in whom there is frequent discordance between measures of cholesterol (LDL-C) and particle number (Apo B, NMR LDL-P). In a discordant setting, CV risk tracks with particle number (Apo B, NMR LDL-P) rather than cholesterol (LDL-C). Moreover, LDL-P is independently predictive of CV events following adjustment for confounding factors and allows clinicians to better judge response to statin therapy. The impact of these factors is evident in data that demonstrated an NNT of 23 for high-risk individuals managed to low LDL-P as part of their usual care vs those managed to similarly low LDL-C on statin therapy. The algorithm previously outlined can assist physicians in using LDL-P measures to identify high-risk patients with persistently high LDL in the presence of acceptable levels of LDL-C or non-HDL-C and adjust therapy to achieve LDL-P levels likely to result in further ASCVD risk reduction.

REFERENCES


