Also in this issue:

Why Advocacy is Important to a Medical Specialty Organization
Beyond the Guidelines: Application of Trial Data to Individual Patients by Focusing on Explanatory vs Pragmatic Trials

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- New Advances in theTreatment of Familial Hypercholesterolemia
- Debates in Lifestyle Therapy
- Keeping Up to Date in Clinical Lipidology
- Application of Innovative Diagnostic Techniques to the Practice of Clinical Lipidology
- Update on Lipid Management in Special Populations

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The National Lipid Association will be providing an app with onsite updates and educational content for attendees. Please check the website for information coming soon about how to download the app in preparation for the meeting. Make sure to bring your device because we will not be providing a printed syllabus. The app is compatible with laptops, tablets, and mobile devices.
How many of you have taken the time to read, from front to back, the recent National Lipid Association (NLA) Recommendations for Patient-Centered Management of Dyslipidemia, Parts 1 and 2? Don’t be embarrassed, because I can’t see how few of you raised your hands. I had the good fortune to co-author this document with a host of outstanding academic leaders in the field of clinical lipidology. As I reflect on the creation of these documents and think of the many hours that we all spent in discussing, debating, writing, and reviewing our sections, I feel proud of the end product of our collaboration.

Collaboration is the essence of all successful organizations. The success of the NLA depends on this process. Our administrative staff supports our activities by actively seeking our input about the goals that we hope to achieve, and by providing the tools that we need to move forward our agenda. The NLA Executive Committee and the Board of Directors work to establish a national agenda, but our organization is not truly successful until we understand and act upon the issues that affect our members at the regional and local level. Active participation in committee work and a commitment to communicate with our regional officers are the best mechanisms to facilitate the accomplishment of these goals.

Despite some philosophical differences in our approaches to lipid management for the prevention of ASCVD, the NLA and the American College of Cardiology (ACC) are actively working together to enhance collaboration in lipid management recommendations. Dr. Alan Brown and I were asked to serve as NLA representatives at the ACC-sponsored LDL Think Tank Meeting in Washington, D.C. Dr. Pamela Morris, who is the co-chair of the ACC Prevention Council and secretary of the SELA chapter, played a major role in organizing this conference. Following the conference, I was asked to serve as a representative of the NLA to participate in the ACC Expert Consensus Panel Lipid Management Writing Group, which is creating a document that will be published in the Journal of the American College of Cardiology in Spring 2016 to address the role of lipid therapy beyond statins in a host of clinical situations confronted by providers in their daily practice. Dr. Morris and I will be participating as panel members in a forum at the ACC’s Annual Scientific Session in April 2016 to discuss this important topic. I was also invited to provide an overview of the NLA Recommendations Part 1 and 2 documents on ACC.org, and this article appeared on Dec. 13, 2015. It is available using the link http://www.acc.org/latest-in-cardiology/articles/2015/12/11/13/50/key-aspects-of-the-nla-recommendations-for-the-patient-centered-management-of-dyslipidemia.

No successful organization operates in a vacuum. The future success of the NLA depends upon our ability to develop, cultivate, and maintain collaboration among our members and with other organizations who have similar interests and goals. I am proud of the steps that that the NLA has taken to meet this important objective.
The National Lipid Association is the premier organization dedicated to advancing our knowledge about the optimal approach to diagnosing and treating patients with lipid disorders. Any success of the organization is directly related to the diverse nature of our members. The NLA membership crosses multiple medical disciplines and specialties in a manner that is totally unique. The unifying theme of the organization is that we operate best “as a team.” This mutual respect among the membership is the single greatest source of our influence and our strength. It is our responsibility to assure that we advocate and approach the best practice model to provide care for our patients’ lipid disorders. It is our obligation to communicate the most accurate level of information — in a timely manner — to our members as well as the many non-member practitioners who look to us for direction.

Recently, as an American College of Cardiology (ACC) representative, I had an opportunity to review the indications for genetic testing for clopidogrel resistance. This is a dilemma facing interventional cardiologists every day because of a wide variation in patient responses to this agent (used to maintain cardiac stent patency). Genetic variations have documented that a patient’s likelihood of a failure of therapeutic response (non-responders) varies between 3 to 4 percent (Caucasian), 12 to 14 percent (African), and almost 40 percent (South Asian Indians). Given this widespread response, it seemed reasonable to explore the possibly of recommending testing to identify clopidogrel responders. The committee was limited in our decision by four benchmarks, which we were able to utilize to make this decision.

1.) An FDA-approved test is readily available.

2.) The laboratory test would be able to differentiate responders from non-responders.

3.) The cost would be reasonable.

4.) When practitioners utilized the test there would be a favorable improvement in the population of patients that we studied compared to a population that did not have access to the test.

The first three benchmarks were relatively easy to attain. It was the fourth benchmark that proved difficult (principally because of a lack of controlled studies). Sadly, because of the fourth benchmark, we were not able to advocate the widespread use of this genetic test as a guideline. This project, I believe, provides the NLA with an opportunity.

Over the course of the last several years, there has been a plethora of guidelines promoted to help us deal with patients’ cholesterol problems. Sadly, only a small
minority of the patients that we see in the office every day would qualify for inclusion in the very studies these guidelines are based. Translating an approach that applies to a few patients (those in clinical trials) into the many (specifically the one patient in your office that you are treating that day) is a challenge encountered by every practitioner on a daily basis. The NLA has an opportunity to serve as a “clinical reservoir” to provide accurate, unbiased, and clinically relevant information. This source of guidance would allow the NLA to maintain the highest standards in clinical lipidology. The NLA’s goal must always remain to maximize opportunity in order to make the best clinical decisions for the patient. It is in this context that I would advocate we consider promoting “guidance lines.” These would be in addition to our current guidelines and, ideally, would be utilized as a “team” approach. The purpose of this approach would not be to replace guidelines, but to partner with them in order to maximize our chances for success, especially in those patients we may have a hard time fitting into the guideline model.

We, as the experts in the field of clinical lipidology, have the ability to provide direction not only for our patients, but also for all practitioners. In addition to the guidelines, the NLA should consider guidance lines to be based on best practice models. We may consider the following points to start the discussion regarding just what a guidance line actually is. Patterned after the previously discussed clopidigrel, the NLA may want to consider the following inclusion points (the four “A’s”):

1.) FDA approved therapy (Available)
2.) Reasonable cost (Affordable)
3.) Capable of identifying non-responders (Accurate)
4.) Our ability to prove that the actions what we are recommending do actually improve our patients’ lives and reduce clinical events (Advantageous)

“We, as the experts in the field of clinical lipidology, have the ability to provide direction not only for our patients, but also for all practitioners.”

Through its membership, the NLA is in an ideal position to provide our patient contacts to populate such a database. This would allow us to create patient registries and would give us the opportunity to partner with our preventative colleagues such as the American College of Cardiology, Preventive Cardiology Nursing Association, or Million Hearts®: Cardiovascular Disease Risk Reduction Model. I can envision the organization playing a vital role in this effort to advance lipid therapy. Many of the questions that our clinical practice poses to us on a daily basis may never be answered by double blind mega trials, but I believe that a small dedicated group of individuals such as the NLA could provide valuable information that will help clinicians help patients. The NLA lipid registry would be able to advance our knowledge in so many areas. The NLA’s determination to commit to such a project has the ability to transform a “could” proposal into “we did” accomplishment. I envision that successful completion of this task may prove to be the NLA’s single greatest achievement and our proudest legacy.

The NLA’s success should be judged on the membership’s ability to favorably advance knowledge, and widely disseminate that information. Our sole focus remains to improve patient chances of survival and lessens their chances of suffering any cardiovascular event. The keystone component in order to reach this goal is that we must consider adding lipid registries to complement the evidence derived from the double blind placebo controlled mega trials. Our success in this endeavor will not only improve cardiovascular care for our patients but also fulfill Ben Franklin’s challenge of “do things worth the writing.”
Letter From the Lipid Spin Editor:
How I Learned to Stop Worrying and Learned to Love the Guidelines

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I've been through many stages with the guidelines. I really think I love them now. Let me tell you why.

I began my internal medicine residency in 1994, and the first time I ever heard that there was such a thing as guidelines, was at residents' clinic in my first year where I was treating a man following hospitalization for non-cardiac chest pain. I asked my attending to explain how a doctor decides when to use cholesterol medicine. We had results from 4S and WOSCOPS, but statins were not nearly as widely used at that time. I knew they were for high cholesterol, but I didn't know how high was high enough to prescribe statins. I feel pretty certain that he did not know the answer, but he did know that there were guidelines and suggested I look them up on my own. This was not a strong introduction to guideline directed care or to clinical lipidology.

By the time I finished my residency, I was well trained in the value and rigor of evidence-based medicine (EBM) and knew that there was strong evidence for the use of pravastatin and simvastatin.

Since atorvastatin had not completed any major outcomes studies by the time I started my career in primary care in 1997, it was several years before I made this more potent statin part of my regular prescribing. Lower is better was not yet a mantra and I can't say that the guidelines were burned into my brain entirely.

In 2001, already a budding lipidologist, I attended a lecture at a local hospital that was titled “How not to get sued” or something like that. The lecturer was an MD and a JD, but practicing mostly as an attorney. He made it clear that the best approach when caring for patients is to keep open lines of communication, to be available and honest, and express caring. He also added that there are no absolute “rules” in medicine — except for the National Cholesterol Education Program (NCEP) Guidelines. He taught us that every individual needs to be addressed individually, and algorithms should never take the place of clinical judgment.

So, in that short period of time, I went from a vague sense that there were guidelines, to they are the “law of the land.”

“At some point, it’s not enough to just be right, it’s critical to promote the value of the message.”

Discuss this article at www.lipid.org/lipidspin
From 2001 (ATP3) to 2013 (American College of Cardiology/American Heart Association Guidelines), I think we all got used to incorporating guidelines into our regular practice. EBM was alive and well. The ATP3 had gotten an early makeover in 2004, but it was long overdue for an update. And I had matured into my new role as a clinical lipidologist.

Budgetary changes and the Institute of Medicine (IOM) mandate changed the process of writing guidelines a bit. The IOM approach meant to maximize strength of recommendations. Under the ACC/AHA banner now, the National Guidelines were based exclusively on outcome based randomized placebo controlled clinical trials (RPCTs) and meta-analyses of the same. But our colleagues on the Guidelines Committee stated very clearly that RPCTs do not answer every clinical question and clinicians should think of this as the foundation of care or the minimum standard. Ultimately, the clinician must use all of the science and improvise strategy that works for the patient, while the guidelines remain the default starting point for the discussion with the patient. In other words, consult your local lipidologist for guidance.

The foundation of recommendations based on RPCTs and meta-analyses coupled with the suggested flexibility in the patient-clinician encounter were somehow lost in the discussion to follow these last two years. Why did this message get lost? How is it that clinicians concluded that there was no evidence for non-statins? Or evidence for LDL-C monitoring? Or standards for judging LDL-C response? I don’t think it was the message. I think it was the messaging. At some point, it’s not enough to just be right, it’s critical to promote the value of the message.

Clinicians are busy. We need simple advice for most things. The ACC/AHA Committee composed an elegant focused guideline, suggested that the patient-clinician relationship and decision making is paramount, and then let others dictate the message. For many, it seemed like there were guidelines, but no guidance. In this issue of LipidSpin, we hope you find both.
Clinical Feature:
Why Advocacy is Important to a Medical Specialty Organization

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In the strictest sense, “advocacy” is defined as the act or process of supporting a cause. With that as the basis of our discussion, why is advocacy important to a medical specialty organization such as the National Lipid Association (NLA)?

The NLA’s mission statement includes comments on professionalism and public service and this service relates to patient outcomes, with specific reference to reducing death and disability related to lipid disorders. Therefore, it is both reasonable and appropriate that the NLA develop strategies to work with state and federal legislators, governmental agencies and all systems that provide healthcare services with the goal of putting patients first.

I come from a decade of advocacy experience with the American College of Cardiology (ACC), serving in two unique but distinctly different roles. I am the chair of the Advocacy Steering Committee that works with the ACC’s advocacy staff to review all major advocacy issues for the ACC Board of Trustees and for the ACC president. We may review as few as two or more than 10 advocacy-related topics each month and render opinions or recommendations for communications on behalf of the ACC. In my role as chair of the ACC’s Political Action Committee (ACCPAC), I am a part of the leadership team of this voluntary, non-partisan political action committee formed to support the ACC’s mission to transform cardiovascular care and improve heart health. ACCPAC opens the doors for ACC members and staff to educate key members of Congress on cardiovascular issues. The funds that we raise amplify the voices of cardiovascular professionals to impact the political process and send a powerful message to Congress regarding the need to preserve and improve cardiovascular care.

When a medical specialty organization commits to an advocacy agenda, the first step is to define its message on behalf of the professionals it serves and the patients it represents. Such action is simple for organizations like the NLA simply based on its current mission statement. While there may be advantages if your organization develops a political action committee, it is neither a requirement nor a strict obligation of a medical specialty organization. The NLA can further pursue advocacy through engaging its members to participate in direct political action activities outside the organization.

Defining pathways for advocacy within the NLA will strengthen the organization’s ability to speak on behalf of patients with lipid disorders and their families. It also will enhance the NLA’s visibility on these particular issues.

Let me share with you some examples of messaging from the ACC. The theme for the 2014 ACC Legislative Conference was “Leading the Transformation in Healthcare.” Our message to Congress included developing a value-driven...
healthcare system, ensuring practice stability and securing the future of cardiovascular care. Our lobbying efforts in 2014 and early 2015 laid the foundation for the repeal of the flawed Medicare Sustainable Growth (SGR) formula.

The ACC believes that clinical data registries are the foundation for educating our cardiovascular care team on the real-world treatment of various cardiovascular disorders. The goal of these registries is to improve the appropriate use of resources, reduce hospitalizations and readmissions, and improve outcomes with a variety of treatment strategies and procedures.

The ACC’s National Cardiovascular Data Registry (NCDR) has expanded to cover a large number of cardiovascular conditions and procedures. We now include the vast majority of cardiac catheterizations/percutaneous coronary interventions (caths/PCIs), transcatheter aortic valve replacements (TAVRs), implantable cardioverter-defibrillators (ICDs), atrial fibrillation ablations and myocardial infarctions managed in the U.S. We have expanded to follow-up and management of adults with congenital heart disease. The companion outpatient Pinnacle Registry is a rapidly growing case depository in the U.S. and has educated researchers and our members on the daily care of cardiovascular patients. We have engaged the American College of Physicians to enrolled patients with diabetes to help us understand the relationship between diabetic care and cardiovascular disease. As part of our international strategy, we have partnered with practices in India and now have more than 100,000 patient records from India in a separate part of the Pinnacle Registry. These activities allow us to speak to Congress, federal agencies, and other partners in care delivery on what we have learned and how we project changes to allow us to be a leader in the improvement of cardiovascular care.

The theme of the 2015 Legislative Conference was “Member Engagement: ACC Members Making a Difference.” Our visits to Capitol Hill centered on thanking our members of Congress for the passage of the Medicare Reform Act and CHIP Reauthorization Act (MACRA) of 2015, while stressing that Congress should work with medical specialty societies and federal agencies to develop alternative payment models that allow clinicians to provide the most effective and efficient care to their patients. We urged Congress to utilize the expertise and experience of medical specialty societies to promote the usability of electronic health records. We made a strong case for Congress to support new funding for the National Institutes of Health (NIH) and the Food and Drug Administration (FDA) at the levels provided in the House-passed 21st Century Cures Act.

“The only through advocacy, careful vigilance, and continued outcomes research will we be able to serve our patients best.”

The practice models for our cardiovascular care teams vary across the country but are unified by the need to improve regulations as related to electronic health records and meaningful use. The ACC remains a strong voice on behalf of our members to streamline regulations and facilitate new programs centered on value-driven healthcare.

The ACC remains a beacon for sounding the message that the NIH needs increased funding for clinical research. We are committed to ensuring an appropriate pipeline of cardiovascular professionals through increased funding for graduate medical education.

The ACC’s new strategic plan has placed an emphasis on “population health.” As part of this, the ACC Population Health Policy and Health Promotion Committee recently convened a meeting of ACC members and a diverse array of experts from government agencies, universities, medical specialty societies, and private sector partners. During this meeting primary prevention, health equity, and social determinants of health, the changing healthcare landscape and the role of primary care professionals in advancing cardiovascular health were all discussed. As our ACC president, Dr. Kim A. Williams Sr., loves to say, “It is time to turn off the faucet rather than mop up the floor.”

NLA members can certainly join with the ACC and other sister cardiovascular organizations in advocacy messaging. As experts on lipid disorders, NLA members may want to consider serving patients through focused messaging from your areas of expertise.

As a cardiologist whose patient population includes a large number of patients with lipid disorders, I see some unique opportunities for the NLA in the years ahead. All of us in cardiovascular care have been spoiled by statins — a major weapon in the prevention and management of atherosclerotic disease — going generic and the cost of care and access to the drugs becoming less of a problem. PCSK9 inhibitors, on the other hand, highlight
issues regarding the cost of care, access to care, and appropriate use of resources. Other professional organizations, such as the ACC, look to the NLA to guide our thinking and approaches as we all deal with government agencies, state Medicaid programs, and health plans to ensure that patients receive the best care possible and have the best possible outcome from the management of their lipid disorder. The interpretation of outcome studies and the generation of guidelines should remain with care professionals rather than with governmental agencies. The ACC and the NLA should continue to work together on future advocacy efforts related to the prevention of cardiovascular disease.

It appears that we are in the infancy of personalized medicine, and the future will likely offer clinicians a variety of genetic tests to identify specific genetic disorders and to identify populations with genetic variations that place them at higher risk for atherosclerotic disease. Again, the interpretation and management of emerging research will depend on professionals in the field. It seems appropriate that the NLA be among the voices to ensure that outcomes and value, not just unit costs, are the drivers of healthcare.

Just as personalized medicine is an emerging paradigm, we know that the passage of MACRA accelerates the change in reimbursement for American physicians. Congress has mandated that the Centers for Medicare and Medicaid Services (CMS) rapidly move to a value-based payment system with a step-wise increase in payments through alternative payment models. Each of us can only speculate about whether such systems will enhance or diminish access to care for patients who may need expensive services. Only through advocacy, careful vigilance, and continued outcomes research will we be able to serve our patients best.

Disclosure statement: Dr. Sherman has no disclosures to report.

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Spring 2016 Testing Window
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(Application Deadline: Friday, March 25, 2016)

Summer 2016 Testing Window
(Application Deadline: Friday, May 27, 2016)

Fall 2016 Testing Window
September 25, 2016 – November 5, 2016
(Application Deadline: Friday, September 16, 2016)
Guest Editorial: Bringing the Nation Together to Save a Million Hearts by 2017

JANET S. WRIGHT, MD, FACC
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Every 39 seconds, a U.S. adult dies from cardiovascular disease (CVD),¹ the nation’s top killer and a leading cause of disability for Americans both young and old. We spend about 1 out of 6 healthcare dollars on this disease each year — an estimated nearly $320 billion in healthcare expenses and lost productivity.¹ But those human and economic costs could be lower if we expanded our national actions in prevention.

About half of U.S. adults (46.5 percent) have at least one of three major — and preventable — risks for cardiovascular disease: uncontrolled high blood pressure, uncontrolled low-density lipoprotein cholesterol (LDL-C), or current tobacco use.² Only around half (53.8 percent) of people who are at risk for CVD use aspirin; blood pressure is controlled in only about half of those with hypertension;³ and only one-third of American adults with a history of high cholesterol have optimal levels.⁴ Common and largely controllable risk factors, hundreds of thousands of preventable events, and under-performance on measures that matter: That’s why Million Hearts® was launched in late 2011.

Million Hearts® is a national public-private initiative with the goal of preventing 1 million heart attacks and strokes by 2017. The initiative brings together communities, healthcare professionals, health systems, nonprofit organizations, federal agencies, and private-sector partners to implement proven interventions in clinical settings and communities and to empower Americans to make healthier choices.

Connecting Public Health and Clinical Care
Reaching the Million Hearts® goal will take focused action by both public health and healthcare professionals. The most powerful public health interventions for preventing cardiovascular events in a five-year time frame are those intended to reduce smoking prevalence, lower daily sodium intake, and eliminate artificial trans-fat from the food supply. These actions can impact millions of people, resulting in a healthier population — and thousands fewer cardiovascular events. Healthcare professionals can have the greatest impact on heart attack and stroke rates by helping their patients achieve excellence in the ABCS:

- Aspirin when appropriate
- Blood pressure control
- Cholesterol management
- Smoking cessation

Achieving high performance on the ABCS will prevent more deaths from cardiovascular disease than will other clinical preventive services,⁵ and recent data show much room for improvement.⁶

The “C” in the ABCS
As experts in the field of lipids, you know that high cholesterol can be well managed for most patients through a combination of effective medications and good habits.⁷ Data from the National Health and Nutrition Examination Survey (NHANES)
show signs of progress with the then-current cholesterol management increasing from 33.0 percent in 2009–2010 to 42.8 percent in 2011–2012. But opportunities to improve abound, especially among subsets of our population. A recent analysis of 2005–2012 NHANES data\(^4\) shows that about 78 million (36.7 percent) U.S. adults were on or eligible for cholesterol treatment based on the 2013 ACC/AHA guidelines.\(^9\) A little more than half (55.5 percent) were on cholesterol-lowering medication and 46.6 percent were modifying their lifestyle with exercise, diet, and/or weight control; more than one third (35.5 percent) were doing neither. These population-level data also shed light on troubling U.S. disparities. A lower proportion of eligible men than women were taking medication (52.9 vs 58.6 percent, \(p=0.01\)), as were a lower proportion of non-Hispanic blacks (46.0 percent) and Mexican Americans (47.1 percent) than non-Hispanic whites (58.0 percent, \(p<0.001\)), highlighting the need to systematically find and address these treatment gaps.

### Partnerships and Focused Actions: Keys to Success

Getting to excellence in the ABCS will take teams, technology, and new workflows — and a strong bridge between clinical and community settings. In an effort to communicate the core tenets of these efforts, “Detect. Connect. Control.” has become a mantra in Million Hearts, applying to communities and clinics for hypertension control and other issues including cholesterol control. Those at high risk for CVD can be found in community settings and also “hiding in plain sight” within practices and systems across the country. For example, we know that three-quarters of the 35 million adults with uncontrolled hypertension saw their usual source of care at least twice in the previous 12 months.\(^{10}\) Algorithms can be designed to sift through electronic health record (EHR) data and find those patients with uncontrolled or not-yet-diagnosed hypertension.\(^7\) Those discovered are then connected to healthcare experts and community resources to help achieve safe control.

With the challenge of helping millions of Americans better manage their cholesterol to reduce cardiovascular risk, Million Hearts depends on organizations and individuals across the country. In addition to the National Lipid Association, Million Hearts partners include numerous public and private sector organizations, such as the Association of State and Territorial Health Officials, National Committee for Quality Assurance, Association of Black Cardiologists, Kaiser Permanente, Men’s Health Network, Preventive Cardiovascular Nurses Association, American Heart Association, National Forum for Heart Disease and Stroke Prevention, Women’s Heart Alliance, and others. All are focused on implementing what works to avoid preventable events and contribute to cardiovascular health.

Million Hearts\(^\circ\) partners are innovating to make our vision a reality. For example, the American Pharmacists Association Foundation’s Asheville Project created a community-based medication therapy management program for high blood pressure, cholesterol, and triglycerides to benefit 12,000 employees of the city and a local hospital system. Participants received employer-sponsored counseling and classes with clinically trained educators and pharmacists. Employers also reduced or eliminated medication co-payments. Over a period of 6 years, the proportion of people who achieved cholesterol targets rose from 50 to 75 percent. Healthcare costs paid by the employer fell by nearly half, and the percentage of health plan costs related to cardiovascular disease dropped substantially, from 30.6 to 19.1 percent.

The Kaiser Permanente Colorado High Blood Pressure and Cholesterol Management Program established patient registries and outreach lists to manage the care of patients with high blood pressure and cholesterol in the company’s
health insurance system. The risk of a cardiac-related death decreased by 88 percent among Colorado residents who enrolled within 90 days of a heart attack, compared with patients who did not enroll in the program. Through the program, the percentage of patients screened for cholesterol rose from 55 to 97 percent, and the percentage of patients who met target cholesterol readings increased from 26 to 73 percent.

2016 and Beyond
In this next year, Million Hearts® will draw an even greater focus on “Detect. Connect. Control.” We will be working with partners to find more people at risk and to match up those individuals with the experts and resources that can help them live healthier, event-free lives. As communities focus on healthy-habit supports for exercise and nutrition, clinical practices and systems can contribute through widespread implementation of standardized treatment protocols, EHR-facilitated registries and algorithms, and proactive, team-based care. As a result, more people can write a new and healthy family history. If you already are one of the many Million Hearts® supporters, thank you. If you’re just learning about the initiative now, we hope you will join us and take action. Together, we can make a difference for one person, one family, one community, one state — and reach 2017 with more than 1 million healthier hearts.

Disclosure statement: Dr. Wright has no disclosures to report.

References are listed on page 35.
This edition of the LipidSpin is devoted to guidelines vs. guidance. We all strive to practice evidence-based medicine (EBM) and our understanding of science has moved forward at a quick pace. Multiple journals come out every week with new articles that advance medicine’s knowledge base. Large, randomized controlled trials (RCTs) get headlines when a positive or negative result is found. However, in the everyday practice of seeing patients and making treatment decisions, how applicable can a large RCT be to your particular patient population? Also, what method do you use to interpret positive and negative results within explanatory (efficacy) vs. pragmatic (effectiveness) trials (Table 1)?

Schwartz and Leoouch first described the explanatory and pragmatic trial approaches in 1967. By definition, an explanatory (efficacy) trial is a traditional RCT designed to evaluate the effects of treatments and is considered the gold standard. A pragmatic (effectiveness) trial is designed to test a treatment or intervention in everyday clinical settings to allow for generalizability and maximum application. An explanatory trial has a main purpose of answering the question, “Does an intervention work under ideal conditions?” The main purpose of a pragmatic trial is to answer the question, “Can an intervention work under usual or real-world conditions?”

So how can a clinician compare the differences between an explanatory and a pragmatic trial? The Pragmatic Explanatory Continuum Indicator Summary (PRECIS) was developed and validated with 10 domains of focus in evaluating trial design and application and has subsequently been refined to focus on nine domains in the PRECIS-2 Tool. These key domains (Figure 1) allow for comparison between explanatory and pragmatic trials. For trials to be clinically meaningful, results must be relevant to specific patient populations in specific settings. Trials have many factors that determine internal and external validity, and these vary between
explanatory and pragmatic trials. Some of these factors include the condition under investigation, drug regimens, compliance, patient characteristics, costs, co-morbidities, and concomitant treatment regimens.

What conclusions can be drawn from explanatory versus pragmatic trials? There are advantages and disadvantages to the application of each trial type on an individual-patient level. The overall strength of explanatory trials is that a “negative” result can directly inform practitioners, because an intervention/treatment that has been shown to not work under ideal conditions is unlikely to work under usual conditions. The main weakness of an explanatory trial is that a “positive” result does not always directly translate to practical use (limited external validity) because the study population may be too narrow or may include optimal circumstances that may not apply to the general population. Pragmatic trials essentially have the opposite strengths and weaknesses. When a “negative” result is seen in a pragmatic trial, it can be unclear as to whether the treatment/intervention is not effective in specific patient populations because it often is studied in the general population under usual conditions. On the other hand a “positive” result can help inform individual patient- and population-based decisions because the study was performed under usual conditions, resulting in increased external validity. Table 1 provides more details on how to evaluate “positive” and “negative” outcomes within explanatory and pragmatic trials.

How else can the relationship between pragmatic trials and explanatory trials be explained? Figure 2 describes key differences as they relate to five factors: validity, size, design, focus population, and study type. Trials often are not purely explanatory or pragmatic, so what factors might suggest trial results are translatable and applicable in a primary care or specialty setting? These factors could revolve around the study design itself. For example:

1) Comparison groups do not represent current standards of care
2) Trial protocols force uncommon clinical scenarios
3) Outcomes include less meaningful end points

In addition, environment of the healthcare delivery system itself could contribute

<table>
<thead>
<tr>
<th>Trial Type</th>
<th>Positive Trial Result</th>
<th>Negative or Neutral Trial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Explanatory (Efficacy)</td>
<td>Intervention works, but it’s limited by a lack of knowledge about whether it will work for clinicians and patients in “real-world practice”</td>
<td>Studied intervention could have no effect or could cause harm, resulting in the abandonment of this approach</td>
</tr>
<tr>
<td>Pragmatic (Effectiveness)</td>
<td>Clear benefit and ability to adopt intervention in “real-world practice”</td>
<td>Ambiguous area because the question remains – Why did intervention fail? Was failure a result of an ineffective intervention vs. limitations with providers or patients following the intervention protocol?</td>
</tr>
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Table 1: Evaluation of Conclusions from Explanatory and Pragmatic Trials (Adapted from reference)
in a negative manner with the following considerations:

1) Limited availability of providers or resources
2) Inadequate levels of reimbursement

Thirdly, therapeutic implementation issues could be part of the problem with effectiveness trials. These might include:

1) Complex and multi-faceted therapies that are challenging to implement
2) The way in which the procedural experience of providers influences outcomes

Finally, patient selection issues can play a factor in pragmatic trials. These include:

1) Biases in the patients who are eligible for a therapy
2) Biases in patients who ultimately are selected for (or agree to) a therapy

In conclusion, when reviewing a clinical trial, keep in mind the factors listed above to help you decide if the results are applicable in your particular practice setting.

Disclosure statement: Dr. Uusinarkaus has no disclosures to report. Dr. Marrs has no disclosures to report.

References are listed on page 35.
The American College of Cardiology/American Heart Association (ACC/AHA) Blood Cholesterol Guideline recommends high-intensity statin therapy for high-risk patients to reduce the risk of major adverse cardiovascular events (MACE).\(^1\) Despite treatment with high-intensity statins, patients continue to experience MACE. Additionally, some patients have a less-than-anticipated response to, or are unable to, tolerate high-intensity statins. Whether additional non-statin lipid-lowering therapies further reduce the risk of MACE in these patients has not been extensively studied. Several trials investigating the benefit of non-statin lipid-lowering medications in addition to statins have been published since publication of the ACC/AHA guideline and provide new information for managing high-risk patients.\(^2,4\)

The Improved Reduction of Outcomes: Vytorin Efficacy International Trial (IMPROVE-IT) compared simvastatin 40 mg/placebo to simvastatin/ezetimibe in patients with low-density lipoprotein cholesterol (LDL-C) of 50-100 mg/dL and recent acute coronary syndrome.\(^2\) The simvastatin/ezetimibe group had a 6 percent reduction in the primary outcome of MACE vs. the simvastatin/placebo group (hazard ratio (HR): 0.936; 95 percent confidence interval (CI), 0.89-0.99; \(p=0.016\)). The mean LDL-C level attained at one year was significantly lower in the simvastatin/ezetimibe vs. simvastatin/placebo group (53.2 vs. 69.9 mg/dL; \(p<0.001\)) with similar rates of adverse effects in both groups. Although IMPROVE-IT used moderate-intensity statin in a population recommended to receive high-intensity treatment, the results of IMPROVE-IT may apply to high-risk patients unable to tolerate high-intensity statins and those at increased risk of statin intolerance. IMPROVE-IT also supports the hypothesis that attaining lower LDL-C with additional non-statin lipid-lowering medication results in lower MACE.

Clinical trials with proprotein convertase subtilisin-kexin type 9 (PCSK9) inhibitors are underway to further test the LDL-C lowering hypothesis. Two of the first clinical trials evaluating PCSK9 inhibitors, Efficacy and Safety of Alirocumab in Reducing Lipids and Cardiovascular Events (ODYSSEY LONG TERM)\(^3\) and Open-Label Study of Long-Term Evaluation against LDL Cholesterol (OSLER)\(^4\) reported significant reductions in LDL-C in patients treated...
with PCSK9 inhibitor vs. placebo. Although not a primary outcome, both studies reported significant reductions in MACE in patients receiving PCSK9 inhibitors compared to placebo.

In ODYSSEY LONG TERM, patients at high-risk of MACE receiving maximally-tolerated statin and an LDL-C > 70 mg/dL were randomized to receive alirocumab or placebo. At 24 weeks, the LDL-C reduction from baseline was 62 percent with alirocumab (mean LDL-C: 48 mg/dL) vs. 0.8 percent with placebo (mean LDL-C: 118 mg/dL). Post-hoc analysis demonstrated a significantly lower rate of MACE with alirocumab compared to placebo at 78 weeks (HR: 0.52; 95 percent CI, 0.31-0.90; p=0.02).

Similar reductions in LDL-C and MACE were reported in the OSLER trial. Patients with or without current statin treatment and a median LDL-C of 120 mg/dL were randomized to evolocumab or placebo. At 12 weeks, the evolocumab group attained a 61 percent reduction in LDL-C from baseline (median attained LDL-C: 48 mg/dL) compared to placebo. Prespecified exploratory analysis of MACE at 1 year favored treatment with PCSK9 inhibitor vs. placebo (HR: 0.47; 95 percent CI, 0.28-0.78; p=0.003).

While previous trials failed to show clinical benefit from adding non-statin lipid-lowering therapies (niacin and fibrates, specifically) to statins vs. statins alone, the medications used in these studies primarily improved high-density lipoprotein cholesterol and triglycerides as opposed to LDL-C. The differences in LDL-C between groups at study end were at the most 10 mg/dL. Alternatively, LDL-C reductions between groups were significant in IMPROVE-IT, ODYSSEY LONG TERM and OSLER suggesting that further LDL-C reduction reduces risk of MACE in high-risk patients. Updates to the current ACC/AHA guidelines should recommend adding non-statin lipid-lowering medications (ezetimibe and/or PCSK9 inhibitors) to maximally-tolerated statin in order to further lower LDL-C in high-risk patients unable to tolerate high-intensity statin or for those who demonstrate a less-than-expected LDL-C reduction despite maximally-tolerated statin.

Disclosure statement: Dr. Kelly has no disclosures to report. Dr. Olson has no disclosures to report.

References are listed on page 35.
In keeping with the theme of this issue of the LipidSpin, this Specialty Corner is devoted to advice for the practicing clinician who has to make daily decisions on how best to protect his/her patients from cardiovascular disease.

It seems since the 2013 publication of the American College of Cardiology/American Heart Association (ACC/AHA) guidelines, we have been engaged in countless discussions about them, either for or against, and all of the reasons to support each view. Well, the world is a complicated place, because we really have at least six new guidelines from which to pick (Figure 1). However, despite all these guidelines, the common thread that joins them all is reduction of low-density lipoprotein cholesterol (LDL-C) to reduce and prevent events. We have to make every clinical decision with this in mind. This is not meant to ignore other important factors, such as smoking, hypertension, and diabetes. My comments as a lipidologist are focused the same as all the lipid guidelines — on how to impact LDL-C. I also plan to further address my belief that apolipoprotein B (ApoB) and low-density lipoprotein particle number (LDL-P) are more useful indices to measure to improve on LDL-C-centric thinking.

A quote from the most talked about guideline — the ACC/AHA guidelines — states, “The expert panel acknowledges that our process did not provide for a comprehensive approach to the detection, evaluation, and treatment of lipid disorders…”[1] As clinicians doing the heavy lifting every day for our patients at risk, who among us does not want a comprehensive approach, and the freedom to treat our individual patients’ lipid disorder with more than just a moderate- or high-intensity statin? We cannot ignore residual dyslipidemia because one group of experts couldn’t find randomized controlled trials (RCTs) to support its treatment. I believe therapy goals are as important for lipids as they are for blood pressure and glucose control. Despite the inability to prove that lowering hemoglobin A1c (HbA1c) prevents events, most clinicians still have therapy goals. Despite having no evidence-based medicine for our individual favorite therapy using three or more antihypertensive agents, most clinicians have a target blood pressure range they try to achieve. Accordingly, I also believe in goals of lipid therapy. In an excellent editorial about guidelines, Dr. Henry Ginsberg reviews for us the list of RCTs that employed goals of therapy in both primary and secondary prevention.[2] We also know from the Cholesterol Treatment Trialist study that lower is better, regardless of where you start.[3] We have data from bile acid binding resins,[4] niacin,[5] and ezetimibe[6] trials that all demonstrate that lowering LDL-C with non-statins reduces events. It is my recommendation to my colleagues that we use the guidelines as guidance, not as commandments. Where the guidelines are able to provide evidence-based data that applies to our individual patient, they clearly make sense. What does not make sense is doing nothing in the absence of evidence-based medicine that has not yet been elucidated for my high-risk patient with complex dyslipidemia.
Therefore, I recommend that all clinicians understand what non-statin drugs such as ezetimibe, niacin, bile acid binding resins, and even fibrates do, and how. Clinicians should understand lipid metabolic pathways to gain an understanding of how triglycerides influence the numbers we see on our test results, as I outlined in an earlier LipidSpin article. We need to understand how the new class of proprotein convertase subtilisin/kexin type 9 (PCSK9) therapies work, when to employ them, and what to expect from them. Only armed with that comprehensive knowledge can we move beyond the tyranny of guidelines, take what we need from them, and then treat the individual patient to address each and every risk by which that patient is confronted.

The constraints of RCTs make it virtually impossible to detect the changes in the progression of atherosclerotic vascular disease that occur in timeframes longer than the trial. We all realize that atherosclerosis is a cumulative phenomenon that manifests over the life of our patient. The key is to know when and in whom to intervene, as well as to quantify the goals and success of our intervention.

I, for one, endorse the National Lipid Association Recommendations as a practical and clinically meaningful guide by which to assess risk and to assist the clinician in knowing when to intervene. They also quantify therapy goals.

Now that we have discussed moving beyond the constraints of therapy guidelines, I would like to address what I believe are the most accurate methods of determining our lipid status. The more accurately we can measure dyslipidemia, the more accurately we can treat it.

In clinical practice, we often are confronted by a significantly variable response to lipid therapy. We also are confronted by so-called residual dyslipidemia when our best statin therapy fails to get our patient to goal. How should we proceed when confronted with this scenario?

My recommendations in this area begin first by most accurately determining the status of the patient’s dyslipidemia. The most recent NLA Recommendations address this issue by setting both non-HDL-C goals and LDL-C goals. These guidelines say that non-HDL-C is superior to LDL-C, with ApoB as a secondary target. This approach also is supported by the European Atherosclerosis Society, Canadian Atherosclerosis Society, and International Atherosclerosis Society guidelines.

Back to the premise that we only take from guidelines what they can prove to us. To consider this, we must first consider the concept of discordance. This term refers to individuals in whom other measures of atherogenic particles are inconsistent with LDL-C measurements. The most classic example of discordance would be a patient who is in the 50th percentile by LDL-C measurement, but in a significantly different percentile by measuring Apo B or LDL-P. This is a painfully simplified example, but it makes the point that some of us transport our lipids differently from others, and may be at more or less risk than LDL-C measurements imply. Simply considering the weight of our cholesterol per deciliter of plasma is, in my view, not always as accurate as I would like. To this point, there also are multiple clinical trials that were not included in ACC/AHA guidelines that demonstrated the superiority of measurements of ApoB and non-HDL-C over LDL-C in patients that demonstrated such discordance. This list includes the Quebec Cardiovascular Study, the Framingham Offspring Study, Interheart, Women’s Health study, and MESA.

This is of critical importance to the practicing clinician, because the patients most likely to be affected by discordance are ones we generally consider to be higher-risk patients — diabetics and those with insulin resistance. If my clinical practice resembles the practices of my colleagues, I would submit that diabetics and patients with insulin resistance are sitting in our waiting rooms every day of our practice lives. What an opportunity we have to recognize the potential severity of their dyslipidemia — if we only take a moment to think about testing beyond LDL-C and consider residual dyslipidemia.

In summary, our clinical practices provide us both the opportunity and the obligation to do the very best for our patients. I believe clinicians and patients are best served when we clinically integrate the suggestions that guidelines provide with our knowledge of pharmacology and physiology — and of our patients trusting us to palliate their risks.

Disclosure Statement: Dr. Lillo was on the speakers bureau for Amgen, Sanofi-Regeneron, Merck, Amarin, Kowa, and Actavis. He also participated in research trials for Amgen, Merck, Abbvie, Ironwood, Pearl, Pfizer, Theravance, and Daich-Sankyo.

References are listed on page 35.
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Clinical question: In statin-treated patients who also require strong CYP3A4 inducers, does statin therapy need to be preemptively modified to account for decreased systemic exposure secondary to increased metabolism?

Recent guidelines endorse the use of appropriate-intensity statin therapy to match an individual’s risk for atherosclerotic cardiovascular disease (ASCVD).1,2 The 2013 American College of Cardiology/American Heart Association (ACC/AHA) guidelines for the treatment of blood cholesterol to reduce ASCVD risk stresses the importance of considering drug-drug interactions that may influence statin safety, but no detail is provided on how to manage these interactions. The 2014 National Lipid Association Statin Safety Task Force guidelines include a detailed review of statin pharmacokinetics (PK).3 These guidelines review drug interactions that increase the risk of statin-related adverse effects, especially myopathy, which is mediated by increased systemic exposure as measured by area under the curve (AUC). On the other hand, little guidance exists to direct clinicians on how to address statin drug-drug interactions with CYP3A4 inducers that result in decreased systemic exposure.

Phase I oxidation reactions carried out by CYP3A4 are responsible for metabolizing more than 50 percent of all manufactured drugs.4 Predicting adverse events from potential CYP3A4 interactions is difficult, in part because a 10-fold difference in activity and a 40-fold difference in enzyme expression may be seen among individuals.5 PK studies have demonstrated that when CYP3A4-dependant statins (lovastatin, simvastatin and atorvastatin) are combined with strong CYP3A4 inducers (e.g. phenytoin, carbamazepine, rifampin), statin AUC is decreased by 53 percent to 82 percent (i.e. < two-fold).6,7 What effect this has on lowering low-density lipoprotein cholesterol (LDL-C) is not entirely clear.

While several case reports have suggested an association between concurrent...
CYP3A4 inducers and inadequate LDL-C response, it also has been demonstrated that a 60 percent reduction in the AUC of certain statins does not result in any statistically significant reduction in LDL-C lowering. Tertiary drug information resources suggest that clinicians either consider changing to a non-CYP3A4-dependant statin or monitoring for LDL-C-lowering response. It is important to recognize that a change in statin therapy may have unintended consequences.

For example, a patient being initiated on phenytoin who is preemptively switched from atorvastatin to rosuvastatin may incur substantial additional costs. Alternatively, if an ASCVD patient were to be switched to pravastatin, the patient would no longer be receiving the high-intensity statin, as recommended, and may not achieve the LDL-C goal.

The Clinical Pharmacy Cardiac Risk Service (CPCRS) at Kaiser Permanente Colorado currently manages more than 16,000 patients with ASCVD. Based on a cursory review of 84 patients on a CYP3A4-dependent statin plus either phenytoin and/or carbamazepine, we determined that the vast majority of patients (94 percent) had attained at least the anticipated percentage of LDL-C reduction based on their individual statin intensity (Figure 1). There were instances in which patients appeared to have a less than or greater than anticipated LDL-C reduction based on statin intensity, which we presume was likely because of variables such as adherence to medication and/or lifestyle.

Statin drug interactions involving CYP3A4 inducers generally are less of a concern than interactions with inhibitors. Based on our collective experience at CPCRS, we feel it is unnecessary to preemptively modify a patient’s statin or dose to accommodate a concurrent CYP3A4-inducer interaction. Overall, patients in our practice seemed to respond as expected to statin therapy when co-administered with CYP3A4 inducers. These observations warrant further investigation in a prospectively designed study to confirm their validity. A final consideration is that a change in therapy may have cost implications and or lead to patients being placed on a lower-than-recommended intensity of statin. Given that there is always the potential for variation in patient response to drug therapy, monitoring for statin efficacy via fasting lipid panel — to ensure adequate LDL-C response to statin therapy — is recommended.

Disclosure statement. Dr. Lamprecht has no disclosures to report. Dr. Todd has no disclosures to report.

References are listed on page 35.
Muscle symptoms in patients on statin therapy are prevalent and offer a complicated differential for providers to entertain. Symptoms range from relatively mild aches and pains to severe and debilitating weakness and pain. Statins are taken by more than 25 million patients across the globe and have been clearly associated with such complaints. The range of muscle complaints attributed to statin use extends from myalgia — a subjective complaint without creatine kinase (CK) elevation (the most common scenario) — to rhabdomyolysis, which is myonecrosis with myoglobinuria with or without acute renal failure.¹ These definitions were developed in 2014 by the National Lipid Association’s (NLA) Muscle Safety Expert Panel.

Parallel to the NLA’s work on developing these definitions, a multidisciplinary international group of experts also convened in 2014 as part of the Phenotype Standardization Project to categorize the wide clinical spectrum of muscle symptoms related to statin therapy (Table 1). They identified categories ranging from myalgias with or without CK elevation to the relatively new identification of statin-induced necrotizing autoimmune myopathy (SINAM).² This classification mirrors the NLA definitions, with the addition of asymptomatic CK elevations and of autoimmune-mediated necrotizing myonecrosis. The autoimmune condition is differentiated from myopathy alone by the presence of 3-hydroxy-3-methyl-glutaryl-coenzyme A reductase (HMG-CoA reductase) auto-antibodies.

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**Case Study:**

Autoimmune-Mediated Necrotizing Myositis Due to Statin Therapy

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ANTOINE PHAM, DO
Hospital Internists of Austin
St. David’s Medical Center
Austin, TX

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1. These definitions were developed in 2014 by the National Lipid Association’s (NLA) Muscle Safety Expert Panel.
2. This classification mirrors the NLA definitions, with the addition of asymptomatic CK elevations and of autoimmune-mediated necrotizing myonecrosis. The autoimmune condition is differentiated from myopathy alone by the presence of 3-hydroxy-3-methyl-glutaryl-coenzyme A reductase (HMG-CoA reductase) auto-antibodies.
A muscle biopsy was sent to the Mayo Clinic and found to have significant CK elevation and incomplete resolution on dechallenge. 

Cardiopulmonary exam was normal. An abdominal exam showed no distention, splenomegaly, or ascites. The patient had a resting blood pressure of 156/80 mmHg. His sclerae were non-icteric and his vitals were stable.

**Case Study**

The patient is a 62-year-old man with a past medical history significant for hypertension, hyperlipidemia, and type 2 diabetes mellitus. He initially was admitted with complaints of muscle aches, weakness, and a sore throat. He was diagnosed with rhabdomyolysis with a CK elevation peaking at 36,734 u/L. He was given intravenous fluids and steroids, and monitored for eight days. He clinically improved and was discharged from the hospital when his CK was trending downward to 24,046 u/L. The evaluation included a negative antinuclear antibody (ANA) panel and a normal serum creatinine. A gastrocnemius muscle biopsy was sent to the Mayo Clinic and results showed muscle symptoms, CK <4x ULN, complete resolution on dechallenge.

The patient was re-admitted to the medical/surgical floor and was immediately given an IV bicarbonate infusion along with serial CK labs. Because of his long-term use of atorvastatin, a serum anti-HMGCR antibody was drawn. A neuromuscular lab. Findings demonstrated type II fiber atrophy and possible re-innervation by type I motor neurons. Two days following his discharge, he returned to the hospital having trouble speaking and swallowing. CK was measured at 26,665 u/L and he was weaker than he had been two days when discharged from the hospital.

Physical exam revealed a weak patient in no respiratory distress. He was afebrile and had a resting blood pressure of 156/80 mmHg. His sclerae were non-icteric and his cardiopulmonary exam was normal. An abdominal exam showed no distention, splenomegaly, hepatomegaly, or ascites. There was bilateral pitting edema from his ankles up to his knees. Neurological exam revealed symmetrical bilateral proximal shoulder girdle and thigh weakness.

<table>
<thead>
<tr>
<th>SRM classification</th>
<th>Phenotype</th>
<th>Incidence</th>
<th>Definition</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>SRM 0</td>
<td>CK elevation &lt;4xULN</td>
<td>1.5–26%</td>
<td>No muscle symptoms</td>
<td>Refs. 1,20,34,35,67</td>
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<td>SRM 1</td>
<td>Myalgia, tolerable</td>
<td>190/100,000 Patient-years; 0.3–33%</td>
<td>Muscle symptoms without CK elevation</td>
<td>Refs. 1,19,21,50,68</td>
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<td>SRM 2</td>
<td>Myalgia, intolerable</td>
<td>0.2–2/1,000</td>
<td>Muscle symptoms, CK &lt;4x ULN, complete resolution on dechallenge</td>
<td>Ref. 20</td>
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<td>SRM 3</td>
<td>Myopathy</td>
<td>5/100,000 Patient-years</td>
<td>CK elevation &gt; 4x ULN ± muscle symptoms, complete resolution on dechallenge</td>
<td>Ref. 1</td>
</tr>
<tr>
<td>SRM 4</td>
<td>Severe myopathy</td>
<td>0.11%</td>
<td>CK elevation &gt;10x ULN &lt;50x ULN, muscle symptoms, complete resolution on dechallenge</td>
<td>Refs. 20,69</td>
</tr>
<tr>
<td>SRM 5</td>
<td>Rhabdomyolysis</td>
<td>0.1–8.4/100,000 Patient-years</td>
<td>CK elevation &gt;10x ULN with evidence of renal impairment + muscle symptoms or CK &gt; 50x ULN</td>
<td>Refs. 4,6,25,44,45</td>
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<td>SRM 6</td>
<td>Autoimmune-mediated necrotizing myositis</td>
<td>~2/million per year</td>
<td>HMGCR antibodies, HMGCR expression in muscle biopsy, incomplete resolution on dechallenge</td>
<td>Refs. 51,70</td>
</tr>
</tbody>
</table>

Table 1.

Their expression on muscle biopsy, and incomplete resolution on cessation of statin therapy. Incidence of SINAM is estimated to be two patients per million per years.²

We present a case of a 62-year-old man with complaints of severe lower extremity weakness, edema, paresthesias in his hands and difficulty swallowing. He was found to have significant CK elevation and serologic confirmation of autoimmune-mediated necrotizing myonecrosis, SRM 6.

CK, creatine kinase; HMGCR, 3-hydroxy-3-methylglutaryl-coenzyme A reductase; SRM, statin-related myotoxicity; ULN, upper limit of normal.

<table>
<thead>
<tr>
<th>SRM</th>
<th>Classification</th>
<th>Phenotype</th>
<th>Incidence</th>
<th>Definition</th>
<th>Reference</th>
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<td>1.5–26%</td>
<td>No muscle symptoms</td>
<td>Refs. 1,20,34,35,67</td>
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<td>0.2–2/1,000</td>
<td>Muscle symptoms, CK &lt;4x ULN, complete resolution on dechallenge</td>
<td>Ref. 20</td>
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<td>CK elevation &gt;10x ULN &lt;50x ULN, muscle symptoms, complete resolution on dechallenge</td>
<td>Refs. 20,69</td>
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<tr>
<td>5</td>
<td>Rhabdomyolysis</td>
<td>0.1–8.4/100,000 Patient-years</td>
<td>CK elevation &gt;10x ULN with evidence of renal impairment + muscle symptoms or CK &gt; 50x ULN</td>
<td>Refs. 4,6,25,44,45</td>
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<tr>
<td>6</td>
<td>Autoimmune-mediated necrotizing myositis</td>
<td>~2/million per year</td>
<td>HMGCR antibodies, HMGCR expression in muscle biopsy, incomplete resolution on dechallenge</td>
<td>Refs. 51,70</td>
<td></td>
</tr>
</tbody>
</table>
anemia, so azathioprine was discontinued. This was compounded by respiratory failure and worsening weakness, including dysphagia. Subsequently, his anti-HMGCR AB was strongly positive at more than 200 units (strong positive is considered >60 U). Thus, we decided to start rituximab 1,000 mg IV weekly for four weeks followed by IVIG 30 gm IV daily for five days. His acute kidney injury was deemed the result of sucrose nephropathy from the level of sucrose in IVIG. This reversed quickly when changed to an IVIG infusion with lower sucrose levels, and his renal function normalized. The patient was then given another round of pulse-dosed IV methylprednisolone for five days.

As the patient’s clinical picture began to improve, he was extubated. His liver function tests normalized. He was able to regain some physical strength and was able to stand and sit upright. His dysphagia was still severe and he was dependent on a percutaneous endoscopic gastrostomy (PEG) tube for nutrition. Finally, his CK levels began to decrease after initiating rituximab. After five weeks of combined therapy with Rituximab, steroids, and intermittent IVIG infusions, his CK levels were in the normal range.

**Discussion**

Statins are among the most prescribed medications in the world because of the excellent evidence demonstrating efficacy in primary and secondary prevention of cardiovascular events. Muscle complaints are common in patients treated with these medications, affecting up to 20 percent of treated patients. Estimates from randomized controlled clinical trials suggest the incidence of true myotoxicity is 1.5 to 2.5 percent. Most cases of muscle symptoms in statin treated patients are mild and improve with cessation of therapy or a change to a different statin or non-statin lipid lowering therapy. The incidence of hospitalization-requiring myotoxicity in one analysis was one in 22,727 patients per year.

We have presented a case of a patient with severe myotoxicity because of autoimmune-mediated necrotizing myositis. This patient had severe muscular weakness, rendering him unable to swallow or breathe on his own, and dependent upon supportive care with endotracheal intubation and a PEG tube.

HMG CoA reductase is a key component of cholesterol biosynthesis, catalyzing the
conversion of HMG CoA into mevalonate, a precursor of cholesterol. Its inhibition by statin therapy is key to their efficacy, and may also be responsible for muscular symptoms due to impaired synthesis of ubiquinone and other cell components. Antibodies to HMG-CoA (anti-HMGCR) develop rarely in patients on statin therapy. However, this may lead to the development of statin-associated myopathy that does not resolve with cessation of statin therapy. These auto-antibodies can be detected using a specific serologic assay and may be detected by muscle biopsy of the affected cells. Regenerating muscle cells express high levels of HMGCR and may sustain the autoimmune response even after statins are discontinued.

Though statin-induced autoimmune-mediated necrotizing myonecrosis is rare, it is important to consider it as a possible etiology for severely ill patients and for patients whose muscle symptoms do not resolve with cessation of statin therapy. Because this autoimmune condition does not resolve with cessation of therapy, aggressive immunosuppressive treatment is needed. In reported cases, weaning off of steroid therapy, even months later, often resulted in a relapse of weakness, elevated CK and hospitalization.

Figure 1 depicts a useful algorithm for the assessment of patients with suspected statin-induced muscle symptoms and allows for consideration of the rare but serious condition found in our patient.

Disclosure statement: Elizabeth Jackson received speakers bureau honorarium from Sanofi/Regeneron. Dr. Chafizadeh has no disclosures to report. Dr. Pham has no disclosures to report.

References are listed on page 36.
Chapter Update:
Goals for the New Year

THOMAS A. HAFFEY, DO, FACC, FACOI, FNLA
President, Southwest Lipid Association
Clinical Professor, Western University/COMP
Denver, CO
Diplomate, American Board of Clinical Lipidology

The Southwest Chapter of the National Lipid Association (SWLA) established three primary goals we planned to achieve at the NLA Scientific Sessions last June in Chicago.

1. Education: Promotion of lipid journal clubs in all of the states in the chapter
2. Introduction to the legislative arena: Improving patient’s health through political advocacy
3. Completion of the SWLA LipidSpin: Part education, part inspiration

Education
On Sept. 19, 2015, SWLA partnered with the Preventive Cardiovascular Nurses Association (PCNA) and co-sponsored a symposium at their 2015 Fall Lecture Series in Denver. SWLA board member Judy Collins, MSN, CLS, presented an update on “Statins and Beyond: New Tools for the Challenging Patient.”

I enjoyed the privilege of attending a Lipid Journal Club of SWLA Vice President Kari Uusinarkaus, MD, FNLA. Dr. Uusinarkaus has been regularly hosting these educational dinner programs in Colorado Springs, Colo., for the last several years. On Sept. 29, 2015, SWLA’s immediate past president Kris Vijay MD, FNLA, was the featured lecturer and lent his extensive expertise to a moderate and spirited educational discussion of current lipid therapy.

James Falko, MD, FNLA, joined board member Joel Marrs, PharmD, FNLA, and preventive cardiology fellow Reynaria Pitts, MD, to present an education symposium to the Colorado Society of Osteopathic Medicine (CSOM) with Dr. Haffey serving as the moderator. More than 75 physicians and healthcare providers were treated to a “question and answer” format of journal club to update community physicians and medical students on the advances in clinical lipidology.

Legislation
The second area of interest that the SWLA Chapter identified as a targeted goal was to educate the SWLA membership to the concept of political advocacy. We initiated the process by offering a scholarship for an interested SWLA member to attend the American College of Cardiology (ACC) Legislative Conference with Dr. Haffey. Elizabeth Jackson, CNS, CLS, a current member of the NLA board, was chosen to represent SWLA. Elizabeth and I were inspired, and had an opportunity to meet with the keynote speaker: Fox News contributor and Pulitzer Prize awardee Charles Krauthammer, MD. We both took inspiration from the keynote address, as he had to overcome so many significant obstacles in his own life to achieve the success he enjoys today.

Dr. Krauthammer impressed both of us with his editorial comments on the ramifications of the presidential election cycle, as well as what effect the process will ultimately have on the medical profession. His anecdotes and personal

Discuss this article at www.lipid.org/lipidspin
stories will continue to inspire us for many years. The ACC conference provided insight into the legislative process and provided a valuable primer that we can expand in the coming years to assist the NLA in achieving our legislative goals.

We are a relatively small organization, but highly dedicated to the concept of enlightening our patient’s health through our devotion to educating members on how to deliver the “best practice” model. We are committed to cooperating with like-minded patient-centered organizations.

We have, and will continue, to reach out to our associated organizations such as the PCNA, CSOM, as well as the Colorado Chapter of the ACC. We will continue to encourage SWLA members to participate in the prevention section of the ACC as we strive to spread the word of the importance of the NLA. We are also committed to sharing the goals of the Million Hearts® program because we too share in their dream to achieve the goal of reduction of cardiovascular events by 1 million over the next three years through the use of evidence-based clinical practices and education.

**LipidSpin**
We have invited M. Eugene Sherman, MD, FACC, to contribute an article in this issue (see page 7) devoted to political advocacy for medical professionals. Serving as the chairman of the ACC Advocacy and PAC, there is no one that is his equal in the field of influencing and educating our legislators. The SWLA chapter has also asked Janet Wright, MD, FACC, medical director of the Million Hearts®, to contribute her vast expertise with this exciting prevention program.

Our SWLA chapter goal is to continue to spread the reputation and expertise of the NLA by engaging preventive organizations in educational events and encouraging education for our members by actively promoting “Lipid Journal Clubs.”

We have had multiple conference calls to update our efforts in SWLA and to promote member engagement. We look forward to being the host chapter for the 2016 NLA Scientific Sessions in New Orleans.

For SWLA, there isn’t a more exciting time than now to be working in the field of clinical lipidology. Fueled by ongoing trials, inspired by the excitement in the area of guidelines, and with the creation of new educational products, advanced diagnostic techniques, and the ability of our patients to access information through the lipid.org, the NLA’s ability to deliver education to its members is second to none. We can also look forward to the excellent educational offerings both in live meetings and on our newly updated website that the NLA offers to its members.

I wish to extend my thanks to the other members of the SWLA board without whose support none of these efforts would be possible. I also would like to extend my thanks to our partner organizations such as the PCNA, CSOM, ACC, and Million Hearts® initiative and look forward to continuing a long and educationally fruitful relationships with them.

![L-R Current SWLA President Thomas Haffey, DO, joins President-Elect Kari Uusinarkaus, MD, and Immediate Past President Krishnaswami Vijayaraghavan, MD, at the Lipid Journal Club in Colorado Springs, CO.](image-url)
A passionate supporter of the National Lipid Association (NLA) and an advocate of clinical lipidology, Elizabeth (Beth) Jackson is a woman of many talents. Jackson works as a clinical nurse specialist and clinical lipid specialist with Edward R. Chafizadeh, MD, at CardioTexas in Austin, Texas. Some states do not work with clinical nurse specialists in the same capacity, but Jackson’s role is an advanced practice provider specializing in cardiology and lipidology. Jackson sees both inpatients (consults and follow-ups) and outpatients, so her patient care extends from preventive cardiology to post-surgical patients.

So what is a regular work day like for her? Well, that depends on the week. Jackson says that she rotates from office to hospital on a somewhat irregular schedule, so there is no work day that is the same as another.

“I see anywhere from 15 to sometimes over 30 patients in the clinic,” says Jackson, “and we follow our existing patients when hospitalized. We rotate with other providers to cover the hospital by week and on weekends. When I am on for hospital coverage, I see existing patients and any consults, plus cover both exercise and nuclear stress testing.”

When asked about the best part of her job, Jackson said: “Learning something new every day and the people I get to surround myself with — both patients and coworkers. My colleagues, from the office and nursing staff, to the advanced practice providers and physicians, are top of the line and we work as a team. All of us wish to elevate the level of comprehensive care for our patients, which is challenging as we have a very large patient population!”

Jackson says she and her fellow staff members strive to create an environment not only for the best care for their patients, but one that facilitates learning and embracing the most up-to-date research findings and medical treatment options. In addition to her day-to-day work, Jackson went on a medical mission trip to Haiti in 2012 — two years after the earthquake struck. There, she did clinics at various places around the area affected by the earthquake and met some amazing survivors.

Jackson says that she would like to see greater awareness about the field of lipidology, and for others to grasp the understanding that there is no “cap” on lipidology. She says that prevention strategies and effectively achieving plaque regression in a way that offers minimal side effects and positive outcomes is the ultimate goal. She hopes that others gain a better understanding of the interplay of the multiple mechanisms affecting plaque development and progression.

Jackson first became interested in lipidology upon attending Preventive Cardiovascular Nurses Association (PCNA) meetings back in the 1990s, and found herself loving the science more than anything else. As someone with significantly high cholesterol in the family, Jackson focused a great deal of attention toward understanding lipid metabolism. Jackson went on to say that: “One of the best things that ever happened for my professional career was getting involved with the National Lipid Association. My interest has never faded and there is always something new to learn.”

Jackson’s journey with the NLA began when she was recommended by the PCNA as a candidate for the Southwest Lipid Association board in 2006. Jackson started there and didn’t look back.
Serving as President of SWLA surely helped build these relationships and facilitated Jackson’s understanding of the organization. Jackson said that becoming a certified clinical lipid specialist has opened so many doors, and has catapulted her career by providing her with speaking roles, consultations, and patient referrals. She looks forward to NLA meetings every year for the educational and networking opportunities.

“The education offered within the NLA is endless,” she says.

When Jackson is not in the clinic or the office, she is spending time with her two teenage daughters, her supportive husband, and her pets (three dogs and a cat). She says her family loves traveling and watching movies. Their favorite form of exercise or recreation is swimming — something that Jackson did competitively as a member of the Texas A&M University Swimming Team. Jackson went on to say that Austin is a great city to raise a family and to do all things outdoors. You can find her and her family on their next adventure in 2016 — rock climbing!

Written by Membership and Marketing Coordinator Nichole Vanderpool.
Register Today for the 2016 Spring CLU

Registration is still open for the National Lipid Association’s Spring Clinical Lipid Update March 18–20, 2016, at the Omni Hotel in San Diego. Don’t miss featured sessions on: PCSK9 Inhibition: A New Paradigm in LDL-C Reduction, New Advances in the Treatment of Familial Hypercholesterolemia, Debates in Lifestyle Therapy, and many more. Make sure to check lipid.org/springclu for updated agenda information and registration.

Pay Your Dues for 2016

The 2016 NLA dues statements and membership cards have been mailed. In addition to paying your 2016 dues, this is a great opportunity to donate to the Foundation of the NLA. To pay your dues online, visit lipid.org/dues. For more information or questions regarding dues, contact Membership Manager Britney Caldwell at bcaldwell@lipid.org.

Call for Honors and Awards Nominations

The NLA Honors and Awards nomination window is now open. The deadline to nominate is Monday, February 29, 2016. The NLA has established several types of recognition: Fellow of the NLA, NLA Distinguished Achievement Award, and NLA Honorary Lifetime Membership Award. To view the awards criteria and requirements or to submit a nomination, visit lipid.org/awards.

NLA and Chapter Board Nominations Now Open

The NLA is now accepting nominations for Officer (President-Elect, Treasurer, and Secretary) and At-Large positions for the NLA Board of Directors and Chapter Boards of Directors. The bylaws, available at lipid.org/about/bylaws, describe the nomination process for both the NLA and its Chapters accordingly. The deadline to submit nominations is Friday, March 4. If you have questions, email Robert Talbert, Esq., at rtalbert@lipid.org. To submit an NLA or Chapter Board nomination, visit lipid.org/about/committees/nominate-board.

NLA Releases Annual Summary of Clinical Lipidology 2016

The second edition of the Annual Summary of Clinical Lipidology was recently released in the Journal of Clinical Lipidology. This Annual Summary is intended to be a “living document,” with future annual updates that will be based on emerging science, clinical considerations, and new NLA position and consensus statements.

The goal is to provide clinicians an ongoing resource that translates the latest advances in medical science toward the evaluation and treatment of patients with dyslipidemia. To read the 2016 edition, visit: lipidjournal.com.

ICD 10 Reference Sheet Now Available

Ralph La Forge, MSc, CLS, FNLA, chair of the Lipid Clinic Operations Committee, created a quick reference sheet of commonly used lipid-centric ICD-10 codes. The sheet highlights some differences between ICD-10 and the old ICD-9 codes. The sheet is designed to help clinicians at level I or level II lipid clinics with most of the key lipid-centric ICD-10 codes. The sheet can be found on lipid.org under Practice & Policy tab and in the Operations Section.

NLA Online Enduring Educational Activities

The NLA partners with various medical education providers to offer many free, online educational opportunities to its members. These include programs on how to lower CVD risk, how to manage lipids beyond statins, and much more. These activities are free of charge and can be completed at your own pace and from your iPad or Android devise. To access these activities, visit lipid.org/education/online/other.

Certification in Clinical Lipidology: Spring Testing Window

The spring testing window for lipid certification will be April 3, 2016–May 14, 2016. Application materials for the American Board of Clinical Lipidology (ABCL) certification and the Accreditation
Get Involved in NLA’s Trainee Travel Grant Program
Are you a fellow-in-training who has a focus in lipid management? Learn about the NLA’s Trainee Travel Grant Program! Fifteen grants are available for the NLA Lipid Academy Courses, each including a $500 travel grant and complimentary attendance for the Lipid Academy — plus NLA membership for lipid focused fellows-in-training is always complimentary. Due to the popularity of Travel Grant Program, please act quickly. For more information, contact Amanda East at aeast@lipid.org. For additional trainee opportunities, visit lipid.org/education/fellows.

Stay Current on NLA News by Following us on Social Media
Are you on Facebook, Twitter, LinkedIn or Instagram? If so, make sure you’re following the National Lipid Association on all of its social media accounts. Doing so will ensure that you’re staying current on all of the NLA’s latest happenings. Like us on Facebook at: facebook.com/nationallipid. Follow us on Twitter at: twitter.com/nationallipid. Follow us on LinkedIn: linkedin.com/company/national-lipid-association. Follow us on Instagram by searching the username: nationallipid.

NLA to Introduce Mobile Meeting App
The National Lipid Association will be providing an app with onsite updates and educational content for attendees at the NLA’s upcoming meetings. Please check lipid.org for information coming soon about how to download the app in preparation for the upcoming Spring CLU in San Diego. Make sure to bring your portable device, because we will no longer be providing a printed syllabus. The app is compatible with laptops, tablets, and mobile devices.

Council for Clinical Lipidology (ACCL) Clinical Lipid Specialist certification are due Friday, March 25, 2016! For more information regarding the ABCL requirements, please visit lipidboard.org. For more information regarding the ACCL-CLS certification, please visit lipidspecialist.org.
2016 National Lipid Association
Clinical Lipid Update—Spring
Hosted by the Pacific and Midwest Chapters
March 18–20, 2016
Omni San Diego Hotel
San Diego, CA
lipid.org/springclu

2016 National Lipid Association
Clinical Lipid Update—Fall
Hosted by the Southeast and Northeast Chapters
August 26–28, 2016
Omni Amelia Island Plantation Resort
Amelia Island, FL
lipid.org/fallclu

2016 National Lipid Association
Scientific Sessions
Hosted by the Southwest Chapter
May 19–22, 2016
Hyatt Regency New Orleans
New Orleans, LA
lipid.org/scientificsessions

NLA Events Calendar

Lipid Academy
March 17–18, 2016
San Diego, CA
May 18–19, 2016
New Orleans, LA
August 25–26, 2016
Amelia Island, FL

Masters in Lipidology
March 17–18, 2016
San Diego, CA
May 18–19, 2016
New Orleans, LA
August 25–26, 2016
Amelia Island, FL

NLA Coding Course
March 17, 2016
San Diego, CA
May 18, 2016
New Orleans, LA
Happy New Year from the Foundation of the National Lipid Association! We are hard at work ensuring that 2016 is as much of a success as 2015.

Part of that success stems from the many fundraising events sponsored by the Foundation. We are pleased to announce the first Foundation event of the year, which will take place during the Spring Clinical Lipid Update (CLU) in San Diego March 18–20. You can join the foundation on the evening of March 19, for a murder mystery comedy dinner show, designed just for our group. This event will feature hors d’oeuvre, dinner, and dessert, and is within walking distance of the Omni Hotel. Buy your ticket now to ensure you don’t miss this opportunity to laugh, interact, and attempt to solve the mystery with your colleagues! For more information about the event, or to purchase tickets, visit lipid.org/springclu.

The Foundation is also wrapping up its collaboration with Sanofi US, Regeneron Pharmaceuticals Inc., and Cohn & Wolfe in their launching of an unbranded cholesterol awareness campaign, “Cholesterol Counts.” Stay tuned for updates on this very important initiative.

Make sure to keep your eye out for the next edition of the LipidSpin, which will feature the Foundation’s 2015 Annual Report.

As always, thank you for your continued support of the Foundation!
Guest Editorial


Specialty Corner


References


Practical Pearls


**Case Study**


Statins are medications that lower LDL-cholesterol (the bad cholesterol). When there is too much LDL-cholesterol, blockages may form inside the arteries (a type of blood vessel).

- When there are blockages inside the arteries in the heart, the condition is called coronary heart disease. A patient with coronary heart disease may have a heart attack or need coronary artery bypass surgery or coronary stent placement.
- When there are blockages inside the arteries in other parts of the body, the condition is called peripheral artery disease. A patient with peripheral artery disease may have a stroke, transient ischemic attacks, abdominal aortic aneurysm, blockages in the leg arteries, or need a procedure to improve the blood's circulation to certain parts of the body.

Many studies have shown that statins lower a person’s risk for coronary heart disease and peripheral artery disease. Before recommending a statin, your clinician considered your risk factors (blood pressure, blood sugar, weight, family history, age, diet, activity level, tobacco use) and whether or not you already have coronary heart disease or peripheral artery disease.

What can I expect while taking a statin?

Your clinician may ask you to get occasional blood tests to check your cholesterol level. Your clinician may recommend that you eat a heart healthy diet and maintain a certain activity level. Most patients can take statins without any side effects or problems. If you experience any unusual symptoms while you are taking a statin, you should discuss it with your clinician.

I have heard that statins may cause problems with my _________________.

- **Muscles:** Most people do not have any problems with their muscles while taking a statin. Tell your clinician if you have any unusual muscle pain, tenderness, or weakness. Muscle symptoms may be caused by many different things, including an increase in your activity level, illness, or other medical conditions. Your clinician may recommend that you stop taking the statin for a short period of time. Your clinician may also ask you to get some blood tests to see if the muscle symptoms could be caused by something else. For instance, low vitamin D levels or low thyroid levels may cause muscle aches. When these conditions are treated appropriately, the muscle aches may go away. Your clinician may ask about other medications or herbal remedies you are taking; some medications and herbal remedies increase the risk of muscle symptoms when used with statins.

If you think you cannot tolerate a certain statin or dose of statin, you may be able to take a different statin or dose without any problems. There are seven statins available in the U.S., and each has different characteristics. For instance, some statins stay in the body longer than others. Some statins may interact with other medications or herbal remedies that you are taking. The clinician will consider all of these things when recommending a statin and dose for you. If you have bothersome side effects with a certain statin or dose, the clinician may recommend that you take the same statin at a lower dose, a different statin, or a non-daily dose of a statin. You may need to try several different statin regimens to find the best one for you. Remember, the reason you are taking a statin is to decrease your risk of having complications from coronary heart disease and peripheral artery disease.

- **Blood sugar:** Some statin doses may increase the risk of high blood sugar and type 2 diabetes in patients who already have risk factors for diabetes (slightly elevated blood sugar; overweight; diet high in fat, starch, simple sugar; low activity levels). It is very important for patients with risk factors for diabetes to eat a heart healthy diet, exercise at least 30 to 40 minutes, four to five days per week, and lose weight to decrease their risk of developing diabetes.
- **Liver:** It is rare for someone to have problems with his or her liver while taking a statin. Tell your clinician if you experience yellowing of the skin or whites of your eyes or unusual nausea, vomiting, or extreme tiredness.
- **Memory:** There is no clear evidence that statins affect memory. If you report memory problems while taking a statin, your clinician should consider all possible causes, including other medications (e.g., sleep aides, pain medicine, antihistamines) and medical conditions (e.g., depression, anxiety, sleep apnea) that affect memory.

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INSTRUMENTAL APPROACHES IN LIPOIDOLOGY

COMPLEX SCIENCE IN THE BIG EASY

Venue: Hyatt Regency
601 Loyola Avenue
New Orleans, Louisiana, USA, 70113

Meeting Room Rate: $229/night++
Room Reservation Cut-Off Date: April 26, 2016

For reservations call 1-800-633-7313 and ask for the National Lipid Association room rate.

For more information visit: lipid.org/sessions