Omega-3 Mechanisms of CVD Reduction

The mechanisms by which omega-3 FA (i.e., EPA and DHA) reduce risk for CHD events are not entirely clear. At doses that have been reported to reduce risk for sudden cardiac death [i.e., around 850 mg/day in the GISSI Prevenzione study (3)], no effects on serum lipids have been observed. The dose of EPA+DHA required to elicit significant triglyceride lowering is 3-4 g/day (4). Kinetic studies have shown that omega-3 FA slow the release of VLDL particles into the plasma (5), and our work has suggested that enhanced TG lipolysis may also play a role (6,7). The effects of these agents on HDL-C are inconsistent, but, as with other triglyceride-lowering agents like fibrates, LDL-C may increase as the triglycerides fall. The molecular (or even cellular) bases for the effects of omega-3 FA on lipid metabolism in humans are not yet known with certainty.

Similarly, although relatively large doses of omega-3 FA mildly inhibit platelet function (8), the effects of approximately 1g of EPA+DHA per day, especially in patients already taking aspirin, is not known but could theoretically play a role. Studies in animal models and in cultured cells have suggested that omega-3 FA have membrane-active effects that reduce the susceptibility of the myocardium to develop malignant arrhythmias in the setting of ischemia-reperfusion (9,10). They appear to inhibit both sodium and calcium channel function so as to maintain electrical stability during ischemic stress. In addition, omega-3 FA may also have direct effects on the autonomic nervous system, possibly by increasing vagal tone (11). Such an effect would be expected to reduce heart rate, and therefore, risk for sudden cardiac arrest.

Clinical Insights continued on page 3
The Year Ahead

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The National Lipid Association (NLA) will be entering its fourth year of existence in November 2005. Conceived as a national organization by the leaders of the Southeast Lipid Association (SELA), the NLA has made great strides towards membership expansion and providing education for all healthcare professionals interested in lipids/lipoproteins, vascular biology and preventive cardiology. As I enter my year as the president of NLA, following in the large footsteps of W. Virgil Brown, MD, and John Guyton, MD, I would like to take a moment to share my thoughts of our organization from the perspective of not only where we have been but also where we are headed.

Educational Focus: Continuous Improvement of Our Programs
Designing and implementing a wide array of continuing education activities for our members is an ambitious task. By offering a diversity of regional programs, self-assessment programs, and lipid-focused courses, we have made great strides providing basic, advanced and board review courses in just a matter of three short years. Our vision is to expand our educational offerings through interactive learning and web-based strategies that not only address educational needs but also provide tools for each member to evaluate and measure their own performance in comparison to established guidelines and patient outcomes. The NLA has made some key alliances and in the coming years we will have exciting activities to offer that meet or exceed board certification practice improvement requirements.

Certification is the Future
The establishment of the American Board of Clinical Lipidology (ABCL) and the first certifying examination for physicians specializing in Lipidology marks a significant milestone in the development of our own subspecialty. As an independent sister organization, formed by the NLA but not part of the NLA (as many believe), the ABCL will hold its first formal examination during the pre-meeting period of the American Heart Association conference on November 12, 2005 in Dallas, Texas. The NLA and other affiliated organizations have recognized the importance of credentialing and certifying our subspecialty as we pursue the promises of cardiovascular disease prevention.

All NLA programs are designed to meet the requirements of the ABCL credentialing criteria. At the present time, the certification program is only offered to physicians, but it is our understanding that exams will be offered for the other medical disciplines within our ranks. The NLA is working with other organizations, such as the Preventive Cardiovascular Nurses Association (PCNA) to shepherd the development of training programs for these new certification programs as they are developed.

Internal Reorganization and Expansion
The NLA continues to expand in size and scope at a rapid pace, achieving a 65% increase in membership in one year. This growth can be attributed in part to the addition of the Northeast Lipid Association (NELA) in January 2005 as well as to the recruitment efforts of our Membership Committee, chaired by Dr. John “Robin” Crouse, and the active involvement of members of the chapters. Already the Southwest chapter, to be called the Southwest Lipid Association (SWLA), has an organizing committee to plan its inaugural meeting, which is set for February 10-12, 2006 at the Hyatt Regency Hill Country Resort in San Antonio, Texas. This leaves the Pacific Lipid Association to be formed and we have a great deal of interest from members to quickly move this forward.

This rapid expansion in 2005 has also brought reorganization, as the chapters became affiliates of the NLA instead of independent organizations. This new organizational structure will provide more unity in our mission and goals, enhanced sharing of resources, and alignment of educational programs that will be offered throughout the country, instead of only in a region. Rest assured, all chapters will continue to be active and will offer their own education programs and services tailored specifically to the unique needs of the region.

In 2006, the Northeast Lipid Association (NELA) will partner with the National Lipid Association to host our Annual Scientific Sessions to be held at the Seaport Hotel in Boston, Massachusetts, April 7-9, 2006, and the Southeast Lipid Association (SELA) will host its 9th Annual Forum in Amelia Island, Florida, at the Amelia Island Plantation, August 11-13, 2006. The Midwest Lipid Association (MWLA) 3rd Annual Forum will take place October 20-22, 2006 at The Fairmont Hotel in Kansas City, Missouri, and preliminary plans are in progress for an Inaugural Forum for the Pacific Lipid Association (PLA) in late 2006 or early 2007.

Tackling Issues in the Public Domain
Remarkably, the NLA has not been shy to tackle controversial issues. The appropriateness of over-the-counter statin therapies was addressed in 2004 and early 2005. This November, the NLA will release an extremely comprehensive peer-reviewed Task Force report on the safety of statin drugs, alone and in combination therapies. In 2006, we plan to tackle the enormous task of reviewing other lipid regulating agents, supplements and nutriceuticals to determine their safety and value to patients.

Many ask if we should do this, and I believe the answer is yes. We have been careful to insist upon an independent analysis of any issue with the full disclosure of funding sources and the disclosure of the interests of our leadership and committee members. The integrity of our reports and the reputation of the NLA, its leadership and members, remains a critical priority.

A Success Story
Given its short history, limited resources and small staff, the success of the NLA as an organization is impressive. All this activity is directly attributable to the dedication of the individual members, represented by the Officers and the Board of Directors of the NLA and each Chapter, who volunteer their time and expertise to important committees (such as CME, communications, membership and consumer affairs), as well as to the unrestricted educational grants so generously donated by our commercial supporters.

I am humbled to be a part of the NLA and its exceptional leadership, and look forward to the year and success in the decade ahead.
Two Doses, Two Endpoints

Omega-3 FA can be used to reduce serum triglyceride levels at doses of 3-4 g/dl (4:12), or they may be able to reduce risk for sudden cardiac death (especially in post-MI patients) at a smaller concentration of 1 g of EPA+DHA per day. The latter dose will not affect the serum lipid profile (3), and whether the former dose will reduce risk for fatal coronary events has not been rigorously tested. Using standard fish oil capsules (most of which contain about 300 mg of EPA+DHA in 1g of fish oil) to lower serum triglycerides, would require the intake of 10-13 capsules per day. However, this dose can be achieved with only 4 capsules of Omacor® (Reliant Pharmaceuticals, Inc.). This FDA-approved omega-3 product, which contains 850 mg of EPA+DHA/g capsule, will be available in the US by prescription in October 2005.

N-6 FA Do Not Block the Benefits of N-3 FA

Data from the Health Professionals Follow-Up Study (HPFS)(13) was used to examine the question, “does a high intake of n-6 FA (notably linoleic acid) negate the beneficial effects of either the short chain (α-linolenic acid) or the long chain (eicosapentaenoic and docosahexaenoic acids, EPA+DHA) on CHD risk?” This is a legitimate question since linoleic and α-linolenic acids compete for the same desaturation/elongation systems for conversion to their respective longer-chain metabolites. The HPFS enrolled 45,722 men and followed them for CHD events over a 14-year period. In that time, 218 experienced sudden death (SD), 1521 had a non-fatal MI, and a total of 2306 had some CHD event. Using intakes of linoleic, ALA, EPA and DHA estimated from diet questionnaires, the investigators found that when EPA+DHA intakes were below the median (250 mg/d), above-average (11.2 g/d) linoleic acid intakes (compared to below-average intakes) tended to reduce risk for Sudden Death (OR 0.76 (95% CI 0.52-1.11) but higher intakes of linoleic acid had no effect on risk for non-fatal MI or total CHD events. In those subjects with background EPA+DHA intakes above the median, risk for Sudden Death was significantly reduced relative to those with less than median EPA+DHA intakes by 48%, and that the n-6 FA intake had no influence on risk. For non-fatal MI or total CHD events, there was no significant effect (good or bad) of either n-6 FA or EPA+DHA.

For ALA intakes above the median (1 g/d) vs. below, there was no significant effect on risk for Sudden Death, but there were significant 11%-12% reductions in risk for the other two endpoints. These effects of high vs low ALA were not affected by high vs low n-6 FA intakes. Finally, they examined the interaction of dietary ALA and EPA+DHA on risk. When the EPA+DHA intake was over 100 mg/d (about the average in the US), then a 1-g/d increase in the intake of ALA had no effect on any CHD endpoint. However, when EPA+DHA intake was below 100 mg/d, a 1-g increase in ALA was associated with a 50% reduction in risk for non-fatal MI and total CHD events. These findings support the view that a relatively high n-6 FA intake does not diminish the beneficial effects of either ALA or EPA+DHA on CHD risk. Indeed, a higher n-6 intake appeared to reduce risk for SD when diets were relatively deficient in the low long-chain n-3 FA. Secondly, it appeared that ALA may only be cardioprotective in the context of a low (<100 mg/d) EPA+DHA intake. Therefore, to reduce risk for CHD, increasing intakes of the long-chain n-3 FA appears to be the most important step, and ALA may only be helpful when EPA+DHA intakes are particularly low. High intakes of n-6 FA do not appear to be detrimental in any setting.

Consumption of N-3 Fatty Acid-Rich Fish Reduces Risk for Stroke

The role of n-3 FA intakes on the risk for stroke in older subjects was also recently described by Mozaffarian and colleagues(14). They examined the relationships between the intakes of higher n-3 FA fish (tuna and other non-fried fish) vs. lower n-3 FA fish (fried fish or fish sandwiches) and both hemorrhagic and ischemic stroke in the Cardiovascular Health Study (CHS). The CHS is a cohort study of 4775 men and women over the age of 64 who were free of clinical CVD at baseline. Participants were followed for stroke endpoints for 12 years. In multi-variable adjusted risk models, there was an intake-dependent reduction in risk for ischemic stroke with increasing intakes of the higher n-3 FA fish (p for trend = 0.03) with risk in the highest intake group (i.e., those reporting 5 or more fish servings per week vs. less than 1 per month) being reduced by 28% (95% CI 0.51-1.03). There was no relationship between intake of these types of fish and hemorrhagic stroke. For increasing intakes of the lower n-3 fish, there was an increasing risk for ischemic stroke (p for trend = 0.01), with risk in the highest intake group (i.e., those reporting more than one serving per week vs. less than 1 time per month) being elevated by 39% (95% CI 1.081-1.79). This study expands upon Mozaffarian’s previous report that higher intakes n-3 FA-rich fish were associated with reduced risk for CHD events, especially arrhythmic death(15). These findings suggest that concerns regarding the possible adverse health consequences of the consumption of large amounts of fish possibly contaminated with mercury(16) or pesticide-herbicide residues(17) may be overblown.

Statins and N-3 Fatty Acids Prolong Life

The ultimate reason for treating patients with anti- lipidemic agents or diets is to prolong their lives. It was, in fact, the demonstration in the 4S trial(18) that treatment of CHD patients with statins (simvastatin) would actually reduce total mortality rates that opened the floodgates for statin use. After all, if lipid-lowering therapy did not prolong life but simply shifted the cause of death from cardiovascular to non-cardiovascular diseases, then there was little compelling reason to treat. A recent meta-analysis examined the effects of a variety of anti- lipidemic agents and diets on total mortality. Studer et al.(19) included 97 studies with over 137,000 patients receiving treatment for lipid disorders vs. a similar number of controls in which risk for death from any cause was reported. There were 35 trials with statins, 7 with fibrates, 8 with bile acid binding resins, 14 with n-3 FA and 18 studying the effects of global dietary changes. The authors found that only two of these interventions were associated with significant reductions in total mortality: statins (risk ratio 0.87, 95% CI 0.81-0.94) and n-3 FA (risk ratio 0.77, 95% CI 0.63-0.94).

There are, however, some very positive studies that, in my opinion, should not have been included in the n-3 FA group. Two were not strictly n-3 FA trials but global dietary interventions(20;21) of which one component was ALA, and one(22) whose validity has been called into question(23). How reclassification/elimination of these studies would have affected the results is not clear. What is evident, however, is that increasing the intake of long-chain n-3 FA is a safe and inexpensive way to significantly reduce risk for CHD, especially sudden cardiac death.
High Fish Intake Slows CAD Progression in Women

An unsettled issue in the n-3 FA – CHD literature is the extent to which these FA impact the atherosclerotic process itself vs. altering the susceptibility of the myocardium to arrhythmias. Evidence for an anti-atherosclerotic effect is mixed, with some reporting no effect of n-3 FA on disease progression in native vessels(24) or on restenosis post-PTCA(25), and others finding these FA to be effective at slowing restenosis(26) and stabilizing (carotid) plaque(27). The effects of n-3 FA in women have also received relatively little attention. Hence, the recent report from the Estrogen Replacement and Atherosclerosis (ERA) study is most welcomed. The ERA was a randomized, double-blind, placebo-controlled trial of hormone replacement therapy in women with documented (by angiography) coronary artery disease (CAD)(28). Women were assigned to estrogen alone, estrogen+progesterone or placebo and the rate of angiographic progression of CAD was followed by repeat angiogram at 3.2 years. The effects of the reported fish intake at baseline (less than 2 vs. at least 2 servings per week) on angiographic progression were recently published(29). CAD progressed more slowly in those women consuming more fish than less after adjusting for 19 relevant covariates (p=0.02). Similarly, there were fewer women with new lesions in the higher fish intake group than in the lower (21% vs. 34%, p=0.03). When fish intake was sub-divided into tuna/dark meat fish vs. light meat fish (the former typically containing more n-3 FA), the association with slowed progression was stronger for dark meat fish. Finally, CAD progression was more clearly evident in the 42% of the women who had diabetes than in those who did not. Hence, atherosclerosis itself may be slowed by dietary n-3 FA.

N-3 FA and Atrial Fibrillation

N-3 Rich Fish and Risk for Developing Atrial Fibrillation

Although most of the attention has focused on the effects of n-3 FA on ventricular arrhythmias, some investigators have begun to explore the relationship between these FA and atrial dysrhythmias, in particular atrial fibrillation. Atrial fibrillation is the most common cardiac arrhythmia and is associated with significant morbidity including chronic fatigue and increased risk for stroke. Mozaffarian et al.(30) reported in 2004 that participants in the Cardiovascular Heart Study (CHS) who consumed the greatest amounts of high n-3 FA fish had a significantly reduced risk of developing of developing atrial fibrillation over 12 years of follow up. In an effort to corroborate the CHS findings, Frost and Vestergaard(31) examined the relationship between incident atrial fibrillation/flutter over a 5.7 year period in the participants in the Danish Diet, Cancer and Health Study. This prospective cohort study included data from nearly 48,000 men and women from whom baseline dietary information had been gleaned. Atrial fibrillation/flutter was detected based on hospital admissions (and outpatient visits) and was confirmed by the appropriate coding (ICD, 2% to 76%). Differences between the results of these studies, one conducted in the US and the other in Denmark, may be explained by differences in the methods of diagnosing atrial fibrillation, the completeness of follow up, the background diets and patient populations themselves.

Atrial Fibrillation Post CABG

Atrial fibrillation is a common and potentially dangerous side effect of coronary artery bypass grafting (CABG). Calo and colleagues from Rome explored the hypothesis that pretreatment of CABG patients with n-3 FA would reduce the incidence of post-CABG atrial fibrillation(32). Patients scheduled for elective CABG (n=160) were randomized to usual care or to n-3 FA supplementation (1.7 g/day of EPA+DHA ethyl esters) for at least 5 days prior to surgery. Supplementation continued for the duration of their hospitalization. Atrial fibrillation developed in 33% of patients in the usual care group and 15% of those in the n-3 FA group (Figure 2; p=0.013), and the mean duration of atrial fibrillation was 15.5 hours vs. 24 hours, respectively (p=0.13). Owing to the reduced incidence of atrial fibrillation in the n-3 FA treated group, the mean length of stay was reduced by 1 day (8.2 to 7.3 days, p=0.017). These effects were observed despite substantial (and equivalent between group) use of perioperative medications. The authors noted that the effects observed with n-3 FA supplementation were similar to those produced by drugs such as amiodarone, beta-blockers and sotalol, but n-3 FA are safer and have no known drug interactions. Clearly, a larger, placebo-controlled trial to confirm these open-label findings is needed.

Conclusion

Most of the recent advances in the field of omega-3 FA and clinical CHD have come from diet questionnaire-based epidemiological studies that have continued to link fish intake with beneficial outcomes. These studies suggested that increased fish oil intake may reduce risk for sudden cardiac death, stroke and CAD progression. The effects on atrial fibrillation are not certain due to major discrepancies in trial data. In addition, it appears that increasing intakes of the long-chain n-3 FA EPA+DHA is the best overall strategy for CHD risk reduction, that increasing the α-linolenic acid intake may be a secondary goal, and that reducing n-6 FA intakes may not only be unnecessary but possibly ill-advised. Future studies clearly linking CHD benefits to EPA and DHA supplementation are needed to determine the proper doses and patient populations most likely to benefit from these essential FA.

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Hurricane Katrina Puts NLA Members on the Front Lines of Patient Care

By Kathryn St. John
NLA Public Relations Consultant

In the first days and the weeks following Hurricane Katrina - the largest, most devastating natural disaster in decades - NLA members found themselves on the front lines of emergency patient care. “In the midst of the most horrific circumstances, our doctors were able to provide medical relief to thousands of evacuees,” said NLA President Peter Jones. “I am very proud of our members’ contributions to the disaster relief effort.”

The Height of the Storm’s Aftermath
The firsthand accounts by physicians who assisted patients and medical staff at the evacuation centers reveal the unprecedented problems and organizational challenges a disaster of this magnitude creates.

In the immediate aftermath, physicians reported chaos. Many were stunned by the disorder that initially existed during the early hours after the storm and the first days following the evacuation. Some reported that there was no process for organizing the local volunteer healthcare professionals or those who later came to the region to help. There was no system in place to transport medical or survival supplies to the designated shelters or evacuation centers in the first hours or days, and there were no stockpiled supplies at most of those locations.

The lack of basic healthcare provisions, including medicines, sanitary supplies, and extremely limited amounts of food and water, complicated an already dangerous situation. How do doctors provide critical care with no electricity, air conditioning, running water or sanitation?

Some physicians reported working 14 consecutive days of 18 to 20-hour shifts, all the while being unsure of their families’ whereabouts. The following are a few snapshots from some NLA members who contributed to the hurricane relief efforts.

Baton Rouge, Louisiana
Dr. David G. Carmouche, of the Baton Rouge Clinic, a multi-specialty facility, lives about 60 miles north and west of New Orleans and he saw the population of his city double overnight. Thousands of patients were taken to Baton Rouge University where the basketball arena was turned into a MASH unit for the critical care patients. They triaged approximately 50,000 patients in the first few days. Additionally, there were two American Red Cross shelters in Baton Rouge, which each housed about 5,000 to 7,000 patients. “For those of us who practice internal medicine, it was stunning. We had more work with a variety of internal medicine issues such as heart failure exacerbations and patients with diabetes who had been without their medicines for 72 hours. You cannot really prepare for something on this scale; the pure numbers of patients was overwhelming. We were running to our clinic pulling samples off the shelves, other doctors were running to their own practices. Houston really stepped up to the plate, Baton Rouge could not have handled much more,” Dr. Carmouche said.

Medicines were in extremely short supply for the first 24 hours or so and then the pharmaceutical companies rushed in much-needed supplies.

“The individual stories were unbelievable…every patient had an incredible story, what you have seen on TV doesn’t even do it justice. Many people had their family members simply die in front of them, unable to help.” Dr. Carmouche said he strongly feels there needs to be a single source for coordinating physician assistance during a disaster. The many nurses, physicians and paramedical support that began to rush in from out of state were greatly appreciated. He said there is no way to thank and demonstrate to those hundreds of people who came in what it meant to have the infusion of resources and skills.

“Our community could not have done the work without the physicians from out of town, in a disaster of this proportion you need physicians from outside. Every physician, every volunteer was appreciated. It took time to get a grasp of what the scope and magnitude of the problems were,” Dr. Carmouche said.

New Orleans 9th Ward
Dr. Keith Ferdinand, who works in Ward 9 of New Orleans, saw the storm’s impact firsthand on his neighbors, his patients and his own family. His Heartbeat Life Center, with three cardiologists and 15 staff members, is in the heart of the hardest impacted area; after the levees broke the area was under 20 feet of water.

“We need to educate our patients about the importance of knowing what medicines they are taking,” Dr. Ferdinand said. “Every one of our patients had a full review and a printed copy of their medicines, their lipid levels and lab values at each assessment. Many of the patients treated after the storm could not articulate their own medicines that they take on a daily basis. This experience has clearly demonstrated for me that we all must aggressively support legislation to implement electronic medical records.”

East Jefferson General Hospital & Kenner “Clinic”
“In the aftermath of Katrina, as the situation at the hospital became more stable, I made the decision that it was time to move to the field. Our hospital was up and running…but the sole hospital in Kenner was closed and therefore its population had no where to go for even its most basic medical needs,” Dr. Madrigal reported. “We went into the sample cabinets of several of our colleagues and removed as much of the samples as we could; next I went to a nursing home that had been evacuated and ‘relocated’ all the medicines and supplies we could find. Armed with these supplies, we approached the Mayor of Kenner and received authorization from him to set up a “walk-in” clinic,” Dr. Madrigal said. “This would have never been accomplished without the enormous support provided by the Kenner Fire Department Volunteers, who responded to our request for assistance in an unprecedented and selfless manner.”

It was later decided that a ‘mobile clinic’ was necessary, Dr. Madrigal reported. This was accomplished by obtaining a large tent, a generator, a large fan, tables, chairs and a trainer to move the medical supplies to where the residents of an apartment complex needed care. Dr. Madrigal praised the Arkansas Air National Guard for providing security and crowd control and the Louisiana National Guard, who provided food and water to distribute at the site. The “walk-in” clinic became the vaccination center for city workers and the general public. About three weeks after the hurricane, they had evaluated 1,500 patients, filled 4,200 prescriptions, vaccinated 828 individuals and distributed almost 1,000 MREs.

“While I have had the honor of leading the effort to undertake this operation, the success of it is a credit to the doctors, nurses, volunteers and National Guardsmen that helped staff and sustain the “walk-in” clinic. Disasters may bring out some bad aspects of human nature, but let me tell you that in the past weeks, I have seen the best side of it more often than not,” Dr. Madrigal said.

Special News continued on page 12
Innovative Approaches to Comprehensive Cardiovascular Disease Risk Reduction: Focus on Therapeutic Lifestyle Changes

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Despite impressive technologic advances in the field of medicine during the 20th century, atherosclerotic cardiovascular disease (CVD) remains the leading cause of death in the United States and most developed countries. Modification of multiple risk factors through a combination of comprehensive lifestyle interventions and appropriate pharmacological therapy is now widely recognized as the cornerstone of initiatives aimed at the primary and secondary prevention of CVD.

Recent studies emphasize the need to intensify efforts aimed at the control of multiple CVD risk factors. To help facilitate this objective, national clinical guidelines advocate a multifactorial lifestyle approach to CVD risk reduction. This approach has been designated “therapeutic lifestyle changes” or “TLC” and includes exercise training together with correct nutrition and other appropriate lifestyle interventions such as cigarette smoking cessation.

Primary care physicians and cardiologists generally work in an intensely busy environment. Typically, physicians do not have the time, infrastructure, or resources to focus adequate attention on certain prevention-related services, especially TLC. Moreover, physicians in the United States receive little or no compensation for the provision of TLC. In view of these and other barriers, it is not surprising that physicians in this country tend to limit most of their attention to acute medical problems presented during office visits, give low priority to preventive interventions in general, and when focusing on CVD risk reduction, prescribe pharmacologic therapy in preference to TLC. Indeed, because of the widespread availability of powerful cardioactive medications, the value of TLC per se in contemporary medical practice is often discounted by physicians, health insurers, and patients.

This article briefly summarizes the findings of the landmark lifestyle intervention trials that refute the commonly held notion among clinicians that TLC is not worth the effort and presents a “case study” of an innovative model for comprehensive lifestyle management and CVD risk reduction that we have successfully integrated into regular medical care.

Landmark Lifestyle Intervention Trials

Overwhelming evidence from a variety of sources, including epidemiological, prospective cohort, and intervention studies, links CVD and most other chronic diseases seen in the world today to physical inactivity, inappropriate diet consumption, and cigarette smoking. Recently, lestra et al. performed a literature search on the effect of the generally agreed upon lifestyle recommendations (Table 1) on mortality in patients with coronary artery disease. Prospective cohort studies and randomized controlled trials of patients with established coronary artery disease were included if they reported all-cause mortality and had at least 6 months of follow-up. Increased physical activity, dietary changes, smoking cessation, and moderate alcohol use were all associated with a statistically significant risk reduction, the magnitude of which was similar to that observed with low-dose aspirin, statins, beta-blockers, and ACE inhibitors after myocardial infarction (Table 2).

In a recent study of ours, 2,390 ethnically diverse men and women with hypertension, hyperlipidemia, and/or impaired fasting glucose or diabetes mellitus and who were not taking medication for these risk factors were evaluated before and after 12 weeks of participation in a community-based comprehensive lifestyle management program. TLC included exercise training, a low fat/cholesterol diet, weight management, smoking cessation, and stress management. Of the participants with an elevated baseline systolic blood pressure, diastolic blood pressure, LDL cholesterol, and/or fasting glucose, 64%, 67%, 11%, and 39%, respectively, achieved the goal value with TLC (without using pharmacotherapeutic agents, Figure 1). Of the patients

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<th>Table 1. Recommendations of Lifestyle and Dietary Factors to Improve Prognosis in Coronary Artery Disease Patients</th>
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<tr>
<td>1. Stop smoking</td>
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<td>2. Engage in moderate intensity physical activity (for &gt;30 minutes on at least 5, but preferably all, days of the week)</td>
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<td>3. If you use alcohol: do so in moderation (maximum 2 alcoholic drinks per day for women and maximum 3 drinks per day for men)</td>
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<td>4. Maintain or attain a healthy body weight (BMI &lt;25 kg/m2); obese patients (BMI &gt;30 kg/m2) should try to lose 10-15% of their current body weight</td>
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<td>5. Limit your saturated fat intake (to a maximum of 10% of daily energy intake) and the intake of trans fatty acids (to a maximum of 1% of daily energy intake)</td>
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<td>6. Consume fish regularly (at least 1 and preferably 2 portions of oily fish per week)</td>
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<td>7. Consume sufficient amounts of fruits and vegetables (&gt;400 g/d)</td>
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<td>8. Use sufficient fiber containing grain products, legumes, and/or nuts (&gt;3 U/d)</td>
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<td>9. Reduce your salt intake (to maximal 2400 mg/day)</td>
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From reference 9.

with a baseline fasting glucose compatible with a diagnosis of diabetes, 37% decreased that value to <126 mg/dl. This study adds to the existing literature by reporting on the effectiveness (i.e., extent to which TLC works in actual practice) rather than on the efficacy (i.e., determining whether TLC can work when administered in a clinical trial) of TLC. Moreover, it should be noted that TLC can generally be implemented less expensively than most medications and, unlike single-drug therapy, favorably affects multiple risk factors. Therefore, these findings also have potentially important policy implications for health care payers, including the federal government, who often do not provide reimbursement for TLC but do provide prescription drug coverage.

Innovative Models for Comprehensive Lifestyle Management and CVD Risk Reduction: A Case Study

Through contact with millions of patients each year, physicians and other health care providers have an opportunity to favorably impact public health by promoting TLC. Clearly, however, innovative approaches are needed to assist physicians in the provision of long-term lifestyle management services to their patients. One example

Therapeutic Lifestyle Changes continued on page 8
of such an approach is a program (called the INTERVENT Lifestyle Management and Cardiovascular Risk Reduction Program) that we have developed, tested and successfully implemented in a variety of clinical and community-based settings in the United States, Canada, and South Africa.11 Outcome data, including results from randomized clinical trials, have documented the clinical- and cost-effectiveness of this approach.10-12

Briefly, the program content is organized into two core sets of services. One set is “mentor-assisted” (involving one-on-one counseling of participants by a non-physician health professional/case manager, called a “mentor”). The other set is an array of individualized “self-help” products, all of which are web-enabled. The programs can be administered in, or from, a variety of physical settings (including physician offices, hospitals, cardiac rehabilitation programs, work sites, and public locations) and via telephone and the Internet. In each of these settings, the program content has been adapted to enhance the applicability to the specific settings and clinical circumstances. Key program steps are as follows:

**Step 1: Participant enrollment.** Typically, patients are referred by their physicians or identified through health risk appraisals or various other referral channels (including, self-referral following marketing of the program to the community, health plan members, or employees). On enrollment, each participant in a mentor-assisted program is assigned to an appropriately trained health professional who serves as the participant’s case manager. Participants in a self-help program are provided instructions for accessing their individualized program via the Internet or mail. Participants often pay to participate in the program themselves (retail pricing currently ranges from approximately $40 for 12 weeks of participation in a web-enabled self-help program to $400 for 1 year of participation in a mentor-assisted program with telephone and Internet counseling). In certain instances, employers and/or health plans pay for program participation (discounted pricing, including capitation, is used when working with employers, health plans, and other groups of program participants).

**Step 2: Initial/intake assessment.** Participants complete a comprehensive medical history and health habits questionnaire, with the option to include biometric measurements (such as, height, weight, waist circumference, blood pressure, fasting serum lipids and lipoproteins, fasting glucose, hemoglobin A1c, C-reactive protein, homocysteine, etc.) and exercise test and other test results, if available. Questionnaires may be completed online via a secure server, in hard copy (“pen and paper”), or via the telephone. The initial assessment evaluates current health status, risk factors for CVD, past medical history, medications, current lifestyle practices, readiness for change, barriers to change, resources for change, and other relevant information.

**Step 3: Goal setting.** Based on the initial assessment, computer-generated individualized short- and long-term goals are set for multiple CVD risk factors and health behaviors in accordance with national clinical guidelines.

**Step 4: Action plan formulation.** Based on the initial assessment, a computer generated individualized action plan is formulated to achieve the short- and long-term goals. The action plan focuses on important health habits (including physical activity/exercise training, nutrition, weight management, tobacco cessation and stress management). In addition to behavior modification, the action plan identifies the need for other self-care activities and physician referrals for prescription medications to optimize CVD risk reduction consistent with national guidelines. Physician letters notify the participants’ physicians of their participation in the program and the CVD risk reduction goals and action plans.

**Step 5: Review/revision of goals and action plan.** For participants in mentor-assisted programs, referring physicians have an opportunity to review, revise, and authenticate the goals and action plan reports for their patients. Using an approach that has been favorably reviewed by the United States’ Department of Health and Human Services, physicians are sometimes compensated for providing this service for their patients. Participants access their goals and action plan reports via program visits, the Internet or mail. Reports are accompanied by an audio explanation, which can be accessed online or via CD. For participants in mentor-assisted programs, mentors review goals and action plans with participants at face-to-face program visits or via the telephone and make revisions, if appropriate. When reviewing reports, mentors are guided by written instructions, referred to as mentor prompt sheets (or lesson plans). If the action plan includes physician referral for consideration of institution or adjustment of prescription medications, the mentor helps facilitate this and documents the outcome of the referral in the program database.

**Step 6: Action plan implementation.** Action plans are implemented using an individualized series of behavior change and education modules, each of which can be read and/or listened to by the participant during a 15-minute or so session. The modules are provided in printed form and via audio recordings, both of which can be accessed via the Internet and via “hard copy” form. The modules are effective in helping modify each participant’s behavior, using single concept learning theory, stages of readiness for change, and other behavior change strategies. Materials and messages are matched with each participant’s stage of readiness for change and personal circumstances, both clinical and otherwise.

<p>| Table 2. Approximate Mortality Reduction Potential of Lifestyle Changes Estimated From Studies in Coronary Artery Disease Patients: Comparison With Preventive Drug Interventions After Myocardial Infarction |
|-------------------|-------------------|</p>
<table>
<thead>
<tr>
<th>Intervention</th>
<th>Mortality Risk Reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-dose aspirin</td>
<td>18%</td>
</tr>
<tr>
<td>Moderate alcohol</td>
<td>20%</td>
</tr>
<tr>
<td>Statins</td>
<td>21%</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>23%</td>
</tr>
<tr>
<td>Physical activity</td>
<td>25%</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>26%</td>
</tr>
<tr>
<td>Smoking cessation</td>
<td>35%</td>
</tr>
<tr>
<td>Combined dietary changes</td>
<td>45%</td>
</tr>
</tbody>
</table>

Adapted from reference 9.
At each program interaction, the participant listens to a CD or web-enabled recording on the specific behavior modification topic, receives the accompanying written educational materials on the specific topic, and, if applicable, meets briefly with his/her program mentor (either physically or telephonically) for further counseling and to update the participant’s individualized lifestyle modification and prevention program. Mentors assist participants in implementing individualized action plans through proactive, structured, one-on-one counseling sessions via face-to-face program visits or prescheduled telephone appointments. With assistance from a web-enabled participant management and tracking database, mentors typically guide participants through approximately 20 modules in the first year of program participation in an individualized, carefully sequenced, structured fashion.

Step 7: Follow-up assessment. After 12 weeks and 1 year of program participation, and at least annually thereafter, participants have an opportunity to complete a follow-up medical history and health habits questionnaire, with the option to include biometric measurements. Questionnaires may be completed online, in hard copy, or via telephone.

Step 8: Progress report and revision of goals/action plan. Based on program participation and the follow-up assessments, participants are provided computer generated reports documenting their progress and updating their goals/action plan. For participants in mentor-assisted programs, progress reports are reviewed at counseling sessions. As with the initial goals and action plan reports, physicians may be asked to review, revise, and sign progress reports for their patients, and in certain instances receive financial compensation for this service. Similarly, if the revised action plan includes physician referral for consideration of institution or adjustment of prescription medications, the mentor helps facilitate this and documents the outcome of the referral in the program database.

Step 9: Maintenance. Participants typically enroll in the program for either 12 weeks or 1 year at a time, but have access to continuing years of mentor-assisted program delivery or to self-help programs. Compliance with scheduled mentoring sessions, lifestyle interventions, and prescribed CVD risk reduction medications is tracked using the web-enabled participant management and tracking database.

Step 10: Outcomes assessment. Using a computerized outcomes analysis system, detailed outcomes reports are generated on a regular basis for specific program locations, individual physicians and groups of physicians, individual mentors, employers, and other groups of program participants. In certain instances, benchmarking is included. To date, the program database has also been used to generate data for approximately 70 published scientific abstracts and/or manuscripts.

It is our belief that the first decade of this new millennium will be remembered as the “decade of CVD prevention.” New and innovative approaches, such as the above “case study,” will be needed to fulfill the potential for improving quality of life and longevity through TLP and other CVD risk reduction interventions.

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REFERENCES
Taking a Pill to Boost HDL Function in Mice


The authors of this study reported that an oral HDL mimetic, D-4F, administered orally together with a statin to a mouse model of atherosclerosis had an anti-atherogenic effect greater than D-4F or the statin alone. The background of this work, [Navab M, Anantharamaiah GM, Reddy ST, Hama S, Hough G, Grijalva VR, Yu N, Ansell BJ, Datta G, Garber DW, Fogelman AM. Apolipoprotein A-I mimetic peptides. Arterioscler Thromb Vasc Biol. 2005 Jul;25(7):1325-31.] relates to the reports made by this group, in that normal HDL is efficient at removing lipid free radicals, lipoperoxides, from LDL and other lipid-containing structures via its apo A-I moiety, rendering them less pro-inflammatory and therefore less proatherogenic. The HDL accumulates these lipoperoxides and becomes more pro-inflammatory in the process. An enzyme carried by HDL, paroxonase, has been shown to degrade the lipoperoxides in HDL, restoring it to its anti-inflammatory status. These authors have previously tested this approach in subjects with coronary heart disease and reported that HDL from these subjects was more pro-inflammatory than normal. This tendency was not obviously related to the level of HDL-cholesterol, which suggests a disparity in at least some individuals between HDL-cholesterol levels and HDL functionality (Ansell BJ, Navab M, Hama S, Kamranpour N, Fonarow G, Hough G, Rahmani S, Mottabedeh R, Dave R, Reddy ST, Fogelman AM. Inflammatory/ anti-inflammatory properties of high-density lipoproteins distinguish patients from control subjects better than high-density lipoprotein cholesterol levels and are favorably affected by simvastatin treatment. Circulation. 2003;108:2751–2756).

The authors then developed small peptides that resembled apo A-I physicochemically and tested them for their ability to bind lipoperoxides and thereby render LDL to be less pro-inflammatory. They found that the number of amino acids with phenylalanine (F) in the peptide had an important effect on the properties of these peptides. They selected the 4F peptide because of its optimal anti-inflammatory properties and its ability to enhance efflux of cholesterol from macrophages. They then re-synthesized 4F with D-amino acids, which are more resistant to intestinal digestive enzymes than the usual L-amino acids, and found that oral D-4F is absorbed intact and can be found in the plasma of animal and human test subjects at concentrations that alter LDL pro-inflammatory activity and increase HDL levels.

Initial testing of this peptide in mouse models of atherosclerosis revealed that D-4F was significantly anti-atherogenic in young animals that had not developed lesions at initiation of the experiment in comparison to the controls. However, no significant effect was seen in older animals that had established lesions at the initiation of the study. They interpreted these results to indicate that the peptide was less effective in producing regression than in preventing progression of lesions. In a proof-of-concept design that anticipates future clinical trials in humans, they then undertook the series of experiments in the report that is referenced at the top of this article. They tested the lowest dose of D-4F that is shown to reduce LDL pro-inflammatory activity with or without a dose of pravastatin and previously not shown to be effective in apo E null mice with established atherosclerotic lesions. They demonstrated that whereas neither D-4F nor pravastatin alone caused any regression of lesions in these extremely hypercholesterolemic mice, the combination reduced lesion size by 62% compared to the untreated controls. This effect was associated with a reduction in the pro-inflammatory properties of LDL from these animals (and in monkeys) and a modest increase in HDL-cholesterol.

These are convincing experimental studies of an anti-atherogenic agent that appears to mimic at least some of the properties of HDL. In many ways they are reminiscent of the work on cholesterol ester transfer protein (CETP) inhibitors, such as torcetrapib, that led to the initiation of ongoing clinical trials with that agent. Perhaps the biggest difference between the two is the fact that the mechanism of action of torcetrapib is well worked out in systems that have a long history of investigation and study, whereas the D-4F studies and the assays used to assess its effects are much more limited. If nothing else, this line of investigation emphasizes that the only convincing approaches to the design of anti-atherogenic drugs that modulate HDL are those in which the action of those agents on HDL functionality and its relationship with atherogenesis are clearly understood. In this respect the authors have been successful.
Ezetimibe: Mechanism of Action and Effects on Serum Lipoproteins

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In a recent addendum to its third Adult Treatment Panel guidelines, the National Cholesterol Education Program promulgated a new set of therapeutic options for LDL-C and non-HDL-C management in patients defined as very high risk, high risk, or moderately high risk (1). Among very high risk patients, recent clinical trials support an LDL-C goal of < 70 mg/dL and a non-HDL-C goal of < 100 mg/dL. Among patients with a 10-yr Framingham risk that exceeds 10%, goals of <100 mg/dL and < 130 mg/dL for LDL-C and non-HDL-C, respectively, are also supported by clinical trial evidence. When treating patients with elevations in other lipoproteins, the concept of “lower is better” is rapidly gaining acceptance among health care providers who treat dyslipidemia. This makes good sense given the fact that cholesterol is one of the most important and potent toxins we are exposed to in our lifetimes. There is also increasing recognition of the need to raise serum HDL levels in patients with low levels of this lipoprotein given its broad-ranging antiatherogenic effects in a variety of experimental models. All practicing clinicians know all too well how challenging it can be to meet these various targets, especially in higher risk patients.

The gastrointestinal tract regulates the extraction and systemic distribution of ingested lipids and steroids. Lipids (phospholipids, triglycerides) and cholesterol esters are hydrolyzed by pancreatic lipases and cholesterol esterase, respectively. The lipids (fatty acids, mono- and diglycerides) and unesterified cholesterol are emulsified with phospholipids by bile salts to form micelles. The cholesterol in these micelles is derived from both biliary and dietary sources. As micelles contact the jejunal brush border, they can interact with a sterol transporter known as Niemann Pick C1-like 1 protein (NPC1L1; 2). NPC1L1 binds cholesterol and phytosterols such as β-sitosterol and campesterol from micelles and transports them into the cytoplasm of enterocytes. The majority of the absorbed cholesterol is then coupled to a free fatty acid to form cholesterol esters in a reaction catalyzed by acyl-coenzyme A:cholesterol acyltransferase.

Under normal conditions, most of the phytosterols and a small percentage of cholesterol is translocated back into the intestinal lumen by a heterocomplex of ATP binding membrane cassette transport proteins, ABCG5/G8. In rare patients, ABCG5/G8 is defective, resulting in excess phytosterol absorption and sitosterolemia. Absorbed free fatty acid not esterified to cholesterol can be reesterified to mono- and diglycerides to yield triglycerides in a reaction catalyzed by acyl-coenzyme A: diglyceride acyltransferase. Cholesterol esters and triglycerides are then combined with apoprotein B48 and phospholipids to form chylomicrons in a reaction catalyzed by microsomal transfer protein. Chylomicrons are released into the mesenteric lymph and conducted into the central circulation via the lymphatic duct.

Ezetimibe is a cholesterol absorption inhibitor that blocks the jejunal uptake of cholesterol by inhibiting NPC1L1 protein (3). Subsequent to absorption, ezetimibe is glucurononidated to phenolic and benzyl conjugates by both hepatic and intestinal uridine-diphosphate-glucuronosyltransferases (4). Ezetimibe can be deconjugated by intestinal bacteria and then reabsorbed and reglucuronidated. In this manner, ezetimibe undergoes enterohepatic recirculation with repeat delivery to its site of action in the intestinal wall with minimal systemic exposure (5). The half-life of ezetimibe is approximately 22 hrs and dosed at 10 mg daily. Ezetimibe does not decrease the absorption of bile salts, triglycerides, free fatty acids, steroid hormones, or fat soluble vitamins (6).

Ezetimibe decreases cholesterol absorption and serum LDL-C by approximately 54% and 20%, respectively (7). The degree of LDL-C reduction is less than that of cholesterol absorption because, as cholesterol uptake from the gastrointestinal tract decreases, the liver upregulates the activity of HMG CoA-reductase, thereby augmenting endogenous cholesterol biosynthesis. Another study evaluating ezetimibe monotherapy in patients with hypercholesterolemia demonstrated a mean reduction in serum LDL-C levels of 17.7% compared to baseline (8). In nearly one-quarter of patients, serum LDL-C can decrease by 25% or more. Ezetimibe decreases serum triglyceride levels approximately 8.3% and increases HDL-C 1-4.2%. Ezetimibe is indicated in the management of patients with sitosterolemia and decreases serum concentrations of campesterol and sitosterol by 24% and 21%, respectively (9).

Ezetimibe can be combined with any currently available statin. It is also available as a formulation (Vytorin, Merck-Schering Plough) that combines 10 mg of ezetimibe with increasing doses of simvastatin (10, 20, 40, 80 mg). With each doubling in the dose of a statin, serum LDL-C levels drop by approximately 5-6% (the “rule of sixes”). By using the combination of ezetimibe and a statin, the need for statin titration is precluded in at least some patients. To illustrate this further, consider the following: 10 mg of ezetimibe combined with 10 mg of simvastatin decreases serum LDL-C by 44%, a reduction essentially identical to that achieved with 80 mg of simvastatin (10). Ezetimibe also significantly augments the ability of atorvastatin, pravastatin, and rosuvastatin to reduce serum LDL-C levels.

Ezetimibe can substantially raise the percentage of high-risk patients meeting their LDL-C goal of < 100 mg/dL, the primary target of dyslipidemia management. After five weeks of therapy with ezetimibe combined with simvastatin 10, 20, or 40 mg, the percentage of these patients achieving an LDL-C < 100 mg/dL was 75%, 83%, and 87%, respectively (11). These are impressive results. Although clinical endpoint trials have yet to be completed with ezetimibe, ezetimibe can substantially boost the capacity of a statin to help patients meet their LDL-C NCEP targets. This type of combination therapy is an especially attractive option when managing patients who cannot get to goal with even high dose statin therapy (e.g., severe mixed dyslipidemia, familial hypercholesterolemia), do not tolerate higher doses of a statin due to adverse events, or who refuse to take moderate to high dose statin therapy because of safety concerns.

REFERENCES
Did You Know?

The benefits of membership to the NLA include members only resources on www.lipid.org. Resources on the website include:

The Clinical Trials Research Center
Learn more about clinical research and new medical therapies for treating lipid related conditions. Topics available include current clinical trial listings, the latest trial results, new FDA drug approvals and more.

Member Forums at the NLA
Discuss topics with other members online. Categories include difficult cases, clinical trials, regional events, membership issues, so create a topic and share it with your peers.

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Special Report continued from page 6

The experiences of these physicians not only reinforce the need for better preparation on behalf of the government, but also the medical community. Hurricane Katrina’s devastating effects revealed the depth of preparedness needed within the medical community to handle patient care emergencies under these conditions.

While these NLA members were at different facilities and in different states, they, almost universally, stressed the same key points they wanted to pass along to their NLA colleagues around the country.

- Encourage your patients to know their medicines and keep a 30-day supply ready; keep basic first aid-supplies in your vehicle; know your local emergency preparedness plan; and know the state and local agencies to contact to sign up as a medical volunteer for your community.
- Keep your own immunizations current.
- Create a method of communication between the designated medical facilities and evacuation/relief centers so information about supplies, available medical volunteers, evacuation plans, etc., can be shared.
- Have faith in your fellow professionals. While the working conditions thrust these professionals into third-world situations, the spirit, efforts, commitment and dedication they saw in their colleagues, other volunteers and the military personnel was astonishing.
- Continue to contribute to the charity of your choice; the need for on-going assistance in the region is deep and will last a long time.

Many of the NLA members also reported that this experience strengthened their belief that the government must quickly implement full electronic medical record keeping. ❤
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Impact of Nutrition Therapy as the Initial Therapeutic Intervention in the Management of Severe Hypertriglyceridemia

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Objective:
To document the degree to which dietary intervention prior to consultation with a lipid specialist impacts severe hypertriglyceridemia in a multidisciplinary lipid management program

Methods:
Of 288 new patients enrolled in a lipid management program, 88 had initial triglycerides over 500 mg/dL. They received usual clinic care consisting of comprehensive laboratory assessment of traditional and novel risk factors, nutrition assessment and initiation of an individualized diet intervention prior to seeing the lipid specialist. During the 75 minute medical nutrition therapy session, registered dietitians provided nutrition prescriptions based on presenting level of triglycerides, carbohydrate tolerance, and initial diet and lifestyle. Nutrition counseling addressed quality and quantity of fat, carbohydrate and protein as well as calories and glucose management with a focus on the proportions and types of foods to eat. A meal plan designed to rapidly clear chylomicrons was used, if indicated, and adjusted after 2 weeks based on response. Alcohol was eliminated or reduced; smoking cessation was addressed, as appropriate. Exercise recommendations were 30 to 60 minutes of aerobic exercise 5 to 6 days per week. Clinical and behavioral outcomes were recorded at each visit. Food intake was assessed with 24 hour recalls estimated by the registered dietitians.

Results:
Eighty-six patients returned for assessment of the diet intervention, brief nutrition consult, and treatment plan from a lipid specialist. For 13 patients, cholesterol-lowering medications were altered by the referring physician prior to the second clinic visit. There were incomplete data on 5 patients. Of the remaining 67 patients (22 female; 45 male; mean age 48.9 ± 10.5; BMI 33.1 ± 5.5 kg/m2), 25 were diabetic and an additional 19 had elevated fasting glucose; 26 reported intolerance to lipid-lowering medication. 32 of 57 patients had prior counseling with a registered dietitian. 30 patients were taking a fibric acid derivative, 15 a niacin compound, 19 a statin, 1 colesevelam, 2 ezetimibe, and 5 a small dose of fish oil. Initial lipids were total cholesterol 329 ± 138 mg/dL; triglycerides 1554 ± 1901 mg/dL (range 503 to 12,723); HDL-C 41.6 ± 12.9 mg/dL; LDL-C 95.0 ± 44.8 mg/dL; and glucose 124 ± 59.6 mg/dL.

Average time between first and second clinic visit was 7.4 ± 5.6 weeks. Patients rated compliance with food choice recommendations as 7.4 ± 2.3 on a scale of 1 to 10. There were significant reductions in weight (215.5 ± 40.9 to 208.4 ± 40.7 lb., p < .001); reported total energy consumption (2110 ± 963 to 1440 ± 574 kcal, p < .001); total carbohydrate (235 ± 111 to 160 ± 59 grams, p < .001); and total fat (86.5 ± 54.6 to 50.1 ± 36.8 grams, p < .001). The number of exercise days per week increased from 1.4 ±2.4 to 2.2 ± 2.5 (p<.05). Triglycerides were reduced from 1554 ± 1901 mg/dL to 592 ± 395 mg/dL (p <.001 for log transformation of triglycerides). Twenty-two per cent of patients reached a triglyceride level < 300 mg/dL. There was no difference in triglyceride reduction between patients on or not on lipid-lowering medication. The reduction in triglycerides was independent of prior RD counseling.

Conclusion:
Aggressive individualized nutrition therapy by registered dietitians results in changes in eating behavior and large reductions in triglyceride levels in patients with severe hypertriglyceridemia independent of lipid-lowering medications and prior nutrition counseling. These results imply that severe hypertriglyceridemia is most effectively treated by individualized dietary therapy, regardless of the usage/non-usage of medications. The initial diet intervention demonstrates the importance of diet to the patient and provides the lipid specialist with a more accurate picture of the underlying lipid disorder.
More than 200 professionals attended this year’s annual meeting, “Practical Applications of Atherosclerosis Research,” which took place at the Pinehurst Resort in North Carolina, August 5-7, 2005. Practical sessions and workshops featured cases and discussions of the clinical applications of advanced lipid research. Registrants and guests also had the option of participating in some new “heart healthy” activities, including a practical pedometry course and a heart healthy recipe contest (see below for the winning recipes).

Make plans now to attend next year’s big event. SELA’s 9th Annual Scientific Forum will take place in Amelia Island, Florida, August 11-13, 2006, at the Amelia Island Plantation. The Masters in Lipidology Advanced Training and Review Course will also be offered on August 10, 2006. Registration will be available online soon. Check the www.lipid.org website for the most up to date meeting information.

SELA Heart Healthy Recipe Contest

The Saturday Evening dinner at this year’s meeting gave attendees the opportunity to sample food high in taste and nutrition from recipes submitted.

**Winning Appetizer Recipe:**

**Mediterranean Vegetable Stack**

By Thomas A. Bridge, MD
American Health Network, Lafayette, IN

A delightful, healthy visual treat for your eyes as well as your body. Suitable as an appetizer or centered on a plate of mixed greens as a salad.

**Ingredients:**

- 1 large eggplant
- Several ripe tomatoes *mixed heirloom tomatoes are recommended
- Fresh spinach leaves stem portion removed
- 8 to 16 oz goat cheese (depending on how many layers and the size you’d like to make)
- Yellow squash and/or zucchini squash
- Extra virgin olive oil spray (or Misto spray w/ extra olive oil)
- Balsamic vinegar

**Directions:**

Thinly slice the eggplant, tomatoes, and squash (if using a mandoline slicer, set at about 1/8 to 3/16). Lightly salt the slices (to reduce the liquid content of the eggplant) and place them between paper towels. Lightly spray the eggplant and squash (not tomatoes) with olive oil spray (or Misto spray). Grill the vegetables over medium heat, taking care not to overcook them.

Line a loaf pan with plastic wrap. Place a layer of eggplant on the bottom of pan, followed by a layer of tomatoes, and a layer of squash. Drizzle or spray a small amount of balsamic vinegar over layers, followed with cracked pepper, if desired. Thinly slice the goat cheese and layer on top of squash. Lay fresh spinach leaves on top of goat cheese. Continue to stack the eggplant, tomatoes, squash, goat cheese; spinach leaves, finishing with eggplant.

Cover stack with a baking sheet lightly weighted with some plates and refrigerate. When chilled and lightly compressed, invert the pan on a carving board and remove plastic wrap. Slice stack in long, thin slices or large rectangle stacks. Drizzle with balsamic vinegar and serve with spinach leaves and garnish.

SELA thanks ACCUSPLIT for providing complimentary pedometers to attendees.
Winning Entrée Recipe:
Lemon Roasted Wild Salmon with a Soy Glaze
by Cheryl Graffagnino RD, LD and James M. Falko MD
The McConnell Heart Health Center, Columbus, Ohio

Ingredients:
1 pound wild salmon filets, with skin
1 tablespoon olive oil
1 ½ teaspoons sesame oil
1 tablespoon rice wine vinegar
1 tablespoon low sodium soy sauce
1 ½ teaspoons brown sugar, packed
1 clove garlic, minced
pinch freshly ground black pepper
1 tablespoon minced green onion
2-3 fresh lemons
Cooking spray

Directions:
In a small bowl, combine olive oil, rice wine vinegar, soy sauce, brown sugar, garlic, pepper and green onion. Mix well to create a marinade and set aside.

Rinse salmon filets and make 3-4 evenly spaced shallow cuts in the skin side of the fish. Place filets in a shallow pan or bowl, skin side down. Pour marinade over salmon filets, cover and refrigerate. Allow salmon to marinate for at least 2 hours.

Preheat oven to 350°. Slice lemons in thick slices. Coat the bottom of a shallow baking pan with cooking spray. Arrange lemon slices in a single layer in the baking pan. Arrange the salmon filets in a single layer on top of the lemon slices with the skin side down. Reserve the marinade liquid.

Bake the salmon for about 20-30 minutes. Cooking time will vary depending on the thickness of the filets. The fish is done when it flakes easily with a fork.

Pour reserved marinade liquid into a small saucepan. Over medium-high heat, bring liquid to a boil for 2-3 minutes. Reduce heat and simmer until sauce is reduced to a syrup-like consistency.

Remove skin from cooked salmon fillets. Top each salmon fillet with the sauce and serve.

Serving Suggestion:
Serve salmon fillets on a bed of lightly sautéed fresh spinach or brown rice. Makes 4 servings.

Nutritional Information (per serving of fish with sauce)
217 Calories, 10 Grams total fat, 2 Grams saturated fat, 5 Grams monounsaturated fat, 2.5 Grams polyunsaturated fat, 63 Milligrams cholesterol, 200 Milligrams sodium, 27 Grams protein, 3 Grams carbohydrate, 0 Grams dietary fiber.

Winning Dessert Recipe:
Unbelievable Chocolate Cake
By Cheryl Kuhta-Sutter, RN, LDN
Presbyterian Center for Preventive Cardiology, Charlotte, NC

Ingredients:
1 3/4 cups all purpose flour
1/2 cup soy flour
1 3/4 cups sugar
2/3 cup cocoa
1 1/4 teaspoons baking soda
1 1/2 teaspoons baking powder
1 teaspoon salt
1/2 cup light silken tofu, firm
1 1/2 cups water, divided
1 teaspoon vanilla
1/2 cup unsweetened applesauce
1 1/2 cup trans fat-free margarine
4 tablespoons cocoa
3 tablespoons skim milk
1 Tablespoon vanilla
3/4 box sifted powdered sugar

Directions:
Preheat oven to 350 degrees. In a large bowl, sift together flours, sugar, cocoa, baking soda, baking powder, and salt. Set aside.

In a food processor, puree tofu with 1/2 cup water, add remaining water and vanilla. Process until mixture is smooth. Add dry ingredients and blend on high. Add applesauce and process until completely mixed.

Spread batter into a 13 x 9 baking dish or two 9 inch round pans sprayed with cooking spray and lightly floured. Bake 35-40 min. Remove cake from oven and let cool.

Meanwhile melt margarine in a small saucepan over low heat. Remove from heat and add cocoa, milk, vanilla and powdered sugar, stir well. Frost cake after it has cooled. Enjoy!

Nutritional Info per serving: 273 Calories, 4 Grams fat, 4 Grams protein, 59 carbohydrates, 2 Grams dietary fiber, 323 Milligrams sodium.
Winning Poster Abstract from the SELA 8th Annual Forum

Aged Rats Lose Estrogen-induced Vasoprotective and Anti-inflammatory Effects in Injured Arteries

Author: Andrew P Miller, MD
Birmingham, Alabama

Objective:
This study attempted to reconcile the fundamentally protective and clinically neutral paradox surrounding estrogen in the vasculature by testing the hypothesis that responsiveness to 17beta-Estradiol (E2) is lost in injured arteries of aged (10-14 mo old) ovariectomized (OVX) rats.

Methods:
Aged and young OVX rats were randomly assigned to treatment with E2 or vehicle (V) and subjected to balloon injury of the right common carotid artery. Rats were sacrificed at 2 wks after injury for morphometric examination of injured arteries, at 24 hrs for assessment of leukocyte infiltration by immunohistochemistry and flow cytometry, and at 2 hrs for quantitation of inflammatory mediator mRNA expression by real-time RT-PCR.

Results:
Neointima formation was significantly reduced in aged compared with young V rats. E2 treatment had directionally opposite effects on intima/media ratios in aged (+75%) and young (-40%) rats. Similar to findings in young rats, injury induced major increases in infiltrating CD45+ leukocytes (4-fold), HIS48+ granulocytes (9-fold), Mar1+ monocyte/macrophages (8-fold), and in expression of inflammatory mediators (2 to 1000-fold) in arteries of aged rats. Unlike findings in young rats, E2 had no effect on these inflammatory responses to injury in vessels of aged rats.

Conclusion:
Aged OVX rats lose the vasoprotective and anti-inflammatory responses to exogenous E2 seen in injured arteries of younger animals. These results may help to explain lack of E2 responsiveness in the vasculature of elderly postmenopausal women.

Congratulations to the SELA Poster Session Contest Winners:

1st Place Winner ($500 award)
Andrew P. Miller, MD of Birmingham, AL

2nd Place Winner ($250 award)
Michelle LaLonde, BS of Columbus, OH

The Effect of a Community Weight Loss Program on Clinical Outcomes in Obese Participants with and without Metabolic Syndrome

Announcing the Newly Elected 2005-2006 Board of Directors

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Betsy LaForge, MPH, RD
Durham, NC

In-Training Member
Ritesh Gupta, MD
Birmingham, AL
This 1½-day intensive Board Review Course is planned by the NLA as a CME activity to prepare physicians seeking certification by the American Board of Clinical Lipidology (ABCL) or an in-depth, advanced review of the specialty of clinical lipidology. This comprehensive course will cover clinical aspects of the prevention, diagnosis and management of dyslipidemias and the metabolic syndrome and the fundamentals of vascular biology. An audience response system will allow participants to test themselves on practice Board questions before and after each subject. After completing the course, attendees will be more confident of their ability to perform well on the ABCL Board Certification Examination and will have improved their clinical knowledge significantly.

Course Features
Registrants for the Masters course will receive all 3 volumes of the NLA Self-Assessment Program (NLA-SAP) to take home and complete. After successful completion of the Masters course and the 3 volumes of the NLA-SAP, you will have earned nearly 200 hours of lipid focused continuing education, which is an eligibility requirement of the ABCL.

Benefits of Attending:
- Receive all 3 volumes of the NLA Self-Assessment Program (included in course fee)
- Prepare for the ABCL certification examination in Clinical Lipidology
- Obtain nearly 200 hours of lipid focused CME credit

Course Curriculum
The didactic curriculum of the Board Review Course will follow the outline of topics covered in the NLA Self-Assessment Program:
- Diagnosis and Management of Dyslipidemias
- Metabolic Syndrome
- Vascular Biology and Advanced Lipoprotein Metabolism.

Each main subject area will include a pre-test and a post-test.

Target Audience
This program is intended for cardiologists, endocrinologists, internists and primary care physicians who wish to specialize and become certified by the American Board of Clinical Lipidology or who desire advanced training in the management of dyslipidemias and the metabolic syndrome.

Accreditation Statement
This activity has been approved for AMA PRA credit.

Commercial Support
This activity is support in part through an educational grant from AstraZeneca.
The NLA-SAP series offers a comprehensive, interactive clinical problem-solving program that will objectively validate, strengthen and reinforce your knowledge of clinical lipidology. Each of the 3 volumes is developed by a group of renowned experts.

Each volume of the NLA-SAP provides up to 60 hours of AMA Category 1 medical education credit. Also, the hours obtained in the NLA-SAP can be applied toward meeting the 200-hour focused lipid education credit requirement necessary to be eligible for the American Board of Clinical Lipidology (ABCL) certifying examination.

Benefits of the NLA-SAP:

1. Prepare for board certification in clinical lipidology or use the credit to renew your state practice license, hospital practice privileges, and for credentialing by managed care organizations.
2. Learn from the experience of leading practitioners and experts in the diagnosis and management of dyslipidemias and in clinical research.
3. Complete the NLA-SAP anywhere – there are no travel or lodging costs – and no time away from your patients and family.

Sponsored for CME Credit by the National Lipid Association.

Funded in part by an educational grant from AstraZeneca.

Developed and published by Professional Evaluation, Inc.
ORDER FORM

NATIONAL LIPID
ASSOCIATION
SELF-ASSESSMENT
PROGRAM

All 3 volumes are available now. NLA members receive a significant savings of $25 per volume. Order all 3 volumes now for a total savings of $150. You may order the NLA-SAP by visiting the NLA website at www.lipid.org/sap, or by returning the order form below. Please allow 2-3 weeks for delivery.

__ I am a member of the NLA

You may apply for membership online at www.lipid.org/membership.php or call the NLA office at 904.998.0854 to request an application.

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Total Enclosed $_______

Name (First) (MI) Last Degree

Address

City State Zip

Email Phone number

☐ Check
☐ MasterCard Card # Exp date
☐ Visa Card is issued in the name of
☐ AmEx Signature of Cardholder Date

By Mail
National Lipid Association
8833 Perimeter Park Blvd, #301
Jacksonville, FL 32216
By Fax (Credit Card Orders Only)
904.998.0855
Online www.lipid.org/sap
**ASSOCIATION NEWS**

**Scientific Experts Gather for Statin Safety Conference**

Washington, D.C. was the site for a conference of impressive international experts who gathered to examine and report on the safety profile of statins and other lipid lowering drugs. The NLA sponsored the Statin Safety Task Force to examine the safety of statins when used alone or in combination with other lipid altering agents.

Chaired by James M. McKenney, PharmD, the Task Force met for two days in July to explore aspects of statin safety and review scientific evidence presented to a distinguished Expert Panel from the United States and other countries.

Dr. McKenney opened the conference by referencing the 20-year history of statins and their proven efficacy based on many scientific studies that show statins reduce morbidity and mortality.

He said there is a clear need to improve the flow of scientific, fact-based information about cholesterol-lowering treatments. “Patients are telling us that they are sometimes hesitant to start or continue their medications because of random reports they hear on the news and that they are unable to sort through what they are being told. It is also clear that many people who are prescribed therapy do not reach their cholesterol goal and sometimes discontinue their treatment without consulting with their physician. We have a long way to go,” Dr. McKenney said. “That is why the NLA has decided to conduct independent research using the FDA AERS database, randomized clinical trials, new drug applications, and managed care claims database to fully understand statin safety issues and to convene this blue ribbon panel of experts. They will give us their opinions and their judgments as to the safety of these products, having heard and studied the evidence in detail.”

On the second day of the conference the panel met in an open forum and received presentations on the Adverse Event Reporting System, an analysis of New Drug Applications (NDA) submissions for marketed statins, a meta-analysis of randomized clinical trials, and an analysis of a managed care claims database. Audience observers were individuals from the scientific, pharmaceutical and government sectors and included Dr. David Orloff from the FDA.

The Task Force’s independent perspective, including their analysis and recommendations, will be published.

**NLA Joins the Peripheral Arterial Disease Coalition**

The NLA recently joined the Peripheral Arterial Disease (PAD) Coalition, a group of organizations formed with the mission of reducing the morbidity and mortality associated with PAD. Led by the Vascular Disease Foundation and in cooperation with the National Institutes of Health, the Coalition’s top priority is to develop a national public awareness campaign that emphasizes key messages regarding early detection, diagnosis and treatment of PAD. Members of the coalition may participate in Coalition governance and program creation. Dr. William James Howard will serve as an NLA representative and attend the Annual Meeting of the PAD Coalition in Northern Virginia on October 11, 2005.

**CHAPTER NEWS**

**NLA and Chapter Reorganization**

The Midwest Lipid Association and Southeast Lipid Association both agreed to become affiliates of the NLA instead of independent organizations at their 2005 Annual Scientific Forums earlier this year. This new organizational structure will provide more unity in the NLA's mission and goals. All chapters will continue to be active and will offer their own education programs and services tailored specifically to the unique needs of the region. Members of the Northeast Lipid Association (NELA) will vote to become an affiliate chapter of the NLA at their 2006 annual meeting in Boston, Massachusetts this April, and the future Southwest and Pacific chapters will be formed as affiliate chapters of NLA as well.

**West Coast/Pacific Chapter Forming**

We are pleased to announce that the development of the Pacific Lipid Association (PLA) is underway. There is a tentative steering committee meeting scheduled to take place at the Southwest Lipid Association’s Inaugural Scientific Forum in San Antonio, Texas, February 10-12, 2006. We wish to encourage members interested in participating in the development of this chapter to attend the steering committee meeting. More details will be available at a later date.

If you have any questions or would like to participate in the formation of this new chapter, please contact Adam Beamer, Assistant Director of Programs at the NLA office (904-998-0854 ext 210) or email at abeamer@lipid.org.

**EDUCATION NEWS**

**Clinical Trials Resource Center Online**

NLA members and patients now have easy online access to the latest information on clinical trials participation and research for patients and healthcare professionals. Located on the lipid.org website homepage, this new feature by Thomson CenterWatch provides members with a listing of relevant clinical trials looking for recruits, the latest FDA drug approvals, news relating to clinical trials, and recently completed and ongoing trials based on published materials from medical conference, journals and company reports.

**2005 NLA Highlights CD-ROM Now Available**

If you were unable to make it to this summer’s scientific meetings, you are in luck. Thanks to an educational grant from Kos Pharmaceuticals, Inc., we are providing all members with a CD-ROM that features presentation highlights from the NLA 2005 Scientific Sessions. Your complimentary CD-ROM accompanies this issue of the Lipid Spin. This sampling of our scientific sessions reflects the outstanding quality of the NLA’s faculty and educational programming. Save the date to attend at least one of the four NLA regional scientific meetings to be held in 2006.

**PCNA Educational Programs Available to NLA Members**

The following programs from The Preventive Cardiovascular Nurses Association (PCNA) are available to NLA members at no cost:

**PCNA’s Online Heart Talk**

The PCNA announces its first online heart health nutrition program: Heart Talk Nourishing Healthy Hearts. The course is complimentary and approved for 3.6 continuing education hours. This interactive educational program will help practitioners stay abreast of the most current information about nutrition in heart health, using evidence-based research and current national guidelines while providing valuable tools for patient education. All healthcare providers are encouraged to take the course at www.pcna.net.

**“What’s Missing in CholesterolALL?”**

This program was designed by PCNA to educate women about the importance of knowing and optimizing all of the components of the lipid profile, with a particular focus on the importance of HDL in women. Visit www.pcna.net or www.RaiseYourCholesterol.com for more information or call 1-877-HDL-GOAL (877-435-4625) to order free brochures – a perfect way to educate your patients about this topic!
Safety Issues: The NLA Extends its Study

Lipid management that employs dietary, lifestyle, and pharmacologic modalities has been demonstrated to be one of the most effective strategies for the prospective treatment of atherosclerosis and the prevention of its sequelae. “Statin” safety, the most questioned area of treatment, is the focus of the recently completed NLA study that will be the centerpiece of an upcoming publication.

However, there is an expansive area regarding the safety of non-statin therapies and additional work on combination issues that still needs to be studied. The Task Force’s new initiative will focus on examining this important area of treatment and prevention. Additionally, the existing NLA Consumer Affairs Committee will be asked to review and report to the Task Force and membership regarding the issues of nutriceutical safety as it relates to the efficacy as a lipid lowering strategy for patients at risk.

Although both the Task Force and Consumer Affairs Committee exist, the NLA is seeking additional participation from members whose unique expertise makes them well suited to participate in the expert panel process. If you are interested in participating, please send an email to the NLA office by the end of November 2005. Availability is limited.

Qualified members will be formally invited in December 2005 and work will begin in January 2006. The expected completion and report of the Task Force and Committee is expected by summer 2006. Details about the task force and committee, the current composition and charge will be found on the NLA website (www.lipid.org) this November. A web based application to volunteer will also be available soon.

Funding of this study was made possible from grants from AstraZeneca, Kos, Sankyo and Merck/Schering-Plough.
ABCL Certification Program Now Available for Qualified Physicians

2005 marks a milestone for the American Board of Clinical Lipidology (ABCL), as well as the NLA, in that the first certifying examination will be offered on November 12, 2005 in Dallas, TX. This is a monumental achievement thanks to the visionary efforts of a group of devoted lipid specialists.

Nearly 150 physicians will pursue the status of “Diplomate” of the American Board of Clinical Lipidology in the 2005 examination group. These physicians have demonstrated their professional commitment to the prevention of cardiovascular disease and will document their expertise in lipid management for patients, professional colleagues and external organizations.

The American Board of Clinical Lipidology is an independent certifying organization offering the only certification program for physicians specializing in Clinical Lipidology. The ABCL has established a rigorous credentialing process and an examination that will assess and validate the specialized knowledge and advanced training required to practice in this dynamic and complex field.

To become credentialed, candidates must meet the basic requirements and the training requirements established by the ABCL. These requirements require candidates to earn 200 credit hour equivalents or “points” based on documented participation in “lipid-focused” CME and expertise in lipid management. The credentialing criteria have been designed to provide any physician with demonstrated knowledge and experience in Lipidology an avenue to become certified as a clinical lipid specialist. Visit [www.lipidboard.org](http://www.lipidboard.org) for the eligibility requirements and an application.

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**Certification for Allied Health Professionals**

We have received many questions from non-physicians seeking certification, since at this time the certification program is open to only licensed physicians. The ABCL Board of Directors will be addressing this situation through discussions with representatives from the various disciplines interested in an advanced certification program. Plans are in progress to develop a similar credentialing and certification process for mid-level providers with demonstrated experience and/or advanced training in clinical lipid management.

For eligibility requirements, examination information and an application you may visit the American Board of Clinical Lipidology website at [www.lipidboard.org](http://www.lipidboard.org) or call the management office at 904-674-0752.

**Special Benefit for NLA members Who Achieve Certification**

The National Lipid Association recognizes the efforts of its members to attain board certification. If you successfully complete the NLA Masters in Lipidology Board Review Course or all three volumes of the NLA-Self-Assessment Program (NLA-SAP) and credential to sit for the ABCL certification exam, you will receive a $300 credit voucher from the NLA. To recognize the achievement of board certification, the NLA is pleased to extend a 10-year pre-paid membership (a $500 value) to active NLA members who successfully pass the exam and become certified by the ABCL.
LIPID EDUCATION PROGRAMS

The following are lipid education programs endorsed or sponsored by the NLA.

Visit page 18 for the NLA Masters in Lipidology Advanced Training and Review Course schedule, and page 23 for the 2006 American Board of Clinical Lipidology exam dates.

November 18, 2005
Duke Lipid Clinic Preceptorship Advanced Program
See below for more information

December 16, 2005
Duke Lipid Clinic Preceptorship Program
Duke University, David Thomas Executive Conference Center
John Guyton, MD, Medical Director
Contact Ralph LaForge, MSc or Email: rlaforge@nc.rr.com
To register, call (919-490-3794)

January 18-20, 2006
American Heart Association: Obesity, Lifestyle, and Cardiovascular Disease Symposium
Co-sponsored by NLA
Grand Hyatt Washington, Washington, DC
Phone: (214) 706-1543
E-mail: scientificconferences@heart.org

January 27-29, 2006
Florida Lipid Foundation Inaugural Meeting & Lipid Clinic Training Program
The KEY to Better Patient Outcomes
Colony Beach & Tennis Resort, Longboat Key, Florida
Call 1-800-4-COLONY and request the FLF room block rate of $199.00 per evening before 1/5/06.
E-mail: ssheridan@lipid.org
Website: www.floridalipidfoundation.org

February 10-12, 2006
Southwest Lipid Association Inaugural Scientific Forum
Hyatt Regency Hill Country Resort
San Antonio, TX
Call 800-233-1324 and request the $175 Southwest or National Lipid Association room rate.
E-mail: ssheridan@lipid.org
Website: www.lipid.org

April 7-9, 2006
National Lipid Association & Northeast Lipid Association
2006 Annual Scientific Sessions
Seaport Hotel, Boston, MA
E-mail: ssheridan@lipid.org
Website: www.lipid.org/chapters/nela/

April 20-22, 2006
Preventive Cardiovascular Nurses Association
12th Annual National Symposium
Denver, CO
Website: www.pcna.net

June 18-22, 2006
International Atherosclerosis Society XIV
International Symposium on Atherosclerosis
Rome, Italy
Contact: Giovanni Lorenzini Foundation or E-mail: info@isa2006.org
Website: www.isa2006.org

August 11-13, 2006
9th Annual Scientific Forum of the Southeast Lipid Association
Amelia Island Plantation, Amelia Island, Florida
E-mail: ssheridan@lipid.org
Website: www.lipid.org/chapters/sela/

October 20-22, 2006
3rd Annual Scientific Forum of the Midwest Lipid Association
The Fairmont Kansas City at the Plaza, Kansas City, MO
E-mail: ssheridan@lipid.org
Website: www.lipid.org/chapters/mwla/

MEETINGS & EDUCATIONAL EVENTS

November 13-16, 2005
2005 American Heart Association Scientific Sessions
Dallas Convention Center, Dallas, TX
E-mail: scientificconferences@heart.org
Website: www.my.americanheart.org

March 2-4, 2006
46th Annual Conference on Cardiovascular Disease
Epidemiology and Prevention in Association with the Council on Nutrition, Physical Activity, and Metabolism
Pointe Hilton Squaw Peak Resort, Phoenix, AZ
E-mail: scientificconferences@heart.org
Website: www.my.americanheart.org

April 27-29, 2006
7th Annual Conference on Arteriosclerosis, Thrombosis, and Vascular Biology
Denver Marriott, Denver, CO
E-mail: scientificconferences@heart.org
Website: www.my.americanheart.org

May 7-9, 2006
7th Scientific Forum on Quality of Care and Outcomes
Research in Cardiovascular Disease
Omni Shoreham Hotel, Washington, DC
E-mail: scientificconferences@heart.org
Website: www.my.americanheart.org

July 23-August 5, 2006
32nd Ten-Day Seminar on the Epidemiology and Prevention of Cardiovascular Disease
Granlibakken Conference Center, Tahoe City, CA
E-mail: scientificconferences@heart.org
Website: www.my.americanheart.org