In February 2000, the American Heart Association formed the Council on Nutrition, Physical Activity, and Metabolism (NPAM) to advance the expansion and exchange of knowledge on nutrition, physical activity, obesity and diabetes. The Council promotes basic and clinical research in these areas to develop approaches for prevention and treatment of coronary heart disease and stroke. Over the past year, several landmark studies have confirmed the significance of our formidable mission. For example, consider the following sobering reports:

Researchers examined the association between body mass index (BMI) and acute coronary events in over 54,000 middle-aged men and women who had no known heart disease at baseline. During an average follow-up of nearly eight years, 1,127 cases of anginal chest pain or acute myocardial infarction occurred. After adjustments for numerous lifestyle factors, each unit of BMI (above 25) was associated with an 8 percent and 7 percent higher risk of coronary events in women and men, respectively.

Another study examined the relationship between BMI and patient age of first heart attack in more than 111,000 people who had heart attacks. After adjusting for baseline demographic data, cardiac risk factors and medications, the age at which a first heart attack occurred went up steadily with increasing BMI: 3.5 years earlier for a BMI of 25.1 to 30.0; 6.8 years earlier for a BMI of 30.1 to 35.0; 9.4 years for a BMI of 35.1 to 40.0; and 12 years earlier for a BMI greater than 40. The investigators concluded that weight reduction could be an effective strategy to reduce cardiovascular risk.

A sedentary lifestyle has now been shown to have a deleterious effect on cellular morphology (leukocyte telomere length) and may accelerate the aging process. Startling new studies indicate that people who exercise regularly are biologically younger – by approximately 10 years — than those who don’t.

Exercise capacity is a strong predictor of all-cause mortality in blacks and whites. The relationship was inverse and graded, with a similar impact on mortality outcomes for both blacks and whites. Adjusted risk was reduced by 13 percent for every 1 metabolic equivalent (MET; 1 MET = 3.5 mL O2/kg/min) increase in exercise capacity.

Collectively, these findings and other recent reports highlight the value of aggressive risk factor reduction via lifestyle modification and complementary pharmacotherapies, if appropriate, in the prevention of initial and recurrent cardiovascular events.

The November AHA Scientific Sessions were highly successful for the NPAM Council, as our members/fellows contributed significantly to the program planning and presentations. Many Council members chaired sessions and presented enlightening oral and poster presentations. Additional program highlights included: The Robert I. Levy Endowed Lecture in Lipid Metabolism, presented by Dr. Robert Eckel; the NPAM Young Investigator Award, presented by Dr. Oliver Rider; and, the prestigious Population Research Award, presented to Dr. Steven Blair. These recipients were also acknowledged at our NPAM Annual Business Meeting and the Annual Reception and Dinner with the Council on Epidemiology and Prevention, as were our new Fellows. The latter include: Fernando A. Costa, MD; Julie St.-Pierre, MD, PhD; Donald R. Dengel, PhD; Kerry J. Stewart, EdD; Eric Larose, DVM; Darren K. McGuire, MD; and Eugene Wolfel, MD.

Each of our subcommittees held successful meetings that were well attended, and our leadership meeting reviewed numerous issues, including forthcoming scientific statements and advisories, a collaborative book project, the revised Web site and Council brochure, conference co-sponsorship requests, budget items, strategic planning, varied committee reports, advocacy initiatives, and plans for our 2009 Spring Conference, March 10–12, 2009, at the Innisbrook Resort and Golf Club/Palm Harbor, Fl.

Needless to say, this is an exciting time to be a member of the NPAM Council. For additional information, feel free to contact me at (bfranklin@beaumont.edu). We’d welcome your involvement!

References


TISSUE-SPECIFIC REGULATION OF LIPOPROTEIN LIPASE AND ENERGY BALANCE: THE STORY GETS EVEN MORE INTERESTING

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Lipoprotein lipase (LPL) is a multifunctional enzyme produced by and studied in many tissues including adipose tissue, cardiac and skeletal muscle, islets, and macrophages. After synthesis by parenchymal cells the lipase is transported to the capillary endothelium where it is rate-limiting for the hydrolysis of the triglyceride (TG) core of the circulating TG-rich lipoproteins, chylomicrons and very low density lipoproteins (VLDL). The reaction products, fatty acids and monoacylglycerol, are in part taken up by the tissues locally where they are processed in a tissue-specific manner, e.g., stored as neutral lipids (TG >> cholesteryl esters(CE)) in adipose tissue, oxidized or stored in muscle, or as CE/TG in foam cells in macrophages. LPL is regulated in a tissue-specific manner. In adipose tissue LPL is increased by insulin and meals but decreased by fasting whereas muscle LPL is decreased by insulin and increased by fasting. In obesity, adipose tissue LPL is increased; however, the insulin dose response curve is shifted to the right. Following weight reduction and stabilization of the reduced obese state, adipose tissue LPL is increased as is the response of the enzyme to insulin and meals. In skeletal muscle, insulin does not stimulate LPL nor is the enzyme activity changed in obesity; however, following weight reduction LPL in skeletal muscle is decreased by 70 percent. These tissue-specific changes in LPL set the stage for lipid partitioning to help explain the recidivism of obesity. To examine this divergent regulation further, transgenic and knockout murine models of tissue-specific LPL expression have been developed. Mice with overexpression of LPL in skeletal muscle develop TG accumulation in muscle, insulin resistance, are protected from excessive weight gain, and increase their metabolic rate in the cold. When placed onto the LPL knockout and leptin-deficient background, overexpression of LPL using an MCK promoter reduces obesity. Alternatively a deletion of LPL in skeletal muscle reduces TG accumulation and increases insulin-mediated glucose transport into muscle but leads to lipid partitioning to other tissues, insulin resistance and obesity. In the heart, loss of LPL is associated with hypertriglyceridemia and a greater utilization of glucose implying that free fatty acids are not a sufficient fuel for optimal cardiac function. LPL is also produced in the brain and that’s where the “story gets even more interesting.” We have just created mice with a neuron-specific deletion of LPL (NEXLPL-/−) using cre recombinase driven by the helix-loop-helix nuclear transcription factor NEX promoter. By 6 months of age NEXLPL-/− mice weigh 50 percent more than their littermates. This phenotype provides convincing evidence that lipoprotein sensing occurs in the brain and is important to energy balance and body weight regulation. Overall, LPL is a fascinating enzyme that contributes in a pronounced way to normal lipoprotein metabolism, tissue-specific substrate delivery and utilization, and to the many aspects of metabolism that relate to cardiovascular disease including energy metabolism, insulin action, body weight regulation, and atherosclerosis.
Numerous studies have documented that racial and ethnic minorities often receive lower-quality care than nonminorities. The same has been found for women. In 1999, the Institute of Medicine (IOM) found significant disparities in the quality of health services received by minorities—even when insurance status, income, age, and severity of condition were comparable. In 2007, the National Committee for Quality Assurance analyzed commercial health plan data and found that women with a history of cardiovascular disease and diabetes were less likely to have their LDL cholesterol under control than men. To help close the disparity gap, the IOM and other groups have recommended that the federal government collect and report data on healthcare access and utilization based on patients’ race, ethnicity, and socioeconomic status; include measures of disparities in performance measures; and monitor progress toward the elimination of healthcare disparities.

No comprehensive action had been taken on these recommendations until July 15, 2008, when President Bush signed into law the Medicare Improvements for Patients and Providers Act (MIPPA) of 2008. Public Law 11-275 includes a provision that requires the Department of Health and Human Services (HHS) to develop approaches for the collection of data that allow for a better evaluation of data on disparities in healthcare services and performance on the basis of race, ethnicity, and gender in the Medicare program.

The legislation gives the Secretary of HHS the authority to (1) develop approaches for data collection that measure disparities in healthcare services, (2) implement strategies that address these disparities in a way that both protects patient privacy and minimizes burdens, and (3) evaluate the success of these efforts in reducing clinical outcome disparities. A report to Congress on data collection approaches is due in 18 months, with implementation of new strategies to occur not later than six months after that. A larger data analysis is expected with recommendations for improving the identification of healthcare disparities in four years, and every four years after that.

The federal government’s leadership in the collection of this data is a pivotal first step to realizing the longstanding national goal of eliminating disparities in health care and can serve as a model for the entire nation. Indeed, quality data broken down by ethnicity and sex does more than shine a light on disparities in health services. It is instrumental to improving the health of all Medicare beneficiaries, many of whom now suffer disproportionately from heart disease, diabetes, and other chronic conditions where disparities have been identified.

The American Heart Association spearheaded the effort to get this law passed and developed a letter that was sent to Congress in support of MIPP and signed by more than 50 groups, including the National Committee for Quality Assurance. AHA staff will monitor the implementation of this new law during the next administration’s reign.