

Article 3

Cedars-Sinai Medical Center, Los Angeles, U.S. Researchers from Cedars-Sinai Medical Center, Los Angeles, U.S., publish recent findings

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2007 JUN 26 - (NewsRx.com) -- Researchers from Cedars-Sinai Medical Center, Los Angeles, U.S., publish recent findings.

This trend article about Cedars-Sinai Medical Center, Los Angeles, U.S., is an immediate alert from NewsRx to identify developing directions of research.

Study 1: Research findings, "Haplotypes in the lipoprotein lipase gene influence high-density lipoprotein cholesterol response to statin therapy and progression of atherosclerosis in coronary artery bypass grafts," are discussed in a new report. According to a study from the United States, "Lipoprotein lipase (LPL) hydrolyzes circulating triglycerides (TGs). We previously showed that 3'-end haplotypes in the LPL gene influence atherosclerosis and insulin resistance."

"This study asked whether these LPL haplotypes influence response to lipid-lowering therapy among 829 subjects from the Post-Coronary Artery Bypass Graft trial. Lipid profiles were obtained at baseline and 4-5 years after treatment with lovastatin. Haplotypes were based on 12 SNPs. The fourth most frequent haplotype, 12-4, was associated with a decreased increment in high-density lipoprotein-cholesterol (HDL-C) following treatment. Haplotypes 12-6, 12-7 and 12-8 were each associated with increased HDL-C response to therapy, and haplotype 12-2 with decreased TG response. The most common haplotype, 12-1, was protective against graft worsening or occlusion. Haplotype 12-4 reduced HDL-C response to lovastatin, possibly consistent with our prior observations of this haplotype as predisposing to coronary artery disease," wrote M.O. Goodarzi and colleagues, Cedars-Sinai Medical Center.

The researchers concluded: "LPL may influence atherosclerosis risk through pleiotropic effects on each aspect of the metabolic syndrome."

Goodarzi and colleagues published their study in The Pharmacogenomics Journal (Haplotypes in the lipoprotein lipase gene influence high-density lipoprotein cholesterol response to statin therapy and progression of atherosclerosis in coronary artery bypass grafts. The Pharmacogenomics Journal, 2007; 7 (1):66-73).

For more information, contact M.O. Goodarzi, Diabetes and Metabolism, Division of Endocrinology, Dept. of Medicine, Cedars-Sinai Medical Center, Los Angeles, CA 9048 USA.

Study 2: David L. Rimoin, MD, PhD, Steven Spielberg chair and director of the Medical Genetics Institute at Cedars-Sinai Medical Center, has been chosen to receive the American Society of Human Genetics' (ASHG) Leadership Award.

Rimoin is the first person to receive the organization's newest award.

"We established the Leadership Award to honor an individual whose professional achievements have fostered and enriched the development of various human genetics disciplines," said **Joann Boughman**, executive vice president of ASHG. "Our nominating committee unanimously selected Rimoin as the inaugural recipient of this award because of his leadership in establishing the American Board of Medical Genetics and the American College of Medical Genetics as well as his internationally recognized research achievements, particularly in the area of skeletal dysplasias."

Rimoin received the award at the ASHG's annual meeting in New Orleans on October 12. "I am honored to accept this tribute from the ASHG and pleased to have played a part in establishing medical genetics as a full medical specialty," Rimoin said.

Rimoin has been affiliated with Cedars-Sinai Medical Center for 20 years. Prior to joining Cedars-Sinai, he served as chief of the Division of Medical Genetics at Harbor-UCLA Medical Center in Torrance, Calif. Before that, he was director of the Genetics Clinic at the Washington University School of Medicine in St. Louis, Missouri.

As the founding president of the American Board of Medical Genetics, Rimoin was instrumental in establishing the educational standards for certification of medical geneticists and the accreditation of training programs throughout the nation. He also served as founding president of the American College of Medical Genetics and then as president of its Foundation, which fosters education in medical genetics to both the practitioner and the public.

Study 3: The homocysteine hypothesis for atherothrombotic cardiovascular disease is not validated in a recent review published in the Journal of the American College of Cardiology.

According to the review from the United States, "Homocysteine has been implicated in promoting atherosclerotic and thrombotic vascular disease. During the last decade, the utility of homocysteine in predicting risk for atherothrombotic vascular disease has been evaluated in several observational studies in a large number of patients."

"These studies show that the overall risk for vascular disease is small, with prospective, longitudinal studies reporting a weaker association between homocysteine and atherothrombotic vascular disease compared to retrospective case-control and cross-sectional studies," wrote S. Kaul and colleagues at Cedars-Sinai Medical Center in Los Angeles.

"Furthermore, randomized controlled trials of homocysteine-lowering therapy have failed to prove a causal relationship. On the basis of these results, there is currently insufficient evidence to recommend routine screening and treatment of elevated homocysteine concentrations with folic acid and other vitamins to prevent atherothrombotic vascular disease," they stated.

"This review outlines the metabolism and pathophysiology of homocysteine, highlights the results of homocysteine observational and interventional trials, and presents areas of uncertainty and potential future work," the authors noted.

Kaul and colleagues published their review in the Journal of the American College of Cardiology (Homocysteine hypothesis for atherothrombotic cardiovascular disease - Not validated. J Am Coll Cardiol, 2006; 48(5): 914-923).

For additional information, contact S. Kaul, Cedars-Sinai Medical Center, Division of Cardiology, 8700 Beverly Blvd., Los Angeles, CA 90048, USA.

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