

**Awardees —
Arteriosclerosis,
Thrombosis, and
Vascular Biology
Conference**

April 28–30, Washington, DC

**ATVB Junior Investigator
Award for Women**

Ranasinghe Silva (Winner)
Hong Lu (Winner)
Rachel Tilley (Winner)
Elke Wagner (Winner)
Greta Hoetzer (Winner)

**Irvine H. Page Young
Investigator Research Award**

Dennis Bruemmer (Winner)
Rohit Khurana (Finalist)
Vyacheslav Korshunov (Finalist)
Angeliki Chroni (Finalist)
Peter Gargalovic (Finalist)

**ATVB Young Investigator
Award in Thrombosis Winner**

Ruhul Abid

**Women's Leadership
Committee Mentoring Award**

Carole Banka

**Recipients of the ATVB New
Investigator Travel Award**

Thomas Barker
Michael Beyea
Wilfried Le Goff
Ramin Zargham
Liam Burnham
Tanya Ramsamy
Qingwei Zhao
Thomas Bell
Boris Boyanovsky
Latika Dhawan
Joanathan Feig
Dardo Ferrara
Sara Police
Stephanie Smith
Elizabeth Westgate
Jenny Zhang
Francesca Zimetti
Zhongmin Zou
James Gallagher

**Jeffrey M. Hoeg Arteriosclerosis, Thrombosis and Vascular Biology
Award for Basic Science and Clinical Research**

Daniel Rader, MD
University of Pennsylvania School of Medicine
Philadelphia, PA

**Molecular Regulation of HDL Metabolism and
Reverse Cholesterol Transport in vivo**

The causal mechanisms of the strong inverse relationship of plasma levels of high density lipoprotein (HDL) cholesterol and the major HDL protein apoA-1 with risk of atherosclerotic cardiovascular disease is complex and highly dependent on the dynamic metabolism of HDL. Unlike the metabolism of apoB-containing lipoproteins, the individual lipid and apolipoproteins components of HDL are largely assembled extracellularly and catabolized independently of each other. Therefore, careful dissection of the pathways of both HDL apolipoprotein and HDL lipid metabolism is critical to understanding the physiological mechanisms by which HDL protects against atherosclerosis. Despite major advances in the cellular and molecular biology and biochemistry of processes such as cellular cholesterol efflux, lipase modification of HDL, and

selective uptake of HDL cholesterol, our understanding of the integrated physiology of HDL metabolism and reverse cholesterol transport (RCT) remains incomplete. A more complete understanding of these processes will be essential for efficient exploitation of the pharmacologic regulation of HDL metabolism and RCT for therapeutic purposes.

Our work has concentrated on the genetic and pharmacologic regulation of HDL metabolism and reverse cholesterol transport in integrated systems, particularly mice and humans. Some recent efforts have been especially focused on the role of phospholipases, particularly endothelial lipase (EL), in HDL metabolism. Since initially reporting EL as a new lipase that hydrolyzes HDL phospholipids and modulates HDL metabolism, we have

continued to explore the physiology and structure-function relationships of EL in HDL metabolism. We have also focused on the molecular regulation of macrophage reverse cholesterol transport in vivo. We developed a novel method to specifically trace RCT from macrophage to feces in mice and used it to demonstrate definitively that apoA-1 overexpression promotes and apoA-1 deficiency impairs macrophage RCT. We have also used this method to study the roles of other gene products such as SR-BI, LCAT, and CETP, as well as pharmacologic interventions, in regulating macrophage RCT in vivo. Insights gleaned from studies of HDL metabolism and RCT in vivo should facilitate the development of new therapeutic approaches to modulating HDL metabolism and RCT in the prevention and regression of atherosclerosis.