

## **Depressive Symptoms and Genetic Influences on Cardiac Outcomes**

Patients with acute coronary syndromes (ACS) who screen positive for depression are at greater risk for subsequent major adverse coronary events (MACE). Depressive symptoms are associated with increased inflammatory protein levels, but only in certain individuals. In a prospective study of ACS patients, we propose to test a biobehavioral model in which inflammatory protein gene polymorphisms interact with depression resulting in even greater increases in inflammatory protein levels than those caused by either gene polymorphisms or depression alone. We expect to identify a well-defined, high-risk subgroup of ACS patients in which the interaction of depression and the genetic polymorphisms identified increases risk of subsequent MACE (myocardial infarctions, revascularization procedures, strokes, and death) more than does either of these factors alone, in part because of their combined effect of increasing inflammatory proteins. Inflammatory proteins and genes measured include: Interleukin (IL) 6, C-reactive Protein (CRP), Tumor Necrosis Factor Alpha (TNF $\alpha$ ), E-Selectin (SELE), and Monocyte Chemoattractant Protein-1 (MCP-1). To test these hypotheses we propose to enroll ACS patients from a single large tertiary care center and obtain blood samples for measurement of inflammatory proteins and genetic testing as soon as possible after hospital admission. Patients will be screened for depression using Beck Depression Inventory (BDI) II scores. Demographic and clinical risk factors will be assessed. Two years of annual follow-up for MACE will be conducted. Data will be analyzed using logistic regression and survival analysis.