

Media Contact: Robin Russell
214-648-3404
robin.russell@utsouthwestern.edu

**UT Southwestern cardiologists identify mechanism
that makes heart disease worse in diabetics**

DALLAS – March 2, 2012 – UT Southwestern Medical Center cardiologists have uncovered how a specific protein’s previously unsuspected role contributes to the deterioration of heart muscle in patients with diabetes. Investigators in the mouse study also have found a way to reverse the damage caused by this protein.

The new research, available online and published in the March 1 issue of the *Journal of Clinical Investigation*, was carried out in the laboratory of Dr. Joseph Hill, director of the Harry S. Moss Heart Center at UT Southwestern.

“If we can protect the heart of diabetic patients, it would be a significant breakthrough,” said Dr. Hill, the study’s senior author who also serves as chief of cardiology at the medical center. “These are fundamental research findings that can be applied to a patient’s bedside.”

Cardiovascular disease is the leading cause of illness and death in patients with diabetes, which affects more than 180 million people around the world, according to the American Heart Association. Diabetes puts additional stress on the heart – above and beyond that provoked by risk factors such as high blood pressure or coronary artery disease, Dr. Hill said.

“Elevated glucose and the insulin-resistant diabetic state are both toxic to the heart,” he said.

Dr. Hill and his colleagues in this study were able to maintain heart function in mice exposed to a high fat diet by inactivating a protein called FoxO1. Previous investigations from Dr. Hill’s laboratory demonstrated that FoxO proteins, a class of proteins that govern gene expression and regulate cell size, viability and metabolism, are tightly linked to the development of heart disease in mice with type 2 diabetes.

“If you eliminate FoxO1, the heart is protected from the stress of diabetes and continues to function normally,” Dr. Hill said. “If we can prevent FoxO1 from being overactive, then there is a chance that we can protect the hearts of patients with diabetes.”

Other UT Southwestern investigators participating in the study were Drs. Pavan Battiprolu,

(MORE)

THE UNIVERSITY OF TEXAS SOUTHWESTERN MEDICAL CENTER AT DALLAS

UT Southwestern Medical School • UT Southwestern Graduate School of Biomedical Sciences • UT Southwestern Allied Health Sciences School
UT Southwestern University Hospitals & Clinics

Office of News and Publications • 5323 Harry Hines Blvd., Dallas, TX 75390-9060 • Telephone 214-648-3404 • Fax 214-648-9119
www.utsouthwestern.edu

Diabetic heart disease – 2

Zhao Wang and Myriam Iglewski, all postdoctoral researchers in internal medicine; Dr. Berdymammet Hojayev, postdoctoral researcher in pathology; Nan Jiang and John Shelton, senior research scientists in internal medicine; Dr. Xiang Luo, instructor in internal medicine; Dr. Robert Gerard, associate professor of internal medicine and molecular biology; Dr. Beverly Rothermel, assistant professor of internal medicine and molecular biology; Dr. Thomas Gillette, assistant professor of internal medicine; and Dr. Sergio Lavandero, visiting professor of internal medicine.

The research was supported by grants from the National Institutes of Health, the American Heart Association, the American Diabetes Association and the Jon Holden DeHaan Foundation.

###

This news release is available on our World Wide Web home page at
<http://www.utsouthwestern.edu/home/news/index.html>

To automatically receive news releases from UT Southwestern via email,
subscribe at www.utsouthwestern.edu/receiveneews