SOUTHWESTERN NEWS

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BLOOD-FLOW STUDY HELPS UT SOUTHWESTERN RESEARCHERS SHED NEW LIGHT ON CHILDHOOD MUSCULAR DYSTROPHY

DALLAS – December 7, 1998 – Researchers at UT Southwestern Medical Center at Dallas have made a critical discovery about blood-flow regulation during exercise and, in the process, have uncovered a clue about a culprit behind Duchenne dystrophy, also known as childhood muscular dystrophy.

In a study published in the Dec. 8 issue of *Proceedings of the National Academy of Sciences*, UT Southwestern researchers detail findings indicating that nitric oxide, one of the substances responsible for increasing blood flow to skeletal muscle during exercise, may derive from a different type of cell than previously thought.

Scientists have believed that the source of nitric oxide was a protein called endothelial nitric oxide synthase, a substance abundant in cells lining blood-vessel walls. But a UT Southwestern team led by Dr. Ronald G. Victor, chief of hypertension, and Dr. Gail Thomas, hypertension researcher, has found another form of the protein, which is widespread in skeletal-muscle fibers, appears to play a major role in the regulation of blood flow to contracting muscles.

Using genetically altered mice with a disease resembling human muscular dystrophy, the team, for the first time, isolated a primary function of nitric oxide in skeletal muscle and pinpointed consequences of a reduction in the protein. In the mice whose levels of skeletal muscle nitric oxide synthase were greatly reduced, the ability to counter normal blood-vessel constriction during muscle contraction was seriously impaired. This impairment may help explain the primary symptom of Duchenne dystrophy, a progressive weakness that occurs as the disease spreads through muscle.

"We're excited about the implications of this latest study and are optimistic that this might help pave the way for further research on possible treatments for muscular dystrophy – a

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terrible disease that still has no cure," said Thomas, an instructor in internal medicine. "It's always exciting when you can solve even a small piece of the puzzle, and we hope that this latest finding will shed light on specific mechanisms contributing to the disease."

Other investigators involved in the project include UT Southwestern researchers Dr. Mikael Sander, hypertension fellow; Dr. Kim Lau, assistant professor of physiology; Dr. James T. Stull, professor and chairman of physiology; and Dr. Paul L. Huang of Massachusetts General Hospital and Harvard Medical School.

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