New molecular signaling cascade increases glucose uptake

August 20 2010

Skeletal muscles combust both lipids and carbohydrates during exercise. The carbohydrates consist of both glycogen stored in the muscles as well as glucose extracted from the blood. Being a major sink for glucose disposal, skeletal muscle represents an important model tissue for studying the intracellular signaling pathways leading to increased glucose transport.

This is important because it is known that the contraction-induced signaling to stimulate glucose transport is distinct from that utilized by insulin. Thus, for individuals in which insulin only has little effect (insulin resistance) the contraction-induced pathway represents an alternative pathway to increase glucose uptake. For pharmaceutical companies this pathway represents a possible and attractive alternative signaling pathway for pharmacological intervention in regulating glucose homeostasis.

Researchers from Department of Exercise and Sport Sciences, University of Copenhagen have in collaboration with scientists at the Joslin Diabetes Center, Harvard University focused on a novel protein called SNARK which was found be activated in skeletal muscle in response to contraction and exercise in both rodents and humans. Furthermore, by the use of transgenic animal models and by over-expressing an inactive mutant of SNARK in mouse skeletal muscle, it could be shown that contraction-induced glucose uptake was severely blunted by 40-50% compared with control animals.

The data in this study clearly support a role for SNARK in regulating glucose transport during muscle contraction and exercise, but it also strongly suggests that multiple, or redundant signals may mediate the effects of contraction on activating glucose transport.

These data have been published in *Proceedings of the Nation Academy of Sciences* (*PNAS*) on 16. August 2010 in an article called "Sucrose nonfermenting AMPK-related kinase (SNARK) mediates contraction-stimulated glucose transport in mouse skeletal muscle".

Provided by University of Copenhagen