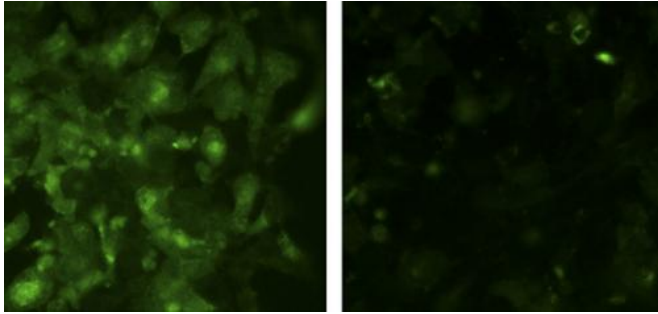


# How brown fat fuels up to combat type 2 diabetes and obesity

10 November 2014



Cold temperature-induced hormones cause GLUT1 (green) to be transported to the surface of brown fat cells (left). Transport is blocked when the mTORC2 pathway is inhibited (right). Credit: Olsen et al., 2014

A newly identified signaling pathway that stimulates glucose uptake in brown fat cells might be useful for treating type 2 diabetes and obesity, according to a study in *The Journal of Cell Biology*.

When the body encounters cold temperatures, the sympathetic nervous system activates adrenoceptors on the surface of [brown fat cells](#) to stimulate glucose uptake from the bloodstream. Brown fat cells then use this glucose as a fuel source to generate body heat. Glucose uptake also can be induced by insulin. However, although insulin-stimulated glucose-uptake is well understood, the mechanisms involved in the adrenoceptor-dependent process have been unclear.

Now, researchers in Sweden reveal that the mTORC2 [signaling pathway](#) is the key regulator of adrenoceptor-stimulated [glucose uptake](#) in brown fat tissue in mice. The pathway, which involves a protein kinase called mTOR, stimulates the transport of a glucose-importing protein called GLUT1 to the surface of brown fat cells.

"One of the most interesting characteristics of this newly discovered signal pathway is that it differs from the signal pathway triggered by insulin," says senior author Tore Bengtsson from the Department of Molecular Biosciences, Wenner-Gren Institute, Stockholm University. "This means that the [signal pathway](#) in brown fat can most likely be activated even in patients with type 2 diabetes, where insulin signaling is impaired."

In addition to being an effective tool for controlling [blood sugar levels](#) in type 2 diabetes patients, these findings suggest that stimulating the mTORC2 pathway to take advantage of the energy-burning power of brown fat might be effective as a weight loss therapy.

**More information:** Olsen, J.M., et al. 2014. *J. Cell Biol.* doi:10.1083/jcb.201403080

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