

# Bad cholesterol inhibits the breakdown of peripheral fat

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The so called bad cholesterol (LDL) inhibits the breakdown of fat in cells of peripheral deposits, according to a study from the Swedish medical university Karolinska Institutet. The discovery reveals a novel function of LDL as a regulator of fat turnover besides its well-established detrimental effects in promoting atherosclerosis.

The study, which is a collaboration of two research groups at Karolinska Institutet, is published in the open-access journal PLoS ONE. It shows that LDL cholesterol slows the rate of fat breakdown (i.e. lipolysis) in adipocytes, the peripheral cells responsible for fat storage. Previously, it has been known that release of free fatty acid from the peripheral fat to the blood stream increases the synthesis of LDL precursors in the liver.

"The results of our study provide evidence of a reciprocal link between the liver and peripheral fat regulating fat turnover", says study-initiator Dr Johan Björkegren.

The discovery also opens up for new theories for the well-established association between blood lipids and the metabolic syndrome.

"If proven of general physiological importance, therapies lowering LDL, as for instances Statins, may also affect the turnover of peripheral fat," continues Dr Björkegren.

The study and has been performed on cell cultures and tissues from humans as well as mouse models with different levels of LDL. The

inhibitory effect was also shown to be dependent on LDL receptors on the surface of the fat cells.

"The exact intracellular mechanism for how the binding of LDL to the surface of the fat cells inhibits the breakdown of intracellular fat remains to be revealed", say project leader Dr Josefin Skogsberg.

Publication: 'ApoB100-LDL Acts as a Metabolic Signal from Liver to Peripheral Fat Causing Inhibition of Lipolysis in Adipocytes" Josefin Skogsberg, Andrea Dicker, Mikael Rydén, Gaby Åström, Roland Nilsson, Hasanuzzaman Bhuiyan, Sigurd Vitols, Aline Mairal, Dominique Langin, Peteris Alberts, Erik Walum, Jesper Tegnér, Anders Hamsten, Peter Arner, Johan Björkegren, PLoS ONE, 20 November 2008

Source: Karolinska Institutet

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