Slimming gene regulates body fat

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Scientists at the University of Bonn, Germany, have discovered a previously unknown fruit fly gene that controls the metabolism of fat. Larvae in which this gene is defective lose their entire fat reserves. Therefore the researchers called the gene 'schlank' (German for 'slim'). Mammals carry a group of genes that are structurally very similar to 'schlank'. They possibly take on a similar function in the energy metabolism. The scientists therefore have hopes in new medicines with which obesity could be fought. Their research bas been published in *The EMBO Journal*.

If scientists decipher the function of a gene, they are allowed to name it. With the fruit fly Drosophila there is a rather paradox convention. The names always indicate what the fly looks like if the respective gene is defective. That is also the case with the schlank gene. If it is unimpaired the fly larva can build up fat reserves. It becomes fat. 'Larvae with a mutation of schlank, however, remain slim,' Professor Michael Hoch from the University of Bonn explains. 'In extreme cases the defect can even lead to death.'

Together with Dr. Reinhard Bauer and other employees the development biologist has explored what exactly 'schlank' does. According to their research the gene contains the instructions of what is known as ceramide synthase. Ceramides serve as raw materials for the gauzy membranes that enclose all of the cells in the body. Moreover, schlank also has a regulatory function. It promotes lipid synthesis and at the same time inhibits the mobilisation of fat from the fat reserves.

Mouse gene saves fly larvae

There is a chance that this is not only the case in fruit flies. Humans also produce ceramide synthases however not just one as Drosophila does but rather as many as six different ones. For this purpose humans rely on a group of genes so-called Lass genes. Ceramide synthases are extremely important for animals. Mutations in the corresponding genes lead to severe metabolic disorders and to malfunctions of organ systems. That is why our Lass genes look surprisingly similar to the schlank gene of fruit flies.

This resemblance is so striking that Lass genes from mice can partially compensate for the defect schlank gene in mutant flies. 'We introduced a mouse Lass gene in mutant Drosophila larvae,' Michael Hoch says. 'Normally the larvae died immediately after hatching. Thanks to the Lass gene they resumed building up body fat and survived until the next development stage.'

Up to now, the Lass genes of mammals have not been connected with the regulation of the lipid metabolism. 'But due to the strong parallels with schlank we think such a function is very probable,' Professor Hoch presumes. 'If this is the case they would be a promising approach for new medications for obesity.'

More information: *The* EMBO Journal, doi: 10.1038/emboj.2009.305

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