

Anti-stress compound reduces obesity and diabetes

December 13 2017



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For the first time, scientists from the Max Planck Institute of Psychiatry in Munich could prove that a stress protein found in muscle has a diabetes promoting effect. This finding could pave the way to a completely new treatment approach.

For some time, researchers have known that the protein FKBP51 is

associated with depression and anxiety disorders. It is involved in the regulation of the stress system – when the system does not function properly; mental disorders may develop. Now, researchers at the Max Planck Institute of Psychiatry have discovered a new, surprising role for this protein: It acts as a molecular link between the stress regulatory system and metabolic processes in the body.

"FKBP51 influences a signaling cascade in [muscle tissue](#), which with excessive calorie intake leads to the development of [glucose intolerance](#), i.e., the key indicator of diabetes type 2," project leader Mathias Schmidt summarizes. An unhealthy diet, rich in fat means stress for the body. If FKBP51 is increasingly produced in the [muscle](#) it leads to reduced absorption of glucose – as a result, diabetes and obesity may develop.

If FKBP51 is blocked, diabetes will not develop, even if too many calories are consumed or the body is still stressed. Less FKBP51 in the muscle tissue means reduced glucose intolerance and thus maintenance of normal metabolism.

Antagonist provides novel treatment approach

The [protein](#) FKBP51 can be pharmacologically blocked by antagonist compounds that were developed at the Max Planck Institute by Felix Hausch (presently at University of Darmstadt). In collaboration with the scientists at the Technical University Darmstadt and funded by the Bavarian State Ministry of Economic Affairs and Media, Energy and Technology, these compounds will be further developed for use in clinical trials. "These findings may provide a completely new treatment approach for [diabetes](#) and other metabolic diseases," states Alon Chen, Director at the Max Planck Institute of Psychiatry.

More information: Georgia Balsevich et al. Stress-responsive

FKBP51 regulates AKT2-AS160 signaling and metabolic function, *Nature Communications* (2017). [DOI: 10.1038/s41467-017-01783-y](https://doi.org/10.1038/s41467-017-01783-y)

Provided by Max Planck Society

Citation: Anti-stress compound reduces obesity and diabetes (2017, December 13) retrieved 3 May 2024 from <https://medicalxpress.com/news/2017-12-anti-stress-compound-obesity-diabetes.html>

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