Researchers validate the potential of a protein for the treatment of type 2 diabetes

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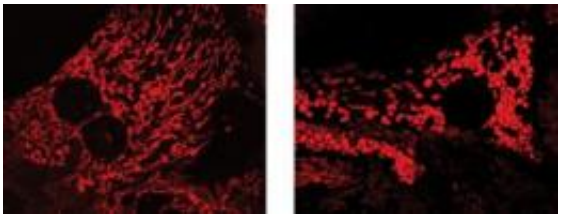

This shows hepatic cells isolated from healthy mice and mice deficient in Mitofusin 2 (right). Morphology of mitochondria (red), more rounded, is altered in the image on the right Credit: Antonio Zorzano laboratory (IRB Barcelona)

Researchers at the Institute for Research in Biomedicine (IRB Barcelona, Spain)) have discovered that deficiency of a single protein, Mitofusin 2, in muscle and hepatic cells of mice is sufficient to cause tissues to become insensitive to insulin, thus producing an increase in blood glucose concentrations. These are the two most common conditions prior to development of diabetes type 2. Published in this week's issue of *Proceedings of the National Academy of Sciences* (*PNAS*), the study validates Mitofusin 2 as a possible target for the treatment of diabetes type 2.

"Resistance to insulin plays a key role in the development of diabetes mellitus, dyslipidemia (alteration of lipid concentrations) and obesity. Mitofusin 2 may provide a specific target for the development of drugs that could hold back a disease that affects millions of people

worldwide", explains the head of the study, Antonio Zorzano, full professor of the University of Barcelona, coordinator of the Molecular Medicine Programme at IRB Barcelona, and head of the Heterogenic and Polygenic Diseases lab at the same centre.

The World Health Organization estimates that there will be 350 million people suffering from diabetes in 2020. Diabetes type 2 accounts for 90% of diabetes cases and is due to a great extent to excess body weight, poor nutrition and sedentary lifestyle. According to the Spanish Society of Diabetes, in Spain 6.5% of the current population between 30 and 65 years has this disease and about 11.6 % of Spaniards are at risk of developing it.

Previous studies performed at IRB Barcelona demonstrate that both obese and diabetes type 2 subjects have low levels of muscle Mitofusin 2. This protein controls the insulin signaling pathway in the liver and muscles. The scientists have observed that deficiency of this protein causes alterations in mitochondria and the endoplasmic reticulum, two crucial organelles for correct cell functioning. "We have shown that the accumulation of dysfunctions in these two structures alters cell behavior and favors the appearance of pre-diabetes symptoms", say the main authors of the article, David Sebastián and María Isabel Hernández-Álvarez, post-doctoral fellows in Zorzano's team.

More information: *PNAS* (2012): Doi:10.1073/pnas.1108220109

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