

FGF21 hormone, key to control obesity, also protects against heart diseases in mice

18 June 2013

A research group has found that FGF21, an endocrine factor which reduces glucose levels, protects against cardiac diseases in mice. The research, published online on the journal *Nature Communications*, was led by Francesc Villarroya, professor from the Department of Biochemistry and Molecular Biology of the UB and Director of the Institute of Biomedicine of the University of Barcelona (IBUB), affiliated centre with the campus of international excellence BKC. Anna Planavila, first author of the paper, from that Department of the UB, the experts Luigi Gabrielli and Marta Sitges (IDIBAPS-Hospital Clínic de Barcelona) and other international experts also collaborated in the research.

Fibroblast growth factor 21 (FGF21), mainly secreted by the liver, is a protein which acts as a metabolic regulator and plays a key role as an antidiabetic and antiobesity agent. In 2010, the cover of the journal *Cell Metabolism* echoed a finding made by the UB research group headed by Dr Villarroya: FGF21 activates thermogenesis in brown adipose tissue—which governs energy expenditure and heat production in the body—, what promotes the burning of calories to release heat, dissipating then large amounts of energy.

FGF21: a cardioprotective agent in mice

According to Professor Francesc Villarroya, member of the Biomedical Research Networking Centres on Physiopathology of Obesity and Nutrition (CIBERObn), "the research has contributed to describe the protecting role against cardiac hypertrophy that FGF21 plays in mice". The research group compared the role of FGF21 in [cardiac tissue](#) in a group of [knockout mice](#) (those lacking FGF21) and in another group which perfectly express the factor. They observed that mice lacking FGF21 are more prone to develop [cardiac diseases](#).

Anna Planavila, expert on metabolism and cardiac

functions study, explains that "echocardiography tests, carried out together with a group from Hospital Clínic, proved how knock-out mice's [heart function](#) had got worse as they showed dilatation and [cardiac hypertrophy](#), electrocardiography waves due to alterations of the mechanisms of cardiac systole and diastole mechanisms, etc. These effects were also observed at histological and gene expression levels".

The heart is able to produce FGF21

Besides FGF21 new function, the paper reveals new scientific findings about cardiac metabolism and physiology. Authors affirm that the heart is also able to produce this factor which works as a protective agent against heart stress situations. "Previous knowledge—Villarroya adds— had already stated that FGF21 is synthesized by the liver, the skeletal muscle and the brown adipose tissue to speed up glucose uptake and energy metabolism. The research has also unveiled that the cardiac muscle produces the endocrine factor too". Experts state that the heart produces the factor under basal conditions. If the heart undergoes more physiological stress, FGF21 production is increased as a protective response. "Unlike the liver, the skeletal muscle and the [brown adipose tissue](#), cardiac cells ability to produce this factor has a local and self-protection effect", Villarroya remarks. The pre-clinical study published on *Nature Communications* shed new light on the metabolic control of molecular signalling pathways of diabetes, obesity and adipose tissue inflammation. The research can contribute to establish FGF21 as a potential tool in the development of new strategies to prevent and treat cardiac damage.

More information: Planavila, A. et al. Fibroblast growth factor 21 protects against cardiac hypertrophy in mice, *Nature Communications*, Volume:4, Article number:2019 [DOI:10.1038/ncomms3019](https://doi.org/10.1038/ncomms3019).

Provided by University of Barcelona

APA citation: FGF21 hormone, key to control obesity, also protects against heart diseases in mice (2013, June 18) retrieved 4 June 2022 from <https://medicalxpress.com/news/2013-06-fgf21-hormone-key-obesity-heart.html>

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.