



PRINCIPE FELIPE
CENTRO DE INVESTIGACION

THE FUTURE OF BIOMEDICAL RESEARCH CIPF Lecture Series

Mitochondrial dynamics and its role in the pathophysiology of metabolic disorders

Speaker: **Dr. Antonio Zorzano**

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Date: **30/11/18- 12:30 h**

Place: Sal6n de Actos CIPF

Abstract: Mitochondrial fusion and fission are key processes that regulate mitochondrial morphology. Mitochondrial fusion is catalyzed by MFN1, MFN2 (Mitofusins) and OPA1 proteins in human cells. MFN2 protein plays a complex set of functions. It regulates mitochondrial morphology, and, in addition, also controls the morphology and function of the endoplasmic reticulum.

Expression of MFN2 is exquisitely regulated in tissues. Thus, it is induced in skeletal muscle in response to chronic exercise and after exposure to cold. In contrast, MFN2 is repressed in muscle or in the hypothalamus of mice fed a high fat diet. On the other hand, MFN2 is repressed in type 2 diabetic patients or in obese subjects. In turn, changes in MFN2 expression have a marked impact on mitochondrial metabolism. Skeletal muscles obtained from MFN2 knockout mice show a reduction in respiratory control, glucose oxidation, and expression of some subunits of oxidative phosphorylation. MFN2 deficiency causes major alterations in muscle biology. Thus, skeletal muscle-ablated MFN2 KO mice show susceptibility to develop glucose intolerance and insulin resistance in response to a high-fat diet or to aging. In addition, Mfn2-deficient muscle show atrophy and a gene signature linked to aging. In summary, available data indicate that the MFN2 protein regulates metabolic homeostasis, insulin signaling, and maintenance of muscle mass.

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