METABOLISM AND CALCIUM SIGNALLING IN BRAIN
ROSER MASGRAU JUANOLA

PROFILE

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Roser Masgrau obtained her PhD degree in Biochemistry from the UAB at 2000 under the supervision of Professor Fernando Picatoste and after completing part of her CNS signalling research at the laboratory of Professor Stefan Nahorski at the Department of Pharmacology of the University of Leicester (UK). She then moved to the University of Oxford to work with Professor Antony Galione and Dr. Steve Aschcorft to elucidate NAADP as a new second messenger mediating insulin secretion. In, 2003 she was awarded a prestigious Marie Curie Fellowship to join the laboratory of Dr. Rebecca Matsas at the Hellenic Pasteur Institute and continue her studies on signalling in the brain. In 2005, she joined the Institut de Neurociències and Department of Biochemistry and Molecular Biology at the UAB thanks to a Ramon y Cajal Senior Researcher Tenure Track. From 2018 she is Associate Professor.

RESEARCH

RESEARCH INTERESTS

Our group is interested in signalling and metabolism in the brain at multiple levels, from single astrocytes, astrocyte-neuron interactions and neuronal circuits, to control of behaviour and memory. We are also interested in how these processes are altered in neurodegeneration, in particular Alzheimer's disease, to elucidate multi-targeted and multi-cellular therapies

STRATEGIC OBJECTIVES

The general objectives of our research group are:

1. To find out molecular mechanisms by which astrocytes regulate memory formation
2. To establish astrocytes as a therapeutic target in neurodegeneration

MAIN RESEARCH LINES

1.- Metabolism of Astrocytes- We have shown that astrocytes, a part of being glycolytic, are also fatty acid and amino-acid oxidative cells. We are currently studying the molecular mechanism of the regulation of fatty acid oxidation in astrocytes and its interplay with calcium signaling. We also investigate how fatty acid metabolism contributes to astrocytic excitability, neuronal network activity and memory processes.

2.-Astrocytes in Alzheimer- Our purpose is to characterize how expression of ApoE4, the main genetic risk of Alzheimer, regulates calcium signaling, lipidome, metabolism and lysosomal funcions in astrocytes.

LAB FEATURED PUBLICATIONS:

Larramona-Arcas R, González-Arias C, Perea G, Gutiérrez A, Vitorica J, García-Barrera T, Gómez-Ariza JL, Pascua-Maestro R, Ganfornina MD, Kara E, Hudry E, Martinez-Vicente M, Vila M, Galea E, Masgrau R. Sex-dependent calcium hyperactivity due to lysosomal-related dysfunction in astrocytes from APOE4 versus APOE3 gene targeted **replacement** mice. Mol Neurodegener.15:35, 2020. doi: 10.1186/s13024-020-00382-8.

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Eraso-Pichot A, Larramona-Arcas R, Vicario-Orri E, Villalonga R, Pardo L, Galea E, Masgrau R. CREB decreases astrocytic excitability by modifying subcellular calcium fluxes via the sigma-1 receptor. Cell Mol Life Sci. 2017; 74(5):937-950. *doi: 10.1007/s00018-016-2397-5.*

Pardo L, Schlüter A, Valor LM, Barco A, Giralt M, Golbano A, Hidalgo J, Jia P, Zhao Z, Jové M, Portero-Otin M, Ruiz M, Giménez-Llort L, Masgrau R, Pujol A, Galea E. Targeted activation of CREB in reactive astrocytes is neuroprotective in focal acute cortical injury. Glia. 2016; 64(5):853-74. *doi: 10.1002/glia.22969.*

Barceló-Torns M, Lewis AM, Gubern A, Barneda D, Bloor-Young D, Picatoste F, Churchill GC, Claro E, Masgrau R. NAADP mediates ATP-induced Ca2+ signals in astrocytes. FEBS Lett. 2011;585(14):2300-6. *doi: 10.1016/j.febslet.2011.05.062.*

España J, Valero J, Miñano-Molina AJ, Masgrau R, Martín E, Guardia-Laguarta C, Lleó A, Giménez-Llort L, Rodríguez-Alvarez J, Saura CA beta-Amyloid disrupts activity-dependent gene transcription required for memory through the CREB coactivator CRTC1. J Neurosci. 2010; 30(28):9402-10. *doi: 10.1523/JNEUROSCI.2154-10.2010.*

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Yamasaki M, Masgrau R, Morgan AJ, Churchill GC, Patel S, Ashcroft SJ, Galione A. Organelle selection determines agonist-specific Ca2+ signals in pancreatic acinar and beta cells. J Biol Chem. 2004;279(8):7234-40. *doi: 10.1074/jbc.M311088200.*

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