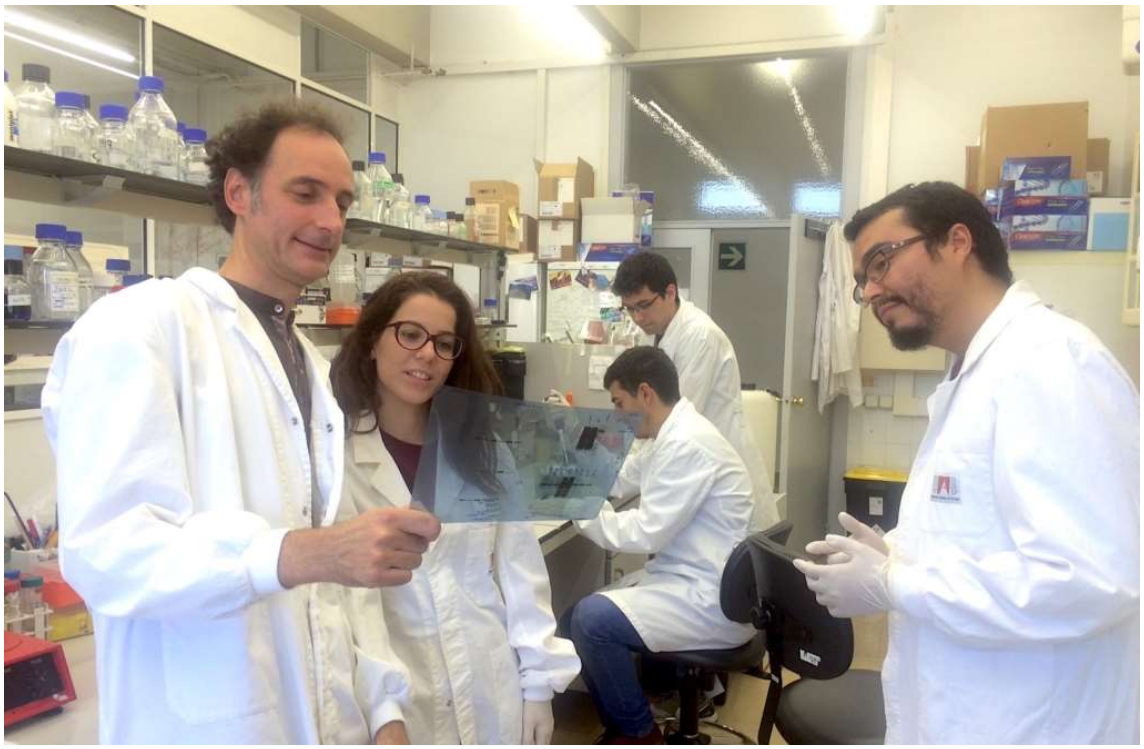
NEUROBIOLOGY OF ALZHEIMER'S DISEASE
CARLES SAURA



PROFILE

Alzheimer´s disease (AD) is characterized by accumulation of phosphorylated tau and β-amyloid (Aβ) peptides in brain regions encoding memory. Aβ is generated by presenilin/γ-secretase-mediated cleavage of the β-amyloid precursor protein. Our investigations focus on molecular mechanisms regulated by presenilins and Aβ that alter gene expression programs leading to synaptic dysfunction and memory loss in Alzheimer´s disease.

RESEARCH

RESEARCH INTERESTS

Neurodegenerative disorders, including Alzheimer's disease, are the main cause of life dissability, memory loss and neurosychiatric symptoms in the elderly. Understanding the pathological mechanisms of brain dysfunction is mandatory to develop novel therapeutic strategies for cognitive and neuropsychiatric disorders. Our lab combines molecular approaches and cutting-edge genomic, transcriptomic and proteomic methodologies to better understand the molecular pathways underlying neuronal dysfunction, memory loss and neurodegeneration in Alzheimer's disease**.**

STRATEGIC OBJECTIVES

Our research efforts are currently focused on understanding how brain circuits regulate memory encoding and storage in order to unravel the cellular and molecular mechanisms involved in brain dysfunction, memory loss and neurodegeneration in age-related cognitive disorders, particularly in Alzheimer's disease.

Saura's lab use cell-specific transcriptomics and high-resolution imaging technologies to investigate the transcriptional pathways that regulate synaptic plasticity underlying cognition and memory, which allows a better understanding of the mechanisms mediating gene expression changes causing synaptic dysfunction and memory loss in Alzheimer's disease. We employ pioneering approaches to generate novel cell-specific transgenic and knockout mouse models of neurodegeneration that are used for testing new therapeutic strategies (pharmacological, gene therapy, cognitive stimulation…) for future treatment of age-related dementia disorders. Our research projects provide knowledge of novel molecular targets and therapeutic strategies for early treatment of Alzheimer's disease and related neurological disorders.

MAIN RESEARCH LINES

1.Cellular and pathological mechanisms in Alzheimer's disease

2.Novel research tools and models of neurodegeneration

3.Development of novel therapies for Alzheimer's disease and dementias

LAB FEATURED PUBLICATIONS

Parra-Damas A., Chen M., Enríquez-Barreto L., Ortega L., Acosta S., Camats Perna J., Fullana N., Aguilera J., Rodríguez-Alvarez J., and Saura C.A. CRTC1 function during memory encoding is disrupted in neurodegeneration (2017). *Biol Psychiatry* 81 (2): 111-123

Van der Jeugd A., Parra-Damas A., Baeta-Corral R., Soto-Faguás CM., Ahmed T., LaFerla FM., Giménez Llort L., D'Hooge R., Saura C.A. Reversal of memory and neuropsychiatric-like symptoms and reduced tau pathology by selenium treatment in 3xTg-AD mice (2018). *Sci Reports,* 8: 6431

Javier-Torrent M., Marco S., Rocandio D., Pons-Vizcarra M., Janes P.W., Lackman M, Egea J. and Saura C.A. Presenilin/γ-secretase-dependent EphA3 processing mediates axon elongation through non-muscle myosin IIA (2019). *eLife*. 8:e43646

Parra-Damas A. and Saura C.A. Synapse-to-nucleus signaling in neurodegenerative and neuropsychiatric disorders (2019). *Biol Psychiatry* 86 (2): 87-96

Florido A., Velasco ER., Soto-Faguás CM., Gómez-Gómez A., Perez-Caballero L., Molina P., Nadal R., Pozo OJ., Saura C.A., Andero, R. Sex differences in fear memory consolidation via Tac2 signaling in mice (2021). *Nat Comm* 12(1):2496

Soto-Faguás CM., Sánchez-Molina, P. and Saura C.A. Loss of presenilin function enhances tau phosphorylation and aggregation in mice (2021). *Acta Neuropath Comm*. 9(1):162

Saura C.A., Deprada A., Capilla-López MD, Parra-Damas A. Revealing cell vulnerability in Alzheimer's disease by single-cell transcriptomics (2023). *Sem Cell Dev Biol*.;S1084-9521(22)00164-1