SEMINARI

"Investigating the molecular substrates of Parkinson's disease"

Ponent:

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Coordina:

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Lloc: aula 0.22, planta baixa part nova Unitat Docent

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Abstract

Parkinson's disease (PD) is the second most common neurodegenerative disease after Alzheimer's dementia and the most common neurodegenerative movement disorder, affecting about 1% of the population above the age of 60. So far, the most consistent risk factor for developing PD is increasing age. With the increase in life expectancy that our society has experienced in the last decades, the incidence and prevalence of PD is expected to increase substantially, which represents a major economic and social burden related to this disabling illness. Clinically, classical PD is characterized by several motor symptoms attributed to a dramatic loss of dopamine (DA)-containing neurons in the substantia nigra pars compacta (SNpc), which leads to DA depletion in the striatum. The neurodegenerative process, however, extends well beyond the nigrostriatal dopaminergic system, which accounts for the increasingly critical role of "non-dopaminergic" symptoms to the quality of life of PD patients. Thus far, the most potent treatment for PD remains the administration of a precursor of DA, levodopa, which alleviates almost all motor PD symptoms although it does not slow or halt disease progression. Therefore, there is an urgent need to acquire a deeper understanding of the pathogenesis of PD in order to identify new molecular targets for potential therapeutic intervention. Our group consists of basic researchers and clinicians so that we can tackle the disease transversally and from different disciplines. Specifically, we are currently mostly focused on the role of neuromelanin, autophagy, the immune system and the microbiota in the development of Parkinson's disease. To this end, we use in vitro and in vivo experimental models of PD-related neurodegeneration, including cell lines, rodent models and human-derived samples from patients affected by PD.

Short bio

My scientific career has been focused in understanding the molecular and cellular mechanisms underlying human diseases affecting the central nervous system, both with a neurodevelopmental or a neurodegenerative etiology. I completed my PhD in the laboratory of Dr. Mariona Arbonés at the Center for Genomic Regulation (CRG, Barcelona) contributing to the field of Down Syndrome and gene-dosage imbalance effects in the central nervous system (Laguna et al., 2008, Developmental Cell; Laguna et al. 2013, Human Molecular Genetics; Aranda et al. 2008, Mol Cell Biology; Ferrón et al. 2010, Cell Stem Cell; Barallobre et al. 2014, Cell Death and Disease). Later, I joined the laboratory of Professor Thomas Perlmann at the Karolinska Institutet (Stockholm, Sweden) and developed projects aimed to elucidate the role of transcription factors in dopaminergic neurons and their contribution to the pathology of Parkinson's disease (PD) (Laguna et al. 2015 Nature Neuroscience; Decressac et al. 2012, Science Translational Medicine; Panmann et al. 2014, Cell Reports; Doucet-Beaupré et al. 2016, PNAS). In 2014, I joined the Neurodegenerative Diseases Group of the Vall d'Hebron Research Institute (VHIR, Barcelona) and participate in the projects of the group related mainly to the role of autophagy, synuclein and neuromelanin in PD (Xicoy et al. 2019 Cells; Vila et al. 2019 Autophagy; Carballo-Carbajal*, Laguna* et al. 2019, Nature Communications; Lassot et al. Cell Reports 2018). Also, I lead a new line of research in our group aimed at the study of patients' biological samples in order to: (i) understand the early molecular mechanisms of PD etiology / pathogenesis, (ii) identify new molecules as biomarkers, and (iii) identify new targets for the development of therapeutic strategies. Since 2016, I received an independent appointment to follow these research lines, first funded by the Spanish Ministry of Economy and Competitiveness (MINECO) and currently by the La Caixa Banking Foundation.