



MARIE SKŁODOWSKA-CURIE POSTDOCTORAL FELLOWSHIPS 2023

EXPRESSION OF INTEREST FOR HOSTING MARIE CURIE FELLOWS

HOST INSTITUTION

NOVA Medical School (iNOVA4Health Research Unit)

RESEARCH GROUP AND URL

Neuronal Trafficking in Aging

<https://www.nms.unl.pt/en-us/research/research-groups/research-group/n/neuronal-trafficking-in-aging>

SUPERVISOR (NAME AND E-MAIL)

Cláudia Guimas Almeida, claudia.almeida@nms.unl.pt

SHORT CV OF THE SUPERVISOR

Claudia Guimas Almeida graduated in Biochemistry and has a master's degree and a Ph.D. in neuroscience from the University of Lisbon. Her doctoral research, which focused on beta-amyloid-dependent synaptic and endosomal dysfunction in Alzheimer's disease, was carried out at the renowned laboratory of Dr. Gunnar Gouras in New York (Weill Cornell Medical College). Dr. Almeida went on to receive postdoctoral training in cell biology as an EMBO and Marie Curie fellow at Dr. Daniel Louvard's laboratory in Paris (Institut Curie). She is an independent researcher since 2013 at NOVA Medical School, supported by the Portuguese Science Foundation (FCT). Dr. Almeida's research has led to 27 publications on intracellular trafficking mechanisms in healthy, aged, and Alzheimer's disease neurons, with 4835 citations.

5 SELECTED PUBLICATIONS

- Burrinha T, Cunha C, Hall MJ, Lopes-da-Silva M, Seabra MC, Guimas Almeida C. Deacidification of endolysosomes by neuronal aging drives synapse loss. *Traffic*. 2023 May 23. doi: 10.1111/tra.12889.
- Burrinha T, Martinsson I, Gomes R, Terrasso AP, Gouras GK, Almeida CG. Upregulation of APP endocytosis by neuronal aging drives amyloid-dependent synapse loss. *J Cell Sci*. 2021 May 1;134(9):jcs255752. doi: 10.1242/jcs.255752.
- Burrinha T, Guimas Almeida C. Aging impact on amyloid precursor protein neuronal trafficking. *Curr Opin Neurobiol*. 2022 Apr;73:102524. doi: 10.1016/j.conb.2022.102524. Perdigão C, Barata MA, Araújo MN, Mirfakhar FS, Castanheira J, Guimas Almeida C. Intracellular Trafficking Mechanisms of Synaptic Dysfunction in Alzheimer's Disease. *Front Cell Neurosci*. 2020 Apr 17;14:72. doi: 10.3389/fncel.2020.00072. Ubelmann F, Burrinha T, Salavessa L, Gomes R, Ferreira C, Moreno N, Guimas Almeida C. Bin1 and CD2AP polarise the endocytic generation of beta-amyloid. *EMBO Rep*. 2017 Jan;18(1):102-122. doi: 10.15252/embr.201642738.

PROJECT TITLE AND SHORT DESCRIPTION

Endolysosomal dysfunction in aging and Alzheimer's disease

Our goal is to uncover druggable molecular mechanisms that underlie synapse dysfunction early on in a still reversible cellular phase of neurodegeneration. For that we are investigating the pathogenicity of



endosomal genetic risk factors associated with late-onset Alzheimer's disease and how neuronal aging impacts the endolysosomal system.

We rely on advanced quantitative microscopy, from live-cell Imaging to super-resolution, to perform mechanistic studies using cultures of primary mouse cortical neurons, neuronal cell lines, human neurons derived from hiPSCs and aged mice. Moreover, we are establishing neuronal models that recapitulate late-onset AD early cytopathology.

SCIENTIFIC AREA WHERE THE PROJECT FITS BEST*

Life Sciences (LIF)