



MARIE SKŁODOWSKA-CURIE POSTDOCTORAL FELLOWSHIPS 2026
EXPRESSION OF INTEREST FOR HOSTING MARIE CURIE FELLOWS

HOST INSTITUTION

NOVA Medical School (NMS)

RESEARCH GROUP AND URL

Lysosomes and Disease Group (<https://www.nms.unl.pt/en-us/research/research/research-groups/research-group/n/lisossomas-em-patologias-cronicas-humanas>)

SUPERVISOR (NAME AND E-MAIL)

Otilia V. Vieira, otilia.vieira@nms.unl.pt

SHORT CV OF THE SUPERVISOR

Otilia Vieira (OV) completed her PhD in atherosclerosis prevention under the supervision of Prof. Robert Salvayre (University Paul Sabatier, Toulouse, France) and Prof. Leonor Almeida (University of Coimbra, Portugal). She carried out her first postdoctoral project on host–pathogen interactions under the supervision of Prof. Sergio Grinstein at the University of Toronto, Canada. Her second postdoctoral position, focused on Trans-Golgi Network sorting in polarized cells, was conducted under the supervision of Prof. Kai Simons at the Max Planck Institute for Molecular Cell Biology and Genetics, Dresden, Germany. In 2006, she returned to Portugal to establish an independent research group at the Center for Neurosciences and Cell Biology, University of Coimbra. Her research focused on plasma membrane resealing by lysosomes and its role during Mycobacterium tuberculosis infection, as well as on the use of surfactants as topical microbicides to prevent sexually transmitted infections (STIs). In 2014, she was awarded the prestigious FCT Investigator Award (Consolidator level) and joined NOVA Medical School, NOVA University Lisbon. She is currently the Principal Investigator of the Lysosomes and Disease group ([link](#)), an Assistant Professor with tenure, and holds a Habilitation degree. At NOVA, she leads translational research on chronic diseases—particularly cardiovascular diseases (CVD)—leveraging her expertise in atherosclerosis and membrane trafficking. Her goal is to elucidate the cellular and molecular mechanisms of atherosclerosis and translate this knowledge into predictive tools and novel therapies for CVD. Her multidisciplinary team includes cardiologists, molecular cell biologists, and biophysical chemists, comprising one senior scientist (ranked among the World’s Top 2% most highly cited researchers), one assistant researcher, and five PhD students. She has published 52 peer-reviewed articles—12 as first author, 24 as last author, and 26 as corresponding author—in high-impact journals such as PNAS, Nature Cell Biology, Journal of Cell Biology, EBioMedicine, and Matter. Four of her papers were highlighted in Journal of Cell Biology, one in PNAS, one in EBioMedicine, and three were recommended by F1000Prime. According to Scopus, her work has been cited over 6,600 times, with an h-index of 33. She is also listed among the World’s Top 2% of highly cited researchers. Since 2006, she has supervised nine postdoctoral researchers, twelve PhD students, and three MSc students. She has secured more than €2.4 million in competitive research funding, including the coordination of an international consortium with Harvard Medical School. She is the recipient of multiple awards and a provisional international patent, and collaborates with a multinational pharmaceutical company on the development of first-in-class therapies for chronic diseases. She serves as an Editor for Scientific Reports (Nature Publishing Group) and as an Associate Editor for Frontiers in Cell and Developmental Biology. She frequently acts as an examiner for MSc and PhD theses and as a reviewer for scientific manuscripts and grant proposals. She has participated in several EU Horizon 2020 projects (Twinning, RISE, COST Actions) and is currently involved in Twinning programs on Extracellular Vesicles in Diagnostics and Therapeutics (EVCA) and Microphysiological Systems (MPS).

5 SELECTED PUBLICATIONS

- Matthiesen R, Lauber C, Sampaio JL, Domingues N, Alves L, Gerl MJ, Almeida MS, Rodrigues G, Araújo Gonçalves P, Ferreira J, Borbinha C, Pedro Marto J, Neves M, Batista F, Viana-Baptista M,

Alves J, Simons K, Vaz WLC, Vieira OV. Shotgun mass spectrometry-based lipid profiling identifies and distinguishes between chronic inflammatory diseases.

EBioMedicine. 2021 Aug;70:103504. doi: 10.1016/j.ebiom.2021.103504. Epub 2021 Jul 24. PMID: 34311325

- Alves LS, Marques ARA, Padrão N, Carvalho FA, Ramalho J, Lopes CS, Soares MIL, Futter CE, Pinho E Melo TMVD, Santos NC, Vieira OV. Cholesteryl hemiazelate causes lysosome dysfunction impacting vascular smooth muscle cell homeostasis. J Cell Sci. 2022 Mar 1;135(5):jcs254631. doi: 10.1242/jcs.254631. Epub 2021 Oct 22. PMID: 34528688
- Domingues N, Gaifem J, Matthiesen R, Saraiva DP, Bento L, Marques ARA, Soares MIL, Sampaio J, Klose C, Surma MA, Almeida MS, Rodrigues G, Gonçalves PA, Ferreira J, E Melo RG, Pedro LM, Simons K, Pinho E Melo TMVD, Cabral MG, Jacinto A, Silvestre R, Vaz W, Vieira OV. Cholesteryl hemiazelate identified in CVD patients causes in vitro and in vivo inflammation. J Lipid Res. 2023 Sep;64(9):100419. doi: 10.1016/j.jlr.2023.100419. Epub 2023 Jul 21.
- Domingues N, Marques ARA, Calado RDA, Ferreira IS, Ramos C, Ramalho J, Soares MIL, Pereira T, Oliveira L, Vicente JR, Wong LH, Simões ICM, Pinho E Melo TMVD, Peden A, Almeida CG, Futter CE, Puertollano R, Vaz WLC, Vieira OV. Oxidized cholesteryl ester induces exocytosis of dysfunctional lysosomes in lipidotic macrophages. Traffic. 2023 Jul;24(7):284-307. doi: 10.1111/tra.12888. Epub 2023 May 2. PMID: 37129279
- Marques ARA*, Ferreira IS, Ribeiro Q, Ferraz MJ, Lopes E, Pinto D, Hall M, Ramalho J, Artola M, Almeida MS, Rodrigues G, Gonçalves PA, Ferreira J, Borbinha C, Marto JP, Viana-Baptista M, Gouveia E Melo R, Pedro LM, Soares MIL, Vaz WLC, Vieira OV*, Aerts JMFG*
Glucosylated cholesterol accumulates in atherosclerotic lesions and impacts macrophage immune response. J Lipid Res. 2025 Jun;66(6):100825. doi: 10.1016/j.jlr.2025.100825.
*Co-corresponding authors

PROJECT TITLE AND SHORT DESCRIPTION

Deciphering Early Atherogenic Mechanisms in 2D and 3D Human Vascular Models

Atherosclerosis is a chronic, non-resolving inflammatory disease of medium and large arteries and the leading pathological basis of cardiovascular disease. One of the earliest steps in atherogenesis is the emergence of lipid-laden “foam” cells, macrophages and vascular smooth muscle cells that accumulate modified low-density lipoproteins (LDL). These lipids build up within lysosomes as poorly degradable material, driving lysosomal dysfunction. The resulting collapse of cellular homeostasis promotes inflammation, senescence, and ultimately cell death. Identifying the molecular events that initiate these changes is essential to enable earlier prevention and to delay disease progression.

Our recent lipidomics studies uncovered a family of cholesterol ester oxidation end-products of cholesteryl hemiesters (ChE) and present in the plasma of cardiovascular disease patients and in human endarterectomy specimens. Importantly, ChE are sufficient to induce lysosomal dysfunction and perturb cell homeostasis, pointing to a direct mechanistic link between patient-derived oxidized lipids and early atherogenic phenotypes.

This project will define how ChE remodel lysosomal membranes and disrupt lysosomal functions using complementary 2D and 3D human systems. Mechanistic work in 2D cultures will be integrated with physiologically relevant 3D blood vessel organoids derived from iPSCs, capturing key cell–cell and cell–matrix interactions that shape vascular responses *in vivo*. Together, these models will enable the construction of a coherent molecular framework connecting disease-relevant oxidized lipids to early cellular dysfunction in the vessel wall.

To achieve these aims, we will combine multi-omics approaches with modern biochemistry and cell biology, including advanced microscopy to resolve lysosomal membrane organisation, trafficking, and functional readouts. The project is expected to pinpoint actionable nodes of vulnerability, such as lysosomal acidification and trafficking pathways, the TFEB programme, and iron-handling mechanisms, and to establish robust 2D/3D platforms suitable for downstream intervention testing.

SCIENTIFIC AREA WHERE THE PROJECT FITS BEST*

Life Sciences (LIF)