MARIE SKŁODOWSKA-CURIE POSTDOCTORAL FELLOWSHIPS 2025

EXPRESSION OF INTEREST FOR HOSTING MARIE CURIE FELLOWS

HOST INSTITUTION

NOVA MEDICAL SCHOOL, FACULDADE DE CIÊNCIAS MÉDICAS, UNIVERSIDADE NOVA DE LISBOA

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

PERSONAL DETAILS

First and last name: Otilia Vieira (OV)
Institutional e-mail address: otilia.vieira@nms.unl.pt
Researcher unique identifier(s): ORCID: 0000-0003-4924-1780; Scopus: 57225361429; Research ID: 5D18-B71A-9E1A

Current position(s)

2020 - Present	*Current Position: Tenured and Habilitated Professor and Principal Investigator at NOVA Medical School (NMS), Universidade NOVA de Lisboa, Portugal, leading the Lysosomes and Disease Group*

Previous position(s)

2014 - 2019	*Principal Investigator (iFCT investigator) of the group Lysosomes and Disease at NMS, Universidade NOVA de Lisboa, Portugal*
2006 - 2014	*Group Leader of the Infection and Pathogens Group, at the Center for Neurosciences and Cell Biology, Universidade de Coimbra, Portugal.*
2002 - 2006	*Postdoc at the Max-Planck Institute for Molecular Cell Biology and Genetics, Dresden, Germany. (Prof. Kai Simons)*
2000 - 2002	*Postdoc at the Hospital for Sick Children, Toronto, Canada. (Prof. Sergio Grinstein)*

OV balances a dual career as a faculty member and researcher. In the last 10 years, Otilia Vieira (OV) published 24 articles, 16 as last author, 1 as single author and 7 as co-author. 1 publication was selected for F1000Prime and subject of a spotlight in the Journal of Cell Biology, 2 publications were selected for the covers of the journals, and another one was the subject of a Comment in EbioMedicine. OV was also a co-author in a Book Chapter. Having worked for the last 10 years on the etiology of atherosclerosis and on the role of ChE, identified by her group, in this pathogenesis, OV is a scientist highly recognized in the field. She has been invited to deliver over 30 talks and seminars at national and international scientific events during this period.

In the last decade, she has successfully supervised 5 postdoctoral fellows, 3 PhD students, and 2 MSc students. Of these, two currently work as research assistants, one is pursuing a PhD, and the remaining have

transitioned to roles in industry and education. Her current lab team consists of one senior scientist, one assistant researcher, three PhD students, and two research fellows.
OV takes an active and committed role in mentoring her team, and under her supervision, four lab members have secured independent salaries through competitive grants from the Portuguese Science Foundation (FCT).
OV´s work has earned several national and international awards: 2024 World's Top 2% Scientists list by Stanford University; 2024 Honorable mention of Bluepharma Innovation Award competition; 2014 FCT investigator (competitive salary for 6 years); 2004 Max-Planck Society Post-doctoral Fellowship; Max-Planck-Gesellschaft, Germany; 2002 Marie-Curie Post-doctoral Fellowship; 2002 EMBO Post-doctoral Fellowship.
She frequently serves as an examiner for MSc and PhD theses both in Portugal and internationally (notably in Italy and Norway), and she is a reviewer for international funding bodies such as the United States–Israel Binational Science Foundation (BSF) and the Austrian Science Fund.
OV is an Editor for Scientific Reports (Nature Publishing Group) and regularly serves as a peer reviewer for leading journals, including Nature Reviews Cardiology and Journal of Cell Biology.
Currently, she is a team member on two Twinning projects funded by the European Commission one in Microphysiological Systems (MPS_NOVA) and the other in Extracellular Vesicles (EVCA) and participates in a COST Action focused on Cellular Senescence.

5 SELECTED PUBLICATIONS

- N. Domingues, ..., **O. V. Vieira[1]**. Inflammatory Profile of human immune cells and zebrafish larvae exposed to a New Lipid Identified in Cardiovascular Disease Patients. **J Lipid Res**. (2023) 21:100419. doi: 10.1016/j.jlr.2023.100419. [1]Corresponding author. This article was selected for the cover of the journal and was highlighted in the ASMB today.
- Domingues N, ……. **O. V. Vieira[1]**. Oxidized cholesteryl ester induces exocytosis of dysfunctional lysosomes in lipidotic macrophages. **Traffic**. 2023 Jul; 24(7):284-307. doi: 10.1111/tra.12888. [1]Corresponding author. This article was selected for the cover of the journal.
- L.S. Alves, ……., **O.V.Vieira[1]**. Cholesteryl hemiazelate cause lysosome dysfunction impacting vascular smooth muscle cells homeostasis. **Journal of Cell Science** (2022) 135, jcs254631. doi:10.1242/jcs.254631. [1]Corresponding author. Published in the SPECIAL ISSUE: CELL BIOLOGY OF LIPIDS
- Matthiesen R, ……..,**O.V.Vieira[1]**. (2021). Shotgun mass spectrometry-based lipid profiling identifies and distinguishes between chronic inflammatory diseases. **EBioMedicine**. Aug;70:103504. DOI: 10.1016/j.ebiom.2021.103504. 1Corresponding author. This article was the subject of a Comment in EbioMedicine.
- M. J. Gerl, …… **O.V. Vieira[1]**. (2018). Cholesterol is Inefficiently Converted to Cholesteryl Esters in the Blood of Cardiovascular Disease Patients. **Scientific Reports** 8(1):14764. doi:10.1038/s41598-018-33116-4. [1]Corresponding author.

PROJECT TITLE AND SHORT DESCRIPTION

TITLE: UNRAVELING THE ROLE OF CHOLESTERYL HEMIESTERS IN LYSOSOMAL DYSFUNCTION AND ATHEROSCLEROSIS PROGRESSION

Atherosclerosis is a progressive, chronic, and often silent disease that underlies the majority of cardiovascular diseases (CVD), which remain the leading cause of death and disability worldwide. The molecular and cellular mechanisms driving the initiation and progression of atherosclerotic lesions are highly complex and remain incompletely understood.
Recently, we identified a novel family of oxidized lipids—cholesteryl hemiesters (ChE)— by shotgun lipidomics in the plasma of CVD patients and in human endarterectomy specimens. These ChE molecules are generated during low-density lipoprotein (LDL) oxidation within the arterial intima and, due to their amphiphilic nature, eventually become detectable in plasma.
All ChE species tested to date have been shown to induce lysosomal dysfunction—a critical event in the pathogenesis of atherosclerosis with significant consequences for inter-organelle communication and cellular homeostasis.

The current objective is to elucidate the molecular mechanisms by which ChE induces lysosomal dysfunction and to investigate how this disruption affects communication between intracellular membrane compartments, contributing to cellular senescence or death.
To achieve this, the study will employ a combination of primary cells, established cell lines, and human blood vessel organoids (hBVOs), alongside a suite of cell biology and biochemical techniques, advanced microscopy, and multi-omics approaches.
Given that lysosomal dysfunction is increasingly recognized as a common pathogenic mechanism in normal aging and numerous age-related diseases, this project has the potential to significantly advance our understanding of aging biology and contribute broadly to improving human health.

SCIENTIFIC AREA WHERE THE PROJECT FITS BEST*

LIFE SCIENCES (LIF)