

Why do women's brains age differently?

Estrogen does more than regulate reproduction — it helps brain cells handle stress. When levels decline after menopause, this ability is reduced, and these hormonal changes are believed to contribute to the increased risk of Alzheimer's disease in women. MPI-IE researcher María José Pérez Jiménez has received the Klaus Tschira Boost Fund to investigate why — and whether these cellular responses can be restored.

Women are significantly more likely than men to develop Alzheimer's disease. One major reason is still poorly understood: how the loss of estrogen after menopause shapes the brain's vulnerability to disease. María José Pérez Jiménez, a postdoctoral researcher at the Max Planck Institute of Immunobiology and Epigenetics (MPI-IE) in Freiburg, has received a Klaus Tschira Boost Fund fellowship to understand how these hormones keep brain cells healthy and resilient.

Her project focuses on a mechanism that has been overlooked for too long: how estrogen regulates the response of brain cells to mitochondrial stress — the strain that occurs when the cell's energy-producing machinery is damaged or overwhelmed. Crucially, this stress response also remodels the epigenetic state of the cell, changing which genes are switched on or off in neurons and glial cells.

"Rather than treating mitochondrial dysfunction as a purely metabolic problem, I want to study how it feeds into chromatin regulation and gene expression programs — and how adaptive cellular states can be maintained or restored," says Pérez Jiménez. Understanding this connection between hormonal signals, mitochondrial function, and gene regulation could help explain why women's brains become more vulnerable to disease after menopause.

Estrogen shapes female brain health across the lifespan

When estrogen levels drop after menopause, this protective state can collapse. Pérez Jiménez wants to understand what that collapse looks like at the molecular level, and whether it could one day be therapeutically restored — potentially opening new ways to protect women's brain health as they age.

To study these mechanisms, Pérez recently joined the lab of Sukanya Guhathakurta in the Department of Chromatin Regulation at MPI-IE. She uses lab-grown human brain cells, including 3D brain organoids, to observe how hormonal changes reshape cellular identity and gene activity. Her approach combines experimental work with single-cell genomics and multi-omics analysis that examine how genes, proteins, and metabolites all interact at once.

About the researcher

María José Pérez Jiménez studied biochemistry in Chile and completed her PhD in Biomedical Sciences in 2018. Supported by Becas Chile and DAAD postdoctoral fellowships, she then held research positions at the German Center for Neurodegenerative Diseases (DZNE) and the Hertie Institute for Clinical Brain Research in Tübingen, and later at the Imagine Institute of Genetic Diseases in Paris — building expertise in mitochondrial dysfunction, neuroinflammation, and human stem cell models of brain disease. An EMBO Long-Term Fellowship further supported her work during this period. She was also selected for the Freiburg Rising Stars Academy 2025/26 cohort — a competitive programme by the University of Freiburg that connects outstanding international early-career researchers with Freiburg's research community, offering mentoring, networking, and a pathway to long-term collaboration in the region. Pérez Jiménez joined MPI-IE in March 2026, where her work now connects this background in neurodegeneration to the emerging field of epigenetic regulation.

About the Klaus Tschira Boost Fund

The Klaus Tschira Boost Fund, awarded by GSO – Guidance, Skills & Opportunities for Researchers in partnership with the Klaus Tschira Stiftung, supports postdoctoral researchers in the natural sciences with up to €120,000 for bold, exploratory projects. Fellows also receive career development training and access to a professional network of alumni across Germany.

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