PhD position at the Biochemistry of Oxidative Stress Group, IRBLleida, Universitat de Lleida

The Biochemistry of Oxidative Stress group (BEOX) focuses on Friedreich Ataxia (FA), a rare, cardio-neurodegenerative disease caused by deficient levels of frataxin, a mitochondrial protein. The group is looking for a researcher interested in carrying out a PhD research project, focused on understanding the molecular mechanisms of FA, and develop new therapeutic approaches for this rare disease.

DURATION: A 4-year contract is offered.

INCORPORATION: First semester 2025

CENTRE: IRBLleida, Universitat de Lleida, Lleida.

ELIGIBILITY REQUIREMENTS: Bachelor and Master's degrees in Biomedicine, Biochemistry, Biotechnology, Biology, or related sciences. Previous experience in biochemistry or cell biology methods will be positively valued.

FUNDING: Ayuda para la formación de personal investigador predoctoral, Convocatoria PID2023 (Proyectos de Generación de Conocimiento) de la Agencia Estatal de Investigación.

PROJECT TITLE: Alteraciones moleculares en el modelo murino FXNI151F de Ataxia de Friedreich y análisis de nuevas estrategias terapéuticas.

PRINCIPAL RESEARCHERS: Jordi Tamarit Sumalla and Elisa Cabiscol Català

CONTACT: If you are interested, send your CV to jordi.tamarit@udl.cat or elisa.cabiscol@udl.cat

MORE INFORMATION ABOUT THE RESEARCH GROUP:

https://www.irblleida.org/en/research/12/biochemistry-of-oxidative-stress

PROJECT SUMMARY: Friedreich Ataxia (FA) is an inherited recessive disease caused by mutations in the frataxin gene. Patients present progressive neurodegeneration and hypertrophic cardiomyopathy, being cardiac dysfunction the leading cause of death. The most common mutation is a GAA triplet expansion in the first intron of the gene which causes a decline in frataxin expression. Around 4% of patients are compound heterozygous for a GAA expansion and a frataxin point mutation or deletion. There is currently no effective cure for FA. In this project we will use a murine model of FA (FXNI151F model) to deeply tackle the mechanisms of neurodegeneration and cardiac dysfunction in this disease, on the contribution of iron to the phenotypes observed, and

to test new therapies. In this regard, we will characterize the role of glial cells in neuronal dysfunction, as several evidence suggest that these cells play an important role in FA. We will also characterize the cardiac phenotype of the FXNI151F mice, analyzing cardiac function and hypertrophic signaling pathways and markers. Regarding iron, although it is well established that dysregulation of this metal plays a central role in FA, it is not clear which is the contribution of this deregulation to neurodegeneration or to the cardiac phenotype, nor the capacity of frataxin-deficient animals to regulate systemic iron absorption or distribution. We will also test several drugs in the FXN151F mice and in patient-derived fibroblasts. These compounds have previously shown benefits in cell cultures models of the disease. We expect that the results obtained will improve our understanding about the tissue-specific consequences of frataxin deficiency, define biomarkers of the disease progression, and contribute to develop new therapeutic approaches for FA.