Master's thesis at the Department of Neurology

Aim: Understand the effect of neuronal sphingolipid accumulation on mitochondrial mobility and function as contributor to peripheral denervation and pain.

Background: Fabry disease (FD) is an X-linked multiorgan disorder caused by mutations in the alpha-galactosidase A (*GLA*) gene. Due to enzyme deficiency, the sphingolipid globotriaosylceramide (Gb3) accumulates in diverse cell types, including sensory neurons, and leads to pain. We have previously seen that Gb3 accumulation affects mitochondrial morphology. In this project we will investigate how Gb3 accumulation affects mitochondrial trafficking and function in a human sensory neuron model. As a translational approach, we aim to treat mitochondria with molecules that enhance their function in order to restore normal sensory neuron processing.

Tasks:

1. To characterize isogenic control cell lines for data comparison and phenotype rescue
2. To determine the effect of Gb3 accumulation on mitochondrial mobility
3. To investigate the effect of Gb3 accumulation on mitochondrial function
4. To evaluate the potential of mitochondrial or FD-specific treatment in restoring mitochondrial mobility and function and neuronal outgrowth

Techniques: iPSC culture, immunofluorescence, confocal microscopy, real time PCR (RT-PCR), Seahorse assay

Requirements: We are looking for a student (m/f/d) of Life Sciences or related Faculty, who is motivated to engage himself/herself in this exciting project and to become part of our enthusiastic research team!

Start and duration: Start is possible from March on for six months.

Team of supervisors: Prof. Dr. Nurcan Üçeyler, Dr. Christoph Erbacher (Erbacher_C@ukw.de), Vijay Medala (Medala_V@ukw.de).

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