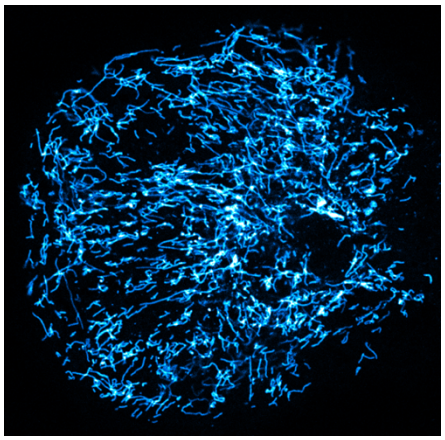
Fully funded PhD position available



http://boecker-lab.uni-goettingen.de

Interested candidates are invited to send their application including a CV to:

alexander.boecker@med.uni-goettingen.de

We are looking for a PhD student to join our team and support our work investigating the role of LRRK2 in the pathogenesis of Parkinson's disease. The project is funded by the Else Kröner-Fresenius Foundation and scheduled to start on April 1st, 2024. We will investigate how hyperphosphorylation of RAB GTPases by pathogenic hyperactivation of LRRK2 affects the turnover of autophagic cargo, and how this may contribute to the neurodegeneration underlying Parkinson's disease.

LRRK2 mutations are the most common genetic cause of Parkinson's disease and lead to pathogenic hyperactivation of LRRK2 kinase, resulting in RAB hyperphosphorylation. However, it is currently unknown how increased levels of phospho-RABs cause the neurodegeneration underlying Parkinson's disease. This project will use advanced light microscopy techniques to investigate in primary neurons and astrocytes how LRRK2-mediated RAB hyperphosphorylation affects organelle degradation through autophagy, a pathway essential for cellular health and with strong links to Parkinson's disease.

Selected previous work:

Boecker, C.A., Goldsmith, J., Dou, D., Cajka, G.G., and Holzbaur, E.L.F. (2021). Increased LRRK2 kinase activity alters neuronal autophagy by disrupting the axonal transport of autophagosomes. *Current Biology*, 1–15. DOI: 10.1016/j.cub.2021.02.061.

Dou, D., Smith, E.M., Evans, C.S., Boecker, C.A.*, and Holzbaur, E.L.F.* (2023). Regulatory imbalance between LRRK2 kinase, PPM1H phosphatase, and ARF6 GTPase disrupts the axonal transport of autophagosomes. *Cell Reports 42*. DOI: 10.1016/j.celrep.2023.112448. *Contributed equally