Postdoc Call 2020

Do you want to work in the lab of world leading neuroscientists then read this document!

Introduction

The Brain Prize, the world's largest brain research prize, is Danish and is awarded annually by the Lundbeck Foundation to one or more neuroscientists who have had a ground-breaking impact in any area of brain research.

The Brain Prize was first awarded in 2011 and has so far honoured 34 scientists from 9 different countries. The Prize is a celebration of outstanding achievements in neuroscience and is at the forefront of the Lundbeck Foundation's ambitions to make Denmark a world leading neuroscience nation.

To strengthen ties between winners of The Brain Prize and young Danish neuroscientists, the Lundbeck Foundation has solicited project proposals from previous winners who are keen to recruit talented Danish post docs via our Lundbeck Foundation Postdoc program, 2020. If you would like to work on one of these projects and gain experience in the lab of a world leading neuroscientist, please use included contact information to get in touch with the relevant prize winner.

Other recognized neuroscientists laboratories have also expressed interest in recruiting talented Danish post docs.

The call closes on November 10th 2020 and you can read the full call text for the Lundbeck Foundation Postdocs 2020 including contact information at the Lundbeck Foundation at https://www.lundbeckfonden.com/en/grants/what-you-can-apply-for/.

The foundation will update this document over the years to come as other Brain Prize Winners express interest in recruiting a Danish post doc and/or establishing a research collaboration with a Danish neuroscience laboratory.

Lundbeck Foundation
September 2020

Laboratories of The Brain Prize Winners

Titel and name	Institution	Country
Prof. Dr. Christian Haass	Ludwig-Maximilians University	Germany
Prof. Gero Miesenböck	University of Oxford	England
Prof. Giacomo Rizzolatti	University of Parma	Italy
Dr. Hugues Chabriat	University Hospital Lariboisiere-Paris	France
Prof. Karen Steel	Kings College London	England
Prof. Ray Dolan	University College London	England
Prof. Richard Morris	University of Edinburgh	Scotland
OTHER RECOGNIZED INTERNATIONAL NEUROSCIENCE LAB.		
Prof. Volker Haucke	Leibniz-Forschungsinstitut für Molekulare Pharmakologie	Germany

Christian Haass **Ludwig Maximillian's University**

What we are looking for: We are looking for a postdoc with extended experience in lipidomics and lipid metabolism. We are specifically interested in identifying lipid related dysfunctions of neurons and microglia in mouse models and human iPSC derived cellular models of neurodegenerative diseases. Experience in big data analysis would be very helpful. The candidate will work in a team of interdisciplinary scientists studying neurodegeneration from a basic mechanistic and translational point of view.

The Haass lab: My lab works since 30 years on the molecular mechanisms of neurodegeneration with a specific focus on Alzheimer's disease and Frontotemporal Lobar Degeneration. The lab started out working on the cell biology of the amyloid generating secretases. More recently, we moved the focus to the role of microglial function and dysfunction during neurodegenerative diseases. We not only want to gain detailed insights how microglia defend or promote neurodegeneration, but we are also very much interested in finding microglial signaling pathways, which can be therapeutically modulated. The lab is part of the German Center for Neurodegenerative Diseases (DZNE) in Munich. The DZNE provides an interdisciplinary environment with research groups working on all aspects of neurodegeneration, from basic mechanisms to translational medicine. Our microglia project is carried out in close collaboration with Denali Therapeutics in San Francisco. Christian Haass is the speaker of the DZNE-Munich as well as the Munich Cluster for Systems Neurology (SyNergy).

https://www.biochemie.abi.med.uni-muenchen.de/about/staff/professors/haass/index.html
https://www.dzne.de/
https://www.synergy-munich.de/index.html

Further reading:

Schlepckow et al. (2020) Enhancing protective microglial activities with a dual function TREM2 antibody to the stalk region. EMBO Mol Med, 12(4): e11227.

Ewers et al. (2019) Increased soluble TREM2 is associated with reduced cognitive and clinical decline in Alzheimer's disease. Sci Transl Med., 1(507). pii: eaav6221. doi: 10.1126/scitranslmed.aav6221.

Parhizkar et al. (2019) Loss of TREM2 function increases amyloid seeding but reduces plaque- associated ApoE. *Nature Neuroscience* 22(2): 191-204.

Suárez-Calvet et al. (2018) CSF progranulin increases in the course of Alzheimer's disease and is associated with sTREM2, neurodegeneration and cognitive decline. *EMBO Molecular Medicine* 2018 Dec;10(12). pii: e9712

Kleinberger et al. (2017) The FTD-like syndrome causing TREM2 T66M mutation impairs microglia function, brain perfusion and glucose metabolism *The EMBO Journal* 36(13): 1837- 1853.

Suárez-Calvet et al. (2016) Early changes of CSF sTREM2 in Dominantly Inherited Alzheimer's Disease follow markers of Amyloid Deposition and Neuronal Injury. *Science Translational Medicine* 8(369): 369ra178.

Willem et al. (2015) eta-Secretase processing of APP inhibits neuronal activity in the hippocampus. *Nature* 526 :443-7.

Contact information:
Prof. Dr. Dr. h.c. Christian Haass
Biomedizinisches Centrum (BMC), Biochemie, Ludwig-Maximilians-Universität München & Deutsches Zentrum für Neurodegenerative Erkrankungen (DZNE) e.V.
Feodor-Lynen Strasse 17; 81377 München; Germany
Phone:
(+49-89) 4400-46549 (C. Haass)
(+49-89) 4400-46550 (Dr. N. Exner; Assistant)

Gero Miesenböck **University of Oxford**

The Project. Sleep is vital and universal, but its biological function remains unknown. This project will seek to understand why we need to sleep by studying how the brain responds to sleep loss. Our previous work in Drosophila showed that rising sleep pressure activates sleep-inducing neurons via a potassium channel β-subunit (Kvβ) that senses changes in cellular redox chemistry. Sleep loss elevates mitochondrial reactive oxygen species, which register this rise by converting Kvβ to the $NADP^+$-bound form. The oxidation of the cofactor boosts the frequency of action potentials and thereby promotes sleep. Future research will seek to demonstrate that redox-sensitive Kvβ subunits regulate sleep also in mammals and furnish a deeper understanding of the underlying molecular mechanism.

The Lab. Although the Miesenböck lab (now at the University of Oxford, formerly at Yale) has been responsible for important methodological advances (most notably the invention of optogenetics), methods development and biological discovery have always gone hand in hand. The group currently studies two neural integrators: those that accumulate evidence during decision-making, and those that accumulate sleep pressure during waking.

Further Information.
http://www.cncb.ox.ac.uk
http://www.cncb.ox.ac.uk/the-science/research-groups/miesenboeck-group/

Gero Miesenböck FRS
Waynflete Professor of Physiology
Director, Centre for Neural Circuits and Behaviour
University of Oxford
Tinsley Building
Mansfield Road
Oxford, OX1 3SR

gero.miesenboeck@cncb.ox.ac.uk

Giacomo Rizzalotti **University of Parma**

Description of the Lab

Grounded on the neurophysiology of the mirror mechanism in humans, the aim of the lab is to collect evidence of the value of action observation not only in supporting the recovery of impaired motor abilities, but also in preserving motor skills and in sustaining the acquisition and perfectioning of new motor abilities.

This topic is timely for two main reasons: First, beyond clinical practices, action observation may act as bulk for a wide number of trainings potentially of great impact on the safety and quality of life of individuals across the whole lifespan. Second, recent technological advances (e.g. 3D environments, virtual reality, etc…) have the potentiality to allow an individualization of procedures, with a consequent maximization of the efficiency of trainings and applications based on action observation.

Our lab is fully equipped for this line of research. We currently use kinematic sensors (stereophotogrammetry, inertial sensors, wearable gloves) to record upper limb and whole body movements, virtual reality visors to reproduce movements in 3D scenarios, TMS and high-density EEG to explore the neural correlates of AOT treatment and efficacy. In addition, we have access to a 3T MRI scanner available for experimental studies.

Candidate profile

Different profiles could fit with this line of research, and research activity can be tailored to individual candidates skills. As an example, clinical researchers could be interested in designing joint clinical trials in their outsourcing centers, researchers with technical skills could join our activities on virtual reality design and editing, post-docs with expertise in human neurophysiology could take part in designing and carrying on experiments aimed at revealing the neural substrates of motor training via observation, and at validating biomarkers of their efficacy. For this reason, research activities will have to be agreed via preliminary contacts between the candidate and our group.

Pr Hugues CHABRIAT (The BRAIN PRIZE 2019)
Department of Neurology, CERVCO (www.cervco.fr), APHP and University of Paris
INSERM U1141 ; Hopital Lariboisiére, 2 rue A Paré, 75010 Paris, France

Request : a Post-DOC candidate for Functional MRI studies and imaging data processing/calculation

Duration : minimum 24 months, maximum 36 months

Location : at the interface between the neuroscience clinical research group at APHP, Lariboisiére Hospital in Paris and an imaging research team located at CEA (Commissariat à l'énergie Atomique) in Saclay (southern suburban area of Paris), in France.

Summary of the project : CADASIL is a cerebral small vessel disease caused by mutations of the NOTCH3 gene on chromosome 19 which is responsible for migraine with aura, stroke, disability and dementia along aging. Accumulation of the extracellular domains of the NOTCH3 receptor leads to aggregates of different vascular matrix proteins at the surface of smooth muscle cells and pericytes in CADASIL patients. This accumulation resuls in severe functional alterations of cerebral microvessels. In animal models, neurovascular coupling was found particularly altered at the early stage of the disease. In humans, some data suggest that the vascular response to visual or motor stimulations is also altered in-vivo. We now aim to develop innovative tools for capturing, measuring and following alterations of neurovascular coupling in CADASIL patients in-vivo. For this purpose, fMRI studies will be obtained in CADASIL patients and healthy individuals in pilot and validating studies. **The final objective is to develop and validate a robust imaging biomarker for longitudinal studies in CADASIL that can be also used for therapeutic development in future.**

Position description : at the interface betwen different research teams (clinicians, imaging researchers, data scientists) we are seeking for a post-doctoral fellow to carry out a research project in neuroimaging within the framework of the "Trt-cSVD" Hospital-University Health Research project, a large program of studies including academic and industrial partners that aims to develop innovative biomarkers for investigating cerebral small vessel diseases at preclinical and clinical level. **The mission will be to contribute to the development of new sensitive and robust biomarkers derived from functional magnetic resonance (MR) images by working in interaction with various participants of the project** (MR physicists, imaging processing specialists, data scientists, clinicians biologists investigating neurovascular coupling) at NeuroSpin at CEA de Saclay, CENIR (location of fMRI studies) and Lariboisiére Hospital. **The post-Doc will intervene in the processing of images, evaluating models and statistical analysis of data and possibly in their acquisition**. She/he will also be a driving force in the progress of fMRI related tasks (storage of data flows, pre-processing, progress reports). She/he will prepare different scientific articles and present her/his main results at international congresses.

Required profile : The candidate should have **a doctorate with experience in neuroimaging studies and imaging data processing and with a good record of publications**. She/he should **master the tools to process MRI images and conduct statistical analyzes.**The candidate should be enough autonomous and efficient in computer programming and image processing. She/he should be rigorous, well organized, curious and ready to work at the interface between MRI methodology and clinical applications.

Danish research groups/institutions can be involved in such collaboration, particularly if the post-DOC is already related to research groups involved in functional neuroimaging studies in animal models or humans or in imaging studies of neurovascular coupling.

Karen Steel **King´s College London**

Lundbeck Foundation proposal

We use genetics as a tool to understand the molecular pathways underlying both childhood and progressive (age-related) hearing loss. My group works with both mouse and human data, using the mouse for understanding the different pathophysiological mechanisms underlying hearing loss and human data to establish the most common types of deafness in the population. Several of the pathways we have identified in the mouse are good targets for drug development. We have several projects available for a suitable postdoc to undertake, including analysing auditory function in mice with human mutations knocked-in using electrophysiological, expression and ultrastructural techniques, investigating the mechanisms involved in age-related hearing loss using single-cell RNAseq, exploring the role of vascular defects in hearing loss, and investigating potential drug treatments.
My laboratory at King's College London has all the facilities required to carry out the proposed research, including three bespoke auditory electrophysiology rigs, mouse holding and breeding facilities within the same building allowing repeated recording of auditory function with age, and dedicated histopathology and molecular biology laboratories We also have access on the campus to state-of-the-art confocal microscopes via the Nikon Centre, electron microscopy facilities including serial block face scanning electron microscopy and tomography, flow cytometry and single-cell RNAseq facilities, and a gene editing core facility that generates our new mouse knockin mutations. We have weekly lab meetings to discuss data and the team usually includes around 8 to 10 people including senior postdocs with expertise in electrophysiology and genome informatics, and a highly collaborative attitude.

I am currently collaborating with Dr Hanne Poulsen in Aarhus University on a Lundbeck-funded project "Delineating the causes of loss of sight and hearing in the CAPOS syndrome using a mouse model". The postdoc working on this project in Aarhus is Dr Tommi Anttonen, and he has expressed an interest in working in my team in London for an extended project. I am also interested in hosting other postdocs who might be interested in working on hearing loss in my team before returning to Denmark. Denmark has a vibrant research effort in auditory research but mostly focussed on hearing aids and cochlear implants, so enhancing the national expertise in the molecular underpinning of hearing loss would build upon this strong foundation and extend it.

Contact Professor Karen P Steel at karen.steel@kcl.ac.uk.
Wolfson Centre for Age-Related Diseases, Guy's Campus
King's College London, London SE1 1UL, UK. https://www.kcl.ac.uk/research

Ray Dolan **University College London**

Project Title: Replay in the human brain in relation to decision making

Effective learning requires incorporating new experience into our existing knowledge of the world. In reinforcement learning (RL) theory non-local value learning can be achieved by "model-based" methods that leverage a learned map or model of the environment to simulate, or simply retrieve, potential trajectories. A potential neural substrate for this process is the phenomenon of hippocampal "replay", where cells in the rodent hippocampus that encode distinct locations in space fire sequentially during rest in a time-compressed manner, recapitulating past or future trajectories. Utilizing novel methods we have developed to measure fast neural sequences noninvasively in my lab, such replay has now been shown in humans subjects. The proposed research aims to examine how replay underpins different forms of learning and inference. The project can include a focus on a key methodological challenge to develop better measures of replay, including how we can improve decoding of states, as well as questions related to how replay relates to the computations that guide decision making.

Location: Max Planck Centre/Dolan Lab

The Max Planck UCL Centre for Computational Psychiatry and Ageing Research is dedicated to studying the causes of psychiatric disorders as well as the causes of individual differences in cognitive development, with an emphasis on adulthood and old age. The Centre was founded in April 2014 and is the result of an existing collaboration between the **Max Planck Society** and **University College London** that began in 2011. The Centre is headed by **Ray Dolan** (University College London) and **Ulman Lindenberger** (Max Planck Institute for Human Development) and is located in London and Berlin. The London site is at Russell Square, in close vicinity to the Wellcome Centre for Human Neuroimaging. The Berlin site is housed at the Max Planck Institute for Human Development.

The Dolan Lab studies reward and human decision making, including its relationship to psychopathology, using a range of methodologies. These include behavioural analysis, computational modelling, psychopharmacological manipulations as well as brain imaging, involving fMRI and MEG. A recent focus of the group addresses how we build cognitive maps and how these support inference during model based decision making. Here the lab has developed sophisticated decoding tools that enable indexing of fast replay of neural representations during learning, episodic memory and decision making. This work has recently been extended to examine the expression of neural replay in patients with schizophrenia.

Dolan Contact:
Email – r.dolan@ucl.ac.uk
Webpages - https://www.mps-ucl-centre.mpg.de/about-the-centre
https://www.fil.ion.ucl.ac.uk/team/cognition-and-computational-psychiatry/
https://iris.ucl.ac.uk/iris/browse/profile?upi=RJDOL46

Richard Morris **University of Edinburgh**

Postdoctoral opportunities in the Morris Lab - The University of Edinburgh

r.g.m.morris@ed.ac.uk

Overview

The "Laboratory for Cognitive Neuroscience" (LCN) at the University of Edinburgh was created by Richard Morris in 1987 and has been running ever since. It is now a constituent part of EDINBURGH NEUROSCIENCE (https://www.edinburghneuroscience.ed.ac.uk) and part of a University Research Centre called the Centre for Discovery Brain Sciences (https://www.ed.ac.uk/discovery-brain-sciences). The University has a thriving neuroscience community ranging from fundamental through to clinical neuroscience.
The primary focus of LCN is fundamental research on the neurobiological mechanisms of learning and memory, with a current focus on "episodic-like" memory and the transformation of representations into "semantic-like" memory via schemas. A secondary facet, since 1995, is translational work. This was initially in the field of animal models of Alzheimer's Disease and, particularly, early detection of cognitive deficits. This project was concluded in 2017 and, since then, a new translational side of the lab has been focused on "social neuroscience models" of autism spectrum disorders, especially Fragile-X.
Innovations include the development of the watermaze to study spatial learning (Morris et al, *Nature,* 1982), the role of NMDA receptors in memory encoding (Morris et al, *Nature* 1986), the synaptic-tagging-and-capture theory of protein synthesis-dependent long-term potentiation (Frey and Morris, *Nature* 1997), and the role of schemas in systems consolidation (Tse et al, *Science,* 2007). The lab has introduced optogenetics techniques to the study of memory consolidation (Takeuchi et al, *Nature* 2016) and, now, endoscopic Ca2+ imaging in freely-moving rats.
From the perspective of the Lundbeck Post-doc Call, there could be one project on **episodic-like/semantic-like memory** and another project on **social neuroscience**. What these two projects have in common is that both use the technique of endoscopic Ca2+ imaging in freely-moving animals.

Expressing interest

Our understanding in Edinburgh is that a postdoc application has to be made by one or more Danish postdocs by 10 November 2020. Richard Morris (email address above) is very happy to discuss either project in detail and offer suitable written assistance with preparing an application. I think the scheme allowing a Danish postdoc to spend most of the time in Edinburgh but then return to Denmark offers the exciting prospect of bringing whatever ideas and techniques that could be acquired with us in Edinburgh back to Denmark. Further information about Project 1 (Social neuroscience) is provided below by way of illustration.

Project 1: Ca2+ imaging in medial prefrontal cortex during social interactions between freely-moving Fragile-X rats

P1.1 Aim

The aim of this project would be to (1) join a small team exploring the neurobiology of social interactions between normal animals, and between normal and Fragile-X animals which display altered responses to social novelty and social dominance induced by this monogenetic disorder. The study would use awake behaving animals and endoscopic Ca^{2+} imaging of transients in the prelimbic region of the medial prefrontal cortex (mPFC). The second facet of the project would be (2) to test treatment options targeting alterations in synaptic plasticity or proteolysis that might alter both behaviour and correlated Ca^{2+} signals.

P1.2 Funding

This is an ongoing project since 2017. I have some funding from the Simons Institute for the Developing Brain (Edinburgh) and matched funding from a Wellcome Trust Investigator Grant for use of the same Ca^{2+} imaging technique to address different questions (associated with episodic- and semantic-like memory). Neither of these sources of funding would cover the salary cost of a visiting postdoc nor the additional costs of the consumables and associated animal costs, which would have to be secured via the Lundbeck Call. There would, however, be no need for equipment purchase

P1.3. Overarching Science

Key idea and findings

Social withdrawal is a key phenotypic feature of Fragile-X in humans. In a pers.comm. exchange with Dr Andy Stanfield (Psychiatry, Edinburgh), he wrote: *"More broadly, children with autism are more likely to both bully their siblings and be bullied by their siblings (Toseeb et al, J. Autism Devel Disorders 2018) and they tend to be the more social impaired children who are both bully and victim."* Now, using a tube-test model, we observe that Fragile-X rats display disturbances of social dominance, and of the stability of social status that are experientially-induced (Saxena et al, Proc. Roy. Soc. B., 2018). While Fragile-X animals are normally submissive, some Fragile-X animals becoming habitually winners in social dominance contests despite failing to provide appropriate social cues. We have recently replicated key findings in animals from which Ca^{2+} imaging was being done simultaneously. Moreover, we have made the discovery in a 3-chamber sociability test that the social interactions of FXS rats reflect their not providing appropriate social signals to others. We are exploring Ca^{2+} transients in mPFC in both tube-test and sociability protocols, and have striking pilot data using state-of-the-art MCA 'manifold' neural population analyses.

In vivo Ca2+ imaging in freely moving animals

The SIDB and Wellcome Trust funded project have proceeded alongside each other to develop expertise in all facets of in vivo awake animal Ca^{2+} imaging. With the assistance of Dr Diane Wermo (an INSCOPIX employee based in Paris), we have learned about use of GCamp6f, virus dose-response control studies, GRIN lens implantation, base-plate surgery and endoscopic recording using the Inscopix IDPS software and CNMFE cell-identification algorithms. The social neuroscience project has, to date, involved behavioural studies of n=16 rats with endoscopic recording in n=11 animals of which n=10 were successful. Best cases establish circa 150 "cells" per animal, with a range from 50-150. Only one animal had to be 'discontinued' as it had only 5 detectable cells.

Treatment opportunities

The findings about social withdrawal are sufficiently exciting to warrant a move to exploring putative treatments in both behavioural tests in association with Ca2+ imaging. We propose to explore the impact of lovastatin and botezomib. Lovastatin is commonly used in the treatment of hypercholesterolemia. It also inhibits the translocation of Ras protein resulting in a dimunition of protein synthesis (Schafer et al., *Science,* 1989; Cerezo-Guisado et al., *Biochem. J.*, 2007). It is known that, in FXS, there is an increase in the activity of ERK/MAPK pathway which could produce excessive protein synthesis (Osterweil et al., *J. Neurosci.,* 2010; Vithayathil et al., *Prog. Brain. Res.,* 2018; Bagni et al., *Neuron,* 2019). Accordingly, lovastatin normalizes this, and so has an important effect on different parameters. Work in the Fmr1-/y mouse model shows that lovastatin corrects excessive protein synthesis, normalized exaggerated mGluR-LTD, reduces epileptiform activity in hippocampus and visual cortex, and prevents audiogenic seizures (Osterweil et al., *Neuron,* 2013). Additionally, early administration of lovastatin in Fmr1-/y rats prevents the emergence of learning and memory deficits, and associated plasticity deficits in the prefrontal cortex (Asiminas et al., *Sci. Transl. Med.,* 2019). Given the efficacy of lovastatin for correcting circuit phenotypes in multiple brain regions, in both mice and rats, this drug stands a good chance of ameliorating abnormal social behaviour phenotypes in the Fmr1-/y rat (Esch et al., *Neurobiol Dis.,* 2015). Botezomib, on the other hand, is a selective inhibitor of the proteasome complex that is responsible for protein degradation. Work in the Osterweil group shows there is a significant elevation of proteasome activity in the Fmr1-/y mouse hippocampus, that is also seen in the Fmr1-/y rat. Bortezomib, which is a highly selective inhibitor of the proteasome catalytic core, can normalize proteasome activity, and thereby restore normal levels of protein synthesis. Importantly, acute injection of bortezomib blocks the occurrence of audiogenic seizures in the Fmr1-/y mouse, indicating it is a pharmacological strategy for correcting FX phenotypes (Fig. 5).

P1.4 Summary

This proposal is relevant to autism research as a window into the neural mechanisms and treatment of social withdrawal. It is an exciting project with clear translational relevance which we hope could be of interest to a suitably qualified Danish postdoc. There are opportunities for publication as part of a team and, in association with the team work, a smaller first-authored paper by the visiting postdoc - subject to the usual qualification that research is an uncertain process with no guarantees. The laboratory encourages creativity by the postdocs, but within a clear framework or "backbone" of ongoing projects.

Volker Hauke **Leibniz-Forschungsinstitut für Molekulare Pharmakologie**

Description of proposed research

Our ability to move, to process sensory information or to form, store and retrieve memories crucially depends on the function of neuronal synapses. However, we know surprisingly little about the pathways that direct the formation, transport, and assembly of the complex molecular machines that make up a functional presynaptic compartment. The proposed research aims to identify the origin and composition of SV and AZ precursors, dissect the mechanisms of their axonal transport and integration into developing synapses and unravel the pathway that controls axonal transport and presynaptic assembly of newly made SV and AZ proteins to set synaptic weight. Postdoctoral projects will either (i) combine genome engineering in stem cell-derived neurons with proteomic and live imaging as well as correlative light and electron microscopy approaches or use (ii) electrophysiology and multimodal imaging in genetically altered mice to unravel the mechanisms of presynaptic biogenesis and dynamic remodeling. These studies will thus fill a crucial knowledge gap in neuroscience.

Description of the Haucke lab

The focus of research in his laboratory is the dissection of the molecular mechanisms of endocytosis and endolysosomal membrane dynamics and its role in the nervous system with a focus on neurotransmission. The laboratory uses a wide range of technologies that include biochemical and cell biological approaches, electrophysiology, chemical biology, super-resolution and electron microscopy as well as genetic manipulations at the organismic level in vivo. The overarching goal of these studies is to mechanistically understand how exo-endocytosis and the endolysosomal system contribute to the development and maintenance of the nervous system function and how dysfunction may lead to neurological diseases.

Selected publications: Kononenko, N.L. et al Haucke, V. (2014) *Neuron*, 82, 981-988; Ketel, K et al. Haucke, V. (2016) *Nature*, 529, 408-412; Soykan, T. et al Haucke, V. (2017) *Neuron*, 93, 854-866; Vukoja, A. et al Haucke, V. (2018) *Neuron* 99, 1216-1232.e7; Kuijpers, M. et al Haucke, V. (2020) *Neuron*, in press

Contact information and links

For further information please contact: Volker Haucke (haucke@fmp-berlin.de)
Twitter: @VolkerHaucke
Lab: www.leibniz-fmp.de/haucke
Institute: www.leibniz-fmp.de/
Cluster of Excellence-Neurocure: www.neurocure.de/