ERA-NET NEURON

NEWSLETTER 35

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ANNOUNCEMENTS FROM NEURON COFUND

DON'T MISS!

2021 Call for Proposals on "Neurodevelopmental Disorders"

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COMING UP!

NEURON COFUND2 KICK OFF MEETING

January 27th, 2021

Click for Details

From the desk of the coordinator | January 2021

Marlies Dorlöchter

Dear All,

Amidst the distress we are all experiencing this autumn with the Corona pandemic and related restrictions and sorrows, a few good news might help to keep up a positive future view. The best from our perspective is that the NEURON proposal for further support from the EU Commission was most successfully evaluated. At the time of the publication of this newsletter, the grant agreement negotiations are proceeding and we can confidently look forward to not only kick-off the new five-year period, but also launch the new NEURON Joint Transnational Call (JTC) on "Neurodevelopmental Disorders" in January 2021.

In the Kick-off meeting on January 27th, 2021, we are excited to explore a new format with virtual round table panel discussions on various topics. Highly renowned neuroscientists, neurologists, and psychiatrists, representatives of the European Commission, as well as of patient organizations will address the coming challenges and opportunities in brain research.

Funded by the European Commission

More information can be found on our website
http://www.neuron-eranet.eu/index.php
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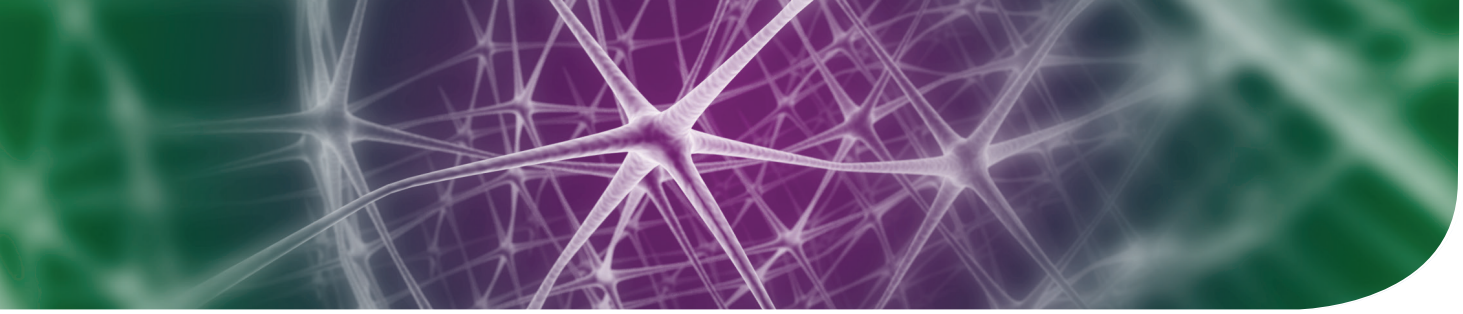


Back-to-back with our Kick-off meeting, we explore in a virtual Midterm symposium the progress of projects in the Joint Transnational Call 2018 on 'Mental Health'. Although this symposium is not public, we would like to share that we are looking forward to hearing promising approaches and results. As usual, the EXCELLENT PAPER IN NEUROSCIENCE AWARD (EPNA) will be awarded during this event. The award ceremony is always a jolly event, which is very well integrated in NEURON's efforts to support Early Career Researchers – read more about the EPNA 2019 in our interview on page 25.

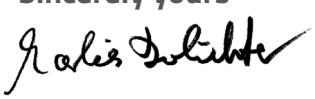
Embedded in the Midterm symposium is a workshop on quality in science, now second in line in conjunction with the project EBRA. These workshops aim to broadly inform and support the research community in the implementation of future (pre-clinical) research, since NEURON is fully committed to the highest possible standards of quality assurance, including working up to nationally and internationally recognized best practices. NEURON's implementation of standards in the design of animal research and full implementation of the 3Rs reflects contemporary good practice for all research using animals, and reinforces that these standards are important for ethical reasons and for obtaining the best possible scientific results.

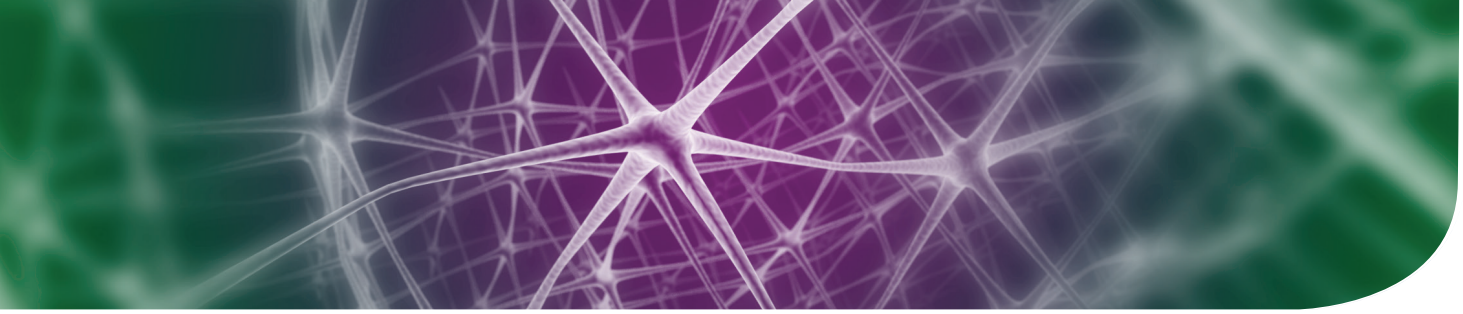
Not least, warmest congratulations to the successful applicants to the NEURON calls in 2020: Twelve multinational research consortia on "Sensory Disorders" and five on "ELSA of Neuroscience". In total, 65 research groups from 12 countries from Europe, Canada, and Israel collaborate in these projects. NEURON calls are always highly competitive, thus we expect excellent research and enthuse about the start of these projects, particularly in difficult times like now. Another highlight in 2020 was the active participation of patients in the proposal review of the "Sensory Disorders" call. With this, NEURON carries on and intensifies the collaboration with patients at various levels. The so-called public involvement is thus not only a lip service to match current fashions, but a serious and continued effort to improve our instruments and measures.

I would like to conclude with the good news that NEURON science and NEURON science funding will continue, and even if the coming times might appear long and cumbersome, I am confident that exciting results, success stories, and careers are to be expected.

Keep up with our website and follow us on twitter @EraNeuron in order not to miss further information about the Kick-off meeting and other events.

Sincerely yours





NEURON Joint Transnational Call 2020: Sensory Disorders

Twelve multinational research consortia were selected for funding under JTC2020 on 'Sensory Disorders'. In total, 48 research groups from 12 European countries, Canada and Israel collaborate in these projects, which cover various sensory systems. The total funding volume of the call amounts to about 9.6 M€. We wish them all remarkable achievements and hope their outcomes may one day assist in the prevention, diagnosis, therapy and/or rehabilitation of people living with sensory deficits!

NEURON Joint Transnational Call 2020: Ethical, Legal and Social Aspects of Neuroscience

Five transnational research consortia were selected to be funded under JTC2020 on 'Ethical, Legal and Social Aspects of Neuroscience'. In total, 17 research groups from 4 countries collaborate in these projects, which cover various ethical, legal and social aspects related to neuroscience. The total funding volume of the call amounts to about 3.5 M€. We wish them all great success with their research!

Sensory Disorders JTC2020

Artificial Intelligence for Diagnosing Retinal Diseases (AI D)

Jan Kremers

Project Coordinator:

Jan Kremers, Dept of Ophthalmology, University Hospital Erlangen, Erlangen, Germany

Project Partners:

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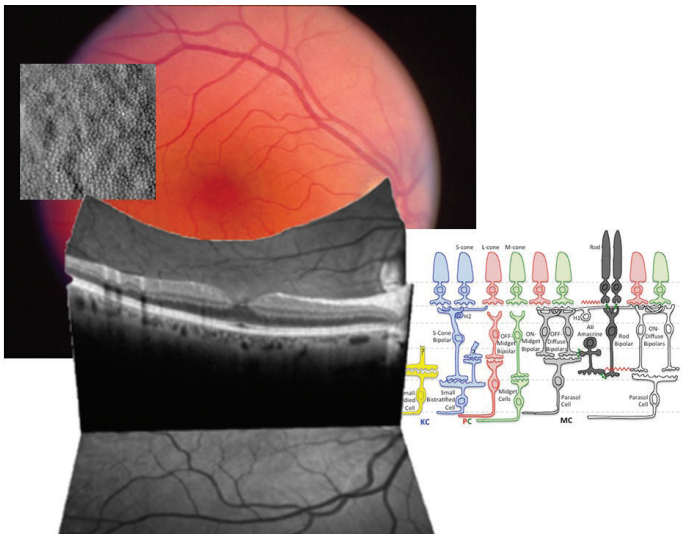
Isabelle Audo and Michel Paques, Centre d'Investigation Clinique (clinical investigation centre) des Quinze Vingts (Inserm-CHNO), Paris, France

Andreas Maier, Dept of Computer Science, Friedrich Alexander University Erlangen-Nürnberg, Erlangen, Germany

Blindness tremendously affects the quality of life of the patients. Treating and helping patients that suffer from diseases that lead to vision loss results in huge economic costs for society. Two major diseases that involve the light sensitive neural tissue at the back of the eye (the retina) are age related macular degeneration (AMD) and Stargardt disease (SD). To help patients optimally, the disease has to be diagnosed and treated as early as possible. In addition, if new treatments are introduced, sensitive methods should be available to monitor their effects. It is the goal of AI D to create these methods and techniques. The consortium will develop novel methods, with which the structure and function of retinal cells and cell circuitries can be studied and compared in patients and healthy subjects. New imaging techniques for the visualization of cell structures that are thought to be involved in AMD and SD will be established. Techniques for capturing the function of retinal cells and cell circuitries will be developed and refined. We expect a wealth of data that are difficult to analyse with conventional methods. Therefore, the large data sets will be analysed by novel techniques, such as artificial intelligence.



The functional role of cochlear synaptopathy for speech coding in the brain (CoSySpeech)

Sarah Verhulst

Project Coordinator:

Sarah Verhulst, Dept. of Information Technology, Ghent University, Belgium

Project Partners:

Jérôme Bourien, Institute of Neuroscience, Inserm/University of Montpellier, France

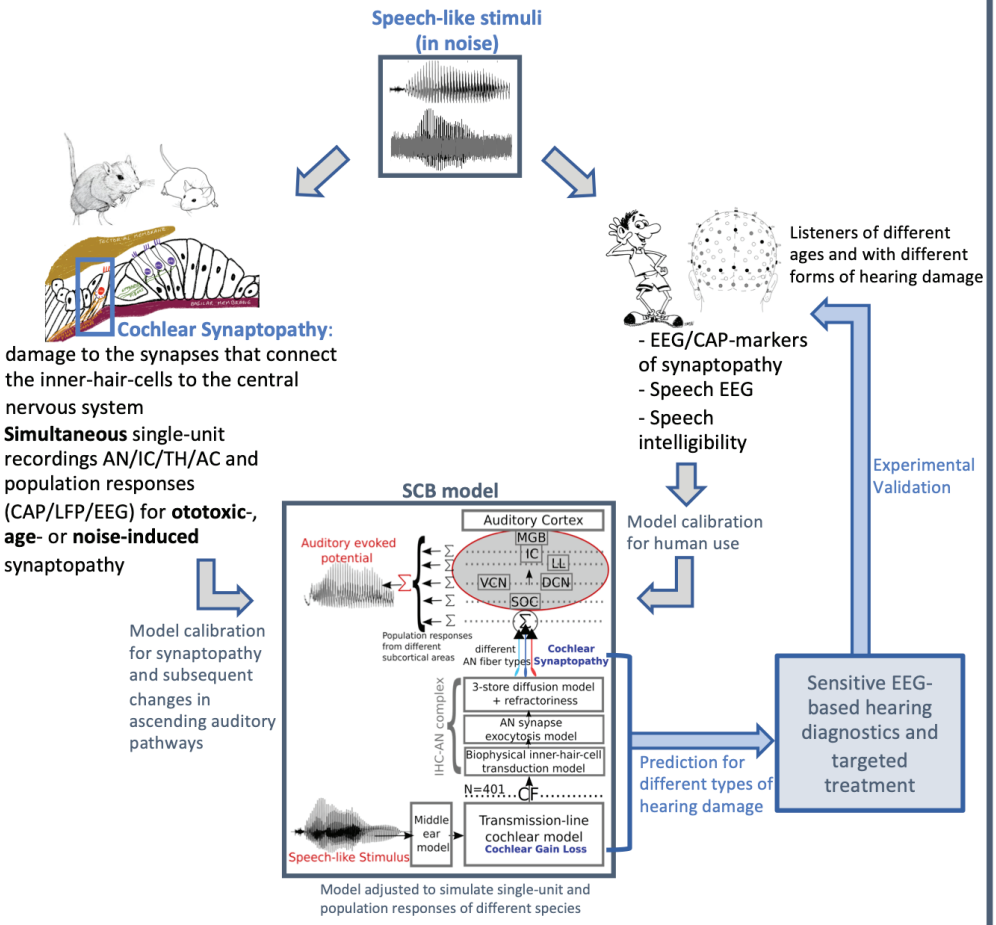
Manuel S. Malmierca, Institute of Neuroscience of Castilla y León, University of Salamanca, Spain

Marlies Knipper, Eberhard Karls University of Tübingen, Germany

Deniz Başkent, University Medical Center Groningen & University of Groningen, The Netherlands

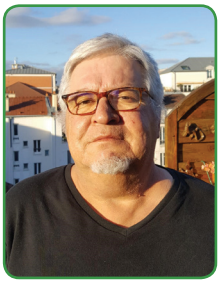
Inner ear (cochlear) synaptopathy is a form of primary neural degeneration, which damages synaptic connections between the auditory cells in the cochlea (sensory inner-hair-cells) and auditory-nerve fibers (neurons of the cochlear spiral ganglion). Our knowledge on synaptopathy is largely confined by animal histology studies which have shown that aging, ototoxic drugs and noise exposure can all cause synaptopathy without affecting hearing sensitivity. Consequently, cochlear synaptopathy is expected to affect more than 5% of the world population presently diagnosed with disabling hearing loss (WHO, 2011), urging the development of clinical screening protocols. At the same time, the functional consequences of synaptopathy for sound perception are poorly understood and therapeutic interventions largely non-existent. CoSySpeech aims to unravel, describe and manipulate the cascade of events occurring along the ascending auditory pathways after synaptopathy. This research will result in a unique, comprehensive framework for the functional aftermath of synaptopathy for speech coding in the brain (SCB-model), trendsetting the development of sensitive hearing screening methods and therapeutic interventions. Our consortium uniquely combines expert knowledge from different brain structures (periphery, brainstem, cortex) and spans various research modalities (histology, physiology, behavior, computational, behavior) to answer: "How does synaptopathy affect speech coding in the brain?"

The functional role of cochlear synaptopathy for speech coding in the brain (CoSySpeech)



Main project outcome: An *in-silico* functional model of speech coding in the brain (SBC-model) that can be personalized to develop future hearing diagnostic and treatment tools aimed to mitigate the aftermath of synaptopathy on speech coding.

Treating inherited binding disease with a slow released form of the rod-derived cone viability factor protein (DrEYE)



Thierry Léveillard

Project Coordinator:

Thierry Léveillard, Sorbonne Université, France

Project Partners:

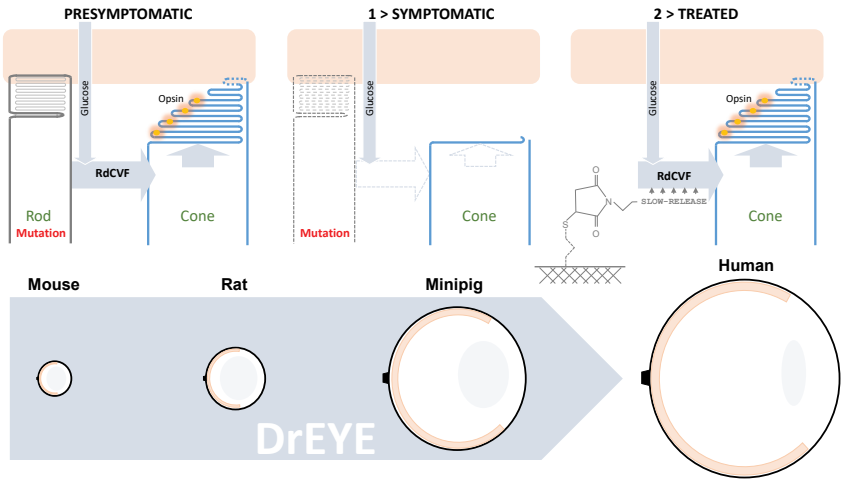
Molly Shoichet, University of Toronto, Canada

Eckhard Wolf, Ludwig-Maximilians-Universität München, Germany

Robert Fluhr, Weizmann Institute of Science, Israel

The cones concentrated at the center of the retina are essential for daily vision. The rods, which are active only in darkness, provide the cones with a signaling protein called rod-derived cone viability factor (RdCVF). RdCVF is necessary for central vision as it facilitates the absorption of bloodborne glucose by cones for the periodic reconstruction of the segment that captures light. Two million patients worldwide suffer from retinitis pigmentosa and become blind from inherited mutations that kill rods and consequently abolish RdCVF production. Our aim is to prevent this from happening by restoring RdCVF using a slow release form of RdCVF.

Our group will study the feasibility of such protein therapy using rodent and porcine models of the targeted disease, retinitis pigmentosa. The success of this proof of concept for a future human therapy relies on the competence of the consortium of experts in their respective domains. Rehovot will produce research grade RdCVF protein, coupled to a matrix for slow release in Toronto. Paris and Toronto will test this therapeutic protein in rodents. Munich will provide the pig model that will ultimately be tested in Paris. Dr. EYE explores a new promising therapeutic approach of this, until now, untreatable disease.



Ion channel modulators to treat itch (ICMI)

Frank Bosmans

Project Coordinator:

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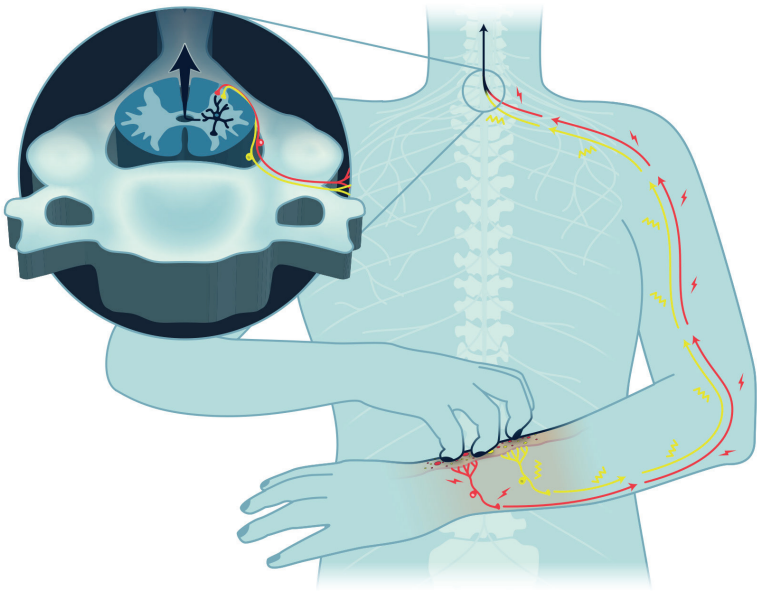
Project Partners:

Michel De Waard, Nantes University, l'institut du thorax, Nantes, France

Filip Van Petegem, University of British Columbia, Life Sciences Centre, Vancouver, Canada

Chronic itch can be devastating to such an extent that some patients report a willingness to accept a shorter lifespan in return for an alleviating treatment. Itch, or pruritis, is commonly associated with a primary skin disorder; however, in a substantial subset of patients, pruritis originates from an unknown origin. Even though pruritus occurs in nearly 20% of adults, treatment options are typically limited to over-the-counter drugs or not effective. To efficiently address itch disorders with safe therapeutics, a vital step is to enable pharmacological manipulation of relevant sensory neuron pathways, which is the essence of the project that we propose here. We will build on our previous work in which we reported a clinical case of debilitating itch resulting from a mutation in SCN11A, a gene that encodes for $Na_V1.9$ which is involved in transmitting sensory signals from the skin to the brain. To further investigate the role of $Na_V1.9$ in itch, we propose to establish a discovery platform to help identify molecules capable of tackling the clinical problem of chronic itch and dramatically expand our understanding of its working mechanism. The results of this project will ultimately lead to new, more effective and safe itch treatments.



Improving postural control by innovative stimulation of the proprioceptive system (IMPULSES)

Stéphane Baudry

Project Coordinator:

Stéphane Baudry, Laboratory of Applied Biology and Research Unit in Applied Neurophysiology (Head of the LAB Neuro), Erasme, Faculty for Motor Sciences, Université libre de Bruxelles, Brussels, Belgium

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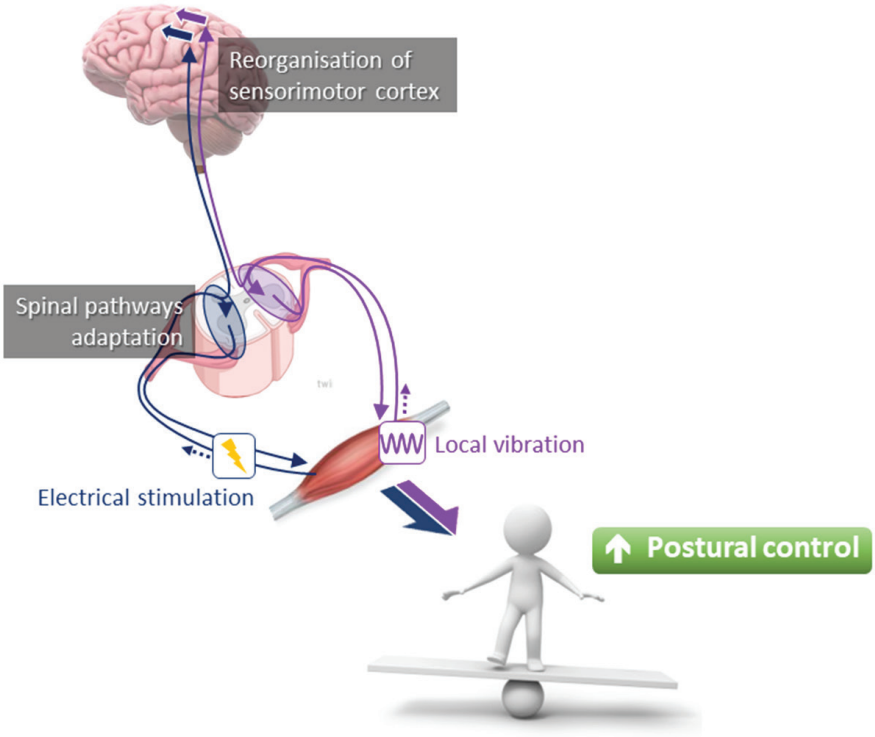
We all know our five senses: hearing, smell, touch, vision and taste. However, there is another one that we use constantly, even if we are not aware of its existence: the proprioception. It is the sense that informs our brain about the position of the different parts of our body, which allows us to know at any time its position and its interactions with the surrounding environment, and guides our movements without resorting to vision.

Proprioceptive sensors and other sensory organs (vision, inner ear) permanently send signals through our nerves to the spinal cord, then to the brain which analyses them, and depending on the situation sends commands to contract or relax certain muscles (sensorimotor integration).

Like all physiological systems, proprioception can dysfunction. When we consider the extent of its action, we realize that the symptoms of proprioceptive dysfunction can be very debilitating, notably by altering our balance and walking capacities; this is the case in children with cerebral palsy (CP) and older people. However, therapeutic interventions to improve or even retain proprioception remain unoptimized.

IMPULSES aims to provide a better understanding of how the alteration of proprioception interferes with balance and gait control, and to study whether the innovative combination of stimulating the proprioceptive system (by small electrical current or mechanical stimuli) while performing postural exercises can optimise the effects of postural exercises on posture and gait control thanks to improved proprioception, in children with CP and the elderly.

The scientific evidence from IMPULSES will open up new concrete perspectives and provide essential basic knowledge about proprioception, which could be extended to other clinical purposes.



IMPROVING POST**U**RAL CONTRO**L** BY INNOVATIVE **S**TIMULATION OF THE PROPRIOCEPTIV**E** **S**YSTEM

IMPULSES

Improving intracortical visual prostheses using complex coding and spontaneous activation states (I-See)

Udo Ernst and David Rotermund

Project Coordinator:

Udo Ernst and David Rotermund, Computational Neurophysics Lab / Theoretical Neurophysics, Institute for Theoretical Physics, Universität Bremen, Bremen, Germany

Project Partners:

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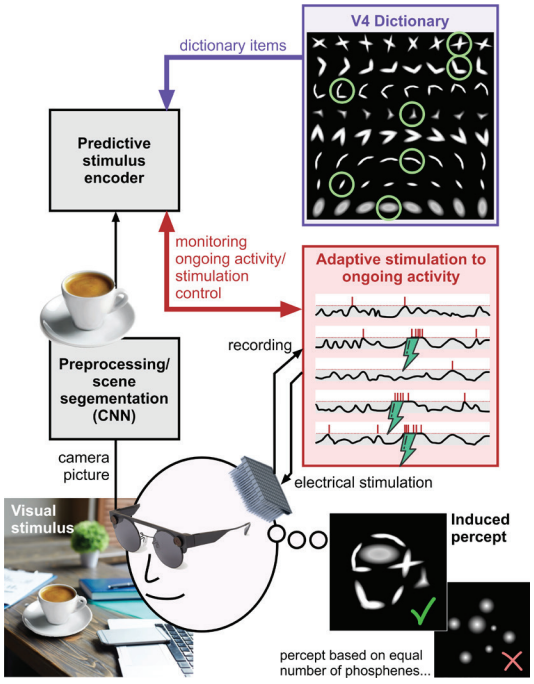
Dirk Jancke, Optical Imaging Lab, Institut für Neuroinformatik, Bochum, Germany
Michael Herzog, Laboratory of Psychophysics (LPSY), École Polytechnique Lausanne, Lausanne, Switzerland
Bogdan Draganski, Laboratoire de Recherche en Neuroimagerie (LREN), Centre Hospitalier Universitaire Vaudois, Université de Lausanne, Lausanne, Switzerland

The lives of many individuals are strongly impaired by missing the sense of vision. One putative option to help them are cortical prostheses. For restoring vision, such devices convert visual scenes into patterns of electrical activity which are then transferred to the visual cortex (typically area V1). This approach aims at using thousands of electrodes, each eliciting the percept of a circular flash of light (phosphene).

Our project evaluates alternative ideas: First, we will stimulate neural populations in visual area V4 with the goal to achieve more specific percepts (e.g. corners, edges, curves) than phosphenes, while using a smaller number of activated stimulation electrodes. Our second aim is to make use of ongoing activation states in cortex that exist even without a visual stimulus. By devising an intelligent stimulation algorithm which boosts these states at the right moment in time we plan to achieve more 'natural' percepts with much lower stimulation currents.



Supported by a strong backbone of computational and theoretical neuroscience approaches, our project encompasses developing subthreshold stimulation techniques in mice, testing novel stimulation paradigms in non-human primates, evaluating sparse stimulus encoding schemes in healthy humans, and assessing brain anatomical and functional constraints of cortical prostheses in the blind.

Tactile sensory impairment of C-LTMR afferents in preterm children and interventional approaches (PreTouch)

Uta Sailer

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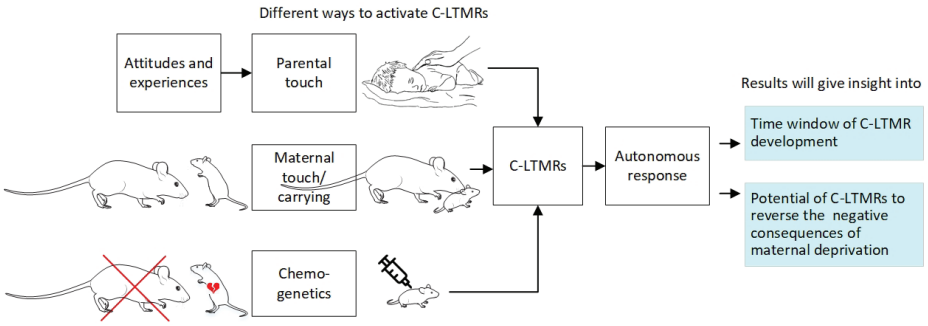
Krisztina Lakatos, Institute for Cognitive Neuroscience and Psychology, Research Centre of Natural Sciences, Budapest, Hungary

Touch is of central importance in social interactions and early development of mammals. For touch used in maternal care - licking behaviour in mice and slow stroking in humans - one type of neural pathway is particularly important. This pathway starts in skin receptors called C-low-threshold mechanoreceptors (C-LTMRs). Touch transmitted by C-LTMRs enables the newborn to regulate arousal and emotions.

The project aims to investigate sensory impairment of C-LTMR-mediated touch in preterm-born infants and to map the time window of C-LTMR development. The effects of such sensory impairment and of parental characteristics on the interaction of parents with their children will also be studied. Using an animal model, we will quantify how selectively inhibiting or activating C-LTMRs influences offspring reactions to arousal and social interaction. We will also investigate if the negative consequences of maternal deprivation in mouse pups can be reversed by selective chemogenetic activation of C-LTMRs.

The project could deliver results important for understanding tactile sensory and social impairments and their potential restitution, thereby providing ground-breaking insights into sensory developmental processes and laying the ground for a clinical intervention in preterm-born infants.



Human organoid system based therapy discovery & development for age-related macular degeneration (ReDiMoAMD)

Mike O. Karl

Project Coordinator:

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Project Partners:

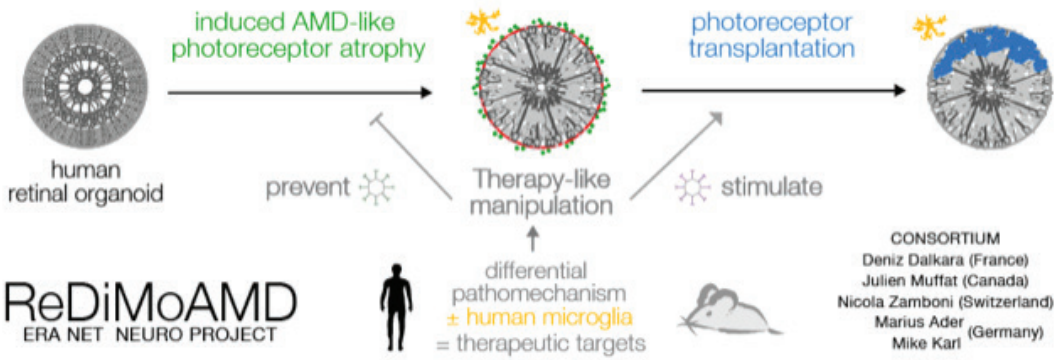
Marius Ader, TU Dresden, CRTD, Dresden, Germany

Deniz Dalkara, Institut de la Vision, Paris, France

Julien Muffat, Hospital for Sick Children, Toronto, Canada

Nicola Zamboni, ETH Zürich, Inst. Of Molecular Systems Biology, Zürich, Switzerland

Age-related macular degeneration (AMD) causes vision loss due to outer retinal atrophy, but disease models for the neural retina and therapies are missing. Photoreceptor cell transplantation represents a promising treatment approach, but clinical translation remains a challenge. Our consortium aims to bridge two related research gaps: modeling of AMD pathology and therapy development (preventive and restorative). We developed a human retinal organoid model with cellular and molecular AMD hallmarks. The photoreceptor pathomechanisms in this model might offer targets to prevent AMD and to restore it by photoreceptor transplantation – since both might share common and/or interrelated processes. For example, metabolic stress and inflammation, which are key in AMD, and known triggers of neurodegeneration in other organs, might cause photoreceptor atrophy and affect transplant integration success. Thus, the aims of the consortium are to develop, optimize and apply organoid and viral tools to decipher pathomechanisms by comparing our human and mouse models, determining the functions of inflammation (microglia) and metabolic changes. We will explore latter findings in preclinical studies to find potential targets for therapeutic prevention of pathogenesis and optimization of photoreceptor transplantation.



Gap junctions serve to distribute health-signals among neurons of the diseased retina (Rethealthsi)

Béla Völgyi

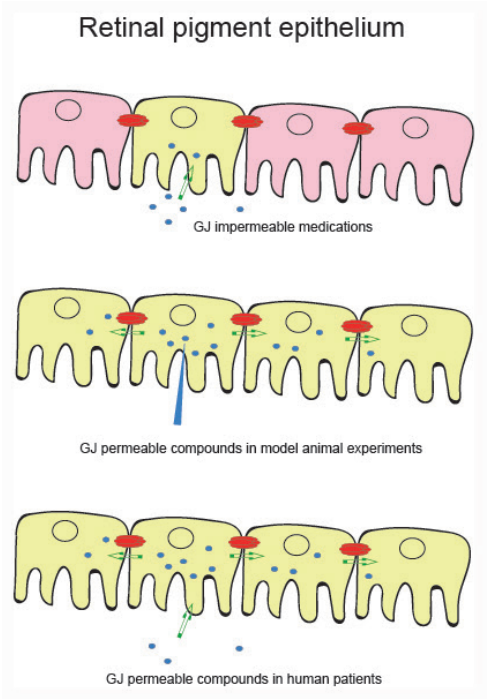
Project Coordinator:

Béla Völgyi, University of Pecs, Hungary

Project Partners:

Stanislao Rizzo, Policlinico Universitario Agostino Gemelli IRCCS, Italy

Karin Dedek, Carl von Ossietzky Universität Oldenburg, Germany



In retinal diseases, the loss of directly insulted cells is accompanied by a secondary en mass cellular death due to the so called bystander effect. Although bystander effect blocking interventions may have therapeutic value, the underlying mechanisms are unknown. Hypothetically, gap junctions (GJs - communication routes between cells) pass 'death-signals' from dying cells to neighbours, thus, it has been put forward that a GJ blockade could rescue cells in progressive diseases, such as diabetic retinopathy, Retinitis Pigmentosa (RP) or glaucoma. However, a GJ blockade for a chronic treatment would deteriorate vision as well. We propose a treatment modality, in which GJs are utilized as avenues for cell-to-cell diffusion of health-signal molecules. We will characterize the physical and chemical barriers of this GJ mediated diffusion of potential health-signals (endogenous molecules, epigenetic factors and medications) in both healthy and RP retinas. RP mutations cause the degeneration of rods (night-vision) and a bystander effect mediated secondary death of cones (daylight vision). Our goal is to characterize health-signals that by crossing GJs, reach many cells in the RP retina and rescue vision. Our long term aim is to develop a new therapeutic modality to arrest vision loss by ocular delivery of health-signal molecules.

Key collaborators: Prof. Enrica Strettoi (Istituto di Neuroscienze Consiglio Nazionale delle Ricerche: Pisa, Toscana, IT) ; Prof. Róbert Gábriel (University of Pécs, Pécs HU).

Targeting Sensory Dysfunctions in Autism Spectrum Disorders (SensingASD)

Peter Soba

Project Coordinator:

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Project Partners:

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Claudia Bagni, Dept of Fundamental Neurosciences, University of Lausanne, Lausanne, Switzerland

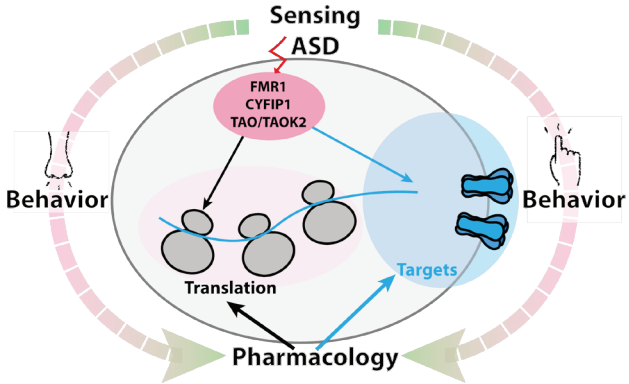
Karun Singh, University Health Network, University of Toronto, Toronto, Canada

Stephen Scherer, The Center for Applied Genomics, Hospital for Sick Children, University of Toronto, Toronto, Canada

Baiba Jansone, Dept of Pharmacology, Faculty of Medicine, University of Latvia, Riga, Latvia

Dysregulation of sensory perception is part of the core symptoms of Autism Spectrum Disorders (ASDs). For the longest time, it was thought that sensory issues in ASDs are due to disruption of the central nervous system (brain). Emerging evidence indicates that sensory and social deficits arise in part due to the abnormal development and function of the sensory nervous system, opening up new ways to study and treat sensory changes in ASDs.

Our multidisciplinary consortium will study whether high confidence genetic factors for ASD cause sensory dysfunction by disrupting the production of proteins required for sensory nerve cell communication. To address this question, we will use a combination of genetically engineered flies, mice, and stem cell-derived human sensory neurons. We will perform behavioral experiments to identify sensory defects and investigate whether the production of proteins in neural cells is abnormal. Finally, we will test whether specific drugs and genetic therapies that correct abnormal protein production will reverse cellular and behavioral dysfunctions. Our studies will generate new evidence that genetic mutations directly impair protein production in sensory neural cells, which leads to sensory and social deficits. Our study will open new avenues for treating sensory abnormalities related to ASDs.



The role of translational dysregulation in sensory neurons in mediating tactile hypersensitivity in neurodevelopmental disorders (TRANSMECH)

Christos Gkogkas

Project Coordinator:

Christos Gkogkas, Foundation of Research and Technology - Institute for Molecular Biology and Biotechnology – Division of Biomedical Research, Greece

Project Partners:

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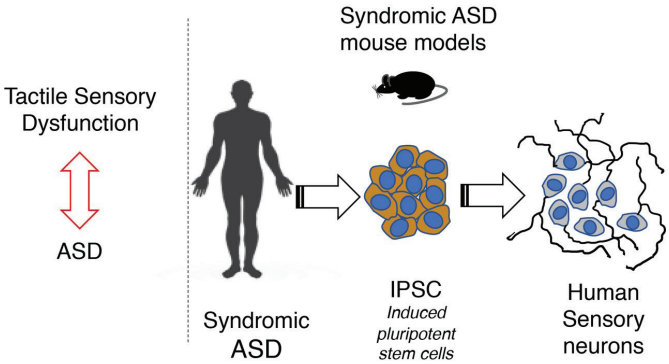
Gary Lewin, Dept of Neuroscience, Max-Delbrück Center for Molecular Medicine, Germany

Approximately 95% of people diagnosed with Autism Spectrum Disorder (ASD) have difficulty processing everyday sensory information, while 60% display altered touch sensation, which can have a profound effect on a person's life. During development, gentle touch communication between mother and infant is essential for proper development of complex behaviours, such as social interaction. Over-sensitivity to touch is observed both in ASD patients and in engineered mouse models carrying mutations of genes linked to ASD. It is plausible that over-sensitivity to touch in ASD patients may be linked to behavioural alterations such as deficits in social interaction. We do not fully understand the mechanisms that account for touch over-sensitivity in ASD. While the precise causes of ASD are unknown, it is established that several risk genes are associated with this disorder. A molecular/cellular pathway that is linked to these ASD risk genes and is commonly affected in ASD patients is involved in regulation of protein synthesis via mTORC1/4E-BP1. We found that selective activation of mTORC1/4E-BP1 only in sensory neurons in mice induced increased synthesis of proteins linked to mitochondria, the cell's powerhouses, which have a central role in energy metabolism. Thus, we hypothesise that the mTORC1/4E-BP1 pathway may be responsible for touch oversensitivity in neurodevelopmental disorders, such as ASD, by favouring the synthesis of certain proteins over others (e.g. mitochondrial proteins). To test this hypothesis in project TRANSMECH, we will use mouse models and human sensory neurons carrying mutations in risk genes found in ASD patients. Understanding this mechanism will lead to the development of novel therapies for treating touch oversensitivity in ASD and also for reversing the core symptoms of ASD (e.g. social behaviour alterations).



Vestibular Loss and Spatial Orientation (VELOSO)

Desdemona Fricker

Project Coordinator:

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Project Partners:

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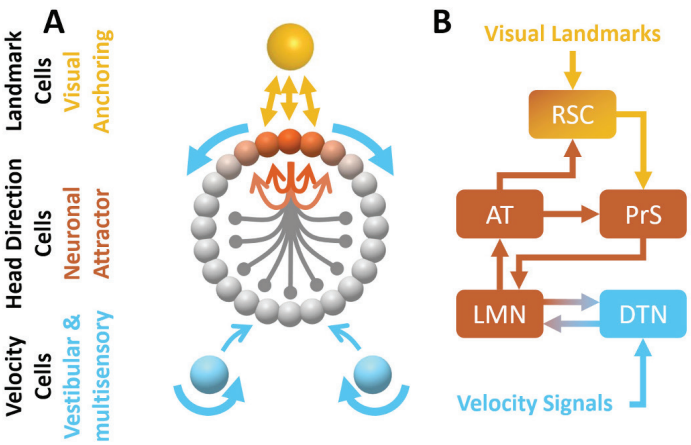
The sense of orientation in space is crucial for survival and navigation in the environment. The brain derives spatial orientation from (1) self-motion signals originating in the vestibular system of the inner ear and (2) visual signals encoding orientation relative to external landmarks.

Loss of vestibular function, which occurs with 7.4% lifetime prevalence, degrades both the perception of orientation in space and the capacity to navigate even in familiar visual environments. We will ask why and how vestibular dysfunction affects the use of visual cues as landmarks in spatial orientation. We hypothesize that cross-modal integration is degraded; vestibular deficits prevent the brain from binding visual landmarks to particular orientations, thus impeding navigation even in familiar visual environments.

Our consortium proposes work in rodent models and non-human primates. We have expertise on the consequences of vestibular deficits, in vivo recordings of neurons in the brain's navigation system, sensory interactions, neuronal circuits and modeling. Anatomical, physiological and, theoretical approaches will let us ask how vestibular deficits compromise the sense of spatial orientation. Our results will serve translational initiatives to help improve diagnosis and develop novel re-education strategies for patients with vestibular dysfunction.



ELSA JTC2020

Optimizing the aging Brain? Situating Ethical Aspects of Dementia Prevention (BEAD)

Silke Schicktanz

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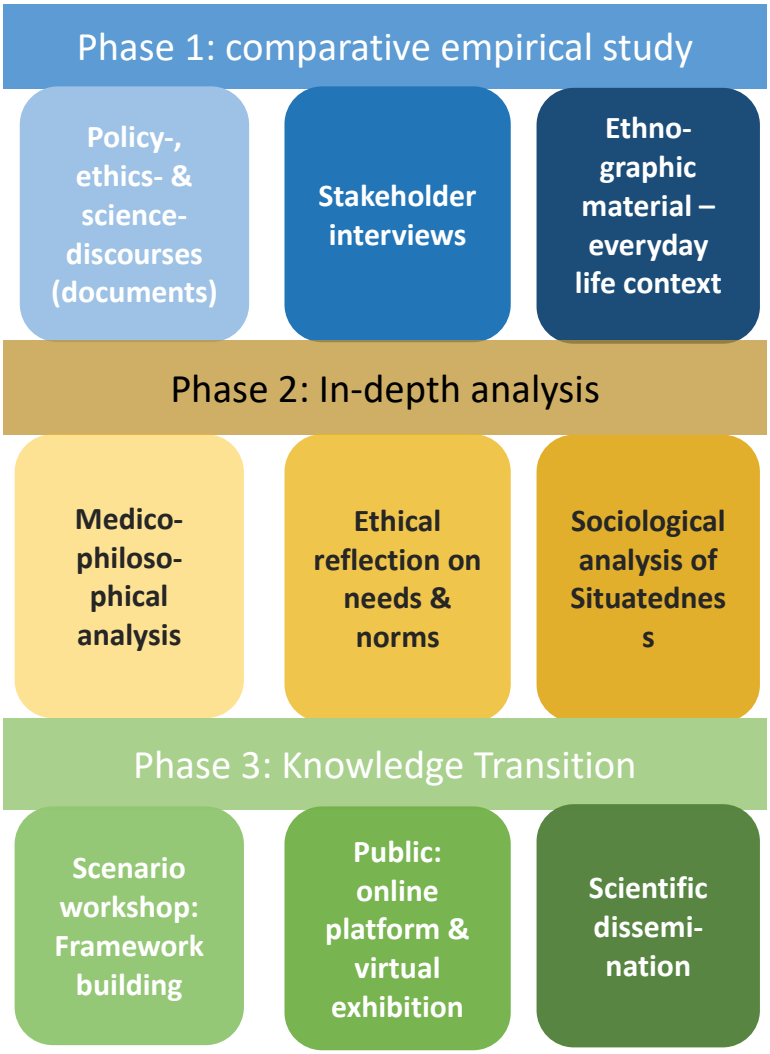
Background: Dementia (the most common form is Alzheimer's disease) has for a long time been conceived of as an unpreventable process of mental deterioration. Now, however, researchers claim that one out of three dementia cases can be prevented if at least nine risk factors are managed. These risk factors include some that have not been traditionally linked to Alzheimer's, such as diabetes, hypertension, social isolation, and hearing loss, and which require management over the life course, making prevention a lifelong endeavour. Early detection through biomarkers and digital tracking technologies have also become a central feature of what we call 'the new dementia'. This raises a number of ethical questions, such as: should individuals be considered personally responsible for their own dementia prevention? Should we accept extensive monitoring of our cognitive functions through digital technologies? What does dementia prevention entail for our healthcare systems and cultures of care? And, do national or local contexts play a role in how prevention is articulated and lived?
Objectives: We propose a research project that studies in three countries the way the 'new dementia' is being articulated, and asks what ethical problems are arising, what the impacts are on health-related and everyday practices, and what solutions might be envisioned.
Methodologies: We will study the 'new dementia' through complementary disciplinary approaches. We will adopt a comparative perspective looking at differences and similarities between three countries: Germany, Canada and Switzerland.
Expected outcomes: Our results aim to make people more aware that preventing dementia is a highly complex issue that needs to be seen in context through an extensive knowledge transfer on multiple levels.


Phase 1: comparative empirical study
Policy-, ethics- & science-discourses (documents)
Stakeholder interviews
Ethno-graphic material – everyday life context
Phase 2: In-depth analysis
Medico-philoso-phical analysis
Ethical reflection on needs & norms
Sociological analysis of Situatedness
Phase 3: Knowledge Transition
Scenario workshop: Framework building
Public: online platform & virtual exhibition
Scientific dissemi-nation

Hybrid minds: experiential, ethical and legal investigation of intelligent neuroprostheses (HYBRIDMIND)

Jennifer A Chandler

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Marcello Ienca, ETH Zurich, Dept. of Health, Sciences and Technology, Switzerland

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Intelligent neuroprostheses represent the next phase in the evolution of devices integrated with the nervous system to assist, replace or alter human sensory, motor, cognitive, and affective functions. These devices include "read out" or output systems that detect, interpret and translate neural signals for various applications such as to move a robotic arm or cursor. They also include "write in" or input systems that deliver signals or stimulation to the brain to alter thinking, emotions, and the ability to move. The technology increasingly incorporates Artificial Intelligence (AI) to create devices characterized by mutual adaptation, in which both user and self-learning algorithm change over time to optimize system output. The integration of AI with human brains and minds into hybrid minds is a departure in terms of its complexity, unpredictability, and psychological impact. Our project pursues a unified theoretical approach to the ethical-legal assessment of intelligent neuroprostheses, informed by the perspectives of users, the neuro-engineering community and other key stakeholders, culminating in the questions: Which AI-elements should future neuroprostheses incorporate or leave out? What technical design choices or regulatory measures are required to proceed safely? How can we support patients in clinical decision-making to avoid overblown hopes and to know, to borrow Thomas Nagel's famous phrase, "what it's like to have a hybrid mind"?

International neuroethics patent initiative (INPI)

Tade M. Spranger

Project Coordinator:

Tade M. Spranger, University of Bonn, Centre for the Law of Life Sciences, Bonn, Germany

Project Partners:

Judy Illes, University of British Columbia, Division of Neurology, Vancouver, Canada

Patents aim to protect inventions and provide protection to inventors for their intellectual achievements. Past research from our teams has shown that, in the area of brain science, such protections can extend beyond methods or devices for which they are intended and permitted, and cover regions of brain tissue and even aspects of behaviour that may be recorded or stimulated. Our goal here is to fully examine patents already granted in the field of neuroscience – drugs, software, technology – examine them for their legal viability, and explore perspectives from experts about their potential benefits and risks to individuals, patients and society. The outcome of this work will be both practical guidance for the future of patents in neuroscience, as well as evidence-based recommendations for boundaries that ought to be drawn.

The predictive turn in alzheimer's disease: ethical, clinical, linguistic, and legal aspects (PreTAD)

Christiane Woopen

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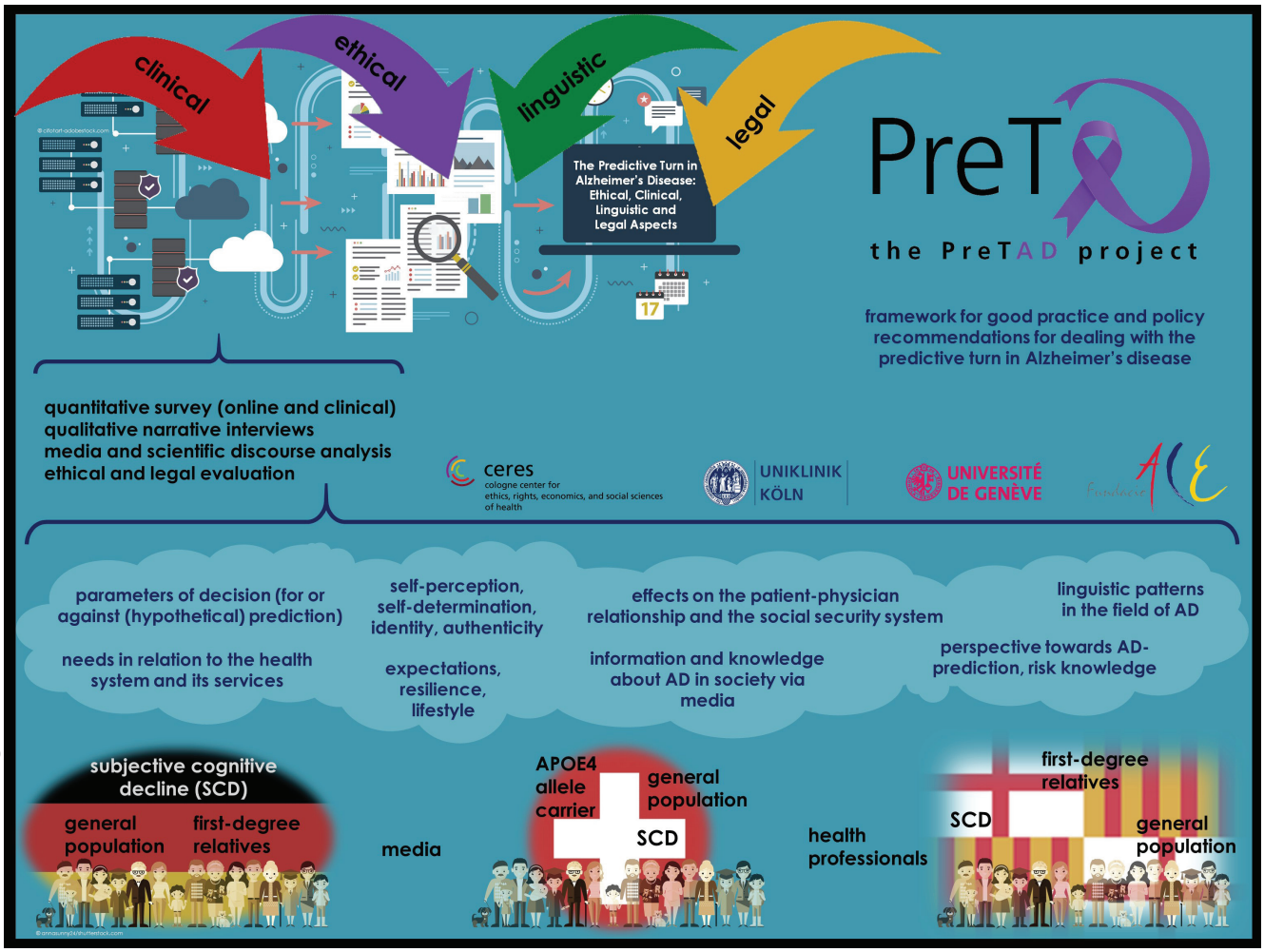
Predictive medicine is becoming better, more reliable, and more accessible. A paradigm shift in medicine is emerging in which diagnostics on the basis of disease symptoms and therapy are complemented or even replaced by predictive diagnostics and prevention. However, this predictive turn does not simply mean improved possibilities of medical care, but deeply affects the life of individuals, the health care system, and our society as a whole.

In the field of Alzheimer's disease (AD), realities such as the constant improvement of risk estimation, or of the accuracy of blood tests for a large-scale screening of AD biomarkers, as well as the possibility of earlier detection in cognitively unimpaired individuals might change the individual and public attitude towards predictive medicine and medicine in general.

PreTAD aims to clarify people's needs in relation to the health care system and its services, people's diversified perspectives towards prediction, and the effects of predictive medicine on individuals, language, public discourse, regulation, and the health care system to develop a framework for good practice regarding prediction of AD, including policy recommendations for dealing with the increasing predictive turn in medicine.



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Brain stimulation reconsidered – participative development of a code of conduct for the european union (STIMCODE)

Martina Schraudner

Project Coordinator:

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Project Partners:

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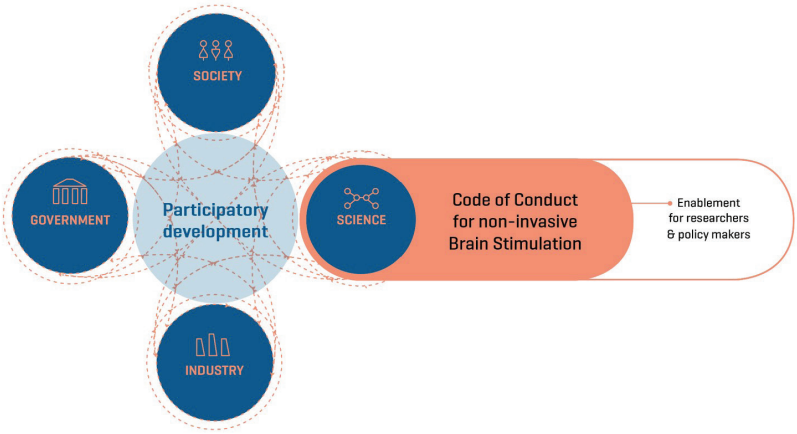
Georg Northoff, Royal Ottawa Mental Health Centre, Canada

Antonio Oliviero, FENNSI Group and Neurology Unit, Hospital Nacional de Parapléjicos, Spain

Nerve cells can be influenced by electrical or magnetic impulses. Noninvasive brain stimulation (NIBS) uses this ability and is able to inhibit or activate certain areas of the brain. NIBS can be used in healthy people, but research has focused primarily on diseases in which certain areas of the brain are restricted. However in addition to clinical research, a large market has developed in which people buy devices for cognitive enhancement or for self-treatment of neuropsychiatric conditions, and use them at home without medical supervision. This development, inter alia, raises questions that are currently difficult for researchers and governments to answer. For example, where exactly is the border between enhancement and treatment? Should the procedures also be used to treat children? In order to address questions like this in a reflective manner, this project aims to develop a participatory code of conduct for NIBS. To this end, actors from various scientific disciplines, society, business and governments will be involved. The involvement of laypersons and various expert perspectives will provide scientists with a tool to reflect on their own research and governments will be able to use the Code of Conduct for NIBS to create long-term guidelines that are valid EU-wide.



2019 EPNA AWARDEE
Dr. Alberto Parras

Personal Interview

The Excellent Paper in Neuroscience Award (EPNA) initiative was first introduced by NEURON in 2009, in order to support and encourage young scientists in the neuroscience field at the early stage of their career. The winners of the award receive a cash prize, as well as an invitation to present their work as special ERA-Net NEURON Young Investigators speakers in an international conference. A fruitful collaboration with the Federation of European Neuroscience Societies (FENS), gives the awardees an opportunity to present their work at the reknowned FENS Forum, which took place virtually this year.

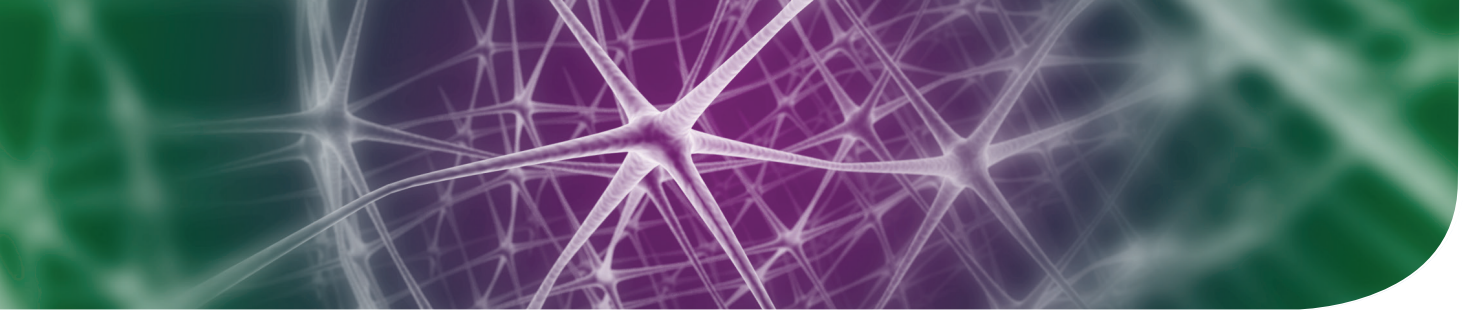
Alberto Parras

The 2019 EPNA awardee is **Alberto Parras**. Dr. Parras is currently a postdoctoral fellow in neuroscience and aging at the University of Lausanne, Switzerland, exploring epigenetic reprogramming in the nervous system. The award was given to Dr. Parras for his publication from his PhD studies in the lab of Prof. Jose Lucas in Madrid: 'Autism-like phenotype and risk gene mRNA deadenylation by CPEB4 mis-splicing' published in Nature 2018: 560, p. 441–446 (www.nature.com/articles/s41586-018-0423-5).

Continue reading to learn more about Alberto and his scientific journey.

1. Please tell us briefly about your research interests.

Since I was a child, I have been interested in biomedical research in general. I could not choose a specific field because I have always been attracted to answer any scientific question that was coming to my mind. Actually, I am more focused on "in vivo" projects and ideas that can be applied in translational research.



2. Please tell us about your scientific journey to-date.

I obtained my Ph.D. in July of 2017 at the Center for Molecular Biology in Madrid. I investigated mRNA polyadenylation and translation within the context of neurological diseases and furthermore we developed a novel therapy for Huntington's disease.
In November of 2018, I continued my postdoctoral training at the Royal College of Surgeons in Dublin where we made a key finding that mRNA polyadenylation is a novel regulatory mechanism of gene expression in temporal lobe epilepsy.
From September of 2019, I got a postdoctoral position in the laboratory of Prof. Alejandro Ocampo in the University of Lausanne. This lab is focused on interrogating epigenetic mechanisms of aging across multiple models along with the development of novel anti-aging interventions based on cellular reprogramming. Recently, I originated an independent line of research focused on epigenetic reprogramming in nervous system.

3. What made you choose a career in your field?

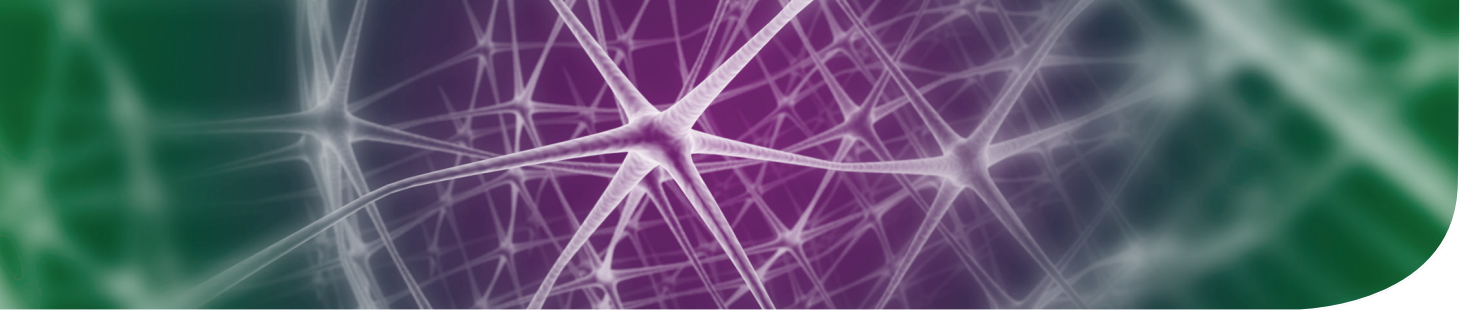
As I mentioned before, I am more interested in the approach than in the field. For that reason, I have always chosen hybrids labs, which work in translational research, combining "in vivo" experiments and bioinformatics analysis.

4. Where do you see your field of research in a few years? What are going to be the major breakthroughs?

Currently, I am studying aging in the context of neurodegenerative disorders. Aging is one of the main risk factors for most human diseases of developed countries but despite decades of research, it is poorly understood. In the last years, funding in aging research has increased drastically and I am sure that in this decade we will see a huge advance in this field. Many new drugs and therapies will be developed, which will have a positive effect on the healthy life span of the society.

5. What were the main challenges you had overcome in your career path and how did you overcome them?

I obtained my PhD in Spain, where the investment in scientific research is not very big and the salaries are relatively low. I was lucky to have worked in Jose Lucas's Lab, one



of the most talented scientists in the country. Particularly, the project for which I was awarded the EPNA was never funded. We had to save money from other projects and to work many extra hours to finish it properly.

6. Had COVID-19 impacted your research, if so how?

Yes, I think like everyone, my research has been adversely impacted by the COVID pandemic this year, specifically "in vivo" projects. However, during this time, I could keep alive and age most of the mice and I also took advantage of my "freed-up" time to write and to publish a couple of papers from my previous postdoc. I also used the time to read a lot and apply for several grants for the following years.

7. What are your goals for the future and where would you like to see yourself 5 years from now?

I got my postdoctoral position in Switzerland 1 year ago. I had always combined my professional research activities with my passion for sports, particularly Triathlon. This country offers me a high quality of live, a very good professional opportunities and the best natural conditions for my sport trainings. For that reasons, I see myself working here for the following years. Actually, last month I applied for a National Grant for senior postdoc researchers, who intend to pursue a scientific and an academic career at a Swiss research institution. This grant would allow me to become scientifically self-reliant at an early stage with an independent research project for 5 years.

8. What advice would you give your younger self or young scientists beginning their research career?

Do not forget that scientific research is our job and in most cases it is something that we really like to do. But, we should always enjoy our work and we should never be obsessed with being successful. We have to be consistent and patient and the success will find us. Moreover, I find my reframe, refocus or reset in sports. By practicing sports, I keep my body and my mind healthy and young. Thus, I would encourage all students to have, additionally to their research, their hobbies and social life in different environments and to feel good about themselves.