ERA-NET NEURON

NEWSLETTER 50

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Announcements from Neuron

JUST LAUNCHED – EP BrainHealth Calls for Proposals
on
Biological, social and environmental factors that impact the trajectory of brain health across the lifespan

DON'T MISS CALL 1!

JTC2026 Call 1:
In the field of
Neurological, Mental and Sensory Disorders

Pre-proposal submission deadline:
10 March 2026 | 14:00 CET

Details

DON'T MISS CALL 2!

JTC2026 Call 2:
In the field of
Neurodegenerative Disorders

Pre-proposal submission deadline:
10 March 2026 | 14:00 CET

Details

NEW PUBLICATION!

ERA-Net NEURON in PLOS Biology-
Human-centered, rigorous, ethical funding in practice
Lessons from ERA-Net NEURON on embedding patient and carer perspectives, ethical awareness, and research quality into biomedical research funding practice.

Read the paper

Funded by the European Commission

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

Produced by CSO-MOH, IL

From the desk of the coordinator | January 2026

Dear All,

As we enter 2026, ERA-Net NEURON does so at a defining moment. This year marks both a culmination and a transition. It is the final phase of ERA-Net NEURON's long-standing commitment to advancing high quality research on brain health, neuroscience, and mental health, and at the same time the beginning of a new chapter with the launch of the European Partnership for BrainHealth (EP BrainHealth). Looking ahead, 2026 offers an opportunity to reflect on the collective achievements built over many years, while ensuring continuity, momentum, and shared ambition as our community moves forward.

This issue of the ERA-Net NEURON newsletter focuses on the 13 consortia funded under our Joint Transnational Call 2025 (JTC2025) on Interdisciplinary Approaches to the Neuroscience of Pain. Further information on the call, its outcomes, and the funded projects can be found on page 4.

As part of our continued support to funded consortia, coordinators of projects selected under the JTC 2025 call are invited to participate in the workshop Open Science Support for Funded Projects, which will take place from February 25th to February 27th, 2026 in Berlin. This workshop is part of a long running ERA Net NEURON series aimed at supporting funded projects with aspects of Responsible Research and Innovation including methodological rigor, ethical awareness, and meaningful patient involvement. In addition to practical guidance, the workshop will provide valuable opportunities for exchange, collaboration, and networking among consortia working together across borders.

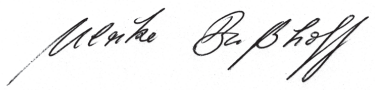
These capacity building activities have also contributed to ERA-Net NEURON's recent publication in PLOS Biology (Lichtenberg & Müller et al., 2025), developed by members of the NEURON coordination office and in collaboration with patient representatives and the Berlin Brain Institute of Health/QUEST Center for Responsible Research. This work presents a comprehensive framework to strengthen methodological rigor, ethical awareness, and patient engagement in biomedical research. The approach integrates patient and healthcare professional perspectives across all research stages - from study design to result interpretation - supported by a structured training program for patient reviewers and a quality assurance workshops for researchers. It demonstrates how participatory, ethically sound research can be systematically implemented in funding schemes by showcasing NEURON's work. According to Open Science principles, all curricula and documents are openly accessible, offering a scalable best practice blueprint for other research funding formats worldwide.

While there are no more calls for proposals launched in the framework of ERA-Net NEURON, we are happy to share the launch of two calls by EP BrainHealth on January 8th. The first call, Call 1, focuses on multinational collaborative research addressing biological, social, and environmental factors that impact the trajectory of brain health across the lifespan in the field of neurological, mental, and sensory disorders. The second call, Call 2, supports similar multinational research in the field of neurodegenerative disorders. Both calls aim to fund ambitious, interdisciplinary, and translational research projects (more details here).

In this final year of ERA Net NEURON, we will continue to engage with the wider community through public oriented educational videos, by highlighting the achievements of NEURON and its funded projects, and by introducing new activities initiated under EP BrainHealth. We encourage you to stay connected by visiting our website, and following us on LinkedIn and X. This will ensure you stay connected with our community and receive timely information about upcoming calls, activities, and events. For access to our lay lecture series, videos featuring funded projects and more, be sure to subscribe to our YouTube channel. For readers interested in the newly launched successor initiative, EP BrainHealth, and the new activities and funding opportunities it offers, we encourage you to subscribe to the EP BrainHealth newsletter and to follow its social media channels (LinkedIn, Bluesky, X, YouTube).

We look forward to a productive and meaningful 2026 and thank you for being part of our community dedicated to advancing brain health, mental health, and neuroscience research.

Sincerely yours



NEURON Joint Transnational Call 2025:

"Interdisciplinary Approaches to the Neuroscience of Pain"

Chronic pain is a major global health challenge, affecting around 20% of people worldwide and significantly reducing quality of life, daily functioning, and societal wellbeing. While acute pain acts as a vital warning signal of injury or disease, chronic pain, which is defined as persistent or recurrent pain lasting longer than three months, often continues even after the original cause has resolved or cannot be identified. Despite its prevalence, the mechanisms that drive and maintain chronic pain remain poorly understood, and many individuals face delayed diagnoses and inadequate treatment. Because pain arises from complex interactions between biological, psychological, and social factors, interdisciplinary research is essential to improve our understanding of chronic pain and to develop more accurate diagnostic tools and effective, personalised treatment strategies.

Thirteen multinational research consortia were selected for funding under JTC2025 on the topic of Interdisciplinary Approaches to the Neuroscience of Pain. In total, 59 research groups from 15 NEURON partner countries collaborate within these consortia, addressing the complex biological, psychological, and social dimensions of chronic pain through diverse experimental, clinical, and translational methodologies. The total funding volume of the call amounts to approximately 16 M€.

In line with ERA-Net NEURON's continued commitment to meaningful patient involvement, all proposals underwent a thorough patient review during the evaluation process. Six international patient experts assessed the full proposals from the perspectives of patients and carers, offering written feedback to applicants and actively participating in discussions during the review panel meeting.

We extend our warmest congratulations to the funded consortia and wish them every success in their ambitious research endeavours. Their work has the potential to deepen our understanding of the intricate mechanisms underlying pain, advance innovative approaches to diagnosis and treatment, and ultimately improve the lives of individuals affected by chronic pain worldwide.

Stuart Brierley

CURE CVP

Collaborative Union of Research Expertise (CURE): Investigating the oxytocin receptor mechanism in chronic visceral pain (CVP) suppression

Project Coordinator:
Stuart Brierley, South Australian Health and Medical Research Institute (SAHMRI), Adelaide, Australia

Project Partners:
Nathalie Vergnolle, INSERM Toulouse, Toulouse, France

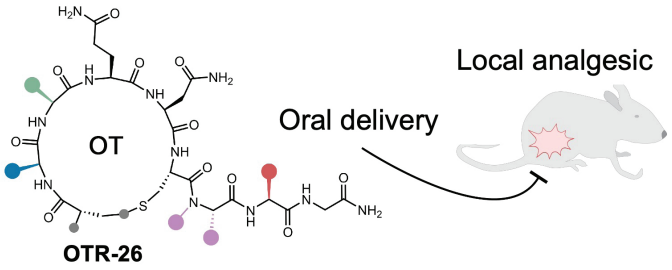
David Bulmer, University of Cambridge, Cambridge, UK

Nicolas Cenac, INSERM Toulouse, Toulouse, France

Markus Muttenthaler, The University of Queensland, Brisbane, Australia

Chronic visceral pain (CVP) is long-lasting pain from internal organs, which greatly reduces quality of life for millions of people worldwide. A major cause is Irritable Bowel Syndrome (IBS), which leads to gut pain, irregular bowel movements, and often anxiety, depression, and pain elsewhere in the body. IBS affects ~11% of people worldwide, yet effective treatments are limited.
We have developed a promising new drug, OTR-26, which targets a receptor linked to oxytocin, a natural hormone known for calming effects. Oxytocin itself breaks down quickly (~8 minutes), but OTR-26 lasts 24 hours and can be taken by mouth- a major advantage. In mice with IBS, a single oral dose of OTR-26 significantly reduced CVP.
This project aims to: 1) Test if regular oral doses of OTR-26 keep reducing CVP. 2) Understand how OTR-26 works to relieve CVP and improve gut health.
IBS symptoms stem from problems in the microbiome-gut-brain axis. OTR-26 may help restore these systems, easing CVP and related issues like anxiety and depression. To test this, we will use advanced human cell models, IBS patient samples, and specialized mice to study how OTR-26 affects gut bacteria, gut lining, and nerve signals between gut and brain. We'll also examine if OTR-26 reverses nervous system changes seen in IBS. If successful, this research could lead to a safe, effective, and easy-to-use treatment for IBS.

Gut-stable oxytocin analogue (OTR-26) for the oral treatment of chronic visceral pain in Irritable Bowel Syndrome



- Potent
- Selective
- Gut-stable
- Gut-specific
- Non-opioid-based
- Reverses IBS induced changes in the microbiome-gut brain axis

Franziska Denk

DECIPHER

Deciphering the role of CD90+ stromal cells in neuropathic pain

Project Coordinator:
Franziska Denk, Wolfson Sensory Pain and Regeneration Centre, King's College London, London, United Kingdom

Project Partners:
Nurcan Üçeyler, Department of Neurology, University Hospital Würzburg, Würzburg, Germany

Mateusz Kucharczyk, Łukasiewicz Research Network – PORT Polish Center for Technology Development, Wroclaw, Poland

Caroline Ospelt, Center of Experimental Rheumatology, University Hospital of Zurich, Zurich, Switzerland

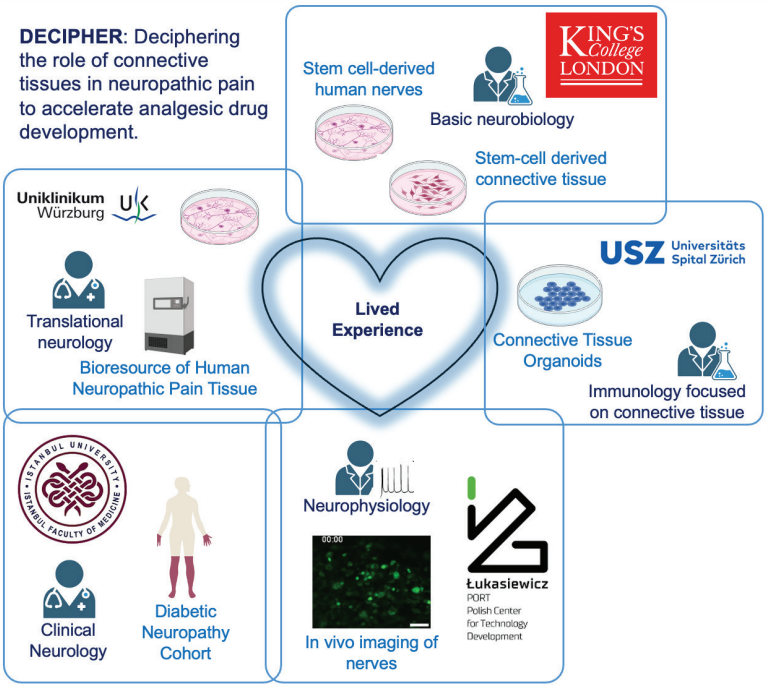
Fatma Yeşim Parman, Istanbul Faculty of Medicine, Istanbul University, İstanbul, Turkey

38 million adults in Europe live with chronic pain caused by nerve damage, called 'neuropathic pain'. Neuropathic pain can be caused, for example, by diabetes or cancer treatment. It feels quite different to other types of pain, causing shooting, stabbing and burning sensations that are very hard to bear. Worse still, painkillers do not work in neuropathic pain for most people. Our network is designed to change this, capitalising on recent research which suggests that neuropathic pain is caused by a particular type of connective tissue cell. We will now:

• test whether the number of these connective tissue cells is related to how much neuropathic pain someone experiences.

• find out whether these connective tissue cells can cause nerves to send abnormal electrical signals that drive pain.

• find new painkillers against neuropathic pain, by identifying ways to soothe and quieten these connective tissue cells.



Our research will use nerves and skin donated by people with neuropathic pain, as well as modern laboratory models, including human nerves derived from stem cells.

Our network includes specialists from many different disciplines and people who live with neuropathic pain themselves. By working together and taking into account everyone's perspective and expertise, we are in a perfect position to finally find better painkillers for the millions of people who live with neuropathic pain every day.

Luke Henderson

GENUS

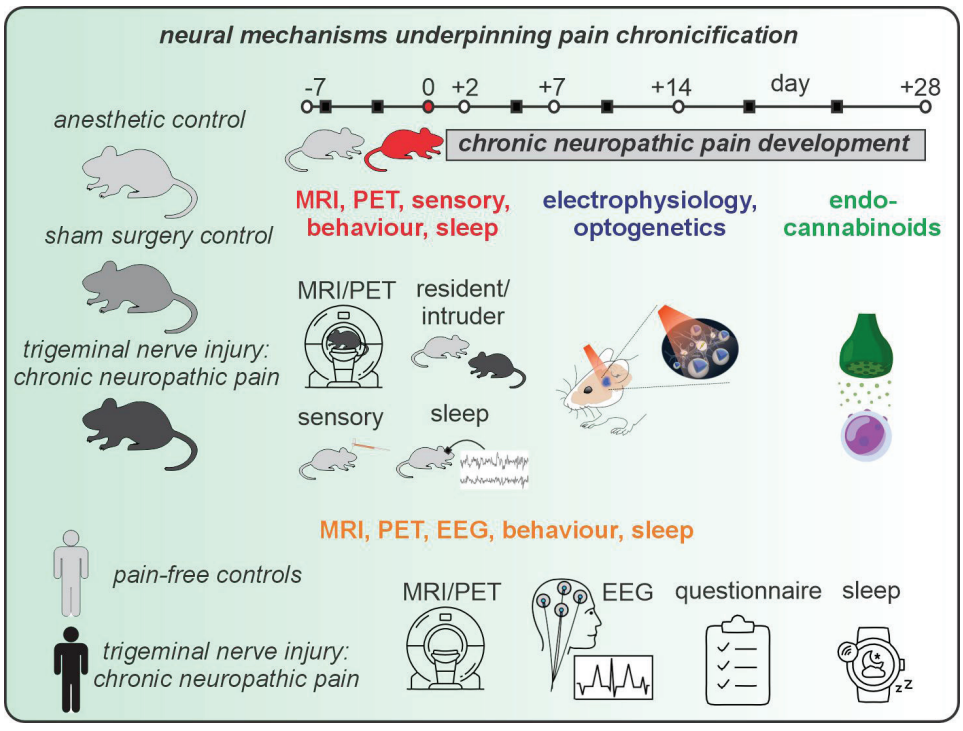
Translational Pain Discovery: BridGing MicE, Rat, aNd HUman Studies

Project Coordinator:
Luke Henderson, School of Medical Sciences (Neuroscience), University of Sydney, Sydney, Australia.

Project Partners:
David Hughes, School of Psychology and Neuroscience, University of Glasgow, Glasgow, UK.

Michelle Roche, Discipline of Physiology, University of Galway, Galway, Ireland.

Chronic pain is extremely difficult to treat, especially that which originates from damaged nerves. Chronic pain that develops in the orofacial region is particularly distressing and debilitating because of the critical roles of the face and mouth in activities such as chewing, swallowing and communication. The majority of individuals do not obtain adequate pain relief from any current treatments. A major roadblock in developing more effective treatments is our limited understanding of the brain changes responsible for chronic pain and in particular how such changes develop over time. This research proposal has four main goals: i) track changes in the brain, nerves, behaviour, and sleep as chronic orofacial nerve pain develops in rodent; ii) study how certain brain circuits respond to nerve injury and inflammation in a rodent model; iii) explore how the brain's natural cannabis-like system (the endocannabinoid system) changes during chronic pain; iv) examine similar changes in people with chronic facial nerve pain. This project brings together a team of experts from different fields to take a new, more complete approach to understanding the reasons why chronic facial pain develops. The goal is to find patterns and changes that are the same in both animals and people, which can help guide the development of new treatments.



Jannis Körner

H-Passion

Human Pain Atlas for Sensory Neurons: A Multimodal strategy of Investigation of Neuropathic Pain Mechanisms

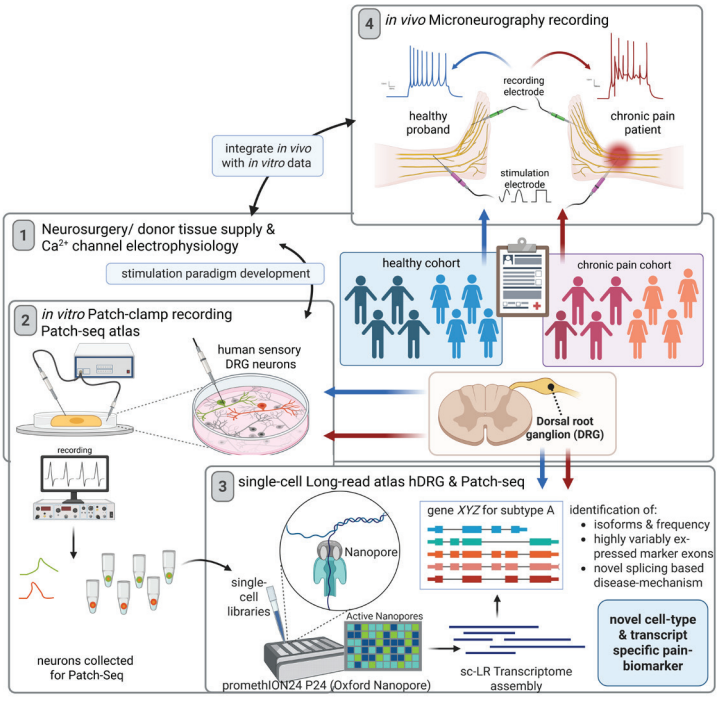
Project Coordinator:
Jannis Körner, Institute of Neurophysiology, Uniklinik RWTH Aachen University, Aachen, Germany

Project Partners:
Natja Haag, Center for Human Genetics and Genomic Medicine, Uniklinik RWTH Aachen University, Aachen, Germany

Emmanuel Bourinet, Institute for Functional Genomics, INSERM U1191, University of Montpellier, Montpellier, France

Jordi Serra, Department of Clinical Neurophysiology, King's College Hospital NHS, London, UK

Chronic pain affects millions of people and is often difficult to treat because we still don't fully understand how the nerves responsible for sensing pain actually work - especially in humans. Our project aims to change that by creating the most detailed map to date of the human pain system, focused on sensory neurons, the nerve cells that detect touch, temperature, and injury. What sets this project apart is our ability to study both how these neurons behave (function) and which genes they express (molecular identity)-all at the level of individual cells. This one-to-one link between a neuron's activity and its genetic code will allow us to uncover how certain nerve cells become overactive in chronic pain conditions. A special focus is on changes in gene processing called alternative splicing, which can subtly alter how proteins in individual cells work and may explain why some people develop pain while others do not. To add a real-time, human-in-the-loop perspective, we are also using a technique called microneurography, where we researchers insert a fine electrode into the nerves of volunteers and patients to record the activity of single nerve fibers directly in the body. This will help us link laboratory findings with the actual experience of pain in humans-something animal studies cannot provide. These unique recordings will guide how we interpret laboratory data and refine which types of neurons are most relevant to different types of pain. By combining live human nerve recordings, cell-by-cell genetic analysis, and tissue from both healthy and pain-affected donors, this project will build a first-of-its-kind atlas of human pain neurons. The insights gained will guide future treatments, moving us closer to personalized, effective therapies for people living with chronic pain.



Angelika Lampert

MiNE

Migraine and Endometriosis: Two Painful Conditions with a Hidden Link

Project Coordinator:
Angelika Lampert, Institute of Neurophysiology, Uniklinik RWTH Aachen, Aachen, Germany

Project Partners:
Xavier Moisset, Université Clermont Auvergne, Clermont Ferrand, France

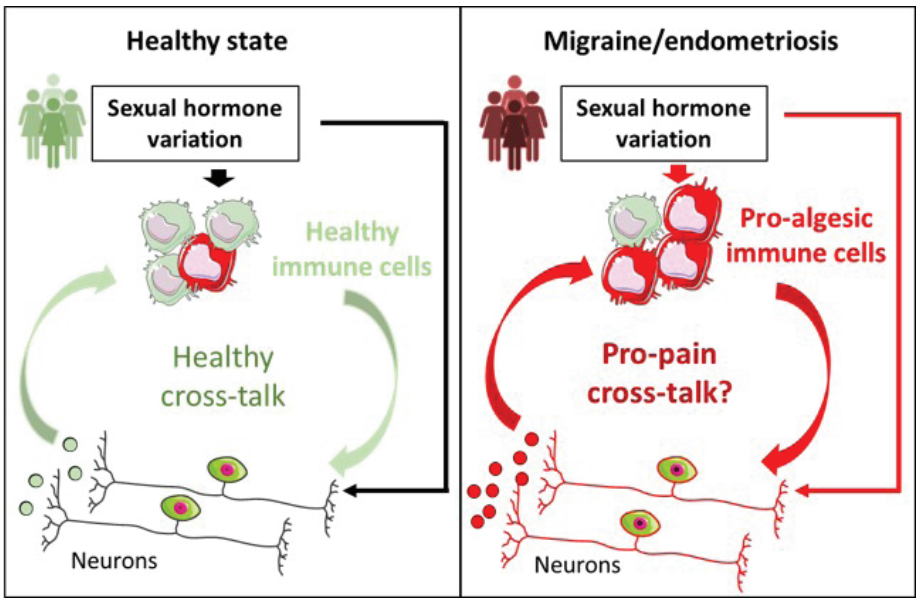
Franziska Denk, King's College London, School of Neuroscience, London, UK

Reza Sharif Naeini, McGill University, Faculty of Medicine, Quebec, Canada

Mario Coric, Department of Gynecologic Surgery and Urology, University Hospital Center Zagreb, Zagreb, Croatia

Dan Tudor Domocos, Faculty of Biology, University of Bucharest, Bucharest, Romania

Migraine and endometriosis are two common, debilitating conditions that cause chronic pain and disrupt daily life. Migraine affects far more women than men. Though different, both conditions often flare up during menstruation. Despite its prevalence, endometriosis remains underdiagnosed and under-researched. Emerging science suggests these diseases may share key biological mechanisms. Pain-related molecules, hormones, and immune cells appear to interact in similar ways. We believe that hormones act as a "bridge", influencing how immune cells communicate with nerve cells, ultimately driving pain in both conditions. However, the exact details of these interactions are still unclear. To uncover these connections, MiNE brings together an international team of experts in neurology, endometriosis, pain models, human sensory neurons, organ donor tissue, neuro-immune interactions, and statistical genomics. Our research focuses on three critical questions: How do hormones shape immune cell behavior? How do these immune cells, in turn, activate pain-sensing nerves? And how can we disrupt this harmful cycle to reduce pain?



Using human cells and models, our findings will pave the way for new treatments. Ultimately, MiNE aims to fast-track clinical trials, discover innovative pain therapies, and explore whether existing drugs could be repurposed. Our mission is clear: to improve the lives of millions of women living with these invisible, yet life-altering, conditions.

Abigail Livny-Ezer

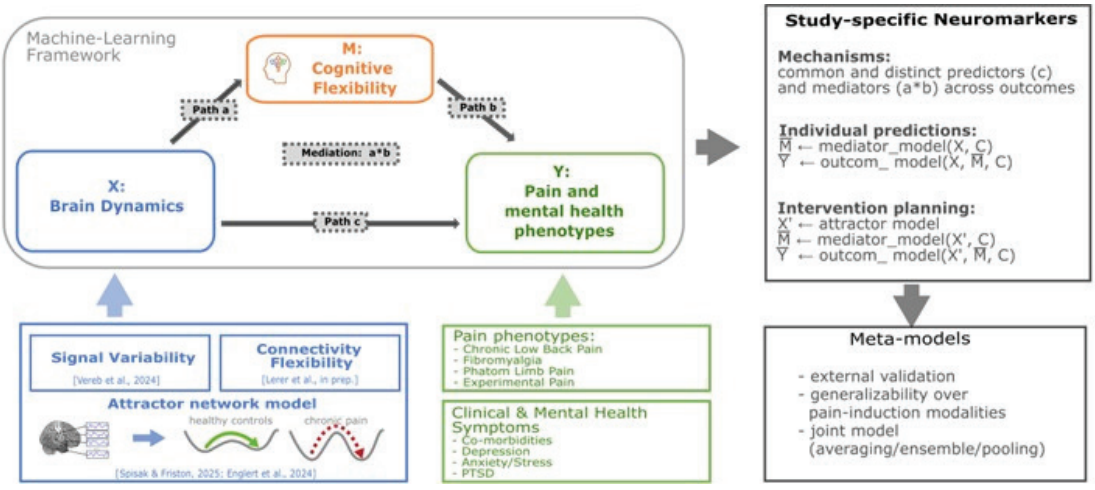
NEUROFLEX

The Flexible Mind: Brain Network and Cognitive Flexibility as Targets to Treat Chronic Pain

Project Coordinator:
Abigail Livny-Ezer, Sheba Medical Center, Tel-Hashomer, Israel

Project Partners:
Tamas Spisak, University Duisburg-Essen, Essen, Germany
Ulrike Bingel, University Duisburg-Essen, Essen, Germany
Zsigmond Tamas Kincses, University of Szeged, Szeged, Hungary
Daniel Vereb, University of Szeged, Szeged, Hungary

Chronic pain represents a challenge in medicine and science. Its variability makes it difficult to uncover the mechanisms that drive it or to develop effective strategies for prevention and treatment. Many chronic pain conditions involve persistent pain in the absence of tissue damage and often appear alongside mood and anxiety symptoms. The NEUROFLEX consortium is developing a computational approach that investigates how cognitive flexibility mediates and predicts the relationship between brain dynamics, pain and mental health outcomes across three chronic pain disorders: chronic low back pain, fibromyalgia, and phantom limb pain. Using resting-state functional MRI, we reconstruct the brain's large-scale "attractor landscape" - the substrate of its recurrent dynamics. From this, we extract brain features and feed them into a mediation-based machine learning framework designed to disentangle direct and indirect pathways through which brain dynamics, mediated by cognitive flexibility, influence clinical pain and mental health symptoms. Cognitive flexibility refers to neurocognitive processes that support adaptive responses to changing environmental demands, relying on flexible communication between large-scale brain networks. This adaptability may promote resilience against pain development and persistence and its associated emotional and functional consequences. We develop computational models within each pain group and validate them across disorders to identify both unique and shared neural signatures of chronic pain. This approach aims to support individual-level predictions and guide personalized treatment strategies.



Enrico Schulz
Veronica Meedt

NeuroPain

Precision Neuromodulation for Chronic Pain: Integrating Functional MRI and Focused Ultrasound for Personalised Treatment

Project Coordinator:
Enrico Schulz, Department of Radiology, LMU University Hospital, Munich, Germany

Veronica Meedt, Department of Radiology, LMU University Hospital, Munich, Germany

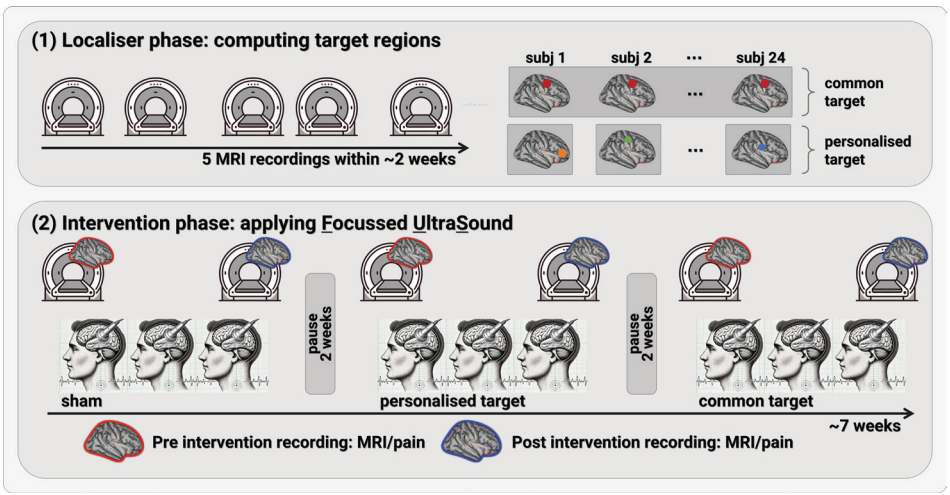
Project Partners:
Daniel Keeser, Department of Psychiatry, LMU University Hospital, Munich, Germany

Viktor Witkovský, Institute of Measurement Science, Slovak Academy of Sciences, Bratislava, Slovakia

Eleanor Martin, Dept of Med Phys & Biomedical Eng, University College London, London, UK

Charlotte Stagg, Nuffield eld Department of Clinical Neurosciences, University of Oxford, Oxford, UK

Chronic pain is a leading cause of disability worldwide, yet current treatments often provide only modest and short-lived relief, frequently accompanied by side effects. Chronic pain is also highly individual: different patients show distinct patterns of brain activity when their pain fluctuates, suggesting that a one-size-fits-all approach to neuromodulation is unlikely to work well for most people. This project aims to develop and test a personalised, non-invasive neuromodulation strategy for chronic low back pain using advanced MRI and focused ultrasound (FUS). Patients will undergo several functional MRI sessions while continuously rating the intensity of their natural, ongoing pain. These data will be used to identify the brain regions in each person where activity most closely tracks their own pain experience. In a subsequent intervention phase, FUS will be applied in three conditions: targeting each patient's personalised pain-encoding region, targeting a common group-level region, and a sham control. Pain ratings and MRI measures of brain activity and connectivity will be collected before and after each FUS intervention to assess both clinical and neurophysiological effects. This study will determine whether individually targeted FUS can provide greater pain relief than conventional group-based targeting. By linking individual pain signatures to tailored neuromodulation, the project aims to lay the groundwork for future clinical trials of safer, more effective, and truly personalised treatments for chronic pain.



Sebastian Wieland

PAINCODE

Unraveling the neural code of predictive dysfunction in chronic pain and anxiety in humans and mice

Project Coordinator:

Sebastian Wieland, Department for Functional Neuroanatomy, Department for General Internal Medicine and Psychosomatics, Heidelberg University Hospital, Heidelberg, Germany

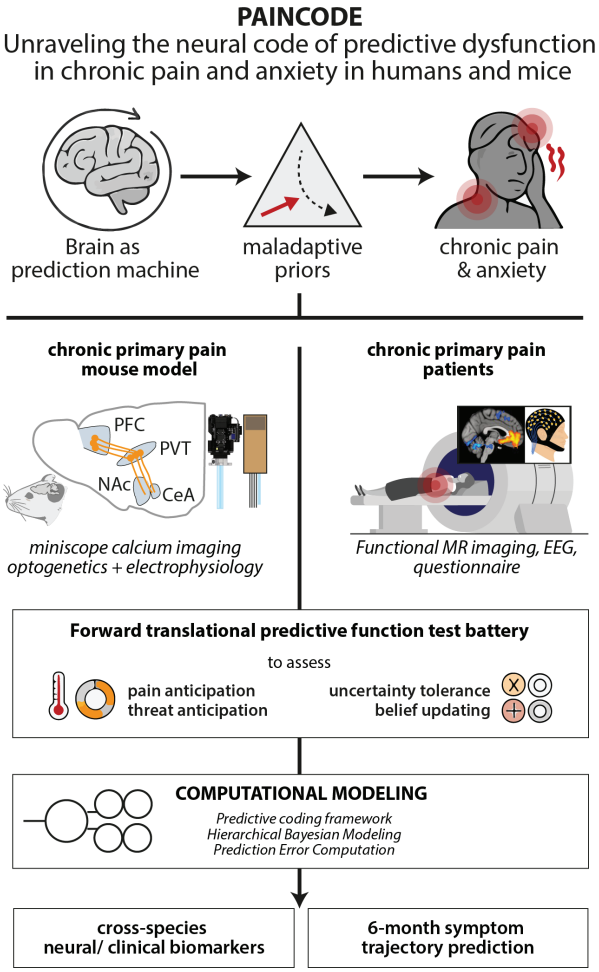
Project Partners:

Yann Quidé, The University of New South Wales, Sydney & Centre for Pain IMPACT, Neuroscience Research Australia (NeuRA), Randwick, Australia

Cyril Herry, Neurocenter Magendie, INSERM U1215, Bordeaux, France

Yael Bitterman, Faculty of Medicine, The Hebrew University of Jerusalem, Jerusalem, Israel

Chronic widespread pain and anxiety often co-occur and reinforce each other, especially in women, and commonly fail to respond to standard treatments. This highlights the need to understand how the brain maintains this persistent state. Growing evidence indicates that the brain operates as a prediction system, using past experiences to anticipate potential threats. In chronic pain, these predictions can become overly rigid and focused on danger, leading the brain to respond as if harm is present even when the body is safe. Such inaccurate predictions may help sustain both pain and anxiety.



PAINCODE aims to identify how these disrupted prediction processes arise and whether specific patterns of brain activity can indicate who will recover or who may need more personalized care. The project applies closely aligned tasks in humans and mice to probe predictive functioning across species. In people with chronic primary widespread pain we will measure brain activity using MRI, EEG, questionnaires, and computer tasks that test expectations of pain or threat, followed by a six-month reassessment. In mice, we will examine the same circuits in greater detail by recording from individual neurons and manipulating specific pathways.

By integrating data with artificial intelligence, PAINCODE seeks to reveal how the brain becomes "stuck" in a threat state and support more precise diagnostics and treatments for chronic pain and anxiety.

Michèle Hubli

PNS-SCI PAIN

The peripheral nervous system as a driver of neuropathic pain after spinal cord injury

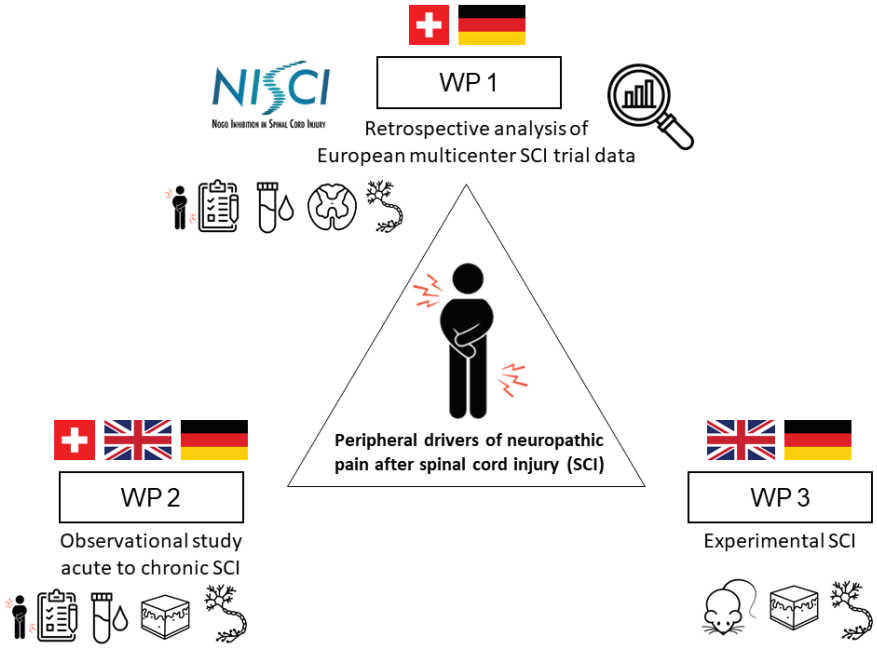
Project Coordinator:
Michèle Hubli, Spinal Cord Injury Center, Balgrist University Hospital, University of Zurich, Zurich, Switzerland

Project Partners:
Norbert Weidner, Spinal Cord Injury Center, Heidelberg University Hospital, University of Heidelberg, Heidelberg, Germany

Annina Schmid, Nuffield Department of Clinical Neurosciences, John Radcliffe Hospital, University of Oxford, Oxford, UK

Martin Schmelz, Department of Experimental Pain Research, Medical Faculty Mannheim, University of Heidelberg, Heidelberg, Germany

Neuropathic pain is a common and debilitating consequence of spinal cord injury (SCI), yet current treatment options provide only limited relief. This underscores the urgent need for a deeper understanding of the underlying mechanisms. PNS-SCI PAIN proposes a paradigm shift from a traditional CNS- towards a newer PNS-centric lens by investigating SCI-induced structural and functional changes in the PNS as key drivers of neuropathic pain. Our consortium integrates clinical and preclinical expertise across three countries into three complementary work packages: WP1 will leverage existing multicenter SCI trial data and analyze neurophysiological, neuroimaging, and protein biomarkers to identify PNS alterations linked to neuropathic pain. WP2 is a prospective, bicentric clinical study tracking 30 acute SCI patients over 6 months. Skin biopsies, neurophysiological testing (e.g. axon reflex flare), and protein biomarker profiling will be used to characterize the temporal dynamics of PNS changes and relate them to the development of neuropathic pain. Ultimately, WP3 will provide a reverse translation to preclinical rodent models and assess pain behavior, PNS histology, ex-vivo nociceptor excitability, and test whether altering promising protein candidates modulate dorsal root ganglion excitability. Through this integrative clinical and translational strategy, PNS-SCI PAIN aims to enhance early diagnostic capabilities, identify novel therapeutic targets within the PNS, and pave the way toward personalized pain management strategies for individuals with SCI.



Maria Maiarú

RESOLVE

Stem Cell-Derived Extracellular Vesicles as Novel Tools for Inflammatory Recalibration and Resolution of Chronic Pain

Project Coordinator:
Maria Maiarú, University of Reading, School of Pharmacy, Reading, UK

Project Partners:
Darius Widera, University of Reading, School of Pharmacy, Reading, UK

Graeme Cottrell, University of Reading, School of Pharmacy, Reading, UK

Fabian Szepanowski, University Medicine Essen, Department of Neurology, Essen, Germany

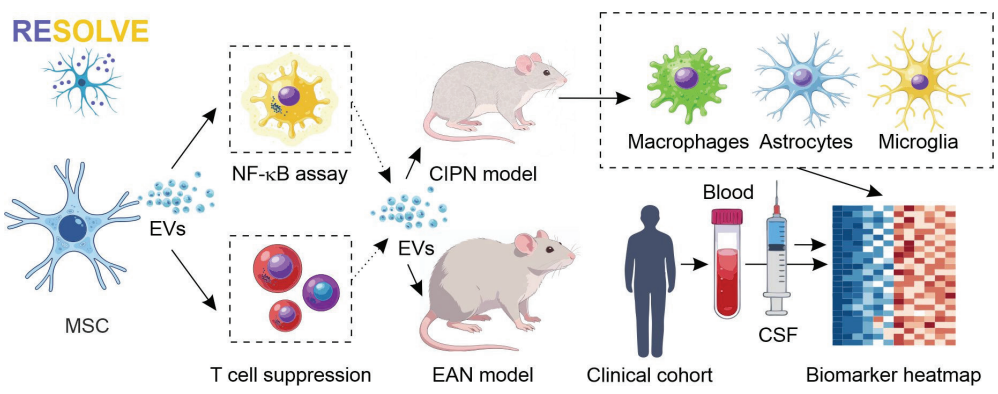
Bernd Giebel, University of Duisburg-Essen, Essen, Germany

Augustas Pivoriūnas, State Research Institute Centre for Innovative Medicine, Vilnius, Lithuania

Aurel Popa-Wagner, University of Medicine and Pharmacy Craiova, Craiova, Romania

Iva Sabolić, Labena d.o.o, Zagreb, Croatia

Chronic pain affects millions worldwide through ongoing inflammation in the nervous system. A key signaling switch called NF-κB plays a central role in starting and maintaining pain by triggering the release of inflammatory substances that increase pain sensitivity and activate immune responses. Simply blocking NF-κB can sometimes backfire and worsen inflammation, so more targeted approaches are needed. Tiny particles called extracellular vesicles (EVs), produced by stem cells called mesenchymal stromal cells (MSCs), offer a promising new treatment strategy. MSC-EVs can powerfully calm the immune system and reduce inflammation in various diseases, making them an excellent candidate for treating nerve pain. This project will test how well MSC-EVs relieve nerve pain and how they work. We believe MSC-EVs reduce pain by adjusting the activity of NF-κB and other inflammatory switches, reprogramming immune and nerve-supporting cells to break the harmful cycles that keep chronic pain going. We will test MSC-EVs from different sources using laboratory and animal models, combined with advanced testing to identify blood markers that show the treatment is working. To prepare for clinical use, we will also examine blood samples from 128 patients with immune-related nerve conditions to find similar markers. These could help identify which patients will respond to treatment in future clinical trials. Ultimately, this study will reveal how MSC-EVs work and identify their most important features: critical steps for developing new treatments for long-lasting nerve pain.



David Finn

Michelle Roche

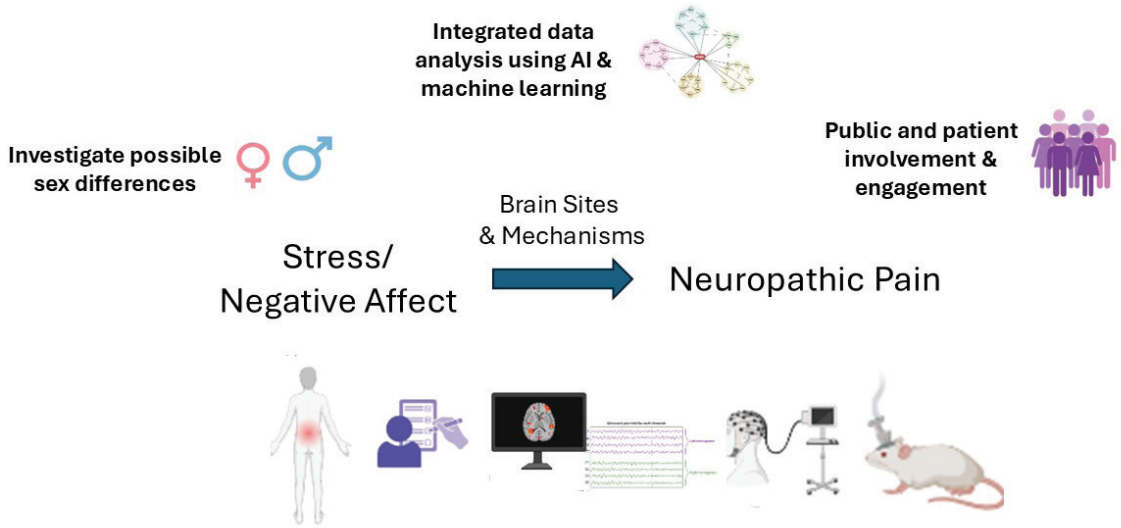
STRESSPAIN

Understanding how stress and negative affect exacerbate chronic neuropathic pain: Unlocking new therapeutic avenues

Project Coordinator:
David Finn and Michelle Roche, University of Galway, Galway City, Ireland

Project Partners:
Ipek Yalcin, CNRS and University of Strasbourg, Strasbourg, France
Kirsty Bannister, Imperial College London, London, UK
Frauke Nees, Ludwig-Maximilians-Universität München, Munich, Germany
Thomas Nevian, University of Bern, Bern, Switzerland
Matus Tomko, Slovak Academy of Sciences, Bratislava, Slovakia

Neuropathic pain is a type of nerve pain caused by a disease or injury to the nervous system. Chronic neuropathic pain affects 7-10% of the population, and over 50% of people living with it struggle with stress, anxiety and/or depression. We still do not fully understand how these emotional changes affect the severity of chronic neuropathic pain and/or prolong the pain state. Current treatments often provide only limited relief, especially for people who face both chronic neuropathic pain and emotional distress. To tackle this problem, this project aims to study how stress and negative emotions influence chronic neuropathic pain, using a combination of research in laboratory animals and humans – an approach called translational neuroscience. We will focus on specialised nerve pathways that travel from the brain to the spinal cord that are known to control pain. We have assembled a network of world-leading experts in the basic and clinical neuroscience of pain, stress, mood disorders, and in advanced data analytics, across 6 countries. We will work closely with patients throughout to ensure our research stays focused on what really matters: finding better answers and improving the lives of people living with chronic neuropathic pain.



Marc R. Suter

SUSEBERI

Microbiota and microglia interactions in neonatal/juvenile insults induced chronic pain: A translational approach

Project Coordinator:
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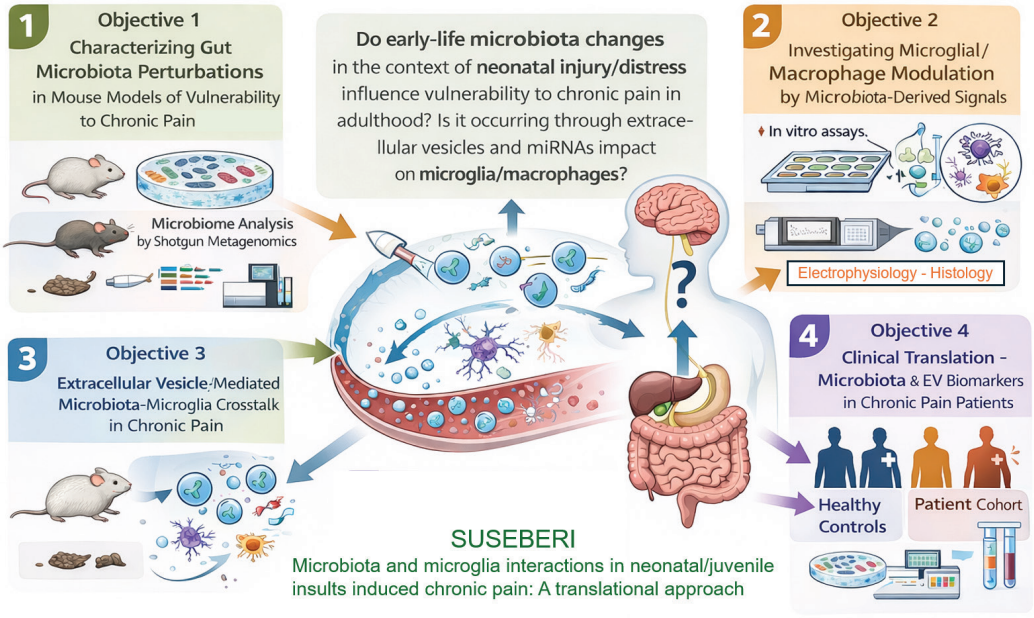
Chronic pain is a major health problem that can last for years and greatly reduce quality of life. Early-life events-such as stress, medical procedures, or injury-can increase the risk of developing long-lasting pain. At the same time, the gut microbiota, the vast community of microbes living in our intestines, is now known to play a key role in shaping the immune system and communicating with the brain. This project investigates whether an injury in early life can produce long-term changes in gut microbiota that make the body more sensitive to pain. Using established models in mice, we will examine how early injury, or stress alters gut microbial composition and function. By transferring microbiota from injured animals into germ-free mice, we will test whether these microbial changes alone are enough to increase pain sensitivity. We will also study how microbial signals-such as metabolites and tiny packages called extracellular vesicles-interact with nerve and immune cells involved in pain, and whether these effects differ between males and females. Finally, we will look for similar microbial patterns in people with chronic pain conditions such as fibromyalgia and hypermobile Ehlers-Danlos syndrome. Together, this work aims to reveal how early-life events shape the gut-immune-brain axis and to identify new microbial markers and treatment targets for chronic pain.



Isabelle Brunet

VASC-in-PAIN

Unraveling the Vascular Contribution to Chemotherapy-Induced Neuropathic Pain for Therapy

Project Coordinator:
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Many cancer patients treated with the chemotherapy drug oxaliplatin – commonly used for colon, gastric, and other cancers– experience a serious side effect called chemotherapy-induced peripheral neuropathy (CIPN). This condition causes long-lasting nerve damage, leading to pain, tingling, or numbness, usually in the hands and feet. More than 70% of patients develop these symptoms, which can be severe and greatly reduce quality of life. Unfortunately, there is no effective treatment for CIPN at the moment. Until now, most research has focused on how oxaliplatin harms nerve cells directly. However, emerging evidence indicates that blood vessels within nerves become dysfunctional during chemotherapy, leading to reduced blood flow and altered vascular properties. This disruption appears to play a key role in the development of chronic pain.

The VASC-in-PAIN project is a collaborative effort between four European research groups with complementary expertise, aimed at understanding how this vascular damage contributes to oxaliplatin-related nerve pain. Our team will utilize animal models and analysis of patient data to test whether vasodilators-drugs that improve blood flow– can reduce nerve pain without weakening the cancer-fighting effects of oxaliplatin. In addition, we will explore whether this approach could help with other chemotherapy drugs that cause similar nerve damage and pain. By targeting blood vessels, we aim to uncover new ways to prevent and treat CIPN using medications already available in the clinic, thus improving quality of life for many people undergoing cancer therapy.