NEUROSCIENCE RESEARCH DAY 2026

DISCOVERY AND NEW FRONTIERS

TBSI | KNOWLEDGE EXCHANGE & TERCENTENARY HALL

9th of June 2026

General information

To reduce the amount of waste generated during the event, **please remember to bring reusable cups and water bottles.** There is a water fountain as well as coffee and tea available.

Posters must be a maximum size of **A0 in portrait** orientation. Velcro tape, scissors, pins, and posterboards will be provided on the day. Please put your poster up on your assigned posterboard number. Posters should be mounted no later than 10:30 for the first poster viewing session.

A total of **six prizes of €100 each** will be awarded during the event, two oral prizes (session 2 and 4), and four poster prizes. These prizes are sponsored by Trinity College Institute of Neuroscience, and the Schools of Psychology, Medicine, Genetics & Microbiology, Biochemistry & Immunology, Pharmacy & Pharmaceutical Sciences and Department of Physiology.

The Inaugural Green Neuroscience Lab Prize

This year, we are delighted to introduce the inaugural **Green Neuroscience Lab Prize**, recognising outstanding commitment to environmental responsibility and sustainable practice within neuroscience research.
Modern scientific research carries significant environmental impact through energy consumption, laboratory waste, travel, and resource use. The Green Neuroscience Lab Prize has been established to celebrate laboratories and research teams that are actively working to reduce this footprint while fostering a culture of sustainability, innovation, and environmental awareness.
The award acknowledges initiatives such as reducing single-use plastics, improving recycling and waste management, promoting energy-efficient laboratory practices, encouraging sustainable procurement, supporting green travel policies, and embedding sustainability into everyday research culture.
By highlighting these efforts, we hope to encourage a shared commitment across the neuroscience community to more sustainable research practices and to recognise that scientific excellence and environmental stewardship can go hand in hand.
We warmly congratulate the recipient of the inaugural Green Neuroscience Lab Prize for their leadership, creativity, and dedication to building a more sustainable future for neuroscience research.

Members of the Neuroscience Research Day Working Group and Organising Committee: Marie Santillo, Áine Travers Dineen, Dara O'Boyle, Christopher Sheridan, Ruaidhri Gollogly Doyle, Iomar McCrea, Serena Pugliano, Anneliese Walsh, Antje De Boer, Brid Murphy, Kieran Byrne, Aline Zoller, Ronan Breathnach, Alejandro Lopez Valdes, Daniela Tropea, and Andrew Harkin

SPECIAL THANKS TO THE FOLLOWING SPONSORS:

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Neuroscience Research Day Morning Sessions

Tercentenary Hall

08:30 – 09:00	**Registration at the entrance of the Knowledge Exchange**
09:00 – 09:05	**Andrew Harkin** | Opening words
09:05 – 10:35	**Discovery and New Frontiers – Principal Investigators** (*Chairperson: Andrew Harkin*)
	9.05-9.20: Iracema Leroi - Interdisciplinarity to explore the Lewy body dementias: A micro-academic health sciences centre approach **9.20-9.35: Eric Downer -** Toll-like receptor signalling as a therapeutic target in multiple sclerosis **9.35-9.50: Mohammed Hankir -** Beyond Ozempic: Weight Loss without Aversion **9.50-10.05: Rob Whelan -** Neural mechanisms of methylphenidate response in young people with ADHD **10.05-10.20: Seán Froudist-Walsh -** Emergent specialisation of distributed networks in cortically-embedded RNNs with macroscopic gradients **10.20-10.35: Dominic Trepel -** From discovery to access: health economics tools for neuroscience innovation
10:35 – 11:00	**Coffee and poster session**

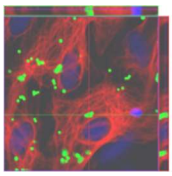
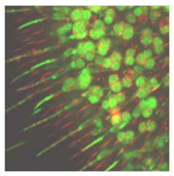
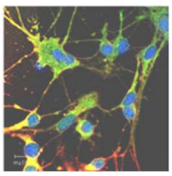
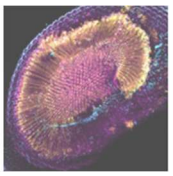
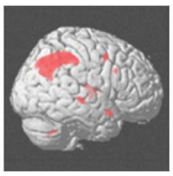
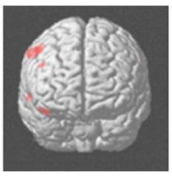
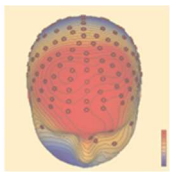
Tercentenary Hall

11:00 – 13:10	**Guest Lecture**
	11:00-11.30: Stefanie Höhl A Developmental Framework of Interpersonal Synchrony *(Chairperson: Rhodri Cusack)*
	Discovery and New Frontiers – Post-doc presentations *(Chairperson: David Loane)*
	11.40-11.50: Clinton Haarlem - Variation in critical flicker fusion: real-world implications
	11.50-12.00: Conor Thornberry - Examining spatial navigation & novel scene imagination in humans using optically pumped magnetoencephalography (OPM-MEG)
Poster viewing **Knowledge Exchange & The Bullnose**	**12.00-12.10: Eike Kofi Buabang** - A neural decoding approach to understand habit formation
	12.10-12.20: Céline Spriet - Acceleration of visual object categorization throughout development
	12.20-12.30: Gloria Vegliante - Use of a high-dimensional proteomic platform to understand secondary injury mechanisms underpinning preclinical traumatic brain injury (TBI) blood biomarker dynamics during brain injury and repair.
	12.30-12.40: Avril Watson - Retinal Barrier-on-a-Chip: Modelling Fibrovascular Remodelling in Neovascular AMD
	12.40-12.50: Rajiv Borah - Wireless electrical stimulation via silk-based conductive hydrogels to boost iPSC-derived astrocytes neuroprotection and guide macrophage polarisation in vitro for spinal cord repair
	12.50-13.00: **Dieter Waschbüsch** - Strategies to activate the LRRK2 counteracting Phosphatase PPM1H
	13.00-13.10: Natalie Hudson - Treatment of Non-Proliferative Diabetic Retinopathy with an oral medication
13:10 – 14:00	Lunch and poster session

Neuroscience Research Day Afternoon Sessions

Tercentenary Hall

14:00 – 15:30		**Guest Lecture** **14:00-14.30: Tsveton Serchov** Linking Brain Circuitry and Neural Plasticity in Antidepressant Response: The mPFC-Reuniens-Hippocampus Pathway *(Chairperson: Daniela Tropea)*
	Poster viewing **Knowledge Exchange & The Bullnose**	**Discovery and New Frontiers – GBHI Fellows** *(Chairperson: Alejandro Lopez-Valdes)* **14.40-14.50: Loretta Norton** - Understanding Cognitive Capacity and Lucidity in Severe Dementia: A Pilot Feasibility Study **14.50-15.00: Maria Kambongi** - Mobile EEG for Brain Health Equity: Expanding Epilepsy Diagnosis in Low-Resource Settings **15.00-15.10: Damián Dellavale** - Non-invasive Biophysical Neuromodulation To Reduce Functional Brain Aging **15.10-15.20: Thuan Anh Nguyen** - iCARE-VN: iSupport Co-created Assistance for dementia caREgivers in Viet Nam
15:10 – 15:40	Comfort Break/ Tea & Coffee in the Knowledge Exchange	
15:40 – 17:00		**Guest Lecture** **15:40-16.10: Silvia Di Angelantonio** The Developmental Roots of Neurodegeneration: Insights from Human Organoids *(Chairperson: Daniela Tropea)*
	Poster viewing **Knowledge Exchange & The Bullnose**	**Discovery and New Frontiers – PhD student presentations** *(Chairperson: Eric Downer)* **16.20-16.30: Marie Santillo** - The Origins of Memorability in Infancy **16.30-16.40: Ruaidhri Doyle -** Temperature Regulated Gene Expression and Alternative Splicing in Drosophila Melanogaster **16.40-16.50: Parnian Rafei -** Does Thinking About Goals Disrupt Habits? Evidence from a Behavioural Outcome-Devaluation Paradigm
16.50-17.00	**Rhodri Cusack** | Prize giving & closing words	
17:10 – 18:30	Nibbles in the Pav *Networking Opportunity*	

Detailed Programme and Abstracts

Guest Lecture 1

Location: Tercentenary Hall

Chairperson: Prof. Rhodri Cusack, Director TCIN, School of Psychology

11:00 – 11:30: Professor Stefanie Höhl, Professor of Developmental Psychology at the University of Vienna

Stefanie Höhl is Professor of Developmental Psychology at the University of Vienna where she directs the Wiener Kinderstudien lab. She received her PhD from the University of Leipzig. From 2016 to 2019 she led the Max Planck Research Group on Early Social Cognition at the Max Planck Institute for Human Cognitive and Brain Sciences in Leipzig. Her research lies at the intersection of developmental psychology and cognitive neuroscience and focuses on social and cognitive development in early childhood.

A Developmental Framework of Interpersonal Synchrony

Interpersonal neural synchrony, the temporal alignment of brain activities between individuals, has recently been proposed as a biomarker for successful communication and smooth social interaction. Surging empirical evidence shows that such synchrony emerges spontaneously between infants, children, and their caregivers from early on in development. Yet, little is known about the developmental preconditions and functions of interpersonal neural synchrony in childhood. In my talk I will present a developmental framework for understanding interpersonal neural synchrony, integrating insights from functional brain maturation and behavioral, social, and cognitive development. I will discuss how early caregiver-infant interactions, characterized by shared perceptual rhythms, facilitate the emergence of neural synchrony, as evidenced in our recent research identifying factors supporting caregiver-child neural synchrony, such as affectionate touch and vocal turn-taking. Given initial limitations in temporal precision of neural processing, early interpersonal neural synchrony is likely constrained to low-frequency brain rhythms and evolves alongside the maturation of neural networks and socio-cognitive abilities. I will outline how interpersonal neural synchrony may support critical developmental processes, including social learning, language acquisition, and attachment formation, through enabling mutual prediction and co-regulation between caregivers and children. This framework highlights the potential of interpersonal neural synchrony as both a marker and a driver of developmental change, offering new avenues for research and intervention.

Guest Lecture 2

Location: Tercentenary Hall

Chairperson: Prof. Daniela Tropea, Department of Psychiatry, School of Medicine

14:00 – 14:30: Dr Tsveton Serchov, Research Group Leader, Institute of Cellular and Integrative Neuroscience in Strasbourg

Tsveton Serchov is research group leader at the Institute of Cellular and Integrative Neuroscience (Strasbourg) and a tenured scientist with the French National Centre for Scientific Research (CNRS) in France. His research aims to better understand how sleep and brain health influence mood and depression. He completed his PhD at Ruhr University Bochum in Germany, studying how the body's internal clock controls behaviour. He developed new mouse models to investigate sleep, mood disorders, and antidepressant treatments. His work identified important brain proteins and signaling pathways involved in how antidepressants work particularly in fast-acting therapies linked to sleep and brain plasticity.

Linking Brain Circuitry and Neural Plasticity in Antidepressant Response: The mPFC-Reuniens-Hippocampus Pathway

The pathophysiology of depression disorder involves multiple biological processes, including circuit dysfunction and impaired neuroplasticity, yet an integrative view linking both processes remains elusive. Here, using circuit manipulation, electrophysiology, behavior, and fiber photometry, we identify a convergent circuit for antidepressant response and plasticity modulation. We demonstrate that chemogenetic activation of the prefrontal cortex (PFC) is sufficient to exert rapid antidepressant effects across multiple behavioral domains in a mouse model of stress-induced depression and exerts top-down control over hippocampal plasticity and processing. We identified the nucleus reuniens (RE) as a necessary mediator of these effects. Our findings demonstrate that the functional PFC → RE → hippocampus circuit plays a central role in the antidepressant response, linking circuit activity, hippocampal plasticity, and depressive-like behaviors.

Guest Lecture 3

Location: Tercentenary Hall

Chairperson: Prof. Daniela Tropea, Department of Psychiatry, School of Medicine

15:40 – 16:10: Professor Silvia Di Angelantonio, Center for Research in Neurobiology 'Daniel Bovet' (CRiN), Sapienza, Universita Di Roma.

Dr. Silvia Di Angelantonio is an Associate Professor of Physiology in the Department of Physiology and Pharmacology at Sapienza University of Rome. As a biophysicist, her research integrates electrophysiology, advanced imaging, and bio-fabrication to study the human nervous system. Her research is prominently focused on the generation and functional characterization of novel 2D and 3D human in vitro brain and retinal models. A central aspect of her work involves exploring the mechanisms of neuroinflammation in neurodevelopmental and neurodegenerative disorders, such as Alzheimer's disease and frontotemporal dementia. Her research investigates microglia-neuron crosstalk and the structural remodelling of the microglial cytoskeleton. By integrating microglial cells into her 3D in vitro models, she examines how microtubule dynamics influence disease progression in the brain and retina.

The Developmental Roots of Neurodegeneration: Insights from Human Organoids

Frontotemporal dementia (FTD) represents a spectrum of clinical syndromes characterized by progressive executive, behavioral, and language dysfunctions associated with the abnormal accumulation of the microtubule-associated protein tau in the brain. The intronic MAPT IVS10+16 mutation alters alternative splicing, disrupting the balanced 1:1 adult brain ratio of three-repeat (3R) and four-repeat (4R) tau isoforms. This molecular imbalance skews expression toward 4R tau isoforms, directly promoting pathogenic tau aggregation and neurofibrillary tangle (NFT) formation. To evaluate the developmental origins and functional consequences of this pathobiology, 2D and 3D human pluripotent stem cell (hPSC)-derived cortical and retinal organoid models were deployed.

Our findings demonstrate that the MAPT IVS10+16 mutation delays both cortical and retinal maturation while disruptively altering early neuroepithelial rosette organization. This genetic alteration stalls development at early polarization foci and significantly delays neuronal rosette lumen formation. During later stages of maturation, mutant organoids exhibit altered neuronal morphology (characterized by fragmented MAP2 and TUJ1 structure) along with profound defects in cortical glutamatergic, cortical GABAergic, and retinal synaptic maturation. This structural impairment correlates with severely compromised functional network activity, including diminished active neuron counts, disrupted calcium firing frequencies, and a loss of network synchronicity.

Mechanistically, this neurodevelopmental and degenerative phenotype is tightly linked to progressive mitochondrial dysfunction driven by soluble oligomeric tau with abnormal post-translational

modifications. This pathological axis causes mitochondrial fragmentation, increased reactive oxygen species (ROS), disrupted axonal transport, and a significant down-regulation in the expression of the master metabolic and mitochondrial biogenesis regulator PPARGC1A.

Remarkably, pharmacological intervention using bezafibrate (BZ), a drug that restores mitochondrial biogenesis via PGC-1α pathways, successfully rescues these deficits. Bezafibrate treatment effectively reverses early rosette patterning defects, lowers hyperphosphorylated and 4R tau expression levels, decreases neuronal fragmentation, and fully restores functional synaptic connectivity and neural network activity. Collectively, these organoid-derived insights expose a critical interplay between tauopathy, mitochondrial health, and early human neurodevelopment, introducing novel therapeutic avenues for mitigating tau-mediated synaptic failure.

Session 1: Discovery & New Frontiers - Principle Investigators

Location: Tercentenary Hall

Chair: Andrew Harkin, Professor in Pharmacology, School of Pharmacy and Pharmaceutical Sciences

9:05 – 9-20: Professor Iracema Leroi, Professor Geriatric Psychiatry, School of Medicine & Director Global Brain Health Institute (GBHI)

Interdisciplinarity to explore the Lewy body dementias: A micro-academic health sciences centre approach

Background: The Lewy body dementias (LBD), including Parkinson's dementia and Lewy body dementia, account for >20% of the nearly 65,000 people with dementia in Ireland; however, an estimated fewer than 5% of those affected receive a formal diagnosis. LBD is characterised by cognitive-behavioural and physical changes which significantly impact quality of life and care burden. Knowledge, awareness and support in Ireland for LBD is low.
Aim: The 4-year *EMERALD-Lewy* program (2024-2028) seeks to improve the diagnosis, care and understanding of the lived experience of people with LBD in Ireland.
Approach: EMERALD lewy is shaped by UK's 'Living Well with Dementia' framework, across 3 workstreams: (1) 'Diagnosing well', is scoping low diagnosis rates in Ireland, to achieve a 'good diagnosis' (timely/accurate/person-centered) and is investigating novel methods to improve diagnostic accuracy. (2) 'Living well' is exploring the meaning of 'quality of life' in LBD and its measurement, is investigating daily lived experiences in real-time using a smartphone app, and is developing and evaluating peer support; (3) 'Participating and translating knowledge well', is impacting health practice and policy in Ireland by enhancing public awareness and knowledge of LBD, and meaningfully including patient and public involvement throughout the program. To address these workstreams, we undertake an interdisciplinary approach using multiple methods including implementation science, observational cohorts, real-time data collection/ecological momentary assessment, health services research, quality of life evaluations, policy economic analysis, and knowledge translation.
Conclusion: The EMERALD Lewy program aims to improve diagnostic rates and quality of care for LBD in Ireland, aligning with existing policy and practice.

9:20 – 9:35: Eric J. Downer, Associate Professor, Discipline of Physiology, School of Medicine & Translational Neuroimmunology Research Group

Toll-like receptor signalling as a therapeutic target in multiple sclerosis

Toll-like receptors (TLRs) are the sensors of pathogen associated molecules that trigger tailored innate immune signalling responses. TLRs have become a focus in biomedical research given the role of this family of innate immune proteins in immune activation, autoimmunity and neuroinflammation. TLRs have been implicated in many diseases, including neurodegenerative diseases such as multiple sclerosis (MS). In this talk I will outline the translational research we undertake in MS cohorts to indicate that TLR dysregulation, and subsequent alterations in TLR-mediated inflammatory signalling, can contribute to MS pathogenesis. I will also present evidence that components of the plant Cannabis sativa L. can target TLR-mediated inflammatory events in MS. Such anti-inflammatory effects of cannabinoids represents a therapeutic avenue for further investigation in neurological disorders.

9:35 – 9:50: Mohammed Hankir, Assistant Professor, School of Biochemistry and Immunology

Beyond Ozempic: Weight Loss without Aversion

GLP-1 receptor agonists such as Ozempic have transformed obesity treatment and cause weight loss largely by suppressing appetite. A major undesired side effect of these drugs is nausea and vomiting, likely because they activate aversive neurons in the hindbrain area postrema. I will present preclinical and clinical evidence showing that the dual-specificity phosphodiesterase PDE10A is a promising target for new obesity drugs. Not only is PDE10A enriched in striatal medium spiny neurons to positively regulate hedonic feeding, but its expression in brown adipose tissue suggests that PDE10A negatively regulates energy expenditure. Thus, PDE10A inhibitors represent a dual-pronged approach to treating obesity, and a non-aversive alternative to GLP-1 receptor agonists.

9:50 – 10:05: Rob Whelan, Professor in Psychology, School of Psychology

Neural mechanisms of methylphenidate response in young people with ADHD

Background. Attention-Deficit/Hyperactivity Disorder (ADHD) is characterised by difficulties in executive functioning. Methylphenidate (MPH) improves cognitive symptoms. However, neural mechanisms of this improved cognition are poorly understood.
Methods. 128-channel EEG in 29 medication-naïve 10-17-year-olds with ADHD and to 27 typically developing controls. EEG data were collected pre- and post-medication initiation in the ADHD group (interval matched for controls). We calculated aperiodic parameters of resting EEG, a 3-stimulus oddball task assayed executive control and an n-back task was used to evaluate working memory.
Results. Young people with ADHD had significantly smaller aperiodic parameters vs. controls, but these parameters were not modified by MPH. In the oddball task, response variability improved under MPH; EEG intertrial coherence (ITC) improved for theta and delta frequencies. Change in ITC correlated with change in behavioral variability. Working memory significantly improved under MPH, with a significant increase in frontomidline theta power, but no change in alpha power.
Conclusion: Aperiodic EEG may reflect ADHD traits rather than stimulant effects. In the oddball task, improved response variability correlated with increased ITC, suggesting MPH augments neural synchrony. In the n-back, performance under MPH was associated with working-memory specific processes (increased theta) and not via suppression of task-irrelevant information.

10:05 – 10:20: Seán Froudist-Walsh, Research Assistant Professor, School of Computer Science & Statistics

Emergent specialisation of distributed networks in cortically-embedded RNNs with macroscopic gradients

Understanding how large-scale cortical organisation supports cognition remains a central challenge for neuroscience. Current recurrent neural network (RNN) models can capture complex neural dynamics during the performance of higher cognitive tasks. However, they largely overlook anatomy, limiting their comparability with experiments. Here, we introduce Cortically-Embedded RNNs (CERNNs), which embed artificial neural networks into a species-specific cortical space, enabling the incorporation of anatomical constraints and facilitating direct comparisons to cortex-wide dynamics during complex cognitive tasks. We trained CERNNs, with macaque or human cortical geometry to perform multiple visual and somatosensory cognitive tasks (e.g. working memory, response inhibition, decision-making), while penalising deviations from known anatomical principles. We found that by penalising a) long-distance connections and b) deviations from a macroscopic gradient of increasing excitation (dendritic spine count), we observed several salient features of brain organisation emerge. These include a) an exponential decay of connectivity weights with distance, b) sparser human vs macaque connectivity, c) propagation along sensory hierarchies, d) emergence of specialised cognitive networks These results suggest that distributed cognitive networks may arise naturally as the brain attempts to solve complex tasks under wiring constraints with systematic gradients of single neuron properties.

10:20 – 10:35: Dominic Trépel, Associate Professor (Health Economics), School of Medicine (Dept. Of Psychiatry) / Global Brain Health Institute

From discovery to access: health economics tools for neuroscience innovation

Health economics provides a toolkit for translating neuroscience discoveries into real-world patient and societal impact. Scientific promise is essential, but innovations must also be affordable, implementable and valuable to health systems, payers and policy-makers.

Using our forthcoming global study of value-based pricing for disease-modifying Alzheimer's therapies as a case study, this talk will introduce key tools from health economics, including economic modelling, cost-effectiveness analysis, value-based pricing and market-access planning. The aim is to show how early collaboration with health economics can help TCIN researchers design studies, strengthen grant applications and create clearer routes from neuroscience innovation to adoption, reimbursement and impact.

Session 2: Discovery and New Frontiers – Post-Doc presentations

Location: Tercentenary Hall

Chairperson: David Loane, Associate Professor in Neuroscience, School of Biochemistry and Immunology

11.40-11.50: Clinton Haarlem, TCIN (Redmond O'Connell Lab)

Variation in critical flicker fusion: real-world implications

The critical flicker fusion threshold (CFF) is a psychophysical paradigm used to assess the temporal resolution of the visual system. It employs a flickering light to quantify the highest frequency at which flicker can be perceived. However, it is unclear how CFF relates to visual perception in general. In psychological- and clinical research, it is often used as a measure for nervous system activation or as an indication of certain pathologies. However, the metric also varies naturally in the general population. In recent research, we have shown this variation to be quite considerable and relatively stable over time. Little is known about how this variation may affect perception or behaviour across individuals. Our current research is aimed at addressing these questions. We have conducted a large-scale study to examine how CFF varies across the animal kingdom and how this variation is associated with different ecological niches and foraging strategies. Additionally, using two different psychophysics paradigms, we have assessed how variation in CFF among humans may be linked with perceptual performance. This presentation will outline our findings and will provide insight into how CFF may relate to the perceptual abilities of individuals and how this may affect behaviour and ecological setting.

11.50-12.00: Conor Thornberry, School of Psychology (Rob Whelan Lab)

Examining spatial navigation & novel scene imagination in humans using optically pumped magnetoencephalography (OPM-MEG)

Spatial navigation deficits and hippocampal atrophy are among the earliest signs of Alzheimer's Disease (AD). However, the underlying neural mechanisms remain poorly understood. Theta oscillations (4-8 Hz), thought to be derived from the hippocampus, are known to support successful spatial learning and memory in humans. However, their precise role in navigation is unclear. This work uses Optically Pumped Magnetometer Magnetoencephalography (OPM-MEG), TCIN's new wearable brain-imaging technology, to capture theta dynamics during naturalistic spatial cognition. Healthy younger adults completed two OPM-MEG tasks: a scene imagination task, generating novel scenes from word cues and an active navigation task in a gamified virtual town requiring route planning and memory-driven navigation. Data were source-localised, with theta-band activity examined during both tasks. OPM-MEG captured task-modulated theta activity in both paradigms. Scene imagination elicited theta increases in hippocampal regions. We anticipate that active navigation will engage a partially overlapping network, with sustained theta in posterior hippocampal regions during route retrieval. These preliminary findings establish OPM-MEG as a feasible tool for studying hippocampal-dependent tasks and provide an analysis pipeline for our planned older-adult cohort. This will guide us towards developing a low-cost, gamified marker of early cognitive decline in AD.

12.00-12.10: Eike Kofi Buabang, School of Psychology (Clare Gillan Lab)

A neural decoding approach to understand habit formation

Goal-directed control allows us to flexibly adapt our actions to achieve desired outcomes, but with repetition behavior becomes habitual, driven by stimulus-response associations rather than current goals and beliefs. But what drives this shift? Outcome representations may weaken as predictable outcomes receive less attention, while response representations may strengthen with practice. I will present results from a study combining EEG with multivariate pattern analysis to decode neural outcome and response representations during habit formation. Participants (N = 76) were randomly assigned to moderate (1-day) or extensive (8-day) training on four stimulus-response-outcome associations. Following training, half the contingencies were reversed and behavior was assessed under time pressure. When goal-directed control was limited in this way, extensively trained participants were more likely to act according to original rather than reversed contingencies, confirming that extended practice promotes habit expression. Furthermore, across both groups, participants who naturally disengaged from outcomes during training — as reflected by poorer knowledge of which outcomes their actions produced — showed greater habitual responding, suggesting that habits form more readily when individuals do not attend to well-predicted outcomes. Understanding these individual differences may shed light on how maladaptive habits become entrenched in compulsive disorders.

12.10-12.20: Céline Spriet (with Dr. Liuba Papeo and Dr. Jean-Remy Hochmann, Cognitive Neuropsychology and Development team and the Baby Lab, Institute of Cognitive Sciences Marc Jeannerod, Lyon)

Acceleration of visual object categorization throughout development

One of the main functions of vision consists in extracting information to identify objects and assign them to meaningful categories. We investigate the development of this function in the first year of life. Previously, we had found that a main distinction guiding infants' looking behavior is that between animate and inanimate objects, found as early as 4 months of age. I will present a new dataset, in which we used frequency-tagging EEG to show that the animate-inanimate categorization is fast and automatic upon stimulus presentation, already at 4 months of age. We also show that infants' object perception is inherently different from adults' one, in terms of processing speed: processing visual objects is seven times slower in 4-month-olds than in adults, but accelerates greatly in the first year of life. I will discuss the cascading effects of slow information-processing on infants' perception and cognition.

12.20-12.30: Gloria Vegliante, School of Biochemistry & Immunology (David Loane Lab)

Use of a high-dimensional proteomic platform to understand secondary injury mechanisms underpinning preclinical traumatic brain injury (TBI) blood biomarker dynamics during brain injury and repair.

Aims: i) To investigate the trajectory of blood-based biomarkers in the acute phase after TBI (baseline, 4 hours, 3 and 7 days post-injury -dpi-); ii) To investigate the relationship between blood, CSF, and brain tissue changes in the acute phase post-injury; iii) To correlate acute blood biomarkers (7 dpi) to long-term neurobehavioural and neuroimaging assessments (up to 5 months).

Methods: Adult (n=77, 9 weeks old) and aged (n=25, 17 months old) mice (male/female: 58/44), were subjected to TBI or sham injury. Blood, CSF, and brain samples were analysed by the Alamar NULISA™ CNS Diseases panel 120 that enables ultra-sensitive, quantification of 120+ neuro-specific and inflammatory proteins. Blood-based biomarkers were correlated to longitudinal sensorimotor assessments (by SNAP), cognitive assessment (by Y-maze) and magnetic resonance imaging (T2w and DTI).

Results: Clinically predicted blood biomarker trajectories are mirrored in our murine TBI model (Gfap, Nfl), along with newly demonstrated TBI-specific biomarkers (Eno2, Vsnl1). Nfh has a sex-specific trajectory acutely after injury. Inflammatory and injury targets are upregulated by physiological ageing and exacerbated by TBI, with a trend toward increase in females. Blood-based biomarkers at 7 dpi have predictive value on neurobehavioral and neuroimaging assessments.

Conclusion: Proteomic approaches help to understand the mechanisms underpinning blood biomarker dynamics and their relevance to brain health and disease.

12.30-12.40: Avril Watson, School of Medicine (Sarah Doyle Lab)

Retinal Barrier-on-a-Chip: Modelling Fibrovascular Remodelling in Neovascular AMD

Fibrosis is a major cause of irreversible vision loss in neovascular age-related macular degeneration (nAMD), yet existing in vitro models lack the multicellular architecture required to study fibrovascular remodelling in the outer retina. This study aimed to develop a retinal barrier-on-a-chip model that recapitulates key features of the fibrotic retinal microenvironment. Human retinal microvascular endothelial cells, retinal pericytes, and choroidal fibroblasts were co-cultured within microfluidic chips to generate choroidal-like vasculature. Vessel integrity and extracellular matrix (ECM) remodelling were assessed using immunocytochemistry and perfusion assays following TGF-β and IFN-γ stimulation. The model formed functional vascular networks with VE-cadherin+ junctions and supported fibrovascular remodelling characterised by increased collagen-I deposition and altered expression of fibrosis-associated ECM proteins. Inflammatory stimulation with IFN-γ disrupted vascular integrity and enhanced multicellular remodelling within the chip. Together, these findings establish a physiologically relevant platform for investigating inflammatory and fibrotic mechanisms underlying retinal degeneration in nAMD.

12.40-12.50: Rajiv Borah, School of Engineering (Michael Monaghan Lab)

Wireless electrical stimulation via silk-based conductive hydrogels to boost iPSC-derived astrocytes neuroprotection and guide macrophage polarisation in vitro for spinal cord repair

Spinal cord injury (SCI) affects over 20 million people globally and more than 3 million in Europe, causing lifelong disability and annual costs exceeding $20 billion in Europe. Yet, no treatment offers full functional recovery. Electrical stimulation (ES) shows good promise, yet most existing systems are electrode-based and invasive for soft and vulnerable tissue such as the spinal cord. To address this, we have developed a minimally invasive conductive silk fibroin/PEDOT:PSS (SF/PEDOT) hydrogel recapitulating the mechanical and electrical properties of spinal cord, that can support capacitive coupling based power transfer wirelessly across dielectrics including cell culture dishes and tissues. Using this, we showed modulation of human blood-derived macrophages (hBDMs) toward a reparative phenotype, while downregulating pro-inflammatory markers even under inflammatory conditions. In parallel, hiPSC astrocytes encapsulated within SF/PEDOT hydrogels showed enhanced functional maturation under ES, evidenced by upregulated Cx43 gap junction protein expression. In an in vitro SCI-like model of reactive astrogliosis, WES via SF/PEDOT partially mitigated astrocytic reactivity. Together, these results provide the first evidence of the dual neuroprotective and immunomodulatory potential of a non-invasive, conductive hydrogel-based WES platform, validated using two human relevant cell types specific to SCI pathophysiology.

12.50-13.00: Dieter Waschbüsch, School of Biochemistry & Immunology (Amir Khan Lab)

Strategies to activate the LRRK2 counteracting Phosphatase PPM1H

Mutations in the Leucine Rich Repeat Kinase 2 (LRRK2) gene are the most common genetic risk factor for Parkinson's Disease (PD). Since these mutations lead to hyper phosphorylation of Rab GTPases, kinase inhibitors of LRRK2 are an important therapeutic strategy. As an alternative to reduce Rab phosphorylation, activator compounds that promote dephosphorylation could provide an alternative startegy. PPM1H phosphatase was shown to be specific for LRRK2-phosphorylated Rabs and antagonizes the cellular pathways associated with LRRK2 activity. Here we describe biochemical and kinetic studies of PPM1H toward possible development of activatos as an alternative to traditional kinase inhibitors. We find that the native enzyme is a dimer and that disrupting dimerization leads to a loss in activity. Structural analyses of PPM1H and a substrate mimicking phospho-Rab8A peptide indicate dramatic movements of a flap-domain that recognizes the peptide. Since the flap domain also enables PPM1H dimerization, these observations suggest a mechanism in which substrate binding at both active sites may allosterically enhance the catalytic rates. We also describe the development of biochemical assays to evaluate binding and catalysis toward the goal of high-throughput screening of small molecule modulators of PPM1H activity. These overall studies have the goal of understanding the allosteric mechanism of an unusual protein phosphatase, which could be exploited for novel therapeutics in future work.

13.00-13.10: Natalie Hudson, School of Genetics and Microbiology (Matthew Campbell Lab)

Treatment of Non-Proliferative Diabetic Retinopathy with an oral medication

Decreased connexin-43 (Cx43) expression is noted in subjects with diabetic eye disease, and alterations can lead to increased retinal permeability. We hypothesise that stabilising Cx43 using a novel orally available medication, danegaptide, could improve inner-blood retinal barrier integrity.

JR5558 mouse model develop spontaneous intra-retinal lesions, and were dosed via oral, systemic, or intravitreal routes with danegaptide at various doses and timepoints, and effect on lesions assessed using fundus fluorescein angiography. In vitro analysis using human retinal endothelial cells (HRMECs) were carried out to understand danegaptide mechanism in a VEGF-mediated manner.

All routes of danegaptide administration was efficacious in reducing intra-retinal lesion area. Selective Cx43 inhibition attenuated the effect of danegaptide on reducing vascular leakage. In vitro analysis found that danegaptide enhanced tight junction integrity in HRMECs in both the absence or presence of VEGF and can attenuate the VEGF-induced suppression of TNFSF15. Danegaptide when orally administered to NPDR subjects showed a statically significant reduction in macular edema.

Danegaptide was able to reduce enhanced vascular permeability in numerous rodent models of neovascular retinal disease and its effect requires the presence and activity of Cx43. Danegaptide action in reducing macular edema in NPDR/mild DME patients further suggests a novel mechanism which can be used as an earlier stage treatment.

Session 3: Discovery and New Frontiers – GBHI Atlantic Fellows

Location: Tercentenary Hall

Chairperson: Alejandro López-Valdés, Assistant Professor in Neural Engineering and Brain Health, School of Engineering / Global Brain Health Institute

14.40-14.50: Loretta Norton

Understanding Cognitive Capacity and Lucidity in Severe Dementia: A Pilot Feasibility Study

Background: As verbal communication becomes unreliable in severe dementia, detecting preserved cognition becomes increasingly difficult. Episodes of paradoxical lucidity suggest intermittent cognitive abilities may persist, yet current clinical care likely misses fluctuating awareness in those who cannot reliably communicate. Research in severely brain-injured patients has demonstrated that covert cognition can exist without purposeful behaviour, suggesting similar unrecognized capacities may exist in advanced dementia.
Method: We will examine stakeholder experiences and attitudes about cognitive capacity and lucidity in severe dementia through semi-structured interviews with clinicians, bedside staff, and family caregivers. Participants will also be asked about the acceptability and feasibility of advanced behavioural and neuroimaging techniques, developed for disorders of consciousness research, in long-term care settings. Expected Results: We will identify themes describing how stakeholders conceptualize cognition in severe dementia and their attitudes toward advanced assessments. Conclusions: This pilot will generate stakeholder perspectives to inform future large-scale studies on covert awareness in severe dementia, advancing scientific understanding and improving personalized care.

14.50-15.00: Maria Kambongi

Mobile EEG for Brain Health Equity: Expanding Epilepsy Diagnosis in Low-Resource Settings

Epilepsy affects over 50 million people worldwide, with the greatest burden in low- and middle-income countries (LMICs), where access to electroencephalography (EEG) remains extremely limited. In Namibia, a single stationary EEG system currently serves the public healthcare sector, creating major barriers to timely diagnosis and continuity of care.
This project aims to explore the potential of cap-based mobile EEG systems to improve equitable access to neurodiagnostic services in resource-limited settings. The work focuses on identifying clinically suitable mobile EEG technologies and evaluating their feasibility for decentralized implementation in Namibia, with particular attention to inclusivity for tightly curled African hair, affordability, portability, and workforce limitations.
Methods include a scoping review of mobile EEG technologies, development of a technology evaluation framework, and planned pilot validation within clinical neurophysiology settings in partnership with the University of Namibia and Windhoek Central Hospital.
The presentation will highlight early findings from the technology assessment process, implementation considerations for LMICs, and the broader implications for brain health equity, task-shifting, and decentralized epilepsy care.
This work demonstrates how resource-constrained settings can drive innovation in equitable neurodiagnostic care while contributing context-specific evidence to the future of global brain health technologies.

15.00-15.10: Damián Dellavale

Non-invasive Biophysical Neuromodulation To Reduce Functional Brain Aging

The human brain undergoes structural and functional changes with age. These changes are influenced by biological determinants, pathological processes, sociodemographic, and economic factors. The complex interaction among some of these factors can lead to frailty and cognitive decline. Given the challenging projections of dementia prevalence worldwide, there is a need to develop interventions that can effectively modify these adverse brain trajectories to maximize the delay of their impact on quality of life. Non-invasive biophysical neuromodulation (NIBN), such as transcranial direct current stimulation (tDCS), has shown promise in modulating brain activity. Recent advances in EEG-based brain clock models allow precise quantification of the Brain Age Gap (BAG) in humans, that is, the discrepancy between the chronological age and the predicted brain age. This project aims to investigate the tDCS effects on human BAG and on the neural network oscillatory and arrhythmic dynamics.

Our results offer preliminary evidence that anodal tDCS is associated with a BAG reduction, warranting further research to assess putative therapeutic potential of NIBN to delay accelerated brain aging trajectories. These findings address the need for scalable and cost-effective interventions to support brain health in marginalized populations.

15.10-15.20: Thuan Anh Nguyen

iCARE-VN: iSupport Co-created Assistance for dementia caREgivers in Viet Nam

Aim: To co-design and pilot-test iCARE-VN, a culturally tailored, digitally enabled, community-embedded intervention to improve wellbeing, caregiving skills, and self-efficacy among family caregivers of people with dementia in Vietnam.
Methods: Using a participatory co-creation approach, we will conduct two in-person workshops with family caregivers and community support workers (CSWs) from the Intergenerational Self-Help Clubs (ISHC) network (4 caregivers and 4 CSWs per workshop). Workshops will use nominal group technique and an expanded User-Task-Context framework incorporating eHealth literacy to identify priorities and co-design solutions. We will then pilot a structured training program with 10 CSWs to build capacity to introduce the iCARE-VN platform, support caregiver engagement, and guide personalised goal-setting. CSW confidence and readiness will be assessed post-training. Finally, a mixed-methods pilot with 24 caregivers and 8 CSWs will evaluate feasibility and acceptability using the Unified Theory of Acceptance and Use of Technology (UTAUT) and think-aloud interviews. Preliminary effectiveness will be explored through changes in caregiver wellbeing, caregiving management skills, and self-efficacy.
Conclusion: iCARE-VN will deliver an optimised, community-embedded digital intervention and generate critical feasibility evidence to inform a future large-scale trial, offering a scalable model for strengthening dementia caregiver support in Vietnam and other LMICs.

Session 4: Discovery and New Frontiers – PhD student presentations

Location: Tercentenary Hall

Chairperson: Eric Downer, Associate Professor, , Discipline of Physiology, School of Medicine

16.20-16.30: Marie Santillo, School of Psychology (Rhodri Cusack Lab)

The Origins of Memorability in Infancy

Some images are consistently more memorable than others, independent of context or prior experience, implying that memorability reflects shared properties of visual processing. While adults show reliable neural signatures of memorability in high-level visual cortex and medial temporal regions, it remains unknown whether this sensitivity depends on visual experience or emerges from the brain's early architecture.

Two-month-old infants (n=125) and adults (n=16) viewed short naturalistic videos during awake fMRI. Framewise memorability scores were predicted using ResMem, a deep neural network trained to predict human memorability. Both infants and adults exhibited activity in ventral visual and medial temporal regions in response to framewise memorability. ROI analyses confirmed the presence of adult-like activity of memorability-sensitive networks in infants. Infants additionally recruited medial and subcortical areas, including the amygdala and hippocampal body and tail, suggesting early involvement of memory-related structures. Memorability effects persisted after controlling for low-level visual features such as attentional saliency and visual entropy.

These findings indicate that neural sensitivity to memorability emerges within the first months of life, before extensive visual experience or conscious recall. Memorability appears to reflect an intrinsic organizational principle of the developing visual system, bridging perception and memory from the earliest stages.

16.30-16.40: Ruaidhri Doyle, School of Genetics and Microbiology (Mani Ramaswami Lab)

Temperature Regulated Gene Expression and Alternative Splicing in Drosophila Melanogaster

Animals developed many different mechanisms to overcome or adapt to environmental challenges. One such adversity are seasonal changes in temperature during winter time. In mammals it has been shown that already slight changes in temperature are enough to induce changes in alternative splicing and one particular alternatively spliced gene, the RNA binding protein RBM3, is an important protein involved in temperature dependent changes in hibernating mammals.

Using the model organism Drosophila melanogaster our primary aim is to understand temperature regulated responses in gene expression and alternative splicing, and how these might underly cold induced changes in certain biological functions, for example ovarian developmental arrest, called Diapause.

Throughout this project, we have employed various techniques including RNA-Seq to identify temperature dependent changes in differential expression and alternative splicing in Drosophila tissue culture, which has helped to identify potential important candidate genes. Additionally, we showed that Doa, a cold activated kinase is a regulator of Diapause. This is of particular interest as the mammalian homologue of Doa, Clk1 has been shown to be involved in cold dependent alternative splicing. We will show how cold induces alternative splicing and differential expression, and have started to identify underlying mechanisms.

16.40-16.50: Parnian Rafei, School of Psychology (Clare Gillan Lab)

Does Thinking About Goals Disrupt Habits? Evidence from a Behavioural Outcome-Devaluation Paradigm

This study tested whether goal simulation reduces habitual responding and enhances behavioural flexibility in an outcome-devaluation task. Participants were assigned to Goal Simulation or Control and completed 6 training sessions, followed by a habit test with varied preparation time. Linear mixed-effects models examined training RT, accuracy, and habit-test responses across preparation times.

During training, RTs decreased across sessions in Control, $\beta(SE)=-0.009(.0023)$, $p<.001$, indicating practice-related improvement. This improvement was attenuated in the Goal Simulation group, session×group $\beta(SE)=0.009(.0032)$, $p=.003$, while accuracy remained high across groups. In devalued trials, habitual responding increased with longer preparation time, $\beta(SE)=16.16(7.24)$, $p=.032$, with higher habitual responding in longer bins relative to the shortest, all $p<.02$. Withheld responses decreased at the longest preparation time, $\beta(SE)=-9.68(4.09)$, $p=.02$. In valued trials, correct responding was high, $\beta(SE)=71.13(6.64)$, $p<.001$, and did not vary by preparation time. Between-group analyses showed no overall effect of Goal Simulation on habitual responding, $\beta(SE)=1.73(5.29)$. However, Goal Simulation moderated withholding in devalued trials at the longest bin, $\beta(SE)=-8.97(4.18)$, $p=.033$.

Goal simulation did not broadly reduce habit expression, but selectively altered response regulation when greater preparation was possible, suggesting context-specific effects on behavioural flexibility.

Posters

Theme 1: Translational Research: Markers, Models and Interventions

1	Devin Seward	In Vitro and In Vivo Assessment of the Anti-inflammatory and Neuroprotective Properties of Formoterol-Loaded Polymeric Nanoparticles
2	Maitreyee Purnapatre	Acute alterations in Blood-brain barrier permeability following biomechanical insults of different severities
3	Emily Iliana Michail	Seed: spatial and temporal exploration of the evolution of brain changes in sepsis-induced cognitive dysfunction and neurodegeneration
4	Myles Corrigan	Beta$_2$-Adrenoceptor Agonist Formoterol Protects Against Deficits in Delayed Non-Match-to-Position Task Following Bilateral LPS Delivery Into the Locus Coeruleus in Rats -relevant in vitro models of inflammatory-driven neurodegeneration
5	Wandi Zhong	Inhibition of the kynurenine pathway protects against poly I:C-induced reactive microglial-associated reductions in the complexity of primary cortical neurons
6	Denis O'Brien	Psychotropic Medication Withdrawal: Characterisation of Antidepressant Dependence in Ex Vivo and In Vivo Models
7	Patricia Iusan	N,N-dimethyltryptamine (DMT) treated astrocytic conditioned media promotes neuronal outgrowth and complexity; suppressed by the viral mimetic Poly I:C
8	Christopher Sheridan	Psilocin attenuates poly I:C induced pro-inflammatory gene expression in human peripheral blood mononuclear cells
9	Taisiia Iurakova	Evaluating Novel Combinatorial Strategies for the Treatment of Glioma and Associated Epilepsy
10	Vincent Healy	Defining the Glioma Peritumoural Zone: An International Multi-Disciplinary Delphi Consensus establishing the Radiology–Pathology–Distance (RPD) Classification
11	Meghamsh Teja Konda	Systemic TNF-α-driven neuroinflammation and delirium vulnerability in ageing: recovery dynamics and the modulatory role of IL-17A
12	Dara O'Boyle	Natural Killer Cell and Macrophage Interactions in Age-Related Macular Degeneration
13	Kathryn Duff	Elucidating Pathological CSF1R Signalling to Inform Therapeutic Development in ALSP
14	Kieran Byrne	Investigating the interaction between Natural Killer and Endothelial Cells in Retinal Neovascular Diseases
15	Raea Michie	Microglial Heterogeneity Following Neonatal Hypoxic-Ischemic Injury Identified by scRNA-seq
16	Kieva Byrne	Investigating Immune Responses in Retinal Neovascular Disease
17	Bríd Murphy	The Integrated Stress Response as a Modulator of Endothelial and Immune Cells: Implications for Traumatic Brain Injury
18	Nila Padmajan Nair	Autophagy and Neuroinflammation interplay in TBI: Invitro modelling using IMG microglia.
19	Fiachra Comber	Fiji-Based Image Analysis for the Quantification of Microglial Responses in Traumatic Brain Injury and Neuropathology
20	Basma Baccouche	EHD4 regulates VEGF-driven permeability and inflammation
21	Avril Reddy	Inner blood-retina barrier dysfunction primes the retina for retinal pigment epithelium atrophy

22	Nathan Strogulski	Microglial NOX2 Couples Metabolic Remodeling to Neuroinflammatory Damage Post-TBI
23	Maria Capdevila Bayo	Extracellular Vesicles of Cerebrospinal fluid from patients with Alzheimer's Disease induce the pathology in wild type mice
24	Sarah Palko	VEGF/Ang2 Dual Inhibition Decreases Collagen Production and Alters Immune Activation in Neovascular Age Related Macular Degeneration
25	Ana Navarro Garcia	Improved oral absorption of pterostilbene via cocrystal engineering: implications for neuroprotection in temporal lobe epilepsy
26	Ximaine van der Burg	Impaired visually-evoked BOLD response in the APP23 mouse model of CAA: preliminary results
27	Zoe da Silva	Human Midbrain Organoids for Modeling α-Synuclein–Associated Mechanisms in Parkinson's Disease
28	Aikaterini Sfyaki	Angelman Syndrome: Therapeutic hijacking of UBE3A absence
29	Javier Sánchez Sánchez-Corral	Targeted Degradation of TDP-43: A PROTAC-Based Therapeutic Approach for ALS and Alzheimer's Disease

Theme 2: Clinical Neuroscience, Psychiatry and Aging

30	Nada Asar	A Computational Network Analysis of Neuropsychiatric Symptoms in Lewy Body Dementia: Implications for Caregiver Burden and Quality of Life
31	Nishka Mishra	Modifiable risk factors and cognitive function in Lewy Body Dementia: Cross-Sectional and Longitudinal Associations
32	Méabh McCrystal	Distinct Sensory-Cognitive and Neuropsychiatric Profiles of Hallucinations and Delusions in Lewy Body Disease
33	Komal Zade	When the background matters: reference lists selection impacts brain genome-wide association studies and exome enrichment analyses
34	Declan Quinn	Electroencephalographic Signatures of Methylphenidate in ADHD: A Systematic Review and 10 Recommendations for Future Research
35	Claudia Seiler	The acute effect of moderate-intensity continuous vs high-intensity 10x1 interval exercise on cerebrovascular blood velocity responses in type 2 diabetes
36	Alma O'Donnell	HFOs in Brain Tumour Related Epilepsy
37	Isabel Milano	Low-Density Sleep EEG Automatic Artifact Rejection: Stage-Specific Considerations
38	Dillon Palmer	Investigating the long-term effects of repetitive head injuries in sport on a proteomic level
39	Nathan Shields	The identification and validation of a biomarker for tinnitus: an objective data-driven personalized approach to diagnosis of chronic tinnitus
40	Sheen Chatta	From Old to the Oldest: Exploring the Needs and Experiences of Ageing Parental Caregivers
41	Ellen Moore	How Well Do Mental Health Dimensions Map onto Brain Organisation? A Precision fMRI Approach

Theme 3: Neural Systems, Development and Behaviour

42	Sarah Power	Time of day influences the brain's immune response to a systemic challenge
43	Prathiksha Math & Margarida Cohen Serra	Uncovering mechanisms of spatial navigation in humans using electroencephalography (EEG) and optically pumped magnetometer magnetoencephalography (OPM-MEG)

44	James Bradshaw	Exploring Neural Synchrony During Group Cognitive Stimulation Therapy in People with Intellectual Disability: Preliminary EEG Hyperscanning Findings
45	Muhammad Faizan	A Multiband EPI Reconstruction Pipeline for Foundcog k-Space Data
46	Bojana Bjegojevic	Two Sides of the Coin: Beta/Alpha and Engagement Index Capture Different Aspects of Attention
47	Jessica Kingston	Peripheral Immune Infiltration and Neuroinflammatory Reprogramming Following Neonatal Hypoxia
48	Renee Le	Active vs. Passive Touch: Neural Responses With and Without Vision
49	Henry Frost	Evaluating the electrophysiological signatures of infantile spasms in a model of neonatal hypoxia
50	Teo Fantacci	Mechanistic computational models of cognition
51	Sarah Desmond	Translating Neural Oscillations into Music: Sonifying EEG Brainwave Bands Through a Data-Driven Compositional Framework
52	Kate Connini	Assessing the impact of environmental enrichment on neuronal network activity
53	Patricia Sim	Mind the Gait
54	Delphine Guichard	Electrophysiological comparison of gamma oscillations in control mice and a mouse model of Rett Syndrome
55	Maria Berjano	Behavioral and fMRI modeling of decision-making under volatility in internalizing psychopathology
56	Chiara Rascona	Sleep-related neuronal network impairments associated with paroxysmal attacks in a mouse model of Alternating Hemiplegia of Childhood
57	Daniel McLoone	Normative Brain Development in Male and Female Rats: A Consensus Clustering Analysis
58	Andrew Breen	A Graph Theory Analysis of the Developing Adolescent Wistar Rat Brain
59	Ronan Murphy	Evaluating the spatial inflammatory profile of neonatal hypoxia and its contributions to neurological outcomes
	Theme 4: Global Brain Health (GBHI fellows) - Knowledge Exchange	
60	Cyprian Mostert	The impact of severe drought on brain health and labor market stability in South Africa
61	Colin Regan	Get Your Head in the Game – The Potential of Sport to Drive Brain Health Awareness
62	John-Paul Omuojine	Co-producing culturally adapted tools for rapid delirium detection in Ghana.
63	Rohith Khanna Deivasigamani	SENSE-Cog Tamil Nadu: Community-health-worker-led hearing and vision screening and support for older adults with cognitive impairment in rural Tamil Nadu: an open-label implementation field study
64	William Dean	Advocacy In Dementia - From Ambiguity To Action
65	Yuen Khuan Chan	When Creativity Meets Memory: an AI-guided methodology for dementia care facilitation
66	GBHI	10 years of Global Brain Health Institute