Symposiwm Niwrowyddoniaeth DU-Japan

Vale Resort
Pont-y-clun
31 Awst –
2 Medi 2023

UK-Japan Neuroscience Symposium

Vale Resort
Pontyclun
31 August –
2 September 2023

Welcome

Symposiwm Niwrowyddoniaeth DU-Japan
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UK-Japan Neuroscience Symposium
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Welcome to the 5th UK-Japan Neuroscience Symposium

As the organising committee, it is our sincere pleasure to welcome you to the 5th UK -Japan Neuroscience Symposium at the Vale Resort in Wales. We hope you will enjoy the talks on a wide range of translational and fundamental Neuroscience topics, featuring the latest results from leading labs in Japan and the UK. We also hope you will take the opportunity to network with one another during the informal meeting times scheduled into the program.

The aim of this symposium series is to disseminate knowledge and foster collaboration between our two country's Neuroscience communities in a convivial atmosphere that encourages interaction. We are particularly pleased to have so many ECRs present at the meeting who we hope will carry this partnership forward for many years to come. We should like to thank the MRC, AMED, The Welsh Government and the Guarantors of Brain for their support for the meeting and making this idea possible. Welcome to Wales or, as the Welsh say, Croeso i Gymru!

Akiko Hayashi-Takagi

Gaynor Smith

Jemeen Sreedharan

Kei Cho

Kevin Fox

Masahisa Katsuno

Tom Macpherson

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Program Day 1

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Day 1	Thursday 31st August
10:00-12:00	Registration (Spa Foyer)
12:00-13:00	Lunch (Castle Suite)
13:00-13:30	Remarks from Mr Kazuhiro KAWASE, Minister and Consul General, Embassy of Japan in the UK. Remarks from Dr Mark PALMER, Director of International Relations, MRC. Remarks from Dr Yasushi OGASAKA, Director of the Department of International Strategy, AMED. Housekeeping Information.
Session 1	Motor Neuron Disease/ Frontal Temporal Dementia (Castle Suite) - Chaired by Jemeen Sreedharan and Haruhisa Inoue
13:30-14:00	**Kotaro Oiwa**—Molecular basis underlying TDP-43 pathology in Amyotrophic Lateral Sclerosis.
14:00-14:30	**John Cooper Knock**—Single-cell integrated RNA-sequencing and ATAC-sequencing in human motor cortex identifies cell-specific genetic drivers of ALS.
14:30-15:00	**Haruhisa Inoue**—iPSC-based translational and reverse translational research for ALS Medicine.
15:00-15:30	Break and Refreshments (Castle Suite Bar)

Program Day 1 (Continued)

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Day 1 (Continued)	Thursday 31st August
15:30-16:00	**Bhuvaneish Thangaraj Selvaraj**—AMPA receptor dysfunction in amyotrophic lateral sclerosis .
16:00-16:30	**Yuka Koike**—DNA demethylation in the TDP-43 autoregulatory region links to aging.
16:30-17:00	**Jenna Gregory**—Using RNA aptamers to detect TDP-43 aggregation in ALS post-mortem tissue.
17:00-17:30	Break
Session 2	New Technologies (Castle Suite) - Chaired by Dezerae Cox and Toshihisa Ohtsuka
17:30-18:00	**Atsushi Kasai**—A hypothesis-free approach using Brain-wide neuronal activation mapping for deciphering complex brain functions.
18:00-18:30	**Omer Bayraktar**—Mapping the rules of glioblastoma using integrated single cell and spatial genomics.
18:30-19:00	**Akihiro Funamizu**—Machine learning approaches to understanding decision-making.
19:00-19:30	**Dezerae Cox**—Evaluating models of Motor Neuron Disease molecule by molecule.
19:30-20:00	**Masanori Matsuzaki**—Auditory mismatch negativity in the common marmoset revealed by calcium imaging.
20:00-20:30	Drinks Reception (Salamanza Bar)
20:30 Onwards	Dinner (Conservatory)

Program Day 2

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Day 2	Friday 1st September
07:00-09:00	Breakfast (Vale Bar and Grill)
08:45-09:00	Tea/Coffee Available (Castle Suite Bar)
Session 3	Neuropsychiatric disorder and computational Psychiatry (Castle Suite) - Chaired by Tomoyuki Furuyashiki and Matt Jones
09:00-09:30	**Hidehiko Takahashi**—Hypothesis testing and data-driven psychiatric research using artificial intelligence.
09:30-10:00	**Cezar Tigaret** —Psychiatric risk gene CACNA1C, synaptic plasticity and associative learning.
10:00-10:30	**Tomoyuki Furuyashiki**—Neuroimmune Mechanisms of Stress: Insights from a Mouse Model.
10:30-11:00	Break and Refreshments (Castle Suite Bar)
11:00-11:30	**Aya Ito-Ishida**—Dissecting the pathogenesis of Rett syndrome by multi-disciplinary approaches.
11:30-12:00	**Matt Jones**—Sleep neurophysiology as a metric of brain health.
12:00-13:00	Lunch (Castle Suite)
13:00-13:10	Symposium Photograph (Castle Suite)
13:30-16:45	Team Building at St Fagans Museum
17:00-17:45	Business Meeting (Plenary Meeting)
17:45-18:00	Break and Refreshments (Castle Suite Bar)
Session 4	Posters
18:00-19:00	Poster Blitz (2 Minute Talks in the Castle Suite) - Chaired by Kei Cho
19:00-19:30	Poster Session (Castle Suite Bar)
19:30 onwards	Buffet Dinner and Poster Session (Castle Suite)

Program Day 3

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Day 3	Saturday 2nd September
07:30-09:00	Breakfast (Vale Bar and Grill)
08:45-09:00	Tea/Coffee Available (Castle Suite Bar)
Session 5	Plenary Talks (Castle Suite) - Chaired by Kevin Fox
09:00-10:00	**Trevor Robbins**—Compulsivity: Neural basis and psychiatric implications.
10:00-11:00	**Haruhiko Bito**—Capturing and measuring circuit dynamics in active ensembles during cognition.
11:00-11:30	Break and Refreshments (Castle Suite Bar)
Session 6	Parkinson's and Alzheimer's Disease (Castle Suite) - Chaired by Gaynor Smith and Masahisa Katsuno/Taku Hatano
11:30-12:00	**Taku Hatano**—α-Synuclein Aggregation and Propagation.
12:00-12:30	**Kathryn Bowles**—Using iPSC-organoids to model tauopathy.
12:30-13:00	**Masami Masuda-Suzukake**—A mouse model of tau propagation using synthetic tau fibrils.
13:00-13:30	**Wendy Noble**—P2X7R influences tau aggregate burden in human tauopathies and shows distinct signalling in microglia and astrocytes.
13:30-14:00	Lunch (Castle Suite)

Program Day 3 (Continued)

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Day 3 (Continued)	Saturday 2nd September
Session 7	Circuits and Behaviour (Castle Suite) - Chaired by Tom Macpherson and Kei Cho
14:00-14:30	**Tom Macpherson**—Striatal control of cue-guided decision-making and its impairment in Schizophrenia.
14:30-15:00	**Makiko Yamada**—Positive illusions: integration from molecules to functional brain networks.
15:00-15:30	**Verity Brown**—Cognitive flexibility requires more than the prefrontal cortex.
15:30-16:00	Break and Refreshments (Castle Suite Bar)
16:00-16:30	**Masako Tamaki**—Roles of NREM and REM sleep in learning and memory in humans.
16:30-17:00	**Abidemi Otaiku**—Do distressing dreams cause neurodegenerative diseases?
17:00-17:30	**Steve McHugh**—Hippocampal circuit mechanisms of memory consolidation.
17:30-18:00	Break and Refreshments (Castle Suite Bar)
Session 8	2022 Poster Prize Winners (Castle Suite) - Chaired by Akiko
18:00-18:30	**Scott Mitchell**—Hypomethylated FUS induced synaptic pathophysiology requires irreversible liquid-liquid phase separation and dendritic localisation of FUS condensates.
18:30-19:00	**Mari Shiozaki**—The Role of Cbln1 in Age-Related Hearing Loss: Insights from Cbln1 Knockout Mice.
19:05-19:30	Champagne Reception (Salamanza Bar)
19:30 Onwards	Banquet Dinner (Conservatory)

Speakers

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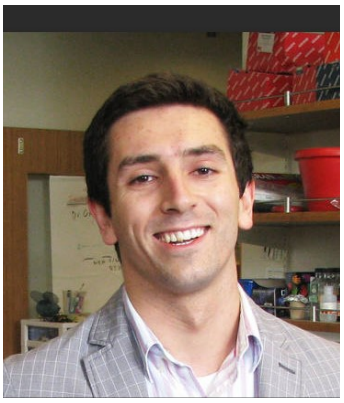
Speakers

Information for speakers

- In giving your presentation, please keep your talk to a maximum of 20-22 minutes and expect about 5 minutes for questions afterwards. This will allow some time for a brief introduction by the chairs and switchover between speakers. For plenary speakers please aim for a 50-minute presentation.

- You will be able to plug laptops directly into the HDMI cable at the lectern, so please remember to bring an adapter if you need one. Alternatively, you can bring the slides on a memory stick to be uploaded into the conference computer. Slide upload should be done in advance of the session during one of the breaks.



Omer Bayraktar

Group Leader

Wellcome Sanger Institute, UK

omer.bayraktar@sanger.ac.uk

Mapping the rules of glioblastoma using integrated single cell and spatial genomics

The malignant brain tumour glioblastoma multiforme (GBM) displays significant levels of cellular plasticity. It is often assumed that the tumour microenvironment (TME) regulates GBM cell states, but we know little about the spatial organisation of GBM tumours and TME-derived signals driving malignant cell state transitions. Here, I will present an integrated single cell and spatial multi-omic approach to resolve the tissue architecture of GBM. First, I will present cell2location, a new computational tool that can map fine-grained cell types in spatial transcriptomics data. Second, I will present GBM-space, a new collaborative effort to discover TME-GBM interactions using multi-modal genomics. Using joint single cell transcriptomic and epigenomic profiling, we define recurrent malignant cell states and expand the description of the TME cells. Using Visium spatial RNA-sequencing, we profile distinct tumour sites and observe significant regional heterogeneity of the GBM microenvironment. Finally, we integrate single cell and spatial transcriptomics using cell2location and discover that malignant cell states regionally segregate in GBM and associate with distinct TMEs. Our efforts reveal the novel spatial organisation of GBM tumours and identify putative TME signals that regulate malignant cell states.

Haruhiko Bito

Professor

University of Tokyo

hbito@m.u-tokyo.ac.jp

Capturing and measuring circuit dynamics in active ensembles during cognition

Two separable yet interactive cellular activity features have been associated with neuronal populations implicated in memory formation. The first feature concerns the induction of synaptic plasticity at synapses that receive characteristic stimuli to trigger postsynaptic NMDA receptor signaling during such cognitive events. The second relates to activity-dependent gene expression via a synapse-to-nucleus signaling mediated mainly by a transcription factor CREB. Synaptic inputs locally trigger neuroplastic signaling at the stimulated synapses while also turning on activity-dependent mechanisms linking synapses and the nucleus, which control long-term memory and late-phase long-term plasticity. By deciphering the bidirectional signaling between synapses and the nucleus, we have elucidated an inverse synaptic tagging mechanism that facilitates long-term maintenance of strong-to-weak synaptic weights ratio based on regulating an activity-regulated CREB target immediate early gene Arc. Human genome analyses have recently revealed numerous disease-linked mutations in the Ca2+-CaMK-CREB-Arc pathway associated with neuropsychiatric disorders. Thus, we sought to create a technology to record the neural activity related to memory processing in vivo and decipher the biochemical basis of the dysfunctional signaling in neuropsychiatric disorders. To do so, we thoroughly used the structural module information of genomes and proteins shown to contribute to neuronal plasticity. We successfully designed a synthetic promoter E-SARE that labels activated neuronal populations with an unsurpassed dynamic range and developed R-CaMP2, G-CaMP9a, and XCaMP, which are next-generation linearized Ca2+ indicators capable of recording fast action potential dynamics in multiple neuronal cell populations. These innovations in the precise measurement and labeling of dynamic neural circuits have begun to uncover interesting rules governing neural information processing during learning and pave the way toward a better understanding of the neuropathological significance of disrupting learning-associated signaling in disorders such as schizophrenia, autism, and intellectual disability.

Kathryn Bowles

Group Leader

University of Edinburgh

kbowles@exseed.ed.ac.uk

Using iPSC-organoids to model tauopathy

Human iPSC models are becoming increasingly popular for modeling human disease, but there is still hesitation surrounding their use for studying age-related neurodegenerative disorders. The advent of 3D organoids has provided a more complex platform for human brain modelling, but they have a reputation for high variability, lack of reproducibility and low replicability, especially when used to study mild phenotypic changes. However, we have developed a new, highly reproducible and consistent protocol for forebrain organoid differentiation that overcomes some of these obstacles. We have applied this model to the transcriptomic and functional study of the impact of autosomal dominant MAPT mutations, which cause frontotemporal dementia. We show that not only do these organoids reproduce key disease phenotypes, as found in human brain, but also reveal early cellular changes that highlight novel disease mechanisms that would not be possible to detect from postmortem tissue. We anticipate these models will continue to increase in their complexity and representation of the human brain, and will be highly valuable to the study of human neurodegenerative diseases.

Verity Brown

Professor

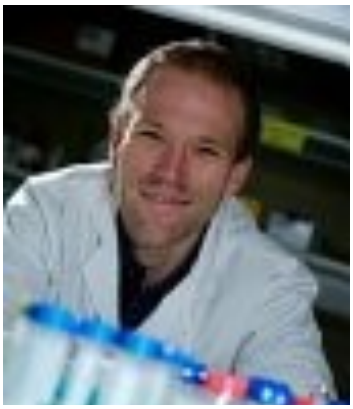
University of St Andrews

vjb@st-andrews.ac.uk

Cognitive flexibility requires more than the prefrontal cortex

A reliable double dissociation, which has been replicated many times and reported in many species, has implicated the rat/mouse medial prefrontal and primate dorsolateral prefrontal cortex in shifting attention when rules change (an “attentional shift”), while the orbital frontal cortex is implicated in reversal learning when contingencies change (an “affective shift”). The most convincing demonstration that this is a double dissociation is where a simple learning task is used that challenges distinct cognitive functions at different points in the task, so that ‘loss of function’ is assessed in the context of cognitive function that is preserved.

In this presentation, I will describe the intradimensional-extradimensional set-shifting task and show how it has been used to demonstrate the double dissociation that characterizes these two regions. Then I will show how small changes to the task can be used to elicit additional information about other functions that are lost, or preserved, following inactivation of the medial and orbital prefrontal cortex that are otherwise masked in the basic task. I will also show data from manipulations of subcortical areas that form part of the mPFC and OFC circuits and how they are involved in behavioural flexibility, and the cognitive processes that mediate it, that seems to require a functional prefrontal cortex. A nuanced understanding of these lost or retained functions can inform us on how best to further explore executive function, not only in rodents, but also with implications for treatment of diseases and disorders in human patients.



Johnathan Cooper-Knock

Principal Investigator

University of Sheffield

j.cooper-knock@sheffield.ac.uk

Single-cell integrated RNA-sequencing and ATAC-sequencing in human motor cortex identifies cell-specific genetic drivers of ALS

Amyotrophic lateral sclerosis (ALS) is an archetypal complex disease with a polygenic architecture. Despite high heritability, the latest ALS genome-wide association study (GWAS) identified a genetic risk factor in <10% of patients. Moreover, where the field has identified genomic regions containing disease-associated genetic variants, we often lack a biological understanding of the effect of the genetic variation, which is a necessary requirement to allow development of therapeutic interventions. We previously applied ATAC-seq to profile chromatin accessibility within normal motor neurons (MNs). By focusing on regulatory regions which are open and active within MNs, our machine learning method reduced the search space by >90% and uncovered 690 ALS risk genes, corresponding to 35% of SNP-based heritability. Here we have applied single-cell integrated RNA-sequencing and ATAC-sequencing to nuclei extracted from human motor cortex from ALS patients and controls. We have discovered cell-specific risk genes across neuronal subtypes and glia thereby increasing further the proportion of explained SNP-based heritability. Trajectory analysis of gene expression within microglia and astrocytes provides a data-driven method for discovering physiological glial phenotypes in vivo. Using this analysis we have identified ALS-associated glial expression of known ALS risk genes and provided previously missing functional annotations for known ALS GWAS loci.

Dezerae Cox

Research Fellow

University of Cambridge

dc597@cam.ac.uk

Evaluating models of Motor Neuron Disease molecule by molecule.

ınappropriate protein aggregation is a central molecular signature of many neurodegenerative diseases, including Alzheimer's and Motor Neuron (MND) diseases. However, decades of research on protein aggregates have not yet isolated the cause-effect relationship and overall role of aggregates in disease pathology and progression. This is in part due to the low abundance and high heterogeneity of aggregate species which has made their quantitation and study challenging. As a consequence, it remains unclear how well current experimental models of MND recapitulate true disease phenomena. Here, we report a novel single-molecule immunoassay for sensitive and specific detection of small soluble oligomeric aggregates. Using the MND-associated aggregation-prone protein TAR DNA-binding protein 43 (TDP-43), we show this assay can detect and characterise aggregate particles ranging from recombinant TDP-43 to complex whole-proteome mixtures. We demonstrate the application of this assay to comparing patient-derived tissue extracts and induced pluripotent stem cell models. Surprisingly, we did not observe consistent differences in the number of aggregate particles extracted from disease-derived tissues compared with age-matched controls. However, we find differences in the physicochemical properties of these aggregates. This knowledge contributes to a growing body of research seeking the fundamental biology underpinning MND and may improve our understanding of the relationship between protein aggregation and disease progression to inform future early diagnosis efforts. In addition, we hope providing a tool for quantitative evaluation of existing models will empower researchers to select the most appropriate systems when investigating aggregate pathology, paving the way for more robust early-stage therapeutic research.

Akihiro Funamizu

Lecturer

University of Tokyo

funamizu@iqb.u-tokyo.ac.jp

Machine learning approaches to understanding decision-making

Our research interest is to investigate the relationship between the algorithm of brain and of machine learning to understand the neural mechanism of decision making. I mainly talk about our recent findings that the mouse cerebral cortex has localized and global computation for integrating prior value and sensory evidence (Ishizu et al, bio-Rxiv, 2013). I also talk about our current challenging which models the mouse choice behavior with artificial neural networks.

Tomoyuki Furuyashiki

Professor

Kobe University

tfuruya@med.kobe-u.ac.jp

Neuroimmune Mechanisms of Stress: Insights from a Mouse Model

Clinical studies have reported inflammatory states in the brain and peripheral blood of depressive patients, even unmedicated ones. Rodent studies have shown that social stress induces inflammatory responses in the brain and peripheral blood, along with behavioral changes, as manifested in microglial activation and myeloid cell mobilization, respectively. Further studies with genetic and cellular manipulations have demonstrated that microglial activation and leukocyte mobilization are essential for social stress-induced behavioral changes. However, the roles and mechanisms of the inflammatory responses remain elusive. Here we performed transcriptomic and epigenomic analyses of microglia isolated from multiple brain areas in different stress conditions and obtained evidence suggesting that microglia are sensitized by acute and chronic social stress via glucocorticoid receptors in stress-susceptible individuals, being further activated by chronic social stress via innate immune receptors TLR2/4 for behavioral changes. Using single-cell RNA-seq analyses and pharmacological and genetic interventions, we also identified specific leukocyte subsets involved in social stress-induced behavioral changes and their transcriptome signatures. These findings elucidate the roles and mechanisms of multiple inflammatory responses in the brain and periphery for social stress-induced behavioral changes.

Jenna Gregory

Senior Clinical Lecturer

University of Aberdeen

jenna.gregory@abdn.ac.uk

Using RNA aptamers to detect TDP-43 aggregation in ALS post-mortem tissue

Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative disease, with a median survival of 18 months following motor symptom onset. Early intervention is key, as at symptom onset, there is already substantial neuronal cell loss that is difficult to recover. There is currently no known cure for ALS, but all licensed medications available for ALS have been shown to have improved efficacy if delivered early. To date, no biomarker exists to facilitate early detection. However, we recently showed that disease-related pathology can be detected in the gut years prior to motor symptom onset allowing the possibility of early detection outside of the central nervous system prior to the onset of motor disability. Further to this we have developed an RNA-based tool to detect disease-related pathology with unprecedented sensitivity and specificity. Using this tool we are able to detect early pathogenic aggregation events and we hope that this unprecedented detection sensitivity can be implemented to enable early disease detection and improve patient outcomes.

Taku Hatano

Senior Associate Professor

Juntendo University

thatano@juntendo.ac.jp

α-Synuclein Aggregation and Propagation

α-Synuclein (AS) is one of the main components of Lewy body and glial cytoplasmic inclusions. Furthermore, pathogenic mutations and multiplications of AS cause familial Parkinson's disease (PD). Therefore, AS is considered the key protein of synucleinopathy. Especially the oligomer of AS is the key to the pathogenesis in PD and disrupts its binding abilities to membranes resulting in inducing the aggregation of membranous organelles, such as mitochondria, lysosomes, synaptic vesicles, and autophagosomes. Thus, the AS-oligomers have been considered as seeds for developing the aggregation of AS and detecting them may shed light on diagnosing PD and understanding the pathomechanisms of synucleinopathy.

Although previous studies indicated that AS might be useful as a diagnostic biomarker of synucleinopathy, there have been controversial. Several reports described that patients with PD are likely to show decreasing monomer AS and increasing oligomer AS in CSF. Moreover, several groups discovered oligomers of AS in PD patients' blood samples. We also revealed serum AS oligomers using real-time quaking-induced conversion combined with immunoprecipitation (IP/RT-QuIC) assays. The technique uses the seeding properties to amplify small quantities of seeds. We found that the assay can detect serum AS-seeds in synucleinopathy, which is useful as a diagnostic biomarker of synucleinopathy. Furthermore, the aggregation, propagation propensity, and microstructures of fibrils obtained from IP/RT-QuIC showed differences between synucleinopathy. We will review AS as a helpful diagnostic biomarker and introduce our study in this session.

Haruhisa Inoue

Professor

Kyoto University

inoue-g@cira.kyoto-u.ac.jp

iPSC-based translational and reverse translational research for ALS Medicine

Since the advent of induced pluripotent stem cell (iPSC) technology 17 years ago, significant progress has been made in a medical field. As the pathophysiology of various diseases is being clarified by using human iPSC-derived neural cells, new drugs derived from the screening of iPSCs are also being developed. We conducted disease modeling, drug screening, in vitro trials, drug discoveries, and a physician-initiated clinical trial for ALS medicine by application of this unprecedented resource.
In this presentation, I would like to talk about our recent efforts and discuss various perspectives of translational and reverse translational research for ALS medicine including “Clinical trial of bosutinib for amyotrophic lateral sclerosis: Induced pluripotent stem cell-based Drug Repurposing for Amyotrophic Lateral Sclerosis Medicine (iDReAM) study”.

Aya Ito-Ishida

Team Leader

RIKEN

aishida@keio.jp

Dissecting the pathogenesis of Rett syndrome by multi-disciplinary approaches

Rett syndrome (RTT) is a neurodevelopmental disorder caused by the loss of methyl-CpG binding protein 2 (MeCP2). Despite decades of research, how MeCP2 loss leads to neurological impairment remains unclear. To clarify the molecular and cellular mechanisms that lead to neurological deficits in RTT, we have been using multi-disciplinary approaches that cover molecular to behavioral analysis. At a molecular level, our studies showed that one of the critical functions of MeCP2 is to maintain heterochromatin structure in neurons. At a behavioral level, we showed that loss of MeCP2 in inhibitory neurons leads to a specific subset of RTT-related behaviors. More recently, we have started to examine the cortical connectivity changes in the female Mecp2-heterozygous mice by conducting wide-field calcium imaging. Finally, to correlate our findings to the higher cognitive functions, we have developed an automated operant conditioning device to measure working memory and flexibility in mice. In this talk, I will share some findings gained through these newly developed approaches.

Matt Jones

Professor

University of Bristol

matt.jones@bristol.ac.uk

Sleep neurophysiology as a metric of brain health

The non-REM sleep EEG of schizophrenia patients consistently reveals abnormal thalamocortical sleep spindles and slow-waves. These oscillatory signatures constitute non-invasive, translational metrics of schizophrenia neurobiology, potentially illuminating mechanistic routes between risk factors, brain development, neural circuit dysfunction, symptoms and personalised therapies. However, grappling with complexity, heterogeneity and causality remains challenging.

I will introduce our approach to iterating between deep-brain, cellular-resolution neurophysiology in rodents and scalp EEG in genotyped volunteers and patients, most recently in young people with 22q11.2 deletion syndrome. Sleep does not hold all the answers, but I hope to make the case that integrating sleep neurophysiology into translational psychiatry can expedite understanding of the neurobiology of individual patients, optimising their diagnosis and treatment.

Atsushi Kasai

Associate Professor

Osaka University

kasai@phs.osaka-u.ac.jp

A hypothesis-free approach using Brain-wide neuronal activation mapping for deciphering complex brain functions

In the brain, the intricate communication between neurons across various brain regions governs complex brain functions such as emotion. Therefore, systematically understanding brain function and dysfunction requires a broad analysis of neuronal activation patterns. Toward this end, we have developed an automated very high-speed imaging system for block-face serial microscopy tomography, FAST (Seiriki et al, Neuron, 2017; Seiriki et al, Nat Protocols, 2019). Combining FAST imaging with the immediate early gene reporter mice, we generated brain-wide activation maps in response to a range of stimuli, including stress exposure and drugs administration. Through machine learning classification analysis of these maps, we identified previously overlooked brain regions and cell populations pivotal in stress responses and antidepressant effects. This hypothesis-free exploration approach, focusing on functional changes in the whole brain, can be applied across various animal conditions and disease models. It holds the promise of significantly enhancing our understanding of the mechanisms underlying brain function and disease pathology in the future.

Yuka Koike

Associate Professor

Niigata University

yukkoike@bri.niigata-u.ac.jp

DNA demethylation in the TDP-43 autoregulatory region links to aging.

Aging is the primary risk factor in sporadic ALS. In the motor neurons of ALS patients, TDP-43 forms cytoplasmic aggregates and is depleted from the nucleus. TDP-43 levels are strictly autoregulated via alternative splicing of TARDBP pre-mRNA. However, it is still being determined how aging affects TDP-43 autoregulation. We hypothesized that DNA methylation status in TARDBP is altered with aging and disrupts the TDP-43 auto-regulatory mechanism. To explore this hypothesis, we used the dCas9 system to de-methylate the alternative splicing-related site of the TARDBP selectively. We found that the demethylation of this region reduced alternative splicing and increased the TARDBP mRNA levels. Next, we demonstrated in the human motor cortex that this region was demethylated with aging and that the demethylation rate was correlated with TARDBP mRNA and TDP-43 protein levels. Moreover, DNA methylation age acceleration in TARDBP was associated with the early onset of ALS. The dysregulation of TDP-43 auto-regulation by age-related DNA demethylation could explain the contribution of aging in ALS pathogenesis.

Tom MacPherson

Assistant Professor

Osaka University

macpherson@protein.osaka-u.ac.jp

Striatal control of cue-guided decision-making and its impairment in Schizophrenia

Learned associations between environmental cues and the outcomes they predict (cue-outcome associations) play a major role in behavioral control, guiding not only which responses we should perform, but also which we should inhibit, in order to achieve a specific goal. The encoding of cue-outcome associations, as well as the performance of cue-guided choice behavior, is thought to involve dopamine D1 and D2 receptor-expressing medium spiny neurons (D1-/D2-MSNs) of the nucleus accumbens (NAc); however their precise functional roles have been unclear. Here, we developed two novel visual discrimination decision-making tasks in mice to separately assess the role of NAc D1-/D2-MSNs in cue-guided facilitation of appropriate behavioral responses, and cue-guided inhibition of inappropriate behavioral responses. Cell-type specific optogenetic silencing and single cell-resolution in-vivo imaging revealed NAc D1-MSN and D2-MSNs to perform separate but complementary roles in optimizing future choice behavior during learning of the tasks. Finally, it will be discussed how these insights are being applied to circuit-based interventions for impaired cue-guided decision-making in a mouse model of schizophrenia.

Masanori Matsuzaki

Professor

University of Tokyo

mzakim@m.u-tokyo.ac.jp

Auditory mismatch negativity in the common marmoset revealed by calcium imaging

Mismatch negativity is an event-related potential that includes deviance detection. The deviance detection component of auditory duration mismatch negativity (dMMN) is reduced in patients with schizophrenia. Thus, examination of the neuronal mechanisms generating the deviance detection component of dMMN in non-human primates is valuable for obtaining an understanding of how abnormalities in neuronal processing cause the symptoms of schizophrenia. We conducted one-photon and two-photon calcium imaging of the auditory cortex in awake common marmosets, and clearly found deviance detection in a dMMN paradigm. Prediction errors to a deviant tone presentation were generated as offset calcium responses of neurons in the rostral parabelt (RPB) of higher-order auditory cortex. Within several hundred milliseconds, the error signals propagated broadly into layer 1 of the primary auditory cortex (A1) and accumulated locally on top of incoming auditory signals. Blockade of RPB activity prevented deviance detection in A1. Optogenetic activation of RPB following tone presentation enhanced A1 tone response. Thus, the feedback error signal is critical for automatic detection of unpredicted stimuli in physiological auditory processing and may serve as backpropagation-like learning.

Stephen McHugh

Research Lecturer

University of Oxford

stephen.mchugh@pharm.ox.ac.uk

Hippocampal circuit mechanisms of memory consolidation

Memory consolidation is thought to require the offline reactivation of neuronal spiking activity. Hippocampal sharp-wave ripples (SWRs) in the Cornu Ammonis (CA) region, and dentate spikes in the dentate gyrus (DG) are prime candidate mechanisms underlying this process. SWRs have been studied extensively but little is known about dentate spikes. By combining triple-(DG-CA3-CA1) ensemble recordings and optogenetic interventions in mice, here we show that dentate spikes synchronize spiking activity across CA and DG principal cells and reactivate the spiking patterns seen during prior waking experience. Notably, the structure and content of this reactivation is distinct from that observed during SWRs. Importantly, suppressing DG granule cell spiking activity selectively during dentate spikes impairs subsequent memory performance, demonstrating their functional role. Thus, dentate spikes are an important hippocampal mechanism underlying offline memory formation.

Scott Mitchell

Postdoctoral Research Associate

King's College London

scott.mitchell@kcl.ac.uk

Hypomethylated FUS induced synaptic pathophysiology requires irreversible liquid-liquid phase separation and dendritic localisation of FUS condensates

The hypomethylation of fused in sarcoma (FUS) in frontotemporal lobar degeneration promotes the formation of irreversible condensates of FUS. However, the mechanisms by which these hypomethylated FUS condensates cause neuronal dysfunction are unknown. Here we report that expression of FUS constructs mimicking hypomethylated FUS causes aberrant dendritic FUS aggregates in CA1 neurons. These hypomethylated FUS aggregates exhibit spontaneous, and activity induced movement. They impair excitatory synaptic transmission, postsynaptic density-95 expression, and dendritic spine plasticity. These neurophysiological defects are dependent upon both the dendritic localisation of the condensates, and their ability to undergo liquid-liquid phase separation and form aggregates. These results indicate that the dendritic localisation and irreversible liquid-liquid phase separation is a key component of hypomethylated FUS pathophysiology in sporadic FTLD, and this can cause synapse dysfunction in sporadic FTLD.

Wendy Noble

Professor

University of Exeter

w.noble2@exeter.ac.uk

P2X7R influences tau aggregate burden in human tauopathies and shows distinct signalling in microglia and astrocytes

The purinoceptor P2X7R is a promising therapeutic target for tauopathies, including Alzheimer's disease (AD). Pharmacological blockade or genetic knockdown of P2X7R ameliorates cognitive deficits and reduces pathological tau changes in mice that model aspects of tauopathy, including mice expressing mutant human frontotemporal dementia (FTD)-causing forms of tau. However, disagreements remain over which glial cell types express P2X7R and therefore the mechanism of action is unclear. Here, we show that P2X7R protein levels increase in human AD post-mortem brain, in agreement with an upregulation of P2RX7 mRNA observed in transcriptome profiles from the AMP-AD consortium. P2X7R protein increases mirror advancing Braak stage and coincide with synapse loss. Using RNAScope we detect P2RX7 mRNA in microglia and astrocytes in human AD brain, including in the vicinity of senile plaques. In microglia, P2X7R activation modulates the inflammasome pathway by promoting the formation of active complexes and release of IL-1β. In astrocytes, P2X7R activates NFkB signalling and increases production of the cytokines CCL2, CXCL1 and IL-6 together with the acute phase protein Lcn2. To further explore the role of P2X7R in a disease-relevant context, we expressed wild-type or FTD-causing mutant forms of tau in mouse organotypic brain slice cultures. Inhibition of P2X7R reduced insoluble tau levels without altering soluble tau phosphorylation or synaptic localisation, suggesting a non-cell autonomous role of glial P2X7R on pathological tau aggregation. These findings support further investigations into the cell-type specific effects of P2X7R-targeting therapies in tauopathies.

Kotaro Oiwa

Research Associate

Nagoya University

k.oiwa@med.nagoya-u.ac.jp

Molecular basis underlying TDP-43 pathology in Amyotrophic Lateral Sclerosis

Cytoplasmic aggregation of TDP-43, also known as TDP-43 pathology, is a pathological hallmark of amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD). However, the mechanisms underlying TDP-43 cytoplasmic mislocalization and its subsequent aggregation remain unclarified. We have revealed that skein-like inclusions immunopositive for TDP-43 in human ALS motor neurons are frequently colocalized with HDAC6, suggesting involvement of aggresome. Moreover, we found that TDP-43 dimerization is impaired in the postmortem brains and spinal cords of sporadic ALS patients. Expression of dimerization-deficient mutant TDP-43 in neuronal cells and iPS-derived motor neurons recapitulates TDP-43 pathology and induces cytotoxicity. Furthermore, TDP-DiLuc, a novel reporter assay, could detect decreased TDP-43 dimerization prior to TDP-43 pathological changes induced by the transcription inhibition and spliceosomal defects linked to aberrant RNA metabolism in ALS. Our findings identify TDP-43 monomerization as a critical determinant inducing TDP-43 pathology in ALS.

Abidemi Otaiku

Clinical Fellow

University of Birmingham

a.otaiku@nhs.net

Do distressing dreams cause neurodegenerative diseases?

Distressing dreams (bad dreams and nightmares) are very common among people with neurodegenerative diseases (NDs) such as dementia and Parkinson's disease. However, until recently, it remained unknown whether these dreams held any clinical significance. This presentation will focus on my recent work which has shown that distressing dreams: (i) predict rapid disease progression in people with newly diagnosed NDs, (ii) precede the onset of NDs by several years or even decades, and (iii) may be strong causal risk factors for developing NDs.

Trevor Robbins

Professor

University of Cambridge

twr2@cam.ac.uk

Compulsivity: Neural basis and psychiatric implications

Impulsivity and compulsivity represent complementary cases of dysexecutive control, of considerable relevance to psychiatry. Whilst there has been intense focus on the dimensional construct of impulsivity, compulsivity has received far less attention. A working definition of compulsivity is of actions persisting inappropriate to the situation, having no obvious relationship to the ultimate goal and often resulting in undesirable consequences. This definition can be dissected neuropsychologically in several ways which I will illustrate by describing behavioural, computational and neuroimaging studies in two prototypical and devastating disorders of compulsivity, stimulant drug addiction and obsessive-compulsive disorder (OCD). One notion is that compulsive behavior is uncontrolled and excessive habitual responding at the expense of adaptive goal-seeking behaviour. Habits are governed by stimulus-response representations that do not involve goals or rewards. These two forms of behavioural control have been characterized by studies in experimental animals and humans indicating mediation by distinct, though interactive, fronto-striatal systems. Hypothetically, addiction and OCD represent imbalance in these neural systems for goal-directed and habitual behaviour. I will examine several different ways of testing this hypothesis in stimulant drug abusers and patients with OCD and make comparisons indicating common, as well as distinctive, features of the two disorders. I will explore the implications of this research for causal accounts of behaviour including psychopathology, new therapeutic approaches, and the functional organization of fronto-striatal systems in the brain and their chemical neuromodulation.

Bhuvaneish Thangaraj Selvaraj

Chancellor's Fellow

University of Edinburgh

bthangar@ed.ac.uk

AMPA receptor dysfunction in amyotrophic lateral sclerosis

MND/ALS is a devastating progressive life-limiting neurological disorder with a lifetime risk of 1 in 300, no cure, only 1 licensed therapy available globally, median life expectancy of only 2-3 years after diagnosis. One of the key mechanisms of ALS pathophysiology includes glutamate-induced excitotoxicity (Williams et al., 1997; Vandenberghe et al 2000; Cleveland & Rothstein, 2001; Van Den Bosch et al., 2006). Indeed, we have previously shown in MN carrying C9ORF72 mutation displayed increased expression of calcium permeable AMPA receptors (ligand gated ion channels) leading to increased vulnerability to excitotoxicity (Selvaraj et al., 2018). However, molecular mechanisms underlying this pathophysiology and its wider relevance to sporadic ALS – which contributes to 90% ALS cases - are unknown. Using human stem cell disease modelling from sporadic ALS and TDP43 ALS cases, patch-clamp electrophysiology, and patient autopsy tissues, in this study we aim to provide mechanistic insight of AMAPR dysfunction leading to glutamate excitotoxicity in ALS MNs.

Mari Shiozaki

PhD Student

Keio University

mari.z6@keio.jp

The Role of Cbln1 in Age-Related Hearing Loss: Insights from Cbln1 Knock-out Mice

Age-related hearing loss (ARHL) is one of the most prevalent sensory deficits in the elderly, affecting over half of the population aged over 75. However, the molecular mechanisms underlying ARHL are not fully understood. Recently, emerging research on Cbln1 and other C1q family molecules has revealed their essential roles in synapse formation and maintenance in the CNS. Interestingly, Cbln1 is specifically expressed in the inner ear throughout life, prompting us to investigate its function in the context of ARHL. We found that Cbln1 knockout (KO) mice exhibited impaired hearing in high-frequency sounds, a characteristic feature of ARHL. Cbln1 was specifically localized at the axon terminal of the cholinergic medial olivocochlear (MOC) neuron, which innervates outer hair cells (OHC) and plays a crucial role in suppressing OHC electromotility, providing protection against acoustic trauma. Furthermore, we observed a progressive decrease in the size of presynaptic terminals in Cbln1 KO mice, leading to a subsequent reduction in the number of OHCs. These results indicate that Cbln1 plays a pivotal protective role in the inner ear by maintaining MOC-OHC synapses. To further validate these findings, we are currently conducting experiments to assess whether Cbln1 expression in the cochlea using adeno-associated virus (AAV) can alleviate vulnerability to acoustic damage. By unraveling the physiological significance of Cbln1 in the cochlear system, we aim to develop potential applications for treating ARHL.

Masami Suzukake

Assistant Investigator

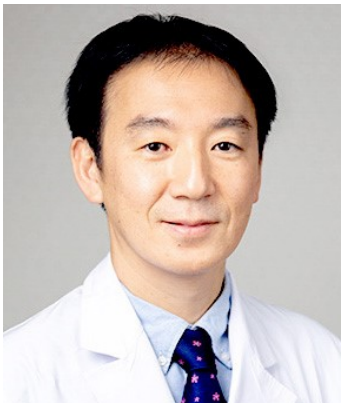
Tokyo Metropolitan Institute of Medical Science

suzukake-ms@igakuken.or.jp

A mouse model of tau propagation using synthetic tau fibrils

Accumulation of assembled tau protein in the central nervous system is characteristic of Alzheimer's disease and several other neurodegenerative diseases, called tauopathies. Recent studies have revealed that the propagation of assembled tau is key to understanding the pathological mechanisms of these diseases. Mouse models of tau propagation have been established by intracerebral injection of human-derived tau seeds. However, there are ethical and legal restrictions on the use of human brain samples and the amount and quality of insoluble tau in brain samples vary among cases, which can affect the reproducibility of experiments. To overcome these issues, we have been working to generate a model mouse using synthetic tau filaments.

We found that dextran sulphate (DS), a sulphated glycosaminoglycan, induces the assembly of recombinant tau protein into filaments in vitro. DS-induced tau filaments had different conformational features than those induced by heparin. Intracerebral injection of DS-induced tau fibrils into wild-type mice caused phosphorylated tau pathology and it spread to anatomically connected regions over time. In biochemical analyses, sarkosyl-insoluble and hyperphosphorylated tau was observed in the mouse brains. In addition, the injected mice had lower levels of acetylcholine in the hippocampus and showed cognitive dysfunction.



Hidehiko Takahashi

Professor and Chair

Toyko Medical and Dental University

hidepsyc@tmd.ac.jp

Hypothesis testing and data-driven psychiatric research using artificial intelligence

The introduction of artificial intelligence in medicine has impacted various stages of diagnosis and treatment, and psychiatry is no exception. Diagnosis of psychiatric disorders, in particular, is currently based on superficial behavioral observations and self-statements, and the development of biomarkers has long been awaited. Using artificial intelligence to develop biomarkers and study pathophysiology in a data-driven, hypothesis-free manner is a natural approach.

On the contrary, advances in computational science and analytical techniques, including artificial intelligence, are making it possible to test hypotheses that were conceived by our predecessors but could not be tested. I would like to introduce the research on imaging biomarkers for psychiatric disorders and visualization of core symptoms of schizophrenia that we have been working on with artificial intelligence researchers.

Masako Tamaki

Team Leader

RIKEN

masako.tamaki@riken.jp

Roles of NREM and REM sleep in learning and memory in humans

The roles of NREM and REM sleep in learning and memory remain unclear. In procedural learning, it has been suggested that NREM sleep plays a role in performance improvements (offline performance gains), while REM sleep makes learning more resilient to interference (stabilization). These suggest that plasticity of learning increases for offline performance gains during NREM sleep, while it decreases for stabilization during REM sleep. To test this hypothesis, in visual perceptual learning (VPL) using magnetic resonance spectroscopy in asleep human subjects, we measured the excitation/inhibition (E/I) ratio which represents the amount of plasticity of learning during sleep. Performance deteriorated significantly after NREM sleep without REM sleep. The E/I ratio increased during NREM sleep while it decreased during REM sleep. The E/I ratio during NREM sleep was correlated with offline performance gains, while the E/I ratio during REM sleep was correlated with stabilization. Furthermore, by utilizing temporary sleep disturbances, known as the first-night effect (FNE), we found that the E/I ratio increases and performance improvements were significantly impaired when the FNE occurred. These results suggest that NREM and REM sleep play complementary roles for learning, which are reflected by significantly different neurochemical processing, and that the quality of sleep matters for such processes to occur.

Cezar M. Tigaret

Hodge Lecturer in Neuroscience

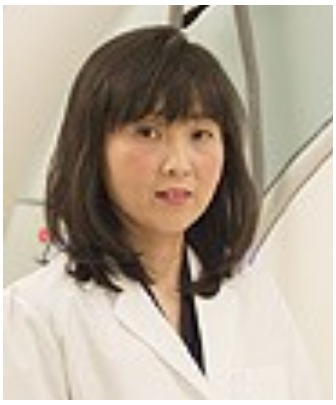
Cardiff University

tigaretc@cardiff.ac.uk

Psychiatric risk gene CACNA1C, synaptic plasticity and associative learning

The CACNA1C gene encoding the pore-forming α1 subunit of CaV1.2 L-type voltage-gated Ca2+ channels (L-VGCCs) has been identified as cross-disorder risk gene for psychiatric conditions including schizophrenia and bipolar disorder. Common CACNA1C risk variants are intronic and are likely to alter CACNA1C gene expression, with recent studies indicating a reduced CACNA1C expression in the hippocampus. To translate genetics to neurobiological mechanisms we investigated the impact of reduced CACNA1C dosage on rat cognitive, synaptic and circuit phenotypes implicated by patient studies. We show that rats hemizygous for Cacna1c (Cacna1c+/- rats) have disrupted Hebbian synaptic plasticity and circuit activity in the hippocampus culminating in markedimpairments in learning to disregards non-salient stimuli, a behavioural deficit preciously associated with schizophrenia.

The activation of ERK pathway by a small-molecule BDNF mimetic compound acting at TrkB/C receptors rescued both the synaptic plasticity and behavioural deficits in Cacna1c+/- rats, indicating that the functional consequences of genetic variation in CACNA1C can be compensated by targeting the signalling mechanisms activated by L-VGCCs.



Makiko Yamada

Group Leader

National Institutes for Quantum Science and Technology

yamada.makiko@qst.go.jp

Positive illusions: integration from molecules to functional brain networks

Cognitive biases, such as biased views and assumptions, have long been studied in cognitive science, social psychology, and behavioral economics. They can lead to what is widely referred to as irrationality in behavioral economics, such as erroneous judgments and illogical interpretations, but on the other hand, they also have adaptive aspects, such as self-serving interpretations that motivate people to look to the future and lead to mental and physical health. My talk will focus on the latter aspect of cognitive bias, "positive illusions," which are positively biased interpretations of oneself, and will provide an overview of its benefits and drawbacks and outline the relationship between positive illusions and brain functions.

Posters

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Information about posters and the poster blitz

- The poster boards are landscape format and sized 1200mm x 900mm.
- The poster blitz will take place before the poster session and is an opportunity to advertise your poster and make sure those interested find it easily amongst the other posters.
- Each poster blitz talk has a 2 minute slot (2 minutes talk, no discussion)
- Please send your slides in advance (one or two slides, 16:9 ratio) to **ukjapanneurosymposium@cardiff.ac.uk**
- All slides by the presenters will be merged on the Secretariat's PC and used by the presenters on the day.
- You can include animated features in your slide, but please be aware that they may not work properly on the Secretariat's PCs.

Information about poster prize voting

- Please visit the following website to vote for your preferred poster or use the associated QR code below.
- https://forms.office.com/e/YkkdypDx2v

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4	Martina	Sassi	CHARACTERIZING GHRELIN AS A NOVEL BLOOD-BASED DEMENTIA BIOMARKER
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6	Natalie	Connor-Robson	Understanding endocytic dysfunction in Late Onset Alzheimer's Disease.
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10	Makoto	Uji	Cerebrospinal fluid dynamics during NREM and REM sleep by a simultaneous sparse-fMRI and EEG method
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12	Aurelio	Cortese	Functional connectivity neurofeedback for depression
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14	Jennifer	Imm	A ROLE FOR EPIGENETIC MECHANISMS IN THE LEWY BODY DEMENTIAS
15	Scott	Mitchell	Segmented synapse weakening by hyperphosphorylated microtubule associated protein Tau in dendritic spines
16	Charlotte	Wiltshire	The role of the supplementary motor area in speech production: Evidence from people who do, and do not stutter.
17	Wei-Li (William)	Kuan	Towards the development of potent small molecules to inhibit ferroptosis as a therapeutic strategy for Parkinson’s disease
18	Tom	Massey	The role of MutL complexes in CAG repeat expansion in human iPSC model of Huntington's disease
19	So	Takasugi	The Localization and Function of Cbln1 in PNS
20	Uroosa	Chughtai	Loss-of-function of ALS/FTD-associated gene TBK1 is associated with cell autonomous microglial dysfunction
21	Stuart	Williams	Investigating the role of cortical feedback between S2 and S1 during whisker-guided texture discrimination learning
22	Hanna	Wyszynska	Interactions between primary and secondary somatosensory cortex
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26	Hannah	Clarke	A Drosophila model of Wwox deficiency in the presence of human amyloid has significantly altered behaviour mediated by a glycolytic shift
27	Maxime	Assous	Role of β2-nAChRs in striatal associated circuits and behaviors
28	Rachel	Hills	Human Induced Pluripotent Stem Cell-Derived Dopamine Neuron Transplants; Neurite Outgrowth and Gene Expression Profile Correlate with Efficacy
29	Koichi	Tabata	Hypozincemia is associated with clinical severities of schizophrenia

Maxime Assous

Senior Lecturer

Cardiff University

AssousM@cardiff.ac.uk

Role of β2-nAChRs in striatal associated circuits and behaviors

Scott J. Mitchell, Seung-Chan Kim, Chotchanit Sunrat, Saviana A. Barbati, Ambra Annibali, Kwangwook Cho.

All authors: Department of Basic and Clinical Neuroscience, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London SE5 9TX, United Kingdom.

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Jackie Casey

Postdoctoral Research Fellow

University College London

ucbtjm4@ucl.ac.uk

Loss of progranulin leads to impaired PINK1/Parkin mitophagy

Jackie Casey[1], Daniela Melandri[1], Charlie Arber[1], Marc Soutar[1], Christopher Holler[4], Thomas Kukar[4], Adrian M. Isaacs[1,3], Jonathan Rohrer[2], Jennifer M. Pocock[5], Helene Plun-Favreau[1], Selina Wray[1].

1 Department of Neurodegenerative Disease, UCL Queen Square Institute of Neurology, University College London, London, WC1N 1PJ.

2 Dementia Research Centre, UCL Queen Square Institute of Neurology, Queen Square, London, WC1N 3BG.

3 UK Dementia Research Institute at University College London, Gower Street, London.

4 Department of Pharmacology and Chemical Biology, Emory University, School of Medicine, Atlanta, Georgia, USA, 30322.

5 Department of Neuroinflammation, UCL Queen Square Institute of Neurology, University College London, London, WC1N 1PJ

Uroosa Chugtai

PhD Student

Cardiff University

ChughtaiUA@cardiff.ac.uk

Loss-of-function of ALS/FTD-associated gene TBK1 is associated with cell autonomous microglial dysfunction

Uroosa Chughtai[1,2], Raja Nirujogi[3], Daniel Cabezas De La Fuente[4], Gaynor Smith[1,4], Meng Li[2,4], Dario Alessi[3], Owen Peters[1,2]

1 UK Dementia Research Institute, Cardiff University, Cardiff, United Kingdom.

2 School of Biosciences, Cardiff University, Cardiff, United Kingdom.

3 MRC Protein Phosphorylation & Ubiquitylation Unit, University of Dundee, Dundee, United Kingdom.

4 School of Medicine, Cardiff University, Cardiff, United Kingdom.

Hannah Clarke

PhD Student

Cardiff University

clarkehj@cardiff.ac.uk

A Drosophila model of Wwox deficiency in the presence of human amyloid has significantly altered behaviour mediated by a glycolytic shift

Hannah Clarke, Daniel Maddison, Lucie Tkacova, Leonardo Amadio, Katie O'Hare, Clement Renault, Wynand Van Der Goes Van Naters, Valentina Escott-Price, Owen Peters, James Hodge, Gaynor Smith.

Natalie Connor-Robinson

Research Fellow

Cardiff University

connor-robsonnl@cardiff.ac.uk

Understanding endocytic dysfunction in Late Onset Alzheimer's Disease

Sri Goberdhan, Cameron Lewis, Atahualpa Castillo Morales, Karolina Nowak, Johanna Maninger, Natalie Connor-Robson

Cardiff University Dementia Research Institute

Aurelio Cortese

ATR Institute International

Principal Investigator

cortese_a@atr.jp

Functional connectivity neurofeedback for depression

Cortese Aurelio [1*], Taylor Jessica [1], Callan Akiko [1], Kawato Mitsuo [1,2#]

1 Department of Decoded Neurofeedback, Computational Neuroscience Laboratories, ATR, Kyoto
2 XNef, Kyoto

*Department Head
#Director of research for the Kokusai-nou AMED project at ATR

Leon Crowley

PhD Student

King's College London

leon.crowley@kcl.ac.uk

Inhibiting glycogen synthase kinase 3 suppresses TDP-43-mediated neurotoxicity in a caspase-dependant manner

Leon Crowley[1], Matthew A. White[1], Francesca Massenzio[1], Xingli Li[2], Michael P. Coleman[3], Sami J. Barmada[2], Jemeen Sreedharan[1].

1 King's College London
2 University of Michigan
3 The Babraham Institute

Chiara Franceschi

PhD Student

Cardiff University

FranceschiC@Cardiff.ac.uk

Is the subiculum involved in long-term spatial memory?

Chiara Franceschi, Sungmin Kang, Jonathan Wilson, John Aggleton, Joseph O'Neill.

All authors: Cardiff University.

Rachel Hills

Research Associate

Cardiff University

HillsR2@cardiff.ac.uk

Human Induced Pluripotent Stem Cell-Derived Dopamine Neuron Transplants; Neurite Outgrowth and Gene Expression Profile Correlate with Efficacy

Rachel Hills[1], Andres M. Bratt-Leal[2,3,4,] Jim A. Mossman[2], Ha Tran[2,3,4,] Roy M. Williams[2,3], David G. Stouffer[3], Irina V. Sokolova[5], Pietro P. Sanna[5], Jeanne F. Loring[3*], Mariah J. Lelos[1*] .

1 Brain Repair Group, School of Biosciences, Museum Avenue, Cardiff University, Cardiff, CF10 3AX, U.K.

2 Aspen Neuroscience, Inc. 10835 Road to the Cure, Ste. 100, San Diego, CA, 92121

3 Department of Molecular Medicine, Centre for Regenerative Medicine, Scripps Research, 10550 North Torrey Pines Road, La Jolla, CA, 92037, USA.

4 Summit For Stem Cell Foundation, San Diego, California, USA

5 Department of Immunology and Microbiology, Scripps Research, 10550 North Torrey Pines Road, La Jolla, CA, 92037, USA

Jennifer Imm

Post Doctoral Research Associate

University of Exeter

j.l.imm@exeter.ac.uk

A role for epigenetic mechanisms in the lewy body dementias

Jennifer Imm[1], Joshua Harvey[1], Byron Creese[1], Leonidas Chouliaras[2], Emma Dempster[1], Clive Ballard[1], John T O'Brien[2], Dag Aarsland[3], Jonathan Mill[1], Ehsan Pishva[4], Katie Lunnon[1].

1 University of Exeter Medical School, University of Exeter.

2 Department of Psychiatry, University of Cambridge.

3 Department of Old Age Psychiatry, Kings College London.

4 Maastricht University, The Netherlands.

Daisuke Ito

Clinical Fellow

Nagoya University

daisuke.ito@med.nagoya-u.ac.jp

Dysregulated N-acyltaurine metabolism is a promising biomarker and a therapeutic target of sporadic ALS

Daisuke Ito[1], Atsushi Hashizume[1,2], Madoka Iida[1], Yohei Iguchi[1], Shinichiro Yamada[1], Yoshiyuki Kishimoto[1], Shota Komori[1], Yuto Takemoto[3], Masahiro Nakatochi[3], Masahisa Katsuno[1,2].

1 Department of Neurology, Nagoya University Graduate School of Medicine.
2 Clinical research education, Nagoya University Graduate School of Medicine.
3 Public Health Informatics Unit, Department of Integrated Health Sciences, Nagoya University Graduate School of Medicine.

Sungmin Kang

Research Associate

Cardiff University

KangS4@cardiff.ac.uk

Reorganization of s1 cell assemblies during learning in freely moving rodent.

Sungmin Kang, Hanna Wyszynska, Chiara Franceschi, Rob Honey, Kevin Fox, Joseph Oneill.

All Authors: Cardiff University.

Wei-Li (William) Kuan

Head of Biology

University of Cambridge

wlk21@cam.ac.uk

Towards the development of potent small molecules to inhibit ferroptosis as a therapeutic strategy for Parkinson's disease

Cathryn Ugalde, David Allendorf, Cara Pedley, Andrew Lim, Renata Lopes Alves, Henriette Willems, Esperanza Agullo Pascual, Nikhita Annaiyappa, Jonathan Clarke, David Winpenny, David Harrison, James Duce, Steve Andrews, Wei-Li Kuan and John Skidmore.

All authors are affiliated to The ALBORADA Drug Discovery Institute, University of Cambridge, Island Research Building, Cambridge Biomedical Campus, Hills Road, Cambridge CB2 0AH, UK.

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Ruixiang Li

Postdoctoral Researcher

RIKEN

ruixiang.li@riken.jp

Probing Functional Brain Network in the Mouse Model of Rett Syndrome by Wide-field Calcium Imaging

Ruixiang Li[1], Toshiki Nomura[1,2] & Aya Ito-Ishida[1].

1 RIKEN Center for Brain Science, Wako, Saitama, 351-0198, Japan.
2 Graduate School of Medicine, The University of Tokyo, Tokyo, 113-0033, Japan.

Vaishnavi Manohar

PhD Student

King’s College London

vaishnavi.1.manohar@kcl.ac.uk

Investigating the burden of non-coding variants of TARDBP on ALS-FTD

Vaishnavi Manohar and Jemeen Sreedharan.

All authors: King's College London.

Tom Massey

Clinical Scientist

Cardiff University

MasseyT1@cardiff.ac.uk

The role of MutL complexes in CAG repeat expansion in human iPSC model of Huntington's disease

Joseph Stone, Caroline Binda, Nicholas Allen, Lesley Jones, Tom Massey*.

Scott Mitchell

Postdoctoral Research Associate

King's College London

scott.mitchell@kcl.ac.uk

Segmented synapse weakening by hyperphosphorylated microtubule associated protein Tau in dendritic spines

Scott J. Mitchell, Seung-Chan Kim, Chotchanit Sunrat, Saviana A. Barbati, Ambra Annibali, Kwangwook Cho.

All authors: Department of Basic and Clinical Neuroscience, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London SE5 9TX, United Kingdom.

Zeynep Okray

Postdoctoral Research Scientist

University of Oxford

Zeynep.Okray@dpag.ox.ac.uk

Multisensory learning binds neurons into a cross-modal memory engram

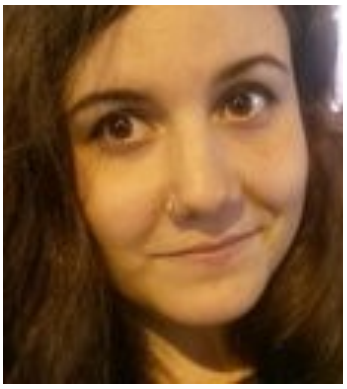
Zeynep Okray[1,3], Pedro F. Jacob[1,3], Ciara Stern[1], Kieran Desmond[1], Nils Otto[1,2], Clifford B. Talbot[1], Paola Vargas-Gutierrez[1] & Scott Waddell[1].

1 Centre for Neural Circuits & Behaviour, University of Oxford, Oxford, UK.

2 Present address: Institute of Anatomy and Molecular Neurobiology, Westfälische Wilhelms-Universität Münster, Münster, Germany.

3 These authors contributed equally: Zeynep Okray, Pedro F. Jacob.

Corresponding author emails: zokray@gmail.com; scott.waddell@cncb.ox.ac.uk.



Martina Sassi

Post Doctoral Research Assistant

martina.sassi@swansea.ac.uk

Characterizing Ghrelin as a novel blood-based dementia biomarker

M. Sassi[1]; B. David[1]; A. Thomas[1]; A. Hornsby[1,2]; N. Jones[1]; C. Thornton[1]; J. Davies[1].

1 Molecular Neurobiology, Institute of Life Sciences, School of Medicine, Swansea University, Swansea, United Kingdom.
2 School of Biosciences, Cardiff University, Cardiff, UK.

Hiroki Shiwaku

Associate Professor

Tokyo Medical and Dental University

shiwaku.npat@mri.tmd.ac.jp

Autoantibodies against NCAM1 and NRXN1 from patients with schizophrenia cause schizophrenia-related behavior and changes in synapses in mice

Hiroki Shiwaku and Hidehiko Takahashi.

All authors: Department of Psychiatry and Behavioral Sciences, Tokyo Medical and Dental University Graduate School.

Koichi Tabata

Researcher

Tokyo Metropolitan Institute of Medical Science

tabata-ki@igakuken.or.jp

Hypozincemia is associated with clinical severities of schizophrenia

Koichi Tabata

Tokyo Metropolitan Institute of Medical Science

So Takasugi

Graduate Student

Keio University

sotakasugi95@gmail.com

The Localization and Function of Cbln1 in PNS

So Takasugi, Keiko Matsuda and Michisuke Yuzaki.

All authors: Keio University, School of Medicine, Department of Physiology.

Jasper Teutsch

PhD Student

Newcastle University

J.A.Teutsch2@newcastle.ac.uk

Trial-resolution neural representations of behavioral strategies during tactile reversal learning

Jasper Teutsch[1], Rohan Rao[1], Fritjof Helmchen[2], Mark Humphries[3], Silvia Maggi[3], Abhishek Banerjee[1].

1 Adaptive Decisions Lab, Biosciences Institute, Newcastle University

2 Brain Research Institute, University of Zürich

3 School of Psychology, University of Nottingham

Cezar Tigaret

Lecturer

Cardiff University

TigaretC@cardiff.ac.uk

Neural mechanisms linking genetic variation in the CACNA1C gene to risk for psychiatric illness

Cezar M. Tigaret[1], Tzu-Ching E. Lin[1], Edward R. Morrell[2,3], Lucy Sykes[1,4], Anna L. Moon [1,5], Michael C. O'Donovan[1,5], Michael J. Owen[1,5], Lawrence S. Wilkinson[1,2,5], Matthew W. Jones[3], Kerrie L. Thomas[1,6], Jeremy Hall[1,5].

1 Neuroscience and Mental Health Innovation Institute, Cardiff University.

2 School of Psychology, Cardiff University.

3 School of Physiology, Pharmacology and Neuroscience, University of Bristol.

4 Neem Biotech, UK.

5 MRC Centre for Neuropsychiatric Genetic and Genomics, Cardiff University.

6 School of Bioscience, Cardiff University.

Makoto Uji

Research Scientist

RIKEN

makoto.uji@riken.jp

Cerebrospinal fluid dynamics during NREM and REM sleep by a simultaneous sparse-fMRI and EEG method

Makoto Uji[1], Xuemei Li[1], An Saotome[1,2], Ryosuke Katsumata[1,3], Sayaka Aritake[1,2], Chisato Suzuki[1], Kenichi Ueno[1], Masako Tamaki[1,4].

1 RIKEN Center for Brain Science, Saitama, Japan.
2 Saitama Prefectural University, Saitama, Japan.
3 Chiba University, Chiba, Japan.
4 RIKEN Cluster for Pioneering Research, Saitama, Japan.

Stuart Williams

Post Doctoral Researcher

Cardiff University

williamss169@cardiff.ac.uk

Investigating the role of cortical feedback between S2 and S1 during whisker-guided texture discrimination learning

Stuart Williams and Kevin Fox.

All authors: School of Biosciences, Cardiff University.

Charlotte Wiltshire

Lecturer

Bangor University

c.wiltshire@bangor.ac.uk

The role of the supplementary motor area in speech production: Evidence from people who do, and do not stutter

Charlotte E.E. Wiltshire[1,2], Nicole Benker[2], Rosa Hufschmidt[2], Anton Gadringer[2], Philip Hoole[2].

1 School of Human and Behavioural Sciences, Bangor University, UK.
2 Institute for Phonetics and Speech Processing, Ludwig-Maximilians-University, Munich, Germany.

Hanna Wyszynska

PhD Student

Cardiff University

wyszynskah@cardiff.ac.uk

Interactions between primary and secondary somatosensory cortex

Hanna Wyszynska and Sungmin Kang.

All authors: Cardiff University.

Biographies

Symposiwm Niwrowyddoniaeth DU-Japan

Vale Resort
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2 Medi 2023

UK-Japan Neuroscience Symposium

Vale Resort
Pontyclun
31 August –
2 September 2023

Biography

Dr Omer Bayraktar

Group Leader at the Wellcome Sanger Institute

Research Overview

Dr Bayraktar uses single cell and spatial transcriptomics technologies to characterize human brain cellular diversity in health and disease. During his training, Omer discovered neural stem cell patterning mechanisms (Nature 2013) and astrocyte layer diversity in the cerebral cortex (Nature Neuro 2020). Omer started his independent research group at the Wellcome Sanger Institute in 2018. His team has developed the cell2location computational tool to map fine cell types in spatial transcriptomics (Nature Biotech 2022). Omer is internationally funded by Wellcome LEAP, SFARI and CZI, and he steers spatial genomic strategy at the Sanger Institute.

Professor Haruhiko Bito

Professor, University of Tokyo

Research Overview

Haruhiko Bito is a Professor and Chair of Neurochemistry at the University of Tokyo. He graduated from the University of Tokyo with an M.D. and a Ph.D. in Biochemistry in 1993. After postdoctoral training at Stanford University as an HFSP Fellow, he started his laboratory in Pharmacology at Kyoto University in 1997 before moving to head the Department of Neurochemistry at the University of Tokyo in 2003. Dr. Bito has deciphered novel functions of many members of the CaMK family, and elucidated the bidirectional neuronal signaling between the synapses and the nucleus, essential for late-phase plasticity and long-term memory. Dr. Bito has also designed powerful molecular tools (E-SARE synthetic activity-dependent enhancer and next-generation Ca2+ indicators XCaMPs) that help capture and measure neuronal ensemble activity critical for cognition. He received the Young Investigator Awards from the Japanese Societies for Pharmacology (2003) and Biochemistry (2004), the Tsukahara Award (2011), AND Investigator Award (Molecular Brain, 2015), and the Setsuro Ebashi Award (2020).

Dr. Kathryn Bowles

Group Leader, UK DRI Centre at the University of Edinburgh

Research Overview

My research interest is in understanding the genetic and mechanistic biology underlying Tauopathy. In particular, I find it fascinating that multiple neurodegenerative diseases are associated with altered tau processing and accumulation, while encompassing a diverse array of neuropathological and clinical phenotypes. The MAPT locus has also been genetically associated with other neurodegenerative diseases that are not characterized by the presence of tau pathology, such as Parkinson's disease. We use a combination of functional genomics, biochemical and cell culture approaches to investigate the mechanisms underlying the regulation of MAPT expression, function and splicing. This knowledge forms the basis for understanding how these processes are dysregulated in, and contribute to Alzheimer's disease, progressive supranuclear palsy, Parkinson's disease and multiple other tauopathies. My lab uses human iPSC models to further understand the biological mechanisms underlying multiple Tauopathies, with a focus on cellular stress, synaptic biology and neuronal-glial interactions.

Selected Publications

1. Bertucci T, Bowles KR, Lotz S, Qi L, Stevens K, Goderie SK, Borden S, Oja LM, Lane K, Lotz R, Lotz H, Chowdhury R, Joy S, Arduini BL, Butler DC, Miller M, Baron H, Sandhof CA, Silva MC, Haggarty SJ, Karch CM, Geschwind DH, Goate AM, Temple S. Improved protocol for reproducible human cortical organoids reveals early alterations in metabolism with MAPT mutations. bioRxiv 2023. doi: 10.1101/2023.07.11.548571 (under review at Cell Stem Cell)

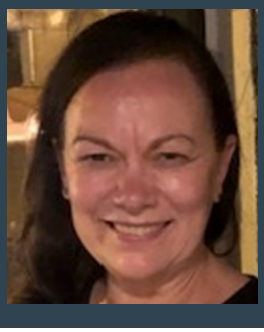
2. Bowles KR, Pugh DA, Pedicone C, Oja L-M, Weitzman SA, Liu Y, Chen JL, Disney MD, Goate AM. Development of MAPT S305 mutation models exhibiting elevated 4R tau expression, resulting in altered neuronal and astrocytic function. bioRxiv 2023. doi: 10.1101/2023.06.02.543224 (under revision at Cell Reports).

3. Bowles KR, Pugh DA, Liu Y, Patel T, Renton AE, Bandres-Ciga S, Gan-Or Z, Heutink P, Siitonen A, Bertelsen S, Cherry JD, Karch CM, Frucht SJ, Kopell BH, Peter I, Park YJ, Charney A, Raj T, Crary JF, Goate AM. 17q21.31 sub-haplotypes underlying H1-associated risk for Parkinson's disease are associated with LRRC37A/2 expression in astrocytes. Mol Neurodegener. 2022. doi: 10.1186/s13024-022-00551-x

4. Bowles KR, Pugh DA, Oja LM, Jadow BM, Farrell K, Whitney K, Sharma A, Cherry JD, Raj T, Pereira AC, Crary JF, Goate AM. Dysregulated coordination of MAPT exon 2 and exon 10 splicing underlies different tau pathologies in PSP and AD. Acta Neuropathol. 2022. doi: 10.1007/s00401-021-02392-2.

5. Bowles KR, Silva MC, Whitney K, Bertucci T, Berlind JE, Lai JD, Garza JC, Boles NC, Mahali S, Strang KH, Marsh JA, Chen C, Pugh DA, Liu Y, Gordon RE, Goderie SK, Chowdhury R, Lotz S, Lane K, Crary JF, Haggarty SJ, Karch CM, Ichida JK, Goate AM, Temple S. ELAVL4, splicing, and glutamatergic dysfunction precede neuron loss in MAPT mutation cerebral organoids. Cell. 2021. doi: 10.1016/j.cell.2021.07.003.

Biography



Professor Verity J Brown

Professor of Behavioural Neuroscience, University of East London and University of St Andrews (Honorary)

Research Overview

My research focuses on the psychological characteristics and neural basis of behavioural flexibility and attentional control. Using a test we devised for rats, we look at the basis of reversal learning and attentional set-shifting. Impairments in cognitive flexibility are seen in many neurological and psychiatric disorders and therefore the work is of interest to pharmaceutical companies developing treatments for these disorders.

Selected Publications

1. Wang J, Tait DS, Brown VJ and Bowman EM (2019) Exacerbation of the credit assignment problem in rats with lesions of the medial prefrontal cortex is revealed by Bayesian analysis of behavior in the pre-solution period of learning. Behavioural brain research,372: Article number: 112037, DOI: 10.1016/j.bbr.2019.112037
2. Tait DS, Bowman EE, Miller S, Dovlatyan M, Sanchez C, and Brown VJ (2021) Escitalopram Restores Reversal Learning Impairments in Rats with Lesions of Orbital Frontal Cortex. Language, Cognition, and Mind 7: 389-409. DOI: 10.1007/978-3-030-50200-3_18
3. Dhawan S, Tait DS, and Brown VJ (2019) More rapid reversal learning following overtraining in the rat is evidence that behavioural and cognitive flexibility are dissociable, Behavioural brain research,363: 45-52. DOI: 10.1016/j.bbr.2019.01.055
4. Tait DS, Bowman EM, Neuwirth LS and Brown VJ (2018) Assessment of intradimensional/extradimensional attentional set-shifting in rats. Neuroscience & Biobehavioral Reviews, 89: 72-84. DOI: 10.1016/j.neubiorev.2018.02.013
5. Gilmour G, Arguello A, Bari A, Brown VJ, Carter C, Floresco SB, Jentsch DJ, Tait DS, Young JW, Robbins TW (2013) Measuring the construct of executive control in schizophrenia: Defining and validating translational animal paradigms for discovery research, Neuroscience and Biobehavioral Reviews, 37 (9), pp. 2125-2140. DOI: 10.1016/j.neubiorev.2012.04.006

Biography

Professor Kei Cho

King's College London

Research Overview

Prof. Kei Cho (Synapse & Drug Discovery Laboratory, Department of Basic and Clinical Neuroscience, King's College London) has considerable expertise in both the fields of neurophysiology and pathophysiology in the hippocampal neuron. Over the last 20 years, Cho has identified several potential mechanistic targets for curtailing synapse weakening. The majority of tauopathy research has mostly focused on the consequences of how misfolded tau results in mislocalisation and neurotoxicity. However, emerging studies show that tau phosphorylation (pTau) has both physiological and pathophysiological roles within the neuron. Cho began to formalize the importance of the caspase-Akt-1-GSK-3β cascade in synapse weakening. Cho is interested in the consequence of AMPA receptor endocytosis with respect to the functional and structural modification of the synapse. Subsequently, Cho identified the role of the microtubule associated protein tau in long-term synaptic plasticity and continues to determine how aberrant synaptic plasticity is expressed and what the physiological and pathological consequences might be. Cho's lab has shown that a tau interacting molecule; protein kinase C and casein kinase substrate in neurons protein 1 (PACSIN1) plays a key role in synapse modification. Very recently, Cho Lab was able to discover previously unrecognised disruption of local translation by neurodegeneration-associated mutant proteins FUS and Tau. Together, we are currently expanding that line of research to build a detailed understanding of how the long-range structures in 3'UTRs of locally translated mRNAs mediate the control of local translation, and how they affect the impact of other disease-causing mutations. Collectively, this multidisciplinary approach will offer a highly sensitive readout of synapse health and can detect the earliest signs of synaptic dysfunction and identify the pathophysiology required for disease progression. Providing the cornerstone to ground-breaking discoveries in the early stage of synapse pathophysiology, "why are some synapses vulnerable to neurodegenerative disorders whereas others are resilient?"

Biography

Uroosa Chugtai

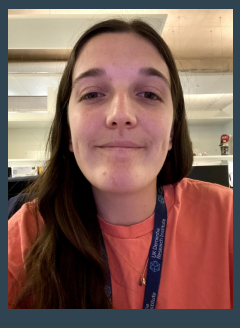
PhD Student at Cardiff University

Research Overview

I am a final year PhD student on the Wellcome Trust Integrative Neuroscience PhD programme at Cardiff University, based in the lab of Dr Owen Peters in the Dementia Research Institute. My PhD research aims to understand the cellular and molecular mechanisms by which loss-of-function mutations in TANK-binding kinase 1 (TBK1) lead to related neurodegenerative diseases amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD), with a particular emphasis on microglia. To this end, I use a combination of pharmacological TBK1 kinase inhibition and CRISPR/Cas9 gene-editing to model TBK1 loss-of-function in microglial cell lines (immortalised and human pluripotent stem cell-derived), followed by unbiased quantitative proteomics and targeted functional phenotyping.

Prior to this, I received a BSc in Biomedical Science from Imperial College London and worked as a research assistant with Professor Wade-Martins at the University of Oxford. My research interests revolve around investigating the cellular and molecular mechanisms of neurodegeneration, specifically using advanced in vitro models of disease, with the ultimate aim of identifying dysfunctional processes that can be translated to patient care.

Biography



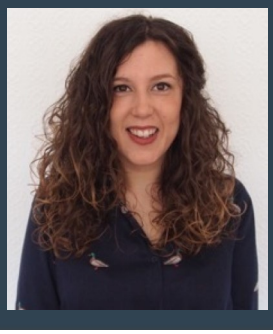
Hannah Clarke

PhD Student – Smith Lab, Cardiff University Dementia Research Institute

Research Overview

My PhD project focusses on the role of the Alzheimer's Disease risk gene WW domain containing oxidoreductase (WWOX) using Drosophila melanogaster as a model system. We are very interested in how Wwox may manipulate amyloid pathology and have taken a variety of behavioural and omic analyses to answer this question. Via transcriptomic and metabolomic analysis, we have discovered that Wwox knockdown and overexpression in the presence of human amyloid may modulate Drosophila metabolism. Specifically, levels of lactate dehydrogenase and lactate are increased when Wwox levels are reduced in neuronal cells also expressing amyloid. We are now working on deciphering the mechanism behind this change, to further understand the role of Wwox in neurodegenerative disease.

Biography



Dr Natalie Connor-Robson

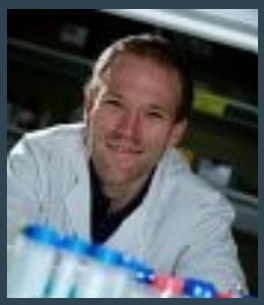
Alzheimer's Research UK Research Fellow, UK Dementia Research Institute Emerging Leader.

Research Overview

Alzheimer's disease (AD) has huge personal and economic costs and as yet there are no effective treatments. The majority, more than 99%, of AD cases are considered to have no clear underlying cause from a single gene and are known as 'sporadic'. However, 60-80% of risk for developing sporadic late onset AD (LOAD) is known to be genetic. The lack of mechanistic understanding of LOAD genetics is the major bottle neck to producing new and effective AD therapies.

Much recent work has highlighted the importance of the endocytic pathway, a cellular mechanism important to recycling and maintenance of normal function, in neurodegeneration. A high proportion of the risk genes for LOAD cluster in the endocytic pathway and enlarged early endosomes are one of the earliest pathological features of the disease. My work focuses on understanding how these genes associated to both LOAD and the endocytic pathway contribute to the earliest molecular and cellular changes which cause the disease. In my lab we have developed a number of novel iPSC models using CRISPR/Cas9 technology and through identifying individuals with high endocytic polygenic risk to make iPSC lines from. We use these lines to study disease mechanisms in neurons and microglia two important cell types in LOAD which use the endocytic pathway in highly specialised ways.

Biography



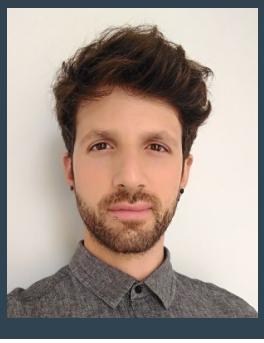
Dr Johnathan Cooper-Knock

Consultant Senior Lecturer, University of Sheffield

Research Overview

Amyotrophic lateral sclerosis (ALS) is an urgent health need and an archetypal complex disease determined by the interaction of multiple genetic and environmental factors. The central aim of our approach is to achieve personalized medicine for ALS through subdivision of patients according to their own specific genetic and environmental disease-determinants. We were the first to describe non-coding genetic risk factors for ALS in a significant proportion of patients (Cooper-Knock et al 2020, Cell Reports); moreover, we have recently increased the number of ALS risk genes by an order of magnitude through new machine-learning analysis (Zhang, Cooper-Knock et al 2022, Neuron). We have pioneered the application of Mendelian randomization (MR) for discovery of environmental risk factors for ALS including the development of new methodology (Julian et al 2021, EBioMed; Boddy et al, 2022, Brain Communications).

Biography



Dr Aurelio Cortese

Computational Neuroscience Laboratories, ATR Institute International (Kyoto, Japan)

Research Overview

My long-term research agenda is to understand the nature of flexible and adaptive behaviours in humans and artificial agents. Specifically, my group investigates the interplay between higher order cognitive functions and reinforcement learning processes, how uncertainty is represented in the brain and what role it plays in decision-making, the computational properties of metacognition and reasoning, and how these functions may be affected by psychiatric disorders. We study these questions in healthy participants as well as in clinical populations. We routinely use neuroimaging (fMRI), particularly in the form of closed-loop experiments, with a combination of behavioural tasks, computational modelling, and machine learning. Our main technical contribution has been the development of the decoded neurofeedback method, an experimental technique whereby brain activity patterns are decoded in real time with machine learning and fed back to participants to lead brain activity to a target state.

Selected Publications

1. Taschereau-Dumouchel V, Cortese A, Lau H, Kawato M. (2021) Conducting Decoded Neurofeedback Studies. Social Cognitive and Affective Neuroscience 16 (8): 838–48
2. Cortese A, Yamamoto A, Hashemzadeh M, Sepulveda P, Kawato K, De Martino B. (2021) Value Signals Guide Abstraction during Learning. eLife 10:e68943
3. Cortese A, Tanaka SC, Amano K, Koizumi A, Lau H, Sasaki Y, Shibata K, Taschereau-Dumouchel V, Watanabe T, Kawato M. (2021) The DecNef Collection, fMRI Data from Closed-Loop Decoded Neurofeedback Experiments. Scientific Data 8 (1): 65
4. Cortese A, Lau H, Kawato M. (2020) Unconscious Reinforcement Learning of Hidden Brain States Supported by Confidence. Nature Communications 11 (1): 4429
5. Cortese A*, Amano K*, Koizumi A*, Kawato M, Lau H. (2016) Multivoxel Neurofeedback Selectively Modulates Confidence without Changing Perceptual Performance. Nature Communications 7 (12): 13669
6. Koizumi A*, Amano K*, Cortese A*, Shibata K, Yoshida W, Seymour B, Kawato M, Lau H. (2016) Fear Reduction without Fear through Reinforcement of Neural Activity That Bypasses Conscious Exposure. Nat Hum Behav 1 (1): 0006

Biography

Dr Dezerae Cox

Lady Edith Wolfson Junior Non-Clinical Fellow, UK Dementia Research Institute, University of Cambridge

Research Overview

My research seeks to uncover the molecular drivers of neurodegenerative disease, with a current focus on Motor Neuron Disease (MND). I have developed a suite of tools spanning whole-proteome 'omics to single-molecule microscopy that allow us to characterise protein aggregates at scale in unprecedented detail. Using these technologies, I have explored fundamental questions of protein homeostasis [1, 2], characterised cellular models of aggregation-associated disorders [3,4], and discovered aggregate features associated with neurodegenerative disease [5]. I am now leveraging these powerful technologies to investigate the accuracy of cellular models of MND, highlighting models of most use in therapeutic testing and uncovering novel mechanisms underpinning disease.

Selected Publications

1. Cox, D, Ang, CS, Nillegoda, NB, Reid, GE, and Hatters, DM. (2022) “Hidden Information on Protein Function in Censuses of Proteome Foldedness.” Nature Communications DOI: 10.1038/s41467-022-29661-2.
2. ^Cox, D, Ormsby, AR, Reid, GE, and ^Hatters, DM. (2022) “Protein Painting Reveals Pervasive Remodeling of Conserved Proteostasis Machinery in Response to Pharmacological Stimuli.” NPJ Systems Biology and Applications DOI: 10.1038/s41540-022-00256-3.
3. *Ruff, KM, *Choi YH, Cox, D, Ormsby, AR, Myung, Y, Ascher, DB, Radford, SE, Pappu, RV, and Hatters, DM. (2022) “Sequence Grammar Underlying the Unfolding and Phase Separation of Globular Proteins.” Molecular Cell DOI: 10.1016/j.molcel.2022.06.024. *Equal contributions
4. Radwan, M, Ang, CS, Ormsby, AR, Cox, D, Daly, JC, Reid, GE, and Hatters, DM. (2020) “Arginine Valency in C9ORF72 Dipolypeptides Mediates Promiscuous Proteome Binding That Stalls Ribosomes, Disable Actin Cytoskeleton Assembly and Impairs Arginine Methylation of Endogenous Proteins.” Molecular & Cellular Proteomics DOI: 10.1101/749127.
5. Böken, D, Cox, D, Burke, M, Lam, JYL, Katsinelos, T, Danial, JSH, McEwan, WA, Rowe, JB, and Klenerman, D. (2023) “Characterization and Super-Resolution Imaging of Small Tau Aggregates in Human Samples.” bioRxiv DOI: 10.1101/2023.06.12.544575.

Biography

Leon Crowley

PhD Student at King's College London

Research Overview

My research focuses on the role of TDP-43 in amyotrophic lateral sclerosis and frontotemporal dementia (ALS-FTD). Mutations in TARDBP, the gene encoding TDP-43, indicate a fundamental role for TDP-43 in ALS-FTD pathogenesis. In my PhD, I am using CRISPR-Cas9 with iPSCs to introduce single nucleotide polymorphisms in UNC13A, a splicing target of TDP-43. UNC13A is one of the top GWAS hits for ALS-FTD and I am interested in exploring the polygenic effects of these UNC13A risk variants together with the M337V TARDBP mutation. Additionally, I will be using Visium spatial transcriptomics in the TARDBPQ331K knock-in mouse model of ALS/FTD, previously established here in the Sreedharan lab, to explore regional vulnerability and cell-type specific molecular changes.

Biography

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Vale Resort, Pontyclun,
31 August – 2 September 2023

Professor Kevin Fox

Professor of Neuroscience, Cardiff University

Research Overview

Our lab is interested in synaptic plasticity, learning and memory. Early work centred on factors involved in development and experience-dependent plasticity of the cerebral cortex. We showed that cortical NMDA receptors were regulated during development by experience, but that they were involved in normal synaptic transmission and slow -wave sleep, not just plasticity. However, auto-phosphorylation of αCaMKI is a specialised enough process to probe plasticity. We were able to show it was critical for experience-dependent potentiation in vivo, neocortical LTP and at least two components of the structural plasticity of dendritic spines (1). More recently, our lab has discovered a diversity of plasticity subtypes within the cortex. For example, two types of layer 5 cortical neurone (the “Regular Spiking” and “Intrinsic Bursting” neurones) exhibit very different combinations of Hebbian and Homeostatic plasticity (2). Our most recent work concerns the plasticity underlying learning. We have characterised the structural plasticity of dendritic spines that results from learning a texture discrimination task and described interactions between cortical areas that appears to gate that plasticity (3). Some of our recent work will be presented in the poster session on day 2.

Selected Publications

1. Seaton, Gillian, Gladys Hodges, Annelies de Haan, Aneesha Grewal, Anurag Pandey, Haruo Kasai, Kevin Fox Dual-Component Structural Plasticity Mediated by αCaMKII Autophosphorylation on Basal Dendrites of Cortical Layer 2/3 Neurones. Journal of Neuroscience 11 March 2020, 40 (11) 2228-2245; DOI: 10.1523/JNEUROSCI.2297-19.202.
2. Pandey, Anurag,1 Neil Hardingham,1 and Kevin Fox1,2,* (2022) Differentiation of Hebbian and homeostatic plasticity mechanisms within layer 5 visual cortex neurons Cell Reports 39 (9) doi.org/10.1016/j.celrep.2022.110892.
3. Pandey, Anurag, Sungmin Kang, Nicole Pacchiarini, Hanna Wyszynska, Aneesha Grewal, Alex M Griffiths, Imogen M Healy-Millett, Zena Masseri, Neil R Hardingham, Joseph O'Neill, Robert C Honey, Kevin Fox (2023) Interdependence of primary and secondary somatosensory cortices for plasticity and texture discrimination learning biorxiv 2023.04. 25.538217.

Biography

Chiara Franceschi – PhD student

School of Psychology, Cardiff University

Research overview

My current research focuses on understanding the involvement of the subiculum in spatial long-term memory, using a radial-arm maze task. Additionally, my project investigates the electrophysiological characteristics of this area during the task, for which we have developed a technique to allow chronic recordings using Neuropixels 1.0 probes in the rat. The subiculum is an area that has been found to be impaired in dementia patients, and understanding its role in spatial memory and the characteristics of its neuronal population can help improve our knowledge on its involvement in the aforementioned brain disorder.

Biography

Symposiwm Niwrowyddoniaeth DU-Japan
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UK-Japan Neuroscience Symposium
Vale Resort, Pontyclun,
31 August – 2 September 2023

Dr Akihiro Funamizu

Lecture (PI) @ Institute for Quantitative Biosciences (IQB), the University of Tokyo

https://www.iqb.u-tokyo.ac.jp/lab/funamizu/

Research Overview

Our lab is interested in understanding the neural mechanism of decision making. We particularly focus on the recent advances in machine learning (i.e., artificial intelligence: AI) and compare the algorithms of AI and brain. Our research targets (i) a Bayesian computation in the cerebral cortex and (ii) a modeling of mouse choice behavior with artificial neural networks.

Selected Publications

1. Ishizu K, Nishimoto S, Funamizu A, “Localized and global computation for integrating prior value and sensory evidence in the mouse cerebral cortex” bioRxiv, 2023 doi: https://doi.org/10.1101/2023.06.06.543645
2. Funamizu A, Marbach F, Zador AM, “Stable sound decoding despite modulated sound representation in the auditory cortex” bioRxiv, 2023 doi: https://doi.org/10.1101/2023.01.31.526457
3. Funamizu A, “Integration of sensory evidence and reward expectation in mouse perceptual decision-making task with various sensory uncertainties” Iscience, 2021 doi: 10.1016/j.isci.2021.102826
4. Funamizu A, Kuhn B, Doya K, “Neural substrate of dynamic Bayesian inference in the cerebral cortex” Nat Neurosci, 2016 doi: 10.1038/nn.4390.

Professor Tomoyuki Furuyashiki

Professor and Chair, Division of Pharmacology, Graduate School of Medicine, Kobe University

Research Overview

Our research focuses on the biological, particularly neuroimmune, mechanisms of psychosocial stress and depression. Using a mouse model of repeated social defeat stress, we discovered that acute stress induces dendritic growth of prefrontal neurons via the dopamine D1 receptor, enhancing stress resilience, whereas chronic stress attenuates the prefrontal dopaminergic response and induces dendritic atrophy of prefrontal neurons via microglia-derived inflammatory mediators, leading to depression-related behaviors. We also found that chronic stress mobilizes bone marrow-derived leukocytes, which critically contribute to the behavioral changes. Using multi-omics analyses, whole-brain imaging, and genetic manipulations, we are now investigating the molecular and cellular processes of these neuroimmune mechanisms and their impact on neural circuit function.

Selected Publications

1. Ishikawa Y, Kitaoka S, Kawano Y, Ishii S, Suzuki T, Wakahashi K, Kato T, Katayama Y, Furuyashiki T (2021). Repeated social defeat stress induces neutrophil mobilization in mice: maintenance after cessation of stress and strain-dependent difference in response. Br J Pharmacol 178, 827-844. 10.1111/bph.15203
2. Nie X, Kitaoka S, Tanaka K, Segi-Nishida E, Imoto Y, Ogawa A, Nakano F, Tomohiro A, Nakayama K, Taniguchi M, Mimori-Kiyosue Y, Kakizuka A, Narumiya S, Furuyashiki T (2018). The innate immune receptors TLR2/4 mediate repeated social defeat stress-induced social avoidance through prefrontal microglial activation. Neuron 99, 464-479. 10.1016/j.neuron.2018.06.035
3. Shinohara R, Taniguchi M, Ehrlich AT, Yokogawa K, Deguchi Y, Cherasse Y, Lazarus M, Urade Y, Ogawa A, Kitaoka S, Sawa A, Narumiya S, Furuyashiki T (2018). Dopamine D1 receptor subtype mediates acute stress-induced dendritic growth in excitatory neurons of the medial prefrontal cortex and contributes to suppression of stress susceptibility in mice. Mol Psychiatry 23, 1717-1730. 10.1038/mp.2017.177
4. Tanaka K#, Furuyashiki T#†, Kitaoka S#, Senzai Y#, Imoto Y, Segi-Nishida E, Deguchi Y, Breyer RM, Breyer MD, Narumiya S† (2012). Prostaglandin E2-mediated attenuation of mesocortical dopaminergic pathway is critical for susceptibility to repeated social defeat stress in mice. J Neurosci 32, 4319-4329. 10.1523/JNEUROSCI.5952-11.2012 (#equally contributed, †co-corresponding author)
5. Furuyashiki T†, Narumiya S (2011). Stress responses: the contribution of prostaglandin E2 and its receptors. Nat Rev Endocrinol 7, 163-175. 10.1038/nrendo.2010.194. (†corresponding author)

Dr Jenna Gregory

Senior Clinical Lecturer at the University of Aberdeen

Research Overview

Dr Jenna Gregory is a Consultant Pathologist and Principal Investigator at the University of Aberdeen. Dr Gregory's research focuses on the molecular mechanisms underlying neurodegenerative diseases with a particular focus on Amyotrophic Lateral Sclerosis (ALS). The work in her lab involves studying patient samples (tissue and biofluids) for molecular differences that could explain why people with ALS have such diverse symptoms, including differences in disease progression and cognitive involvement. Her work is funded by the NIH, Target ALS and MND Scotland and her lab recently discovered that markers of ALS can be detected in the gut years before motor symptoms begin. Example research outputs listed below show how the Gregory lab make use of cutting edge molecular and digital pathology techniques to probe deeply clinically phenotyped human tissues. The aim of her lab's work is to identify targets that could be used for early diagnosis or to monitor disease progression, or ultimately, for therapies to improve the outlook for people with ALS.

Selected Publications

1. Rifai OM, O'Shaughnessy J, Dando OR, Munro AF, Sewell MDE, Abrahams S, Waldron FM, Sibley CR, Gregory JM. Distinct neuroinflammatory signatures exist across genetic and sporadic amyotrophic lateral sclerosis cohorts. Brain 2023.

2. Pattle SB, O'Shaughnessy J, Kantelberg O, Rifai OM, Pate J, Nellany K, Hays N, Arends MJ, Horrocks MH, Waldron FM, Gregory JM. pTDP-43 aggregates accumulate in non-central nervous system tissues prior to symptom onset in amyotrophic lateral sclerosis: a case series linking archival surgical biopsies with clinical phenotypic data. Journal of Pathology: Clinical Research 2023; 9: 44-55.

3. Rifai OM, Longden J, O'Shaughnessy J, Sewell MDE, Pate J, McDade K, Daniels MJD, Abrahams S, Chandran S, McColl BW, Sibley CR, Gregory JM. Random forest modelling demonstrates microglial and protein misfolding features to be key phenotypic markers in C9orf72-ALS. Journal of Pathology 2022.

4. Zacco E, Kantelberg O, Milanetti E, Armaos A, Panei FP, Gregory JM, Jeacock K, Chandran S, Clarke D, Ruocco G, Gustincich S, Horrocks M, Pastore A, Tartaglia GG. Probing TDP-43 condensation using an in silico designed aptamer. Nature Communications 2022.

Professor Jeremy Hall

Director of Psychological Medicine and Clinical Neurosciences, Cardiff University

Research Overview

My overarching interest is in the role of genetic and environmental risk factors in the development of neurodevelopmental disorders such as schizophrenia, autism and related personality disorders. In my work I employ a translational approach to study how genetic and environmental factors enhance risk for mental illness. I am particularly interested in how identified genetic risk factors affect learning processes in the brain; abnormalities in which underlie the key symptoms seen in a range of mental health problems. Overall I believe that understanding how genetic risk factors influence the brain and how these responses are modulated by environmental stimuli is crucial to the development of new treatments for psychiatric illness. In addition to my pre-clinical work I also conduct clinical work and research in the fields of adult neurodevelopmental disorders and early psychosis.

Current Research Projects

Role of psychiatric risk genes in learning and memory

Expression and regulation of autism and schizophrenia associated genes

Modulatory effects of early life experience on gene expression and psychiatric risk

Genetic effects on brain function and structure

Selected Publications

1. Kopal, J. et al. 2023. Rare CNVs and phenome-wide profiling highlight brain structural divergence and phenotypical convergence. Nature Human Behaviour 7, pp. 1001-1007. (10.1038/s41562-023-01541-9)
2. Chawner, S. J. et al. 2023. Neurodevelopmental dimensional assessment of young children at high genomic risk of neuropsychiatric conditions. JCPP Advances 3(2), article number: e12162. (10.1002/jcv2.12162)
3. Dec, K., Alsaqati, M., Morgan, J., Deshpande, S., Wood, J., Hall, J. and Harwood, A. J. 2023. A high ratio of linoleic acid (n-6 PUFA) to alpha-linolenic acid (n-3 PUFA) adversely affects early stage of human neuronal differentiation and electrophysiological activity of glutamatergic neurons in vitro. Frontiers in Cell and Developmental Biology 11, article number: 1166808. (10.3389/fcell.2023.1166808)
4. Moreau, C. A. et al. 2023. Brain functional connectivity mirrors genetic pleiotropy in psychiatric conditions. Brain 146(4), pp. 1686-1696. (10.1093/brain/awac315)
5. Gasalla Canto, P., Manahan-Vaughan, D., Dwyer, D. M., Hall, J. and Méndez-Couz, M. 2023. Characterisation of the neural basis underlying appetitive extinction & renewal in a Cacna1c rats. Neuropharmacology 227, article number: 109444. (10.1016/j.neuropharm.2023.109444)
6. Lynham, A. J. et al. 2023. DRAGON-Data: A platform and protocol for integrating genomic and phenotypic data across large psychiatric cohorts. BJPsych Open 9(2), article number: e32. (10.1192/bjo.2022.636)

Biography

Senior Associate Professor Taku Hatano

Department of Neurology, Juntendo University Faculty of Medicine

Research Overview

My research focuses on unraveling the pathomechanisms of Parkinson's disease and developing biomarkers of synucleinopathy. Specifically, I am interested in the aggregation mechanism of α-synuclein. Our team created a Drosophila model of PLA2G6 (PARK14) and found abnormal α-synuclein aggregation due to phospholipid metabolism abnormalities. Additionally, our team discovered that mutations in the D domain of prosaposin, which is crucial for glycolipid metabolism, can cause familial Parkinson's disease.

Furthermore, we conducted blood metabolomics in patients with Parkinson's disease and PARK2 as potential biomarkers, revealing decreased caffeine levels and increased oxidative stress markers. Recently, our team identified α-synuclein seeds in serum, discovering their potential usefulness in differentiating synucleinopathies.

Selected publications

1. Okuzumi A, Hatano T, Matsumoto G, et al. Propagative α-synuclein seeds as serum biomarkers for synucleinopathies. Nat Med. 29:1448-1455 (2023)
2. Oji Y, Hatano T, Ueno SI, et al. Variants in saposin D domain of prosaposin gene linked to Parkinson's disease. Brain. 143:1190-1205 (2020)
3. Mori A, Hatano T, Inoshita T, et. al. Parkinson's disease-associated iPLA2-VIA/PLA2G6 regulates neuronal functions and α-synuclein stability through membrane remodeling. Proc Natl Acad Sci U S A. 116:20689-20699 (2019)
4. Okuzumi A#, Hatano T#*, Ueno SI, et al. Metabolomics-based identification of metabolic alterations in PARK2. Ann Clin Transl Neurol. 6:525-536, (2019) #equally contribution, *Corresponding Author.
5. Hatano T, Saiki S, Okuzumi A, et al. Identification of novel biomarkers for Parkinson's disease by metabolomics technologies. J Neurol Neurosurg Psychiatry. 87:295-301 (2016)

Biography

Symposiwm Niwrowyddoniaeth DU-Japan
Vale Resort, Pont-y-clun,
31 Awst – 2 Medi 2023

UK-Japan Neuroscience Symposium
Vale Resort, Pontyclun,
31 August – 2 September 2023

Dr Jennifer Imm

Post Doctoral Research Associate at the University of Exeter

Biography

My research focuses on investigating the links between DNA methylation with neuropathology and clinical diagnosis in the Lewy body dementias. To do this we have generated a cohort of 921 post-mortem brain samples from over 450 different individuals. In this cohort we have both pre-frontal cortex and anterior cingulate gyrus tissue and have so far have used the Illumina EPIC array to assess bulk methylation changes. We have identified significant changes in DNA methylation associated with both clinical diagnosis and neuropathology, including genes that have been previously associated with synucleinopathies, including PTPRN2, DGKI and SYN3. Currently we are in the process of using fluorescence activated nuclei sorting to look at the cell type specificity of these changes. I also have experience working with induced pluripotent stem cells (iPSCs) and iPSC-derived neurons as throughout my PhD I used them to model Alzheimer's disease related exposures such as differentiation and maturation, drug treatment and immune challenge.

Selected Publications

1. Imm J., Pishva E., Ali M., Kerrigan T.L., Jeffries A., Burrage J., Glaab E., Cope E.L, Jones K.M., Allen N.D., and Lunnon K., 2021. Characterization of DNA methylomic signatures in induced pluripotent stem cells during neuronal differentiation. Frontiers in Cell and Developmental Biology. eCollection 2021.
2. Steg L., Shireby G., Imm J., et al, 2021. Novel epigenetic clock for fetal brain development predicts prenatal age for cellular stem cell models and derived neurons. Molecular Brain. 14(1):98.
3. Thei L., Imm J., Kaisis E., Dallas M., and Kerrigan T.L., 2018. Microglia in Alzheimer’s disease: a role for ion channels. Frontiers in Neuroscience, vol. 12.
4. Crawford B., Craig Z., Mansell G., White I., Smith A., Spaull S., Imm J., Hannon E., Wood A., Yaghootkar H., Ji Y., Mullins N., Lewis C.M., Mill J., and Murphy T., 2018. DNA methylation and inflammation marker profiles associated with a history of depression. Human Molecular Genetics, vol. 27(16).
5. Imm J., Kerrigan T.L., Jeffries A., and Lunnon K., 2017. Using induced pluripotent stem cells to explore genetic and epigenetic variation associated with Alzheimer’s disease. Epigenomics, vol 9., pp. 1455-1468.
6. Tieu K., and Imm J., 2014. Mitochondrial dynamics as a potential therapeutic target for Parkinson’s disease? Advances in Clinical Neuroscience and Rehabilitation, vol. 4(1) pp:6-8.

Professor Haruhisa Inoue

Center for iPS Cell Research and Application (CiRA), Kyoto University, RIKEN

Research Overview

My research focuses on the basic biology of neurodegeneration including amyotrophic lateral sclerosis (ALS). We modeled ALS by reprogramming technology, and found that the Src/c-Abl pathway is a potential therapeutic target in ALS. We further conducted a phase I dose escalation study of bosutinib, a Src/c-Abl inhibitor, for ALS patients (induced pluripotent stem cell–based Drug Repurposing for ALS Medicine: iDReAM) to evaluate the safety and tolerability of bosutinib in ALS patients as well as to assess its efficacy in an exploratory manner.

Selected Publications

1. Egawa, N., Izumi, Y., Suzuki, H., Tsuge, I., Fujita, K., Shimano, H., Izumikawa, K., Takahashi, N., Tsukita, K., Enami, T., Nakamura, M., Watanabe, A., Naitoh, M., Suzuki, S., Seki, T., Kobayashi, K., Tida, T., Kaji, R., Takahashi, R., Inoue, H. (2022). TDP-43 regulates cholesterol biosynthesis by inhibiting sterol regulatory element-binding protein 2. Scientific Reports 12:7988.

2. Imamura, K., Izumi, Y., Nagai, M., Nishiyama, K., Watanabe, Y., Hanajima, R., Egawa, N., Ayaki, T., Oki, R., Fujita, K., Uozumi, R., Morinaga, A., Hirohashi, T., Fujii, Y., Yamamoto, T., Tatebe, H., Tokuda, T., Takahashi, N., Morita, S., Takahashi, R., Inoue, H. (2022). Safety and tolerability of bosutinib in patients with amyotrophic lateral sclerosis (iDReAM study): A multicentre, open-label, dose-escalation phase 1 trial. eClinical Medicine 53,101707.

3. Imamura, K., Izumi, Y., Banno, H., Uozumi, R., Morita, S., Egawa, N., Ayaki, T., Nagai, M., Nishiyama, K., Watanabe, Y., Hanajima, R., Oki, R., Fujita, K., Takahashi, N., Ikeda, T., Shimizu, A., Morinaga, A., Hirohashi, T., Fujii, Y., Takahashi, R., Inoue, H. (2019). Induced pluripotent stem cell-based Drug Repurposing for Amyotrophic lateral sclerosis Medicine (iDReAM) study: Protocol for a phase 1 dose escalation study of bosutinib for amyotrophic lateral sclerosis patients. BMJ Open 9(12): e033131.

4. Imamura, K., Izumi, Y., Watanabe, A., Tsukita, K., Woltjen, K., Yamamoto, T., Hotta, A., Kondo, T., Kitaoka, S., Ohta, A., Tanaka, A., Watanabe, D., Morita, M., Takuma, H., Tamaoka, A., Kunath, T., Wray, S., Furuya, H., Era, T., Makioka, K., Okamoto, K., Fujisawa, T., Nishitoh, H., Homma, K., Ichijo, H., Julien, JP., Obata, N., Masato, H., Akiyama, H., Kaneko, S., Ayaki, T., Ito, H., Kaji, R., Takahashi, R., Yamanaka, S., Inoue,

5. H. (2017). The Src/c-Abl pathway is a potential therapeutic target in amyotrophic lateral sclerosis. Science Translational Medicine 9(391): eaaf3962.

6. Egawa, N., Kitaoka, S., Tsukita, K., Naitoh, M., Takahashi, K., Yamamoto, T., Adachi, F., Kondo, T., Okita, K., Asaka, I., Aoi, T., Watanabe, A., Yamada, Y., Morizane, A.,

7. Takahashi, J., Ayaki, T., Ito, H., Yoshikawa, K., Yamawaki, S., Suzuki, S., Watanabe, D., Hioki, H., Kaneko, T., Makioka, K., Okamoto, K., Takuma, H., Tamaoka, A., Hasegawa, K., Nonaka, T., Hasegawa, M., Kawata, A., Yoshida, M., Nakahata, T., Takahashi, R., Marchetto, MC., Gage, FH., Yamanaka, S., Inoue, H. (2012). Drug Screening for ALS Using Patient-Specific Induced Pluripotent Stem Cells. Science Translational Medicine 4 (145):145ra104.

Biography

Daisuke Ito M.D., PhD.

Clinical Fellow, Nagoya University Graduate School of Medicine, Department of Neurology

Research Overview

My research focuses on clinical biomarkers of motor neuron disease, especially amyotrophic lateral sclerosis. Our research strategy is to explore clinical findings on preclinical disease models in reverse translational method. I have reported the prodromal clinical elevation of serum creatine kinase with reverse translational confirmation using SOD1G93A transgenic mice. Now I am focusing on metabolic changes of ALS and their relationship with disease progression. In this study, we identified the alteration of serum endocannabinoids, and their elevations were related to rapid progression of ALS. FAAH inhibitor which increases the level of endocannabinoids including N-acyl taurines ameliorated the cell models and a murine model of ALS and we revealed the effects of a FAAH inhibitor in SOD1G93A transgenic mice using RNA-Seq and scRNA-Seq of murine spinal cords.

Selected Publications

1. Torii R, Hashizume A, Yamada S, Ito D, Kishimoto Y, Moriyoshi H, Inagaki T, Nakamura R, Nakamura T, Naoi T, Morita M, Katsuno M. Clinical features of female carriers and prodromal male patients with spinal and bulbar muscular atrophy. Neurology in press.
2. Kishimoto Y, Hashizume A, Imai Y, Nakatochi M, Yamada S, Ito D, Torii R, Nagano Y, Fujimoto H, Katsuno M. Quantitative evaluation of upper limb ataxia in spinocerebellar ataxias. Ann Clin Transl Neurol. 9(4):529-539. 2022
3. Ikenaka K, Maeda Y, Hotta Y, Nagano S, Yamada S, Ito D, Torii R, Kakuda K, Tatebe H, Atsuta N, Aguirre C, Kimura Y, Baba K, Tokuda T, Katsuno M, Kimura K, Sobue G, Mochizuki H. Serum asymmetric dimethylarginine level correlates with the progression and prognosis of amyotrophic lateral sclerosis. Eur J Neurol. 29(5):1410-1416. 2022
4. Yamada S, Hashizume A, Hijikata Y, Ito D, Kishimoto Y, Iida M, Koike H, Hirakawa A, Katsuno M. Ratio of urinary N-terminal titin fragment to urinary creatinine is a novel biomarker for amyotrophic lateral sclerosis. J Neurol Neurosurg Psychiatry. 92(10):1072-1079. 2021
5. Ito D, Hashizume A, Hijikata Y, Yamada S, Iguchi Y, Iida M, Kishimoto Y, Moriyoshi H, Hirakawa A, Katsuno M. Elevated serum creatine kinase in the early stage of sporadic amyotrophic lateral sclerosis. J Neurol. 266 (12):2952-2961.2019.
6. Hijikata Y, Hashizume A, Yamada S, Inagaki T, Ito D, Hirakawa A, Suzuki K, Atsuta N, Tsuboi T, Hattori M, Hori A, Banno H, Sobue G, Katsuno M. Biomarker-based analysis of preclinical progression in spinal and bulbar muscular atrophy. Neurology. 24;90(17):e1501-e1509. 2018.

Biography

Aya Ito-Ishida

RIKEN Center for Brain Science

Research Overview

An infant's brain undergoes dynamic changes and eventually matures into a fully developed circuitry. My research focuses on the biological mechanisms that regulate this process by addressing how synaptic connections are formed and disrupted in developmental disorders. My previous works revealed that Cbln1 is critical for synapse formation in the cerebellum. By studying the mouse models of Rett syndrome, my work identified the roles of inhibitory neuron subtypes and heterochromatin structural alteration in neurological deficit. More recently, my lab has been studying the developmental process of cortical connectivity and possible impairment in Rett syndrome.

Selected publications

1. Ito-Ishida A, Baker SA, Sillitoe RV, Sun Y, Zhou J, Ono Y, Iwakiri J, Yuzaki M, Zoghbi HY. “MeCP2 Levels Regulate the 3D Structure of Heterochromatic Foci in Mouse Neurons.” Journal of Neuroscience. 40(45), 8746-8766. (2020)
2. Ito-Ishida A, Yamalanchili HK, Shao Y, Baker SA, Heckman LD, Lavery LA, Kim J, Lombardi LM, Sun Y, Liu Z, Zoghbi HY. “Genome Wide Distribution of Linker Histone H1.0 is Independent of MeCP2.” Nature Neuroscience. 21(6), 794-798. (2018)
3. Ito-Ishida A, Ure K, Chen H, Swann JW, Zoghbi HY. “Loss of MeCP2 in parvalbumin-and somatostatin-expressing neurons in mice leads to distinct Rett Syndrome-like phenotypes.” Neuron. 88(4), 651-8. (2015).
4. Ito-Ishida, A, Miyazaki, T., Miura, E., Matsuda, K., Watanabe, M., Yuzaki, M. and Okabe, S. “Presynaptically released Cbln1 induces dynamic axonal structural changes by interacting with GluD2 during cerebellar synapse formation.” Neuron 76(3), 549-564. (2012)
5. Ito-Ishida, A, Miura, E., Emi, K., Matsuda, K., Iijima, T., Kondo, T., Kohda, K., Watanabe, M. and Yuzaki, M. “Cbln1 regulates rapid formation and maintenance of excitatory synapses in mature cerebellar Purkinje cells in vitro and in vivo.” Journal of Neuroscience. 28(23): 5920-5930. (2008)

Biography

Symposiwm Niwrowyddoniaeth DU-Japan
Vale Resort, Pont-y-clun,
31 Awst – 2 Medi 2023

UK-Japan Neuroscience Symposium
Vale Resort, Pontyclun,
31 August – 2 September 2023

Matt Jones

Professor of Neuroscience, University of Bristol

Research Overview

I trained as a neuroscientist at the University of Cambridge, the UK National Institute for Medical Research and the Massachusetts Institute of Technology before establishing my research team at the University of Bristol. My lab strives to understand how distributed neural networks spanning hippocampus, striatum and prefrontal cortex process and store information, and how this processing becomes impaired in neuropsychiatric disorders. To do this, we record and modulate brain activity (with Neuropixels in rodents and EEG in humans), then apply computational modelling and analyses to try and decode the terabytes. Current projects include analyses of sleep's contributions to cognition, the diagnostic and translational utility of sleep neurophysiology and the circuit architecture of psychedelic drug action.

Selected Publications

1. Domanski A.P.F., Kucewicz M.T., Russo E., Tricklebank M.D., Robinson E.S., Durstewitz D. and Jones M.W. (2023) Prefrontal cortical contributions to working memory loading, maintenance and recall are parsed by hippocampal-prefrontal oscillatory assembly dynamics. bioRxiv https://doi.org/10.1101/2021.12.20.473436 (in press, Current Biology).
2. Donnelly N.A.*, Bartsch U.*, Moulding H.A., Eaton C., Marston H., Hall J.E., Hall J., Owen M.J., van den Bree M.B.M. and Jones M.W. (2022) NREM sleep signatures of memory disruption and psychiatric symptoms in young people with 22q11.2 deletion syndrome eLife, https://doi.org/10.7554/eLife.75482.
3. Kersanté F., Purple R.J. and Jones M.W. (2022) The GABA-A receptor modulator zolpidem augments hippocampal-prefrontal coupling during non-REM sleep. Neuropsychopharmacology, https://doi.org/10.1038/s41386-022-01355-9.
4. Phillips K.G.#, Bartsch U.#, McCarthy A.P., Edgar D.M., Tricklebank M.D., Wafford K.A. and Jones M.W. (2012) Decoupling of sleep-dependent cortical and hippocampal interactions in a neurodevelopmental model of schizophrenia. Neuron 76: 1-8.
5. McHugh T.J.#, Jones M.W.#, Quinn J.J., Balthasar N., Coppari R., Elmquist J.K., Lowell B.B., Fanselow M.S., Wilson M.A. and Tonegawa S. (2007) Dentate Gyrus NMDA Receptors Mediate Rapid Pattern Separation in the Hippocampal Network. Science 317: 94-99.

Dr Sungmin Kang

Research Associate, Cardiff University, UK

Research Overview

This ongoing research delves into the roles of the primary (S1) and secondary somatosensory cortices (S2) in texture discrimination learning within freely moving rodents. By employing behavioral studies and electrophysiology, the study aims to unravel how S1 and S2 contribute to the neural representations, learning dynamics, and cross-cortical interactions involved in texture discrimination. The insights gained from this research could advance our understanding of sensory perception and learning processes, with potential implications for enhancing cognitive flexibility in both animals and humans alike.

Selected Publications

1. A Pandey, S Kang, Pacchiarini, H Wyszynska, A Grewal, A Griffiths, I Healy-Millett, Z Masseri, Nl Hardingham, J O'Neill, R C. Honey, K Fox (2023), Interdependence of primary and secondary somatosensory cortices for plasticity and texture discrimination learning, Biorxiv .
2. S Kang, Y Hayashi, M Bruyns-Haylett, E Delivopoulos, Y Zheng (2020), Model-predicted balance between neural excitation and inhibition was maintained despite of age-related decline in sensory evoked local field potential in rat barrel cortex, Frontiers in Systems Neuroscience 14, 24 .
3. S Kang, Y Hayashi, M Bruyns-Haylett, Y Zheng (2019), Supplemental vitamin B-12 enhances the neural response to sensory stimulation in the barrel cortex of healthy rats but does not affect spontaneous neural activity, The Journal of Nutrition 149 (5), 730-737.
4. S Kang, M Bruyns-Haylett, Y Hayashi, Y Zheng (2017), Concurrent recording of co-localized electroencephalography and local field potential in rodent, JoVE, e56447.

Atsushi Kasai

Associate Professor of Department of Pharmaceutical Sciences, Osaka University

Research Overview

My research delves into the pathogenesis and neurobiology of mental disorders, with a particular emphasis on stress-related psychiatric conditions. I have innovated a technique to map neuronal activation across the entire brain, which has enabled the identification of previously overlooked claustral ensembles that regulate stress-induced emotional responses. To further decipher the intricate dynamics of neuronal ensembles and circuits, and their influence on anxiety-related behaviors in both health and disease states, I employ cutting-edge methodologies. These include calcium imaging in freely-moving mice, chemo/opto-genetic interventions, and single-cell transcriptome analyses.

Selected Publications

1. Niu M1., Kasai A1,*., Tanuma M., Seiriki K., Igarashi H., Igarashi H., Kuwaki T., Nagayasu K., Miyaji K., Ueno H., Tanabe W., Seo K., Yokoyama R., Ohkubo J., Ago Y., Hayashida M., Inoue KI., Takada M., Yamaguchi S., Nakazawa T., Kaneko S. Okuno H., Yamanaka A., and Hashimoto, H*. (2022). Claustrum mediates bidirectional and reversible control of stress-induced anxiety responses. Science Advances 8:eabi6375.
2. Endo F, Kasai A, Soto JS, Yu X, Qu Z, Hashimoto H, Gradinaru V, Kawaguchi R, and Khakh BS. (2022) Molecular basis of astrocyte diversity and morphology across the CNS in health and disease. Science, 378:eadc9020.
3. Tanuma M1, Kasai A1,*, Bando K, Kotoku N, Harada K, Minoshima M, Higashino K, Kimishima A, Arai M, Ago Y, Seiriki K, Kikuchi K, Kawata S, Fujita K*, and Hashimoto H*. (2020) Direct visualization of an antidepressant analog using surface-enhanced Raman scattering in the brain. JCI Insight 5:e133348.
4. Seiriki K, Kasai A*, Nakazawa T, Niu M, Naka Y, Tanuma M, Igarashi H, Yamaura K, Hayata-Takano A, Ago Y, and Hashimoto H*. (2019) Whole-brain block-face serial microscopy tomography at subcellular resolution using FAST. Nature Protocols, 14:1509-1529.
5. Seiriki K1, Kasai A1,*, Hashimoto T, Schulze W, Niu M, Yamaguchi S, Nakazawa T, Inoue KI, Uezono S, Takada M, Naka Y, Igarashi H, Tanuma M, Waschek JA, Ago Y, Tanaka KF, Hayata-Takano A, Nagayasu K, Shintani N, Hashimoto R, Kunii Y, Hino M, Matsumoto J, Yabe H, Nagai T, Fujita K, Matsuda T, Takuma K, Baba A, and Hashimoto H*. (2017) High-speed imaging and scalable whole-brain imaging in rodents and primates. Neuron 94:1085-1100.

Professor Masahisa Katsuno

Neurology, Nagoya University

Research Overview

My research focuses on the molecular pathogenesis and therapy development of neurodegenerative disorders including motor neuron disease and Lewy body disease. As for amyotrophic lateral sclerosis (ALS), I have been focusing on the pathological molecules TDP-43 and FUS, and showed that TDP-43 exits the nucleus not only of motor neurons but also of pancreatic beta cells in ALS patients, and that TDP-43 regulates the exocytosis of insulin vesicles in beta cells. In the field of Lewy body disease, my colleagues and I are analyzing functional and structural brain images of patients to identify abnormalities in neural circuit function, as well as developing biomarkers and preemptive treatments by establishing a cohort at a prodromal stage before the onset of the disease.

Selected Publications

Hattori M, Hiraga K, Satake Y, Tsuboi T, Tamakoshi D, Sato M, Yokoi K, Suzuki K, Arahata Y, Hori A, Kawashima M, Shimizu H, Matsuda H, Kato K, Washimi Y, Katsuno M. (2023). Clinico-imaging features of subjects at risk of Lewy body disease in NaT-PROBE baseline analysis. NPJ Parkinsons Dis. 9, 67. doi: 10.1038/s41531-023-00507-y.

Torii R, Hashizume A, Yamada S, Ito D, Kishimoto Y, Moriyoshi H, Inagaki T, Nakamura R, Nakamura T, Naoi T, Morita M, Katsuno M. (2022). Clinical features of female carriers and prodromal male patients with spinal and bulbar muscular atrophy. Neurology. 100, e84-e93. doi: 10.1212/WNL.0000000000201342.

Tsujikawa K, Hamanaka K, Riku Y, Hattori Y, Hara N, Iguchi Y, Ishigaki S, Hashizume A, Miyatake S, Mitsuhashi S, Miyazaki Y, Kataoka M, Jiayi L, Yasui K, Kuru S, Koike H, Kobayashi K, Sahara N, Ozaki N, Yoshida M, Kakita A, Saito Y, Iwasaki Y, Miyashita A, Iwatsubo T; Japanese Alzheimer's Disease Neuroimaging Initiative (J-ADNI), Ikeuchi T; Japanese Longitudinal Biomarker Study in PSP and CBD (JALPAC) Consortium, Miyata T, Sobue G, Matsumoto N, Sahashi K, Katsuno M. (2022). Actin-binding protein filamin-A drives tau aggregation and contributes to progressive supranuclear palsy pathology. Sci Adv. 8, eabm5029. doi: 10.1126/sciadv.abm5029.

Kiryu-Seo S, Matsushita R, Tashiro Y, Yoshimura T, Iguchi Y, Katsuno M, Takahashi R, Kiyama H. (2022). Impaired disassembly of the axon initial segment restricts mitochondrial entry into damaged axons. EMBO J. 41, e110486. doi: 10.15252/embj.2021110486.

Araki K, Araki A, Honda D, Izumoto T, Hashizume A, Hijikata Y, Yamada S, Iguchi Y, Hara A, Ikumi K, Kawai K, Ishigaki S, Nakamichi Y, Tsunekawa S, Seino Y, Yamamoto A, Takayama Y, Hidaka S, Tominaga M, Ohara-Imaizumi M, Suzuki A, Ishiguro H, Enomoto A, Yoshida M, Arima H, Muramatsu SI, Sobue G, Katsuno M. (2019). TDP-43 regulates early-phase insulin secretion via CaV1.2-mediated exocytosis in islets. J Clin Invest. 129, 3578-3593. doi: 10.1172/JCI124481.

Biography

Assistant Professor Yuka Koike

Department of Molecular Neuroscience, Niigata University

Research Overview

My research contributions focused on RNA metabolism and DNA methylation in ALS/FTD. We have focused on the autoregulatory mechanism of TDP-43 in ALS/FTD pathogenesis and clarified its epigenetic modifiers. We have shown that (i) the autoregulatory region of TDP-43 is demethylated in the motor cortex with aging, and (ii) the more autoregulatory region is demethylated, the younger the age at the onset of ALS. Results from our research were highly relevant as they provided new details into the workings of epigenetic regulation in gene expression and allowed for further extrapolations into neurodegenerative disease and epigenetic factors.

Selected Publications

1. Koike Y, Pickles S, Estades-Ayuso V, Jansen-West K, Qi YA, Li Z, Daughrity LM, Yue M, Zhang Y, Cook CN, et al. TDP-43 and other hnRNPs regulate cryptic exon inclusion of a key ALS/FTD risk gene, UNC13A. PLoS Biology, 21(3): e3002028, 2023.
2. Pickles S, Gendron T, Koike Y, Yue M, Song Y, Kachergus J, Shi J, DeTure M, Thompson EA, Oskarsson B, et al. Evidence of cerebellar TDP-43 loss of function in FTLD-TDP. Acta Neuropathol Commun, 10:107, 2022.
3. Ma XR, Prudencio M, Koike Y (co-first author), Vatsavayai SC, Kim G, Harbinski F, Briner A, Rodriguez CM, Guo C, Akiyama T, et al. TDP-43 represses cryptic exon inclusion in the FTD-ALS gene UNC13A. Nature, 603:124-130, 2022.
4. Koike Y, Sugai A, Hara N, Ito J, Yokoseki A, Ishihara T, Yamagishi T, Tsuboguchi S, Tada M, Ikeuchi T, Kakita A, Onodera O. Age-related demethylation of the TDP-43 autoregulatory region in the human motor cortex. Commun Biol, 4: 1107, 2021.
5. Sugai A, Kato T, Koyama A, Koike Y, Konno T, Ishihara T, Onodera O. Non-genetically modified models exhibit TARDBP mRNA increase due to perturbed TDP-43 autoregulation. Neurobiol Dis, 130: 104534, 2019.

Biography

Dr Wei-Li (William) Kuan

Head of Biology, Alborada Drug Discovery Institute (University of Cambridge)

Research Overview

My research focuses on disease modelling and the development of novel therapeutics for neurodegenerative conditions. I have devised a method to model Parkinson's disease pathology in nontransgenic rodents through the transvascular delivery of misfolded alpha-synuclein into the central nervous system. Furthermore, I have pioneered the strategy of transvascular administration of biologics across the blood-brain barrier. Presently, I oversee the processes of target identification and validation at the Alborada Drug Discovery Institute. There are dedicated biology and chemistry teams in our institute to assess promising new targets, establish assays facilitating high-throughput screening of compound libraries, and refine hits into potent and selective lead molecules. These lead molecules serve as tools for pharmacological proof-of-concept and provide the basis for the next generation of medicines to treat neurodegeneration.

Relevant Publications

1. Kuan WL, Stott K, He X, Wood TC, Yang S, Kwok JCF, Hall K, Zhao Y, Tietz O, Aigbirhio FI, Vernon AC, Barker RA. (2021). Systemic α-synuclein injection triggers selective neuronal pathology as seen in patients with Parkinson's disease. Mol Psychiatry. 2021 Feb;26(2):556-567.
2. Kuan WL, Alfaidi M, Horne CB, Vallin B, Fox S, Fazal SV, Williams-Gray CH, Barker RA. (2023). Selective neurodegeneration generated by intravenous α-synuclein pre-formed fibril administration is not associated with endogenous α-synuclein levels in the rat brain. Brain Pathol. 2023 May;33(3):e13128.
3. Kuan WL, Poole E, Fletcher M, Karniely S, Tyers P, Wills M, Barker RA, Sinclair JH. (2012). A novel neuroprotective therapy for Parkinson's disease using a viral noncoding RNA that protects mitochondrial complex I activity. J Exp Med. 2012 Jan 16;209(1):1-10.
4. Rooney TPC, Aldred GG, Boffey HK, Willems HMG, Edwards S, Chawner SJ, Scott DE, Green C, Winpenny D, Skidmore J, Clarke JH, Andrews SP. (2023). The Identification of Potent, Selective, and Brain Penetrant PI5P4Kγ Inhibitors as In Vivo-Ready Tool Molecules. J Med Chem. 2023 Jan 12;66(1):804-821.
5. Scott DE, Rooney TPC, Bayle ED, Mirza T, Willems HMG, Clarke JH, Andrews SP, Skidmore J. (2020). Systematic Investigation of the Permeability of Androgen Receptor PROTACs. ACS Med Chem Lett. 2020 Jun 8;11(8):1539-1547.

Dr Ruixiang Li

Postdoctoral Researcher at RIKEN

Research Overview

I am a postdoctoral researcher in Dr. Aya Ito-Ishida's lab at the RIKEN Center for Brain Science. Our lab currently focuses on Rett syndrome, which is a rare neurological disorder caused by mutations in the MeCP2 gene. By studying MeCP2 knockout mice, we hope to understand what happens at molecular, synaptic, cellular, and network level and what to do to relieve the symptoms in patients with Rett syndrome.

I graduated from the University of Tokyo in April 2023. I am interested in the dynamics of the brain. I use both macroscopic and microscopic imaging as well as optogenetics to record and manipulate the mouse brain activity at different scales and hope to understand how brain activity is organized and associated with behaviors and disorders.

Selected Publications

1. Li, R., Ohki, K., & Matsui, T., (2023). Ketamine-induced 1-Hz oscillation of spontaneous neural activity is not directly visible in the hemodynamics. Biochemical and Biophysical Research Communications (in press).
2. Noro, Y., Li, R., Matsui, T., & Jimura, K. (2023). A method for reconstruction of interpretable brain networks from transient synchronization in resting-state BOLD fluctuations. Frontiers in Neuroinformatics, 16, 960607.
3. Matsui, T., Jimura, K., & Li, R. (2023) Recent Studies of Human Resting-state Brain Activity Using a Public Open Database. Proceedings of the Institute of Statistical Mathematics, Vol. 71, No. 1, 81–95 (in Japanese).

Assistant Professor Tom Macpherson

Institute for Protein Research, Osaka University

Research Interests

Dr Macpherson's research is motivated by the need to develop a neurobiologically and computationally grounded understanding of the cellular and molecular mechanisms underlying intelligent behaviour, as well their disruption in various neurodevelopmental and neuropsychiatric conditions. Using a multi-disciplinary approach combining in-vivo neural imaging, genetics, artificial intelligence, and detailed cognitive-behavioural analysis, his research aims to identify how coordinated activity within specific cell types of basal ganglia neural circuits enables complex cognitive and limbic functions, including reward and aversive learning, attention, motivation, cognitive flexibility, and economical decision-making.

Recent Publications:

1. Nishioka, T., Attachaipanich, S., Hamaguchi, K. Lazarus, M., de Kerchove d'Exaerde, A., Macpherson, T*., Hikida, T*. Error-related signaling in nucleus accumbens D2 receptor-expressing neurons guides inhibition-based choice behavior in mice. Nat Commun 14, 2284 (2023). https://doi.org/10.1038/s41467-023-38025-3 (*Co-senior corresponding author).
2. Attachaipanich, S., Ozawa, T., Macpherson, T*., Hikida, T*. Dual roles for nucleus accumbens core dopamine D1-expressing neurons projecting to the substantia nigra pars reticulata in limbic and motor control in male mice. eNeuro 10(6), 0082-23-2023 (2023). https://doi.org/10.1523/ENEURO.0082-23.2023 (*Co-senior corresponding author).
3. Macpherson, T. & Hikida, T. Role of basal ganglia neurocircuitry in the pathology of psychiatric disorders. Psychiatry Clin. Neurosci 73, 289-301 (2019). https://doi.org/10.1111/pcn.12830.

Biography

Vaishnavi Manohar

PhD/RA, Sreedharan lab, Institute of psychiatry, psychology, and neuroscience, King's College London

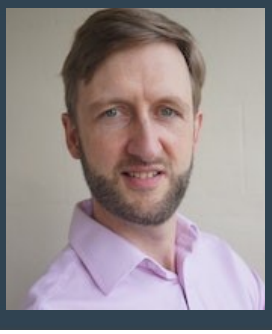
Research Overview

For my current research project, I am interested in looking at the effect of variants in the non-coding region of the TARDBP (encodes TDP-43) gene on ALS/FTD pathology. I am using a CRISPR-based screening technique called GenIE (genome engineering-based interrogation of enhancers) to screen for over 100 TARDBP UTR variants for their influence on TDP-43 gene expression, using iPSC derived cell models. I have previously worked on a screening project in flies to identify interactors of rdgB (retinal degeneration B), a membrane contact site protein critical to lipid transfer across ER-PM junction.

Selected Publications

1. Manohar, V., Crowley, L., & Sreedharan, J. (2023). TARDBP-Related Amyotrophic Lateral Sclerosis-Frontotemporal Dementia.
2. Mishra, S., Manohar, V., Chandel, S., Manoj, T., Bhattacharya, S., Hegde, N., ... & PADINJAT, R. (2023). A genetic screen to uncover molecular mechanisms underlying lipid transfer protein function at membrane contact sites and neurodegeneration. bioRxiv, 2023-07.

Biography



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UK-Japan Neuroscience Symposium
Vale Resort, Pontyclun,
31 August – 2 September 2023

Dr Tom Massey

Senior Clinical Research Fellow, Cardiff University

Research overview

My research interest is the molecular pathogenesis of Huntington's disease and other repeat expansion disorders. Most of these conditions are characterised by progressive neurodegeneration and none currently has a disease-modifying treatment. The overarching aim of my research programme is to leverage insights from human genetics, cell models and biochemistry to identify new therapeutic targets that can cut across a number of repeat expansion disorders. We use a combination of next-generation sequencing techniques, induced pluripotent stem cell models, CRISPR/Cas9 editing and biochemistry to go from clinical phenotypes to molecular mechanisms.

As a Neurologist I see patients in clinic and have been heavily involved in clinical trials in Huntington's disease. My hope is that by understanding the molecular details of repeat expansion disorders we can develop novel treatments with the potential to slow or prevent these currently incurable diseases.

Selected Publications

1. McAllister, B, Donaldson, J…& Massey, TH* (2022) Exome sequencing of individuals with Huntington's disease implicates FAN1 nuclease activity in slowing CAG expansion and disease onset. Nature Neuroscience 25, 446-457; doi: 10.1038/s41593-022-01033-5.
2. Lobanov, S, McAllister, B, McDade-Kumar, G, Landwehrmeyer, GB, Rosser, AE, Paulsen, JS, Lee, J-M, MacDonald, ME, Gusella, JF, Ryten, M, Williams, N, Holmans, P, Massey, TH* & Jones, L (2022) Huntington's disease age at motor onset is modified by the tandem hexamer repeat in TCERG1. NPJ Genomic Medicine, 7, 53. 10.1038/s41525-022-00317-w.
3. McAllister, B, Gusella, JF, Landwehrmeyer, GB, Lee, J-M, MacDonald, ME, Orth, M, Rosser, AE, Williams, NM, Holmans, P, Jones, L & Massey, TH* (2021) The timing and impact of psychiatric, cognitive and motor abnormalities in Huntington's disease. Neurology, doi: 10.1212/WNL.0000000000011893.
4. Genetic Modifiers of Huntington's Disease (GeM-HD) Consortium (2019)* CAG repeat not polyglutamine length determines timing of Huntington's disease onset. Cell, 178, 887-900. Doi: 10.1016/j.cell.2019.06.036.
5. Tabrizi SJ, Phase 1–2a IONIS-HTTRx Study Site Teams* et al. (2019) Targeting Huntingtin Expression in Patients with Huntington's Disease. N. Engl. J. Med., 380, 2307-2316. Doi: 10.1056/NEJMoa1900907.

Biography

Professor Masanori Matsuzaki

Department of Physiology, Graduate School of Medicine, The University of Tokyo

Research Overview

My research focuses on the neuronal circuits for motor control and decision-making. Especially, we study the cerebral cortex-basal ganglia/cerebellum circuits in the mouse and frontal-parietal/temporal networks in the common marmoset, a small New World monkey. We developed mouse forelimb motor tasks to reveal motor information flows in cortico-cortical circuits and cortico-subcortical circuits. In our marmoset research, we have developed two-photon calcium imaging and optogenetic techniques and forelimb motor tasks from scratch. it is now possible to use similar motor tasks, the same genetically encoded indicators, the same imaging and manipulating methods, and the same analyses, to compare the neural activity over the broad cerebral cortex between rodents and marmosets. I believe that study of the marmoset brain will be of benefit for understanding the evolution of the brain function, and also the basic neural mechanisms of primate-specific higher order functions and human brain diseases.

Selected Publications

1. Terada S., Kobayashi K., and Matsuzaki M. (2022). Transition of distinct context-dependent ensembles from secondary to primary motor cortex in skilled motor performance. Cell Rep. 10.1016/j.celrep.2022.111494.
2. Kondo M. and Matsuzaki M. (2021). Neuronal representations of reward-predicting cues and outcome history with movement in the frontal cortex. Cell Rep. 10.1016/j.celrep.2021.108704.
3. Ebina T., Obara K., Watakabe A., Masamizu Y., Terada S., Matoba R., Takaji M., Hatanaka A., Nambu A., Mizukami H., Yamamori T., and Matsuzaki M. (2019). Arm movements induced by non-invasive optogenetic stimulation of the motor cortex in the common marmoset. Proc Natl Acad Sci U S A. 10.1073/pnas.1903445116.
4. Terada S., Kobayashi K., Ohkura M., Nakai J., and Matsuzaki M. (2018). Super-wide-field two-photon imaging with a micro-optical device moving in post-objective space. Nat Commun. 10.1038/s41467-018-06058-8.
5. Tanaka Y.H., Tanaka Y.R., Kondo M., Terada S., Kawaguchi Y., and Matsuzaki M. (2018). Thalamocortical axonal activity in motor cortex exhibits layer-specific dynamics during motor learning. Neuron. 10.1016/j.neuron.2018.08.016.
6. Ebina, T., Masamizu Y., Tanaka Y.R., Watakabe A., Hirakawa R., Hirayama Y., Hira R., Terada S., Koketsu D., Hikosaka K., Mizukami H., Nambu A., Sasaki E., Yamamori T., and Matsuzaki M. (2018). Two-photon imaging of neuronal activity in motor cortex of marmosets during upper-limb movement tasks. Nat Commun. 10.1038/s41467-018-04286-6.

Biography

Dr. Stephen McHugh

University Research Lecturer, University of Oxford

Research Overview

My research focuses on the neurophysiological underpinnings of learning and memory, and lies at the interface of systems neuroscience and pre-clinical models of neurological and psychiatric disease. Specifically, I am interested in how memories are encoded, consolidated, and expressed via the temporal coordination of ensembles of single-neurons, and how different cell types and neurotransmitters influence this neuronal activity and associated behaviours. My recent work has discovered a key role for adult-born dentate granule cells in the temporal coordination of spiking activity in downstream hippocampal neurons, which is essential for the emergence of sparse hippocampal population activity and flexible memory recall. My current research is dedicated to exploring the circuit mechanisms governing memory consolidation during sleep.

Selected Publications

1. McHugh SB, Lopes-dos-Santos V, Gava GP, Hartwich K, Tam SKE, Bannerman DM, Dupret D (2022). Adult-born dentate granule cells promote hippocampal population sparsity. Nature Neuroscience 25(11): 1481-1491. https://doi.org/10.1038/s41593-022-01176-5.

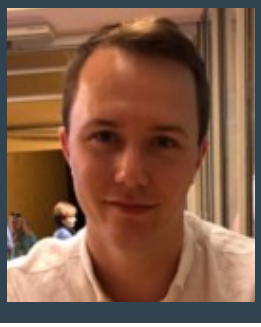
2. Gava GP, McHugh SB, Lefevre L, Lopes-dos-Santos V, Trouche S, El-Gaby M, Schultz SR, Dupret D (2021). Integrating new memories into the hippocampal network activity space. Nature Neuroscience 24: 326–330. https://doi.org/10.1038/s41593-021-00804-w.

3. Lima J, Sharp T, Bannerman DM, McHugh SB (2019). Enhanced discriminative aversive learning and amygdala responsivity in 5-HT transporter mutant mice. Translational Psychiatry 9(1): 139. https://doi.org/10.1038/s41398-019-0476-8.

4. McHugh SB, Barkus C, Huber A, Capitao L, Lima J, Lowry J, Bannerman D (2014) Aversive prediction error signals in the amygdala. Journal of Neuroscience 34 (27): 9024-9033. https://doi.org/10.1523/JNEUROSCI.4465-13.2014.

5. Barkus C, Line SJ, Huber A, Capitao L, Lima J, Jennings K, Lowry J, Sharp T, Bannerman DM, McHugh SB (2014) Variation in serotonin transporter expression modulates fear-evoked hemodynamic responses and theta-frequency neuronal oscillations in the amygdala. Biological Psychiatry 75: 901-908. https://doi.org/10.1016/j.biopsych.2013.09.003.

Biography



Dr Scott Mitchell

Postdoctoral Research Associated, Synapse & Therapeutic Discovery Laboratory, King's College London

Research Overview

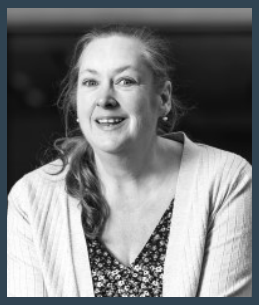
My research centers on understanding the mechanisms which alter synapse and receptor function during development, in response to behavioral stimuli, and during neurodegeneration. As part of the Synapse & Therapeutic Discovery Laboratory (King's College London), our recent focus has been on the impact of neurodegeneration-related proteins such as fused in sarcoma, amyloid beta, and hyperphosphorylated tau on synapse weakening. Using a range of neurophysiological, molecular, and multiphoton imaging assays, we aim to examine the intricate molecular relationship between pathophysiological proteins and synapse weakening. Notably, we found that phosphorylation of tau at the PHF1E region disrupts its interaction with PACSIN1, and this reducing binding is crucial for Tau-PHF1E induced synapse weakening. Expanding on this, we are currently investigating how the spatial distribution of synapses within the dendritic arbor influences Tau-PHF1E mediated synapse weakening (i.e., do all synapses respond the same way). This sheds light on dendrites as essential sites for synaptic integration, signal processing, and their role in pathophysiological progression.

Selected Publications

1. Regan et al (2021) Regulation of synapse weakening through interactions of the microtuble associated protein tau with PACSIN1. J Neurosci 41(34): 7162-7170 10.1523/JNEUROSCI.3129-20.2021.

2. Yi et al (2020) M1 muscarinic acetylcholine receptor dysfunction in moderate Alzheimer's disease pathology. Brain Communications 2(2). 10.1093/braincomms/fcaa058.

3. Kelly et al (2020) Identification of intraneuronal amyloid beta oligomers in locus coeruleus neurons of Alzheimer's patients and their potential impact on inhibitory neurotransmitter receptors and neuronal excitability. Neuropathology and Applied Neuroscience 47(6): 1-18 10.1111/nan.12674.



Professor Wendy Noble

Professor of Molecular Neurobiology, University of Exeter

Research Overview

My research programme aims to elucidate the molecular mechanisms underlying the development of Alzheimer's disease (AD) and other tauopathies. These neurodegenerative diseases, for which there are no effective treatments, are characterised by deposits of abnormal tau that spread across the brain as disease progresses. We use rodent and human (iPS and iNP)-cells, transgenic rodent models of disease and post-mortem human brain to examine how and why tau becomes altered in neurodegenerative diseases, and how protein modifications affect tau function and cause toxicity. Our recent research has also led us to examine how glial cells, and particularly astrocytes, influence tau biology. We work closely with clinicians to better investigate novel strategies to prevent tau-associated damage and treat neurodegenerative diseases.

Selected Publications

1. Rupawala H, Shah K, Davies C, Rose J, Colom Cadena M, Peng X, Granat L, Aljuhani M, Mizuno M, Troakes C, Perez-Nievas BG, Morgan A, So P-W, Hortobagyi T, Spires-Jones TL, Noble W*, Giese KP* (2022). Amyloid plaque associated CSPalpha deposition as a marker of pre-synaptic dysfunction in Alzheimer's disease. Brain Comms. 4(4): fcac192. doi: 10.1093/braincomms/fcac192. PMID: 35928052. *co-corresponding authors.

2. Perez-Nievas BG, Johnson L, Beltran Lobo P, Hughes MM, Gammallieri L, Tarsitano F, Myszczynska MA, Vazquez-Villasenor I, Jimenez-Sanchez M, Troakes C, Wharton SB, Ferraiuolo L, Noble W. (2021). Astrocytic C-X-C motif chemokine ligand-1 mediates b-amyloid-induced synaptotoxicity. J. Neuroinflammation. 18(1):306. doi: 10.1186/s12974-021-02371-0. PMID: 34963475.

3. Staurenghi E, Cerrato V, Testa G, Giannelli S, Leoni V, Caccia C, Buffo A, Noble W*, Perez-Nievas BG*, Leonarduzzi G* (2021). Oxysterols present in Alzheimer's disease brain induce synaptotoxicity by activating astrocytes: a major role for lipocalin-2. Redox Biology. 39:101837. doi: 10.1016/j.redox.2020.101837 PMID:33360775. *co-corresponding author.

4. Glennon EB, Lau DH-W, Gabriele RMC, Taylor MF, Troakes C, Opie-Martin-Sarah, Elliott C, Killick R, Hanger DP, Perez-Nievas BG, Noble W (2020). BIN1 protein loss in Alzheimer's disease promotes synaptic tau accumulation and disrupts tau release. Brain Comms. 2(1): fcaa011. doi: 10.1093/braincomms/fcaa011 PMID: 32500121.

5. Howard R, Zubko O, Bradley R, Harper E, Pank L, O'Brien J, Fox C, Tabet N, Livingston G, Bentham P, McShane R, Burns A, Ritchie C, Reeves S, Lovestone S, Ballard C, Noble W, Nilforooshan R, Wilcock G, Gray R; Minocycline in Alzheimer Disease Efficacy (MADE) Trialist Group. (2019). Minocycline at 2 Different Dosages vs Placebo for Patients With Mild Alzheimer Disease: A Randomized Clinical Trial. JAMA Neurology. 77(2):164-174. doi: 10.1001/jamaneurol.2019.3762 PMID: 31738372.

Designated Assistant Professor Kotaro Oiwa

Department of Neuroscience and Pathobiology, Research Institute of Environmental Medicine, Nagoya University

Research Overview

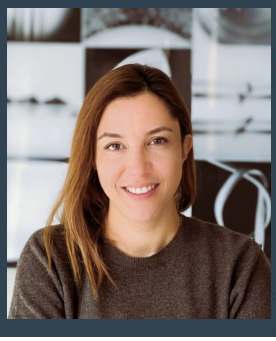
My research interest is focused on the cellular and molecular mechanisms of TDP-43 underlying ALS/FTLD.

I discovered that the physiological TDP-43 dimerization/multimerization via its N-terminal domain is reduced in ALS patients' brain and spinal cord tissues. Interestingly, we found that TDP-43 monomerization preceded and recapitulated TDP-43 pathological changes. I created a novel high-throughput reporter for quantifying TDP-43 dimerization/multimerization, “DiLuc-TDP-43”, which is a powerful and sensitive tool for detecting TDP-43 aberrations.

Selected Publications

1. Oiwa K, et al. “Monomerization of TDP-43 is a key determinant for inducing TDP-43 pathology in amyotrophic lateral sclerosis.” Science Advances. 2023 Aug 4; 9 (31): eadf6895.
2. Watanabe S, Inami H, Oiwa K, et al. “Aggresome formation and liquid-liquid phase separation independently induce cytoplasmic aggregation of TAR DNA-binding protein 43.” Cell Death Dis. 2020 Oct 23;11(10):909.
3. Watanabe S, Oiwa K, et al. “ALS-linked TDP-43M337V knock-in mice exhibit splicing deregulation without neurodegeneration.” Mol Brain. 2020 Jan 20;13(1):8.
4. Nishino K, Watanabe S, Shijie J, Murata Y, Oiwa K, et al. “Mice deficient in the C-terminal domain of TAR DNA -binding protein 43 develop age-dependent motor dysfunction associated with impaired Notch1-Akt signaling pathway.” Acta Neuropathol Commun. 2019 Jul 25;7(1):118.

Biography



Dr Zeynep Okray

Postdoctoral scientist, Centre for Neural Circuits and Behaviour, Oxford

Research Overview

My main research interest lies in how neural circuits encode different types of memory. I am currently a postdoctoral fellow in Scott Waddell's laboratory at the University of Oxford, and my work here focuses on how the brain integrates different modes of sensory information to create meaningful and adaptive memories.

I obtained my doctorate from the University of Leuven/VIB, where I worked in the laboratory of Bassem Hassan, focusing on the genetic mechanisms and neurobiology of Fragile X Syndrome.

I am passionate about leveraging the power of the fruit fly model (Drosophila melanogaster) to address fundamental questions exploring brain function and disorders.

Selected Publications

1. Okray Z*, Jacob PF*, Stern C, Desmond K, Otto N, Talbot CB, Vargas-Gutierrez P, Waddell S. Multisensory learning binds neurons into a cross-modal memory engram. Nature (2023).
2. Jacob PF, Vargas-Gutierrez P, Okray Z, Vietti-Michelina S, Felsenberg J, Waddell S. Prior experience conditionally inhibits the expression of new learning in Drosophila. Current Biology (2021).
3. Franco LM, Okray Z, Linneweber GA, Hassan BA, Yaksi E. Reduced Lateral Inhibition Impairs Olfactory Computations and Behaviors in a Drosophila Model of Fragile X Syndrome. Current Biology (2017).
4. Okray Z, de Esch CE, Van Esch H, Devriendt K, Claeys A, Yan J, Verbeeck J, Froyen G, Willemsen R, de Vrij FM, Hassan BA. A novel fragile X syndrome mutation reveals a conserved role for the carboxy-terminus in FMRP localization and function. EMBO Molecular Medicine (2015).
5. Okray Z, Hassan BA. Genetic approaches in Drosophila for the study of Neurodevelopmental Diseases. Neuropharmacology (2013) *equal contribution.

Biography

Dr Abidemi Otaiku

Clinical Research Fellow, UK Dementia Research Institute Care Research & Technology Centre, Imperial College London

Research Overview

My research examines the neurobiological basis of sleep and dreaming. In particular, my work explores how sleep and dreaming change with healthy ageing and neurodegenerative diseases, and how this knowledge can be utilised to optimise brain health.

Selected Publications

1. Otaiku AI. Distressing dreams in childhood and risk of cognitive impairment or Parkinson's disease in adulthood: A national birth cohort study. EClinicalMedicine. 2023; 57:101872.
2. Otaiku AI. Distressing dreams, cognitive decline, and risk of dementia: A prospective study of three population-based cohorts. EClinicalMedicine. 2022; 52:101640.
3. Otaiku AI. Distressing dreams and risk of Parkinson's disease: A population-based cohort study. EClinicalMedicine; 48:101474.
4. Otaiku AI. Dream content predicts motor and cognitive decline in Parkinson's disease. Movement Disorders Clinical Practice. 2021;7:1041-1-51.
5. Otaiku AI. Association of sleep abnormalities in older adults with risk of developing Parkinson's disease. Sleep. 2022; 45: zsac206.

Biography

T.W. Robbins

Professor of Cognitive Neuroscience, University of Cambridge

Research Overview

My research has focused on executive functions of the prefrontal cortex and associated structures including the striatum, and their chemical modulation by ascending monoaminergic and cholinergic neurotransmitter systems. These executive functions comprise working memory, cognitive flexibility and inhibition, investigated using the CANTAB computerised neuropsychological test battery, which I co-invented to enhance cross-species translation in rodents, non-human primates and humans, including patients with neurological or psychiatric disorders. I have used a variety of techniques, involving several neuroimaging modalities, in vivo neurochemical monitoring, and neuropharmacological methods. I have helped to develop a novel theory of addiction, applied also to obsessive-compulsive disorder, and to characterise behavioural traits of impulsivity and compulsivity. I have also investigated several modes of treatment in neuropsychiatric patients, including psychopharmacological and neurosurgical approaches.

Selected Publications

1. Biria M., Banca P., Healy, M., Keser, E., Sawiak, S., Rodgers C.T., Rua, C., Pereira de Souza, A.M.F.L., Marzuki, A.,Sule,A, Ersche, K.D. & Robbins, T.W. (2023). Cortical glutamate and GABA are related to compulsive behaviour in individuals with obsessive compulsive disorder and healthy controls. Nature Communications. 14 (1):3324. doi:10.1038/s41467-023-38695-z.

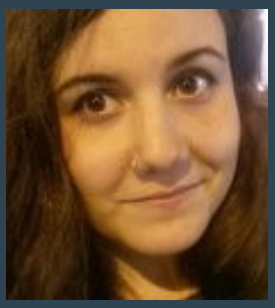
2. Duan, L. Y., Horst, N. K., Cranmore, S., Horiguchi, N., Cardinal, R. N., Roberts, A. C., & Robbins, T. W. (2021). Controlling one's world: Identification of sub-regions of primate PFC underlying goal-directed behavior. Neuron, 109(15), 2485–2498. e5. https://doi.org/10.1016/j.neuron.2021.06.003.

3. Robbins TW, Vaghi MM, Banca P. (2019) Obsessive-Compulsive Disorder: Puzzles and Prospects. Neuron., 102(1), 27-47. https://doi.org/10.1016/j.neuron.2019.01.046.

4. Everitt, B.J. & Robbins, T.W. (2016) Drug Addiction: Updating Actions to Habits to Compulsions Ten Years On. Annual Review of Psychology, 67, 23-50. (Review) https://doi.org/10.1146/annurev-psych- 122414-033457.

5. Dalley, J.W., Fryer, T.D., Brichard, L., Robinson, E.S.J., Theobald, D.E.H., Lääne, K., Peña, Y., Murphy, E.R., Shah, Y., Probst, K., Abakumova, I., Aigbirhio, F.I., Richards, H.K., Hong, Y., Baron, J.C., Everitt, B.J. & Robbins, T.W. (2007) Nucleus accumbens D2/3 receptors predict trait impulsivity and cocaine reinforcement. Science, 315, 1267-1270. https://doi.org/10.1126/science.1137073.

Biography



Dr. Martina Sassi

PDRA in Molecular Neurobiology, Swansea University

Research Overview

My studies involved understanding the neuroprotective and neurogenic properties of the stomach peptide hormone, ghrelin. Its unique biology includes a post-translational modification (via the addition of a fatty acid) that is essential for binding and activating its receptor. This modification represents a putative drug target to regulate ghrelin signalling. My post-doctoral studies involve characterizing the opposing actions of the acylated and non-acylated version of ghrelin within the CNS and the adaptive immune system. The aim of my work is to identify mechanisms to slow or even stop progression of neurodegenerative diseases.

Selected publications

1. Acylation, a Conductor of Ghrelin Function in Brain Health and Disease. Thomas, AS: Sassi, M; Angelini, R; Morgan, AH and Davies, JS, Frontiers in Physiology 30 June 2022.
2. Ghrelin Acylation—A Post-Translational Tuning Mechanism Regulating Adult Hippocampal Neurogenesis. Sassi, M.; Morgan, A.H.; Davies, J.S. Cells. 22 Feb 2022.
3. Unacylated-Ghrelin Impairs Hippocampal Neurogenesis and Memory in Mice and Is Altered in Parkinson's Dementia in Humans. Hornsby AKE, Buntwal L, Carisi MC, Santos VV, Johnston F, Roberts LD, Sassi M, Mequinion M, Stark R, Reichenbach A, Lockie SH, Siervo M, Howell O, Morgan AH, Wells T, Andrews ZB, Burn DJ, Davies JS. Cell Rep Med. 20 Oct 2020.
4. Ghrelin regulation of adult hippocampal neurogenesis – implications for health and disease. Buntwal L*, Sassi M*, Morgan AH, Andrews ZB, Davies JS. Trends in Endocrinology and Metabolism. 21 Aug 2019.

Biography

Dr. Bhuvaneish T Selvaraj

Chancellors' fellow – Centre for Clinical Brain Sciences, Emerging leader – UK Dementia Research Institute, University of Edinburgh

Research Overview

I am an engineering graduate from Anna University, India. My research focus is on understanding the pathomechanisms of neurodegenerative diseases with a specific focus on amyotrophic lateral sclerosis (ALS). During my PhD at University of Wuerzburg – Germany, I elucidated a key scientific question whether local axonal maintenance would improve motor function in mouse model of ALS. Firstly, I identified the novel axonal function of ciliary neurotrophic factor (CNTF) whereby it modulates microtubule dynamics locally in axons to reverse motor neuron degeneration in progressive motor neuronopathy mouse model of ALS. Secondly, I also assessed if accumulation of neurofilament in axons, a characteristic hallmark in neurodegenerative diseases, contributes to the disease progression in ALS. These crucial studies showed that local axonal maintenance and thereby preserving axon from degeneration is a beneficial neuroprotective strategy for ALS. I then took an early decision to enhance my skill set in human experimental medicine platforms – human stem cell disease modelling and autopsy studies - therefore, I did my post-doctoral research in University of Edinburgh where I led a study to elucidate motor neuron specific vulnerability to glutamate mediated excitotoxicity – a major pathophysiology in ALS. I also elucidated the non-cell autonomous role of ALS astrocytes in contributing to loss of neuronal activity in motor neurons. Since 2020 as a Chancellor's fellow at University of Edinburgh, I lead research programme that aims to gain greater understanding of the molecular pathomechanisms leading to selective vulnerability of motor neurons and axonal homeostasis dysfunction in ALS.

Selected Publications

1. Banerjee P et al., Cell-autonomous immune dysfunction driven by disrupted autophagy in C9orf72-ALS iPSC-derived microglia contributes to neurodegeneration. Science Advances. 2023.
2. James, O.G., Selvaraj, B.T., …. Chandran, S. Human iPSC-derived myelinoids for investigation of myelin disorders and adaptive myelination. Developmental Cell, 2021.
3. Perkins, E.M., Burr, K., Banerjee, P., Mehta.A.R., Dando, O., … Selvaraj, B.T., … Wyllie, D.J., Chandran, S., Livesey, M. Altered network properties in C9ORF72 repeat expansion cortical neurons are due to synaptic dysfunction. Molecular Neurodegeneration, 2021.
4. Mehta, A.R., Gregory, J., …. Selvaraj, B.T (corresponding author). Mitochondrial bioenergetic deficits in C9orf72 amyotrophic lateral sclerosis motor neurons cause dysfunctional axonal homeostasis. Acta Neuropathologica, 2021 https://doi.org/10.1007/s00401-020-02252-5.
5. Zhao, C., Devlin, A. C., …. Selvaraj, B. T., …. Miles, G. B., Chandran, S. Mutant C9ORF72 human iPSC-derived astrocytes cause non-cell autonomous motor neuron pathophysiology. Glia 2019. DOI: 10.1002/glia.23761.
6. Selvaraj, B.T.*, Livesey, M. R.*, Zhao, C., …. Wyllie, D. J. A., Chandran, S. C9ORF72 repeat expansion causes vulnerability of motor neurons to Ca2+-permeable AMPA receptor-mediated excitotoxicity. Nat Commun, 2018. DOI:10.1038/s41467-017-02729-0.* Equal contribution.
7. Yadav, P., Selvaraj, B.T., …. Sendtner, M. Neurofilament depletion improves microtubule dynamics via modulation of Stat3/stathmin signalling. Acta Neuropathol., 2016. DOI: 10.1007/s00401-016-1564-y.
8. Selvaraj, B.T. Frank, N., Bender, F. L., Asan, E., Sendtner, M. Local axonal function of STAT3 rescues axon degeneration in the pmn model of motoneuron disease. J. Cell Biol., 2012. DOI: 10.1083/jcb.201203109. Book Chapter.

Biography

Mari Shiozaki

Ph.D. student at the Department of Physiology (Yuzaki Lab), Keio University School of Medicine

Research Overview

Synapses are essential components of neuronal circuits, and many neurological and neuropsychiatric disorders are considered synaptopathies. My current research explores the molecular mechanisms by which cochlear synapse dysfunction leads to hearing loss. Cbln1, an essential synaptic organizer found in the CNS, is also highly expressed in the inner ear. We found that Cbln1 knockout mice exhibited impaired hearing in high-frequency sounds, a characteristic feature of Age-related hearing loss (ARHL). Morphological analysis indicates that Cbln1 plays a pivotal protective role in the inner ear by maintaining medial olivocochlear (MOC) neuron-outer hair cell (OHC) synapses. By unraveling the physiological significance of Cbln1 in the cochlear system, we aim to develop potential applications for treating ARHL.

Biography

Hiroki Shiwaku, M.D., Ph.D.

Associate Professor, Department of psychiatry and behavioral sciences, Tokyo Medical and Dental University

Research Overview

I am a clinical psychiatrist and am investigating the molecular pathology of schizophrenia.

My recent research focuses on finding unknown autoantibodies in schizophrenia patients that shape its pathology. We found autoantibodies against synaptic adhesion molecules, NCAM1 and NRXN1, from patients with schizophrenia. Administration of anti-NCAM1 and anti-NRXN1 autoantibodies from patients with schizophrenia into the cerebrospinal fluid of mice reduced the number of spines/synapses in the frontal cortex and induced schizophrenia-related behavior such as reduced cognition, impaired pre-pulse inhibition, and reduced social novelty preference. Removal of anti-NCAM1 and anti-NRXN1 autoantibodies may be therapeutic targets for a subgroup of patients who are positive for these autoantibodies.

Selected Publications

1. Shiwaku H, Katayama S, Gao M, Kondo K, Nakano Y, Motokawa Y, Toyoda S, Yoshida F, Hori H, Kubota T, Ishikawa K, Kunugi H, Ikegaya Y, Okazawa H, Takahashi H. Analyzing schizophrenia-related phenotypes in mice caused by autoantibodies against NRXN1α in schizophrenia. Brain Behav Immun. 2023;111:32-45.
2. Shiwaku H, Katayama S, Kondo K, Nakano Y, Tanaka H, Yoshioka Y, Fujita K, Tamaki H, Takebayashi H, Terasaki O, Nagase Y, Nagase T, Kubota T, Ishikawa K, Okazawa H, Takahashi H. Autoantibodies against NCAM1 from patients with schizophrenia cause schizophrenia-related behavior and changes in synapses in mice. Cell Rep Med. 2022;3(4):100597.
3. Shiwaku H, Yoshimura N, Tamura T, Sone M, Ogishima S, Watase K, Tagawa K, Okazawa H. Suppression of the novel ER protein Maxer by mutant ataxin-1 in Bergman glia contributes to non-cell-autonomous toxicity. EMBO J. 2010;29(14):2446-60.

Biography

Symposiwm Niwrowyddoniaeth DU-Japan
Vale Resort, Pont-y-clun,
31 Awst – 2 Medi 2023

UK-Japan Neuroscience Symposium
Vale Resort, Pontyclun,
31 August – 2 September 2023

Gaynor Ann Smith

Senior Lecturer at Cardiff University

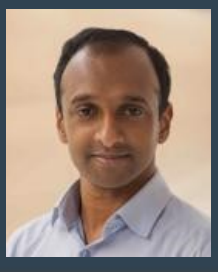
Research Overview

1) To discover new genes which control mitochondria maintenance in the axons of neurons using an unbiased in vivo genetic approach.

We know relatively little about the basic biology of mitochondrial biogenesis, morphological changes, transport, or function in axons in vivo, yet mitochondrial abnormalities in the terminals have been strongly linked to the etiology of several neurodegenerative disorders. We perform unbiased genetic screening in Drosophila to discover new mitochondrial regulators in axons and characterize their function. Mitochondrial mechanisms and dynamics are applicable to neurodegenerative disease and axonopathies, where mitochondrial dysfunction is typically an early feature. Other interests include understanding how mitochondria “communicate” with other organelles such as peroxisomes and endoplasmic reticulum to drive metabolic processes.

2) To investigate how new genes discovered from GWAS approaches contribute to the pathological mechanisms of Alzheimer’s disease.

Biography



Jemeen Sreedharan

Wellcome Trust Senior Research Fellow, King's College London

Research Overview

I am a neurologist and neuroscientist specializing in amyotrophic lateral sclerosis (ALS). My laboratory develops preclinical human, mouse and fly models of ALS and the related condition frontotemporal dementia (FTD). We focus on TDP-43, a complex DNA/RNA binding protein that is central to the pathogenesis of ALS and FTD. I have made significant discoveries in the course of our studies: the identification of mutations in TDP-43 as a cause of ALS, the discovery that said mutations can disturb the ability of TDP-43 to autoregulate in a novel knock-in mouse, and the discovery of several modifiers of TDP-43 toxicity in flies and mice. We have continued to develop the TDP-43 knock-in mouse as a translational tool using in vivo MRI. My group is also using human stem cell derived neurons and glia to develop novel models of disease, identify suppressors of neurotoxicity, and dissect the mechanisms by which TDP-43 misregulation occurs. I continue to see patients in the motor nerve clinic at King's College Hospital and I am actively involved in several ALS clinical trials. Everything my group does is directed towards finding better treatments for patients with ALS.

Masami Masuda-Suzukake, Ph.D.

Researcher, Tokyo Metropolitan Institute of Medical Science

Research Overview

I am interested in the pathological mechanisms of sporadic neurodegenerative diseases with an accumulation of disease-specific protein aggregates. I study the prion-like propagation mechanisms of α-synuclein and tau. We established an animal model of α-synuclein propagation by injecting synthetic α-synuclein filaments into the brains of wild-type mice1. In this model, pathological α-synuclein was phosphorylated and ubiquitinated, and spread through neural networks2. For tau, we found that synthetic tau fibrils induced in the presence of dextran sulfate have seeding propensity in vivo3. Recently, we developed new tau knock-in mice expressing 6 isoforms of murine tau (3R + 4R)4. Our goal is to elucidate the mechanisms of neurodegenerative diseases and find new therapeutic targets through the analysis of these animal models.

Selected Publications

1. Masuda-Suzukake M, Nonaka T, Hosokawa M, Oikawa T, Arai T, Akiyama H, Mann DM, and Hasegawa M. Prion-like spreading of pathological α-synuclein in brain. Brain, 136, 1128-1138, 2013.
2. Masuda-Suzukake M, Nonaka T, Hosokawa M, Kubo M, Shimozawa A, Akiyama H, and Hasegawa M. Pathological alpha-synuclein propagates through neural networks. Acta Neuropathol. Commun. 2, 88 2014.
3. Masuda-Suzukake M, Suzuki G, Hosokawa M, Nonaka T, Goedert M, and Hasegawa M. Dextran sulphate-induced tau assemblies cause endogenous tau aggregation and propagation in wild-type mice. Brain Commun. Vol. 8;2(2):fcaa091. doi: 10.1093/braincomms/fcaa091.
4. Hosokawa M, Masuda-Suzukake M, Shitara H, Shimozawa A, Suzuki G, Kondo H, Nonaka T, Campbell W, Arai T, and Hasegawa M. Development of a novel tau propagation mouse model endogenously expressing 3 and 4 repeat tau isoforms. Brain. Vol.145,p349-361, doi: 10.1093/brain/awab.

Researcher Koichi Tabata

Schizophrenia Research Project, Tokyo Metropolitan Institute of Medical Science and Department of Psychiatry and Behavioral Sciences, Tokyo Medical and Dental University

Research Overview

My research focuses on the role of zinc deficiency in the cause of schizophrenia. Zinc is an essential trace element that plays an important role in immune cell differentiation and function. Recently, lower serum zinc levels have been shown in a variety of autoimmune diseases and psychiatric disorders, including schizophrenia, compared to healthy controls. We previously reported the association with hair zinc levels and psychosis risk among adolescents, using data from a population-based birth cohort study of the Tokyo Teen Cohort. Subsequently, we found that schizophrenia with hypozincemia (a serum zinc level of less than 80 µg/dL) showed clinical severities, including a higher proportion of inpatients, longer durations of hospitalization and higher scores of PANSS (Positive and Negative Syndrome Scale) compared to schizophrenia without hypozincemia.

Selected Publications

1. Tabata K, Miyashita M, Yamasaki S, Toriumi K, Ando S, Suzuki K, Endo K, Morimoto Y, Tomita Y, Yamaguchi S, Usami S, Itokawa M, Hiraiwa-Hasegawa M, Takahashi H, Kasai K, Nishida A, Arai M. Hair zinc levels and psy chosis risk among adolescents. Schizophrenia, 8(1):107, 2022.

Biography

Professor Hidehiko Takahashi

Chair of Psychiatry and Behavioral Sciences, Graduate School of Medical and Dental Sciences, Tokyo Medical and Dental University

Director of Center for Brain Integration Research, Tokyo Medical and Dental University

Research Overview

I have been conducting brain imaging research with an interest in the neural basis of cognition, emotion, and decision making in healthy subjects and in neuropsychiatric disorders. More recently, I have become involved in computational psychiatry. In particular, I am using AI and natural language processing techniques to understand the neural basis of thought disorder in schizophrenia, as well as data-driven research in the development of imaging biomarkers and neurofeedback for psychiatric disorders.

Selected Publications

1. Matsumoto Y, Nishida S, Hayashi R, Son S, Murakami A, Yoshikawa N, Ito H, Oishi N, Masuda N, Murai T, Friston K, Nishimoto S, Takahashi H. Disorganization of Semantic Brain Networks in Schizophrenia Revealed by fMRI. Schizophr Bull. 2023 Mar 15;49(2):498-506. doi: 10.1093/schbul/sbac157.
2. Takeuchi H, Yahata N, Lisi G, Tsurumi K, Yoshihara Y, Kawada R, Murao T, Mizuta H, Yokomoto T, Miyagi T, Nakagami Y, Yoshioka T, Yoshimoto J, Kawato M, Murai T, Morimoto J, Takahashi H. Development of a classifier for gambling disorder based on functional connections between brain regions. Psychiatry Clin Neurosci. 2022 Jun;76(6):260-267. doi: 10.1111/pcn.13350.
3. Yoshihara Y, Lisi G, Yahata N, Fujino J, Matsumoto Y, Miyata J, Sugihara GI, Urayama SI, Kubota M, Yamashita M, Hashimoto R, Ichikawa N, Cahn W, van Haren NEM, Mori S, Okamoto Y, Kasai K, Kato N, Imamizu H, Kahn RS, Sawa A, Kawato M, Murai T, Morimoto J, Takahashi H. Overlapping but Asymmetrical Relationships Between Schizophrenia and Autism Revealed by Brain Connectivity. Schizophr Bull.
4. 2020 Apr 17;46(5):1210-8. doi: 10.1093/schbul/sbaa021.
5. Tei S, Kauppi JP, Jankowski KF, Fujino J, Monti RP, Tohka J, Abe N, Murai T, Takahashi H, Hari R. Brain and behavioral alterations in subjects with social anxiety dominated by empathic embarrassment. Proc Natl Acad Sci U S A. 2020 Feb 25;117(8):4385-4391. doi: 10.1073/pnas.1918081117.
6. Takahashi H, Kato M, Matsuura M, Mobbs D, Suhara T, Okubo Y. When your gain is my pain and your pain is my gain: neural correlates of envy and schadenfreude. Science. 2009 Feb 13;323(5916):937-9. doi: 10.1126/science.1165604.

Biography

So Takasugi – Graduate student

Department of Physiology, Keio University School of Medicine

Research Overview

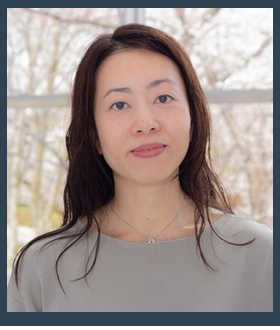
My research focuses on the molecules that regulate synapse functions (synapse organizers) in the peripheral nervous systems (PNS).

Our laboratory has studied the C1q complement family as the synapse organizers in the central nervous systems (CNS) and revealed their function and association with some neurological disorders (synaptopathy) in the CNS.

By revealing their localization and function in the PNS, I aim to understand better the physiological function of the synapses and develop new therapeutic strategies for neurological disorders in the PNS.

So far, I discovered that Cbln1, a member of the C1q complement family proteins, is expressed in the DRG neurons, and it has an essential role in the transduction of certain types of itch sensation that might be related to hypersensitivity in autism spectrum disorders or chronic itch state in atopic dermatitis.

Biography



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UK-Japan Neuroscience Symposium
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31 August – 2 September 2023

Masako Tamaki, Ph.D.

RIKEN Hakubi Team Leader, Cluster for Pioneering Research / Center for Brain Science, RIKEN

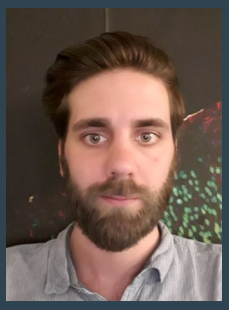
Research Overview

Why do we sleep? Although this question has been asked for centuries, it remains enigmatic. Our research interests lie in understanding how the sleeping brain facilitates healthy cognitive functions in humans. Notably, after only one night of sleep or a nap, our skills can dramatically improve to a level that surpasses our past performances, without any additional practice. By combining psychophysics, multimodal neuroimaging, and physiological measurements, our research has elucidated the neural mechanisms of visual and motor learning during sleep, clarified the mechanisms of sleep disturbances, and led to the development of a novel method to investigate dreaming, and more recently, investigations of cerebrospinal fluid dynamics during sleep.

Selected Publications

1. Uji M, Tamaki M. Sleep, learning, and memory in human research using noninvasive neuroimaging techniques. Neuroscience Research, 189, 66-74, 2023.
2. Tamaki M, Sasaki Y. Sleep-dependent facilitation of visual perceptual learning is consistent with a learning-dependent model. Journal of Neuroscience, 42(9), 1777-1790, 2022.
3. Tamaki M, Wang Z, Barnes-Diana T, Guo D, Berard AV, Walsh EG, Watanabe T, Sasaki Y. Complementary contributions of NREM and REM sleep to visual learning. Nature Neuroscience, 23(9), 1150-1156, 2020.
4. Tamaki M†, Berard AV†, Barnes-Diana T, Siegel J, Watanabe T, Sasaki Y. Reward does not facilitate visual perceptual learning until sleep occurs. Proceedings of the National Academy of Sciences of the United States of America, 117(2), 959-968, 2020. †Co-first authorship
5. Tamaki M, Bang JW, Watanabe T, Sasaki Y. Night watch in one brain hemisphere during sleep associated with the first-night effect in humans. Current Biology, 26(9), 1190-1194, 2016.
6. Tamaki M, Huang TR, Yotsumoto Y, Hämäläinen M, Lin FH, Náñez JE Sr, Watanabe T, Sasaki Y. Enhanced spontaneous oscillations in the supplementary motor area are associated with sleep-dependent offline learning of finger-tapping motor-sequence task. Journal of Neuroscience, 33(34), 13894-13902, 2013.
7. Horikawa T, Tamaki M, Miyawaki Y, Kamitani Y. Neural decoding of visual imagery during sleep. Science, 340 (6132), 639-642, 2013.

Biography



Jasper Teutsch, M.Sc.

PhD Student, Adaptive Decisions Lab, Newcastle University

Research Overview

My main research interests are adaptive decision-making, learning and relearning, and conceptual representations and their relation to value. I implemented and refined an operant working memory and attention task for mice, enabling performance tests during pharmacological and optogenetic perturbations and large-scale screening of disease models. Currently, I am conducting head-fixed reversal learning experiments using longitudinal In vivo two-photon Ca2+ imaging and behavioral measurements to investigate experience-induced changes in hierarchical inter-areal computations between prefrontal and primary sensory areas, finding that subpopulation in somatosensory cortex remapped according to task context and reflect reward history dependent on prediction error signalling from the orbitofrontal cortex.

Selected publications

1. Banerjee A, Teutsch* J, Parente* G, Lewis C, and Helmchen F (2020); Value-guided remapping of sensory circuits by lateral orbitofrontal cortex in reversal learning Nature 585, pages245–250

2. Teutsch J, Dennis K (2019). Operant assessment of DMTP spatial working memory in mice; Front. Behav. Neurosci. 13:193

3. Banerjee A, Wang BA, Teutsch J, Helmchen F, Pleger B (2023). Analogous cognitive strategies for tactile learning in the rodent and human brain; Progress in Neurobiology 222

4. Gigliucci V, Teutsch J, Woodbury-Smith M, Luoni M, Busnelli M, Chini B, Banerjee A (2021); Region-Specific KCC2 Rescue by rhIGF-1 and Oxytocin in a Mouse Model of Rett Syndrome Cerebral Cortex bhab388

Biography

Dr. Cezar M. Tigaret

Hodge Lecturer in Neuroscience, NMHII, School of Medicine, Cardiff University

Research overview

I am interested in understanding the causality between psychiatric risk factors and the emergence of symptoms and cognitive deficits in psychiatric illness, underpinned by alterations in neural synaptic and circuit functions. I have a special interest in synaptic plasticity as a principal cellular mechanism of associative learning, which forms the basis of our capacity to adapt to the environment and is disrupted in psychosis. I combine state-of-the-art two-photon imaging and ex vivo slice electrophysiology in animal models and in silico modeling techniques.

Selected publications

1. Rodrigues, Y.E., Tigaret, C.M., Marie, H., O'Donnell, C., Veltz, R.. (2023) A stochastic model of hippocampal synaptic plasticity with geometrical readout of enzyme dynamics. eLife, 12:e80152, doi: 10.7554/eLife.80152.
2. Tigaret, C.M., Lin, T-C.E., Morrell, E.R., Sykes, L., Moon, A.L. Moon, O'Donovan, M.C., Owen, M.J., Wilkinson, L.S., Jones, M.W., Thomas, K.L., Hall, J. (2021). Neurotrophin receptor activation rescues cognitive and synaptic abnormalities caused by hemizygosity of the psychiatric risk gene cacna1c. Mol Psychiatry, doi: 10.1038/s41380-020-01001-0.
3. Tigaret, C.M., Chamberlain, S.E.L., Sadowski, J.H.L.P., Ashby, M.C., Mellor, J.R. (2018) Convergent metabotropic signalling pathways inhibit SK channels to promote synaptic plasticity in the hippocampus. J Neurosci. doi:10.1523/JNEUROSCI.1160-18.2018.
4. Tigaret C.M., Olivo V., Sadowski J.H.L.P., Ashby M.C., and Mellor J.R. (2016) Coordinated activation of distinct Ca2+ sources and metabotropic glutamate receptors encodes Hebbian synaptic plasticity. Nature Communications, 7:10289, 2016.
5. Sainlos, M., Tigaret, C., Poujol, C., Olivier, N.B., Bard, L., Breillat, C., Thiolon, K., Choquet, D., Imperiali, B. (2011) Biomimetic divalent ligands for the acute disruption of synaptic AMPAR stabilization. Nat. Chem. Biol., 7(2): 81-91.
6. Opazo, P., Labrecque, S., Tigaret, C.M., Frouin, A, Wiseman, O. W., De Noninck, P., Choquet, D. (2010) CaMKII triggers the diffusional trapping of surface AMPARs through phosphorylation of stargazin. Neuron, 67(2):239-252.

Dr Makoto Uji

Research Scientist, Cognitive Somnology RIKEN Hakubi Research Team (PI: Dr Masako Tamaki), RIKEN CBS

Research Overview

My research interest is to better understand how the human sleeping brain works, the functional role of sleep in maintaining healthy cognitions and brain maintenance, and also to unveil a link between the healthy brain functions and aging brain during sleep. I have applied non-invasive neuroimaging methods (EEG, fMRI, simultaneous EEG-fMRI) to study human brain function. Especially, for the last 5 years, I have been applying the simultaneous EEG-fMRI method in sleep and sleep disorder studies.

Selected Publications

1. Uji, M., Tamaki, M. (2023). Sleep, learning, and memory in human research using noninvasive neuroimaging techniques. Neuroscience Research. https://doi.org/10.1016/j.neures.2022.12.013.
2. Uji, M., Cross, N., Pomares, F.B., Perrault, A.A., Jegou, A., Nguyen, A., Aydin, U., Lina, J-M., Dang-Vu, T.T., Grova, C. (2021). Data-driven beamforming techniques to attenuate ballistocardiogram (BCG) artifacts in EEG-fMRI without detecting cardiac pulses in electrocardiography (ECG) recordings. Human Brain Mapping. https://doi.org/10.1002/hbm.25535.
3. Uji, M., Wilson, R., Francis, S.T., Mullinger, K.J., Mayhew, S.D. (2018). Exploring the advantages of multiband fMRI with simultaneous EEG to investigate coupling between gamma frequency neural activity and the BOLD response in humans. Human Brain Mapping. https://doi.org/10.1002/hbm.23943.

Biography

Dr Stuart Williams

Postdoctoral research associate, Cardiff University

Research Overview

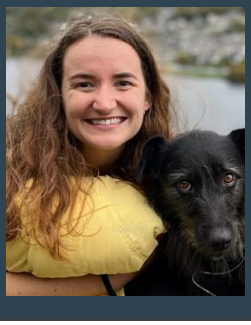
Currently my research is focused on cortico-cortical feedback circuits in the integration of sensory information, with a particular focus on feedback circuits within the somatosensory cortex. To understand these feedback circuits, we utilise in vivo calcium imaging techniques and optogenetics in combination with rodent behaviour in the form of a tactile discrimination task. Previously my doctoral research focused on how hippocampal GABA dysfunction, which has been implicated in brain disorders including schizophrenia and Alzheimer's disease, leads to changes in cognition and connected neural networks using rodent behaviour and imaging techniques.

Selected Publications

S. A. Williams, M. Gwilt, R. Hock, C. Taylor, J. Loayza, C.W. Stevenson, H.J. Cassaday, T. Bast. In Press (2021) Hippocampal disinhibition reduces contextual and elemental fear conditioning while sparing the acquisition of latent inhibition. eNeuro.

S. Kohli, M.V. King, S. Williams, A. Edwards, T.M. Ballard, L. J. Steward, D. Alberati & K. C. F. Fone. (2019) Oxytocin attenuates phencyclidine hyperactivity and increases social interaction and nucleus accumbens dopamine release in rats. Neuropsychopharmacology, 44, 295–305.

Biography



Dr. Charlotte Wiltshire

Lecturer in Psychology, Bangor University

Research overview

My research aims to understand the neural control of speech production, with a specific interest in developmental stuttering. My work combines brain stimulation techniques (Transcranial Magnetic Stimulation), brain imaging (functional MRI, Multi-Parametric-Mapping) and articulography (Electromagneticarticulography, vocal tract MRI) to investigate the neural processes underlying the initiation and inhibition of speech movements in people who stutter and people who are typically fluent. I completed my DPhil at the University of Oxford in 2020. During my DPhil, I contributed to a large randomised control trial looking at whether brain stimulation (tDCS) can enhance fluency in people who stutter (INSTEP trial).

Selected publications

1. Wiltshire, C. E. E. & Hoole, P. (Stage 1 Registered Report IPA). The role of the Supplementary Motor Area in Speech Production: Evidence from People Who Do, and Do Not Stutter. Brain Communications.
2. Lu, Y., Wiltshire, C. E. E., Chiew, M., Watkins, K. E., & Goldstein., L. (2022). Characteristics of articulatory gestures in stuttered speech: A case study using real-time magnetic resonance imaging. Journal of Communication Disorders.
3. Cler, G., Krishnan, S., Papp, D., Wiltshire, C. E. E., Chesters, J., & Watkins., K. E. (2021). Grey matter microstructural differences in developmental stuttering. Brain.
4. Wiltshire, C. E. E., Chiew, M., Chesters, J., Healy, M. P., & Watkins, K. E. (2021). Speech Movement Variability in People Who Stutter: A Vocal Tract Magnetic Resonance Imaging Study. Journal of Speech, Language, and Hearing Research.
5. Wiltshire, C. E. E., & Watkins, K. E. (2020). Failure of tDCS to modulate motor excitability and speech motor learning. Neuropsychologia.

Biography

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Hanna Wyszynska

PhD Student, Cardiff University

Research Overview

The aim of my PhD project is to investigate how memory consolidation is organised in the primary somatosensory cortex (S1) and the hippocampus during a rewarded tactile discrimination task. To achieve that, we use chemogenetics approach (DREADDs) to inhibit region of interest during different timepoints following the behavioural task. We also use novel Neuropixel probes to record neural activity in S1 and S2 both in anesthetised animals and in freely moving behaving animals. My PhD project integrates the expertise of two labs that focus on different anatomical structures involved in learning and memory – neocortex and hippocampus – and the development of computational models of learning and memory processes.

Selected Publications

1. Interdependence of primary and secondary somatosensory cortices for plasticity and texture discrimination learning. Anurag Pandey, Sungmin Kang, Nicole Pacchiarini, Hanna Wyszynska, Aneesha Grewal, Alex Griffiths, Imogen Healy-Millett, Zena Masseri, Neil Hardingham, Joseph O'Neill, Robert C. Honey, Kevin Fox. bioRxiv 2023.04.25.538217; doi: https://doi.org/10.1101/2023.04.25.538217.

Professor Makiko Yamada

National Institutes for Quantum Science and Technology

Research Overview

My research focuses on the neural and molecular mechanisms of cognitive biases. In particular, I am interested in how people are ignorant about themselves, how this ignorance arises from the brain, how it is expressed physically, and its effects on social adaptability and mental and physical health. To understand the brain mechanisms of cognitive biases, I attempt to quantify the magnitude of cognitive bias using psychological and psychophysical measures, and to integrate it with molecular systems measured by positron emission tomography (PET) and brain function measured by fMRI. In order to apply these basic researches in society, I am currently working as a project manager for the Moonshot R&D program, promoting the development of science and technology to improve human well-being through the use of cognitive biases.

Selected Publications

1. Isato A, Yokokawa K, Higuchi M, Suhara T, Yamada M. Resting-state functional connectivity relates to interindividual variations in positive memory. Behav Brain Res 419, 2022 Feb 15: 113663.
2. Kojima K, Hirano S, Kimura Y, Seki C, Ikoma Y, Takahata K, Ito T, Yokokawa K, Hashimoto H, Kawamura K, Zhang MR, Ito H, Higuchi M, Kuwabara S, Suhara T, Yamada M. Brain 5-HT2A receptor binding and its neural network related to behavioral inhibition system. Brain Imaging Behav. 2022 Jun;16(3):1337-1348.
3. Ito T, Kimura Y, Seki C, Ichise M, Yokokawa K, Kawamura K, Takahashi H, Higuchi M, Zhang MR, Suhara T, Yamada M. Histamine H3 receptor density is negatively correlated with neural activity related to working memory in humans. EJNMMI Res 8(1), 2018 Jun 14: 48.
4. Yokokawa K, Ito T, Takahata K, Takano H, Kimura Y, Ichise M, Ikoma Y, Isato A, Zhang MR, Kawamura K, Ito H, Takahashi H, Suhara T, Yamada M. Neuromolecular basis of faded perception associated with unreality experience. Sci Rep 8(1), 2018 May 23: 8062.
5. Ito T, Yokokawa K, Yahata N, Isato A, Suhara T, Yamada M. Neural basis of negativity bias in the perception of ambiguous facial expression. Sci Rep 7(1), 2017 Mar 24: 420.
6. Yamada M, Uddin LQ, Takahashi H, et al. Superiority illusion arises from resting-state brain networks modulated by dopamine. Proc Natl Acad Sci U S A 110(111), 2013 Mar 12: 4363-4367.
7. Yamada M, Camerer CF, Fujie S, et al. Neural circuits in the brain that are activated when mitigating criminal sentences. Nat Commun 3(759), 2012 Mar 27.

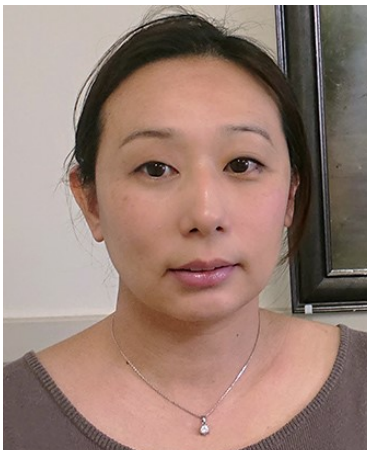
Organizing Committee

Professor Kevin Fox

Dr Gaynor Smith

Professor Kei Cho

Dr Akiko Hayashi-Takagi



Dr Tom Macpherson

Dr Jemeen Sreedhan

Professor Masahisa Katsuno

AMED and MRC

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Attendees from the Japan Agency for Medical Research and Development (AMED) and the Medical Research Council (MRC):

Dr Yasushi Ogasaka, Director of the Department of International Strategy, AMED.

Yumiko Miyashita, Deputy Director, Division of Basic Medical Research, AMED.

Martin Gadsden, London Liaison Associate Manager, AMED.

Dr Akira Yoshida, Manager for the Department of International Strategy, AMED.

Dr Mark Palmer, Director of International Relations, MRC.

AMED Japan Agency for Medical Research and Development

UKRI Medical Research Council

St Fagans Team Building

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"St Fagans National Museum stands in the grounds of the magnificent St Fagans Castle and gardens, a late 16th-century manor house donated to the people of Wales by the Earl of Plymouth in 1948.

Since 1948 over forty original buildings from different historical periods have been re-erected in the 100-acre parkland, among them houses, a farm, a school, a chapel and a splendid Workmen's Institute."

- St Fagan's Website

St Fagans Team Building

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Team Building Event Information

As part of the team building event, attendees will be split into small groups to complete activities. Please see below your assigned team number. Please note that these teams are based on those who indicated they would like to go on the excursion on their registration form however there is space remaining for additional attendees on the day. Please speak with a member of the organising committee for more details.

Team 1—Gaynor Smith, Stuart Williams, Tomoyuki Furuyashiki, So Takasugi

Team 2—Abidemi Otaiku, Hanna Wyszynska, Masanori Matsuzaki, Akihiro Funamizu

Team 3—Kei Cho, Cezar Tigaret, Tom Macpherson, RuixiangLi

Team 4—Uroosa Chughtai, Sungmin Kang, Atsushi Kasai, Hiroki Shiwaku

Team 5—Zeynep Okray, Chiara Franceschi, Akiko Hayashi-Takagi, Kotaro Oiwa

Team 6—Hannah Clarke, Masako Tamaki, Aurelio Cortese

Team 7—Leon Crowley, Maxime Assous, Makiko Yamada, Daisuke Ito

Team 8—Jemeen Sreedharan, Rachel Hills, Mari Shiozaki, Aya Ito-Ishida

Team 9—Charlotte Wiltshire, Natalie Connor-Robson, Daisuke Ito, Akira Yoshida

Team 10—Haruhiko Bito, Yumiko Miyashita, Haruhisa Inoue

The Vale Resort

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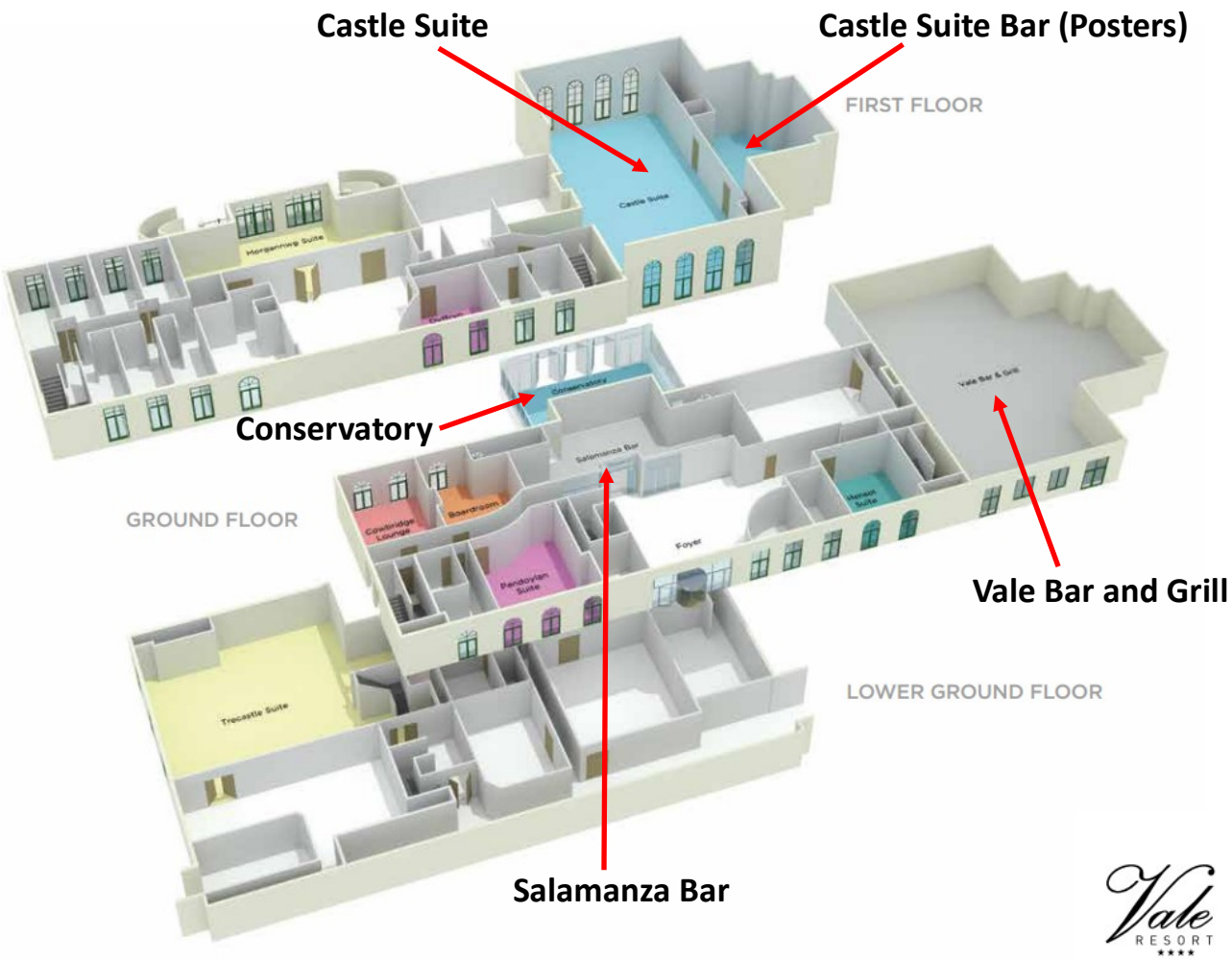

Useful Information

Breakfast will be in the Vale Grill each day.

Lunch is in the meeting room—Castle Suite Bar.

Dinner is in the Conservatory on Thursday 31st August and Saturday 2nd September and in the Castle Suite on Friday 1st September.

Drinks receptions are held within the Salamanza Bar.

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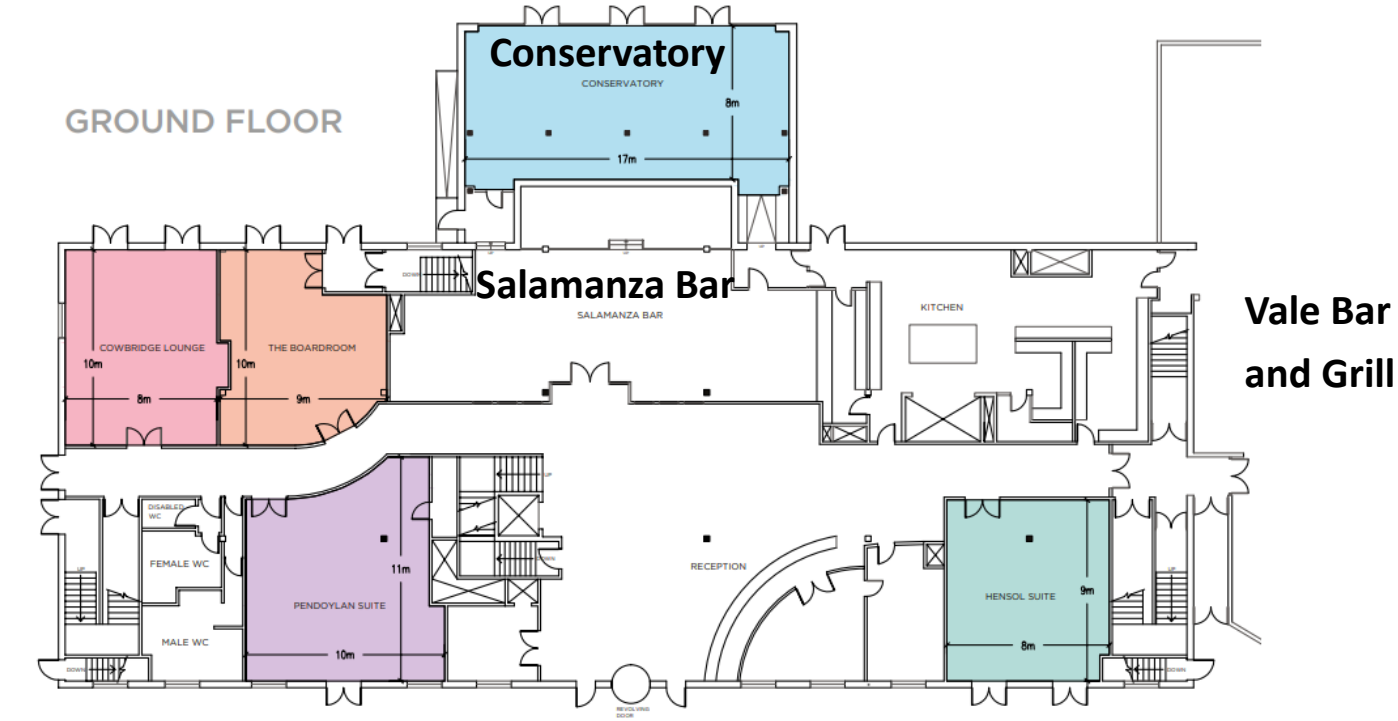
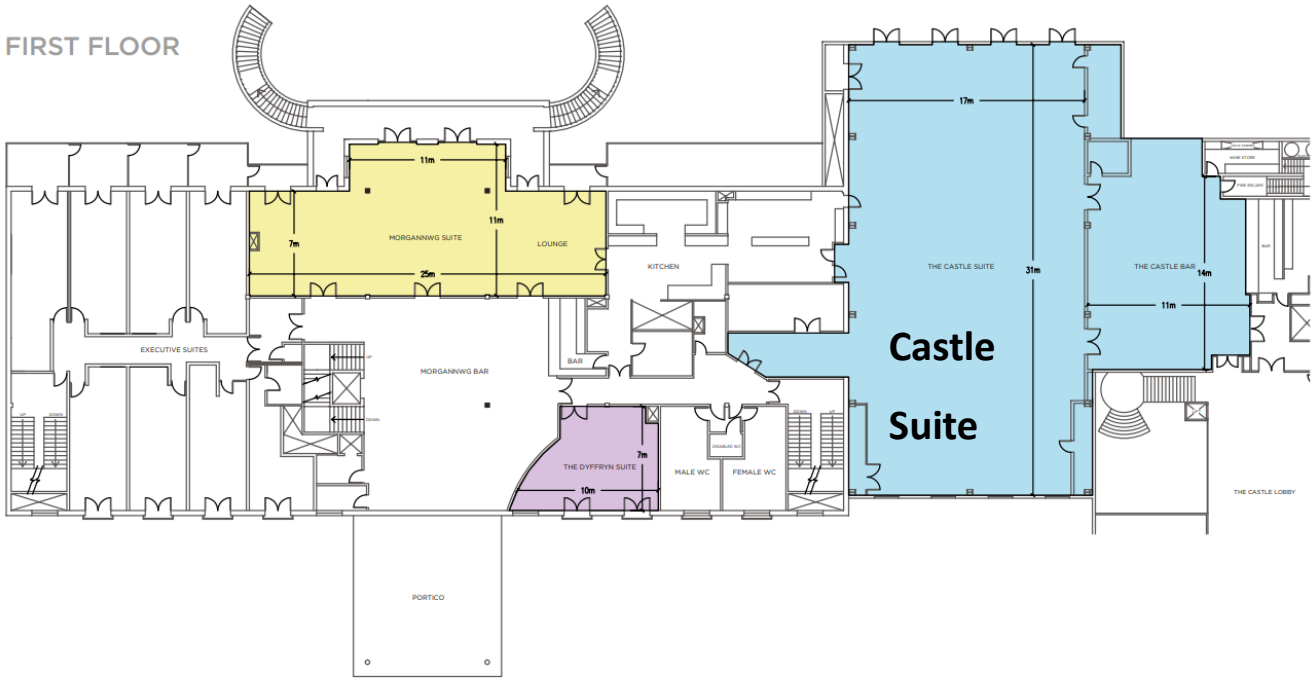


Vale Bar and Grill



Castle Suite

The Vale Resort

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The Vale Bar and Grill

Travel Information

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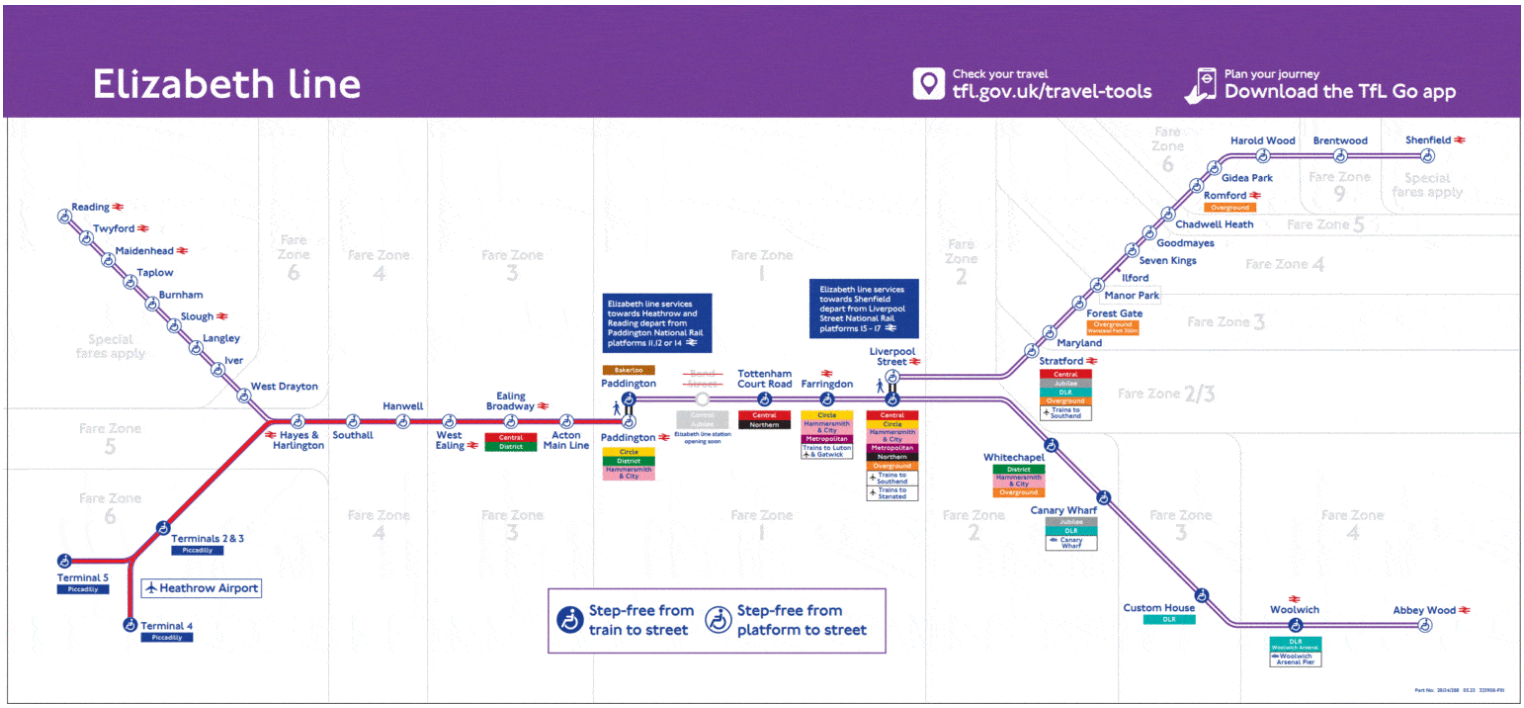
Train from Heathrow Airport to Cardiff, Taxi to the Vale Resort

Step 1: From Terminal 2, 3, 4 or 5 catch the train to Paddington Station as highlighted by the red line on the map below. This can be done by the Elizabeth Line or by the Heathrow Express.



Heathrow Express — Heathrow to Paddington

Mondays to Fridays (except Public Holidays)														
Heathrow, Terminal 5	0512	0527	0542	0557	Then every 15 minutes until	2157	2212	2227	2242	2257	2312	2327	2342	2357
Heathrow, Terminals 2&3 *arr.*	0515	0530	0545	0600		2200	2215	2230	2245	2300	2315	2330	2345	0001
Heathrow, Terminals 2&3 *dep.*	0517	0532	0547	0602		2202	2217	2232	2247	2302	2317	2332	2348	0003
London, Paddington	0533	0548	0603	0617		2217	2232	2248	2304	2317	2333	2347	0008	0023

Saturdays													
Heathrow, Terminal 5	0512	0527	0542	0557	Then every 15 minutes until	2127	2142	2157	2212	2227	2242	2257	2312	2327	2342	2357
Heathrow, Terminals 2&3 *arr.*	0515	0530	0545	0600		2130	2145	2200	2215	2230	2345	2300	2315	2330	2345	0001
Heathrow, Terminals 2&3 *dep.*	0517	0532	0547	0602		2132	2147	2202	2217	2232	2247	2302	2317	2332	2347	0003
London, Paddington	0532	0548	0602	0617		2147	2202	2218	2232	2248	2302	2317	2332	2347	0003	0018

Sundays & Public Holidays																							
Heathrow, Terminal 5	---	0527	Then every 15 minutes until	0657	0712	0727	0742	0757	0812	0827	Then every 15 minutes until	2127	2142	2157	2212	2227	2242	2257	2312	2327	2342	2357
Heathrow, Terminals 2&3 *arr.*	---	0530		0700	0715	0730	0745	0800	0815	0830		2130	2145	2200	2215	2230	2245	2300	2315	2330	2345	0001
Heathrow, Terminals 2&3 *dep.*	0517	0532		0702	0717	0732	0747	0802	0817	0832		2132	2147	2202	2217	2232	2247	2303	2317	2332	2347	0003
London, Paddington	0533	0548		0718	0735	0750	0804	0818	0834	0848		2148	2203	2219	2233	2248	2303	2320	2337	2352	0008	0020

Step 2: From Paddington you can catch a train to Cardiff Central or to Swansea (which has a stop in Cardiff Central). Please exit the train at Cardiff Central station. Please use the link below to find specific train times. https://www.thetrainline.com/en-us

Step 3: As you come out of Cardiff Central Station there will be taxis available to the right of the main entrance. The postcode for the Vale Resort is CF72 8JY.

Airport Coach Information

30th Aug

From Heathrow to the Vale Resort Hotel:

The coach driver will track NH211 (HND>Heathrow) that is scheduled to land at 16:20 on 30th August at Terminal 2. The coach driver will wait for everyone to clear customs before coming to the pick up point.

**Please note passengers arriving on JL041 (landing at 06:25), TG910 (07:15), EK313/Y (14:40), EK29 (14:45), BA008 (15:30), JL43 (15:50), or other earlier flights can also travel on this coach, but please make your way to Terminal 2 arrivals hall where AMED staff (Yumiko Miyashita) will meet you.*

3rd Sept

From the Vale Resort Hotel to Heathrow:

A coach will depart for Heathrow airport at 8AM, from outside the Vale Resort.

**estimated arrival time at Heathrow is 10:40*

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Check-in Information

Attendees will all require a credit/debit card to check in. Please note that this will not be charged for your accommodation. It is only used for cases where the minibar has been used for example.

30th August (Japanese attendees only)

The coach will arrive at the hotel approximately between 7:30pm-9pm on the 30th August. At attendees own discretion there will be opportunity to order food from the Vale Bar and Grill until 9pm and room service will be available after this time.

31st August

Check-in to the Vale Resort on the 31st August will be from 3pm. There will be a secure place to leave your bag at the hotel Reception as you arrive. There will be a chance to check in during the breaks or before the opening dinner.

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Maxime Assous	AssousM@cardiff.ac.uk
Abhishek Banerjee	abhi.banerjee@newcastle.ac.uk
Omer Bayraktar	ob5@sanger.ac.uk
Haruhiko Bito	hbito@m.u-tokyo.ac.jp
Kathryn Bowles	kbowles@ed.ac.uk
Verity Brown	vjb@st-andrews.ac.uk
Jackie Casey	ucbtjm4@ucl.ac.uk
Kei Cho	kei.cho@kcl.ac.uk
Uroosa Chughtai	ChughtaiUA@cardiff.ac.uk
Hannah Clarke	clarkehj@cardiff.ac.uk
Natalie Connor-Robson	connor-robsonnl@cardiff.ac.uk
Johnathan Cooper-Knock	j.cooper-knock@sheffield.ac.uk
Aurelio Cortese	cortese_a@atr.jp
Dezerae Cox	dc597@cam.ac.uk
Leon Crowley	leon.crowley@kcl.ac.uk
Kevin Fox	Foxkd@cardiff.ac.uk
Chiara Franceschi	FranceschiC@Cardiff.ac.uk
Akihiro Funamizu	funamizu@iqb.u-tokyo.ac.jp
Tomoyuki Furuyashiki	tfuruya@med.kobe-u.ac.jp
Martin Gadsden	m.gadsden@amedjp-uk.org
Jenna Gregory	jenna.gregory@abdn.ac.uk
Jeremy Hall	hallj10@cardiff.ac.uk
Taku Hatano	thatano@juntendo.ac.jp
Akiko Hayashi-Takagi	akiko.hayashi-takagi@riken.jp
Rachel Hills	HillsR2@cardiff.ac.uk
Jennifer Imm	j.l.imm@exeter.ac.uk
Haruhisa Inoue	inoue-g@cira.kyoto-u.ac.jp
Daisuke Ito	ito.daisuke.k4@f.mail.nagoya-u.ac.jp
Aya Ito-Ishida	aya.ishida@riken.jp
Matt Jones	matt.jones@bristol.ac.uk
Sungmin Kang	KangS4@cardiff.ac.uk
Atsushi Kasai	kasai@phs.osaka-u.ac.jp
Masahisa Katsuno	ka2no@med.nagoya-u.ac.jp
Yuka Koike	yukkoike@bri.niigata-u.ac.jp
Wei-Li (William) Kuan	wlk21@cam.ac.uk

Contact Information

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Ruixiang Li	ruixiang.li@riken.jp
Tom Macpherson	macpherson@protein.osaka-u.ac.jp
Vaishnavi Manohar	vaishnavi.1.manohar@kcl.ac.uk
Tom Massey	MasseyT1@cardiff.ac.uk
Masanori Matsuzaki	mzakim@m.u-tokyo.ac.jp
Stephen McHugh	stephen.mchugh@pharm.ox.ac.uk
Scott Mitchell	scott.mitchell@kcl.ac.uk
Yumiko Miyashita	yumiko-miyashita@amed.go.jp
Wendy Noble	w.noble2@exeter.ac.uk
Yasushi Ogasaka	yasushi-ogasaka@amed.go.jp
Toshihihisa Ohtsuka	tohtsuka@yamanashi.ac.jp
Kotaro Oiwa	k.oiwa@med.nagoya-u.ac.jp
Zeynep Okray	Zeynep.Okray@dpag.ox.ac.uk
Abidemi Otaiku	a.otaiku@nhs.net
Mark Palmer	mark.palmer@mrc.ukri.org
Beverley Plummer	plummerbj@cardiff.ac.uk
Trevor Robbins	twr2@cam.ac.uk
Martina Sassi	martina.sassi@swansea.ac.uk
Mari Shiozaki	mari.z6@keio.jp
Hiroki Shiwaku	shiwaku.npat@mri.tmd.ac.jp
Gaynor Smith	SmithGA@Cardiff.ac.uk
Jemeen Sreedharan	jemeen.sreedharan@kcl.ac.uk
Masami Suzukake	suzukake-ms@igakuken.or.jp
Koichi Tabata	tabata-ki@igakuken.or.jp
Hidehiko Takahashi	hidepsyc@tmd.ac.jp
So Takasugi	sotakasugi95@gmail.com
Masako Tamaki	masako.tamaki@riken.jp
Bhuvaneish Thangaraj Selvaraj	bthangar@ed.ac.uk
Cezar Tigaret	TigaretC@cardiff.ac.uk
Makoto Uji	makoto.uji@riken.jp
Stuart Williams	williamss169@cardiff.ac.uk
Charlotte Wiltshire	c.wiltshire@bangor.ac.uk
Hanna Wyszynska	wyszynskah@cardiff.ac.uk
Makiko Yamada	yamada.makiko@qst.go.jp
Akira Yoshida	akira-yoshida@amed.go.jp

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