Name: Cathy Abbott

Email: C.Abbott@ed.ac.uk

Brief Summary of Research Interests:
We are interested in the way in which mutations in translation elongation factors, and particularly eEF1A2, cause neurodevelopmental disorders (NDD) including epilepsy, autism and intellectual disability. In contrast, complete ablation of eEF1A2 causes catastrophic neurodegeneration. We also explore the switch between eEF1A variants during neuronal development and the implications of this switch for canonical and non-canonical functions of the variants. We use CRISPR/Cas9 gene editing to create mouse and cell based models of the NDD-causing missense mutations in eEF1A2 and carry out in-depth phenotyping to identify disease relevant end-points with which to assess therapeutic strategies.

Lab website URL:
https://www.ed.ac.uk/centre-genomic-medicine/research-groups/abbott-group

Recent/Relevant Publications:

Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
I have collaborated for some years with Maria Vera, who is just setting up her lab at McGill. She is a molecular biologist rather than a neuroscientist but we are doing complementary work on the role of translation elongation factors in proteostasis with implications for neurodevelopmental disorders and neurodegeneration.
**Name:** Dr Thomas H Bak

**Email:** thomas.bak@ed.ac.uk

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**Brief Summary of Research Interests:**
The main topic of my research is the relationship between language and other cognitive as well as motor functions in the brain, in healthy ageing as well as in neurodegenerative diseases such as dementia, motor neuron disease (MND) and parkinsonian syndromes including Progressive Supranuclear Palsy (PSP), Corticobasal Degeneration (CBD) and Multiple System Atrophy (MSA).

More recently, my research focused on the influence of language learning and multilingualism on cognitive ageing, stroke and dementia. My contributions to this field include the largest study of the influence of bilingualism on dementia and stroke as well as the first studies examining the impact of bilingualism on cognitive ageing controlling for childhood intelligence. The results point to a possible role of language learning and use as a mechanism of cognitive reserve counteracting the effects of brain pathology.

A continuation of this research, which would combine both topics of my previous work (cognition in movement disorders and bilingualism) would be to examine the effects of bilingualism on Parkinson's Disease and atypical parkinsonian subjects; Montreal, as one of the leading centres of bilingualism research in the world would be an ideal place to conduct this type of research.

**Lab website URL:**
[https://www.ed.ac.uk/profile/thomas-bak](https://www.ed.ac.uk/profile/thomas-bak)
[http://healthylinguisticdiet.com/](http://healthylinguisticdiet.com/)

**Recent/Relevant Publications:**

**Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.**
I have been in touch with Prof. Debra Titone, one of the leading researchers in cognitive science of multilingualism, with connections to clinicians working with parkinsonian patients: [https://www.mcgill.ca/psychology/debra-titone](https://www.mcgill.ca/psychology/debra-titone)
Brief Summary of Research Interests:
Currently, neurodegenerative diseases are attracting the interest of the scientific community due to the dramatic increase in the number of cases and their impact on public health. Many of these diseases, such as Alzheimer’s disease, Parkinson’s disease, Huntington’s disease and prion diseases, share common features and molecular patterns including protein misfolding, protein aggregation, and cell death. There is an unrevealed understanding on the specific mechanisms of how these aggregation process starts, and how they are implicated with complex pathways to trigger finally toxicity and neuronal loss. Prion research has been utilized to reveal details of protein misfolding disorders expanding the possibility to develop an early diagnostic method and future treatments for prions disease and to other protein misfolded disorders.

Dr Barria is currently the Head of the Protein Biochemistry Laboratory at the National CJD Research & Surveillance Unit, Centre for Clinical Brain Sciences, University of Edinburgh. His research interests lie in the field of neurodegenerative disorders, and for the last two decades he has been investigating the molecular pathology of prion diseases using tissue-base approaches, and animal and in vitro models. Prion diseases are associated with the conformational change of a normal protein, termed the prion protein, to a misfolded isoform. They can affect both animals and humans, and they are transmissible and fatal. Dr Barria current research interests are to explore (i) the molecular basis of human and animal prion diseases and their potential for zoonotic transmission; (ii) the effect of cofactors which may influence prion conversion; (iii) the events that trigger their spontaneous formation; and (iv) developing of highly sensitive molecular tools for the early diagnosis of neurodegenerative illnesses.

Recent/Relevant Publications:

Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.

No
Name: Dr David P. Breen

(Wellcome Clinical Research Career Development Fellow and Honorary Consultant Neurologist, University of Edinburgh)

Email: David.Breen@ed.ac.uk

Brief Summary of Research Interests:
I am a movement disorders neurologist. In 2016, after completing neurology training, I won an Edmond J. Safra Fellowship via the Michael J. Fox Foundation to spend two years in Toronto under the mentorship of Prof. Anthony Lang. I have since returned to Edinburgh to lead the development of the Parkinson’s disease (PD) clinical research infrastructure.

My previous research was novel in shedding light on the causes of sleep and circadian rhythm disruption in early PD. We found that sleep fragmentation was common, related to reduced 24-hour melatonin output, and correlated with hypothalamic volume loss. We also uncovered fundamental alterations in peripheral clock gene expression. I continue to be interested in the influence of sleep and circadian disruption on brain health. I have forged new collaborations with large prospective cohort studies in order to study subjective (questionnaires) and objective (accelerometry-derived) sleep and circadian measures and relate them to brain health outcomes in healthy individuals, both cross-sectionally (cognitive performance, brain MRI) and longitudinally (incident AD/PD, post-mortem neuropathology).

I have considerable experience in PD cohort studies. Alongside other colleagues, I ran two longitudinal cohort studies (PICNICS and ICICLE-PD), which continue to identify new mechanisms and predictors of cognitive impairment in PD.

Lab website URL:
http://annerowlingclinic.org/people/dr-david-breen

Recent/Relevant Publications:


*Denotes joint authors

Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.

No
Name: Paula J Brunton

Email: p.j.brunton@ed.ac.uk

Brief Summary of Research Interests:
Our research focusses on the long-term impact of the early life environment on the development and neuronal organisation of the brain. Our main areas of interest involve understanding the consequences of early life stress exposure on subsequent physiology and behaviour and the underlying central mechanisms. We have developed an ethologically relevant social stress paradigm for pregnant rats to study the effects of adverse early life environment on the development of various systems in the offspring. We have demonstrated that maternal exposure to social stress is linked with low birth weight, anxious behaviour, hyperactive stress axis activity, insulin resistance, cognitive deficits and abnormal social and behaviours in the offspring. Current projects are focused on understanding: (i) the central mechanisms involved in early life programming of the brain and behaviour and whether these adverse effects can be prevented or reversed; (ii) the mechanisms involved in transmission of maternal stress effects from the mother to the fetus; (iii) the role of the gut microbiota in stress axis dysfunction and anxiety behaviour in developmentally programmed rats; and (iv) the cumulative effects of stress and inflammatory challenge in early life on the development of mood disorders in adulthood.

Lab website URL:
https://www.ed.ac.uk/discovery-brain-sciences/our-staff/research-groups/dr-paula-brunton

Recent/Relevant Publications:

Do you have an existing collaboration with a colleague at McGill?  If so, please give brief details.
No
Name: Emanuel Busch

Email: emanuel.busch@ed.ac.uk

Brief Summary of Research Interests:
The global aim of my research is to understand the mechanisms that govern the stability and plasticity of neural circuit function across a lifetime; how neural circuits dynamically process and store information about the outside world to generate appropriate behavioural output. Little is known about how signalling in neural circuits dynamically changes to adapt to different contexts but can also be reliably sustained over long periods of time – and how these circuit functions deteriorate with age. We use the well-defined C. elegans neurons that mediate oxygen responses as an exemplar for neural circuit function. These neurons set the long-term behavioural state of C. elegans, operate over broad time frames and show complexity on multiple levels – their output integrates sensory integration, shows experience-dependent plasticity, is shaped by environmental context and by genetic background. We dissect this sensory processing at the molecular, cellular, circuit and behavioural level with the help of the strong experimental toolbox available in C. elegans. We have found, for example, that the decline of experience-dependent plasticity of oxygen responses with age depends on environmental stress levels. We also have a strong interest in gap junctions and explore the plasticity of electrical coupling in neural circuits of C. elegans.

Lab website URL:
https://www.ed.ac.uk/discovery-brain-sciences/our-staff/research-groups/emanuel-busch

Recent/Relevant Publications:

Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
No
Name: Mike Cousin

Email: M.Cousin@ed.ac.uk

Brief Summary of Research Interests:
My research investigates how presynaptic function adapts to maintain neurotransmission across the range physiological inputs. This includes recruitment of specific SV endocytosis modes and SV cargo retrieval mechanisms. To answer these questions we use a multi-disciplinary approach employing a variety of in vitro model systems which combine population and single cell fluorescent imaging of nerve terminals with functional, biochemical, cell biological and molecular biological techniques. We are increasingly making use of preclinical disease models to determine how dysfunctional SV recycling can impact on synaptic failure.

Our current projects include –

1) A molecular dissection of the process of activity-dependent bulk endocytosis and a determination of its physiological role in neurotransmission.

2) Investigations into how interactions between synaptic vesicle cargo molecules can determine their copy number on these vesicles.

3) A determination of the molecular mechanism underpinning activity-dependent presynaptic dysfunction in Huntington’s Disease (joint project with Dr. Karen Smillie).

4) Identification and correction of presynaptic dysfunction in a series of preclinical models of monogenic epilepsy, epileptic encephalopathy, intellectual disability and autism.

Lab website URL:
https://www.ed.ac.uk/discovery-brain-sciences/our-staff/research-groups/mike-cousin

Recent/Relevant Publications: (selected from total of 85):


Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
No.
Name: Paul De Sousa

Email: paul.desousa@ed.ac.uk

Brief Summary of Research Interests:
My track record over the last twenty years since leaving Canada (for what was supposed to be a 3 year adventure) has been in developmental and stem cell biology and biotechnology from animal cloning to human pluripotent stem cells. Most of this period I have been focused on development of applications centred on human induced and embryo pluripotent stem cell systems in biomedical discovery and as a source of products for tissue rejuvenation in neurodegenerative diseases. Ten years ago, I sought to transition into neuroscience following my wife’s genetic diagnosis of HD. In addition to HD I have interests in AD and Mucopolysaccharidosis Type III, drawing in collaborators with expertise and resources in these arenas. Current ongoing research is developing and using tools to sense and report oxidative stress via NRF2 activated responses and as a focus of therapeutic intervention. I also have an IP position and intent around human pluripotent stem cell derived mesenchymal cells as products to attenuate inflammation and as bio-vectors.

Lab website URL:
https://www.ed.ac.uk/profile/dr-paul-de-sousa

Recent/Relevant Publications:
5. Lineage-specific distribution of high levels of genomic 5-hydroxymethylcytosine in mammalian development https://www.nature.com/articles/cr2011113
7. Rapid establishment of the European Bank for induced Pluripotent Stem Cells (EBiSC) : The Hot Start experience(10 pages) 1 Apr 2017 In: Stem cell research, vol. 20, pp. 105-114 DOI: https://doi.org/10.1016/j.scr.2017.03.002
9. Development and production of good manufacturing practice grade human embryonic stem cell lines as source material for clinical application

Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
No
Name: Javier Escudero

Email: javier.escudero@ed.ac.uk

Brief Summary of Research Interests:
I create and apply data analysis tools to extract information from biomedical signals and clinical time series. My main aim is to reveal the subtle changes that major diseases (e.g., Alzheimer's and epilepsy) cause in the brain activity.

By developing and applying signal processing methods, I aim at increasing our understanding of how several brain conditions progress. Of particular interest is the evaluation of brain functional connectivity in both neurodevelopmental and neurodegenerative diseases to understand how they affect the way in which different brain regions interact with each other. I am also interested in the interplay between structure and function in the brain and in the application of pattern recognition techniques to highly-dimensional clinical datasets to support decision making.

Current Research Interests
- Multiway analysis
- Brain connectivity
- Network theory
- Multivariate signal processing
- Applications of pattern recognition to physiological data
- Electroencephalogram (EEG) and Electromyogram (EMG)
- Alzheimer's disease
- Epilepsy

Lab website URL:
http://www.research.ed.ac.uk/portal/jescuder

Recent/Relevant Publications:

Full list at:
http://www.research.ed.ac.uk/portal/jescuder https://scholar.google.co.uk/citations?user=slBtm3AAAAAJ&hl=en

Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
No
**Name:** Kathy Evans  

**Email:** Kathy.Evans@igmm.ed.ac.uk

**Brief Summary of Research Interests:**

We are interested in identifying biomarkers, understanding mechanisms and identifying treatment targets for neurodegenerative and psychiatric disorders. We utilise both computational modelling and cell-based analyses to improve understanding of these disorders at the level of the genotype, epigenome and cellular phenotype. A common aspect to these research strands is the use of various ‘omics techniques, i.e. characterisation of the epigenome, transcriptome and proteome, in order to facilitate an unbiased assessment of potential pathogenic mechanisms. For example, we are currently carrying out an epigenome-wide association study of dementia risk scores using DNA methylation data on thousands of subjects from Generation Scotland (https://www.ed.ac.uk/generation-scotland). Here we hope to understand mechanisms of pre-morbid risk for dementia. Such computational approaches are complemented by functional analyses of cellular phenotype. For example, we have used CRISPR/Cas9 genome editing in iPSC-derived neurons (and other neuronal cell lines) to generate mutations in the sortilin family, which comprises signalling and trafficking molecules linked to neurodegenerative and psychiatric conditions. These analyses are uncovering cellular mechanisms that may predispose to these illnesses. In the longer term, future plans include development of cellular assays amenable to high throughput drug screening.

**Lab website URL:**  
https://www.ed.ac.uk/centre-genomic-medicine/research-groups/evans-group

**Recent/Relevant Publications:**

As the functional work on neurodegenerative disorders is a recent change of direction for me, I don’t have any publications directly relevant to that yet. A recent publications (2018) plus a selection of previous relevant papers are listed below:


*Joint Senior Authors

Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.

No
Name: Alfredo Gonzalez-Sulser

Email: agonzal2@ed.ac.uk

Brief Summary of Research Interests:
Epilepsy is increasingly considered a disease of neuronal networks with multiple nodes within the brain contributing differentially to seizures. As a consequence, both pharmacological treatments and surgical resections to decrease the activity of epileptic foci often fail to prevent seizures, as although some nodes may be controlled, other anatomical regions may still be able to generate network pathologies.

We are recording network activity utilizing rodent EEG and intracranial LFP recordings and identifying electrophysiological biomarkers in both rodent models of neurodevelopmental disorders and temporal lobe epilepsy. Furthermore, we are researching populations of brain cells that may have powerful effects over network activity and utilizing optogenetics to modulate these cells in an attempt to ameliorate network abnormalities and block seizures. This may lead to translatable treatment strategies and an increase understanding of epileptic networks within the brain.

Lab website URL:
https://www.ed.ac.uk/discovery-brain-sciences/our-staff/research-groups/alfredo-gonzalez-sulser

Recent/Relevant Publications:


Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
No
Name: Seth Grant

Email: seth.grant@ed.ac.uk

Brief Summary of Research Interests:
My research aims to understand how the organisation of the synapse, in particular the postsynaptic proteome of excitatory synapses in the brain, informs fundamental mechanisms of learning, memory, and innate and learned behaviours and how these go awry in neurological dysfunctions including Alzheimer’s, depression, autism and intellectual disability. We are interested in the diversity of postsynaptic proteins and the dynamics of their assembly into complexes and supercomplexes in different neurons and brain regions. These studies have also provided insight into synapse evolution and the establishment of vertebrate behavioural complexity. We discovered the Genetic Lifespan Calendar, a genomic programme that could explain how schizophrenia susceptibility genes are timed to exert their effects in young adults. Recently, we have produced the first synaptome maps of the whole mouse brain, revealing the molecular and morphological features of a billion synapses. This has uncovered unprecedented spatiotemporal synapse diversity organised into an architecture that correlates with the structural and functional connectomes, and shown how mutations that cause cognitive disorders reorganise these synaptome maps. We are now exploiting this technology to characterise how the brain changes during normal development and with degeneration, with a view to facilitating new pathways to diagnosis and therapy.

Lab website URL:
www.genes2cognition.org

Recent/Relevant Publications:
6. Skene NG, Roy M, Grant SGN (2017). A genomic lifespan program that reorganizes the young adult brain is targeted in schizophrenia. eLIFE 6, e17915.

Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
No
**Name:** Sebastian Greiss

**Email:** s.greiss@ed.ac.uk

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**Brief Summary of Research Interests (200 words max):**

*Using Genetic Code Expansion for Precise in vivo Control of Protein Function*

My lab develops tools based on genetic code expansion, which allows the site-specific introduction of chemically synthesized non-canonical amino acids (ncAA) into proteins in vivo. While we mostly focus on *C. elegans* at present, genetic code expansion can be easily applied to cultured cells and has been established in higher organisms such as mice and zebrafish.

To expand the genetic code we modify the translational machinery by adding an archael aminoacyl-tRNA-synthetase/tRNA_{CUA} pair. The archael tRNA_{CUA} then incorporates the ncAA at an amber stop codon introduced into the gene of interest.

We can use photo-caged amino acids to create photo-activatable versions of potentially any protein by introducing such a caged amino acid into the catalytic site of an enzyme in lieu of the native residue, thus rendering the enzyme inactive. Short exposure to 365nm light can then be used to remove the caging group and activate the enzyme either globally or using a targeted laser.

Other available ncAA include amino acids carrying chemical handles for site specific labeling of proteins, photo-crosslinking amino acids, or amino acids to site specifically install post translational modifications.

We are interested in collaborations aimed at dissecting biological processes in any organisms of interest, which could benefit from the possibilities offered by genetic code expansion / ncAA.

**Lab website URL:**

https://www.ed.ac.uk/discovery-brain-sciences/our-staff/research-groups/sebastian-greiss

**Recent/Relevant Publications:**


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**Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.**

In discussions with Prof. Oliver Hardt, Department of Psychology, McGill.
**Name:** Dr Mandy Jackson

**Email:** mandy.jackson@ed.ac.uk

**Brief Summary of Research Interests:**

My lab is particularly interested in a group of inherited diseases (spinocerebellar ataxias) where patients lose the ability to coordinate their movement due to a change in the activity and then subsequent death of Purkinje cells. Using a variety of techniques we are investigating what cellular processes underlie the decline in cerebellar function and with this information attempting to develop therapeutic interventions to improve the physical wellbeing of individuals afflicted with SCA. Furthermore because the cerebellum is increasingly being implicated in a wide variety of clinical disorders, such as Alzheimer’s disease, Parkinson’s, schizophrenia, autism, cognitive cerebellar affective syndrome, dyslexia, depression and other cognitive and emotional deficits we are also interested in investigating cerebellar dysfunction in these disorders and identifying common mechanisms.

**Lab website URL:**
https://www.ed.ac.uk/discovery-brain-sciences/our-staff/research-groups/mandy-jackson

**Recent/Relevant Publications:**


**Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.**

No
Name: David Lyons

Email: David.lyons@ed.ac.uk

Brief Summary of Research Interests:
We are interested in understanding the mechanisms underpinning the formation, health and function of myelinated axons in vivo. Our lab uses zebrafish as a model system, due to their amenability for live cell imaging and high-resolution cellular analyses, their ability to be used to carry out large-scale genetic and chemical screens, and their relatively simple nervous system with well characterised circuitry that now allows system-level interrogation of function. To support our work in zebrafish we have a wide range of collaborators with expertise in in vitro and in vivo analyses in both rodent and human experimental platforms, with both academic and industrial partners.

Lab website URL:
http://www.lyons-lab.com/

Recent/Relevant Publications:

Do you have an existing collaboration with a colleague at McGill?  If so, please give brief details.
No.
Name: Dr Tom MacGillivray

Email: t.j.macgillivray@ed.ac.uk

Picture (of yourself, if possible):

Brief Summary of Research Interests:
I have more than 15 years’ experience facilitating clinical research at the University of Edinburgh that features retinal imaging, computational analysis and machine learning. This includes studies and publications on stroke, cardiovascular disease, MS, diabetes, kidney disease, dementia and age-related cognitive change. In close collaboration with the University of Dundee (Prof E. Trucco, School of Computing), I co-ordinates an interdisciplinary initiative called VAMPIRE (Vascular Assessment and Measurement Platform for Images of the Retina; vampire.computing.dundee.ac.uk) in which our aim is efficient, semi-automatic analysis of retinal images and the pursuit of biomarker identification. Of particular interest is the use of the eye to provide a unique “window” to detect both microvascular and neurodegenerative changes in the brain.

Lab website URL:
https://www.ed.ac.uk/profile/dr-tom-macgillivray
https://www.edinburghcrf.ed.ac.uk/Content.aspx?uirefid=2532&dbid=crf_admin_live&areaid=855&name=Imaging+and+Image+Analysis&oid=Screen&type=StaticContent&theme=CrfPublic&

Recent/Relevant Publications:

Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
No
Name: Dr Barry McColl

Email: barry.mccoll@ed.ac.uk

Brief Summary of Research Interests (200 words max):
The overall goal of our research is to understand neuroimmune mechanisms influencing brain injury, repair and disease in order to identify new targets for treatments. We have particular interests in cerebrovascular disease (stroke and vascular cognitive disorders) and neurodegenerative diseases linked to dysfunction of microglia and neuroimmune regulation. Research in the lab largely encompasses three major strands each of which involves a range of molecular, cellular and organismal approaches using preclinical models, human samples and patient-based investigation: (i) Microglial mechanisms of resilience and susceptibility to neurodegenerative disease, (ii) Myeloid cells in brain injury and repair, (iii) Neuroimmune signalling and systemic immune dysfunction after stroke

Lab website URL:
https://www.ed.ac.uk/discovery-brain-sciences/our-staff/research-groups/dr-barry-mccoll

Recent/Relevant Publications:
Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
Yes, with Prof Sam David around role of microglia/macrophages in CNS injury and repair, including MTA to share reagents. See also recent PLoS Biol publication above. I have previously given seminar at their Neuroinflammation Center symposium.
Name: Veronique Miron  
Email: vmiron@ed.ac.uk

**Brief Summary of Research Interests:**
Central nervous system (CNS) white matter repair often fails in neurological disease, e.g. following developmental brain injury leading to cerebral palsy, and in various neurodegenerative disorders such as multiple sclerosis, contributing to axon dysfunction/loss and clinical impairment for which there is an unmet therapeutic need. My lab focuses on identifying novel strategies to overcome this failure of myelin repair by taking the innovative approach of investigating the regenerative properties of inflammation, in particular those of microglia. Previously I showed that successful adult white matter repair is driven by a transition in activation of microglia from a pro-inflammatory to a pro-regenerative phenotype [reference 2]. Since then, my lab’s primary objective has been to determine how we can harness the regenerative properties of microglia to support myelin repair across the lifespan. We are addressing this by investigating:

1) Which regenerative factors are released by microglia and how they exert their effect [reference 1,2],
2) How the successful transition in microglia activation takes place [reference 4],
3) How this transition is dysregulated in disease (e.g. perinatal brain injury [underway], MS [reference 4]),
4) How interaction with other cell types influences the regenerative potential of microglia e.g. astrocytes, monocyte-derived macrophages [underway].

**Lab website URL:**  
https://www.ed.ac.uk/centre-reproductive-health/dr-veronique-miron

**Recent/Relevant Publications:**


**Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.**
I have discussed a possible collaboration with Jack Antel at the Montreal Neurological Institute regarding investigating microglia function across the life span in health and disease. This would combine his unique access to human neurosurgical tissue across the lifespan with our access to various models of injury.
Name: Matthew Nolan

Email: mattnolan@ed.ac.uk

Brief Summary of Research Interests:
My lab investigates how neural circuits implement computations important for cognition and how these computations are altered in neuropsychiatric disorders. Our primary focus is on how neural circuits that tell us where we are integrate memories with sensory stimuli. We are also investigating how functions of these circuits are modified in rodent models of autism spectrum disorders. Approaches that we use include in vivo and ex-vivo electrophysiology and optogenetics, virtual reality-based behaviours, light sheet microscopy, molecular circuit analysis and computational modelling.

Lab website URL:
http://nolanlab.mvm.ed.ac.uk/

Recent/Relevant Publications:


Do you have an existing collaboration with a colleague at McGill? If so, please give brief details:
No
Name: Dr Cyril Pernet

Email: cyril.pernet@ed.ac.uk

Brief Summary of Research Interests:
I am mostly interested in methodological and (bio) statistical methods, including machine learning, for brain imaging (MRI, fMRI, PET, MEG, EEG) with clinical applications (e.g. adapting methods to work on patients, developing pipelines for automated data processing, etc).

Lab website URL:
http://www.sbiric.ed.ac.uk/cyril/

Recent/Relevant Publications:
1. Pernet et al. (2019) **BIDS-EEG: an extension to the Brain Imaging Data Structure Specification for electroencephalography.** In revision for *Sci Data.*

Do you have an existing collaboration with a colleague at McGill? If so, please give brief details
Informal collaboration with Prof Evans and Poline: bioinformatics for brain data imaging management and processing.
Name: Nathalie Rochefort

Email: n.rochefort@ed.ac.uk

Brief Summary of Research Interests:
The global aim of my research group is to understand how brain neuronal networks represent the outside world and how experience durably modifies the activity of such networks.

By using two-photon calcium imaging combined with electrophysiological recordings in awake behaving mice, our current projects investigate:
- how behavioral context modulates neuronal activity in the primary visual cortex
- which mechanisms underlie the action of neuromodulators in cortical networks
- how visual experience durably modifies the activity of cortical neuronal networks.

Using such information, we apply the same combination of methods to study how this network activity is disrupted in the brain of mouse models for autistic spectrum disorders and intellectual disabilities.

Lab website URL:
https://www.ed.ac.uk/discovery-brain-sciences/our-staff/research-groups/nathalie-rochefort

Recent/Relevant Publications:

Articles

Review

Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.

No
Name: Tara Spires-Jones

Email: tara.spires-jones@ed.ac.uk

Brief Summary of Research Interests:

My research focuses on the mechanisms and reversibility of neuronal degeneration in Alzheimer’s disease and ageing. We hypothesize that the degeneration of synapses causes memory impairments and that targeting the proteins that cause synaptic degeneration will allow recovery of cognitive function. Previous work from our group has shown that both of the proteins involved in the neuropathological lesions in Alzheimer’s (amyloid beta and tau) contribute to synapse loss in Alzheimer’s disease, and further that reducing the levels of soluble amyloid beta or tau prevents synaptic degeneration and improves memory in disease models. These experiments indicate that the plasticity of synapses will allow recovery after treatments, giving hope for some functional recovery in patients if we can develop therapies that remove the toxic protein species from the brain.

For our experiments, we apply high-resolution imaging techniques, including multiphoton imaging and array tomography, to examine the structure and function of synapses in healthy and diseased brain. Array tomography is a post-mortem imaging technique involving high-throughput imaging of thousands of synapses to determine whether disease-related proteins are associated with synapse shrinkage and loss. We have accumulated the world’s first brain bank of human tissue prepared for the array tomography technique, which we are using to investigate the relationship between synaptic changes and dementia.

Lab website URL:

Recent/Relevant Publications:


Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.

No
**Name:** Sergiy Sylantyev  

**Email:** s.sylantyev@ed.ac.uk

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**Brief Summary of Research Interests:**
In our work we monitor electrical currents through individual ion channels or groups of channels to increase our knowledge of channel function and the regulatory role of different pharmacological and biophysical agents. This furthers our understanding of the signalling mechanisms among molecular complexes inside the cell, between different cells and within neural networks.

There are three main areas which we work with:
1. Spontaneously opening GABA$_A$-receptors, their role in interneuronal crosstalk and intracellular factors that regulate their function; potential role of this receptor subtype in epilepsy therapy.
2. High-speed interaction between metabotropic and ionotropic glutamate receptors via scaffolding proteins; possible role of this type of crosstalk in development of Parkinson’s disease.
3. Tonically active glycine receptors and their role in the brain function; cellular and molecular mechanisms of hyperekplexia initiated by loss-of-function and gain-of-function mutations in glycine receptors.

**Lab website URL:**  
https://www.ed.ac.uk/profile/dr-sergiy-sylantyev

**Recent/Relevant Publications:**

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Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
I have no collaboration with McGill.
Brief Summary of Research Interests:
The cerebral cortex is responsible for all higher mental and cognitive functions unique to humans. Disruption of its function underlies a variety of different neurological disorders such as intellectual disability, autism and certain forms of epilepsy. To fulfil its role the cortex requires an enormous variety of different neurons, far more than in any other part of the brain. This striking degree of neuronal diversity is generated from stem cells. During corticogenesis, the activity of these stem cells, their proliferation and differentiation are tightly controlled and defective growth control can lead to macrocephaly or microcephaly.

The general aim of our research is to better understand the mechanisms which lead to the generation of these different types of cortical neurons and how cell signalling and transcriptional regulation converge to control cortical neuron formation. We specifically investigate the role of (i) the Gli3 zinc finger transcription factor and (ii) of the primary cilium, a signalling hub crucial for embryonic development and tissue homeostasis, in cortical stem cell development. To this end, we are using the mouse as a model system as well as human brain organoids derived from iPS cells that we engineered to carry mutations in the ciliary INPP5E gene.

Lab website URL:
https://www.ed.ac.uk/discovery-brain-sciences/our-staff/research-groups/thomas-theil

Recent/Relevant Publications:


Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
N/A
**Name:** Dr Carole Torsney  
**Email:** carole.torsney@ed.ac.uk

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**Brief Summary of Research Interests:**
Persistent pain arises following tissue damage or due to nervous system injury/disorder and is characterised by spontaneous pain, allodynia (touch-evoked pain) and hyperalgesia (exaggerated pain). Using electrophysiological and behavioural approaches in preclinical models I have identified neural circuits and plasticity relevant for allodynia (Torsney & MacDermott 2006), mechanical hyperalgesia (Torsney 2011) and thermal hyperalgesia (Dickie et al 2017). As part of the University of Edinburgh Translational Pain Research Programme I have collaborated with clinical colleagues to study chemotherapy-induced neuropathic pain. This work has established that antioxidants that are specifically targeted to mitochondria are able to limit the development of chemotherapy-induced neuropathic pain (McCormick et al. 2016; Galley et al. 2017). This includes melatonin, which has a low toxicity profile and is well tolerated in humans and so has strong potential for translation to patients.

The International Association for the Study of Pain has defined 2019 as the ‘Global Year Against Pain in the Most Vulnerable’ because pain in vulnerable populations including dementia, intellectual disability and autism/neurodevelopmental disorders has been underexplored despite increasing awareness of environmental and genetic influences upon pain. I am therefore interested in investigating the somatosensory phenotype of these vulnerable populations in preclinical models.

**Lab website URL:**
https://www.ed.ac.uk/discovery-brain-sciences/our-staff/research-groups/carole-torsney

**Recent/Relevant Publications:**

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**Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.**  
No
Brief Summary of Research Interests:
My primary interest is drug discovery, working across therapeutic areas with expertise spanning protein biochemistry, pharmacology, medicinal chemistry and technology development. I specialise in translational research with a focus on discovering and developing small molecules as medicines for the treatment of diverse conditions such as Alzheimer’s disease, multiple organ failure, fibrotic disease and Multiple Sclerosis. My research has led to the discovery of a number of potent small molecule modulators of target enzymes and receptors. Notably within the University context my team discovered and advanced a novel compound (UE2343, Xanamem™), through preclinical discovery and Phase 1 clinical development. UE2343 was recently licensed to a commercial company and is currently progressing through Phase 2 clinical trials in the UK, US and Australia in patients with Alzheimer’s disease. A second molecule (GSK3335065), discovered as part as a collaboration with GSK, is in Phase 1 clinical development for acute pancreatitis.
I am interested in developing collaborations on novel therapeutic targets and pathways involved in neurodegeneration. My group has experience in this area with an ongoing project focused on the development of inhibitors of a key target involved in remyelination. We have developed expertise in various in vitro assays and screening technologies to identify and triage emerging small molecule modulators.

Lab website URL:
http://edin.ac/2dqzzUB

Recent/Relevant Publications:


Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.

No
Name: Anna Williams

Email: anna.williams@ed.ac.uk

Brief Summary of Research Interests (200 words max):
The Williams group has two programmes of research linking two diseases of the brain white matter, multiple sclerosis and cerebral small vessel disease (SVD):

1) Mechanism of remyelination in Central Nervous System (CNS) repair.
   Building on our previous work, we investigate a) which signals in the oligodendrocyte precursor cell (OPC) microenvironment direct their activation, migration and maturation for repair, b) how OPCs themselves can be manipulated to ensure more efficient repair c) how oligodendroglia are heterogeneous and how this affects their biology MS (Nature, 2019, Nature Medicine 2018), and d) how they interact with other cells, including axons via synapses in the context of demyelination and remyelination, using a hierarchy of in vitro and in vivo models and human post mortem tissue.

2) Mechanism of cerebral small vessel disease (SVD).
   SVD is common, important as a cause of dementia and stroke, yet relatively understudied and its aetiology is unknown. We have discovered that endothelial cell dysfunction causes oligodendrocyte damage as the initial mechanism of the disease, published in Science Translational medicine (2018). We are now investigating upstream of this, to understand genetic changes which provoke dysfunction, and downstream to determine the effectors of this dysfunction and their subsequent effects on the surrounding brain.

Lab website URL:
http://www.crm.ed.ac.uk/research/group/remyelination-multiple-sclerosis

Recent/Relevant Publications:


Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.
No
Name: David J A Wyllie

Email: david.j.a.wyllie@ed.ac.uk

Brief Summary of Research Interests:
I am the Director of the Centre for Discovery Brain Sciences at the University of Edinburgh and a Principal Investigator in the Centre for Brain Development and Repair at the Institute for Stem Cell Biology and Regenerative Medicine in Bangalore, India. I have a long-standing research interest in physiology, pharmacology and function of ligand-gated ion channels, particularly those activated by the neurotransmitter, L-glutamate. Through electrophysiological studies, my lab seeks to understand the structure-function properties and physiological roles of the various subtypes of NMDA receptors. In related research we use pre-clinical models of single gene causes of neurodevelopmental disorders to study the properties of altered synaptic function and to assess the extent to which pharmacological intervention can ameliorate the changes that are observed in such models. In addition, our research extends to the electrophysiological and functional characterization of defined neuronal and glial populations derived from human pluripotent stem cells and specifically those from individuals suffering from neurodevelopmental and neurodegenerative diseases. Our overall aim is to develop an integrated approach to research that begins with the study of single protein molecules and synaptic function and extends, through collaboration with colleagues, to whole animal studies with an ultimate goal of the clinical study and treatment of disease.

Lab website URL:
https://www.ed.ac.uk/discovery-brain-sciences/our-staff/research-groups/david-wyllie

Recent/Relevant Publications:


**Do you have an existing collaboration with a colleague at McGill? If so, please give brief details.**

No