MRC Research Experience Placements Sumer 2026

Supervisors and Projects

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- **Ruth Williams -** Identifying cis-regulatory elements governing sensory placode specification, with implications in placode associated birth defects.

Evaluation of acceptability: intervention aimed at autistic children and others with social communication differences

Name of Supervisor: Alexandra Sturrock
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Description: Autism is a neuro-difference affecting ~1:10 individuals across the UK. Social communication, i.e., the use of language in a social context, is a core feature of autism diagnosis. It can also be a standalone condition (i.e., Social Pragmatic Communication Disorder).

Speech and Language Therapists are significant contributors to the support package offered to children in these groups. However, there are few interventions with a solid evidence base, and fewer still that are endorsed by the autistic community.

SCIP is an intervention package which already has an evidence base for children in these groups (6-11y): https://sites.manchester.ac.uk/scip/. The online training aimed at S&LTs is new. In this project we would:

1. Evaluate the acceptability of the online training resources with practicing S&LTs working with these clinical populations
2. Collaborate with neurodivergent S&LTs to understand how to make the training more affirming

As the intern, you would recruit ten S&LTs and host 1-2 focus groups on the SCIP training programme. Also, recruit and host ~6 interviews with neurodivergent S&LTs. You would collect and analyse data thematically, ready for publication. You would contribute updating this important new resource for open access to clinicians and may contribute to writing a publication on this subject.

The supervisor will support the intern to develop skills in all areas. An interest in supporting children with social communication difficulties is essential. A knowledge of working with qualitative data collection/analysis and online platforms would be beneficial.

Understanding cognitive and neural correlates of paranoia and unusual beliefs in schizotypy.

Name of Supervisor: James Gilleen
Email: James.gilleen@manchester.ac.uk

Description: This project explores how people with high schizotypy, or those at risk of psychosis or in its early stages, form and update their beliefs about the world. Psychosis often involves auditory verbal hallucinations (AVH i.e. hearing voices), paranoia (sense of threat), and delusions (strong beliefs despite contrary evidence). The study uses a set of computer-based tasks and questionnaires to examine how people interpret perceptions, respond to social situations containing threat, and revise beliefs when given new information – and how this is modulated by stress. Data will come from healthy controls with low/high schizotypy, with the potential for data from clinical groups assessed in NHS services – the latter will be to contribute to data collection of a PhD student. The objectives of the placement student will be to (i) collect study data at one of the time points being assessed (T1 or T2), (ii) work with the PI and PhD student to analyse data and test specific hypotheses to understand the role of stress on mechanisms of paranoia or AVH. There is potential for a student to be involved in a neuroimaging project examining the role of glutamate in belief management and paranoid / unusual thinking using MRS.

Airway epithelial cell response to infection in idiopathic pulmonary fibrosis.

Name of Supervisor: Jenny Herbert
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Description: Idiopathic pulmonary fibrosis (IPF) is a severe progressive chronic respiratory condition. The disease is characterised by progressive damage to the airways, with aberrant tissue repair. It is unclear the exact causes of IPF, although genetic factors seem to contribute 1,2. Other risk factors such as smoking, have been shown to contribute to disease inception 3.Exacerbations in IPF are significant events, with diffuse alveolar damage occurring in the airways, with one study stating a 50% survival rate for patients 3-4 months post exacerbation 4. The role of infections in exacerbations still remains unclear, however a recent study found 34% of acute exacerbation in IPF were due to respiratory infections 5. In other respiratory conditions exacerbations (COPD, asthma, CF) are commonly due to infectious causes 6,7. The airway is the key site of IPF disease pathology therefore respiratory viruses and bacteria are likely one of the main culprits of airway damage and inflammation, as seen in other chronic respiratory conditions. In a 2017 study, 22.5% of IPF patients were positive for respiratory viruses 8. Another study found during exacerbations, patients had viral (17.9%) or bacterial (10.4%) respiratory mono infections. Within this study they also found 59.7% of patients had co-infections, and patients with co-infections had significantly reduced FVC and increased mortality rates 9. Understanding the role of infections, IPF disease exacerbations is still in its infancy. Therefore, the aims of this project are to:

1. Characterise differing inflammatory profiles of airway epithelial cells (AECs) (healthy/ IPF donors) in response to respiratory bacteria.
2. Evaluate the cellular damage to AECs (healthy/ IPF donors) in response to respiratory bacteria.
3. Quantify respiratory bacterial adherence to AECs (healthy/ IPF donors) This study will use easily accessible nasal airway epithelial cells as a surrogate for hard to access lower AECs.

Investigating mechanisms of brain metastasis in small cell lung cancer.

Name of Supervisor: Kathryn Simpson
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Description: Small cell lung cancer (SCLC) is a highly aggressive, neuroendocrine tumour. Most patients present with extensive stage disease which results in 5-year survival rates of <7%. SCLC is among the most brain-metastatic of all cancer types. Brain metastasis is present in 20% of patients at diagnosis, increasing to 80% during disease progression. Our understanding of SCLC metastasis remains limited, due to a lack of patient material and preclinical models, and biomarkers and therapeutic options are urgently needed. Our lab has developed a biobank of Circulating Tumour Cell (CTC)-derived explant (CDX) models which reflects the heterogeneous molecular landscape of SCLC. Multiple CDX models tractably metastasise following resection of the subcutaneous (S.C) tumour to frequent metastatic sites observed in SCLC patients, including brain, providing an excellent tool to investigate the mechanisms driving SCLC metastasis.

The student will work with a postdoc who is working to identify the biological mechanisms which drive small cell lung cancer metastasis to the brain. They will be exposed to our studies, working with CDX models to identify the biological changes which occur in SCLC cells which facilitate their growth and survival in the brain.

The student will learn how to culture cancer cells and dissociate tumours to isolate single cells for further downstream analysis. The student will be exposed to a range of molecular biology techniques including flow cytometry of fluorescently labelled cancer cells and immunohistochemical analysis of cancer cells in mouse tissues to probe for specific proteins of interest in both subcutaneous tumours and at sites of metastases.

Aims

1. Become competent in tissue culture techniques of CDX cells.
2. Learn how to identify proteins of interest in tissues using immunohistochemistry and perform analysis of study samples using HALO.

Dissociate CDX tumours and perform immunofluorescence staining to confirm presence of human mitochondria in isolated cells.

Investigating genetic origins of plasmid impact on bacterial fitness

Name of Supervisor: Mato Lagator
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Description: Horizontal acquisition of plasmids by bacterial hosts is a major source of both, virulence and antibiotic resistance. Plasmids carry myriad genes, which they introduce into their host to provide novel functions. While these functions can be beneficial in a given environment (for example, in the presence of an antibiotic), a key factor that determines whether a plasmid will be maintained in the long-term is how it affects host fitness *across many* environments. The impact of a plasmid on host fitness in each environment, in turn, depends on the interactions between host and plasmid genes.

To interrogate this relationship, we have inserted a clinical antibiotic resistance plasmid pLL35 into >3000 single gene *E. coli* knockout strains. We measured the fitness of each gene knockout (as growth rate over 24h) with and without the plasmid, which allowed us to capture whether and how each bacterial gene interacts with the plasmid to affect fitness. However, these measurements were performed in only one growth condition (Muller Hinton rich media). This limitation means that, currently, we characterized host genome-plasmid interactions in only a single environment, limiting our ability to capture how genetic interactions shape long-term plasmid maintenance.

The aim of this internship is to measure the fitness of *E. coli* knockout strains with and without the pLL35 plasmid in three additional conditions: minimal media; enriched minimal media; and media mimicking growth conditions in a human body.

To achieve this aim, the student will utilize state-of-the-art robotics to facilitate high throughput growth measurements of 1000s of bacterial strains simultaneously. The student will be trained in all the required techniques and will work under S2 microbiology conditions. The student will also learn how to use R and Python to run existing scripts to analyze the data, produce figures and conduct statistical analyses.

Identifying mechanisms that drive pathogen virulence.

Name of Supervisor: Fiona Whelan/Melissa Lawson
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Description: For people with cystic fibrosis (CF), illness and early mortality are often driven by chronic bacterial infections. A major challenge is that disease severity varies widely between patients. For several CF-associated pathogens, virulence is influenced by their environment, including the presence of other microbes—an area the Whelan group is actively investigating. Work in *Drosophila* models has shown that specific microbe–pathogen associations can either enhance or reduce the virulence of CF-related pathogens, although the mechanisms behind this remain unclear.

This project uses our established *Drosophila* chronic infection model to dissect these interactions, focusing on pathogens (e.g. *Pseudomonas aeruginosa* or *Stenotrophomonas maltophilia*) and other microbes, including *Staphylococcus* sp. or *Streptococcus sp.* to address the following questions: (1) Does microbial abundance change over time, and are these changes linked to killing and/or the type of microbe-pathogen association (e.g. for associations that perpetuate pathogen virulence do you see greater pathogen abundance in these flies?). (2) According to published literature, 'priming' pathogens by growing them in different media prior to infection can influence virulence. Therefore, we would like to determine if 'priming' pathogens prior to infection means they kill flies faster. (3) Finally, to complement these objectives we also want to investigate how the pathogen changes (before and) after infection/fly death? To do this we will use robotics to make a high-throughput workflow and examine changes in pathogen growth rate and/or protease activity before and after fly colonisation.

At the end of the project, the student will have identified key insights into the mechanism of how microbes regulate pathogen virulence. In addition, the student will gain valuable experience in a variety of fields including microbiology, the use of model organisms, imaging, high-throughput robotics, and analysis of experimental data using R.

Multimodal Large Language Model for Early Detection of Appendix Cancer

Name of Supervisor: Mobarak Hoque
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Description: This project aims to develop a multimodal LLM prototype for early detection of appendix cancer by integrating CT imaging, pathology, and clinical information. The objectives are

1. Building on our prior multimodal LLM research, develop a lightweight pipeline that encodes CT scans, pathology slides, and clinical variables, and fuses them within an LLM framework for early disease identification.
2. Investigate whether this form of cross-modal fusion can produce clearer early-detection signals, more informative reasoning, and improved performance, using both public benchmarks and anonymised in-house data from the Christie NHSFT.

Project Description

Appendix cancer is often detected late because early imaging and pathology features are subtle and easy to miss. This six-week project explores whether a multimodal LLM can integrate CT, pathology, and clinical inputs to support earlier detection.

Building on our previous multimodal LLM work (Huang et al. 2025; He et al. 2024; Bian et al. 2023; Martínez-Quintanilla et al. 2024), the student will assemble a lightweight workflow using existing encoders for CT scans and pathology slides, combined with structured clinical data. The model will be trained and tested on a mix of public benchmark datasets and anonymised in-house data provided by Prof Aziz (in supervisory team), a surgeon at the Christie NHSFT, which conducts advanced translational research in peritoneal tumours.

These multimodal features will be merged through an LLM-based fusion module that learns shared representations linked to early disease indicators. A small explanation component will allow the model to highlight key imaging or pathology cues behind each prediction.

The work focuses on a practical proof-of-concept rather than a full diagnostic system. Evaluation will assess feasibility, early detection performance, and basic model behaviour. The project will conclude with a functioning prototype and a short report outlining the approach, results, and recommended next steps.

Real-time pose capture in freely moving mice for closed loop experiments

Project Supervisor: Riccardo Storchi
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Description: To understand mechanisms of neural disorders it is important to determine how they affect daily activities. In our group we have pioneered high-throughput 3D tracking in mice that are free to move and can express their natural behaviours (Storchi, Curr Bio 2020; Orlowska-Feuer, Curr Bio 2022; Ebrahimi, Sci Rep 2023). We have been using these techniques to study mechanisms of ocular disease (Storchi, Sci Rep 2019) and the effects of ablation of specific retina cell types (Mouland, Nat Comm, 2025).

We are now combining simultaneous neural recording and 3D tracking to understand neural responses to postural challenges in visual brain regions (Ebrahimi, biorxiv, 2025). In these experiments mice actively explore an arena; at irregular intervals, the arena is tilted, introducing postural challenges. To better capture specific behavioural responses, we want to induce the tilt when the animal is in a specific pose (e.g. vertical posture vs four-limb support; with body oriented parallel vs orthogonal to the tilt axis). To achieve this, we need to perform closed-loop experiments in which animals' 3D poses are reconstructed in real-time and used to trigger the tilt events. However, so far, pose 3D reconstruction has only been performed offline after the experimental sessions.

The student will generate a pipeline to combine a fast/light head and body tracker with our in-house code for 3D reconstruction. They will be provided with source code for 3D reconstruction, support during the implementation phase and a GPU-powered PC. Upon successfully passing tests on previously acquired datasets, the pipeline will be validated on a small set of pilot experiments.

We are looking for a student with a passion for neuroscience, machine learning and practical programming skills. Applicants would preferably have experience with computer vision and real time signal processing, be prepared to work with hardware-software integration and be available in person throughout the project duration.

Identifying cis-regulatory elements governing sensory placode specification, with implications in placode associated birth defects.

Name of Supervisor: Ruth Williams
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Description: The neural plate border (NPB) generates the neural crest and sensory placodes. Neural crest cells contribute to a wide range of derivatives in the vertebrate body, including elements of the peripheral nervous system, pigment cells and the craniofacial skeleton. While placode cells form cranial sensory structures (nose, ears, lens) and cranial ganglia. With such plasticity, NPB cells are an attractive model for therapeutic purposes. Aberrant development of the neural crest and sensory placodes is associated with one-third of all congenital birth defects. The over-arching aim of my lab is to understand the precise molecular mechanisms controlling cell fate decisions from the NPB. We use the chicken embryo as our model since their early development closely mirrors human development, plus many of the genes and regulatory elements are well conserved. Furthermore, chicken embryos are readily accessible without needing to dissect the mother and they are easily manipulated ex ovo and in ovo for CRISPR perturbation and enhancer reporter experiments.

Specifically, this project aims to identify novel cis-regulatory interactions underlying the activity of genes known to be involved placode specification. This will involve exploring existing epigenomic data sets, in particular; ATAC-seq generated from NPB cells isolated from developing chick embryos and single-cell Multiome data generated from early human cranial tissue, for putative cis-regulatory elements (enhancers) driving key placode genes. Putative enhancers we then be cloned into a fluorescent reporter plasmid and tested for enhancer activity in vivo using the chick embryo. Fluorescent in situ hybridisation will also be used to detect expression of candidate genes in developing chick embryos. The student will therefore learn some bioinformatic skills as well as molecular biology, embryology and fluorescent confocal imaging techniques.
Identifying enhancers driving placode specification will further resolve the gene regulatory network governing this crucial developmental process. Furthermore, we will determine the conservation of chick placode enhancers with human, which will allow us to infer putative enhancer activity in human and thus provide novel loci at which to search GWAS data sets for causative SNPs underlying birth defects and determine enhancer pleiotropy in during placode development and disease.