**Available Projects for Intercalated BSc (iBSc) in Medical Science**

**2024-2025**

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**Projects Hosted by the Wellcome-Wolfson Institute For Experimental Medicine**

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| **Project Title** | **Exploring mechanism of bacterial resilience against last-resort antibiotics** |
| **Supervisor(s)** | Prof. Miguel A. Valvano, M.D. (Paediatrics & Infectious Diseases); Chair in Microbiology and Infectious Diseases |
| **School / Centre** | Medicine, Dentistry and Biomedical Science, Wellcome-Wolfson Institute for Experimental Medicine |
| **Principal Supervisor’s Contact Details** | Email: m.valvano@qub.ac.uk | Tel: 028 9097 6025 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry | ✓✓ |
| Microbiology | ✓✓ |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | (✓) | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) |  |
| **Background information:** | Antibiotics are the greatest success story of modern medicine, but the steady global increase of infections caused by multidrug antibiotic resistant (AMR) bacteria has turned into one of the greatest threats to human health. We investigate *Enterobacter* species, which are identified by the WHO among the most dangerous bacteria. AMR *Enterobacter* clinical isolates can also become resistant to colistin, a last-resort antibiotic, either by horizontal transfer of modifying genes or by the expression of heteroresistance in the bacterial population. Uncovering the molecular basis of intrinsic resistance in *Enterobacter* is paramount to devise control measures. |
| **Aims / objectives** | The aim of this project is to investigate the mechanisms of intrinsic resistance in AMR *Enterobacter cloacae* complex isolates by: (1) testing the virulence potential of the *E. cloacae* complex isolates in the *Galleria mellonella* moth larvae infection model; (2) conducting qRT-PCR assays and global transcriptomics comparing the expression of selective colistin. resistance associated genes and potential regulators in pre- and pots-infection isolates; (3) comparing the lipid a profile of colistin-sensitive and -resistant isolates.; and (4) investigating the role of an *Enterobacter* type VI secretion system in the bacterial competition against gut colonizing bacteria to explain how AMR *Enterobacter* can colonize patients providing a reservoir to transmit superbugs.  |
| **Techniques employed:** | The student will learn general microbiology and molecular biology techniques, PCR amplification, infection model in *Galleria mellonella*, and rudiments of mass spectrometry. General techniques of biochemistry and molecular biology will be applied, as well as rigorous hypothesis-driven thinking process to learn scientific method. ***The student will join a highly accomplished and motivated research team environment and s/he is expected to learn and apply experimental design and execute the experiments with technical proficiency after initial training.*** Attendance and presentation to weekly lab meetings will complement training, as well as individual meetings with Prof. Valvano to assess progress. To learn more about the Valvano lab ethos please visit: <https://publish.uwo.ca/~mvalvano/> |

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| **Project Title** | **Probing the signal transduction mechanisms involved in neuroinflammation induced by astrocytes in response to substance P**  |
| **Supervisor(s)** | Dr Bianca Plouffe (PI) and Dr Carole Daly (PDRA) |
| **School / Centre** | SMBMS / WWIEM |
| **Principal Supervisor’s Contact Details** | Email: b.plouffe@qub.ac.uk | Tel: 07865422928 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry |  |
| Microbiology |  |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation AwardThe Medical Undergraduate Intercalated Scholarship | ✓✓ | *Subject-specific awards*Sir Colin Dollery Clinical Pharmacology Award | ✓ |
| **Background information:** | Infection to pathogens can cause neuroinflammation, a condition associated to excessive production of cytokines promoting neuronal death. Astrocytes, a type of glial cells in the central nervous system, play a major role in neuroinflammation. These cells express neurokinin-1 receptor (NK1R) and stimulation of NK1R by the neuropeptide substance P induces cytokine secretion. Exposition of astrocytes to bacterial pathogens dramatically increases NK1R expression in astrocytes and consequently exacerbates the cytokine production by substance P. Although the role of NK1R in cytokine secretion by astrocytes is well established, the molecular mechanisms translating NK1R activation into this cellular response remains obscure as it mainly occurs at endosomes rather than at the plasma membrane. |
| **Aims / objectives** | The objective is to understand how endosomal NK1R activates nuclear ERK1/2 and NFκB, two major drivers of transcription involved in cytokine production, by investigating: 1. The production of the canonical second messenger diacylglycerol at the membrane of endosomes following stimulation with substance P.
2. The role of phosphatidylinositol-3-phosphate (PI3P), the most abundant phosphoinositide at the membrane of endosomes, as a potential substrate for phospholipase Cβ.
 |
| **Techniques employed:** | The student will use a wide panel of state-of-the art biosensors with high subcellular resolution based on Förster resonance energy transfer (**FRET**), bioluminescence resonance energy transfer (**BRET**) and nano-luciferase Binary Technology (**nanoBiT**) to track in real time the signalling events following NK1R activation in astrocytoma. **Confocal microscopy** will also be used to visualise these events.  |

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| **Project Title** | **The role of Alveolar Type 2 cell derived HE4 in pulmonary fibrosis** |
| **Supervisor(s)** | 1. Dr BC Schock
2. Dr K Dib
 |
| **School / Centre** | SMDBS, WWIEM |
| **Principal Supervisor’s Contact Details** | Email: b.schock@qub.ac.uk | Tel:02890972258 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry | ✓ |
| Microbiology |  |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) | ✓ |
| **Background information:** | Pulmonary fibrosis (PF) such as Idiopathic Pulmonary Fibrosis (IPF) and Systemic-Sclerosis-associated-Interstitial-Lung-Disease (SSc-ILD) is a life-limiting lung disease and increasing rates are observed particularly in Northern Ireland. There are limited therapies available and new treatments are urgently needed. Hypoxia is common in PF determining the disease specific microenvironment that activates fibroblasts. We identified **Human Epididymis protein 4 (HE4)** as a hypoxia-induced pro-fibrotic mediator proposing **HE4 as a therapeutic target in PF.** Exposure of bronchial epithelial cells significantly increases HE4 protein induction (intracellular) and secretion (into the microenvironment/cell culture medium), but the effect of hypoxia on HE4 in alveolar type 2 (AT2) cells has not been explored. Further, the anti-diabetic Dapagliflozin reduces hypoxia induced HE4 in epithelial cells, and fibroblasts exposed to such medium show reduced fibrosis and inflammation.Here we will extend our investigation to AT2 cells and investigate the effect of the SGLT2 inhibitor dapagliflozin on HE4 induced fibrotic and inflammatory responses. |
| **Aims / objectives** | We will determine the hypoxia induced HE4 secretion from AT2 cells using an AT2 cell line. We will further investigate if treatment with dapagliflozin is effective in preventing hypoxia induced HE4 secretion from AT2 cells and if the this leads to a reduction of the fibrotic and inflammatory responses in pulmonary fibroblasts. |
| **Techniques employed:** | 1. Cell culture of an AT2-like cells line (A549) and pulmonary fibroblasts (CCD11-Lu).
2. Characterization of the hypoxia induced response in AT2 cells (HE4 and inflammatory cytokines) using ELISA techniques.
3. Characterization of the expression and function of HE4 on differentiated fibroblasts by analyses of specific myofibroblast markers at transcriptome (qRT-PCR) and protein level (Western blot, ELISA).

The student will be able to present data regularly at laboratory meetings and at a national respiratory conference (e.g., BALR) |

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| **Project Title** | **Developing a vaccine to protect sexual health**  |
| **Supervisor(s)** | 1. Rebecca Ingram 2. Rachael Bell  |
| **School / Centre** | Wellcome-Wolfson Institute of Experimental Medicine  |
| **Principal Supervisor’s Contact Details** | Email: b.ingram@qub.ac.uk | Tel: 02890 97 6389 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry | ✓ |
| Microbiology | ✓ |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓✓ | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) | ✓ |
| **Background information:** | Sexually transmitted bacterial infections are increasing and unfortunately, the levels of antibiotic resistance seen in these bacteria are increasing. Two of the most common infections are Chlamydia and Gonorrhoea. *Chlamydia. trachomatis* is the most common sexually transmitted bacterial pathogen in the world with a global prevalence estimated at 4.2%. Worldwide approximately 130 million new infections occur annually. In 2020, WHO estimated 82.4 million new infections with *N. gonorrhoeae* among adults aged 15 to 49 years. These infections can cause serious complications such as pelvic inflammatory disease, ectopic pregnancy and infertility. Both of these infections increase the risk of HIV infection. WHO has recognized sexually transmitted infections as a significant public health problem and has set ambitious targets to reduce the global incidence of infection by 90% by 2030. A key part of meeting this challenge is the development of a vaccine to prevent infection. The Ingram lab has developed a reverse vaccinology approach for identifying novel anti-bacterial antigens. This approach has successful identified antigens that protective against bacterial infection; we are currently in the process of patenting a vaccine to prevent *Pseudomonas*. In this project the student will clone and express potential vaccine candidates that have been identified using in silico analysis of the genomes of *Chlamydia trachomatis* and *Neisseria gonorrhoeae* |
| **Aims / objectives** | 1. To design primers and PCR the genes encoding the top 10 vaccine candidates from
2. To clone the genes into a vector and transform *E. coli*
3. To induce expression of the recombinant protein and verify the expression using western blot
 |
| **Techniques employed:** | PCR, cloning, expression, western blot |

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| **Project Title** | **DNA methylation as a key determinant of endothelial progenitor cell dysfunction associated with cardiovascular disease** |
| **Supervisor(s)** | 1. Professor David Grieve2. Dr. Karla O’Neill |
| **School / Centre** | SMDBS / Wellcome-Wolfson Institute for Experimental Medicine |
| **Principal Supervisor’s Contact Details** | Email: d.grieve@qub.ac.uk | Tel: 02890976468 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry |  |
| Microbiology |  |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) |  |
| **Background information:** | Impaired angiogenesis is known to influence the progression of ischaemic cardiovascular disease (CVD), with stresses such as hyperglycaemia and hypoxia (ischaemia) promoting endothelial cell (EC) dysfunction. Recent attention has focused on the therapeutic potential of endothelial progenitor cells (EPCs), which are mobilised by ischaemia and are important in vascular homeostasis. Our group has characterised a distinct EPC subtype, termed endothelial colony-forming cells (ECFCs), with well-defined endothelial progenitor properties, which promote new blood vessel formation in both health and disease. Whilst DNA methylation (critical for appropriate gene expression) regulates mature EC homeostasis and stress-induced dysfunction, its role in determining the angiogenic response of ECFCs is not defined. This is critical given their capacity for vascular repair, reported dysfunction in CVD, and therapeutic potential. Importantly, we have recently demonstrated that important stress-induced DNA methylation alterations correlate with reduced angiogenic capacity and therefore hypothesise that this key epigenetic modification is central in driving ECFC dysfunction in CVD and could be targeted to promote therapeutic angiogenesis. |
| **Aims / objectives** | This project therefore aims to investigate the specific role of DNA methylation and associated modifying enzymes (DNMT1 etc.) on *in vitro* ECFC angiogenic function. It is hoped that the results will identify both key adverse DNA methylation changes and alterations in methylome-linked signalling pathways which 1) may become dysregulated in response to cardiovascular stresses (hyperglycaemia and hypoxia) and 2) could represent potential targets to enhance the reparative capacity of ECFCs given their clear potential for the treatment of ischaemic cardiovascular disease. |
| **Techniques employed:** | In order to characterise the effects of DNA methylation and modifying enzymes on ECFC function, studies will be undertaken in cultured cells (healthy and diseased) post-genetic modification (using siRNA, plasmids) to produce attenuated/augmented levels of proteins critical to maintenance of the methylome (DNMT1, UHRF1 etc.). An inhibitor of DNA methylation (5’AZA; clinically approved drug for treatment of specific disease) will also be utilised in order to examine the role of this gene-regulating modification in the ECFC angiogenic response. Expression of key signalling genes will be quantified by real-time RT-PCR and/or western blot and *in vitro* ECFC tube formation (Matrigel) assays will be performed to assess functional effects.The student can realistically expect to make an important contribution to ongoing ECFC research which will be acknowledged through manuscript co-authorship. |

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| **Project Title** | **Exploring the role of bone related proteins in vascular calcification** |
| **Supervisor(s)** | 1. Prof David Grieve
2. Dr Chris Watson
3. Dr Paul Hamilton
 |
| **School / Centre** | 1. Wellcome-Wolfson Institute for Experimental Medicine (DG, CW)
2. Centre for Medical Education (PH)
 |
| **Principal Supervisor’s Contact Details** | Email: d.grieve@qub.ac.uk | Tel: 02890976468 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry |  |
| Microbiology |  |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) | ✓ |
| **Background information:** | Calcification of the coronary arteries is a poorly understood pathological process. People affected with it will often have no symptoms but are known to be at high risk of a future myocardial infarction, particularly those with elevated circulating levels of low-density lipoprotein (LDL) cholesterol which is often linked with coronary artery calcification.The widespread adoption of computed tomography (CT) scanning of the coronary arteries now allows for non-invasive assessment of coronary artery calcification in at-risk patients. Once identified, individuals are targeted for aggressive risk factor modification, but there are currently no effective therapeutic options for reversing calcification. |
| **Aims / objectives** | By studying coronary microvascular endothelial cells in a laboratory environment, this project will explore mechanisms underpinning vascular calcification. This will enhance our understanding of how blood vessel disease develops and open up the possibility of better disease prediction and more focused treatment options in the future. |
| **Techniques employed:** | In order to characterise mechanisms underlying vascular calcification, studies will be undertaken in cultured human coronary microvascular endothelial cells treated with oxidised LDL to mimic early clinical disease. Expression of key signalling genes and proteins will be quantified by real-time RT-PCR, western blot and/or ELISA in parallel with assessment of in vitro function. The student can realistically expect to make an important contribution to ongoing translational research which will be acknowledged as appropriate through manuscript co-authorship. |

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| **Project Title** | **To investigate the role of histamine in neutrophil phagocytosis** |
| **Supervisor(s)** | 1. Dr K Dib2. Dr B. Schock |
| **School / Centre** | Welcome-Wolfson Institute for Experimental Medicine, School of Biomedical sciences, Dentistry and Medicine |
| **Principal Supervisor’s Contact Details** | Email:k.dib@qub.ac.uk | Tel:02890694244 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry | ✓ |
| Microbiology | ✓ |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*Sir Colin Dollery Clinical Pharmacology AwardOther (Please specify): British Society for Immunology | ✓✓ |
| **Background information:** | Neutrophils constitute the first line of defence against a large number of microorganisms. These leukocytes have developed different means to kill blood borne or tissue-resident microorganisms. One of this mechanism is phagocytosis. It is a complex process starting with the capture of opsonised microorganisms (microorganisms coated with complement or antibodies) by phagocytic receptors, followed by the internalisation of the microorganisms inside phagosomes where they are killed. Pro-inflammatory cytokines, produced in response to infection, stimulate neutrophil phagocytosis. During periods of infection histamine is produced but its role in the inflammatory response is not well known. Recently, we showed that histamine is a potent regulator of neutrophil phagocytosis. We found that both the histamine two receptor (H2R) and the histamine four receptor (H4R) in neutrophils have opposite roles in terms of phagocytosis. The H4R controlled intracellular killing of *E. coli* whereas the H2R negatively controlled the capture of *E. coli*. We would like now to investigate if histamine also regulates the capture and/or killing of *P. aeruginosa*, a bacteria found in the airways of patients with cystic fibrosis (CF) causing lung tissue damage. The rationale behind this study is the fact that bacteria colonising the CF airways produce histamine and thereby may block neutrophil phagocytosis.  |
| **Aims / objectives** | **The main objective** of this proposal is to investigate whether neutrophil histamine receptors can be targeted pharmacologically to boost *P. aeruginosa* eradication by neutrophils.**Aim 1**: To investigate the role of histamine in *P. aeruginosa* phagocytosis. We will test if histamine controls *P. aeruginosa* capture and/or killing by neutrophils. We will optimise *P. aeruginosa* phagocytosis by neutrophils including incubation time, neutrophil to bacteria ratio, and opsonisation.**Aim 2**: To investigate whether H2R antagonists or H4R agonists affect histamine-dependent neutrophil phagocytosis.Once the *P. aeruginosa* phagocytosis assay has been optimised (see aim 1), we will test if pre-incubation of neutrophils with the H2R antagonist famotidine or the H4R agonist VUF affects histamine-induced *P. aeruginosa* capture and/or killing by neutrophils. We will conclude on whether or not histamine receptor antagonist/agonist can be used to boost neutrophil phagocytosis in vitro. If this is the case, we can envision using them *in vivo* to treat patients who have their lungs colonized by P. aeruginosa or other Gram-negative bacteria. |
| **Techniques employed:** | Isolation of neutrophils from human blood. Blood will be collected from healthy volunteers and neutrophils will be isolated by using the Dextran/Ficoll method. Preparation of bacteria for phagocytosis. *P. aeruginosa* will be grown in LB medium after which they will be opsonised with human serum.Neutrophil phagocytosis. Neutrophils will be incubated with serum-opsonised bacteria for different time periods in the absence or presence of histamine. Thereafter, the amount of viable bacteria left will be measured by colony counting on LB-agar plates. |

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| **Project Title** | **Macrolide antibiotic resistance in *Mycoplasma pneumoniae* in Northern Ireland.** |
| **Supervisor(s)** | 1. Dr. Helen Groves (QUB / RBHSC)
2. Dr. Derek Fairley (BSHCT Regional Virus Lab / QUB)
3. Dr. Kathy Li (BSHCT Regional Virus Lab)
 |
| **School / Centre** | Wellcome Wolfson Institute for Experimental Medicine |
| **Principal Supervisor’s Contact Details** | Email: h.groves@qub.ac.uk | Tel: +44 (0)28 9097 2601 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry | ✓ |
| Microbiology | ✓ |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) |  |
| **Background information:** | *Mycoplasma pneumoniae* is a human host-restricted bacterial pathogen that causes community-acquired respiratory tract infection and atypical pneumonia, especially in children and adolescents. Fulminant *M. pneumoniae* infections can also occur, with significant mortality, so timely and appropriate antibiotic treatment is essential. Diagnosis of *M. pneumoniae* infection can be challenging because routine clinical bacteriology methods fail to isolate mycoplasma, so detection of this pathogen relies on PCR testing. This also means that conventional culture-based phenotypic drug susceptibility testing is not possible. First-line treatment of *M. pneumoniae* infection is empirical and relies heavily on macrolide antibiotics (clarithromycin or azithromycin) that target protein synthesis by binding to the peptidyl-transfer loop of the bacterial 23S rRNA. Single point mutations in the 23S rRNA gene can confer high-level macrolide resistance, and many countries including the UK are seeing rapidly rising rates of macrolide resistant *M. pneumoniae*. In parts of Europe and the US, levels of reported resistance are up to ~30%, and in some Asian countries, resistance rates of 90-100% are reported. No data are available on the prevalence of macrolide resistance mutations in Northern Ireland, so this project aims to address this. |
| **Aims / objectives** | 1. To identify 23S rRNA mutations that confer macrolide resistance in circulating *M. pneumoniae* strains in Northern Ireland.
2. To develop and validate a panel of molecular assays to rapidly identify common resistance mutations in clinical samples.
3. To estimate the prevalence of macrolide resistance in *M. pneumoniae* infections in Northern Ireland.
 |
| **Techniques employed:** | The project will work with residual diagnostic respiratory specimens from the Regional Virus Laboratory that have tested positive for *M. pneumoniae* by PCR.The student will use end-point PCR and DNA sequencing methods to obtain 23S rDNA sequences, and bioinformatics methods to investigate the presence of mutations that are known or expected to confer resistance to macrolide antibiotics.The student will then develop and validate a small panel of molecular assays using real-time PCR and/or LAMP to rapidly detect key mutations.Finally, the student will use these tests to evaluate the prevalence of macrolide resistance in circulating *M. pneumoniae* in Northern Ireland. |

**Projects Hosted by the Centre for Public Health**

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| **Project Title** | **Why do kidney transplants fail so early in young people?** |
| **Supervisor(s)** | 1. Dr Gareth McKay1 (Reader) 2. Dr Michael Corr1,2 (Clinical Fellow- Nephrology Registrar)  |
| **School / Centre** | 1. Centre for Public Health
2. Regional Nephrology and Transplant Centre, Belfast City Hospital
 |
| **Principal Supervisor’s Contact Details** | Email: g.j.mckay@qub.ac.uk | Tel: |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry | ✓ |
| Microbiology | ✓ |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) |  |
| **Background information:** | Kidney transplantation is the best form of treatment for young people with end stage renal disease (ESRD). Not only does it have a transformative impact on their quality of life, it dramatically reduces morbidity and mortality. In the last 20 years, over 300 young people have received a renal transplant in Northern Ireland (NI). Unfortunately, young recipients often lose their transplant much earlier than expected. Why transplant failure is more common in young people remains unclear.This study will characterise transplant loss in young renal transplant recipients in NI and assess epidemiological associations with long-term outcomes. Understanding why young recipients lose their transplant at higher rates will inform health services and interventions to prevent early loss of kidney transplants. |
| **Aims / objectives** | 1.) Compare the incidence of transplant graft loss in Adolescent / Young Adult (A/YA) to the wider transplant population in NI.2.) Identify the disease aetiology of NI A/YA recipients returned to ESRD i.e. acute kidney injury, immunological injury, disease recurrence3.) Investigate key demographic details with long-term transplant outcomes in this population (such as, age of transplant, age of transition from paediatric to adult care, socioeconomic group etc.)  |
| **Techniques employed:** | The study population will be NI renal transplant recipients transplanted before the age of 30 from 2001-2021 inclusive. Data will be extracted from a prospective database of renal transplant recipients which is maintained by clinical staff in the Belfast Health and Social Care Trust (IRAS ID 239344, REC 18/NI/0004). This database contains demographic information such as age of transplantation, HLA matching and details of both donors and recipients. Additional data including subsequent outcomes (graft failure, mortality) will be ascertained from the electronic care record. Simple and descriptive bioinformatics statistical testing will investigate correlations between evidence of immunological injury and graft loss to determine associative links.Students will be supported to transform their work into academic outputs e.g., poster presentations, oral presentations, and publications. They will benefit from having supervisors from both a scientific and clinical renal background.  |

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| **Project Title** | **A scoping review of concussion guidelines in sailing sports**  |
| **Supervisor(s)** | 1. Dr Neil Heron
 |
| **School / Centre** | Medicine; Centre for Public Health  |
| **Principal Supervisor’s Contact Details** | Email: N.Heron@qub.ac.uk | Tel: |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry |  |
| Microbiology |  |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) |  |
| **Background information:** | Concussion is an important public health concern and the UK recently released their grassroot concussion guidelines for use in community sport (https://www.sportandrecreation.org.uk/policy/research-publications/concussion-guidelines). However, this guidance needs to be adapted for the specific requirement of the individual sports. This project is therefore to undertake a scoping review of concussion guidelines within sailing sports and to then develop sailing-specific concussion guidelines from the scoping review results.  |
| **Aims / objectives** | To undertake a scoping review of concussion guidelines used within sailing sports. From these results, to then develop sailing-specific concussion guidance for use in sailing sports.  |
| **Techniques employed:** | Scoping review; literature review.  |

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| **Project Title** | **Exploring the impact of Food Insecurity on nutrition and health in Northern Ireland**  |
| **Supervisor(s)** | 1. Dr Claire McEvoy2. Dr Anne Nugent  |
| **School / Centre** | 1. School of Medicine, Dentistry and Biomedical Sciences/CPH2. School of Biological Sciences |
| **Principal Supervisor’s Contact Details** | Email: c.mcevoy@qub.ac.uk | Tel:07545246985 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry |  |
| Microbiology |  |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) |  |
| **Background information:** | Food insecurity, a condition of limited food availability due to inadequate resources, is a significant social determinant of health. The adverse effects of food insecurity on physical and mental health, including obesity, diabetes, and depression, have been suggested in children and adults. However, there is a paucity of research on food insecurity among Northern Irish adults, particularly as it relates to diet quality and risk of physical and mental health conditions. In Northern Ireland, food insecurity disproportionately affects younger adults/families and those who are unemployed or on low incomes. Understanding the impact of food insecurity on nutrition and health in vulnerable adults/families is critical to inform future policy and interventions to mitigate food insecurity.  |
| **Aims / objectives** | The overall aim of the project is to understand the determinants of food insecurity and the impacts of household food insecurity on nutritional intake and reported health status. |
| **Techniques employed:** | * Recruitment of food insecure adults/families
* Collection of quantitative data on food security, nutritional intake, nutritional status, and self-reported health
* Dietary assessment and analysis
* Data input and statistical analysis
* Academic write-up

 Depending on the student ability there will be an option to conduct qualitative research to understand the lived experience of food insecurity |

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| **Project Title** | **Exploring the relationship between depression and cognition: A secondary analysis of the NICOLA Harmonised Cognitive Assessment Protocol (HCAP) sub-study.** |
| **Supervisor(s)** | 1. Prof Bernadette McGuinness
2. Dr Leeanne O’Hara
3. Dr Calum Marr
 |
| **School / Centre** | Centre for Public Health  |
| **Principal Supervisor’s Contact Details** | Email:B.Mcguinness@qub.ac.uk | Tel:+44 (0)28 9097 8959 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry | ✓ |
| Microbiology |  |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) |  |
| **Background information:** | Depression has previously been associated with the incidence of cognitive impairment and dementia, suggesting that it may represent an important risk factor. It is therefore important to understand which individual factors might predict depressive symptoms, particularly among older adults who are at greater risk of cognitive decline. The Northern Ireland Cohort for the Longitudinal Study of Ageing (NICOLA) study is the largest study of ageing in Northern Ireland. The Harmonised Assessment Protocol (HCAP) is a sub-study of NICOLA designed to assess cognitive impairment and dementia in ~1000 individuals aged 65 and over. The NICOLA-HCAP sub-study is funded by the National Institute on Ageing (NIA) as part of a network for enhancing cross-national research within a worldwide group of population-based, longitudinal studies of ageing, all of which are centred around the US based Health and Retirement Study (HRS). The NICOLA-HCAP study has collected data on cognitive function and depressive symptoms, as well as various demographic variables that will allow for an exploration of potential risk/protective factors.  |
| **Aims / objectives** | 1. To examine the profile of depressive symptoms in the NICOLA HCAP cohort.
2. To explore the relationship between cognition and depression in NICOLA HCAP.
3. To identity risk and protective factors of depression in the NICOLA HCAP cohort.
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| **Techniques employed:** | The student will familiarise themselves with the HCAP study and HCAP study data file. They will create an analysis plan to answer the research questions presented. It is anticipated that the student will manipulate the data to create new variables, where necessary, for analysis. Descriptive statistics will be run on the data followed by tests of correlation (if required). Regression analysis will be performed. The results will be written up into a report that will form the basis of the student dissertation. |

**Projects Hosted by the Centre for Medical Education**

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| **Project Title** | **An investigation into the impact of repeated simulation in enhancing the learning experience of undergraduate medical students** |
| **Supervisor(s)** | 1. Dr Andrew D Spence
2. Professor Gerry Gormley
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| **School / Centre** | Centre for Medical Education |
| **Principal Supervisor’s Contact Details** | Email:a.spence@qub.ac.uk | Tel:02890 972215 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry |  |
| Microbiology |  |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) |  |
| **Background information:** | To meet the challenges of modern healthcare simulation-based training is being increasingly used as an educational tool. Commonly, simulation offers training in high acuity events that health professionals, and students, may not regularly encounter during training, e.g. cardiac arrests, challenging conversations or mental health crises. It enables skill development in a safe environment where students learn to prepare for a clinical environment.Within simulation-based learning, there is an increased focus on interprofessional education (IPE), often with medical and nursing students at its core. This has been shown to enhance knowledge, skills and confidence through a shared learning experience. Traditionally, simulation-based education is embedded as a component of university curricula, however many institutions also provide opportunities for students to practise skills in an extra-curricular setting, including Queen’s University Belfast (QUB). It is known simulation organised within the university curricula improves skill acquisition, however it can provoke powerful emotions, which may enhance or hinder learning. It is not known, however, if this is also found with students undergoing simulation that is not part of a scheduled class and where they have signed up voluntarily.Our proposed project will investigate the experiences of healthcare students undergoing simulation exercises in a non-mandatory extra-curricular setting. Participants from undergraduate medicine and nursing will be combined to create an IPE team approach to the simulations. These sessions will be organised to take place outside of the regular scheduled classes and participation will be voluntary. Semi-structured interviews will be conducted after the students have performed the simulation exercises where they have the opportunity to reflect on the event. Areas that will be explored include emotions prior to the simulation, experiences during the scenario and on its completion. These qualitative data will be analysed to establish themes to determine the experiences of students who undertake simulation as a non-mandatory activity. |
| **Aims / objectives** | ***Aims***The overall aim of this project is to investigate the effects of IPE simulation in an extracurricular setting on learner experience for undergraduate medical and nursing students. Publication of the results will inform the wider literature on the impact of non-mandatory additional simulation classes on learner experience.***Objectives***1. Perform a literature review of the experiences of healthcare students who undergo simulation scenarios.
2. Design a study protocol where students from both medical and nursing schools attend for a simulation scenario followed by semi-structured interviews.

Determine the year group of student that will be invited and how students will be recruited.Create simulation scenarios for the sessions.1. Implement this design and collect data using qualitative methodology.
2. After the data has been interpreted, aim to inform the academic community through presentations at conferences and publications in peer-reviewed journals.
 |
| **Techniques employed:** | 1. Perform a literature review.
2. Design a process using simulation-based education to assess the participant experience of simulation training in a non-mandatory extra-curricular setting.
3. Create a study protocol and achieve ethical approval.
4. Recruit students to participate in the research study.
5. Conduct the simulations using interprofessional based scenarios.
6. Perform semi-structured interviews after the scenarios.
7. Conduct thematic qualitative analysis on the collected audio data recordings.
8. Disseminate results through peer-reviewed publications and conference presentations.
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| **Project Title** | **Embedding diversity into Case-Based Learning (CBL) in healthcare education** |
| **Supervisor(s)** | 1. Dr Mairead Corrigan
2. Dr Paul Hamilton
 |
| **School / Centre** | Medical Education |
| **Principal Supervisor’s Contact Details** | Email: m.corrigan@qub.ac.uk  | Tel: 07860251957 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry |  |
| Microbiology |  |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*British Assoc DermatologistsDigestive Disorders FoundationPathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) |  |
| **Background information:** | CBL is core to teaching and learning in undergraduate medical education at Queen’s. Clinical cases used in CBL aim to reflect the diverse populations that doctors will be caring for. Challenges in embedding diversity into cases include striking a balance between the probability of certain diseases occurring among certain minoritized populations and the risk of stereotyping (John Cookson Ethnicity in clinical vignettes, Medical Teacher. 2023). Developing patient identities that intersect is another challenge. There does not exist any guidance about how to embed diversity into clinical cases. |
| **Aims / objectives** | Aims: To explore how to embed diversity into clinical cases.Objectives* Identify good practice;
* develop guidelines that can be used to review and write cases;
* develop a small number of clinical cases for different protected characteristics as exemplars.
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| **Techniques employed:** | * Review the literature;
* focus groups & surveys of students from different protected characteristics and analysis of these;
* interviews with third sector organisations;
* identify practices used by other medical schools.
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**Projects Hosted by the Patrick G Johnston Centre for Cancer Research**

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| **Project Title** | **Testing of a potential Cathepsin S PROTAC System for the Treatment of Inflammatory Disease** |
| **Supervisor(s)** | 1. Dr Rich Williams2. Emma Clyde-Allen |
| **School / Centre** | PGJCCR |
| **Principal Supervisor’s Contact Details** | Email: rich.williams@qub.ac.uk | Tel: 02890972791 |
| **Degree Pathway for which project is suitable (**✓**)** | Medical Science | ✓ |  |
| Biochemistry | ✓ |
| Microbiology |  |
| **Is project of suitable standard / subject for studentship application? (**✓**)** | *General awards*Wolfson Foundation Award | ✓ | *Subject-specific awards*Pathological SocietySir Colin Dollery Clinical Pharmacology AwardOther (Please specify) |  |
| **Background information:** | Cathepsin S (CatS) is a cysteine protease from the C1 clan of proteases. Research has shown that active CatS is expressed in immune and cancer cells, within the early and late-stage lysosomal structures, as well as being excreted from the cell. This cysteine protease has also been reported to play a significant role in several human inflammatory diseases, such as CF, COPD and cancer ([McKelvey, M. C.](https://pure.qub.ac.uk/en/persons/michael-mckelvey-2),  [Taggart, C. C.](https://pure.qub.ac.uk/en/persons/cliff-taggart) & [Weldon, S.](https://pure.qub.ac.uk/en/persons/sinead-weldon), 01 Apr 2022, In: [American Journal of Respiratory and Critical Care Medicine.](https://pure.qub.ac.uk/en/persons/cliff-taggart/publications/) 205, 7, p. 769-782 14 p., Wilkinson, R., Williams, R., *et a*l *Biological Chemistry*, **2015**, *396*, 867-882,). Research here at Queen’s University has shown that this protease leads to significant tissue re-modelling and damage within models of disease. Taggart and co-workers have shown that CatS is essential in the hallmarks of CF (Brown, R., [Small, D.](https://pure.qub.ac.uk/en/persons/donna-small), 20 Mar 2021, In: [Mediators of Inflammation.](https://pure.qub.ac.uk/en/persons/cliff-taggart/publications/) 2021, 10 p., 6682657). Both genetic manipulation and small molecule inhibitor treatment can block these disease phenotypes and highlight the potential of targeting this protease therapeutically. However, despite a significant effort in developing novel small molecule inhibitors there has been limited success in the clinical setting. From each of the clinical studies there is no reported safety issue in targeting CatS in dose escalation studies. Where each of these drugs appears to fail within clinical trials appears to be due to a lack of efficacy as measured by block antigen present (p10 fragment accumulation). In addition, we and others observe a stabilization in CatS expression upon CatS inhibition with a small molecule inhibitor. Whether this stabilization of CatS expression is responsible for the lack of efficacy as measured by the downstream biomarker (p10) is still matter of debate as there is currently no selective substrate available to measure activity.Targeted Protein degraders have been developed to target enzymes and receptors throughout the cell from the nucleus to the cell surface with significant success (Crews, C. Nat Rev Clin Oncol. 2023 Apr;20(4):265-278). These degrader systems are generated via coupling of protein of interest (POI) binder to a recruiter or cellular response trigger, such as an E3 ligase binder or Lipo tag. To date, there is no evidence of any research group developing a CatS specific degrader system, which could make a significant breakthrough in inflammatory disease treatment. Through this project you will test a series of Lipo-tagged CatS inhibitors for their ability to affect biomarker (p10 expression) and levels of the protease itself. |
| **Aims / objectives** | Aim 1: Test a series of tagged CatS inhibitors for intra-cellular activity via measuring p10 accumulation over time.Aim 2: Determine intracellular IC50 values at selected time point.Aim 3: Measure and determine changes in CatS expression in time and dose dependent manner. |
| **Techniques employed:** | Western BlotCell cultureCell assaysDrug Treatment |