Available Projects for Intercalated BSc (iBSc) in Medical Science

QUEEN'S UNIVERSITY BELFAST

2026-2027

5.1.26

Contents

Projects Hosted by the Johnston Cancer Research Centre

Projects Hosted by the Centre for Public Health

Projects Hosted by the Wellcome-Wolfson Institute For Experimental Medicine

Projects Hosted by the Johnston Cancer Research Centre

<table>
<tr><td>Project Title</td><td colspan="4">Exploring iRHOM2 as a Biomarker of Inflammation in the Acute Respiratory Distress Syndrome</td></tr>
<tr><td>Supervisor(s)</td><td colspan="4">1. Dr Colin Adrain, Johnston Cancer Research Centre

2. Dr Andrew Boyle, Clinical Lecturer and Specialty Trainee in Intensive Care Medicine at the Royal Victoria Hospital</td></tr>
<tr><td>School / Centre</td><td colspan="4">Johnston Cancer Research Centre</td></tr>
<tr><td>Principal Supervisor's Contact Details</td><td colspan="2">Email: c.adrain@qub.ac.uk</td><td colspan="2">Tel: 028 9097 2700</td></tr>
<tr><td>Is project of suitable standard / subject for studentship application? (ü)</td><td>General awards

Wolfson Foundation Award</td><td>✓</td><td>Subject-specific awards

British Assoc Dermatologists

Digestive Disorders Foundation

Pathological Society

Sir Colin Dollery Clinical Pharmacology Award

SK Chin Intercalated Degree Scholarship

Other (Please specify)</td><td>✓</td></tr>
<tr><td>Background information:</td><td colspan="4">Acute Respiratory Distress Syndrome (ARDS) is a severe inflammatory lung condition affecting critically ill patients and is associated with high mortality. Despite advances in supportive care, there are no effective drug treatments, in part because ARDS is biologically heterogeneous and driven by dysregulated immune responses. Pro-inflammatory cytokines such as tumour necrosis factor-α (TNFα) and interleukin-6 (IL-6) play central roles in disease pathogenesis.

The membrane protease ADAM17 regulates the release of TNFα, IL-6 receptor α, and epidermal growth factor receptor ligands, linking it to inflammation and tissue repair. ADAM17 activity is controlled by inactive rhomboid proteins (iRHOMs), particularly iRHOM2, which is selectively required for ADAM17 function in inflammatory cells. Targeting iRHOM2 is therefore an attractive strategy to suppress pathological inflammation while preserving beneficial ADAM17 activity in other tissues. Neutralising iRHOM2 antibodies have now progressed into early-phase clinical trials for inflammatory disease.

This project will investigate whether iRHOM2 expression is elevated in ARDS and will pilot a simple ELISA-based assay to explore its potential as a measurable biomarker.</td></tr>
<tr><td>Aims / objectives</td><td colspan="4">• Determine whether iRHOM2 expression is increased in ARDS patient samples versus non-inflamed controls
• Pilot an ELISA-based approach to detect iRHOM2 in extracellular vesicles</td></tr>
</table>

Techniques employed:	• RNA extraction from biobanked bronchoalveolar lavage samples • Quantitative real-time PCR (qPCR) to measure iRHOM2 mRNA expression • SDS-PAGE and immunoblotting to assess iRHOM2 protein levels • Isolation of extracellular vesicles from cell culture supernatants • Basic statistical analysis and data presentation This project will provide the student with hands-on experience in wet lab molecular biology techniques alongside the analysis and interpretation of biological datasets within a clinically relevant context.

Project Title	**Investigating the effects of SGLT2 inhibitors on haematopoietic cells to explore novel approaches to chronic anaemia in myeloid blood cancers**			
Supervisor(s)	1. Dr Graeme Greenfield (Clinical Senior Lecturer)			
School / Centre	Johnston Cancer Research Centre			
Principal Supervisor's Contact Details	Email: g.greenfield@qub.ac.uk			
Is project of suitable standard / subject for studentship application? (✓)	*General awards* Wolfson Foundation Award	✓	*Subject-specific awards* British Assoc Dermatologists Digestive Disorders Foundation **Pathological Society** Sir Colin Dollery Clinical Pharmacology Award SK Chin Intercalated Degree Scholarship Other (Please specify)	✓
Background information:	Many patients with chronic forms of myeloid blood cancers including myelodysplasia (MDS) and myelofibrosis (MF) have significant chronic anaemia. This significantly impacts on quality of life for these patients, may reduce long term survival and can result in transfusion dependence placing a large resource demand on the health service. There is a current unmet need for novel effective and tolerable approaches to help improve erythropoiesis in these patients. SGLT2 inhibitors increase the haemoglobin and haematocrit in other patient groups and clinical experience suggests they may have utility in the treatment of blood cancer patients with chronic anaemia. This project aims to assess the effects of these drugs directly on haematopoietic cell models.			
Aims / objectives	Investigate the effects of SGLT2 inhibitors on the gene expression profiles of haematopoietic cell models			
Techniques employed:	• Cell culture and *in vitro* drug treatment assays • RNA extraction • Real time Polymerase Chain Reaction (qPCR) • RNA sequencing • Bioinformatic analysis of large RNA-sequencing datasets This project will provide the student with experience in both wet lab molecular biology techniques and the analysis of large datasets			

<table>
<tr><td>Project Title</td><td colspan="4">Investigating Cellular Responses to DNA Abasic Sites Under Metabolic Stress</td></tr>
<tr><td>Supervisor(s)</td><td colspan="4">Melissa LaBonte Wilson

Robert Ladner</td></tr>
<tr><td>School / Centre</td><td colspan="4">Johnston Cancer Research Centre</td></tr>
<tr><td>Principal Supervisor's Contact Details</td><td colspan="2">Email:
m.labontewilson@qub.ac.uk</td><td colspan="2">Tel: 028 9097 2789</td></tr>
<tr><td>Is project of suitable standard / subject for studentship application? (✓)</td><td>General awards

Wolfson Foundation Award</td><td>✓</td><td>Subject-specific awards

British Assoc Dermatologists

Digestive Disorders Foundation

Pathological Society

Sir Colin Dollery Clinical Pharmacology Award

SK Chin Intercalated Degree Scholarship

Other (Please specify)</td><td>

✓

✓</td></tr>
<tr><td>Background information:</td><td colspan="4">DNA abasic sites, locations where the DNA base has been removed, occur frequently as part of normal cellular metabolism and are further induced under conditions that disrupt the nucleotide pool or DNA repair pathways. Although cells possess robust mechanisms to repair these lesions, accumulation of abasic sites can alter genome stability, replication dynamics, and cell survival.

Our lab investigates how abasic lesions form, persist, and influence cellular stress responses, specifically after chemotherapy treatment (e.g. 5-Fluorouracil (5-FU)). This intercalated project will focus on a small, well-defined component of that larger effort: establishing simple, quantitative readouts of how cultured cancer cells respond to conditions that modestly increase abasic site burden. Our collaboration with CV6 Therapeutics, has led to the evaluation of a first-in class dUTPase inhibitor in clinical trials with 5-FU and this project builds on the underlying biology of this promising new combination treatment.

This introductory project will suit an intercalated medical student interested in cancer biology, DNA repair, or mechanisms that influence therapeutic response. The work will also introduce practical laboratory skills and scientific reasoning applicable across biomedical research.</td></tr>
<tr><td>Aims / objectives</td><td colspan="4">The goal of this project is to generate pilot data describing cellular responses to experimentally induced abasic DNA lesions under controlled conditions.

Specific objectives are:
• To optimise a simple assay for detecting cellular stress or viability changes following induction of abasic lesions using metabolic or chemical perturbations known to influence nucleotide balance.
• To quantify how cells adapt to or tolerate mild abasic DNA stress, using standard readouts such as metabolic activity, cell viability, or checkpoint-related protein markers.
• To perform basic statistical analysis (N=3) to evaluate the reproducibility and significance of observed responses.

The project is intentionally narrow in scope to allow the student to complete all experimental cycles, data interpretation, and write-up within the intercalated timeframe.</td></tr>
</table>

Techniques employed:	Students will receive full training in all techniques, to include: • Cell culture of established human cancer cell lines • Induction of mild DNA/base lesion stress using metabolic or chemical treatments • Cell viability/metabolic assays (e.g. CellTiter-Glo or equivalent) • Basic protein analysis (e.g. Western blot for one or two stress-response markers such as γH2AX or pKAP1) • Detection of DNA abasic sites using an aldehyde-reactive probe (ARP)–based AP site assay. • Data handling, graphing, and statistical analysis appropriate for N=3 • Literature review, project planning, and dissertation writing

<table>
<tr><td>Project Title</td><td colspan="4">Molecular stratification of colorectal cancer</td></tr>
<tr><td>Supervisor(s)</td><td colspan="4">1. Philip Dunne</td></tr>
<tr><td>School / Centre</td><td colspan="4">SMBDB/PGJCCR</td></tr>
<tr><td>Principal Supervisor's Contact Details</td><td colspan="2">Email:
p.dunne@qub.ac.uk</td><td colspan="2">Tel:
02890972792</td></tr>
<tr><td>Is project of suitable standard / subject for studentship application? (✓)</td><td><u>General awards</u>

Wolfson Foundation Award</td><td>✓</td><td><u>Subject-specific awards</u>

British Assoc Dermatologists

Digestive Disorders Foundation

Pathological Society

Sir Colin Dollery Clinical Pharmacology Award

SK Chin Intercalated Degree Scholarship

Other (Please specify)</td><td>✓</td></tr>
<tr><td>Background information:</td><td colspan="4">Colorectal cancer (CRC) is a heterogeneous disease in which differences in tumour biology profoundly influence prognosis and treatment response. The Dunne group is a computational biology group (no "wet-lab" researchers) that focuses on understanding phenotypic plasticity and molecular stratification in CRC, as demonstrated in our recent work (Malla et al., Nature Genetics, 2024). Leveraging large-scale transcriptomic and multi-omic datasets, we seek to uncover robust tumour subtypes, integrate genomic and clinical data, and explore biological pathways that underpin disease progression.

For students that have existing R-based programming skills, this project offers an exceptional opportunity to apply cutting-edge computational biology to a major clinical challenge, contribute to ongoing research in an internationally recognised cancer research programme, and develop outputs of potential publishable quality.</td></tr>
<tr><td>Aims / objectives</td><td colspan="4">This project requires existing R expertise as a gatekeeper skill to unlock the exciting biology locked within molecular datasets. It offers the opportunity to work with real patient-derived datasets (e.g., TCGA, GEO), applying advanced bioinformatic approaches to generate biologically and clinically meaningful insights.

Analyses will include unsupervised clustering (e.g. hierarchical clustering), dimensionality reduction (PCA, UMAP), differential expression (DESeq2, edgeR), pathway enrichment (clusterProfiler, fgsea), integration with clinical metadata, and high-quality visualisation (ggplot2, ComplexHeatmap).

All analyses are aimed at improving our understanding of CRC and will be performed in R, using reproducible workflows documented via R Markdown.</td></tr>
<tr><td>Techniques employed:</td><td colspan="4">Essential Skills: This is a fully computational project and is not designed as an introduction to R. To get the most out of this project, applicants must already possess a working proficiency in R programming, including data wrangling, statistical analysis, and visualisation. This is an essential technical requirement.

Informal discussions are encouraged before application, please contact Dr Philip Dunne (p.dunne@qub.ac.uk) to arrange a short meeting to review prior experience and ensure suitability, where we can discuss your previous R-based projects, including code examples or a link to a GitHub repository where possible.</td></tr>
</table>

<table>
<tr><td>Project Title</td><td colspan="4">Great vessel toxicity following thoracic and neck radiotherapy: a systematic review of clinical studies</td></tr>
<tr><td>Supervisor(s)</td><td colspan="4">1. Dr Gerard Walls
2. Prof Karl Butterworth</td></tr>
<tr><td>School / Centre</td><td colspan="4">Johnston Cancer Research Centre</td></tr>
<tr><td>Principal Supervisor's Contact Details</td><td colspan="2">Email:
g.walls@qub.ac.uk</td><td colspan="2"></td></tr>
<tr><td>Is project of suitable standard / subject for studentship application? (✓)</td><td>General awards

Wolfson Foundation Award</td><td>✓</td><td>Subject-specific awards

British Assoc Dermatologists

Digestive Disorders Foundation

Pathological Society

Sir Colin Dollery Clinical Pharmacology Award

SK Chin Intercalated Degree Scholarship

Other (Please specify)
• The Medical Undergraduate Intercalated Scholarship
• Association of Physicians</td><td>✓</td></tr>
<tr><td>Background information</td><td colspan="4">Radiotherapy comprises a standard component of treatment for patients diagnosed with breast cancer, oesophageal cancer, unresectable lung cancer, combined with surgery, chemotherapy and immunotherapy. Despite technological advances in radiotherapy planning and delivery, the risk of damage to surrounding organs in the chest remains high. Cardiovascular problems, including arrhythmia, heart failure and ischaemic events, occur in 10–25%, and result in hospitalisations and additional medications are detrimental for quality of life during survivorship (PMID 39506136) and are also associated with a 20% excess risk of death. However, while the literature on the cardiac tissues features strongly in clinical multidisciplinary meetings and scientific congresses, the vasculature receives comparatively little consideration. The impact of high-dose irradiation on the aorta, carotids other large vessels is known to include stenosis, accelerated atherosclerosis and thrombosis, but the prevalence of these problems in the setting of modern radiotherapy is not known. There has been no synthesis of the available data on great vessels to date, or any attempt to refine the 30-year old guidelines on the safe radiation dose limits for these structures. In this project, the results of all relevant published studies will be collated and summarised to enable a contemporary analysis of radiotherapy effects on the great vessels.</td></tr>
<tr><td>Aims / Objectives</td><td colspan="4">The objective of this intercalated project is to conduct a systematic review of clinical impact of thoracic radiotherapy on the great vessels. A literature database (MEDLINE, Embase, Web of Science) search will be employed to identify relevant studies, with registration on PROSPERO in advance. The student will lead the screening of articles with support from the supervisor and other team members. Data will then be extracted regarding vessel toxicities, timepoints, radiotherapy factors and patient baseline health. The resulting data will then be synthesised into tables and plots for analysis and interpretation and write-up.</td></tr>
</table>

Techniques employed	The objective of this intercalated project is to conduct a systematic review of clinical impact of thoracic radiotherapy on the great vessels. A literature database (MEDLINE, Embase, Web of Science) search will be employed to identify relevant studies, with registration on PROSPERO in advance. The student will lead the screening of articles with support from the supervisor and other team members. Data will then be extracted regarding vessel toxicities, timepoints, radiotherapy factors and patient baseline health. The resulting data will then be synthesised into tables and plots for analysis and interpretation and write-up.

<table>
<tr><td>Project Title</td><td colspan="4">Uncovering How Genetic Variants Influence Breast Cancer Risk Using Functional Genomics</td></tr>
<tr><td>Supervisor(s)</td><td colspan="4">1. Prof Nick Orr

2. Dr Sarah Maguire</td></tr>
<tr><td>School / Centre</td><td colspan="4">SMDBS / JCRC</td></tr>
<tr><td>Principal Supervisor's Contact Details</td><td colspan="2">Email: nick.orr@qub.ac.uk</td><td colspan="2">Tel: x2095</td></tr>
<tr><td>Is project of suitable standard / subject for studentship application? (✓)</td><td>General awards

Wolfson Foundation Award</td><td>✓</td><td>Subject-specific awards

British Assoc Dermatologists

Digestive Disorders Foundation

Pathological Society

Sir Colin Dollery Clinical Pharmacology Award

SK Chin Intercalated Degree Scholarship

Other (Please specify)</td><td></td></tr>
<tr><td>Background information:</td><td colspan="4">Genome-wide association studies (GWAS) have identified hundreds of loci associated with breast cancer risk. However, for the vast majority of these loci, the causal variants, target genes, and underlying biological mechanisms remain unresolved. Most risk-associated variants lie in non-coding regions of the genome and are thought to influence disease susceptibility by altering the activity of enhancers and other regulatory elements that control gene expression in relevant cell types.

Post-GWAS functional characterisation is therefore essential to bridge the gap between statistical associations and biological understanding. Modern high-resolution experimental approaches now allow systematic evaluation of regulatory variants and candidate enhancers, enabling researchers to identify functional single nucleotide polymorphisms (SNPs), define their transcriptional consequences, and link them to the genes they regulate.

This project will focus on characterising one or more breast cancer predisposition loci using a combination of reporter assays, CRISPR-based perturbation systems, and high-throughput enhancer activity screens. The study will contribute to the broader effort to map the regulatory architecture underlying breast cancer susceptibility mechanisms and may highlight novel pathways relevant to disease risk, tumour initiation, and precision prevention.</td></tr>
<tr><td>Aims / objectives</td><td colspan="4">Overall Aim:
To functionally characterise regulatory variants and candidate enhancers at breast cancer GWAS loci using a combination of targeted and high-throughput experimental approaches.

Specific Objectives:
1. Prioritise candidate causal variants at selected GWAS loci using fine-mapping and regulatory annotation datasets (e.g., chromatin accessibility, histone marks, transcription factor binding).
2. Assess allele-specific regulatory activity of prioritised SNPs using dual-luciferase reporter assays (DLRAs) and electrophoretic mobility shift assays (EMSAs).
3. Interrogate enhancer function in situ using CRISPR activation (CRISPRa) and CRISPR interference (CRISPRi) to determine the effect of enhancer perturbation on gene expression.</td></tr>
</table>

	4. **Integrate functional data** to identify likely effector genes and propose plausible mechanistic pathways linking variants to breast cancer susceptibility.
Techniques employed:	Students will receive training in, and undertake, a range of wet-lab and computational techniques including: **Molecular and Cellular Techniques** • **Dual-luciferase reporter assays (DLRAs):** Testing allele-specific enhancer activity of candidate regulatory elements. • **Electrophoretic mobility shift assays (EMSAs):** Assessing differential transcription factor binding to risk vs reference alleles. • **CRISPRa/CRISPRi perturbation assays:** Modulating enhancer activity in breast epithelial cell models to measure expression changes in putative target genes. **Computational and Analytical Approaches** • Prioritisation of candidate causal variants using fine-mapping tools and publicly available epigenomic datasets. • Sequence design and cloning strategies for reporter constructs. • Analysis of reporter assay outputs • Integration of functional findings with gene expression and chromatin interaction data.

Project Title	**What is the real-world clinical experience for targeted treatment in patients with poor prognostic V600E *BRAF*MT colorectal cancer in the United Kingdom.**			
Supervisor(s)	1. Dr. Sandra Van Schaeybroeck (Clinical Reader – QUB-JCRC/BHSCT) 2. Dr. Ian Overton (Reader – QUB JCRC)			
School / Centre	Medicine, dentistry, biomedical sciences Johnston Cancer Research Centre			
Principal Supervisor's Contact Details	Email: s.vanschaeybroeck@qub.ac.uk		Tel: +2890972954	
Is project of suitable standard / subject for studentship application? (✓)	*General awards* Wolfson Foundation Award	✓	*Subject-specific awards* British Assoc Dermatologists **Digestive Disorders Foundation** Pathological Society Sir Colin Dollery Clinical Pharmacology Award SK Chin Intercalated Degree Scholarship Other (Please specify)	✓
Background information:	*BRAF*V600E mutated metastatic (stage IV) colorectal (CRC) cancer accounts for 10-15% of CRC patients, and has a 3 times poorer survival rate compared to patients with BRAF and KRAS non-mutated CRC tumours. These CRC patients have also a poorer response to standard of care chemotherapy treatment and in contrast to BRAFV600E mutated melanoma (skin cancer) patients, do not respond to specific targeting of the BRAFMT protein alone. Based on recent positive data from a phase III clinical study, targeted combination of BRAF specific inhibition (called encorafenib) in combination with specific inhibition of the epidermal growth factor receptor (using monoclonal antibody cetuximab) has been approved by EMEA, FDA and NICE. The new combination targeted treatment encorafenib/cetuximab has been introduced for patients in NI and other parts of the UK in 2021. This study will explore now real-world efficacy of this novel treatment regimen in NI and other part in the UK and assess clinical associations with long-term responses/outcome. Understanding why some patients have better outcomes with this novel treatment will inform health services and future combinations with this novel treatment.			
Aims / objectives	1) Compare real-world outcome and safety of this novel treatment regimen with its data published in the phase III clinical trial. 2) Compare outcomes of this novel combination in patients treated in NI and other parts in the UK. 3) Investigate key clinical details with long-term benefit from this treatment in this population (such as sites of secondary metastases etc).			
Techniques employed:	The study population will be NI and UK stage IV BRAF mutated colorectal cancer group who received this novel targeted approach (encorafenib – cetuximab) from 2021 and 2025 inclusive. Anonymous data from NI patients is already available, anonymous UK data will be collected through REDCap. The database contains anonymous clinical information such as age, date of surgery and start treatment, duration on treatment and date of death. Additional data including toxicity, type of organs involved with disease will be available. Simple and descriptive bioinformatics statistical testing will investigate eg. outcome and associations between type and extent of organs involved and outcome. 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 colorectal cancer background.			

Projects Hosted by the Centre for Public Health

<table>
<tr><td>Project Title</td><td colspan="4">Why do some kidney transplants last longer than others?</td></tr>
<tr><td>Supervisor(s)</td><td colspan="4">1. Dr Gareth McKay[1] (Reader)

2. Dr Michael Corr[1,2] (Clinical Fellow- Nephrology Registrar)</td></tr>
<tr><td>School / Centre</td><td colspan="4">1. Centre for Public Health
2. Regional Nephrology and Transplant Centre, Belfast City Hospital</td></tr>
<tr><td>Principal Supervisor's Contact Details</td><td colspan="2">Email:
g.j.mckay@qub.ac.uk</td><td colspan="2">Tel: +44 (0)28 9097 8958</td></tr>
<tr><td>Is project of suitable standard / subject for studentship application? (✓)</td><td>General awards

Wolfson Foundation Award</td><td>✓</td><td>Subject-specific awards

British Assoc Dermatologists

Digestive Disorders Foundation

Pathological Society

Sir Colin Dollery Clinical Pharmacology Award

SK Chin Intercalated Degree Scholarship

Other (Please specify)</td><td></td></tr>
<tr><td>Background information:</td><td colspan="4">Kidney transplantation is the gold-standard treatment for individuals living with end-stage kidney disease (ESKD), profoundly improving quality of life and significantly reducing morbidity and mortality. In Northern Ireland (NI) alone, over 3,000 people have received a kidney transplant.

Whilst the average kidney transplant lasts 15–20 years, an increasing number of patients experience ultralong-term transplant survival (greater than 20 years). The reasons why these patients achieve such exceptional outcomes remains poorly understood. Exploring the unique characteristics of these cases may reveal insights that could help extend the lifespan of all kidney transplants. This could have profound implications, reducing the need for patients to return to dialysis or undergo more complicated second or third transplants.</td></tr>
<tr><td>Aims / objectives</td><td colspan="4">This project seeks to identify factors associated with ultralong-term kidney transplant survival through two objectives:

1. Review existing literature to map current knowledge on ultralong-term graft survival.
2. Analyse demographic and clinical characteristics associated with ultralong-term transplant survival using a prospective clinical database of transplant recipients in NI.</td></tr>
<tr><td>Techniques employed:</td><td colspan="4">Students will gain experience in the following areas:

1. Literature Review: Use systematic scoping review methodology to synthesise findings from current research on ultralong-term graft survival.
2. Clinical Epidemiology and Data Analysis: Utilise the NI Kidney Transplant Database (IRAS ID 239344, REC 18/NI/0004), which contains comprehensive demographic and clinical data for transplant recipients. Will perform descriptive bioinformatics and statistical analysis to identify potential correlations between patient characteristics and prolonged transplant survival.

Student Support and Opportunities</td></tr>
</table>

	This project offers mentorship from both scientific and clinical nephrology supervisors. Previous students have successfully transformed their work into academic outputs, including poster and oral presentations at conferences, and peer-reviewed publications.

<table>
<tr><td>Project Title</td><td colspan="4">Agreement between self-reported and registry-recorded cancer prevalence in the Northern Ireland Cohort for the Longitudinal study of Ageing</td></tr>
<tr><td>Supervisor(s)</td><td colspan="4">1. Dr Gareth McKay[1] (Reader)

2. Dr Damien Bennett[1,2] (Clinical Lead Northern Ireland Cancer Registry)</td></tr>
<tr><td>School / Centre</td><td colspan="4">1. Centre for Public Health
2. Norther Ireland Public Health Agency</td></tr>
<tr><td>Principal Supervisor's Contact Details</td><td colspan="2">Email:
g.j.mckay@qub.ac.uk</td><td colspan="2">Tel: +44 (0)28 9097 8958</td></tr>
<tr><td>Is project of suitable standard / subject for studentship application? (✓)</td><td>General awards

Wolfson Foundation Award</td><td>✓</td><td>Subject-specific awards

British Assoc Dermatologists

Digestive Disorders Foundation

Pathological Society

Sir Colin Dollery Clinical Pharmacology Award

SK Chin Intercalated Degree Scholarship

Other (Please specify)</td><td></td></tr>
<tr><td>Background information:</td><td colspan="4">Cancer-specific health literacy may influence individual patient access to relevant cancer information, and their adherence to cancer prevention guidelines, including screening. For those diagnosed with cancer, health literacy may influence understanding of the diagnosis and associated treatment, leading to possibly poorer outcomes, including reduced quality of life and survival. Comparison of self-reported cancer with clinically confirmed medical diagnosis may highlight patients' perception of their diagnoses and assess the validity of self-reported information.
Reliability of self-reported health conditions may be influenced by multiple factors including age, sex, education, socioeconomic status and health condition, although, the validity of self-reported measures remains relatively underexplored.
Our objective is to assess agreement with, and describe the validity of, the self-reported measures in a cohort of participants from the Northern Ireland Cohort for the Longitudinal study of Ageing (NICoLA) with linked data from the Northern Ireland Cancer Registry (NICR).</td></tr>
<tr><td>Aims / objectives</td><td colspan="4">Reliance on self-reported health status information as a measure of population health can be challenging due to errors associated with participant recall. We seek to investigate agreement between self-reported and registry-recorded site-specific cancer diagnoses from a cohort of participants from the NICoLA study. We have linked self-reported cancer history information from the NICoLA cohort and the NICR which will be used to evaluate validity measures (sensitivity, specificity, positive predictive value, negative predictive value, kappa etc).</td></tr>
<tr><td>Techniques employed:</td><td colspan="4">Students will gain experience in the following areas:

1. Literature Review: Use systematic scoping review methodology to synthesise findings from the current published literature to evaluate the reliance of self-reported site-specific cancer diagnoses in relation to clinically specified outcomes.
2. Multiple logistic regression statistical modelling to assess independent associations of demographic and socioeconomic variables with incorrect reporting of cancer diagnoses.

Student Support and Opportunities

This project offers mentorship from both scientific and clinical supervisors. Previous students have successfully transformed their work into academic outputs, including poster and oral presentations at conferences, and peer-reviewed publications.</td></tr>
</table>

Project Title	**A scoping review of concussion guidelines in blind and/or deaf football**			
Supervisor(s)	1. Dr Neil Heron			
School / Centre	Centre for Public Health, Queen's University Belfast			
Principal Supervisor's Contact Details	Email: N.Heron@qub.ac.uk		Tel:	
Is project of suitable standard / subject for studentship application? (✓)	*General awards* Wolfson Foundation Award	✓	*Subject-specific awards* British Assoc Dermatologists Digestive Disorders Foundation Pathological Society Sir Colin Dollery Clinical Pharmacology Award SK Chin Intercalated Degree Scholarship Other (Please specify)	
Background information:	Physical activity and sport is important to promote to everyone to maintain a healthy lifestyle, including those with a disability. However, injuries do occur in sport and can affect people's ability to participate and therefore gain the associated health benefits. One important injury to be aware of is concussion and concussion is an important public health concern. However, concussion guidance needs to be adapted for the specific requirement of the individual athlete and sports. This project is therefore to undertake a scoping review of concussion guidelines within blind and/or deaf football and to then develop specific concussion guidelines for these sports from the scoping review results.			
Aims / objectives	1. Undertake a scoping review of concussion guidelines in blind and/or deaf football. 2. From the scoping review results to then develop concussion guidelines for the blind and/or deaf football sports.			
Techniques employed:	Scoping review; evidence synthesis.			

<table>
<tr><td>Project Title</td><td colspan="4">A systematic review and meta-analysis on the physical demands of squash</td></tr>
<tr><td>Supervisor(s)</td><td colspan="4">1. Dr Neil Heron</td></tr>
<tr><td>School / Centre</td><td colspan="4">Centre for Public Health, Queen’s University Belfast</td></tr>
<tr><td>Principal Supervisor’s Contact Details</td><td colspan="2">Email: N.Heron@qub.ac.uk</td><td colspan="2">Tel:</td></tr>
<tr><td>Is project of suitable standard / subject for studentship application? (✓)</td><td>General awards

Wolfson Foundation Award</td><td>✓</td><td>Subject-specific awards

British Assoc Dermatologists

Digestive Disorders Foundation

Pathological Society

Sir Colin Dollery Clinical Pharmacology Award

SK Chin Intercalated Degree Scholarship

Other (Please specify)</td><td></td></tr>
<tr><td>Background information:</td><td colspan="4">Background: Squash is a multidirectional high-intensity intermittent sport for male and female individuals. Although several studies have attempted to characterise the physical demands of squash, a meta-analysis is still lacking.

Methods: PubMed, Embase, CINAHL and SPORTDiscus will be searched from inception. A backward citation search will be conducted for included articles using Scopus. The PECOS framework will be used to formulate eligibility criteria.

Population: squash players of regional, national or international playing levels (juniors and adults).

Exposure: singles and doubles match play. Comparison: sex (male/female), playing levels.

Outcomes: duration of play, on-court movements and stroke performance.

Study design: cross-sectional, longitudinal. Pooled means or mean differences with 95% confidence intervals will be calculated. A random-effects meta-analysis with robust variance estimation will be performed. The measures of heterogeneity will be Cochrane Q and 95% prediction intervals.</td></tr>
<tr><td>Aims / objectives</td><td colspan="4">Objective: We will aim to describe and synthesise the physical demands of squash across the different performance levels and sexes.</td></tr>
<tr><td>Techniques employed:</td><td colspan="4">Systematic review; meta-analysis; evidence synthesis.</td></tr>
</table>

Projects Hosted by the Wellcome-Wolfson Institute For Experimental Medicine

<table>
<tr><td>Project Title</td><td colspan="4">Investigating the function of CARD-only proteins in human inflammasome regulation</td></tr>
<tr><td>Supervisor(s)</td><td colspan="4">1. Dr Rebecca Coll

2. Dr Chloe McKee</td></tr>
<tr><td>School / Centre</td><td colspan="4">WWIEM</td></tr>
<tr><td>Principal Supervisor's Contact Details</td><td colspan="2">Email: r.coll@qub.ac.uk</td><td colspan="2">Tel: 6473</td></tr>
<tr><td>Is project of suitable standard / subject for studentship application? (✓)</td><td>General awards

Wolfson Foundation Award</td><td>✓</td><td>Subject-specific awards

British Assoc Dermatologists
Digestive Disorders Foundation
Pathological Society

Sir Colin Dollery Clinical Pharmacology Award

SK Chin Intercalated Degree Scholarship

Other (Please specify)</td><td>✓
✓
✓</td></tr>
<tr><td>Background information:</td><td colspan="4">Inflammasomes are intracellular protein complexes that form part of the innate immune response to infection and injury. Inflammasomes provide a platform for the activation of caspase-1 which cleaves inflammatory cytokines such as interleukin(IL)-1beta into their active forms and causes a form of cell death known as pyroptosis.

Excessive activation of inflammasomes is associated with many diseases including Alzheimer's, Parkinson's, atherosclerosis, liver disease, and asthma. It is therefore critical to understand how inflammasomes are regulated in human immune cells, in order to develop new therapies for inflammatory diseases.

In humans (but not mice) several caspase recruitment domain (CARD)-only proteins (COPs) such as CARD16 have been identified as potential inflammasome regulators. The function of CARD16 is controversial with studies suggesting is has an anti-inflammatory effect while another suggest it has a pro-inflammatory role (1).

Importantly, the expression of CARD16 can be increased by stimulation of Toll-like receptors (TLRs) and the type 1 interferon (IFN) receptor, and transcriptomic data indicates that four differentially spliced isoforms of CARD16 are expressed in human macrophages(2). The alternative splicing of mRNAs is an important post-transcriptional mechanism that regulates protein expression across many systems including the innate immune response(3). The CARD16 isoforms include a longer form of CARD16 with an uncharacterised C-terminus of unknown function.

We hypothesise that different forms of CARD16 may be induced to differentially regulate inflammasome signalling.

References:
(1) Devi et al. Int J Mol Sci 2020 https://doi.org/10.3390/ijms21186901
(2) Vollmers et al J Biol Chem 2021 https://doi.org/10.1016/j.jbc.2021.100784</td></tr>
</table>

	(3) Carpenter et al Nat Rev Immunol https://doi.org/10.1038/nri3682
Aims / objectives	This project will examine how different CARD16 isoforms interact with components of the inflammasome such as apoptosis-associated speck-like protein containing a CARD (ASC) and caspase-1. This will be compared to interactions with the closely related proteins CARD17 and CARD18. Protein-protein interactions will be examined using co-immunoprecipitation assays and immunocytochemistry. The effect of CARD16 isoforms on inflammasome formation (NLRP3, NLRP1 and AIM2) will also be examined by flow cytometry. The results from this project will provide important insights into the expression and function of the inflammasome regulator CARD16.
Techniques employed:	The student will learn a range of lab techniques and skills such as cell culture, plasmid preparation and transfection, flow cytometry, Western blotting, immunocytochemistry and microscopy. The student will also learn data analysis and presentation skills.

<table>
<tr><td>Project Title</td><td colspan="4">Unseen enemies – Chasing bacteria hiding in human macrophages</td></tr>
<tr><td>Supervisor(s)</td><td colspan="4">Prof. Miguel A. Valvano, M.D. (Paediatrics & Infectious Diseases); Chair in Microbiology and Infectious Diseases</td></tr>
<tr><td>School / Centre</td><td colspan="4">Medicine, Dentistry and Biomedical Science, Wellcome-Wolfson Institute for Experimental Medicine</td></tr>
<tr><td>Principal Supervisor's Contact Details</td><td colspan="2">Email: m.valvano@qub.ac.uk
https://publish.uwo.ca/~mvalvano/</td><td colspan="2">Phone: 028 9097 6025</td></tr>
<tr><td>Is project of suitable standard / subject for studentship application? (✓)</td><td>General awards

Wolfson Foundation Award</td><td>✓</td><td>Subject-specific awards

British Assoc Dermatologists

Digestive Disorders Foundation

Pathological Society

Sir Colin Dollery Clinical Pharmacology Award

SK Chin Intercalated Degree Scholarship

Other (Please specify)</td><td>✓</td></tr>
<tr><td>Background information:</td><td colspan="4">Infections by Enterobacter species, steadily rising in the UK, are difficult to treat due to multidrug antibiotic resistance (AMR), including resistance to last-resort antibiotics like carbapenems and polymyxins. Enterobacter, represented by the last "E" of the ESKAPE acronym, are included in the WHO's list of priority global threat pathogens for which new treatments are urgently needed. Many Enterobacter are gut commensals, but they can cause bacteraemia, neonatal sepsis, and multi-organ infections. We have recently discovered that by hiding inside macrophages, Enterobacter lies dormant and does not stimulate any inflammatory responses, allowing it to escape the action of the few antibiotics that remain available for treating the infection (https://doi.org/10.1093/infdis/jiaf099) (https://www.qub.ac.uk/News/Allnews/2025/antibiotic-resistant-bacteria-hide-human-cells-without-alerting-immune-system.html)</td></tr>
<tr><td>Aims / objectives</td><td colspan="4">This intercalated student project is ideal to any medical student interested in an academic medicine future career.
The student will investigate the hypothesis that Enterobacter clinical isolates, traditionally viewed as extracellular bacteria, can establish hide in macrophages to delay macrophage cell death and detection by the innate immune system. Two key questions on the cellular microbiology of Enterobacter will be investigated: (1) What are the features of the Enterobacter containing vacuole? and (2) What are the molecular bases of the intramacrophage survival of Enterobacter? Unveiling the cellular microbiology of Enterobacter-macrophage interactions will open a door to enhance Enterobacter clearance by macrophages through the development of novel host-directed therapeutics.</td></tr>
<tr><td>Techniques employed:</td><td colspan="4">A combination of molecular genetics, deletion mutagenesis, state-of-the-art confocal microbiology and bioinformatic approaches will be used by the candidate to address these questions. While this project is on fundamental science, it has direct clinical relevance since bacterial intramacrophage survival may complicate the treatment of Enterobacter infections and explain documented treatment failures even in patients infected with antibiotic susceptible strains. Any medical student interested in academic medicine will benefit from acquiring direct laboratory experience and learning rigorous, hypothesis driven, scientific methodology.</td></tr>
</table>

<table>
<tr><td>Project Title</td><td colspan="4">Investigations into the role of endothelial injury to the progression of lung fibrosis in systemic sclerosis</td></tr>
<tr><td>Supervisor(s)</td><td colspan="4">1. Dr Bettina Schock

2. Mrs Amal ElBanna</td></tr>
<tr><td>School / Centre</td><td colspan="4">WWIEM</td></tr>
<tr><td>Principal Supervisor's Contact Details</td><td colspan="2">Email:
b.schock@qub.ac.uk</td><td colspan="2"></td></tr>
<tr><td>Is project of suitable standard / subject for studentship application? (✓)</td><td>General awards

Wolfson Foundation Award</td><td>✓</td><td>Subject-specific awards

British Assoc Dermatologists

Digestive Disorders Foundation

Pathological Society

Sir Colin Dollery Clinical Pharmacology Award

SK Chin Intercalated Degree Scholarship

Other (Please specify)</td><td>✓

✓

✓</td></tr>
<tr><td>Background information:</td><td colspan="4">Systemic sclerosis (SSc) is a rare autoimmune disease characterised by vascular dysfunction and fibrosis. One of its severe and life limiting complications is the development of interstitial lung disease (ILD), which occurs in up to 50% of SSc patients.
This project will explore the role of endothelial cells and their secretions in SSc. In particular, we are interested in hypoxia induced secreted proteins, that can reach and activate fibroblasts in the interstitium.

We will investigate the effects of these secretions on SSc and SSc-ILD fibroblasts, obtained from patients. For this we will use our established methos of peripheral blood derived induced pluripotent stem cells differentiated into fibroblasts.

The work will identify endothelial cell-derived molecular mechanisms leading to activation of fibroblasts. The most pro-fibrotic factors secreted by hypoxia-injured endothelial cells, will be further investigated as therapeutical targets to limit progression of lung fibrosis in SSc (e.g., Dapagliflozin) with the aim to identify already licenced drugs that can be repurposed in SSc-ILD.</td></tr>
<tr><td>Aims / objectives</td><td colspan="4">To investigate the role of the endothelium on fibroblast activation we will:

1. Determine the cytokine signature of hypoxia-injured endothelial cells (HUVEC cells)

2. Confirm the expression of hypoxia-injury derived mediators (HUVEC cells) at protein and gene expression level

3. Investigate the effect of hypoxia-injured endothelial secretions on the activation of patient-derived fibroblasts using functional assays.</td></tr>
</table>

Techniques employed:	As the successful candidate, you will join the vibrant pulmonary fibrosis laboratory of Dr Schock where you will learn: • cell culture of endothelial cells and fibroblasts, • hypoxia exposure, • protein analyses (soluble proteins by ELISA, cellular proteins by Western blotting), • gene expression (mRNA by qRT-PCR) and • functional assays (wound closure). We also run a weekly journal club / jab meeting where you can present your lab work and identify the role of the endothelium in driving fibrosis.

Project Title	**Unlocking the role of insulin receptor substrate-2 (IRS2) in diabetic kidney disease**			
Supervisor(s)	1. Dr. Derek Brazil 2. Dr. Sam Lockhart			
School / Centre	SMDBS/WWIEM			
Principal Supervisor's Contact Details	Email:d.brazil@qub.ac.uk		Tel: 6469	
Is project of suitable standard / subject for studentship application? (✓)	*General awards* Wolfson Foundation Award	✓	*Subject-specific awards* British Assoc Dermatologists Digestive Disorders Foundation Pathological Society Sir Colin Dollery Clinical Pharmacology Award SK Chin Intercalated Degree Scholarship Other (Please specify)	✓
Background information:	Insulin receptor substrate 2 (IRS2) is the key protein that regulates insulin-stimulated glucose uptake in liver, muscle, and fat cells. IRS2 acts as a scaffold of phosphorylation to integrate extracellular insulin signals to intracellular PI3Kinase, Akt, ERK, and other signalling pathways. Mice lacking IRS2 develop type 2 diabetes, and a loss of IRS2 expression is associated with insulin resistance in humans. Our team has identified a range of mutations in IRS2 that increase the risk of patients developing kidney disease in diabetes. These mutations are predicted to alter IRS2 protein expression and phosphorylation, reducing insulin signalling in kidney cells.			
Aims / objectives	This project aims to characterise these mutations by expressing these IRS2 mutant proteins in kidney cells and assessing insulin signalling, glucose uptake, and kidney cell survival. Responses to hypoxia and cell metabolism changes will also be assessed.			
Techniques employed:	This project is a molecular cell biology project involving techniques such as kidney cell culture, transfection, Western blotting, qPCR, immunocytochemistry, and a range of cell biology assays. The project is well suited to an ambitious, hard-working medical student who is interested in diabetes and kidney disease. The project will be co-supervised by Dr. Sam Lockhart, a clinical endocrinologist.			