2025 WWIEM SUMMER RESEARCH PROJECTS

QUEEN'S UNIVERSITY BELFAST | WELLCOME-WOLFSON INSTITUTE FOR EXPERIMENTAL MEDICINE

Program Information: https://www.qub.ac.uk/research-centres/wwiem/EducationandTraining/SummerStudentProgramme/

Apply at: https://forms.gle/Z5hbjmSVGgLtd3SH7

Email: cemsummerstudents@qub.ac.uk

Program Coordinator: Dr Rebecca Coll

***Project code is required for the application form.** ****LB=laboratory-based**

PROJECT CODE*	SUPERVISOR	PROJECT TITLE	PROJECT DESCRIPTION	LB**	RESEARCH GROUP/PI WEBSITE
P01	Dr Derek Brazil d.brazil@qub.ac.uk	Identifying the molecular mechanisms of GREM1 signalling in human disease	Levels of the secreted BMP antagonist Gremlin1 1 (GREM1) are increased in a range of human diseases including lung fibrosis, diabetic nephropathy and a range of human cancers including colorectal, breast and lung cancer. The canonical model of GREM1 signalling is binding to bone morphogenetic proteins (BMPs) and preventing BMP receptor signalling. However, many reports have identified additional non-BMP signalling modalities for GREM1 that may be significant for the pathogenic role of GREM1 in human disease. This project will examine a recently reported GREM1 signalling pathway involving MEK1/2 and ERK1/2 in prostate cancer cells. Aims: 1. Validate GREM1 inhibition of BMP signalling in cancer cells. 2. Treat cells with rhGREM1 over a range of concentrations and time-points and examine pERK1/2 and pMEK1/2 readouts. 3. Measure levels of pERK1/2 and pMEK1/2 in FFPE sections from WT and villin1-GREM1 transgenic mice using immunohistochemistry. This project represents an exciting opportunity for a student to learn a range of molecular cell biology techniques as part of the Brazil lab team. These experiments will help us to shed new light on the pathogenic signalling for GREM1 in human disease.	YES	https://pure.qub.ac.uk/en/persons/derek-brazil
P02	Prof. Mei Chen	Investigating a new treatment for Retinal Fibrosis	This project provides an exciting opportunity to explore innovative, cell-free therapies for retinal fibrosis, a major cause of vision loss. The student will work on generating and characterizing extracellular vesicles (EVs) derived from induced pluripotent stem cell (iPSC), iPSC-derived mesenchymal stem cells (iMSCs) and MSCs. Using advanced techniques like iPSC reprogramming and culture, differentiation protocols, immunostaining, and flow cytometry, EV isolation, Western blotting, nanoparticle tracking analysis (NTA), and immunofluorescence, the student will assess the anti-fibrotic potential of these EVs in in vitro models of retinal fibrosis. This hands-on experience will include exposure to state-of-the-art molecular biology and cell culture techniques. The project will not only enhance the student's technical skills but also provide a deeper understanding of translational research and the potential of regenerative medicine. These skills and experiences will be invaluable for pursuing future academic or industrial research opportunities in biomedical sciences.	YES	https://pure.qub.ac.uk/en/persons/mei-chen
P03	Dr Bianca Plouffe b.plouffe@qub.ac.uk	Insight into the neuroprotective function of the prostaglandin receptor EP2	G protein-coupled receptors (GPCRs) are the largest family of membrane proteins in humans. They are essential to human physiology and play key roles in disease. Approximately 35% of approved drugs target GPCRs so understanding their signalling is important for drug development. One such receptor, the prostaglandin EP2 receptor, has been associated to several functions in the central nervous system, including neuroprotection against excitotoxicity. Excitotoxicity occurs when excessive release of the neurotransmitter glutamate causes damage to surrounding neurons via overactivation of glutamate receptors in conditions such as stroke, seizures and neurodegenerative diseases. This project will study G protein activation by EP2 in the presence of EP2-activating compounds and the effect of EP2 activation in a cellular model of excitotoxicity.	YES	https://pure.qub.ac.uk/en/persons/bianca-plouffe

			The student will gain experience in cell culture, transfection, the use of bioluminescence resonance energy transfer (BRET)-based biosensors, RT-qPCR and excitotoxicity assays.		
P04	Dr Yvonne Dombrowski y.dombrowski@qub.ac.uk	Identifying novel immune targets for brain repair in multiple sclerosis and neurodegenerative diseases	Myelin is the protecting sheath around nerve fibres that enables nerve signalling. Damage to myelin can lead to neurodegeneration and permanent disability for patients. To date, there is no cure for neurodegenerative diseases such as Multiple Sclerosis (MS), Alzheimer's disease or dementia. Our research investigates novel immune targets to repair myelin damage and to prevent neurodegeneration. Inflammasomes are danger sensors of the immune system that initiate inflammation. In the brain too much inflammation can lead to neurodegeneration; conversely, however, myelin repair is also dependent on inflammation. It is not known if inflammasomes are beneficial or detrimental for brain repair. This project will investigate inflammasomes in brain repair in a model of MS, which could lead to the development of future therapeutics for MS patients. Students will learn tissue dissection, immunofluorescent staining, confocal microscopy, image analysis, and qPCR as well as transferable skills such as project/time management and communication skills.	YES	https://pure.qub.ac.uk/en/persons/yvonne-dombrowski
P05	Dr Rebecca Coll r.coll@qub.ac.uk	Do macrophages like it hot? Examining the thermal biology of the innate immune response	The innate immune system is our bodies' first line of defence against infection and injury. Sentinel cells like macrophages express a range of pattern recognition receptors (PRRs) that sense pathogens and danger. PRRs trigger signalling pathways that induce the expression of inflammatory cytokines and enhance phagocytosis and antigen presentation. PRR signalling triggers local inflammation but can also cause a systemic fever response. Fever is a highly conserved element of immunity, and heat was recognised by Celsus in the first century BC as a cardinal sign of inflammation. The evolution of the fever response indicates that it is essential to the control of infection, but the exact mechanisms that make it protective are not fully understood. This project will examine whether fever range temperatures effect PRR signalling in our macrophage model systems. The student will learn a range of techniques and skills including cell culture, ELISAs, Western blotting, and metabolism assays.	YES	https://pure.qub.ac.uk/en/persons/rebecca-coll
P06	Prof. Miguel A. Valvano m.valvano@qub.ac.uk	Exploiting a bacterial toxin to modulate the function of infected macrophages and epithelial cells	Burkholderia lethal factor 1 (BLF1) is a virulence-associated toxin produced by Burkholderia pseudomallei, the causative agent of melioidosis. BLF1 blocks protein synthesis by inactivating the eukaryotic initiation factor 4A, and it has been proposed as an anticancer agent. We have demonstrated that BLF1 can cross the bacterial cell membrane using a signal peptide-independent novel pathway without the assistance of any proteins secretion system; secreted BLF1 ends up associated with membrane vesicles (MVs) in the bacterial supernatant. Both membrane crossing and association to MVs require a novel type of N-terminal region in the protein that is critical for its transport. This region is sufficient to export heterologous recombinant proteins across bacterial membranes providing a host of medical applications to target proteins to eukaryotic cells. This project involves using learn molecular recombinant and cellular biology techniques to construct and deliver novel proteins to modulate macrophages and epithelial cells responses during infection.	YES	https://publish.uwo.ca/~mvalvano/
P07	Dr Bettina Schock b.schock@qub.ac.uk	How different are fibroblasts in systemic sclerosis (SSc)?	"Systemic Sclerosis (SSc) is characterised by irreversible scarring and organ failure and 40% of patients transition into Systemic Sclerosis with Interstitial Lung Disease (SSc-ILD), a disease with significant lung fibrosis and a higher mortality risk. However, the mechanisms underlaying this transition is still unknown. We have successfully developed	YES	https://pure.qub.ac.uk/en/persons/bettina-schock

			an iPSC-derived fibroblasts cell line from a patient with SSc, SSc-ILD and a healthy control. Here we will compare the responses of these fibroblasts to pro-fibrotic stimuli between the two disease phenotypes to understand why some patients develop SSc-ILD. Aim: Characterisation of iPSC-derived fibroblasts from patients with SSc and SSc-ILD. Methods: Fibroblasts will be stimulated with TGFβ1 and conditioned medium from airway epithelial cells exposed to hypoxia. Specific markers of fibroblast activation (Col1A1, SMAalpha, Vimentin, S100A4) will be determined at gene/protein level. Wound healing ability will be determined by analysis of collagen deposition and migration assay."		
P08	Dr Gunnar Schroeder g.schroeder@qub.ac.uk	Understanding host subversion by bacterial pathogens	Infections with bacterial pathogens are a global health problem, which is intensified by fast spreading antimicrobial resistance (AMR) and increasing populations with immunodeficiencies or underlying health conditions. To design urgently needed new treatments, we must improve our understanding about the molecular infection mechanisms. This project will focus on Legionella pneumophila, the etiological agent for a severe form of pneumonia in immunocompromised patients, known as Legionnaires' disease. L. pneumophila uses a Dot/Icm Type IV secretion system to inject >330 effector proteins into the host cell cytoplasm to hijack cellular processes, ensuring its own intracellular survival and extracting nutrients for replication. Integrating microbiology, genetic engineering, mammalian cell culture, infection assays, and advanced microscopy to investigate the function of Legionella effectors, this project will offer an exciting opportunity to gain experience in key biomedical research techniques, design and analysis of experiments and contribute to the fight against an emerging health threat.	YES	https://pure.qub.ac.uk/en/persons/gunnar-neels-schroeder
P09	Dr Guilherme Costa g.costa@qub.ac.uk	How do RNA-protein interactions shape blood vessels?	Blood vessels are formed during embryonic development, and later in life, they grow under particular circumstances to maintain tissue homeostasis. In some pathological conditions, such as cancer and diabetes, blood vessel formation is activated but occurs in a dysregulated manner, with critical consequences for tissue health. In this project, you will explore the importance of RNA biology in vascular endothelial cells and how these molecules regulate vessel formation, in homeostasis and disease. To do so, you will use a fascinating toolkit of techniques that allow for the visualisation of RNAs and RNA-binding proteins (RBPs) in endothelial cells. You will have the opportunity to be trained by a group of enthusiastic scientists and gain skills in cutting-edge techniques involving cell culture, molecular biology and microscopy.	YES	https://gcosta064.wixsite.com/costa-lab
P10	Dr David A. Cisneros d.cisneros@qub.ac.uk	Molecular mechanisms of bacterial competition in the gut microbiome and their influence on chronic intestinal inflammation and colorectal cancer	This project investigates how bacterial competition in the gut microbiome influences chronic inflammation, aiming to identify key microbial genes and mechanisms driving intestinal inflammation; focusing on pathobionts from the human microbiome. Utilizing metagenomic sequencing, in vitro assays, or animal models, the project seeks to uncover the causes and consequences of pathobiont colonisation of the gut. For students, this project offers a rich learning experience and valuable job opportunities. They will gain interdisciplinary knowledge in microbiology, immunology, or bioinformatics. The project contributes to develop skills necessary in biotechnology, pharma, healthcare, and public health, as well as opportunities in startups focused on microbiome-based therapies.	YES	https://www.cisneros-lab.org/