DARWIN TRUST OCTOBER 2026 ENTRY

Project Catalogue

Instructions for applicants

King's College London accepts applications for the Darwin Trust PhD studentship awards up until 23:59 UK time on 30th January 2026. **Applications will be capped at 50**

Applications will open on the 15th December 2025. Any applications submitted before then won't be accepted.

INSTRUCTIONS:

By the deadline of 23:59 UK time on 30th January 2026, you MUST:

- Submit an application to King's College London for a full-time MPhil/PhD degree programme with a 2026-27 start date (i.e., from 1 October 2026) using the King's online admissions application system – King's Apply
- **Choose ONE project** from the project catalogue – enter this project choice (A -R) in the box that asks for project description and put the title on your personal statement.
- Enter the correct funding code in your application:
    - On the Funding section of your online King's Admissions Application Form, tick the box at Item 5 (Award Scheme Code or Name).
    - Award Scheme code: **This will be available on the 15th December 2025**
    - Important: Applicants who fail to input this exact code (the bit in bold) as instructed will not be identified during the admissions data extraction process and will therefore not be considered for the scholarship, so it is vital that you do this correctly!
- Ensure that you have submitted all the required supporting documentation as part of your online Admissions application, to include:
    - Official degree transcripts and certificates, including professional/university-made translations.
    - Proof of English language proficiency (see below for further guidance).*
    - Two references
    - Most up to date CV
    - Personal statement **– include rationale for the project you have picked**

** English Language Requirements: PLEASE NOTE: if you are currently studying a master's degree in a majority English speaking country, such as the UK and USA, you may still be required to provide a valid English language test certificate by 14 January 2026. If you have previously taken an English language test, please upload your test certificate to your admissions application. Test results are valid if the test was taken within two years of the start date of your chosen programme.*

For full guidance about Postgraduate English language entry requirements, please refer to the webpage at: https://www.kcl.ac.uk/study/postgraduate-taught/how-to-apply/entry-requirements/english-language-requirements.

For any queries relating to English language requirements, please contact the King's Admissions Team via your online application.

NOTE: if you experience problems accessing an English Language test centre, King's accepts online test options such as, such as the TOEFL Home Edition and IELTS (Online). NB: we currently are not accepting results from the Pearson Test of English (Academic) Online.

Important: The results turnaround time for some English tests is up to 10 working days, therefore, if you need to take a test, please plan accordingly.

PLEASE BE AWARE that material from your admissions application will be extracted and used as part of the assessment/selection process. As part of the standard admissions process, you may be contacted about missing material/documents so please ensure you log in to King's Apply regularly to check for any messages that the Admissions team send you, however this is not guaranteed, and it is your responsibility to ensure that all the relevant information and supporting materials are provided in time. Applications that are incomplete by the given deadline will not be considered.

Project A

Title
Antimicrobial gene delivery for the prevention of preterm birth

Project Details
Co-Supervisor 1A: Dr Ashley Boyle School: School of Life Course & Population Sciences KCL/KHP email: Ashley.boyle@kcl.ac.uk KCL/KHP Website: https://www.kcl.ac.uk/people/ashley-boyle
Co-Supervisor 1B: Dr Natalie Suff School: School of Life Course & Population Sciences KCL/KHP email: natalie.suff@kcl.ac.uk KCL/KHP Website: https://www.kcl.ac.uk/people/natalie-suff
Project description: Preterm birth (delivery before 37 weeks) is a global health issue with no effective methods of prevention or treatment. In the UK, 60,000 babies are born prematurely every year, often leading to brain damage, disability, or death. Preterm birth is commonly caused by bacteria travelling up from the vagina into the womb. The cervix forms a barrier to protect the baby, producing protective mucus with antibacterial proteins to fight infection. Damage to the cervix can weaken this defence and increase the risk of premature birth. Increasing antibacterial proteins around the cervix may prevent infection spreading and protect the baby's developing organs including the brain and gut. We have established a mouse model that mimics this infection pathway. By making bacteria glow, we can track their movement through the cervix with imaging. Gene therapy is a technology that can deliver DNA, as a medicine, to prevent or treat diseases. This project will develop a therapy that increases antibacterial proteins in the cervix using gene delivery technology. This project has the potential to generate novel therapeutic insights for one of the most urgent challenges in maternal and child health. Year 1 will focus on optimising this therapy to specifically target cervical cells using cloning techniques and comparing viral vectors with biodegradable nanoparticles. Year 2 will test these therapies our mouse model of preterm birth, investigating the effects on the maternal and neonatal tissues. Year 3 will explore the molecular mechanisms of these therapies. The student will gain skills and training in animal models of disease, live imaging, molecular biology techniques, gene therapy production, data interpretation and statistics. The student will be supported to present their work at conferences and publish their findings in peer-reviewed journals.
<u>One</u> representative publication from <u>each co-supervisor</u>: Boyle, A. K., Tetorou, K., Suff, N., Beecroft, L., Mazzaschi, M., Karda, R., Hristova, M., Waddington, S. N. & Peebles, D., 2025, Ascending Vaginal Infection in Mice Induces Preterm Birth and Neonatal Morbidity, American Journal of Pathology, 195:891-906 Suff N, Karda R, Diaz JA, Ng J, Baruteau J, Perocheau D, Taylor PW, Alber D, Buckley SMK, Bajaj-Elliott M, Waddington SN, Peebles D. Cervical Gene Delivery of the Antimicrobial Peptide, Human β-Defensin (HBD)-3, in a Mouse Model of Ascending Infection-Related Preterm Birth. Front Immunol. 2020 Feb 11;11:106. doi: 10.3389/fimmu.2020.00106. PMID: 32117260; PMCID: PMC7026235.

Project B

Title
RNA binding protein regulation of cancer cell metabolism and tumorigenesis

Project Details
Co-Supervisor 1A: Maria R Sasi Conte School - BMBS KCL/KHP email sasi.conte@kcl.ac.uk KCL/KHP Website: https://www.kcl.ac.uk/people/sasi-conte
Co-Supervisor 1B: Agi Grigoriadis School – FoDOCS, CCRB KCL/KHP email agi.grigoriadis@kcl.ac.uk KCL/KHP Website: https://www.kcl.ac.uk/people/agamemnon-agi-grigoriadis
Project description: Cancer cells have altered metabolism to cope with high energy demands associated with cell proliferation, migration and survival. The Conte and Grigoriadis labs explore the use of metabolic profiling, transcriptomics and proteomics to elucidate the role of two RNA binding proteins, LARP4A and LARP4B, that are key regulators of cancer cell behaviour and tumour progression. We recently demonstrated that silencing of LARP4A and 4B reduce tumour formation and alters the metabolism of cancer cells. LARP4A and 4B regulate the translation of a subset of mRNAs but the full list of target RNAs regulated by each LARP4 protein and the mechanisms linking mRNA translation regulation, cancer cell behaviour, metabolism and tumourigenesis is not known. We have identified a few metabolic and signalling pathways affected by these proteins. Moreover, LARP4A and LARP4B deletion appears to show differential cellular response at mitochondrial level. In this project the student will be trained in complementary skills of both Conte and Grigoriadis labs, including RNA structural biology, cancer cell biology and *in vivo* technology to achieve the following goals: Year 1-2: Perform metabolomics, respirometry and proteomics profiles of cancer cell lines under different stress conditions (hypoxia, starvation) that are depleted in LARP4A and/or LARP4B using siRNA and CRISPR/Cas9 technologies. Experiments will be analysed by targeted and untargeted profiling for analysis of global changes. Year 2-3: Validation of selected dysregulated genes, analyse metabolic profiles and function following small molecule inhibition and selected mutagenesis. Analysis of changes of RNA interactome profile in cells and in vitro under different stress conditions Years 3-4: Functional investigation of targets *in vivo* using multiple xenograft transplantation approaches; molecular analysis of LaRP4A/B RNA target interactions and write-up. **These studies will shed light for the first time on the mechanisms underlying the cancer-related functions of LARP4A and LARP4B.**
<u>One</u> representative publication from <u>each co-supervisor</u>: Cruz-Gallardo, I., Martino, L., Kelly, G., Atkinson, A., Trotta, R., De Tito, S., Coleman, P., Ahdash, Z., Gu, Y., Bui, T.TT **Conte, M.R.*** (2019) LARP4A recognises polyA RNA via a novel binding mechanism mediated by disordered regions and involving the PAM2w motif, revealing interplay between PABP, LARP4A and mRNA. *Nucleic Acids Res.,* 47:4272-4291*,* doi: 10.1093/nar/gkz144 Coleman JC, Tattersall L, Yianni V, Knight L, Yu H, Hallett SR, Johnson P, Caetano A, Cosstick C, Ridley A, Gartland A, **Conte MR*, Grigoriadis AE***. The RNA binding proteins LARP4A and LARP4B promote sarcoma and carcinoma growth and metastasis *iScience.* 2024, 27(4):109288. doi: 10.1016/j.isci.2024.109288

Project C

Title
Project title: Effects of Type 1 Diabetes on ovarian follicular cells

Project Details
Co-Supervisor 1A: Dr James Bowe School: School of Cardiovascular and Metabolic Medicine & Sciences KCL/KHP email: james.bowe@kcl.ac.uk KCL/KHP Website: https://kclpure.kcl.ac.uk/portal/en/persons/james.bowe
Co-Supervisor 1B: Dr Kim Jonas School: Life Course and Population Health Sciences KCL/KHP email: kim.jonas@kcl.ac.uk KCL/KHP Website: Kim Jonas - King's College London
Project description: Polycystic Ovary Syndrome (PCOS) is the most common endocrine disorder in women of reproductive age, with a prevalence of 6-15% worldwide. The aetiology of PCOS is linked to altered levels of the pituitary hormones LH and FSH, dysfunctional ovarian follicle development and elevated androgen levels. This results in reduced fertility and additional adverse effects including obesity and Type 2 Diabetes (T2D). The deleterious interaction between T2D and PCOS has been well-characterised in previous studies. It is less appreciated that approximately 25% of women with Type 1 Diabetes (T1D) also develop PCOS, a prevalence considerably higher than the general population. T1D is characterised by autoimmune destruction of the insulin-producing beta-cells of the pancreas. While insulin resistance and hyperinsulinaemia drives the interaction between T2D and PCOS, individuals with T1D classically have normal insulin sensitivity but an inability to produce insulin. Thus, the association between T1D and PCOS is likely to involve distinct pathophysiological mechanisms, though these are poorly understood. This study aims to assess how T1D influences the transcriptional profile of ovarian follicles at different stages of development. This is a collaborative project between Dr James Bowe who has a background in animal models of diabetes and Dr Kim Jonas who has a background in ovarian physiology, the student will receive training from experienced members of both labs. The first year objective will use in vivo techniques to characterise the metabolic and reproductive phenotype of T1D mice. They will learn to isolate the ovarian follicles and targeted qPCR approaches to assess changes in key marker of follicular function. From year 2 the student will use scRNAseq approaches to assess the broader transcriptional profile of follicular cells at different stages of development. Subsequent studies towards the end of the PhD will use histological approaches including RNAscope to assess transcriptional changes alongside morphological effects.
One representative publication from each co-supervisor: Bowe JE, Hill TG, Hunt KF, Smith LI, Simpson SJ, Amiel SA, Jones PM (2019) A role for placental kisspeptin in β cell adaptation to pregnancy. JCI Insight, 4(20). doi: 10.1172/jci.insight.124540 Johnson GP, Onabanjo CGA, Hardy K, Butnev VY, Bousfield GR, Jonas KC (2022) Follicle-Stimulating Hormone Glycosylation Variants Distinctly Modulate Pre-antral Follicle Growth and Survival. Endocrinology 163(12); DOI: 10.1210/endocr/bqac161

Project D

Title
Modelling Manganese Neurotoxicity in Slc30a10 knockout mice

Project Details
Co-Supervisor 1A: Karin Tuschl School: School of Cancer & Pharmaceutical Sciences KCL/KHP email: karin.tuschl@kcl.ac.uk KCL/KHP Website:
Co-Supervisor 1B: Po-Wah So School: IoPPN KCL/KHP email: po-wah.so@kcl.ac.uk KCL/KHP Website: https://kclpure.kcl.ac.uk/portal/en/persons/po-wah.so
Project description: Background and Aims Manganese is an essential trace metal vital for brain physiology. However, its levels must be tightly regulated, as both deficiency and excess impair neuronal function. Loss-of-function mutations in the metal transporters SLC30A10 and SLC39A14 cause distinct manganese dyshomeostasis phenotypes, offering valuable insights into mechanisms of metal regulation and neurotoxicity. Using a Slc30a10 knockout mouse model of manganese overload, this project will characterise the role of Mn in different brain cell populations and determine how its dyshomeostasis affects neuronal function and synaptic integrity. **Objectives** 1. Identify neuronal cell types that accumulate manganese (Year 1). Mass cytometry (immunostaining with heavy metal–conjugated antibodies followed by laser ablation-inductively coupled- mass spectrometry) will map manganese distribution across the brain of Slc30a10 knockout mice. Thus, we will be able to discriminate multiple neuronal and glial subtypes within the same sample to identify those with preferential manganese accumulation. 2. Determine neuronal subtype-specific effects of manganese excess (Year1-3). Acute brain slices from Slc30a10 knockout mice will be used to assess calcium dynamics and synaptic vesicle release using fluorescent probes and electrophysiology, identifying vulnerable neuronal populations. 3. Assess the molecular impact of manganese excess (Year 3-4). Transcriptomic and proteomic profiling of synaptosomes will identify the molecular consequences of manganese neurotoxicity. Genes and proteins of interest will be validated using CRISPRi technology, Western blotting and immunohistochemistry. **Skills Development** The student will gain interdisciplinary training in neuroscience, molecular biology, and analytical chemistry, including confocal microscopy, mass cytometry, proteomics, and electrophysiology. They will acquire molecular biology expertise in genotyping, qPCR, Western blotting, molecular cloning, and CRISPRi technology. The project also provides experience in experimental design, quantitative data analysis, and scientific communication, preparing the student for a career in molecular and cellular neurobiology, metal homeostasis, and translational neuroscience.
One representative publication from each co-supervisor: Tuschl K, White RJ, Trivedi C, Valdivia LE, Niklaus S, Bianco IH, Dadswell C, González-Méndez R, Sealy IM, Neuhauss SCF, Houart C, Rihel J, Wilson SW, Busch-Nentwich EM. Loss of slc39a14 causes simultaneous manganese hypersensitivity and deficiency in zebrafish. Dis Model Mech. 2022;15:dmm044594. Ashraf A, Jeandriens J, Parkes HG, So PW. Iron dyshomeostasis, lipid peroxidation and perturbed expression of cystine/glutamate antiporter in Alzheimer's disease: Evidence of ferroptosis. Redox Biology. 2020;32:101494. doi: 10.1016/j.redox.2020.101494.

Project E

Title
New Insights in the spatiotemporal dynamics of the KNDy neural network shaping reproduction.

Project Details
Co-Supervisor 1A: Professor Kevin OByrne School of Life Course & Population Sciences KCL/KHP email kevin.obyrnekcl.ac.uk KCL/KHP Website: https://kclpure.kcl.ac.uk/portal/en/persons/kevin-obyrne
Co-Supervisor 1B: Professor Paul Taylor School of Life Course & Population Sciences KCL/KHP email paul.taylorkcl.ac.uk KCL/KHP Website: https://kclpure.kcl.ac.uk/portal/en/persons/paul.taylor
Project description: ***Scientific basis:*** Successful reproduction requires normal function of a neural oscillator, the gonadotrophin-releasing-hormone (GnRH) pulse generator, which drives pulsatile release of gonadotrophic-hormones (LH/FSH) to control ovulation and spermatogenesis. This oscillator comprises hypothalamic Kisspeptin neurones co-expressing Neurokinin-B (NKB) and Dynorphin (acronym-KNDy). Using *in-vivo* real-time monitoring of *single*-KNDy neurones in-situ we have revealed for the first-time that there is a complex repertoire of mini-synchronised events (mSEs; synchronisation between 2-or-more neurones) building up to the full-blown SEs characteristic of LH pulses. However, the precise role of the key neurotransmitters (NKB, dynorphin and glutamate) in orchestrating the emergence of these mSEs and SEs remains to be established. ***Techniques:***We have a unique opportunity to establish how individual KNDy neurones entrain and synchronise to produce the dynamic SE architecture underlying normal reproduction. To interrogate the system, we will use cutting-edge *in-vivo* GRadient-INdex-(GRIN)-lens miniendoscopy with the latest *in-vivo* CRISPR-Cas9-gene-editing methods to selectively knockdown glutamate, NKB, or dynorphin expression in KNDy neurones. All techniques are well established in the laboratory and training provided by PIs and postdocs. *Kiss1*-Cre mice will receive an injection of AAV1-FLEX-SaCas9-U6-**sgVlut2** or **-sgNkB** or **-sgDyn** together with Cre-dependent GCaMP6m-AAV for calcium imaging of KNDy neurones via GRIN-lens placement in the hypothalamus. ***Aim and Objectives:*** **Aim:** To test the hypothesis that synchronisation in the KNDy-neural network is a dynamic phenomenon driven by mini-synchronisation events (mSEs) involving intricate interactions between glutamate, neurokinin-B, and dynorphin signalling, which collectively orchestrate GnRH pulse generation required for successful reproduction. **Objectives:** Using *in-vivo* GRIN-lens recordings from *single*-KNDy neurones across the female ovarian-cycle and in males, and CRISPR-Cas9 mutagenesis, interrogate the integrative role of glutamate (Year-1), neurokinin-B (Year-2) and dynorphin (Year-3) transmission in synchronising the KNDy-network to initiate and sustain pulsatile dynamics of the GnRH pulse generator. Year-4 will be used to consolidate remaining analysis, write papers and complete thesis.
One representative publication from each co-supervisor: Voliotis M, Li XF, De Burgh R, Lass G, Lightman SL, **O'Byrne KT**, Tsaneva-Atanasova K. Mathematical modelling elucidates core mechanisms underpinning GnRH pulse generation. J Neuroscience. 2019; pii: 0828-19. doi: 10.1523/JNEUROSCI.0828-19.2019. Maragkoudaki X, Naylor M, Papacleovoulou G, Stolarczyk E, Rees D, Pombo JM, Abu-Hayyeh S, Czajka A, Howard JK, Malik AN, Williamson C, Poston L & **Taylor PD**. (2020) Supplementation with a prebiotic (polydextrose) in obese mouse pregnancy improves maternal glucose homeostasis and protects against offspring obesity. Int J Obes DOI: 10.1038/s41366-020-00682-5.

Project F

Title
Molecular dissection of cancer-related anaemia

Project Details
Co-Supervisor 1A: Dr Stephan Menzel **School:** School of Cancer and Pharmaceutical Sciences **KCL/KHP email:** stephan.menzel@kcl.ac.uk **KCL/KHP Website:** https://www.kcl.ac.uk/people/dr-stephan-menzel
Co-Supervisor 1B: Dr Mohammad Mahdi Karimi **School:** School of Cancer and Pharmaceutical Sciences **KCL/KHP email** mohammad.karimi@kcl.ac.uk **KCL/KHP Website:** https://www.kcl.ac.uk/people/mohammad-mahdi-karimi
Project description: The daily production of billions of red blood cells packed with haemoglobin in the bone marrow is a life-critical, albeit extremely resource-intensive cellular and molecular processes. Anaemia is often caused by erythropoietic stress, which can be induced through cancer-associated processes that are often present at the time of diagnosis for solid cancers. Stress erythropoiesis is characterised by the increased occurrence of so-called F cells, which retain a production of fetal haemoglobin. Our group has recently discovered that such F cells are produced through 3 separate erythropoietic sub-lineages. F cells and stress erythropoiesis are poorly characterised so far. The student will use cutting edge molecular biological tools, such as single-cell multiomics, single cell ATAC-seq and cut-and run investigation of the binding of critical transcription factors to characterise such cells at an early developmental stage in the human system. In addition to these wet-lab experiments, the student will also apply cutting-edge analytical tools to analyse her/his own results under the supervision of Dr Karimi.
<u>One</u> representative publication from <u>each co-supervisor</u>: (1) Fetal-hemoglobin-expressing red blood cells ("F cells") consist of three distinct types as revealed by single-cell transcriptomic analysis of circulating reticulocytes. Rooks H, Ng C, Oikonomopoulos S, Hoss SE, Turner C, Kong KL, Mian S, Daniel Y, Ojewunmi OO, Brewin J, Rees D, Mufti GJ, Ragoussis J, Strouboulis J, Menzel S. Hemasphere. 2025 Jun 25;9(6):e70174. doi: 10.1002/hem3.70174. eCollection 2025 Jun. (2) Prompt-based bioinformatics: a new interface for multi-omics analysis. Awan AR, Oveisi M, Karimi MM. Nat Rev Genet. 2025 Aug 11. doi: 10.1038/s41576-025-00889-0.

Project G

Title
Rebuilding the Brain: Molecular Biology of Neuroregeneration in Earthworms

Project Details
Co-Supervisor 1A: Stephen Sturzenbaum School: School of Cancer and Pharmaceutical Sciences KCL/KHP email: stephen.sturzenbaum@kcl.ac.uk KCL/KHP Website: www.toxicogenomics.info
Co-Supervisor 1B: Christer Hogstrand School: School of Cancer and Pharmaceutical Sciences KCL/KHP email: christer.hogstrand@kcl.ac.uk KCL/KHP Website: https://www.kcl.ac.uk/people/christer-hogstrand
Project description: Background This project builds on Charles Darwin's pioneering studies of earthworms, extending his legacy with modern molecular science. In 1881 Darwin published a book to reveal the extraordinary importance of earthworms in shaping ecosystems and behaviour. Today, we know that earthworms can regenerate their cerebral ganglion (brain) after surgical removal, an ability mammals largely lack. Preliminary work highlights iron as a crucial factor: while the brain appears structurally intact after four weeks, iron is only replenished by 10–12 weeks. This suggests that metal homeostasis is tightly coupled to functional recovery. Transcriptomic analyses have also identified unannotated genes differentially expressed during regeneration, offering novel candidates for discovery. This makes earthworms a powerful but underutilised model for studying neural repair. **Training and Skills** The student will train in molecular biology (RNAseq, RNAscope, qPCR), metallomics (LA-ICP-MS, XRF, XANES), ROS assays, advanced imaging (confocal microscopy, TEM, histology), and bioinformatics (transcriptome assembly, WGCNA, phylogenetics). Alongside technical skills, the project emphasises communication, experimental design, and data integration, combining Darwin's observational tradition with cutting-edge molecular biology. The student will benefit from a supportive environment as reflected in Co-Supervisor 1A's 2014 and 2019 Supervisory Excellence Award and a 2024 Student Support Award. **Objectives** In the **first year**, the student will establish the regeneration model, optimise iron supplementation and chelation, and build expertise in histology and imaging. In the **second year**, they will map iron distribution and oxidative stress during regeneration, echoing Darwin's observational studies. The **third year** will focus on identifying and validating unannotated genes using RNAseq, qPCR, and RNAscope. In the **final year**, the datasets will be integrated into a systems-level model of brain regeneration, culminating in thesis completion and dissemination. By uniting Darwin's earthworm with modern molecular approaches, this project illuminates fundamental principles of biology while addressing questions central to regenerative medicine.
One representative publication from each co-supervisor: **Stürzenbaum SR**, Hoeckner M, Panneerselvam A, Levitt J, Bouillard J-S, Taniguchi S, Dailey L-A, Ahmad Khanbeigi RA, Thanou M, Rosca EV, Suhling K, Zayats A, Green M (2013) Biosynthesis of luminescent quantum dots in an earthworm. Nature Nanotechnol. 8(1):57-60. Hu X, Xiao W, Lei Y, Green A, Lee X, Maradana MR, GaoY, Xie X, Wang R, Chennell G, Basson MA, Kille P, Maret M, Bewick GA, Zhou Y, **Hogstrand C** (2023) Aryl hydrocarbon receptor utilises cellular zinc signals to maintain the gut epithelial barrier. Nature Commun. 14, 5431.

Project H

Title
Molecular Pathways of Neutrophil Dysregulation by Air Pollution in Chronic Airway Disease.

Project Details
Co-Supervisor 1A: Dr. Annika Warnatsch **School:** School of Immunology and Microbial Sciences, Peter Gorer Department of Immunobiology **KCL/KHP email:** Annika.warnatsch@kcl.ac.uk **KCL/KHP Website:** https://www.kcl.ac.uk/research/warnatsch-group
Co-Supervisor 1B: Dr Rocio Martinez-Nunez **School:** School of Immunology and Microbial Sciences, Dept Infectious Diseases **KCL/KHP email:** rocio.martinez_nunez@kcl.ac.uk **KCL/KHP Website:** Lab website: https://www.kcl.ac.uk/research/martinez-nunez-lab CDDU: https://www.kcl.ac.uk/research/cddu
Project description: SCIENTIFIC BASIS: Neutrophils are the most abundant immune cell in blood and first responders upon infection. Dysregulated, they can cause inflammation in chronic airway diseases such as severe asthma and COPD, which is worsened by exposure to Particulate Matter (PM) air pollution. Neutrophils deploy powerful antimicrobial weapons such as phagocytosis, production of Reactive Oxygen Species (ROS) and formation of Neutrophil Extracellular Traps (NETs) - webs of DNA decorated with histones and antimicrobials that trap pathogens. Our findings show that NET composition varies with the stimulus: PM, viral infection, or cytokine exposure each generate distinct signatures. Aberrant NET formation is increasingly recognised as a driver of tissue injury and chronic inflammation. This project will dissect how PM induces stimulus-specific NET phenotypes, how these are sensed by immune receptors such as Toll-like receptors, and how altered NET composition affects antiviral immunity and inflammatory responses in the lung. **TECHNIQUES AND SKILLS:** The student will gain expertise in immune cell isolation, culture, flow cytometry, protein and gene expression analysis, advanced microscopy, and high-throughput imaging for NET quantification. They will apply proteomics and RNA-sequencing to profile NETs and their effects, and use molecular tools to interrogate ROS/Nox2 and PAD4 pathways. Training will include experimental design, image and transcriptomic data analysis, and scientific communication. **OBJECTIVES: Year 1:** Establish foundational methodologies including neutrophil isolation, PM exposure, and imaging methods to quantify NET release and composition under different stimuli. **Year 2:** Define molecular pathways through which PM alters neutrophil function. **Year 3:** Characterise PM-induced NET signatures using proteomics and identify receptors on immune and epithelial cells that sense these structures. **Year 4:** Dissect downstream signalling and secondary effects of NET recognition, including cytokine production and modulation of antiviral responses during respiratory viral infection.
One representative publication from each co-supervisor: **Inflammation. Neutrophil extracellular traps license macrophages for cytokine production in atherosclerosis.** Warnatsch A, Ioannou M, Wang Q, Papayannopoulos V. 2015 Jul 17, In: ***Science***. 349(6245):316-20. doi: 10.1126/science.aaa8064. Epub 2015 Jul 16. PMID: 26185250; PMCID: PMC4854322. **The RNA binding proteins ZFP36L1 and ZFP36L2 are dysregulated in airway epithelium in human and a murine model of asthma.** Rynne J, Ortiz-Zapater E, Bagley DC, Zanin O, Doherty G, Kanabar V, Jackson D, Parsons M, Rosenblatt J, Adcock I, Martinez-Nunez RT. Front Cell Dev Biol. 2023;11:1241008. doi: 10.3389/fcell.2023.1241008.

Project J

Title
Investigating the role of PAK4 in the nucleus of breast cancer cells

Project Details
Co-Supervisor 1A: Prof Claire Wells School Cancer and Pharmaceutical Sciences KCL/KHP email claire.wells@kcl.ac.uk KCL/KHP Website: https://www.kcl.ac.uk/research/invasion-and-metastasis-research-group
Co-Supervisor 1B: Dr Fiona Wardle School BMBS KCL/KHP email fiona.wardle@kcl.ac.uk **KCL/KHP Website:** https://www.kcl.ac.uk/research/wardle-group
Project description: PAK4 kinase is overexpressed in breast cancer (BC) and has been associated with proliferation/matrix invasion. PAK4 has both cytoplasmic and nuclear localisations. However, very little is known about PAK4 nuclear function, whether it requires kinase activity and if it is related to cell invasion. Moreover, PAK4 transcriptional activity has not been explored. We have preliminary data suggesting PAK4 regulates its own expression. We work with CRUK to develop PAK kinase inhibitors (PAKi). It will be important to understand how these inhibitors might/might not interfere with PAK4 nuclear functionality. There is also evidence that PAK4i leads to double stranded DNA breaks (DSBs). This suggests that PAK4i could synergize with PARP inhibition. We aim to investigate four key questions: 1. Is PAK4 regulating gene expression? 2. Is PAK4 associated with the repair of DSBs? 3. Does PAK4 nuclear function require kinase activity? 4. Does PAK4 nuclear function influence matrix invasion? **Year 1:** Test appearance DSBs in PAK4KO/PAKi BC cells using immunofluorescence and investigate kinase dependence. Use CUT&RUN to identify PAK4 binding sites in genomic sequence. Optimize PAK4 nuclear localisation for RNA-Seq analysis. **Year 2:** RNA-Seq of PAK4 driven gene expression using nuclear targeting /PAKi with a focus on DNA repair/cell invasion networks. Validate DNA interactions (Y1) to identify gene regulation mechanisms. **Year 3** Identify DNA repair proteins that interact with PAK4 in BC. Test synergising PARPi with PAKi in BC proliferation. **Year 4** Test the ability of PAK4KO BC cells to invade collagen using kinase/nuclear localisation mutants. Consolidate experiments from Year 1-3 , write thesis. **Training:** Genomics techniques including CUT&RUN, RNA-seq and library preparation, bioinformatic analysis of genomics datasets, high resolution live/fixed cell imaging, biochemistry, cell biology. This project will be a collaboration with Prof Iweala, Covenant University Nigeria, proving expertise in cancer genomics and networking support for the student.
One representative publication from each co-supervisor: *PAK6 acts downstream of IQGAP3 to promote contractility in triple negative breast cancer cells* (2024) Aikaterini Pipili , Nouf A. Babteen, Lujain Kuwair, Mahfuja Bulu Jannet, Jelmar Quist, Karine K.V. Ong , Ryan Pitaluga, Anita G. Grigoriadis, Andrew Tutt, Claire M. Wells *Cellular Signalling*, Volume 121, Article 111233 Yaa, R.M., Schilder, B.M., Acemel, R. D., Wardle, F.C. (2025). Chromatin Interaction and Histone Mark Signatures Associated With TBXT Expression in Metastatic Lung Cancer. Genes Chromosomes Cancer, 64(3):e7004.

Project K

Title
Smart Functional Electrospun Nanofibre Textiles for Shade and Shield: Rethinking Sunlight Modulation in Protective Clothing for Darker Skin

Project Details
Co-Supervisor 1A: Paul F. Long School: School of Cancer & Pharmaceutical Sciences KCL/KHP email: paul.long@kcl.ac.uk KCL/KHP Website: Paul Long | King's College London
Co-Supervisor 1B: Bahijja Raimi-Abraham School: School of Cancer & Pharmaceutical Sciences KCL/KHP email: Bahijja.raimi_abraham@kcl.ac.uk KCL/KHP Website: Bahijja Raimi-Abraham | King's College London
Project description: Sunlight-induced reactive oxygen species (ROS) can disrupt gene and protein function, impairing skin physiology. Electronically excited species formed during ROS-mediated lipid peroxidation and protein or nucleic acid oxidation produce high-energy intermediates. Their decomposition results in ultraweak photon emission (UPE), primarily within the visible spectrum. Blue light (~450 nm), the most energetic visible wavelength, has been shown to significantly elevate both UPE and ROS levels in skin. Blue light can exacerbate post-inflammatory hyperpigmentation by stimulating melanin production, particularly in individuals with darker skin tones. This may lead to long-term discolouration, psychological distress, and an increased risk of keloid or hypertrophic scarring (both conditions have limited therapeutic options with suboptimal outcomes). Functional textiles offer a promising intervention. These may incorporate antioxidants (e.g., vitamin C), mineral barriers (e.g., zinc oxide), niacinamide (vitamin B3), or structural/light-filtering features using materials like titanium dioxide. Electrospun nanofibres can be engineered for selective blue-light attenuation. Aim: 1. Determine whether UPE generated from blue-light-induced ROS in the epidermis can be absorbed by dermal chromophores, establishing a novel mechanism of epidermal–dermal signalling. **Aim 2.** Translate this insight into the development of electrospun nanofibre-based functional textiles to selectively modulate blue light exposure. **Objectives: Year 1:** Use cultured skin models to identify oxidative photodamage markers post-blue light exposure. Isolate epidermal and dermal cells to assess ROS decay and damage correlation.**Year 2:** Design and fabricate electrospun textiles incorporating bioactive agents and light modulators. Characterise optical (400–470 nm), mechanical, and biological performance. Develop wearable prototypes and conduct pilot wearability studies.**Year 3:** Conduct a human volunteer study (+/– textile). Use biopsies, antibody labelling, and mRNA/protein assays to investigate UPE-mediated dermal remodelling.**Year 4:** Thesis writing, publication preparation, and conference presentation (e.g., International Union of Photobiology).
One representative publication from each co-supervisor: Gacesa R, Lawrence KP, Georgakopoulos ND, Yabe K, Dunlap WC, Barlow DJ, Wells G, Young AR, **Long PF.** The mycosporine-like amino acids porphyra-334 and shinorine are antioxidants and direct antagonists of Keap1-Nrf2 binding. Biochimie. 2018 Nov;154:35-44. doi: 10.1016/j.biochi.2018.07.020. Lina Wu, Driton Vllasaliu, Qi Cui, and **Bahijja Tolulope Raimi-Abraham** In Situ Self-Assembling Liver Spheroids with Synthetic Nanoscaffolds for Preclinical Drug Screening Applications ACS Applied Materials & Interfaces 2024 16 (20), 25610-25621 DOI: 10.1021/acsami.3c17384

Project L

Title
Exploring the 4D Nexus Between RNA-Binding Proteins and RNA

Project Details
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Project description: The heterogeneous nuclear ribonucleoprotein A1 (hnRNPA1) is a prototypical RNA-binding protein (RBP)involved in diverse aspects of nucleic acid metabolism. We and others have shown that hnRNPA1 enables cancer cells to reprogram mRNA translation initiation to adapt to severe microenvironmental stress. Under such conditions, hnRNPA1 binds to Internal Ribosomal Entry Sites (IRESs)—cis-regulatory sequences in mRNAs encoding pro-survival proteins—and enhances their translation. Despite its central role in cell fate decisions, the molecular basis of IRES regulation remains unknown. We hypothesise that stress-induced conformational changes in hnRNPA1 "switch on" its IRES-binding activity. This project aims to define the molecular mechanism by which hnRNPA1 recognises IRES elements, thereby advancing our understanding of how RBPs regulate IRES-mediated translation during stress responses. **Years 1–2 – Defining RNA structures and functional modules** The student will determine structures of hnRNPA1 bound to key IRESs (c-Myc, XIAP, Bcl-xL, Cyclin D1) using SEC-SAXS and assess its ability to unwind RNA secondary structures using atomic force microscopy (AFM). Long RNAs (>300 nt) will be generated by in vitro transcription at our BBSRC-funded RNA-factory hub. The student will gain hands-on training in large scale RNA synthesis, purification and sample preparation. **Years 2–3 – Identifying RNA specificity rules** The student will map sequence and structural RNA motifs within selected IRESs recognised by hnRNPA1. Binding affinities will be measured using high-throughput Spectral Shift (Dianthus, NanoTemper) and validated through reciprocal NMR titrations with identified RNA motifs. **Years 3–4 – Establishing a molecular model of RNA recognition** Experimental data will guide computational energy landscape analyses and molecular dynamics (MD) simulations to elucidate hnRNPA1–RNA structural dynamics, including the impact of post-translational modifications. Integration of SAXS, NMR, and computational data will yield a general molecular model of hnRNPA1 sequence- and structure-specific RNA recognition. This project will provide training in molecular biology, biophysics (NMR, SAXS, AFM), and advanced computational modelling, equipping the student with a broad, interdisciplinary skill set.
One representative publication from each co-supervisor: • S Chrysostomou*, R Roy*, Prischi, F.*, et al. (2021) Repurposed floxacins targeting RSK4 prevent chemoresistance and metastasis in lung and bladder cancer. Science translational medicine. 13 (602), eaba4627, doi: 10.1126/scitranslmed.aba4627 • Röder, K., Barker, A. M., Whitehouse, A., Pasquali S. (2022) Investigating the structural changes due to adenosine methylation of the Kaposi's sarcoma-associated herpes virus ORF50 transcript. PLoS Comput. Biol. 18(5): e1010150, doi: 10.1371/journal.pcbi.1010150

Project M

Title
Project title: Myosin-based regulation of contractility in slow and fast fibres of human skeletal muscle

Project Details
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Co-Supervisor 1B: Stephen Harridge School BMBS KCL/KHP email s.harridge@kcl.ac.uk KCL/KHP Website: https://www.kcl.ac.uk/people/stephen-harridge
Project description: Skeletal muscle weakness is central to many myopathies and wasting disorders, yet effective therapies remain scarce due to gaps in understanding the molecular regulation of contractility. Human skeletal muscle comprises fast-contracting type-2A and slow-contracting type-1 fibres, which differ in myosin isoforms and regulatory proteins. Traditionally, contraction has been explained by calcium-driven activation of actin thin filaments. However, recent studies support a dual-filament paradigm, where myosin-containing thick filaments also contribute via calcium-independent regulation. These mechanisms are established in animal fast fibres but remain unexplored in slow fibres and largely unknown in human muscle. Early evidence suggests human fibre types possess distinct thick-filament regulatory mechanisms, offering potential for fibre-specific therapies.This PhD project will combine molecular biology and in situ biophysics to investigate thick-filament regulation in human type-1 and type-2A fibres. Approaches will include purification and fluorescent labelling of human thin and thick filament proteins, proteomics of single fibres from healthy donor biopsies, muscle mechanics, and fluorescence microscopy. The student will develop expertise in experimental design, quantitative analysis, and molecular interpretation of biophysical data, alongside transferable skills in scientific writing and communication. Objectives: **Year 1:** Expression, purification, and labelling of fast and slow human isoforms of myosin regulatory light chain and troponin C; training in single-fibre isolation and fibre-typing.**Year 2:** Investigation of myosin motor and troponin regulation in human fibres using the probes generated in Year 1 and advanced microscopy and biophysical assays.**Year 3:** Proteomics on single human muscle fibres and analysis of post-translational modifications of myosin and regulatory proteins in type-1 and type-2 fibres.**Year 4:** Evaluation of novel small-molecule myosin modulators on the myosin-based regulation of contractility in type-1 and type-2 fibres. By revealing mechanisms of thick-filament regulation in human muscle, this project will advance fundamental knowledge and inform rational development of myosin-targeted therapies to alleviate muscle weakness in disease and ageing.
One representative publication from each co-supervisor: Brunello, E., Marcucci, L., Irving, M., & Fusi, L. (2023). Activation of skeletal muscle is controlled by a dual-filament mechano-sensing mechanism. Proc Natl Acad Sci U S A, 120(22), e2302837120. doi:10.1073/pnas.2302837120 12Kalakoutis M, Pollock RD, Lazarus NR, Atkinson RA, George M, Berber O, Woledge RC, Ochala J & Harridge SDR (2023) Revisiting specific force loss in human permeabilized single skeletal muscle fibers obtained from older individuals. American Journal of Physiology - Cell Physiology 325(1):C172-C185. doi: 10.1152/ajpcell.00525.2022.

Project N

Title
Dysregulation of cellular gene expression by influenza A virus infection induces the inflammatory response

Project Details	
Co-Supervisor 1A:	Chad Swanson
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Project description: Viral infection alters the molecular landscape of a cell through large scale changes in the transcriptome. Influenza A virus (IAV) infection affects host cell gene expression through multiple mechanisms including changes in epigenetic regulation and post-transcriptional RNA processing pathways. IAV-infection leads to cytoplasmic accumulation of RNA that is usually not expressed or should be retained in the nucleus including mRNAs with retained introns or extended 3' UTRs, transposable element RNA and natural antisense transcripts. These RNAs have the propensity to form double stranded RNA (dsRNA) that activates innate immune pattern recognition receptors (PRRs), leading to inflammation and cell death. However, as transposable elements and RNAs with processing defects are difficult to characterise with traditional short-read sequencing methods, their contribution to the inflammatory response to IAV infection remains ill-defined.

To understand how influenza A virus infection changes the cellular transcriptome and how these transcripts interact with PRRs, this project will employ long-read sequencing (Oxford Nanopore Technologies) that can characterise repeat or unconventional transcripts. Year 1: To determine how IAV infection alters the cellular transcriptome present in the cytoplasm, viral infection will be combined with subcellular fractionation and long read sequencing. Computational tools will focus on expression of transcripts containing transposable element sequences, natural antisense transcripts and other transcripts with the potential to form dsRNA. Year 2: IAV with mutations in specific viral proteins will be used to test which viral proteins are required to induce specific changes in cellular transcripts that form dsRNA. Year 3: Cellular RNA bound by cytoplasmic dsRNA PRRs such as MDA5, PKR and OAS3 will be identified and characterised to determine how specific characteristics of the RNA have led to dsRNA formation. The effect of this RNA on innate immune signalling will be analysed by qRT-PCR, viral replication kinetics and cell death assays.

One representative publication from each co-supervisor:

Supervisor 1A Lista MJ, Ficarelli M, Wilson H, Kmiec D, Youle RL, Wanford J, Winstone H, Odendall C, Taylor IA, Neil SJD, Swanson CM. (2023) A Nuclear Export Signal in KHNYN Required for Its Antiviral Activity Evolved as ZAP Emerged in Tetrapods. J Virol. 97:e0087222.

Supervisor 1B Mischo HE[*,+], Chun Y, Harlen KM, Smalec BM, Dhir S, Churchman LS, Buratowski S*, (2018) "Cell-cycle modulation of transcription termination factor Sen1", Mol Cell, **70**:312-326 ([*] joint corresponding authors, [+] lead author)

Project P

Title
DNA repeat instability as a driver of bacterial infection heterogeneity

Project Details
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Co-Supervisor 1B: Joseph Wanford School: School of Immunology and Microbial Sciences KCL/KHP email: joseph.wanford@kcl.ac.uk KCL/KHP Website: https://www.kcl.ac.uk/people/joseph-wanford
Project description: Scientific basis of the project: Hypervirulent *Klebsiella pneumoniae* (HV*Kp*) *represents a growing antimicrobial resistance (AMR) threat and an emerging global health concern*. A key virulence factor is the polysaccharide capsule, a protective layer which enables survival in the bloodstream but impedes epithelial colonisation. We have recently discovered that simple sequence DNA repeats (SSRs) within the *rmp* locus (a gene involved in capsule biosynthesis) drive a high-frequency, reversible ON/OFF switching of capsule expression, termed phase variation. This process generates phenotypic heterogeneity enabling the pathogen to adapt to contrasting host environments. Importantly, the molecular determinants of this switch, and whether they promote adaptation to the host during infection remains unknown.This project will leverage cutting edge molecular biology and 'omics'-based measurements of mutation rate to define the molecular basis of phase variation, before employing our newly established HV*Kp* zebrafish infection model, to conduct real-time analysis of host–pathogen interactions at single-cell resolution *in vivo*. Overall, the successful candidate will define how localised hypermutation at simple sequence repeats alters HVKp colonisation and immune evasion in the host. **Techniques and skills:** Training will encompass molecular microbiology and genetic engineering in bacteria (for example construction of recombinant bacteria with altered DNA repeat lengths, next-generation sequencing of bacterial genomes), infection modelling in zebrafish larvae (for example survival assays, bacterial burden quantifications, recruitment of immune cells, infection dissemination quantification, analysis of host gene expression by qRT-PCR) and advanced live imaging (for example confocal microscopy). **Overarching Objectives: Year 1**: Define the molecular basis of phase variation rate using reporters and next-generation sequencing. **Year 2**: Isolate and construct phase variants for phenotypic characterisation. **Year 3**: Define the role of the host in mutation rate and selection for different phase variants. **Year 4**: Writing of publications and thesis submission.
One representative publication from each co-supervisor: **Torraca, V.,** Brokatzky, D., Miles, S. L., Chong, C. E., De Silva, P. M., Baker, S., Jenkins, C., Holt, K. E., Baker, K. S., & Mostowy, S. (2023). *Shigella* serotypes associated with carriage in humans establish persistent infection in zebrafish. *The Journal of Infectious Diseases*, 228(8), 1108-1118. https://doi.org/10.1093/infdis/jiad326 Stawarska, O., Nair, A. R., Man, A. W. Y., Green, L. R., Gazioglu, O., Flandi, F., ... & **Wanford, J. J.** (2025). Simple sequence repeats mediate phase variation of the mucoid phenotype in hypervirulent *Klebsiella pneumoniae*. *bioRxiv*, 2025.09.12.675794. https://doi.org/10.1101/2025.09.12.675794

Project R

Title
Decoding epicardial fat–driven cardiac dysfunction using hiPSC-derived 3D tissues: a translational approach to heart failure.

Project Details
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Project description: EAT, derived from epicardial progenitors via epicardial-to-fat transition (EFT), plays a pathogenic role in obesity-associated heart failure (HF) by disrupting myocardial structure and electrophysiology, contributing to diastolic dysfunction and arrhythmogenesis. Rodent models exhibit species-specific limitations in EFT, hindering mechanistic exploration. This PhD project circumvents these constraints by leveraging hiPSC-derived three-dimensional EHTs to model epicardial lineage transitions and dissect EAT–myocardium interactions at cellular and molecular resolution. This project enables translational identification of therapeutic targets and offers comprehensive interdisciplinary training across experimental, analytical, and professional domains. Trainees will gain expertise in s**tem cell and tissue engineering**, including hiPSC culture, directed differentiation into cardiac and epicardial lineages, and generation and functional assessment of 3D EHTs. **Molecular and cellular biology** techniques such as immunostaining, confocal microscopy, flow cytometry, RNA extraction, qPCR, and Western blotting will be employed alongside **omics and bioinformatics** approaches, including mining single-cell RNA-sequencing datasets and perform metabolomics analysis. The project will also facilitate the development of essential soft skills for research, including hypothesis generation, experimental design, research ethics, data reproducibility, and presentation skills. Overarching objectives for this PhD project include: **Year 1**: master technical foundations and establishing the EHT model; characterise baseline architecture, contractility, and electrophysiology. **Year 2**: explore EAT–myocardium crosstalk through co-culture systems and omics profiling to identify key inflammatory, metabolic, and structural pathways. **Year 3**: validate candidate mechanisms using perturbation assays and integrate findings with patient-derived datasets or clinical biomarkers. **Year 4**: synthesise results, finalise data analysis, and support dissemination through thesis writing, manuscript submission, and conference presentations. This project provides a unique opportunity to bridge cellular/molecular biology, cardiovascular pathology, and translational research, equipping the candidate with a robust skillset to address complex questions in cardiac disease and regenerative medicine. Expertise and training needed for the project are in-place *via* the co-supervisory team.
One representative publication from each co-supervisor: Chapman, B., Klaourakis, K., de Villiers, C., Gunadasa-Rohling, M., Cosma, M.-A., Copper, S., Mohan, K., Weinberger, M., Carr, C., Greaves, D., Jackson, D., Pezzolla, D., Choudhury, R., **Vieira, J. M.***, & Riley, P*. (2025). *Nature Cardiovascular Research*. https://doi.org/10.1038/s44161-025-00711-4 **Zoccarato, A*.**, Smyrnias, I., Reumiller, C. M., Hafstad, A. D., Chong, M., Richards, D. A., Santos, C. X. C., Visnagri, A., Verma, S., Bromage, D. I., Zhang, M., Zhang, X., Sawyer, G., Thompson, R., & Shah, A. M*. (2025). *Cardiovascular research*, *121*(2), 339–352. https://doi.org/10.1093/cvr/cvae250