



THE UNIVERSITY
OF QUEENSLAND
AUSTRALIA

School of
Biomedical Sciences

HONOURS APPLICATION AND ENROLMENT GUIDE

2019 - 2020

Bachelor of Science
(Hons) Biomedical
Sciences

Bachelor of Biomedical
Science (Hons)

Bachelor of Advanced
Science (Hons)



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This handbook is intended to give information on applying and enrolling in the Honours program in the School of Biomedical Sciences. This information is advisory and does not in any way supersede The University of Queensland Calendar & electronic course profile.

ENROLMENT CHECKLIST

1. Check if you meet the entry requirements. Bachelor of Science graduates can check their eligibility to enrol in Honours by reading the program rules and requirements [here](#). Three year Bachelor of Biomedical Science graduates can check their eligibility to enrol in Honours by reading the program rules and requirements [here](#). Students currently in the four year Bachelor of Biomedical Science (Honours) program can check their eligibility to enrol in the final Honours year by reading the program rules and requirements [here](#). Students currently in the four year Bachelor of Advanced Science (Honours) program can check their eligibility to enrol in the final Honours year by reading the program rules and requirements [here](#).
2. Choose a research area you would like to work in and discuss potential projects with a range of possible supervisors. Research profiles of our academics can be found on the SBMS web site (<https://biomedical-sciences.uq.edu.au/research/themes>) and UQ websites. You can also find a list of Honours projects at: <https://biomedical-sciences.uq.edu.au/study/honours> and at the end of this booklet.
3. Select supervisor(s) and project and ensure your supervisor has agreed to enrolment. It is required that at least one of the primary or co-supervisors be an SBMS academic or SBMS affiliate. In addition, SCMB academics can act as supervisor or co-supervisor for students enrolled in BIOM6501/2.
4. Return completed School application form to the SBMS Honours administrator (Email: sbms@enquire.uq.edu.au or Level 1, Sir William MacGregor Building (#64)).

Important: All BSc Honours applicants, one year BBiomedSc applicants and new to UQ students must also complete the [UQ Online application form](#).

Deadline: 17th June 2019 for commencement in Semester 2 2019
6th December 2019 for commencement in Semester 1 2020

5. We check that you meet the GPA requirements and application details.
 6. You will receive notification advising which courses you need to add in mySI-net. You need to enrol in these courses before the census date (census dates are indicated on the University calendars). If awaiting results, students are not enrolled until the results are released.
 7. Official starting date for Honours (**can vary by a week**):
First week in February for commencement in Semester 1
Third week in July for commencement in Semester 2
 8. The Honours calendar of events and deadlines will be finalised before commencement of your Honours program. It will be emailed to you as well as posted on the SBMS Honours website (<https://biomedical-sciences.uq.edu.au/study/honours>) and the course Blackboard site.
- *Steps 1-3 should be completed well before the proposed commencement date*

GENERAL INFORMATION

After completing your Bachelor of Science or equivalent degree, Honours will be the most intensive – and for many the first – contact with original research. Through Honours you will experience the different facets of research: the excitement of discovering something new, the satisfaction that comes with being an expert in your chosen field as well as frustrations, problem-solving and communication of your findings. You will be part of a research team, learning from more experienced researchers around you, such as your supervisors and other members of the laboratory.

Whether you consider Honours a stepping stone to a Masters or PhD and onto a career as a researcher, or a vital research experience giving you credibility in science and research-associated careers, you will find the course will add significantly to your training as a Science graduate.

Honours with SBMS

The Honours year with SBMS is a hands-on experience in research and associated skills. This includes the development of technical skills in scientific methodology as well as intellectual skills in experimental design, critical appraisal of scientific literature and assessment of the impact of your original data on current knowledge. Research projects are selected by negotiation between you and supervisor(s) and are reviewed by the SBMS Honours Committee. We encourage you to seek contact with staff members to discuss likely research projects early in your Level 3 studies.

In order to find a supervisor and suitable project we advise you identify research areas you are interested in and approach staff working in these areas. Our staff at the School of Biomedical Sciences research and teach in a wide range of areas from the genomic level through to the structure and function of intact humans and other organisms. We have a strong focus on molecular, cellular and structural biology. Students who want to study physiology will focus their research on how organ systems, tissues, cells and molecules function together; those who concentrate on anatomical studies will investigate how structures are created and how they function whilst pharmacology/toxicology students will research how drugs and toxins modify or affect biological functions.

The School and its associated centres and companies are heavily involved in cutting edge biomedical research and most of our academics have an active research program with projects available for Honours students. More details can be found at <https://biomedical-sciences.uq.edu.au/research/themes>. The research areas of SBMS staff members are grouped under the following themes:

- Cell Architecture
- Chronic Diseases: cancer, cardiovascular disease, diabetes
- Drug Design and Development
- Functional Morphology
- Innovation in Biomedical Education
- Musculoskeletal and Motor Control
- Neurobiology and Brain Function
- Receptors and Signalling
- Reproduction

Collaborative projects may also be available with several University Institutes including the Queensland Brain Institute (QBI), the Institute for Molecular Bioscience (IMB), The Australian Institute for Bioengineering and Nanotechnology (AIBN), the Diamantina Institute, the UQ Centre for Clinical

Research and The Mater Research Institute More information about these institutes and centres can be found on their respective UQ websites.

Bachelor of Biotechnology (Drug Design and Development)

Laboratories within SBMS frequently host students who are engaged in the Biotechnology degree program. Details regarding this program and its requirements can be obtained from the following website: <http://scmb.uq.edu.au/biotech>

Information for Students of other Faculties

The School is within the Faculty of Medicine and most of our Honours students are enrolled in either Bachelor of Science (Honours), Bachelor of Biomedical Science (Honours) or Bachelor of Advanced Science (Honours). However, there are avenues for students enrolled in Medicine, Veterinary Science, Dentistry and other professional courses to undertake research studies with us. Students enrolled in professional courses may obtain more detailed information from their relevant faculty and discuss their interest with the Chair of the Honours committee.

The main focus of this handbook is for Honours in Biomedical Science. Students wishing to pursue similar degrees with us should also contact the faculty in which they are enrolled.

SBMS-Oxford Honours Scholarships

We have established a program with Oxford University (UK), designed to promote learning in the form of special lectures and Honours scholarships for students in the biomedical sciences. For details on applying for one of these competitive scholarships please see the Honours website: (<https://biomedical-sciences.uq.edu.au/study/honours>)

SBMS-Ludwig Maximilians University Honours Scholarships

We have established a program with Ludwig Maximilians University (LMU) in Munich, Germany that gives students the opportunity to undertake their Honours research project at this world-leading institution. For details on applying for one of these competitive scholarships please see the Honours website: (<https://biomedical-sciences.uq.edu.au/study/honours>)

Careers

An Honours degree is the qualification most often required for employment in research positions and industry. Numerous career opportunities await students with backgrounds in biomedical science, where universities and research institutions are the major employers. Many students have opted to study biomedical sciences as a prelude to careers in professional disciplines such as medicine, dentistry and speech therapy. Increasingly an Honours degree is a minimum requirement for entry-level employment in industry.

Candidates who obtain Honours I or Honours IIA may proceed directly to studies for the degree of Doctor of Philosophy (PhD). An Honours IIB is the minimum requirement for entry to the degree of Master of Science (MSc). The path for students aspiring to careers as academics or research scientists is usually the PhD.

HONOURS ENROLMENT REQUIREMENTS

Entry Criteria

For entry into Honours, SBMS requires a satisfactory background in Level 2 and 3 relevant courses. For BSc and BBiomedSc graduates, the minimum requirement is a GPA of at least 4.5 in the “most relevant 8 units of third level (or advanced) study”. In addition, an overall GPA of 4 (minimum) for the BSc and BBiomedSc degree (or for the first three years of the BBiomedSc (Hons) and BAdvSc (Hons) degree) is required. In some circumstances, and subject to the approval of the Head of School and Executive Dean, BSc graduates, BBiomedSc graduates, BBiomedSc (Hons) students and BAdvSc (Hons) students who do not fulfil the above requirements may be permitted to enter the Honours program if they have a high GPA in other science courses and if their proposed project fits well with their background.

Commencement of Study

Studies may commence on the following dates: (slight variations are possible if there is any change in the UQ Academic Year).

1st Semester enrolments (can vary by a week)

Application Due: Friday 6th December 2019

Start Date: First week in February 2020 (check the eCP for confirmed date)

2nd Semester enrolments (can vary by a week)

Application Due: Monday 17th June 2019

Start Date: Third week in July 2019 (check the eCP for confirmed date)

PROGRAM DESCRIPTION

General Information

- The Honours program consists of a research project with associated research proposal, research report, seminars, journal clubs and evaluation of laboratory performance.
- It is required that at least one of the primary or co-supervisor be a SBMS academic or SBMS affiliate. In addition, SCMB academics can act as supervisor or co-supervisor for students enrolled in BIOM6501/2.
- It is very important for students and supervisors to be aware that the research report represents the bulk of the year's work and is therefore the primary indicator of the level of the student's research and communication skills.
- On receiving the application, the Honours committee will evaluate the candidate and the project descriptions. Any questions or concerns will be discussed with the supervisor or candidate before approval is given. Any subsequent major changes to the research project throughout the Honours year will require approval from the SBMS Honours Committee chairperson.

Assessment Items

- **Research Proposal:** Submitted as a document of 4000 words (maximum) outlining and justifying the proposed project and introducing the background literature.
- **Proposal Seminar:** Students will give a 10 minute oral presentation (with 5 minutes of questions) on the background and rationale for their study. This will include a statement of aims and hypotheses along with research methods to be used.
- **Research Report:** Submitted as a document of 7500 words describing and critically appraising the research work undertaken during the Honours year.
- **Seminar Diary:** Students will attend Honours student presentations and at least 12 seminars (these can be external to SBMS) given by academic/research staff or invited speakers.
- **Supervisor's Report:** Supervisors will provide a report based on the student's performance over the course of the Honours year.
- **Final Research Seminar:** This component includes the final seminar presented at the end of the year (15 minute talk & 10 minutes for questions).

Assessment Marking

- Two examiners are invited by the SBMS Honours Committee to assess the research proposal and research report. Their feedback will be made available to the students, although examiners have the option of remaining anonymous. If appropriate, examiners may be from another department or institution.

- Research proposal seminars will be examined by a supervisor of the students presenting within that session. Each supervisor will examine all speakers within that session. A suitable proxy can be recruited provided they have > 3 years post-PhD research experience.
- Final research seminars are examined by two members of the SBMS Honours Committee.
- Templates of marking sheets used by examiners for the assessment of items of work can be found in the learning resources section of the Blackboard site.
- Students will be informed of the grading of any item's assessment at the end of each semester. Students should direct any queries in relation to marks to the Honours Coordinators or Committee.
- Final results are recommended by the SBMS Honours Committee to the Head of School, who advises the Executive Dean. The award of various classes of Honours is also made by the Head of School and relevant Executive Dean.
- Criteria marking sheets for all Honours assessment items will be posted on Blackboard sites for the courses.

Assessment Summary BSc, BBiomedSc and BAdvSc Honours

Subject Code	Subject Title	Credit Unit
BIOM6191 or BIOM6192	Research Project in Biomedical Sciences	16
BIOM6501 or BIOM6502	Research Project in Biomedical Sciences	16

Assessment Task	Weighting
<i>Seminar</i> Research Proposal Seminar	5%
<i>Report</i> Research Proposal	10%*
<i>Diary</i> Seminar Diary	5%
<i>Report</i> Research Report	55%*, ^h
<i>Report</i> Supervisor's Report	5%
<i>Seminar</i> Final Research Seminar	20%

* submission of final version via Turnitin, ^h indicates a hurdle on the assessment task

HOW TO FIND AN HONOURS SUPERVISOR/PROJECT

- Decide what broad research field you are interested in
 - Also consider what techniques you would like to learn
- Find a supervisor in that field
 - Lecturer or Researcher
 - Are they actively publishing?
 - Do they have other students/staff (to provide you with extra support)?
- Contact your potential supervisor
 - Don't send a generic email
 - Be familiar with the Supervisor's work
 - Tell them why you want to do an Honours project in **their** lab
 - Contact them early – i.e. NOW!!
 - Meet with them in person – will they be supervising you day-to-day or will it be a post-doc? If it is the latter then ask to meet the post-doc.
- It is required that at least one of the primary or co-supervisor be a SBMS academic or SBMS affiliate. In addition, SCMB academics can act as supervisor or co-supervisor for students enrolled in BIOM6501/2.

WHERE DO I LOOK FOR A SUPERVISOR/PROJECT?

The following pages have information for specific Honours projects that have been submitted by potential supervisors. This is not an exhaustive list and you might also be able to find a project by contacting a researcher directly. Information and contact details for researchers can typically be found on School and Institute web sites, some of which are listed below:

School of Biomedical Science - <https://biomedical-sciences.uq.edu.au/research/themes>

School of Clinical Medicine - <https://medicine-program.uq.edu.au/school-of-clinical-medicine/research>

Diamantina Institute - <http://www.di.uq.edu.au/study/honours>

Mater Research Institute – <http://www.materresearch.org.au/students.aspx>

UQ Centre for Clinical Research - <https://clinical-research.centre.uq.edu.au/honours>

UQ Child Health Research Centre - <https://child-health-research.centre.uq.edu.au/study-chrc>

Queensland Brain Institute - <https://qbi.uq.edu.au/study/honours>

Institute for Molecular Biosciences - <http://postgraduate.imb.uq.edu.au/>

QIMR-Berghofer Medical Research Institute - http://www.qimrberghofer.edu.au/student_projects/



Prof Elizabeth Coulson

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Neurotrophin Biology Lab

My lab is currently focusing on the reasons why cholinergic neurons of the basal forebrain die in Alzheimer's disease, what contribution their loss makes to cognitive decline and whether manipulating neurotrophic signalling (NGF, BDNF, TrkA/B, p75) can protect or restore cognitive function, and what role the neurotrophins play in the normal function of these neurons. Possible projects include:

- Project 1: The role of neurotrophins in regulating the synaptic function of cholinergic basal forebrain neurons (mouse studies: slice electrophysiology, mouse surgery for *in vivo* genetic manipulation, mouse behaviour and potentially mouse MRI)
- Project 2: The role of cleavage of p75 neurotrophin receptor in neurodegeneration (cell biology and/or mouse studies)
- Project 3: Studying basal forebrain function in Alzheimer's disease and sleep apnea (cell signalling pathways OR human MRI and PET image analysis)
- Project 4: Developing optimised peptides for treating neurotrophic dysfunction in neurodegenerative disease (biochemistry)

Techniques you will learn in our group may include: histology, microscopy, cell biology, biochemistry, electrophysiology and potentially, advanced imaging analysis methods.



Dr Sean Millard

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Millard Lab – Molecular mechanisms for wiring the brain

The overall goal of the Millard lab is to understand how specificity is generated in the brain. This problem is best exemplified by considering that 100 trillion synapses are generated and maintained in the human brain using a toolkit of only 20,000 genes. We have been approaching this problem using molecular genetics in the fruit fly, *Drosophila melanogaster*. Most projects in the lab revolve around how a broadly expressed cell surface protein, called Down syndrome cell adhesion molecule 2 (Dscam2), is able to perform specific functions in different neurons. We are also interested in mechanisms of neurological disease, particularly those that involve changes in synaptic function.

Available Projects:

1. Mapping binding sites for the factor that regulates Dscam2 alternative splicing using CRISPR-Cas9 in *Drosophila*.
2. Validating sporadic Motor Neuron Disease candidate genes in the fruit fly.
3. Investigating whether the splicing factor, Rbfox1, represses Dscam2.10B isoform expression.
4. Investigating whether Dscam2 and Presenilin function in the same signaling pathway in motor neurons.
5. Assessing whether different cytoplasmic isoforms of Dscam2 confer dendritic localization.

Techniques: Molecular biology, genetics, immunohistochemistry, microscopy

**A/Prof Peter Noakes**

School of Biomedical Sciences
Neuroscience

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Noakes Lab / Cell and Molecular Biology of the Neuromotor System

Peter and his collaborators are investigating the cell and molecular mechanisms that underlie the development and breakdown of the neuro-motor system. This includes formation and development of input synapses onto motor neurons and their output synapses onto muscle. As a consequence of these interests, they are also investigating the cell and molecular mechanisms of several neuromuscular diseases, such as motor neuron disease, muscular dystrophies, and associated neuromotor disorders.

Project Description 1: A loss of Agrin-LRP4 Musk trans-synaptic signalling leads to a loss of neuromuscular connections in patients with Motor neuron disease. Muscle stem cells grown from Motor Neuron Disease patients appear not to effectively respond to Agrin to make post-synaptic specializations (the site that the motor neuron connects with muscle to make it contract). The molecular basis of this will be investigated using biochemical (Western Blot) and Molecular methods (PCR, RNA Seq).

Project Description 2: Perineuronal nets regulate the numbers of pre-synaptic input onto motor neurons: their loss leads to neuronal hyperexcitability neuronal dysfunction. Perineuronal nets are extracellular matrices that cover the excitable neurons and their proximal dendrites. They offer neuronal protection and act to regulate the balance of excitatory and inhibitory inputs that a post-synaptic neuron receives. Our idea is that malformation or breakdown of these nets changes this balance of inputs onto motor neurons to render them hypoexcitable. This idea will be explored at the histological and electrophysiological levels in mice models of motor neuron disease or mice that have had their inhibitory synaptic inputs perturbed.

Project Description 3: Making human skeletal muscle from skin fibroblasts: a new way to help model severe neuromuscular disorders. Many neuromuscular diseases are characterized by severe muscle wastage making it unethical to obtain muscle samples to grow and test under controlled conditions in the laboratory. One way around this is to make multinucleated muscle cells from skin fibroblasts (i.e. from a skin scraping). This project will generate and characterize such cells. It will involve tissue culture, immunostaining, and bioassays (functional and molecular).

Project Description 4: Human neuromuscular synapses cell and molecular characterization from patients with motor neuron disease and healthy donors: high resolution microscopy and image analyses (2D and 3D rendering of synapses).

Project Description 5: The final number of motor neurons needed to connect with muscle is determined in part by the synaptic inputs onto motor neurons - independent of innervation feedback from muscle. This project will use single knockout and double knockout mutant mice, that will have both input and output motor neuron synapses lost (double knockout), compared to mice with a loss of only input synapses or only output neuromuscular synapses (single knockouts). It will involve histology, immunostaining and confocal microscopy.

**A/Prof Michael Piper**

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Neuroscience

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Neural Stem Cells in Development and Disease

We use the cortex, cerebellum and spinal cord of the developing and adult mouse as model systems to elucidate the biology of neural stem cells within the brain. Ultimately, we hope to define the genes that drive the differentiation of neural progenitor cells into either neurons or glia, work that will provide insights into neurodevelopmental disorders, ageing and cancer. Dr. Piper currently has funding from the ARC (Discovery Project grant; 2018-2020) to study the molecular determinants governing how neural stem cells proliferate sufficiently to generate a brain of the correct size. In addition to this, he holds a CIB grant from the NHMRC in association with A/Prof Helen Cooper (QBI) to study how abnormal ependymal cell development contributes to hydrocephalus, as well as seed funding from the Australian Skin and Skin Cancer Research Centre to study the malignant invasion of certain types of brain cancer.

Finally, Dr Piper is studying how abnormal neural stem cell differentiation may contribute to autism, work that is funded by the Simons Foundation Autism Research Initiative (USA). Collectively, we envisage that this research will provide important insights into both the control of neural stem cell differentiation within the developing brain, and the consequences of aberrant neural stem cell biology with regards to disease progression.

Project 1

How neural stem cell biology contributes to macrocephaly

Project 2

Understanding the genetic determinants driving cerebellar development

Project 3

NFIB and melanoma metastasis

Techniques you will learn in our group may include: Immunohistochemistry, Microscopy, qPCR, In situ hybridisation, Histology, Behavioural analysis, Diffusion tensor magnetic imaging



Dr Jana Vukovic

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Neuroscience, Cancer
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Laboratory for Neuroimmunology and Cognition

Targeting microglia to alleviate side-effects of leukaemia treatments on cognition

Haematological malignancies (i.e. leukaemia) represent the fifth most commonly occurring cancers globally and are the second leading cause of cancer death. Hematopoietic stem cell transplantation (SCT) is the most effective curative therapy, and hence the therapy of choice, for the majority of these cancers of bone marrow origin. The curative property of SCT lies in the graft-versus-leukemia effect which is required for ablation of residual cancer burden. This process is absolutely dependent on donor T cells contained within the graft, however, these T cells are also the primary mediators of graft versus host disease (GVHD), a life-threatening complication that significantly limits the success of SCT therapy. Importantly, GVHD has been shown to significantly impact neurocognitive function, which after SCT, is a serious cause of morbidity. Symptoms include memory impairment, depression and difficulty in performing multiple tasks simultaneously, all of which adversely affect the patient's quality of life. Although these neurocognitive side effects / complications are recognised, very few studies have been undertaken to date to identify how GVHD affects the brain.

This project will employ established models and methodology to examine the effects of GVHD on neuronal connectivity and cognitive behaviour (in particular hippocampal function), and also the contribution of microglia, the resident macrophage-like cells of the brain, to these processes. This project is highly novel and will generate important data into the immune mechanisms driving GVHD of the brain.

This is a challenging project, suitable for high achieving and motivated Honours or PhD students. Successful applicants will have a unique opportunity to undertake research in leading labs across two fields: Neuroimmunology and Cognition (Dr. Jana Vukovic, SBMS) and Transplant Immunology (A/Prof Kelli MacDonald, QIMR Berghofer).

Techniques you will learn in our group may include: Small animal handling and experimentation (incl. behaviour), transplant models, flow cytometry, immunostaining, imaging and quantification, quantitative PCR, etc.



A/Prof Trent Woodruff

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Pharmacology, Neuroscience

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Neuroinflammation Laboratory

Our laboratory focuses on the role of inflammation and the immune system in neurodegeneration, and the identification of novel drug targets to slow progressive brain disease in humans. Our projects encompass the identification and development of novel drugs, to the testing of novel drugs in animal models of neurodegenerative disease, and the exploration of novel inflammatory pathways in diseased patients. In doing so, we hope to accelerate the clinical translation of our findings and novel drugs candidates to clinical trials.

Project 1

Identification of new immune system contributors to neurodegenerative disease progression

Project 2

Novel therapeutic strategies to treat motor neuron disease

Project 3

Pharmacological characterisation of novel anti-inflammatory compounds in cell-based models

Project 4 (with Dr Nemat Khan)

Development of a novel treatment strategy for Multiple Sclerosis by targeting the complement system.

Project 5 (with Prof. Maree Smith and Dr Nemat Khan)

The role of complement system (C5a-C5aR signalling) in development of peripheral neuropathic pain

Techniques you will learn in our group may include: pharmacological profiling (cellular signalling assays), animal behaviour and drug testing (cognitive and motor testing), immune cell profiling (flow cytometry), neuropathology (immunohistochemistry) and molecular analysis (qPCR, western blotting).

**Dr Richard Clark**

School of Biomedical Sciences
Pharmacology

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Peptide Chemical Biology Lab

We are interested in studying the role of naturally occurring bioactive peptides in a broad range of human diseases. Peptides have a diverse range of functions in human biology including acting as hormones, neuro-regulators and in the protection against pathogens. Our work in this area is focused on understanding the molecular mechanism that these peptide use to elicit a biological response with the hope of using this knowledge to develop new drug leads.

Project Description: Discovery and development of peptides for the treatment of inflammation and infection

This project involves the discovery of novel peptides from nature that modulate the immune response to prevent inflammation or infection by pathogens. Using peptide engineering we can modify these leads to understand how they mechanism of action, improve their drug-like properties, and develop therapies that are targeted to one location in the body.

Techniques you will learn in our group may include: Solid phase peptide synthesis, High Performance Liquid Chromatography (HPLC), NMR Spectroscopy, Mass Spectrometry, Cell culture and cell-based assays, Compound stability assays.



Dr Lachlan Rash

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Pharmacology & Neuroscience

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Ion Channel Pharmacology

Given the crucial role of ion channels in normal physiology, many venomous predators (spiders, scorpions, sea anemones, wasps etc.) have evolved libraries of molecules that potently interfere with ion channel function in order to rapidly paralyse prey. The Ion Channel Pharmacology lab uses animal venoms as well as man-made drugs to help understand the function and modulation of various ion channels in health and disease. We have a particular focus on acid-sensing ion channels and voltage-gated sodium channels and their role in pain, inflammation and neurological disorders such as stroke and spinal cord injury.

The broad research fields of the group cover pharmacology, physiology, biochemistry and toxinology.

Project 1: Characterisation of novel voltage-gated sodium channel (Nav) modulators from spider venoms and their use to gain structural insights into Nav channels.

Project 2: Venom peptides and herbal medicines as tools to study acid-sensing ion channels structure and molecular function.

Project 3: Biological and pathological roles of acid-sensing ion channel. Pathological conditions of interest can include neurogenic inflammation, ischemia, tumors, neurodegeneration.

Techniques you will learn in our group may include: electrophysiology (*Xenopus* oocytes), high-performance liquid chromatography, mass spectrometry, molecular biology, peptide and protein production & mutagenesis, cell culture/assays, organ bath assays.

**Dr Dominic Ng**

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Cell Biology/Signalling

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Kinase Biology and Intracellular Signal Transduction Pathways

Our research is focused on signal transduction pathways that are required for normal human development and that are dysfunctional in disease states including cardiovascular, neurological and oncological conditions. In particular, we are interested in the regulation and function of protein kinases (enzymes that mediate phosphorylation reactions) that control key cellular process including proliferation, differentiation and cell death. The human genome encodes over 500 kinases and pseudokinases (kinome) but the vast majority of these are poorly characterized. Honours projects in the Ng lab will contribute to fundamental research to understand how the substrate selectivity, subcellular distribution and functional pleiotrophy of kinases are determined and the effect of targeting kinases for therapy in animal and cellular models of human disease.

Project Description 1: Centrosome mechanisms in the developing heart

Centrosomes are small organelles that co-ordinate kinase signalling and cytoskeletal organization in mammalian cells. There is increasing appreciation for the contribution of centrosomes in cell fate specification and the structural reorganization of the centrosome in many somatic cell types including myocytes that coincide with their differentiation. In this project, you will investigate the role of dynamic centrosome reorganization in myocyte differentiation, maturation and terminal differentiation. A secondary objective would be to define the contribution of centrosomal-associated kinases in myocyte differentiation. This study will provide insights into the molecular control of the postmitotic state of myocytes and address a long-standing question of whether these processes can be manipulated for cellular regeneration.

Project Description 2: Kinase mechanism and function in stress granule formation.

The inside of a cell is a crowded place with ~42 million proteins co-existing within a single cell volume (typically ~100 μm^3). Recent studies have demonstrated that membrane-less compartments such as centrosomes, P bodies and stress granules offer a level of intracellular organisation that was previously unappreciated. These structures are formed through liquid-liquid phase separation of proteins into droplets in order to co-ordinate specific signalling activities. There is also strong evidence that link defects in these higher ordered protein states to the toxic aggregation of proteins associated with neurodegenerative conditions such as Alzheimers and motor neuron disease. This project will investigate how stress-induced kinase signalling is co-ordinated within stress granules and how this ultimately defines cell survival and stress responses.

Project Description 3: How do microcephaly proteins control brain growth?

Microcephaly is a serious neuropathological condition that encompasses a spectrum of reduced brain size and cortical malformations that significantly impact learning, memory and behaviour. A number of

genetic mutations have been identified as causative of heritable or primary microcephaly (MCPH). Although these mutations are rare, they provide novel insights into non-redundant molecular processes in neural stem and progenitors cells that are required for growth and normal development of the human brain. This project will study MCPH proteins that integrate microtubule cytoskeleton, cell cycle regulation and cell stress responses within neural stem cells.

Techniques you will learn in our group may include:

- live and fixed cell fluorescence microscopy
 - immunohistochemistry
 - immunoblotting
 - proteomic techniques
 - mammalian cell culture including primary isolation of myocytes and neurons
 - viral transduction of mammalian cells
 - recombinant protein expression/purification
 - flow cytometry
 - reverse genetic approaches (siRNA, shRNA, CRISPR-Cas9)
 - transmission electron microscopy
 - sub-cellular and cytoskeletal purification
 - in vitro enzymatic assays
 - in vivo disease models
-



A/Prof Rohan Teasdale

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Cell Biology, Cell Architecture, Chronic Diseases

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The highly co-ordinated movement of the thousands of distinct membrane proteins between the cell surface and intracellular compartments is a critical factor in health and disease, controlling the organisation of cells in tissues and communication between cells and their environment. The success of this process depends on the regulated sorting and trafficking of proteins within the highly dynamic intracellular endosomal compartments. Protein trafficking dysfunction are important drivers of neurodegenerative disease, cancer and metabolic pathologies. An understanding of how endosomal traffic is regulated is critical for providing insights into is critical for providing insights into disease and the numerous physiological processes associated with these intracellular organelles.

Project 1 - Retromer's role in neurodegeneration. Multiple neurodegenerative diseases are caused by defects in the degradation of biological materials. This degradation is dependent on the trafficking of proteolytic enzymes to the endosome/lysosome system within cells. Recently, we have defined the underlying molecular causes of Parkinson's disease for familial mutations identified in Retromer, a central endosome protein machine responsible for the sorting of membrane cargo to a range of destinations. We have projects focused on determining the rigorous mechanistic understanding that result in the changes in cellular homeostasis that occur during disease induction and progression that impair the cell's ability to degrade protein aggregates like that associated with Parkinson's disease.

Project 2 - Retromer – A master regulator of endosome protein trafficking. Fidelity of transport through the endosomal system requires mechanisms that precisely sort cargoes for delivery to a range of different destinations. This is achieved by cargo engaging specific sorting machinery that is responsible for their accumulation into tubules that then undergo scission to generate endosome-transport carriers (ETCs). Once formed, these carrier vesicles engage the machinery at the target membrane, resulting in cargo delivery to the specific membrane, e.g. plasma membrane. Retromer has a central role in this process through interaction with associated proteins that determines the properties of the individual ETCs formed. We have projects available to investigate the contribution each of the variant Retromer complexes has on the formation of the distinct ETCs types and defining the cargo transported by these vesicles.

Techniques you will learn in our group may include: Cell Biology, Tissue Culture, Microscopy (super-resolution; live cell imaging, confocal etc), Cell based trafficking assays, Molecular Biology and Biochemical techniques.

**Dr Julia Pagan**

Lecturer, School of Biomedical Sciences
Cells and Genes, Cancer

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Regulation of cell growth and survival by protein degradation pathways

Although a great deal is known about the regulation of gene transcription and translation, much less is known about the processes targeting individual proteins for degradation, which are equally important for cell regulation. Indeed, protein degradation plays a critical role in the regulation of virtually all cellular processes, and can be misregulated in many diseases, including cancers and neurodegenerative diseases. My lab is interested in understanding how ubiquitin ligases (the enzymes that provide the specificity of protein degradation) recognize their target substrates. We focus on understanding the role of protein degradation in cell cycle progression, centrosome biology, and mitochondrial quality control.

We have a number of interesting projects available, including, but not limited to, the following:

Project Description 1***Regulation of mitochondrial quality control by ubiquitin ligase signalling pathways***

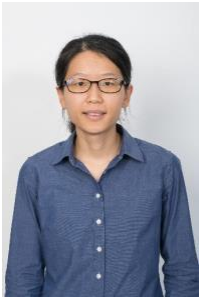
Mitochondria are essential organelles that control energy synthesis and cell signalling, as well as apoptosis. Due to their important function in cell metabolism, the quantity and quality of mitochondria must be regulated tightly, through mitochondrial fusion and fission, biogenesis, and degradation. Damaged and excessive mitochondria are recycled and degraded by mitophagy, a form of selective autophagy, a process that is misregulated in Parkinsons disease. This project will characterise the mechanisms regulating the degradation of two proteins that control the amount of mitophagy within the cell.

Project Description 2***Role of a novel ubiquitin ligase in cell division***

Accurate chromosome segregation is essential for cells to grow and divide. Errors in this process can generate changes in chromosome content, which can lead to cell death or problems with embryonic development. This project will use interdisciplinary approaches incorporating proteomics, biochemistry, genome-editing and molecular cell biology, to characterize the role of a novel microtubule-binding protein in mitosis.

Techniques you will learn in our group may include:

- CRISPR-Cas9 gene editing
 - Molecular Biology techniques (gene cloning, structure-function mutagenesis, etc)
 - Cell Biological techniques and assays
 - Biochemical techniques
 - Large-scale purifications of protein complexes
 - Microscopy (super-resolution, high resolution, live cell, etc)
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Dr Sherry Wu

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Cancer Therapeutics Lab/ Harnessing the immune system to battle ovarian cancer: A novel approach using non-coding RNAs

The high recurrence rate presents a major challenge in the clinical management of high grade serous ovarian cancer (HGSC). While stimulating our own immune system to recognize and attack tumour cells represents an attractive means to facilitate complete elimination of tumours, emerging data suggest that many of the immunotherapy tools, such as immune checkpoint inhibitors (e.g., α PDL1/CTLA4), are minimally active in ovarian cancer. The overall goal of this study is to develop effective strategies to enhance the infiltration and function of cytotoxic T lymphocytes in ovarian tumours.

While cytotoxic T-cells are strongly associated with improved patient survival in ovarian cancer, only ~30% of tumours have T-cells. This difficulty in inducing an effective anti-tumour immune response stems from the highly immunosuppressive microenvironment present in ovarian tumours. We propose to use microRNAs (miRNAs) to relieve the immunosuppressive networks in ovarian tumours, allowing T-cells to infiltrate and kill tumour cells. MiRNAs are a class of naturally occurring non-coding RNAs that are often deregulated in tumours. We and others have shown that modulation of miRNA levels in tumours can offer tremendous therapeutic benefit. The ability of miRNAs to regulate multiple pathways simultaneously can prevent pathway redundancy or resistance, a feature which cannot be achieved by many other therapeutics.

There are two specific aims for this project. Aim 1 focuses on examining the ability of immunostimulatory miRNAs to sensitize ovarian tumours to existing TGA-approved immune checkpoint antibodies. Aim 2 assesses the ability of these immunostimulatory miRNAs to induce a potent tumour antigen-specific immune response in ovarian cancer. Ultimately, strategies developed here could harness the power of the immune system to eliminate tumours and significantly increase the survival of patients with ovarian cancer.

Techniques you will learn in our group may include: Cell culture techniques, immunological assays, animal handling experience, molecular biology techniques including PCRs, nanoparticle synthesis

**Dr Anne Conibear**

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Protein chemical biology

My research aims to understand how post-translational modifications (PTMs) affect protein structure, dynamics and interactions. PTMs regulate many fundamental cellular processes and their patterns change in diseases such as cancer, diabetes and neurodegenerative diseases. Despite their prevalence, the challenges of making modified proteins have, until recently, limited our ability to understand these regulatory and disease processes at a molecular level. Developments in protein synthesis and ligation, however, now allow us to access site-specifically modified and isotope-labelled proteins. Integrating protein and peptide synthesis techniques with structural biology using Nuclear Magnetic Resonance (NMR) spectroscopy, we provide atomic-resolution insights into the structure and interactions of post-translationally modified proteins.

Project Description 1

The role of posttranslational modifications in the structure and neuronal receptor activity of alpha-conotoxins – venom peptides from cone snails.

Project Description 2

Regulation of proteins by modification of alpha-helical structures.

Techniques you will learn in our group may include:

Protein expression and isotope labelling

Protein ligation and modification – native chemical ligation, protein conjugation

Solid phase peptide synthesis

Protein structural biology – circular dichroism, peptide and protein NMR spectroscopy

Protein purification – affinity purification, HPLC

Protein characterisation – mass spectrometry, stability, binding interactions

Dr Claire Aland

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Anatomy

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There are many areas of human anatomy that require further study. Anatomical descriptions found in modern anatomy texts are very general, and do not reflect the range of morphology that structures, such as normal, healthy muscles, display between and within individuals in a population.

Project Description 1 Morphology of plantar intrinsic foot muscles

The intrinsic muscles of the plantar surface of the human foot are arranged into 4 layers. As these muscles are fully contained within the foot, their actions pertain solely to foot and toe movements. It is not known, for example, how similar or different the equivalent muscles of the left and right feet might be. Dysfunction of some of these muscles has been implicated in various foot pathologies, and some of these muscles are used in reconstructive surgeries. For these reasons, more complete information on these muscles is needed.

Building on previous projects, this project will examine either the plantar intrinsics that move the great toe (hallux) OR plantar intrinsics that move the 2- 5th toes, using dissection, radiography and ultrasonography to map the detailed anatomy of these muscles, including segmentation and attachments, innervation, vascular supply, and calculation of potential force production.

Project Description 2 Morphology of the Suboccipital Muscles

The suboccipital muscle group consists of 4 small muscles just inferior to the base of the skull. There are 2 additional associated muscles, on each side between the neck and the base of the skull.

These muscles attach to the base of the skull, and to the first two cervical vertebrae, and are involved in the fine movements of head and neck, and controlling and stabilizing the head on neck.

There are various neurovascular structures in this region which are often targeted for regional anaesthesia. The relationship of some of the neurovascular structures to the muscular, osseous and ligamentous structures in this region have been implicated in occipital neuralgia, or chronic pain over the skull.

There is not much known of the fine structure, attachments or intra and inter-individual variability of these muscles, or of the neurovascular structures in this region.

This project aims to investigate the anatomy of the muscles and neurovascular structures in this region, with a view to their function, using radiography, ultrasonography and dissection.

Techniques you will learn in our group may include:

- Dissection
 - Radiography
 - Ultrasonography
 - Functional anatomy techniques
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Dr Kylie Tucker and Dr Taylor Dick

School of Biomedical Sciences
Musculoskeletal and motor control

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Motor Control and Pain Research & Neuromuscular Biomechanics Research

Our research provides insight into (i) the development of movement and postural control in children and adolescents, (i) the effects of acute pain and chronic injury on motor control parameters, and (iii) the neuromuscular and biomechanical mechanisms that underlie healthy and pathological locomotor function. We use innovative methodologies to improve knowledge about muscle mechanical properties and assess movement in healthy and pathological populations across the lifespan, from paediatrics to geriatrics. Integration of our data into clinical models aims to shape our understanding of how neuromuscular properties are altered with throughout development, and in musculoskeletal pain and neuromotor conditions. This information is critical in the diagnosis, treatment, and prevention of movement impairments throughout the lifespan.

The project that will be offered will depend on the funding received just prior to the honours year beginning, and the work of other students within our laboratories. However, the project(s) will broadly focus on assessing motor coordination in health and/or musculoskeletal disorders, with potential to feed into Hill-type models. We are very happy to talk with you more specifically about the project(s). Please see our latest publications via the research profile links below and email us to find out more.

Techniques you will learn in our group may include: B-mode ultrasound and shear wave elastography (muscle- tendon length changes and stiffness properties), electromyography (muscle activation), force dynamometry (musculoskeletal forces, centre of pressure), and kinematic analysis via 3D motion capture (limb movements and joint angles).

KT Researcher Profile: <https://researchers.uq.edu.au/researcher/1745>

TD Researcher Profile: <https://researchers.uq.edu.au/researcher/19714>

Web KT: <https://biomedical-sciences.uq.edu.au/research/groups/motor-control-pain-research>

Web TD: <http://biomedical-sciences.uq.edu.au/research/labs/neuromuscular-biomechanics>



Professor Chen Chen

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Physiology and Cell Biology

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Chen Lab - Endocrinology and metabolism/ hyperinsulinemia and hyposomatotropism in obesity and diabetes

Disruption of growth hormone (GH) secretion is a common pathological change in obesity. Reduced GH secretion accelerates fat accumulation and impedes lean mass preservation, thus, exaggerating the development of obesity. The mechanisms underlying the impaired GH secretion is not fully clear. Ghrelin is a GH-stimulating, stomach-derived peptide that also has orexigenic action. Low levels of ghrelin in obesity are thought to contribute to the reduction of GH. Ghrelin supplement may restore decreased GH secretion in obesity, but it may worsen obesity by its orexigenic action. Therefore, novel molecules able to restore GH secretion without worsening obesity are in urgent need. Recently, an endogenous molecule, Leap-2, has been identified as a non-competitive allosteric antagonist of the GH secretagogue receptor 1a (GHSR1a). Overexpression of Leap-2 impairs the ability of ghrelin to maintain the body weight and blood glucose levels in a period of extreme food shortage by diminishing ghrelin-induced GH release in vitro. It remains unknown whether Leap-2 contributes to the markedly decreased ghrelin and GH secretion in obesity.

Project Description 1

Are circulating levels of Leap2 and gastric expression of leap2 mRNA differ in obese animal models compared to controls?

Project Description 2

How does LEAP2 modify GH secretion on somatotrophs in vitro and regulate GH patterning in mice in vivo?

Project Description 3

Will LEAP2 centrally act on GH regulating neurons to influence GHRH and somatostatin in vitro?

Techniques you will learn in our group may include: The project will include cell biology techniques including cell culture, polymerase chain reaction (PCR), western blot, Enzyme-Linked Immuno Sorbent Assay (ELISA) and immunohistochemistry. In addition, small animal research techniques will also be applied to the project, including animal handling and restraint, injections, anaesthesia, oral gavage, blood sampling, tissue dissection and fixation.

A/Prof Glenda Gobe



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Physiology/Pathology

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Centre for Kidney Disease Research

Role of Tissue Factor in Kidney Disease

(with A/Prof David Vesey, david.vesey@health.qld.gov.au)

Patients with kidney disease often present in an enhanced thrombotic state. Recent studies have shown elevated levels of tissue factor (TF) are present in the circulation in various diseases. The kidney can produce TF and it can be detected in the urine.

AIMS & METHODS

1. Establish and validate assays or methods to measure TF (Protein: Western blotting, ELISA, blood clotting assays, activity assays, expression: qPCR).
2. Measure levels of TF in body fluids (urine, plasma, saliva) using these methods. The student will collect samples from patients with kidney disease and measure levels of TF. A bank of samples is already available but further samples will need to be collected.
3. Investigate TF produced by kidney cells in culture. The student will use established cell lines and primary cultures of kidney cells. Previous studies have revealed that TF released by kidney cells is present in micro-vesicles.
4. Develop methodology for isolation of micro-vesicles/particles (MV/MP) and their characterisation. Are MV/MP a better starting material for TF measurement.
5. Measure TF and proteases in human kidney tissue section by IHC.
6. Studies will also focus on Protease-Activated receptor-2 (PAR2) as we have found PAR2 activation enhances TF production by Kidney cells.

OUTCOMES

1. Discovery of new biology around TF and kidney disease.
 2. Determine if TF is a useful biomarker for chronic kidney disease progression.
 3. Uncover links between TF and kidney inflammation & PAR2 signalling.
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Cardiorenal Laboratory

General focus of the lab: Development of novel treatments for cardiorenal syndrome

Heart failure patients often develop secondary renal dysfunction. When a patient develops both cardiac and renal dysfunction it is termed the cardiorenal syndrome. Currently, there are no effective treatments to rescue renal dysfunction in heart failure patients and there is an unmet clinical need to develop new treatment strategies. Recent data indicate that reduced nitric oxide bioavailability plays a central role in inducing renal injury in the setting of heart failure. Our data indicate that drug interventions which restore nitric oxide bioavailability also reduce renal injury and restores renal function in preclinical animal models.

Project Description

In this project, we will test whether a new drug developed by us can rescue renal function in mice with heart failure. Our data indicate that this new drug can increase the activity of an enzyme (Angiotensin converting enzyme 2; ACE2) in the in vitro setting. ACE2 can increase the bioavailability of angiotensin 1-7 and this peptide has been demonstrated to exert cardio and reno-protective effects in both pre-clinical and clinical settings.

Techniques you will learn in our group may include: Cell culture experiments, measurement of ACE2 activity, measurement of Ang 1-7 in cell based systems, in vivo mice experiments involving measurement of glomerular filtration rate, albuminuria, renal fibrosis and inflammation together with cardiac measurements via echocardiography.



Dr David Simmons

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Developmental Biology

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Simmons Lab/Placental Biology

The placenta is a highly specialized organ of pregnancy that facilitates the exchange of nutrients and wastes between a mother and her developing fetus (or fetuses if you happen to be a mouse). Abnormalities in placentation can result in a range of serious pregnancy complications such as preeclampsia and intrauterine growth restriction. Our lab has a particular focus on the development of the fetomaternal exchange interface within the placenta, both from the perspective of cell lineage/differentiation and at the level of tissue morphogenesis. We use transgenic mice and trophoblast stem cells to investigate these aspects of placentation.

Project Description: Understanding Trophoblast-Endothelial Cell Interactions at the Feto-Maternal Interface

Within the placenta, the maternal circulation (lined by trophoblast cells) becomes intimately apposed with the developing fetal circulation (lined by endothelial cells), to create a highly ordered transporting structure called the interhemal membrane. The morphogenesis of the two circulations is interdependent, and an understanding of the reciprocal crosstalk between trophoblast and endothelial cells regulating this process is critical to understanding both the normal formation of this structure, and how defects in these processes contribute to pregnancy complications.

This project will interrogate the effects of endothelial-derived signalling pathways on trophoblast differentiation. We will use a combination of tissue culture models and histological analysis of placental tissue to define important regulators of trophoblast differentiation, with an emphasis on endothelial pathways responsive to changes in fetal blood flow.

Techniques you will learn in our group may include:

Working with transgenic mouse lines, trophoblast stem cell cultures, histological techniques such as in situ hybridization and immunohistochemistry, and general molecular biology techniques (such as RNA isolation, qPCR, western blotting, etc).



Biomedical Education Research Group (BERG)

Leader: Dr Kay Colthorpe

School of Biomedical Sciences
Biomedical Education

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Biomedical Education Research Group

Our research group brings together academics and students whose research interests are in understanding student learning in the biomedical sciences. These include topics such as understanding how students learn to think, act and communicate science, the learning processes students develop and use as they enter and progress through university, and their engagement with learning aids and technologies such as feedback and mobile devices. Ultimately, our research aims to inform education, to improve students' experience of learning science and aid their success. As a student in our research group, we value the insights you bring, and during your research you will deepen your understanding of learning, aiding your future endeavours.

Specific projects are listed below, but within these general themes, can be tailored to suit your interest.



Dr Louise Ainscough



Dr Judit Kibedi



Dr Hardy Ernst



Ms Tracey Langfield

Project Descriptions

Student learning in the sciences - Dr Kay Colthorpe

<https://biomedical-sciences.uq.edu.au/profile/717/kay-colthorpe>

Science can be a challenging discipline to learn, both in terms of the content and the skills needed to perform and communicate science. Students can use a variety of learning strategies to tackle science, with some approaches being more effective than others. My research investigates how undergraduate students learn science. Specific areas of interest include science communication, the acquisition of scientific and research skills, 'meta-learning' and self-regulated learning.

Developing learning skills in the early years of university - Dr Louise Ainscough

<https://biomedical-sciences.uq.edu.au/profile/876/louise-ainscough>

The transition from school to university can be challenging. To cope with the expectations of university learning, students need to develop independent learning skills, time management and academic resilience.

My research involves asking students about how they learn in the early years at university in order to understand how students can improve their academic performance.

Supporting students' development of effective communication and thinking – Dr Judit Kibedi

<https://biomedical-sciences.uq.edu.au/profile/1320/judit-kibedi>

Students' mastery of science communication and thinking skills is a highly complex and nuanced process. My research is focused on supporting their acquisition of the conventions in science writing, critical thinking and science literacy, particularly in their early academic years, and better understanding the strategies they employ to facilitate this development. Feedback is an important component of this growth, so I am also interested in how students use and apply feedback, as well as developing tutors' skills in the construction and delivery of good feedback.

Mobile learning and student-generated multimedia - Dr Hardy Ernst

<https://biomedical-sciences.uq.edu.au/profile/603/hardy-ernst>

I am interested in supervising research projects that investigate either (1) student engagement through student-generated learning, especially student-generated multimedia, or (2) how mobile technologies can enrich learning on and off campus.

How do we learn anatomy? - Ms Tracey Langfield [https://biomedical-](https://biomedical-sciences.uq.edu.au/profile/747/tracey-langfield)

[sciences.uq.edu.au/profile/747/tracey-langfield](https://biomedical-sciences.uq.edu.au/profile/747/tracey-langfield)

Learning anatomy can be challenging for students and poor performance in the fundamental anatomy courses may negatively impact future performance in anatomy-based science or clinical programs. I am interested in facilitating student learning of anatomy and, more broadly, students' development of self-regulation skills for lifelong learning.

Techniques you will learn in our group may include: Qualitative and quantitative analysis techniques, including data acquisition, thematic analysis, and a range of statistical analyses. In addition, our students gain particular insight into learning processes and develop their science communication skills, these have proved valuable to them in their further studies and careers.



Dr Mary-Louise Roy Manchadi

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Biomedical Education

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Student Engagement with Lectures and “Blended Learning”

Universities around the world are grappling with cultural changes and shifts in educational strategies as well as student expectations towards their university experiences. In an age where students can do on-line courses at universities far from Australia, lecturers are carefully considering student engagement and success here at UQ and on other Australian campuses.

Lecture slots currently have the bulk of contact hours in most courses, with ~39 lectures across a semester. These are largely recorded and used as a teaching resource. Student attendance to the traditional lecture spaces has decreased significantly across all campuses, with a UQ average of ~60% across all courses.

My work seeks to better understand what motivates students to attend these spaces in their traditional sense and in the transition to an increasing number of flipped classroom models and blended learning, where the academics are less on the stage, and are instead facilitating activities for learning, while students will be engaged in learning content largely outside the lecture slots.

Project Description 1

In association with the Spaces Management team at UQ, we have access to accurate student numbers across the university so as to track lecture attendance based on body heat entering and exiting lecture venues. Aligning the use of these sensors with course, lecturer and topic will give a rich qualitative data set. In addition to this, surveys will be done with key courses across the university for those which have high attendance and those with low – and drivers identified and shared with the Course Coordinators, along with such learnings being used to inform the Teaching and Learning Community.

Project Description 2

In collaboration with Dr Amy Johnston in the School of Nursing (HABS), assessing these same parameters in the professional cohort is the goal, but cross-referencing year groups with those of similar students enrolled at Griffith University. As a professional degree approved by the national accreditation body, the content is approved, but the method of delivery is varied. Aligning similar courses and comparing academic achievement and engagement is the goal of this project.

Project Description 3

I am very happy to work with a student who may have their own ideas around educational research using scientific method. Please feel free to contact me and discuss what possible options might be to assist you in such a project.

Techniques you will learn in our group may include: statistical analysis, qualitative and quantitative techniques, including analysis of student responses, writing, presentation, and communication skills, interactions with UQ professional staff throughout the university.

**A/Prof Kai-Hsiang Chuang**

Queensland Brain Institute
Neuroscience

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Laboratory of Functional and Molecular Neuroimaging

Neuroimaging, such as functional magnetic resonance imaging (fMRI), is a powerful tool enables *in vivo* mapping structural, functional and connectivity changes of the brain noninvasively. As the same technique can be applied in both humans and animals, it allows direct translation of findings in animal models to humans, or vice versa. The laboratory aims to identify imaging-based neuro-endophenotype of brain functions and disorders (such as learning, memory and dementia) to improve our understanding of cognitive functions and to facilitate early diagnosis and evaluation of treatment. Three active projects are ongoing:

Project 1: Imaging brain connectome of learning and memory

How memory is formed and stored has been one of the most intriguing question in neuroscience. Besides cellular and molecular changes in this process, recent studies indicate that learning shapes large-scale brain networks. However, the relationship between brain connectivity and behaviour is still elusive. This project aims to identify connectivity signature of memory formation so as to determine key brain areas and pathways in this process. We will use advanced magnetic resonance imaging (MRI) techniques to characterise the structural and functional connectivity changes in memory consolidation in mouse models following behavioural training. Network analyses will be applied and correlated with behaviour. The behaviour-related brain networks identified will be validated by opto- and chemo-genetic methods.

Project 2: Imaging brain disorders and treatment response

Neurodegenerative diseases, such as dementia, are irreversible and generally incurable. Therefore early detection is essential so that interventions can be applied to slow down its progression. Sufficient and efficient nutrient delivery and waste removal by the cerebrovascular system are vital for the brain health. Although cerebrovascular dysfunction has been observed in various neurodegenerative diseases, its role in the pathogenesis is still unclear. This project will investigate the cerebral blood flow, a key indicator of cerebrovascular function, and its relationship with brain function and disease progress in mouse models of dementia and human patients. We will use noninvasive MRI to quantify cerebral blood flow and correlate with structural and functional connectivity, blood biomarkers and behaviour to understand the interplay among cerebrovascular function, brain connectivity and the cognition.

Project 3: Understand neural basis of resting-state network

An interesting phenomenon of the brain is the spontaneous formation of synchronous low frequency oscillation across the whole brain at the resting state. These resting-state networks not only indicate the intrinsic organization of the brain, but also changes with brain state, attention, learning, memory and disorders. Therefore it could be a biomarker of relevant brain functions and diseases. As these networks can

Biomedical Sciences Honours Projects

be detected by functional MRI (fMRI) noninvasively, it has been widely applied in human and even animal models. However, the neural mechanism of such slow oscillation and the basis of resting-state fMRI are largely unknown. We aim to understand the neural basis underlies the resting-state networks, the axonal connectivity that supports the network topology and their relevance to behaviour, such as learning and memory. We will apply fMRI in rodent under pharmacological and behaviour manipulations and validated by electrophysiology, neuronal tract tracing, lesion and opto-/chemo-genetics to determine the neural underpinning of the fMRI signal oscillation and its relationship with particular neural pathway and transmission system.

Techniques you will learn in our group may include: functional MRI, diffusion tensor imaging (DTI), image processing, neuroanatomy, brain function, behaviour



A/Prof Paul Dawson

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Neurosciences and Cognitive Health

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Developmental Disorders

Associate Professor Paul Dawson is the Mater Research Head of Education, a Principal Research Fellow and Program Leader of the Neuroscience and Cognitive Health research program at Mater Research, as well as being an Honorary Associate Professor at the University of Queensland. Paul leads the Developmental Disorders Research Group at the Mater with a team of more than 10 biomedical and clinical researchers.

Paul has authored over 80 scientific publications, with a strong research interest in the developmental origins of neuro-disability.

Paul's research group collaborates very closely with neonatologists, maternal fetal medicine specialists, obstetricians, bioinformaticians and biochemical pathologists at Mater to investigate clinical, biochemical and genetic markers that predict adverse neurodevelopmental outcome. In particular, his research is focussed cerebral palsy, non-syndromic intellectual disability, autism and adverse neurodevelopmental outcomes in preterm infants.

Project Description 1

Molecular analysis of the *SLC26A1* gene.

This project aims to investigate sequence and splice variants of the *SLC26A1* gene.

Project Description 2

Molecular analysis of sulfate biology in pregnancy.

Sulfate is an important nutrient for fetal growth and development. This study will investigate the expression profile of genes involved in sulfate biology during mouse pregnancy.

Techniques you will learn in our group may include: In silico analyses of human and mouse genes, RNA isolation, cDNA synthesis, PCR, sub-cloning into mammalian expression vectors, cell culture and Western blot analysis.



Prof Barry Dickson

Queensland Brain Institute
Neuroscience, Neural Circuits, Genetics and Behaviour

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Neural circuits underlying locomotion

As animals walk, run, or hop, motor circuits in the spinal cord convert descending “command” signals from the brain into the coordinated movements of many different leg muscles. How are command signals from the brain deconvolved into the appropriate patterns of motor neuron activity? We aim to answer this question for *Drosophila* by studying the functional organization of leg motor circuits in the ventral nerve cord, the fly’s analogue of the spinal cord. In *Drosophila*, individual neuronal cell types can be reproducibly identified and manipulated using genetic reagents that have been developed to target specific descending neurons, interneurons, or motor neurons. We have also established imaging pipeline to identify novel neurons that are behaviourally relevant and probe how they talk to each other. A range of projects involving optogenetics, two-photon imaging, machine learning assisted behavioural analysis and circuit modelling are currently open to honours students with a background in any area of molecular biology or experimental or theoretical neuroscience.

Techniques you will learn in our group may include: fly genetics; two-photon imaging; histology and confocal imaging; quantitative behavioural assays; basic programming and data analysis



Prof Geoff Goodhill

QBI / SMP
Neuroscience

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Development of brain wiring and neural coding

We are interested in how brains process information, particularly during development. This includes how brain activity develops to represent sensory information, and how growing nerve fibres (axons) use molecular cues to make guidance decisions. The laboratory uses a combination of experimental and computational techniques.

Project Description 1

Using fluorescent calcium indicators it is possible to image the activity of large populations of neurons in the larval zebrafish brain. We are using this system, combined with advanced mathematical and computational analyses, to investigate how neural activity represents information about the world, and how this representation changes over neural development. We also use assays of zebrafish prey-capture to correlate brain activity with behaviour. The project will involve (i) recording both spontaneous and stimulus-evoked neural activity in zebrafish using 2-photon and/or light sheet microscopy and analysing how this changes over development, (ii) analysing prey-capture behaviour in developing zebrafish.

Project Description 2

Our recent work suggests that axon growth and guidance may be linked at the level of signal transduction in a way not previously appreciated. In this project you will test this idea by growing neurons cultured from rats in new types of custom-engineered microfluidics chambers.

Techniques you will learn in our group may include: advanced microscopy, behavioural assays, tissue culture, computational modelling (for those with a strong mathematical background).



Dr Rodrigo Medeiros

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Neurula Lab/Investigating the underlying molecular mechanisms of Alzheimer's disease

Role of the immune system in neurodegeneration

Dr Medeiros discovered that Alzheimer's disease promotes defects in fundamental molecular events that limit and resolve inflammation, and demonstrated that such changes account for a substantial portion of the disease pathogenesis. Currently, the Neurula lab is undertaking the challenge of using and developing novel laboratory models in parallel with studies on affected human subjects to elucidate the underlying molecular mechanisms linking inflammation to β -amyloid, tau pathology and cognitive decline. Understanding these mechanisms will allow definition of the biological pathways involved in the onset and progression of Alzheimer's disease, and identify potential therapeutic targets for the management of this devastating disorder.

Impact of comorbidities in brain ageing and disease

The Neurula Lab also studies the impact of comorbidities in neurodegeneration and Alzheimer's disease. We seek to understand how concurrent diseases that commonly occur in the elderly may modulate neurodegeneration and age-related changes in the brain. We have been particularly interested in infections, diabetes and traumatic brain injury as major regulators of biological processes, and are developing genetic and pharmacological agents to manipulate these pathways in Alzheimer's disease.

Techniques you will learn in our group may include: Human and mouse brain slicing, cell cultures, protein and RNA extraction from tissues and cells, western blot, immunofluorescence, microscopy and ELISA.



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Single Molecule Neuroscience Laboratory

The overall goal of our research is to determine how brain cells communicate and survive in health and disease. Our lab focuses on the molecular events that govern vesicular trafficking within presynaptic nerve terminals and neurosecretory cells. We mainly use super-resolution microscopy to assess the dynamic change occurring during trafficking events.

Project 1 - Dynamic nanoscale organisation of the neuronal communication machinery

This project will investigate the dynamic nanoscale organisation of proteins implicated in neurotransmitter release such as Munc18, Munc13 and syntaxin1A.

Project 2 - Dynamic nanoscale organisation of the neuronal communication machinery

This project will investigate the dynamic nanoscale organisation of proteins implicated in endocytosis such as clathrin and dynamin.

Project 3 - The lipidomic of memory

This project will investigate the change in lipid metabolites associated with memory acquisition in various areas of the healthy and diseased brain

Techniques you will learn in our group may include: Single molecule imaging, super-resolution microscopy, mass spectroscopy, lipidomics, proteomics

Prof Bryan Mowry & Dr Zhaoyu Li

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Li lab and Mowry Lab *C. elegans* as a model to study neuropsychiatric disorders

Abnormalities of neural plasticity, including pre-pulse inhibition and facilitation have been reported in schizophrenia (SCZ). However, how these defects are generated by SCZ- associated genes are poorly understood. Here we want to address this question using *C. elegans* as a model.

Project Description

C. elegans comprises a small nervous system with only 302 neurons. Nevertheless, this organism is able to perform a wide range of behaviours including simple forms of neural plasticity such as pre-pulse facilitation. Defects in pre-pulse inhibition (PPI) and pre-pulse facilitation (PPF) are observed in SCZ patients, so *C. elegans* can be potentially used to identify and functionally characterise susceptibility genes for schizophrenia. In this project, we will first setup stable behavioural assays for PPI and PPF. Using these assays, we aim to functionally characterise the SCZ- associated genes and dissect the regulatory network of these genes.

Techniques you will learn in our group may include:

optogenetics, behavioural test and analysis, genetics



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Mimicking sleep-related sleep rhythms to promote neuroplasticity

Amongst its many functions, sleep plays a critical role in consolidating the memories and skills that were acquired during the day. When we learn a new skill or store a memory, certain physiological processes, known collectively as **neural plasticity**, take place that retain the acquired information. During a particular phase of sleep known as slow wave sleep (SWS), large, highly synchronous bursts of low-frequency brain activity known as **slow-wave oscillations** are critical in consolidating these plastic changes. Consolidation is important in promoting long-term storage of information.

The projects being offered will investigate a novel approach to harnessing the beneficial effects of sleep. Rather than boosting sleep quality to improve learning, this study will *mimic* the components of sleep that are beneficial to enhancing plasticity - in the awake human brain. This will be achieved by using a weak, often imperceptible form of electrical brain stimulation known as transcranial alternating current stimulation (tACS).

Project Description

The specific project(s) being offered in the lab will be decided and agreed upon by the student and supervisor. They will involve brain stimulation, induction of neuroplasticity, and quantifying the induced changes.

Techniques you will learn in our group may include: Transcranial magnetic stimulation (TMS), transcranial alternating current stimulation (tACS), electromyography (EMG), electroencephalography (EEG)

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Neurodegeneration Pathobiology Lab

Motor neuron disease (MND) and frontotemporal dementia (FTD) are both fatal neurodegenerative diseases that currently lack effective treatments. These diseases are characterised by aggregation and cytoplasmic accumulation of a nuclear protein known as TDP-43, within neurons. By uncovering the molecular mechanisms involved in TDP-43 malfunction, our lab aims to identify new potential therapeutic targets for MND and FTD.

Project 1: Identifying new protein targets to modulate TDP-43 pathology

In screening experiments, we have identified hundreds of proteins that are altered very early in disease in genetically modified TDP-43 mice. In this project, you will investigate changes in protein levels and sub-cellular localisation of several of our top-priority targets, in brains and spinal cords from these TDP-43 mice. You will also investigate whether the most promising protein targets can modulate TDP-43 aggregation and mislocalisation in cell lines and primary neurons, using CRISPR gene knockout and plasmid/lentiviral-mediated over-expression studies.

Project 2: New neuronal cell models to study TDP-43 proteinopathy

We are developing new methods to track TDP-43 alterations in disease. In this project, you will use CRISPR gene editing to introduce specific disease-mimicking changes, and to create genetically-encoded fluorescent protein markers, in neuronal cell lines. You will then use these cell lines to analyse how TDP-43 aggregation occurs and how changes in TDP-43 localisation affect neuronal function using biochemical assays, immunoblotting, and fluorescent live-cell imaging.

Project 3: The role of neuroinflammation in MND/FTD

In this project, you will characterise how the levels of several newly-identified neuroinflammatory regulators change over time in TDP-43 mice. Several of these proteins represent potential therapeutic targets, and so you will also use drugs and viruses to modify these proteins in the TDP-43 mice, and then analyse whether these alterations affect disease onset, progression, motor phenotype, brain and spinal cord neuropathology, and mouse survival.

Techniques you will learn in our group may include: cell line and primary neuron culture, FACS, CRISPR, molecular biology, virus production, biochemical assays, immunoblotting, qPCR, fluorescent microscopy, mouse handling/drug administration/behavioural testing, mouse surgery and dissection, and analysis of mouse and human brain and spinal cord samples. You will work as part of a cooperative team, and also attend seminars, participate in lab meetings and learn how to effectively organise, analyse and present your data to others.



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Emma Hamilton-Williams Lab

Immunology and pathogenesis of type 1 diabetes

During her PhD in France, Anne-Sophie worked on regulatory T cells in cancer immunotherapy in mice in Prof. David Klatzmann lab. She showed that a subpopulation of activated/memory Tregs were constantly activated by self-antigens and recognized self-tumor-antigens when a tumor was present, leading to rapid reactivation, suppression of the conversion of naïve T cells to effector T cells, and protection of the tumor cells. She then moved to Brisbane, Australia, where she undertook her first postdoc under the supervision of Prof. Ian Frazer, who is internationally renowned for the co-creation of the technology for the cervical cancer vaccines Gardasil and Cervarix. She carried out a highly successful and independent program of work on mast cells in a model of papillomavirus-associated pathologies in mice. After a period of maternity leave in New Caledonia, she returned to Brisbane in March 2016 and joined Prof Ranjeny Thomas group, co-supervised/mentored by Dr Emma Hamilton-Williams, to work on Immunotherapy for type 1 diabetes (T1D).

Anne-Sophie has an outstanding track record in immunology and immunotherapies.

Project Description

We are currently investigating the use of a liposome for antigen-specific therapy in the mouse model of T1D, to restore tolerance in autoreactive islet-specific T cells. Its role is dual: fostering regulatory T cell function and terminating pathogenic T cells.

Selected peptide and drug are encapsulated into liposomes, injected to mice, taken up by antigen-presenting cells (APCs) and then presented to T cells.

- (1) Effects of the liposomes on APCs
- (2) Effects of the liposomes on T cells (regulatory and effector T cells)

Techniques you will learn in our group may include: flow cytometry, cell sorting, PCR, histology,

**Prof David Evans**

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Statistical Genetics Laboratory

We are interested in the genetic basis of common complex diseases in humans and using this information to improve human health. We work on a variety of diseases and traits including (but not limited to) osteoporosis, severe septic shock, autoimmune disease, and early life phenotypes. We are primarily a dry lab that uses computational approaches like genome-wide association analysis and Mendelian randomization on large-scale datasets to answer interesting questions in genetic epidemiology. Ideally we are looking for students who have training and good grades in statistics and are interested in medicine/biology.

Project 1

Asthmatics often use glucocorticoids (GCs) to control their asthma, but the evidence regarding whether they should continue to use them during pregnancy is mixed. Animal models have consistently demonstrated that prenatal exposure to GCs results in reduced size at birth. In humans, male and female growth patterns throughout pregnancy differ in response to high cortisol exposure. This project will use a technique called Mendelian randomization to examine the causal effect of corticosteroid exposure in mothers on offspring birthweight. Analyses will be performed using >300,000 individuals from the UK Biobank Study and Early Growth Genetics Consortium who have genome-wide SNP data and measures of offspring birthweight.

Project 2

The project involves the systematic curation, downstream analysis and biological annotation of genome-wide association summary statistics of over 4000 human complex traits and diseases. The project will also involve identifying the overlap of genes and biological pathways between different complex traits and diseases. The student will learn to use the high-performance computer cluster to conduct the required analyses. Data resulting from this project will be deposited in a public database for researchers to browse.

Techniques you will learn in our group may include: Mendelian randomization; Genome-wide association analysis (GWAS);



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Haass Lab/Microtubule-Dependent Mechanosensing in Cell Migration and Cancer Invasion

The majority of cancer deaths are due to metastatic disease, highlighting the need for ‘migrastatics’, therapeutics which act to inhibit migration. Metastatic success requires cells to navigate complex cellular environments, adapting either their shape to navigate between matrix fibres or adapting their environment to facilitate movement between tight spaces. Innovative imaging and cell biology approaches have recently uncovered novel biology that is unique to cells navigating confined 3D dimensional spaces vs 2D, underlining the significance of understanding melanoma invasion in mechanically relevant cell culture models. Our lab focuses on understanding the fundamental mechanisms governing the bi-directional relationship between melanoma and extracellular matrix during 3D dimensional cell culture models of melanoma invasion.

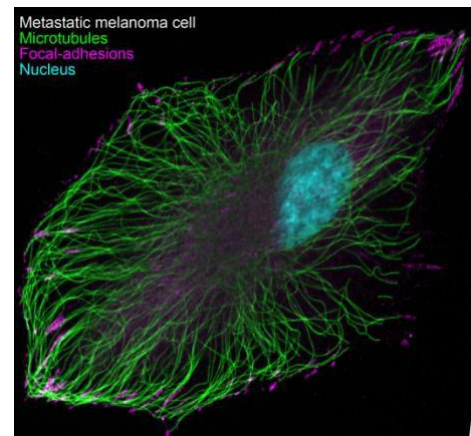
Project Description 1

Establish mechanically relevant three-dimensional cell culture models of MBM (melanoma brain metastases). Investigate MBM motility, proliferation and survival in extra-cerebral (collagen I) and brain (HA) matrices using a combination of high-resolution live-cell microscopy, cutting-edge bio-reporters, immunofluorescence and 3D cell culture.

Project Description 2

Target the SxIP-EB1/microtubule protein-protein interaction using novel peptides in metastatic melanoma. Utilise high-resolution, multi-wavelength, live-cell spinning disc confocal microscopy to

analyse microtubule +TIP protein dynamics in response to novel peptides. Investigate alterations to 3D melanoma invasion.



See: Stehbens et al., *Nature Cell Biology* 2014, Stehbens et al., *Methods in Enzymology* 2012, Stehbens and Wittmann, *Journal of Cell Biology*, 2012. Spoerri et al., *Methods Mol Biology*, 2017.

Techniques you will learn in our group may include: cell culture, 3D hydrogel models, cell migration analysis, immunofluorescence, live-cell microscopy, spinning-disc confocal microscopy, quantitative image analysis



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Genome Plasticity and Disease Group

Cancer is one of the leading causes of mortality and morbidity worldwide. However, many of the genetic causes of tumour initiation, progression and metastasis remain unknown. Retrotransposons and pseudogenes were previously thought to be inert “junk DNA”, but new research demonstrates that they play key roles in cancer. In the Genome Plasticity Group of Professor Geoff Faulkner we are interested in understanding the roles of retrotransposons and pseudogenes contribute to the tumour formation and metastasis.

Project 1: Deciphering global and locus-specific regulation of LINE-1 retrotransposons in cancer

In cancer, but not healthy cells, ~100 L1 “jumping genes” can copy and paste themselves into the human genome. L1s can contribute to cancer initiation by activating oncogenes and inactivating tumour suppressor genes and can drive tumour evolution, underpinning resistance to chemotherapy. This project aims to determine the cause of L1 activation in cancer. We hypothesise that L1 activation is controlled at the transcriptional level by proteins that bind to L1 regulatory sequences. We further hypothesise that some proteins may control the activity of only individual L1s, explaining the considerable differences in activity observed for L1s at different genomic locations. This project will identify novel factors that regulate L1 expression in cancer, transforming our understanding of the mechanism of L1 activation. As L1 expression is highly correlated with cancer severity, these factors may hold important prognostic and diagnostic value.

Project 2: Identifying functional pseudogene-derived noncoding RNAs in cancer

Pseudogenes are dysfunctional copies of genes that have accumulated mutations and no longer encode functional proteins. Many pseudogenes retain transcriptional activity and can evolve new roles as noncoding RNAs. These pseudogene-derived noncoding RNAs are potential novel regulators of cancer growth and metastasis. We have identified a novel class of pseudogenes that inhibit the protein-coding genes from which they are derived. Several of these pseudogenes are derived from oncogenes and may serve as tumour suppressors. We will use genetic and biochemical approaches to identify the cellular functions of cancer-associated pseudogenes and the molecular mechanisms through which they act.

Techniques you will learn in our group may include: Genomics, molecular cloning, real-time PCR, proteomics, transcriptomics, cell culture.

**Prof Josephine Forbes**

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Glycation and Diabetes Complications Research

Josephine's lab is investigating new treatments for diabetes and the devastating chronic complications associated with it such as kidney disease, blindness, amputations and heart disease. With this research, Josephine aims to build a greater understanding of the biological basis of diabetes in connection with a broad range of chronic diseases and develop preventative strategies and innovative treatments to improve patient outcomes. Josephine is a scientist at heart, who likes to be close to discovery and innovation as she believes this is to be the key to future health discoveries. She keeps a clear focus on the health outcomes of her research for individuals affected by diabetes and is passionate about training our future leaders in medicine and science. "Diabetes has become so prevalent that it touches all of our lives. My inspiration is

to discover something that will better someone's life".

Project Description 1: A cohort study of changes in kidney energetics in young people with type 1 diabetes and early kidney disease

More than 1.2 million Australian have been diagnosed with by diabetes. A majority of these individuals will develop chronic complications, which make up most of the cost of diabetes to Australia. Diabetic complications include kidney disease which is a major risk factor for heart attacks and stroke. The development and progression of diabetic kidney disease remains poorly understood and current therapies used to treat this disease only slow the progression. There is evidence that early impairments in kidney function are present in young people with type 1 diabetes, well before traditional clinical parameters can identify this. This project will investigate the connection between impaired kidney function, and our cellular power stations, the mitochondria which manufacture fuel for metabolism from sugars, fats and oxygen.

The aim here is to provide a therapy in the future which will prevent progression to kidney and cardiovascular disease in individuals with diabetes, in particular adolescents and young adults.

Project Description 2: Investigating pathways to alleviate the burden of diabetes and kidney disease

A frequent complication of patients with diabetes is that the uncontrolled high blood sugar can cause significant damage to the kidney. This project aims at alleviating the burden of diabetes and kidney disease by investigating the underlying pathways involved.

Project Description 3: Advanced glycation and pancreatic islets: Using the Network of Pancreatic Organ Donors with diabetes (nPOD) resource

Type 1 diabetes is the most common chronic disease which manifests in early life. Although comprising only 10% of diabetes cases, type 1 diabetes makes up ~40% of the total cost of diabetes to Australia, due to its early life onset and complex clinical management. Type 1 diabetes is an autoimmune disease where the

body "turns on itself" and actively destroys the cells which produce the sugar storage hormone insulin. Hence, there is no cure and individuals require life long insulin administration for survival. This project aims to investigate the connection if the receptor for advanced glycation end products (RAGE) can be targeted to arrest the development of type 1 diabetes. There is some evidence that changes in sugar related molecules termed AGEs and their cellular receptor, RAGE within the body could alter sugar homeostasis as well as influencing the immune system, which ultimately culminate in the development of type 1 diabetes. In this project, we hope to gain a better understanding of the relationship between AGEs and their "receptors" at sites that are relevant for the development of type 1 diabetes and develop specific medicines to prevent type 1 diabetes development.



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Infection, Immunity and Metabolism Group

Annually over 10 million people contract tuberculosis (TB) and 1.7 million dies from this disease. Patients with type 2 diabetes (T2D) are particularly susceptible to TB and other infectious diseases and they are less likely to respond to standard antibiotic therapy. Katharina's research investigates the underlying immunological mechanisms that lead to this increased risk of TB in patients with T2D for which she received a 5-year NIH R01 grant (2015-2020). In addition, her research aims at manipulating the host immune system to better control the infection, ultimately leading to the development of novel host directed therapies to improve treatment outcomes not only in patients with T2D, but also in multi-drug resistant bacterial infections.

Project Description 1: Impact of ketone bodies on innate immunity to infection

We and others have demonstrated that diabetes impairs immune responses to *Mycobacterium tuberculosis* and other pathogens. Ketogenic diets improve symptoms of metabolic syndrome and type 2 diabetes such as insulin sensitivity and glucose tolerance. This project aims to establish whether ketones can also improve immune function to infections.

Project Description 2: Contribution of lung dysbiosis to increased susceptibility of T2D patients to respiratory infections

We have collected bronchoalveolar lavage fluid from healthy controls and T2D patients. This project will identify any alterations in the microbiome in the lung vs. the oral cavity during diabetes and identify any associations with a dysbiosis and immune function in patients with T2D vs. controls.

Techniques you will learn in our group may include: Tissue culture, bacterial cultures, ELISA, Western Blot, qRT-PCR, Flow cytometry, Isolation of immune cells from human blood, animal models, PC2 and PC3 laboratory skills.

Prof Vicki Clifton



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Mothers', Babies and Women's Health Research

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Pregnancy and Development

Professor Vicki Clifton is a National Health and Medical Research Council (NHMRC) Senior Research Fellow and a Senior Research Fellow at Mater Research. Vicki is currently one of the Program Leaders of the Mothers, Babies and Women's Health Research Theme and leader of the Pregnancy and Development research group, with a team of six at Mater.

Vicki has established an international reputation as leader in the field of asthma and pregnancy research and has authored more than 160 publications. She has a specific interest in the sex specific differences in the fetal-placental response to complications of pregnancy understanding the different strategies male and female fetuses institute to cope with adverse events in pregnancy and how this ensures their survival in early life. Her work has focussed on the recruitment of pregnant women and following them longitudinally to determine how maternal health and stress can change in pregnancy, and how that may affect the placenta, fetus and health of the child in the long term.

Project Description 1: Effect of asthma during pregnancy on maternal health, placental function, fetal growth and childhood development

Asthma is the most common disease to affect pregnant women in Australia and is associated with a number of poor outcomes for the mother and baby including asthma exacerbations, reduced growth of the baby, preterm delivery and stillbirth. There is no clinical or basic science information about how to treat pregnant asthmatic women for the best outcome for the mother and baby. This research is looking to address these issues by determining how asthma changes during pregnancy and how that effects the baby.

Project Description 2: Effect of severe asthma during pregnancy on placental function and fetal outcome

Asthma is the most common disease to affect pregnant women in Australia and is associated with a number of poor outcomes for the mother and fetus. Research suggests asthma during pregnancy can alter placental function and this may be critical for poor foetal outcomes. This study aims to identify how asthma affects placental function.

Project Description 3: The human fetal-placental glucocorticoid receptor: mechanisms that confer a sex- specific difference in glucocorticoid sensitivity

In this research project we will collect and study the placenta and the cord blood from the placenta as it tells us a lot about how the baby developed during pregnancy and how it responded to stress in the mother. We have an interest in a protein called the glucocorticoid receptor which can change in response to mothers' being stressed or sick during pregnancy. We think it plays a role in how the baby develops. We want to compare placentas from normal healthy pregnancies to placentas from pregnancies where there may be a problem such as a preterm delivery, a small baby, hypertension or diabetes in pregnancy.

Project Description 4: What is the impact of environmental exposures on families in the Queensland Family Birth Cohort Study

The Environmental and Occupational Interactions Theme will use biological samples, demographic data and general health self-reporting information to understand the impact of air quality on families enrolled in the Mater Queensland Family Birth Cohort Study. This theme also has an interest in comparing this data to a Chinese Birth Cohort Study. Specifically, this sub-study of the Queensland Family Cohort will aim to understand:



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Ian Frazer lab

The Frazer lab investigates the immunology of malignant disease, in particular cancers of the skin, head and neck, and cervix.

Project 1: Immune escape of human Squamous Cell Carcinoma (Supervision: Dr. Katharina Noske)

Background:

Keratinocytes of the skin accumulate sun-damage throughout a person's life which leads to high mutational burden. In nearly 100% of the Queensland population of 80 years or older, these sun-damaged areas develop into Actinic Keratosis (AK). Most of these pre-cancerous cells are recognised and eliminated by the immune system. Still, this process often fails, leading to progression of AK to SCC, and making SCC the most common cancer in Australia. Identifying the mode of immune escape the malignant cells use is crucial in advancing current treatments of this highly prevalent cancer.

Research Question:

Why do immune cells sometimes fail to recognise and/or eliminate AK cells and allow for progression of SCC?

Hypothesis:

During progression from sun-damaged cells to AK, some of the keratinocytes lose their ability to initiate an effective immune response, either through defective presentation of tumour-antigen or through secretion of signals that create an inhibitory environment for the immune system.

Project outline:

Preliminary findings have shown that the antigen-processing and –presenting mechanism in these cells seems to be intact. The project will therefore focus on the immune environment in human tissue across the spectrum of SCC to determine whether the keratinocytes create an inhibitory niche which prevents effective immune response.

Materials/methods used:

- Human tissue from the clinic: normal skin, sun-exposed, sun-damaged, SCC
- Cell and tissue culture
- RNA and DNA extraction and analysis by qPCR, PCR
- Protein (cytokine) analysis with FACS bead array
- Immunofluorescent staining of cellular markers
- Potentially co-culture of keratinocytes with T cells
- Genetic analysis of sequencing data to identify pathways involved in the immune escape

Expected results obtained during Honour's year:

- Immune profile of tissue across the spectrum of SCC (cytokines)
- Identify pathways involved in SCC development and validation in vitro

Project 2: Immune kinetics of HPV-infected epithelium (Supervision: Dr. Katharina Noske and Dr. Janin Chandra)

Background:

Human Papillomavirus (HPV) is a DNA virus implicated in the development of cervical cancer and Head-and-Neck cancer. While in many cases the immune system can eradicate virus-infected cells, the infection can become latent. Unknown mechanisms contribute to the development of malignancy caused by chronic HPV-infection.

Our lab uses a mouse model where the HPV E7 oncogene is expressed under a K14 promoter, meaning all keratinocytes express the E7 protein. E7 binds to the cell-cycle regulator Rb, leading to a hyperproliferative, inflammatory epithelium. The ears of these K14E7 mice are transplanted onto the flank of recipient mice (either wild-type or knockouts of interest) to study the interaction of immune cells with the HPV-infected keratinocytes.

Our preliminary data shows that the grafts expressing E7 protein are not rejected by healthy mice, even though the intact immune system of wild-type mice should recognise the virus and eliminate the infected cells.

Investigating the mechanism of immune escape of HPV is crucial in understanding the development of HPV-induced cancers, and the first step in developing immunotherapy against those cancers.

Research question:

How does HPV “hide” from the immune system, i.e. why are immune cells not able to efficiently kill virus-infected cells?

Using the grafting model, this project investigates which immune cell populations are active in the grafts and surrounding tissue over a time course. Preliminary data shows that the immune cells present in the graft at time of transplantation die within 2 weeks, and the graft is populated with cells from the recipient mice. Further studies are necessary to determine a) which cell types infiltrate the graft, b) whether their phenotype changes when they come in contact with the E7-expressing cells and c) which factors contribute to their tolerant state.

Hypothesis:

The E7-expressing keratinocytes of the graft induce an immune-suppressive environment which leads to tolerant phenotype of the infiltrating immune cells and tolerance of E7-expressing cells.

Project outline:

Ears from K14E7 mice will be grafted onto WT mice and grafts will be harvested over a time course of up to 4 weeks.

Materials/methods used:

- K14E7 mouse model
- FACS
- Potentially tissue culture and immunofluorescent staining

Expected results obtained during Honour's year:

- Timeline of infiltrating cell populations after grafting
 - Phenotyping of cell populations
 - In combination with preliminary data from our lab: factors contributing to immune- inhibitory microenvironment and involved molecular pathways
-

Project Description 3: Antigen presentation of HPV-infected cells in K14E7 mouse model (Supervision: Dr. Katharina Noske and Dr. Janin Chandra)

Background: see project 2

Using the K14E7 mouse model, this project investigates the antigen-presenting capacity of E7-expressing keratinocytes. Normally, keratinocytes expressing the E7 protein should present the peptide on their surface as part of normal peptide turnover. When this tissue is transplanted onto a mouse that does not express E7, the recipient's immune system should recognise the E7 peptide as "foreign" and elicit a killing response against these cells. From previous research in our lab we know that this is not the case.

Hypothesis:

The keratinocytes of K14E7 grafts on WT hosts fail to effectively present E7 peptide on the surface and do not elicit T cell response.

Project outline:

Keratinocytes will be isolated from the K14E7 mouse model for in vitro studies.

Materials/methods:

- K14E7 mouse model
- Primary cell culture of mouse cells
- Immunofluorescent/IHC staining/FACS for cell markers to find out whether peptide is presented or not; if not, does peptide get processed/transported
- Antigen-processing/presenting assays
- Identify potential pathways that play a role (from sequencing data obtained previously) and validate in vitro

Expected results obtained during Honour's year:

- Characterise defect in antigen presenting pathway of K14E7 keratinocytes in terms of which pathway components are involved

Techniques you will learn in our group may include:

- FACS
 - Mouse handling, dissection, tissue harvest and processing
 - Culture of primary tissue and cells, as well as cell lines
 - Immunofluorescent stainings
 - Protein assays
 - Bead arrays
 - RT-qPCR, PCR
 - Data analysis of sequencing data
-

A/Prof Allison Pettit

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Bones and Immunology Laboratory

Understanding the role of macrophages in bone marrow transplantation and using them to improve the clinical application of transplantation therapy

Haematopoietic stem cell (HSC) transplantation is a potential curative approach for haematological malignancies as well a therapeutic option to facilitate high dose cytotoxic therapy in treatment resistant solid tumours. However, HSC transplantation has significant risks that constrain its clinical application, in particular lengthy myelosuppression in the post-transplant (Tx) period due to Tx conditioning and/or poor engraftment of donor HSC. Long term complications of allogeneic transplantation, including chronic graft versus host disease (GVHD) and infection remain significant clinical problems resulting in patient mortality.

Our laboratory has identified a population of resident bone marrow macrophages that survive post transplantation and are required for HSC engraftment and haematopoietic reconstitution. The projects listed below will investigate the role of these resilient bone marrow macrophages in stem cell engraftment and blood reconstitution and determine if donor macrophage resilience extends to other organs post transplantation.

Project Description 1

Demonstrate that resilience and long term persistence of recipient BM resident Macrophages is a common tissue protective mechanism in clinically relevant cytotoxic treatments and transplantation models.

Our published data demonstrates that resilience and persistence of recipient resident macrophages is essential for successful transplantation. These outcomes are based upon the pre-transplantation myeloablation strategy, total body irradiation, that is not commonly applied in the clinical setting. In this project, we will characterise the resilience and persistence of resident bone marrow macrophages after clinically-relevant chemotherapy or combination chemotherapy plus radiation-based pre-transplant myeloablation. This strategy will also determine whether recipient resident macrophages persist in the more complex scenario of allogeneic transplantation.

Project Description 2

Determine if recipient macrophages are essential to transplantation success in the early post-transplant period.

Resilience and persistence of recipient resident bone marrow macrophages is essential for successful transplantation outcomes; however, our preliminary data suggests that these cells are also critical to stem cell engraftment. This project will investigate the impact of recipient macrophage depletion in the early post-transplant period (first 4 weeks post-transplant) on donor stem cell engraftment using multiple in vivo models of macrophage depletion.

Project Description 3

In situ characterisation of tissue macrophage repopulation kinetics following bone marrow transplantation.

Recent experimental evidence has identified that certain macrophage populations undergo replacement via self-renewal instead of, replenishment by differentiating monocyte precursors. In this project we will use two transgenic mice strains that express different fluorescent markers in their macrophages (MacApple and MacGreen mice) in a bone marrow transplantation model to distinguish in which tissues do macrophage populations regenerate via self-renewal or via monocyte recruitment, or via a combination of both regeneration strategies.

Techniques you will learn in our group will include:

Animal handling techniques: including injections, total body irradiation, chemotherapy and stem cell transplantation.

Imaging: Immunohistochemistry and fluorescent imaging of tissue sections, whole-mounted tissues and intravital live imaging.

Flow cytometry: analysis of surface and intracellular markers of HSC and macrophage populations in various tissues.

**Dr Janin Chandra**

UQ Faculty of Medicine – Diamantina Institute
Infection and immunity

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Frazer Laboratory: Establishing novel immunotherapy approaches

Immunotherapy aims to correct a mal-functioning immune system to combat a variety of diseases including cancer, autoimmunity or allergies. Many immunotherapy targets have been identified and some have been developed into powerful treatments. Immunotherapy largely targets effector T cell responses which are either suppressed or falsely activated. Professional antigen-presenting cells such as dendritic cells (DCs) play a pivotal role in T cell fates and are hence a desired immunotherapy target. Immunotherapy has the potential to effectively harness DCs to prime e.g cancer-specific cytotoxic T cell responses or shut-down self- or allergen-reactive T cells. My research focusses on understanding dendritic cell diversity and fate, and how manipulation of dendritic cells can improve disease outcomes.

Project Description

DCs, and especially cross-presenting DCs, are a highly desired target for immunotherapy. The current paradigm describes mature DC subtypes as terminally committed and specialized. However, my previous studies indicate that this model is incomplete. Understanding mechanistic underpinnings of DC commitment and fate is of significant relevance to improve DC-targeting immunotherapy approaches. The aim of this project is to determine if mature CD8⁺ lineage DCs can convert to CD11b⁺ DCs and vice versa in mice using cutting edge multi-parameter flow cytometry and CITE sequencing combining single cell RNA sequencing approaches and proteomics. Furthermore, we will investigate if DC subtype divergence can be induced, manipulated and utilized for immunotherapy approaches.

Techniques you will learn in our group may include: multi-parameter flow cytometry, cell sorting, in vitro cell cultures and manipulation, imaging flow cytometry, in vivo and ex vivo immune assays, in vivo imaging, reverse transcription quantitative PCR, analysis of single cell RNA sequencing

**Prof Jürgen Götz**

Queensland Brain Institute
Neuroscience, Cell Biology/Signalling

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Web: <https://researchers.uq.edu.au/researcher/2692>

Google Scholar:

https://scholar.google.com.au/citations?hl=en&user=67UrhCQAAAAJ&view_op=list_works&sortby=pubdate

Lab Name/Research Title: *Goetz Laboratory in Clem Jones Centre for Ageing and Dementia Research*

Professor Jürgen Götz is the Foundation Chair of Dementia Research and Director of the Centre for Ageing Dementia Research (CADR) at the Queensland Brain Institute (QBI), The University of Queensland. Prof Götz studied biochemistry at the University of Basel, and earned his PhD in immunology in the laboratory of Nobel Laureate Georges Köhler at the Max-Planck-Institute in Freiburg, Germany (1989). The Götz lab is undertaking research into better understanding the underlying causes of Alzheimer's disease (AD) and investigating potential therapeutic avenues for the disease. The lab has developed a novel ultrasound-based treatment of amyloid and tau depositing mice that we are currently developing for clinical application in AD patients. Jürgen Götz published 180+ peer-reviewed articles (3 Cell, 1 Science, 1 Science Transl Med, 2 Neuron, 8 PNAS, 8 EMBO J, 7 J. Biol. Chem., 1 Lancet, 3 Nature Rev Neurosci, 3 Nature Rev Neurol, 1 Nature Rev Drug Disc, 3 Nature Prot). His work attracted >17,200 cit (Google Scholar), with >8,100 citations since 2014 (h-index: 66).

REVIEWS

Tau-based therapies in neurodegeneration: opportunities and challenges

Chuanzhou Li and Jürgen Götz

REVIEWS

Amyloid- β and tau complexity — towards improved biomarkers and targeted therapies

Juan Carlos Polanco, Chuanzhou Li, Liviu-Gabriel Bodea, Ramon Martinez-Marmol, Frederic A. Meunier and Jürgen Götz

REVIEWS

ALZHEIMER DISEASE

Rodent models for Alzheimer disease

Jürgen Götz^{1*}, Liviu-Gabriel Bodea¹ and Michel Goedert²

The team has >24 members, working on pathomechanisms, developing antibodies and exploring therapeutic ultrasound as a treatment modality for neurodegenerative diseases. Some of the ultrasound projects are commercial in nature.

Selected projects by our researchers:

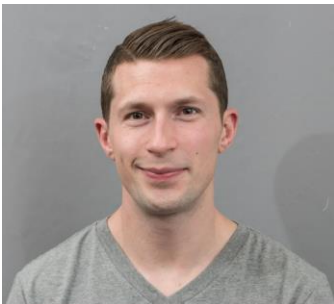
Dr Juan Polanco: <https://researchers.uq.edu.au/researcher/1464>



Dr Liviu Bodea: <https://researchers.uq.edu.au/researcher/10356>



Dr Andrew Kneynsberg: <https://qbi.uq.edu.au/profile/8525/andrew-kneynsberg>



Project Description 1: Supervisor [Prof J Goetz j.goetz@uq.edu.au](mailto:j.goetz@uq.edu.au)

Understanding the dynamics of the blood-brain barrier in response to therapeutic ultrasound

The brain is considered to be the last frontier, both in terms of understanding how it operates and in accessing it for therapeutic intervention. Our laboratory works in both spaces: deciphering the role of key molecules and signalling pathways in Alzheimer's disease (AD) and developing novel ultrasound-based techniques to overcome the blood-brain barrier (BBB). The BBB restricts the flow of blood-borne substances into the interstitial brain space, facilitated by tight junctions between the cells that constitute the blood vessels thereby hindering paracellular transport, and a very low number of vesicles, thereby reducing transcytosis. Application of pulsed ultrasound (US) in the presence of intravenously injected microbubbles has been shown to transiently open the BBB in different model organisms. In previous studies by our laboratory, we demonstrated that delivery of ultrasound to Alzheimer's mice was sufficient to remove amyloid plaques and tau tangles and restore cognitive function. The study will use two-photon microscopy to investigate the dynamics of BBB opening following ultrasound treatment aided by the use of fluorescently labelled

dextrans, thereby contributing to our strategies of using ultrasound as a therapy for Alzheimer's disease.

Project Description 2 [Dr Liviu Bodea](#) l.bodea@uq.edu.au

Characterisation of novel animal models generated by CRISPR technology

Our lab has a long-standing history of generating different animal models helpful for the understanding of the brain physiology under both healthy and pathological conditions. Recently, we have applied CRISPR-based cutting-edge genomic approaches to generate several animal models targeting different molecules important for brain function or neurodegenerative progression. The student candidate will analyse these models under the guidance of Dr. Liviu Bodea. We expect that the results from this project will have a significant impact on our understanding of how the brain is functioning in healthy or pathological conditions.

Project Description 3 [Dr Liviu Bodea](#) l.bodea@uq.edu.au

Identification of changes in the brain's molecular composition in response to the environment

Brain molecular composition is dynamic, with the surrounding environment being one of the most important elements affecting it. Based on our tools designed to identify and isolate different cell types from the brains of animals undergoing behavioural tasks, the student candidates will investigate these molecular-level changes in the brain under the guidance of Dr. Liviu Bodea. This work will provide important advancements in our understanding of brain's response to the environment at the molecular level.

Project Description 4 [Dr Liviu Bodea](#) l.bodea@uq.edu.au

Developing CRISPR based *in vitro* tools

Simplistically, normal cellular functions are based on intracellular signalling that rely in turn on interactions between particular proteins. To be able to study these functions, either a genetic or pharmacologic approach is applied to alter one of these proteins and investigate its effect. The recent advances in CRISPR-based genome editing opened the possibility of fast and targeted introduction of proteins modification. During this project, the candidate will work under the guidance of Dr. Liviu Bodea to generate *in vitro* mutants using cutting-edge CRISPR-based techniques. The tools generated in this project will significantly enrich and update the available toolset useful to study brain's functions.

Project Description 5 [Dr Juan Carlos Polanco](#) j.polanco@uq.edu.au

Unravelling how exosomes induce and propagate tau pathology

Alzheimer's Disease (AD) is an irreversible and progressive dementia characterized by neurodegeneration and concomitant neuronal cell loss. The pathological progression of AD involves the aggregation and deposition of two proteins, β -amyloid and tau. The tau pathology in AD usually starts in the hippocampus and the entorhinal cortex, and as AD progresses it spreads to other cortical areas in a pattern that suggests that neuronal interconnectivity facilitates AD

progression, characterized by increasing tau aggregation and the presence of cytoplasmic neurofibrillary tangles (NFTs) in an increasing number of neurons. This suggests an active spreading mechanism, which could be via extracellular vesicles such as exosomes or microvesicles. We have established that brains from mice with tau pathology produce tau seeds 'encapsulated' within exosomes. Furthermore, exosomes containing tau seeds can induce tau aggregation in recipient cells, and exosomes spread from neuron to neuron by hijacking endosomes. Exosomes are internalised by recipient cells via endocytosis. Consequently, tau seeds must not only be able to exit the exosomal membranes, but they must also be able to escape the endosomes in order to access cytosolic tau and induce corrupting cycles of tau aggregation. We aim to determine how exosomal tau seeds escape endosomes, and whether controlling the exosome production or traffic can halt or reduce tau pathology.

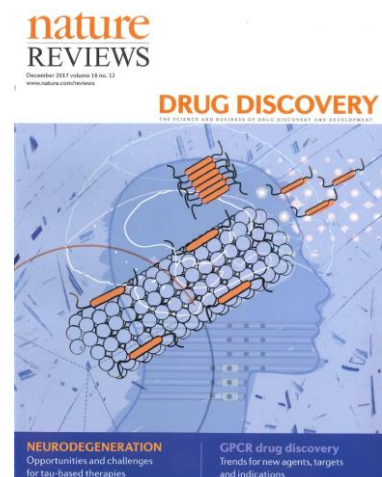
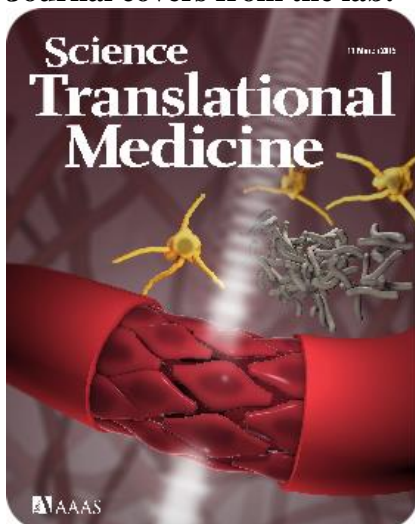
Project Description 6 [Dr Andrew Kneynsberg](#) a.kneynsberg@uq.edu.au

Understanding the fundamental biology of microtubule associated protein tau

Alzheimer's disease and other neurodegenerative diseases (termed tauopathies) are characterized by the accumulation of the tau protein into identifiable brain pathologies. The role of tau in disease, however, is not well understood, nor are the functions tau in healthy neurons. In order to better understand the role of tau in disease pathophysiology, our group investigates tau's basic cellular functions. In addition to tau regulating microtubule dynamics, tau is known to play a role in axonal trafficking, phosphotransferase activity, and protein translation through interaction with Fyn kinase. These functions require specific regulation of tau localization and activity, within the intracellular compartments of neurons. The Honours candidate will investigate tau biology, localization, and involvement in cellular functions using techniques in primary cell culture, protein biochemistry, and advanced microscopy.

Techniques you will learn in our group may include: Mice handling procedures, Behaviour experiments, Cell culture, Histology and Imaging of cells and mice brain sections, Cloning and Molecular Biology techniques.

Journal covers from the lab:





Dr Helmut Schaider

UQ Diamantina Institute - Dermatology Research Centre
Infection and immunity

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Web: <https://researchers.uq.edu.au/researcher/8126>

Project title: Stress tolerance in human keratinocytes and fibroblasts

Project description: Adaptive cellular reprogramming due to chronic sub-lethal stress like hypoxia or UV irradiation in differentiated cells like fibroblasts is not very well understood. Our lab has recently developed an in vitro stress tolerant model in cancer cells. This model describes a state- wise transition represented by a slow cycling phenotype and a colony state preceding permanent resistance. The project will use the model to develop a similar approach in human keratinocytes and fibroblasts isolated from foreskin and adult human skin. The outcome of this project will pave the way to obtain novel mechanistic insights into adaptive stress tolerance in differentiated cells.

Preferred educational background: B.Sc. or M.Sc., Masters by Research. A background in Biochemistry & Molecular Biology, Cell Biology or Pharmacology is essential, additional background in epigenetics and metabolism would be advantageous.

This project is available until September 2019 unless a suitable candidate is found prior.

Contact details: Dr. Helmut Schaider, Dermatology Research Centre, The University of Queensland Diamantina Institute, Translational Research Institute, 37 Kent Street, 4102 Woolloongabba, Brisbane, QLD, Australia



Professor John Fraser MB ChB, PhD, FRCP, FFARCSI, FRCA, FCICM

Critical Care Research Group, Northside Clinical School, Faculty of Medicine
Physiology

Phone: 07 3139 6880

Email: Dr. Jacky Suen (j.suen1@uq.edu.au)

Web: <http://ccrg.org.au>

Critical Care Research Group (CCRG)

The CCRG is based at the Australia's largest cardiothoracic hospital, The Prince Charles Hospital, and regularly realises the huge potential of integrating technology and biology in combating cardiovascular disease. The group is a multi-disciplinary team consisting of clinicians, scientists, statisticians, allied health professionals and engineers. The CCRG aims to improve both the understanding and use of technologies to improve the outcomes of patients via organ transplantation and mechanical assist device (MAD) options, used by clinicians in the management and treatment of patients suffering from cardiovascular disease.

Since its establishment by Professor John Fraser in 2004, the CCRG has attracted more than \$28 million in grants and industry funding, published over 300 papers, and was awarded the first NHMRC Centre of Research Excellence for MADs. The CCRG also has active ongoing collaborations with The Alfred Hospital and St Vincent's Hospital, which, combined with the CCRG's base location of The Prince Charles Hospital gives the group a strong network across the three major cardiac hospitals in Australia. In addition, the group belongs to multiple national and international research networks, including ECMOnet, EuroELSO, APELSO, ANZICS amongst others. The group specialises in clinically validated large animal models, within a state of the art research facility. CCRG also has an extensive network of collaborators across UQ, and both within Australia and overseas.

Research Projects are available in a number of areas for honours, masters (research/coursework), and PhD students. Projects can range from animal experiments, to biological characterisation of tissue samples, through to translation of research findings to improve current clinical outcomes. Large animal, pre-clinical studies are a major focus of the CCRG, but through collaborations with other research scientists (at UQ and overseas), the group encourages multidisciplinary projects. Students will be expected to apply for research funding, and the group has a strong track-record in mentoring students to obtain novice grants successfully (up to \$10,000 each). Students will also gain valuable experience working with leading clinician researchers from major hospitals around Australia, as well as cutting edge researchers in medical engineering and biomedical science.

Currently available projects:

1. The Dead Heart Project – When is a dead heart truly dead?

Students will be involved in a multi-disciplinary team across the 3 biggest cardiac hospitals in Australia. The project aims to improve the quantity and quality of donor hearts through organ reconditioning and new donor sources, as well as understanding ischemia-induced molecular damage to cardiomyocytes during transplant. The model is clinically relevant with study transplants performed by leading Australian cardiothoracic surgeons.

2. Endotyping Acute Respiratory Distress Syndrome (ARDS)

ARDS is a critical illness with unacceptably high mortality rate (up to 45%). Currently, there is no effective treatment, partly due to its heterogeneity nature. Recent retrospective studies proposed the possibility of endotypes among ARDS patients. CCRG is exploring this topic with our established large animal model. The project is in collaboration with international partners, including Queen's University Belfast and UCSF. Students will have the opportunity for a short-term visit to our collaborator in the UK.

3. Impact of Extra-Corporeal Membrane Oxygenation (ECMO) on leukocytes

ECMO is a life saving device for patients with severe cardiac and/or respiratory dysfunction. It allows patients to rest in otherwise life-threatening situations. However, mortality remains high. This undesirable outcome is often associated with immune perturbation mediated by the contact of patient blood cells with the foreign surface of ECMO. This project aims to better understand the impact of ECMO on leukocyte fate.

4. Gastrointestinal bleeding caused by ventricular assist device (VAD)

Gastrointestinal bleeding is one of the most common complications in patients with continuous flow VAD (Ventricular Assist Devices - artificial mechanical hearts), but the exact cause is unknown. This project involves testing whole blood and endothelial responses to different flow conditions by connecting vascular models to commercially-available VADs (some of these may have been removed from patients.). Students will develop skills in cell culture, blood circuit assembly, flow cytometry and fluorescent microscopy

For more information of the group and available projects, please contact Dr. Jacky Suen (j.suen1@uq.edu.au).

Techniques/Knowledge you will learn in our group may include:

General: experimental design, good laboratory practise, ethics application, site specific approval application, good record keeping, pipetting and dilution technique

In vitro/ex vivo: SDS-PAGE, Western Blot, ELISA, RT-PCR, RNAseq, ddPCR, single cell PCR, immunofluorescence, flow cytometry, confocal microscopy, electronic microscopy (SEM, TEM), mitochondria profiling, oxygraph, organ bath.

In vivo: animal ethics application, animal handling, dissection, tissue/blood handling, blood differential, histology

Clinical/Human: electrocardiogram, extra-corporeal membrane oxygenation, echocardiogram, cardiopulmonary bypass, mechanical ventilation, rotational thromboelastometry (ROTEM), multiplate analysis, electrophysiology, arterial blood gas, ex vivo lung perfusion, patient recruitment process
