



THE UNIVERSITY
OF QUEENSLAND
AUSTRALIA

School of
Biomedical Sciences

HONOURS APPLICATION AND ENROLMENT GUIDE

2019

Bachelor of Sciences
(Hons) Biomedical
Sciences

Bachelor of Biomedical
Sciences (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 entry requirements. [BSc graduates](#), can check their eligibility to enrol in honours by reading the program rules and requirements [here](#). [BBiomSci \(Hons\)](#) students can check their eligibility to enrol in honours by reading the program rules and requirements [here](#). In some circumstances, and subject to the approval of the Head of School and Executive Dean, BSc and BBiomSci graduates 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
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.
4. [Return completed application](#) to the SBMS Honours administrator (Email: sbms.hons@uq.edu.au or Level 1, Macgregor Building (#64)).

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

Deadline: 7th December 2018 for commencement in Semester 1 2019
17th June 2019 for commencement in Semester 2 2019

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 programs:

- 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 or Bachelor of Biomedical Science. 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 fellowships 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 degree (or for the first three years of the BBiomedSc degree) is required. In some circumstances, and subject to the approval of the Head of School and Executive Dean, BSc graduates and BBiomedSc 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 7th December 2018

Start Date: First week in February 2019 (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 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

- **Journal Club:** This component involves the presentation and contribution to discussion of a research paper amongst your peers.
- **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 8000 words (maximum) describing and critically appraising the research work undertaken during the Honours year.
- **Seminar Diary:** Students will attend at least 12 seminars (these can be external to SBMS) given by academic/research staff, invited speakers or PhD students (M1 confirmation only).
- **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.

- Seminars are examined by two members of the SBMS Honours Committee or appropriate proxies.
- 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 and BBiomedSc 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>Journal</i> Journal Club Presentation and Participation	5%
<i>Seminar</i> Research Proposal Seminar	5%
<i>Report</i> Research Proposal	10%*
<i>Diary</i> Seminar Diary	Mandatory
<i>Report</i> Research Report	55%*
<i>Report</i> Supervisor's Report	5%
<i>Seminar</i> Final Research Seminar	20%

* submission of final version via Turnitin

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.

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/

The Critical Care Research Group - Prince Charles Hospital - <http://ccrg.org.au/>



Prof. Elizabeth Coulson

School of Biomedical Sciences
Neuroscience

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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 basalforebrain neurons (mouse studies: slice electrophysiology, mouse surgery for *in vivo* geneticmanipulation, 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 inneurodegenerative 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**

School of Biomedical Sciences
Neuroscience

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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

**Assoc. 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**

SBMS
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; 2016-2018) to investigate how neural stem cell quiescence in the adult brain is coordinated. This work will provide pivotal insights into how ongoing neurogenesis in the adult brain is regulated, and the behavioural consequences of deficits to this process. Dr. Piper also holds another grant 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

We are currently investigating the role of transcription factors in cerebellar development and disease. We have an Honours Project available for 2019 that will investigate how NFIX mediates stem cell differentiation within the postnatal cerebellum.

Techniques you will learn in our group may include:

Immunohistochemistry

Microscopy

qPCR

In situ hybridisation

Histology

**Dr Jana Vukovic**

SCHOOL OF BIOMEDICAL SCIENCES

Neuroscience

Cancer

Infection and Immunity

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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

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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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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**

School of Biomedical Science
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 co-incide 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
-



Associate Professor Rohan Teasdale

School of Biomedical Sciences
Neuroscience/Cancer and/or Cell Signalling

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Protein Trafficking in Disease

Protein trafficking controls the spatial organisation of individual proteins within cells. This highly co-ordinated movement of the thousands of distinct membrane proteins within cells is essential for the regulated localisation of proteins to the plasma membrane which in turn controls the organisation of cells within tissues and coordinates their communication with the environment. The success of this process depends on the regulated sorting and trafficking of proteins within the highly dynamic endosomal compartments of the cell in processes that are also emerging as important drivers of neurodegenerative disease, cancer and metabolic pathologies. An understanding of how endosomal traffic is regulated, and how lysosomal traffic and degradation are modulated, is critical for providing insights into the physiological processes associated with proteins that traffick through 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 a network of intracellular compartments that form the mammalian 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 thus 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**

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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***

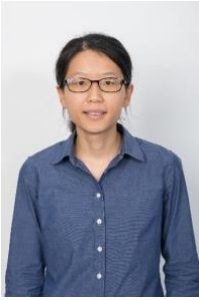
Mitochondria are essential organelles that control energy synthesis and cell signalling, as well as apoptosis. Due to their important function in cell metabolism, the quantity and quality of mitochondria must be regulated tightly, through mitochondrial fusion and fission, biogenesis, and degradation. 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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**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 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
-

**Dr Taylor Dick**

School of Biomedical Sciences

Musculoskeletal and motor control

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Neuromuscular Biomechanics Research Laboratory

Our research aims to understand the neuromuscular and biomechanical mechanisms that underlie healthy and diseased locomotor function. Through development of innovative imaging techniques to measure *in vivo* muscle and tendon properties, our research is advancing our understanding of the interactions between muscles and tendons. Integration of our data into clinical models is shaping our understanding of how neuromuscular properties are altered with ageing, obesity and disease.

Project Description 1

Shear wave elastography is an ultrasound-based technology capable of evaluating, in real-time, the mechanical properties of tissues, including muscle and tendon. Over the last decade, there has been growing evidence that shear wave elastography may be a useful tool in detecting subtle changes in musculotendinous mechanical properties that occur early in the course of an injury or disorder. Despite promising results, elastography has not yet earned its place, owing to insufficient information about its relationship with other gold standard methods used to study muscle-tendon properties.

In the proposed project, shear wave elastography will be compared with classical methods that assess the mechanical properties of the Achilles tendon by studying force-length and stress-strain relationships. The objective of the proposed project is to determine the relationship between the two experimental approaches by studying a diverse sample of participants, for example participants of normal weight and healthy obese participants or participants of varying age.

Project Description 2

Obesity is strongly linked with musculoskeletal impairments, such as osteoarthritis and tendinopathy, and is known to significantly contribute to a decline in physical function and quality of life in older adults. Indeed, obese but metabolically healthy adults have nearly six-times greater odds of worsening of bodily pain than normal weight adults who are similarly healthy (Bell et al., 2017).

The ankle is arguably the most critical joint for locomotion— providing up to 80% of the push-off power required to move the body from one step to the next (Neptune et al., 2009). Yet with disease and age, the calf muscles weaken and the Achilles tendon loses its stiffness (Onambele et al., 2006). This causes a reduction in ankle push-off power and initialises a cascade of unfavourable mechanical and sensory consequences that underpin mobility impairment. To date, we lack an understanding of how the structure and function of key locomotor muscle groups (ie: ankle plantarflexors and Achilles tendon) are altered with

obesity. This has, in part, limited our ability to design effective exercise and rehabilitation programs in this population. The objective of the proposed project(s) are to (1) determine the structural and mechanical changes in muscle-tendon properties that underpin obesity and its associated risk factors and (2) evaluate the effects of an exercise training regime known to induce muscle and tendon adaptation.

Techniques you will learn in our group may include: B-mode ultrasound and shear wave elastography (muscle- tendon length changes and material properties), electromyography (neural drive), force dynamometry (musculoskeletal forces), and kinematics (limb movements and joint angles).

Collaborators: This project will be a collaboration with co-advisor Dr Brooke Coombes, who is a Physiotherapist and Lecturer at Griffith University who specializes in the assessment and treatment of tendinopathy and Dr Kylie Tucker, who is a Senior Lecturer with expertise in motor control and pain within the School of Biomedical Sciences.



Dr Kylie Tucker

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Laboratory of Motor Control and Pain research

We aim to gain insight into the development of movement and postural control in children and adolescents, and determine the affect of acute pain and chronic injury on motor control parameters in children and adults. We use innovative methodologies to improve knowledge about muscle mechanical properties and the way we drive our muscles to produce force and movement.

Project Description

Idiopathic patellofemoral (PF) pain is the most common cause of knee pain in female adolescents, affecting 14-21% of this population. Despite being prescribed evidence-based treatments, less than half of adolescents recover after 12 months, and 1 in 3 still have pain at 5 year follow up. A leading model for the genesis and persistence of PF pain in adults is that altered muscle coordination of the vastus lateralis and vastus medialis (the two largest knee extensor muscles), fosters altered PF kinematics, which leads to suboptimal PF joint loading. In contrast, for adolescents, overuse (i.e. excessive loading of the PF joint structures) is thought to underpin pain development.

The potential for a force imbalance between vastus muscles to underlie altered PF kinematics and contribute to the development and persistence of PF pain, needs to be thoroughly tested in the adolescent population. Advances in this field will support the development of treatments that lead to higher recovery rates, and support adolescents progressing through to adulthood without knee pain limiting their physical, emotional, educational, and intellectual development.

Here we will determine if 2 commonly used rehabilitation interventions (a motor control and an orthodic intervention) are associated with an immediate change in the neural drive to the vastus lateralis and vastus medialis in adolescents and young adults with PFP.

Techniques you will learn in our group may include: Electromyography (neural drive), Force Plate (centre of pressure), Kinematics (joint angles), Shear-wave elastography (muscle stiffness).

Collaborators: This project is part of a larger study that involves multiple national and international collaborators. In particular this work will be completed with co-advisor Dr Natalie Collins, who is a physiotherapist and researcher within the School of Health and Rehabilitaiton Sciences at UQ.

<http://researchers.uq.edu.au/researcher/12040>



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Chen Lab - Endocrinology and metabolism/Long period insulin treatment improve healing of diabetic foot

In 2014, 422 million people lived with diabetes, a serious chronic disease. The prevalence doubled from 1980. People with diabetes have dramatically increased risk of amputation because of non-healing foot ulcer, so called diabetic foot. Local injection of insulin shows beneficial effects to diabetic foot through improved wound healing. As insulin is a protein hormone, it is quickly degraded *in vivo*. A system, which provides sustained insulin to the wound sites continuously for a long duration---a period that required for a complete wound healing, likely helps wound healing significantly in diabetes. For such purpose, insulin secreting living cells with controllable insulin releasing ability would be a potential strategy. In this project, we will develop such a system using mouse insulin secreting MIN6 cell line transfected by light-sensitive ion channels to control insulin secreting pattern. We will test if the supernatant of this MIN6 cell culture will be applicable to help wound healing using a HaCaT scratch *in vitro* wound healing model.

Project Description 1

MIN6 cell is an insulin secreting β cell line. Intracellular increase in calcium signalling alone induces insulin secretion from MIN6 cell.

Project Description 2

A light activatable calcium selective ion channel that opens during blue light exposure will be stably transfected into MIN6 cells, so that MIN6 cells will secrete insulin during light exposure.

Project Description 3

The supernatant of transfected MIN6 culture will be collected after cells are exposed to light, and this supernatant will be applied to HaCaT scratch *in vitro* wound healing model.

Techniques you will learn in our group may include: The project will include cell culture, optogenetics, transfection, calcium imaging, *in vitro* wound healing experiments, microscopy, and molecular biology.

Chen Lab - Understanding of the adipo-pancreatic axis: effects of fatty acid binding protein 4 on pancreatic β cells

Insulin resistance is an early hallmark of type 2 diabetes and is greatly associated with obesity. As a complex endocrine organ, adipose tissue plays an intricate role on insulin secretion from pancreatic β cells

and insulin sensitivity on central and peripheral tissues. This is thought to be mediated through adipocyte derived adipokines and cytokines. Although efforts have cast light on the crosstalk between adipose tissues and β cells, a full concept of the adipo-pancreatic axis remains vague with new emerging adipokines. The project aims to investigate the role of fatty acid binding protein 4 (FABP4), one of the most abundant intracellular lipid transport proteins produced by mature adipocytes and the key regulator of lipid trafficking, signalling, membrane synthesis and oxidation, on pancreatic β cells and its interaction with other adipokines such as leptin on insulin signalling both *in-vivo* and *in-vitro*.

Techniques you will learn in our group may include: The project will include molecular biology techniques including tissue 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, tissue dissection and fixation.



Dr Niwanthi Rajapakse

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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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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. Our lab has a particular focus on the development of the feto-maternal 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 1: 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 surface 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.

Project Description 2: Regulation of Trophoblast Giant Cell Differentiation

Within the mouse placenta there is a class of cells that is truly remarkable; trophoblast giant cells (TGCs). As the name suggests, these cells are enormous, growing to an impressive size by undergoing endoreduplication. Some TGC subtypes have DNA contents as high as 1024N, and are many times the size of normal diploid cells. TGCs line the whole of the maternal circulation of the placenta, and have important endocrine functions. However, our understanding of how TGCs differentiate from stem cell progenitors is limited.

This project will investigate the role of a newly identified transcription factor in TGC differentiation, using trophoblast stem cell cultures as a model system.

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

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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-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.



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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 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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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

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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



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.



Prof. Frederic A. Meunier

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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 area 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

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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.



Dr Anne-Sophie Bergot

FoM/UAQI

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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,



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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);



Dr Samantha Stehbens

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

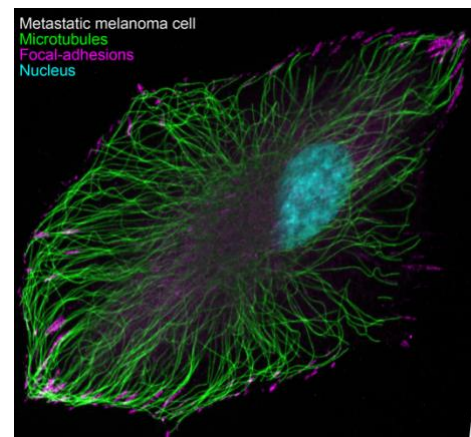
The majority of cancer deaths are due to metastatic disease, highlighting the need for ‘migrastatics’, therapeutics which act to inhibit migration. Metastatic success requires cells to navigate complex cellular environments, adapting either their shape to navigate between matrix fibres or adapting their 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 3Dimensional 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 3Dimensional 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.



A/Prof Katharina RONACHER

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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.

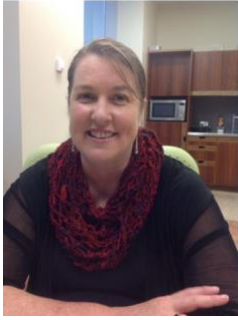
Project Description 1: Impact of ketone bodies on innate immunity to infection

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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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: