THE UNIVERSITY OF MELBOURNE

# DAP Research Projects 2027

# Welcome

The decision to undertake an Honours year or a Masters degree is an important one that adds real substance to your undergraduate degree. It gives you a close-up view of how a research laboratory works, and the chance to put your scientific knowledge into practice.

For a small minority, it will be the first step towards an independent research career. But the skills you learn along the way, critical thinking, experimental design, resilience, and the ability to communicate complex ideas clearly, are valuable well beyond the laboratory, and will serve you in whatever career you choose. An Honours or Masters qualification also sets you apart when you're seeking employment or applying for further study.

There is a lot to weigh up: the research topic itself, whether you want the additional coursework, the laboratory and its resources, your potential supervisor, and the support the department offers its students.

The Department of Anatomy and Physiology has a genuinely strong record of research training and mentorship. Our graduates go on to leadership roles across universities, institutes, industry and the private sector, and we are enormously proud of them. We've built a carefully structured coursework program to complement your growing laboratory and analytical skills, and our Honours and Masters students consistently tell us that the friendship, advice and mentoring they receive from fellow students is one of the best parts of the experience.

***This booklet provides information to help you decide on a potential research project in Anatomy and Physiology at Honours and Masters level, and perhaps beyond, to your PhD.***

Our research spans metabolic and cardiovascular sciences, muscle biology, sensory and systems neuroscience, stem cells, developmental biology, and learning and teaching. Take your time looking through the projects on offer. Identify the ones that appeal to you, and reach out to potential supervisors to ask questions and visit their laboratories. Talk to lab heads, staff and students about the projects and about your career options with the qualification, supervisors are genuinely glad to hear from you, so don't hesitate to make that approach.

I did my own Honours and PhD here at the University of Melbourne, and after 23 years leading a research group at the University of Queensland, I've returned to this department because of the calibre of research and the collaborations on offer across the Bioscience precinct. I can honestly say this is a wonderful department in which to do research. We offer a huge breadth of exciting opportunities, and we would love the chance to discuss them with you.

Professor Elizabeth Coulson
Head of Department of Anatomy and Physiology

# How to apply

## Honours

### What is Honours?

Honours is a fourth-year undergraduate course that consists of a combination of a research project and coursework subjects. The course is designed to develop the student's capacity to solve problems, to analyse data, to read and think critically, and to communicate clearly.

- Honours can give you a taste of what working as a scientist would be like as a career, allows you to demonstrate academic excellence in an area of special interest to you, and provides an entry point for further research higher degree study (i.e. PhD). These skills are highly sought after by employers in biological, medical and industrial areas.

### What are the entry requirements?

To be considered for entry, applicants must have completed a suitable undergraduate degree (Bachelor of Biomedicine, Bachelor of Science or equivalent) with a major in a relevant discipline with a WAM (weighted average mark) of at least H3 (65%) or equivalent. Students who have completed or are due to complete a Bachelor of Biomedicine at the University of Melbourne should apply to complete Biomedicine Honours. Students who have completed or are due to complete a Bachelor of Science at the University of Melbourne or an equivalent course at another institution should apply to complete Science Honours.

Meeting the minimum Faculty level is not a guarantee of admission and students must be accepted by a supervisor before entry into the course.

### How long is Honours?

Honours is a one-year course consisting of 75 points of research and 25 points of coursework, that commences mid-February and finish in November

### How to apply

#### Step 1: Contact Potential Supervisor(s)

Decide which departments, institutes, supervisors and projects you wish to apply for and make contact with the relevant supervisor. Applicants must contact potential supervisors either before or soon after submitting an online application for entry to an MDHS Honours course. Department and Institute Honours project booklets and websites, the individual information sessions held by departments and institutes are ways of helping you to make contact with potential Honours supervisors.

#### Step 2: Online Application

Lodge an online application

1. Apply online and select either the Returning Applicants, Current Students and Previous Students or First Time Applicants. Do not select the First Time Applicants option if you have previously completed study or applied to any program at the University of Melbourne.
2. Select 'MDHS Specialisations' as requirement response in the online application form.
3. Provide original or certified transcript(s) for any study not undertaken at the University of Melbourne. You are not required to provide transcripts for study undertaken at this university.

#### Step 3: Project Preference

Once you have submitted an online course application, you will receive an email within 3 working days with your personal login details to access the Honours Project Preference System - SONIA. Please follow the instruction in the email to set up your password and select your preferences for projects offered within MDHS departments. You may select up to 4 project preferences in Round 1 or 3 project preferences in Round 2, 3 and mid year. You must only preference projects after making contact with the relevant supervisor(s). You are allowed to log into Sonia to change your preferences any time by the closing date.

More information including application dates and online application link:
mdhs-study.unimelb.edu.au/degrees/honours/apply-now

## Master of Biomedical Science

### What is the Master of Biomedical Science?

- The Master of Biomedical Science at the University of Melbourne is a coursework master's degree incorporating a substantial research project. This course is an alternative to the Honours as a PhD pathway. Students undertake a major research project and discipline-specific coursework subjects. In addition, a suite of professional business and communication subjects are offered to complement and enhance the research undertaken and to progress students' career opportunities.

The course encourages students to think innovatively and provides an awareness of the health and economic benefits of biomedical research. Graduates of this course gain an understanding of the research process, specialist knowledge and professional skills that are attractive to employers.

### What are the entry requirements?

To be considered for entry, applicants must have completed a suitable undergraduate degree with a major in a relevant discipline with a WAM (weighted average mark) of at least H3 (65%) or equivalent. Meeting this requirement does not guarantee selection.

Note

- Quotas may be applied to the degree as a whole, or to individual disciplines, and preference may be given to applicants with evidence of appropriate preparation or potential to undertake research.
- Entry is subject to the capacity of a participating department to provide adequate supervision in a research project appropriate to the interests and preparation of the individual student and is subject to the agreement of an academic staff member to supervise the project.
- Students entering this course are expected to organise an academic supervisor in the relevant academic unit, and select a research project, as part of the application process. You will be provided with a list of current projects once your application has been assessed and deemed eligible. The theme and scope of the research project is negotiated between the student and supervisor prior to commencement of the course.

### How long is the Master of Biomedical Science?

The Master of Biomedical Science is a two year (full time) course consisting of 125 points of research and 75 points of coursework. The course can be commenced at the start of the year or at mid year.

### How to apply

1. Apply online and select either Current Students and Previous Students or First Time Applicants. Do not select the First Time Applicants option if you have previously completed study or applied to any program at the University of Melbourne.
2. Provide original or certified transcript(s) for any study not undertaken at the University of Melbourne.

### Selecting a Project

Once you have submitted an online course application, you will receive an email with your personal login details to access the Master of Biomedical Science Project Preference System - SONIA. Please follow the instruction in the email to set up your password and review projects offered within MDHS departments. You must make direct contact with the supervisor and obtain permission to work on their project before submitting your project preference. Once your project has been endorsed, you will be allocated to this project in SONIA.

More information including application dates and online application link:study.unimelb.edu.au/find/courses/graduate/master-of-biomedical-science/how-to-apply/

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## Difference between Honours and the Master of Biomedical Science

| | Honours | Master |
|---|---|---|
| Duration | 1 year (full time) | 2 years (full time), part time available |
| Level | Undergraduate | Graduate |
| CSP (commonwealth supported places) available? | Yes | Limited |
| PhD Scholarship scoring | Considers marks from 3rd year of Bachelor's degree and Honours marks | Only Masters marks are considered |
| International Market recognition | Australian Honours degrees may not be recognised overseas, as many countries do not have an equivalent degree. | Recognised as a graduate master's degree |

# How to apply

## Research Higher Degrees

### What is a PhD?

- A PhD (Doctor of Philosophy) is a 3-year supervised research degree with the possibility of up to 12 months extension. A candidate may be required to supplement their research with enrolment in additional subjects if considered necessary. The research is written up as a thesis (80,000 – 100,000 words) and examined by external experts in the field.

### What is a MPhil?

A MPhil (Master of Philosophy) is similar to a PhD but carried out over a shorter period of time of 18months to 2 years. The research work is written up as a thesis (30,000 – 40,000 words) which demonstrates your knowledge and contribution to the field of research.

### What are the entry requirements?

To be considered for entry into a PhD, applicants must have completed

- a four-year bachelor degree (BSc Hons, BBiomed Hons) in a relevant discipline which includes a substantial research component equivalent to at least 25% of one year full time study and achieved a minimum WAM of 80% (the University of Melbourne) or equivalent; or
- a masters degree in a relevant discipline which includes a substantial research component equivalent to at least 25% of one year of full time study and achieved a minimum weighted average of 80% or (the University of Melbourne) equivalent.

To be considered for entry into a MPhil, applicants must have completed

- a four-year bachelor degree (BSc Hons, BBiomed Hons) in a relevant discipline which includes a substantial research component equivalent to at least 25% of one year full time study and achieved a minimum WAM of 75% or higher; or
- a masters degree in a relevant discipline which includes a substantial research component equivalent to at least 25% of one year of full-time study and achieved a minimum weighted average of (University of Melbourne) 75% or higher.

### Choosing a supervisor and research area

A critical element of success is choosing a research area that interests you. Departmental websites have information on the range of research areas on offer, as well as areas of interest of academic staff members who can supervise your project.

It is very important for you to talk to supervisors as well as current or previous students. It is one thing to be interested in the project but you need to get along with your supervisor too. If possible, try to get some work experience in the lab to get an idea about the environment.

For future information regarding Research Higher Degrees: https://study.unimelb.edu.au/find/courses/graduate/doctor-of-philosophy-medicine-dentistry-and-health-sciences/ https://study.unimelb.edu.au/find/courses/graduate/master-of-philosophy-mdhs-biomedical-science/ https://biomedicalsciences.unimelb.edu.au/departments/anatomy-and-physiology

### How to apply

- Review the list of prospective projects and supervisors in this handbook or online at https://biomedicalsciences.unimelb.edu.au/departments/anatomy-and-physiology
- Identify projects of interest and contact the project supervisor to explain your research interests and provide your curriculum vitae (CV) and academic transcripts.
- Once you have confirmed a project and supervisor apply online at https://study.unimelb.edu.au/how-to-apply/graduate-research

## Scholarship

### Honours

Honours applicants who accept and enrol in an Honours course will automatically be considered for available Honours Scholarships. These are awarded on academic merit.

Highly ranked full-time students who have enrolled in an MDHS program through the Bachelor of Biomedicine (Degree with Honours) and the Bachelor of Science (Degree with Honours) and demonstrated a level of financial needs will automatically be considered for a Frances Elizabeth Thomson Trust Scholarship. The Scholarship will award eligible students with a one- off payment of $5,000. mdhs.unimelb.edu.au/study/scholarships/n/frances-elizabeth-thomson

### Honours & Masters

If you a third-year student currently enrolled in Bachelor of Biomedical Science or Bachelor of Science and are considering enrolling to do Honours or Masters within the Department of Anatomy and Physiology in 2027 you can apply for a Summer Research Studentship or Vacation Scholarship to work on a supervised research project. The studentship provides a small living allowance to enable you to work on a laboratory-based project for a period of 4 or more weeks over the summer break. The purpose of the Summer Research Studentships and Vacation Scholarships is to provide an opportunity for undergraduates to gain first-hand experience in research.

Over the summer break, up to four Studentships and Vacation Scholarships may be available to the best qualified candidates.

You will need to discuss a project with a potential supervisor before applying. Most supervisors listed in this booklet will be happy to discuss summer projects with you.

For application forms and information, Please check out the following pages for further information: mdhs.unimelb.edu.au/study/scholarships/n/vacation-scholarship mdhs.unimelb.edu.au/study/scholarships/n/r.d.-wright-prize-and-scholarship or Biomedical Science Academic Services (biomedsci-academicservices@unimelb.edu.au) for further information.

### Graduate degrees

The Melbourne Scholarships Program is one of the most generous and comprehensive in Australia, with a wide range of scholarships available for domestic and international students. There are many different types of scholarships available, with some varying in value, duration and eligibility. Most University of Melbourne graduate students have scholarships to aid with living expenses and course fees. Some scholarships also assist with relocation fees and insurance costs whilst studying at the University of Melbourne.

Graduate Research Scholarships for domestic and international students are awarded on a competitive basis. If successful, students must also meet the entry requirements for a Doctoral degree at the University of Melbourne. More details on the different types of scholarships available, what they cover and eligibility can be found here:scholarships.unimelb.edu.au/awards/graduate-research-scholarships.

# Contact

## Honours Coordinators

Email: DAP-Honours@unimelb.edu.au

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## Masters Coordinators

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## Graduate Research Coordinator

Prof. Erica Fletcher
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MEDIflex
X-SMALL

# Project Index

## Neuroscience

# Metabolic & Cardiovascular Sciences



# Scholarship of Teaching & Learning (SOTL)



# Muscle Biology

# Stem Cell & Developmental Biology



# Orthopaedic Surgery & Applied Anatomy

# Neuroscience

# Belaidi Group

**Contact:** Dr Abdel Belaidi
**Email:** abdel.belaidi@unimelb.edu.au
**Location:** Florey Institute of Neuroscience and Mental Health

Our research group aims to develop strategies to preserve brain health by pinpointing and targeting the earliest molecular triggers of neuronal death. Our work bridges rare genetic/metabolic disorders (e.g. molybdenum cofactor deficiency, sulphite toxicity) and common neurodegenerative diseases, investigating how pathways such as apolipoprotein E (APOE) signalling, lipid and iron metabolism, and cofactor homeostasis shape vulnerability or resilience in the brain.
We combine mechanistic neuroscience, biomarker discovery, and translational therapeutics. Our ambitions extend from fundamental biochemistry to preclinical development, with particular emphasis on mRNA-based therapies targeting APOE and other molecular targets.

**Apolipoprotein E-dependent lipid trafficking as a regulator of neuronal proteostasis and tau aggregation**

Apolipoprotein E (apoE) is a central regulator of lipid transport in the brain and a major genetic determinant of Alzheimer's disease risk. While apoE isoforms (apoE2, apoE3, apoE4) are known to differentially influence tau aggregation and neurodegeneration, the mechanistic link between lipid delivery, vesicle trafficking, and protein aggregation remains poorly defined.

This project will investigate how apoE isoforms and their lipidation state control neuronal proteostasis through vesicular trafficking pathways, with a focus on tau handling. The central hypothesis is that apoE-dependent lipid import modulates endosomal–lysosomal function and extracellular vesicle dynamics, thereby regulating the balance between tau degradation, aggregation, and intercellular spread.

**Primary Supervisor:** Dr Abdel Ali Belaidi
**Co-Supervisor:** TBC
**Availability:** PhD;MC-BMEDSC ;Honours

# Fletcher group

**Contact:** Professor Erica Fletcher
**Email:** e.fletcher@unimelb.edu.au
**Location:** Medical Building

The Visual Neuroscience laboratory is interested in understanding the structure and function of the retina. We study two broad classes of retinal disease:

1. Retinal degenerations, including Age-Related Macular degeneration
Retinal degenerations are a family of hereditary diseases that cause a gradual loss of photoreceptors leading to blindness. These diseases occur in around 1:5500 people, but 1:50 are carriers. We are examining the mechanisms of photoreceptor death and whether specific treatments ameliorate or slow the loss of photoreceptors. Understanding how photoreceptors die is of relevance to diseases such as Age-Related Macular Degeneration (AMD), which is one of the leading causes of blindness especially in older people. Photoreceptor death and abnormal growth of blood vessels into the retina are major contributors to blindness in this disease. We use pre-clinical models to understand the underlying mechanisms involved and whether treatments slow the progression of disease. Our ultimate goal is to develop ways of slowing photoreceptor death and also to investigate ways of replacing lost photoreceptors.
2. Retinal vascular diseases, including Diabetic retinopathy
Diabetes affects 3.8% of the population of Australia, at an annual cost of AUS$1 billion dollars. Diabetic retinopathy is a common complication associated with diabetes and is the leading cause of blindness in those under 65 years of age. Approximately 10% of all diabetics experience vision threatening retinopathy. One of the major reasons for vision loss is the growth of new blood vessels in the retina (neovascularization). Although a great deal of attention has focussed on the vascular changes associated with diabetes, it is now emerging that changes in neuronal, glial and microglial cell function often occur prior to overt vascular abnormalities. Understanding the link between microglial, glial cell dysfunction and changes in vasculature is vital for gaining a better understanding of the pathogenesis of diabetic retinopathy. The major thrust of our work is understanding the changes in the retina that lead to neovascularization. Ultimately, we seek to identify and implement novel treatments for retinal vascular disease so that we can prevent or slow vision loss .

**Pharmacological and laser therapies for age related macular degeneration**

Age related macular degeneration (AMD) is a major cause of vision loss in the older community. There are currently no specific treatments for preventing late stage AMD or slowing the progression of the disease to the later vision threatening forms. In this project we will characterise morphological and functional changes in the eye of a pre-clinical model of AMD and test novel pharmacological and laser therapies to ameliorate these changes. This project will involve the use of wide ranging techniques such as assessment of visual function, immunohistochemistry and molecular biology. Ultimately, this study will help to answer whether novel pharmacological or laser therapies can be used as a preventative treatment for AMD.

**Primary Supervisor:** Professor Erica Fletcher
**Co-Supervisor:** Una Greferath
**Availability:** PhD;MC-BMEDSC ;Honours

**Microglial-induced mechanisms involved in diabetic retinopathy and macular oedema**

The retina is highly susceptible to damage arising from the high glucose concentrations present during diabetes. Individuals with type I and II diabetes often develop retinopathy (a vascular pathology) and oedema (fluid-induced swelling). Both these pathologies lead to the development of potentially blinding conditions. The development of retinopathy and oedema results from changes in neurons, glia, microglia and blood vessels. In preclinical models, this project will use immunohistochemistry, molecular biology and in vivo imaging techniques to examine the changes in retinal microglia and how this relates to altered retinal vascular response and swelling of particular glial cells. Understanding these microglial-induced changes is critical to explaining the retinal pathology that develops during diabetes.

**Primary Supervisor:** Professor Erica Fletcher
**Co-Supervisor:** Dr Andrew Jobling
**Availability:** PhD;MC-BMEDSC ;Honours

# Hannan Group

**Contact:** Professor Anthony Hannan
**Email:** anthony.hannan@florey.edu.au
**Location:** Florey Institute of Neuroscience and Mental Health

The Epigenetics and Neural Plasticity Laboratory at the Florey Institute of Neuroscience and Mental Health. We explore how genes and the environment combine via experience-dependent plasticity in the healthy and diseased brain. Our research includes models of specific neurological and psychiatric disorders which involve cognitive and affective dysfunction, investigated at behavioural, cellular and molecular levels so as to identify pathogenic mechanisms and novel therapeutic targets. Most recently, this has included studies of intergenerational and transgenerational epigenetic inheritance.

We investigate how genetic and environmental factors combine to cause specific cognitive and affective disorders, including Huntington's disease, dementia, depression, anxiety disorders, schizophrenia and autism spectrum disorders. Our research also links data at behavioural and cognitive levels to underlying cellular and molecular mechanisms. We use a variety of behavioural tools, including automated touchscreen testing of cognition and high-throughput data analysis of vocalization and communication, that are directly translatable to clinical tests. We are establishing the extent to which experience-dependent plasticity can modulate behavioural and cognitive endophenotypes, in models with targeted genome editing. This cellular level of understanding is linked, in turn, to molecular mechanisms, including epigenetics, transcriptomics, proteomics and metabolomics. We are also exploring the concept of 'enviromimetics', therapeutics that mimic or enhance the beneficial effects of cognitive stimulation and physical exercise.

**Investigating multigenerational effects of paternal immune activation (PIA) on brain function**

Brain disorders account for a significant global burden of disease, yet the "missing heritability" in common brain disorders like autism, schizophrenia, depression, and anxiety suggests the involvement of environmental factors, including paternal exposures before conception. This project aims to explore the impact of paternal pathogenic infections on epigenetic inheritance and offspring phenotypes, as part of a paradigm shift in understanding the intergenerational effects of pathogens on public health.

Recent research has revealed the potential of pathogens to influence paternal germ cell epigenetics and modify offspring phenotypes, highlighting the urgent need to investigate the intergenerational impacts of paternal infection on brain development, behavior, and cognition. This is particularly relevant in the context of the ongoing COVID-19 pandemic.

While maternal immune activation's impact on offspring brain function has been studied, the influence of immune activation in the male germline and subsequent offspring phenotypes remains largely unexplored. We will investigate the mechanisms leading to heritable epigenetic changes in sperm due to paternal immune activation and examine their impact on offspring.

By utilizing mimics of infection and inflammation to induce immune responses in fathers, we will evaluate the infection-mediated immune response as an epigenetic modulator of offspring brain and immune system functioning. Through a combination of behavioral tasks, cellular/molecular approaches, and state-of-the-art epigenetic analyses, we aim to gain a comprehensive understanding of the offspring's endophenotypes and elucidate the modulation of the sperm epigenome and its consequences on offspring physiology, metabolism and behaviour. This research may uncover previously unknown interactions between infectious agents and their hosts, shedding light on the intergenerational effects of paternal infection and informing future public health strategies.
**Primary Supervisor:** Professor Anthony Hannan
**Availability:** PhD;MC-BMEDSC ;Honours

**Cognitive, social and psychiatric behavioural traits and their pharmacological treatment in a mouse model of neurofibromatosis type I**

Patients suffering from neurofibromatosis type 1 have a mutation in one allele of the neurofibromin 1 (NF1) gene, leading to a variable clinical presentation which may include the formation of tumours, hyperpigmentation marks, vision disorders and scoliosis. However, patients also typically show a range of neuropsychological symptoms including cognitive impairments and learning difficulties, attention deficits, as well as autism and depression. Despite the profound impact on the lives of patients and their families, these symptom complexes remain inadequately addressed by current treatments.
Nf1 +/- mice used in this project are a widely used model for neurofibromatosis type 1, though the study of cognitive and psychiatric behavioural traits has been limited to date and warrants further investigation.
The initial approach will involve the phenotypic characterisation of this mouse model using a broad battery of state of the art rodent behavioural tests, with the aim of examining specific aspects of cognition and attention, social function, as well as anxiety-like and depression-like behaviour. This will include the use of rodent touchscreen tests, which are directly translatable to human patients. In parallel, biochemical and molecular techniques will be used to investigate the brain mechanisms underlying the behavioural deficits observed in these mice. This will be followed by the application of promising drug candidates in a preclinical study using the Nf1 +/- mouse line, with the aim of progressing successful candidates to a clinical study in the near future with our clinical collaborators in Parkville.

**Primary Supervisor:** Prof Anthony Hannan
**Co-Supervisor:** Dr Sonali Reisinger
**Availability:** MC-BMEDSC ;Honours;PhD

**Investigating molecular mechanisms of viral paternal immune activation on offspring brain and behaviour**

Brain disorders such as autism spectrum disorder, schizophrenia, depression, and anxiety contribute significantly to the global burden of disease. However, despite substantial progress in genetic research, much of the heritability of these complex conditions remains unexplained. This gap points to the important role of environmental factors, including paternal and maternal exposures prior to conception, in shaping offspring neurodevelopment and risk of psychiatric illness.
This project addresses a critical question: How do viral infections in fathers affect the germline and, in turn, influence the health and behaviour of their offspring? We propose to investigate the role of viral-like paternal immune activation as a driver of heritable epigenetic changes that impact offspring brain development, behaviour, and physiology. This represents a fundamental shift in how we understand the intergenerational consequences of infection, particularly in light of global public health challenges such as the recent COVID-19 pandemic.
We have developed a mouse model of viral paternal immune activation using the viral mimic Poly I:C, where offspring of exposed fathers show significant behavioural alterations and brain developmental abnormalities. Offspring of immune-activated fathers are evaluated using a comprehensive battery of behavioural assays to assess cognition, affect, and social behaviour. In parallel, we will characterise cellular and molecular phenotypes in the brain and immune system, focusing on known pathways disrupted in neurodevelopmental and psychiatric disorders. Advanced statistical analyses will be integrated with behavioural and physiological readouts to identify potential mechanisms linking paternal infection to offspring phenotype.
This research has the potential to uncover a novel dimension of infectious disease impact—one that extends beyond the exposed individual to influence the health of the next generation.

**Primary Supervisor:** Prof Anthony Hannan
**Co-Supervisor:** Dr Sonali Reisinger
**Availability:** PhD;MC-BMEDSC ;Honours

# Mo Group

**Contact:** Dr Christina Mo
**Email:** christina.mo@florey.edu.au
**Location:** Florey Institute of Neuroscience and Mental Health

We are interested in how neural circuits underlie our perception, decision-making, and cognitive processes. We investigate this by live imaging neurons within specific circuits between the cortex and the thalamus while mice make sensory decisions. We also casually manipulate the circuits to see how this affects the behavioural performance and and neuronal activity. Computational analysis of population and single cell data help tease neural how electrical activity gives rise to our experiences.

**Identifying and manipulating the neural circuits of decision-making**

Making choices is a part of daily life yet one of the most challenging but fascinating questions in neuroscience is the neural basis of decision-making. Decision-making arises from activity between cortical areas but this communication also occurs via higher order thalamus. These cortico-thalamo-cortical pathways ubiquitously parallel direct corticocortical pathways in the brain, yet their role in perception has largely been ignored. Our previous work has shown that the transthalamic pathway in the somatosensory system is an essential neural pathway for perceptual decisions (Mo et al., Nat Comms, 2024). This project delves deeper into the role of transthalamic pathways in perceptual choice by combining trans-synaptic tracing (rabies virus), live calcium imaging (miniscopes) and controlled silencing or activation of neural circuits (optogenetics) during a whisker-based perceptual task. Students with experience in mouse behaviour, coding or an interest in neural circuits are encouraged to apply.

**Primary Supervisor:** Dr Christina Mo
**Availability:** PhD; MC-BMEDSC; Honours

# Vaughan Group

**Contact:** Dr David Noel Vaughan
**Email:** david.vaughan@florey.edu.au
**Location:** Florey Austin

The Vaughan research group, based at the Heidelberg campus of the Florey Institute at Austin Health, is dedicated to advancing the diagnosis and treatment of epilepsy through innovative brain imaging. We specialise in the clinical translation of advanced neuroimaging methods to understand where seizures begin, how they spread, and how best to guide therapy.

Our research spans the full spectrum of epilepsy, from the first seizure through to chronic disease that requires surgical intervention. A major focus is on people whose MRI scans appear “normal” using standard clinical techniques. We apply cutting-edge methods—including functional MRI, diffusion MRI, quantitative susceptibility mapping, and fixel-based analysis—to reveal subtle structural abnormalities and network dysfunction. Our work has helped identify imaging-based epilepsy subtypes and uncovered network-level changes, such as in MRI-negative temporal lobe epilepsy.

We are leaders in the use of simultaneous EEG-fMRI, a technique that combines brainwave recording with functional imaging to map the brain regions responsible for epileptic discharges. Our lab has pioneered methods for modelling these networks and identifying seizure-generating regions, including deep and non-lesional targets. We work closely with neurologists and neurosurgeons to validate our findings using stereo-EEG, an invasive technique for localising seizures and delivering targeted lesions through radiofrequency ablation.

We have strong expertise in quantitative and structural MRI, particularly through our leadership in the Australian Epilepsy Project. Our team developed the imaging protocol that is now used nationally for this study, and analysed imaging data from over 1,200 participants across multiple centres. We use advanced techniques to detect focal lesions that may cause epilepsy—especially when conventional imaging is inconclusive—and to characterise how epilepsy affects the brain more broadly. We also investigate how epilepsy affects cognition and mental health, using imaging to explore the basis of symptoms such as memory impairment, depression, and psychosis of epilepsy. Our recent work also explores how anti-seizure medications influence brain network organisation.

Our lab offers a rich, multidisciplinary environment where students gain experience in neuroimaging analysis, clinical neuroscience, and collaborative translational research. We welcome students who are passionate about improving care for people with epilepsy and contributing to the future of personalised neurological medicine.

**Where should the Radiologist Look? Predicting the Brain Location of Epilepsy-causing Lesions**

Detecting the brain lesion that causes epilepsy in a particular person is critical to diagnosis and targeting of curative surgery. Yet many lesions are small and easily missed, and effective detection can depend on knowing which brain region to scrutinise. Detection rates rise when an epilepsy neurologist can use clinical clues to offer a strong localizing hypothesis. This project aims to reproduce that expertise computationally, pointing radiologists to brain regions most likely to harbour an epilepsy-causing lesion before they review the MRI.
You will develop and train a machine learning classifier to predict lesion location from clinical and EEG features. Using retrospective well-characterised MRI and clinical data, you will define lesion locations and generate predictive features from clinical text documents such as neurologist and EEG reports. This will include developing methodology to select the neurologist's account of seizures, and extract their characteristics, using AI-assisted text processing. You may also build a literature-derived database linking seizure features to localisation, to generate transferable pre-training localization heuristics. The initial goal is for laterality and lobe-level prediction with probabilities, with scope to refine toward gyrus-level resolution or a whole-brain probability map.
Training data will be drawn from the Australian Epilepsy Project (over 1,200 people with epilepsy), a national large-scale study that has collected high-quality research imaging data. Specifically, we will select participants with a single small lesion (<2-3 cm) such as focal cortical dysplasia, low-grade tumour, or cavernoma, as typical representative examples of hard-to-find pathologies. An independent validation cohort will be drawn from the Austin Health First Seizure Clinic database. The long-term goal is to deliver pre-reporting predictions within the radiology workflow, and evaluate the impact of this tool.
You will gain skills in brain neuroanatomy, epilepsy pathology, machine learning, and AI-assisted medical text processing. The project suits students enthusiastic about computational research; familiarity with, or strong motivation to learn, Python/R and machine learning skills is important.

**Primary Supervisor:** Dr David Noel Vaughan
**Availability:** PhD;MC-BMEDSC

**Metabolic Signatures of Epilepsy-causing Brain Lesions**

Structural MRI shows where epilepsy-causing lesions sit, but says little about the disturbed biochemistry within them. This project asks what the metabolism of epileptogenic tissue can reveal, and conversely, what are the metabolic biomarkers of seizure-generating brain regions.
You will work with Magnetic Resonance Spectroscopic Imaging (MRSI) data acquired at ultra-high-field 7T, part of a larger program studying focal epilepsy. Unlike conventional single-voxel spectroscopy, which averages one large region, MRSI is a new technique to map metabolic spectra across the whole brain at 3.4 mm isotropic resolution. For the first time we can assess metabolites in regions within, adjacent and contralateral to irregularly-shaped brain pathology. Glutamate and glutamine (excitatory neurotransmitters), N-acetyl-aspartate (a marker of neuronal integrity), and choline and myo-inositol (markers of gliosis and membrane turnover) are of particular interest.
The core design is within-subject: comparing metabolites inside and adjacent to each lesion against the contralateral, normal-appearing brain. Across roughly 20 participants with focal cortical dysplasia, low-grade tumours, or vascular lesions, you will explore how these signatures differ, and relate metabolic changes to both lesion characterics and features of each persons' epilepsy (e.g. seizure types and frequency).
Day to day, you will process the MRSI data using Python, MATLAB, FSL, and LCModel to identify the target pathology, extract and analyse metabolite concentrations. There are also opportunities to assist with participant recruitment, scanner bookings, and data acquisition.
You will gain skills in MRI image processing, MR spectroscopy analysis, brain anatomy, and epilepsy neuropathology. The project suits students enthusiastic about hands-on imaging analysis and keen to learn these computational methods.

**Primary Supervisor:** Dr David Noel Vaughan
**Availability:** PhD;MC-BMEDSC

# Scott Group

**Contact:** Professor Ethan Scott
**Email:** ethan.scott@unimelb.edu.au
**Location:** Medical Building

A fundamental challenge to understanding the brain is its complexity: its genetic and developmental programs, its neurons and connections, its balance of permanence and plasticity, and the nuanced information flow through its networks. Across biology, emerging technologies are revolutionising the scope and scale at which we can address such questions. Our group has developed technologies for studying brain-wide sensory networks using calcium imaging and house-built light-sheet microscopes. Because zebrafish larvae are small and transparent, we can image tens of thousands of neurons, simultaneously and individually, as animals perceive and respond to sensory stimuli. We have used this approach to produce the first functional maps, brain-wide at cellular resolution, for auditory, vestibular, and water flow perception in any vertebrate.

**Brain-wide networks for perception and learning**

While our calcium imaging studies have taught us where and when neurons are active, they have not shown us how or why. In our available projects, we are interested in using a diverse array of methods, including microscope engineering, calcium imaging, optogenetics, mathematical modelling of brain-wide networks, and behavioural analysis to better understand how information flows thorough the brain during perception, and how changes in information flow encode memories. Applications from people with interest or experience in any of these approaches are welcome to apply.

**Primary Supervisor:** Professor Ethan Scott
**Co-Supervisor:** Dr. Conrad Lee
**Availability:** PhD;MC-BMEDSC ;Honours

# Dodd Group

**Contact:** Prof Garron Dodd
**Email:** garron.dodd@unimelb.edu.au
**Location:** Medical Building

Our lab explores innovative strategies to treat glioblastoma, the most aggressive form of brain cancer. We use cutting-edge tools in molecular biology, neuroimmunology, and drug delivery to uncover new therapeutic targets. We're seeking motivated students eager to advance brain cancer research and drive meaningful clinical impact.

**The Next Frontier in Brain Cancer Therapy**

Glioblastoma is the most aggressive and lethal form of brain cancer. Despite decades of research, survival is still measured in months and treatment options remain painfully limited. One of the biggest reasons is that these tumours build their own defences. Our lab has discovered that glioblastoma surrounds itself with a unique extracellular matrix, a dense molecular scaffold that fuels tumour growth, drives recurrence, and acts as a physical shield that stops drugs from ever reaching the cancer.
In this project you will help dismantle that shield. You will develop and test a new generation of inhibitors designed to break down this matrix in advanced pre-clinical models, with the goal of making glioblastoma vulnerable to treatment for the first time. Along the way you will gain hands on expertise in molecular biology, neuroimmunology, drug delivery, and advanced imaging. This work sits at the heart of an active drug development program with real commercial potential, and you will see first hand how a laboratory discovery is built into intellectual property and moved toward the clinic.
This is bold, translational science aimed squarely at a disease that desperately needs new ideas. Every experiment moves us a step closer to a therapy that could change outcomes for patients. We are looking for curious, ambitious students ready to gain cutting edge skills and make a real impact at the intersection of neuroscience and cancer research.

**Primary Supervisor:** Prof Garron Dodd
**Co-Supervisor:** Cait Beddows
**Availability:** PhD;MC-BMEDSC

# Ivanusic Group

**Contact:** Professor Jason Ivanusic
**Email:** j.ivanusic@unimelb.edu.au
**Location:** Medical Building

Neurobiology of pain

**Pathogenesis of skeletal pain**

Skeletal pain is transmitted by two classes of peripheral nociceptors. Aδ nociceptors are medium-diameter myelinated neurons that transmit fast, intense pain, of the sort experienced in fracture and breakthrough cancer pain. C nociceptors are small-diameter unmyelinated neurons that encode slow, burning pain, of the sort experienced in cancer and osteoarthritis. A number of different ion channels and receptors are emerging as important modulators of the activity of peripheral bone nociceptors. Identifying these regulators of nerve activity and better understanding their role in generation of bone pain could open up avenues for development of tools to selectively manipulate pain originating from bone. In this project, we will use a variety of techniques and animal models to explore roles for different sensory neuronal subtypes, ion channels and receptors in generating and/or maintaining skeletal pain. We are currently interested in modelling experimental inflammation of the bone marrow, osteoarthritis and bone cancer induced skeletal pain.
Students can expect to gain experience in working with one or more animals models of skeletal pathology, an in vivo electrophysiological bone-nerve preparation, neuroanatomical tracing and immunohistochemistry, tissue clearing and light sheet microscopy for 3D visualisation of nerves in bone, small animal handing, anaesthesia, surgery and/or dissection.

**Primary Supervisor:** Professor Jason Ivanusic
**Co-Supervisor:** Dr John-Paul Fuller-Jackson
**Availability:** PhD;MC-BMEDSC ;Honours

# Crouch Group

**Contact:** Professor Peter Crouch
**Email:** pjcrouch@unimelb.edu.au
**Location:** Medical Building

Prof. Crouch's team investigates neurodegenerative diseases of the central nervous system such as motor neuron disease and multiple sclerosis. Our primary interests are elucidating the cellular mechanisms that lead to neuronal death and assessment of putative neuroprotective therapeutic interventions. We utilise models of disease that span in vitro and in vivo paradigms and we also examine human, disease-affected central nervous system tissue.

**Investigating fundamental causes of neurodegeneration and assessing putative therapeutic interventions**

There is a dearth of effective treatments for neurodegenerative diseases due to an incomplete understanding of disease biology and the underlying processes that lead to neuronal death. To address this, we seek to elucidate disease processes and assess potential neuroprotective therapeutic interventions with a focus on motor neuron disease and multiple sclerosis. Our in vitro paradigms include patient-derived neural cultures and cells obtained directly from mice, with particular interest in the role of the normally supportive glial cells, microglia and astrocytes, investigating how they become disrupted in disease in order to reveal novel therapeutic targets. This cellular work is complemented by in vivo investigations utilising robust transgenic and induced mouse models of neurodegenerative diseases to explore disease processes and assess putative therapeutic interventions in the complete animal. The relevance of these discoveries to neurodegenerative disease is validated by assessment of human disease-affected tissue. Projects utilize methodologies encompassing cell culture, treatment and phenotypic assessment of animals, established biochemical methods (qPCR, western blot, immunolabelling, histochemistry), and more advanced approaches including transcriptomics, proteomics and elemental analyses.

**Primary Supervisor:** Professor Peter Crouch
**Co-Supervisor:** Dr Jeffrey Liddell
**Availability:** PhD;MC-BMEDSC ;Honours

# Furness Group

**Contact:** Dr Myat Noe (Cherish) Han
**Email:** cherish.han@unimelb.edu.au
**Location:** Medical building

Our research group investigates the structure and function of the enteric nervous system along the gastrointestinal tract, focusing on gut-brain communication circuits and the pathways controlling gut function. By studying these complex neural networks and their interactions, we aim to develop a deeper understanding of gastrointestinal disorders associated with many conditions. One of our ultimate goals is to translate this knowledge into novel therapeutic approaches that can improve the quality of life for patients suffering from these debilitating symptoms.

**Development of treatments of constipation in Parkinson's disease**

Constipation is a common, debilitating condition that significantly impacts the quality of life for Parkinson's disease patients, and current treatments often provide inadequate management. The study builds on our discovery that centrally penetrant ghrelin receptor agonists, when administered systemically or orally, induce defecation in both laboratory animals and humans. In this research, you will learn to record and analyse the physiological effects of potential therapeutic compounds on rodent defecation. You'll develop skills in quantifying and interpreting physiological data to assess the compounds' therapeutic potential. This research aims to develop novel therapeutic approaches for chronic constipation in Parkinson's disease, potentially advancing both gastrointestinal pharmacology and patient care.

**Primary Supervisor:** Dr Myat Noe (Cherish) Han
**Co-Supervisor:** Dr Pratik Thakkar
**Availability:** PhD;MC-BMEDSC ;Honours

**Gut-Brain axis and mood regulation**

Early studies, dating back decades, and traditional references to 'gut feelings', indicate that the state of the digestive system influences mood and emotion. Vagal nerve stimulation (VNS) can reduce depression and anxiety and be beneficial for people with Post-Traumatic Stress Disorder (PTSD). In this study we are investigating the mechanisms through which the gut communicates with the brain to influence emotion.

**Primary Supervisor:** Dr Pratik Thakkar
**Co-Supervisor:** Dr Myat Noe (Cherish) Han
**Availability:** PhD;MC-BMEDSC ;Honours

**Gut barrier integrity in 6-OHDA mouse model**

Patients with Parkinson's disease often suffer from an array of debilitating gastrointestinal dysfunction like constipation. These symptoms are contributed to by a compromised gut barrier called the "leaky gut". This project aims to investigate the gut barrier integrity in a 'brain-first' model of Parkinson's disease. Techniques may include tissue histology, immunofluorescence, molecular assays and using chamber studies to localise and assess tight junction proteins and inflammatory markers profile.

**Primary Supervisor:** Dr Myat Noe (Cherish) Han
**Co-Supervisor:** Dr Rachel McQuade
**Availability:** PhD;MC-BMEDSC ;Honours

**Characterisation of mouse stomach innervation**

One of the major problems impeding our understanding of gastric function is incomplete knowledge of the nerve circuitry, both within the gastric wall (the gastric enteric nervous system; ENS) and the central connections (gut-brain axis). It is crucial to better understand the organisation of the controlling nerve circuits so that we can make progress in developing treatments for upper gastrointestinal (GI) disorders such as gastroparesis, achalasia and gastric problems in diseases such as Parkinson's. We have made considerable progress on aspects of this work in the last 4-5 years.

The project aims to resolve a major current problem, which is to relate functional identities of enteric neurons (determined using a range of structural and functional investigations) with their molecular identities determined primarily by RNAseq.
In this project, we intend to use immunohistochemical studies, including super-resolution confocal microscopy, to define all gastric neurons and their connections. This involves multi-colour fluorescence visualisation to be able to locate nerve cells and their innervating synapses. To determine where nerve fibres come from, we will use surgical lesions (that remove incoming nerve fibres) and dye tracing.

**Primary Supervisor:** Prof John Furness
**Co-Supervisor:** Dr Myat Noe (Cherish) Han
**Availability:** PhD;MC-BMEDSC ;Honours

# Stamp & Hao Group

**Contact:** Dr Lincon Stamp
**Email:** lstamp@unimelb.edu.au
**Location:** Medical Building

Proper development and function of the digestive tract is crucial for good health. Gut function relies on the co-ordinated activity of neural circuits in the enteric nervous system (ENS), a network of neurons and glia located within the wall of the gastrointestinal tract. Our lab focuses on investigating the plasticity of the enteric nervous system and the development of stem cell therapy to treat digestive diseases.

**Examining the impact of environmental toxins on gut health**

Environmental pollutants, such as microplastics and per- and polyfluoroalkyl substances (PFAS), have become a growing concern due to their widespread presence and potential adverse effects on human health. The gastrointestinal tract is a primary route of exposure to these pollutants, and emerging evidence suggests that they may disrupt gut homeostasis and contribute to the development of various gastrointestinal disorders. This project aims to investigate the impact of microplastics and PFAS on gut health using a combination of in vivo and in vitro approaches. Using a mouse model of chronic exposure, the effects of these pollutants on gut function, intestinal barrier integrity, and gut microbiome composition will be investigated. Concurrently, high-throughput screening assays with intestinal epithelial cell lines and organoid models will be developed to identify cellular pathways and mechanisms underlying the potential gut-disrupting effects of microplastics and PFAS, employing methods like cytotoxicity assays, transcriptomics, and proteomics. This comprehensive approach aims to elucidate the functional consequences and cellular mechanisms underlying the impacts of these pollutants on health, contributing to preventive measures, and potential therapeutic interventions for associated gastrointestinal disorders.

**Primary Supervisor:** Dr Lincon Stamp
**Co-Supervisor:** Dr Madeleine Di Natale
**Availability:** PhD;MC-BMEDSC

**A gut feeling about new therapies for brain cancer treatment**

Gliomas are a very aggressive form of brain cancer, with a very poor 5-year survival rate. Gliomas can arise from over-proliferation of glial cells or neural stem cells in the brain. Glial cells are a prominent part of the enteric nervous system, but gliomas in the gut are very rare and are generally benign. How are enteric glial cells protected against developing aggressive tumours? In this project, we will use a novel line of transgenic mice to investigate enteric glial cell proliferation and changes in their DNA repair pathways.

**Primary Supervisor:** Dr Marlene Hao
**Co-Supervisor:** Dr Lincon Stamp
**Availability:** PhD;MC-BMEDSC

# Palmer Group

**Contact:** Professor Lucy Palmer
**Email:** lucy.palmer@florey.edu.au
**Location:** Florey Institute of Neuroscience and Mental Health

The Neural Networks group uses various techniques to record from neurons in vivo including two photon calcium imaging, somatic and dendritic patch-clamp electrophysiology and optogenetics. Using these advanced techniques, we investigate neural function during learning and memory in health and disease.

**The neural basis of learning and memory**

Our memories define who we are. Whether it's a memory from our childhood or a memory from eating breakfast in the morning, all memories combine to contribute to how we react to everyday life. It is crucial that memories are formed and can be recalled at will. How the brain does this is largely mysterious. The brain consists of billions of individual neurons that are connected to one another forming a complex wiring pattern. An individual neuron receives thousands, sometimes tens of thousands, of inputs from other brain cells. Almost all of these inputs land onto a neuron's complex, tree-like branches, called dendrites. Dendrites then combine these thousands of inputs into action potentials, which is transferred to thousands of other neurons (and the process continues). How neural encoding is modulated during learning and memory is largely unknown, and will be explored here using advanced recording techniques.

**Primary Supervisor:** Professor Lucy Palmer
**Co-Supervisor:** Professor Christopher Reid
**Availability:** PhD;MC-BMEDSC ;Honours

**The influence of brain cancer on neural encoding**

Brain cancer is unique amongst cancers in that they arise, and grow, within the electrically active microenvironment of the brain. It is now realised that brain cancer cells are electrically active and receive synaptic input from nearby neurons. This synaptic input has a functional impact and has been shown to promote tumour growth. This project will investigate how brain cancer influences neural function using advanced monitoring techniques including two-photon microscopy and patch clamp electrophysiology.

**Primary Supervisor:** Professor Lucy Palmer
**Co-Supervisor:** Professor Katharine Drummond
**Availability:** PhD;MC-BMEDSC ;Honours

# McColl Group

**Contact:** A/Prof Gawain McColl
**Email:** gmccoll@florey.edu.au
**Location:** Florey Institute of Neuroscience, Kenneth Myer Building

We develop simple animal models to explore the fundamental processes of ageing and neurodegeneration

**Developing optogenetic approaches to explore age-related cell death in Caenorhabditis elegans**

Biological age is the single greatest risk factor for a range of sporadic diseases, including neurodegenerative conditions such as Parkinson's disease. But what drives this increase in disease risk with age, and how does cell death contribute? A poor understanding of how ageing alters cell signalling and cellular resilience has limited the development of effective therapeutic strategies. In this project you will investigate these questions in Caenorhabditis elegans, using optogenetics to trigger cell death in specific cell types, including muscle cells, intestinal cells, neurons, and their glial support cells, and examine the consequences for whole-animal health and ageing. You will learn core techniques in C. elegans handling, behavioural assays, and genetic analysis, gaining hands-on experience exploring how cell death processes drive age-related decline and frailty in ways that are directly relevant to human ageing and disease.

**Primary Supervisor:** A/Prof Gawain McColl
**Availability:** MC-BMEDSC ;Honours

**Dopamine signalling in behavioural adaptation to environmental change in Caenorhabditis elegans**

Dopamine is a signalling molecule best known for its role in reward and motivation in the human brain, but it also plays conserved roles in simpler animals. In the tiny nematode worm Caenorhabditis elegans, dopamine controls how animals adjust their behaviour when their environment changes, for example, worms slow down when they encounter food, and this response requires dopamine. Because C. elegans has a completely mapped nervous system of just 302 neurons and a powerful set of genetic tools, it offers a uniquely transparent window into how dopamine circuits work. In this project, you will use genetics to alter dopamine synthesis, signalling, or specific dopamine receptors to investigate how this signalling system controls behavioural responses to changes in environmental stimuli. You will learn core techniques in C. elegans handling, behavioural assays, and genetic analysis, gaining hands-on experience with questions that are directly relevant to understanding dopamine function in the human brain.

**Primary Supervisor:** A/Prof Gawain McColl
**Availability:** MC-BMEDSC ;Honours

# Clark group

**Contact:** Dr Mike Clark
**Email:** michael.clark@unimelb.edu.au
**Location:** Melbourne Brain Centre / Kenneth Myer Building

Neuropsychiatric disorders such as Bipolar Disorder and Schizophrenia are severe and incurable diseases in urgent need of improved treatments. Our genes play a key role in who develops neuropsychiatric disorders, but we have little understanding of how. Our research sits at the intersection of genomics and neuroscience, utilising a number of genomic approaches to investigate gene expression and function in the human brain and in neuropsychiatric disorders. We are investigating how the expression and splicing of genes can change to create disease risk and how characterising these changes can help us understand what causes neuropsychiatric disorders and identify novel treatment targets.

A second interest of our research is to develop novel sequencing methods, with a focus on Nanopore sequencing, a technology that can sequence both DNA and native RNA. We are applying Nanopore sequencing to many research questions and developing novel applications for this technology.

**How do our genes cause neuropsychiatric disorders?**

Our genes play a key role in our risk for developing neuropsychiatric disorders such as schizophrenia, bipolar disorder and depression. However, while many risk genes have been discovered, we don’t have a good understanding of what goes wrong with these genes or how they increase risk of disease. Our research has identified many new RNA products from neuropsychiatric risk genes, some of which may confer disease risk. This project will use a combination of cutting-edge technologies, (including Nanopore long-read sequencing, stem-cell models of disease, antisense oligonucleotides, mass spectrometry and bioinformatics) to investigate the function of these RNAs and their protein products and decipher how these genes cause risk for disease. This project will provide crucial new knowledge into the causes of neuropsychiatric disorders and potentially identify novel targets for future therapeutics. The opportunity exists to focus on the lab side and/or the bioinformatic aspects of this project and it would suit students interested in either laboratory work or computational analysis.

**Primary Supervisor:** Dr Mike Clark
**Co-Supervisor:** Dr Ricardo de Paoli-Iseppi
**Availability:** PhD;MC-BMEDSC ;Honours

**Deciphering how our genes control human brain development**

Human brain development is an exquisitely complex process, which is tightly controlled by networks of gene products. Almost all human genes make multiple mRNA products (known as isoforms), but current technologies lack the ability to identify and functionally characterise the repertoire of gene isoforms controlling brain development. This project will profile gene isoforms in developing brain cells using long-read single cell RNA sequencing, a ground-breaking new technique for characterising isoform expression in individual cells, and determine their functional roles. We are using both in-vivo samples and the differentiation of stem-cells into brain organoids to investigate human brain development. This project will illuminate the role of gene isoforms in brain development and form a foundation for understanding how gene isoforms regulate brain cell functions and fates. The opportunity exists to perform bioinformatic analysis of single-cell expression data and/or lab-based discovery of isoform functions and would suit students interested in either laboratory work or computational analysis.

**Primary Supervisor:** Dr Mike Clark
**Co-Supervisor:** Dr Sefi Prawer
**Availability:** PhD;MC-BMEDSC ;Honours

**Discovering the genetic causes of rare diseases with long-read RNA sequencing**

While individually rare, collectively rare diseases affect around 10% of the global population, with 30% of children with a rare disease not reaching their fifth birthday. At least 70% of rare diseases have a genetic cause, however, despite increasing genomic diagnostic capacity in Australia, some 50% of individuals with a rare genetic disorder remain genetically undiagnosed. The application of new genomic technologies, such as long-read RNA-seq, has the potential to solve previously unsolvable cases, providing answers for patients and enabling personalised treatments. This project will analyse cutting-edge long-read Oxford Nanopore RNA sequencing datasets from control and patient human tissues, helping to identify disease causing variants and their effects on gene expression, splicing and function. The student will have the exciting opportunity to work with a national team containing many of Australia's leading researchers in rare disease genomics. The opportunity exists to focus on the lab side and/or the bioinformatic aspects of this project and it would suit students interested in either laboratory work or computational analysis.

**Primary Supervisor:** Dr Mike Clark
**Co-Supervisor:** Dr Ricardo de Paoli-Iseppi
**Availability:** PhD;MC-BMEDSC ;Honours

**Uncovering the role of RNA modifications in brain function and evolution**

While all primates have advanced cognitive abilities, humans have a distinct aptitude for complex cognitive tasks, as well as being prone to neurological and psychiatric disorders. Substantial changes have occurred in gene expression regulation (e.g., RNA splicing and RNA modifications) over evolution and such changes may underly many of the cognitive differences between primate species, including humans. The brain has the most complex transcriptome of major organs and over 35 different RNA chemical modification are known. These modifications, (the epitranscriptome) regulate many critical aspects of RNA metabolism, from splicing to translation. Despite their importance, little is known about them in non-human primates and the role they have played in the human brain evolution. This project will analyse unique Oxford Nanopore direct RNA sequencing datasets from human and marmoset brains to provide the first exploration of transcriptome complexity and RNA modifications in this non-human primate. It will provide crucial new knowledge to understand how RNA modifications patterns have evolved in primates, how human brains are unique, and how regulation of genes that cause neurological and psychiatric disorders have changed in humans. This is primarily a bioinformatics project and it would suit students interested in computational and “big data” analysis.

**Primary Supervisor:** Dr Mike Clark
**Co-Supervisor:** A/Prof Irene Gallego Romero
**Availability:** PhD;MC-BMEDSC ;Honours

# Reid Group

**Contact:** Dr Khaing Phyu Aung
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**Location:** Florey Institute of Neuroscience and Mental Health

- Developing and characterising various genetic mouse models of epilepsy.
- Using genetic mouse models of epilepsy to understand mechanisms and identify new targets and treatments. This involves multiple techniques including behavioural studies, brain-slice electrophysiology, EEG recording, immunohistochemistry, transcriptomics and drug administration.
- Understanding biophysics of ion channels using electrophysiological methods. Techniques involved include two-electrode voltage clamp, voltage-clamp fluorometry and patch-clamp.
- Investigating the relationship between genetic cardiac arrhythmia in sudden unexpected death in epilepsy. Techniques involved include behavioural studies, EEG and ECG recordings, and drug administration.

**(1) Biophysics of leaky HCN ion channels**
**(2) Understanding mechanisms and identifying new targets and treatments using various genetic mouse models of epilepsy**

Project (1) aims to understand the biophysics of ion channels (HCN and potassium channels) using a combination of electrophysiological methods including two-electrode voltage clamp, voltage-clamp fluorometry and patch-clamp. Elucidating the biophysics of these channels that are involved in epilepsy will allow us to understand how epilepsy variants affect the properties of these channels and develop targeted treatments that correct the broken channels.

Project (2) is an umbrella project which aims to develop and characterise various genetic mouse models of epilepsy, which will be used to understand mechanisms and identify new targets and treatments. This project involves multiple techniques including behavioural studies, brain-slice electrophysiology, EEG recording, immunohistochemistry, transcriptomics and drug administration. Some of the mouse models are already up and running in the lab, including mouse models of developmental and epileptic encephalopathy (Hcn1M294L, Gabra, Cux2 etc) and Dravet syndrome (Scn1a). These mice are modelled after genetic variants of patients and mainly recapitulated patient phenotypes. The next step of this project is to understand mechanisms underlying epileptic phenotypes and identify effective therapeutics (which involes screening small molecules, currently available antiseizure medications and toxins) that can reduce symptoms.

**Primary Supervisor:** Dr Khaing Phyu Aung
**Co-Supervisor:** Dr Ming Soh
**Availability:** PhD;MC-BMEDSC

# McQuade Group

**Contact:** Dr Rachel McQuade
**Email:** Rachel.McQuade@unimelb.edu.au
**Location:** Medical Building

Our lab is broadly interested in gut physiology and the potential role of impaired gut health on organ function and the subsequent development and progression of disease. We take a holistic approach to gut physiology, with a focus on gut barrier integrity, and the role of enteric neurons, enteroendocrine cells, immune cells and microbiota in the modulation of gut function.
Our projects are multidisciplinary, spanning basic cell biology, pre-clinical models, and human samples with the aim of generating findings that are relevant across experimental systems. Our projects are spread across a variety of conditions/diseases, with current areas of interest in Parkinson's disease, obesity, ageing and chronic systemic inflammation.

**breaking barriers: understanding the impact of gastrointestinal ageing on longevity**

Food and gastrointestinal (GI) barrier function are central to life and critical for longevity. This project aims to identify cellular and molecular drivers of GI barrier decline and its systemic consequences and to examine how macronutrients shape GI barrier architecture and physiology over a lifetime, providing novel insights into interactions between diet and ageing. Using molecular, biochemical, and proteomic techniques, this project expects to identify basic mechanisms of ageing, offering a fundamental understanding of how in the aged GI barrier shapes systemic physiology, nutrition, and organismal homeostasis. The outcomes of this work may guide future innovations in nutritional science and strategies to optimize ageing trajectories

**Primary Supervisor:** Dr Rachel McQuade
**Co-Supervisor:** A/Prof Jessica Biesiekierski
**Availability:** PhD;MC-BMEDSC ;Honours

**Modelling Chronic "Leaky Gut" Using Faecal Microbiota Transfer**

Intestinal barrier dysfunction, or "leaky gut," is increasingly recognised as a contributor to systemic inflammation and neurodegenerative disease. However, progress in understanding these links has been limited by the lack of reproducible long-term models. This project aims to establish a new, stable model of chronic gut leakiness using faecal microbiota transplant (FMT) from Winnie mice, which naturally develop gut barrier dysfunction. FMTs from human inflammatory bowel disease (IBD) and irritable bowel syndrome (IBS) donors will be used for comparison. Mice receiving these microbiota will be monitored for up to six months using behavioural, gut, and immune assessments. Metagenomic and biochemical analyses of faeces and blood will track microbial engraftment, systemic inflammation, and nutrient absorption. Gut tissues will be examined for epithelial integrity and immune cell infiltration. Together, this study will test whether FMT from Winnie mice provides a reproducible model of chronic leaky gut and help reveal how gut dysfunction contributes to systemic disease.

**Primary Supervisor:** Dr Rachel McQuade
**Co-Supervisor:** Prof Joel Bornstein
**Availability:** Honours

**Gut barrier integrity in 6-OHDA mouse model**

Patients with Parkinson's disease often suffer from an array of debilitating gastrointestinal dysfunction like constipation. These symptoms are contributed to by a compromised gut barrier called the "leaky gut". This project aims to investigate the gut barrier integrity in a 'brain-first' model of Parkinson's disease. Techniques may include tissue histology, immunofluorescence, molecular assays and using chamber studies to localise and assess tight junction proteins and inflammatory markers profile.

**Primary Supervisor:** Dr Myat Noe (Cherish) Han
**Co-Supervisor:** Dr Rachel McQuade
**Availability:** PhD;MC-BMEDSC ;Honours

# Maljevic Group

**Contact:** A/Prof Snezana Maljevic
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**Location:** Florey Institute of Neuroscience and Mental Health

The most severe paediatric epilepsies are associated with genetic mutations in ion channels and synaptic proteins, and they are defined by the presence of medication-refractory seizures, neurodevelopmental delay, psychiatric presentations, dystonic movements and autism spectrum disorder. Our group specialises in modelling these epilepsy forms (also known as developmental and epileptic encephalopathies) using human induced pluripotent stem cells to investigate the disease mechanisms and test innovative therapies through applications of cutting-edge functional and genomic approaches.

**Disruption of early neurogenesis in iPSC-derived models of neurodevelopmental disorders**

Developmental and epileptic encephalopathies (DEE) are severe conditions marked by early-onset seizures and significant cognitive and developmental impairments. Genetic mutations in a range of genes are central to DEE onset and progression. Our project aims to investigate the underlying developmental disruptions in DEE using patient-specific induced pluripotent stem cell (iPSC)-derived models of early neurogenesis.

To dissect the complex mechanisms of DEE pathogenesis, we will combine stem cell culture and immunocytochemistry with high-throughput imaging and AI-based analysis. This integrated approach will enable detailed, unbiased quantification of neurodevelopmental phenotypes across large datasets, advancing our understanding of early disease mechanisms.

**Primary Supervisor:** A/Prof Snezana Maljevic
**Co-Supervisor:** Dr Cristiana Mattei
**Availability:** PhD;MC-BMEDSC

# Yao Group

**Contact:** A/Prof Song Yao
**Email:** song.yao@unimelb.edu.au
**Location:** Medical Building

The Yao laboratory is focused on understanding how dysregulation of the autonomic nervous system contributes to diseases such as hypertension and heart failure, sepsis and inflammation. We take a multi-disciplinary approach to understanding how changes in the neurochemistry and function of key autonomic brain cells and circuits lead to autonomic dysregulation and disease progression. Techniques we frequently use in the laboratory include: in vivo electrophysiology, immunohistochemistry, anatomical neural circuit tracing and advanced microscopy. Current projects include, investigating the role of inflammatory cytokines in hypertension and studying the brains immune cells and their dysregulation in driving adverse outcomes in sepsis.

**Neuroinflammatory cytokines and their impact on autonomic brain circuits in blood pressure control**

Hypertension contributes to cardiovascular disease, which is one of the most prevalent causes of morbidity and mortality in the world. There is good evidence to suggest that there are changes in the brain centres that control cardiovascular function in pre-clinical animal models of hypertension such that there is impaired regulation of cardiovascular neurons in hypertension. This project will investigate inflammatory cytokines and their role in modulating critical brain circuits that control cardiovascular function. This project will involve experiments using in vivo techniques, surgery, immunohistochemistry and advanced microscopy.

**Primary Supervisor:** A/Prof Song Yao
**Co-Supervisor:** Dr Willian Korim
**Availability:** MC-BMEDSC ;Honours;PhD

**Mapping the expression of cytokine receptors in normotensive and hypertensive rat brains**

This project investigates the distribution of cytokine receptors within the brainstem of normotensive and hypertensive rats. By mapping cytokine receptor expression across key cardiovascular regulatory nuclei, we aim to elucidate the neuroinflammatory mechanisms underpinning hypertension, identifying potential therapeutic targets for blood pressure dysregulation.

**Primary Supervisor:** A/Prof Song Yao
**Co-Supervisor:** TBC
**Availability:** MC-BMEDSC ;Honours;PhD

# Metabolic and Cardiovascular Sciences

# Hossain Group

**Contact:** Professor, Akhter Hossain
**Email:** akhter.hossain@unimelb.edu.au
**Location:** Florey Institute of Neuroscience and Mental Health

Peptidomimetics are therapeutic compounds designed to replicate the function of natural hormones and growth factors. These agents offer a unique blend of benefits-combining the bioavailability and stability typical of small molecules with the high specificity and safety profile associated with larger biologic therapies. Our team has pioneered and patented innovative approaches for designing peptidomimetic drugs that target G Protein-Coupled Receptors (GPCRs), which represent the most expansive category of druggable proteins.
To achieve this, we draw on a broad suite of advanced technologies, integrating modern solid-phase peptide synthesis, cutting-edge cellular signalling assays, and in vivo physiological models. This multidisciplinary strategy enables us to explore a wide array of GPCR targets with therapeutic potential across cardiovascular diseases (including fibrotic conditions), metabolic and neurological disorders, and gastrointestinal dysfunctions.

### Targeting RXFP4 for treating colon motility disorders

INSL5 (Insulin-like peptide 5) and its receptor RXFP4 (Relaxin Family Peptide Receptor 4) are key regulators of colonic motility and have been increasingly implicated in the pathophysiology of constipation. INSL5 is primarily produced by enteroendocrine L-cells in the distal colon, where it acts on RXFP4-expressing neurons within the enteric nervous system to modulate colonic propulsion. We aim to therapeutically target RXFP4 through the development of INSL5-based drugs, including both agonists and antagonists, to treat disorders of gut motility such as constipation and diarrhoea. To achieve this, we will employ advanced solid-phase peptide synthesis, state-of-the-art cellular signalling assays, molecular pharmacology techniques, and in vivo physiological models.

**Primary Supervisor:** Professor, Akhter Hossain
**Co-Supervisor:** Ross Bathgate
**Availability:** Honours;MC-BMEDSC ;PhD

# Parker Group

**Contact:** Associate Professor Benjamin Parker
**Email:** ben.parker@unimelb.edu.au
**Location:** Medical Building

The Metabolic Proteomics and Signal Transduction Group is focused on understanding how signal transduction regulates metabolism with the goal of identifying new therapeutic targets to treat disease. We are particularly interested in studying dynamic changes in proteins and their post-translational modifications, and understanding how these are regulated by genetic variants and metabolic insults to ultimately shape cellular physiology. We primarily focus on metabolic tissues such as the heart, liver and adipose with a strong emphasis on muscle.

**Developing spatial proteomics methods to treat cardiometabolic disease**

Cells are constantly talking to each other and this communication is defective in nearly every human disease. Spatial-'omics technologies have revolutionized our understanding of how cells in precise locations of a tissue are dysregulated in disease. Single cell and spatial proteomics is the new frontier in this technology and offers distinct advantages to understand cell communication. This project will develop new spatial proteomics methods to understand how proteins in precise locations within specific cells are disrupted in cardiometabolic diseases such as obesity, insulin resistance and type-2 diabetes. Identified proteins will be genetically manipulated using CRISPR/Cas9 followed by physiological assessment of a range of in vivo phenotypes such as metabolic assessment, muscle/heart function, exercise tolerance amongst several others. The outcomes of this project will lead to pre-clinical evaluation of therapeutic targets to treat cardiometabolic disease.
Primary Supervisor: Associate Professor Benjamin Parker
Availability: PhD;MC-BMEDSC ;Honours

**Modulating skeletal muscle signal transduction to treat diabetes**

Insulin resistance (or pre-diabetes) is the fastest growing disease in the world and it's estimated >2 million Australians are at risk of developing type-2 diabetes. We urgently need new therapeutic treatments to use in conjunction with diet/exercise to treat these diseases. Insulin resistance is characterised by a major defect in the ability of insulin to promote glucose uptake into skeletal muscle. This results in hyperglycemia and several other diabetic complications. We have identified a series of lead candidates that promote insulin sensitivity and glucose uptake into skeletal muscle. These lead candidates include several kinases and phosphatases that regulate phosphorylation-based signaling pathways. This project will perform ex vivo functional screening in a series of pre-clinical models to understand how signaling pathways regulate glucose uptake and metabolism. The project will involve a variety of techniques including isotopic tracing of metabolism, phosphoproteomics and biochemistry.

**Primary Supervisor:** Associate Professor Benjamin Parker
**Co-Supervisor:** na
**Availability:** PhD;MC-BMEDSC ;Honours

# Dodd Group

**Contact:** Prof Garron Dodd
**Email:** garron.dodd@unimelb.edu.au
**Location:** Medical Building

Our lab investigates how the brain regulates metabolism and how this is disrupted in obesity and type-2 diabetes. Using cutting-edge tools, we identify new drug targets. We seek motivated, curious students eager to explore neuroscience, metabolism, and translational research in a collaborative, high-impact environment.

**Cracking the Brain's Hidden Barrier to Reverse Diabetes**

More than one in ten people now live with type 2 diabetes, and the number is climbing fast. The brain sits at the centre of the problem. Normally, the hormone insulin acts on a small population of neurons deep in the brain to switch off hunger and keep blood sugar in check. In diabetes, this conversation breaks down. We have discovered that these neurons become wrapped in a dense molecular barrier that physically blocks insulin from getting through, leaving the brain effectively deaf to one of the body's most important signals.
In this project you will work out how this barrier forms, and whether stripping it away can restore the brain's sensitivity to insulin and reverse features of diabetes. You will gain hands on training in a powerful toolkit that few students ever get to use, including CRISPR gene editing, precision brain surgery, and whole tissue clearing that renders the brain transparent so you can image neural circuits in three dimensions.
This is discovery science with a clear translational endpoint. Every result feeds directly into our search for new drug targets for obesity and type 2 diabetes. We are looking for curious, driven students who want to learn cutting edge skills, ask big questions, and launch a career at the frontier of neuroscience and metabolic disease.

**Primary Supervisor:** Prof Garron Dodd
**Co-Supervisor:** Cait Beddows
**Availability:** PhD;MC-BMEDSC

**Why Exercise Resets the Obese Brain**

Obesity is one of the defining health challenges of our time, and at its core lies a brain that has stopped listening. In obesity, the brain becomes resistant to leptin and insulin, the two hormones that normally tell us when we have eaten enough. Remarkably, exercise can restore the brain's sensitivity to these signals, sometimes even before any weight is lost. How it does this remains one of the most intriguing open questions in metabolism, and answering it could unlock an entirely new class of treatments.
In this project you will uncover how exercise reaches into the brain and resets the circuits that control appetite and body weight. You will learn a suite of cutting edge technologies, including precision brain surgery, CRISPR gene editing, advanced imaging, and tissue clearing that lets you visualise whole neural circuits in three dimensions.
This is fundamental neuroscience with a direct line to the clinic. Your findings will help explain why exercise is so protective for the brain, and how we might bottle those benefits as a therapy for the millions of people who cannot exercise enough on their own. We are seeking curious, motivated students who want to gain rare technical skills and build a career at the cutting edge of neuroscience and metabolic disease.

**Primary Supervisor:** Prof Garron Dodd
**Co-Supervisor:** Cait Beddows
**Availability:** PhD;MC-BMEDSC

# Delbridge/Weeks Group

**Contact:** Dr Kate Weeks
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**Location:** Medical Building

The Cardiac Phenomics & Signalling Lab seeks to understand how the heart response to stress can be managed to minimize the damaging impacts of a variety of disease conditions. We investigate responses of the working 'pumping' heart, of specialized muscle tissues and cells from different regions of the heart, and of molecular signalling processes. As our name suggests, we look at how the cardiac 'genome' (the genetically defined heart) is translated in different stressor situations to create the 'phenome' (the structurally and functionally defined heart). One of our focus areas is to explore the molecular signalling events involved in various heart failure types. Our pre-clinical work focuses on cardiac pathology arising from Type 1 and Type 2 diabetes and on the factors which determine how female and male hearts respond differently to stress and disease challenges. These areas of heart health are of critical significance in shaping the demographics of cardiovascular disease. We use experimental models to mimic human disease conditions, and we look for links between the performance of single muscle cells and the functioning heart. Our goals are to inform the development of new treatments for diabetic cardiomyopathy and heart failure and to understand how for women and men, cardiac 'difference' may be managed with optimized therapeutic tools. Student projects could incorporate a range of methodologies including animal dietary and pharmacologic treatments, instrumented working heart preparations, immunohistochemistry, cell culture and adenoviral expression manipulation, cell kinetic imaging, biochemical assays, confocal microscopy, big data "omics" (e.g. transcriptomics, proteomics, phosphoproteomics), real-time PCR, and Western blot techniques. Projects are particularly suitable for MSc students, as there is scope for progression to publication within the degree time frame and research work is supported by complementary skills development coursework. As part of the Cardiac Research Consortium (Australia and New Zealand), our projects involve multiple campus opportunities in Melbourne (LaTrobe University, Baker Heart & Diabetes Institute) and New Zealand (University of Auckland). There are also the prospects of Study-away opportunities with international collaborators (USA, Canada, Europe & UK).

**New therapeutic leads in heart failure - developing protein phosphatase directed treatments**

Heart failure is a debilitating condition in which the ability of the heart to meet the body's demands for oxygenated blood is compromised. Prognosis is poor, with approximately 50 per cent of patients with heart failure dying within 5 years of diagnosis. There is a clear need for new therapeutic strategies for the treatment of heart failure. Much of the research focus to date has been on the 'kinase' superfamily of enzymes – which are cardiometabolic 'on' switches. This project will explore the role of a family of proteins known as 'protein phosphatases' which provide the physiological 'off' switch to oppose kinase action. In this project the particular role of selected phosphatase treatments will be explored. The goals are to examine how phosphatases can be protective in suppressing the development of heart failure, and whether phosphatases can be selectively targeted to delay progression of heart failure. This project will involve work with new 'gene-editing' technology developing experimental models of disease and also involves work with clinical biopsy samples. Students have the opportunity to experience research in the University academic setting in Parkville and also to interact with collaborators at the Baker Heart & Diabetes Institute.

**Primary Supervisor:** Dr Kate Weeks
**Co-Supervisor:** Prof Lea Delbridge
**Availability:** PhD;MC-BMEDSC

**Investigating sex differences in heart failure**

Heart failure with preserved ejection fraction (HFpEF) accounts for more than 50% of heart failure patients and is particularly prevalent in women. An understanding of the cellular mechanisms underlying HFpEF is limited with no clincial treatments identified. In particular, gender-specific aspects of HFpEF etiology have not been well characterised. We have recently published new findings which reveal the cardiac muscle cell basis for increased cardiac 'stiffness' observed in heart failure. The goal of this project is to explore these new molecular leads, comparing female and male disease states, using Crispr-Cas9 'gene editing' to create animal models. This project involves training and use of cutting edge proteomics analysis approaches to apply big-data investigations searching for disease-causing candidate proteins linked with full-scale disease characterization involving cardiac echo imaging and single cell functional studies.

**Primary Supervisor:** Prof Lea Delbridge
**Co-Supervisor:** Dr Kate Weeks
**Availability:** PhD;MC-BMEDSC

**Developing novel biomarkers for diabetic heart failure**

Impaired diastolic relaxation is an early sign of diabetic cardiomyopathy and involves increased heart wall stiffness and abnormal filling during the diastolic period of the cardiac cycle. The early occurrence of diastolic dysfunction in otherwise 'healthy' asymptomatic diabetic patients has been extensively reported, and is prognostic of later occurrence of heart failure and increased mortality. In the diabetic heart, irreversible modifications of certain cardiac proteins is correlated with impaired heart relaxation. Our data demonstrate that these protein modifications may contribute to impaired cardiac relaxation, indicating that small changes in protein structure can have large implications for diastolic function in diabetes. Specific characterisation of these key protein modifications offers the opportunity for biomarker development for use in the early detection of subclinical diabetic cardiomyopathy and monitoring of therapies. This project will involve work with experimental models of disease and custom designed cell systems. . Opportunities for study away to pursue project research in New Zealand (in Dr Mellor's Laboratory) are also available. This work also involves extensive collaboration with our Los Angeles partners at Cedars Sinai Medical Center.

**Primary Supervisor:** Prof Lea Delbridge
**Co-Supervisor:** Dr Kate Weeks, Dr Kim Mellor
**Availability:** PhD;MC-BMEDSC

**Leveraging exercise biology to protect the heart from toxic cancer drugs**

Anthracyclines remain a cornerstone of chemotherapy for many cancers. However, their clinical utility is limited by dose-dependent cardiotoxicity, which can lead to heart failure. Emerging evidence suggests that factors released into the circulation during exercise, known as exerkines, may mitigate these adverse effects by modulating gene expression in cardiomyocytes. This project will investigate novel transcriptional regulatory mechanisms that underlie the cardioprotective actions of exerkines in the context of doxorubicin-induced cardiotoxicity. Employing a combination of biochemical, histological, and functional assays, the study will assess the impact of gene therapy interventions on gene expression and cardiac function in organoid and/or in vivo models. The findings may reveal new therapeutic targets to mitigate chemotherapy-associated cardiac damage.

**Primary Supervisor:** Dr Kate Weeks
**Co-Supervisor:** Prof Lea Delbridge
**Availability:** Honours

# Montgomery Group

**Contact:** Dr Magdalene Montgomery
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**Location:** Medical Building

Metabolic dysfunction-associated steatotic liver disease (MASLD), until recently referred to as NAFLD, is the most common chronic liver condition in developed countries, with a global prevalence of >30%. The later stages of MASLD show marked fibrosis, which is associated with the development of life-threatening diseases, including cirrhosis and liver cancer.

Despite this increasing clinical epidemic, the first pharmacotherapy for the treatment of MASLD and liver fibrosis has only been provisionally approved by the FDA in 2024, and novel approaches and ideas are instrumental. That's where we come in!

In addition, there is a close bidirectional relationship between MASLD and insulin resistance/type 2 diabetes, and our lab is additionally interested in understanding the inter-connect between hepatic lipid and glucose metabolism, and blood glucose control.

**The identification of novel regulators of lipid metabolism in the liver - potential therapeutic targets for metabolic liver disease and liver cancer?**

Metabolic dysfunction-associated steatohepatitis (MASH), previously denoted as non-alcoholic steatohepatitis, is characterized by lipid accumulation, inflammation and frequently fibrosis, and is associated with the development of liver cancer. Hepatocellular carcinoma (HCC) is the second most frequent cause of cancer-related death worldwide with a low 5-year survival rate of only 18%, highlighting the need for novel therapies for both MASH and HCC.

Our team has performed the worlds' first genome-wide CRISPR screen targeting 19,050 genes in liver cells combined with functional assessment of lipid accumulation and sensitivity to cancer therapy. This screen identified 100's of novel regulators of lipid metabolism and cancer growth highly relevant for therapeutic targeting.

This project will focus on uncovering the function and therapeutic potential for some of these novel regulators which will pave the way to understanding if targeting those proteins could be beneficial for MASH and HCC.

**Primary Supervisor:** Dr Magdalene Montgomery
**Co-Supervisor:** Li Dong
**Availability:** PhD;MC-BMEDSC

# Tham Group

**Contact:** Dr Yow Keat Tham
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**Location:** Baker Heart and Diabetes Institute

The Group investigates the role of plasmalogen lipids in cardiometabolic disease using preclinical, in vitro, and in vivo models, while also exploring plasmalogens as therapeutic targets and interventions for the prevention and treatment of cardiometabolic diseases.

**Can plasmalogen-enhancing therapies protect the heart after a heart attack?**

Heart attacks (myocardial infarction; MI) remain a leading cause of global mortality. Heart attacks cause ischemic damage that arise from the reduction of blood flow to the heart, resulting in cell death. While the most effective clinical intervention for MI is to allow for the prompt restoration of blood flow (reperfusion) via stenting or vessel bypass grafting, this often resulting in additional heart damage caused when blood supply returns to the heart tissue. Collectively this is termed ischemia reperfusion (IR) injury. There are no current therapies to address this, highlighting an urgent need for novel treatments that reduce damage and enhance recovery.

Innovative Therapeutics via Plasmalogen Enhancement: At the Baker Heart and Diabetes Institute, our research is pioneering the therapeutic modulation of plasmalogens—a unique class of protective lipids. Plasmalogens have potent antioxidative and anti-inflammatory properties, which we believe can significantly reduce cardiac damage from IR injury. This project leverages cutting-edge lipidomics, proteomics, and advanced preclinical models to explore this potential breakthrough.

Research Objectives:

1. Evaluate Cardioprotective Effects:
    - Examine how dietary plasmalogen enhancement reduces heart injury after ischemia-reperfusion events in mouse models.

2. Explore Underlying Mechanisms:
    - Investigate how plasmalogen supplementation influences inflammation, immune cell activity, mitochondrial function, and oxidative stress.

Your Role and Opportunities: You will actively participate in groundbreaking research aimed at transforming how we mitigate cardiac IR injury. You will:

- Conduct experiments using animal models of cardiac IR injury.
- Learn and apply sophisticated laboratory techniques including lipidomics, flow cytometry, histology, mitochondrial function assays, and microscopy.
- Gain experience in data analysis and interpretation in lipidomics, proteomics, echocardiocardiography and providing skills highly sought-after in both academia and industry.
- Contribute to publications and present your findings at scientific conferences, boosting your research profile.

Why Choose This Project?

- Transformative Impact: Your research will directly contribute to developing new therapeutic strategies that could significantly reduce the burden of heart attacks worldwide.
- Skill Development: Receive comprehensive training in advanced techniques that are pivotal in modern biomedical research.
- Multidisciplinary Collaboration: Join a dynamic and supportive team within a leading research institute, benefiting from robust mentorship and extensive collaborative networks.

Ideal Candidate: We seek enthusiastic, motivated students passionate about biomedical research and keen to make meaningful contributions to cardiovascular health. Ideal candidates will demonstrate strong analytical skills, dedication to scientific inquiry, and an eagerness to learn advanced laboratory and analytical techniques.

Keywords: Cardiovascular disease, lipidomics, ischemia-reperfusion injury, inflammation, therapeutics

**Research Webpage:** https://www.baker.edu.au/research/laboratories/metabolomics
**Primary Supervisor:** Dr Yow Keat Tham
**Co-Supervisor:** Prof Peter Meikle
**Availability:** Honours

**Can restoring plasmalogens protect macrophages from plaque-destabilising lipid stress?**

Atherosclerosis is a chronic inflammatory disease in which macrophages play a central role in plaque progression and vulnerability. Under atherogenic lipid stress, macrophages can accumulate lipid, become inflammatory, lose phagocytic function, and contribute to necrotic core formation. Recent work from our group suggests that plasmalogen enhancement via the use of alkylglycerol (AKG) supplementation may reduce features of plaque vulnerability in vivo, but whether this directly reflects improved macrophage function remains unknown.
This Honours project will test whether AKG-mediated plasmalogen enhancement protects RAW264.7 macrophages from oxLDL-induced dysfunction. The student will assess foam-cell formation, inflammatory gene expression, phagocytic function, and mitochondrial oxidative stress using cell culture, Oil Red O staining, qPCR, bead-based or pHrodo-labelled uptake assays, and MitoSOX analysis.
This project would suit a student interested in cardiovascular disease, macrophage biology, lipid metabolism, inflammation, and preclinical therapeutic discovery. The project provides hands-on training in cell-based cardiovascular research and will contribute to a broader program investigating plasmalogen restoration as a potential strategy to improve plaque stability.

**Primary Supervisor:** Dr Yow Keat Tham
**Co-Supervisor:** Prof Peter Meikle
**Availability:** Honours, MMC-BMEDSC

# Hossain Group

**Contact:** Prof Akhter Hossain
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**Location:** Florey Institute of Neuroscience and Mental Health

Our lab is interested in developing new peptide-based drugs for diabetes, obesity, pain, and colon motility disorders

**Developing novel peptidomimetics for diabetes and obesity**

The project will investigate a novel drug traget in the brain and probe its physiological function in treating diabetes and obesity

**Primary Supervisor:** Prof Akhter Hossain
**Co-Supervisor:** TBC
**Availability:** PhD, Masters and Honours

# Scholarship of Teaching and Learning (SOTL)

# IGNITE Research Group

**(Insights into Generative and Networked Innovation in Teaching & Education)**

**Contact:** A/Prof Angelina Fong
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**Location:** Medical Building

We are keenly interested in how technology and digital tools can enhance, and support student learning and improve educational outcomes. As part of the “age of technology”, current students are presented with a broad range of learning resources and educational tools. These tools can range from online videos, discussion boards, virtual environments, to Chatbots powered by Generative Artificial Intelligence (GenAI). It is unclear if and how these tools can be used to benefit students and their learning as rate of change increases with the emergence of the “intelligence age” and GenAI. We are also interested in understanding the underlying motivations and expectations of students as they engage with their learning and education, whether it be with the subject material, or with their learning environments. Together, thes e research areas will provide insights into how to design learning environments, and resources to best support students to promote their education.

**Eye on Learning in the Digital Age: Clicks, Views, and What Helps Students Thrive**

This project examines how students engage with digital and AI-enabled learning tools, and how these interactions shape learning outcomes in contemporary higher education. With the rapid expansion of online platforms, lecture capture, virtual environments, and generative AI, there is a need for deeper, evidence-based understanding of what actually supports meaningful learning. Student researchers will contribute to a mixed-methods investigation of both observable engagement behaviours (e.g., interactions with videos, platforms, and AI tools) and underlying learning motivations that drive how, when, and why students use these resources. The project offers opportunities to work with innovative research methods, including learning analytics, behavioural data, and eye tracking or other real-time measures of engagement.
There is also scope to be involved in the co-design and evaluation of new digital learning interventions, enabling educators to translate findings into practical improvements in teaching and resource design.
This project is well suited to students interested in education research, digital learning, human behaviour, or emerging AI applications in education. The outcomes will contribute to evidence-based approaches that enhance student learning at scale.

**Primary Supervisor:** A/Prof Angelina Fong
**Co-Supervisor:** TBC
**Availability:** Honours;MC-BMEDSC ;PhD

**Mapping the Landscape of Student Perceived Skill Acquisition.**

This project explores how undergraduate students recognise, understand, and value the transferable skills they develop throughout their degree. While curricula are intentionally designed to build capabilities such as teamwork, communication, leadership, digital literacy, and feedback literacy, students may not always be aware of these skills or able to clearly articulate them.
The study focuses on student perceptions of skill development, examining how students identify the skills they acquire, how they value them, and how they link these capabilities to future career or academic pathways. It may also investigate how different teaching approaches and learning environments support (or hinder) students' ability to recognise and communicate their skills.
There is scope for projects analysing student reflections, surveys, or interviews, as well as designing and evaluating interventions to improve skill awareness and articulation. The findings aim to inform curriculum design and strengthen graduate employability outcomes, with project direction tailored to student interests in consultation with supervisors.
The exact nature of the research project may be directed by the student's individual interest in discussion with the supervisors.

**Primary Supervisor:** A/Prof Angelina Fong
**Co-Supervisor:** TBC
**Availability:** PhD;MC-BMEDSC ;Honours

**Co-designing Inclusive Scientific Diagrams for Diverse Learners in STEM**

This project explores how students engage with scientific diagrams and aims to improve how complex scientific concepts are visually communicated in STEM education. While diagrams are widely used to support learning in disciplines such as physiology, microbiology, immunology, and chemistry, they are often designed with limited consideration of diverse learning needs.
We are seeking students to participate as collaborators in evaluating, redesigning, and co-creating scientific diagrams to make them more inclusive and accessible. Students will work alongside educators, learning designers, and researchers to explore how diagram design influences understanding, cognitive load, and engagement—particularly for neurodiverse learners (e.g., ADHD, dyslexia).
Through workshops, feedback sessions, and co-design activities, participants will help develop improved diagram formats and contribute to evidence-based guidelines for inclusive visual learning in STEM. This is an opportunity to gain experience in educational research, user-centred design, and collaborative problem-solving, while directly shaping teaching practices that impact thousands of students.

**Primary Supervisor:** A/Prof Angelina Fong
**Co-Supervisor:** TBC
**Availability:** Honours;MC-BMEDSC ;PhD

# SMART (Student Motivation, Activity, and Research for Teaching)

**Contact:** Associate Professor Joseph (Yossi) Rathner
**Email:** Joseph.Rathner@unimelb.edu.au
**Location:** Medical Building

Our research group applies the Science of Learning to better understand how students approach their studies in today's higher education landscape and to develop more effective academic skills. We employ a rigorous, mixed-methods approach that combines student surveys with controlled experiments to test hypotheses about learning approaches.

In the rapidly evolving educational environment, accelerated by the COVID-19 pandemic, we're investigating the impact of asynchronous learning models and educational technologies on student engagement and outcomes. Our research focuses on several key areas:

1) Note-taking strategies in digital environments: We're examining the differences between transcription and effective note-taking, particularly in the context of recorded lectures. Through controlled experiments, we're testing how various note-taking methods affect information retention, comprehension, and academic performance.

2) Cognitive load and information processing: Our studies investigate how the cognitive demands of different learning approaches impact students' ability to process and understand course material. We're particularly interested in how multitasking (e.g., listening while transcribing) affects learning outcomes.

3) Elaborative learning techniques: We're exploring the effectiveness of strategies like mind mapping and concept mapping in enhancing understanding and retention. Our experiments compare these techniques with more traditional study methods to identify best practices for deep learning.

4) Pre- and post-lecture learning strategies: Through both surveys and controlled studies, we're assessing how pre-lecture preparation and post-lecture review techniques influence learning outcomes. This includes investigating the impact of providing lecture transcripts on student engagement and comprehension.

5) Developmental aspects of learning strategies: Our longitudinal studies track how students' learning approaches evolve throughout their academic careers, helping us understand how to tailor support at different stages of higher education.

6) Balancing accessibility and active engagement: We're investigating how to optimize the use of learning aids like transcripts and recorded lectures to enhance accessibility without promoting passive learning behaviors.

7) Technology-enhanced learning environments: Our research examines how various digital tools and platforms affect student engagement, time management, and overall academic performance.

learning resources, and institutional policies, ultimately leading to improved student outcomes and a more engaging, efficient educational experience.

By combining survey data on student perceptions and experiences with empirical evidence from controlled experiments, we aim to bridge the gap between student practices, institutional policies, and effective pedagogical approaches. This comprehensive approach allows us to develop evidence-based recommendations for both students and educators.

Our ultimate goal is to enhance student learning by promoting strategies that foster deep understanding, critical thinking, and long-term retention of knowledge. We're committed to helping students develop robust academic skills that will serve them throughout their educational journey and beyond.

Through this research, we're contributing to the ongoing transformation of higher education, ensuring that technological advancements and pedagogical innovations are harnessed to truly enhance the learning experience. Our findings inform the development of more effective teaching methods, learning resources, and institutional policies, ultimately leading to improved student outcomes and a more engaging, efficient educational experience.

**Optimising Student Learning Strategies in Higher Education: A Scientific Approach to Understanding and Enhancing Academic Performance in the Digital Age**

Key skills students will develop:

1. Survey Design and Analysis
2. Focus Group Interviews
3. Controlled Intervention Studies
4. Statistical and Qualitative Data Analysis
5. Participant Management:
    - Recruiting participants
    - Main contact for participants
    - Maintaining communication
    - Managing relationships

Research focuses on technology-enhanced learning:

- Online simulated practical classes
- Asynchronous learning environments
- Augmented/virtual reality experiences
- Digital tools for note-taking, retention
- Adaptive learning platforms
- AI in learning and teaching

Students involved in all research stages:

- Research design discussions
- Ethics applications
- Developing data collection instruments
- Data collection and analysis
- Interpreting results

Project offers opportunity to contribute to cutting-edge research in educational psychology and learning sciences. Students develop skills in mixed-methods research and participant management, enhancing future employability.
Findings contribute to evidence-based practices in higher education, potentially influencing teaching methods and institutional policies.

Students gain:

- Understanding of learning behaviours and technology's impact on education
- Critical evaluation skills for educational interventions
- Experience with quantitative and qualitative methods
- Ability to approach complex problems from multiple angles
- Relevant skills for technology-driven educational contexts

Research experience equips students with:

- Practical skills
- Analytical thinking
- Understanding of educational research methodologies

This project develops transferrable skills, enhances understanding of learning processes, and provides opportunity to impact educational practices through rigorous, innovative research. Students will be well-positioned as valuable contributors to the field of education and beyond, prepared for evolving digital learning environments and various career paths.

**Primary Supervisor:** Associate Professor Joseph (Yossi) Rathner
**Co-Supervisor:** Makhala Khammy
**Availability:** PhD;MC-BMEDSC ;Honours

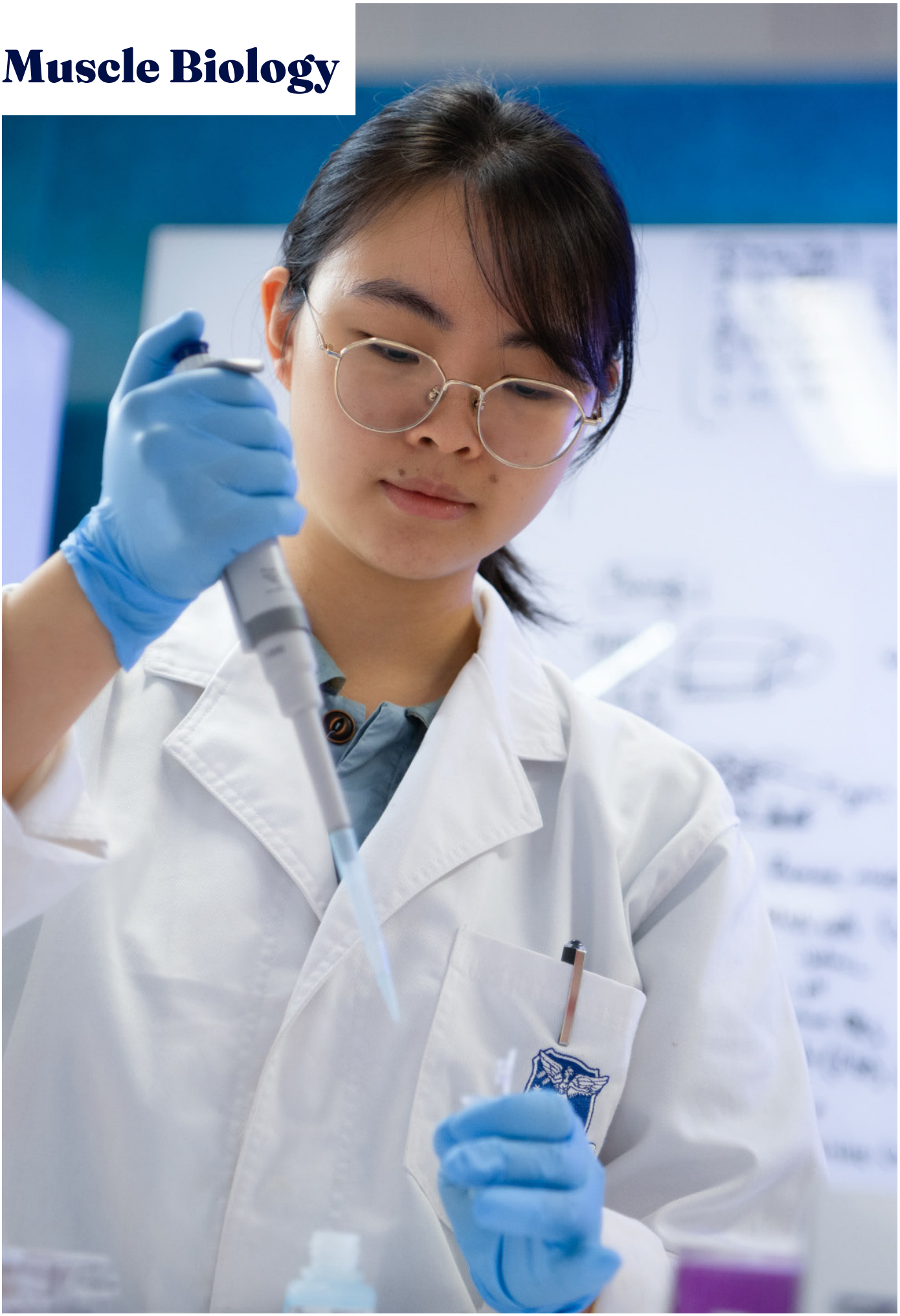
**Fit for Success: Leveraging Exercise to Enhance Academic Performance**

1. Survey Design and Analysis:
   - Create and implement surveys on student perceptions, experiences, and learning behaviours
2. Focus Group Interviews:
   - Conduct and analyse qualitative data
   - Develop qualitative research skills
3. Controlled Intervention Studies:
   - Design and execute experiments assessing physical activity's impact on academic outcomes
   - Develop skills in experimental design, participant recruitment, and data collection
4. Physical Fitness Assessments:
   - Conduct VO2 max tests and other fitness evaluations
   - Operate specialized equipment and interpret physiological data
5. Participant Management:
   - Develop skills in managing people and tasks, ensuring participant engagement
6. Data Management and Analysis:
   - Organize and manage large datasets
   - Apply statistical methods to interpret quantitative data
   - Use AI tools for coding and interpreting interview data
7. Ethics Applications:
   - Assist in preparing ethics applications for human research
   - Understand ethical considerations in human participant research
8. Collaboration with External Partners:
   - Work with Melbourne University Sport staff
   - Gain experience in interdisciplinary collaboration and stakeholder management

Students will be involved in all research stages, from study design to data analysis and interpretation. They'll gain hands-on experience with quantitative and qualitative methods, developing a well-rounded skill set applicable to various research contexts.
This project offers an opportunity to contribute to innovative research that could significantly impact university policies and student support practices. Students will develop valuable transferable skills in project management, data analysis, scientific communication, and participant management, preparing them for future careers in research or related fields.

**Primary Supervisor:** Associate Professor Joseph (Yossi) Rathner
**Co-Supervisor:** Dr Keit Loi
**Availability:** PhD;MC-BMEDSC ;Honours

# Muscle Biology



# Ruparelia lab

**Contact:** Dr Avnika Ruparelia
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**Location:** Medical Building

The Ruparelia lab is investigating the fundamental biology of muscle wasting disorders such as sarcopenia, which is the age-related decline in muscle mass and function, and genetic conditions such as Duchenne muscular dystrophy - examining processes such as muscle fiber atrophy and growth, muscle stem cell dynamics, muscle regeneration, and metabolism. We not only use the unique advantages of the zebrafish model, but also uses a new experimental system – that of the African killifish, whose extremely short lifespan makes it an attractive model for ageing research.

### Identifying therapies for muscular dystrophies using zebrafish

Muscular dystrophies have one of the highest burdens of disease, and yet, there is currently no cure for this devastating group of muscle wasting disorders. Therefore, using the advantages of the zebrafish model system combined with genetic approaches, confocal microscopy, immunofluorescence techniques and live imaging, we aim to identify novel disease mechanisms and therapeutic strategies for the treatment of muscular dystrophy.

**Primary Supervisor:** Dr Avnika Ruparelia
**Availability:** MC-BMEDSC ;PhD;Honours

### Characterising muscle ageing in the short lived killifish

Sarcopenia, the age-related decline in muscle mass and function, places a great burden on the health care system. Despite this, very little is known about the molecular pathways that drive this process. To address this, we have developed a new model of ageing - that of the African killifish which has an extremely short lifespan. Here, using cellular, molecular and genetic techniques, we will characterise the muscle of these short-lived vertebrates to determine if they undergo sarcopenia.

**Primary Supervisor:** Dr Avnika RUparelia
**Availability:** PhD;Honours;MC-BMEDSC

# Parker Group

Contact: Associate Professor Benjamin Parker
Email: ben.parker@unimelb.edu.au
Location: Medical Building

The Metabolic Proteomics and Signal Transduction Group is focused on understanding how signal transduction regulates metabolism with the goal of identifying new therapeutic targets to treat disease. We are particularly interested in studying dynamic changes in proteins and their post-translational modifications, and understanding how these are regulated by genetic variants and metabolic insults to ultimately shape cellular physiology. We primarily focus on metabolic tissues such as liver and adipose with a strong emphasis on muscle and bone.

**Proteome-wide systems genetics and physiology of muscle function**

We all poses genetic variants that make us unique. They predispose you to disease or they are protective and make you strong. Identifying these genetic variants is not only the essence of personalised medicine but they can be used as a research tool to identify the function of uncharacterised genes. This study is built on a massive proteomic and genomic dataset that has identified specific genetic variants and novel genes that are correlated with muscle function. This project will use CRISPR/Cas9 to genetically manipulate these genes followed by physiological assessment of cardiac and/or skeletal muscle function combined with biochemistry and molecular analysis. The discovery of novel genes that provide functional protection of muscle is fundamental to the development of novel therapeutic targets.

**Primary Supervisor:** Associate Professor Benjamin Parker
**Co-Supervisor:** na
**Availability:** PhD;MC-BMEDSC ;Honours

# Lynch Group

**Contact:** Prof. Gordon Lynch
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**Location:** Medical Building

Skeletal muscle is essential for survival. Not only is muscle the vital organ for movement but the diaphragm muscle sustains life by inflating the lungs for breathing. Skeletal muscle is also an endocrine organ that contracts and releases hormones and factors that communicate with other body tissues to sustain life. Skeletal muscle accounts for half a person's body mass yet we take for granted its crucial role in our health and lifestyle. Many diseases and conditions are linked with changes in muscle structure and function, including: ageing and frailty; cancer; muscle injury, sepsis and other forms of metabolic stress; nerve injury; disuse through inactivity and microgravity; burns; and different forms of muscular dystrophy. These conditions are major health problems globally and contribute to a large burden of disability and suffering. Tackling these muscle-related health conditions requires a coordinated research effort from discovery biology to understand disease mechanisms and translational approaches to take these discoveries from bench to the clinic. Researchers in the Centre for Muscle Research seek to understand the mechanisms that regulate muscle growth, wasting and metabolism, and to develop new approaches for preventing or treating muscle related conditions, utilising the latest techniques in biology and biomedicine. We also consider skeletal muscle in the context of other diseases, such as heart and cardiovascular diseases, cancer and osteoporosis. We are interested in understanding muscle development and growth, injury and repair, studying the biology and metabolism of muscle stem cells and their commitment to becoming functional muscle fibres. Our researchers design, manufacture and utilise viral vectors to alter gene expression in mouse models of disease and interrogate cellular mechanisms of muscle adaptation, techniques that provide a unique combination of speed, precision and efficacy not achieved through other approaches. The Centre for Muscle Research offers a wonderful training environment for studying muscle biology in health and disease and exceptional career-training opportunities for Honours, Masters and Ph.D. students.

**Understanding the plasticity of skeletal muscle in health and disease**

Skeletal muscle is comprised of diverse fibre types that differ in size, metabolic and contractile properties; classically referred to as either 'slow, oxidative' or 'fast, glycolytic'. These properties are not fixed but can change in response to imposed demands, a process known as 'plasticity'. Understanding the biological mechanisms regulating fibre phenotype and the adaptive response across muscles of varying phenotypes has not been fully resolved. Addressing these research gaps may also identify potential therapeutic targets to improve quantity and quality of life across many disease conditions. The objectives of this project are to: 1) understand the biological mechanisms regulating fibre size, phenotype and plasticity; and 2) whether modifying skeletal muscle attributes can protect against injury and disease. This project will utilise genetic, pharmacological and lifestyle approaches to interrogate the molecular, metabolic and contractile properties of fast and slow muscles in a variety of healthy and pathological states; including but not limited to muscular dystrophies, cancer cachexia, muscle injury and repair, and ageing.

**Primary Supervisor:** Prof. Gordon Lynch
**Co-Supervisor:** Marissa Caldow
**Availability:** PhD;MC-BMEDSC ;Honours

**Characterising the pathways responsible for ICU-acquired weakness**

Muscle wasting is the most common complication of critical illness, occurring in 25-50% of patients. The extent of wasting is determined by the severity of organ failure and lung injury, however, a loss of 20-30% of muscle mass over the first 10 days in ICU is not uncommon.
ICU patients generally have increased muscle protein breakdown relative to muscle protein synthesis, leading to a net catabolic state and rapid loss of muscle mass and function. To allow the development of novel and effective treatments to attenuate muscle wasting in ICU patients it is important to identify the signalling pathways and proteins that drive this catabolic state. This project uses animal-based experiments and analyses of muscle biopsies from ICU patients to comprehensively test these mechanisms. Findings from this project will enhance our knowledge about the regulation of skeletal muscle mass during critical illness and will aid in the further development of treatment strategies.

**Primary Supervisor:** Prof. Gordon Lynch
**Co-Supervisor:** Marissa Caldow
**Availability:** PhD;MC-BMEDSC ;Honours

# Cheng Group

**Contact:** A/Prof Louise Cheng
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**Location:** Peter MacCallum Cancer Centre

We are interested in organ size control and how inter-organ communication mediates crosstalk between organs during development and in cancer.

**How do tumours grow at the expense of other tissues in cancer cachexia?**

Cancer cells are known to drive altered metabolic circuits to meet the bioenergetic and biosynthetic demands of increased cell growth and proliferation. Under nutrient restriction, when growth of most organs shut down, cancer cells can bypass these brakes imposed on cellular growth, thus gaining a growth advantage under these conditions. Furthermore, during cachexia, which causes more than one third of cancer death, tumour derived factors can also induce the breakdown of fat and skeletal muscles, in order to generate metabolic intermediates necessary for the preferential tumour growth. The signaling between tumors and other tissues is highly complex, and the adaptations that allow cancer cells to preferentially activate growth are largely unknown. The student will work within a existing team to discover some of the mediators of cancer cachexia using Drosophila genetics, confocal microscopy, proteomics, metabolomics; the findings will be further validated in human samples.

**Primary Supervisor:** A/Prof Louise Cheng
**Availability:** PhD;Honours

# Gregorevic Group

**Contact:** Prof Paul Gregorevic
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**Location:** Medical Building

Skeletal muscle powers movement, metabolism, and inter-organ signaling, making it vital for life. Reduced muscle function is an important contributor to poor outcomes associated with serious illness, ageing, and injury. As members of the Centre for Muscle Research, our team studies the mechanisms that control muscle attributes in health and disease, with the aim of developing innovative, much-needed, muscle-directed therapeutics. By leveraging our team's extensive expertise across a wide range of techniques (including gene therapies) and strong collaborative networks, the projects offer rich training opportunities for those interested in discovery biology and its biomedical translation.

### Learning from skeletal muscle to treat cancer

Many cancer patients suffer significant complications associated with cachexia (a debilitating wasting syndrome marked by severe loss of muscle and fat), and from metastasis (the spread of cancer to other organs). Building on discoveries by our team, this research theme investigates mechanisms driving cachexia and explores why skeletal muscle is rarely colonized by metastatic cancers. Understanding and manipulating the underlying mechanisms may reveal novel strategies for preventing and treating cancer progression and associated complications in patients with advanced disease.

**Primary Supervisor:** Prof Paul Gregorevic
**Co-Supervisor:** Dr Rachel Thomson, Dr Alastair Saunders, Dr Chris Karagiannis
**Availability:** PhD;MC-BMEDSC ;Honours

### Developing innovative animal and human cell models to study and treat muscular dystrophies

Many neuromuscular disorders lack effective therapies due to the limitations of existing models for studying disease mechanisms. This program uses innovative gene- and cell-based approaches to develop new in vivo and in vitro models. Using these novel modles to accelerate research will help to uncover disease mechanisms and support the development of much-needed therapeutic strategies for neuromuscular conditions.

**Primary Supervisor:** Prof Paul Gregorevic
**Co-Supervisor:** Dr Craig Goodman, Dr Chris Karagiannis, Dr Alastair Saunders
**Availability:** PhD;MC-BMEDSC ;Honours

**Exploring new roles for the TGFβ signalling network as a cause of skeletal muscle disorders, and a target for new muscle therapeutics.**

The TGFβ signalling network regulates skeletal muscle development, adaptation, and repair. This research explores how TGFβ is controlled in muscle during health and disease, and how it influences muscle traits. Using gene delivery, cell culture, animal models, and molecular analyses, we aim to discover new strategies to prevent or treat muscle loss.

**Primary Supervisor:** Prof Paul Gregorevic
**Co-Supervisor:** Dr Craig Goodman
**Availability:** PhD;MC-BMEDSC ;Honours

**Unravelling the mysteries of ubiquitin biology as a regulator of skeletal muscle in health and disease.**

Muscle size and function are vital for health, metabolism, performance, and aging. E3 ubiquitin ligases and De-Ubiquitinases regulate cell homeostasis, but their roles in muscle are unclear. This research investigates which of these genes influence muscle health and disease, aiming to understand their function and develop therapeutic strategies.

**Primary Supervisor:** Prof Paul Gregorevic
**Co-Supervisor:** Dr Craig Goodman
**Availability:** PhD;MC-BMEDSC ;Honours

# Stem Cell and Developmental Biology

# Wells Group

**Contact:** Professor Christine Wells
**Email:** wells.c@unimelb.edu.au
**Location:** Melbourne Brain Centre / Kenneth Myer Building

This project will characterise a group of putative growth factors that haven't been previously described. The student will learn some simple bioinformatic approaches to model protein function and evolution. Using pluripotent stem cells where the factors have been conditionally knocked out, the student will characterise cell growth and differentiation potential, learning stem cell culture and fundamentals of cell and molecular biology.

**Discovery of novel growth factors made by macrophages during tissue repair.**

Students will undertake characterisation of a newly discovered isoform of a growth factor made by macrophages. They will gain experience in basic cell and molecular biology techniques, including culture of pluripotent stem cells, isolation of mRNA and protein, quantitative PCR, flow cytometry and microscopy. Students will also receive training in primer design, and the use of genome browsers. Students will use genetic databases and the scientific literature to investigate potential disease associations with genetic polymorphisms associated with newly discovered growth factors.

**Primary Supervisor:** Professor Christine Wells
**Co-Supervisor:** A/Professor Brooke Farrugia
**Availability:** PhD, Masters, Honours

**Dynamic consent: Exploring community platforms for ethical use of stem cell lines.**

Students will work closely with an interdisciplinary team of researchers spanning stem cell scientists, data curators and social scholars. The project will explore how consent is usually obtained and maintained when a stem cell line is made, and how these consent conditions are shared when the stem cell line is shared. We will examine the attitude of the Australian public/stem cell donors to current consent practises with the aim of building standardised approaches to ethical sharing of stem cell lines in the future.

**Primary Supervisor:** Professor Christine Wells
**Co-Supervisor:** Dr Suzy Butcher
**Availability:** Masters, Honours.

# Hime Group

**Contact:** Prof Gary Hime
**Email:** g.hime@unimelb.edu.au
**Location:** Medical Building

The Hime groups studies regulation of organ development and regeneration using Drosophila as a model organism. Many differentiated but renewable cell types are derived from relatively small populations of dedicated precursors, or stem cells.
The ability to replenish differentiated cells depends on the continued survival and proliferation of their respective stem cell populations. If we are to realise the goals of re-programming tissue differentiation, growing organs for transplantation in vitro, regeneration of damaged organs in vivo and targeted effective treatments for cancer it is essential that we understand the molecules and mechanisms that stem cells utilise for renewal and differentiation. We also utilise Drosophila to model human genetic variants that are suspected to be associated with disease states in order to gain evidence for pathogenicity.

### Identification of novel regulators of stem cell differentiation

We have conducted genetic screens which have identified new mutations that affect the ability of Drosophila male germline and somatic stem cells to differentiate. This project will involve genetic analysis and DNA sequencing to identify genes associated with specific mutations and phenotypic characterization of the mutant to determine the mechanism affecting stem cell differentiation. Techniques will involve genetic manipulation via RNA interference and CRISPR, microdissection and immunostaining of tissues, qRT-PCR analysis of gene expression and confocal microscopy.

**Primary Supervisor:** Prof Gary Hime
**Co-Supervisor:** TBC
**Availability:** PhD;MC-BMEDSC ;Honours

### Drosophila models of human disease

The rapid advances in sequencing of human genomes has identified many variant gene sequences that may be associated with genetic diseases. It can be difficult to unambiguously associate genetic variants with phenotypes without a direct assay. We are using Drosophila to model the effects of genetic variants associated with human disease to determine how the variants affect gene function. This project will involve generation of transgenic strains that express human gene variants to attempt to genetically rescue loss of function alleles, or generation of Drosophila alleles analogous to the human genetic variants via CRISPR. Variant alleles will be phenotyped via immunostaining, confocal microscopy and transcript expression to determine if they are pathogenic in nature.

**Primary Supervisor:** Prof Gary Hime
**Co-Supervisor:** TBC
**Availability:** PhD;MC-BMEDSC ;Honours

**Regulating fertility: identifying new regulators of gamete formation**

Production of spermatozoa and oocytes are highly regulated processes that involes genes interacting with environmental signals to produce spermatozoa from a population of germline stem cells and oocytes from dedicated progenitors. This project will use a variety of genetic techniques (RNA interference, CRISPR, mutant analysis) with advanced microscopy and cellular analysis to identify new regulators of this process. We work with groups studying human infertility to identify genes that affect human male and female fertility and as these genes are often involved in other disease processes they provide warnings for overall human health.

**Primary Supervisor:** Prof Gary Hime
**Co-Supervisor:** TBC
**Availability:** PhD;MC-BMEDSC ;Honours

**Amitosis: Understanding how cells can divide without undergoing mitosis**

We have identified that during Drosophila salivary gland development an increase in cell number occurs without mitotic activity. This is an unusual form of cell division that is specific to polyploid cells, and occurs in greater than 30% of human tumours. We will use Drosophila genetics to discover how this process is regulated and potentially identify new ways of treating tumour growth. These cells also display a variant form of cell polarization and we also seek to understand how this affects secretory tissue function.

**Primary Supervisor:** Prof Gary Hime
**Co-Supervisor:** TBC
**Availability:** PhD;MC-BMEDSC ;Honours

# Smith Group

**Contact:** A/Prof Kelly Smith
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**Location:** Medical Building

The Smith group is focussed on identifying the genetic and cellular regulators of heart development; in otherwords: how do we build a heart? The heart develops by differentiating cells and organising them in a stereotypical pattern to build this organ. The fact that this structure is more or less identical between individuals demonstrates that a tightly controlled genetic program instructs this process. The lab is interested in identifying such genes, determining how they function, and uncovering the cellular processes they regulate.  We use the zebrafish model for much of our discovery-based projects. The zebrafish is an excellent genetic model and the transparency of the embryos and availability of fluorescent transgenic reporter lines permits live imaging of organogenesis. For some projects, we translate our discoveries to mouse models to investigate evolutionary conservation. The long-term objective of the lab is to contribute to our knowledge of how to build a heart, gathering along the way information that will assist bioengineering efforts and help with diagnosis and treatment of genetic-based heart disease.

**Formation of the trabecular layer during cardiac development**

The project is focussed on understanding a specialised tissue in the heart, called cardiac trabeculae. Trabeculae are ridges or protrusions that grow into the lumen of the heart, helping to make it more efficient. Questions around this project include understanding the genetic regulation of how this tissue is patterned, and what signalling cues are needed to both promote it's growth as well as make sure there isn't excessive growth. We use the zebrafish model for much of our research. Methods used in the project will include embryology (of zebrafish), microinjection techniques, drug and chemical treatments, genetic crosses, molecular techniques (such as DNA extraction, PCR, gel electrophoresis, RNA synthesis), phenotypic screening by bright-field and fluorescence microscopy, confocal microscopy, image analysis, and statistical analysis.

**Primary Supervisor:** A/Prof Kelly Smith
**Co-Supervisor:** Veronica Uribe Sokolov
**Availability:** MC-BMEDSC ;PhD

# Stamp & Hao Group

**Contact:** Dr Marlene Hao
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**Location:** Medical Building

Proper development and function of the digestive tract is crucial for good health. Gut function relies on the co-ordinated activity of neural circuits in the enteric nervous system (ENS), a network of neurons and glia located within the wall of the gastrointestinal tract. Our lab focuses on investigating the plasticity of the enteric nervous system and the development of stem cell therapy to treat digestive diseases.

**Modelling Digestive Diseases in vitro**

Neurodevelopmental disorders such as autism spectrum disorder (ASD) and attention deficity/ hyperactive disorder (ADHD) arise from alterations in the development of the central nervous system. Many patients also experience severe gastrointestinal dysfunction, as a major and debilitating comorbidity. However, despite its clinical impact, the biological basis of gut dysfunction in these neurodevelopmental disorders remains poorly understood. We hypothesise that mutations which result in miswiring of the central nervous system also impact the enteric nervous system (ENS), a key network of neurons and glial cells located within the gut that is essential for the control of gastrointestinal motility and digestive function. In this study, we will use novel human pluripotent stem cell (hPSC) technology and transgenic mouse models to examine how enteric neurons develop and wire together. Using hPSC and mouse lines that carry genetic mutations associated with ASD and ADHD, we aim to examine how the ENS becomes directly impacted during embryonic development.

**Primary Supervisor:** Dr Marlene Hao
**Co-Supervisor:** Dr Atefeh Namipashaki
**Availability:** MC-BMEDSC ;Honours

# Eckersley-Maslin Group

**Contact:** A/Prof Melanie Eckersley-Maslin
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**Location:** Peter MacCallum Cancer Centre

The Eckersley-Maslin (EckMas) laboratory investigates how chromatin landscapes shape current and future cell identity. Both embryogenesis and tumourigenesis are associated with changes in cellular phenotypes and functions, a process termed plasticity. Our laboratory uses insights from development, where cell plasticity is tightly controlled and regulated, to get new insights into how cell plasticity is adopted by cancers to promote therapy resistance, metastases and relapse.

**Investigating novel epigenetic regulators using embryonic stem and cancer models**

Epigenetics helps define current cell states, yet also shapes how cells respond to external cues such as differentiation or stress. The epigenetic plasticity of a cell describes how flexible this regulation is. Early embryonic cells are highly plastic in that they are able to generate all adult cell types. As development progresses, this plasticity is lost as normal healthy adult cells are locked in their identity. Crucially, aberrant reactivation may contribute to pathologies such as cancer. Our laboratory explores how epigenetic plasticity is controlled and regulated in development, applying these principles to understand how it is exploited by cancers.

This project will investigate poorly understood epigenetic regulators in developmental biology and/or cancer cell models. The project will use high throughput CRISPR screens to systematically assess functional importance of these epigenetic regulators in embryonic stem cell and/or cancer cell models. Subsequent molecular experiments will uncover the mechanism and functional significance of these epigenetic regulators in shaping developmental and/or cancer processes. The prospective student will have the opportunity to use a range of cutting-edge technologies including chromatin assays, CRISPR-Cas genome engineering, epigenomic and transcriptomic analyses, cell culture and molecular biology techniques. The project is suitable for a student wishing to be solely lab based or those who also wish to do both experiments and bioinformatic analysis.

**Primary Supervisor:** A/Prof Melanie Eckersley-Maslin
**Co-Supervisor:** tbc
**Availability:** PhD; honours

**AI-designer peptides targeting regulators of cancer cell plasticity**

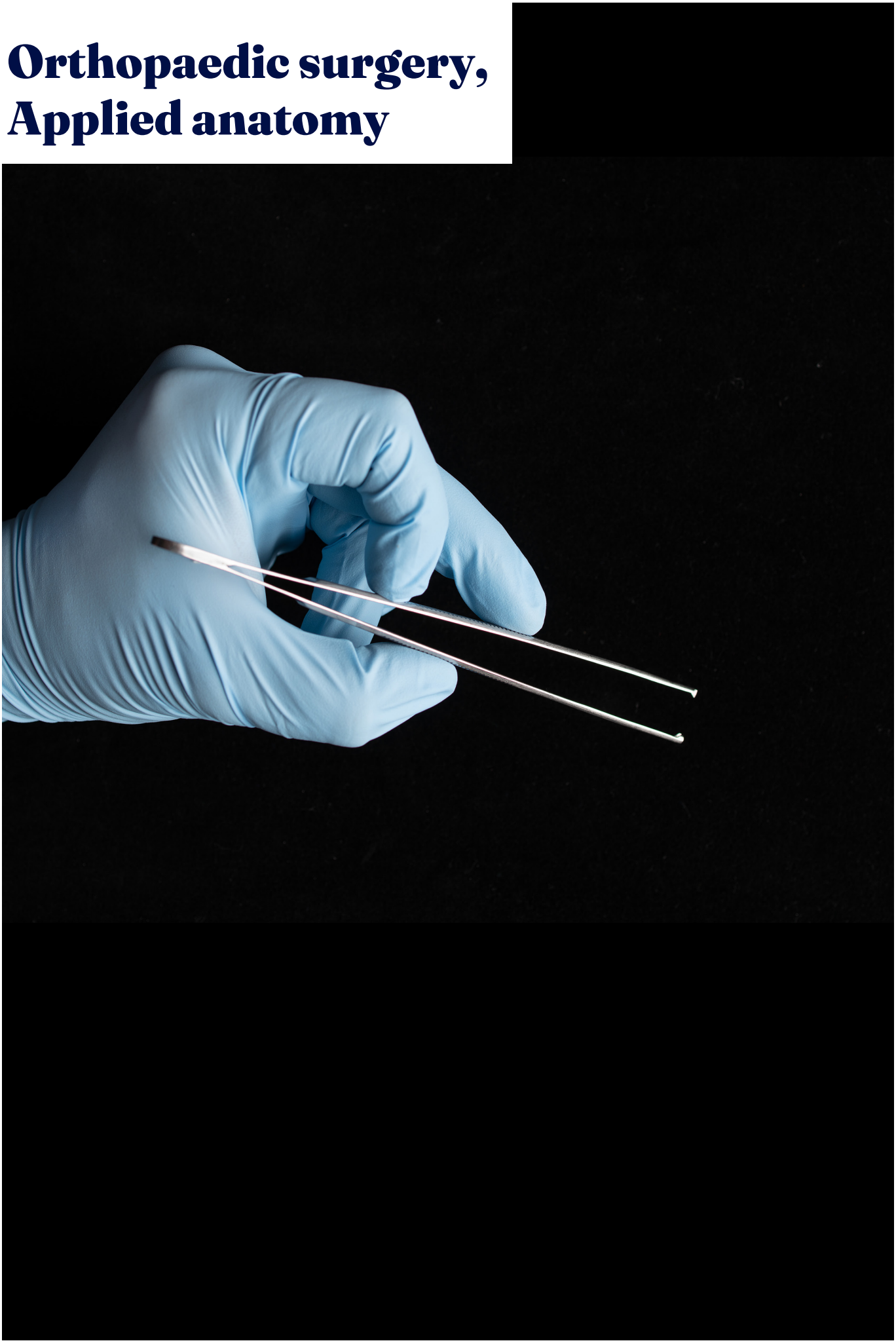
Cancer cell adaptation remains a major challenge in successfully treating cancers. The heightened plasticity of cancer cells enables them to rapidly change their phenotypes, often without acquiring additional genetic mutations. Plasticity is proposed to facilitate metastasis, immune evasion and treatment resistance. We have recently identified a series of putative upstream regulators of cancer cell plasticity. The aim of this project is to establish a pipeline to design and develop peptides that selectively bind these regulators that may be further developed as lead drug compounds.

This project will use available or computationally generated 3D protein structural data of the target regulators to identify residue hotspots that can be pursued for selective binding. Hotspots will then be defined as inputs to generate peptides and/or mini-proteins using AI-accelerated design for specific targeting of the regulator to block its function. Expression libraries encoding these designer peptides fused to E3 ubiquitin ligase binding moieties will be used to screen for candidate PROTAC prototypes by flow cytometry in fluorescence reporter cell lines generated as part of this project. The final novel peptide prototypes can then be studied further using both in vitro and in vivo assays to assess its applicability as a novel drug prototype targeting cancer cell plasticity.

The project will require someone with strong foundations in molecular biology and cell biology with a strong conceptual understanding of structural biology. A strong knowledge of gene regulation and chromatin biology is essential. The project will involve both computational biology and experimental laboratory work. The project is part of a collaboration between the Eckersley-Maslin and Knott laboratories and will be based across both Peter MacCallum Cancer Centre and Monash Biomedicine Discovery Institute.

**Primary Supervisor:** A/Prof Melanie Eckersley-Maslin
**Co-Supervisor:** A/Prof Gavin Knott
**Availability:** PhD

# Orthopaedic surgery, Applied anatomy



# Ackland Group

**Contact:** Prof David Ackland
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**Location:** Department of Biomedical Engineering

Orthopaedic Research Laboratory

**Elbow fracture and reconstructive surgery**

The elbow joint is essential for undertaking most activities of daily living. Fractures of the coranoid process of the elbow are most often associated with elbow dislocation; however, the role of surgical repair on post-operative joint stability remains poorly understood. The aim of this study is to employ a cadaver model to assess joint stability in the elbow following sequential coronoid fractures, and assess the effectiveness of plating fixation in restoration of elbow function. Testing will be undertaken using a testing rig that simulates elbow joint function by simulated force application. The results of this study will inform clinical practice in the treatment of coranoid fracture.

**Primary Supervisor:** Prof David Ackland
**Co-Supervisor:** A/Prof Lukas Ernstbrunner; Dr Kate Hatzopoulos
**Availability:** PhD:Honours

THE UNIVERSITY OF MELBOURNE

# Get in touch

If you're considering studies at the University of Melbourne,
we'd love to hear from you online or meet you on campus.

Sign up and submit enquiries online at: https://biomedicalsciences.unimelb.edu.au/departments/anatomy-and-physiology



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