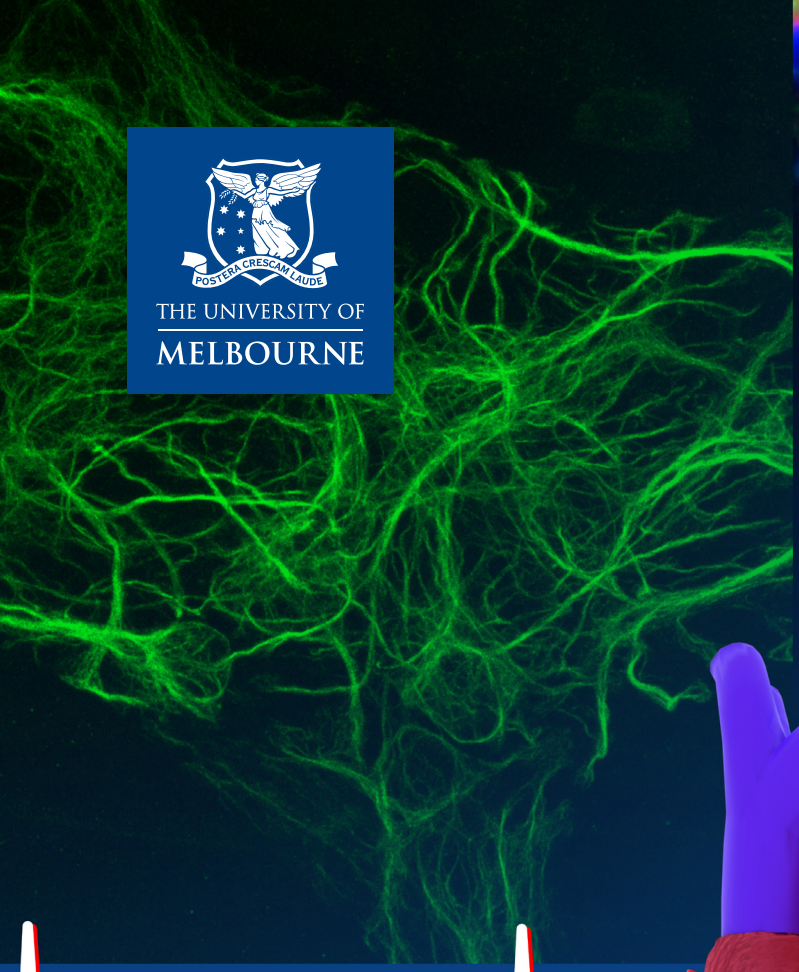
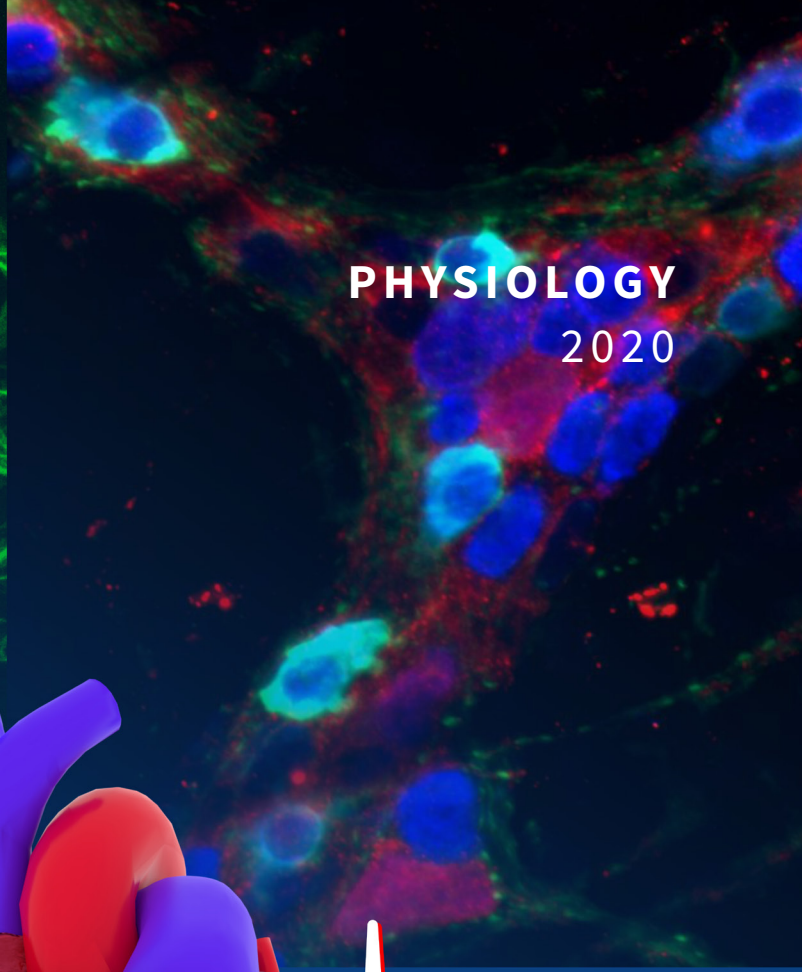
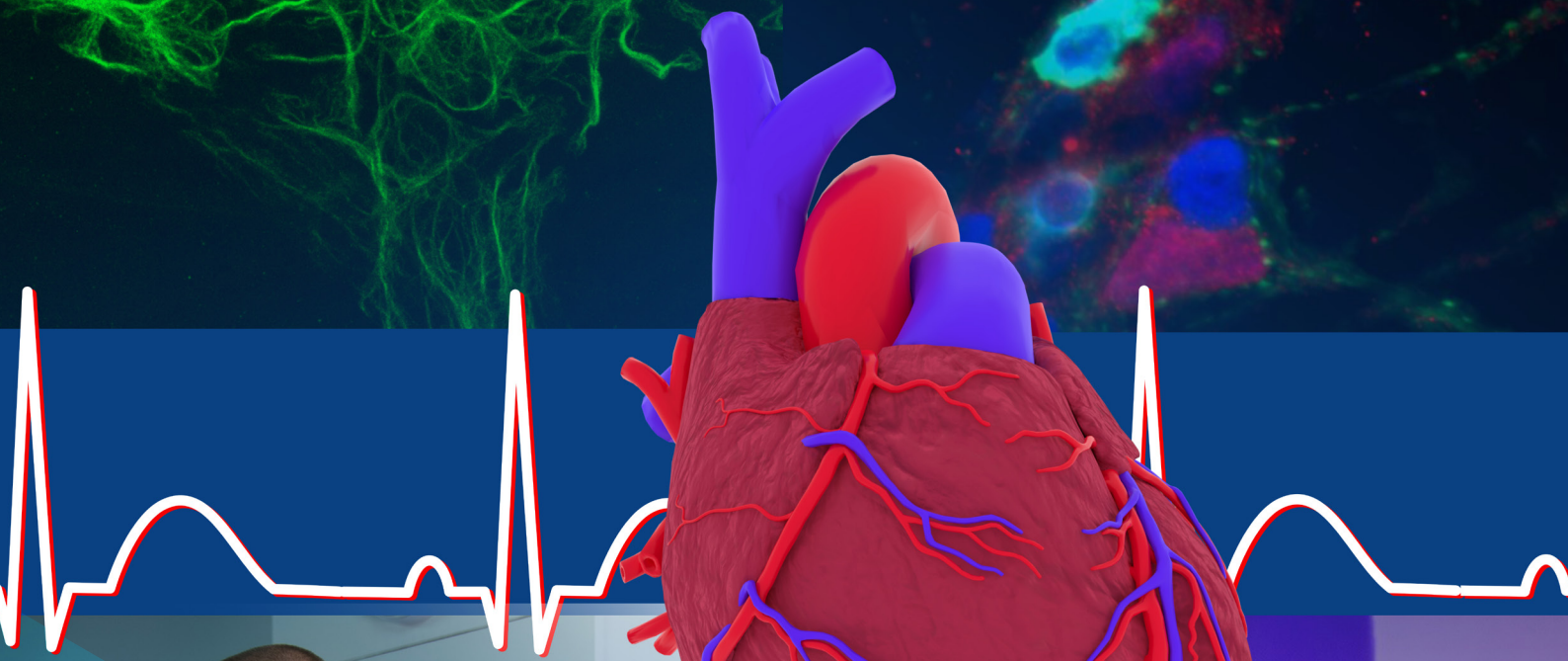
THE UNIVERSITY OF MELBOURNE

PHYSIOLOGY
2020







RESEARCH PROJECTS
HONOURS, MASTERS AND PHD

WELCOME

The decision to undertake an Honours year or a Masters degree is an important one that provides substance to your undergraduate degree. You get to see up close the workings of a research laboratory and gain the ability to put your scientific knowledge into practice.

It can be the first step towards an independent scientific career when you get the chance to pursue a research area of interest. This is likely to be the path for a small minority, but the skills learnt are valuable in many areas of life and in multiple careers. The extra qualification will also help set you apart from competitors when seeking employment or entry into other courses or specialties.

There are many things to consider – the research topic, your need and desire to undertake additional, advanced coursework, the laboratory and its resources, the potential supervisor and the departmental support of students.

The Department of Physiology has a strong record of award-winning research training and mentorship with our graduates securing leadership roles in universities, institutes, industry and in the private sector. We are very proud of our students and have developed a carefully structured program of coursework to complement your developing laboratory and analytical skills. The Department environment provides support in a number of ways for our Honours and Masters students, but perhaps none is more important than the friendship, advice and mentoring they receive from other graduate students.

This booklet provides information that will help you decide on a potential research project in Physiology at Honours and Masters Level and perhaps beyond to your PhD.

Our research is focused on themes related to cardiovascular health, neurophysiology, and skeletal muscle and exercise, and metabolism in health and disease. Take your time and look at the different projects on offer. Identify projects that appeal to you and contact potential supervisors for more information and visit their laboratories. Ask lab heads, staff and students about the projects and your potential career options with the new qualification. Be assured, supervisors are very interested in talking to you and you should be confident in making that approach.

Physiology offers many exciting research opportunities and we welcome the chance to discuss these with you.

Professor Matthew Watt

Head of Department

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 75points of research and 25points 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: https://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 Masters of Biomedical Science?

The Masters 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: https://study.unimelb.edu.au/find/courses/graduate/master-of-biomedical-science/how-to-apply/

Difference between Honours and the Master of Biomedical Science

	Honours	Masters
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

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% (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 (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/physiology

How to apply

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

SCHOLARSHIPS

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 an 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 Physiology in 2020 you can apply for a Summer Research Studentship to work on a supervised research project. The studentship provides a small living allowance ($2,000) to enable you to work on a laboratory-based project for a period of 5 or more weeks over the summer break. The purpose of the Summer Research Studentships is to provide an opportunity for undergraduates to gain first-hand experience in research.

Over the 2019/2020 summer break, up to four Studentships 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.

Application forms are available from the Department of Physiology. Please contact our Department by emailing Physiology-Info@unimelb.edu.au to request a copy. The deadline for application is the 22nd of November, 2019.

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

CARDIOVASCULAR HEALTH

DELBRIDGE GROUP

Contact: **Prof Lea Delbridge**
Email: **lmd@unimelb.edu.au**
Location: **Department of Physiology**

Cardiovascular

Translational and Clinical Research

Discovery research

The Cardiac Phenomics Laboratory research is about understanding 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 signaling 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).

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 to understand how for women and men, cardiac 'difference' may be managed with optimized therapeutic tools.

Student projects in the Cardiac Phenomics lab 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 assay, confocal microscopy, microarray gene profiling, realtime 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.

Project: Understanding glycogen dysregulation in diabetic heart failure

Globally, diabetes is an epidemic disease with a specific cardiopathology independent of associated cardiovascular risk profiles. Diabetic hearts are more vulnerable to developing failure, especially after a myocardial infarct. Our work focuses on understanding the metabolic and structural changes leading to both diastolic and systolic dysfunction, examining how circulating glucose and insulin impacts on cardiopathology and identifying potential molecular targets for intervention. This project will utilise experimental models of type 1 and type 2 diabetes to investigate the molecular and structural adaptations in the diabetic heart. We use cutting edge gene delivery and gene editing tools to create boutique experimental models of disease.

Project supervisor
Professor Lea Delbridge

Project co-supervisor
Dr Kimberley Mellor

- PhD
- Master of Biomedical Science

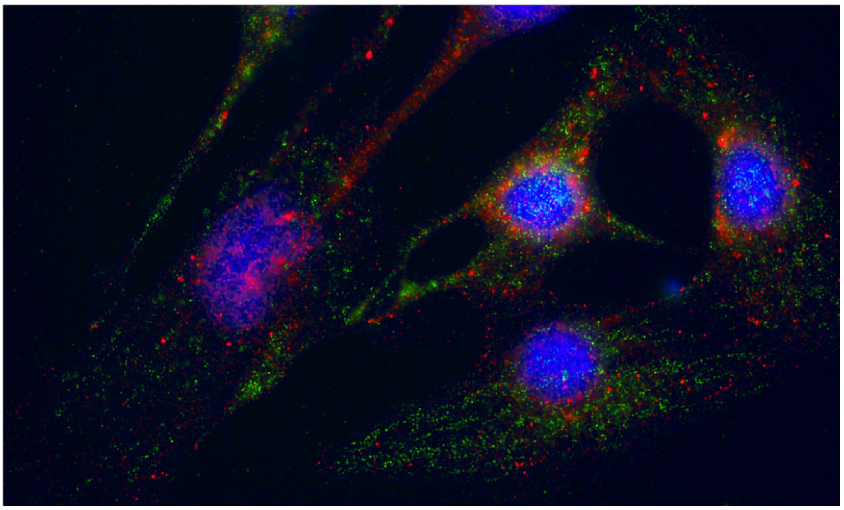
Project: Is a 'fat heart' an especially vulnerable heart?

Maintaining normal rhythm properties is essential to heart function. Sustained arrhythmias (including atrial fibrillation) increase significantly with aging and in obesity. Often evident in otherwise 'healthy' asymptomatic patients, these sustained arrhythmias represent a primary component of cardiac demise. Understanding the cellular mechanisms driving arrhythmias is crucial to developing new effective therapies. Recent evidence has emerged indicating that accumulation of the fat around the heart (pericardial adipose) may be crucial to the development of sustained arrhythmias in the aged/obese population. Pericardial adipose levels are known to increase markedly in obesity, with aging, and in post-menopausal women – all important risk factors for cardiovascular disease. Our very recent data indicate that pericardial adipose may release proteins that exert a paracrine effect on the heart muscle to increase vulnerability to arrhythmias. This project will use molecular and tissue recording studies of human and rodent tissues to further understand how cardiac adipose contributes to the development of cardiac arrhythmias.

Project supervisor
Professor Lea Delbridge

Project co-supervisor
Dr James Bell

- PhD
- Master of Biomedical Science



Project: Defining the molecular mechanisms underlying heart failure

Among the growing number of patients with heart failure, as many as half have heart failure with preserved ejection fraction (HFpEF). In this condition the signature symptoms relate to relaxation abnormality in diastole, which limit the cardiac output due to compromised ventricular filling. To date, clinical trials have yet to find an effective and specific treatment for this condition. This project will utilise our unique experimental models of HFpEF to investigate the underlying molecular changes that occur with HFpEF and aims to identify therapeutic targets for this disease. We work with clinical partners in developing specialized imaging methods to benchmark laboratory derived measures of diastolic dysfunction against patient characteristics.

Project supervisor
Professor Lea Delbridge

Project co-supervisor
Dr Claire Curl

- PhD
- Master of Biomedical Science

Project: 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 clinical biopsy samples, as part of an associated project to develop biomarkers for early detection of cardiomyopathic disease.

Project supervisor
Professor Lea Delbridge

Project co-supervisor
Dr Kimberley Mellor

- PhD
- Master of Biomedical Science

Project: Understanding the role of locally synthesized steroids in the heart

Important differences exist between women and men with regard to cardiovascular disease. This is likely related to sex steroid (estrogen and testosterone) actions on the heart. However, recent controversies about the use of sex steroid therapies in men and women highlight a lack of understanding of the underlying mechanisms by which sex and sex steroids influence the heart. We have recently shown in humans that both the myocardium and cardiac adipose express the enzyme aromatase – showing that estrogen synthesis can actually occur within the myocardium. In aging/obesity, when the onset of cardiovascular disease is prominent, the influence of this locally-synthesised estrogen likely increases. This project will use molecular and tissue recording studies of human and rodent tissues to determine how estrogens synthesised within the heart contribute to the development of cardiac rhythm and relaxation abnormalities.

Project supervisor
Professor Lea Delbridge

Project co-supervisor
Dr James Bell

- PhD
- Master of Biomedical Science

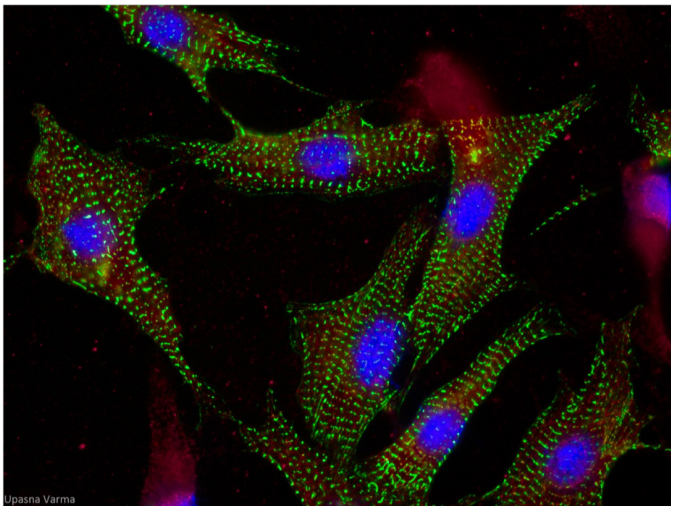
Project: Investigating sex differences in heart failure

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. There are few animal models of HFpEF currently available and those that are utilised generally investigate male animals only. We have used our unique model of HFpEF to produce preliminary experimental evidence which suggests that the cellular mechanisms underlying this disease are different in males and females. This project will expand and extend these findings to evaluate sex differences in the cellular and molecular mechanisms of HFpEF and aims to identify sex specific therapeutic targets for this disease.

Project supervisor
Professor Lea Delbridge

Project co-supervisor
Dr Claire Curl

- PhD
- Master of Biomedical Science



Project: Targeting altered cardiac glucose metabolism in the cardiac complications of diabetes

The increasing global prevalence of type 2 diabetes (T2D) and our aging population has given rise to an epidemic of heart failure (HF). Up to one-third of patients in clinical HF trials are diabetic, and diabetes is an independent predictor of poor outcome. Despite the higher rate of HF in these patients, no specific treatment for HF exists for T2D patients. We have identified novel mechanisms for limiting T2D-associated cardiomyopathy that could pave the way for the development of much needed, novel therapies that are specific for diabetic HF. Increased glucose flux through the hexosamine biosynthesis pathway (HBP) has now emerged as a key mediator of the adverse effects

of diabetes on the heart. As a result of this HBP overdrive, increased cardiac levels of the glucose metabolite called O-GlcNAc increases susceptibility of a range of proteins to O-GlcNAc modification, altering their function. We propose that this route of glucose metabolism impairs left ventricular (LV) function, and will focus in particular on O-GlcNAcylation of key components within the cardiomyocyte. The aim of this project is to demonstrate that cardiac-directed therapeutic targeting of this ROS-hexosamine biosynthesis axis delays or even overcomes diabetes-induced

cardiac dysfunction in the intact heart in vivo, and to investigate susceptibility of specific components within the cardiomyocyte to O-GlcNAcylation, and how this impacts on diabetes induced HF.

Project supervisor
Professor Lea Delbridge

Project co-supervisor
Professor Rebecca Ritchie

- PhD
- Master of Biomedical Science

Project: Nitroxyl-based therapies to overcome diabetes-induced impairments in cardiac nitric oxide signalling

In patients with cardiovascular disease, impaired NO• signalling is an independent predictor of poor outcomes, including mortality. This loss of NO•-responsiveness (termed 'NO•-resistance') is particularly debilitating in type 2 diabetes (T2D), where cardiovascular emergencies occur more frequently, but where NO•-based pharmacotherapies are less effective. Identifying strategies to circumvent cardiovascular complications in the diabetic heart and vasculature, both in an acute emergency situation and over the longer-term, will improve prognosis in these patients. We have identified an exciting potential strategy for circumventing this impaired NO• signalling, utilizing the novel NO-like molecule, nitroxyl (HNO). Ultimately, HNO- based strategies may offer new treatment options for cardiac disease, particularly T2D. The aim of this project is to determine the extent of NO resistance in T2D, and whether HNO can overcome this in the short and longer-term. HNO may be superior to NO with respect to limiting diabetes-induced myocardial dysfunction and changes in cardiac structure, key characteristics of diabetes-induced heart failure.

Project supervisor
Professor Lea Delbridge

Project co-supervisor
Professor Rebecca Ritchie

- PhD
- Master of Biomedical Science

PORELLO GROUP

Contact: **Dr Enzo Porrello**

Email: **enzo.porrello@unimelb.edu.au**

Location: **Murdoch Children's Research Institute**

Website: **https://biomedicalsciences.unimelb.edu.au/sbs-research-groups/physiology/Porrello-lab-Cardiac-Regeneration**

https://www.mcri.edu.au/heartregeneration

https://www.ncbi.nlm.nih.gov/pubmed/?term=porrello+er

	Cardiovascular
	Translational and Clinical Research
	Discovery research
	Genomics

The Cardiac Regeneration Group is focused on the development of novel therapeutic approaches for congenital and acquired forms of heart disease based on a deep understanding of developmental and regenerative biology. Our laboratory uses a variety of approaches including molecular genetic studies in mice through to single cell transcriptomics, gene editing (CRISPR/Cas9) in human pluripotent stem cells, as well as disease modelling and drug screening in human cardiac organoids. We are working closely with clinicians and scientists both nationally and across the Melbourne Children's precinct to foster knowledge transfer and translation of research discoveries from bench to bedside.

Project: Modelling heart disease in a dish using patient-derived stem cells and drug screening.

One key area of our heart research focuses on creating stem cells from patients with congenital heart disease and recreating their heart tissue in our laboratories. This allows us to recreate and study their disease more closely – this method of research is called disease modelling. If we are able to determine a genetic cause for the disease through studying a patient's tissue in the laboratory, we now have the gene editing capability and technology to correct mutations found in that patients' genome. By comparing a patient's genetically mutated and corrected cell lines, we are able to better understand a disease's cause and progression, which informs our understanding of any potential preventative measures, tests for that disease, developing new treatments and hopefully cures. We are also investigating whether iPS-derived cardiac organoids can be used to screen for drugs that promote regeneration of cardiomyocytes in children with heart disease. This project will develop iPS disease models of congenital heart disease for high-content screening to discover novel disease mechanisms and potential drug targets. PhD students will develop skills in iPS cell culture and differentiation including 3D organoids, gene editing, microscopy, transcriptomics/proteomics and cardiomyocyte physiology.

High-content screening will be facilitated by the development of genetically encoded reporters to assess calcium handling, electrophysioloigcal properties, cell cycle status and biomechanical forces in iPS-derived cardiomyocytes. Candidate genes/pathways identified in the screen will be further validated and characterised using sophisticated genetic, biochemical and physiological approaches including gain/loss of function, contractility assays in 3D organoids and transcriptomics approaches (including single cell profiling).

Project supervisor
Dr Enzo Porrello

Project co-supervisor
Dr David Elliott

- PhD
- Master of Biomedical Science

WLODEK GROUP

Contact Name: **Prof Mary Wlodek**
Email address: **m.wlodek@unimelb.edu.au**
Location: **Department of Physiology**
Website: **http://go.unimelb.edu.au/e7hr.**

Metabolism	**Cardiovascular**
Muscle	**Neurophysiology**
Exercise	**Translational and Clinical Research**

Small birth size affects 10% of pregnancies, which increases the risk of adult diseases. The Wlodek laboratory explores how impaired organ development programs cardiorenal and metabolic diseases in rat offspring born small. We are currently exploring the impact and timing nutritional, exercise and lifestyle interventions have on disease outcomes, and whether this disease is transmitted to the next generation. Our studies will provide the scientific basis for the design and testing of appropriately targeted lifestyle interventions to reduce adverse health effects in individuals born small, which is increasingly important due to the ever-increasing incidence and earlier age of diseases onset.

Project: Impact high-fat feeding and endurance exercise training has on physiological and behavioural outcomes in male rats born small and second-generation offspring

Many experimental and human studies worldwide have shown that babies born small for gestational age (or who are light at birth) are strongly and consistently at an increased risk of developing behavioural, cardiovascular and metabolic diseases as adults, and that this risk is passed onto subsequent generations. Being overweight or obese can exacerbate these consequences. The relative role of paternal transmission is less well understood. We have demonstrated in our model of uteroplacental insufficiency that the fetal growth restriction is associated with hypertension, a nephron deficit and metabolic dysfunction. The aim of this study is to determine whether the behavioural, cardiorenal and metabolic phenotypes in males born small and their offspring are exacerbated with high-fat feeding and whether endurance exercise can prevent the emergence of disease.

Project supervisor
Prof Mary Wlodek

Project co-supervisor
Dr Jessica Griffith

- PhD
- Master of Biomedical Science
- Honours

Project: Impact treatments for gestational diabetes have on maternal, fetoplacental and offspring outcomes.

Gestational diabetes mellitus (GDM) is one of the most common pregnancy complications that has profound health risks beyond pregnancy for the child. As many women do not adopt healthy lifestyles after GDM diagnosis, pharmaceutical treatments are often required. While commonly used clinically, the long-term effects of these treatments for GDM on child health are surprisingly unknown. Without knowledge of benefits or risks associated with these treatments for GDM on their child's short-term (fetal and placental health) and long-term health, women are less likely to comply with taking pharmaceuticals during pregnancy, despite the short-term benefits for their health. The aim of this proposal is to determine the impact GDM treatments during pregnancy have on short-term (fetal and placental) and long-term maternal and offspring outcomes.

Project supervisor
Prof Mary Wlodek

Project co-supervisor
Dr Jessica Griffith

- PhD
- Master of Biomedical Science
- Honours

SMITH GROUP

Contact: : **Kelly Smith**
Email: **kelly.smith1@unimelb.edu.au**
Location: **Department of Physiology**

Cardiovascular

Discovery research

The focus of the Smith group is to identify the genetic and cellular processes that regulate heart development. The heart develops by differentiating and integrating multiple tissue types via a specific sequence of events to generate the stereotypical structure of the 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 the genes in this program, 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 particularly important projects, we translate our discoveries to the 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.

Project: Characterisation of novel genes in cardiac morphogenesis

To identify genes required for cardiac development, the lab has undertaken a forward genetic screen in zebrafish and screened for mutants with cardiac defects. This process involved mutagenizing animals, inbreeding to isolate recessive mutants with inherited heart abnormalities and mapping the causative mutation, identifying which genes are important for cardiac development. From this screen, we have identified several novel mutants and the affected genes are either completely novel or have not been previously implicated in heart development. The project will involve characterising the nature of the cardiac defect, the timing of the onset of the heart defect and may involve one or more of the following: determining the genetic pathway the gene functions in, which tissue the gene is expressed in, which cellular compartments are disrupted in mutants and whether the phenotype/s can be rescued by modification of downstream components.

Project supervisor
A/Prof Kelly Smith

- PhD
- Master of Biomedical Science
- Honours

Project: Investigating left-right patterning of the heart

The heart is an asymmetric organ. Not only is it positioned on the left side of the body (and is therefore subject to overall left-right axis patterning in the embryo) but it possesses asymmetry intrinsic to the organ itself. Symmetrical organs in humans form a collection of diseases called heterotaxia and, for the heart, symmetry causes improper connections with the major vessels, valve defects and poor cardiac chamber development. These defects are debilitating and require multiple surgeries to correct and can be fatal. These defects arise at the very early stages of heart development. The heart begins as a simple symmetrical tube and asymmetry is imposed as the heart twists and bends to form what is called the “looped heart”. This asymmetric morphogenesis always occurs with left-right bias in the same direction and is, therefore, patterned genetically. The lab has identified an early left-right asymmetry that precedes asymmetric looping of the heart and we believe is instructive to directional cardiac looping. We have developed a number of transgenic, genetic and chemical tools to study this process.

Project supervisor
A/Prof Kelly Smith

- PhD
- Master of Biomedical Science
- Honours

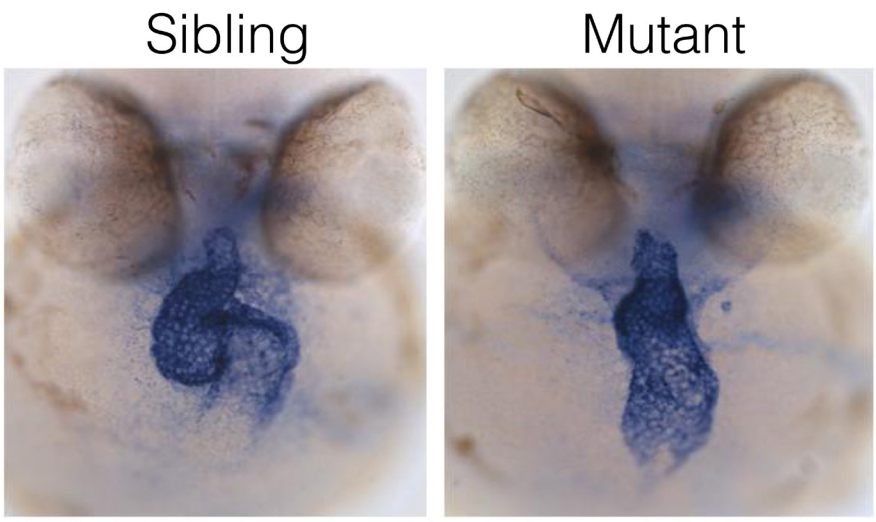
Double transgenic zebrafish embryo at two days old. Green: myocardium (muscle), Red: endocardium & vasculature

Project: Mammalian coronary vascular development and its interaction with the extracellular matrix

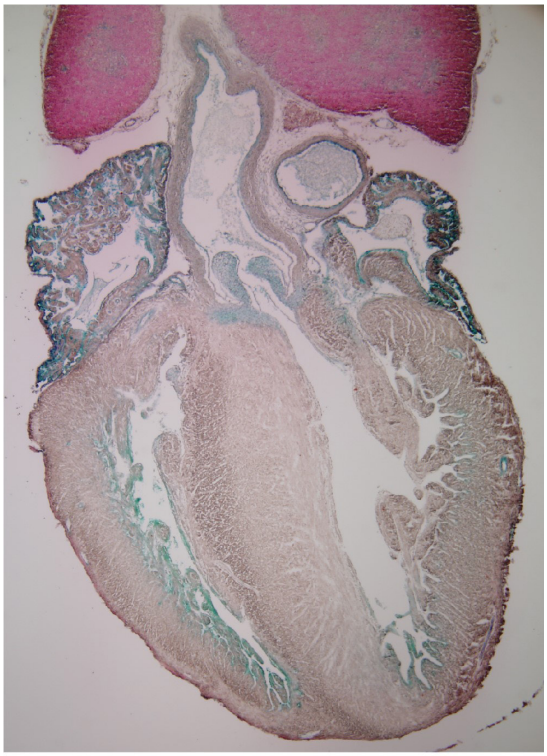
The heart is a large and highly metabolic organ that requires its own blood supply to continue to respire and function. The coronary vasculature is a specialised network of blood vessels that carries oxygenated and deoxygenated blood to and from the heart. Cardiac arrest or myocardial infarction occurs due to occlusions of the coronary vasculature. It is the leading cause of death in the western world, providing a testament to how important this vascular network is. Blood vessels grow via sprouting angiogenesis, invading tissues that need a blood supply. This growth is dependent on growth factor signalling and growth factor signalling involves modification of the extracellular matrix. As the name suggests, the extracellular matrix (or ECM) exists outside the cell and is often described as a scaffold or network for cell-cell communication and for cells to adhere to. The ECM is composed of many different components, one of which is Hyaluronic Acid. We have identified a new enzyme that degrades Hyaluronic Acid and have shown an early role in embryonic angiogenesis in the trunk of the zebrafish embryo. We hypothesise this enzyme is essential for coronary vascular formation and have generate a mouse model to study this. The project will involve the analysis of mouse embryonic hearts to determine how the coronary vasculature is developing under normal and conditions of disturbed Hyaluronic Acid turn-over.

Project supervisor
A/Prof Kelly Smith

- PhD
- Master of Biomedical Science
- Honours



In situ hybridisation stain outlining the heart and showing a looped wildtype heart (sibling) and an unlooped (mutant) heart



1 day old mouse heart, LacZ-stained for the expression of an enzyme that degrades the extracellular matrix

METABOLISM

WATT GROUP

Contact Name: **Matthew Watt**
Email: **matt.watt@unimelb.edu.au**
Location: **Department of Physiology**

Metabolism

Translational and Clinical Research

Discovery research

Our innovative research program seeks to identify how defects of lipid metabolism and inter-tissue communication cause obesity-related disorders, including type 2 diabetes and non-alcoholic fatty liver disease (NAFLD). We use this information to discover novel targets that can be transitioned to clinical therapeutics. Our research themes are:

1. Understanding how insulin resistance develops in obesity.
2. Understanding how proteins that are secreted by NAFLD / non-alcoholic steatohepatitis (NASH) liver affect metabolism and contributes to the development of type 2 diabetes.
3. Regulation of lipid metabolism: identifying novel proteins that control lipid metabolism and how they are altered in metabolic diseases (e.g. diabetes, cancer).

Project: Discovery of new proteins that lead to the development of type 2 diabetes

A major goal of our research program is to understand how obesity changes liver function and how this contributes to the development of type 2 diabetes. An excessive accumulation of fat in the liver is known as non-alcoholic fatty liver disease (NAFLD) and occurs in 70% of obese individuals, with up to 30% of those individuals progressing to the more severe disease state known as non-alcoholic steatohepatitis (NASH). We aim to understand how proteins termed 'hepatokines' are secreted by the NAFLD/NASH liver affect metabolism in tissues of the body, and how this contributes to the development of type 2 diabetes.

We have previously used a high-throughput screen to identify several proteins whose secretion is increased in NAFLD and NASH and we now aim to determine whether these proteins affect (1) glucose metabolism and blood glucose control, (2) insulin sensitivity and (3) lipid metabolism in skeletal muscle, liver, adipose tissue and the pancreas.

In this project, you will evaluate the effects of newly identified hepatokines on muscle, liver and fat cell metabolism and extend these studies to mouse models of pre-diabetes and diabetes.

Project supervisor
Prof. Matthew Watt

- PhD
- Master of Biomedical Science
- Honours

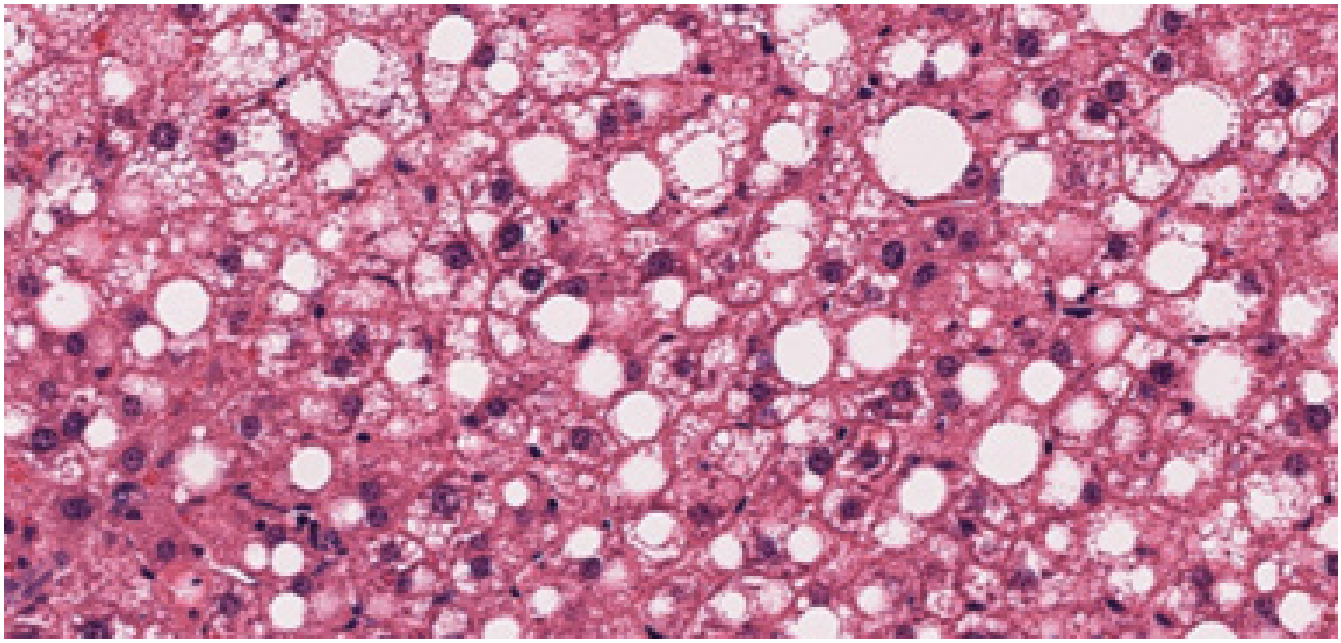


Image showing liver section from a patient with fatty liver disese. Note the abundance of lipid droplets (white circles).

Project: New ways to improve metabolism: understanding mitochondria and lipid droplet interactions in health and disease

Although many biology text books indicate that organelles, such as mitochondria and lipid droplets, are static within cells, recent discoveries have transformed this view and show dynamic interactions between organelles in the same 'neighbourhood'. Mitochondria are critical for generating energy, lipid droplets provide the fuel for mitochondrial energy production and these organelles come into close contact, particularly during metabolically demanding situations. However, we do not know how and why mitochondria are in physical contact with lipid droplets. We aim to test the hypothesis that the inter-organellar interaction of mitochondria and lipid droplets is essential for normal energy metabolism and that this process is dysregulated in metabolic diseases such as obesity and diabetes. The student in this project will identify novel proteins that are essential for mitochondria-lipid droplet interactions and determine their metabolic consequences. This will be achieved with proteomic profiling (with space and time resolution), by generating knock-out cell lines using state-of-art genetic editing tool CRISPR-Cas9, imaging of cells using super-resolution microscopy and performing detailed assessment of metabolism. The results of these studies will provide new information regarding the regulation of cell metabolism, information that could be harnessed to develop new therapies for metabolic diseases.

Project supervisors
Prof. Matthew Watt

Co supervisor
Dr. Ayenachew Bezawork-Geleta

- PhD
- Master of Biomedical Science
- Honours

Project: Chewing the fat: characterisation of protein-protein interactions regulating lipid metabolism

Defective lipid (fat) metabolism is associated with the development of many diseases, including type 2 diabetes, cardiovascular disease and fatty liver disease. Lipids are contained within specialised organelles called 'lipid droplets' and are required for survival. It is known that proteins located on the surface of lipid droplets can regulate lipid synthesis and lipid breakdown. We have recently discovered several novel proteins at the surface of the lipid droplet and the aim of this project is to determine their role in regulating metabolism. This will be achieved by using state-of-the-art protein labelling techniques, confocal microscopy to assess protein-protein interactions and detailed assessment of metabolism. Targeting lipid droplet proteins will provide insight into new treatment strategies for metabolic disease.

Project supervisors
Prof. Matthew Watt

Co supervisor
Dr. Stacey Keenan

- PhD
- Master of Biomedical Science
- Honours

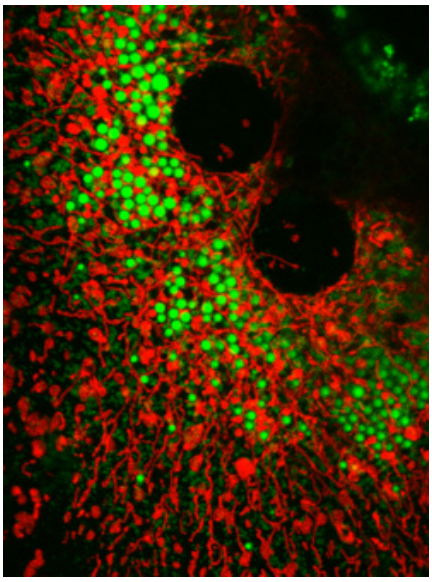


Image showing lipid droplets (green) in close association with mitochondria (red) in liver cells.

Project: Awkward conversations: understanding how exosomes from fatty liver cause metabolic dysfunction

Non-alcoholic fatty liver disease (NAFLD) and type 2 diabetes are common co-morbidities, suggesting there may be communication between these two conditions. Exosomes are small vesicles that contain a variety of proteins, miRNAs, and lipids that can be delivered to peripheral cell types and alter recipient cell function. We have preliminary evidence showing that exosomes secreted by fatty liver differ from healthy control mice, suggesting that changes in exosome secretion might drive metabolic dysfunction in NAFLD.

In this project, you will investigate the role of exosomes in regulating metabolism and insulin resistance in cultured cells and mice. These studies will provide valuable new insights into the pathogenesis of metabolic diseases such as type 2 diabetes.

Project supervisors
Prof. Matthew Watt

Co supervisor
Dr. Paula Miotto

- PhD
- Master of Biomedical Science
- Honours

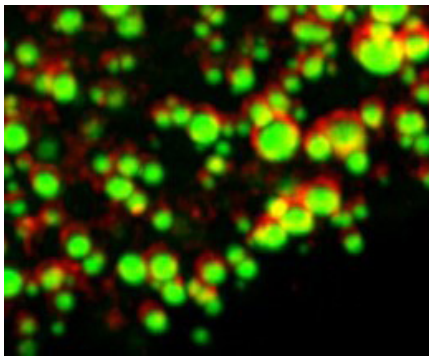


Image of lipid droplets (green) within liver cells in close contact with a protein that regulates lipid metabolism (red).

MONTGOMERY GROUP

Contact Name: **Magda Montgomery**
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Location: **Department of Physiology**
Weblink: **http://go.unimelb.edu.au/u7hr.**

Metabolism

Translational and Clinical Research

Discovery research

One major aim of our research stream is to understand how the liver contributes to metabolic disease - ie. obesity, glucose intolerance, and insulin resistance. Non-alcoholic fatty liver disease (NAFLD, fat accumulation in the liver) is found in 70% of obese individuals, with up to 30% of those further progressing to the more severe disease state - non-alcoholic steatohepatitis (NASH), which is characterized by liver inflammation, hepatocyte ballooning and significant fibrosis. We aim to understand what proteins a fatty liver secretes and if these proteins contribute to changes in systemic metabolism (Can a fatty liver drive type 2 diabetes?).

Project: Understanding the effects of NASH-secreted factors on glucose and lipid metabolism

In a high-throughput screen we identified several proteins whose secretion was increased in the presence of NASH and we now aim to determine if these proteins affect (1) glucose metabolism and glycaemic control, (2) insulin sensitivity and (3) lipid metabolism in the key peripheral tissues of metabolism, including skeletal muscle, liver, adipose tissue and the pancreas.

Project supervisor
Dr Magda Montgomery

- Master of Biomedical Science
- Honours

DODD GROUP

Contact: **Garron Dodd**
Email: **garron.dodd@unimelb.edu.au**
Location: **Department of Physiology**

Metabolism	**Translational and Clinical Research**
Molecular mechanisms of disease	**Discovery research**
Biomedical neuroscience	**Proteomics**

Metabolic diseases, such as obesity and type-2 diabetes, represents one of the biggest biomedical challenges of our time. With the ever-increasing metabolic disease epidemic and the insurmountable costs of treating comorbidities (cancer, cardiovascular disease and stroke), there has never been a more desperate need to discover novel pharmacological treatments.

Our research mission is to understand how metabolic hormones signal to the brain and how this becomes defective in obesity and type-2 diabetes. To do this we use cutting-edge technologies and innovative mouse models to genetically dissect, manipulate and image the brain to discover novel drug targets.

Project: When does the brain become broken in obesity?

A major goal of our research program is to understand how obesity affects the brain and how this contributes to the development of obesity and type 2 diabetes. When we eat, insulin is secreted from the pancreas where it travels, via the blood, to signals to neurons in the brain's hypothalamus. Insulin signalling to the hypothalamus informs our brain to stop eating and coordinates tissues such as the liver and fat to metabolise glucose. As such, the actions of insulin signalling within the brain functions to maintain energy and blood glucose levels within a safe range.

We have previously shown that in obesity, neurons of the hypothalamus become resistant to the actions of insulin, resulting in an inability for the brain to appropriately control feeding behaviour and blood glucose levels. Despite this exactly when neuronal insulin resistance occurs during the development of obesity is unknown.

In this project, you will use state of the art technology alongside transgenic mouse models of obesity to evaluate when neurons of the hypothalamus become insulin resistant and determine the exact neuronal subtypes that become defective. The results of these studies will provide new insights into how and when insulin signalling in the brain becomes defective in obesity, information that will be harnessed to develop new therapies for metabolic diseases.

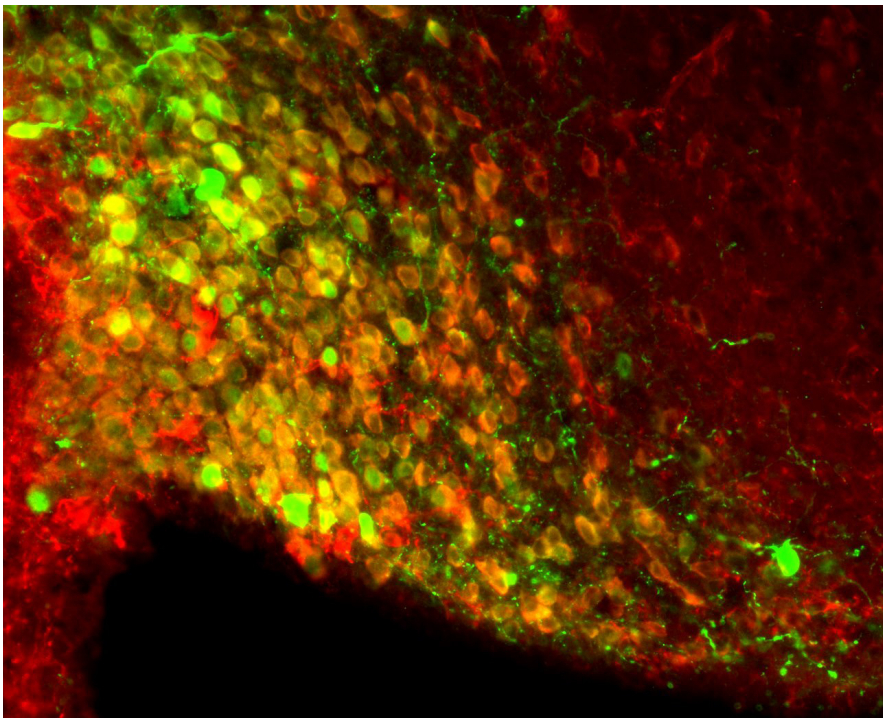
Project supervisor
Dr. Garron Dodd

- PhD
- Master of Biomedical Science
- Honours

Project: Exercising the brain to treat obesity

The increase in obesity has become one of the most important clinical-epidemiological challenges facing our society. Obesity arises when the energy we intake as food, chronically exceeds the energy we expend via exercise. Despite this simplistic overview the mechanisms underlying the development of obesity are incredibly complex.

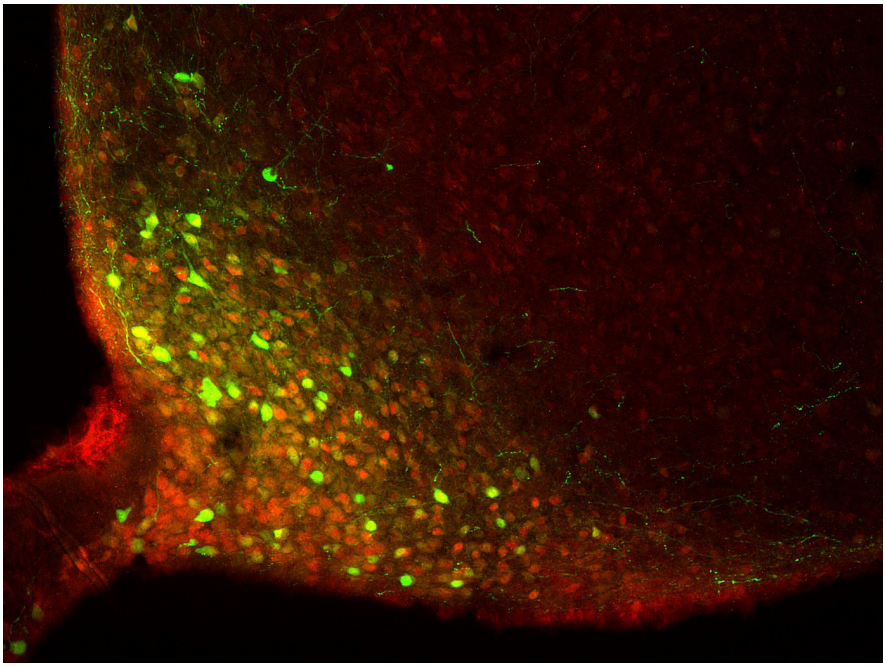
It is well established that metabolic hormones such as leptin and insulin regulate our appetite and energy expenditure by signalling to neurons in an area of the brain termed the hypothalamus. During the development of obesity hypothalamic neurons become



resistant to the actions of leptin and insulin which result in uncontrollable food intake and attenuated energy expenditure. The development of leptin and insulin resistance within neurons of the hypothalamus is a critical mechanism as to why obesity persists and places the development of drugs capable of reinstating leptin and insulin signalling at the forefront of metabolic research.

Physical activity contributes to the prevention and treatment of obesity, not only by increasing energy expenditure but also by modulating appetite and reducing food intake. Exciting new evidence shows that physical activity can re-sensitising hypothalamic neurons to the actions of leptin and insulin however the molecular mechanism underlying this are not fully understood.

In this project, you will use state of the art proteomic profiling (with space and time resolution) alongside transgenic mouse models of obesity and exercise training to evaluate the molecular mechanisms by which neurons of the hypothalamus become defective in obesity and how exercise restores them. The results of these studies will provide new insights into how exercise regulates neuronal functional, information that will be used to discover novel drug targets to treat obesity.



Project supervisors
Dr. Garron Dodd

Project Co supervisor
Dr. Benjamin Parker

- PhD
- Master of Biomedical Science
- Honours

CENTRE FOR MUSCLE RESEARCH

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.

BASIC & CLINICAL MYOLOGY

Contact: **Kate Murphy**
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Location: **Department of Physiology**

Muscle

Discovery research

Project: Investigating the role of cachexia in the response to surgical tumour resection in mice

Cancer cachexia is the progressive skeletal muscle wasting and weakness observed in 80% of cancer patients. Cachexia reduces mobility and quality of life and in the most severe cases, can lead to death. Unfortunately, there are currently no effective treatments for cachexia, with one of the reasons being a lack of understanding of the cellular mechanisms responsible for this profound wasting and weakness.

Chemotherapy and surgical interventions exist only to address primary tumour burden and the efficacy of both are dramatically limited by cachexia itself. This project will use cell- and animal-based experiments to comprehensively identify how skeletal muscle responds to chemotherapy and surgical tumour resection and will lead to developing more targeted therapies to address cancer associated muscle wasting.

Project supervisor
Dr Kate Murphy

Project co-supervisor
Prof Gordon Lynch
A/prof Paul Gregorevic

Project availability (indicate what course the project is available to). If all three, leave all three options. Indicate the places available for Masters and Honours

- Master of Biomedical Science
- Honours

Contact: **Gordon Lynch**
Email: **gsl@unimelb.edu.au**
Location: **Department of Physiology**

Metabolism

Muscle

Discovery research

Project: Therapeutic potential of skeletal muscle plasticity and slow muscle programming for muscular dystrophy

Duchenne muscular dystrophy (DMD) is a devastating, life-limiting, muscle disease that causes progressive, severe muscle wasting in boys and young men. There is currently no cure. A potential therapy may come from altering muscle phenotype based on slower, more oxidative muscle fibres being better protected from the dystrophic pathology than faster, more glycolytic muscle fibres.

Muscle plasticity can be achieved through exercise and/or through well described pharmacological approaches like activation of AMP-activated protein kinase (AMPK). Physical activity has many beneficial effects on muscle health but unfortunately many patients simply cannot exercise, especially those with DMD. Modulating muscle activity patterns through low-frequency electrical stimulation (LFS) protocols could mimic the benefits of exercise and promote a slow muscle phenotype. No studies evaluating the therapeutic merit of LFS have been conducted on the accepted mouse models of DMD nor have they determined whether muscle wasting can be attenuated or reversed.

Similarly, no studies have examined the therapeutic merit of LFS in conjunction with AMPK activators. These studies are essential for enhancing the clinical translation to improve patient quality of life.

Project supervisor
Prof. Gordon Lynch

Project co-supervisor
Dr. Justin Hardee
Dr. Rene Koopman

- PhD
- Master of Biomedical Science
- Honours

Contact: **James Ryall**
Email: **ryalljg@unimelb.edu.au**
Location: **Department of Physiology**
Weblink: **https://jgryall.wordpress.com/**

Metabolism

Muscle

Discovery research

Project: Metabolic reprogramming in skeletal muscle stem cells

One of the most important and unanswered questions in stem cell biology is how decisions regarding cell fate are regulated, specifically whether a cell undergoes self-renewal or commitment to a specific lineage. While the better part of the 20th century focused on the importance of cellular metabolism for the generation of energy, recent work has uncovered an essential role for metabolism in the generation of the building blocks (nucleotides, phospholipids, and amino acids) required by rapidly dividing cells. Additionally, the metabolite balance of both stem and differentiated cells has been found to directly influence the epigenome through post-translational modifications of histones, DNA and transcription factors and therefore has important implications for stem cell lineage progression. The overall goal of research into the link between metabolism and stem cell identity is to improve stem cell transplantation and regenerative medicine, nuclear reprogramming, transdifferentiation, and stable ex vivo expansion of stem cells. This project will utilise cutting-edge techniques such as RNAseq, metabolomics and imaging mass-spectrometry, and will have broad application in the fields of regenerative medicine, synthetic biology and cellular agriculture (the growth of so-called “clean-meat”).

Project supervisor
Dr. James Ryall

Project co-supervisor
Prof. Gordon Lynch

- PhD
- Master of Biomedical Science
- Honours

Contact: Justin Hardee

Contact: **Justin Hardee**
Email: **justin.hardee@unimelb.edu.au**
Location: **Department of Physiology**

Metabolism

Muscle

Project: 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.

Project supervisor
Dr. Justin Hardee

Project co-supervisor
Prof. Gordon Lynch
Dr. Rene Koopman

- PhD
- Master of Biomedical Science
- Honours

Contact: **Kristy Swiderski**
Email: **kristys@unimelb.edu.au**
Location: **Department of Physiology**

Muscle

Discovery research

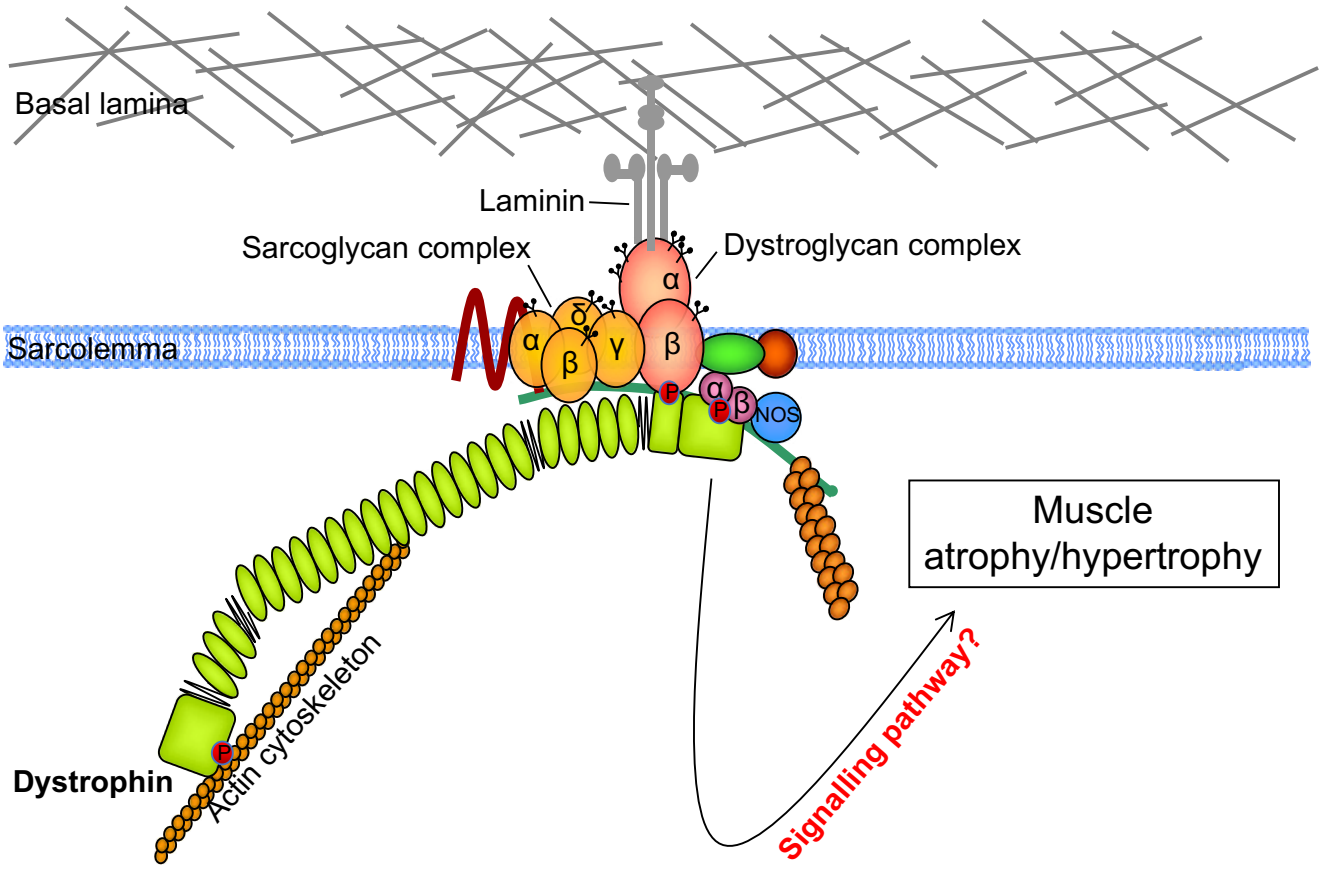
Project: Investigating the Dystrophin-Glycoprotein Complex to protect muscles from wasting conditions

The dystrophin-glycoprotein complex (DGC) is a multi-protein structure required to maintain integrity of the muscle fibre membrane and to transmit force, by linking the actin cytoskeleton with the extracellular matrix. Importantly, we and others have shown the DGC also plays a critical role in the signalling mechanisms that maintain muscle homeostasis and membrane localisation of dystrophin is perturbed in muscles wasting as a consequence of cancer cachexia, sepsis, unloading, denervation and advanced ageing, which are all associated with low level, chronic inflammation. Identifying therapeutic approaches to restore the DGC at the muscle fibre membrane is essential for improving clinical outcomes for patients whose muscles are wasting and seemingly unresponsive to other treatments. This project will test the hypothesis that loss of DGC integrity at the fibre membrane is implicated in multiple wasting conditions and that post-translational modification modulates these DGC interactions to preserve and protect muscles in different muscle wasting states.

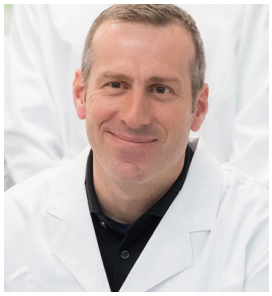
Project supervisor
Dr. Kristy Swiderski

Project co-supervisor
Prof. Gordon Lynch
Assoc. Prof. Paul Gregorevic

- PhD
- Master of Biomedical Science
- Honours



MUSCLE RESEARCH AND THERAPEUTICS



Contact: **Paul Gregorevic**
Email: **pgre@unimelb.edu.au**
Location: **Department of Physiology**

Metabolism

Muscle

Translational and Clinical Research

Discovery research

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

The Transforming Growth Factor β (TGFβ) signalling network is one of the most important regulators of processes associated with skeletal muscle development, adaptation, and repair. However, many questions remain as to how this network is regulated in skeletal muscle in health and disease, how it controls processes that determine skeletal muscle characteristics, and how best to control network elements to prevent/treat muscle conditions. Combining gene delivery-based methods with cell culture and animal models and analyses of gene expression and protein regulation, this research theme seeks to examine novel processes that control the TGFβ network in skeletal muscle, and determine how unique components of the TGFβ network control skeletal muscle structure and function. These discoveries will help to develop novel strategies for preventing/treating the loss of skeletal muscle mass and strength associated with disease and advancing age.

Project supervisor
Assoc. Prof. Paul Gregorevic

- PhD
- Master of Biomedical Science
- Honours

Project: Unravelling the mysteries of E3 ubiquitin ligase biology as a regulator of skeletal muscle in health and disease.

Regulation of muscle size and function impacts on all aspects of human health and well-being. From performance on the sports-field, to regulation of whole-body metabolism, and independence in aging. A large family of genes known as E3 ubiquitin ligases are paramount in regulation of muscle homeostasis. Changes in the activity of specific members can provoke muscle frailty and wasting, whilst others promote growth and function. Skeletal muscle expresses over 250 E3 ubiquitin ligases, yet only a handful have been characterised. This research program is investigating which E3 ligases have important functions in muscle health and disease. The projects focus on charting novel E3 ubiquitin ligases, understanding how they regulate muscle size and function, and developing therapeutically relevant methods to control their activity.

Project supervisor
Assoc. Prof. Paul Gregorevic

- PhD
- Master of Biomedical Science

Project: Developing innovative animal and human cell models to study and treat muscular dystrophies.

Many neuromuscular disorders remain poorly studied and without adequate therapies due to a lack of suitable models in which to study mechanisms and test possible interventions. This research program combines novel gene- and cell-based approaches to generate new in vitro and in vivo models of neuromuscular disorders. Characterisation and manipulation of these new model systems will enable us to a) study the underlying mechanisms of action associated with muscle disease and b) devise novel much-needed therapeutic strategies for these conditions.

Project supervisor
Assoc. Prof. Paul Gregorevic

Project co-supervisor
Dr. Kevin Watt

- PhD
- Master of Biomedical Science
- Honours

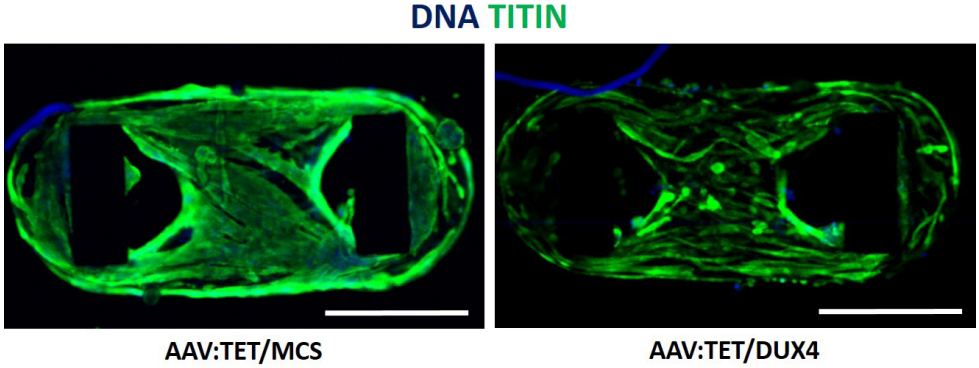
Project: Learning from skeletal muscle to treat cancer.

Patients with cancer frequently succumb to complications arising from cachexia - a condition characterised by debilitating loss of functional muscle mass, and adipose tissue. Projects within this theme are examining the mechanisms involved in the development of cachexia, in the hopes of helping to develop new therapeutic strategies. Patients with cancer also frequently succumb to complications arising from metastasis - the spread of tumour cells to other sites distant from the tissue of origin. However, the colonisation of metastatic cancers within muscle is remarkably infrequent, and the mechanisms underlying these discrepancies between muscle and other tissues remain unclear. Projects within this theme will examine why skeletal muscles are resistant to metastatic cancers, to identify new strategies for preventing and treating the development and progression of metastatic cancers.

Project supervisor
Assoc. Prof. Paul Gregorevic

Project co-supervisor
Dr. Rachel Thomson

- PhD
- Master of Biomedical Science
- Honours



Contact: **Kevin Watt**
Email: **kevin.watt@unimelb.edu.au**
Location: **Department of Physiology**

Metabolism

Muscle

Discovery research

Project: Investigating the Hippo signalling pathway as an emerging regulator of skeletal muscle

The Hippo signalling pathway regulates skeletal muscle stem cell proliferation and differentiation. Recently we found that this pathway also operates as a vital post-natal regulator of the size of skeletal muscle fibres, and is disrupted in some settings where control of muscle mass is impaired. How the Hippo pathway regulates muscle size and function, and what regulates the Hippo pathway in adult muscle remains unknown.

This research program will use innovative gene therapy-based techniques to manipulate the Hippo pathway in vitro and in vivo to a) define the critical target genes controlled by Hippo signalling in skeletal muscle, b) identify the essential elements that regulate Hippo pathway activity to control aspects of skeletal muscle biology, and c) test interventions that target the Hippo pathway, as novel muscle therapeutics.

Project supervisor
Dr. Kevin Watt

Project co-supervisor
Assoc. Prof. Paul Gregorevic

- PhD
- Master of Biomedical Science
- Honours

CLINICAL NUTRITION AND MUSCLE METABOLISM

Contact: **Rene Koopman**
Email: **rkoopman@unimelb.edu.au**
Location: **Department of Physiology**

Metabolism

Muscle

Project: 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.

Project supervisor
Dr. Rene Koopman

Project co-supervisor
Prof. Gordon Lynch

- PhD
- Master of Biomedical Science
- Honours

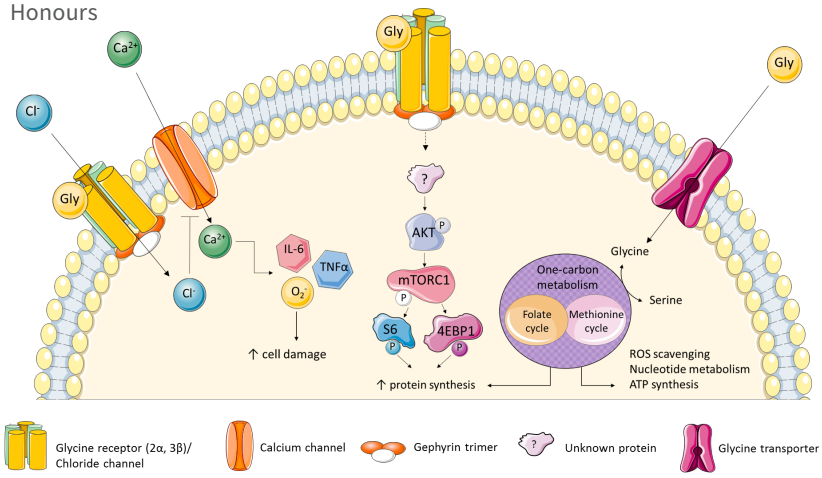
Project: Establish the effect of glycine/serine metabolism on skeletal muscle cell growth

Skeletal muscle cell proliferation and growth require the production of building blocks for new cellular components (proteins, lipids and nucleic acids) as well the maintenance of cellular redox status. Observations in other cells suggest that the metabolism of the amino acid L-serine and its intermediate glycine can provide carbon units that satisfy many of these requirements. However, the cellular demand for L-serine is much greater than its uptake suggesting that the de novo production of L-serine is of critical importance to sustain cellular growth. Surprisingly, to date no detailed investigation of the role of L-serine biosynthesis in skeletal muscle has been performed and whether L-serine can support the production of biomass in growing muscle cells remains to be established.

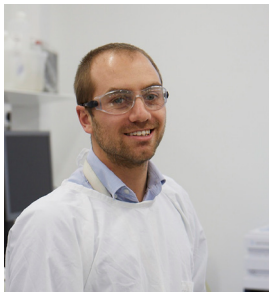
Project supervisor
Dr. Rene Koopman

Project co-supervisor
Dr. Marissa Caldow
Prof. Gordon Lynch

- PhD
- Master of Biomedical Science
- Honours



METABOLIC PROTEOMICS AND SIGNAL TRANSDUCTION



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Location: **Department of Physiology**
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- **Metabolism**
- **Metabolic Disease**
- **Cell Signalling**
- **Proteomics**
- **Systems Biology**

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 metabolic diseases. We primarily focus on metabolic tissues such as brain, liver, adipose, and muscle. Our research integrates physiology with systems biology techniques such as proteomics to understand how metabolic tissues develop, how they are regenerated, how they are affected by physical activity, how defects and genetic variants contribute to insulin resistance, and the identification and development of novel therapeutics.

Project: Modulating skeletal muscle signal transduction to treat pre-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.

Project supervisor
Dr Benjamin L. Parker

- PhD
- Master of Biomedical Science
- Honours

Project: The role of Galectin-1 on myogenesis and skeletal muscle development

The maintenance of muscle mass is one of the most important processes for a healthy and long life. Myogenesis is critical in the development and regeneration of muscle and involves the differentiation and fusion of myoblasts to form multi-nucleated myofibers. This process involves complex changes in cell surface and cell adhesion receptors. Many of these receptors are modified with glycans that regulate cell:cell interactions. We have recently performed a comprehensive glycoproteomic analysis of myogenesis revealing several changes in the glycosylation of cell adhesion receptors and glycan-binding proteins including galectin-1 (LGALS1). CRISPR-based knock out of LGALS1 inhibited myogenesis in vitro. To further characterise the role of LGALS1 on muscle physiology, we have generated an recombinant adeno-associated virus (rAAV) to overexpress LGALS1 in vivo. This project will characterise the role of LGALS1 overexpression in adult mouse skeletal muscle physiology. The project will involve a variety of techniques including physiology, proteomics and biochemistry.

Project supervisors
Dr Benjamin L. Parker

- PhD
- Master of Biomedical Science
- Honours

NEUROPHYSIOLOGY

BORNSTEIN GROUP

Contact: **Joel Bornstein**
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Location: **Department of Physiology**

Neurophysiology

Discovery research

Our major research interests are the neural mechanisms and circuits that control intestinal motor functions underlying the digestive process, including both muscle movement and the secretion of water and salt by the mucosa, and how these are disturbed by bacterial toxins. This work involves experimental methods ranging from electrophysiological analysis of synaptic transmission in reflex pathways, to immunohistochemical analysis of enteric neural circuits, to measurements of intestinal movements and secretions both in vitro and in vivo and computer simulation of the networks of neurons that mediate these functions. Much of this work, especially that involving interactions between intestinal movements and secretion, is carried out in close collaboration with Dr Tor Savidge of Baylor College of Medicine in Texas. Other international collaborations include a consortium led by Professor Marthe Howard (University of Toledo, Ohio) and funded by NIH whose goal is a predictive anatomical map of the enteric nervous system.

Project: Mechanisms underlying synaptic transmission in the enteric nervous system

Transmission between enteric neurons is an essential therapeutic target for many gastrointestinal diseases, but the molecular mechanisms are not clearly established. In this project, key molecules and the dynamic properties of enteric synapses will be identified using immunohistochemistry and calcium imaging to determine how these molecules participate in transmission.

Project supervisor
Prof Joel Bornstein

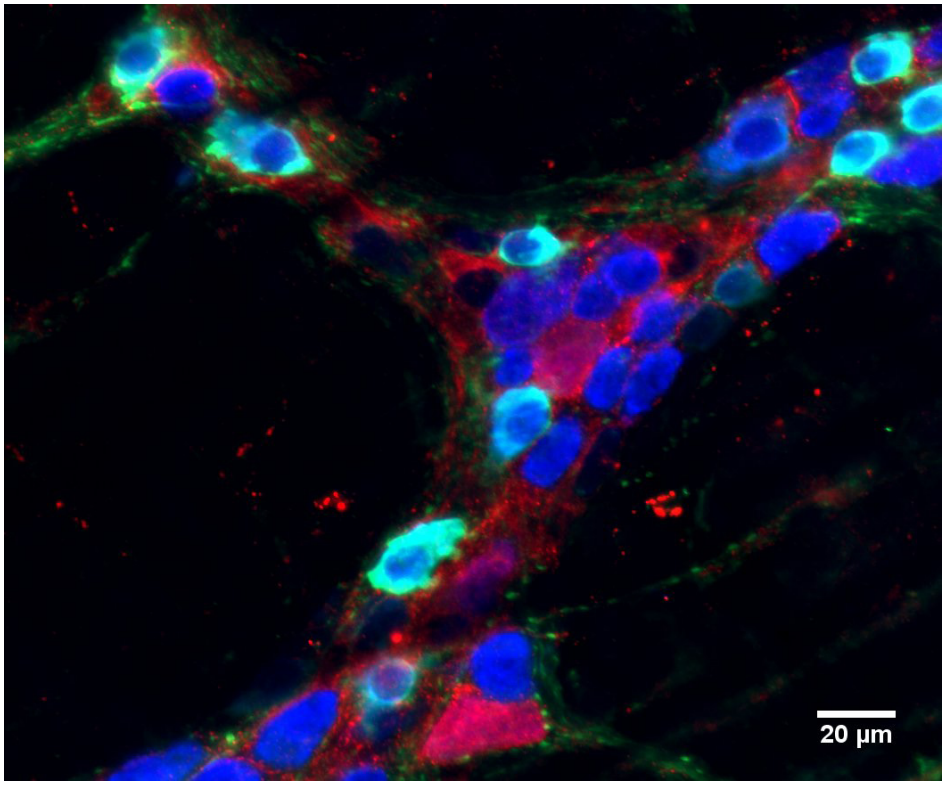
- Master of Biomedical Science
- Honours

Project: Reproductive cycle dependent plasticity within the enteric nervous system

We have recently found that enteric neural circuits that control gut function change their properties according to the stages of the reproductive cycle in mice. These changes include changes in the neurochemical phenotype of myenteric neurons and appear to depend on circulating estrogens. In this project, you will investigate whether rapid changes in the phenotype of enteric neurons are associated with changes in function using immunohistochemistry, calcium imaging and functional analysis.

Project supervisor
Prof Joel Bornstein

- Master of Biomedical Science
- Honours



Project: Role of bacterially generated GABA in antibiotic associated diarrhoea

Antibiotic treatments frequently produce diarrhoea as a major side effect and this can be lifethreatening. We have data indicating that antibiotic treatments that cause antibiotic associated diarrhoea change the gut microbiome so that it produces large amounts of the neurotransmitter GABA. In this project, you will investigate the chronic effects of bacterially derived GABA to identify how this transmitter affects diarrhoeal disease.

Project supervisor
Prof Joel Bornstein

- Master of Biomedical Science
- Honours

Project: Impact of early life antibiotics on the nervous system of the gut and host physiology

Exposure to antibiotic during critical developmental windows have been linked to increased susceptibility to several diseases, including gastrointestinal and metabolic disorders later in life. We have found in mice that exposure to antibiotics early in life during the neonatal period, and in utero (via the female dam) disrupts the developing microbiota, nervous system of the gut and host metabolism. This project will provide critical insights into how antibiotics impacts host physiology, which will aid in elucidating potential circumventive measures for the unwanted side-effects of antibiotic therapy.

Project supervisor
Dr Jaime Foong

Project co-supervisor
Prof Joel Bornstein

- PhD
- Master of Biomedical Science

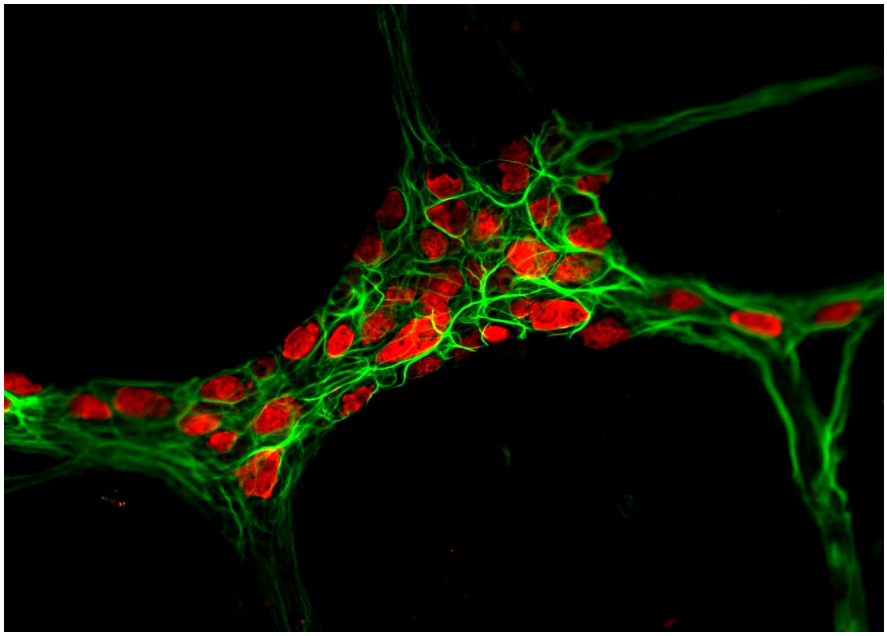
Project: Development of a functional Enteric Nervous System

Proper development of the Enteric Nervous System (ENS) is essential for regulating vital gastrointestinal functions. However, the development of a functioning ENS is still unclear. This project will use advanced microscopy and a robust method of measuring dynamic activity and neurotransmission of the developing enteric circuitry by employing mice in which enteric neurons express a genetically encoded calcium indicator. Findings from this study will elucidate factors that affect maturation of synaptic transmission within the ENS.

Project supervisor
Dr Jaime Foong

Project co-supervisor
Prof Joel Bornstein

- PhD
- Master of Biomedical Science



MCDOUGALL GROUP

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Neurophysiology

Discovery research

The Viserosensory group lab at the Florey Institute studies the basic neurophysiology underpinning the integration of sensory information within the brain. Our focus of study is at the level of the brain that first receives signals from visceral organs including those of the cardiorespiratory and gastrointestinal systems. This knowledge is pertinent to several disease states including; hypertension and obesity, and mental health related including epilepsy. The primary techniques utilised within the laboratory revolve around in vitro slice electrophysiology. We possess a large skill-set and toolkit to answer a variety of experimental questions including optogentics through to behavioural paradigms.

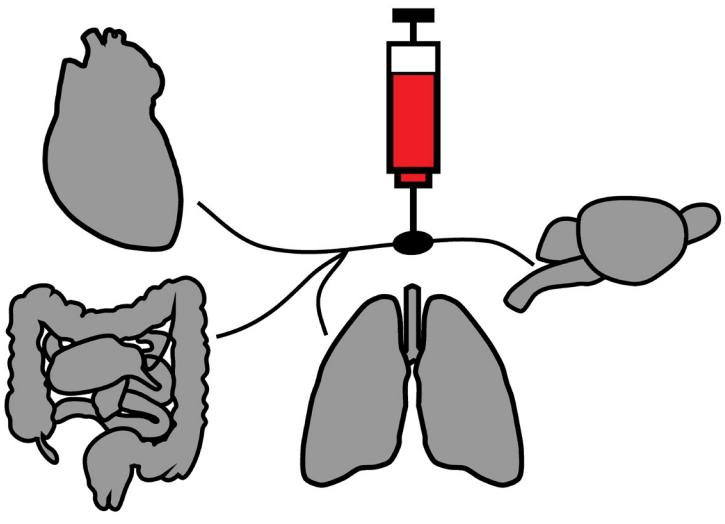
Project: Optogenetic activation of vagal afferents to decode viscerosensory signal processing within the brain.

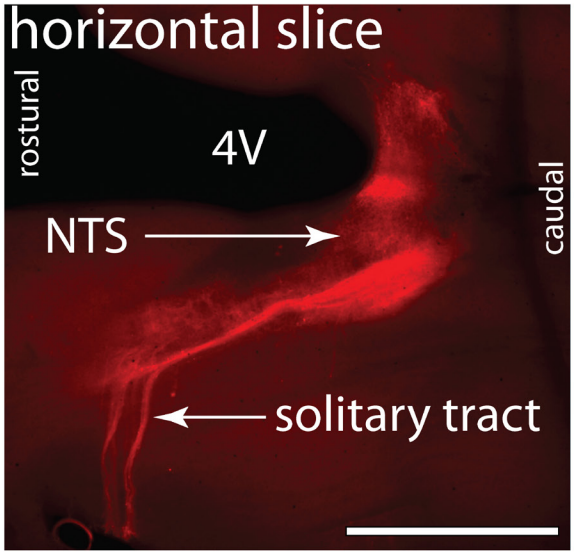
How different sensory signals from internal organs are organised and processed upon first entering the brain is ill defined. Viscerosensory signals arise from several functional modalities; baroreceptors, chemoreceptors, lung stretch afferents, gastrointestinal etc. These varied signals all terminate in the solitary nucleus with overlapping terminal fields. We will use optogenetic tools, that allow for the selectively activation of neurons of interest, to unravel how signals from these different sensory modalities 'talk' to the brain. Optogentic and electrical activation will be compared to further understand to functional determine how the local circuits are organised. This work will be highly relevant to current and future strategies to manipulation behavior and/or autonomic function.

Project supervisor
Dr Stuart McDougall

Project co-supervisor
Professor Andrew Allen

- PhD
- Master of Biomedical Science
- Honours





LANKADEVA GROUP

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Weblink: **http://go.unimelb.edu.au/89hr.**

Cardiovascular

Translational and Clinical Research

Discovery research

The Translational Cardiovascular and Renal Research group's research focuses on understanding the mechanisms driving cardiovascular and renal diseases arising from severe infections (sepsis) and heart (cardiac) surgery. Our aim to understand the changes that occur during disease and armed with this knowledge to develop effective diagnostics and therapeutics that can be rapidly translated to improve health outcomes for patients.

Project: Reversing renal medullary hypoxia and acute kidney injury from sepsis

Sepsis is the main cause of death in intensive care units worldwide. Acute kidney injury (AKI) develops in up to 50% of septic patients leading to increased mortality (up to 40%). There are no therapies for septic AKI, apart from dialysis, which is invasive and expensive and is not a cure for AKI. A major barrier to the discovery of new therapies for septic AKI is the lack of animal models that replicate the complex pathophysiology of human septic AKI.

We have established a clinically relevant sheep model of sepsis that has a similar phenotype to human septic AKI. In recent pioneering studies, we found that in early sepsis there is reduced blood flow (ischemia) and reduced oxygen levels (hypoxia) in the renal medulla, which may be driving AKI. The aim of this project is to investigate novel and clinically feasible therapies and drug delivery techniques to reverse hypoxia and acute kidney injury during sepsis.

Project supervisor
Dr Yugeesh Lankadeva

Project co-supervisor
Professor Clive May

- PhD
- Master of Biomedical Science
- Honours

YAO GROUP

Contact: **Dr Song Yao**
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Location: **Howard Florey Laboratories Parkville**

	Cardiovascular
	Neurophysiology
	Discovery research

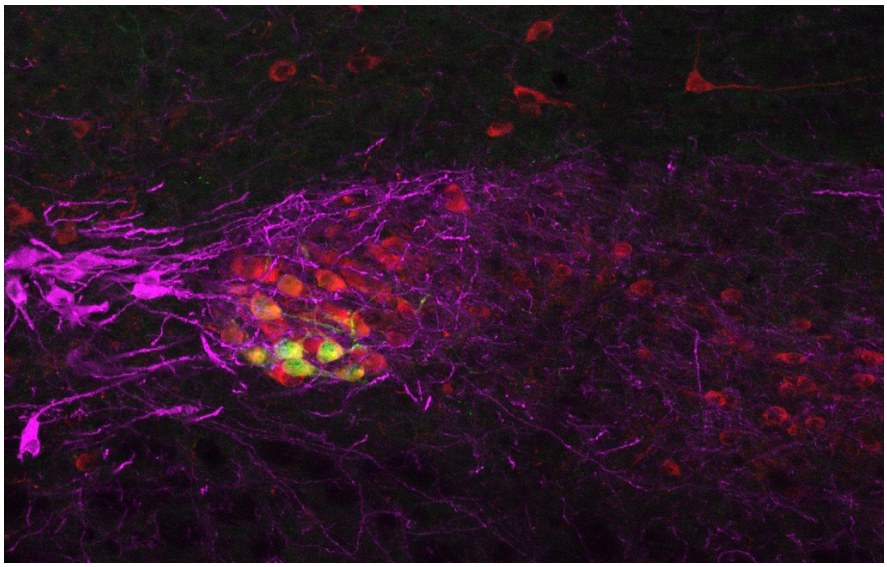
The Neurovascular Biology Laboratory is located in the Howard Florey Institute. The laboratory's main interest is to understand how the brain controls the cardiovascular system and how these mechanisms become dysfunctional in diseases such as heart failure and hypertension. Recently, we have explored mechanisms whereby inflammation can cause increased activity in certain brain areas which ultimately causes an increase in sympathetic nerve activity and blood pressure. Our laboratory is particularly interested in how inflammation and inflammatory mediators might lead to increases in blood pressure in hypertension. We use a range of techniques in the laboratory. These include, neuropharmacology, electrophysiology, radiotelemetry, immunohistochemistry, confocal microscopy.

Project: Central cardiovascular control: Does MCP-1 act on area postrema neurons to increases in blood pressure?

The area postrema is a circumventricular organ located in the brain stem. Because it lacks a blood-brain barrier the area postrema is exposed to a wide range of factors found in the circulation. There is much evidence to suggest that inflammatory cytokines are increased in a number of cardiovascular diseases such as hypertension and heart failure. However, the cardiovascular effect of these cytokines when exogenously applied to the area postrema is not currently known. This project will investigate the effects of the monocyte chemoattractant protein-1 (MCP-1), within the area postrema. Plasma levels of MCP-1 has been previously shown to be increased in hypertension when administered centrally (via intracerebroventricular cannula) but whether it acts within the area postrema is not known. The successful completion of the project will increase our understanding of the role of cytokines in driving changes in blood pressure at the level of the area postrema and how this signaling might be altered in disease states such as hypertension.

Project supervisor
Dr Song Yao

- Honours



BAKER HEART AND DIABETES INSTITUTE MACEFIELD GROUP

Contact: **Prof Vaughan Macefield**
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Location: **Baker Heart and Diabetes Institute**

Cardiovascular

Neurophysiology

Discovery research

Investigating vestibulosympathetic reflexes in humans

While several methods to activate the human vestibular apparatus have been used, galvanic vestibular stimulation (GVS) is a means of selectively modulating vestibular afferent activity via electrodes over the mastoid processes, causing robust vestibular illusions of side-to-side movement. Sinusoidal GVS (sGVS) causes partial entrainment of sympathetic outflow to muscle and skin. Modulation of muscle sympathetic nerve activity (MSNA) and skin sympathetic nerve activity (SSNA) from vestibular inputs competes with baroreceptor inputs, with stronger temporal coupling to the vestibular stimulus being observed at frequencies remote from the cardiac frequency. Moreover, the vestibular modulation of SSNA, but not MSNA, is augmented in individuals experiencing nausea. In this project we will extend our work on investigating vestibulosympathetic reflexes in humans, exploring how the frequency and pattern of stimulation modulates sympathetic outflow to muscle and skin. MSNA and SSNA can be recorded directly via metal microelectrodes inserted percutaneously into a peripheral nerve in awake humans (microneurography), and the student will acquire the skills for recording and analysing MSNA and SSNA, which can then be applied to a more detailed PhD project.

Project supervisor
Prof Vaughan Macefiled

- Honours
- Masters

The contribution of the dorsolateral prefrontal cortex to the control of blood pressure

The sympathetic branch of the autonomic nervous system plays a critical role in the control of blood pressure, both through its effects on the heart and, importantly, through the beat-to-beat control of blood flow through systemic blood vessels. Arterioles in the skeletal muscles are particularly important in this regard (the muscle vascular bed has a very high volume), and an increase in muscle sympathetic nerve activity (MSNA) is known to occur during psychological stress. MSNA can be recorded directly via metal microelectrodes inserted percutaneously into a peripheral nerve in awake humans (microneurography), and by recording MSNA at the same time as performing functional Magnetic Resonance Imaging (fMRI) of the brain we have shown that the dorsolateral prefrontal cortex (dlPFC) is involved in the regulation of MSNA. Here we shall use transcutaneous Direct Current Stimulation (tcDCS) and transcutaneous Alternating Current Stimulation (tcACS), delivered by surface electrodes applied to forehead, to change the activity of the dlPFC, and thereby investigate how such changes in activity modulate MSNA and blood pressure. The student will acquire the skills for recording and analysing MSNA, skills which can then be applied to a more detailed PhD project.

Project supervisor
Prof Vaughan Macefiled

- Honours
- Masters

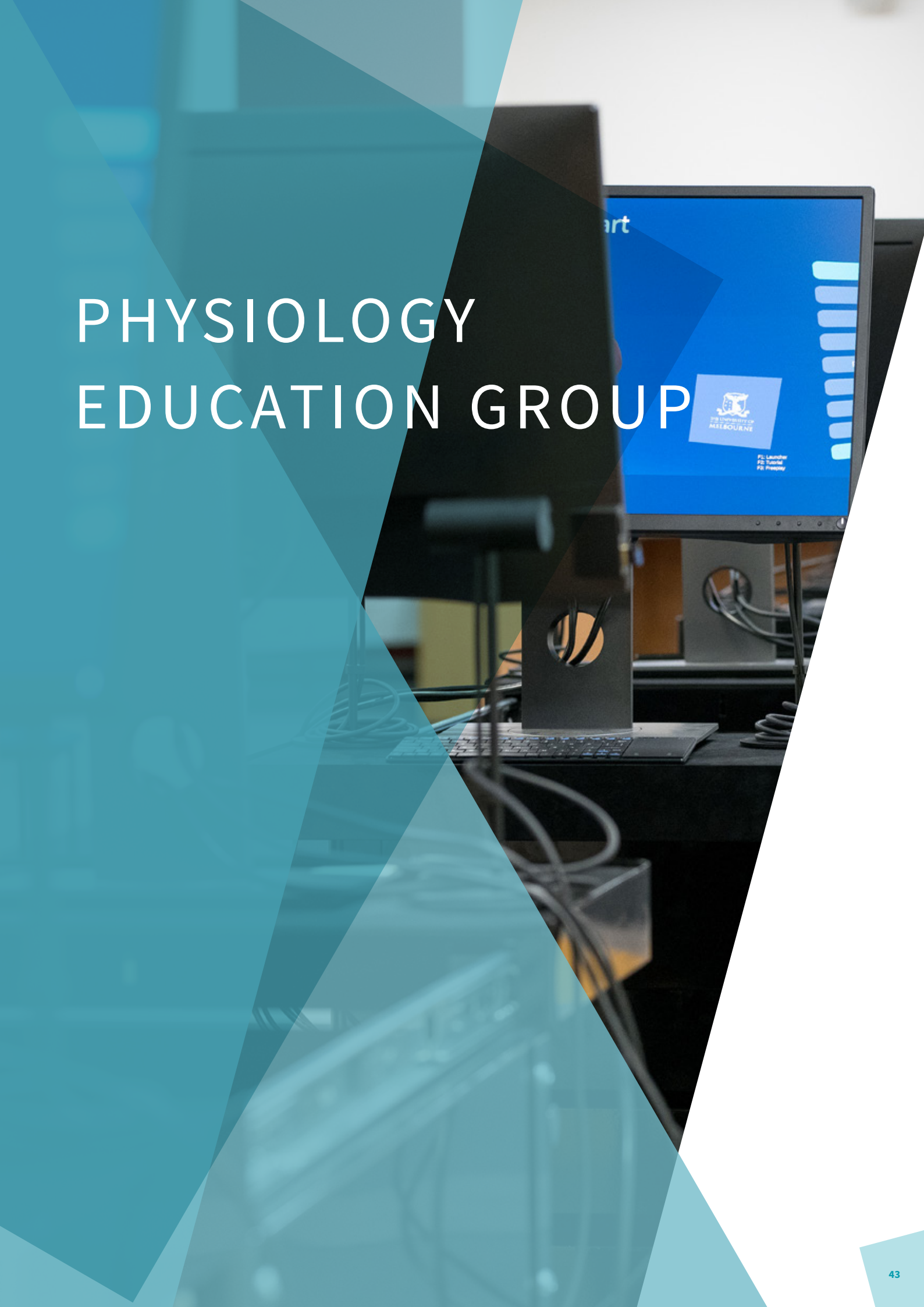
The effects of pain on the sympathetic and immune systems

Chronic pain - now defined as ongoing pain lasting more than 3 months - is frequently established from activation of nociceptors located in deep tissues such as muscle, but can be sustained in the absence of persistent peripheral noxious input by plastic changes within the brain. The incapacitating effects of long-lasting pain are not just psychological - reflexes driven by nociceptors during the establishment of chronic pain may cause serious physiological consequences that affect many systems, including the cardiovascular system. Using a model of experimental muscle pain - intramuscular infusion of hypertonic saline - we have shown that long-lasting muscle pain causes a sustained increase in muscle vasoconstrictor drive, blood pressure and heart rate in some subjects but sustained decreases in others. This may explain why some people develop high blood pressure following surgery, but why this is occurs we do not know. The purpose of this project is to understand the processes by which noxious stimulation causes an increase in muscle vasoconstrictor drive and blood pressure in some people, but not in others. By recording muscle sympathetic nerve acitivty (MSNA) at the same time as performing functional magnetic resonance imaging (fMRI) of the brain we have recently identified differences in specific regions of the brain in a group showing an increase in MSNA and blood pressure and a group showing a decrease. The current project will determine whether the differential sympathetic responses to muscle pain also lead to differences in inflammatory markers. The project will combine microelectrode recordings of MSNA in awake human subjects with intravenous blood sampling at rest and during one hour of experimental muscle pain. This approach will allow us to determine whether sympathetic activation leads to increases in release of inflammatory products, which in turn may contribute to long-term changes in the brain. The student will acquire the skills for recording and analysing MSNA, skills which can then be applied to a more detailed PhD project.

Project supervisor
Prof Vaughan Macefiled

- Honours
- Masters

PHYSIOLOGY EDUCATION GROUP



Contact: **Dr Charles Sevigny**
Email: **sevignyc@unimelb.edu.au**
Location: **Department of Physiology**

Pedagogical Research

The Physiology Teaching Team is committed to excellence and continuing development of educational strategies and resources. Recently, we have been developing immersive 3D learning tools, including Virtual Reality (VR) applications to enhance student engagement and understanding.

The School of Biomedical Sciences (SBS) Digital Learning Hub (DLH) develops and deploys modern and engaging learning experiences to undergraduate and graduate students within the SBS. The DLH mission is to make learning within the SBS engaging, effective, efficient, and collaborative. The Department of Physiology, utilising the exciting resources within the DLH, is currently developing a Virtual Reality Human Heart which will be integrated into the Biology, Physiology, and Anatomy curricula across all year levels.

Project: The use of an immersive virtual reality human heart application to improve student learning outcomes

The implementation of virtual reality solutions in biomedical science fields, as well as other STEM fields opens a great number of opportunities for both undergraduate and postgraduate teaching. Recent examinations of virtual reality within the classroom at all levels of education has shown a considerable improvement in student learning outcomes when compared to current teaching methods.

This study aims to assess the efficacy of a highly contextual environment for learning in biomedical science. The study will examine whether an immersive environment (virtual reality) will enable better learning outcomes of abstract principles, i.e. the visualisation of the heart and the haemodynamic principles, that are often difficult to replicate in real-life environment. The outcomes from this study will provide evidence of whether immersive learning tools are beneficial for learning abstract concepts in STEM.

Project supervisor
Dr Charles Sevigny

Project co-supervisor
Dr Angelina Fong
Dr Joseph Rathner

- Honours

Contact: **Yossi Rathner**
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Pedagogical Research

The teaching team in the Department of physiology focuses on building curriculum and learning resources that enhance student learning, and attract students to study within the department. We are active in scholarship of teaching and learning (SoTL), which is research into teaching practice, with a specialization of teaching and learning in physiology and biomedicine.

Project: Enhancing the student learning experience: Evaluating the impact of learning design on the student self-efficacy and assessment performance.

The scholarship of teaching and learning encompasses a broad array of educational outcomes. These include student perception of their learning experience and the impact of teaching design on student learning. In the higher education sector, there is increasing pressure to make learning relevant to the workplace. Graduate attributes for degrees will include soft skills like 'lifelong learning' and 'communication skills'. There is also an increasing tension between teaching content (what instructors want students to learn) and learning process (how students learn). Typically STEM educators value content over process.

Understanding the motivation of students selecting physiology subjects to study could potentially provide insight into how best to design learning activities, and guide instructors in determining what students need to know. Research projects in SoTL can be driven by student's individual interest but may include (but not limited to):

- Evaluation of the impact of feedback on student assessment results
- Development of tools to enhance student meta-learning (self-efficacy)
- Evaluation of learning design approaches on student outcomes
- Development and deployment of online or e-learning resources, and evaluation of their efficacy.
- Analysis of the internal and external factors that predict assessment performance.

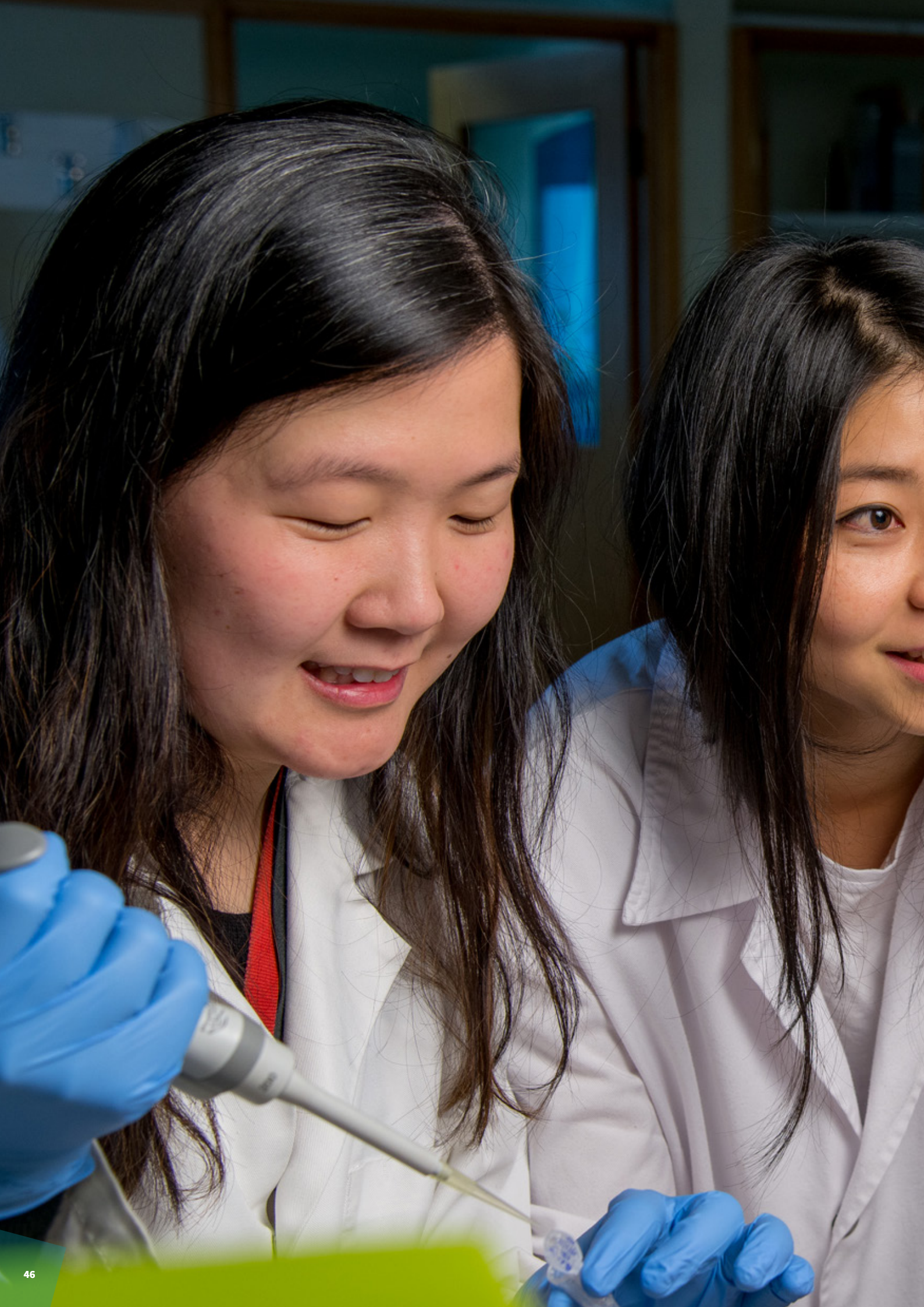
Students who undertake projects in SoTL will develop a deeper knowledge in ways of learning. You will also develop research skills, particularly related to writing and designing survey instruments, qualitative analysis of survey results, as well as quantitative statistical analysis. This project will be suitable for students who are interested in understanding the methodological and ethical issues associated with research on people. You will also deepen your understanding of physiology by simply asking the questions "how do we teach physiology?" and "how is physiology relevant to our real world experience?".

Project supervisor
Dr Joseph (Yossi) Rathner

Project co-supervisors

Professor David Williams
Dr Angelina Fong
Dr Charles Sevigny

- Honours
- Master of Biomedical Science



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Website: biomedicalsciences.unimelb.edu.au/departments/physiology

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