THE UNIVERSITY OF MELBOURNE

Department of Biochemistry and Pharmacology

Research Projects 2026

Welcome

The Department of Biochemistry and Pharmacology is a teaching and research Department of the School of Biomedical Sciences, Faculty of Medicine, Dentistry and Health Sciences based on the main campus in the Melbourne Biomedical Precinct in Parkville.

The Department of Biochemistry and Pharmacology has teaching responsibilities and very active research programs and expertise that underpin our key themes of molecular understanding of disease, translational research and drug discovery and development. Our department houses 30+ research groups working in the areas of biophysics, cell biology, chemical biology, pharmacology, computational biology, drug design, immunology, metabolism, proteomics and structural biology. Disease foci include infection, cancer, neurodegeneration, respiratory disease, inflammatory/ autoimmune diseases and genetic diseases. We place a strong emphasis on research and research training with over 75 graduate research students currently enrolled.

The Department is situated within the University of Melbourne's Parkville Biomedical Precinct, one of the largest biomedical research precincts in the world that is home to several research institutes that are household names. Laboratories in our department are located both in the Medical Building and in the Bio21 Molecular Science and Biotechnology Institute, a multidisciplinary research centre specialising in biochemistry, drug discovery, neuroscience, environmental science, dentistry and engineering. In addition to housing the research facilities of CSL, Australia's largest pharmaceutical and biotechnology company, the Bio21 Institute also hosts the University's major technology platforms including the Melbourne Advanced Microscopy Facility, the Magnetic Resonance Facility, the Melbourne Mass spectrometry and Proteomics Facility, Metabolomics Australia, the Macromolecular Interactions Facility, a peptide synthesis facility, as well as High Performance Computing and facilities for animal models. The department and the Bio21 Institute provide outstanding career opportunities for staff and superb training facilities and research environment for our students.

It is a great pleasure to introduce you to the research projects that are on offer by the Department of Biochemistry and Pharmacology for 2026.

Most projects offered will be in our departmental laboratories on the 8th and 9th floors of the Medical Building and in the Bio21 Molecular Science and Biotechnology Institute. The remainder will be conducted in affiliated research institutes with external supervisors and co-supervision by Department staff.

The Department of Biochemistry and Pharmacology Honours and Masters courses are directed at students with above average academic ability. Both courses aim to transition students from formal lectures and teaching, to self-directed learning and exploration of your own scientific problem. We will introduce you to skills in communication, data analysis and assessment of scientific papers. Your supervisor and their laboratory staff will guide you through the challenges, strengthen your technical skills and introduce you to the excitement of research - its rewards and its disappointments. You will have the opportunity to use the latest in equipment and work alongside other researchers to expand biomedical knowledge. The Honours and Masters "experience" will require self-motivation and discipline, and you will learn a lot about your own problem-solving ability. Research Higher Degrees such as a PhD are longer courses with a focus on self-directed research. Graduate researchers are typically students with both an above average academic ability and a desire to further develop a specific field of research. Entry into these programs requires completion of an Honours or Masters degree.

It is not a simple task to select a project, laboratory and supervisor. We suggest you talk to several potential supervisors, as well as to their current Honours, Masters or Graduate Researchers, to gain some appreciation of the research problems being addressed and the related techniques. You will find them friendly and welcoming! We hope you will join us in The Department of Biochemistry and Pharmacology for a 2026 Honours, Masters, or PhD project.

How to apply

Honours

What is Honours?

Honours is a fourth-year undergraduate course that consists of a major research project complemented by 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 or Biomedicine Honours.

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

How long is Honours?

Honours is a one-year course consisting of 75 points of research and 25 points of coursework, that commences mid-February and finishes in November. Mid-year entry is also possible, commencing late July and finishing in June the following year.

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. We suggest you provide the potential supervisor with a recent academic transcript on first contact. 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 a course application online, 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 instructions 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 before the closing date.

More information including application dates and the online application form can be found in the following links:

mdhs-study.unimelb.edu.au/degrees/honours/apply-now

biomedicalsciences.unimelb.edu.au/departments/department-of-biochemistry-and-pharmacology/study/honours-and-masters-by-coursework-biochemistry-and-pharmacology

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 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, alongside 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?

Masters is a two year (full time) course consisting of 125 points of research and 75 points of coursework. The course can be commenced in Semster 1 or Semester 2 each year.

How to apply

- 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.
- 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. We suggest you include an academic transcript and any other relevant information when making first contact with a potential supervisor. Once your project has been endorsed, you will be allocated to this project in SONIA.

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

https://biomedicalsciences.unimelb.edu.au/departments/department-of-biochemistry-and-pharmacology/study/honours-and-masters-by-coursework-biochemistry-and-pharmacology

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

How to apply

Research Higher Degrees

What is a PhD?

A PhD (Doctor of Philosophy) is a three-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 18 months 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's 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 master's 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% (University of Melbourne) or equivalent.

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

- a four-year bachelor's 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% (University of Melbourne) or higher; or
- a master's 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 75% (University of Melbourne) 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.

For future information regarding Research Higher Degrees:

study.unimelb.edu.au/find/courses/graduate/doctor-of-philosophy-medicine-dentistry-and-health-sciences/

study.unimelb.edu.au/find/courses/graduate/master-of-philosophy-mdhs-biomedical-science/

biomedicalsciences.unimelb.edu.au/departments/biochemistry/research/graduate-research-opportunities

How to apply

1. Review the list of prospective projects and supervisors in this handbook or online.
2. Identify projects of interest and contact the Project Supervisor: to explain your research interests and provide your curriculum vitae (CV) and academic transcripts.

Once confirmed a project and supervisor apply online at **https://study.unimelb.edu.au/how-to-apply/graduate-research**

Scholarships and awards

Honours

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

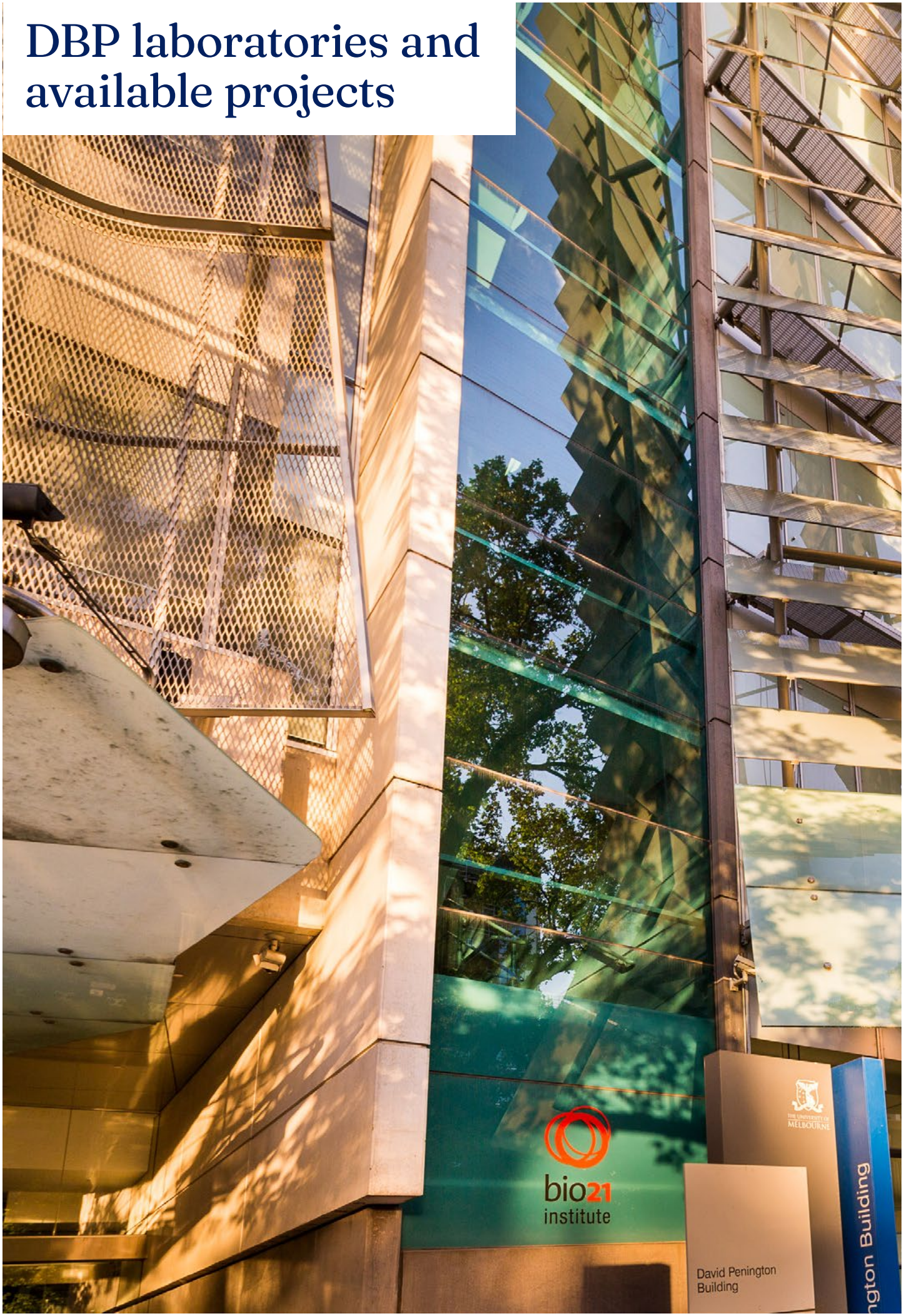
Highly ranked full-time students who have enrolled in an MDHS program through the Bachelor of Biomedicine (Degree with Honours) and the Bachelor of Science (Degree with Honours) and demonstrated a level of financial needs will automatically be considered for an Frances Elizabeth Thomson Trust Scholarship.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

Research Higher 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**

DBP laboratories and available projects



Anderson Group

Contact: Professor Gary Anderson
Location: Department of Biochemistry & Pharmacology (Bio21 Molecular Science and Biotechnology Institute)
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Website: go.unimelb.edu.au/2z5i

Key focus areas

- Cell Biology
- Discovery research
- Immunology
- Lung Health
- Metabolism
- Respiratory diseasesogy

Our research is focused on understanding the molecular basis of chronic degenerative lung diseases, in particular severe refractory asthma, Chronic Obstructive Lung Disease (COPD), Asthma-COPD Overlap, the COPD-lung cancer interface and fibrotic lung diseases. We are interested in understanding the reasons why lung disease becomes chronic and resists the normal processes that help resolve tissue damage, as well as why the damaged lung is so susceptible to viral and bacterial infections. Our research also focuses on developing and testing experimental medicines in preclinical models. We work with leading clinicians/researchers at the RMH and international consortiums to translate our basic findings into useful medicines. Our development ethos is that the end goal should be a 'cure' rather than small incremental steps towards improving current therapeutics and our laboratory has helped create lung medicines used in clinical practice and trials, we are a lead laboratory of the Australian Cure Asthma Initiative research network.

Project: Using airway organoids to model chronic lung diseases and T2 immunity

Mucus obstruction is a major determinant of disease morbidity, progression and mortality in multiple "muco-obstructive" respiratory diseases including chronic obstructive pulmonary disease, non-cystic fibrosis bronchiectasis, Primary Ciliary Dyskinesias, asthma and Cystic Fibrosis. T2 is a specialized tissue injury and immune response program now explicitly linked to asthma and COPD but of unknown significance in other lung diseases. This project utilizes the derivation and reprogramming of iPSC "stem cell" airway organoids from patients across different muco-obstructive respiratory diseases to better understand the contribution of T2 immunity in progressive pathophysiology and to test new potential treatments.

Project availability:

PhD
Honours
Master of Biomedical Science

Project supervisor:

Professor Gary P Anderson

Project co-supervisors:

Dr Aowen Zhuang and Dr Rhiannon Werder (Royal Children's Hospital)

Project: Understanding the persistent lung disease mechanisms following viral infection

Viruses are major triggers of lung disease worsening (exacerbation) and loss of lung function. They are now also suspected to be fundamental causes but how? This project examines now viruses cause persistent changes in the lung lining epithelium that we suscept are a fundamental cause. This project focuses on new insights into persistent "muco-obstructive" pathology where excessive and pathogenic mucus impairs host defense and increases the likelihood of recurrent viral infections. This project utilises in vitro and in vivo models of natural infection to better understand the reprogramming of the lung epithelium post-viral and how this causes persistent airway disease and destruction.

Project availability:

PhD
Honours
Master of Biomedical Science

Project supervisor:

Professor Gary P Anderson

Project co-supervisors:

Dr Aowen Zhuang and Dr Rhiannon Werder (Royal Children's Hospital)

Project: Discovery of novel polygenic disease signatures associated that predict lung function decline

COPD affects more than 360 million people. It is a heterogeneous and complex condition that was once mainly caused by smoking but is now caused mostly by air-pollution. By the time COPD is diagnosed it has destroyed up to 80% of the lung so we want to be able to detect it much earlier in the “pre-COPD” stage where mucus pathology is increasingly recognised as a central pathogenic feature- mucus and cough are central parameters. We have proven epidemiologically in the TAHS cohort study, that productive cough with sputum marks “at risk” individuals (now called “pre-COPD”) for a rapidly declining lung function trajectory and future COPD. This cross-disciplinary project will incorporate methods of machine learning and bioinformatics and clinical epidemiology to better understand novel risk signatures that could predict individuals most at risk for developing COPD and finding intervention pathways for advanced new treatment.

Project Availability:

PhD
Honours
Master of Biomedical Science

Project Supervisor:

Professor Gary P Anderson

Project Co -supervisors:

A/Prof Michael Menden; A/Prof Caroline Lodge; Prof Shyamali Dharmage

Bathgate Group

Contact: Professor Ross Bathgate
Location: The Florey Institute of Neuroscience and Mental Health
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Website: go.unimelb.edu.au/id5i

Key focus areas

- Biochemistry
- Cell Signaling
- Drug Design
- Pharmacology
- Structural Biology

The largest single class of drug targets is the G Protein-Coupled Receptor (GPCR) family. However, current drugs only target a small proportion of the GPCR family and peptide GPCRs, although showing great potential as targets for treating many diseases, are poorly targeted with drugs. Our laboratory targets peptide GPCRs for drug development utilizing state-of-the-art molecular pharmacology, biochemical and structural techniques, including Nuclear magnetic resonance (NMR) and cryo-EM microscopy. This enables us to map the native peptide binding sites of these receptors and determine the mechanisms of receptor activation which is essential to design drugs targeting these receptors. We then utilize the latest chemical, biochemical and pharmacological tools to design and test peptide, small molecule and antibody drug leads targeting these receptors. Finally, we work closely with pharmaceutical industry partners to facilitate the development of these lead compounds into drugs.

Project: Targeting peptide G protein-coupled receptors (GPCRs) for drug development

Targeting peptide G protein-coupled receptors (GPCRs) for novel drug development.

Project a vailability:

PhD
Honours

Project supervisors:

Professor Ross Bathgate

Project co-supervisors:

Professor Paul Gooley

The Bathgate lab focusses on understanding the interactions of peptide ligands with their G protein-coupled receptor (GPCR) targets for the development of new therapeutics. He works closely with a number of pharmaceutical companies interested in the clinical development of drugs targeting receptors for peptides of the relaxin family. Projects are available on multiple therapeutically relevant GPCR targets with training in various techniques including peptide mimetic design, cell signalling assays, molecular pharmacology, structural biology and structure-based drug design.

Project: Peptidomimetic drug design targeting G protein-coupled receptors

Peptidomimetics are drugs that mimic the actions of native hormones and growth factors. They combine the advantages of both small molecule drugs (e.g. bioavailability and stability) and the specificity and safety of larger biologic drugs. Our group have developed novel patented methodologies for the development of peptidomimetic drugs targeting G Protein-Coupled Receptors (GPCRs), the largest single class of drug targets. We utilize multidisciplinary cutting-edge technologies, including modern solid phase peptide synthesis, state-of-the-art cell signalling assays, molecular pharmacology, structural biology (see complementary project) and animal physiology to target numerous GPCR targets for the treatment of cardiovascular disease, neurological disorders, fibrosis, diabetes and gut dysfunction. Importantly, we work closely with pharmaceutical industry partners to facilitate the development of our peptidomimetics into drugs.

Project availability:

PhD
Honours

Project supervisors:

Professor Ross Bathgate

Project co-supervisors:

Professor Akhter Hossain

Cox Group

Contact: A/Professor Andrew Cox
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Email: andrew.cox@petermac.org
Website: go.unimelb.edu.au/pd5i

Key focus areas

- Cancer
- CRISPR/Cas9
- Molecular mechanisms of disease
- Biochemistry
- Discovery research
- Metabolism

Associate Professor Andrew Cox leads a lab at the Peter MacCallum Cancer Centre. His research team use zebrafish as a model system to understand the metabolic regulation of growth control in the context of development, regeneration and cancer (EMBO J 2018, Developmental Cell 2022, PNAS 2023, Developmental Cell 2024).

Project: Investigating the interplay between oxidative stress and oncogenic KEAP1-NRF2 pathway in liver cancer

Many of the major risks factors for developing liver cancer such as alcohol, obesity, smoking and toxin exposure share in common a role for oxidative stress. NRF2 is a transcription factor that plays a pivotal role in the cell's adaptation to oxidative stress. The NRF2 pathway is frequently mutated in solid tumours, leading to NRF2 activation. Despite the high prevalence of NRF2 activation in cancers, we still lack a clear appreciation of how this major oncogene drives tumorigenesis. We have recently used transcriptomic and metabolic profiling in zebrafish models to examine the role NRF2 plays in remodeling metabolism during liver development, regeneration and cancer. Building on these preliminary studies, we currently have research project that aims to:

1. Develop new zebrafish models of liver cancers driven by oncogenic NRF2 activation.
2. Identify the metabolic dependencies of NRF2-driven liver cancer.
3. Reveal therapeutic vulnerabilities of NRF2-driven liver cancer.

Students will gain experience in a variety of biochemistry/cell biology/molecular biology techniques including CRISPR/Cas9-mediated gene editing, transcriptomics, tissue clearing and advanced microscopy.

Project availability:

Honours
Master of Biomedical Science
PhD

Project supervisor:

A/Professor Andrew Cox

Project co-supervisor:

Dr Athena Ong

Project: Role of the Hippo-YAP pathway in tumour initiation

The Hippo-YAP pathway is an evolutionarily conserved cascade that plays a fundamental role in governing organ size control, stem cell homeostasis and cancer. The Hippo-YAP pathway is regulated by a range of environmental cues including nutrient status. Although many of the inputs into the Hippo pathway have been identified, less is known about how YAP drives tumourigenesis. Our previous studies revealed that the oncogenic Hippo-YAP pathway reprograms anabolic metabolism (nucleotide and lipid biosynthesis) to fuel liver cancer. Building on these studies, we currently have research projects that aim to:

1. Examine how YAP initiates liver tumour formation.
2. Elucidate the mechanisms by which YAP reprograms metabolism.
3. Examine role of YAP in regulating cancer-associated cachexia.

Students will gain experience in a variety of biochemistry/cell biology/molecular biology techniques including CRISPR/Cas9-mediated gene editing, Next generation sequencing (transcriptomics), Mass Spectrometry (metabolomics), tissue clearing and advanced microscopy.

Project availability:

Honours
Master of Biomedical Science
PhD

Project supervisor:

A/Professor Andrew Cox

Project co-supervisor:

Associate Professor Kristin Brown

Crack Group

Contact: Professor Peter Crack
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Website: go.unimelb.edu.au/fz5i

Key focus areas

- Biomedical neuroscience
- Cell Signaling
- Drug Design
- Neuroinflammation
- Neurotrauma
- Neuropharmacology

Professor Peter Crack's laboratory at the University of Melbourne investigates the molecular mechanisms underlying neuroinflammation and neurodegeneration, with a particular focus on the type-I interferon and STING (Stimulator of Interferon Genes) pathway. The lab integrates cutting-edge techniques—including functional genomics, pharmacology, and multi-omics—to explore how innate immune signaling drives pathological changes in the brain. A key research priority is developing and testing novel CNS-penetrant STING inhibitors to treat conditions such as traumatic brain injury, motor neuron disease, and other neurodegenerative disorders.

Project: Understanding neuroinflammation in acute brain injury

This project, supervised by Professor Peter Crack, investigates the role of neuroinflammation in acute brain injury, with a focus on the STING (Stimulator of Interferon Genes) signaling pathway. Using transgenic mouse models, pharmacological tools, and advanced imaging and molecular techniques, the project aims to identify the key immune cell populations and signaling events that drive secondary brain damage. The findings will inform the development of novel therapies targeting neuroinflammatory mechanisms to improve outcomes after traumatic brain injury.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Professor Peter Crack

Project co-supervisors:

Dr Bruce Wong

This project, supervised by Professor Peter Crack, investigates the role of neuroinflammation in acute brain injury, with a focus on the STING (Stimulator of Interferon Genes) signaling pathway. Using transgenic mouse models, pharmacological tools, and advanced imaging and molecular techniques, the project aims to identify the key immune cell populations and signaling events that drive secondary brain damage. The findings will inform the development of novel therapies targeting neuroinflammatory mechanisms to improve outcomes after traumatic brain injury.

Project: Understanding neuroinflammation in Parkinson's disease

Supervised by Professor Peter Crack, this project explores the contribution of neuroinflammation to the progression of Parkinson's disease, with a focus on type-I interferon and STING signaling pathways. Using preclinical models, the project will investigate how innate immune activation contributes to dopaminergic neuron loss and motor dysfunction. Techniques include immunohistochemistry, gene expression profiling, and pharmacological modulation. Insights gained will support the development of targeted anti-inflammatory strategies to slow neurodegeneration and improve therapeutic outcomes in Parkinson's disease.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Professor Peter Crack

Project co-supervisors:

Dr Bruce Wong

Project: Neuroinflammation and motor neurone disease

Supervised by Professor Peter Crack, this project investigates the role of neuroinflammation in motor neurone disease (MND), focusing on the STING and type-I interferon signaling pathways. Using established MND mouse models, the project will examine how innate immune activation contributes to motor neuron degeneration and disease progression. Techniques include immunofluorescence, transcriptomic analysis, and pharmacological intervention. The goal is to identify novel therapeutic targets within inflammatory pathways to slow disease progression and improve outcomes for individuals living with MND.

Project availability:

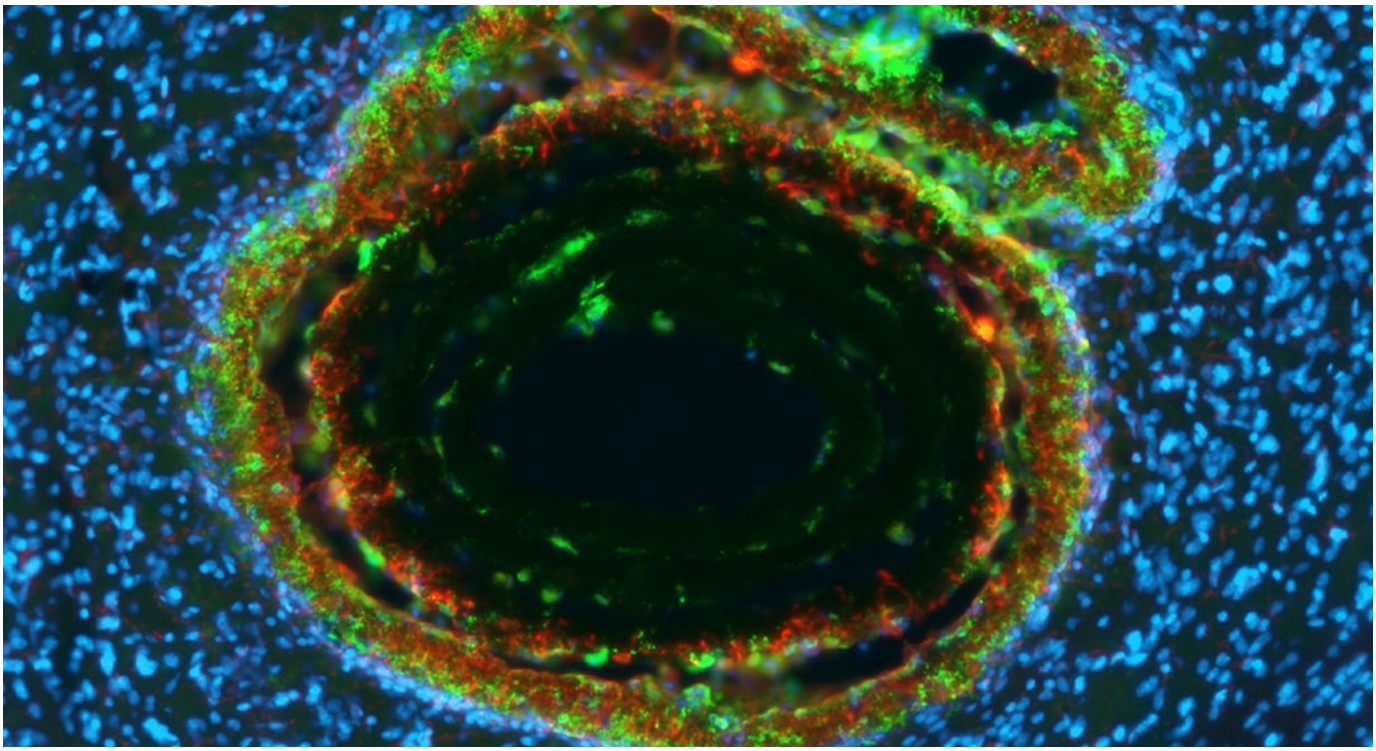
PhD

Project supervisor:

Professor Peter Crack

Project co-supervisors:

Dr Bruce Wong



Edgington-Mitchell Group

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Website: go.unimelb.edu.au/iz5i

Key focus areas

- Biochemistry
- Cancer
- Cell Biology
- Chemical Biology
- Molecular mechanisms of disease
- Proteomics

The Protease Pathophysiology Lab uses a chemical biology approach to understand the mechanisms by which proteases contribute to normal cellular function and how these mechanisms can go wrong to cause disease. Our overarching goals are to establish proteases as diagnostic or prognostic biomarkers for inflammation and cancer and as novel drug targets for the treatment of these diseases.

Project: Beyond the Lysosome: Unveiling New Roles of Canonically Lysosomal Proteases

Lysosomal proteases, known as the cell's "garbage disposals," degrade proteins to supply nutrients for new protein synthesis. In the context of disease, including cancer and inflammation, lysosomal proteases can be misdirected to extra-lysosomal locations, including the nucleus, cytoplasm, and the extracellular space. How this occurs and the resulting impact on cell function remain incompletely understood. This project utilizes advanced proteomics techniques to investigate how these proteases shape extra-lysosomal proteomes. Combining chemical tools and molecular biology techniques, we will explore the functional consequences of mistrafficked proteases on cellular processes and uncover mechanisms that sustain their activity outside the lysosome. The findings will provide critical insights into how extralysosomal proteases contribute to disease pathogenesis, and aid in their validation as therapeutic targets.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Dr Laura Edgington-Mitchell

Project co-supervisors:

Nichollas Scott

Project: Molecular scissors moonlighting as glue

Proteases are enzymes that cut proteins for degradation or to regulate functions. To understand the multi-faceted roles of proteases and how they can go rogue to cause disease, complete characterisation of their substrates is required. This has been hampered by limitations in technology to identify cut sites under physiological conditions in a systematic, unbiased manner. This project seeks to innovate methodology to globally profile the cuts made by specific proteases by applying state-of-the-art technology. The method will be applied to study the mechanisms and consequences of these cuts to assign function to a bifunctional enzyme that exhibits both protease and ligase activity.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Dr Laura Edgington-Mitchell

Project co-supervisors:

Nichollas Scott

Ghosal Group

Contact: Dr Debnath Ghosal
Location: Bio21 Molecular Science and Biotechnology Institute
Email: debnath.ghosal@unimelb.edu.au
Website: ghosallab.com

Key focus areas

- Biochemistry
- Cell Biology
- Microscopy
- Microbiology
- Structural Biology

Our lab is focused on electron cryo-tomography (cryoET) and cryo-focused ion beam milling (cryoFIBSEM) to investigate protein structures in situ (inside cells, in their endogenous environment, without expression and purification). Very few laboratories in the world have this capability, we are situated in bio21 which has Australia's most advanced electron microscopy facility.

Project: In situ characterisation of host-pathogen interactions by cryoEM

Microbial pathogenicity is dependent on host-pathogen interactions. Microbes often adhere to host cells and inject effectors using macromolecular machines, this enables them to remodel the host cells causing adherence, infection, or cell death. Previously, these interactions could not be visualised at high resolution, light microscopy is methods are limited to >10 nm and cells are too thick for conventional electron microscopy. Our laboratory uses cutting-edge technologies called: cryo-focused ion beam milling (cryoFIBSEM), and electron cryo-tomography (cryoET) to study these interactions at atomic resolution in their native conditions (in situ). In this project we will visualise active infections in cryo-conditions, we will target the interaction sites using cryo-light microscopy and create thin sections called lamella (100 nm thick) using cryoFIBSEM, these thin sections can be visualised by cryoET to determine the high resolutions structures of protein complexes involved in host-pathogen interactions. Understanding the structure and function on host-pathogen interactions will provide crucial information for the development of antimicrobial therapies.

Project availability:

Honours
PhD

Project supervisor:

Dr Matthew Johnson

Project co-supervisors:

A/Prof Debnath Ghosal

Grinter Group

Location: Department of Biochemistry & Pharmacology (Bio21 Molecular Science and Biotechnology Institute)
Contact: Dr Rhys Grinter
Email: rhys.grinter@unimelb.edu.au

Key focus areas

- Ai Artificial Intelligence
- Biophysics
- Biochemistry
- Chemical Biology
- Structural Biology
- Microbiology

The Grinter lab has a broad interest in understanding the structure and function of cells and biomolecules at a molecular level. We have a focus on the structural biology of proteins, but employ a multidisciplinary approach encompassing genetics, genomics. biochemistry, proteomics, microbiology, and structural biology. We work across several research themes, with a focus on understanding the outer envelope of Gram-negative bacteria as a target for novel antibiotics, and on novel metalloproteins in microbial respiratory chains. We have a strong interest in computational structural biology and the use of artificial intelligence for the design of new proteins as medicines and biotechnology.

Project: Targeting heme-piracy using de novo designed proteins

This project will use cutting-edge artificial intelligence-based de novo protein design to generate novel binders to heme-uptake proteins in the outer membrane of Gram-negative bacterial pathogens. Heme is an essential nutrient for many bacterial pathogens during infection, and bacteria use outer-membrane transporters to import it directly and obtain it from host proteins like hemoglobin. Blocking heme-uptake using de novo designed protein represents a promising new strategy for combating infection by antibiotic-resistant bacterial pathogens like E. coli, Haemophilus influenzae, or Neisseria meningitidis. This project will involve the computational design of novel binders, their production using recombinant technology, followed by their testing using biophysical/biochemical methods and microbiology methods.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Dr Rhys Grinter

Project: Developing outer membrane protein assembly as a target for novel antibiotics

The outer membrane of Gram-negative bacteria is essential for their structural integrity. It also serves as an important permeability barrier, selectively preventing antibiotics and other unwanted molecules from entering the cell. The outer membrane is predominantly composed of beta-barrel integral membrane proteins, which are assembled by the BAM complex. BAM complex function is essential for bacterial survival, and proteins or peptides that inhibit it are promising novel antimicrobials. In this project, we are developing a class of antimicrobial protein that tightly binds and inhibits the BAM complex at sub-nanomolar concentrations. We are working to understand the structural and molecular basis for BAM inhibition; to understand the effect this has on the bacterial cell, and to test and engineer these proteins as next-generation antimicrobials.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Dr Rhys Grinter

Project: Understanding long-range quinone transport by respiratory chain coupling proteins

The respiratory chain is one of the most fundamental cellular structures. It uses a series of membrane-associated enzyme complexes to transfer electrons from fuel compounds to oxygen, generating a proton motive force across the plasma membrane that drives ATP production. Membrane-bound quinones are fundamental to electron transport chains, acting as electron carriers between these enzymes. In this project, we are studying a novel family of protein complexes that are common across microbial respiratory chains. These complexes extract quinone from the membrane and deliver it to diverse respiratory enzymes. The structural and molecular basis for this process is poorly understood and represents a fundamental mechanism in respiratory chain function. This project will use recombinant protein production to isolate novel enzyme complexes and use biochemistry and structural biology to study their function. It will combine this with cellular microbiology to understand their role in the producing microbe.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Dr Rhys Grinter

Project: Developing modulators of G-protein coupled receptors using de novo protein design

G-protein coupled receptors (GPCRs) are a diverse and abundant family of membrane proteins in human cells, responsible for transducing signals across the plasma membrane in response to chemical stimuli. They are the most important family of proteins for drug development, with ~33% of all drugs targeting them. Despite this, many GPCRs are important for diseases, like autoimmunity, cancer, Alzheimer's disease, and heart failure are not currently targeted by drugs. In this project, we will use artificial intelligence-based de novo protein design to develop binders capable of modulating the function of GPCRs involved in disease-causing processes. This project will involve the computational design of binders targeting GPCRs, their recombinant expression and screening using biochemical/biophysical methods and cellular assays.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Dr Rhys Grinter

Hatters Group

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Key focus areas

- Proteomics
- Cell Biology
- Biophysics
- Organelles
- Neurodegeneration

The Hatters group investigates the molecular origins of neurodegenerative diseases with an emphasis on Huntington and Motor Neuron diseases. The lab is particularly focused on the quality control mechanisms of protein folding, and aberrant mechanisms involving protein misfolding and aggregation. Key interests include developing novel biosensors and tools to explore the biology in cellular and animal models of disease.

Project: Exploring how protein aggregation causes toxicity in Huntington disease

A signature of Huntington's Disease is the formation of inclusion bodies, which are large micrometre-sized aggregates in neurons by the Huntingtin protein. We know from model systems that these aggregates lead to cytotoxicity. However, we do not understand the mechanisms behind this. Insight into this question is critical to planning new therapeutic strategies for this otherwise incurable and fatal condition. Our lab has uncovered intriguing proteomics data, suggesting that specific endogenous proteins are sequestered inside the inclusion bodies in cell models. This novel finding leads us to hypothesise that their sequestration depletes them from their normal function, potentially explaining the observed toxicity. This unique perspective on the role of these proteins in Huntington's Disease could open up new avenues for therapeutic interventions.

Project availability:

Master of Biomedical Science

Project supervisor:

Professor Danny Hatters

Project co-supervisors:

Dr Michal Ugarenko

Project: Determining the proteins that bind to specific protein complexes in Huntington Disease

Our lab strongly focuses on developing and applying proteomics methods to understand how specific protein interactions form in neurodegenerative diseases. In this exciting project, you will work with our team to build a new proteomics method dedicated to finding binding partners to a specific complex formed between the Huntingtin Protein and a protein called PCNA, which is involved in DNA mismatch repair. The benchmark approaches to finding protein interactors involve fusing APEX2, an engineered ascorbate peroxidase, to a target protein. APEX2 catalyses biotin-phenols instantly conjugated to proteins within a few nanometres of proximity in the presence of hydrogen peroxide. Here, you will be developing an approach that will restrict the activity of APEX2 to fold only when a specific complex is formed (e.g., Huntingtin and PCNA). The strategy will involve expressing Huntingtin and PCNA fused to two split fragments of APEX2. These fragments should reconstitute into a functional enzyme upon binding between Huntingtin and PCNA. The project will involve testing various design configurations to establish the assay and then implementing it to find novel proteins associated with the Huntingtin:PCNA complex. You will learn molecular cloning, proteomics, cell biology, and imaging methodologies.

Project availability:

Master of Biomedical Science

Project supervisor:

Professor Danny Hatters

Project co-supervisors:

Dr Michal Ugarenko

Hinde Group

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Key focus areas

- Cell Biology
- Biophysics
- Molecular Imaging
- Microscopy
- Epigenetics

Inside the nucleus of a living cell, DNA is folded into a multi-layered 3D structure called chromatin. At any moment in time, thousands of proteins are diffusing throughout this structural framework - scanning the genome for a target DNA sequence. The question is "does the chromatin network serve as 'road map' for molecular traffic during DNA target search?" To investigate this, we are developing microscopy methods - based on fluorescence lifetime and correlation spectroscopy - to track how proteins move throughout the DNA networks of a living cell. Using this technology we have discovered sub-micron rearrangements in chromatin density that direct the diffusive route of DNA repair factors to damage sites and mediate transcription factor accumulation at specific nuclear locations. This body of work demonstrates an active role for 3D chromatin organisation in nuclear factor navigation. This is important because a hallmark of cancer is genome dysregulation. We therefore now aim to investigate how disruptions to chromatin structure redirect nuclear traffic and the implications this has for maintaining genome function. The current projects in the lab are: (1) mapping the spatial connectivity of nuclear architecture in health and disease, (2) chromatin dynamics during the DNA damage response, and (3) the role of protein oligomerisation in transcription factor DNA target search.

Project: Quantitative imaging of three-dimensional genome architecture in a living cell

Live cell nucleus architecture has emerged as a key player in DNA target search and maintenance of genome integrity. In recent work we have developed a series of fluorescence microscopy methods to track the movement of molecules around the complex DNA networks within the nuclei of live cells. Based on fluorescence lifetime and fluctuation spectroscopy, this technology has the spatial and temporal resolution to map the impact genome organisation has on nuclear traffic. From using these methods, we have discovered that DNA networks rearrange to genome structures that facilitate DNA repair and transcription factor recruitment to target DNA sites. The aim of this biophysical project is to investigate how genome organisation serves as 'road map' for DNA-binding proteins to navigate the nucleus and maintain genome function.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

A/Prof Elizabeth Hinde

Project co-supervisors:

Xiaomeng Zhang

Project: Role of chromatin dynamics during the DNA damage response

Tumour suppression relies on the preservation of genome integrity and this is maintained by a cellular surveillance system called the DNA damage response (DDR). Amazingly the DDR can almost instantaneously detect a genomic lesion. However, how it spatiotemporally coordinates DNA repair factor recruitment to that damage site is not currently understood. We recently demonstrated that double-strand breaks (DSB) induce a transient local 'opening' of chromatin at the damage site that is bordered by an increased level of chromatin compaction (Hinde et al. 2014 Biophys. J 107(1)). These dynamic rearrangements in local DNA density appear to facilitate DNA repair factor accumulation at only the damage site. The overall aim of this project is to investigate how these changes in chromatin compaction modulate genome topology to facilitate the DDR. By use of a fluorescence lifetime imaging microscopy (FLIM) based assay, we will test the hypothesis that spatial heterogeneity in chromatin compaction during the DDR serves to facilitate DNA repair factor access whilst blocking transcription of neighbouring genes.

Project availability:

Honours
Master of Biomedical Science
PhD

Project supervisor:

A/Prof Elizabeth Hinde

Project co-supervisors:

Xiaomeng Zhang

Keenan Group

Location: Department of Biochemistry & Pharmacology (Bio21 Molecular Science and Biotechnology Institute)
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Key focus areas

- Epigenetics
- Immunology
- Molecular mechanisms of disease
- Respiratory diseases
- Virology
- Genomics
- Biostatistics
- CRISPR/Cas9
- Pharmacology
- Translational and Clinical Research

Research in the Keenan laboratory aims to understand how epigenetic mechanisms cause or contribute to diseases of the immune system. The ultimate goal of this research is to develop new therapeutic strategies to restore healthy immunity. We apply a wide-range of cutting-edge experimental approaches including Flow Cytometry, Next-Generation Sequencing (e.g. RNA-seq, ATAC-seq, ChIP-seq, CUT&Tag, Chromosome Conformation Capture), CRISPR/Cas9, Immunological assays, and in vivo models of disease.

Project: Unravelling the epigenetic consequences of respiratory viral infection

Early life respiratory viral infections are a major cause of hospitalistion in children, and predispose individuals to the development of asthma. This project will explore the epigenetic changes that occur in the lung and/or immune system following infection with common respiratory viruses (e.g. RSV, rhinovirus, influenza, SARS-CoV-2) in order to determine whether these epigenetic changes predispose individuals to allergen-induced asthma. This project will suit a student with an interest in mucosal immunity and epigenomic techniques.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Dr Christine Keenan

Project co-supervisors:

Dr Michelle Ruhle

Project: Investigating the role of Kmt2d in haematopoiesis

Kmt2d is an epigenetic enzyme frequently mutated in cancers such as leukemias, and is the main causative gene for the developmental disorder Kabuki syndrome (KS). KS patients frequently present with immunological issues such as immune deficiency and autoimmunity, suggesting Kmt2d plays important roles in both the development and function of white blood cells. This project aims to understand how Kmt2d controls the development and function of blood cells using a novel mouse model where Kmt2d is specifically deleted in the haematopoietic compartment using an inducible Cre-loxP system. This project will characterise the phenotype of this novel mouse model using multi-dimensional flow cytometry and fluorescence microscopy in order to assess the role of Kmt2d in predisposition to leukemia / immune deficiency / autoimmunity.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Dr Christine Keenan

Project co-supervisors:

Dr Michelle Ruhle

Project: Targeting epigenetic mechanisms to improve immunity

Targeting epigenetic mechanisms is emerging as an exciting therapeutic strategy with potential to re-wire the genome in disease. The Keenan lab has particular interest in diseases of the immune system. We have exciting preliminary data that implicates methylation of lysine 4 of histone H3 (H3K4me) as an important molecular controller of the B cell lineage of the immune system, the cell-type responsible for producing functionally diverse antibodies to protect us from infection. This project will investigate the role of H3K4 demethylase enzymes such as LSD1 and JARID1 in B cells using pharmacological and CRISPR/Cas9 approaches. By understanding the molecular regulation of B cells, we hope to develop new therapeutic approaches to improve immune responses to vaccination and infection. Other techniques that will be learned include multi-dimensional flow cytometry, advanced genomics techniques such as RNA-seq/ChIP-seq/ATAC-seq/CUT&Tag/CUT&Run/Hi-C, and mouse models of immune responses. The successful student will ideally know (or be willing to learn) some basic R coding to enable you to interact with your data.

Project availability:

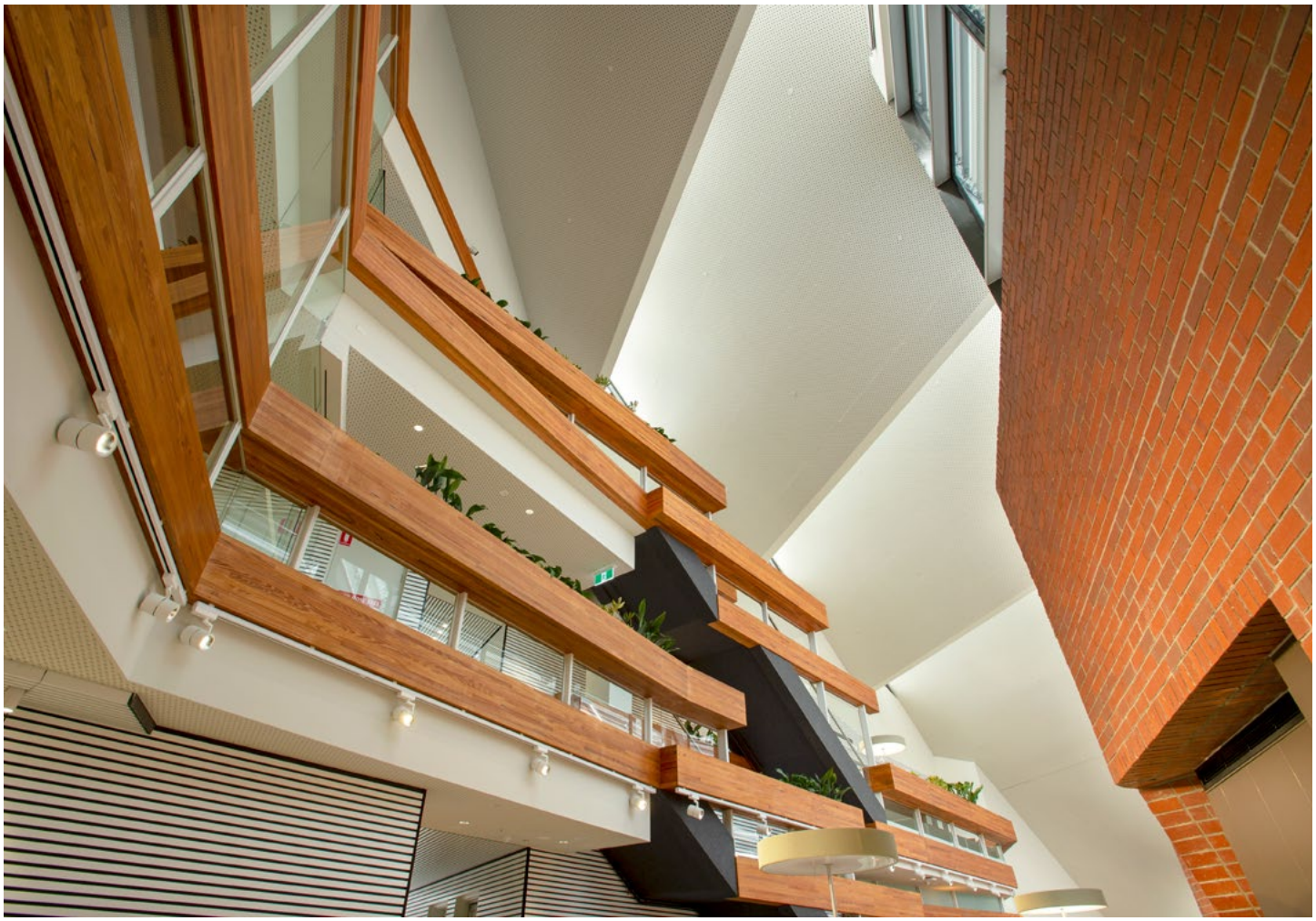
PhD

Project supervisor:

Dr Christine Keenan

Project co-supervisors:

Dr Michelle Ruhle



Kristin Brown Group

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Key focus areas

- Cancer
- Cell Biology
- Cell Signalling
- Metabolism

Almost a century ago, pioneering studies by Otto Warburg revealed a fundamental difference between the metabolism of normal and cancer cells. However, it is only in the last decade that significant inroads have been made to understanding the critical importance of deregulated cellular metabolism in cancer. Consequently, there is growing interest in developing therapeutic strategies to exploit the metabolic vulnerabilities of cancer cells. The Brown laboratory uses a variety of biochemistry, molecular biology and cell biology techniques to investigate the fundamental mechanisms contributing to regulation of cellular metabolism, and the impact of aberrant cellular metabolism on tumour initiation and progression.

Project: Targeting cell metabolism to overcome chemotherapy resistance in triple-negative breast cancer

Triple-negative breast cancer (TNBC) is defined by the lack of estrogen receptor (ER), progesterone receptor (PR) and absence of human epidermal growth factor receptor-2 (HER2) amplification. Consequently, TNBCs are impervious to therapies commonly used in other breast cancer subtypes and treatment options are largely limited to conventional chemotherapy agents that are only effective in approximately 30% of patients. Identification of novel and actionable strategies to treat TNBC would represent a major advance for the management of this deadly disease. Cancer cells exhibit dramatic alterations in cell metabolism, which support cell growth, proliferation and survival. Our studies have revealed that reprogramming of cellular metabolism is also a component of the highly coordinated response to chemotherapy exposure. Moreover, we have shown that the unique metabolic requirements of chemotherapy-treated cancer cells can be targeted for therapeutic gain. The aims of this project will be to:

1) identify adaptive metabolic reprograming events triggered by chemotherapy exposure
2) determine how these metabolic reprogramming events drive chemotherapy resistance
3) identify novel therapeutic approaches to exploit adaptive metabolic reprogramming events and sensitize TNBC cells to chemotherapy.

Students will gain experience in a variety of biochemistry and molecular biology techniques including metabolomics, transcriptomics and CRISPR-Cas9 genetic screens.

Project availability:

PhD
Honours
Master of Biomedical Science

Project supervisors:

A/Prof Kristin Brown

Project: Metabolic regulation of antigen presentation and tumour immune escape

Tumour development and progression are dependent on the ability of cancer cells to evade immune control. The strategies employed by cancer cells to escape immune control are diverse but one of the primary mechanisms involves loss of immune recognition as a result of dysfunctions in antigen processing and presentation (APP). APP is the process by which antigenic peptides are generated and loaded onto MHC-I molecules for display on the cell surface. Reduced MHC-I expression has been observed across diverse tumour types and is associated with poor prognosis. Cancer cells are dependent on reprogramming of cellular metabolism to promote cell growth, proliferation and survival. Surprisingly, the contribution of metabolism to the regulation of APP and immune escape is poorly understood. The aims of this project will be to:

1) identify specific metabolic pathways that contribute to the regulation of MHC-I expression
2) determine how these metabolic pathways impact APP
3) identify strategies to target metabolism to overcome immune escape and enhance the efficacy of currently existing immunotherapies.

Students will gain experience in a variety of biochemistry and molecular biology techniques including metabolomics, transcriptomics and CRISPR-Cas9 genetic screens.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisors:

A/Prof Kristin Brown

Project: Cutting off the fuel supply to starve cancer: identifying the nutrient dependencies of cancer cells

Project Description: To meet the demands associated with rapid proliferation, cancer cells must be able to synthesise critical nutrients or acquire them from the surrounding microenvironment. Until recently, little attention has been paid to the fact that cell culture media does not mimic the in vivo metabolic environment. We have therefore adopted the use of a physiologically relevant media that can be manipulated to investigate the impact of nutrient availability on cancer cells. Coupled with genetic approaches to perturb metabolic pathway activity, we seek to identify the nutrient dependencies of cancer cells with a view to developing novel strategies for cancer therapy. These studies will focus on determining whether key oncogenic transcription factors that we have discovered to play a significant role in the cellular response to altered nutrient availability (e.g. the YAP/TAZ-TEAD pathway), can be targeted to improve treatment options for cancer patients. Students will gain experience in a variety of biochemistry and molecular biology techniques including metabolomics, transcriptomics and CRISPR-Cas9 genetic screens.

Project availability:

PhD
Honours
Master of Biomedical Science

Project supervisors:

A/Prof Kristin Brown

Mackay Group

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Key focus areas

- Immunopharmacology
- Cell Biology
- Cell Signaling
- Allergic disease

Our team is focused largely around the fascinating mast cell and its role in allergic and non-allergic disease. In particular, we want to better understand how these cells are activated, the mediators they release and if we can translate this knowledge to generating new therapeutics.

Project: MRGPRX2-mediated drug hypersensitivity reactions

Exciting new work has identified that mast cells have a receptor called MRGPRX2 that can be directly activated by many clinically used drugs including certain antibiotics. This might explain a significant number of drug hypersensitivity reactions. However, it is unclear as to why only some individuals react so adversely to these drugs. In this project you will examine mast cell activation through MRGPRX2 and identify pathways that enhance the activity of this pathway. This information will hopefully be of utility in better understanding, predicting and treating MRGPRX2-mediated drug hypersensitivity.

Project availability:

Master of Biomedical Science
Honours
PhD

Project supervisor:

A/Prof Graham Mackay

Maher Group

Location: Department of Biochemistry & Pharmacology (Bio21 Molecular Science and Biotechnology Institute)
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Key focus areas

- Biochemistry
- Bioinorganic chemistry
- Chemical Biology
- Infection and Immunity
- Structural Biology
- Protein Chemistry

Our research sits at the interface of chemistry and biology and investigates the roles of transition metals such as iron, copper, manganese and zinc in biological systems. These trace nutrients are essential for all forms of life – approximately 30% of all proteins bind transition metals and 50% of enzymes requires these cofactors for activity.

Project: Nutritional Immunity and Antibiotic Design

We have several exciting projects examining the role of transition metals in infection and immunity. We know that bacteria (like all other organisms) require transition metals for life, but that they are also toxic in excess. In fact, these elements form part of the human innate immune response where they are harnessed as antibacterial agents. The investigation of these systems is of critical importance currently, as the world is facing a crisis of antibiotic resistance. At the current trajectory, it is estimated that by 2050, 10 million people per year will die because of antimicrobial resistance. Therefore, there is an urgent need to develop new strategies for the treatment of bacterial infections. These projects aim to characterise the protein systems that control bacterial transition metal nutrition and that are impacted by metal toxicity. We do this using structural biology, protein chemistry, biochemical, bioinorganic and biophysical techniques.

Project availability:

Master of Biomedical Science
Honours

Project supervisor:

Professor Megan Maher

McConville Group

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Key focus areas

- Analytical Biochemistry
- CRISPR/Cas9
- Chemical Biology
- Metabolomics
- Microbiology
- Pathogens

The McConville group is interested in understanding how different microbial pathogens cause diseases in humans, with the view of identifying new and more effective drug targets. The primary focus of the group is on two parasitic protists, Leishmania spp and Toxoplasma gondii, which both cause devastating diseases in humans. We investigate aspects of parasite and host cell metabolism that are essential for virulence and have established expertise in cutting-edge metabolomics technologies, biochemical, molecular and cell biology techniques (including CRISPR/Cas9), infection models and drug screening.

Project: Identifying essential metabolic genes in Leishmania: Linking Metabolism to Parasite Survival and Virulence

Leishmania are protozoan parasites responsible for leishmaniasis, a neglected tropical disease that affects millions of people worldwide. These pathogens specifically target macrophages in the mammalian host, proliferating within the hydrolytic lysosome of these host cells. How these parasites adapt metabolically to this niche remains poorly defined. This project will systematically investigate the role of selected, potentially essential metabolic genes in Leishmania survival, proliferation, and virulence. These include genes involved in carbohydrate (ribokinase, galactokinase) amino acid (amino acid permeases) and fatty acid (b-oxidation) catabolism. Using CRISPR-Cas9–based gene knockout (KO) and inducible KO approaches (where null mutants are not viable), mutants will be generated and validated using LeishGEdit and conditional gene deletion strategies. Phenotypic characterisation of KO lines will involve a suite of complementary approaches, including:

- Fluorescent tagging and microscopy to determine subcellular localisation
- 13C-stable isotope labelling and mass spectrometry-based metabolomics to assess metabolic impact
- Plate-based growth and viability assays
- Macrophage infection models and high-content imaging to evaluate intracellular survival and infectivity This work will define the major carbon sources used by intracellular parasite stages and help prioritise new metabolic targets for therapeutic development.

Key Techniques and Concepts: CRISPR-Cas9, inducible gene knockout, fluorescence microscopy, metabolomics, 13C-labelling, mass spectrometry, growth assays, macrophage infection, high-content imaging.

Key References:

- Beneke & Gluenz (2019) Methods Mol Biol 1971:189–210
- •Yagoubat et al. (2020) Cell Microbiol 22(5):e13159
- Saunders et al. (2018) Mol Microbiol 108(2):143–158
- Dagley et al. (2015) Assay Drug Dev Technol 13(7):389–401

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Professor Malcolm McConville

Project co-supervisors:

Dr Eleanor Saunders, Dr Fleur Sernee, Dr Vinz Hofferek

Project: Immunometabolism in Leishmania Infection: Exploring Host–Parasite Metabolic Interplay

Leishmania are intracellular protozoan parasites responsible for leishmaniasis, a neglected tropical disease. Following infection, Leishmania parasites reside within the parasitophorous vacuole (PV) of host macrophages, where they must adapt to an environment characterised by oxidative stress, nutrient limitation, and host immune pressure. The metabolic state of the host macrophage—whether inflammatory (M1) or anti-inflammatory (M2)—profoundly influences parasite survival and disease progression. However, the metabolic crosstalk between parasite and host remains poorly understood. This project investigates the interplay between Leishmania metabolism and host macrophage immunometabolism, with a focus on how different macrophage polarisation states affect parasite survival, and vice versa. Leveraging a library of over 40 Leishmania metabolic enzyme knockout (KO) strains developed by the McConville lab, we will screen these mutants in M0, M1, and M2 polarised human macrophages using high-content imaging. This assay will evaluate both: Parasite survival and proliferation, and Host cell metabolic and inflammatory readouts (e.g. lipid droplet accumulation, mitochondrial membrane potential). To further dissect host-parasite metabolic interactions, the nutritional environment of macrophages will be modulated using defined media (e.g. high glucose, glutamine, or arginine),

metabolic inhibitors (e.g. 2-deoxyglucose, sodium fluoroacetate), and potentially host KO models. In parallel, we will conduct 13C-labelled metabolic flux analysis and mass spectrometry-based metabolomics on polarised, infected, and uninfected macrophages. This will help identify key metabolic pathways that are altered during infection and contribute to parasite persistence or clearance. The outcomes of this study will provide novel insights into how Leishmania exploits or adapts to different host metabolic states, identifying potential vulnerabilities in both parasite and host that could be targeted for therapy. Key Techniques and Concepts: Macrophage polarisation (M0, M1, M2), Leishmania metabolic mutants, metabolomics, 13C-labelling, mass spectrometry, fluorescence microscopy, high-content imaging, metabolic inhibitors, host-pathogen interactions. Key Reference: Dagley MJ, Saunders EC, Simpson KJ, McConville MJ. (2015) Assay Drug Dev Technol 13(7):389–401.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Professor Malcolm McConville

Project co-supervisors:

Dr Eleanor Saunders, Dr Fleur Sernee, Dr Vinz Hofferek

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Key focus areas

- Biochemistry
- Cell Biology
- Metabolism
- Metabolomics
- Microbiology
- Molecular Imaging

The McConville group is interested in understanding how intracellular microbial pathogens, responsible for diseases such as malaria, human leishmaniasis and toxoplasmosis, are able to survive within their human/animal hosts and cause disease. We utilize advanced mass spectrometry approaches, as well as a range of biochemical and genetic techniques to identify microbial metabolic pathways that are switched on during infection and are essential for pathgenesis. We are also interested in identifying metabolic pathways in the host cell that are required for pathogenesis. These studies are directed at understanding fundamental aspects of microbial pathogenesis, as well as developing new anti-microbial or host-directed therapies for these important diseases.

Project: Single-cell metabolite imaging of the coral-microalgal symbiosis

Corals sustain some of the most diverse ecosystems on Earth but are at risk due to warming and acidifying oceans. Coral survival critically depends on the photosynthetic microalgae that live inside the coral and provide the coral with nutrients. Many aspects of this coral-algal relationship remain poorly defined. This project aims to unravel coral-algal interactions with single-cell imaging. Insights from extreme environment corals will reveal how these microalgae may facilitate coral survival under future climate change, providing vital information for reef managers and restoration practitioners. By establishing a novel method, databases and networks, this project will create a powerful forward momentum for coral-algal research.

Project availability:

PhD

Project supervisor:

Dr Wing Yan Chan

Project co-supervisors:

Malcolm McConville

Menden Group

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Key focus areas

- Ai Artificial Intelligence
- Biostatistics
- Machine learning
- Translational and Clinical Research
- Precision medicine

The Menden Lab develops advanced biostatistical, machine learning, and artificial intelligence frameworks to analyse biomedical data, unlock insights into complex diseases, and discover novel intervention strategies. By leveraging deeply characterized biomedical datasets and integrating disease-specific knowledge from collaborations, literature, and data-driven analyses, we aim to empower the next generation of drug target identification, drug repositioning, and precision medicine.

Project: Computational Modeling for Drug Response and Precision Medicine

In this project, the Master's student will develop and apply computational models—ranging from machine learning to biostatistics—to predict drug response, synergy, or resistance using high-throughput screening data from cancer and other diseases. A key focus will be the integration of multi-omics datasets to identify predictive biomarkers and explore patient stratification strategies based on deep molecular profiles. The student will gain hands-on experience in Python and/or R programming, large-scale biomedical data analysis, and the application of state-of-the-art tools in computational biology. Depending on interest and progress, there will be opportunities to explore innovative approaches, such as generative AI and digital twins, to enhance model interpretability and clinical translatability. The project will be conducted within an interdisciplinary research environment, offering close collaboration with clinicians, molecular biologists, and computational scientists. This will provide the student with a unique opportunity to work at the interface of data science and precision medicine.

Project availability:

Master of Biomedical Science
Honours
PhD

Project supervisor:

A/Prof Michael P Menden

Project co-supervisors:

Dr Teresa Sadras

Parker Group

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Key focus areas

- Cancer
- Cell Signaling
- Drug Design
- Neurodegeneration
- Pathogens
- Structural Biology

The focus of our research is to visualise the three-dimensional structures of medically important proteins using structural biology techniques including X-ray crystallography, cryo electron microscopy and AI. A particular focus is proteins that play a role in infection (bacterial, parasitic or viral), cancer (particularly leukaemia, breast and prostate) and neurological diseases (e.g. Alzheimer's, Parkinsons, Lewy body Dementia). The structures provide a detailed understanding of how each protein works and how it contributes to disease. Most importantly, the structures can be used to discover and develop drugs using computational and biophysical approaches.

Project: New approaches to treat Alzheimer's disease

Alzheimer's disease (AD) is the fourth biggest killer in developed countries. Amyloid precursor protein (APP) plays a central role in the development of AD, through generation of the toxic Abeta peptide by proteolytic breakdown of APP. Here we will use X-ray crystallography at the Australian Synchrotron or cryogenic electron microscopy at Bio21 to determine the 3D atomic structures of Abeta bound to therapeutic antibodies currently in clinical trials in order to understand how these molecules recognise Abeta. We use this information to engineer more potent antibodies as treatments for AD. We also have structure-based small molecule drug discovery projects on APP itself and on immune cell receptors that clear toxic molecules from the brain.

Project availability:

PhD
Honours
Master of Biomedical Science

Project supervisor:

Professor Michael Parker

Project co-supervisors:

Dr Emma van der Westhuizen

Project: Drugging the undruggable to combat cancer

Modulating protein–protein interactions represents a huge potential for cancer therapeutic intervention with over 300,000 protein interaction pairs already identified in the human genome. Historically, targeting these interactions with small molecules was not thought possible because the corresponding interfaces would be "undruggable". However, the last decade has seen encouraging breakthroughs via refinement of existing techniques and development of new ones, together with the identification and exploitation of unexpected aspects of protein–protein interaction surfaces. In this project we wish to discover new drugs that disrupt protein-protein interactions in cancer using a mixture of techniques including computational biology, protein expression and purification, X-ray crystallography and measurement of protein interactions.

Project availability:

PhD
Honours

Project supervisor:

Professor Michael Parker

Project co-supervisors:

Dr Tracy Nero

Project: New antibiotics to treat deadly infections

Project Description: Bacterial biofilms are matrix-enclosed bacterial populations adherent to each other and/or to surfaces or interfaces. Bacterial biofilm formation is an extremely common phenomenon with a major economic impact in different industrial, medical and environmental fields. Klebsiella pneumoniae is a Gram-negative, opportunistic pathogen that is frequently associated with infections such as urinary tract infections, pneumonia and septicemia. An important clinical pathogenic mechanism of K. pneumoniae is its ability to form robust biofilms on inanimate surfaces (eg. plastics used in surgical implants). This project aims to inhibit biofilm formation by targeting adhesion molecules using biophysical, computational and structure-based drug design techniques. We also have projects to develop new antibiotics based on key protein targets in the bacterium that have been discovered by our collaborators.

Project availability:

PhD
Honours

Project supervisor:

Professor Michael Parker

Project co-supervisors:

Dr Michelle Christie

Project: Understanding how bacterial pore-forming toxins punch holes in membranes

The beta-barrel pore-forming toxins constitute the largest group of functionally related protein toxins. They are expressed in many species in the prokaryotic and eukaryotic kingdoms and are related to the membrane attack complex/perforin (MACPF) family of mammalian immune defence proteins. Despite their widespread occurrence and role in bacterial pathogenesis and immune defence, the detailed mechanism by which they form pores remains an enigma. The overall aim here is to visualise the 3D structures of family members as a basis for functional studies to reveal the molecular details of how these toxins insert into membranes to form beta-barrel pores and how the process is regulated. The structures will shed light on one of the most fundamental biological events (namely, protein insertion into cell membranes) and also provide the basis for the design of novel tools with various biotechnology applications and the design of novel antibiotics.

Project availability:

PhD
Honours

Project supervisor:

Professor Michael Parker

Project co-supervisors:

Dr Michelle Christie

Project: Overcoming cancer drug resistance

Conventional cancer chemotherapy kills rapidly growing cells indiscriminately, causing significant side-effects and can lead to disease re-occurrence and resistance to the drugs. One of our interests is the Glutathione S-Transferase (GST) family of proteins that function by recognising foreign small molecule toxins in the body, causing them to be eliminated from the cell. Unfortunately, commonly used anti-cancer drugs are also recognised as toxic by GSTs, which are often overexpressed in cancer tissues and are associated with transformation to malignancy and the adaptive resistance to anti-cancer drugs. There is thus an urgent need for the design of new anti-cancer drugs that circumvent the development of GST-mediated resistance to treatment. Recently, there has been an increasing interest in the development of metal-based drugs as effective and potent protein targeted chemotherapies. We are investigating, though structural and biochemical means, how a range of ruthenium, arsenic and osmium-based drugs and drug-like compounds interact with GSTs. Students will investigate how these compounds work, as well as any drug-like molecules we develop, using X-ray crystallography and a range of biophysical techniques.

Project availability:

Honours

Project supervisor:

Professor Michael Parker

Project co-supervisors:

Dr Claire Weekley

Ralph Group

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Key focus areas

- Biochemistry
- Cell Biology
- Genomics
- Infection and Immunity
- Pathogens

The Ralph lab explores the molecular cell biology of Plasmodium falciparum, with a focus on protein trafficking, organelle function, and gene regulation. We characterise promising drug targets from Plasmodium falciparum and other parasites, as well as studying the modes of action and mechanisms of resistance for existing drugs.

Project: Drug resistance in the human malaria parasite Plasmodium falciparum

Resistance to the frontline antimalaria Artemisinin a growing threat to malaria control, particularly in Southeast Asia and parts of Africa. Resistance is associated with mutations in the kelch13 (K13) gene in the causative agent of malaria, Plasmodium falciparum. The native function of K13 in wild-type parasites and how mutations alter its role in drug resistant parasites remain poorly understood. Our recent data show that K13 forms distinct ring-like structures localized to the neck of the cytostome—the parasite's endocytic organelle for haemoglobin uptake. We will compare wild-type and K13 mutant P. falciparum lines to elucidate the function of K13, with a focus on its involvement in parasite feeding and haemoglobin uptake. We will use super-resolution microscopy and electron microscopy to visualize K13 localization, morphology and ultrastructural changes and to measure how K13 mutations impact parasite feeding. Understanding K13's physiological role in wild-type parasites, and how resistance mutations modify this role, will clarify the biological basis of artemisinin resistance. This knowledge could uncover novel aspects of parasite biology and inform new strategies for monitoring and overcoming resistance in endemic regions.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Professor Stuart Ralph

Project co-supervisors:

Dr Emma McHugh

Key Focus areas

- RNA biology and Cancer
- Vaccines
- Pathogens
- Microbiology
- Genomics & Functional genomics

Project: RNA modifications to modulate gene expression

The success of mRNA vaccines with chemically modified RNA bases in immunising against COVID has established the enormous potential of this technology. Current mRNA products make use of synthetic RNA modifications, but disregard how those modifications will be further altered after the RNA is taken up by a cell. We will demonstrate how RNA modifications can fine-tune RNA vaccines and therapies to optimise the amount and duration of protein produced to improve vaccines and RNA therapies. We are also investigating the role RNA modifications play in medically important human parasites including the parasites that cause Malaria and Toxoplasmosis. We will determine how these modifications impact host pathogen interactions.

Project availability:

PhD

Project supervisor:

Professor Stuart Ralph

Project co-supervisors:

Dr Emma McHugh

Robyn Brown Group

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Key focus areas

- Behavioral neuroscience
- Motivated Behaviour
- Translational and Clinical Research
- Neuropharmacology

The Brown group aims to understand the neural mechanisms underlying pathological forms of motivated behaviour such as compulsive forms of eating and addiction, as well as explore the impact of high-fat high-sugar foods on brain and behaviour. We use a multi-disciplinary approach including behavioural models, electrophysiology, chemogenetics, optogenetics, fibre photometry, viral methods and transgenic mouse models.

Project: Understanding the brain mechanisms underlying stress eating

It is well established that stress and emotional state trigger binge eating however the neural drivers of this behaviour remain underexplored. The aim of this project is to investigate brain and blood correlates of stress-induced binge eating in order to gain an understanding of possible neural mechanisms driving this behaviour. Techniques include transcriptomic analysis of blood samples from individuals with binge eating disorder as well as animal behaviour combined with drug testing and neurochemical analyses.

Project availability:

PhD
Master of Biomedical Science

Project supervisors:

A/Prof Robyn Brown

Project co-supervisors:

Dr Christine Keenan; Dr Trevor Steward; Associate Professor Priya Sumithran

Project: Gut-brain mechanisms underlying the negative and positive health outcomes following bariatric surgery

Bariatric surgery is remarkably successful as it produces profound and sustained weight loss in combination with metabolic benefits. These benefits are thought to occur via actions on the gut-brain axis which lead to alterations in the neuroendocrine regulation of appetite and glycaemia. Despite its remarkable effectiveness, evidence is emerging which shows that subset of patients experience adverse mental health effects, including suicide, self-harm, depression and substance use disorders. The causative and contributing factors behind these adverse effects are not well understood. This project explores the utility of using a mouse model of bariatric surgery to determine the mechanisms underlying the impact of this procedure on affective behaviour. Neurochemical analysis of brain tissue will investigate the changes in central gut hormones and their receptors potentially responsible for these changes in behaviour.

Project availability:

PhD
Master of Biomedical Science

Project supervisors:

A/Prof Robyn Brown

Project co-supervisors:

Associate Professor Priya Sumithran

Project: Towards improved therapeutics for binge eating disorder

Binge eating disorder is a debilitating mental health condition which involves a loss of control over eating accompanied by subsequent feelings of distress, guilt and shame. There is only one approved medication for binge eating disorder which is a stimulant medication that has tolerability issues and has a risk of abuse. Thus, there is a need for new medications for binge eating disorder. This project will investigate the brain mechanisms driving binge eating with a focus on the orexin system as a possible therapeutic target. The project will involve behavioural pharmacology, fibre photometry and neurochemical analysis.

Project availability:

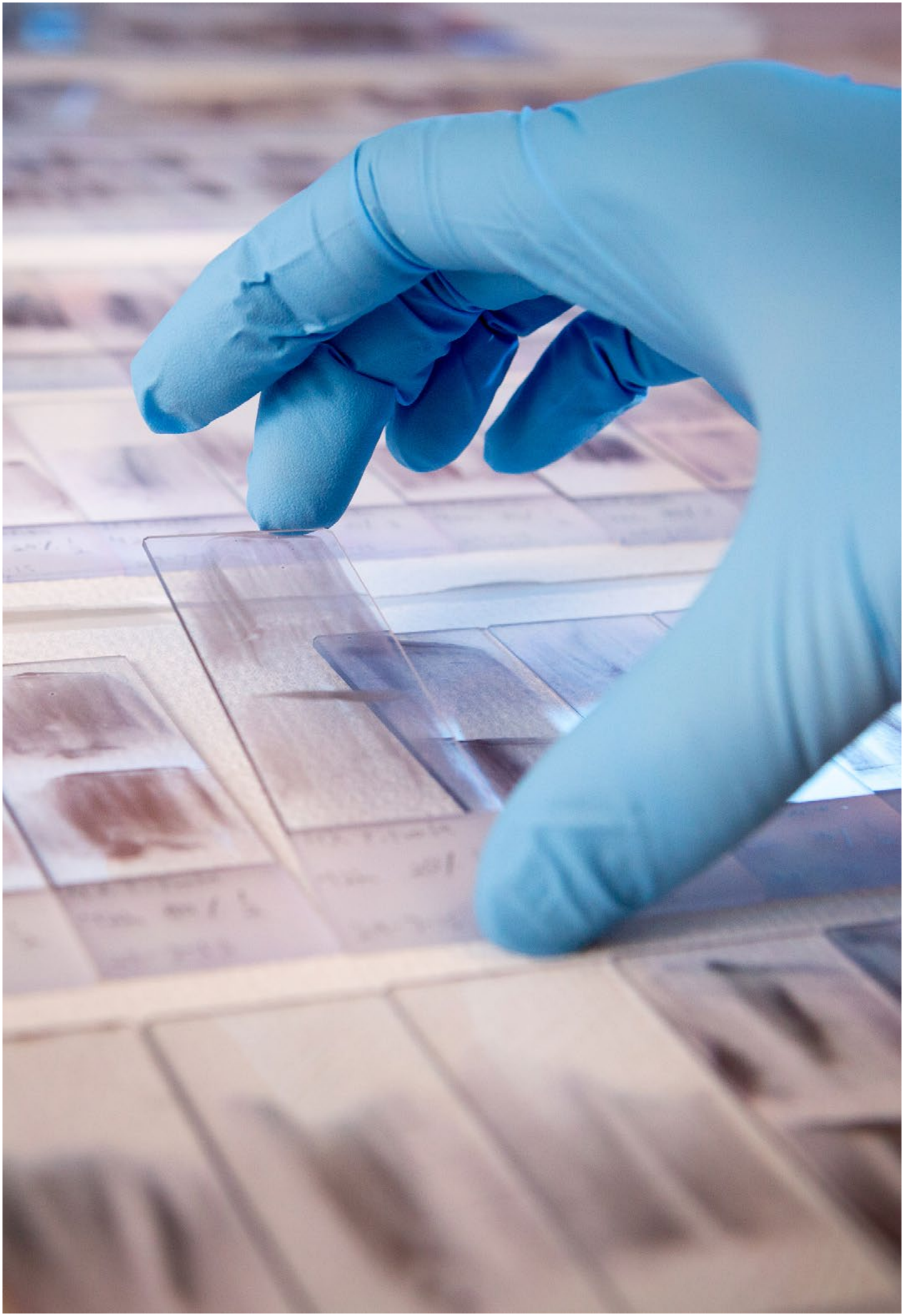
PhD
Master of Biomedical Science

Project supervisors:

A/Prof Robyn Brown

Project co-supervisors:

Dr Morgan James



Rouiller Group

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Key focus areas

- Biochemistry
- Biophysics
- Molecular Imaging
- Structural Biology
- Protein Chemistry

Our lab studies how tiny molecular machines keep cells healthy—or go wrong in disease. We use tools from protein science—like cryo-electron microscopy, mass spectrometry and biochemical assays—to capture and analyse proteins in action. We focus on proteins like p97 (linked to cancer and ALS), membrane transporters (involved in antibiotic resistance and pain), and viral proteins (important for vaccine design). By understanding how these machines work, we aim to uncover new ways to treat diseases.

Project: Unlocking the Secrets of Mitochondrial Shape and Function

This project explores how a key mitochondrial protein, OPA1, helps shape the internal architecture of mitochondria and powers the fusion of their membranes. In this project, you will investigate how OPA1 assembles and functions using a mix of cutting-edge techniques—including structural biology, mass spectrometry, and biophysical tools—and gain hands-on experience in protein science. This project is perfect for students with a strong foundation in biochemistry who are fascinated by how cells work at the molecular level and curious about the molecular machines that keep cells healthy and functioning. Your work will contribute to a deeper understanding of how mitochondria change shape and communicate within cells—processes that are disrupted in neurodegenerative diseases, metabolic disorders, heart disease, and cancer. In the long run, your discoveries could help lay the groundwork for new therapeutic strategies.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Professor Isabelle Rouiller

Project co-supervisors:

Dr Naveen Vankadari

Using powerful imaging tools like cryo-electron microscopy, we capture 3D "snapshots" of proteins in action. We focus on proteins like p97 (linked to cancer and ALS), membrane transporters (involved in antibiotic resistance), and viral proteins (important for vaccine design). By understanding how these machines work, we aim to uncover new ways to treat diseases and develop better therapies.

Project: How Do Molecular Machines Unfold Proteins? Discover the Secrets of p97!

This project dives into p97, a powerful protein-unfolding machine (ATPase) that plays a central role in regulating many cellular processes. In this project, you'll investigate how cofactor proteins help p97 recognize, load, and unfold its targets. You'll use state-of-the-art techniques- including mass spectrometry, cryo-electron microscopy, high-resolution light microscopy and biophysical approaches—you'll uncover how these cofactors control p97's shape and activity. This project is ideal for students with a strong background in biochemistry who are eager to apply their knowledge to the molecular study of protein structure and function. You'll gain hands-on experience with powerful techniques at the cutting edge of protein science and structural biology. By joining this project, you'll contribute to understanding how protein unfolding is regulated in cells—a process that goes wrong in neurodegeneration and is linked to ALS, dementia and Paget's disease. Your discoveries could deepen our understanding of how cells regulate protein quality—and how we might disrupt p97 in cancer or restore its function in neurodegenerative disease.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Professor Isabelle Rouiller

Project co-supervisors:

Dr Naveen Vankadari

Project: Targeting HIV's Weak Spot: Using Structural Biology to Fight Infection

Despite major advances like antiretroviral therapy (ART), HIV remains a global challenge, with over 39 million people currently infected and millions of lives lost. A key obstacle in both treatment and vaccine development is the virus's surface protein, Env, which constantly changes shape and hides behind a shield of sugars—making it hard for the immune system to target. This project focuses on ultra-potent antibodies that can penetrate the virus's protective shield and target its most vulnerable site. You'll use a combination of cryo-electron microscopy and computational analysis to understand how these antibodies work and help design improved versions that stay effective across diverse HIV strains. Ideal for students interested in structural biology and protein engineering, this project offers hands-on experience with cutting-edge tools to support future HIV therapies. This project is ideal for students with a strong computational background who want to combine computational biology and structural biology to tackle global health challenges. Your work will help reveal how the immune system can be harnessed to fight HIV—supporting the design of new therapeutic strategies.

Project availability:

PhD
Master of Biomedical Science

Project supervisor:

Professor Isabelle Rouiller

Project co-supervisors:

Dr Naveen Vankadari

Schneider-Futschik Group

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Key focus areas

- Anti-microbial pharmacology
- Lung Health;
- Pharmacology
- Precision medicine
- Respiratory diseases
- Translational and Clinical Research
- Molecular mechanisms of disease
- Women's Health

The Lab interest focuses on rare and complex lung diseases and chronic illness including ME/CFS and Long COVID. These diseases can have genetic, autoimmune, and/or environmental origins and are often difficult to diagnose. The interdisciplinary nature of our research, blending genetics, molecular biology, and therapeutics, underscores its novelty, offering hope for transformative treatments and ultimately, a brighter future for individuals living with chronic, complex diseases.

Project: Improving lung health for rare and complex lung diseases

In this study, we will correlate the functional manifestations of cystic fibrosis with pathological changes in histopathology; and investigate whether administration of ivacaftor, the first CF gene modulator that has significantly improved the life of patients with CF; is beneficial in improving inflammation. a) cystic fibrosis and treatment options for vulnerable sub populations b) investigational compounds for bronchiectasis c) finding biomarkers for ME/CFS.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Dr Elena Schneider-Futschik

Project co-supervisors:

Dr Chris Armstrong, Dr Natalie Thomas, Dr Kathy Huang

Project: ME/CFS & Long COVID Longitudinal Omics & Women's Health

Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) and Long COVID are debilitating, multisystem conditions with a poorly understood pathophysiology. Female sex is one of the most consistent risk factors, yet sex-specific mechanisms remain largely underexplored. A key challenge in ME/CFS and Long COVID research is biomarker heterogeneity, compounded by study designs that fail to account for sex differences, reproductive lifespan, circadian rhythms, and menstrual cycle fluctuations. Sex hormones broadly influence immune, endocrine, nervous, and metabolic function, likely driving the observed sex differences in symptom prevalence, severity, and disease progression. However, such endocrine variability is rarely considered, reducing biomarker signal detection and reproducibility. The MELLOW study (ME/CFS + Long COVID Longitudinal Omics and Women's Health) addresses these challenges through a comprehensive, dense-sampling study-design in women, precisely mapping menstrual cycle dynamics and sex hormone fluctuations alongside multi-omic data integration.

Project availability:

PhD
Honours

Project supervisor:

Dr Natalie Thomas

Project co-supervisors:

Dr Elena Schneider-Futschik

Stewart Group

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Key focus areas

- Cell Signaling
- Lung Health
- Mechano-pharmacology
- Pharmacology
- Respiratory diseases
- Microphysiological Systems
- Cell Biology
- Chemokine biology

https://biomedicalsciences.unimelb.edu.au/sbs-research-groups/biochemistry-and-pharmacology-research/stewart-laboratory-mechanopharmacology

Our lab seeks to investigate novel aspects of chemokine biology and how it relates to innate immunity in the lung with an aim of better understanding chronic respiratory pathologies

Project: Microphysiological Systems models of fibrosis

You will learn how to make microfluidic devices in hydrogel material for measuring force in medium throughput, perfusion based human pirmary myofibroblast cultures. The project involves fabrication of biomaterials, assembly of microfluidics perfusion imaging and pharmacological benchmarking.

Project availability:

Master of Biomedical Science
Honours

Project supervisor:

Professor Alastair Stewart

Project co-supervisors:

Dr Bryan Gao

Project: Chemokines as innate immune sensing molecules - a novel atypical function

Chemokines facilitate a range of cellular functions via the activation of G protein-coupled receptors (GPCR), a mechanism of action that is well-established and understood. In the lung chemokines are central mediators of the innate immune response and help to protect against external pathogens by recruiting and activating immune cells. We have identified an atypical function of chemokines that allow them to act independently of GPCRs and rather as direct modulators of pattern recognition receptor signalling. This is exemplified by our work on the novel chemokine CXCL17. We hypothesize that direct modulation of pattern recognition receptor signalling is a key function of CXCL17 that helps protect the lung from infection. In this project, you will dissect the interplay between CXCL17 expression and modulation of pathogen-associated signalling using model immune cells lines as well as cells from the airways. Methods to be used will include immunoassay, real-time quantitative PCR, cell culture, flow cytometry, CRISPR/Cas9 genome editing, molecular biology and fluorescent confocal microscopy.

Project availability:

Master of Biomedical Science
Honours

Project supervisor:

Dr Carl White

Project co-supervisors:

Professor Alastair Stewart

Stojanovski Group

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Key focus areas

- Cell Biology
- Intracellular transport
- Mitochondrial disease
- Molecular mechanisms of disease
- Organelles
- Pathogens
- Biochemistry
- Infection and Immunity

THE STOJANOVSKI GROUP is working to understand the inner workings of one of the cells most intriguing and important organelles, the Mitochondrion. Mitochondria are the cell's power plant, where sugars from the food we eat are converted into energy that our bodies need to survive. Our lab is interested in how mitochondria are created and how they function in health, but also situations of dysfunction that lead to disease.

Project: The machines and pathways that facilitate mitochondrial protein import in human cells.

"Inside each mitochondrion is a workforce of approximately 1500 proteins, which collaborate to perform the many critical functions of the organelle. These proteins are trafficked from their site of synthesis in the cytosol to their correct location within mitochondria, a process referred to as 'protein import'. If mitochondrial protein import fails, energy production and general mitochondrial health and function is affected. Therefore, not surprisingly dysfunctional protein import is linked to mitochondrial disease and other human pathologies. In spite of this, our understanding of mitochondrial protein import and import machineries in human cells remains poor. Our lab is interested in elucidating the molecular architecture of human import machines and elucidate how they govern protein import in mammalian cells. For instance, we recently identified novel metazoan specific subunits, Tim29 and AGK (Kang et al., 2016; Kang et al., 2017), which are components of the inner membrane translocation machine, TIM22 complex. Current efforts in the lab are focused on: (i) characterizing known mitochondrial import machines across human cell types; (ii) identifying novel facilitators of these pathways; and (iii) investigating how protein import dysfunction leads to mitochondrial disease. Projects in this space utilise a variety of cell and molecular biology techniques, including mammalian cell culture, CRISPR/Cas9 gene editing techniques, molecular biology, confocal microscopy, Blue-native PAGE electrophoresis, protein chemistry and proteomics.

Project availability:

PhD

Project supervisor:

A/Prof. Diana Stojanovski

Project: Mitochondria as targets during bacterial pathogenesis.

"Invasion of a human cell by pathogenic bacteria often requires that the bacteria manipulate the eukaryotic cell biology to create a replicative niche and evade killing. To achieve this, intracellular bacterial pathogens transport virulence proteins, termed effectors, into the host cytosol and beyond to other cellular compartments. Effector proteins from different bacteria and viruses are known to target mitochondria and try and manipulate organelle function. We are currently investigating the intracellular bacterial pathogens Coxiella burnetii and Legionella pneumophila, the causative agents of Q-fever and Legionnaire's disease, respectively. In collaboration with Dr Hayley Newton we are: (i) working towards establishing the entire repertoire of effector proteins from these two bacterial species that are targeted to mitochondria; (ii) establishing how infection manipulates the mitochondrial proteome and function. Participation in this project will involve characterizing of novel effector proteins, how they are delivered to the mitochondrion and how they interact with mitochondrial proteins to allow these intracellular bacterial pathogens to replicate within a eukaryotic cell. Projects in this space utilise a range of cell and molecular biology techniques, including mammalian cell culture, bacterial culturing, molecular biology, confocal microscopy, Blue-native PAGE electrophoresis, protein chemistry and proteomics."

Project availability:

Honours
Master of Biomedical Science
PhD

Project supervisor:

A/Prof. Diana Stojanovski

Project co-supervisors:

Prof. Hayley Newton

Stroud Group

Contact: A/Professor David Stroud
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Key focus areas

- Functional genomics
- Inborn errors of metabolism
- Biochemistry
- Genetics
- Mitochondrial disease
- Translational and Clinical Research
- CRISPR-Cas9

Research in the Stroud laboratory aims to raise the diagnostic rate for monogenic rare diseases, including mitochondrial disease, understand rare disease pathology, and ultimately improve human health through the application new multi-omic based technologies and bespoke cell-based disease models to functional genomics.

Project: New functional genomics technologies in diagnosis and study of rare genetic disorders

Rare diseases are defined as a disease affecting less than 5 in 10,000 people. While rare diseases are individually rare, there are more than 7,000 conditions with most being pediatric disorders and having devastating outcomes. Rare diseases are typically monogenic where mutations in a single gene explains patient pathology. An accurate molecular diagnosis is critical as it can guide treatment and enable access to family planning approaches such as pre-implantation genetic testing. The front line approach for diagnosis of suspected rare disease is whole genomic sequencing, however less than half of patients will not receive a definitive answer. The most common reason for this is lack of biochemical evidence supporting pathogenicity of novel mutations in known and novel genes. Our lab has developed a quantitative proteomics-based approach to rapidly generate functional data linking novel variants to pathogenicity (see: Hock et al., 2025 Genome Medicine; Lunke et al., 2023 Nature Medicine; Amarasekera et al., 2023 Human Molecular Genetics for examples). Through our Australian Government funded initiative RDMassSpec, we are actively recruiting undiagnosed patients with suspected mitochondrial and non-mitochondrial rare genetic disease (see: https://go.unimelb.edu.au/obi8) and in parallel developing the technique for routine clinical use through automation of sample preparation, benchmarking of utility against groups of patients with a known diagnosis, and demonstrating utility in diagnosis of non-mitochondrial disorders. We have multiple Honours and Master projects available and would be happy to discuss a project within this research theme that aligns with your specific interests and career goals. When undertaking a project in this theme you will become skilled in cell culture, proteomics and metabolomics sample prep and data analysis, application of various functional assays such as Seahorse XF oxygen consumption rate measurements, and potentially the use of CRISPR/Cas9 and molecular biology approaches to generate and analyse disease models. You will also become familiar with the analysis of human genomic data and curation, and will work alongside clinicians, medical scientists, genetic counsellors and clinical genetics experts.

Project availability:

Master of Biomedical Science
Honours

Project supervisor:

A/Prof David Stroud

Project co-supervisors:

Dr. Daniella Hock

Project: Forward CRISPR/Cas9 genetic screen to identify novel mitochondrial protein function

Is has been estimated that, even at rest, our bodies turn over ~70kg of ATP each day. More than 90% of this is generated through mitochondrial oxidative phosphorylation (OXPHOS), which occurs on the five membrane protein complexes comprising the respiratory chain. Mitochondria are comprised of ~1500 different proteins. Over 80 of these are subunits of the OXPHOS complexes and >50 others known as assembly factors are needed for their biogenesis and regulation. Our lab recently identified a protein factor that accumulates in mitochondria when the OXPHOS system is damaged, either through mutations in genes affecting OXPHOS function, hypoxia, or inhibition of OXPHOS through treatment with small molecule inhibitors. In this project you will harness this knowledge to perform a forward CRISPR/Cas9 genetic screen with the goal of identifying new proteins involved in maintenance of mitochondrial OXPHOS function. Throughout your Honours or Masters research project you will become skilled in cell culture, molecular biology and biochemistry techniques such as cloning, lentivirus transduction, imaging approaches such as confocal fluorescence microscopy, fluorescence activated cell sorting, and bioinformatic approaches related to genetic screening.

Project availability:

Master of Biomedical Science
Honours

Project supervisor:

A/Prof David Stroud

Project co-supervisors:

Dr. Nikeisha Caruana

Villadangos Group

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Key focus areas

- Cell Biology
- Cell Signaling
- Immunology & Immunity
- Proteomics
- Metabolism

Our laboratory studies the first event that triggers adaptive immune responses: the presentation of pathogen or tumor antigens to T cells by Dendritic Cells, B cells and Macrophages. We are characterizing the development, regulation and impairment of antigen presenting cells by pathogens, inflammatory mediators and tumours. We are also dissecting the biochemical machinery involved in antigen capture, processing and presentation. We use this knowledge to understand how T cell-dependent immunity is initiated and maintained, and apply it to design better vaccines and immunotherapies against infectious agents and cancer.

Project: Control of dendritic cell tissue niche adaption by O-GlcNAcylation

This project will delineate how the addition of a small sugar to cellular proteins, known as O-GlcNAcylation, links nutrient-sensing and cell development. We have developed one few models where this important process can be studied in living animals. This project will determine how the loss of O-GlcNAcylation in dendritic cells (an important immune cell type), impacts cell development in specific tissues. The outcomes include breakthrough new knowledge, publications and commercial opportunities.

Project availability:

PhD
Master of Biomedical Science
Honours

Project supervisor:

Dr Adam Balic

Project co-supervisors:

Prof. Jose Villadangos

Index

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MELBOURNE

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

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