Te Tari Mātai Whaiaroaro

Department of Physiology

Taumata-400 2025

400-level 2025

Ngā whakamārama | Information

Te tukanga tono | Application process

Please read this document carefully because it contains important information about the 400-level programme in Physiology, including entry requirement and how to arrange a PHSL/GENE/NEUR PGDipSc or BSc (Hons), or BBiomedSc (Hons) project with a supervisor in the Department of Physiology.

Te tukanga tono | Application process

If you are interested in a 400-level qualification in the Department of Physiology, as well as obtaining the necessary grades to be eligible (see "400-level Degree Options and Entry Requirements" below), you will need to secure a supervisor for your research project. The process for arranging a PHSL/NEUR PGDipSc or BSc (Hons) project, or a BBiomedSc (Hons) project in the Department of Physiology is below.

How to apply for a research project

1. Read the project descriptions (Appendix I).
2. E-mail the supervisors offering the projects in which you are interested to arrange a meeting to discuss the projects in-person or by Zoom.
3. Decide the projects for which you would like to be considered (up to three, in rank order) and the qualification for which you are applying.
4. Complete the online application form by the **8th November 2024**:
5. Late applications will be considered if there are projects available.

What happens next?

1. Your research project application and academic record will be given to the academic with whom you are interested in working, who will decide on whether to accept your application.
2. You will be informed of your application outcome by email in **early December**.
3. If your research project application is successful:
4. For **PGDipSci** or **BSc (Hons)**, complete your formal application for entry on eVision by **10th December 2024**. Once we (or GENE/NEUR) confirm admission with the Division of Sciences Administration, you will be notified of acceptance on eVision.
5. For **BBiomedSc (Hons)**, entry is subject to approval of the Pro-Vice-Chancellor (Health Sciences) on the advice of the Board of Studies for Biomedical Sciences. Acceptance into the programme is organised by the BBiomedSc Administration but is dependent on securing a research project.
6. If you are eligible but not matched with any of your choices, you will be provided an opportunity to discuss alternative projects with other supervisors if they still have projects available.

Ngā karahipi | Scholarships

The University offers various scholarships for 400-level students. In addition, Māori and Pacific Peoples students can apply for a School of Biomedical Sciences Scholarship for 400-level ($7,500 tuition fee waiver). The Department of Physiology also offers one stipend of $5,000 to a Māori or Pacific Peoples student undertaking a 400-level project in the Department, preferably in a neurophysiology topic.

Application instructions:

All scholarships can be found here.

Māori Biomedical Sciences Scholarships can be found here.

Pacific Peoples Biomedical Sciences Scholarships can be found here.

For the Māori or Pacific Peoples Physiology stipend, please forward a copy of a School of Biomedical Sciences application directly to: physiology.postgrad.admin@otago.ac.nz.

Questions? Contact:

- Prof Colin Brown (colin.brown@otago.ac.nz), kaituitui kaupapa taumata-400-level convener, for questions about the 400-level programme.
- the relevant supervisor for questions about the projects.
- physiology.postgrad.admin@otago.ac.nz for questions about your Research Project application.

Ngā Herenga Tohu Paetahi | Degree Requirements

PGDipSci

Prerequisites: BSc (B average recommended in four of PHSL 341-345 or equivalents).

Programme: PHSL 471 and 472 (20 pts each), PHSL474 (20 pts) and PHSL490 (60 pts).

BSc (Hons)

Prerequisites: At least five 300-level papers including at least four of PHSL341-345 (B+ average recommended in the four PHSL 300 papers).

Programme: PHSL 471 and 472 (20 pts each), PHSL474 (20 pts) and PHSL490 (60 pts).

BBiomedSc (Hons) in Functional Human Biology

Prerequisites: A BBiomedSc degree with an average grade of at least B+ for the four prescribed 300 papers, must have passed a fifth 300-level paper in their third year of study (for a total of 90 points at 300-level), and should normally have passed papers worth at least 108 points at 200-level or above in their third year of study.

Programme: A 120-point programme, comprising a research thesis and course work.

See: https://www.otago.ac.nz/courses/qualifications/bbiomedschons.html.

N.B. Entry into the two-year MSc programme is organized through a different process; please contact the Physiology Postgraduate Coordinator, Assoc Prof Martin Fronius (martin.fronius@otago.ac.nz) for information on the process.

Pārongo Hōtaka | Programme information

PHSL 471 Systematic Physiology and PHSL 472 Neurophysiology

These 20-point papers each consist of seminars on research frontiers in physiology. Each paper requires preparation and participation (e.g. discussion, presentation, etc.), and is assessed by written online examination.

PHSL 474: Research Topics

This 20-point paper is a self-directed literature survey of physiology topics that complement, but are distinct from, the research project. It is specifically designed for each student, guided by the supervisor and is internally assessed by three essays.

PHSL 490: Research Dissertation

This is a 60-point laboratory project involving original research and is assessed by a dissertation in the form of a thesis. All steps of the project are guided by the supervisor. PHSL 490 also includes oral presentations to the Department in April and September/October. Dissertation submission is in late October.

Appendix I: Research Projects

Our research falls into the following main areas:

- **Cardiovascular Physiology:** Jeff Erickson, Martin Fronius, Pete Jones, Rajesh Katare, Megan Leask, Michelle Munro, and Daryl Schwenke.
- **Neurophysiology:** Colin Brown, Rosie Brown, Rebecca Campbell, Karl Iremonger, Joon Kim, and Alex Tups.
- **Membrane & Ion Transport:** Tanya Cully, Fiona McDonald.

Cardiovascular Physiology Projects

Associate Professor Jeff Erickson & Dr Luke Worthington

(jeff.erickson@otago.ac.nz)

The role of CaMKII in diabetes-induced heart failure

CaMKII activation is a primary pathological event in heart failure and arrhythmia, particularly for patients with diabetes mellitus. Thus, CaMKII has emerged as a potential therapeutic target in the treatment of heart disease. With this in mind, our research focuses on investigating the role of CaMKII in the diabetic heart. Contributions by a motivated 400 level student would be possible for a project examining cardiac function in diabetic animal models using protein blotting, histochemistry, and cell imaging techniques.

Associate Professor Martin Fronius (martin.fronius@otago.ac.nz)

Investigating the effect of vapes on lung epithelia

The project aims to investigate if and how vapes interfere with ion transport in human lung epithelial cells. Gas exchange and the innate immune system in our lungs rely on transepithelial ion transport processes, facilitated by the epithelia covering the surfaces of the lungs. Cigarette smoke has been identified to interfere with ion transport processes, which contributes to lung pathologies including chronic bronchitis and chronic obstructive pulmonary disease (COPD). Although the number of cigarette smokers decreased over time, vaping is becoming more and more popular. Vaping is perceived as a 'healthy' alternative to smoking. However, there are growing reports of lung damage associated with vaping

termed e-cigarette or vaping product use–associated lung injury (EVALI). Interestingly, the damage observed is different in comparison to cigarette smoke.

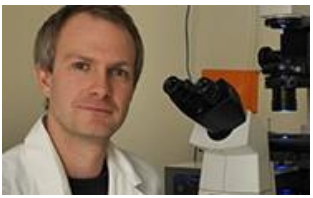
The project will expose human lung epithelial cells (H441 cells) to vapes and measure how this affects ion transport processes by performing Ussing chamber electrophysiology. The study will identify a putative mechanism by which vaping interferes with normal epithelial cell function that is crucial for normal breathing.

Characterising TRPV channel function underpinning hypothalamic vasopressin release

Vasopressin release from osmosensitive neurones in the hypothalamus is essential for maintaining plasma osmolality homeostasis and blood pressure regulation. Elevated vasopressin release is associated with hypertension indicating impaired function of these osmosensitive neurones. Osmosensitivity in these neurones relies on the function of Transient Receptor Potential Vanilloid (TRPV) ion channels. Activation of these channels in response to increased plasma osmolality activates the channels to depolarise the cell membrane potential. This increases action potential frequency and drives the exocytosis of vasopressin. The Fronius and C. Brown laboratories are interested in understanding the processes of how TRPV channels sense cell shrink and cell swell due to changes of plasma osmolality. Further, if changed TRPV channel function is a contributor to elevated vasopressin release in hypertension. The project provides the opportunity to study TRPV channel function by patch clamp electrophysiology and characterise how cell swell and shrink (mimicked by the application of positive and negative pressure) affects channel activity. Main goal of the study is to understand if and how TRPV channels contribute to hypertension.

Professor Pete Jones (pete.jones@otago.ac.nz)

Role of RyR2 mediated calcium release in arrhythmia and Alzheimer's disease.



We are seeking students to join our research group for various projects that relate to the following research theme: Calcium release is critical for contraction in the heart and synaptic transmission in the brain. In both tissue types, the ryanodine receptor (RyR2) mediates a large part of this release. When the carefully controlled release of calcium through RyR2 goes wrong it leads to disease. Our lab aims to understand the molecular mechanisms which lead to abnormal RyR2 function.

Several projects are available looking at the function and structure/ location of RyR2

within cardiac cells and neurons with the purpose of better understanding arrhythmias and Alzheimer's disease.

Associate Professor Rajesh Katare (rajesh.katare@otago.ac.nz)

Molecular mechanisms underlying healthy ageing

Ageing is an inevitable process accompanied by a gradual decline in physiological functioning. Advances in healthcare and improvements in diet have significantly increased life expectancy. However, this increased longevity has also led to a higher incidence of chronic diseases. The Honours project will explore the molecular mechanisms associated with cardiovascular disease. The project provides an opportunity to learn several molecular techniques, such as PCR, protein quantification, immunohistochemistry, and ELISA, and how to apply contemporary statistical analyses to larger clinical datasets.

Dr Megan Leask (Kāi Tahu) (megan.leask@otago.ac.nz) & Dr Oluwatobi Eboda

Establishing a zebrafish model for the study of genetic predisposition to polycystic ovary syndrome

Polycystic ovary syndrome (PCOS) is common in Aotearoa and is the leading cause of infertility in women worldwide. Despite its prevalence, little is known about the underlying biological mechanisms of PCOS, and current treatment modalities are limited. In PCOS there are alterations in the hypothalamic-pituitary-gonadal axis hormone release in the brain affecting ovarian function. Previous studies have shown there is a substantial genetic component in PCOS development. Currently, mammalian mice and rat models are used to study PCOS circuitry however, these models present a difficult and expensive challenge when it comes to studying how genetics may contribute to the disruption in the feedback between the ovary and the brain. We will establish an alternative zebrafish model of PCOS at the University of Otago that is far less expensive and amenable to genetic study. Contributions from a motivated 400 level student will be to establish a model of PCOS in zebrafish and perform subsequent ELISAs on harvested muscle tissue, glucose tolerance tests and confocal imaging of the brain and ovary.

Dr Michelle Munro (michelle.munro@otago.ac.nz)

Calcium handling proteins and cardiac ultrastructure

Contraction of the heart relies on the tightly regulated movement of calcium within the myocytes. Calcium mishandling occurs in cardiac diseases, which can lead to impaired contractility and the development of irregular contractions (arrhythmias). We are seeking students to join our group to work on projects exploring the organisation, expression and regulation of key calcium handling proteins in cardiac diseases including atrial fibrillation, diabetes and heart failure. A range of techniques are used in our lab including calcium imaging, immunolabelling, fluorescent imaging, super-resolution microscopy and western blotting, with projects using cell models or patient samples.

Sex differences in the diabetic heart

Diabetic patients have an increased risk of developing cardiovascular complications, including heart failure. While there is a higher prevalence of diabetes in men, diabetic women have a higher risk of developing these complications. The cause for this disparity is unclear, however one potential mechanism is a difference in fibrotic remodelling between female and male diabetic hearts. This project investigates the pattern of cardiac fibrosis and response to anti-fibrotic treatment in male and female diabetic mice. Techniques in this project include echocardiography, immunofluorescence and confocal imaging.

Neurophysiology Projects

Professor Colin Brown (colin.brown@otago.ac.nz)

Regulation of blood pressure by vasopressin neurons

The project will determine how the brain controls blood pressure. Long-term regulation of blood pressure depends on body fluid balance. Body fluid balance is principally controlled by the hormone, vasopressin, which inhibits urination. A genetic variant has been identified that is associated with low blood pressure in humans. A new genetically modified rat strain that expresses the variant urinates excessively and has low blood pressure. Hence, decreased vasopressin secretion might increase urination to cause low blood pressure in rats that carry the variant. Vasopressin secretion is driven by action potential firing in vasopressin neurons. Therefore, this project will use electrophysiology to determine whether vasopressin neurons of variant rats have a lower action potential firing rate than those of non-variant rats.

Dr Rosie Brown (rosemary.brown@otago.ac.nz)

Investigating the role of neurochemicals in maternal mood regulation

Maternal mood disorders affect 1 in 5 new mothers in Aotearoa New Zealand, and yet how the brain controls maternal mood is poorly understood. Neurochemicals including dopamine and serotonin are known to be key regulators of mood and behaviour in mothers. We have recently found that interactions with offspring, drives release of these neurochemicals in a region of the mother's brain that is important for pleasure and reward. This project aims to investigate how neurochemical release is disrupted in preclinical mouse models of maternal mood disorders, and whether stimulating neurochemical release can rescue maternal mood and behaviour.

Interrogating a neural circuit regulating parental care-giving behaviour

Neurons expressing hormone receptors in the brain are essential for parental care-giving behaviour and survival of newborn offspring. This project will test how specific neural circuits govern discrete aspects of parental care-giving behaviour, and whether stimulation of circuits can drive these behaviours in animals that would normally ignore newborn young.

Professor Rebecca Campbell (rebecca.campbell@otago.ac.nz)

Using pre-clinical models to understand the PCOS brain

Research in our lab is aimed at understanding the brain circuits that regulate fertility and the central defects that contribute to infertility. We are particularly focused on understanding how brain wiring and communication is altered in the common endocrine disorder Polycystic Ovary Syndrome (PCOS). For the appropriate student, a 400-level project will be developed to better understand the central defects that may underpin the neuroendocrine pathology of PCOS in a pre-clinical model of the syndrome. The project will likely involve working with transgenic mouse models, immunohistochemistry, light and confocal microscopy, and the application of imaging software and analysis.

Associate Professor Karl Iremonger (karl.iremonger@otago.ac.nz)

Studying brain circuits controlling stress responses

Our research group focuses on understanding how the brain connects internal states and behaviour. We are particularly interested in the role of brain stress circuits on generating anxiety, depressive, and reward-seeking behaviours. In our laboratory, honours projects start at the behavioural level, using animal models to explore these behavioural changes. We will then manipulate the activity of stress neurons in the brain to gain insights into their role in controlling the behaviour of interest.

To develop our complex behavioural models, we custom-build various tools, such as operant conditioning devices. In addition to learning animal behaviour research techniques, you will gain entry-level experience in neural engineering and coding.

Projects available in 2025:

* Inhibition of stress neurons to reduce anxiety
* Protective role of social connectedness on mental health
* Stress and reward seeking behaviours

Dr Joon Kim (joon.kim@otago.ac.nz)

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Associate Professor Alex Tups (alexander.tups@otago.ac.nz)

Neuroendocrine control of obesity and diabetes

If you want to find out why jetlag causes obesity or why Alzheimer's disease is called type 3 diabetes join the Tups lab for pursuing your BSc Honours studies. Projects are available to study the neuroendocrine control of obesity and glucose homeostasis or their interaction with the circadian clock as well and brain function. You can choose to either work with zebrafish or mouse models. We will focus on mechanistic work to combine genetic, pharmacological, or nutritional approaches to find novel treatments for metabolic health.

Membrane & Ion Transport Projects

Dr Tanya Cully (tanya.cully@otago.ac.nz)

Calcium and ROS signalling in skeletal muscle

The Cully research lab has honours projects focusing on the interplay between calcium and reactive oxygen signalling in healthy and diseased skeletal muscle. Calcium is known to have a critical role in the degenerative phase of many muscular diseases. A leaky calcium channel in skeletal muscle can be a result of the influence of reactive oxygen species (ROS) which can promote damage and inflammation in the muscle. Projects will focus on rodent models and utilise several techniques, such as biochemical measurements and microscopy. We will seek to understand the role of two ROS producing isoforms and their influence on calcium handling in different skeletal muscle fibre types, to gain a greater understanding of these processes as well as potential drug targets.

Professor Fiona McDonald (fiona.mcdonald@otago.ac.nz)

Epithelial sodium channel (ENaC) and its effects on breast cancer progression

Breast cancer is the most common cancer affecting New Zealand women and 90% of breast cancer deaths occur due to metastasis. A process called epithelial-mesenchymal transition (EMT), whereby cells change their structure and shape, and begin to proliferate and migrate, contributes to breast cancer progression and metastasis. During EMT cancer cells lose their epithelial phenotype, including cell-cell and cell-basement membrane connections, and gain mesenchymal characteristics, such as increased proliferation and migration. Our data has highlighted a role for the epithelial sodium channel (ENaC) in EMT, with a loss in ENaC expression potentially driving EMT. Projects will investigate the effect of ENaC on cancer hallmarks, signalling pathways, or Ca^{2+} levels in breast cancer cells, using techniques such as cell migration, cell proliferation, invasion, cell-extracellular matrix interactions, western blot and Ca^{2+} imaging.