
Auckland Medical
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ANNUAL REPORT
2018
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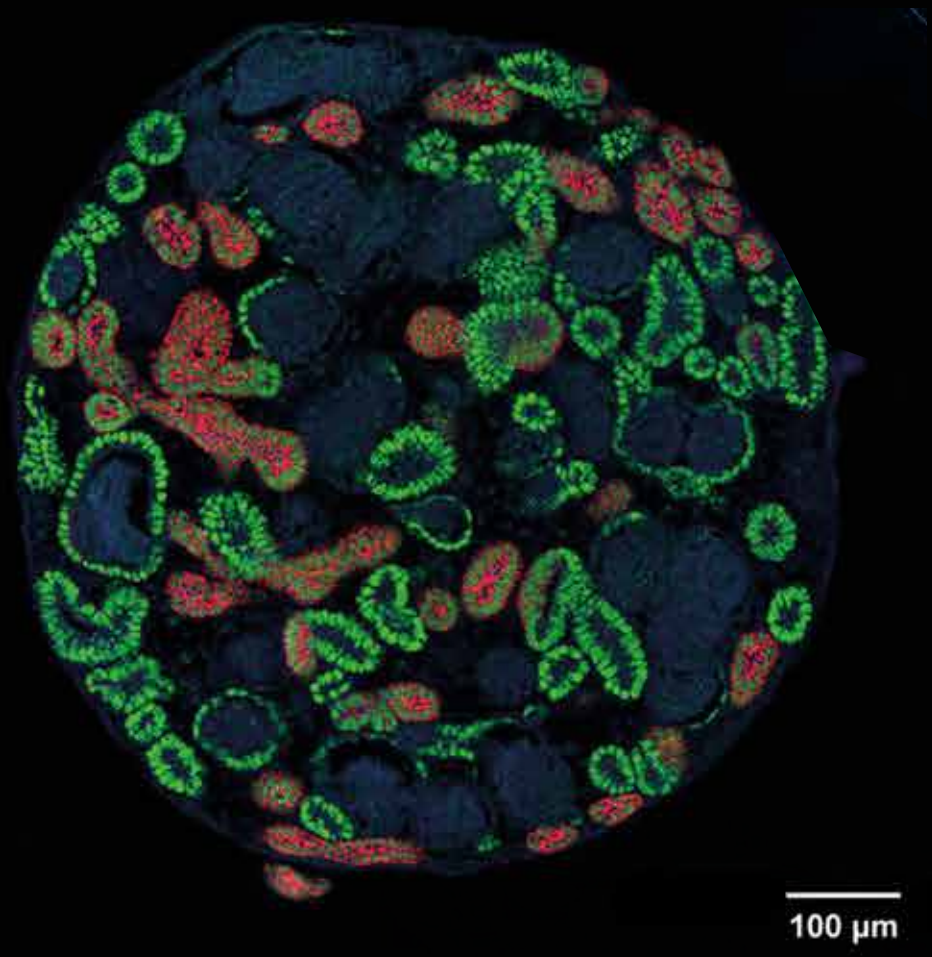
FOR OVER
60 years



MODELLING HUMAN KIDNEYS IN A DISH

Research fellow Dr Veronika Sander and the team lead by A/Prof Alan Davidson from the Department of Molecular Medicine and Pathology at The University of Auckland have developed a method to grow thousands of small kidney organoids (3D mini kidneys grown in a culture dish) from human induced pluripotent stem cells (iPSC) at low-cost and high efficiency. The organoids develop up to approximately 1 mm in diameter and are composed of renal tissues that closely resemble those of the actual human kidney. The new method enables the researchers to generate kidney tissue in the quantities needed to develop new drug treatments for renal disorders such as acute kidney injury and fibrosis. Shown in the photo here and on the inside of the back cover are cross sections of a kidney organoid with renal tissues stained in green, red and light blue. See inside back cover for additional image.

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President's Report & Medical Committee Report

YEAR ENDED 31 DECEMBER 2018

Richard Taylor

President's Report

The 2018 year was another successful year for the AMRF, amidst a year of change. In June, after 16 years on the Board and eight of those as President, Jeff Todd retired having made an outstanding contribution to the AMRF during his tenure. I felt humbled and privileged to take up the Presidency mantle from Jeff and have enjoyed walking in the footsteps of the exceptional leaders that have gone before.

The financial performance this year was strong with $4.37 million being awarded for research projects, fellowships, scholarships, travelling fellowships and travel grants. This represented a $350,000 increase year-on-year and the approved funding was awarded for a diverse range of research, spanning development of needle-free vaccinations through to identification of more effective treatments for aggressive brain tumours.

The success of the AMRF can be attributed to many. However, the ongoing funding of world-class medical research would not be possible without the generosity of our donors and sponsors. On page 38 of this Report, we acknowledge you, our members and donors and we cannot thank you enough for your support. Alongside our long-term and very loyal supporters, a number of new donors have helped to make this year such a strong one.

We are pleased to welcome the Goodwin Charitable Trust as a new partner with their commitment to support the Helen Goodwin Doctoral Scholarship. You can read about the 2018 recipient of their generosity, Ms Zoe Woolf, whose work is featured on pages 3 & 14. Grants from the Estate of Ernest Hyam Davis & the Ted & Mollie Carr Endowment Trust enabled Dr Peter Freestone to continue with his vital research into Parkinson's disease through the awarding of a three year Davis & Carr Senior Research Fellowship.

As always, bequests play a significant role in the AMRF's ability to fund major research projects and we are very grateful to those who have left, or are considering leaving, a legacy to us. It is heartening to know that people understand the critical role medical research plays in the continuous improvement of health and quality of life for generations past, present and future.

I extend my sincere gratitude to our Executive Director, Sue Brewster, and her hard working team at the AMRF for their expertise and passion that continues to drive the engine room of our organisation.

To the AMRF trustees, committee chairs and committee members, thank you for your generous donation of time and expert knowledge and the ongoing commitment you have to the achievement of our AMRF mission.

It would be remiss of me not to acknowledge and thank the Goodfellow family and their associated charitable trusts which fund AMRF's operational expenses. This means every dollar donated by you, our generous supporters, goes directly into funding medical research and to making an immense difference in the lives of so many.

Richard Taylor
President

Prof Peter Browett

Medical Committee Report

Once again the Medical Committee have been busy this year, assessing 180 grant applications over the course of the year split between five grant rounds. In a tough funding environment, we awarded 67 grants at a total cost of $4.37 million – a success rate of 37.2% - which although it may seem high, still means that many worthy applications are unable to be supported.

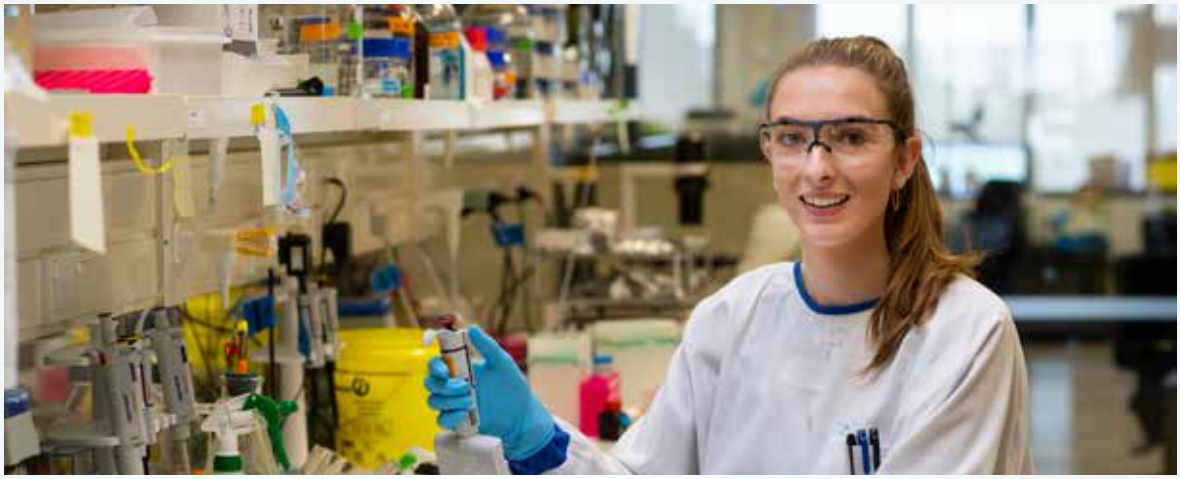
Within our funded grants, the AMRF has continued to support researchers and clinicians in all stages of their careers. A particular highlight from this year was the awarding of the inaugural AMRF Senior Research Fellowship to Dr David Musson, in partnership with The University of Auckland's Faculty of Medical and Health Sciences. Another highlight was the awarding of a total of seven scholarships and fellowships to support researchers in the early stages of their career. In addition to personal awards, we have funded 20 projects that span the full breadth of what is considered to come under our remit of medical and/or health research.

The awarding of these grants would not be possible without the generous gifting of time and expertise of our Medical Committee members who ensure a contestable and robust assessment process is followed for all of the applications. In 2018, we welcomed Dr Jane Alsweiler from the Department of Paediatrics: Child & Youth Health, The University of Auckland and Neonatologist, Auckland District Health Board. Her clinical expertise will add great value to the already diverse experience of our Medical Committee. I also want to extend my gratitude to Dr Justin Dean, Associate Professor Alan Davidson, and Associate Professor Greg O'Grady for their input into the Committee over several years. I could not write this report without expressing my sadness at the passing away of Associate Professor Nigel Birch after short battle with cancer. We enjoyed his company and appreciated his involvement the AMRF Medical Committee meetings – both academically and the dry follow up comments he provided, always with a wry smile on his face. He will be missed.

On behalf of the Medical Committee, I would like to thank the AMRF team, under the expert guidance of Sue Brewster for the administration and background work that enables us to distribute more funding to the research community each year where possible. In particular, my sincere thanks go to Dr Hannah Gibbons (Research Programme Manager) for her stewardship of the Grants Portfolio and management of the Medical Committee. I would also like to thank our Board of Trustees and in particular our new President, Richard Taylor, for their hard work and belief that funding the highest quality medical research will improve the health and quality of life of all New Zealanders.

Peter Browett
Chair, Medical Committee
Professor of Pathology, Department of Molecular Medicine and Pathology, The University of Auckland; and Haematologist, Auckland District Health Board

INAUGURAL RECIPIENT OF THE HELEN GOODWIN DOCTORAL SCHOLARSHIP



Recent BSc graduate, Ms Zoe Woolf, was inspired by a personal connection to study and research glioblastoma multiforme (GBM), the most common and aggressive brain tumour in adults.

She says, "GBM carries a dismal mean survival period of only 15 months. There have been numerous advances in recent years, but aggressive cancers such as GBM remain incurable. Given the rapid progression of these tumours, there is an urgent need for the development of successful treatments."

With her prestigious AMRF Helen Goodwin Doctoral Scholarship, Zoe's PhD research will help decipher the role of immune cells, like microglia and tumour associated macrophages, in GBM tumours. These cell types, she says, are an often over-looked contributor to tumour development and metastasis.

"My Aunty was affected by this aggressive cancer when I was younger, so I really want to try and better understand the immune environment within these brain tumours, because I think this may be the best way to open up new avenues for immunotherapy and treatments to help patients."

Doctoral scholarships are vital in helping young researchers progress and prosper in their studies and the Gooduck Charitable Trust wanted to be able to make a critical difference in an emerging researcher's career pathway.

Helen Goodwin, the founder of the Gooduck Charitable Trust, has a life long interest in education and also appreciates the importance and scarcity of medical research in New Zealand. Accordingly Helen was thrilled to be able to make a grant through her Charitable Trust to the Auckland Medical Research Foundation, to enable Zoe to further her education and important research.

"I am very grateful to Helen Goodwin and the trustees of the Gooduck Charitable Trust who are willing to support us as researchers and make our work possible."

The Helen Goodwin Doctoral Scholarship is awarded at the 2018 Research Awards - from L to R Professor Peter Browett, Richard Taylor, Rowan Kingstone, Zoe Woolf

Thank you to all our supporters for joining us in the AMRF mission: to fund world class medical research to provide genuine advances in medical and health sciences

100% OF YOUR DONATION DIRECTLY SUPPORTS *Medical Research*

AMRF Success Stories

SHINING A LIGHT ON PARKINSON'S DISEASE

DR PETER FREESTONE

"I am humbled to have been awarded a Senior Research Fellowship from the Estate of Ernest Hyam Davis & The Ted and Mollie Carr Endowment Trust. This financial support will go a long way to advancing my research to develop new treatment strategies for Parkinson's disease."

Parkinson's disease (PD) is a particularly cruel disorder. It impacts your movement and mobility and even the ability to interact socially. Investigating why one section of the brain deteriorates and how to combat the condition has been a 10-year quest for **Dr Peter Freestone**.

His research aims to deepen the understanding of what changes in the Parkinson's brain and identify more effective therapies to improve the lives of those living with the disease.

Peter is pioneering the use of cutting-edge technology, called optogenetics, to improve the understanding of what happens to the brain of someone with PD. Optogenetics is a relatively new technique in neuroscience, involving the use of light to activate single neurons within a tightly interconnected network, and allows a very high level of precision when studying brain function.

"We can actually use light to activate specific cells that we choose and that means we can study these networks in much greater detail," he says. "I am not pursuing ways to prevent the disease – in the vast majority of cases we do not even know what causes it."

"I want to improve existing treatment strategies. Parkinson's represents a huge loss for families, communities and the country. Anything we can do to restore people with Parkinson's to their normal life will be an important improvement for these patients and their families."

A SENIOR RESEARCH FELLOWSHIP FOR TENDON RESEARCHER

DR DAVID MUSSON

"I've been really fortunate to have support from AMRF throughout my career. AMRF has absolutely helped me establish myself as a tendon researcher."

Tendon injuries cost the ACC more than $280 million each year. And this kind of injury cannot be repaired, only made functional, leaving people susceptible to further issues. Around 20% of us unknowingly have damaged tendons, and the impact of a tendon injury affects day-to-day tasks for life, whether you know you have it or not.

Unfortunately, it gets worse as you get older, with about 50 to 60% of people in their fifties having tears in shoulder and hip tendons, and as we age the numbers keep increasing. This is a serious concern for New Zealand's aging population, and it's surprising for so little to be known about tendons and tendon injury when they affect so many people. The number of surgeries is increasing at an unprecedented rate – New Zealand has seen a 400 percent increase over the past 10 years.

Dr David Musson says, "My research aims to heal tendons and find treatments for tendons and ligaments of older people so they heal better. If we can get this overlooked tissue to recover better after damage, to look and act more like younger tendons then we will reduce costs for the government but most importantly give people back their pain-free, functional joints."

Already, one part of David's research is paying dividends having identified key structural and bio-mechanical differences in the hamstring tendons normally used as graft material for ACL reconstruction in younger patients. The result? Clinicians now use an alternative, safer graft source which has improved the patient's likelihood of recovery.

CHANGING SLEEPING POSITION WHILE PREGNANT - A SIMPLE FIX THAT CAN SAVE LIVES

DR ALYS CLARK

"Funding from supporters The MRI ERD Trust has been critical for us to begin generating world-class quality images that will help identify risks from sleeping position to mum and baby."

Mum and baby's physiology changes when a pregnant woman sleeps in a particular position, but how and why? When mum sleeps on her side late in pregnancy the risk of stillbirth is halved compared to sleeping on her back. The reasons for this are being investigated using world-leading magnetic resonance imaging (MRI) techniques by **Dr Alys Clark** from the Auckland Bioengineering Institute, Prof Peter Stone from the Department of Obstetrics and Gynaecology at The University of Auckland, and their collaborators.

This imaging can show what actually changes when a pregnant woman moves from one posture to another by creating maps of both mum and baby's oxygen levels and blood flow within the placenta, the organ responsible for delivering baby all the nutrients it needs to thrive and grow.

"We know that lying on your back late in pregnancy compresses major blood vessels which can reduce the amount of blood flow to the uterus, but we don't know yet whether long periods lying on your back could have any effect on the amount of oxygen baby can receive from mum," Alys says. "These amazing new technologies provide the first opportunity to observe the placenta when mum moves position to begin to determine whether this is the case. Looking at the placenta in this level of detail, and late in pregnancy, is a first for New Zealand, and contributes to a major effort in New Zealand to reduce the risk of stillbirth."

NEEDLE-FREE VACCINES FOR NEW ZEALAND'S WORST COMMUNICABLE DISEASES

DR CATHERINE TSAI

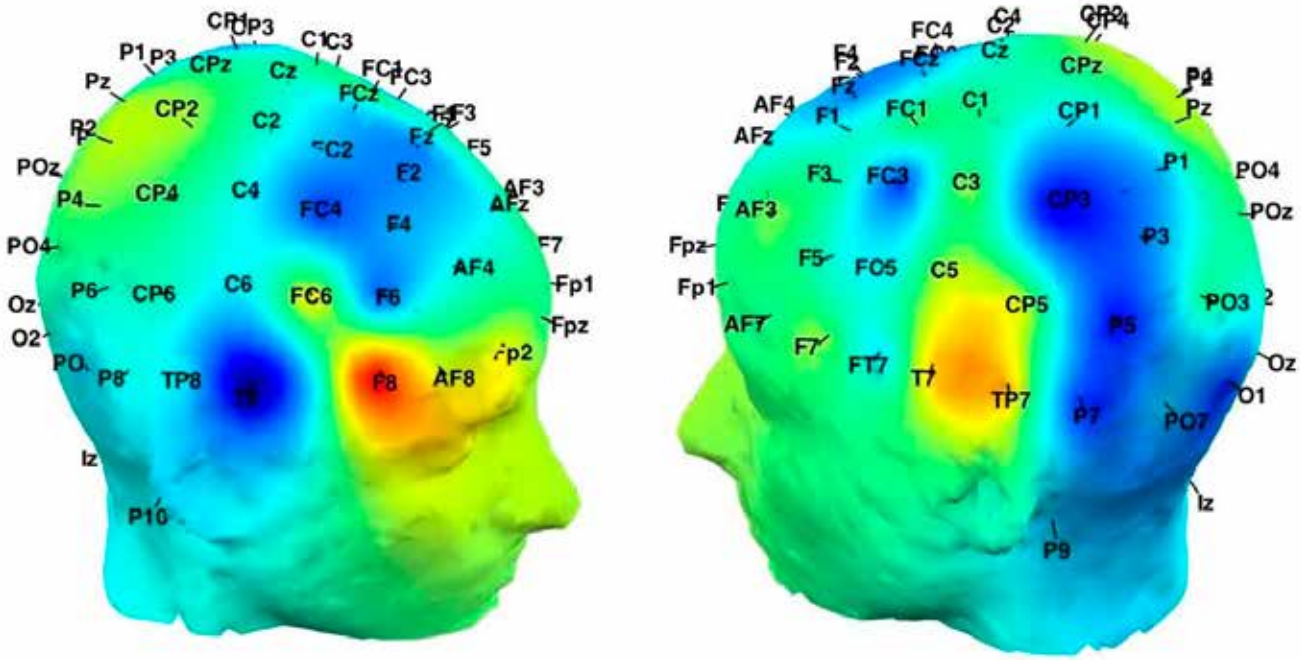
"As the mother of a young child, I have a truly personal stake in my work on effective, inexpensive, needle-free vaccines that can protect good health without causing tears and fears in the clinic."

A strategy to overcome the pitfall of synthetic peptide vaccines, namely the lack of stability and insufficient ability to elicit strong immune responses on their own, is a challenge **Dr Catherine Tsai** has undertaken in her early research career, thanks to the generosity of AMRF donors.

"Vaccines remain the most cost effective and feasible method for infectious disease control. Modern vaccines are carefully designed so are safer and more specific. But to compensate, the trade-off of improved efficacy, they sometimes require expensive processing or potentially toxic additives," she says. "I am often dismayed by how infectious diseases negatively affect people's life and well-being, and how challenging the control measures can be in high risk communities."

"In my current postdoctoral research, I want to develop a novel vaccine strategy that uses a non-pathogenic bacterium found in dairy foods. I plan to improve the stability of the vaccine and still be able to elicit a strong, beneficial immune response - and an added bonus is it will be needle-free. These lactococcus-based vaccines can be administered through the mouth or nose which is the same place that many of the pathogens we aim to target enter the body."

With the support of a prestigious **AMRF Postdoctoral Fellowship**, Catherine's work will improve the efficacy and safety of this novel vaccine technology, and could develop into useful vaccines against a range of diseases, including tuberculosis, flu, colorectal cancer and gonorrhoea.



MAPPING BRAIN WAVES
WHEN TINNITUS IS SUPPRESSED

Tinnitus is the perception of sound in the absence of an external physical sound source. Researchers lead by A/Prof Grant Searchfield in the Section of Audiology at The University of Auckland are testing the ability of EEG (electroencephalography or "brain wave" recordings) to map what happens in the brain in patients with or without tinnitus.

This tool will allow us to piece together the parts of the puzzle of tinnitus and how it can be treated with future therapies.

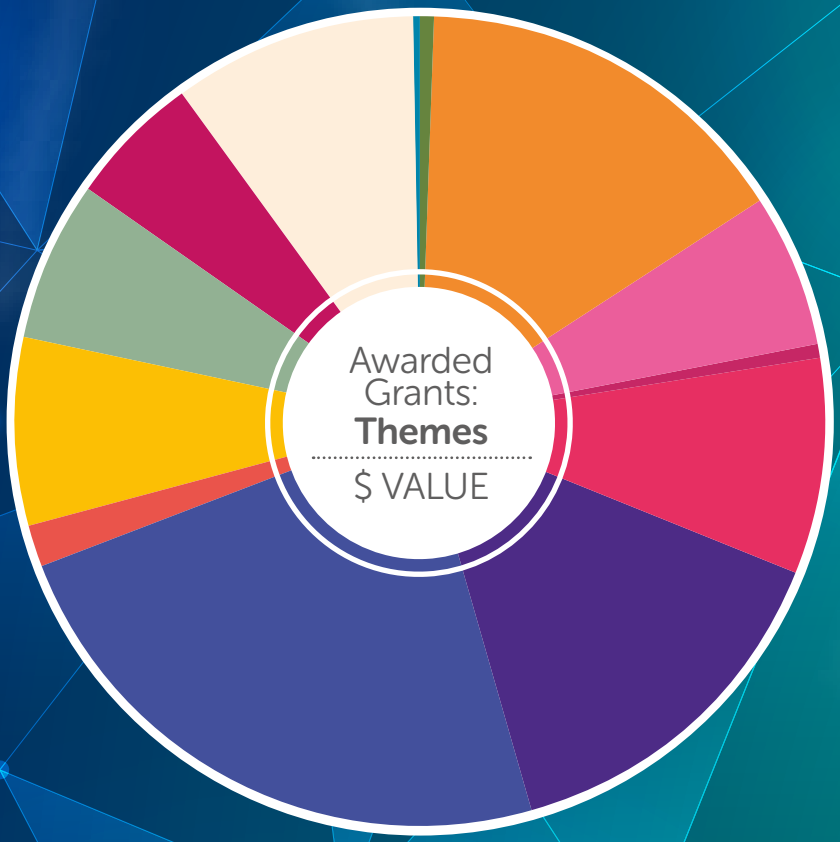
In this image of an EEG head plot, we have taken a snap shot of brain activity immediately following a simple, loud sound, which is known to temporarily reduce or suppress tinnitus.

During this suppression there is repeated activation of unexpected parts of the brain. On the left we see a 'hotspot' of activation on the frontal cortex, shown in orange and red and marked F8, while in the image on the right, the auditory cortex is also activated, also in yellow and orange and marked T7 and TP7.

This tool will allow us to match tinnitus treatments, from drugs to music therapies, that have similar and lasting effects in the brain but are less damaging than additional loud sound.

Image kindly supplied by Dr King, Dr Shekhawat, Dr Chan, Dr Kobayashi and A/Prof Searchfield.

GRANTS AWARDED



2018 AWARDED GRANTS – THEMES : 67 GRANTS AWARDED TOTALLING $4,370,740

- Biomedical Imaging (1) | **$24,600** **0.56%**
- Cancer (7) | **$677,123** **15.49%**
- Cardiovascular Science (4) | **$265,952** **6.08%**
- Cellular and Molecular Biology (3) | **$22,121** **0.51%**
- Endocrinology, Metabolism and Nutrition (2) | **$4,262** **0.10%**
- Infection and Immunity (6) | **$368,588** **8.43%**
- Musculo-skeletal Science (8) | **$633,363** **14.49%**
- Neuroscience (12) | **$1,028,184** **23.52%**
- Other (8) | **$75,500** **1.73%**
- Population Health (4) | **$324,893** **7.43%**
- Pulmonary, Renal, Nephrology and Gastrointestinal Sciences (2) | **$283,209** **6.48%**
- Reproduction, Development, Maternal and Newborn Health (4) | **$236,470** **5.41%**
- Sensory Sciences (4) | **$417,813** **9.56%**
- Stem Cell Biology (1) | **$1,847** **0.04%**
- Surgery (1) | **$6,815** **0.16%**

$ Value each theme **% Total expenditure**
(n) Number of grants

Grants Awarded

PROJECTS

NOVEL PEPTIDE ANTIBIOTICS TARGETING ANTIMICROBIAL RESISTANCE ($159,824 – 2 years) 1118013

Dr Ghader Bashiri, Dr Paul Harris, Dr Stephen Ritchie
School of Biological Sciences, The University of Auckland

Antimicrobial resistance is a pressing global health issue of the modern age. In February 2017, the World Health Organisation published a list of antibiotic-resistant "priority pathogens" that pose an imminent threat to human health. These include both methicillin- and vancomycin-resistant Staphylococcus aureus (MRSA and VRSA), which are major causes of hospital-acquired and community infections. We have recently discovered a novel antibiotic with potent activity against these antibiotic-resistant bacteria, however, the antibiotic is complicated to make synthetically and can only be extracted in minute amounts from the natural source. This multidisciplinary project will use a powerful combination of chemical peptide synthesis and recombinant enzyme engineering to yield sufficient quantities of this new antibiotic for clinical application. The expected outcomes of this research address a key National Science Challenge to reduce the burden of major health problems in New Zealand, but its impact will be far-reaching as new-generation antibiotics are urgently required worldwide.

INCIDENCE STUDY OF SUDEP IN NEW ZEALAND ($75,367 – 2 years) 2118014

Dr Peter Bergin, Prof Jonathan Skinner, Dr Yannan Jiang, Dr Claire Spooner, Dr Simon Stables, Dr Elizabeth Walker, Dr Nicholas Child, Dr Ian Rosemergy, A/Prof Roderick Duncan, Dr Melinda Nolan
Neurology, Auckland District Health Board

Sudden unexpected death in people with epilepsy (SUDEP) is an under-appreciated tragedy. It has devastating effects on families, friends and colleagues of those who die. The incidence of SUDEP is not well known, and it has not previously been studied in New Zealand; however, the life-time risk of SUDEP for someone who develops epilepsy before age one and who continues having regular seizures may be as high as 8%. We will undertake a prospective study to identify all people who die from SUDEP in New Zealand. We intend to identify patients from multiple sources: doctors, coroners, pathologists, Epilepsy New Zealand field workers, the epilepsy community generally, and the Ministry of Health national administrative datasets. All cases of possible SUDEP will be reviewed by two adult or paediatric neurologists and at least one pathologist, and cases will also be discussed at a meeting of the Cardiac Inherited Diseases Group. Patients in whom no clear cause of death is identified will have a 'molecular autopsy' in which known genes associated with sudden death are assessed. Patients who are deemed to have died from SUDEP will have information about their epilepsy and the circumstances of their deaths entered into the EpiNet database. We will calculate the incidence of SUDEP in New Zealand and determine which people with epilepsy are at greatest risk of dying. We will monitor the incidence over time to see if changes in epilepsy management result in a change in SUDEP incidence.

CO-FUNDED WITH: The Neurological Foundation of New Zealand

Neurological Foundation
A pathway to hope

IMMUNOONCOLOGY OF MERKEL CELL CARCINOMAS ($25,000 – 6 months) 1118005

Dr Cherie Blenkiron, Dr Kate Parker
Dept. of Molecular Medicine & Pathology, The University of Auckland

Merkel Cell Carcinoma (MCC) is a rare tumour that is often identified as small red, quickly growing lumps on the skin. As well as growing quickly they can also spread or metastasise to other sites in the body. MCCs are caused either by an infectious virus or more often here in New Zealand by genetic damage caused by sun exposure. These two environmental triggers themselves can be the downfall for the tumour, causing an immune response or white blood cell attack. The tumour can however overcome this attack through molecular camouflage. This is where new immunotherapies, like the melanoma treatment Keytruda can be used to reawaken the immune cells. In order to understand whether people with MCC could benefit from immunotherapy, this study will identify the types of immune cells present in these tumours and decipher their molecular camouflage signals. The project will provide new biological information about an understudied rare cancer and in the long term it could support the provision of funded immunotherapy for people with MCC.

FUNDED BY: J.I Sutherland Fund for Melanoma Research

HOUSEHOLD TRANSMISSION OF BACTERIAL RESISTANCE ($145,078 – 2 years) 1118020

A/Prof Catherine Byrnes, Dr Emma Best, Dr Rachel Webb, Dr Adrian Trenholme, Dr Susan Morpeth, Dr Catherine Bremner, Dr Hamish McCay
Dept. of Paediatrics, The University of Auckland

The increased prevalence of antibiotic-resistant bacteria in New Zealand is concerning. It is driven by high antibiotic use. Worldwide infection by untreatable bacteria is projected to cause more deaths than cancer by 2025. Recently prolonged oral azithromycin (an antibiotic and anti-inflammatory medication) resulted in reduction of infections and hospitalisations for children with the lung scarring 'bronchiectasis'. This is a group with few other available treatments leading to PHARMAC recently funding oral azithromycin for children with bronchiectasis and frequent infections for up to 12 months. As this policy is taken up, we will investigate the development of antibiotic resistance in bacteria carried by these children. However, more importantly we will also determine if the resistant bacteria are transmitted to otherwise healthy siblings in the household. Regular nose and throat swabs taken at the beginning, during and after azithromycin treatment will be cultured for bacteria and resistance patterns identified. If we show there is spread of resistant bacteria, this could significantly impact the wider community by reducing future antibiotic treatment options for others. We will also

describe any factors that could improve treatment to the individual but prevent acquisition of resistant-bacteria to other household contacts.

FUNDED BY: AC Horton Estate

INOSINE FOR BONE HEALTH ($160,000 – 2 years) 1118012

Prof Nicola Dalbeth, Dist. Prof Ian Reid
Dept. of Medicine, The University of Auckland

Recent observational studies have reported that high urate levels are protective in the development of thin bones (osteoporosis) and fractures. Inosine is a nutritional supplement that increases serum urate levels. We plan a six month randomised controlled trial of 120 postmenopausal female participants. Participants will be randomised to one of two groups: placebo or inosine tablets. We will study markers to bone health to understand whether this supplement has effects on bone. We will also study whether this supplement can be used safely in this study population, by monitoring measures of kidney function, blood pressure and other features of metabolic syndrome. If inosine does have positive effects on bone markers, this supplement may represent a new treatment strategy for prevention of osteoporosis.

WHANAU EXPERIENCE OF A HEALTHY HOMES INITIATIVE ($14,259 - 1 year) 1118001

Dr Kyle Eggleton
Dept. of General Practice & Primary Health Care, The University of Auckland

Cold damp houses are associated with poorer health outcomes. Significant evidence exists demonstrating that housing insulation may reduce exacerbations of asthma and respiratory illnesses. There are a number of healthy homes initiatives nationwide and this research intends to explore one such initiative, based in Northland, to ascertain whānau experiences of the programme. The research will determine the wider health benefits of insulating and warming damp homes and whether the implementation of the project has met whānau expectations. The intent of the research is to improve the delivery of healthy homes initiatives in order to align with whānau expectations.

CROSS TALK BETWEEN TISSUES OF THE EYE ($158,981 – 2 years) 1118009

Dr Julie Lim, Prof Paul Donaldson, A/Prof Dipika Patel, Dr Angus Grey, A/Prof Philip Polkinghorne, Dr Rasha Altaie
Dept. of Physiology, The University of Auckland

With an aging population, age related pathologies of the eye are increasingly common resulting in the need for surgery. However, clinical evidence suggests that in two of the most commonly performed eye surgeries, cataract surgery and vitrectomy, the risk of secondary eye diseases is significantly increased resulting in patients requiring treatment within two years post-surgery. To investigate the molecular mechanisms that contribute to these secondary pathologies, we propose a novel hypothesis in which the lens and vitreous humor work together to maintain high concentrations of antioxidants in the vitreous humor. These antioxidants consume oxygen to ensure low oxygen levels which is required to protect the lens from cataract and the vitreous humor from degeneration. If this hypothesis is correct, a major impact of this project is the development of new post-cataract or vitrectomy treatments to enhance antioxidant levels in the eye and improve vision outcomes for the elderly.

FUNDED BY: Anonymous donor

DOES CYREN DETERMINE RADIATION-INDUCED TUMOUR MUTATIONAL BURDEN? ($156,091 – 2 years) 1118016

Dr Barbara Lipert, Prof William Wilson, Dr Francis Hunter, Prof Cristin Print
Auckland Cancer Society Research Centre, The University of Auckland

Targeted antibodies (immunotherapy) that harness the body's natural defence mechanisms have changed the paradigm of oncology. As one example, immunotherapy with T cell checkpoint inhibitors has shown success in shrinking even advanced tumours. Yet, at present these drugs help only a minority of patients predominantly due to the ability of cancer cells to make themselves invisible to the immune system (so-called 'cold' tumours). Radiotherapy can induce DNA mutations leading to formation of altered proteins (neoantigens), which can target tumour cells for elimination. Several studies have shown that high levels of mutation increase the number of neoantigens and that combining radio- and immunotherapy improves tumour control, including for patients failing radiotherapy alone. However, there are no methods for identifying patients who may benefit from this novel approach. CYREN is a small protein that we have recently discovered to be a suppressor of radiation-induced mutations. We will test whether CYREN influences mutational burden in cancers of the head and neck, and whether its inhibition enhances the antitumour activity of radiotherapy in combination with T cell checkpoint immunotherapy. If confirmed, this would help identify patients who would benefit from this treatment and would open the way for CYREN inhibition for enhancing cancer therapy.

FUNDED BY: Anonymous donor

CGRP AND BONE HEALING ($160,000 - 2 years) 1118008

Dr Brya Matthews, Dr Dorit Naot, Dr Christopher Walker
Dept. of Molecular Medicine & Pathology, The University of Auckland

Bone fractures cause severe morbidity in both young, active people and in the elderly. Fracture healing is significantly delayed or fails to occur in approximately 10% of patients. Limited treatment options are available to treat poor fracture healing, so more insight is needed into the mechanisms involved in the healing process. Sensory nerves are abundant in bone tissue, more so following injury, however their function in the healing process is not understood. One of the neurotransmitters produced by these nerves is known as CGRP and has previously been shown to promote bone formation. We will try to understand the role of CGRP in bone healing using a mouse model that lacks CGRP, as well as performing cellular studies to understand the mechanisms by which CGRP promotes bone cell activity.

Grants Awarded continued

CHARACTERISING THE ROLE OF CARDIAC NEURONS IN HEART RHYTHM ($154,539 – 1.5 years) 1118003

A/Prof Johanna Montgomery, Prof Julian Paton, Prof Bruce Smaill, Dr Martin Stiles, Dr Kirsten Finucane, Dr Jesse Ashton

Dept. of Physiology, The University of Auckland

Abnormal heart rhythms, termed arrhythmias, are devastating disorders. The most common arrhythmia, atrial fibrillation (AF), significantly increases the risk of stroke, heart failure, and dementia. Treatment strategies are limited, and more precise therapies are essential. One treatment target is the neurons located on the heart. These neurons regulate heart rhythm, and they can trigger AF. However, how these neurons trigger AF is unknown, which limits our ability to precisely target them to increase treatment success. We have assembled a team of scientists and clinicians with expertise in neurophysiology, cardiac physiology, cardiology and cardiac surgery. This enables us to examine the properties of cardiac neurons in animal models and human tissue for the first time. We hypothesise that changes occur in these neurons, termed "plasticity", driving abnormal neuron activity in AF. We will identify the changes in cardiac neuron function during normal heart rhythm versus AF, and whether these changes can be reversed to stop AF. We will also establish protocols to record from human cardiac neurons collected during heart surgery. Our novel data sets will advance our understanding of cardiac neuron function during normal and abnormal heart rhythm, which is critical to develop more precise therapies to treat AF.

SYNBIOTICS AND LIVER TRANSPLANTATION ($123,943 – 2 years) 1118011

A/Prof Lindsay Plank, A/Prof Mike Taylor, Prof John McCall, Prof Edward Gane, Dr Adam Bartlett

Dept. of Surgery, The University of Auckland

Liver transplantation is the only effective treatment for patients with advanced liver disease. This is a major operation which is associated with a high rate of complications over the early postoperative period which prolongs stay in hospital and is a major cause of early death. These complications are predominantly bacterial infections. A combination of prebiotics and probiotics (synbiotics) provided for one day before and 14 days after transplant has been shown to almost eliminate bacterial infections. This remarkable result requires confirmation for this treatment to be accepted into routine clinical practice. We propose to conduct a similar study using the same synbiotic. If this simple and inexpensive treatment reduces infections after transplant we expect it to lead to routine use in our liver transplant unit and adoption by other centres worldwide. This result and the associated reductions in antibiotic use and length of hospital stay are significant both for patients undergoing this life-saving procedure and for the hospital, given the consequential cost savings. We will also examine potential mechanisms that might explain the purported benefits of this treatment.

NEUTIN ARI ($104,556 – 2 years) 1118018

Dr Grant Searchfield, Prof Nikola Kasabov

Section of Audiology, The University of Auckland

Tinnitus is the perception of sound in the absence of an external physical sound source. It is a highly prevalent condition affecting approximately 10% of all New Zealanders. Tinnitus disrupts: hearing, attention, sleep, and can lead to anxiety and depression. There is no cure for tinnitus. However a short loud sound can partially, and in many cases totally, suppress tinnitus for a brief time. This effect has been labelled Acoustic Residual Inhibition (ARI) and is the focus of the proposed research. This research will investigate the behavioural effects and neural correlates of ARI. It will use Electroencephalography (EEG, "brain waves") and a computer model of neurons developed in NZ called the NeuCube to understand the processes responsible for tinnitus generation and its suppression. Knowledge of the neuronal responses to tinnitus suppression will lead to the further development of novel sound therapy paradigms, and potential targets for tinnitus medicines. This innovative research builds on previous successful AMRF funded research.

BENZENESULPHONAMIDES: A PROMISING NEW CLASS OF IMMUNOSUPPRESSANTS ($158,808 – 2 years) 1118004

Dr Julie Spicer, Prof Geoff Hill, Dr Stephen Jamieson, Dr Kate Gartlan

Auckland Cancer Society Research Centre, The University of Auckland

Stem cell transplantation is used to treat cancers such as leukaemia, lymphoma and myeloma, but in many cases the patient's immune system sees the incoming cells as 'foreign' and they are rejected, often with fatal consequences. This is because without a perfectly-matched donor, the procedure relies on mis-matched grafts from stored umbilical cord blood or partly-matched grafts from relatives. The cells responsible for early rejection overwhelmingly use a protein called perforin to kill the transferred stem cells. We have developed a class of small molecules that can block this process in the critical period of four to five days after the transplant. This will allow far more stem cells to survive and migrate to the marrow where they will essentially be in a 'safe haven' and able to multiply to produce red blood cells, white blood cells and platelets. Effective perforin inhibitors would increase the number of successful stem cell transplants, improving survival from potentially fatal cancers and with applications in solid organ transplantation more generally. In this project we plan to optimise the potency, physicochemical and pharmacological characteristics of these molecules to give safe and efficacious candidates suitable for pre-clinical development.

MRI STUDY OF PLACENTAL OXYGENATION IN PREGNANCY ($24,600 – 1 year) 1118010

Prof Peter Stone, Dr Alys Clark, Dr Seyed Ali Mirjalili

Dept. of Obstetrics & Gynaecology, The University of Auckland

Late stillbirth in normally formed babies is responsible for a majority of perinatal deaths. We were the first to show that maternal sleep position may be a major modifiable risk factor for late stillbirth, but exactly how and why position impacts on fetal well-being remains to be determined. Using MRI we showed that the supine position in late pregnancy caused a 15%

reduction in cardiac output and a 36% reduction in blood flow in the lower aorta. This project aims to determine if these changes in blood flow cause reduced oxygen delivery to the baby. We have developed an international collaboration to use novel techniques to measure blood flow in the arteries supplying the womb and to measure both the maternal and fetal oxygen signals recorded by MRI. We plan to study healthy pregnancies first but in the future we would plan to apply our new knowledge to women at particular risk of stillbirth such as when the baby is not growing as expected or when the mother notices reduced movements of the baby.

FUNDED BY: MRI ERD Trust

IDENTIFYING IMPAIRED INFANT MUSCLE GROWTH ($154,878 – 2 years) 1118015

Prof Susan Stott, A/Prof Malcolm Battin, Dr Seyed Ali Mirjalili, Dr Justin Fernandez, Dr Sian Williams, Dr Geoffrey Handsfield, A/Prof Alicia Spittle

Dept. of Surgery, The University of Auckland

The first years of a child's life are fundamentally important in shaping the child's future health, growth and development. During infancy, extraordinarily rapid muscle growth occurs as both the brain and movement develops. Unfortunately not all children will develop at the same rate or to the same extent. For a baby born prematurely, we know that in many cases there may be lifelong effects on neurodevelopmental functioning, including impaired movement development and risk of cerebral palsy. Surprisingly, there is little research documenting typical muscle growth in infants, and how the muscle growth changes once infants begin to reach motor-milestones. Even less is known of muscle development in premature babies, who may be at risk of cerebral palsy, a condition in which children are known to have substantially smaller and weaker muscles already by the age of two years. Understanding typical muscle growth through this important period of development has the potential to help us identify 'at risk' infants and plan earlier intervention for those struggling to achieve these key motor-milestones.

INFLAMMATION AND COCHLEAR IMPLANTATION ($158,942 - 2 years) 1118002

Prof Peter Thorne, Dr Ravindra Telang, Dr Andrew Wise, A/Prof Srdjan Vlajkovic, A/Prof Phil Bird

Dept. of Physiology, The University of Auckland

Deafness is a leading cause of disability worldwide and affects over 18% of New Zealanders. It occurs predominately from injury or disease of the cochlea of the inner ear and the primary treatment is with hearing aids or cochlear implants. Cochlear implants, normally for people with total deafness, are now available for people with some residual hearing but in many cases the hearing can deteriorate following implantation, possibly due to inflammation from surgery. This research will investigate a novel approach to reduce the inflammation by applying an activator (or agonist) of the adenosine receptors in the cochlea that have been shown to inhibit inflammation in other tissues. We will test this in an animal model by applying the drugs loaded into nanoparticles, which are inserted surgically along with the implant. Our preliminary findings show that these drugs can reduce the post-implant deterioration in hearing and in this study, we will confirm if this is due to inhibition of inflammation. If these studies are successful, it should be easily translatable to treating patients during implant surgery as the compounds are already approved for use in humans. Protecting the ear during surgery is important to maximise the outcome of the cochlear implant.

FUNDED BY: W & WAR Fraser

MEDICATION USE IN BREAST CANCER PATIENTS ($159,253 – 2 years) 1118017

Dr Sandar Tin Tin, Prof Diana Sarfati, Prof Ross Lawrenson, Prof Mark Elwood, A/Prof Ian Campbell, Prof Bruce Arroll, A/Prof Vernon Harvey

Section of Epidemiology & Biostatistics, The University of Auckland

Cancer patients are commonly burdened with comorbidities and often use multiple medications. This may have an impact on cancer treatments and outcomes. This research aims to investigate the use of prescription medications for non-cancer related indications in women with primary invasive breast cancer. The data consolidated from four regional breast cancer registers will be used, which covers about 63% of all breast cancer registrations in New Zealand. Patient data will be linked to pharmaceutical, hospital discharge and mortality data. The findings will provide insight into medication use and related consequences in breast cancer patients in New Zealand, and will inform policy, practice and efforts to improve cancer care and outcomes and reduce inequities.

THE ROLE OF EPAC IN DIABETIC HEART DISEASE ($155,688 - 2 years) 1118006

Dr Marie-Louise Ward, Prof Peter Ruygrok, Mr Nicholas Kang, Dr Sarbjot Kaur

Dept. of Physiology, The University of Auckland

Type 2 diabetes (T2D) is one of the fastest growing health issues in New Zealand, and is closely linked with the development of cardiovascular disease. Decades of research using animal models of diabetes suggest that the development of diabetic heart disease is progressive, beginning soon after the onset of diabetes, and resulting in changes to the heart cells that impact upon their function. Recently, exchange proteins activated by cAMP (known as "Epac") have been shown to induce alterations in intracellular calcium regulation when activated in many cell types, including heart muscle. Cyclical changes in intracellular calcium control contraction and relaxation of heart muscle, which enables it to function as a pump. Experimental activation of Epac in isolated heart cells upsets calcium cycling. This results in less calcium available for excitation-contraction coupling, and an increased susceptibility to developing arrhythmias. Incomplete relaxation between beats can also occur if calcium within the muscle cells remains high. We will investigate Epac in tiny human atrial tissue samples obtained from consenting patients undergoing routine surgery. We hypothesise that altered Epac activation and location in T2D promotes abnormal muscle cell calcium handling. We will test this by measuring the relative abundance and distribution of

the Epac isoforms, as well as their function and ultrastructural organisation in atrial tissue samples from non-diabetic and T2D patients. This study will provide new knowledge of human T2D and the cellular changes that are detrimental to the heart during diabetes.

NOVEL TREATMENT FOR ACUTE PANCREATITIS (159,266 - 2 years) 1118007

Prof John Windsor, Dr Jiwon Hong
Dept. of Surgery, The University of Auckland

Acute pancreatitis is a common and potentially fatal disease for which there is no specific drug treatment. Our studies have provided a new treatment paradigm that is based on the concept that the gut becomes leaky during acute pancreatitis and that toxic factors are taken up by gut lymph and drained into the main blood circulation. These toxic factors cause severe inflammation and injury to vital organs such as the heart, lungs and kidneys, which in turn results in a worse clinical outcome. This project is the first to test two types of drugs which specifically target key factors in the gut-lymph responsible for it's toxicity in acute pancreatitis. The first study is designed to show that these two drugs are taken up preferentially by gut-lymph and as a result reduces the toxicity of the gut-lymph when tested on cultured cells. The second study is designed to show that one of the drugs is effecting in reducing inflammation and organ injury. These critical proof-of-principle studies will directly contribute to further drug development and to the design of larger clinical studies which are necessary to translate this new treatment strategy for acute pancreatitis.

EXOSOME-LIPOSOME HYBRIDS FOR TUMOUR TARGETED DRUG DELIVERY ($160,000 – 2 years) 1118019

A/Prof Zimei Wu, Dr Euphemia Leung, Prof Larry Chamley
School of Pharmacy, The University of Auckland

Nano-sized liposomes are perceived as 'magic bullets' for tumour-targeting and have been successful in clinical translation. However, challenges remain including their poor tumour tissue penetration and slow release of drugs once in cells. Recently, exosomes (30-100 nm), vesicles secreted from cells, have emerged as attractive drug carriers. As natural transporters, exosomes play a major role in cell-to-cell communication, and have the ability to travel to remote locations and penetrate tissues. In this proposal, we aim to engineer a liposome-exosome hybrid, combining the beneficial features of exosomes and liposomes for targeted intracellular drug delivery to cancer cells, while sparing healthy cells. Macrophage-derived exosomes will be utilised to engineer hybrids by fusion with pH-sensitive liposomes (PSL). Unlike conventional liposomes such as Doxil® (liposomal doxorubicin), PSL rapidly release their payload into cells. This will be paired with a tumour-targeting ligand to promote specific uptake of the hybrids by cancer cells. We will then test the doxorubicin-loaded hybrids for augmented cell- and tumour-tissue penetration capabilities. Utilising the applicants' research strengths in different fields, this cutting-edge collaborative research proposal has the potential to revolutionise strategies for tumour-targeted drug delivery.

FUNDED BY: Anonymous donor

JEAN CATHIE FUND FOR TINNITUS RESEARCH

SOMATSENSORY STIMULATION TO TREAT TINNITUS ($199,987 – 2 years) 7415002

Dr Yiwen Zheng
Pharmacology and Toxicology, University of Otago

Chronic tinnitus is a debilitating condition affecting approximately 10% of the population and for which there are limited treatment options. It has long been known that the perception of auditory signals can be modulated by the stimulation of the somatosensory system and effective modulation requires precise pairing of both the somatosensory and auditory stimulations. The proposed project will test the efficacy of a paired stimulation protocol on tinnitus perception in an animal model. Specifically, we will induce tinnitus in rats using the acoustic trauma method and confirm the animal's perception of tinnitus using a well-established behavioural paradigm. We will then electrically stimulate the somatosensory system and pair it with either a tinnitus sound or non-tinnitus sound with a specified order and timing. We will compare tinnitus perception in rats before and after the paired stimulation, to determine the efficacy of the stimulation. We will also compare the tonotopic map between tinnitus rats with and without paired stimulation by recording neuronal responses to tones of different frequencies and intensities. The results will provide important preclinical information on the efficacy of a new stimulation protocol for tinnitus and may lay the foundation for the development of a more effective treatment for tinnitus.

FUNDED BY: Jean Cathie Research Fund

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SENIOR RESEARCH FELLOWSHIPS

AMRF SENIOR RESEARCH FELLOWSHIP

IDENTIFYING THE FACTORS THAT CONTRIBUTE TO THE POOR HEALING RESPONSE OF TENDONS ($525,276 – 5 years)

Dr David Musson

Dept. of Medicine, The University of Auckland

Tendon damage (tendinopathy) is a severe clinical problem, costing the NZ healthcare system in excess of $280M a year, which significantly affects the quality of life of both young and aged patients, limiting movement and resulting in significant time off work. Once damaged, tendons never recover their original structural and functional integrity; instead, disorganised scar tissue forms, diminishing both biomechanics and function. There are currently no successful therapies for treating tendinopathies, suggesting there is a clear unmet clinical need for new strategies that will improve the poor healing potential of tendons.

ADMINISTERED BY: Faculty of Medical and Health Sciences, The University of Auckland

THE DAVIS & CARR SENIOR RESEARCH FELLOWSHIP

PARKINSON'S DISEASE RESEARCH PROGRAMME: TO DISCOVER POTENTIAL NEW THERAPIES FOR PARKINSON'S DISEASE ($375,000 – 3 years) 1718008

Dr Peter Freestone

Dept. of Physiology, The University of Auckland

Parkinson's disease is a debilitating brain disorder that arises from loss of the chemical dopamine produced by nerve cells in the part of the brain called the basal ganglia. The resulting change in brain activity ultimately leads to the loss of normal movement, which is the main feature of the disease. A key element of the basal ganglia is the subthalamic nucleus. Previous discoveries by my group have identified this nucleus to be involved in regulating dopamine release; a mechanism that involves cannabis-like substances called endocannabinoids. My research will apply state-of-the-art techniques to investigate the role the subthalamic nucleus and endocannabinoids on brain function, in normal animals and animal models of Parkinson's disease. Optogenetics is one such technique, using light to precisely control the activity of individual brain cells, allowing us to study brain function in new and exciting ways. I will also test new implantable optogenetic devices that could one day be used to treat Parkinson's disease and other brain disorders, replacing existing treatments based on electrical brain stimulation. This research will deepen our understanding of how the basal ganglia works, and explore new potential therapies for Parkinson's disease that could one day improve the lives of the ~2% of New Zealanders over the age of 65 that suffer from the disease.

FUNDED BY: Estate of Ernest Hyam Davis & The Ted and Mollie Carr Endowment Trust

POSTDOCTORAL FELLOWSHIPS

DAVID AND CASSIE ANDERSON RESEARCH FELLOWSHIP

ULTRASOUND IN ASYMPTOMATIC HYPERURICEMIA ($201,363 – 2 years) 1318001

Dr Sarah Stewart

Dept. of Medicine, The University of Auckland

High levels of urate in the blood (hyperuricemia) can lead to the deposition of urate crystals in musculoskeletal structures and acute arthritis, known as symptomatic gout. However, many individuals with hyperuricemia remain asymptomatic; a condition present in 20% of adults. Despite the absence of symptoms, ultrasound imaging has demonstrated crystals and inflammation are still present. The pathological mechanisms involved in the transition from hyperuricemia to crystal presence, nor the long-term effects of crystal deposition in the development of gout have been investigated. This two-year project aims to explore the role of sonographically-evident crystal deposition in the transition from asymptomatic hyperuricemia to symptomatic gout as well as determine the clinical factors which predict underlying crystal deposition. An optimal ultrasound protocol and score for screening people with hyperuricemia at risk of gout will be developed which will facilitate early management strategies directed towards preventing the transition to symptomatic disease. This research has particular relevance to New Zealand, which has one of the highest global prevalence rates of gout, particularly in Māori and Pacific populations. This research has the potential to reduce the New Zealand and global prevalence of gout, improve health outcomes and reduce the healthcare economic burden associated with gout.

FUNDED BY: David and Cassie Anderson Medical Trust

A NOVEL PEPTIDE DELIVERY PLATFORM FOR THE DEVELOPMENT OF MUCOSAL VACCINES ($198,264 – 2 years) 1318002

Dr Catherine Tsai

Dept. of Molecular Medicine & Pathology, The University of Auckland

Vaccines remain the most cost-effective and feasible means of infectious disease control in the community. Well-defined synthetic vaccines based on individual peptides are specific and safe. However, peptide antigens are usually poorly immunogenic and sensitive to proteolytic degradation, thus require costly conjugation to carrier proteins and administration with potentially toxic adjuvants. Lactic acid bacteria have become promising mucosal vaccine vehicles. We have developed PilVax, a novel peptide delivery platform that utilises the group A streptococcus (GAS) pilus structure (hair-like protrusions) to carry a stabilised and highly amplified peptide on the surface of the food-grade bacterium Lactococcus lactis. The PilVax system provides

the possibility to produce effective, safe and inexpensive vaccines. To demonstrate the versatility of PilVax, we initiated a pilot study to construct a vaccine against the important infectious disease tuberculosis (TB) caused by Mycobacterium tuberculosis (Mtb). Preliminary results showed that intranasal immunisation of PilVax generated strong mucosal and systemic immune responses in mice. The aim of this project is to further investigate and improve the safety and efficacy profile of TB PilVax, in order to facilitate clinical applications of this novel vaccine technology.

RUTH SPENCER MEDICAL RESEARCH FELLOWSHIP

OPTIMISING THE CARE OF WOMEN AT HIGH RISK OF SPONTANEOUS PRETERM BIRTH ($66,061 – 1 year) 1418001

Dr Lisa Dawes

Dept. of Obstetrics & Gynaecology, The University of Auckland

Preterm birth, the birth of a baby before 37 weeks, is the leading cause of death in newborn babies worldwide and may impact the lifelong health of surviving babies. Despite considerable research, there is no effective way to stop preterm labour once it starts and so the focus of current practice is on prevention and optimising outcomes for babies that are born preterm. The Ruth Spencer Research Fellowship will allow completion of a series of projects which aim to improve the care provided to women who are at high risk of having their baby born early. Specialised preterm birth clinics provide a package of care for these women. We will analyse the existing international literature on preterm birth clinics and will review the treatments and outcomes from five years of practice in the only specialised preterm birth clinic in New Zealand. We will also explore the psychological wellbeing of women who are cared for in a preterm birth clinic. We expect that the new knowledge obtained from these studies will support the establishment of more preterm birth clinics throughout New Zealand to provide evidence-based care and appropriate support for all women at high risk of having a preterm birth.

FUNDED BY: Ruth Spencer Estate

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DOCTORAL SCHOLARSHIPS

HELEN GOODWIN DOCTORAL SCHOLARSHIP

DECIPHERING THE ROLE OF TWO MACROPHAGE-LIKE POPULATIONS IN HIGH GRADE GLIOMA (128,000 – 3 years) 1218002

Zoe Woolf

Dept. of Pharmacology & Clinical Pharmacology, The University of Auckland

Glioblastoma multiforme (GBM) is the most common and aggressive brain tumour in adults, and carries a dismal mean survival period of only 15 months. The presence of an immunosuppressed microenvironment is a hallmark of GBM, and this environment is in part due to the presence of dysfunctional immune cells. Microglia from the brain and tumour associated macrophages from the body are two cell types which are seen to have altered function within these tumours. Despite these being two different immune cell populations, due to their overlapping functions, they are often grouped as a single population. Using patient-derived tumour specimens and tumour cells, we have developed methods of identifying these populations for downstream experiments. Our goal is to investigate the respective roles of these cells in GBM and find more targeted therapies.

FUNDED BY: The Gooduck Charitable Trust

KERATOCONUS IN DOWN SYNDROME (97,890 – 2 years, 4 months) 1218001

Joyce Mathan

Dept. of Ophthalmology, The University of Auckland

Keratoconus is a potentially blinding disease of the cornea, the front window of the eye. In keratoconus, the cornea becomes thinner, weaker and protrudes which results in significant vision loss. International studies show that keratoconus may be several hundred times more common in Down Syndrome. Individuals with Down Syndrome experience a number of challenges and may be unaware of decreasing vision. Therefore, timely and appropriate treatment is delayed. In advanced keratoconus, corneal transplantation from a human donor is required. This may be avoided by a less invasive surgical procedure known as corneal collagen cross-linking, which has been shown to slow or halt the progression of disease. This treatment is more effective when it is provided in the early stages of keratoconus. Visual impairment affects many aspects of life such as learning, mobility, independence and autonomy. To reduce the burden of vision related impairment, we aim to introduce a screening and treatment initiative in New Zealand, tailored to the unique needs of this population. By preserving vision, we may preserve the unique potential and abilities of those with Down Syndrome.

FUNDED BY: Stilson Endowment Trust

THE ROLE OF HYALUONAN IN HIPPOCAMPAL NEURON DEVELOPMENT (87,000 – 2 years) 1218004

Molly Abraham

Dept. of Physiology, The University of Auckland

The growth and connectivity of brain cells (neurons) is critical for normal brain development and function. Alterations to the normal development of these cells can disrupt their ability to form connections and create neural networks. Deficits in neuronal connectivity are observed in a range of neurodevelopmental disorders including autism, attention deficit hyperactivity disorder, and can induce impairments in learning and memory. However, there is limited progress in the treatment of such disorders, as the mechanisms underlying neuronal developmental and connectivity in the normal brain remain unclear. Hyaluronan is a sugar molecule expressed throughout the body and brain, which has been shown to support non-neural cell development. Evidence suggests that this sugar is expressed in the developing brain, however its specific role in brain cell development is unknown. Thus, this research will provide a novel insight into the role of hyaluronan in normal brain function, and whether disruption of hyaluronan and the extracellular matrix contributes to various neurodevelopmental disorders. Further, this study will provide information on whether targeting hyaluronan disruption is a potential therapeutic strategy to promote normal brain function.

FUNDED BY: John Alfred Jarrett Trust

PROSTATE CANCER SCREENING IN NEW ZEALAND: TRENDS AND THE DEVELOPMENT OF CANCER RISK CALCULATOR (128,000 – 3 years)

1218005

Dr Bashar Matti

Dept. of Surgery, The University of Auckland

Prostate cancer is the most diagnosed cancer in New Zealand. It has significant attributed morbidity and mortality. Prostate Specific Antigen (PSA) is the main test currently used for prostate cancer screening. Despite being available for more than two decades, New Zealand data on PSA testing are scarce. Furthermore, the New Zealand guidelines on the utilisation of PSA for cancer screening purposes are not currently supported by validated international clinical studies. This project will investigate the patterns of PSA testing in New Zealand. This includes establishing an ethnicity specific, age-based reference values for PSA results. Also, it will explore the ethnic and socioeconomic disparities in cancer screening. Moreover, we will be creating a New Zealand specific prostate cancer risk calculator. This will be invaluable for patients (and their health providers) to better understand their risk of harbouring cancer. This project is the first of its kind in Australasia and will positively impact the quality of care that New Zealand men receive.

GAVIN AND ANN KELLAWAY MEDICAL RESEARCH FELLOWSHIP

$37,556 1518002

Professor Edwin Mitchell

Dept. of Paediatrics: Child and Youth Health, The University of Auckland

Application of new "Big Data" analytic techniques to identify new risk factors and insights for Sudden Unexpected Death in Infancy (SUDI).

$18,300 1518002

Professor Debbie Hay

School of Biological Sciences, The University of Auckland

Collaborative visit to the Danish Headache Center.

$60,000 2518003

Dr Chang-Ho Yoon

Auckland District Health Board

To participate in the Accelerated Master's Program in Biomedical Informatics, Harvard University, USA, August 2019 - July 2020.

SIR HARCOURT CAUGHEY AWARD

$14,127 1718001

Professor Cynthia Farquhar

Dept. of Obstetrics & Gynaecology, The University of Auckland

Core Outcome Measures for Infertility Treatments COMMIT Project.

$16,121 1718005

Dr Haruna Suzuki-Kerr

Dept. of Physiology, The University of Auckland

Research Project: P2X receptors and hemichannels as targets to prevent diabetic cataract.

SIR DOUGLAS ROBB MEMORIAL FUND

$1,338 1718002

Professor Cynthia Farquhar

Dept. of Obstetrics & Gynaecology, The University of Auckland

Verbal histories: Early Medical Women in New Zealand.

$6,815 1718006

Dr Celia Keane

Dept. of Surgery, The University of Auckland

To send two patient representatives to the 2018 International Consensus Definition of Low Anterior Resection Syndrome (LARS) meeting.

KELLIHER CHARITABLE TRUST EMERGING RESEARCHER START-UP AWARDS

$30,000 1718004

Dr Brigid Ryan

Department of Anatomy & Medical Imaging, The University of Auckland

Research support for her Edith C Coan Postdoctoral Fellowship "Early changes in frontotemporal dementia".

$15,000 1718007

Tamsin Robb

Department of Molecular Medicine & Pathology, The University of Auckland

Research support for her Doctoral Scholarship "Tumour heterogeneity: a patient-specific multi-layered investigation".

ALL KELLIHER AWARDS **FUNDED BY:**
Kelliher Charitable Trust

Kelliher Charitable Trust

SUMMIT POSTDOCTORAL RESEARCH PRESENTATION AWARD

$3,000 Travel Award 6718001

Dr Marie-Claire Smith

Dept. of Medicine, The University of Auckland

To attend a conference to present her research in the field of stroke outcome prediction.

Grants Awarded continued

HEALTHEX EMERGING RESEARCHER AWARDS

$3,000 Travel Award 6718002

Miss Farha Ramzan

Liggins Institute, The University of Auckland

To attend a conference to present her research on the comprehensive profiling of the circulatory miRNAome response to a high protein diet in elderly men.

FUNDED BY: Wellington Sisters Charitable Trust

$2,000 Travel Award 6718003

Mr Sam Blanchett

Dept. of Molecular Medicine & Pathology, The University of Auckland

To attend a conference to present his research on PilVax: a novel peptide carrier for the development of vaccines against tuberculosis.

FUNDED BY: Wellington Sisters Charitable Trust

$2,000 Travel Award 6718004

Mr Luis Knight

Dept. of Physiology, The University of Auckland

To attend a conference to present his research on the Role of the Cystine/ Glutamate Antiporter in Glutamate Metabolism in the Mouse Retina.

TRAVEL GRANTS

Dr Debbie Bean

Dept. of Psychological Medicine, The University of Auckland

To attend the 17th World Congress on Pain & present at the pre-conference Satellite Symposium on Complex Regional Pain Syndrome & Acute Pain, Boston, USA, 11 - 16 September 2018.

Dr Kristi Biswas

Dept. of Surgery, The University of Auckland

To visit a laboratory at Technische University Dresden, Germany and to attend the International Society of Microbial Ecology (ISME) conference, Leipzig, Germany, 12 - 21 August 2018.

Dr Melissa Cadelis

School of Chemical Science, The University of Auckland

To attend the American Society of Pharmacognosy 2018 Annual Meeting, Kentucky, USA, 21 - 27 July 2018.

Dr Peter Choi

Auckland Cancer Society Research Centre, The University of Auckland

To attend the 121st KCS General Meeting & Exposition, Jeju, South Korea, 18 - 20 April 2018.

Dr David Crossman

Dept. of Physiology, The University of Auckland

To attend the Europhysiology 2018 Conference, Oslo, Norway, and to visit collaborators at the University of Exeter, United Kingdom, 7 - 23 September 2018.

Dr Joanne Davidson

Dept. of Physiology, The University of Auckland

To attend the Fetal Neonatal Physiological Society annual congress, Maastricht, The Netherlands, 24 - 27 June 2018.

Dr Victor Dieriks

Dept. of Anatomy & Medical Imaging and Centre for Brain Research, The University of Auckland

To attend the Neurodegenerative Diseases: Biology & Therapeutics Conference, 28 November - 1 December 2018, Cold Spring Harbor, USA, and undertake laboratory visits, NIH Bethesda, USA.

Dr Desney Greybe

Auckland Bioengineering Institute, The University of Auckland

To attend the 8th World Congress of Biomechanics, Dublin, Ireland, 8 - 12 July 2018.

A/Prof Sarah Hetrick

Dept. of Psychological Medicine, The University of Auckland

To attend the Asia Pacific Regional Meeting of the International Association of Suicide Prevention, Bay of Islands, New Zealand, 2 - 5 May 2018.

Dr Maximilian Joret

Dept. of Neurosurgery, Auckland District Health Board

To attend the 13th Meeting of the European Association of Neuro-Oncology (EANO), Stockholm, Sweden, 10 - 14 October 2018.

Dr Maria Kleinstaeuber

Dept. of Psychological Medicine, The University of Auckland

To attend the 15th International Conference of Behavioural Medicine (ICBM) and pre-conference meetings of the International Society of Behavioural Medicine (ISBM), Santiago, Chile, 12 - 17 November 2018.

Dr Bobbi Laing

School of Nursing, The University of Auckland

To attend the 2018 ISONG World Congress, and invited panel member, Orlando USA, 26 - 28 October 2018.

Dr Christopher Lear
Dept. of Physiology, The University of Auckland

To attend the Fetal Neonatal Physiological Society annual congress, Maastricht, The Netherlands, 24 - 27 June 2018.

Dr Sophia Leung
Dept. of Medicine, The University of Auckland

To attend the 8th World Congress of Biomechanics, Dublin, Ireland, 8 - 12 July 2018.

Dr Mataroria Lyndon
Centre for Medical and Health Sciences Education, The University of Auckland

To undertake research titled 'Measure the Learning Outcomes of Healthcare Hackathons', and present data, Bejing, China, 30 November - 2 December 2018.

Dr Jordan McIntyre
Dept. of Paediatrics: Child and Youth Health, The University of Auckland

To attend the Australasian Sleep Association ASM, "Sleep Down Under" conference, Brisbane, Australia, 17 - 20 October 2018.

Dr David Musson
Dept. of Medicine, The University of Auckland

To attend the 8th World Congress of Biomechanics, Dublin, Ireland, 8 - 12 July 2018.

Dr Simon O'Carroll
Dept. of Anatomy & Medical Imaging, The University of Auckland

To attend the Society for Neuroscience Meeting, San Diego, USA, 3 - 7 November 2018, and to discuss a collaboration with Professor Elizabeth Bradbury.

Dr Sarah Primhak
Dept. of Paediatrics: Child and Youth Health, The University of Auckland

To attend ID (Infectious Diseases) week, San Francisco, USA, 3 - 6 October 2018.

Dr Veronika Sander
Dept. of Molecular Medicine & Pathology, The University of Auckland

To attend the International Workshop of Developmental Nephrology, Ein Gedi, Israel, 22 - 26 April 2018.

Dr Anna Serlachius
Dept. of Psychological Medicine, The University of Auckland

To attend the Society of Behavioural Medicine annual conference, New Orleans, USA, 10 - 16 April 2018.

Dr Dharshini Sreenivasan
Auckland Bioengineering Institute, The University of Auckland

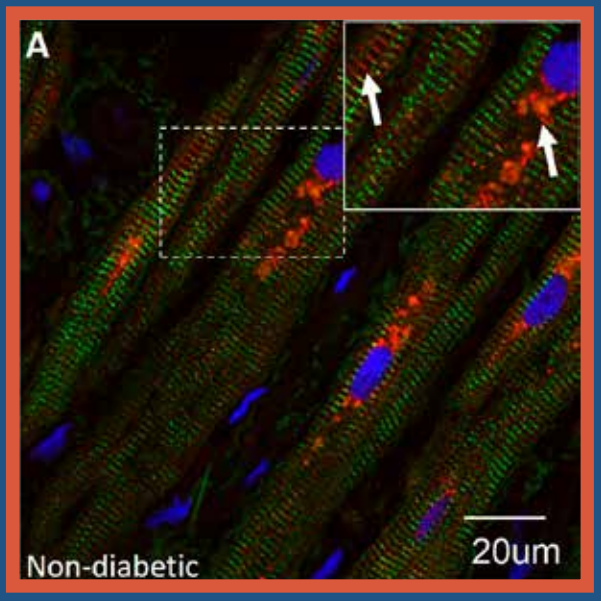
To attend the 8th World Congress of Biomechanics, Dublin, Ireland, 8 - 12 July 2018.

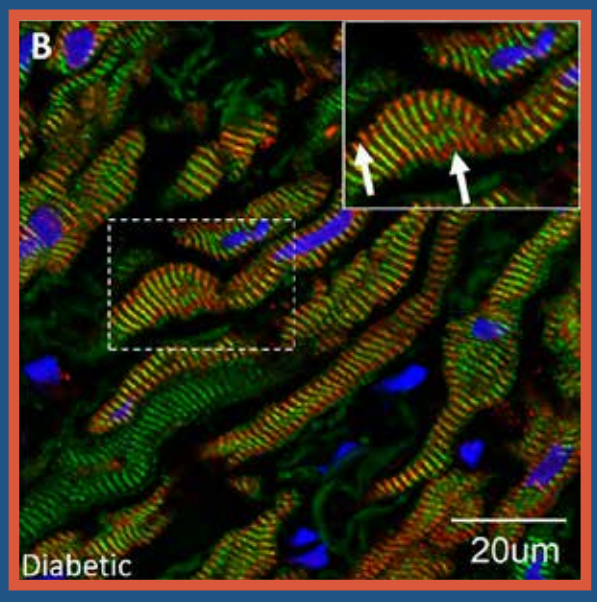
Dr Sachin Thakur
Dept. of Ophthalmology, The University of Auckland

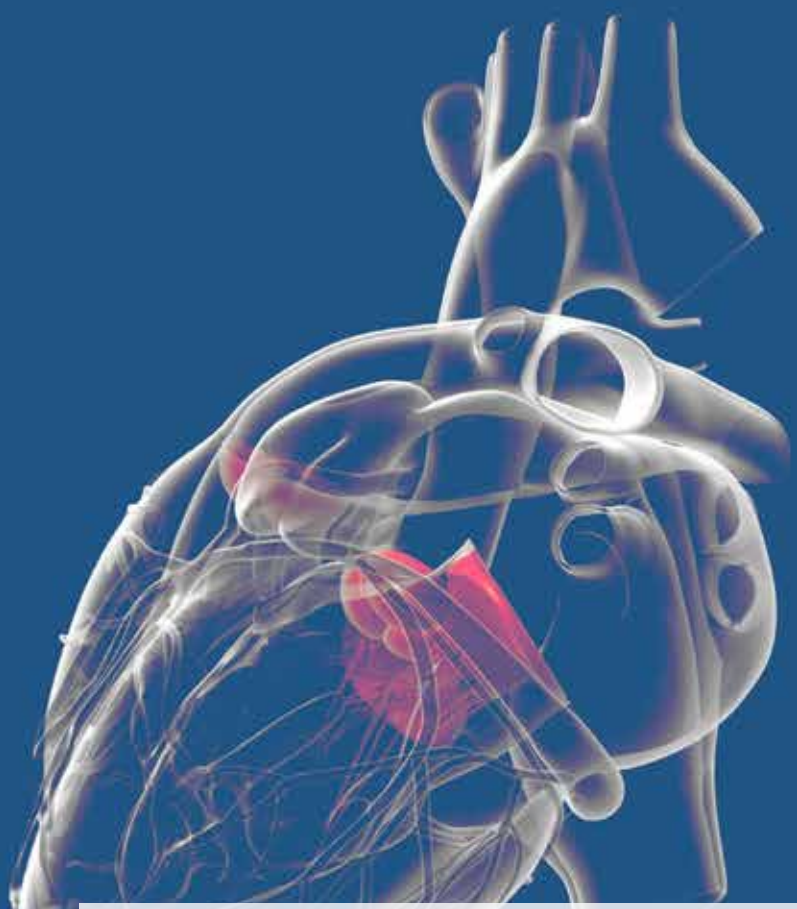
To attend the 2018 Annual Meeting of the Association for Research in Vision and Ophthalmology (ARVO), Honolulu, Hawaii, USA, 27 April - 7 May 2018.

Dr Jia-Yun Tsai
Dept. of Molecular Medicine & Pathology, The University of Auckland

To attend the American Society of Microbiology Microbe (ASM Microbe) 2018 Meeting in Atlanta, USA, followed by laboratory visit at the McGovern Medical School, The University of Texas, Houston, USA, 7 - 29 June 2018.







CARDIAC CHANGES IN DIABETIC HEART DISEASE

Normal contraction and relaxation of the heart is controlled by rapid changes in calcium levels. This is brought about by the interaction of key proteins that move calcium ions within heart muscle cells.

As a result of disease or injury, these normal interactions are impaired and the heart undergoes changes collectively known as cardiac remodelling. The result is heart damage.

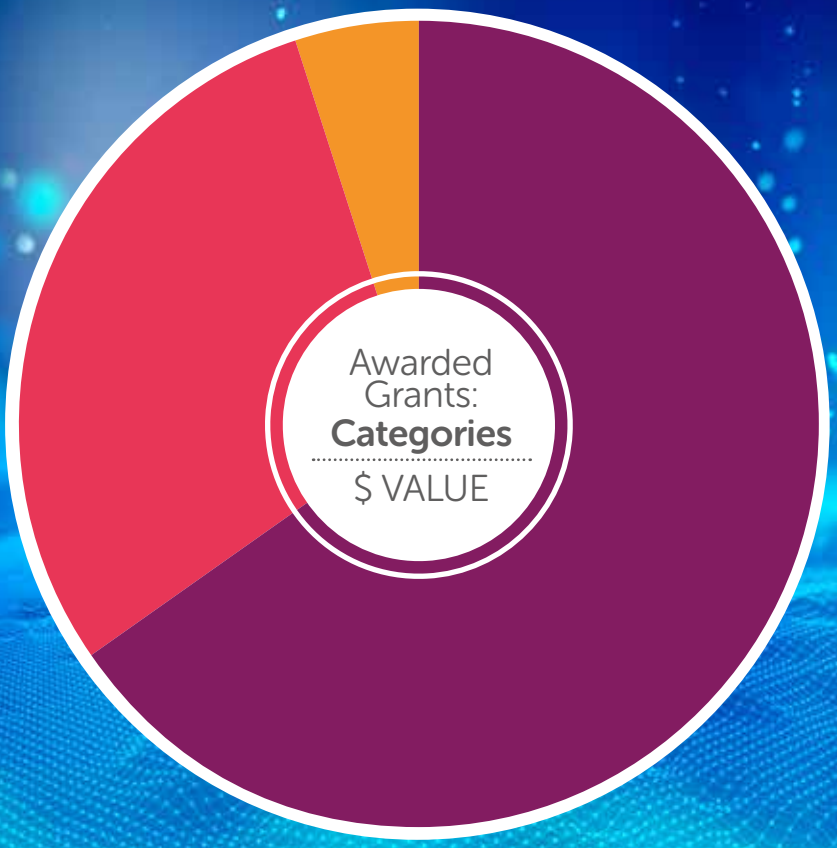
Dr Marie-Louse Ward and her team from the Department of Physiology at The University of Auckland study the effect of cardiac remodelling in people with diabetes.

Recently they showed that proteins known as "Epac" induce alterations in calcium regulation inside heart tissue cells. White arrows in the images indicate Epac, stained in red, which is distributed quite differently within the heart cells of non-diabetic (Panel A) and diabetic (Panel B) patients. Overall cellular structure is also notably disrupted.

They now hold an AMRF grant to examine the role of Epac in human heart disease, particularly in people with diabetes.

They hypothesise that prolonged activation of these Epac proteins results in changes to the heart's ability to contract and cardiac remodelling. Epac may therefore represent an attractive therapeutic target to improve diabetic heart function.

GRANTS COMPLETED



2018 AWARDED GRANTS – CATEGORIES
67 GRANTS AWARDED TOTALLING $4,370,740

	$ Value each category	% Total expenditure
Biomedical (43)	$2,861,346	65.5%
Clinical (19)	$1,293,988	29.6%
Population Health and Community (5)	$215,406	4.9%

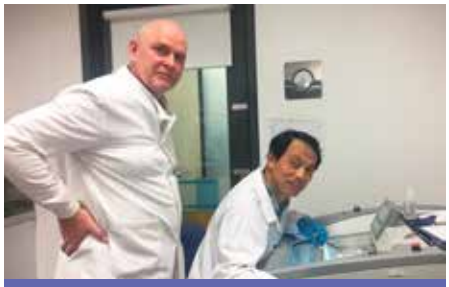
(n) Number of grants

Grants Completed

PROJECTS

MELATONIN AND PREECLAMPSIA ($53,274 – 1.5 years) 1115006

Dr Qi Chen, Dr Katie Groom, Prof Larry Chamley, Prof Peter Stone
Dept. of Obstetrics & Gynaecology, The University of Auckland



Prof Larry Chamley with Dr Qi Chen (right)

Preeclampsia, a human pregnancy-specific hypertensive disease, affects 3-5% of pregnancy and is a leading cause of maternal and perinatal mortality and morbidity globally. However to date the only effective treatment is delivery of placenta, which causes preterm birth. Antiphospholipid antibodies (aPL) are a maternal risk factor that increases the chance of developing preeclampsia by up to 10 fold. This could be due to the production of toxic placental debris by aPL, which can activate endothelial cells seen in preeclampsia. It has been suggested that an increase in oxidative stress in the placenta is associated with the pathogenesis of preeclampsia. Melatonin is a lipid soluble hormone released mostly by the pineal gland, but it is also released from the placenta. More recently, melatonin has been found to have indirect antioxidant activities in both mother and fetus. In this project, we investigated whether melatonin can reverse the production of toxic placental debris induced by aPL and we found that exogenous melatonin reversed the production of toxic placental debris. Since the pathogenesis of preeclampsia is thought to be partially dependent on the maternal response to placental factors including toxic placental debris, our results suggest that melatonin may be a potential therapy for preeclampsia.

URATE CRYSTAL-INDUCED INFLAMMATION IN BONE EROSION DUE TO GOUT ($159,162 – 2 years)

1115015

Prof Nicola Dalbeth, Prof Jillian Cornish, Dr Ashika Chhana
Dept. of Medicine, The University of Auckland

Prof Nicola Dalbeth

Gout is caused by urate crystals that deposit in the joints. It is the most common form of inflammatory arthritis affecting adults in Aotearoa New Zealand. Some people with gout develop bone and cartilage damage, which can cause deformity and difficulty with function of the joints. This project studied how inflammation triggered by urate crystals can contribute to bone damage in gout. This involved studying bone cells and samples from people with gout in the laboratory. This research showed that inflammation triggered by urate crystals has a major impact on both osteocyte and osteoblast cells, leading these cells to release factors that contribute to bone damage. We also identified that non-steroidal anti-inflammatory drugs (medications that are widely used as short-term treatment for gout attacks) can inhibit these effects in bone cells, suggesting a potential new treatment approach to prevent joint damage in people with gout.

FUNDED BY: The Richardson No. 2 Trust

CLINICAL PROGNOSTIC MODELS IN BREAST CANCER ($72,423 - 1 year)

1116017

Prof Mark Elwood, Prof Ross Lawrenson, Dr Sandar Tin Tin, A/Prof Vernon Harvey, A/Prof Ian Campbell
Section of Epidemiology and Biostatistics, The University of Auckland

Dr Essa Tawfiq, Prof Mark Elwood and Dr Sandar Tin Tin

We developed and validated a prediction model to estimate the probability of 10-year breast cancer survival, using the breast cancer registries in Auckland (n=9358) for development and internal validation and in Waikato (n=2627) for external validation. External, independent, validation confirmed good discrimination and calibration; the C-statistic was 0.83, and the predicted 10-year breast cancer survival were within 95% CI of the observed survival in each of ten patient groups. Two conference presentations and a draft paper have been produced. We plan to assess the model in sub-groups of women (such as Maori, women aged under 40 years, and women aged 70 years and older), and to compare its performance to other prediction models developed overseas.

FUNDED BY:
The Hugh Green Fund

CRISPR/CAS9 SCREENING IN HUMAN TUMOUR XENOGRAFTS ($147,536 – 2 years) 1115022

Dr Stephen Jamieson, Prof William Wilson, Prof Cristin Print, Dr Francis Hunter

Auckland Cancer Society Research Centre, The University of Auckland

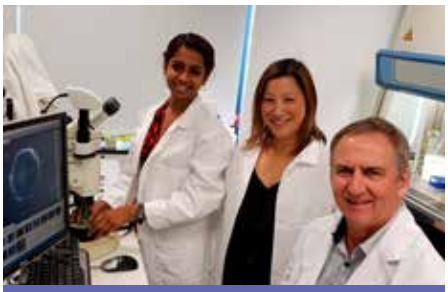
Dr Stephen Jamieson in the laboratory

We have developed a new tumour xenograft model that allows us to discover genes in tumours that determine sensitivity to anticancer agents. We first evaluated multiple head and neck cancer cell lines to find cell lines that required small (UT-SCC-54C) or large (UT-SCC-74B) numbers of cells to form tumours in mice. Using CRISPR/Cas9 technology, we knocked out all genes individually (one gene per cell) in large populations of UT-SCC-54C or UT-SCC-74B cells then injected the knockout cells into mice. The knockout cells from both cell lines formed tumours in mice, but there was much higher representation of individual gene knockouts in the UT-SCC-54C tumours, indicating that a majority of the cancer cells had contributed to tumour growth in this tumour model. The high representation of gene knockouts in our UT-SCC-54C CRISPR/Cas9 tumour xenograft model suggests that this model is ideal for discovery of therapeutic genes and to that end, we have tested this tumour model with two therapeutic agents, evofosfamide and 6-thioguanine, and are currently evaluating the genes that influenced sensitivity to these agents on tumour growth.

CYSTINE/CYSTEINE REDOX SIGNALLING IN THE AGING EYE ($106,260 - 2 years) 1116006

Dr Julie Lim, Prof Paul Donaldson, Dr Monica Acosta

Dept. of Physiology, The University of Auckland



Renita Martis, Dr Julie Lim and Prof Paul Donaldson

With advancing age, oxidative stress results in eye diseases which threaten the sight of the elderly. We have identified the cystine/glutamate antiporter (CGAP) in the eye to play a role in minimising oxidative stress. Clinical assessments on CGAP knockout mice reveal the early onset of eye diseases. To determine the molecular pathways that contribute to these distinct pathologies in the eye, we performed a series of molecular biology, biochemical and imaging experiments on the lens and retina of the CGAP knockout mice. We showed that in the absence of CGAP, the lens produces high levels of reactive oxygen species, resulting in oxidative damage and the early development of cataract. In the retina, we showed that in the absence of CGAP, glutamate levels were altered and neurotransmission was delayed, indicative of the early onset of retinal degeneration. Collectively, our findings demonstrate the CGAP knock out mouse to be an excellent model for studying the underlying pathways that contribute to age related eye diseases. These complementary studies will be used to inform future work towards the design of effective therapies that target a specific tissue of the eye against oxidative stress to delay the onset of age related eye diseases.

FUNDED BY: John Jarrett Trust

perpetual guardian

LONG QT SYNDROME AND HYPERTROPHIC CARDIOMYOPATHY GENE ANALYSIS ($50,000 – 1.5 years) 2113017

Dr Donald Love, Dr. Jonathan Skinner, Dr. Ivone Un San Leong

Diagnostic Genetics, LabPLUS, Auckland Hospital

Dr Donald Love

The principal aim of our research was to establish a high throughput approach to screen for mutations in a large number of genes in patients with heritable cardiac disorders. We successfully developed an approach to screen for mutations in 29 genes in a batch of eight patients in a simultaneous sequencing pipeline. We validated this approach for implementation in a clinical diagnostic setting. Finally, we developed a web-based application for analysing sequence variants using 10 in silico prediction programmes in order to identify those variants with disease-causing potential. This approach subsequently led to the identification of prediction programmes that were most relevant for gene-specific mutation assessment.

Grants Completed continued

PREVENTING THE DEVELOPMENT OF IMPAIRED GAIT PATTERNS AFTER STROKE ($159,046 – 2 years) 1115016

Dr Andrew McDaid, Ms Anna McRae, Dr James Stinear, A/Prof Cathy Stinear
Dept. of Mechanical Engineering, The University of Auckland

Dr Andrew McDaid

Our long-term aim is to improve clinical practice by demonstrating that a simple mechanical device can prevent stroke patients from developing the inefficient and unstable gait pattern that typically afflicts chronic stroke survivors. The aim of this project was to develop and pilot test a novel device for retraining walking in acute stage stroke rehabilitation. The Re-Link Trainer was shown to effectively constrain a limb to a set walking pattern and preliminary results demonstrate its feasibility to be used for chronic and acute stroke patients.

FUNDED BY:
W & WAR Fraser Charitable Trust

PAIN IN THE BACK! DECIPHERING WHICH CELLS DRIVE DISC DEGENERATION ($147,194 – 18 months) 1114015

Dr Sue McGlashan, A/Prof Ashvin Thambyah, Dr Taryn Saggese
Dept. of Anatomy & Medical Imaging, The University of Auckland

Dr Sue McGlashan

Intervertebral disc degeneration is a major cause of back pain. The intervertebral disc consists of an outer fibrous ring, the annulus fibrosus, which surrounds an inner gel-like centre, the nucleus pulposus. Strong annular fibers contain the nucleus pulposus and distribute pressure evenly across the disc, whereas the nucleus pulposus acts as a shock absorber. With degeneration, the nucleus pulposus becomes fibrous and stiff, unevenly transferring loads to the annular walls creating areas of high stress, increasing the risk of disc herniation. Although changes in the nucleus pulposus are thought to initiate disc degeneration, how this occurs is still poorly understood. In this study, we examined two major cell types present in the nucleus pulposus and how they signal to each other. In contrast to other studies, we found that the 'potent' cell type which is thought to have a protective function to the disc had effect on the biological behaviour of the other 'maintenance' cell type, and vice versa. We are further examining if mechanical stress has an effect on the two-way signalling relationship of these two important cell types.

FUNCTIONAL OUTCOMES AFTER FONTAN SURGERY ($32,224 – 18 months) 1115019

Dr Kathryn Rice, Dr Tom Gentles, Dr Tim Hornung
Dept. of Paediatrics, The University of Auckland

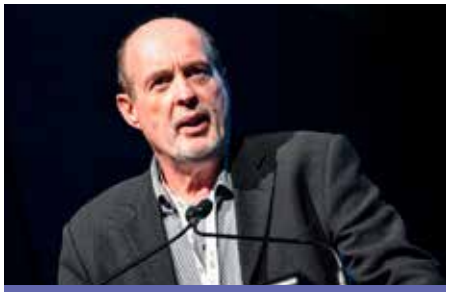
Dr Kathryn Rice

This study of functional assessment in teenagers and adults born with half a heart was conducted across Australasia from February 2016 until 31 January 2018. This currently incurable heart condition requires a series of operations, the last of which called the Fontan operation, to create an imperfect heart circulation. There is an expanding population of around 1350 people living with a 'Fontan circulation' in Australasia. The Australia and New Zealand Fontan Registry has been set up to advance knowledge and improve the quality and quantity of life for people with this condition. This is the first extensive functional study of patients with a Fontan circulation undertaken worldwide. Investigations included assessment of bone density, vitamin D levels, brain application and imaging, heart imaging with echocardiography and MRI, exercise performance and imaging with echocardiography and MRI, and quality of life assessment. Travel and accommodation funding for New Zealand participants was gratefully received from the Auckland Medical Research Foundation. The investigations were funded through an Australian National Health and Medical Research Council Grant. 129 people have participated across Australia and New Zealand. Analysis of the results from the investigations is currently underway with publication expected from the end of this year onwards.

EFFECTIVENESS OF FOOTWEAR IN PEOPLE WITH GOUT ($106,553 – 2 years) 5114003

Prof Keith Rome, A/Prof Nicola Dalbeth, Prof Peter Gow, Prof Peter McNair, A/Prof Alain Vandal

Dept. of Podiatry, Auckland University of Technology



Prof Keith Rome

The objective of the study was to investigate the effectiveness of podiatric foot care packages on foot pain and disability for people with gout. Participants with gout were randomly allocated to receive standardised podiatric care or standardised podiatric care with footwear. The primary outcome was foot pain at 6-months. Key secondary outcomes were general pain, patient global assessment, activity limitation, foot impairment and disability. Ninety-four (n=94) participants were recruited into the study (47 in the intervention arm and 47 in the control group). Perceptions of footwear, intervention adherence and adverse events were also measured. We found no difference in foot pain between the two arms at 6-months. General pain was significantly reduced at 2-months with the intervention arm, but not at 6-months. Foot-related disability was reduced at 2-months in the intervention arm, but not at 6-months. Differences in footwear fit, comfort, ease and weight were observed at 6-months in the intervention arm compared to control arm. No other differences were observed at 6-months. In conclusion, foot care packages comprising of gout, foot health and footwear education are associated with improvements in foot impairment, foot disability, footwear comfort and fit in people with gout.

HNF1B-ASSOCIATED DISEASE IN A HUMAN KIDNEY ORGANOID MODEL ($120,000 - 1 year) 1116018

Dr Veronika Sander, Prof Alan Davidson, Dr Rinki Murphy

Dept. of Molecular Medicine & Pathology, The University of Auckland

Dr Veronika Sander at the lab bench

The aim of this project was to establish a human-based model system to study kidney birth defects that are frequently caused by mutation of the gene HNF1B. These defects include malformed kidney tubules, renal cysts and early onset diabetes and are largely untreatable, thus frequently proceed to chronic and end-stage kidney disease. Our approach was to grow kidney organoids (mini kidneys in a dish) from induced pluripotent stem cells. To mimic HNF1B-related birth defects, we created HNF1B-mutant organoids by using the powerful genetic engineering tool CRISPR/Cas9. Over the course of this project, we have successfully established this system and compared normal kidney organoids to HNF1B-mutant organoids. These results show that the mutant organoids form incomplete kidney tubules similar to the defects found in patients. This validates the use of kidney organoids to study human kidney conditions. Ongoing work is focused on elucidating the mechanisms of how HNF1B regulates kidney development in the human foetus and how malformations arise when the gene is mutated. We hope that these findings will contribute to the development of new therapeutic treatments for patients suffering from genetic kidney defects.

IMPROVING PATIENT RECOVERY AFTER ABDOMINAL SURGERY USING A LONG ACTING LOCAL ANAESTHETIC IMPLANT ($154,940 – 2 years) 1114011

Dr Manisha Sharma, Prof Andrew Hill, Dr Darren Svirskis

School of Pharmacy, The University of Auckland

Dr Manisha Sharma

A drug loaded polymeric implant formulation with desired mechanical properties was successfully developed. Lidocaine release from the developed implants was performed both in synthetic media, phosphate buffered saline (PBS) and in human peritoneal fluid (PF) to better understand the expected drug release in vivo. The implant formulation was able to provide sustained release of lidocaine over a period of 10 days. The release of lidocaine occurred at a faster rate in PF as compared to PBS, owing to the increased solubility and better wettability in PF. The in vivo study using a sheep animal model confirmed the suitability of implant formulation to be used to treat post-operative pain. Sustained lidocaine release was demonstrated without adverse effects from the implant. Histological examination revealed a similar tissue response between the implants and widely used silicon drains after 3 days. The data generated is supportive to advance to the next stage, which is a human clinical trial. Research outputs generated from this study included three peer-reviewed journal articles in international scientific journals of repute, four international conference presentations and one doctoral thesis. An application to file the provisional patent is currently in preparation phase.

Grants Completed continued

EPIGENETIC TARGETING OF METASTASIS ($106,725 - 2 years)

1116002

Dr Dean Singleton, A/Prof Adam Patterson

Auckland Cancer Society Research Centre, The University of Auckland

Dr Dean Singleton in the laboratory

Each year around 500 New Zealand women are diagnosed with Triple receptor Negative Breast Cancer (TNBC). TNBC is an aggressive disease with 77% four year survival rate. A greater understanding of the molecular characteristics of this disease is needed to enable development of effective new treatment strategies. A key driver of the metastatic progression of breast cancer cells is a protein called HIF-1α. HIF-1α is usually found only in oxygen starved cells but it becomes abnormally expressed in TNBC. This project evaluated two new strategies for blocking HIF-1α activity in TNBC. One approach used drugs that can displace HIF-1α from the DNA and dampen down its pathogenic effects. Another, more successful strategy, utilised drugs that block the metabolism of glutamine, an abundant amino acid that is favoured as a nutrient source by TNBCs. We found that inhibiting glutamine metabolism strongly depleted HIF-1α levels and reduced the invasive potential of TNBC cells. This newly discovered link between glutamine metabolism and HIF-1α biology may be employed to guide the clinical development of drugs designed to inhibit glutamine metabolism.

FUNDED BY: Anonymous donor

CREATING NEURAL BRIDGES: A CONDUCTING POLYMER NEUROTRANSMITTER RELEASING SYSTEM ($150,215 – 2 years) 1114010

Dr Darren Svirskis, A/Prof Johanna Montgomery, Prof Jadranka Travas-Sejdic

School of Pharmacy, The University of Auckland

(from left to right) Saiful Azmi, Dr Zaid Aqrawe, A/Prof Darren Svirskis and Mahima Bansal

We sought to advance our understanding of conducting polymer (CP) based materials for use at the Brain-Machine interface. We have developed and tested CP coatings on microelectrode arrays and applied these to both sense and to stimulate nerve cells. A number of delivery platforms have been developed where the release of a medicine can be tuned by electrical stimulation. We are continuing to explore how electrical stimulation can tune glutamate release from our delivery systems. Together, our technologies will be used to record the activity of one population of nerve cells and to pass the signals on to a separate population of nerve cells. This research has opened up new capabilities and we are exploring how communication between different nerve cells can be applied to spinal cord injury where communication pathways are damaged.

TREATMENTS FOR COCHLEAR NEUROPATHY ($146,463 – 2 years)

1115013

A/Prof Srdjan Vlajkovic, Prof Peter Thorne

Dept. of Physiology, The University of Auckland

A/Prof Srdjan Vlajkovic

Hearing loss affects 10-13% of New Zealanders and this prevalence will increase with the aging population. The most common causes of acquired hearing loss in humans are aging and noise exposure. These are associated with the loss of sensory cells and auditory neurons in the cochlea of the inner ear. Prosthetic rehabilitation via hearing aids and cochlear implants cannot repair cochlear injury, hence it is essential to develop therapies that can protect the delicate structures of the inner ear and thus preserve hearing. In this study, we investigated how the blocking of two proteins that bind together and function as a molecular switch for adenosine receptors in the cochlea can improve the survival of cochlear tissues after exposure to noise and rescue hearing. The results of our study were encouraging, and potentially represent a critical translational research for prevention and therapeutic management of acquired hearing loss.

JEAN CATHIE FUND FOR TINNITUS RESEARCH

CHANGES IN BRAIN NETWORKS UNDERLYING TINNITUS DUE TO BRAIN STIMULATION AND HEARING AIDS ($163,686 – 2 years) 1415001

Dr Giriraj Shekhawat
Section of Audiology, The University of Auckland

Dr Giriraj Shekhawat

Tinnitus, or "ear and head noise", is a highly prevalent condition affecting approximately 10% of the New Zealand population. Severe tinnitus can lead to disruption of work, social activities, sleep; and lead to anxiety and depression. There is a pressing need for greater understanding of how tinnitus arises, and evolves over time, in order to develop effective therapies to address this common problem. Traditional group based studies have failed to answer questions about the individual neurophysiological components underlying tinnitus. Considering the heterogeneity in tinnitus sufferers, we are using a new research model (longitudinal multiple case studies) that investigates brain connectivity (the pattern of how individual or clusters of brain cells connect to others) and individual factors in response to non-invasive brain stimulation and hearing aids. This longitudinal case study design may also provide prognostic indicators for client specific tinnitus management. Ten participants with chronic tinnitus have undergone one session of brain stimulation (HD-tDCS) and a digital hearing aid fitting. We collected data from behavioural tests and experiments examining brain connectivity every three months up to 18 months of hearing aid use (using resting state functional magnetic resonance imaging and psychoacoustical tinnitus measurements). We are currently in the process of analysing the behavioural and fMRI data. Once the data is analysed, it will be published in a peer reviewed journal.

FUNDED BY: Jean Cathie Research Fund

SELECTIVE ACTIVATION OF GABAERGIC NEURONS TO TREAT TINNITUS ($199,987 – 2 years) 7415002

Dr Yiwen Zheng
Dept. of Pharmacology and Toxicology, University of Otago

Prof Paul Smith (top left), A/Prof Yiwen Zheng (bottom left) and the tinnitus and balance research team

Tinnitus is a ringing, buzzing or roaring sound in a person's ear or a person's head without the corresponding external sound. Tinnitus can cause sleep disturbances, cognitive problems, work impairment and sometimes, even suicide. There is no effective treatment currently available, mainly due to a lack of understanding of the underlying mechanisms of tinnitus. Research has suggested that tinnitus may be caused by neuronal hyperactivity in the brain. This project aims to make one type of neurons that is responsible for reducing the activity of other neurons respond to light stimulations and to test if light stimulation will change neurotransmission and tinnitus perception in an animal model of tinnitus. So far, we have transduced light sensitive proteins into the target neurons and measured discrete neurochemical changes following tinnitus-inducing noise. These methods will be used to test optimal stimulation paradigms for tinnitus prevention and treatment in animals. These results would ultimately lead to the development of target-specific therapies in humans.

FUNDED BY: Jean Cathie Research Fund

DOUGLAS GOODFELLOW MEDICAL RESEARCH FELLOWSHIP

DETERMINANTS OF SERIOUS SKIN AND SOFT TISSUE INFECTION IN NEW ZEALAND CHILDREN ($282,500 – 3 years) 1414001

Dr Mark Hobbs
Centre for Longitudinal Research, The University of Auckland

Dr Mark Hobbs

This project investigated the epidemiology of skin and soft tissue infections (SSTI), amongst children enrolled in the Growing Up in New Zealand longitudinal cohort study. The key findings of this project were that: (1) New Zealand children received more antibiotics than children in comparable developed countries, and Māori and Pacific children received more than European or Asian children. (2) Hospitalisations for infectious diseases in general, and for SSTI in particular, were common and disproportionately affected Māori and Pacific children. Some potentially modifiable risk factors were identified. (3) The burden of SSTI managed in the community was many times greater than that managed in the hospital, and also fell disproportionately on Māori and Pacific children. (4) Colonisation with *Staphylococcus aureus* or *Streptococcus pyogenes* was associated with an increased risk of SSTI. While the genotype of the colonising strain of S. aureus did not appear to influence the risk of SSTI, methicillin resistance of the colonising strain was associated with an increased risk. Co-colonisation with both S. aureus and S. pyogenes was associated with a high risk of SSTI, and when co-colonisation was taken into account, colonisation with either organism alone was no longer associated with an increased risk of SSTI.

Grants Completed continued

POSTDOCTORAL FELLOWSHIPS

EDITH C. COAN RESEARCH FELLOWSHIP

EFFECTS OF CALCIUM ON INDICES OF BONE AND CARDIOVASCULAR HEALTH ($175,863 and $30,000 – 2 years) 1314001 & 1715001

Dr Sarah Bristow Dept. of Medicine, The University of Auckland

Dr Sarah Bristow with a patient

Calcium supplements have been associated with increased cardiovascular risk, but how this occurs is uncertain. In a randomised cross-over trial of postmenopausal women, we showed that blood pressure was higher in the hours after a calcium supplement was taken compared with a placebo. If repeated long-term, this could contribute to increased cardiovascular risk. As calcium supplements have been recommended to be taken in the morning and evening, we are presently undertaking a second trial examining how this dosing regimen affects blood pressure over 24 hours. Strategies to prevent or treat osteoporosis are urgently required. High intakes of calcium have been believed to be important for bone health, but most people do not meet the recommended intakes. We therefore investigated whether dietary calcium intake was related to bone loss in 800 women and 100 men, finding calcium intake did not affect the amount of bone people lost. We then searched for and combined all studies that had ever examined calcium intake in relation to bone loss, finding that most studies reported no relationship between calcium intake and bone loss. These findings suggest that public-health strategies aimed at increasing dietary calcium intake are unlikely to reduce the prevalence of osteoporosis.

IMPACT IN CANCER ($182,861 and 30,000 – 2 years) 1315001 & 1716001

Dr Petr Tomek Auckland Cancer Society Research Centre, The University of Auckland

Dr Petr Tomek

Cancers use a number of cunning strategies to escape the host immune system. In one strategy, cancers produce high amounts of an enzyme called IDO1 to starve the patient's immune cells of the essential amino acid tryptophan. Immune cells are very sensitive to tryptophan deficiency, and become dysfunctional and die. Yet, cancer cells in the same environment remain alive. In this research we investigated how cancer cells overcome tryptophan starvation. Brain cells and skin cells have been shown to use a protein called IMPACT which allows them to withstand amino acid shortage. We hypothesised that cancer cells also hijack IMPACT for the same protective purpose. We showed that a broad range of human tumours produce substantially higher amounts of IMPACT compared to normal healthy tissues. When brain cancer cells were cultured in low levels of tryptophan, the cells with higher levels of IMPACT survived better than the cells with the low IMPACT abundance. These observations support the hypothesis that IMPACT helps cancer cells to overcome tryptophan starvation. Overturning survival mechanisms mediated by IMPACT in cancer cells could therefore represent a novel strategy to combat cancer.

FUNDED BY: Edith C Coan Trust and the Kelliher Charitable Trust

perpetual guardian

Kelliher Charitable Trust

DOCTORAL SCHOLARSHIPS

J.I SUTHERLAND FUND FOR MELANOMA RESEARCH

THE MC SUBSETS IN HEALTH AND DISEASE ($126,500 – 3 YEARS) 1214002

Miss Jennifer Eom School of Biological Sciences, The University of Auckland

Miss Jennifer Eom in the laboratory

Tumours consist of malignant cancerous cells as well as normal cells that help the cancer cells survive and grow. Tumour-Associated Fibroblasts (TAFs) - or more correctly "mesenchymal cells" (MCs) - are one class of these normal cells that support tumour development in a number of ways. Unfortunately, these cells remain poorly characterised. From 2015 to 2017, we analysed MCs in cancer tissue donated by melanoma patients after their surgery. For the first time, we developed techniques that allow us to distinguish between two different types of MCs in melanoma tissues. We also analysed the kinds of molecules those two types of MCs are making that might help tumours grow or evade attack from the immune system. This analysis suggested one type of MCs produced thick walls, while the other type attracted immune cells. Since these two types of cells are present in different proportions in different tumours they may help explain some of the differences in responses to therapy that different patients experience. These results will enable deeper analysis of melanoma fibroblasts and the development of new cancer therapies that target the molecules used by different types of MCs to support cancer cells.

FUNDED BY: J.I Sutherland Fund for Melanoma Research

HUMAN TROPHOBLAST STEM CELLS ($126,500 – 3 years) 1214004

Miss Teena Gamage

Dept. of Obstetrics & Gynaecology,
The University of Auckland

Miss Teena Gamage

The placenta is a vital foetal organ essential for survival of the baby in utero. Inadequate placental development is associated with pregnancy complications including foetal growth restriction (FGR), which affects approximately 5000 pregnancies in New Zealand annually. Currently there is no effective treatment for FGR, and how or why placentation is impaired in FGR is unknown. However, impaired growth and maturation of specialised placental cells called trophoblasts, are likely to be a major contributing factor. This PhD aimed to expand our knowledge of a candidate trophoblast stem cell population (side-population trophoblasts) in order to understand their role in normal placentation, and their contribution to FGR. This work showed that side-population trophoblasts are present throughout gestation, and exhibit stem-like characteristics at the level of the methylome. Moreover, the proportion of side-population trophoblasts are markedly reduced in FGR compared to healthy pregnancy and may contribute to the pathogenesis of FGR. Conditions were developed to propagate side-population trophoblasts short term, which will enable comparisons of transcriptomic differences found in this work to functional differences in cell differentiation in the future. The knowledge gained in this PhD provides insights into the pathogenesis of FGR that will allow development of treatments for this disorder.

SIR DOUGLAS ROBB MEMORIAL FUND

$6,815 1718006

Dr Celia Keane ($6,815 – 1718006)
Dept. of Surgery, The University of Auckland

Participants in the International Consensus Definition of Low Anterior Resection Syndrome (LARS) meeting, France

This funding enabled two patient representatives and one health professional (nurse) representative from New Zealand to attend the International Consensus Meeting on Low Anterior Resection Syndrome (LARS). The meeting was held in Nice, France on 28 September and was very successful. The consensus meeting was the culmination of a years of work towards developing an internationally accepted consensus definition of LARS. Throughout this process both patients and providers have been involved at all stages. It is vital for patients to be involved and have their voices heard so that any outcomes are clinically relevant and will be able to be applied in real world practice. The New Zealand representatives input was well received at the meeting, they were able to provide valuable insights throughout the discussion, and they formed collegial relationships with other attendees. The New Zealand patient representatives also discussed undertaking additional work to develop patient resources in partnership with the other patient representatives who attended the meeting. The 'academic' outcome of the meeting (and the wider project) will be the production of an international consensus statement on the definition of LARS (currently being drafted). This will enable: better awareness of LARS; improved recognition of those patients who are at risk of or who are suffering from LARS and require additional support, management, or follow-up; construction of measurement tools with increased clinical utility; and improved standardisation of the research into LARS including assessment of the incidence, natural history, risk factors, and potential management or treatment strategies for LARS.

GAVIN AND ANN KELLAWAY MEDICAL RESEARCH FELLOWSHIP

$18,300 1518002

Prof Debbie Hay

School of Biological Science,
The University of Auckland

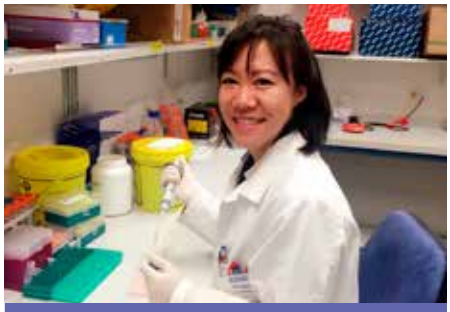
(from left to right) Dr Mohammad Al-Mahdi Al-Karagholi, Prof Debbie Hay and Dr Hashmat Ghanizada

The major purpose of this Gavin and Ann Kellaway Medical Research Fellowship was to visit the Danish Headache Centre in Copenhagen. Severe headache disorders, especially migraine, have a major impact on New Zealanders. During her visit to this world-leading centre, Professor Hay gained a much greater understanding of the causes, impacts and new treatments for headache disorders. She learned about research into hormones that can trigger migraine attacks, which aligns with her own research on hormone receptor targets. In addition to visiting this centre, Professor Hay presented her research at the University of Cambridge and attended two migraine-related conferences. Overall, this was a valuable experience, which will lead to enhanced international research efforts and will further enable headache research projects in the Hay lab.

HEALTHEX EMERGING RESEARCHER AWARD

$3,000 Travel Award 6716001

Miss Grace Gong
Dept. of Molecular Medicine & Pathology, The University of Auckland



Miss Grace Gong

The award allowed me to travel to Cambridge and Oxford, and visit some of the best laboratories in the world. This in itself was a privilege, that I was able to talk to these world leading scientists about their research, listen to their ideas, learn from them, not just in the academic setting, but also as a person. Their ways of doing things, their humbleness and their attitude towards both their work and the people around them - it was so impressive and inspiring. I also came to realise that having this award on my CV worked so much in my favour, I received a lot of compliments about it, and was even invited to give a talk at University College London.

Furthermore, I attended a prestigious Keystone Symposia conference in New Mexico, meeting the leaders and big names in my field, I was like a little girl who finally gets to see her superstars. Through this conference I was able to learn and expand my understanding of the topics covered, build relationships and potential collaborations with these top scientists, and most importantly, I had the opportunity to present some of the work I did during my PhD. The research was well received and sparked interest from scientists all around the world, including those from Cambridge, Harvard, and big pharmaceutical companies. This shows that although we are a small country, the quality of our research, and what we are capable of doing is definitely up there with these leading scientists. I was very proud.

This trip was invaluable to me, it helped me grow so much both as a scientist and as a person. It gave me mind opening experiences that have shaped my path going forward, and all of this would not have been possible without the AMRF Emerging Researcher Award. I have been very fortunate, and can't express my gratitude enough.

FUNDED BY:
Wellington Sisters Charitable Trust

$3,000 Travel Award 6717002

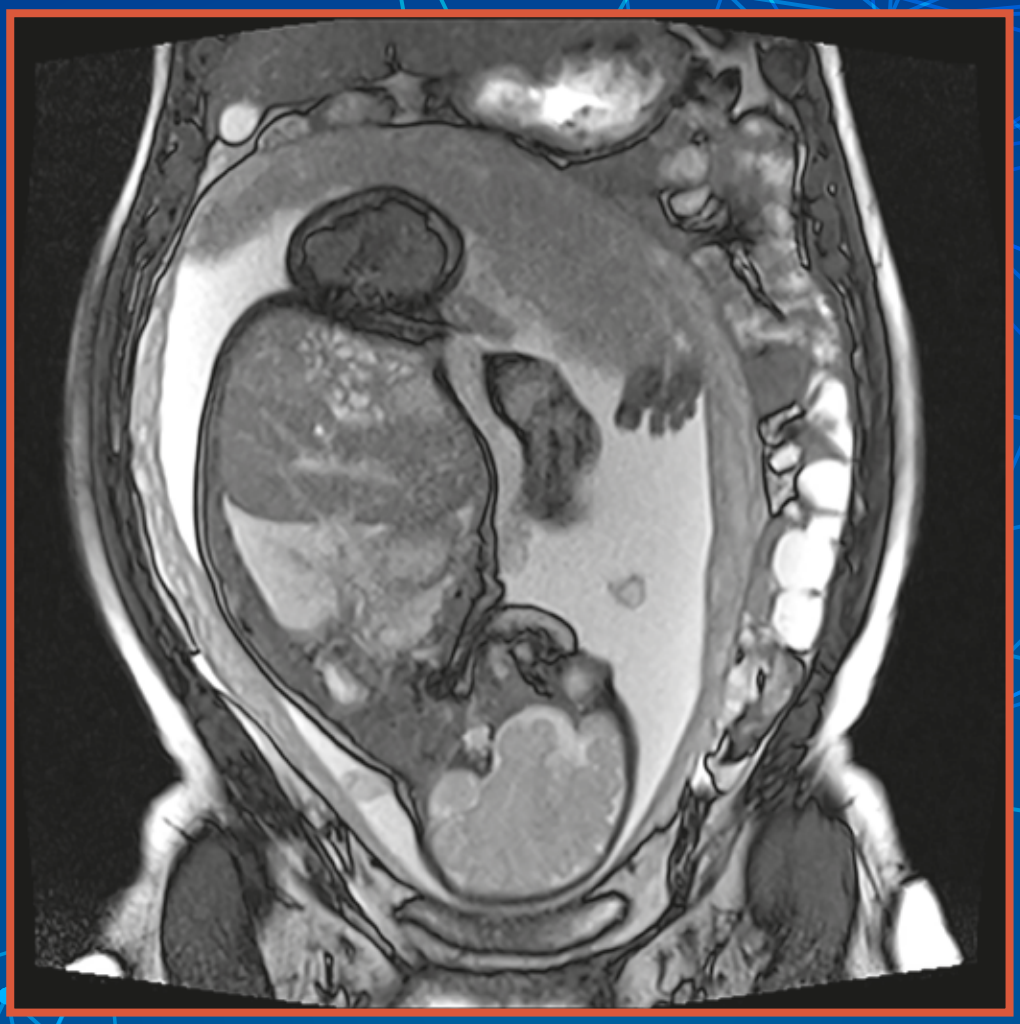
Miss Melanie MacFarlane
Dept. of Paediatrics, University of Auckland

Melanie MacFarlane in Glasgow

My PhD research focuses on Māori sudden unexpected death in infancy (SUDI). SUDI deaths occur in infants up to one year of age and include SIDS (sudden infant death syndrome), which are deaths that remain unexplained even after a thorough investigation. Today, the most significant SUDI risk factors for all infants, regardless of ethnicity, are smoking in pregnancy and infant bed sharing. When infants are exposed to both factors, their SUDI risk increases dramatically. More than half of all SUDI in this country is associated with unsafe sleeping, including bed sharing.

Part of my research investigated the reason for the continuing SUDI disparity experienced by Māori from a contemporary perspective. Our findings were consistent with previous results, which stated that the difference in SUDI rates for Māori and non-Māori can be explained by the higher exposure among Māori infants to the combination of smoking in pregnancy and bed sharing.

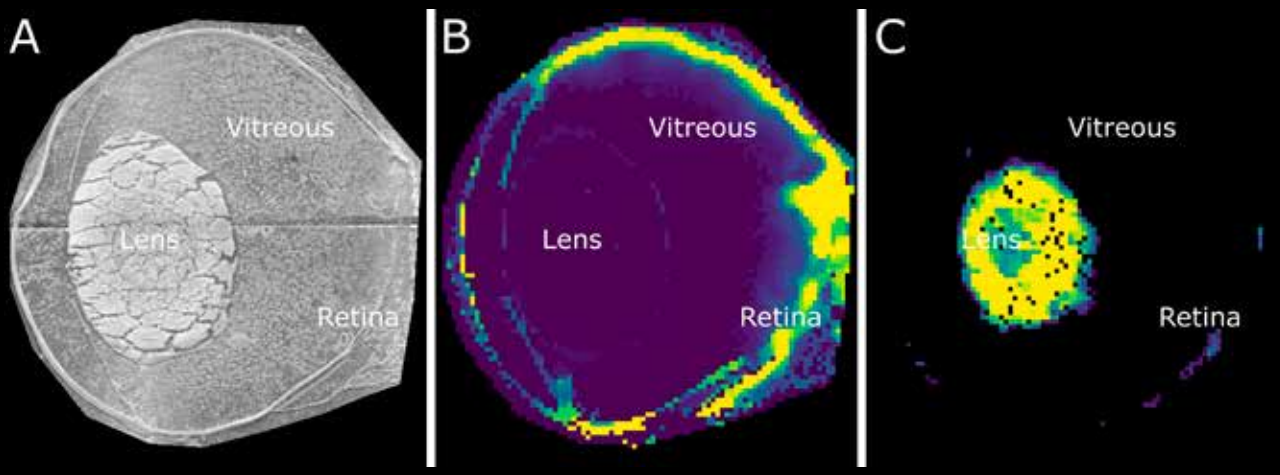
This travel award enabled me to share this research with international researchers. I attended the Paediatric Society of New Zealand 69th Annual Scientific Meeting in Christchurch in November 2017, followed by the International Conference on Stillbirth, SIDS and Baby Survival in Glasgow, Scotland in June 2018. The most rewarding aspects of this travel, apart from communicating my research to others, was having the opportunity to listen and talk to SUDI experts from around the world. Most poignant was the involvement in Glasgow of families that have experienced stillbirth, SIDS or SUDI, and hearing their stories of loss, courage and hope. It served as a reminder to the clinicians, researchers and policy makers about why we work in this field, and that is to help prevent sudden and unexpected deaths among the most vulnerable members of our population - infants.



MOTHER AND BABY

The image is a magnetic resonance imaging cross section of a pregnant mother's abdomen in the third trimester of pregnancy. You can see baby (head down) in the uterus, as well as the placenta (the grey 'fluffy' region near the baby at the top of the picture). Images like this form part of our study to understand how mum's body position impacts on oxygen delivery to the baby. We are creating maps of blood flow and oxygen levels in the uterus and placenta, which, using images like this one we can relate to the anatomy of pregnancy (things like the size or position of the placenta).

The image has been kindly supplied by Dr Alys Clark of The Placental Blood Flow Group, The University of Auckland. Read more about her important work funded by the MRI ERD Trust on page 5.



CROSS TALK BETWEEN TISSUES OF THE EYE

Antioxidants are important as they protect tissues of the eye from chemical and UV damage. It is well established that a depletion of antioxidants plays a major role in the onset of age related eye diseases. Antioxidants themselves are small molecules that can be very difficult to measure and detect.

To help understand how overall antioxidant levels are maintained in the eye, AMRF-funded researchers Dr Julie Lim and Dr Angus Grey from the Department of Physiology, The University of Auckland have been studying the inter-tissue exchange of antioxidants and related molecules between the lens and other tissues of the eye.

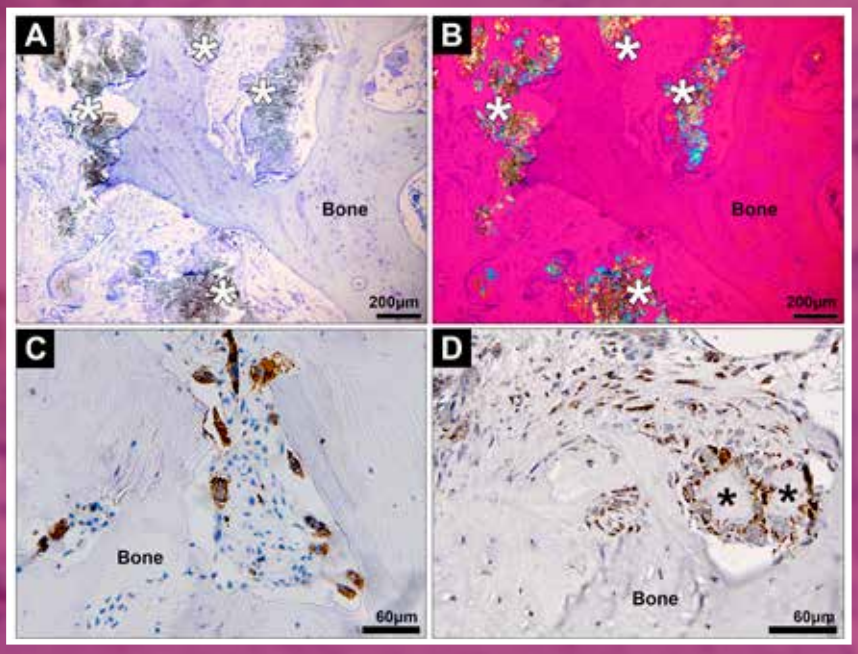
Using a tool known as imaging mass spectrometry (IMS), they have for the first time been able to visualise and detect antioxidants and related molecules in specific tissues of the rabbit eye.

Panel A is a simple cross section of a rabbit eye showing the main structures.

Using IMS, Panel B shows the detection of antioxidants (yellow) mainly in the retina, while Panel C shows them only in the lens. When used together with different eye health treatments, IMS will much more easily show researchers which drugs and therapies can move antioxidants to the tissues that need them most to prevent loss of sight and eye damage.

Image kindly supplied by Dr Angus Grey.

PUBLICATIONS



GOUT CAUSES BONE EROSION

Bone erosion is a frequent complication of gout, but it's not well understood how. In this peer-reviewed publication, AMRF-supported researchers show that the crystals that form in gouty joints may kill important bone cells, called osteocytes, and those cells which do survive behave in an erratic manner, further promoting the loss of bone. This is a newly discovered way gout can lead to painful and disfigured joints.

This image shows microscopic anatomy of human joint tissue affected by tophaceous gout. Representative photomicrographs of joint samples affected by tophaceous gout, showing both monosodium urate crystals (indicated by asterisks) and associated inflammatory tissue in close proximity to bone (A) toluidine blue staining viewed using light microscopy; (B) viewed using polarizing light microscopy with a red compensator). Immunohistochemistry staining for (C) CD68+ cells (macrophages, a type of immune cell) and (D) COX-2 (an inflammatory factor) expression in human joint tissue affected by tophaceous gout.

Image provided by the authors from publication:
Chhana A, Pool B, Callon KE, Tay ML, Musson D, Naot D, McCarthy G, McGlashan S, Cornish J, Dalbeth N. (2018). Monosodium urate crystals reduce osteocyte viability and indirectly promote a shift in osteocyte function towards a proinflammatory and proresorptive state. Arthritis Research & Therapy, 20(1):208.

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FELLOWSHIPS

DAVID & CASSIE ANDERSON POSTDOCTORAL FELLOWSHIP

DR SARAH STEWART

DEPARTMENT OF MEDICINE, UNIVERSITY OF AUCKLAND

Dr Sarah Stewart aims to develop an ultrasound screening programme that will detect the deposition of urate crystals in the joints of people before they show any symptoms of gout, enabling clinicians to treat the disease before it becomes problematic. For the 20% of adults who struggle with the pain of gout every day, Dr Stewart's research has the potential to prevent the development of gout and the associated chronic symptoms that can be debilitating and disabling for so many.

THE RUTH SPENCER MEDICAL RESEARCH FELLOWSHIP

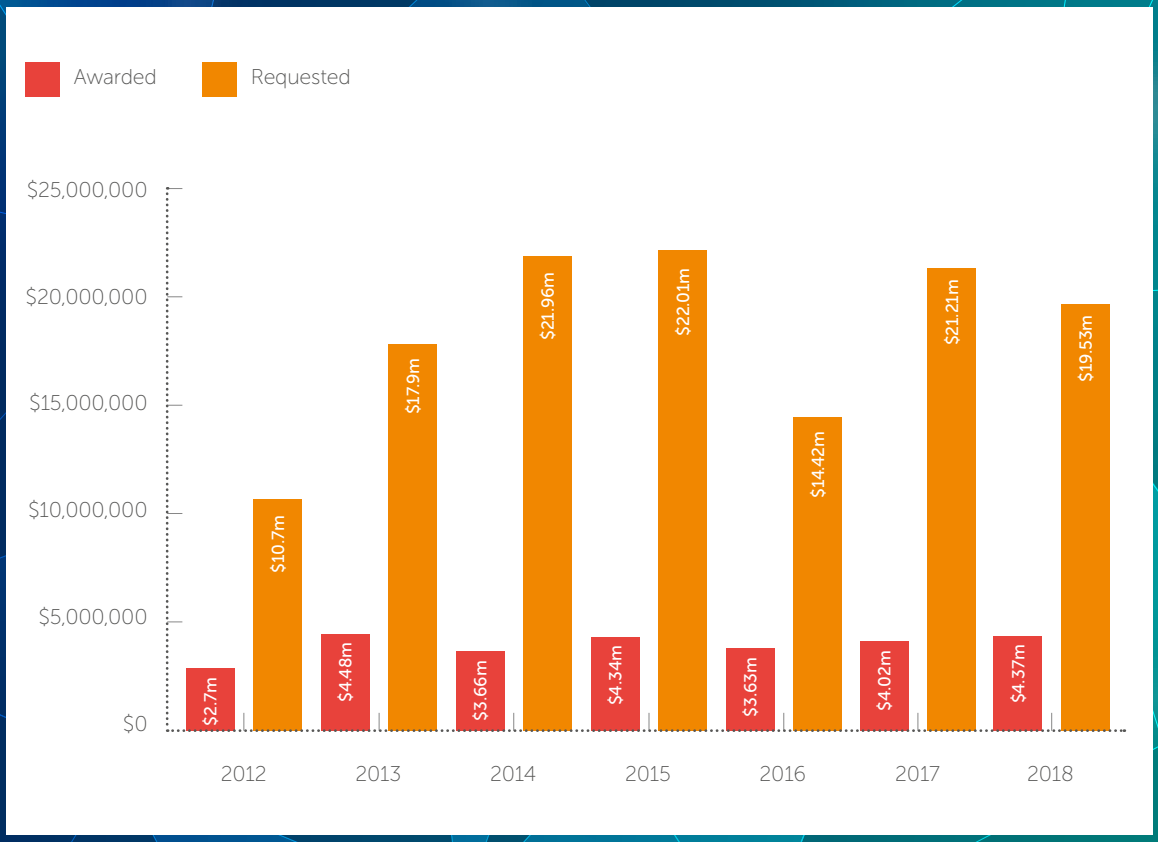
DR LISA DAWES

LIGGINS INSTITUTE, UNIVERSITY OF AUCKLAND

Dr Lisa Dawes will use her fellowship to complete a series of projects which aim to improve the care provided to women who are at high risk of having their baby born early.

Left to right: Professor Peter Browett, Chair of the AMRF Medical Committee, Richard Taylor, President of AMRF, and Gail Stevens from Perpetual Guardian with Dr Lisa Dawes.

FINANCIALS 2018



There are many worthy requests for funding that we cannot support.

Thank you for your generosity.

Financial Highlights 2018

RESEARCH FUNDING 2018 $4,370,740 TOTAL RESEARCH FUNDING SINCE 1955 $75,970,740

FINANCIAL PERFORMANCE

	Note	2018 $		2017 $	
Income					
Donations / Subscriptions	1	654,752		629,399	
Investment Income		1,933,166		2,307,993	
Trust Income / External Funding	1	1,116,432		879,118	
Legacies/Bequests/Specific Donations	2	1,171,968		3,013,788	
Net Gain on realisation of investments	3	22,208,931		451,959	
Net Gain on currency fluctuations		(30,300)		16,010	
Total		27,045,949		7,298,267	
Expenditure					
Operational expenses		504,969		461,012	
(Less Donation)	4	(500,182)	4,787	(461,012)	Nil
Research Grants	5		4,037,550		3,976,305
Depreciation on Grant Funded Assets			-		3,750
Reduction in value of investments			3,697,410		-
Total			**7,739,747**		**3,980,055**
Net Surplus / (Deficit)			**19,306,202**		**3,318,212**

The summary of financial highlights above have been extracted from the Audited Financial Statements which can be obtained by contacting the Foundation's office.

2018			2017
$645,752		Donations / Subscriptions	$629,399
$1,933,166		Investment Income	$2,307,993
$1,116,432		Trust Income and External Funding	$879,118
$1,171,968		Legacies / Bequests / Specific Donations	$3,013,788
$22,178,631		Net Gain on realisation of investments	$451,959

Notes to the 2018 Financial Report

1. Donation & Trust Income includes grants, donations and external funding received from the following organisations:

Perpetual Guardian Administered Funds	
David & Cassie Anderson Medical Trust	201,363
Jean Cathie Research Fund	198,446
Est of Ernest Hyam Davis / The Ted & Mollie Carr Endowment Trust	375,000
The J & P Stilson Endowment Trust	100,000
Rose Richardson Estate	10,000
Room-Simmons Charitable Trust	35,000
Ruth Spencer Estate	66,016
Stichbury Charitable Trust	10,000
N H Taylor Charitable Trust	20,000
NR & JH Thomson Charitable Trust	25,000
Edith C Coan Trust	120,000
John A Jarrett Trust	46,673
C E Lawford Estate	4,000

Public Trust Administered Funds	
Acorn Charitable Trust	20,000
Audrey Simpson Trust	4,550
Pritchard Coutts Charitable Trust	22,500
Ralph Dingle Trust	2,100
Pauline Gapper Charitable Trust	5,400
Reed Charitable Trust	10,000
Wellington Sisters Trust	11,000

Other Trusts/Funds	
Auckland Airport - 12 Days of Christmas	10,000
Gooduck Charitable Trust	128,000
Douglas Goodfellow Charitable Trust	360,000
The Kelliher Charitable Trust	45,000
Marion Ross Fund	17,884

2. Legacies, Bequests and Specific Use Donations 3,511,407

Anonymous

The JI Sutherland Fund

Estate of Rosemary Erson

Estate of Thomas Campbell

Hugo Charitable Trust

Douglas Goodfellow Charitable Trust

3. Net Gain on realisation of investments

During the financial year the Foundation restructured its investment portfolio moving from individual securities into managed funds. This required the sale of all individual bonds and shares and the proceeds invested in managed funds . The 'net gain on realisation' is the difference between the sale price and the original cost of those investments, some of which had been held for many years. This net gain does not form part of operational income.

4. Research Funding Approved 2018

PROJECT GRANTS (20)	2,519,176
AMRF General Purpose & Named Funds Supporting Research Projects:	
AC Horton Estate	
JI Sutherland Fund	
MRI ERD Trust	
W & WAR Fraser	
Room-Simmons Charitable Trust	
POSTDOCTORAL FELLOWSHIPS (2)	399,627
AMRF Postdoctoral Fellowship	
David and Cassie Anderson Research Fellowship	
DOCTORAL SCHOLARSHIPS (4)	
AMRF Doctoral Scholarships (3)	440,890
Helen Goodwin Doctoral Scholarship	
AMRF TRAVEL GRANTS (24)	62,328
OTHER GRANTS	
AMRF Senior Research Fellowship	100,000
Kelliher Charitable Trust Emerging Researcher Start-up Grant (2)	45,000
Sir Harcourt Caughey Award (2)	30,248
Gavin and Ann Kellaway Medical Research Fellowship (3)	115,856
Ruth Spencer Medical Research Fellowship	66,016
The Ted & Mollie Carr Endowment / Ernest Hyam Davis Est - Research Fellowship	375,000
Jean Cathie Research Fellowship	198,446
Douglas Robb Memorial Award	8,153
HealtheX Emerging Research Awards (3)	7,000
Summit Award	3,000
TOTAL GRANT FUNDING 2018	4,370,740
Less amounts allocated but not required	(333,190)
TOTAL GRANTS AWARDED 2018	**4,037,550**

Members, Sponsors & Supporters 2018

WE ARE MOST GRATEFUL TO ALL THE INDIVIDUALS, TRUSTS AND ORGANISATIONS LISTED BELOW WHO HAVE GIVEN GENEROUS SUPPORT TO THE FOUNDATION DURING THE YEAR.

MEMBERS

Honorary Life Members

Byrne, Judi and Peter
Chan, Rebecca and David
Hart, Cliff
Lawrence, Dr Dick
Levene, Sir David
Maclaurin, Dr CH
Nicholson, Prof Louise
Stevenson, Bill & Nari

Life Members

Bain, Roy
Baird, Dr Tony
Batt, Leonie
Bunning, Natalie
Christie, A/Prof David
Collings, Margaret
Crookbain, Margaret
Davies, Amelia
Davies, Matthew
Davies, Noel & Heather
Dickey, Mr KL
Ding, Allan
Ding, Christine
Ding, Thomas
Fish, Barbara
Friedlander, Sir Michael
Gibbons, Dr Hannah
Glass, Paul
Glover, Donna
Glover, Bill
Goodfellow, Dr Bruce & Mary Ann
Goodfellow, Mr & Mrs TB
Goodfellow, Peter & Desley
Green, Prof Colin & Paula
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Hall, Judith
Hall, Richard & Yvette
Hall, Simon
Hendry, Ian
Herle, Suryashobha
Hobbs, Emmet
Howie, Dr Ross & Helena
Jenkinson, Vivienne
Jollands, Elizabeth
Keeling, Paul
Kellaway, Ann
Lake, Margaret
Lawry, Jean
Londeen, Maree
Lorimer, Michael
Lu, Jun
MacCulloch, Donald
MacCulloch, Robert
MacDonald, Cathrine
McElroy, Robyn
McWilliams, Kim
Menzies, Mr & Mrs P
Moffitt, Dr AR
Mount, Elspeth
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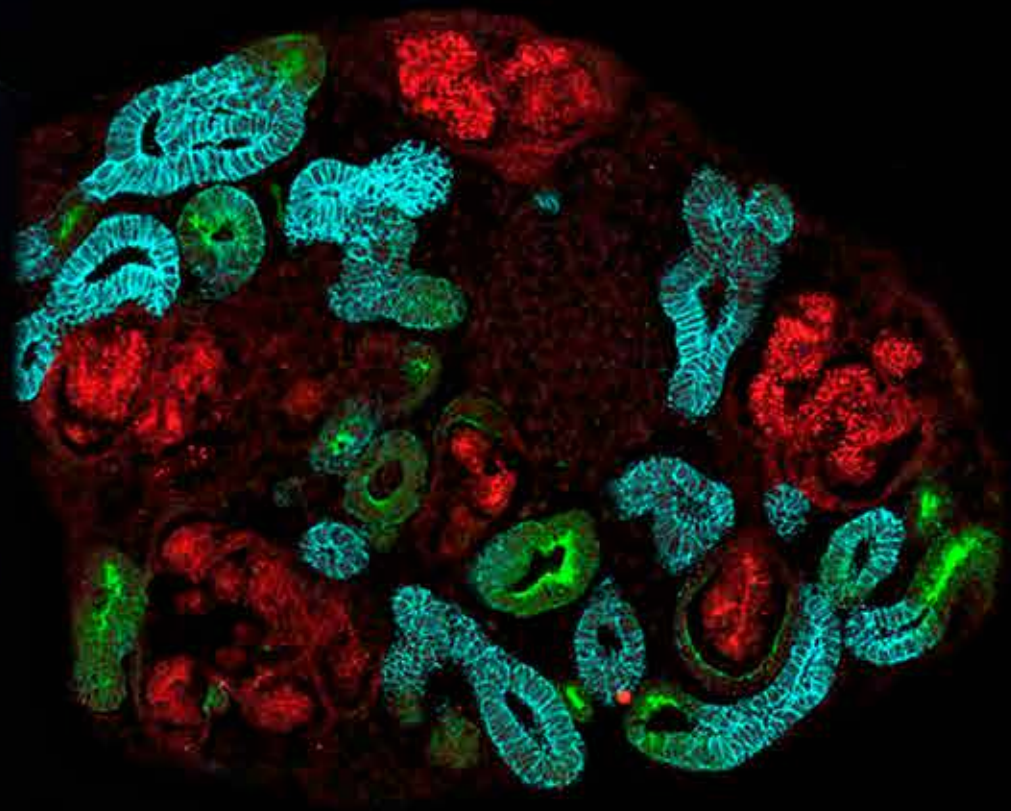

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