

20
15



**ANNUAL
REPORT**

CONTENTS

01	OBJECT OF THE FOUNDATION	02
	OMRF COUNCIL	
03	REPORT FROM THE DIRECTOR OF DEVELOPEMENT	04
	UNDER THE MICROSCOPE	
08	FUNDING BREAKDOWN	09
	EVENTS	
12	CLUB OTAGO	14
	CHAIRPERSON'S REPORT	
16	SCIENTIFIC COMMITTEE REPORT	25
	FINANCIAL HIGHLIGHTS	
26	AUDITOR'S REPORT	27
	INFORMATION ABOUT THE FOUNDATION	
28	LIST OF MEMBERS	29
	FUNDING PATHWAYS	

OBJECT OF THE FOUNDATION

There is not one person alive today who has not benefitted from medical research.

The Object of the Otago Medical Research Foundation: Is for the furtherance of medical research in Otago.

We fund world class research, equipment and facilities for Otago's highly talented medical community of scientists, students, practitioners and lecturers.

Our recipients contribute invaluable medical knowledge that can be applied to medicine and prevention in future, and in doing so we also retain top medical talent and intellectual property in Otago.

MEDICAL RESEARCH IS A LIFE CHANGER. YOU'RE A LIFE CHANGER.

The answers unearthed through medical research irrefutably lead to greater quality of life for society – through earlier diagnosis and treatment. Since the Foundation was established in 1967 it has identified and funded close to \$7 million worth of grants and scholarships, with much of the work undertaken now acclaimed around the world.

The lives of millions of people have ultimately been improved by the research funded by the Otago Medical Research Foundation. Made possible by our generous supporters.

IT ALL STARTS SOMEWHERE.

The Foundation helps to fund medical research projects and scholarships which are highly novel and scientifically worthy, but due to their early exploratory nature don't attract the interest of larger funding agencies.

However in the world of medical research what the Foundation launches cannot be underestimated. Once that initial research has been completed and the answers unearthed, it often opens up new areas of investigation for bigger entities to develop.

So the research never stops and many of our esteemed alumni are now global leaders in their medical fields.

EVERYONE BENEFITS FROM MEDICAL RESEARCH.

There is not one person who has not benefitted from answers unearthed through medical research; whether that be personally, through parents or children, partners or siblings, work mates or their friends. We will all know many who wouldn't be with us had it not been for the discoveries made and the earlier diagnosis and less invasive treatment that research unveils.

It is irrefutable that from medical research we all benefit.

THE OTAGO MEDICAL RESEARCH FOUNDATION COUNCIL

Professor Barry Taylor
Dean Dunedin School of Medicine
ex-officio

Assoc Prof P A Cragg
Chair of Scientific Committee
ex-officio

Mr M C Horne
Deloitte (Secretaries)
ex-officio

Prof A van Rij
Otago University Faculty of Medicine

Dr P Gootjes
N.Z. Medical Association (Otago Division)

Mrs. Sarah Ramsay
Co-opted

Assoc Prof Greg Jones
Co-opted

Prof Vernon Ward
Dean of the Otago School of Medical Sciences

EXECUTIVE

Mr K G Dempster - Chairperson
Assoc Prof P A Cragg - Deputy Chair
**Deloitte representative - Secretary/
Treasurer**

SCIENTIFIC COMMITTEE

**Assoc Prof P A Cragg - Chair Otago
Medical School Members**
(see report on page 17)

DIRECTOR OF DEVELOPMENT

Mr S. Davie

Prof J Highton
General Medical Staff, Otago District Health Board

Mr R Bunton
Otago District Health Board

Dr M Coleman
Elected by Members of the Foundation

Mr K G Dempster
Elected by Members of the Foundation

Mr R P Lewis
Elected by Members of the Foundation

Dr J McMahon MBE
Elected by Members of the Foundation

Ms S Saunderson-Warner
Elected by Members of the Foundation

Assoc Prof Joel Tyndall
President of the Otago Medical School of Research
Society

Dr Victoria Scott
Co-opted

SECRETARIES

Deloitte

HONORARY SOLICITOR

Mr J Anderson
(Galloway Cook Allan)

AUDITORS

Crowe Horwath

PATRON

Emeritus Professor Gil Barbezat

REPORT FROM THE DIRECTOR OF DEVELOPMENT

In tough economic times, the Otago Medical Research Foundation continues to make very steady progress in increasing its capacity to identify and fund world-class, ground-breaking research projects and scholarships.

A mixture of revenue generated through the Foundation's growing calendar of events, individual gifting, bequests, charity and trust grants has seen more than \$3.5 million in new revenue attracted by March 31 2015, five years since the launch of a structured profile and fundraising campaign.

With a growing list of business owners, chief executives and individuals who are now regular benefactors, gaming machine trusts who have made several donations, and many within the charitable industry financially supportive of the Foundation's vision, the funding base is expanding monthly.

The Foundation's calendar of events is growing rapidly.

This is highlighted by a number of 'must attend' functions including the annual dinner 'A Night to Remember', the Club Otago lunch series and the annual golf tournament. Other events through the year included a memory-laden 'Blast from the Past' rugby lunch as the Foundation celebrated its alliance with the Otago Rugby Football Union, a motivational lunch with the inspirational Lisa O'Neill staged in association with the Dunedin Casino, and two marvellous nights alongside Armstrong Prestige - the annual movie night and a fun gaming night.

The Foundation continues to strengthen its partnerships with RD Petroleum in offering a fuel card to supporters and with Payless Energy, which donates funds as residential and business account holders switch their electricity needs.

The Foundation's Bequest Society is also gaining a greater awareness.

As the Foundation's profile builds, so does its ability to establish funding opportunities. That, in turn, increases the long-term capacity to identify and tangibly support world-class medical research.

The Foundation prides itself on its ability to identify and nurture research which, although highly scientifically worthy, does not attract attention from larger funding bodies. However, without the Foundation acting as a catalyst for this investigation to be established, more in-depth study simply wouldn't occur.

My thanks go to all supporters and friends of the Foundation. Your generosity and foresight in supporting the terrific work our scientists and researchers undertake is very much appreciated.

As I continually note at all Foundation functions and events - it is irrefutable that from medical research we all benefit.



Steve Davie
Director of Development

UNDER THE MICROSCOPE

Why do females have a higher risk of heart disease?

OMRF ANNUAL GRANT \$30,150

DR RAJESH KATARE & DR REGIS LAMBERTS
(Department of Physiology)

Title: Why do females have a higher risk of diabetic heart disease?

A team of University of Otago researchers, lead by Dr Rajesh Katare, are making ground breaking discoveries into why heart disease is the number one killer of people with diabetes.

Diabetes is a chronic metabolic disease and figures from the Ministry of Health in 2013, show almost a quarter of a million New Zealanders have been diagnosed with diabetes (mostly type 2), and it is estimated another 100,000 people are unaware they have the disease. Worldwide, diabetes affects more than 365 million people. Worryingly, rates are on the increase and that figure is expected to double by 2030. Unfortunately, people with diabetes are at risk of developing other health problems and recent studies show that at least 60% of people with diabetes die because of cardiovascular complications.

It is known that diabetes damages cardiac cells directly, reducing their ability to function and survive, ultimately leading to heart failure. However, until Rajesh Katare's research, the actual mechanisms that cause the extreme heart damage have long remained a mystery. Over the years, Rajesh and his team have identified harmful molecular changes in the cells of diabetic hearts that begin before the

cardiovascular symptoms even appear. This pioneering work has been published in internationally renowned journals in the field, including the International Journal of Cardiology (August 2015) and Cardiovascular Diabetology (April 2014).

Indian-born, Rajesh said he was inspired to undertake research in this field following the sudden death of his father in 2008, who had diabetes. "When you start doing the research, you always want to save as many people [as you can]."

"We are using diabetes as the disease model because people with diabetes are more prone to develop heart disease. But this therapy is applicable to any form of heart disease," Dr Katare said in a recent interview with the Heart Foundation (<http://www.heartfoundation.org.nz>)

The OMRF is delighted they have been involved in the innovative research of Dr Rajesh Katare by awarding an annual grant in 2012 to himself and Dr Regis Lamberts for a project titled: 'Why do females have a higher risk of diabetic heart disease?'

Cardiac complications of diabetes are prevalent in women and the damaging effects on the heart muscle begin especially early in females. The project funded by the OMRF in 2012, used an established type-2 diabetic mouse model to analyse the changes of cardio-protective and -destructive genes at different stages of diabetes. Not surprisingly, Rajesh and Regis discovered a gender specific progression of diabetic heart disease and in female cells there is a down-regulation of a pro-survival gene and subsequent protein. Although a mouse model is always used by Rajesh, for the initial research, he collaborates with cardiothoracic surgeons at Dunedin Hospital to collect and study heart tissue samples from diabetic coronary bypass patients, in order to confirm the laboratory based results. The findings from the OMRF funded study have provided vital information which in the future will be used to help develop gender specific treatments against diabetic heart disease.

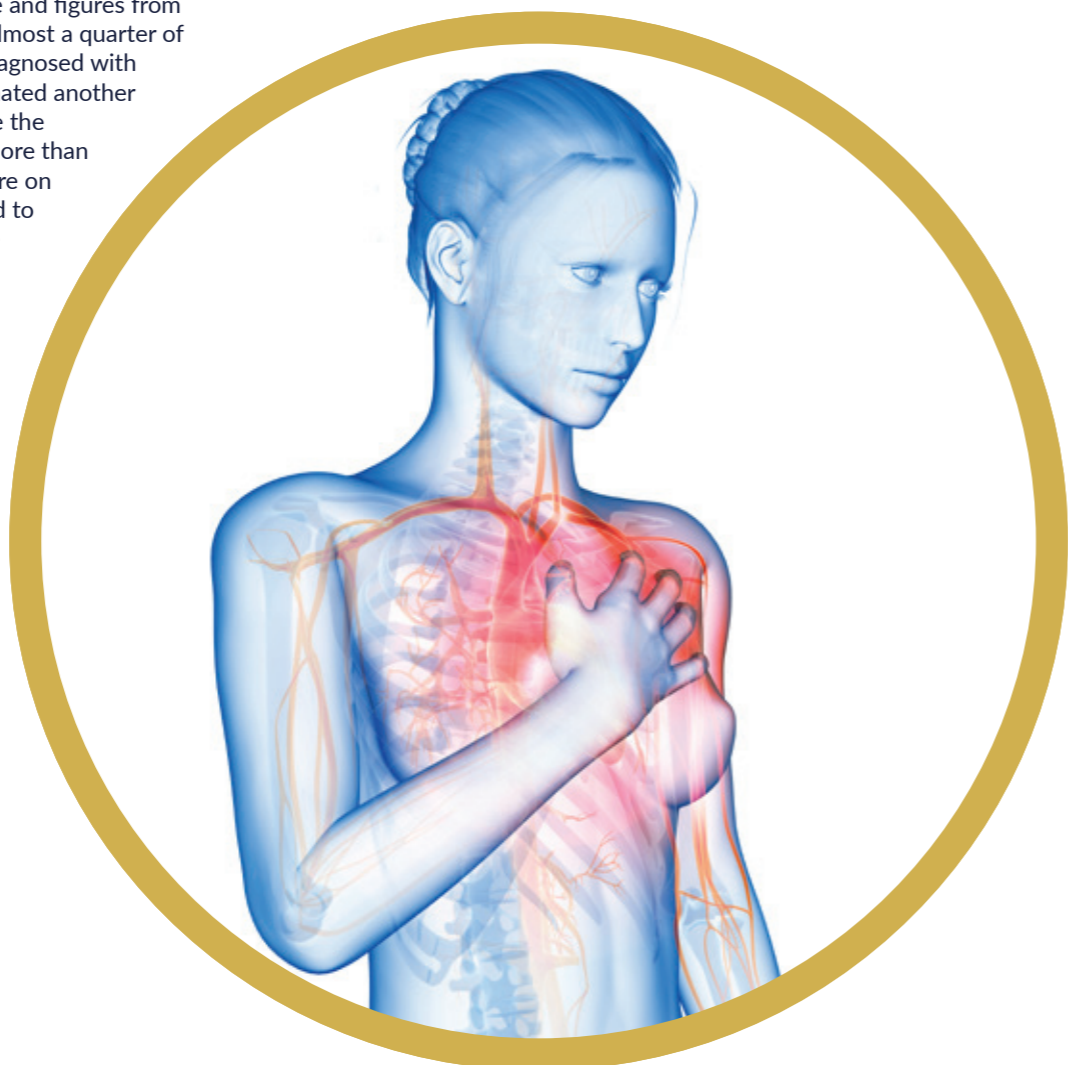
RAJESH AND HIS TEAM HAVE IDENTIFIED HARMFUL MOLECULAR CHANGES IN THE CELLS OF DIABETIC HEARTS THAT BEGIN BEFORE THE CARDIOVASCULAR SYMPTOMS EVEN APPEAR.

Rajesh has also had an OMRF funded summer student, Sophie Gandhi, working with him this year (MM & JH Hughes Family Trust Scholar). Because the harmful molecular changes to the cells happen early, before symptoms appear, it's crucial to find ways of detecting these changes. As part of her summer project, Sophie measured the levels of HDL cholesterol in blood from people with and without diabetes. Sophie discovered the levels of HDL were lower in diabetics and might therefore provide a marker for early detection of heart disease. Sophie also analysed levels of a small molecule called microRNA-34a, which is involved in regulating cardiac function. Rajesh is optimistic about the future possibility of non-invasive blood tests to determine the level of diabetic damage to the heart, by analysing levels of microRNA-34a, which Sophie found to be raised in the diabetic patients.

"Given that the growing diabetes epidemic is set to create major global economic and social costs in coming decades, it is very exciting to have opened up a new research avenue that could greatly decrease the disease's burden," Rajesh said in a recent interview in the Otago Daily Times.

"Doctors and patients would benefit from doctors being able to determine levels of markers in the diabetic heart, and this could help provide early warning of heart health risks, and could help doctors refine treatment options"

WORLDWIDE DIABETES AFFECTS MORE THAN 365 MILLION PEOPLE.



Long term relationships and having kids... a contributing factor to lowering the risk of prostate cancer and decreasing low cholesterol.

YAECHAN (DAVID) JU

(Associate Professor Nigel Dickson and Dr Claire Cameron, Department of Preventative and Social Medicine)

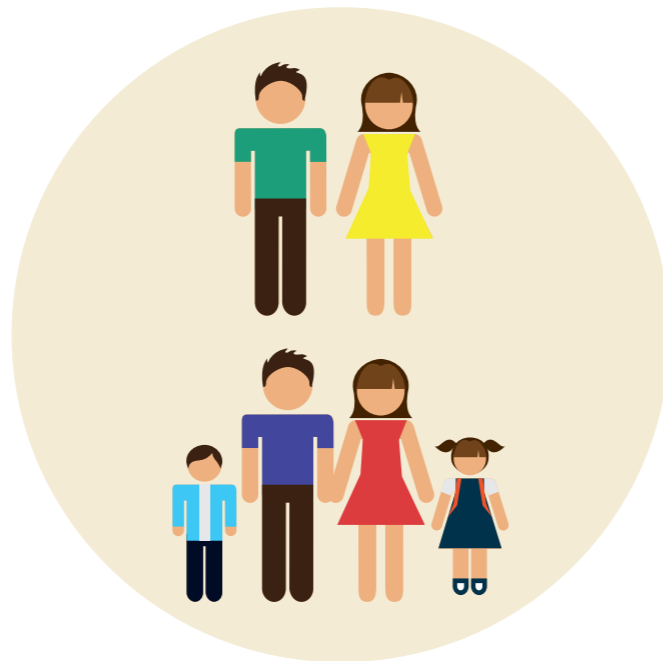
Title: The association of serum testosterone with relationship status and parenting at ages 26 and 38 in men: preliminary analyses

(Dunedin Casino/Media Works Otago Scholar)

Testosterone is well known as the hormone that makes men 'men', controlling male physical features like musculature and facial hair. Testosterone is also needed for behaviours associated with masculinity like risk, aggression and competitiveness, which are all important in the contest of winning a mate.

However, what happens after the women is won? That was the question being asked by summer student David Ju, who analysed the testosterone levels of men at ages 26 and 38 (data that has been gathered as part of the Dunedin Multidisciplinary Health and Development study). David discovered that at both ages, the testosterone levels were lower in men in long term relationships.

Humans are unusual as a species, in the fact we are bi-parental, with women and men both taking a part in childcare. As part of his summer project, David also investigated the impact of fatherhood on testosterone levels and found that



the men (at age 38) living in a household with children, had lower testosterone levels, compared with men living without children.

Although men might worry that this drop in testosterone is a bad thing, it's likely to have health benefits, including lowering the risk of prostate cancer and decreasing high cholesterol. So what better reason for a man to get married and have children!

Does weight loss improve vitamin D status?

SIMONETTE MALLARD

(Dr Lisa Houghton, Department of Human Nutrition)

Title: Does weight loss improve vitamin D status? A pooled analysis of weight loss trials and bariatric surgery studies

(Jan Warburton Scholar)

Known as the sunshine vitamin, vitamin D is produced by the body in response to skin being exposed to sunlight. It also occurs naturally in a few foods, including fish and egg yolks. Vitamin D helps the body use calcium from the diet and a deficiency is associated with soft bones. Interestingly, recent research indicates that too little vitamin D can also have other serious health risks including cardiovascular disease and cancer.

Vitamin D is stored in body fat and is released when fat is broken down for energy; therefore vitamin D deficiency is common in obesity due to a decreased use of body fat



and instead, increased storage. During her summer project, Simonette Mallard collected and collated data from weight loss intervention studies, in order to understand better the link between obesity and vitamin D status. This information will be used in further analysis and in the future might help to direct the research into a number of different conditions associated with both obesity and vitamin D deficiency, including: type 2 diabetes, hypertension, glucose intolerance and cancer.

Could beetroot bread reduce blood pressure?

LAUREN SHARP

(Dr Rachel Brown and Dr Katherine Black, Department of Human Nutrition)

Title: Effects of two types of beetroot bread on consumer acceptability, health markers and exercise

(OceanaGold NZ)

Here in New Zealand we enjoy our toast for breakfast and sandwiches at lunch and about 80% of the population eat up to six slices of bread a day. Some bread is already fortified with folate, so summer student Lauren Sharp wondered if our love of bread could be used as a way to provide other nutrients which might benefit our health. One such nutrient is nitrate, which is found in beetroot, and previous research has shown that eating beetroot can cause a reduction in blood pressure. Lauren hypothesised that adding beetroot to bread could be a novel way to improve health and she had participants of her trial eat bread containing beetroot juice or beetroot purée, before having their blood pressure measured. Although the participants enjoyed eating the beetroot bread, unfortunately there wasn't a drop in their blood pressure afterwards but further tests are being carried out to confirm this result. We might be waiting a little longer for the next best thing since sliced bread!



2014 Otago Community Trust Grant \$25,000

Professor Alison Heather

(Department of Physiology)

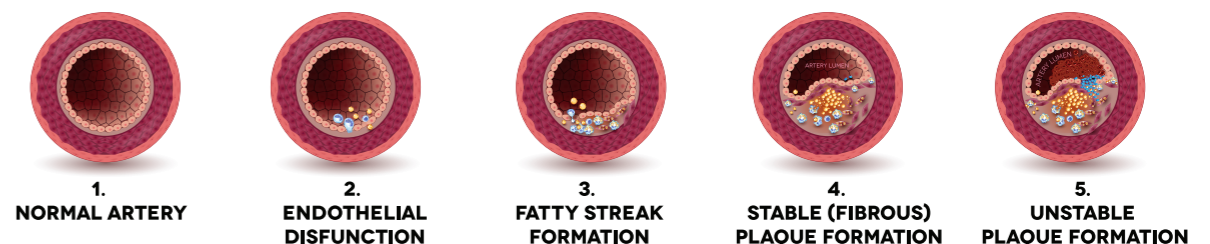
Title: Defining estradiol's bad effects on atherosclerosis: targeting safe HRT for women

Although most of us would like to 'stop the clock' and women especially are keen to try various anti-ageing treatments, getting older is unfortunately an inevitable part of life, and for women, so is the menopause. Due to today's longer life spans, women can expect to spend over a third of their life in their postmenopausal years. A few generations ago, women had to just accept hot flushes, mood swings, and the increase risk of osteoporosis, but the arrival of hormone replacement therapy (HRT) in the 1960s liberated millions of women from "l'enfer des femmes" (a woman's hell). HRT is known to have various health benefits, including prevention of dementia, reversing muscle ageing and improving bone density, but unfortunately it can also have an adverse effect on the heart.

Atherosclerosis is the build-up of fats on the walls of arteries and calcification of these atherosclerotic plaques, increases the risks of heart attacks and strokes, and it is a major medical problem affecting almost 60% of the population. Hormone therapies that increase estrogen in women (androgens in men), can increase levels of calcification, therefore increasing the risk of heart disease.

The important work being carried out by Professor Alison Heather with financial support from a Community Trust grant, administered by the OMRF, will investigate the mechanisms of increased calcification promoted by estrogen, so that more safe and successful hormone replacement therapies can be developed. This will allow women in the future to protect their hearts, while still having the health benefits of HRT.

ATHEROSCLEROSIS



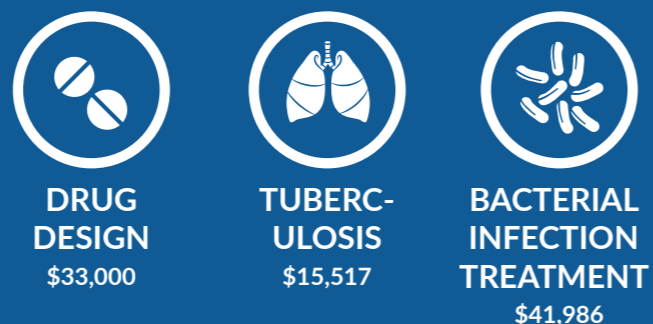
FUNDING BREAKDOWN

OF STUDENTSHIPS, GRANTS, TRUST GRANTS, LAURENSEN GRANTS AND JACK THOMSON GRANTS 1ST SEPTEMBER 2014 - 31ST AUGUST 2015

Summer Studentships



Annual Grants



Community Trust Grants



Laurenson



Jack Thomson



EVENTS

A Night to Remember 2015

Already seen as a 'must attend' event after just two years, the Foundation's annual black tie fundraising dinner - A Night to Remember - reached new heights in late-February.

More than \$75,000 was raised through sponsorships, ticket sales, auction items and raffle with donations received after the dinner lifting the overall amount to past the \$80,000 mark.

Funds generated go directly to the Foundation's work of establishing world-class, life-changing medical research in Dunedin ... for the benefit of tens of thousands of New Zealanders and millions more around the globe.

Our keynote speaker was the inspirational Tony Christiansen, who has packed more into his 56 years than most can dream of; magician and illusionist Andre Vegas amazed and, occasionally, shocked us; and Shane Cortese and his 8 Track Band had the dance floor packed in a two-hour non-stop grand finale.

Nine of the 2014/2015 summer research scholarship recipients were on hand to assist, auctioneer Rob Fowler's performance was a show in itself, and the Hi-Fives promotion gave two guests - lucky raffle ticket buyers on the night - the chance to vie for prizes worth almost \$150,000.

It was a terrific night of entertainment and bookings are already being made for the 2016 event, scheduled for late-February.

This year's naming rights' sponsor was Oyster Executive Recruitment which was joined by OceanaGold NZ (associate sponsor) and Armstrong Prestige, Volvo New Zealand, Air New Zealand, Forsyth Barr, House of Travel Dunedin, NZI, Vero Liability, QBE, Speight's Brewery and Misha's Vineyard as supporting partners.



GOLF: Foundation's annual golf tournament

After near-freezing temperatures 24 hours earlier, players in the fifth annual Foundation golf tournament on the St Clair course were greeted with a balmy 18 degrees in early-October.

The course was in magnificent condition and it rained birdies – and the occasional eagle – as the field enjoyed a fine start to the Spring charity tournament season, banding together to raise funds for the Foundation's annual research grants' allocation.

OceanaGold's naming rights' sponsorship extended into its fourth year and with strong support from hole sponsors and team entries, individual benefactors and prize sponsors, and through a generous uptake of the Mulligans promotion and raffle, a profit of just under \$18,500 was made on the day.

Those funds will be directed into the OceanaGold research grant, identified and allocated in May 2015. Money raised at the 2011 event was invested in a study into diabetic heart disease and why the occurrence of such is higher in women. The findings suggest that changes at cellular level appear much earlier in female diabetics compared to males, and the poor cell survival protein, Pim-1, also plays a major role in the deterioration of cardiac function in female diabetics. Restoration of the protein markedly improved the survival of female diabetic heart patients. Work is underway on the development of new strategies for the treatment of diabetic heart disease.

That is a major breakthrough as a result of the 2011 golf tournament.

In 2012 just under \$22,000 was raised and was directed towards investigation into a specific treatment for a particularly virulent prostate cancer where there is just a 25% survival rate five years after diagnosis. That research is ongoing.

The 2013 tournament generated \$18,000 which was directed towards an investigation into a specific bacterium that is a common cause of hospital-acquired infections and which is significantly resistant to antibiotics. This is an international study, which is gathering speed, and in collaboration with researchers in Italy where a compound has been discovered which inhibits these bacteria from causing infection.



Mark Cadzow, chief development officer of OceanaGold (NZ) Ltd – the tournament's naming rights' sponsor – celebrates his team's effort in finishing eighth alongside St Clair Golf Club's marketing executive Phillipa Calvert.



The winning team at the 2014 Otago Medical Research Foundation's annual golf tournament, staged in association with OceanaGold, pose with the champion's trophy: Brett McCormack, Greg Flannery, Chris Idour and Will Hepburn.

Supporting the OceanaGold commitment were our hole sponsors and the Foundation acknowledges their enthusiasm. Our thanks to Silicon Coach, Orbit Corporate Travel, Dr John Greaves and Keith Newton (Mornington Health Centre), Dr Alan Wright, Dr Patrick Dawes, Dr David Peart and Mr Andrew Swan (all Marinoto Clinic), Forsyth Barr, Deloitte, Palmers Mechanical, Sport Otago, Southern Colour Print, Craigs Investment Partners, the Hong Kong and Shanghai Banking Corporation, Armstrong Prestige and GJ Gardner Homes Otago.

Our appreciation is also extended to our prize and refreshment sponsors, and others who played a part in the success of the day: Dr Brian McMahon, Dr Jenny McMahon, Aravin Central Otago, Dunedin Venues, Watty! New Zealand, Rialto Cinemas (Dunedin), Cadbury Confectionery, Henry's Beer Wines & Spirits, Rockburn Wines, Gardens New World, Craft Bar, Otago Cricket, Luna Bar & Restaurant, Scotia Bar & Bistro, Mitchells Tavern, Opus International, HSBC, Dunedin Casino, Perseverance Estate, McDonald's Dunedin, Brittain Wynyard and Armstrong Prestige.

There were also a number of team entries and their support was also appreciated: Ken & Liz Dempster, Whatsoever Ltd (Dave Sharp), Lab Supply Ltd (Adam Spurway), Mr Simon McMahon and Steve Davie's Tuesday afternoon golfing mates representing the Foundation.

The day's results were:

Closest to the pin – 4th; Luke Murdoch, 7th; Alan Nichols, 13th; Alan Carne, 16th; Luke Murdoch, 1st (with the second shot); Robin Bates

Straightest drive – 11th; Dan Gin

Team results:

- 1st playing off a team handicap of 5.75, net score of 50.25 – GJ Gardner Homes Otago (13 birdies and an eagle off the tee)
- 2nd 6.5, 53.5 – Otago Medical Research Foundation
- 3rd 7.25, 54.75 – Mornington Health Centre
- 4th 8.25, 55.75 – OceanaGold # 1
- 5th 5.75, 56.25 – Whatsoever Ltd
- 6th 4.625, 56.375 – Palmers Mechanical
- 7th 9, 57 – Dr David Peart, Mr Andrew Swan
- 8th 10.87, 57.13 – OceanaGold # 2
- 9th 4.75, 57.25 - Deloitte
- 10th 6.5, 58.5 – Craigs Investment Partners

CLUB OTAGO

Established in early-2012, Club Otago continues to bring together the very best in speakers, camaraderie and charity.

The lunch club has been enthusiastically embraced by the region's corporate sector and individual enthusiasts alike and is a high profile component of the Foundation's calendar of events.

Four lunches are hosted each year with these featuring the topical speakers of the moment. All funds raised are directed towards the Foundation and its on-going mission of identifying and nurturing world-class medical research in the city.

The Club's speakers to date have been All Black coach Steve Hansen; broadcaster Keith Quinn; former doctor and now successful businessman David Kirk; Sir Peter Leitch (the 'Mad Butcher'); the inaugural captains of the Highlanders and Warriors, John Leslie and Dean Bell, as Dunedin celebrated the hosting of a Super Rugby/ National Rugby League double; outspoken businessman Sir Bob Jones; New Zealand cricket coach Mike Hesson; new

Dunedin Venues boss Terry Davies; UK-based brain researcher and science communicator James Piercy; head of New Zealand Cricket World Cup team Theresa Walsh; and former New Zealand Police Commissioner Howard Broad, now Deputy Chief Executive of Security & Intelligence in the New Zealand Department of Prime Minister & Cabinet.

Funds raised in 2012 tallied \$53,000 with more than \$70,000 generated in each of the last two years through the Club's activities.



OUR MEMBERS ARE:

Patrons



Senior Fellow

Mercy Hospital

Fellow

Allied Press
Carpet Court Dunedin
Crombie Lockwood
Deloitte
Dunedin City Motors
Farmlands Co-operative
Fitzgerald Family Trust
Fulton Hogan
McMahon Investments
Orbit Corporate Travel
RD Petroleum
Stu Stevenson

Associate Fellow

Asteron Life
Body Synergy
Dunedin Casino
Dunedin Airport
Forays Consulting Ltd
Forsyth Barr
Harvie Green Wyatt
HSBC
Immersion Marketing
Jenepher Glover
Kiwibank
Living Corporation
Octagon Dental Suite
Opus International Consultants
Otago Cricket
Otago Orthodontics
Reid, Paterson, Scully, Cassidy syndicate
Richard Joseph & Associates
Sassanachs RFC
Seperex Nutritionals
SF Waller Family Trust
Southern Wide Real Estate
This Way Ltd

Individual

Sarah Anderson (Regent Theatre)
Peter & Paula Anstey (Progressive Plastics)
Martyn Ballantyne, John Larsen (Suits on Wall Street)
Hudson Biggs (Keogh McCormack)
Adam Binns (PRP Barlow Justice Binns Ltd)
Michael Bird (Storesafe Ltd)
Steve Brocklebank (PWC)
Paul Buckner (Downie Stewart)
Dave Callon (Share NZ)
Andrew Campbell (Wattyl NZ)
Andrew Carmody (Brooker Travel)
Bruce Carvell (Williams Signs & Graphix)
Grant Chirside (Southern Realty)
Garry Clarke (Arbi Monograms)
Malcolm Dore (Magoo Auto Dunedin)
Malcom Farry (Farry Group)
Dr Norman & Mrs Barbara Fitzgerald (NW Fitzgerald Family Trust)
John Freeland (AON, Mosgiel)
Adam Gain (Metro Realty)
Donna Gale (NZI)
Ross Gamble (Roslyn Storage)
Bill Haydon (Roman Catholic Diocese of Dunedin)
Ian Hogg (ANZ Bank, Commercial)
Murray Hughes (Aotea Electric Group)
Sharon Hyndman (Metro Realty)
Dr Rod Keillor (Keillor Ophthalmology)
Adam La Hood (Cook Brothers Construction)
Ron Lewis (Craigs Investment Partners)
James Lovelock (Webb Farry Lawyers)
Neil & Jamie Lyons (Signature Property Ltd)
Amy McFadzien (Cook North & Wong)
Stuart McLauchlan (GS McLauchlan & Co)
Mr Will McMillan (McMillan Medical Specialist)
Dave McPhedran (YBT)
Bill Marshall (ASB Commercial)
Nadene Moore (International Freight Logistics)
Alan Nicholls
Simon Parker (Parker Warburton Team Architecture)
Mike Pearce (Strawberry Sound)
Russell Quin (Quintessentially Financial Services)
Jules Radich (ActionCOACH)
Richard Roberts (Dunedin Airport)
Sergio Salis (London Street Specialists)
Sarah Saunderson-Warner (Barrister & Solicitor)
Dr Michael Schultz (Gastroenterology Otago Ltd)
Carl Spruyt (10X)
Justin & Etere Stonelake (McDonald's Dunedin)
Peter Taylor (Peter J Taylor & Associates)
Dr Paul Templer (Sandman Anaesthesia Services)
Mark Thompson (Thompson Accounting)
Barry Timmings (Timmings Partners)
Chris Timms (Craigs Investment Partners)
Michael Turner (Polson Higgs)
Sherman Weatherall (Agility Logistics)
Tom West (Tom West Risk Advisors Ltd)
John White (Telfer Electrical Otago Ltd)

CHAIRPERSON'S REPORT

47TH
ANNUAL REPORT
YEAR
2015

\$398,214
Decrease of
\$25,751
Since 2014

Total amount funded
Since the Foundations inception
\$7,621,213

The extract from the Financial Statements, as published elsewhere in the Annual Report, shows a surplus for the year of \$91,251 which is \$113,363 less than last year but as noted in my report last year, last year's result was boosted by Bequest income and two Evenings to Remember falling in the same financial year. In the financial year under review we only had a small bequest and one Evening to Remember, which, once again, proved very successful. The Foundation endeavours to invest surpluses in project grants rather than build up funds but further injections of capital for investment are vital if the Foundation is to continue supporting research at, at least the same rate that we have been.

The Investment Sub-Committee has continued to face the challenge of finding suitable low risk investments while acknowledging that income and growth are also important. It is pleasing to report that at balance date, the market value of our Company Securities and Shares shows an unrealised gain on cost of \$556,272, which is 14% of cost, with the New Zealand and Australian investments being the main contributors.

At March 31 2015, Accumulated General Funds total \$486,474, and Accumulated Special Funds \$4,383,311, both these figures comprising Capital and Income.

This year marked the 18th year in which the Otago Community Trust awarded an Annual Grant to the Foundation with the details of grants awarded from this year's funding being published in the Scientific Committee Report. This brings the total grants received from the Otago Community Trust to \$1,281,000 a truly generous contribution. On behalf of all members of the Foundation and all Researchers based in Dunedin I would like to sincerely thank the Otago Community Trust for their very generous, and much needed, contributions.

The Foundation is deeply indebted to those people who have named the Foundation as a beneficiary in their wills. Medical research is a never ending activity and the role of the Foundation will continue as long as there are medical scientists willing to ask critical questions and people willing to help fund these researchers in their quest for the vital answers. I would ask members to consider the Foundation when preparing their wills. A bequest to the Foundation will be effectively used and your influence will be felt beyond your lifetime.

Council Membership

Since my report last year the following changes have occurred in the membership of Council. Prof Vernon Ward, the new Dean of the Otago School of Medical Sciences, along with Assoc Prof Joel Tyndall, President of the Otago Medical School Research Society and Assoc Prof Greg Jones, Deputy Chair of the Scientific Committee are all new members, Greg being co-opted. We welcome Vernon, Joel and Greg and farewell Assoc Prof Colin Brown and thank Colin for his contribution to Council as President of the Otago Medical School Research Society.

Thanks

- Firstly, to all those Trusts, Companies, Individuals, Members and Non-Members listed in this Annual Report who have supported the Foundation in the year under review. The Foundation is very grateful that it has continued to receive the support that it has in these difficult economic times.
- To Steve Davie, our Director of Development, for yet another year of commitment and belief in the work of the Foundation, continuing to raise funds for our work, and, just as importantly, continuing to raise the profile of the Foundation. Steve's report can be found on page 5.
- To my fellow Investment Sub-Committee members, Mike Horne, Ron Lewis and Jenny McMahon for their wise counsel, advice and time so willingly given to serve on this Sub-Committee, I thank you most sincerely.
- Once again, we must acknowledge the contribution of the Scientific Committee and special thanks must go to the members of this Committee, under their longstanding and dedicated Chairperson, Associate Professor Patricia Cragg. Although all busy with their own career activities, the Scientific Committee still continue to find the time to provide professional assessment and advice on applications submitted for funding. The time spent by this Committee

on the assessing and recommendations to Council is greatly appreciated. Without this group of dedicated people we would not be able to achieve the object of the Foundation, "The Furtherance of Medical research in Otago".

- To all Council Members, for your contribution and support, my sincere thanks for your continued interest in, and work done, for the Foundation.
- To the Deloitte team of Mike Horne, Megan Vintiner, Trudy Corbett and Luke Murdoch for continuing to provide very professional, friendly and efficient administration services for the Foundation.

On behalf of the Council
Ken Dempster
Chairperson



SCIENTIFIC COMMITTEE REPORT

1 September 2014 to 31 August 2015

1. MEMBERSHIP

Chair: Associate Professor Pat Cragg
(Nominee of the Otago School of Medical Sciences)

Deputy Chair: Associate Professor Greg Jones
(2014 Nominee Otago Medical School Research Society)

Professor Antony Braithwaite
(2014 Co-opted);

Dr Heather Cunliffe
(2015 Co-opted)

Associate Professor Colin Brown
(until September 2014 President Otago Medical School Research Society)

Dr Tamlin Conner
(2014 Co-opted);

Dr Damian Scarf
(2015 Co-opted)

Dr Peter Gootjes
(Nominee Otago Branch of the NZ Medical Association)

Associate Professor Bob Hancox
(Nominee Dunedin School of Medicine)

Dr Nick Heng
(Co-opted)

The Scientific Committee is primarily concerned with adjudicating on applications for Research Grants and on applications from students for Summer Research Scholarships. To cover the breadth of topics submitted, the committee is relatively large to ensure it has representatives from all the major sub-disciplines of medical research.

At the end of 2014 there were three retirements from the committee: Professor Antony Braithwaite, Associate Professor Colin Brown and Dr Tamlin Conner who joined the committee, respectively, in June 2013, May 2013 and March 2008. All have provided excellent input to our deliberations and in particular we thank Tamlin for 6 years of exceptional service. For 2015 we welcome Dr Heather Cunliffe, Dr Gill Johnson and Dr Damian Scarf as a co-opted members from the University of Otago representing the Department of Pathology, School of Physiotherapy and Department of Psychology respectively. We also welcome Dr Reuben

Dr Gill Johnson
(2015 Co-opted)

Dr Reuben Johnson
(2015 Nominee Otago Medical School Research Society)

Dr Joanna Kirman
(Co-opted)

Dr Beulah Leitch
(Co-opted)

Associate Professor Russell Poulter
(Co-opted)

Associate Professor Ivan Sammut
(Co-opted)

Dr Paula Skidmore
(Nominee Otago Medical School Research Society)

Associate Professor Joel Tyndall
(2014 Co-opted) (since October 2014 President Otago Medical School Research Society)

Professor Rob Walker
(Co-opted)

Johnson, from the Department of Surgical Sciences, as the nominee of the Otago Medical School Research Society replacing in this role Associate Professor Greg Jones who however continues as the Deputy Chair.

Note: Most, but not all research projects, have protocols that require approval by the appropriate Ethics or Safety Committee prior to commencement of the research. Agreement by the Foundation to fund research projects is thus subject to receipt by the Chair of the Scientific Committee of a letter from the University of Otago's Animal Ethics Committee, Human Ethics Committee or Human Ethics Committee (Health) (or the Ethics Committee of a Health Funding Authority) indicating that the research has received full ethical approval. Work involving genetically modified organisms requires evidence of approval from ERMA or from the University of Otago's Institutional Biological Safety Committee.

The scientific activities of the Foundation (advertising of up-coming grants and listings of awards) can be found on the following web site <http://www.omrf.org.nz>

2. SUMMER RESEARCH SCHOLARSHIPS 2014/2015

One hundred and thirteen applications (compared with 102 the previous year) for an OMRF summer research scholarship were received from the University of Otago in late August 2014, of which 22 were recommended for funding by the OMRF (and at least 62 of the other applicants gained scholarships from other funding bodies or the Division of Health Sciences and its Schools). Of the 22 students funded by the OMRF, two were studying dentistry, eight medicine, one pharmacy and eleven science or biomedical science. It should be noted that the ten-week summer research is not part of the study required in a student's tertiary qualification and any data obtained during the summer research cannot contribute to the dissertation or thesis of such a qualification.

Each scholarship was worth \$4,000 except for the two students with the highest scores who were awarded named Summer Research Scholarships (\$5,000) – named in honour of the late Allan Wilkinson and the late Emeritus Professor Garth McQueen. Allan was Secretary of the Foundation from its inception in 1967 until his retirement in 1993 and Garth was a foundation member of the Foundation and one of the instigators of the formation of the Foundation's Auxiliary. One of the projects was funded from the Foundation's Iverach Fund and another was administered by the OMRF but sponsored by the Otago Diabetes Research Trust.

Due to the continuing sponsorship drive of the OMRF, all the other 18 OMRF scholarships were funded by: Ailsa Goulding, Crowe Horwath, Deloitte, Dunedin Casino/MediaWorks Otago, Fortune Theatre, Healthcare Otago Charitable Trust, Hughes Family Trust, Jan Warburton, Kingston Sedgfield Charitable Trust/ACE Shacklock Charitable Trust, Lions Club of Dunedin South, OceanaGold NZ (2), Otago Service Clubs Medical Trust, PricewaterhouseCoopers Foundation, Southern Trust, Southern Victorian Charitable Trust (2) and Southern Wide Real Estate. The involvement of Otago commercial companies and the Otago community for a fourth year in supporting summer research by tertiary students is much appreciated.

All scholars returned good to excellent reports by the end of February 2015. The Renshaw Prize (\$250) for the best report was awarded this year to: Alice McSweeney, who worked under the guidance of Professor Vernon Ward and Dr Zabeen Lateef of the Department of Microbiology & Immunology.

The following is a list of the summer scholars and summaries of the projects undertaken – additional information on these projects can be obtained from the Chair of the OMRF Scientific Committee or from the supervisor concerned.

ALICE McSWEENEY

(Professor Vernon Ward and Dr Zabeen Lateef, Department of Microbiology & Immunology)

Title: How does mouse norovirus exit from cells?

(Garth McQueen Scholar and Renshaw Prize Winner)

Noroviruses are well known pathogens but currently there is no treatment, vaccine or ability to easily grow the virus in cell lines. Mouse norovirus (MNV), however, can be propagated in the laboratory and was used in this project to investigate if exocytosis was a possible exit strategy for MNV. Inhibitors were used to block aspects of the exocytosis pathway and

the effects on intracellular and extracellular virus production analysed. No change in virus production was observed, demonstrating that MNV does not require the exocytosis pathways blocked by the inhibitors to exit cells.

EMILY BRIDSON

(Professor Iain Lamont, Department of Biochemistry)

Title: A possible mechanism for increased antibiotic resistance in the bacterial pathogen *Pseudomonas aeruginosa* isolated from the lungs of cystic fibrosis patients

(Kinston Sedgfield Charitable Trust/ACE Shacklock Charitable Trust Scholar)

The majority of cystic fibrosis adults are infected with the bacterium *Pseudomonas aeruginosa* which can cause life-threatening illness. *P. aeruginosa* antibiotic resistance is rapidly advancing, so monitoring antibiotic effectiveness is essential. *P. aeruginosa* possesses an ampC gene which provides resistance to certain antibiotics. This project investigated whether highly antibiotic resistant strains of *P. aeruginosa* carry multiple copies of ampC which could increase their resistance. Most strains investigated appeared to have a single copy of ampC while two strains may have multiple copies (further research would provide conclusiveness) and one non-clinical strain may have lost ampC altogether.

CHANTAL CHEN

(Professor Cliff Abraham, Department of Psychology)

Title: Minimising effort to maximise a memory mechanism

(OceanaGold Scholar)

In order to store information, neural synapses must be capable of regulated change. This memory-related plasticity, for example long-term potentiation (LTP), must be maintained within a functional range by metaplastic mechanisms. Previous studies have shown that single hippocampal neurons could be positively primed for increased future LTP via intracellular stimulation but not extracellular stimulation, possibly because bulk stimulation activates other types of priming. We hypothesise that hippocampal CA1 neurons can be extracellularly primed by applying MRS1754, an adenosine 2B receptor inhibitor which blocks negative priming. We found a trend supporting our hypothesis, although it was not statistically significant.

KHAI HOW (NICHOLAS) CHOO

(Professor Richard Cannon, Dr Ann Holmes and Dr Hee Ji Lee, Department of Oral Sciences)

Title: Which *Candida albicans* strains adhere to acrylic dentures?

(Lions Club of Dunedin South Scholar)

Candida albicans is the principal fungal cause of denture stomatitis - inflammation of mucosa beneath dentures. The ecology of *C. albicans* strains causing denture stomatitis is poorly understood. We used multilocus sequence typing to track changes in the *C. albicans* strains in the mouths of people following delivery of new dentures. We found *C. albicans* strains that were common to several individuals. Some people had more than one strain type. Some strains were maintained in individuals for up to six months despite renewal of dentures, others were acquired during the study. There was preliminary evidence for strain microevolution with time.

SAMUEL COSGROVE

(Professor Rob Walker, Department of Medicine, and Dr Dan Wright, Department of Pharmacy)

Title: Metformin dosing in patients with impaired kidney function: a pilot study

(Otago Service Clubs Medical Trust Scholar)

Our aim was to evaluate the renal clearance and dosing of metformin in subjects with reduced kidney function. Metformin 500 mg and gentamicin 40 mg (a marker of the kidney's glomerular filtration rate [GFR]) was given to six volunteers with impaired (2) and normal renal function (4). Blood and urine samples were collected over 24 hours. Metformin clearance was compared to estimates of GFR using linear regression. Metformin clearance was strongly correlated with GFR (gentamicin clearance, R2 = 0.94, P <0.01), and published eGFR equations (R2 >0.90). Thus our results suggest that a proportional dose adjustment based on estimated GFR is a reasonable dosing strategy for metformin in renal impairment.

NICOLA DAVIS

(Dr Jody Miller and Dr Lisa Houghton, Department of Human Nutrition)

Title: The impact of increasing iodine levels in bread on the iodine status and intakes of elderly New Zealand rest-home residents

(Ailsa Goulding Scholar)

In response to re-emerging iodine deficiency, in 2009, the NZ government mandated the fortification of breads with iodised salt. Iodine intakes and urinary iodine concentrations were determined for participants from a 2014 nation-wide survey of rest-home residents, allowing the impact of fortification on iodine intake and status to be examined. Fortification increased iodine intakes, from 66.6 µg/day to 97.3 µg/day (P <0.001). However, the post-fortification median urinary iodine concentration was 72.5 µg/L, indicative of mild iodine deficiency. Fortification of other staple foods or supplementation should be considered as additional interventions aimed at improving the iodine status of NZ's rest-home population.

SOPHIE GANDHI

(Dr Rajesh Katare, Department of Physiology)

Title: Blood based biomarkers for diabetic heart disease

(MM & JH Hughes Family Trust Scholar)

Diabetes is a chronic metabolic disorder associated with acquiring heart disease, commonly known as diabetic heart disease. Heart disease among diabetics develops at an earlier stage than in non-diabetics. Changes in the structure and function of the heart occur as a consequence of stages at the molecular level. The expression of circulating microRNA-34a (miR-34a) and high-density lipoproteins (HDL) cholesterol were measured to determine use as a biomarker for early detection of heart disease. Results demonstrated diabetic plasma samples to have an increased level of miR-34a compared to non-diabetic plasma samples. Furthermore, HDL concentrations were lower in diabetics than non-diabetics.

DANYON GRAHAM

(Associate Professor Brian Monk, Sir John Walsh Research Institute, Faculty of Dentistry)

Title: The effect of a second-site mutation on triazole resistance in yeast: A research project seeking to facilitate structure-directed antifungal drug design

(Crowe Horwath Scholar)

Fungal infections caused by drug-resistant fungi are a serious and growing public health concern. Mutations in yeast lanosterol 14α-demethylase (Erg11p) reduce susceptibility to triazole drugs, severely limiting therapeutic options. A *Saccharomyces cerevisiae* hyperexpression system was used to investigate the effects of two common, clinically relevant mutations (Y140H and I471T) in Erg11p. Cell-based analysis revealed that the Y140H + 1 471T double mutation conferred resistance to short-tailed but not long-tailed triazole drugs. The chemistry of the linkage between the triazole head group and the rest of the drug, together with a long tail, may stabilise the drug in the binding pocket and limit the desensitising effect of the double mutation. Improved design of long-tailed azole drugs may lead to a new generation of antifungals that will circumvent the resistance problem.

SAMUEL GRAINGER

(Dr Antje van der Linden, Department of Pathology, and Dr James Ussher, Department of Microbiology & Immunology)

Title: Can MALDI-TOF mass spectrometry be used to distinguish between methicillin-resistant and methicillin-sensitive *Staphylococcus aureus*?

(Southern Victorian Charitable Trust Scholar)

Rapid and accurate discrimination between methicillin-resistant *Staphylococcus aureus* (MRSA) and methicillin-susceptible *S. aureus* (MSSA) is essential for effective treatment and prevention of transmission. This project investigated the ability of matrix-assisted laser desorption/ionisation time-of-flight mass spectrometry (MALDI-ToF MS) to discriminate between MRSA, MSSA and the seven major MRSA strains found in New Zealand. The hierarchical analysis created from the mass spectral profiles showed that isolates were randomly distributed. Thus, we cannot recommend MALDI-ToF MS for discrimination of *S. aureus* isolates beyond the species level.

KATIE HOEKSEMA

(Dr Chris Baldi and Associate Professor Gerry Wilkins, Department of Medicine)

Title: Impaired heart function in type 2 diabetes

(J. A. Iverach Scholar)

Heart rate regulation and contractility are carefully modulated. In a pathophysiological system such as type 2 diabetes mellitus (T2DM), heart function is impaired and the heart is unable to meet the demands of exercise. However, the mechanisms underlying this impairment are not understood. We aimed to compare heart responses of T2DM patients to controls during pharmacological stimulation. Contrary to our hypothesis, no significant differences were seen in contractility, suggesting T2DM patients retain their ability to increase contractility and more extensive study is required to clarify the underlying pathology.

YAECHAN (DAVID) JU

(Associate Professor Nigel Dickson and Dr Claire Cameron, Department of Preventive & Social Medicine)

Title: The association of serum testosterone with relationship status and parenting at ages 26 and 38 in men: preliminary analyses

(Dunedin Casino/MediaWorks Otago Scholar)

Consistently serum testosterone (T) has been associated with relationship status in men, and in some studies with fathering. This was examined at ages 26 and 38 in the Dunedin Multidisciplinary Health and Development study. At both ages both total and free serum T were lower in men in long-term relationships; they were not associated with ever fathering a child, but were associated at age 38 (but not 26) with living with a child. Serum T was also associated with weight and smoking. Further analyses are planned to examine for independent effect of relationship status and of changes over time in individuals.

STENAR KIRS

(Mr Ahmad Taha and Dr Noelyn Hung, Department of Surgical Sciences, Neurosurgery)

Title: Metastatic brain tumours in a Dunedin cohort and their molecular markers

(PricewaterhouseCoopers Foundation Scholar)

Brain metastases are even more common than primary brain tumours and there are limited treatments available for them. This study aimed to identify possible clinical and molecular prognostic factors for this devastating disease. Although the study had a small sample size, modality of treatment and location of tumour were found to be prognostic factors in this cohort as well as MGMT (O6-methylguanine DNA methyltransferase) and CD163 (macrophage/activated microglia associated antigen) marker status. These factors should be considered when a decision is made about how aggressively a patient is treated particularly if the treatment threatens the patient's quality of life in their final days.

RICHARD LAMB

(Dr Bill Hawkins and Dr Eng Wui Tan, Department of Chemistry)

Title: Rational drug design: Covalent inhibitors targeted towards improving Alzheimer's treatment

(Southern Wide Real Estate Scholar)

Molecules containing reactive electrophilic centres are generally discouraged from being considered as potential drug candidates and often removed from screening libraries before biological evaluation. This central belief seems unfounded when considering the \$US33 billion/yr in worldwide sales generated by covalently binding drugs (including aspirin and penicillin). As a case study, we explored the rational modification of the current standard for treatment of Alzheimer's, that of using donepezil. Reactive electrophilic centres were incorporated into different analogues to improve its binding and selectivity, which could potentially improve the drug's half-life, cellular resistance, and dosing as well as its pharmacokinetic profile. Synthetic studies were carried out directed towards obtaining these compounds which will eventually be tested for their biological activity. The enone analogue of donepezil, where R = H, was successfully synthesised as well as donepezil itself. However analogues of donepezil, where R = Me and Et, were unable to be synthesised using an aldol condensation from the precursors.

LAURELLE LOCK

(Dr Greg Walker, School of Pharmacy and Professor Vernon Ward, Department of Microbiology & Immunology)

Title: Nanofibre delivery of next generation vaccines

(Healthcare Otago Charitable Trust Scholar)

A controlled release virus like particle (VLP) loaded with vaccine has the potential to improve the efficiency of immunisations. Nanofibre scaffolds could be used as a matrix to bind VLP as a controlled release platform. In this research we used an electrospinning technique to fabricate nanofibres with various properties known to bind VLP: polycaprolactone (PCL) for hydrophobic attraction, PCL-chitosan blend for ionic interaction and fucoidan coated nanofibres for sugar binding. Scanning electron microscopic analysis showed a stable nanofibre network had been fabricated for all formulations, with the hydrophobic PCL nanofibre appearing to have the highest affinity for VLP.

SIMONETTE MALLARD

(Dr Lisa Houghton, Department of Human Nutrition)

Title: Does weight loss improve vitamin D status? A pooled analysis of weight loss trials and bariatric surgery studies

(Jan Warburton Scholar)

To clarify the nature of the association between obesity and vitamin D deficiency, we undertook a meta-analysis of weight loss studies, aiming to describe the effect of decreasing body weight on vitamin D status. I developed a systematic literature search strategy, and from the 6766 articles identified, 95 met our inclusion criteria. This number was greater than anticipated, which will increase our ability to detect a significant finding. Data extraction sheets were developed and completed. Upon completion of duplicate data extraction by a second researcher, I will in future perform the meta-analyses and draft a manuscript for publication in 2015.

ADRIENNE MORALES

(Associate Professor Ruth Empson, Department of Physiology)

Title: Reducing mGluR1 hyperactivity in a mouse model of spinocerebellar ataxia type-1 – helpful or harmful? A behavioural study

(Fortune Theatre Scholar)

Spinocerebellar ataxia type-1 (SCA1) is a disorder that destroys the cerebellum - the part of the brain crucial for executing co-ordinated movements. Previous research has found hyperactivity of cerebellar metabotropic glutamate receptors (mGluR1) in the early stages of SCA1. My experiment aimed to answer the question whether this hyperactivity is helpful or harmful. I found that acute pharmacological blockade of mGluR1 improved the motor performance of SCA1 mice suggesting that mGluR1 hyperactivity may herald the onset of harmful cerebellar destruction. Our findings suggest that moderation of mGluR1 may be a good way to treat the early symptoms of human ataxia.

HAZEL NISSEN

(Dr Regis Lamberts and Dr Carol Bussey, Department of Physiology)

Title: How does diabetes affect the microcirculation of the heart?

(Otago Diabetes Research Trust Scholar)

Diabetes Mellitus (DM) is strongly associated with cardiovascular disease and is escalating worldwide. DM-induced disease of small heart vessels, the coronary microvessels, contributes to increased cardiovascular morbidity and mortality in diabetic patients. However, our limited ability to directly measure coronary microcirculation restricts progress in our understanding of DM heart disease. I aimed to establish an innovative tool, vascular casting, to measure coronary microvascular perfusion and investigate how type 2 DM impairs this. My study supports the validity of this technique, demonstrates its potential to enhance our understanding of coronary microcirculation in DM, and generates novel opportunities for future cardiovascular research.

PHILIPPA ROSS

(Dr Ben Wheeler, Department of Women's and Children's Health)

Title: Incidence and characteristics of insulin-pump adverse events in New Zealand children and adults

(Deloitte Scholar)

Insulin pumps are widely used in the treatment of type 1 diabetes mellitus (T1DM). Perhaps because of their popularity, there have been few recent studies considering the adverse events (AE) associated with their use. This study sought to describe the incidence and characteristics of insulin pump-associated AEs in New Zealand children and adults with T1DM, using a self-report retrospective questionnaire covering the previous 12-month period. We found a high rate of AEs of all types, with set/site problems most commonly reported. As AEs appear common, anticipatory patient education may therefore be important in order to minimise AB-associated complications.

LAUREN SHARP

(Dr Rachel Brown and Dr Katherine Black, Department of Human Nutrition)

Title: Effects of two types of beetroot bread on blood pressure, endothelial function and consumer acceptability

(OceanaGold Scholar)

Bread is a dietary staple of the New Zealand population, making it an ideal vehicle to provide nutrients known to benefit health. One such nutrient is nitrate found in beetroot. In this randomised, cross-over trial, subjects consumed bread containing beetroot juice or beetroot puree. Then blood pressure, endothelial function and gastro-intestinal acceptability were measured over a four-hour period. The beetroot bread was well accepted by participants, however there was no reduction in blood pressure or endothelial function. Fifteen more participants will be recruited to strengthen these findings and determine whether an association is present.

ANTHONY SHAW

(Professor Parry Guilford and Associate Professor Mik Black, Department of Biochemistry)

Title: Detecting tumour cells in urine as a potential diagnostic test for prostate cancer

(Southern Victorian Charitable Trust Scholar)

Current tests for early detection of prostate cancer have relatively low effectiveness, often only being able to detect

late-stage cancers, or in the case of PSA testing having high false positive rates, leading to unnecessary biopsies and potentially serious complications. Single cell sequencing provides the ability to identify urine-based prostate tumour biomarkers, which may offer improved diagnostic performance. By analysing data from single cell sequencing experiments we attempted to identify genes that are only active in prostate tumour cells, and accurately estimate the number of transcripts in single cells to help optimise the development of an improved urine-based diagnostic test.

HAYDEN SMITH

(Professor Warren Tate, Department of Biochemistry)

Title: An investigation into the interaction partners of a protective, Alzheimer's disease-related, brain protein

(Allan Wilkinson Scholar)

An Alzheimer's disease-related protein has drawn great interest because it has been shown to be neuroprotective. My project intended to begin a study to look for the interaction partner(s) of secreted amyloid precursor protein alpha (sAPP α) in order to determine how it might mediate its positive effects on the brain. I carried out the first step by producing the complementary DNA (cDNA) for the gene of one potential protein partner, sortilin. I then inserted this into a larger DNA construct that can enter human cells in culture and express the protein, ready for interaction testing in the future.

NICOLAS THEIS

(Dr Keith Ireton, Department of Microbiology & Immunology)

Title: Purification of a bacterial protein essential for infection of human cells and its recruitment of human proteins to facilitate entry

(Southern Trust Scholar)

Yersinia species are food-borne bacteria that commonly cause intestinal disease. To establish infection, a protein on the surface of Yersinia, called invasin, interacts with a specific human receptor on the surface of intestinal cells. We aimed to purify invasin and introduce it to human cell cultures to observe its effects on human proteins associated with structural change. Introduction of invasin-coated beads to the surface of human intestinal cells resulted in recruitment of several proteins responsible for remodeling the cellular skeleton. This indicates that Yersinia invasin protein is capable of actively triggering structural changes in the human cell to facilitate infection.

3. RESEARCH GRANTS AWARDED

(A) ANNUAL GRANTS AND OTAGO COMMUNITY TRUST GRANTS

These one-year grants are for research concerned with human health and the scientific basis of medicine. In June 2014 there were 38 applications from the University of Otago (cf 43 the previous year) totalling \$975,468 and seven of these were funded at a total expenditure of around \$145,500 of which \$60,000 was provided most generously by the Otago Community Trust. These grants commenced between August and October 2014 and are nearing completion with full reports due 3 months after the one-year grant ends. Progress as at the end of May 2015 is summarised below:

(I) ANNUAL GRANTS

Dr John Ashton and Professor Rhonda Rosengren

(Department of Pharmacology & Toxicology)

Synergy in cytotoxic and targeted drug combinations for ALK+ lung cancer – AG 327

Sponsored by the JN Lemon Trust

Non-small cell lung cancer is the biggest cause of cancer deaths in the developed world. New drugs that target specific cancer genes have extended the life expectancy of nearly 10% of lung cancer patients. However, resistance develops to these drugs such that they are usually only effective for less than a year. We have been experimenting with combinations of anti-cancer drugs on lung cancer cell lines with a specific cancer mutation (in the “ALK” gene). Our original hypothesis, that crizotinib would show synergistic activity in killing these cells in combination with pemetrexed turned out to be false (although the two drugs did have additive activity). However, we did identify another drug combination - crizotinib with drugs that block the protein “IGF-1R” – that was extremely potent, with the combination of the drugs showing far greater effect than either alone. We are pursuing this line of investigation aggressively, and are using new (“CRISPR/Cas9”) gene editing technologies to understand this phenomena further – including manipulating closely related cancer drug targets such as “MET”. This work is now the basis for grant applications to NZ grant-funding bodies, as well as to the USA Department of Defence which has identified these themes as key research objectives. Ultimately, we aim to discover biomarkers that will identify potential responders to a range of drug combinations, and thus help extend the lives of lung cancer patients.

Dr Bill Hawkins

(Department of Chemistry)

A new paradigm in drug design – AG 328

Sponsored by Southern Trust

In the field of pharmaceutical development, there is currently an extreme bias to remove "reactive" functional groups from molecules. Yet some of our most pervasive pharmaceuticals (e.g. Aspirin and Penicillin) are dependent upon this reactivity. For these globally-utilised clinical drugs, their mode of action was either identified after development, or before current design practice. Today's policy of ignoring molecules containing reactive functionality is depriving clinicians of more effective pharmaceuticals (e.g. lower dosing, extended half-life). As a case study we incorporated a reactive centre into a clinically used drug. Preliminary data indicate that these structural modifications are well tolerated, with the original, desirable biological activity conserved. We are continuing to synthesise molecules with reactive functional groups and study the precise nature of binding and pharmacokinetics, to inform the development of more clinically effective pharmaceuticals.

Dr Joanna Kirman

(Department of Microbiology & Immunology)

Deciphering the memory T cell response to tuberculosis (TB) – AG 329

Sponsored by the JN Lemon Trust

The current vaccine against tuberculosis (TB) is ineffective and

we urgently need an improved vaccine. In order to develop a new vaccine we must understand the memory immune cells that drive protection against TB. Most new TB vaccines are being designed to stimulate memory CD4 T cell development; however these is no solid evidence to suggest that this is appropriate. For this project we tested which memory T cells are required for protection afforded by vaccination, by specifically removing these cells after vaccination and before an infectious challenge. We found that although CD4 T cells were important for the protection afforded by vaccination, interestingly CD8 T cells were equally as important for protection, and both T cell subsets were necessary for full protection. These findings will help inform future vaccine development. The findings from this research have been presented in seminars at the Centenary Institute (Sydney, Australia), the University of Oxford (Oxford, UK) and by oral presentation at the 4th Global Forum on TB Vaccination, held in Shanghai, China. A manuscript based on this project is in preparation. Our laboratory has greatly appreciated the support from the Otago Medical Research Foundation for this important project.

Professor Iain Lamont

(Department of Biochemistry)

Understanding and overcoming antibiotic resistance of Pseudomonas aeruginosa – AG 330

Sponsored by OceanaGold

Pseudomonas aeruginosa is a species of bacterium that is a very common cause of hospital-acquired infections and also infects patients with cystic fibrosis or who are immunocompromised. The bacteria become resistant to antibiotics during the course of prolonged infection, making infections difficult or impossible to treat. Collaborating researchers in Italy have recently identified a compound fluorocytosine that inhibits the bacteria from causing infection. The aims of this research are to understand how P. aeruginosa adapts during infection to have very high levels of antibiotic resistance, and how fluorocytosine bypasses antibiotic resistance mechanisms and inhibits bacterial infection. Bacteria resist antibiotics by activating genes that stop the antibiotics from working. We will compare the activities of all of the genes in a strain of Pseudomonas that is highly resistant to antibiotics with those of the genes in a strain that is killed by antibiotics in order to identify genes that collectively contribute to high-level resistance. We will determine how fluorocytosine works by comparing the total activity of all of the genes in bacteria grown with and without the compound. Genes that have altered activity in the present of fluorocytosine will reveal how the compound affects the bacteria. For both of these approaches we need to optimise the growth conditions for the bacteria to allow us to precisely address our aims and then prepare the samples for gene activity analysis by New Zealand Genomics Ltd.

Dr Robin Simmonds, Dr Heather Brooks

(Department of Microbiology & Immunology) and

Professor Kelly Doran

(Department of Biology - San Diego State University)

Role of a bacteriocin-like inhibitory substance in the pathogenicity of group B streptococci – AG 331

Sponsored by the Southern Victorian Charitable Trust

All three protein expression systems required for the project have been successfully developed i.e. one producing the

full-length endopeptidase, one producing a truncated protein corresponding to the predicted catalytic domain, and one producing a truncated protein corresponding to the predicted target recognition domain. Characterisation to date has been undertaken using the full-length endopeptidase protein which, as predicted, has been shown to have antibacterial activity against group B streptococci (GBS) and has been shown to have muralytic activity i.e. it hydrolyses the bacterial cell wall. Eighteen different GBS strains and three mutant GBS strains have been imported from our collaborators in San Diego, to ensure consistency in results between the respective laboratories. The three mutants consist of two GBS strains in which only the endopeptidase gene has been deleted and one GBS strain in which a global virulence regulator gene has been deleted. The pattern of activity of the endopeptidase protein against these 21 GBS strains has been established and the effect of the protein against cell wall material derived from each of these strains is currently underway. The development of a cell culture attachment assay is underway and preliminary results show clear differences in the levels of attachment of the three mutant GBS strains, both between themselves and their wild-type parent.

(ii) OTAGO COMMUNITY TRUST GRANTS

The Otago Community Trust supports biomedical research in the Otago area with the proviso that the research is selected on topics that can relate well to issues understandable by the layperson. The two projects selected were:

Professor Alison Heather

(Department of Physiology)

Defining estradiol's bad effects on atherosclerosis: targeting safe HRT for women – CT 332

The aim of this project is to define how hormone replacement therapy (HRT) can adversely affect calcification in arteries. In particular, the research is exploring how women are protected from the major heart disease, atherosclerosis, by the female sex hormones, estrogens, up until menopause. After menopause, HRT has a bad effect on the heart, increasing heart attacks and strokes. It is not clear why estradiol has these opposing effects and this is what the project is hoping to explain. Atherosclerosis is the formation of large fatty lesions in the arteries that eventually block blood flow causing a heart attack. From the work completed so far in an animal model, the project is showing that if estradiol therapy is started while these lesions are still growing, the therapy is protective. Estradiol slows down both the growth and the calcification of the lesion. In striking contrast, if estradiol treatment is started after the lesions have developed, then estradiol accelerates calcification. The findings, thus far, are important because they are providing clues as to what may be underlying some of the adverse effects of hormone therapies. As the project continues, the underlying mechanisms driving the injurious effects of estradiol will be determined.

Dr Julia Horsfield, Professor Mark Hampton

(Department of Pathology) and

Dr Joseph Antoun

(Department of Orthodontics – School of Dentistry)

A novel model for exploring the causes and treatment of craniofacial birth defects in children – CT 333

Cleft lip and cleft palate are common craniofacial birth defects.

The incidence of cleft lip and/or palate (CL/P) is 1/700 live births, and treatment over a lifetime can be very costly. The general view is that clefts are caused by a combination of genetic and environmental factors. In this research we want to determine how genetic and environmental causes of CL/P affect the growth and survival of cells contributing to the palate during embryo development. More importantly, we want to determine whether factors that enhance cell survival can actually rescue development of CL/P. The present study is being carried out in developing zebrafish embryos, which is an ideal animal model for these experiments. Catherine Carleton is studying toward a Doctorate of Clinical Dentistry in Orthodontics. Using the zebrafish as a model, she is determining if jaw and palate defects caused by an oxidative stress-inducing compound, auranofin, can be rescued by treatment with antioxidants. Auranofin mimics oxidative damage encountered in the environment. Together with Assistant Research Fellow Bryony Leeke, Catherine has shown that auranofin causes death of cells that will develop into the facial bones of zebrafish. Remarkably, an antioxidant called RiboCeine rescued the jaw defects caused by auranofin. Excitingly, RiboCeine rescued the effects of auranofin, even when applied after auranofin has already damaged cells. Future research in Catherine's project will uncover why RiboCeine works – we suspect it is protecting the cells from dying. Next, our research will address whether antioxidants can rescue genetic causes of jaw defects. This fundamental discovery-based research in zebrafish could eventually identify a potential for treatment with antioxidants to prevent CL/P in children, especially in families where there is an identified genetic or environmental risk.

(iii) RECENT ANNUAL GRANT ROUND

In June 2015 there were 33 applications from the University of Otago totalling \$794,644. Recipients of funding from the OMRF (\$98,000), and the Otago Community Trust (\$70,000) will be announced soon and the abstracts of the proposed work will then be available on the following web site <http://www.omrf.org.nz>

(B) LAURENSEN AWARDS

Laurenson Awards are one-year grants for research concerned with the effects of diet and/or drugs on human health. In December 2014 there were 18 applications (compared with 21 the previous year) from the University of Otago totalling \$466,532 and three of these were funded at a total expenditure of around \$93,000. Final reports are not due until the end of March to June 2016 or in one case September 2016, depending on the start date of the grant. Work in progress as at the end of May 2015 is summarised below:

Associate Professor Greg Anderson, Associate Professor John Reynolds and Dr Maggie Evans

(Department of Anatomy)

Hormonal restraint of hedonic eating behaviour – LA 337; commencing July 2015

Body weight regulation is usually ascribed to homeostatic mechanisms involved in stimulating and inhibiting food intake based on energy requirements. However, the role of hedonic eating (i.e. eating for pleasure rather than energy needs) in the promotion of food intake, and the neural mechanisms associated with reward that drive hedonic eating, have

recently emerged as active areas of obesity research due to their contribution to overeating and 'food addiction'. Perhaps not surprisingly, many of the same metabolic hormones critically involved in regulating the homeostatic drive to eat, such as leptin and insulin, have also been shown to play a role in modulating hedonic feeding behaviour. We propose to characterise the discrete functional roles of endogenous leptin and insulin signaling via the neuronal circuit of midbrain dopamine neurons that mediates reward, motivational, and hedonic mechanisms using transgenic mouse models in combination with metabolic and behavioural phenotyping.

Professor Sarah Hook

(School of Pharmacy)

Use of a dual COX/LOX inhibitor for treatment of cancer – LA 338

Non-steroidal anti-inflammatory drugs (NSAIDs) can be used to prevent and treat cancer. The aim of this research is to optimise the use of an NSAID in combination with a cancer vaccine to treat metastatic melanoma. Work so far has concentrated on examining the timing of administration of the NSAID in relation to the cancer vaccine, with initial results suggesting that reducing immune suppression at the time of administration of the vaccine has the greatest impact on anti-tumor responses. Ongoing work will investigate the route of drug administration, with oral delivery being preferred over an injection.

Professor Ian Morison and Mr Robert Weeks

(Department of Pathology)

The mechanism of action of glucocorticoids revealed through changes in DNA methylation – LA 339

Glucocorticoids such as prednisone have been widely used to treat a wide range of inflammatory disorders and cancer for decades. Surprisingly we have found marked changes in epigenetic marks on numerous genes in response to glucocorticoid treatment of a leukaemia cell line (an example is shown below). A small number of the top ranking changes have been assessed and the differences are yet to be confirmed by an independent method. Additional analysis of the epigenetic changes has been performed and further genes are being assessed for differences as proposed. In addition, other dexamethasone-sensitive blood-cancer cell lines have been imported and the effects of dexamethasone will be determined.

Associate Professor Ivan Sammut

(Department of Pharmacology),

Professor Robert Walker

(Department of Medicine),

Associate Professor Fiona McDonald and Dr Martin Fronius

(Department of Physiology)

The cardio-renal syndrome: targeting aldosterone inhibition to reduce cardiac and renal injury – LA 340

There is now increasing awareness that heart failure, particularly in the presence of hypertension, can produce chronic kidney injury, and combined kidney and heart disease has a worse outcome than heart failure alone. The key pathway mechanisms linking heart and kidney disease are not well understood, but research has indicated that by targeting

aldosterone receptors found within the vasculature we can improve outcomes in this syndrome. This Laurenson grant has allowed us to establish a model of heart failure in hypertensive rats, in which we are investigating the pathological developments in heart, kidney and vascular function and structure. Aiming to alleviate the pathology, we are using clinically available antagonists of aldosterone receptors to improve our understanding of this complex pathophysiology. This study, conducted by a collaborative group of researchers from Pharmacology, Medicine and Physiology, will provide vital insight into the use of targeted drug therapy to reduce the impact of poor cardiac function on kidney injury in the setting of chronic hypertension. The study is further supported by the key research contributions of Ms Catherine Leader, Dr Zoe Ashley and Dr Joanne Harrison. The benefits of preventing or reducing kidney injury extend beyond the kidney as the drug therapy would also decrease morbidity, and improve survival in patients.

(C) JACK THOMSON ARTHRITIS FUND

This OMRF fund was made possible by a bequest from the late Jack Thomson and commenced in 2011. For the fourth grant round in December 2014 there were three applications (compared with five in the previous year) from the University of Otago totalling \$71,756 and all three of these were funded at a total expenditure of around \$71,750. All grants commenced on 1 March or 1 April 2015 and final reports are due at the end of April or May 2016. Work in progress is summarised below:

Dr Melanie Bussey

(School of Physical Education) and

Dr Simon Stebbings

(Department of Medicine)

Can manual sacroiliac joint tests detect early signs of sacroiliitis in non-radiographic axial spondyloarthritis? – JT 334

Axial spondyloarthritis (AS) is the name for a family of inflammatory rheumatic diseases that cause arthritis in the axial skeleton. The term axial spondyloarthritis is now used to classify not only patients with ankylosing spondylitis but also patients presenting with inflammatory spinal pain associated with other forms of spondyloarthritis including reactive arthritides, psoriatic arthritis, enteropathic arthropathies, and undifferentiated spondyloarthritis. Chronic low back pain caused by inflammation in the sacroiliac joints is the early trademark of this disease that affects approximately 1% of New Zealand population. Recent advances on the treatment for AS have emphasised the importance of early identification of the disease. The advent of effective therapy, biological therapies or even continuous doses of anti-inflammatory drugs, has led to the suggestion that earlier diagnosis and earlier initiation of therapy may retard radiological progression and reduce disability. Our aim is to improve early diagnosis by validating a set of physical therapy tests of the pelvis that may be used by non-rheumatology practitioners to identify potential AS disease amongst chronic low back pain sufferers. To date we have obtained our HDEC (Health and Disability Ethics Committee) consent from the regional health authority. We have identified all 40 patients with non-radiographic axial spondyloarthritis (nr-AxSpA) whom we have recruited and initially screened from the rheumatology outpatient clinic

at the Dunedin Hospital. These participants are nr-AxSpA diagnosed in the last 2 years. While we have only just begun our testing we are currently seeing two participants per week, thus we aim to have all data collected from these participants by end of October. We will be presenting our findings at the World Congress on Low Back and Pelvic Girdle Pain on Dec 30th 2015.

Dr Daniel Cury Ribeiro and Dr Gisela Sole

(School of Physiotherapy)

Shoulder muscle activity: a study on patients with pain-limited shoulder elevation – JT 335

Shoulder disorders commonly cause pain during arm elevation and are a predisposing factor for shoulder osteoarthritis. Shoulder mobilisation decreases pain and improves function for up to a week, and our current research suggests that shoulder mobilisation leads to reduced activity levels in asymptomatic individuals. It is unclear what effect this technique has on patients with shoulder disorders. This two-treatment, crossover, participant-blinded, randomised trial will compare shoulder muscle activity between the baseline and post-mobilisation periods. Results from this study will inform the development of a novel treatment for shoulder rehabilitation.

Dr Prasath Jayakaran, Dr Meredith Perry, Dr Cathy Chapple, Professor David Baxter

(School of Physiotherapy) and

Dr Gareth Treharne

(Department of Psychology)

Early detection of hip/knee osteoarthritis to improve physical activity and self-efficacy – JT 336

Osteoarthritis (OA) of hip/knee is a painful condition which significantly impacts daily activities. Individuals with OA often resort to a sedentary lifestyle for fear of progression of the disease. Ironically, this can lead to development of co-morbidities such as obesity and set up a vicious cycle. Nevertheless, undertaking physical activity is a challenge in later stages of OA with significant pain and disability, reinforcing the fear of movement. The aims of this study are to identify individuals with early signs of OA in the community and pilot the effect of a physical activity intervention on the ability to self-manage the condition and exercise behaviour.

4. OTHER ACTIVITIES OF THE SCIENTIFIC COMMITTEE

OMRF Student Speaker Awards at the Otago Medical School Research Society:

The Student Speaker awards are given to the student speakers who, in the opinion of a panel of three to five judges, gives the best and second best oral presentation – based on both the components of the presentation and its scientific merit. To be eligible the candidates must report work that has been performed under the auspices of the University of Otago.

(1) At the September 2014 scientific meeting of the Otago Medical School Research Society (OMSRS) there were 10 doctoral candidates (selected from 25 applicants based on

their submitted abstracts). The first Prize (\$1,000) funded by Otago Postgraduate Medical Society was awarded to Sam Norton (supervised by Dr Ros Kemp, Department of Microbiology & Immunology) on the topic of "Colorectal tumour associated macrophages are more pro-inflammatory than adjacent control bowel tissue macrophage populations". The second prize (\$500), which was funded by the OMRF, was awarded to Nicole Neverman (supervised by Dr Stephanie Hughes, Department of Biochemistry) on the topic of "Identification and correction of prenatal synaptic pathologies in CLN6 ovine Batten disease".

(2) At the May 2015 scientific meeting of the OMSRS there were ten candidates (selected from 15 applicants based on their submitted abstracts). All were summer research scholars and four of the ten (and five of the 15) had been sponsored by the OMRF. The first prize (\$500) funded by the OMRF was awarded to Philippa Ross (supervisor Dr Ben Wheeler, Department of Women's and Children's Health, sponsored via the OMRF by Deloitte) on the topic of "Incidence and characteristics of insulin pump-associated adverse events in New Zealand children and adults with type 1 diabetes mellitus". The second prize (\$250) funded by the OMRF was awarded to Terry Zhang (supervisors Professor Margaret Baird, Professor Antony Braithwaite and Associate Professor Marilyn Hibma, Department of Pathology) for "Evaluating the immune modulating effects of microparticles from human papillomavirus type 16-E7 expressing keratinocytes on Langerhans cell-like cells".

The OMRF summer research prizes have been called, commencing this year, "The Pat Cragg Summer Scholar Speaker Prizes" in recognition of the long-standing involvement by Associate Professor Pat Cragg in the summer research scholarship assessing committee.

OMRF-sponsored Invited Speaker for the Otago Medical School Research Society:

The opportunity for such sponsorship occurred in September 2014 and was reported in the 2013/14 Annual Report.

OMRF-sponsored prizes at the Otago School's Science Fair:

The Foundation sponsors four prizes (\$50 each) each year in the Special Prize category at the Otago Aurora Science & Technology Fair for secondary schools for projects involving medically orientated topics. The August 2014 awards were reported in the 2013/14 Annual Report.

ACKNOWLEDGEMENTS

The Foundation continues to play an ever increasing role in funding Medical Research in Otago – may I thank the Scientific Committee for its dedicated efforts in the arduous, though satisfying, work of assessing the scholarship and merit of the many summer research projects and grant applications that it receives. We thank the Council of the Foundation for the support, advice and enthusiasm with which our funding recommendations are endorsed and the many Benefactors and Sponsors of the Foundation whose financial support has made all this possible.

- Associate Professor Patricia A. Cragg
Chairperson, Scientific Committee
30 June 2015

FINANCIAL HIGHLIGHTS

Otago Medical Research Foundation Inc.

This summary financial report has been authorised for issue by the Chairperson of the Council Mr Ken Deropster. The results presented in the summary financial report have been extracted from the full financial report for the year ended 31 March 2015. As such, this summary report cannot be expected to provide as complete an understanding as provided by the statements of financial performance, financial position and movements in equity of the Otago Medical Research Foundation Incorporated. A full copy of the audited financial report for the Otago Medical Research Foundation Incorporated for the year ended 31 March 2015 is available from the office of the Foundation's administrators - Deloitte, Otago House, 481 Moray.

Statement of Financial Performance

For the Year Ended 31 March 2015

	2015	2014
	\$	\$
Operating Income		
Donations, Bequests, Subscriptions	538,790	708,195
Investment Income	288,536	249,610
Profit (Loss) on Disposal of Investments	(16,811)	19,162
	610,515	976,967
Less Expenses		
Administration	83,418	68,485
Promotion Costs	237,632	279,903
Total Expenses	321,050	348,388
Net Surplus before Research Grants	489,465	628,579
Research Grants - Current year	398,214	423,965
Net Surplus for the year	91,251	204,614

Statement of Financial Position

As at 31 March 2015

	Market	2015	2014
		\$	\$
Current Assets		137,857	202,679
Investments	5,252,055	5,099,392	4,972,622
Total Assets		5,237,249	5,175,301
Current Liabilities		367,464	396,967
Total Liabilities		367,464	396,967
NET ASSETS (EQUITY)		4,869,785	4,778,334

Statement of Movements in Equity

For the Year Ended 31 March 2015

	2015	2014
	\$	\$
Revenue		
Net Surplus	91,251	204,614
Total Recognised Revenues and Expenses	91,251	204,614
Equity at the Beginning of the Year	4,778,534	4,573,920
Equity at the End of the Year	4,869,785	4,778,534

The full financial report of the Otago Medical Research Foundation for the year to 31 March 2015 was authorised for issue by the Chairperson of the Council. The full financial statements applied different reporting conventions. The auditor expressed an unqualified opinion. The summary financial report has been examined by the auditor for consistency with the full financial report. The auditor has expressed an unqualified opinion.

Deloitte



AUDITOR'S REPORT

INFORMATION ABOUT THE FOUNDATION

Charities Registration Number CC33444



REPORT OF THE INDEPENDENT AUDITOR ON THE SUMMARY FINANCIAL STATEMENTS

To the Council of the Otago Medical Research Foundation

The accompanying summary financial statements, which comprise of the summary statement of financial position as at 31 March 2015, the summary statement of financial performance and the summary statement of movements in equity for the year then ended and related notes, are derived from the full audited financial statements of the Otago Medical Research Foundation for the year ended 31 March 2015. We expressed an unmodified audit opinion on those financial statements in our report dated 30 July 2015. Those financial statements and the summary financial statements, do not reflect the effects of events that occurred subsequent to the date of our report on those financial statements.

The summary financial statements do not contain all the disclosures required for full financial statements under generally accepted accounting practice in New Zealand. Reading the summary financial statements, therefore, is not a substitute for reading the full audited financial statements of the Otago Medical Research Foundation.

Council's Responsibility for the Financial Statements

The Council are responsible for the preparation of a summary of the audited statements in accordance with FRS-43: *Summary Financial Statements*.

Auditor's Responsibility

Our responsibility is to express an opinion on the summary financial statements based on our audit procedures, which were conducted in accordance with International Standard on Auditing (New Zealand) (ISA (NZ)) 810, "Engagements to Report on Summary Financial Statements".

Other than in our capacity as auditor we have no relationship with, or interests in, Otago Medical Research Foundation.

Opinion

In our opinion, the summary financial statements derived from the audited full financial statements of the Otago Medical Research Foundation for the year ended 31 March 2015 are consistent, in all material aspects, with those financial statements, in accordance with FRS-43.

Crowe Horwath New Zealand Audit Partnership
CHARTERED ACCOUNTANTS
30 July 2015

Crowe Horwath
New Zealand Audit Partnership
Member Crowe Horwath International
44 York Place
Dunedin 9016 New Zealand
PO Box 188
Dunedin 9054 New Zealand
Tel +64 3 477 5790
Fax +64 3 474 1564
www.crowehorwath.co.nz

SUBSCRIPTIONS

Current subscriptions are \$30 per annum for Ordinary Members, \$100 per annum for Research Patrons (business firms or corporate bodies), and a minimum of \$500 paid by individuals, (\$1,000 for corporate bodies), applying as Life Members.

Although business firms are welcomed as Ordinary Members, in order to assist in expanding the work of the Foundation, they are invited to consider joining as Research Patrons or Life Members. The Foundation is an approved body for Income Tax purposes, and is registered for GST purposes. The taxation position in respect of donations and subscriptions is as follows:

COMPANIES

- From 1 April, 2008 a company making cash donations, or paying a membership subscription to any one donor may treat the amount as a deductible item for tax purposes up to the amount of their net income.

MEDICAL PRACTITIONERS

- Annual subscriptions - claim as a deduction.
- Donations - can be claimed as a rebate as for individual taxpayers.

INDIVIDUAL TAXPAYERS (INCLUDING FULL TIME SALARIED DOCTORS)

- All taxpayers are entitled to a rebate on subscriptions and donations in excess of \$5. Receipts should be attached to the Donations

Rebate Form in support of the claim. From 1 April, 2008 taxpayers are able to claim a 33.33% tax rebate on all donations up to their annual net income.

GIFT AND DEATH DUTIES

- No gift duty is payable by an individual on gifts to the Foundation.

REMEMBRANCE DONATION

- When you consider this substitute in place of a floral tribute, write or telephone the Secretary giving the name of the deceased, the relationship to the deceased, the relationship to the bereaved, and the name and address of the bereaved. A letter of condolence will be sent to the bereaved notifying them that you have made a donation in place of a floral tribute. An acknowledgement, with a receipt for your donation (which may be tax deductible), will be sent to you. This is a dignified and practical way of expressing your condolence, which is invariably appreciated by the bereaved.

MEMBERSHIP

- A form for membership application or donations is included within this report. Further information or brochures will be supplied on request to the Secretaries, Deloitte, P.O. Box 1245, Dunedin. Telephone (03) 474-8630.

LIST OF MEMBERS

ORDINARY MEMBERS

Prof W C Abraham	Dr P R F Gootjes	Dr H Nukada
Dr F J Austin	Prof A Goulding	Assoc Prof D Oorschot
Assoc Prof M A Baird	Dr S J Greaves	Assoc Prof D J Perez
Dr G Barbezat	* Ashburn Hall	Dr E L Phelan
Mr M G Bell	Dr R J Harvey	Assoc Prof J J Reid
Rev Dr John R Brinsley	** Mr J H Heslop	Assoc Prof A Rich
Mr John Burton	Dr M Hibma	Prof A M van Rij
Mr N A Carroll	Prof J Highton	S Saunderson-Warner
Caversham Pharmacy(2005)Ltd	Mrs L Homersham	Dr M Schlup
* Dr S O Chin	Mr M C Horne	Prof D C G Skegg
* Mr E J Chronican	* Prof J B Howie	Dr W Sutherland
Dr J I Clayton	+ Mr A K Jeffery	Mr M Thompson-Fawcett
Dr M Coleman	Mr & Mrs S D Jones	Dr M Turner
Dr A Cook	Dr R B Keillor	Dr & Mrs G P White
Assoc Prof P A Cragg	Assoc Prof I L Lamont	Dr S Wilbanks
Mr K G Dempster	Dr Liz Ledgerwood	* Mrs S M Wilkinson
Dr J M Faed	Mr R Lewis	Mr T J Williams
* Fairmaid Chance & Crawford	Prof A C B Molteno	Prof D Wilson
Mr M Farry	Prof J G Mortimer	Dr R A Wright
* Prof F N Fastier	Dr R Nada-Raja	Dr M E Wyatt
Prof W Gillett	Dr J Ng	Dr A I Yelavich

* Indicates Founder Member

+ Recently deceased

RESEARCH PATRONS

Respiratory Research Unit (University of Otago)
Hope & Sons Limited
Asthma Society Inc.

LIFE MEMBERS

Cadbury Confectionary Ltd	HealthCare Otago Ltd	Dr J A McMahon
Mrs J Callon	Dr R S Henderson	Northern Southland Transport Ltd
Cerebos Gregg Ltd Mr L Chronican	Janssen-Cilag Pty Ltd	Schering (NZ) Limited
Ciba-Geigy NZ Ltd	Lions Club Dunedin South	Roche Products NZ Ltd
Donaghys Industries Ltd	Ms S Mackinlay	St Margaret's College Council
Dunedin City Council	Mr D Marsh	Mr I A Thomson
Mr S Davie	Mr G J Marsh	Mr H R Wilson & Mrs N Ellis
Farra Dunedin Engineering Ltd	Mr W J Marsh	
Dr C M Goodall	Marsh Family Trust	

HONORARY LIFE MEMBERS

Mr & Mrs L J Brown	Rotary Club of St Kilda
Rotary Club of Dunedin South	Prof J I Mann
Mr G T Adams	Dr C N A & Mrs J Trotman
Mr P C L Gibson	

FUNDING PATHWAYS

ACE Shacklock Charitable Trust
Dr Ailsa Goulding
Barry Kloogh
Crowe Horwath
Deloitte
Dunedin Casino
Eastern Dunedin Charitable Club
Estate of Mrs SA Rowley (bequest)
Foodstuffs Community Trust (South Island)
Howard & Jane Fraser
Infinity Foundation
Jan Warburton
JN Lemon Charitable Trust
Kingston Sedgfield Charitable Trust
Lions Club of South Dunedin
MM & JH Hughes Family Trust
OceanaGold NZ Ltd
Otago Diabetes Research Trust
Otago Service Clubs Medical Trust
Oyster Executive Recruitment
Payless Energy
RD Petroleum
SpecSavers Dunedin
Southern Trust
Southern Victorian Charitable Trust
Zonta Club of Metropolitan Dunedin

These contributions are in addition to sponsorships and memberships associated with the annual dinner, Club Otago, the annual dinner and the like – as noted in specific reports.





OTAGO MEDICAL RESEARCH FOUNDATION
Annual Report to 31st March 2015 & Notice of Annual General Meeting