

breakthrough

Ellie

is living proof
of the power of
genomic testing

Could we teach
the heart to
repair itself?

Taking the fight
to autoimmune
disease



GARVAN
INSTITUTE
OF MEDICAL RESEARCH



Welcome from our Executive Director

Dear Garvan family,

It's with a great sense of positivity that I greet you in our final *Breakthrough* magazine for the year.

It has been a year of change and growth for many of us at Garvan, not the least me in stepping into this role. I would like to acknowledge and thank you all for your support throughout the year. With your help, we are leading the charge in a profound global transformation in medicine and healthcare.

In this issue of *Breakthrough*, we reveal the latest update on the Hope Research project, our ambitious approach to finding and treating the root cause of autoimmune diseases. You'll also meet little Ellie, who is alive today because of the incredible collaborative efforts of the Zero Childhood Cancer program. Read her story on page 6.

I'd also like to thank all of you who filled out our reader survey, as we strive to bring you the news and stories that you want to read. The overwhelming majority of you told us that you want to know more about our clinical trials, so in this issue we bring you an update on one of our recent human studies for an osteoporosis treatment, on page 12.

Please read on for updates on more of the successes and breakthroughs from the brilliant scientific minds made across Garvan over the previous few months.

We'd like to wish you and your family a wonderful end of 2018 and a healthy, happy new year.

With very best wishes,

Professor Chris Goodnow FAA FRS

Executive Director

The Bill and Patricia Ritchie Foundation Chair

Cover image courtesy of the Zero Childhood Cancer personalised medicine program, led by Children's Cancer Institute and the Kids Cancer Centre at Sydney Children's Hospital, Randwick.

NEW RESEARCH

Insight into the immune system

Our researchers have identified a new anatomical structure within the immune system – and it could help to make better vaccines. Using sophisticated high-resolution 3D microscopy in living animals, Associate Professor Tri Phan and his team identified where immune cells gather to mount a rapid response against an infection the body has seen before. This is the first time these structures have ever been seen, and they were uncovered by using technology developed by Tri and others at Garvan.

The researchers could see that several classes of immune cells gathered together in the new structure – including memory B cells, which carry information, or 'memories' about how best to attack the infection. The researchers could also see that memory B cells were changing into infection-fighting plasma cells. This is a key step in the fight against infection.

The new discovery is an important step in understanding the infection-fighting response that the body mounts to an attack it has been exposed to before. Understanding this anatomical structure within the immune system could help us to make better vaccines. The study was made possible by generous support from Peter and Val Duncan.

 Read more at: garvan.org.au/immune-structure.

Does fat drive diabetes?

Our researchers have discovered that, beyond the liver and the pancreas, one of the root causes of diabetes may lie in fat tissue. This could have important implications for the development of treatments.

Associate Professor Carsten Schmitz-Peiffer and his team have examined the protein PKCε, long known to be involved in the worsening of diabetes. In mice, removing PKCε from all tissue protects them from becoming diabetic. PKCε has always been assumed to be working in the liver, but upon removal in the liver alone, they found the mice were not protected.

Remarkably, they have now found that removing PKCε from fat tissue protects the mice from becoming diabetic – indicating that fat tissue may be playing a major role in the progression of this disease.

Currently, our team is collaborating with the Monash Institute of Pharmaceutical Sciences to try to develop an orally available peptide that can disrupt PKCε activity. Therapeutically targeting PKCε, and fat tissue, would be a brand new approach to treating diabetes.

 Read more at: garvan.org.au/fat-diabetes.



How do you mend **an injured heart?**

The problem: hearts can't fix themselves. Now, we've uncovered a huge amount of previously hidden information that may help us find ways to teach the heart to repair itself.

As humans, we have the capacity to repair much of ourselves, but not all tissues. For example, the skin, liver, skeleton and limb muscles can all regenerate. But unlike those tissues, the heart does not have the capacity for self-repair after damage (such as a heart attack). This is one reason why heart disease is the leading cause of death worldwide.

However, we may be able to help the heart heal itself. And the answer is likely to lie in understanding exactly how the heart builds itself in the developing embryo, one cell at a time.

Growing a heart from stem cells

Researchers from Garvan and the University of Queensland have just reported on the most in-depth study to date of exactly how human stem cells can be turned into heart cells. The work involved measuring changes in gene activity in tens of thousands of individual cells as they move through the stages of heart development.

To explore human heart development, the researchers mimicked, in the lab, how a heart develops in the embryo. They started with skin-derived human stem cells (from adults). These cells are capable of becoming any cell type in the body. They were able to guide the cells, over time, to become heart cells (cardiomyocytes).

"The development of the heart is like a tightly plotted novel – or an intricate dance," says Associate Professor Joseph Powell, head of the Garvan-Weizmann Centre for Cellular Genomics, who co-led the research.

"Each cell goes through its own series of complex, nuanced changes. They are all different, and changes in one cell affect the activity of other cells. By tracking those changes across the different stages of development, we can learn a huge amount about how different subtypes of heart cells are controlled, and how they work together to build the heart."

The information we've gained from this work positions us to take on new and bigger questions in cardiovascular disease. "We are now building on the knowledge gained from this work to investigate at what stages during heart development, and in what cell subtypes, the genetic risks of cardiovascular disease become most dangerous," says Joseph.

The researchers' next challenge is to find new approaches and insights into ways to help the heart repair itself.

You can help us to uncover the mysteries of the heart by making a donation at garvan.org.au/donate.

A tribute to Tracey Lea

In the hope that others will never suffer in the same way his sister did, Greg Goodley has become a Garvan Partner for the Future.



Greg Goodley remembers precisely the moment he found out about his sister Tracey Lea's teratoma tumour. He was 11 years old and she was 13. He'd seen her lying on her bed after school and insisted his parents tell him what was going on. "I knew something was wrong. She was in so much pain," Greg recalls.

Greg's memories of his sister are vivid, even though it is decades since her death. "She was the go-to person in our family, the peacemaker. When she was 12 she decided to become a journalist. At that age I was mucking around, making trouble, doing a bit of surfing, but she put her heart and soul into everything. She used to write long poems. They were beautiful."

Before she died, at age 16, Tracey Lea was misdiagnosed three times and went through rounds of chemotherapy that made no difference to her condition. Greg's hope is that no one else ever has to go through the same suffering.

"When she had chemo, she got sick but she never lost her hair. They'd told her

she would, so she'd bought a couple of wigs. Even though she didn't need them, she loved to dress up in those wigs. That's the kind of person she was."

Though he's still years from retirement age, Greg decided to plan ahead and leave a tribute to Tracey Lea in his Will. "If Tracey Lea had been diagnosed today, I know that her cancer could have been identified through the technology and expertise at Garvan. Perhaps one day, by the time my bequest reaches Garvan, they will be able to sequence everyone's DNA, or be able to identify the tumour and plan treatment for someone like Tracey Lea within an hour or two. I know the idea of that would have made Tracey Lea very happy."

If you would like information about leaving a gift to Garvan in your Will as a tribute to your loved ones, please contact Donna Mason, Bequest Manager, on (02) 9295 8559, or visit garvan.org.au/bequest.

PHILANTHROPY WITH STYLE

This November, we celebrate our partnership with iconic Australian pearl company Paspaley

Two years ago, Garvan and Paspaley formed a partnership committed to progressing Garvan's cancer research. Through donations from its Kimberley Bracelets, Paspaley provides considerable support to Garvan's pioneering clinical trials.

"Garvan's program has the potential to significantly improve the survival rate of cancer sufferers globally, now and for all future generations," says Chris Paspaley, Director of Merchandise, Retail at Paspaley. "This is world-changing and exciting. We feel privileged to be able to be part of it."

As an organisation, Paspaley has a strong commitment to philanthropy. "My grandfather and family received generosity from the local community that helped them establish their lives and led to the foundation of the Paspaley company," Chris explains. "I grew up hearing stories of my grandfather's generosity and focus on community. As a company, we share these same values today."

Paspaley generously donates to Garvan 25% of proceeds of sales from each of its unique Kimberley Bracelets. One of Chris's personal designs – a bracelet he had made in 2008 and still wears today – is the basis for the Kimberley Bracelet range. The mission of the range is just as close to his heart. "On a personal level, I find Garvan's work so inspiring: their leadership, vision and focus on developing a new future for medicine, while treating those who need it the most."

"The program has provided options for hundreds of patients with advanced stage rare cancers, for whom conventional treatments are no longer available," says Professor David Thomas, head of Garvan's Cancer Division. "With Paspaley's support, Garvan is leading the way in providing new therapies for cancer patients who have exhausted all other treatment options."



See the beautiful Kimberley Bracelet designs on [page 12](#).

Chris Paspaley, in the company's Sydney boutique, wears his original Kimberley Bracelet.

BREAST CANCER BREAKTHROUGHS

Some tumours have a pause button

When cancer cells break away from the primary tumour, they travel elsewhere and can grow into secondary tumours. New research from our Cancer Division has uncovered a natural process in breast cancer, in which some primary tumours can signal the immune system to follow the breakaway cells and 'freeze' them. In this 'frozen' state, the cells can't grow effectively – thereby stopping secondary tumour growth in its tracks.

"We know the response is a consequence of the tumour eliciting an immune reaction. We want to understand exactly what the tumour is releasing to activate this response, and how immune cells are targeting the secondary sites," explains Dr Christine Chaffer, who co-led the research.

The hope is that if we can exploit this signalling process in breast cancer, we may find controls that pause other types of cancer as well. Dr Christine Chaffer holds the Rebecca Wilson Fellowship in Cancer Research, funded by The NELUNE Foundation.



Read more at: garvan.org.au/pause-button.

Promising new breast cancer treatment

Our researchers have found a molecule that reduces the spread of cancer, slows tumour growth, increases sensitivity to chemotherapy and improves survival in mouse models.

Surprisingly, the potential treatment targets non-cancerous cells within breast tumours, instead of the cancer itself. The team found that triple negative breast tumours – which are the most aggressive and have the fewest treatment options – could be susceptible to an existing drug. It works by stopping tumour cells from 'talking' with nearby normal cells, which effectively stops the tumour from growing.

The collaboration between our researchers Dr Aurélie Cazet, Dr Mun Hui and Associate Professor Alex Swarbrick, the Centre for Cancer Biology (Adelaide) and GEICAM, Spain's leading breast cancer research group, was supported by funding from Love Your Sister, John and Deborah McMurtrie, the National Breast Cancer Foundation, RT Hall Trust and Novartis. The researchers will next explore whether this has potential to work on other cancer types that behave in the same way.



Read more at: garvan.org.au/breast-cancer-treatment.

Living Proof

The world-first Zero Childhood Cancer national personalised medicine clinical trial together with outstanding support from Lions mean that Ellie's story has a happy ending.

Two years ago, Mina and Rob gave birth to a beautiful baby girl – Ellie. All seemed well, yet only 11 months later, Ellie was admitted to the Sydney Children's Hospital, Randwick.

A scan revealed a tumour in her chest so large it was pushing her tiny heart and lungs to one side. Within days, she was on life support, no longer able to breathe. The tumour was aggressive, rare and resistant to chemotherapy.



Ellie's cancer struck when she was only 11 months old

Ellie's case was immediately referred to the Zero Childhood Cancer program, Australia's child cancer personalised medicine program. The world-first clinical trial, led by Children's Cancer Institute and the Kids Cancer Centre at Sydney Children's Hospital, Randwick, finds treatments for children with serious and aggressive cancers.

The race was on

The teams jumped to find out what was pushing Ellie's cancer to grow so large – and how it might be stopped. Through Genome Power (the Lions Kids Cancer Genome Project), Garvan scientists sequenced the whole genome of Ellie's tumour.

Then, at speed, they worked with the Zero Childhood Cancer team to zero in on the specific genetic change that was driving the cancer's growth. The Zero Childhood Cancer team were then able to identify a drug that targeted the particular genetic change.

The timing was just right. The drug had very recently been discovered, and the US company Loxo Oncology agreed to provide it on compassionate grounds, so treatment could begin.

Life-saving impact

Within four weeks of beginning treatment, Ellie's cancer had shrunk to a point where she could breathe on her own and no longer needed life support. And six weeks later, she was home. This targeted therapy also appears to be both more effective and less toxic than standard chemotherapy.

Now, a clinical trial of larotrectinib, the drug used to treat Ellie, is open in Australia for all children whose cancer is identified by the Zero Childhood Cancer program as having the same genetic marker. This means that the work done to save Ellie will help other children as well.

If Ellie had been diagnosed with this cancer even two years earlier, she would have died. She is only alive today because of the Zero Childhood Cancer collaborative program and the contribution of whole genome sequencing by Garvan, supported by Lions.

Ellie's case was immediately referred to the Zero Childhood Cancer Program.

One year since its launch, Zero Childhood Cancer has produced remarkable results for children like Ellie. Almost 130 children with serious and aggressive cancers have been enrolled. This state-of-the-art personalised medicine clinical trial aims to give them the best possible chance of survival and quality of life.

As Ellie's mum Mina explains, "We were told to think about saying goodbye, she was so sick we didn't even know if she would reach her first birthday. Today she is such an active and energetic two-year-old ... beyond our wildest dreams. We can't thank the teams at the children's hospital and research institute enough."



**If Ellie had been
diagnosed with this
cancer even two
years earlier, she
would have died.**

Ellie, now two,
with mum Mina
and dad Rob.

The Lions Kids Cancer Genome Project (Genome Power), primarily funded by the Lions Clubs International Foundation and the Australian Lions Childhood Cancer Research Foundation, is bringing whole genome sequencing and informatics capability at the Kinghorn Centre for Clinical Genomics (Garvan Institute of Medical Research) to all children with high risk cancer participating in the Zero Childhood Cancer program.

Genome Power is also supported by the John Brown Cook Foundation, the Vodafone Foundation and Lions Clubs across Australia.

Help children like Ellie

**You can help children like Ellie by
supporting medical research today.**

HOPE

Grows

Type 1 diabetes, multiple sclerosis, lupus, rheumatoid arthritis... this seemingly disparate group of conditions are all caused by the same disease process – autoimmunity. Garvan’s revolutionary Hope Research project is looking to find the root cause.

In 2017, we introduced you to Hope Research and asked you to help us make it a reality. Thank you – because today, it is.

Autoimmune disease is when the body’s own immune system becomes overactive and begins to attack itself. Hope Research is tackling the disease like never before, by getting to the root cause underlying all autoimmune disease. Our project has begun by hunting down the identity of the ‘rogue’ clones that cause 36 types of autoimmunity.

Hope Research is based on a premise first proposed by Garvan’s Executive Director, Professor Chris Goodnow, more than 10 years ago – that ‘rogue’ clones cause autoimmunity. At the time, he couldn’t prove his theory because the necessary technology didn’t exist. It now does, here at Garvan.

The project will, step-by-step, tackle a significant number of autoimmune diseases while simultaneously adding more to the list. “We’ve started with some of the most common disorders, and then we’ll add more,” says Dr Dan Suan, a team leader on the study who works with Professor Goodnow. “We’re starting with the diseases where we

already have a really clear idea of how to identify and isolate rogue clones.”

Dan explains that a research project this large couldn’t get off the ground without multiple funding sources. “We could not even consider embarking on this momentous task without many, many supporters.”

“Doctors around Sydney are identifying patients who will give us the best opportunity to find rogue clones.”

“We have funding from disease-specific research groups, such as Multiple Sclerosis Research Australia. We have philanthropic support from Garvan donors, many of whom have friends and family members affected by autoimmune disorders. And, we are funded by government through the National Health and Medical Research Council and other peer-reviewed funding through universities,” Dan describes.

How, what, where?

Hope Research is not a clinical trial, Dan explains. He doesn’t want to give those suffering from autoimmune disease unrealistic expectations. “Doctors around Sydney are identifying patients who will give us the best opportunity to find rogue clones,” he says.

“This study is difficult because we need to find patients with newly diagnosed autoimmune disorders. This gives us the best chance of catching the disease-causing rogue clones to then study in detail. Many patients have had their immune systems blasted by powerful drugs, so as a starting point we won’t be studying patients already on treatment.”

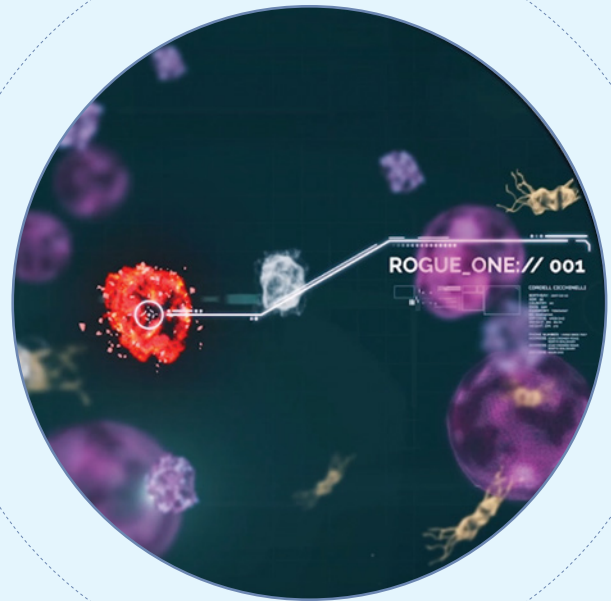
The rogue clones that cause these terrible diseases are floating around in your blood, but they’re mixed in among all the other normal cells of the immune system. Dan likens it to the proverbial needle in the haystack, “Out of a million completely normal cells, there might be five bad ones.”

However, technology has advanced to a stage that we can now find the rogues. Using our state-of-the-art

Hope Research is a flagship collaborative research project of the Garvan-Weizmann Partnership and is led by Garvan's Executive Director Professor Chris Goodnow, the Bill and Patricia Ritchie Foundation Chair.

The project has had philanthropic support from several families and foundations that have made significant donations: The Bill and Patricia Ritchie Foundation, Ken and Jill Allen, The Richard and Sue Croall Foundation and Scleroderma NSW.

You too can support the Hope Research project.
[Click here to make a donation on our website.](#)



equipment, the expert technicians in the Garvan-Weizmann Centre for Cellular Genomics can single out those five cells.

“Out of a million normal cells, there might be five bad ones.”

“When we have isolated the cells, we can probe them for unique weaknesses and use that new information to design personalised therapies,” Dan explains. “It’s the advent of single cell genomic technologies at an acceptable cost that has finally allowed us to undertake this world-first study.”

Many diseases, one at a time

The initial proof of concept has come via the group’s study of Sjögren’s syndrome. “Sjögren’s is a disease I’ve been working on for about 10 years now,” says Dr Joanne Reed, a group leader on the study.

“Using the cutting-edge technologies in the Garvan-Weizmann Centre to look at an enormous number of parameters in a single cell, we first were able to find these cells we call rogue clones in a patient with Sjögren’s.”

From the new understanding of the underlying causes of the disease that came from this research, the team identified an immunotherapy treatment for this patient. “By comparing rogue clones to normal immune cells, we identified a molecule unique to rogue clones that could be targeted to eliminate them,” recalls Joanne.

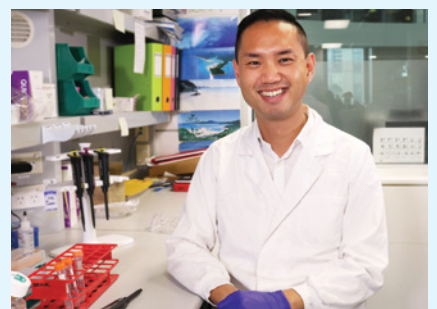
Perhaps even more important is the potential predictive value of tracking rogue clones. “We went back to older blood samples, and could identify rogue clones even in the very early stages of disease. This indicates we could predict patients who are going to develop severe symptoms and treat far earlier, before irreparable damage has been caused,” says Joanne.

A future free from autoimmune disease

As a clinician, Dan also treats patients at Westmead Hospital in Sydney, and sees the suffering that autoimmune disease causes. “While it might not be any of my patients who benefit from our findings, it might be their children or grandchildren, and many thousands of people around the world. The true effect of what we are doing now may be seen after all our lifetimes, but, one day, it will be worth it.”



Professor Chris Goodnow



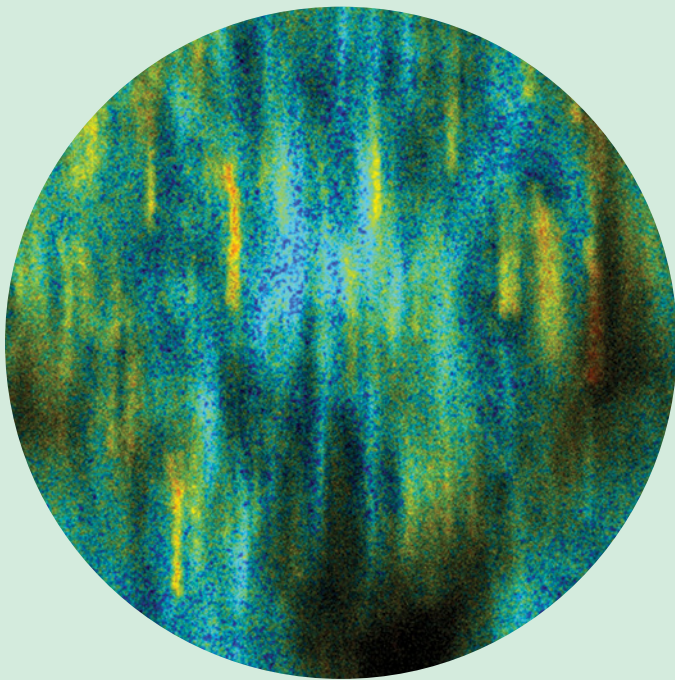
Dr Dan Suan



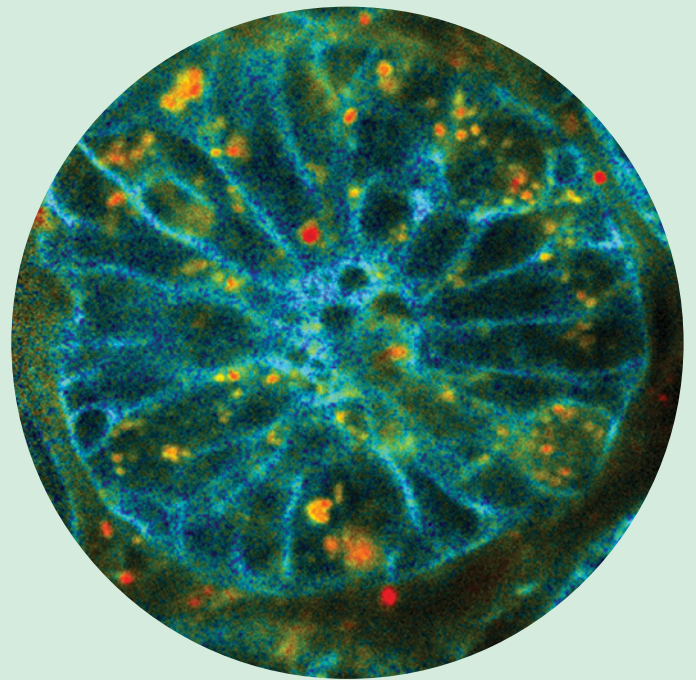
Dr Joanne Reed

THROUGH THE MICROSCOPE

A new software, created here at Garvan, allows us to see, far more clearly than ever before, what is happening in the body.



Before: A high resolution microscopy image of cells inside a living mouse model.



After: The same image with Garvan's Galene software applied.

The two images above show high resolution microscopy of the same activity – a protein called Rac1 in an intestinal crypt. The image on the left is as it was captured through the microscope. It is very difficult to discern the activity because even the tiniest movements – like those of a heartbeat – can blur the images. This movement makes it very difficult to study living animals, including humans.

The image on the right is the same view, after it has been processed with the software tool called Galene, which we've developed here at Garvan.

By correcting for these small movements in microscopy images, Galene makes it possible for us to see events happening in single cells, or even specific parts of a cell. This is essential for studying how well drugs work in real life.

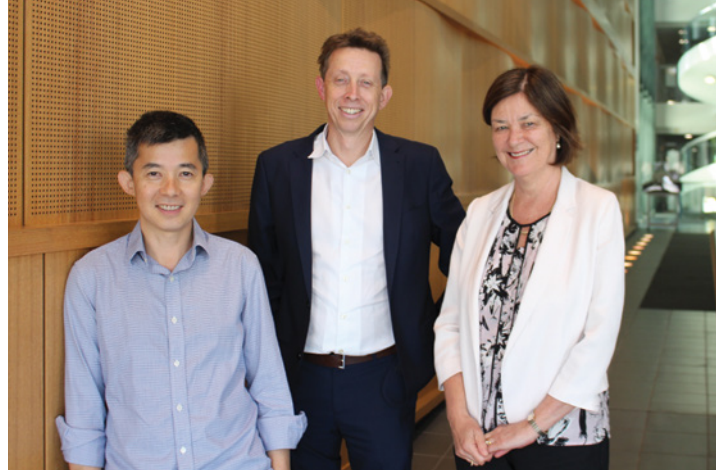
We can now see in the above image the activity of Rac1. The red indicates high Rac1 activity and blue is low Rac1 activity. Rac1 is known to be a key driver of stem cell activation in the intestinal crypt.

In another use of the software, we were able to take steady 3D images of human skin in a volunteer's arm – a technique that could be used to study drug penetration.

Next, we'll work to integrate the tool into the imaging techniques used during surgery. By helping surgeons to find the edges of tumours, for example, this could help to improve the success rates of cancer surgery.

**Dr Sean Warren, Research Officer,
Cancer Invasion and Metastasis**

Associate Professor
Tri Phan, Professor
Peter Croucher and
Professor Susan Clark



The dream team

challenging cancer's biggest secret

Three Garvan researchers from three different research divisions are working to unlock the mystery of why many cancers reoccur, long after an apparent cure.

At Garvan, collaboration is second nature. Our researchers work across diseases and disciplines to implement innovative approaches to fast-track the rate of discovery.

A clear example of this is the joint effort between Professor Peter Croucher, Professor Susan Clark and Associate Professor Tri Phan. They joined forces to answer one of cancer's deepest mysterious – why cancer cells can 'go to sleep' in bones for months or years, only to wake and begin to form a new cancer.

How did you meet?

Peter: My team had been working for years to understand why sleeping cancer cells inside our bones reawaken without warning, often with devastating effect. We knew that it would be crucial to find and track individual sleeping cells inside living animals, something that many thought would be insurmountably difficult. So, when I came to Garvan in 2011 and saw the work Tri had done in setting up a two-photon microscope for imaging cells in real time, I knew this was a great opportunity to collaborate and push our understanding further.

What led you to working together?

Peter: Sue's work in cancer epigenetics is well known in the science world. She has pioneered the field globally. So, it was exciting to realise jointly with Sue that her expertise could shed light on exactly what's going on in these sleeping cancer cells – and what changes as they start to awaken.

Tri: My work with Peter and his team got started in 2013, when we were the first to witness, in real time, a cancer cell arriving at the bone marrow of a living mouse and 'climbing in'.

Sue: The mystery of cancer dormancy has eluded the science community to date. When I heard about Peter's project to address the issue of cancer dormancy in bone, I realised it had enormous potential. We agreed that the collaboration of our divisions, to combine our unique skills, would bring new insights to help solve the mystery of dormancy.

What's been the outcome?

Sue: As a team, we have the capabilities to understand the changes in the cancer cells themselves, as well as

in the evolving ecosystem in which they reside. We hope to identify biomarkers that can be used clinically to help eradicate sleeping cells, and to develop computational models and data visualisation approaches to help us understand dormancy more fully. We're then looking to share our work with patients and the wider public.

Tri: This year, our team was shortlisted for a Cancer Research UK Grand Challenge Award, which is the world's most ambitious cancer research grant. Reaching the final stages of this prestigious award is an extraordinary feat in itself and is testament to the calibre of our work and researchers.

Peter: Sue and Tri and I, with a host of leading researchers from around the world, are committed to solving the problem of sleeping cancer cells. The solution will revolutionise our understanding of cancer.

Please show your support for our world-renowned researchers by making a donation today.

Donations made in memory of loved ones between 1 June and 31 August, 2018

Adriana	Brian Davis	Frank Ewing
Bill and Barbara	Janelle Kaye Davis	Albert Mason
John	Keith Willis Day	Norm McDonald
Julie	Glenda Dollin	James McMurray
Louise	Sally Donovan	Kate S Mignot
Maisie	Deceased	Ronald Miles
Marjorie and Jeffrey	members of the	Ronald Miller
Rochelle	Drechsler family	Shirley Moran
David Abbott	Bridget Laing Dunn	Graham Frank
Stuart Allsopp	Salim El-Chami	Muggridge
Trevor William	Margaret Chisholm	Ann Margaret
Annetts	Rosemary Faber	Nadonza
Ruby Antoniadis	Julie Anne Fakes	Margaret Mae
Greg Ayre	Mr Gino Fazekas	Nelson
Therese Battendier	Judy Feder	Adrian Notley
Norman Beilby	Chris Fishburn	Marjorie Ohlback
Jon Bell	Janie Forsterling	Mr Reginald J Olliff
Randy Sullivan	Stuart Furler	Barry Orrock
Bennett	Domenico Galimi	Gilda Palinginis
Emilie and Lucie	Jan Gardner	Hazel Paton
Bernard	Corel Garling	Val Patterson
Arthur and	Olive M Geldard	John Phillips
Sharron Bexon	Mr and Mrs Godi	Maryanne Pickup
John Bolzan	John Lawrence	Gerry Piels
Barbara Bosher	Goldsmith	Delia Ponti
Gordon Bower	Trish Goldsmith	Fred Povey
Mary Brayshaw	Stewart John	Angelo Pruscino
Jane Bryant	Graham	Leonie Purcell
Valerie Bullus	Peter Harman	Nicodemo
Michael Burgess	Martha (May) Heard	Quattroville
Gail Burgin	Elias Heiki	Douglas Raupach
Viola Butters	Linus Hellqvist	Michael Peter
Ronald Button	Philip Hemstritch	Richardson
Georgina Camenzuli	Maree E Hicks	Katrina Marie
Shirley Munroe	Douglas Hilton	Robison
Carroll	Stuart John Hoy	Roger Rose
Natasha Casey	Ash Hugget	Grace Sadiki
David Campbell	Reeta Humlin	Martin Samociuk
Cathels	Ann Hunt	L Sanchez
Gary Chadwick	Liz Jackson	Norma and
Mee-Na Robyn	Anne Jensen	Ken Scully
Cheok	Evi Joannou	John Sewell
Robert Chi Kau	Barry Jones	Una Short
Ching	Lloyd Gwyn Jones	Giovanna Sieminski
Peter Clarke	Vanessa Juresic	Kevin Skelton
Stewart Clode	Ian King	Fred Sperber
Matilda Ann	Tamara Kogan	Neville Starr
Coleman	Sue Leahy	Elisabete Stewart
Noel Connor	Daryl Lewis	Ivy Stokes
Nyrie Contor	Graham Levy	Vera Strong
Norma and	Paul Lewis	Bert Taylor
Robert Cook	Ian Lindenmayer	Suzanne Taylor
Jean Cooney	Julia May Linyard	Tara Tobin
Helen Cooper	Peter Lownds	Darren Tuxford
June Evelyn	Tim and Andrew	Maureen Twaits
Corkery	Lynch	Carol Wake
Lewis Cox	Colin MacGregor	Robyn Walpole
Peter Cummins	J Mackenzie	Jenny Wareham
Simon Curtis	Michael MacNeil	Jan Weber
Dale Lionel	John Mansell	Joyce Wegner
Cuthbert	Geoffrey Gilbert	Jean Weiss
Anna D'Alberto	Marlow	

Donations made to celebrate a special occasion

Patricia Allsopp's 80th birthday	Dr Corazon Francisco's birthday
Yola Center's 80th birthday	Paulette Goldberg's birthday
Paul and Sue Dowd's wedding	May Tan's 90th birthday

The most generous gift

The gorgeous Paspaley Kimberley Bracelet reflects the raw beauty of Australia's North-West coast, through a striking combination of pearls, sandalwood and onyx.

Even more beautiful than the bracelet itself is the sentiment behind it. The family-owned company donates 25 per cent from each bracelet sold to Garvan, making a huge impact on the lives of people with rare cancers.

For a very special gift this Christmas that gives and gives back, visit garvan.org.au/paspaley today.



CLINICAL STUDIES

We offer a range of clinical trials at The Kinghorn Cancer Centre for the treatment of patients with breast cancer. Find the full list at garvan.org.au/breast-cancer-clinical-trials.

Endocrine therapy for breast cancer

We are seeking patients with advanced oestrogen receptor positive breast cancer. This study will investigate the combination of GDC-9545, a next-generation oral endocrine therapy, alone or in combination with palbociclib (Ibrance), another oral targeted therapy for advanced breast cancer. We are one of two Australian sites in this international study. HREC ref: 17/PMCC/158

Immunotherapy-based study for triple negative breast cancer

We are seeking patients with advanced triple negative breast cancer. This study will investigate the combination of atezolizumab (Tecentriq, an immunotherapy drug) in combination with Ipatersertib (a targeted therapy) and chemotherapy for advanced triple negative breast cancer. We are one of three Australian sites in this international study. HREC ref: 17/PMCC/186

For further information, please contact:
Claire Gray, Breast Cancer Clinical Research Nurse
Email: claire.gray@svha.org.au. Phone: (02) 9355 5708.

Clinical trial spotlight

To develop new medical interventions, Garvan researchers study the effects of treatments with human volunteers.

Garvan researchers are involved in the development of new medical interventions. Often we take part in early animal studies but we also contribute to human studies.

Only when a large number of studies in animals confirm that a drug treatment might work and also that it is safe are we able to start small human studies. To get from a promising treatment in mice to something that can be used in humans takes years, if not decades.

Mice studies can't tell us if a drug causes symptoms like nausea or headache. A crucial part of human studies is to record any and all symptoms that a person has. Human studies carefully compare all symptoms between those taking the study drug and those not taking it.

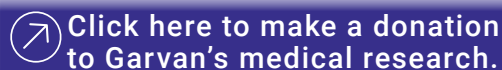
In the Bone Division, we have been part of significant clinical trials exploring new ways to treat osteoporosis. This is a very common disease that makes your bones more fragile so they then fracture easily.

Our most recent clinical trial for a new treatment for osteoporosis has just finished. For this trial, our volunteers visited us at the Garvan every three months for two years. At these visits, a doctor took blood tests and recorded any and all symptoms a participant may have had while in the study. They were part of over 4000 volunteers world-wide who contributed to the results.

Unfortunately many clinical trials don't result in a treatment that can be used for humans but this is not so with our most recent trial. It is likely that this new treatment will become available in 2019 after it has been approved by regulatory authorities. This new treatment may build up lost bone rather than just stopping the rate of bone loss.

The development of a new treatment breakthrough requires extensive human studies and the generosity of volunteers.

Dr Yvonne Selecki,
Translational Research Medical Officer,
Osteoporosis and Translational Research



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