Issue 48

breakthrough

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Garvan Institute of Medical Research

Welcome from our Executive Director

Dear Garvan family,

As we are again faced with lockdowns across much of the country, I hope you and your loved ones are managing well during this challenging period. In times like this, we must support each other however we can and ensure the most vulnerable in our community are protected. As the vaccine rollout continues, we must all do our part to stop the spread of COVID-19; staying home where possible, wearing masks and frequent handwashing are simple things we can all do to help reduce transmission. We pulled together well as a community over the past year, we can do it again.

In our second Breakthrough of 2021, I'm delighted to be able to share with you just a selection of diverse and high impact research being undertaken at Garvan.

As you know, scientists at Garvan focus on a wide array of diseases. This includes the rarest diseases to more common ones, like cancer, metabolic disease, osteoporosis, immune and neurological diseases. On page 5 you'll learn about an exciting collaboration to improve outcomes for people with glioblastoma, an aggressive form of brain cancer.

On page 6 you'll read about the discovery of a new bone cell discovered by Garvan researchers which could lead to new therapeutic targets for skeletal diseases. This is followed by the role that inflammation could play in pregnancy-associated breast cancer. You will also read about how targeting a heat production 'brake' on fat tissues may be a safer way to treat obesity than current medication on page 11.

Finally, we introduce you to two of Garvan's newest recruits: Dr Owen Siggs and Dr Kylie James. I hope you enjoy reading about the exciting research they have underway.

Thank you for your unwavering support of medical research. We could not do what we do without our Garvan family.

Regards,

Professor Chris Goodnow FAA FRS
Executive Director
The Bill and Patricia Ritchie Foundation Chair

RESEARCH NEWS

Vitamin D deficiency may impair muscle function

New research has uncovered a possible link between vitamin D deficiency and impaired muscle function, which could help older adults better maintain muscle strength as they age.

Vitamin D is a hormone the body produces in response to sun exposure, and can be supplemented through fortified foods or oily fish. However, almost one in four Australian adults are estimated to be vitamin D deficient.

A study led by Dr Andrew Philp used experimental models to determine the effects of diet-induced vitamin D deficiency in the mitochondria of skeletal muscle cells. Mitochondria are specialised organelles within cells that convert nutrients into energy, which in skeletal muscle is used to power the movements needed to perform everyday tasks.

The team found that after three months of diet-induced vitamin D deficiency, skeletal muscle mitochondrial function was found to be impaired by up to 37%.

"Impaired mitochondrial function reduces the amount of energy produced in the muscles, which may lead to poor muscle function. Therefore, preventing vitamin D deficiency in older people may help maintain muscle performance and reduce the risk of muscle related diseases, such as sarcopenia," says Dr Philp.

Visit: **garvan.org.au/impair**

Blood test for cancer DNA validated by international research team

Garvan researchers led an international effort to independently assess five commercially-available assays for tumour DNA sequencing – a fast, cheap and less invasive method to diagnose and monitor cancer.

The assays measure fragments of circulating tumour DNA (ctDNA) – fragments of DNA that shed or break off of tumours and enter the bloodstream when cancer cells break down.

The researchers revealed that all assays could reliably detect so-called circulating tumour DNA (ctDNA) when it made up 0.5% of the total DNA in blood, a level of sensitivity that allows detection, genetic analysis and monitoring of late-stage and metastatic tumours.

The study, which was conducted with the US Food and Drug Administration (FDA) and the University of Arkansas for Medical Sciences, forms part of an FDA project that aims to develop standard protocols and quality control metrics to guide the use of next-generation sequencing technologies for precision medicine, to make it a reality for patients.

Visit: **garvan.org.au/blood-tests**

THROUGH THE MICROSCOPE

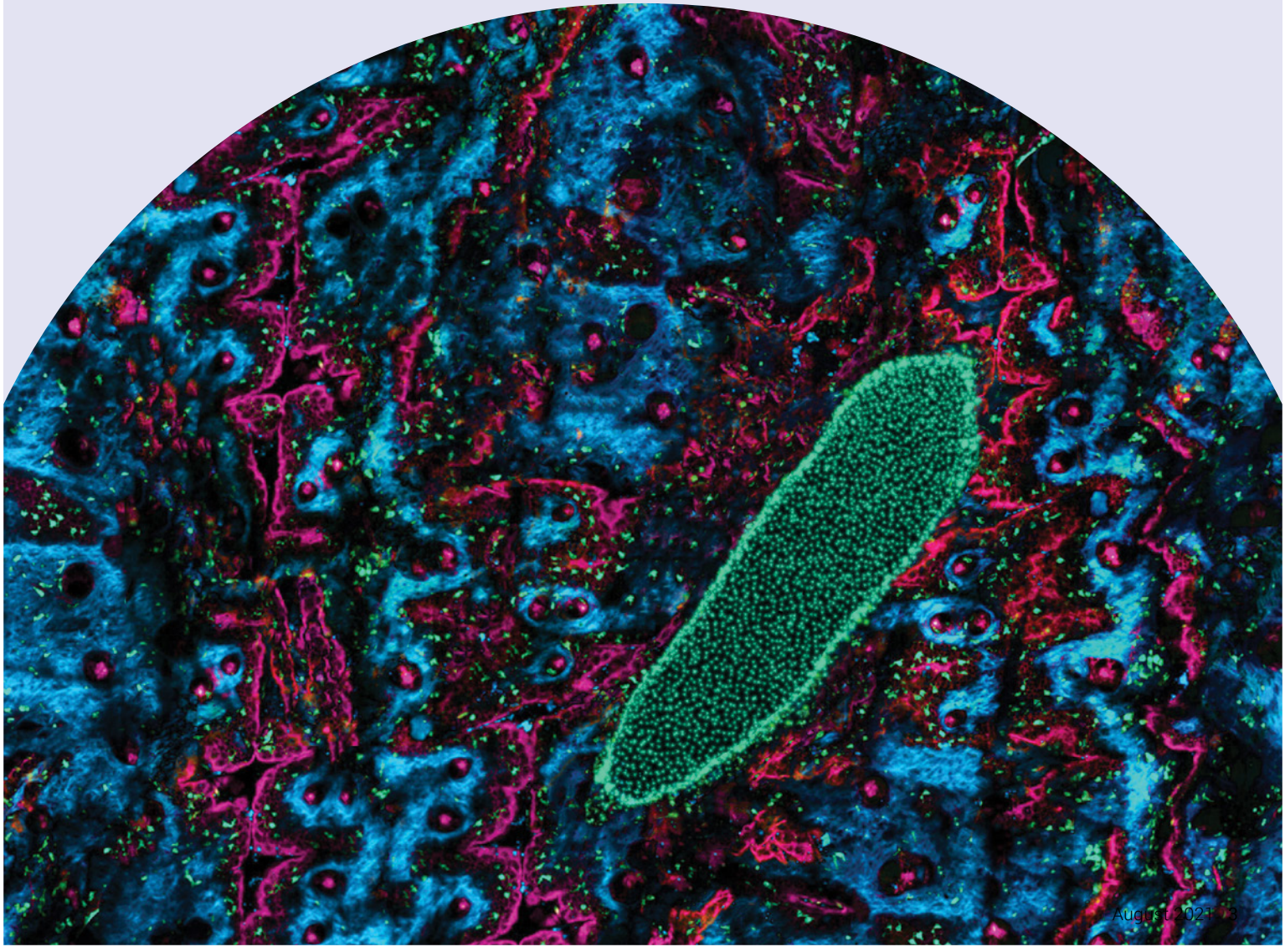
The study of how wounds heal may reveal new targets for cancer therapy.

A papercut and a tumour appear to have little in common – but at the molecular level, the immune system responds to them both in a similar way. This discovery, made in the late 80s, prompted scientists to introduce the concept of tumours as 'wounds that never heal'.

As part of Dr Tatyana Chtanova's Innate and Tumour Immunology Lab at the Garvan Institute, PhD student Arnolda Jakovija studies the microscopic processes involved in wound healing to uncover new insights that may help improve current cancer therapies.

Using next-generation imaging technology, she investigates how immune cells interact with the cell environment, known as the extra-cellular matrix, around a healing wound in real time. Pictured in this image are immune cells called myeloid cells (green), migrating to a small skin incision (centre). The collagen of the skin is shown in blue, with other skin components highlighted in purple.

This image is the winner of the 2020 Light Microscopy Australia Image Competition.



A lifelong, lifechanging partnership

For over 20 years, Kent and Dianne have been involved with the Garvan Institute in more ways than one.

Kent and Dianne McNab

It was a chance conversation with a pharmacist that led Kent McNab to have his bone density checked for signs of osteoporosis. After a positive result on both ankles that indicated low bone density, he was advised to go to his doctor for follow up tests. At first, Kent's doctor was sceptical – osteoporosis normally affected older women, not men his age. After persisting, subsequent testing revealed he had osteoporosis.

At the time, Kent and his wife Dianne had been enthusiastic supporters of Garvan for five years, and had read about the Osteoporosis Clinic and various bone disease studies run by researchers at Garvan. In an attempt to answer questions about his osteoporosis diagnosis, and knowing how important patient data for research is, Kent reached out to the clinic.

"We were lucky enough to be involved with the fantastic Professor John Eisman and his magnificent team at the clinic. Over the years they have inspired me, on my yearly visits to the clinic, to keep fighting for improvement of my bone density," says Kent.

"I always hoped that I would turn the corner and improve my bone density. After 15 years of treatment, including attention to diet and exercise, the hard work and many years of holding my bone density numbers steady has paid off and I have now turned it into a small positive increase. Professor Eisman and his team should be proud of their great efforts and their inspiration to enable me and many other people to be positive and persist with our treatment and life improvement."

"I hope that the information gained from my involvement will assist with the treatment of others to improve their bone density too."

Both Kent and Dianne understand how important medical research is, now and for the next generation. They have generously chosen to be *Garvan Partners for the Future*, by including a future bequest in their Will to Garvan.

"I would encourage all Garvan friends to please, like us, include a bequest to the Garvan Institute in your Will, to ensure their important research can continue into the future." – Kent McNab

Would you consider this special way of giving to the future of medical research?

To request our Bequest Giving brochure or for a no obligation conversation, please contact our Bequest Manager, Donna Mason on **(02) 9295 8559** or **bequests@garvan.org.au** or visit **garvan.org.au/bequest**

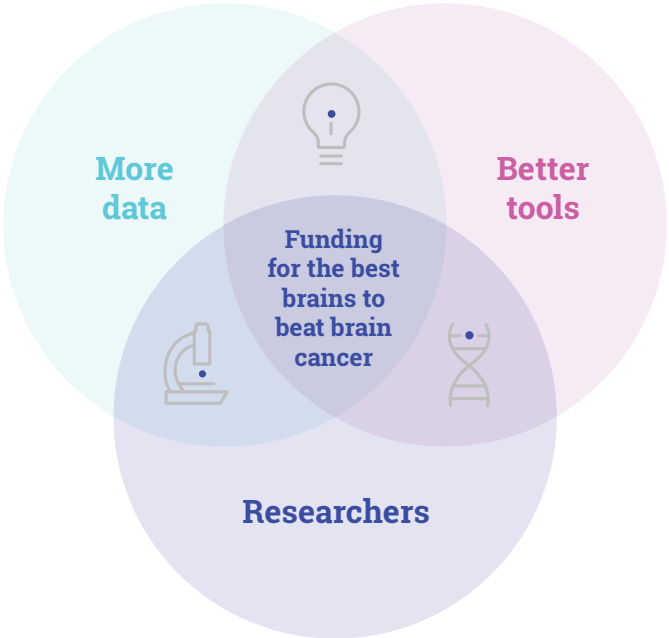
Brain cancer genomics: hitting the bullseye

In a world-first collaboration between the Garvan Institute's Associate Professor Joseph Powell and the Charlie Teo Foundation, this project will organise the billion million bytes of genetic data from Australian brain cancer patients. Using single cell sequencing, this project aims to identify tumour differences and rare cellular subtypes that may contribute to the cancer's development.

Currently, standard genomic and transcriptomic methods profile complex brain tumours in bulk, masking cellular diversity and providing only limited insight into the tumour. Single-cell analysis techniques provide an opportunity to access information about cellular biology at an unprecedented level, opening urgently needed avenues to novel treatment strategies and personalised medicine for patients.

Despite an average 20% increase in five-year survival rates for Australians diagnosed with cancer over the last 30 years, brain cancer survival rates have barely changed over the same period, increasing by only 1%. Only 2 in 10 people with brain cancer will survive for 5 years, with this number decreasing to less than 1 in 10 for people diagnosed with glioblastoma multiforme (GBM).

This project could potentially change how clinicians currently diagnose and treat brain cancer. Targeted therapies are powerful, but brain cancer is a target with many bullseyes that shift position whenever struck. Understanding how tumours become masses of varied cells, all with their own unique mutations will allow researchers to continue to strike the deadliest parts of the tumour until the bullseye is hit.



RESEARCH NEWS

Antibodies selected for better immunity

Researchers led by Professor Rob Brink have uncovered an antibody selection process that is critical to our immune system's ability to destroy pathogens and a key objective for the development of effective vaccines.

The team revealed that the selection happens in germinal centres – the body's 'antibody tuning centres' located in lymphoid organs such as the lymph nodes, spleen and tonsil. The new form of antibody selection is based on the antibody's 'constant' region, which is responsible for recruiting immune 'disposal systems' that can destroy foreign antigens.

The study advances our in-depth understanding of how the body generates an effective immune response and may underpin the design of experimental models of immune disease that will help researchers develop new therapeutic approaches in future.

"Our findings have solved a long-standing paradox and provided an entirely new perspective of how the germinal centre shapes our immune response," says Professor Brink.

This research was supported by Mr & Mrs John and Megan Wade, Wade Civil Engineering Pty Ltd.

Visit: **garvan.org.au/immunity**

Study shows vast potential of genomics-driven medicine approaches

Garvan researchers have made a crucial step forward in the understanding of how our own DNA information influences our personal disease risk, thanks to cutting-edge cellular genomics technology.

By analysing the gene activity of 64,018 individual skin cells, the team revealed for the first time that common DNA variants – the kind that make each individual unique – have distinct effects on the gene activity of different cell types.

The research forms part of a global effort to map disease-causing genes and demonstrates the significant potential cellular genomics technology has for developing more personalised ways to diagnose and treat disease.

"Genetic variation is almost always a root cause of why disease develops, however until recently we've only been able to study its effects on gene activity at the level of tissues, where data is generated from millions of cells together," says Associate Professor Joseph Powell, who led the study.

"We revealed that DNA variants can change the gene activity of distinct cell subtypes, which we and others will further investigate to understand how individual DNA profiles influence disease risk."

Visit: **garvan.org.au/potential**

New type of bone cell discovered

The discovery of a new type of bone cell may uncover therapeutic targets for a range of skeletal diseases.

Garvan Researchers have discovered a new type of bone cell that may reveal new therapeutic approaches for osteoporosis and other skeletal diseases.

The new cells, described in a paper published in the journal Cell, have been named 'osteomorphs' and are found in the blood and bone marrow. Named after the Mighty Morphin Power Rangers, osteomorphs fuse together to form osteoclasts, specialised cells that break down bone tissue.

"This discovery is a game-changer, which not only helps us understand bone biology but presents significant new in-roads for osteoporosis therapy," says Professor Tri Phan, co-senior author of the paper and Head of the Intravital Microscopy and Gene Expression Lab at the Garvan Institute.

"Osteomorphs express several genes that seem to be linked to bone disease, which could lead scientists to entirely new ways to target osteoporosis." – Professor Tri Phan

The skeleton under the microscope

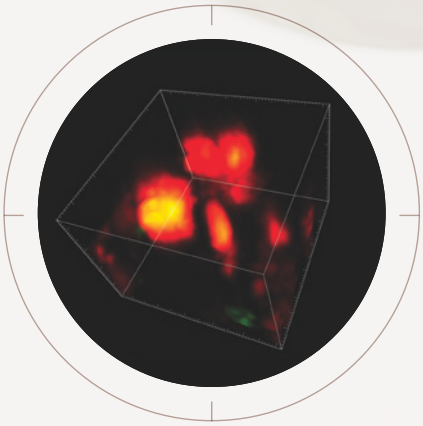
Our bones are constantly changing at a microscopic level. Specialised cells on the bone surface break down old bone tissue (known as resorption) and build it back up to support healthy growth, maintenance and repair to damage. Imbalances in these processes can lead to diseases such as osteoporosis, which affects over 900,000 Australians.

Garvan researchers used intravital imaging technology to look deep inside living bone to better understand how osteoclasts work. They noticed that these cells were doing something unusual – they split up into smaller cells and then joined back to form osteoclasts again.

"This process was completely new to us. The consensus until now has been that osteoclasts undergo cell death after they've done their job, but we saw they were recycling by splitting up and joining back together again, a process which we hypothesise may increase their lifespan," says Dr Michelle McDonald, first author of the paper and leader of the Bone Microenvironment Group at Garvan.

"We also found these cells in the blood and bone marrow, suggesting they can travel to other parts of the skeleton, as a likely 'reserve' of cells that are ready to fuse and deploy when osteoclasts are needed again."

Credit: Dr Kate Patterson



Intravital imaging of osteomorph cells

Authors of the paper published in Cell

A unique genetic signature

Using cutting-edge single cell RNA sequencing technology, which the researchers developed specifically for studying these cells in bone, the team confirmed that these small new cells had a unique genetic profile separate to that of osteoclasts.

"The profile of genes that were switched on in these cells was really interesting – while many genes were also expressed by osteoclasts, several were unique. This, together with the evidence of the new re-fusion processes observed by intravital imaging, convinced us that we had discovered a new cell type, which we called osteomorphs, after the Mighty Morphin Power Rangers," says author Dr Weng Hua Khoo.

Together with colleagues at Imperial College London, the researchers deleted 40 of the genes switched on in osteomorphs in experimental models. They found the deletion of 17 genes impacted on the amount of bone and bone strength, highlighting how important osteomorphs are in bone maintenance.

"When we further investigated human genomic data in publicly available databases, we found that genes switched on in osteomorphs were linked to human gene variants that lead to skeletal dysplasia and control bone mineral density," says co-senior author Professor Peter Croucher, Deputy Director of the Garvan Institute and head of the Bone Biology Lab.

"Together, these findings revealed just how crucial osteomorphs are in bone maintenance, and that understanding these cells and the genes that control them may reveal new therapeutic targets for skeletal disease."

Explaining a common side effect

Beyond revealing new avenues for treating bone disease, the team's findings provide a possible explanation of a commonly observed clinical phenomenon.

"Some individuals who discontinue the osteoporosis treatment denosumab experience a reduction in bone mass and an increase in so-called 'rebound vertebral fractures'," explains Professor Phan.

The authors say that denosumab blocks a molecule that they found is needed for the osteomorphs to form osteoclasts. They suspect that patients who receive denosumab accumulate osteomorphs in their body, and that these are released to form osteoclasts, which resorb bone, when treatment is stopped.

The researchers say studying the effects of denosumab and other osteoporosis medication on osteomorphs may inform how those treatments could be improved and how their withdrawal effects could be prevented.

This research was supported by Mrs Janice Gibson and the Ernest Heine Family Foundation, a Cancer Institute NSW Career Development Fellowship, a Future Fellowship from the Australian Research Council, Fellowships from the National Health and Medical Research Council of Australia (NHMRC) and a Wellcome Trust Strategic Award (101123), an American Society of Bone and Mineral Research Rising Star Award, and a UNSW Cellular Genomics Futures Institute grant.

The impact

The researchers say the discovery of osteomorphs may lead to new ways to target osteoporosis, which affects over 900,000 in Australia alone.

To find out more visit
garvan.org.au/new-bone-cell

Inflammation a key to targeting pregnancy-associated breast cancer

Targeting the tumour environment may help improve treatments for breast cancers affecting pregnant women and young mothers.

New research led by the Garvan Institute of Medical Research has revealed how breast cancer cells that develop during or after pregnancy change their environment to form more aggressive tumours.

In experimental models of pregnancy-associated breast cancer, researchers found that cancer cells send signals to the connective tissue around them to trigger uncontrolled inflammation and remodel the tissue, which in turn helps the cancer to spread.

"Breast cancers that arise during or shortly after pregnancy are highly aggressive as they often become resistant to standard therapies," says Dr David Gallego-Ortega, Leader of the Tumour Development Group at Garvan who co-led the research.

"Our study has revealed a crosstalk between these breast cancer cells and their environment that is fuelling the right conditions for cancer to metastasise, and reveals the inflammation itself as a potential new therapeutic target for the disease."

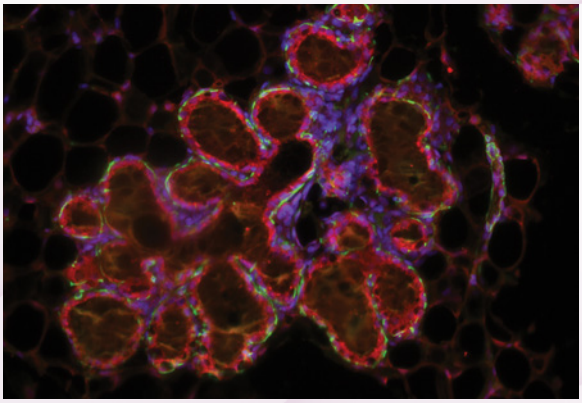
Breast cancer during pregnancy

While a breast cancer diagnosis is devastating for any patient, the prognosis is far worse for pregnant women and new mothers. One in every two women diagnosed with pregnancy-associated breast cancer, which affects up to 40 of every 100,000 women giving birth, will lose their battle within five years of diagnosis.

"We set out to understand the cellular basis of how pregnancy triggers a more aggressive breast cancer and how current treatment could be improved," says Dr Fatima Valdes-Mora from the Children's Cancer Institute, who conducted the research at Garvan.

Using next-generation cellular genomics and experimental models, the researchers analysed a snapshot of gene activity of the individual cells found within pregnancy-associated breast cancers. The researchers observed a number of changes not just in the cancer cells themselves, but in the surrounding connective tissue cells.

Dr David Gallego-Ortega



Microscopy image of mammary gland alveoli during lactation

Credit: Dr David Gallego-Ortega

An environment for cancer to thrive

"In normal breast tissue cells, when mothers stop breastfeeding, changes in hormonal signals tell the connective tissue around milk-producing cells to revert to its pre-pregnancy form. But we found when similar signals were sent by breast cancer cells, they triggered changes that enabled inflammation and faster cancer spread," says Dr Gallego-Ortega.

"The genomic data showed us that as they received pregnancy cues, breast cancer cells transitioned to the more malignant 'basal' breast cancer subtype. At the same time, the cells were 'tweaking' their environment, signalling to cells around them to turn on inflammation within the tumour tissue."

In their model, the researchers found that cells in the tumour environment drove changes that make pregnancy-associated breast cancer highly aggressive – uncontrolled inflammation, tissue remodelling and the generation of new blood vessels.

Path to improving therapy

The findings have prompted the researchers to explore new therapeutic targets for pregnancy-associated breast cancer. In experimental models, the team is next aiming to test whether treating the inflammatory pathways in the tumour environment – some of which can be targeted with existing medication such as ibuprofen – could reduce the spread of pregnancy-associated breast cancers and improve outcomes of current treatments. "Pregnancy can provide cancers with a route of escape from therapy and from their location in the breast," explains Prof Chris Ormandy, Head of the Cancer Biology Lab at Garvan who co-led the research. "Our study suggests that targeting inflammation is one way to stay a step ahead of the disease."

The impact

The researchers hope the study will lead to urgent new treatments for pregnancy-associated breast cancer, which claims the life of one in every two women diagnosed within five years.

To find out more visit garvan.org.au/pregnancy

This research was supported by the Cancer Institute NSW, the Cancer Council NSW, the National Breast Cancer Foundation and the National Health and Medical Research Council.

Boosting body heat to treat obesity

A receptor that helps conserve energy when food is scarce may be the key to a safer approach to treating diet-induced obesity.

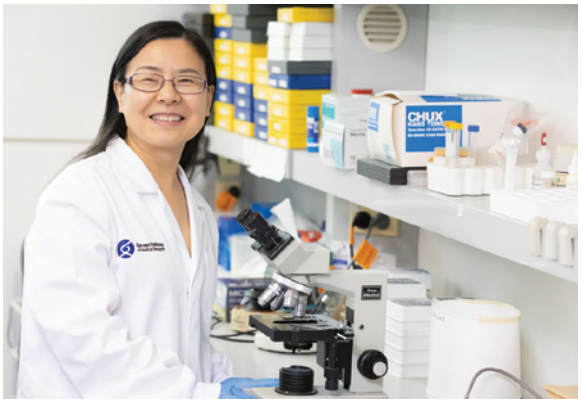
In a study using experimental models and fat tissue biopsies from obese individuals, researchers revealed that blocking a specific receptor of the molecule neuropeptide Y (NPY), which helps our body regulate its heat production, could increase fat metabolism and prevent weight gain.

"The Y1 receptor acts as a 'brake' for heat generation in the body. In our study, we found that blocking this receptor in fat tissues transformed the 'energy-storing' fat into 'energy-burning' fat, which switched on heat production and reduced weight gain," says Dr Yan-Chuan Shi, Leader of the Neuroendocrinology Group at Garvan and co-senior author of the study.

"Most of the current medications used to treat obesity target the brain to suppress appetite and can have severe side effects that limit their use. Our study reveals an alternative approach that targets the fat tissues directly, which may potentially be a safer way to prevent and treat obesity."

Y1 receptor linked to obesity

The authors of the study investigated Y1 receptors controlled by the molecule NPY, which is released under conditions of starvation to reduce energy expenditure and increase fat storage. Surprisingly, the team discovered that Y1 receptors were produced at higher levels in the fat tissue of obese individuals. The team then blocked the Y1 receptor using the experimental treatment BIBO3304 in a mouse model of obesity.



Dr Yanchuan Shi

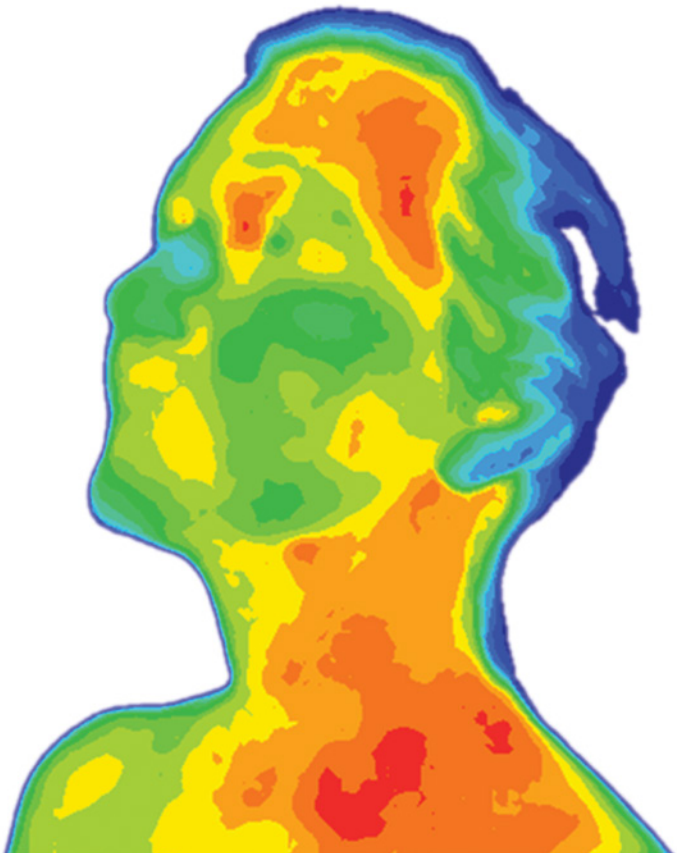
"In our study, we found that mice that were administered BIBO3304 and fed a high-fat diet gained about 40% less body weight over seven weeks than mice on a high-fat diet alone. This significant reduction of body weight gain was caused by an increase in body heat generation and reduction in fat mass," says Dr Shi.

The same mechanism was observed in human fat cells isolated from obese individuals, suggesting the treatment may also be effective in humans.

Importantly, the researchers say a crucial component of the study was to demonstrate that the experimental treatment BIBO3304 did not cross the blood brain barrier, and that the anti-obesity effects of blocking the Y1 receptor pathways occurred not via the brain, but specifically only in peripheral tissues.

This research was supported by Australia's National Health & Medical Research Council and a Diabetes Australia Research Programme grant.

Visit: **garvan.org.au/heat**



Two bright new minds join the Garvan family

Fellowship Spotlight

Thanks to the very generous philanthropic support from the Yarranabbe Foundation provided by Ms Jillian Segal AO and Mr John Roth and their family and the Snow Medical Research Foundation provided by Terry and Ginette Snow and family, Garvan has been lucky enough to welcome two bright new minds to the Garvan family – Dr Kylie James and Dr Owen Siggs. Their expertise will bring new perspectives to Garvan, and will allow us to amplify our pioneering research projects.

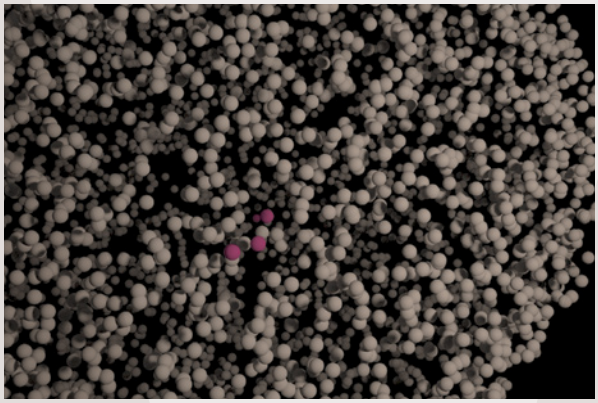
As the Spinak Fellow at Garvan, Dr Kylie James will join Garvan's HOPE and OneK1K initiatives and continue to investigate cell functions in the gastrointestinal tract and how the immune system contributes to Crohn's disease and Inflammatory Bowel Disease. Her team hopes to complete the picture of cellular and microbial landscape of the intestines during Inflammatory Bowel Disease to develop strategies to restore the healthy balance between these intestinal communities.

Named in honour of the late Mr Jeremy Spinak, Ms Jillian Segal AO and Mr John Roth are delighted to be supporting Dr Kylie James. "We are thrilled that Garvan was able to attract someone of Dr James' calibre to work on this important project. Jeremy Spinak was a talented community leader with a bright future whose life was cut short at 36 when he passed away from a rare cancer complicated by underlying Crohn's disease. We hope that this research will not only relieve the suffering of the 75,000 Australians who suffer from inflammatory bowel diseases including Crohn's (with diagnosis commonly between 5 and 35 years of age), but also prevent similar tragedies in future."

Dr Owen Siggs, who is a returned John Monash Foundation Scholar will be supported by an $8 million 8-year Snow Fellowship to focus on debilitating diseases that severely impact the quality of life of those affected. His team will use high-performance computing to analyse the genomes of thousands of individuals with autoimmune and inflammatory conditions to identify acquired genetic changes linked to disease. While this research will help provide an in-depth understanding of some of the most common inflammatory conditions, identifying these disease-causing immune cells opens the possibility of eliminating them altogether; offering potential cures for otherwise incurable conditions.

"We believe that Owen has an exceptional ability to combine his research, training, impressive international networks and the outstanding environment and mentorship provided by the Garvan Institute to achieve a significant impact in the field. With strong financial backing and support from their institutions, we hope young scientists like Owen will produce paradigm-shifting research that wouldn't otherwise be possible. We also want to enable these outstanding young researchers to become the next generation of Australia's research leaders," says Mr Tom Snow, Chair of the Snow Medical Research Foundation.

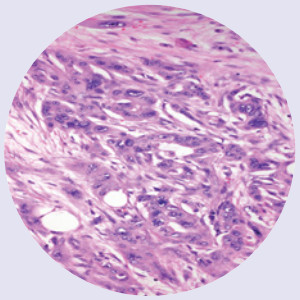
Securing talent like Dr James and Dr Siggs at Garvan is a real investment into Australia's future – bringing a powerful combination of skills in genome computation, genetics, immunology and clinical medicine. We look forward to the breakthrough research that they will drive at Garvan.



3D image of a cell cluster

Clinical Trial **Spotlight**

Pancreatic cancer clinical trial program to target genome and scar tissue



Pancreatic cancer

Pancreatic cancer is one of the deadliest forms of cancer, with a five-year survival rate of just 9% in Australia. More than 3,000 cases are diagnosed in Australia each year, often in the later stages of disease as pancreatic tumours rarely show obvious signs or symptoms in the early stages. By the time most cases are diagnosed, the cancer has already begun to spread outside the pancreas and is often inoperable.

A national clinical trial program will test a promising new targeted therapy for the disease. Garvan and UNSW Sydney researchers will treat pancreatic cancer patients by using genetic information or targeting the tumour environment, known as the stroma.

The research team is working with Garvan's successful Molecular Screening and Therapeutics (MoST) trials program, where the genome of a pancreatic cancer patient's tumour is sequenced and matched to available treatments which can be delivered at a number of different sites around Australia.

If no matched treatment is available, patients will be placed in one of two trials testing the efficacy of therapies targeting the stroma, which in pancreatic cancers can form an impenetrable barrier to treatment.

These two treatments, RXC004 (trial led by the Garvan Institute in collaboration with biotech Redx) and sulfasalazine (trial led by UNSW Sydney), have both been shown to target the scar tissue around pancreatic cancers and prevent tumour growth in experimental models.

This trial aims to bring these lab findings to patients in a meaningful timeframe to improve treatment efficacy and patient outcomes.

For questions about the program clinical trial please email: gcmp@garvan.org.au

CLINICAL STUDIES

New study aims to improve treatment for sarcoma

A national clinical trial will test whether an existing therapy for psoriasis can help treat patients with sarcomas, cancers arising in connective tissue.

The phase II clinical trial, which is a collaboration between the Garvan Institute of Medical Research, Omico (the Australian Genomic Cancer Medicine Centre) and the University of Sydney's NHMRC Clinical Trials Centre, will investigate if treatment with a drug targeting the immune molecule IL23 could improve outcomes for sarcoma patients.

The new trial is the first globally to test a new anticancer pathway for sarcoma. It is supported by the Cooper Rice-Brading Foundation, the Australian Government, the NSW Office of Health and Medical Research, SunPharma, The Kids' Cancer Project, the Matthew Fisher Sarcoma Research Fund and the Daniel Allchin Race for a Cure, and brings new hope to those affected by sarcoma.

To register interest for the trial please contact most@ctc.usyd.edu.au or 02 9562 5000 (ACTRN12620000984998)

PREDICT prediabetes clinical trial

We are seeking men and women aged 20-70 years who have pre-diabetes or who have been recently diagnosed with types 2 diabetes and have not yet been treated with a sugar-lowering medication. This study investigates blood sugar response to personalised diet and diabetes medication. HREC Approval: SVH 17/080.

For further information, please contact
Dr Dorit Samocha-Bonet (02) 9295 8309
predict@garvan.org.au

Jewellery with Purpose

The Paspaley Kimberley Bracelet reflects the raw beauty of Australia's North-West coast, through a striking combination of pearls and renewable sandalwood. With the help of Paspaley's generous support, the MoST program has recruited over 2,200 patients who have exhausted all other treatment options, with 10 major centres now involved in the program across the country. We thank Paspaley for their continued generosity and visionary commitment to breakthrough medical research. To give a gift that means more, **please visit garvan.org.au/paspaley today.**

DONATIONS

Made to celebrate a special occasion

Doreen Cheong's 80th birthday
In Celebration of Dr James Gnanapragasam
In Celebration of the birth of Aiden
Bernard Nightingale's 90th birthday

Donations made in memory of loved ones

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