breakthrough

Decoding kids cancer

The promise of a single cell

Exercise under the microscope

The Parkinson’s puzzle
Here at Garvan, we are truly privileged to have the support of very passionate and dedicated members of the community: philanthropists, community fundraisers, corporations, and Partners for the Future (those who have chosen to leave a lasting legacy through a bequest in their will). While people support Garvan in various ways, they all have one thing in common: they are making a tangible impact on the future of medicine.

You have heard me say it before but, truly, it cannot be overstated: such support is fundamental to what we do here at the Garvan Institute. Without it, our scientists wouldn’t be able to continue their ground-breaking research into some of the most complex and devastating diseases affecting society today. While government funding is essential, there is still a significant gap that must be filled for this vital work to continue.

I hope that you never underestimate the real impact of your support.

It is thanks to our donors that Garvan has been able to remain a leader in research into bone biology, cancer, diabetes and metabolism, genomics and epigenetics, immunology, and neuroscience, not to mention overlaps and collaborations between these six divisional areas.

Philanthropic support provides another, perhaps unexpected benefit to our researchers: it encourages innovation. Donations are vital for funding “novel projects” – that is, promising projects in their very early stages that do not yet have enough basic data behind them to be eligible for government funding. Such projects, in their initial exploratory phase, are the nurseries for major discoveries. Donations are also crucial for the purchase of equipment and technology that is essential to modern day medical research and where, similarly, government funding can be in short supply.

Thank you for your ongoing support and for helping Garvan’s inspirational researchers to continue making important breakthroughs that have the potential to improve disease diagnosis, treatment and outcomes, for the benefit of our community and beyond.

From the CEO

Andrew Giles, Chief Executive Officer, Garvan Research Foundation.

Making news

Outwitting the silent thief

In a world first, Garvan research has revealed that genetic profiling can help predict whether an individual will break a bone through osteoporosis. The findings, which arise from the globally recognised Dubbo Osteoporosis Epidemiology Study, are likely to contribute to clinical decision-making in future, bringing us one step closer to personalised medicine for bone disease.

Osteoporosis is dubbed “the silent thief” because bone loss occurs without obvious symptoms until a bone is broken. Because of this, it is very difficult to predict who will or will not fracture. A key goal of osteoporosis research is to identify those who have a high risk of breaking a bone, with the ultimate ambition of preventing avoidable fractures.

Die another day

New research has shown how the immune system avoids attacking its own tissues with antibodies, while still maintaining a strong defence against invaders.

Researchers from Garvan and the John Curtin School of Medical Research have identified that a type of antibody called Immunoglobulin D (IgD), which sits on the surface of immune cells termed B cells, is responsible for stopping “traitor” cells from producing damaging antibodies against the body’s own tissues (auto-antibodies). IgD keeps the cells in lockdown – unresponsive to the body’s tissues, yet still capable of producing antibodies against invaders.

The findings solve a longstanding mystery surrounding IgD’s function, whose role in the immune system has been unclear since it was first observed 50 years ago.

Two is better than one

Garvan researchers have identified a new driver in the spread of breast cancer to other tissues. The team showed that blocking a survival protein, MCL-1, decreases cancer spread (metastasis). Additionally, this can turbo-charge the anti-metastatic drug dasatinib, suggesting possible combination therapies for metastatic breast cancer. The researchers went on to show that, in cancer cells, MCL-1 regulates proteins called Src-family kinases.

The discovery could have broad potential as MCL-1 and Src-family kinases are important factors in a wide range of cancers. “These findings have implications that could reach far beyond triple-negative breast cancer and in fact beyond all breast cancers,” said Dr Samantha Oakes, who co-led the research.

Adelaide Young, who contributed to this research, answers a reader’s question in Ask Garvan, page 11.
From little things, big things grow

One human cell. It’s only a few microns in size but it contains whole worlds. Now, thanks to a visionary international partnership, we can start to unlock its secrets

Each of our cells is unique, yet until recently the vast majority of medical research has only been able to investigate large numbers of cells at once. The result is an averaging of information that, while useful, cannot hope to uncover how each cell functions within a group. The capacity to sequence the genomes of single cells would enable discovery of unprecedented detail and open up previously inaccessible insights.

The Garvan-Weizmann Centre for Cellular Genomics is getting closer to doing exactly this. This broad-ranging collaboration brings together the research strengths of two institutes under one roof: whole genome sequencing from Garvan, and single-cell genomics and emerging technologies from Israel’s Weizmann Institute of Science.

“It is rare that two research institutes find such complementarity of expertise and sharing of purpose,” says Professor Chris Goodnow, Garvan’s deputy director. “Building a collaborative research centre strengthens ties not just between our institutes and respective countries, but crucially between our research visions.”

Made feasible by rapidly advancing genomic and cell-handling technologies, cellular genomics will revolutionise our understanding of complex diseases, particularly cancer, immunological and neurological diseases – and will pave the way for new therapies and interventions.

The Garvan-Weizmann Centre for Cellular Genomics would never have progressed beyond the dream phase without significant support from the NSW Government and generous philanthropic investment from three groups of visionary donors, including Jillian Segal AM and John Roth.

“When John and I visited the Weizmann Institute a number of years ago I was struck by its overlap with Garvan in many areas of endeavour, and always thought that some sort of collaboration would be ideal,” says Jillian. “Health care is going to become an ever-increasing part of every nation’s focus given the ageing of the population and the only way to deal with the situation is to collaborate internationally and become more effective.”
The active ingredient

It’s no surprise that exercise is good for you, but did you know just how amazingly good, and in what an incredible abundance of different ways?

Many health wish-lists go something like this: stronger, slimmer and more energetic. More smiles and fewer grey hairs. While we’re at it, let’s also protect against serious chronic diseases such as type 2 diabetes, cardiovascular disease, depression, osteoporosis, osteoarthritis, polycystic ovarian syndrome, Parkinson’s and Alzheimer’s and even some types of cancer. Lastly, let’s achieve it without negative side-effects and at potentially zero cost. Failing a personal fairy godmother, there is a way to do all this and more: exercise.

We’ve known for millennia that exercise is beneficial but the details of how it works on the body are still not fully understood. For instance, research has found that losing weight is not as straightforward as energy in versus energy out, as the amount of exercise you do seems to influence how the body burns fat. To unravel such mysteries, Garvan researchers have been looking at the small picture – to cells and molecules – to try to discover exactly what happens when we exercise beyond that smug sheen of sweat.

About 15 years ago, a landmark study found that muscles secrete special molecules, dubbed “myokines”, into the blood during exercise, affecting the function of other organs much like hormones.

“We discovered that a cytokine, namely interleukin-6, which is normally associated with inflammation and the immune system, was released from skeletal muscle,” says one of the study’s authors, Professor Mark Febbraio, who now leads Garvan’s Diabetes and Metabolism Division. “We showed that it signalled the liver to increase its production of glucose so that the contracting muscle could use it as a fuel source. That was really the first example that muscle is an endocrine organ and can communicate information to other parts of the body.”

The implications of this are huge: your muscles do more than move limbs and exercise does more than trim fat. Exercise initiates a chemical change in your body that affects it from top to toe in all kinds of unexpected ways. The discovery launched researchers worldwide on a mission to find and describe new myokines, and the results continue to pile up.

You don’t have to run a marathon every day to get the benefit. Any exercise is good, even walking.
It’s potentially great news for the countless people who suffer from chronic disease, such as type 2 diabetes. “Many people suggest that type 2 diabetes is irreversible. That’s not exactly the case. If you’re diagnosed with type 2 diabetes and your pancreas still has some functionality, you can rescue your health,” says Febbraio. “There are a couple of myokines that have been identified that result in ‘browning’ of white fat, and if you brown white fat then you can increase the energy expenditure of that fat.”

Happily, a dose of myokines is not the sole preserve of elite athletes. “You don’t have to run a marathon every day to get the benefit. Any exercise is good, even walking,” says Febbraio. “Everyone who is reasonably able-bodied should wear a pedometer and aim for 10,000 steps a day.”

Alongside such encouraging news for diabetes, research is beginning to probe myokines’ potential to extend to other diseases, notably cancer. “Excitingly we are now starting to find that the muscle releases proteins that seem to be protective against a range of cancers. That’s something we’re working on a lot now,” says Febbraio. It is known that inactivity contributes to 10 per cent of breast and colon cancer cases in western countries, making exercise a compelling preventative measure.

In cases of existing cancer, exercise appears to slow the growth of tumours, promote cancer cell death and helps prevent the cancer from spreading. The next phase of Febbraio’s research involves exercise-training people with breast cancer to measure how exercise can stop or reverse the progress of the disease.

FEBBRAIO SEES IT AS HIS MISSION TO PROVIDE THE PROOF TO INSPIRE PEOPLE TO MAKE THE LIFESTYLE CHANGE NEEDED TO CASH IN ON EXERCISE’S LIMITLESS BENEFITS. “THE MORE EVIDENCE SHOWING THE PROTECTIVE EFFECTS OF EXERCISE, THE MORE LIKELY PEOPLE ARE TO DO IT,” HE SAYS.

So if exercise isn’t something that comes naturally to you, remember that every time you flex a muscle, you’re releasing your own private army of mini do-gooders that make your bones strong, keep your cells firing and your brain happy. And isn’t that a rejuvenating thought?

Mark Febbraio

A version of this story was originally published in the March/April 2017 issue of Diabetic Living magazine.
To seek to understand Parkinson’s disease is to journey into unknown territory. In most cases, there is no clear cause. There is no known prevention or cure. There’s no definitive test, and it is diagnosed through clinicians’ assessment of a range of symptoms. Yet the disease itself is all too familiar. Parkinson’s is the second most common neurological disease after dementia, with about 70,000 Australians living with the condition.1 Journalist Liz Jackson recently shared a candid portrait of her experience in a Four Corners documentary: “[When I was diagnosed], Parkinson’s meant a bad tremor, an awkward gait and difficulty with handling small change. I’ve since learned it’s a complicated disease of the brain that can have different effects on different people. For me it’s meant pain and panic attacks.”2

So where does such a lack of indicators leave researchers, let alone patients and their families?

“This is a major problem with brain disease. We know the least about diseases of the brain,” says Associate Professor Antony Cooper, who heads Garvan’s Neuroscience Division. “If you have a cancer then often it can be biopsied and swiftly tested. But no one is going to say, ‘Please take a piece of my brain for testing,’ with good reason. Someone might have been diagnosed with Parkinson’s 17 years earlier, and the disease might have potentially started 10 years before they had symptoms, slowly getting worse. So we’re investigating the brain decades later, asking, ‘What caused this to start?’”

Diagnosis typically arrives as the classic “Parkinsonian” symptoms become apparent, such as a resting tremor or slowed movement, which has led to the misconception that Parkinson’s is solely a movement disorder. In truth it has many symptoms, both motor and non-motor. A loss of the sense of smell is often an early sign, which may be followed by chronic constipation, then restless legs while sleeping. Symptoms accumulate in number and intensity as the disease progresses through the brain, region by region, undermining the function of each as it goes.

“One may dismiss some of those symptoms when you’re aging, but then it’s hard to ignore a resting tremor,” says Cooper. “When the tremor appears it is thought 50 to 60 per cent of neurons in a specific part of the brain are already lost. So by the time you’re diagnosed you’re entering the mid-stage of the disease. Most therapies will work best in the early stages.” Current Parkinson’s drugs such as L-DOPA are only effective for treating certain symptoms.

Given this, Cooper and his team study Parkinson’s with two main objectives: finding ways to diagnose before the onset of symptoms, and developing treatments. Both are significant, but their combined value is greater. “Diagnosing someone earlier is most beneficial if you also have a therapy that will slow or stop their disease,” he says. “Such a therapy would be most effective when given early. We need to simultaneously detect and monitor people’s disease, as well as have something to give them. It’s hand in glove.”

Developing a test and a therapy for a disease we know next to nothing about isn’t easy, but thankfully there are some clues. While 15 per cent of Parkinson’s cases have an identifiable genetic cause, the remaining 85 per cent are less clear-cut. For them, genetic factors may also play a role but as an accumulation of small changes overlaid with environmental influences, which combine to cross a biological threshold. Such complex interactions cannot be readily observed through analysing DNA. Instead, Cooper and team have turned to its lesser-sung companion molecule, RNA (ribonucleic acid), in order to map the early disease changes.

If DNA is the book of genetic information, RNA is the transcript of a relevant page. Double-stranded DNA remains sequestered in the cell’s nucleus, while as many as 100,000
different types of single-stranded RNAs migrate into the cytoplasm to perform various functions, such as templating protein production or in regulatory or managerial roles. Being short-lived copies, RNAs are produced according to supply and demand. As such, sequencing and measuring the complete set of RNA in a cell, the “transcriptome”, tells us which parts of the genome are active and by how much.

For researchers, the transcriptome is an Aladdin’s cave of insight into complex diseases such as Parkinson’s. In revealing the cellular pathways that are disrupted it can provide a focus for therapies. Furthermore, Parkinson’s-specific RNAs could potentially be used as “biomarkers”, the nano-sized red flags that signal the early presence of disease in clinical pathology, such as a blood test. But first, they would need to be identified.

From samples of donated brains of people who had Parkinson’s and healthy people who did not, Cooper and his team sequenced the transcriptome, compared the results and found more than 40,000 changes: prospective biomarkers. The next step was to extend the findings to a living cohort. For them, the researchers went to a more accessible source, the blood, to see which among the 40,000 RNA changes were still apparent. “We found that about 3200 RNAs were still different in the blood of known Parkinson’s patients,” says Cooper. Sophisticated computer algorithms found 20 of these RNA changes that, in combination, could distinguish patients from healthy individuals. “From there we will see if we can identify people who later go on to develop Parkinson’s – in other words diagnose them before they have symptoms.”

A pre-symptomatic Parkinson’s blood test would be a major achievement, and not just for its diagnostic potential. Having a way to measure the extent of a patient’s disease would also assist with finding therapies, as it would enable researchers to assess whether a drug is working.

Drug development is notoriously challenging: expensive, protracted, swathed in red tape and with a low success rate. The urgency for a Parkinson’s treatment requires an alternative approach. Garvan is leading an innovative clinical trial in Australia that repurposes existing drugs as possible Parkinson’s therapies. The beauty of this concept is that candidate drugs are already in use for other diseases and thus have attained the safety all clear. The trial is part of an international program coordinated by the Cure Parkinson’s Trust UK, and will be Australia’s first to address Parkinson’s progression rather than symptoms alone.

For Cooper, it is also an ideal opportunity to test his biomarkers. “We’ll be testing drugs to help patients but at the same time we’ll see if our biomarkers change as the patients improve,” he says. It may also prompt a more nuanced understanding of Parkinson’s, with potential for personalised medicine. “We’ll probably find that, under the umbrella clinical term of Parkinson’s disease, we can cluster patients into sub-types. One day we might understand sub-type 1 and find a drug that works better for them than for sub-type 2, for example.”

Such a future may seem distant but, as can happen in science, breakthroughs breed breakthroughs. With energy and ingenuity, what is cutting edge now will become commonplace.

Antony Cooper

You’re a researcher and a clinician. How do the two streams of practice intersect?

I have a clinic at St Vincent’s Hospital where I see patients with immunological diseases. A lot of patients have diseases that are caused by defects in the B cells. This is a type of immune cell that turns into a plasma cell and makes antibodies, which are key weapons that fight off bacteria and viruses. The interactions with the clinic have allowed us to do what’s becoming more common, which is bedside to bench, and then back to bedside research. We have a number of patients who have extremely interesting clinical manifestations of their diseases. With the help of Professor Robert Brink (Lab Head, B Cell Biology) and the MEGA (Mouse Engineering at Garvan/ABR) facility we can make mouse avatars that have the same mutations as the patients, which allow us to examine under the microscope the molecular processes that have gone awry. It’s a really exciting development that embraces some of Garvan’s core strengths in genomics and intravital imaging. I also think a real strength that’s under appreciated is the fantastic connections between the clinic and the Institute.

Your research also moves beyond immunology through cross-divisional collaborations. Why is this?

You go where the science takes you. We’re interested in bone because plasma cells live in bone. We hooked up with Professor Peter Croucher (Head, Bone Biology Division) who is studying multiple myeloma, where the plasma cell becomes a cancer. So there are some very natural synergies there. It’s a great example of how Garvan nurtures collaborations and interactions across different fields. Some of the greatest discoveries, I think, are not because someone had a brilliant idea that no one’s ever had before, but rather someone taking a very simple idea from one area across to a completely different area. So Garvan is really a hotbed for that sort of activity and it’s only getting better.

What motivates you in your work?

In science, we’re just like kids in a sandbox. The really exciting thing is that we’re not just playing with toys; we’re in a really lucky position where we might be able to help somebody out there in the real world with a real-world health problem. That is really inspiring.

What did you do before starting at Garvan in October last year?

When I was in France I was a lecturer in neuroscience. Then I decided to move to Australia to do an MBA in finance and management at UTS. During my MBA, I worked in banking, in strategy. When I completed my MBA in June 2015 I was seven months pregnant with my second child! I went back to banking after maternity leave but I was missing one of my deepest drives: working to make the world a better place.

What prompted you to move into the medical research sphere?

I’ve always been interested in medical research. When I did my masters in neuroscience I was working on hearing impairment, and when I was a lecturer I specialised in disability – physical and visual impairment. My dad was a GP in the countryside of France; my brother is a physio for physically disabled children. I do need a purpose in life, so I thought it was a perfect combination to be an executive officer in such a great institute, and in a role where I can use my MBA as well.

Your background blends science and business. How do they come together in your work?

In the way I understand stakeholders. We engage with stakeholders in government, institutions like hospitals, universities, charities, with supporters, donors, but also other medical research institutes and universities all over the world, not just nationally. I spent some time in research, and then in the corporate world, so I can adapt and understand the jargon from both. It’s great to be at the boundaries of two different industries. That’s where you can add value I think.

Outside of work, what do you do with your time?

I have two young boys, aged four and one and a half. I love to spend time with them and with friends. I do yoga, I love to walk, read and I play the piano, even though it’s hard to find the time to practise!

Staff profile:
Maud Dumont
Executive Officer

Researcher profile:
Dr Tri Phan
Lab Head, Intravital Microscopy – Immunology Division
The concept is simple: every child deserves a chance at a healthy life. Making that happen is now one step closer, thanks to a combination of good deeds, dollars and genome power.

Cheeky, smart, obsessed with Frozen: at first glance Ava seems to be an average six year old. Rewind to January 2011, however, and it’s a different story. When Ava was six months old, doctors found a five-and-a-half centimetre neuroblastoma in her chest and abdomen, with secondary bone cancer. Over the next year, little Ava endured 10 rounds of chemotherapy and three operations. Then, just before Christmas, the family received the best possible seasonal gift: news that Ava required no further treatment. Now, with part-time monitoring, Ava can enjoy the everyday pleasures of any other healthy child, oblivious of her babyhood illness, just as it should be.

Sadly, others aren’t so fortunate. Cancer is the most common cause of disease-related death in Australian children. Every year, more than 800 children and adolescents are diagnosed with cancers, and nearly three will die from cancer every week.

It’s children like Ava – alongside the less happy stories – that have inspired a landmark partnership to combat children’s cancer. Garvan has joined forces with the Children’s Cancer Institute, the Kids Cancer Centre at Sydney Children’s Hospital, the Lions Club International Foundation (LCIF) and the Australian Lions Childhood Cancer Research Foundation (ALCCRF) to launch the Lions Kids Cancer Genome Project.

The project funds whole genome sequencing of tumour and normal tissue for 400 Australian children with high-risk cancer, which will help clinicians to plan the best treatment for each child. In addition, this information will establish a database of genomic factors that predispose children to cancer and assist with prevention and treatment strategies into the future.

The three-year project brings together Garvan’s state-of-the-art capability in whole genome sequencing and analysis, and Australia’s national personalised medicine program in childhood cancer, the Zero Childhood Cancer Program, led by the Children’s Cancer Institute and the Kids Cancer Centre at Sydney Children’s Hospital, Randwick.

Such an exceptional vision, however, requires exceptional generosity and dedication. While LCIF has committed $2.7 million and ALCCRF $0.5 million – the country’s biggest philanthropic donation for kids cancer – the remaining $0.8 million required for project completion still needs to be raised. To learn more, visit genomepower.org.au.

Following her experience with cancer, Ava is getting on with growing up.

Professor David Thomas, Dr Marie Dziadek and Professor John Mattick from Garvan, with Lions leaders from Australia and abroad, including Dr Jitsuhiro Yamada (Chairman of the Lions Clubs International Foundation, top, centre), Dr Joe Collins (Founding Chairman of the Australian Lions Childhood Cancer Research Foundation, top, second from right), and Barry Palmer AM (past president of Lions Clubs International, bottom, third from right).
Witness Rebecca Wilson in full journalistic flight and there’s no mistaking why she is hailed as a legend. Formidable, incisive and passionate, she changed sports reporting in this country forever — and not just for the women who follow her into this notorious testosterone zone.

Though sadly Wilson passed away from breast cancer on 7 October 2016, aged 54, her legacy will continue to inspire positive change in another of her lifelong pursuits: improving cancer outcomes for patients.

In Wilson’s memory, the NELUNE Foundation established the Rebecca Wilson Fellowship in Breast Cancer Research at Garvan. This wonderful initiative has enabled Garvan to recruit a rising star of cancer research, Dr Christine Chaffer, who returns to Australia after several years in the US and Canada.

“My research focuses on something we call ‘cell plasticity’,“ says Chaffer. “This is essentially the ability of cells to change from one type to another — a process that we think is at the heart of cancer cells’ ability to grow into tumours and spread around the body. We’ve demonstrated that by stopping or slowing cell plasticity in cancer cells we can also stop or slow tumours from growing and spreading. The fellowship provides financial security to allow me to continue my research for another five years.”

Long before her own diagnosis, Wilson had supported the NELUNE Foundation as its ambassador, donor and MC at fundraising events. While patient support will always be the Foundation’s main focus, the Rebecca Wilson Fellowship represents a special extension into research in the expectation that it will ultimately contribute to its overarching ambition through better cancer treatments.

“I am very grateful to the NELUNE Foundation, and honoured to receive an award celebrating the life of someone as inspiring as Rebecca Wilson,” says Chaffer. “After seeing the power of philanthropy in North America, it is encouraging to see such support of medical research here in Australia. The more funding we have, the faster we can achieve our ultimate goal of developing effective therapies for advanced cancer.”

Bec’s legacy lives on

A new fellowship in honour of the late, great Rebecca Wilson aims to hasten a brighter future for breast cancer
For her first year, Barbara May Guy seemed to be a normal baby. Then as the months went by it became clear that she was not reaching developmental milestones. She did not learn to talk and her family gradually came to accept that Barbara would live with a disability, one that would never be fully understood.

Back then, in the 1930s, the options were few. “As far as I’m aware the philosophy at the time was that nothing can be done, and that it was best for everyone’s sake to put her life aside,” says John Guy, Barbara’s younger brother. John has a fleeting memory of his sister at home before she went into full-time care at Newcastle’s Stockton Hospital (later the Stockton Centre). Barbara lived there for 74 years until she died in 2013, aged 82, from pneumonia following surgery for a hip fracture.

While this was a life no one would choose, Barbara was never abandoned or forgotten by her family. Her needs were met in a safe and caring environment. She grew to accept her life and was happy to return to her secure place after outings.

Wishing to recognise the value of Barbara’s life, John and his wife Pamela posthumously enrolled her as a Partner for the Future by granting her entire estate to Garvan. “In this way Barbara can thank those who assisted her on life’s pathway and extend hope to people like her in the future. We will be honoured to do the same,” says John.

As we have seen with children such as Alan (see garvan.org.au/alan), a better future for people living with genetic conditions is tantalisingly close.

Thank you to John and Pamela, and especially to Barbara, for entrusting your hopes to Garvan with your bequests.

If you would like information about leaving a gift to Garvan in your will and becoming a Partner for the Future, please contact our Bequest Manager, Carol O’Carroll on (02) 9295 8117.
Can breast cancer recur if there’s been both chemotherapy and radiotherapy?

A: When a patient receives both chemotherapy and radiotherapy it is effectively a two-pronged attack to increase the likelihood that all breast cancer cells are destroyed. While they are very effective and have saved many lives, unfortunately there is always a risk some cancer cells may be able to resist these therapies.

Chemotherapy kills cells that are multiplying quickly, like cancer cells. A small proportion of breast cancer cells, however, may divide slowly or be dormant (alive, but not multiplying) and are therefore resistant to this type of therapy, even after multiple rounds of treatment. Radiotherapy on the other hand causes damage to cells within a targeted area and breaks their genetic code or DNA. For most cancer cells the DNA damage is catastrophic and causes them to die, however, it is possible for a small population of breast cancer cells to repair the damage and survive.

So, although these therapies have greatly increased the survival of women and men with breast cancer, in some cases cancer cells are able to survive and the disease can recur. It is these that researchers at the Garvan Institute are attempting to understand and develop new therapies for.
In celebration

Thank you to those who marked special occasions with gifts to Garvan. Your donations will help us continue our vital work.

Warm wishes to newlyweds Robert Jackson and Laura Curotta. May you enjoy many years of health and happiness together.

Happy anniversary, James and Stephanie! Congratulations to Carrie Jacobi for your wonderful years of teaching.

Coming up

Please join us for another public seminar in our popular program. In July, we take a special look at diabetes research from across the institute. You can meet the people behind the research and see first-hand how your donations are changing the future of medicine.

**Topic:** Diabetes (type 1 and type 2)

**Date:** Tuesday 11 July

**Time:** 10am – 12pm

**Location:** Garvan Auditorium

This seminar is free of charge, but space is limited and bookings are essential. Seminars are held at the Garvan Institute of Medical Research, 384 Victoria Street, Darlinghurst, Sydney.

To reserve your place, call 1300 73 66 77 or visit garvan.org.au.

Clinical study

**Brown fat and blood pressure**

Brown fat is a special kind of fat which burns fat in the body. We are looking for volunteers who have high blood pressure to participate in a trial investigating the effect of a medication on brown fat. Participants must be aged 18 to 45 years and currently on one blood pressure medication.

For further information please contact:

Dr Paul Lee (02) 9295 8416 or email p.lee@garvan.org.au

(St Vincent’s HREC Ref 14/SVH/105).