2017–2018

SVI YEAR

RISING STARS THE NEXT 60 YEARS AT SVI

SVI
ST VINCENT'S INSTITUTE
MEDICAL RESEARCH

60 YEARS OF DISCOVERY

CONTENTS

THIS IS SVI

April 2018 marked the 60th anniversary of the opening of SVI.

When racehorse trainer Jack Holt died in 1951, he left £200,000 (equivalent to around $81 million today) to establish a medical research hub at St Vincent's. Holt's vision was an organisation that nurtured medical research excellence, with a focus on the diseases that affected those people in the Hospital just next door.

The laboratories at SVI were officially opened on the 23rd of April, 1958, led by inaugural Director, Pehr Edman. Edman was a quiet man who made a major contribution to our understanding of the mechanisms of disease. His work at SVI in developing the world's first automated protein sequencer prefaced many of the major breakthroughs in medical research that came in the decades that followed.

While many of the stars of SVI's past have now faded, researchers at SVI today continue to execute Jack Holt's vision: giving hope, through research, to Australians affected by disease.

MISSION
To carry out the highest quality laboratory-based biomedical research in order to make discoveries that will improve the health of the community.

VISION
To be a thriving medical research institute that makes discoveries with impact.

VALUES
Excellence, passion, creativity, collaboration, individual drive, integrity and questioning of dogma

Front cover: Mouse bone marrow cells stained to show the nucleus. (Dr Gavin Tjin, Stem Cell Regulation, SVI)

SVI
ST VINCENT'S INSTITUTE MEDICAL RESEARCH
60 YEARS OF DISCOVERY

SVI'S RESEARCHERS HAVE ALWAYS BEEN AT THE HEART OF OUR SUCCESS. THE COMING YEAR WILL SEE A FOCUS ON PROVIDING OUR PEOPLE...

This April marked the 60th anniversary of the opening of the research laboratories at SVI. When we take people through the Institute today, we always begin by highlighting the contribution that was made by Jack Holt, our founding benefactor, and by Pehr Edman, our inaugural Director.

The vision of both Holt and Edman for the Institute in its early days is echoed today with our focus on excellent fundamental biomedical research that has a clear line of sight to improved health outcomes. The medical research institutes (MRIs) in Melbourne, of which we are the third oldest, and with which we have close links both culturally and collaboratively, have an undeniable impact on Victoria's economy, health outcomes and reputation as an international hub of discovery.

The last 12 months have been a successful period for SVI in terms of grant funding success. It has also been notable for increased funding from philanthropy, industry and commercial sources. This has been driven by a sharper focus on sustainability of funding at the Institute. Sourcing funding from outside the traditional government grants not only insulates us from the vagaries of the grant system, but also helps us to achieve our goal of delivering impact through research excellence and its translation. This will continue to drive both the Institute and Foundation Boards in the year to come.

2017 saw a 'changing of the guard' for the leadership and governance of the Institute.

A very significant change occurred with the retirement of Brenda Shanahan, who was the Chair of the SVI Board from 2004-2017 and who had been a Board member since 1995 – a remarkable tenure. During this time, she was also Chair of the Board of St Vincent's Hospital Melbourne. Being simultaneously Chair of a hospital and its co-located research institute is unique in the Melbourne medical scene.

Brenda brought a wealth of experience to SVI from her career as a leading member of the financial markets community and her broad network in that sector. She has been an advocate for a closer relationship with the Hospital and for collaboration generally, building strong relationships with the leaders of our affiliated organisations and the Chairs of the other MRIs. Brenda established the SVI Foundation by hiring our first fundraiser using her own financial resources and she has been a generous donor to the Institute over her long period of involvement with us. It was great to celebrate her many contributions late last year.

In 2017, we also farewelled Sue O'Neill and Sue Alberti from the Institute Board and welcomed new Directors Chris Pearce, Fiona Rowland and the new CEO of St Vincent's Hospital Melbourne, Angela Nolan. Chris' background is varied, with extensive experience in business as well as government. Angela knows the campus well, having been in numerous senior roles at St Vincent's Hospital in recent years. Fiona is a lawyer with extensive not-for-profit and philanthropy experience. We have a strong Board and we

...WITH CUTTING-EDGE TECHNOLOGY TO ALLOW THEM TO EXECUTE SVI'S VISION, AND ON RECRUITING TOMORROW'S 'RISING STARS'.

are very grateful to them and to our Foundation Board for their hard work on our behalf.

We also have many new staff, including new senior administrative appointments. In early 2018, Associate Professor Natalie Sims was appointed Deputy Director of SVI and we are already benefitting greatly from her involvement. She is an experienced researcher with expertise in the biology of the skeleton, which is a long-standing focus of research at the Institute. Kate Barnett (CEO of SVI Foundation) and Philippa (Pip) Hetzel (CEO of NRL) were also significant new appointments in recent months.

Of course, change is an exciting opportunity for renewal and fresh talent – and this is the main theme of this year's Research Report. The Report highlights the tremendous contribution that is being made by our 'rising stars', as Chair of our Foundation Board, Karen Inge, likes to call them. Supporting these researchers at an early stage of their careers is a priority for us.

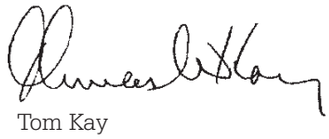
Funding from peer-reviewed national and international grants is extremely competitive and will remain so. Our researchers have done well in a competitive environment, but the difficulty of obtaining these grants and the increasing cost of the sophisticated technologies that support world-leading research highlights the role that philanthropy and other sources of funding play.

SVI's researchers have always been at the heart of our success. The coming year will see a focus on providing our people with cutting-edge technology to allow them to execute our vision, and on recruiting tomorrow's 'rising stars'. We continue to work with the Hospital and other partners on the plans for the Aikenhead Centre, our healthtech innovation initiative that sits at the intersection of clinical practice, engineering and technology, and industry. This has great promise in involving us in multi-disciplinary, cross-institutional collaborations with a focus on improved patient outcomes. It will help our researchers get their discoveries out of the laboratory and into clinical use, delivering greater impact.

We thank all of our supporters, including our donors, the State Government of Victoria (for the Operational Infrastructure Support Scheme), the NHMRC and many Trusts and Foundations. Thanks too to the national Board of St Vincent's Health Australia, and the Trustees of The Mary Aikenhead Ministries.

Thanks to the contribution from our staff and stakeholders, past and present, we look forward to a further 60 years of discovery at SVI.

Tony Reeves
Chair, SVI Board



Tom Kay
Director

TYPE 1 DIABETES

TYPE 1 DIABETES IS A CHRONIC AUTOIMMUNE DISEASE CAUSED BY THE IMMUNE SYSTEM DESTROYING THE INSULIN-PRODUCING CELLS OF THE PANCREAS.

Children are the most commonly diagnosed group

Long-term health complications include heart attack and stroke, vision impairment, kidney disease and nerve pain

Cause is unknown – thought to be genetic and environmental

5% of people with a parent or sibling affected will also get the disease

Warning signs include increased thirst and urination, hunger & weight loss

1,500 shots of insulin required per person each year

AUSTRALIA

Australia has one of the **highest rates** of type 1 diabetes in the world

130,000 Australians have type 1 diabetes

$570 million, the estimated annual healthcare cost

SVI at a glance:

SVI has the largest group of type 1 diabetes researchers in Australia.

Research covers fundamental to clinical studies, working towards better treatments and a cure.

Since 2007 SVI has performed human islet isolations to treat more than 29 people with type 1 diabetes.

DAVID DANKS WAS 13 WHEN HE WAS DIAGNOSED WITH TYPE 1 DIABETES.

"I was with my Aunt Kate one night, and while I wasn't feeling unwell, she noticed I was drinking and weeing a lot. She has type 1 diabetes, so she told my Mum. The next day we went to the doctor.

After a urine test, the doctor sent us straight to the Royal Children's Hospital where they confirmed the diagnosis after a few blood tests. While above the normal range, they initially weren't sure if I had type 1 diabetes. Looking at my family's health history we found that, in addition to Aunt Kate, there were multiple occurrences back in the 1920s."

David's mother Jeni says that for the first 6 months, she thought life would never be the same again.

"Initially we felt unprepared, out of our depth and shattered. But our life has returned to normal.

I recall my sister Kate saying that 'Nobody understands my condition', and now I know what she means. Most people have very little knowledge of type 1 diabetes, and when they do, it is often confused with type 2.

> "MOST PEOPLE HAVE VERY LITTLE KNOWLEDGE OF TYPE 1 DIABETES, AND WHEN THEY DO, IT IS OFTEN CONFUSED WITH TYPE 2."

While David's diagnosis came as a shock, type 1 diabetes hasn't stopped him from leading an active life. A year after his diagnosis, he's ridden 300km in 5 days, sailed in a state championship, rowed in the Head of the River competition, and participated in a week-long surf camp.

He constantly uses his blood monitor that transmits to our phones and, when he is on camp, his teachers' phones. An alarm sounds when his blood glucose level is too low or high. It doesn't replace finger pricks, but gives greater peace of mind.

It is wonderful to know of the research that is going into prevention for people predisposed to type 1, and I hope the knowledge continues improving at the current rapid rate to help those living with the condition."

SVI's researchers have always been at the heart of our success. The coming year will see a focus on providing our people with cutting-edge technology to allow them to execute our vision, and on recruiting tomorrow's 'rising stars'.

Image: David Danks (right) with his mother and brother

DR MICHELLE SO'S WORK WOULDN'T BE POSSIBLE WITHOUT THE ASSISTANCE OF THE PEOPLE WHO HAVE DONATED TO SVI'S LIVING BIOBANK.

Endocrinologist and PhD student Michelle So has spent the last 3 years of her life doing the medical research equivalent of a 'Where's Wally?' puzzle, with a layer of added difficulty. She has been trying to identify a specific type of immune T cell – her version of Wally – without knowing exactly what it looks like.

Her PhD project aimed at identifying immune cells in the bloodstream in people with type 1 diabetes, specifically targeting those cells that are on their way to the pancreas with the task of killing insulin-producing beta cells.

Michelle says that if they were able to biopsy the pancreas of a person newly diagnosed with the disease, the job would be considerably easier.

"It would be like organising to meet Wally and knowing that he is going to be standing at a certain spot at a certain time, wearing his striped red top and beanie. But as we can't do a biopsy, what we are trying to do is find these cells in the bloodstream. That is a bit like trying to find Wally by turning up at a place far from where he was last spotted, without a good description of what he might look like."

Michelle's work wouldn't have been possible without the assistance of the people who have donated to SVI's Living Biobank. These people agree to provide a sample of blood on demand. This has the advantage of providing fresh, rather than frozen, blood cells – which is necessary for the delicate cells that Michelle is trying to find.

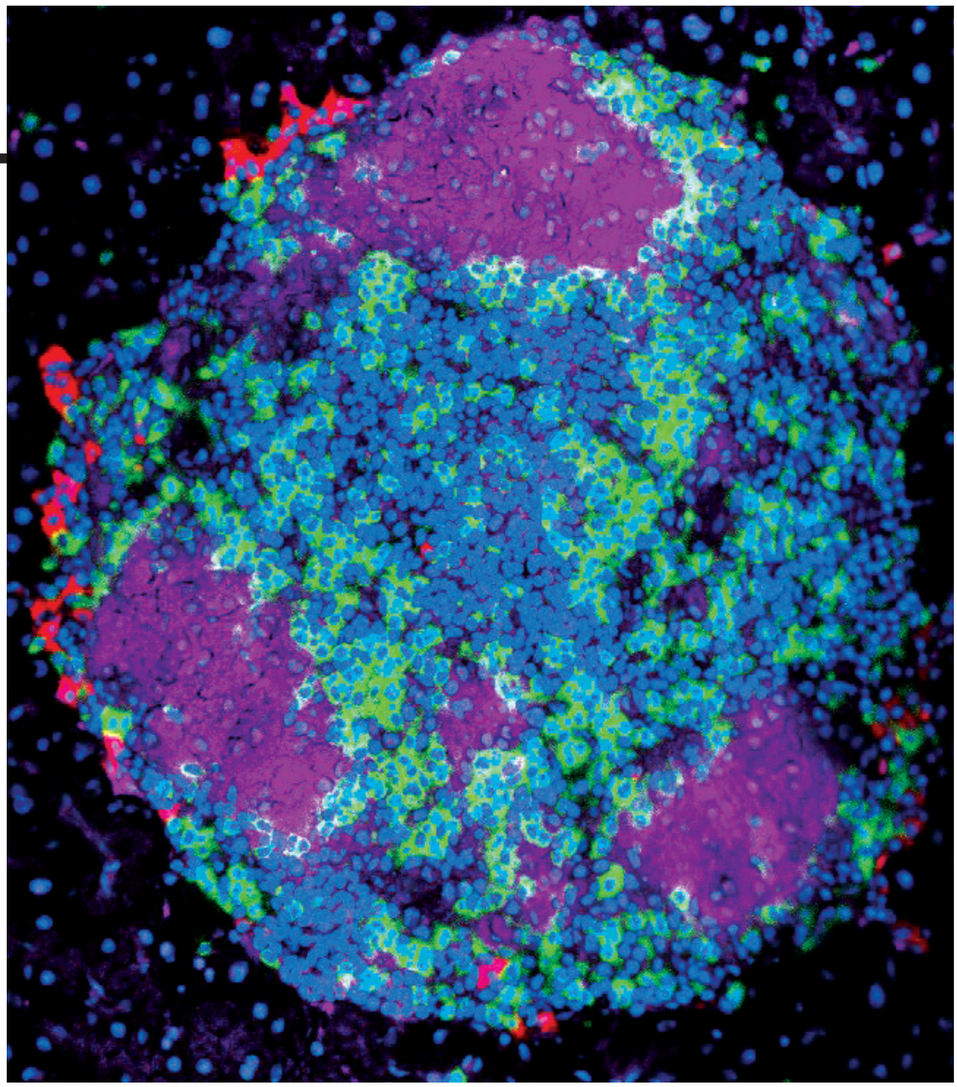
Using these samples, Michelle was able to show that immune cells in the blood from people who had recently been diagnosed with type 1 diabetes responded to a particular protein sequence that hadn't been studied before. This indicates that the sequence, which is part of the precursor to insulin, may play a role in triggering the immune cells to attack, or in amplifying the immune response in people with the disease.

The team at SVI are now working towards a blood test which can identify people at risk of developing type 1 diabetes. This test will be used to measure if therapies to prevent the condition are working as hoped, which will enable new therapies to be evaluated rapidly.

Once she has submitted her PhD, Michelle plans to continue her training in the USA.

Michelle says, "We now know much more about what these cells look like and how they respond – we have a sort of identikit for Wally. I hope that our work will allow us to give people at risk of type 1 diabetes more certainty about whether they are likely to develop the disease and also give us a way to test the efficacy of new treatments as they come on line."

THE NEXT STEP.



In 1922, Canadian teenager Leonard Thompson, lying in a diabetic coma, was the first person to be given an experimental treatment for type 1 diabetes – an injection of insulin. This momentous occasion signalled a shift in the diagnosis of type 1 diabetes from being a fatal condition, to a chronic one.

Although almost 100 years separates us from that event, the treatment for type 1 diabetes essentially remains the same today.

Insulin treatment addresses the deficit that is caused when someone's pancreatic insulin-producing cells are killed by their body's own immune system.

However, researchers will only be able to design new treatments to stop the disease from developing if they can understand what it is that triggers the immune system to attack these cells in the first place.

In 2017, the team from SVI's Immunology and Diabetes Unit published an article in the journal *Diabetes* in which they identified a potential culprit.

The researchers were interested in the role of a protein called granzyme A. It was thought that this protein was an important part of the immune response. To dissect its role, they removed the protein from mice that were genetically prone to developing the disease.

> HELEN SAYS THEIR FINDINGS INDICATE THAT THE TRIGGER FOR TYPE 1 DIABETES COULD COME FROM WITHIN THE CELL ITSELF.

Associate Professor Helen Thomas says that the results were very surprising. "We thought that by making a genetic modification to these mice – essentially taking away this protein involved in activating the immune system – we would protect the mice from developing diabetes."

To the researchers' surprise, the modified mice actually developed diabetes at a much faster rate. They then showed that granzyme A's role in protecting cells from viral attack could provide a reason why.

Granzyme A acts like a pair of molecular scissors, cutting up double-stranded DNA that appears where it is not supposed to be. This mechanism evolved because the presence of DNA in the body of the cell, as opposed to the nucleus where it normally resides, is a hallmark of viral infection and prompts the cell to activate immune pathways to try and protect itself.

It has been long thought that an environmental trigger such as a viral infection is the most likely cause of the initial immune response in type 1 diabetes. Conventional thinking goes that immune cells reacting to a virus become overzealous and attack specific cells in the body by mistake.

The mice in the study developed accelerated diabetes because when granzyme A wasn't able to carry out its normal role, the DNA that accumulated caused the immune system to ward off a non-existent viral infection, with the response eventually leading to accelerated killing of the insulin-producing cells.

Helen says their findings indicate that the trigger for type 1 diabetes could come from within the cell itself.

"Our next step is to identify whether this same trigger has a role in humans with type 1 diabetes."

Image: A stained pancreatic islet showing the immune T cells in green, insulin-producing cells in purple and single stranded DNA in red

TYPE 2 DIABETES

TYPE 2 DIABETES IS A METABOLIC DISORDER THAT OCCURS WHEN THE CELLS IN THE BODY BECOME RESISTANT TO INSULIN'S EFFECTS AND/OR THE PANCREAS DOESN'T MAKE ENOUGH INSULIN.

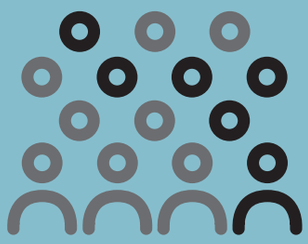
Most common
form of diabetes

Risk factors
include family history, being overweight & physical inactivity

Complications
can affect the eyes, heart and circulation

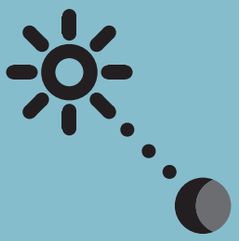
AUSTRALIA

1 million Australians have type 2 diabetes



About 60% of cases are thought to be preventable

Fastest growing chronic condition in Australia



Every day 280
Australians are diagnosed with diabetes

500,000 Australians are thought to live with undiagnosed type 2 diabetes

$14.6 billion, the total estimated annual cost of diabetes in Australia per year

SVI at a glance:

Research in the Protein Chemistry and Metabolism Unit centres on the role of AMP-activated protein kinase (AMPK), which acts as the body's master regulator of metabolism.

In 1994, researchers at SVI played a key role in identifying and sequencing AMPK.

Researchers at SVI are focussed on understanding AMPK and its broad role in the body, including in heart disease, rebound weight gain and finding ways to control its functions with drugs.

KARLA STRUGGLED AFTER BEING DIAGNOSED WITH TYPE 2 DIABETES AT 30 YEARS OLD.

"The day of my diagnosis was devastating. I knew it was coming after feeling extremely sick when I had my glucose tolerance test the week before.

I spent days crying and really struggling with the diagnosis. It felt different to being diagnosed with gestational diabetes – I knew that would end when the pregnancy did. But type 2 is a chronic disease and the thought of that tore me apart.

My children were 5 and 4 at the time. They couldn't understand why I was upset, and they didn't know what diabetes was. After a while they started to ask me if what I was eating had sugar in it, and they would tell me I couldn't eat something if they were eating something sweet! It made me less sad because I knew they were looking after me, and they were the biggest reason that I needed to look after myself.

My mum was amazing. She also has a chronic illness and understood the enormity of being diagnosed with something that won't go away. My husband felt sad for me and supported me in giving up sugar after the diagnosis, but only as long as it didn't affect him!

Lately I have been struggling with food issues again. My family have gone through a lot in the last year with my husband having cancer, and I turned to food to help get me through it. I've been trying to pull myself back to my version of normal but it's difficult.

I currently don't have any of the side effects of diabetes and I would like to keep it that way.

The team of medical support that is behind me is amazing. My dietitian and diabetic educator are right on top of every change in my blood tests. They tell me that something's a bit low, or a bit high, and how to fix it so that it minimizes the chance of any major complications.

When I was first diagnosed, I guess I was concerned that it was a bit of a death sentence. I know now that it doesn't have to be that way and that I can lead a full, long and happy life if I just take care of myself in the right ways.

> "...I CAN LEAD A FULL, LONG AND HAPPY LIFE IF I JUST TAKE CARE OF MYSELF IN THE RIGHT WAYS."

In terms of medical research, my hopes are for a cure for all types of diabetes. As much as I complain, I know that the way my diabetes manifests is a lot better than others. It would be amazing to give everyone a life without worrying about their health."

Image: Karla with her family

DR JOHN SCOTT BECAME INTERESTED IN THE ROLE OF LITHIUM BECAUSE OF HIS FOCUS ON THE PATHWAYS THAT CONTROL ENERGY USAGE IN THE BODY.

Since Australian psychiatrist John Cade employed lithium to successfully treat one of his bipolar patients in the 1940s, scientists have tried, and largely failed, to understand why the treatment is so effective. The difficulty stems from the fact that lithium interacts with many proteins and other molecules in the brain, making it hard to determine which interaction is responsible for its mood stabilisation effects. This is also the root of lithium's undesirable side effects.

Dr John Scott in SVI's Protein Chemistry and Metabolism Unit became interested in the role of lithium because of his focus on the pathways that control energy usage in the body. His particular focus is a protein called Calcium/calmodulin-dependent protein kinase kinase 2 (CAMKK2).

"CAMKK2 is one of the key steps along the pathway that leads to the activation of a protein called AMP-activated protein kinase, which acts as the body's energy gauge. AMPK has been a focus of research in our Unit for decades," says John.

"One way of dissecting the action of a protein is to look at the effects in people who have a genetic mutation in that gene. A couple of years ago we became interested in mutations in CAMKK2 in people who are affected by behavioural disorders such as anxiety, bipolar and schizophrenia."

John and his team showed that when they mimicked the human CAMKK2 mutation in the test-tube, the ability of CAMKK2 to activate the next step in the pathway was reduced.

John says, "We basically found a 'switch' in the protein that is normally able to be turned on and off. In people with a mutation in CAMKK2, this switch is permanently off. Excitingly, we were able to show that treatment with lithium could overcome the effects of the mutation."

"Our hypothesis is that at least one of the ways that lithium works is by creating a bypass of the defective circuit, essentially leapfrogging the broken switch."

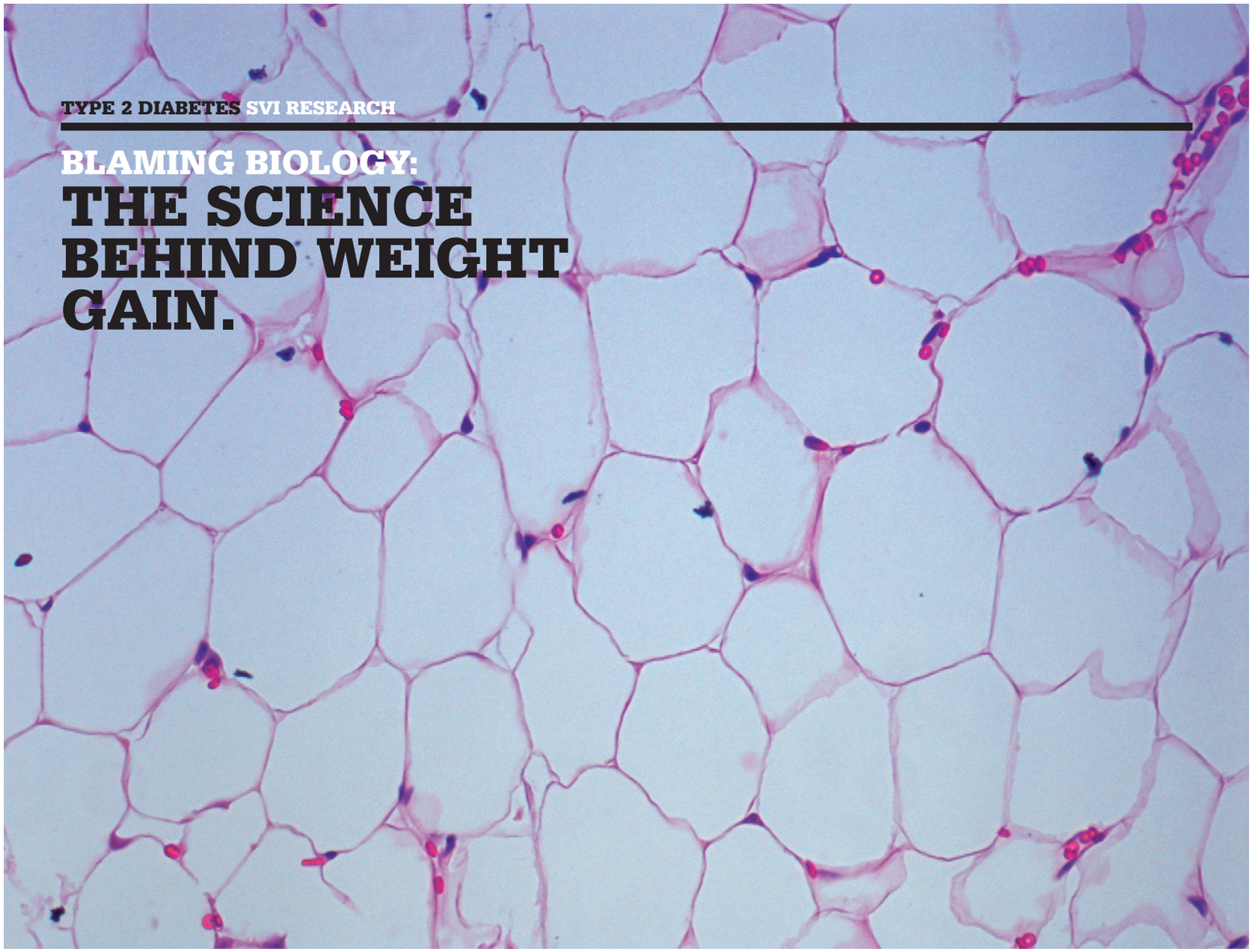
In order to prove this hypothesis John needed to create a genetically modified mouse in which the CAMKK2 switch was mutated in a similar fashion. His goal was to show that this one isolated change would result in similar behavioural changes as seen in humans with the mutation.

In a funding environment that is so competitive, it has become more difficult to get support for early stage research. John says that a grant where a mouse model of disease is already in hand is a much more compelling proposition for grant reviewers.

This is where the Angior Family Foundation stepped in. A grant from the Foundation in 2016 enabled John to do the work that he needed.

Thanks to the support, John has spent the last year examining the effects of the mutation on the behaviour of his mice. He is now a step closer to his goal of understanding how lithium works, with a longer term aim of developing safer and more effective therapies for debilitating mental disorders.

BLAMING BIOLOGY: THE SCIENCE BEHIND WEIGHT GAIN.



Dr Sandra Galic says that throughout history, being fat has generally been a good thing. Fat insulates from cold and provides a stockpile of nutrients when food is scarce.

But with obesity the prime driver behind the first modern decline in life expectancy, Sandra says that there is a compelling need to understand how our bodies manage the storage and expenditure of energy.

Sandra's most recent work sheds light on the metabolic processes that cause people to regain weight after dieting. She aims to use her findings to develop treatments that will help keep weight off for good.

"Most weight-loss strategies focus on creating a negative energy balance, where you burn more calories than you consume. However, these strategies frequently fail because our bodies compensate by reducing activity and increasing appetite," explains Sandra.

SANDRA'S MOST RECENT WORK SHEDS LIGHT ON THE METABOLIC PROCESSES THAT CAUSE PEOPLE TO REGAIN WEIGHT AFTER DIETING.

The team focused their study on a molecule called AMP-activated protein kinase (AMPK), which controls how the body generates and uses energy under different conditions.

They generated mice lacking a key step which regulates fat synthesis in the AMPK signalling pathway.

The researchers noticed that their mice lost weight when they were exposed to low temperatures. Normally, in these conditions mice increase their food intake to meet the energy demands of maintaining body temperature. Sandra's mice didn't do this even though their body temperature was normal. This indicated that the pathways that control heat production and regulate appetite are independent of each other.

The researchers then showed their mice were not able to respond to a hormone called ghrelin, which is known as the 'hunger hormone' for its role in boosting appetite. Ghrelin is thought to be responsible for the rebound weight gain that is seen after diet-induced weight loss.

Sandra hopes that her further research into understanding the nuances of these signalling pathways could lead to new ways to control appetite and achieve enduring weight loss. This could have major implications for those Australians who suffer the health consequences of obesity.

Image: A tissue section of white fat

CANCER

A DIVERSE GROUP OF DISEASES IN WHICH SPECIFIC CELLS BEGIN TO MULTIPLY OUT OF CONTROL, EVENTUALLY SPREADING TO OTHER PARTS OF THE BODY.

A leading cause of death worldwide

Risk factors include smoking, alcohol intake, UV radiation, family history and poor diet

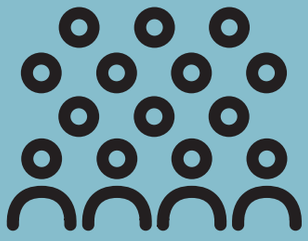
1 in 3 cases of cancer are considered preventable

AUSTRALIA

1 in 2 Australians will be affected in their lifetimes

Around 17,500 more people die each year from cancer than 30 years ago

In 2018, it is estimated that **138,321 cases** will be diagnosed

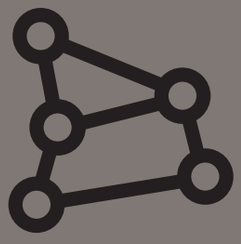


Cancer will kill more than 48,000 Australians in 2018

Lung cancer causes the most deaths and breast cancer is the most common type

$4.5 billion in direct health system costs

SVI at a glance:



SVI has five groups researching different aspects of cancer.

Research focuses on genetic causes, specific cancer types and understanding the role of stem cells in disease development.

Recent advances include understanding how the body repairs DNA damage, how stem cells in the bone marrow affect blood cell disease and the role of RNA editing in cancer development.

ONE BY ONE, ALL THE WOMEN IN DI'S IMMEDIATE FAMILY WERE DIAGNOSED WITH BREAST CANCER.

"In 1994, I had a routine mammogram that showed up a lump. Following the diagnosis of breast cancer, I had a lumpectomy, chemotherapy, radiotherapy and hormone therapy.

My family have always been very supportive. My mother's mother and her sister had breast cancer many years ago, but I was the first in my immediate family to be affected. One by one, however, the rest of the women in my family have been also been diagnosed with breast cancer.

My mother was diagnosed in 1996, and received a second diagnosis in 2001. My twin sister's turn came in 2003. In that same year, I was diagnosed again and had a bilateral mastectomy, chemotherapy and hormone therapy.

In 2005 my twin sister received another diagnosis and in 2006 my older sister was told she had the disease.

Genetic testing showed no sign of the BRCA1 or 2 genetic mutations – it is a real puzzle as to why we have all been affected.

I feel I cope well with the disease. I have a wonderfully supportive group of family and friends who try to keep me on an even keel!

In 1995 a support group was formed by friends who wanted to help me with my chemo and radiotherapy treatments. This group is called "Bosom Buddies", and 23 years later, is still going strong! (bosombuddies.org.au)

I have maintained an involvement with Bosom Buddies over the years, having served in different committee positions. I now belong to our Support Group, giving phone and personal support to women with breast cancer, and their families.

"IT WOULD ALSO BE WONDERFUL IF THE HIGH SUCCESS RATE OF BREAST CANCER TREATMENT – DUE TO THE SUCCESSES OF MEDICAL RESEARCH – COULD BE TRANSLATED INTO TREATMENTS FOR OTHER CANCERS."

Like many people who have had a cancer diagnosis – and particularly with my family history – there is always a concern about future recurrences. But it is important to keep it in perspective: I have a wonderful oncologist and GP and I have regular check-ups.

I think it would be great if we understood more about the genetic component of breast cancer so we could understand why families like mine are affected. It would also be wonderful if the high success rate of breast cancer treatment – due to the successes of medical research – could be translated into treatments for other cancers which have very low survival rates, like pancreatic cancer."

Image: Di (top left) with her mother and siblings

DR JACKI HERAUD-FARLOW IS MOTIVATED BY THE THRILL OF NEW DISCOVERIES AS WELL AS BY THE SUPPORT THAT SHE HAS RECEIVED.

Dr Jacki Heraud Farlow has a little more on her plate than the average early career researcher. She returned to SVI in February after maternity leave that followed the birth of her twin girls, Indiana and Frida. Her older daughter, Florence, turned 3 just before the twins were born.

On her return to work, Jacki was appointed as The Marian and EH Flack Fellow. The Flack Trust has worked with SVI to specifically tailor their support for Jacki. The Fellowship has been awarded for 2 years full-time, even though Jacki has returned to work, for the moment, on a part-time basis. The balance of funds will be used to pay a research assistant, enabling Jacki to maximise her lab presence for the research project, while still being able to devote time to her children.

Jacki's research in the Cancer and RNA Biology Laboratory focuses on a pathway that allows cells to identify the difference between 'self' and 'non-self'. This pathway enables a function called 'editing', by which the cell tags the molecules that belong to it. The presence of an unlabelled molecule is taken as a sign that there has been a viral infection and the cell is prompted to mount an immune response.

Jacki says, "Unfortunately, some children are born with mutations in this editing pathway, meaning their immune system reacts even though there is no infection present. This response results in profound neurodegeneration and loss of motor and communication skills early in life. There is currently no treatment for this disease, termed Aicardi-Goutières syndrome."

Jacki hopes that her research can shed light on the process in both normal cells and in those that carry a mutation in this important pathway.

Combining her busy research and family environments has led to a can-do attitude when it comes to life at home.

"My husband and I are both very fortunate to work in family-friendly environments with flexible work hours. We coordinate our hours, so the kids get time with both of us each day. A chest freezer with lots of ready-to-go meals helps too!"

Despite the difficulty of coordinating a research career with a particularly busy home life, Jacki is motivated by the thrill of new discoveries, and by the philanthropic support she has received.

"I am really lucky to have philanthropic support that takes into account my circumstances, increasing my chances of future funding success, but more importantly I believe, giving our research the best chance of coming to fruition."

UNRAVELLING THE MYSTERIES OF DNA.

Our DNA is constantly changing – during the course of evolution, genes have been created, mutated, duplicated and even lost. And while it is this ability to modify our DNA that lies at the very heart of our existence, our cells have also developed sophisticated mechanisms to protect DNA from changes that might cause problems.

SVI's Dr Andrew Deans has based his career around understanding this tension between stability and variability in our DNA. He believes that knowledge of these processes will help us to understand the underlying basis of cancer and develop new ways of treating the disease.

In 2017, Andrew and his colleagues, SVI's Associate Professor Jörg Heierhorst and Dr Wojciech Niedzwiedz from the Institute of Cancer Research in the UK, were awarded a 4-year NHMRC Project Grant to examine in detail one of the mechanisms by which the cell protects its DNA from mutation.

The grant focuses on the role of a protein called FANCM. Recently, it was shown that people with mutations in FANCM were predisposed to early-onset cancer, including head and neck cancers, leukaemia and breast cancer.

Andrew says, "We have shown that this protein – FANCM – plays a role in stopping the accumulation of what is called an R-loop: an area where the DNA double helix has been disrupted and exposed to damage."

> "OUR WORK WILL UNCOVER HOW FANCM SUPPRESSES CANCER, AND HOW IT PLAYS A MAJOR ROLE IN RESPONSE TO SEVERAL IMPORTANT CHEMOTHERAPY DRUGS IN CANCER TREATMENT."

Andrew compares it to a knitted scarf in which a loop of wool is sticking out. If the loop is not pulled back into the knit, it is more likely to become frayed or ripped. FANCM recognises that there has been a mistake and goes to the site of the problem, pulls and pushes on the loop to bring it back into place, and restores the integrity of the knit so that the whole scarf doesn't unravel.

In the work funded by the NHMRC, Andrew and his team plan to examine the way that FANCM works in detail. They will look at the proteins that partner up with FANCM to remove the loop and determine the consequences of disabling FANCM. They will also look closely at the relationship between FANCM mutation and breast cancer development.

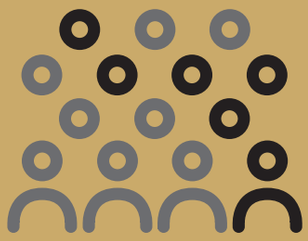
"Our work will uncover how FANCM suppresses cancer, and how it plays a major role in response to several important chemotherapy drugs in cancer treatment. Importantly, this will tell us why particular FANCM mutations predispose to cancer and help us improve the use of these chemotherapies."

Image: Chromosomes from a single cell, coloured with blue dye. The telomeres at the ends of the DNA are labelled in red and the centromeres, where the DNA is held together, are labelled in green.

CANCER SURVIVORSHIP

PEOPLE LIVING WITH, THROUGH AND BEYOND CANCER CAN BE AFFECTED BY UNANTICIPATED CONSEQUENCES OF THEIR TREATMENT.

Treatment can lead to a number of serious side effects, including fatigue, lymphoedema and increased risk of other cancers

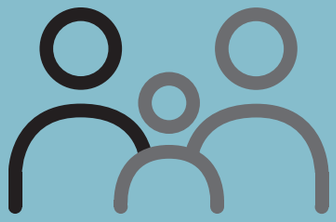


Two thirds of all cancer survivors will be over the age of 65 by 2020

Survivors who pass the 5-year milestone are living longer

AUSTRALIA

Over 1 million live with a cancer diagnosis

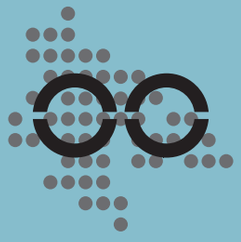


2 in 3 will live more than 5 years from diagnosis

Australia has one of the highest rates of cancer survival

The 5-year survival rate from cancer in Australia increased from 47% in 1987 to 66% in 2010

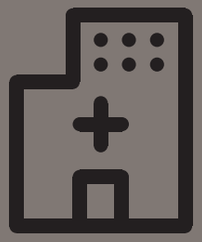
Fewer are dying although more people are being diagnosed



Breast cancer has a 5-year survival rate of 90% in Australia

SVI at a glance:

In 2015, the O'Brien Institute merged with SVI, to form the OBI Department of SVI.



Researchers in the OBI Department have strong clinical links and focus on practical application of research to patients.

Recent advances include harnessing stem cells to grow liver tissue outside the body and understanding the role of adult stem cells in the heart.

Image: Sheryl with her husband, David

AFTER TREATMENT FOR BREAST CANCER, SHERYL DEVELOPED LYMPHOEDEMA OF HER LEFT ARM.

"I had no symptoms, but I was 6 months late having my mammogram. A few days afterwards I had an ultrasound, which then led to an appointment to have a biopsy. Two days later I was speaking to a surgeon.

She told me I had an 11mm mass that was invasive and aggressive, and I would have to have a lumpectomy and chemotherapy. I opted for a double mastectomy and decided to have a breast reconstruction take place at the same time.

Ten days after my double mastectomy and reconstruction, I had 16 nodes taken out of the left armpit; a couple of days later my left arm was sore. I had developed what they call 'cording' in my arm and it started to swell. I saw a friend, Rosemary, who was a physiotherapist and specialised in treating people with lymphoedema. She worked on my arm and I got suppression sleeves; 3-4 weeks later the lymphoedema was under control, but I had to continue to wear a compression garment.

Then I started my chemo treatment; I had four sets of chemo 3 weeks apart. After the last session the lymphoedema came back. I went back to my physiotherapist for massage treatment every day, bought a pulse pump machine and purchased more compression garments. She told me that she knew of a world expert, Dr Ramin Shayan, who she had met at conferences. When I saw him on her suggestion, Ramin said he thought he could help me, as I had only had lymphoedema for 3 months at that time.

"I'VE HAD THE BREAST CANCER AND THE LYMPHOEDEMA AND NOW THEY'RE GONE – I'M BACK TO MY NORMAL LIFE NOW."

I went back home and in early December had permanent breast implants put in. I was back in Melbourne early the next year, where Ramin did 3½ hours microsurgery on my left hand. It has been fine ever since – no physio, massages, pain or compression garments needed! The top of my arm has gone down by 5-6cm and I feel normal again. I'm so lucky that my physio knew about Ramin.

I have absolutely no fear about my future health; in my mind I'm done and dusted. I've had the breast cancer and the lymphoedema and now they're gone – I'm back to my normal life now.

As for medical research, I'm blown away with how far it has come, and if medical researchers keep going the way they're going, we've got a fabulous future ahead for everybody. I truly believe there's hope for all of us; and that chronic diseases and other conditions can be eradicated."

CLINICIAN-RESEARCHER RAMIN SHAYAN'S WORK IS FOCUSED ON THE PEOPLE THAT THE SYSTEM FORGETS.

Clinician-researcher Ramin Shayan spends his research time trying to do himself out of a job. Ramin's work in SVI's O'Brien Institute Department is focused on understanding the molecular mechanisms of a condition known as lymphoedema – an increasingly common consequence of breast cancer treatment. He is also a highly sought-after plastic surgeon who has developed innovative surgical solutions for people with the condition.

"Happily, a growing number of cancer patients have had their life spared thanks to new therapies. But because of this there is also a growing cohort of people who suffer debilitating side-effects resulting from their treatment," says Ramin.

Lymphoedema occurs when removal of lymph nodes or damage to the lymphatics system during treatment for cancer – mainly breast cancer or melanoma – causes lymph fluid to build up under the skin.

The condition can be debilitating, with an affected arm in a very bad case weighing up to 6 kilos more than an unaffected arm.

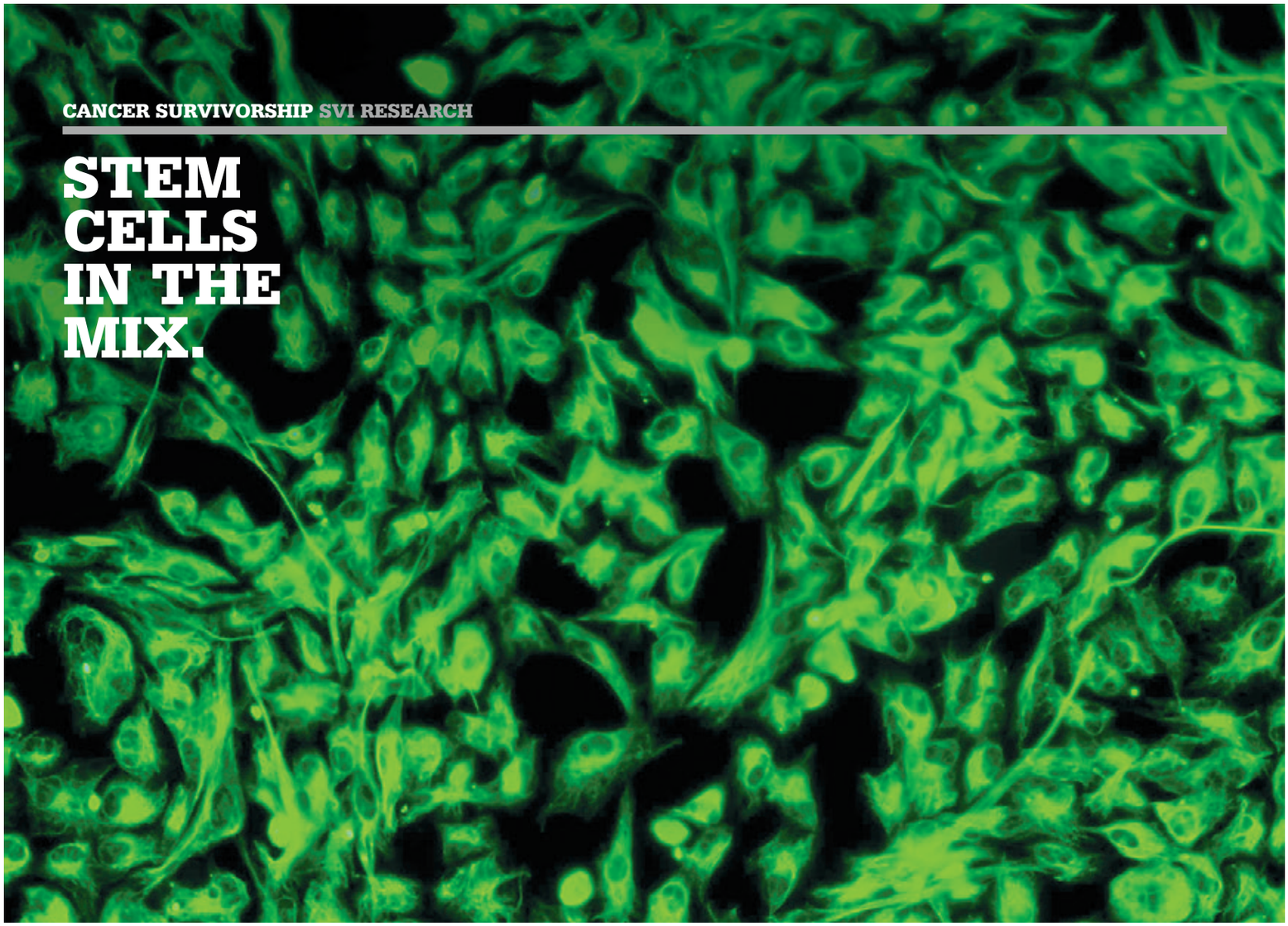
"Our approach to treating the cancer is so sophisticated, it is hard to imagine that these people often felt that they couldn't get anyone to acknowledge the consequences of that treatment as a serious problem. These people were 'medical orphans' – there was no-one who was dedicated to understanding what caused lymphoedema, much less finding a way to treat it," says Ramin.

Ramin's list of patients has grown steadily since 2013 when he started performing complex surgery on these patients – painstakingly repairing damaged vessels in surgical sessions that can take up to 6 hours.

"Surgery allows me to see the problem at a microscopic level, but it is limited by the size and type of surgical instruments available, by my manual dexterity and also by the resolution of our imaging. Molecular biology allows us to understand at a deeper level – at a cellular level – what is going wrong and will hopefully allow us to develop new ways to stop the problem from occurring in the first place."

He says that he would happily forego his surgical work on these patients if he could develop a therapy to solve the problem before it became so serious that surgery was required.

STEM CELLS IN THE MIX.



In the Cardiac Regeneration Laboratory, Dr Max Lim and his team can take a scraping of skin cells from a willing volunteer and put them in culture, exposed to appropriate conditions and a specific mix of growth factors. About a month later, the cells have grown to resemble heart muscle cells that constrict and contract in unison in the culture dish, beating to their own particular rhythm.

This 'reprogramming' of stem cells into heart cells is one of the ways that researchers would like to treat hearts that have been damaged by heart disease, or by the onslaught of chemotherapy, in the future.

But the cells generated in this manner, while they beat convincingly, do not appear to be able to do everything that is asked of them.

An alternative, if slightly more complicated strategy, is to harvest adult stem cells from the heart itself. When transplanted into damaged heart tissue, these adult cardiac stem cells work not by repopulating the damaged heart muscle with new cells, but by secreting factors which repair the damage done by heart attack.

> MAX LIKES TO THINK OF THE STEM CELLS AS LITTLE BIOLOGICAL FACTORIES.

In 2017, Max and his colleagues published a paper in the journal *Scientific Reports*, in which they painstakingly examined the factors that were released by adult cardiac stem cells that had been isolated from biopsies taken from patients undergoing heart bypass surgery.

The team had previously shown that the stem cells could powerfully influence other cells within the heart to promote cardiac repair and regeneration.

Max likes to think of the stem cells as little biological factories.

"We call the products of these cells the 'secretome' – the cells secrete a complex mix of beneficial factors. Our ultimate aim was to isolate the different components and assign an action to each of these."

Extensive analysis allowed the researchers to identify a large number of different factors in the mix. In fact, the breadth of the factors identified suggested to Max that they act in concert, rather than individually, to drive the observed repair and regeneration of heart tissue.

Max says that from a clinical perspective, harnessing the secretome of the stem cells could provide the benefits of stem cell therapies in a more controlled, reversible and safe manner than is offered by traditional stem cell transplantation.

"The challenge now is to identify the right mix of factors secreted by the cells so that we can mimic the effect with a drug cocktail."

Image: A population of human cardiac stem cells in culture

BONE DISEASE

BONE DISEASES DAMAGE THE SKELETON, MAKING BONES WEAK AND PRONE TO FRACTURE. THE MOST COMMON BONE DISEASE IS OSTEOPOROSIS.

Bone diseases include osteoporosis, osteogenesis imperfecta and the spread of cancer to bone

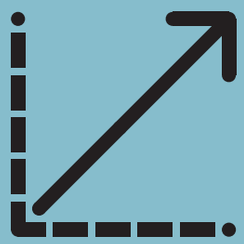
Risk factors for osteoporosis include poor diet and low activity levels, older age and some genetic factors

At 30, bones begin to break down faster than they are made

AUSTRALIA

2 in 3 over 50 are affected by low bone density or osteoporosis

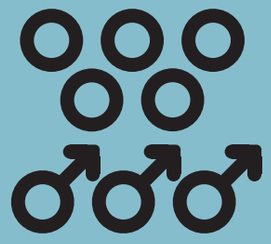
Every 3.4 minutes an Australian breaks, snaps or cracks a bone due to poor bone health



Bone health can be improved by weight bearing exercise, increased calcium intake and vitamin D

More women than men are affected by osteoporosis

$3 billion in direct and indirect costs each year is attributed to poor bone health



Men are twice as likely as women to die in the year after a hip fracture

SVI at a glance:

Research on bone at SVI has led to new understanding of how bone cells communicate, both with each other and with other cells in the body.

Researchers at SVI discovered parathyroid hormone-related protein, which plays a role in cancer spread to bone and in bone development in the embryo.

Recent highlights include increased understanding of the differences in bone development between men and women, detailed analysis of bone composition and how osteoporosis drugs affect bone growth.

Image: Jane with her youngest son, Liam

BEING DIAGNOSED WITH OSTEOPOROSIS AT 38 CAME AS A REAL SHOCK TO JANE.

"I went through premature menopause in 2006. My obstetrician recommended a bone mineral densitometry (BMD) scan, and I was subsequently diagnosed with osteoporosis. The diagnosis came as a real shock to me, not only due to my age, but also my excellent health record.

I was pretty upset at the time. I suspected I may have had a predisposition to osteoporosis, since my mum and my aunties had it. I thought I could have developed it in older age, but I was only 38 and had three young children. I'd always consumed foods high in calcium, exercised a lot, and never smoked.

In order to qualify for government-subsidised medication, I had to undergo X-rays. They found a fracture in the middle of my back, in the thoracic section of my spine. I do recall in 2000, at the age of 32, I lifted my two-month-old son out of his cot and experienced sudden, severe back pain. The pain continued for weeks on end, without respite. In hindsight that was my first fracture. At the time, I had no X-rays. No one suggested it, and no one was looking for osteoporosis, given my age.

A six-monthly injection and strict adherence to a healthy lifestyle is part of my osteoporosis management plan. I prioritise time for step aerobics and Pilates, only drink decaffeinated coffee, take calcium and vitamin D tablets daily, and even alight my train a few stops early to fit more exercise into my day.

I also attend a Melbourne osteoporosis support group to keep current with osteoporosis information and tips from other members.

As a nurse specialising in orthopaedics, for one day each week, I ensure patients admitted to hospital with minimal trauma fractures are screened for osteoporosis. I'm concerned about fracturing another bone, because I see what people go through. It can be quite debilitating, and I certainly don't have the time to be out of action.

I'm also concerned that any of my children could develop osteoporosis, with such a strong genetic predisposition from me. I'm forever educating them about eating well and encouraging them to be active.

"EVEN SINCE MY DIAGNOSIS, NEW TREATMENTS HAVE COME ONLINE..."

I hold great hopes for medical research in the area of bone health, especially for my children and generations to come. Even since my diagnosis, new treatments have come online, and it is my hope that one day a cure will be available."

ASSOCIATE PROFESSOR NATALIE SIMS IS HIGHLY SOUGHT AFTER AS A WORLD EXPERT IN BONE STRUCTURE.

Associate Professor Natalie Sims says that the design of the Eiffel Tower was based on the structure of bone. This isn't just the obsession of a single-minded scientist who sees her work reflected in the world around her. Apparently, Gustav Eiffel, when designing his now famous tower, was influenced by the work of anatomist Hermann von Meyer, who published some of the first detailed studies on the internal structure of the human thighbone.

Natalie says, "The pattern of struts and spars in the Eiffel Tower distributes weight and stress in the same way that the internal structure of bone – called trabecular bone – does. This means that our bones are light, but are still able to absorb the forces that occur when we jump a puddle or run for the tram."

Natalie says that while we need our bones to be hard, this feature poses some unique difficulties for researchers like her.

First, the nature of the tissue itself. "Because bone is so hard, we can't easily see what is happening inside it – it is particularly complicated to cut through bone without destroying its internal structure unless you use specialised methods. For obvious reasons, we also can't easily get samples from patients. Also, bone structure is different in every part of the body - you can't take a sample from the base of the Eiffel Tower and use that to understand what a bit at the top might be like. In the same way, what is happening inside say, the ankle, is often different to what is happening in another part of the skeleton."

Natalie says that as a PhD student, she spent a lot of time looking at bone slices down a microscope.

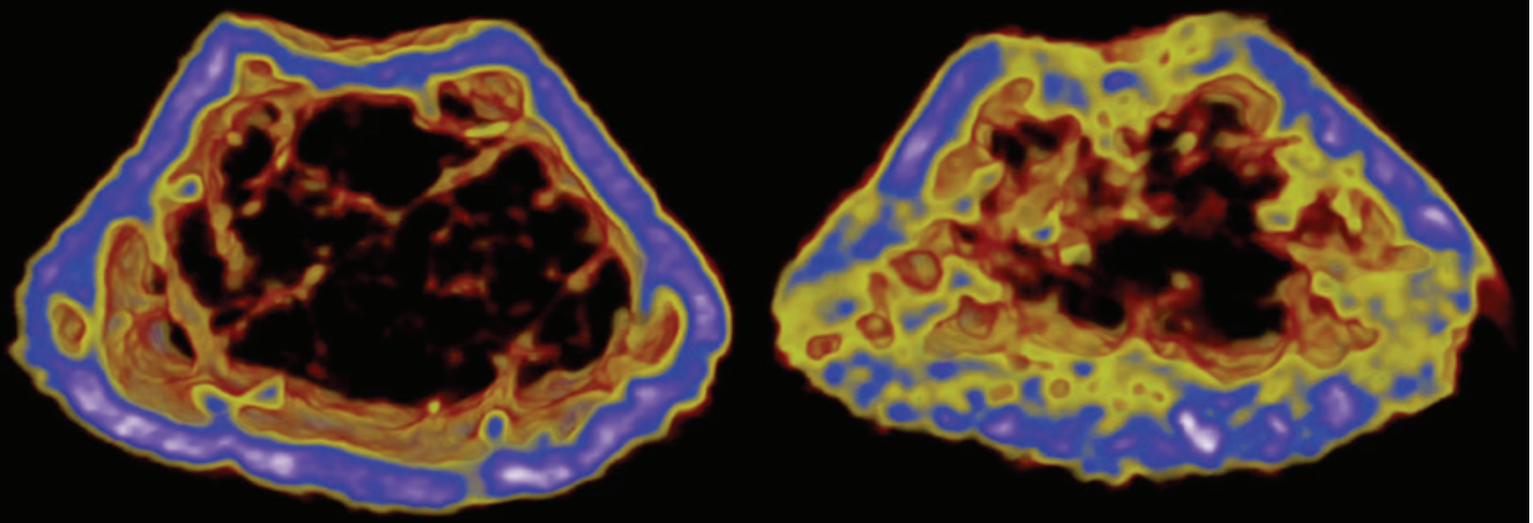
"Right about that time, people were starting to develop genetically modified mice and there was a real need for expertise in bone structure to help understand the influence of specific genes on that structure."

Thanks to the skills that she has developed since then, Natalie has been highly sought after as an expert on bone structure.

Much of her own career has been focused on studying the cells and signalling pathways that are responsible for the way that bone is composed. In 2018, she took on new responsibilities when she was appointed Deputy Director of SVI, an acknowledgement of her leadership role at the Institute, and of the high esteem in which she is held in the scientific community.

Recent studies from Natalie's team have used genetically modified mice and the power of the Australian Synchrotron to examine bone structure in unprecedented detail – detail which Hermann Von Meyer himself could not possibly have imagined.

TOUGH ON THE SURFACE.



If you slice a bone open, you'll find that it's a bit like a baguette – hard on the outside and porous on the inside.

The internal honeycomb-like structure of bone, called the trabecular bone, is covered by a thick crust – an outer shell of hard cortical bone.

While the signals between bone cells that control the growth of trabecular bone are relatively well understood, until recently, the signals that control cortical bone formation were unknown.

In 2017, researchers from SVI's Bone Cell Biology and Disease Unit identified a key pathway that is responsible for making the external surface of male bones stronger than that of female bones.

In research published in the journal *Nature Communications*, the team showed that removing a signaling protein called SOCS3 from specialised bone cells of mice caused a delay in the formation of the external surface of the bone.

Male mice recover from this impairment at puberty, but female mice do not. This is because testosterone interacts with other proteins that enable the process of cortical bone formation to continue in male mice.

Senior author of the study, Associate Professor Natalie Sims, says that treatments for osteoporosis are best at preventing vertebral fractures, which is the most common site of fracture.

"These treatments are effective because they work by increasing the strength of the trabecular bone. However, they are less successful at strengthening cortical bone. Strengthening cortical bone would help prevent fractures in the hip and wrist, which are common in sufferers of osteoporosis."

"Most research that has identified genes controlling bone mass has focused on the trabecular bone," continues Natalie. "Until this study, the specific signals that control the strength of cortical bones were unknown, and we also didn't know why some people develop stronger cortical bones than others – it was a big black box."

> "A MAJOR GAME CHANGER IN THE FIGHT AGAINST OSTEOPOROSIS WOULD BE THE DEVELOPMENT OF THERAPIES THAT PREVENT THE EROSION OF CORTICAL BONE."

Having identified this new role for SOCS3, the researchers now wish to identify the other signalling molecules that are involved in cortical bone production.

Natalie says, "A major game changer in the fight against osteoporosis would be the development of therapies that prevent the erosion of cortical bone."

Image: Normal bone (left) has a thick and dense outer shell (blue), but bones from mice lacking SOCS3 have an outer shell that is full of holes and low density patches (yellow).

ALZHEIMER'S DISEASE

A PROGRESSIVE NEURODEGENERATIVE DISEASE THAT AFFECTS MEMORY, THINKING & REASONING.

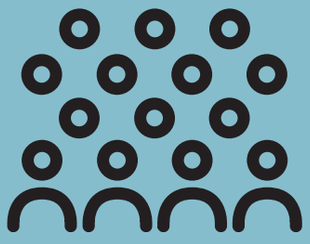
Single most common form of dementia

Memory loss that disrupts daily life is a common symptom

At any age adults can be affected, but usually occurs after age 65

AUSTRALIA

Dementia is a leading cause of disease burden in Australia



900,000 people will be living with dementia by 2050 if no new treatment is found

Dementia is the second leading **cause of death** in Australia

Every 6 mins there is a new case of dementia in Australia

$15 billion, the expected cost of dementia in 2018

No treatments are currently available to stop the progression of Alzheimer's

SVI at a glance:

Research in the Structural Biology Unit centres on understanding the structure of proteins and using this knowledge to develop new disease treatments.

The group researches neurodegenerative diseases with a major focus on Alzheimer's.

One exciting new project is aimed at harnessing the body's own mechanisms to counter the development of Alzheimer's.

Image: Althea with her grandson, Art

WANDA'S MOTHER-IN-LAW, ALTHEA, HAS ALZHEIMER'S DISEASE AND HAS LIVED WITH HER SON AND DAUGHTER-IN-LAW FOR THE LAST 3 YEARS.

"Althea would frequently call my husband in the middle of the night, asking when he would be coming over and declaring that she had no food despite us delivering her groceries earlier in the week. In early 2014 her phone calls for help became more urgent."

The main factors that resulted in her diagnosis were loss of memory skills, wandering, and heightened episodes of anger and psychotic behaviour towards her and medical staff at the memory clinic. The aged care assessment service assessor deemed that she was no longer able to live on her own; she then came to stay with us.

Althea's mother died early and her father left his children with the grand-parents, so we don't know if there was a history of dementia in the family. Her other children, who both live interstate, find it very difficult to deal with her memory loss, repetition of the same conversation over and over again, and her lack of understanding of who they are.

Althea has been on a mix of prescribed medications since 2015. She doesn't know she has Alzheimer's Disease or what it means. At times we feel she realises that she has lost her memories.

"SHE IS FULLY RELIANT ON SOMEONE ELSE MAKING ALL HER DECISIONS FOR HER."

Althea is now 91 years of age and is grossly demented. She is content in the moment and loves listening to music and reading children's books out aloud, especially Dr Seuss. She is speaking less and less. She attends a day centre during the week and enjoys these social outings immensely. She is fully reliant on someone else making all her decisions for her and directing her to dress, shower, go to bed and eat her meals and more. She is still continent.

We worry about how to manage her end-of-life process. We have made a lot of concessions and adjustments to our lives in order to accommodate Althea's needs and have undertaken numerous courses and family counselling to understand the impact of having an elderly person with Alzheimer's living with us.

Alzheimer's Disease relentlessly steals memories. It is a degenerative disease that is debilitating not just to the individual affected, but also to their family."

JASMINA MARKULIC SAYS THAT IT IS EXCITING TO BE WORKING IN AN AREA THAT IS THE FOCUS OF SUCH INTENSE INTEREST.

Growing up in the suburbs of Adelaide, PhD student Jasmina was extremely close to her great-grandmother.

"I was the oldest grandchild and so was blessed to have more than 20 years with my great-grandmother, who lived with us while I was growing up. Sadly, during my undergraduate studies in science, she was diagnosed with dementia."

"It was never a question of who would care for her as her health began to deteriorate in her later years; I helped my mum and my grandmother with doctors' visits, hospital stays and at-home care. It was difficult to witness the change in the most important person in our lives."

"Her passing was very difficult for me. She was one of the strongest and most fierce women I have known; she continues to motivate me and inspire me every day. That is why, when Professor Michael Parker offered me the opportunity to join SVI to focus my studies on neurodegenerative diseases, I knew this was where I was meant to be."

Jasmina's PhD focuses on neurodegenerative disease, including Alzheimer's and Parkinson's Disease.

Jasmina says, "One in ten Australians over 65 live with dementia and that number will continue to increase as our population ages. After dementia, Parkinson's is the most common neurodegenerative disease, with approximately 80,000 Australians affected. But if you know someone who is suffering from these diseases, you know it is not about numbers: this is about people's lives."

Jasmina says that it is exciting to be working in an area that is the focus of such intense interest.

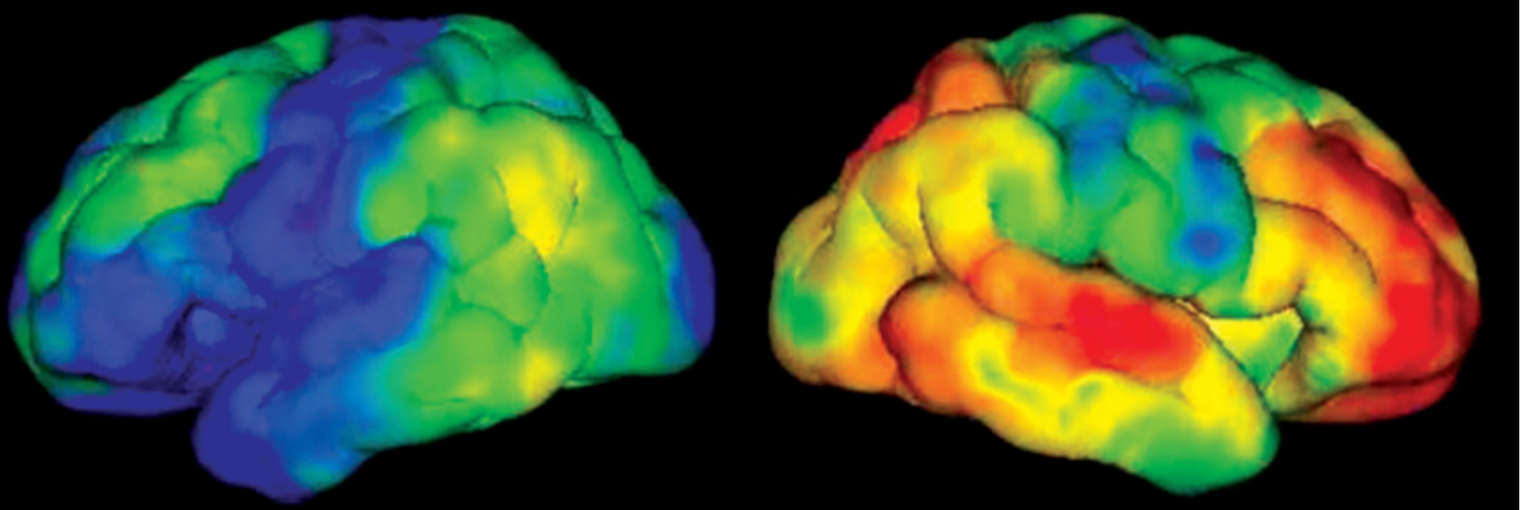
"We know that both Alzheimer's and Parkinson's are linked to accumulation of toxic protein deposits in the brain. We also know that the brain has natural ways of removing these deposits."

"I am working on understanding the 3D atomic structure and the function of the proteins involved in removing toxic proteins that build up in the brain. The aim is to design drugs to enhance this naturally occurring process. This could lead to a slowing of the disease, or even its reversal."

Jasmina has also been inspired by the trust that has been put in her by supporters who are investing in her career. Her PhD Scholarship is sponsored by Rotary Health International, her Top-up Scholarship has been generously donated by SVI supporter Elaine Walters, and support from the Yulgilbar Foundation has enabled her to travel to international conferences and keep up to date with cutting-edge research findings in her field.

"Importantly, attending conferences has provided me with the opportunity to meet and foster networks with some of the world's leading researchers and clinicians as well as other students in the field. I am indebted to the generous supporters who have enabled me to take advantage of opportunities that will be of great significance to my research career."

PARTNERSHIP OF NEW THINKING.



A degenerative brain condition, Alzheimer's Disease is the most common form of dementia. The disease is characterised by memory loss and increasingly impaired cognitive function. It is most commonly diagnosed in people over 65 years of age, although an early-onset variant may occur earlier. In 2018, it is estimated that the costs of dementia in Australia will reach $15 billion.

While the the causes of Alzheimer's Disease aren't yet fully understood, its effect on the brain is clear: Alzheimer's damages and kills brain cells. Currently available medications only treat the symptoms of the disease and the race is on to find new treatments, particularly as our population ages.

Researchers in SVI's Structural Biology Unit are focusing on finding new ways to treat the disease.

Head of the Unit, Professor Michael Parker, says, "Without new treatments, it is expected that something like 900,000 Australians will be affected by dementia in 2050."

> "THE BURDEN OF ALZHEIMER'S ON OUR AGEING SOCIETY IS EVER-INCREASING, SO THERE IS A GREAT NEED FOR EFFECTIVE TREATMENTS."

In 2017, SVI entered into a collaboration and license agreement with Janssen Pharmaceuticals, Inc. ("Janssen"), one of the Janssen Pharmaceuticals Companies of Johnson & Johnson. The partnership is aimed at developing and commercialising small molecule modulators of microglial function and inflammation, with the aim of reducing amyloid plaque burden and Alzheimer's Disease severity. The collaboration was facilitated by Johnson & Johnson Innovation.

The collaboration leverages SVI research capability and the expertise of Michael Parker – one of Australia's leading structural biologists – with Janssen's drug discovery and development expertise.

SVI Director Professor Tom Kay says, "We welcome this collaboration with Janssen to develop a novel class of Alzheimer's therapies. SVI is proud of its research excellence and we are excited to partner with Janssen, a pharmaceutical industry leader in neuroscience research and development. At SVI, we continue to work to translate our fundamental discoveries into future medicines to improve health outcomes."

Michael says, "The burden of Alzheimer's on our ageing society is ever-increasing, so there is a great need for effective treatments to improve the quality of life for people not only in Australia, but throughout the world. We look forward to combining our expertise with Janssen's drug discovery and development capabilities."

Image: PET scans of a living human brain from someone with Alzheimer's showing low levels (blue) and high levels (red) of the amyloid beta peptide (left); and hyperphosphorylated tau, another pathological hallmark of Alzheimer's disease (right). Image courtesy of A/Prof Victor Villemagne, Dept of Molecular Imaging & Therapy, Austin Health

SVI STAFF

Executive

Prof Thomas WH Kay (Director)
A/Prof Natalie Sims (Associate Director)
A/Prof Jörg Heierhorst (Associate Director)
Prof Bruce E Kemp (Pehr Edman Fellow)
Prof Jack Martin (John Holt Fellow)
A/Prof Louise Purton (Associate Director)
A/Prof Helen Thomas (Associate Director)
A/Prof Carl Walkley (Associate Director)

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Prof Michael Parker
Dr Brett Bennetts
Dr Sophie Broughton
Karen Steffi Cheung Tung Shing (PhD Student)
Dr Michelle Christie
Gabriella Crespi
Dr Urmi Dhagat
Larissa Doughty
Dr Michael Gorman
Nancy Hancock
Dr Stefan Hermans
Dr Jessica Holien
Dr Jacinta Holmes
Bronte Johnstone (Honours Student)
Dr Sara Lawrence
Jasmina Markulic (PhD Student)
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Dr Craig Morton
Dr Tracy Nero
Dr Lorien Parker
Janelle San Juan
Brendan Stephenson (Honours Student)
Dr Claire Weekley

Immunology and Diabetes Unit
Prof Thomas Kay
A/Prof Helen Thomas
Dr Tom Brodnicki
Dr Mark Chong
A/Prof Stuart Mannering
Dr Satoru Akazawa
Matthew Andrews (Masters Student)
Elena Batleska
Nicole Bleasdale
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Bill Chow (U/Grad Student)
Marie Kruse Christensen (Masters Student)
Marie Tessa Doger de Speville (U/Grad Student)
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A/Prof John Slavin
A/Prof Gregory Steinberg

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Prof Harshal Nandurkar
A/Prof Evange Romas

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Nadeeka Bandara, PhD Charles Sturt University, Wagga
Gaurang Jhala, PhD University Melbourne
Nick Scott, PhD University Melbourne
Christina Vrahnas, PhD University Melbourne

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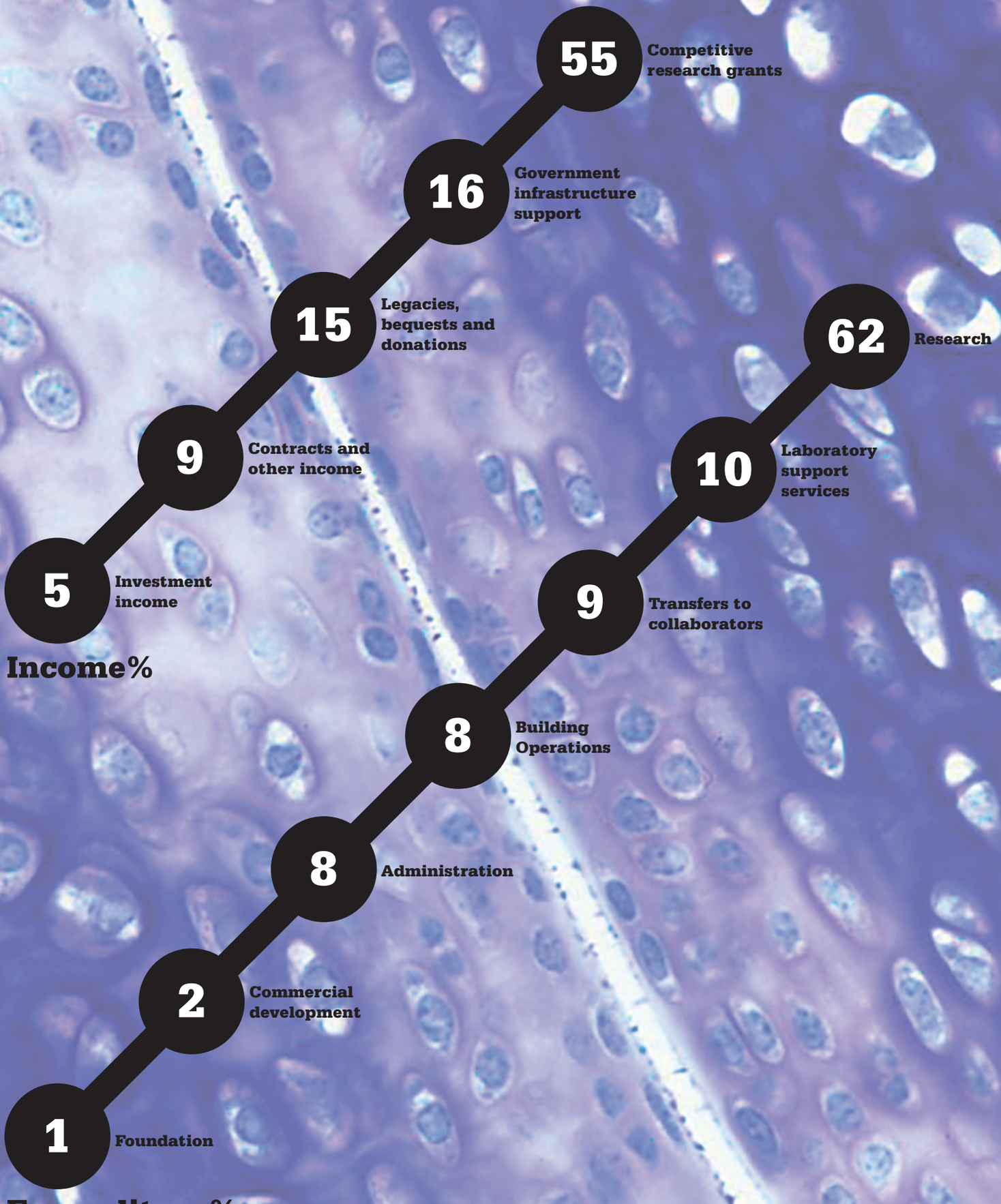
FINANCIAL SNAPSHOT



STATEMENT OF FINANCIAL POSITION AS AT 31 DECEMBER

	2017 ($)	2016 ($)
ASSETS		
Current assets	14,736,055	15,343,925
Non-current assets	18,294,718	16,777,736
TOTAL ASSETS	**33,030,773**	**32,121,661**
LIABILITIES		
Current liabilities	7,093,134	7,045,571
Non-current liabilities	148,957	141,900
TOTAL LIABILITIES	**7,242,091**	**7,187,471**
NET ASSETS	**25,788,682**	**24,934,190**
EQUITY		
– Retained surplus	24,304,706	24,021,076
– Reserves	1,483,976	913,114
TOTAL EQUITY	**25,788,682**	**24,934,190**

STATEMENT OF PROFIT OR LOSS AND OTHER COMPREHENSIVE INCOME FOR THE YEAR ENDED 31 DECEMBER

	2017 ($)	2016 ($)
Revenue	15,686,695	14,462,724
Other income	6,722,197	7,802,432
TOTAL REVENUE	**22,408,892**	**22,265,156**
Consumables and general research expenses	(3,817,160)	(4,084,747)
Employee benefits expense	(12,694,951)	(12,403,021)
Depreciation and amortisation	(1,692,191)	(2,192,558)
Administration expenses	(1,838,479)	(1,603,381)
Transfers to collaborators	(2,082,480)	(1,959,489)
TOTAL EXPENSES	**(22,125,261)**	**(22,243,196)**
Surplus/(Deficit) for the year	283,631	(21,960)
Other Comprehensive income (loss):		
Net gain/(loss) on revaluation of financial assets	570,861	(421,415)
Total Comprehensive income for the year	**854,492**	**443,375**
Total Comprehensive income attributable to members of the entity	**854,492**	**443,375**

NOTE 1: GOVERNMENT GRANTS

National Health and Medical Research Council:		
– Independent Research Institutes Infrastructure Support Scheme	1,462,680	1,118,987
– Research grants	7,548,435	7,050,242
Australian Research Council	365,171	608,703
Victorian State Government – Operational Infrastructure Support Program	1,866,017	1,608,868

The summary financial information shown above does not include all the information and notes included in the entity's statutory set of financial statements. The full set of Statutory Financial Statements can be obtained upon request to the Chief Financial Officer. The Statutory Financial Statements comply with the Australian Accounting Standards and an unqualified audit opinion was issued by the auditors, William Buck Audit (Vic) Pty Ltd.

SVI FOUNDATION REPORT

We are deeply grateful to all the people in our community who make regular gifts, hold fundraising events for research, remember the Institute in their Wills, and ask for donations in memory of a cherished loved one. Our donors are the driving force behind our progress.

SVI was born through an act of philanthropy – Jack Holt's generous gift in his Will, in 1951 – which led to the opening of the Institute in 1958. Sixty years on, the Institute's future largely depends on a new generation of philanthropic partners stepping forward to support the bold and collaborative science undertaken here.

Thanks to the generosity of nearly one hundred supporters, SVI's Discovery Fund has reached its goal of $5 million. The fundraising campaign has created a permanent endowment to support the Institute's fundamental research into common diseases like cancer, osteoporosis, type 1 and type 2 diabetes, and Alzheimer's. We congratulate SVI Foundation Board member, and Discovery Fund Committee Chair, Christine Tarascio, for her exceptional support and tireless advocacy for this initiative.

In 2017, we partnered with 5Point Foundation to create a Fellowship at the Institute, named in honour of Christine Martin, the wife of former Director, Jack Martin. The Fellowship is designed specifically to support outstanding female researchers to continue their medical research after a career disruption. The structure of the Institute's leadership team today is remarkable in the sector, in that our Deputy Director and two of our three Associate Directors are women. In addition, we have five female laboratory heads, seven female senior researchers and 11 female junior post-doctoral researchers, with expertise across a range of research fields. This demonstrates how philanthropy in partnership with an institute willing and able to support its female leaders can lead by example.

The ongoing generosity of Tony Schiavello has been crucial in funding new technology at St Vincent's Institute. Leading-edge technology has been a feature of the Institute since the early days, when the inaugural director, Pehr Edman, designed and produced a machine that automated protein sequencing and became widely used in laboratories across the world. Tony Schiavello's support provides vital funding for new scientific tools that enable our dedicated, curious and skilled group of scientists to conduct high-quality research.

We seek to be the best that we can be because we believe our mission to make discoveries to improve the health of the community is profoundly important. To that end, the SVI Foundation's goal is to help current and prospective St Vincent's Institute donors understand the importance of their support and the extraordinary impact they can have by powering our scientists' work. We look forward to sharing with you our passion and commitment for advancing health and making a significant difference to the lives of Australians now and for future generations.

Karen Inge
Chair, SVI Foundation

Kate Barnett
Chief Executive, SVI Foundation

DISCOVERY FUND

"THERE IS A LARGE PERSONAL MOTIVATION FOR WANTING TO ENSURE SCIENTISTS HAVE ENOUGH MONEY TO TACKLE COMMON DISEASES."

Image: Christine Tarascio with her husband, Sam

Christine Tarascio's motivation is simple. She wants the SVI Discovery Fund she established a little more than a decade ago to help scientists continue their research into the most common and crippling of diseases.

But, like all great causes, there is a personal impetus to her ambition.

"One of my sons, Sam, has psoriatic arthritis, and I have seen the way he has had to manage that condition," Christine says.

"My father also died of lung cancer. There is a large personal motivation for wanting to ensure scientists have enough money to tackle common diseases."

It's a personal motivation that isn't confined to Christine, either, with many of the 100 or so members and donors to the Discovery Fund motivated by a close brush with disease.

"It's a theme I have noticed with a lot of the supporters of the Fund," says Christine, who is Chair of the Discovery Fund and an SVI Foundation Board member.

"They have all been touched in some way by illness or disease, whether it's a close friend or family member who is sick. Sometimes they have had therapies or treatments themselves that would not have been possible without medical research. It becomes a big motivator in their commitment to the Fund."

It partly helps explain, too, the exponential growth of the Fund since its inception.

Earlier this year, the endowment fund exceeded its milestone of $5 million, helping to ensure researchers can be supported in perpetuity.

"We started off by inviting people to give $10,000 for 5 years, with each donating $50,000 all up," Christine notes.

"We now have 45 members of the Fund and many are on to their third term of donations."

During the early years of the popular annual Discovery Fund lunch, Christine would host the donors and scientists at her home.

"I would push the furniture out of the living room and into one of the bedrooms," she says.

"Then the gathering got too big, so now we go to a restaurant and then back home, which is where the real party starts. The donors love hearing from the scientists, who are unbelievably humble and thankful for the money we're raising, but, really, we're the ones thanking them for the important work they're doing."

The endowment fund model will ensure a steady income stream for SVI researchers by enabling a reliable cash injection through income generated from the Fund.

"In a way, it's filling the gaps in traditional grant funding," Christine says.

"You need to have a grander vision and look for ways to guarantee income further down the track."

Christine is not about to rest on her laurels either, and can't see why the Fund cannot reach a $100 million milestone one day.

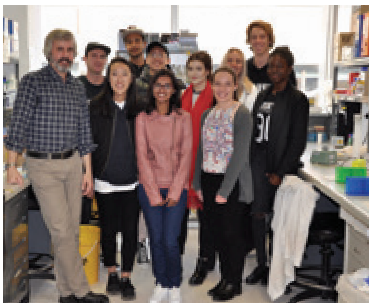
"The greater the endowment fund, the greater the income for the scientists," she says.

"I can feel that it's going to be a phenomenal success. I really do believe that the sky is the limit."

A YEAR IN PICTURES

SVI's 60th anniversary year provided memorable opportunities for our community of researchers, students, alumni and supporters to celebrate.









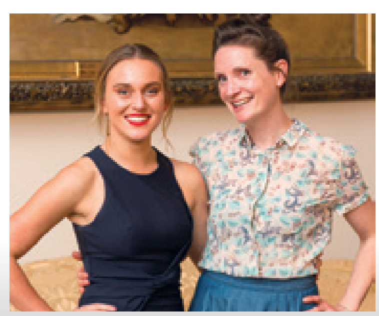










2017-2018











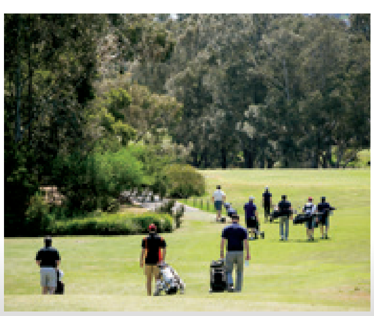








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Wolf Foundation
Maxwell Woods
Adam Woolcock
Adam Wulff
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Edward & Mandy Yencken
The Yulgilbar Foundation
Toni Zagame & The Late Vic Zagame
Zig Inge Foundation
Leon Zwier
15 Anonymous

Estates

Estate of The Late Geraldine Nicoll
Estate of The Late Margaret Littledale Tutton
Estate of The Late Hilton John Nicholas

Whilst every endeavor is made to ensure accuracy, we apologise for any unintended errors or omissions. If you have any queries please contact Kate Barnett, Chief Executive, SVI Foundation on (03) 9231 3265 or kbarnett@svi.edu.au

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