2018–2019

SVI YEAR

SVI
ST VINCENT'S INSTITUTE
MEDICAL RESEARCH

RESEARCH. DISCOVERY. IMPACT.

THIS IS SVI
SVI is an independent medical research institute that conducts research into the cause, prevention and treatment of diseases that are common and have serious effects on health. We strive, through our research, to help alleviate the enormous financial, emotional and physical impacts of these diseases on individuals, their families and the community.

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.

St Vincent's Institute acknowledges the Aboriginal lands on which we live and work and pays respect to Traditional Owners, ancestors and Elders.

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DIRECTORS' REPORT

Australians rank amongst the healthiest people in the world.

However, we face some sombre truths.

An Australian currently spends 11 years in ill health over a lifetime. Our ageing population will be increasingly affected by health issues, including neurodegenerative disorders, type 2 diabetes and heart disease, and other chronic diseases.

The research carried out at SVI is based on the strong belief that many of these challenges can be met only with a greater understanding of disease processes.

Success in delivering this impact would not be possible without our world-class scientists.

In early 2019, SVI researchers Helen Thomas and Natalie Sims were appointed as honorary Professors by the University of Melbourne. Helen and Natalie play significant roles at the Institute, both as leaders in type 1 diabetes and bone disease, respectively, as well as on the Institute's leadership team.

Another highlight in the last 12 months was Professor Bruce Kemp being honoured as an Officer of the Order of Australia for his distinguished service to biomedical research, particularly the study of protein phosphorylation. Fittingly, this fell on the 30th anniversary of Bruce's arrival at the Institute.

The SVI Board and Foundation Board also make a vital contribution to the Institute. We are very grateful for the contribution of JT

Macfarlane and Paul Holyoake, who recently retired from the Institute Board after many years of service, and Simone Carson, who retired from our Foundation Board in 2018.

Our relationships form an important foundation of our success. In 2018, we established a joint appointment with the University of Melbourne for newly recruited bioinformatician, Dr Davis McCarthy, who returned to Australia after 7 years in the UK. This was facilitated by a generous donation from SVI supporters Paul Holyoake and Marg Downey.

We also work closely with our campus partner, St Vincent's Hospital Melbourne, and its clinicians, who have first-hand knowledge of the day-to-day challenges of illness. The relationship between these experts and our researchers is invaluable. We have continued to work with the Hospital throughout 2018 and 19 on our partnership centred around the Aikenhead Centre for Medical Discovery.

"None of our achievements would be possible without support from the community."

This initiative has recently received significant Federal Government funding to add to previously committed State Government support.

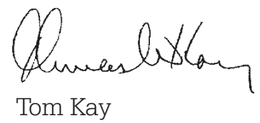
None of our achievements would be possible without support from the community. This includes our donors and volunteers, as well as people who are willing to share their stories and act as advocates for others affected by disease.

Philanthropic support is particularly important. At all stages of a researcher's career it gives a huge boost to see that others value what we do and are prepared to help. At SVI, we are particularly focused on philanthropic funding for the next generation of leaders, who are supported through our Rising Stars Program.

Philanthropy also helps to recruit new researchers and to provide our scientists with cutting-edge research technologies that help them and others to achieve research outcomes and contribute to our goals of improved understanding and treatment of disease.

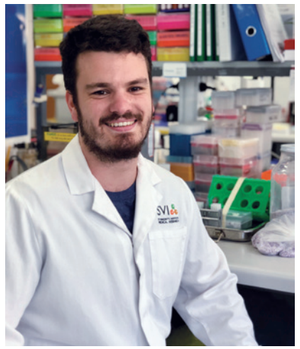
Thanks also to the State Government of Victoria for their Operational Infrastructure Support Scheme, the Australian Government, the Board of St Vincent's Health Australia and the Trustees of the Mary Aikenhead Ministries for their ongoing support of our research efforts.

Tony Reeves
Chair, SVI Board



Tom Kay
Director

YEAR IN FOCUS



THE HEART OF THE PROBLEM

In early 2019, Dr Jarmon Lees, from SVI's OBI Department, was awarded a Young Investigator Grant by the US-based National Ataxia Foundation. Friedrich's Ataxia is a genetic disorder that causes progressive nervous system damage and movement problems. Heart failure is the leading cause of death in people with the disease. Jarmon will use stem cells from people with Friedrich Ataxia to find out what goes wrong within the heart of people with the disease.

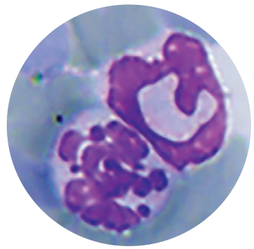
SVI CELEBRATES 60 YEARS

Wednesday 23rd April 1958 saw the opening of the first laboratories at SVI. "During our 60th year, we have celebrated with those people who have helped fulfil the vision of our founder, successful racehorse trainer and philanthropist Jack Holt, to provide hope to those who suffer with diseases that remain, unfortunately, all too common," said SVI Director, Professor Tom Kay.

Breaking the glass ceiling

In early 2019, SVI researchers Natalie Sims and Helen Thomas were appointed as honorary Professors by the University of Melbourne. Helen says that when she started out in research, her aims were short term, but this has changed over time. "I have met many people with type 1 diabetes, and parents whose children have the disease. The idea that I can personally make a difference is a big motivation." Natalie says that doing research requires an active choice every day. "Every grant that you write; every paper that you produce; every experiment that you run; it's a commitment that you're going to finish that. When you get new data, that reminds you why you're still in the game."

NEW RESEARCH INTO BLOOD CANCER



Research from Associate Professor Carl Walkley's laboratory in 2018 delved into the causes of myelodysplastic syndrome – a type of blood cancer which has limited treatment options. In a publication in the prestigious journal *Blood*, Carl's team showed how a gene called SRSF2 is involved in the development of the disease. By creating a mouse model in which SRSF2 is mutated, the researchers recapitulated the symptoms seen in humans with the disease, and they now have a valuable tool with which to test new treatments.

A TEAM EFFORT

Professor Bruce Kemp was included in the 2019 Australia Day Honours List for his distinguished service to biomedical research. Bruce's career, for which he is internationally recognised, has been based around a particular type of protein called a kinase. Bruce says, "This recognition wouldn't have been possible without the postdocs, students and colleagues who have been involved along the way - my success has really been the result of a lot of hard work by many others."

PHILANTHROPY FUELS BIOINFORMATICS APPOINTMENT

In late 2018, Dr Davis McCarthy joined SVI as Head of the new Bioinformatics and Cellular Genomics Laboratory, as a joint appointee with the University of Melbourne. The position was facilitated by a generous donation to the University by former SVI Board member Mr Paul Holyoake and his wife, Ms Marg Downey. Prior to joining SVI, Dr McCarthy was at the European Bioinformatics Institute in the UK. In his time overseas, Dr McCarthy worked at the interface of cutting-edge statistics, computing and cell biology. He brings this unique expertise back to Australia.

Action on HTLV-1

SVI's NRL division has been working with partners, including at the Baker Heart and Diabetes Institute and Flinders Medical Centre, to improve the outcomes for indigenous Australians living with human T-lymphocytic virus (HTLV-1). Indigenous communities in Central Australia have the highest-reported rates of HTLV-1 in the world. The virus can cause severe health conditions, including leukaemia, and can affect the lungs and nervous system. The NRL's expertise has supported both making the diagnosis and the development of new techniques for monitoring the disease.

IF YOU COMBINED ALL OF THE INSULIN-PRODUCING CELLS IN YOUR BODY IN ONE PLACE, THEY WOULD FILL A SINGLE TEASPOON

In a healthy person, insulin-producing cells clump together in groups called islets which are scattered throughout the pancreas. The pancreas itself is buried deep inside the abdomen.

In someone developing type 1 diabetes, the body's immune system comes to see its insulin-producing cells as a threat. Like an elite special operations force sent in to eliminate the wrong target, immune T cells gather their forces, infiltrate and sweep through the pancreas, specifically killing the cells that make insulin.

It is not known why the T cells receive the message to kill the body's own cells. Also unknown is the sequence of the events that leads to their death.

Type 1 diabetes places an extraordinary burden on individuals and their families.

TYPE 1 DIABETES

The Immunology & Diabetes Unit comprises the largest group of Australian researchers focused on type 1 diabetes. Their goal is to understand how and why the immune system attacks insulin-producing cells. They are using this information to develop safe and effective ways to stop the disease.

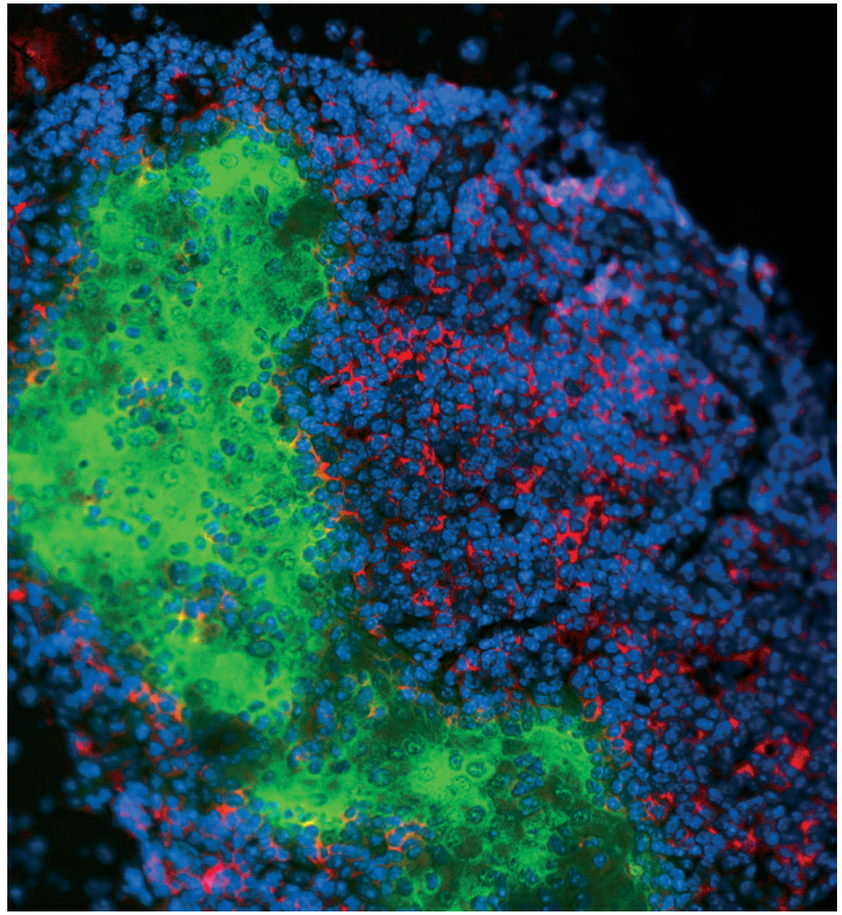


Image: Immune T cells (red) in the process of destroying insulin-producing cells (green) in a pancreatic islet

FINDING A NEW PURPOSE

Developing a new medicine is a fraught process.

On average, bringing a new drug to market costs $2-3 billion and takes more than 13 years of intense study and clinical trials. Many drugs fall at one or other of the hurdles put before them – they have unintended side effects, or are toxic, or patients don't show the benefits that have been seen in animal models of disease.

Because of the great investment of time, money and resources spent on getting a drug to the clinic, researchers are increasingly combing through the medicine chest to identify approved drugs that might work for a different indication.

Professor Helen Thomas

This is why, when researchers in SVI's Immunology & Diabetes Unit learnt that a drug had been approved to treat the autoimmune disease rheumatoid arthritis, they were excited.

Head of the Unit, Professor Helen Thomas, says that they knew that this drug inhibited a family of proteins that her group had been investigating for some years. They had shown that the proteins played a role in type 1 diabetes, in which they amplified the body's immune attack against insulin-producing cells.

"When we tested this drug in a type of mouse that develops type 1 diabetes, we found that not only did it prevent the disease but, to our delight, it even reversed it in newly diagnosed mice, effectively curing them."

Helen says that when type 1 diabetes is first diagnosed in both mice and humans, there are still a substantial number of insulin-producing cells left. If protected from attack by the immune system, at least in Helen's mice, these cells can make sufficient insulin for normal health.

"We are now planning a clinical trial to test this drug in people with newly diagnosed type 1 diabetes, to see if we can reproduce the benefits we saw in the mice."

Helen adds that if the trial proceeds as planned, it is likely that in the long term the drug will need to be combined with other treatments to stop the immune attack.

Insulin – first used almost 100 years ago – remains the mainstay of treatment for type 1 diabetes.

Helen and her group hope to soon change that.

"...not only did it prevent the onset of the disease; to our delight, it even reversed it..."

130,000 AUSTRALIANS LIVE WITH TYPE 1 DIABETES

OTTO WAS DIAGNOSED WITH TYPE 1 DIABETES WHEN HE WAS 10.

"I was waking up every two hours because I needed to go to the toilet and was barely getting any sleep.

One weekend, I was at a ukulele festival. Normally I can't contain my excitement, but I had blurred vision, was tired and not having a good time.

We drove to the hospital, where I was diagnosed with type 1 diabetes. I remember feeling really worried and afraid.

It's a tough ride having diabetes. Making sure my blood is on track and I can respond correctly is now vital. It adds an extra step that I'm going to have to take everywhere I go.

I inject insulin three times a day and check my blood before eating and whenever I feel like I'm running low or high.

To all those people who donate to type 1 diabetes research, I say thank you."

AUSTRALIA IS DUE TO HAVE ITS FIRST MODERN DECLINE IN LIFE EXPECTANCY DUE TO OBESITY

Over two thirds of Australians are overweight or obese.

These people have an increased risk of developing chronic diseases, including type 2 diabetes and heart disease, and are more likely to die as a result.

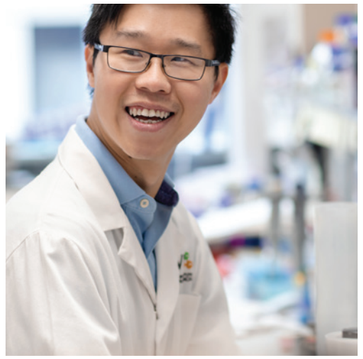
Overcoming obesity is difficult because the body's control mechanisms for appetite and energy expenditure fight any attempt to lose weight.

AMP-activated protein kinase (AMPK) acts as the body's fuel gauge, controlling the balance between how much energy is stored as fat, and how much is used for exercise and other activities. It also helps to regulate appetite.

HEART DISEASE & TYPE 2 DIABETES

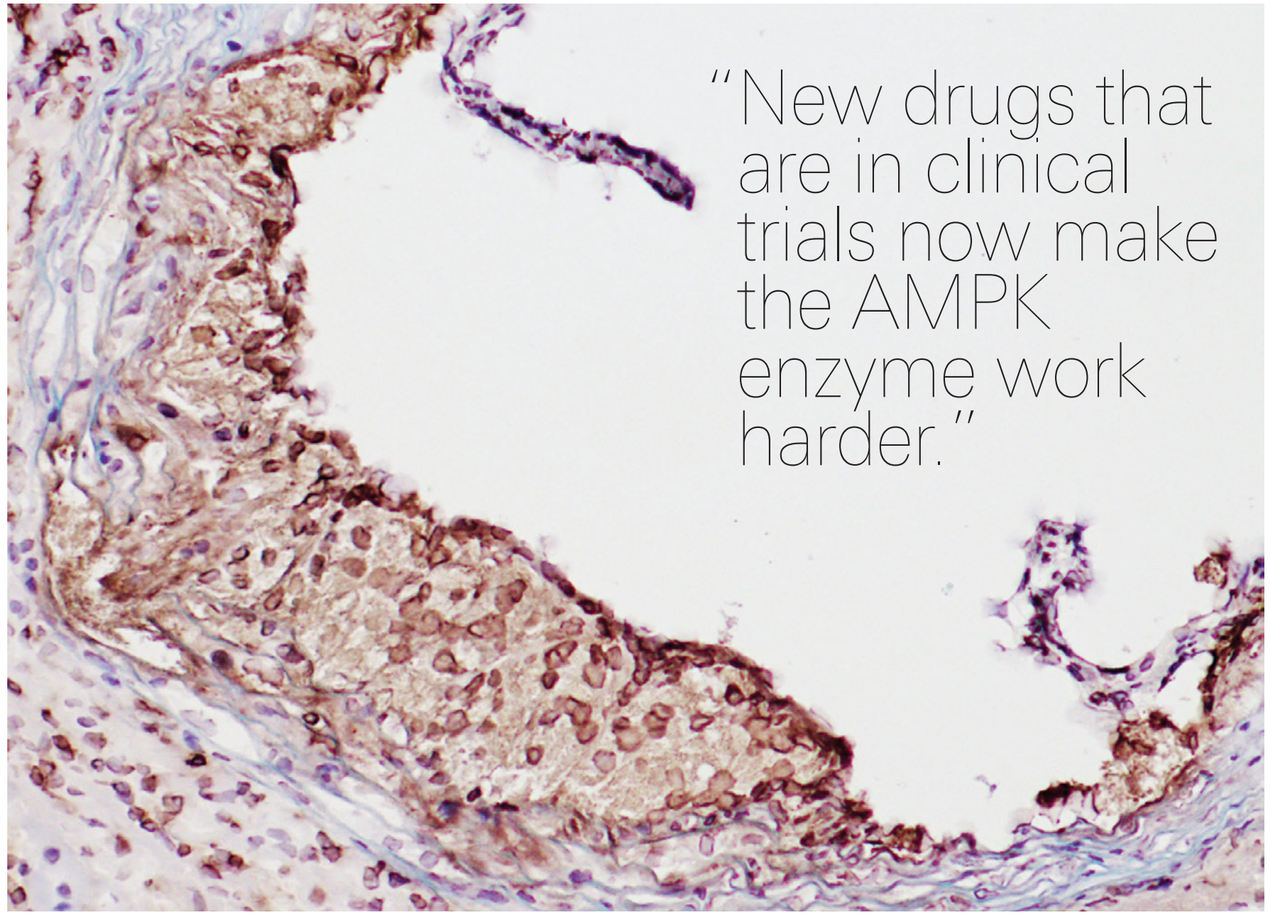
Researchers in SVI's Protein Chemistry & Metabolism Unit are looking for new ways to fight Australia's obesity epidemic by working out how the body controls its use of energy and understanding the molecular pathways that underlie the development of type 2 diabetes and heart disease.

AMPK is a prime target for the development of drugs to combat heart disease and type 2 diabetes.



Dr Kim Loh

Image above right: Cross section of an atherosclerotic plaque from a mouse in which certain signalling pathways have been dampened, resulting in an increase in the number of macrophages (stained brown) accumulating in the aortic wall.



AT THE HEART OF IT

The relationship between type 2 diabetes and heart disease is complex.

Dr Kim Loh from SVI's Protein Chemistry and Metabolism Unit says that a unifying feature of these often-intertwined conditions is the involvement of an enzyme called AMP-activated protein kinase (AMPK).

AMPK, he says, plays a central role in controlling the balance between energy production and energy storage. When AMPK is active, energy production is increased and less energy is stored as fat.

Kim says, "New drugs that are in clinical trials now make the AMPK enzyme work harder. It is believed that turning the enzyme on in people with type 2 diabetes and heart disease will have beneficial effects."

In 2018, Kim received support from the L.E.W. Carty Charitable Fund and subsequently from the National Health and Medical Research Council to look at the role that AMPK plays in heart disease.

"Heart disease is primarily caused by atherosclerosis: a process in which fat, including cholesterol, accumulates on the inside of arteries. These fatty deposits can progress to block blood flow, causing heart attacks, stroke, or heart failure."

One of the hallmarks of the early stages of atherosclerosis is the accumulation of specialised immune cells called macrophages in the blood vessels. Their job is to locate and engulf unwanted particles, such as cellular debris and foreign substances.

"It is thought that the macrophages arrive to deal with inflammation and the fat build up and then themselves contribute to the formation of the plaque and its consequences. There is some evidence that AMPK is involved in this process."

Kim has developed a sophisticated suite of research tools to determine the role that AMPK plays. He has access to drugs that specifically turn the AMPK enzyme on and off, mice that are prone to the development of atherosclerosis, and others that carry mutations in specific parts of the AMPK enzyme.

Kim's aim is to clarify the exact contribution that AMPK makes to the development of heart disease, in order to find new, more effective, ways of treating it.

MORE THAN ONE MILLION AUSTRALIANS HAVE BEEN DIAGNOSED WITH TYPE 2 DIABETES

MIGNONNE FOUND OUT SHE HAD TYPE 2 DIABETES WHEN SHE WAS 38.

"I swam occasionally for exercise but I was overweight. I felt unwell and had skin infections before my doctor diagnosed type 2 diabetes. Diabetes and heart disease run in my family.

That was more than 20 years ago now and my life has totally changed since then; it sparked a lot of things in me. I changed my diet completely and started going to the gym three times a week.

Living with diabetes is incredibly hard. Every day is a battle, you can't expect it to get any easier.

You do have to watch every mouthful you eat.

I had my first child at 43 and my son and partner are my motivation to keep healthy. I want to see my son grow up."

MOST PEOPLE DIAGNOSED WITH CANCER IN AUSTRALIA ARE STILL ALIVE 5 YEARS LATER

One in three Australian men and one in four women are affected by cancer before the age of 75 and over 100,000 new cases are diagnosed every year.

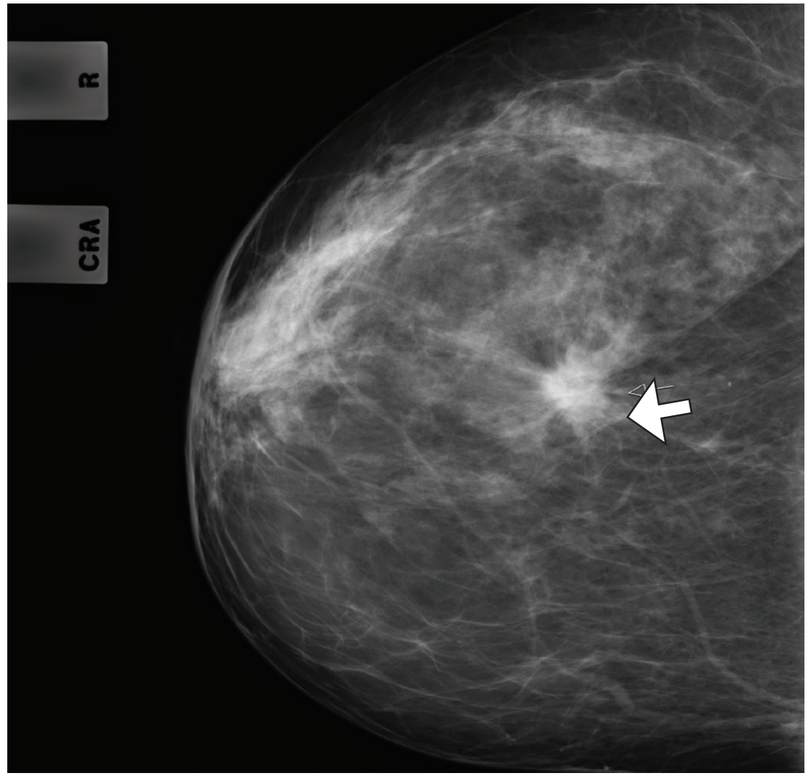
While survival rates have increased significantly over the past 20 years, cancer remains a leading cause of death: every year more than 40,000 Australians die of the disease.

Cancer costs Australian health services over $6 billion a year. Its cost in terms of pain and suffering is inestimable.

The most common cancers in Australia (besides non-melanoma skin cancer) are prostate, breast, colorectal, melanoma and lung cancer.

C CANCER

Researchers at SVI approach cancer from a number of angles, from fundamental research into the mechanisms of the disease, to understanding diseases of cancer survivorship.



"...we need a whole arsenal of tools to beat the disease."

CANCER'S ACHILLES HEEL

Cancer starts with DNA damage.

This damage occurs randomly and constantly – simply as a consequence of our daily lives – because of aging, exposure to radiation, environmental carcinogens and other factors.

Luckily, our cells have a number of different methods to repair DNA damage. However, if it is not repaired, or repaired incorrectly, the mutations that arise can lead to the development of cancer.

Maddie Riewoldt's Vision Fellow, Dr Wayne Crismani, from SVI's Genome Stability Unit, aims to find new treatments for people who have 'cancer in the family', caused by inherited mutations in the BRCA1 and BRCA2 genes.

Wayne and an international team of collaborators were recently awarded an NHMRC grant to fund the research project.

Related research in his group, on inherited bone marrow failure, is supported by funding from Maddie Riewoldt's Vision.

Wayne says that BRCA1/2 normally protect our cells from cancer by mending DNA as it is damaged.

"Nearly every cell in our bodies has two copies of each gene – one from our mother and one from our father. Some unlucky people inherit a defective copy of BRCA1/2, and then their other 'healthy' copy acquires a mutation that stops it from working. In these cells, BRCA1/2 can no longer fix DNA damage. This causes mutations to accumulate, leading to cancer."

A new drug treatment, known as PARP inhibitors, introduced in 2018, works by specifically targeting those cells in which BRCA 1/2 no longer works. This concept is known as synthetic lethality.

Wayne explains, "Imagine you are riding a bike downhill, hurtling towards a busy intersection. If you pull on your handbrakes, you come to a stop. If either your left or right handbrake didn't work, you would still manage to stop to avoid a collision, but if both were faulty, you would have a bad accident."

Wayne says that a cell in which BRCA1/2 is not working has the equivalent of a faulty brake. These cells are predisposed to cancer. PARP inhibitors work by selectively knocking out the other brake – targeting an alternate way that our cells repair DNA. With both options for DNA repair gone, catastrophic damage to its DNA causes the cancer cell to die.

"However, relapse during PARP inhibitor treatment has already been seen in the clinic, because cancers are very good at evolving. This is why clinicians talk about using first-line, second-line and third-line therapies. We urgently need other drugs to treat advanced disease," says Wayne.

Wayne is working to develop a new set of compounds that act in the same way as PARP inhibitors, but on a different arm of the DNA repair pathway, one in which he is an expert.

By exploiting DNA damage – the very thing that starts cancer in the first place – Wayne and his team hope to be able to find new ways to treat it.

The best chance we have to change these statistics is through medical research.

MORE THAN HALF OF THE WOMEN WHO CARRY BRCA1/2 MUTATIONS DEVELOP BREAST CANCER

RACHELLE WAS 36 WHEN SHE WAS DIAGNOSED WITH BREAST CANCER

"Genetic testing and a positive BRCA2 mutation meant there was a high chance that my sister Corinne and I would develop breast cancer.

I was diagnosed with stage IV metastatic breast cancer. My yearly mammogram, breast ultrasound and MRI had shown no evidence of cancer in the 12 months prior.

I was angry; I wasn't prepared for life-long treatment and the fact that I may not see my two young boys become adults.

I've now had 24 months without progression on my second line of treatment – a clinical trial for people with a BRCA mutation.

My sister Corinne responded to my diagnosis by getting a double mastectomy.

I am forever thankful for the work that medical researchers do; if I could request anything of them, it would be to discover more mutations that cause cancer and to develop targeted therapies for them."

IT'S EASY TO THINK OF OUR SKELETON AS A COAT HANGER FOR OUR BODY

In reality, however, bone is a living tissue with important functions, like storing minerals in its hard outer shell and producing red blood cells in its spongy interior.

Different types of cells, which act much like little construction crews, are tasked with looking after the health of our bones. The crews are made up of osteoclasts, demolition teams that are called in to break down bone. Osteoblasts, on the other hand, are the builders which lay down new bone in areas where it is required. A perfect balance between these two crews keeps bone strong.

However, as we age, the balance shifts. Bone is broken down faster than it is made, which makes our bones weaken over time. The hard surface of the bone becomes thinner, and its spongy interior becomes even spongier.

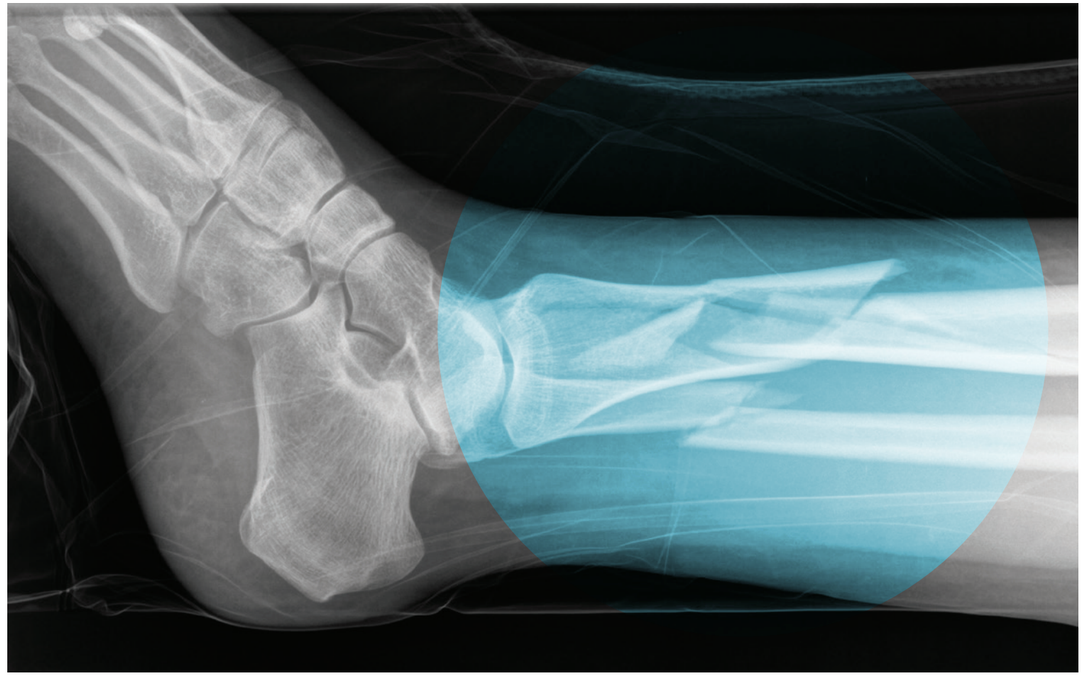
Osteoporosis is often diagnosed only after someone has already broken a bone.

B

BONE DISEASE

Research in SVI's Bone Disease & Biology Unit is focused on understanding the cells within the bone and how they interact with each other and their environment. Ultimately, the researchers' aim is to identify new pathways to promote bone formation, in order to help people suffering from diseases like osteoporosis.

"...we are not currently able to reliably predict who is at risk..."



Professor Natalie Sims

DIGGING FOR CLUES

Bones are hard in more ways than one.

It's not surprising to discover that medical researchers find it tricky to get access to samples of living human bone. For this reason, much of our understanding of how bone cells work has derived from experiments carried out in animals.

SVI's Professor Natalie Sims says that understanding what is occurring within human bone during the development of osteoporosis is essential in order to find new ways to detect and treat the condition.

"Osteoporosis is becoming more common as the population ages. The standard test is a bone mineral density scan, but about 60% of fragility fractures actually happen in people who have normal scans. This means that we are not currently able to reliably predict who is at risk."

What's more, Natalie says that the most common treatments for osteoporosis stop bone from becoming more fragile, but do not make it stronger. She says that research using human bone samples is required to understand more about how the skeleton works, and how it reacts as a person ages.

Natalie has found a way to address the difficulties in accessing human bone samples. When someone has a major bone fracture, they often undergo surgery to stabilize the bone. Surgeons use a process called reaming – essentially coring out a hole in the bone – to provide an anchor point for a pin or hip replacement.

Natalie has marshalled doctors from Western Hospital, Monash Medical Centre and St Vincent's Hospital Melbourne to provide her group with the bone debris from surgeries that they carry out in postmenopausal women with osteoporotic fractures. This bone is usually discarded as a useless by-product of the surgery.

Natalie and her team will collect the precious samples and grow them in the laboratory. She will then isolate the different bone cell types. By comparing which genes are active or inactive in these cell types, she hopes to identify the profile of genes that make some people more prone to particular fractures than others.

Bones are hard. So is medical research. But for Natalie, the effort is worthwhile.

1.2 MILLION AUSTRALIANS HAVE OSTEOPOROSIS

JUDITH WAS 63 WHEN SHE WAS DIAGNOSED WITH OSTEOPOROSIS

"I had a heavy fall and 6 weeks later I had chest pains. The doctor eventually picked up a fracture in my back.

Thirteen years on, I have had another six back fractures, ongoing disc problems and have shrunk 4 inches. I have also had four rounds of infusions to strengthen my bones as well as a procedure to relieve back pain caused by vertebral compression fractures, in which bone cement is injected through a hole in the skin into a fractured vertebra.

I'm getting the humpback look, but I live with it. Each day I think 'I hope I don't break any more'. Despite these challenges, I walk my dog every day and still enjoy gardening.

I hope that researchers can develop a simple test to quickly confirm whether or not someone has osteoporosis."

OUR ABILITY TO INTERACT WITH THE WORLD IS THE RESULT OF THE SIGNALS THAT RUN BETWEEN OUR NEURONS LIKE CARS ON A BUSY NETWORK OF FREEWAYS

Alzheimer's disease occurs when these networks within our brains are disrupted.

While there is some dispute about what triggers the disease, it is known that the brains of people with Alzheimer's are characterised by the accumulation of proteins called beta amyloid and tau.

These proteins are thought to build up in the neurons themselves and in the spaces between them, with the prevailing theory being that, once in place, they disrupt the ability to transmit the signals that allow our brain to function. More recently, the immune system has been implicated in the disease.

Every day, around 250 more Australians are diagnosed with dementia.

A

ALZHEIMER'S DISEASE

Researchers in SVI's Structural Biology Unit are working to find new treatments for Alzheimer's disease through better understanding of the three-dimensional structure of the proteins involved.

Image: Mouse microglial cells

Dr Luke Miles

PUTTING THE BRAKES ON ALZHEIMER'S

Some of the players involved in Alzheimer's disease have been well defined: toxic protein plaques made up of beta amyloid and tangles made up of a protein called tau.

More recently, the finger has also been pointed at immune cells called microglia – cellular scavengers tasked with scouring the brain and gobbling up damaged material.

Dr Luke Miles, from SVI's Structural Biology Unit, says that despite all the work that has been done in the field, the exact chain of events that leads to the development of Alzheimer's is still unclear.

"We know that toxic proteins accumulate in the brain of people with the disease and we also know that the mechanisms normally in place to clean up these proteins are disrupted. How the timing of these two processes work is still unclear."

Luke and the team at SVI have spent the last few years trying to find ways to enhance the action of microglia. They have been focused on a protein that is found on the surface of the microglia themselves.

Using sophisticated technology, Luke has drawn up a molecular map of this protein – a blueprint that may allow the researchers to develop drugs which can physically interfere with its function.

"It is thought that this protein acts as a sort of brake on the microglia. Now that we know its shape, we are beginning to design compounds which can turn it off. In theory, this could allow the microglia to do their job better and possibly slow the effects of the disease process or even stop it from occurring in the first place."

With trials still far off, the compounds that the team are developing will nevertheless provide important tools to help understand the tragic chain of molecular events that leads to Alzheimer's.

ALMOST HALF A MILLION AUSTRALIANS LIVE WITH DEMENTIA

PHIL WAS DIAGNOSED WITH ALZHEIMER'S 4 YEARS AGO, WHEN HE WAS 55

"My dad had been diagnosed with Alzheimer's when he was 88, and he's 92 now. We thought it was unreal that I got it early and he got it late in life.

I wasn't surprised when I was diagnosed, because I'd started having symptoms the year before. If I was with the boys in the bar, there was no way I could remember them. I was forgetting things around the house and couldn't cook.

In my experience, doctors don't have a clear understanding of what it means to live well with dementia. I take every single opportunity to keep myself independent. I take day flights to Adelaide with my assistance dog Sara to visit Mum and Dad. I get confused sometimes, but I still go.

So many people are affected by dementia. If we all donated towards medical research, it could make a huge difference."

SVI STAFF 2018

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

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Dr Sophie Broughton
Karen Steffi Cheung Tung Shing (PhD Student)
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Dr Jon Gooi
Dr Michael Gorman
Nancy Hancock
Dr Stefan Hermans
Dr Jessica Holien
Dr Jacinta Holmes
Jasmina Markulic (PhD Student)
Dr Belinda Michell
Dr Luke Miles
Dr Craig Morton
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Dr Tom Brodnicki
A/Prof Mark Chong
A/Prof Stuart Mannering
Prof Thomas Kay
Dr Satoru Akazawa
Matthew Andrews (Masters Student)
Mark Antonopoulos (Summer Student)
Jonathan Bebbington (MDRP Student)
Dr Federico Bonnet (Visiting Academic)
Tara Catterall
Anthony Di Carluccio
Anna Chan
Marie Christensen (Masters Student)
Oliver Cicanic (U/Grad Student)
Dr Colleen Elso
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Dr Anna Galligan (Masters Medicine Student)
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Tinting Ge (PhD Student)
Dr Shayarana Gooneratne
Karen Gu (PhD Student)
Allison Irvin
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Katrina Kiburg
Monika Kitzerwetter
Cameron Kos
Natali Kosger (Masters Student)
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Catharine McKay
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Lina Mariana
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Dr Dijana Miljkovic
Katerina Mitsinikos (U/Grad Student)
Dr Lawrence Mok
Dr Zia Mollah
Marco Montes de Oca (Masters Student)
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Sean Oh (PhD Student)
Miha Pakusch
Evan Pappas
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Dr Sandra Galic
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Dingy Yu

MOLECULAR CARDIOLOGY

A/Prof Duncan Campbell
Fei Fei Gong (PhD Student)

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Prof Natalie Sims
Prof Jack Martin
Niloufar Ansari (PhD Student)
Martha Blank (PhD Student)
Blessing Crimeen-Irwin
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Diannita Kwang (Masters Student)
Christina Nelson
Slavisa Ninkovic (PhD Student)
Kelli Schleibs
Lenny Straszkowski
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Dr Wilson Castillo-Tandazo (PhD Student)
Dr Alistair Chalk
Ankita Goradia
Dr Jacki Heraud-Farlow
Xining Li (Masters Student)
Dr Brian Liddicoat
Lokman Pang
Dr Monique Smeets
Scott Taylor
Dr Mannu Walia
Jane Xu (PhD Student)

MOLECULAR GENETICS

A/Prof Jörg Heierhorst
Emily Derrick (Summer Student)
Ashleigh King (PhD Student)
Lingli Li (PhD Student)
Dr Rui Lui
Nora Tenis

GENOME STABILITY

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Dr Wayne Crismani
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Winnie Tan (PhD Student)
Eiffel Tolentino (Honours Student)
Vanessa Tsui (PhD Student)
Sylvie van Twest

BIOINFORMATICS & CELLULAR GENOMICS

Dr Davis McCarthy

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Husni Idris (Masters Student)
Dr Musarat Ishaq
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Jarmon Lees
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Steven Morgan (Masters Student)
Jason Palmer
Georgina Riddiough (PhD Student)
Ritika Saxina (Honours Student)
Lipi Shukla (PhD Student)
Priyadharshini Sivakumaran
Beryl Tan (Masters Student)
Kam Truong (PhD Student)
Kiryu Yap (PhD Student)
Mr Stephen Tham (Honorary Research Fellow)

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Prof Anthony d'Apice
Prof Kong Wah Ng
Prof Richard MacIsaac

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A/Prof Duncan Campbell
Prof Peter Cowan
Dr Barry Dixon
Prof Michael Henderson
Prof Darren Kelly
A/Prof John Slavin
Prof Gregory Steinberg

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Prof Harshal Nandurkar
A/Prof Evange Romas

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Dr Renwick Dobson
Dr Johannes Kern
Dr Raymond Martyres
Dr Hang Quah
Dr Nirupa Sachithanandan
Dr George Varigos
Dr Meaghan Wall

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Ivan Munanto (Database Administrator)
Nicola Peniguel (Events and Donor Relationship Manager)

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Maria Pineda-Haufe (Head of Finance; Company Secretary)
Dr Anne Thorburn (Head of Operations)
Trent Anderson (IT Administrator)
Steven Boz (Administration Officer)
Elizabeth Campbell (Legal Counsel)
Theresa Clarke (Administration Officer)
Metta Clarissa (Finance Officer)
Christophe Demaison (Head of Business Development)
Susanne Fernandes (HR Adviser)
Kate Gaebele (Administration Officer)
Pam Jones (Administration Officer)
Lisa Kuspira (Media & PR Adviser)
Patrick Martin (Facilities & Safety Officer)
Dr Rachel Mudge (Grants Manager)
Kathryn O'Connell (Administration Officer)
Dr Neil Owens (Grants Manager)
Rowan Quigley (IT Officer)
Jon Rhoades (IT Manager)
Helen Ritchie (Human Resources Manager)
Dan Thomas (Facilities & Safety Manager)
Tiffany Tran (Administration Officer)
Lisna Wirrawan Liauw (Payroll Officer)
Jing Zhang (Finance Officer)
Monica Zhang (Snr Finance Officer)

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Dr Philippa Hetzel (from March 2018)
Lena Arvanitis
Dr Thein Thein Aye
Dr Susie-Jane Braniff
Penny Buxton
Liza Cabuang
JingJing Cai
Jenny Catimel
Roderick Chappel
Shannon Curley
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Anna Swierc-Lech
Roseena Taqi
John Tomosov
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Rosanna Torzillo
Marlene Tschaepe
Guiseppe Vincini
Sandra Walker
Dr Kim Wilson
Kate Zhang

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Toby Dite, PhD The University of Melbourne
Jingjing Ge, PhD The University of Melbourne
Vy Huang, PhD La Trobe University
Matthew O'Brien, PhD The University of Melbourne
Julienne O'Rourke, PhD The University of Melbourne
Michelle So, PhD The University of Melbourne

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Prof Richard Fox
Mr Paul Holyoake (to May 2018)
Ms Karen Inge
Prof Shitij Kapur
Prof Thomas WH Kay
Mr John Macfarlane (to December 2018)
Mr Stephen Marks
Ms Angela Nolan
The Honourable Chris Pearce
Ms Fiona Rowland
Dr Marcus Tierney
Mr Michael Wachtel
The Honourable Katharine Williams (from September 2018)

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Ms Kallie Blauhorn
Mrs Simone Carson AM (to November 2018)
Ms Jennifer Coutts
Mrs Caroline Daniell (from May 2018)
Ms Bernadette Dennis
Mr James Hatzimoisis
Prof Thomas WH Kay
Ms Margaret Lodge (from May 2018)
Mrs Claire O'Callaghan
Mrs Maria Palazzolo
Mr Tony Reeves (from January 2018)
Ms Brenda M Shanahan
Mrs Christine Tarascio
Ms Yan Li Wang

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Sir Gustav JV Nossal AC

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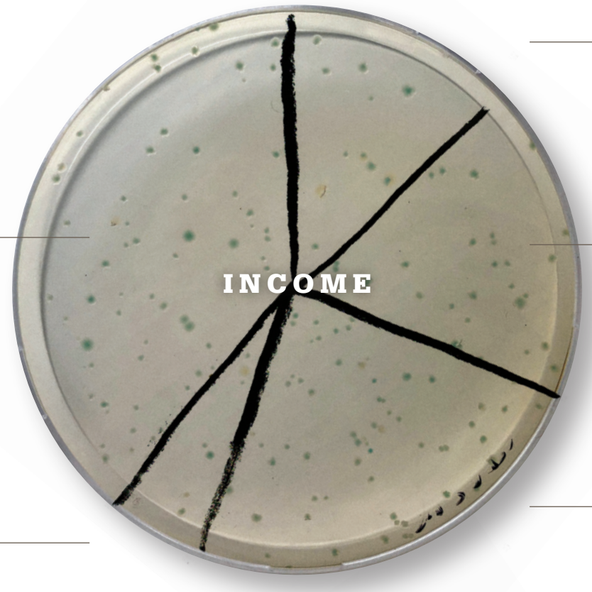
FINANCIAL SNAPSHOT



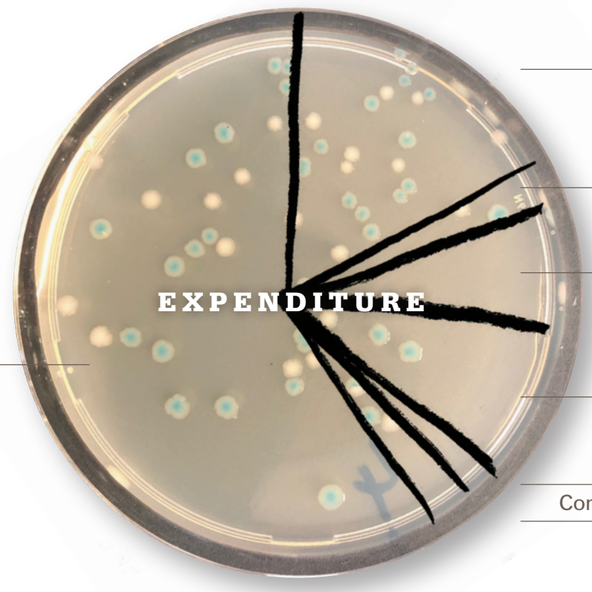
Government infrastructure support 13%

Competitive research grants 40%

Legacies, bequests and donations 18%

Contracts and other income 24%

Investment income 5%



Contract services 17%

Transfers to collaborators 2%

Building operations 8%

Research 60%

Administration 9%

Foundation 2%

Commercial development 2%

STATEMENT OF FINANCIAL POSITION AS AT 31 DECEMBER

	2018 ($)	2017 ($)
ASSETS		
Current assets	18,363,357	14,736,055
Non-current assets	18,336,792	18,294,718
TOTAL ASSETS	**36,700,149**	**33,030,773**
LIABILITIES		
Current liabilities	9,248,524	7,093,134
Non-current liabilities	187,896	148,957
TOTAL LIABILITIES	**9,436,420**	**7,242,091**
NET ASSETS	**27,263,729**	**25,788,682**
EQUITY		
– Retained surplus	26,708,690	24,304,706
– Reserves	555,039	1,483,976
TOTAL EQUITY	**27,263,729**	**25,788,682**

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

	2018 ($)	2017 ($)
Revenue	13,866,845	15,686,695
Other income	12,665,021	6,722,197
TOTAL REVENUE	**26,531,866**	**22,408,892**
Consumables and general research expenses	(6,557,567)	(3,817,160)
Employee benefits expense	(14,688,545)	(12,694,951)
Depreciation and amortisation	(1,448,812)	(1,692,191)
Administration expenses	(2,748,917)	(1,838,479)
Transfers to collaborators	(507,930)	(2,082,480)
TOTAL EXPENSES	**(25,951,771)**	**(22,125,261)**
Surplus/(Deficit) for the year	580,095	283,631
Other comprehensive income (loss):		
Transfer of retained surplus from the National Serology Reference Lab	1,823,889	0
Net gain (loss) on revaluation of financial assets	(928,937)	570,861
Total comprehensive income for the year	**1,475,047**	**854,492**
Total comprehensive income attributable to members of the entity	**1,475,047**	**854,492**

NOTE 1: GOVERNMENT GRANTS

National Health and Medical Research Council:		
– Independent Research Institutes Infrastructure Support Scheme	1,074,130	1,462,680
– Research grants	5,354,475	7,548,435
Australian Research Council	473,728	365,171
Victorian State Government – Operational Infrastructure Support Program	2,312,305	1,866,017

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

It has been 15 years since the launch of our SVI Foundation and we're immensely proud of what has been achieved so far.

As many of you may know, SVI began its life thanks to a generous bequest from racehorse trainer Jack Holt.

The generosity of community and business leaders, founding members and donors has been integral to not just our beginnings, but also our continued successes.

In particular, the tireless work of our SVI Foundation Board ensures we continue to support the Institute's world-leading medical research. Some of these board members have been with us from the very beginning, including Brenda Shanahan, whose vision brought about the establishment of the Foundation.

Brenda became a founding director of the SVI Foundation in 2004, while Chair of the SVI Board. She is currently the Chair of the Aikenhead Centre for Medical Discovery, a former Chair of St Vincent's Health Melbourne, and former Director of St Vincent's Health Australia.

Brenda shares her passion for medical research with her vast network in finance and business and leads by example in her extraordinarily generous personal giving.

The Foundation Board is also indebted to Christine Tarascio for her impressive contribution.

Christine Tarascio was Deputy Chair of the SVI Foundation Board between 2004 and 2018 and established the SVI Discovery Fund, in which donors pledge $50,000, in some cases, multiple times over.

The fund has reached its first target of $5 million, which will go a long way to supporting the work of SVI's researchers in perpetuity. Christine's annual Discovery Fund lunch, hosted at her Melbourne home, is a testament to her heartfelt commitment, empathy and passion for the work of the Institute.

Fellow founding Foundation Board member, Claire O'Callaghan, has been fundraising for SVI for the past three decades. A former nurse at St Vincent's Hospital, Claire was approached by the Director of SVI at the time, Professor Jack Martin, and then-Chair of the Institute, Jock Chappell, to help with fundraising. Claire set up a committee with Jack's wife, Christine, and they launched the inaugural SVI Christmas Ball in 1989. Sadly, Christine passed away from cancer, but she inspired many of her friends to join the committee, now known as the SVI Support Group. They have raised $500,000 over the past 15 years for scholarships to support honours and PhD students.

These three amazing women are a stellar example of the calibre of people we are lucky to have on the SVI Foundation Board.

Their support, wisdom and endurance has been both unwavering and instrumental, and we know that the SVI Foundation would not be nearly as successful without their considerable talents.

Karen Inge
Chair, SVI Foundation

Kate Barnett
Chief Executive, SVI Foundation

Image (l-r): Christine Tarascio, Brenda Shanahan, Claire O'Callaghan

AN INTENTIONAL LEGACY

Right up until his final days, Hilton Nicholas AM OBE had an abiding passion for medical research and the work of St Vincent's Institute (SVI).

"A month before he died, I remember bringing him a newspaper to read and although his mind was quite confused, he still managed to pick out a story on SVI research," recalls his wife, Marjorie Nicholas OAM.

"By that stage he had been retired from the (SVI) Board for more than 20 years, but he always maintained a vital interest in the Institute."

Hilton served on the Board for more than 30 years, including as Chair from 1988 to 1993.

He died on January 25, 2017, at the age of 91, leaving a generous bequest in his will to ensure the continuation of first-class and independent medical research at the Institute.

This passion for medical research was born out of his childhood experiences, a belief in the importance of medical advancement in changing lives, and an unshakeable value system.

Hilton lost his mother when he was just 18 months old and his father went on to build the successful Aspro pharmaceutical business.

"I don't think it was easy having such a famous father and he had no mother in his life from very early on," Marjorie says.

"Then he went to boarding school as a teenager and it gave him a sense of who he was outside the family. He learned how to deal with life's challenges and he received no special treatment, despite his background. He was enormously grateful for that defining experience."

Hilton enlisted in the RAAF during World War II where he flew Hurricanes. After the war ended, he returned to Australia to join the family firm, Nicholas Pty Ltd.

"He was only 21 at that stage, and he wanted to do a chemical engineering degree at Massachusetts Institute of Technology (MIT) in the US, but his father wanted him in the business," Marjorie says.

"I never got the sense that he was an academic person, he was more of a businessman.

"So I think maybe God moves in mysterious ways as he was able to go on and help many people through his business support of medical research."

His commitment to the Institute was also guided by a strong humanitarian streak.

"Hilton had a strong social conscience and that came from his family background, where he was taught there was a moral responsibility to share the good fortunes in your life," Marjorie says.

"He was keen on medical research and development as a way of helping people. He understood the importance of funding medical research projects even if not all of them succeeded."

Hilton's passion for philanthropy is one that Marjorie shares.

"We had similar values in that we both believed in the importance of giving back to causes we believe in," she says.

Marjorie's enduring impression of Hilton was the first time she saw him when she was 17. He was 31 and married to his wife, Brenda, who has since passed away.

"A friend introduced him as her 'recalcitrant cousin' and there he was, leaning against the wall with his hand up against the refrigerator," Marjorie says.

"I didn't see him again for another 25 years, but the image I have always had of him was of this tall, ginger-haired man leaning against a wall."

> "He understood the importance of funding medical research projects even if not all of them succeeded."

A YEAR IN PICTURES

We had the opportunity to create wonderful memories with our supporters, researchers and visitors to the Institute during the year.

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Edward & Mandy Yencken
Shay Zayontz
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Leon Zwier

ESTATES

Estate of The Late Geraldine Nicoll
Estate of The Late Arthur Stokes
Estate of The Late Margaret Littledale Tutton

THANKS TO THOSE WHO SHARED THEIR PERSONAL STORIES WITH US FOR THIS REPORT:

Mignonne De Witt
Rachelle Gebert
Phil Hazell
Judith Spriggs
Otto Zagala

Whilst every endeavour 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 at kbarnett@svi.edu.au.