the NeuRA magazine

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NeuRA
Discover. Conquer. Cure.

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Untangling TAU *in* ALZHEIMER'S DISEASE

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01

welcome

Message from our EXECUTIVE DIRECTOR

The beginning of the year is grant-writing time for our researchers. The process of developing new and innovative proposals to tackle brain disorders that impact our community is always demanding, but it provides us with the opportunity to refine our ideas and develop partnerships for success. We are fortunate to have the support of federal government grants from the National Health and Medical Research Council, the Australian Research Council and other agencies, but sadly, these grants don't cover all our research costs.

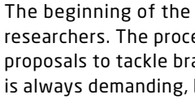
Partnerships between donors, philanthropists and NeuRA are essential. They allow us to ensure that there is funding security for our younger scientists as they pursue their areas of research for three to four years at a time. It also allows our more established researchers to plan studies that are wide-reaching in their goals and scope, as they aim to find better treatments and cures.

In this issue we meet three such researchers; brothers Dr Arne Ittner and Prof Lars Ittner, and Dr Nic Dzamko. Their work offers an amazing amount of hope to those affected by Alzheimer's disease or Parkinson's disease, and presents us with new information about the early stages of both diseases.

Crucially these new realisations have the potential to reveal how we might better treat them. It's early days yet, but these research paths hold incredible promise.

This issue also looks at the benefits that are gained when people who have had a stroke use a Wii-based rehabilitation therapy, and we touch base with some of our young researchers working in the areas of back pain and injury prevention in children.

Thank you so much for your continued support. Your donations are imperative to our ability to deliver the neuroscience research that leads to discovery, treatment and cure.



Prof Peter R Schofield *FAAHMS PhD DSc*
Executive Director and CEO

news

IN BRIEF

02

NEW MEASURE OF COGNITION MAY PREDICT LIKELIHOOD OF FALL

The Falls and Balance team are seeking volunteers aged 65 years or older to participate in their smart±step research study. This study aims to investigate the benefits of balance training and brain training on physical functions (such as balance and mobility), cognitive functions, general health and accidental fall events. The smart±step training system has been designed to enable you to undertake training in your own home, by playing engaging and enjoyable computer games. The system connects to a TV or computer monitor. The games are played with either a step mat or a touch pad. These games have been designed to train important balance and cognitive functions, while also being fun. To participate, you must be over the age of 65, English-speaking, living in the Sydney metropolitan area, able to perform everyday activities independently, have no neurodegenerative condition such as Parkinson's disease, Multiple Sclerosis, Dementia or Alzheimer's disease, and are able to participate for one year. *Please contact the smart±step team on (02) 9399 1127 or email smartstep@neura.edu.au.*

03

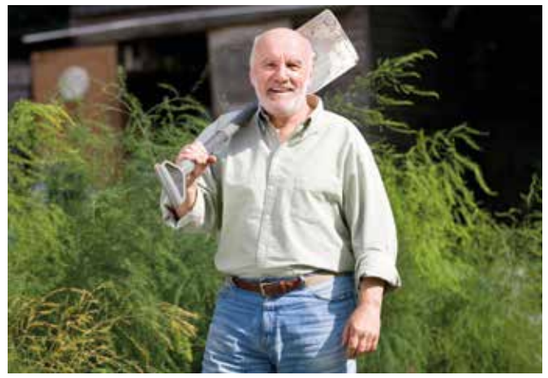
SLEEP AROUSALS IN OSA

Researchers have identified a potential new cause for obstructive sleep apnoea. Specifically, how 'intensely' a person wakes during the night. Interruptions to breathing are often associated with brief awakenings, which disrupt sleep continuity. Historically, arousals have been assumed to be vitally important in restoring airflow at the end of obstructed breathing events. However, it is now believed that different levels or intensity of arousal may have quite different effects on sleep and breathing. A recent study led by Dr Jason Amatoury, in collaboration with investigators in Canada and the USA, revealed that intense arousals caused larger breathing responses, and had different effects on the muscles around the throat that act to stabilise and open the airway. "Higher arousal intensity can cause increased hyperventilation, resulting in excessive reductions in carbon dioxide levels, which can then lead to hypoventilation (breathing at an abnormally slow rate) and airway collapse upon return to sleep," says Dr Amatoury. The study also suggests that arousal intensity may be an inherited characteristic. Future studies will focus on whether targeted individual or combined therapies could be used to treat OSA in this cohort.

04

OAM FOR NORBERT SCHWEIZER

NeuRA board member Norbert Schweizer has been made a member of the Order of Australia in the General Division for his "service to the community through voluntary roles". The Governor-General today announced Mr Schweizer's Medal of the Order of Australia award in recognition of his contributions to the Australian community – in particular his dedicated service to the care and support of those less able to fend or provide for themselves. On being informed about the award, Schweizer said, "I am very humbled by this honour". NeuRA congratulates Norbert and his family.



05

CAN A BUSY LIFE INCREASE RISK OF FALL?

In trying to understand the yet unknown causes about why older people fall over, Dr Daina Sturnieks looked at fatigue, which is a common complaint for older people. More than 50 percent of people aged 70 and over report fatigue in their daily activities. Her study compared the effects of a busy day and a restful day on fall-related measures of physical and cognitive function in 50 older people. Interestingly, tests of balance, strength, reaction time and cognitive function showed no differences between the busy and rest day. However, tests of mobility and sensation were negatively affected by the busy day, compared with rest. These few differences provide little evidence that a busy day increases fall risk in older people.

About NeuRA

Neuroscience Research Australia (NeuRA) is a not-for-profit research institute based in Sydney, Australia. Our goal is to prevent, treat and cure diseases, disorders and injuries of the brain and nervous system through medical research. Find out more at neura.edu.au or call 02 9399 1000.

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Credits

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Writers: Chelsea Hunter **Designer:** Kristian Molloy

feature story

UNTANGLING *the role of tau*

01

A new understanding of the intricate biological steps that lead to the development of Alzheimer's opens the door to new treatments that could halt the progression of the disease.

"We've been looking at Alzheimer's disease all wrong," says Prof Lars Ittner, discussing his latest study. "Until now, research has missed the first essential step in the development of Alzheimer's, which involves tau and its protective effect."

> *"This finding has opened the door to possible treatments, given time."*

This startling new discovery is a result of research that Prof Ittner has been conducting since 2010, when he first identified that two proteins – tau and amyloid-beta – together created a toxicity in the brain that lead to Alzheimer's disease. This work was published in *Cell*.

To further understand why this toxicity occurred, Prof Ittner recruited his brother, Dr Arne Ittner, a cell biologist, to participate in the study. Together, the brothers sought to understand the complex relationship between tau and amyloid-beta.

This led them to their most recent discovery, which has changed the way they view the development of Alzheimer's disease altogether. Their study revealed that tau, which has long been thought to contribute to the cell death that leads to Alzheimer's, actually has a protective effect on the brain in the early stage of disease.

This finding, which was published in the journal *Science*, overturns previously held ideas of how the disease develops and opens the door to new treatment options that could halt or slow the progression of Alzheimer's disease.

PLAQUES AND TANGLES

Around 354,000 Australians are living with Alzheimer's and other dementia-related illnesses. This figure is likely to rise to 900,000 by 2050, unless a treatment is found in the meantime.

There are many theories about what causes Alzheimer's, but the most well-supported concept involves "plaques" and "tangles". Plaques are clusters of amyloid-beta, which builds up between nerve cells. And tangles are formed by tau, which accumulates within nerve cells.

The accumulation of these plaques and tangles is associated with degeneration of brain tissue and memory loss. The Ittners' research has revealed that a crucial step in the process that leads to tangles has been misunderstood.

02

03

Initially, it was thought that amyloid-beta prompted a change in tau, causing it to become toxic. However, results from the new study suggest that tau changes itself in order to protect neurons, and that amyloid-beta assaults this protective functionality until it is progressively lost. This is the stage at which toxicity levels cause the destruction of neurons and results in the memory loss and confusion associated with Alzheimer's disease.

POTENTIAL THERAPY

Their study revealed that a third protein, kinase p38, aided tau in its efforts to protect against damage. But, as levels of p38 became depleted in the brain, so too were the protective benefits reduced.

"This is a change in concept," Prof Ittner explains. "We have completely changed our view on the whole disease process involving this protein, p38, which is central to the disease. When other researchers recognise this, it will change their views on the disease process too."

Crucially, this study has revealed that p38 is a potential therapeutic target. Using animal model studies, the brothers found that Alzheimer-like symptoms emerged in mice when p38 was blocked. When they reintroduced the protein, however, the symptoms disappeared.

"This finding has opened the door to possible treatments, given time," says Lars. "The protein has a relatively clear function," adds Arne, which he believes makes it a particularly valuable treatment target.

"As the protein is lost during the progression of the disease, you lose the protective function. But a small amount of protein still remains, so if you can stimulate that function, we may be able to delay the onset of deficits.

"The development of therapeutics is a whole different area of research, but this will push the boundaries forward," says Lars.

01 Prof Lars Ittner, Dr Arne Ittner 02 Dr Arne Ittner 03 Prof Lars Ittner
04 Dr Penelope McNulty tailors a Wii-based rehab programme for a study participant

news

Improving health WITH A Wii

USING Wii COMPUTER GAMES AS REHABILITATION THERAPY HAS PROVEN TO BE BENEFICIAL FOR PEOPLE AFTER A STROKE. NEW RESEARCH HAS SHOWN IT CAN ALSO IMPROVE THEIR FITNESS.

Wiiii-based Movement Therapy (WMT) not only restores upper limb mobility, but also improves lower limb movement and cardiovascular health in people after a stroke, according to two new studies by Dr Penelope McNulty.

Both studies compared WMT with current best practice in stroke rehabilitation. Results indicate that WMT is equally as effective, with better lifestyle outcomes at six months.

"Our study shows us that Wii-based therapy provides three essential benefits to stroke survivors," says neurophysiologist Dr Penelope McNulty. "After receiving this treatment their stepping, as well as arm and hand movements were improved and many enjoyed the additional benefit of increased cardiovascular fitness. We were pleasantly surprised with these results."

The Wii-based therapy involved 60-minute sessions per day of an individually tailored program involving Wii-Sports (golf, bowling, baseball, tennis or boxing). Game activities were introduced and varied according to motor function and progress of each patient.

"Our research emphasises the need to increase physical activity post-stroke. We have shown that WMT is as effective for upper limb rehabilitation as current best practice and, crucially, it has the added benefit of having higher patient preference, so they're likely to adhere to their rehabilitation training for longer."

04

Researchers say that WMT can be tailored to address aerobic deconditioning that affects around 50 percent of stroke survivors without compromising its focus on improving upper limb function.

Dr McNulty believes that with a few minor modifications, Wii-based Movement Therapy can be individualised to provide a carefully controlled cardiovascular rehabilitation option for stroke survivors.

"Our research highlights the importance of developing a therapy that focuses on enabling increased independence post-stroke, and that the Wii-based Movement Therapy can deliver benefits that have been overlooked by current standard therapies."

New insights INTO DOPAMINE DYSFUNCTION

01

Knowing more precisely how dopamine is changed in the brains of people with schizophrenia will help us to better understand the main path that leads to developing the disorder.

While there are many roads leading to the development of schizophrenia, one often considered a final common pathway is the dysregulation of dopamine. Too much dopamine in a particular region of the brain – the subcortex – contributes to the psychotic symptoms seen in schizophrenia.

Dopamine is a neurotransmitter that helps to control the brain's reward and pleasure centre, and regulates our emotions. Antipsychotics are designed to block dopamine receptors, and reduce the amount of dopamine action in the brain. Unfortunately, antipsychotics do not work for everyone and have serious side effects.

A better understanding of how and where dopamine is changed in the brains of people with schizophrenia will help us to know how to more accurately correct or prevent this disruption and thus help to create more targeted approaches to treatment.

A new study from the Schizophrenia Research Lab has identified molecular changes in the brains of people with schizophrenia, which offers support for and extends the dopamine hypothesis.

The new study from Dr Tertia Purves-Tyson compared tissue from deep in the brain (midbrain) of people with and without schizophrenia.

This brain region has not previously been given the attention it deserves in schizophrenia research on human brains donated after death. The study found that the genes of molecules that are responsible for regulating the amount of dopamine and for regulating the reaction to dopamine (receptors) are altered in people with schizophrenia.

These alterations in gene expression implicate a new suspect as a major contributor to dopamine dysregulation, namely a massive decrease in a dopamine transporter. The 66 percent reduction in this important molecule would mean that dopamine may be allowed to stay in the synapse longer than it should and suggests that novel treatments aimed at ramping up the synthesis and function of this in schizophrenia could be of benefit. To our knowledge, the dopamine transporter has not been used as a treatment target ever before.

This study begins to address a vital knowledge gap in schizophrenia research with regards to how dopamine in the midbrain contributes to dopamine dysfunction. This will help us to better understand the dopamine dysregulation that is found in schizophrenia and, potentially, how we can better treat it.

01 Dr Tertia Purves-Tyson views proteins from the midbrain
02 Dr Yann Quidé 03 Arkiev D'Souza

CHILDHOOD TRAUMA *and psychosis*

UNDERSTANDING THE VARIOUS CAUSES OF PSYCHOSIS IS A KEY WAY TO IMPROVE TREATMENTS.

There is increasing evidence that exposure to childhood trauma is a risk factor for some psychiatric conditions, including post-traumatic stress disorder, depression, anxiety and psychotic disorders. Trauma may present a different pathway to illness compared to those who have a psychiatric condition but who did not experience childhood trauma, and so may require a different treatment approach.

Within the Imaging Genetics in Psychosis project, lead by Assoc Prof Melissa Green, around 60 percent of psychosis patients and around 40 percent of healthy participants were exposed to significant levels of childhood trauma. This includes emotional and physical abuse, neglect and sexual abuse.

To identify these trauma-related brain abnormalities in psychosis, participants underwent functional magnetic resonance imaging (fMRI) while performing cognitive tasks. Results indicated that being exposed to trauma was associated with inefficient activation of key brain regions for working memory in psychosis patients.

02

The study, led by Dr Yann Quidé, also found that some people with schizophrenia or schizoaffective disorder who experienced childhood trauma present abnormal patterns of brain function in regions important to understanding another person's mental state, also called theory-of-mind. These findings confirm that trauma-exposure leads to distinct brain abnormalities in psychosis, and may help to improve personalised approaches to treatment of psychosis.

Dr Yann Quidé recently received an 2016 Early-Career Project Grant award from the Society for Mental Health Research (SMHR) in order to further investigate this topic.

03

NEW MOVEMENT *in muscle study*

HOW DTI IS HELPING IN THE PREVENTION AND TREATMENT OF MUSCLE CONTRACTURE IN CHILDREN WITH CEREBRAL PALSY.

A new study by Prof Rob Herbert and his team will look specifically at muscle contracture in children with cerebral palsy. It is not yet known whether contracture is a result of muscles becoming more rigid, preventing normal movement, or if it is due to changes in the length or arrangement of tendons.

Around 53 percent of children with cerebral palsy have contractures at the lower leg, which prevent normal joint mobility and can result in deformity. Understanding the causes of contracture will help to create effective prevention and treatments.

The motor impairment team, which includes Dr Bart Bolsterlee and PhD student Arkiev D'Souza, is using diffusion tensor imaging (DTI) to determine the changes in muscle architecture that accompany contracture.

DTI is an imaging technique typically used to measure white matter in the brain. Recent advancements in technology have allowed researchers to more accurately measure muscle fibre bundles as well.

"While the technology was initially developed to examine neural connectivity of the brain, over the last decade it has been increasingly applied to study skeletal muscle," Arkiev D'Souza explains. "This will allow us to compare the architecture of calf muscles in 20 children with cerebral palsy and 20 of their healthy peers."

The MRI scan takes approximately 45 minutes and the DTI data is used to generate a 3-dimensional model of the muscles in the lower leg. Differences in muscle architecture between healthy children and children with contracture will help identify the mechanism causing the condition. It is hoped this information will help to create new techniques to overcome the difficulties caused by contracture.

If you are interested in learning more about this study or are interested in participating, please contact Arkiev D'Souza at a.dsouza@neura.edu.au or 9399 1832.

01

5 minutes with

DR NICOLAS **DZAMKO**

Dr Nicolas Dzamko works with Prof Glenda Halliday to understand the causes of Parkinson's disease. He has recently published two landmark studies that provide hope for early detection and possible treatment of Parkinson's disease. He tells us more about the two studies.

YOUR FIRST STUDY LOOKED AT ONE OF THE KEY CAUSES OF PARKINSON'S DISEASE. WHAT DID YOU FIND?

We modelled the early stages of Parkinson's disease so we could gain a better idea of its causes and a possible treatment. This is the result of four years' worth of work, and we're really excited by the study's outcomes.

We've shown how inflammation within the brain is related to the development of Parkinson's disease, and we've identified a potential mechanism that can prevent this inflammation.

This gives us a new target for therapeutic research, which we're now working on.

YOU'VE USED A NEW APPROACH FOR THIS STUDY. TELL US ABOUT THAT.

This is the first time we've used the human-induced pluripotency stem cell model.

This was the Nobel-prize winning discovery from a couple of years ago and we've got it up and running now, so we can take someone's skin cells, turn them into brain cells and study them in a dish.

ONE OF THE KEY CAUSES OF PARKINSON'S IS THE 'CLUMPING' OF α-SYNUCLEIN IN THE BRAIN, WHICH CAUSES A LOSS OF CELLS AND EVENTUALLY LEADS TO THE SYMPTOMS OF PARKINSON'S. YOU WERE ABLE TO STOP THAT PROCESS IN YOUR STUDY.

Yes, that's what we were able to do. We could activate the inflammatory pathways, see the α-synuclein clumping and introduce drugs in order to stop that from happening. Given that we were able to find this association in the post-mortem brain tissue, then model this relationship in tissue culture, we're confident that we've understood a key process in the development of Parkinson's.

WHAT HAPPENS NEXT?

The next stage will be to identify a drug that can be used in human trials, which acts on the pathway we've identified and prevents the increase in α-synuclein.

YOU'VE BEEN WORKING ON A SECOND STUDY THAT HAS FOUND A POSSIBLE EARLY INDICATOR OF PARKINSON'S, IS THAT CORRECT?

We conducted one of the largest post-mortem brain studies in the world, and confirmed that a protein (LRRK2) associated with the development of Parkinson's disease is increased in the pre-symptom stages. This leads us to believe that we may be able to treat Parkinson's disease sooner.

WHAT IS THE LRRK2 GENE?

This is a gene that is found in people with a family background of Parkinson's disease and is a known genetic contributor. The study found that there are increased levels of LRRK2 in the pre-symptomatic stages of Parkinson's, suggesting that this may be an appropriate time to administer pharmaceutical therapies. Previous studies have shown that Parkinson's-associated genetic mutations increase the activity of LRRK2, and that this activity can be reduced by drug therapies.

HOW DID THIS COLLABORATION COME ABOUT?

The Michael J Fox Foundation got in touch with Prof Glenda Halliday and myself because we have access to brain tissue. We collaborated with the who's who in the world of studying post-mortem brain tissue. Most of these types of studies use a sample size of eight to 12 brains. We've got up to 30 for each of our groups and we've studied not just one part of the brain, but several parts of that brain that are affected differently in the disease. So we have a really comprehensive picture of what is happening with the LRRK2 protein.

01 Dr Nic Dzamko 02 Dr Lauriane Jugé
03 Edel O'Hagan

YOUNG **RESEARCHERS**

02

Understanding soft tissue changes in children

WHEN HUMAN TISSUE, SUCH AS MUSCLES OR SOME ORGANS, ARE AFFECTED BY DISEASE, THEY CAN BECOME STIFFER THAN SURROUNDING MUSCLE TISSUE.

Traditionally, medical practitioners have used the palpation technique – using their hands to determine the firmness of tissue, for instance around the abdomen – to feel for changes in tissue stiffness in order to diagnose illness or disease.

While this is an effective technique, not all tissue is accessible to a physician's hand. In these cases magnetic resonance elastography (MRE), a non-invasive medical imaging technique, has been developed to assess the stiffness of tissue such as the brain.

Prof Lynne Bilston's team, which includes Dr Lauriane Jugé is particularly interested in changes in tissue stiffness in the brain and muscles and how this changes in neurological and muscle disorders.

During their research they came to realise that while there is a lot of data on stiffness in adult tissue, there was little to none when it came to children. To answer this they are working on new methods to measure the properties of tissue when it is in use or under stress, either as a result of accident or disease.

One of the areas they are particularly interested in studying involves keeping children safe during car accidents. Current injury criteria and anthropomorphic test dummies, for example, are based on scaling adult anatomy to match children's anatomy. Despite this, the dummies use adult tissue properties, even though there is evidence that this can result in flawed information that cannot predict injury outcome in real children.

One of their current studies involves using MRE and diffusion tension imaging to find a more accurate way to assess and measure soft tissue changes in children.

In doing this they hope to be able to quantify the mechanical properties and microstructure of tissues in healthy children in order to better predict the responses of these tissues in situations such as car accidents or disease.

They're confident that they will be able to fill in the critical gap in knowledge so they can create accurate computational models of the body for use in child injury prevention, and other medically-related fields.

Sleep and back pain

A NEW STUDY THAT SEEKS TO FIND IMPROVED WAYS TO DEAL WITH BACK PAIN SEEKS PARTICIPANTS.

Back pain researchers at NeuRA know that there is a shared relationship between sleep and pain. Typically, the higher the pain intensity the worse a person sleeps. Conversely, after a few bad nights' sleep a person with lower back pain may perceive their pain to be even worse.

PhD student and physiotherapist Edel O'Hagan is currently working on a study that investigates whether using a medication, usually used for sleep disturbances, can help people with acute back pain – that is, pain that has lasted less than three months.

"In this trial we are investigating whether improving sleep has a knock-on effect on improving lower back pain intensity," she explains.

The medication used in the study acts on a neurotransmitter called GABA, which has a number of roles in the brain, but is primarily involved in calming overexcited neurons, such as those involved in transmitting pain.

The procedure involves a visit to NeuRA, where participants are reviewed by a physician and given the intervention tablets – either a sleep medication or a sugar pill. They take one tablet a night for 14 nights. Over this time participants keep a sleep diary and wear a monitor on their back to measure movements during sleep. They will also fill out questionnaires online on day one, at two weeks and at six weeks.

03

"Participants don't need to change anything they are currently doing to manage their back pain," Edel assures. It is hoped that this research will identify a new way to stop low back pain from developing into a long-term chronic condition.

Edel, who is part of the McAuley group, is looking to recruit more patients for the study. To get involved, email pain@neura.edu.au or call 02 9399 1618.

Making a difference

LISA JOINS NEURA *in sending her thanks*

In November last year we shared a story that we expect was tough for you to read. Bob Rushton – husband, father, grandfather, salt of the earth – had died of dementia, aged just 53. This Christmas past was the first without him, and it was terribly hard for the family. Your response to the story was very encouraging to our research teams and, most importantly, it lifted the spirits of Lisa (Bob's wife) and his family. Your kind hearts also meant that more than 50 Christmas cards, expressing love and support were sent to the family. These genuine, heartfelt messages brought tears to our eyes, so we can only imagine the impact it had on the Rushton family. Once again we thank you for your support, and showing you care.

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DO YOU REMEMBER *this face?*

Nearly two years ago Amanda Ayliffe - aged just 46 and living with an inherited form of Alzheimer's disease - graced the cover of the NeuRA Magazine. It was an exciting time for Amanda, Dr Bill Brooks of NeuRA and dementia researchers around the world. Why? Because Amanda had just received her first dose in a landmark international clinical trial aimed at stopping Alzheimer's disease before symptoms become a problem. We catch up with Amanda this May to find out how the trial is going, how she is feeling, and most importantly, what the clinical trial has taught us. There is very encouraging news to share, and we look forward to telling you about it in our winter appeal.

JOIN AN EVENT OR CREATE YOUR OWN TO SUPPORT NEURA

2017

Date Of Event	Name Of Event	Location Of Event
April 9th	Gold Coast Triathalon	Gold Coast
April 8-9th	Canberra Running Festival	Canberra
May 1st -7th	Bridge For Brain Research Challenge	National
May 21st	SMH Half Marathon	Hyde Park Sydney
May 28th	Run Noosa	Noosa
May 14th	Mothers Day Classic	Brisbane
May 28th	HBF Run For A Reason	Perth
June 11th	Perth Marathon	Perth
July 30th	Run Melbourne	Federation Square Melbourne
August 13th	City2surf	Sydney
August 26th	ASX Thompsons Reuters Charity Race Day	Rosehill
August 27th	City2surf	Perth
August 27th	Pub2Pub Run	Dee Why To Newport
September 3rd	Canberra Times Fun Run	Canberra
September 17th	Blackmores Family Running Festival	Sydney
October 30th - November 9th	Conquer Kokoda Trek	New Guinea
November 5th	Noosa Triathalon	Noosa

CREATE YOUR OWN EVENT

Walk / Run / Swim / Bake / Sky Dive / Run A Morning Tea / Trivia Night / Host A Dinner / Bootsale Birthday4Charity / Open Your Garden / Memory Lunch / Colour Your Hair / Clothes Swap — Anywhere, anytime

LEAVING A LEGACY

Following trade training and an Industrial Engineering degree from the UNSW in the 1950s, Stewart Horwood found himself working in San Francisco in the late 1970s. Networking with local professionals, mainly through Rotary, exposed him to the inherent open-minded spirit of philanthropy where ordinary, everyday people supported their chosen organisations through leaving bequests.

"Over my working lifetime, I have seen the demise of manufacturing in Australia. This sector was not only a creator of jobs, many for semi-skilled people, but more importantly for engineering, designing and production, all characterised by the need for a rigorous intellectual approach," says Stewart.

03

"On retirement, I decided to follow the US model of philanthropy and went about selecting organisations for a bequest. With no locally controlled manufacturing left, medical research stood out as a prime target. It is one in which Australia does well and has a proud record and which, by its very complexity, attracts highly skilled professionals not only from Australia, but around the world," he added.

"There is also the prospect that, with entrepreneurial management, a future income stream can be obtained from intellectual property. I am happy to be involved with, and to support, NeuRA in the quest to alleviate the burden of disease many Australians face today and in the future," Stewart concluded.

A SWING AND A HIT!

On Saturday, February 13 more than 130 people gathered to attend the Taylor Fundraising Golf Day, which raised close to $70,000 for NeuRA. As well as a few rounds of golf the teams also enjoyed a lunch and auction at St Michael's Golf Course Little Bay. NeuRA's team of four enjoyed a great day of camaraderie. We're incredibly grateful to Taylor Construction for such strong support from a committed company.

04

01 Bob Rushton with his family 02 DIAN participant Amanda Ayliffe
03 Stewart Horwood 02 Taylor Fundraising Golf Day

DONATION & RESEARCH VOLUNTEER FORM

- ☐ Yes, I would like to donate to research at NeuRA
- ☐ Yes, I am interested in participating in research at NeuRA

Title:

First Name:

Surname:

Address:

Suburb:

State:

Postcode:

Phone:

Best time & day to call:

Email:

Step 1: How I choose to give my gift:

- ☐ Please accept this one-off gift to support research at NeuRA
- ☐ I would like to make a regular donation of $ _____ on a monthly/ quarterly (please select one) basis deducted from my credit card until further notice to support research at NeuRA

Step 2: My gift:

☐ $50 ☐ $100 ☐ $250 or

☐ A cheque payable to the NeuRA Foundation is enclosed OR

☐ I wish to make my gift by credit card:

☐ Visa ☐ Mastercard ☐ American Express ☐ Diners

Card No:

Expiry Date:

Cardholder's Name:

Cardholder's Signature:

Please send me:

☐ Details about how I can support NeuRA in my will

Step 3: How to make a donation

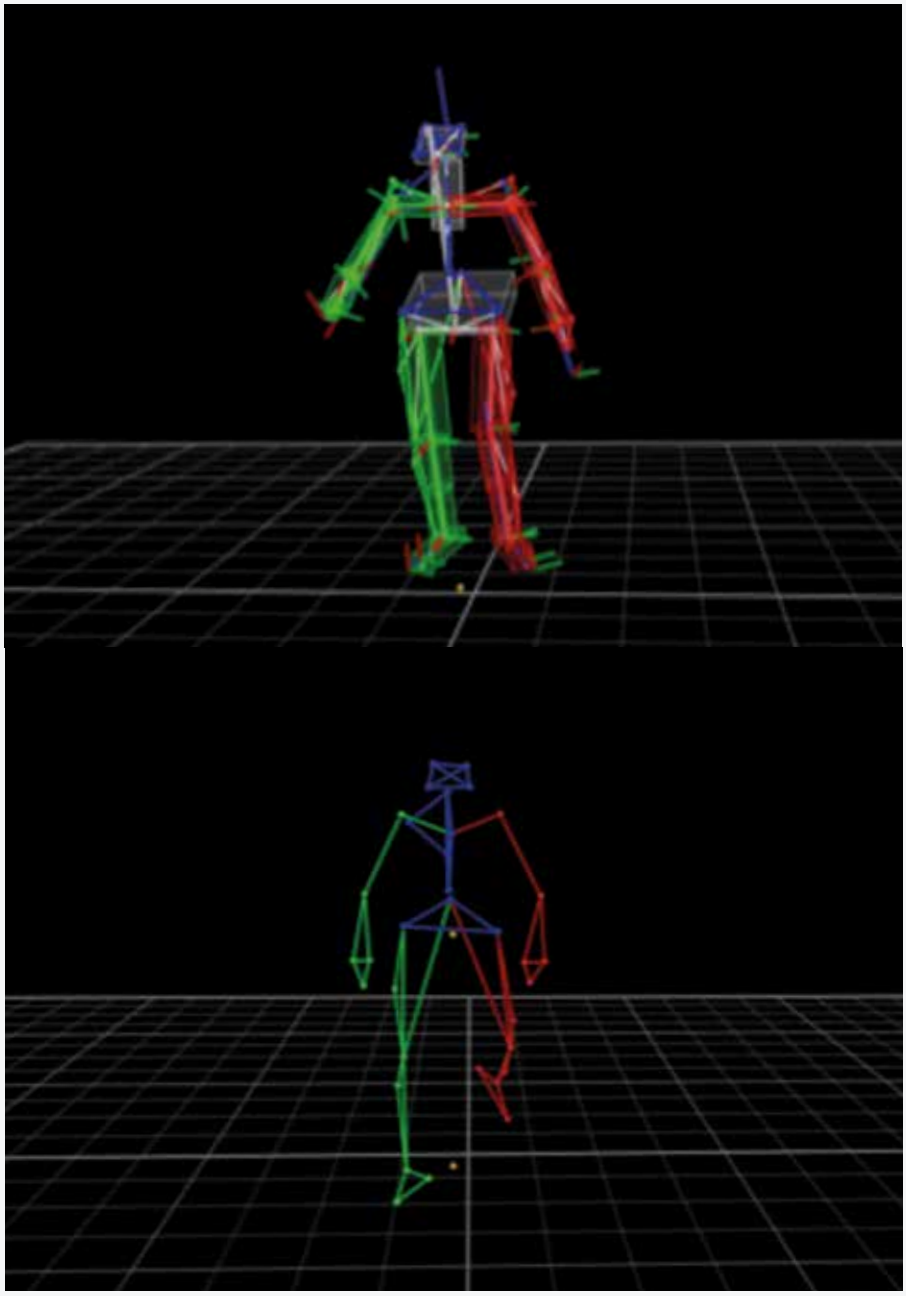
- Mail this coupon in the reply paid envelope
- Call us on **1300 888 019** to make a donation over the phone
- Make a secure online donation at **neura.edu.au/donate**
- Fax this form **(02) 9399 1082**

NeuRA
Discover. Conquer. Cure.

A message from the NeuRA Foundation: The NeuRA Foundation may co-operate with other like-minded reputable Australian charities to promote our work to our respective donors. If you'd prefer that NeuRA does not share your information with other charities, please phone us on **1300 888 019**, email us at **foundation@neura.edu.au** or write to us using the enclosed envelope.

Thank you for generously supporting our research into diseases of the brain and nervous system.

Neuroscience Research Australia Foundation, PO Box 1165, Randwick NSW 2031 ABN 57 008 429 96



in **focus**

LIGHTING **the way**

As part of our research to better understand the biomechanical factors that can lead to a fall, we use the Vicon Vantage 3D motion capture system to measure people's gait as they walk along our specially constructed pathway in the gait lab.

The Vicon system is capable of measuring movements from fine finger manipulations, to walking, running and negotiating obstacles.

During our studies, we place reflective markers on the skin (over bony landmarks) of participants to capture then analyse their movement.

This system is integrated with in-floor force-measuring plates to give us an accurate understanding of how a person's gait and balance responses may help to prevent or increase the risk of a fall.

The Vicon system and force plates all work together to provide us with 3D kinematic (movements) and kinetics (forces) of gait, balance and other human movements, similar to what is pictured above, left, without the need for restrictive devices and cables.

The synchronisation of equipment and established processing software offers an incredible opportunity for researchers and research participants to work together to understand normal and atypical movement and inform fall prevention strategies.

05 Dr Daina Sturnieks affixing sensors used to measure gait

Click on the icons on our website to view.

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