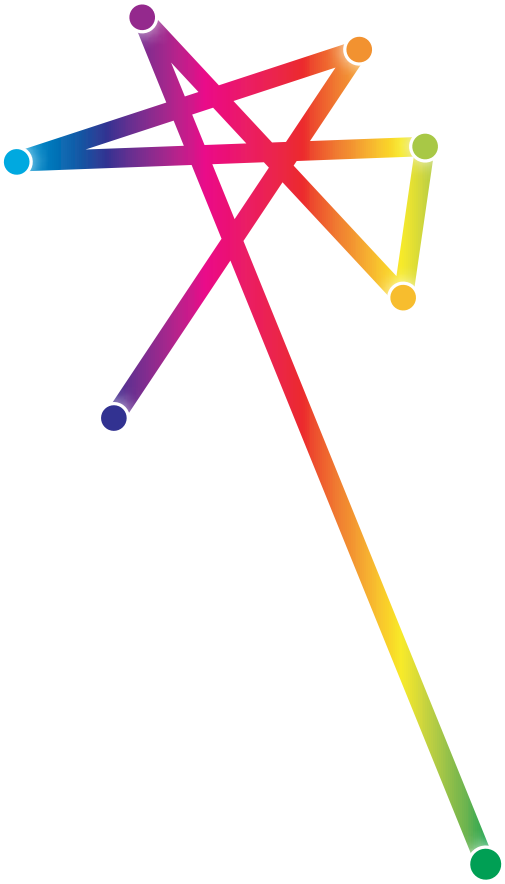


NeuRA 2010 in review

Picture a senior researcher, an older woman participating in a falls study, a young PhD student, a daughter of someone with frontotemporal dementia, a young man with a spinal cord injury – they are all seeking answers. These are the people who pass through the hallways of our institute, every day.

In 2010, this vibrant and productive place became Neuroscience Research Australia. We invite you to explore these pages and discover for yourself what we do and how much we have achieved this year.

While our scientists have profound challenges to address, our capacity for finding answers is growing day by day. If we keep an open mind to the possibilities, just imagine what we can achieve together.

Neuroscience Research Australia

Discover. Conquer. Cure.


Brain development Bipolar disorder Autism Asperger's syndrome Dyslexia Car safety Child injury Spinal cord injury Schizophrenia Binge drinking Post traumatic stress disorder Depression Sleep apnoea Chronic pain Nerve damage from chemotherapy Muscle fatigue Frontotemporal dementia Motor neurone disease Stroke rehabilitation Parkinson's disease Healthy ageing Alzheimer's disease Whiplash injury Falls Genetics Balance Brain abnormalities Brain mapping Dementia Mental illness Respiratory disorders Progressive supranuclear palsey Paralysis

It all starts with an open mind...

Pip Kuczerawy
took care of her mother who had frontotemporal dementia
See her story on page 24

Dr Tim Karl
is working on the effects of cannabis and schizophrenia
See his story on page 12

Valerie Ardler
is an Aboriginal elder and participant in our Koori Growing Old Well Study
See her story on page 30

just imagine

Caidos Sapsford
likes knowing what it's like
to be inside an MRI machine
See his story on page 8

The reason we are able to make great advances in our understanding of the brain and nervous system is due to the dedication, and generosity, of the hundreds of people who come to Neuroscience Research Australia every year – scientists, research participants, families, carers and supporters.

These are the people who are part of our Neuroscience Research Australia community

Prof Simon Gandevia
cofounded the institute
almost 20 years ago
See his story on page 18

Neuroscience Research Australia
Discover. Conquer. Cure.

Welcome to NeuRA

2010 was an extraordinary year of change and progress. After almost 20 years as the Prince of Wales Medical Research Institute, we officially became Neuroscience Research Australia, or NeuRA. With our new name, our intention is clear: we are dedicated to understanding and decreasing the burden that disorders of the brain and nervous system place on our community.

This year, we saw the introduction of new national child restraint legislation based on our research; we started a drug trial for motor neurone disease, a universally fatal neurodegenerative disorder that currently has few treatment options; we announced our plans to test the use of high speed broadband to deliver rehabilitation therapy to stroke patients in remote areas of the country; and we published our discovery of a gene and a potential treatment for frontotemporal dementia. These are just a few of our achievements in 2010. We invite you to explore these pages to find out more.

Our research touches the lives of all Australians, young and old. We have divided our research report into five sections – childhood, adolescence, adulthood, middle age and old age – to reflect the considerable range and diversity of our research.

As our population ages, the need for research on the brain and nervous system has never been greater. This year, we commenced construction on the first stage of the Neuroscience Research Precinct on our existing site. The precinct will house hundreds of researchers, all working towards preventing and curing brain disorders. Construction is forecast for completion in December 2011. The precinct will provide the opportunity to consolidate the research activities of NeuRA, the University of New South Wales, the Local Health Network and neighbouring organisations with a focus on neuroscience. This is our vision for the future. Find out more about this ambitious project on page 42 of this review.

Our significant achievements in 2010 are a testament to the diligence of the directors, and the generosity of our friends and supporters. Most importantly however, our achievements are a direct result of the dedication and enthusiasm of our talented team of researchers.

Paul Brassil *Chairman*

Prof Peter R Schofield *Executive Director & CEO*

2010 in review

Prof Peter Schofield

Paul Brassil

Our research touches the lives of all Australians, young and old

Imagine growing up to be anything you want to be

Caidos Sapsford – a research participant in our empathy study

I am eleven this year and I just started high school. My favourite class is chemistry, even though so far all we've done is boil water. I am looking forward to making a battery out of a lemon. It sounds strange, but it can be done.

I like science and that's why I wanted to be part of research. I wanted to know what it felt like to be in an MRI machine. I had to take off my glasses and put on a plastic pair so the magnetism wouldn't make them stick. I thought the noises were pretty weird. I also had to do a questionnaire, but we had a kerfuffle with that because I have a bit of Asperger's.

Asperger's means that sometimes I am not that good in social situations. I may annoy some people, or maybe hurt their feelings, and I will not know. I am working on this. If I think people do not like me, I will ask why or ask if I have done anything to offend them.

I do not mind having Asperger's. Maybe it's good because I think about things that other people don't think of and I come up with different solutions to problems. I just seem to get computers, maths and science. Others hate maths and when I try to help them I see how hard it is for them.

It sounds silly, but I really want to be a racing car driver when I grow up. I like to be competitive. I won a competition where I got the opportunity to drive a racing car. I didn't win the race, but I had a great time.

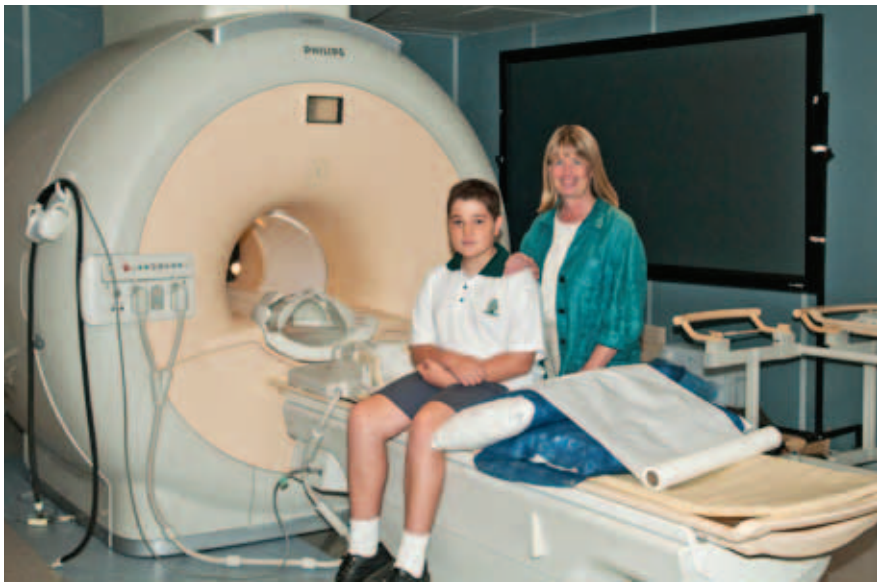
Would I like to be part of more research? Yes, I would.

In childhood

Our early years are a crucial time for brain development, from the moment in utero when our neurons first begin to fire, to infancy when our neural networks make connections at a breathtaking pace. Our research at NeuRA starts at this critical phase of life.

Autism and empathy

We are using brain scans to examine why some children have a hard time understanding what other people are feeling



Caidos Sapsford helps Prof Rhoshel Lenroot with her empathy study

> *This is one of the biggest challenges for children with disorders such as autism, and may also contribute to aggressive behaviour in others.*

Without empathy we would all find it extremely difficult to communicate with our family and friends. Yet this is how some children experience the world; they struggle to recognise what others are feeling and to adjust to social situations. This is one of the biggest challenges for children with disorders such as autism, and may also contribute to aggressive behaviour in others.

We are using magnetic resonance imaging (MRI) to identify subtle brain differences that may have developed in these children. In particular, we are examining the structure and function of the brain regions that help us recognise when someone is happy, sad or afraid. For example, we are exploring whether autistic or aggressive children pay less attention to the eyes of the person they are interacting with by asking them to identify different facial expressions while in the MRI scanner.

Pinpointing these and other differences will help us to craft interventions that we hope will minimise or even prevent symptoms from emerging so that children with these kinds of disorders can lead happy and healthy lives.

This year, we recruited more than 60 out of a total of 120 participants for this study, gathering benchmark data from healthy children. We also started to scan the first participants with autism or chronic aggressive behaviour. By the close of this project, we will have gained vital insight into how differences in brain development might lead to problems with empathy in disorders such as autism and, most importantly, clues for better treatments.

Ethan Smith demonstrates appropriate shoulder height labels on a child restraint

Safe travels

We have helped make car travel safer for children through the introduction of important new legislation

Despite substantial improvements in car safety, road trauma is still a leading cause of death and disabling injury for children in Australia. However, our studies on injuries sustained by children in car crashes indicate that much of this trauma is preventable simply by using the right type of restraint correctly.

Children aren't big enough to fit into adult seats until they are around 11 years of age. Our findings have informed important legislation introduced in 2010, which states that children must travel in an appropriate restraint or booster in the rear seat until they are at least seven years old.

We have also trialled a new system of labels, which will soon be mandatory on all restraints and booster seats sold in Australia. These shoulder height labels will prevent parents from inadvertently moving their children to a bigger seat too early and putting them at risk in the car.

This year, our research has given parents clear guidelines on how to keep their children safe in the car, which we hope will soon translate into fewer injuries and, in particular, fatalities of our youngest passengers.

How early can we see bipolar disorder?

Genes active in early brain development may predispose an individual to bipolar disorder and schizophrenia later in life

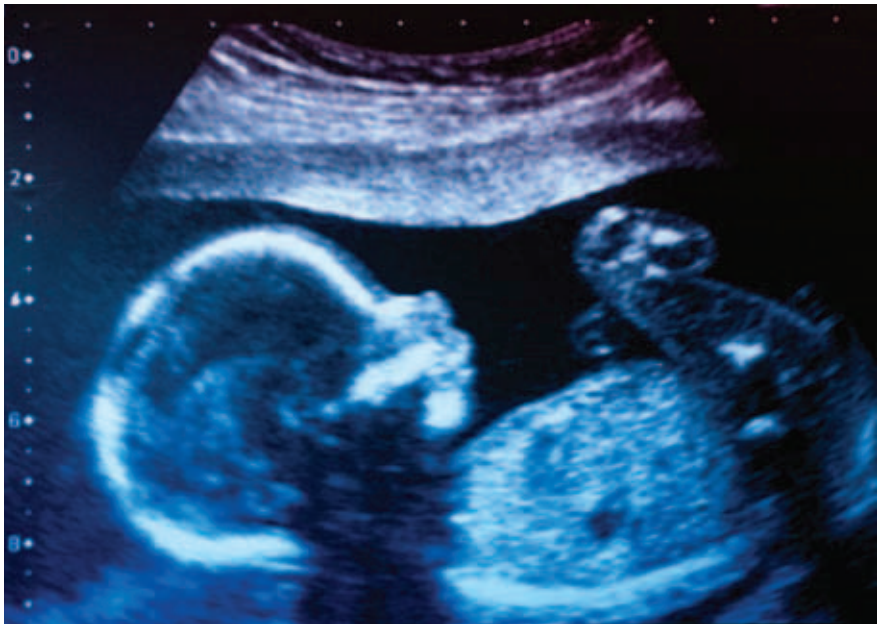
While most of us associate bipolar disorder with young adults – when symptoms of mania and depression first appear – our research has shown that the groundwork for this mental illness may in fact be laid *in utero*. We are the first group to have located and identified a gene – sialyltransferase – that may affect brain development during this early stage, thereby increasing susceptibility to mental illness later in life.

We are currently working on determining the function of sialyltransferase in more detail. Early work suggests that this gene confers a general susceptibility for mental illness, including schizophrenia. This gene may encourage faulty connections to form between neurons in the developing brain, increasing the brain's vulnerability to other damaging genetic and environmental influences, which may later trigger the onset of mental illness.

In 2010, we sequenced the gene in people with bipolar disorder to identify 'risky' variations in their DNA, and have begun to look at the effects of these variations on the sailytransferase protein and how it may function. Our next step is to determine the effects of 'risky' variations on the position and function of neurons in the brain.

Too little is known about what increases susceptibility to bipolar disorder. By uncovering the genetic and molecular mechanisms responsible, we will be able to improve diagnosis and, ultimately, treatment of the disease.

Faulty connections formed during early brain development could increase susceptibility to mental illness later in life



Read more about our research at ***www.neura.edu.au***

Imagine if we could shed light on schizophrenia

Dr Tim Karl – Senior Research Fellow and innovator in animal welfare

My research is in animal models of disease, in particular mice bred to mimic schizophrenia in humans. I really like working with animals, even if working with them in a research context may not be your typical way of showing it. Working with these animals, we can learn things about how genes and the environment interact that would be impossible to observe in human populations.

Some people say that you can't measure hallucinations and delusions in a mouse. To an extent that's true, but it's these limitations of animal model research that interest me. My aim is to improve the animal models out there so that the quality of our research is always getting better.

I feel I can justify my work with animals because I have seen people suffer with schizophrenia. When I finished high school in Germany, I worked for a time in an emergency psychiatric ward. I saw people who had had psychotic episodes, people who were suicidal. I felt deeply sorry for them because their options were so limited. It's probably why I believe that what I'm doing makes sense.

I don't believe that my work will cure schizophrenia, but it will give insights into the biological mechanisms of the disorder and might help us develop better treatments. I saw first-hand the limited effectiveness of the drugs available to the patients in the psychiatric ward.

In parallel, I also hope to improve the welfare and wellbeing of the animals we use in research. My team deals with our animals in the best ethical way possible and we're testing the effects of more stimulating cage systems on our animals. This will make our research stronger, more biologically relevant. I think as a society this should be of interest to us all.

In adolescence

Adolescence is a critical time when the brain undergoes changes to prepare for adulthood. As the brain undergoes its transformation, it's a time when latent mental illnesses often first appear. On the cusp of maturity, teenagers are still inclined to take risks that can leave an indelible mark on the brain and nervous system.

Brain plasticity in schizophrenia

A loss of brain plasticity may cause some of the symptoms of schizophrenia

Dr Leonora Long examines sections of the frontal cortex

The brain is full of connections that allow cells to communicate with each other. These connections – called synapses – are like electrical plugs in that they can be unplugged and reconnected between cells, depending on what the brain needs.

Conventional wisdom states that, during adolescence, the brain becomes more efficient at processing information by reducing some of these unwanted connections between brain cells. It's also a commonly held view in the field of schizophrenia research that schizophrenia may be caused in part by an over reduction of these connections in adolescence so that there are fewer than in healthy people.

However, there is little direct evidence that the pruning process is an important event in the development of the healthy adult brain. In 2010, we set out to measure molecular markers of these connections in participants of different ages, ranging from 1 month to 45 years.

We found little evidence for a consistent decrease in these connections at any age. Instead, we found clear patterns of a gradual increase with age. Our evidence supports the notion that growth and strengthening of these connections between brain cells is important throughout childhood, and that their number does not decrease during adolescence but remains steady.

We also looked for evidence of reduced connectivity in participants with schizophrenia. However, we found little change in the type or density of connections compared with healthy people.

We did find evidence to suggest that these connections are less able to be unplugged and reconnected in response to the needs of the brain. This ability of the brain to change and adapt, called plasticity, is important for learning and memory. This loss of plasticity may, in part, be responsible for the deficits in working memory seen in schizophrenia and may, in fact, be more important in causing the symptoms of schizophrenia than the pruning of connections.

The good news is that people with schizophrenia have not lost these important brain cell connections. This means that we can stop worrying about rebuilding connections and concentrate on finding ways to make them easier to rearrange.

Does cannabis have antipsychotic properties?

Cannabis contains dozens of chemical components, all with differing effects on the brain

> *The benefit of this research will be a better understanding of the risks involved in early cannabis abuse...*

In addition to psychoactive chemicals, cannabis contains components that may have a protective effect in mental illness

Despite the anecdotal evidence, the link between smoking marijuana, or cannabis, and schizophrenia is far from clear.

Cannabis is, in fact, made up of dozens of chemical components, called cannabinoids, that can have widely varying effects. Depending on the strain of cannabis and its origin, cannabinoid levels and ratios can differ dramatically, thereby modulating the effect the drug has on the brain. We are looking at two cannabinoids – △9-tetrahydrocannabinol (THC) and cannabidiol (CBD) – to elucidate their roles in triggering the development of schizophrenia in genetically predisposed individuals.

While both cannabinoids co-exist in cannabis plants, THC is known to increase psychotic symptoms in people with schizophrenia, while CBD is thought to reduce these psychoactive effects. We are investigating whether CBD blocks the effects of THC or has an independent anti-psychotic effect. Encouragingly, our laboratory and others have recently shown that CBD reduces the effects of other psychoactive drugs, such as methamphetamine, in rodents.

In 2010, we began looking at the effects of CBD on THC using various THC:CBD ratios, using mice that carry a genetic risk factor for schizophrenia called neuregulin 1. These mice are more susceptible to THC in a similar way to human subjects genetically predisposed to experiencing psychosis if they use cannabis during adolescence. This research will tell us whether particular cannabis strains are more likely than others to induce psychotic-like symptoms based on their THC:CBD ratio.

The benefits of this research will be a better understanding of the risks involved in early cannabis abuse and the interactive nature of genetic and environmental risk factors for schizophrenia. Furthermore, we will clarify whether CBD has a protective effect and therefore potential as an anti-psychotic therapeutic.

Does binge drinking affect the adolescent brain?

Research into how excessive drinking affects the teenage brain is essential for informing our public health response to alcohol

Binge drinking – more than five drinks on one occasion – has become a common and more extreme pattern of drinking among young Australians. With adolescence a critical period for brain development, our public health response to alcohol is desperately lacking an understanding of how binge drinking affects the teenage brain.

In adolescence, the brain undergoes active rewiring of circuitry that is necessary for successful development of 'adult' adaptive patterns of behaviour and cognitive functioning, with particular focus on the frontal lobe and its connections. We are examining this connectivity in the brains of young binge drinkers and comparing these 'tracks' to those from controls. We are also studying the size of brain regions known to be affected by alcohol, such as the hippocampus, to see whether brain structure is altered by binge drinking.

So far, we have studied adolescents using questionnaires, cognitive testing and magnetic resonance imaging (MRI) to look for changes in structure and connectivity as well as chemical differences in the brain.

Alcohol is thought to induce a state where the glutamate system – the major excitatory system in the brain – becomes unbalanced. We have found significant elevations in frontal lobe glutamate in boys who binge drink. Binge drinkers showed impaired neuro-cognitive function, with significantly slower responses and greater errors on the tests of inhibition and poorer emotional face recognition compared to non drinkers. These changes were positively correlated with binge drinking episodes and alcohol consumption.

Australia has one of the highest rates of alcohol use disorders in the world, with the age of onset often in late adolescence. This research will be crucial to informing our alcohol licensing laws and public health advice.

There is a critical lack of data on the impact of binge drinking on brain development

Prof George Paxinos is one of the world's foremost brain mappers

Mapping the brain and spinal cord

We are creating maps to guide the testing of treatments for people with spinal cord injury

While spinal cord injury can happen at any time in life, unfortunately adolescents and young adults are the most likely of any age group to sustain this type of injury, which results in lifelong suffering.

Research with stem cells in mice holds the allure of ameliorating paralysis and other symptoms of spinal cord injury. This research suggests that, while damaged areas of the spinal cord are unable to regenerate, new connections from healthy neurons in the brain can potentially reach the spinal cord and compensate for the injured ones.

However, very little is known about the connections between the brain and spinal cord of the mouse that would allow scientists to test treatments that target these healthy neurons.

Our research is comprehensively identifying the connections between the mouse brain and spinal cord. In 2010, we identified in the mouse brain more than 30 neuronal groups that project to the spinal cord. Among these is the precuneiform nucleus in the midbrain locomotor area, a nucleus previously not known to project to the spinal cord. The projections from this nucleus travel to the cervical and upper thoracic spinal cord. It is possible that these connections influence the spinal cord pattern generator, which controls the movement of the limbs.

Through the creation of our maps, we hope to assist other scientists in their quest to help people with spinal cord injury to regain movement.

Retraining the body after spinal cord injury

Specially designed training can give independence back to people with spinal cord injuries

Rachel McBain demonstrates the use of the 'coughalator'

Beyond losing the ability to walk, many people with spinal cord injuries also lose the ability to perform everyday tasks such as sitting unsupported and coughing.

We are investigating ways of retraining people with such injuries to perform these tasks so that they can lead more independent lives.

Coughing is essential for keeping the lungs clear from infection, but people with high level spinal cord injuries are less able to cough due to paralysis of their abdominal muscles. We have previously shown that electrical stimulation of these muscles can induce coughing. In 2010, we showed that, by additionally training these muscles with electrical stimulation, we can further improve the strength of the cough.

This kind of technology can lead to a longer and better quality of life. We hope to develop a self-triggered, portable device to offer even greater independence.

Sitting unsupported also requires the use of any non-paralysed trunk muscles to perform daily activities, such as dressing and reaching forward.

We evaluated intensive, task-specific training to determine its effectiveness in improving our participants' ability to sit unsupported. While we found this training to be effective, in the future we need to conduct more research into the type of people who are most likely to benefit, including those with more recent spinal cord injuries.

Read more about our research at ***www.neura.edu.au***

Imagine creating a unique place

Prof Simon Gandevia – Senior Principal Research Fellow and a cofounder of the institute

Four of us started the Prince of Wales Medical Research Institute back in 1991: Potter, McCloskey, Burke, and me. It was unique; at the time there were no big neuroscience and experimental neurology research institutes like we were proposing. Many thought we were crazy, but we were determined to make it work. We sat around my kitchen table for many hours planning how we would do it. It was really exciting.

It was a big deal moving our small research groups to what was essentially a non-existent institute. When we set up the Prince of Wales Medical Research Institute as a company, the buildings we acquired had been used as hospital wards for palliative care and various other services, not laboratory research. We renovated one of the buildings and added a second story on one side of it. For a while we didn't have the funds to fit this part out. We even played indoor cricket in the space at one of our early Christmas parties.

I guess we were all risking something. We were all under pressure to do good science. If we didn't, then the whole venture would fail. But a critical reason we set the place up was so that we would be better together than as individuals, and we've produced some extremely fruitful lines of research as a result.

In 20 years, we've grown from a core of 20 researchers to 280 or so; that's more than a ten-fold increase, which is amazing. But I think we've kept the flavour of the place as friendly and collegiate. When you walk through the institute, you don't see a narrow set of offices; you notice the wide and welcoming hallways and a vibrant tea room. We always wanted it to be that way.

In adulthood

Adulthood is the prime of life for many, but it can also be a time when the unexpected occurs. Psychological or physical trauma can take many years to heal, while something as seemingly innocuous as a sleep disorder can lay the foundations for poor health later in life.

Breathing in obstructive sleep apnoea

We are investigating what happens in the airway to cause this condition

Obstructive sleep apnoea (OSA) is more than just an extreme form of snoring. In this disorder, the muscles in the throat and upper airway repeatedly collapse during sleep, leading to a decrease in the amount of oxygen in the blood. This leads to poor sleep and an increased risk of accidents, hypertension, heart attack and stroke.

While there are several treatments available for OSA, we don't truly understand how the muscles of the upper airway cause the condition. In this project, we are taking the first direct biomechanical measurements of the upper airway. By examining how the mechanical properties of these muscles are altered in people with OSA and how the brain drives these muscles, we will be better able to improve treatments.

We are using MRI to measure the stiffness of the upper airway muscles and how they move during normal breathing, so we can understand what predisposes them to collapse. We are also measuring the electrical signals that travel from the brain to the major muscle in the upper airway, the genioglossus, using electromyography. This will allow us to understand how signals from the brain cause the muscle to contract with breathing to keep the upper airway open.

In 2010, we compared the characteristics of the tongue and the motion of the genioglossus in people with obstructive sleep apnoea compared with healthy

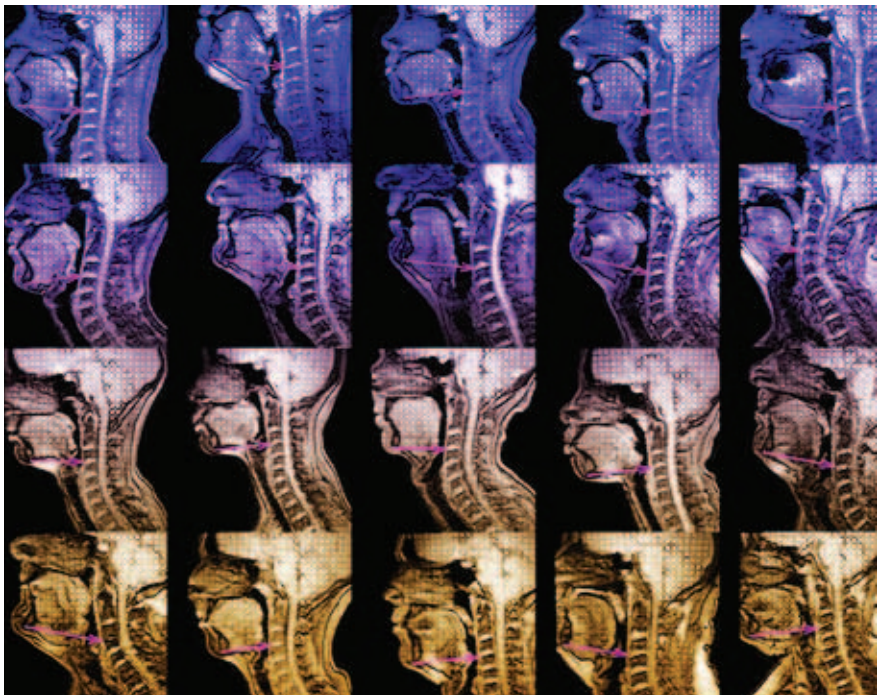
Dr Carol Dobson-Stone analyses DNA from people with post-traumatic stress disorder

By examining how the mechanical properties of these muscles are altered in people with obstructive sleep apnoea and how the brain drives these muscles, we will be better able to improve treatments.

controls. We found that the motion is significantly different in OSA and also varies with the severity of the disease. As the disease develops, tongue motion increases, but is not well coordinated, so that instead of opening the airway, contraction can widen one region and narrow another. In the most severe cases, motion appears to reduce or cease, so that the genioglossus does not dilate the airway during each breath.

With these findings, we are in a unique position to visualise the neuro-mechanical coupling in the upper airway. This will provide critical information about how the upper airway works normally and how its function is impaired in OSA.

In the longer term, we will apply this knowledge to assess the treatment of OSA and explore further how the upper airway behaves in different conditions.



These MRI images are used to study how the tongue and other muscles keep the airway open during sleep

The DNA of trauma

Do genes play a role in our response to trauma therapy?

Post-traumatic stress disorder is a type of severe anxiety disorder that can develop after experiencing an extremely traumatic event, such as one that involves the threat of injury or death. While people with this disorder are offered cognitive behaviour therapy, many people continue to suffer from anxiety after treatment.

In collaboration with the University of New South Wales, we are investigating whether our genes play a role in how we respond to treatment for this disorder, and whether we can use particular DNA variants to predict which people are more likely to respond to treatment.

We are focussing on the serotonin transporter gene, which codes for a protein that transports a neurotransmitter called serotonin in and out of neurons. A common variant of this gene leads to lower levels of serotonin transporter in the brain and has previously been associated with a higher risk of depression.

In 2010, we analysed DNA collected by our UNSW colleagues from people with this disorder who were undergoing cognitive behaviour therapy. We reported that those people with post-traumatic stress disorder, who have this variant of the gene, were less likely to benefit from cognitive behaviour therapy than other patients.

The implication is that people receiving therapy may benefit from being tested for a genetic predisposition to therapy response, thereby helping clinicians tailor their treatment.

Pain that doesn't make sense

We are investigating why some people develop excessive pain in response to injury

If you broke your wrist, you could reasonably expect the pain to reduce by the time the break heals. Yet, for some people, that pain not only continues but becomes incapacitating.

For reasons as yet unknown, a small percentage of people who experience a physically traumatic event, often a wrist fracture, develop a condition called Complex Regional Pain Syndrome (CRPS). Their painful limb may swell, sweat, become red, hot and immobile and, in the longer term, they may develop localised osteoporosis.

In 2010, we began recruiting people with wrist fractures to test the hypothesis that an excessive inflammatory response in the first two weeks after injury triggers the development of this syndrome. So far, we have enrolled 50 people, and plan to recruit a total of 1000 over the next three years.

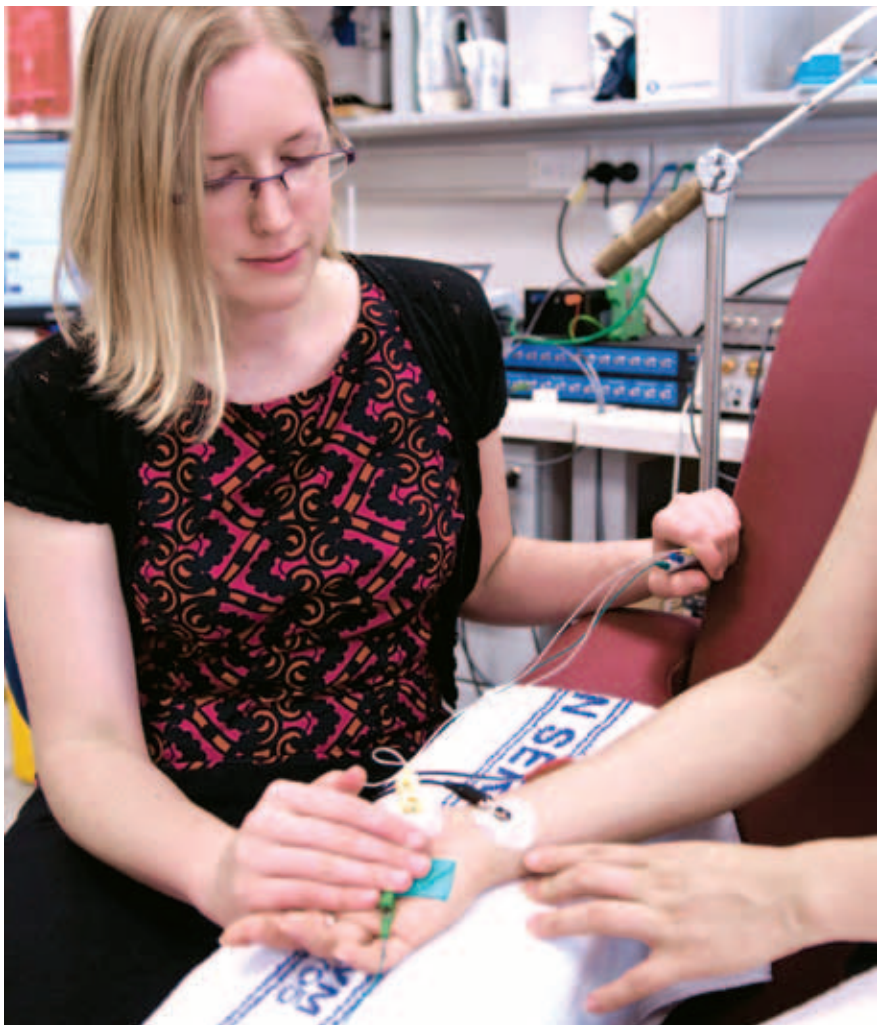
If we are able to identify a dysfunctional inflammatory response in people who develop CRPS, and pinpoint when these changes occur, we might be able to understand how the syndrome develops and how we might prevent it.

Because this syndrome shares many characteristics with other pathological pain disorders, such as chronic back pain and phantom limb pain, our research will hopefully open new avenues for investigation of those disorders also.

Luke Parkitny and Flavia Di Pietro measure inflammation in chronic pain

Nerve damage in cancer treatment

Dr Susanna Park tests a patient for nerve damage



Nerve damage following chemotherapy is no longer inevitable

Cancer specialists are already using our findings to inform treatment decisions.

Cancer treatment has progressed so far in recent years that many people have an excellent prognosis after treatment. Yet despite these advances, nerve damage is still a common side effect of many chemotherapy treatments. Nerve damage limits the amount of treatment that people with cancer can receive and may leave them with lifelong neurological symptoms including pain, numbness, weakness and difficulty walking. Up until now, the mechanisms underlying this nerve damage have been a mystery and oncologists have lacked sufficient strategies to prevent it from occurring.

Throughout 2010, using new methods of measuring nerve function developed at NeuRA, we have followed cancer patients receiving treatment to identify early predictive markers of nerve damage. We have been able to identify 80% of patients most at risk of developing severe symptoms following treatment for a range of cancers, particularly breast and bowel cancer. As a consequence, we now have a means of identifying susceptible patients early. We have also gained considerable insight into how chemotherapy induces nerve damage, which will assist us in developing protective strategies down the track.

Cancer specialists are already using our findings to inform treatment decisions. Our techniques are also being adapted for use in an international clinical trial of the chemotherapy treatment oxaliplatin.

Read more about our research at ***www.neura.edu.au***

Imagine if we had a cure for dementia

Pip Kuczerawy – cared for her mother, Anne

My mum Anne was an amazing and vibrant woman. She had been a model, a fitness instructor and had just been admitted as a lawyer when she was diagnosed with frontotemporal dementia. I vividly remember the day she was diagnosed. We all began to cry as the doctor explained what we could expect over the coming years.

Bit by bit we lost the person we loved. Mum's personality changed dramatically. She became very withdrawn from society and would yell at people on the street. She would sneak food and eat compulsively. I don't think many people understood what we were going through.

Mum passed away on Thursday 6 May 2010 aged 54 years. She had lost the ability to speak and could not walk, eat or drink. My sister and I visited Mum the night before she died. Even in her weakened state Mum still managed to give my sister one last kiss on the cheek.

Frontotemporal dementia cut my mother's life short. Mum will never move to the beach and grow old with my Dad like they planned. She will never watch my sister be admitted as a lawyer, she will never see her children marry or meet her grandchildren.

When Mum was initially diagnosed, I could not have comprehended what lay ahead. My mission now is to raise awareness of dementia so that other people out there know they're not alone. Mum gave so much of herself to others and I am honoured to be able to do something in her memory.

In middle age

Many baby boomers believe they will never have to slow down. Yet our research also shows that they are worried about their brain health. Their 50s and 60s are when some types of dementia begin to appear, and events related to poor cardiovascular health, such as stroke, can significantly affect the function of the brain.

Slowing the progression of frontotemporal dementia

One Australian family's gene has helped point to a treatment for frontotemporal dementia

Dr John Kwok investigates a mutant gene from a family with frontotemporal dementia

Over several generations, a large family living in Western and South Australia found that many of its members were developing a range of strange and troubling symptoms. At around age 50, brothers, sisters, mothers and uncles would develop changes in their personality and behaviour and sometimes their ability to control their muscles and the movement of their body. Eventually this strange disease would be the cause of their death.

After working with this family for many years, we now know that some of them have an inherited form of frontotemporal dementia, sometimes combined with motor neurone disease, that appears later in life.

Frontotemporal dementia occurs at a younger age than other dementias, such as Alzheimer's disease, appearing most commonly between the ages of 50 and 60 years. Damage to brain cells begins in the frontal (front) or temporal (side) lobes of the brain and causes changes in personality and behaviour, and the ability to speak or understand language.

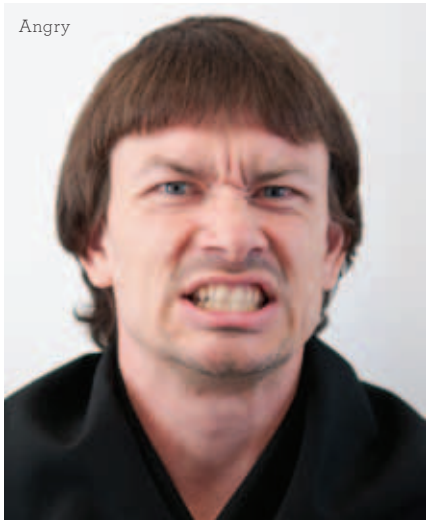
Frontotemporal dementia is not always genetically inherited, but by scanning DNA samples from this family, we were able to identify a gene responsible for their disease. SIGMAR1 causes the build-up of a protein called TDP-43 in the brain, bringing about brain cell death and the changes in behaviour that caused such distress to the family.

While not everyone who develops frontotemporal dementia will carry this gene, by studying its role in degenerative brain disease, we can gain insight into how we might prevent the abnormal build up of protein in the brains of people with frontotemporal dementia more generally.

Fortuitously, a drug known to act on this gene is already available: Haloperidol, a psychiatric drug used to treat schizophrenia. We are conducting research to investigate whether Haloperidol can slow down or even prevent the abnormal protein build-up and hence reduce the speed at which this dementia progresses. Preliminary work with Haloperidol suggests that a dose 10-100 times less than that used for treating psychosis may be effective, reducing the risk of side effects.

Through this significant research, we may have an effective treatment for frontotemporal dementia on the horizon. In fact, because Haloperidol is already on the market, a treatment may be available more rapidly than would normally be the case. For people with this disease who currently have no prospect of effective treatment, this is a crucial step forward.

Losing emotion in dementia



Cristian Leyton, Bonnie Lam, Fiona Kumfor and Marshall Dalton demonstrate some of the emotions used in a test for frontotemporal dementia

A hallmark of the disease is a loss in the ability to recognise emotions, particularly negative ones such as anger and disgust.

We are examining how people with frontotemporal dementia lose the ability to understand emotion

A loss in the ability to recognise negative emotions, such as anger and disgust, is a hallmark of frontotemporal dementia. As a result, people with this disease lose empathy. They become less able to judge and understand emotional states in others, may behave inappropriately, making tactless comments or may fail to respond to emotional cues.

We are charting how emotion recognition and empathy decrease as the disease progresses, as well as examining changes in the brain and corresponding MRI scans. We have so far recruited 90 participants. In 2010, we continued to review our patients, evaluating cognitive abilities and memory changes in the brain and the ability to process and recognise emotions and the impact on carers and family members.

So far, we have identified differences between subtypes of frontotemporal dementia, namely that people with certain subtypes can still recognise emotions when shown faces displaying stronger emotions, whereas others are unable to benefit from such cues.

We have also shown that some people with frontotemporal dementia lose the ability to recognise emotion conveyed through music. In addition, because emotion is closely linked to memory, we have found that the ability to remember significant events from the recent past differs between the subtypes of frontotemporal dementia and Alzheimer's disease.

In the longer term, our research will lead to a better understanding of the clinical symptoms and pathology of frontotemporal dementia subtypes and their progression. It will improve our ability to diagnose this disease accurately and prevent misidentification with other dementias such as Alzheimer's disease. Our research will also improve our ability to treat the disease and meet patients' and carers' needs for support services.

Treating motor neurone disease

We are testing a new drug for motor neurone disease, an illness that is universally fatal

Treatment trials for diseases with very few treatment options are always significant events, for both researchers and patients. We are conducting a clinical trial of a new treatment for people with motor neurone disease, a degenerative disease that typically affects people in their mid-50s. Currently, we have very few drugs available to treat this disease and survival is as short as between two and five years.

Motor neurone disease (MND) affects nerve cells in the upper spinal cord that control the muscles for moving, speaking, breathing and swallowing. After symptoms first appear, these muscles rapidly become weaker.

Our study, run in conjunction with the Multidisciplinary MND Clinic at the Prince of Wales Hospital, is a double-blind, randomised controlled trial of a drug that blocks the function of sodium channels in cells. Our aim is to determine whether we can slow the progression of the disease, which we will measure by monitoring markers of disease severity.

By 2010, we had enrolled 53 participants in the study, who we initially monitored over a 12-week lead-in phase and then randomised to receive active treatment or placebo for 32 weeks. We are currently following up with our participants to record how they have responded to treatment. Our goal is to provide a treatment for patients with motor neurone disease worldwide.

Ben Cheah demonstrates a method of measuring the effectiveness of a treatment for motor neurone disease

The Sydney Brain Bank is an invaluable resource for researchers

The brain bank

Research in life and after death deepens our understanding of brain diseases

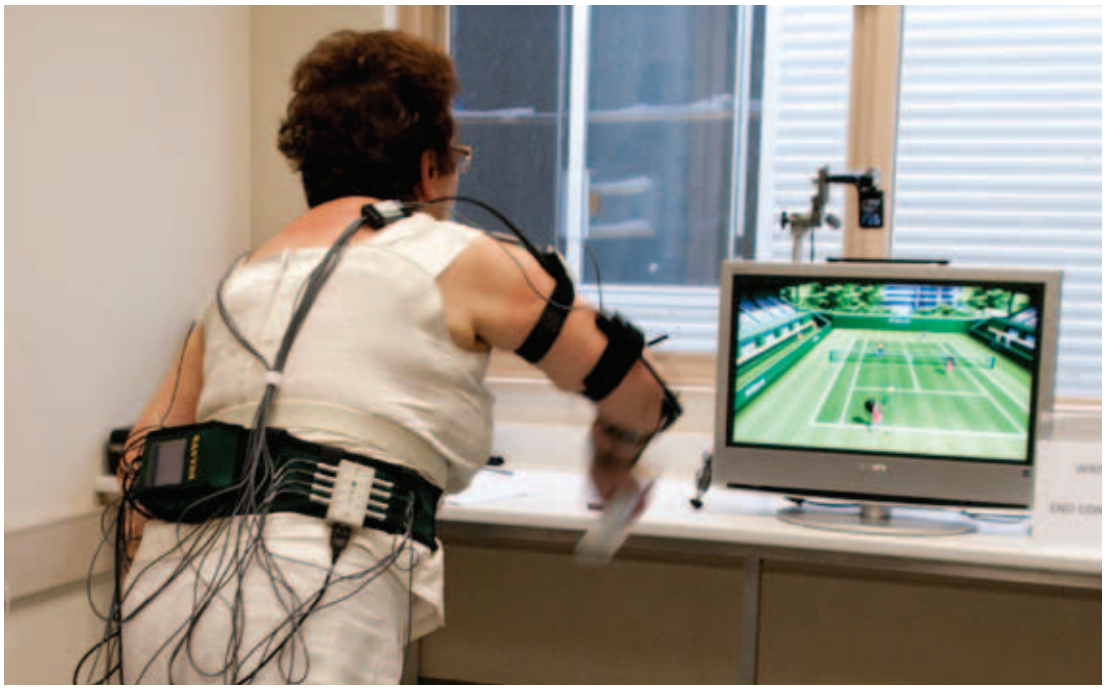
Brain tissue arrives at the Sydney Brain Bank from all over New South Wales. It provides an invaluable research resource for scientists in Australia, and around the world, seeking to alleviate the suffering caused by neurodegenerative diseases such as frontotemporal dementia, Parkinson's disease, Huntington's disease and Alzheimer's disease.

We currently store tissue from approximately 800 donors and have bequests from 400 more who are interested in supporting our research.

In 2010, the Sydney Brain Bank established agreements with a number of clinical research programs in Sydney that study individuals in life and recruit them for brain donation at death. The value of working with these programs is that the brain tissue collected also has comprehensive clinical information, which enriches the research that can be carried out.

Over the coming years, the Sydney Brain Bank's intention is to continue to expand the collection of high-quality and well-characterised cases to enable the best possible research outcomes.

Stroke rehab via high speed broadband



Tania Opadchy plays virtual tennis to improve movement in her stroke-affected arm

Our research has shown that Wii therapy can significantly improve movement after only two weeks.

We are designing rehabilitation therapy for stroke patients who have difficulty travelling to receive care

Every year, over 60,000 Australians have a stroke and rehabilitation is the only method to recover movement of stroke-affected limbs.

We are working on a project to offer rehabilitation therapy to people who have had a stroke using the Nintendo Wii gaming system. Delivering therapy using high-speed broadband to people living in non-metropolitan areas of Australia will become a reality.

'Wii therapy' requires complex movements combined with progressively increasing skill levels. Our research in 2010 has shown that Wii therapy can significantly improve movement after only two weeks. Our participants increased use of their more-affected hand and were able to move the joints of their more-affected arm further. They also enjoyed a cardiovascular workout which is important as fitness is typically poor after stroke.

We plan to recruit 50 participants who will take part in ten one-hour sessions at home over a two-week period, performing exercises designed to help improve the range of movement in their stroke-affected hands and arms. Each patient will be supervised by a therapist in Sydney who will use high-speed broadband to receive high quality video images and sensor data to analyse progress and provide feedback.

The ultimate goal of this research is to provide quality rehabilitation therapy to people who have had a stroke and have difficulty travelling to receive care. With much of Australia's population living outside of the major cities, this technique will contribute significantly to our ability to provide effective healthcare to all Australians.

Read more about our research at ***www.neura.edu.au***

Imagine if we could all age well

Valerie Ardler – Aboriginal Elder and participant in our Koori Growing Old Well Study

I've lived in La Perouse for most of my life. La Pa is a beautiful place; I've had a good life out here. I used to love swimming.
At La Pa, if you didn't know how to swim, you soon learned because they'd throw you in the water.

I'm 67 now and they call me an Elder. I didn't think I was that old but then my nephew is nearly 50 so I must be!

A lot of my friends are already gone. I've got a friend who's my age and she's got the start of dementia. She keeps asking me the same questions all the time. There's a lot of that going around now, memory problems. That's one thing I wouldn't like to get.
I'm worried that some of these young ones won't even reach the old age, the way they're drinking and getting on these rotten drugs. Even some of the older ones don't think anything bad is going to happen to them.

I found out I had breast cancer in 2000. After I had my breasts off, I had a triple bypass. I have a big cross on my stomach and chest. People come up to me because they can see how good I've come. They'll ask me things and I find that I'm helping them. I did a breast cancer talk and they said how good it was that I spoke.

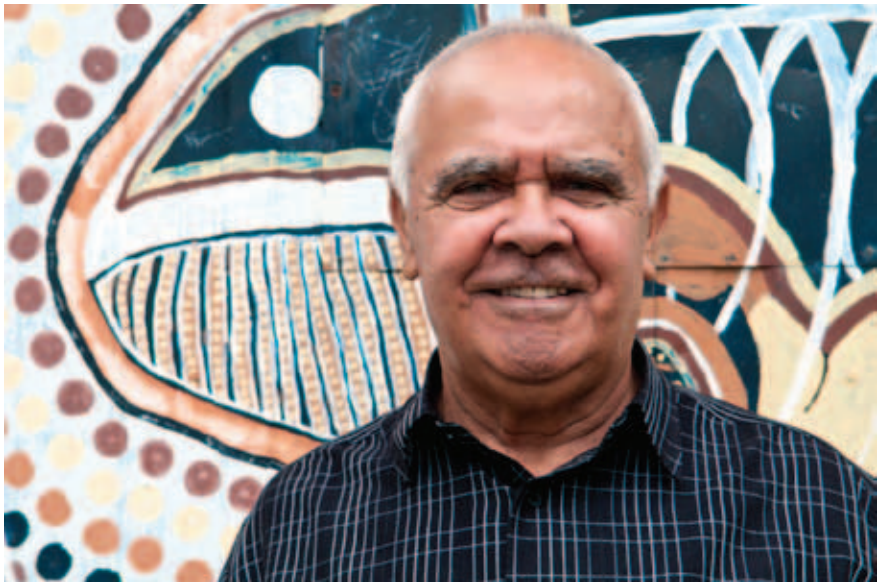
I offered to help with this study because researchers need to find out these things about health and getting older. Otherwise, how are they going to learn, especially about my people?
I'm just glad that I can be helpful.

In old age

Growing old well is important to every one of us. We are investigating how we can maintain the health of the brain and body in older age by detecting dementias such as Alzheimer's disease early, preventing injury through better car safety and understanding why we fall in order to prevent them.

Growing old well

Our research into healthy ageing is providing evidence to help improve the health and longevity of Aboriginal people



Athol Dixon lives in La Perouse, one of the five indigenous communities taking part in our study

> *We are taking every opportunity to promote the research, which is already putting Aboriginal ageing on the national health agenda.*

We all hope to remain healthy into old age, and that includes retaining our memory and ability to think clearly. While we have a good sense of how dementia will affect the general population in the coming years, we know very little about how Indigenous Australian people will be affected. A 2006 study found that people in remote communities in the Kimberley in Western Australia are five times more likely to suffer from dementia than other Australians. The aim of our Koori Growing Old Well Study is to learn more about ageing and dementia in Indigenous people living in urban and regional areas.

In the course of the study, we will talk to around 600 Aboriginal people aged 60 years and over, in five communities in NSW, to investigate risk factors and prevalence of dementia and mild cognitive impairment. We also aim to build the capacity of the community to access services and care for people with dementia.

After completing our pilot study, we improved our questionnaires based on community feedback and began data collection in earnest. By the end of 2010, we had completed over 100 interviews. We hope to complete the majority of assessments during 2011.

We are taking every opportunity to promote the research which is already putting Aboriginal ageing on the national health agenda. This research is laying the foundations for a project to translate current knowledge about dementia into education for health workers in Aboriginal communities, so that Aboriginal people benefit directly from our work.

Sam Bush, whose mother had Alzheimer's disease, is a participant in the early onset Alzheimer's disease study

Alzheimer's: a family affair

People with inherited forms of Alzheimer's disease are helping us find ways to detect the disease early

While Alzheimer's is not normally passed down through the generations, in this study we are working with a very special group of people – those who may have inherited a genetic mutation that is the cause of Alzheimer's disease in the family. Those who carry these mutations will almost certainly develop the disease, usually at an early age.

We know of about 20 such families in Australia, some of whom are participating in the international Dominantly Inherited Alzheimer Network (DIAN) study. The aim of this project is to identify markers of Alzheimer's before there are outward symptoms. These markers will allow us to detect the disease early and treat people before significant damage to the brain has occurred.

In DIAN, adult children of an affected parent give blood and spinal fluid samples for genetic and biomarker studies. They also undergo clinical and cognitive assessment, MRI scans and PET scans to measure protein and metabolic activity in the brain.

In 2010, we recruited ten participants from six families. We hope to recruit a further 20 to 30 participants during the course of the study.

Using ultrasound to 'see' Parkinson's in the brain

Our goal is to diagnose Parkinson's disease early using ultrasound

Ultrasound is a medical imaging technique usually associated with looking at babies in the womb, not the brain. Yet we believe ultrasound could be used as an effective screening tool for Parkinson's disease.

Parkinson's destroys brain cells that control the body's movement, causing trembling, stiffness, slowness and a loss of fine motor control. Currently, there is no diagnostic test. Rather, we rely on detecting subtle problems with movement that, by the time they become obvious, are associated with an enormous amount of damage in the brain.

Our goal is to be able to diagnose Parkinson's early. Ultrasound is non-invasive, readily available and, if our technique proves effective, could be used to inexpensively screen people before the disease takes hold.

In 2010, we began scanning the brains of healthy research participants, focusing on the substantia nigra, an area of the brain that controls the body's movements. In a small proportion of these healthy people, we have identified brain changes which are also typically seen in individuals with Parkinson's. This suggests the marker might point to the disease before movement problems develop.

Up to 70% of susceptible brain cells can die before symptoms become noticeable. Ultrasound will also help us understand these extraordinary coping mechanisms. Our ultimate goal is to mimic, or prolong, these mechanisms to help people with Parkinson's remain symptom-free for longer, even indefinitely.

Assoc Prof Kay Double takes an ultrasound scan of volunteer Lorraine Rayward's brain

Dr Kim Delbaere assesses Max Titterton's confidence when walking

Don't worry, be happy

Fear can increase the risk of falls in older people

Many older people are understandably fearful of falling, as a fall can have devastating consequences such as a loss of independence and placement in institutional care. Yet, having an excessive fear of falling can discourage people from taking part in activities where they believe they might fall. This can lead to a loss of fitness, strength and balance and, ironically, an increased risk of falling.

In 2010, we recruited 500 people, aged 70 to 90 years, to examine their actual physiological fall risk and their perceived fall risk, or fear of falling. We found that almost a third of elderly people under or overestimated their risk of falls. Those people in the study who were overly anxious about falling also had symptoms of depression and neurotic personality traits. We found these psychological characteristics to be just as important a contribution to falling as physical incapacity. In contrast, those people who were overly confident, even if they had a high actual risk, were actually protected against falling. Our study showed that having a positive outlook helps people keep active, which protects against falls.

We have shown that older people can reduce their risk of falls by exercising at least twice a week for an hour each time. They can improve their balance through Tai Chi or other standing exercises that require coordination, agility and quick stepping. However, staying positive is essential to preventing falls. In the future, we plan to look at preventing fear of falling through cognitive behavioural therapy, combined with exercises to improve balance.

A dose of sunshine may prevent falls

Sunshine and vitamin D are essential for maintaining physical strength and cognitive abilities, and may also help prevent falls

While there is no doubt that too much sun can be harmful, many older Australians appear to have too little sunshine exposure and, as a result, insufficient vitamin D levels.
With ageing, the human skin has less capacity to synthesise vitamin D and many older people also spend more time indoors as a result of frailty, immobility or illness.

While low levels of vitamin D have been associated with an increased risk of falls in people living in aged care facilities, we don't know whether the same is true for all older people. In 2010, we investigated the relationship between vitamin D levels and falls in people aged 70 to 90 years living in the community.

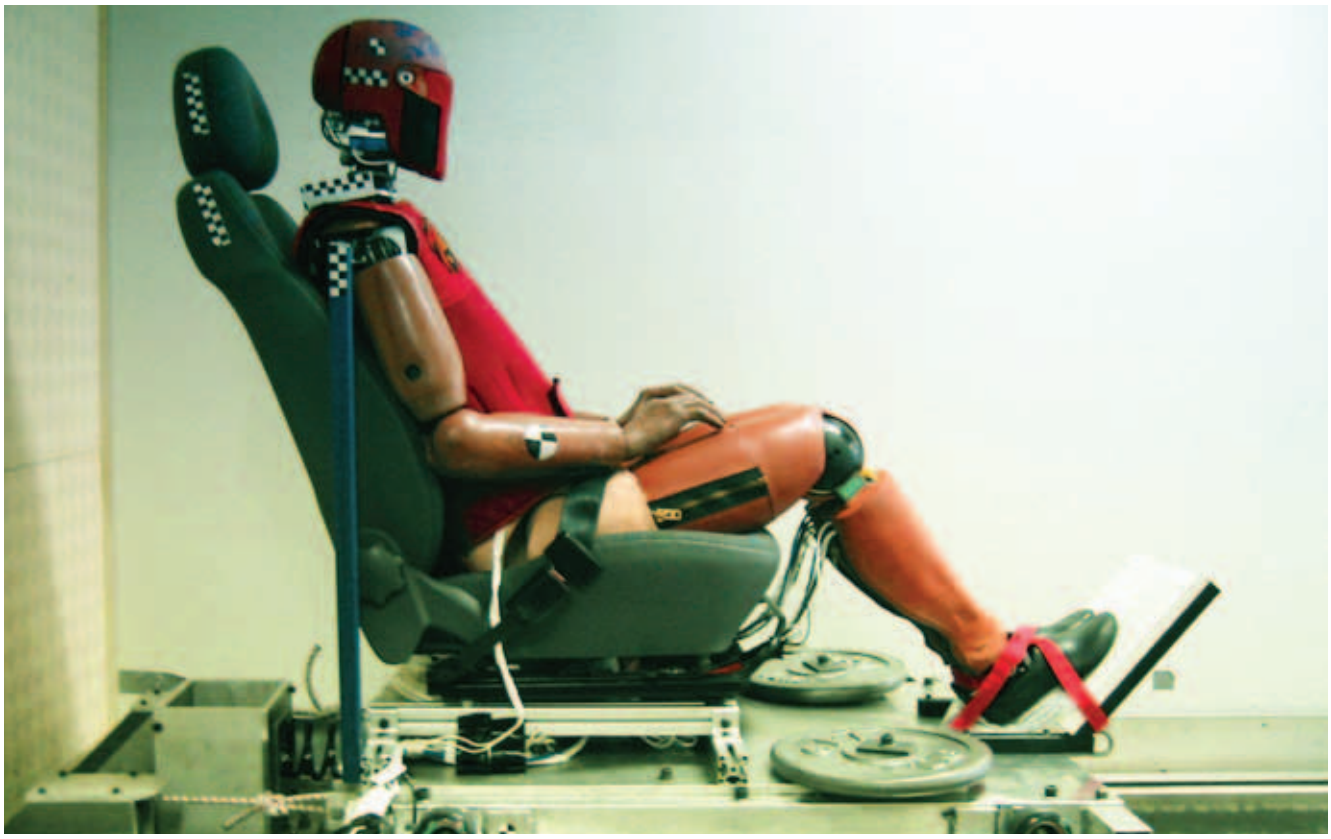
We found that approximately one third of our participants were vitamin D deficient. These people were also weaker, had a slower reaction time, poorer balance and slower gait, and performed worse in cognitive function tests. In men, we found that vitamin D insufficiency was associated with an increase in falls.
Our study suggests that a balance is required between avoiding an increase in the risk of skin cancer by excessive sun exposure and achieving enough sun exposure to maintain adequate vitamin D levels. In particular, those with lower cognitive and physical performance may benefit from spending more time outdoors or taking vitamin D supplements.

These results provide further evidence for ensuring that vitamin D levels are adequate in all older people, particularly as the benefits seem to extend beyond cognition and the musculoskeletal system to our ability to prevent ourselves from falling.

Esma Graham and Dorothy Polden enjoy the many benefits of sunshine

Safety in the rear seat

Counter-measures to reduce injury will improve rear seat safety



Crash test dummies will help us develop new rear seat safety technology

For a long time, the rear seat of cars has been considered safer in an accident than the front seat. However, while safety technology has improved in the front seat over the past decade, rear seat safety has not kept up. Approximately 23% of serious injuries are suffered by people travelling in the rear seat, but only 15% of all journeys are made seated in the rear of the car, which suggests that the rear seat could be safer. This is particularly important for older people, as they have been shown to have the highest risk of injury of rear seat occupants.

In 2010, we began an in-depth investigation of how people are injured in a crash. We are recruiting rear seat occupants injured in accidents, reviewing their medical records and inspecting the vehicle and site of the crash. We will also evaluate new and existing restraint technologies, such as airbags and advanced seat belt designs, on a crash sled using crash test dummies.

Over the past year, we have investigated the effect of the rear seat cushion on abdominal injuries, and have published a study on the reduction in protection provided to rear seat occupants in newer vehicles.

Once complete, this research will enable us to determine the best counter-measures to reduce injury to rear seat occupants. Our aim is to highlight to the Government and the Australasian New Car Assessment Program the need to shift some of their focus to the rear seat.

Read more about our research at ***www.neura.edu.au***

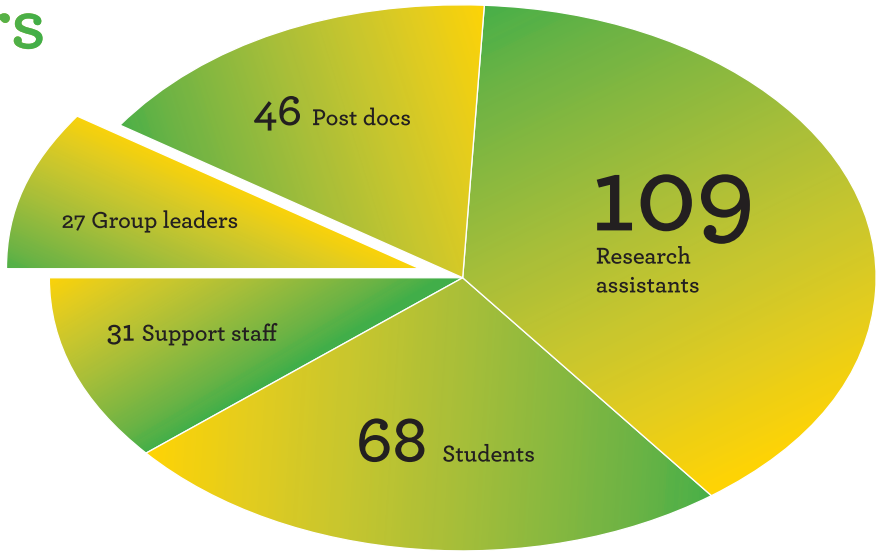
A shared vision

The dedication of our scientists is what makes progress in neuroscience research possible.

In 2010, we expanded by 11.2% to a total of 281 staff members

In the 20 odd years since we realised our vision of an internationally recognized medical research institute, we have grown significantly.

We now number 281 staff members, including 27 group leaders who are not only responsible for leadership and mentoring, but also for growing and developing research at NeuRA. We'd like to introduce two new group leaders, Dr Penelope McNulty and Dr Julie Brown.



Profile

Dr Penelope McNulty

Dr Penelope McNulty has always been fascinated by the science of human movement, and leads a group looking at how muscles and sensory organs provide information to help control the hand and upper limbs.

"We want to understand how this changes after stroke and spinal cord injury, so that we can improve people's response to rehabilitation. Our goal is to help people live more independently," she says.

Penelope is developing rehabilitation therapy that is fun as well as effective. Having originally trained as a classical dancer, she understands that enjoyment is key to perseverance. "If therapy is fun, people do more and recover more quickly. They also receive ancillary benefits, such as a cardiovascular work out and alleviation of their depression," she says.

Penelope says one of the most rewarding aspects of her work is being able to give people hope. "I had one patient who had been a keen golfer, but could barely use his arm. After Wii therapy, he felt ready to try putting using a real golf club. He was over the moon."

An Australian first

We continue to explore myriad aspects of neuroscience and contribute to scientific literature, with almost 200 journal articles and four books published this year. In 2010, Prof Matthew Kiernan was selected to be the first Australian editor of the prestigious Journal of Neurology, Neurosurgery and Psychiatry.

When Prof Matthew Kiernan was a young medical registrar, he took a piece of advice that would change the course of his career: "Any doctor can merely treat the patient. Your job is to find a cure."

This counsel became Matthew's compass and led him to a small, yet amazingly productive research team housed in a small villa on a dusty plot behind the Prince of Wales Hospital. Today that site is NeuRA, where Matthew is a senior scientist and, most significantly, the editor of the prestigious international Journal of Neurology, Neurosurgery and Psychiatry.

The seven-year tenure is a win for Australasian research and NeuRA in particular, with Prof Kiernan now a conduit for the world's top research into the full gamut of neurological disorders, from stroke, epilepsy, Parkinson's disease and multiple sclerosis through to rare and as-yet unfathomable diseases.

Connections like this will ensure that NeuRA remains a world hub for producing and attracting cutting-edge research and scientists. It is a testament to the maturation of local research that Prof Kiernan is the journal's first Australian editor – a journey from those first early days in makeshift laboratories to steering one of the world's foremost neuroscience journals.

Profile

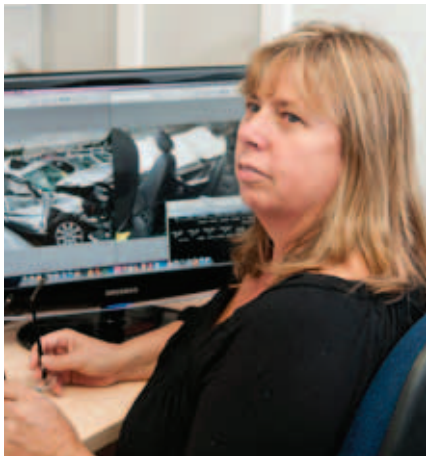
Dr Julie Brown

Having worked at the Roads and Transport Authority for over a decade, Dr Julie Brown is well-versed in developing policy to improve the safety of passengers travelling in cars. When she saw the need for more robust scientific evidence to support these policies, she decided to make the move to research.

Julie's group focuses on reducing injury among the most vulnerable Australians: children and the elderly.

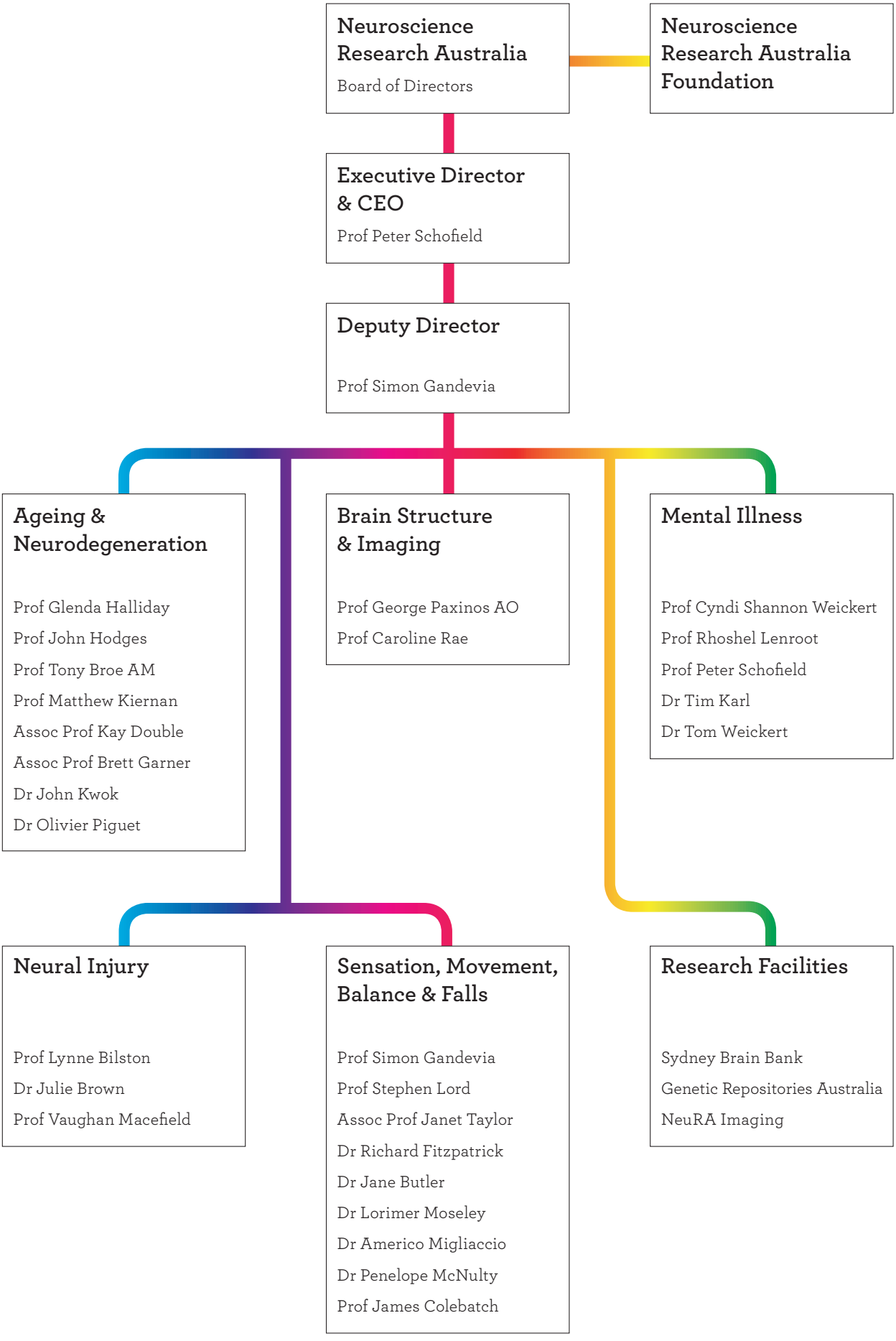
"We look primarily at transport-related injuries, but I'm also looking at falls in young children and, in particular, why some groups in the community, for example diverse cultural and linguistic groups, are at a higher risk of these types of injuries."

"My background in policy development helps me design research that has translatable outcomes. I was at the motor registry the other day and the posters advertising restraint fitting stations referenced one of my papers. I really enjoy seeing our results make a tangible difference."



Read more about our research at ***www.neura.edu.au***

Our research leaders



Funding our important research

Caitlin Harris, St Catherine's School Waverley presents a cheque to Prof Peter Schofield

As a not-for-profit organisation, Neuroscience Research Australia is funded by a combination of government grants and private donations

Of a total of $28.26 million in 2010, approximately one half came from competitive external grant funding from a number of national and international organisations including the National Health and Medical Research Council and the Australian Research Council. In 2010, our researchers held a total of 157 grants, fellowships, scholarships and awards totalling $12.84 million.

Donations and bequests play a key role in allowing us to continue our important research. Approximately 12% of our income came from fundraising in 2010, while 3% came from bequests. It is only through this ongoing support that we can hope to improve the health and wellbeing of so many people in the community.

We spent 29% of our income on capital works. Turn to page 42 to read about our exciting progress in building the first stage of the Neuroscience Research Precinct.

A full copy of the audited Financial Statements, including Notes to the Financial Statements and the Audit Opinion, can be obtained online at www.neura.edu.au or free of charge on request to the Finance Manager, Neuroscience Research Australia, Barker Street, Randwick NSW 2031.

Our board of directors

Paul Brassil, BEc LLB ACA FTIA
Director, 1997 – present
Chairman of NeuRA Board, 2004 – present
Chairman of NeuRA Foundation Board, 2007 – present
Chairman, Audit Committee
Member, MRI Committee
Independent Director

John Grill, BSc, BE(Hons), Hon DEng
Director, July 2010 – present
Independent Director

Michael Quigley, BSc BE
Director, 2008 – present
Independent Director

Andrew Bernard, BSc MPH
Director, 2008 – present
Member, Investment Committee
Nominee of South Eastern Sydney & Illawarra Area Health Service

Prof Mike Calford, BSc(Hons) PhD
Director, 2009 – present
Nominee of National Health & Medical Research Council

The Hon Dr Andrew Refshauge, MBBS FAICD
Director, 2005 – present
Member, Audit Committee
Member, Building Committee
Member, MRI Committee
Nominee of NSW Minister for Health & Medical Research

Barry Shepherd, PSM, Grad.Dip PSM
Director, 2005 – present
Chairman, Building Committee
Nominee of South Eastern Sydney & Illawarra Area Health Service to Sept 2010 then Independent Director

David Thomas
Director, 1997 – April 2010
Independent Director – appointed Honorary Life Governor on retirement

John Walton,
AM BEc MBA FCPA AASA FAICD FAIM
Director, 1991 – May 2010
Member, Investment Committee
Member, MRI Committee
Independent Director – appointed Honorary Life Governor on retirement

Prof Peter Smith,
RFD MD FRACP FRCPA FAICD
Director, 2005 – present
Nominee of University of New South Wales

Gabrielle Upton, BA LLB MBA FAICD
Director, 2007 – present
Nominee of University of New South Wales

Prof Peter Schofield, PhD DSc
Executive Director and Chief Executive Officer, 2004 – present

Foundation board

Graeme Bradshaw
Director, NeuRA Foundation, 2007 – present

Ian Kennedy, OAM
Director, NeuRA Foundation, 2009 – present

Sally Manion, BCom CA CFP
Director, NeuRA Foundation, June 2010 – present

James Williams, BCom LLB LMusA FCIS
Director, NeuRA Foundation, 2007 – March 2010

Endless possibilities

The first stage of the precinct, the Neuroscience Research Australia (NeuRA) building, is due for completion in December 2011. Its design emphasises the strong connection between our research and the well-being of the community. From the welcoming, glass-walled entrance to the open, light-filled reception area, patients and visitors will feel they are in an environment mindful of the human side of our research.

Improving the lives of people with neurological or psychiatric conditions demands facilities that foster intense scientific investigation. We will deliver this, and so much more, through rapidly translating our laboratory research findings to clinical care and, ultimately, to cures.

Our new building provides seven floors of laboratory and clinical research space designed to foster innovation within multidisciplinary teams of scientists and clinicians. Working side by side, their collaborative efforts will overcome today's most pressing medical research challenges.

Our current studies on prevention of childhood injury are saving lives, while our research on autism and reading disabilities addresses the urgent needs of our young children. Our internationally recognised work on schizophrenia assists vulnerable people just as their adult lives are starting. In an ageing community, our focus on

Designed to enhance progress in science and ultimately patient care, the Neuroscience Research Precinct will be a place of endless possibilities.

Alzheimer's and other dementias, and the prevention of falls in the elderly, will greatly improve the quality of life of the elderly and reduce the healthcare burden that greatly impacts on our community.

Government grants and private donations have played a critical role in funding this first stage of the Neuroscience Research Precinct. The spirit of generosity among those private individuals who have already given is not only inspiring, but vital.

The current $44m budget funds the construction of the building shell and the fit-out of two of the seven research floors. We now need to raise $15 million to transform the remaining empty floor space into vibrant, productive laboratories.

Will you help us? As an independent, not-for-profit research institute, we urgently need your support. We will keep you up to date with how your donation has been used and, as the fit-out of the building progresses, we will show you tangible results of your philanthropy in action.

To discuss a gift in support of our research, please contact the NeuRA Foundation on ***02 9399 1262.***

You can also make a donation by using the form at the back of this publication or by visiting us at ***www.neura.edu.au.***

Our community of supporters

The NeuRA community supports our research in many ways, from one-off gifts and bequests to regular donations, while thousands of people take part in our Bridge for Brain Research Challenge

Marion Rice

A Bridge for Brain Research Challenge participant

“It’s up to the community to support and donate to research. Without research there would be no advances in prevention and cures.”

At 101 years young, Marion Rice is the oldest member of the NSW Bridge Club in Surry Hills. She says she has been playing Bridge for ‘only’ 40 years. “I started late,” says Marion. “I wish I had learnt earlier.”

Marion has participated in our annual Bridge for Brain Research Challenge for the past eight years. “Nowadays Bridge is everything to me. It’s my only interest. It gets me out of the house, I make and meet kind friends, it gives me intellectual stimulation and a chance to socialise. It’s probably what keeps me going,” she says.

Marion says she’s been fortunate that none of her family or friends has been affected by diseases like Alzheimer’s, but she still believes in the importance of supporting medical research. “It’s up to the community to support and donate to research. Without research there would be no advances in prevention and cures.”

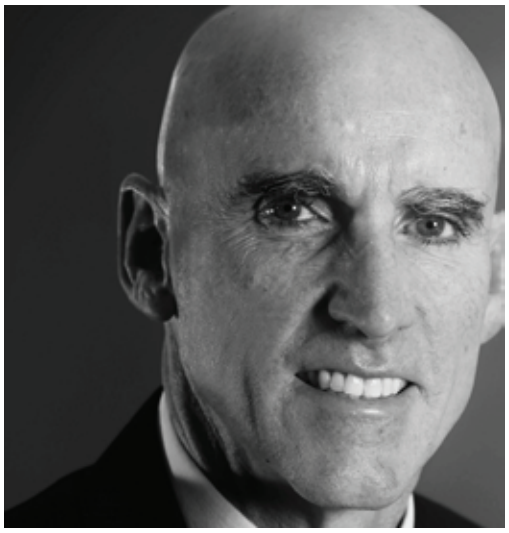
Mike Quigley

NeuRA board member and Chief Executive of the National Broadband Network (NBN)

Mike Quigley says that after surviving a life-threatening battle with leukaemia, he's a true believer in what science can do for society.

"Anyone of us can succumb to these diseases and I think, as a society, we only advance by understanding science and then finding the technologies that spin out of that. I was a direct recipient of that, and I wouldn't be here if people didn't pump money into leukemia research 20 and 30 years ago. Fortunately I got through that and now I'm working on the NBN."

Mike donated his first year's pay cheque to aid our research into brain diseases and stroke rehabilitation. The donation will fund a project to deliver remote rehabilitation therapy to stroke patients using the Nintendo Wii games console and the high speed broadband provided by the NBN.



My gift

Please use this form to make your tax-deductible donation today. You can also donate online at **www.neura.edu.au** or by calling **1300 888 019**

YES, I want to support the important work of Neuroscience Research Australia with the following:

One-off gift: $

Or monthly gift: $50, $100, $200, $500

Please direct my gift towards:

◯ NeuRA's area of greatest need

◯ NeuRA's building project

About me

Title

First name

Surname

Date of birth

NeuRA is privacy compliant. Providing your date of birth can be helpful in proving your identity when contacting us.

Street address

City / State / Postcode

Phone (home / work)

Mobile

Email

Payment

My cheque/Money Order payable to Neuroscience Research Australia is enclosed or

I would like to make my donation by:

◯ Visa ◯ MasterCard ◯ Amex ◯ Diners

Card number

Expiry date

Cardholder's name

Cardholder's signature

See over

My gift

I am most interested in the following areas of NeuRA's work:

(please tick all applicable)

- Alzheimer's disease
- Autism
- Balance disorders
- Bipolar disorder
- Child injury
- Chronic pain
- Dementia
- Dyslexia
- Falls in the elderly
- Frontotemporal dementia
- Motor neurone disease
- Muscle fatigue
- Sleep apnoea
- Parkinson's disease
- Schizophrenia
- Spinal cord injury
- Stroke rehabilitation
- The Neuroscience Research Precinct
- Please send me information on leaving a gift in my Will to NeuRA

Thank you for your generous support

Please return this form by fax to 02 9399 1082 or by mail to:
Neuroscience Research Australia
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ABN 94 050 110 346

Joan Heaney

A NeuRA bequestor

When he was in his sixties, Joan Heaney's younger brother, Brian, developed a degenerative brain disorder, related to Parkinson's disease, called corticobasal degeneration.

Joan was devastated. "Brian was like a second dad to my daughters. He and my husband, Kevin, were great mates; he was more than just a brother-in-law."

Brian gradually lost the ability to control his body and speak. "When he was little, Brian couldn't say 'Joan' so he called me 'La La'. Near the end, when he could no longer speak or move, he could still say 'La La' to me and follow me with his eyes to let me know he was still there," says Joan.

Brian died on 14 November, 2007. He had already made the decision to donate his brain to research in the hope that, one day, NeuRA would discover a cure for his disease. "Brian felt so passionately about this that I thought, I'm going to leave a gift in my Will," says Joan.

"I have a lot of confidence in research," she says. "My grandson's girlfriend is studying neuroscience at university at the moment. That gives me hope that a cure for this disease will be discovered one day."

Leaving a gift in your Will to NeuRA, however large or small, will help us find the cures so urgently needed by millions of Australians. It won't cost you anything now, but is one of the simplest and most powerful ways to support our work into the future. Visit our website or call us on 1300 888 019 to find out how.

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