

Queensland Brain Institute 2013 Annual Report



THE UNIVERSITY
OF QUEENSLAND
AUSTRALIA

QBI

Queensland Brain Institute

TABLE OF CONTENTS

UQ Vice-Chancellor and President's Report	2	Jiang Laboratory	34	Students	58
QBI Director's Report	3	Lynch Laboratory	35	Student stories and profiles	60
Discovery	4	Mangelsdorf Laboratory	36	Master of Neuroscience students	61
Blocking a protein could be key to treating spinal cord injury	6	Marshall Laboratory	37	Community	62
New learning and memory neurons uncovered	8	Mattingley Laboratory	38	Lectures	64
Shedding light on brain computations	10	McGrath Laboratory	39	Events	66
Older grandfathers pass on autism risk through generations	12	Meunier Laboratory	40	Community Outreach	68
Research into axon degeneration	14	Mowry Laboratory	41	Australian Brain Bee Challenge	69
Study reveals molecular networks of mental health disorders	16	Osborne Laboratory	42	Recognition	70
Research	20	Piper Laboratory	43	Fellowships and Awards	72
Bartlett Laboratory	22	Reinhard Laboratory	44	Commercialisation	73
Bredy Laboratory	23	Richards Laboratory	45	Publications	74
Burne Laboratory	24	Sah Laboratory	46	Grants	84
Cheung Laboratory	25	Srinivasan Laboratory	47	Neuroscience Seminars	86
Claudianos Laboratory	26	van Swinderen Laboratory	48	Professional Service	88
Cooper Laboratory	27	Visscher Laboratory	49	Editorial Boards	89
Coulson Laboratory	28	Williams Laboratory	50	UQ Appointments	90
Cunnington Laboratory	29	Wray Laboratory	51	Staff	91
Eyles Laboratory	30	Clem Jones Centre for Ageing Dementia Research	52	In Appreciation	94
Goodhill Laboratory	31	Science of Learning Research Centre	54	Supporting QBI	95
Götz Laboratory	32	Joint Sino-Australian Neurogenetics Laboratory	55		
Hilliard Laboratory	33	Joint Laboratory of Neuroscience and Cognition	56		
		Joint Sino-Australian Laboratory of Brainnetome	57		

UQ VICE-CHANCELLOR AND PRESIDENT'S REPORT



In 2013, the Queensland Brain Institute (QBI) celebrated its 10th anniversary.

In the past 10 years, the Institute has achieved remarkable success and made credible inroads into neuroscience research, one of the final frontiers of science. Since Founding Director Professor Perry Bartlett co-authored QBI's first publication in the prestigious *Nature Neuroscience* journal in 2002, the Institute has published over 1200 papers.

QBI's excellence in the field saw The University of Queensland (UQ) attain the highest possible score of five for neuroscience, "well above world standard", in both the 2010 and 2012 Excellence in Research for Australia (ERA) reviews — one of only two universities in Australia to achieve this.

The quality of work produced by QBI researchers is further demonstrated by the Institute's National Health and Medical Research Council and Australian Research Council grant success, attracting over \$110 million in competitive grant funding to date — a success rate far above the national average.

QBI's research power is further augmented by its many international partnerships. From its first international research assignment with the Chinese Academy of Sciences Institute of Neuroscience in Shanghai in 2006, QBI is now a truly global research hub. It has grown to establish a Joint Laboratory of Neuroscience and Cognition with the Institute

of Biophysics, Beijing, in 2010 and a Joint Sino-Australian Neurogenetics Laboratory with the Second Military Medical University, Shanghai, in 2011. This past year saw the inception of the Joint Sino-Australian Laboratory of Brainnetome with the Chinese Academy of Sciences Institute of Automation, Beijing, using advanced neuroimaging techniques and computational analysis to understand brain behaviour.

A growing area in neuroscience, namely computational analysis and neuroimaging, will also be used in the newly established Science of Learning Research Centre (SLRC). These techniques will be used to understand the networks involved during learning and the formation of memories, and then applied to deliver more effective education models in schools and universities — a first for Australian education.

The opening of the SLRC in November along with the unveiling of the Clem Jones Centre for Ageing Dementia Research in February, have helped strengthen the University's relationship with State and Federal Government. Over the past 12 months the Centres have attracted many representatives from State and Federal Parliament to QBI, including Prime Minister Tony Abbott. We are incredibly grateful for the significant support these areas have received from Government, at a local, state and national level.

QBI is committed to unearthing the next generation of neuroscientists by creating pathways for high school students into the field. Since 2006, it has facilitated the Australian Brain Bee Challenge (ABBC) neuroscience competition. It is designed to motivate students to learn about the human brain and inspire them to consider careers as clinicians and researchers. Today, ABBC engages with over 5500 students from 300 schools across Australia and New Zealand. The initiative is further supported by a Summer Scholarship scheme at QBI for students who excel in the competition. The demonstrated passion and outstanding leadership of QBI's Professor Linda Richards in initiating the internationally recognised competition in Australia, saw her awarded first place in the Vice-Chancellor's Equity and Diversity Award in May. Her commitment to growing the calibre of young Australian neuroscientists has seen the previous two Australian winners, Jackson Huang (2013) and Teresa Tang (2012) both going on to win the International Brain Bee Competition.

As we embark on the next decade of research at QBI, I wish to thank Professor Bartlett, Professor Pankaj Sah, Deputy Director (Research) and Mr John Kelly, Deputy Director (Operations) and their able team of research and administration support for shaping the Institute into the world-class research hub that stands today.

This annual report highlights the breadth of QBI's work and showcases some recent findings, including a fundamental breakthrough into how the brain decodes the visual world.

I have no doubt that we will continue to see many, many more significant findings in neuroscience emerge from QBI in the decades to come.

Professor Peter Høj
Vice-Chancellor and President



QBI DIRECTOR'S REPORT



This year marks 10 years since the Queensland Brain Institute (QBI) was established.

As the Founding Director, I have had the enormous privilege of observing first-hand this exciting period in which we grew from 10 staff to more than 400, from 3 laboratory heads to 33, from publishing 2 papers to 239, and brought in competitive grant funding in excess of \$110 million and philanthropic funding in excess of \$43 million. More important has been the impact and recognition our research has garnered over this period, which is exemplified by the increasing number of papers published in frontline journals, such as *Nature*, *Science* and *Proceedings of the National Academy of Sciences USA*, as well as UQ receiving the highest possible rating for neuroscience excellence in both the 2010 and 2012 Excellence in Research for Australia (ERA) reviews. This success has been due to the exceptional scientists – whom we have been fortunate to attract from all over the globe – who have made key discoveries that greatly contribute to our understanding of how brain function is regulated at the most fundamental level. Equally important has been the incredible support given to us by the people of Queensland through philanthropy and government contributions.

We have made great strides in understanding the circuitry regulating brain function in health and disease. We now have begun applying this knowledge to the development of new therapeutics and approaches to combat a wide range of neurological and psychiatric diseases, as well as to develop new innovative approaches to deliver learning into our schools. Thus, the future decade looks even more exciting and fruitful as we build upon the scientific foundations of the past decade.

I was especially delighted by the immense success QBI achieved in 2013, as outlined below and more fully in this Annual Report, as it showcased the impressive achievements of the last decade.

In 2013, on February 28, the Clem Jones Centre for Ageing Dementia Research (CJCADR) was officially opened by Queensland Premier Campbell Newman. Establishing this, Australia's first and only facility focussed entirely on research into the prevention and treatment of dementia, was made possible through the generosity of philanthropic partners, in particular the Clem Jones Trustees whose financial support allowed us to assemble an unparalleled research team, led by Professor Jürgen Götz. The significance of the establishment of CJCADR captured the attention of both State and Federal Governments, who collectively committed a further \$18 million in funding, to accelerate our research towards finding a cure for dementia.

This year, the Australian Research Council (ARC) provided \$21 million for a Science of Learning Research Centre (SLRC), a Special Research Initiative housed at QBI. The SLRC was opened by the Honourable Ian Walker, Minister for Science, Information Technology, Innovation and the Arts in November, and attended by ARC CEO Professor Aidan Byrne. Based around the three themes of understanding learning, measuring learning and promoting learning, the SLRC's research findings will help Australia build a solid scientific evidence base that directly informs teaching practices, applying benchmark discoveries directly to the blackboard. The establishment of the SLRC was especially gratifying for me, as I had been part of an expert working group that had successfully presented the concept four years earlier to the Prime Minister's Science, Engineering and Innovation Council.

QBI scientists were extremely successful in the 2013 grant rounds of the National Health and Medical Research Council (NHMRC) and the ARC. In the NHMRC round, 17 projects were funded, totalling more than \$10.47 million at a success rate of 44.7 per cent – this was considerably higher than the 16.7 per cent national average. In addition, Professors Joe Lynch and Fred Meunier received NHMRC fellowships worth \$1.39 million, and were promoted.

In the ARC round, QBI had a 44.4 per cent success rate (again, well above the national average of 19.9 per cent), totalling \$2.6 million. Among the successful applicants was Professor Mandyam Srinivasan, who was awarded

two grants totalling almost \$1.5 million. One of these grants also included the very prestigious Discovery Outstanding Researcher Award.

I am delighted to report that QBI scientists received several prestigious accolades for their work this year. The NHMRC John Cade Fellowship in Mental Health Research was awarded to Professor John McGrath. A leader in this field of research, Professor McGrath discovered that low vitamin D during early life is a risk factor for schizophrenia. This Fellowship, worth \$3.75 million, will enable Professor McGrath to expand his current research to examine the association between early life vitamin D status and childhood brain development, as well as mental health in later life. Dr Jian Yang was awarded \$1.22 million to unlock the genetic basis of thousands of diseases including schizophrenia, obesity and cancer. As one of only two recipients of the highly prestigious Sylvia and Charles Viertel Charitable Foundation's Senior Medical Research Fellowship, Dr Yang will apply his background in genetics and statistics, along with his outstanding skills in computational biology, to answer fundamental questions about the genetics of common disease, and to develop software tools that will analyse millions of DNA markers. Dr Yang's achievement is particularly pleasing with the knowledge that QBI's Deputy Director (Research) Professor Pankaj Sah, was the first recipient of this Fellowship when it was launched in 1995.

Two new Fellowships were established in 2013 through the generosity of philanthropic support. A \$1.4 million Senior Research Fellowship was jointly funded by the charitable arm of the Freemasons Queensland, the Board of Benevolence of Aged Masons, Widows and Orphans' Fund, and UQef, a private fund established by Wotif.com founders Andrew Brice and Graeme Wood, and will support an outstanding researcher in the area of learning and memory over 5 years. A further \$500,000 was donated by Robyn Hilton to establish the Peter Hilton Research Fellowship in Ageing Dementia in honour of her late husband. The Fellowship will fund an early-career researcher based at CJCADR for 5 years, who will explore how memory and learning functions are disrupted in dementia, and develop procedures to test and manage these dysfunctions.

As exemplified above, philanthropic support has been fundamental to the success of QBI and we are enormously grateful for the support of our donors.

Since the Maclean family made the very first donation to the Institute 10 years ago, we have attracted more than \$43 million in support from generous individuals, businesses and from charitable organisations. My heartfelt thanks to all of our donors, and to our Development Board, chaired by Jeff Maclean, who invest so much time and energy into ensuring the continuation of the philanthropic support. Our Director of Advancement and Communications Mikaeli Costello also has been central to the success of our philanthropic effort.

Also, I wish to thank our Advisory Board, chaired by Dr Sallyanne Atkinson AO, whose support and counsel have played an invaluable role in guiding QBI to become the global-leading research organisation it is today.

In addition, I would like to acknowledge the unwavering support I have received from my scientific colleagues at QBI – not only over the past 12 months but also throughout our exciting journey over the past decade. Our dedicated operations team, led by Deputy Director (Operations) John Kelly, and Manager Helen Weir, have provided the unparalleled, outstanding support so vital to the success of our research programs.

My very great gratitude goes to the Deputy Director (Research) Professor Pankaj Sah who has been instrumental in orchestrating the success we have achieved in obtaining research grants, and his team of Rowan Tweedale and Dr Sylvie Pichelin who are peerless in this area.

Finally, my warm thanks to Vice-Chancellor Professor Peter Hoj, Senior Deputy Vice-Chancellor Professor Deborah Terry and Deputy Vice-Chancellor (Research) Professor Max Lu for their guidance and support.

I hope you enjoy reading about QBI's research discoveries in more detail in this Annual Report.

Professor Perry Bartlett FAA
Director

DISCOVERY



Discovery

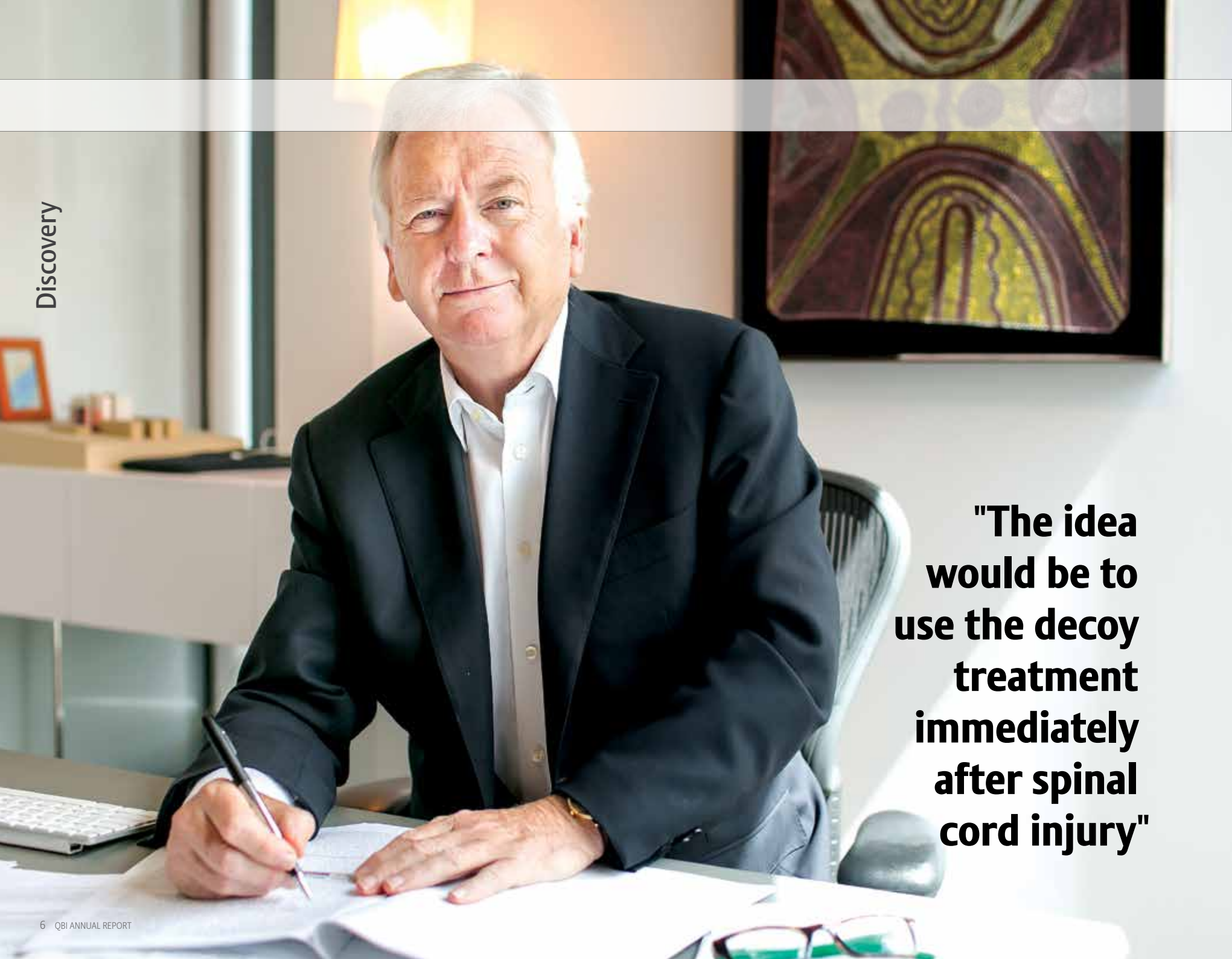
In only 10 years, the Queensland Brain Institute has positioned itself as one of the world's leading neuroscience research facilities.

QBI fosters an environment of discovery that will ultimately lead to the development of much needed therapeutic treatments to combat diseases in which brain function has failed or is compromised.

Here, we celebrate some of QBI's fundamental breakthroughs in 2013.

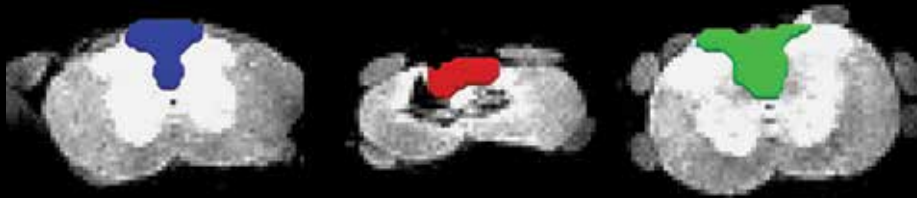


Vinod Narayana, Meunier laboratory.

A man with white hair, wearing a dark suit jacket over a light-colored button-down shirt, is seated at a desk. He is holding a pen in his right hand and appears to be writing on a document. His left hand rests on the desk. In the background, there is a framed abstract painting with yellow, green, and purple patterns. A computer keyboard and a mouse are visible on the desk to the left. A pair of glasses is on the desk in the foreground.

**"The idea
would be to
use the decoy
treatment
immediately
after spinal
cord injury"**

Blocking a protein could be key to treating spinal cord injury



Many people who have suffered spinal cord injury experience permanent deficits in movement and sensation, which is primarily due to the inability of the mammalian central nervous system to repair itself.

In 1998, Professor Perry Bartlett, together with Professor Andrew Boyd, first identified that a protein called EphA4 may be the key to solving this problem.

“Our work demonstrated that the EphA4 protein was critical to the development of the nerves that control walking and other complex muscle functions,” Professor Bartlett said.

The study was the first to show this relationship and paved a new path for research into the treatment of diseases and injuries involving motor nerves.

Subsequent studies by the Bartlett and Boyd team showed that after spinal cord injury, the production of EphA4 increased and stopped severed nerve endings from regrowing through the injury site.

The team then developed a “decoy” protein, a competitive antagonist called EphA4-Fc, to block EphA4 function.

The most recent study the team conducted assessed the effect of blocking the activity of EphA4 using this decoy protein in a rat model of thoracic contusive spinal cord injury.

Using tests of locomotion and limb function – a ledged tapered balance beam and open-field testing – they observed significant improvements in recovery of movement after treatment with EphA4-Fc.

Using high-resolution magnetic resonance imaging, they found that rats treated with EphA4-Fc also showed structural changes in the tissue near the injury site.

More specifically, they found a significantly increased cross-sectional area of the dorsal funiculus caudal to the injury epicentre, when compared with rats that had not received the decoy protein treatment.

“The idea would be to use the decoy treatment immediately after spinal cord injury to try to improve the patient’s recovery,” Professor Bartlett explained.

“And as a neurologist or neurosurgeon will tell you, if you could improve function even marginally for a quadriplegic, you could make a massive difference to their life.”

This research was supported by funding from the Lisa Palmer Spinal Research Consortium and SpinalCure Australia.

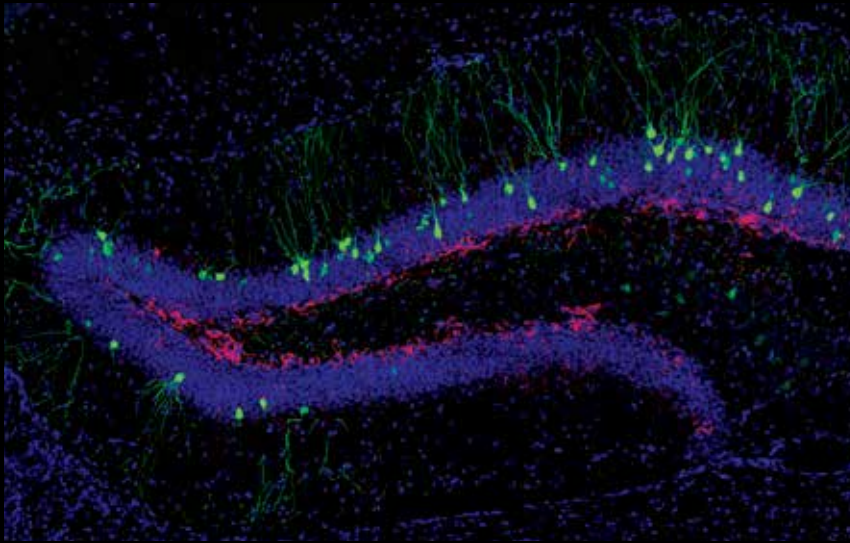
The paper, “Acute delivery of EphA4-Fc improves functional recovery after contusive spinal cord injury in rats”, was published in the *Journal of Neurotrauma*.

Above: Diffusion tensor imaging (DTI) analysis of spinal cord injury after treatment with EphA4-Fc (mid-sagittal view). *Journal of Neurotrauma*, 2013.
Far left: Professor Perry Bartlett.

"This research allows for better understanding of the processes underlying learning and memory"



New learning and memory neurons uncovered



Though an area of the brain called the hippocampus is known to be important for learning and memory, exactly how the cells in this region are involved in these processes has remained an area of intense research.

New neurons are continually produced in the brain throughout life, and pass through a number of developmental stages before becoming fully mature.

A recent discovery from the Bartlett laboratory has demonstrated precisely when these neurons become important for learning in adult animals.

Dr Jana Vukovic, a postdoctoral researcher in the Bartlett laboratory, was part of a team that developed a mouse model in which newborn hippocampal neurons could be specifically deleted through the injection of a toxin.

Using a challenging behavioural test (a modified version of the active place avoidance test), the team has direct evidence that immature neurons are required for successful acquisition of spatial learning but are not necessary for memory retrieval.

“Using a genetic technique to delete immature neurons in animal models, we found they had great difficulty learning a new spatial task.

“On the other hand, if the animals needed to remember a task they had already mastered in the past, before these immature neurons were deleted, their ability to perform the task was the same – so, they had remembered the task they learned earlier,” Dr Vukovic explained.

Importantly, the observed learning deficits were rescued as newly generated immature neurons repopulated the ablated area once the toxin treatment ceased.

If the pool of immature neurons was deleted again and the mice challenged with a novel spatial learning task, the behavioural deficits were again evident.

“The new neurons appear particularly important for the brain to detect subtle but critical

differences in the environment that can impact on the individual,” Dr Vukovic said.

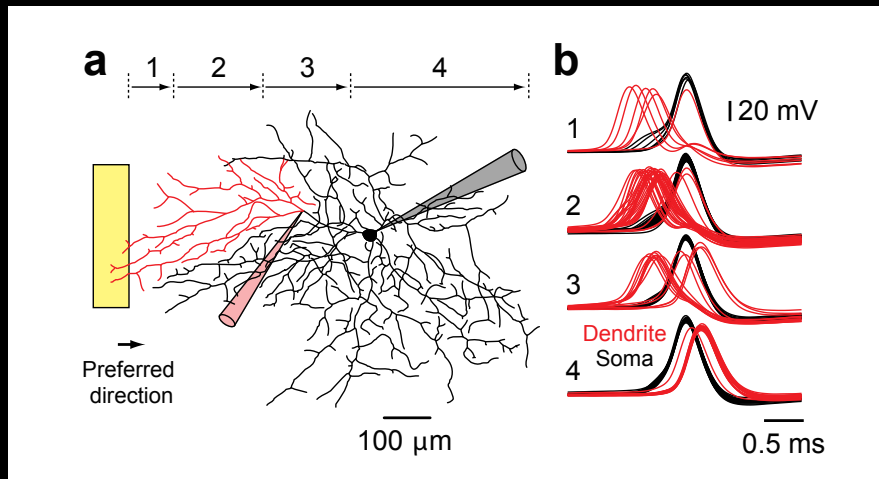
She said that this research allows for better understanding of the processes underlying learning and memory formation and highlights the potential of stimulating neurogenesis as a means to enhance learning.

The paper, “Immature doublecortin-positive hippocampal neurons are important for learning but not for remembering”, was published in the *Journal of Neuroscience*.

Above: Section through the mouse hippocampus, showing immature, doublecortin-positive neurons (red) and activated, Arc-positive neurons (green) in the granule cell layer of the dentate gyrus. Far left: Dr Jana Vukovic.

**"Discovering how
nerve cells process
information is
fundamental to
understanding
how we learn"**

Shedding light on brain computations



The retina, a thin neuronal network located at the back of the eye, transforms light input into patterns of action potential output, signalling key aspects of visual stimuli to higher visual centres. The mechanisms underlying such retinal neuronal computations are, however, poorly understood.

Using advanced electrical recording techniques, Dr Ben Sivyer and Associate Professor Stephen Williams have discovered how electrical activity in the dendrites - the thin cable-like extensions of neurons - of a class of retinal ganglion cell is used to compute the directional movement of light.

“Our work over the last ten years has shown that dendrites provide neurons with powerful information processing capabilities, however the function of dendritic processing in the real-time

operation of neuronal networks has remained elusive,” Associate Professor Williams said.

“We used multi-site electrophysiological recording techniques to demonstrate that active dendritic integration underlies the computation of direction selectivity in rabbit retinal ganglion cells.”

To gain greater insight, the group measured electrical activity from multiple sites in retinal ganglion cells when visual stimuli moved through space.

“The retina is an ideal system to investigate the role of active dendritic integration in neuronal circuit function, because this network can be maintained intact in a dish and retains its responsiveness to natural stimuli,” he said.

Whilst it has long been known that the retinal network extracts and signals specific aspects of visual stimuli, the new work is the first to discover how such responses are computed.

“We found that retinal ganglion cells compute the direction of light stimuli through exquisitely controlled local integration compartments in

the dendritic tree, a finding which highlights the key function that dendrites play in brain computations,” Associate Professor Williams said.

Discovering how nerve cells process information is fundamental to understanding how we learn, and to developing new strategies to enhance learning in education and in disease processes in the brain.

The paper was published in the prestigious journal *Nature Neuroscience*.

Above: Light-evoked dendritic spike generation in a direction selective retinal ganglion cell.
Far left: Associate Professor Stephen Williams.

"The evidence suggests that the biological clock ticks for men and not just for women"

Older grandfathers pass on autism risk through generations



Men who have children at older ages are more likely to have grandchildren with autism compared to younger grandfathers, according to a joint international study.

The study, a collaboration between Professor John McGrath and researchers from King's College London's Institute of Psychiatry (IoP) and Karolinska Institute in Sweden, shows for the first time how risk factors for autism with older fathers may accumulate and be passed down to the grandchildren.

The researchers used Swedish national registers, and identified 5,936 individuals with autism and 30,923 healthy controls born in Sweden since 1932.

"We had complete data on each individual's parents' and grandfathers' age of reproduction," Professor McGrath said.

The study found men who had a child when they were 50 years or older had about a 70 per cent increased risk of having a grandchild with autism compared to men who had children when they were 20-24.

Professor McGrath said the evidence suggests that the biological clock ticks for men and not just for women.

"Autism is caused by a combination of genetic and environmental factors," Professor McGrath said.

"Previous studies have shown that older paternal age is a risk factor for autism in children: fathers aged 50 or older have a more than double the risk of having a child diagnosed with autism compared to younger fathers.

"The mechanism behind this link is unknown, but may be explained by mutations occurring in the male sperm cells.

"Sperm cells divide over time, and on each division there is an increased risk of new mutations being introduced."

Professor McGrath said this Swedish study suggested that some of these new mutations could skip a generation.

"The new findings suggest that these 'silent' mutations are passed on to the otherwise healthy child, but may influence the risk of future generations developing autism."

Dr Avi Reichenberg, from IoP and co-author of the paper, said the research provided evidence on how fathers' and grandfathers' lifestyle choices affected the following generations.

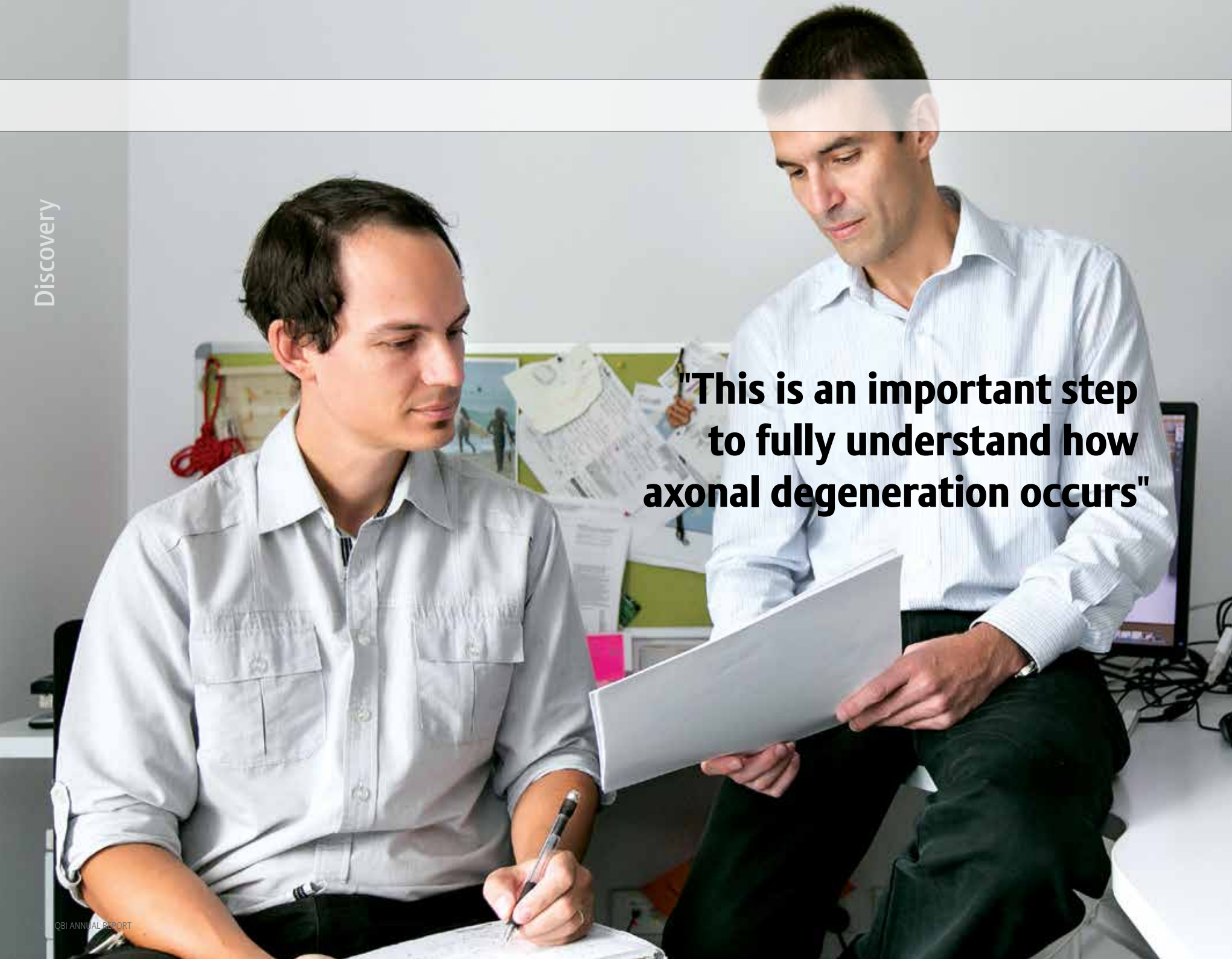
"This doesn't mean that you shouldn't have children if your grandfather was old when he had your father or mother, because while the risk is increased, it is still small," Dr Reichenberg said.

"However, the findings are important in understanding the complex way in which autism develops."

Recent reports have also suggested that the prevalence of autism spectrum disorder may be increasing.

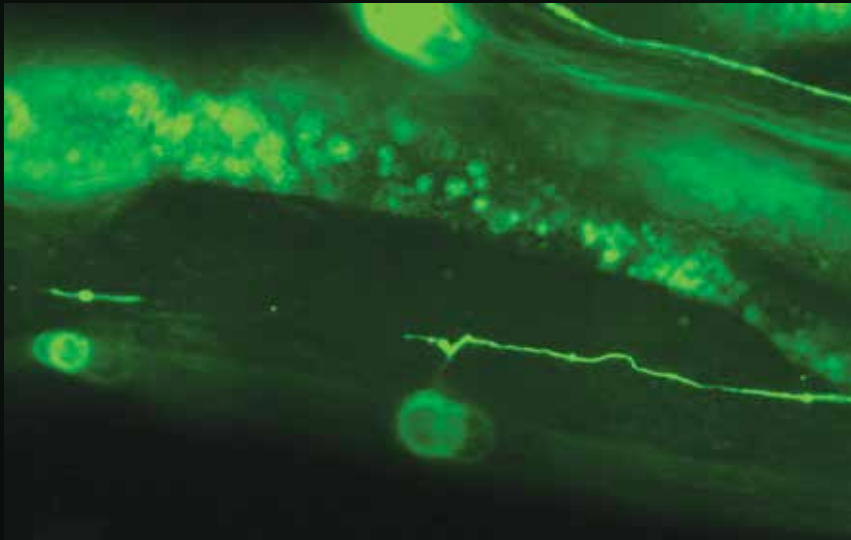
In Australia, one child in 160 is diagnosed with autism and it was four times more common in boys than girls.

Above: Professor John McGrath. Far left: Henry Simila, McGrath laboratory.

A photograph of two men in a laboratory or office environment. The man on the left, wearing a light blue button-down shirt, is seated and writing in a notebook with a pen. The man on the right, wearing a light blue striped button-down shirt, is standing and holding a large sheet of paper, looking at it intently. In the background, there is a bulletin board with various papers and a computer monitor on a desk.

**"This is an important step
to fully understand how
axonal degeneration occurs"**

Research into axon degeneration



A significant discovery at QBI could one day halt a number of neurodegenerative diseases, with the identification of a gene that protects against spontaneous, adult-onset, progressive nerve degeneration.

Dr Massimo Hilliard said that the discovery of gene *mec-17* causing axon (nerve fibre) degeneration could open the door to better understand the mechanisms of neuronal injury and neurodegenerative diseases characterised by axonal pathology, such as motor neuron disease, Parkinson's, Alzheimer's and Huntington's diseases.

"This is an important step to fully understand how axonal degeneration occurs, and thus facilitates development of therapies to prevent or halt this damaging biological event," Dr Hilliard said.

Dr Hilliard's laboratory specialises in neuronal development, and focusses on how nerves both degenerate and regenerate.

The team found that the protein MEC-17 protects the neuron by stabilising its cytoskeletal structure, allowing proper transport of essential molecules and organelles, including mitochondria, throughout the axon.

This discovery also has the potential to accelerate the identification of human neurodegenerative conditions caused by mutations in genes similar to *mec-17*.

"It's our hope that this could one day lead to more effective treatments for patients suffering from conditions causing neuronal degeneration," Dr Hilliard said.

This discovery highlights the neuron's longest process, the axon, as a major focal point for the health of the neuron.

Findings of the research have been published in journal *Cell Reports*, and lead author Dr Brent Neumann anticipates that the research into the gene will soon lead to further discoveries.

"This study demonstrates that *mec-17* normally functions to protect the nervous system from damage," Dr Neumann said.

"This knowledge can now be used to understand precisely how the gene achieves this and to discover other molecules that are used by the nervous system for similar protective functions," he said.

"We can now start to look into means of bypassing the function of *mec-17*, such as activating other genes or alternative mechanisms that can protect the nervous system from damage."

Previous research has shown that *mec-17* is conserved across species, including humans, suggesting a possible shared function of protection.

"We identified *mec-17* from a genetic screening method aimed at identifying molecules that cause axonal degeneration when they become inactive through genetic mutations," Dr Neumann said.

This project was conducted in the tiny nematode *Caenorhabditis elegans*, which is a very powerful genetic model system commonly used for addressing and understanding neurobiology questions at a basic biological level.

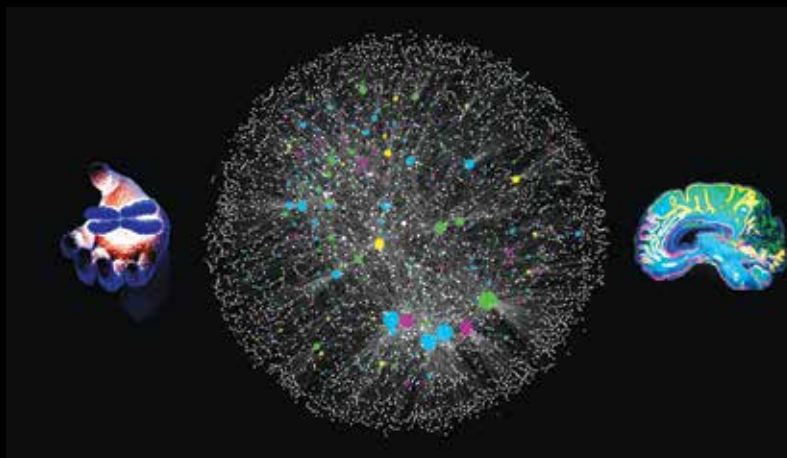
The project was funded by the National Health and Medical Research Council and an Australian Research Council Future Fellowship.

Above: Mutations in the *mec-17* gene cause neuronal fibres to break and degenerate. Far left: Dr Brent Neumann (L) and Dr Massimo Hilliard (R).



**"There are
overrepresented
patterns of gene
variations that
define differences
between disorders"**

Study reveals molecular networks of mental health disorders



Early diagnosis and intervention of neurodevelopmental and psychiatric disorders could be made possible after QBI scientists discovered the molecular networks that underpin autism, X-linked intellectual disability (XLID), attention deficit hyperactivity disorder (ADHD) and schizophrenia. This study represents a step forward in understanding the causal basis of these principally genetic disorders.

Associate Professor Claudianos and his team used complex network concepts and computational methods to investigate whether hundreds of genes associated with these conditions were related through mechanisms of gene regulation and functional protein-protein interactions.

Many studies have identified genes associated with these mental health disorders, albeit this is often done in a piecemeal fashion with little regard to the inherent molecular complexity.

The international research team led by QBI scientists sought to abstract the detailed molecular relationships of disorder genes to understand the impact of DNA variations on biological pathways and process at the whole genome level.

The discovery of a large gene network comprised of over 4,000 genes was used to successfully predict association of genetic screening data with each disorder. The study shows that although genetic variations converge in some functional pathways, including how nerve cells connect (synapses), there are overrepresented patterns of gene variations that define differences between disorders.

The research paves the way for a 'gene network model' that can potentially be used to analyse the many DNA variations detected from genomic

screening of families with a neurodevelopment disorder and help in the early diagnosis and clinical intervention for one to two per cent of children with autism. Identifying the unique pathogenetic profile for individuals affected by a disorder will also greatly facilitate the accurate application of therapeutics.

The molecular network approach AXAS™ is now being used by the Autism Cooperative Research Centre Ltd (Australia) in collaboration with the Sick Kids Hospital Toronto Canada to analyse genome sequence information from 6,500 families with autism.

Above: Schematic representation of the AXAS™ network comprised of over 4,000 genes that identified molecular pathways and processes overrepresented in four different neurodevelopmental and neuropsychiatric disorders: autism (blue), XLID (magenta), ADHD (yellow) and schizophrenia (green). This gene network model provides a means to associate genetic screening data with brain disorders. Far left: Associate Professor Charles Claudianos.





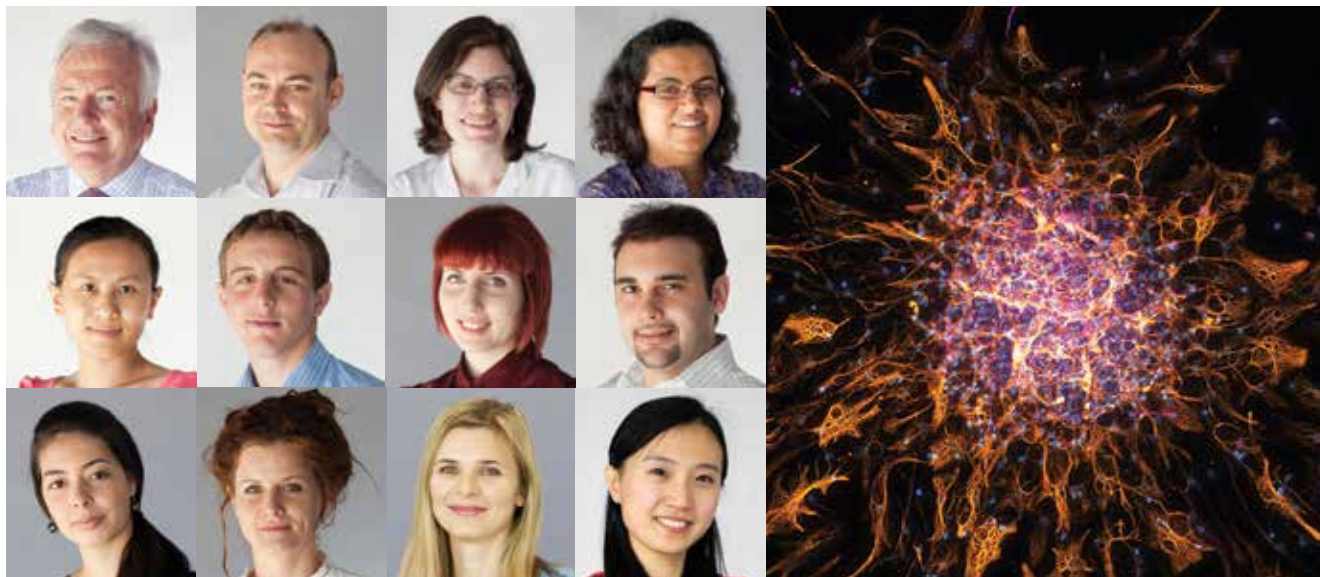
Beth Kita, Goodhill laboratory.

Research

QBI is a world-leading research facility whose staff are committed to discovering the fundamental mechanisms regulating brain function.

QBI's research provides the opportunity to address the overwhelming tide of neurological disease and mental ill health in the community.

Laboratory Head Professor Perry Bartlett



2013 Laboratory Members L-R: Perry Bartlett, Daniel Blackmore, Lavinia Codd, Dhanisha Jhaveri, Jing Lu, Cornel Mirciov, Estella Newcombe, Gregory Robinson, Sophie Tajouri, Chanel Taylor, Jana Vukovic, Jing Zhao. *Not pictured:* Weichuan Mo (based in China), Imogen O'Keeffe. **Background:** A differentiated neurosphere, grown from a single stem cell, showing glial cells (orange), neurons (red) and cell nuclei (blue).

Neurogenic regulation of cognition

Professor Perry Bartlett's laboratory is dedicated to understanding the mechanisms that drive the continuous production of new neurons in a region of the adult brain known as the hippocampus. This process, called neurogenesis, slows as we age, and this loss of neurons has been associated with a loss of cognitive function.

In a study published in *The Journal of Neuroscience* in 2013, the Bartlett laboratory demonstrated the importance of immature adult-born neurons to the learning process. By developing a mouse model in which they could specifically delete these immature neurons,

without affecting the rest of the cell population, they were able to show that loss of these cells resulted in a failure of the mice to perform well in a hippocampal-dependent spatial learning task compared to normal animals. Once these cells were allowed to regenerate, the mice performed as well as they had done prior to the immature neurons being ablated. Interestingly, the group also demonstrated that though important for memory, these immature neurons were not necessary for recall of a familiar spatial task. This work is important for the future development of means to stimulate neurogenesis to alleviate learning deficits. The Bartlett laboratory is

now focussed on identifying the factors that can trigger activation and production of these newborn neurons.

Also in 2013, the Bartlett laboratory continued their research into the treatment of spinal cord injury. In work published in *The Journal of Neurotrauma*, the group demonstrated that blocking the activity of the EphA4 receptor using the competitive antagonist, EphA4-Fc, in a rat model of contusive spinal cord injury significantly improved recovery of motor function after injury.

EMX1-positive cells (green) in the CA3 region of the mouse hippocampus.

The image is a composite. On the left is a large, textured, abstract shape in shades of blue and green, resembling a biological specimen or a microscopic view. On the right is a grid of seven headshots of researchers, arranged in two rows: four in the top row and three in the bottom row. The researchers are of various ethnicities and are smiling or looking directly at the camera.

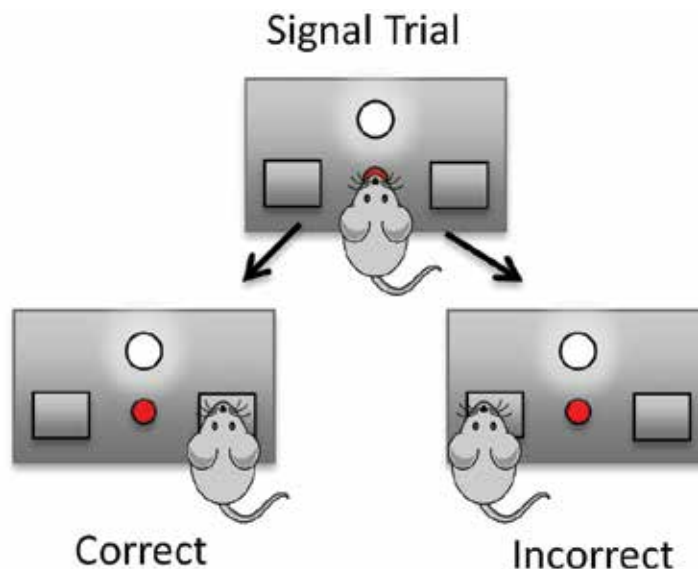
Epigenetic mechanisms regulating memory

lifespan. The group is particularly interested in how epigenetic mechanisms, including DNA methylation, histone modifications and the activity of non-coding RNAs regulate the formation and maintenance of memory.

2013 was a productive year for the laboratory, which published two important review articles on DNA methylation and neural plasticity in the journals *Trends in Neurosciences* and *Neurobiology of Learning and Memory*. In other work, which appeared in the journal *Learning and Memory*, the group demonstrated that the gene encoding BDNF is differentially methylated in the male and female cortex and that targeting

BDNF signalling can overcome sex differences in fear-related learning. These findings have significant implications for the development of novel therapeutic approaches for the treatment of fear-related anxiety disorders. Finally, recent work on a novel DNA modification, 5-hydroxymethylcytosine, and a new method for genome-wide profiling of this epigenetic mark have received positive reviews with invitations to present their work at the Society for Neuroscience annual meeting as well as the Society for Biological Psychiatry and the American College of Neuropsychopharmacology meetings.

Laboratory Head Associate Professor Thomas Burne



2013 Laboratory Members L-R: Thomas Burne, Suzy Alexander, Natalie Groves, Lachlan Harris, Pauline Ko, Emilia Lefevre, Aung Aung Moe, Chris Simpson, Karly Turner. *Not pictured:* James Peak, Michelle Sanchez Vega. **Background:** Translational research is being improved by developing a novel perceptual decision-making task for use in animal models based on the cognitive symptoms of schizophrenia. Here the rodent learns the rule about the stimuli and receptacle pairing, responding to the correct side to receive a food reward.

Behaviour and brain function in animal models of neuropsychiatric disorders

Associate Professor Thomas Burne's group studies brain development and behaviour in animal models. The group is focussed on investigating the underlying biological basis for schizophrenia, with the goal of finding public health interventions that will alleviate the burden of this disease. The group has been exploring the impact of developmental vitamin D deficiency on brain development, the impact of adult vitamin D deficiency on brain function and behaviour and, more recently, establishing novel ways to assess cognitive behaviour in rodents.

In 2013, the Burne group, in collaboration with Associate Professor Darryl Eyles and Professor John McGrath, built on its previous

research showing that low prenatal vitamin D (the 'sunshine hormone') is associated with alterations in behaviour, brain neurochemistry and receptor profile associated with vitamin D deficiency in animal models. Ongoing National Health and Medical Research Council funding allows the group to dissect the exact neural pathways involved in cognitive impairments of attentional processing in developmentally vitamin D deficient rats to model the cognitive symptoms of schizophrenia. The Burne group has now shown that low vitamin D levels during adulthood affects the balance of excitatory and inhibitory neurotransmitters in the brain, as well as altering cognitive behaviour in rodents. These

results provide the first evidence in mice to show that adult vitamin D deficiency impacts on neurotransmitter systems that are affected in a number of neuropsychiatric conditions, including autism, schizophrenia and depression.

The team is also trying to create and validate a unique cognitive task for rodents that mirrors the continuous performance task in humans. While there are a number of cognitive tests currently in use, the results are difficult to translate. The group's goal is to provide a novel tool for cognitive research in rodents and to uncover more about the pathophysiology and drug treatment of cognitive symptoms in schizophrenia.

Hippocampal neurogenesis is maintained throughout life. How adult vitamin D deficiency impacts on this process is being explored.

Laboratory Head Dr Allen Cheung

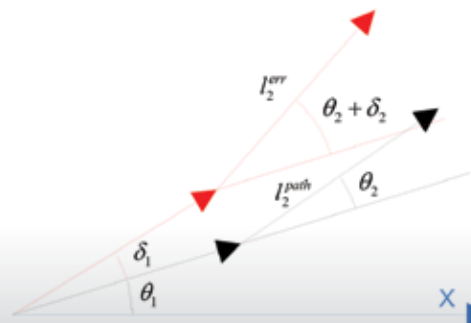
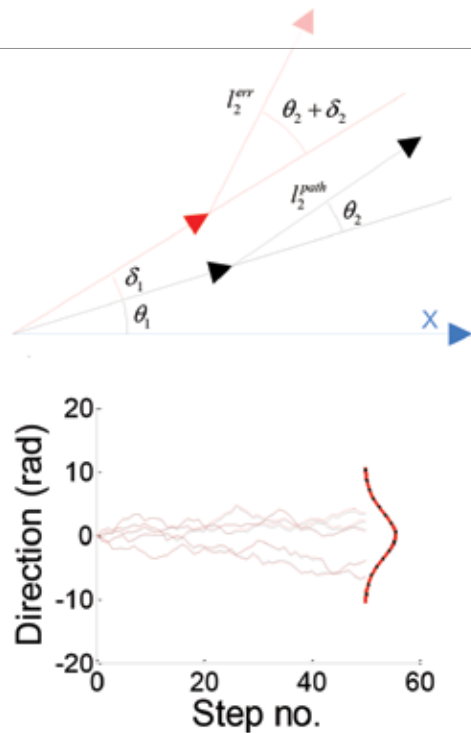
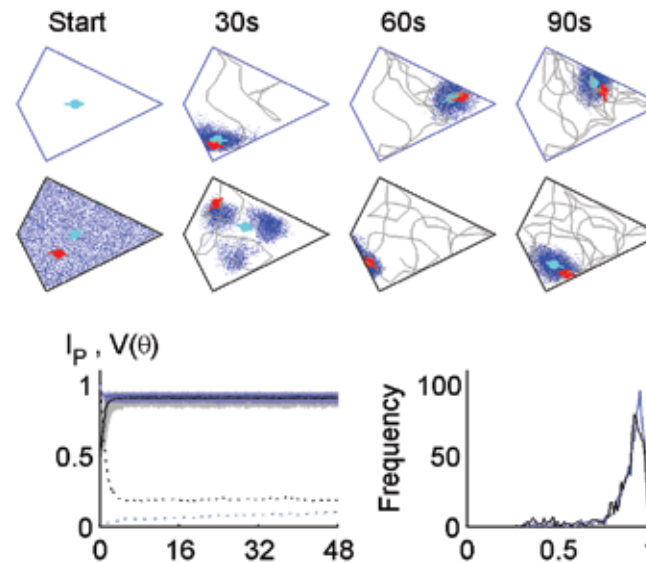


Illustration of path tortuosity and positional estimation errors.



2013 Laboratory Members top-bottom: Allen Cheung, Ashvin Srinivasan. Not pictured: Matthew Kennett, Zoltán Kósci. Background: Idiopathic localisation in bounded spaces.



Understanding the brain computations needed for spatial navigation

The core research of Dr Cheung's laboratory is aimed at understanding the fundamental brain computations required for spatial navigation. Spatial navigation is one of the oldest and most widespread brain functions in the animal kingdom. The cells, circuits and computations required for animals to search for resources, return home, and go back to those resources later, are subjects of intense research worldwide.

Path integration is one strategy used by vertebrates and invertebrates alike, and may be the common 'scaffold' required for spatial navigation. It is the process whereby estimated self-motion is integrated over time to yield an approximate vector between the starting location and current location. This form of

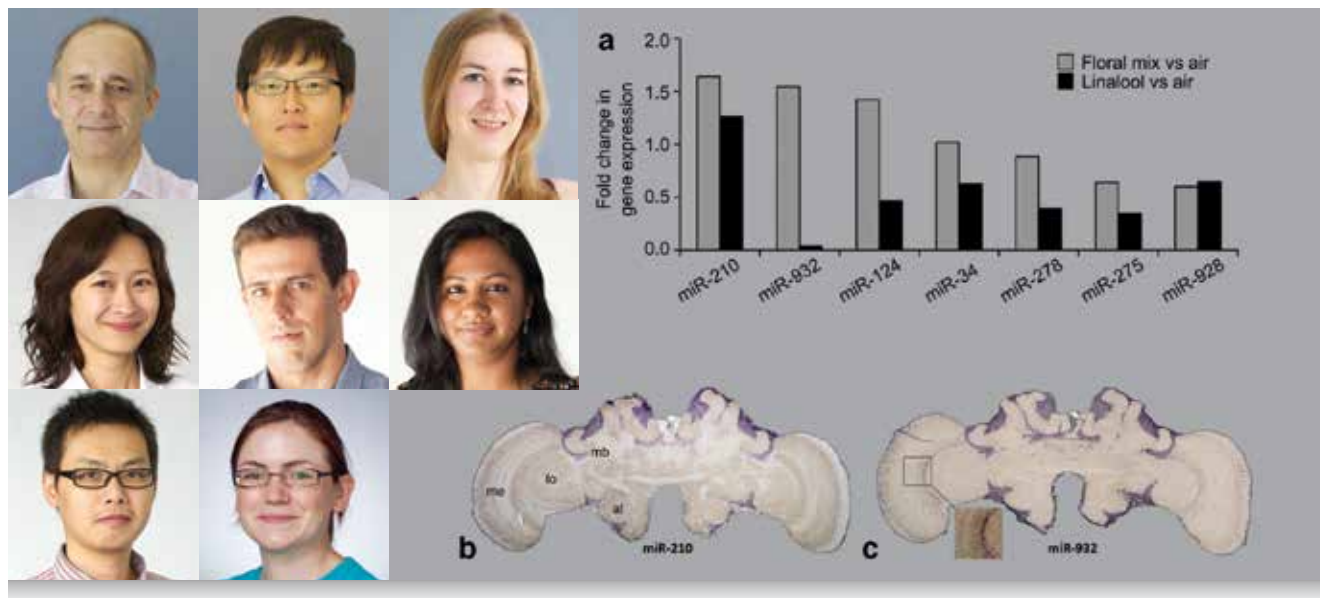
navigation is prone to noise, which leads to errors in navigation.

It was previously unknown how cumulative errors affect uncertainty during path integration along the types of tortuous paths that foraging animals take. Recent mathematical modelling work from the Cheung laboratory showed that path tortuosity has a surprising effect on positional uncertainty – the more tortuous a path, the less uncertainty. The effect was pronounced when animals only used internal cues. These results show that the path taken by an animal could significantly change its navigation performance, with implications for the design and interpretation of experiments.

In a recent collaboration with Professor Pankaj Sah's laboratory, *in vivo* recordings were performed in the brains of freely moving rats. A digital wireless system was used to record the spikes from individual neurons in the rat's hippocampus while it foraged in a large outdoor arena. Recently, place fields have been identified using this system. Exciting opportunities and challenges lie ahead to develop the experimental and theoretical tools to study the rodent brain in larger, more naturalistic environments.

Research from the Cheung laboratory was published in the *Journal of Theoretical Biology* in 2013.

Laboratory Head Associate Professor Charles Claudianos



2013 Laboratory Members L-R: Charles Claudianos, Joon An, Stephanie Biergens, Ming-yu Chen, Alexandre Cristino, Nivetha Gunasekaran, Shao-chang Huang, Aoife Larkin. *Not pictured:* Flavia Freitas, Sarah Williams. **Background:** MicroRNAs found to be upregulated (a) during olfactory learning and memory in the honeybee showing localisation of miR-932 and miR-210 is associated with higher brain centres (b & c).

Senses and synapses

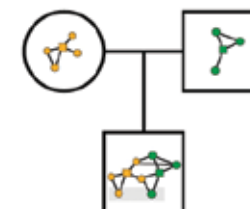
The Claudianos laboratory uses a 'genome to phenome' approach, whereby genome sequence information is used to abstract a molecular basis for mental health disorder. The biological relevance of human genetic variations is examined using simple model species such as *Drosophila* and the honeybee. The goal is to unravel how polygenic DNA variations that often occur in synaptic molecules contribute to physiological and behavioural deficits that characterise disorders like autism.

A major aim of the laboratory is to characterise key molecular processes involved in neurodevelopment and synapse development. Many data now show that a loss of synapses or aberrant synaptic

connection between neurons affect brain function. Molecules involved in synapse development such as neuroligin and neuroligin head a list of causative molecular associations in the pathogenesis of autism spectrum disorders (ASDs). The group use systems biology and complex network approaches to abstract a molecular basis for human mental health disorders that provides a 'hypothesis' to assess genetic screening data from affected individuals. They can then functionally characterise these molecules in the laboratory using small animal models.

In 2013, the Claudianos group showed neurodevelopmental and neuropsychiatric disorders such as autism, X-linked intellectual disability,

attention deficit hyperactivity disorder and schizophrenia are underpinned by an interconnected molecular network of causal genes (*Molecular Psychiatry*). Together with collaborators from the University of Western Australia and the Telethon Institute, the laboratory completed the first whole genome sequence (exome) analysis of Australian autism families. The project was part of a successful Cooperative Research Centre application for 'Living with Autism Spectrum Disorders' that received \$32 million from the Australian Federal Government, which helped establish Autism CRC Ltd. The Claudianos laboratory leads the genetic screening projects associated with this national initiative.

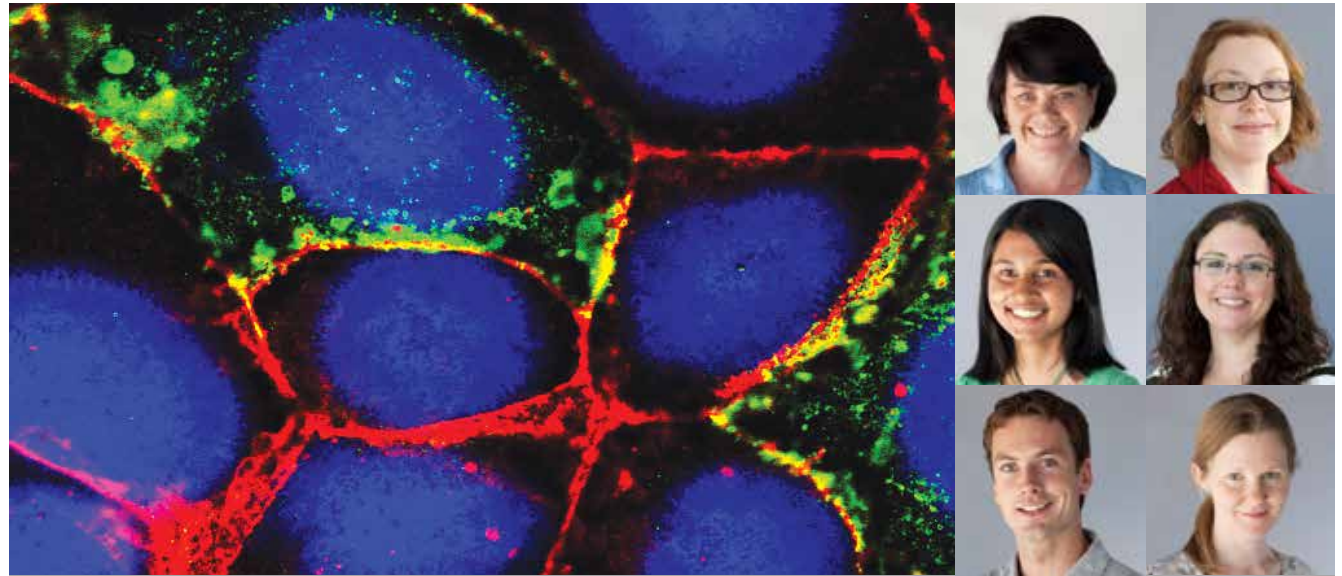


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Networks of causal DNA variations detected from whole genome (exome) sequencing of individual autism families that cluster in pathways associated with neurodevelopment.

Laboratory Head **Associate Professor Helen Cooper**

2013 Laboratory Members L-R: Helen Cooper, Charlotte Clark, Jayani Hewage, Natalie Lee, Conor O'Leary, Amanda White. *Not pictured:* Michael Langford. **Background:** The birth of individual neuron populations in the embryonic mouse cortex. Neogenin (green) is localised to the junctions (red) between cells.

Molecular mechanisms regulating new neurons in the brain

Cortical malformations are responsible for up to 40 per cent of drug-resistant childhood epilepsies and often cause mild to severe mental retardation. The aetiology of these syndromes is linked to the disruption of neural stem cell function and the failure to adopt the correct neuronal identity within the embryonic cortex. Identification of the aberrant mechanisms responsible for these devastating malformations will reveal fundamental insights into the pathophysiology of cortical malformations at both the molecular and cellular levels.

The Cooper laboratory has discovered that mutations in the cell surface receptor, Neogenin, severely disrupt the structure of the embryonic

stem cell niche, resulting in the formation of cortical malformations. Strikingly, these phenotypes closely parallel those seen in humans, thereby implicating Neogenin in the aetiology of human cortical malformations. Recent experiments suggest that Neogenin is part of the trafficking machinery that replenishes important cell surface proteins that anchor neural stem cells to their local environment. Disruption of this stem cell architecture leads to a failure in neuron production. Studies in the adult neural stem cell niche have further revealed that this important receptor also regulates the birth of new neurons in the adult brain by controlling the ability of stem cells to progress through the

cell cycle. Therefore, Neogenin is a key regulator of neurogenesis in both the embryo and adult.

The Cooper laboratory has also identified a second receptor, the Wnt receptor, Ryk, as a key control point in the decision of newborn cortical neurons to adopt the correct identity – an essential requirement for the development of a fully integrated and functioning neocortex. They have uncovered evidence that Ryk mutations lead to neuron mis-specification and inappropriate positioning within the developing cortex. Similar disruptions in humans have profound consequences for neocortical function and have been linked to schizophrenia, epilepsy and mental retardation.

The spectrum of colours reveals the diversity of pyramidal neuron subtypes in the developing mouse neocortex.

Laboratory Head Associate Professor Elizabeth Coulson



2013 Laboratory Members L-R: Elizabeth Coulson, Fabienne Alfonsi, Earlene Ashton, Zoran Boskovic, Georg Kerbler, Dusan Matusica, Linda May, Nika Mohannak, Nick Palstra, Lei Qian, Bree Rumballe, Aanchal Sharma, Sune Skeldal, Toni Turnbull. *Not pictured:* Mirela Wagner. **Background:** Virtual maze.

Measuring neuronal loss in Alzheimer's disease through navigation task

The Coulson laboratory is investigating why certain neurons die in Alzheimer's disease (AD) and how that affects cognition. Work focusses on the p75 neurotrophin receptor and its role in neuronal loss. By blocking signalling through this receptor, nerve cell degeneration in the basal forebrain in an animal model could be stopped.

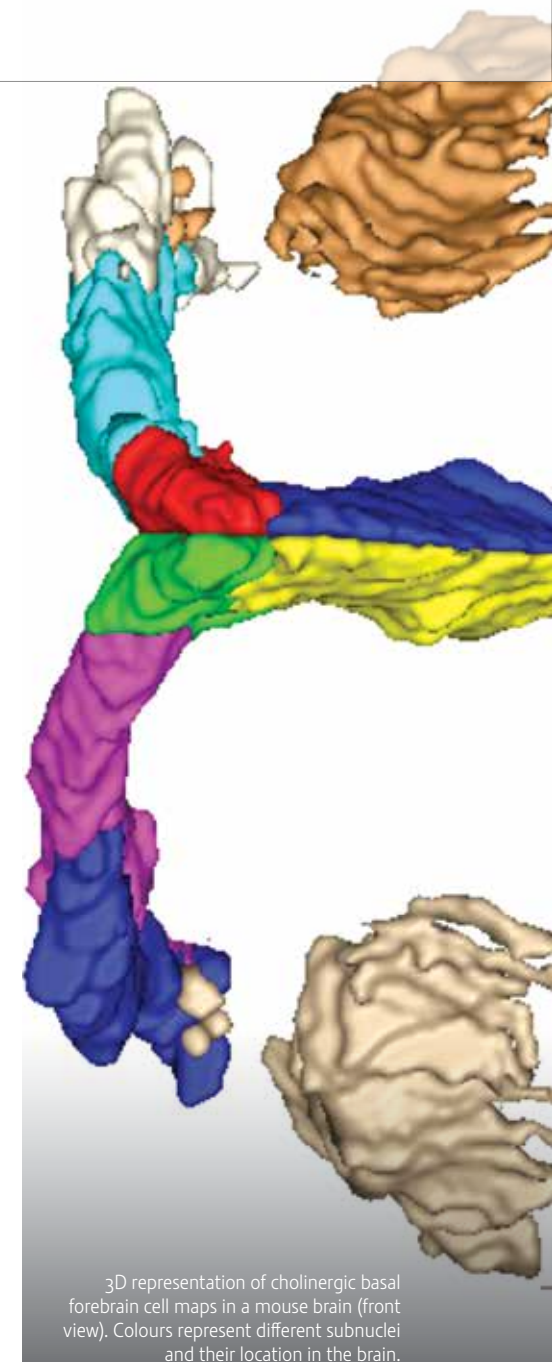
The basal forebrain is important for learning and memory and is known to be selectively lost early in AD. The only class of drugs currently given to AD patients and shown to be effective at slowing cognitive decline in clinical trials increases the levels of the neurotransmitter released by the basal forebrain neurons. However significant

loss of these neurons has already occurred in the majority of AD patients prior to treatment and because these drugs are only efficacious while the neurons are alive, it is not surprising the treatment is of limited value.

The Coulson group asked if basal forebrain function, assessed by cognitive testing, could be used as an earlier indicator of dementia. Using mouse models they discovered that the basal forebrain is important for spatial processing used in a certain type of navigation sometimes referred to as dead reckoning. These findings are of particular significance as poor navigation is a cognitive change observed in humans with mild

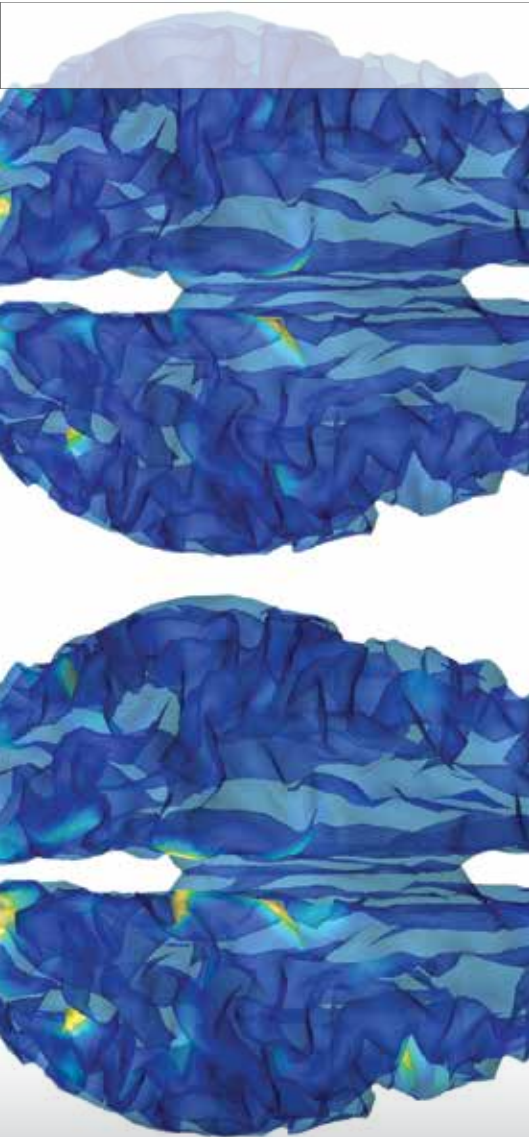
cognitive impairment, the prelude to AD.

The Coulson laboratory also teamed-up with collaborators Drs Hort and Lazco at Charles University in the Czech Republic, who had developed dead reckoning-style spatial memory tests for humans. Joint studies have revealed that this test might be useful in assessing the efficacy of current AD treatments or novel treatments which, like blocking P75 neurotrophin receptor cell death signalling, aim to stop degeneration of the basal forebrain neurons and restore the cognitive deficits that characterises dementia.

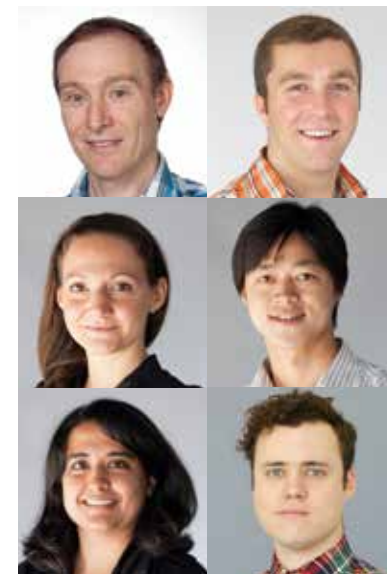
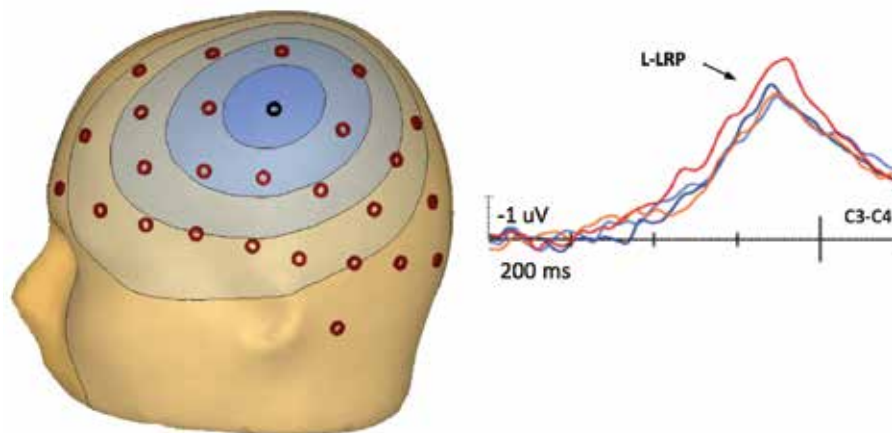


3D representation of cholinergic basal forebrain cell maps in a mouse brain (front view). Colours represent different subnuclei and their location in the brain.

Laboratory Head Associate Professor Ross Cunnington



Activity of the brain generates very small magnetic fields that can be measured with a technique known as magnetoencephalography (MEG). This shows the very early neural responses in the visual areas at the back of the brain when we see hand gesture actions, important for understanding others' gestures.



2013 Laboratory Members L-R: Ross Cunnington, Jeff Bednark, Veronika Halász, Vinh Nguyen, Simmy Poonian, Chase Sherwell. *Not pictured:* Megan Campbell, Samuel Fynes-Clinton, Jessica McFadyen, Jessica Ogden, Kelsey Palghat, Eva-Maria Reuter, Elysia Sokolenko, Michelle Steffens, Özlem Yetim. **Background:** When planning voluntary actions, neural activity increases in the motor cortex of the brain before movement initiation. This shows motor cortex activity increasing 200 ms before movement - that's about the fastest possible reaction time.

Brain processes for human voluntary action

The Cunnington laboratory focusses on the brain processes involved in perceiving and understanding the actions of others, as well as planning and preparing for our own voluntary actions.

Brain circuits involving the motor cortex and deep-brain regions known as the basal ganglia are crucial for the voluntary control of everyday actions and for learning movement skills. The Cunnington group is using high-resolution brain imaging with functional MRI (fMRI) to examine how different parts of this brain circuitry are important for the learning and control of complex movements. This is important for understanding movement disorders such as Par-

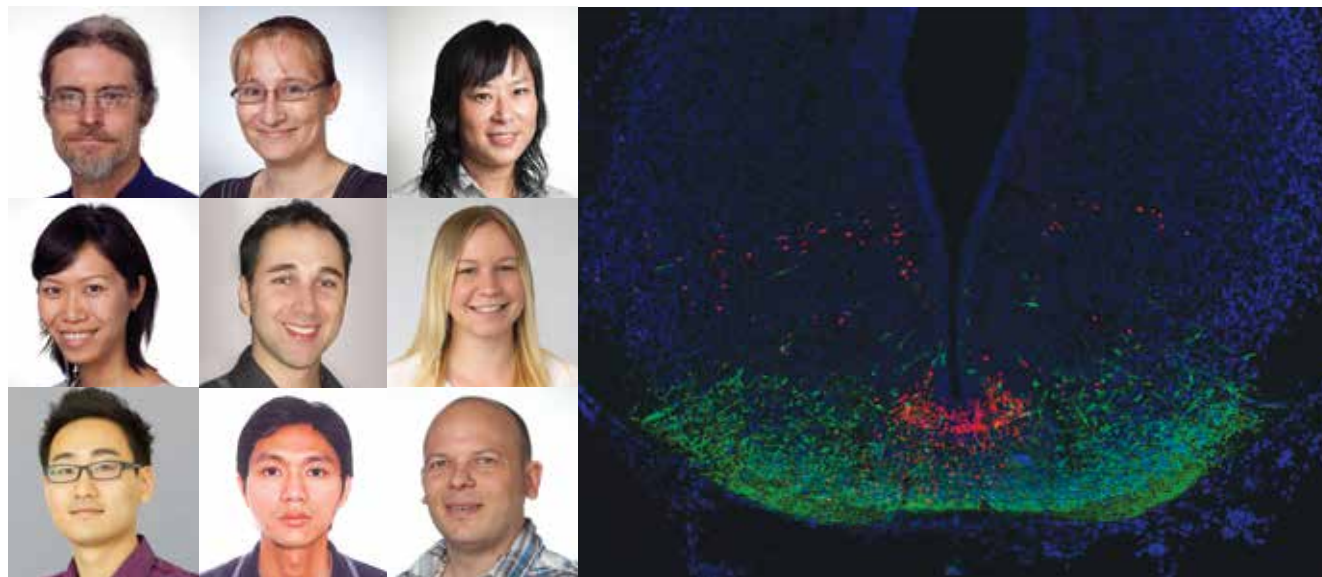
kinson's disease in which basal ganglia function is impaired.

The group is also combining fMRI with direct measurement of brain activity using electroencephalography (EEG) to model the dynamics of brain function during action planning. Research has revealed how another deep region of the brain known as the cingulate cortex is crucial as a "hub" region and drives activity in the motor circuits of the brain as people plan and prepare for voluntary actions.

Research from the laboratory also examines how these same motor circuits of the brain

are important for our ability to perceive and understand others' actions through "mirroring" processes. Whenever we observe another person's actions, gestures or emotions, their states appear to be "mirrored" in our own brain, leading us to understand their actions, intentions and emotions through a process of simulation or mirroring. Controversially, this neural empathy appears to be stronger when we see people of our own race express emotion than when seeing people of a different race express the same emotion. The Cunnington group is examining how this bias in neural empathy or mirroring changes with learning and familiarity with other races.

Laboratory Head Associate Professor Darryl Eyles



2013 Laboratory Members L-R: Darryl Eyles, Suzy Alexander, Xiaoying Cui, Pauline Ko, David Kvaskoff, Emilia Lefevre, Leon Luan, Aung Aung Moe, Henry Simila. *Not Pictured:* Lachlan Ferguson, Verena Landel. **Background:** Fluorescence microscopy image of a coronal section of the embryonic rodent midbrain showing the location of potential dopaminergic progenitor neurons (Nurr 1, red) and mature dopaminergic neurons [tyrosine hydroxylase (TH), green].

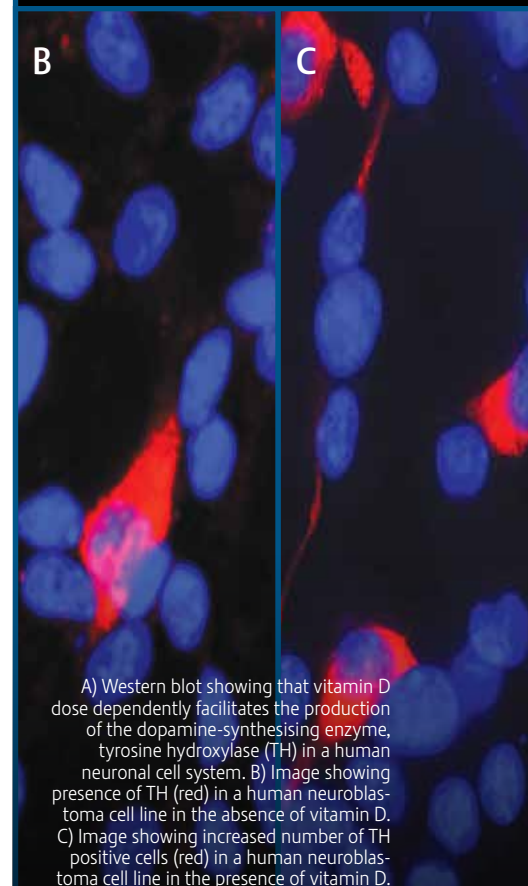
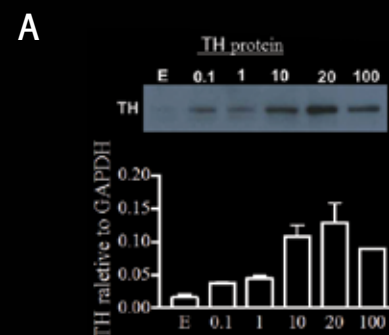
Vitamin D and brain development

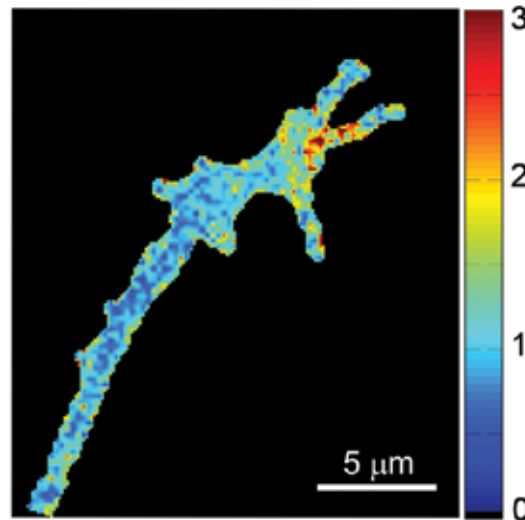
The Eyles laboratory focusses on how risk-factors for schizophrenia, such as developmental vitamin D (DVD) deficiency, change brain development. The group has developed an extremely sensitive LC/MS/MS assay for vitamin D species in blood spot cards. This assay allowed the 2010 landmark study implicating low maternal levels of vitamin D as a risk factor for schizophrenia to be conducted. The group is now examining the relationship between DVD-deficiency and autism. Wide-spread international interest led to discussions with the National Institute of Standards and Technologies in Washington regarding the production of blood spot standard reference material to use this assay in paediatric settings internationally.

Schizophrenia is closely associated with abnormalities in dopamine transmission. The group's work in DVD-deficient animals confirms there are early abnormalities in dopamine development and turnover, and its work in human cell systems describes the direct control vitamin D exerts over dopamine production via the vitamin D receptor.

The group fast-tracks discoveries regarding abnormal dopamine ontogeny in rodent models into other model systems, including the fruit fly and zebrafish. Collectively, the work represents a synthesis of the two major theories of schizophrenia, the "dopamine hypothesis" and the "neurodevelopmental hypothesis" into the "dopamine ontogeny hypothesis of schizophrenia".

For 14 years the Eyles group has explored the role of vitamin D in the developing brain and how DVD-deficiency may affect brain function and behaviour in adult offspring. With National Health and Medical Research Council funding success in 2013, the group intends to expand the scope of its existing animal model in two critical ways. Firstly, the group will examine the effect of varying the duration and level of DVD-deficiency duration on brain development and function. Secondly, the group will examine whether abnormalities in the ontogeny of dopamine systems observed in DVD-deficient animals are shared by other prominent animal models of this disease.



Laboratory Head **Professor Geoffrey Goodhill**

2013 Laboratory Members L-R: Geoffrey Goodhill, Lilach Avitan, Kelsey Chalmers, Richard Faville, Nicholas Hughes, Elizabeth Kita, Huyen Nguyen, Zac Pujic, Biao Sun, Daniel Sutherland. *Not Pictured:* Brendan Bicknell. **Background:** Calcium distribution inside a growing nerve fibre.

Computational, systems and developmental neuroscience

Professor Goodhill's laboratory is interested in how brains process information, particularly during development. This includes how growing nerve fibres (axons) use molecular cues to make guidance decisions, how map-like representations of visual inputs form in the optic tectum and visual cortex, and how these maps code sensory information. The laboratory is addressing these questions using a combination of experimental, mathematical and computational techniques. Members of the group come from diverse backgrounds, including biology, mathematics, physics and computer science.

One area of focus for the laboratory is how nerve fibres are guided by molecular gradients to find appropriate targets in the developing nervous

system. The laboratory recently developed a theoretical model to understand quantitatively how levels of calcium and cAMP in axons determine whether they are attracted or repelled by guidance cues. The model made surprising predictions that the group confirmed experimentally. This may help to explain the behaviour of developing and regenerating axons *in vivo*.

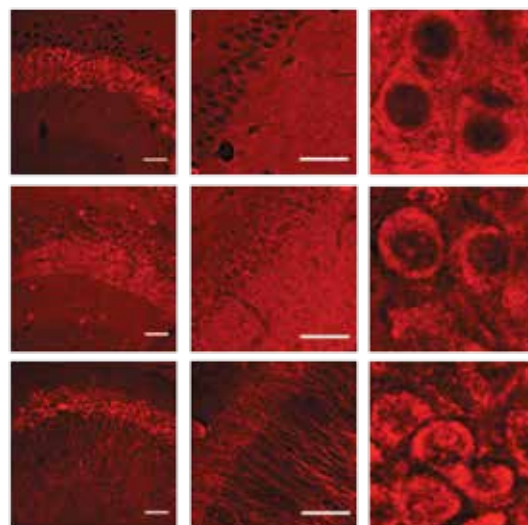
The laboratory has also investigated the shape of growth cones, the structures at the tip of developing axons. This morphology is complex and highly dynamic but the significance of these changes for either the sensory or motor roles of growth cones is mostly unknown. Sophisticated mathematical techniques for characterising shape in general have been adapted to develop a more quantita-

tive understanding of the role growth cone shape plays in effective axon guidance.

The group is also using fluorescent labelling techniques to visualise the simultaneous activity of many neurons in the developing zebrafish brain in response to simple visual stimuli. By using mathematical techniques from statistics and information theory, it is then possible to predict how the zebrafish could optimally decode these patterns of activity in order to determine what visual stimulus was actually present. A better understanding of neural decoding is important for optimising the design of brain-computer interfaces.

Receptive fields of visual neurons predicted from a mathematical model.

Laboratory Head Professor Jürgen Götz



2013 Laboratory Members L-R: Jürgen Götz, Sian Baker, J Bertran-Gonzalez, Xia Di, Harrison Evans, Hon Mun Lee, Gerhard Leinenga, Jing Lu, Miriam Matamalas, Rebecca Nisbet, Tishila Palliyaguru, Linda Wernbacher. *Not pictured:* Nadia Cummins, Julia Gutmann, Henry Harding, Chuanzhou (Joe) Li, Juan-Carlos Polanco, Zoe Wood. **Background:** Analysis of murine brain sections with antibodies raised against specific tau isoforms (oN, 1N and 2N) reveals a distinct subcellular staining pattern.

Mechanisms of neurodegeneration and therapies

At present there is no cure for Alzheimer's disease and other forms of dementia. This poses an unprecedented social and economic challenge to Australia, a country with the second highest life expectancy worldwide.

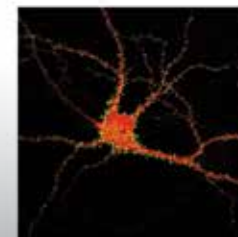
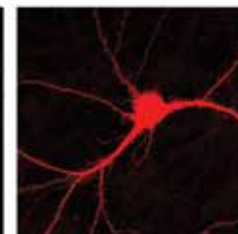
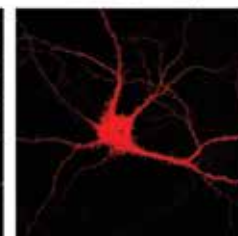
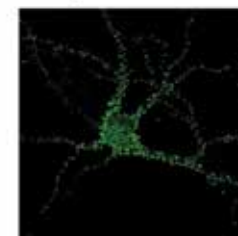
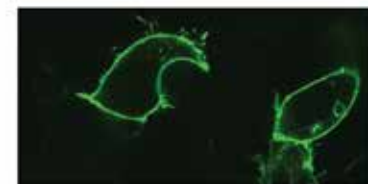
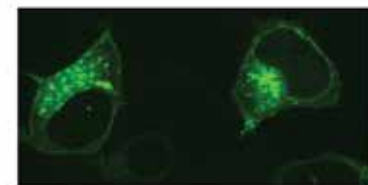
In the Götz laboratory, which forms part of the Clem Jones Centre for Ageing Dementia Research, there are four major streams of research: (1) understanding pathogenic mechanisms of key players in dementia, such as the microtubule-associated protein tau or the kinase Fyn; (2) understanding the physiological role of proteins implicated in disease; (3) developing novel methods to visualise *de novo* protein synthesis and of delivering drugs to the brain; and (4)

developing therapies for treatment.

Following on from the discovery that tau causes a pathological elongation of mitochondria (*Neuron*, 2012), Professor Götz and colleagues provided an integrated view of their findings in the journal *Trends in Neurosciences* (2013). In work published in the journal *Neurobiology of Aging* (2013), Professor Götz and his colleagues at the University of Cologne showed that the tyrosine kinase Pyk2 becomes activated in neurons with tau pathology. In a signalling cascade, Pyk2 activates the serine/threonine kinase GSK3 to phosphorylate tau at pathological epitopes. Interestingly, the gene encoding Pyk2 is one of the recently identified novel Alzheimer's disease risk genes.

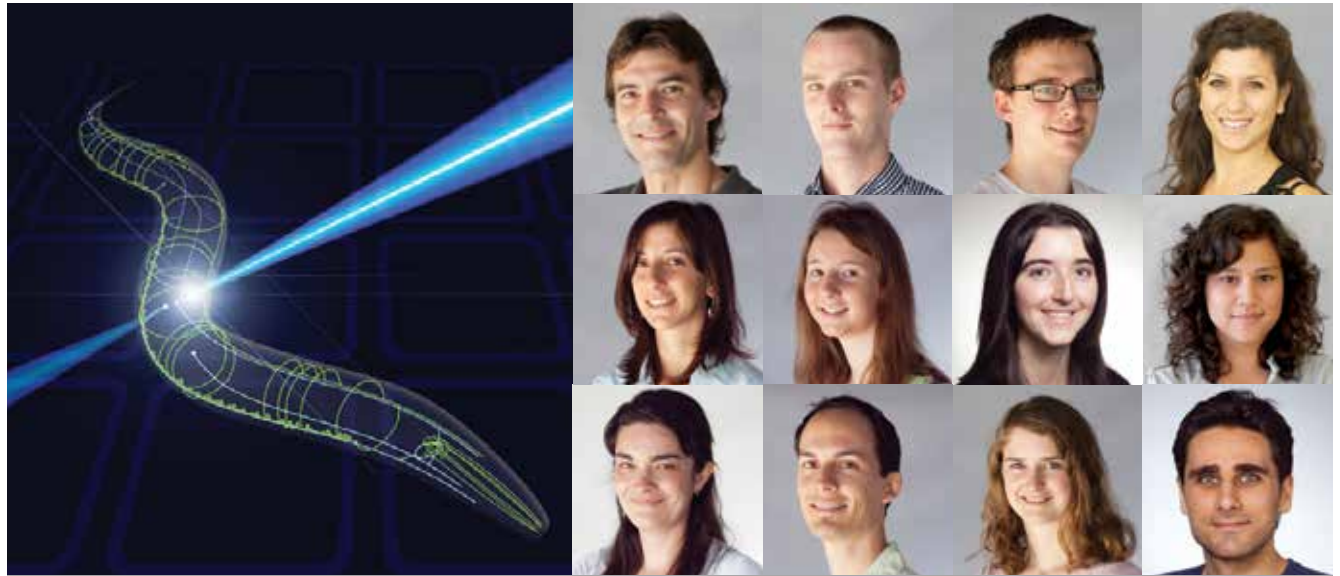
The group also succeeded in establishing the gene editing method TALEN in the laboratory. Furthermore, they established a novel protocol, using ultrasound and microbubbles, which enables them to open the blood-brain barrier transiently, allowing drugs of a defined size to enter the mouse brain.

A highlight of 2013 was the 7th Alzheimer's + Parkinson's Disease Symposium that was organised by Professor Götz, and was this year held at QBI. With 175 registered delegates and international speakers from the USA, Germany and China, the conference was hailed as a success.



Palmitoylation, and therefore membrane association, is required for the localisation of Fyn into dendritic spines.

Laboratory Head Dr Massimo Hilliard



2013 Laboratory Members L-R: Massimo Hilliard, Justin Chaplin, Sean Coakley, Alessandra Donato, Rosina Giordano, Rhianna Knable, Casey Linton, Ellen Meelkop, Paula Mugno, Brent Neumann, Annika Nichols, Nicholas Valmas. *Not pictured:* Laura Frederiksen, Joseph Yunis. **Background:** *C. elegans* being targeted by a light stimulus to induce neuronal inactivation. Inactivation of selected neurons *in vivo* can define their contribution to specific developmental outcomes, circuit functions, and behaviours. The optogenetic tool KillerRed can selectively, rapidly, and permanently inactivate different classes of neurons. Williams et al., *Cell Reports*, 2013 (Image: Nick Valmas, Dee McGrath and Sean Coakley.)

Axonal development, regeneration, and degeneration

The Hilliard laboratory is interested in understanding how axons (nerve fibres conducting impulses from the neuron) develop and are guided to their targets. The group investigates how axonal structure is maintained over time, and how it can be repaired after injury.

Neurons are highly polarised cells with neurites, dendrites and an axon, forming distinct morphological and functional domains. How a neuron decides on the number of neurites to extend is not well understood. Using *Caenorhabditis elegans* mechanosensory neurons as a model system, the Hilliard group has discovered MEC-7/ β -tubulin, a component of the microtubules, to have a critical role in this process. These results support the emerging evidence that microtubules provide

critical signals for axon formation (*Molecular Biology of the Cell*, 2013).

The axon is the neuron's longest process, but the mechanisms that allow it to maintain its structural integrity, or facilitate repair following injury, are still poorly understood. Reactive oxygen species (ROS) are major neuronal damaging components generated in a number of neurodegenerative conditions. In a collaborative project, the Hilliard group has developed an approach to generate ROS in selective classes of neurons, making it possible to determine the molecular mechanisms underlying ROS-mediated degeneration (*Cell Reports*, 2013a).

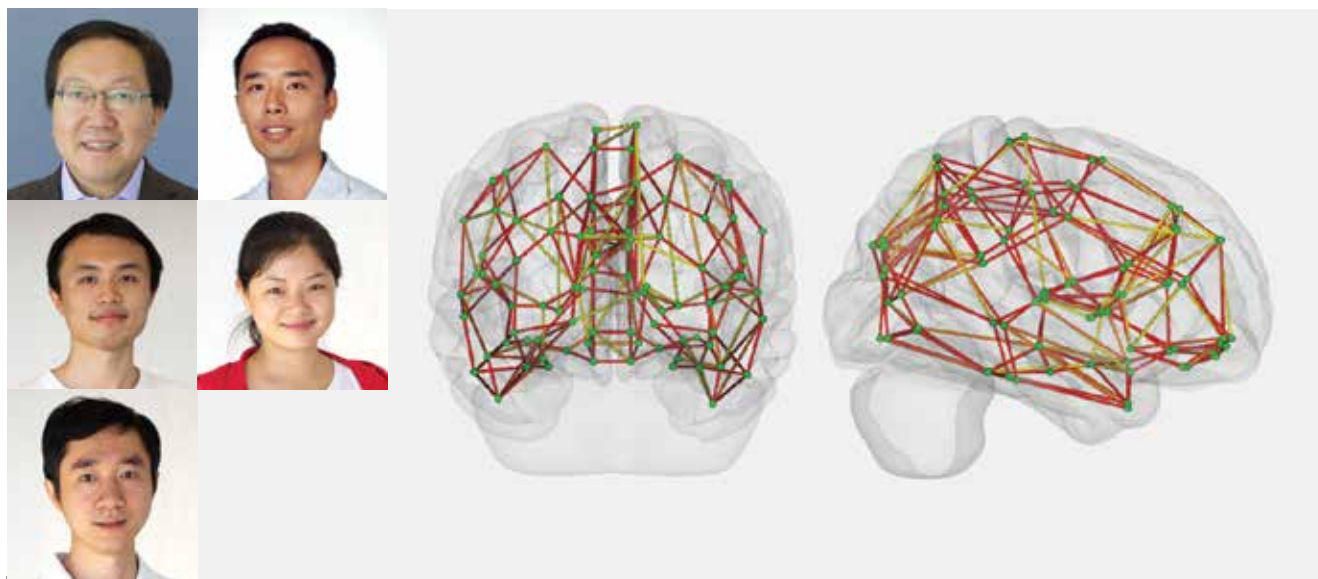
More recently, the team has uncovered an axonal

protective function for MEC-17, an alpha-tubulin acetyltransferase, which acts by stabilising the cytoskeleton allowing proper transport of molecules and organelles throughout the axon (*Cell Reports*, 2013b).

Using laser-based technology to axotomise single neurons in *C. elegans*, the Hilliard group has characterised neuronal regeneration in different classes of sensory neurons. In earlier work they demonstrated that axonal regeneration can occur as a result of axonal fusion, when two separated axonal fragments re-attach and restore the original axonal tract (*Developmental Dynamics*, 2011). In 2013, they identified some of the molecules that regulate this process.

A *Caenorhabditis elegans* mechanosensory neuron expressing GFP undergoes axonal degeneration due to a mutation in the gene *mec-17*, which has an axonal protective effect. Neumann & Hilliard, *Cell Reports*, 2013. (Image: Brent Neumann).

Laboratory Head Professor Tianzi Jiang



2013 Laboratory Members L-R: Tianzi Jiang, Yonghui Li, Cirong Liu, Tong Wu, Xianfeng Yang. **Background:** 3D presentation of binary connective matrixes; adapted from Li et al., *Cerebral Cortex*, 2013.

Mapping human and animal brain networks with neuroimaging

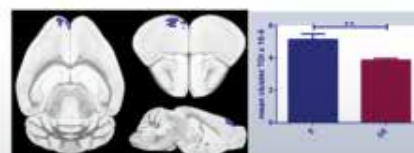
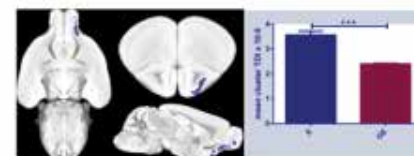
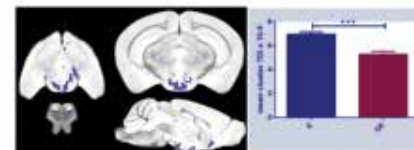
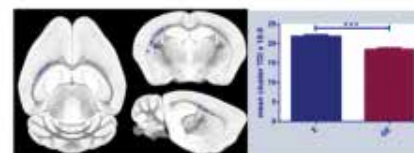
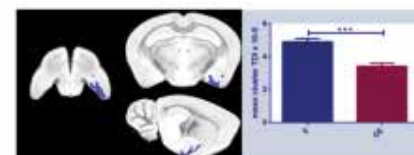
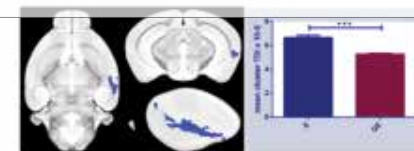
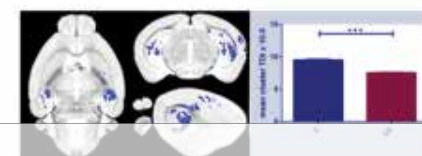
Convergent evidence has shown that brain functions can manifest on brain networks on different scales, and that the brain malfunctions associated with most psychiatric disorders are the result of faulty brain networks.

The “brainnetome” (www.brainnetome.org) is an emerging avenue to integrate the multi-level network features obtained with various functional and anatomical brain imaging technologies on different scales.

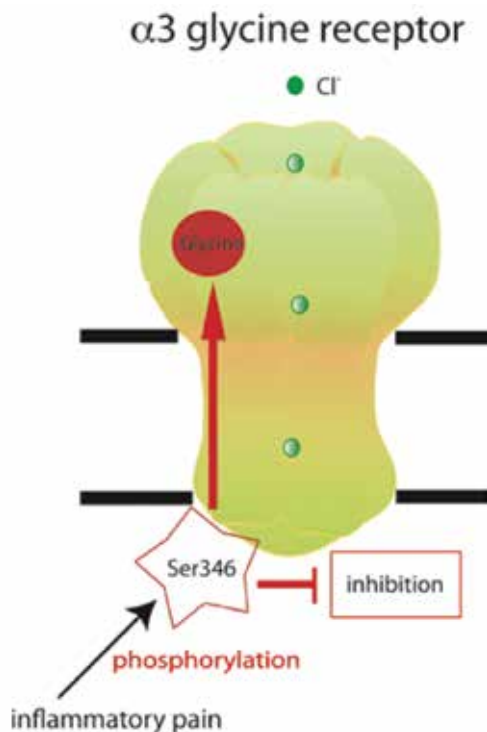
The Jiang laboratory is studying basic theory, methodologies and algorithms underpinning the brainnetome platform, and their applications in neurological and psychiatric diseases. In

2013, their research mainly involved the mouse brainnetome, focussing on the Disrupted-In-Schizophrenia-1 (*DISC1*) gene. *DISC1* plays critical roles in neurodevelopment by regulating neurogenesis, migration, and dendrite/spine density via its interaction with many other proteins. However, the roles of the *DISC1* in white matter development, oligodendrocyte differentiation and myelination are unclear, despite the fact that *DISC1* is a promising risk gene for many mental illnesses associated with white matter abnormalities and disconnection syndromes. By sending the same animals through behavioural and neuroimaging examinations, the Jiang laboratory have shown that significant abnor-

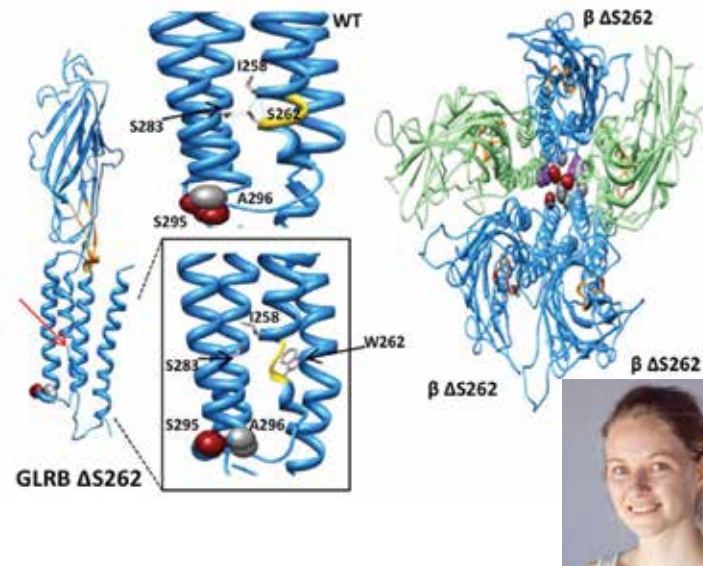
malities can be detected in one of their *DISC1* transgenic mouse strains, in which human mutant *DISC1* is expressed under the control of the prion protein promoter (Prp-DN-*DISC1*). The group observed abnormal behavioural performances in the transgenic animals by open-field, Y-maze and fear-conditioning tests and abnormal brain structures were found to be associated with the behavioural phenotypes. Most importantly, the abnormalities were only observed in *DISC1* transgenic mice that went through social isolation during adolescence postnatal day 35 to 56, suggesting the “Gene & Environment” interaction may underlie a variety of neuropsychiatric disorders such as schizophrenia.



Results of track-density analysis based on diffusion MRI in isolated groups (wild-type versus *DISC1* mice). In each subfigure, the left shows clusters that have a significant difference ($P < 0.005$, uncorrected, cluster size $> 0.1 \text{ mm}^3$), while the right shows the average track-density of each cluster after being inverse-transformed back into the native space.

Laboratory Head **Professor Joe Lynch**

A cartoon showing how pain-induced phosphorylation of glycine receptors produces a conformational change in the glycine binding site.



2013 Laboratory Members L-R: Joe Lynch, Anna Bode, Christine Dixon, Justine Haddrill, Angelo Keramidas, Han Lu, Suzanne Scott, Ming Shiuan, Sahil Talwar, Azra Zamri. Not Pictured: Sharifun Islam, Robi Islam, Yan Zhang. **Background:** A molecular model of a glycine receptor showing how hyperekplexia mutations affect their structure and function.

Targeting inhibitory neurotransmitter receptors in neurological disorders

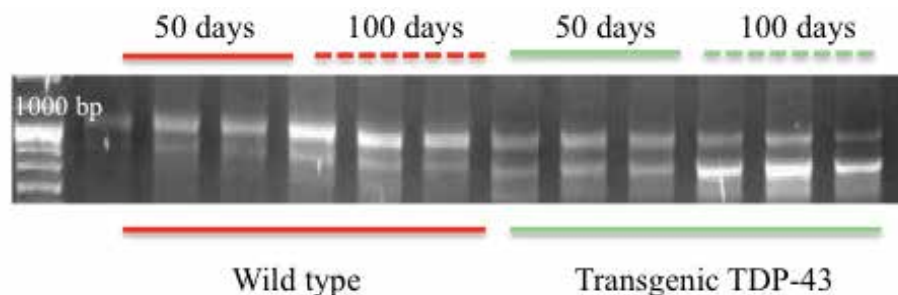
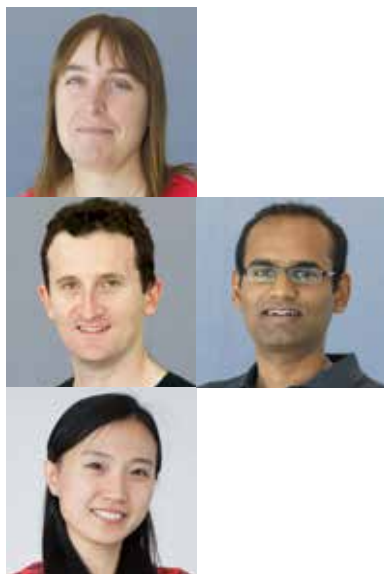
The Lynch laboratory's major research interest concerns the molecular structure and function of the glycine and GABA_A receptor chloride channels that mediate inhibitory neurotransmission in the brain. The GABA_A receptor is an important therapeutic target for sedative and anxiolytic drugs and the glycine receptor has recently emerged as a therapeutic target for pain, spasticity, epilepsy and tinnitus. The Lynch laboratory is discovering new drugs active at these receptors and the molecular mechanisms by which their structures and functions are disrupted in hereditary neurological disorders.

Chronic inflammatory pain is caused by the prostaglandin-induced phosphorylation of a type of glycine receptor found in pain sensory neurons in the spinal cord. These 'pain-modulated' or phosphorylated receptors represent a promising therapeutic target for chronic pain, but the problem has always been to prevent the drugs from affecting normal phosphorylated glycine receptors. The laboratory recently showed that the phosphorylation event that underlies this pain produces a specific change in structure of a drug binding site on this receptor. These results raise the possibility of designing new analgesic drugs that specifically target only

those glycine receptors that are modulated by pain. The laboratory is currently attempting to design such drugs.

Hyperekplexia (or startle disease) is a rare human neurological disorder that produces an exaggerated startle in response to unexpected auditory or tactile stimuli. In collaboration with geneticists at the University of Swansea, the laboratory has recently shown that the glycine receptor beta subunit is an important new protein targeted by hereditary startle disease mutations.

Laboratory Head Dr Marie Mangelsdorf



2013 Laboratory Members L-R: Marie Mangelsdorf, Tim Butler, Ramesh Narayanan, Jing Zhao. *Not pictured:* He Ji. **Background:** TDP-43 regulates many aspects of gene regulation, including alternative splicing. Alternative splicing is a normal process where several transcripts (isoforms) are produced from the same gene, which can encode different proteins with different cellular functions. The image shows alternative splicing that changes with disease progression in a mouse model of MND.

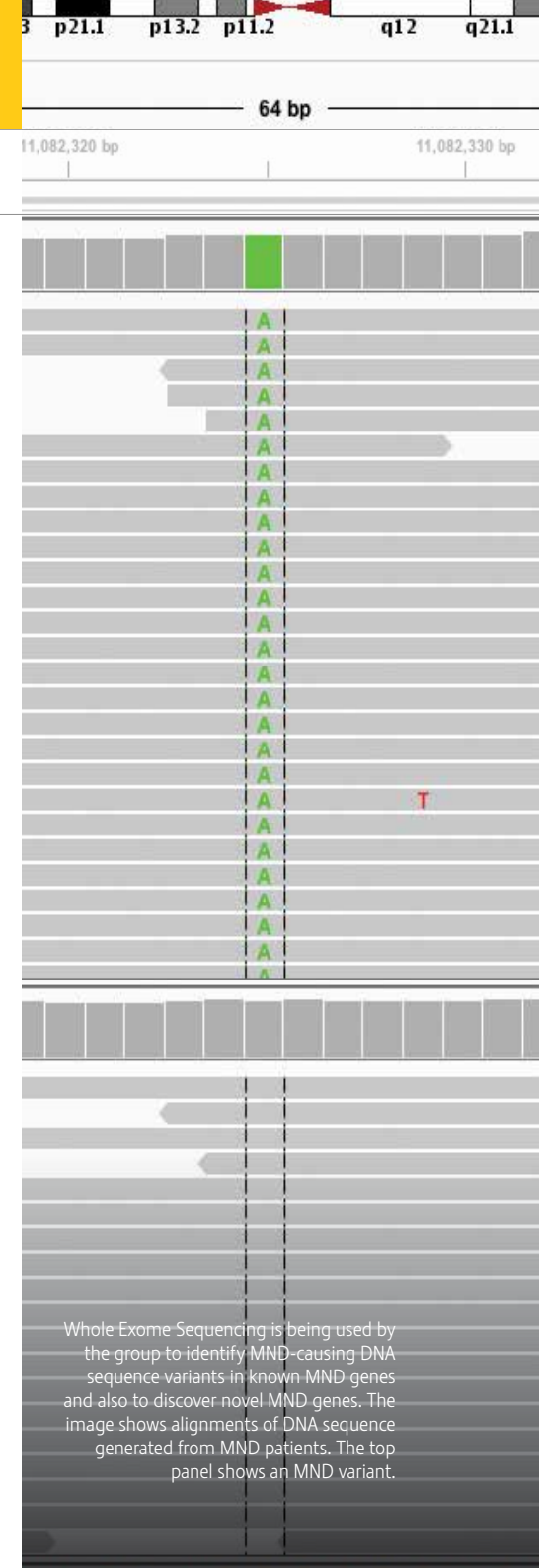
Genetics and molecular mechanisms of motor neuron disease

Genetic mutations have been found in familial forms of motor neuron disease (MND) and the same mutations can be found in non-familial cases. Currently ~60 per cent of familial cases can be explained by a genetic mutation, suggesting there are still more MND genes to be found. The group is using whole exome sequencing of a large cohort of MND patients from China to identify novel MND genes. This research is part of a large collaboration including Professors Perry Bartlett and Peter Visscher and Associate Professor Naomi Wray at QBI, as well as Professors Matt Brown (UQ's Diamantina

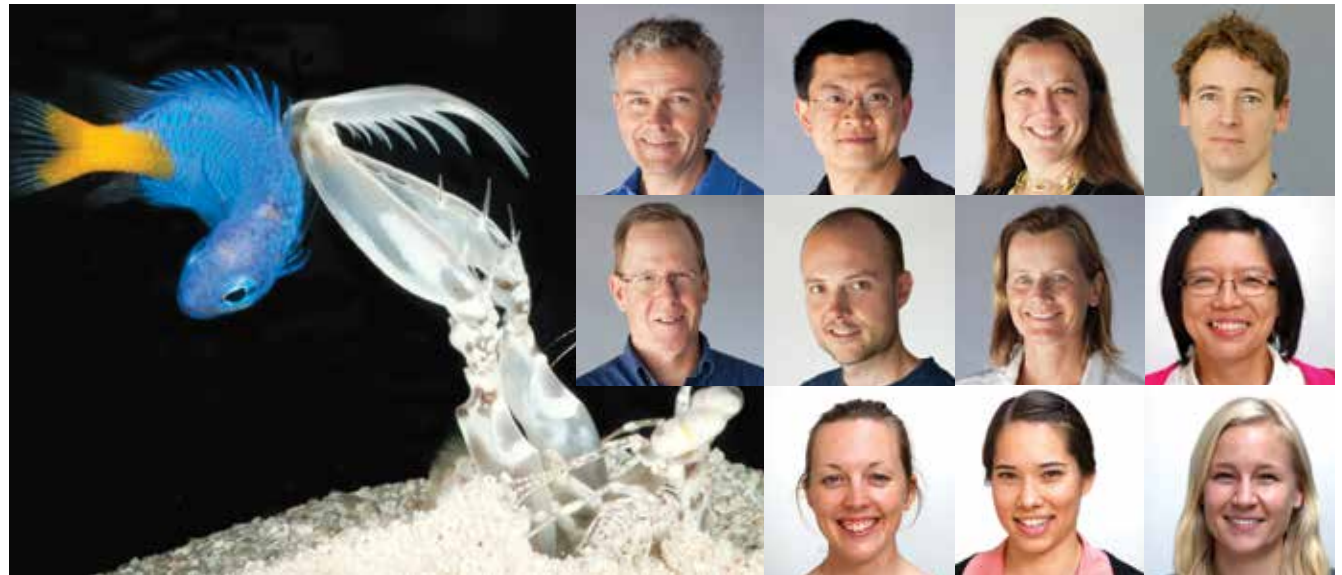
Institute), Huji Xu (Shanghai) and Dongsheng Fan (Beijing). The group is also sequencing a cohort of patients from Queensland to identify novel disease causing genes.

Several of the known MND genes encode proteins involved in gene regulation. The group is striving to understand the normal role of one of these proteins, namely TDP-43, in the central nervous system, and how mutation leads to disease. In work published in 2013, the group reported mRNAs to which TDP-43 binds and found it regulates genes involved in synaptic activity. The same year the group investigated

TDP-43 mediated pre-mRNA splicing in a mouse model of MND using exon microarrays. Genes have been identified that are misspliced in TDP-43 transgenic animals, prior to the onset of MND symptoms. These genes have the potential to become biomarkers of progression of MND in patients. The team is also collaborating with the Hilliard group to study TDP-43-mediated RNA transport in *Caenorhabditis elegans* neurons to determine the role of this cellular process in MND pathology.



Whole Exome Sequencing is being used by the group to identify MND-causing DNA sequence variants in known MND genes and also to discover novel MND genes. The image shows alignments of DNA sequence generated from MND patients. The top panel shows an MND variant.

Laboratory Head **Professor Justin Marshall**

2013 Laboratory Members L-R: Justin Marshall, Wen-Sung Chung, Angela Dean, Yakir Gagnon, Alan Goldizen, Martin How, Diana Kleine, Yi-Hsin Lee, Genevieve Phillips, Rachel Templin, Hanne Thoen. *Not pictured:* Karen Cheney, Fabio Cortes, Kyra Hay, Santi Krisantini, Qamar Schuyler, Anne Winters. *International members/trainees:* Simon Giggins, Sara Stieb, Margarete Uberfuhr. **Background:** The spearing mantis shrimp *Lysiosquilla sulcata* narrowly misses a brightly coloured damselfish. Both animals are studied for their visual capabilities. As the mantis shrimp strikes faster than any other marine creature, it is rare for fish to escape.

A systems approach to neuroscience, neuro-circuitry and information flow

As sensory neurobiologists, the Marshall laboratory investigates the information and circuitry that allows colour and polarisation information to flow from the outside world, through the eyes and into the brain. The end result is a decision based on the quality of light reflected from an object; a food item, member of the opposite sex or the flash of a potential predator.

The laboratory takes a multi-disciplinary approach. Research areas include the physics of light, colour and polarisation; the sensory cells in the eyes of invertebrates and vertebrates; neural connection and informational translation behind the eye

and into the brain; and the resulting behaviours from this information flow. Using molecular, anatomical, electrophysiological, physical and behavioural methods, the group aims to reveal the visual world of animals through the languages of colour and polarisation communication.

The Marshall laboratory works with non-standard model animal systems such as crustaceans, reef fish, cephalopods and turtles. The voracious predator, the mantis shrimp (stomatopod), is a favourite as its eye has the capacity for better colour and polarisation vision than any animal known so far. The group also investigates the beautiful world of colour communication,

camouflage, sex and death on the reef through the fish that live there.

This comparative approach, sometimes called Visual Ecology, yielded many exciting discoveries with over 20 publications in 2013, including work appearing in *Science*, *Current Biology*, *PNAS* and many media and public domain outlets. Talking to non-scientists is a priority, and collaborations with David Attenborough through Atlantic Productions, BBC, Discovery Channel NHK (Japan), along with publications for CoralWatch (the group's environmental section) and several mainstream magazines and newspapers showcase the group's work to the public.

Close up view of mantis shrimp compound eye. The six rows of enlarged facets in the mid-band area contain many specialised photoreceptors for colour and polarisation vision.

Laboratory Head Professor Jason Mattingley



2013 Laboratory Members L-R: Jason Mattingley, Oliver Baumann, Luca Cocchi, Daina Dickens, Eve Duperrix, Hannah Filmer, Marta Garrido, Oscar Jacoby, Marc Kamke, David Lloyd, Natasha Matthews, Claire Naughtin, David Painter, Martin Sale, Susan Travis, Lisa Wittenhagen. *Not pictured:* Michael Dwyer, Michelle Hall, William Harrison, Luke Hearne, Sang Hoon Jee, Abbey Nydam, Cooper Smout, Amy Taylor, Joe Wagner. **Background:** Yellow concentric circles, crosshairs and red spot help an experimenter guide a TMS coil into position on a human volunteer's head. The underlying grey area shows the brain's cortical surface as visualised with magnetic resonance imaging.

Brain oscillations reveal how we see

Researchers from the Mattingley group made several important discoveries in 2013 about how the human visual system works, in health and disease.

PhD student David Painter developed a novel method for recording stimulus-evoked oscillations in brain activity in human volunteers using electroencephalography (EEG). He had participants identify briefly flashed coloured targets in the centre of a cluttered display, while they ignored distracting stimuli in the periphery of their visual field. He discovered that the brain continues to respond to distracting information, but only if it shares a key feature with the target stimulus, such as its colour. Using a similar

experimental approach, David also recorded how the brain responds to visual information following localised damage caused by stroke, a discovery that has important implications for rehabilitation therapy.

In further work, Masters student Lisa Wittenhagen used EEG to measure how visual areas of the brain represent elements of a scene that are momentarily occluded from view, such as when we see a cyclist pass behind a tree. She developed a novel protocol to identify a unique activity signature in visual neurons, which arises when portions of an object are rendered invisible due to occlusion. This important discovery provides new insights into how we continue to

“see” in the absence of direct visual input.

Sometimes the brain produces visual experiences that have no basis in reality. Dr Michael Dwyer used EEG to characterise changes in the brains of individuals who experience visual hallucinations due to macular degeneration (an acquired loss of central vision due to damage to the light sensitive retina in the eye). He showed, for the first time, that visual neurons in these individuals are pathologically hyperactive. This discovery provides an important clue as to the mechanisms underlying visual hallucinations, and will help guide new treatments for this debilitating disorder.



A human volunteer receives transcranial magnetic stimulation (TMS) during an experiment on brain plasticity. The stimulating coil is guided into position on the scalp using a magnetic resonance image of the volunteer's own brain. Brief, high intensity magnetic pulses are delivered which activate neurons in the underlying cortex.

Laboratory Head **Professor John McGrath**

2013 Laboratory Members, top-bottom: John McGrath, Henry Simila. *Not pictured:* Peter Josh. **Background:** Vitamin D is known as the 'sunshine hormone'.

Modifiable risk factors for schizophrenia

The aim of the McGrath laboratory is to explore risk factors that are linked to schizophrenia. They focus on non-genetic factors that are potentially modifiable. In recent years the team has been examining the impact of low vitamin D (the 'sunshine hormone') during early brain development and on adult brain function. In collaboration with Associate Professors Darryl Eyles and Thomas Burne, they have developed animal models to examine the impact of low vitamin D during gestation on brain development.

To date, the group has clearly shown that low vitamin D during early life alters brain development in rodents. However, they are now exploring whether this is also relevant

to humans by investigating the association between vitamin D concentrations at birth and later risk of schizophrenia. Like folate and spina bifida, if vitamin D is linked to the risk of schizophrenia, then it offers the opportunity to use supplements to reduce the incidence of this disorder. The research team at QBI has pioneered this innovative hypothesis.

In 2013, Professor McGrath was awarded a prestigious National Health and Medical Research Council John Cade Fellowship in Mental Health Research. These funds will allow the research focus to include: (a) a wider range of modifiable risk factors (e.g. infectious agents, stress, cannabis, vitamin D), (b) a more diverse

range of brain related outcomes (e.g. prenatal and neonatal brain growth, childhood neuro-cognition, autism, schizophrenia, other mental disorders), (c) a wider range of epidemiological samples (in collaboration with national and international groups), (d) explore new ways to combine genetic and epidemiological clues to uncover the risk architecture of brain-related disorders (in collaboration with Professor Peter Visscher and Associate Professor Naomi Wray), and (e) build skills related to clinical trials in those with Early Psychosis (in collaboration with the UQ Centre for Clinical Research's Associate Professor James Scott).

Artwork by Glenn Brady.

Laboratory Head Associate Professor Frederic Meunier*



2013 Laboratory Members L-R: Frederic Meunier, Rachel Gormal, Callista Harper, Ravikiran Kasula, David Kvaskov, Regine Low, Nancy Malintan, Sally Martin, Nika Mohannak, Vinod Narayana, Tam Hong Nguyen, Shona Osborne, Andreas Papadopoulos, Vanesa Tomatis, Tong (Iris) Wang. *Not pictured:* Adekunle Bademosi, Shi Min (Priscilla) Goh. **Background:** Super-resolution microscopy enables the visualisation and tracking of lipid molecules on the plasma membrane. The movements of cholera toxin, which binds to the ganglioside GM1, are shown. Studying the diffusion of these individual molecules provides information on how the plasma membrane responds to stimuli. Image: Callista Harper.

The mechanism underpinning neuronal communication and survival

2013 was a year of many achievements for the Meunier laboratory, including the award of two grants, namely a National Health and Medical Research Council Project grant and an Australian Research Council LIEF grant to equip QBI with some of the most exciting new technologies in super resolution microscopy. In 2013, the laboratory published eight peer-reviewed publications and was asked to join the Clem Jones Centre for Ageing Dementia Research (CJCADR).

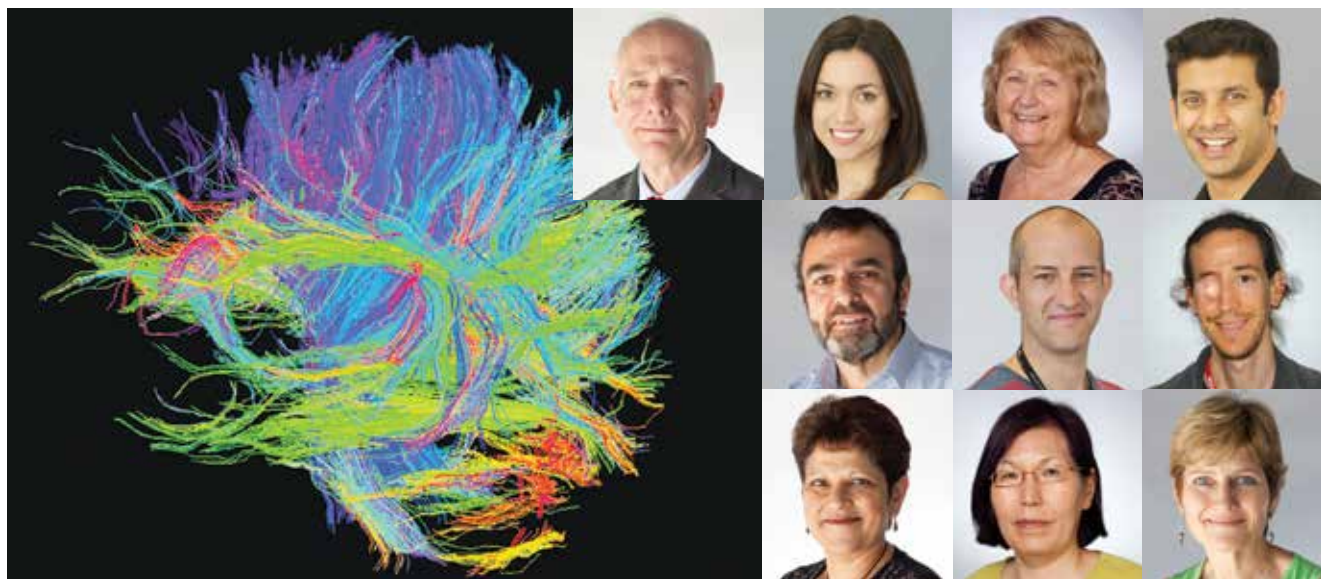
The team has continued to pursue its work into the mechanism of neuroexocytosis. This year the group published the discovery that Myosin VI plays a key role in capturing secretory vesicles on the cortical actin network in an activity-de-

pendent manner (*Journal of Cell Biology*). They also revealed that Munc18-1 not only controls the delivery of Syntaxin-1 to the plasma membrane but also regulates neuroexocytosis itself through a key role of its domain 3a (*Journal of Cell Science*). Additionally, they showed a novel type of plasticity allows filopodial extensions to create new release sites in response to secretagogue stimulation (*Journal of Neuroscience*).

In terms of vesicular trafficking, the Meunier laboratory demonstrated that pharmacological inhibition of PIKfyve activity leads to an apoptosis-independent neuronal cell death, implicating a dysregulation of autophagy. This pathway is thought to be involved in a number of neuro-

degenerative diseases, including Charcot-Marie Tooth and motor neuron disease (*PLOS ONE*). The group investigated the traffic of synthetic self-assembling clostridial chimera neurotoxin in neurons in collaboration with Professor Bazbek Davletov (MRC Laboratory of Molecular Biology, UK). They also reviewed the literature on the potential of blocking endocytic pathways to counteract pathogens internalisation (*Trends in Cell Biology*) and pursued their collaboration with Professor Phillip Robinson (University of Sydney) and Professor Adam McCluskey (University of Newcastle) on a range of highly effective dynamin inhibitors (*Traffic*).

Mobile autophagosomes in neuronal networks.

Laboratory Head **Professor Bryan Mowry**

2013 Laboratory Members L-R: Bryan Mowry, Ilvana Dzafic, Cheryl Filippich, Javed Fowdar, Bill Mantzioris, Andrew Martin, Samuel Nayler, Kalpana Patel, Chikako Ragan, Heather Smith. *Not pictured:* Mahdod Eftekar, Duncan McLean. **Background:** White matter images using diffusion tensor imaging in a schizophrenia patient.

Neural rosettes exhibiting expression of core neurogenic genes, PAX6 and MASH1 generated from schizophrenia patient induced pluripotent stem cells.

The Mowry laboratory aims to identify and functionally characterise susceptibility genes for schizophrenia and related disorders. The group aims to achieve this by combining genome-wide association studies (GWAS), DNA sequencing and transcriptome profiling with neuropsychological testing and neuroimaging in people with schizophrenia. Current studies include: (i) the recruitment of a large Indian case-control and family cohort in collaboration with Dr Rangaswamy Thara (Schizophrenia Research Foundation, Chennai); (ii) neuroimaging and neuropsychological phenotyping of schizophrenia patients with major copy number variations, and comparing patients with a matched sample of healthy individuals; (iii)

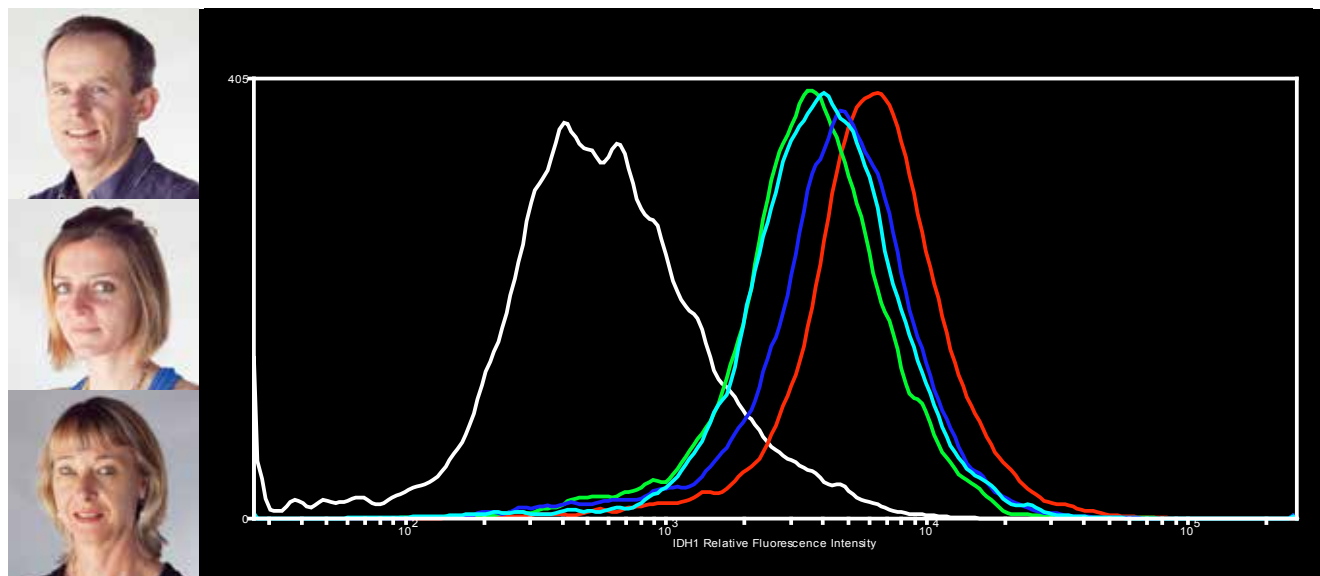
GWAS in homogeneous Indian and Sarawak populations, and relating the results to the latest European study results; (iv) transcriptome-wide analysis of small non-coding RNAs in post-mortem brain samples from schizophrenia patients and unaffected individuals; (v) targeted resequencing of a previously identified schizophrenia linkage region on chromosome 1 in an Indian case-control sample, using QBI's next-generation sequencing facility; (vi) whole exome sequencing of a large cohort of families in order to identify *de novo* and inherited mutations contributing to disease; (vii) derivation of neuronal cells using induced pluripotent stem cell (iPSC) technology, in a subset of schizophrenia patients and controls in

Psychiatric genomics

order to establish an *in vitro* model of disease; (vii) immunological subtyping of schizophrenia cohorts; (vii) coordinating the inclusion of Australian Schizophrenia Research Bank data in the latest Psychiatric Genetics Consortium (PGS) of schizophrenia.

Highlights during the year included contributions to the latest PGC schizophrenia GWAS that has identified in excess of 100 genetic susceptibility loci (due to be published in 2014), and publication of a perspective in *Nature Genetics* on the interpretation of *de novo* protein-coding mutations in neuropsychiatric disorders. The group also published a review in *Schizophrenia Bulletin* on the role for iPSCs in schizophrenia research.

Laboratory Head Mr Geoffrey Osborne



2013 Laboratory Members, top-bottom: Geoffrey Osborne, Anne-Sophie Bedin, Virginia Nink. **Background:** Flow cytometric comparison of IDH1 levels in glioma cell lines, with the proneural subtype cell line (red line), that has the highest levels of IDH1, detected above over cell lines. In this way quick comparisons yielding prognostic indications can be made. This approach is an example aimed at developing rapid prognostic indicators of patient survival.

Implementing novel approaches to solve fundamental problems

As Director of Flow Cytometry for both QBI and the Australian Institute for Bioengineering and Nanotechnology, Mr Geoffrey Osborne leads a team that provides crucial cell sorting and analysis services to researchers both within QBI and across the broader university. The laboratory specialises in the analysis and separation of cells derived from a variety of sources such as solid tissue, blood and cultured cell lines.

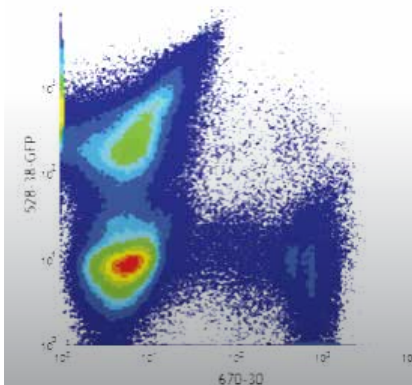
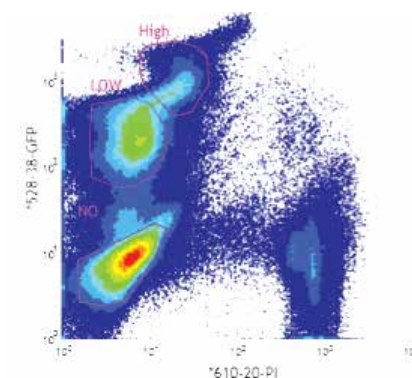
In 2013, investigations continued in the area of characterisation of cells from neural tissue. Many studies have used surface markers to enrich for and help purify, murine neural stem cells for research applications, however, there have been few effective intracellular labels that

stain putative stem cells. In 2013, the Osborne group published a paper demonstrating that a novel intracellular labelling dye, Cdy1, selectively enriches for neural stem cells from primary tissue from the mouse subventricular zone and hippocampal regions. The group plans to conduct further studies of brain cancer samples utilising Cdy1.

Working with collaborators from the QIMR Berghofer Medical Research Institute and the Royal Brisbane and Women's Hospital, the Osborne group has continued to investigate the surface marker expression present on tumour cells. It is hoped that combinations of markers present on these cells will provide information

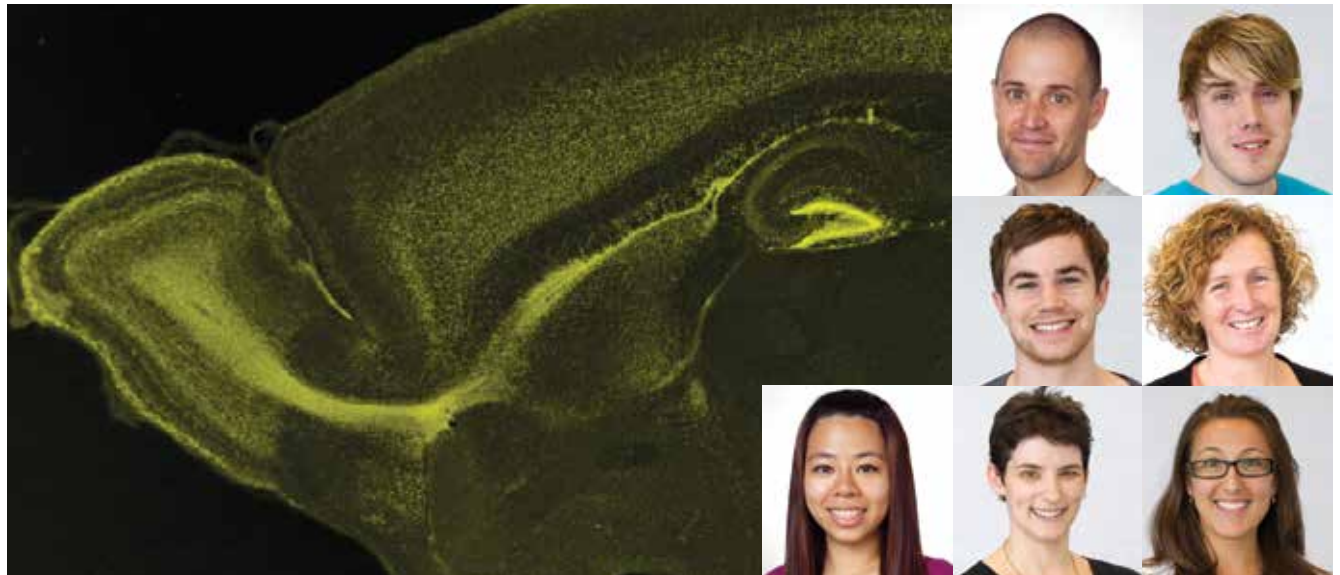
that is of both diagnostic and prognostic value.

On the instrumentation front, development and testing of prototype software that provides the first implementation of a novel cell sorting algorithm conceived at QBI has been undertaken. This software allows the selection of particular subsets of cells from a larger group of cells, with each cell having unique measured characteristics. The method has important applications in neuroscience and other areas of science.



Custom linear optical filters in a precision fabricated housing designed and built at QBI (top panel). Results generated from conventional bandpass filters (centre panel), with those of the QBI designed (bottom panel) system show comparable output, however the custom filter device can be tuned to any spectral range.

Laboratory Head Dr Michael Piper



2013 Laboratory Members L-R: Michael Piper, Joshua Eeles, Lachlan Harris, Tracey Harvey, Evelyn Heng, Chantelle Reid, Diana Vidovic. **Background:** Expression of the transcription NFIX (yellow) within the postnatal brain is shown in this sagittal section of a mouse brain. NFIX is expressed by neural stem cells and neurons within many regions of the brain.

Cell self-renewal and differentiation

The subventricular zone is one of the few regions of the brain in which the birth of new neurons continues throughout life. Many proteins are expressed within the subventricular zone of the adult mouse brain, including NFIA (red) and GFAP (green).

The brain is ultimately derived from neural stem cells, which differentiate to give rise to both neurons and glia within the developing and adult brain. Controlling how these neural stem cells either self-renew or differentiate is critical during development, and changes to the normal trajectory of these processes can lead to severe functional consequences. Moreover, many of the genes that control neural stem cell differentiation are misexpressed aberrantly within brain cancers such as glioma.

The Piper laboratory is investigating the molecular mechanisms underlying progenitor cell self-renewal and differentiation in order to

deepen our understanding of brain development and function, as well as to reveal the underlying deficits in brain cancers such as glioma. To do this it uses mouse model systems and *in vitro* cell culture paradigms to investigate the key processes behind the biology of neural progenitor cells, and to reveal the genetic hierarchy that controls neural progenitor cell differentiation.

In a paper recently published in *Cerebral Cortex*, the Piper laboratory revealed that a group of transcription factors named the Nuclear factor one family (NFI) play a central role in regulating how neural progenitor cells differentiate within the developing brain by revealing that NFI tran-

scription factors repress the expression of genes central to stem cell self-renewal. Moreover, in collaboration with Professor Francois Guillemot (MRC National Institute for Medical Research, UK) the group revealed a key role for NFI genes in adult neural stem cell quiescence (*Genes and Development*).

Current work in the Piper laboratory is aimed at further elucidating how NFI transcription factors control stem cell biology, and their contribution to the formation of glioma.

Laboratory Head Dr Judith Reinhard



2013 Laboratory Members L-R: Judith Reinhard, Stephanie Biergans, Julia Canning-Ure, Ming-Yu Chen, Alexandre Cristino, Shao-chang Huang, Homayoun Kheyri, Aoife Larkin, Morgane Nouvian, Amanda Robinson. **Background:** Honeybee (*Apis mellifera*) at a training feeder used in experiments to investigate learning of Monet and Picasso paintings by insects.

Cognitive capacities of small brains

Researchers in the Reinhard laboratory investigate how the brain processes sensory information and translates it into behavioural activity, thus linking brain function to behaviour. Particular focusses are the mechanisms underlying learning of sensory information, and how these memories support cognitive function. The laboratory uses insect model systems in combination with human research, and integrates behavioural studies with physiological and molecular approaches.

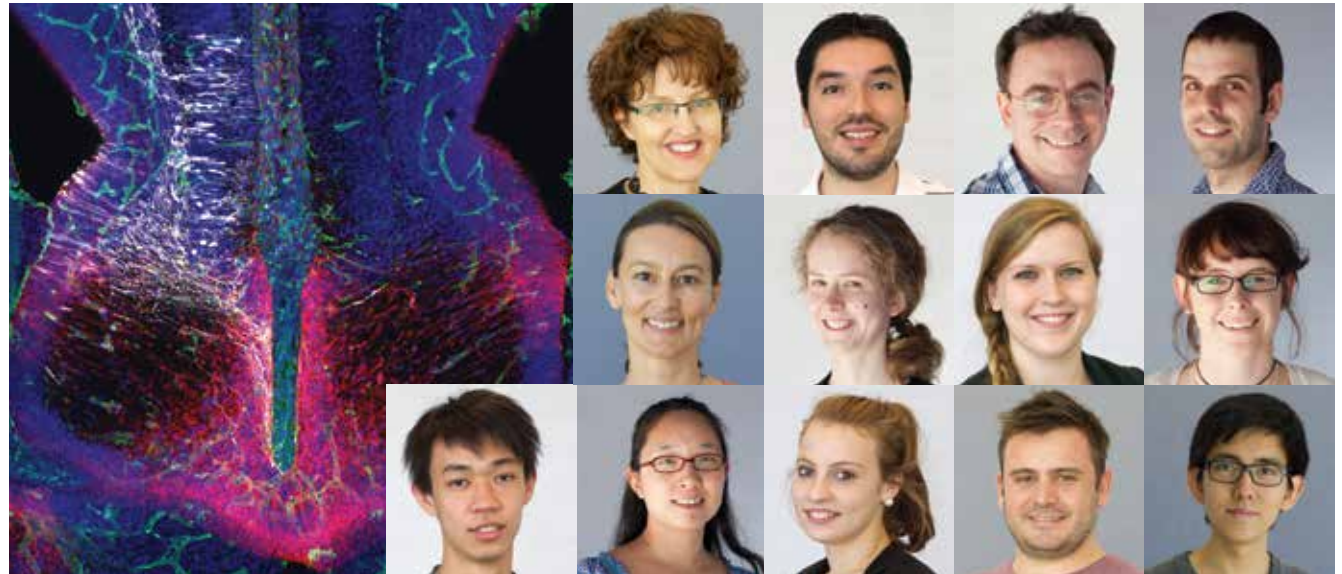
It has long been known that honeybees have remarkable visual learning abilities that extend beyond learning simple colours, shapes or patterns. They can learn and discriminate landscape scenes, types of flowers, and even

human faces. This suggests that in spite of their small brain, honeybees have a highly developed capacity for processing complex visual information, comparable in many respects to vertebrates and even humans. In collaboration with psychologists from UQ, the Reinhard laboratory investigated whether this capacity of honeybees extends to learning complex visual images that are thought to be specific to human cognition, namely paintings by Monet and Picasso. The team showed that honeybees indeed learned to discriminate between different Monet and Picasso paintings, and that they did not rely on luminance, colour, or spatial frequency information for discrimination. They also discovered that after training bees even

distinguished novel paintings by the same artists, suggesting that honeybees are able to discriminate Monet's paintings from Picasso's by extracting and learning the characteristic visual information inherent in each painting style. This research indicates that discrimination of artistic styles is not a higher cognitive function that is unique to humans, but simply due to the capacity of animals – from insects to humans – to extract and categorise the visual characteristics of complex images. This research was published in the *Journal of Comparative Physiology A*, and was reported in the BBC World News.



Examples of an Impressionist Monet painting (above) and a Cubist Picasso painting (below) that honeybees learnt to discriminate based on artistic style.

Laboratory Head **Professor Linda Richards**

2013 Laboratory Members L-R: Linda Richards, Gonzalo Almaraz, John Baisden, Jens Bunt, Ilse Buttiens, Amelia Douglass, Laura Fenlon, Ilan Gobius, Jonathan Lim, Samantha Liu, Laura Morcom, Rodrigo Suarez, Dennis Yeow. *Not pictured:* Michael Binzer, Tim Edwards, Lu Zhao. **Background:** Fluorescent confocal microscopy demonstrates how pioneering callosal axons (white) utilise midline glia (red) to navigate the interhemispheric fissure (green) during brain development. Image by I. Gobius.

Wiring the brain for function

Structured illumination microscopy demonstrates how microglial (red) and astroglial (magenta) cells interact with the surface of the interhemispheric fissure (green) during brain development. Image by I. Gobius.

The brain functions by integrating information from many sources and requires precise connections between functionally similar areas to achieve this goal. Researchers in the Richards laboratory are investigating how the brain forms these precise connections during prenatal stages and after birth. Determining how the brain is wired is of fundamental importance to understanding how the brain functions and can be repaired following injury, or in mental illnesses where brain wiring is disrupted.

The Richards laboratory is focussed on glial development, glioma and formation of the corpus callosum, the largest fibre tract in the human brain. This year, projects, performed in

collaboration with scientists at the Institute of Neuroscience, Shanghai, involved determining how callosal axons are sorted within the tract, enabling them to target the correct region in the contralateral hemisphere and discovered the basis of callosal defects in mice with disruptions in the *Midi1* gene associated with Opitz syndrome. Further work demonstrated that callosal axons integrate multiple molecular guidance cues at the midline of the brain in order to cross. The group discovered that axons use attractive cues to dampen the effects of repellents, allowing them to continue to grow and cross the midline effectively.

The laboratory also produced a systematic review of the literature on clinical syndromes associated with malformations of the corpus callosum, including hypotheses on the underlying causes of some of these syndromes. Medical student Tim Edwards, who is first author on the paper, spent time working with Professors Elliott Sherr and Jim Barkovich, at the University of California, San Francisco, to complete some of the work.

Laboratory Head Professor Pankaj Sah



2013 Laboratory Members L-R: Pankaj Sah, Eleanora Autuori, Suzanne Campbell, Peter Curby, Christine Dixon, Helen Gooch, Sarah Hunt, Roger Marek, John Morris, Margreet Ridder, Peter Stratton, Cornelia Strobel, Robert Sullivan, Yajie Sun, Tim Tattersall, Fabrice Turpin, Francois Windels, Li Xu, Shanzhi Yan. *Not pictured:* Madhusoothanan Bhagavathi Perumal, Petra Sedlak. **Background:** The medial amygdala is a part of the brain that receives olfactory information to drive defensive and reproductive behaviours. Shown is a neuron in the medial amygdala with the recording electrode attached. The neuron has been filled with a fluorescent indicator (Alexa 594) and imaged using a multi photon imaging system.

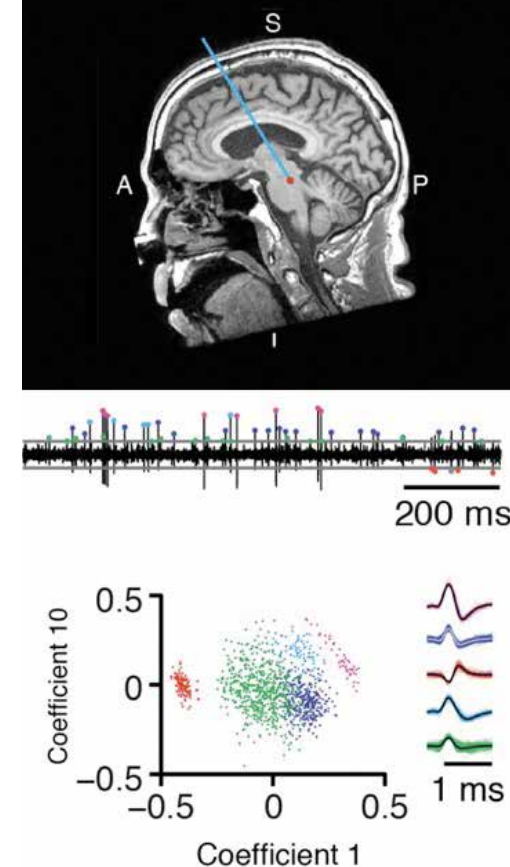
Neural circuits and mechanisms underpinning learning and memory

The Sah laboratory studies the physiological and molecular mechanisms that underlie learning and memory formation, focussing on a part of the brain called the amygdala. The amygdala is involved in emotional processing, and neural circuit dysfunction in the amygdala is thought to underlie a range of anxiety disorders. The Sah group uses electrophysiology and molecular techniques, in conjunction with behavioural studies, to understand the neural circuitry and cellular changes that underpin amygdala-dependent learning. The group uses viruses to deliver optogenetic constructs to neurons in defined regions, and then records the electrical activity in acute brain slices to study the properties of

the connections in these neural circuits. The role of these circuits in behaviour is being explored using multiunit recordings in awake, behaving animals. In the last year, the group has mapped the circuits that provide auditory information to the amygdala. They have also studied the circuits that connect the amygdala with the prefrontal cortex and hippocampus.

In collaboration with Professor Joe Lynch, the group is exploring the molecular identity of GABA receptors in the amygdala that could be targets for the development of new anxiolytic drugs. They have determined the properties of receptors that contain gamma1 subunits and are enriched in the amygdala.

Professor Sah also collaborates with Professor Peter Silburn and Dr Terry Coyne (UQ Centre for Clinical Research) to study neural activity in the human brain in patients undergoing neurosurgery for deep brain stimulation. These recordings are revealing the activity in the human brain in a range of movement disorders, such as Parkinson's disease, essential tremor and Tourette's syndrome. In 2013, the group studied the properties of cells in a part of the brain called the pedunculo pontine nucleus, which is involved in locomotor control.



Recordings from the human pedunculo pontine nucleus (PPN). The upper panel shows an MRI scan of human brain. The blue line marks the location of the recording electrode in the PPN. A- anterior, P- posterior, S- superior, I- inferior. The trace below the MRI shows a recording in an awake patient that identified five different neurons each marked with a different colour. The bottom panel shows the results from spike sorting to identify the different cell types, and the shape of each spike is shown on the right.

Laboratory Head **Professor Mandyam Srinivasan**

At the biorobotics laboratory, we usually take inspiration from biology to build better robots. Sometimes the inspiration goes the other way as well. Authors: Michael Knight, Saul Thurrowgood and Gavin Taylor.



2013 Laboratory Members L-R: Mandyam Srinivasan, Kathy Asmussen, Samuel Baker, Julia Groening, Michael Knight, Nikolai Liebsch, Ingo Schiffner, Dean Soccol, Saul Thurrowgood, Hong Vo. *Not pictured:* Peter Anderson, Aymeric Denuelle, Cassandra Guilfoyle, Marcel Schumacher, Gavin Taylor, Trevor Weatherhead. **Background:** An aircraft endowed with vision inspired by insect research is released to explore its environment, avoiding obstacles by watching passing objects and navigating by patterns in the sky. Image: Saul Thurrowgood.

Visual guidance in insects and birds, and aircraft navigation

Flying insects display remarkable visual agility, despite their diminutive brains. The Srinivasan laboratory is using honeybees and budgerigars as models to understand how vision guides flight and enables navigation. They are also using these insights to design novel, biologically inspired strategies for the guidance of aircraft.

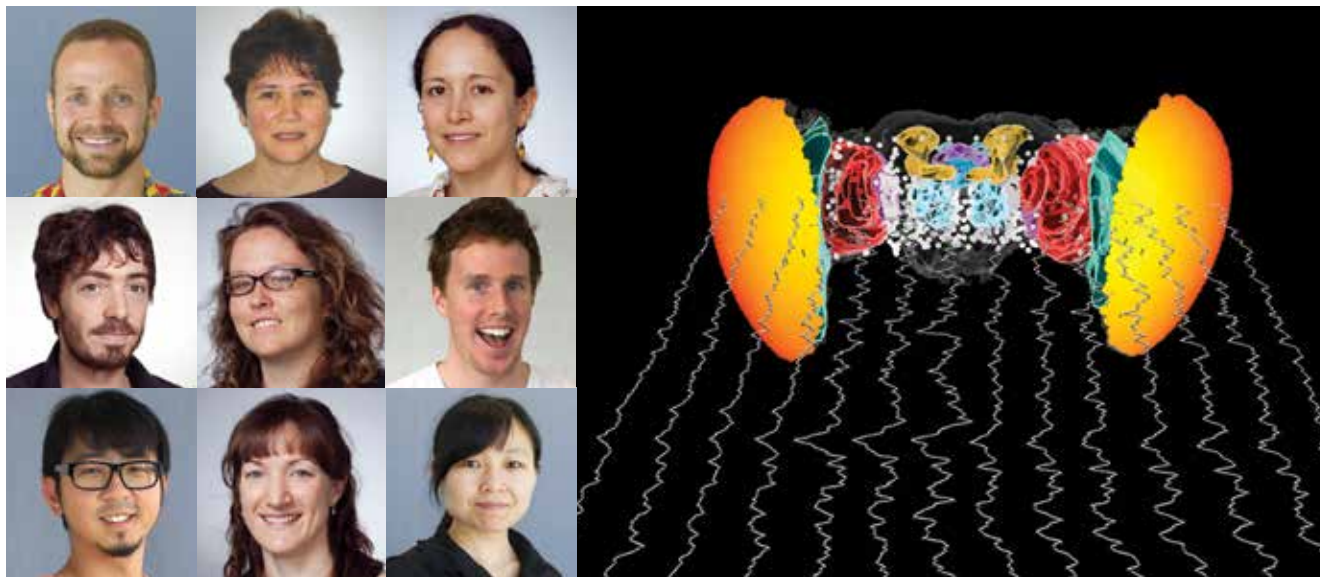
In the honeybee laboratory, analysis and modelling of the way in which honeybees land has led to a better understanding of how this intricate manoeuvre is orchestrated, and enabled the formulation of a universal, visually-guided strategy for landing that can be used by animals as well as flying machines. In another study, a collaboration with researchers in the

School of Information Technology and Electrical Engineering, has led to a quantitative model that characterises how flying honeybees avoid mid-air collisions. The results are of potential relevance to aviation safety.

In the bird laboratory, studies of budgerigar flight along a 25-metre tunnel with moving patterns projected on the inside walls is revealing that birds control the speed of their flight, at least in part, by monitoring the speed of the image of the environment in their eyes. Further studies are working on the decision making of budgerigars, with the aim of understanding how flocks navigate through complex environments such as forests without birds colliding with each other.

In the robotics laboratory, a novel, insect-like, multi-legged landing gear has been developed to enable a multirotor aircraft to land safely on uneven terrain. Another highlight has been the development of an aircraft-mounted vision system that is capable of detecting a moving object in the environment while the aircraft itself is in motion. The system can potentially be used to track the moving object, or avoid collisions with it.

Laboratory Head Associate Professor Bruno van Swinderen



2013 Laboratory Members L-R: Bruno van Swinderen, Kathy Asmussen, Leonie Kirszenblat, Ben Kottler, Angelique Paulk, Michael Troup, Melvyn Yap, Oressia Zalucki, Yan-Qiong Zhou. Not Pictured: Alice Petty. Background: Electrical activity is recorded from across the *Drosophila* brain.

Drosophila behaviour and cognition laboratory

The van Swinderen laboratory uses the fruit fly model *Drosophila melanogaster* to investigate perception and cognition. By combining powerful molecular genetic tools with high-throughput behavioural assays and electrophysiology, they are able to study the underpinnings of complex phenomena such as selective attention, memory, general anaesthesia, and sleep in the more simple fly brain. To pay attention, learn, and sleep a brain must be able to suppress parts of the outside world effectively. Understanding how this suppression mechanism works is a central question of the laboratory, with a focus on visual systems.

Using high-throughput behavioural assays, the laboratory has developed a model for studying attention-like defects in a fly model for schizophrenia. This research, done in collaboration with the laboratory of Associate Professor Darryl Eyles, revealed a role for developmental dopamine in modulating visual attention in adult animals.

In collaboration with colleagues in Beijing, the laboratory is also developing electrophysiological and behavioural approaches to studying visual selective attention. They have discovered frequency-specific responses in the fly brain, and how transient control of key neurons modulates these responses (*Journal of Neurophysiology*).

In other work focussed on sleep and general anaesthesia, the laboratory has discovered distinct sleep stages in flies (*Journal of Neuroscience*), and has found that a defined sleep circuit in the fly brain controls sensitivity to common general anaesthetics such as isoflurane (*Current Biology*).

In addition to *Drosophila* work, the laboratory also studies the honeybee in order to gain insight on neural activity associated with visual attention and learning. In recent work, the laboratory has localised attention-like processes in the bee optic lobes.



Brain activity is recorded from a fly walking on an air-supported ball.

Laboratory Head **Professor Peter Visscher**

Jian Yang received a 2013 UQ Foundation Research Excellence Award.



2013 Laboratory Members L-R: Peter Visscher, Beben Benyamin, Marie-Jo Brion, Guo-Bo Chen, Gibran Hemani, Hong Lee, Allan McRae, Gerhard Moser, Joseph Powell, Sonia Shah, Konstantin Shakhbazov, Anna Vinkhuyzen, Jian Yang, Zhihong Zhu. *Not pictured:* Anita Goldinger. **Background:** Joint analysis for each chromosome for estimating the genetic variance in risk to Alzheimer's disease from SNP data.

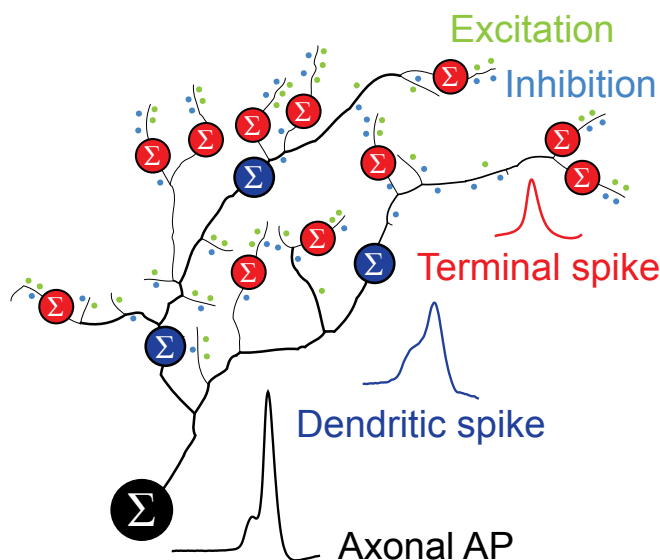
Genes in human populations for common diseases and their risk factors

The Visscher laboratory specialises in quantitative and statistical genetics, population genetics, human genetics and bioinformatics, with the ultimate aim of trying to understand the genetic basis of differences in risk to disease and other phenotypes between individuals. Applications of the research include dissection of genetic variation underlying cognition and cognitive change, and quantification and deciphering of the genetic architecture of psychiatric disorders. The group uses theoretical derivations, simulation studies, development of new analytical methods and software tools, and the application of advanced statistical analysis methods to genetic and phenotypic data.

In 2013, the group demonstrated that individual differences in complex traits, including cognitive ability, learning and memory, and risk of Alzheimer's disease, are caused by the cumulative effect of many genes. In addition to a number of significant publications, this also resulted in Dr Jian Yang receiving a UQ Foundation Research Excellence Award and was one of two recipients of the Sylvia and Charles Viertel Charitable Foundation's Senior Medical Research Fellowship. The group has also contributed analysis expertise to a large number of international research consortia that have found genes affecting schizophrenia, cardiac rhythm, asthma and obesity.

In collaboration with researchers from the QIMR Berghofer Medical Research Institute, Professor Visscher has established the Brisbane Systems Genetics Study, with the aim of understanding genetic variation in gene expression and its correlation with individual differences in complex traits. They have shown that gene expression itself is under genetic control and that gene expression is correlated with disease. A long-standing collaboration with Professor Ian Deary (University of Edinburgh, UK) has been expanded through joint projects on the genomics underlying cognitive ageing.

Laboratory Head Associate Professor Stephen Williams



2013 Laboratory Members L-R: Stephen Williams, Arne Brombas, Florence Cotel, Lee Fletcher, Ben Sivyer. **Background:** Schematic representation of active dendritic integration in an output cell of the retina.

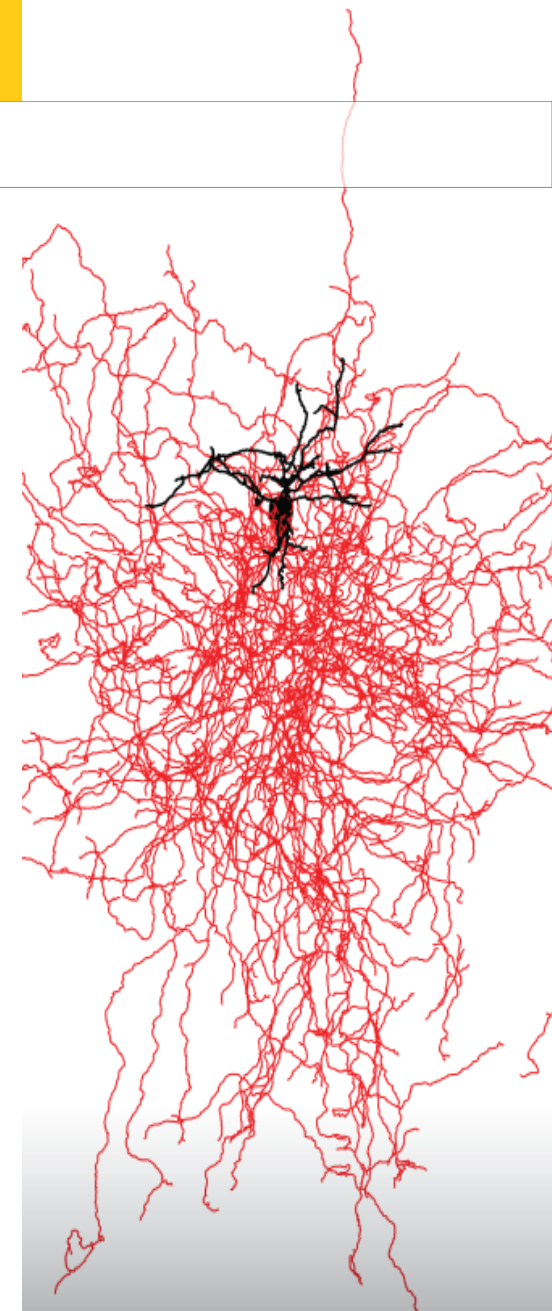
Computation in neurons and circuits

The brain is fundamentally a computational device. Unlike in a personal computer, the components underlying brain computations are nerve cells, not transistors. In the brain, nerve cells are arranged in intricate networks, in which neuronal computations are performed that underlie all aspects of behaviour. The Williams laboratory is investigating how nerve cells and neural circuits implement computations. They use advanced electrophysiological and optical techniques to investigate how neurons integrate input signals, termed synaptic potentials, received throughout their dendritic tree, to produce an output signal. This work has shown that single neurons can operate as complex computational devices; acting to produce finely tuned output signals through

the engagement of active dendritic synaptic integration. The processing capacity of single neurons has been found to be equivalent to that of a vast array of transistors, highlighting how the brain can operate in a fast and energy efficient manner. The laboratory seeks to understand the rules and mechanisms that form and control this rich neuronal integrative process and explore the relevance to the operation of neuronal networks in health and disease.

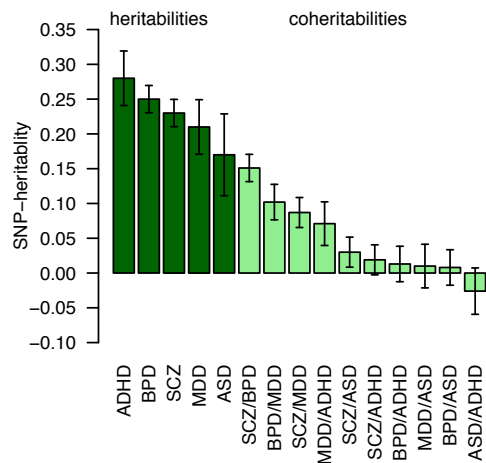
Recent work has revealed that active dendritic integration is recruited by natural stimuli and implements circuit based computations in the neuronal networks of the neocortex and retina. Researchers have discovered that complex

processing by single neurons implements multi-modal processing in neocortical circuits, acting to compute the location of objects in the environment by the integration of motor and sensory information. In the output neurons of the retina, members of the Williams laboratory have found that dendritic integration underlies the computation of image motion. Ongoing work is exploring how these and other neuronal computations are engaged and controlled. This work will enable neuroscientists to better understand how networks of neurons function and ultimately how these processes are disturbed in disease.



Dendritic (black) and axonal arbor of an inhibitory neuron in the neocortex.

Laboratory Head Associate Professor Naomi Wray*



Evidence for the common genetic contribution (SNP-heritability) to psychiatric disorders. *Nature Genetics*, 2013.



2013 Laboratory Members L-R: Naomi Wray, Enda Byrne, Suzanne Campbell, Guo-Bo Chen, Jake Gratten, Hong Lee, Divya Mehta, Gerhard Moser, Wouter Peyrot, Matthew Robinson, Anna Vinkhuyzen. QBI Bioinformatics Core: Zong-Hong Zhang, Qiongyi Zhao. Background: Flow chart for prediction of genetic risk from genetic data. *Nature Reviews Genetics*, 2013.

Probing of the genomic complexity between & within psychiatric disorders

Research in the Wray laboratory focusses on understanding the genetic contribution to psychiatric disorders. The group specialises in the development of new analytical methods and the application of advanced statistical methods to the analysis of genetic and phenotypic data of psychiatric disorders. Group members play leading roles in international consortia including the International Psychiatric Genomics Consortium, which has brought together large samples of genetically informative cases and controls from the five major psychiatric disorders: schizophrenia, bipolar disorder, major depressive disorder, attention deficit-hyperactivity disorder and autism spectrum disorder.

In 2013 the Wray laboratory published a novel analysis in *Nature Genetics* showing the shared genetic relationship attributable to common DNA variants between these five disorders. It has long been speculated that there may be a common genetic vulnerability across the spectrum of psychiatric disorders. However, demonstrating this from epidemiological data is difficult since very large cohorts of families are needed, with family members recorded for status on multiple disorders. Even if such data sets were collected, separating shared genetic from shared family effects is challenging. The novel analyses of the Wray laboratory used independently collected case-control data sets for two disorders, and use the directly measured

DNA variation to demonstrate the relationship between disorders. They confirmed a shared genetic relationship between schizophrenia and bipolar disorder, and between bipolar disorder and major depression, but also showed that genetic vulnerability is shared between schizophrenia and major depression. These results contribute to discussions on psychiatric nosology and help shape future experimental design in understanding the complexity of human disorders. Research has focussed also on strategies that define more homogenous diagnostic classes for genetic studies of psychiatric disorders, for example using major depression, postnatal depression, seasonal affective disorder or sleep dysregulation.

*Promoted to Professor, effective 1 January 2014.

Clem Jones Centre for Ageing Dementia Research

Housed within QBI, the Clem Jones Centre for Ageing Dementia Research (CJCADR) was officially opened by Queensland Premier Campbell Newman on 28 February.

Dementia affects more than 320,000 Australians and is the nation's third leading cause of death, after heart disease and stroke.

There is no cure, and without a significant medical breakthrough, the number of Australians living with dementia is expected to soar to almost one million by 2050.

Mr Newman said dementia research was vitally important and congratulated those involved with the establishment of the new Centre.

"Research leads to better healthcare practices, less disease, and improvements to quality and longevity of life.

"It also helps to address the significant pressures facing the public health system," he said.

Headed by world-renowned neuroscientist, Professor Jürgen Götz, the Centre is Australia's first and only facility focussed entirely on research into the prevention and treatment of dementia.

Along with early detection and preventative strategies, therapeutic intervention is key to minimising the social and economic impact of dementia in Australia.

In May, the Premier announced that CJCADR would receive \$9 million in funding over the next five years, to support research within the Centre.

The funds will help attract more of the world's brightest neuroscientists and advance diagnostic tools for early intervention and clinical trials of new therapeutic approaches currently being explored.

"This is a fantastic boost to our capabilities of translating our discoveries into new diagnostics and therapeutic treatments of dementia," Professor Perry Bartlett said.

"In addition it will fast track our work, which aims to bolster the function of the ageing brain and offers the opportunity to improve learning and memory in an ageing population," he said.

Clem Jones Foundation Chairman, David Muir, said the State Government support would have been welcome news for the late Lord Mayor Clem Jones, after whom the Centre was named.

"If Clem was alive he would be over the moon with the announcement by Premier Campbell Newman of the grant of \$9 million to the Centre," he said.

"Clem would applaud the Queensland Government for being prepared to spend millions of dollars on medical research to find a cure for ageing dementia in order to save billions of dollars of expense in caring for future generations."

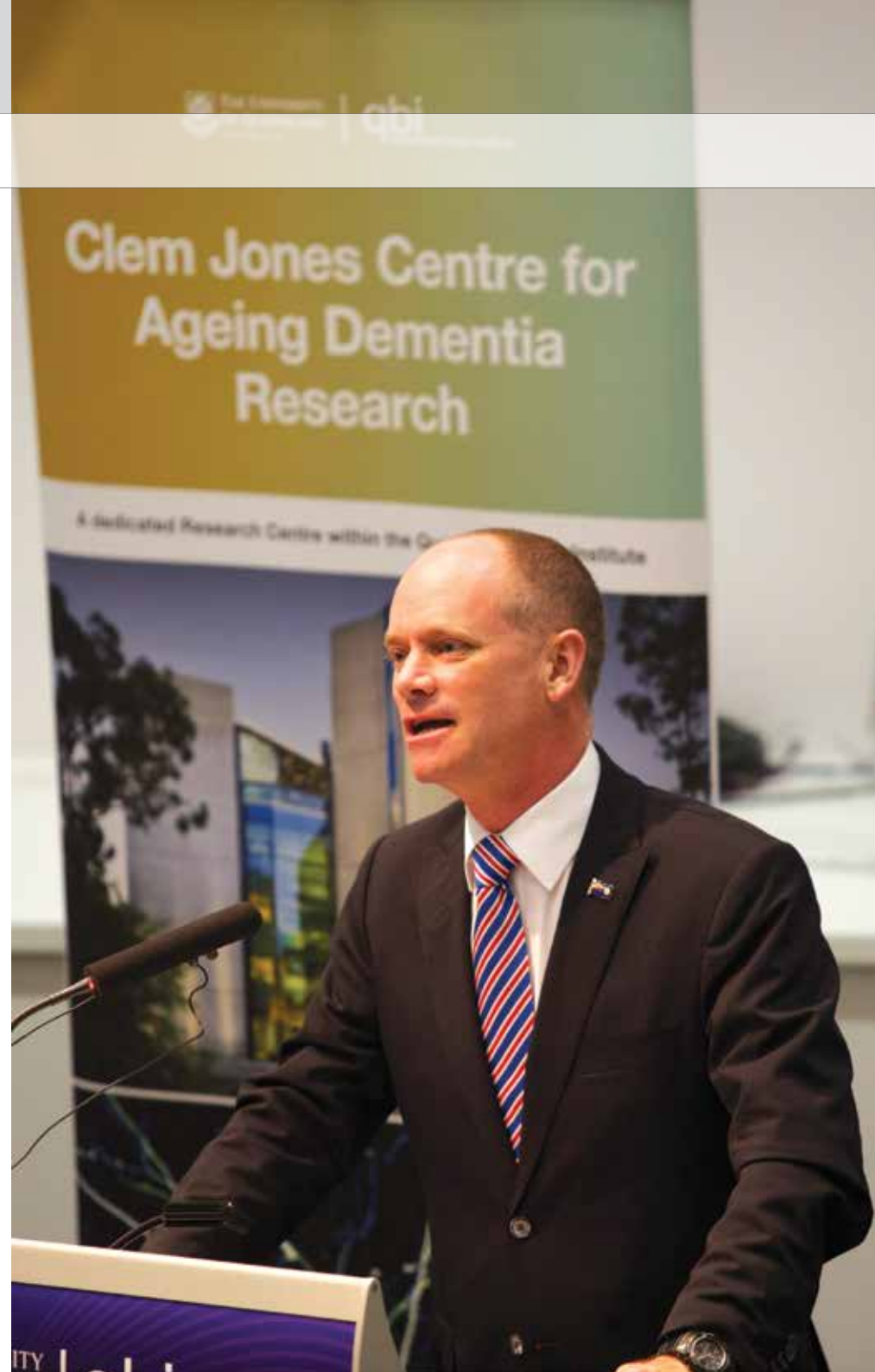
The State Government funding was matched with a commitment of \$9 million, announced by Prime Minister Tony Abbott during the Federal Election campaign.

Federal Member for Ryan, Jane Prentice, joined with new Federal Health Minister, The Hon Peter Dutton MP, at QBI on 20 September for his first official event, to confirm the Federal Government's \$9 million funding commitment to CJCADR.

This funding forms part of the Federal Government's \$200 million commitment to dementia research.

In addition to the planned expansion through new recruits, researchers from the following QBI laboratories all undertake dementia-related research within CJCADR: Bartlett, Coulson, Hilliard, Mangelsdorf and Meunier.

Right: Queensland Premier Campbell Newman officially opened CJCADR.



Clem Jones Centre for Ageing Dementia Research



Professor Jürgen Götz, Director CJCADR

CJCADR research is already making waves in the neuroscience space and is being used to develop therapeutic interventions.

Professor Götz has earned international acclaim for the discovery of how the molecule tau causes neuronal demise in Alzheimer's disease.

"Highly enriched in neuronal cells, this protein forms insoluble clumps in the brains of Alzheimer patients, causing their neuronal demise and, ultimately, dementia," Professor Götz said.

After discovering how tau exerts its damage, CJCADR scientists are developing small compounds, peptides and antibodies in order to block the disease process. The team is also developing methods to assist in the clearance of another molecule that forms toxic aggregates in the Alzheimer's brain, amyloid-beta. How amyloid-beta and tau interact and cause neurons to degenerate is a long-standing interest of Professor Götz.

Also high on CJCADR's research agenda is cell death and discovering molecules that might interfere with or block this death pathway.

"While more intensive research is needed to bring these discoveries to the patient, CJCADR scientists are on a launching path to make discoveries that help the growing number of dementia patients, both in Australia and overseas," he said.



Dr Victor Anggono

The Anggono group uses an integrative approach to understand the molecular mechanisms underlying neuronal communication by combining biochemistry, cell biology, molecular manipulation of genes, electrophysiology, the use of animal models and behavioural analysis.

This fundamental biological process is crucial for information processing in the brain. It depends on the ability of neurons to transmit chemical signals (neurotransmitters) across the synapses, which in turn bind to neurotransmitter receptors on the other neurons.

The AMPA-type neurotransmitter receptors mediate most of the fast synaptic transmissions in the brain. The ability of neurons to modulate the strength of their connections, termed synaptic plasticity, is determined in part by the number of these receptors at synapses. Dysregulation in AMPA receptor trafficking has been implicated in various neurological disorders, such as Alzheimer's disease, schizophrenia, bipolar disorders and autism. The major aim of the Anggono group is to understand the detailed molecular mechanisms regulating AMPA receptor trafficking and synaptic plasticity, one of the cellular correlates of learning and memory.

In a paper recently published in the *Proceedings of the National Academy of Sciences of the United States of America*, the Anggono group revealed a novel interaction between two membrane-sensing proteins, PICK1 (Protein Interacting with C-Kinase 1) and PACSIN (Protein Kinase C and Casein Kinase II Substrate in Neuron), and that they form a complex with AMPA receptors. The phosphorylation-regulated interaction between these two proteins is required for AMPA receptor internalisation, and more importantly, for the expression of cerebellar long-term depression, which is thought to control fine motor movement.

Currently, the Anggono group aims at uncovering the roles of post-translational ubiquitination in regulating AMPA receptor functions, and their contribution to synaptic plasticity, learning and memory.

Group Members: Victor Anggono, Ye Jin Chai, Yu Qian Chau, Se Eun (Joanne) Jang, Tong (Tina) Lin.

Purkinje cells of the rat cerebellum were visualised by immunohistochemical staining with specific antibodies against the calcium binding protein, calbindin.

Science of Learning Research Centre

In May, the Australian Research Council (ARC) awarded \$16 million over four years for a Science of Learning Research Centre (SLRC), a Special Research Initiative led by The University of Queensland (UQ) and involving researchers from QBI, the School of Psychology, the School of Education and the Centre for Advanced Imaging.

This collaboration between UQ, The University of Melbourne, Australian Council for Educational Research (ACER), Deakin University, Flinders University, Charles Darwin University, University of New England and Macquarie University will establish new means to assess the impact of different types of learning and strategies to inform teaching practices to benefit all Australians.

Officially opened by the Honourable Ian Walker, Minister for Science, Information Technology, Innovation and the Arts on 27 November, the Centre brings together researchers in education, neuroscience, and cognitive psychology to work with teachers to enhance our understanding of the learning process.

“The objectives of the SLRC are to identify, research and understand effective learning processes and factors that influence successful human learning,” Professor Perry Bartlett said.

“The establishment of the SLRC will, for the first time, allow us to take what we know about how the brain learns and translate that into educational outcomes,” he said.

“A benchtop-to-blackboard approach is revolutionary for the learning community.”

The grant comes four years after the concept was recommended by the Prime Minister's Science, Engineering and Innovation Council Expert Working Group, of which Professor Bartlett was a member, in their report, *Transforming Learning and the Transmission of Knowledge*.

The grant was led by Professor Ottmar Lipp from UQ's School of Psychology, in cooperation with Professor Pankaj Sah, Professor John Hattie (University of Melbourne) and Dr Mike Timms (ACER). Professor Lipp says the Centre will place learning at the focus of its research and develop an evidence based approach to educational practice.

“It is essential that this is done in collaboration between researchers from different disciplines on the one side and practitioners on the other. This collaboration will be facilitated by centerpieces of the Centre, two experimental classrooms, one at UQ and one at The University of Melbourne.”

In addition to the eight research organisations, the SLRC is supported by nine partner organisations, including the Queensland Department of Education and Training, the Victorian Department of Education and Early Childhood Development, the South Australian Department of Education and Child Development, Questacon, North Carolina State University, Institute of Education, London, Carnegie Mellon University, University College London, and the Benevolent Society.



Joint Sino-Australian Neurogenetics Laboratory

The Joint Sino-Australian Neurogenetics Laboratory, an initiative of QBI, UQ's Diamantina Institute (UQDI) and the Second Military Medical University (SMMU) in Shanghai, is dedicated to exploring how genes influence brain development and function, and focusses on discovering the genes that cause or make individuals susceptible to certain neurological and mental illnesses.

Under the guidance of Professor Huji Xu, who holds an appointment with QBI and SMMU, researchers are probing the neurogenetics of motor neuron disease (MND), schizophrenia and epilepsy.

Researchers in the laboratory are engaging with a network of neurologists and psychiatrists at various hospitals and universities across China to study these diseases and disorders. It is anticipated that differences in ancestral genetic diversity will enable mapping of genes in certain populations that are not easily identified in other populations.

Project profile

Motor neuron disease

MND is a devastating neurodegenerative disease characterised by progressive muscle weakness and wasting. About 10 per cent of MND is familial, that is more than one person in the family has been or is affected, and is usually the result of a genetic mutation. Genetic mutations are also found in cases where there is no family history – sporadic cases.

Professors Perry Bartlett and Peter Visscher, in collaboration with Professor Matt Brown (UQDI) and Professor Huji Xu (SMMU), are conducting large population studies to identify genes that are associated with MND. This research will lead to new genetic markers, aid development of diagnostic tools and identify new therapeutic targets for MND.



Right: Professors Perry Bartlett (left) and Matt Brown (right) show UQ Vice-Chancellor and President, Professor Peter Høj (centre) through the Joint Laboratory during a Senior Executive Mission to China in March.

Joint Laboratory of Neuroscience and Cognition

Researchers within the Joint Laboratory of Neuroscience and Cognition, between QBI and the Institute of Biophysics (IBP) in Beijing, bring together complementary expertise and advanced technologies in cellular and molecular systems.

They share a goal to discover how functions like learning and memory are regulated, and use these discoveries to develop new techniques to treat the many neurological and mental diseases facing both countries.

In three short years QBI and IBP have built a truly collaborative laboratory of research excellence that continues to expand, both in terms of the number of projects being undertaken, the number of researchers involved in the projects, and the many visits by students, postdoctoral fellows and Faculty that occurred between the two countries.

Professor Jürgen Götz, inaugural Director of the Clem Jones Centre for Ageing Dementia Research (CJCADR), has developed a new collaborative project with Professor Rongqiao He (IBP) looking at the role of formaldehyde in cognitive impairment, by investigating the role of formaldehyde in tau modification and aggregation and dysfunction.

Dr Cornelia Strobel, who works with Professors Pankaj Sah and Jianyuan Sun (IBP) studying the neural circuits that mediate pain pathways in the amygdala, spent a length of time in Beijing in 2013, and was successful in obtaining a Chinese Academy of Sciences (CAS) Visiting Scientist Fellowship to return in 2014.

IBP received further funding of 1.1 million RMB from the External Cooperation Program of the Bureau of International Cooperation, CAS, to support the joint project “The mechanisms of learning-memory and the related brain disorders”. The project is being undertaken by Professors Rongqiao He (IBP), Li Liu (IBP), Yan Zhu (IBP) and Perry Bartlett, and Associate Professors Ying Liu (IBP), Bruno van Swinderen and Helen Cooper. It aims to reveal the neural circuits involved in learning and memory, and the genetic and epigenetic mechanisms of the related brain dysfunctions.



Joint Sino-Australian Laboratory of Brainnetome

In March, the Joint Sino-Australian Laboratory of Brainnetome was opened as a joint initiative between QBI and the Institute of Automation at the Chinese Academy of Sciences (CASIA) in Beijing.

Professor Tianzi Jiang, a neuroimaging researcher who has a joint appointment between UQ and CASIA, is overseeing the development of the new laboratory, which is focussed on using advanced imaging techniques and computational analysis to understand brain behaviour.

Researchers within the Joint Laboratory are using neuroimaging technology to track and map brain behaviour when performing tasks, thereby developing a greater understanding of how neural networks function in the healthy brain.

The findings will be used to develop computational models to diagnose changes in brain wiring associated with diseases.

Computational analysis and neuroimaging will also be applied to other areas of research at QBI, such as learning and memory. In particular, this approach will be used in the Science of Learning Research Centre (SLRC) to understand the networks involved during learning and the formation of memories, and then applied towards delivering new innovative education models in schools and universities.

The Joint Laboratory also hosted the second Symposium on Brainnetome Meets Genome (SBMG) in September, bringing together experts in neuroscience, neuroanatomy, medical genetics, neuroimaging and neurotechnology, all of whom have a special interest in understanding the relationships between brain networks, brain functions and malfunctions, and risk genes of neuropsychiatric diseases. The inaugural SBMG took place at QBI in May 2012.

Far left: Associate Professor Bruno van Swinderen, who has a joint research project with Professor Li Liu at IBP, examining a vial of *Drosophila* food. Below: Professors Perry Bartlett (left) and Tianzi Jiang (right) sign the agreement underpinning the Joint Laboratory that opened in Beijing in March. Image courtesy of CASIA.



STUDENTS



Hanne Theon,
Marshall laboratory.

Students

Students play an integral role in the cutting-edge research undertaken at QBI. Students travel from as far afield as China, Latin-America and Europe to study, bringing fresh, innovative and international approaches to neuroscience research at the Institute.

STUDENT STORIES AND PROFILES

QBI had a total of 97 research higher degree students enrolled in 2013, of whom 46 were international candidates from Argentina, Austria, Bangladesh, Canada, Chile, China, Ecuador, France, Germany, India, Iran, Italy, Korea, Malaysia, Burma, New Zealand, Norway, Singapore, Switzerland, Taiwan, Thailand, the United Kingdom, the United States of America and Vietnam.

In 2013, QBI welcomed 21 new domestic and international students who commenced their research candidature during the year. QBI was also delighted to see the conferral of 12 PhD awards and one MPhil upon the following students across two graduation ceremonies held in July and December:

Jessica Barnes (Bellgrove* laboratory), Charlotte Clark (Cooper laboratory), Michael Dwyer (MPhil, Mattingley laboratory), Lu Han (Lynch laboratory), Thuan Huynh (Adams* laboratory), Sepideh Keshavarzi (Sah laboratory), Inga Laube (Bellgrove* laboratory), Sha Liu (Richards laboratory), Roger Marek (Sah laboratory), Ramesh Narayanan (Wallace* laboratory), Kian Bee Ng (Cunnington laboratory), Daniel Stjepanovic (Bellgrove laboratory) and Jiajia Yuan (Goodhill laboratory). These graduates have subsequently gone on to postdoctoral work or into research administration.

QBI students were awarded multiple highly competitive scholarships during the year. Some of the most successful international scholarship recipients were: Natalie Lee (USA) who was awarded the top international scholarship offered at UQ, the International Postgraduate Research Scholarship (IPRS), in conjunction with the Australian Postgraduate Award (APA) and the UQ Advantage Top-Up. Morgane Nouvian (France) was awarded the UQ Centennial Living Allowance and QBI Top-Up Scholarship. Seven domestic PhD students who commenced their studies in 2013 each secured the top Australian Government scholarship of an Australian Postgraduate Award (APA), and one commencing student received the UQ Research Scholarship.

The annual UQ Summer Research Program and the UQ Winter Research Program also provides QBI the opportunity to welcome undergraduate and postgraduate coursework students. For the Summer Research Program 2013/2014, QBI welcomed 16 domestic and international students to undertake a range of projects across 10 different laboratory groups within QBI. The Winter Research Program involved a total of four students who participated in various laboratory-based projects across three different laboratory groups over a six-week period.

Students chart course to success

Students at QBI are provided with opportunities to pursue their research interests, while working closely with dedicated neuroscientists. This research and work experience provides the students with a solid foundation for career success.

Dr Roger Marek

Dr Roger Marek commenced his PhD in the laboratory of Dr Louise Faber, where he focussed on the investigation of prefrontal neuronal correlates and ion channels that are implicated in higher cognitive and executive functions using electrophysiological techniques. After Dr Faber left QBI, Roger later joined Professor Pankaj Sah's laboratory where he investigated the neuronal circuitry between the prefrontal cortex and fear-related structures. He showed that prefrontal regions integrate and process synaptic information on a sub-region and layer-specific manner. Moreover, Roger also collaborated with Dr Tim Bredy to study epigenetic changes of prefrontal regions in fear learning. Specific proteins were identified to modulate synaptic plasticity and the extinction of conditioned fear. These findings are important to understand the cause for the development of anxiety disorders such as phobias and post-traumatic stress disorder.

Since the completion of his PhD thesis, Roger has remained in Professor Sah's laboratory to deepen the understanding about this fear-related neuronal circuit using optogenetic techniques in freely behaving animals.

Dr Inga Laube

Dr Inga Laube joined the cognitive neuroscience group of QBI in 2009 to begin her doctoral research investigating the control mechanisms of attention in the human brain. During her candidature she was supervised by Professor Mark Bellgrove, Professor Jason Mattingley and Dr Angela Dean.

Inga's research focussed on the attentional mechanisms that allow the goal-dependent filtering of visual information. Using functional magnetic resonance imaging (fMRI) as well as electroencephalography (EEG), Inga investigated the brain areas involved in this cognitive function as well as its neurochemical modulation. Another focus of her research was the question of how attentional control mechanisms are affected in patients with hemispatial neglect – an attentional disorder that often occurs after unilateral stroke. The results of this study indicate that the attentional control system in hemispatial neglect is partially intact, suggesting a potential gateway for improving current rehabilitation strategies for this disorder.

Since the completion of her PhD at UQ in 2013, Inga has taken up a postdoctoral position at the Neuroscience Research Centre in Lyon (CRNL), France, to continue her research on hemispatial neglect and the development of rehabilitation strategies for patients who have suffered from a stroke.

*Former QBI Laboratory Heads.

MASTER OF NEUROSCIENCE STUDENTS

Master of Neuroscience

The Master of Neuroscience program was introduced in 2010 as an initiative of QBI Director Professor Perry Bartlett and Senior Deputy Vice-Chancellor Professor Deborah Terry, as a pathway for students who wish to shift their career focus to neuroscience and pursue independent research and teaching careers.

The program is coordinated by QBI and the Faculty of Social and Behavioural Sciences (now Faculty of Health and Behavioural Sciences), but also spans other centres for neuroscience research at UQ. To ensure quality of student experience and teaching, a quota of 12 students per semester has been imposed upon the course.

Providing research training and core professional skills, the program is a pathway to specialist streams including molecular and cellular neuroscience, neural imaging and computational neuroscience, developmental neurobiology, cognitive and behavioural neuroscience, visual and sensory neuroscience and epigenetics. The Master of Neuroscience runs for three semesters (24 units), although students with Honours or equivalent can complete the program in two semesters (16 units).

In 2013, a total of nine students graduated with the Master of Neuroscience degree. They were: Richard Carey, Brett Kagan, Matthew Kennett, Elliot Lambert, Kim Loong Lim, Jessica Ogden, Janaina Videira Pinto, Michael Troup, and Azra Zamri.

In 2013 the program also welcomed three international and three domestic students. They were: Adekunle Bademosi, Samuel Fynes-Clayton, John Jordan, Muthmainah, Ozlem Yetim, and Diana Zanfirache.

Students completing their Master of Neuroscience program say that the experience has encouraged them to pursue further study opportunities, such as PhDs.

Compulsory core lecture-based courses in the Master of Neuroscience program are:

- ▮ Systems Neuroscience: Sensory and Motor (NEUR7004), which uses a systems approach to explore the brain with respect to circuits that integrate and process information.
- ▮ Cognitive and Behavioural Neuroscience (NEUR7005), which focusses on the elucidation of the neural basis of cognitive and behavioural phenomena.
- ▮ Molecular and Cellular Neuroscience (NEUR7006), which is concerned with cellular and molecular biology of the neuron.

Together with the three Master of Neuroscience laboratory rotations, which offer 300 hours of supervised practical experience; these courses provide a cohesive introduction to the theoretical and practical aspects of neuroscience. Rotations can be undertaken in a wide number of participating schools, including QBI, UQ's Schools of Psychology, Pharmacy, Medicine, Biomedical Sciences, Microbial and Molecular Biosciences, Information Technology and Electrical Engineering, the Perinatal Research Centre, Centre for Clinical Research, the Institute for Molecular Bioscience, the Centre for Advanced Imaging and the QIMR Berghofer Medical Research Institute.



COMMUNITY



Community

QBI's goal is to make a positive impact on the Australian community by helping to reduce the huge social and financial cost of neurological and mental illness.

In 2013, QBI hosted a series of high profile events and conducted a range of community outreach events. In addition to educating Australians about the latest research findings, staff also continued their efforts to encourage the next generation to consider careers in neuroscience.



Left: MND Breakfast held at QBI.

LECTURES

PETER GOODENOUGH LECTURE

Killer Viruses and Killer Cells

This year's Peter Goodenough Lecture was delivered by Nobel Laureate Professor and former Australian of the Year, Peter Doherty.

On June 18, Professor Doherty shared his insights into his work with immunity and killer viruses, and the invaluable role philanthropy plays in funding the research that leads to the development of drugs to combat infection.

Professor Doherty trained as a veterinarian, spent a decade researching infectious diseases of domestic animals and has, for more than 40 years, been involved in basic biomedical research.

He and his Swiss colleague Rolf Zinkernagel shared the 1996 Nobel Prize for Physiology or Medicine for the discovery of how the immune system recognises virus-infected cells.

The Peter Goodenough annual lecture is named in honour of the late Mr Peter Goodenough (1936–2004), a QBI benefactor, whose personal battle with motor neuron disease led to a bequest to fund fundamental scientific research.

MERSON LECTURE

Cortical Interneurons, Creation, Wiring and Disease

On November 7, New York University's (NYU) Professor Gordon Fishell, addressed guests at QBI's fifth annual Merson Lecture.

The Julius Raynes Professor of Neuroscience and Physiology at the NYU School of Medicine and Associate Director of the NYU Neuroscience Institute, shared his insights into wiring and disease in the nervous system.

Professor Fishell's interest is with the inhibitory cells that, he explained, act as the yin to the excitatory cells' yang.

Excitatory cells carry information and allow an external world to be internalised and represented in the brain.

"The inhibitory cells, the cortical interneurons, keep the excitatory cells in check and prevent the networks from overloading and causing problems such as epilepsy or schizophrenia," he said.

Professor Fishell explores the developmental events by which these cells acquire their identities, and discovered that the very electrical signals that ultimately allow one to perceive the world are the same ones that are used to assemble the nervous system.

Further, not all dysfunction results in brain disease. "A false dichotomy is that disease can be caused by genes or environment, while in fact the two are interlinked. Environment affects gene expression and genes affect environment," he said.

The Merson Lecture is named in honour of Dr David Merson, member of the QBI Advisory Board, whose philanthropic sponsorship of this lecture is indicative of a strong community interest in neuroscience.



L-R: Dr David Merson, Professor Gordon Fishell and Professor Perry Bartlett



Attendees at the UQ Fellows of the Australian Learned Academies Lecture 2013.

EVENTS

‘BEATING OF THE DRUMS’ CEREMONY

To celebrate the arrival of traditional Papua New Guinean slit-drums to UQ, as well as QBI’s advances in motor neuron disease (MND) research, a ‘beating of the drums’ ceremony was held on June 3, with the Institute drum’s journey beginning at the UQ Anthropology Museum and ending at QBI.

Known as a garamut, these ancient artifacts are used as a voice in villages to announce meetings, call individuals, and issue warnings over long distances.

The skill of making slit-drums has been the focus of PhD student Alphonse Aime, the recipient of The Peter Goodenough and Wantoks PhD Scholarship in Anthropology, established in memory of Mr Peter Goodenough, who had extensive business interests and friends in Papua New Guinea.

Mr Goodenough was sadly diagnosed with motor neuron disease and died in 2004, leaving a major bequest to QBI to further invest in research to treat this neurological disease.

Mr Aime is encouraging Papua New Guineans to preserve their rich cultural heritage by passing on the traditional knowledge and skills of garamut-making to young people.

At present, the average age of garamut carvers in Papua New Guinea is 50, with few young people interested in the art.

Mr Aime’s research has triggered a renewed interest among the people to hold onto their fast disappearing indigenous cultural knowledge and skills.

Two garamuts were acquired, through Mr Aime, from a village in the Madang Province of Papua New Guinea, and are housed separately in the UQ Anthropology Museum and QBI.

The ‘beating of the drums’ not only celebrated the arrival of the garamuts at UQ, but also symbolised QBI’s advances into ‘beating’ motor neuron disease through ongoing intensive research.

SYMPOSIUM ON BRAINNETOME MEETS GENOME

The Second Symposium on Brainnetome Meets Genome (SBMG 2013) was held in Beijing, China, from 12-13 September.

Hosted by the Joint Sino-Australian Laboratory of Brainnetome, the symposium brought together researchers from neuroimaging, network neuroscience, neuroscience methodology and experimental neuroscience with a special interest in understanding the relationships between brain networks, function and malfunction, and risk genes of neuropsychiatric and neurological diseases.

Themes of the scientific sessions of the symposium included anatomical and structural brain networks, brainnetome methods and applications, how genes modulate brain networks, and clinical applications of brain networks.

Time was also dedicated to a discussion of the major challenges in the field and how to address them through integrating disciplines and multinational organisations.

7th ALZHEIMER’S AND PARKINSON’S DISEASE SYMPOSIUM

On September 23, QBI was host to more than 175 researchers from throughout Australia and across the globe for the 7th Alzheimer’s and Parkinson’s Disease (A+PD) Symposium.

The event featured two mornings of speaker presentations followed by two consecutive workshops, one on Alzheimer’s disease and the second on frontotemporal dementia and motor neuron disease.

The impressive line-up of speakers included a Keynote Lecture from the University of Melbourne’s Professor Ashley Bush, who has conducted ground-breaking work in the diagnosis and treatment of Alzheimer’s disease.

International Keynote Lectures were given by Professors Christian Behl (University of Mainz), Christian Haass (German Center for Neurodegenerative Diseases, Munich) and Angus Nairn (Yale University).

Organised by Professor Jürgen Götz (QBI) and Professor Lars Ittner (The University of New South Wales), the event now alternates on an annual basis between QBI and UNSW.



Traditional Papua New Guinean slit-drum, known as the garamut.

COMMUNITY OUTREACH

QBI's community outreach program is designed to engage people interested in discovering more about neurological disorders. The program's success is proof of the public's thirst to learn more about the latest developments in this area of research.

In addition to regular tours through QBI's world-class facilities, the Institute's researchers frequently conduct lectures, talks and discussions that are the anchor of the outreach program. This interaction has continually proven beneficial for the public and scientists alike.

As part of this community outreach program, QBI held a number of events designed to celebrate the support both individuals and community groups have provided towards the Institute's research.

NEW FELLOWSHIPS

The charitable arm of the Freemasons Queensland, the Board of Benevolence of Aged Masons, Widows and Orphans' Fund, and UQef, a private fund established by Wotif.com founders Andrew Brice and Graeme Wood, have established a \$1.4 million fund to support a Queensland Freemasons Senior Research Fellowship in Learning and Memory at QBI.

The Fellowship was formally recognised by the Honourable Ian Walker MP, Minister for Science, Information Technology, Innovation and the Arts at a celebratory event in Sandgate.

Also recognised at this event was the Peter Hilton Research Fellowship in Ageing Dementia, which was established in honour of the late Peter Hilton by his wife Robyn.

The \$500,000 Fellowship will support an outstanding early-career researcher for five years based at the Clem Jones Centre for Ageing Dementia Research (CJCADR).

The primary role of the Peter Hilton Research Fellow will be to explore the interface between biological and physical research in memory and learning, how these functions are disrupted in dementia, and develop procedures to test and manage these dysfunctions.

BREAKFAST SERIES

The introduction of the QBI Breakfast Series in 2013 enabled meaningful engagement with our donors, corporates and community groups.

Topics covered throughout the period included:

- | *Parkinson's and deep brain stimulation*
Professor Pankaj Sah and
Professor Peter Silburn
- | *Discovering the causes of and treatments for brain cancer*
Professor Linda Richards and Mr Geoff Osborne
- | *What is a stroke and how does it affect the brain and behaviour?*
Professor Jason Mattingley and
Ms Lavinia Codd
- | *Ageing and dementia: Delaying and reversing cognitive decline*
Professor Jürgen Götz and Associate
Professor Elizabeth Coulson
- | *Mental illness: Is it nature or nurture?*
Professor John McGrath and
Associate Professor Naomi Wray
- | *MND: Uncovering the genetic causes*
Professor Pam McCombe and
Dr Marie Mangelsdorf

AUSTRALIAN BRAIN BEE CHALLENGE

out-smart, out-think, out-last

The Australian Brain Bee Challenge (ABBC) is QBI's major public outreach program. Each year the ABBC encourages high school students to learn about neuroscience. The program aims to capture students' interest in brain structure, function and anatomy and to educate students, teachers and the wider community about the importance of neuroscience research to society. The ABBC is Australia's only neuroscience competition for high school students and provides opportunities for students from all over Australia, including regional areas, to participate and consider a career in science and in particular, neuroscience.

2013 was another successful year for the ABBC, with Brisbane student and 2012 ABBC Champion, Jackson Huang, becoming the second Australian student to win the International Brain Bee (IBB) Championship. The IBB was held in Vienna, Austria, in September where Jackson competed against students from 18 countries and completed five neuroscience challenges to become the International Winner. Jackson followed in the footsteps of 2011 ABBC Champion Teresa Tang, who also went on to win the IBB in 2012.

The ABBC has three rounds, with Round 1 taking place during the annual Brain Awareness Week in March. Round 1 of the ABBC is an online quiz in which students have to demonstrate their knowledge and understanding of brain structure, function, anatomy, neurological diseases and disorders. In 2013, there were 5,550 Year 10 student participants in Round 1, taking the total number of participants to nearly 30,000 students over the 8 years since the ABBC was introduced in Australia.

The Round 2 Queensland event took place at QBI in June and was attended by 200 Year 10 students and teachers from across Queensland. The full-day competition was complemented with an impressive speaking program featuring

Nobel Prize winner and Laureate Professor Peter Doherty and Dr Chelsea Bond from UQ's Aboriginal and Torres Strait Island Studies Unit. In addition to competing in the competition, students and teachers had the opportunity to tour the facilities at QBI, participate in experiments and hear from QBI scientists discussing their research, discoveries and how they became involved in science research as a career. Somerville House student Eva Wang was the winning student on the day, becoming the 2013 Queensland Brain Bee Challenge Champion.

Round 3, the National Final, in which each State Champion competes to become the Australian Brain Bee Champion, is held annually at the Australasian Neuroscience Society (ANS) meeting and in 2014 will be in Adelaide.

In 2013, the ABBC and QBI also hosted a number of ABBC participants for work experience. Students came from across Queensland and had the opportunity to spend a week at QBI experiencing what it is like to be a neuroscientist. The ABBC also continued their collaboration with indigenous organisations including the Queensland Aboriginal and Torres Strait Islander Foundation, and Generation One to ensure the competition is accessible to indigenous students and to encourage involvement. ABBC Founder, Professor Linda Richards, was awarded the 2013 Vice-Chancellor's Equity and Diversity Award for her work in this area.

With neurological and mental illness accounting for almost half of the total disease burden in Australia, the ABBC is one way that we can encourage students to be interested in neuroscience research and to join QBI in finding treatments and cures for disease. The ABBC is continuing to develop meaningful ways to engage students, teachers and the community, and will continue to grow in 2014.



Above: Eva Wang, ABBC Champion.

RECOGNITION



Recognition

QBI is home to more than 300 dedicated researchers working to discover the cellular and molecular mechanisms that underlie the ability of the adult brain to generate new nerve cells and form functional connections.

QBI researchers represent the Institute on a number of pivotal scientific organisations and serve on prestigious editorial boards. QBI's track record in terms of publications, grants and awards further attests to the high standard of research being undertaken at the Institute.

FELLOWSHIPS AND AWARDS

Australian Research Council

Discovery Early Career Researcher Awards

Dr Marta Garrido and Dr Hong Lee were successful in obtaining funding in the second round of the ARC's Discovery Early Career Researcher Award (DECRA) scheme.

Dr Garrido's research program will build on her ground-breaking work in the application of brain imaging methods, including electroencephalography (EEG) and functional magnetic resonance imaging (fMRI), for modelling large-scale brain networks in humans. Her key goal is to understand the role of uncertainty in human behaviour and brain function. This will provide a deeper knowledge of the brain mechanisms and structural elements that contribute to the way in which uncertainty shapes decision making, and provide insight into disorders that are linked to intolerance of uncertainty.

Dr Hong Lee's project will establish new theories and statistical models to estimate the genome pleiotropy (the phenomenon whereby genes exert effects on multiple traits) between complex traits. By applying a novel design using multiple independent genome-wide association studies to provide an unbiased estimation of genome pleiotropy not affected by uncontrolled environmental effects, Dr Lee will be able to address important biological questions such as whether human cognition is genetically identical across genders or different population groups.

National Health and Medical Research Council

John Cade Fellowship in Mental Health Research

In a major achievement, Professor John McGrath became one of only two national recipients of a prestigious NHMRC John Cade Fellowship in Mental Health Research. Professor McGrath and his close colleagues Associate Professors Darryl Eyles and Tom Burne are renowned for their research showing that low levels of vitamin D during early life pose a risk for the later development of schizophrenia. The John Cade Fellowship will enable Professor McGrath to explore further the association between early life vitamin D and childhood brain development, as well as subsequent mental health outcomes. It will also allow clinical trials to be established in Queensland and will promote the training of junior psychiatrists in clinical neuroscience. Psychiatric disorders are a key area of QBI's research program, and it is noteworthy that Professor McGrath's proposal also includes new collaborations with three other QBI faculty, Professor Peter Visscher, Professor Pankaj Sah and Associate Professor Naomi Wray, building additional capacity in cross-disciplinary mental health research.

Early Career Fellowships

As part of his CJ Martin Overseas Biomedical Fellowship, Dr Enda Byrne is spending two years in the laboratory of Professor Hakon Hakonarson at the Centre of Applied Genomics at the Children's Hospital of Philadelphia Research Institute. Understanding the genetic aetiology of complex

disorders such as type 1 diabetes, cancer and schizophrenia offers hope of developing new therapies and preventative measures. The goal of Dr Byrne's proposal is to develop new statistical methodologies for analysing genomic data, and to apply these and existing methods to the very large cohorts available at the Children's Hospital of Philadelphia. His primary interest is in the genetic underpinnings of psychiatric disorders, and he will use his new methods to analyse schizophrenia, autism and attention deficit hyperactivity disorder datasets.

Dr Ben Sivyver was also the recipient of a CJ Martin Overseas Biomedical Fellowship, the first two years of which are being undertaken in the laboratory of Dr Gabe Murphy at the Janelia Farm Research Campus of the Howard Hughes Medical Institute in Virginia. There are many visual areas in the brain, and these are interconnected in a complex fashion. Determining how these different streams interact to process visual information is critical to our understanding of vision. During the US phase of his fellowship, Dr Sivyver will focus on a brain region known as the superior colliculus. This area is of particular interest because it transforms multimodal sensory information into a coordinated representation of the external world, enabling head and eye movements to be directed towards behaviourally relevant stimuli.

The University of Queensland

Vice-Chancellor's Equity and Diversity Award

Professor Linda Richards was the recipient of the 2013 UQ Vice-Chancellor's Equity and Diversity Award as testament to her outstanding leadership in developing the Australian Brain Bee Challenge

(ABBC), and for her unflagging commitment to ensuring that this competition provides equal opportunities to those in regional centres as well as students from an indigenous background. Professor Richards has been passionate in her efforts to make the ABBC more inclusive, drawing in students from beyond the major urban centres, and introducing them, their teachers and their families to neuroscience research on a major university campus. More recently she has also collaborated with indigenous organisations including the Queensland Aboriginal and Torres Strait Islander Foundation and GenerationOne, who, through financial support and awareness programs, have ensured that the competition is more accessible to indigenous students. This is an exciting development in raising the diversity of the ABBC, which is also being enhanced by the implementation of a work experience program for indigenous students with an ongoing interest in science.

UQ Foundation Research Excellence Award

Dr Jian Yang's research excellence and leadership potential was acknowledged with a UQ Foundation Research Excellence Award. Dr Yang will use this award to develop advanced statistical methods to quantify the overall contributions of all DNA variants (both common and rare single nucleotide polymorphisms or SNPs) to motor neuron disease, in order to test the hypothesis that rare SNPs explain a large proportion of the heritability of this disease. A software tool that implements the methods developed in this project will also be made freely available to the wider scientific community for research into other diseases of interest.

COMMERCIALISATION

Despite a restructure of the Institute's commercialisation team during the year, QBI continued to discover and develop new methods for treating, preventing and diagnosing neurological diseases, as well as develop strategies for applying biologically-inspired mechanisms to machines.

QBI continues to maintain its strategic relationship with UniQuest, the main commercialisation company of The University of Queensland, providing access to commercialisation expertise, processes and resources. UniQuest is recognised as an Australian leader in research commercialisation.

2013 saw a restructure within UniQuest, replacing the hub-and-spoke model with a more versatile approach. UniQuest's new model will be of great benefit to QBI as its application of brain research diversifies from disease and mental illness to mechanical applications and learning, with the Australian Research Council's award of the Special Research Initiative Science of Learning Research Centre. Moving forward, QBI's commercialisation activities will continue to be overseen by the Intellectual Property and Translational Committee.

Clem Jones Centre for Ageing and Dementia Research Director, Professor Jürgen Götz, made a significant finding this year that could revolutionise the future treatment of dementia sufferers, particularly those affected by amyloid plaques. In work still to be published Professor Götz has managed to clear a significant amount of amyloid plaques in an animal model of dementia. A patent application is currently being prepared for this technology.

In 2013, QBI, in partnership with Boeing Research & Technology - Australia and the University of Newcastle, was the recipient of an Australian Research Council Linkage Project grant. The project, "Strategies for mid-air collision avoidance in aircraft: lessons from bird flight" will study the mechanisms by which birds successfully avoid collisions, even in confined places such as aviaries, and seek to apply some of these strategies to collision avoidance in unmanned aircraft. This research partnership may one day lead to Professor Mandyam Srinivasan's research literally guiding the way in aircraft navigation throughout the world. Professor Srinivasan's research first came to Boeing's attention in 2010 and the collaboration has been building steadily since then.

Left: Professor Mandyam Srinivasan's laboratory is among those working together with commercial partners.

PUBLICATIONS

QBI researchers (noted in bold) have contributed to the following publications. Some publications appeared as electronic publications ahead of print. These are signalled as “epub 2012 print 2013”. Publications that appeared as electronic publications in 2013 and print in 2014 are signalled as “epub 2013 print 2014”. Any publications omitted from this report will appear in the 2014 Annual Report.

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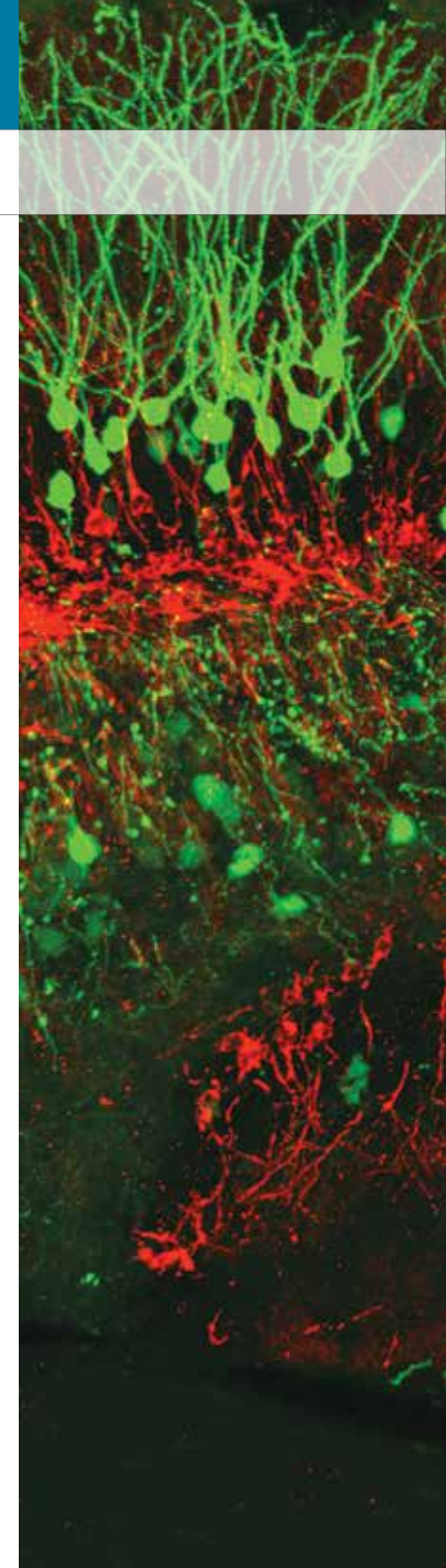
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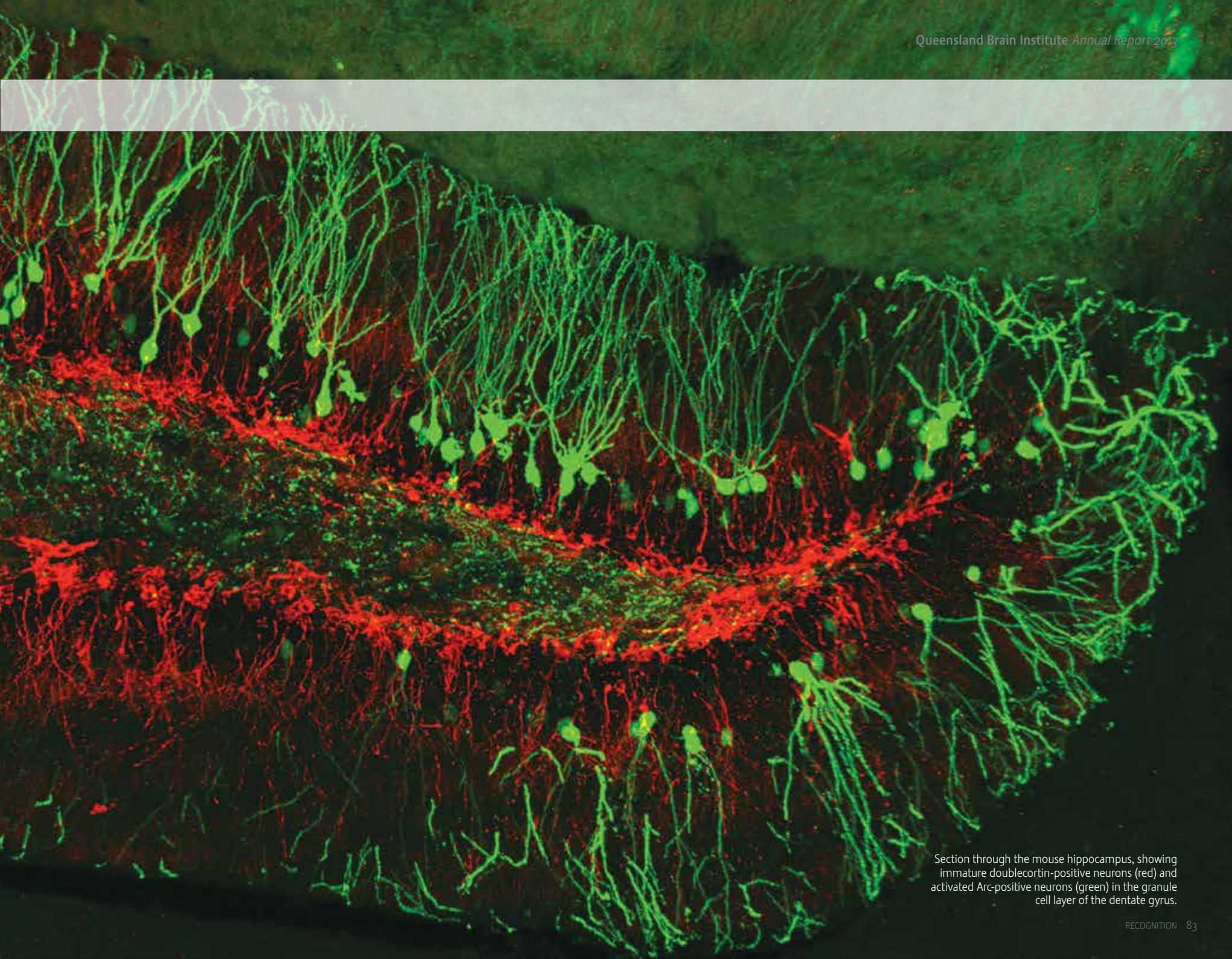
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- Zhou J, Wen Y, She L, Sui YN, Liu L, **Richards LJ**, Poo MM (2013) Axon position within the corpus callosum determines contralateral cortical projection. *Proceedings of the National Academy of Sciences of the USA* 110: E2714-E2723
- Zhu Y, Li Z, **Chen M**, **Cooper HM**, Lu GQM, Xu ZP (2013) One-pot preparation of highly fluorescent cadmium telluride/cadmium sulfide quantum dots under neutral-pH condition for biological applications. *Journal of Colloid and Interface Science* 390: 3-10 epub 2012 print 2013
- Zhu Y, Li Z, **Chen M**, **Cooper HM**, Xu ZP (2013) Tuning core-shell SiO₂@CdTe@SiO₂ fluorescent nanoparticles for cell labeling. *Journal of Materials Chemistry B* 1: 2315-2323
- ### Book Chapters
- Blackmore DG**, Rietze RL (2013) Distribution of neural precursor cells in the adult mouse brain. In *Methods in Molecular Biology*, eds. B. A. Reynolds, L. P. Deleyrolle, 183-194. New York, United States: Humana Press [Protocol]
- Jhaveri DJ**, **Prosper BW**, **Bartlett PF** (2013) Culturing and expansion of precursor cells from the adult hippocampus. In *Methods in Molecular Biology*, eds. B. A. Reynolds, L. P. Deleyrolle, 41-51. New York, United States: Humana Press [Protocol]
- Osborne GW** (2013). Flow cytometry of neural cells. In *Methods in Molecular Biology*, eds. B. A. Reynolds, L. P. Deleyrolle, 135-44. New York, United States: Humana Press [Protocol]
- Yang J, **Lee SH**, Goddard ME, **Visscher PM** (2013). Genome-wide complex trait analysis (GCTA): methods, data analyses, and interpretations. In *Methods in Molecular Biology*, eds. C. Gondro, J. van der Werf, B. Hayes, 215-236. New York, United States: Humana Press
- ### Refereed Conference Proceedings
- Baker S**, **Soccol D**, Postula A, **Srinivasan MV** (2013) Passive landing gear using coupled mechanical design. *Australasian Conference on Robotics and Automation*. University of New South Wales, Australia
- Bossomaier T, Delaney AJ, **Crane J**, Gobet F, Harre M (2013) The neurological scaling of human expertise. *Cognitive The Fifth International Conference on Advanced Cognitive Technologies and Applications*. Valencia, Spain
- Ng KB**, Bradley AP, **Cunnington R** (2013) Effect of posterized naturalistic stimuli on SSVEP-based BCI. *Annual International Conference of the IEEE Engineering in Medicine and Biology Society*. Osaka, Japan
- Strydom R, **Thurrowgood S**, **Srinivasan MV** (2013) Airborne vision system for the detection of moving objects. *Australasian Conference on Robotics and Automation*. University of New South Wales, Australia
- Thiruvurudchelvan V, **Crane JW**, Bossomaier T (2013) Analysis of spikeprop convergence with alternative spike response functions. *IEEE Symposium on Foundations of Computational Intelligence*. Singapore
- Wang AG, Kurniawati H, Singh S, **Srinivasan M** (2013) Animal locomotion *in silico*: a POMDP-based tool to study mid-air collision avoidance strategies in flying animals. *Australasian Conference on Robotics and Automation*. University of New South Wales, Australia





Section through the mouse hippocampus, showing immature doublecortin-positive neurons (red) and activated Arc-positive neurons (green) in the granule cell layer of the dentate gyrus.

GRANTS

The following contains information on national and international competitive funding for grants and fellowships that received funding starting in 2013; GST and other yearly increments are not included in the amounts shown. Internal grants and fellowships awarded by The University of Queensland have been included this year.

Alzheimer's Australia Dementia Research Foundation

Alzheimer's Australia Dementia Research Grant

Matamalas M - Axonal transport defects in Alzheimer's disease and related disorders: mechanisms of tau pathology, \$50,000, 1 year

Australian Government

Department of Foreign Affairs and Trade (Australia), Australia-Indonesia Institute Grants

Dean A, Kleine D - Selamat Datang di CoralWatch - Supporting marine science education in Indonesia via internet-based, interactive resources, \$20,108, 1 year

Australian Government Cooperative Research Centres (CRC)

Whitehouse A, Carrington S, Falkmer T, Dissanayake C, **Claudianos C**, Eapen V, Ashburner J, Sofronoff K, Roberts J, Richdale A, Sofronoff K, Lennox N - CRC for Living with Autism Spectrum Disorders, \$31,000,000, 8 years [Managed by Sylvia Rodger, Director of Research and Education]

Australian Research Council

ARC Discovery Early Career Researcher Awards

Garrido M - Neurobiological mechanisms of decision under uncertainty, \$375,000, 3 years

Lee SH - Novel statistical algorithms and methods to quantify and partition pleiotropy between complex traits in populations, \$375,000, 3 years

Yu X - Ultra-fast functional magnetic resonance imaging (fMRI) mapping of layer-specific interhemispheric plasticity, \$375,000, 3 years [relinquished]

ARC Discovery Projects

Goddard M, **Visscher P** - The genetic architecture and evolution of quantitative traits, \$270,000, 3 years [awarded to and administered by UQ's Diamantina Institute]

Götz J - Nuclear functions of the microtubule-associated protein tau, \$300,000, 3 years

Hayes I, Jones C, **Colvin R**, Meinicke L - Understanding concurrent programs using rely-guarantee thinking, \$305,000, 3 years [awarded to and administered by UQ's School of Information Technology and Electrical Engineering]

Lynch J - The molecular basis of ionic selectivity in nicotinic-type ion channel receptors, \$350,000, 3 years

Sah P - Neural circuits that mediate fear conditioning and extinction, \$600,000, 3 years

Williams S - Dendritic information processing during sensory-motor behaviour, \$441,000, 3 years

Yang J, Visscher P, Goodard M - Estimation of non-additive genetic variance for complex traits using genome-wide single nucleotide polymorphisms and sequence data, \$309,000, 3 years [awarded to UQ's Diamantina Institute but now administered by QBI]

ARC Linkage Infrastructure, Equipment and Facilities Grants

Meunier F, Goodhill G, Kaunanithi S, Yap A, Drennan J, Mackay-Sim A, Wei M, Avery V, Rubinsztein-Dunlop H - Live molecular imaging using super resolution microscopy, two photon and spinning disk confocal microscopy, \$800,000, 1 year

Reutens D, Watson C, Paxinos G, **Götz J**, Meikle S, Bernhardt P, Mardon K, Dodd P, Banati R - A facility for ex-vivo molecular imaging in cells and tissues, \$310,000, 1 year [awarded to and administered by UQ's Centre for Advanced Imaging]

ARC Linkage Project

Srinivasan M, Williams B, Perez T - Strategies for mid-air collision avoidance in aircraft: lessons from bird flight, \$652,374, 3 years

ARC Special Research Initiative

Ottmar L, John H, Timms M, **Sah, P, Jiang T, Mattingley J, Cunningham R**, Dux P, Reutens D, Hester R, Gos M, Carroll A, Clarke D, Gillies R, Kennedy G, Tayler C, Koo S-T, Westwell M, Tytler R, **Bredy T**, Thomson S, Pegg J, Griffin P, Silburn S, Lockyer L - Science of Learning Research Centre, \$16,000,000, 4 years

Human Frontier Science Program Organisation

Research Grant Program

Altshuler D, Lentink D, **Srinivasan M** - Visual control of flight modes and transitions in birds, \$1,050,000, 3 years

Motor Neurone Disease Research Institute of Australia Inc

Research Grant

Henderson R, McCombe P, Rose S, **Wallace R** - Use of biomarkers to understand Amyotrophic Lateral Sclerosis, \$100,000, 1 year [awarded to and administered by UQ's Centre for Clinical Research]

National Health and Medical Research Council

NHMRC Career Development Fellowships

Yang J - Dissecting genetic variation for human complex diseases and traits, \$397,724, 4 years [awarded to UQ's Diamantina Institute but now administered by QBI]

NHMRC Early Career Fellowships

Byrne E - Functional organisation of visual inputs to the superior colliculus, \$299,564, 4 years

Sivyer B - Functional organisation of visual inputs to the superior colliculus, \$299,564, 4 years

NHMRC John Cade Fellowships in Mental Health Research

McGrath J - Modifiable risk factors for serious mental illness - an integrated program of epidemiology, genetics and clinical trials, \$3,750,000, 5 years

NHMRC Program Grant

Halliday G, **Götz J**, Ittner L, Kril J, Hodges J, Kiernan M - Frontotemporal dementia and motor neurodegenerative syndromes, \$11,011,390, 5 years [awarded to and administered by the University of NSW]

NHMRC Project Grants

Bartlett P, Jhaveri D - Defining the function of two discrete precursor cell populations in the adult hippocampus: potential for the treatment of cognitive and mood disorders, \$559,003, 3 years

Bredy T - MicroRNA regulation of fear-related memory, \$473,222, 3 years

Collins B, Teasdale R, **Coulson E**, King G - Understanding how membrane trafficking controls the levels of Alzheimer's disease causing Aβ peptides, \$152,262, 1 year [awarded to and administered by UQ's Institute for Molecular Bioscience]

Coulson E, Bellingham M - Sleep disturbance and cholinergic dysfunction in Alzheimer's disease, \$824,641, 4 years

Eyles D, McGrath J, Burne T - Early pharmacological intervention in an animal model of schizophrenia, \$424,139, 3 years

Goodhill G, Scott E - Combining timelapse imaging and computational modelling to understand the mechanisms of axon guidance in the developing retinotectal system, \$423,647, 3 years

Martin N, **McGrath J**, White M - Exploring modifiable candidate risk factors for mental illness in young adults: Infection, vitamin D and stress, \$827,611, 3 years [awarded to and administered by Queensland Institute of Medical Research]

Meunier F, Collins B - Unravelling Munc18 dual function in exocytosis, \$497,031, 3 years

Richards L - Cellular and molecular regulation of interhemispheric fusion, \$433,889, 3 years

Richards L - Guidance mechanisms regulating the development of axonal projections from the cingulate cortex, \$467,522, 2 years

Scott J, **McGrath J**, Najman J, Alati R, Mamun A, Clavarino A - The outcomes of adolescents and young adults who experience hallucinations: A birth cohort study, \$603,862, 3 years [awarded to and administered by UQ's Centre for Clinical Research]

Visscher P - Exploiting SNP data in epidemiology and genetics through multivariate analysis of complex traits, \$460,518, 3 years [awarded to and administered by UQ's Diamantina Institute]

Visscher P, Montgomery G - CAGE: Consortium for the architecture of gene expression, \$484,191, 1 year [awarded to and administered by UQ's Diamantina Institute]

GRANTS

Wray N, Visscher P, Yang J - Estimation and partitioning of the still-missing heritability for complex disease, \$282,202, 3 years

Wray N, Lee SH, Mowry B - Statistical analyses of whole genome genotype data to better understand psychiatric disorders, \$525,405, 3 years

National Institutes of Health (USA)

NIH Ro1 [subcontract]

Visscher P, Keller M (US lead) - Estimating the frequencies and population specificities of risk alleles, \$388,000, 5 years [NIH subcontract awarded to UQ's Diamantina Institute and administered by the University of Colorado and UQ's Diamantina Institute]

PADI Foundation

Dean A - Improving our understanding of coral bleaching using CoralWatch, \$8,550, 2 years

Queensland Department of Development and Innovation

Queensland Department of Development and Innovation Smart Futures Fund - QCAS Biotechnology Projects Fund

Chen C, Esteban M, McCombe P, **Wallace R** - A model for understanding and treating amyotrophic lateral sclerosis using induced pluripotent stem cells, \$140,000, 4 years [awarded to and administered by UQ's School of Biomedical Sciences]

Swiss National Science Foundation Candoc Fellowship

Cortesi F - Swiss National Science Foundation Candoc Fellowship, \$53,000, 1 year

Travel Grants

Australian Flow Cytometry Group Conference Travel Grant

Osborne G - Travel grant for AFCG conference in New Zealand, \$800, 1 year

Contributing to Australian Scholarship and Science

Anggono V - PICK1 interacts with syndapin to regulate AMPA receptor internalisation and cerebellar long-term depression, \$2,000, 1 year

Ian Potter Foundation Travel Grant

Wei W - Ian Potter Foundation Travel Grant, \$1,200, 1 year

Widagdo J - Ian Potter Foundation Travel Grant, \$1,500, 1 year

International Brain Research Organization Travel Grant

Martin S - International Brain Research Organization Travel Grant, \$2,094, 1 year

International Symposium on Chromaffin Cell Biology

Martin S - International Symposium on Chromaffin Cell Biology, \$977, 1 year

Motor Neurone Disease (Victoria)

Mangelsdorf M - The Nina Buscombe Award 2013, \$3,000, 1 year

The Queensland Emory Development Alliance Travel Bursary

Jenkins A, **Lynch J** - High throughput assay for biomarkers of and therapeutics for neurological disease, \$5,000, 1 year [awarded to and administered by Queensland Institute of Medical Research/UQ's QBI/Emory University]

UniQuest

UniQuest Pathfinder

Osborne G - Slice and dice sorting software, \$15,000, 1 year

The University of Queensland

UQ Early Career Researcher Grants

Cocchi L - Mapping changes in human neural networks associated with local cortical plasticity, \$30,000, 1 year

Sivyer B - Dendritic integration in direction-selective retinal ganglion cells, \$20,000, 1 year

UQ Foundation Research Excellence Awards

Yang J - Quantifying the overall contribution of all the DNA variants to motor neuron disease, \$70,000, 1 year

UQ Major Equipment and Infrastructure and NHMRC Equipment Grant

Goodhill G, Bredy T, Götz J, Cooper H, Hilliard M, Richards L, Coulson E, Scott E, Ruitenber M, Noakes P - Spinning disk confocal for advanced high-speed histocytometry and neuromorphology analysis, \$205,123, 1 year

King G, Alewood P, Capon R, Cooper M, Craik D, Fry B, Lewis R, **Lynch J**, Markovich D, Mobli M, Smith M, Sweet M - High throughput electrophysiology platform, \$342,000, 1 year [awarded to and administered by UQ's Institute for Molecular Bioscience]

Mattingley J, Dux P, Vanman E, Henry J, Nielsen M, Kritikos A, **Cunnington R**, Reutens D, Carroll T, Body R - A brain stimulation and portable eye-tracking suite for human behavioural research, \$96,427, 1 year [awarded to and administered by UQ's School of Psychology]

Porrello E, Phipps S, **Piper M, Eyles D**, Mazzone S, Wolvetang E, Key B, Walter T, Powell E, Thorn P, Smythe M, Moritz K, Launikonis B, Saunders N, Woodruff T, Borges K - Establishment of an integrated facility for single cell analysis, \$164,629, 1 year [awarded to and administered by UQ's School of Biomedical Sciences]

UQ Postdoctoral Research Fellowships

Garrido M - Neurobiological mechanisms of decision under uncertainty, \$311,305, 3 years [relinquished]

Tong W - Ras family small GTPases as Phosphatidylinositol 4, 5-Bisphosphate effectors regulate secretory vesicle release in endocrine cell and neuron, \$301,342, 3 years

UQ ResTeach

Colvin R - ResTeach, School of ITEE, \$28,308, 1 year

UQ Travel Awards for International Collaborative Research

Carleton K, **Marshall J** - UQ Travel Awards for International Collaborative Research Category 1, awarded return airfare from USA, 1 year

Féron F, **Eyles D** - UQ Travel Awards for International Collaborative Research Category 1, awarded return airfare from USA, 1 year

UQ Vice-Chancellor's Senior Research Fellowship

Sah P - Neural circuits that mediate learning and memory formation in the mammalian brain, funding for 3 years

Srinivasan M - From flying animals to airborne machines and back, funding for 3 years

NEUROSCIENCE SEMINARS

The Queensland Brain Institute conducts a weekly seminar program, which gives neuroscientists an opportunity to learn more about the latest scientific developments, often before research is published. The series is designed to challenge researchers in their thinking, promote excellence through the exchange of ideas and lead to future collaborations.

Dr Fabienne Alfonsi

Queensland Brain Institute, The University of Queensland
P75NTR has opposing roles in pre- and post-mitotic cells to regulate neuronal number

Dr Ehsan Arabzadeh

The John Curtin School of Medical Research,
Australian National University
*Neuronal encoding in rat whisker-barrel system
– from sensory representation to behaviour*

Professor Bernard Balleine

Brain & Mind Research Institute, The University of Sydney
Dysexecutive syndrome: The cortical- and thalamostriatal pathways and disorders of goal-directed action

Professor Bernhard Baune

Professor of Psychiatry, School of Medicine,
The University of Adelaide
Brain–inflammation interface: Molecular mechanisms and systems biology – novel diagnostic and therapeutic opportunities?

Dr Sophie Billa

Queensland Brain Institute, The University of Queensland
*Development of a new approach in preclinical research:
The touchscreen testing method for mice and rats*

Dr Stefan Bode

Decision Neuroscience Laboratory,
The University of Melbourne
Informing decision-making models by decoding patterns of brain activity

Professor Michael Breakspear

Queensland Institute of Medical Research
A phase transition in neonatal cortex during recovery from hypoxia

Dr Timothy Bredy

Queensland Brain Institute, The University of Queensland
*Characterising the role of the "6th" base:
How experience-dependent changes in DNA hydroxymethylation contribute to memory*

Dr Murray Cairns

Schizophrenia Research Institute
Genetic and environmental determinants of small non-coding RNA expression in neural development and schizophrenia

Associate Professor Tim Carroll

School of Human Movement Studies,
The University of Queensland
Multiple coordinate systems for new sensorimotor maps

Dr Adam Cole

Garvan Institute of Medical Research
GSK3 regulation of vesicle trafficking: Implications for neurotransmission and mood disorders

Associate Professor Lynn Dobrunz

Department of Neurobiology, University of Alabama at Birmingham, USA
Alterations in the excitation/inhibition balance in the hippocampus in an animal model of schizophrenia

Dr Mirella Dottori

Department of Anatomy and Neuroscience,
The University of Melbourne
Building the nervous system with pluripotent stem cells

Associate Professor Darryl Eyles

Queensland Brain Institute, The University of Queensland
The dopamine ontogeny hypothesis of schizophrenia

Associate Professor Geoff Faulkner

Mater Medical Research Institute
Single cell genomics reveals mosaic L1 retrotransposition in hippocampal neurons

Dr Marta Garrido

Queensland Brain Institute, The University of Queensland
Surprise induced changes in brain dynamics

Dr Ilan Gobijs

Queensland Brain Institute, The University of Queensland
Fgf8 signalling regulates interhemispheric fusion and corpus callosum formation

Dr Marcus Gray

Centre for Advanced Imaging, The University of Queensland
Neural correlates of the autonomically mediated integration of cognitive, affective and physiological activity

Professor Richard Gronostajski

State University of New York at Buffalo,
Department of Biochemistry
New York State Center of Excellence in Bioinformatics and Life Sciences, USA
Nuclear Factor I (NFI) transcription factors: A small family that does big things in brain development

Associate Professor Ulrike Grunert

Save Sight Institute, The University of Sydney
Parallel pathways in the primate visual system

Dr Martin How

Queensland Brain Institute, The University of Queensland
Polarisation vision in the marine environment

Professor Wieland Huttner

Max Planck Institute of Molecular Cell Biology and Genetics, Germany
Neural stem and progenitor cells and the evolution of the cerebral cortex

Professor Michael R. Ibbotson

National Vision Research Institute
Australian College of Optometry &
Department of Optometry and Vision Sciences, The University of Melbourne
How looking helps you see

Professor Allan Jones

Allen Institute for Brain Science, USA
Mapping gene expression and connections in the CNS: Tools and data from the Allen Institute for Brain Science

Dr Marc Kamke

Queensland Brain Institute, The University of Queensland
The influence of selective attention on plasticity induction in the human brain

Dr Hideaki Koizumi

Fellow and Corporate Officer, Hitachi Ltd. &
Director, Chairman of the Committee on Foreign Affairs, The Engineering Academy of Japan
Brain–science-based education and psychiatric diagnosis with optical topography

Professor Shu Li

Institute of Psychology, Chinese Academy of Sciences
Is risky choice actually guided by a compensatory rule? Converging evidence from eye-tracking and fMRI studies

Dr Divya Mehta

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The yin and the yang of post-traumatic stress disorder

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National Vision Research Institute
Visual stability in the primate visual-system

Professor Henrik Østergaard Mouritsen

Department of Biology and Environmental Sciences, University of Oldenburg, Germany
The magnetic compass of migratory birds: From behaviour to molecules and cognition

NEUROSCIENCE SEMINARS

Emeritus Professor Phillip Nagley

School of Biomedical Sciences, Monash University
Mitochondria and me: From yeast to mammalian cells and much neuroscience

Dr Angelique Paulk

Queensland Brain Institute, The University of Queensland
*Neural mechanisms of attention in the honeybee and the fly, *Drosophila melanogaster**

Dr Magreet Ridder

Queensland Brain Institute, The University of Queensland
Unraveling the pathophysiology of the white matter disorder MLC; Implications for white matter water homeostasis

Professor Marcello Rosa

Department of Physiology, Monash University
Anatomical and physiological organisation of the visual pathways following lesions of primary visual cortex in early life

Dr Martin Sale

Queensland Brain Institute, The University of Queensland
Enhancing neuroplasticity induction in human cortex – from observing actions to sleep rhythms

Professor Thomas Suddendorf

School of Psychology, The University of Queensland
Reflecting on reflection. The nature of visual self-recognition

Professor Stefan Thor

Department of Clinical and Experimental Medicine, Developmental Biology, Linköping University, Sweden
From neural stem cell to unique neuron: Controlling cell fate and cell number in the developing nervous system

Dr Fabrice Turpin

Queensland Brain Institute, The University of Queensland
NCC, the “kidney specific” sodium-chloride cotransporter is expressed in the brain and modulates fear extinction

Associate Professor Vidita Vaidya

Department of Biological Sciences, The Tata Institute of Fundamental Research, India
The right thing at the wrong time is the wrong thing: The ups and downs of Prozac

Professor Trichur Vidyasagar

Visual Neuroscience, The University of Melbourne
Reading into top-down mechanisms in visual attention: Neural synchrony to dyslexia

Professor Boyer Winters

Department of Psychology, University of Guelph, Canada
Investigating crossmodal cognition in rats using a new spontaneous object recognition paradigm

Dr Trent Woodruff

School of Biomedical Sciences, The University of Queensland
The opposing roles of innate immune complement proteins C3a and C5a in neurodegenerative disease

Professor Robert Zorec

Laboratory of Neuroendocrinology - Molecular Cell Physiology, Institute of Pathophysiology, Medical Faculty, University of Ljubljana, Slovenia
The fabrics of astrocyte vesicle traffic in health and disease



PROFESSIONAL SERVICE

In addition to playing a role in grant assessment for major national and international funding bodies, our staff provide the follow external professional service to the discipline.

Perry Bartlett

- SpinalCure Australia Director and Scientific Board Chairman
- Centre for Brain Research, University of Auckland, Scientific Advisory Board Member
- Garvan Institute of Medical Research, University of New South Wales, Scientific Appointments and Promotions Committee Member
- Motor Neurone Disease Research Institute of Australia, Research Committee Member
- NHMRC Research Translation Faculty Member
- Australia Israel Research Exchange Steering Committee Member
- Brainnetome Center, Institute of Automation, The Chinese Academy of Sciences, Beijing, International Advisory Committee Member
- Science of Learning Research Centre, Advisory Board Member
- Mater Medical Research Institute Limited, Board of Directors, Member

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- Fonds Nationale de la Recherche (Luxembourg) Grant Review
- Agence Nationale de la Recherche (France) Grant Review

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- Biological Psychiatry Australia, Committee Member
- NHMRC Grant Review Panel Member

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- Queensland Cyber Infrastructure Foundation, Architecture Consultant

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- Brisbane Chapter of the American Society for Neuroscience, Committee Member
- Australian Huntington's Disease Association, Queensland Branch, QBI Representative

Elizabeth Coulson

- Alzheimer's Australia Dementia Research Foundation Scientific and Medical Panel Member
- Australian Academy of Science Theo Murphy High Flyers Think Tank
- Australian Brain Bee Challenge, Northern Territory Coordinator
- Friedreich's Ataxia Research Association, Scientific Advisory Committee Member
- NHMRC Grant Review Panel Member

Ross Cunnington

- Australasian Cognitive Neuroscience Society, National President
- NHMRC Grant Review Panel Member

Darryl Eyles

- Biological Psychiatry Australia, Vice President
- NSW Brain Bank Network Scientific Review Committee Member

Geoffrey Goodhill

- NHMRC Grant Review Panel Member

Jürgen Götz

- NHMRC Grant Review Panel Member
- Member of the Alzheimer Research Forum
- Member of the DZNE (German Center for Neurodegenerative Diseases) Scientific Review Panel Member

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- Institute of Automation of the Chinese Academy of Sciences, Scientific Committee Member
- National Laboratory of Pattern Recognition, Scientific Committee Member
- Tianjing Key Laboratory of Brain Functional Imaging, Scientific Committee Chair

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- National Collaborative Research Infrastructure Strategy Imaging Facilities, Board Member
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- Australian Course in Advanced Neuroscience Scientific Program, Advisory Group Member
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- Australian Coral Reef Society, Past President and Council Member
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- ProjectAWARE, Honorary Board Member
- Great Barrier Reef Research Expeditions Advisory Board Member

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- Academy of Social Sciences in Australia, Panel D (Psychology, Social Medicine, Education) Committee Member
- Association for Attention and Performance, Advisory Council Member
- ARC Centre of Excellence for Cognition and its Disorders, Scientific Advisory Committee Member
- Australian Academy of Science National Committee for Brain and Mind Member
- NHMRC Grant Review Panel Member

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- Schizophrenia International Research Society, Board Member
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- Australian Schizophrenia Research Bank, Access Committee Member
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- NHMRC Australian Health Ethics Committee Member
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Frederic Meunier

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- Australian Schizophrenia Research Bank: Science Committee Member, Genetics Committee Member and Access Committee Member
- Royal Australian and New Zealand College of Psychiatrists, Member, Committee for Research

Michael Piper

- Australian Cell and Developmental Biology Society, Queensland Representative
- Brisbane Chapter of the Society for Neuroscience, Treasurer
- NHMRC Assigners' Academy Member
- Australasian Neuroscience Society, Queensland Representative

Judith Reinhard

- Australasian Association for Chemosensory Science Council Member
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- Agence Nationale de la Recherche (France) Grant Review
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- Australian Disorders of the Corpus Callosum (AusDoCC), Scientific Advisor
- Hunter Cell and Developmental Biology Meeting Committee Member
- International Brain Bee, Vice-President

Pankaj Sah

- Australian Course in Advanced Neuroscience, Course Management Committee Member
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- NHMRC Assigners' Academy Member
- NHMRC Career Development Fellowship Panel Member
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- Chinese Rheumatology Association, Vice President
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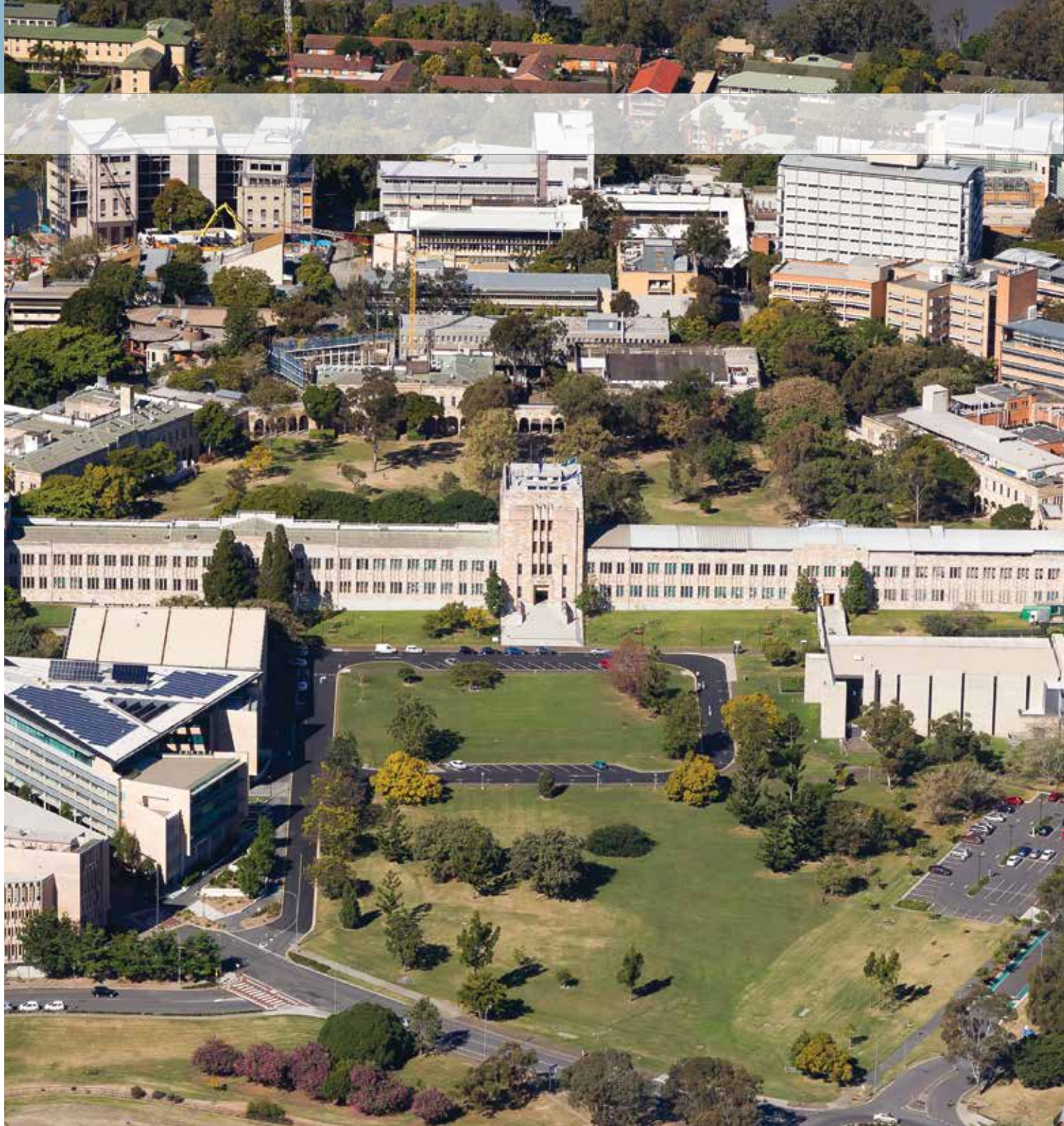
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