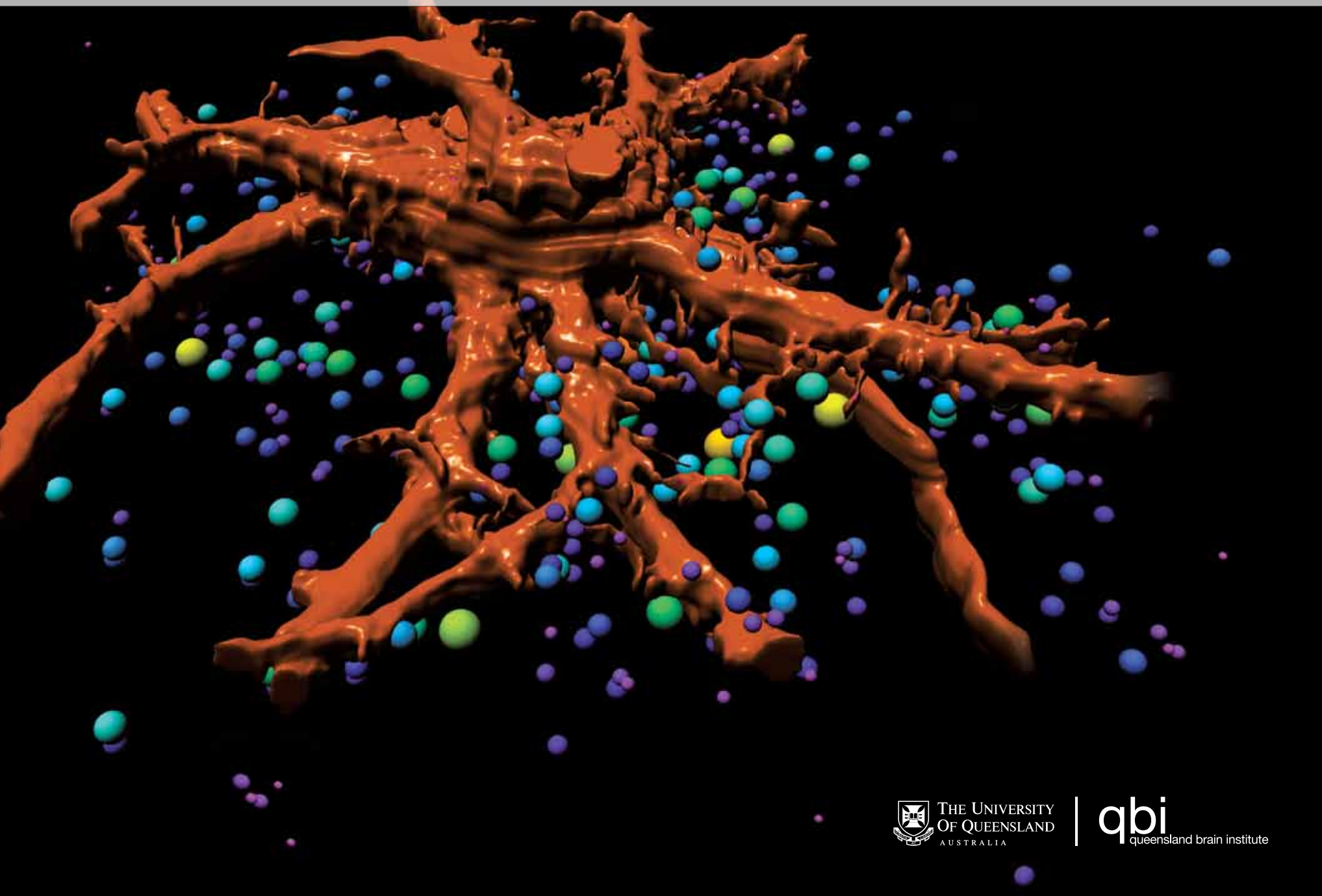




THE UNIVERSITY
OF QUEENSLAND
AUSTRALIA

| qbi
queensland brain institute

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Cover Image: Computational neuron. This image represents a single neuron (orange) surrounded by synaptic connections (spheres) of unlabelled and subsequently invisible neurons. Image: Luke Hammond, Refik Kanjhan and Matt Fogarty.

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Vice-Chancellor's Report



Since its inception in 2003, the Queensland Brain Institute (QBI) has emerged as one of The University of Queensland's flagship research facilities. With a growing team of almost 300 dedicated neuroscientists undertaking research under the guidance of 33 laboratory heads, the Institute covers a broad base of neuroscience over seven major themes. Their efforts are expected to yield multiple discoveries in the near future and thus pave the way for the development of new therapies and diagnostics to facilitate the treatment and management of mental and neurological disorders.

This optimistic outlook has been fully supported by independent reviews of the Institute's performance. The most recent, chaired by Professor Richard Faull, Director of the Centre for Brain Research with the University of Auckland's Faculty of Medical and Health Sciences, was the Septennial Review of QBI, required of all Institutes and Schools at UQ by the Academic Board. Among other things, it commended QBI for its quality, quantity and breadth of ongoing research, and for its number of noteworthy initiatives, including the Science of Learning Centre, the Centre for Advanced Imaging and the Centre for Ageing Dementia Research.

An earlier review of the Institute's performance, chaired by Dr Peter Riddles, noted that QBI had delivered outstanding outcomes to Queensland. These include scientific discoveries, the attraction of an internationally competitive faculty, establishment of cutting-edge experimental facilities, the mentorship and training of postgraduate students and postdoctoral scientists, engagement with the biotechnology and engineering industries, the generation of substantial economic returns on the Queensland Government's investment and strategic contributions to the development of the state through a knowledge-based society and economy.

As the major centre for neuroscience at The University of Queensland, QBI also helped the University to secure the top rating of five in neurosciences in the recent Excellence in Research for Australia (ERA) assessment.

This strong research base is supported by a wealth of international collaborations, including the Joint Sino-Australian Neurogenetics Laboratory in Shanghai and Joint Laboratory of Neuroscience and Cognition in Beijing. QBI also features a busy outreach program including the annual Australian Brain Bee Challenge (ABBC), a competition for high school students designed to stimulate, encourage and support their interest in neuroscience.

All in all, it has been another successful and productive year for QBI. I congratulate Perry and all staff and students on their efforts and accomplishments of the year and look forward with anticipation to an even brighter future.

A handwritten signature in dark ink, appearing to read 'Deborah Terry'.

Professor Deborah Terry
Vice-Chancellor



Director's Report



As the founding Director of the Queensland Brain Institute (QBI), my vision was to create an environment in which leading neuroscientists working across a range of sub-disciplines and animal models could interact and collaborate to discover the fundamental mechanisms regulating brain function in health and disease. My *raison d'être* for this was the belief that deeper understanding of these mechanisms would provide the insight required to develop novel therapeutic approaches to address the growing burden of neurological and mental illness in our community.

I am delighted to report this approach is bearing fruit as you will discover in reading the Annual Report. Among the highlights is the discovery of molecules regulating repair of nerve injury in the nematode, *C. elegans*, by Dr Massimo Hilliard's team. This discovery has taken us a few steps closer to understanding how we may be able to promote nervous system repair in humans. Meanwhile, Dr Tim Bredy's laboratory has discovered a previously unrecognised layer of gene regulation modulating fear extinction in mice, providing fresh understanding of how fear-related memories are formed, updated and extinguished at the molecular level. This new insight provides the basis for new

therapeutic approaches to tackle anxiety, phobias and post-traumatic stress disorders. Associate Professor Stephen Williams' work is unlocking the fundamental mechanisms by which synaptic connections between neurons in different parts of the brain form the basis for complex behaviour, findings that have ramifications in all aspects of health and disease. Associate Professor Frederic Meunier's team has uncovered how lipids regulate release of neurotransmitters, a finding that will have application in promoting improved neuronal function in a number of disease processes.

Also, the detailed study of human disease is leading to more fundamental understanding of the neural mechanisms involved: Professor Bryan Mowry's laboratory has been part of the largest genome-wide association study of schizophrenia ever undertaken, and is leading to a deeper understanding of the mechanisms underlying schizophrenia predisposition. Professor Jason Mattingley's studies into mechanisms that promote brain plasticity in stroke patients have direct implications to effective rehabilitation. In addition to applications in disease, studies from Professor Mandyam Srinivasan's team revealing how birds regulate flight by optic flow has relevance in a range of areas, including biorobotics.

I am delighted, also, that significant progress has been made in establishing two research centres within QBI that bring increased focus and resources to important areas. The Centre for Ageing Dementia Research, the long-term goal of which is to develop new therapeutic approaches to treat ageing dementia, has been established and we have recruited an outstanding scientist to be its inaugural director, Professor Jürgen Götz. The Science of Learning Centre is an exciting new venture which brings together researchers with expertise in neuroscience, cognitive neuroscience, psychology, advanced brain imaging and education

to translate our increasing scientific knowledge about how the brain learns into practical protocols that can be used to promote better education in our schools, universities and the workplace.

This year we built on our strong scientific interactions with China by establishing the Joint Sino-Australian Neurogenetics Laboratory with the Second Military Medical University (SMMU) in Shanghai. The joint laboratory was opened by the then Minister for the Department of Innovation, Industry, Science and Research, Senator Kim Carr. During the ceremony, I was delighted and honoured to receive an Honorary Professorship at the SMMU. This, along with the Joint Laboratory of Neuroscience and Cognition with the Institute of Biophysics in Beijing, provides us with enormous collaborative opportunities in China.

During 2011, we welcomed several leading researchers who established new laboratories within QBI. These included Dr Allen Cheung, Associate Professor Naomi Wray, Professor Peter Visscher and Professor Tianzi Jiang. We also bade farewell to Associate Professor Mark Bellgrove, who left to establish a centre that will further expand Australia's capabilities in the area of cognitive neuroscience in his new role as Professor of Cognitive Neuroscience at Monash University.

Following a change of focus and subsequent restructuring, I wish to thank the members of QBI's Development Board, Commissioner Bob Atkinson, Dr Sallyanne Atkinson, Mr Milton Dick, Mr Mark Gray, Mr John Lyons, Mr Jeff Maclean, and ably chaired by Dr David Merson, for their service and support, since the first meeting in 2006. We have dissolved this Board and formed a new Development Board which will focus on fundraising. I am delighted that Mr Jeff Maclean has agreed to chair this vital group.

All of this has, of course, occurred in a year that commenced with the Brisbane River at St Lucia breaking its banks and enveloping our campus – an event which, fortunately, had minimal impact on QBI. I wish to thank the many staff who worked tirelessly throughout the crisis, in particular Jake Carroll, Dave Wheeldon and Ross Dixon, and their respective teams. Though several staff members' homes and possessions sustained significant damage from floodwaters, the Institute provided assistance with relocation and rental costs.

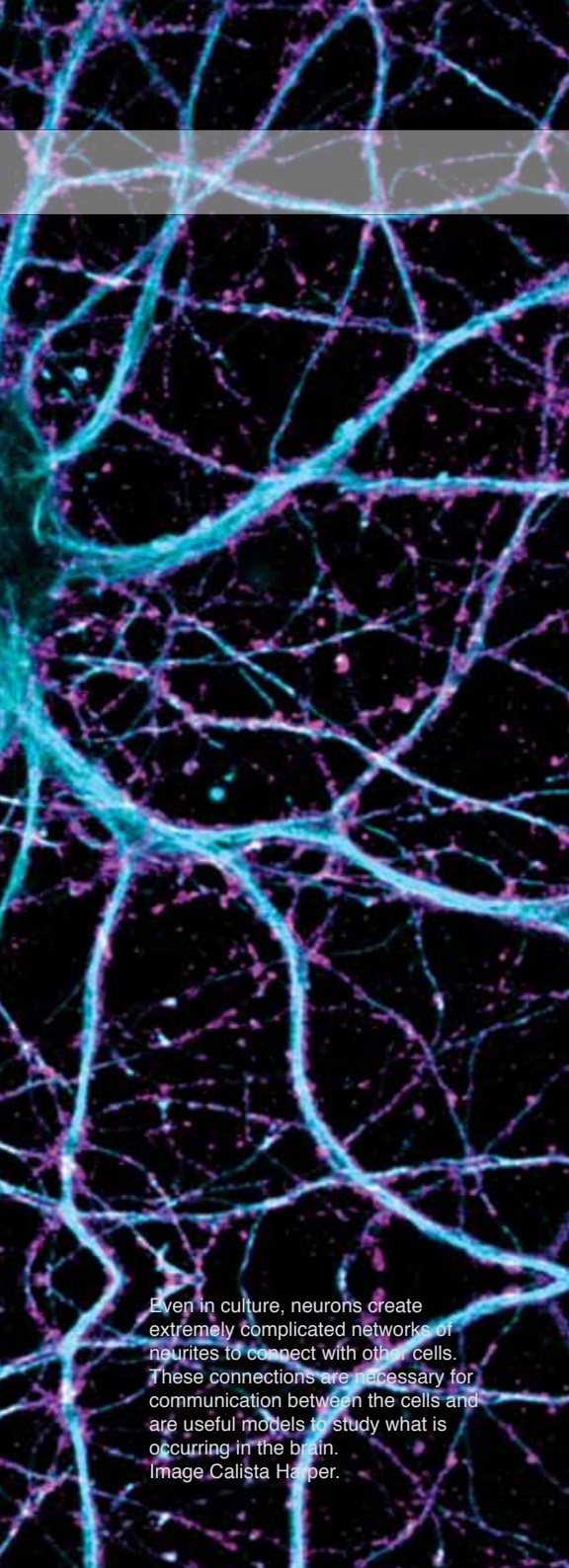
In conclusion, I would like to express my appreciation to everyone – our Faculty, postdoctoral fellows, research assistants, students and support staff. I am particularly grateful to my Deputy Directors for Research, Professor Pankaj Sah, and Operations, Mr John Kelly, for their support and ongoing commitment to the success of the Institute. I would also like to acknowledge Vice-Chancellors Professor Paul Greenfield and Debbie Terry, Senior Deputy Vice-Chancellor Professor Michael Keniger and Deputy Vice-Chancellor (Research) Professor Max Lu, for their continuing support, guidance and friendship. Last, but by no means least, I am extremely indebted to our donors and supporters and wish to express our gratitude for their contribution to the work of the Institute.

Professor Perry Bartlett FAA
Director

Discovery

The Queensland Brain Institute continues to maintain its status as one of the world's leading neuroscience institutes.

Improvements to the lives of everyday Australians are being made by our leading researchers; hand-picked from around the globe. Together with our high calibre students, these researchers create an environment of discovery that will ultimately lead to the development of much needed therapeutic treatments for neurological disorders and neurotrauma.



Even in culture, neurons create extremely complicated networks of neurites to connect with other cells. These connections are necessary for communication between the cells and are useful models to study what is occurring in the brain.
Image Calista Harper.

"Contrary to popular belief, fear-related memories are not set in stone"

Groundbreaking experiments in the Psychiatric Epigenomics Laboratory have revealed that microRNAs are key regulators of neural plasticity and cognition.

Regulating the formation of fear extinction memory



QBI neuroscientists have discovered a previously unrecognised layer of gene regulation associated with fear extinction.

This is an inhibitory learning process thought to be critical for controlling fear-related behaviour when the fear response is no longer required.

Lead researcher Dr Timothy Bredy said the findings shed new light on the processes involved in loosening the grip of fear-related memories, particularly those implicated in conditions such as phobias and post-traumatic stress disorder.

Published in *Nature Neuroscience*, the study explores how fear-related memories are formed, updated, and extinguished at the molecular level.

It also provides fresh understanding of the actual function of genes expressed at the time of retrieval of fear memories, and how they are regulated to facilitate fear extinction.

“This is the first demonstration of how small non-coding RNAs contribute to the formation of fear extinction memory, and highlights the adaptive significance of activity-dependent microRNA expression in the adult brain,” Dr Bredy said.

Non-coding RNAs are believed to function by directing the epigenome to activate or silence genes although the genome itself remains the same.

Small non-coding RNAs, such as the microRNAs studied here, can regulate gene function by complementary binding to the 3' untranslated end of their protein-coding target genes, resulting in transcriptional silencing.

Dr Bredy said that the extinction of fear-related memories occurred in the face of a competing memory process called reconsolidation, which saw memories potentially undergo modification every time they were retrieved.

“Contrary to popular belief, fear-related memories are not set in stone,” Dr Bredy said.

“Extinction learning involves retrieval and expression of the original fear memory, which naturally permits either the restabilisation of the original trace, or new extinction learning.”

“And in order for new memories to be firmly established, the genes associated with the original fear memory trace must be transiently inhibited, so that the fear extinction process can proceed.”

QBI neuroscientists probed how fear memories could be strengthened or shaken loose by intervening during extinction training.

“These findings indicate that activity of this brain-specific non-coding RNA is necessary for the formation of fear extinction memory,” Dr Bredy said.

It seems to do this by disrupting the stability of several plasticity-related target genes including one called regulator of calmodulin signalling (RCS), which is important for dopamine signalling in the brain.

“This research explains how fear extinction proceeds despite competition with the original fear memory for control over behaviour, where we have evidence to suggest that the brain specific microRNA miR-128b is essential for tipping the scale in favour of fear extinction memory rather than restabilisation of the original fear,” Dr Bredy said.

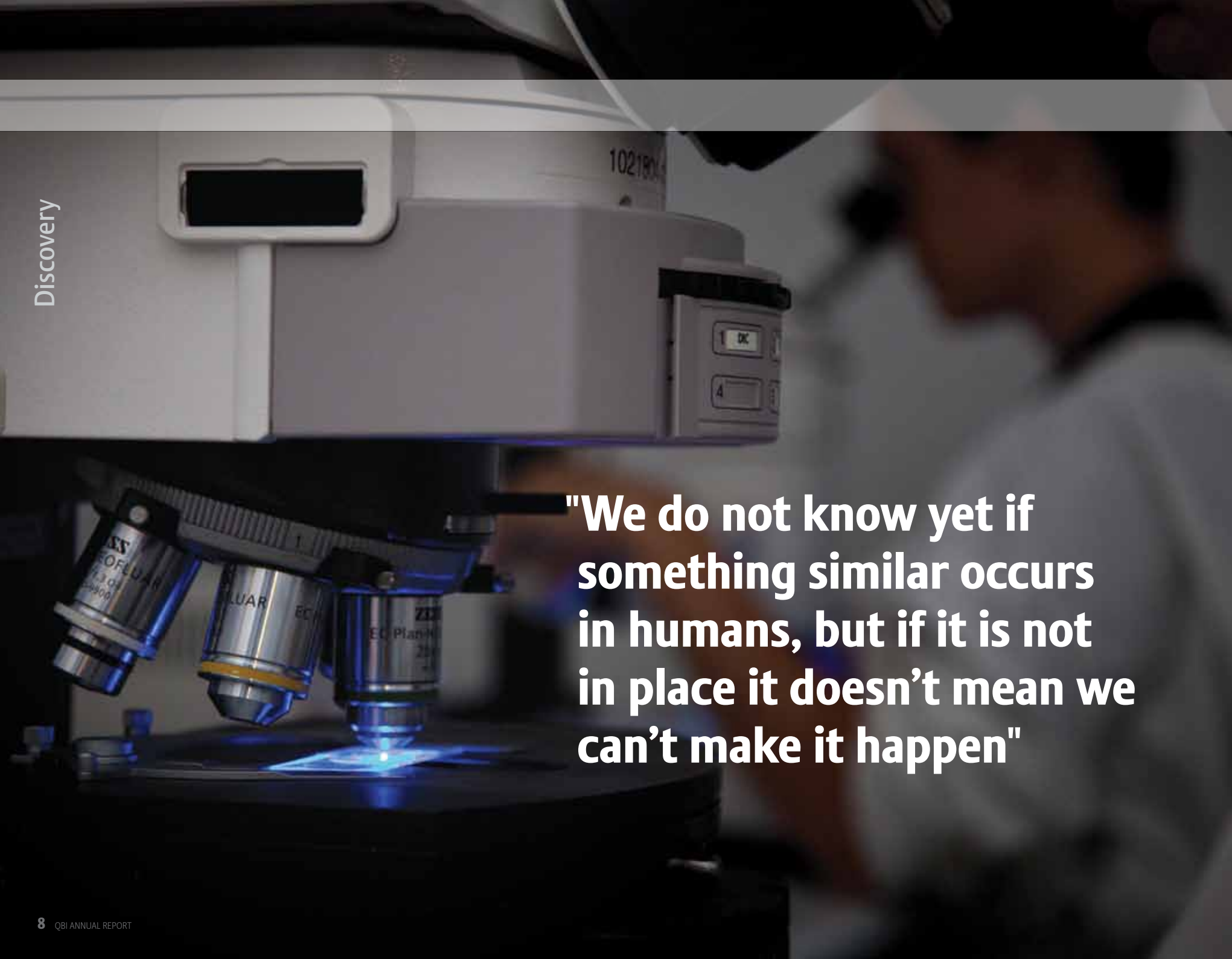
“These findings represent significant advance in our understanding of the neural mechanisms of memory updating and the processes by which fear memories can be inhibited through extinction learning.”

Dr Bredy's laboratory is interested in elucidating how the genome is connected to the environment, and how this relationship shapes brain and behaviour across the lifespan.

Embedded within the chromatin landscape, directly at the interface between intracellular signalling and genomic DNA, epigenetic mechanisms including histone modifications, DNA methylation and non-coding RNAs represent an attractive foundation for experience-dependent, long-lasting changes in gene expression, cellular function and behaviour.

In contrast to the information conveyed by a static genome, the epigenome is very dynamic and can be modified by exposure to a variety of environmental stimuli including fear-related learning, exposure to drugs of abuse, environmental toxins, dietary factors, and social interaction.

Above: A Ph.D. Candidate in the Psychiatric Epigenomics Laboratory, Mr Virkam Ratnu, employs nucleotide mass spectrometry to analyze activity-dependent changes in DNA methylation levels in cortical neurons.

A close-up photograph of a microscope, showing the eyepiece, objective lenses, and stage. The microscope is white and blue. In the background, a person wearing a white lab coat is visible, looking down at something. The image is dimly lit, with a blue light source illuminating the microscope's stage.

"We do not know yet if something similar occurs in humans, but if it is not in place it doesn't mean we can't make it happen"

Researchers probe nervous system repair



In humans, regeneration of the peripheral nervous system after injury is variable, and brain and spinal cord damage usually results in lifelong disabilities.

However, by studying nerve injury in roundworms, QBI researchers have revealed a different modality by which regeneration can occur – one that promises to shine light on how to promote repair in the nervous system.

“Though damaged nerves reconnect in a number of different ways, the underlying mechanisms remain poorly understood,” said Dr Brent Neumann, the postdoctoral fellow who carried out the work in the laboratory of Dr Massimo Hilliard.

The research, published in *Developmental Dynamics*, examined a process called axonal fusion, which has been observed in crayfish, earthworms, leeches and now in the roundworm *Caenorhabditis elegans* (*C. elegans*).

C. elegans is a nematode which, when fully grown, is less than a millimetre long.

The QBI study offers a fundamentally different mechanism for regeneration of axons (long structures that look like cables and conduct electrical impulses between neurons) from those traditionally proposed.

Using fluorescent imaging, it showed that axonal fusion is a highly effective way to restore neuronal connections with the target tissue.

Dr Neumann noted that transected (severed) axons can restore their trajectories by bridging just the damaged site instead of regrowing their entire length beyond an injury site.

“In the worm, this process happens automatically a certain percentage of the time,” Dr Hilliard explained.

“As *C. elegans* are highly accessible for genetic analyses, future research will focus on the cellular and molecular mechanisms regulating the process – and how to make it happen when it doesn’t do so naturally.”

“We do not know yet if something similar occurs in humans, but if it is not in place it doesn’t mean we can’t make it happen.”

The research was carried out in collaboration with Professor David Hall of the Albert Einstein College of Medicine in New York, and Associate Professor Adela Ben-Yakar of the University of Texas at Austin.

Other research undertaken by the Hilliard laboratory during the year probed the molecular mechanisms which prompt the development of dendrites in *C. elegans*.

Dendrites are the branch-like structures in nerve cells, which receive electrochemical signals from other nerve cells or sensory inputs from the external environment.

Together with axons, dendrites are crucial to nervous system function but their development has been poorly understood to date.

The QBI team has discovered that a ligand called LIN-44 and a receptor called LIN-17 work together to coax certain neurons in *C. elegans* to extend dendrites towards their targets.

“This is the first study to demonstrate, *in vivo*, that the initial outgrowth of a dendrite is controlled by these ligands and receptors,” says Ms Leonie Kirszenblat, the research assistant who carried out the study in Dr Hilliard’s lab.

Understanding these fundamental mechanisms of neuronal development may have practical, as well as theoretical implications, says Dr Hilliard.

“Having the ability to control dendritic growth may be important for growing neurons from stem cells, which could be useful in a range of neurological conditions, including spinal injury,” he said.

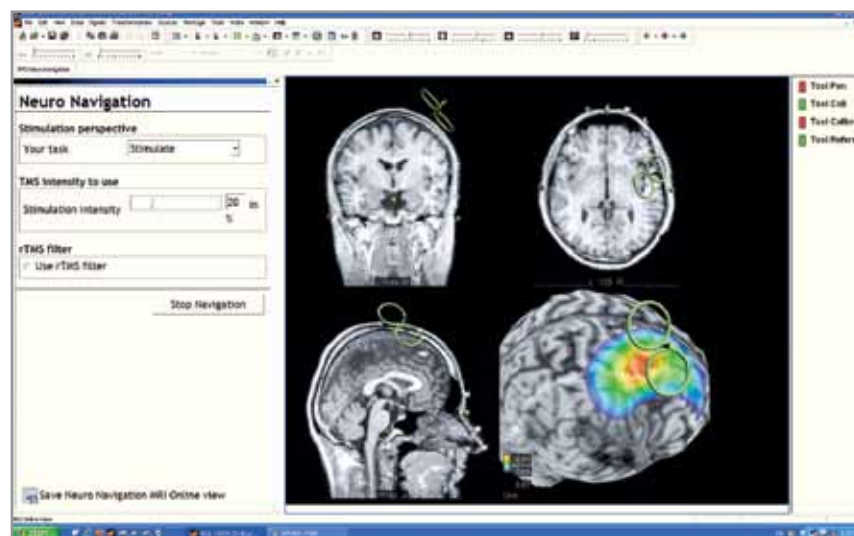
The findings were published in *PLoS Biology*.

Left: Researchers in the Hilliard Lab. Above: A red fluorescent protein expressed at the synapses (presynaptic sites) of *C. elegans* motor neurons (DD and VD classes). Image Justin Chaplin.

**"We know that attention
is a potent modulator
of brain plasticity after
damage"**



Research holds out hope for stroke patients



People with a curious condition called ‘**unilateral spatial neglect**’ are a subset of stroke patients who tend to have the worst outcomes in relation to regaining function in affected parts of their bodies.

Typically caused by strokes on the right hand side of the brain, spatial neglect manifests in patients ignoring the left side of their body and, in severe cases, behaving as though the left side of their world does not exist.

For example, affected people may ignore food on the left hand side of their plate or, if asked to draw a clock, might squash all 12 numbers into the right side of the clock face, leaving the other side blank.

Patients will also fail to shave or to put make-up on the left hand side of their faces.

“What makes this condition particularly difficult to manage is that people have no insight into their own difficulties,” says Professor Jason Mattingley.

“It’s very hard to develop interventions to help a person who doesn’t know they have a problem.”

But current research being undertaken by the Mattingley laboratory, which is exploring patterns of electrical activity in the brains of healthy volunteers, may ultimately change this outlook.

“We know that attention is a potent modulator of brain plasticity after damage,” explains Professor Mattingley.

“What we’re trying to do is explore what effect attention has on this process, and how it might be used in neurorehabilitation.”

Volunteers first undergo a magnetic resonance imaging (MRI) scan, which provides researchers with a three-dimensional picture of the brain.

“In terms of their structure, brains are like fingerprints – no two are exactly the same, even though superficially they seem very similar,” Professor Mattingley explains.

The MRI scan allows researchers to guide a transcranial magnetic stimulation (TMS) coil into position on a volunteer’s scalp.

The device induces a small electrical current in the underlying brain tissue, causing it to become more active.

The researchers specifically target a part of the motor cortex that controls the thumb muscle in the left hand.

Experimenters also place a small electrode on the wrist to activate a nerve bundle that sends signals from the hand back to the brain.

When this volley of nerve activity from the hand is timed to arrive in the motor cortex at the same time as a pulse of TMS is delivered there, a form of local brain plasticity is induced, which can last for several minutes.

“It’s well established that the more often neurons activate at the same time, the more likely they are to communicate efficiently in the future. This is how the brain learns,” Professor Mattingley explains.

“We’re exploiting that general principle in this research.”

But what the researchers have found is that the effects of stimulation on a brain’s plasticity are dependent on where a person’s attention was focussed.

“When we ask people to pay attention to small flashing lights located near to the left thumb, we observe very strongly enhanced brain plasticity,” Professor Mattingley explains.

“But when people are paying attention to the same flashing lights positioned near to the right thumb, plasticity effects for the ignored left hand are diminished.

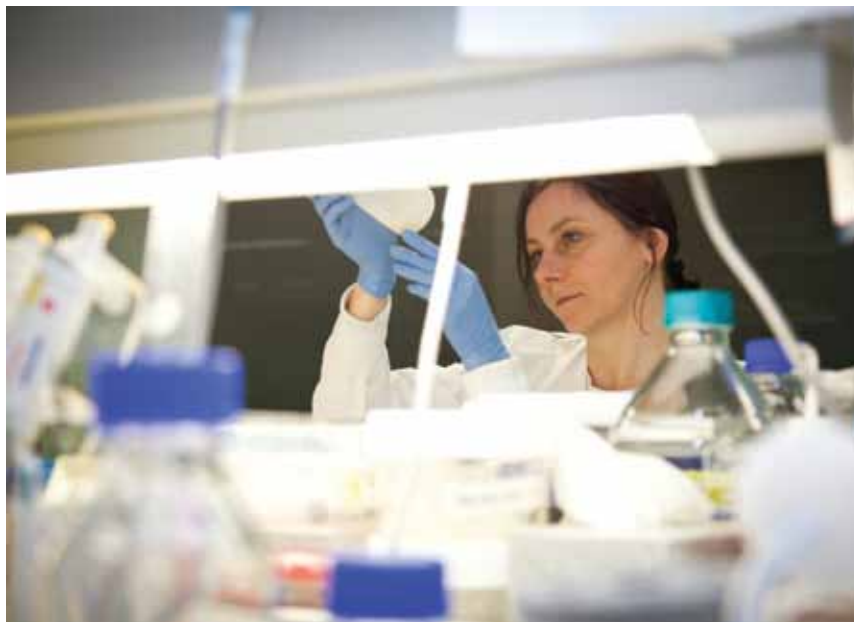
So, in other words, people who aren’t paying attention to the side of their body that is being stimulated tend not to show any alterations in brain plasticity, and we suspect something similar is what’s happening in cases of spatial neglect.”

Their brains just aren’t learning any more.

While practical applications remain several steps away, this knowledge may ultimately help us develop more effective strategies for physical therapy after stroke.”

**"Dyngo-4a™
significantly delayed
the onset of paralysis,
botulism's most lethal
symptom, by more
than 30%"**

Study brings secrets of brain cell communication closer



Researchers in the Meunier laboratory this year took a significant step towards unravelling the mechanism by which communication between brain cells occurs.

Findings from a study published in *Nature Communications* revealed that the lipid (fat) from the membranes of brain cells controls the movement of vesicles containing chemical messengers called neurotransmitters.

QBI's Associate Professor Frederic Meunier, who led the study, said these findings were made possible through experimentation with very selective compounds affecting the membrane.

"Our findings explain how minute changes in the lipid composition of our neurons can have a

dramatic effect on the way these cells communicate with each other in the brain," he said.

"We found that the lipid phosphatidylinositol(4,5) bisphosphate orchestrates the mobilisation and movement of secretory vesicles towards the plasma membrane of neurosecretory cells."

According to Associate Professor Meunier, a better understanding of the mechanism underpinning neurotransmitter release will aid scientists' ongoing fight against the plethora of diseases affecting neuronal communication in the brain.

"Changes in lipid composition have already been shown to be a factor contributing to the development of dementia in Alzheimer's disease," he says.

"We hope that developing novel compounds targeting the fat lipid composition of biological membranes could ultimately help in the treatment of such brain disorders."

The study was carried out in conjunction with colleagues from The University of Queensland's School of Biomedical Sciences, Flinders University in South Australia, the Centre for Cell Signalling within the Institute of Cancer in London, the Australian Centre for Blood Diseases in Melbourne's Monash University and the Max Planck Institute of Biochemistry in Germany.

During 2011, the Meunier laboratory was also part of a team which discovered a new way to block the action of botulinum toxin – a finding which promises to pave the way for more effective treatments of the life-threatening disease botulism.

The team, comprising scientists from QBI, the University of Newcastle and the Children's Medical Research Institute, have found a novel way of blocking the uptake of the toxin using a new class of drug called dynamin inhibitors.

"We have designed and tested a new molecule called Dyngo-4a™ which prevents botulinum toxin

from entering nerve cells," Associate Professor Meunier explained.

"Dyngo-4a™ works by blocking the action of a protein called dynamin which plays a key role in controlling how most molecules can enter nerve cells."

Botulism is a rare but potentially fatal condition that involves progressive weakness.

It is caused by botulinum toxin, which is made by the *Clostridium botulinum* bacterium found naturally in soil, sediments, raw foods (including seafood) and honey.

As terrorists have also attempted to use botulinum toxin as a bioweapon, development of more effective treatments to counter this type of health threat is a high priority for countries such as the United States.

"The toxin that causes botulism is one of the most deadly agents known" Associate Professor Meunier said.

Currently, the only known treatment for botulism is antibodies that bind some of the toxin before it reaches nerve cells.

"Dyngo-4a™ significantly delayed the onset of paralysis, botulism's most lethal symptom, by more than 30 per cent" Associate Professor Meunier explained.

"This is significant because it may provide extra time for antibodies to take effect and minimise symptoms."

"Our research is the first to identify the protein dynamin as a suitable drug target for preventing botulinum toxin entering nerve cells throughout the body."

The research was published in the *Journal of Biological Chemistry*.

Left: A mouse hippocampal neuron labeled with the heavy chain of botulinum neurotoxin type-A (BoNT-A, white) and VAMP2 (red), a synaptic vesicle marker.
Above: Dr Sally Martin in the Meunier laboratory.

**"The study also confirmed
genetic overlap between
bipolar disorder and
schizophrenia"**

Study reveals fresh insights into genetics of schizophrenia

Researchers are a step closer to unravelling the genetic underpinnings of schizophrenia following the largest genome-wide association study (GWAS) of the disorder ever undertaken.

An international consortium of 190 researchers from 135 institutions, including QBI, found significant associations with schizophrenia for five new and two previously implicated locations on the human genome.

It has long been recognised that schizophrenia is highly heritable.

However, this new study has pinpointed novel regions of the human genome significantly associated with disease, and confirmed other recently reported genomic regions that may harbour disease-causing genetic variation.

According to Professor Bryan Mowry, who initiated and coordinated the Australian contribution to this study, these findings were made possible because of the unprecedented size of the study, which involved more than 50,000 participants.

“It provides a solid foundation for beginning to understand the mechanisms underlying the substantial genetic predisposition to schizophrenia,” Professor Mowry said.

The research was published in *Nature Genetics*.

Schizophrenia affects 1 in 100 people and its onset typically occurs in adolescence or early adulthood.

Psychosis (comprising hallucinations and delusions) is the hallmark of schizophrenia, but other symptoms such as personal neglect and amotivation are common, as is an increased risk of suicide.

Professor Mowry says that gaining a better understanding of the genetic architecture of schizophrenia will ultimately aid the earlier diagnosis and management of the disorder.

“If your genetic profile suggests you have a predisposition towards developing schizophrenia, it will be particularly important for you to avoid known environmental risk factors – such as smoking cannabis,” he says.

“We also expect that understanding the biological mechanisms underlying the disorder will lead to more robust therapeutics in future.”

GWAS such as this one are aimed at discovering genetic variations associated with disease, particularly common diseases such as asthma, cancer, diabetes, heart disease and mental illnesses.

These studies involve rapidly screening hundreds of thousands of DNA markers (single nucleotide polymorphisms, or SNPs) of known location on the human genome across thousands of DNA samples of people with a particular disease and thousands of healthy people (controls).

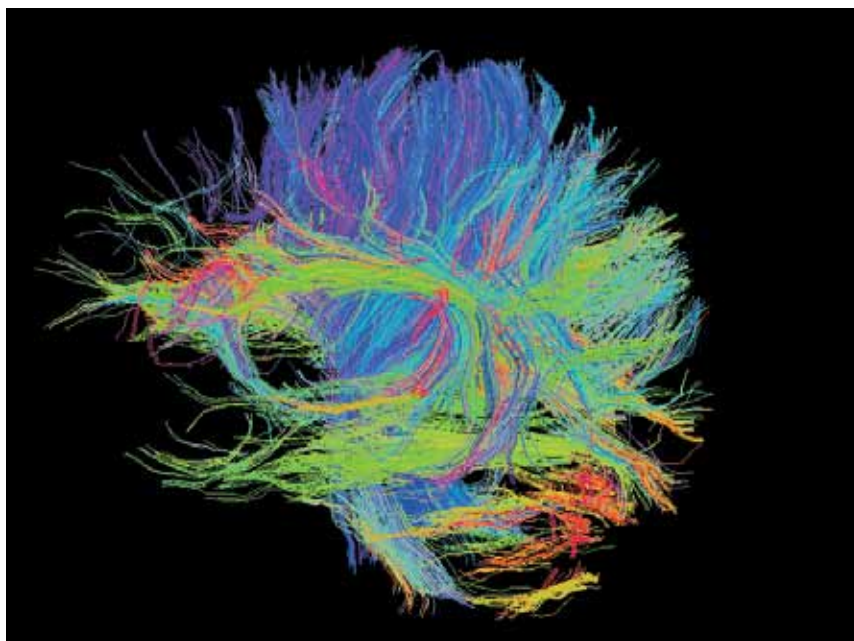
SNPs are relatively evenly spaced across the human genome (approximately one every 80 bases) and are strategically selected to comprise a dense panel of markers of genetic variation for use in GWAS.

If certain SNPs are associated with disease – that is, if they are significantly more frequent in disease cases than controls – they highlight a region of the genome where disease-causing variants may be located.

The strongest genome-wide association finding in the current study was to SNPs in a region containing numerous immune-related genes, suggesting that schizophrenia may be triggered by autoimmune responses or infection.

Another SNP in a region linked to neuronal development was also implicated, suggesting a novel mechanism underlying schizophrenia.

The study also confirmed genetic overlap between bipolar disorder and schizophrenia, suggesting that these disorders have shared rather than separate roots.



Opposite: Heather Smith at work in the Mowry Laboratory. Above: White-matter images acquired using Diffusion Tensor Imaging (DTI) as part of ongoing study into endophenotypes of schizophrenia.



**"QBI researchers have
unlocked the secrets
of how birds avoid
collisions"**

This page: Abstract rendition of a sequence of images illustrating the flight of a budgerigar through a tunnel in which the walls are decorated with various visual patterns, in experiments designed to investigate visual guidance of bird flight. Image: Dr. Ingo Schiffner. Opposite: Professor Mandyam Srinivasan

Research illuminates birds' use of **optic flow** cues to guide flight



The beauty and majesty of birds in flight has long captured the attention of artists and photographers.

Now QBI researchers have unlocked the secrets of how birds avoid collisions as they soar, swoop, dive, glide and engage in other aeronautic manoeuvres.

The grace of birds in even cluttered environments is all a function of their perception of something called optic flow, according to lead researcher Dr Partha Bhagavatula.

“Our findings show, for the first time, that birds regulate their speed and negotiate narrow gaps safely by balancing the speed of image motion, or optic flow, experienced by the two eyes,” said Dr Bhagavatula.

In order to undertake the study, researchers trained budgerigars to fly through a seven-metre corridor.

Researchers then lined the corridor with different combinations of thick black horizontal and vertical stripes and filmed the birds’ flight trajectories.

They found that the budgerigars flew down the centre of the corridor when optic flow cues were balanced (with identical, vertical stripes on either side of the corridor) but more closely towards one wall or another when these cues were unbalanced (such as when one wall was lined with horizontal stripes and the other with vertical stripes).

The birds flew faster when the tunnels were lined with horizontal stripes (rather than vertical stripes), indicating that they were using optic flow cues to regulate their flight speed.

Dr Bhagavatula explains that because the birds naturally flew in a horizontal direction within the tunnel, horizontal stripes (parallel to the direction of flight) would provide only weak motion

cues, whereas vertical stripes (perpendicular to the direction of flight) would provide strong motion cues.

While similar flight behaviours have previously been demonstrated in honeybees, bumblebees and flies, this is the first time the use of optic-flow signals has been demonstrated in birds.

“The findings suggest that some of the principles that underlie visually guided flight may be shared by all diurnal flying animals,” says Professor Mandyam Srinivasan, head of the laboratory.

According to Professor Srinivasan, these findings also have important implications for robotics.

Specifically, the speed, agility and accuracy with which birds fly through a thicket of branches will teach scientists a lot about designing vision systems for guiding autonomous aerial vehicles through densely cluttered environments.

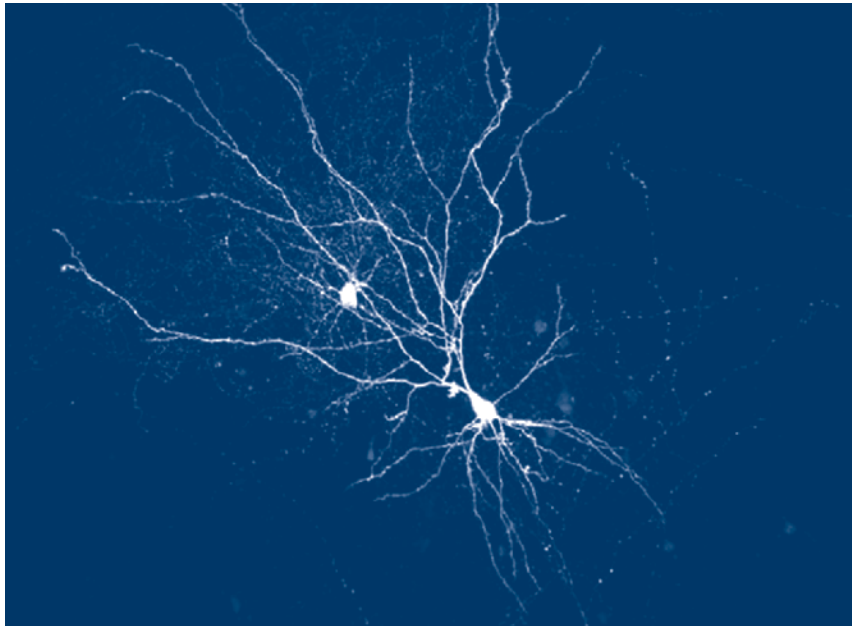
The research was published in *Current Biology*.

During the year, the Biorobotics Laboratory within Professor Srinivasan’s group has been able to use the results of the group’s work on birds and honeybees to devise biologically inspired algorithms for the visual guidance of aircraft that enable automated execution of a range of extreme aerobatic manoeuvres, as well as automated landing.

This work was highlighted in ABC’s science show *Catalyst* and in *New Scientist* magazine.

"such findings may shed light on how the brain works so efficiently to compute enormous amounts of information"

Increasing our understanding of how the **neocortex** operates



Deep within the grooves and wrinkles of the neocortex lies humans' ability to read maps, appreciate artworks, pen poems, recall memories and undertake a range of other higher order tasks.

Yet despite a century of experimental and theoretical work, the neural computations that underpin behaviour remain poorly understood.

This is an area of neuroscience that Associate Professor Stephen Williams is attempting to shed some light on, as he turns new optical and electrophysiological recording techniques to the task of probing the neuronal and circuit computations that underlie sensory-motor behaviour in rodents.

"We know that the neocortex is divided into specialised areas, which manage functions such as vision, motor control and somatosensation," Associate Professor Williams explains.

"However, there's also a rich reciprocal connectivity between different areas."

"This connectivity may underlie multi-modal sensory processing and contribute to directed attention, and so is thought to play a decisive role in the control of animal behaviour."

Rodents, for example, employ vibrissal (whisker) tactile discrimination as a primary sensory modality.

It's believed that the primary vibrissal motor neocortex is reciprocally connected, by long-range intercortical pathways, with the primary vibrissal somatosensory neocortex.

"This strongly suggests that the two systems operate in concert," Associate Professor Williams explains.

Through undertaking experiments involving a range of techniques, including *in vivo* recording techniques from animals exploring their environments, Associate Professor Williams and his col-

laborators at the Howard Hughes Medical Institute in the USA aim to probe and map these little-understood connections.

"This work will significantly increase our knowledge of the operation of the neocortex, by linking the integrative operations of pyramidal neurons to behaviour," he explains.

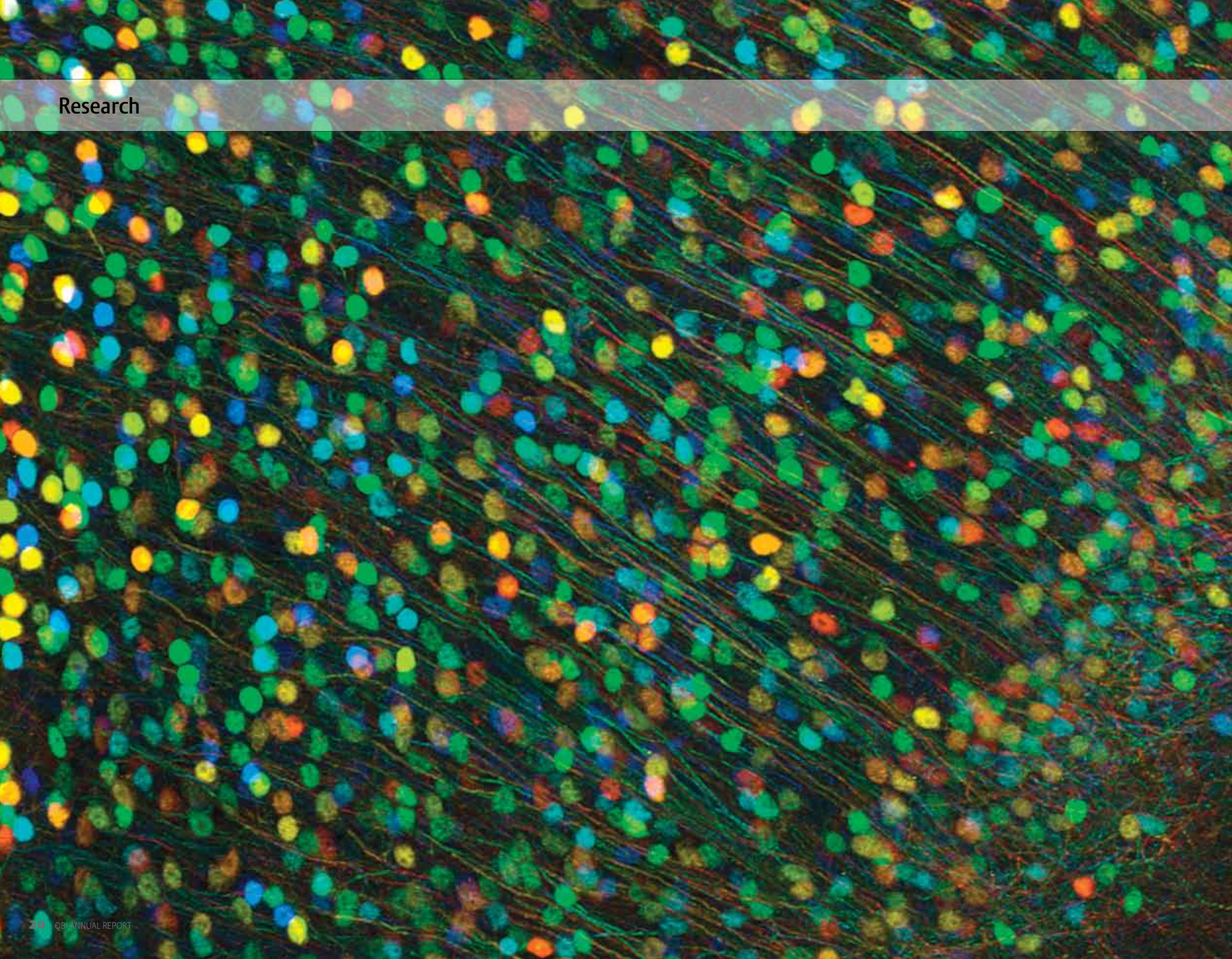
"Moreover, our collaborative work links the electrical operation of the dendrites of neurons with animal behaviour."

"As individual neurons are believed to perform independent integrative steps in their complex dendritic arbors, such findings may shed light on how the brain works so efficiently to compute enormous amounts of information and integrate across a number of modalities."

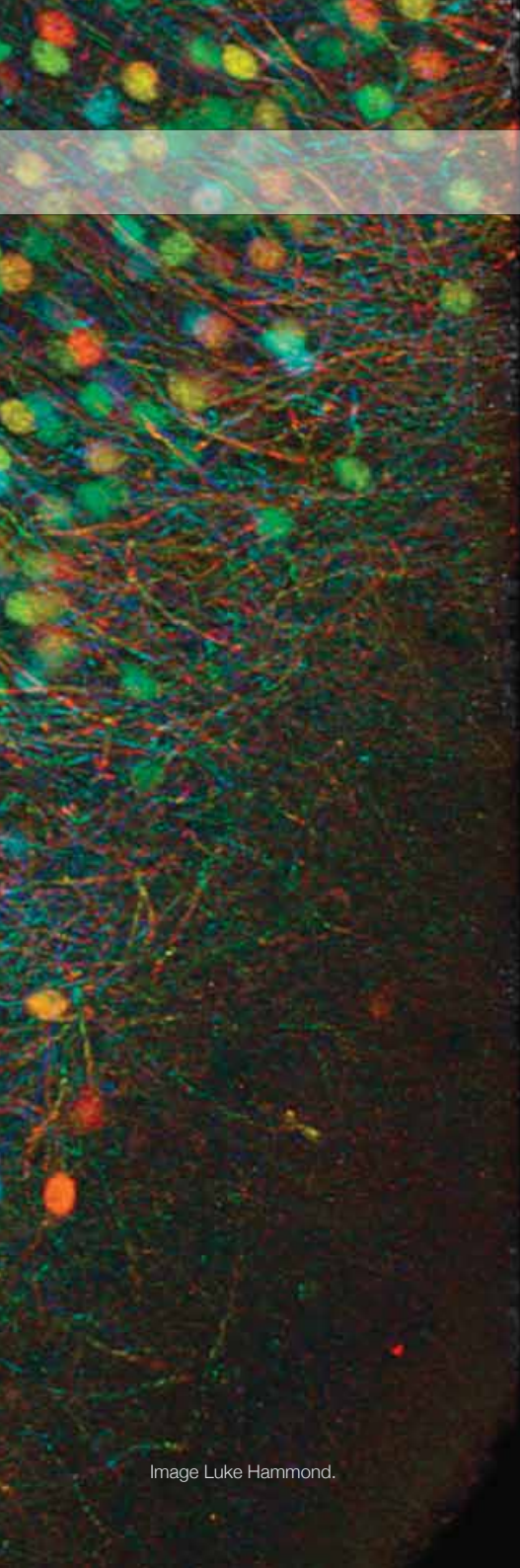
Associate Professor Williams has pioneered the investigation of dendritic excitability in central neurons and his laboratory is one of only three to four groups in the world who are working in this highly specialised field.

During the year, the work of its members has been published in a range of influential journals, including the *Journal of Neuroscience* and *Nature Communications*.

Left: Associate Professor Steven Williams.
Above: Synaptically connected layer 1 inhibitory interneuron and layer 2/3 pyramidal neuron.

The background of the entire page is an abstract, high-resolution image. It features a dense field of small, out-of-focus, multi-colored circular spots (bokeh) in shades of green, blue, yellow, orange, and red. Overlaid on these spots are numerous thin, dark, fiber-like or thread-like structures that crisscross the entire frame, creating a complex, textured appearance reminiscent of a microscopic view of neural tissue or a dense network of fibers.

Research

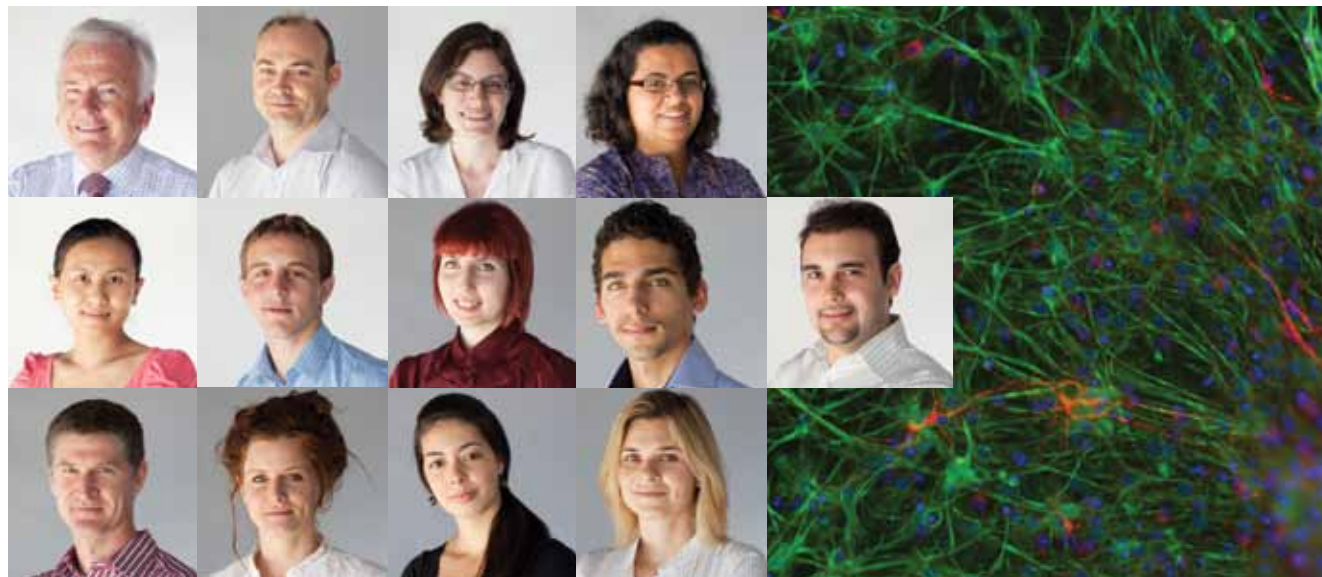


Research

Unlike other research organisations which focus on particular diseases or conditions, QBI is structured to study the brain's fundamental molecular and physiological mechanisms. Research is conducted across the seven key themes of cognition and behaviour, computation and neuronal circuits, neurogenesis and neuronal survival, genetics and epigenetics, neuronal development and connectivity, sensory systems and synaptic function.

QBI neuroscientists use the world's most advanced investigative technologies for their research, including flow cytometry and mass spectrometry equipment. They use human volunteers in their research and also study a range of animal models including the mouse, honeybee, fruit fly, frog, zebrafish and flatworm.

Laboratory Head Professor Perry Bartlett



2011 Laboratory Members L-R: Perry Bartlett, Daniel Blackmore, Lavinia Codd, Dhanisha Jhaveri, Jing Lu, Cornel Mirciov, Estella Newcombe, Boris Prosper, Gregory Robinson, Mark Spanevello, Chanel Taylor, Sophie Tajouri, Jana Vukovic. **Background:** A differentiated neurosphere, Image Dan Blackmore.

Regulating precursor cell activity and neurogenesis in the brain

Professor Perry Bartlett's laboratory previously identified that, although the production of new neurons declines progressively with age, a latent neural precursor cell population remains in the adult brain throughout life. Work in the Bartlett laboratory has since focussed on the triggers and mechanisms controlling the activation and production of new nerve cells from this latent cell population in the two key neurogenic areas of the adult brain, the hippocampus, which is critical for some forms of learning and memory, and the subventricular zone.

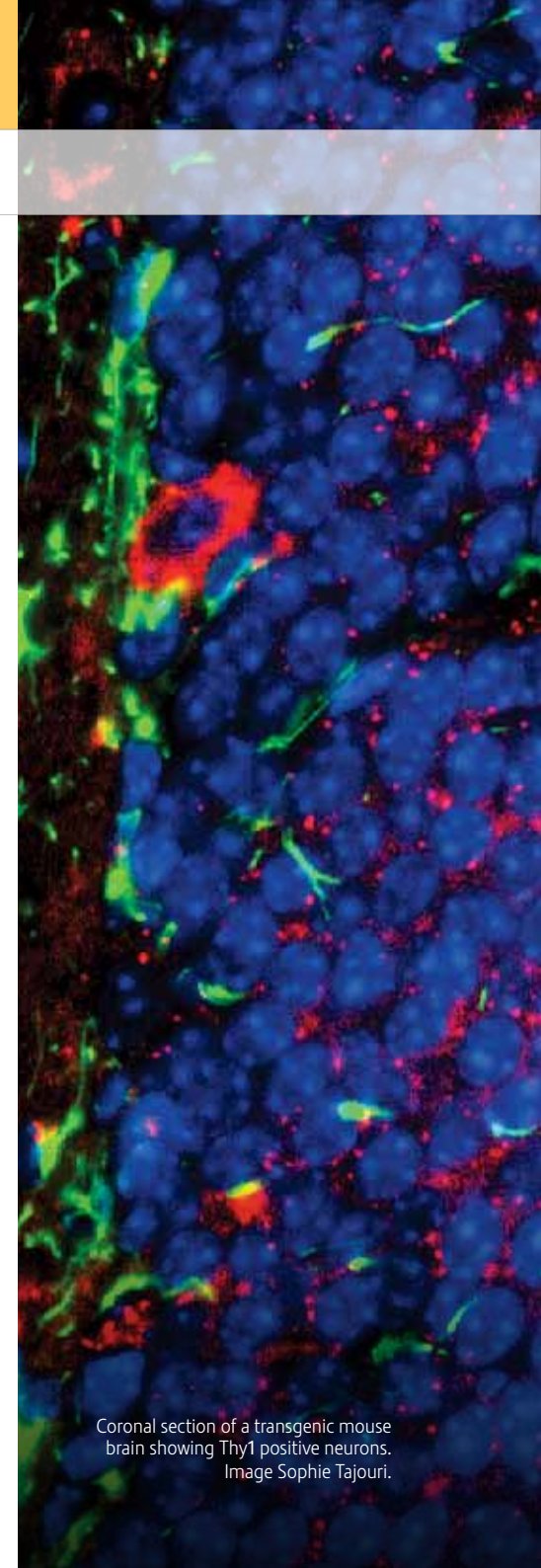
Understanding the regulation and activity of neural precursor cells in the adult brain has

continued to be a focal point of research in the Bartlett laboratory in 2011.

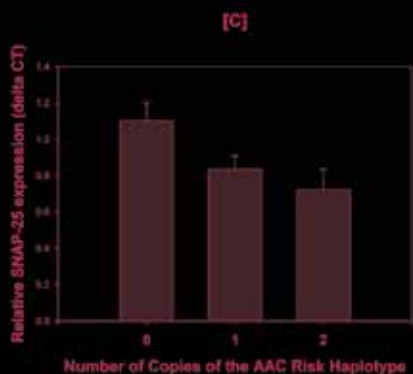
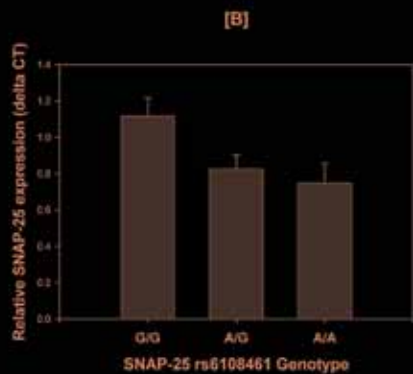
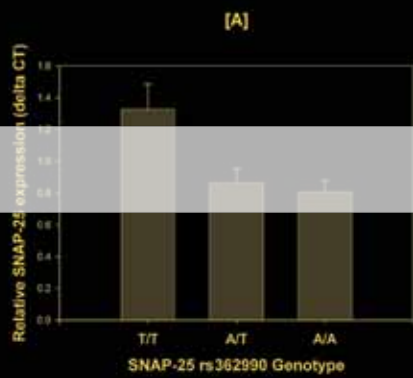
The group has recently reported that signalling of the cytokine Oncostatin M is important for neural precursor cell homeostasis. More specifically, Oncostatin M was found to directly inhibit proliferation of neural precursor cells in the subventricular zone and hippocampus both *in vitro* and *in vivo*. Prior to this finding, no role had been established for Oncostatin M in neurogenesis.

Further, during 2011, the Bartlett laboratory reported that whereas hippocampal precursor cells decline with age in wild type mice, a latent

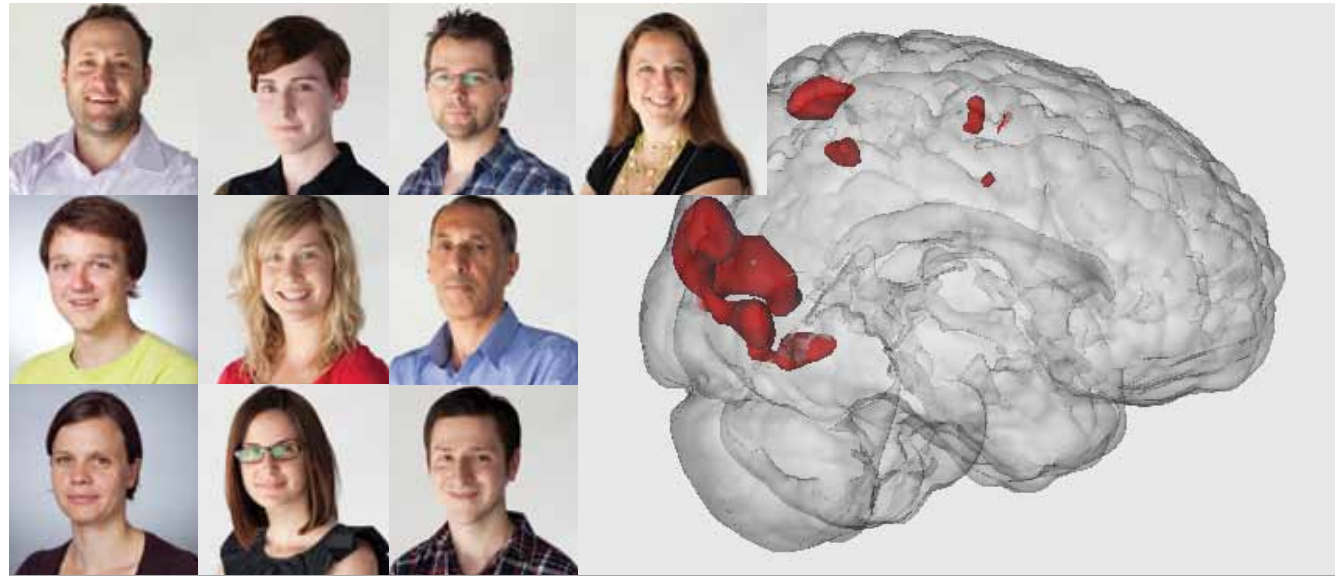
population of neural precursor cells is retained in the hippocampus of a mouse model of Huntington's disease. This raises the possibility that if the dormant precursor and stem cells could be activated to produce new cells, thereby replacing the degenerating neurons, the motor, psychiatric and cognitive deficits that are characteristic of Huntington's disease could be ameliorated. Once a more detailed understanding of the mechanism is established, it may be possible to use this method to repair other areas of neuronal cell loss.



Coronal section of a transgenic mouse brain showing Thy1 positive neurons.
Image Sophie Tajouri.

Laboratory Head **Assoc. Professor Mark Bellgrove**

Relative expression level of SNAP25 in non pathological samples; A and B showing decreased expression with ADHD associated alleles; C shows the level of expression relative to ADHD risk haplotypes. Image Ziarhi Hawi



2011 Laboratory Members L-R: Mark Bellgrove, Jessica Barnes, Tarrant Cummins, Angela Dean, Jack Goodrich, Teresa Hall, Ziarhi Hawi, Inga Laube, Natasha Matthews, Daniel Stjepanovic. **Absent:** Mykolas Byrne, Lara Campbell, Kelly Garner, Sanjay Nandam, Daniel Newman, Joe Wagner. **Background:** Highlighted in red, brain areas become more active when volunteers attempt to complete a difficult task of paying attention to important parts of a complex visual display while ignoring others. Image Daniel Stjepanovic

The genetics and pharmacology of human cognition

Work in the Bellgrove laboratory is investigating the biological mechanisms underpinning human cognition. The laboratory has a multi-disciplinary focus with staff and students trained in molecular genetics, pharmacology, psychiatry and cognitive neuroscience. The laboratory conducts basic human experimental work that attempts to link DNA variation in chemical signalling genes to our abilities to behave and think flexibly in an ever-changing social world.

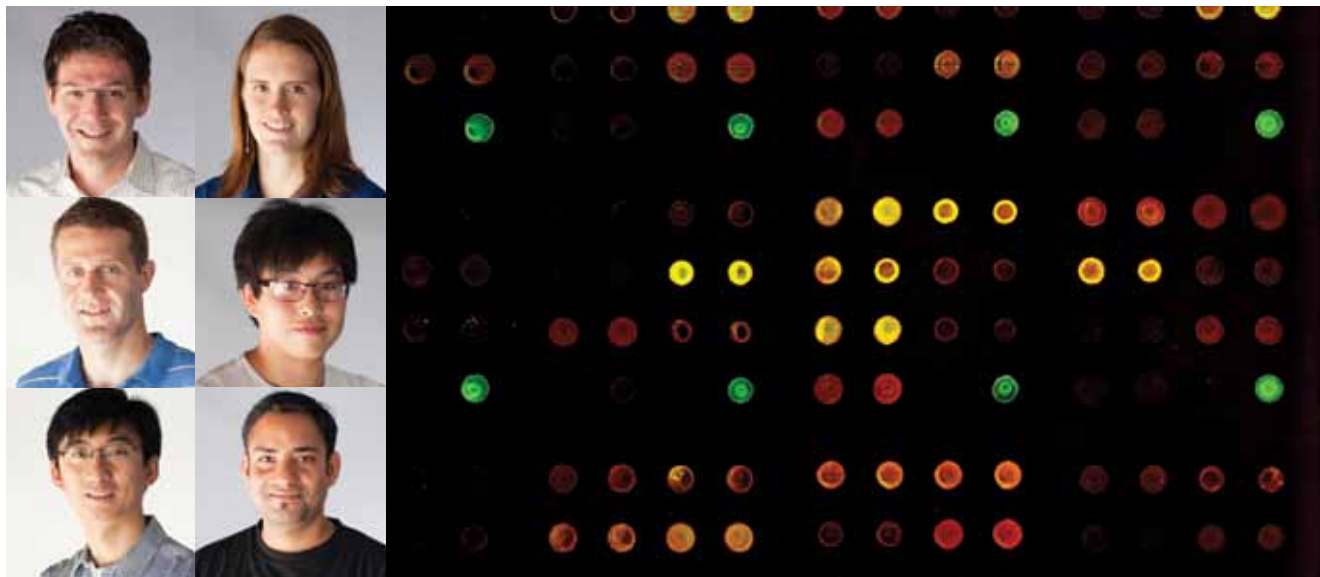
The search for candidate genes is informed by pharmacological challenge studies in which the impact of pharmacological agents on brain and

behaviour is examined. A major goal of the work within the laboratory is to map susceptibility pathways for psychiatric disorders. Thus the identification of 'genes for attention' in healthy populations can provide vital clues to the genetics of heritable disorders of attention, such as attention deficit hyperactivity disorder (ADHD). Major discoveries include the identification of polymorphisms of the dopamine D2 receptor gene that predict the degree to which individuals are aware of errors in their performance. These results also have important implications for disorders such as schizophrenia and drug addiction where there is a loss of

insight and awareness and fundamental deficits in dopamine D2 signalling.

Other work in the laboratory has, in collaboration with a number of other centres, identified DNA variants in the SNAP-25 gene as a susceptibility mechanism for ADHD. Further, by studying expression of the gene in the post-mortem brain, the group has shown that variants of the SNAP-25 gene that confer risk to ADHD have altered expression in the key regions of the frontal cortex that are known to be dysfunctional in individuals with ADHD.

Laboratory Head Dr Timothy Bredy



2011 Laboratory Members L-R: Timothy Bredy, Danay Baker-Andresen, Kevin Dudley, Xiang Li, Wei Wei, Vikram Ratnu. **Absent:** Paola Spadaro. **Background:** Protein antibody microarray is used to determine the epigenetic regulatory proteins associated with fear extinction learning.

Epigenetic mechanisms regulating the formation & maintenance of memory

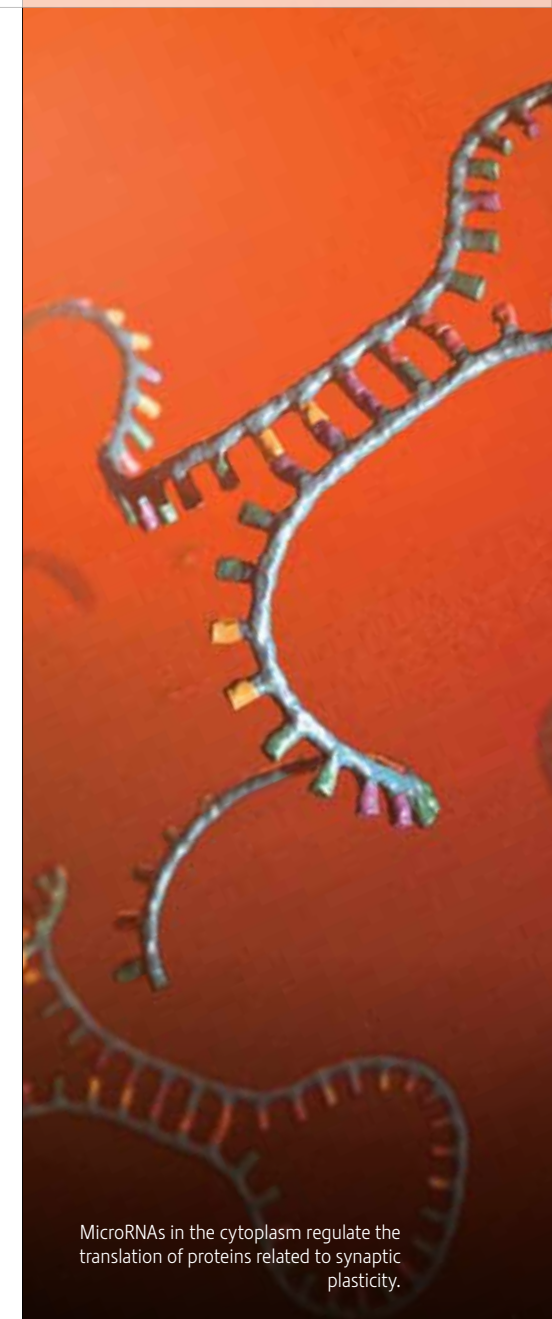
The extinction of conditioned fear, the reduction in responding to a feared cue when the cue is repeatedly presented without any adverse consequence, is an important model for the treatment of anxiety disorders. Like other forms of learning, long-lasting memory for fear extinction depends on coordinated gene expression and the synthesis of new synaptic proteins. This process involves a tightly controlled interplay between transcriptional machinery and enzymes that regulate chromatin structure. Research in the Bredy laboratory is elucidating how the genome is connected to the environment, and

how this relationship shapes behaviour across the lifespan. The group is particularly interested in how epigenetic mechanisms, including DNA methylation, histone modification and the activity of small non-coding RNAs, regulate the formation and maintenance of memories such as those associated with fear extinction.

2011 was a highly productive year for the laboratory, which published two review articles in the journals *Neurobiology of Learning and Memory* and *Neuroscience and Biobehavioural Reviews*, as well as two major discoveries on the

role of epigenetic mechanisms in fear extinction.

The first of these, which appeared in the *Journal of Neuroscience*, demonstrated that inhibition of the histone acetyltransferase p300 enhances memory for fear extinction. The second, published in *Nature Neuroscience*, revealed the important role of a brain-specific microRNA, miR-128b, in regulating fear extinction memory. These findings have significant implications for the development of novel therapeutic approaches for the treatment of fear-related anxiety disorders.



MicroRNAs in the cytoplasm regulate the translation of proteins related to synaptic plasticity.

Laboratory Head Dr Thomas Burne



2011 Laboratory Members L-R: Thomas Burne, Suzanne Alexander, Claire Foldi, Pauline Ko, Natalie Groves, Lauren Harms, Karly Turner. **Background:** Section from Painting by Glenn Brady, Cold day at Lutwyche, 1996, Acrylic on canvas. Image thanks to QCMHR Gallery.

Exploring brain development and behaviour in animal models

Research in the Burne laboratory is focused 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 (DVD) deficiency on brain development, the impact of adult vitamin D deficiency on brain function and behaviour and, more recently, the neurobiological effects of having an older father.

In 2011 the Burne group, in collaboration with QBI's Dr 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 in animal models. Now the collaboration is investigating the impact of DVD deficiency on social and cognitive behaviours. Research into the impact of adult vitamin D deficiency on brain function has also started.

The Burne group has also expanded its research tools, with a suite of cognitive behavioural tasks to assess attentional processing in rodents. The

goal now is to investigate the neurobiology of altered cognition in animal models, by looking at selected cognitive domains – sensorimotor gating, working memory, attention and speed of processing, learning and memory, and problem solving – that are known to be disrupted in schizophrenia. In collaboration with other neuroscientists at QBI, they have also begun to use other species, such as zebrafish and fruit flies, to capitalise on the advantages of the 'small brain' for such research, work which was featured on the cover of *Molecular Psychiatry*.

Section from Front panel of Schizophrenia/ Internal Symmetry, Craig Finn. Collage Acrylic on polycarbonate corrugated surface. Image thanks to QCMHR Gallery.

Laboratory Head Dr Allen Cheung



2011 Laboratory Member Above: Allen Cheung. Background: Seeing is believing – a tethered honeybee in a virtual tunnel displayed by computer monitors. Image Chen Wu.

Explaining neurocomputational theory of 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.

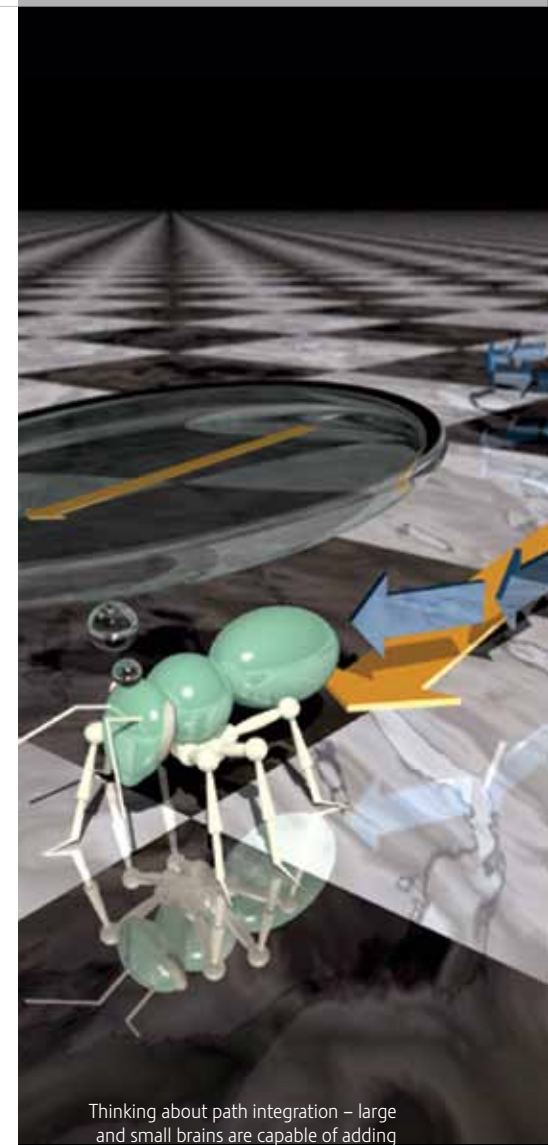
Path integration is one strategy used by vertebrates and invertebrates alike, and may well be the common 'scaffold' required for spatial navigation generally. It is the process whereby estimated self-motion is integrated over time

to yield an approximate vector between the starting location and current location. Recently, the laboratory proved mathematically that there are key computational constraints governing all neural circuitry involved in path integration, irrespective of species differences.

However, even an ideal path integration circuit cannot work effectively if an animal doesn't see, smell or detect external landmarks. Yet, in experiments, rats have neurons (place cells and grid cells) which consistently 'know' where they are in total darkness. In collaboration with computer scientists and roboticists, the group recently showed that this can be explained if the

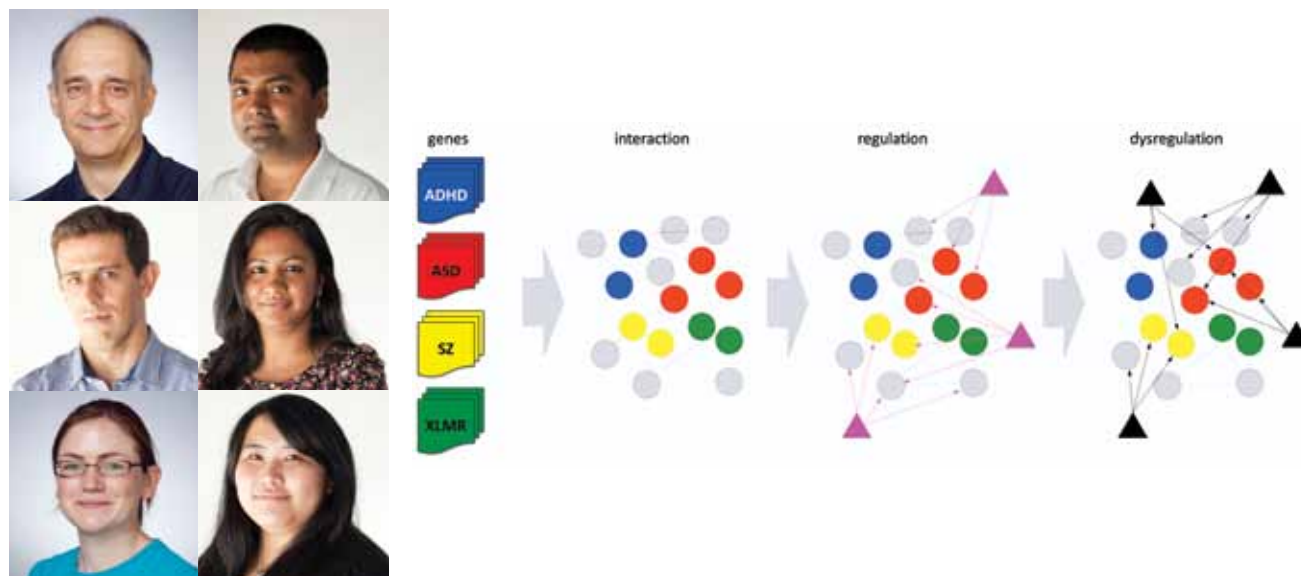
rat acquired a map-like representation of the arena boundary, which is combined with path integration in an optimal probabilistic manner. There are important implications for the neural networks involved in such a system.

In collaboration with experimentalists, a virtual reality system has also been developed which is capable of initiating and maintaining flight behaviour in tethered honeybees. An unexpected visually driven 'streamlining' response has been discovered. In the future, it may be possible to record from the neural circuits of this superb invertebrate navigator, while flying in a virtual world.



Thinking about path integration – large and small brains are capable of adding up displacements through space, leading to an internal estimate of position. Errors impact on path integration in vastly different but predictable ways, depending on the computations performed by the brain circuits involved.

Laboratory Head Dr Charles Claudianos



2011 Laboratory Members L-R: Charles Claudianos, Partha Bhagavatula, Alex Cristino, Nivetha Gunasekaran, Aoife Larkin, Yuelin Liu. Absent: Joon An, Mathew Brugioni. Background: Network analyses used to abstract a molecular basis for cognitive disorder.

Molecular mechanisms of senses and synapses

The major aim of the Claudianos laboratory is to characterise key molecular processes involved in synapse development that provide detailed insight into the aetiology and diagnosis of cognitive disorder. Many data now show that a loss of synapses or aberrant synaptic connection between neurons will affect brain function. Molecules involved in synapse development such as neuroligin and neuroligin now head a list of causative molecular associations in the pathogenesis of autism spectrum disorders (ASD) and schizophrenia.

Using tractable insect genetic and behavioural models, the fly and the honeybee, the laboratory

examines the role of these molecules and their fundamental biological relevance to healthy brain function. They then go back to the human and use genome network analyses to identify the molecular processes and pathways in which these and other candidate ASD, schizophrenia, attention deficit hyperactivity disorder (ADHD) and X-linked mental retardation (XLMR) genes are involved. This systems approach highlights the central role that synaptic molecules play within a novel *in silico* profile of human cognitive disorder.

The Claudianos group, in collaboration with QBI colleagues, has demonstrated that the

small brain can be a useful tool for researching neuropsychiatric disorders. They use the *Drosophila* fly and the honeybee to examine the biological role of synaptic molecules with regard to healthy brain functions. Biochemical interaction, gene expression and targeted-disruption of these molecules are examined in context to learning and memory and sensory response. The approach is helping unravel how key neurological molecules contribute to behaviours that underlie cognitive disorder endophenotypes. This work was published in *Molecular Psychiatry*.

Experimental honeybees live in a natural environment, the hive.

Laboratory Head **Dr Robert Colvin**



2011 Laboratory Members: Above: Robert Colvin. *Absent:* Belinda Craig **Background:** Students in a classroom.

Using neuroscience to optimise delivery of learning

Education and learning research has discovered literally hundreds of effective teaching techniques that assist learning, ranging from modifications to the timing of lessons and assessment, to the use of visual aids, and providing examples and practice. Dr Colvin's laboratory, within QBI's Science of Learning Centre is addressing the question of what combination of those techniques, and in what proportion, will make the most effective lesson.

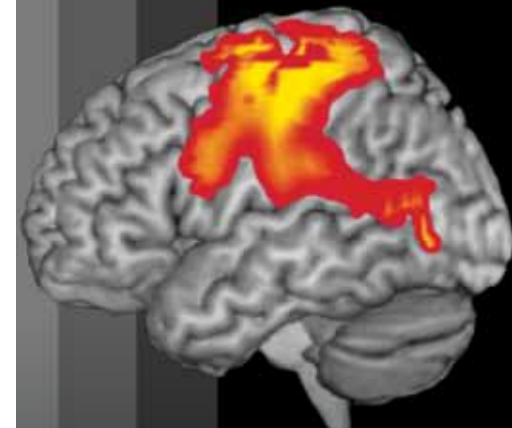
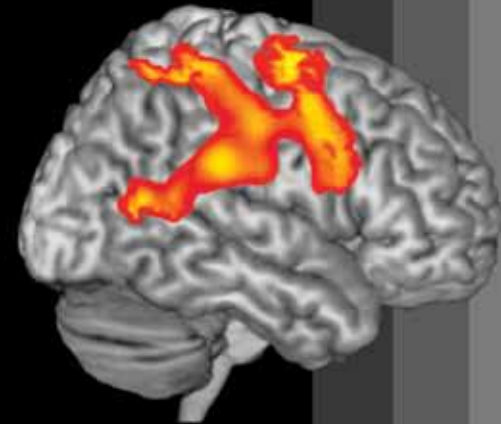
The Colvin group tackles this problem from a biological perspective, with a particular focus on how attention modulates our ability to store and recall memories. The laboratory works

with professional educators and education researchers on classroom-based studies in which teaching techniques are modified, within the complex constraints of a real classroom, to make effective use of our brain's mechanisms for strengthening memories.

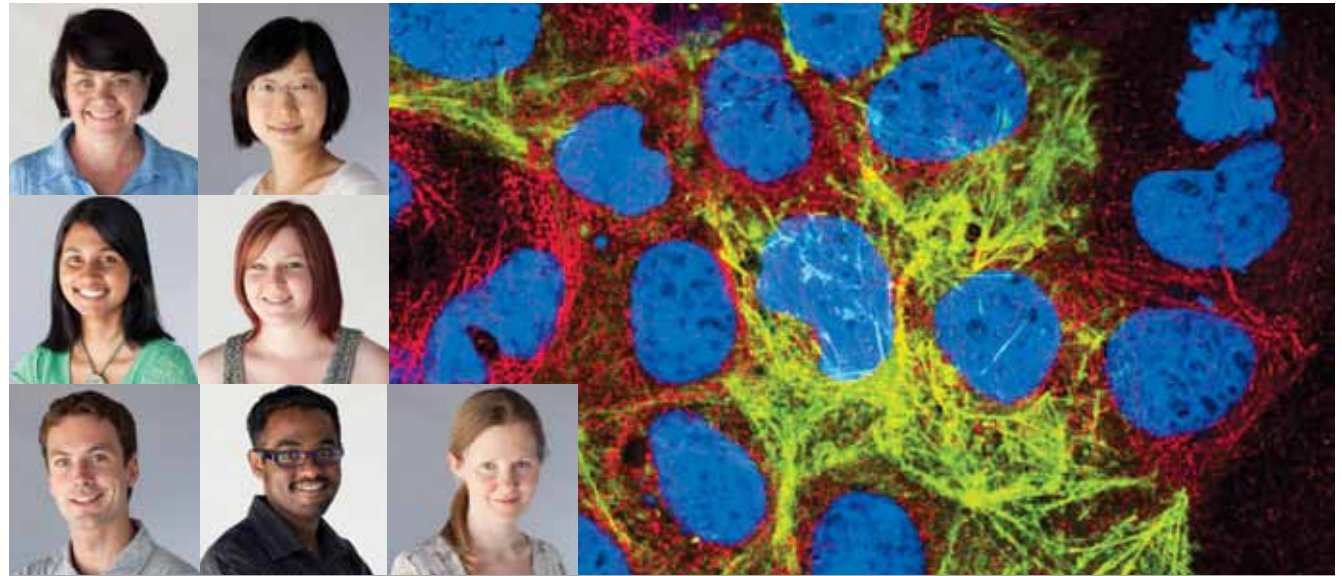
Underlying the research on memory optimisation are computational models which integrate at a high level of abstraction the biological components of learning. This research stream uses classical (or Pavlovian) conditioning as the basic paradigm by which memories (associations) are formed, and extends the models with notions of real-time

and attention constraints. The models are used to elucidate the connection between behaviour and brain function, and relate phenomena from the classroom to biology.

In 2011, the laboratory developed the underlying mathematical framework in which behavioural concepts such as performance on an exam can be related to the activity of regions in the brain, and in collaboration with a Brisbane state school, we began an investigation of the effects of attention training on struggling readers.



The motor network of the human brain is active when we plan voluntary actions, imagine movements, and even when we observe others performing actions.
Image: Cunnington Lab.

Laboratory Head **Assoc. Professor Helen Cooper**

2011 Laboratory Members L-R: Helen Cooper, Min Chen, Jayani Hewage, Casey Holding, Conor O'Leary, Murugesh Sheekar, Amanda White. *Absent:* Charlotte Clark, Haley Cox, Cathrin Nourse, Melissa de Vries. **Background:** Cytoskeletal scaffolding (actin, green; tubulin, red) within epithelial cells. Image Cooper lab.

Understanding the molecular mechanisms regulating the birth of new neurons

The early vertebrate embryo comprises a highly organised neural stem cell population which gives rise to all neurons in the central nervous system. The Cooper laboratory is working to understand how the local environment of the stem cell influences the decision to generate new neurons. The group has now identified two cell surface receptors that control the birth of new neurons in the embryonic cortex. The Neogenin receptor controls the initial events that designate one daughter cell as a neuron during stem cell division, whereas the Wnt receptor, Ryk, controls the next phase of neuronal differ-

entiation by relaying local environmental information to the newborn neuron, ensuring that it takes up the correct fate. Recent studies also show that Neogenin controls the birth of new neurons in the adult brain. Understanding these fundamental processes is of major importance, as aberrant division and subsequent failure in neurogenesis is linked to debilitating conditions such as epilepsy, schizophrenia, and autism.

The Cooper laboratory is also heavily involved in The NanoNeuro Project. Presently, there are no effective therapies to combat conditions such as

Alzheimer's or Huntington's disease. This project aims to develop a novel class of nanoparticles as an effective drug delivery system for the treatment of neurodegenerative disease. This collaborative project between the Cooper laboratory, Professor Perry Bartlett, Professor Max Lu and Dr Zhi Ping Xu of the Australian Institute for Bioengineering and Nanotechnology has demonstrated for the first time that these nanoparticles can efficiently deliver small interfering RNAs to neurons in the adult mouse brain, indicating that they may have great clinical potential.

Loss of the cell surface receptor Neogenin results in severe disruption to the developing zebrafish brain (section through forebrain). Image Cooper lab.

Laboratory Head **Assoc. Professor Elizabeth Coulson**



2011 Laboratory Members L-R: Elizabeth Coulson, Fabienne Alfonsi, Earlene Ashton, Zoran Boskovic, Sophie Hill, Nicola Marks, Dusan Matusica, Linda May, Nick Palstra, Aanchal Sharma, Sune Skeldal. **Absent:** Georg Kerbler, Mei-Fong Ho, Mirela Wagner. **Background:** MRI image of the connections between the basal forebrain and the hippocampus. Image Georg Kerbler.

Promoting neuron survival in Alzheimer's disease with neurotrophic factors

The Coulson group aims to understand the factors which promote the survival of nerve cells throughout the life of the organism. By understanding the molecular and environmental factors that control neuronal survival, their goal is to develop molecules that can mimic naturally occurring mechanisms to treat neurodegenerative diseases.

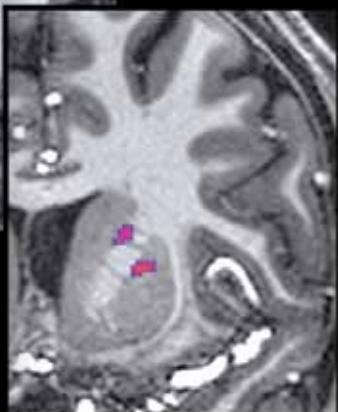
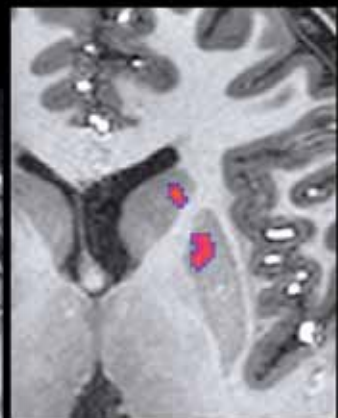
The prevailing theory of Alzheimer's disease, the most common form of ageing dementia, is that an endogenous amyloid- β peptide (A β) is either overproduced or poorly cleared from the brain, resulting in neurotoxicity. Although high levels of A β may be the proximal cause of the neuro-

degeneration that underpins cognitive decline associated with the condition, the aetiology of disease in the 90 per cent of people who develop it sporadically is unknown, with age alone being the greatest risk factor.

The Coulson laboratory has delineated a key molecular pathway by which A β causes neuronal death. This has led to the identification of two molecules, the p75 neurotrophin receptor and a potassium channel Kir3, which if disabled, can prevent neuronal death. They have also demonstrated that this pathway is normally regulated by neurotrophic factors, the production of which declines in humans as they

age. The group hypothesises that dysregulation of these neurotrophic factors may be a major trigger of dysfunction of neurons that reside in the cortex and hippocampus - the seats of memory and executive thinking - resulting in increased production of A β and hence neurodegeneration. They have recently devised a novel magnetic resonance imaging method of assessing the extent of neuronal degeneration in a specific brain region (the basal forebrain) in people with suspected Alzheimer's disease which, together with animal models of the condition, is being used to test this theory.

Phase contrast micrograph of neuronal PC12 cells showing extensive neurite growth following treatment with a p75-derived peptide. Image Dusan Matusica.

Laboratory Head **Assoc. Professor Ross Cunnington**

"The basal ganglia in detail." - With ultra-high resolution fMRI, function can be probed within specific parts of the basal ganglia as people perform simple finger movements inside the MRI scanner. This shows activity within the striatum - the area of the brain most affected by Parkinson's disease. Image Cunnington lab.



2011 Laboratory Members L-R: Ross Cunnington, Katharine Baker, Veronika Halász, Kian Ng, Vinh Nguyen, Simmy Poonian. *Absent:* Megan Campbell, Luis Sebastian Contreras Huerta, Shashenka Milston, Aaron Warren. **Background:** Setting up for a functional MRI brain imaging experiment on the UQ 3 Tesla MRI scanner.

Decoding brain activity during action planning and perception

Research in the Cunnington laboratory focuses on the brain processes involved in planning and preparing for voluntary actions, and for perceiving and understanding the actions of others. Whenever we plan, imagine, or observe others performing actions, representations of those actions are encoded in the motor areas of the brain.

Research from the Cunnington laboratory has revealed how, when we observe others' actions, specialised regions of the brain appear to be important for decoding the intention or goal of the person's action. As humans, we are very quick to understand the intentions or goals

of people's actions. This seems to be a highly automatic process, mediated by specialised areas of the brain, through the "mirroring" or simulation of others' actions in our own brain states.

The Cunnington group is discovering how neural predictions of others' actions and goals can influence the way we perceive those actions, and how other events that occur at the time of the action – sudden sights or sounds – get bound together in the brain so that we often perceive those events as being caused by the action. They are also examining how such "mirroring" for action understanding is influenced by social relationships such as team-membership and race.

Using new technology for brain imaging, the group is also combining electroencephalography and functional magnetic resonance imaging (fMRI) to examine the temporal dynamics of human brain activity, and using machine-learning computational methods to decode brain activity in real time. Using ultra-high resolution human fMRI they are now able to see brain activity in small regions of deep brain areas, such as the basal ganglia and thalamus that are crucial to motor control, with never before achieved detail. These methods are crucial for understanding clinical disorders affecting the deep brain circuits, such as Parkinson's disease.

Laboratory Head **Assoc. Professor Darryl Eyles**



2011 Laboratory Members L-R: Darryl Eyles, Suzanne Alexander, Xiaoying Cui, Claire Foldi, Isabella Formella, Pauline Ko, David Kvaskoff, Pei-Yun Ashley Liu, Henry Simila.
Background: Research assistant Henry Simila assaying archived dried blood spots for 25 hydroxy-vitamin D levels. Low levels of vitamin D at birth have been correlated with increased risk of developmental psychiatric conditions such as schizophrenia.

Understanding brain development and psychiatric illness

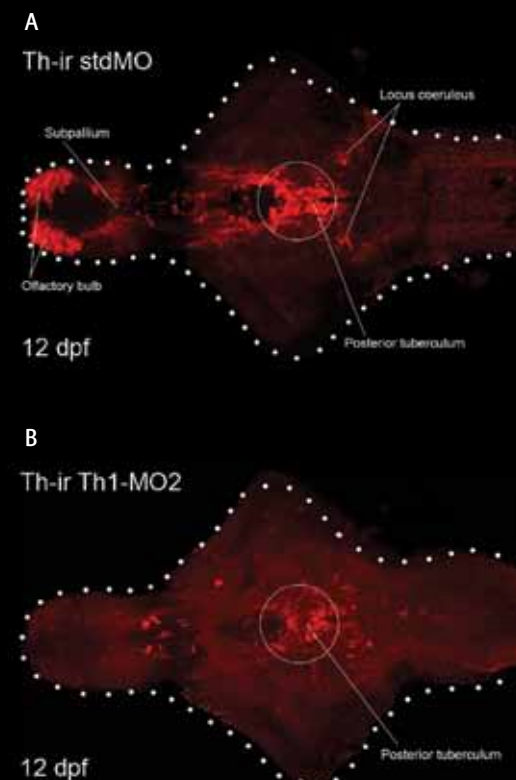
Adverse events *in utero* increase risk for serious psychiatric disorders such as schizophrenia. Research in the Eyles laboratory focuses on how known risk-factors for schizophrenia, including developmental vitamin D (DVD) deficiency, change the way the brain develops. The Eyles group has developed an extremely sensitive LC/MS/MS assay for vitamin D species in blood spot cards. This assay allowed the landmark study in 2010 implicating maternal levels of vitamin D as a risk factor for schizophrenia to be conducted.

Vitamin D has long been known to be important for calcium absorption and bone health, but it is only relatively recently that attention has

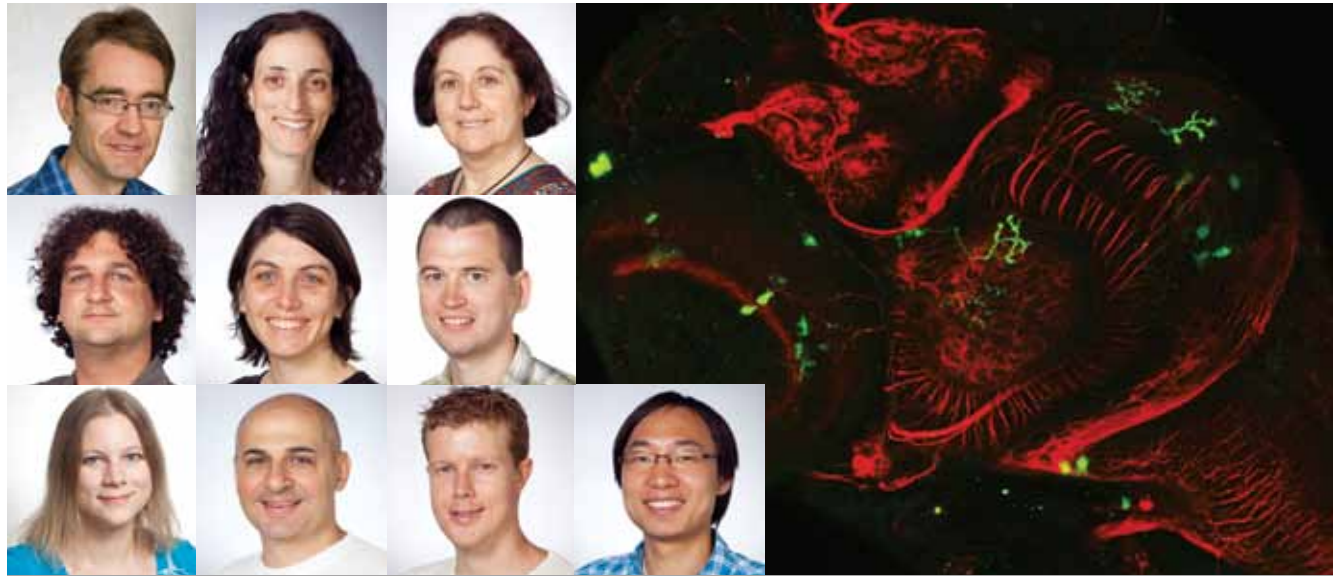
been directed to its role in cellular differentiation and brain development. In 2005 we mapped the distribution of the vitamin D receptor in human brain, and showed the close anatomical relationship between vitamin D signalling and metabolism in both rodent and human brain. Over the past 12 years the Eyles lab has been exploring what role vitamin D plays in the developing brain and how the maternal absence of this vitamin may affect brain function and behaviour.

They have recently discovered a mechanism of post-translational modification of the vitamin D receptor that may profoundly affect vitamin D signalling selectively in the developing brain.

Schizophrenia is also closely associated with abnormalities in dopamine transmission. The group's work in DVD-deficient animals confirms this. They have now fast-tracked some of their discoveries in rodent models into other model systems more amenable to high-throughput neuroscience such as the fruit fly and zebrafish. Using these model species the team has established models of restricted early transient impairments in dopaminergic development which they predict may be of aetiological relevance to what is happening in the developing human brain in schizophrenia.



Morpholino-induced knock-down of tyrosine hydroxylase-expressing neurons in a 12 day old zebrafish larva. Compare control (A) with the morphant (B). Altering tyrosine hydroxylase expression (rate limiting enzyme in dopamine synthesis) early in zebrafish brain development has been shown to alter anxiety-like behaviour in the adults.

Laboratory Head **Professor Geoffrey Goodhill**

2011 Laboratory Members L-R: Geoffrey Goodhill, Lilach Avitan, Maria Caldeira, Richard Faville, Clare Giacomantonio, Jonathan Hunt, Elizabeth Kita, Zac Pujic, Hugh Simpson, Jiajia Yuan. *Absent:* Clement Bonini, Elizabeth Forbes, Andrew Thompson. **Background:** Axons growing from the eye to the brain in the head of a larval zebrafish. Image Hugh Simpson

Using mathematical models to understand neural wiring development

For the brain to function properly, its neurons must be connected correctly. Research in the Goodhill laboratory uses a unique combination of experiments and theoretical modelling to understand how the nervous system becomes wired up during development. The laboratory's guiding philosophy is that building mathematical models allows a much more precise understanding of the underlying phenomena than relying on purely qualitative reasoning.

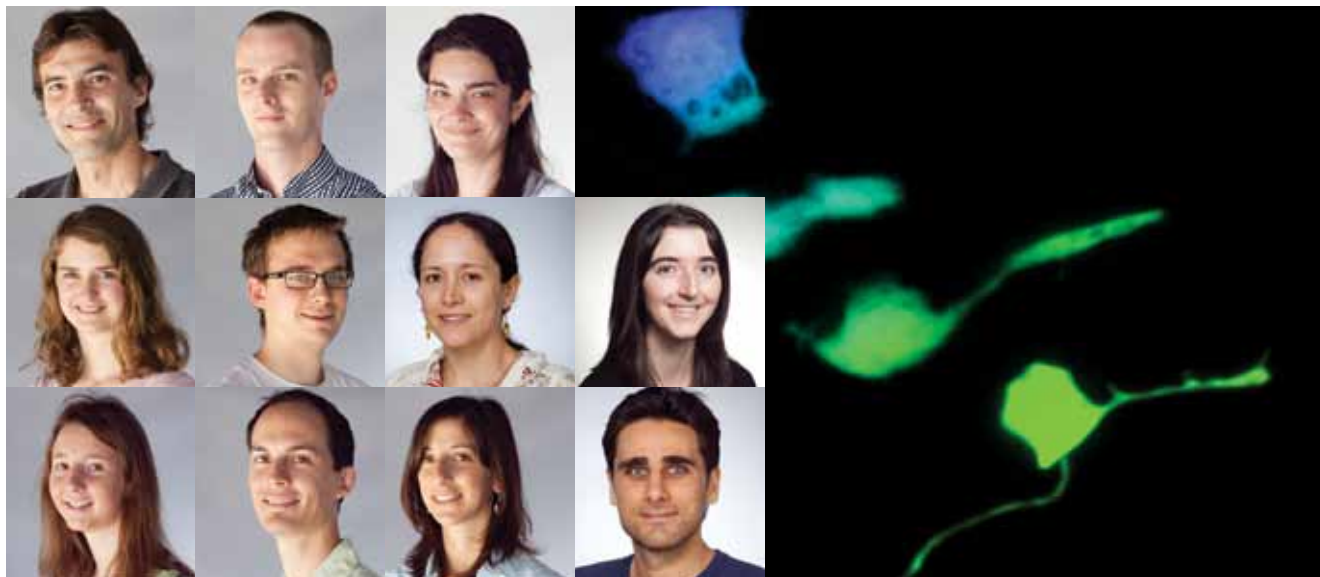
One area of focus for the Goodhill laboratory is how nerve fibres (axons) are guided by molecular gradients to find appropriate targets in the developing nervous system. The laboratory

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. This may help to explain the behaviour of developing and regenerating axons *in vivo*. The Goodhill group is also investigating the shape of growth cones, the structures at the tip of developing axons. Sophisticated mathematical techniques for characterising shape in general are currently being adapted to develop a more quantitative understanding of the role growth cone shape plays in effective axon guidance.

Further, the Goodhill laboratory is studying visual system development, particularly maps in the primary visual processing centres of the brain. Through a combination of theoretical modelling and timelapse imaging of real axons growing in the zebrafish brain, the laboratory has been investigating the mechanisms that lead to precise targeting of retinal axons in the brain. Theoretical predictions suggest that the precision of these connections may vary with the distribution of underlying molecular guidance cues; this prediction is now being tested experimentally.

Neuronal growth cone in culture. Stained for TrkA receptors (green) and F-actin (phalloidin). 100X confocal image. Image Zac Pujic.

Laboratory Head Dr Massimo Hilliard



2011 Laboratory Members L-R: Massimo Hilliard, Justin Chaplin, Paula Mugno, Annika Nichols, Sean Coakley, Leonie Kirszenblat, Casey Linton, Rhianna Knable, Brent Neumann, Rosina Giordano-Santini, Nicholas Valmas. **Background:** Different developmental stages of the PQR oxygen sensory neuron, with the dendrite (right) and axon (left) extending progressively. Image Hilliard lab.

Discovering the molecular mechanisms of axonal development & regeneration

Determining how individual neurons develop is crucial for understanding how highly complex neuronal structures, such as the brain and spinal cord, are formed. The Hilliard laboratory is interested in understanding how axons (nerve fibres conducting impulses from the neuron) and dendrites (nerve processes conducting impulses to the neuron) develop and how they are guided to their targets. The group also investigates how axonal structure is maintained over time and how it can be reconstituted after injury.

Neurons are highly polarised cells, the dendrites and axons of which form distinct morphologi-

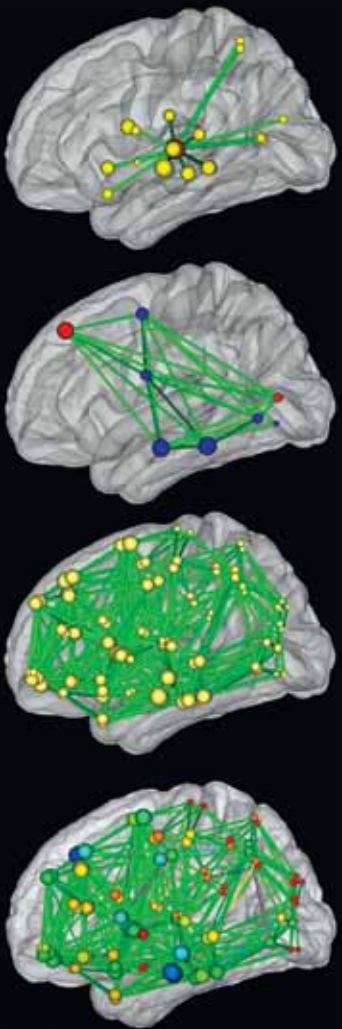
cal and functional domains. Using a *C. elegans* oxygen sensory neuron as a model system, the Hilliard group has identified two Wnt ligands and two Frizzled receptors that regulate dendrite development *in vivo*. The team has discovered that Wnt ligands can function as attractant cues for the developing dendrite.

The axon is the neuron's longest process, but the mechanisms that allow it to maintain its structural integrity and its regeneration capacity following injury are still poorly understood. The Hilliard group has identified mutant animals in which the axons of *C. elegans* mechanosensory

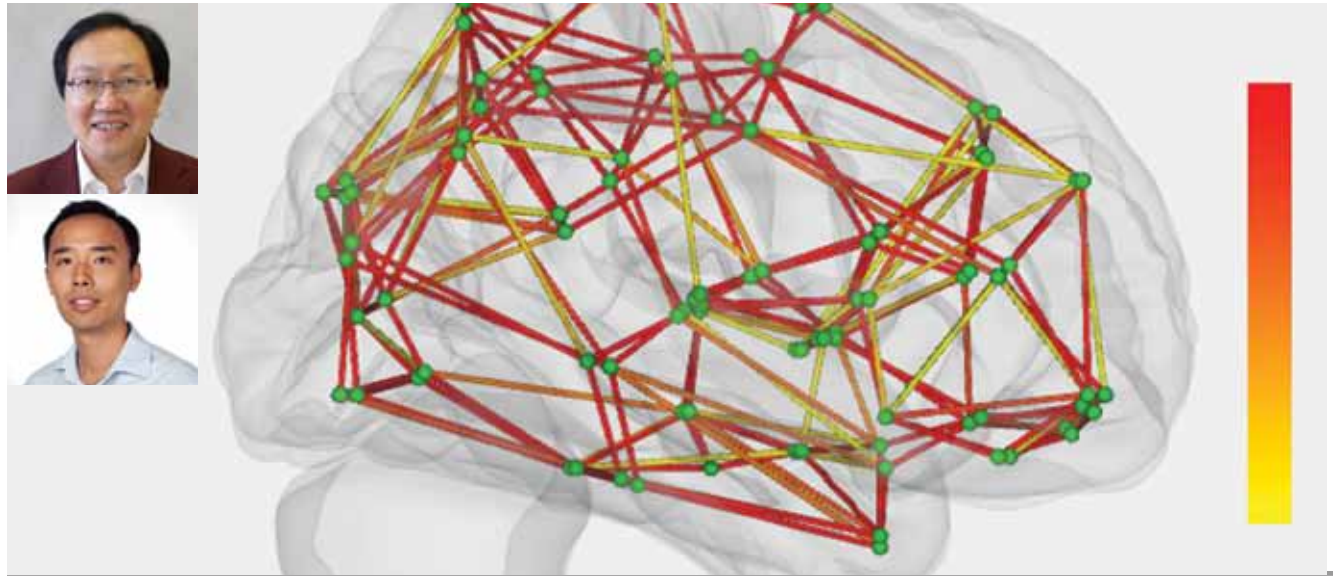
neurons spontaneously degenerate. Molecular identification of one of these genes has revealed a high level of conservation across animal phyla, including humans.

Using a laser-based technology to axotomize single neurons in living *C. elegans* animals, the team has also characterised neuronal regeneration in different classes of sensory neurons. They have shown that axonal regeneration can occur by a mechanism of axonal fusion, whereby the two separated axonal fragments can specifically re-attach and restore the original axonal tract.

The axon of the ALM mechanosensory neuron has regenerated bridging the injury site and restoring the original axonal tract. Image Hilliard lab.

Laboratory Head **Professor Tianzi Jiang**

On a macroscale, Brainnetome can be studied from at least four aspects: networks based on illness special region of interest, networks related to special cognitive function, whole brain networks, and performance of networks. Image Jiang lab



2011 Laboratory Members, top - bottom: Tianzi Jiang, Yonghui Li. Background: People with DISC1 risky allele have less global efficiency of brain networks based on diffusion MRI. Image Jiang lab.

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. "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.

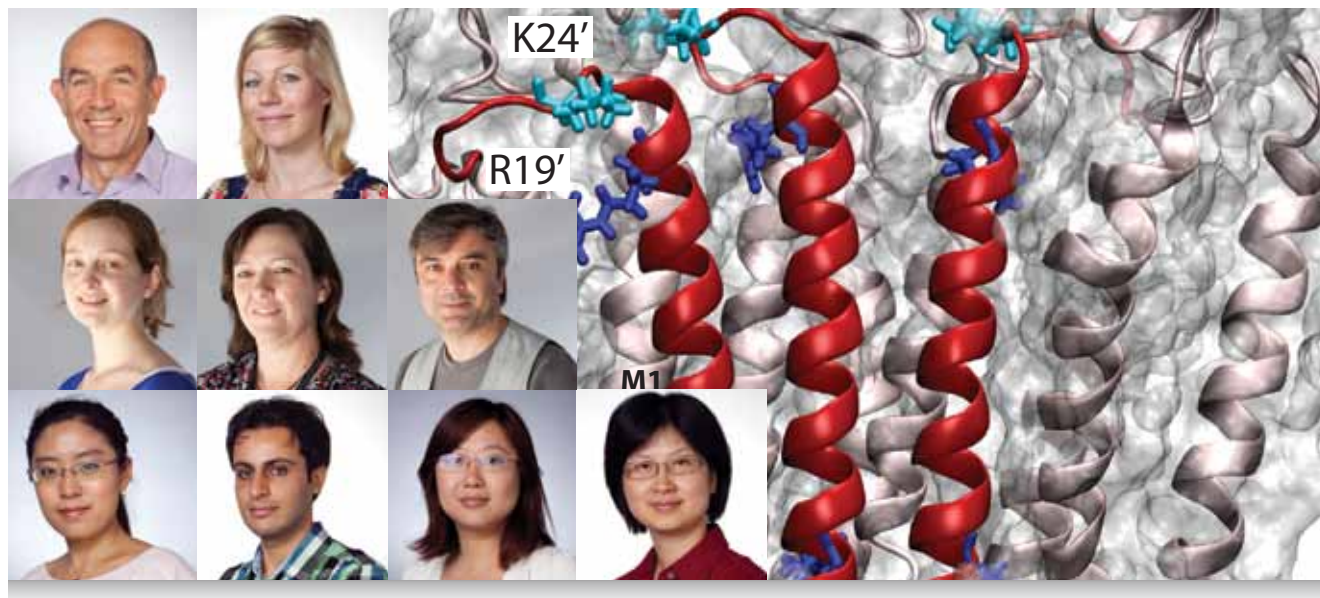
The Jiang laboratory is studying basic theory, methodologies and algorithms underpinning the Brainnetome platform, and their applications in neurological and psychiatric diseases. In 2011, their research has followed two streams. The first

of these involves the Brainnetome of age-related brain disorders, such as mild cognitive impairment and Alzheimer's disease. They have found that Alzheimer's disease patients have decreased long distance functional connectivity specifically between the default network regions. Moreover, the team has shown that the longer the distance, the more significant the alteration in functional connectivity, and the more severe the illness, the lower the connection strength and global efficiency in the patient group. These results provide direct support for the idea that Alzheimer's disease is a disconnection syndrome.

Another research stream is the Brainnetome

of neurodevelopment disorders, such as schizophrenia and autism. One such study is focussed on how the Disrupted-in-Schizophrenia-1 (DISC1) gene modulates brain anatomical network properties based on diffusion magnetic resonance imaging (dMRI). The group has found that in healthy young adults, A-allele carriers show significantly lower global efficiency of their brain network compared with TT homozygotes. This indicates that less efficient information transfer may be associated with the high-risk genotype of DISC1.

Laboratory Head Professor Joe Lynch



2011 Laboratory Members L-R: Joe Lynch, Anna Bode, Christine Dixon, Justine Haddrill, Angelo Keramidas, Han Lu, Sahil Talwar, Qian Wang, Yang Zhe.

Background: View of the ion pore of the glycine receptor showing amino acids that give rise to fluctuations in single channel current when they bind and unbind protons.

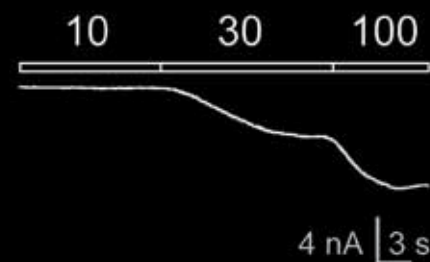
Inhibitory neurotransmitter receptors as therapeutic targets

The major research interest of the Lynch laboratory 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 group is attempting to identify the locations of drug binding sites on these receptors. They are also discovering new drugs active at these receptors which could lead not only to improved therapies but also to better tools for basic research.

Recent research in the laboratory has led to the development of an improved 'neuronal silencing receptor' for inhibiting electrical activity in defined populations of neurons in behaving animals. This technology holds promise for treating human neurological disorders that are caused by excessive levels of neuronal activity, including motor neuron disease, Parkinson's disease, addiction, anxiety and epilepsy.

Most synaptic receptors in the brain exhibit 'desensitisation', which is the progressive fading of ionic current in the prolonged presence of agonist.

This process involves conformational changes that close the channel despite continued agonist binding. Despite the physiological and pathological importance of desensitisation, little is known about the molecular basis of this process in any Cys-loop ion channel receptor. The Lynch group has recently discovered how this process occurs. This has important consequences for understanding how these receptors work and will help in designing new drugs that modulate these receptors in specific ways.



Location of a critical ivermectin binding determinant on the inhibitory glycine receptor.

Laboratory Head **Professor Justin Marshall**

2011 Laboratory Members L-R: Justin Marshall, Tsyr-Huei Chiou, Wen-Sung Chung, Angela Dean, Alan Goldizen, Martin How, Anna Kleine. *Absent:* Andy Dunstan, Adrian Flynn, Genevieve Phillips, Chris Talbot, Hanne Thoen. **Background:** The stomatopod crustacean *Odontodactylus cyllarus* catches prey in less than 50 milliseconds. The visual system of these crustaceans is one of our current research themes.

The coastal squid *Sepioteuthis lessoniana* is one of the marine animals whose visual system is being investigated in the Marshall laboratory. Neuroscience originally used cephalopods and crustaceans as models systems to discover basic neural function and we are following this lead to discover more about their sensory systems.

The Marshall laboratory is interested in “neuro-ecology”, the study of neuroscience in the outside world. The group specialises in visual systems and their animal models include stomatopods (a marine crustacean), cephalopods (octopus, squid, cuttlefish and Nautilus), fish (mostly the colourful assemblage of reef fish), turtles and occasionally birds. The environments they work in are largely marine (although birds and forests have also recently featured) and go from The Great Barrier Reef to the icy depths of the deep-sea. They also have an environmental wing, CoralWatch, an award winning citizen

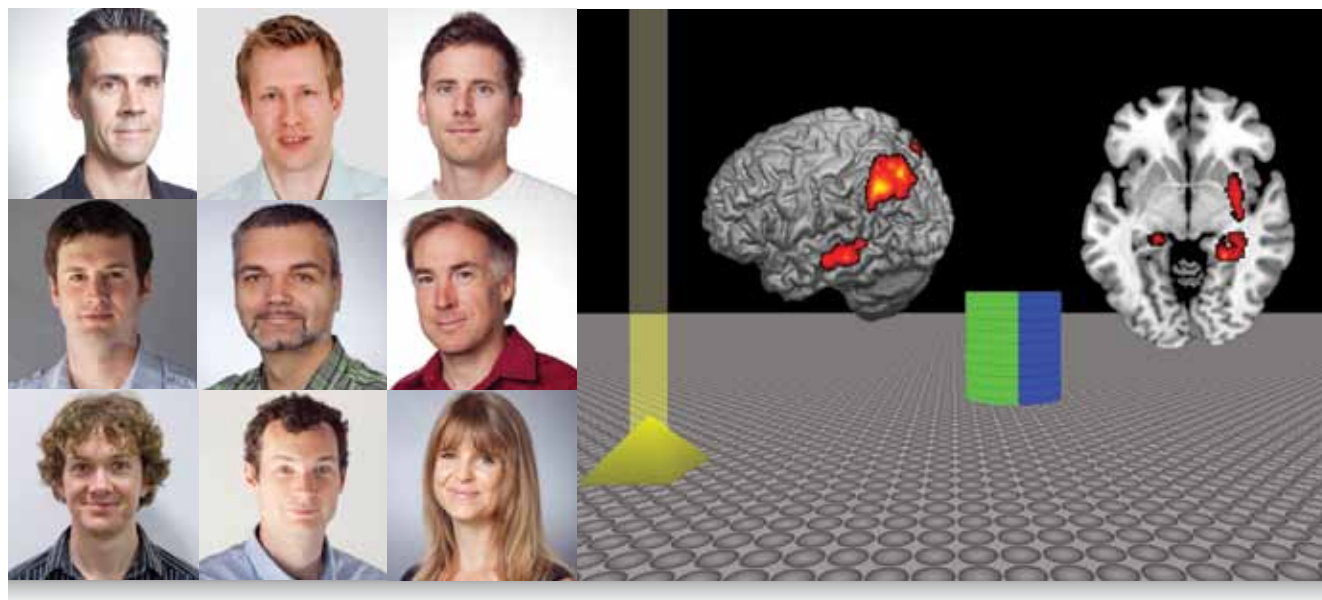
science program that communicates science direct to the public.

Highlights for 2011 include 15 publications from the team, all in top-tier journals, including *Current Biology* and a theme issue of *The Philosophical Transactions of the Royal Society* of which Professor Marshall was edition co-editor with long-term US collaborator Tom Cronin. The group has made significant discoveries in polarisation vision and colour vision with collaborators in the USA and UK. The Deep-Australia project received fresh input with two new

industry partners and a pair of 600m capable Deep-Worker submersibles that will be available for use in early 2012.

Stomatopod visual systems are now providing bio-inspiration for the nanofabrication of optical data storage devices and one new theme for 2012 will be to further explore this area with funding from the US Airforce and American colleagues. Our aim is to capitalise upon the power of evolutionary design to enhance the opto-electrical engineering of camera systems and other optical devices.

Laboratory Head Professor Jason Mattingley



2011 Laboratory Members L-R: Jason Mattingley, Oliver Baumann, Luca Cocchi, Oscar Jacoby, Marc Kamke, David Lloyd, David Painter, Martin Sale, Susie Travis. *Absent:* Michael Dwyer, Jill Harris, Will Harrison. **Background:** Neuroimaging and a virtual navigation task (behind) to isolate differences in the neural activity underlying the formation of navigation-based metric and categorical spatial representations. Brain images show neural activity as participants learn their way around a new environment.

Understanding selective attention in health and disease

Attentional processes are crucial for most cognitive functions in humans, including learning and memory. How does the brain select just a few important inputs from the myriad of data entering our senses?

In 2011, Mattingley lab researchers made several breakthroughs in understanding how attention operates in health and disease. Mr Will Harrison (PhD) is investigating what happens to visual attention when we move our eyes. Mr Harrison's work has shown that the brain effectively anticipates each eye movement a fraction of a second before it occurs, giving the visual system

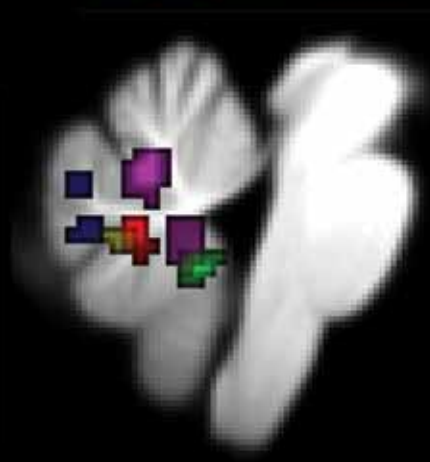
a "head start" in updating its current picture of the visual world.

In related work, Dr Michael Dwyer (MPhil) and Mr David Painter (PhD) have used brain recording methods to investigate changes in the brain that occur when people lose part of their vision due to macular degeneration. Their findings have led to a better understanding of how the brain reorganizes itself when the senses are compromised due to ageing or disease.

Mr Oscar Jacoby (PhD) and Ms Susie Travis (PhD) have examined the processes that enable the human visual system to search rapidly and efficiently through cluttered visual environments,

such as looking for a set of car keys on a messy desk. Their work has shown that the brain uses predictable elements within visual scenes to improve search efficiency.

Post-doctoral fellows Dr Marc Kamke, Dr Martin Sale and Dr Luca Cocchi have used non-invasive brain stimulation to examine how attention and training influence neural plasticity. They have also used brain imaging combined with novel analytic methods to uncover changes in the brain's functional networks after local stimulation of the motor cortex. This work has important implications for people who lose limb mobility after a stroke.



The cerebellum has an important and well-known role in the control and coordination of movements. Here neuroimaging was used to investigate the role of the human cerebellum in the regulation of emotions. Participants were shown emotion-evoking pictures (see examples top and middle) while undergoing fMRI measurements of their neural activity. The bottom image displays emotion-related activity in the cerebellum for five primal emotions (Happiness=Red; Anger=Green; Fear=Blue; Sadness=Yellow; Disgust=Purple).

Laboratory Head **Professor John McGrath**

2011 Laboratory Members L-R: John McGrath, Suzanne Alexander, Trudi Flatscher-Bader, Pauline Ko, Pei-Yun Ashley Liu, Henry Simila. **Background:** Painting by Glenn Brady, *The Schizophrenic, the bipolar and the manic-depressive*, acrylic on canvas. Image thanks to QCMHR Gallery.

Modifiable risk factors for schizophrenia

Schizophrenia is a poorly understood group of brain disorders that affects about one in a hundred Australians. The aim of the McGrath group is to explore risk factors that are linked to schizophrenia. In particular, they focus on nongenetic 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. In collaboration with Dr Darryl Eyles and Dr Tom Burne, they have developed animal models that examine the impact of low vitamin D during gestation on brain development. Recently the group and their Danish collaborators have found that low

vitamin D levels at birth double the risk of later developing schizophrenia.

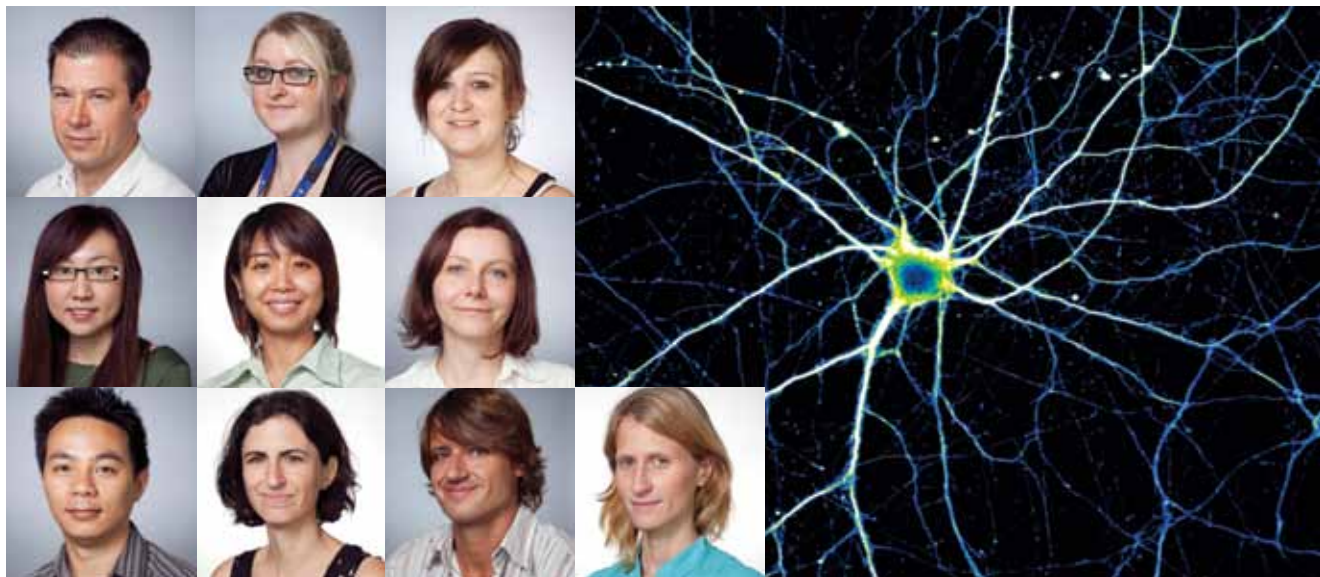
The group has also found that the offspring of older fathers have an increased risk of schizophrenia. In 2011 they published findings of a mouse model of advanced paternal age. In collaboration with Professor Emma Whitelaw at the Queensland Institute of Medical Research, Dr Trudi Flatscher-Bader from the McGrath team reported that the offspring of older males have a significantly increased risk of a type of genetic mutations (copy number variants). This study also found that paternal age was associated with

mutations in genes linked to autism and schizophrenia. Age of parenthood is increasing in many societies and it is feasible that paternal-age related genetic mutations will increase in the years ahead.

This research raises the tantalizing prospect of the primary prevention of schizophrenia. It is feasible that the use of vitamin D supplementation in at-risk groups could reduce the incidence of schizophrenia, in a manner comparable to folate supplementation and the prevention of spina bifida.

The McGrath laboratory investigates the genetic risk factors associated with paternal age. Image Garrison Photography.

Laboratory Head **Assoc. Professor Frederic Meunier**



2011 Laboratory Members L-R: Frederic Meunier, Rachel Gormal, Callista Harper, Pei C Low, Nancy Malintan, Sally Martin, Tam Hong Nguyen, Shona Osborne, Andreas Papadopoulos, Vanesa Tomatis. **Absent:** Peter Wen. **Background:** Even in culture, neurons create extremely complicated networks of neurites to connect with other cells. These connections are necessary for communication between the cells and are useful models to study what is occurring in the brain. Image Callista Harper.

Deciphering the mechanism underpinning neuronal communication

In 2011, the Meunier laboratory has focused on the major role played by vesicular trafficking in health and disease.

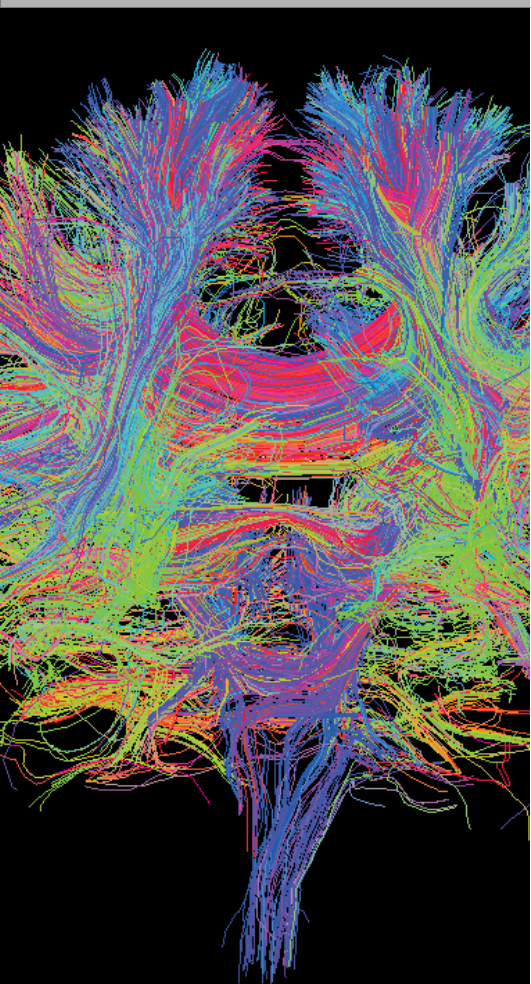
In a paper published in the international journal *Nature Communications*, the group has demonstrated that the lipid (fat) from the membranes of brain cells controls the movement of vesicles containing chemical messengers called neurotransmitters. Their results show that the lipid phosphatidylinositol(4,5)bispophosphate orchestrates the mobilisation and movement of secretory vesicles towards the plasma membrane of the cell. These findings explain

how minute variations in the lipid composition of our neurons can have dramatic effects on the way in which these vital cells communicate with each other in the brain, with changes in lipid composition already being shown by others to be a contributing factor to the development of dementia in Alzheimer's disease. The team therefore hopes that developing novel compounds targeting the fat lipid composition of biological membranes could ultimately help in the treatment of such brain disorders.

In another study published in the *Journal of Biological Chemistry*, the Meunier laboratory

has demonstrated that botulinum neurotoxin type-A exploits synaptic vesicle recycling and endosomes to gain access to neurons, leading to intractable flaccid paralysis. This process requires the activity of dynamin-1. In collaboration with the groups of Phil Robinson (University of Sydney) and Adam McCluskey (University of Newcastle), who provided a highly effective dynamin inhibitor, the team has demonstrated that such drugs could be used to prevent toxins such as botulinum neurotoxin from entering our neurons.

A cultured neuron treated with two toxins (red and green) that are used to study trafficking pathways. The blue staining (VAMP2) shows where the neuron connects with other cells, which is often where endocytosis occurs. Despite the majority of both toxins being taken up in the same regions of the cellular membrane, they follow very different pathways within the neuron. Image Callista Harper.

Laboratory Head **Professor Bryan Mowry**

White-matter images acquired using Diffusion Tensor Imaging (DTI) as part of ongoing study into endophenotypes of schizophrenia.



2011 Laboratory Members L-R: Bryan Mowry, Cheryl Filippich, Jake Gratten, Chikako Ragan, Vasilis Mantzioris, Kalpana Patel, Lauren Simpson, Heather Smith, Xi Yao. **Absent:** Andrew Martin, Duncan McLean. **Background:** Researcher Kalpana Patel loading a microtitre plate on to a real time PCR machine.

Psychiatric genomics

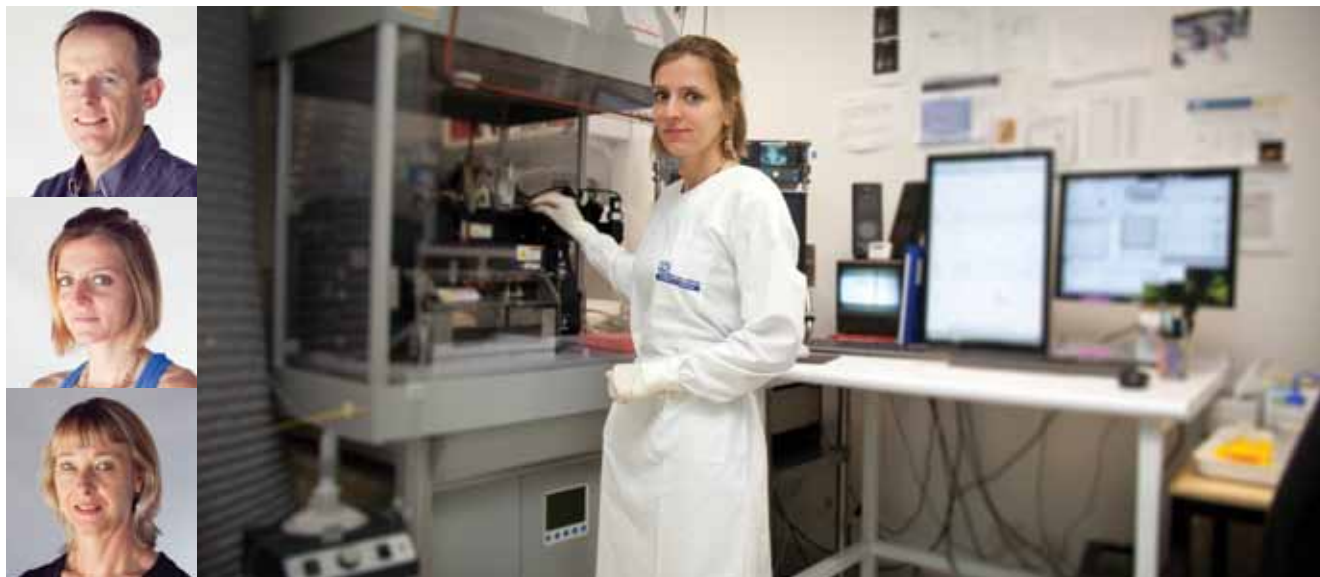
The Mowry group aims to identify and functionally characterise susceptibility genes for schizophrenia and related disorders. The group achieves this by combining genome-wide association studies (GWAS), DNA sequencing and transcriptome profiling with neuropsychological testing and neuroimaging in people with schizophrenia. Ongoing studies include (i) the recruitment of a large Indian case-control cohort in collaboration with Dr Thara, Schizophrenia Research Foundation, Chennai, India (ii) neuroimaging and neuropsychological phenotyping of schizophrenia patients with major copy number variations, (iii) GWAS in homogeneous Indian and Sarawak populations, and (iv) involvement in the

Australian Schizophrenia Research Bank.

During the year, the group made a substantial contribution to the landmark Psychiatric Genetics Consortium schizophrenia GWAS, reported in the September issue of *Nature Genetics*. They contributed approximately 950 individuals (multiplex schizophrenia families, affected sibling pair pedigrees and unrelated cases) in the discovery phase, and coordinated the Australian component (558 schizophrenia cases; 957 controls) of the replication phase. Other highlights included two applications of next-generation sequencing technology (a pilot RNA-Seq study of post-mortem schizophrenia

brain samples; targeted resequencing of a schizophrenia linkage region) development of a neuro-immunology of schizophrenia project involving new group member, Dr Vasilis (Bill) Mantzioris, and a collaborative induced pluripotent stem cell study involving samples from people with schizophrenia and healthy controls. The group includes four PhD students: Chikako Ragan (small non-coding RNAs in schizophrenia); Andrew Martin (neuropsychological and neuroimaging endophenotypes in schizophrenia); Xi Yao (statistical analysis of rare genetic variants in schizophrenia), and Duncan McLean (Analysing the expression of psychotic disorders in ethnically different populations).

Laboratory Head Mr Geoffrey Osborne



2011 Laboratory Members, top - bottom: Geoffrey Osborne, Anne-Sophie Bedin, Virginia Nink. Absent: John Wilson. Background: Researcher Anne-Sophie Bedin using the BD Influx cell sorter.

Laying the groundwork for brain tumour therapies

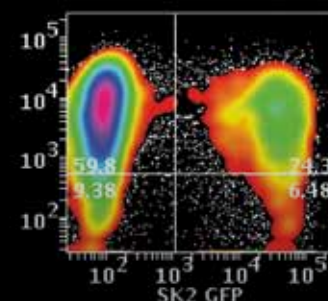
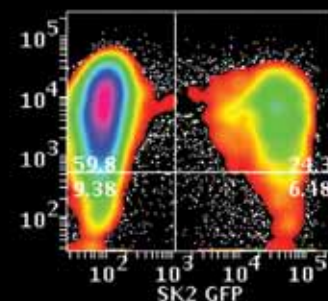
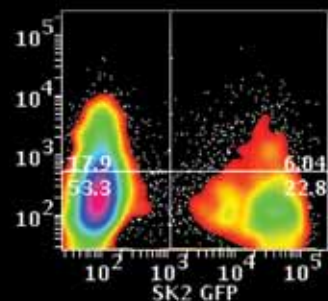
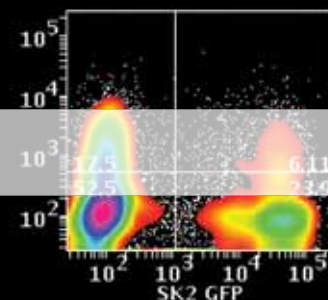
Mr Geoffrey Osborne is unique among QBI researchers, in that he combines his work as a neuroscientist with a facility management role. As Director of Flow Cytometry for both QBI and the Australian Institute for Bioengineering and Nanotechnology, Mr Osborne leads a team that provides crucial cell sorting 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.

The research focus of the laboratory encompasses a wide range of neuroscience areas where it is

felt that flow cytometry is, or could become, an important technology. As in previous years, investigations continued in the area of characterisation of cells from brain tumours. These studies focus on the heterogeneity of cell surface receptors present on tumour cells, as these important receptors regulate interactions that occur between the cell and its immediate environment. This year saw the publication of work showing that even within heterogeneous populations of cells, identifiable sub-populations have different tumour-forming potentials, which goes some way to explaining tumour resistance to current treatment approaches. Other related

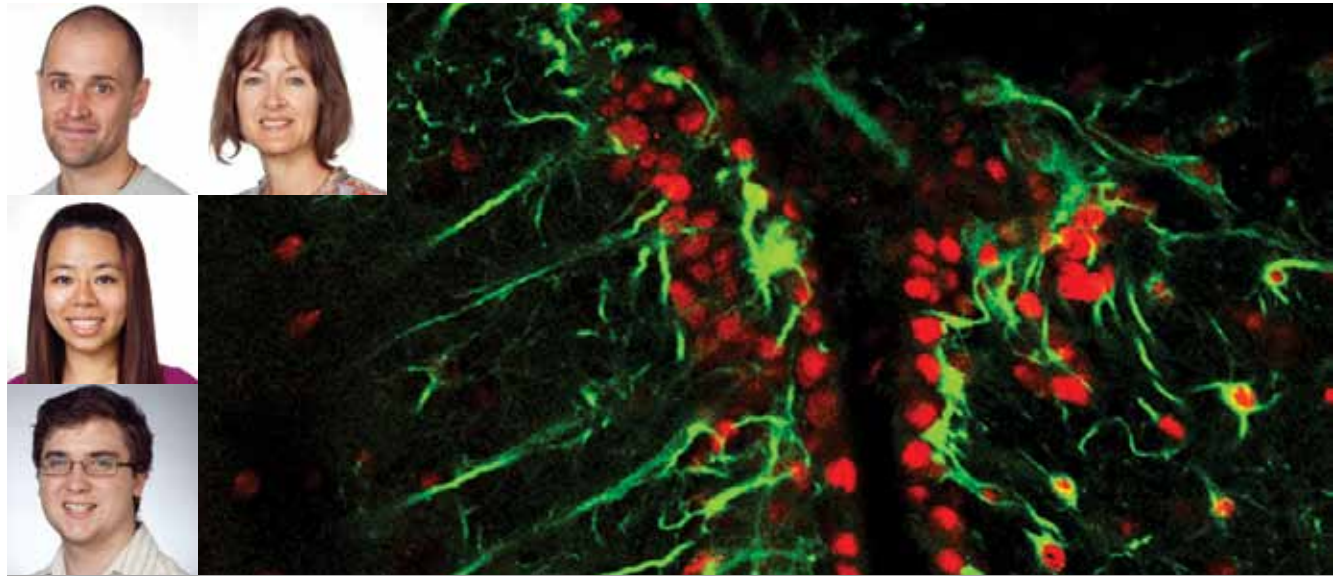
published work focused on the detection and flow cytometric separation of cells that are newly committed to differentiating down a neuronal lineage. These cells have potential use for assay development or transplantation. Given that the first clinical trials for transplanted cells for the treatment of spinal injury have recently been approved, this approach may prove important in the future.

Other collaborative projects to develop specific antibodies for neural stem cells and ion channel targets are ongoing, with putatively clones developed and undergoing testing for specificity.



Studies utilising antibody phage display libraries are performed to identify specific fragments of antibodies that can bind specifically to ion channels of interest in the neurosciences. Image Osborne lab.

Laboratory Head Dr Michael Piper



2011 Laboratory Members L-R: Michael Piper, Kathleen Cato, Evelyn Heng, Kynan McInnes. *Absent:* Chantelle Reid. **Background:** Expression of the glial marker, GFAP (green), and the transcription factor NFIA (red), in the subventricular zone of the adult mouse brain. The subventricular zone is one of the few places in the adult brain that makes new neurons throughout life.

Neural stem cell differentiation

Glial supporting cells in the hippocampus of an embryonic mouse brain show in white. The hippocampus is a part of the brain that is crucial for learning and memory.

The regulation of cellular proliferation and differentiation is critical during both development and disease. For instance, controlling the balance between the proliferation of neural progenitor cells and their subsequent differentiation into neurons or glia during development is central to the formation of the brain. Moreover, when proliferative cells in the adult brain fail to differentiate correctly, brain cancers such as glioma can arise. 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 they are investigating the role of a suite of transcription factors called the Nuclear Factor One (Nfi) family, and investigating how they drive the differentiation of neural progenitor cells. In 2011 in collaboration with QBI's Professor Linda Richards, the group has shown that Nfi members are required for the formation of the cerebellum, a part of the brain crucial for movement, and elucidated some of

the genes that act to regulate the expression of the Nfi genes during development. They have also shown that these transcription factors are vital for neurogenesis in the adult brain, and that they are misregulated in brain cancers including glioma. Recently, the team has also begun to discover the transcriptional targets of the Nfi proteins, work that aims to reveal the genetic hierarchy that controls neural progenitor cell differentiation.

Laboratory Head **Dr Judith Reinhard**



2011 Laboratory Members L-R: Judith Reinhard, Nicole van der Burg, Alexandre Cristino, Nivetha Gunasekaran, Shao-chang Huang, Homayoun Kheyri, Amanda Robinson. *Absent:* Shanzhi Yan. **Background:** Nicole van der Burg preparing a mixture of selected odorants to test human scent perception.

Uncovering mechanisms underlying scent perception in humans

Researchers in the Reinhard laboratory investigate how the brain processes sensory information from the environment and translates it into behavioural activity, thus linking brain function to behaviour. A particular focus is the sense of smell and its effect on memory, social behaviour, and cognitive performance. The laboratory uses insect model systems such as the honeybee and the fruit fly in combination with human research, and integrates behavioural studies with physiological and molecular approaches.

Natural scents such as floral bouquets or food aromas are complex mixtures, composed of

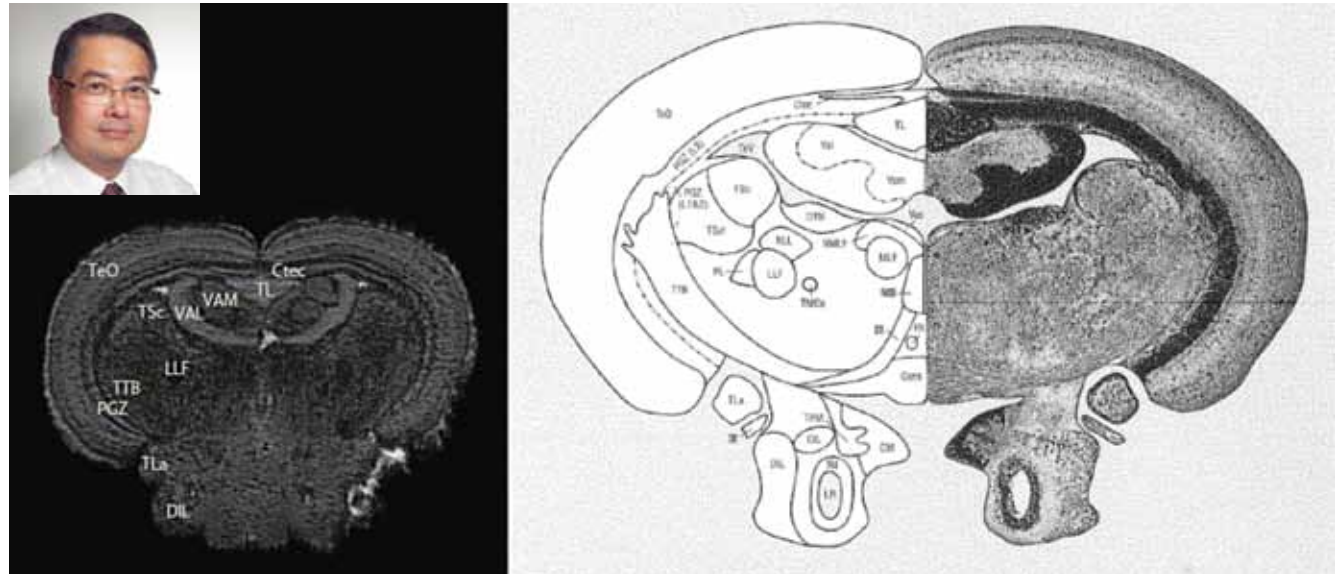
hundreds of different odorants. The Reinhard laboratory had previously demonstrated that honeybees encode such natural scents via a selection of key odorants. In 2011, they investigated how the human sense of smell recognises complex natural scents. A large-scale behavioural study revealed that humans also use the key odorant strategy of scent processing. That is, our brains filter out the majority of olfactory information from an aroma and we only learn a specific key odorant signature for each complex scent. This simple reductionist strategy helps the brain to manage the massive amount of information contained in natural

scents, thereby enhancing processing speed and capacity.

The study also revealed that the environment in which we live significantly affects key odorant signatures. People who had lived in Australia for several years all had similar key odorant signatures, irrespective of their gender, age, or genetic background. This exciting new discovery suggests that our sense of smell is not hard-wired, but highly plastic, adapting throughout life to the scent environment surrounding us.



Honeybee tongue in a drop of scented sugar water during olfactory conditioning of the bee.

Laboratory Head **Professor David Reutens**

2011 Laboratory Member, above: David Reutens. Background: Zones of a Zebra fish brain.

Creating images to explore living organisms

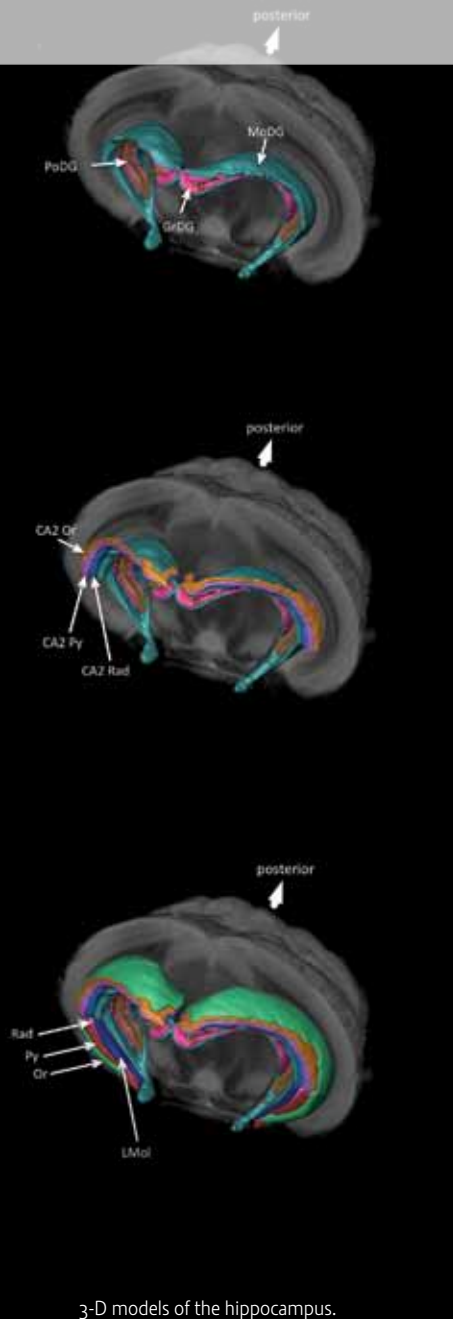
The emergence of powerful new imaging techniques such as magnetic resonance imaging (MRI), electroencephalogram (EEG), transcranial magnetic stimulation (TMS) and positron emission technology (PET) has significantly driven advances in neuroscience in recent times. The Reutens laboratory takes full advantage of these advances in technology to study functions such as memory in the healthy brain, the mechanisms behind diseases such as epilepsy and stroke and how the brain responds to overcome injury.

One aspect of the Reutens group research is the creation of *in vivo* mouse brain atlases using MR microscopy. These atlases make it possible to study and compare *in vivo* changes over time of diseased and healthy brains. The group is also examining new ways of mapping neuronal activation with MRI using diffusion and ultra-low field imaging techniques. This year the group has commenced another project to create a zebrafish MRI library.

The Reutens laboratory continues the development of biomarkers of epileptogenesis,

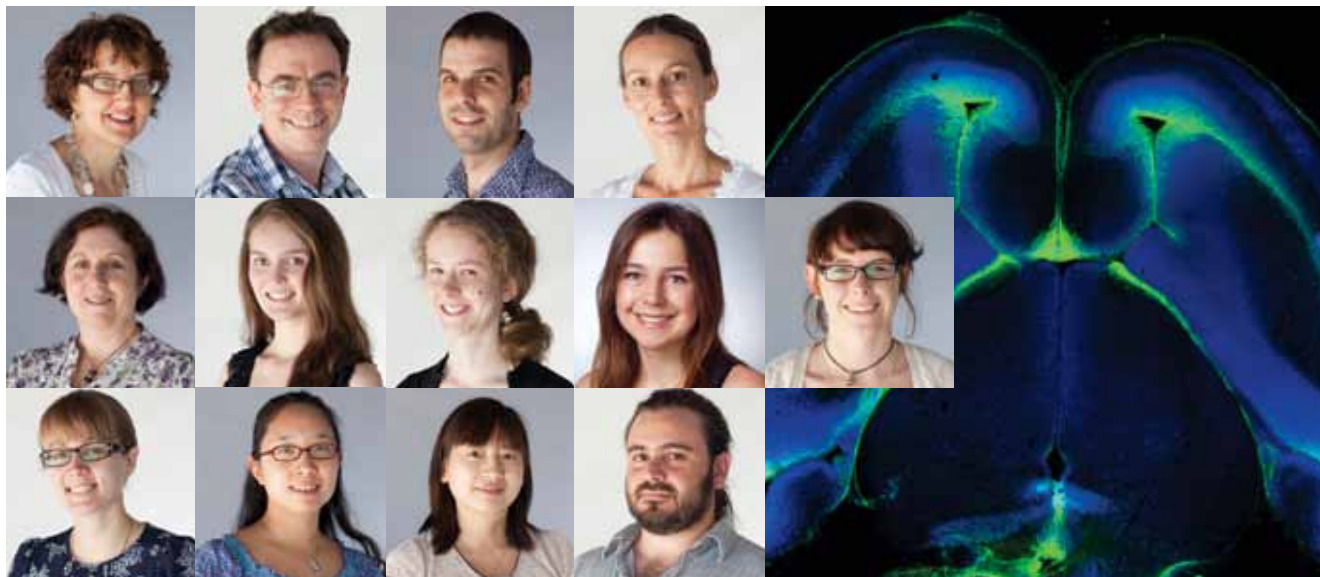
correlating imaging changes with spike seizure frequency and with histological changes in mouse models. These biomarkers may be used to test novel antiepileptogenic therapies.

Meanwhile, another group of researchers in the laboratory is working on new ways of detecting brain hypoxia using novel MRI contrast agents and susceptibility imaging techniques, resulting in two *Journal of Chemical Crystallography* papers in 2011.



3-D models of the hippocampus.

Laboratory Head Professor Linda Richards



2011 Laboratory Members L-R: Linda Richards, John Baisden, Jens Bunt, Ilse Buttiens, Maria Caldeira, Lauren Crumlish, Amelia Douglass, Tess Evans, Ilan Gobius, Katelin Haynes, Samantha Liu, Yolanda Liu, Rodrigo Suárez. *Absent:* Tim Edwards, Justin Jin, Gayeshika Leanage, Sharon Mason, Laura Morcom, Wachizya Nyirenda, Jeffrey Thompson, David Yap. **Background:** Glial cells (green) in an embryonic mouse brain shown in horizontal section.

Wiring the brain for function

Prior to birth, nerve cells form long processes called axons that connect to other neurons creating functional circuits in the brain. Researchers in the Richards laboratory are studying how the brain forms these connections during embryonic and foetal development. This year marked a new phase in the laboratory with the departure of a number of students and postdoctoral fellows and the recruitment of new scientists to join the team. Of special note was the departure of Dr Michael Piper who became a group leader in the School of Biomedical Sciences, UQ and QBI. Ongoing projects include our work on the nuclear factor one genes and

resulted in two publications this year, one in the *Journal of Comparative Neurology* and another in the *Proceedings of the National Academy of Sciences* in collaboration with Dr Shubha Tole, Tata Institute, Mumbai. Imaging work led by postdoctoral scientist Dr Randal Moldrich resulted in one paper in *Developmental Dynamics*, as a collaborative effort with Tomoko Iwata's laboratory in Scotland.

Two new postdoctoral scientists joined the laboratory this year: Dr Rodrigo Suárez from the University of Chile, Santiago, Chile and Dr Jens Bunt from the Academic Medical Centre,

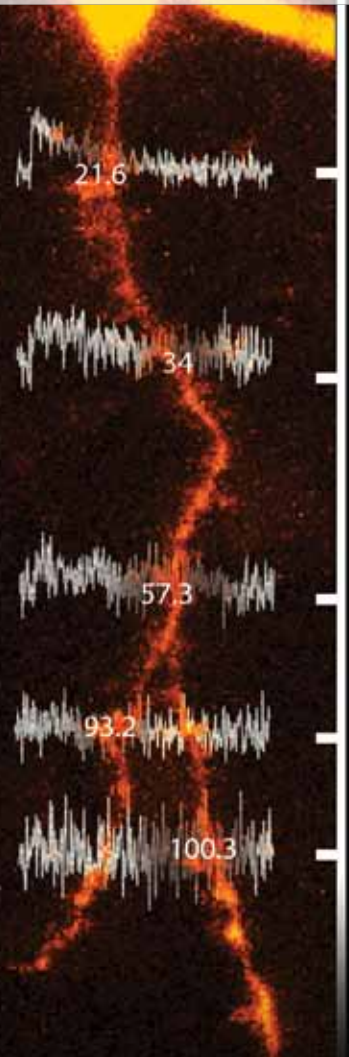
Amsterdam, The Netherlands. Rodrigo is pioneering our efforts to understand callosal axon targeting in the contralateral hemisphere. Jens is beginning our efforts to understand the array of genes regulated by the nuclear factor one family of transcription factors in brain development and disease. Both Rodrigo and Jens are bringing exciting new approaches to the laboratory and we look forward to reporting their progress next year. Finally Amber-Lee Donahoo successfully completed her PhD and graduated this year, and Amelia Douglass completed her honours degree in the Bachelor of Biomedical Sciences program.

Pyramidal neurons (green) in the somatosensory cortex of the mouse labeled with green fluorescent protein. Image Rodrigo Suárez.

ventral

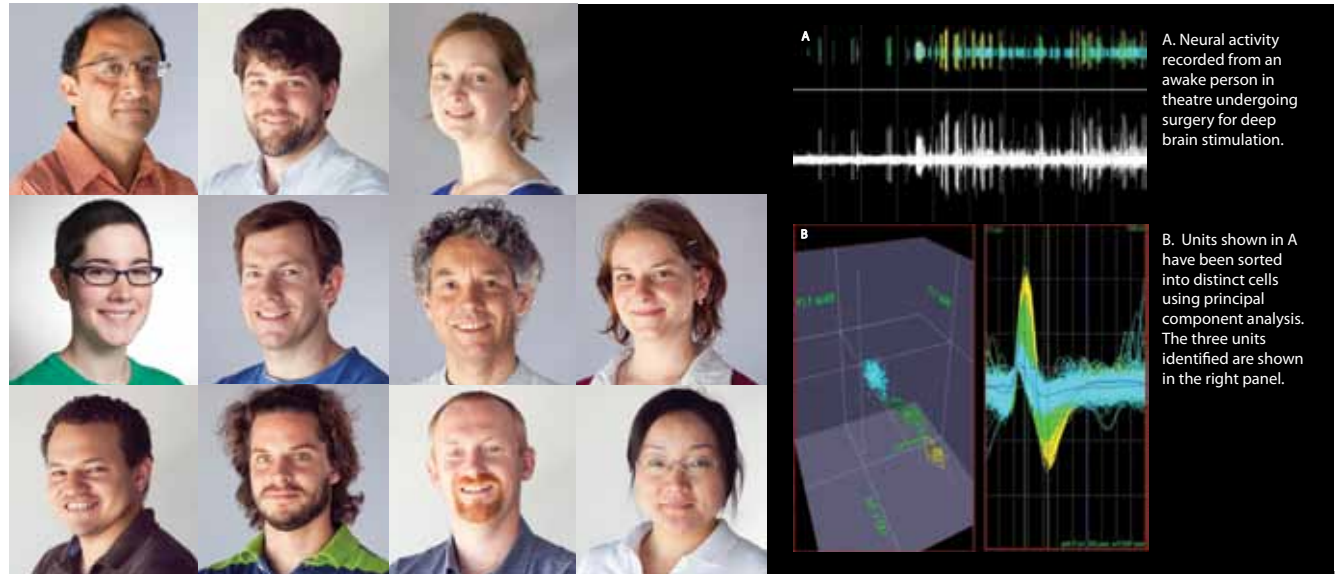


dorsal



Multi photon imaging shows partial backpropagation of action potentials in an intercalated neuron. Panel shows neuron with recording pipette attached that has been filled with a calcium indicator. The numbers measure distance from the soma in microns. An action potential evoked at the cell body raises calcium in the proximal dendrite but not at distances $> 60 \mu\text{m}$

Laboratory Head Professor Pankaj Sah



2011 Laboratory Members L-R: Pankaj Sah, Peter Curby, Christine Dixon, Helen Gooch, Roger Marek, John Morris, Cornelia Strobel, Robert Sullivan, Fabrice Turpin, Francois Windels, Li Xu. *Absent:* Sepideh Keshavarzi, Chris Nolan, Jay Spampinato, John Power, Petra Sedlak, Peter Stratton.

Background: Recording of neural activity from the human subthalamic nucleus. This area of the brain is stimulated to relieve the symptoms of Parkinsons disease.

Exploring learning and memory formation

The Sah laboratory aims to understand the circuits that mediate learning and memory formation in the mammalian brain using a variety of experimental techniques.

One of the major goals in neuroscience today is to understand the mechanisms that underlie learning and memory formation. This information is a prerequisite not only for understanding the biology of mind but also for the discovery of therapies that can alleviate disorders such as anxiety, depression and stress. The Sah laboratory studies the physiological and molecular mechanisms that underlie learning and memory formation. To achieve these goals they

focus on a part of the brain called the amygdala. The amygdala is an almond shaped structure in the mid temporal lobe that is responsible for assigning emotional salience to sensory stimuli. In particular it is involved in a simple learning paradigm – fear conditioning - that involves the rapid and long lasting acquisition of ‘emotional’ memories. Understanding the mechanisms that underlie fear-related learning is therefore most likely to yield a mechanistic understanding of the biology of learning and memory storage. The group uses a combination of electrophysiological recording, calcium imaging, molecular analysis, anatomical reconstruction and behavioural studies to understand the circuitry of the

amygdala and how activity in this circuitry leads to learning and memory formation.

One major advance in 2011 was the discovery that synaptic inputs to interneurons in the amygdala express different types of receptors, but only one of these has the potential to change with experience.

The team has also established collaboration with Professor Peter Silburn (UQ Centre for Clinical Research) to analyse recordings from brain regions in humans. These recordings are revealing how the human brain processes information and how it changes in disease.

Laboratory Head Professor Mandyam Srinivasan



2011 Laboratory Members L-R: Mandyam Srinivasan, Partha Bhagavatula, Brenda Campbell, Julia Groening, Michael Knight, Nikolai Liebsch, Laura McLeod, Ingo Schiffner, Dean Socol, Saul Thurrowgood, Tien Luu. **Absent:** Daniel Bland, Natalie Bland, William Warhurst. **Background:** Experimental honey bees live in natural hive environments. Image UQ Photography.

From flying insects and birds to aircraft autopilots

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.

Honeybees are highly efficient flying machines, requiring barely an ounce of honey to circumnavigate the world. Recently the Srinivasan team discovered that honeybees streamline their flight by raising their abdomens at higher speeds. They now find that this response is driven by the airflow on the body, as well as the motion of the

image of the environment, and are examining how the brain combines these signals to achieve optimal streamlining.

Aggressive honeybees are very adept at detecting and pursuing intruders. The group's experiments are revealing that moving targets are best detected when they are darker than the background – regardless of the colour of the target or the background. This research is helping them to understand how visual systems detect moving targets in natural environments.

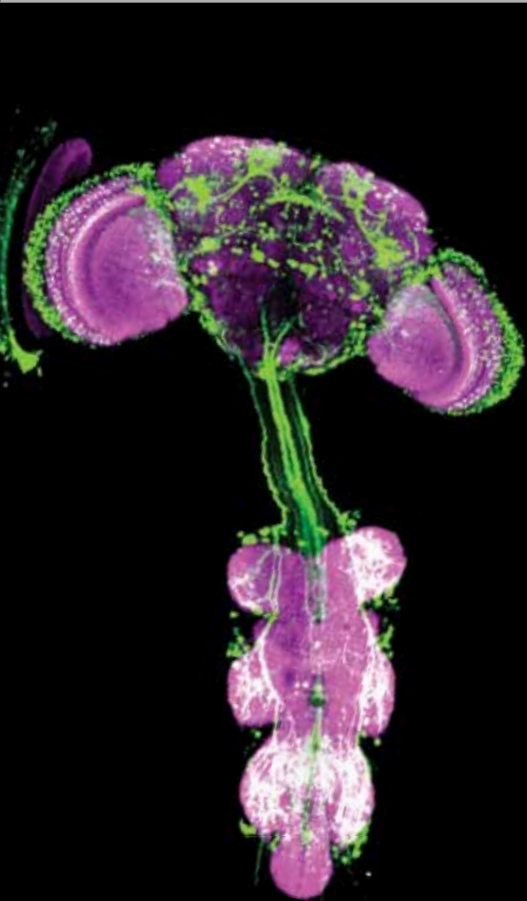
How do birds fly rapidly and safely through dense foliage? Members of the Srinivasan team have found that birds display characteristics

when they choose routes through a complex environment. Mathematical modelling reveals that this behaviour can expedite the passage of a flock of birds through a cluttered environment. This study is the first to report lateralisation in bird flight, and to postulate a function for it.

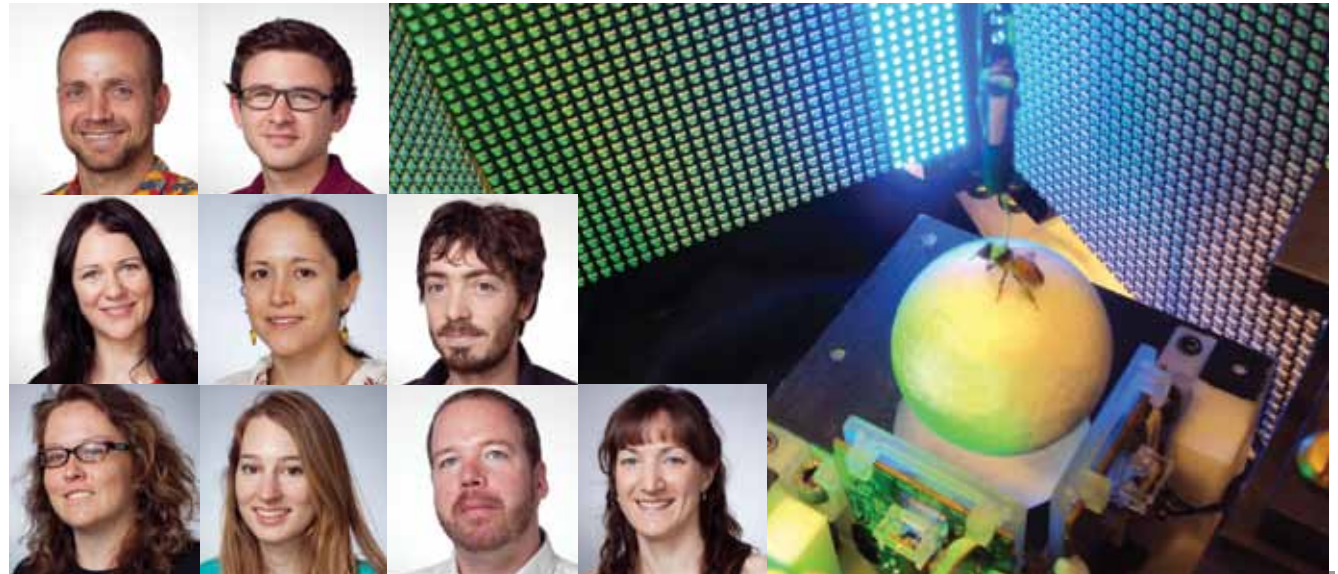
In the area of robotics, other members of the group have developed and successfully tested a vision system that enables fully automated aircraft landings, using principles gleaned from their earlier study of landing bees. This system is computationally simpler, lighter, and cheaper than conventional landing systems.



Researcher Saul Thurrowgood working in the robotics area of the Srinivasan laboratory.

Laboratory Head **Assoc. Professor Bruno van Swinderen**

Green fluorescent protein expresses in a select set of neurons in the central nervous system of a *Drosophila* fly mutant. By turning on and off the activity of this subset of neurons in mutants, we can begin to understand how individual neurons contribute to attention, sensory processing, and sleep. The magenta staining denotes synapses in the central nervous system.
Image: Angelique Paulk.



2011 Laboratory Members L-R: Bruno van Swinderen, Ben Calcagno, Wendy Imlach, Leonie Kirszenblat, Ben Kottler, Angelique Paulk, Jacqui Stacey, Bart van Alphen, Oressia Zalucki. **Absent:** Rebecca Morley, Melvyn Yap. **Background:** The honeybee controls the movement of the green bar by manipulating the Styrofoam ball. These experiments highlight the bees' ability to adapt to novel situations, their attention capabilities, and can be extended for use with electrophysiological tools.

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.

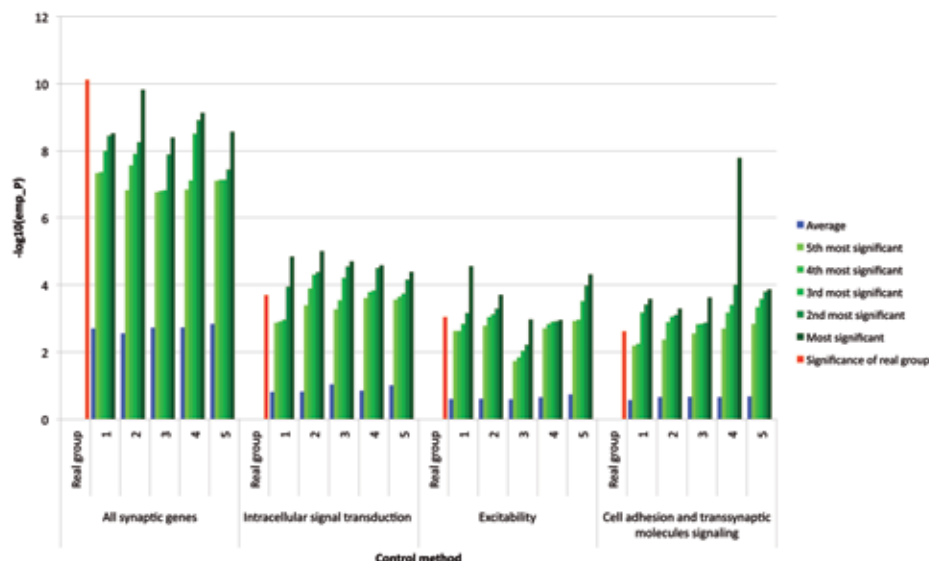
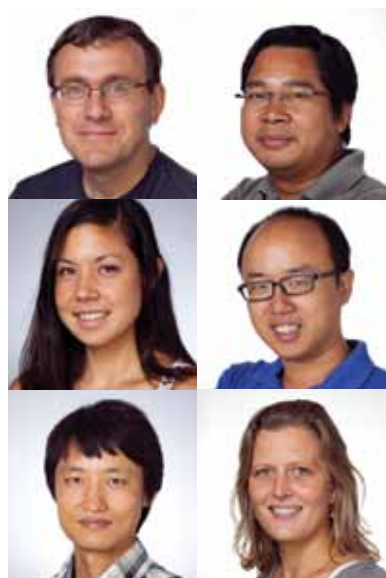
In order to efficiently screen *Drosophila* genetic variants for visual phenotypes, the laboratory has designed a unique system in which populations of flies display visual choices while navigating a maze, and these are then automatically scored for visual responsiveness levels. This efficient system, first used to fully characterise visual behaviour in a learning and memory mutant called *dunce*, will be crucial for screening mutants in the van Swinderen laboratory and in that of our collaborators at the Institute of Biophysics in Beijing.

Also in collaboration with colleagues in Beijing and the USA, the laboratory is focusing on a

population of neurons in the fly's central brain thought to be involved in sleep regulation as well as learning and memory. Convergence of behavioural effects stemming from activity of these central neurons is being studied by behavioural genetics and electrophysiology.

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. The honeybee offers valuable behavioural repertoires and a larger brain to complement similar questions addressed in the fruit fly.

Laboratory Head Professor Peter Visscher



2011 Laboratory Members L-R: Peter Visscher, Beben Benyamin, Marie-Jo Brion, Guo-Bo Chen, Hong Lee, Anna Vinkhuyzen. **Background:** Using data from genome-wide association studies, the Visscher laboratory showed the genes involved in synaptic plasticity are enriched for risk variants for schizophrenia.

Understanding genetic variation for complex traits in human populations

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, such as cognitive ability, between individuals. They use 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 2011, the group continued to demonstrate, using innovative statistical methods, that complex traits in human populations, including

common diseases and traits such as height, body-mass-index and cognitive ability, are caused by the cumulative effect of hundreds of genes.

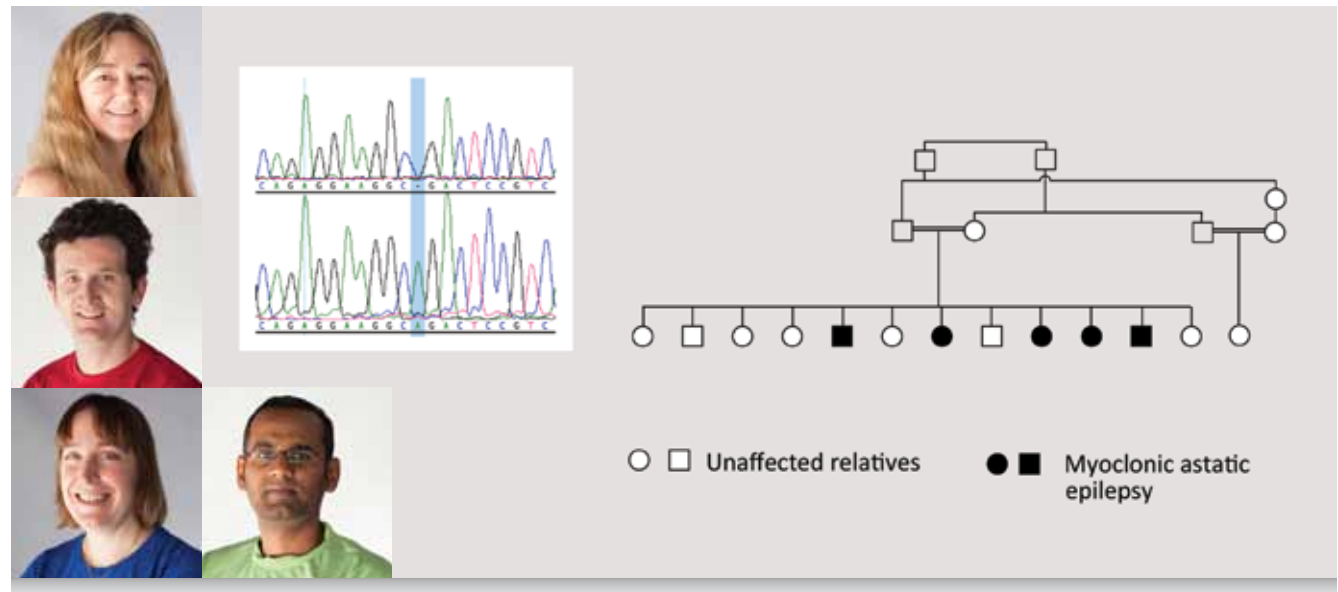
The group has also contributed analysis expertise to a large number of international research consortia that have found genes affecting endometriosis, schizophrenia, asthma, circulating lipid levels, rheumatoid arthritis and stature. The Visscher laboratory continues to lead the analysis efforts of a number of international research consortia.

They have developed widely used statistical

methods and software to estimate the effects of genes, chromosomes and the whole genome on disease susceptibility. Using data on 3,500 individuals from Scotland and England for whom they had measures of DNA variation on 500,000 genetic markers and outcomes of psychometric tests, they showed that adult intelligence is highly heritable and caused by the cumulative of hundreds if not thousands of genes. This work is the continuation of a long-standing collaboration with Professor Ian Deary from the University of Edinburgh. In collaboration with researchers from QIMR, Professor Visscher has also established the Brisbane Systems Genetics Study.



In a long-standing collaboration with Professor Ian Deary (University of Edinburgh), the Visscher laboratory showed that human cognition and cognitive ageing is partly genetic and due to many genes of small effects.

Laboratory Head **Dr Robyn Wallace**

2011 Laboratory Members, top - bottom: Robyn Wallace, Tim Butler, Marie Mangelsdorf, Ramesh Narayanan. **Background:** A single nucleotide deletion was discovered in a family with myoclonic astatic epilepsy.

Genetics of neurological disorders

The main focus of the Wallace laboratory is the genetics of neurological disorders such as motor neuron disease (MND) and epilepsy. MND is a rare, incurable disorder with late onset. Although most MND cases are not familial, a small percentage are due to genetic mutations. The group is using advanced genomic techniques to understand how these genes cause MND and to test potential treatments. Epilepsy is a common, complex disorder with a strong genetic component. The laboratory has successfully identified several human epilepsy genes and is continuing to characterise the functional consequences of the mutations.

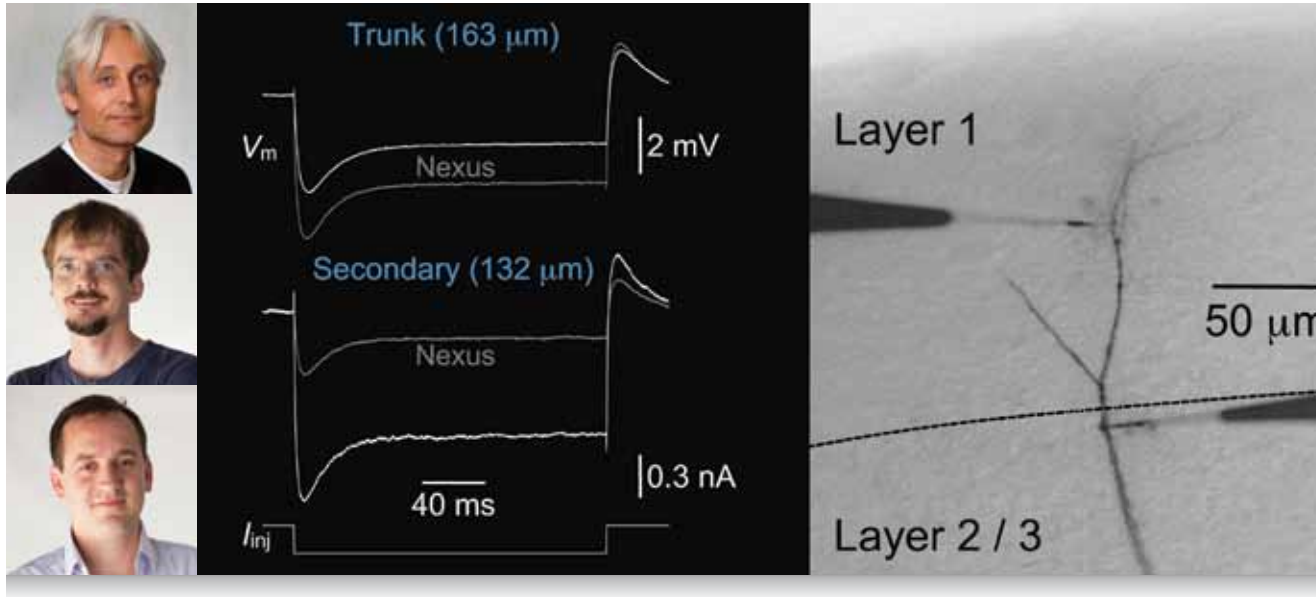
In 2011 the Wallace laboratory secured funding to study the role of a newly discovered MND gene, TDP-43. TDP-43 is a protein involved in gene regulation. However, the function of TDP-43 in the nervous system is currently unknown and its role in the pathogenesis of MND remains unclear. The team has identified gene targets of TDP-43 using a mouse model of the disease and are now examining these genes in MND patients. Out results suggest that TDP-43 is involved in maintaining the cell machinery that controls communication between motor neurons and their target muscle cells, a hypothesis that is now being investigated in collaboration with the

Hilliard laboratory using a nematode (*C. elegans*) model of MND. Potential outcomes of this project include crucial insights to understanding how motor neurons degenerate in MND and the identification of novel therapeutic targets.

The Wallace laboratory has also been studying families with inherited forms of epilepsy. Using high throughput "next generation" sequencing technology, they have recently identified two novel epilepsy-causing genes. The discovery of these genes has increased our understanding of the mechanisms that contribute to seizure activity in patients with myoclonic epilepsy.

Representation of genes regulated by the MND-causing TDP-43 gene. Image Ramesh Narayanan.

Laboratory Head Assoc. Professor Stephen Williams



2011 Laboratory Members, Top-Bottom: Stephen Williams, Arne Brombas, Ben Sivyer. **Background:** The figures show voltage recordings from the apical dendritic tree of neocortical pyramidal neurons.

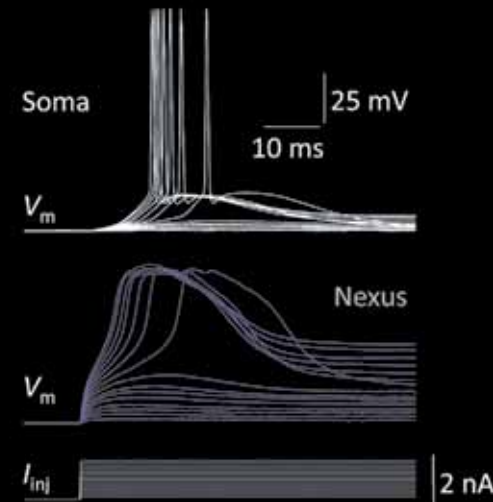
Computation in neocortical neurons and circuits

Researchers in the Williams laboratory are investigating how single neurons and circuits of neurons in the neocortex carry out computations that ultimately control behaviour. They use advanced electrophysiological and optical techniques to investigate how neurons integrate input signals, termed synaptic potentials, received throughout their tree-like morphology. Work has shown that synaptic inputs received at defined areas of the dendritic tree uniquely control the output of neurons.

The laboratory seeks to understand the rules and mechanisms that form and control this rich integrative process and explore the relevance of dendritic synaptic integration in the regulation of neuronal network function.

Through active collaboration with research groups at the Howard Hughes Medical Institute, Janelia Farm Research Campus, in the USA, the Williams laboratory is directly investigating how dendritic integration is engaged in the working brain using optical recording techniques.

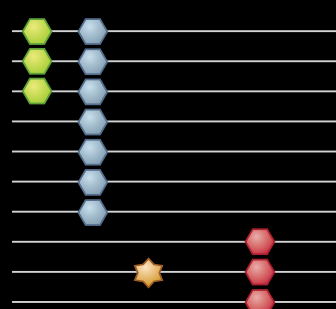
In the forthcoming year these techniques will be established at QBI, allowing the Williams team and other research groups to explore the operation of neuronal circuitry underlying defined behaviour. This research will enable neuroscientists to better understand how networks of neurons function in the neocortex and how these processes are disturbed in disease.



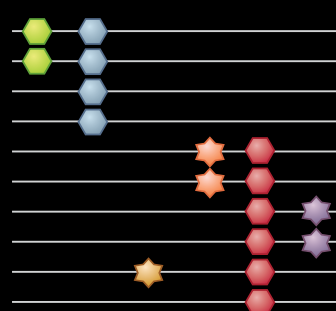
Synapses connect networks of neurons and facilitate information transfer between cells. A single neuron in the central nervous system may receive thousands of synaptic inputs distributed widely across its dendritic arbor. Neurons must integrate such time-varying input signals to form an output signal, or action potential, which is communicated to other neurons and/or effector systems like muscles.

Laboratory Head **Assoc. Professor Naomi Wray**

Controls

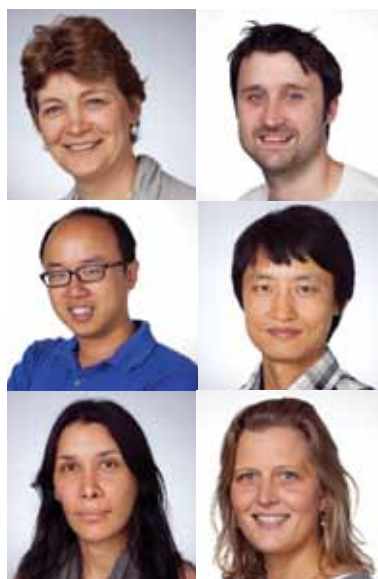


Cases



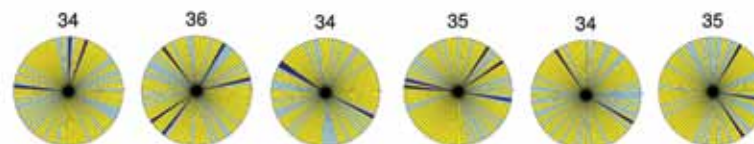
It has been suggested that synthetic associations can explain associations of common variants with disease. A synthetic association is illustrated in the cartoon of 10 control and 10 case chromosomes. The coloured shapes are DNA polymorphisms and the hexagonals represent common polymorphisms that have been genotyped. The common red locus is more frequent in cases than controls and so is associated with disease. However, the association is driven by three much rarer mutations (stars) that are all associated with the red locus. We argued that, while synthetic associations are possible, empirical data does not support a hypothesis that most common genetic associations reflect multiple causal variants.

Wray et al (*PLoS Biology*, 2011)

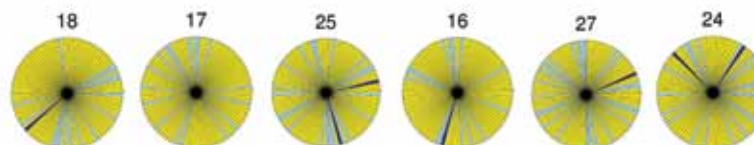


2011 Laboratory Members L-R: Above: Naomi Wray, Enda Byrne, Guo-Bo Chen, Hong Lee, Tania Carillo Roa, Anna Vinkhuyzen. *Absent:* Natalie Mills. **Background:** The Wray lab uses risk models to investigate genetic risk to disease. This cartoon illustrates the huge heterogeneity in genetic profiles between individuals under a genetic architecture of many variants of small effect. (From Visscher, Derks, Goddard and Wray, 2011 epub *Molecular Psychiatry*).

Affected individuals



Unaffected individuals



Each pie represents a genomic profile of an individual based on 100 risk loci (pie slices). Risk alleles have frequency of 10% in the population. Loci with 0, 1 or 2 risk loci are coloured yellow, blue or black, respectively. On average individuals carry 20 risk loci. Affected individuals carry > 33 risk loci.

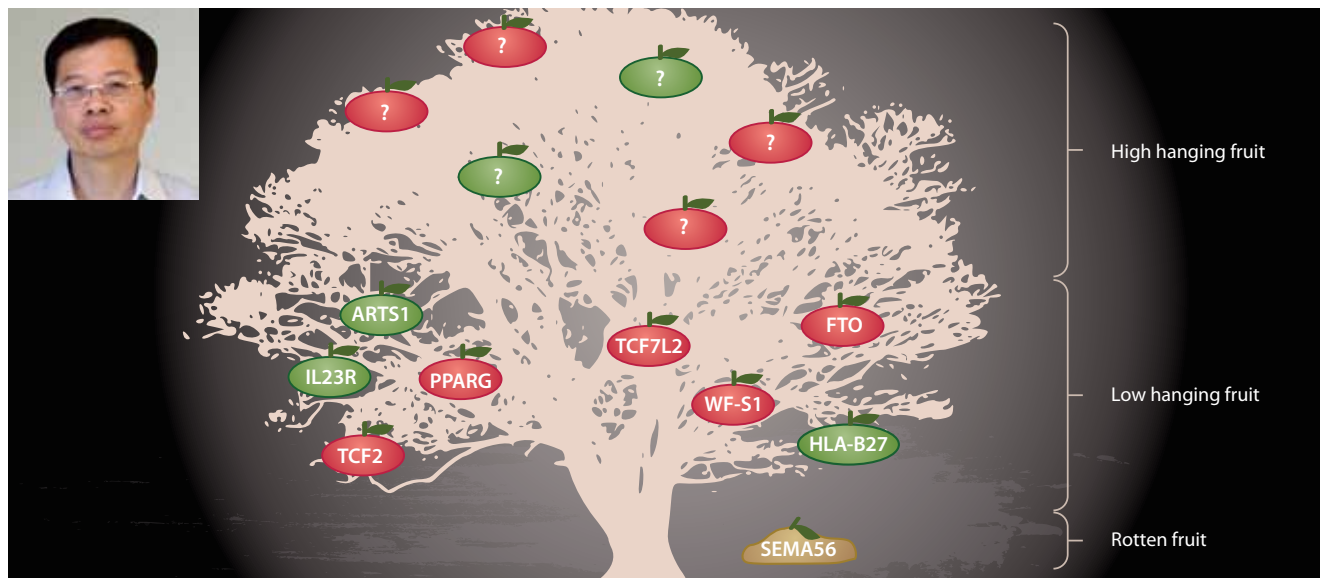
Understanding the genetic contribution to psychiatric disorders

The Wray laboratory joined QBI in September 2011. Their research focuses 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. In 2011 they published the largest genome-wide association study (GWAS) to date for major depression and they also play a leading role in the international Psychiatric GWAS Consortium (PGC) for Major Depression. Using GWAS data from the PGC for schizophrenia they have shown that at least 23 per cent

of the variance in liability to schizophrenia is attributed to common genetic variants and that this has important implications for the design of future studies. Psychiatric disorders are characterised by significant heterogeneity within diagnostic classes, as well as significant overlap of symptoms between disorders. For example, the anxiety disorders (panic, generalised anxiety, agoraphobia, and social and obsessive compulsive disorders) commonly co-occur, as do anxiety and major depression; more than 50 per cent of individuals with major depression will develop anxiety disorders in their lifetime. The Wray laboratory is developing novel strategies

to interrogate the shared genetic relationship between disorders, and have quantified the shared genetic relationship between schizophrenia and bipolar disorder. They are also focussing on strategies that define a more homogenous diagnostic class, for example postnatal depression or sleep dysregulation in collaboration with researchers at the Queensland Institute of Medical Research. Their theoretical work focuses on possibilities of prediction of genetic risk under a genetic architecture of many risk variants of small effect and this has led to invited plenary presentations about the prospects for genetic testing in psychiatry.

Joint Sino-Australian Neurogenetics Laboratory Professor Huji Xu



2011 Lab Head, top left: Huji Xu. **Background:** Identifying the casual variants of complex diseases remains an intricate challenge for researchers. Some loci with relatively high odds ratio, like the low hanging fruits of the trees, can be easily discovered. Some loci with moderate odds ratios, like the higher hanging fruits, can be investigated by well organized studies such as GWAS.

New laboratory probes neurogenetics of brain disease

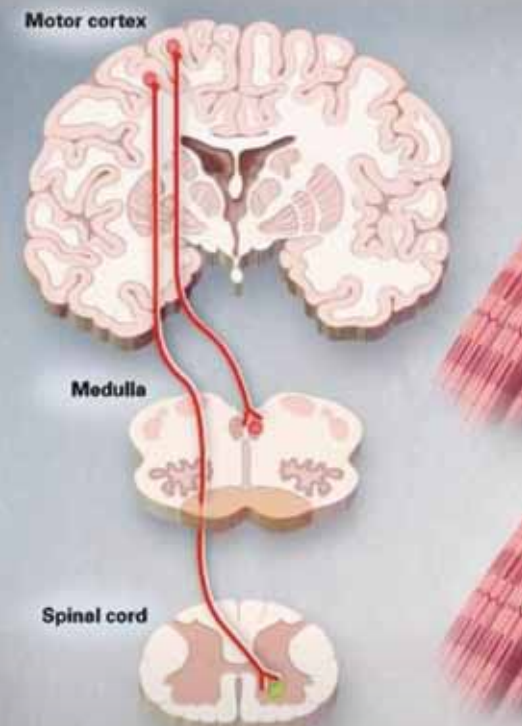
QBI and The University of Queensland Diamantina Institute (UQDI) further strengthened research ties with China with the 2011 opening in Shanghai of a joint laboratory dedicated to exploring how genes influence brain development and function.

A collaboration with the Second Military Medical University (SMMU), the Joint Sino-Australian Neurogenetics Laboratory was officially opened by then Federal Minister for Innovation, Industry, Science and Research, the Hon Kim Carr, on 2 August.

The goal of the Joint Sino-Australian Neurogenetics Laboratory, headed by Professor Xu, who holds an appointment with QBI and SMMU, is to uncover the genes that cause or make individuals susceptible to certain neurological and mental illnesses.

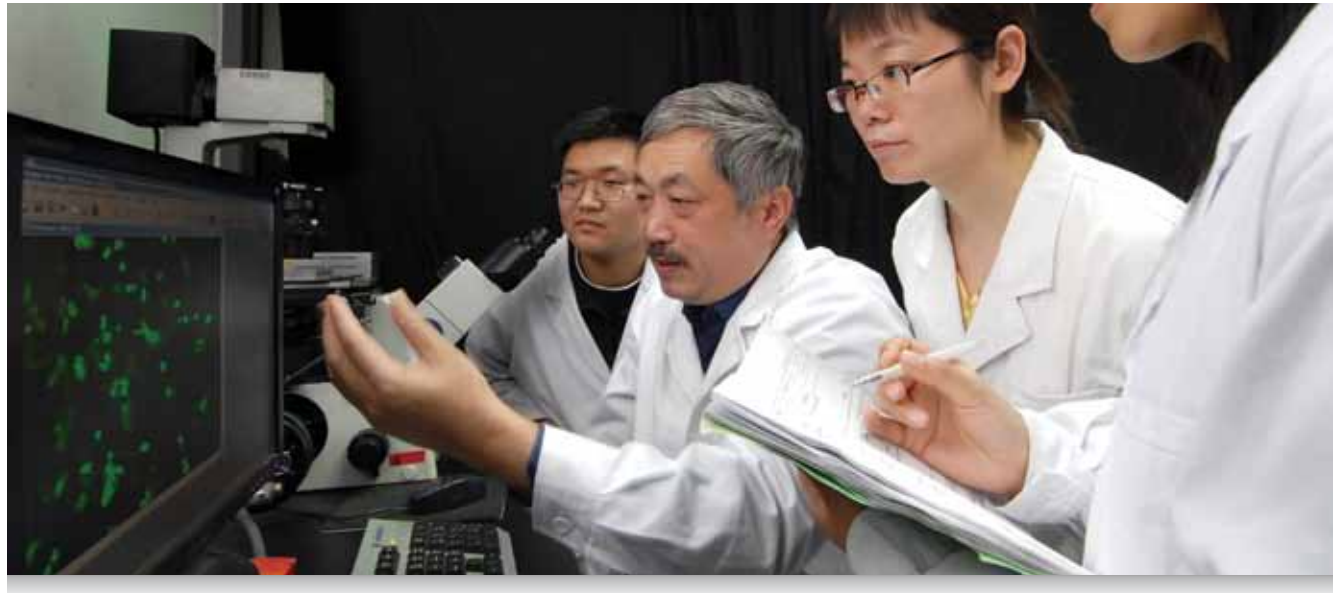
Researchers will initially focus upon the neurogenetics of motor neuron disease (MND), schizophrenia, stroke and epilepsy, but expect to extend their investigations to other disorders such as depression and dementia as the laboratory develops.

The research program will facilitate, for the first time within the Chinese population, genetic studies which have so far been conducted only in patient cohorts of European descent. Studying ethnically remote cohorts has the potential advantage that differences in ancestral genetic diversity may enable mapping of genes in Han Chinese that are not easily identified in other populations.



The motor neuron diseases (MND) are a group of neurological disorders that selectively affect motor neurons, the cells that control voluntary muscle activity including speaking, walking, breathing, swallowing and general movement of the body. Genetic factors are suspected to be important in determining an individual's susceptibility to disease. Several genes such as SOD1, ALS2, NEFH are known to be linked to ALS. The group is collaborating with Prof Brown's group in UQDI and Prof Bartlett's group in QBI as well as Chinese colleagues to identify more risk genes for this disease.

Joint Laboratory of Neuroscience and Cognition



Above: Professor Rongqiao He, from the IBP in Shanghai and Co-Director of the joint laboratory, with students.

Utilising brain plasticity to promote function and ameliorate disease

The Joint Laboratory of Neuroscience and Cognition between QBI and the Chinese Academy of Sciences Institute of Biophysics in Beijing, established in 2010, continues to gain momentum. QBI hosted a number of researchers from IBP in 2011 and several QBI researchers spent time at IBP working on collaborative projects.

There are currently six key projects underway in this initiative:

Screening molecules important in regulating neurogenesis - working towards better understanding of dementia - Perry Bartlett (QBI), Rongqiao He (IBP) and Ying Liu (IBP).

Working toward understanding visual attention and learning using *Drosophila* genetic tools - Li Liu (IBP) and Bruno van Swinderen (QBI).

Studying the way painful stimuli lead to emotional outcomes - resulting in a better understanding of how we respond to pain and

stress - Pankaj Sah (QBI) and Jianyuan Sun (IBP).

Identifying molecules that encourage repair of axon tracts after spinal cord injury - Helen Cooper (QBI) and Yaobo Liu (IBP).

Developing behavioural paradigms that measure selective attention - Raymond Chan (Institute of Psychology), Jason Mattingley (QBI), and David Shum (Griffith University).

Investigating bulk endocytosis at the molecular level which will lead to a greater understanding of how neurons "talk" to each other - Fred Meunier (QBI) and Jianyuan Sun (IBP).

A neuron in the medial amygdala in an acute brain slice. Image Pankaj Sah.

Science of Learning Centre

A major area of research excitement in coming years will involve exploring how the latest advances in neuroscience can inform educational practice.

Could school terms and tests be overhauled to take advantage of what we know about memory formation? What implications are there for teachers in our burgeoning understanding of mirror neurons? Can working memory be trained to boost problem-solving performance?

Researchers are a step closer to answering these and other questions through an ever-expanding range of projects within the Science of Learning Centre (SoLC) at QBI.

“Advances in technologies such as brain imaging have given neuroscientists a new understanding of brain function that has the potential to transform education in the same way biological understanding has transformed medicine,” says QBI Director Professor Perry Bartlett.

Such knowledge provides the basis for developing new approaches and tools which could revolutionise the delivery of learning, both for children in classrooms and adults in workplaces.

“These developments have the potential to put Australia at the very forefront of innovation and to realise the education revolution,” says Professor Bartlett.

“Science of Learning” is an international research initiative which aims to foster collaborations between educators, neuroscientists and psychologists.

“It’s about using the needs of classroom teachers to drive neuroscience research, and designing effective and practical learning techniques and tools based on neuroscientists’ growing understanding of what happens in the brain when learning takes place,” Professor Bartlett says.

Ms Belinda Craig joined the SoLC team in 2011 to conduct memory training experiments which replicate and extend findings from the Universities of Michigan and Bern, and found performance on IQ tests could be improved through an unrelated memory training game.

She is continuing to investigate whether these results extend to improvement in mathematical concepts, and hence can be applied in schools.

SoLC will also provide a structure through which practitioners can explore a range of issues, from attention in fruit flies, through to the strategies of students who find it easy to ‘pay attention’ in class.



Centre for Ageing Dementia Research

International expert heads new centre

QBI has recruited a leading international expert in the field of neurodegeneration to head its new Centre for Ageing Dementia Research.

Professor Jürgen Götz will be the inaugural director of the Centre, after several successful years undertaking dementia research at the Brain and Mind Research Institute in Sydney.

QBI Director Professor Perry Bartlett says the Centre is poised to tackle one of the country's most pressing health problems.

In the absence of significant medical breakthroughs, close to a million people will be dealing with dementia by 2050, according to a *Dementia Across Australia 2011-2050* report prepared for Alzheimer's Australia by Deloitte Access Economics.

Professor Götz has already made multiple discoveries in the area of neurodegenerative disorders and has contributed to lifting Australia's research profile internationally.

Research has shown that the brains of people with Alzheimer's disease are characterised by amyloid- β plaques and Tau-containing neurofibrillary tangles.

Professor Götz has played an instrumental role in these and many associated discoveries – for example, he established the first mouse model of Tau pathology and proved the amyloid cascade hypothesis.

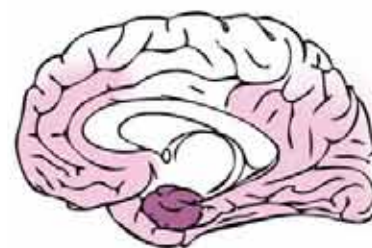
"My interest is in understanding the pathogenesis of dementias such as Alzheimer's disease and eventually finding a cure," Professor Götz explains.

"I firmly believe that in order to achieve this ambitious goal, it will be necessary not only to understand the pathological function of specific disease-related proteins but also to determine their physiological roles."

"Taking this a step further we not only want to understand why memory functions are impaired in dementia but also to discover the molecular correlates of memory and how memory 'works'."

Professor Götz said there was no better place to undertake this research than QBI, with its many outstanding experts in neuronal signalling and memory processing.

"I will be in an ideal environment to pursue these research directions, and the funding possibilities are there to recruit within QBI and externally to make a major contribution to ageing and dementia research," he says.



Students





Students

Students play a central role in the cutting-edge research undertaken at the Queensland Brain Institute. Attracted by QBI's outstanding reputation, local, domestic and international students from as far afield as China, Latin-America and Europe, are a familiar sight in the Institute's laboratories. Enrolling in either a PhD or QBI's Masters of Neuroscience program, these students bring a fresh, innovative and international approach to the institute. They represent, in a very real sense, the future of neuroscience research.

Students

Students form an integral part of life at QBI. Research higher degree students, in particular, help to maintain the smooth running of the Institute's laboratories and contribute ideas to the Institute's work. Accordingly, QBI aims to maintain a cohort of around 80 PhD and MPhil degree students each year.

In 2011, QBI had 70 research higher degree students enrolled at the Institute, of whom 29 were from a range of overseas countries, including China, Vietnam, Taiwan, Austria, Switzerland and Canada. Eleven students commenced their candidature and six PhDs were conferred – to Dana Bradford (Cooper lab), Timothy Lynagh (Lynch lab), Jonathan Hunt (Goodhill lab), Amber-Lee Donahoo (Richards lab), Nicola Watts (Sah lab) and Md Robiul Islam (Lynch lab). These graduates have subsequently gone on to postdoctoral work or into research administration.

The high quality of students at QBI is evidenced by their success in securing coveted scholarships and awards. Elizabeth Kita, who commenced work in the Goodhill laboratory in 2011, received the top international scholarship available at UQ, the International Postgraduate Research Scholarship (IPRS), in conjunction with the UQ Centennial Living Allowance and the UQ Advantage Top-up. Meanwhile, Oscar Jacoby and Leonie Kirszenblat were successful in securing the top domestic scholarship, the Australian Postgraduate Award (APA) and UQ Advantage Top Up. Two scholarships administered by the Queensland Centre for Mental Health Research were awarded to students Chikako Ragan and Xi Yao in the Mowry laboratory, to facilitate their research into schizophrenia and

related mental illnesses, while Georg Kerbler (Coulson lab) received an ANZ Trustee PhD Scholarship in Medical Research.

QBI also opens its doors to undergraduate students through the annual Summer Research program. In 2011/12, the Institute welcomed 25 Summer Researchers, its largest number ever, into the laboratories to undertake a range of projects ranging from computational rules in zebrafish development to the resequencing of pathogenic copy number variations in schizophrenia. For many students, time spent as a Summer Researcher paves the way to further study with QBI.



Above: Award winning scientist, Dana Bradford.

Students chart course to success

Students at QBI enjoy unparalleled opportunities to pursue their research interests, while working shoulder-to-shoulder with world-leading neuroscientists. This broad grounding provides a solid foundation for stellar career success.

Dr Dana Bradford

Dana Bradford joined QBI in 2004 as a casual research assistant during her final undergraduate year, before continuing on to undertake both Honours and a PhD in Associate Professor Cooper's laboratory. Dana's PhD thesis (2011) demonstrated a role for the multi-functional receptor, Neogenin, in neuronal differentiation in the adult mouse brain. Through successful collaboration with Professor Richard Faulk at the Centre for Brain Research at The University of Auckland, Dana was also able to establish that human forebrain tissue expresses Neogenin, suggesting that its function is the same in adult human brains. In addition to her publications, Dana received several awards during her PhD candidature, most notably a US Society for Neuroscience Travel Award, the Australian Neuroscience Society Istvan Törk Prize, the Sir Grafton Elliott Prize, and an Australian Society for Medical Research Oral Competition prize. Dana has recently begun a promising career working with the CSIRO.

Dr Tim Lynagh

The focus of Tim Lynagh's PhD (2011) was to determine the molecular basis by which the anthelmintic drug, ivermectin, activates inhibitory synaptic glycine receptors. Under the supervision of Professor Joe Lynch, Tim discovered the ivermectin binding site, a finding that was subsequently verified by the publication by another group of the crystal structure of a related receptor with which ivermectin docks. These results provide a molecular template for the design of new drugs that target this important receptor. During the course of this project, Tim also developed an improved 'neuronal silencing receptor' to inhibit electrical activity in defined populations of neurons in behaving animals, as well as discovering a means of identifying highly ivermectin-sensitive receptors that will be useful in characterising ivermectin resistance mechanisms in parasitic nematodes. To date, three first-author papers have been published from this work, two in the *Journal of Biological Chemistry* and another in the *International Journal of Parasitology*. Tim, who was the recipient of a Dean's Award for Research Higher Degree Excellence, is now working as a postdoctoral fellow in the laboratory of Dr Bodo Laube at the University of Darmstadt.

Students

Master of Neuroscience

Since its launch in 2010, UQ's flagship Master of Neuroscience program has gone from strength to strength, welcoming eight new students in the first semester of 2011 and a further six in the second semester. Six international students were among the year's intake.

An initiative of QBI's Professor Perry Bartlett and The University of Queensland's Vice-Chancellor, Professor Deborah Terry, the program is suited to both international and domestic students who wish to shift their career focus to neuroscience and pursue independent research and teaching careers. A per-semester quota of 12 is imposed to ensure a quality experience.

The program is coordinated by QBI and the Faculty of Social and Behavioural Science, but also spans many other exceptionally strong centres for neuroscience research at UQ. 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), though students with Honours or equivalent can complete the program in two semesters (16 units).

In 2011, three students who commenced the 24 unit program in 2010 graduated with Master of Neuroscience degrees. They were Di Xia, Shanzhi Yan and Zoran Boskovic, who subsequently accepted an offer to join the Coulson Laboratory as a Research Assistant. There were also five graduates who commenced the 16 unit program in 2011:

Kylie Cuthbertson, Jeff Thomson, Yin Wan, Aaron Warren and Megan Campbell. Without exception, students completing the Master of Neuroscience program say that the experience has whet their research appetites and encouraged them to pursue further study opportunities, such as PhDs.

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

- Molecular and Cellular Neuroscience (NEUR7006) which is concerned with cellular and molecular biology of the neuron.
- 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 focuses on the elucidation of the neural basis of cognitive and behavioural phenomena.

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, Human Movement Studies, Health & Rehabilitation Sciences, Microbial and Molecular Biosciences, Information Technology & Electrical Engineering, the Perinatal Research Centre, Centre for Clinical Research, the Institute for Molecular Bioscience, the Centre for Advanced Imaging (CAI) and the Queensland Institute of Medical Research.

Undergraduate Student, Clara Tang, was a summer student in Professor Panjak Sah's laboratory at QBI.

Community



Community

Queensland Brain Institute researchers form an integral part of the communities in which they work and live. They regularly discuss the latest research discoveries with community groups, while also engaging with their peers at scientific conferences.

In 2011, QBI hosted a series of high profile lectures and conducted a range of community outreach events. In addition to educating Australians about the latest research findings, staff also expanded their efforts to encourage the next generation to consider careers in neuroscience.

Staffing many of the QBI community events, receptionists Jill Wardropper (left) and Sue Earnshaw (right) are always ready with a smile.

QBI Events

Toshiya Yamada Memorial Lecture

Research and Remembrance

RNA binding proteins and neurodegeneration were among the topics under the spotlight at the Toshiya Yamada Memorial Lecture held at QBI in August.

Guest speakers Dr Jane Wu and Dr Yi Rao acknowledged their debt to the work of Dr Yamada, an outstanding mid-career scientist at UQ's Institute for Molecular Bioscience, who died suddenly in 2001.

Dr Wu, the Charles Louis Mix Professor of Neurology at Northwestern University Feinberg School of Medicine in Chicago, spoke on the subject of RNA binding proteins and neurodegeneration.

She noted that Dr Yamada's important discoveries relating to the molecules and pathways critical for the development and correct wiring of the nervous system "has inspired many people around the world".

Dr Rao, the Peking University Chair Professor and Dean of the Peking University of Life Sciences in Beijing, then spoke on the molecular biology of social interactions.

He took the packed lecture theatre on an entertaining journey through his research on the social and sexual lives of fruit flies and mice and the

crucial role of the 5-HT "molecule of love" in both insects and mammals.

QBI Director Professor Perry Bartlett took pleasure in welcoming members of the Yamada family to the lecture, including his widow, Linda Yamada, and her partner Bruce Moore.

Dr Yamada's children, Lisa, Kenji and Akira, also attended, as did his niece Ai-chan, who had travelled from Japan specially for the occasion.

Dr Yamada's groundbreaking work forms the basis of modern neurobiology and was instrumental in the resurgence of Australia as a world leader in this field.

Merson Lecture

The Emotional Brain

The QBI auditorium was filled to capacity when Professor Joseph LeDoux presented the Merson Lecture in October, taking audience members on a tour through the emotional centre of the brain, the amygdala.

According to LeDoux, the study of emotion has been hampered by a fixation on feelings.

"Rather than imposing concepts based on human introspective experience to the brains of other creatures, we should attempt to understand how human and animal brains are alike," said Professor LeDoux.

"Much of what is called 'emotion' in studies of other animals is accounted for by the operation of circuits involved in defence, energy and nutrition supplies, fluid balance, thermoregulation and procreation.

"These circuits are highly conserved in mammals, including humans."

The lecture is named in honour of Dr David Merson, who served for many years as chair of QBI's Development Board, and whose philanthropic sponsorship of this lecture indicates growing community interest in neuroscience.

Dr Merson, who was the founder of Mincom Ltd, from which he retired in 2002, has become director of a number of Australian software companies, research institutes and charitable bodies.



Left: Image from the Merson Lecture Poster. Image Dee McGrath. Right: Professor Perry Bartlett with speaker Professor Joseph LeDoux and sponsor Dr David Merson.

QBI Events

Peter Goodenough Memorial Lecture

Advances in ALS understanding

Professor Christopher Shaw, Professor of Neurology and Neurogenetics and Director of the MRC Centre for Neurodegeneration Research within the Institute of Psychiatry at King's College London, was the guest speaker at QBI's 2011 Peter Goodenough Memorial Lecture in December.

His address, delivered to a rapt audience, covered major advances in scientists' understanding of amyotrophic lateral sclerosis (ALS), which places ribonucleic acid (RNA) processing centre stage.

Professor Shaw has worked on the genetics and pathobiology of ALS for more than 15 years.

His group was the first to discover mutations in the TDP-43 gene in familial and sporadic motor neuron disease (MND) and demonstrated their toxicity *in vivo*.

Although mutations are rare, they provided a powerful biological tool to discover how TDP-43 is mislocalised and causes motor neuron degeneration.

Professor Shaw's group contributed to the discovery of another ALS gene called FUS, which has very similar function to TDP-43.

QBI's Director Professor Perry Bartlett said Professor Shaw delivered an engaging and stimulating talk which was interjected with humour while remaining sensitive to the many people who are afflicted with MND.

"It was especially wonderful to note the attendance of two of Peter Goodenough's four children, Christine and Alexandre, who travelled from the UK to attend the lecture," he adds.

"Christine was also accompanied by her two children, Toby and Marshall."

This annual lecture is named in honour of the late Mr Peter Goodenough (1936 – 2004), whose personal battle with MND led to an inspirational

bequest for fundamental scientific research.

These funds have primarily been used to establish, staff and support the Peter Goodenough and Wantoks Research Laboratory.

Completed in November 2007, this laboratory is home to the Molecular Genetics of Human Disease team led by QBI's Dr Robyn Wallace.



Right: Members of the Goodenough family and QBI staff listen to researcher Tim Butler explain the Wallace Lab's work.

QBI Conferences

The Australian Science of Learning meeting

Schools ahead

The program featured keynote addresses delivered by local and international experts who outlined the latest findings in the field.

The event marked the entry into Australia of what is known internationally as the 'Science of Learning', an initiative which aims to foster collaborations between educators and researchers.

"With the infrastructure in place to support collaborations between educators and neuroscientists, and increased investment in understanding the fundamental biology and physiology of the brain, we can look forward to an exciting time in learning discoveries," says Professor Bartlett.

Many of the speakers flew in from the US to attend, including Professor Soo-Siang Lim, Director of

the NSF Science of Learning Centres Program, who gave an overview of the US system. Other speakers included Professor Kenneth Koedinger from the Pittsburgh Science of Learning Center, and Professor Gary Cottrell from the Temporal Dynamics of Learning Center.

The US has already established a long-term Science of Learning program with a US\$500 million budget over 10 years.

QBI's Science of Learning Centre, launched in 2010, is one of the first steps towards building similar capabilities locally – but there remains an urgent need for Australia to increase its investment in this area.

"This in turn leads to the very attractive prospect that the next generation of Australians will have the learning capacity to lead the world in discovery and innovation," Professor Bartlett says.

Symposium on Attention and Learning

Educational engagement

The inaugural Symposium on Attention and Learning held at QBI on 21 July provided more than 120 registrants with opportunities to further explore concepts and issues raised during the previous day's Science of Learning meeting.

According to organiser Dr Robert Colvin, attention is fundamental to learning because, without it, we cannot filter sensory information or focus our concentration appropriately.

"Educational research emphasises engagement and motivation as key factors in student performance at school," Dr Colvin says.

The symposium brought together researchers from across the broad spectrum of attention research as it relates to learning.

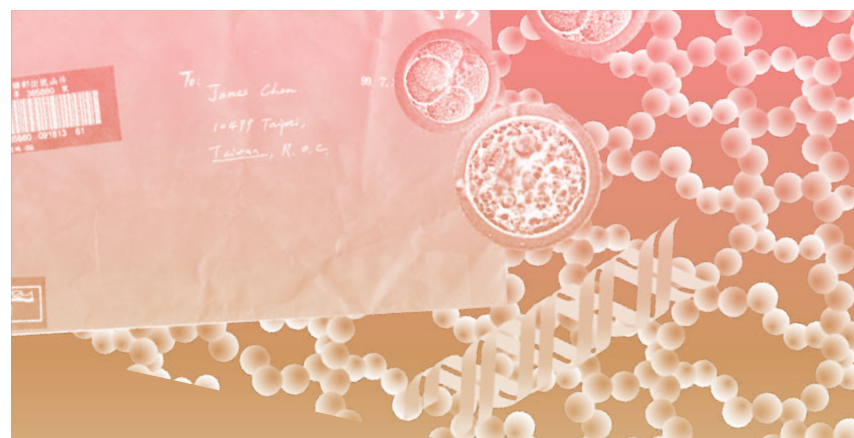
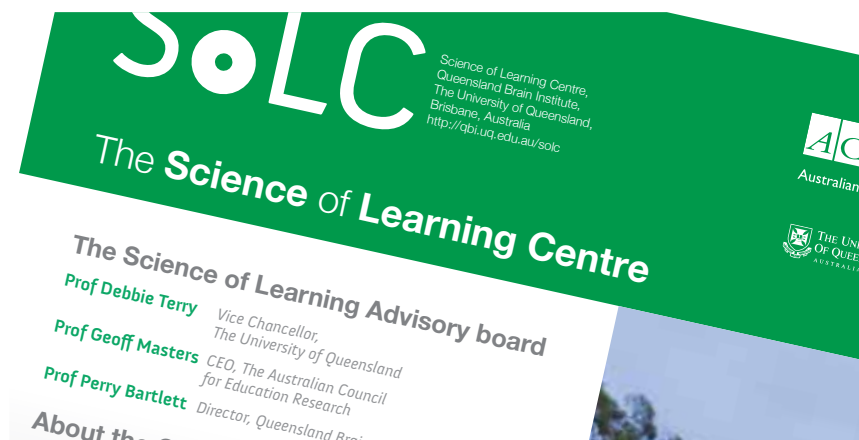
"They explored such things as classroom strategies

designed to engage 20 or more students at a time, behavioural research examining specific modes of attention and performance, and neuroscience research investigating neuronal interactions and the interactions of brain regions that characterise attentive and inattentive states," Dr Colvin explained.

"There was particular emphasis on how research across these different levels can be integrated to improve delivery of learning in today's classrooms."

Speakers included internationally renowned researchers from Australia and overseas, as well as experts in the quantitative analysis of educational outcomes.

QBI's Associate Professor Bruno van Swinderen took audience members through his investigations into the role of dopamine in attention using a fly model, while Associate Professor Ross Cunnington explained the mirror system and the perception of actions.



QBI-MCN Joint Symposium on Systems Neuroscience

Systems secrets

The secrets of stomatopod vision, the dynamics of binaural hearing in mammals and the mysteries of insect navigation were among the many fascinating topics canvassed at the first QBI-MCN Symposium on Systems Neuroscience.

Held at QBI in conjunction with the Munich Center for Neurosciences (MCN) of the Ludwig-Maximilians-University from 28-30 September, the symposium showcased the work of leading researchers in sensory, cognitive, cellular and molecular neuroscience.

More than 160 participants registered for a lecture program which spanned more than two days and featured local and international speakers, including Professor Mark Hübener from the Max-Planck-Institute of Neurobiology in Germany.

QBI Director Professor Perry Bartlett said MCN was the pre-eminent centre for neuroscience research in Europe and that the Institute was honoured to host the inaugural symposium.

"This event, and others which are expected to follow, provide us with exciting opportunities to forge strong interactions with other international leaders in this field," he says.

Joint Academies Public Forum

Expert insights

QBI staff attended a public seminar on 8 November hosted by the Queensland Chapter of the Australian Academy of Science, the Queensland Division of the Australian Academy of Technological Sciences and Engineering and the Queensland Chapter of the Australian Academy of the Humanities.

The keynote speaker was Professor Ian Frazer, Director of Research and Chief Executive Officer of the Translational Research Institute, Brisbane, who spoke on the subject of 'Cancer and how to avoid it'.

The Australian Academy of Science was represented by Professor Mark Blows, Professor of Evolutionary Biology and Head of School of Biological Sciences, UQ, who asked the question "Why does evolution fail?", and Professor Emma Whitelaw, Head of the Epigenetics Laboratory, Queensland Institute of Medical Research, who discussed "Epigenetics – the new genetics". Professor Suresh Bhatia, ARC Professorial Fellow within the School of Chemical Engineering, UQ, representing the Australian Academy of Technological Sciences and Engineering, spoke about engineering at the nanoscale, and Professor Gillian Whitlock, ARC Professorial Fellow within the School of English, Media Studies and Art History, UQ, representing the Australian Academy of the Humanities, gave an insight into the importance of archival material.

Opposite. Left: Detail from the SoLC flyer. Right: Detail from the advertising of the Joint Academies Public forum. Right: The program flyer for the QBI - MCN Joint Symposium Systems Neuroscience.
All images Dee McGrath.



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 and talks and hold discussions that are integral to the outreach program. These interactions – in libraries, bookstores, schools, hospitals and other community settings – have continually proven beneficial for the public and scientists alike.

As the community learns more about the research being conducted at QBI, the lectures provide an unparalleled opportunity for scientists to meet people who, in many cases, know someone affected by a neurological condition. Engaging with people who will potentially benefit from QBI's research in the longer term provides an additional impetus for neuroscientists to advance their work.

In 2011, QBI's researchers were involved in more than 30 outreach events, including:

Professor Perry Bartlett updated the judges of the Queensland Supreme Court on the latest research into ageing dementia.

Dr Dusan Matusica presented a talk at the University of the 3rd Age Winter School about his research into Alzheimer's disease.

Dr Brent Neumann addressed the Logan Village Library at their monthly Tips 'n Tea on his research using *C. elegans* as a model for understanding the human brain.

Dr Daniel Blackmore delivered a talk on the affect of exercise on the brain to patrons of the Green Apple Wellness Centre.

Dr Bill Mantzioris spoke to staff and affiliates at the Logan Adult Mental Health Service about mental health.

Associate Professor Elizabeth Coulson addressed the members of the Association of Independent Retirees, Gold Coast about her research into Alzheimer's disease.

Professor Joe Lynch gave a presentation to members of The Faculty of Pain Medicine (a faculty of the Australian and New Zealand College of Anaesthetists) on his research into ion channel receptor proteins.

Image right: Dr Robyn Wallace gives a tour of the QBI facilities.



Australia New Zealand Brain Bee Challenge



out-smart, out-think, out-last

Each year, the Australian Brain Bee Challenge (ABBC) creates a distinctive buzz in the laboratories and offices of QBI. Established in 2006, the Challenge aims to encourage high school students to learn about the brain and to educate students, teachers and the wider community about the importance of neuroscience research to society. Providing opportunities for students from all over Australia and New Zealand, including rural areas, to participate in the competition and to consider careers in neuroscience, is another major goal of the Challenge.

The ABBC has three rounds, with Round 1 taking place during Brain Awareness Week in March. This round is an online quiz in which students have to demonstrate their knowledge and understanding of brain structure, function and anatomy, as well as neurological disease and disorders. In 2011, 5,629 Year 10 students participated in the ABBC, taking the total number of participants over the five years since the competition was introduced in Australia to 18,547.

Round 2 of the ABBC, the State Final, is held in each state at a research institute or elsewhere on a university campus. The Queensland State Final was held on 19 July 2011 at QBI and gave 140 high-achieving Year 10 Queensland students, from as far away as Cairns, the opportunity to tour the facilities at QBI, participate in experiments, and attend lectures where various scientists discussed their discoveries and how they became involved in science research as a career.

Round 3 is the National Final, in which each State Champion competes to become the Australian Brain Bee Champion. The ABBC National Final is held annually at the Australian Neuroscience Society (ANS) meeting.

The Australian Brain Bee Champion for 2010, decided at the 2011 ANS meeting in Auckland, was Ben Thompson from the ACT. As part of his prize, Ben was able to attend the International Brain Bee Competition held in Florence in July 2011 during the International Brain Research Organisation's World Congress of Neuroscience. Ben competed against Brain Bee Champions from all over the world and came second by just half a point, which was an outstanding result for Australia!

With neurological and mental illness accounting for almost half of the total disease burden in Australia, the ABBC is a key vehicle to help students become interested in neuroscience research. Organisers continue to develop meaningful ways to engage students, teachers and the community – and look forward to the ABBC growing bigger and better in 2012.

Professor Perry Bartlett congratulating Teresa Tang from Brisbane State High school, the 2011 Queensland Brain Bee Champion.

Recognition



Recognition

The Queensland Brain Institute boasts almost 300 dedicated investigators working to elicit the fundamental mechanisms that regulate brain function. QBI researchers consistently shine in the neuroscience community, representing the Institute on a number of pivotal scientific organisations and serving on prestigious editorial boards.

QBI's track record in terms of publications, grants and awards further attest to the high standard of research being undertaken with the aim of discovering the fundamental mechanisms regulating brain function.

Dr Cheryll Filippich separating white cells from the blood of a patient with schizophrenia. These cells will be used to extract DNA for genetic studies.

Fellowships and Awards

Australian Research Council

Australian Laureate Fellowship

Professor Jason Mattingley was awarded a prestigious Australian Research Council Australian Laureate Fellowship, one of only 17 awarded from 139 applications. Professor Mattingley's Australian Laureate Fellowship will provide novel insights into how attentional processes are controlled in the human brain. The project will investigate how people use attention to filter sensory information and how the brain controls attention in health and disease. The findings will support new initiatives in a range of fields, from the development of more effective teaching practices to improved rehabilitation strategies for people with brain injuries.

The funding is for five years, and will support three postdoctoral scientists and three PhD students.

Future Fellowships

Four QBI researchers have recently taken up Australian Research Council Future Fellowships. Associate Professor Stephen Williams will use his fellowship to continue his investigations into the operation of nerve cell networks in the neocortex. Associate Professor Bruno van Swinderen's fellowship will support his work probing perceptual suppression mechanisms in the *Drosophila* brain. Dr Massimo Hillard's award will support his research into the regulation of axonal degeneration and regeneration in *C. elegans* while Dr Charles Claudianos will continue his investigation into the role of synapse development in cognitive disorders.

National Health and Medical Research Council

Principal Research Fellowship

Professor Linda Richards was successful in securing a sought-after NHMRC Principal Research Fellowship, recognising her longstanding work exploring embryonic and foetal brain development. Professor Richards is studying the development of the mammalian cerebral cortex, an area of the brain responsible for higher order cognitive processes in particular, how connections form between the two cerebral hemispheres, thereby enabling the brain to coordinate information from the two sides of the body. Malformations of these connections occur in more than 50 different human congenital syndromes, and can result in mental retardation as well as sensory and motor deficits.

Professor Richards is working with paediatric neurologists in Australia and the USA to develop diagnostic genetic and imaging tools to characterise human congenital syndromes associated with malformations of these connections.

Training Fellowship

Dr Martin Sale's research into the effect of prism therapy on attention in right-parietal lobedamaged stroke patients with spatial neglect earned him a 4 year NHMRC Training (Postdoctoral) Fellowship, commencing in 2011. He will use the fellowship to identify brain areas critical in restoring normal attention, and to investigate whether repeated therapy sessions improve treatment effectiveness.

Awards

UQ Foundation Research Excellence Award

Dr Timothy Bredy was one of only 11 researchers to be honoured with a UQ Foundation Research Excellence Award, for his work, probing the origins, maintenance and extinction of fear. The UQ Foundation Research Excellence Awards recognise outstanding performance and leadership potential among early to mid-career researchers. Dr Bredy's award will specifically explore how fear extinction memories, which serve to minimise fear, are established and maintained.

Research Australia Griffith University Discovery Award

Dr Oliver Baumann was awarded the Research Australia Griffith University Discovery Award in November, acknowledging his research into how humans use and process visual landmarks to navigate around unfamiliar terrain. This award, which was established to recognise achievements in the health and medical research community, highlights the importance of Dr Baumann's work as an early career researcher in human spatial navigation and cognitive neuroscience.



Commercialisation

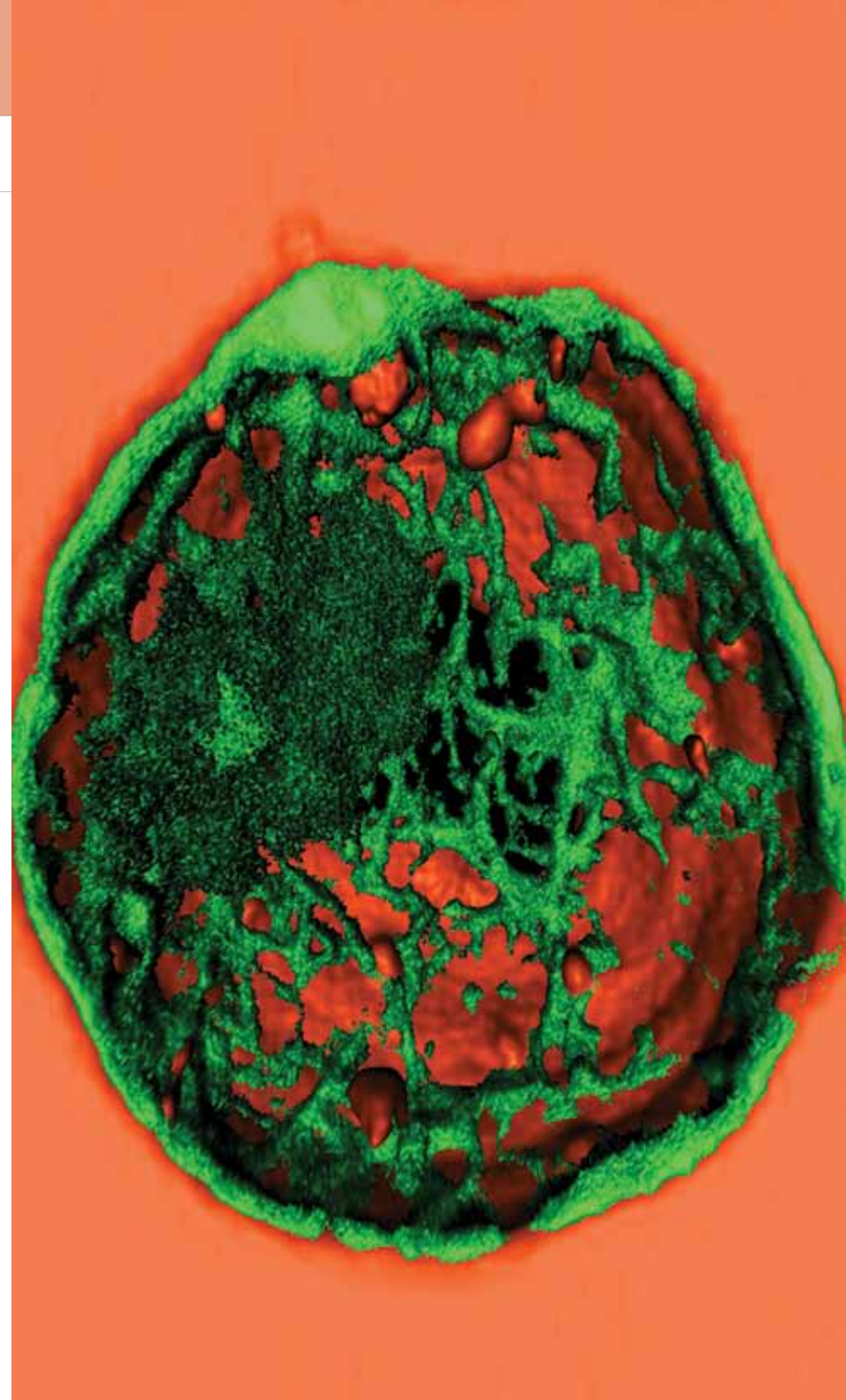
Commercialisation of QBI's discoveries continued to be actively pursued during 2011, including treatments to prevent neurodegenerative diseases such as dementia and motor neurone disease (MND), as well as treatments for spinal cord repair and botulism poisoning.

QBI researcher Associate Professor Frederic Meunier, in collaboration with researchers from the University of Newcastle and the Children's Medical Research Institute, has identified a novel treatment to prevent the uptake of pathogenic agents such as botulinum toxin. A provisional patent application directed to this research was filed in early 2011 and is being pursued by life science business development company Biolink.

NuNerve Pty Limited, a company focused on the development of novel technologies for the treatment and prevention of neurodegenerative diseases, has also continued its support of QBI's discovery pipeline this year. Projects being supported by NuNerve include screening for compounds which could potentially prevent over-excitation, and subsequent death, of neurons in MND sufferers (Professors Joe Lynch and Pankaj Sah), and development of a molecule which has been shown to block apoptotic neuron cell death in a dementia model and is currently being tested in an MND model (Associate Professor Elizabeth Coulson and Dr Robyn Wallace).

The development of a treatment for acute spinal cord injury is also ongoing. This project had been a collaboration with CSL Limited, to whom the technology was licenced, since 2007. Unfortunately, due to a change in strategic focus, CSL terminated the licence in late 2011. QBI is extremely grateful to CSL for its contribution to this promising technology, both financially and intellectually. The development of this treatment continues to be funded with the support of philanthropy.

QBI's commercialisation activities are supported by UniQuest Pty Limited, the main commercialisation company of The University of Queensland. UniQuest provides commercialisation expertise and resources through a Manager of Innovation and Commercial Development, Dr Bronwyn Battersby, who is based at QBI.



Right: Meunier Lab - Cellular staining of cytoskeletal proteins that are thought to be required for endocytosis.

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Book and Book Chapters

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Conference Proceedings

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Publications

Ng KB, Bradley AP, **Cunnington R**. (2011). Effect of competing stimuli on SSVEP-based BCI. In *33rd Annual International Conference of the IEEE EMBS*, 6307-6310. Boston, USA

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Protocols and Laboratory Manuals

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Editorials, letters, reviews and others.

Carter A, **Hall W** (2011) Proposals to trial deep brain stimulation to treat addiction are premature. *Addiction* 106: 235-237

Cronin T, **Marshall JN**, Wehling MF (2011) Talbot H. Waterman. *Philosophical Transactions of the Royal Society B* 366: 617-618

Frey C, **Marshall NJ**, Sherrell AJ (2011) Designing modular unmanned landers to better observe life in the deep ocean. *Sea Technology* 52: 25-28

Hall W, Carter A (2011) Science, safety and costs make deep brain stimulation for addiction a low priority: a reply to Vorspan *et al.* (2011) and Kuhn *et al.* (2011). *Addiction* 106: 1537-1538

Marshall J, Cronin T, Wehling MF (2011) New directions in the detection of polarized light. *Philosophical Transactions of the Royal Society B* 366: 615-616

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McGrath JJ, Hannan AJ, Gibson G (2011) Decanalization, brain development and risk of schizophrenia. *Translational Psychiatry* 1: e14

McGrath J (2011) Migrant status, vitamin D and risk of schizophrenia. *Psychological Medicine* 41: 892-895

McGrath, J. J. & Lawlor, D. A. (2011) The search for modifiable risk factors for schizophrenia. *American Journal of Psychiatry* 168: 1235-1238

Mulley JC, Heron SE, **Wallace RH**, Gecz J, Dibbens LM (2011) "Blinders, phenotype, and fashionable genetic analysis": setting the record straight for epilepsy! *Epilepsia* 52: 1757-1758

Suárez R (2011) Molecular switches in the development and fate specification of vomeronasal neurons. *Journal of Neuroscience* 31: 17761-17763

* some publications appeared first as electronic publications (epub) in 2010 ahead of their print version in 2011. These have been labeled accordingly.



Grants

For funding commencing in 2011, GST exclusive.

Australia-Indonesia Institute, Australian Department of Foreign Affairs and Trade

Dean A (CoralWatch) - Reef Education through Active Learning, \$11,000, 1 year

Australian Research Council

Fellowships

Australian Laureate Fellowship

Mattingley J - Cognitive control of attention and its role in regulating brain function in health and disease, \$2,649,836, 5 years

Future Fellowships

Claudianos C - The role of synapse development in cognitive disorder, \$708,377, 4 years

Hilliard M - Molecules and mechanisms regulating axonal degeneration and regeneration in *C. elegans* neurons, \$714,528, 4 years

van Swinderen B - Perceptual suppression mechanisms in the *Drosophila* brain, \$813,192, 4 years

Wray N - Dissecting the shared genetic architecture of psychiatric and psychological traits with application to prediction of genetic risk, \$686,400, 4 years [awarded to Wray while at the Queensland Institute of Medical Research but transferred to QBI in 2011]

Williams S - Operation of nerve cell networks in the neocortex, \$813,192, 4 years

Projects

Discovery Projects

Bartlett P, Boyd A - Development of novel reagents that specifically counteract EphA4 to enhance axonal regeneration, \$420,000, 3 years

Cunnington R - The mirror system and the

perception of actions, \$244,753, 3 years

Duncan J, Lawrence A, Bredy T, Gavrilescu M - The long-term consequences of toluene exposure on the maturing brain, \$360,000, 3 years [awarded to and administered by the University of Melbourne]

Dux P, Mattingley J - Bottlenecks in the brain: a causal role for the frontal-parietal network in multitasking limitations, \$382,000, 3 years [awarded to and administered by UQ School of Psychology]

Goodhill G - Mechanisms of nerve fibre guidance by molecular gradients, \$300,000, 3 years

Marshall J, Cheney K, Temple S, Cribb T - The functions of reef fish colour patterns: how did the coral trout get its spots? \$230,000, 3 years

Rubinsztein-Dunlop H, Nieminen T, Meunier F - Dynamics of constrained Brownian motion of neuro-secretory vesicles, \$510,000, 3 years [awarded to and administered by UQ Centre for Biophotonics and Laser Science]

Srinivasan, M - Visual guidance of flight in birds, \$500,000, 3 years

Goddard ME, Visscher P* - Why is most of the genetic variance for complex traits undetected by large powerful screens of common variants? \$360,000, 3 years [awarded to and administered by the University of Melbourne] (*Visscher appointed UQ Diamantina Institute 60%; QBI 40%)

Large Infrastructure, Equipment and Facilities

Bredy T, Claudianos C, Richards L, Grimmond S, Lawrence A, Mowry B - Mass spectrometry platform for high-throughput genotyping, epigenetic analysis and validation of genome-wide sequencing studies, \$240,000, 1 year.

Linkage Projects

Brown M, Xu H, Bartlett P, Wallace R, Visscher P, Mowry B, Reutens D - Sino-Australian

Neurogenetics Initiative, \$690,000, 3 years

Coulson E, Wallace R, Gearing D - Regulation of neuronal cell death signalling for the treatment of neurodegenerative diseases, \$430,000, 3 years

Townsend K, Marshall J, Wilcox C, Hardest B - Identifying the risks and assessing the impacts of marine debris on sea turtles in Australian waters, \$465,000, 3 years [awarded to and administered by UQ School of Biological Sciences]

Special Research Initiative in Stem Cells Science

Pera T, Kilpatrick T, Gardner D, Hilton D, Rosenthal N, Elefanty A, Stanley E, Gray P, Little M, Grimmond S, Wolvetang E, Nielsen L, Bartlett P, Wells C, Harvey R, Graham R, Alexander W - Stem Cells Australia, \$21,000,000, 7 years [awarded to and administered by the University of Melbourne]

Cancer Council Queensland

Richards L, Boyd A, Piper M - Suppression of high-grade glioma by Nf1b over-expression, \$196,452, 2 years

Mason Foundation

Coulson E, Rose S, Hort J, Byrne G - Measures of cholinergic basal forebrain degeneration as an early screen for Alzheimer's disease, \$50,000, 1 year

Motor Neurone Disease Research Institute of Australia

Wallace R, Blair I, Mangelsdorf M, Nicholson G - Identifying genes that are regulated by TDP-43 and FUS using high-throughput sequencing, \$96,540, 1 year

National Health and Medical Research Council

Fellowships

Research Fellowships

Richards L - NHMRC Principal Research Fellowship: Development of the

cerebral cortex, \$690,370, 5 years

Wray N - NHMRC Senior Research Fellowship level A: Dissecting the shared genetic architecture of psychiatric and psychological traits with application to prediction of genetic risk, 5 years [awarded to Wray while at the Queensland Institute of Medical Research but transferred to QBI in 2011; held in Honorary capacity while on ARC Future Fellowship]

Training (Postdoctoral) Fellowship

Sale M - Investigation of the effect of prism therapy on attention dysfunction in right-parietal lobe damaged stroke patients with spatial neglect, \$290,032, 4 years

Projects

Development Grant

Bartlett P, Boyd A - Therapeutic development of novel EphA4 antagonists for spinal cord injuries, \$663,390, 3 years

Project Grants

Bellgrove M, Hester R, Chambers C, Garavan H, Hawi Z - Genetic and physiological mechanisms of executive control, \$541,049, 3 years

Burne T, McGrath J, Eyles D - Attentional processing in developmentally vitamin D deficient rats: Modelling the cognitive symptoms of schizophrenia, \$244,182, 3 years

Claudianos C, van Swinderen B, Reinhard J - Neurexin and neuroligin: a code for synaptic development, \$337,524, 3 years

Cooper H, Neogenin: a regulator of neuronal differentiation and migration in the adult brain, \$322,524, 3 years

Coulson E, Nykjaer A - How do p75 and sortilin facilitate TrkA-mediated survival signalling? \$540,048, 3 years

Grants

Gunnarsen J, **Power J** - Sez6 and neuronal calcium signalling in synapse development [awarded to and administered by the University of Melbourne], \$596,072, 3 years

Hawi Z, Bellgrove M, Wallace R, Vance A - Functional characterisation of genetic risk variants for ADHD: from association to biology, \$507,255, 3 years

McGrath J, Eyles D, Burne T, Pedersen C, St Clair D, Mortensen P - Neonatal vitamin D status and risk of schizophrenia: a replication in two independent samples, \$334,615, 3 years

Meunier F, Arumugam T - TNF traffic and secretion in astrocytes and microglial cells: unveiling new targets for ischemic stroke, \$565,048, 3 years

Piper M - Investigation of the role of Nfix in adult neurogenesis, \$337,524, 3 years

Sah P, Windels F - Neural correlates of fear conditioning and extinction, \$855,650, 5 years

Williams S - Cellular basis of cortico-cortical integration, \$337,524, 3 years

Wray N, Montgomery G, Martin N, Middeldorp R, Freimer N - Towards an etiological understanding of the comorbidity of psychiatric disorders, \$845,939, 3 years [awarded to Queensland Institute of Medical Research but transferred to QBI in 2011]

Wray N, Visscher P - Better methods for individual risk prediction of complex traits in human populations, \$578,416, 3 years [awarded to Queensland Institute of Medical Research but transferred to QBI in 2011]

National Institutes of Health, USA

National Institute of Neurological Disorders and Stroke

Shaw P, van Swinderen B - Functional analysis of sleep promoting neurons in health and disease USD\$1,574,042, 5 years [awarded to and

administered by Washington University, USA; subaward of USD\$616,023 to van Swinderen]

Queensland State Government

Queensland-Chinese Academy Sciences Biotechnology Projects Fund

Bartlett P, Cooper H, Sah P, He R, Li B, Liu Y - The effect of hypogeomagnetic field on brain function and development, \$250,000, 3 years

Queensland-Chinese Academy Sciences Early Career Fellowships Program

Nguyen T - Uncovering the mechanisms underpinning the sustainability of neurotransmission at a central synapse, \$17,000, 1 year.

Indo-Queensland Biotechnology Project Fund

Bartlett P, Jhaveri D, Vaidya V - Stimulation of adult neural stem cells by norepinephrine: a promising target for the treatment of depression, \$250,000, 5 years

UniQuest

Coulson E - UniQuest Pathfinder, \$50,000, 1 year

Richards L, Boyd A - UniQuest Pathfinder, \$50,000, 1 year

Researcher Irina Kharatishvili working in the laboratory.



Neuroscience Seminars

QBI conducts a weekly seminar program giving 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 Victor Anggono

Howard Hughes Medical Institute, The Johns Hopkins University School of Medicine, Baltimore
Activity-dependent regulation of AMPA receptor trafficking

Professor Dana Ballard

University of Texas, Austin
Modular reinforcement learning as a model of embodied cognition

Dr Daniel Blackmore

Queensland Brain Institute, The University of Queensland
Physical exercise activates endogenous neural stem cells in the aged brain

Dr Tom Burne

Queensland Brain Institute, The University of Queensland
Modelling cognitive symptoms in developmental vitamin D-deficient rats

Professor Brian Butterworth

Institute of Cognitive Neuroscience and Department of Psychology, University College, London
The science of failing to learn arithmetic

Professor Xiangjun Chen

Department of Neurology, Fudan University, Huashan Hospital, Shanghai
Axonal transport and motor neuron disease: what we can learn from Swl mouse study?

Dr Louise Cheng

Department of Developmental Neurobiology, National Institute for Medical Research, London
Food for thought: how the growth of Drosophila CNS is spared under nutrient restriction

Dr Allen Cheung

Queensland Brain Institute, and School of Information Technology and Electrical Engineering, The University of Queensland
Neurocomputational principles of animal navigation

Dr Charles Claudianos

Queensland Brain Institute, The University of Queensland
The neuroligin and neuroligin complex: codes, circuits and cognitive disorder

Professor Justin Cooper-White

Australian Institute for Bioengineering and Nanotechnology and the School of Chemical Engineering, The University of Queensland
Tailored surfaces, scaffolds and diagnostic cell-based microfluidic platforms for small molecule screening, stem cell expansion and controlled tissue genesis

Professor Michael Corballis

Department of Psychology, The University of Auckland, Auckland
Language, time, and the lopsided brain

Associate Professor Jeremy Crook

Department of Surgery, St Vincent's Hospital, and The University of Melbourne
Physical exercise activates endogenous neural stem cells in the aged brain

Professor Brian Dean

Rebecca L. Cooper Research Laboratories, Victorian Brain Bank Network, and The Mental Health Research Institute
The role of muscarinic receptors in the pathophysiology and treatment of schizophrenia

Professor Carlos Dotti

Center for Human Genetics, Katholieke Universiteit Leuven, Belgium
Establishment of neuronal polarity in vivo

Dr Kevin Dudley

Queensland Brain Institute, The University of Queensland
Epigenetic mechanisms associated with vulnerability and resilience to psychiatric disease

Dr Ben Emery

Centre for Neuroscience and Howard Florey Institute, The University of Melbourne
Transcriptional control of CNS myelination

Ms Claire Foldi

Queensland Brain Institute, The University of Queensland
Mouse models of advanced paternal age: relevance to neuropsychiatric disorders

Professor Wayne Hall

UQ Centre for Clinical Research and the Queensland Brain Institute, The University of Queensland
The ethical and social implications of proposals to allow normal adults to use neuropharmaceutical drugs to enhance cognitive functioning or replace recreational drugs

Ms Callista Harper

Queensland Brain Institute, The University of Queensland
Mechanisms segregating local from retrograde trafficking in presynaptic terminals

Professor Cheng He

Department of Neurobiology, Second Military Medical University, Shanghai
Biology of olfactory ensheathing cells in CNS regeneration

Dr Massimo Hillard

Queensland Brain Institute, The University of Queensland
Neurite development and regeneration in C. elegans neurons

Mr Robiul Islam

Queensland Brain Institute, The University of Queensland
Discovering novel drugs targeting glycine receptor Cl⁻ channels in pain sensory pathways

Professor Tianzi Jiang

Institute of Automation, Chinese Academy of Sciences, Beijing
Vascular development in the primate retina: the role of guidance and anti-angiogenic factors and How do brain networks correlate with intelligence?

Professor Roland Jones

University of Bath, Bath
The role of presynaptic NMDA and kainate receptors in synaptic plasticity, synchrony and epileptic activity in the entorhinal cortex

Dr Shanker Karunanithi

School of Biomedical Sciences, The University of Queensland
Neuronal homeostasis in Drosophila

Ms Sepideh Keshavarzi

Queensland Brain Institute, The University of Queensland
The medial nucleus of the amygdala: neuronal classification and synaptic transmission

Assoc. Professor Rupert Lanzenberger

Department of Psychiatry and Psychotherapy, Medical University of Vienna, Vienna
Molecular and functional neuroimaging in psychiatric neuroscience using PET and fMRI

Professor Andrew Lawrence

Howard Florey Institute and Centre for Neuroscience, The University of Melbourne
Reinforcement vs extinction: the yin & yang of mGlu5 signalling

Dr Yonghui Li

Institute of Computing Technology, Chinese Academy of Sciences, Beijing
Brainnetome study based on diffusion MRI

Ms Nancy Malintan

Queensland Brain Institute, The University of Queensland
Role of Munc18-1 in regulating neuroexocytosis: unravelling the trafficking pathway underpinning Syntaxin-1 delivery to the plasma membrane

Dr Sally Martin

Queensland Brain Institute, The University of Queensland
Regulation of autophagic trafficking in neurons: clues to a novel neurodegenerative pathway

Neuroscience Seminars

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Queensland Brain Institute,
The University of Queensland
*The role of Nuclear Factor One (NFI)
in cortical development*

Professor Colin Masters

Mental Health Research Institute,
The University of Melbourne
*The natural history of Alzheimer's disease:
strategies for diagnosis and therapy*

Professor Jason Mattingley

Queensland Brain Institute and School of
Psychology, The University of Queensland
*Parietal control of attention, saccadic remapping
and audiovisual integration: insights from
human brain stimulation studies*

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The University of Queensland
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death signalling is mediated by GIRK channels*

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Institute of Psychiatry and Maudsley
NHS Foundation Trust, London
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School of Biomedical Sciences,
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*Highlights in the making and breaking
of the neuromotor system*

Professor Patricio O'Donnell

Departments of Anatomy & Neurobiology
and Psychiatry, University of Maryland

School of Medicine, Baltimore
*Altered peri-adolescent maturation of prefrontal
cortical circuits in animal models of schizophrenia*

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Centre for Advanced Imaging,
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Professor Nadia Rosenthal

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Institute, Monash University
Immune modulation of mammalian regeneration

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Institute of Neurology, University
College London, London
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and patients with Parkinson's disease studied
with transcranial magnetic stimulation*

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School of Mathematics and Physics and
Centre for Biophotonics and Laser Science,
The University of Queensland
Catch, move and twist using optical tweezers

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The University of Queensland
Fear, synaptic plasticity and the amygdala

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Queensland Brain Institute,
The University of Queensland
*Computational modelling of
retinotectal map development*

Dr Samuel Solomon

School of Medical Sciences Bosch
Institute, The University of Sydney
Some building blocks for motion vision

Ms Cornelia Strobel

Queensland Brain Institute
The University of Queensland
The medial intercalated cells of the mouse amygdala

Professor Yi Sun

Department of Psychiatry and Behavioral
Sciences and the Department of Molecular and
Medical Pharmacology, University of California,
Los Angeles Medical School, Los Angeles
Epigenetic regulation of stem cell differentiation

Professor Misha Tsodyks

Department of Neurobiology,
Weizmann Institute of Science, Rehovot
*Neuronal population coding of
parametric working memory*

Ms Qian Wang

Queensland Brain Institute,
The University of Queensland
*Investigating GABA-A and glycine receptor structure
and function using voltage clamp fluorometry*

Professor Sam Weiss

Hotchkiss Brain Institute and Department of Cell
Biology and Anatomy, University of Calgary, Calgary
*Adult neural stem cells: from basic
science to therapeutic applications*

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Department of Biological Sciences, National
University of Singapore, Singapore
Lipidomics, new tools and applications

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*Corollary discharge abnormalities in patients
with schizophrenia: evidence from electroen-
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*How birds use the magnetic field
of the earth for navigation*

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Genetic Epidemiology, Molecular Epidemiology
and Queensland Statistical Genetics Laboratories,
Queensland Institute of Medical Research
*What have we learnt about psychiatric disorders
from genome-wide association studies?*

Dr Xin Yu

Laboratory of Functional and Molecular
Imaging, National Institute of Neurological
Disorders and Stroke, Bethesda
*Characterizing the sensory deprivation-induced
plasticity along the whisker-barrel system
of adolescent rats: from systemic MRI brain
mapping to single cell electrophysiology*

Associate Professor Yimin Zou

Division of Biological Sciences, University
of California, San Diego
*Wnt/Planar cell polarity signalling in axon
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Professional Service

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- Garvan Institute of Medical Research, University of New South Wales, Scientific Appointments and Promotions Committee Member
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- Australian Huntington's Disease Association, Queensland Branch, QBI representative
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John McGrath

- Schizophrenia International Research Society, Board Member
- Orygen Youth Health Research Centre, Research Committee Member
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- Australian Schizophrenia Research Bank, Access Committee Member
- Ernst Strüngmann Forum on Schizophrenia, Program Advisory Committee
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- Research Evaluation Committee, National ICT Australia Ltd Member
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- NHMRC Grant Review Panel Member
- NHMRC Program Grant Panel Member

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- National Natural Science Foundation of China, Program Review Committee Member

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By improving the understanding of the fundamental mechanisms that regulate brain function, QBI researchers are working to develop new, more effective therapeutic treatments for conditions such as dementia, stroke, motor neuron disease, multiple sclerosis and neurotrauma.

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