Aerial view of the St Lucia campus looking towards the QBI building (bottom left), the Brisbane River and downtown Brisbane.
The opening of the Queensland Brain Institute building in November 2007 was a signature event for The University of Queensland. The award-winning building, commissioned by Queensland Premier Anna Bligh, does justice to QBI Director Professor Perry Bartlett’s ambitious vision to establish a globally renowned neuroscience institute at UQ. We were particularly honoured by the presence at the opening of Mr Chuck Feeney, the founder of The Atlantic Philanthropies. A gift from Atlantic, combined with funding from the Queensland Government and UQ, made the building possible.

Perry and his QBI team are energised by the goal of growing an institute that is internationally acknowledged for high-calibre research and for training excellent neuroscientists. They communicate the achievements and promise of neuroscience to community members, including children involved in the International Brain Bee. Successful engagement with people of all ages, with corporations, and with international and Australian research organisations, is an important characteristic of QBI.

In its first five years, the QBI has accomplished a remarkable body of work, won numerous prestigious awards and fellowships, and achieved high success rates in applying for external funding.

It is no small task to build a new institute from the ground up and to continually recruit, nurture and promote talented staff and students.

Professor Paul Greenfield AO
Vice-Chancellor
2007–2008 has been an exciting period for QBI, with the opening of our new building by the Queensland Premier Anna Bligh on 19 November 2007, in the presence of all the members of her cabinet and our benefactor Mr Chuck Feeney, founder of The Atlantic Philanthropies. The occasion was also marked by the Premier’s announcement of an additional $25 million in operational funding for QBI over the next five years. This generous funding provides the support that is so vital in meeting the running costs associated with QBI’s research programs. In Australia, these indirect research costs are not met by our funding bodies, but they are nonetheless essential to ensuring that our facilities can be appropriately maintained and staffed.

The opening of the new building finally allowed us to assemble all the Institute’s principal investigators under one roof, and to enhance the cross-disciplinary interactions that we envision will lead to a new and deeper understanding of how the brain functions. I am delighted to report, as you will see below, that these collaborations have already proven very fruitful.

It is exciting to observe how groups working in nervous systems as seemingly diverse as those of fruit flies (Drosophila) and humans are integrating their approaches to discover common mechanisms underpinning learning and memory. Equally important is the interaction between electrophysiologists, molecular biologists, mathematicians and cognitive neuroscientists occurring throughout the Institute.

During 2007–2008, QBI expanded from a core group of fewer than 30 scientists to a research institute with more than 200 staff, many of whom have come from interstate or overseas to work in Queensland. In January 2007, we were fortunate to attract Professor Mandyam Srinivasan FAA FRS as Professor of Visual Neuroscience. Winner of the 2006 Australian Prime Minister’s Prize for Science, ‘Srini’ and his colleagues have demonstrated that many relatively simple nervous systems nevertheless display a rich behavioural repertoire.

The Srinivasan Group’s work with honeybees seeks to elucidate principles of flight control and navigation, and to explore the limits of the ‘cognitive’ capacities of small brains. In July 2007, Srini was awarded the 2008 Rank Prize for Optoelectronics (a prestigious UK award), which was followed by the Smart State Premier’s Fellowship, the Queensland Government’s highest honour for a research scientist.

In the same year, Dr Bruno van Swinderen joined us from the Neurosciences Institute in San Diego to continue his work with Drosophila, investigating mechanisms of perception and providing insights into attention and memory, anaesthesia and sleep. Because more than half of all known human disease genes have a recognisable matching sequence in the Drosophila genome, fruit flies provide an ideal research model for many human neurological conditions.

Dr Massimo Hilliard was also recruited to the Institute in 2007, joining us from the Rockefeller University, New York. Massimo’s rapidly expanding research program is based on the nematode worm C. elegans, another valuable genetic model system that provides much sought after insights into early brain development, including neuronal polarity and axonal guidance, as well as axonal degeneration and regeneration.

“During 2007–2008, QBI expanded from a core group of fewer than 30 scientists to a research institute with more than 200 staff, many of whom have come from interstate or overseas to work in Queensland”
We have also focused on the human side of cognition with the recruitment of Professor Jason Mattingley, Professor of Cognitive Neuroscience, who has recently been made a Fellow of the Academy of Social Sciences in recognition of his outstanding contributions to the field. The cognitive area has also been substantially bolstered by the addition of Associate Professors Ross Cunnington and Mark Bellgrove, who use functional magnetic resonance imaging and molecular genetics to study human behaviour. These appointments have been made jointly with the School of Psychology and I thank Professor Debbie Terry for the support and insight that has enabled us to assemble this world-leading cohort of cognitive neuroscientists. I was delighted, also, that several senior neuroscientists who had been working for many years at the University of Queensland, and who have helped bring about the establishment of QBI, have now joined us. These included Professor David Adams, the immediate past Head of the School of Biomedical Sciences, and an acknowledged leader in the study of ion channels and the basis of pain, Professor David Vaney, a foundation member of the internationally recognised Vision, Touch and Hearing Research Centre at UQ and an expert in retinal neuroanatomy, Professor Joe Lynch, who has made a significant contribution to our understanding of the molecular basis of functional signalling in chloride channels, and Associate Professor Frederic Meunier, who is providing valuable new insights into how membrane vesicles are trafficked within neurons.

In light of this growth in research capacity, it was a significant milestone for QBI when November 2008 coincided not only with the first anniversary of the opening of the new building but also a time of outstanding success by our researchers in the annual Australian Research Council (ARC) and National Health and Medical Research Council (NHMRC) funding rounds. QBI scientists were lead investigators on twelve NHMRC project grants and three ARC Discovery grants. I was also delighted that the NHMRC Program Grant that I lead was refunded for another 5 years. Also noteworthy was the recognition accorded QBI’s Deputy Director Professor Pankaj Sah, who received an NHMRC Excellence Award as lead investigator on the top-ranking project grant in any discipline in the 2008 funding round. Pankaj’s extensive contribution to neuroscience through the use of electrophysiology, imaging and molecular techniques continues to provide insights into amygdala function that push our level of understanding about how the brain retains emotional memory.

I am also impressed by our growing number of publications, many in top neuroscience journals such as Neuron, Nature Neuroscience and Journal of Neuroscience, as these provide another indicator of the scientific excellence of QBI researchers. In addition to our human capital, the move into the new building has also provided the opportunity to realise the establishment of a number of major new facilities. First amongst these was the Australian Cancer Research Foundation (ACRF) Brain Tumour Research Centre. Funded by a $1.14 million grant from ACRF, the Brain Tumour Research Centre provides researchers with sophisticated tools to isolate, enumerate and purify populations of cells from human gliomas, and to use this capability to identify the unique characteristics of the tumour-producing cells, with the aim of eventually developing novel ways to block the growth and spread of this devastating brain cancer.
We also marked the official opening of the Peter Goodenough and Wantoks Research Laboratory, headed by Dr Robyn Wallace. Thanks to the far-sighted determination of the late Peter Goodenough, his $6 million bequest to fight motor neuron disease has already provided encouraging new insights into a disease that reaps a cruel harvest of human lives every year.

Peter’s legacy has also allowed us to honour his memory through the annual Peter Goodenough Memorial Lecture. In July 2008, eminent Australian scientist Sir Gustav Nossal presented the first of these public lectures. Sir Gus is currently a consultant for both the World Health Organisation and the Bill and Melinda Gates Foundation, as well as Chairman of the Global Foundation Advisory Committee. The range of issues Sir Gus highlighted in his presentation – Some Rays of Hope in Global Health Reform – amounted to a call-to-action for developed countries seeking to manage the burden of disease on a global scale.

Finally, towards the end of 2008, we celebrated the opening of QBI’s $2.5 million All Weather Bee Flight Facility. With nearly 200 square metres of useable flight space, the laboratory is the world’s largest indoor, climate-controlled insect flight-testing facility, and will undoubtedly play a key role in helping scientists to better understand how complex small brains function.

QBI’s new facilities, which also include a 200-seat auditorium, have become the locus for numerous neuroscience-related conferences and seminars. As well as our own long-running neuroscience seminar series (which featured more than 80 speakers in the 2007–2008 period), leading neuroscientists from Australia, China and the USA attended QBI’s Brain Plasticity Symposium in September 2008, which formed a platform for scientific discussion about the fundamental processes that regulate brain function and disease.

Earlier in the year, over a period of five weeks, we also hosted a series of well attended lectures exploring the theme of Learning and Memory, presented by internationally renowned neuroscientists from Australia and abroad.

The Institute also achieved considerable satisfaction from its interactions with various business and community organisations. Nor was the next generation of scientists and community leaders overlooked. Initiated by QBI’s Associate Professor Linda Richards, the Australian Brain Bee Challenge (ABBC) has evolved from a local neuroscience awareness initiative to a major event on the Australian school calendar, involving thousands of Year 10 students.

The ABBC is a showcase for the brightest young minds in neuroscience. In 2008, for example, more than 8500 students enrolled nationally in round one of the competition. With the support and assistance of neuroscientists around Australia, the event introduces a wide cross-section of young people to neuroscience in a positive environment that encourages and rewards excellence.

Despite the importance of our local and interstate links, QBI is also taking to the international stage with a growing number of strategic alliances. In 2008, these included the establishment of formal research ties with Japan’s most prestigious medical school, the Keio University School of Medicine in Tokyo, which is recognised around the world for its teaching excellence and leading research into neural stem cells. As such, the synergies between Keio University and QBI promise some exciting collaborations.

The Memorandum of Understanding between QBI and Keio University School of Medicine was the fifth strategic research alliance that QBI has made in the Asia-Pacific region during the past three years. QBI also has research links with the RIKEN Brain Science Institute (Japan), the University of Auckland (NZ), the Chinese Academy of Science (CAS) Institute of Neuroscience in Shanghai, the Medical University of South Carolina (USA) and Zeiss Australasia. Toward the end of 2008, we also embarked on an major new initiative with CAS’s Institute of Biophysics which will lead to the establishment of joint laboratories in Beijing and Brisbane.

The past two years have been tremendously exciting for the advancement of neuroscience. While highlights of our successes has been singled out for special mention, I must emphasise that QBI is privileged to enjoy loyal support from its donors and benefactors. Furthermore, our dedicated postdoctoral fellows, research assistants, students and support staff, led by Deputy Director (Operations) John Kelly, have all contributed to the Institute’s burgeoning success.

I would also like to thank our Development Board, chaired by David Merson, for their strong support and generous help with raising the public profile of QBI during this period, and for initiating a program aimed at raising funds to underpin the Institute’s future research initiatives. I especially want to thank Jeff Maclean and his family for their continued generosity, enthusiasm and friendship, which was vital in the early growth of QBI and has never flagged in the ensuing years.

Finally, I wish to acknowledge the vision of two Vice Chancellors, Professor John Hay and Professor Paul Greenfield, who helped drive the establishment of QBI, and who continue to give me enormous personal support.

Professor Perry F Bartlett FAA
Director
The imposing foyer of QBI provides views of the research laboratories on four floors through Fiona Hall’s mural Out of Mind, which is a transparent digital graphic design incorporated into an internal glass wall.
QBI BUILDING

The Queensland Brain Institute building is the outcome of a competition to design a research facility for the University of Queensland dedicated to understanding the brain. The new building houses a remarkably broad gathering of scientific researchers with strong links to associated schools, institutes, centres and commercial bodies.

This great diversity was the catalyst for an overarching theme for the project – A Theatre for Research. It frames an architectural approach that encourages the fertile cross-pollination of ideas. To this end, the building exaggerates the visibility of research activity, placing the laboratories on display upon entry. Circulation routes are elevated from the prosaic to become lively promenades that are attached to informal conversation spaces important for conducting impromptu research in a more social environment. Our philosophy was to maximise staff interaction and collaboration. Communal hubs and informal meeting spaces are the settings for discussion and exchanging ideas.

The network of circulation leads to the public spaces of auditorium, seminar room and terrace and the communal lounge at the top of the building. We understand that these spaces are heavily utilised within the University and are also important for bringing the wider realm of government, industry, schools and public visitors to the Institute to better understand its ongoing research. To reach these upper spaces, the visitor is drawn past the researchers at work, the intellectual activity on theatrical display, and so as a prelude they gain insight into the Institute.

Overlaid on this window into the laboratories is a new artwork entitled Out of Mind by Adelaide-based artist Fiona Hall. This work, which was especially commissioned by the University of Queensland, comprises a large digital print image that interprets the research activity beyond. This overlap of the creative arts and creative research emphasises the multi-disciplinary approach for the Institute.

As architects for the project, we have received very positive feedback and commentary from Institute members and their visitors, particularly in relation to the success of the theme of A Theatre for Research. The building appears to have created a foothold in the imagination of the University, Brisbane and beyond, from which the research groups within can successfully launch their own work. The project has also been recognised by our architectural peers and has received several awards including:

- National Commendation for Interior Architecture. Australian Institute of Architects National Architecture Awards
- Brisbane Region Building of the Year. RAIA Queensland Regional Architecture Awards 2008
- Inaugural Art and Architecture Award for Out of Mind by Fiona Hall. RAIA Queensland Regional Architecture Awards 2008
- Award for Public/Institutional Interior Design. 2008 Interior Design Awards

Both John Wardle Architects and Wilson Architects feel very fortunate to have had the opportunity to work with such an enthusiastic and passionate client group. The clarity of vision for the Institute, and the collaborative spirit in which the project was undertaken, provided the starting point for a project that we are all proud to have been part.

John Wardle Architects and Wilson Architects

Contemporary Australian artist Fiona Hall with installation architect Ted Chen and QBI Director Perry Bartlett at the official launch of Ms Hall’s artwork Out of Mind
The Premier of Queensland, The Hon Anna Bligh MP, officially opened the Queensland Brain Institute on 19 November 2007, just a month after the scientists moved in from their temporary home in UQ's Ritchie Laboratories.

Speaking at the opening of the $63 million facility, QBI's Inaugural Director, Professor Perry Bartlett, thanked those people whose vision and generosity had made the Institute a reality, all of whom were in the audience. "It is with enormous appreciation that I thank both UQ Vice-Chancellor Professor Hay and the executive members of the Queensland Government for the outstanding support and leadership they have demonstrated in backing this initiative" said Perry Bartlett.

"I also thank Mr Chuck Feeney and The Atlantic Philanthropies, without whose generosity and farsightedness QBI's members would not have such a wonderful home in which to pursue scientific excellence and increase our international leadership in neuroscience."

Professor Bartlett also acknowledged that QBI was built on the strong foundations in neuroscience at the University of Queensland that had been laid by Professors Jack Pettigrew, David Vaney and David Adams of the School of Biomedical Sciences.

Prior to the opening, QBI had the honour of hosting a Queensland Government Community Cabinet, which provided the Institute with an opportunity to showcase its achievements to the Premier and her ministerial team. Anna Bligh used the occasion to announce that the Queensland Government would provide an additional $25 million for the operational funding of QBI over the next five years, bringing its total support to $45 million.
The $2.5 million All Weather Bee Flight Facility was opened on 20 August 2008 by the Hon Desley Boyle MP. With nearly 200 m² of useable flight space, it is the world's largest indoor, climate-controlled insect flight-testing facility, and is playing a key role in helping QBI scientists understand how relatively simple brains are capable of complex functioning.

Professor Perry Bartlett noted at the opening of the facility that “Bees offer a good research model because they can be studied in a natural setting that is easily accessible to scientists. Important from a neuroscience perspective is the fact that, although the bee brain is only about the size of a sesame seed, it has many of the characteristics of the human brain, including complex behaviours such as learning and memory.”

ACRF Chairman, Mr Tom Dery, said that the Centre “would be the first automated high throughput screening facility designed for testing and identifying stem cells from human brain tumours.”

A generous $1,140,000 grant from the Australian Cancer Research Foundation (ACRF) has funded a new tumour-cell testing facility that is being used to develop more effective treatments for brain cancer. The ACRF Brain Tumour Research Centre was officially opened on 5 March 2008 by the Queensland Minister for Health, the Hon Stephen Robertson MP.

Professor Perry Bartlett said that, despite significant advances in treatments, the average life expectancy of patients with aggressive forms of brain cancer was often less than a year. There was an emerging view amongst neuroscientists that such cancers may contain a population of stem cells responsible for tumour initiation and malignancy. “Until now, one of the prime difficulties in studying these stem cells was that scientists lacked the tools to identify and collect them – this is the first time researchers will be able to isolate, enumerate and purify tumour stem cells from human gliomas with such high levels of efficiency.”

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Mr Peter Goodenough was a charismatic north Queensland businessman who forged a trucking and construction business empire in Papua New Guinea (PNG) in the 1970s and 1980s. Before motor neuron disease ended his life in 2004, Mr Goodenough bequeathed $6 million to QBI to assist finding a cure for this debilitating neurological disease. This philanthropic donation, which was the second largest ever made to UQ, has also enabled both the creation of three PhD scholarships for PNG students in the disciplines of engineering, law and neuroscience, and the endowment of the annual Peter Goodenough Memorial Lecture in philanthropy.

The Peter Goodenough and Wantoks Research Laboratory was opened on 19 March 2008 by the State Minister for Tourism, Regional Development and Industry, the Hon Desley Boyle MP. ‘Wantoks’ means close friends or relatives in pidgin English – a reference to Mr Goodenough’s three pet dogs (pictured below) and ‘best mates’, whom he wanted recognised. The Peter Goodenough and Wantoks Research Laboratory is home to the Molecular Genetics of Human Disease Lab headed by Dr Robyn Wallace.

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The facility is a vital part of the research work of the Visual and Sensory Neuroscience Group headed by Professor Mandyam Srinivasan, who is internationally recognised for his groundbreaking discoveries concerning bee vision, navigation, perception and cognition.
Honours for Professor Srinivasan

Professor Mandyam Srinivasan FAA FRS, who received the Prime Minister’s Prize for Science in 2006, has added to his impressive list of honours. In July 2007, ‘Srini’ was awarded a Smart State Premier’s Fellowship. The Queensland Government provided $1,250,000 funding over five years, with matching funding from UQ. Srini’s research will investigate how unmanned aerial vehicles can be improved by a better understanding of how bees detect, chase and intercept moving targets.

In July 2007 Srini also gave the ANS Plenary Lecture at the 7th IBRO World Congress of Neuroscience in Melbourne. Then, in August, it was announced in London that Srini had been awarded the 2008 Rank Prize in Optoelectronics for his work on insect vision and its implications for robotics.

QBI Researcher Awarded Smart State Fellowship

Dr Xiaoying Cui, who is a postdoc in the McGrath Group, was awarded a Smart State Fellowship by the Queensland Government in March 2008. Xiaoying is studying whether low levels of vitamin D in the foetus can impair brain functions in the adult and increase the risk of developing schizophrenia.

International Charity Supports Young QBI Faculty Members

Assoc Prof Mark Bellgrove and Dr Louise (Luli) Faber received Young Investigator Awards from NARSAD (National Alliance for Research on Schizophrenia and Depression), which is the world’s leading charity dedicated to mental health research. The subject of Mark’s project is Studying attention networks in the attention-deficit hyperactivity disorder (ADHD) brain with functional MRI: influence of genetics and stimulant medication. Luli is investigating Mechanisms underlying the functioning of the prefrontal cortex.
QBI Research Fellow Receives Prestigious Award

The 2009 AW Campbell Award of the Australian Neuroscience Society was awarded to Dr Michael Piper. This annual award recognises the Australasian researcher who has achieved the highest standard of work in neuroscience during their first five postdoctoral years. The list of previous winners reads like a Who’s Who of Australian neuroscientists, and includes QBI researchers, Pankaj Sah and Elizabeth Coulson.

Michael undertook a PhD with Melissa Little at UQ’s Institute for Molecular Bioscience before heading to Cambridge University for a three-year postdoc. Since returning to UQ in 2006 as a NHMRC Howard Florey Centenary Research Fellow, Michael has been working in the Richards Lab at QBI. Michael’s research examines how neural stem cells acquire their identity to form the many different cell types that make up brain tissue. His outstanding track record was also recognised by the recent award of a NHMRC Career Development Award for 2009-2012.

NHMRC Grants & Fellowships Awarded in 2008

QBI researchers were awarded 12 NHMRC project grants in the 2008 grant round. A 5-year NHMRC Program Grant totaling $7,627,200 was awarded to Professor Perry Bartlett and Professor Pankaj Sah of QBI, and to Professor Seong-Seng Tan and Professor Trevor Kilpatrick of the Howard Florey Institute, to study the development and refinement of neural connections in the adult brain in health and disease.

NHMRC Senior Research Fellowships were awarded to Professor Joe Lynch (Level B) and Assoc Prof Frederic Meunier (Level A) for 2009–2013. NHMRC Career Development Awards were awarded to Assoc Prof Mark Bellgrove (Level 2), Dr Elizabeth Coulson (Level 2) and Dr Michael Piper (Level 1) for 2009–2012. A NHMRC Training Fellowship was awarded to Dr Adam Hamlin for 2009–2012. These awards follow on from the previous years’ awards of a NHMRC Career Development Award (Level 1) to Dr Robert Hester for 2008–2011, and a NHMRC Training Fellowship to Dr Tim Silk for 2007–2010. These NHMRC-funded research fellows join Assoc Prof Linda Richards, who holds a NHMRC Senior Research Fellowship (Level B), and Professor David Vaney, who transferred his NHMRC Principal Research Fellowship from the School of Biomedical Sciences to QBI in 2008.

UQ Foundation Research Excellence Award

The NHMRC Career Development Award capped an exceptional year for Dr Elizabeth Coulson. She was also awarded a 2008 Research Excellence Award by the UQ Foundation. These Awards recognise outstanding performance and leadership potential in early-career researchers at UQ.

Lizzie is studying what causes healthy nerve cells to switch off and die – a characteristic associated with many neurodegenerative conditions, including Alzheimer’s disease.

QBI Study Judged Top Neuroscience Paper

The Paxinos-Watson Prize is awarded for the most significant neuroscience paper published by a member of the Australian Neuroscience Society. The 2008 Prize was awarded to a 2006 paper by Thomas Keeble, Michael Halford, Clare Seaman, Nigel Kee, Maria Macheda, Richard Anderson, Steven Stacker and Helen Cooper, published in Journal of Neuroscience 26: 5840-5848.
IBRO Satellite Meeting on Neurodevelopment 2007

Assoc Prof Linda Richards from QBI and Professor Seong-Seng Tan from the Howard Florey Institute organised an international conference on Stem cells, migration and axon guidance in the developing and adult brain. This 3-day satellite meeting of the 7th IBRO World Congress of Neuroscience was held in Cairns in July 2007 and attended by 120 registrants from 14 countries.

Invited lectures were given by Perry Bartlett (QBI), Alain Chedotal (Uni Paris), Gord Fishell (NYU), Magdalena Gotz (Uni Munich), Arnold Kriegstein (UCSF), Zoltan Molnár (Uni Oxford), Peter Mombaerts (Max Planck Institute), Fujio Murakami (Osaka Uni), Hideyuki Okano (Keio Uni), John Parnavelas (UCL), Mu-Ming Poo (UC Berkeley & ION Shanghai), John Rubenstein (UCSF), Li-Huei Tsai (MIT), and Samuel Weiss (Uni Calgary). Another 17 speakers gave shorter talks and there were 48 poster presentations. Social highlights included a snorkeling trip to Green Island, an excursion on the Kuranda Railway, and an Australian BBQ dinner on the final night.

Brain Plasticity Symposium 2008

Two years after the inaugural Brain Plasticity Symposium was held in 2006, the 2nd Symposium brought together scientists from Australia, China, New Zealand, the United Kingdom and the United States for a 3-day meeting at the Institute in September 2008.

Perry Bartlett noted at the opening that: “Our understanding of brain function in both health and disease is growing at a very fast rate and possible cures for conditions such as age-related dementia can be envisaged in the not too distant future. This symposium provides a unique opportunity to hear world-leading neuroscientists discuss their latest research into the mechanisms that underlie learning and memory as well as disease states.”
Vision Down Under 2007:
Jack Pettigrew’s Festschrift

QBI and the ARC Centre of Excellence in Vision Science were the principal sponsors of Vision Down Under 2007, which was held near Cairns from 19-22 July 2007. VDU 2007 was the largest gathering of visual neuroscientists ever held in Australia, attracting 140 registrants from this country and 100 from overseas. The conference, which was organised by Professor David Vaney, incorporated Festschriften for two of Australia’s most distinguished visual neuroscientists, Professor John (Jack) Pettigrew of the University of Queensland and Professor Bogdan Dreher of the University of Sydney.

For 24 years, Jack Pettigrew combined responsibilities both as the Chair of Physiology and as the Director of the Vision, Touch & Hearing Research Centre, which was Queensland’s first ARC Special Research Centre. VTHRC’s researchers were highly regarded internationally and 14 of the Centre’s former postdocs now hold professorships at universities in Australia, Canada, China, England and the USA. In 1987, Jack was awarded a Fellowship of the Royal Society of London, becoming only the third person at UQ to be made a member of the world’s oldest and most prestigious scientific academy.

Jack is renowned for his innovative and eclectic science but his most enduring legacy may be the strong foundation he laid for brain research at UQ. At Jack’s Festschrift Dinner, Perry Bartlett announced that QBI would be endowing a PhD scholarship in neuroscience to commemorate Jack’s contributions to UQ. The inaugural Jack Pettigrew Scholar is Melissa de Vries from the Cooper Lab, who is studying the role of guidance receptors in zebrafish neurulation.

“Jack is renowned for his innovative and eclectic science but his most enduring legacy may be the strong foundation he laid for brain research at UQ.”
LECTURES & COURSES

Peter Goodenough Memorial Lecture
The outstanding bequest of Mr Peter Goodenough that established The Peter Goodenough and Wantoks Research Laboratory also endowed a Memorial Lecture in Peter’s name. The Lecture was established not only to remember the benefactor but also to highlight the importance of philanthropy in research.

The inaugural lecture, A search for global health: a new 21st century paradigm, was given by Sir Gustav Nossal AC CBE FRS FAA on 15 July 2008. In summing up his broad-ranging lecture, Sir Gus noted that “Somehow the penny’s dropped, we’re all living on the one spaceship and we’ve got to figure out some way of getting a better health deal for mankind’s poorest.”

Toshiya Yamada Memorial Lecture
The Toshiya Yamada Memorial Lecture, a joint initiative of QBI and the Institute for Molecular Bioscience (IMB), aims to highlight the work of leading neuroscience researchers. This public lecture, held on an annual basis during Brain Awareness Week, was named in honour of the late Dr Toshiya Yamada, a gifted neuroscientist who passed away in 2001.

Toshi, as he was affectionately known, was an IMB researcher who discovered the molecules that are essential for regulating the correct wiring of the spinal cord and parts of the brain. Toshi played an important role in the resurgence of Australia as a leader in the field of developmental neurobiology, and many of his conclusions have since become textbook entries, a legacy few scientists can claim.

The 2007 lecture was given by Professor Ryoichiro Kageyama, Director of the Institute for Virus Research, Kyoto University, Japan. His talk was entitled Hes and proneural bHLH genes antagonistically regulate maintenance of neural stem cells, neurogenesis and gliogenesis.

The 2008 lecture was given by Assoc Prof Jozef Gecz, Head of the Neurogenetics Research Program, Department of Genetic Medicine, University of Adelaide. His talk was entitled The genetic landscape of learning and memory: what do we learn from naturally occurring mutations?

Each lecture was attended by about 150 people – both professionals and interested members of the public.

Advanced Neuroscience Lectures 2008
In April–May 2008, QBI hosted a series of Advanced Neuroscience Lectures on the general theme of Learning and Memory. The five public lectures occupied an extended timeslot of 1.5–2 hours, to give the lecturers plenty of time to introduce and develop their topics.

The first lecture was given by Professor Cliff Abraham of the University of Otago, New Zealand, on The search for the engram: mechanisms of learning and memory. This was followed in successive weeks by lectures from three QBI neuroscientists. Associate Professor Bruno van Swinderen talked about Learning from flies, Professor Jason Mattingley talked about Mechanisms of selective attention and their role in human perception, and Professor Mandyam Srinivasan talked about The honeybee as a model for the study of vision, navigation, learning, memory and ‘cognition’. The final lecture was given by Professor Greg Stuart of the Australian National University on Cortical function.
The Mammalian Brain – ‘A Guide for Tourists’

Professor Charles Watson from Curtin University and Prince of Wales Medical Research Institute conducted a two-day Brain Mapping Workshop at QBI in August 2008. This workshop was an opportunity for participants to improve their confidence in identifying major structures in the brain and spinal cord.

The workshop was based around a series of presentations, each followed by a practical session in which groups of participants attempted to label photographs of brain sections. Over 160 registered for the workshop, but only about 80 returned for the second day – an indication of the demands of the practical sessions that some had not expected. The level of interaction during the workshop was high; a quiz at the end of the workshop showed that almost all of those who came for the second day had reached an admirable level of mastery of brain anatomy, and some were outstanding.

Charles Watson is a neurobiologist and public-health physician. He has had over 25 years’ experience teaching brain anatomy to medical and science students. He and George Paxinos have collaborated for 30 years in the production of a notable series of brain atlases, one of which has been cited over 43,000 times. Charles’ current research is focused mainly on gene expression in hindbrain development, mapping MRIs of the mouse brain, and the construction of an atlas of the marmoset brain.

Australian Course in Advanced Neuroscience (ACAN)

ACAN is Australia’s answer to the legendary summer programs in biomedical and biological sciences run at Cold Spring Harbor and Woods Hole in the USA. The course, which was founded by Dr Alan Finkel and is now run under the auspices of the Australian Neuroscience Society, has been held every year since 2005 at UQ’s Moreton Bay Research Station on Stradbroke Island, off the coast of Brisbane.

Leading Australian and overseas researchers work with a group of 12 graduate students and postdocs to provide rigorous training in the theory and practice of cellular neuroscience. During the three-week course, the young neuroscientists attend lectures on the fundamentals of cellular neuroscience, receive extensive hands-on laboratory training in patch-clamping and neuronal imaging using the latest equipment provided by the manufacturers, and undertake a mini research project of their own choosing.

QBI researchers have played an integral role in the success of ACAN. Pankaj Sah, Joe Lynch and Louise Faber serve as instructors on the course, Andrew Delaney and John Power have assisted with the teaching, and Pankaj and David Vaney currently serve on the ACAN Management Committee. Usually one or two of the students on each course come from QBI. In addition, the proximity of the Institute to the Research Station means that QBI can be relied upon to provide missing consumables at short notice or replace faulty equipment.

In Australia there is little support for postgraduate training from the ARC and NHMRC, outside of the direct scholarship funding awarded to individual students. Moreover, any new initiatives in postgraduate education established by individual universities are only available to students at that institution, so the scope for intermural training in Australia is regrettably limited. ACAN provides a superb model of how Australian scientific societies can play an active role in intermural postgraduate research training. However ACAN costs about $100,000 to run each year and its continued success will require an injection of funds from external sources.
QBI neuroscientists maintain active research collaborations with many colleagues throughout Australia and the rest of the world.

QBI has established formal partnership agreements with a number of key institutions in the Asia-Pacific region, reflecting the sustained growth of neuroscience research in our part of the world. Five agreements were signed on behalf of QBI in 2006–2008.

Within the University, the Institute has close ties with the Centre for Magnetic Resonance, the Institute for Molecular Bioscience, the Australian Institute for Bioengineering & Nanotechnology, the Queensland Centre for Mental Health Research, and UQ’s School of Biomedical Sciences, School of Psychology, School of Information Technology & Electrical Engineering, and School of Mathematics & Physics.

Institute of Neuroscience, Chinese Academy of Sciences, Shanghai, China

The Institute of Neuroscience (ION), which was established in 1999, is devoted to research in all areas of basic neuroscience. ION currently has 25 faculty members and aims to reach a steady-state of 30 labs by 2010.

Perry Bartlett said that “QBI and ION have many common interests, so it makes good sense to work together to accelerate the discovery processes that will lead to the development of new therapeutics to treat mental and neurological diseases. The importance of this relationship should not be underestimated, since China is set to become a major force in the Asia-Pacific.”

University of Auckland, Faculty of Medical & Health Sciences, New Zealand

The Faculty of Medical and Health Sciences was established in 1968 as The University of Auckland School of Medicine. The School of Medical Sciences is very strong in neuroscience research and has a variety of outstanding facilities that complement those at QBI, including an internationally recognised Human Brain Bank and the Biomedical Imaging Research Unit.

According to Tom Barnes, DVC Research at Auckland University, “Interest in neuroscience is growing strongly in New Zealand and the new agreement will encourage the exchange of ideas as research in this field has doubled in the past five years.” Perry Bartlett and Richard Faull from the Department of Anatomy at Auckland University later spoke at a QBI public seminar about neurogenesis and new approaches to treating brain disease.
Professor Hideyuki Okano and Professor Perry Bartlett sign the partnership agreement at QBI on 26 November 2008.

Dr Ray Greenberg, President of MUSC, and Mr John Kelly, QBI Director of Operations, sign the agreement in the presence of Dr Mark Kindy and the Queensland Premier.

Professor Shun-ichi Amari, Director of RIKEN BSI, and Professor Perry Bartlett sign the partnership agreement in Saitama on 6 September 2007.

Mr Beattie noted that “MUSC has great expertise in animal models of dementia, like Alzheimer’s, which will allow QBI to test recent discoveries of how to delay the onset of dementia using molecules to stimulate production of nerve cells.” At the signing ceremony on 9 May 2007, Mr Beattie was presented with MUSC’s Presidential Merit Award for promoting collaboration between South Carolina and Queensland.

Medical University of South Carolina, Charleston, South Carolina, USA

South Carolina is Queensland’s Sister State in the USA. While on a trade mission to the state, the Premier of Queensland, The Hon Peter Beattie MP, observed the signing of the Memorandum of Agreement between MUSC and QBI.

Mr Beattie noted that “MUSC has great expertise in animal models of dementia, like Alzheimer’s, which will allow QBI to test recent discoveries of how to delay the onset of dementia using molecules to stimulate production of nerve cells.” At the signing ceremony on 9 May 2007, Mr Beattie was presented with MUSC’s Presidential Merit Award for promoting collaboration between South Carolina and Queensland.

Keio University, School of Medicine, Tokyo, Japan

The Keio University School of Medicine is Japan’s most prestigious medical school and is recognised internationally for its teaching excellence and leading research into neural stem cells.

Perry Bartlett said that “The synergies between Keio University and QBI promise some exciting collaborations. Like QBI, researchers at the Keio University’s Department of Physiology are also seeking to elucidate the basic mechanisms of brain function and development. Both research groups are seeking to develop new therapeutic strategies for the treatment of neurological diseases and injury.”

Hideyuki Okano, Chair of the Department of Physiology at Keio University School of Medicine, is internationally recognised for his research on the development and regeneration of cells of the nervous system. He leads a large research group that has been working to isolate stem cells from bone marrow.
The University of Queensland and the Queensland Brain Institute have established the QBI Development Board to assist in the strategic planning and development of the Institute. This includes helping to build greater community awareness of the neuroscience research being undertaken at QBI and to provide advice and leadership in all aspects of external fundraising. The Board members also represent the public interest in terms of accountability.

The Board, which meets quarterly in an honorary capacity, is comprised of Professor Paul Greenfield, (Vice-Chancellor of UQ), Professor Perry Bartlett (Director of QBI) and six external members.

Mr David Merson (Chairman), who was the founder of Mincom Ltd, has become director of a number of Australian software companies, research institutes and charitable bodies since retiring from Mincom in 2002. His achievements have been recognised with an Honorary Doctorate from UQ, a Centenary Medal from the Australian Government, the Export Hero award from the Australian Institute of Export, the inaugural Gold Award of the Australian Information Technology Association, and the inaugural CSIRO iAward for individual achievement in the IT industry.

Commissioner Bob Atkinson APM has had a 40 year career with the Queensland Police Service, serving throughout the State from Goondiwindi to Cairns, and performing a wide range of operational and managerial roles. He was appointed Commissioner of the Queensland Police Service in 2002.

Ms Sallyanne Atkinson AO is Co-Chair of the Commonwealth Government’s Dementia Advisory Group and Chair of the Crawford Fund (Queensland). She is a former Lord Mayor of Brisbane, Chair of Tourism Queensland, and Australian Senior Trade Commissioner to France.

Mr Mark Gray has served in senior management roles for over 35 years in both the private and public sectors, including extensive experience in the Queensland and Commonwealth Governments. Prior to joining BDO Kendalls as an Executive Director, he was the Brisbane Office Head for the Macquarie Group from 2002–2008 and a Division Director in Macquarie Capital Advisers.

Mr John Lyons, who was the Founder and Chairman of Marketshare, is now an independent company director with a special interest in high-growth innovative companies. He is also a board member of the Royal Children’s Hospital Foundation and the Jupiters Casino Community Benefit Fund, and a Queensland Councillor of the Australian Institute of Company Directors.

Mr Jeff Maclean is CEO and Executive Director of the Index Group of Companies. The Maclean family were early supporters of QBI: in 2004 they established the Ross Maclean Senior Research Fellowship to fund research into motor neuron disease, which sadly claimed the life of Jeff’s father Ross in 2005. Jeff is also on the Brisbane Red Shield Appeal Launch Committee.
Indeed, this has already proven to be the case. The Meunier Lab, which studies the molecular dynamics of synaptic plasticity, has identified a possible mechanism for preventing the neurological effects of certain bacterial toxins. This research is still in its early stages, with work being undertaken to validate the findings and potentially develop therapeutic outcomes. Research in the Reinhard and Claudianos Labs is unravelling the molecular and neurological mechanisms of odour detection, providing fundamental knowledge about how complex odours are processed. This research has many applications, particularly in the food industry, where it may shed light on the sensory basis of consumer preferences for different wines.

Many neurological disorders, such as Alzheimer’s disease and Parkinson’s disease, are associated with a reduction in the number of nerve cells. QBI researchers are taking two approaches to address this problem: the first is to develop therapeutics that prevent cell death and the second is to develop therapeutics that stimulate the production of new neurons.

Research in the Coulson Lab has shown that a region of the p75 neurotrophin receptor, called the Chopper domain, is critical for inducing cell death. QBI has engaged an industry expert to assist in the development of a protein to block this receptor-induced cell death.

Building on the previous discovery that the brain contains stem cells, research in the Bartlett Lab has also identified several factors involved in the proliferation, differentiation and migration of stem cells to brain sites in need of repair. The QBI researchers are working to apply these findings to the generation of therapeutics that can stimulate the production of new neurons in the brain.

Each year more than 300 people in Australia suffer a paralysing spinal cord injury. The Bartlett Lab has been working with researchers from CSL Limited, the University of Melbourne, the Queensland Institute of Medical Research, the University of Western Australia, and elsewhere at UQ, to develop a therapeutic to stimulate the repair of newly damaged spinal cord. After nearly three years of involvement with the project, CSL Limited took an exclusive license to this promising technology in February 2009.
The Australian Brain Bee Challenge (ABBC) was initiated by the Queensland Brain Institute in 2006 with the support of the University of Queensland, the Faculty of Science, and a major external sponsor, Carl Zeiss Australia.

ABBC, a competition for Year 10 high school students, is designed to stimulate, encourage and support their interest in science generally and in neuroscience in particular. Moreover, by engaging the students’ teachers, families and the broader community, ABBC is an important part of QBI’s outreach activities.

ABBC was founded by Assoc Prof Linda Richards, who continues to guide and develop the initiative as the National Chair of ABBC. Rhonda Lyons from QBI is responsible for the national administration of ABBC and Ian Duncan has provided invaluable software support.

The students taking part in Brain Bee learn about neuroscience through three rounds of challenges spread over a year. The students begin with a quiz in their schools and then progress to the State Finals held in universities throughout Australia and New Zealand. At the State Finals, students compete in Team Challenges to win Zeiss microscopes for their schools and in Individual Challenges to progress to the National Final. The students take part in focused activities created to provide an understanding of what it means to work in science.

The Australia-New Zealand National Final was held at QBI in 2006 and 2008, and at the International Brain Research Congress in Melbourne in 2007. Each National Final winner was then eligible to represent their country in the International Brain Bee, competing against students from around the world.

In 2006 the ABBC targeted students from South-East Queensland and Northern New South Wales and 240 students took part. The 2006 champion was Timothy Mew from St Paul’s School in Brisbane, who came fifth at the International Brain Bee competition in Baltimore, Maryland.

In 2007 the ABBC was expanded to include four Australian States and one district in New Zealand, resulting in a tripling of the number of competitors. The 2007 champion was Quinn McGennisken from Lavalla Catholic College in Traralgon, Victoria, who came fourth at the International Brain Bee competition in Montreal, Canada.

In 2008 almost 5,000 students competed in the ABBC, including entrants from every Australian state, the ACT, and the North and South Islands of New Zealand. The 2008 Australian Champion was Jayson Jeganathan of Baulkham Hills High School in Sydney, with Stephen Mackereth of King’s College in Auckland taking out the New Zealand title. The runner-up, Casey Linton of Somerset College in Brisbane, represented Australia at the International Brain Bee held in Toronto, Canada, in 2009. Casey came second and Stephen came fourth, which is a magnificent result.

Top students are choosing science at university as a direct result of being involved in the ABBC and a number of students who participated in this competition now work in neuroscience and associated fields while they undertake their university studies.

ABBC National Chair, Linda Richards, with Timothy Mew (Australian Champion 2006) and James Bennett (Queensland Champion 2007), both from St Paul’s School in Brisbane.

2008 ABBC Finalists. Front row, from left: Jayson Jeganathan (NSW), Kieran Bunn (NZ-South Island), Jack Lowe (SA), Stephen Mackereth (NZ-North Island), Stephanie Mercuri (Vic) Back row, from left: Casey Linton (Qld), Yasmin Soliman (WA), Hayden Lee (ACT), Katie Dyke (Tas)
Michelle Wang, an honours student in the Mattingley Lab, wears an EEG recording cap for a brain-wave study.
Jay Spampinato, a postdoc in the Bartlett/Sah Labs, studies the microcircuitry of the amygdala using a patch-clamp recording rig.
The human brain is composed of 100 billion brain cells, or neurons; each of which communicates with 1,000 other neurons on average. One of the biggest challenges in neuroscience is to understand the processes underlying communication between connected neurons.

Neurons are connected by synapses, which allow the electrical signal in one neuron to pass across the gap to the next neuron, in a process called synaptic transmission. At the synapse, the conveyed electrical signal is converted into a chemical signal.

In presynaptic neurons, the chemical signal (neurotransmitter) is stored in small vesicles and released by the fusion of these vesicles with the outer membrane of the neuron. Once released, the neurotransmitter diffuses across the synapse and binds to selective receptors on the postsynaptic neuron. This activates these receptors, thereby eliciting an electrical response in the postsynaptic neuron. During life, the strength of connections between neurons changes (synaptic plasticity) and it is this process that is thought to underlie learning and memory in the brain.

Scientists at QBI are working at unravelling the processes underlying synaptic transmission and synaptic plasticity, using approaches that focus on each stage of these processes, at both the pre- and postsynaptic neuron. They are investigating the mechanisms underlying fusion of vesicles to the plasma membrane in the presynaptic neuron, how neurotransmitters act on postsynaptic receptors to relay information, and the role that other proteins, called ion channels, play in relaying synaptic transmission.

The Sah Lab is investigating the mechanisms underlying synaptic transmission and synaptic plasticity in a brain region called the amygdala. This region is a limbic structure that generates emotions, with malfunctions in the amygdala leading to anxiety disorders and post-traumatic stress disorder. By understanding synaptic transmission and plasticity in this brain area, the researchers hope to pave the way for new treatments for anxiety-related conditions.

The Faber Lab aims to understand synaptic transmission and synaptic plasticity in another part of the brain involved in emotion — the prefrontal cortex. This region is also essential for higher brain functions such as personality, imagination and memory, and brain disorders such as schizophrenia, depression and attention deficit hyperactivity disorder are thought to arise from problems in this brain region.

The Lynch Lab is involved in understanding the functioning of two neurotransmitter receptors, called the GABA and glycine receptors. Both of these receptors are important in regulating pain transmission and, through this work, the researchers hope to find new drugs to alleviate pain. The Lynch Lab studies these receptors by looking at how the structure of the receptors governs their function.

Finally, the Adams Lab uses toxins derived from cone shells to understand the functioning of ion channels. Cone shell toxins contain a very high number of compounds that may be useful clinically. The researchers study the mechanisms of action of these compounds in the hope of developing new drugs for chronic pain or psychiatric disorders.

“Scientists at QBI are working at unravelling the processes underlying synaptic transmission and synaptic plasticity, using approaches that focus on each stage of these processes, at both the pre- and postsynaptic neuron.”
A bullwhip amacrine cell in the chicken retina injected with dye and volume-rendered following confocal microscopy. (Nick Nacsa, research assistant in the Vaney Lab)
The Sensing Brain

Research at QBI on sensory processing in the brain provides knowledge about both the basic biology of sensation and health problems associated with our senses.

We receive sensory input via sight (eyes), smell (nose), taste (mouth), sound (ears), and touch (skin). Disorders in our sense organs or difficulty processing sensory inputs leads to problems interpreting the world, potentially compromising learning ability, social relationships, emotions, and even self-esteem. To understand sensory processing in the brain, QBI neuroscientists are working in diverse areas such as behaviour, physiology, molecular genetics, anatomy, engineering and computation.

Sensory information is translated both in the peripheral sense organs and centrally in the brain, as exemplified by research on the visual system at QBI. The Vaney Lab is studying how the many types of neurons in the retina process visual information before it is transmitted along the optic nerve to the higher visual centres in the brain. Recordings of the synaptic currents in individual retinal neurons show how excitatory and inhibitory inputs interact to code complex visual information. The Mattingley Lab studies how the human brain further processes visual information, using a combination of brain-wave recordings and advanced brain-imaging techniques to map visual responses.

How do we discriminate between thousands of sensory inputs so that our brain makes sense of our world? The Reinhard and Claudianos Labs use complex behavioural methods to help identify the molecular, cellular and physiological changes in the brain resulting from sensory stimulation.

Evidence from studies on the honeybee’s sense of smell suggests that changes in sensory acuity (the ability of the sense organs to receive input) are based on specific molecules on nerve cells in the antennae (insect nose) that can detect and interact with a smell molecule. The brain simplifies a world of different smells by selectively filtering information before forming new nerve connections, which in turn form new memories and generate a physical response. Understanding molecular and cellular processes that respond to sensory stimulation gives us new insights into the causes and possible cures of sensory processing disorders such as autism.

How can knowledge of the ‘sensing brain’ be used to develop new technology? The Srinivasan Lab studies how flying creatures as diverse as birds and bees use vision to navigate safely in cluttered environments.

Have you ever noticed when travelling in a car that trees in the distance are not moving while trees at the side of the road flash by? This phenomenon is called ‘optic flow’: the amount of motion experienced by our eyes is used to gauge how quickly we are moving through our environment, and how close an object is to us. Visual cues derived from optic flow are commonly used by organisms to navigate to a food source or to intercept a threat. These observations are inspiring engineering solutions for developing machine vision systems to guide planes and explore the surface of neighbouring planets.

Our observations point to several common underlying principles. The brain is a plastic organ moulded by our sensory experience: it never stops changing and requires stimulation throughout life to remain healthy.

"The brain is a plastic organ moulded by our sensory experience: it never stops changing and requires stimulation throughout life to remain healthy."
Jocelyn Bosse from the Queensland Academy for Science, Mathematics & Technology competes in the 2009 Queensland Final of the Brain Bee Challenge
A fundamental principle of learning and memory is that the changes in behaviour associated with learning are directly linked to events at a molecular and cellular level.

Therefore, to fully study learning and memory requires multiple approaches in different experimental systems. Human brain imaging can tell us where learning occurs in the brain. Whole animal preparations, such as brain recordings in behaving rats or insects, inform us on electrical processes involved in the brain during learning. Recording from single neurons can further inform us on how electrical processes are coupled to cellular processes such as synaptic plasticity. To then understand synaptic plasticity, or how neurons are wired, requires cellular and molecular tools.

Using state-of-the-art brain imaging techniques, researchers at QBI are showing how brain structures such as the hippocampus are recruited to retrieve place memories in human subjects exploring virtual reality worlds. By investigating behaving subjects, the researchers are beginning to unravel the complex relationship between selective attention, skill learning, and fact learning – all intertwined aspects of learning. Similar complex questions are also being investigated in animal models such as honeybees and fruit flies. The Srinivasan Lab is exploring conceptual learning in honeybees, and the van Swinderen Lab is finding that flies pay attention and learn in much the same way as humans.

Animal models provide an effective way of proceeding from the behavioural level to the actual brain mechanisms involved in learning and memory. A crucial first step in animal studies involves defining the correct behavioural paradigms to test different learning processes relevant to human behaviour. To effectively connect molecular changes with the appropriate behaviour, the Burne Lab has developed a wide array of behavioural paradigms to carefully assay rodent learning.

Similarly, to make studies of learning more ecologically relevant, the Reinhard Lab studies honeybee learning in more natural contexts, for example by using floral scent mixtures as stimuli. By studying mechanisms of learning and memory in widely different species, QBI researchers are uncovering processes that are fundamental to learning in all animals. Memory is strange: it can last a lifetime if associated with strong emotions, but often lasts only just long enough to briefly remember a phone number. What are the different events in the brain behind these distinct processes? A major effort in studies at QBI involves relating memory with the activity of neurons. To this end, the Faber Lab is dissecting working memory by recording firing patterns in the rat prefrontal cortex and thereby finding the cellular requirements to keep a memory alive long enough to accomplish a task. The Sah Lab, focusing on a brain structure called the amygdala, is finding those long-lasting changes associated with emotions that consolidate our memories for life.

As we grow and age, our brain is continuously reorganising, yet somehow our lifetime of memories persist through these changes. The Claudianos Lab has discovered key molecules involved in re-wiring during honeybee learning, and many of these have extensive homology in humans. Using rodent models, the Bartlett Lab has uncovered reservoirs of neurons in the hippocampus capable of regeneration, suggesting a neural substrate for the persistence of our memories through life. By manipulating these systems in mammalian and insect models, QBI researchers are determining how we can reverse the neurodegenerative trends that often unravel the skills and memories we have acquired through a lifetime of experience.

Fruit flies provide a model system for investigating the mechanisms underlying attention and learning. (Oliver Evans, research assistant in the van Swinderen Lab)
Pascal Molenberghs, a postdoc in the Cunnington and Mattingley Labs, conducts an fMRI experiment on the human mirror neuron system at UQ’s Centre for Magnetic Resonance.
Researchers in cognitive neuroscience at QBI aim to understand the processes in the brain that mediate our everyday behaviour, such as the way we perceive the world around us, how we maintain or shift our focus of attention, how we make choices, and select and initiate appropriate actions. Our brains constantly receive vast amounts of information about our environment from our senses – particularly vision, touch, and hearing. This information is also constantly changing. Even the visual information our brain receives from the world changes every time we move our eyes or head. We also need to make choices to select appropriate actions and to react to events. This allows us to co-ordinate our actions with objects and with other people in our surrounding environment to achieve our desired goals.

How do we make sense of the rich and changing information coming into our brain to gain a stable perception of the world around us? How do we select and control appropriate actions within this rich sensory environment to achieve our goals? And how do we learn within this changing environment to guide our choices and behaviour in the future? QBI researchers are examining what mechanisms in the brain underlie the cognitive processes of perception, attention, and selection of action that are a fundamental part of our everyday life.

Research in the Mattingley Lab examines how mechanisms of selective attention influence vision, touch and hearing, and how information from these different senses gets integrated in the brain. Attention is the process by which we select and focus on what is relevant in our environment and prevent distraction from competing stimuli. The Mattingley Lab also examines impairments of attention that can arise following stroke. A major goal of this work is to develop more effective techniques for rehabilitation.

The Cunnington Lab focuses on the preparation and control of voluntary action, examining the brain processes that precede movement and are responsible for selecting, planning, and coordinating voluntary movements before they are initiated. Research in the Cunnington Lab also examines how we perceive and understand the actions of others, and how motor areas that are important for controlling our own actions are also involved in our ability to recognise others’ actions.

The Bellgrove Lab examines the influence of genes on cognitive processes related to attention, perception and response selection. Neurons in the brain communicate via neurotransmitters, which are largely determined by our genes. Dopamine is a neurotransmitter system in the brain that is crucial for attention, decision-making and response selection. Researchers in the Bellgrove Lab are examining how individual variations in dopamine genes, for example, may affect our ability to select and maintain attention and prevent distraction, and may account for problems with attention and inhibition of responses in children with attention deficit hyperactivity disorder (ADHD).

The van Swinderen Lab studies selective attention and memory in fruit flies. While the brain of a fruit fly is vastly different from that of humans, most of the neurotransmitter systems and their regulatory genes are very similar. By studying visual selective attention in this genetic model, researchers in the van Swinderen Lab are able to discover some of the genes and neural mechanisms that control attention and memory. This research can then provide important insights into how neural mechanisms for attention may operate in the human brain.

EEG recordings show that brain activity increases in readiness for voluntary action. (Marianna Lemonis, honours student in the Cunnington Lab)

QBI researchers are examining what mechanisms in the brain underlie the cognitive processes of perception, attention, and selection of action that are a fundamental part of our everyday life.
Neuronal fibre tracts (‘wiring’) in the mouse brain revealed by axonal tractography on magnetic resonance images obtained with a 16.4 Tesla scanner. (Nyoman Kurniawan, Centre for Magnetic Resonance, and Randal Moldrich, CJ Martin Fellow in the Richards Lab)
Our brains are constantly performing astonishing feats of computation without us being aware of it. Tasks as apparently simple as catching a ball involve vast amounts of calculation as the ball flies towards you: tracking the ball in the visual scene, predicting where it will be next, directing one’s hands to the right place at the right time, even unconsciously estimating the weight of the ball and the degree of friction offered by its surface.

We can do all this in half a second yet, even given unlimited time, modern digital computers are still incapable of solving these computational problems nearly as well as humans. In fact, in many important ways, even the brain of a humble honeybee is a far more sophisticated computing device than the largest man-made supercomputer.

How do biological nervous systems achieve such feats? It’s not that they have more powerful hardware – brain cells are slow and unreliable compared to the transistors of digital computers. Rather, millions of years of evolution have crafted brains to be extremely efficient at solving very particular types of problems. Two key examples of such problems are visual perception and spatial navigation, both of which are typically characterised by noisy, ambiguous and incomplete information.

To understand how biological brains solve such problems, QBI researchers are working at the interfaces between areas such as neuroscience, computer science, mathematics, physics, engineering and psychology. For instance, by studying the retina of rabbits, the Vaney Lab is uncovering how visual motion information is computed. Other researchers are studying how humans process visual images of faces, in order to understand how we are extract biologically important information from facial expressions.

Moving from large brains to small brains, the Srinivasan Lab is studying how honeybees solve tasks such as navigating to and from food sources. A major discovery of this work is that bees use simple algorithms based on optic flow to help them land smoothly. The researchers are now putting these principles to work in the design of autonomous flying vehicles.

QBI is also home to a large government-funded Thinking Systems project, one of only three in Australia. This involves several QBI laboratories that are taking complementary approaches to understanding the general principles by which bees, rats and humans efficiently navigate in the world. Again the researchers are turning the insights gained from this work into the design of autonomous robotic systems.

The Goodhill Lab continues the themes of vision and navigation, but now in the context of the developing brain. The researchers study how the visual system becomes wired up during early development, with particular emphasis on the role the visual environment plays in this process. The Goodhill Lab is also using a combination of experiments and computational modelling to uncover how growing nerve fibres navigate through the developing brain to make the right ‘wiring’ connections. A remarkable recent discovery from this work is that principles of optimal computation that explain some aspects of human behaviour can also explain the decisions that nerve fibres make when choosing in which direction to grow.

Through its unique combination of researchers, expertise and facilities, QBI is at the forefront of research in understanding how brains compute. This area of research is expanding both at QBI and worldwide, and it is becoming clear that a computational perspective will be a crucial component of work in almost all areas of neuroscience in the future.

"In many important ways, even the brain of a humble honeybee is a far more sophisticated computing device than the largest man-made supercomputer"
The development of nerve fibre tracts, magenta, in the mouse brain is regulated by the protein Nuclear Factor One A, green. (Ilan Gobius, PhD student in the Richards Lab)
How is the astonishingly complex architecture of the brain generated? The answers to this question lie not in the adult animal but in the developing embryo. Research carried out by QBI’s developmental neurobiologists is providing important insights to the cellular and molecular mechanisms driving the establishment of the central nervous system (CNS).

To understand the processes underpinning brain development, QBI researchers employ a range of animal models, including the mouse, the toad *Xenopus laevis*, the zebrafish, and the nematode *C. elegans*. At first glance, the nervous systems of some of these animals may seem rudimentary when compared to the human CNS. However, the fundamental developmental principles and molecular signalling pathways utilised by the common evolutionary ancestors of vertebrates and invertebrates continue to be exploited today by their descendants.

One of the major goals of the Cooper Lab is to unravel the molecular mechanisms responsible for the formation of the early nervous system. The earliest neural structure in vertebrates is the neural tube, comprising a single layer of neural stem cells surrounding a central cavity. These neural stem cells undergo countless rounds of division to reproduce themselves and to ultimately generate all neural cell types found in the adult CNS. Using both the Xenopus and zebrafish models, members of the Cooper Lab have recently identified a cell surface receptor that is essential for neural tube formation. Mutations in this receptor produce neural tube defects that closely resemble the congenital neural tube abnormalities seen in humans. The researchers have also discovered that this multi-talented receptor is important for the production of new neurons from neural stem cells throughout embryonic development and into adulthood.

Once neurons are born, they immediately sprout processes that will become axons or dendrites when mature. Intriguingly, axons and dendrites originate from opposite sides of the young neuron, indicating that the cell has an intrinsic ability to become polarised. Understanding how neurons establish and orient polarity with respect to extracellular cues is an important and challenging problem in neurobiology. The Hilliard Lab uses the nematode *C. elegans* to investigate how exposure to complex mixtures of molecular cues regulates the establishment of neuronal polarity and promotes outgrowth of neuronal processes.

How do growing axons navigate for long distances through complex environments to reach their specific targets? The Richards Lab focuses on the largest axon tract in the mammalian brain, the corpus callosum, which connects neurons in the left and right cerebral hemispheres. Using the mouse as a developmental model, the researchers have now identified several indispensable guidance molecules that steer axons from one hemisphere to the other. As defects in the development of the corpus callosum are associated with a large number of human congenital syndromes, resulting in mild to severe cognitive deficits, these studies are providing clues as to what may be going wrong during the developmental phase.

Human congenital brain abnormalities and many psychiatric syndromes are known to be the direct result of failure in developmental processes within the embryonic CNS. While pursuing their research goals, the QBI developmental neurobiologists are also indentifying key molecules that, when mutated, contribute to the abnormal development of the CNS. This knowledge is providing valuable clues that may lead to novel preventative or treatment strategies.
Untitled by Zelko Maric; acrylic on canvas; courtesy of the Queensland Centre for Mental Health Research Art Collection
THE AGEING BRAIN

Society is poised to face a health crisis as the burden of age-related brain disorders overwhelms both the monetary and human resources needed to provide adequate care for the sufferers of neurological conditions.

Researchers at QBI are working on understanding what goes wrong in the ageing brain and how we might prevent and treat these diseases in the future. By employing a variety of scientific techniques including genetics, cell culture, animal models and human studies, QBI researchers have made several breakthroughs in understanding the processes that occur in the ageing brain.

Neurodegenerative conditions, which include injuries to the nervous system such as occur in stroke, spinal cord injury and neurotrauma, as well as diseases such as Alzheimer’s, Huntington’s and motor neuron disease, are underpinned by the death of cells in the nervous system. There are several approaches taken by QBI researchers that are leading to potential treatments for these conditions. These include slowing or preventing cell death, and promoting repair and regeneration of the damaged cells.

Regenerative treatments for functional decline associated with neurodegeneration are the focus of several laboratories at QBI. The goal of the Hilliard Lab is to understand how brain cells grow and make connections during development. This research has identified molecules that prevent recovery after spinal cord injury. Drugs that overcome the actions of these molecules have been successful in allowing recovery from paraplegia in animal trials performed by the Bartlett Lab and collaborators. The best of these drugs is currently undergoing further preclinical trials.

Until quite recently, it was thought that the number of brain cells steadily declines from birth. An exciting discovery to come out of QBI is that, in some parts of the human brain, new brain cells continue to be born throughout life. How this contributes to normal brain function and the role it plays in the ageing brain is the focus of a number of laboratories at QBI. Animal models have provided us with some unique insights into the processes that control this neurogenesis.

Research from the Bartlett and Rietze Labs has found that simple voluntary daily exercise, as well as some drugs such as antidepressants, can increase the rate at which new brain cells are born in both the normal and ageing brain, leading to improved brain function.

By harnessing and promoting the brain’s innate ability to generate new neurons, the Bartlett and Cooper Labs aim to repair regions of the brain impaired by normal ageing, dementia, stroke and Huntington’s disease.

Another approach used by QBI researchers to understand disease processes is to identify cellular and genetic abnormalities that occur in people with disease. The Wallace, Coulson and Osborne Labs study cells collected from human sufferers of motor neuron disease, Alzheimer’s disease and brain tumours, comparing their cells and genes with those from healthy individuals. A recent key discovery of the Wallace Lab was the detection of two genes with mutations in people with motor neuron disease. This information is then used to develop models of disease using cell culture techniques and whole animal studies in an attempt to understand how these genetic mutations lead to disease progression.

This multidisciplinary approach taken by QBI scientists is helping us understand the ageing and damaged brain, assisting in the clinical pursuit of the treatment and prevention of neurological disorders.

“By harnessing and promoting the brain’s innate ability to generate new neurons, we aim to repair regions of the brain impaired by normal ageing, dementia, stroke and Huntington’s disease”
3 wise men by Craig Finn; acrylic on laminate and cannite table top; courtesy of the Queensland Centre for Mental Health Research Art Collection
QBI’s research is advancing our basic understanding of how the healthy brain develops and functions. Through understanding how a normal brain works, we can determine what goes wrong in the case of disease.

Using world-class facilities, QBI researchers from a variety of disciplines are working together to investigate the underlying causes of common brain disorders such as epilepsy, multiple sclerosis, schizophrenia, autism and attention deficit hyperactivity disorder (ADHD).

QBI researchers work closely with local, national and international clinicians to access patient samples. The clinical symptom details and samples provided by patients are essential to understanding what goes wrong in cases of disease. Animal models of disease are also an important part of clinical research. QBI maintains several colonies of mice and rats that develop brain disorders mimicking human conditions. These animals not only allow us to examine the disease process in detail, but are also essential to testing newly developed treatments for disease.

Genetic factors play some role in almost all brain disorders. At QBI we are assessing the role of genetic factors in various brain disorders. Research in the Wallace Lab is focusing on identifying genes for familial forms of epilepsy and has led to the recent discovery of a new gene that causes a form of epilepsy associated with twitching muscles. This research will ultimately lead to improved treatments for epilepsy.

The Bellgrove Lab is investigating genes that are associated with attention and attention deficit. ADHD is one of the most common and controversial childhood disorders and research suggests a strong genetic component. By using sophisticated behavioural and brain measures of attention, QBI researchers are trying to establish subgroups of children with ADHD with potentially distinct genetic bases.

So called ‘environmental factors’ also play a major role in brain disorders. In particular, the McGrath Group is investigating the role of nutrition and paternal age as factors influencing the development of cognitive disorders. For example, there is evidence to suggest that low prenatal vitamin D (the sunshine hormone) may alter brain development. Using mouse and rat models to explore this interesting research question, QBI scientists were the first to discover that low prenatal vitamin D changes the shape of the brain and changes the behaviour of the animal. Some of these changes involve parts of the brain that are known to be altered in people with schizophrenia.

The offspring of older fathers have an increased risk of developing disorders such as autism and schizophrenia. This is thought to be due to mutations in the developing sperm. The McGrath Group has shown in a mouse model that the offspring of older fathers have changes in brain shape and in behaviour, similar to some findings in autism.

The Faber Lab is focusing on disorders of the prefrontal cortex, the region of the brain that controls awareness and personality, and distinguishes humans from other mammals. The functioning of the prefrontal cortex is disrupted in many neurological disorders, such as schizophrenia, ADHD, dementia, depression, anxiety, bipolar disorder, post-traumatic stress disorder, stress, eating disorders and sleep disorders. The research will provide insights into how we might improve prefrontal cortex performance for sufferers of these brain disorders.

These are just a few of the advances gained from QBI’s research that will improve the lives of people suffering from brain disorders.
The Adams Lab is exploring the workings of the nervous system, focusing largely on the native receptors and ion channels that transmit pain signals from peripheral sensory nerves to the brain. The lab has a long-standing collaboration with researchers in UQ’s Institute for Molecular Bioscience to investigate the function of venom peptides (conotoxins) derived from cone snails. The complexity of the venoms reflects the smorgasbord of potential prey species in the communities of tropical and sub-tropical reefs.

**Analgesic Conotoxins**

Each of the 700 Conus species has evolved its own specialised cocktail of ~200 neuroactive peptides. These peptides target specific classes and subtypes of membrane receptors and ion channels in the prey’s nervous system, with paralysing or lethal effect. Over the past year, the lab has focused on the analgesic α-conotoxins Vc1.1 and Rg1A, which are naturally occurring peptides that are currently in development as a treatment for neuropathic pain. Recently it was proposed that the primary target of Vc1.1 and Rg1A is the α9α10 neuronal nicotinic acetylcholine receptor. Surprisingly, however, we found that Vc1.1 and Rg1A more potently inhibit the N-type Ca2+ channel currents in rat sensory ganglion neurons via a G protein-coupled receptor mechanism. To our knowledge, this is the first demonstration of α-conotoxins acting as agonists at G protein-coupled GABA<sub>B</sub> receptors modulating native CaV2.2 channels. The prevailing view in the literature until now has been that α-conotoxins primarily target nicotinic acetylcholine receptors (nAChRs), so our current findings have the potential to introduce a paradigm shift in thinking about the targets of α-conotoxins. GABA<sub>B</sub> receptors may play a critical role in pain pathways and are a clear therapeutic target for these and novel ‘designer’ conotoxins.

Structure-activity relationships obtained from mutational studies of Vc1.1 have also been carried out. Initially, all residues except the highly conserved cysteines, typical of conotoxins, were mutated to a lysine, an aspartic acid or an alanine. Secondary <sup>α</sup>H shift data were collected via NMR spectroscopy to characterise the mutants and determine the effect that the substitution had on the secondary structure of the peptide. The mutants were tested against the α9α10 nAChR and the key conserved residues were identified. From these scans, the [S4K] and [N9A] mutants were surprisingly more potent then synthetic Vc.1. Data from a second...
Postdoc Simon Nevin makes voltage-clamp recordings to test the effects of conotoxins on neurotransmitter receptors expressed in Xenopus oocytes.

Ion Channels and Neurogenesis

In a second distinct research project in the Adams Lab, we are investigating the expression and role of membrane receptors and ion channels in neurogenesis, that is, how neural precursor cells acquire function by activating specific ion channels. Neural precursor cells are normally quiescent and unresponsive to most biochemical cues. We have identified several potassium channel types expressed in adult neural precursor cells that contribute to the regulation of cell proliferation and differentiation. As neural stem cells express potassium channels before they can divide and differentiate, understanding the regulation of their expression and function may highlight ways of stimulating adult neurogenesis. This is being investigated by knocking down genes for candidate ion channels using RNA interference, and studying the effects on cell differentiation. In a related study, we have also established a medium- to high-throughput neuronal lineage commitment assay system using neural precursor cells obtained from transgenic mice, which has been implemented for quantitative evaluation of compounds that affect membrane ion transport, such as voltage-gated ion channel inhibitors.
The evidence for the importance of the continual production of new neurons in the brain of adult animals to maintaining fundamental functions such as learning and memory is becoming increasingly cogent. The goal of the Bartlett Lab is to understand how this process of adult neurogenesis is regulated and to investigate whether the molecular regulators can be employed to reverse diseases including dementia and depression, which appear to result from a decline in neurogenic activity.

**Discovering the Stem Cells in the Hippocampus**

In previous work, members of the lab had successfully isolated and characterised the precursor population in the subventricular zone (SVZ) of the lateral ventricle that gives rise to interneurons in the olfactory bulb. A similar precursor cell population was hypothesised to reside in the hippocampus, as this brain region also has continual production of neurons in adult animals, including humans. Initially, however, this assumption appeared to be incorrect, as it proved challenging to grow precursors in vitro that demonstrated the cardinal stem cell property of self-renewal.

Given reports that neurogenesis in the hippocampus is increased through synaptic stimulation associated with learning and exploration, we subsequently reasoned that such stimulation may activate dormant precursors. To mimic synaptic activity in vitro, postdoc Tara Walker added depolarising levels of potassium to the culture medium, and found an enormous increase in the number of precursors. Importantly, she also revealed that some of these precursors had stem cell properties, and that the same dormant precursor population was not present in the SVZ, since no increase in precursor number was evident there following depolarisation.

Tara and postdoc Masahiro Kameda, in collaboration with the Wallace Lab and Professor Cliff Abraham’s Lab in Otago, have since used models of epilepsy and direct stimulation of pathways that innervate the hippocampus to show that this reservoir of precursors can also be activated by synaptic input in vivo. Furthermore, Tara has discovered that this synaptic input leads to the release of molecules that are responsible for the precursor activation, and is currently identifying these molecules, which we believe may become front-line therapy for disease associated with loss of neurogenesis.

In a related line of investigation, postdoc Dhanisha Jhaveri has also shown that norepinephrine-mediated antidepressive medication directly activates a different dormant population of precursors, raising the possibility of new therapeutics to treat this debilitating condition.
Regulation of Hippocampal Stem Cells: Role in Learning and Ageing

As animals age, there is an increased incidence of decline in their ability to learn hippocampal-dependent tasks such as spatial navigation. This decline is matched by a decline in the number of neurons produced and by the number of precursors that can be detected using conventional approaches. We have shown that although there is a decline, synaptic stimulation leads to a 10-12 fold increase in the number of precursors and a concomitant increase in neuronal production. As recent evidence suggests that an increase in neurogenesis may reverse, at least in part, the learning deficiencies found in older animals, the lab is pursuing the idea that stimulation of the dormant precursors in the ageing brain may be a novel way to reverse cognitive decline in humans. In order to achieve this, the team has developed several strains of transgenic mice in which we can delete discrete populations of precursors at various times before or after the learning process to determine the precise impact of impaired neurogenesis on specific learning tasks.

Developing EphA4 Antagonists for Spinal Cord Repair

In association with colleagues at the Queensland Institute of Medical Research, Professor Andrew Boyd and Dr Mark Spanavelllo and Assoc Prof Ann Turnley and Professor Mary Galea at the University of Melbourne, the Bartlett lab has demonstrated that by blocking the activity of EphA4 following spinal cord injury, we can promote nerve cell regrowth and functional recovery. The group is currently testing a wide range of blocking reagents prepared by our corporate colleagues at CSL in a variety of animal models in preparation for human clinical trials.
In evolutionary terms, the development of such ‘cognitive control’ capacities has, in large part, paralleled the development of the frontal lobe of the human brain. It is perhaps unsurprising, then, that individual differences in cognitive control may be attributed, at least in part, to genetic variation. The importance of understanding cognitive control is reinforced by the number of disorders, both neurologic and psychiatric, in which deficits of control impair everyday functioning. The Bellgrove Lab is working towards understanding the biological bases of these higher cognitive processes in both healthy and clinical populations.

**Attention Deficit Hyperactivity Disorder (ADHD)**

ADHD is one of the most common and controversial psychiatric disorders of our time. The disorder is strongly genetic and it is likely that a number of genes add-up to increase risk for the disorder. A major focus of the Bellgrove Lab is to understand the nature of the cognitive problems experienced by children and adults with ADHD, and their relationship to genetics. One perplexing aspect of the attention deficit associated with ADHD is that some children and adults present with impairments of cognition, whereas others have behavioural disturbance without any apparent cognitive deficit. We are currently investigating why some individuals with ADHD experience problems of cognition and others do not. One possibility under study is that individuals with and without cognitive problems have a fundamentally different genetic basis. We have identified a subgroup of children with ADHD for which genetic risk predicts both cognitive deficits and the degree of symptom improvement achieved by drug treatments, such as Ritalin. Through the use of functional magnetic resonance imaging (fMRI), we also hope to understand how genetic risk influences brain function in individuals with ADHD.

**Dr Mark Bellgrove**

I completed a PhD in experimental psychology at Monash University, working on neurocognitive deficits in schizophrenia, before moving in 2002 to Trinity College Dublin, where I interfaced my skills in cognitive neuroscience with a developing interest in psychiatric genetics.

Over the next three years, I travelled around Ireland performing cognitive testing on children with ADHD and this research allowed patterns of cognitive deficit to be linked to susceptibility genes for ADHD.

I returned to Australia in 2005 under a NHMRC Howard Florey Centenary Fellowship to continue my work in attention and ADHD at the University of Melbourne. In 2007 I relocated to QBI where I am continuing to research the genetic and cognitive correlates of ADHD, as well as undertaking pharmacological challenge studies with functional MRI to reveal the chemical bases of attention in the healthy brain. I currently hold an NHMRC Career Development Award and a NARSAD Young Investigator Award.
Genetics of Cognition

Science has long had a fascination with studying how genetic variation contributes to individual differences in cognitive ability. In the Bellgrove Lab, Joe Wagner, Dana Schneider and Daniel Stjepanovic are examining how DNA variation in genes that control chemical (neurotransmitter) signalling in the brain influences a range of cognitive control processes such as the ability to selectively attend to one object in a visual scene.

By using techniques from cognitive neuroscience, such as the human electroencephalogram and functional magnetic resonance imaging, we are able to determine when (EEG) and where (fMRI) DNA variation might influence brain activity. By studying the genetics of cognition in non-clinical populations, it is hoped that novel insights might be gained regarding the genetic basis of clinical conditions marked by cognitive problems such as ADHD or schizophrenia.

Pharmacology of Cognition

The Bellgrove Lab is also working towards understanding how chemicals, or neurotransmitters, in the brain influence our ability to attend to and control behaviour. By using drugs that selectively alter chemical signalling pathways in the brain, one can begin to build models of the chemical control of cognitive function. For example, we are currently examining the influence of agents that modulate dopamine, noradrenaline and serotonin compared with a placebo, on the ability of humans to control impulsive behaviour.

Pitting one agent against the other allows us to determine which system(s) are important for impulse control.

This work may have important implications for treating disorders where the control of behaviour is problematic, such as schizophrenia, ADHD or obsessive-compulsive disorder.

From left: Ajay Panwar, Melany Christofidis, Mark Bellgrove, Daniel Stjepanovic, Dana Schneider,
The Claudianos Lab seeks to elucidate the molecular principles underlying sensory processing, learning, and postnatal brain development, using the honeybee as a model organism. The honeybee is an attractive model system for this research agenda given the bee’s relatively simple and accessible nervous system, complete genome sequence, extensive behavioural repertoire and social development.

The Claudianos Lab investigates the molecular changes that occur in the neuronal substrate both in the periphery and in higher brain centres during sensory experience. Their aim is to unravel a sub-code of molecular events associated with sensory processing.

**Synaptic Development and Brain Plasticity**

Charles Claudianos and Sunita Biswas characterised the Neurexin/Neuroligin complex in honeybees. These pre- and postsynaptic binding molecules play a central role in the development and specification of new synaptic connections. The team showed that these molecules are highly conserved between most animals including humans, in whom they have been associated with neurological disorders such as autism and schizophrenia. The team also discovered a great range of gene arrangements during synaptic development that indicate Neuroligins and Neurexins have multiple and subtly different roles that are likely to provide a code for how new nerve circuits are specified under sensory stimulation. This extensive first-of-its-kind study was recently published in *PLoS ONE*.

In collaboration with Judith Reinhard, the Claudianos Lab then investigated the functional role of Neuroligins and Neurexins in bees. Using a number of behavioural assays combined with molecular and immuno-histochemical analyses, the team discovered that the expression of these molecules is dynamically linked to sensory experience. Neuroligin/Neurexin expression was significantly down-regulated in higher brain centres when honeybees were kept in isolation, deprived of olfactory and visual input, but was up-regulated when bees were stimulated with intense learning assays. These results confirm that sensory experience stimulates synaptic development, be it in size or the number of synapses in brain centres that are involved in sensory learning and memory.

**Dr Charles Claudianos**

I completed a PhD in biochemistry and molecular biology under the supervision of John Oakeshott and Hugh Campbell at the Australian National University (ANU) in 1999, after which I was awarded an NHMRC CJ Martin Fellowship to pursue my postdoctoral training with Bob Sinden and Hans Dessens at Imperial College London (2000–2002).

On my return to Australia, I continued my work with Hugh Campbell at ANU's Research School of Biological Sciences, before joining Mandyam Srinivasan’s Visual Sciences Group in 2006. The following year I relocated to QBI where I was appointed as a Senior Research Fellow. My research interests focus on the sensory response of insects to environment. In particular, I use the honeybee and the *Drosophila* fly to examine the molecular basis of visual and olfactory perception, including how the brain responds to sensory stimulation by changing synaptic connections via key adhesive molecules such as neuroligin and neurexin.
Molecular Basis of Olfactory Adaptation

Charles also investigated the molecular basis of sensory learning by studying molecules in the periphery of the central nervous system: olfactory receptor proteins (ORs). ORs are transmembrane proteins located on olfactory sensory neurons and are responsible for binding odorants, which triggers a signalling cascade resulting in action potentials being sent to higher-order brain centres for further processing. Honeybees have approximately 160 ORs. Using a calibrated molecular clock analysis, Charles and his colleagues have shown that ORs in honeybees have only recently expanded over the last 50 million years, during the same time as the emergence of flowering plants.

In collaboration with Judith Reinhard, the team then studied expression patterns of ORs and found that the OR expression varied significantly depending on geographic location, time of year, and bee age. This indicated that, contrary to common assumption, OR expression patterns are highly plastic and responsive to sensory experience. This hypothesis was confirmed in a controlled experiment, where bees experienced a specific floral scent and the pattern of OR gene expression changed after this odorant exposure. These ORs were biochemically specific for the floral odorant and the number of OR molecules on olfactory neurons changed even after a few hours of specific scent experience. This molecular sensory adaptation is thought to be a general paradigm that would also occur in humans.

Charles received a prestigious award from the Royal Entomological Society for the ‘Best Paper’ published during the period 2006-2007 (Insect Molecular Biology 2006 15: 615-636). The study was part of the Honeybee Genome Project (Nature 2006 443: 931-949) to which Charles contributed as a theme leader.

From left: Charles Claudianos, Julie Lim, Melanie Havler, Sunita Biswas, Judith Reinhard, Michael Sinclair, Peter Carlyle
Cell and axon migration are fundamental processes essential for establishing the architectural plan of the central nervous system during vertebrate embryogenesis. Newly born neurons migrate along predefined pathways to establish the variety of distinct structures present in the adult brain. In addition, young neurons must also extend nascent axons to their appropriate targets in order to establish the extensive network of connections found between neurons in the adult brain.

Research in the Cooper Lab focuses on key molecular guidance systems driving both axon pathfinding and neuron migration during embryonic development. Our team has now identified several important guidance receptors (Neogenin and Ryk) that govern the migration of important neuronal and axonal populations in the embryonic brain.

Neogenin and Cortical Development

The intricate neural architecture of the six-layered mammalian neocortex is dependent on the accurate and efficient migration of young cortical neurons during embryogenesis. In humans, mutations in genes controlling this process have severe consequences for cortical development, leading to intractable epilepsy, mental retardation and other common disorders such as schizophrenia, dyslexia and autism. PhD student Stacey Cole, together with Amanda White, is investigating the hypothesis that one cell surface molecule, Neogenin, guides new interneurons through the embryonic brain into the early cortex. Their analysis of embryos from mouse strains carrying a loss-of-function mutation in the Neogenin gene has revealed that loss of this receptor results in marked perturbations in cortical development. They are now beginning to unravel how Neogenin regulates neuronal migration at both the molecular and cellular level.

Neogenin and Adult Neurogenesis

Neogenin is present in the adult forebrain in regions where adult neural stem cells reside. It is also found along the rostral migratory stream, a pathway followed by newly born adult neurons as they travel from their place of birth to their final destination in the olfactory bulb. PhD student Dana Bradford has been investigating the possibility that Neogenin regulates neurogenesis and migration in the adult mouse brain. She has discovered that this receptor is present on adult neural stem cells within the forebrain and on maturing neurons as they navigate along the migratory stream. In collaboration with Richard Faull and colleagues at the University of...
Auckland, Dana has also demonstrated that Neogenin is present on neural stem cells and migrating neurons within the adult human brain. This is an exciting result because it suggests that it may be possible to harness the Neogenin signalling pathway for promoting the migration of new neurons to regions of damage or disease in the human brain.

Ryk and Cortical Development

The corpus callosum is the major interhemispheric commissure in the human brain, comprising approximately 3 million myelinated fibres that connect one side of the cortex to the other. More than 50 different human congenital syndromes, often associated with mental retardation and epilepsy, have been described in which cortical axons fail to cross the midline of the forebrain during late embryonic development. The Cooper Lab has recently identified a guidance receptor, Ryk, that acts on the contralateral side of the midline (on the opposite side of the midline from where the axons originate) to promote callosal axon escape from the midline into the adjacent hemisphere. PhD student Charlotte Deverson has continued to explore the function of Ryk during cortical development and has now discovered that Ryk has multiple roles in corpus callosum development.

In addition to its axon guidance role, Charlotte has shown that it is also important in promoting the growth of these axons. This project may identify molecular targets that can be manipulated to encourage axon regrowth and correct pathfinding in the adult human brain and spinal cord damaged as a result of disease or injury.

Cultured cortical neurons from a developing mouse brain, labelled to show the nucleus, blue, the actin cytoskeleton, red, and an intracellular signalling molecule GSK3-beta, green. (Charlotte Clark, PhD student in the Cooper Lab)
Alzheimer’s is the most common form of dementia in the western world. However, as definitive diagnosis is only available upon autopsy, the disease’s aetiology is still not well understood. Despite the widely held belief that accumulation of a naturally produced peptide known as amyloid-beta (Aβ) is the proximal cause of Alzheimer’s disease, how this results in brain-area-specific degeneration and the manifestation of clinical symptoms of cognitive decline, particularly memory loss, remains highly debated.

One of the persisting hypotheses of the mechanisms underpinning the memory decline in Alzheimer’s disease involves the degeneration of basal forebrain cholinergic neurons (cBF neurons), which are critical for learning, memory and attention. The susceptible cBF neurons innervate brain regions such as the cortex and hippocampus, where high Aβ load is found post-mortem. In addition, the only classes of drugs with clear efficacy in clinical trials are those targeting the cholinergic system, although these drugs are only beneficial for as long as the cBF neurons remain alive. Therefore, Aβ-induced cBF neuronal degeneration is likely to underpin the cognitive decline observed in Alzheimer’s disease.

Neuronal loss in neurodegenerative disease often results from activation of specific cell death pathways triggered either by a toxic agent, such as Aβ, or indirectly through secondary disease processes. One pathway activated in a wide range of neurodegenerative conditions (including motor neuron disease, stroke and spinal cord injury) is that mediated by a cell death-inducing receptor called p75. p75 is expressed in the developing nervous system, acting to prune excess and misconnected neurons in order to produce functional brain circuits. In the adult, p75 is down-regulated in most brain regions. There are, however, some cell populations that continue to express p75 throughout adult life, including the cBF neurons, which may make them particularly susceptible to neurodegeneration.

Neurodegenerative diseases are underpinned by the dysfunction and death of particular neurons. The Coulson Lab investigates the processes by which neurons make the decision to die or stay alive, with a current focus on the mechanism of neuronal death in Alzheimer’s disease.

I obtained my science degree from the University of Melbourne, majoring in genetics and biochemistry. I then undertook a PhD with Colin Masters in the Department of Pathology, studying the normal function of the amyloid β protein precursor of Alzheimer’s disease.

Following a year at the ZMBH, University of Heidelberg, Germany, I pursued postdoctoral work at the Walter and Eliza Hall Institute with Perry Bartlett, studying the neuronal cell death that is mediated by the cell death receptor p75, both in normal development of the nervous system and in neurodegeneration.

In 2003 I was recruited to the University of Queensland as a founding member of QBI, which allowed me to establish an independent laboratory. Although the main focus of my work remains on the molecular and cellular mechanisms by which neurons make life and death decisions, I am also investigating the role of p75 in neurogenesis and its behavioural outcomes.
Areechun Sotthibundu, a recipient of Thailand’s Mahidol University Science and Technology Talent PhD Scholarship, found that Aβ-induced cell death was almost completely abolished in cultured neurons unable to make p75. Furthermore, loss of p75 expression attenuated any Aβ-induced loss of cBF neurons observed in animals following application of Aβ into the hippocampus. Areechun’s results were the first to definitively show a principal role for p75 in Aβ-induced death of cBF neurons. However, the precise mechanism by which Aβ results in dysfunction is an area of ongoing research by other members of the lab. PhD student Linda May is investigating how stimulation and inhibition of neuronal activity affects p75-mediated death signalling induced by Aβ, and postdoc Alex Sykes, recently awarded a grant from Alzheimer’s Australia Research, is biochemically characterising how p75 death signalling is activated, with the aim of developing inhibitors of the process.

The Coulson Lab is also investigating whether cBF neuron degeneration is responsible for the cognitive and behavioural changes that characterise the clinical presentation of Alzheimer’s patients. Adam Hamlin, the recipient of an NHMRC Training Fellowship, joined the lab in 2008 to drive forward this area of research. He will also assist in the lab’s pursuits to understand the normal role of p75 in the cBF neurons, an area of research arising from the discovery that mice unable to make p75 in their cBF neurons have subtle changes to their behaviour.
Research in the Cunnington Lab focuses on the brain processes underlying the preparation for action and the perception of others’ actions in the human brain. Whenever we plan, imagine, or observe others performing actions, representations of those actions are encoded in the neural activity of motor areas of the brain. Researchers in the lab use brain imaging methods to examine how we form these plans for action before initiating voluntary movement and how we perceive and understand the actions of others.

**Preparation for Voluntary Action**

Activity in premotor and supplementary motor areas of the brain begins up to two seconds before the initiation of voluntary action. This activity is generally thought to reflect the planning and preparation for movement and is known as the ‘readiness potential’. A study by Marta Bortoletto is examining the different cognitive and motor processes that precede the initiation of voluntary movement. Using combined fMRI brain imaging and EEG event-related potentials, Marta has found that different parts of the brain co-ordinate the timing of movement initiation and the sequencing of sub-movements within complex actions. Voluntary decisions on when to initiate actions are mediated by prefrontal brain areas and occur over the earliest part of the readiness potential, whereas the specific programming of actions to be performed involve premotor and parietal brain regions and occur immediately before the initiation of movement. This research is beginning to uncover the various neural processes that must be co-ordinated before the initiation of voluntary action.

**Perception of Actions**

Research in the Cunnington Lab also focuses on the human ‘mirror neuron’ system, thought to underlie the perception of others’ actions. The mirror system has become an important topic in cognitive neuroscience because of its speculated role in human abilities crucial to our social life, such as empathy, understanding others’ intentions, and social development in children. These all stem from our ability to perceive and understand the actions of others.

A study by Pascal Molenberghs is examining brain activity associated with observing and imitating the actions of the others. This work has shown that those parts of the inferior parietal cortex and the dorsal premotor cortex that are important for planning and performing action are also involved when observing the same actions performed by others. This highlights the common role of motor areas of the brain in both the execution of movement and the perception of others’ actions.

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**Dr Ross Cunnington**

After growing up in rural Victoria, I completed my BSc and PhD at Monash University, Melbourne. My research examined the brain processes involved in the preparation for voluntary action in people with Parkinson’s disease. I then moved to Vienna to complete a postdoctoral fellowship with Lueder Deecke in the Department of Neurology and Institute for Medical Physics (MRI Centre), University of Vienna, where I learnt the art of brain imaging with functional MRI.

After another two years as a research associate at the Peter Mansfield Magnetic Resonance Centre and the School of Psychology, University of Nottingham, I returned to Australia as an NHMRC RD Wright Fellow and established my cognitive neuroscience group at the Howard Florey Institute, Melbourne. In 2007 I joined QBI, where my research now focuses on the brain processes underlying action and attention in the human brain.
Parkinson's Disease and Perception of Actions

In collaboration with Peter Silburn and David Copland of the UQ Centre of Clinical Research Excellence, researchers in the Cunnington Lab are examining the neural processes associated with the perception of actions in people with Parkinson’s disease. This condition involves impaired output of the basal ganglia to the higher motor areas of the brain and results in severe deficits in the initiation and performance of voluntary movements. These same brain areas are also important for the perception of others’ actions. This work is finding that parts of the inferior parietal cortex, known to contain mirror neurons, are impaired in people with Parkinson’s as they observe and imitate actions of others. This research is important for understanding wider cognitive and motor deficits that might be associated with impairment of motor areas of the brain in people with Parkinson’s.

Mind-Reading and Brain-Computer Interfaces

Finally, new projects in the Cunnington Lab are aiming to decode the brain activity measured during the preparation for action, as a kind of mind-reading, in order to predict the actions people are about to make. This technology forms the basis of brain-computer interfaces, which could be used in the future to control devices such as computers, robots, or prosthetic limbs.
The prefrontal cortex, which is the newest part of our brains in evolutionary terms, sets humans and primates apart from other mammals. It is the evolution of this part of the brain that makes humans the intelligent beings we are, allowing us to conceptualise ideas, be imaginative and creative, modify our emotions, and make future plans.

One particular function of the prefrontal cortex is the process called working memory. This is the ability to keep a piece of information in mind for about 15 seconds, and then to use it, for example, to guide behaviour, form a sentence, or play a musical instrument.

The functioning of the prefrontal cortex is disrupted in many neurological disorders, such as schizophrenia, attention deficit hyperactivity disorder, dementia, depression, anxiety, bipolar disorder, post-traumatic stress disorder, stress, eating disorders and sleep disorders. By understanding the way in which the prefrontal cortex operates in a healthy brain, we can gain insights into how we might modulate activity to improve prefrontal cortex performance for sufferers of these neurological disorders.

The Faber Lab studies the medial prefrontal cortex in the rat. Although the rat’s prefrontal cortex is substantially smaller than that of a primate, it is thought to operate in a similar way during working memory tasks and in controlling emotion. Therefore it provides a good model for studying how the prefrontal cortex functions. The Faber Lab makes electrical recordings from single neurons in vitro, with a technique known as patch clamping.

The way that information is kept in mind during working memory is by the repetitive firing of action potentials by neurons in the prefrontal cortex. This repetitive firing is believed to be mediated by both the properties of individual neurons and the activation of connections between neurons.

Connections between Neurons
Louise (Luli) Faber’s project focuses on the connections between neurons. This involves looking at the processes of synaptic transmission and synaptic plasticity. Synaptic transmission is the way in which neurons communicate with each other, while synaptic plasticity is thought to be the process that

Dr Louise Faber

I was born in London and grew up in England. I obtained my BSc in pharmacology from the University of Edinburgh in 1994 and my PhD in neuropharmacology from the University of Bristol in 1998. In 1999 I moved to the John Curtin School of Medical Research at the Australian National University, where I studied a region of the brain known as the amygdala with Pankaj Sah.

This postdoctoral work continued in the Sah Laboratory after the team moved to Brisbane in 2003 to join QBI, culminating in the establishment of my independent laboratory in 2007. My work now focuses on the cellular physiology of the medial prefrontal cortex, a region that is essential for executive functions and cognitive processing, and one that is known to malfunction in neurological disorders such as schizophrenia and attention deficit disorder.
Postdoc Matthew Ireland makes patch-clamp recordings from brain slices containing the prefrontal cortex. Luli has been focusing on the role of one particular ion channel called the SK channel. Her experiments have shown that these channels play an important role in dampening down communication between neurons: SK channels reduce synaptic transmission and act as a brake on synaptic plasticity during bursts of activity.

Properties of Single Neurons

Matthew Ireland's project focuses on the inherent, or intrinsic, properties of neurons within the prefrontal cortex. These neurons show a tendency to be in either an active state or a quiet state, known as up and down states, respectively. It is during up states that neurons fire repetitively, so the mechanisms underlying this process are likely to be relevant to working memory. Matthew is aiming to find out which ion channels mediate the transitions between up and down states, and how these are modulated by neurotransmitters. Since working memory is impaired in a number of neurological disorders, such as schizophrenia and attention deficit hyperactivity disorder, understanding how to modulate the firing or the transitions between up and down states is likely to offer new ways to modulate working memory performance.
I did an honours degree in mathematics and physics at Bristol University, followed by a MSc in artificial intelligence at Edinburgh University and a PhD in cognitive science at Sussex University.

Following postdoctoral work with David Willshaw at Edinburgh University, I moved to the USA for further postdoctoral study, working with Terry Sejnowski at the Salk Institute in San Diego. I formed my own lab at Georgetown University Medical Center in 1996 and was awarded tenure in the Department of Neuroscience in 2001. In 2005 I moved to the University of Queensland, holding a joint appointment between QBI and the School of Mathematics and Physics.

My principal research interests are in the computational mechanisms underlying the development of neuronal connections; I have been a pioneer in introducing mathematical and computational analyses to two areas of developmental neuroscience, visual map development and axon guidance.
theoretical modelling, PhD student Clare Giacomantonio has reproduced recent experimental data regarding how the shadows of blood vessels on the retina affect maps in the visual cortex. However, the model suggests a more subtle explanation for some of the observed effects than was apparent from the original data. In a related project, PhD student Jonathan Hunt has investigated the extent to which certain statistical regularities of real-world visual images leave their signature in the structure of the visual cortex. Surprisingly, he has found that there is a much more complex link between input statistics and cortical structure than was initially expected. This raises many new questions regarding how visual cortical structure actually develops. PhD student Hugh Simpson is combining insights from theoretical work in both axon guidance and visual map formation to develop a new understanding of map development in the optic tectum, and is also directly testing these theoretical ideas using time-lapse imaging of map development.

Future Directions
Looking ahead, the Goodhill Lab sees many new opportunities for the synthesis of theoretical and experimental insight into the development of neuronal wiring. Grants to the lab from the ARC, NHMRC and HFSP are now supporting new collaborations with experimental neuroscientists in Europe and Japan, as well as elsewhere in Australia. Refinements in experimental technologies constantly inspire new avenues for exposing the key principles by which the nervous system develops. Ultimately, understanding development means understanding how we become the people we are.
The brain is a highly complex organ which, together with the rest of the nervous system, contains more than 100 billion neurons, forming a network that underlies the large variety of human sensitivities and behaviours. In both humans and other animals, correct development is absolutely essential for proper functioning of the nervous system.

Determining how individual neurons develop is crucial for understanding how more complex neuronal structures are formed. A newborn neuron goes through many different steps during its development. It needs firstly to migrate to the right position, then establish an axis of polarisation, extend an axon and dendrites, guide these long processes to the right targets, and finally maintain its structure and organisation over time.

Research in the Hilliard Lab is focused on understanding how neuronal polarity is established and how the axonal structure is preserved over time. Our experimental model is the small free-living nematode worm, Caenorhabditis elegans. There is an extensive set of genetic and molecular biological tools available to study this organism, which has a simple nervous system made of only 302 individually characterised neurons. C. elegans therefore provides an excellent model system for addressing neurodevelopmental questions.

Neuronal Polarity

Neurons are highly polarised cells with distinct functional domains such as axons and dendrites. The polarity of a developing neuron determines the precise exit points of its processes as well as their initial trajectory of outgrowth. Understanding how neurons establish and orient polarity with respect to extracellular cues is a crucial and challenging problem in neurobiology. We have discovered that specific molecules (Wnt ligands and Frizzled receptors), conserved from lower invertebrates right through to higher vertebrates, regulate the anterior-posterior nerve growth by determining the initial polarity of the developing neuron. In mutant animals lacking the Wnt ligand LIN-44 or the Frizzled receptor LIN-17, the growth of specific nerves occurs in the opposite direction compared to wild-type animals. We also found that a precise asymmetric localisation of the receptor molecule on the neuronal membrane is essential for the generation of correct anterior-posterior polarity and nerve growth.
We are now exploring if similar mechanisms are in place for a different class of sensory neurons, or if other molecules regulate neuronal polarity and dendrite outgrowth in these cells.

**Axonal Degeneration and Regeneration**

The processes protruding from neuronal cells can extend extraordinary long distances, in some cases reaching almost the full length of an animal's body. As such, mechanisms are in place to maintain the structural integrity of these long axons over the lifetime of the animal. However, axonal degeneration does occur in some conditions and it is a critical and common feature of many peripheral neuropathies, neurodegenerative diseases and nerve injuries. The genetic factors and the cellular mechanisms that prevent axonal degeneration under normal conditions, and that trigger it under pathological conditions, are still largely unknown. We are using C. elegans genetics to identify the molecules and the mechanisms that control these processes.

Using a forward genetic screen, we have identified mutant animals in which the axons of a subset of mechanosensory neurons spontaneously degenerate as the animal age. We are now using mapping and cloning techniques to identify the mutated genes responsible for this degenerative condition.

A complementary and crucial question in neurobiology is how some axons can regenerate following nerve damage while others cannot. The answer will be of great value for the treatment of many neurodegenerative diseases and of traumatic nerve injuries. The molecules and the mechanisms underlying this important biological process remain largely uncharacterised. In C. elegans, a new laser-based technology allows single neuron axotomy in living animals, and axonal regeneration can now be visualised in real-time and tackled with a genetic approach. In our lab, we are using this approach to identify the mechanisms behind axonal regeneration.

*From left: Elia Di Schiavi, Brent Neumann, Massimo Hilliard, Leonie Kirszenblat, Divya Pattabiraman*
The major research interest in the Lynch Lab is the molecular structure and function of the structurally related glycine- and GABA,-receptors, both of which are chloride channels that mediate inhibitory neurotransmission in the central nervous system. The Lynch Lab investigates these receptors using a variety of experimental approaches, including molecular biology, protein chemistry, manual and automated patch-clamp electrophysiology, voltage-clamp fluorescence, and automated high-throughput fluorescence-based imaging.

The GABA,- receptor (GABA,R) is an important therapeutic target for neuro-active drugs such as sedatives and anaesthetics, whereas the glycine receptor (Gly,R) has recently emerged as a drug target for inflammatory pain and spasticity. The lab investigates the structure of these receptors, the mechanisms by which they open and close, and their molecular pharmacology. The lab is also interested in identifying novel compounds that are active at these receptors, both as leads for therapeutic development and as pharmacological tools for basic research.

Glycine Receptor Structure and Function

Both types of inhibitory channels belong to a large family of structurally related receptors termed the Cys-loop receptor family. Similar structure implies similar operation, so any findings made about the molecular operation of GlyRs apply to all related receptors, many of which are prime targets for therapeutic drugs. A technique called voltage-clamp fluorometry was developed by PhD student Stephan Pless (now graduated) to understand how GlyRs open and close. It involves inserting a cysteine into a receptor domain of interest and specifically labelling this with the fluorescent label, rhodamine. By simultaneously measuring channel current and fluorescence changes, conformational changes in multiple domains can be monitored in real time.

So far, the Lynch lab has made two important discoveries that dramatically revise our understanding of how GlyRs open and close. Furthermore, Ph.D student Qian Wang has recently adapted this technique to studying GABA,R.

The lab is also engaged in identifying the GlyR binding sites for molecules of potential therapeutic interest. Recently, important insights have been made into the binding mechanisms of several drugs including dihydropyridines, cannabinoids and tropones. Defining such sites is an important first step in designing novel therapeutics. However, it is often difficult to identify drug-binding sites by...
PhD student Han Lu fabricates glass micropipettes for patch-clamp recordings

conventional strategies. Postdoctoral fellows Timothy Webb and Daniel Gilbert, together with PhD student Timothy Lynagh, have established a project employing high-throughput methods to automate the discovery and characterisation of novel drug-binding sites. This has already provided important insights into the binding mechanisms of the anti-parasitic agent, ivermectin.

Drug Discovery

The Lynch Lab is also interested in discovering novel drugs with high potency and specificity for GlyRs and GABA\(\text{A}\)Rs. This mainly involves screening natural product fractions supplied by Rob Capon from UQ’s Institute for Molecular Bioscience. Initial screening is performed on a fluorescence-based cell-screening robot constructed in the lab. Compounds of interest identified in this way are then tested at higher precision by automated patch-clamp electrophysiology.

The GlyR drug-discovery project is well advanced and has identified several compounds of possible therapeutic interest, as well as a range of other compounds that are potentially of use as pharmacological probes for basic neuroscience research. Many of these compounds belong to structural classes previously unknown to science. The lab has also commenced screening GABA\(\text{A}\)Rs, which are much more important clinical targets. Due to the large variability in the GABA\(\text{A}\)R subunit composition at brain synapses, it is particularly important to develop subtype-specific pharmacological probes for these receptors, and this is one of the major goals of this program.
The Mattingley Lab aims to understand the neural processes that underlie human perception, attention and motor behaviour. The lab’s researchers use cutting-edge brain imaging and brain stimulation techniques, including functional magnetic resonance imaging (fMRI), electroencephalography (EEG) and transcranial magnetic stimulation (TMS). Although much of the work involves normal healthy volunteers, a substantial portion of the research effort is directed toward understanding neuropsychological impairments that arise from cerebral stroke, dementia and neurodevelopmental conditions such as ADHD.

Sorting the Wheat from the Chaff
Our sensory world is a confusion of information. How does the brain select just those sensory inputs that are currently relevant for guiding behaviour and filter out the rest? The Mattingley Lab is examining this question by focusing on how mechanisms of selective attention influence vision, touch and hearing. Studies by Jason Mattingley and postdoctoral fellow Marc Kamke have employed TMS to reversibly activate areas of the brain thought to control voluntary attention. The aim of this work is to discover why people have difficulty perceiving distinct visual objects that are presented in rapid succession (“attentional blink”). Using fMRI imaging, Jason and his colleagues have discovered that activity in visual-processing areas of the brain is dramatically reduced during the attentional blink. This work has overturned the textbook view that sensory areas of the cerebral cortex are driven passively by stimulation of the peripheral sense organs and instead demonstrates that activity within these cortical regions is strongly modulated by voluntary attentional processes.

Neglecting One Half of the World
Attentional processes can be catastrophically affected by cerebral stroke and, in some cases, patients show a syndrome called ‘spatial neglect’, in which one half of the sensory world disappears from awareness. Postdoctoral fellow Ranmalee Eramudugolla has tested the efficacy of a new technique for treating attention deficits after stroke. By having patients adapt to optical wedge prisms, many of the visual and auditory impairments that characterise neglect can be reduced or eliminated. The challenge ahead is to understand the brain processes that underpin these dramatic therapeutic effects.

Knowing ‘Where’ and Knowing How to Get There
Most people take for granted their ability to navigate the environment successfully. However this ability belies the enormous computational complexity of storing and recalling spatial information in the natural world. The Mattingley Lab is investigating the brain processes responsible for spatial memory and navigation, as part of a larger
Thinking Systems project that includes other researchers at QBI. One crucial aspect of successful navigation involves encoding of distinctive landmarks, such as signs and buildings. Postdoctoral fellow Oliver Baumann and PhD student Edgar Chan have conducted behavioural and fMRI studies of navigation in healthy humans using computer-generated ‘virtual’ environments. Their work has shown that not all landmarks are treated with equal importance, and that distinct brain networks are directly involved in creating and retrieving accurate ‘mental maps’ of the visual world.

A Mirror on the World

Although most human communication involves language, a significant portion of our thoughts and feelings are conveyed non-verbally, e.g. by hand gestures or facial expressions. But how are such non-verbal cues decoded in the brain? Research carried out by Jason Mattingley in collaboration with Ross Cunnington and postdoctoral fellow Pascal Molenberghs has begun to shed light on the brain processes that underlie such uniquely human abilities as imitation. This work has revealed for the first time in humans the existence of ‘mirror neurons’ within a region of the parietal lobe. Such brain cells encode specific hand gestures and respond during passive observation of the identical actions; they are thought to underlie humans’ ability to understand the intentions of others. A challenge for the future is to determine whether dysfunction within the mirror system might underlie the problems of social communication that are common in neurodevelopmental conditions such as autism spectrum disorder.
In 1996 I completed my PhD in neurobiology at the CNRS in Gif-sur-Yvette, under the supervision of Jordi Molgo, before crossing the Channel as the recipient of a European Community Postdoctoral Training Fellowship to work with Oliver Dolly at Imperial College London on motor nerve terminal sprouting and neuroexocytosis. In 1999 I moved to Giampietro Schiavo’s laboratory at Cancer-Research UK to continue this work and, in 2002, accepted a visiting position at the MRC Cambridge.

I emigrated to Australia to take up my first independent academic position in the School of Biomedical Sciences, the University of Queensland, and I was promoted to Associate Professor in 2007.

I moved my laboratory to QBI and was appointed an NHMRC Senior Research Fellow in 2008. My research interests centre on the mechanisms underpinning neurotransmission, with a particular focus on exocytosis and endocytosis.

Phosphoinositide Effectors for Neuroexocytosis

The Meunier Lab recently developed a novel phosphoinositide (PI) pull-down strategy coupled with the power of tandem mass spectrometry that allows us to identify selective PI-binding proteins from highly purified subcellular fractions, such as secretory granules. Using this method, we have identified five PtdIns(4,5)P2 effectors from large dense-core vesicles, including synaptotagmin 1 and 7. We have further demonstrated by mutational analysis that the synaptotagmin7 binding site for PtdIns(4,5)P2 is critical for neuroexocytosis. Synaptotagmin7 is therefore an important effector for exocytosis of large dense-core vesicles in neurosecretory cells – a finding that has now been confirmed by others.

This new method has the potential to identify other phosphoinositide effectors in a variety of organelles and cellular systems.

The presynaptic nerve terminal is filled with synaptic vesicles that contain a neurotransmitter chemical. Upon arrival of a nerve action potential, vesicles synchronously fuse with the plasma membrane, thereby releasing the neurotransmitter and conveying the signal to the postsynaptic neuron. The research focus of the Meunier Lab is on deciphering the dynamics of the molecular events underpinning the release of neurotransmitter from vesicles located in neurosecretory cells.

Neuro-exocytosis relies on a series of protein-protein and protein-lipid interactions leading to the fusion of secretory vesicles with the plasma membrane. The Meunier Lab has accumulated evidence for a critical role played by several lipids at distinct steps of the membrane-fusion process.

PI3-Kinase C2a Controls Vesicle Priming

The Meunier Lab recently discovered that, in neurosecretory cells, the lipid PtdIns3P is produced in an activity-dependent manner on a subpopulation of secretory vesicles by the lipid kinase, PI3 kinase-C2a. The PtdIns3P produced plays a critical role in priming secretory granules, which is the process whereby the vesicles acquire the ability to fuse with the plasma membrane.

PIKfyve Controls Vesicle Exocytosis

For more than 20 years, research has focused on the positive regulation of exocytosis by the lipid PtdIns(4,5)P2. The lab recently discovered that PtdIns3P can be used as a substrate by the lipid kinase, PIKfyve, to produce PtdIns(3,5)P2 on secretory vesicles. We found that PIKfyve is a negative regulator of neuroexocytosis.
**Ca\(^{2+}\) Channels Mediating Vesicle Exocytosis**

The mechanism of vesicle fusion is highly regulated and is initiated by an influx of Ca\(^{2+}\) through Ca\(^{2+}\) channels. Following the purification of a novel stimulatory neurotoxin called glycero-toxin, which specifically up-regulates Cav2.2 (N-type) Ca\(^{2+}\) channels, the lab demonstrated that both Cav2.1 (P/Q-type) and Cav2.2 channels contribute equally to glutamate release in rat brain synaptosomes. We are now in the process of using this neurotoxin to dissect the molecular mechanism of bulk endocytosis, a process critical in maintaining a pool of synaptic vesicles during neuronal stimulation.

**Ciguatoxin-Evoked Neurosecretion**

Ciguatoxin, from the dinoflagellate *Gambierdiscus toxicus*, is responsible for a disease called ciguatera. An acute gastroenteritis occurs after consumption of intoxicated fish, followed by neurological disturbances, musculoskeletal symptoms, and paradoxical dysaesthesia. Ciguatera is considered one of the most widespread seafood-related diseases affecting humans, with at least 50,000 cases annually. Pacific ciguatoxin-1B (P-CTX-1B) activates voltage-sensitive Na\(^{+}\) channels and promotes an increase in neurotransmitter release believed to underpin the symptoms associated with ciguatera. However, the mechanism through which slow Na\(^{+}\) influx promotes neurosecretion was not fully understood. We have used chromaffin cells as a model to reconstitute the sequence of events responsible for ciguatoxin-evoked neurosecretion. This revealed that P-CTX-1B induces a slow rise in intracellular Na\(^{+}\), closely followed by an increase in cytosolic Ca\(^{2+}\) responsible for promoting SNARE-dependent catecholamine secretion. In an attempt to find a cure for ciguatera, we discovered that brevenal and beta-naphtoyl-brevetoxin prevent P-CTX-1B secretagogue activity without affecting nicotine- or barium-induced catecholamine secretion. Brevenal is therefore a potent inhibitor of ciguatoxin-induced neurotoxic effect and a potential treatment for the condition.
Schizophrenia is a poorly understood group of disorders that contribute substantially to the global burden of disease. Schizophrenia affects about one in a hundred people, tends to be more common in men than women, and usually emerges in the second or third decades of life. The symptoms of schizophrenia include hallucinations (e.g., hearing voices), delusions (e.g., false beliefs, often of a persecutory nature), impaired communication, and poor planning and motivation.

Despite advances in medications and better community mental health services, a sizeable proportion of people with schizophrenia have recurrent or persistent symptoms. There is, therefore, an urgent need to find better treatments for people with this disorder, and to understand its underlying neurobiology. In particular, there is a pressing need to evaluate known risk factors for schizophrenia, which may eventually lead to preventive strategies.

Three QBI Faculty members, John McGrath, Darryl Eyles and Tom Burne, work closely in a multidisciplinary team to help unlock the mysteries of the ‘disordered’ brain. Each member brings special skills to the team, which is part of the Queensland Centre for Mental Health Research (a body funded by Queensland Health). John McGrath, an epidemiologist with a particular interest in looking for the causes of schizophrenia, is a psychiatrist with clinical duties at the Park Centre for Mental Health. Darryl Eyles is a neuroscientist with experience in developing animal models related to schizophrenia; he is looking at structural, neurochemical and neuroanatomical measures in order to understand how disruptions during early life (e.g., changes in prenatal nutrition) can impact on the trajectory of brain development. Tom Burne is an experienced behavioural neuroscientist; by studying the behaviour of rat or mouse models related to schizophrenia, he can explore brain mechanisms known to be disrupted in such disorders. Overall, they have an international reputation for developing innovative animal models of schizophrenia. In particular, the group has built a collaborative research platform for translational epidemiology – one that encourages cross-fertilisation between schizophrenia epidemiology and neuroscience.

**Does Low Prenatal Vitamin D Alter Brain Development?**

Many studies have shown that those born in winter and spring have a significantly increased risk of developing schizophrenia, and that those born at higher latitudes are also at increased risk. Given that vitamin D levels in the population fluctuate across the seasons...
and decrease with latitude, low prenatal vitamin D “fits” these key environmental features and is therefore a plausible candidate risk factor for schizophrenia. Based on these clues, QBI researchers have developed rat and mouse models that examine the impact of low prenatal vitamin D on adult brain function. This model reproduces several core features observed in patients with schizophrenia, such as changes in brain shape and altered dopamine chemical signalling in the brain. In recent years, considerable progress has been made in unravelling the mechanisms of action, linking developmental vitamin D deficiency and brain development. This has involved a range of laboratory skills crossing many categories of observation: molecular biology, molecular pharmacology, brain architecture and cell density, behavioural neuroscience, and developmental biology including the timing and regional patterns of gene and protein expression. Several PhD students have made important contributions to this research. Louise Harvey, who recently completed her PhD and is now working at McGill University in Canada, found the first evidence that low prenatal vitamin D permanently alters adult immune function in a rat model – this could have important implications for disorders such as multiple sclerosis, which is also linked to low prenatal vitamin D. James Kesby, who was awarded the inaugural Sunshine Coast ARAFMI Scholarship, has been exploring how low prenatal vitamin D alters dopamine neurotransmission in a rat model (dopamine is linked to the symptoms of schizophrenia). Finally, Lauren Harms has developed a mouse model of developmental vitamin D deficiency and has discovered important differences in this species compared to the rat.

continued next page...
I am a Brisbane local and obtained my PhD in the neuropharmacology and neurotoxicology of antipsychotic drugs, working with Susan Pond in the Department of Medicine at the University of Queensland. My postdoctoral years between 1995 and 1997 were spent with Neil Castagnoli at Virginia Tech, and with Catriona Mytilineou at Mt Sinai, New York, examining how antipsychotic drugs induce movement disorders. I also investigated the neuropathological basis for schizophrenia in post-mortem tissue with Glenda Halliday at the Dr Darryl Eyles Prince of Wales Medical Research Institute, Sydney.

In 1998 I returned to the University of Queensland to model schizophrenia epidemiology in animals, first in the School of Biomedical Sciences and later at QBI from 2007. My work has led to the development of two main models of schizophrenia, one based on developmental vitamin D deficiency and the other on advanced paternal age. These models reveal mechanisms for abnormal brain development that may be relevant to patients with schizophrenia.

Dr Darryl Eyles

Apart from using rodent models to explore the link between developmental vitamin D deficiency and brain development, researchers at QBI are exploring this question using biological ‘banks’. Samples of blood are routinely collected from newborn babies in order to screen for various clinical disorders. These samples are often kept for many decades. A highly sensitive assay has been developed that can, for the first time, accurately measure vitamin D in tiny samples of dried whole blood. In collaboration with Danish researchers, the group is measuring vitamin D in blood from individuals who have developed schizophrenia versus healthy control subjects.

What is the Best Way to Measure Vitamin D in Newborn Babies?

Do the Children of Older Fathers have Altered Brain Development?

There is evidence from epidemiology that suggests the offspring of older fathers have an increased risk of schizophrenia, and also of autism. It is thought that mutations in the cells that produce sperm increase with age. These genetic alterations could be passed on to the children of older fathers, and subsequently disrupt the orderly development of the brain. QBI researchers have developed a mouse model of advanced paternal age, with PhD student Claire Foldi doing pioneering work in this field. There is preliminary evidence to suggest that the offspring of older mice differ in terms of behaviour and brain structure.

This project will develop an innovative ‘discovery platform’ that can help unravel the mechanisms of action that link advanced paternal age and altered brain development.

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Dr Thomas Burne

I studied at the University of New England, obtaining a Rural Science degree before gaining my PhD in neurophysiology and behaviour with Lesley Rogers in 1997. I then went to the UK as a postdoc with Steven Rose, examining correlates of learning and memory in humans and chicks.

In 1998 I moved to the Babraham Institute in Cambridge where I first investigated neural correlates of maternal behaviour in pigs and then behavioural paradigms of learning and memory in knockout and transgenic mouse models with Keith Kendrick and Lawrence Wilkinson. In 2002 I moved to Brisbane to work with Alan Mackay-Sim at Griffith University and, in 2005, joined the Queensland Centre for Mental Health Research, where my research is focused on aspects of behavioural neuroscience using animal models of altered brain development.

Awards and Prizes

In 2008 Xiaoying Cui was awarded a prestigious Queensland Smart State Fellowship for three years. The PhD students working with the group have also won awards: Claire Foldi was awarded Best Poster at the 2008 Australian Neuroscience Society Meeting in Hobart, and James Kesby was awarded Best Poster at the 2008 European Congress of Neuropsychopharmacology in Barcelona.
Neurodegenerative diseases, like Alzheimer’s (230,000 patients in Australia), Parkinson’s (80,000 patients), motor neuron disease (13,000 patients) and Huntington’s (1500 patients), represent a major portion of the burden of disease in our community. With an increasingly older population, this load will become more severe at both the economic level and the social level. For example, it is projected that there will be a 330% increase in the number of Alzheimer’s patients by 2050. Presently there are no therapies that are effective in ameliorating these conditions and, although agents like small interfering RNAs (siRNAs) have shown great promise, failure to deliver these novel drugs to the affected brain region has severely limited their clinical development.

One major hurdle is the inability of bioactive compounds to penetrate the blood-brain barrier and specifically target damaged or susceptible neuronal populations. To overcome this hurdle, scientists at QBI have teamed up with Max Lu and Gordon Xu at UQ’s Australian Institute for Bioengineering and Nanotechnology (AIBN) and Dongyuan Zhao at Fudan University, China, to develop a more efficient and reliable drug-delivery system based on a novel class of hybrid inorganic nanoparticles, the layered double hydroxides (LDHs). These special nanoparticles have unique properties that make them particularly adept at penetrating cell membranes, including those comprising the blood-brain barrier. The Nano-Neuro scientists are also exploiting the unique properties of LDH nanoparticles to carry magnetic resonance imaging (MRI) contrast agents into the brain.

**Huntington’s Disease Model**

The Nano-Neuro team has chosen to study the efficacy of LDH-based siRNA delivery to the damaged brain in the Huntington’s disease (HD) mouse model. Although siRNA approaches show great potential as HD treatments, there is still a major hurdle in their application due to the lack of an effective delivery system. Recent research in the Nano-Neuro program has now provided convincing evidence that LDH-siRNA complexes are efficiently taken up by cultured neurons and then released into the cytoplasm, where they deplete the levels of targeted proteins. Importantly, exposure to the LDH particles has no adverse effects on neuronal viability.

These studies provide strong evidence that LDH nanoparticles represent an effective and efficient drug-delivery system for the transport of bioactive molecules such as siRNAs into diseased neurons. The next step is to chemically modify the nanoparticles so that they can penetrate the blood-brain barrier in both healthy and HD mice. The long-term goal of the Nano-Neuro program is to adapt this LDH-siRNA drug delivery system to the treatment of other CNS diseases including Alzheimer’s, Parkinson’s, motor neuron disease, and the aggressive CNS cancer, glioblastoma multiforme.

**LDH-Based Contrast Agents for MRI**

CMR’s Nyoman Kurniawan and AIBN’s Gordon Xu have explored the possibility of using LDHs as carrier molecules for the MRI contrast agent, Gd-DTPA. The aim of this project is to enable transport of Gd-DTPA to many regions of the body. Of particular interest is the potential of LDH nanoparticles to act as Trojan horses to carry Gd-DTPA across the blood-brain barrier, which is not normally accessible to this MRI contrast agent. The intercalation of Gd-DTPA between the LDH layers creates LDH-Gd(DTPA) hybrid particles that undergo a change in their overall shape from hexagonal plate-like sheets to rod-like nanoparticles. The Nano-Neuro scientists believe that this shape change will enhance the ability of the LDH-Gd(DTPA) particles to cross the blood-brain barrier. In addition, the intercalation of Gd-DTPA into LDHs is likely to improve the sensitivity of the Gd(DTPA) as an MRI contrast agent. The next challenge for the team is to increase the stability of LDH-Gd(DTPA) in biological solutions by developing a more biologically friendly form of the nanoparticle complex.

**Nano-Neuro Personnel**

Professor Perry Bartlett, QBI  
Assoc Prof Helen Cooper, QBI  
Professor Gaoqing Max Lu, AIBN  
Professor Dongyuan Zhao, Fudan University

**Postdoctoral Fellows**

Dr Nyoman Kurniawan, CMR  
Dr Kathryn Markham, QBI  
Dr Zhi Ping ‘Gordon’ Xu, AIBN

**PhD Students**

Yunyi Wong, QBI/AIBN  
Ryan Harrison, AIBN
On leaving school, I completed an Associate Diploma in Applied Biology at the Queensland University of Technology and a Bachelor of Science at the Australian National University (ANU). I worked in a variety of areas in the 1980s, including bacteriology for a large company and water advisor with Queensland Water Resources, before specialising in flow cytometry at the John Curtin School of Medical Research, Australian National University.

From 1989 to 1999, my interest lay in developing novel flow cytometry technologies in software and hardware, with a particular emphasis on cell sorting and high-content screening applications. In 2000 I moved to the University of British Columbia in Canada to establish a core facility before returning to ANU in 2001, where I remained until accepting a position at QBI in 2004. My research interests involve novel implementation of flow technology in neuroscience, with a major focus on brain tumour and stem cell research.
Populations of dissociated cells labelled with different fluorescent markers to provide a unique optical signature (UOS) are displayed in three dimensions prior to software decoding.

From left: Virginia Nink, Geoff Osborne, John Wilson

Hardware and Software Development

Neural cells are of sufficient size that they have difficulty in passing through the small analysis channels present in flow cytometers. In order to address this shortcoming in conventional instrumentation, the Osborne Lab is developing a range of microfluidic devices that will integrate with existing instrumentation or be microscope mounted, and will allow the assessment and separation of cells, or even small animals such as C. elegans, based on their fluorescence characteristics.

The integration of this technology with imaging cytometry holds great promise for separating putative neural stem cells in a relatively high-throughput manner, based on a combination of morphological characteristics and fluorescence signals.

From left: Virginia Nink, Geoff Osborne, John Wilson

automated microscope-based robotic systems. However, by pooling samples using the UOS method, Virginia Nink and John Wilson (Osborne Lab) and Daniel Gilbert (Lynch Lab) have shown viable cells from 10 different sources, each containing a different chloride channel mutation, can be combined and the responses to agonists assessed in a high-content, high-throughput assay. The team has continued to expand this approach by developing online mixing hardware, which allows the rapid addition of various agonists delivered via a multiwell platform.
The Reinhard Lab seeks to elucidate principles of sensory processing, learning and memory in animals with small brains, such as the honeybee. Judith Reinhard investigates how honeybees process, learn and recall olfactory and visual information from their natural environment, and how the sensory information is encoded and stored in the brain. The lab uses elaborate behavioural methods for measuring sensory performance, from which the underlying neural mechanisms can be inferred.

**Scent Learning: From Complex Information to a Sparse Code**

In their daily search for food, honeybees encounter countless floral scents, which are highly complex mixtures of dozens or even hundreds of different odorants. Despite this, honeybees show an impressive capacity to discriminate and learn floral scents with high sensitivity and specificity. How does the bee brain successfully process such complex olfactory information?

To investigate this question, the Reinhard Lab used a learning assay custom-designed for bees, the proboscis-extension-reflex assay. Live bees were restrained in little holders and exposed to a mixture of 14 floral odorants. While smelling this scent, the bees were fed a drop of sugar water, thus quickly learning to associate the scent with the reward. The next time they were exposed to the scent, they stuck out their tongue (proboscis) in expectation of the reward. Using this method, Judith Reinhard tested which of the 14 odorants of the mixture were actually learnt by the bees. She discovered that bees only learn a handful of key odorants from a complex scent as representative for the entire mixture, and that most odorant information is ignored.

What makes a key odorant? The research showed that neither the chemical nature of an odorant, nor innate preferences for certain odorants play a role. It seems that the network of inhibitory and excitatory neurons in the first olfactory neuropil is responsible for reducing incoming olfactory information from a complex scent to the relevant key odorants.

This sparse code of key odorants is then passed on to higher brain centres where the information is learnt and stored, available for recall to trigger motor output.
Olfactory Plasticity: Effect of Scent Exposure

Judith also observed that honeybees varied in their olfactory learning performance, responding well to specific odorants at some times and poorly at others. In collaboration with the Claudianos Lab, she investigated whether continued exposure to a scent has an effect on the molecular basis of scent processing and learning.

The team studied expression patterns of olfactory receptor proteins (ORs), which are located on the sensory neurons and responsible for binding odorants. Bees were either trained to a specific scent by associating it with a sugar reward, or merely exposed to the same scent over two days. Judith found that OR expression changed significantly when bees had to learn the scent, but did not change when bees were merely exposed to the scent without learning an association. This suggests that there is a feedback mechanism from the higher brain centres (scent learning) to the periphery (scent detection) that controls OR levels according to the association formed with a scent. This adaptation mechanism is the likely cause for the observed plasticity in olfactory learning, i.e. a scent is learnt better when a prior memory for it already exists.
The Reutens Lab, which is in a development and recruitment phase, works on imaging techniques to explore the workings of the nervous system in health and disease.

**PET Ligands for Alzheimer’s Disease**

The aim of this project is to develop a new method of diagnosing Alzheimer’s disease and to assess response to treatment. Current PET imaging agents suffer from the disadvantage of requiring an on-site cyclotron. The research in this laboratory is oriented towards the development of ligands labelled with long half-life positron emitters so as to remove the requirement for proximity to a cyclotron. A number of candidate molecules have been synthesised and their binding characteristics are currently being tested in vitro in collaboration with the Coulson Lab.

**MRI Contrast Agents**

In collaboration with the School of Chemistry and Molecular Biosciences, we are evaluating a number of compounds that are sensitive to local redox potential. Ultimately these may be of value in identifying the ischaemic penumbra following stroke, tissue that is threatened but potentially salvageable.

**PET Attenuation Correction**

The Reutens Lab is also working on MR-based attenuation correction of PET data. This is an important technical issue that must be overcome if hybrid MR-PET scanners are to become useful for PET quantitation. Current PET-CT scanners use information from CT to correct for attenuation of photons that result from positron annihilation in tissue. Because conventional MR sequences provide less information than CT on high-attenuation tissues such as bone, new techniques are required for attenuation correction for MR-PET.

**Ultra low-field MRI**

In collaboration with the School of Mathematics and Physics, we are working on the development of a MRI device that can operate at microtesla fields. The ability to perform MRI at low field is an important step towards the ultimate goal of using MRI to measure neuronal currents directly.

**Mouse Brain Mapping**

The Reutens Lab is developing methods for registration of MR and histological data acquired for the Australian Mouse Brain Mapping Consortium. This will allow the generation of three-dimensional volumes of histological data, a necessary step towards statistical comparison of brain morphology between groups of animals. Others in the lab are involved in segmentation of hippocampal, thalamic and cerebellar subregions as part of the endeavour to create the next generation of digital atlases. An important part of this is the definition, in collaboration with Charles Watson in Perth, of operational criteria for standardised segmentation of structures on high-resolution MR images.

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**Professor David Reutens**

I graduated in medicine from the University of Western Australia in 1984 and trained in neurology at Royal Perth Hospital, the Austin Hospital and the National Hospital for Neurology and Neurosurgery, Queen Square, London. I completed my MD at the University of Melbourne in 1992 and a postdoctoral fellowship in neuroimaging and epilepsy at Montreal Neurological Institute (1993–1996). I have been Assistant Professor of Neurology and Neurosurgery, MNI and McGill University (1996–1998), Associate Professor, Department of Medicine, the University of Melbourne (1998–2003), and Professor of Neuroscience, Monash University (2003–2008). Between 2003 and 2008, I was Director of Neurology at Southern Health, Victoria’s largest health care network. In 2008 I was recruited to UQ as the Foundation Chair in Experimental Neurology and to head the new Centre for Advanced Imaging. My research interests centre on the development and use of new methodological and analytical imaging tools to study disorders such as epilepsy and stroke.
The Centre for Advanced Imaging (CAI) is a strategic initiative developed through the Queensland Brain Institute. The establishment of the Centre reflects the burgeoning demand for improved imaging research capabilities, arising from the growth in biotechnology and biomedical research at UQ. Professor David Reutens, who joined UQ in September 2008, is the inaugural Executive Director of the Centre.

In establishing CAI, the overall goal is to create a world-class facility for research and training in biomedical imaging. The Centre will provide an integrated, multidisciplinary and multimodal research framework for basic, translational and clinical research in imaging. CAI brings together the skills of a critical mass of researchers and state-of-the-art imaging instruments such as high-field human MRI scanners (1.5T, 3T and 4T), animal MRI scanners (4.1T and 16.4T), human and animal PET scanners, and radiochemistry facilities. The equipment includes flagship instruments of the National Imaging Facility, such as the combined PET/MR scanner to be delivered in 2010.

CAI has nodes at clinical research sites with the aim of maximising the impact of imaging on translational research. CAI will be the only facility of its type in Australia and one of only a handful in the world. It will progress treatments for major diseases such as stroke, dementia, heart disease and cancer by comprising a comprehensive “end-to-end” biomedical imaging capability. This will allow integrated progression from the laboratory bench, through animal models, and finally into a clinical setting. Imaging techniques are now key platform research technologies for studying the structure and function of living organisms, in health and disease, from the laboratory mouse to the human. The ability of ultra high-field MRI to characterise the blood flow and structure of living systems, together with developments in MRI biomarkers, will allow UQ researchers to better phenotype animal models of disease and to map the cognitive function of the brain. PET measures the distribution and fate of molecular markers using radiolabelled ligands, therefore providing UQ researchers with the capacity to perform in vivo studies of metabolism, receptor-ligand binding and gene expression.

The ability to study the living organism enables longitudinal studies of normal development, of the natural history of disease, and of responses to novel therapies. Consequently, in vivo imaging methods have also become platform technologies for drug discovery and validation. By providing surrogate end points to assess the effectiveness of new therapies in clinical trials, a coherent imaging framework also speeds translation of scientific discoveries to clinical realisation. MRI and PET are now core investigative modalities in virtually all clinical specialties, informing clinical diagnosis and prognosis and facilitating the goal of personalised medicine by better characterising both disease and its response to treatment in the individual patient.

Standing, from left: Jana Vukovic, Greg Pierens, Steven Yang, Nyoman Kurniawan, David Reutens, Deming Wang, Giang Nguyen, Tarac Venkatachalam (Venku), Natalie Alexopoulos, Georg Kerbler. Seated, from left: Viktor Vegh, Marianne Keeler
The goal of the Richards Lab is to investigate the mechanisms that regulate development of the forebrain. Three major projects are currently under investigation in the laboratory.

**Cortical Axon Guidance**

The corpus callosum connects neurons in the left and right cerebral hemispheres and is the largest fibre tract in the brain. It is required for the coordination of brain information processed in each cerebral hemisphere. Defects in the development of the corpus callosum are associated with a large number of human congenital syndromes resulting in severe to mild cognitive deficits. The Richards Lab has had a long-standing interest in understanding the developmental mechanisms that regulate the formation of the corpus callosum.

Postdoc Tom Fothergill and PhD students Amber-Lee Donahoo and Divya Unni are investigating the molecular regulation of axon guidance at the cerebral midline, including the function of molecules such as Slit and Netrin, and their receptors Robo and DCC. Classically these ligand-receptor pairs have been studied independently but new data from our laboratory is showing how these signaling families interact to modulate axon growth and guidance. Recently, honours student Oressia Zalucki and postdocs Michael Piper and Tom Fothergill have discovered that Neuropilin 1 and its ligands, Semaphorin 3A and 3C, regulate the development of the pioneering axons of the corpus callosum.

**Development of All Forebrain Commissures**

The correct molecular patterning of the forebrain is essential for the formation of commissural projections such as the corpus callosum. In this project, postdoc Randal Moldrich and PhD student Ilan Gobius are investigating whether the mechanisms that regulate corpus callosum formation also regulate the formation of all commissural projections in the forebrain, including the hippocampal commissure and the anterior commissure. Such mechanisms include the formation of a region of the cortical midline called the commissural plate, and the fusion of the two telencephalic hemispheres, providing a substrate for axon growth. Using an *in vitro* assay, Ilan Gobius and Tom Fothergill have shown that fibroblast growth factor-8 (FGF8), expressed by the commissural plate, regulates hippocampal axon guidance.
Using magnetic resonance imaging, Randal Moldrich and masters student Bob Ren have also discovered how each commissural projection develops in relation to the commissural plate and the other commissural projections in this region of the brain. In a related project, PhD student Janette Thurley is investigating the postnatal development of the corpus callosum and a cellular population known as the subcallosal sling.

**Nuclear Factor One (Nfi) Genes in Cortex and Hippocampus Development**

The Nfi genes are transcription factors that regulate neuronal and glial differentiation as well as the formation of axonal tracts within the brain. Mutations in the Nfi genes cause severe brain defects in patients, as well as in mouse models of these disorders. There are four different Nfi family members and the Richards Lab is investigating how each family member, either alone or in combination, acts to regulate important developmental events. Postdocs Guy Barry and Michael Piper showed that the Nfi genes are essential for the morphological development of the hippocampus by regulating the differentiation of glial populations. Postdoc Charlotta Lindwall, PhD student Sharon Mason and summer scholar Sean Coakley have found that these genes also regulate neuronal development, specifically in the developing cerebral cortex. Michael Piper, Guy Barry and Sharon Mason continue to probe the molecular cascades driven by these transcription factors in an effort to gain a deep understanding of their function. Research assistants Erica Little and John Baisden facilitate our discoveries and work across all the projects in the laboratory.
Our stored memories, and the emotions that go with these memories, in large part make us who we are. There is little doubt that the mammalian brain, a complex organ, is responsible for all our activities, including the control of our behaviour, our memory formation and our cognitive skills. 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 the mind but also for the discovery of therapies that can alleviate many of the related disorders that afflict us, such as anxiety, depression and stroke. The Sah Lab studies the cellular and molecular mechanisms that underlie learning and memory formation. To achieve these goals, the lab focuses 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 lab uses a combination of electrophysiological recording, calcium imaging, molecular analysis, anatomical reconstruction and behavioural analysis to understand the circuitry of the amygdala and how activity in this circuitry leads to learning and memory formation.

Using molecular biological tools combined with electrophysiological recordings, postdoctoral fellow Andrew Delaney is examining the molecular identity of different receptors in the amygdala. The aim of these studies is to determine the subunit composition of receptors at defined connections in the amygdala. Many of the pharmacological agents that affect nervous system function (such as valium or diazepam) do so by acting at neurotransmitter receptors and ion channels at connections in the brain. Unfortunately, because we do not understand the exact molecular composition of many receptors, these therapeutic agents also cause many side-effects. A better knowledge of receptor composition will facilitate the development of more specific drugs that can be targeted
to specific synapses, thus avoiding a number of the adverse side-effects of currently available pharmaceutical agents.

John Power, also a postdoctoral fellow, is studying how synaptic activity changes intracellular calcium in neurons in the basolateral amygdala. Using multiphoton imaging of calcium, John has found that repetitive stimulation of synapses onto amygdala neurons generates a wave of rising calcium, which begins in the dendritic tree and propagates into the soma and the nucleus of the neurons. These calcium rises are well known to initiate changes in gene transcription and may underlie how long-term memory is stored.

Two other postdoctoral fellows in the lab, Francois Windels and James Crane, are studying how large assemblies of neurons process incoming sensory information in awake behaving animals. By using implanted electrodes in the amygdala, it is possible to study the electrical activity of large numbers of neurons simultaneously while animals explore their environment. These studies are providing key data on how information is encoded and transmitted to the amygdala.

Finally, in collaboration with the Bartlett Lab, members of the Sah Lab are also investigating how newly born neurons in the adult brain are integrated into the existing circuitry to form connections and fulfill functional roles.
Flying insects display remarkable visual agility despite their small brains and relatively simple nervous systems. The honeybee is being used as a model to elucidate the principles of flight guidance and navigation. A related aim is to explore insect-inspired strategies for the guidance of autonomous aerial vehicles.

**Polarisation Compass in Navigation**

Little is known about how information on the distance and the direction of flight are combined to pinpoint the location of the destination. This question has been addressed through two studies conducted principally by Carla Evangelista. The first study shows that bees, flying in tunnels covered with polarisation filters, use the orientation of the polarised light as a compass to signal the direction of the food source in their dances. The second study shows that bees can be trained to use the overhead polarisation as a compass to find their way through a maze.

**Mid-Air Collision Avoidance**

How do flying insects avoid collisions with other insects? This question was investigated in the lab through two approaches crafted by Peter Kraft: (1) video-taping bees flying head-on towards each other in a tunnel and manipulating the resulting visual stimuli (2) analysing high-speed films of bees flying in high-density air spaces. The results suggest that collision avoidance is mediated primarily by cues derived from image motion induced by translatory flight.

**Honeybee Landing – Final Moments**

Although some studies have examined how flying insects control their approach during landing, little is known about how the final touchdown is orchestrated. High-speed movies of landing bees are now revealing that honeybees enter a hover phase at a distance of ~1.5 cm from the target surface, just prior to touchdown. The hover distance is remarkably constant, irrespective of whether the bee lands on the floor, on an inclined surface, or on the ceiling. Carla Evangelista, Peter Kraft, Marie Dacke (University of Lund) and Judith Reinhard are exploring the roles of various sensory cues in guiding the final phase of landing.

**Visually Driven ‘Streamlining’ Response**

Bees are also being studied in tethered flight, with the aim of combining behaviour with the underlying electrophysiology. Tien Luu finds that when tethered bees are exposed to image motion that simulates forward flight in a virtual-reality arena, they raise their abdomens in a systematic velocity-dependent way, even in the absence of airflow.

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**Professor Mandyam Srinivasan**

My career began with a bachelor’s degree in electrical engineering from Bangalore University, followed by a master’s degree in electronics from the Indian Institute of Science, and a PhD in engineering and applied science from Yale University in 1976. By then my interests had turned towards insect vision and, after periods spent working at Yale and the University of Zurich, I was appointed a Fellow in Visual Sciences at ANU’s Research School of Biological Sciences.

By now my major focus was visual processing in the honeybee, and what began as a one-person operation (I even did my own beekeeping) has since grown to the flourishing group I have established at QBI, since being recruited as Professor of Visual Neuroscience in 2007. Here my research continues to focus on the principles of visual processing, perception and cognition in simple natural systems, and on the application of these principles to machine vision and robotics.
Target Tracking and Interception by Aggressive Honeybees

Eliza Middleton is studying how aggressive bees detect, pursue and intercept a motorised moving target. Aggression is induced by an ‘aggravator’ device placed at the hive entrance, and the bees’ flights are recorded on high-speed stereo video cameras. Eliza finds that aggressive bees can discriminate a moving target from a stationary one. While a foraging bee decelerates to a smooth landing on a flower, an aggressive bee accelerates constantly, impacting its target at high speed. Timothy Mew, a summer student, has developed a mathematical model to characterise the pursuit trajectories.

Visual Guidance of Bird Flight

Do the principles of visual guidance that this laboratory has been investigating in honeybees also apply to other flying creatures, such as birds? Partha Bhagavatula finds that: (1) budgerigars, like bees, tend to land in the vicinity of highly contrasting visual features; (2) during landing, the process of edge detection appears to be ‘colour-blind’, even though these birds possess excellent tetrachromatic colour vision; (3) budgerigars, like bees, negotiate narrow passages safely by balancing the speeds of image motion that are experienced by the two eyes.

Biologically Inspired Machine Vision and Robotics

The biorobotics group has two major aims: (1) to test our understanding of the principles of insect visual guidance by implementing them in vehicles; (2) to enhance the performance of machines through biologically inspired algorithms. Dean Soccol fabricated vision systems for model aircraft, Saul Thurrowgood refined computer algorithms for the measurement of image motion, Navid Nourani successfully implemented honeybee-like visual odometry in terrestrial vehicles, and Richard Moore developed vision algorithms for use in obstacle detection, terrain following and landing.
The Thinking Systems program brings together five CIs from QBI and four CIs associated with the School of Information Technology & Electrical Engineering (ITEE), under the project direction of Professor Janet Wiles (ITEE), to study how brains understand spatial systems, both physical and conceptual. Insights from neurocognitive systems are being used to develop computational models, autonomous robots and intelligent software agents. The program is divided into five broad themes, which together are leading to a deeper understanding of the relationship between neurocognitive mechanisms and their behaviour in whole systems.

Theme 1. Neural Mechanisms for Navigation
This theme is investigating neural mechanisms for navigation in insects and freely behaving rodents via electrophysiology and behavioural observation. This research is helping design computationally efficient and reliable algorithms for autonomous navigation. A virtual reality set-up has been developed in which the behavioural responses of honeybees can be observed and recorded while they believe they are flying through a virtual world. This experimental set-up led to a novel finding – the abdomen of the flying honeybee changes position in response to the change in optic flow. A freely behaving rat laboratory has also been set up for the project. This lab will allow us to test theories of rodent navigation, as well as directly examine the neural mechanisms that underlie navigation behaviour.

Theme 2. Spatial Maps and Neurally Inspired Algorithms
This theme is uncovering fundamental computational principles involved in generating, updating and using efficient spatial representations, and developing mathematical and computational models inspired by neurophysiological and neurocognitive data from insects, rodents and humans. Several papers have been published on the abilities of, and the limits implied by, different navigational strategies. These abilities and limits inform and constrain both the potential sensory and neural mechanisms of navigation, and the computational algorithms we develop. Also, a functional model of a component of the rat navigation system has been created. This model may explain the need for a certain type of neuron that had not been explained before.
Theme 3. Biologically Inspired Robot Navigation

Robotics plays two roles in this project. Firstly, robots are serving as mechanisms for embodying the neurocomputational models of navigation in real physical spaces, with focus on the biological plausibility of the models. Secondly, the neurocomputational models are serving as inspiration for frontier technologies for sensing and navigation in autonomous robots. The focus in the second role is on engineering effectiveness.

Work in this theme has demonstrated for the first time that a robot can successfully map and navigate through an unknown cluttered dynamic environment for days at a time with no human assistance. The robot navigation system, called RatSLAM, appeared on the Australian national science program Catalyst. A lightweight MATLAB version of RatSLAM has been released for use by other researchers.

Other mechatronics engineering solutions being developed for the project include: wireless telemetry for neural recording, an omni-directional drive platform, interactive robot web interface, and an air-cushioned treadmill ball for insects. The robot navigation model consists of a network of location- and orientation-sensitive cells and an episodic spatial memory map.

Theme 4: From Physical to Conceptual Spaces

Perceptual and cognitive representations of the spatial layout of visual environments in humans are subserved by a complex network of cortical and subcortical brain regions. To what extent has the uniquely human ability to acquire and manipulate complex representations of conceptual space been dependent upon the neural hardware that has evolved for representing real (sensory) spatial maps? This theme is seeking convergent evidence for the neural underpinnings of real and conceptual space representations using functional neuroimaging, reversible neurodisruption, and focal brain lesions.

fMRI experiments have been conducted on healthy subjects during navigation tasks designed to find brain regions involved in landmark learning and navigation. These results provide the first evidence that purely landmark-related learning is mediated by hippocampal, parahippocampal and striatal systems, even within a single trial and without reinforcement. In other work, behavioural tests on healthy subjects were conducted, focused on investigating the effects of proximal and distal landmarks on object-location memory. These tests had not been done before in humans. Results suggest that subjects were fastest and most accurate at recalling locations using proximal landmarks.

Theme 5: Navigating Information Spaces

This theme is using insights from biological navigation systems to develop algorithms for automated concept mapping. In particular, the aim is to understand how trajectories through information spaces are used to build maps and how to use maps to achieve goals.

A patent for using minimum spanning trees and shortest paths in concepts maps with a distal component in their cost function has been commercialised. A concept mapping software system, called ConceptSLAM, is being designed using principles of the biological hippocampal and parietal systems as well as the physical robot navigation system RatSLAM. The research has identified metrics useful for measuring movement in concept space, and methods for extracting conceptual landmarks using lexical statistics. In addition, new methods for concept and word association have been developed, as well as a series of interactions for mobile robots using generated language. Together with the underlying cognitive map representation, these methods and interactions enable the formation and grounding of spatial concepts in mobile robots, including toponyms (place names) and spatial relations (directions and distances).

Thinking Systems Personnel

Chief Investigators
Professor Janet Wiles (Director), ITEE
Professor Perry Bartlett, QBI
Professor Kevin Burrage, IMB
Assoc Prof Geoffrey Goodhill, QBI
Professor Jason Mattingley, QBI
Professor Pankaj Sah, QBI
Dr Andrew Smith, ISSR
Professor Mandyam Srinivasan, QBI & ITEE
Dr Gordon Wyeth, ITEE

QBI Affiliates
Dr Bruno van Swinderen
Dr Charles Claudianos
Dr Judith Reinhard
The structure and function of the retina have been elucidated in remarkable detail and this is facilitated by the highly ordered neuronal architecture. Because the intact retina can be readily isolated from the rest of the central nervous system, the retina has been described as ‘Nature’s brain slice’. The Vaney Lab is studying how excitation and inhibition interact in the inner retina to produce the complex receptive-field responses of retinal ganglion cells (RGCs), which are the output neurons of the retina.

**Uniformity Detector RGCs**

RGCs convey information by increasing their firing in response to an optimal visual stimulus or ‘trigger feature’. However, one class of RGC responds to changes in the visual scene by decreasing its firing. These ‘uniformity detectors’ are encountered only rarely and the synaptic mechanisms underlying their unusual responses have not been investigated.

Ben Sivyer has been able to target uniformity detectors with a high success rate in a whole mount preparation of the rabbit retina, which has enabled him to characterise the synaptic mechanisms that govern their unusual light responses.

The maintained firing of uniformity detectors is transiently suppressed by bright or dark contrast. Patch-clamp recordings show that the action potentials arise within ‘complex spikes’, each comprising a burst of 2 or 3 Na+ spikelets riding on top of a slower Ca2⁺-mediated depolarization. Both ON and OFF visual stimuli elicit only inhibitory synaptic input, the immediate effect of which is to suppress the maintained firing. However, this inhibition also alters the properties of the resurgent spiking by increasing the amplitude of the spikelets within each burst, suggesting that this may increase the efficacy of spike propagation and transmission. This appears to be the first report of a RGC that (1) produces complex spikes and (2) receives negligible bipolar cell input.

**Intrinsic Properties of RGCs**

Although RGCs are excited and inhibited by inputs from retinal interneurons, they are not relay stations that simply sum their synaptic inputs. RGCs have intrinsic membrane properties, allowing them to amplify or filter-out specific signals. Refik Kanjhan has been studying the intrinsic membrane properties of RGCs, revealing distinct types of RGCs with special ion channels embedded in their membranes. These channels are critical for burst firing and spontaneous rhythmic activity, firing in a way similar to that seen in heartbeat and the respiratory system.

These rhythmic activities are particularly important during early development prior to eye opening. With maturation, the rhythmic activities are mostly...
PhD student Ben Sivyer makes patch-clamp recordings from neurons in the isolated retina and measures the synaptic inputs to the cells in response to controlled visual stimuli.

Inhibited or blocked by light-evoked synaptic responses, and by leak currents and potassium channels in the RGC membranes. In the adult, these channels contribute to the shape and duration of the responses to light. However, when inputs are reduced during sleep, these channels may provide continuity of interaction with the brain.

**Pattern Discrimination in Reef Fish**

Uli Siebeck has been studying the ability of reef fish to learn and distinguish complicated natural and artificial patterns. This research was inspired by Uli’s previous finding that some reef fish species have complicated UV-reflective facial patterns, which differ between species and, on a much smaller scale, also between individuals. Since then, Uli and her colleagues have been able to show that the Ambon damselfish (*Pomacentrus amboinensis*) uses these facial patterns for species and individual recognition and that the fish are very sensitive to small changes within these natural patterns.

Uli is now using this system to investigate the underlying mechanisms.

Are the fish relying on simple spatial frequency differences between patterns, or do they have the ability to distinguish complicated patterns using shape information only? This question is being investigated with behavioural experiments using classical conditioning for training and a two-alternative forced choice procedure for testing. The results show that the fish are able to learn and discriminate between complicated artificial patterns using shape information only. This indicates that, rather than relying on simple low-level spatial frequency analyses, higher level processes are involved in pattern discrimination in reef fish.

*From left: Nick Nacsa, Ben Sivyer, David Vaney, Uli Siebeck, Refik Kanjhan*
Attention and memory seem to be intertwined processes: when we pay attention to an object it helps us remember it, but our memories also bias the objects to which we pay attention. This ongoing interaction between attention and memory influences our behaviour and the choices we make when experiencing the world.

But what is the nature of this interaction at the level of brain function? In humans, we might be overwhelmed by the complexity of the problem and just call it “consciousness”. However, the relationship between attention and memory can effectively be investigated in simpler organisms such as the fruit fly Drosophila melanogaster.

Drosophila has been extensively used to uncover molecular and cellular mechanisms of learning and memory, and many discoveries in the fly have been found to generalise across species, including humans. Studying attention in a fly is a bit trickier as it is difficult to really know what an animal perceives. To study selective attention in an animal requires measures of suppression, namely of the competing stimuli being actively ignored. This can be accomplished by both behavioural approaches and brain recordings.

The van Swinderen Lab has developed unique paradigms to study visual selective attention in Drosophila, with the goal of understanding how attention works in a small insect brain. Novel behavioural assays allow for efficient screening of genes and circuits relevant to visual perception, while electrophysiology in select fly strains permits detailed investigation of neural correlates of selective attention. Using these new technologies, questions typically reserved for human visual attention studies can now be asked of a fly.

There are great advantages to using Drosophila for studying complex processes such as attention and memory. First, the fly accomplishes these functions via a much simpler brain quite unlike our own. This means that mechanisms uncovered in Drosophila will reveal fundamental properties of memory and attention not necessarily tied to the anatomy specific to humans. Also, to fully understand how a brain works, molecular genetic techniques in Drosophila allow for a level of manipulation unparallelled in any other organism.
Molecular Genetics of Attention

Suppression mechanisms characteristic of selective attention require proper wiring in the brain. Neuronal connections made during brain development and during learning in adulthood depend on a set of molecules involved in synaptic plasticity and adhesion. Narelle Tunstall, in collaboration with the Claudianos Lab, is designing transgenic constructs to manipulate wiring in the fly brain, in order to target attention and memory processes.

Behaviour Genetics of Attention

An automated, high-throughput assay for visual responses in flies has been developed in the lab to screen mutant strains for attention-like behaviour. A comparative survey of visual responsiveness in the short-term memory mutant dunce and wild-type flies has revealed a number of visual paradigms most relevant to detecting attention defects in Drosophila. Oliver Evans, a research assistant in the lab, is using these paradigms to uncover the basic circuitry supporting visual attention in the fly brain.

Electrophysiological Studies of Attention

The project of postdoctoral fellow Angelique Paulk focuses on neural correlates of visual attention. Using tiny electrodes and competing visual stimuli, Angelique follows the patterns of stimulus selection and suppression in the insect brain by recording activity from single cells (‘units’) and populations of neurons (‘local field potentials’). Oscillations in brain activity are often associated with attention, and the insect brain provides an ideal preparation to investigate how these oscillations might cause selection or suppression of stimuli. Honeybees are used to uncover the physiology of these processes, and Drosophila to manipulate the processes.

From left: Nigel Thomas, Narelle Tunstall, Bruno van Swinderen, Oliver Evans, Angelique Paulk
The Wallace Lab is using a multidisciplinary approach to research disorders of the central nervous system such as epilepsy and motor neuron disease. Techniques include human and mouse genetics, molecular and cellular biology, and magnetic resonance imaging.

**Epilepsy**
Epilepsy is a common complex disorder with a strong genetic component. In 2008, the Wallace Lab identified two new human epilepsy genes. The first gene, GLI3, is associated with rare brain lesions known as hypothalamic hamartomas. These lesions frequently cause an unusual type of epilepsy associated with repetitive laughing (gelastic seizures). Patients with hypothalamic hamartomas and gelastic seizures were found to have mutations in the transcription factor GLI3.

The second gene discovered, PRICKLE1, causes progressive myoclonic epilepsy and ataxia. The discovery of novel epilepsy genes is increasing our understanding of the mechanisms involved and provides the basis for the development of new treatment strategies. It will also provide the opportunity for accurate diagnosis and determination of carrier status, within families where a gene is identified. Future research is aimed at using animal models to study the changes that occur following severe seizures, after which there is marked cell loss in certain regions of the brain, followed by increased production of new neurons. However, very little is known about this process. In collaboration with the Bartlett Lab, experimental mouse models of epilepsy have been instrumental in demonstrating that the neural excitation produced by seizures is capable of activating stem cells in the hippocampus.

**Motor Neuron Disease (MND)**
MND is the third most common adult neurodegenerative disease, after Alzheimer’s and Parkinson’s. In patients with MND, the neurons that control motor functions begin to die, affecting the muscles of the arms and legs, as well as those which control breathing and speech. With no nerves to activate the muscles, the patients begin to waste, resulting in paralysis and difficulty in speaking, swallowing and ultimately breathing. The causes of MND are poorly understood; there is no diagnostic test and no cure.

**Dr Robyn Wallace**

After completing a science degree at Flinders University in 1994, I undertook a PhD at the University of Adelaide supervised by the renowned geneticists, John Mulley and Grant Sutherland. The highlight of this period was my discovery of the first gene for a form of idiopathic generalised epilepsy.

Following the award of my PhD in 1998, I worked with Bionomics Ltd to develop drug targets and genetic tests for epilepsy. In 2002 I was appointed Assistant Professor at the University of Tennessee Health Science Center, where I extended my genetics experience to include the analysis of mouse epilepsy models.

In 2005 I joined QBi as the inaugural Ross Maclean Senior Research Fellow, and I am now applying my expertise in human and mouse genetics to the analysis of motor neuron disease. My research goal is to understand how genetic variation contributes to human disease.
Although mouse models are invaluable for studying the genetic and molecular mechanisms associated with brain disorders, very few models currently exist that are relevant to MND. Through the Australian Phenomics Facility, the Wallace Lab has access to hundreds of mice carrying thousands of random point mutations. Tim Butler, a research assistant in the lab, screened these mice for loss of motor function and identified four lines of mice with MND-like symptoms. Postdoctoral fellow Marie Mangelsdorf and research assistant Ajay Panwar are now focusing on further characterising these mice and identifying the genetic mutations. Mutations in two different ion channel genes were recently discovered in two of the four selected mouse lines. Identifying mouse MND genes will improve our understanding of what causes MND and potentially provide new drug targets.

Mouse models are invaluable for testing potential therapies for MND. The Wallace group is currently using a well established MND mouse model (SOD1 transgenic) to investigate ways of preventing the motor neuron loss associated with MND. A postdoctoral fellow in the lab, Clare Underwood, developed new MRI techniques to track disease progression, which will be invaluable for assessing the efficacy of treatments for MND. In collaboration with the Coulson Lab, the team is also assessing the role of a nerve growth factor receptor (p75) by eliminating the gene from our SOD1 mouse to determine whether this prevents MND onset. In addition, the therapeutic potential of QBI-developed peptides specifically targeted to the p75 protein is being investigated.
The Queensland Brain Institute is welcoming three new Faculty members in 2009, making a total of 28 Faculty going into 2010.

### Professor Bryan Mowry

After graduating in medicine from UQ and completing an arts degree at the University of Western Australia, I specialised in psychiatry. In 1998, I spent a sabbatical studying statistical genetics with Jürg Ott at Rockefeller University and, in 1999, I was awarded a MD degree from UQ in the molecular genetics of schizophrenia.

I am Director of Genetics at the Queensland Centre for Mental Health Research and a Professor in the Department of Psychiatry at UQ. Before joining QBI, my lab was located at the Queensland Institute of Medical Research, where I am an Honorary Principal Research Fellow.

My research goal is to identify genetic variants predisposing to schizophrenia. I have recruited and diagnosed large collaborative samples, with a particular focus on ethnically homogeneous populations in India and Sarawak. My program is currently expanding to study the functional effects of identified genetic variants in these data.

### Dr Timothy Bredy

Originally from Halifax, Canada, I completed an honours degree in experimental psychology at Dalhousie University. I then moved to McGill University to work with Michael Meaney, earning a PhD in 2004 on the subject of gene-environment interactions and the influence of early life experience on cognitive development.

From 2005–2009, I was awarded FRQS, NSERC and CIHR research fellowships to pursue postdoctoral training at the University of California, Los Angeles, where I worked with Mark Barad (fear-related anxiety disorders) and Yi Sun (epigenetics and stem cell biology).

In 2009 I am establishing my own lab at QBI. The aim of my research is to understand how epigenetic mechanisms contribute to the formation and maintenance of long-term memories, particularly within the context of psychiatric disorders such as phobia, post-traumatic stress disorder, and the addictions.

### Dr Stephen Williams

Originally trained as an electronics engineer, I became deeply interested in neuroscience while a medical technician at a neurology hospital. I returned to university to study neuroscience, receiving an honours degree and PhD from the University of Wales.

I was a postdoctoral fellow at the University of California, Los Angeles, and later at the John Curtin School of Medical Research, Canberra. In 2003, I formed my own lab at the MRC Laboratory of Molecular Biology in Cambridge, England, and was awarded tenure in 2008.

I am excited about moving to QBI at the end of 2009 to establish a research program focused on understanding the operation of neocortical neuronal circuits.

Our lab has helped to pioneer direct recording techniques from the dendrites of central neurons and we have introduced many technical innovations that have helped to elucidate the electrical operation of neurons.
Tim Silk, an Australian Clinical Research Fellow in the Cunnington Lab, studies how the brain interprets the world in attention deficit hyperactivity disorder.
QBI Director
Professor Perry Bartlett FAA

QBI Deputy Director
Professor Pankaj Sah

QBI Faculty Members
Professor David Adams (from Jan 2008)
Assoc Prof Mark Bellgrove
Dr Thomas Burne – Adjunct (from Jan 2008)
Dr Charles Claudianos (from Feb 2007)
Assoc Prof Helen Cooper
Dr Elizabeth Coulson
Assoc Prof Ross Cunnington
Dr Geoffrey Ericksson (until Jan 2008)
Dr Darryl Eyles – Adjunct (from Jan 2008)
Dr ES Louise Faber
Assoc Prof Geoffrey Goodhill
Dr Robert Hester (until Dec 2008)
Dr Massimo Hilliard (from Sep 2007)
Professor Joe Lynch (from Jan 2008)
Professor Jason Mattingley
Professor John McGrath – Adjunct
Assoc Prof Frederic Meunier (from July 2007)
Mr Geoffrey Osborne
Dr Judith Reinhard (from Feb 2007)
Professor David Reutens (from Aug 2008)
Professor Brent Reynolds (until Feb 2008)
Assoc Prof Linda Richards
Dr Rodney Rietze (until Dec 2008)
Professor Mandyam Srinivasan FAA FRS
Dr Bruno van Swinderen (from Jan 2008)
Professor David Vaney (from Jan 2008)
Dr Robyn Wallace

University of Queensland Affiliates (Current)
Professor Andrew Boyd (QIMR)
Professor Chen Chen (SBMS)
Professor Shaun Collin (SBMS)
Professor Justin Marshall (SBMS)
Assoc Prof Peter Noakes (SBMS)
Dr Ethan Scott (SBMS)
Professor Walter Thomas (SBMS)
Assoc Prof Guy Wallis (HMS)
Professor Janet Wiles (ITEE)

Honorary Professors
Professor Dexter Irvine (Monash University)
Professor Peter Mombaerts (Max Planck Institute)
Professor Hideyuki Okano (Keio University)
Professor Seong-Seng Tan (Howard Florey Institute)
Research Fellows and Postdocs

Dr Daniel Angus
Dr Emily Baird (Dec 07–Jan 08)
Dr David Ball (from June 2008)
Dr Guy Barry (from Jan 2008)
Dr Oliver Baumann (from Oct 2007)
Dr Paul Beatus (until June 2007)
Dr Sunita Biswas (from May 2008)
Dr Daniel Blackmore
Dr Brid Callaghan
Dr Allen Cheung (from July 2007)
Dr Robert Colvin (from Oct 2008)
Dr James Crane
Dr Xiaoying Cui (from Jan 2008)
Dr Denis de Assis (from July 2007)
Dr Robert Colvin (from June 2008)
Dr Timothy Silk
Dr David Smith (Mar 07–Mar 08)
Dr Jay Spaman (from Mar 2008)
Dr Mark Spamer – Adjunct (from June 2008)
Dr Melissa Starfield (July 07–Feb 08)
Dr Peter Stratton (from Feb 2007)
Dr Robert Sullivan (from June 2008)
Dr Alexander Sykes
Dr Huajin Tang (from Feb 2007)
Dr Narelle Tunstall – Adjunct (from Nov 2008)
Dr Matthew Ireland (from Feb 2008)
Dr Angela Boyce (from Nov 2007)
Dr David Lloyd (from Dec 2007)
Dr Eirinn Mackay (from Feb 2008)
Dr Matthew Pellekanos (Nov–Dec 2008)
Dr Qiang Shan (from Dec 2007)
Dr Timothy Silk
Dr David Smith (Mar 07–Mar 08)
Dr Jay Spaman (from Mar 2008)
Dr Mark Spamer – Adjunct (from June 2008)
Dr Melissa Starfield (July 07–Feb 08)
Dr Peter Stratton (from Feb 2007)
Dr Robert Sullivan (from June 2008)
Dr Alexander Sykes
Dr Huajin Tang (from Feb 2007)
Dr Narelle Tunstall – Adjunct (from Nov 2008)
Dr Matthew Ireland (from Feb 2008)
Dr Angela Boyce (from Nov 2007)
Dr David Lloyd (from Dec 2007)
Dr Eirinn Mackay (from Feb 2008)
Dr Matthew Pellekanos (Nov–Dec 2008)
Dr Qiang Shan (from Dec 2007)
Research Assistants

Suzanne Alexander – Adjunct (from Oct 2007)
Cameron Anderson (from Jan 2008)
John Baisden (from Nov 2007)
Jessica Barnes (from Mar 2008)
Debra Black
Daniel Bland (from April 2008)
Angela Boyce (from Nov 2007)
Tim Butler
Maria Caldeira (until May 2008)
Nissa Carrodus (Jan–May 2008)
Dr Preethi Eliy (Jan–Mar 2007)
Carla Evangelista (from July 2007)
Oliver Evans (from Apr 2008)
Briony Fox (until Feb 2007)
Kathryn French (from April 2008)
Damien Gardiner (until Jan 2007)
Clare Giacomantonio (until Feb 2008)
Ilan Gobius (July 08–Sep 08)
Justine Haddrell (from Jan 2008)
Carmen Haines (from Nov 2008)
Melanie Havler (from Feb 2008)
Candidates for Research Higher Degrees 2007 – 2008

**Noura Al-Menhali.** (Enrolled through SBMS) Role of p75NTR in BDNF-stimulated neurogenesis in the adult hippocampus. (Principal Advisor: Elizabeth Coulson) PhD awarded August 2008.

**Parthasarathy Bhagavatula.** (Enrolled at Australian National University) Visually guided flight in birds using budgerigar as a model system. (Principal Advisor: Mandyam Srinivasan)

**DanaKai Bradford.** The role of Netrin receptors in neural differentiation and migration in the adult mammalian brain. (Principal Advisor: Helen Cooper)

**Adrian Carter.** The ethical and philosophical implications of the neuroscience research on addiction and mental disorders. (Principal Advisor: Wayne Hall, School of Population Health)

**Kathleen Cato.** Overcoming therapy resistance in glioblastoma multiforme. (Principal Advisor: Angus Harding)

**Xuebin Chen.** (Enrolled through SBMS) Investigation into the molecular pharmacology of α1 and α3 glycine receptors. (Principal Advisor: Joe Lynch)

**Charlotte Clark.** Wnt-Ryk signalling in the establishment of major axon tracts in the embryonic mouse brain. (Principal Advisor: Helen Cooper)

**Lavinia Codd.** Neuroprotection and neurogenesis in the adult mouse brain after transient global cerebral ischaemia. (Principal Advisor: Perry Bartlett)


**Stacey Cole.** The role of the netrin receptor, Neogenin, in neuronal progenitor migration in the embryonic brain. (Principal Advisor: Helen Cooper)

**Melissa de Vries.** The role of the guidance receptor Neogenin in zebrafish neurulation. (Principal Advisor: Helen Cooper)

**Amber-Lee Donahoo (née Dawson).** Molecular mechanisms regulating the development of the corpus callosum. (Principal Advisor: Linda Richards)

**Michael Dwyer.** Charles Bonnet Syndrome: an investigation into the mechanisms underlying the development of visual hallucinations in acquired visual loss. (Principal Advisor: Jason Mattingley)

**Claire Foldi.** The effects of advanced paternal age on brain development and behaviour in the mouse. (Principal Advisor: Thomas Burne & Darryl Eyles)

**Clare Giacomantonio.** Self-organised wiring of the cerebral cortex. (Principal Advisor: Geoffrey Goodhill)

**Ilan Gobius.** The role of commissural plate glia, Slit and FGF protein family members in forebrain commissure formation. (Principal Advisor: Linda Richards)

**Lauren Harms.** The effect of developmental vitamin D deficiency on brain development and behaviour in the mouse. (Principal Advisor: Thomas Burne)

**Kristin Hatherley.** Modelling the formation of the neurosphere. (Principal Advisors: Rod Rietze & Pamela Burrage) MPhil awarded September 2008.

**Jonny Hunt.** Natural scene statistics and the architecture of the visual system. (Principal Advisor: Geoffrey Goodhill)

**Md Robiul Islam.** Identification and characterisation of new drugs targeting glycine receptor Cl- channels in pain sensory pathways. (Principal Advisor: Joe Lynch)
Sepideh Keshavarzi. Electrophysiology and morphological study of interneurons in lateral nucleus of amygdala. (Principal Advisor: Pankaj Sah)

Inge Laube. Neurochemical substrates of attention and attention deficit. (Principal Advisors: Mark Bellgrove & Jason Mattingley)

Tim Lynagh. Identifying the binding sites of analgesic lead compounds at the glycine receptor. (Principal Advisor: Joe Lynch)

Nancy Malintan. Role of Munc18-1 in neuroexocytosis. (Principal Advisor: Frederic Meunier)

Sharon Mason. The role of the Nuclear Factor I transcription factors NFI-A, NFI-B and NFI-X in regulating the development of the pallium. (Principal Advisor: Linda Richards)

Linda May. Synaptic activity and the p75I pan neurotrophin receptor death signalling pathway. (Principal Advisor: Elizabeth Coulson)

Richard Moore. Vision systems for autonomous aircraft guidance. (Principal Advisor: Mandyam Srinivasan)

Duncan Mortimer. A Bayesian model of axon guidance. (Principal Advisor: Geoffrey Goodhill)

Navid Nourani-Vatani. Biologically inspired robot navigation using vision. (Principal Advisor: Mandyam Srinivasan)


Sumiti Saharan. Modulation of adult mammalian neurogenesis by sirtuin proteins. (Principal Advisor: Perry Bartlett)

Hugh Simpson. Computational modelling of retinotectal map development. (Principal Advisor: Geoffrey Goodhill)

Ben Sivyer. Complex processing in the retina. (Principal Advisor: David Vaney)

Mark Stafford. (Enrolled through SBMS) Purinergic receptor-mediated signalling in neural stem cells and progenitor cells from the adult mouse subventricular zone. (Principal Advisors: David Adams & Perry Bartlett) PhD awarded September 2007.

Daniel Stjepanovic. Imaging the genetics of emotion and attention. (Principal Advisor: Mark Bellgrove)

Cornelia Strobel. Mechanisms of synaptic plasticity in intercalated cell masses of the amygdala. (Principal Advisor: Pankaj Sah)

Janette Thurley. Guidance mechanisms in the development of the caudal corpus callosum. (Principal Advisor: Linda Richards)

Clare Underwood. (Enrolled through SBMS) Regulation of p75 neurotrophin receptor-mediated neuronal death through post-translational modifications. (Principal Advisor: Elizabeth Coulson) PhD awarded June 2007.

Divya Unni. Role of Robo1 and DCC in Slit 2-induced repulsion in the corpus callosum. (Principal Advisor: Linda Richards)

Qian Wang. Investigating GABA\(_J\) receptor structure and function using voltage-clamp fluorometry. (Principal Advisor: Joe Lynch)

Nicola Watts. Neurogenesis in the adult amygdala and its modulation by fear conditioning. (Principal Advisors: Pankaj Sah & Perry Bartlett)

Peter Wen. (Enrolled through SBMS) Role and regulation of 3-phosphorylated phosphoinositides in neuroexocytosis. (Principal Advisor: Frederic Meunier)

Nikki Zuvela. SK2 channels: localisation and trafficking in central neurons. (Principal Advisor: Pankaj Sah)
PROFESSIONAL SERVICE

David Adams
Australian Physiological Society, President, 2005–10
Australian Academy of Science, National Committee for Biomedical Sciences, 2003–09
Multiple Sclerosis Research Australia, Research Management Council, 2007–09
Beijing Joint Conference of Physiological Sciences 2008, Honorary Advisory Committee
International Society for Autonomic Neuroscience, 2009 Sydney Congress, Local Organising Committee
NHMRC Project Grants Review Panel, 2007, 2008 (Deputy Chair)

Perry Bartlett
Motor Neurone Disease Research Institute of Australia, Research Committee, 1993–
SpinalCure Australia, Director and Scientific Chairman, 1995–
Human Frontier Science Program, Research Grant Review Committee, 2006–08
Australia 2020 Summit, Health Stream, 2008
NHMRC GRP Member & Assessor Selector, 2008
NHMRC Program Grant Review Panel B, 2008

Thomas Burne
Australasian Society for Psychiatric Research, Committee, Queensland Representative, 2008–
NHMRC Project Grants Review Panel, 2008

Helen Cooper
Australian and New Zealand Society for Cell and Developmental Biology, Secretary, 2007–08

Australian Neuroscience Society, Council, Queensland Representative, 2005–2008
Health Research Council of New Zealand, Biomedical Science Assessing Committee 1, 2008
NHMRC Project Grants Review Panel, 2007 (Deputy Chair)

Elizabeth Coulson
Friedreich Ataxia Research Association, Scientific Advisory Committee, 2006–

Ross Cunnington

Daryl Eyles
NHMRC Project Grants Review Panel, 2008

Louise Faber
NHMRC Project Grants Review Panel, 2008

John Kelly
National Collaborative Research Infrastructure Scheme, Sub-Facilitator Imaging, 2006–07
National Imaging Facility, Queensland Delegate, 2007–

Joe Lynch
Australian Physiological Society, Secretary, 2007–
Australian Course in Advanced Neuroscience, Faculty Member, 2005–
NHMRC Project Grants Review Panel, 2007

Jason Mattingley
Australian Academy of Science, National Committee for Brain and Mind, 2008–
NHMRC Project Grants Review Panel, 2008

John McGrath
Global Burden of Disease Study, Mental Disorders and Illicit Drugs Expert Group, 2007–
Melbourne Neuropsychiatry Centre, Scientific Advisory Board, 2008–
Centre for Clinical Research in Neuropsychiatry, University of Western Australia, Scientific Advisory Board, 2008–
NHMRC Project Grants Assessor Selector, 2008
NHMRC National Health Committee, Expert Health Panel, 2008

Frederic Meunier
Australian and New Zealand Society for Cell and Developmental Biology, Committee, Queensland Representative, 2007–
IBRO Satellite Meeting on The Secretory Vesicle Cycle, Brisbane, July 2007, Co-Organiser

Linda Richards
Australian Brain Bee Challenge, National Chair, 2006–
National Association of Research Fellows of NHMRC, Queensland Representative, 2006–
American Association of Anatomists, CJ Herrick Award Committee, Chair, 2007
IBRO Satellite Meeting on Neurodevelopment, Cairns, July 2007, Co-Organiser
NHMRC Project Grants Review Panel, 2008

Judith Reinhard
Australasian Association for Chemosensory Science, Committee, 2005–
10th AACSS Meeting, Brisbane, Organiser, 2008
Two of the leading scientific societies in Australia are now presided over by QBI Faculty members. Professor David Vaney is the President of the Australian Neuroscience Society (ANS) for 2008–2009, having served as President-Elect in 2007. David is the first Queensland scientist to be elected as ANS President, although Perry Bartlett served as ANS President from 2000–2001 before his move to Brisbane.

Professor David Adams was reappointed as President of the Australian Physiological Society (AuPS) for 2009–2010, having held the position since 2005. The two Davids have facilitated the organisation of a joint meeting of the two societies in Sydney in January 2010, which will mark the 50th Anniversary of AuPS and the 30th Annual Meeting of ANS.

### QBI Professors Head National Scientific Societies

**Pankaj Sah**
- Addiction Neuroscience Network Australia, Scientific Advisory Board, 2005–
- Australian Course in Advanced Neuroscience, Faculty Member, 2005–
- Neurosciences Australia, Integrative Neuroscience Facility, Scientific Advisory Committee, 2003–
- NHMRC Career Development Awards Biomedical Panel, 2007, 2008 (Chair)
- NHMRC Project Grants Review Panel, 2008

**Mandyam Srinivasan**
- Prime Minister’s Science, Engineering and Innovation Council, 2006–2007
- PMSEIC Working Group on Water for Our Cities, Deputy Chair, 2007
- Australian Academy of Science, Sectional Committee on Applied Physical and Engineering Sciences, 2008–
- ARC Network for Intelligent Signal Sensors & Information Processing, Advisory Board, 2006–

**David Vaney**
- Australian Neuroscience Society, President-Elect, 2007; President 2008–2009
- International Brain Research Organization, Governing Council, 2008–09
- Federation of Asian and Oceanian Neuroscience Societies, Council, 2008–09
- Neurosciences Australia, Non-Voting Board Member, 2008–2009
- National Vision Research Institute, Board of Administration, 2008–
- Australian Course in Advanced Neuroscience, Management Committee, 2007–
- IBRO Satellite Meeting on Visual Neuroscience, Cairns, July 2007, Organiser
- NHMRC GRP Member & Assessor Selector, 2008

**Robyn Wallace**
- NHMRC Scholarships Medical Panel, 2008
Acta Psychiatrica Scandinavica
John McGrath
Editorial Advisory Board

Advances in Artificial Neural Systems
Mandyam Srinivasan
Editorial Board

Australian and New Zealand Journal of Psychiatry
John McGrath
International Advisory Board

BMC Biology Image Library
Jason Mattingley
Section Editor for Behavioral & Cognitive Neuroscience

BMC Physiology
Pankaj Sah
Editorial Board

BMC Psychiatry
Darryl Eyles, John McGrath
Editorial Board

Brain and Cognition
Jason Mattingley
Editorial Board

Clinical Schizophrenia & Related Psychoses
John McGrath
Editorial Board

Cognitive Neuropsychiatry
John McGrath
Editorial Board

Cortex
Jason Mattingley
Associate Editor

Developmental Dynamics
Linda Richards
Editorial Board

Faculty of 1000 Biology
David Adams
Faculty Member

Frontiers in Cellular Neuroscience
Louise Faber
Review Editor

Hippocampus
Pankaj Sah
Editorial Board

International Journal of Developmental Neuroscience
Perry Bartlett
International Editorial Board

Journal of Applied Clinical Pediatrics
Robyn Wallace
Editorial Board

Journal of Attention Disorders
Mark Bellgrove
Editorial Board

Journal of Comparative Neurology
David Vaney
Editorial Board

Journal of Comparative Physiology A
Mandyam Srinivasan
Editorial Advisory Board

Journal of Insect Physiology
Mandyam Srinivasan
Editorial Board

Journal of Neurochemistry
Frederic Meunier
Handling Editor & Reviews Handling Editor

Journal of Neurophysiology
Pankaj Sah
Associate Editor

Journal of Neuroscience
Pankaj Sah
Associate Editor, Cellular/Molecular

Journal of Neuroscience Research
Perry Bartlett
Editorial Board

Network – Computation in Neural Systems
Geoffrey Goodhill
Editor-in-Chief

Neural Development
Perry Bartlett
Editorial Board

Neural Plasticity
Pankaj Sah
Editorial Board

Neurocase
Jason Mattingley
Editorial Board

Neuropsychologia
Jason Mattingley
Editorial Advisory Board

Neuroscience Letters
Pankaj Sah
Associate Editor

Neuroscience Research
Perry Bartlett
Editorial Board

Neurosignals
Perry Bartlett
Editorial Board

Open Evolution Journal
Charles Claudianos
Editorial Advisory Board

Pflügers Archiv – European Journal of Physiology
David Adams
Editorial Advisory Board

PLoS Biology
Mandyam Srinivasan
Editorial Board

PLoS ONE
Thomas Burne, Frederic Meunier
Editorial Board

Revista Brasileira de Psiquiatria
John McGrath
International Editorial Board

Schizophrenia Bulletin
John McGrath
Editorial Board

Schizophrenia Research
John McGrath
Editorial Board

Stem Cell Research
Perry Bartlett
Editorial Board

Yonsei Medical Journal
Perry Bartlett
Editorial Advisory Board
**UQ APPOINTMENTS**

**David Adams**  
Chair of Physiology, 1995–2009  
Head, School of Biomedical Sciences, 2000–07  
Director, UQ Neuroscience Program, 2008–09  
UQ Academic Board Standing Committee, 2008–09

**Perry Bartlett**  
Foundation Chair of Molecular Neuroscience, 2002–  
Chair, UQ Animal Ethics Committee, 2008  
Chair, BACS Local Confirmation and Promotions Committee, 2008

**Mark Bellgrove**  
Associate Professor, School of Psychology, 2007–

**Helen Cooper**  
Associate Professor, School of Biomedical Sciences, 2002–  
UQ Research Academic Promotions Committee (Levels A-D), 2006–07  
UQ Institutional Biosafety Committee, 2008–

**Elizabeth Coulson**  
UQ Research Higher Degree Committee, 2007–

**Ross Cunnington**  
Associate Professor, School of Psychology, 2007–

**Ian Duncan**  
UQ Information Technology Consultative Group, 2005–

**Louise Faber**  
UQ Biological Sciences Library Advisory Committee, 2007–08

**Geoffrey Goodhill**  
Associate Professor, School of Mathematics and Physics, 2005–

**John Kelly**  
UQ Research Services Review Committee, Secretary, 2007  
UQ Biological Resources Steering Committee, 2007–  
UQ Administrative Services External Audit, 2008–  
UQ INSIGHT Steering Committee, 2008–  
UQ CRM Project Steering Committee, 2008–

**Joe Lynch**  
Associate Professor, School of Biomedical Sciences, 2003–08  
Professor, School of Biomedical Sciences, 2008–  
Coordinator, UQ Masters of Neuroscience Program, 2008–

**Jason Mattingley**  
Foundation Chair of Cognitive Neuroscience, 2007–

**John McGrath**  
Director, Epidemiology and Developmental Neurobiology, Queensland Centre for Mental Health Research, 1999–  
Professor, Discipline of Psychiatry, School of Medicine, 2001–  
Professorial Affiliate, Queensland Brain Institute, 2005–

**Frederic Meunier**  
Associate Professor, School of Biomedical Sciences, 2007–

**Linda Richards**  
Associate Professor, School of Biomedical Sciences, 2005–  
Chair, UQ Biological Resources Animal Users Advisory Committee, 2007–

**David Reutens**  
Foundation Chair of Experimental Neurology, 2008–

**Pankaj Sah**  
Professor, Queensland Brain Institute, 2003–  
UQ Academic Board, 2005–07  
UQ Research Committee, 2005–  
UQ Library Committee, 2009–

**Mandyam Srinivasan**  
Professor of Visual Neuroscience, Queensland Brain Institute and School of Information Technology & Electrical Engineering, 2007–

**David Vaney**  
Professorial Research Fellow, School of Biomedical Sciences, 2002–07  
Professorial Research Fellow, Queensland Brain Institute, 2008–

**Robyn Wallace**  
Anatomical Biosciences Animal Ethics Committee, 2006
OPERATIONS STAFF

Director of Operations
John Kelly

Institute Manager
Helen Weir

Institute Operations Manager
Ian Duncan

Senior Personal Assistant to the Director
Deirdre Wilson (from Mar 2008)
Veronica Baldry (until Mar 2008)

Projects and Development
Alison van Niekerk, Manager: Projects and Executive Support
Jenny Valentine, Development and Community Relations (from April 2007)

Research Management
Rowan Tweedale, Senior Research Manager
Dr Sylvie Pichelin, Grants and Postgraduate Student Coordinator (from April 2007)

Commercialisation
Annita Nugent, Manager: Innovation and Commercial Development

Laboratory Support
Clare Seaman, Scientific Services Manager
Judy Bracefield (from Sep 2007)
Jane Ellis (from Feb 2008)
Colin Macquien (from Oct 2007)
Virginia Nink
Lida Stjepcevic
Nana Sunn (from Jan 2008)
Mary White
Janette Zlamal (from July 2007)

Occupational Health & Safety
Dr Paul Lovelock, OH&S Manager (from May 2007)

Communications and Information Technology
Ronald Hohenhaus, Communications Manager
Ian Glidden (from Jan 2008)
Jake Carroll, Senior Systems Programmer
Phillip George
Toby O’Brien (from July 2007)

Technical Services
David Wheeldon, Technical Services Manager
Adam Barry (from Mar 2007)
Trent Bell (from Jul 2008)

Finance Section and Store
Katherine Parsonage, Finance Manager
Wade Ebeling
Michael Perren (from Aug 2007)
Elizabeth Power (from Apr 2008)
Nathan Weir (from Sep 2008)
Jason White (from Mar 2008)

Administration Support
Brenda Campbell (from Aug 2008)
Amy Cook (from Mar 2008)
Shani Doig (until May 2007)
Susan Earnshaw (from Apr 2008)
Phillip Harris (Sep 07–Sep 08)
Millie Lin (Sep 07–Jan 08)
Rhonda Lyons (from Mar 2008)
Debra Mc Murtrie (from Oct 2007)
Charmaine Paiva
Jacqueline Perren (from Mar 2008)
Lauren Sheraton (from Nov 2008)

From left: John Kelly, Jacqueline Perren, Helen Weir, Deirdre Wilson, Alison van Niekerk, Annita Nugent, Debra McMurtrie, Sylvie Pichelin
Institute Manager
Helen Weir manages the Institute’s administrative functions, including human resources, finance, events, communications, and students.

Institute Operations Manager
Ian Duncan manages the Institute’s operational functions, including scientific services, technical services, ICT and OH&S.

Projects and Executive Support
Alison van Niekerk and Deirdre Wilson coordinate specific projects including government and international relations and provide administrative support for the Executive.

Development and Community Relations
Jenny Valentine coordinates the activities of the QBI Development Board, develops relationships with community groups, and is responsible for the Institute’s donors and sponsors program.

Senior Research Manager
Rowan Tweedale coordinates the development of research grant applications and helps edit the scientific manuscripts of QBI researchers.

Grants and Postgraduate Student Coordinator
Dr Sylvie Pichelin coordinates the submission and management of research grants and is also responsible for student administration.

Innovation and Commercial Development Manager
Annita Nugent coordinates the commercial activities of QBI through UniQuest (UQ’s commercialisation company) and the QBI Executive.

Scientific Services Manager
Clare Seaman coordinates the laboratory support facilities of the Institute, including imaging, histology and media, and is responsible for the acquisition and operation of major equipment.

Occupational Health & Safety
Dr Paul Lovelock is responsible for all health and safety aspects of the Institute, including OGTR and AQIS.

Communications Manager
Ron Hohenhaus is responsible for QBI’s internal and external communications and for media relationships.

Technical Services Manager
Dave Wheeldon coordinates the workshop facilities, ensures the building is properly maintained, and is responsible for the building management systems.

Finance Manager
Katherine Parsonage manages the Institute’s financial services and provides budget support to the Executive.

Rhonda Lyons, the national administrator for the Australian Brain Bee Challenge, organises materials for participating schools.

From left: Wade Ebeling, Elizabeth Power, Katherine Parsonage, Michael Perren, Nathan Weir, Susan Earnshaw, Rezaa Nazer, Charmaine Paiva
The Queensland Brain Institute took possession of its new $63 million building in September 2007. After testing the support systems in October 2007, 20 research groups from seven geographic locations were moved into the new facilities. The first experiments were conducted a week after the move – a credit both to the careful planning and to the good will of everyone involved.

The seven-storey building was designed to accommodate 250 researchers and to house their state-of-the-art equipment. The design team of John Wardle Architects and Wilson Architects combined with the builders, Watpac and the UQ Property & Facilities Division, were able to deliver the project on time and within budget – an unusual achievement for the construction industry in recent times.

The QBI building was modelled on the theme of A Theatre for Research, with informal interaction spaces opening to the imposing four-storey foyer on one side and to the research laboratories on the other side, thus show-casing QBI’s research environment. QBI staff and visitors come together on Level 7, which includes a 200-seat auditorium, a 40-seat seminar room, and interaction areas that can accommodate 300 people.

Research spaces have been designed for maximum flexibility; they can be configured to support molecular biology, tissue culture, electrophysiology and even robotics. On each of the four research floors, a core facility has been placed to one side, with both direct access to the main research labs and external access for affiliate researchers. These includes core facilities for flow cytometry (Level 6), advanced microscopy (Level 5), histochemistry (Level 4) and cognitive testing (Level 3). Another feature of the building is a dedicated SPF animal facility on Level 2, which includes surgeries, a behavioural-testing suite, aquaria and a micro-ultrasound facility.

Scientific officer Janette Zlamal manages the core facility for media preparation and quality control.
Microscopy Core Facility
The microscopy facility, which is managed by Luke Hammond and Colin Macqueen, is built around nine high-end Zeiss instruments. Six epifluorescence microscopes, both upright and inverted, are capable of automated imaging, optical sectioning, and tiled imaging of large areas. Three confocal systems allow high-resolution imaging (LSM510 META), high-speed live imaging (LSM 5 LIVE) and immersion microscopy (LSM PASCAL). On several systems, controlled-temperature atmospheric incubation supports live imaging of cells and tissues.

Researchers also have access to the Zeiss Imaging Suite, which is housed within QBI. This centre provides researchers with the latest Zeiss instruments and software, keeping them abreast of current advances in microscopy.

Training is provided to researchers in the use of all instruments, including advice on experimental design and advanced imaging techniques. The combination of outstanding equipment and expert personnel means that QBI researchers have the tools to undertake studies as diverse as the 3-D reconstruction of brain sections and the rapid imaging of neuronal outgrowth and intracellular trafficking.

Micro-Ultrasound Core Facility
The micro-ultrasound facility, which is managed by Nana Sunn, uses the same real-time imaging technology that is used to monitor the development of a human foetus. The high-frequency high-resolution preclinical imaging is applicable for research on a variety of small animals, including rodents, chick embryos, zebrafish and honeybees. This safe non-invasive imaging technique permits longitudinal studies of live animals from early implantation of the embryo to adulthood.

The facility offers a wide range of specialised imaging services for pregnancy status, embryonic implantations and development, embryonic lethality, as well as in vivo and in utero visualisation of anatomical regions. In addition, intervention studies or therapies are possible with ultrasound-guided microinjection of DNA plasmids, siRNA, lentivirus, stem cells, drugs, proteins, tract tracers, and fluorescent beads, as well as extraction procedures such as amniocentesis.

Histochemistry Core Facility
The histochemistry facility, which is managed by Jane Ellis, provides expert advice and support to QBI researchers in elucidating the structure, function and pathology of the brain and nervous system in normal and disease states. Histology relies on preserving animal tissues and processing this material to produce stained slides for microscope analysis.

The facility is well equipped to process tissues for a variety of staining techniques, including histochemistry, immunocytochemistry and electron microscopy. Currently the facility provides access to equipment for processing and sectioning frozen, paraffin-embedded and resin-embedded tissues. It also provides researchers with access to a Leica sledge microtome and cryostat, rotary microtomes for paraffin sectioning, vibrating microtomes, and an ultramicrotome. Other associated equipment includes a paraffin processor, an embedding centre and a multi-head teaching microscope.


Jane Ellis cuts frozen brain sections on the cryostat in the histochemistry core facility.
Since its foundation, the Queensland Brain Institute has played a leadership role in coordinating the Neuroscience Seminar Program, which is designed to promote excellence through the exchange of ideas, establishing new collaborations, and augmenting existing partnerships.

In 2007 and 2008, this program was instrumental in attracting 22 interstate scientists and 39 overseas scientists from 9 countries to share their neuroscience knowledge and experience with the UQ neuroscience community.

The program also provided the opportunity for 28 Queensland neuroscientists, both at QBI and other institutions, to showcase their research and ideas to their colleagues.

Cliff Abraham (University of Otago, Dunedin, New Zealand) Mechanisms of hippocampal LTP and LTD in vivo. 7 March 2007
Cliff Abraham (University of Otago, Dunedin, New Zealand) Advanced Neuroscience Lecture Series. The search for the engram: mechanisms of learning and memory. 30 April 2008
David Adams (Queensland Brain Institute) Analgesic conotoxins targeting neuronal nicotinic receptors or GABA-B receptors? 3 September 2008
Dana Ballard (University of Texas, Austin TX, USA) Distributed synchrony: a model of cortical signalling. 12 March 2008
Andrew Barron (Macquarie University, Sydney) Cocaine and the honey bee brain. 3 October 2008
Daniel Blackmore (Queensland Brain Institute) Physical exercise stimulates resident stem cells and augments the regenerative capacity of the ageing brain. 28 November 2007
Ian Blair (ANZAC Research Institute, Sydney) Molecular genetic insights into ALS. 27 June 2008
Timothy Bredy (Semel Institute for Neuroscience and Human Behavior, Los Angeles CA, USA) Epigenetic mechanisms associated with the extinction of conditioned fear. 30 October 2008
Heinrich Bülthoff (Max Planck Institute for Biological Cybernetics, Tübingen, Germany) An image-based approach to perception and action. 16 July 2007
Charles Claudianos (Queensland Brain Institute) Neurexin and neuroligin in the synapse: fundamental roles in brain development, learning and memory. 4 June 2008
Helen Cooper (Queensland Brain Institute) The multi-talented receptor Neogenin regulates key events in neural development. 6 August 2008
Elizabeth Coulson (Queensland Brain Institute) Alzheimer’s disease peptide Aβ1-42 acts through the p75 neurotrophin receptor to induce cell death and neurogenesis. 14 November 2007
Ross Cunnington (Queensland Brain Institute) The mirror system and the perception of actions and gestures. 13 June 2007
Marien DeBruijne (Monash University, Melbourne) Encoding a chemical world: behavioural and neuronal responses to odours in Drosophila. 2 April 2008
Andrew Delaney (Queensland Brain Institute) Noradrenaline inactivates release sites at a glutamatergic basket synapse in the central amygdala: presynaptic inhibition via a change in N. 18 June 2008
Derek Denton, (Howard Florey Institute, Melbourne) The evolutionary emergence of consciousness. 21 April 2008
Yu-Qiang Ding (Institute of Neuroscience, Shanghai, China) Transcription factor Lmx1b: from development to brain functions. 9 May 2007
Mike Dragunow (University of Auckland, New Zealand) The life and death of human brain cells. 20 February 2008
Anna Dunaevsky (Brown University, Providence RI, USA) Neurons and glia take shape in development, disease and learning. 29 August 2008
Sally Dunwoodie (University of New South Wales, Sydney) Notch signalling, somitogenesis and abnormal vertebral segmentation. 1 October 2008
Darryl Eyles (Queensland Brain Institute) Translating schizophrenia epidemiology into neuroscience: developmental vitamin D deficiency and advanced paternal age. 30 July 2008
Eyleen Goh (John Hopkins University, Baltimore MD, USA) Novel approaches for functional regeneration in the adult central nervous system. 26 April 2007

Bruce Graham (University of Stirling, Scotland) Modelling short term plasticity and information transmission at an auditory synapse. 12 February 2008

Mary Hayhoe (University of Texas, Austin TX, USA) Factors controlling allocation of gaze in natural, dynamic environments. 12 March 2008

Fritjof Helmchen (University of Zurich, Switzerland) New imaging modalities for studying cellular and network computations in vivo. 18 July 2007

Robert Hester (Queensland Brain Institute) Executive dysfunction in drug dependence. 17 September 2008

Massimo Hilliard (Rockefeller University, New York NY, USA) Wnt signals and Frizzled activity orient anterior-posterior neuronal polarity and axonal outgrowth in C elegans. 21 February 2007

John Horn (University of Pittsburgh School of Medicine, Pittsburgh PA, USA) Synaptic amplification in sympathetic ganglia: a building block for homeostatic control. 28 June 2007

Fahmeed Hyder (Yale University, New Haven CT, USA) Energetics of neural signaling and fMRI activity. 4 August 2008

Anthony Isles (University of Cardiff, Wales) RNA-editing and serotonin 2C function in a mouse model for Prader-Willi syndrome. 19 September 2008

Terrance Johns (Ludwig Institute for Cancer Research, Melbourne) Glioma: therapeutic antibodies targeting key signalling pathways. 26 March 2008

Marc Kamke (Queensland Brain Institute) Cholinergic modulation of lesion-induced plasticity in mature sensory cortex. 26 September 2007

Damien Keating (Flinders University of South Australia, Adelaide) Identifying novel regulators of cell communication as potential linkers to neurodegeneration. 22 September 2008

Trevor Kilpatrick (University of Melbourne, Victoria) Exploring frontiers in multiple sclerosis research. 2 May 2007

Simon Koblar (University of Adelaide, South Australia) Human dental pulp stem cells: the tooth fairy and brain repair? 21 May 2008

Kyoko Koshibu (Queensland Brain Institute) PP1-mediated histone coding: a mechanism underlining memory. 3 October 2007

Leah Krubitzer (University of California, Davis CA, USA) How does evolution build a complex brain? 18 August 2008

Cath Latham (Institute for Molecular Bioscience, UQ) Munc-y business in membrane fusion: deciphering Munc18/SNARE interactions regulating exocytosis. 23 July 2008

Rob Lewis (Monash University, Melbourne) Detailed real-time images of live subjects using medical imaging beamlines. 14 May 2008

Terrance Johns (Ludwig Institute for Cancer Research, Melbourne) Glioma: therapeutic antibodies targeting key signalling pathways. 26 March 2008

Justina Marshall (School of Biomedical Sciences, UQ) Colour, sex and violence: a day on the reef. 1 August 2007

John Mattick (Institute for Molecular Biosciences, UQ) RNA control of brain development, function and memory. 19 September 2007


Randal Moldrich (Queensland Brain Institute) The neurophenotype of Down syndrome. 22 August 2007

Mark Murphy (University of Melbourne, Victoria) Genetics and memory of stress, fear and anxiety. 29 October 2008

David O’Carroll (University of Adelaide, South Australia) Detection of moving objects and features by a small brain. 15 October 2008

Geoff Osborne (Queensland Brain Institute) Positional sorting and other recent advances in flow cytometry. 9 April 2008

Peregrine Osborne (University of Sydney, NSW) TRPV1 inhibition by estrogen and the GDNF family ligand artemin in somatosensory nociceptors. 9 July 2008

Tracie Paine (Harvard Medical School, Cambridge MA, USA) Attenuation of cortical PKA disrupts attention and stimulates locomotor activity in rodent models. 12 November 2008
Giles Plant (University of Western Australia, Perth) Autologous human cell transplants for spinal injury repair. 28 February 2007

Marcus Rattray (King’s College, London, England) Glutamate transporters and excitotoxicity in ALS. 18 January 2007

Judith Reinhard (Queensland Brain Institute) Making sense of scents: how bees detect, process and learn complex odours. 2 July 2008

Adina Roskies (Dartmouth College, Hanover NH, USA) Decision-making and freedom. 23 May 2007

Mark Schira (University of New South Wales, Sydney) 40 years of mystery: how the fovea is represented on human visual cortex. 19 November 2008

Ethan Scott (School of Biomedical Sciences, UQ) Observation and manipulation of neural circuits using Gal4 enhancer trapping in zebrafish. 5 November 2008

Floyd Qiang Shan (Queensland Brain Institute) Ca2+ stimulated type 1 adenylyl cyclase is essential for the remote memory maintenance. 27 February 2008

Tatyana Sharpee (Salk Institute, San Diego CA, USA) From noise to natural scenes: optimization principles for adaptation in visual cortex. 27 June 2007

Hartwig Siebner, (University of Kiel, Germany) Brain imaging of neurogenetics in Parkinson’s disease. 11 June 2008

Horst Simon (University of Heidelberg, Germany) Differentiation, maintenance and survival of mesencephalic dopaminergic neurons: a route to the mechanisms of Parkinson’s disease? 17 May 2007

Philip Smith (University of Melbourne, Victoria) An integrated theory of attention and decision making in visual signal detection. 17 July 2008

Russell Snell (ViaLactia Biosciences, Auckland, New Zealand) Neurodegenerative diseases: models and genetics. 16 May 2007

Nelson Spruston (Northwestern University, Chicago IL, USA) Synapse distribution and dendritic excitability optimize integration and plasticity in hippocampal pyramidal neurons. 29 April 2008

Mandyam Srivinvasan (Queensland Brain Institute) Bees and robots: recent progress and current challenges. 24 October 2007

Mandyam Srivinvasan (Queensland Brain Institute) Advanced Neuroscience Lecture Series. The honeybee as a model for the study of vision, navigation, learning, memory and ‘cognition’. 21 May 2008

Greg Stuart (Australian National University, Canberra) Advanced Neuroscience Lecture Series. Cortical function. 28 May 2008

Nana Sunn (Queensland Brain Institute) From implantations to adulthood: applications of in vivo ultrasound biomicroscopy technology in mice. 27 March 2008

Jeremy Turnbull (University of Liverpool, England) Decoding heparan sulphate function in neural development and neurodegeneration. 21 October 2008

David Vaney (Queensland Brain Institute) Visual processing in the inner retina: timing is everything. 15 July 2008

Bruno van Swinderen (Neuroscience Institute, San Diego CA, USA) Neurophysiology of attention-like processes in Drosophila. 14 February 2007

Bruno van Swinderen (Queensland Brain Institute) Advanced Neuroscience Lecture Series. Learning from flies! 7 May 2008

Jane Visvader (Walter and Eliza Hall Institute of Medical Research, Melbourne) Mammary stem cells in development and cancer. 6 June 2007

Michael Walker (University of Auckland, New Zealand) Animal magnetism: structure, function and use of the magnetic sense in animals. 22 October 2008

Charles Watson (Curtin University of Technology, Sydney) Genetic neuroanatomy: from ontogeny to ontology. 19 August 2008

Cyndi Weickert (University of New South Wales, Sydney) Neuregulin, neurogenesis and neuropsychiatry. 30 May 2007

David Willshaw (University of Edinburgh, Scotland) Measuring and modelling maps of retinocollricular connections. 6 February 2008

Francois Windels (Queensland Brain Institute) Aversive stimuli reset membrane potential oscillations in the amygdala. 31 October 2007

Zhi-Qi Xiong (Institute of Neuroscience, Shanghai, China) Regulation of dendritic growth and synaptic plasticity by transducers of regulated CREB (TORCs). 28 August 2007

Yuchio Yanagawa (Gunma University School of Medicine, Japan) Transgenic rodents and the study of GABAergic neurons. 19 July 2007

Benjamin Yee (Neuroscience Centre, ETH, Zurich, Switzerland) Modulation of cognitive function via glycine transporter 1: relevance to schizophrenia. 26 November 2008

Ling Yiu (Vanderbilt University, Nashville TN, USA) Transcription factors and cell fate specification. 21 June 2007

Andrea Yool (University of Adelaide, South Australia) Role of Aquaporin-1 as a dual water and ion channel. 27 August 2008

Shingo Yoshikawa, (Salk Institute, San Diego CA, USA) Whnt and axon guidance. 21 March 2007

Angela Yu (Princeton University, Princeton NJ, USA) Seeing patterns in randomness: irrational superstition or adaptive behaviour? 11 February 2008

James Zheng (UMDNJ-Robert Wood Johnson Medical School, Piscataway NJ, USA) Spatial signalling and directional control of growth cones during axon guidance. 24 July 2007
In 2007–2008 the Queensland Brain Institute received major research funding from the National Health & Medical Research Council, the Australian Research Council, the Queensland State Government, and the University of Queensland. External research grants that commenced in 2007–2008 are listed below, along with older grants to new researchers who transferred their grants to QBI in 2007–2008. Grants awarded to QBI that commenced in 2003–2006 but continued in 2007–2008 are listed in the 2003–2006 QBI Report. QBI researchers are shown in bold.

**Australian Research Council (ARC) Discovery Projects**


Hester R (2005–2007) Examining the relationship between error processing, cognitive control and emotion: a cognitive neuroscience approach. $267,000 (Transferred to QBI in 2007)

Lynch J (2007–2009) Molecular structure and function of the glycine receptor. $263,000


**ARC Centre of Excellence**

Srinivasan M (2005–2010) ARC Centre of Excellence in Vision Science. $1,000,000,000 (Transferred to QBI in 2007)


**ARC Linkage Infrastructure Equipment & Facilities (LIEF)**


**ARC Linkage Project**


**Australian Stem Cell Centre**


**CSIRO Flagship Collaboration Research Fund**

RESEARCH GRANTS

Fondation Jérôme Lejeune

Garnett Passe & Rodney Williams Memorial Foundation
Kamke M (2008–2011) The role of cross-sensory brain plasticity and attention in limiting the benefits of cochlear prostheses. $263,450

Human Frontier Science Program

Motor Neurone Disease Research Institute of Australia

Multiple Sclerosis Society of Queensland
Bartlett P (2008–2010) Funding for MS Research Fellowship. $75,000

NARSAD Young Investigator Awards

Faber ESL (2008–2010) Functions of SK channels in the medial prefrontal cortex. $60,000

National Health & Medical Research Council (NHMRC) Project Grants
Cunnington R, Mattingley J, Williams MA (2008–2010) Selective attention and the processing of observed actions. $326,250
Eyles D, Burne T, Mackay-Sim A, McGrath J (2007–2009) The developmental vitamin D model (DVD) as an animal model for schizophrenia. $247,589
Faber ESL (2008–2010) Modulation and trafficking of SK channels in the lateral amygdala. $250,500

Meunier F, Wei M, Lavdis N (2007–2009) Gene therapy to cure botulinum toxin intoxication and new motoneuron delivery system. $375,000
Richards L (2007–2009) The role of Netrin-DCC in the development of the corpus callosum. $492,000
Sah P, Delaney A (2008–2010) NMDA receptor function in the amygdala. $345,000

NHMRC Career Development Award (Biomedical)

NHMRC Enabling Grant

NHMRC Equipment Grants
Bartlett P (2008) AKTA Explorer fast protein liquid chromatography equipment. $88,932
Faber ESL (2007) Electrophysiology rig for probing the neural circuitry of the brain. $51,000

ARC Centre of Excellence in Vision Science
Since 2008, QBI has hosted the Queensland node of the ARC Centre of Excellence in Vision Science (ACEVS). The Centre, which is based at the Australian National University, has 16 chief investigators located in four nodes at the ANU, the University of Sydney, the University of Queensland and the University of Western Australia. The UQ Chief investigators are Professor Mandyam Srinivasan and Professor David Vaney, who study invertebrate vision and mammalian vision, respectively. ACEVS has three major research themes: Vision for living, Vision for action and robotics, and Vision for life.

NHMRC Equipment Grants
Bartlett P (2008) AKTA Explorer fast protein liquid chromatography equipment. $88,932
Faber ESL (2007) Electrophysiology rig for probing the neural circuitry of the brain. $51,000
NHMRC Program Grant

NHMRC Public Health Postgraduate Research Scholarship

NHMRC Research Fellowships

NHMRC Training Fellowships
Silk T (2007–2009) Australian Clinical Research Fellowship. A fMRI investigation of the prefrontal- striatal-parietal brain function in attention deficit hyperactivity disorder. $269,000

National Institute of Neurological Disorders and Stroke (NINDS)

Queensland Cancer Fund

Queensland State Government – Dept of Tourism, Regional Development and Industry
Bartlett P (2008–2010) Operational Funding for the Queensland Brain Institute. $27,500,000

Queensland State Government – National Collaborative Research Infrastructure Strategy (NCRIS)

Queensland State Government – Innovation Skills Fund
Cui X (2008–2011) Smart State Fellowship: How does developmental vitamin D deficiency alter dopaminergic signalling in adulthood? $150,000
Power J (2007–2010) Smart State Fellowship: Calcium dynamics in amygdala neurons. $225,000
Srinivasan M (2007–2012) Smart State Premier’s Fellowship: From small brains to novel aerospace technology. $1,250,000

US Air Force Office of Scientific Research
Srinivasan M (2007–2009) Target tracking and interception by aggressive honeybees. $225,000

US Army Research Office

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<th>Granting Agency</th>
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<td>National Health &amp; Medical Research Council</td>
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<td>Commonwealth Government: Department of Health &amp; Ageing</td>
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<td>Commonwealth Scientific &amp; Industrial Research Organisation</td>
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<td>Garnett Passe &amp; Rodney Williams Memorial Foundation</td>
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<td>Multiple Sclerosis Society</td>
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<td>National Alliance for Research on Schizophrenia &amp; Depression</td>
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<td>Queensland Government – Smart State Scheme</td>
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<td>Queensland Government – Queensland Health</td>
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<td>Ramaciotti Foundation</td>
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<td>Ross Maclean Fellowship</td>
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<td>SpinalCure Australia</td>
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<td>US Army Research Office</td>
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<td>Total</td>
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Power J (2007–2010) Smart State Fellowship: Calcium dynamics in amygdala neurons. $225,000
Srinivasan M (2007–2012) Smart State Premier’s Fellowship: From small brains to novel aerospace technology. $1,250,000

Srinivasan M (2007–2009) Target tracking and interception by aggressive honeybees. $225,000

US Army Research Office
Papers available online in 2008 ahead of print publication in 2009 will be listed in the 2009–2010 QBI Report.


mediated by a phenylalanine residue.

The binding site of the glycine receptor is interaction in the


QBI has an active outreach program that focuses on taking neuroscience to the wider community and making information on current research accessible to the public. QBI scientists regularly give talks about their work to various community organisations, business groups, hospitals and support groups throughout Queensland. In addition, scientists and other staff provide guided tours of the QBI facilities. More than 30 outreach events take place each year.

**Cool Night Classic**

PricewaterhouseCoopers (PwC) staged a fun run called the Cool Night Classic on 31 October 2007. Proceeds from the event were donated to two worthy causes: the Down Syndrome Association of Queensland and the Queensland Brain Institute. 4,500 runners participated in the Cool Night Classic, which followed a 5.75 km course through inner Brisbane finishing at the Riverside Centre.

About 30 staff from QBI staff helped a small army of volunteers ensure that the event ran smoothly and safely, acting as marshals and water-station attendants. After the event, the Managing Director of PwC, Mr Rob Baker, presented cheques for $22,000 to QBI and the Down Syndrome Association – a fantastic outcome and much appreciated!

**Motor Neuron Disease Awareness Week**

On 10 May 2008, the Motor Neuron Disease Association of Queensland and the Queensland Brain Institute hosted a public lecture at QBI to mark Motor Neuron Disease Awareness Week. The event began with a tour of the Peter Goodenough and Wantoks Research Laboratory, which is QBI’s laboratory devoted to research on motor neuron disease. This was followed by a presentation from Dr Robyn Wallace, who spoke about her research on the genetics of motor neuron disease.

**The Brisbane Institute**

On 28 October 2008, Professor Jason Mattingley gave an address to the Brisbane Institute, which is a Queensland forum for innovative information, ideas and solutions for business, government and the community. Jason talked about what neuroscience tells us about the nature of conscious experience. In his presentation, Jason discussed recent experimental work directed towards understanding the brain mechanisms that play a fundamental role in human consciousness. The research draws on studies of individuals with psychiatric and neurological conditions using the technique of functional magnetic resonance imaging, which allows the living brain to be visualised in action.

How can we determine when someone is conscious? Are humans the only animals capable of conscious experience? Such questions have occupied the thoughts of philosophers, ethicists and scientists for centuries. Tentative answers to these questions can now be provided due to advances in techniques for measuring brain activity as people think, feel, and behave. Recent breakthroughs in the scientific study of consciousness could have significant implications for situations as diverse as driving a car or making decisions about when to terminate life-support.
QBI relies on both public and private sources for the ongoing funding of its research programs and the Institute is very grateful for the support and generosity of its benefactors.

**Major Donors and Supporters**
- Peter Goodenough Estate
- Deborah Kelly & Gregg Thompson
- Lisa Palmer Estate (Spinal Research Consortium, SpinalCure Australia)
- Eric Sussman
- Frank & Patsy Youngleson
- The Index Group of Companies
- PricewaterhouseCoopers
- Australian Federal Government
- Queensland State Government

**Bequests and Donations of more than $1000**
- Mary Cross
- Lynette Davis
- Selwyn & Beryl Davis
- Ray Donaldson
- Ronald Fuller
- Maureen Gilmartin AM
- Michael Gordon
- Robert & Tricia Hammond
- Glenn Howell
- David Matthew Hunter
- Steve Knight
- Bob MacDonnell
- James & Terriann MacDonnell
- Daphne Maclean
- Colin Paroz
- Sylvia Peach
- Leah Perry
- David & Debra Thompson
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- Lions Club of Brisbane Macgregor
- Nucrush Pty Ltd
- Soroptimist International Brisbane South
- Southern Districts Rugby Union Football Club Inc

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**How to Support the Queensland Brain Institute**

QBI researchers seek to understand the fundamental mechanisms that regulate brain function – and to translate this knowledge into the development of effective treatments for neurological disorders.

The broad scope of QBI’s operations provides many avenues for supporting brain research in ways that can be personally tailored to the donor’s interests and resources. These include:
- Donations applied to a specific area of research
- Purchase of scientific equipment
- Scholarships for talented students
- Fellowships for early career scientists
- Laboratory dedications
- Professorial Chairs
- Gifts in memoriam

Gifts made during your lifetime may be structured in a way that maximises support to QBI by taking advantage of the tax deductibility of donations.

By leaving a bequest to QBI in your Will, you can provide a lasting legacy that will accelerate current research as well as underpin future research. Gifts to QBI through the University of Queensland are tax deductible under current legislation.

Please contact the Institute to discuss how you can support brain research at QBI.