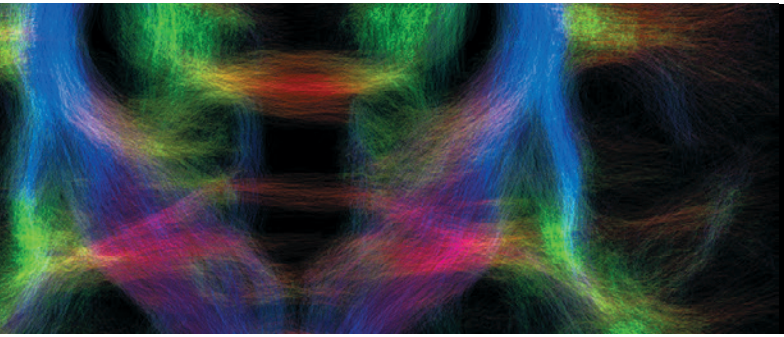
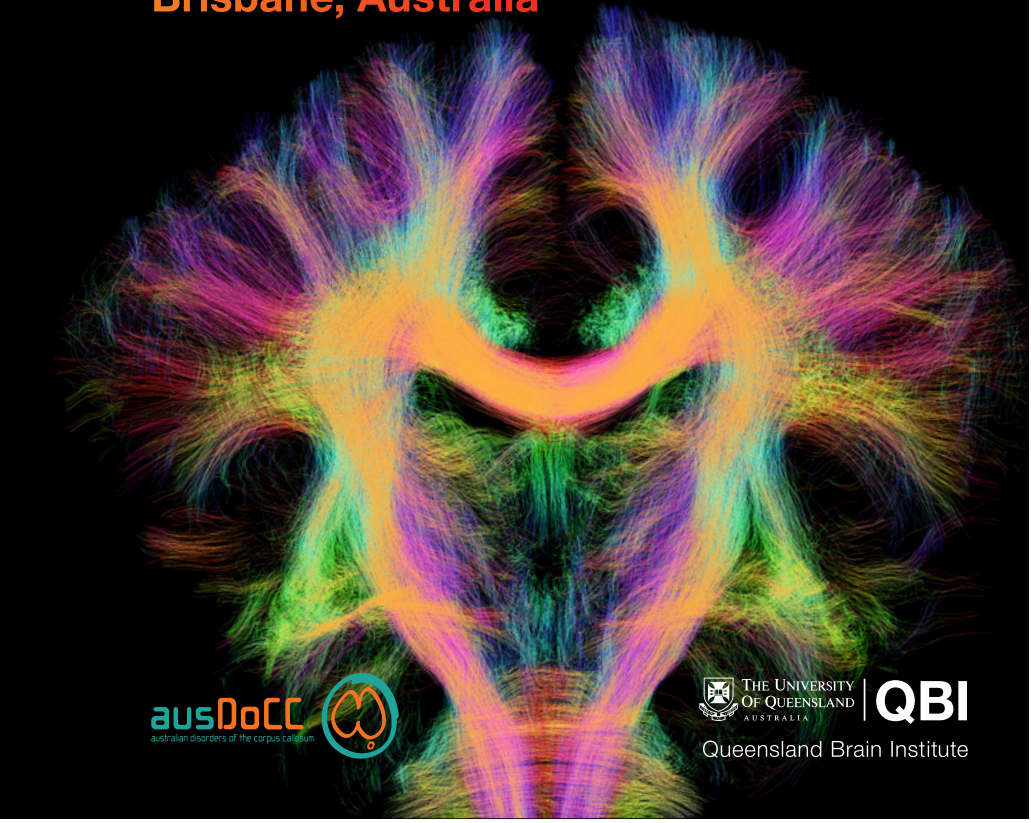


Cortical Connections 2015

19th and 20th of March, 2015
Queensland Brain Institute
Brisbane, Australia



ausDoCC
australian disorders of the corpus callosum

The University Of Queensland
AUSTRALIA
QBI
Queensland Brain Institute

Program, Speaker Biographies, and Abstracts

Programme Day 1 Thursday March 19

8:15AM	**Registration open**	
8:45AM	**Welcome by Prof. Linda Richards** Queensland Brain Institute	

Session 1: Development of cortical wiring

9:00–10:00AM	**Prof. Roberto Lent** Federal University of Rio de Janeiro ***Distance plasticity - on connectomes and dysconnectomes***	Session chairs **Dr Ilan Gobius** and **Ms Laura Fenlon**
10:00–10:30AM	**Dr Richard Leventer** Murdoch Childrens Research Institute, Melbourne ***Agenesis of the corpus callosum and the company it keeps***	
10:30–11:00AM	**Prof. Stephen Williams** Queensland Brain Institute ***Active dendritic integration underlies circuit-based neocortical computations***	
11:00–11:30AM	**Morning tea on the QBI terrace**	
11:30AM–12:00PM	**Prof. Linda Richards** Queensland Brain Institute ***Development of the corpus callosum***	

Programme Day 1 Thursday March 19

Session 2: Genetics of cortical wiring disorders

12:00–1:00PM	**Prof. Elliott Sherr** University of California, San Francisco ***Cerebral connectivity: from genes to cognition***	Session chairs **Dr Jens Bunt** and **Dr Peter Kozulin**
1:00–1:30PM	**Lunch at QBI**	
1:30–2:00PM	**Prof. Jozef Gécz** University of Adelaide ***Protocadherin 19 and female limited epilepsy and intellectual disability***	
2:00–2:30PM	**Prof. Kathryn North** Director, Murdoch Childrens Research Institute, Melbourne ***Learning disabilities in childhood – lessons from Neurofibromatosis type 1***	
2:30–3:00PM	**Prof. Ingrid Scheffer** The University of Melbourne, Florey Neurosciences Institute, Murdoch Childrens Research Institute, Melbourne ***Genetics of the epilepsies—framing cortical connections***	
3:00–3:30PM	**Afternoon tea**	
3:30–4:30PM	**Prof. Tania Attié-Bitach** Hospital Necker-Enfants Malades & Université Paris Descartes, Paris ***Unraveling the causes of corpus callosum anomalies: from fetal pathology to NGS and reverse phenotyping***	Session chairs **Mr Jonathan Lim** and **Ms Laura Morcom**
4:30–5:00PM	**Dr George McGillivray** Murdoch Childrens Research Institute, Melbourne ***Antenatal diagnosis of ACC: How do we predict a child's future?***	
5:00–5:30PM	**Dr Paul Lockhart** Murdoch Childrens Research Institute, Melbourne ***Using modern genomic technologies to identify genes associated with agenesis of the corpus callosum***	
5:30–6:30PM	**Discussion of international consortium (optional)**	

7:00PM	**Conference dinner at Customs House**

Programme Day 2 — Friday March 20

Session 3: Imaging cortical wiring

9:00–10:00AM	**Prof. Fernanda Tovar-Moll** Federal University of Rio de Janeiro and D'Or Institute for Research and Education (IDOR) ***Imaging brain connectivity and plasticity in the living human brain***	Session chairs **Dr Yonghui Li** and **Mr Tim Edwards**
10:00–10:30AM	**Prof. Tianzi Jiang** Queensland Brain Institute ***How do risky genes of brain diseases affect the brainnetome?***	
10:30–10:45AM	***short talk:*** **Dr Rodrigo Suarez** Queensland Brain Institute ***Homotopy, axonal segregation and cortical hubs in early-branched mammals reveal a pre-callosal template of interhemispheric cortical connections***	
10:45–11:00AM	***short talk:*** **Mr Luke Hearne** Queensland Brain Institute ***Interactions between default mode and control networks with increases in complexity during cognitive reasoning***	
11:00–11:30AM	**Morning tea on the QBI terrace**	

Programme Day 2 — Friday March 20

Session 4: Cognitive function and cortical wiring disorders

11:30AM–12:30PM	**Prof. Lynn Paul** Caltech, USA ***Cognitive and behavioral consequences of callosal malformation***	Session chairs **Dr Annalisa Paolino** and **Mr Gonzalo Almarza**
12:30–12:45PM	***short talk:*** **Ashley Marsh** Murdoch Childrens Research Institute ***Complete callosal agenesis, pontocerebellar hypoplasia and axonal neuropathy caused by mutation of AMPD2***	
12:45–1:00PM	***short talk:*** **Dr David Graham** The University Of Sydney ***A systematic review of corpus callosotomy outcomes in children***	
1:00–1:15PM	***short talk:*** **Fenna Bacchus** Independent Interdisciplinary Researcher ***A preliminary survey of the fundamental neurocognitive differences among 'cyclical thinking': subgroupings and linear thinking***	
1:15–1:30PM	***short talk:*** **Michael Valente** Monash Health ***Validation of a novel proverb test for frontal function***	

Wrap up and conference conclusion: Prof. Linda Richards

1:30–2:30PM	**Lunch at QBI**	
3:00–4:00PM	**ausDoCC tours of the Queensland Brain Institute** **(meet in the QBI reception area)**	Tours conducted by **Ms Katherine Robbins,** **Dr Peter Kozulin,** **Dr Rodrigo Suarez,** **Mr Tim Edwards,** **Mr Thomas Pollak**
4:00–5:00PM		

Professor Roberto Lent

Federal University of Rio de Janeiro

Roberto Lent is Professor of Neuroscience at the Institute of Biomedical Sciences, Federal University of Rio de Janeiro, Brazil. He also earned his MD and PhD at this University. Professor Lent worked at the Institute of Biophysics for almost 20 years, until he moved to the institute of Biomedical Sciences in 1994, where he runs the Laboratory of Neuroplasticity. He is currently the Director of the Institute.

He has conducted studies on neuroplasticity, neurodevelopment and evolution of the nervous system, employing different techniques, from cell biology to neuroimaging. His studies have recently revisited some widely held dogmas of quantitative neuroscience, including that which wrongly attributes the round numbers of one hundred billion neurons to the human brain, with ten-fold more glial cells. More recently in neuroplasticity, he has contributed to unravelling the Sperry paradox on acallosal subjects, revealing extensive white matter rewiring in the human brain during development.

Besides his scientific publications in specialised journals, Professor Lent dedicates part of his time to the popularisation of Neuroscience, with books for adults and for children. For this activity he has been awarded the Brazilian National Prize on Popularization of Science in 2010, and the Prize for Public Understanding and Popularization of Science by the Academy of Sciences of the Developing World (TWAS) in 2007.

Long distance plasticity - on connectomes and dysconnectomes

Roberto Lent

Institute of Biomedical Sciences, Federal University of Rio de Janeiro, Brazil

Defined as the immensely complex set of neuronal circuits in the normotypic brain, the human connectome undergoes a sequential development to establish gradually in embryos, children, and youngsters until adulthood. Under abnormal genetic and/or environmental conditions, a dysconnectome is formed instead, with novel circuits that either (re)establish normal function or cause miswiring and produce symptoms. The mechanisms underlying these circuit-forming events – both in normal and in pathological circumstances – may be called *long-distance plasticity*, to differentiate it from the subtle, synaptic phenomena that give the brain its great adaptability throughout life. Long-distance plasticity, therefore, may be considered the key concept to explain individual variation of normal brain circuits, and the aberrant circuitry that explains a number of neuropsychiatric disorders. One of the greatest long circuits in the brain is the corpus callosum. To form it, an orchestral sequence of molecular and cellular signals is expressed orderly from the 12th gestation week onwards, recognized by callosal axons in the cortex as they grow towards the targets. When a particular group of these signals fails, callosal axons grow astray, and aberrant tracts form within and between the hemispheres. This condition is known as callosal dysgenesis. As an example of the concept of long-distance plasticity, we will here compare the circuitry of commissural fibers in animals and humans as formed under normal developmental mechanisms, with those formed in cases of callosal dysgenesis. Work with animal models and with humans will be reviewed, describing normal and abnormal circuitry and their functional significance.

Funding sources: Brazilian Ministry of Science, Technology and Innovation (CNPq, INNT), Ministry of Education (CAPES), and the Rio de Janeiro Foundation for the Support of Science (FAPERJ).

Associate Professor Richard Leventer

Murdoch Childrens Research Institute, Melbourne

A/Prof. Richard Leventer is a consultant paediatric neurologist at the Royal Children's Hospital (RCH) and a Group Leader of Neuroscience Research within the Clinical Sciences theme of the Murdoch Children's Research Institute (MCRI). He was awarded his PhD on the topic of brain malformations in 2007, which included research that was commenced whilst doing a Neurogenetics Fellowship in the Brain Malformation Program at the University of Chicago.

A/Prof. Leventer is Director of the RCH/MCRI Brain Malformation Program and Clinic, which is the referral centre for children with brain malformations from Australia and New Zealand. He is a Chief Investigator on the MCRI Accelerated Gene Identification Program within the MCRI Bruce Lefroy Centre. He conducts collaborative research on brain malformations with local, national and international colleagues including active projects on agenesis of the corpus callosum with genetics, basic science and neuropsychology colleagues. A/Prof. Leventer is the Australian representative on a new international consortium studying white matter disorders, known as the Global Leukodystrophy Initiative. A/Prof. Leventer was president of the Australia and New Zealand Child Neurology Society from 2002 - 2007.

Agenesis of the corpus callosum and the company it keeps

Richard Leventer

Murdoch Childrens Research Institute, Melbourne

Whilst developmental abnormalities of the corpus callosum may occur in isolation, they are often just one component of a more widespread disorder of brain development, often invoving abnormalities of the cerebral cortex. A/Prof. Leventer will provide a brief overview of normal cortical development, and will highlight the most common disorders of brain development that may be seen in combination with agenesis or hypogenesis of the corpus callosum. The presentation will highlight the imaging features of these disorders and what is currently known regarding their aetiologies.

Professor Stephen Williams

Queensland Brain Institute

Stephen Williams received his BSc and PhD degrees in the United Kingdom, and conducted postdoctoral studies in the United Sates and Australia. He started an independent laboratory at the MRC Laboratory of Molecular Biology, Cambridge, UK in 2002 and was tenured in 2008. He relocated to the QBI in 2010. He is currently supported by an ARC Future Fellowship.

Professor Williams laboratory studies how information is processed and computed by neuronal circuits to affect behaviour. He uses multi-site electrophysiological and optical recording techniques to study these circuits in the neocortex and retina.

Active dendritic integration underlies circuit-based neocortical computations

Stephen Williams
Queensland Brain Institute

Neocortical circuits have been subject to detailed anatomical and functional investigation for over a century, yet our understanding of how circuit-based computations are executed remains in its infancy. The development of functional recording techniques, which enable the direct examination of physiologically engaged neuronal circuit operations are, however, beginning to mechanistically dissect the rules underlying neocortical information processing. Using electrophysiological and imaging techniques we have identified that single neocortical pyramidal neurons possess powerful information-processing capabilities, exhibiting multiple sites for active dendritic integration that locally compute synaptic input and drive the action potential output. Recently, we have directly demonstrated that active dendritic integrative mechanisms underlie physiologically engaged circuit-based neuronal computations in the rodent somato-motor neocortex.

Funding: ARC, NHMRC, Freemasons Queensland.

Professor Linda J. Richards

Queensland Brain Institute

Professor Richards did her undergraduate degree at Monash University and obtained her BSc (Hons) and a PhD from The University of Melbourne and The Walter and Eliza Hall Institute in the laboratory of Professor Perry Bartlett. Her thesis was on the determination of neuronal lineage in the developing spinal cord. She then moved to the USA to complete a postdoctoral fellowship at The Salk Institute for Biological Studies where she worked with Professor Dennis O'Leary on cortical development and formation of the lateral cortical projection through the internal capsule. She began her independent laboratory at The University of Maryland Medical School in 1997, in the Department of Anatomy and Neurobiology chaired by Professor Michael Shipley.

In 2005 she moved her laboratory to The University of Queensland and was appointed as an Associate Professor in the Queensland Brain Institute and The School of Biomedical Sciences and in 2006, she was appointed as an NHMRC Senior Research Fellow. In 2010, she was promoted to Professor at The University of Queensland and promoted to NHMRC Principal Research Fellow in 2011. In addition to running her laboratory, Professor Richards is passionate about informing the public about science. In 2006 she founded the Australian Brain Bee Challenge, a program that inspires and excites high school students about science.

Development of the corpus callosum

Linda J. Richards

Queensland Brain Institute

The corpus callosum is the largest fiber tract in the human brain and connects the two cerebral hemispheres. The mechanisms regulating the formation of the corpus callosum are complex as they involve all of the developmental mechanisms required to form a functioning brain. Our laboratory not only investigates the mechanisms involved in the formation of the corpus callosum, but also how other seemingly unrelated structural brain malformations may be related to callosal dysgenesis via the disruption of similar cellular and molecular mechanisms. Subjects with corpus callosum agenesis/dysgenesis are currently identified based on their structural midline phenotype, a limit imposed by current clinical neuroimaging capabilities. However, these subjects are likely to represent sub-groups with more severe forms of callosal dysgenesis. More subtle changes in callosal targeting could represent a much larger and diverse group of subjects that have normal midline crossing, but disrupted targeting in the contralateral hemisphere. This area of research is in its formative stages but could offer potentially major breakthroughs in how the brain is normally wired during development and what mechanisms may be disrupted in disorders of brain connectivity.

Acknowledgement of funding: Our research is funded by project grants from the National Health and Medical Research Council, Australia, the Australian Research Council, and the National Institutes of Health, USA. LJR is supported by an NHMRC Principal Research Fellowship.

Professor Elliott Sherr

University of California San Francisco

Elliott Sherr is a Professor in Neurology and Pediatrics at the Institute of Human Genetics at UCSF. He directs the Brain Development Research Program, a group that studies the genetics and biology of autism and epilepsy (http://brain.ucsf.edu). Specific areas of interest include understanding the link between cerebral connectivity and brain function (as exemplified by agenesis of the corpus callosum) and understanding the biological underpinnings of autism. Dr Sherr also is part of a large epilepsy genetics consortium (www.epgp.org/epi4k/) in which he leads a team trying to understand the genetic causes of severe childhood epilepsies, such as infantile spasms. For his research, Dr Sherr was the 2006 recipient of the Philip R. Dodge Young Investigator Award from the Child Neurology Society. Dr Sherr is a board certified Child Neurologist and co-directs the Comprehensive Center for Brain Development at UCSF. In this capacity, he cares for children with neurodevelopmental disorders, including autism, intellectual disability and epilepsy.

Dr Sherr is a native of California and completed his undergraduate degree in Philosophy and Biology at Stanford University. He obtained his MD and PhD at Columbia University in New York and completed his clinical training in Pediatrics and Neurology at UCSF. He lives in San Francisco with his wife (a biotechnology financial executive) and his three children.

Cerebral connectivity: from genes to cognition

Elliott Sherr

University of California San Francisco

Professor Sherr will present work from his research group investigating disorders of the corpus callosum (agenesis of the corpus callosum; ACC), focusing on gene discovery, insights gained from novel imaging modalities and functional assessments of cognition in individuals born without the corpus callsoum, which is the main connection between the two cerebral hemispheres. In addition, he will present data highlighting the connection between autism spectrum disorder and ACC, and will address the direction of new research in this field, including implications for novel therapeutics.

Professor Jozef Gécz

Robinson Research Institute and School of Paediatrics and Reproductive Health, The University of Adelaide, Adelaide, Australia

Jozef Gécz, PhD is a NHMRC Senior Principal Research Fellow and Professor of Human Genetics at the Department of Paediatrics, University of Adelaide. Dr. Gécz established and currently heads the Neurogenetics Research Program at the Women's and Children's Hospital, Adelaide. Dr. Gécz has completed his undergraduate studies at the Comenius University and PhD at the Slovak Academy of Sciences in Bratislava, Slovakia under the supervision of Dr. Ferak and subsequently trained for two years with Prof. M. Fontes in Marseille, France as an INSERM postdoctoral fellow. In 1994 he joined the group of Professor G. Sutherland in Adelaide where he so far discovered or contributed to the discovery of numerous (>70) genes for various forms of X-chromosome linked intellectual disability, epilepsy and cerebral palsy (e.g. FMR2, ARX, CDKL5, PHF6, PCDH19, UPF3B or USP9X). Dr. Gécz published in excess of 220 peer reviewed publications. Dr. Gécz is an active member of various professional societies and national and international committees. He has served, among others, on the International Congress of Genetics (2003) and Human Genetics (2006 and 2011), as well as HUGO meetings (2006 and 2007) scientific program organising committees. The main research interest of Dr. Gecz's group is gene identification and functional, cellular and molecular modelling of intellectual disability, epilepsy, autism and cerebral palsy.

Protocadherin 19 and female limited epilepsy and intellectual disability

Jozef Gécz *et al*

Robinson Research Institute and School of Paediatrics and Reproductive Health, The University of Adelaide, Adelaide, Australia

TPCDH19 Girls Cluster Epilepsy (PCDH19 GCE) is an intriguing X-chromosome disorder that primarily affects females. Males with loss of function mutations are spared. PCDH19 GCE encompasses a broad clinical spectrum from infantile epileptic encephalopathy resembling Dravet syndrome to epilepsy with or without intellectual disability and autism spectrum disorders. PCDH19 is a non-clustered protocadherin that interacts with N-cadherin and is expressed in the brain, particularly in the cerebral cortex and hippocampus. We found that PCDH19 is involved in the regulation of steroid hormone receptors and as such regulates gene expression. By studying PCDH19 GCE patient cells we identified a set of sex-biased genes with significant differential expression (p=2.51E-47). Among these we studied in greater detail the allopregnanolone, estradiol and testosterone metabolizing enzymes AKR1C1-4. Subsequently we demonstrate that PCDH19 GCE girls are allopregnanolone deficient in their blood, when compared against age-matched controls. Steroids and neurosteroids are known to play a crucial role in normal neuronal development. These findings open realistic opportunities for targeted therapeutic interventions for PCDH19 GCE.

Acknowledgement of funding: This work is supported by NHMRC Program grant 628952 and NHMRC senior principal research fellowship 1041920 to JG, and Insieme per la Ricerca PCDH19 – ONLUS Foundation grant (Italy).

Professor Kathryn North AM

Director, Murdoch Childrens Research Institute, David Danks Professor of Child Health Research, University of Melbourne

Professor Kathryn North AM is Director of the Murdoch Children's Research Institute and the David Danks Professor of Child Health Research at the University of Melbourne.

Professor North is trained as a paediatric physician, neurologist and clinical geneticist and in 1994, was awarded a doctorate from the University of Sydney for research into Neurogenetics. She completed a postdoctoral fellowship in the Harvard Genetics Program. Her previous positions include the Douglas Burrows Professor of Paediatrics, Faculty of Medicine, University of Sydney (2004-2012) and Head of the Institute for Neuroscience and Muscle Research (2008-2012). Her laboratory research interests focus on the molecular basis of inherited muscle disorders – particularly the muscular dystrophies and congenital myopathies – as well as genes which influence normal skeletal muscle function and elite athletic performance. Her clinical research focusses on clinical trials of therapies for muscular dystrophy as well as the development of interventions for children with neurofibromatosis type 1 and learning disabilities.

Professor North has received a number of awards for her research including the GSK Australia Award for Research Excellence (2011), the Ramaciotti Medal for Excellence in Biomedical Research (2012) and the Member of the Order of Australia (AM) for service to medicine in the field of neuromuscular and neurogenetics research (2012). In 2012, Professor North was appointed Chair of the NHMRC Research Committee and Member of NHMRC Council and in 2014 was appointed Vice Chair of the Global Alliance for Genomics and Health, an international consortium of more than 200 institutions promoting the sharing of genomic and clinical data.

Learning disabilities in childhood – lessons from Neurofibromatosis type 1

Kathryn North AM

Director, Murdoch Childrens Research Institute

David Danks Professor of Child Health Research, University of Melbourne.

Neurofibromatosis type 1 (NF1) is an autosomal dominant disorder with a frequency of 1 in 3,000. NF1 is a multisystem disorder characterised by pigmentary skin lesions and nerve tumours. However the most common neurological 'complications' of NF1 in childhood are cognitive deficits and academic learning difficulties– including dyslexia, deficits in organising and planning and difficulties with sustaining and switching their attention. Approximately 35-40% of children with NF1 have ADHD.

Understanding the neuropathological basis of the different types of cognitive dysfunction in NF1 is crucial to the development and design of potential therapies and interventions. Some cognitive deficits are associated with developmental/structural brain anomalies while other deficits are associated with biochemical defects and are more likely to be reversible and amenable to pharmacological therapy.

The *NF1* gene is a tumour suppressor gene that modulates activity of the *ras* oncogene. Studies in *Nf1* mouse models have shown that abnormal *ras* activation is associated with impaired hippocampal based learning and that abnormal dopamine homeostasis is the primary biochemical defect underlying attention deficits in NF1. These animal studies have provided the preclinical evidence for current clinical trials in children with NF1, and also provide insight into the etiology of learning disabilities in the general population.

Professor Ingrid Scheffer

AO MBBS PHD FRACP FAHMS FAA

Chair of Paediatric Neurology Research, Departments of Medicine and Paediatrics, The University of Melbourne, Austin Health and Royal Children's Hospital, Melbourne
Senior Principal Research Fellow, The Florey Institute of Neuroscience and Mental Health
Director of Paediatrics, Austin Health, Melbourne, Australia

Professor Ingrid Scheffer is a physician-scientist whose work as a paediatric neurologist and epileptologist at the University of Melbourne and Florey Institute has led the field of epilepsy genetics over more than 20 years, in collaboration with Professor Samuel Berkovic and molecular geneticists. This resulted in identification of the first epilepsy gene and many more genes subsequently. Professor Scheffer has described many novel epilepsy syndromes and refines genotype–phenotype correlation. She recently led the first major reclassification of the epilepsies in two decades as Chair of the International League Against Epilepsy Commission for Classification and Terminology. She has received many awards: 2007 American Epilepsy Society Clinical Research Recognition Award, 2009 RACP Eric Susman Prize, 2013 GSK Award for Research Excellence, ILAE Ambassador for Epilepsy Award, 2013 Australian Neuroscience Medallion, 2013 Emil Becker Prize for child neurology and the L'Oréal-UNESCO Women in Science Laureate for the Asia-Pacific region for 2012. In 2014, she was elected as a Fellow of the Australian Academy of Science and also elected as Vice-President of the Australian Academy of Health and Medical Sciences. In the same year, Professor Scheffer was awarded the Order of Australia in the Queens Birthday Honours List and, together with Professor Berkovic, was awarded the Prime Minister's Prize for Science.

Genetics of the epilepsies—framing cortical connections

Ingrid E. Scheffer
Florey Institute of Neuroscience and Mental Health, The University of Melbourne, Austin Health and Royal Children's Hospital, Melbourne, Australia

With the advent of next generation sequencing, there has been a virtual explosion in the number of known epilepsy genes which has been especially prolific in the last two years. The major areas of impact are the focal epilepsies, a group previously considered predominantly acquired disorders apart from rare large families with autosomal dominant disorders. With the new gene discoveries, there is a paradigm shift with regard to evaluation of children and adults with focal epilepsies, and now includes both lesional and non-lesional cases. These findings have implications for aetiology but also for management, as particular genes may suggest that the patient should be scrutinized for a focal cortical dysplasia. New focal epilepsy genes are shedding light on novel mechanisms with the mammalian target of rapamycin pathway becoming a critical pathway leading to focal epilepsy, apart from its role in the relatively rare disease, tuberous sclerosis.

The epileptic encephalopathies are proving to be genetically highly heterogeneous, with many patients having *de novo* dominant mutations. Phenotypic heterogeneity is also seen with many genes. As new genes are discovered, the phenotypes of specific genetic encephalopathies are emerging, and may have distinctive features that aid diagnosis. Cohorts of patients with specific diseases will rapidly grow and this, in turn, will inform treatment choices and long term prognosis.

Professor Tania Attié-Bitac

Département de Génétique et INSERM U1163, Institut Imagine, Université Paris Descartes Sorbonne Paris Cité, Paris, France

Professor Tania Attié-Bitach is a human geneticist specialising in molecular genetics of congenital malformations at Paris Descartes University. She has developed clinical and genetic research on human fetal disorders at the Imagine Institute, Necker Medical School. She has driven several projects leading to the identification of genes responsible for Meckel syndrome, related ciliopathies and other lethal disorders such as Matthew-Wood and Fowler syndromes. The identification of KIF7 mutations as the genetic cause of acrocallosal syndrome and its clinical heterogeneity has raised the question of the involvement of primary cilia defects in corpus callosum anomalies. She currently leads a national project (ANR Cilaxal) aiming to characterise the genetic causes of isolated and syndromic causes of corpus callosum anomalies in humans.

Unraveling the causes of corpus callosum anomalies: from fetal pathology to NGS and reverse phenotyping

Tania Attié-Bitac

Département de Génétique et INSERM U1163, Institut Imagine, Université Paris Descartes Sorbonne Paris Cité, Paris, France

Corpus callosum (CC) is the major brain commissure connecting the homologous areas of both hemispheres at the midline. CC malformations (CCM) are the most frequent brain malformations with an incidence of 1/4000 newborn often associated with chromosomal anomalies or Mendelian syndromes with recessive and dominant inheritance. Recurrence is observed in 5 % of cases. Children with CCM have an uncertain neurodevelopmental outcome. Therefore, counseling remains challenging, especially prenatally.

We conducted a clinical, chromosomal and molecular analysis of a cohort of fetus with CCA. We first systematically reviewed the data of 141 fetuses with CCM as isolated or associated autopsy findings in our center. Neuropathological examinations were performed in 133 of them. We completed the cytogenetic analysis by a CGH array when the underlying etiology was not found and then used a NGS strategy to unravel the genetic causes.

We found a spectrum of CCM classified as follows: agenesis of corpus callosum (ACC, 54), corpus callosum hypoplasia (HCC, 23), CC dysplasia (DCC, 23) and CCM due to malformations of cortical development (MCD, 31). Interestingly, following fetal pathology examination, only 25 (19%) remained isolated, highlighting the importance of autopsy following termination of pregnancy. Following fetal imaging, fetal examination, and cytogenetic analysis, the underlying etiology was found in 40 cases: vascular, infectious, chromosomal rearrangements (15%) or Mendelian diseases (15%).

We then used whole exome targeted NGS sequencing of 423 selected genes in undiagnosed cases. This strategy allowed a diagnosis in 10 % of undiagnosed individuals, such as a PDH deficiency (PDHA1), PCH with ACC (AMPD2), genitopatelar (KAT6B), Primrose (ZBTB20), Coffin-Siris (ARID1A and ARID1B) or Chudley Mac Cullough syndrome (GPSM2). Interestingly some diagnoses were not possible antenatally due to the absence of specific signs, but reevaluation of fetal pathological data (reverse phenotyping) supported NGS findings. These situations will be illustrated.

All together, combined fetal imaging, fetal examination, cytogenetic and molecular analysis allowed the identification of the cause in 40% of individuals, with several candidate genes requiring further investigation. The classification based on the underlying neurodevelopmental defects pave the way for further genetic study and genotype-phenotype correlations. A better knowledge of the genetic causes and prognostic factors for CCM is the challenge of the next decade for the clinical management of fetuses and children.

George McGillivray

Murdoch Childrens Research Institute, Melbourne

George McGillivray is a Clinical Geneticist with expertise in fetal medicine. He has appointments as a senior specialist at the Victorian Clinical Genetics Services, the Mercy Hospital for Women, and the Royal Women's Hospital in Melbourne. He consults at the Perinatal Unit at The Mercy Hospital, the Fetal Medicine Unit at The Women's Hospital and the Neurogenetics Clinic at The Royal Children's Hospital. He provides clinical liaison for the prenatal cytogenetics laboratory at VCGS Pathology offering high resolution prenatal chromosome microarray testing to pregnant women and their partners in Australia. He is a guest author currently reviewing national and international statements on prenatal screening and diagnosis for the Royal Australasian College of Obstetricians and Gynaecologists and the International Society of Ultrasound in Obstetrics and Gynecology respectively. He contributes to research into the genetic causes of human brain malformations through the "Genetics of Brain Disorders" and "Accelerated Gene Identification" research programs at the Murdoch Childrens Research Institute in Melbourne.

Antenatal diagnosis of ACC: How do we predict a child's future?

George McGillivray

Murdoch Childrens Research Institute, Melbourne

Agenesis or dysgenesis of the fetal corpus callosum (ACC/DCC) is a rare but relatively common abnormality of fetal brain development. ACC/DCC can be identified in the second trimester ultrasound screening from 20 weeks onward and confirmed by fetal MRI. In this context, genetic counselling is the interactive process by which prospective parents are advised about structural and genetic anomalies in their unborn child. This is done so that parents can make informed decisions about their pregnancy. ACC/DCC can be associated with other anomalies of fetal brain development, structural anomalies in other fetal organ systems or pathogenic chromosome changes on microarray. The prognosis for a future child with these associations is more certain and the risk of severe disability is often high. Providing accurate antenatal advice is more difficult with apparently isolated fetal ACC/DCC. The risk of childhood developmental delay or intellectual disability in this setting is 25-30% but it is not currently possible for clinicians to distinguish between fetuses with a good or poor prognosis. This is despite a range of imaging findings. We hope that advances in the understanding of genetic causes of ACC/DCC and the introduction of more sensitive clinical genomic testing in pregnancy will improve the diagnostic rate and the quality of advice we are able to give prospective parents at this difficult time.

Associate Professor Paul Lockhart

Bruce Lefroy Centre, Murdoch Childrens Research Institute, Melbourne, Australia

A/Professor Paul Lockhart received his PhD from the University of Melbourne in 2000. He was awarded a NHMRC CJ Martin Fellowship to study the genetics of neurodegenerative disorders, specifically Parkinson's disease, with Professor John Hardy and Professor Matthew Farrer at The Mayo Clinic, Florida. Paul returned to Australia in 2004 and joined the newly formed Bruce Lefroy Centre (BLC) at the Murdoch Childrens Research Institute.

In 2009 he became Co-Director of the Bruce Lefroy Centre and expanded his research activities to include gene identification utilising modern genomic technologies. He works in close collaboration with the Victorian Clinical Genetics Service to identify and characterise the genetic basis of rare Mendelian genetic disorders in families presented to the clinic. Currently he is a NHMRC Career Development Fellow and leads a laboratory group that investigates the molecular basis of neurogenetic disorders.

Using modern genomic technologies to identify genes associated with agenesis of the corpus callosum

Paul J. Lockhart

Bruce Lefroy Centre, Murdoch Childrens Research Institute, Melbourne, Australia

Agenesis of the corpus callosum (ACC) is a genetically complex, congenital brain malformation, characterised by partial or complete absence of the corpus callosum, the structure that connects the two cerebral hemispheres. ACC is associated with diverse neurological deficits and affects ~1:4500 children. While there is considerable evidence that genetics contributes to ACC, in approximately 50% of cases the cause remains unknown.

The recent development of massively-parallel sequencing and associated genomic technologies has led to the rapid identification of genes underlying many human disorders. In particular, gene discovery can now be performed in small families that were previously underpowered for such analysis. This presentation will review the methodologies being applied in the research group to identify novel genes associated with ACC. Specific examples using different genetic models will be presented.

This research has been supported by grants from the National Health and Medical Research Council (Australia) and philanthropic donations to the Bruce Lefroy Centre.

Professor Fernanda Tovar-Moll MD

D'Or Institute for Research and Education (IDOR); Institute of Biomedical Sciences and National Center for Structural Biology and Bioimaging (CENABIO), Federal University of Rio de Janeiro, Rio de Janeiro, Brazil

Fernanda Tovar-Moll earned her MD degree from the Federal University of Rio de Janeiro (UFRJ), Brazil. She completed a Medical Residency program in Radiology, with an emphasis in Neuroradiology, and obtained a PhD in Morphological Sciences at UFRJ. Her PhD thesis explored the use of MR tractography in assessing neuroplasticity in children with callosal dysgenesis. She was a postdoctoral Fellow at the National Institute of Neurological Diseases and Stroke, National Institutes of Health (USA), from 2004-2007, where she investigated MRI anatomical biomarkers in demyelinating and neurodegenerative central nervous system disorders.

Dr Tovar-Moll is currently an adjunct professor at the Institute of Biomedical Sciences and the director of the Bioimaging Unit for Small Animals (CENABIO) at UFRJ. In addition, she also holds a position as the scientific director of the D'Or Institute for Research and Education (IDOR), a private not-for-profit research institute, which she co-founded in 2009. She has been working in projects related to basic, clinical and translational research in neurodegenerative and neurodevelopmental conditions. Her main research interest is to employ novel *in vivo* imaging techniques in human and rodents to map brain circuits in order to improve the understanding of patho-physiological mechanisms related to functional and structural brain connectivity and brain plasticity in normal and pathological conditions. Another focus of interest is to employ neuromodulatory techniques, such as tDCS, TMS and MRI neurofeedback to induce changes in brain circuits to improve neurological function in stroke and other abnormal conditions.

Imaging brain connectivity and plasticity in the living human brain

Fernanda Tovar-Moll

D'Or Institute for Research and Education (IDOR); Institute of Biomedical Sciences and National Center for Structural Biology and Bioimaging (CENABIO), Federal University of Rio de Janeiro, Rio de Janeiro, Brazil

Over the last two decades, structural and functional neuroimaging have promoted a great advance on the characterization of the brain connectome, and its capacity to reorganize in response to internal and environmental influences, opening new windows for the investigation of changes in brain networks in normal and pathological conditions. These new concepts and techniques will be presented, including fMRI (especially resting state fMRI) that allows us to probe functional coupling of networks, and diffusion tensor imaging, which characterizes white matter tracts interconnecting nodal structures among brain networks. We will present the use of these methods in investigating brain plasticity in two main conditions, [long-term] traumatic amputation and callosal dysgenesis. Although both conditions are associated with brain circuitry reorganization, they exhibit different levels of changes. Our data indicates that amputees suffering from phantom limb sensation show enlarged functional stump cortical representation and changes in intra- and inter-hemispheric functional connectivity, associated with microstructural white matter disorganization of the corpus callosum. On the other hand, patients with callosal dysgenesis show a massive white-matter reorganization, leading to the formation of anomalous tracts interconnecting distant cortical areas. Supported by neuropsychological data, we will describe how this structural long–distance plasticity may explain the maintenance of inter-hemispheric functional communication in callosal dysgenesis despite the lack of the corpus callosum, a paradox that has defied neuroscientists for decades. We will also address how these findings are inspiring new research on non-human models to establish a more detailed view of brain plasticity mechanisms.

Funding sources: Brazilian Ministry of Science, Technology and Innovation (CNPq, INNT), Ministry of Education (CAPES), the Rio de Janeiro Foundation for the Support of Science (FAPERJ) and intramural grants from D'Or Institute for Research and Education (IDOR).

Professor Tianzi Jiang

Queensland Brain Institute

Tianzi Jiang is Director of the Brainnetome Centre and Professor of Neuroimaging and Brain Disorders at Institute Automation of the Chinese Academy of Sciences and Professor at the Queensland Brain Institute, The University of Queensland. His research interests include neuroimaging, brainnetome, imaging genetics, and their clinical applications in brain disorders. He is the author or co-author of over 200 reviewed journal papers in these fields and the co-editor of six issues of the Lecture Notes in Computer Sciences. He is Associate Editor of IEEE Transactions on Medical Imaging, IEEE Transactions on Autonomous Mental Development, and Neuroscience Bulletin.

How do risky genes of brain diseases affect the brainnetome?

Tianzi Jiang
Queensland Brain Institute

The Brainnetome (Brain-net-ome) is a new “-ome” in which the brain network is its basic research unit. Convergent evidence from multimodal imaging studies has demonstrated that brain networks are heritable both structurally and functionally and this suggests that brain connectivity and networks are under genetic control. Therefore, the mechanisms by which specific genetic variants affect brain networks are attracting more and more attention. In this talk we present the advances on how the brainnetome meets genome. First, we give an introduction about the brainnetome. Second, we present some imaging techniques, computational methodologies and software for the brainnetome. Second, we review some imaging genetic studies that took brain connectivity and brain networks as intermediate phenotypes from functional MRI and diffusion MRI, respectively. Finally, we give a perspective on the brainnetome meets genome.

short talk

Homotopy, axonal segregation and cortical hubs in early-branched mammals reveal a pre-callosal template of interhemispheric cortical connections

Rodrigo Suárez[1], Annalisa Paolino[1], Laura Morcom[1], Peter Kozulin[1], Laura R. Fenlon[1], Nyoman Kurniawan[2] & Linda J. Richards[1,3]

[1]Queensland Brain Institute, [2]Centre for Advanced Imaging, [3]School of Biomedical Sciences. The University of Queensland

Precise integration of left and right brain hemispheres is crucial for sensory, motor and associative processes. In placental mammals, this is accomplished by the corpus callosum, the largest tract in the brain. However, non-placental mammals, such as egg-laying monotremes and marsupials lack a corpus callosum; instead, all connections between neocortical hemispheres course through the anterior commissure. An important developmental and functional feature of the corpus callosum is the topographic arrangement of axons between homotopic regions of both hemispheres. Whether the origin of the corpus callosum in the ancestors of modern placentals led to establishing these connectivity patterns, or instead whether these patterns were already in place by the time of callosal origin, are open questions required to understand the events that led to callosal evolution.

To elucidate this, we studied commissural anatomy in the naturally acallosal platypus (monotreme) and dunnart (marsupial). We combined diffusion tensor magnetic resonance imaging (dtMRI) and stereotaxic injections of tract tracers in adults and found a remarkable segregation of commissural axons according to the location of their cell bodies. Moreover, by electroporating reporter genes in the developing brain of dunnarts inside the pouch and colabeling with molecular markers and tracer injections, we found ipsilateral and contralateral projections to hyper-connected regions (hubs), coexistence of homotopic and heterotopic contralateral projections, and independent populations of upper-layer cortical neurons distinguished by their ancestral versus proto-callosal projection fates. These results reveal that the evolution of the corpus callosum involved rearrangement of axonal routes while conserving an ancestral mammalian commissural template that originated more than 200 million years ago.

short talk

Interactions between default mode and control networks with increases in complexity during cognitive reasoning

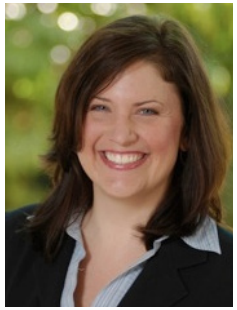
Luke Hearne

Queensland Brain Institute, University of Queensland

Successful performance of challenging cognitive tasks depends upon a consistent functional segregation of activity within the default-mode network, on the one hand, and control networks encompassing fronto-parietal and cingulo-opercular areas on the other. Recent work, however, has suggested that in some cognitive control contexts, nodes within the default-mode and control networks may actually cooperate to achieve optimal task performance. Here we used functional magnetic resonance imaging to examine whether the ability to relate variables while solving a cognitive reasoning problem involves transient increases in connectivity between default-mode and control regions. Participants performed a modified version of the classic Wason Selection Task, in which the number of variables to be related is systematically varied across trials. As expected, areas within the default-mode network showed a parametric deactivation with increases in relational complexity, compared with neural activity in null trials. Critically, some of these areas also showed enhanced connectivity with task-positive control regions. Specifically, task-based connectivity between the striatum and the angular gyri, and between the thalamus and right temporal pole, increased as a function of relational complexity. These findings challenge the notion that functional segregation between regions within default-mode and control networks invariably support cognitive task performance, and reveal previously unknown roles for the striatum and thalamus in managing network dynamics during cognitive reasoning.

Dr Lynn Paul

Caltech, Los Angeles



Lynn K. Paul, PhD is a Senior Research Scientist at California Institute of Technology, where she is directing a research program studying brain-structure, cognition and social processing in Agenesis of the Corpus Callosum (AgCC). Dr Paul received a PhD in Clinical Psychology from Fuller Graduate School of Psychology and completed a post-doctoral fellowship in clinical neuropsychology from the Department of Neurology, UCLA.

In graduate school, Dr Paul began working with Dr Warren Brown to describe the cognitive and behavioral profile of individuals with agenesis of the corpus callosum. Currently, she is an Associate Research Professor at Fuller Graduate School of Psychology, where she continues to collaborate with Dr Brown on research describing the AgCC profile.

In 2002, Dr Paul collaborated with other professionals and family members to found the National Organization for Disorders of the Corpus Callosum (NODCC). The NODCC is a 501c3 not-for-profit that brings families, clinicians, and scientists together in the effort to improve quality of life for people with callosal disorders. During her tenure as NODCC president, she co-authored “ACC and Me”, a children’s book about a boy with callosal agenesis.

Dr Paul is also more broadly interested in understanding the role that cortical connectivity plays in development of higher-order social cognition. In addition to research on AgCC, she collaborates with Dr Ralph Adolphs on studies of social processing and brain structure in high functioning adults with autism spectrum disorders and individuals with congenital bilateral amygdala lesions. She is also the Director of the Psychological Assessment for Research Laboratory at Caltech and principal investigator for the Psychological Asessment Core of the NIH-funded Conte Center for Social Decision Making.

Finally, Dr Paul maintains a clinical psychology practice (L.K.Paul and Associates) in Pasadena, where she sees adult outpatient psychotherapy clients and conducts neuropsychological assessments on individauls with callosal agenesis.

Cognitive and behavioural consequences of callosal malformation

Lynn Paul

Caltech, Los Angeles

The corpus callosum, whose roughly 200 million axons constitute the largest white matter tract in the human brain, remains enigmatic in its contribution to cognition and behavior. For two decades we have been characterizing the cognitive and behavioral features shared among individuals with agenesis of the corpus callosum (AgCC), a congenital brain malformation defined by the complete or partial absence of callosal structures (i.e. ~200 million axons fail to cross midline that typically interconnect the cerebral hemispheres). AgCC can be an isolated finding but can also co-occur with additional brain malformations and systemic conditions. Adolescents and adults with isolated AgCC and normal-range intellectual scores exhibit a shared pattern of limitations in fundamental aspects of cognition including inter-hemispheric transfer of complex information and processing speed, as well as higher order cognitive skills such as complex novel problem solving, comprehension of second-order meanings in language, and complex theory of mind and social interpretation. We have recently begun longitudinal studies of cognitive development in infants with AgCC. Our research aims to provide a foundation for more accurate cognitive and psychosocial profiling and intervention for people with corpus callosum disorders, while also informing wider central issues in neuroscience, such as functional development of the cortex and neuroplasticity.

Acknowledgment of funding: Our research on AgCC has been supported by the Simons Foundation, a NARSAD Young Investigator Grant to LKP, and the Pfeiffer Foundation. LKP is supported in part by a Conte Center grant from the NIH.

short talk

Complete callosal agenesis, pontocerebellar hypoplasia and axonal neuropathy caused by mutation of *AMPD2*

Ashley Marsh

Bruce Lefroy Centre for Genetic Health Research, Murdoch Childrens Research Institute
Department of Paediatrics, The University of Melbourne

Agenesis of the corpus callosum (ACC) is a genetically complex developmental brain malformation associated with diverse neurological deficits that affect ≈1:4500 children. Despite its prevalence, 55-70% of ACC cases have no identifiable cause. We have characterised a large, consanguineous family with five offspring presenting with complete ACC and pontocerebellar hypoplasia (PCH). The offspring are severely affected, their phenotype including profound cognitive impairment, spastic quadriplegia and postnatal onset-microcephaly.

Both fetal and postnatal imaging studies showed complete ACC and hypoplasia of the cerebellum and/or brainstem. Nerve conduction studies revealed an axonal neuropathy. Linkage analysis and whole exome sequencing identified a stopgain mutation in the gene encoding adenosine monophosphate deaminase 2 (*AMPD2*). Experimental studies demonstrated that the mutation segregated with disease and resulted in complete loss of protein. Mutation screening of 42 genetically undiagnosed individuals with related imaging phenotypes failed to identify any candidate variants, suggesting that *AMPD2* mutations are not a common cause of combined callosal and pontocerebellar defects.

The combination of ACC and PCH was recently reported as PCH type 9 (OMIM #615809), secondary to mutations in *AMPD2*. This is the second report of mutations in *AMPD2* as a cause of a callosal abnormality associated with PCH, and is the largest multiplex pedigree described with this disorder. We expand the phenotypic spectrum of emerging PCH disorders to include complete ACC and axonal neuropathy and provide evidence for the prenatal onset of the neurodegenerative process in PCH9.

short talk

A systematic review of corpus callosotomy outcomes in children

David Graham[1,2], Deepak Gill[1,2] and Martin Tisdall[3]

[1] Faculty of Medicine, the University of Sydney, [2] T.Y. Nelson Department of Neurology and Neurosurgery, Children's Hospital at Westmead, [3] Neurosurgery Unit, Great Ormond Street Hospital

Overview: Corpus callosotomy is a palliative treatment for patients with either generalised or multifocal refractory epilepsy with injurious drop attacks. There are a number of systematic reviews of surgical treatment for refractory epilepsy, but to date only one has focused on corpus callosotomy and this was not in the paediatric population.

Methods: Medline, Embase, Web of Knowledge and Scopus were systematically searched for published articles on treatment outcomes of corpus callosotomy for refractory epilepsy. The review of the literature on corpus callosotomy was limited to articles in English or German. Studies were included if the patient population was under 18 at the time of surgery and mean/median follow-up was greater than 1 year. Studies were excluded if resective surgery was also conducted.

Results: A total of 13 papers met inclusion criteria. All papers were retrospective case series, with the exception of one being a prospectively designed retrospective case series. The patient population was significantly heterogeneous, although Lennox-Gastaut Syndrome was the most common diagnosis. There was very little agreement among authors on the definition of a good seizure outcome, with only six papers using the Engel classification. Greater extent of callosotomy was associated with a greater reduction in drop attacks among these papers; 90.2% of total corpus callosotomy patients had a worthwhile reduction in drop attacks compared with 70.5% of patients who underwent partial corpus callosotomy ($p<0.05$). Nevertheless, disconnection syndrome was significantly more likely in total corpus callosotomy (12.5% vs 0%, $p<0.05$). Improvements in quality of life, behaviour and intelligence/development quotient, as well as parental satisfaction, were generally correlated with seizure outcome. There was no post-callosotomy change in the number of anti-epileptic drugs in any of the studies that reported this. Predictors of good outcome included structural abnormalities on MRI and extent of the corpus callosotomy.

Conclusions: Total corpus callosotomy is significantly more likely to result in a reduction in drop attacks but partial corpus callosotomy is unlikely to result in disconnection syndrome. While all of the papers drew a similar conclusion, their retrospective design introduces a risk of over-estimating the effectiveness of corpus callosotomy. It is clear that a case-control trial is warranted.

short talk

A preliminary survey of the fundamental neurocognitive differences among 'cyclical thinking': subgroupings and linear thinking

Fenna Bacchus
Independent Interdisciplinary Researcher

80% of the world's traditional people think 'cyclical' or have a variety of underlying elements of 'cyclical thinking', yet most decisions that directly affect their lives, are made for them by 'linear thinkers'. 'Cyclical thinking' manifests itself in many different cultural specific patterns, likely grounded in the interaction of the neuronal networks of an individual based on his/her cultural experience. This dynamic of the neuronal networks is a determinant of the individual's neuroplasticity. 'Cyclical thinking' is an executive neurocognitive process. This functional process is finalized in the Third functional unit (Luria,1973). Varieties exists in the 'future' in terms of 'time' being 'unknown', seen through 'the present' and the 'past', while non-existent in others. 'Time' is associated with cultural actions, seasons and nature. Transcultural fMRI studies established that one's cultural content can influence neural activity that underlies both high-and low-level cognitive functions (Han et al., 2008). Studies in human subjects revealed this to occur in the lateral intraparietal area (Eagleman et al., 2005), the left parietal cortex (Coulle et al., 2008), functional networks and Dopamine (Droit et al., 2011). Research on rats revealed a neuronal code for extended time in the hippocampus, a neural activity pattern in CA1 to distinguish between time intervals (Mankin et al., 2012). There is need for scientific research to reveal the fundamental neurocognitive differences between 'cyclical' and 'linear' thinking. Is there a different functional interaction between the networks, and if so how, and what are the implications for future corpus callosum research? The potential significance of this study is to establish markers and protocols which will lead to the development of universal models to educate.

short talk

Validation of a novel proverb test for frontal function

Michael Valente[1], Elsdon Storey[2], Helene Roberts[1], Mahima Kapoor[2]
[1]Monash Health, [2]Alfred Health

Interpretation of proverbs has traditionally been used by clinicians as a simple bedside measure of abstraction (ability to derive a higher concept from observed information) . Impairment of the frontal lobe of the brain is thought to lead to the "concrete" interpretation of proverbs, where only the literal meaning of a proverb is accepted. In addition, a well known proverb may only require memory for its interpretation. This study compared the responses of 37 participants with focal brain lesions to 55 control subjects with no brain injury. The test battery administered involved the administration of an MCQ format questionnaire, of 12 novel proverbs intermixed with 6 known proverbs, to participants recruited as inpatients or in the community. All subjects were also examined with the "F,A,S" test of verbal fluency, the 5-point design fluency test, the WAIS-3 similarities subtest and the Stroop test, National Adult Reading Test, and MMSE. Novel Proverbs not only correlated more strongly with each of the executive function measures, but also demonstrated a deficiency of interpretation in the frontal lesioned group (p=0.041), whilst traditional proverbs could not (p=0.118). Novel proverb interpretation was also significantly deficient in the left frontal lobe when compared to the right (p=0.025). Novel proverbs significantly outperformed traditional proverbs on every comparison, and should be considered in future studies.

Funding sources: None

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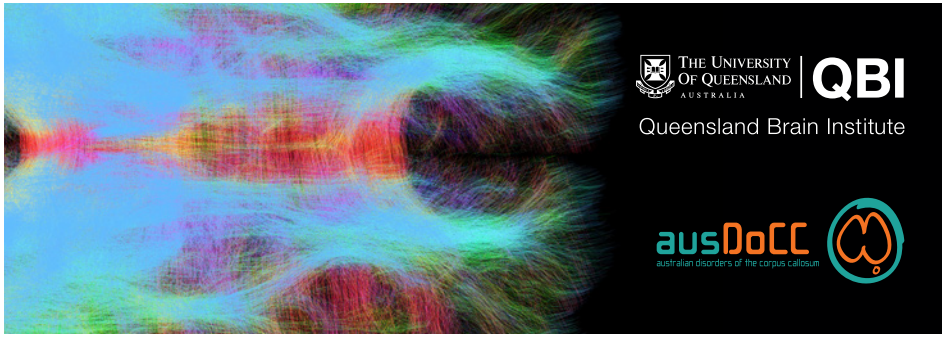

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