NNIDR Australian Dementia Forum
QIMR Berghofer Medical Research Institute
1-3 May 2016

Abstracts

Australian Government
NHMRC National Institute for Dementia Research
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Sunday 1 May 2016

3.00pm - 5.00pm  Registration

5.00pm – 6.30pm  
**Welcome to the 2016 NNIDR Symposium**
Professor John McCallum  
Director, NHMRC – National Institute for Dementia Research and Symposium Chair

**Introduction to the Forum**
Graeme Samuel AC  
President, Alzheimer’s Australia National Board and NNIDR Board

**Welcome to Country**

**Welcome to QIMR**
Professor Michael Breakspear

**Keynote address from the Breakthrough Prize Winner 2015**
**Whole genome analysis of neurodegeneration**
Professor John Hardy  
Head, Department of Molecular Neuroscience and Chair of Molecular Biology of Neurological Disease, UCL Institute of Neurology, United Kingdom

6.30pm - 7.30pm  Networking Drinks – Level 6 outdoor terrace
Monday 2 May 2016

8.00am - 5.00pm  Registration
8.00am – 8.30am  Arrival tea and coffee

**Session 1  Diagnosis/Assessment**

Chairperson  Carol Bennet, CEO, Alzheimer’s Australia National Office

8.30am – 9.20am  Keynote address - Catching dementia – does the evidence stack up?
Professor Glenda Halliday
Director, Sydney Brain Bank, UNSW and NeuRA Neuroscience Research Australia (NeuRA), Australia

9.20am – 9.30am  Consumer discussion
John Doull

9.30am – 9.50am  Prospective Imaging Study of Ageing: Genes, Brain and Behaviour (PISA)
Professor Michael Breakspear & Dr Christine Guo

9.50am – 10.10am  Vascular determinants of dementia
Associate Professor Amy Brodtmann

10.10am – 10.30am  Developing insight into the molecular origins of familial and sporadic frontotemporal dementia and amyotrophic lateral sclerosis
Associate Professor Ian Blair

10.30am – 11.00am  Morning Tea

11.00am – 12.30pm  Rapid fire Talks – NHMRC – ARC Dementia Research Development Fellows
Chair: Professor Colin Masters

12.30pm – 2.00pm  Lunch & Poster Session

**Session 2  Care/Living with Dementia**

Chairperson  Dr Jane Thompson

2.00pm – 2.10pm  Consumer discussion
Christine & Paul Bryden

2.10pm – 2.30pm  What is the 'Australian Community of Practice in Research in Dementia’?
Professor Rob Sanson-Fisher
2.30pm – 2.50pm Moving Forward in the NNIDR Unitary DCRC Model: The Lens of DCRC: CC
Professor Elizabeth Beattie

2.50pm – 3.10pm NHMRC Partnership Centre Dealing with Cognitive and Related Functional Decline in Older People: improving the quality of care for people with dementia and their carers.
Professor Susan Kurrle

3.10pm – 3.30pm Dementia Collaborative Research Centre - Assessment and Better Care
Professor Brian Draper

3.30pm – 4.00pm Afternoon Tea

4.00pm – 5.00pm Panel Discussion: Innovations in care research
Chair: Dr Jane Thompson
Panel members: Professor Rob Sanson-Fisher
Professor Elizabeth Beattie
Professor Brian Draper
Professor Susan Kurrle
Tuesday 3 May 2016

8.00am - 5.00pm Registration
8.00am – 8.30am Arrival tea and coffee

Session 3 Intervention/Treatment

Chairperson Professor Peter Schofield

8.30am – 8.50am Alzheimer's disease: Aβ amyloid is the critical target for primary (pre-AD) and secondary (preclinical) disease-modifying strategies
Professor Colin Masters

8.50am – 9.10am Clem Jones Centre for AGEing Dementia Research - From basic mechanisms to therapeutic interventions
Professor Jürgen Götz

9.10am – 9.30am Structural imaging in dementia with Lewy bodies
Dr Rosie Watson

9.30am – 10.10am Dementia in Indigenous Communities
Professor Leon Flicker
Commentator: Dr Tammy Kimpton
This session will provide opportunity for discussion and perspectives from Indigenous people on dementia in their communities.

10.10am – 11.00am Keynote Address from the Head of Leading German Dementia Research Institute
Professor Pierluigi Nicotera
Scientific Director and Chairman of the Board, German Center for Neurodegenerative Diseases (DZNE), Germany

11.00am – 11.30am Morning Tea

11.30am – 1.00pm Rapid fire Talks – NHMRC – ARC Dementia Research Development Fellows
Chair: Professor John McCallum

1.00pm – 2.40pm Lunch & Poster Session

Session 4 Prevention

Chairperson Professor John McCallum

2.40pm – 3.00pm Dementia Collaborative Research Centre: Early Diagnosis and Prevention – overview and update
Professor Kaarin Anstey
3.00pm – 3.20pm  Maintain Your Brain (MYB): A large scale multi-modal online randomised placebo-controlled intervention to reduce cognitive decline
Professor Perminder Sachdev

3.20pm – 3.40pm  Next Generation Brain Training in the Maintain Your Brain Trial
Professor Michael Valenzuela

3.40pm – 4.00pm  Evaluating dementia risk reduction eHealth tools for the Australian community
Dr Maree Farrow

4.00pm – 4.15pm  Afternoon Tea

4.15pm – 5.00pm  Panel Discussion: Innovations in intervention/treatment and prevention research
Chair: Professor Peter Schofield
Panel members: Professor John Hardy
Professor Pierluigi Nicotera
Professor Glenda Halliday
Christine Bryden
Keynote speakers

Professor John Hardy
Head, Department of Molecular Neuroscience and Chair of Molecular Biology of Neurological Disease
UCL Institute of Neurology United Kingdom

Professor John Hardy is the Head of the Department of Molecular Neuroscience and Chair of Molecular Biology of Neurological Disease at the UCL Institute of Neurology. In 2015, John was awarded the $3 million Breakthrough Prize in Life Sciences for his pioneering research into the genetic causes of Alzheimer’s disease, other forms of dementia and Parkinson’s disease. In recognition of his exceptional contributions to science, he was also elected a Fellow of the Royal Society in 2009.

Professor Glenda Halliday
Director, Sydney Brain Bank, UNSW and NeuRA Neuroscience Research Australia (NeuRA), Australia

Professor Glenda Halliday is an Australian Professor of Neuroscience leading a research program of 70 researchers tackling non-Alzheimer’s neurodegeneration that stems from her work on frontotemporal and motor neurodegenerative syndromes, and Parkinson’s disease. She is also Director of the Sydney Brain Bank. She received her degrees at University of New South Wales, and postdoctoral training at Flinders University prior to an ARC Queen Elizabeth II Fellow and NHMRC research fellowships since 1988, joining NeuRA in 1993. She has published more than 300 research papers and 2 books, and attracted $30m in grant funding. Prof. Halliday is on the editorial boards of 5 international journals, on Scientific Advisory Boards for 3 research institutes (one international), and is a committee member for a number of international organizations, including the International Brain Research Organization (a member organization of UNESCO). She was elected president of the Australian Neuroscience Society (ANS 2006-2007), awarded the 2011 ANS Nina Kondelos Prize, and named a high achiever in Australian Health and Medical Research by NHMRC.

Professor Pierluigi Nicotera
Scientific Director and Chairman of the Board
German Center for Neurodegenerative Diseases (DZNE), Germany

Prof Pierluigi Nicotera, a renowned scientist and leading international expert in the field of neuronal cell death, was appointed Scientific Director of DZNE in April 2009. Prof Nicotera was trained in General Medicine and Cardiology at the University of Pavia, Italy. He obtained his Ph.D. at the Karolinska Institute in Stockholm, where he worked subsequently as associate professor. His research has been centred on the molecular mechanisms that lead to neuronal demise following chronic and acute insults. He was awarded the International Prize Gerolamo Cardano by the Rotary Club of Pavia (Italy) for scientific credits in the research of mechanisms determining neuronal death.
Invited speakers

Professor Michael Breakspear
Group Leader, QIMR Berghofer Medical Research Institute & Coordinator program of Mental Health research

Michael Breakspear is Group Leader at QIMR Berghofer and coordinator of the Program of Mental Health Research. He trained in Medicine and Physics at the University of Sydney and completed his psychiatry training at the BlackDog Institute, Sydney. He combines computational modelling with advanced neuroimaging techniques to study neurodevelopmental and neurodegenerative disorders. He is a psychiatrist in the Brisbane Prison Mental Health Service.

Dr Christine Guo
Team Head, QIMR Berghofer Medical Research Institute

Christine Guo is a Team Head at QIMR Berghofer. She came to Australia in 2013, after finishing her PhD at Stanford University and postdoctoral training at UCSF. Dr Guo’s research experience extends from molecular biology and genetics to systems neuroscience, and the diversity of her techniques ranges from electrophysiology in animal models to clinical neuropsychology and functional neuroimaging. Dr Guo has led several studies using a multidisciplinary approach, combining functional MRI imaging analysis, clinical anatomy and clinical neuropsychology.

Associate Professor Amy Brodtmann
Co-Division Head, Behavioural Neuroscience, NHMRC Clinical Career Development Fellow at the Florey Institute for Neuroscience and Mental Health in Melbourne, Australia; Stroke Neurologist, Austin Health; Cognitive Neurologist and Clinic Director, Eastern Cognitive Disorders Clinic, Box Hill Hospital

Associate Professor Amy Brodtmann is a stroke and cognitive neurologist at Austin Health and director of the Eastern Cognitive Disorders Clinic. She is the recipient of many awards and grants for her work in stroke and dementia, including NHMRC project grants, post-Graduate, post-Doctorate, and clinical Career Development Fellowships, and is CIA on a Dementia Research Team Grant. She sits on the editorial boards of Neurology and the International Journal of Stroke, the board and committee of Alzheimer’s Australia Victoria Dementia Research Grants, is an inaugural member of the Wicking Strategic Review Panel, and is a founding member of the Australian Frontotemporal Dementia Association. Her research focuses on the imaging of brain network degenerations following stroke, post-stroke behavioural syndromes, and the diagnosis and management of focal onset dementias.

Associate Professor Ian Blair
Faculty of Medicine and Health Science, Macquarie University

A/Prof Ian Blair’s research career has focussed on determining the molecular basis of a variety of neurological disorders including ALS/MND, FTD, hereditary sensory neuropathy (HSN), Charcot Marie Tooth disorder (CMT), the spinal cerebellar ataxias (SCA), Joubert syndrome, and bipolar disorder. At Macquarie University, his team works to unravel the molecular and cellular basis of ALS and FTD. His group has played a key role in several ALS/FTD gene discoveries including identification of mutations in the TDP-43 and FUS genes. These discoveries have opened new chapters in ALS/FTD research and led to effective diagnostic tests for ALS, CMT1A and HSN1.
Professor Rob Sanson-Fisher
Director, Health Behaviour Research Group, School of Medicine and Public Health, University of Newcastle

Laureate Professor Rob Sanson-Fisher is Director of the Health Behaviour Research Group, School of Medicine and Public Health, University of Newcastle. An internationally recognised leader in health behaviour research, his work successfully combines behavioural approaches to knowledge translation, health promotion, health service evaluation and chronic disease control. He has published over 470 peer-reviewed journal articles and obtained some 100 competitive research grants, with a total value over $36 million. His research interests include exploring health care provider behaviour and adoption of best evidence practice, and the development, implementation and evaluation of interventions to improve health outcomes for vulnerable population groups.

Professor Elizabeth Beattie
Professor of Aged and Dementia Care, School of Nursing, Queensland University of Technology

Elizabeth Beattie, Professor of Aged and Dementia Care, School of Nursing, Queensland University of Technology, is a psychgeriatric nurse educated in Australia, the UK and the US who has been involved in dementia-focused clinical practice, education and research for 30 years. She directs the Dementia Collaborative Research Centre Carers and Consumers and the Queensland Dementia Training Study Centre. Elizabeth has an international nursing leadership profile and a sustained record of competitive research funding and publication. Her research is focused on improving the quality of care and quality of life of people living with dementia and those who support them.

Professor Susan Kurrle
Geriatrician, Ku-ring-gai Hospital, Sydney

Susan Kurrle is a geriatrician practising at Hornsby Ku-ring-gai Hospital in northern Sydney, and Batemans Bay Hospital in southern NSW, and she holds the Curran Chair in Health Care of Older People in the Faculty of Medicine at the University of Sydney. Since 2012 she has led the NHMRC Partnership Centre on Dealing with Cognitive and Related Functional Decline in Older People. This Centre focuses on research and implementation projects dealing particularly with the care aspect of dementia.

Professor Brian Draper
Professor (Conjoint), School of Psychiatry, University of NSW, and Clinical Director, Academic Department for Old Age Psychiatry, Prince of Wales Hospital

Brian Draper is an old age psychiatrist and Conjoint Professor, School of Psychiatry, UNSW, Sydney Australia. He is Clinical Director, Academic Department for Old Age Psychiatry, Prince of Wales Hospital, Randwick & Deputy Director of the Dementia CRC-ABC at UNSW. He is Board Member, International Psychogeriatric Association. He has published over 300 scientific articles on clinical aspects of dementia and cognitive disorders in the community, hospitals and residential aged care. Other areas of research include late life suicidal behavior, substance use, depression and carer stress.

Professor Colin Masters
Division Head, the Florey Institute

Colin Masters has focused his career on research in Alzheimer’s disease and other neurodegenerative diseases. His work over the last 35 years is widely acknowledged as having had a major influence on Alzheimer’s disease research world-wide, particularly the
collaborative studies conducted with Konrad Beyreuther in which they discovered the proteolytic neuronal origin of the A\(\beta\) amyloid protein which causes Alzheimer’s disease. This work has led to the continued development of diagnostics and therapeutic strategies. More recently, his focus has been on describing the natural history of Alzheimer’s disease as a necessary preparatory step for therapeutic disease modification.

**Professor Jürgen Götz**  
*Inaugural Director, Clem Jones Centre for Aging Dementia Research, Queensland Brain Institute, Brisbane*

Professor Jürgen Götz is the inaugural Director of the Clem Jones Centre for Ageing Dementia Research at the Queensland Brain Institute in Brisbane. Götz studied biochemistry in Switzerland and earned his PhD in immunology with Nobel Laureate Köhler in Germany. After postdoctoral work at UCSF and at Novartis, he became a group leader in Zürich, before moving to Sydney in 2005, and Brisbane in 200x. A major focus of his laboratory is the generation and analysis of transgenic animal models to gain a better mechanistic understanding of Alzheimer’s disease and to develop therapeutic interventions targeting two key molecules in disease, tau and amyloid-beta.

**Professor Leon Flicker**  
*Professor of Geriatric Medicine, University of Western Australia*

Leon Flicker is the inaugural Professor of Geriatric Medicine at the University of Western Australia since 1998. He helped establish a research unit aimed at translational issues focusing on the health needs of older people, the Western Australian Centre for Health and Ageing. He has been interested in the risk factors, assessment and management of the common problems of older people. He also pursues studies on why some older people achieve healthy ageing. He has published over 300 peer-reviewed articles on a wide variety of health issues affecting older people.

**Dr Rosie Watson**  
*Consultant Geriatrician, Royal Melbourne Hospital, Cognitive, Dementia and Memory Service and the Florey Institute, University of Melbourne*

Rosie Watson is a Consultant Geriatrician with current appointments at the Royal Melbourne Hospital, including the Cognitive, Dementia and Memory Service and the Florey Institute of Neurosciences – The University of Melbourne. After completing her clinical training in geriatric medicine in Melbourne, she undertook her PhD studies at the Institute of Ageing and Health, Newcastle upon Tyne, UK investigating the use of MRI techniques in dementia with Lewy bodies and Alzheimer’s disease. Her current research interests include how the use of neuroimaging can help better understand dementia, disease trajectories and improve the clinical diagnosis.

**Professor Kaarin J Anstey**  
*Professor of Psychology and Population Health, Australian National University*

Kaarin J. Anstey is a Professor of Psychology and Population Health at the Australian National University and Director of the Dementia Collaborative Research Centre - Early Diagnosis and Prevention. Her research interests focus on the prevention of dementia, and the impact of cognitive impairment on activities such as driving. Anstey led the first online dementia risk reduction intervention called Body Brain Life that is soon to be trialled in Primary Care. Anstey is a Director of the Alzheimer’s Australia Dementia Research Foundation and the Global Council on Brain Health, an initiative of the US AARP and UK HelpAge organisations.
Professor Perminder Sachev  
*Scientia Professor of Neuropsychiatry, Co-Director, Centre for Healthy Brain Ageing (CHeBA), School of Psychiatry, UNSW, Australia*

Perminder Sachdev AM MBBS MD FRANZCP PhD MFPOA is Scientia Professor of Neuropsychiatry, Co-Director of the Centre for Healthy Brain Ageing (CHeBA), School of Psychiatry, UNSW Australia. He is Director of the Neuropsychiatric Institute (NPI), Prince of Wales Hospital, Sydney. He is a past President of the International Neuropsychiatric Association and the International College of Geriatric Psychoneuropharmacology. He has broad research interests, with a major focus on dementia and cognitive ageing, drug-induced movement disorders, neuroimaging and brain stimulation. He has over 700 peer-reviewed journal papers and 5 books, including one for lay readers (*The Yipping Tiger and other tales from the neuropsychiatric clinic*). He was awarded a Member of the Order of Australia (AM) for service to medical research in the field of neuropsychiatry, and to professional associations at a national and international level, an International Distinguished Fellowship from the APA (USA) and a Deans Award for Outstanding Achievement (Academic) for his outstanding contribution to research and teaching in the Faculty of Medicine.

Associate Professor Michael Valenzuela  
*Leader, Regenerative Neuroscience Group, NHMRC Clinical Career Development Fellow, Brain & Mind Research Centre, University of Sydney*

Michael trained in psychology, medicine and neuroscience and for his PhD work was awarded the Australian Museum’s Eureka Prize for Medical Research. In 2010, he received a NHMRC Excellence Award as the top-ranked Career Development Fellow and in 2012 moved to the University of Sydney to establish the Regenerative Neuroscience Group at the Brain and Mind Centre. Michael’s research focuses on lifestyle-based interventions to help prevent dementia and his team is developing an all-new stem cell therapy. He is the author of the popular science title ‘*Maintain Your Brain*’ and was part of the team that developed BrainyApp.

Dr Maree Farrow  
*Cognitive Neuroscientist & Lecturer, Wicking Dementia Research and Education Centre, University of Tasmania*

Dr Maree Farrow is a cognitive neuroscientist. She is a Lecturer with the Wicking Dementia Research and Education Centre at the University of Tasmania, and a Visiting Fellow with the Centre for Research on Ageing, Health and Wellbeing at The Australian National University. Her current research interests include dementia risk reduction, timely diagnosis and early intervention for cognitive impairment, and translating evidence into programs for the community and primary care. She has developed and evaluated a range of resources and eHealth tools for community education about dementia and risk reduction.
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- **Hardy, John** | Keynote Address from the Breakthrough Prize Winner 2015 - Whole genome analysis of neurodegeneration  
- **Nicotera, Pierluigi** | Keynote Address from the Head of Leading German Dementia Research Institute

## Invited Speakers
*Sorted alphabetically by surname*

- **Anstey, Kaarin** | Dementia Collaborative Research Centre: Early Diagnosis and Prevention – overview and update  
- **Beattie, Elizabeth** | Moving Forward in the NNIDR Unitary DCRC Model: The Lens of DCRC: CC  
- **Blair, Ian** | Developing insight into the molecular origins of familial and sporadic frontotemporal dementia and amyotrophic lateral sclerosis  
- **Breakspear, Michael** | Prospective Imaging Study of Ageing: Genes, Brain and Behaviour (PISA)  
- **Brodtmann, Amy** | Vascular determinants of dementia  
- **Draper, Brian** | Dementia Collaborative Research Centre - Assessment and Better Care  
- **Farrow, Maree** | Evaluating dementia risk reduction eHealth tools for the Australian community  
- **Flicker, Leon** | Dementia in Indigenous Communities  
- **Götz, Jürgen** | Clem Jones Centre for Ageing Dementia Research - From basic mechanisms to therapeutic interventions  
- **Guo, Christine** | Prospective Imaging Study of Ageing: Genes, Brain and Behaviour (PISA)  
- **Kurrle, Susan** | NHMRC Partnership Centre Dealing with Cognitive and Related Functional Decline in Older People: improving the quality of care for people with dementia and their carers  
- **Masters, Colin** | Alzheimer’s disease: Aβ amyloid is the critical target for primary (pre-AD) and secondary (preclinical) disease-modifying strategies  
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Yassi, Nawaf | Vascular Cognitive Risk Score: quantifying the vascular burden in Alzheimer’s disease

Yates, Mark | National Roll Out and Evaluation of the Dementia Care in Hospitals Program

Yates, Paul | Untreated Hypertension is Associated with Longitudinal Aβ Accumulation over Six Years: Results from the AIBL Study of Ageing

Yates, Paul | Association of Cerebrovascular disease and Alzheimer’s disease Biomarkers with and Longitudinal Cognitive Decline
**Keynote Speaker Abstracts**

**Professor Glenda Halliday**  
Neuroscience Research Australia AUSTRALIA  
**Email:** g.halliday@neura.edu.au  
Keynote Presentation  
**Theme:** 1. Diagnosis/Assessment  
Monday 2 May 2016  
8.30am – 9.20am

**Catching dementia – does the evidence stack up?**

Glenda Halliday*¹

1 UNSW Medicine & Neuroscience Research Australia, Sydney, Australia

Prions are proteins that can take on an infectious form and spread through the brain of anyone infected, and also can infect others if ingested. After infection, there is a long incubation period and the infectious form of the protein builds up in the brain, the neurons swell and burst, and the brain becomes inflamed. It is now apparent that the spread of pathological forms of normal brain proteins in association with inflammation occurs in many neurodegenerative diseases, and recent publications have suggested that pathological proteins can be transmitted through contact with already infected brain material. This has now been suggested for Alzheimer’s disease, but the data is strongest for multiple system atrophy (MSA), which has some similarities to prion disease. There include considerable inflammation, considerable neuronal death, and transmission along white matter tracts. Compared to Parkinson’s disease, MSA is usually rapidly progressive, similar to prion disease. Unlike Parkinson’s disease, extracts from the brains of patients with MSA appear to be able to transmit MSA pathology to particular genetic mice, and then the aggregates from these mice can also be transmitted in similar mice [1]. However, unlike prions, not all mice can be infected [1], indicating that a predisposition is important for the phenomenon. What is it that could predispose people to MSA? Genetic polymorphisms in the prion protein predispose people to prion diseases, although recent data suggests that common genetic variations predisposing to MSA are unlikely [2]. There is some evidence that compared with Parkinson’s disease, patients with MSA are more likely to have similar polymorphisms in the prion protein gene [3]. They also have polymorphisms in genes involved in oxidative stress, mitochondrial dysfunction, inflammatory processes, as well as parkinsonism- and ataxia-related genes [4]. It will be important to determine what may predispose people to the more rapidly progressive alpha-synucleinopathy of MSA, or other forms of neurodegeneration.


**Professor John Hardy**

**Email:** r.williamson@unimelb.edu.au  
Keynote Presentation  
**Theme:** No Theme Allocated  
Sunday 1 May 2016  
5.40pm – 6.30pm

**Abstract to come**
Professor Pierluigi Nicotera

Email: pierluigi.nicotera@dzne.de
Keynote Presentation
Theme: 3. Intervention/Treatment
Tuesday 3 May 2016
10.10am – 11.00am

Abstract to come
Invited Speaker Abstracts

Professor Kaarin Anstey
Centre For Research On Ageing, Health & Wellbeing, ANU
Email: kaarin.anstey@anu.edu.au
Oral Presentation
Theme: 4. Prevention
Tuesday 3 May 2016
2.40pm – 3.00pm

Dementia Collaborative Research Centre: Early Diagnosis and Prevention – overview and update

Professor Kaarin Anstey*¹

1 Centre for research on ageing, health and wellbeing, ANU, Australia

The Dementia Collaborative Research Centre: Early Diagnosis and Prevention (DCRC-EDP) focuses on translating basic research related to early detection of dementia and risk reduction, into practical tools, interventions and policy. Compared with other areas of population health, dementia prevention is relatively new and methodologies and effective interventions are still being developed. The DCRC-EDP has contributed to global advances in early diagnosis and prevention at several levels. The groundbreaking AiBL study has been at the forefront of biomarker development in Alzheimer’s disease. Systematic reviews of cohort studies funded by DCRC have led to knowledge that has informed government policies internationally as well as intervention design. Risk assessment tools have been developed that are now widely in use online and as apps. The DCRC has supported the development of interventions to reduce risk of dementia. Recently completed interventions have been conducted in nutrition, lifestyle modification, and dementia literacy. Interventions are in progress in cognitive training, sitting reduction and physical activity. The presentation provides an overview of the program and discusses the challenge and opportunity of knowledge translation in this field.

Professor Elizabeth Beattie
QUT
Email: elizabeth.beattie@qut.edu.au
Oral Presentation
Theme: 2. Care/Living With Dementia
Monday 2 May 2016
2.30pm – 2.50pm

Moving Forward in the NNIDR Unitary DCRC Model: The Lens of DCRC:CC

Elizabeth Beattie*¹

1 Queensland University of Technology, Brisbane, Australia

The research agenda of the Dementia Collaborative Research Centre: Carers and Consumers has historically focused on basic and applied studies and knowledge translation designed to meet gaps in understanding of, or provide strong evidence for interventions designed to improve, the issues affecting the care, support and quality of life of people living with dementia and carers. Pressing issues include: systematic reviews of meaningful activities and carer resilience, supporting carer wellness, respite care options, home care support, decision making about care choices, palliative care, responsive interventions for behavioural
and psychological symptoms and the use of technologies such as companion robots to connect and enrich lives. Projects undertaken by DCRC: CC partners span all contexts of care (community, RACF, acute care) and have been determined in increasingly close consultation with the Consumer Dementia Research Network. In the absence of a cure for dementia, and with limited effective treatments, improving care, carer support and QoL across the dementia trajectory is critical. A highlight of our achievements is the completion of the first nationally representative study of Quality of Life for people living with dementia in residential aged care (N =53 facilities across 5 states and 1 territory, including 430 people with dementia, over 400 family carers and over 900 staff members). Australia now has world class benchmarking on a set of outcome variables essential to improving daily QoL: depression, behavioural symptoms, activities, staff factors, person centred care uptake and environmental characteristics, in addition to perceived QoL from three perspectives (person with dementia, family and staff). We are currently disseminating study outcomes in local, national and international fora and hope to use study findings to develop targeted research and KT projects consistent with NNIDR priorities. We look forward to uniting, extending and energising the pool of investigators skilled and committed to care focused applied research and KT around these NNIDR priorities and growing the next generation of world class care researchers. Challenges for care-focused researchers from any original DCRC within the unitary DCRC model will include creating and sustaining new collaborations and synergies across the dementia initiative to best respond to and meet priorities.

A/Professor Ian Blair
Macquarie University
Email: ian.blair@mq.edu.au
Oral Presentation
Theme: 1. Diagnosis/Assessment
Monday 2 May 2016
10.1 0am – 10.30am

Developing insight into the molecular origins of familial and sporadic frontotemporal dementia and amyotrophic lateral sclerosis

Julie Atkin1, Roger Chung1, Gilles Guillemin1, Lezanne Ooi2, William Wilson3, Mark Molloy1, Justin Yerbury2, Nicholas Cole1, Tim Karl4, Carol Dobsom-5, Denis Bauer1, Dominic Rowe1, Gaetan Burgio6, John Kwok2, Kelly Williams1, Roger Pamphlett7, Ian Blair1

1 Macquarie University
2 The University of Wollongong
3 CSIRO
4 Western Sydney University
5 Neuroscience Research Australia
6 Australian National University
7 University of Sydney

There is strong evidence that frontotemporal dementia (FTD) and amyotrophic lateral sclerosis (ALS) represent a spectrum of neurodegenerative disease with common origins. A combined study of FTD/ALS patient cohorts will provide greater power to identify these shared molecular origins. We aim to discover gene variants that cause, predispose, or modify onset and progression of inherited and sporadic FTD/ALS, and validate and study our discoveries in new cell and animal models of these disorders. In addition to familial genetic studies in FTD/ALS, we are performing large-scale whole-genome sequencing in sporadic cases to identify risk alleles. Epigenetic studies are also underway, which coupled with WGS data, aim to identify modifiers of disease including the age-of-onset and rate of disease progression. In vitro studies will assess the pathogenicity of these candidate sequence variants and transgenic studies in zebrafish will inform the development of mouse models.
Using our comprehensive patient biobank, we are generating and investigating a bank of fibroblast-derived iPSC lines from patients with candidate gene variants. Coupling cellular and proteomic analysis with genetic, epigenetic and clinical data provides a powerful approach to unravel the molecular origins of these disorders.

**Professor Michael Breakspear**
QIMR Berghofer
Email: mjbreaks@gmail.com
Oral Presentation
Theme: 1. Diagnosis/Assessment
Monday 2 May 2016
9.30am – 9.50am

**Prospective Imaging Study of Ageing: Genes, Brain and Behaviour (PISA)**

Professor Michael Breakspear*¹, Dr Christine Guo*¹
1 QIMR Berghofer Medical Research Institute, Brisbane, Australia

While the burden of dementia occurs late in life, neuropathology accumulates for decades prior to dementia onset. Disease modifying interventions have the greatest potential to avert neuronal death and later disease burden if introduced during this crucial window, well before the onset of clear cognitive decline. To reduce Australia’s future dementia burden, the PISA study aims to develop methods to identify those Australians at the very early stage of dementia. I will introduce our strategies in establishing a younger healthy cohort enriched for high risk of dementia. Risk prediction is enabled by recent advances in genome wide association study (GWAS) studies on Alzheimer’s study and the computation of polygenetic risk scores. To elucidate the neurobiology of prodromal Alzheimer’s disease, we will use cutting-edge bioinformatics, brain imaging, cognitive testing and lifestyle monitoring to follow up this cohort longitudinally. Overall, PISA aims to (1) Discover biological markers of early neuropathology; (2) Identify modifiable risk factors, and (3) Establish the very early phenotypic and neuronal signs of disease conversion.

**A/Professor Amy Brodtmann**
The Florey Institute of Neuroscience and Mental Health
Email: amy.brodtmann@florey.edu.au
Oral Presentation
Theme: 1. Diagnosis/Assessment
Monday 2 May 2016
9.50am – 10.10am

**Vascular determinants of Dementia**

Associate Professor Amy Brodtmann*¹
1 The Florey Institute of Neuroscience and Mental Health Australia

The evidence is compelling: vascular burden is the greatest determinant of late life cognition. This risk is not just for vascular dementia. In 2013, the American Alzheimer’s Association conceded that the volume of evidence linking vascular risk and dementia was conclusive, announcing that vascular contributions to cognitive decline were a priority area of research focus for their international grant program. All late-onset dementia syndromes, especially Alzheimer’s disease (AD), are driven or exacerbated by vascular brain burden. We
aim to examine how vascular burden causes dementia. Understanding the mechanisms means that we can prevent and treat the global epidemic of dementia.

**Professor Brian Draper**  
*University of NSW*  
*Email: b.draper@unsw.edu.au*  
*Oral Presentation*  
**Theme:** 2. Care/Living With Dementia  
**Monday 2 May 2016**  
**3.10pm – 3.30pm**

**Dementia Collaborative Research Centre – Assessment and Better Care**

Professor Brian Draper*¹

1 University of New South Wales, Australia

The Dementia Collaborative Research Centre – Assessment and Better Care (DCRC-ABC) was established in 2006 under the National Dementia Initiative. The pillars of our centre continue to be research, collaboration, capacity building, consumer involvement and knowledge translation. The foci of our collaborating research partners across Australia have been assessment, treatment, primary care, nursing, community care, acute care, behavioural and psychological symptoms of dementia (BPSD), transitions in care, environment and technology, special groups (e.g. Aboriginal & Torres Strait Islanders, CALD, Intellectual disability, Young Onset Dementia) and physical comorbidity. DCRC-ABC has received $11.8 million in funding over last 9.5 years with approximately half going to internal research and knowledge translation projects within UNSW and half to research partners. We have a local consumer advisory committee and have collaborated with Alzheimer’s Australia’s Consumer Dementia Research Network and its National Quality Dementia Care Network. Other important collaborations have been with the Dementia Training Study Centres, the Dementia Behaviour Management and Advisory Services, the Australian Institute of Health and Welfare, the Australian Department of Health, 10 University partners and various service providers. There have been 89 Centre based (61 completed, 28 in progress) and 59 Partner based (45 completed, 14 in progress) projects. Our model has been to fund smaller/ pilot projects that can be the basis for larger grants usually from NHMRC and Dept. of Health. Three examples follow. DCRC funding for a review of non-pharmacological funding of management of BPSD resulted in an NHMRC project grant which was successfully completed. Collaboration between Centre researchers and AIHW led to the NHMRC funded Hospital Dementia Services Project. A pilot of humour therapy in nursing homes led to the NHMRC-funded SMILE study which was the basis for the Arts Health Institute establishment and adoption of humour therapy across ≈100 nursing homes in Australia. Capacity building has included part or full-time funding for 14 PhD and 5 Masters Students, top-up scholarships for PhDs, travel scholarships for early career researchers and training for service providers to evaluate their innovative programs. Recent short-term arrangements for funding have limited DCRCs’ ability to fund projects and capacity building over last three years. The process of combining the three DCRCs into a unitary DCRC is underway.

**Dr Maree Farrow**  
*Wicking Dementia Research & Education Centre, University of Tasmania*  
*Email: maree.farrow@utas.edu.au*  
*Oral Presentation*  
**Theme:** 4. Prevention  
**Tuesday 3 May 2016**  
**3.40pm – 4.00pm**
Evaluating dementia risk reduction eHealth tools for the Australian community

Maree Farrow¹,²

¹ Wicking Dementia Research and Education Centre, University of Tasmania
² Centre for Research on Ageing, Health and Wellbeing, The Australian National University

Several modifiable health and lifestyle factors are consistently associated with the risk of developing dementia. It has been estimated that millions fewer cases worldwide would result from reducing the population incidence of dementia risk factors. However, knowledge in the general population is low, especially about the link between cardiovascular risk factors and brain health. In conjunction with Alzheimer’s Australia’s dementia risk reduction campaigns, we have undertaken a program of research to gain an understanding of Australians’ perceptions, knowledge and health behaviours related to dementia risk reduction, and to determine whether the advice provided by eHealth resources (websites and smart device apps) can improve knowledge, increase motivation to adopt healthier lifestyles, and change behaviour. Surveys of consumers’ perceptions of a dementia risk reduction website and community presentation revealed many were already concerned about cognitive decline and their immediate risk of dementia and would like personalised risk assessments and risk reduction programs. Brief dementia risk reduction eHealth interventions were found to achieve improved knowledge and increased motivation, as well as improvements in self-rating of health behaviours for mental, social and physical activity and diet. An evaluation of user experiences and perceptions of the online Australian National University Alzheimer’s Disease Risk Index found two thirds of respondents were likely to change their behaviour based on their results. Strong community interest in access to dementia risk assessment and risk reduction information was confirmed by large responses to recruitment advertisements for these studies. Findings suggest those who already have healthy lifestyles are more likely to be attracted to dementia risk reduction resources and more work is needed to reach those most at risk. Findings also suggest eHealth resources can make a difference, raising awareness and also improving behaviour, but that interactive elements may need to be individually targeted and provide structured guidance.

Professor Leon Flicker
Western Australia Centre for Health & Ageing, UWA
Email: leon.flicker@uwa.edu.au
Oral Presentation
Theme: 3. Intervention/Treatment
Tuesday 3 May 2016
9.30am – 10.10am

Dementia in Indigenous Communities

Leon Flicker*¹

1 Western Australia Centre for Health and Ageing, University of Western Australia, Australia

Indigenous peoples represent up to 5% of the world’s population (almost 400 million people), representing thousands of individual cultures and language groups. In Australia the Indigenous population is undergoing rapid ageing despite life expectancy being considerably lower than the non-Indigenous population. In 2008, we reported that the prevalence of dementia in older Indigenous people in the Kimberley area of Western Australia was 12.4%, some five times greater than the overall Australian population rate of
2.4% (age standardized). Since then, another study has demonstrated a similarly high prevalence of dementia in rural and urban dwelling Aboriginal people in NSW. In both studies the most common specific form of dementia was Alzheimer’s type dementia. Five year follow-up of the original study performed in the Kimberley has confirmed the stability of diagnosis and risk factors associated with dementia and cognitive impairment that include, age, head injury, hypertension and stroke. Of the original participants with dementia 77% had died by the 5 year follow-up. Overall, the major predictors of mortality included age (Hazard ratio (95% CI)), 1.03 (1.01, 1.05), male sex, 2.17 (1.39, 3.39), poor mobility, 2.11 (1.34, 3.30) and cognitive impairment 2.19 (1.31, 3.65). Dementia and cognitive impairment are major problems for Australian Indigenous people. Provision of care of Indigenous people with dementia needs to be culturally sensitive, taking into consideration the whole community and not only individuals and carers.

Professor Jürgen Götz
The University of Queensland, Queensland Brain Institute, Clem Jones Centre for Ageing Dementia Research
Email: j.goetz@uq.edu.au
Oral Presentation
Theme: 3. Intervention/Treatment
Tuesday 3 May 2016
8.50am – 9.10am

Clem Jones Centre for Ageing Dementia Research – From basic mechanisms to therapeutic interventions

Professor Jürgen Götz*¹
1 The University Of Queensland, Queensland Brain Institute, Clem Jones Centre For Ageing Dementia Research

In an ageing society, we are in a race against time to find new, effective treatments for Alzheimer’s disease, the most prevalent of all dementias. At the Clem Jones Centre for Ageing Dementia Research (Queensland Brain Institute), we have the depth of talent to research the disease at its molecular, cellular and systems level and translate these discoveries into health outcomes. In my presentation I will highlight selected research activities, driven by a mix of curiosity and innovation, including the vital work underway into a novel ultrasound-based technology that can be employed for the clearing of toxic protein aggregates and the delivery of biologics past the blood-brain barrier.

Dr Christine Guo
QIMR Berghofer
Email: christine.guo@qimrberghofer.edu.au
Oral Presentation
Theme: 1. Diagnosis/Assessment
Monday 2 May 2016
9.30am – 9.50am

Prospective Imaging Study of Ageing: Genes, Brain and Behaviour (PISA)

Professor Michael Breakspear*¹, Dr Christine Guo*¹
1 QIMR Berghofer Medical Research Institute, Brisbane, Australia
While the burden of dementia occurs late in life, neuropathology accumulates for decades prior to dementia onset. Disease modifying interventions have the greatest potential to avert neuronal death and later disease burden if introduced during this crucial window, well before the onset of clear cognitive decline. To reduce Australia’s future dementia burden, the PISA study aims to develop methods to identify those Australians at the very early stage of dementia. I will introduce our strategies in establishing a younger healthy cohort enriched for high risk of dementia. Risk prediction is enabled by recent advances in genome wide association study (GWAS) studies on Alzheimer’s study and the computation of polygenetic risk scores. To elucidate the neurobiology of prodromal Alzheimer’s disease, we will use cutting-edge bioinformatics, brain imaging, cognitive testing and lifestyle monitoring to follow up this cohort longitudinally. Overall, PISA aims to (1) Discover biological markers of early neuropathology; (2) Identify modifiable risk factors, and (3) Establish the very early phenotypic and neuronal signs of disease conversion.

Professor Susan Kurrle
NHMRC Cognitive Decline Partnership Centre
Email: kurrle@bigpond.com
Oral Presentation
Theme: 2. Care/Living With Dementia
Monday 2 May 2016
2.50pm – 3.10pm

NHMRC Partnership Centre Dealing with Cognitive and Related Functional Decline in Older People: improving the quality of care for people with dementia and their carers

Professor Susan Kurrle*¹, Jennifer F Thompson¹

1 NHMRC cognitive decline partnership centre, Australia

The NHMRC Partnership Centre Dealing with Cognitive and Related Functional Decline in Older People (known as the Cognitive Decline Partnership Centre – CDPC) is the first of the NHMRC Partnership Centres for Better Health. In 2013, $25M in funding was made available by the National Health and Medical Research Commission (NHMRC), Department of Health (DOH), Brightwater Care Group, HammondCare, Helping Hand Aged Care, and Alzheimer’s Australia (the Funding Partners) for creation of this Centre, which was envisaged to utilise initial funding within 5 years, with a second 5 years of funding potentially available. NHMRC mandated objectives for NHMRC Partnership Centres are: implementation of research informed change; synthesis and dissemination of existing research; undertaking collaborative new research; and capacity building. Capacity building, it should be noted is inherent across most activities. CDPC Investigator teams bring together government, researchers, consumers, aged care providers, and practitioners. The fields of medicine, nursing, allied health, pharmacy, social work, law, sociology, health economics, and change management are represented within the CDPC membership. Institutional and individual involvement spans 5 States (NSW, WA, SA, VIC, QLD), the ACT, 10 Universities, numerous other research institutions, aged care facilities, and multiple professions. CDPC Consumer representatives include people with dementia and their carers, drawn from the Alzheimer’s Australia Consumer Dementia Research Network (CDRN). The reach and influence of CDPC research spans urban, rural and regional areas and examples from 28 CDPC Activities funded to date are:

Implementation - the Confused Hospitalised Older Persons) (CHOPs) program enabling acute hospital staff in identifying, treating, and caring for older people presenting with confusion; and the implementation of Vitamin D supplements for residents of aged care facilities.

Synthesis and dissemination - development and launch of the Clinical Guidelines for Management of Dementia in Australia; and development and dissemination of a national approach to advance care planning in dementia.

Collaborative new research - the effect of regulations on care provided in residential care; understanding long-term care
configurations for older people with cognitive decline; and optimizing the quality use of medicines for people with cognitive and related functional decline. Capacity building - activities include workshops on health economics, policy development, and technology and telehealth. CDPC research funding has also enabled support through employment for numerous early career researchers since its inception.

Professor Colin Masters
The Florey Institute
Email: c.masters@unimelb.edu.au
Oral Presentation
Theme: 3. Intervention/Treatment
Tuesday 3 May 2016
8.30am – 8.50am

Alzheimer’s disease: Aβ amyloid is the critical target for primary (pre-AD) and secondary (preclinical) disease-modifying strategies

Professor Colin Masters*¹
1 The Florey Institute, Australia

There are two basic forms of Alzheimer’s disease (AD). The common (>95%) form is sporadic, and is caused by the failure to clear the Aβ peptide (mean age at onset 80 years). The rare (< 5%) autosomal dominant familial form is caused by the over-production of this peptide (mean age at onset 45 years). In both forms, the kinetics of Aβ accumulation are similar, taking about 30 years to accumulate approximately 10mg of Aβ. Thus we estimate that sporadic AD starts about the age of 50 years and the autosomal dominant form starts about 15 years of age. A disease modifying strategy will be needed to keep the total brain Aβ burden close to normal levels (<2.5 mg) and prevent/delay onset of both forms. Such a strategy may encompass lowering production, stabilizing / neutralizing the toxic Aβ species, and promoting it’s clearance from the brain. Interventions targeting Aβ in the earliest / mildest stages of the natural history of AD are beginning to show efficacies.

Professor Perminder Sachdev
UNSW
Email: p.sachdev@unsw.edu.au;angie.russell@unsw.edu.au
Oral Presentation
Theme: 4. Prevention
Tuesday 3 May 2016
3.00pm – 3.20pm

Maintain Your Brain (MYB): A large scale multi-modal online randomised placebo-controlled intervention to reduce cognitive decline

Professor Perminder Sachdev*¹
1 UNSW, Australia

Background: A number of health and lifestyle-related modifiable risk factors for dementia have been identified. It is not known whether addressing these risk factors at the population level will lead to a reduced incidence of dementia as indicated by a slowed rate of cognitive decline in a middle-aged to older population. Methods: MYB is a randomised controlled trial of multiple online interventions designed to target modifiable risk factors (physical inactivity, cognitive inactivity, depression, overweight and obesity, and poor diet)
for Alzheimer’s disease and dementia. Four intervention modules (physical activity, diet and nutrition, cognitive training and depression, each of 3 months’ duration) will be customised to individual risk profiles and delivered over 12 months online through the newly developed MYB eHealth platform. Booster sessions and monitoring will continue for four years. Follow-up assessments measuring these risk factors and cognition will be completed annually for 4 years. The comparison control group will receive basic psychoeducation with up-to-date information on risk factors and care as usual. The sample will be drawn from the NSW 45-and-Up study, with target N=18,000 and will include individuals aged 55-75 years, with at least 1 risk factor on the ANU Dementia Risk Index, without history of dementia or other neurological or major psychiatric disorder, access to computer and home internet, and reasonable proficiency in English. **Co-primary outcomes:** change in cognition as measured by Cogstate Plus, and incident dementia. **Multiple secondary outcomes and linkage to electronic health records and databases.**

**Results and Conclusions:** The pilot study will begin in mid-2016 and the trial launched in early 2017. MYB is the largest dementia prevention trial internationally and introduces novel elements and state-of-the-art concepts in mode of delivery and behavioural change theory that make it unique among existing strategies. It has the potential to be scalable at the population level and make a significant impact on the burden of dementia internationally.

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**Professor Rob Sanson-Fisher**

*University of Newcastle*

**Email:** Rob.Sanson-Fisher@newcastle.edu.au

**Oral Presentation**

**Theme:** 2. Care/Living With Dementia

Monday 2 May 2016

2.10pm – 2.30pm

**What is the ‘Australian Community of Practice in Research in Dementia’?**

Professor Rob Sanson-Fisher*¹

1 University of Newcastle, Australia

The Australian Community Of practice in Research in Dementia (ACcORD) program will conduct research aimed at improving the wellbeing and health outcomes of people with dementia and those who support and care for them. The ACcORD program is made up of a national group of researchers, consumers, legal and clinical experts, who will work together to undertake this research. This program is one of 6 funded by the National Health and Medical Research Council for the period 2016-2020 under the Dementia Research Team Grants scheme. L/Prof Sanson-Fisher will provide an overview of the research and highlight ways people can get involved in this work.

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**A/Professor Michael Valenzuela**

*Brain and Mind Centre*

**Email:** michael.valenzuela@sydney.edu.au

**Oral Presentation**

**Theme:** 4. Prevention

Tuesday 3 May 2016

3.20pm – 3.40pm

**Next Generation Brain Training in the Maintain Your Brain Trial**

Associate Professor Michael Valenzuela*¹
Brain training is an effective method for improving cognitive outcomes in older adults with and without cognitive impairment. However, efficacy wanes quickly after training offset and current technology is only effective when supervised in a group environment. Therefore, the main obstacles for establishing a genuine role for brain training in dementia prevention are scalability and sustained long-term engagement. We are tackling these challenges in the Maintain Your Brain Trial through co-development of next generation brain training technology together with our industry partner, NeuroNation. This includes innovation in training regimen, exercise content and context of online delivery. Based on our research, we will employ distinct loading, peak-finding and maintenance doses in order to target long-term cognitive gains without over-burdening participants. Cognitive exercises are to be designed on “gaming for training” principles that prioritize affective immersion and customized learning architecture. Our aim is for participants to experience their assigned tasks as fun, challenging and inherently rewarding. The new “Training with Friends” eHealth platform is intended to solve the main implementation challenge, the inefficacy of unsupervised home-alone training. From the consumer’s perspective, the platform will allow training “alongside” other participants using Skype-like audiovideo streaming, post-training debriefs, self-identified opt-in groups and competitive leader boards. From the clinician’s side, the platform will flag participants who are struggling on exercises or protocol adherence, such that an online trainer can be “called up” during training sessions to instruct, mentor and motivate. Further, we will engage the participant’s wider circle of friends and family, inviting them to upload motivational messages (text, photos, video) delivered to participants at key progress milestones. Overall, the Maintain Your Brain strategy is to socialize the online brain training experience, connecting participants with like-minded peers, expert trainers and their own social network for long term engagement and hence sustained cognitive benefit.

Dr Rosie Watson
Florey Institute of Neurosciences and the Department of Medicine - The Royal Melbourne Hospital
Email: rosie.watson@florey.edu.au
Oral Presentation
Theme: 3. Intervention/Treatment
Tuesday 3 May 2016
9.10am – 9.30am

Structural imaging in dementia with Lewy bodies
Rosie Watson*

1 Florey Institute of Neurosciences and the Department of Medicine – The Royal Melbourne Hospital

Dementia with Lewy bodies is a common form of dementia in older age, but it can be difficult to distinguish from other dementias. Due to overlapping clinical features, people with DLB are often misdiagnosed as having Alzheimer’s disease (AD) during life, and therefore investigating the neurobiological changes in vivo using MRI can assist our understanding of the common characteristics of the disease and could help improve the clinical diagnosis.

One hundred and six older participants (35 DLB, 36 AD and 35 healthy controls) underwent 3 Tesla MR scanning along with clinical and neuropsychological assessments. The FreeSurfer analysis package was used to investigate patterns of cortical thinning and subcortical volume differences across groups. Compared to controls, AD was associated with significant cortical thinning in the bilateral temporal and parietal regions extending into the frontal lobes, while in DLB; cortical thinning was less diffuse with focal areas of change mainly affecting bilateral...
posterior structures. Analysis of subcortical structures indicated that whilst not significantly different from AD, volumetric loss relative to healthy subjects in basal ganglia and brainstem were more pronounced in DLB. For similar levels of dementia severity, DLB was associated with less cortical thinning, relative preservation of the medial temporal lobe and more subtle subcortical volume changes. Future work needs to concentrate on longitudinal imaging studies with pathological correlates to evaluate the influence and interaction of differing pathologies on the DLB clinical syndrome, disease trajectories and patient outcomes. This may assist with treatment development and appropriate stratification for clinical trials.

Poster Abstracts

Dr Scott Ayton
Florey Institute of Neuroscience and Mental Health
Email: scott.ayton@florey.edu.au
Poster Presentation
Theme: 1. Diagnosis/Assessment
Poster number: P001

Cerebrospinal ferritin determines the risk of cognitive decline in pre-clinical APOE-Îµ4 carriers

Scott Ayton*1,2, Noel Faux1,2, Ashley Bush1,2
1 Florey Institute on Neuroscience and Mental Health, Victoria, Australia
2 CRC for Mental Health, Victoria, Australia

Introduction: The Îµ4 allele of APOE confers the greatest risk for Alzheimer’s disease (AD), however the pathologic mechanisms for this are uncertain, as are the reasons for variable disease penetrance. We recently that CSF ferritin, a reporter of brain iron load, predicted longitudinal outcomes of AD comparable to that of the combined performance of CSF tau and Aβ (an established biomarker for AD). Here, we explored whether CSF ferritin levels could be use in combination with other AD risk factors such as APOE Îµ4 to predict early cognitive changes.

Methods: Subjects classified as Cognitively Normal (CN; n=91), Mild Cognitive Impaired (MCI; n=144) and AD (n=67) were recruited to the Alzheimer’s Disease Neuroimaging Initiative (ADNI) study, which collected demographic, genetic (e.g. APOE isoform), clinical (e.g. cognition) and biochemical (e.g. CSF) information at baseline, and clinical appraisal was performed annually for up to seven years. Results & Discussion: Baseline CSF ferritin was associated with a marked acceleration of cognitive deterioration over 7 years in CN subjects carrying the APOE Îµ4 risk allele (RAVLT: P=0.0008, r²=0.21), but there was no association in Îµ4-ve subjects. In contrast, the ratio of tau and Aβ levels in CSF was more modestly associated with cognitive change (RAVLT: P=0.039, r²=0.005), and did not vary according to Îµ4 genotype. A threshold value of 6.6 ng/ml CSF ferritin predicted stable and cognitively declining Îµ4+ve CN subjects with an accuracy (area under receiver-operator characteristic) of 0.96. Conclusions: CN Îµ4+ve subjects with comparatively low ferritin may not deteriorate in the foreseeable future, which could explain why ~30% of Îµ4+ve subjects do not develop AD. The effect of CSF ferritin on cognitive deterioration was remarkably different between APOE Îµ4 carriers and non-carriers, which provides insight into the pathomechanisms of this major risk factor, and supports lowering brain iron levels as a therapeutic strategy.

Dr Amee Baird
Macquarie University
Email: amee.baird@mq.edu.au
Poster Presentation

NNIDR Australian Dementia Forum www.nnidr2016.com
Music, memory and me: An investigation of the beneficial effects of music on autobiographical memory and self identity in persons with dementia

Amee Baird*1,2, William Thompson1,2

1 Department of Psychology, Macquarie University
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Introduction: Music is highly effective at evoking personal (autobiographical) memories. Autobiographical memory is closely linked to our self identity. Our objective is to explore music evoked autobiographical memories (MEAMs) and the impact of music on self identity in persons with mild cognitive impairment (MCI), dementia of any type, and healthy elderly. We aim to characterise MEAMs compared with memories evoked by photos and to explore the impact of favourite music on self-identity. Methods: 25 persons with MCI or dementia (any type) and 25 aged matched healthy persons will recall personal memories during music listening or photo viewing. Stimuli will be 15 famous songs (rated number 1 in Australia) and 15 photos of famous world events from 3 time periods, when participants were aged (a) 10-30 years (reminiscence bump), (b) 31-50 years, and (c) 51-70+ years. A measure of self-identity that involves generating ‘I am’ statements (e.g. I am a grandfather) and associated memories while listening to their favourite music (or an auditory control), in addition to measures of cognition and music engagement will also be completed. Anticipated results: We predict that compared with participants with MCI or dementia, healthy participants will produce more specific and frequent MEAMs, particularly for the recent lifetime period (50+ years), but frequency or type of MEAMs from aged 10-30 years (reminiscence bump) will not differ between the groups. Furthermore, compared with photo evoked memories, MEAMs will be more specific and contain higher emotional content, and favourite music should facilitate production of self-identity statements and associated memories in all participants. Discussion: This study will be the first systematic characterisation of MEAMs and the relationship between favourite music and self-identity in persons with dementia. The findings will have implications for how MEAMs can be used to enhance well-being and maintain self-identity in this population.

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Vitamin D to Reduce Falls: Planning to Implement Evidence into Australian Residential Aged Care Facilities

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Introduction: There is strong evidence of the effectiveness of vitamin D supplements in preventing falls in residential aged care facilities. This project aims to understand the barriers and enablers to knowledge translation in aged care homes for the implementation of vitamin D supplements for falls prevention. Methods: A literature review on interventions focused on the implementation of vitamin D supplements in aged care homes was
conducted. Factors influencing implementation were also considered by an expert advisory group, who were consulted over an 18 month planning period. **Results:** Ten studies were identified that have focused on the implementation of vitamin D supplements. Evidence for the effectiveness of implementation strategies is inconclusive, with a general consensus for the use of multifaceted interventions that include strategies such as education, audit and feedback and appointing a champion. It is clear that the attitudes and beliefs of relevant stakeholders, and organisational readiness and capacity for change are pivotal for the success of implementation. The experience with the expert advisory group and with partner organisations was instructive in that there is resistance to implementation of vitamin D supplements despite clear guideline evidence supporting this. **Conclusions:** Whilst there is limited evidence for effective knowledge translation in this setting, it is clear that there will be no ‘one size fits all’ answer. Although the evidence for vitamin D supplement use in this population is strong, the individual contexts of aged care homes must be understood to develop and implement a multifaceted knowledge translation intervention specific to their needs.

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Poster Presentation
Theme: 3. Intervention/Treatment
Poster number: P047

**Using yeast to understand intracellular pathways that promote the removal of Alzheimer beta amyloid**

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**Introduction:** The aggregation and accumulation of the beta amyloid (Abeta) protein in the brain is one of the major contributors to neurodegeneration in AD. Enhancing the removal of Abeta aggregates is one approach to prevent neuronal cell death. Intracellular clearance pathways such as autophagy play a vital role in cell survival by removing damaged organelles and aggregated proteins, including Abeta. However, the cellular mechanisms underlying autophagy mediated Abeta clearance and protection against toxicity is poorly understood. **Methods:** We originally developed a yeast cell model expressing Abeta tagged with green fluorescent protein (GFP-Abeta) to investigate aggregation, toxicity and intracellular clearance pathways that regulate Abeta accumulation in cells. In yeast, aggregated Abeta is selectively targeted for degradation in the cell and stimulating the autophagy-lysosomal pathway reduces Abeta accumulation and toxicity. To identify protective autophagy genes against Abeta, a genetic screening of Abeta expression and toxicity was undertaken in a knock-out yeast autophagy mutant library. To further validate the protective effects of the autophagy genes identified in yeast, GFP-Abeta accumulation and clearance was assessed in cells lacking and overexpression the desired genes. **Results & Discussion:** Autophagy regulators encoding regulatory subunits of AMPK, PP2A and heat shock protein complexes were identified as the lead candidate genes that altered Aβ accumulation and toxicity in the yeast screening. In addition, overexpression of these autophagy regulators showed reduced aggregation and accumulation of Abeta in yeast. **Conclusion:** Understanding mechanisms for effective removal of Abeta will be paramount for developing novel therapies for Alzheimer's. This study has identified novel genes that protect against the accumulation of Abeta aggregates. Further investigation is underway to determine whether these genes are altered in the Alzheimer's brain and if they have
neuroprotective functions. In addition to identifying protective genes, this yeast model is suited for high-throughput screening techniques for drug discovery in AD.

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Theme: 3. Intervention/Treatment
Poster number: P048

Role of Apolipoprotein D in Alzheimer's disease and Frontotemporal dementia

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Dementia is an umbrella term that describes a wide range of symptoms associated with cognitive dysfunction due to gradual brain atrophy. The loss of cognitive ability results in impairment of memory, planning, reasoning and behaviour, profoundly affecting the lives of the patients. Alzheimer's disease (AD) and Frontotemporal Dementia (FTD) are the two major forms of dementia. These are characterised by extracellular deposition of amyloid β (Aβ) and TDP43, tau and FUS deposits respectively. Oxidative stress manifested in the form of lipid peroxidation and neuroinflammation are considered to play an important role in dementia. Apolipoprotein D (apoD) is a highly conserved protein known for its antioxidant function. Recent studies have demonstrated that apoD inhibits lipid peroxidation and plays a role in regulating inflammatory pathways. The exact role of apoD in regulating inflammatory pathways is unknown. The main aim of this project is to study the role of apoD in AD and FTD brain. The project will investigate the association of apoD to inflammatory markers, TNFα, IL1β, LTB4 and PLA2 in AD and FTD post-mortem brain tissues and lipid peroxidation markers, malondialdehyde and 4-hydroxynonenal in post-mortem tissues of FTD patients in a case controlled study. This data will provide information about the role of apoD in these biochemical processes in AD and FTD brain. We will also study the impact of apoD on generation and processing of Aβ and examine the effect of apoD on TDP43 expression and its effect on TDP43 shuttling between the nucleus and cytoplasm using neuronal cells. Hence this study will advance the knowledge of role of apoD in dementia related neuroinflammation and its effect on pathological markers of dementia. We believe that apoD may help prevent neuronal damage in dementia using its antioxidant nature by inhibiting oxidative stress and/or effecting Aβ and TDP43 pathology.

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Theme: 3. Intervention/Treatment
Poster number: P079

The role of mobile DNA in Parkinson’s disease

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Parkinson’s disease (PD) is a complex neurodegenerative condition, which affects more than 7 million people worldwide and often occurs after the age of 65. About one-quarter of the
affected individuals, experience PD associated dementia and the prevalence of dementia increases with the severity of PD. The main hallmark of PD is the selective loss of dopamine (DA) neurons which control voluntary movement. Despite recent advances, current PD treatments only ameliorate symptoms but cannot cure the disease. PD aetiology is multifactorial, with genetic and environmental factors interacting via a yet unclear mechanism to induce PD pathology. Recent studies have proposed that environmental and genetic factors may trigger hyper activation of DNA mobile elements which can alter the genome by insertional mutagenesis, recombination and deletions, contributing to the susceptibility and pathophysiology of neurological disorders. Long interspersed element-1 (L1) is the only active and autonomous mobile element in the human genome, and accounts for about 17% of human DNA. L1 is active in somatic cells and can ‘jump’ from one place in the genome to another by first copying itself into RNA and then reversing the process, thus altering the activity of genes were it relocates. This study proposes to investigate the role of L1 activity at the intersection of environmental and genetic factors known to contribute to PD aetiology and, as such, presents an opportunity to transform and deepen our understanding of how PD develops. The study will go far beyond establishing the core parameters of L1 mobilisation in PD, by also testing whether L1 insertions are likely to alter DA neuronal phenotype, and whether chemical modulation of L1 activity could ameliorate PD phenotype. To achieve these aims, the project will employ imaging techniques in a mouse model combined with cutting edge single-cells genomics in mouse and human samples.

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Theme: 3. Intervention/Treatment
Poster number: P076

A deprescribing intervention to reduce the inappropriate use of antipsychotics to manage BPSD in residential aged care: The Halt Project

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Background/Objectives: Inappropriate use of antipsychotic medications to manage Behavioural and Psychological Symptoms of Dementia (BPSD) continues despite evidence for the associated risks and side-effects including apathy, stroke and death. The aim of the HALT project was to identify residents of aged care facilities on antipsychotic medications, and undertake an intervention to deprescribe antipsychotic medications and improve non-pharmacological behaviour management. Training was provided to nursing staff using a train-the-trainer model, and to pharmacists and GPs. Method: Twenty-four Residential Aged Care Facilities (RACFs) were recruited across metropolitan and regional areas. Potential participants were aged over 60 years, on regular antipsychotic medication, and without a primary psychotic illness or too severe neuropsychiatric symptoms, defined as total Neuropsychiatric Inventory (NPI) score above 50, with individual symptom scores score of 12 and occupational disruptiveness scores of at least 4 in at least two of the domains delusions, hallucinations, agitation/aggression, anxiety and disinhibition. Consenting participants were assessed one month and one week prior to commencement of deprescribing. Training was provided for nurses on how to manage neuropsychiatric symptoms and a dose reduction schedule was sent to and approved by GPs before deprescribing commenced. Participants were re-assessed 3, 6 and 12 months later. The primary outcome measure was reduction of regular antipsychotic medication without use of substitute psychotropic medications. The secondary outcome measures were NPI total and domain scores and Cohen-Mansfield Agitation Inventory score. Results To date: of 137 residents recruited, 126 had commenced
Deprescribing of antipsychotic medication. Of these, 109 had achieved cessation; 22.2% had not or later recommenced an antipsychotic medication. Preliminary analyses of 71 participants assessed 6 months after deprescribing showed NPI and CMAI scores remained stable from baseline to follow-up, including those for whom an antipsychotic was recommenced. **Conclusion:** Deprescribing of antipsychotics in nursing home residents with previous BPSD is feasible; however one quarter of those whom commence deprescribing either do not reach cessation, or are later recommenced on an antipsychotic medication. Preliminary results show BPSD do not significantly change in the 6 months after deprescribing.

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**Theme:** 4. Prevention  
**Poster number:** P059

**The role of intensity physical activity in protecting the ageing brain**

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**Introduction:** With no available cure for Alzheimer’s disease (AD, the most common form of dementia), interest has turned to preventative measures, such as changes in lifestyle that may delay or prevent the onset of cognitive decline leading to AD. A vast literature supports a link between higher levels of physical activity and cognitive function and reduced risk of AD. Furthermore, previous research findings indicate that there may be a physical activity intensity threshold beyond which cognitive benefits become more pronounced. The hypothesis that the level of physical activity intensity moderates the level of cognitive response is quite logical; however, this has yet to be thoroughly investigated in an intervention in which physical activity intensity is systematically manipulated. My proposed project will address this hypothesis by evaluating the effect of a 6 month high- and low-intensity cycle-based exercise intervention on measures of cognitive health, including neuropsychological assessment and MRI-derived brain volume, connectivity and activation.  

**Method and Result:** One hundred and five community dwelling individuals aged 65-80 years will be recruited and randomised into one of three groups: high-intensity, low-intensity and control (n=35 in each group). Participants allocated to an exercise intervention will complete 6 months of either low-intensity or high-intensity cycling program consisting of two sessions per week. Neuropsychological assessment, volumetric and functional MRI will be conducted pre-intervention, post-intervention and 12 months post-intervention.  

**Discussion and Conclusion:** At an individual level, findings from this study should result in an increased awareness of the benefits of exercise beyond the highly publicised benefits to cardiovascular health. At a national and international level, findings from this research, combined with other research corroborating these results, will provide important information which could be used to develop evidence based physical activity programs specifically aimed at enhancing cognitive health and decreasing the incidence of AD in Australia.

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The role of the neuronal epigenome in natural brain ageing and Alzheimer’s disease

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Introduction: Although the prevalence of Alzheimer’s disease (AD) is increasing, the origins and mechanisms that underlie the characteristic cognitive decline remain elusive. The emerging field of neuroepigenomics is offering new insights into the role of the epigenome in neurodegenerative disorders, and several recent studies have revealed an incredibly complex neuronal epigenetic landscape. Levels of DNA methylation in the brain are correlated with age, and affect genes involved in nervous system development, neurogenesis and AD. Furthermore, limited profiling of DNA methylation in AD brains identified aberrant DNA methylation near genes with neurological and cognitive functions. These compelling data indicate a role of the epigenome in age-related cognitive decline. Given the complexity of the neuronal epigenome, and the profound insights already gained through genome-wide DNA methylation profiling, there is a immediate need to determine the role of the neuronal epigenome in ageing and AD. Method: In this study, we will determine the extent of neuronal epigenomic reconfiguration during ageing and in AD. Epigenomic profiling of neurons isolated from non-pathological human brains spanning 45-95 years and AD-affected brains will be analysed using whole-genome DNA bisulfite sequencing (methylC-seq), accessible chromatin profiling (ATAC-seq) and gene expression profiling (RNA-seq) to enable the first comprehensive investigation of epigenomic changes in AD. Discussion: These comprehensive epigenome profiles of AD and normal human brain ageing will provide unprecedented insights into the role of the epigenome in neuronal genome regulation throughout the natural ageing process and in AD. This project has the potential to identify many new candidate genes involved in cognitive decline, which could aid in the generation of new animal and cell models for AD. Comprehensive investigation of the different forms of DNA methylation has proven to be a powerful discovery tool that has consistently resulted in major new and unanticipated discoveries that are rapidly accelerating neuroepigenomic research.

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Poster Presentation
Theme: 1. Diagnosis/assessment
Poster number: P084

Investigating mGluR5-amyloid-beta interactions in cognitive decline using translational touchscreen paradigms

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Cognitive decline is a core feature of Alzheimer’s disease (AD) and there is no cure or treatment. Metabotropic glutamate receptor 5 (mGluR5) has been identified as a novel...
therapeutic target through the recent demonstration of its involvement in a key pathogenic pathway. As yet, the role of mGlu5 in mediating specific cognitive phenotypes has not been explored using clinically translatable tests. This is an important step towards developing novel mGlu5-based therapies for cognitive dysfunction. Genetic mouse models are major tools to investigate mechanisms underlying cognitive decline however, to date, assessment of cognition in mice has been largely unrelated to the clinic. This project investigates how mGluR5 mediates cognition in AD mouse models using recently developed touchscreen tasks that allow the assessment of cognitive domains, directly relevant to impairments described in AD patients. Mice containing disease-causing mutations in genes encoding the amyloid precursor protein (APP), and presenilin-1 (PS1) will be examined using touchscreen tests relevant to the principal cognitive domains affected by AD. APP/PS1 mice show cognitive deficits in standard tests; however these transgenic mice have not been characterised using clinically relevant tasks. Animals will be trained to discriminate between two visual stimuli projected onto a touch-sensitive computer screen. Tasks will then be scaled in complexity to mimic tests used in the clinic. To establish the role of mGlu5 in mediating cognitive phenotypes in APP/PS1 mice, we will employ genetic and pharmacological tools to spatially and temporally restrict the receptor’s expression. By establishing behavioural testing protocols that can be directly translated into clinical practice, this project has potential to facilitate the translation of pre-clinical treatments into clinical trials. Furthermore, investigating how mGluR5 modulates cognitive function in AD, and using this information to implement pharmacological interventions, will inform novel drug targets of direct clinical relevance.

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Theme: 3. Intervention/Treatment
Poster number: P049

Improving pain assessment and treatment for people with cognitive impairment in the Emergency Department.

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Introduction: People with cognitive impairment (CI), including dementia, face substantially greater obstacles in receiving effective pain relief, as validated pain assessment tools and protocols for people with CI are not generally used in Australian emergency departments (ED). Methods: Two-year RCT in 8 Australian EDs in a pre/post-test design in 602 older people and a long bone fracture, with/without CI, and random assignment to pain assessment with the PAIN-AD (breathing, vocalization, facial expression, body language and consolability) and associated pain management. Primary outcome: Time to analgesia; Secondary outcomes: pain assessment, analgesia given. Analysis: Binary logistic model, adjusted for age, triage code, gender, ambulance analgesia and documented pain score. Results: 271 (45.1%) participants had CI (mean age 86), 84% were female and 94% presented after a fall. Participants with no CI waited 127.7 (average) minutes vs. participants with CI waited 162 minutes (average) for pain assessment and analgesia. 45% of participants with CI were given no analgesia and 19.4% were given one dose of paracetamol. PAIN-AD was used for 160 (44.6%) participants with CI, reducing analgesic wait time from 176.11 (SD 213) minutes to 123.9 (SD 123) minutes; < 60 minutes (n=180, 29.9%, 33min); >60 minutes (n=422, 70.1%, 182min. Discussion: Inadequate analgesia in the ED arises from non-use of pain screening
tools for people with CI, poor knowledge of pain as a reason for agitation/delirium and belief that people with CI are vulnerable to analgesic side effects. **Conclusion:** An acceptable pain response by ED clinicians for all older people, including people with CI, requires urgent attention: clinical procedure review, standardised pain assessment screen, nurse-initiated analgesic standing orders, targeted education/training in pain assessment.

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Poster Presentation  
Theme: No Theme Allocated  
Poster number: P073

**Disruption of myelin lipid biosynthesis precedes tau pathology in the cortical pathogenesis of Alzheimer's Disease**

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**Introduction:** The anatomical progression of neurofibri llar tangle (NFT) pathology, a hallmark of Alzheimer's Disease (AD), runs inverse to the course of developmental myelination, with regions of thinner myelin sheaths preferentially affected by AD. Lipids constitute more than 70% of myelin, with sulfatide and galactosylceramide (GalCer) comprising almost 30% of myelin lipids. Depletion of these prototypical myelin lipids has been reported in various age-related neurological diseases, including AD. Expression of sulfatide and GalCer is dependent on the production of their biosynthetic precursor, very long chain ceramide (VLC). This study addressed whether loss of myelin-enriched lipids correlates with increased NFT pathology and whether perturbations in this myelin lipid biochemical pathway occur in the pre-clinical AD state. **Method:** Lipids were extracted from brain tissue of subjects graded according to the Braak staging scheme for post-mortem NFT pathology. Mass spectrometry was used to quantify lipid levels in hippocampus, cerebellum, inferior temporal and superior frontal grey and white matter. Ceramide synthase activity in crude tissue homogenates was assayed using our recently-published fluorescent assay. **Results and Discussion:** Severe depletion of GalCer and sulfatide was identified in our tissue cohort with increasing AD pathology. Depletion of these myelin-enriched lipids was traced metabolically to loss of VLC. Synthesis of VLC is catalysed by ceramide synthase 2 (CERS2). We observed a deficiency in CERS2 activity as early as Braak stage I/II in tempor al cortex, and Braak stage III/IV in hippocampus and frontal cortex, indicating that loss of myelin-specific ceramide synthase activity precedes NFT pathology in cortical regions. **Conclusion:** Decreased myelin ceramide synthesis is indicative of a defect in myelin maintenance during the pre-clinical stages of AD pathogenesis. We propose that this defect contributes significantly to myelin deterioration, synaptic dysfunction, and neurological decline. Our results support the notion that demyelination is a significant driving influence in AD pathogenesis.

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Poster Presentation  
Theme: 3. Intervention/Treatment  
Poster number: P050
Discovery of functionally selective C5aR2 ligands: novel modulators of C5a signalling activity in vitro and in vivo

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Introduction: Neuroinflammation has now been widely associated with a number of dementia related diseases. Activation of the innate immune system, specifically the complement system has been attributed a key role in many neurodegenerative diseases. The complement activation peptide, C5a, binds two seven transmembrane receptors; namely the C5a receptor 1 (C5aR1) and C5a receptor 2 (C5aR2). C5aR2 is a non-G-protein-signalling receptor whose biological role remains controversial. Some of this controversy arises due to the lack of selective ligands for C5aR2. This study aimed to discover novel selective C5aR2 ligands to explore the functional role of C5aR2.

Methods: A small peptide library of 61 ligands based on the C-terminus of C5a was tested for the ability to displace 125I-C5a from C5aR2 membranes and counter-screened against C5aR1. A C5aR2 β-arrestin 2 recruitment assay was used to test ligand activity, and ligands were counter-screened for β-arrestin 2 recruitment via C5aR1. The functional role of ligands was investigated by looking at ERK1/2 phosphorylation and LPS-stimulated cytokine release in human monocyte derived macrophages (HMDM). Additionally, C5aR2 activity was confirmed in the presence of a C5aR1 antagonist. Finally one ligand was tested for the ability to modulate C5a-induced neutrophil mobilisation in vivo.

Results & Discussions: Two ligands (P32 and P59) were identified as functionally selective C5aR2-ligands, exhibiting selective recruitment of β-arrestin 2 via C5aR2, partial inhibition of C5a-induced ERK1/2 activation, and LPS-stimulated IL-6 release from HMDM in a C5aR2 dependent manner. Importantly, neither ligand could induce ERK1/2 activation or inhibit C5a-induced ERK1/2 activation via C5aR1 directly. Finally, P32 inhibited C5a-mediated neutrophil mobilisation in wild-type, but not C5aR2-/- mice.

Conclusion: Here we report the first functionally selective ligands for C5aR2 with novel pharmacology that can selectively modulate C5a activity in vitro and in vivo, and thus will be valuable tools to interrogate C5aR2 function in dementia models.

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Poster Presentation
Theme: 3. Intervention/Treatment
Poster number: P082

Sleep, plasticity and neurodegeneration: Targeting sleep to improve cognition in Mild Cognitive Impairment

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A compelling body of evidence underscores the critical role sleep plays in learning, memory and brain plasticity. Two key components of sleep physiology which are intimately linked with memory consolidation include sleep spindles and slow wave activity (SWA, 0.5 - 4.0 Hz) in non-rapid eye movement sleep. These distinct electroencephalogram (EEG) features are important for sleep maintenance and neuronal plasticity, and age-related reductions in sleep spindles and SWA may explain cognitive decline in older age. Importantly, more than 60% of individuals with mild cognitive impairment MCI experience significant sleep disturbance. However, the relationship between disrupted sleep, cognitive impairment and neurodegeneration in this group is poorly understood. At present it is unclear whether altered sleep physiology in MCI is a risk factor for ongoing cognitive decline and progression to dementia. It is also necessary to identify the sleep characteristics which relate to neurodegeneration in critical key brain areas that are fundamental for sleep-dependent memory consolidation. Sleep spindles and SWA may serve as novel brain biomarkers of poor cognitive outcomes in MCI and by targeting these we can potentially modify the risk factors for developing dementia. Experimentally boosting sleep spindles and SWA during sleep results in improved memory and cognition in healthy individuals. Therefore if sleep quality is optimised by enhancing sleep microarchitecture, an exciting new treatment approach to prevent or slow cognitive decline becomes possible.

AIMS

1. Investigate key EEG features known to be associated with sleep-dependent memory processes and brain plasticity, to identify novel brain biomarkers of cognitive impairment and neurodegeneration,
2. Examine prospectively whether altered sleep microarchitecture predicts cognitive decline and disease progression in MCI over a 2 year follow-up period,
3. Conduct a clinical trial to deliver an early pharmacological intervention to enhance sleep spindle EEG features to optimise sleep quality and improve memory.

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Poster Presentation
Theme: 1. Diagnosis/Assessment
Poster number: P002

Moderate consumption of alcohol modifies the risk of Alzheimer’s disease and is associated with lower levels of cortisol

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www.nnidr2016.com
Introduction: The search for a definitive set of validated blood based biomarkers that relate directly to either Alzheimer's disease (AD) pathology, or the onset of clinical symptoms is far from complete. A possible reason for this, is the underlying biological interaction between lifestyle and biology. In the current study we investigate the relationship between the well know stress hormone cortisol, and alcohol consumption with regards to risk of AD. Method: Using data from the Australian Imaging, Biomarkers and Lifestyle (AIBL) study, we tested for the presence of biological interaction between cortisol and alcohol consumption for the risk of AD. Result: Mean cortisol level in the AIBL HC group (N=422) at baseline was lower compared with the AD group (N=95). (HC: 143.3 (67.1) vs AD: 160.1 (72.1)). We found a significant interaction between alcohol consumption, cortisol levels and clinical classification (P=0.02), with no difference between HC and AD cortisol levels for those who consumed alcohol (P=0.5), compared with a large significant difference for those who consumed a moderate amount of alcohol (P=0.004). Individuals with high cortisol, who reported no alcohol consumption were 7.2 times more likely to develop AD compared with those with low cortisol who reported any alcohol consumption (p<0.0001, Synergy Index (SI): 11.6 (95%CI: 6.4-16.9)). Reducing the sample to only those who consistently consumed wine (N=293) compared with those reporting no alcohol consumption (N=224), we saw the effect increase, with those with high cortisol who reported no alcohol consumption 9.4 times more likely to develop AD compared with those with low cortisol who reported any alcohol consumption (p<0.0001, SI: 9.5 (2.9-15.9)). Discussion and Conclusion: These findings demonstrate the benefit of regular moderate consumption of alcohol to combat the increased risk of AD due to the stress hormone cortisol.

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Poster Presentation
Theme: 4. Prevention
Poster number: P060

Characterising the associations between sleep and physical activity in older adults at-risk for dementia

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Purpose: Sleep-wake dysfunction (SWD) is evident in up to 60% of individuals with Mild Cognitive Impairment (MCI). MCI is considered an ‘at-risk’ or transitional stage between normal ageing and dementia, with conversion rates of approximately 50% over five years reported in the literature. Of concern, SWD is linked with poorer cognition and clinical outcomes, and more rapid decline. Increasing evidence suggests that moderate and vigorous physical activity (MVPA) has a beneficial effect on sleep and cognition, however it is unclear how MVPA relates to SWD in older adults with MCI. Methods: Fifty-four participants with MCI were recruited from the Healthy Brain Ageing Clinic, Sydney, Australia. All participants underwent comprehensive medical, psychiatric and neuropsychological assessments. Total MVPA was calculated using the Active Australia Questionnaire (MVPA=[minutes of vigorous activity*2]+minutes of moderate activity). For comparison purposes, participants were stratified into the following MVPA groups: 0-9, 10-149, 150-299
and >300 minutes/week. Participants also wore an actigraphy watch for 2-weeks to assess SWD. **Results:** There were no significant differences between MVPA groups in terms of age, gender, depressive symptoms, global cognition and sleep onset, sleep offset or total sleep times. However, participants who reported 0-9 minutes/week of MVPA had significantly greater sleep disturbance (wake after sleep onset [F=11.5, p<0.000] and night-time awakenings [F=6.8, p=0.001]) in comparison to each of the other MVPA groups, which remained highly significant with post-hoc analyses. **Conclusion:** These findings suggest that even low levels of MVPA (below current recommendations of >150 mins/week) may have a beneficial effect on SWD in older adults with MCI. Further research is now required to examine how MVPA relates to sleep macro and microarchitecture as well as cognitive functioning in ‘at-risk’ individuals. Longitudinal follow-up of participants is also required to determine how MVPA relates to clinical trajectory.

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Poster number: P003

Amyloid Imaging in Sleep Apnoea: Findings from AIBL-VETS

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**Introduction:** An association between sleep disturbance and Alzheimer's dementia (AD) has been suggested. Recent amyloid imaging studies have shown increased tracer retention associated with shorter sleep duration, initial insomnia and impaired generation of Non Rapid Eye Movement (NREM) Slow Wave Activity. Obstructive Sleep Apnoea (OSA) is a common disorder with a prevalence of 24% in men aged between 30 and 60 years. OSA has been associated with cognitive impairment and increased dementia risk but no amyloid imaging studies have been performed in subject suffering from this condition. **Methods:** We assessed Vietnam Veterans in the AIBL-VETS study of AD risk chronic combat related Post-Traumatic Stress Disorder. Veterans with polysomnographic confirmation of OSA were compared to those who did not have features of OSA according to the Pittsburgh Sleep Index Questionnaire. Amyloid imaging was performed using florbetaben (18^F-FBB). Standardised Uptake Value Ratios (SUVR) as a measure of amyloid burden were generated using the cerebellar cortex as reference region. **Results:** 18 veterans with OSA (mean age, 68.18±3.56 years) and 48 Veterans without OSA (mean age 68.17±3.74 years) underwent 18F-FBB PET. SUR was significantly higher in the OSA group than in the non-OSA group (1.39±0.31 vs 1.27±0.17, respectively, p <0.05). **Discussion and conclusion:** This preliminary result suggests an association between OSA and increased amyloid deposition of brain amyloid. OSA is a treatable condition so it may represent a treatable risk factor for AD. Prospective studies in larger cohorts are warranted.

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Poster Presentation  
Theme: 2. Care/Living With Dementia  
Poster number: P033
Work4Dementia: Development of an evidence-based intervention to build capacity and resilience for the Australian dementia care workforce

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Introduction: Dementia is a public health priority. A three-fold increase in the number of care workers in Australia is required to meet the demands associated with dementia over the next three decades. There are concerns aged care organisations cannot keep up with the increasing high care demands associated with this condition. This is worsened by high rates of turnover in the sector. Innovative strategies are required to attract and retain aged and dementia care staff as the unstable workforce has deleterious effects on both care workers and people with dementia (e.g., high job stress and poor quality care). Despite this urgent need it is not clear which interventions best enhance the dementia care workforce. **Aim:** I will apply findings from my PhD and postdoctoral research, as well as my clinical psychology expertise to offer unique insights on how to effectively improve the capacity and resilience of the dementia care workforce. **Previous Findings:** I identified a new construct, occupational communion (OC), which has implications for retaining dementia care workers in their jobs for longer. OC facilitates a sense of belonging based in social interaction at work, essential for positive coping with high job demands. Results showed that OC is a valid and reliable construct. OC has been tested in a theoretical model showing social interaction with clients and colleagues and can influence positive ways to cope with job demands such as isolation or grief and loss. **Research Program:** This fellowship will test the application of OC to inform the development of an innovative evidence-based intervention (Work4Dementia). The acceptability, feasibility and efficacy of Work4Dementia will be tested in six aged and dementia care organisations across Australia. Work4Dementia will ultimately reduce job stress and subsequent turnover, enhance work engagement and improve quality care outcomes for people with dementia and their families.

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Dementia in people with Intellectual Disability: A longitudinal study with a knowledge translation focus

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**Introduction:** The population with intellectual disability (ID) is ageing rapidly. People with ID appear to be at increased risk of dementia, often with younger onset and faster decline. Yet little research has investigated risk factors for dementia in ID, and there is a dearth of information pertaining to screening tools and diagnostic instruments for this group. Unclear service models and inadequate clinician training further impact their care. This project has two aims: firstly, investigating correlates of dementia in ID, and appropriate instruments for assessment; secondly, developing clinician resources and a pathway to care for people with ID and dementia. **Methods:** A longitudinal pilot study will be expanded to create a sample of 230 participants with ID aged over 40, recruited from disability services in NSW, with a high-risk sub-sample recruited via clinicians and aged-care services. Carer-report questionnaires will assess demographics, lifestyle, medical history, adaptive behaviour, life events, and declines...
for the person with ID, and semi-structured carer interviews will assess symptoms of dementia. A self-report questionnaire for family carers will measure carer mental health, burden and supports. A sub-sample of participants with ID will complete medical and neuropsychological examinations. Potential improvements to current care models will be developed via a Delphi process with experts in the field. A new model will then be proposed. **Results:** Results will be used within a knowledge translation framework which includes developing an online training module for clinicians, and guidelines for assessing dementia in ID. **Discussion & Conclusions:** The study holds potential to identify the most appropriate screening and assessment tools to detect dementia in people with ID, and to reveal potentially modifiable risk factors for dementia in ID. The project aims to produce outcomes which can then be used to rapidly improve the nature of dementia assessment for older Australians with ID.

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Positive association of MR susceptibility and amyloid-\(\beta\) in non-demented individuals suggesting a potential role of iron in frontal circuit dysfunction

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**Introduction:** Normal aging and Alzheimer’s disease (AD) are associated with increased levels of cerebral iron. Recent developments of magnetic resonance imaging (MRI) techniques such as quantitative susceptibility mapping (QSM) is believed to reflect non-heme iron deposition, allowing to investigate the association of iron with other well-established biomarkers of AD such as amyloid-\(\beta\) (\(A\beta\)) prior to the onset of the AD pathology. It has been shown that in AD iron level increases mainly in tau tangles and \(A\beta\) plaques, however, the process and timeframe leading to this accumulation is not well understood. This study aims to investigate the association between regional iron and \(A\beta\) among non-demented subjects prior to AD. **Methods:** MRI data including QSM and T1W as well as 11C PiB-PET from 51 HC and 15 individuals with mild cognitive impairment (MCI) were collected as part of the Australian Imaging Biomarkers and Lifestyle (AIBL) study. Different anatomical regions were obtained by spatially normalizing anatomical templates to T1W image of each subject followed by a rigid alignment to QSM and PET images. The PiB SUVR and iron concentration were obtained by intensity normalized the PET and QSM images using the cerebellum gray matter and posterior ventricle, respectively. For regional analysis, robust mean signal intensity was computed in four cortical lobes and sub-cortical regions (caudate, putamen and pallidum). The normalized values were adjusted for age, gender and APOE \(\varepsilon4\) carrier status. **Result:** Correlation analysis showed a significant correlation between regional iron and \(A\beta\) in the frontal lobe \((r=0.27, p<0.05)\) and caudate \((r=0.334, p<0.01)\) as shown in Fig. 1. **Conclusions:** Significant associations were found between cerebral iron and amyloid in non-demented elderly individuals. The positive correlation in caudate and the frontal lobe suggests a potential role of iron in frontal circuit dysfunction which leads to impairment of executive function and inhibitory processes.
**Association of cerebral blood flow and amyloid-β status in preclinical Alzheimer’s disease**

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**Introduction:** Global and regional reductions in cerebral blood flow (CBF) have been reported in Alzheimer’s disease (AD). However, these CBF changes have been only investigated in prodromal AD and no study has used MRI to identify such differences between healthy controls (HC) with high or low amyloid-β (Aβ) deposition. This study aims to investigate the association between Aβ and rCBF in preclinical AD using state of the art multiphase pseudo-continuous ASL (MP-PCASL). **Methods:** 42 HC underwent PiB-PET and MP-PCASL as part of the AIBL study. Aβ status was determined using CapAIBL while rCBF was computed using an in-house processing pipeline comprising of motion correction, spatial and temporal denoising and quantification. The regional CBF values were adjusted for age and thalamus was used as a reference region for CBF normalisation. **Results:** Using a cut-off value of 1.5 for SUVR values, 32 HC were classified as low Aβ (HC-) and 10 as high Aβ (HC+). Comparing HC+ to HC-, CBF was significantly lower in the neocortex as well as inferior temporal, frontal, parietal and posterior cingulate regions (p<0.01). **Conclusions:** Significant differences in CBF were found between HC with high and low Aβ burdens. MP-PCASL offers a highly sensitive and non-invasive tool that can detect reduced cerebral flow in preclinical AD. Furthermore, the high sensitivity of MP-PCASL could be useful for subject screening in clinical trials.

**Depression in Dementia**

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**Introduction:** Neuropsychiatric symptoms like depression are common in people with Alzheimer’s disease (AD) and are a frequent cause of distress and reduced quality of life. This study will identify factors that may lead to the development of depression and other neuropsychiatric symptoms in AD and additionally test a novel intervention to try and...
prevent these symptoms from occurring. Cognitive bias modification (CBM) targets attentional and interpretative biases associated with anxiety and depression. This has been shown to be effective in reducing depressive symptoms in younger adults without cognitive impairment and may also have a role in preventing the development of depressive symptoms in people with dementia. **Methods:** We will recruit 300 depression-free individuals with mild/moderate severity AD and randomise them to active or control CBM over a 2-year period. The primary outcome of interest is the difference in the incidence of depression between the groups. Additional outcomes of interest will include quality of life, other neuropsychiatric symptoms, cognitive performance and carer burden. Participants will undergo regular assessments to determine any clinical and lifestyle factors that may be important in the development of these outcomes. Additionally, a sub-group of 40 individuals will undergo 2 magnetic resonance imaging scans (baseline and 2 years) to explore neuroanatomical predictors of depression and other neuropsychiatric symptoms. **Discussion:** Alzheimers dementia is a common disorder with devastating consequences for sufferers and their families. The impact of this disease is undoubtedly accentuated by the presence of neuropsychiatric symptoms. Our understanding of the aetiology of these symptoms is relatively poor and few interventions exist for their prevention and treatment, and these are frequently associated with unacceptable side effects. The proposed study will enable a better understanding of neuropsychiatric symptoms in AD and trial a simple, safe and cost-effective treatment that could be easily implemented into everyday clinical practice.

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**Theme:** 4. Prevention  
**Poster number:** P070  
**Systematic review of associations of sedentary behaviour with cognitive function or dementia in mid-age and older adults**  
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**Introduction:** There is a need to identify behavioural risk factors to reduce the risk of cognitive decline and dementia and progression of those conditions. Sedentary behaviour is ubiquitous in contemporary society and preliminary evidence suggests that sedentary behaviour may affect cognitive outcomes at various stages of the lifecourse. The aim of this study was to review the literature to examine associations of sedentary behaviour with cognitive function or dementia in mid-aged and older adults. **Methods:** Databases were searched for articles published to April 2016. Studies were included if they measured sedentary behaviour/s and cognitive function or dementia, and were conducted in the relevant study population, i.e. aged 45 years and older. Information on study characteristics and results were extracted. **Results & Discussions:** Fifteen studies (14 observational and one case control), representing 25,905 participants from six countries were included. 26 of 41 associations were statistically significant. Objectively-measured sedentary behaviour was associated with lower integrity in parahippocampal white matter, poorer visual memory, and in people with a genetic risk for Alzheimer’s Disease, higher cerebral blood flow. Computer use was beneficially associated with global cognitive function, dementia risk, verbal memory, and executive function. Television viewing was detrimentally associated with psychomotor speed, executive attention, immediate and delayed verbal memory, episodic
memory, and global cognition, and development of Alzheimer’s Disease. One study found higher self-report total sedentary behaviour was associated with faster visual search and perceptual speed, however no associations for television time or objectively-measured sedentary behaviour were observed. Different sedentary behaviours were beneficial or detrimental for cognitive function and development of dementia in mid-aged and older adults. Research is needed to understand the physiological mechanisms underpinning these relationships and causality. **Conclusion:** Sedentary behaviour/s are associated with cognitive function or dementia. This study highlights the importance for cognitive health of activities undertaken while being sedentary.

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**Music Playlists and Mood Regulation in People with Dementia and Depression**

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**Introduction:** Depression is common in dementia and is a primary factor associated with a decline in the quality of life of people with dementia and their caregivers. It is also both a risk factor and a prodromal symptom of dementia, and is associated with accelerated cognitive decline. There is evidence that music can have a powerful effect on people with dementia even in cases of significant cognitive impairment. However, previous research suggests that depression has an important influence on the affective impact of music on the listener. While music therapy interventions involving the presence of a trained therapist are generally tailored to the individual needs of patients, interventions using pre-recorded musical playlists seldom account for both individual music tastes and psychological profile. The research to be conducted will further explore ways for designing music programs for self-management of moods in people with dementia that will consider both individual taste and mental health status.  
**Method:** An initial phase of this research will involve a 3x2x2 factorial experiment to test the relative impact of tempo, mode and lyrics on the mood of people with mild dementia, in order to test the contribution of these components of music on people with differing symptomatic profiles. Pre and post-mood self-report mood measures will be triangulated with behavioural observation and physiological measures.  
**Results:** Not available at this time.  
**Discussion:** This research will result in an increased understanding of the impact of music on quality of life of people with dementia and will demonstrate how multiple variables interact in the effect that music has on mood. The findings will ultimately inform the development of an engaging and cost-effective tool for reducing depression in people with dementia using music.

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**Impact of a virtual dementia experience on medical and pharmacy students knowledge, attitudes and self-reported behaviour toward people with dementia**
Introduction: Medical practices and community pharmacies should be structured according to dementia-friendly community guidelines to ensure accessibility for people with dementia. Medical practitioners and pharmacists should be equipped with the necessary knowledge and skills to create these environments. Alzheimer's Australia Victoria's Virtual Dementia ExperienceTM (the Experience) uses a multi-sensory simulation of light, sound, colour and visual content to immerse participants into the virtual world of a person with dementia. This Experience can educate medical and pharmacy students about dementia-friendly communities in preparation for their future healthcare roles. This study aims to quantitatively and qualitatively evaluate the impact of the Experience on medical and pharmacy students knowledge, attitudes and self-reported behaviour toward people with dementia.

Method: Third year medical (105) and fourth year pharmacy (250) Monash University students will be invited to participate in a non-randomised controlled study in 2016-17. Of these students, 110 will undertake the Experience. To evaluate the Experience, all 355 students will be invited to complete pre- and post-Experience surveys. The 20-item Dementia Attitudes Scale will be used to evaluate the affective, behavioural, and cognitive components of students attitudes (O'Connor et al 2010). Students who undertake the Experience will also be invited to participate in one of eight focus groups.

Result & Discussion: This study is due to commence in 2016 as part of the lead author's NHMRC-ARC Dementia Research Development Fellowship.

Conclusion: Education about dementia-friendly communities should be provided to future medical practitioners and pharmacists during their undergraduate training. This research is focussed on an area where there is a clear identified need for greater healthcare professional understanding and engagement. Knowledge gained will be used to provide an evidence base for Australian academics designing medical and pharmacy curriculum, to build capacity of Australia's future healthcare professionals and optimise clinical care for older Australians with dementia.

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Poster number: P036

Optimising medication use to maintain or improve quality of life in aged care facility residents with and without dementia

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Introduction: In the absence of a cure, maintaining or improving quality of life is a central goal of dementia management. This often involves using medications to treat signs and symptoms of dementia and co-morbidities. Despite this, the impact of medication use on quality of life has not been extensively explored in Australian aged care facility residents with and without dementia. This study aims to investigate the relationship between medication use and quality of life in aged care facility residents with and without dementia.

Method: The objectives of this study are to: systematically review factors related to medication use and their association with quality of life; prospectively investigate the association between...
medication use and quality of life; design, implement and evaluate a targeted intervention to optimise medication use for the purpose of maintaining or improving quality of life; identify factors important for wider implementation of the intervention into clinical practice; and evaluate the outcomes of this study internationally. **Result & Discussion:** This study is due to commence in 2016 as part of the lead author’s NHMRC-ARC Dementia Research Development Fellowship. **Conclusion:** This study offers an innovative approach towards improving Australian dementia management, by investigating the under-researched association between medication use and quality of life. The results of this research will guide health professionals to better manage medications.

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**Poster number:** P006

**Spatial learning and memory in Huntington’s disease**

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**Background:** Cognitive assessment research in the neurodegenerative diseases is lacking tasks that allow direct translation of findings from animal models to human trials, resulting in a critical gap in assessment of potential treatments. This project will focus on spatial memory and dementia in Huntington’s disease (HD) to create a brain-informed cognitive outcome measure for testing treatments. Spatial memory deficits in HD are related to striatal and hippocampal pathology, and animal models of HD link reduced hippocampal brain-derived neurotrophic factor (BDNF), a key neuroplasticity protein that declines in HD, to spatial memory impairments. Despite clear functional disability in HD (e.g., finding one’s way home), the association between spatial memory deficits and BDNF levels in human HD is unknown. Several treatments in the pipeline for HD are specifically targeted at restoring BDNF. The specificity of spatial memory to the hippocampus and striatum in HD makes spatial memory an ideal cognitive outcome for testing BDNF-relevant treatments for HD. **Method:** During the project we will create the first comprehensive analysis of spatial memory impairments in HD, and determine patterns of brain volume loss in striatal and hippocampal formations. With the use of this battery of spatial memory tests, we will assess the efficacy of treatment in the context of an international HD clinical trial. Prof Stout leads the cognitive assessment component of several clinical trials in HD, creating an opportunity to translate measures of spatial memory to test treatments. **Significance:** This project is in the initial stages, but its results will yield cognitive assessment strategy that specifically maximises the potential for homologies across species in cognitive phenotyping, which will improve translational outcomes in HD research.

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**Poster number:** P007

**Frontoparietal cortical network connectivity and executive functioning in older adults**
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Introduction: Advancing age is associated with a decline in executive functioning, with marked deficits in this cognitive domain among the earliest clinical signs of neurodegeneration and future dementia. Long-range connectivity between remote brain regions is an important factor underpinning a number of cognitive processes, with connectivity between frontal and parietal cortices strongly implicated in working memory and executive control. In this study, we used resting-state electroencephalography (EEG) to characterise the relationship between long-range synchronisation of oscillatory activity (a marker of connectivity) in the frontoparietal network and executive functioning in older adults. Method: Fifteen healthy older adults (aged 53-76 years) were assessed using the Spatial Working Memory (SWM) subtest from the Cambridge Neuropsychological Test Automated Battery (CANTAB). Resting EEG (3 min; eyes open) was recorded in a separate session, and frontoparietal network connectivity was determined by averaging debiased weighted phase-lagged index (wPLI; a conservative measure of phase synchronisation) for electrode pairs F3-P3 and F4-P4. Results & Discussion: A significant correlation was observed between SWM task performance and frontoparietal network connectivity, with stronger phase synchronisation between frontal and parietal electrodes, specifically in the alpha frequency band (8-12 Hz), associated with fewer errors and more efficient strategy use. Conclusion: These findings suggest that frequency-specific long-range connectivity in the frontoparietal network is a strong predictor of higher-order executive functioning in older adults. This may have important implications for the early detection of cognitive decline and dementia.

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Poster number: P008

Modelling age-related changes in brain dynamics

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Introduction: The brain exhibits structural adaptations as we age. Such structural changes make the white matter sparser and increase the path length between cortical areas. The functional consequences of such structural changes remain poorly understood. Method: We model the whole-brain dynamics utilising the anatomical connectivity obtained from cohorts of young (15-30 years) and elderly (76-94 years) adults. The model is tuned to match the statistical dependences observed between cortical regions. Result: The results of this study demonstrate that the influence of the structural connectivity over the functional connectivity is stronger for the younger cohort, and the overall metabolic cost is higher for the older cohort. As a function of age, regions that show increased synchronisation are mostly located within the same hemisphere, whereas regions that show decreased synchronisation are mostly located in different hemispheres. Discussion and Conclusion: Our results suggest that our modelling approach constitutes a promising framework to characterise changes in brain dynamics with age. The understanding of healthy brain ageing is a fundamental benchmark for diagnosis of neurodegenerative age-related disorders such as dementia. Our framework allows the test of numerous hypothesis and interventions, which will be explored in the future.
**Retinal vascular changes are associated with neocortical beta amyloid scores in the elderly**

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**Introduction:** There is mounting evidence in recent years suggesting that the retina can provide an easily accessible window to brain pathology during early stages of dementia, specifically Alzheimer's disease (AD). The retina shares common physiological processes with the brain and thus is an excellent target area to study AD-related symptoms in vivo. We studied retinal vascular changes and their association with cerebral beta amyloid plaque load in an elderly cohort to further establish the link between retinal biomarkers and cerebral pathology in AD.

**Methods:** 75 patients (79±5 yrs, 22 male) with no clinical diagnosis of AD were studied. All patients had a magnetic resonance image (MRI) and a florbetaben positron emission tomography (PET) scan. PET images were analysed based on the standardised uptake value ratio (SUVR). Following this, all patients were asked to attend Macquarie Eye clinic for a thorough clinical ocular examination and measurement of arterial pulse (RAP) retinal venous pulse (RVP) amplitude using the Dynamic Vessel Analyser.

**Results:** The mean neocortical beta amyloid (Aβ) SUVR was 1.35±0.31 (0.97-2.32). Intraocular pressures were normal (14±3 mmHg). We observed a positive association between RAP amplitude and Aβ-SUVR (p<0.05, r=0.33). The correlation between RVP amplitude and Aβ-SUVR was negative (p<0.005, r=0.4).

**Discussion and Conclusion:** This study demonstrated a significant correlation between amplitude of retinal vascular pulsatility and neocortical Aβ scores. Future studies will investigate this correlation in an established AD cohort to further elucidate whether these biomarkers have a correlation not only with cerebral Aβ plaques but with the development of subsequent clinical dementia. If a clinical correlation is confirmed, screening of eyes in those considered at risk of AD may provide an alternative non-expensive simple tool for establishing increased risk profiles and potentially for monitoring potential therapies.

**Knowledge translation in dementia care: A review of the evidence for 'Appreciative Inquiry' as a method to facilitate organizational change**

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Introduction: It can be difficult for service providers to be aware of, and use, relevant research-derived knowledge of best practice in dementia care. Timely, effective knowledge translation becomes more challenging when policy reforms demand rapid change - and the evidence base may be unclear but commercial stakes are high, e.g. consumer directed care. One approach gaining popularity is Appreciative Inquiry - a strengths-based technique with roots in positive psychology and business management. Some Australian service providers have engaged change consultants to facilitate this process. Our question: How has Appreciative Inquiry been used in dementia care settings, and with what types of outcomes?

Method: We conducted a scoping conceptual review of the dementia care literature (2010+) to determine how Appreciative Inquiry has been used and evaluated (measures and outcomes).

Result & Discussions: Fewer than 20 relevant articles were found. Beyond qualitative process reports on creating ‘team vision’, no compelling evaluations of knowledge translation or sustainable change outcomes in dementia care were identified. One study reported dementia care staff enjoyed the imaginative narrative approach of Appreciative Inquiry, despite it being initially deemed ‘woolly thinking’ by sceptical clinicians. The method was also used with consumers in participatory action research, e.g. to learn what older people want from care. Conclusion: In dementia care, Appreciative Inquiry has been used to broker vision-setting conversations with staff and/or consumers. There is a dearth of evidence for outcomes - should this obstruct using Appreciative Inquiry as a technique for facilitating change in translating research-derived knowledge into practice? Strategies harnessing ‘feel good’ and ‘innovative thinking’ may have value, e.g. help re-engage staff in settings already negative for change efforts. While awaiting quality research to demonstrate improved measureable care-related, staff-based, or organization-derived change outcomes, we explore practical considerations for dementia care providers interested in Appreciative Inquiry. Hallmarks of transformational leadership will be highlighted.

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Poster Presentation
Theme: 3. Intervention/Treatment
Poster number: P057

The role of copper in Ubiquitin-dependent protein degradation in Alzheimer’s disease

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Disruption to copper homeostasis is a feature of Alzheimer’s disease (AD). Recently it was discovered that copper reduces Amyloid Precursor Protein (APP) endocytosis from the plasma membrane and promotes its ubiquitination. The importance of this finding is underlined by studies that indicate that endocytosis is a key step in amyloidogenic processing of APP to form neurotoxic amyloid beta (Aβ) peptides. Ubiquitin plays a fundamental signaling role in proteasome-mediated protein degradation, endocytic protein sorting and targeting membrane proteins to lysosomes for degradation via autophagy. My hypothesis is that APP amyloidogenic processing is modulated by copper-responsive ubiquitination of APP, signaling it towards a degradative pathway rather than an endocytic pathway where it encounters the enzymes responsible for Aβ generation, namely P- and P-secretase. Specific aims: Aim 1: To determine the role of Cu-responsive ubiquitination of APP
on its localization and degradation in cultured mouse neurons. Aim 2: To compare Cu-responsive ubiquitination of APP in differentiated neurons that have been re-programmed from healthy and AD patient human fibroblasts using induced pluripotent stem cells (iPSCs). Aim 3: To determine if mutations that cause familial AD affect Cu-responsive ubiquitination of APP using cultured mouse and human fibroblasts. Aim 4: To identify novel Cu-responsive ubiquitin targets in AD-affected and healthy control fibroblasts using an ubiquitin-omics approach. I propose that copper is a physiological co-factor for the ubiquitination of APP, a neuroprotective mechanism that reduces the level of amyloidogenic processing. There is evidence from human clinical and animal trials that drugs designed to restore brain metal homeostasis provide therapeutic benefit for the treatment of AD. However, knowledge of their mechanism of action is still limited. This study will provide vital molecular information on the effect of copper on ubiquitination and provide new insight into the mechanism of action of metal ionophores, a promising disease modifying AD therapy.

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Promoting microglial function in Alzheimer’s disease through copper delivery

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Despite intensive research into disease mechanisms of Alzheimer’s disease (AD), an overarching problem is the lack of translatability of treatments to human disease. A significant number of promising therapeutics emerged from animal studies yet have failed to translate clinically, reflecting the limitations of currently available AD models. Thus the need for a physiological preclinical model for drug screening in AD is clear. The importance of microglia to inflammatory and phagocytic processes in AD, particularly in late onset AD, and their emerging role in synaptic plasticity indicates their presence as vital to a preclinical AD model. Additionally, the reported differences between human and murine microglia indicate that human-derived models are essential to fully appreciate the role that these cells play in AD. Currently no effective protocols exist for the generation of human microglia, either through direct reprogramming or from stem cells. Our collaborator recently published a novel bioinformatic tool, Mogrify, to predict transcription factor networks that control cell identity allowing effective reprogramming from one somatic cell to another. In order to develop patient-specific preclinical models with improved translatability we will use Mogrify and transcription factor-mediated reprogramming to directly obtain mature microglia from fibroblasts, stem cells and monocytes from AD and cognitively normal patients. We will use the reprogrammed Alzheimer’s microglia and 3D co-cultures containing microglia, neurons and astrocytes to investigate the mechanism of action of potential therapeutics on amyloid phagocytosis, transcriptional changes and synaptic remodelling in AD. We will test a family of neuroprotective and bioavailable copper-delivering compounds developed by our team. These studies will provide mechanistic insights into action of copper compounds on microglia to inform the development of next generation compounds for AD.
The utility of mass spectrometry for investigating iron proteins in Alzheimer's disease

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Metals such as copper, zinc and iron exist in trace amounts, but are essential to all life and play a critical role in cellular processes in the brain. When metal homeostasis in the brain is altered, such as via the accumulation of metals with age, neuronal damage, oxidative stress and brain pathologies are observed. This accumulation includes labile metals and metalloproteins, and can lead to neurodegenerative effects such as those seen in Alzheimer’s disease. Alzheimer’s is the leading cause of dementia, and affects nearly 350,000 Australians at a cost of $4.9 billion in direct health care expenditure. Many proteins involve a metal co-factor, but only a small fraction of these have been characterised. Traditional proteomics techniques do not reveal information on the metal status of a protein. Further, the current approach to metal biology involves bulk analysis of total metal levels in tissue, and does not provide information on the number or type of metalloprotein alterations. The lack of mechanistic detail from these approaches has led to only limited insight. Rather than focusing solely on the presence of the individual metal species, advancement of the field hinges on understanding the specific relationship between biometals like iron and proteins. This project will generate functional information on iron-containing proteins in the Alzheimer's brain using newly developed mass-spectrometry techniques targeted for the measurement of metal-containing proteins. This will provide detailed information on the fundamental molecular mechanism of iron in Alzheimer's disease, and more broadly, aid in understanding the role of metals in neurodegenerative disease.

Forging New Links: A New Theory for the Role of Iron Metabolism in Neurodegenerative Disease

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Curing dementia requires new targets for drug discovery. Using Caenorhabditis elegans as a tractable model system, I will examine deleterious changes in redox metabolism by creating new transgenic C. elegans, expressing both genetically encoded redox sensors and proteins associated with neurodegenerative disease, e.g. Aβ. These novel animal models will be used to examine how age-related change in iron/mitochondrial/neuronal redox chemistry drive neurodegenerative disease pathogenesis. I will use my knowledge of X-ray spectroscopy to characterise the changes in iron coordination that drive neurodegenerative disease and evaluate the neuronal proteome to identify age- and disease-specific changes to the...
complement/function of mitochondrial proteins. These activities will accelerate the search for a cure by identifying gene products that modulate mitochondrial function/iron homeostasis during neurodegeneration. By harnessing world-leading technology and implementing novel analytical approaches to data reduction I have already demonstrated that X-ray microscopy can visualise the distribution of metalloproteins in vivo [1]. This provides a unique tool for studying ferrobiology of disease. This fellowship capitalises on unique Australian resources, including the renowned dementia research expertise of the Florey Institute for Neuroscience and Mental Health and world-leading capabilities of the Florey, University of Melbourne and Australian Synchrotron. [1] SA James et al, ‘XANES: In vivo imaging of metal-protein coordination environments’, Sci Reports (2016) 6: 20350

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Poster Presentation
Theme: No Theme Allocated
Poster number: P081

Novel targeted degradable multifunctional poly(vinyl-co-ester) nanoparticles for Alzheimer’s disease applications

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The aim of the proposed project is the design of novel biodegradable multifunctional carriers based on poly(vinyl-co-ester)s, which can be readily imparted with stealth and targeting properties, as the next generation brain delivery systems. The nanoparticle systems will be designed to be able to co-deliver diagnostic and therapeutic cargo for Alzheimer’s disease. A range of well-defined glycopolymers will be synthesized and tested for their ability to prevent the aggregation of amyloid β.

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Poster Presentation
Theme: 1. Diagnosis/Assessment
Poster number: P041

A scoping study for the Australian National Dementia Registry

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Introduction: Clinical registries are databases collecting information about patients diagnosed with a particular disease, patients who use a particular health resource or undergo a particular procedure. The current scoping study looks at the feasibility and sustainability of establishing an Australian national registry of patients diagnosed with dementia, irrespective of type of dementia or age. Method: The scoping study is informed by review of the policy documents on clinical (quality) registries in Australia (ACSQHC, 2014), a systematic review of the international literature and online resources on dementia registries.
worldwide. The study involves consultations with relevant stakeholders and experts, including dementia researchers, carers of patients with dementia, managers of existing clinical registries, data linkage specialists, and ICT services. **Result:** The review of the literature and online sources identified 24 dementia and/or Alzheimer’s disease registries worldwide, including population-based, research, quality, and case registries, as well as research volunteer registries. Stakeholders and expert consultations, and the review of relevant Australian policy documents, have helped to identify potentially effective recruitment strategies, and the minimum dataset for the planned Australian National Dementia Registry. This process has also helped to identify practical, ethical, and legislative challenges in development of the online database for the registry, in recruitment of patients with dementia and their carers, and in future data linkage. The next major step in the ongoing scoping study is determining funding sources which will ensure successful development and long-term operation of the registry. **Discussions and Conclusion:** The planned Australian National Dementia Registry has a potential to be an invaluable tool to collect clinical and epidemiological data on dementia, to monitor performance of health and aged care services, and facilitate participation in treatment trials. These data can improve the quality of care for people with dementia and their carers, and support and stimulate dementia research in Australia. Australian Commission on Safety and Quality in Health Care (2014). Framework for Australian clinical quality registries. Sydney: ACSQHC.

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Poster Presentation  
Theme: 1. Diagnosis/Assessment  
Poster number: P010

**Understanding irony: Employing The Awareness of Social Inference Test to inform differential diagnosis of dementia**

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**Introduction:** Assessment of social cognition is increasingly recognised as an important component towards an accurate clinical diagnosis of behavioural-variant frontotemporal dementia (bvFTD). It is also helpful for differentiating bvFTD from typical Alzheimer's disease (AD). Surprisingly, however, few validated clinical instruments to assess social cognition exist. The Awareness of Social Inference Test (TASIT) assesses interpretation of basic emotions, sincere and sarcastic interactions, using ecologically valid video vignettes. While it has shown good sensitivity to detect bvFTD, its lengthy administration has limited the translation of the TASIT to the clinic. **Method:** Here, we evaluated the new short version of the TASIT “the TASIT-S” in 25 bvFTD patients, 23 AD patients and 25 healthy controls. The TASIT-S was recently developed using Rasch analysis and confirmatory factor analysis to reduce the number of items, while maintaining the structure of the original TASIT and is divided into: Part 1 (Emotion Evaluation) and Part 2 (Social Inference), which is subdivided into ‘Sincere’ and ‘Sarcastic’ exchanges. **Results & Discussions:** On Part 1, both bvFTD and AD groups were impaired when compared with controls (p values <.001). After controlling for cognitive impairment, using Addenbrooke’s Cognitive Examination-Revised, however, only bvFTD were impaired (p=.034), whereas the AD group was not significantly different from controls.
(p=.492). On Part 2, both bvFTD and AD group performed within normal limits in their ability to interpret sincere exchanges (p values >.05). Importantly, however, the bvFTD group was impaired in the interpretation of sarcastic exchanges (p=.004), whereas again AD performed within normal limits (p=.477), even after accounting for cognitive ability. **Conclusion:** These results confirm the utility of the TASIT-S in identifying social cognition impairment in bvFTD. The test is much shorter than the original TASIT (administration time ~20 mins) and should be included in the clinical assessment when considering a differential diagnosis of bvFTD.

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**Theme:** 4. Prevention  
**Poster number:** P062

**Computerised Cognitive Training in Older Adults with Parkinson disease, Mild Cognitive Impairment or Dementia: Convergence and Divergence across Meta-Analyses**

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**Introduction:** Computerised cognitive training (CCT) is a safe and efficacious intervention for cognition in healthy older adults, but efficacy varies across domains and design choices, and little is known about the effects of CCT in older adults with cognitive impairments. **Method:** We searched Medline, Embase, PsychINFO, CINAHL and CENTRAL for randomised controlled trials (RCTs) of CCT in older adults with Parkinson disease, mild cognitive impairment (MCI) or dementia. Overall cognition, individual cognitive domains, psychosocial functioning and everyday function were pooled separately for each population. **Results:** The overall effect on cognition in MCI across 13 RCTs was small (Hedges g=0.29, 95%CI 0.126\(\pm\)0.45). Small to moderate effects were found for global cognition, attention, working memory as well as learning and memory with the exception of non-verbal memory. In dementia RCTs statistically significant effects were found on overall cognition (k=11, g=0.26, 95%CI 0.016\(\pm\)0.52) as well as visuospatial skills and psychosocial functioning, but these pooled effects were driven by three trials of virtual reality or Nintendo Wii. Seven RCTs in Parkinson disease revealed a small and statistically significant effect size on overall cognition (g=0.23, 95%CI 0.014\(\pm\)0.44). Larger effects were noted on working memory, processing speed and executive function. There were no evidence for statistical heterogeneity or publication bias and no adverse events were reported. **Discussions and conclusion:** CCT is efficacious on overall cognition in people with MCI or Parkinson disease. Domain-specific effects vary across populations. This technique warrants longer-term and larger trials that examine effects on conversion to dementia. Conversely, evidence for efficacy in people with dementia is weak and limited to trials of immersive technologies.

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**Theme:** 4. Prevention  
**Poster number:** P063

**Zebrafish models of familial Alzheimer’s disease for understanding molecular mechanisms and drug discovery**
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Introduction: To prevent or delay Alzheimer’s disease (AD) onset we must understand its underlying molecular mechanisms. In human disease modeling, zebrafish can offer the most sensitive detection of changes in gene and protein expression in response to mutation. We have developed and analysed the first zebrafish models of familial AD (fAD) mutations in the PRESENILIN genes (in which the majority of fAD mutations occur). Methods: We analysed early changes in brain gene and protein expression due to single, heterozygous mutations in the zebrafish’s endogenous PSEN1 gene. These represent the initial molecular events and stresses that, in humans, lead to development of AD decades later. Results: 1) Single, heterozygous, endogenous fAD mutations cause very significant changes in young adult (6 month) brain gene and protein expression 2) Changes in behavior and gene expression are seen in 6- and 3-day-old fish respectively. This permits use of these fish in screening of chemical libraries 3) Systems biology analysis of gene and protein expression data implies changes in ATP, insulin signalling, and other cellular systems as early, common effects of fAD mutations. 4) These effects apparently occur in the absence of Amyloidbeta accumulation but may promote Amyloidbeta accumulation at later stages 5) Systems biology analysis shows that recognised risk factors for late onset AD cause similar profiles of altered brain gene expression as the fAD mutations. 6) Neurodegenerative changes may exist in 2-year-old (aged, infertile) fish. Conclusions: A) We can use zebrafish to understand the initial molecular changes that ultimately lead to AD B) Both systems biology-based interrogation of drug databases and chemical library screening with zebrafish may find drugs to prevent or delay AD onset C) The dramatic molecular and behavioural changes observed from single, heterozygous, endogenous fAD mutations imply that animals with multiple mutant transgenes are not close models of the disease.
is still seen as largely consistent with a central role for Amyloidbeta peptide in disease pathogenesis. But is it? Can the genetic data be interpreted from a different angle to provide a more predictive view of disease mechanism? Methods & results: The majority of dominant mutations causing fAD occur in the PRESENILIN genes PSEN1 and PSEN2. These genes encode ‘holoproteins’ that become cleaved internally to activate the gamma-secretase activity that produces Amyloidbeta. In a recent review in the Journal of Alzheimer’s Disease we proposed that the PSEN genetic data is consistent with a central role in fAD of the PSEN holoprotein rather than gamma-secretase activity. Data from our PSEN fAD mutation model zebrafish now indicate that failure of PSEN holoprotein function may also contribute to the sporadic, late onset form of AD. Published evidence indicates a central role of PSEN holoproteins in cells’ ability to cope with hypoxia and that PSEN holoprotein is required for efficient lysosome function and breakdown of protein aggregates. Conclusions: Research into the function of the holoprotein forms of the PSENs may be a fruitful avenue for understanding the basis of both early and late onset forms of AD. Interventions promoting healthy brain vasculature and lysosome function may prevent or delay AD onset.

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Poster Presentation
Theme: 2. Care/Living With Dementia
Poster number: P039

A telehealth intervention to delay functional decline in community-dwelling people with dementia

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Introduction: Functional decline is one of the core features of dementia and is associated with reduced quality of life, considerable impact on carers, high healthcare costs and institutionalisation. There is evidence that non-pharmacological interventions that promote functional independence and provide the carer with skills training can delay decline, reduce carer impact and improve quality of life of the person with dementia. The use of telehealth technologies to deliver the intervention may reduce the costs of delivering the intervention, increase accessibility and facilitate research translation. Method: This research project involves two phases. First, we will conduct a pilot study involving ten people with dementia and their carers to determine the feasibility of delivering the intervention using telehealth. The first two consultations will take place in the home and involve assessment and familiarisation with the technology. The remaining consultations (up to eight) will be conducted via videoconferencing technology. The feasibility study will inform a larger study in which we compare the efficacy of telehealth delivery of the program with face-to-face delivery. Result: The feasibility project will provide information about the proportion of people (and their carers) that are willing and able to participate, the modifications required for telehealth delivery and the acceptability of telehealth delivery. The larger trial will provide information regarding efficacy (functional independence, quality of life, activity engagement and symptoms) and costs of care. Discussions and Conclusion: There is a need to investigate new strategies to translate evidence based interventions that delay functional decline into clinical practice. If found to be effective, this approach may be particularly useful for people living in rural and remote areas who have limited access to staff with expertise in dementia care.
Common and divergent neural correlates of anomia in amnestic and logopenic presentations of Alzheimer's disease

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Introduction: The majority of logopenic variant primary progressive aphasia (lv-PPA) cases harbour Alzheimer pathology in the brain, suggesting that lv-PPA constitutes an atypical presentation of Alzheimer's disease (AD). However, even if caused by Alzheimer pathology, clinical manifestations of lv-PPA differ from those observed in typical AD: in lv-PPA, aphasia is the main feature while typical AD is characterised by impaired episodic memory. Anomia or impaired naming, however, is present in both disorders. Whether these AD presentations share anatomical and mechanistic neurocognitive processes of anomia has not been fully investigated. Methods: We studied naming and other single-word performance, and its relationship with regions of brain atrophy in 23 typical AD and 22 lv-PPA cases with presumed underlying Alzheimer pathology. All cases underwent MRI and cortical thickness calculations using FreeSurfer. Result & Discussions: Whereas both AD groups displayed some degree of anomia and impaired word comprehension, those deficits were severe in lv-PPA and accompanied by a range of linguistic deficits, comprising phonological substitutions, superordinate semantic paraphasias and abnormal single-word repetition. Analysis of cortical thickness revealed that anomia was correlated with thinning in the left superior temporal gyrus in both groups. In typical AD it was also associated with thinning in the right inferior temporal regions. The analysis of single-word comprehension in turn evidenced convergent cortical thinning involving both fusiform gyri in typical AD and in lv-PPA. These findings suggest that these common areas of atrophy are involved in the shared semantic deficits of both groups. Conclusion: This evidence shows that anomia in lv-PPA and typical AD results from the common involvement at multiple steps of word processing, in particular semantic and lexical retrieval; however, lv-PPA display a more marked involvement spanning phonological processing.
Background: The brain-derived neurotrophic factor (BDNF) Val66Met polymorphism is implicated in synaptic excitation and neuronal integrity, and has previously been shown to moderate Aβ-related memory decline and hippocampal atrophy in preclinical sporadic Alzheimer's disease (AD). However, the effect of BDNF in autosomal dominant AD (ADAD) is unknown. We aimed to determine the effect of BDNF Val66Met on cognitive function, hippocampal function, tau and Aβ in preclinical ADAD. We explored effects of apolipoprotein E (APOE) ε4 on these relationships. Methods: The Dominantly Inherited Alzheimer Network (DIAN) conducted clinical, neuropsychological, genetic, biomarker and neuroimaging measures at baseline in 131 mutation non-carriers (NC) and 143 preclinical ADAD mutation carriers (MC) on average 12 years prior to clinical symptom onset. BDNF genotype data were obtained for MCs (95 Val66 homozygotes, 48 Met66 carriers).

Results: Among preclinical MCs, Met66 carriers had worse memory performance, lower hippocampal glucose metabolism and increased levels of CSF tau and phosphorylated tau (p-tau) than Val66 homozygotes. Cortical Aβ and CSF Aβ42 levels were significantly different from NC’s but did not differ between preclinical MC Val66 homozygotes and Met66 carriers. There was an effect of APOE on Aβ levels but not cognitive function, glucose metabolism or tau.

Discussions and Conclusions: As in sporadic AD, the deleterious effects of Aβ on memory, hippocampal function, and tau in preclinical ADAD mutation carriers are greater in Met66 carriers. To date, this is the only genetic factor found to moderate downstream effects of Aβ in ADAD.

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Poster Presentation
Theme: 1. Diagnosis/Assessment
Poster number: P013

Genetic risk prediction for selection of those at extremes of risk for Alzheimer's disease in the PISA study

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Introduction: While the burden of dementia occurs late in life, neuropathology accumulates for decades prior to onset of dementia. Interventions to modify the course of the disease have the greatest potential to avert neuronal death and later disease burden if they are introduced during this crucial window, well before the onset of clear cognitive decline. It is thus imperative to develop methods to identify those at the very early stage of dementia. This is the aim of the Prospective Imaging Study of Aging: Genes, Brain and Behaviour (PISA) study, which seeks to 1) Identify healthy middle-aged Australians at high risk of dementia; 2) Discover biological markers of early neuropathology; 3) Identify modifiable risk factors, and 4) Establish the very early phenotypic and neuronal signs of disease conversion. Method: We are utilizing APOE genotype and polygenic risk scores (PRS) to identify individuals at high and low risk of AD. We are leveraging our extensive in-house cohorts, comprising ~16,000 individuals between the ages of 40 and 70yrs with available GWAS data to generate a genetically enriched cohort for studying the precursors and lifestyle risk factors for AD. Result & Discussions: For AD, a high prediction accuracy of an AUC of 78.2% can be achieved by a prediction model including APOE genotype, and PRS (containing GWAS association SNPs
with P value <0.5) with the PRS adding significant predictive value over APOE alone. Our own work has shown that the AD PRS is associated with reduced hippocampal volume in those without a dementia diagnosis, including healthy older adults and those with mild cognitive impairment (MCI). **Conclusion:** Using cutting-edge genetic prediction, the PISA study is an at-risk AD cohort enriched by genome-wide risk prediction, aiming to identify early markers of prodromal Alzheimer’s disease that are detectable as early as middle age.

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Theme: 4. Prevention  
Poster number: P064

**A multi-faceted intervention to enhance cognition in older people at risk of cognitive decline**

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**Introduction:** Rapid population ageing is leading to an increasing proportion of the population living with cognitive impairment and dementia. There is an urgent need to focus on preventative actions to reduce the future number of dementia cases. The pathophysiology of dementia and cognitive decline is complex, therefore a multi-faceted approach incorporating both exercise and dietary factors may convey greater cognitive benefits than a single intervention administered in isolation. Few studies have taken the approach of combating cognitive decline by combining exercise with dietary supplementation. This study will be a 6-month randomised controlled trial (RCT) to investigate the combination of a multimodal exercise program, combined with omega-3 fatty acid, vitamin D and protein supplementation, to enhance cognition in older people experiencing early signs of memory impairment. This study will also evaluate the longer-term impact of the multi-faceted intervention, including the ability of individuals to incorporate these changes into their lifestyle. **Methods:** This 12-month, community-based, double-blind, placebo controlled, randomised trial will involve a 6-month supervised and structured program followed by a 6-month maintenance (translation) phase. Participants (n=148) with subjective memory impairment (SMI) aged 60-85 years will be randomised to: 1) a multi-modal exercise program involving progressive resistance training (PRT) and aerobic training combined with omega-3, vitamin D and protein supplementation, or 2) a sham exercise program and placebo supplements. **Results and discussion:** Recruitment for this study will commence in October 2016 and baseline testing will commence in January 2017. It is anticipated data collection for this study will be complete in early 2019. **Conclusion:** In summary, this study will target prevention and early intervention through a novel combination of exercise and dietary supplements in elderly who are at risk of further cognitive decline.

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Theme: 3. Intervention/Treatment  
Poster number: P052

**The effect of aromaticity on short peptide hydrogels for cell culture applications**
Introduction: Self-assembled hydrogels assemble through non-covalent interactions such as stacking and hydrogen bonding, and have been used to culture a variety of cell lines including HeLa, Caco-2 and fibroblasts. Their tuneable nature makes them ideal candidates for three dimensional cell culture, as their properties can be tuned upon modification of either the peptide backbone or aromatic N-terminal capping group. This capping group is often the widely used as a protecting group fluorenylmethyloxycarbonyl, or Fmoc, however in this work a variety of heterocyclic capping groups are used to control the properties of an N-capped diphenylalanine gelator. Method: Gelators were prepared through standard solid phase peptide synthesis techniques. Hydrogels were formed using a pH switch method, whereupon the gelator was dissolved at pH 9, followed by acidification using glucono-delta-lactone, resulting in gel formation. Hydrogels were characterised using rheology, atomic force microscopy (AFM), circular dichroism (CD) and electrochemical impedance spectroscopy (EIS). Results & Discussion: Four different capping groups were employed, where either the hydrogen bonding potential or degree of nitrogen substitution on the N-terminal capping group is varied, to probe the effect of these parameters on self-assembly. Zeta potential measurements and EIS were used to monitor the self-assembly process during gelation. The morphology of the hydrogel has been studied by AFM, Figure 1, and the mechanical strength of these hydrogels tested using rheology. Conclusion: From characterisation across different length scales, a model of the self-assembly for these four gelators can be discerned. The morphology of the fibres seen by AFM is due to different self-assembly pathways, and this strongly correlates to the different stiffnesses observed by rheology. This work gives an insight into the factors governing the self-assembly of short peptide gelators and will be useful in the future use of these hydrogels as three dimensional cell culture materials.

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Poster Presentation
Theme: No Theme Allocated
Poster number: P075

Biometal Dyshomeostasis in Dementia with Lewy Bodies

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Dementia with Lewy bodies (DLB) is the second most common form of dementia after Alzheimer’s disease (AD) accounting for up to 1 in 4 of all dementia cases. DLB is characterised by fluctuations in mental state, visual hallucinations and parkinsonism. The predominant pathological feature of DLB is the presence of ß-synuclein Lewy bodies and Lewy neurites in the brainstem, limbic region and cortical areas. Lewy bodies are round, filamentous inclusions whereas Lewy neurites are diffuse presynaptic ß-synuclein aggregates which affect synaptic function. Lewy pathology is also a feature of several other neurodegenerative diseases including Parkinson’s disease (PD) and multiple system atrophy. Collectively, these conditions are termed synucleinopathies and likely share common pathogenic mechanisms that lead to cell death and tissue atrophy. Of these synucleinopathies, only PD has been extensively studied to date. In addition to its PD-like
pathology, DLB also shares pathology with AD including amyloid-beta (A-beta) plaques and tau neurofibrillary tangles. There is little known about the pathogenesis of DLB and it is largely assumed to be similar to AD and PD given the pathological and symptomatic overlap. Due to this lack of understanding, there are no specific therapies for DLB and treatment relies on AD therapeutics which do not alter progression of the disease. Understanding underlying disease mechanisms is crucial for identifying valid targets for therapeutic intervention. Metal dyshomeostasis has been implicated in the pathogenesis of both AD and PD and is a major target of ongoing development of new therapeutics for these conditions. The convergence of ß-synuclein, A-beta and tau pathology in DLB suggests a role for metal dyshomeostasis in the pathogenesis of DLB. This fellowship will extend current knowledge of AD and PD to investigate biometal dyshomeostasis in DLB and whether it represents a valid target for the development of disease modifying therapeutics.

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**Theme:** 2. Care/Living With Dementia
**Poster number:** P040

**BPSD-CARE: a person-centred approach to managing behavioural and psychological symptoms of dementia in residential care**

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**Introduction:** Rates of behavioural and psychological symptoms of dementia (BPSD) amongst people living in residential aged care facilities (RACF) are high. Over 90% of Australian aged care residents exhibit clinically significant BPSD. BPSD, especially when severe, are difficult to manage and can put patients, carers and residents at risk. Sweden has a long tradition of quality registries aimed at securing high quality care in a variety of clinical settings. The Swedish BPSD registry program was initiated to ensure and improve residential care for people with BPSD, reduce BPSD and improve quality of life. This project adapts the Swedish BPSD program for use in Australia (BPSD-CARE). **Method:** Participants (care staff, residents, next-of-kin) will be recruited from Goodwin Aged Care. Eligible residents will be continuously recruited into the study over a 24 month period and complete the 10 month intervention program. The intervention comprises the active use of the BPSD-CARE program in combination with regular online tutoring and education of staff. **Results & Discussions:** Efficacy of the BPSD-CARE intervention program will be evaluated using pre- and post-measures and a within-subjects repeated measures design. Semi-structured interviews with RACF staff and next-of-kin will assess perceptions of the efficacy of the intervention. This project will evaluate the efficacy of BPSD-CARE to reduce the prevalence of BPSD and the use of medication to manage BPSD in Australian RACF. The unique contribution of this research is the evaluation of BPSD-CARE on RACF staff, their attitudes towards dementia care and quality of interactions with residents. For the first time, impact of BPSD-CARE on next of kin satisfaction with the care provided will also be evaluated. **Conclusion:** This project will inform the development of a program which will provide the multidisciplinary teams working within RACF with the specialised training and skills needed to provide care for residents with BPSD.

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Poster Presentation  
**Theme:** 3. Intervention/Treatment  
**Poster number:** P053

**Effects of BACE inhibition on synaptic connectivity**

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**Overview:** Inhibition of BACE1 (β-site amyloid precursor protein cleaving enzyme 1) is a promising future treatment for Alzheimer’s disease which aims to decrease production of the amyloid-β peptide from the amyloid precursor protein. BACE inhibitors also affect the functions of multiple proteins which are not associated with Alzheimer’s disease pathology, but rather have important roles in the brain. In particular, the Seizure-related gene 6 (Sez6) family of proteins, Sez6, Sez6-like (Sez6L) and Sez6-like 2 (Sez6L2), are major BACE1 substrates. Sez6 is required for the normal development of dendrites and excitatory synapses(1) and plays an ongoing role in excitatory synapse function in the adult mouse brain (Munro & Carrodus et al, unpublished). In this study, we will assess whether long-term BACE1 inhibition compromises synapse function, focusing initially on the altered activity of Sez6 family proteins.  

**Aims:** 1) Identify neuronal and behavioural changes associated with chronic BACE inhibition, and the extent to which key BACE1 substrates contribute to these outcomes. This is conducted in vitro in neuron cultures and through the use of wild-type and Sez6 family knockout mouse lines. 2) Quantify changes in the synaptic proteome following chronic BACE inhibition. 3) Identify consequences of BACE1 deletion in neurons important for learning and memory. 4) Identify clinically relevant biomarkers of BACE inhibitor efficacy.  

**Results:** When a β-secretase inhibitor (C3, Millipore) was applied to cultured mouse cortical neurons (after the development of dendrites), a decrease in synapse number was observed in wild-type but not Sez6 family knockout neurons. This indicates that preventing the production of BACE1 shed Sez6 family ectodomains decreases synapse number in vitro.  

**Conclusion:** Sez6 family proteins are BACE1 substrates that play an important role in synapse formation, maintenance and behaviour. (1) Gunnersen, J.M., et al., Sez-6 proteins affect dendritic arborization patterns and excitability of cortical pyramidal neurons. Neuron, 2007. 56(4).

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**Poster Presentation**  
**Theme:** 4. Prevention  
**Poster number:** P065

**Development of a unified list of drugs associated with drug-induced cognitive impairment**

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**Introduction:** To date 406 medicines including 225 currently marketed in Australia have confusion listed as a side effect in their product information or in reports from post-marketing surveillance. The Canadian Adverse Reaction Database also highlights that this potential adverse event (AE) occurs frequently, holding 6193 reports of suspected AEs of drug-related confusion. Despite this wealth of data, we still do not know the full contribution of medicines.
to iatrogenic cognitive impairment. This research will be conducted to develop a comprehensive list of probable drugs inducing cognitive impairment. **Methods:** First, different signal detection methods will be used in corroborating data sources to detect drugs inducing AEs of neurocognitive disorders (NCDs). Bayesian techniques will be used in the US Food and Drug Administration Adverse Event Reporting System and Australian Government Department of Veterans’ Affair (DVA) claims database. Prescription sequence symmetry analyses will also be used in DVA database to detect medicines inducing delirium. Detected associations will be classified into known or new by reviewing product information documents and conducting systematic reviews and meta-analyses of published literature on drugs inducing NCDs. The new identified signals will then be adjudicated. The drugs’ mechanism of action will be reviewed; whether the drug crosses the blood-brain barrier and causes apoptosis. Also, sources of false-positive signals will be identified; an example being using the Longitudinal Evaluation of Observational Profiles of Adverse Events Related to Drugs to examine protopathic bias when a drug is prescribed for a prodrome of the conditions of concern. Confirmatory analyses will be conducted using formal epidemiological studies in DVA dataset and Australian ongoing longitudinal population-based cohorts. **Conclusion:** The research outputs will assist prescribers in clinical decision-making, possibly avoiding the prescribing of high risk drugs for patients with a high index of suspicion for drug-induced dementia, thus preventing this type of dementia from developing.

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Poster Presentation
**Theme:** 3. Intervention/Treatment
**Poster number:** P054

**Fatty acid-binding protein 5: an intracellular protein regulating the blood-brain barrier transport of docosahexaenoic acid and cognitive function**

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**Introduction:** Docosahexaenoic acid (DHA) is an essential fatty acid required for cognitive function. The brain has limited ability to synthesise DHA and therefore plasma-derived DHA must be transported across the blood-brain barrier (BBB). This study investigated whether fatty acid-binding protein 5 (FABP5) regulates the BBB transport of DHA and therefore cognitive function. **Methods:** The uptake of 14C-DHA was measured in human brain microvascular endothelial cells (hCMEC/D3) with and without FABP5 genetic silencing and in brain microvascular endothelial cells isolated from wild-type (FABP5+/+) and FABP5 deficient (FABP5-/-) mice. The BBB transport of 14C-DHA was assessed in FABP5+/+ and FABP5-/- mice using an in situ transcardiac perfusion technique. Endogenous brain concentrations of DHA were measured in FABP5+/+ and FABP5-/- mice using gas chromatography with flame ionization detection, and cognitive function was assessed using a modified water maze, novel object recognition, and T-maze memory paradigms. **Results:** FABP5 siRNA transfection decreased FABP5 mRNA in hCMEC/D3 cells by 53.2 ± 5.5%, and this was associated with a 44.8 ± 13.7% reduction in FABP5 protein expression and 14.1 ± 2.7% reduction in 14C-DHA cellular uptake. 14C-DHA uptake into brain endothelial cells from FABP5-/- mice was reduced by 48.4 ± 14.5% relative to those from FABP5+/+ mice. The BBB transport of 14C-DHA was decreased by 36.7 ± 12.4% in FABP5-/- mice and this was associated with a 27.4 ± 10.3% reduction in endogenous brain DHA levels. FABP5-/- mice exhibited decreased spontaneous alternations (T-maze), a lower discrimination index (novel object recognition) and impaired spatial learning (water maze). **Discussion and Conclusion:** This study has demonstrated that
FABP5 regulates the BBB transport of DHA, playing an important role in maintenance of cognitive function. FABP5 may therefore be potentially manipulated to enhance the CNS access of DHA in conditions where the brain levels of DHA are decreased, such as Alzheimer’s disease.

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Poster Presentation
Theme: 3. Intervention/Treatment
Poster number: P083

The role of proteoglycans in neurodegeneration

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With Dementia affecting a large number of people worldwide, often with lifelong consequences, new and effective approaches to enable long-lasting therapeutic interventions are required. An estimated 298,000 Australians were diagnosed as having dementia in 2011 and this is estimated to increase to 400,000 by 2020. Stem cells have provided some positive results in the treatment of neurodegenerative disorders however, further information is required to more fully understand the mechanisms controlling disease onset and potential models of repair. Proteoglycans (PGs) are proteins ubiquitous to the cell surface and the extracellular matrix (ECM) and include two major families, the chondroitin sulfate proteoglycans (CSPGs) and the heparan sulfate proteoglycans (HSPGs). Both CSPGs and HSPGs are key components of the ECM and play important roles in neural development. Mesenchymal stem cells (MSCs) are relatively easy to obtain, have a large capacity for self-renewal, and can differentiate into a variety of cell types including neural cells. In contrast, human embryonic derived neural stem cells (NSCs) are much less abundant, difficult and controversial to obtain, and importantly have a much lower capacity for expansion and self-renewal. The proposed study will examine the HS and CS PG sugars in human MSCs compared to human NSCs during neural lineage commitment. Multiple studies have demonstrated a role for these sugars during normal development of the nervous system, however how these sugars interact with and control human stem cells and in particular how they control neural lineage commitment is as yet unknown. Methods developed by our group have improved our ability to differentiate human MSCs toward specific neural lineages and may enable us to generate lineage-specific cells in substantially greater numbers for use in therapeutic applications for treatment of dementias.

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Poster Presentation
Theme: 4. Prevention
Poster number: P066

In search of an active solution to alcohol-related dementia
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Introduction: Alcohol-related dementia is one of the leading causes of preventable dementia in Australia, and the only treatment currently available is alcohol rehabilitation. Emerging evidence from animal models, however, shows that exercise may protect against the neurotoxic effects of alcohol1. We plan to investigate whether neurotoxic and cognitive deficits arising from alcohol abuse may be recovered with abstinence combined with voluntary exercise.

Method: We will use rodent models to provide the first comprehensive analysis of how chronic alcohol exposure precipitates behavioural and neuropathological symptoms of dementia. Rats will be allowed to consume alcohol for 6 months, and then subjected to a period of enforced abstinence. Throughout abstinence, rats will have free access to a running wheel, or housed under standard conditions. Extensive analysis of neural injury and cognitive ability will be carried out at various points across the experiment. Cognitive capacity will be measured using a battery of complex tasks available on a novel touchscreen platform. These provide measures across a range of cognitive domains, are analogous to tests used on humans and therefore translationally relevant. They will provide a systematic analysis regarding the specific cognitive impairment that follows alcohol abuse, and further which domains are recoverable and which undergo irretrievable damage.

Results and Discussions: Here I will explain the validity of the behavioural models, and the clinical implications of this research. I will also present the cognitive and neuropathological profile of young naive rats which provide the baseline for this study.

Conclusion: This research will provide important evidence regarding the potential for a readily translatable intervention (voluntary exercise) to be employed in the treatment of alcohol-related dementia.

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Poster Presentation
Theme: 2. Care/Living With Dementia
Poster number: P042

Consumer Directed Care: Understanding and promoting participation and care outcomes for people living with dementia in receipt of a Home Care Package

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Overview: In Australia, policy reforms which emphasise consumer choice and control are occurring in both the aged and disability sectors. One example is Home Care Packages (HCPs) for older Australians which are now delivered within a Consumer Directed Care (CDC) framework. International literature suggests CDC presents challenges for service providers and older consumers, especially for those living with dementia. These include: the desire and capacity of the person with dementia to direct their care; the capacity and approach of care providers; and the presence and capacities of a carer. To evaluate the extent to which CDC within the HCPs program can deliver quality care outcomes for people with dementia this program of research will explore to what extent, and by what strategies the objectives of CDC can be met for people living with dementia within the HCP program.

Methods: Study 1. A systematic literature review will define concepts associated with CDC for
people with dementia. Approaches to evaluation of participation in care planning and management will also be identified. Study 2. A Nominal Group Technique will generate consumer and expert consensus regarding CDC service and client variables and methods for use with people with dementia and their carers. Study 3. Tools and methodologies will be developed and validated to assess: Capacity, Activation and Satisfaction with CDC for people with dementia and their carers; and CDC orientation of care providers. Study 4. A multi-method approach will explore control and participation in the delivery of HCPs for people with dementia including: interviews; repeated observations and use of the client and carer (proxy) measurement tools. Study 5. Outcomes will be translated in a pilot intervention to build the capacity of care providers to deliver CDC, and the capacities of consumers with dementia (and their carers) to be active participants in their care.

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Poster Presentation
Theme: 1. Diagnosis/Assessment
Poster number: P086

**Communicating the diagnosis of dementia**

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3 School of Nursing, Midwifery & Indigenous Health, Faculty of Science, Charles Sturt University, Orange

**Background:** A project is underway to revise the 2003 General Practitioner (GP) Dementia Guidelines. This poster will report one section of the revised guideline – “Communicating the Diagnosis of Dementia”. **Methodology:** Nine general practice dementia guidelines released since 2008 were reviewed. A list of topics covered by these and the 2003 GP Dementia Guidelines were constructed. Questions were formulated on each topic using an iterative process. A literature review was conducted around each topic following the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) protocol. Two groups, a Guidelines Update Committee and an advisory group, consisting of consumers, carers and experts in dementia from a variety of backgrounds provided input on this process. Sections were further developed using information from forums of carers, consumers and health professionals. The guide will be trialled in general practice and further developed. **Results & Discussion:** Section on Communicating the Diagnosis: GPs need to use a person-centred approach (e.g. consider language, culture, education, and whether the person wants to know their diagnosis); have carer(s) present if available; assess readiness and the risk of raising a strong emotional reaction to the possibility of dementia; talk to the person with dementia, not only their carer(s); provide the person with dementia, and the carer(s) with support and information when discussing the diagnosis; discuss the implications of the diagnosis and make follow-up plans. Suggestions from the forums included an example of specific wording that could be used by the GP. **Conclusion:** The guidelines provide the GP with practical advice about communicating the diagnosis of dementia.

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Poster Presentation
**Supporting the carer of a person who is living with dementia**

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**Background:** A project is underway to revise the 2003 General Practitioner (GP) Dementia Guidelines. This poster will report one section of the revised guideline – “Supporting the carer of a person who is living with dementia.” **Methodology:** Nine general practice dementia guidelines released since 2008 were reviewed. A list of topics covered by these and the 2003 GP Dementia Guidelines were constructed. Questions were formulated on each topic using an iterative process. A literature review was conducted around each topic following the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) protocol. Two groups, a Guidelines Update Committee and an advisory group, consisting of consumers, carers and experts in dementia from a variety of backgrounds provided input on this process. Sections were further developed using information from forums of carers, consumers and health professionals. The guide will be trialled in general practice and further developed.

**Results & Discussion:** Section on carer support: GPs need to provide information on dementia as a disease process with education on dementia including available financial support and tailored to the carer’s situation; consult with carer(s) on the impact of caregiving / advise carer(s) to consult with their own GP in this regard while acknowledging the positive aspects of caregiving and the good job being done; provide practical strategies to support the carer; include the carer in management of the patient whenever possible and appropriate; refer as necessary to sources of support in the community; provide written information and refer to consumer / carer organisations. **Conclusion:** The guidelines cover a range of strategies that the GP may use to provide carer support.

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**Assessing differences in demographics and risk factors between autopsy proven dementia and Parkinson’s disease patients**

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**Introduction:** Lewy bodies, which are abnormal aggregations of the synaptic protein alpha-synuclein, are a very common pathology found in the brains of patients with dementia. Recent studies have shown that more than 50% of patients with Alzheimer’s disease (AD) pathology have some Lewy bodies in the brain, and in some dementia patients (~10-15%) Lewy bodies are the major pathology found. These patients are called Dementia with Lewy bodies (DLB). Lewy bodies are also found in the brainstem of patients with Parkinson’s disease (PD). It remains unclear whether there are demographic differences between pathologically confirmed patients with PD versus DLB versus AD, and whether they have different or...
overlapping risk factors. **Methods:** Longitudinally followed AD, DLB and PD patients and controls (N=373) who donated their brains to the Sydney Brain Bank for research purposes were selected following ethics approval. All cases with AD or Lewy body pathology, as well as cases with no significant neuropathology were included. Cases with infections, cerebrovascular disease and neoplasms were excluded. Data extraction from research records will utilise a designed proforma to capture key demographic, family history, clinical and risk factor features. Chi-square analyses will be performed to determine differences between groups in these key variables. **Expected Outcomes:** From the captured data, important demographic differences and inheritance patterns for each different disease cohort will be identified, including identifying DLB families for future gene discovery. **Conclusions:** This study will determine any demographic and risk factors differences (or similarities) between DLB, AD and PD, providing cohorts for further tissue based studies to elucidate differences in pathomechanisms initiating these different dementia phenotypes.

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**Theme:** 4. Prevention  
**Poster number:** P068

**Ageing and dementia in Aboriginal Australians: promoting vitality, identifying decline and supporting communities**

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**Introduction:** Like many populations, the Aboriginal population of Australia is ageing rapidly and dementia is a growing concern and burden on communities. Prevalence of dementia is at least 3 times higher in Aboriginal peoples compared to the general Australian population, often with younger onset. However, little is known regarding the risk factors for cognitive decline, nor about the most accurate and culturally appropriate ways to identify cognitive decline in its early stages. This has a significant impact on the ability to plan and provide for appropriate prevention and early intervention strategies. **Method:** The initial development stage of the project will include analysis of existing databases and new surveys, semi-structured interviews and focus groups with Aboriginal community members to gain insight into Aboriginal perspectives on cognitive assessment and healthy ageing, along with key risk factors for cognitive decline. This will support development of an evidence-based healthy brain ageing program in collaboration with older Aboriginal people. The second evaluation stage will involve a preliminary randomized controlled trial with 100 older Aboriginal people to assess whether a multifaceted program using computer technology can improve cognitive function, increase physical activity levels and improve quality of life. **Results:** This study will generate culturally appropriate cognitive assessment resources and validated strategies for promoting healthy brain ageing. This will improve functioning and wellbeing for older Aboriginal people in the short-term and ultimately aims to prevent or delay onset of dementia in this high risk population. **Discussion and Conclusion:** Globally, many cases of dementia are potentially preventable and this is also likely in Aboriginal communities, but culturally sensitive approaches are required. The extensive formal consultation and analysis at the core of the project will provide the foundations for future development of innovative dementia prevention strategies, with the potential for all Australians to benefit.
How do mutations in autophagy receptors cause FTD and ALS?

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Introduction: Frontotemporal dementia (FTD) and amyotrophic lateral sclerosis (ALS) share genetic, clinical and pathological overlap. A number of genes implicated in both diseases code for autophagy receptors. In living cells autophagy breaks down protein aggregates, however these aggregates are characteristically present in post-mortem diseased neurons. Autophagy receptors target specific proteins to the autophagic machinery, including aggregate-prone proteins that are important in FTD/ALS pathology (e.g. SOD1, tau, TDP-43). We will determine if disease-associated autophagy receptor variants (e.g. SQSTM1/p62, OPTN, UBQLN2, VCP) alter common pathways that impair the targeting or breakdown of protein aggregates. Methods: We will express autophagy receptors (wild type or variant) in NSC34 cells, then isolate the expressed proteins and bound interacting partners by immunoprecipitation. Proteins will be trypsinised and identity determined by mass spectrometry. Our bioinformatics pipeline (R-studio, String and KEGG analysis) will determine biological pathways affected by expression of disease variants when compared with normal counterparts. This will identify proteins and protein pathways that are aberrantly regulated in the presence of defective autophagy receptors. Results & discussion: We have performed pilot studies and show that the autophagy receptor SQSTM1/p62 lacking an ubiquitin-associated (UBA) domain, ΔUBA, has a different protein interaction network compared with the wild type protein. The ΔUBA protein interactome was enriched for heat shock proteins (protein chaperones), autophagy mediators known to be involved in autolysosome formation and proteins associated with Alzheimer’s disease and ALS/FTD (including TDP-43). Conclusions: Our proof-of-concept pilot study shows that protein networks, including those that may be involved in neurodegeneration, are affected by the expression of a disease variant. Future experiments will expand on this concept to include additional SQSTM1/p62 variants as well as variants in the autophagy receptors OPTN, UBQLN2 and VCP. Using an integrated bioinformatics approach we will identify pathways that may later be targeted for therapeutic benefit.

Development and implementation of evidence-based deprescribing guidelines for people with dementia

Emily Reeve*1, 2, 3, 4, Barbara Farrell5, 6, 7, Kenneth Rockwood3, 4, Sarah Hilmer1, 2, 8
Introduction: People with dementia (PWD) often take multiple medications to treat the symptoms of dementia and their other co-morbidities. Approximately 50% of PWD take 5 or more regular medications, so-called polypharmacy, which is associated with increased adverse drug reactions, hospitalisations and mortality. Ensuring optimal medication use in PWD involves consideration of medical, functional and social issues and goals of care. It involves both prescribing medications that will help achieve these goals and deprescribing medications for which risk may outweigh benefit. Unfortunately, half of older adults with dementia are taking at least one medication where the potential harms outweigh the potential benefits, and therefore this medication(s) should be considered for deprescribing. There are currently no deprescribing guidelines for PWD, which GPs report as a significant barrier to optimising medication use in this population. The aim of this project is to develop medication class-specific, evidence-based deprescribing guidelines for people with dementia and implement them in Australia.

Methods: The guidelines will be developed following a GRADE based process. Briefly, a guideline development team will review the literature and formulate recommendations which will then undergo external review by clinical experts, end-users and stakeholders. The guidelines will address when deprescribing of specific medications (cholinesterase inhibitors, memantine and benzodiazepines) may be considered and how to conduct deprescribing of these medications (i.e. whether it needs to be tapered and what monitoring should be conducted). It will also include a review of clinical considerations including patient and carer views towards withdrawal, with a focus on the individuals’ lived experience and consumer relevant outcomes. Implementation will consist of a multi-faceted approach including online educational activities and consumer directed strategies. Discussion and Conclusions: Evidence-based deprescribing guidelines for people with dementia will address a significant barrier to deprescribing inappropriate medications in practice which may result in improved patient outcomes.

Implementation and validation of the Centiloid transformation

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Background: A common quantitative output value for A\(\beta\) imaging across tracers and methods will improve clinical and research use. A method has recently been developed by an international team of A\(\beta\) imaging experts for this purpose that produces a unit of measurement called the Centiloid (Klunk et al, Alzheimers Dement, 2015). This approach was
implemented on Aβ imaging studies performed with 18F-NAV4694 (NAV) and 11C-PiB (PiB).

**Methods:** Fifty-five participants underwent PET imaging between 50-70 min after injection of PiB and NAV: 10 healthy young controls (33±7 yo), 25 healthy elderly controls (74±8 yo, MMSE 29±1), 10 mild cognitive impairment (75±9 yo, MMSE 27±3), 3 frontotemporal dementia (68±5 yo, MMSE 27±1), and 7 Alzheimer’s disease (73±11 yo, MMSE 24±2) patients. Spatially normalized images were analyzed using the standard Centiloid regions (cortex and whole cerebellum reference region) downloaded from the Global Alzheimer’s Association Interactive Network website (GAAIN; http://www.gaain.org). The non-standard reference regions, cerebellar cortex, pons, and whole cerebellum+pons were also investigated. **Results:** Both radiotracers presented an almost identical dynamic range of neocortical SUVR (linear slopes=1.09±0.01) and Centiloid values, the latter ranging from -30 to 130 Centiloids. Both tracers were highly correlated (R2>0.97), irrespective of the reference region used for the scaling. We further validated the Centiloid transformation by comparing the results from the standard approach and our own imaging analysis software, while using the same cortical and whole cerebellum masks from GAAIN. Our software yielded results that differed by 1-2% from the standard SPM approach. A correction was implemented to adjust for this small discrepancy. **Conclusions:** Both 11C-PiB and 18F-NAV4694 results can now be calculated in the common language of Centiloids by centers across the world using the data supplied through the GAAIN website. This is an important step towards better use of the clinical and research potential of Aβ imaging.

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**Theme:** 1. Diagnosis/Assessment  
**Poster number:** P018

Generating continuous and categorical measures from tau imaging studies with 18F-AV1451, 18F-THK5317 and 18F-THK5351: The Tau MeTeR scale

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**Objectives:** It has been postulated that tau stereotypically spreads from the mesial temporal cortex (MTC) into neocortex and that tau deposition restricted to MTC might be just part of the ageing process, suggesting that both the amount and the location of these tau deposits are likely to be relevant in regards to disease staging, prognosis and progression. We implemented a stereospecific approach to generate both continuous and categorical measures that reflect tau spreading and deposition in order to make results from tau imaging studies clinically relevant and easy to interpret. **Methods:** Sixty-five participants underwent tau and Aβ imaging with 18F-AV1451 and 18F-florbetapir (58 HC, 6 MCI, 1 AD), while 79 received 18F-THK5317 or 18F-THK5351 and 18F-flutemetamol (25 HC, 19 MCI, 5 AD). Three tau-masks were constructed: Mesial-temporal (Me) comprising entorhinal cortex, hippocampus, parahippocampus and amygdala; Temporoparietal (Te) comprising inferior temporal, fusiform, supramarginal and angular gyri, posterior cingulate/precuneus, superior and inferior parietal, and lateral occipital; and Rest of neocortex (R) comprising dorsolateral & ventrolateral prefrontal, orbitofrontal, gyrus rectus, superior and middle temporal, and anterior cingulate. A threshold was established for each mask and tracer. A global SUVR was determined by averaging the SUVR of the three composite regions. Categorically, a study was deemed high when at least two of three regions showed high tracer retention. The
relationship between Aβ and tau was also explored. Results: A categorical classification using global cut-offs of 1.35 SUVR (18F-AV1451), 1.25 SUVR (18F-THK5317) and 1.85 SUVR (18F-THK5351), yielded similar classification than obtained through the three composites. While the sample size is still low, both tracers showed that MTC was high irrespective of Aβ levels, in contrast with the other two neocortical regions where high cortical tau was mainly associated with high Aβ. Conclusion: We have developed a scale that accounts for the particularities of tau deposition, yielding both continuous and categorical measures of tau imaging studies.

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Theme: 1. Diagnosis/Assessment  
Poster number: P021

Assessing Aβ & tau pathology in Vietnam war veterans with chronic Post-Traumatic Stress Disorder

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Objectives: Epidemiological studies indicate a nearly twofold increase in risk of dementia associated with Post Traumatic Stress Disorder (PTSD) in military cohorts, however mechanisms contributing to this relationship are poorly understood. The aim of this study was to investigate if Vietnam war veterans without mild cognitive impairment or dementia, but with chronic combat related PTSD show evidence of Alzheimer’s disease (AD) pathological markers, as assessed by amyloid and tau imaging with PET. Methods: Sixty-seven male participants -30 veterans with chronic PTSD (aged 67.9±2.6 years) and 37 controls (aged 74.3±8.3 years)- underwent both tau and amyloid PET imaging scans with 18F-AV1451 and 18F-florbetaben or 18F-flutemetamol, respectively. While 18F-AV1451 SUVR was calculated using the cerebellar cortex as reference region, the whole cerebellum and the pons were used as reference regions for 18F-florbetaben and 18F-flutemetamol, respectively. While 18F-AV1451 SUVR was calculated using the cerebellar cortex as reference region, the whole cerebellum and the pons were used as reference regions for 18F-florbetaben and 18F-flutemetamol, respectively. Results: Despite the PTSD cohort being significantly younger than the controls, there was a significant difference in the age-corrected 18F-AV1451 retention between the PTSD and control groups in the mesial temporal cortex (1.19±0.12 vs. 1.12±0.17, p=0.03), temporoparietal (1.21±0.12 vs. 1.13±0.13, p=0.01) and frontotemporal (1.14±0.12 vs. 1.06±0.13, p=0.012) regions. There was no significant difference in amyloid burden between the groups conclusions: Our preliminary findings suggest that chronic PTSD might be associated with higher neocortical tau deposition later in life. Further work is required to determine if chronic PTSD itself, or associated lifestyle factors account for this observation.

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Poster Presentation  
Theme: 1. Diagnosis/Assessment  
Poster number: P024
Revisiting, revising and refining the natural history of Aβ deposition and its effects on neurodegeneration and cognitive decline in sporadic Alzheimer’s disease

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Background: We used 72-month longitudinal data from the AIBL study to calculate the rates of Aβ deposition. Methods: 201 participants (149HC; 34MCI; 20AD) were evaluated at enrollment and every 18 months for a mean follow-up of 4.9 (range 2.5-10.6) years. Participants underwent neuropsychological examination, MRI, and a PiB-PET scan. A 1.4 SUVR (25 Centiloids-CL-) was used to discriminate high from low Aβ burdens. Irrespective of their Aβ-status, participants with a positive rates of Aβ deposition, deemed to be on the AD-pathway were used for the analyses. Results: At baseline significantly higher Aβ burdens were observed in AD (2.3±0.4 SUVR/91±26 CL) and MCI (2.0±0.7 SUVR/77±27 CL) when compared to HC (1.4±0.4 SUVR/25±7 CL). At follow-up 164 (82%) of the participants showed positive rates of Aβ accumulation. Confirming our previous findings our new assessment with a longer follow-up showed Aβ deposition spanning more than two decades, averaging 30 (CI 25-39) years to go from the levels observed in Aβ-HC (1.2±0.1 SUVR/10±1 CL) to those observed in mild AD, with rates of 0.048 -CI 0.041-0.056- SUVR/yr (3.8 -CI 3.2-4.4- CL/yr), between the threshold of PiB abnormality to the levels observed in AD. As AD progresses, the rate of Aβ deposition slows, approaching a plateau. There were no significant associations between the rates of Aβ deposition and the rates of hippocampal or grey matter atrophy. There was a significant association between rates of Aβ deposition and rates of episodic memory decline only in Aβ+HC accumulators (R2=0.20; p=0.04), association that disappeared after adjusting for baseline Aβ-burden. Conclusions: Our new assessment with a longer follow-up confirmed our previous findings that Aβ deposition is a slow and protracted process, likely to extend for more than two decades. Despite this wide time-window, the effects of Aβ accumulation over cognition seem to be limited to the early stages of accumulation suggesting that anti-Aβ therapeutic interventions aimed at modifying the course of AD, should be administered at preclinical stages of the disease.

Computational Analysis of PET by AIBL (CapAIBL): A cloud-based processing pipeline for the quantification of PET images

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Objectives: Evaluate a recently developed cloud-based analysis method for Positron Emission Tomography (PET) on a database of C11 and F18 β-Amyloid (Aβ) tracers as well as F18-FDG. Methods: The Computational Analysis of PET by AIBL (CapAIBL) is a publically available cloud-based platform (https://capaibl-milxcloud.csiro.au) where PET images are spatially normalised to a standard template using an adaptive atlas approach [1]. SUVR normalised and quantified. Four hundred and fifty four participants underwent MRI and PET scans with 18F-Flutemetamol (N=180), 11C-PiB (N=381), 18F-Florbetapir (N=171), 18F-Florbetaben (N=148), 18F-NAV4694 (N=47) and 18F-FDG (N=34). Each PET image was analysed using CapAIBL. The SUVR normalisation was performed using each tracer's reference region (Cerebellum GM for 11C-PIB, 18F-Florbetaben, 18F-NAV4694 and 18F-FDG, Pons for 18F-Flutemetamol and Whole Cerebellum for 18F-Florbetapir). For validation purposes, the images were also quantified using their corresponding MR. The error in neocortical SUVR between CapAIBL PET-only approach and the MR-based quantification was assessed using the coefficient of determination (R2) and mean absolute percentage error (MAPE). Results: The error in neocortical SUVR quantification was lower than 5% and was comparable across tracers. Conclusions: As the use of PET Aβ tracer becomes more prevalent, there is going to be a greater need for standardised methods to analyse and quantify these images. CapAIBL can accurately quantify Aβ PET images without MR, with a similar degree of accuracy across tracers.

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Poster Presentation
Theme: 1. Diagnosis/Assessment
Poster number: P029

Differential Diagnosis in Alzheimer's Disease and Dementia with Lewy Bodies via VMAT2 and Amyloid Imaging

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Background: The noninvasive evaluation of nigrostriatal dopaminergic integrity by PET can provide useful information for the differential diagnosis between dementia with Lewy bodies (DLB) and Alzheimer's disease (AD). Objectives: To evaluate the diagnostic potential of imaging striatal monoaminergic terminal integrity with the novel vesicula monoamine transporter type 2 (VMAT2) radioligand [18 F]AV-133 and PET to distinguish DLB from AD. Methods: Fifty participants [9 DLB, 11 AD, 20 Parkinson's disease (PD) and 10 healthy age-matched control subjects (HC)] underwent [18F]AV-133 PET studies. Additionally, 20 participants underwent amyloid imaging PET scans with either [11C]PiB or [18F]florbetaben. VMAT2 density was calculated through normalized tissue uptake value ratios (RT) at 120-140 min after injection using the primary visual or the cerebellar cortex as reference region. Comparison of the RT for [18F]AV-133 was done between the different clinical diagnostic groups. Results: Significantly lower striatal VMAT2 densities were observed in DLB and PD when compared to AD and HC, especially in the posterior putamen. In contrast to PD and DLB, no reductions were observed in AD patients when compared to HC. Conclusions: [18F]AV-133 allows assessment of nigrostriatal degeneration in Lewy body diseases. In contrast to amyloid imaging, VMAT2 imaging with [18F]AV-133 can robustly detect reductions of dopaminergic nigrostriatal afferents in DLB patients, assisting in the differential diagnosis from AD.
Web-Based PET and MR quantification using CurAIBL

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Anatomical MR imaging for Alzheimer’s disease looks at patterns of atrophy in key structures associated with the disease. Visual inspection is typically limited to the identification of gross changes, in the inferior temporal lobe and ventricles. Using the same cloud computing platform as CapAIBL for PET quantification, a new atrophy report is publically available. CurAIBL (Computational qUantification of mRi fr om AIBL) enhances visual inspection with z-score map of cortical thickness, normative graphs, and tables with volume of key structures in relation to a reference population. MR images are first rigidly registered to the MNI average brain, and segmented into gray and white matter and CSF using Expectation Maximisation Segmentation algorithm. The images are then parcellated using the 20 most similar atlases, selected from a database of 843 images. Hippocampus volume is extracted using the Harmonized Protocol for Hippocampal Volumetry. Cortical GM and hippocampal volumes are reported on a graph with confidence interval for an aged-matched normal population. Cortical thickness is computed and mapped to a normalised template. Z-score map of cortical thickness is computed and reported as a 3D rendering. Key volumes, graphs and mesh rendering are display on a pdf report which is emailed to the user at the end of the procedure. If a PET image is also provided, the CapAIBL platform is used to analyse the PET using the extracted MR information, and a combined PET-MR analysis report is sent. CurAIBL is a tool to aid visual interpretation of MR images. Volumes of key structures in relation to a normal population can provide early warnings of abnormal atrophy. Patterns of cortical thinning can be also used as a tool for differential diagnosis (such as FTD). CurAIBL provides an efficient clinical inspection tool for MR imaging and when used in conjunction to CapAIBL for PET quantification such as Amyloid-beta or Tau markers, it offers a comprehensive imaging assessment of Alzheimer’s disease.
Introduction: AIBL commenced in late 2006 with 1,100 participants and included beta-amyloid imaging and MRI in 30%. Assessments have been repeated every 18 months and imaging was expanded and participants replenished so that presently AIBL has baseline amyloid scans on 1,070 participants and 3 or more time point imaging in over 300. Method: Participants were recruited from advertising and Memory Clinics, were aged 60+ with no history of stroke or serious medical disease and normal (HC) (60%), MCI (20%) and AD (20%). Cognitive assessment, blood analysis and imaging were performed at each time point. Follow up of the initial cohort to 90 months is almost complete. Results: At baseline the prevalence of positive amyloid scan in HC rises steeply from 10% in persons aged 60-69 to over 50% if aged >80. The prevalence is strongly influenced by APOE genotype. Amyloid burden is higher in those with untreated hypertension and diabetes. Amyloid burden increases at 2-3% per year in those accumulating and this process takes 30 years to reach the typical level of mild AD. Rate of cognitive decline and clinical progression (HC:MCI:AD) is strongly related to the presence of amyloid but in HC this is moderated by genetic factors including APOE and BDNF alleles. Clinical progression is faster when hippocampal atrophy or episodic memory impairment is also present. Discussion and Conclusion: AIBL discoveries have contributed substantially to new guidelines for earlier diagnosis of Alzheimers disease and to the implementation of dementia prevention trials in those with asymptomatic or prodromal Alzheimers pathology. The large cohorts with and without AD pathology provide gold standard cohorts invaluable to researchers in lifestyle, blood and CSF biomarker, genetic and other discovery areas. The addition of tau imaging and in-depth genetic and epigenetic characterization to AIBL promises further novel and important discovery.

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Poster Presentation
Theme: 1. Diagnosis/Assessment
Poster number: P037

The effect of Aβ deposition, neurodegeneration and their interactions on the cognitive trajectories of healthy older adults in the AIBL cohort

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Background: Beta-amyloid deposition and/or neurodegeneration have been documented in a considerable number of reports about cognitively unimpaired healthy elderly individuals, however, it is still unclear what are the long-term cognitive consequences of their presence, and if they have an independent or synergistic effect on cognition. The objective of the study was to characterise the clinical and cognitive trajectories of healthy elderly controls
using both a two-imaging (AD pathology and neurodegeneration) marker construct. **Materials and Methods:** Five hundred and seventy-three (573) cognitively unimpaired individuals (73·1±6·2 years; 58% female) from the Australian Imaging, Biomarker and Lifestyle (AIBL) study were assessed. Beta-amyloid status (A) was determined with either PiB, flutemetamol, or florbetapir; while neurodegeneration (N) was established using hippocampal volume. For the two-marker construct individuals were categorised as either A-N-, A+N-, A+N+, or suspected non-Alzheimer disease pathophysiology (A-N+, SNAP). Domain-specific and global cognitive composite scores were assessed longitudinally over six years using linear mixed effect models. **Results:** Nine percent of HC were classified as A+N+, 15% as A+N-, 54% as A-N-, and 22% as SNAP. APOE Æ 4 carriage was more frequent in A+N+ (54%) and A+N- (48%) than in A-N- (21%) and SNAP (18%). Generally, no significant differences in baseline cognitive scores were observed for A+N- and A+N+ compared to A-N-, however, they presented significantly faster cognitive decline than A-N-. The A-N- and SNAP groups did not show significant decline over time, although SNAP was sometimes associated with lower baseline cognitive scores. **Conclusions:** Increasing marker abnormality was reflected in faster cognitive decline, indicating a synergistic effect. Completely distinct cognitive trajectories were observed in those with AD and non-AD pathology, likely suggesting different underlying pathophysiological mechanisms.

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Poster Presentation
**Theme:** 1. Diagnosis/Assessment
**Poster number:** P015

**Recruiting a preclinical cohort with AlP positive subjects**

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**Introduction:** As therapy strategies and clinical studies focus on preclinical individuals, there is a need to recruit large cohorts of asymptomatic elderlies with high cortical AlP deposition. PET scanning can image in vivo AlP deposition in the brain, but with high cost. This study suggests several strategies to minimize the cost of recruiting healthy elderlies as confirmed by PET scan. **Methods:** Subjects older than 60 from the AIBL study were used. Amyloid PET scan (AU$2000) was considered the ground truth to identify individuals with abnormally high AlP brain level (sensitivity 100% and specificity 100%). Three options were considered. 1) Scan each recruited subjects with PET until reaching the targeted cohort size (100); 2) perform prior to PET scan a blood test (AU$100) to screen APOE Æ 4 (E4) allele carriers (sensitivity of 60.4% and specificity of 79.5%); 3) perform a more expensive blood test (AU$200) to screen in addition to E4 allele carrier, subjects positive for a blood panel (Abeta1-42, CXCL-13, IgM-1, IL17, PYY & VCAM-1; sensitivity of 50.5% and specificity of 96.8%). The total cost of recruitment was compared between the three options. **Results:** Prevalence of high AlP was 39%. For a final cohort of 100 individuals, the number of subjects to recruit and the total cost for the three strategies were respectively: 256 and AU$564,000 (~US$395,000), 425 and AU$348,000 (~US$244,000), and 508 subjects for a total cost of AU$321,000 (~$225,000). **Conclusions:** Close to 50% cost reduction in recruiting individuals older than 60
with confirmed Aβ by PET could be achieved by selecting subjects carrying at least one E4 allele. A further 8% cost saving could be achieved by adding extra blood exams.

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Poster Presentation
**Theme:** 1. Diagnosis/Assessment
**Poster number:** P020

**Age of Onset for different bio-markers of Alzheimer's disease**

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**Background:** Abnormal levels of Aβ deposition in the brain are reached prior to evidence of neurodegeneration and cognitive impairment. An understanding of the age of onset of these different markers, and what factors affect them, is of clinical interest for the understanding of underlying mechanisms that may delay onset or stop progression of the disease. **Methods:** The ages at which 317 AIBL participants reached abnormal levels on six markers of disease (namely Aβ deposition (SUVR≥1.5), Hippocampus Volume (<5.87cm3), AIBL-PACC4 (≤-6 [four summed z-scores, therefore 4*1.5SD]), Episodic Memory, Executive Function and Language Composites5 (≤-1.5SD)) were calculated using models of disease progression. Cox proportional hazards models of survival and Kaplan-Meier plots were employed to determine if the six different markers had varying ages of onset of abnormal levels. **Results:** The Kaplan-Meier plots suggested that the ordering or age of onset for the six markers was Aβ deposition/Hippocampus Volume/AIBL-PACC followed by Episodic Memory, Executive Function and Language. However, in Æ4 carriers it was clear that Aβ deposition had the earliest onset. A 40% risk of having abnormal Aβ deposition occurred 22 years earlier in Æ4 vs non-Æ4 carriers. **Conclusions:** The order of onset of abnormal levels of disease markers as measured here appears to align with other findings previously reported in the literature.

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Poster Presentation
**Theme:** 2. Care/Living with Dementia
**Poster number:** P078

**Study protocol: translating and implementing a support and education based intervention to improve driving cessation outcomes for people with dementia**

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The issues around driving and dementia have emotional, social, legal, and ethical ramifications. Health professionals acknowledge the difficulties of managing driving cessation with people with dementia and their families. Dementia has a profound effect on capacity for driving, although a diagnosis of dementia does not immediately preclude someone from safe driving, at some stage they will have to stop. Without intensive practical and emotional support to plan for, and eventually cease driving, people with dementia are at risk for depression, reduced community mobility, social isolation, unsafe and unlicensed driving, and injury or loss of life. Despite the concerns for community safety and the safety and wellbeing of people with dementia, no theory-driven driving cessation interventions are in routine clinical practice in Australia. The UQDRIVE-People-with-Dementia intervention is a theoretically driven, comprehensive support and education based driving cessation intervention for people with dementia and their families that it is individualised according to geographic location and to lifestyle goals of participants. The intervention includes seven modules covering education and practical support. It will be embedded in primary care and community settings to optimise the timing of delivery for people with dementia and their families. A pragmatic cluster randomised controlled trial with regions as the unit of randomisation, and a wait-list control will be undertaken. An iterative mixed methods approach will be applied to understanding outcomes, including wellbeing and community mobility. Community mobility will be measured objectively with novel Smartphone GPS technology. A process analysis will be conducted to understand the facilitators and barriers to intervention delivery in primary care settings in metropolitan and regional areas.

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Poster Presentation
Theme: 1. Diagnosis/Assessment
Poster number: P016

The Surface Functionalization of Upconversion Nanocrystals for Blood-Brain Barrier Crossing and Potential Theranostics

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Introduction: Blood-Brain Barrier (BBB), a crucial physiological structure between blood and brain tissues, strictly regulates the movement of cells, molecules and ions between the circulatory system and brain to protect the brain, heavily limiting the delivery of drug to the brain circulation. [1] This BBB remains the therapy of central neuron diseases (e.g. amyotrophic lateral sclerosis, Alzheimer’s disease, Parkinson’s disease, brain tumour) a formidable challenge although tremendous efforts to develop effective strategies for neurological disorders treatment have been made in the past decades [2]. Lanthanide doped upconversion nanoparticles (UCNPs) show their unique advantages to cross BBB, such as fine tuning shape/size/surfaces, background free, photo stable, and high deep tissue penetration, [3] which will benefit to investigate the underlying mechanisms of how nanoparticles cross the BBB. Method, Result & Discussions: In this study, we investigate how nanoparticles with different surfaces effect on BBB penetration using UCNPs as model. We synthesize UCNPs with varieties of function groups, and then identify the preferable surfaces of UCNPs that can efficiently pass the adhesive BBB through testing in cell and animal model.

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Poster Presentation
Theme: 1. Diagnosis/Assessment
Poster number: P017

Development and validation of the first culturally based quality of life tool for Aboriginal Australians living with dementia or cognitive impairment

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Introduction: Dementia is highly prevalent in Aboriginal Australians, with Aboriginal caregivers reporting that major improvements are required to the quality and cultural security of dementia care. Enhancing quality of life (QoL) is the central goal of residential and community care services for their clients with dementia. A valid QoL tool enables person-specified areas of need to be identified over a number of domains, and treatment and care strategies to be planned and evaluated accordingly. Despite the need there is no valid QoL measure for Aboriginal Australians living with dementia or other forms of cognitive impairment. This project aims to develop such a tool. Method: A mixed methods approach will be applied in which Aboriginal consultation and participation is at the forefront. Aboriginal Australians living in residential care or accessing community care in Perth will be invited to take part in focus groups and in-depth interviews for tool development. This will be followed by reliability and validity testing of the QoL tool with older Aboriginal Australians with cognitive impairment (including dementia) living in Perth and Melbourne. Result and Discussions: Networking has begun with different stakeholders for their participation in a steering committee. Derbarl Yerrigan Aboriginal Health Service in Perth has recently approved this project. An Aboriginal research officer is to be employed towards the end of this year. The resulting QoL tool will be disseminated with training packages for inclusion into policy and procedures of service providers and interested organisations. Data on the factors affecting the QoL of this population will be provided to leading authorities and policy makers. Conclusion: A quality of life tool can be used by service providers to evaluate the effectiveness of dementia treatment, care and support, with enhancing the quality of life of elders being an area of genuine need for Aboriginal Australians.

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Poster Presentation
Theme: 1. Diagnosis/Assessment
Poster number: P019

An investigation into the neural substrates of the cognitive deficits in Mild Cognitive Impairment, and the mechanisms of action of a novel treatment
Introduction: Mild Cognitive Impairment (MCI) causes a slight but noticeable decline in cognitive abilities, and is associated with an increased risk of developing dementia. Currently, there is no treatment for MCI, and Alzheimer’s disease medications do not satisfactorily consider its diverse underlying pathophysiology. Furthering our understanding of the neural mechanisms underpinning the cognitive deficits present in MCI is vital for the conceptualisation of the condition’s underlying pathophysiology, and the development of targeted treatments. This NHMRC-ARC Dementia Research Development Fellowship project aims to: 1.) further our understanding of MCI pathophysiology, and 2.) determine the mechanisms of action of a potential multi-target treatment for MCI. Two studies will be conducted to assess the two central aims of this project. Method and Proposed Outcomes: Study 1 will assess the neural correlates of episodic memory, executive function, and perceptual reasoning in people with MCI, mild Alzheimer’s disease, and healthy age-matched controls. This study will triangulate a range of physiological measures and biomarkers with cognitive function including electroencephalography (EEG), functional magnetic resonance imaging (fMRI), genetic risk factors, and plasma inflammatory markers. This work will look for changes in the function and structure of neural networks with the aims of elucidating individuals who may have an increased risk of developing Alzheimer’s disease, and detecting novel pathways for possible treatments. Building on the findings from Study 1, Study 2 will investigate the mechanisms of action of Sailuotong (SLT) in people living with MCI with a 12 week randomised placebo-controlled pilot trial. SLT is a three-herb formula consisting of standardised extracts from Ginkgo biloba, Panax ginseng, and Crocus sativus (saffron), and has been shown to improve cognitive function in healthy adults and people with vascular dementia. This work will determine whether SLT may be a viable treatment for MCI.

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Poster Presentation
Theme: 1. Diagnosis/Assessment
Poster number: P022

Cognitive Assessment Strategies for Clinical Trials in Dementia

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Introduction: Dementing disorders are defined by cognitive impairment linked to functional disability, thus cognitive assessment forms the cornerstone of the neurodegenerative diseases. No guidelines have emerged, however, to support the selection of rational, evidence-based approach to cognitive assessment in clinical trials. Furthermore, even in major dementia trials, cognitive outcomes with severely limited psychometric properties and sensitivity are often used, thus limiting the detectability of treatment effects. Method: As an illustrative example, we describe the Australia-led development of a sensitive cognitive battery and composite score for Huntington’s disease clinical trials. The method used expert opinion, systematic literature review, and a clinical-trial like study of 250 participants at 20 sites internationally to determine practice effects, psychometric properties, feasibility and
tolerability for participants and site staff, and pragmatic issues such as time of testing, and the development of composite measures that can be used as primary outcomes. **Results and Discussion:** The HD-CAB is now being administered in five commercially-sponsored Huntington's clinical trials across a total of nearly 100 clinical sites and by more than 120 trained cognitive examiners using methods that meet GAMP-5 and regulatory standards. Quality controls procedures show that the data are high quality, with few missing observations, and that standardised methods can be implemented by examiners following rigorous training with good results. Trial results will begin to emerge in 2017, but the processes we have put into place provide a framework for developing evidence-based cognitive assessment methods and implementing them using industry standards and across many research sites. **Conclusion:** The rational development of cognitive battery, along with psychometric considerations for the construction of cognitive composites that can be used as primary outcomes in clinical trials, has the potential to accelerate the progress and quality of clinical trials in neurodegenerative diseases, thus increasing the efficiency of progress toward treatments.

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**Poster Presentation**  
**Theme:** 2. Care/Living with Dementia  
**Poster number:** P077

**Therapeutic signing and music interventions to improve wellbeing and connection between community dwelling people with dementia and their primary caregivers**

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In spite of the prevalence of dementia in the Australian population and its significant negative effect on people with dementia (PWD) and their caregivers (CG), there is limited evidence-based research to support the use of music interventions for PWD/CG dyads living at home. Areas of the brain responsible for music processing are retained until late in the trajectory of dementia. For the PWD this capacity to respond to music activities facilitates reminiscence and successful social engagement. As a consequence, CGs can relate with their loved one in meaningful and satisfying ways. Music interventions also provide a non-pharmacological alternative to assist with management of challenging dementia symptoms (agitation, anxiety, and apathy), offering CGs strategies to use in the home. This project aims to investigate effects of community signing groups and home-based music interventions for PWD/CG dyads on:  
1. anxiety, quality of life, agitation, apathy and cognitive function in PWD.  
2. life satisfaction, carer satisfaction, flourishing, and depression in CGs.  
3. quality of the PWD/CG relationship.

This therapeutic music program has the potential to: 1) support a sustained and fulfilling relationship between the PWD and their primary caregiver; 2) alleviate psychosocial and emotional difficulties that are commonly experienced by PWDs and their CGs; and 3) assist PWD and their CGs to remain together in the family home for as long as possible. As people will be recruited with early-mid stage dementia, supportive strategies will be established before the disease progresses, thereby building on knowledge that music memory is retained into later stages of the disease. These outcomes are recognised as important ethical and economic considerations in dementia care. Supporting the CG to manage dementia symptoms and care for their loved one at home may improve quality of life for the PWD/CG.
dyad while also significantly reducing healthcare costs for society.

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Poster Presentation  
Theme: 1. Diagnosis/Assessment  
Poster number: P023

**Dual and multiple proteinopathies in neurodegenerative dementias – risk factors, prognostic indicators and clinical ramifications**

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**Background:** ‘Dementia’ encompasses a number of different clinical syndromes, each subserved by distinct histopathological signatures of insoluble protein aggregates and spread. Converging evidence now indicates the overlapping deposition of pathologic proteins in at least 50% of dementia syndromes. The deposition of dual/multiple proteinopathies impacts on dementia phenotype and severity, and has important clinical implications for diagnosis and development of substrate-specific interventions, particularly since targeting and alleviating one proteinopathy without the other is unlikely to successfully ameliorate dementia in affected patients. However, the prevalence, clinical ramifications and associated risk factors of dual/multiple proteinopathies remains largely unknown with the 2014 updated consensus criteria for Alzheimer's disease stating that the clinical phenotype of mixed pathologies is uncertain. **Methods:** The Sydney Brain Bank (SBB) holds 643 longitudinally-studied patients with a pathologically-confirmed clinical dementia syndrome and 90 cognitively normal individuals with no significant neuropathology. The present study will assess all dementia cases categorized by disease duration into three groups of short, average and prolonged disease to determine: (1) The prevalence of dual/multiple proteinopathies in the main neurodegenerative clinical dementia syndromes, which proteins most commonly co-occur and the predilection sites of these; (2) When during the disease course do multiple proteins become pathological and/or the clinical relevance of dual/multiple proteinopathies; (3) If the presence of dual/multiple proteinopathies in the main neurodegenerative clinical dementia syndromes change the clinical course; (4) If there are separate risk factors for the presence of dual/multiple proteinopathies in the main neurodegenerative clinical dementia syndromes. **Project Significance:** By investigating the prevalence, risk factors and clinical ramifications of dual/multiple proteinopathies in patients with dementia, this study will provide critical information to aid the clinical recognition of patients at-risk, and the identification of substrate-specific targets for the development of therapeutic interventions.

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Theme: 2. Care/Living With Dementia  
Poster number: P044

**Understanding and preventing physical and cognitive decline and falls in older people with dementia**
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Introduction: More than 60% of community-dwelling older people with dementia (CDWD) fall annually and approximately 40% have multiple falls. There is limited evidence that falls can be prevented in CDWD. There is also a lack of evidence in relation to preventing cognitive and physical decline in this population, particularly with home-based programs. This fellowships’ research program aims to promote independence, prevent functional decline/falls, with the overarching goal of improving quality of life for CDWD. Specifically it will a) explore the association between physical and cognitive performance, and falls in CDWD, b) develop and pilot novel approaches to fall prevention in CDWD, c) assess the feasibility of adapting new and emerging technologies in falls prevention to the needs of CDWD and d) implement and evaluate the impact of assessing functional cognition in Aged Care Rehabilitation. Methods: Prospective cohort study (Study 1; n=177, 12-months), uncontrolled exercise intervention study (Study 2; n=42, 6-months), pilot feasibility studies, pilot randomised control trial (cognitive training), translational/implementation study (assessing functional cognition). Results: Study 1: Poorer executive function increases the risk of multiple falls in CDWD. Taking ≥6 medications, reaction time and balance were mediators of the relationship between executive function and falls. Study 2: A tailored, home-based exercise program improved balance, concern about falls and planned physical activity, but resulted in worse knee extension strength and no change in depression scores in CDWD. Discussion and conclusions: Furthering our understanding of fall risk and decline in CDWD will enable us to continue working towards developing interventions that target the identified amenable factors and reduce fall risk, fall injury and decline. Successful strategies that maintain independence, optimise physical, functional and cognitive performance and reduce falls are desperately needed for CDWD. The economic benefit and potential positive impact for older people with dementia and their carers is substantial.

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Poster Presentation
Theme: 2. Care/Living With Dementia
Poster number: P045

Improving dementia care by creating research connections that encourage increased collaboration and translation of research into real outcomes for people with dementia

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Introduction: Improving care for people with dementia remains critical for the 353,800 people living with dementia in Australia. Through the Partnership for Better Health Initiative, the National Health and Medical Research Council (NHMRC) recognised bringing together clinicians, consumers, researchers and decision makers to work on priority areas as essential for translating research into health and health systems improvement. This poster reports on the
extent to which this model, adopted by the NHMRC funded Partnership Centre Dealing with Cognitive and Related Functional Decline in Older People (CDPC), is developing a growing collaborative environment focused on improving care for people living with dementia.

Method: The CDPC’s knowledge-to-action approach brings together consumers, clinicians, academic, and industry partners to: support research and implementation of tested models of care; synthesise and disseminate existing research; conduct collaborative new research; and build capacity to translate research into practice. Mixed method longitudinal analysis of CDPC activities is conducted through ongoing collection of data, measuring how the CDPC network increases connectedness resulting in increased translation of research into practice. Analysis draws together interviews and surveys with CDPC network members and CDPC quarterly monitoring data.

Results & Discussions: Ongoing social network analysis shows new collaborative ties among increased CDPC membership with a measurable shift from within sector collaboration to cross-sector collaboration. At the same time there has been considerable increase in numbers of organisations implementing CDPC research-supported system change projects. A shift in number of non-academic based researchers, including consumers themselves, involved in the CDPC has also occurred, as has the increased dissemination of research findings.

Conclusion: The CDPC is successfully developing collaborative relationships between clinicians, consumers, researchers and decision makers. Continued growth of these partnerships will enhance research translation into best practice care for people living with dementia in Australia.

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Poster number: P046

Promoting age friendly communities preferences for inter-generational respite care services for older people with cognitive decline and children residing in the community: preliminary results

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Introduction: For long term sustainability of respite programs and to improve outcomes for clients, it is important that service models reflect an understanding of consumers’ preferences and needs rather than clinical viewpoints alone. Preferable respite services may include Intergenerational care (InGen) that encourage the care of children and older people in a shared setting for mutual benefits. As part of exploring the acceptability of integrated models, an economic feasibility study provides analytical rigour to inform stakeholder decision-making regarding the introduction of new respite services. This project explored the initial phase of a feasibility study that involved: 1) identification of feasible models of InGen services in the Australian setting; 2) collection of information relating to preferences and willingness to pay for new types of InGen services.

Method: Using the Delphi process, a panel of experts developed and identified feasible InGen. The design of the survey tool that was used to elicit preferences and estimate the demand for InGen models involved a systematic literature review, interviews and pilot studies. Based on the Contingent Evaluation Method, individuals were asked about their preferences and willingness to pay for status quo services versus innovative InGen services.

Result: The Delphi process narrowed the feasible InGen services in the Australian setting to two models, i.e. shared campus and visiting campus.
Preliminary results from the survey indicate that respondents do have a preference for InGen services, however, their additional willingness to pay compared to the status quo is low. **Discussion and Conclusion:** Future research will identify the economic drivers of demand and the price levels consumers are willing to pay for InGen services. Once demand for these services is understood, information regarding the supply side will be collected and analysed. The demand and supply analyses are then brought together to identify probability of outcomes under various scenarios.

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**Poster number:** P025

**Prevalence of dementia among Australian women aged over 70: application of capture-recapture methodology on data from multiple sources**

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**Introduction:** Accurate estimates of dementia are needed to plan for health service needs. This study used linked data to estimate the prevalence of dementia in Australian women.  
**Methods:** There were 12,432 women born between 1921 and 1926 who completed an Australian Longitudinal Study on Women's Health survey. These data were linked to records of aged care assessments and services, hospital admissions, drug prescriptions, and death certificates, to estimate the prevalence of dementia. Capture-recapture methods were used to estimate the number women with dementia not identified from any of the available sources.  
**Results:** Over 16 years follow-up, 20.4% (95% CI (19.7%, 21.1%)) of women were recorded as having dementia from at least one data-source. Using capture-recapture methods, this estimate increased to 26.2% (95% CI (25.5%, 27.0%)).  
**Discussions and Conclusion:** This analysis demonstrates the importance of using multiple data sources, and capture-recapture methods, when estimating the total number of women with dementia.

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**Supporting healthy ageing in postmenopausal women with resveratrol: study protocol**

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**Introduction:** Postmenopausal women suffer disproportionately from dementia, which we hypothesise may be partly attributable to loss of cerebrovascular benefits of estrogen. Our previous research has demonstrated the efficacy and safety of resveratrol (an ingredient found in berries and grapes) on vasodilator function and cognition. We now aim to test whether regular supplementation with resveratrol (a phytoestrogen) can improve cerebral perfusion, cognition, mood, physical function, bone health and well-being in...
Methods: In a randomised, double-blind, placebo-controlled, crossover intervention, 170 women aged 45-85 years who are at least 12 months postmenopausal will take 75mg of resveratrol or placebo, twice daily, each for 12 months. They will undertake the NIH Toolbox battery of cognitive tests that covers domains of attention, executive function, processing speed, episodic and working memory. The primary outcome will be the overall cognitive performance, which is the sum of Z-scores of all tests. Transcranial Doppler (TCD) ultrasound will be used to record basal blood flow velocity and intracranial stiffness in the middle cerebral artery. Cerebrovascular responsiveness to hypercapnia, cognitive testing and photic stimuli using TCD will assess the ability of the cerebral vasculature to increase delivery of blood in response to demands. Other outcomes will include measures that are relevant to the overall well-being and quality of life of postmenopausal women. They include mood, physical function (i.e. balance and grip strength, dexterity), pain perception, menopausal symptoms, DEXA assessment of bone mineral density and adiposity, blood lipids, glucose, insulin, HbA1C, selected inflammatory biomarkers, osteocalcin, estradiol and follicle-stimulating hormone levels. Conclusion: This will be the first study to explore whether postmenopausal deficits in cognition, mood and self-perception of well-being are modifiable by enhancing cerebral perfusion. Findings will also provide the first clinical evidence if early intervention (<10 years since onset of menopause) can attenuate the decline in cognition with aging.

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Poster number: P085

Vascular Cognitive Risk Score: quantifying the vascular burden in Alzheimer’s Disease

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Understanding the variable underlying pathophysiological substrate of the dementias is of great importance as this represents a key target for putative therapies. The two most common pathologies in dementia, Alzheimer’s disease and cerebrovascular disease, are often coexistent in an individual patient to a variable degree, and hence treatment approaches should account for this balance. Characterising the contribution of each pathology in a single patient represents a critical step towards developing more effective treatments. This research project will explore multiple patterns of cerebrovascular disease using MRI studies in the Australian Imaging, Biomarker and Lifestyle Study of Ageing (AIBL), with the aim of developing a numeric score, the vascular cognitive risk score, reflecting the contribution of cerebrovascular disease to cognitive impairment in this cohort. Furthermore, concurrent assessment of PiB PET data will allow the estimation of the relative contribution of both Alzheimer pathology and vascular pathology in individual patients. We will aim to validate the findings in external cohorts and using a new prospective cohort of patients with vascular cognitive impairment and mixed dementia. A quantitative assessment of the relative contribution of vascular disease to a given patient’s cognitive decline represents a major paradigm shift in diagnosis and prognosis, as well as informing future therapeutic trials.
National Roll Out and Evaluation of the Dementia Care in Hospitals Program

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Background: The Dementia Care in Hospitals Program (DCHP) is an all-of-hospital training and education program to improve awareness of, and communication with, patients with cognitive impairment (CI) in the acute sector. The DCHP was developed at Ballarat Health Services and has been implemented in twenty-five hospitals across Victoria. Department of Health funding is supporting a national rollout with a detailed evaluation by Deakin University.

Method: A stepped-wedge methodology will be implemented in leadership hospitals in Adelaide, Canberra, Perth and Hobart. The target population on participating wards is all acute admissions aged over 65 and found to have CI using a validated assessment tool. The primary outcome is the change in the rate of Adverse Events experienced by participants with CI at invention compared to baseline. Sub-analyses will adjust for each adverse event using a Generalised Linear Model. The impact of the DCHP on patient quality of life, hospital length of stay and costs, carer satisfaction, staff knowledge and change in practice will also be evaluated.

Results & Discussion: Three of the four sites have developed cognition pathways, completed baseline, and commenced intervention. Introducing universal screening for over-65s has proven challenging and has impacted on overall participant numbers. The pooled prevalence of CI at baseline is 37%. Screening rates vary across sites due to reluctance to change existing hospital processes.

Conclusion: Translational research in the complex environment of acute hospitals presents constant challenges when research requirements must be balanced against everyday needs and external environmental factors. Nevertheless, this project demonstrates that the DCHP can be implemented nationally in regional and metropolitan settings. Environmental factors, such as implementation of revised National Safety and Quality Health Service (NSQHS) standards and variation in the use of CI tools, require further investigation. A longer-term evaluation to identify the determinants of maintenance and sustainability is recommended.
Introduction: Midlife hypertension is associated with a significantly higher risk of both AD-dementia and dementia due to cerebrovascular disease, and antihypertensive treatment is associated with better cognitive outcomes in clinical trials. Hypertension has been associated with cross-sectional Aβ PET imaging measures, however whether antihypertensive treatment influences the longitudinal accumulation of Aβ is not known. We used Aβ PET imaging to determine whether treatment of hypertension influenced accumulation of Aβ over six years’ follow-up. Methods: 140 cognitively-normal participants from the AIBL Study with 11C-PiB PET imaging at 18-monthly intervals over six years. Only participants with three or more PET assessments were included in analysis. Linear mixed models regression was performed for Aβ SUVR (dependent variable) and Baseline Hypertension Status (Normotensive/Treated Hypertension/Untreated Hypertension), Time (and their two- and three-way interactions), as well as age, gender, education, APOE ε4, cholesterol, glucose, smoking and BMI. Results: Age, APOE ε4, Gender, Time, and APOE ε4 x Time were all associated with longitudinal measures of Aβ burden. There was also a significant difference in change in Aβ over time between individuals with normal blood pressure, untreated- and treated hypertensives, with participants with untreated high blood pressure at baseline showing greatest increases in Aβ over time. Findings remained significant after adjustment for BMI, cholesterol, glucose and smoking. Conclusion: In hypertensive cognitively-normal controls, use of antihypertensive medication was associated with less Aβ accumulation over time compared with their untreated peers. Although observational only, this study provides some in vivo biomarker evidence supporting that lifestyle risk factor modification may mitigate the pathology of AD.

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Poster number: P030

Association of Cerebrovascular disease and Alzheimer's disease Biomarkers with and Longitudinal Cognitive Decline

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Background: Cerebrovascular disease (CVD) is commonly seen to co-exist with Alzheimer’s disease. Recent studies suggest that the two pathologies may mediate distinct, additive
We examined the contribution of subclinical CVD (SCVd) and A\(\beta\) burden at baseline to risk for incident dementia over six years. **Methods:** 219 non-demented participants from the AIBL Study (169 normal cognition, 50 mild cognitive impairment) with 3-Tesla MRI and 11C-PiB PET at baseline and clinical assessments over 18-monthly intervals over six years. Persons with a history of clinical stroke were excluded from AIBL. Participants were classified as A\(\beta\)+ if PiB Neocortical SUVR ≥ 1.5 and SCVd+ if MRI evidence of stroke or significant SCVd. Incident cognitive decline and dementia were determined from clinical panel consensus following neuropsychological test performance at each timepoint. Cox proportional hazard regression was performed including A\(\beta\) and SCVd, age, APOE \(\varepsilon4\) status, gender and education as covariates, and cognitive decline, or dementia, as outcome variables. **Results:** 25% of participants were classified as having cognitive decline and 16% progressed to dementia. While both SCVd and A\(\beta\) were associated with incident dementia in univariate analyses, the interaction between SCVd and A\(\beta\) was not. Only the association with A\(\beta\) remained significant after adjustment for all covariates (Hazard ratio [for decline] 3.8, p<0.001; [for dementia] HR=7.4, p<0.001). In participants with normal cognition at baseline, risk for incident dementia at six years was only significant in those with A\(\beta\) and SCVd at baseline (HR=25.9, p=0.004). **Conclusion:** In this non-demented cohort, A\(\beta\) more strongly predicts incident cognitive decline and dementia than subclinical CVD. Subclinical CVD lowered the threshold for incident dementia in those with A\(\beta\), although SCVd alone was not sufficient to predict future dementia. These data also have implications for clinical trials in preclinical and prodromal AD.