www.csiro.au

Submission No. 034
(Dementia)
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CSIRO Submission

House of Representatives Committee Inquiry into Dementia: Early diagnosis and Intervention

May 2012

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AND

Co-Chair of the Australian Imaging Biomarkers and Lifestyle Study of Ageing (AIB)

Introduction and context of CSIRO's submission

The Commonwealth Scientific and Industrial Research Organisation (CSIRO) is pleased to be able to respond to your Inquiry into Dementia: Early diagnosis and Intervention.

As indicated in the Terms of Reference, early diagnosis and intervention has been shown to improve the quality of life for people with dementia as well as for family members and carers. CSIRO's focus with respect to early diagnosis and intervention has been one of attempting to identify the first measurable signs of the onset of the disease in humans and thereby produce a platform to soundly test both lifestyle and therapeutic approaches to slow progression toward disease or prevent its initiation. Understandably, success in this challenging area would have a significant impact on the issues that you have identified within the Terms of Reference. Accordingly, the remainder of our submission is focused on highlighting the importance of Australian research in a global context in this area and how we believe it can be accelerated into the future.

CSIRO has identified dementia, and particularly Alzheimer's disease, as an area of high priority and one in which we have devoted significant activity and resources within the Organisation's research priorities and in national collaborations as part of its national Preventative Health Flagship. This approach has been embodied in the initiation of the major cohort in Australia in Alzheimer's disease "The Australian Imaging Biomarkers and Lifestyle Study of Ageing" (AIBL). The AIBL brings together world leading researchers to improve the understanding of the causes and diagnosis of Alzheimer's disease. The longitudinal study involves over 1 000 volunteers from a cross-section of Australia's population for a 'cohort' study which will integrate expertise in neuroimaging, biomarkers, psychometrics, and lifestyle interventions.

Further details are available on www.aibl.csiro.au.

Why is early diagnosis in dementia a critical issue?

The historical success of reducing the impact of chronic disorders on individuals and society rests on two fundamental pillars - early diagnosis and intervention. The ability to detect early and reliably is an absolute prerequisite for the design and evidence based testing of intervention strategies including those relating to lifestyle choice and intervention by way of therapeutics.

Cardiovascular disease is a wonderfully successful example of the importance of the relationship between early detection and intervention and holds key lessons for research into dementia. Success in the cardiovascular area was dependent upon the generation of early knowledge that identified that elevated diastolic blood pressure (a biomarker) was a key risk measure for subsequent end organ damage, including stroke and that elevated blood lipids (biomarkers) a risk factor for subsequent development of atherosclerosis and coronary artery disease.

We need to identify with precision those biomarkers that identify the risk of developing dementia, and particularly Alzheimer's disease in a reliable way to permit intervention. Those biomarkers need to be formatted into a test for population screening identifying those at risk and requiring further clinical assessment.

Some lessons we have learnt in chronic disorders from clinical and basic sciences in the areas of early detection and intervention

It is fundamental that you cannot intervene or offer advice until you have reliable detection processes that enable efficacy to be assessed on intervention strategies. Secondly, most chronic disorders adhere to a similar pattern, ie they have a long incubation time (sometimes decades) and it is clear that intervention is more biologically effective and more cost effective to society and government when it is applied at the earlier stages of the pathogenesis of the disease. Therefore, and particularly with respect to dementia, validated early detection is critical.

Where inroads have been made, success has come from multidisciplinary approaches to the identification of the key biological signatures for the early development of that disease. It is unlikely that any one

discipline is able to achieve this, and that a combination of integrated clinical sciences, biological sciences, physical sciences and mathematical sciences offers the way forward.

The science in detection and intervention- global standardisation and future direction

Recently, the partners involved in the AIBL collaboration, held a key international meeting (Research and Standardisation in Alzheimer's Disease (RASAD) in Melbourne with the major global players in Alzheimer's disease attending. This meeting addressed the specific need for the standardisation (from a global perspective) of imaging, biomarkers, lifestyle and psychometric measures to enable early detection and to monitor progression. The RASAD program and the key players are attached to this submission (refer to **Attachment A**) and the proceedings of that key gathering will be assembled in the form of an international paper. Immediately following the RASAD meeting, a meeting was held in Perth, WA to specifically address the issue of Lifestyle Approaches for the Prevention of Alzheimer's disease and again the key areas of significance that were addressed are attached (refer to **Attachment B**).

Australia (through AIBL) has taken a lead in driving the recent forum for standardisation. The critical issue for Australian and global researchers, in tackling this disease, is the importance of the amyloid pathway in the pathogenesis of the disease. The discovery of the amyloid protein was fundamental to that process. This protein was co-discovered by one of the Australian AIBL collaborators Professor Colin Masters. More recently, CSIRO and Professor Masters have pioneered an understanding of an elusive structure of that molecule (Streltsov, Varghese, Masters & Nuttall 2011[1]). It is the measurement of amyloid that forms the basis of the Positron Emission Tomography (PET) imaging in the powerful AIBL cohort study. In summary, Australia is intellectually well represented in this area – the challenge is to keep supporting that drive into the future.

In summary, the key approaches to early detection and intervention are as follows:

1. In terms of early detection, advances in imaging technology particularly Magnetic Resonance Imaging (MRI) and PET have been impressive, and one can expect those technologies to be more refined and more sophisticated in the immediate future. Those technologies combined with psychometric measures make a very powerful combination heading strongly towards early detection.
2. There is an increased focus on the presence of signature molecules in blood (and cerebral spinal fluid) that may not only travel in parallel with established Alzheimer's disease but herald an approach to Alzheimer's disease. Australia is playing a major role through AIBL and the Cooperative Research Centre for Mental Health in this area and you can logically expect that globally this will be an area where Australia must be cognisant of not only its own developments but those from overseas.
3. Lifestyle measures, while in many ways are in need of powerful biomarkers, are progressing on many fronts. Outstanding work by individuals in Australia, in the area of physical activity, social interactions defining broad based food, and dietary patterns are continually being recognised nationally and globally. Australia has played a major role in other chronic disorders providing evidence based advice in terms of lifestyle and can continue to play a key role in this area.

The position in Australia with respect to early detection and intervention

In the most recent edition of the Medical Journal of Australia, there is a summary article (refer **Attachment C**) which describes some of the activities of and the findings coming from the AIBL cohort study in Australia. Australia is driving strongly in the direction of earlier detection as being significant not only for identifying early stages of this disease but also providing the platforms for intervention.

Optimizing Australian investment in the research efforts into early detection and intervention?

As indicated in our recent submission into the McKeon Review into Health and Medical Research in Australia, the major health challenge for Australia is to reduce the impact of chronic diseases, including Alzheimer's disease and dementia. More broadly, by increasing the healthy life expectancy (disability free life expectancy), and by having effective management systems for people who already have a chronic disease, the emphasis should be on primary and secondary prevention. This is also the essence of this current submission, namely early detection and intervention. The fundamental emphasis must be upon a translational approach to the development of early detection and intervention. The integration of traditional health and medical research with a translational approach requires high level priority setting and coordination of a whole of systems and whole of government approach. In some cases, this may require a fundamental change to the way health and medical research is funded and managed. What must be avoided is fragmentation, subcritical approaches and lack of coordination in areas of research in dementia for early detection and intervention. The critical lessons from the AIBL activity have been:

1. Addressing key and defined challenges on scale requires the assembly of multi-institutional teams across Australia and the supportive management of those teams.
2. To have global impact, the research must be powerfully embedded not only multidisciplinary but interdisciplinary science with mathematicians, statisticians, biologists, clinical imaging experts, psychometric clinical experts working collaboratively around highly refined and targeted goals with adoption partners. Additionally it is important to have early involvement of those responsible for the translation of science to the community.
3. There is significant value of well coordinated longitudinal cohorts and Australia has the ability to manage these well.

This may require in many cases a change in the way that we operate – it requires a program that integrates unmet need, to due diligence, to science application, adoption partnership, to impact and we have described this most recently in the literature (O'Keefe & Head (2011[2]).

Driving the activities for early detection and intervention faster in an Australian context

Taking as an example the success of the AIBL cohort study can be attributed to a visionary model of funding, namely the Collaboration Cluster Funds – a funding process which aligns deep input from multiple research institutes across Australia with a fundamental set of goals and more particularly, in the case of AIBL, an alignment with the national science institution, CSIRO. This approach could be continued and also enhanced in the following ways:

1. Maintain both consistency and longevity of legacy cohorts in Alzheimer's in Australia, not only AIBL but others.
2. Provide funding mechanisms where research institutions across Australia are attracted to bringing their best to bear on these national cohorts, ie fundamental processes of discovery and adoption of early markers of the disease and approaches to lifestyle interventions that can be implemented in society early using the power of these national cohorts.

In summary

This submission focuses on validated early detection with successful intervention to improve quality of life, effective participation and assistance for people with dementia and their carers. Clearly, this is an area of great importance to Australia and one in which CSIRO is proud to have played a collaborative role together with outstanding institutions and colleagues across Australia in a model recognized globally and one which will hopefully serve as a model for future engagement.

References

[1] Streltsov, VA, Varghese, JN, Masters CL & Nuttall, SD (2011) Crystal structure of the amyloid-β P3 fragment provides a model for oligomer formation in Alzheimer's disease. J Neuroscience 31:1419-1426.

[2] O'Keefe, CM & Head, RJ (2011) Application of logic models in a large scientific research program. Evaluation and Program Planning 34:174-184.

Attachment A

27-29 MARCH 2012

MELBOURNE BRAIN CENTRE
MELBOURNE AUSTRALIA

2012 RASAD

Research and Standardisation in Alzheimer's Disease Conference

CONFERENCE PROGRAM

aibl
The Australian Imaging, Biomarkers and Lifestyle Flagship Study of Ageing

SCIENCE AND INDUSTRY ENDOWMENT FUND

alzheimer's association

FNIH
Foundation for the National Institutes of Health

AIBL research is supported by the Science and Industry Endowment Fund: www.sief.org.au

RASAD CONFERENCE PROGRAM

In 2011, we have seen the emergence of consensus positions on diagnosis of AD based on clinical assessment, psychometric testing, CSF-based biomarkers (the "AD signature"), MRI-volumetrics and molecular imaging of Aβ amyloid by PET techniques. The time is now ripe for a global effort to seek harmonization, standardisation and validation of these various diagnostic and prognostic parameters.

The objective of the 2012 Research & Standardisation in Alzheimer's Disease Conference (RASAD 2012) is to bring together a selected group of key researchers, clinicians, industry, regulatory and government leaders in Alzheimer's disease to generate consensus on standardisation and validation for clinical practice of psychometrics, imaging, biomarkers and lifestyle parameters used in multiple centres around the world.

Registration and conference details are available at: http://www.aibl.csiro.au/rasad2012/

International Speakers will include:

John Morris
Greg Savage
Ron Peterson
Cliff Jack
Bill Jagust
Reisa Sperling
Giovanni Frisconi
Charles DeCarli
Satoshi Minoshima
Chris Rowe
Kaj Blennow
Simon Lovestone
Harald Hampel
Takeshi Iwatsubo
Bill Potter
Les Shaw
Henrick Zetterberg
Sid O'Bryant
Colin Masters
Ralph Martins
Nikos Scarmeas
Carl Cotman
Paul Taylor
Steve Collins
Joyce Suhy
John Seibyl
Richard Margolin
Dan Skovonski
Lennert Thurfjell
Suzanne Craft
Art Toga

Sessions will focus on:

- Neuroimaging
- Biomarkers
- Psychometrics
- Lifestyle
- Future Directions

MONDAY 26 MARCH 2012

4.00PM – 6.00PM

Registration

Melbourne Brain Centre

6.00PM – 8.00PM

Welcome Reception

Melbourne Brain Centre

TUESDAY 27 MARCH 2012

8.30AM

Welcome Address

Professor Chris Baggoley BVSc (Hons), BM BS, BSocAdmin, FACEM

Australian Government Chief Medical Officer

> COGNITION SESSIONS

9.00AM – 9.30AM

Plenary Session

John Morris – Diagnostic criteria and biomarkers

9.30AM – 10.00AM

Session 1

Ron Petersen – Challenges of Longitudinal Data Sets

10.00AM – 10.30AM

Session 2

Greg Savage – Being too smart for your own good: issues in the cognitive assessment of high functioning individuals

10.30AM – 11.00AM

BREAK

11.00AM – 11.50AM

Cognition Panel Session

Chair David Ames

Panel John Morris, Ron Petersen, Greg Savage – Current issues in cognitive assessment for AD research

11.50AM – 12.00NOON

Maria Carrillo and Art Toga – Global Alzheimer's Association Interactive Network (GAAIN)

12.00NOON – 1.00PM

BREAK

> IMAGING SESSIONS

1.00PM – 1.05PM

Introduction

Christopher Rowe

1.05PM – 1.40PM

Plenary Session

Reisa Sperling – The role of neuroimaging in AD and why we need standardization (earlier and more accurate diagnosis in the clinic; subject selection and therapeutic response for drug development)

1.40PM – 2.10PM

Plenary Session

Cliff Jack – Translating 20 years of research with volumetric MRI into multicentre trials and routine clinical practice. Has ADNI solved all the problems?

2.10PM – 2.30PM

Session 1

Charles DeCarli – Visual vs volumetric MRI in the clinic and for research

2.30PM – 2.50PM

Session 2

Giovanni Frisconi – The European perspective on Standardization of Neuroimaging, including commentary on EMA statements on vMRI

2.50PM – 3.10PM

BREAK

3.10PM – 3.50PM

Plenary Session

Cliff Jack – Automated MRI analysis: the academic and commercial options

3.50PM – 4.20PM

Plenary Session

Bill Jagust – Multicenter standardization of PET data processing

4.20PM – 4.40PM

Session 3

Satoshi Minoshima – Automated FDG Analysis: the commercial and academic options

4.40PM – 4.50PM

Session 4

Christopher Rowe – Amyloid PET in clinical practice: is a binary yes or no report enough? Should the patients' age influence the interpretation? Can the different tracers be compared?

4.50PM – 5.05PM

Session 5

Dan Skovonski (Avid/Lilly) – Standardization of Florbetapir for clinical practice; recent evidence of clinical utility and autopsy confirmation of amyloid load

5.05PM – 5.20PM

Session 6

Lennert Thurfjell (GE) – Standardization of Flutemetamol and recent trials of clinical utility in MCI

5.20PM – 5.40PM

TBD – Amyloid PET in therapeutic trials – what has been learnt so far?

5.40PM – 6.00PM

Richard Margolin – Sources of variance in Amyloid PET

6.30PM – 7.40PM

Public Lecture – Consumers and researchers fighting Alzheimer's Disease together

Lecture Theatre, Melbourne Brain Centre

David Ames, Co-Chair of AIBL, and Maria Carrillo, Alzheimer's Association with an opening address by Ita Buttrose AO, OBE, President of Alzheimer's Australia.

Followed by light refreshments.

WEDNESDAY 28 MARCH 2012

> IMAGING SESSIONS (CONTINUED)

8.25AM – 8.30AM

Morning Welcome

8.30AM – 9.00AM

Session 1

Joyce Suhy (Synarc) and John Seibyl (Molecular Neuroimaging (MNI)) – Current Industry Neuroimaging Experience in Clinical Trials: What are the critical challenges and standardization efforts used within the organization, could they be applied in clinical practice and if not why, what is missing?

9.00AM – 9.30AM

Comments from Regulatory Authorities

CAMD, EMEA, TGA, Japan

9.30AM – 10.15AM

Panel Session

Chairs: Keith Johnson and Maria Carillo

Panel (all imaging session speakers) and Audience Participation to address the major issues arising from the presentations and comment on draft recommendations.

Meeting recommendations to be consolidated and presented at close of meeting. To include consensus on visual vs quantification and recommended standards to be met for techniques to be used by researchers and for developers.

Outcomes: Recommended acquisition, processing and interpretation for 1) clinical MRI, FDG and Amyloid PET; 2) clinical trials with MRI, FDG and amyloid PET.

10.15AM – 10.30AM

BREAK

> BIOMARKER SESSIONS

10.30AM– 10.35AM

Introduction

Colin Masters – Rates of change in Biomarkers

10.35AM – 11.05AM

Plenary Session

Kaj Blennow – CSF diagnostics: Overview and use in monitoring therapeutic interventions

11.05AM – 11.30AM

Session 1

Takeshi Iwatsubo – Japanese perspective on CSF diagnostics

11.30AM – 11.55AM

Session 2

Bill Potter – Perspective from Industry

11.55AM – 12.20PM

Session 3

Henrik Zetterberg – Quality controls and validation of CSF tests

12.20PM – 12.30PM

Session 4

Steve Collins – Lessons from running CSF diagnostics for CJD: National and International perspectives

12.30PM – 1.30PM

BREAK

1.30PM – 2.00PM

Session 5

Harold Hampel – Development of hypothesis driven candidate biomarkers (CSF and Blood)

2.00PM – 2.25PM

Session 6

Les Shaw – Validation studies arising from ADNI experience

2.25PM – 2.50PM

Plenary Session

Simon Lovestone – Candidate gene/protein vs proteomic approaches

2.50PM – 3.20PM

BREAK

3.20PM – 3.45PM

Session 7

Sid O'Bryant – Proteomic approaches

3.45PM – 4.10PM

Session 8

Ralph Martins – Perth AIBL approach. Lipidomic approaches and ApoE

4.10PM – 4.30PM

Session 9

Andrew Watt, Kevin Barnham, Blaine Roberts, Noel Faux, Qiao-Xin Li, Alan Rembach – Melbourne AIBL approach: Collection methods, Aβ oligomers, Apo E

4.30PM – 5.00PM

Biomarkers Panel Discussion

8.00PM

Conference Gala Dinner

The Langham Hotel, 1 Southgate Avenue, Southbank, Melbourne

THURSDAY 29TH MARCH 2012

> LIFESTYLE SESSIONS

8.45AM – 8.55AM

Introduction

Ralph Martins

8.55AM – 9.55AM

Lifestyle Plenary

Nick Scarmeas – What is the current level of evidence for dietary intervention to delay the onset of Alzheimer's disease? How can we improve our measures?

9.55AM – 10.40AM

Lifestyle Panel Session

Paul Taylor, Nicola Lautenschlager, Carl Cotman, Nick Scarmeas and Suzanne Craft

1) The strength of current evidence for lifestyle and dietary modifications which prevent or delay the onset of Alzheimer's disease.
2) The messages we should be giving to the general public on lifestyle and dietary modifications which prevent or delay the onset of Alzheimer's disease or other chronic disorders.
3) The studies needed and the collaborative partnerships to address the knowledge gaps for lifestyle and dietary modifications which prevent or delay the onset of Alzheimer's disease.
4) What are the best dietary and lifestyle tools for cohort studies?

10.40AM – 11.00AM

BREAK

> REGULATORY

11.00AM – 1.10PM

BREAK

> FUTURE DIRECTIONS

1.10PM – 3.00PM

Maria Carrillo (Alzheimer's Association) and Tim O'Meara (CSIRO)

Attachment B

McCUSKER ALZHEIMER'S RESEARCH FOUNDATION INC

Lifestyle Approaches for the Prevention of Alzheimer's disease

30th and 31st March 2012

Hyatt Regency Hotel, Perth, Australia

PROGRAM & ABSTRACTS

www.alzheimers.com.au/conference

SCIENCE AND INDUSTRY ENDOWMENT FUND

HALL & PRIOR

HOLLYWOOD PRIVATE HOSPITAL
A proud member of Ramsay Health Care

ARJUNA

AUSTRALIA ECU EDITH COWAN UNIVERSITY

fB fisher biotec australia

Braemar PRESBYTERIAN CARE

McCusker Charitable Foundation

CommonwealthBank

CONTENTS

WELCOME

McCUSKER
ALZHEIMER'S
RESEARCH FOUNDATION INC

Welcome to the inaugural Lifestyle Approaches for the Prevention of Alzheimer's disease (LAPAD) Conference. Lifestyle approaches for the prevention of Alzheimer's disease is an exciting new frontier in the fight against this devastating disease.

Alzheimer's disease is this century's most challenging health issue. It threatens the lives and livelihoods of millions of citizens around the globe as our population ages.

As researchers working in the area we have a responsibility to share our knowledge, increase our collaboration and take these findings to the community.

During this conference we will examine various areas that offer hope in the prevention of Alzheimer's disease. These areas range from the latest advances in diet and lifestyle, to hormonal therapies, and emerging novel technologies for measuring biomarkers.

We will then explore how we translate this knowledge into practice in community-based interventions in Australia and internationally.

One of the most vexing issues for an ageing population is primary prevention of memory loss and Alzheimer's disease, so this conference, with its lifestyle and prevention focus is very timely.

We believe our deliberations and outcomes will galvanise efforts to implement evidence-based intervention strategies in the community, to help prevent and ameliorate Alzheimer's disease.

Conferences don't happen by chance and this one has been made possible by the presence of eminent speakers from all parts of the world; the support from generous sponsors; and the work of a superb organising committee.

We believe you will find the conference an enriching experience from which you will take away new ideas, more collaborations and great friendships.

AUSTRALIA ECU EDITH COWAN UNIVERSITY

Professor Ralph Martins

8.00 – 8.45	Registration	Plaza Foyer
8.45 – 9.00	Welcome to Country – ***Prof Colleen Hayward*** PVC, Equity and Indigenous Head, Kurongkurl Katitjin Centre for Indigenous Australian Education and Research Faculty of Education and Arts, Edith Cowan University Opening Address –His Excellency the Governor Mr Malcolm McCusker AC CVO QC	Plaza Ballroom 1
9.00 – 11.00	***SESSION 1 – Lifestyle (Diet, Physical and Mental Activity) and AD*** Sponsored by Hollywood Private Hospital Chair: ***Prof Ralph Martins***	Plaza Ballroom 1
9.00 – 9.30	Speaker: ***W/Prof Lawrie Beilin*** School of Medicine and Pharmacology, Royal Perth Hospital Unit, UWA Lifestyle Cardiovascular Disease and Healthy Longevity	
9.30 – 10.00	Speaker: ***A/Prof Nikos Scarmeas*** Columbia University, New York Nutrition and Dementia: dietary patterns and mechanisms	
10.00 – 10.30	Speaker: ***Dr Serge Gauthier*** McGill Centre for Studies in Ageing, McGill University How can we prove that lifestyle changes and other interventions are effective to delay the onset of Alzheimer's disease, what do you tell persons at risk and how safe should the treatments be?	
10.30 – 11.00	Speaker: ***Dr Michael Valenzuela*** Regenerative Neuroscience Group, University of NSW Cultivating a Cognitive Lifestyle: Why it's good for your Brain	
11.00 - 11.3	Morning Tea	Plaza Ballroom 2 & 3
11.30 – 13.30	***SESSION 2 – Biomarkers and Lifestyle*** Chair: ***Dr Stephanie Rainey-Smith***	Plaza Ballroom 1
11.30 – 12.00	Speaker: ***A/Prof Sid O'Bryant*** Department of Neurology and the F. Marie Hall Institute for Rural and Community Health, Texas Tech University Health Sciences Centre Diabetes and Drinking Water as Targets for Alzheimer's Disease Prevention	

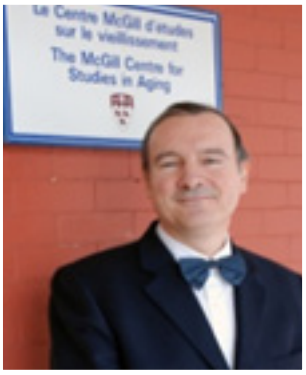
12.00 – 12.30	Speaker: ***A/Prof Markus Wenk*** National University of Singapore Natural lipidomic variations during synaptic function and neurodegeneration	
12.30 – 13.00	Speaker: ***Prof Suzanne Craft*** University of Washington School of Medicine Food for Thought: Diets Associated with Insulin Resistance and Type 2 Diabetes Affects AD Biomarkers	
13.00 – 13.30	Speaker: ***Dr Allen Roses*** Duke University A pharmacogenetic-assisted primary prevention of cognitive impairment clinical trial	
13.30 – 14.30	Lunch Poster Display	Plaza Ballroom 2 & 3
14.30 – 16.30	***SESSION 3 – Hormones and Biomarkers*** Chair: TBA	Plaza Ballroom 1
14.30 – 15.00	Speaker: ***Prof Carl Cotman*** Institute for Brain Ageing and Dementia, University of California, Irvine The role of BDNF in Lifestyle Behavioural Interventions	
15.00 – 15.30	Speaker: ***Dr Malcolm Carruthers*** Centre for Men's Health Clinic, Harley Street, London Testosterone in Health and Disease	
15.30 – 16.00	Speaker: ***A/Prof Christian Pike*** Davis School of Gerontology, University of Southern California Testosterone and the Prevention of Alzheimer's disease	
16.00 – 16.30	Speaker: ***Dr Judith Miklossy*** International Alzheimer Research Center, Switzerland Alzheimer's disease - A neurospirochetosis	
16.30 – 17.00	Afternoon Tea – Sponsored by Fisher Scientific	Plaza Ballroom 2 & 3
18.50	Buses leaving Hyatt Regency Perth to Conference Dinner Venue	
19.00 – 00.00	Reception Drinks, four course dinner and entertainment	
23.15	First bus to hotel	
23.45	Last bus to hotel	

7.45 – 9.00	Registration	Plaza Foyer
8.00 – 9.00	Young Investigator Presentations Chair: ***Dr Michael Fenech***	Plaza Ballroom 1
8.00 – 8.10	Speaker: ***Ms Sarah Brooker*** CSIRO, Adelaide	
8.10 – 8.20	Speaker: ***Ms Diane Hosking*** CSIRO Human Nutrition, Adelaide	
8.20 – 8.30	Speaker: ***Ms Gloria Castellano Gonzalez*** School of Medical Sciences, University of New South Wales	
8.30 – 8.40	Speaker: ***Mrs Ruth Wallace*** School of Exercise and Health Science, Edith Cowan University	
8.40 – 8.50	Speaker: ***Ms Samantha Gardener*** School of Medical Sciences, Edith Cowan University	
9.00 – 12.30	***SESSION 4 – Intervention Strategies*** Sponsored by Hall and Prior Chair: ***Graeme Prior***, Hall and Prior Aged Care Organisation	Plaza Ballroom 1
09.00 – 09.30	Speaker: ***A/Prof Laura Baker*** Department of Psychiatry and Behavioural Sciences, University of Washington School of Medicine Aerobic Exercise as a Cognition-Enhancing and Disease-Modifying Intervention: Early Signals From Pilot Randomized Controlled Trials	
09.30 – 10.00	Speaker: ***Prof Nicola Lautenschlager*** University of Melbourne Physical activity interventions to protect brain health in older adults	
10.00 – 10.30	Morning Tea	Plaza Ballroom 2 & 3
10.30 – 11.00	Speaker: ***A/Prof Amit Dias*** Department of Preventive and Social Medicine, Goa Medical College From Research to Policy: A successful psychosocial intervention for people with dementia in India	
11.00 – 11.30	Speaker: ***Prof Paul Taylor*** University of San Francisco Sharpening The Axe: The Impact of Positive Lifestyle Intervention on Brain Function and Performance	

11.30 – 12.30	Debate: Holistic lifestyle intervention designs for improved cognitive function in those at risk of MCI/AD Chair: ***Prof Richard Head***	Plaza Ballroom 1
12.30 – 13.30	Lunch	Plaza Ballroom 2 & 3
13.30 – 16.45	Public Lectures Chair: ***Dr Judy Edwards MLA***	Terrace Ballroom
13.30 – 13.50	Speaker: ***A/Prof Nikos Scarmeas*** Columbia University, New York Nutrition and Dementia: dietary patterns and mechanisms	
13.50 – 14.10	Speaker: ***Prof Suzanne Craft*** University of Washington School of Medicine, Seattle Food for thought: the role of insulin resistance in brain ageing	
14.10 – 14.30	Speaker: ***Dr Michael Valenzuela*** Regenerative Neuroscience Group, University of NSW Cultivating a Cognitive Lifestyle: Why it's good for your Brain	
14.30 – 15.00	Tea and coffee – Sponsored by Braemar Presbyterian Care Book Signing – 'Maintain your Brain' by Dr Michael Valenzuela	Terrace Ballroom
15.00 – 15.10	Drawing of the Raffle	Terrace Ballroom
15.10 – 15.30	Speaker: ***Dr Radha Murthy*** Nightingales Medical Trust, Bangalore Dementia Risk Reduction - A working model from India	
15.30 – 15.50	Speaker: ***Prof Paul Taylor*** University of San Francisco Prevention vs. Cure	
15.50 – 16.30	'Ask an Expert' Question and Answer Session featuring the Public Lecture speakers and invited guests Chair: ***Prof Gerald Muench***, University of Western Sydney	
16.30 – 16.45	Closing Address : ***Prof Ralph Martins***	
17.30 – 20.30	'Sundowner' barbecue – Supported by the Lions Club of WA Volunteers	Matilda Bay Reserve
17.15	Buses leaving Hyatt Regency Perth to Matilda Bay Reserve	
19.30	First bus to hotel	
20.30	Last bus to hotel	

NIKOS SCARMEAS

NIKOS SCARMEAS was born and raised in Athens, Greece. After obtaining an M.D. degree from the University of Athens he moved to the US and had Neurology residency training (Columbia University Medical Center). He subsequently completed a 2-year clinical fellowship in Ageing and Dementia at Columbia University Medical Center. He also completed a Masters degree in Biostatistics – Epidemiology at the Mailman School of Public Health at Columbia University. He shares his time between clinical work and research. The clinical work includes seeing elderly patients with dementias and cognitive dysfunction in general, supervising and teaching of Neurology residents. His research interests have so far included the following; he has been investigating how factors that can affect cognitive reserve (i.e. higher IQ, education, more demanding occupational attainments, or more engagement in cognitive-social-physical leisure - lifestyle activities) can help elderly cope better with the damage caused to their brains by Alzheimer's disease and ageing, and therefore reduce their risk for dementia and slow down their rates of cognitive and functional decline. He has also used brain imaging to uncover differences in Alzheimer's disease patients and in healthy elderly subjects with either different degrees of cognitive reserve or a genetic predisposition to Alzheimer's disease. More recently, he has developed a special interest in the contribution of diet (in particular composite dietary patterns such as a Mediterranean-type diet and others) and physical activity in dementias and healthy ageing.



SERGE GAUTHIER

SERGE GAUTHIER carried out his medical studies at the Université de Montréal and specialty training in Neurology at McGill University, followed by a Medical Research Council of Canada Fellowship in Neurochemistry at Professor Ted Sourkes' laboratory, Allen Memorial Institute, Montreal. He was on the staff at the Montréal Neurological Hospital and Institute from 1976 to 1985, then Director of the McGill Centre for Studies in Aging, from 1986 to 1997 and currently Professor in the Departments of Neurology & Neurosurgery, Psychiatry, and Medicine, at McGill University. His contributions to research include design and implementation of randomized clinical trials to establish the safety and efficacy of cholinesterase inhibitors, memantine and agents possibly modifying progression of Alzheimer's disease. Special interests include consensus approach to the management of dementia in different stages, and the ethics of research involving persons with dementia.

SID O'BRYANT

SID O'BRYANT has focused much of his work on biomarkers of cognition and Alzheimer's disease. His team has created and cross-validated a blood-based screener for Alzheimer's disease through the Texas Alzheimer's Research & Care Consortium (TARCC). His search for biomarker mediators of cognitive dysfunction/decline among elders is designed to identify modifiable pathways that can be capitalized on for preventative efforts. His work has highlighted the importance of markers related to diabetes, obesity, and cardiovascular disease all of which are modifiable through lifestyle interventions. Additionally, he has generated a method for estimating the health consequences related long-term consumption of drinking water containing "acceptable" levels of toxicants (e.g. arsenic, aluminum, selenium). Dr. O'Bryant completed his PhD in clinical psychology with a neuropsychology emphasis from the University at Albany and his fellowship at the New Orleans VA medical center. He is a fellow of the National Academy of Neuropsychology and is funded by multiple federal and state grants and has published extensively in the field of Alzheimer's disease, cognition, and cultural issues that impact cognition.

MARKUS WENK

MARKUS WENK has been interested in membrane lipids, their structure and function since his undergraduate years at the Biozentrum of the University of Basel. At Yale he introduced and established novel techniques for analysis of phospholipid metabolism at the neurological nerve terminal. His work resulted in scientific publications which have major impact on conceptual advancements in the field of lipid metabolism. He is now spearheading novel approaches in systems level scale analysis of lipids and their interactors (lipidomics). His laboratory is considered one of the leading groups worldwide in this emerging field (Wenk 2010). Markus Wenk is currently Associate Professor of Biochemistry at the National University of Singapore (NUS), Privatdozent at the University of Basel and director of SLING, the Singapore Lipidomics Incubator, a new strategic program at NUS dedicated to Innovation, Education and Partnership in Lipidomics research (http://sling.nus.edu.sg/). He is founder and organizer of the biennial International Singapore Lipid Symposia and an Executive Editor of Progress in Lipid Research.

SUZANNE CRAFT

SUZANNE CRAFT completed her Ph.D. specializing in Neuropsychology at the University of Texas at Austin, and then completed fellowships in Behavioural Neuroscience at Boston University and Harvard Medical School. She is Professor of Psychiatry and Behavioural Sciences at the University of Washington and directs the Memory Disorders Clinic at the VA Puget Sound. Her research investigates the role of insulin resistance in the development of Alzheimer's disease. Her team has identified mechanisms through which insulin resistance may increase the risk of Alzheimer's disease and has now begun clinical trials of intranasal insulin, exercise, and dietary intervention to treat or prevent Alzheimer's disease. Dr. Craft received a National Institute of Health MERIT award for research excellence and an Alzheimer's Association Zenith Award for special contributions to Alzheimer's research.

ALLEN D. ROSES

ALLEN D. ROSES has established an international reputation on his work in pharmacogenetics, exploratory drug discovery, and clinical neuroscience. Dr. Roses is currently appointed at Duke University as the Jefferson-Pilot Professor of Neurobiology and Genetics, Professor of Medicine (Neurology), Director of the Deane Drug Discovery Institute, and Senior Scholar at the Fuqua School of Business. He recently returned to Duke after a decade-long career as a Senior Vice President at GlaxoSmithKline (GSK). Upon joining GW in 1997, he organized genetic strategies for susceptibility-gene discovery, pharmacogenetics strategy and implementation, and integration of genetics into medicine discovery and development. In 2009, Dr. Roses founded Zinfandel Pharmaceuticals to organize and sponsor a diagnostic qualification of a new genetic testing paradigm and to initiate a delay of onset-prevention study of Alzheimer's disease with a commercial partner.

CARL W. COTMAN

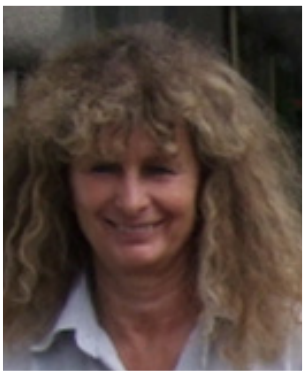
CARL W. COTMAN is Professor of Neurobiology and Behaviour and Neurology at the University of California, Irvine. He obtained his BA from Wooster College, Ohio and his PhD from Indiana University in the Department of Chemistry. His major research interest is to discover interventions which alleviate cognitive dysfunction that occurs with ageing and can reduce the risk for developing Alzheimer's disease. He has published extensively, currently with over 700 publications in the field. He first discovered axon sprouting and new synapse formation in the Alzheimer's disease brain, discovered that brain derived neurotrophic factor (BDNF) is increased with exercise and initially reported that exercise will improve learning and reduce brain amyloid accumulation in transgenic mouse models. His work on exercise is a cornerstone of current interventions involving the impact of lifestyle on brain ageing and Alzheimer's disease. He founded the Institute for Memory Impairments and Neurological Disease (formerly Institute for Brain Ageing and Dementia), established the UCI Alzheimer's Disease Research Center (ADRC) and is a member of the steering committee for the NIA Alzheimer's Disease Cooperative Study group (ADCS). In addition to his basic science research, he has been involved in several clinical trials over the years, including the use of Vitamin E for delaying the onset of Alzheimer's disease, estrogen replacement therapy for Alzheimer's disease, and the use of antioxidants to reduce Alzheimer's disease biomarkers.

MALCOLM CARRUTHERS

MALCOLM CARRUTHERS is the founder and Medical Director of the Centre for Men's Health Clinic. Dr. Carruthers started in General Practice in London over 40 years ago, and is a Life Member of The Royal College of General Practitioners. His career is best summarised as bedside to bench, and back again. Armed with the knowledge of general medicine obtained by a decade of bedside experience, he then went on to specialise in Chemical Pathology at The Middlesex Hospital in London, and became a Senior Lecturer and Consultant at Saint Mary's Hospital, and later Consultant Director of clinical laboratory services at the Maudsley Hospital and Bethlem Royal Psychiatric Hospitals, with a research laboratory in the Institute of Psychiatry. He obtained his MD degree with widely publicised research on 'Stress, Tension and Heart Disease', described in his book 'The Western Way of Death' published by Pantheon Books in London and New York in 1974. He is a Member of the British Cardiovascular Society, and past President of the Society for Psychosomatic Research. He became interested in testosterone when in 1977 he saw the remarkable results being obtained by this hormone treatment used in circulatory disorders by the Danish pioneer, Dr Jens Moller, and frequently visited his Cardiovascular Clinic in Copenhagen over the following 10 years. In 1988 he set up what is now the Centre for Men's Health in Harley Street, specialising in the still controversial diagnosis and treatment of testosterone deficiency, male menopause or andropause. He became a member of The European Academy of Andrology, and International and European Societies for the Study of the Ageing Male. Since then he has done extensive original research on the subject and given talks at numerous international meetings and conferences world-wide. In 2000 was a founder member, Chairman and now President of The Society for the Study of Androgen Deficiency (Andropause Society), a registered charity which has held five International Conferences in London, and training courses in testosterone treatment in England, Russia, and Australia, with others planned in South America and Ireland.

CHRISTIAN PIKE

CHRISTIAN PIKE heads a lab where research is broadly focused on Alzheimer's disease (AD), with the general goals of elucidating factors that regulate AD pathogenesis and pursuing translational approaches that will be useful in the prevention and/or treatment of the disease. A primary area of focus in the laboratory is the relationship between age-related loss of steroid hormones and the development of AD. For example, our research with post-mortem human brain has helped to identify testosterone loss in ageing men as a risk factor for AD. In rodent models, we observe that depletion of androgens accelerates development of AD-like neuropathology and increases neuronal vulnerability to toxic insult. Cell culture studies continue to identify the relevant underlying mechanisms for these androgen effects, including investigation of classic genomic actions as well as activation of rapid cell signalling pathways. In ongoing translational studies, we are building upon our basic science advances to develop specific therapeutic interventions that selectively activate protective androgen pathways (e.g., synthetic testosterone mimetics). Using this general research strategy, we are pursuing conceptually parallel basic science and translational projects to evaluate the interactions between estrogen and progesterone actions in the regulation of neurodegenerative cascades associated with AD.



JUDITH MIKLOSSY

JUDITH MIKLOSSY obtained her MD, PhD, DSc degrees and board certificates in neurology, psychiatry, psychotherapy and neuropathology with conformity certificate for European Union (EU) and AELE in the Faculty of Medicine of the University of Debrecen, Hungary and University of Lausanne, Switzerland. She conducted independent research from 1985 and was head of "Neurodegeneration" research group in the University Institute of Pathology, Division of Neuropathology, Lausanne, Switzerland. She participated in molecular biology research in the Center of Virology and Cancer Biology in Temple University, Philadelphia, and helped to introduce Alzheimer's research between 2001-2004. She was head of the Neuropathology laboratory of Kinsmen Laboratory of Neurological Research in the University of British Columbia, Vancouver, Canada, between 2004-2007. Judith Miklossy is actively involved in research on Alzheimer's disease, other neurodegenerative disorders and Lyme disease from more than 20 years. She is on the scientific advisory board of the Canadian Lyme Foundation and the German Borreliose Centrum. She is founder and president of the Prevention Alzheimer International Foundation and presently director of the International Alzheimer Research Center in Switzerland.

LAURA BAKER

LAURA BAKER is Assistant Professor of Psychiatry and Behavioural Sciences at the University of Washington (UW) School of Medicine in Seattle, WA, USA. She completed her graduate work in experimental psychology/cognitive neuroscience at Washington University in St. Louis, MO, and then a postdoctoral fellowship in her current UW department. Dr. Baker has conducted clinical research in the areas of ageing, mild cognitive impairment (MCI), and Alzheimer's disease (AD) for over 16 years. The focus of her work involves aerobic exercise as a potential cognition-enhancing and disease-modifying intervention for vulnerable older adults at increased risk of cognitive decline. Dr. Baker is also beginning to look at potential synergistic effects of exercise and diet on cognition in these adults, and has examined potential mechanisms to support exercise effects on glucoregulation, growth factor activity, the stress response, and AD biomarkers in blood and cerebrospinal fluid. She is the PI of 6 randomized controlled clinical trials, and co-investigator of more than 40 studies. Over the past 2 years, Dr. Baker has worked closely with Dr. Carl Cotman to plan a pivotal multi-site randomized controlled trial of aerobic exercise in adults with MCI, which will be conducted through the Alzheimer's disease Cooperative Study (ADCS) consortium. Drs. Baker and Cotman will be co-Directors of this important work planned to start in January 2013, work that may ultimately affect standard of care for older adults with MCI.

AMIT DIAS

AMIT DIAS is an epidemiologist and geriatrician by training and is currently the Asst. Professor at the department of Preventive and Social Medicine at Goa Medical College. Dr. Dias is the founder secretary of the Dementia Society of Goa and the coordinator of the 10/66 Dementia research group in India. He is also the coordinator of the Medical and Scientific Advisory Panel for the Alzheimer's and Related Disorders Society of India (ARDSI). He was the former Jt. Secretary of ARDSI. He was one of the authors of the National dementia report that was presented to the Government of India last year. He has a number of publications to his credit in national and international peer reviewed journals. His research on interventions for families of people with dementia won the International FMA-ADI prize for being the best evidence based psychosocial research in 2010. He has been involved in research on a range of subjects like Japanese encephalitis, rabies, iodine deficiency disorders, polio, HIV/AIDS, neurological disorders and heart disease. He is a key member of several Non Governmental Organisations in India and is currently the honorary project director for Sangath's dementia services in Goa supported by the Rotary club of Crosby, UK.

PAUL TAYLOR

PAUL TAYLOR is an Exercise Physiologist, Nutritionist, Neuroscientist and Research Professor at The University of San Francisco, as well as being a former British Royal Navy Aircrew Officer. He is the Director of The Body-Brain Performance Institute, where he delivers Executive Performance and Leadership workshops to large multi-nationals such as SAP, Google, NAB and Accenture and in late 2010, opened Australia's first Body-Brain Fitness gym, Acumotum. He is also the founder and Director of The Personal Training Academy, a Registered Training Organisation that delivers Fitness Education and recently co-launched a worldwide initiative, PTA Global. In 2010 Paul created and co-hosted the Channel ONE HD TV series, Body and Brain Overhaul, and appears regularly on The Biggest Loser TV series as a consultant. He is also widely published in health magazines and is the creator of BioAge fitness testing software that is used by many Australian and overseas gyms. In addition to an extensive background in health and fitness, Paul has a proven track record in leadership, management and dealing in high-pressure situations, through his former roles as an Airborne Anti-submarine Warfare Officer and a Helicopter Search-And-Rescue Crew Member with the Royal Navy Fleet Air Arm, and has undergone rigorous Combat Survival and Resistance-to-Interrogation Training.

RADHA S. MURTHY

RADHA S. MURTHY is the founder and Managing Trustee of the Nightingales Medical Trust (NMT) established in 1998, a nongovernmental organization that works exclusively for the welfare of senior citizens in Bangalore. Moved by the plight of elders and the disabled who have difficulty in visiting hospitals, she started Nightingales Home Health Services in January 1996 to provide all possible medical care at the doorstep. At a time when old age homes were mushrooming in the city, NMT supported the family as a unit and strived to provide support systems to maintain elders with their families. Dr Radha started several projects under NMT to address various issues concerning the elderly. The services include an elders enrichment centre ,dementia day care, elders helpline, rural mobile Medicare and the Nightingales Centre for Ageing and Alzheimer's which is India's first comprehensive care facility for people with Dementia. Dr Radha's research interest includes the effect of Indigenous therapies and Lifestyle modification in Dementia risk reduction and non pharmacological interventions for management of challenging behaviours in Dementia. The numbers with Dementia are increasing and the services available in India currently cannot meet this need. Dr Radha is keen to establish a rural dementia care model which would be holistic and cost effective. For her commitment towards elder care she was awarded the Namma Bengaluru Award in 2011.

LAWRIE BEILIN

LAWRIE BEILIN was a Winthrop Professor of Medicine with the School of Medicine & Pharmacology at the Royal Perth Hospital Campus for the University of Western Australia and Consultant Physician at Royal Perth Hospital from 1977 until his retirement from these positions at the end of 2011. He will continue work in an Honorary Senior Research Fellow appointment and has Emeritus Professor title with the University of Western Australia and is an Emeritus Physician at Royal Perth Hospital. He is currently the Chair of the RPH Medical Research Foundation and has been since November 2000. He has had a distinguished academic and scientific research career engaged in research into high blood pressure over the last 48 years beginning in London and Oxford and since 1977 he has developed a research team in the University of Western Australia Cardiovascular Research Centre which has been responsible for major advances in understanding the role of diet, and lifestyle in high blood pressure and cardiovascular disease. He has over 550 publications and his contributions to knowledge in his area of research have been recognised Nationally and Internationally by incorporation and reference to his group's work in Heart Foundation Guidelines, World Health Organisation and International Society of Hypertension Guidelines. For his contributions to medical education and research he was awarded the Prime Ministers Centenary Medal in 2001 and the Officer of the Order of Australia in the General Division (AO) in 2003 as well as other teaching and research awards. Professor Beilin has held many positions on National and International Committees over the above period and was President of the International Society of Hypertension from 2002 to 2004 and from 2002 he still holds the position of Vice President of the World Hypertension League.

MICHAEL VALENZUELA

MICHAEL VALENZUELA is a Senior Research Fellow at the School of Psychiatry, and leader of the Regenerative Neuroscience Group, UNSW. His background is in psychology, clinical medicine and neuroscience research. Dr Valenzuela's PhD focused on the topic of brain reserve, specifically how complex mental activity affects the development and expression of dementia. This question was tackled using multiple different technologies including epidemiology, neuroimaging and cognitive research. For this work he was awarded the prestigious Eureka Prize for Medical Research in 2006. More recently, he was honoured to receive a NHMRC Excellence Award for the top-ranked clinical Career Development Award in the 2010 round. Dr Valenzuela's current research is aimed at further understanding the competing forces of neuroplasticity and degeneration in the human brain, and how these can either lead to, or help prevent, dementia. This research includes studies with stem cells, animal models, brain tissue, human clinical trials and large multinational population-based samples. Dr Valenzuela is the author of popular science book Maintain Your Brain and believes that the problem of dementia is a critical one for modern society, and is committed to communicating dementia-prevention health ideas to the public.

NICOLA LAUTENSCHLAGER

NICOLA LAUTENSCHLAGER is an academic old age psychiatrist who received her undergraduate and postgraduate training, including an MD in 1994, at the Technical University in Munich, Germany. In 1995 she spent one year as a postdoc fellow at Boston University in the United States. From October 2000 to June 2008 NL worked at the University of Western Australia in Perth, Australia where her last position was Professor in Old Age Psychiatry and Deputy Head of School. In July 2008 she took up the position of University of Melbourne Professor & Chair of Old Age Psychiatry at the Department of Psychiatry, where she is also Head of the Academic Unit for Psychiatry of Old Age. NL is the Director for the St. Vincent's Aged Mental Health Service at St. George's Hospital in Kew. NL is the current editor-in-chief of the scientific journal International Psychogeriatrics. Her current research focus is early diagnosis of cognitive impairment and intervention trials for older adults to improve mental health outcomes.

LAWRIE BEILIN

LIFESTYLE CARDIOVASCULAR DISEASE AND HEALTHY LONGEVITY

This presentation will review data illustrating the potential impact of both specific and comprehensive lifestyle changes to prevent and help manage hypertension and cardiovascular disease and with regard to their effects on health and longevity. Particularly emphasis will be placed on weight control and avoidance of overweight and obesity, physical activity, tobacco and alcohol consumption and other dietary measures other than dietary salt and potassium discussed elsewhere at this meeting.

Evidence for lifestyle effects on risk factors for cardiovascular disease are well established from randomised controlled trials. For example significant and sometimes substantial blood reductions have been shown with weight reduction, increased physical activity, alcohol moderation, vegetarian diets, non vegetarian diets increasing fruit and vegetable consumption and decreasing saturated fat intake, increased dietary fish or protein or fibre or combinations of the above. The ability for exercise, weight control and various dietary changes to favourably influence serum lipids, insulin resistance and circulating inflammatory markers is also well established. Many of the behaviours influencing the risk of cardiovascular disease also affect the risk of other chronic disorders such as some common cancers, diabetes mellitus, chronic lung diseases and dementia.

Evidence for effect of lifestyle changes on morbidity and mortality in populations cannot always meet the gold standard of randomised controlled trials. Such was the case with the absence of hard outcome trial evidence for the benefits of smoking cessation or avoidance. However inaction is likely to have disastrous consequences given current trends in overweight, obesity and diabetes. Nonetheless there is a high priority for research into the most effective and acceptable nutritional and lifestyle measures for reducing the risk of a range of chronic diseases associated with ageing, the mechanisms by which they operate, and the importance for optimising effects of medication. Not the least important is research into the most cost effective means for both rich and poorer nations to ensure the widespread uptake and maintenance of healthier lifestyles in the face of the major cultural/societal changes taking place worldwide.

NIKOS SCARMEAS

NUTRITION AND DEMENTIA: DIETARY PATTERNS AND MECHANISMS

There has been considerable research on the relationship between diet elements and risk for Alzheimer's disease, cognition and dementia but the results have been conflicting. Among the various methodological reasons for non-consistent results, the examination of individual foods or nutrients (vs. dietary patterns) stands as an important one because we do not consume foods in isolation but as part of an overall diet. We briefly outline our previous investigations of the relationship between dietary patterns and cognition and more specifically of associations between a Mediterranean-type diet and Mild Cognitive Impairment, Alzheimer's disease and Alzheimer's disease mortality. We have found that higher adherence to this diet is associated with lower risk for developing Alzheimer's disease and Mild Cognitive Impairment. We also found that higher adherence to this diet even after clinical onset of Alzheimer's disease is associated with prolonged survival. We also present data on other, non-Mediterranean-type dietary patterns derived using modern nutritional epidemiology techniques). The biological mechanisms of all the above associations may include vascular, anti-inflammatory, anti-oxidative, metabolic and even amyloid-related ones. We summarize some of our recent studies investigating the above potential mediating pathways.

SERGE GAUTHIER

HOW CAN WE PROVE THAT LIFE STYLE CHANGES AND OTHER INTERVENTIONS ARE EFFECTIVE TO DELAY THE ONSET OF ALZHEIMER'S DISEASE, WHAT DO YOU TELL PERSONS AT RISK AND HOW SAFE SHOULD THE TREATMENTS BE?

There are a growing number of treatment options for persons at risk of Alzheimer's disease later in life. Genotyping and biomarkers are available to further characterize the level of risk for individuals, raising issues of disclosure of personal information that can cause distress and impact employability. Ethical considerations require that preventive treatments for asymptomatic persons are not above minimal level of risk (equivalent to risks encountered in average daily life activities).

The trial methodology to prove the safety and efficacy of prevention studies is still evolving, but two placebo-controlled trials using Gingko Biloba are good models to learn from: persons with no cognitive complaints or with minimal cognitive complaints were treated for 7 and 5 years respectively, monitored regularly by their family doctors, and seen yearly at memory clinics. Although the drug was ineffective to delay cognitive decline and dementia, the high retention rate and the involvement of primary care practitioners are useful lessons for designing new studies involving life style changes or medications targeted at specific etiologic factors.

MICHAEL VALENZUELA

CULTIVATING A COGNITIVE LIFESTYLE: WHY IT'S GOOD FOR YOUR BRAIN

An active cognitive lifestyle translates to continually challenging one's mind to learn new things, take up new activities, and meet new people. What medical research has revealed in the last 10 years is that in the same way an active physical lifestyle is good for one's heart health, so an active cognitive lifestyle is good for one's brain health. In particular, our group has identified an active cognitive lifestyle as important for helping protect individuals from mental decline and dementia. This simple idea has many interesting facets. In this talk, A/Prof Valenzuela will give examples of the various strands of evidence, how they may join together in a coherent theory, and what the implications are for how we live our life.

SID O'BRYANT

DIABETES AND DRINKING WATER AS TARGETS FOR ALZHEIMER'S DISEASE PREVENTION

To date, there remains to be a preventative option for Alzheimer's disease (AD). The current talk will discuss (1) how recent work on blood-based biomarkers for AD may identify biological pathways for preventative efforts targeting the diabetes – AD link and (2) possible drinking water sources of AD risk. (1) Many of the recently identified markers contained in blood screeners for AD are relevant to the biology of diabetes, which is the strongest mid-life risk factor for AD when considering the cardiovascular related diseases. A review of these markers as they relate to both AD and DM will be presented. (2) While a great deal of information is available regarding the neurodevelopmental and neurotoxic consequences of acute environmental exposures, much less work has been conducted regarding chronic exposures to "acceptable" levels of toxicants in drinking water. Our group has conducted a series of projects utilizing geospatial information systems (GIS) analyses to model current and chronic residential (and regional) groundwater exposures. We have shown that such exposures are harmful to human health and increase risk for poor health outcomes including cognitive dysfunction. Together this work points towards several lifestyle opportunities for preventative efforts for AD both at the individual and population level.

MARKUS R WENK

NATURAL LIPIDOMIC VARIATIONS DURING SYNAPTIC FUNCTION AND NEURODEGENERATION

Once viewed simply as a reservoir for carbon storage, lipids are no longer cast as bystanders in the drama of biological systems. The emerging field of lipidomics is driven by technology, most notably mass spectrometry, but also by complementary approaches for the detection and characterization of lipids and their biosynthetic enzymes in living cells. The development of these integrated tools promises to greatly advance our understanding of the diverse biological roles of lipids (Wenk 2010 Cell 143(6):888-95).

Lipid levels are governed by genetics, diet and the environment and lipid metabolism forms a central basis of homeostasis (structure, energy) and communication (cell signalling). Genetic studies in mice have provided insights into alterations in inositol signalling lipids which are compatible with life but which lead to abnormal neuronal function (Cremona et al 1999 – Cell 99(2):179-88; Di Paolo et al 2004 – Nature 431(7007):415-22). However, lower and upper boundaries of natural variations in lipid levels are to a large extent unknown. Apolipoprotein E4 is a major risk factor for late onset Alzheimer's disease but variations in ApoE isoforms do not per se affect bulk lipid homeostasis in mouse brain (Sharman et al 2010 - J Alzheimers Dis 20(1):105-11). Comparative lipidomic analysis of brain tissue from animal models and human patients with Alzheimer's disease reveals a multitude of changes as well as some commonalities in different brain regions (Chan et al 2012 – J Biol Chem 287(4):2678-88). Changes in cholesterol and sphingolipid metabolism are consistent with a view that adaptation in lipid homeostasis during

ageing may affect specialized membrane organization and trafficking. Sterol homeostasis was also shown to be correlated with increased risks to Parkinson's disease. Lanosterol, (the first sterol with the characteristic cycloalkane ring structure during biosynthesis of cholesterol, induces mitochondrial uncoupling and protects dopaminergic neurons from cell death (Lim et al 2012 – Cell Death Differ 19(3):416-27).

Understanding better the fundamentals of natural variation in lipidomes as well as specific recognition of individual lipid species are main scientific aims of SLING, the Singapore Lipidomics Incubator (http://www.sling.nus.edu.sg/). Shaped by a five year competitive research program supported by the National Research Foundation and the National University of Singapore, this centre is a major global magnet for collaborating parties in lipidomics – from academia and industry – delivering new technologies and intellectual capital.

SUZANNE CRAFT

FOOD FOR THOUGHT: DIETS ASSOCIATED WITH INSULIN RESISTANCE AND TYPE 2 DIABETES AFFECTS AD BIOMARKERS

Type 2 diabetes, pre-diabetes, and insulin resistance have reached epidemic proportions in the US, affecting more than 40% of adults over 20 years of age, and 70% of adults over 65 years of age. These statistics are troubling in many respects, and in particular because diabetes and related conditions increase the risk of developing Alzheimer's disease (AD). There has been much interest in modifiable lifestyle factors that have contributed to the widespread prevalence of diabetes and insulin resistance. Diet is one such factor, and excess calories, saturated fat, and sugar all increase the risk for diabetes. Interestingly, epidemiologic studies also show a correlation between dietary patterns and risk of AD. Similarly, intriguing animal studies suggest that diet can promote the abnormal accumulations of β-amyloid in the brain. Based on these findings, we examined the effects of a diet intervention on β-amyloid in spinal fluid, given that spinal fluid levels are thought to reflect concentrations in brain. Twenty-nine adults with mild cognitive impairment (a condition thought in most cases to represent early AD) and 19 normal adults received one of two diets for four weeks: a high saturated fat/high simple sugar diet ('HIGH' diet) or a low saturated fat/low sugar diet ('LOW' diet). The diets provided the same amount of calories as participants' normal diet, so their weight did not change. Participants received a spinal tap before and after the four week diet. We showed that for normal older adults, the HIGH diet raised β-amyloid, moving levels in a direction of increased AD pathology, and the LOW diet lowered it. For adults with MCI, the LOW diet moved levels of β-amyloid in a healthy direction. We also examined how the diets affected a marker of injury to brain cells, known as F2-Isoprostane. The LOW diet lowered and the HIGH diet raised F2-Isoprostane levels in spinal fluid. For normal adults consuming the HIGH diet, lowered insulin was associated with increased F2-Isoprostane. Finally, apolipoprotein E plays an important role in clearing β-amyloid, and its efficiency is related in part to whether its molecules are lipidated, that is, combined with healthy fats. Lipid-free apolipoprotein E does not bind to and chaperone β-amyloid effectively. We found that adults who have a genetic risk factor for AD that is related to the apolipoprotein E gene (the ε4 variant of this gene) had strikingly higher levels of lipid-free apolipoprotein E in general, and that lowering of these levels with the LOW diet was associated with lowering of the toxic free form of β-amyloid. These results suggest that diet may have protective or pathologic influences on brain health, and support further studies on the role of diet in AD.

ALLEN ROSES

A PHARMACOGENETIC-ASSISTED PRIMARY PREVENTION OF COGNITIVE IMPAIRMENT CLINICAL TRIAL

A large Phase III prevention trial designed to test the effect of a safe drug on the delay of onset in cognitive impairment of the Alzheimer type is planned to launch later this year. The trial will be a simultaneous qualification of a newly discovered genetic predictor, and an assessment of delay of cognitive impairment by a therapeutic agent. The trial is made possible by using the newly discovered genetic marker [TOMM40] to enrich a High Risk group of normal subjects for treatment. The trial must both verify the utility of the genetic marker in guiding subject selection and test drug efficacy against the delay of cognitive impairment endpoint. In order to facilitate this clinical prevention study: Zinfandel has consulted with regulatory bodies at the US FDA, 2) key investigators at suitable sites around the world were identified, 3) registries of cognitively normal subjects were developed, and 4) suitably qualified personnel were engaged. The clinical trial is designed and executed by a Zinfandel-Takeda Pharmaceutical Alliance. Perth Australia is one of the Testing Center Sites. Professor Ralph Martins will be the Principal Investigator on site and it is expected to enrol approximately 350 subjects. The design and details will be discussed.

CARL COTMAN

THE ROLE OF BDNF IN LIFESTYLE BEHAVIORAL INTERVENTIONS

Behavioral and lifestyle interventions including diet, exercise, and cognitive training are emerging as potentially powerful strategies for promoting brain health. Studies in animal models offer the advantage over human research that dietary and behavioral conditions such as exercise and learning can be precisely controlled for prolonged periods, allowing the relative contributions of each intervention to be evaluated. In rodent and canine studies we have found exercise and behavioral enrichment is a powerful means to induce BDNF. BDNF can mediate cellular responses supporting synaptic plasticity and improve learning. Recent work in the aged canine also suggests that BDNF may be a common endpoint for the synergistic beneficial effects of diet and behavioral enrichment interventions. BDNF is able to translate behavioral influences to cellular and molecular events that support brain health and plasticity.

MALCOLM CARRUTHERS

TESTOSTERONE IN HEALTH AND DISEASE

From the womb onwards, as the 'king of hormones and hormone of kings', testosterone shapes a man's body, mind, health and destiny. It is not, as often portrayed, just a sex hormone affecting libido and reproductive function, but hard-wires the developing brain, and through its action on the stem cells affects the development and repair of virtually every organ in the body. Testosterone deficiency has been implicated in the causation of heart disease, diabetes, osteoporosis, and Alzheimer's disease.

The diagnosis of testosterone deficiency is not as simple as measuring its level in the blood. Not only are laboratory measures notoriously unreliable, but they ignore the multiple complex metabolic paths affecting the production and action of testosterone. This is underlined by the very poor correlation between levels of this hormone and clinical signs and symptoms. What is needed is a 'patient centred' clinical approach rather than the orthodox 'lab-centred' view. Because of a varying degree of 'testosterone resistance', the situation is similar to the insulin resistance seen in adult onset diabetes. As with diabetes, the condition can be defined as 'An absolute or relative deficiency of testosterone or its metabolites according to the needs of that individual at that time in his life.'

It could be stated as inconvenient truth that testosterone deficiency is (other than Type 2 Diabetes in which it also plays a part in up to 50% of cases), the commonest endocrine disorder in the adult male, and yet the least commonly diagnosed and treated.

It is in dementia, including both Alzheimer's disease and cerebrovascular disease, that some of the greatest possibilities for prevention and treatment with hormones, either directly or by modifications in life-style factors are opening up. There are many reasons why testosterone shares many of the neuroprotective actions of oestrogens, and an increasing amount of research evidence in favour of their clinical use. Also, the therapeutic application of testosterone to mitigate the cognitive decline following traumatic brain injury in both peace and war, and following major surgery are areas of major research potential.

CHRISTIAN PIKE

TESTOSTERONE AND THE PREVENTION OF ALZHEIMER'S DISEASE

Although ageing is the most significant risk factor for the development of Alzheimer's disease (AD), the age changes that confer increased susceptibility to AD are not well defined. One normal age change in men that is significantly associated with increased AD risk is testosterone depletion. In aged men, low levels of testosterone in both brain and blood have been observed to precede neuropathological and clinical diagnoses of AD, suggesting that low testosterone occurs prior to and thus may promote AD pathogenesis. Consistent with potential protective roles of testosterone against development of AD are numerous beneficial neural actions, including neuroprotection, inhibition of beta-amyloid accumulation and tau phosphorylation, and promotion of spine density and select aspects of cognition. Importantly, work in transgenic mouse models of AD has demonstrated that testosterone negatively regulates development of AD-like pathology. Together, these lines of evidence suggest that testosterone-based hormone therapy in ageing men may be a useful strategy to prevent AD.

JUDITH MIKLOSSY

ALZHEIMER'S DISEASE - A NEUROSPIROCHETOSIS

Recognition that pathogens can produce slowly progressive chronic diseases has resulted in a new concept of infectious diseases. The pioneering work of Marshall and Warren has established that Helicobacter pylori (H. pylori) causes stomach ulcer. Increasing number of recent reports claim that infectious agents are also associated with atherosclerosis, cardio- and cerebrovascular disorders, diabetes mellitus, chronic lung and bowel diseases, and various neuropsychiatric disorders.

The possibility that a slow acting unconventional infectious agent, acquired at an early age and requiring decades to become active may be involved in Alzheimer's disease (AD) has been proposed decades ago. It is established that chronic spirochetal infection can cause slowly progressive dementia, brain atrophy and amyloid deposition in syphilis. Recent observations suggested that various types of spirochetes, similarly to Treponema pallidum, could also cause dementia and reproduce the pathological and biological hallmarks of AD. Statistical analysis of all data available on the association of various spirochetes with AD, and critical analysis of the association following Koch's and Hill's criteria were in favour of a causal relationship. Following Hill, in such cases prompt action is needed. Syphilitic dementia was almost eradicated by Penicillin, suggesting that with appropriate targeted therapy one might also prevent AD-type dementia.

ABSTRACTS SATURDAY

LAURA BAKER

AEROBIC EXERCISE AS A COGNITION-ENHANCING AND DISEASE-MODIFYING INTERVENTION: EARLY SIGNALS FROM PILOT RANDOMIZED CONTROLLED TRIALS

Aerobic exercise has well-established benefits on cardiovascular health, glucoregulation, lipid metabolism, growth factor activity, and physiological response to stress, any one of which when compromised confers additional risk of developing Alzheimer's disease (AD). Aerobic exercise has favourable effects on brain structure, function, and pathological processes in AD animal models, and is associated with reduced risk of cognitive impairment and dementia in observational human studies. In two randomized controlled pilot clinical trials, we reported cognition-enhancing effects following 6 months of supervised vigorous aerobic exercise in older adults at increased risk of cognitive decline and dementia. In these studies, exercise also modulated circulating levels of the AD biomarker, beta-amyloid 1-42 (Aβ42). Recently, in a controlled 4-week study examining the effects of a low fat/low sugar vs. a high fat/high sugar diet on AD biomarkers in cerebral spinal fluid (CSF), we showed that higher frequency of vigorous exercise (by self-report) protected against the negative effects of the unhealthy diet on CSF Aβ42 in cognitively normal adults, and potentiated positive effects of the healthy diet on this biomarker in adults with mild cognitive impairment (MCI). These findings, together with those from a growing number of other clinical studies, provide early support for the therapeutic potential of vigorous physical exercise to attenuate age- and disease-related changes in cognitive function and other processes associated with pathological brain ageing.

NICOLA T. LAUTENSCHLAGER

PHYSICAL ACTIVITY INTERVENTIONS TO PROTECT BRAIN HEALTH IN OLDER ADULTS

This presentation will highlight recent developments in research focusing on intervention strategies with physical activity. Target groups are older adults with normal cognition, subjective memory complaints, mild cognitive impairment and dementia. The aim of these interventions is to determine whether regular physical activity can have a positive impact on cognition, function and mental health outcomes. Whilst this is an increasingly active research area, many challenges remain in the quest to develop effective strategies for health promotion: what is the correct time window of exposure, what type of physical activity, duration and intensity is necessary, how to motivate sedentary older adults, how to make the programs relevant to the participants, how to avoid adverse events, how to design research trials and how to best translate findings into the community.

AMIT DIAS

FROM RESEARCH TO POLICY: A SUCCESSFUL PSYCHOSOCIAL INTERVENTION FOR PEOPLE WITH DEMENTIA IN INDIA

The 10/66 dementia research group was established at the ADI meeting in India more than a decade ago. This was in recognition of the fact that only 10% of the funds for research on dementia go to developing countries where more than 66% of the people with dementia live. To begin with, there was hardly any information available on dementia, and the community at large often misconstrued it to be normal ageing and would not visit medical facilities. The treatment gap for dementia as seen in India was more than 90%. India is now estimated to have 3.7 million people with dementia and services are negligible (Dementia India report 2010). There was a need for developing a culturally appropriate service to bridge the treatment gap for dementia in India using low cost, locally available resources. The following study is an attempt to develop and evaluate the effectiveness of a home based intervention in reducing caregiver burden, promoting caregiver mental health and reducing behavioural problems in elderly people with dementia.

This was a randomized controlled trial in which the person with dementia-caregiver dyad was randomly allocated either to receive the intervention immediately or to a waiting list group which received the intervention after 6 months. It was carried out in communities based in two talukas (administrative blocks) in Goa, India. Mild to moderate cases with dementia (diagnosed using the DSM IV criteria and graded using the Clinical Dementia Rating scale) and their caregivers were included in the trial. Community based intervention provided by a team consisting of Home Care Advisors who were supervised by a counsellor and a psychiatrist, focusing on supporting the caregiver through information on dementia, guidance on behaviour management, a single psychiatric assessment and medication if needed. We measured caregiver mental health (General Health Questionnaire), caregiver burden (Zarit Burden Score), distress due to behavioural disturbances (NPI-D), behavioural problems in the subject (NPI-S) and activities of daily living in the elder with dementia (EASI). Outcome evaluations were masked to the allocation status.

Eighty one families enrolled in the trial; 41 were randomly allocated to the intervention group. 59 completed the trial and 18 died during the trial. The intervention led to a significant reduction of GHQ (21.12, 95% CI 22.07 to 20.17) and NPI-D scores (21.96, 95%CI 23.51 to 20.41) and non-significant reductions in the ZBS, EASI and NPI-S scores. We also observed a non-significant reduction in the total number of deaths in people with dementia in the intervention arm (OR 0.34, 95% CI 0.01 to 1.03).

Home based support for caregivers of persons with dementia, which emphasizes the use of locally available, low cost human resources, is feasible, acceptable and leads to significant improvements in caregiver mental health and burden of caring. The trial has formed the basis for developing dementia services for India as mentioned in the 'Dementia India report' released on World Alzheimer's Day 2010. The Alzheimer's and related Disorders society of India is working with the government to help develop a national dementia policy based on these guidelines.

PAUL TAYLOR

SHARPENING THE AXE: THE IMPACT OF POSITIVE LIFESTYLE INTERVENTION ON BRAIN FUNCTION & PERFORMANCE

Chronic stress is now well recognized to impact negatively upon overall health, productivity, brain function and long-term brain health. Although there is some evidence in the US that employee wellness programs have a positive impact on absenteeism, presenteeism, productivity and the bottom line, there is a different dynamic between the US and Australia in healthcare costs incurred by a company.

In addition, the vast majority of employee wellbeing programs examine markers of physical health, but there is a paucity of information regarding the impact of wellness programs on mental health and clinical markers of brain function.

In 2011 The Body-Brain Performance Institute, in conjunction with The Centre of Human Psychopharmacology at Swinburne University, conducted a clinical trial to examine the impact of an integrated lifestyle program on employee brain function, wellbeing and productivity.

The employer was SAP and the trial period coincided with the lead-up to the end of the financial year, the most stressful time for employees. This presentation will discuss the results of the clinical trial and highlight a new mode of thinking that integrates positive lifestyle and more vigorous physical activity with employee brain function, wellbeing and productivity.

NIKOS SCARMEAS

NUTRITION AND DEMENTIA: DIETARY PATTERNS AND MECHANISMS

There has been considerable research on the relationship between diet elements and risk for Alzheimer's disease, cognition and dementia but the results have been conflicting. Among the various methodological reasons for non-consistent results, the examination of individual foods or nutrients (vs. dietary patterns) stands as an important one because we do not consume foods in isolation but as part of an overall diet. We briefly outline our previous investigations of the relationship between dietary patterns and cognition and more specifically of associations between a Mediterranean-type diet and Mild Cognitive Impairment, Alzheimer's disease and Alzheimer's disease mortality. We have found that higher adherence to this diet is associated with lower risk for developing Alzheimer's disease and Mild Cognitive Impairment. We also found that higher adherence to this diet even after clinical onset of Alzheimer's disease is associated with prolonged survival. We also present data on other, non-Mediterranean-type dietary patterns derived using modern nutritional epidemiology techniques). The biological mechanisms of all the above associations may include vascular, anti-inflammatory, anti-oxidative, metabolic and even amyloid-related ones. We summarize some of our recent studies investigating the above potential mediating pathways.

SUZANNE CRAFT

FOOD FOR THOUGHT: THE ROLE OF INSULIN RESISTANCE IN BRAIN AGEING

Type 2 diabetes, pre-diabetes, and insulin resistance have reached epidemic proportions in the US, affecting more than 40% of adults over 20 years of age, and 70% of adults over 65 years of age. These statistics are troubling in many respects, and in particular because diabetes and related conditions increase the risk of developing Alzheimer's disease (AD). There has been much interest in modifiable lifestyle factors that have contributed to the widespread prevalence of diabetes and insulin resistance. Diet is one such factor, and excess calories, saturated fat, and sugar all increase the risk for diabetes. Interestingly, epidemiologic studies also show a correlation between dietary patterns and risk of AD. Similarly, intriguing animal studies suggest that diet can promote the abnormal accumulations of a toxic protein that is a primary contributor to AD, the β-amyloid protein. Based on these findings, we examined the effects of a diet intervention on β-amyloid in spinal fluid, given that spinal fluid levels are thought to reflect concentrations in brain. Adults with mild cognitive impairment (a condition thought in most cases to represent early AD) and normal adults received one of two diets for four weeks: a high saturated fat/high simple sugar diet ('HIGH' diet) or a low saturated fat/low sugar diet ('LOW' diet). We showed that for normal older adults, the HIGH diet raised β-amyloid, moving levels in a direction of increased AD pathology, and we also observed similar changes in other markers of brain injury. LOW diet lowered it. For adults with MCI, the LOW diet moved levels of β-amyloid in a healthy direction. These results suggest that diet may have important protective or pathologic influences on brain health, and support further studies on the role of diet in AD.

MICHAEL VALENZUELA

CULTIVATING A COGNITIVE LIFESTYLE: WHY IT'S GOOD FOR YOUR BRAIN

An active cognitive lifestyle translates to continually challenging one's mind to learn new things, take up new activities, and meet new people. What medical research has revealed in the last 10 years is that in the same way an active physical lifestyle is good for one's heart health, so an active cognitive lifestyle is good for one's brain health. In particular, our group has identified an active cognitive lifestyle as important for helping protect individuals from mental decline and dementia. This simple idea has many interesting facets. In this talk, A/Prof Valenzuela will give examples of the various strands of evidence, how they may join together in a coherent theory, and what the implications are for how we live our life.

RADHA MURTHY

DEMENTIA RISK REDUCTION - A WORKING MODEL FROM INDIA

Dementia has become one of the most important health care issues with the changing trend in demographic ageing. It is estimated that over 3.7 million people are affected by dementia in India. This is expected to double by 2030. As resources for management of dementia are limited developing preventive strategies becomes important.

A growing body of research supports the protective effects of late-life intellectual stimulation on incident dementia. Recent research from both human and animal studies indicates that cognitive stimulation, physical activity and socialization in old age are an important predictor of enhancement and maintenance of cognitive functioning. An engaged lifestyle during adulthood has been shown to be correlated with a variety of benefits, including enhanced longevity, reduced risk of dementia, enhanced cognitive resilience in the face of brain pathology, and enhanced mental flexibility.

Nightingales medical trust, an NGO based in Bangalore working for the welfare of senior citizens conceptualized an elder enrichment centre in 1999. To combat the loneliness and other psychological problems in the elderly a series of planned activities were conducted on daily basis. The membership of the centre currently stands at 300 and a cross sectional study of the cognitive states of the 300 current members revealed no symptoms of Alzheimer's disease. A detailed retrospective analysis of the program is currently underway to understand the positive impact it has had on cognition. The outcomes would help to plan a dementia risk reduction program.

PAUL TAYLOR

PREVENTION VS. CURE

It is now well accepted that primary prevention is more effective, cheaper and less painful than treatment for every chronic disease, including Alzheimer's disease. This talk will focus on the lifestyle factors that are known to contribute to AD and give simple to implement solutions that can prevent AD and other chronic diseases.

POSTERS

1. Discovering Cytome Biomarkers of Alzheimer's Disease Risk in Isolated Buccal Cells Using Laser Scanning Cytometry

[1,2]Maxime François, [1]Wayne R Leifert, & [1]Michael Fenech.

[1]CSIRO Food and Nutritional Sciences, Adelaide, SA and [2]Centre of Excellence in Alzheimer's Disease Research and Care, Edith Cowan University, Joondalup, WA.

2. Self-rated memory and its association with socio-economic and health parameters

Syed Ziaur Rahman, Anton Rahmadi, Sanja Lujic, Louisa Jorm, Phillipa Hay, Gerald Münch

School of Medicine, University of Western Sydney, NSW, Australia.

3. Dietary patterns and cognitive functioning and change in community-dwelling older adults

Vanessa Danthiir[1], Nick Burns[2], Carlene Wilson[3], Ted Nettelbeck[2], Gary Wittert[4]

[1]CSIRO Food and Nutritional Sciences, Adelaide, Australia, [2]School of Psychology, University of Adelaide, [3]Flinders University, Adelaide, [4]School of Medicine, University of Adelaide.

4. Physical activity is associated with hippocampal volume in an elderly population

Belinda Brown[1,2,3], Jeremiah Peiffer[4], Veer Gupta[2,3], Kevin Taddei[1,2,3], Pierrick Bourgeat[5], Olivier Salvado[5], Victor Villemagne[6], Stephanie Rainey-Smith[2,3], Kathryn Ellis[7,8,12], David Ames[8,12], Christopher Rowe[6], Colin Masters[7,10], Cassandra Szoeke[11], Ralph Martins[1,2,3] and the AIBL Research Group

1. School of Psychiatry and Clinical Neurosciences, University of Western Australia, Perth, Australia 2. School of Exercise Biomedical and Health Sciences, Edith Cowan University, Joondalup, Australia 3. McCusker Alzheimer's Research Foundation, Nedlands, Australia 4. School of Chiropractic and Sport Science, Murdoch University, Perth, Australia 5. CSIRO Preventative Health, Brisbane, Australia 6. Austin Health, Heidelberg, Australia 7. Mental Health Research Institute, The University of Melbourne, Parkville, Australia 8. Dept of Psychiatry, University of Melbourne, Australia, 9. Academic Unit for Psychiatry of Old Age, Department of Psychiatry, St. Vincent's Aged Psychiatry Service, St George's Hospital, Melbourne, Australia 10. Centre for Neuroscience, The University of Melbourne, Parkville, Australia 11. CSIRO, Parkville, Australia 12 .National Ageing Research Institute, Parkville, Victoria, Australia

5. An Australian-type rodent diet and cognitive decline: Is there a benefit to nutrient supplementation?

S. Brooker, C. McIver, G. Patten, M. Fenech

CSIRO, Adelaide, SA

6. Lifetime dietary patterns and longitudinal cognitive outcomes in an older Australian population

D Hosking [1, 2] V Danthiir [1, 2]

[1] CSIRO Human Nutrition PO Box 10041 Adelaide S. A, [2] School of Psychology, University of Adelaide, S. A.

7. Evaluation of a dementia-specific nutrition education intervention

Wallace, R & Devine, A.

School of Exercise and Health Science, Edith Cowan University

8. Adherence to a Mediterranean Diet and Alzheimer's disease Risk in an Australian Population

Gardener S[1,2], Gu Y[3,4], Rainey-Smith SR[1,2], Keogh JB[6], Clifton PM[14,15], Mathieson SL[2], Taddei K[1,2], Mondal A[1,2], Ward VK[1,2], Scarmeas N[3,4,5], Barnes M[9], Ellis KA[7,8,11], Head R[9], Masters CL[7,16], Ames D[8,11], Macaulay SL[12], Rowe CC[10], C. Szoeke[12], Martins RN[1,2], For the AIBL Research Group[13].

[1]Centre of Excellence for Alzheimer's Disease Research & Care, School of Medical Sciences, Edith Cowan University, Joondalup, Western Australia, Australia. [2]Sir James McCusker Alzheimer's Disease Research Unit (Hollywood Private Hospital), Perth, Western Australia, Australia. [3]Taub Institute for Research of Alzheimer's Disease and the Ageing Brain, Columbia University, New York, NY, USA. [4]Gertrude H. Sergievsky Centre, Columbia University, New York, NY, USA. [5]Department of Neurology, Columbia University College of Physicians and Surgeons, New York, NY, USA. [6]School of Pharmacy & Medical Sciences & Sansom Institute for Health Research, Division of Health Sciences, University of South Australia, Adelaide, Australia. [7]Mental Health Research Institute, The University of Melbourne, Parkville, Victoria, Australia. [8]National Ageing Research Institute, Parkville, Victoria, Australia. [9]CSIRO, Preventative Health Flagship, Adelaide, Australia. [10]Department of Nuclear Medicine & Centre for PET, Austin Health, Heidelberg, Victoria, Australia. [11]Academic Unit for Psychiatry of Old Age, Department of Psychiatry, The University of Melbourne, St. Vincent's Aged Psychiatry Service, St George's Hospital, Victoria Australia. [12]CSIRO Preventative Health Flagship, CMSE Parkville, Victoria, Australia. [13]http://www.aibl.csiro.au (for the AIBL Research Group). [14]Department of Medicine and Department of Biomedical Science, University of Adelaide. [15]Nutritional Interventions, Baker IDI Heart and Diabetes Institute. [16]Centre for Neuroscience, The University of Melbourne, Parkville, Victoria, Australia.

9. Natural inhibitors of the Kynurenine pathway: Novel applications in Alzheimer's disease treatment

[1]Gloria Castellano Gonzalez, [2]Helder Marcal, [1]Alban Bessede, [1,2]Gilles J. Guillemin

[1]Neuroinflammation Group, School of Medical Sciences, Faculty of Medicine, University of New South Wales, Sydney, Australia. [2]Traditional Medicine Research Group, School of medical Sciences, Faculty of Medicine, University of New South Wales, Sydney, Australia

AN AUSTRALIAN-TYPE RODENT DIET AND COGNITIVE DECLINE: IS THERE A BENEFIT TO NUTRIENT SUPPLEMENTATION?

S. Brooker, C. McIver, G. Patten, M. Fenech

CSIRO, Adelaide, SA

Alzheimer's disease (AD) is the third largest cause of death in Australia. Lifestyle factors, such as diet have been demonstrated to modify risk for developing AD. A diet that is high in polyphenolic compounds, B vitamins, and ω-3 fatty acids has been associated with a decreased risk for AD, when compared to a diet that is high in saturated fats. The purpose of this research was to investigate the effect of an Australian-type diet (Oz-AIN diet), with and without nutritional supplementation, on cognitive decline in a mouse model of AD.

Macro- and micro- nutrient content of an ideal rodent diet (AIN93-M) were adjusted to represent the nutrient intake of middle-aged Australian women, a population who are at high risk for developing AD. The resulting diet (Oz-AIN diet) was 35 % fat, 47 % carbohydrate, 17 % protein (kJ/g).

Mice pre-disposed to develop AD were fed the Oz-AIN diet, with or without supplements of polyphenolic compounds, B vitamins, and ω-3 fatty acids, for 15 months. Cognitive decline was assessed by time taken (sec) to find a hidden platform in the Morris Water Maze (MWM).

AD mice that were fed Oz-AIN diet without nutritional supplements performed significantly worse in the MWM at 15 months (38.9 ±6.14 sec) when compared to those fed either the nutrient supplemented (32.43 ±5.73 sec) or control (31.53 ±9.05 sec) diet ($p < 0.05$).

This study demonstrates that supplementing an Australian-type diet, which is high in fat and has nutrient intakes that deviate from recommended levels, with B vitamins, ω-3 fatty acids and polyphenolic compounds reduced the rate of cognitive decline in a mouse model of AD.

LIFETIME DIETARY PATTERNS AND LONGITUDINAL COGNITIVE OUTCOMES IN AN OLDER AUSTRALIAN POPULATION

D Hosking [1, 2] **V Danthiir** [1, 2]

[1] *CSIRO Human Nutrition, Adelaide S. A.* [2] *School of Psychology, University of Adelaide, S. A*

Background: Dietary pattern research is a promising approach to identify potential dietary influences on cognitive decline and dementia. The aetiology of cognitive decline suggests that past dietary intake may be relevant to cognitive status in later life; therefore relationships between lifetime dietary patterns and later life cognitive outcomes are of interest.

Methods: A sample of 352 community-dwelling older adults (Females = 189) completed a lifetime diet questionnaire. Ages ranged from 65 to 91 years (M = 73.15, SD = 5.48) with a mean of 13 years of education (SD = 5.7). Participants undertook an extensive battery of computerised and pencil and paper cognitive tasks at four time points over 18 months. The cognitive constructs are invariant over time, as assessed by confirmatory factor analytic models. Exploratory factor analysis was performed on each of the life periods from the Lifetime Diet Questionnaire. Factor scores for each of the dietary patterns were used as predictor variables in regression models, with scores from the cognitive factors at time-point four as the outcome variables. Cognitive outcomes at time-point one were controlled for to investigate whether the dietary variables predicted cognitive change over time. Other covariates in the models included current diet, age, sex, education and smoking status.

Results: Dietary patterns from childhood, early adulthood, adulthood and middle age were associated with changes in cognitive outcomes over 18 months after controlling for demographic variables known to influence both dietary intake and older-age cognition.

Conclusion: Dietary patterns across the lifetime predicted longitudinal cognitive outcomes in a non-clinical older population. This finding supports taking a life course approach to ameliorating later life decline via earlier life dietary choices.

NATURAL INHIBITORS OF THE KYNURENINE PATHWAY: NOVEL APPLICATIONS IN ALZHEIMER'S DISEASE TREATMENT.

[1]Gloria Castellano Gonzalez, [2]Helder Marcal, [1]Alban Bessede, [1,2]Gilles J. Guillemin

[1]Neuroinflammation Group, School of Medical Sciences, Faculty of Medicine, University of New South Wales, Sydney, Australia. [2]Traditional Medicine Research Group, School of medical Sciences, Faculty of Medicine, University of New South Wales, Sydney, Australia

The major degrading biochemical pathway of tryptophan is the kynurenine pathway (KP), which ultimately leads to production of nicotinamide adenine dinucleotide (NAD+). Some of the metabolites of KP can either be neurotoxic or neuroprotective. For example, quinolinic acid (QUIN), an N-methyl D-aspartate (NMDA) receptor agonist, and 3-Hydroxykynurenine (3-HK), a free radical generator, are both neurotoxic. Conversely, Kynurenic acid (KYNA) is neuroprotective against QUIN-induced neuronal cell-death. The first step of the KP is initiated and regulated by the enzyme Indoleamine 2, 3 dioxygenase (IDO-1), which synthesizes kynurenine (KYN) from tryptophan. The succeeding step is directed by kynurenine 3-Monooxygenase (KMO), which metabolises KYN to 3-HK, and then metabolised to QUIN. Overproduction of neurotoxic KP compounds is known to play a key role in neurodegenerative diseases such as the Alzheimer's disease (AD).

The KP also has major influences on the regulatory mechanisms involved with immune responses. Activation of inflammatory pathways has been shown in brains of AD patients. Microglia and infiltrated macrophages are considered to play a key role on the innate immune inflammatory responses in the CNS. Activated monocytic macrophages secrete a wide range of inflammatory mediating factors, which may impact on neurons survival. While QUIN has shown to activate the pro-inflammatory pathways, KYN has shown to activate an immune tolerance; therefore by modulating KP, the monocytic phenotype can be modulated.

Current research is focussed on IDO-1 inhibition as a therapeutic strategy for neurodegenerative diseases because of its ability to decrease QUIN and 3-HK. Numerous natural compounds have been described as IDO-1 inhibitors; however, IDO-1 inhibition also decreases the neuroprotective metabolite KYNA and its substrate, with immune modulatory activity, KYN. This study screens a large collection of natural polyphenols and flavonoids for their ability to inhibit KMO activity in human derived macrophages and microglia. KMO inhibition is a superior strategy since it will not only decrease the production of neurotoxic metabolites, however, it will also increase the neuroprotective KYNA and favour the production of inmunotolerant products. The best compounds (alone and/or in combination) have been assessed in vitro and in vivo in mouse models for AD and for their abilities to ameliorate the disease progression and/or severity. In conclusion, the identification of a novel combination of natural non-toxic molecules targeting KMO may lead to new therapeutic strategies to treat neurodegenerative diseases.

EVALUATION OF A DEMENTIA-SPECIFIC NUTRITION EDUCATION INTERVENTION

Wallace, R & Devine, A.

School of Exercise and Health Science, Edith Cowan University

Objective: Dementia is a significant and costly health issue currently without a cure or effective treatment. This study piloted a dementia-specific nutrition education intervention (NEI) to community members aimed at increasing knowledge and improving dietary patterns and cooking behaviours which may delay or prevent the onset of dementia.

Methods: Following a free public seminar about the dietary and lifestyle aspects of dementia, attendees were recruited (n=72) to a 4-week NEI comprising of education sessions, food preparation and meal sharing. Impact and outcome evaluation were assessed from a questionnaire administered at baseline, post-NEI and 3-8 months later. Results from 45 participants were available for analysis and qualitative findings were determined from four focus group sessions (n=19).

Results: After the 4-week NEI participants knew more about dementia 27(IQR: 23-31) vs. 20.0(IQR: 15-22), T=1697.50, z= -6.136, p<0.001), consumed a greater variety of fruit (T=50.0, z = -3.777, p = 0.0001) and amount (T=72.0, z = -3.354, p = 0.001), used less salt (T=72.5, z= -3.358 p = 0.001) and increased herb (T = 122.5, z = -3.997, p = 0.001) and spice use (T=96.0, z = -2.418, p = 0.016). At the outcome evaluation point 3-8 months later, participants demonstrated that they had maintained this increased level of knowledge about dementia and favourable dietary patterns, as no significant changes were reported. Descriptive qualitative data support these findings.

Conclusion: The NEI had a medium to large effect on favourable dietary patterns, cooking behaviours and knowledge about dementia which may delay or prevent the onset of dementia and be beneficial to overall health. These changes were maintained in the longer term (3-8 months post-NEI), thus highlighting the need for further dementia specific NEI's to be delivered within a wider community setting.

ADHERENCE TO A MEDITERRANEAN DIET AND ALZHEIMER'S DISEASE RISK IN AN AUSTRALIAN POPULATION

Gardener S[1,2], Gu Y[3,4], Rainey-Smith SR[1,2], Keogh JB[6], Clifton PM[14,15], Mathieson SL[2], Taddei K[1,2,] Mondal A[1,2], Ward VK[1,2], Scarmeas N[3,4,5], Barnes M[9], Ellis KA[7,8,11], Head R[9], Masters CL[7,16], Ames D[8,11], Macaulay SL[12], Rowe CC[10], C. Szoeke[12], Martins RN[1,2], For the AIBL Research Group[13].

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Background: The focus of the current research climate is shifting from understanding the pathology of Alzheimer's disease (AD) and its diagnosis to primary prevention and intervention strategies. Early detection combined with intervention strategies could reduce disease effects. Diet represents one potential intervention strategy accessible to all. The Mediterranean diet (MeDi), due to its correlation with a low morbidity and mortality for many chronic diseases, has been widely recognised as a healthy eating model. In fact, recent reports suggest that adherence to the MeDi may affect not only the risk of AD, but also of pre-dementia syndromes and their progression to overt dementia. However, the investigation of dietary factors, AD risk and disease course, is a relatively young field of research and there is a critical need for data collected from a well-characterised ageing cohort.

Methods: Our work reports data collected from the Australian Imaging, Biomarkers and Lifestyle (AIBL) Study of Ageing; a cohort that has garnered international attention. Food frequency questionnaires were used to evaluate the intake of foods and beverages including components reported as potentially beneficial for AD and cognition. A MeDi score was generated for each participant (0-9 point scale): higher scores indicate higher adherence, and scores were compared between the AIBL classification groups.

Results: Compared with healthy control subjects, subjects with AD had lower MeDi Scores ($p < 0.001$); and compared with healthy control subjects, subjects with MCI had lower MeDi Scores ($p < 0.05$). MCI compared with AD lacks statistical significance.

Conclusion: Our analysis suggests that lower adherence to the MeDi is associated with MCI and AD. This is consistent with previous studies. In the Australian AIBL cohort, both MCI and AD patients have a lower adherence to the MeDi compared with healthy controls. We intend to investigate conversion rates from HC to MCI and AD in a longitudinal analysis.

McCusker Alzheimer's Research Foundation Conference & Charity Dinner

7pm cocktails for a 7:30pm start

Bus departing the Hyatt at 6:50pm
Bus returning to the Hyatt at
11:15pm and 11:45pm

You will be joining the Foundation, distinguished guests and internationally renowned clinicians and researchers visiting Australia, for an evening of fine food, wine and entertainment at Perth's iconic WA Italian Club.

217-225 Fitzgerald Street, West Perth

We welcome renowned businesswoman & patron of the arts Mrs. Janet Holmes à Court as our distinguished guest speaker. Live musical entertainment will be provided courtesy of the 'Musicantes'.

Attire: Lounge Suit


SUNDOWNER BBQ
PLEASE JOIN US FOR A RELAXING EVENING AT MATILDA BAY RESERVE, OVERLOOKING PERTH CITY
Saturday, March 31st 5:30pm
Come and enjoy a casual BBQ of sausages & cold beverages and enjoy the opportunity to mingle with fellow researchers and conference delegates.
Buses will depart the Hyatt at 5:15pm to Matilda Bay
Buses will pick up at 7:30pm and 8:30pm to return to the Hyatt.
Supported by the Lions Club of WA volunteers

MAP OF THE HYATT REGENCY

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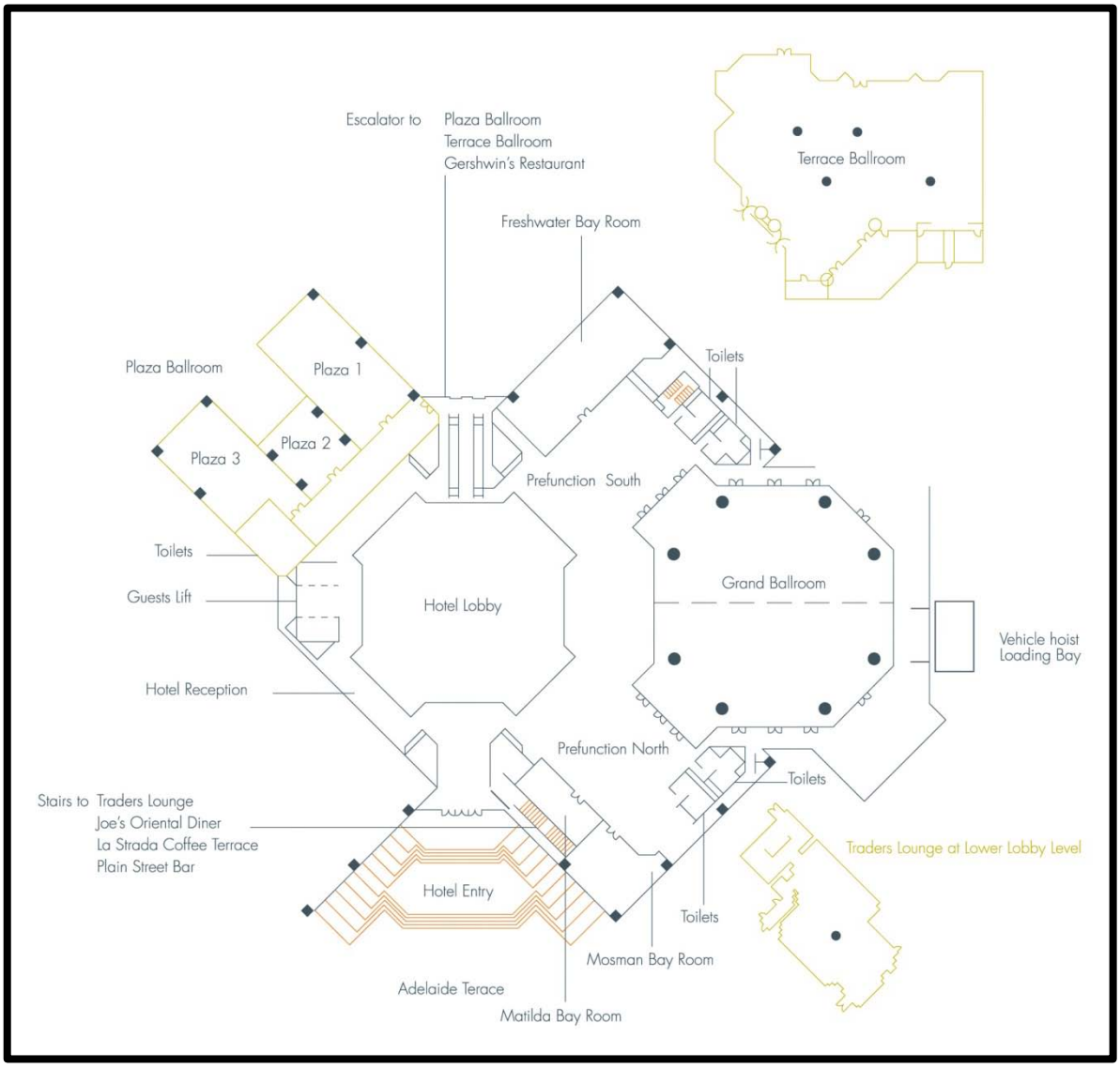

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Attachment C

In brief

Reuters

Milk containers hang from the windows of a passenger train in Ghaziabad on the outskirts of New Delhi, India, last month. According to a survey by India's food safety authority earlier this year, most of the country's milk is diluted, or tainted with products such as bleach and detergent. The survey reportedly found that 70% of samples in urban areas and 30% in rural areas were contaminated.

From the CSIRO Preventative Health National Research Flagship

The AIBL study: throwing light on amyloid-β and Alzheimer's disease

Increasing health costs and lost productivity due to Alzheimer's disease, exacerbated by an ageing population, are a major national and global problem. However, there are signs of light at the end of the tunnel. The Australian Imaging, Biomarkers and Lifestyle Flagship Study of Ageing (AIBL study) is providing new hope for understanding and treatment of Alzheimer's disease.

A key clue to solving the puzzle of Alzheimer's disease was the characterisation of amyloid-β plaques deposited in the brains of patients with Alzheimer's, discovered in the 1980s by one of the present lead AIBL scientists, Colin Masters, and his collaborator Konrad Beyreuther. The hypothesis postulating amyloid-β as the causative agent has placed this peptide as the central target for understanding Alzheimer's disease.

Building on this discovery, the AIBL collaboration between researchers from CSIRO (Commonwealth Scientific and Industrial Research Organisation), the Mental Health Research Institute, University of Melbourne, National Ageing Research Institute, Austin Health and Edith Cowan University (http://www.aibl.csiro.au) has demonstrated the critical significance of amyloid-β in disease development. Christopher Rowe and his team's pivotal and groundbreaking studies using positron emission tomography have shown that amyloid-β plaque is deposited in the brain well before volumetric brain changes can be detected by magnetic resonance imaging and up to 15 years before clinical symptom onset, when damage may be largely irreversible. AIBL research provides hope for blood-based screening tools to facilitate early presymptomatic detection and therapeutic intervention.

There is now renewed interest from the pharmaceutical industry in developing treatments, including antibody therapeutics, for the presymptomatic phase of Alzheimer's disease. CSIRO scientist Jose Varghese and his team, in collaboration with Masters, recently characterised the structure of the amyloid-β peptide at the Australian Synchrotron. It is hoped this will provide clues to inform drug development. In addition, the newly formed Cooperative Research Centre for Mental Health will work with AIBL and CSIRO scientists to help develop diagnostic and therapeutic approaches to delay and treat this insidious disease. The global Research and Standardisation in Alzheimer's Disease Conference was hosted by the AIBL group in March to set standards for development of imaging and biomarker strategies, which should speed up research approaches to diagnostic methods.

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