Australian Imaging, Biomarkers and Lifestyle Study of Ageing Participant Information Day 2019

Kaele Stokes

Executive Director of Consumer Engagement, Policy and Research

Dementia Australia

Friday 21 June 2019

Dementia in Australia

447,115

Australians living with dementia

dementia australia™

Dementia Australia

- Works with individuals and families, all levels of government, and other key stakeholders.
- We are an important representative for those impacted by dementia.
- We provide input on policy matters.
- We collaborate with a range of stakeholders.
- We provide support services, education and information.

dementia australia™

Activity

- Established the Centre for Dementia Learning.
- Memory Walk & Jog – 9 events this year.
- Dementia Action Week.
- Research - $1 million in funding.
- Suite of technology using virtual reality.
- Royal Commission into Aged Care Quality and Safety.
- Federal election campaign.

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Highlights from last financial year

- 22,000 calls to the National Dementia Helpline.
- 28,000 people who attended community education, information and awareness sessions.
- 447,000 Help Sheets downloaded.
- 644,000 visits to the Dementia Australia website.

dementia australia™

Australian Imaging, Biomarkers and Lifestyle Study of Ageing

- One of the most recognised longitudinal study of Alzheimer’s disease.
- Contributes to incredible clinical advances.
- Works towards early diagnosis.

Thank you

dementia australia™

dementia australia™

dementia.org.au

National Dementia Helpline

1800 100 500

For language assistance
call 131 450

AIBL Feedback Day 2019

Emeritus Professor David Ames AO, AIBL Chair

University of Melbourne Academic Unit for Psychiatry of Old Age, National Ageing Research Institute and Florey Institute for Neuroscience and Mental Health

dames@unimelb.edu.au

Running order

- Welcome – Kaele Stokes Dementia Australia
- Introduction - David Ames
- PET Imaging – Chris Rowe
- Blood, CSF Biomarkers & trials – Colin Masters AO
- Cognitive and brain change in the absence of Alzheimer's disease – Krista Dang
- Questions
- Tea

AIBL Publications in peer-reviewed journals to November 2017

- 2007 – 4
- 2008 – 1
- 2009 – 8
- 2010 – 8
- 2011 – 16
- 2012 – 21
- 2013 – 37
- 2014 - 31
- 2015 - 31
- 2016 – 25
- 2017 – 25
- 2018 - 58
- 2019 - TBA
- Total – at least 265

Retinal imaging in Alzheimer's disease

A/Prof Peter van Wijngaarden MBBS PhD FRANZCO
Dr Xavier Hadoux M.Eng M.Sc PhD

CENTRE FOR Eye Research Australia

THE UNIVERSITY OF MELBOURNE

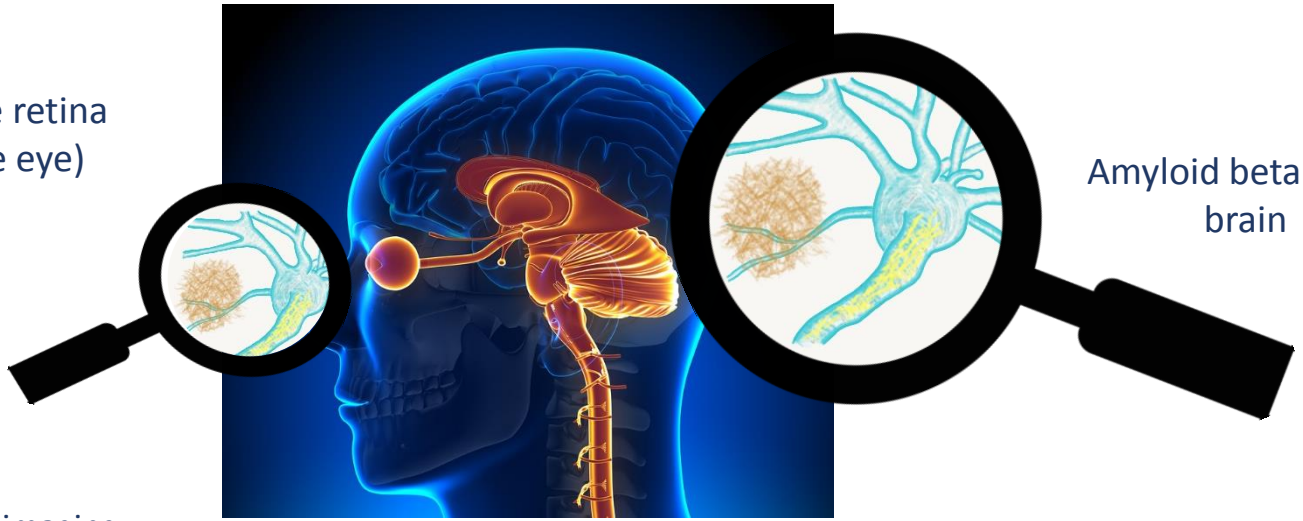
the royal victorian eye and ear hospital

enlighten imaging

The eye is a window to the brain

Amyloid beta accumulates in neural tissue of people with **Alzheimer's disease**
10-20 years before the onset of symptoms

Amyloid beta in the retina
(at the back of the eye)



Amyloid beta in the brain

Advantages of eye imaging

- ✓ Convenient: quick, non-contact, non-invasive
- ✓ Low cost
- ✓ Safe
- ✓ Can be repeated as often as needed

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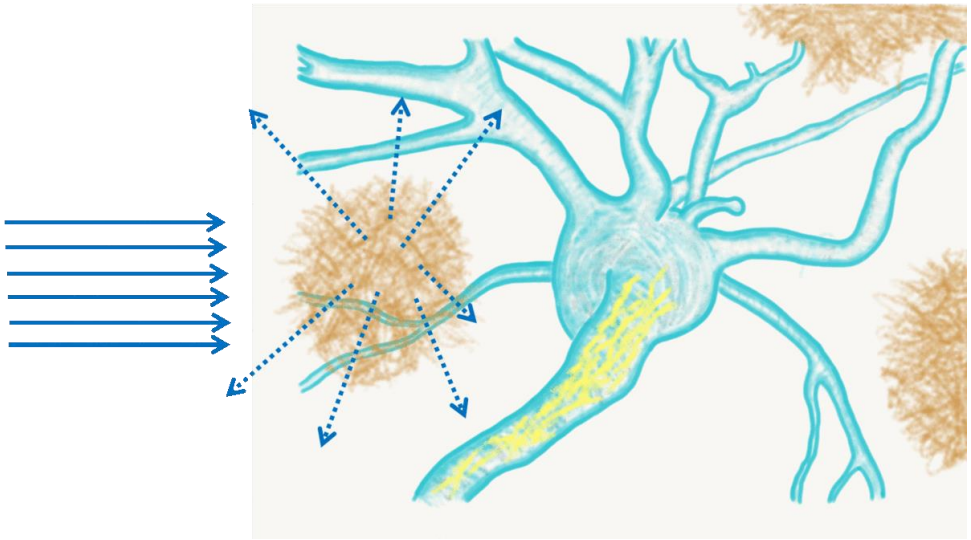
THE UNIVERSITY OF MELBOURNE
the royal victorian eye and ear hospital
enlighten imaging

Amyloid beta scatters light in a characteristic way…



… the scatter varies with different colours of light

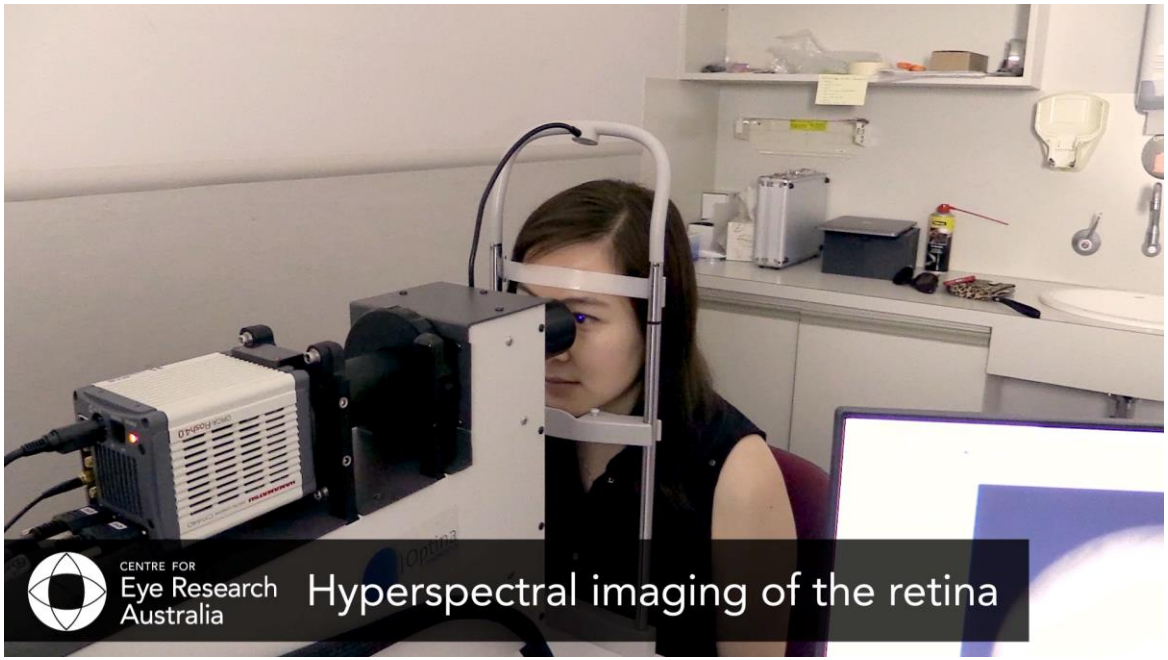
✓ We can use this property as a measure of amyloid beta in the retina
✓ We have shown that the eye imaging signal corresponds with the amount of amyloid beta in the brain

CENTRE FOR Eye Research Australia
THE UNIVERSITY OF MELBOURNE
the royal victorian eye and ear hospital
enlighten imaging

It's as simple as a rainbow-coloured flash

The imaging process is very similar to a standard photo of the eye, but with a **rainbow-coloured flash**

The imaging is performed by our team at the **Royal Victorian Eye and Ear Hospital** in East Melbourne



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NeuroVision

- Another simple eye-test for early detection of Alzheimer's disease
- Investigating whether β amyloid plaques can be identified in the retina at an earlier age before symptoms emerge or in people with MCI
- Correlation between retinal β amyloid and those in the brain
- *Curcumin* allows fluorescence of retinal β amyloid, so it is visible to the camera

NeuroVision Update

- Study closed December 2018
- 145 participants enrolled
  - 2 sessions of retinal imaging
    - one at screening/baseline
    - one after 3 doses of Curcumin and Vitamin E
      - Vit E known to enhance absorption of curcumin
- In the process of data analysis
- Results to follow…

The Australian Imaging Biomarkers and Lifestyle Study of Ageing

Professor Christopher Rowe
Director – Molecular Imaging and Therapy, Austin Health
Director – Australian Dementia Network

The Australian Imaging Biomarkers and Lifestyle Study of Ageing

Commenced 2006

Amyloid PET + MRI with follow-up in 288 of the 1100 original participants.

Now in 75% of 2,335 participants

Biomarkers to assist diagnosis of AD

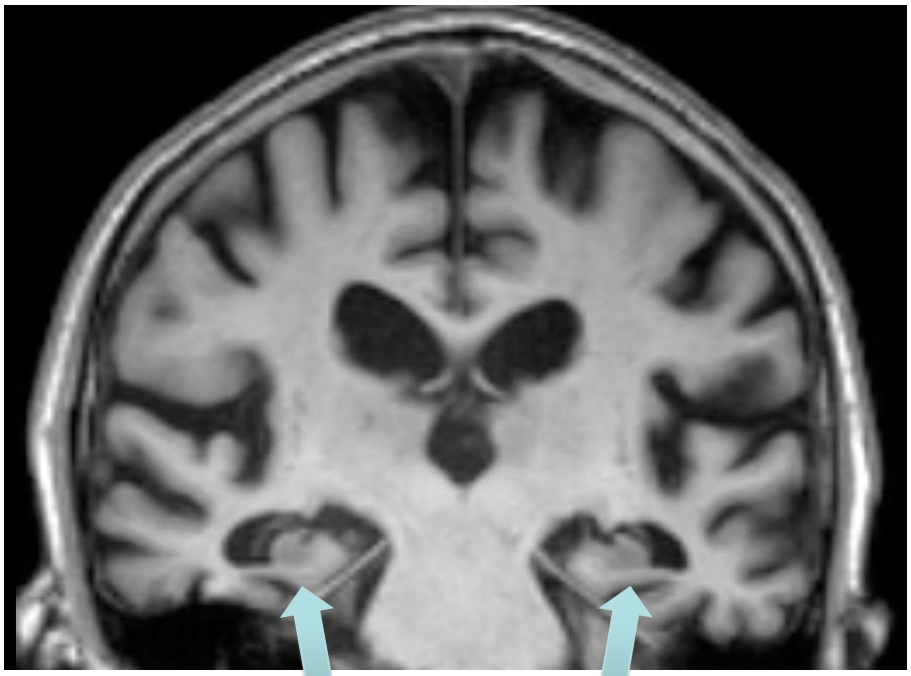
Pathology Markers

- Beta-amyloid imaging with PET
- CSF $A\beta_{42}$ assay

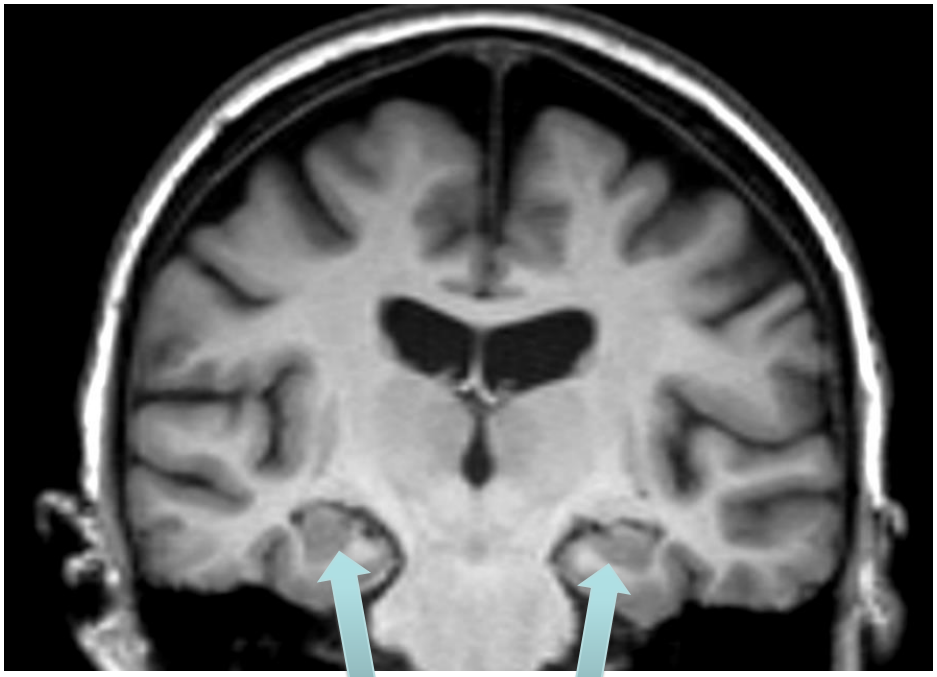
Neuronal damage markers

- MRI
- FDG PET
- CSF Tau assay

Neuronal Injury Biomarkers on MRI: Hippocampal Atrophy

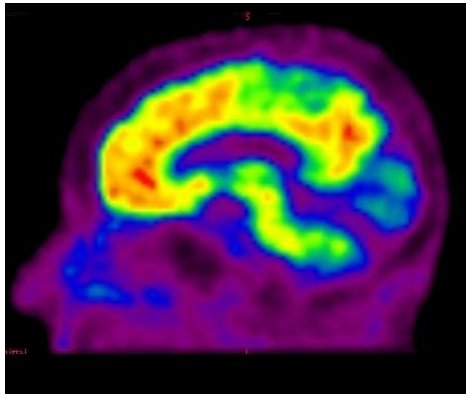


Hippocampal atrophy



Normal hippocampi

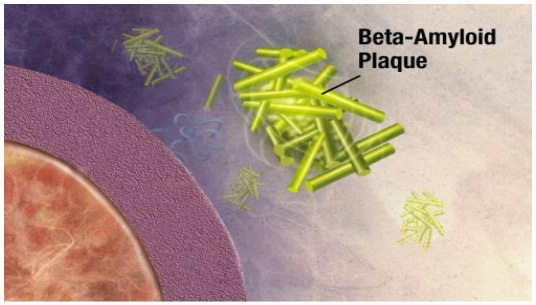
Positron Emission Tomography (PET)

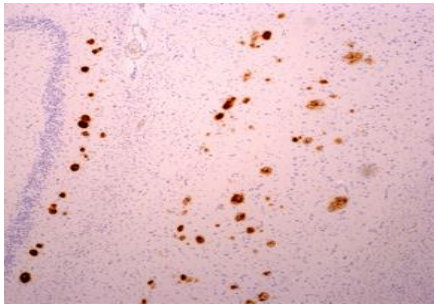


Austin Health

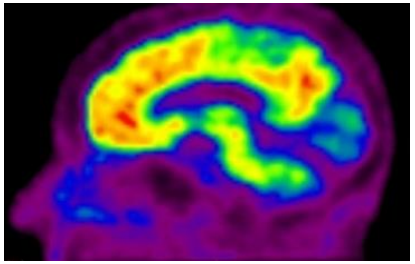
Alzheimer's Pathology

1. Extracellular Beta-amyloid Plaques

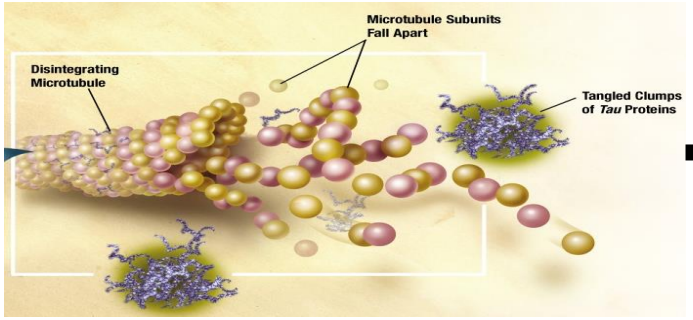


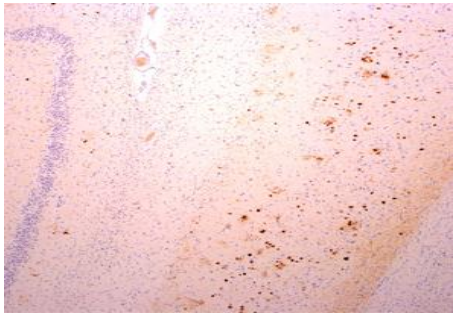


2004

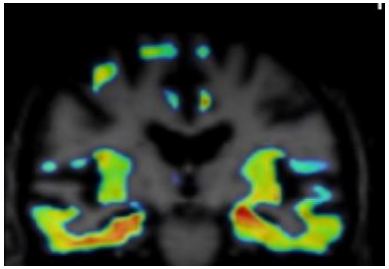


2. Intracellular Neurofibrillary Tangles (tau aggregates)



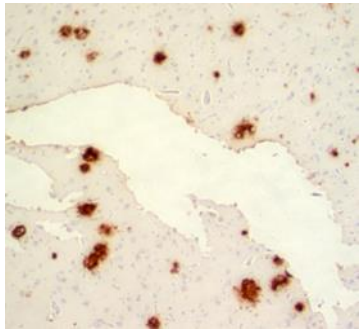


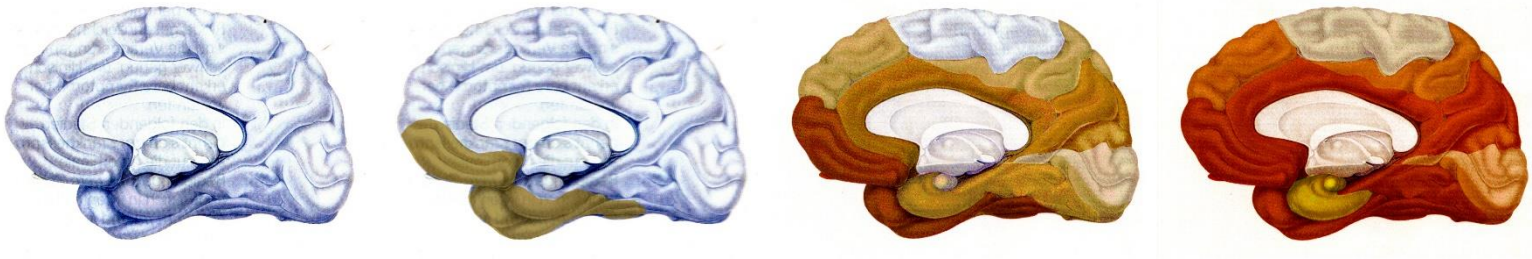
2013



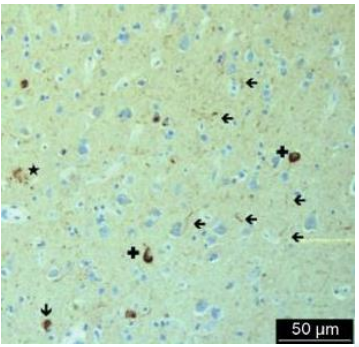
Neuropathology of AD

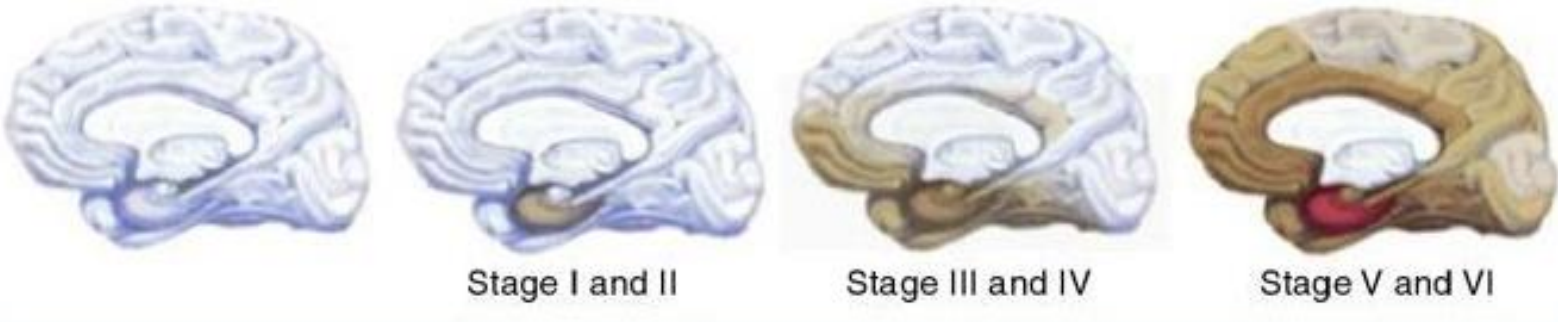
- β-Amyloid





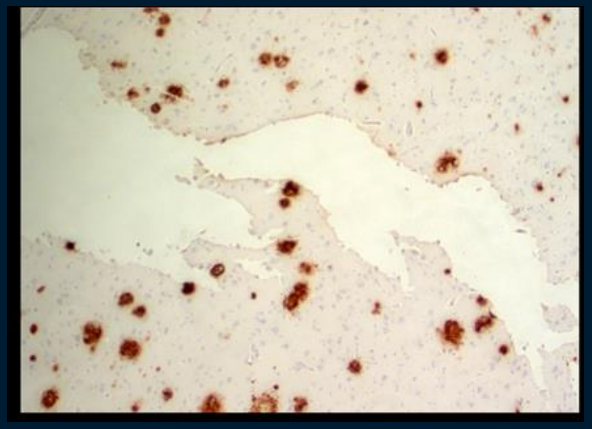
- Phospho-tau tangles

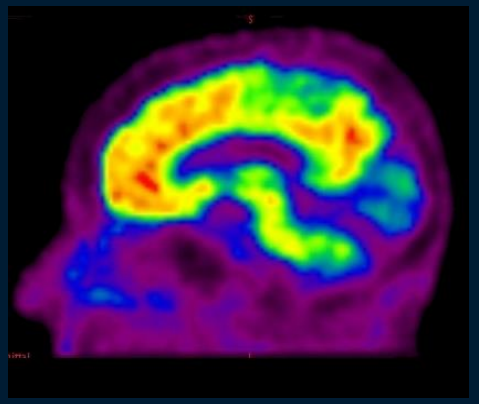




Braak and Braak 1991

Beta-amyloid PET

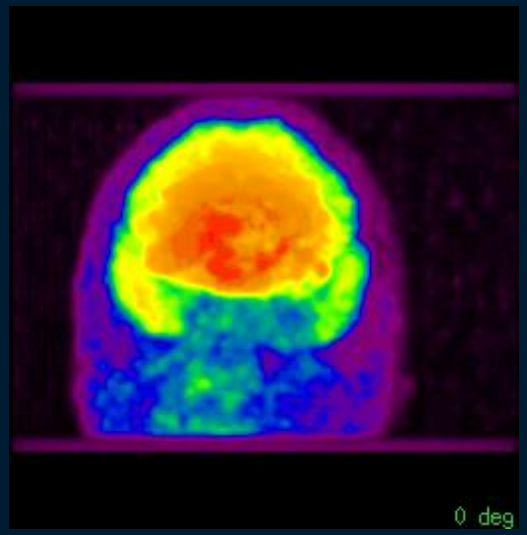




^{11}C-PiB

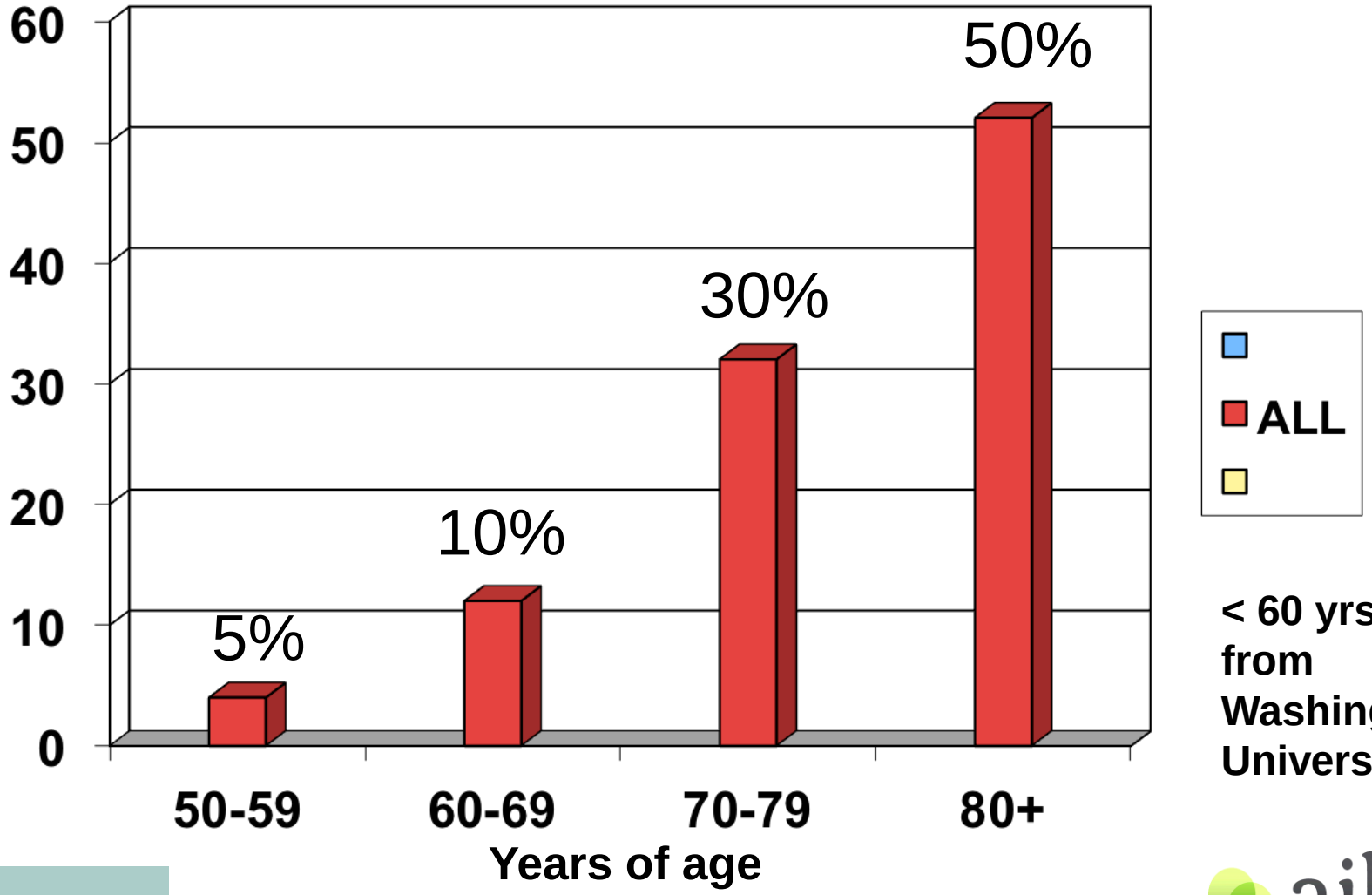
Inventors:
Chet Mathis
William Klunk
University of Pittsburgh

First Publication 2004.



Alzheimer's Disease

% of cognitively healthy population who have plaques



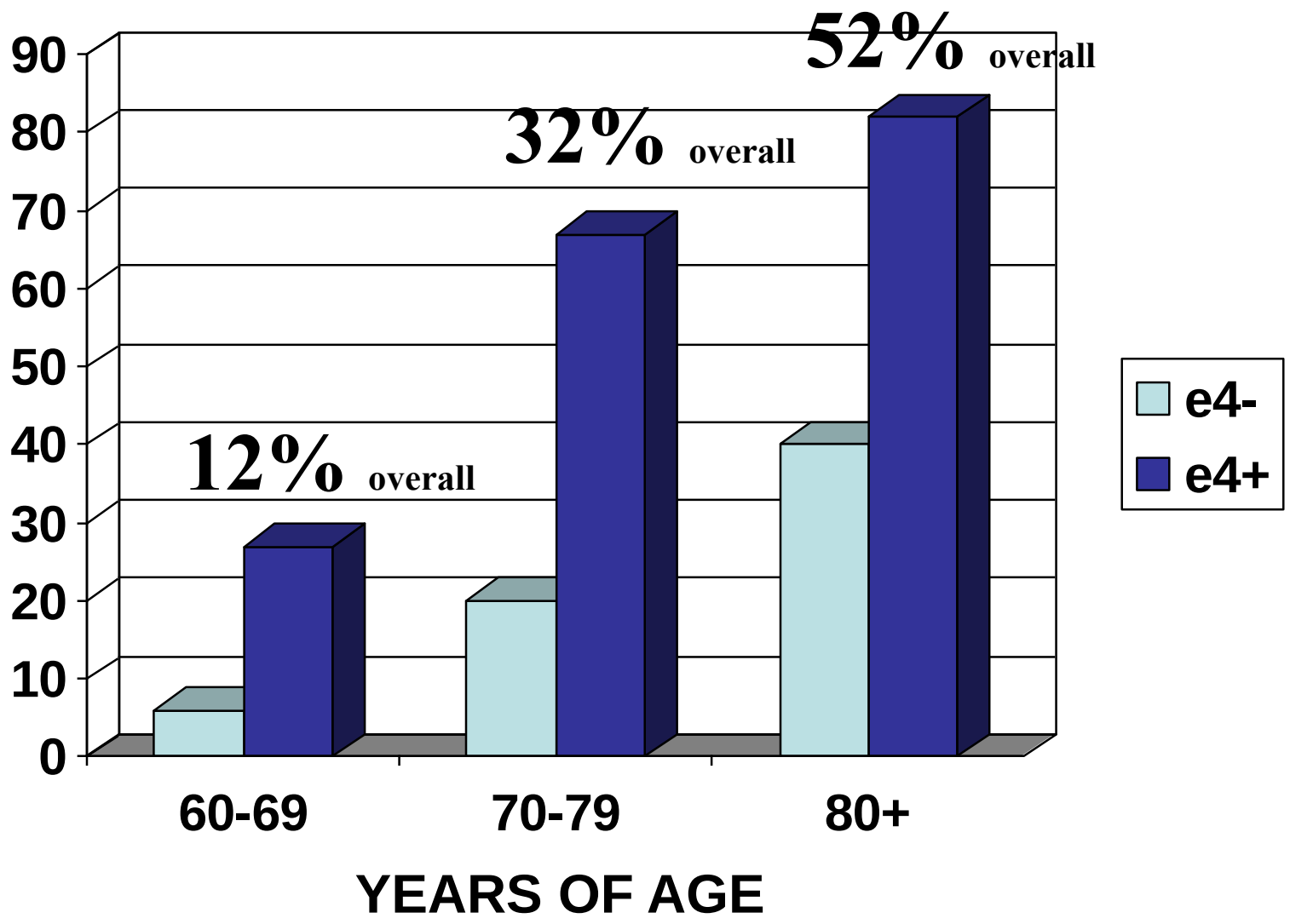
< 60 yrs data from Washington University

Austin Health

aibl

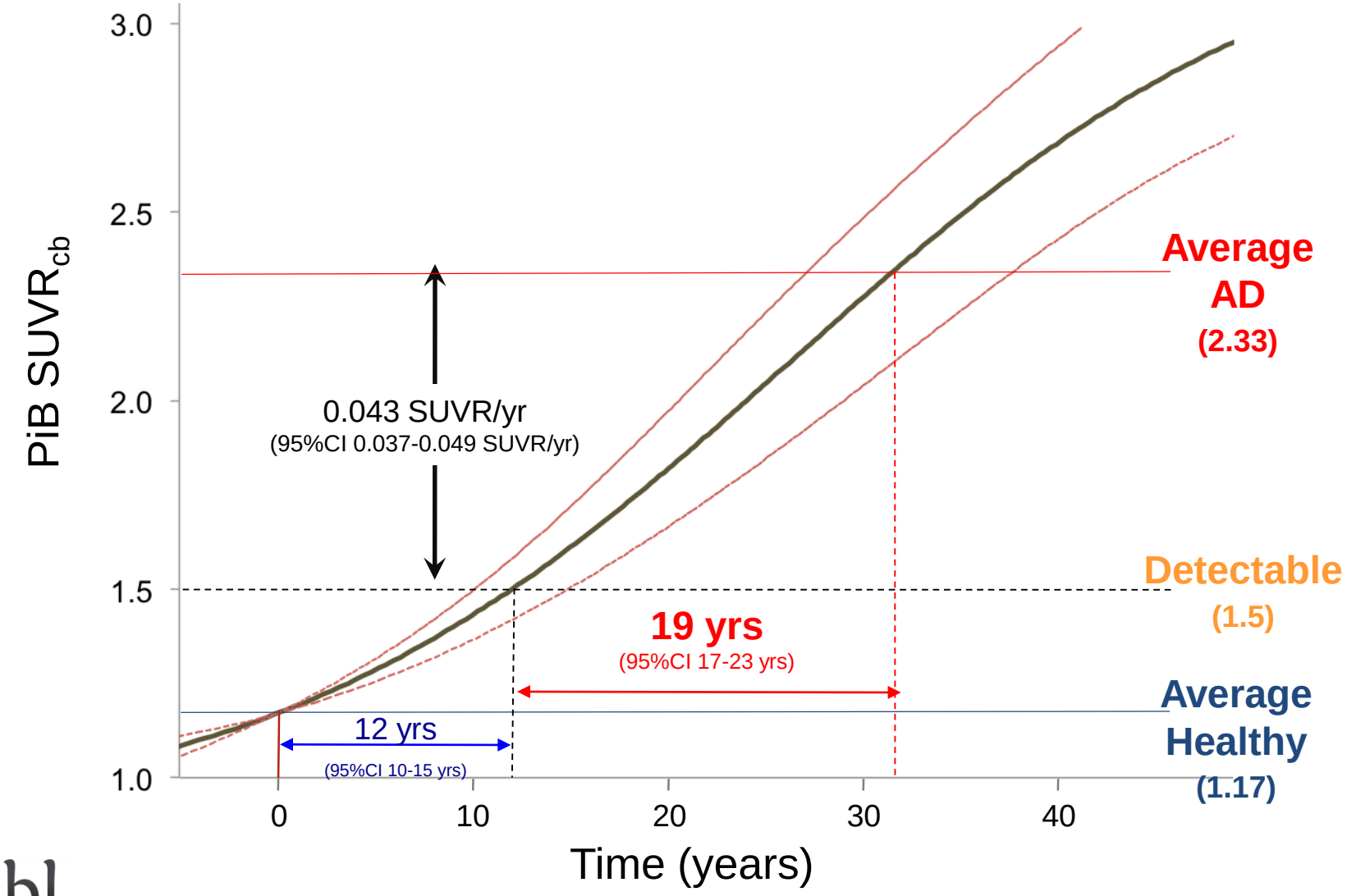
Rowe CC, et al. Neurobiology of Aging. 2010 (680 citations)

% of persons with elevated amyloid as a function of the APO-E gene



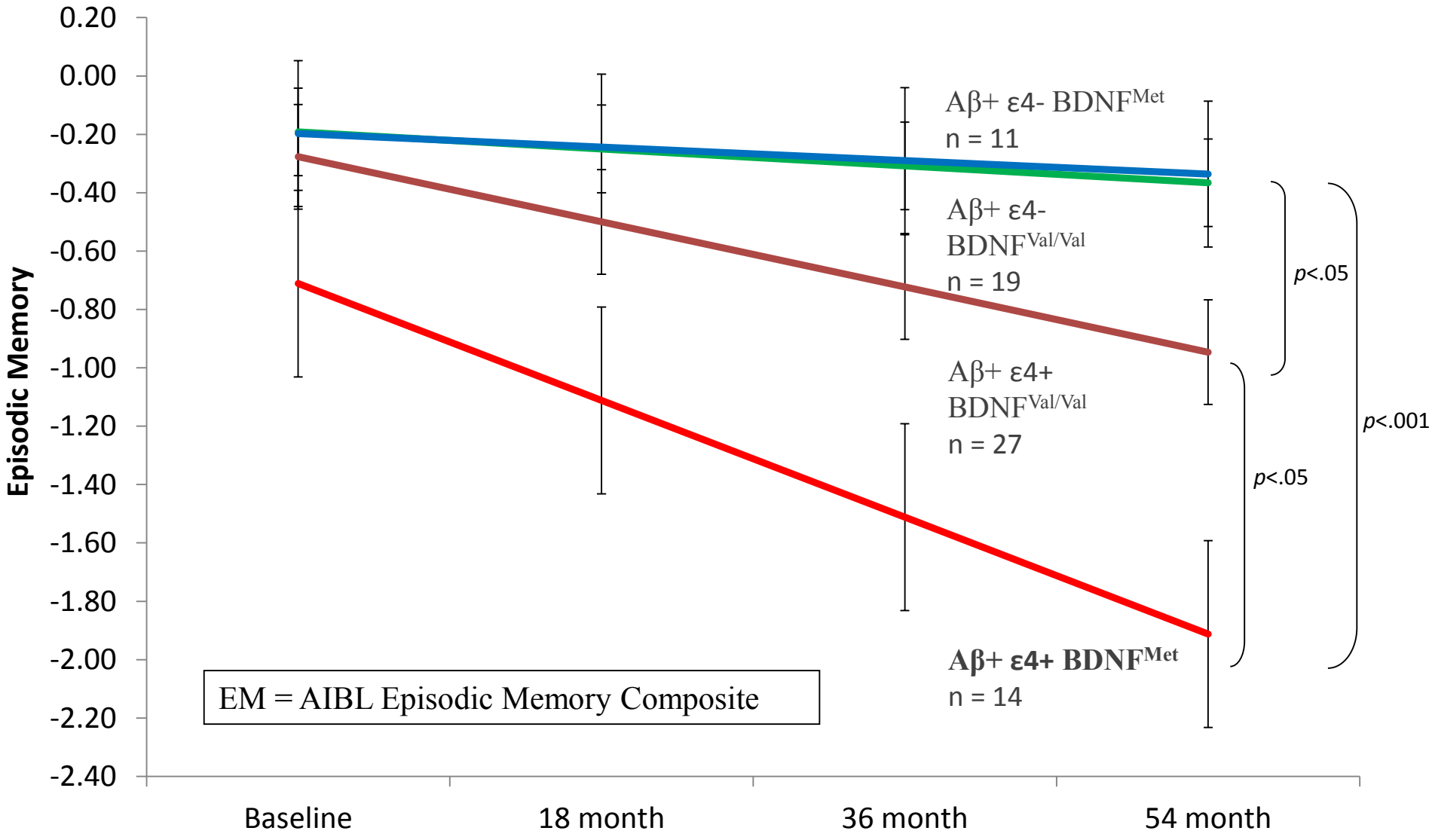
Villemagne VL, et al. 2013 Lancet Neurology
(587 citations)

Aβ deposition in sporadic AD



aibl

Memory decline and amyloid: Effect of *APOE & BDNF*



New diagnostic criteria for Alzheimer's disease

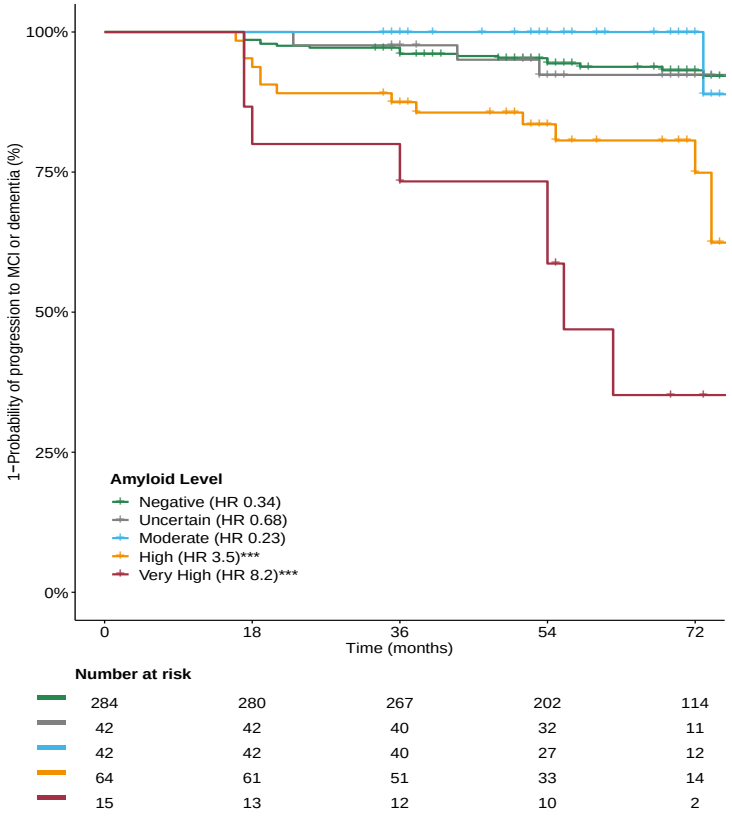
*Dubois B, et al. Lancet Neurology **2014**.*

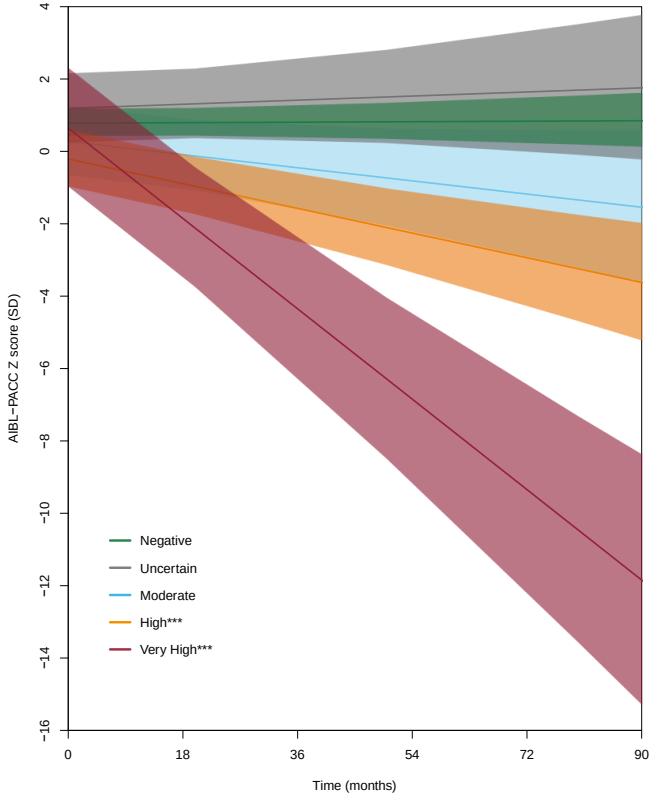
- Progressive memory impairment

plus

- **Positive Aβ PET** or CSF (low $A\beta_{42}$ with high tau)

A little amyloid plaque is not so bad

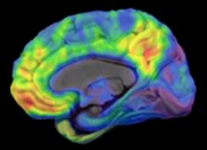




Achievements of Aβ PET imaging

- Improved diagnosis of AD vs FTD
- Earlier diagnosis of AD in MCI phase ("Prodromal" AD)
- Opened a broad window (15 years) for preclinical intervention
- Improved subject selection for therapeutic trials
- Measures effectiveness of anti-Aβ agents
- Permits the study of AD related pathology in non-demented persons and its relationship to genetic and potentially modifiable environmental factors.

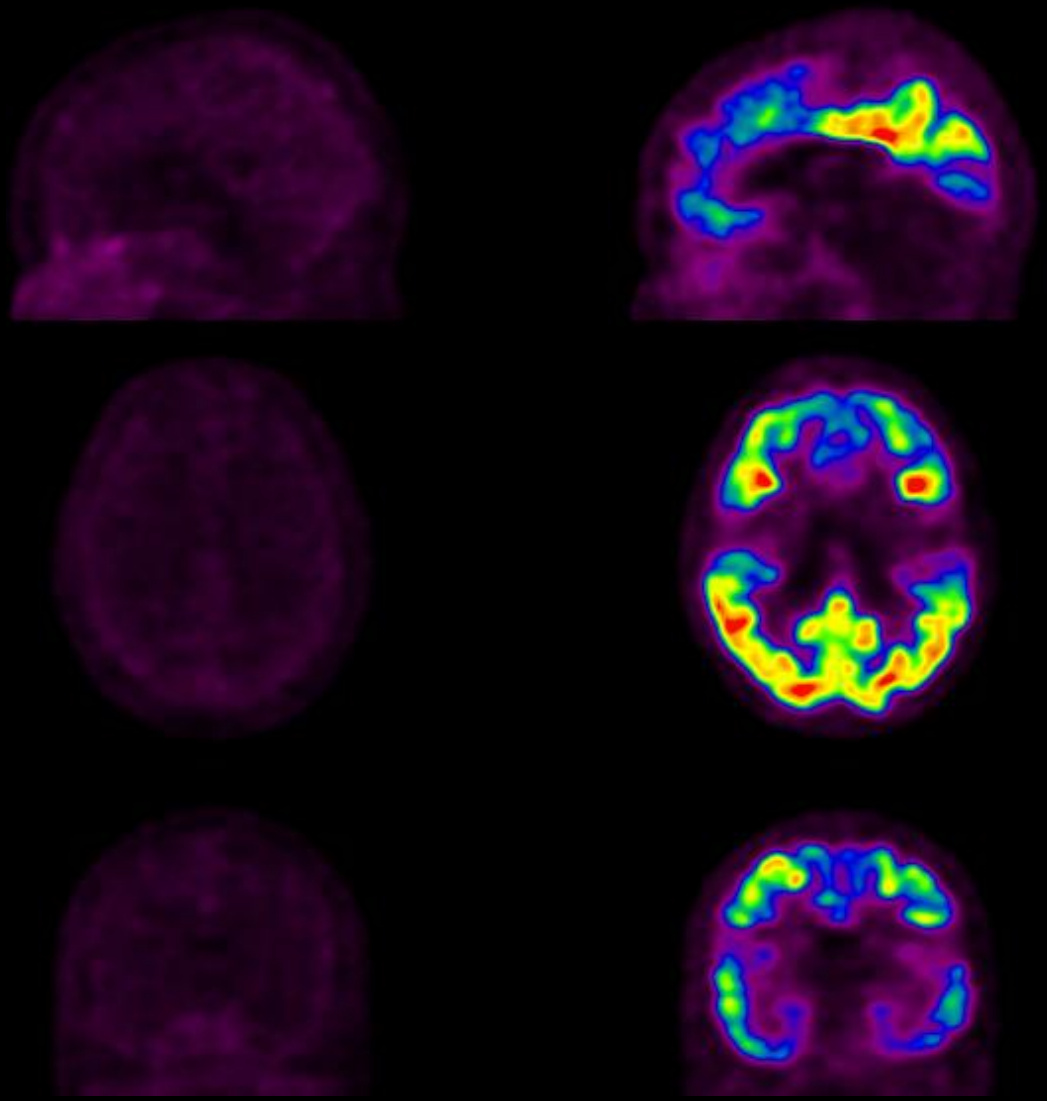
Tau Imaging: ^{18}F-MK6240



AADVaNCe
Australian Ageing and Dementia
Vanguard Neuroimaging Centre

HC

AD



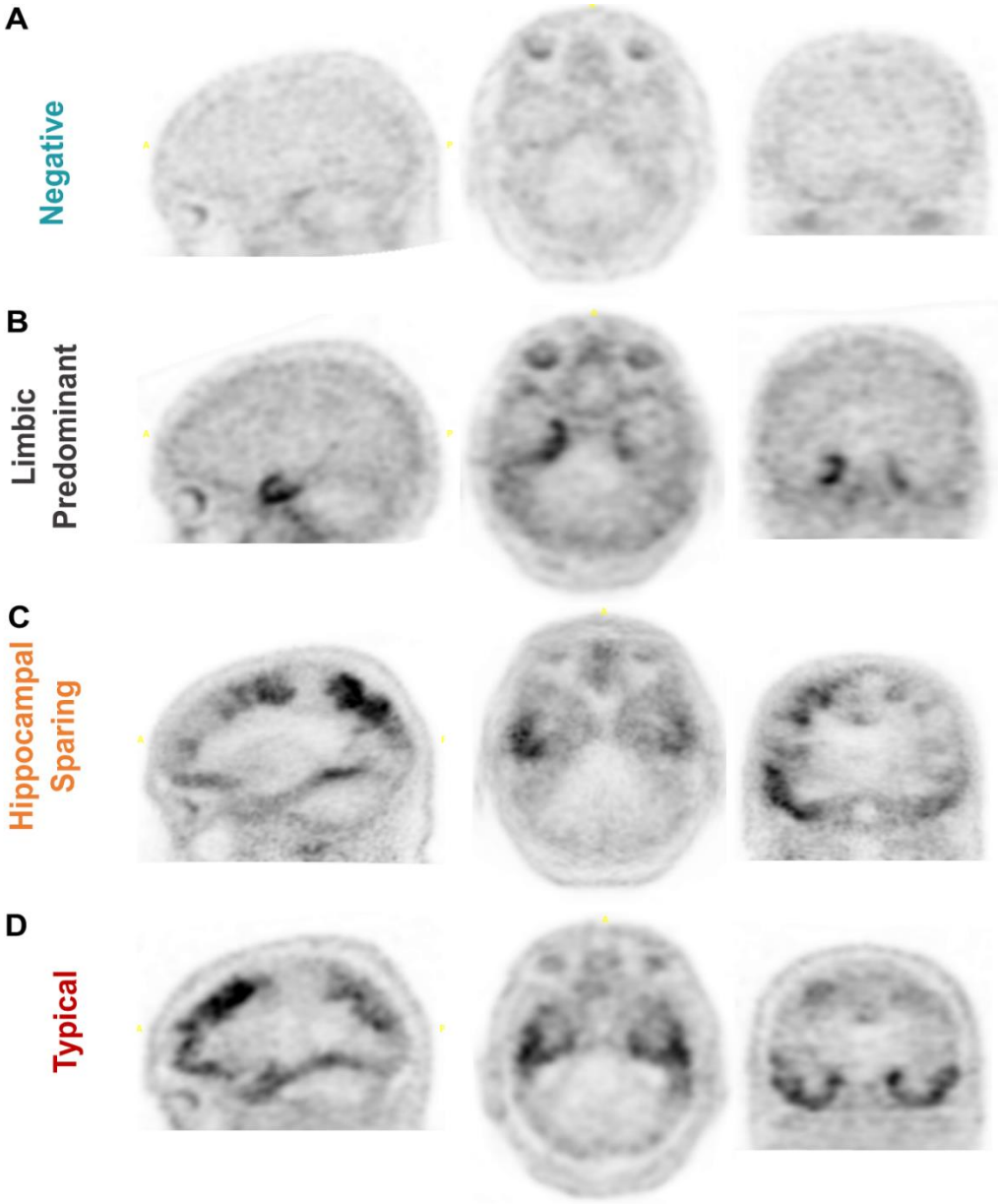
SUVR

7.0

3.5

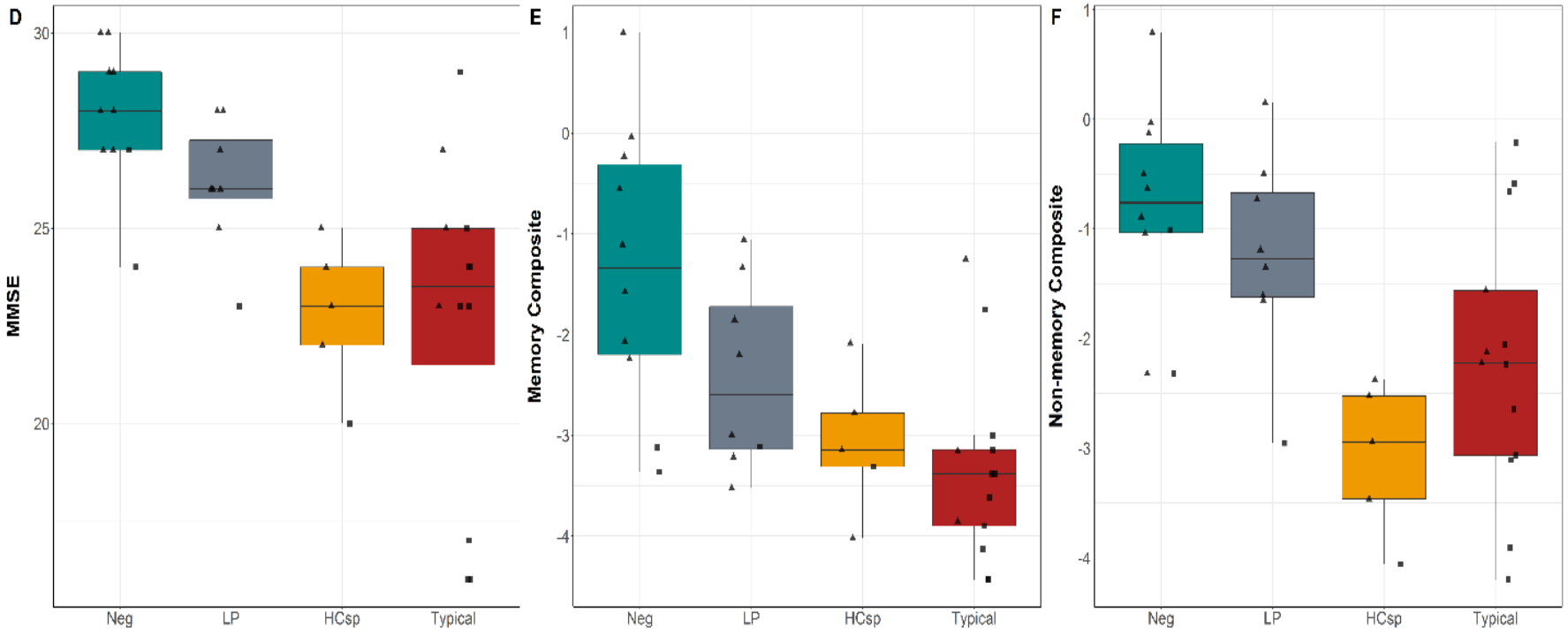
0.0

Austin Health



Patterns of MK-6240

A is an Older Control,
B-D are MCI/early AD



In the MCI/early AD group, the Limbic Predominant had mildly reduced MMSE, moderately reduced memory function and relatively intact non-memory cognitive functions

The Australian Imaging Biomarkers and Lifestyle Flagship Study of Ageing.

4.5 year data release

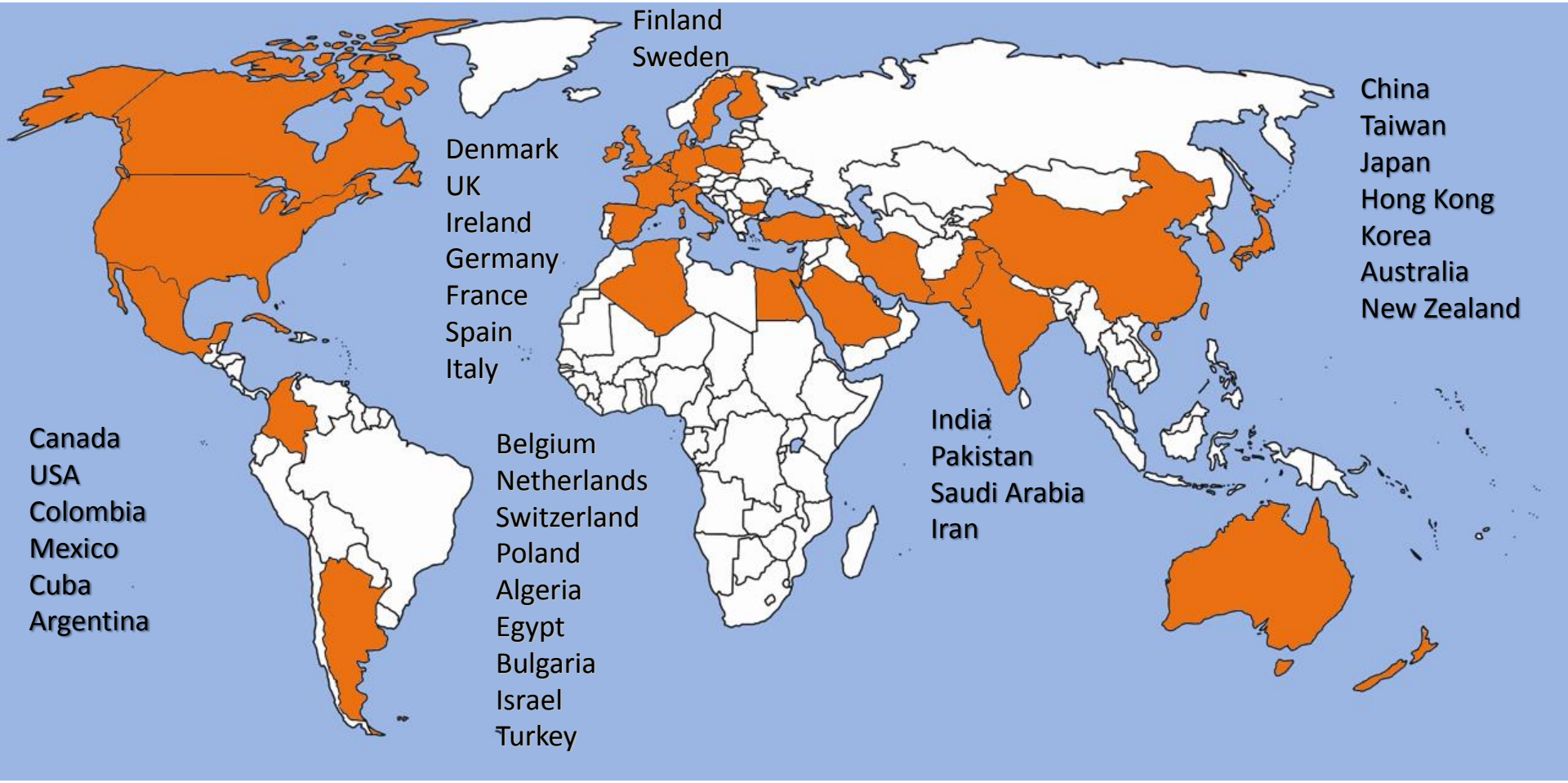
amyloid scan status known in 371 subjects with 4.5 yrs of follow-up and 250 new recruits

www.adni.loni.usc.edu

- Data and Samples
- Access Data

CSIRO · Austin Health · Mental Health Research Institute · NARI · The University of Melbourne · ECU Edith Cowan University

1550 research groups granted access to AIBL@LONI through ADNI website



Includes access granted to the following companies:

Abbott Labs, Abiant, ADM diagnostics, Astra Zeneca, Avid, BioClinica, Biogen Idec, Bristol-Myers Squibb, Cogstate Cytokinetics, Eisai, Elan, Eli Lilly, GE Health Care, General Resonance, Genetech, Imorphics, Iris Biotechnologies, Janssen, Johnson Johnson, M and M Scientific, Merck & Co, Mimvista, Pentara Corp, Pfizer, Philips, Predixion software, Rancho Biosciences, Servier, Siemens, Soft team solutions, UCB, United Biosource Corp

What is ADNeT?

- **A**ustralian **D**ementia **NeT**work = **ADNeT**
- Funded by $18 Million NHMRC-NNIDR (Boosting Dementia Research Grant)
- Network of national dementia research experts

ACKNOWLEDGEMENTS

AIBL would like to thank the study participants and their families

AIBL Study team

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Tania Taddei
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Andrew Watt
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Rob Williams
Bill Wilson
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Paul Yates
Ping Zhang

AIBL is a large collaborative study and a complete list of contributors can be found at www.aibl.csiro.au

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

THE FLOREY INSTITUTE OF NEUROSCIENCE & MENTAL HEALTH
CRC for Mental Health
AUSTRALIA ECU EDITH COWAN UNIVERSITY
Austin Health
McCusker ALZHEIMER'S RESEARCH Foundation
CSIRO

Collaborators

COGSTATE ASSESS · MONITOR · IMPROVE
NARI
THE UNIVERSITY OF MELBOURNE
THE UNIVERSITY OF WESTERN AUSTRALIA
DCRC Dementia Collaborative Research Centres
janssen
Pfizer
Alzheimer's Australia Living with dementia
GE
Navidea BIOPHARMACEUTICALS

The Australian Imaging Biomarkers and Lifestyle Study of Ageing

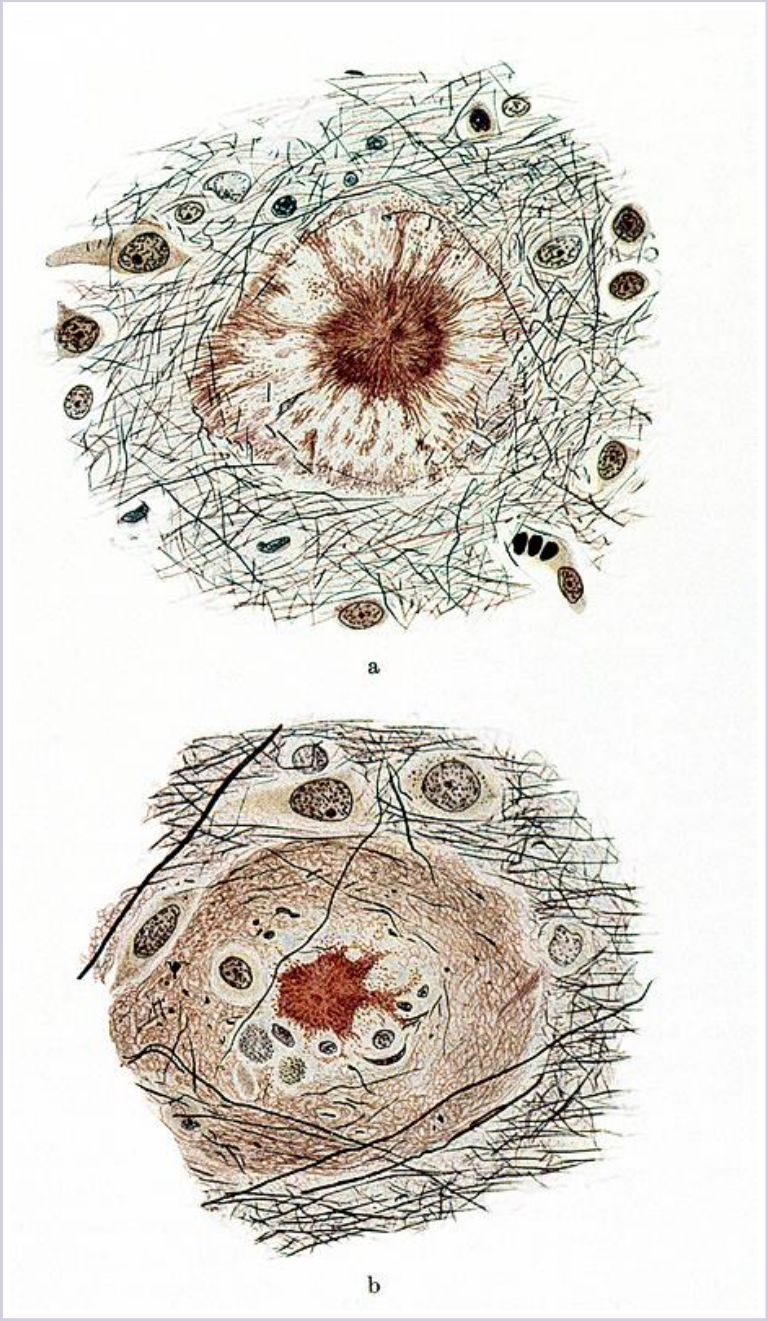
Professor Colin Masters
Laureate Professor of Dementia Research

Florey Institute and The University of Melbourne

Molecular origins of Alzheimer's disease: when does it start, and what strategies for primary prevention?

1. How much Aβ amyloid accumulates in the AD brain?
2. When does it start and how long does it take?
3. How much is the clearance mechanism impaired in sporadic AD?
4. Can quantitative real-time biomarker read-outs be used in clinical trial design to monitor drug efficacy?
5. How to quantitatively define the onset of AD using biomarkers?

The Amyloid Plaque



From W Spielmeyer,
Histopathologie des Nervensystems.
1922

Abb. 201 a und b. Zwei senile Plaques. Bielschowskysche Silberimprägnation. In 201 a gehen von dem amorphen Kerne Strahlen aus, welche wie Kristallnadeln einen hellen Hof durchsetzen und am Rande in einer schmalen, ringartigen Zone ansetzen. In 201 b ist der Kern massiger, ebenfalls amorph, stellenweise etwas ausgezogen. In dem hellen Hof liegen Gliazellen und dürftige Achsenzylinderauftreibungen. Die Außenzone besteht aus einem breiten dichten Wall.

Disclosures

PRANA BIOTECHNOLOGY Limited

(and current consultancies with Prana/Alterity, NeuroBio, Recuerdo and Actinogen)

Two types of Alzheimer's disease

Autosomal/Dominantly Inherited (Early Onset):

Over-production of Aβ

Mean age dementia onset: 45 y

Mutations in APP/PSEN1,2

18% increased production of $[A\beta_{42}]_{CSF}$

Aβ-PET accumulation rates same as sporadic AD (below)

Sporadic (Late Onset):

Failure of Aβ clearance

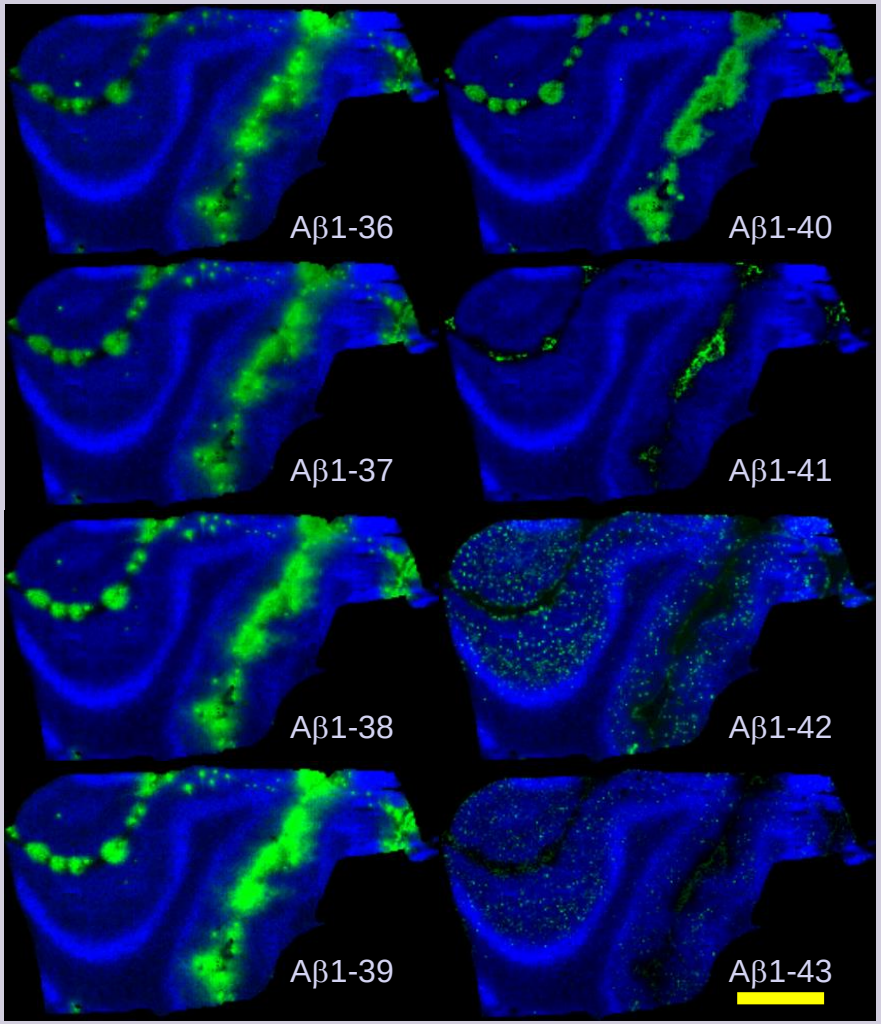
Mean age dementia onset: 78 y ($\varepsilon_4^{+/+}$ 68y, $\varepsilon_4^{+/-}$ 76y, $\varepsilon_4^{-/-}$ 86y)

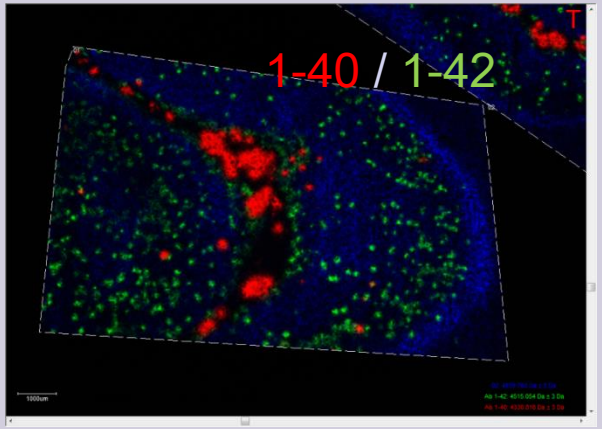
$[A\beta]_{CSF}$: turnover 19h (13h control): 49% slower than control

$T_{1/2}$ 9.4 h (3.8 h young control)

Aβ-PET: accumulation 0.048 SUVR/y; 28 ng/hr

MALDI-MS imaging





Ikegawa/Ihara: 2019

The Australian Imaging, Biomarkers and Lifestyle Study of Aging

(Australian ADNI)

AIBL cohort (now collecting 144 month/12 year data)

Total enrollments: 2502

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

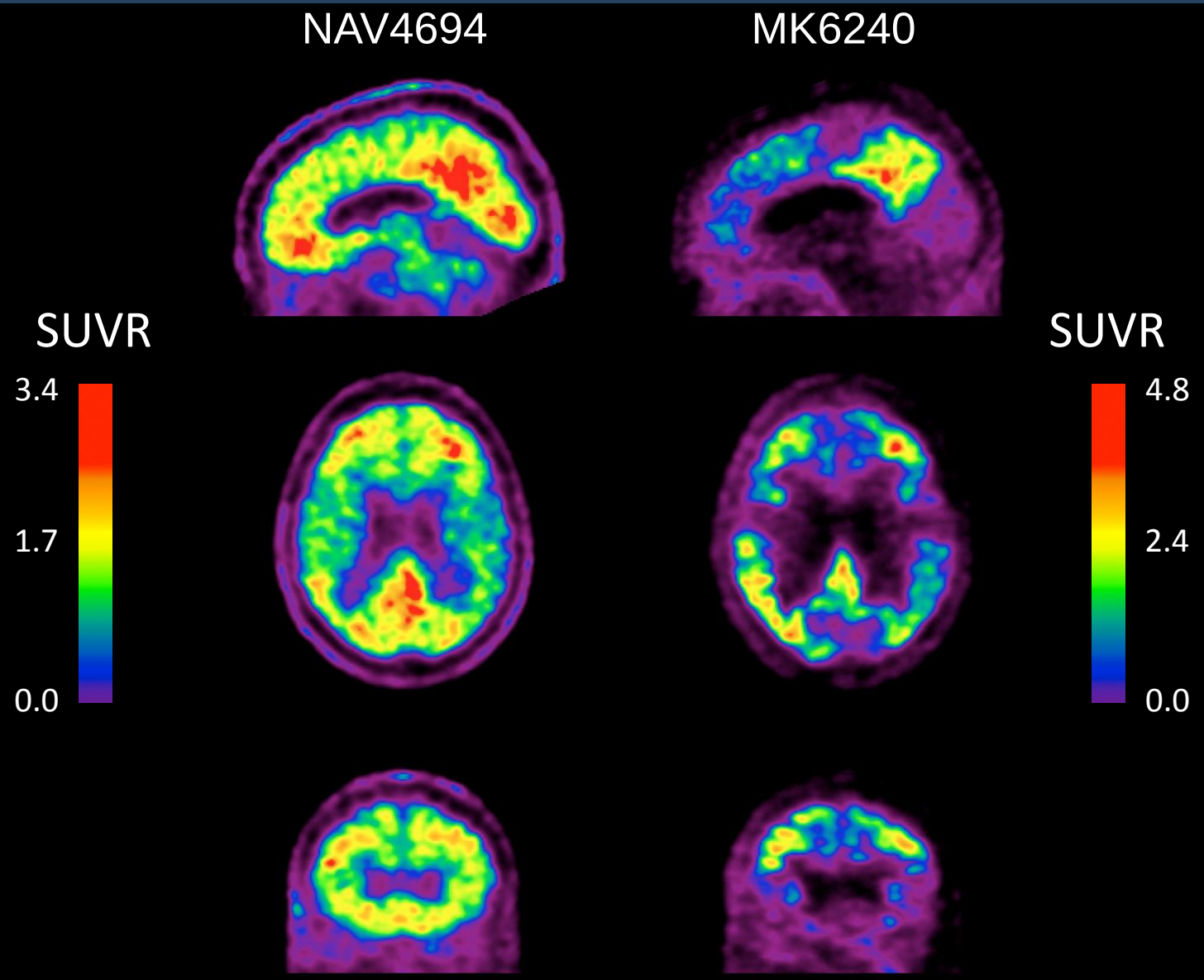
April 2019

Time point (n)					
Baseine (2502)	1488 CN	411 MCI	437 AD	17 Non-AD	149 Excl.
18 month (1582)	1103 CN	182 MCI	292 AD	5 Non-AD	
36 month (1181)	833 CN	111 MCI	227 AD	10 Non-AD	
54 month (910)	688 CN	80 MCI	130 AD	12 Non-AD	
72 month (675)	539 CN	59 MCI	69 AD	8 Non-AD	
90 month (503)	419 CN	34 MCI	44 AD	6 Non-AD	
108 month (439)	377 CN	28 MCI	30 AD	4 Non-AD	
126 month (322)	290 CN	21 MCI	8 AD	3 Non-AD	

Person contact years

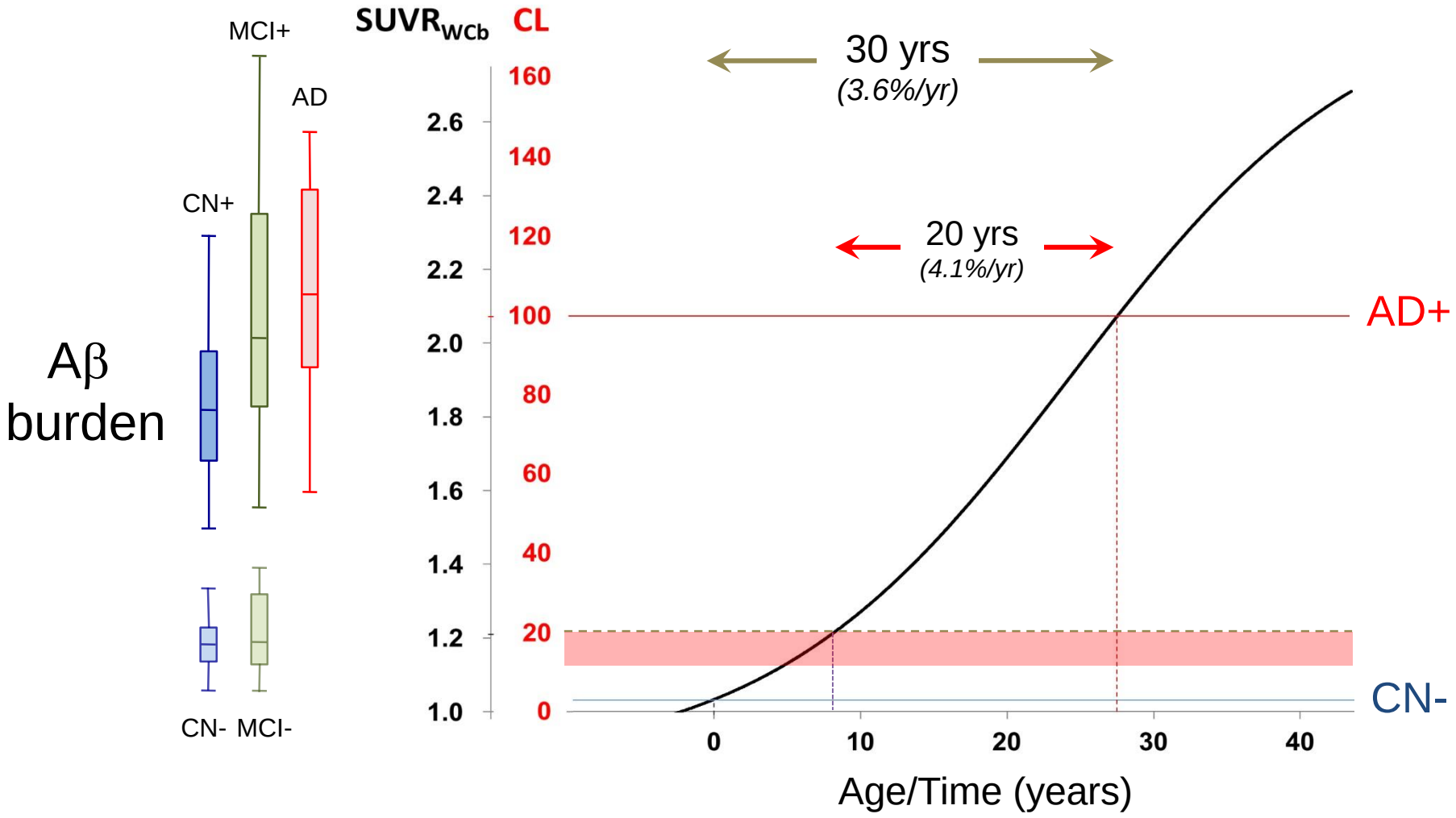
Clinical classification	CN	MCI	AD	Total
Years	6373	772	1200	8418

Aβ and Tau Imaging in AD (^{18}F) (Villemagne and Rowe)



Aβ deposition over time

5-9 year follow-up (PiB/NAV)



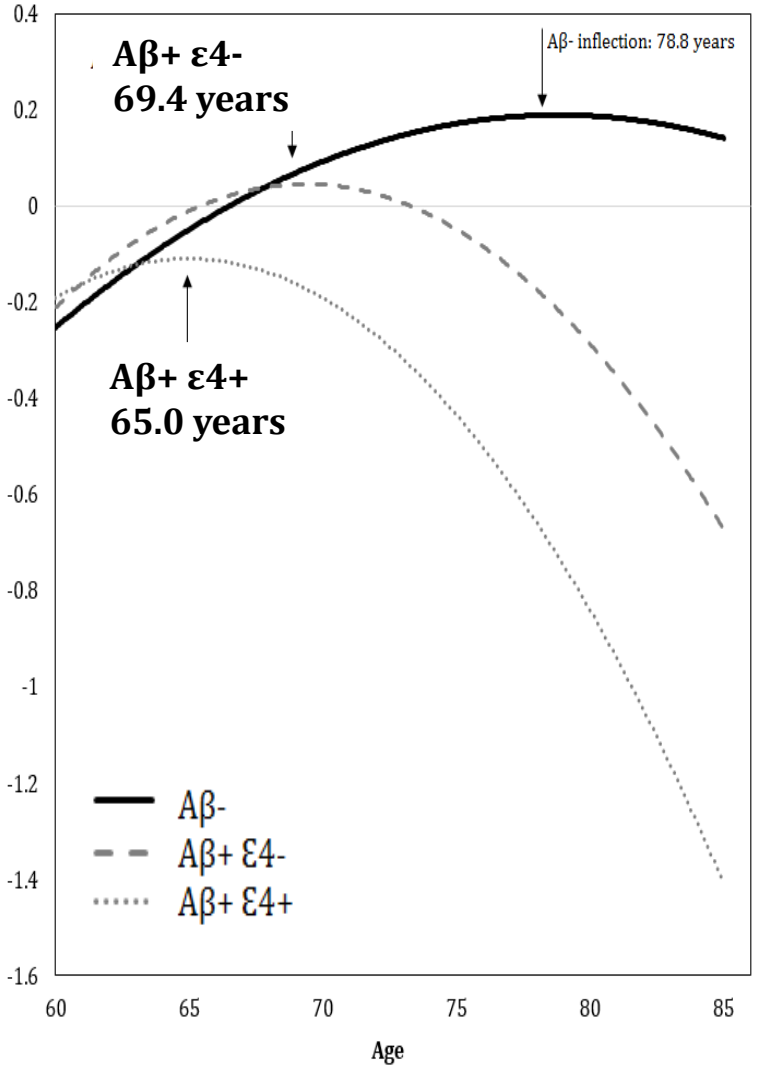
(Villemagne and Rowe)

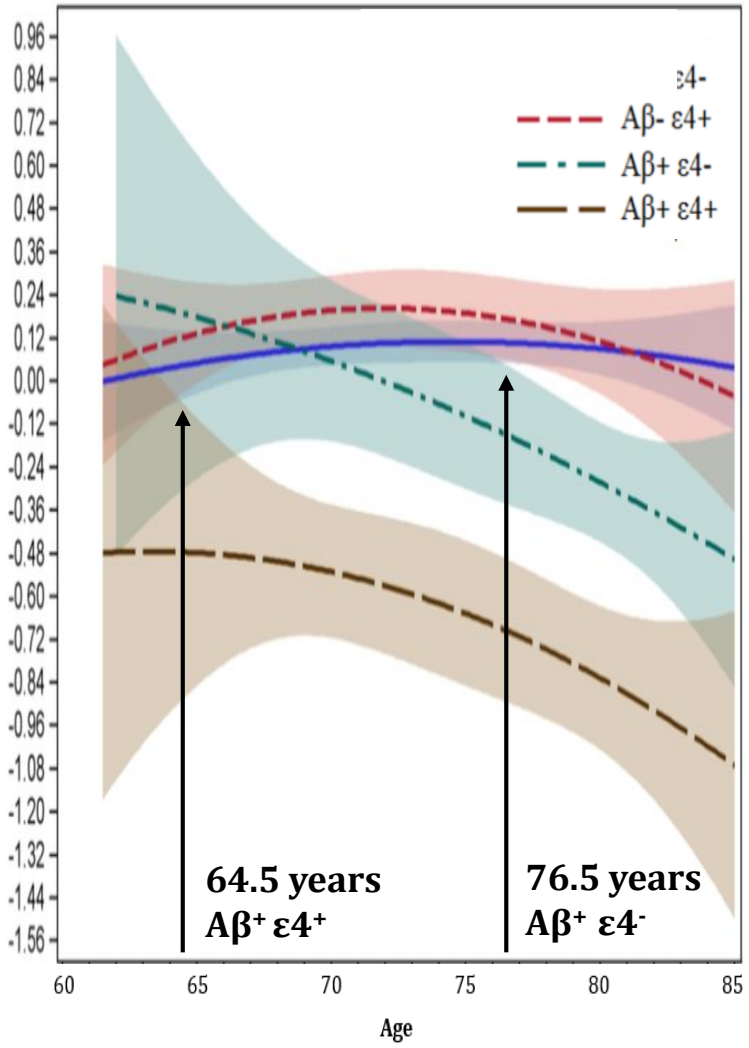
Age 50

Age 70

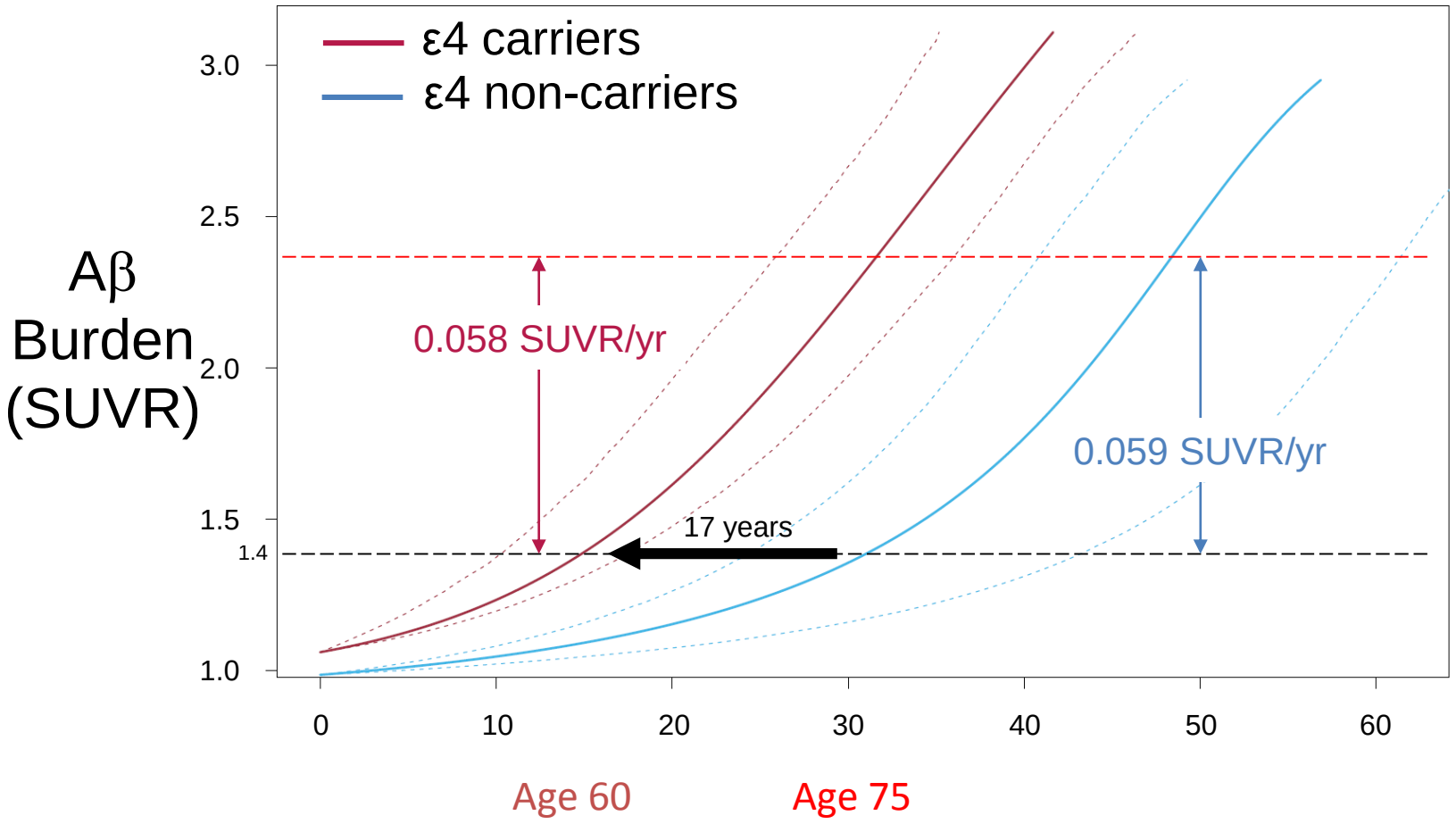
Preclinical AD age at onset of episodic memory decline: effect of APOE4; quadratic curve fits, differences from Aβ- subjects compared to inflexion points (Lim et al., JAMA Neurol 2018).

Episodic Memory Composite



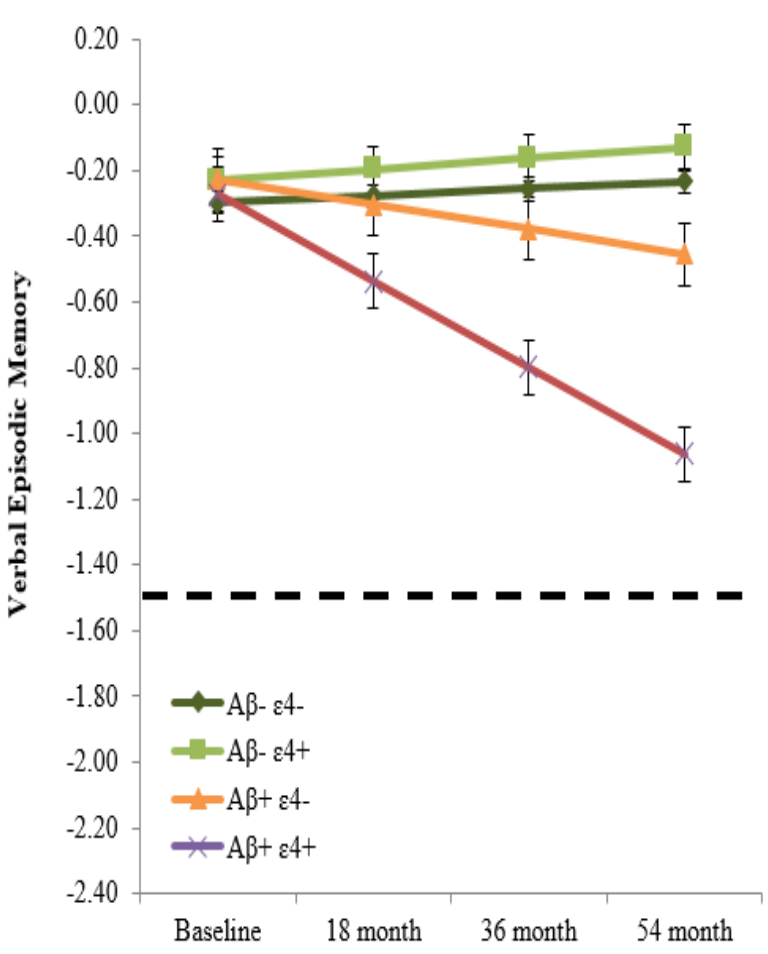


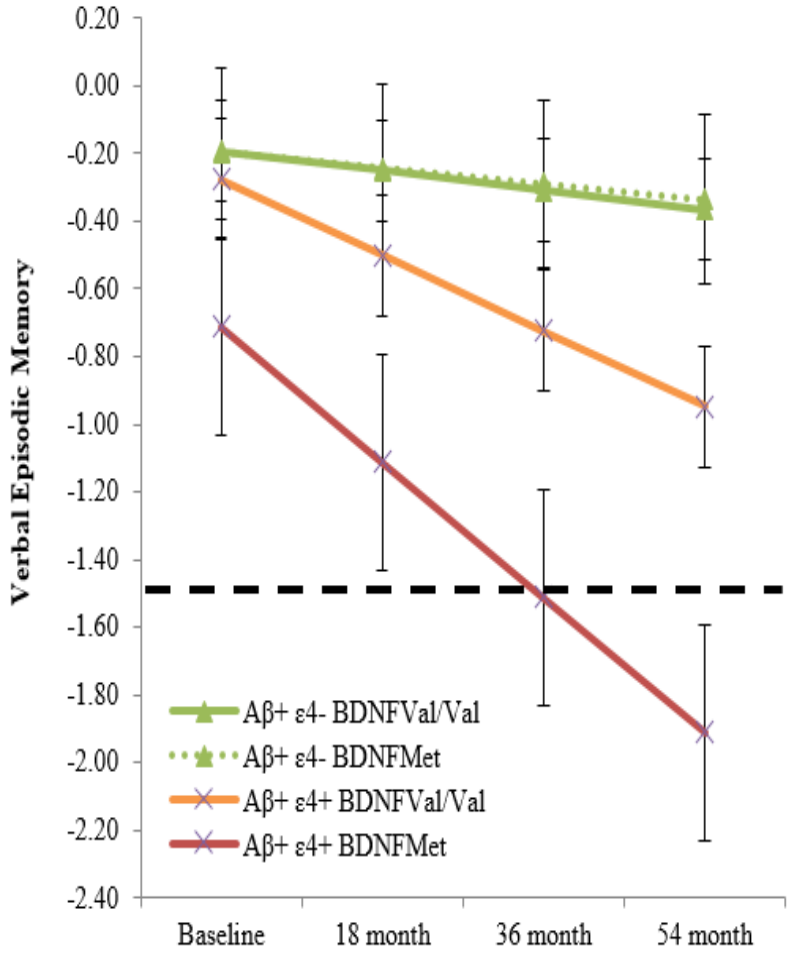
Impact of ε4 carriage on the progression of Aβ-amyloid accumulation (AIBL)



(Burnham & Villemagne)

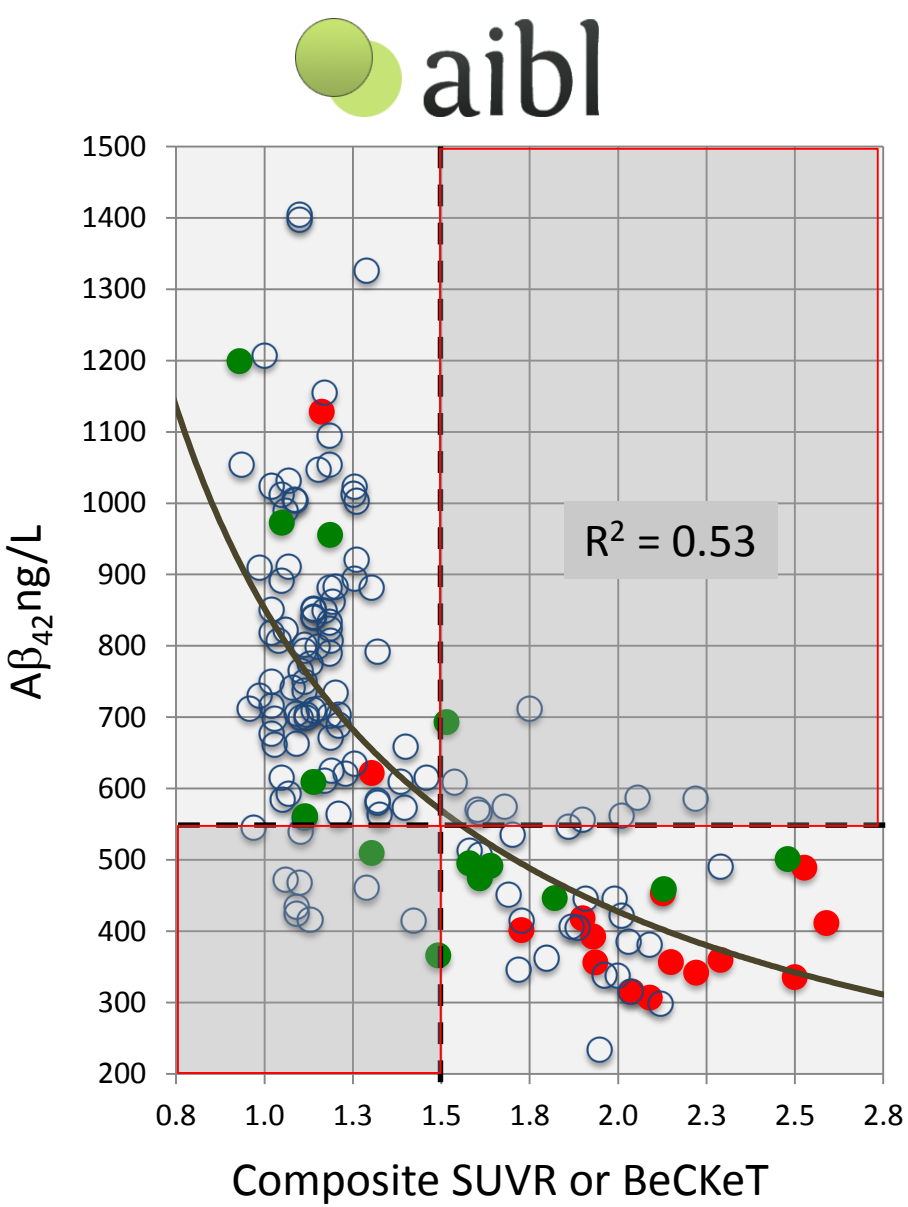
Trajectories of cognitive decline over 54 months in preclinical AD: effect of ApoE and BDNF polymorphisms (Lim et al. 2015)

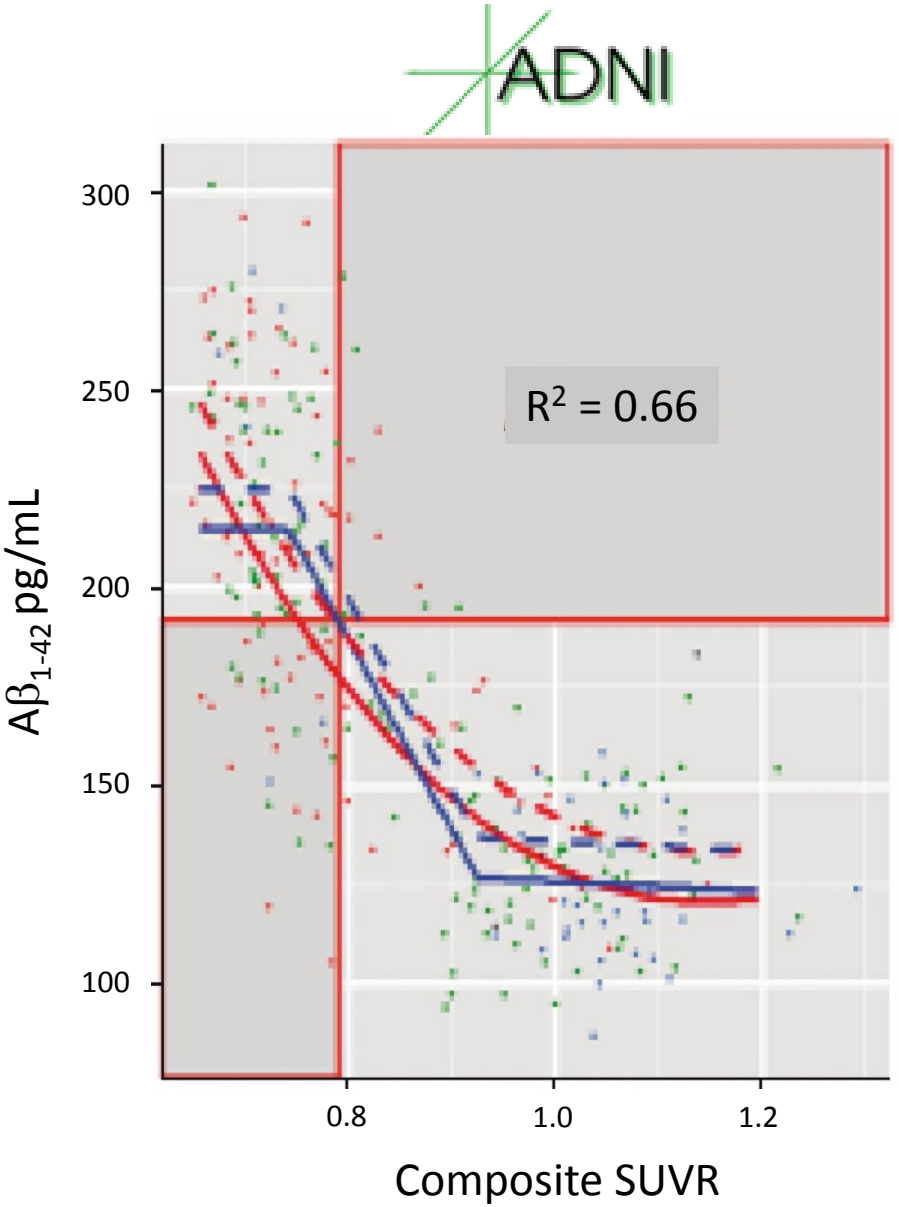




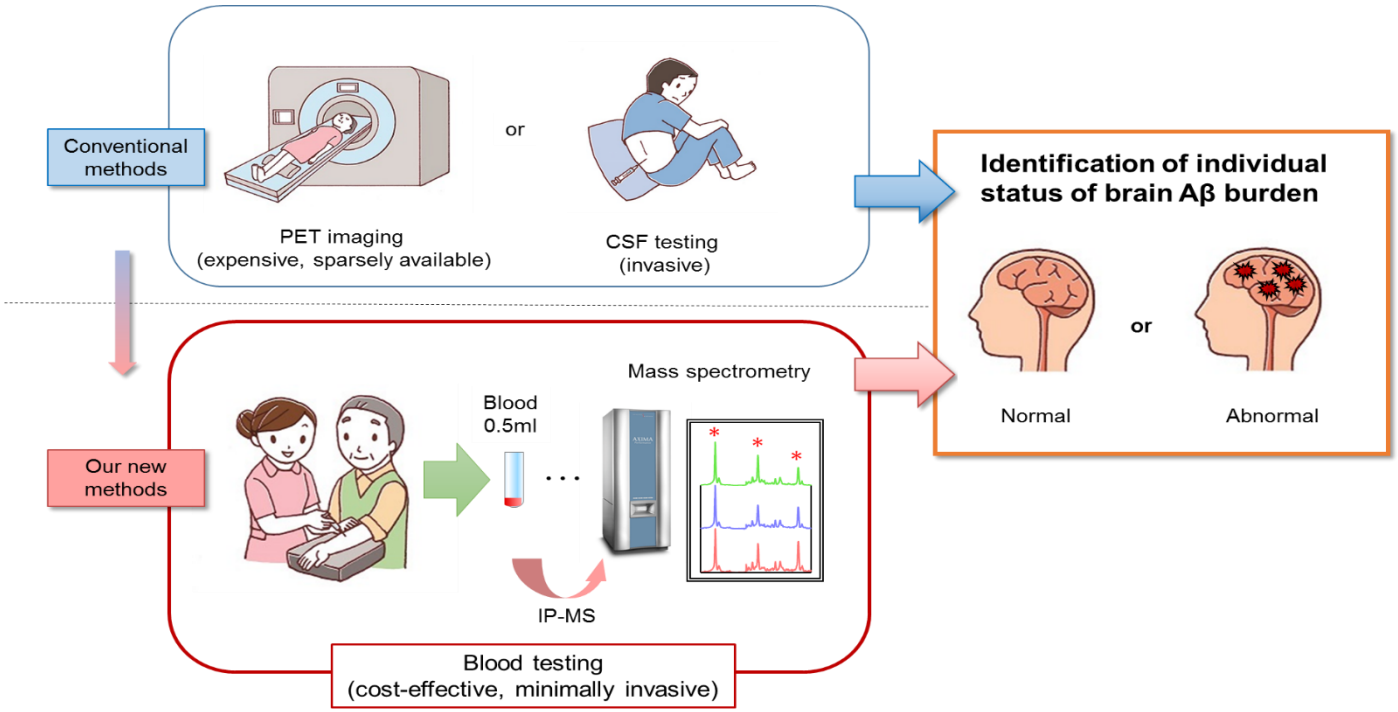
$A\beta^{+}$ $E4^{-}$ $BDNF^{+/-}$ 30 yrs; $A\beta^{+}$ E^{+} $BDNF^{val}$ 10yrs; $A\beta^{+}E^{+}BDNF^{met}$ 3yrs

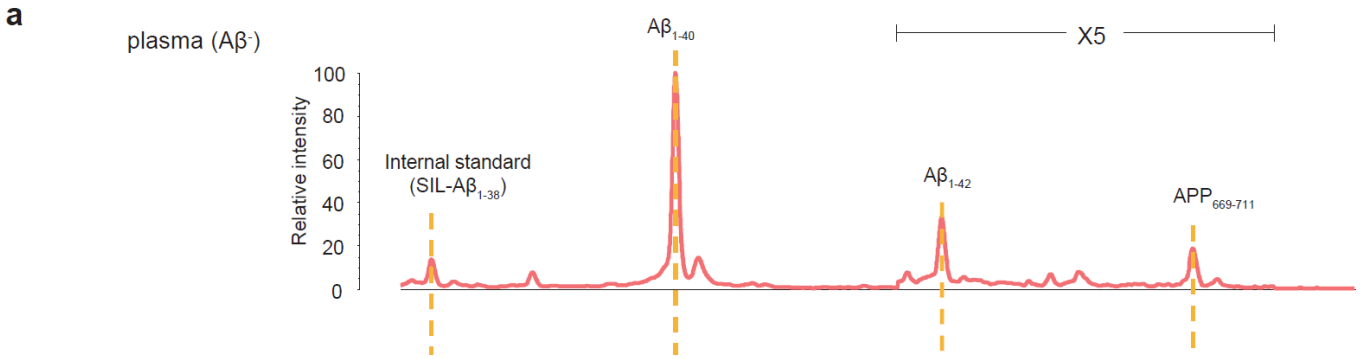
Relationship between brain Aβ and CSF $A\beta_{42}$





Shimadzu blood test (Nakamura, et al., 2018)





High performance plasma Aβ-amyloid biomarkers for Alzheimer's disease (Nakamura et al., Nature 2018: 554: 249-254)

IP- MALDI-TOF MS

$[A\beta_{42}]_{plasma}$ 38.5 ± 5.7 ng/L ($A\beta^{-}$) $[A\beta_{40}]_{plasma}$ 233.5 ± 43.1 ng/L ($A\beta^{-}$)

28.5 ± 2.5 ng/L ($A\beta^{+}$) 220.7 ± 36.8 ng/L ($A\beta^{+}$)

(26% lowering : 8.6 pm to 6.3 pm)

Composite ratios of: $(APP_{669-711})$ $(A\beta_{(-3)-40})$ / $A\beta_{1-42}$ + $A\beta_{1-40}/A\beta_{1-42}$:

AUC	94-97%
Accuracy	90%
Sensitivity	88%
Specificity	87%

Compared to ^{11}C-PiB, 10% lower accuracy with ^{18}F tracers (FLUTE, FBP)

For preclinical AD (30% prevalence)

Sensitivity	88%
Specificity	87%
PPV	74%
NPV	94%

For prodromal AD (66% prevalence)

Sensitivity	90%
Specificity	87%
PPV	74%
NPV	94%

Primary vs secondary prevention of AD

- Primary (pre-AD) in populations who fall below the Aβ cut-offs for CSF/PET. Subjects who meet prognostic algorithm of “age x genes x PET/CSF” change over three years. Characterised as “Aβ accumulators”. Design of trial in development recruiting from failed screens in A4.

- Secondary prevention in subjects with preclinical AD (over the threshold for Aβ PET/CSF) now underway: DIAN-TU in pathogenic mutation carriers and A4 with solanezumab in sporadic preclinical AD

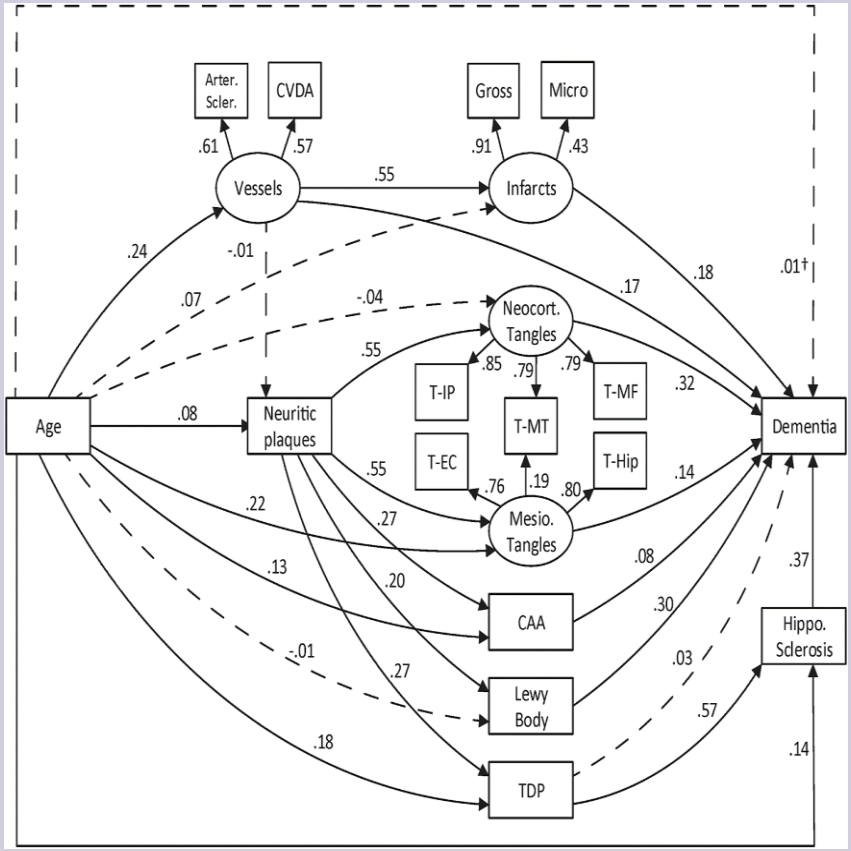
Alzheimer's disease: future strategies for disease modification

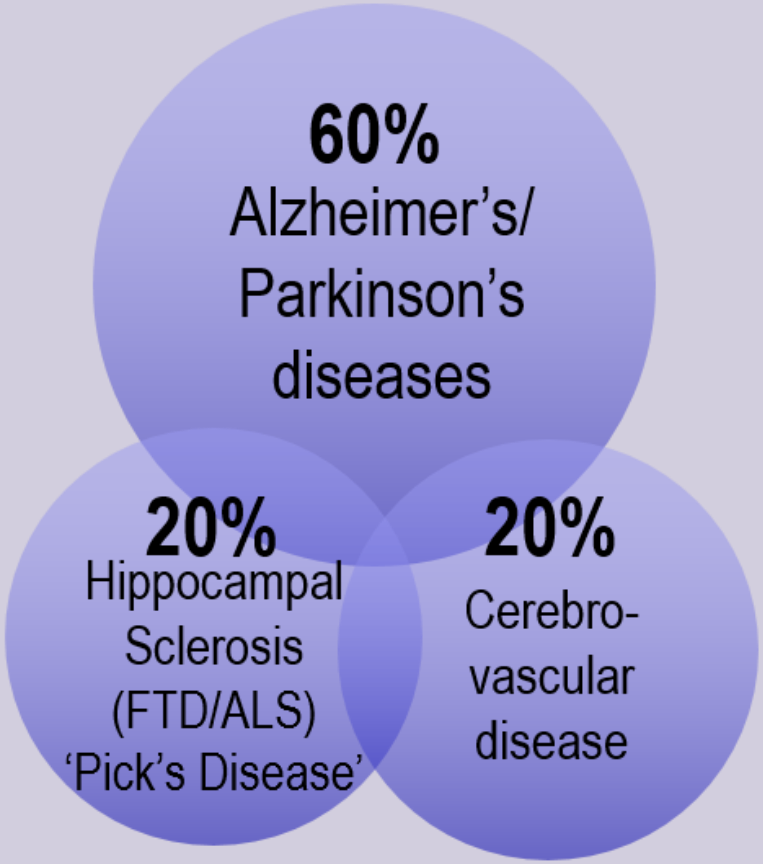
- Determine and use Maximum Tolerated Dose (MTD)
- Develop rational combination therapeutics:

  Lower production by 10-20% (β/γ secretase inhibitors)
  Stabilize and neutralize (8OH-quinoline; Mcab to mid-region)
  Clear (Mcab to N-terminus)

- Design "super-adaptive" trials with frequent, interim, quantitative real-time biomarker evaluations
- Consider lowering Aβ burden to baseline (Mcab) in earliest stage, followed by maintenance therapy with inhibitors of production and aggregation, dimer stabilization, and improved clearance strategies
- May require use of "co-morbid disease-free" subjects

The burden of age-associated cognitive impairment lies in the co-morbidities of only a few major disease pathways; these need to be controlled in clinical trial designs.





Power CM et al. Ann Neurol 2018; 84: 10-22.

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Cognitive and brain aging in the absence of preclinical Alzheimer's disease

AIBL Participant Information Day – 21 June 2019

Christa Dang, PhD Candidate

Alzheimer's disease (AD)

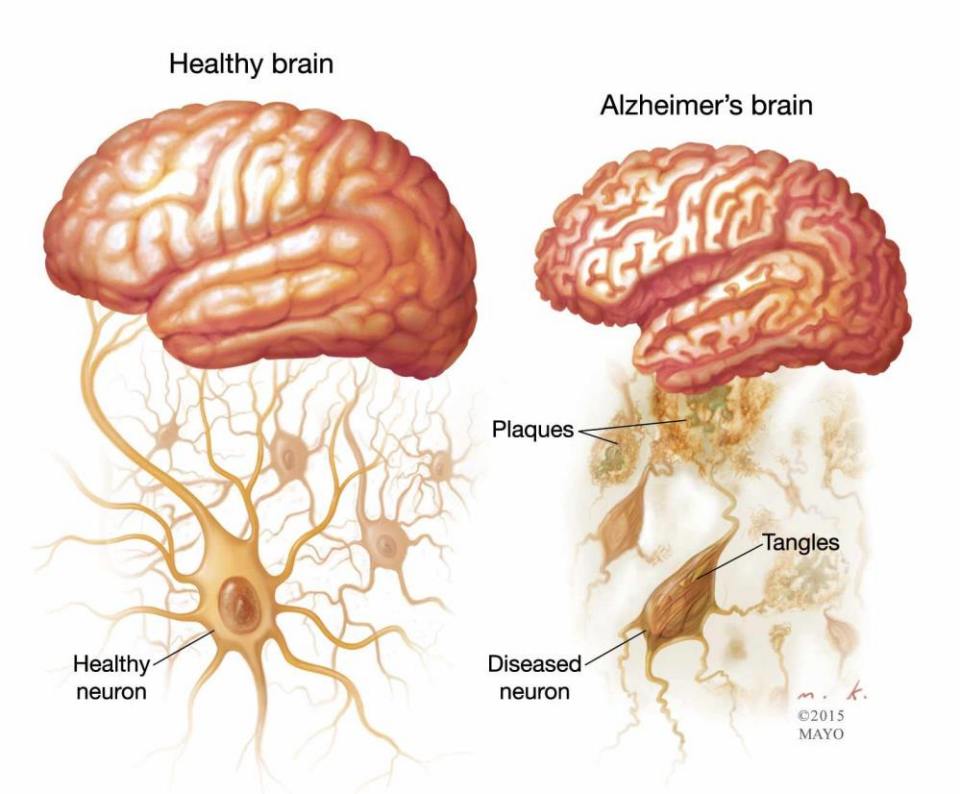


Figure from the Mayo Clinic

Amyloid-β and preclinical AD

- Aβ+ is associated with higher risk of AD, and faster rates of cognitive decline[1] and cortical atrophy[2]

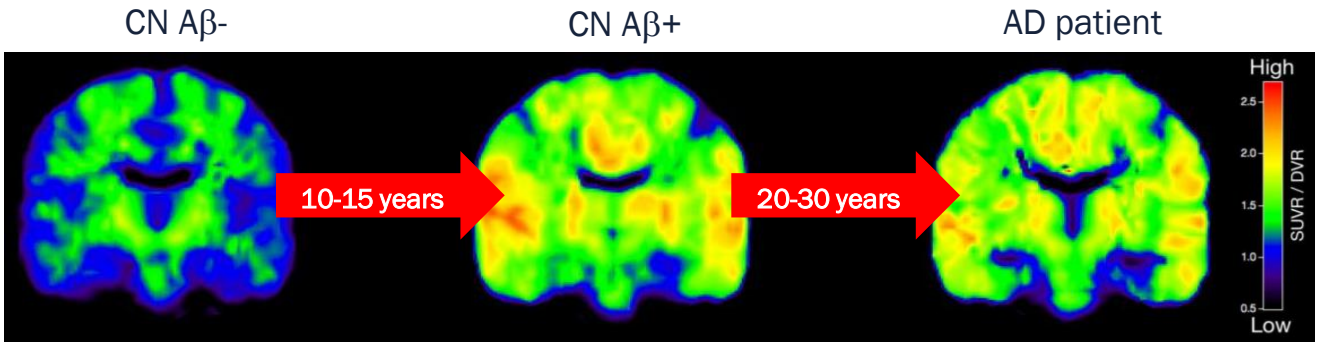
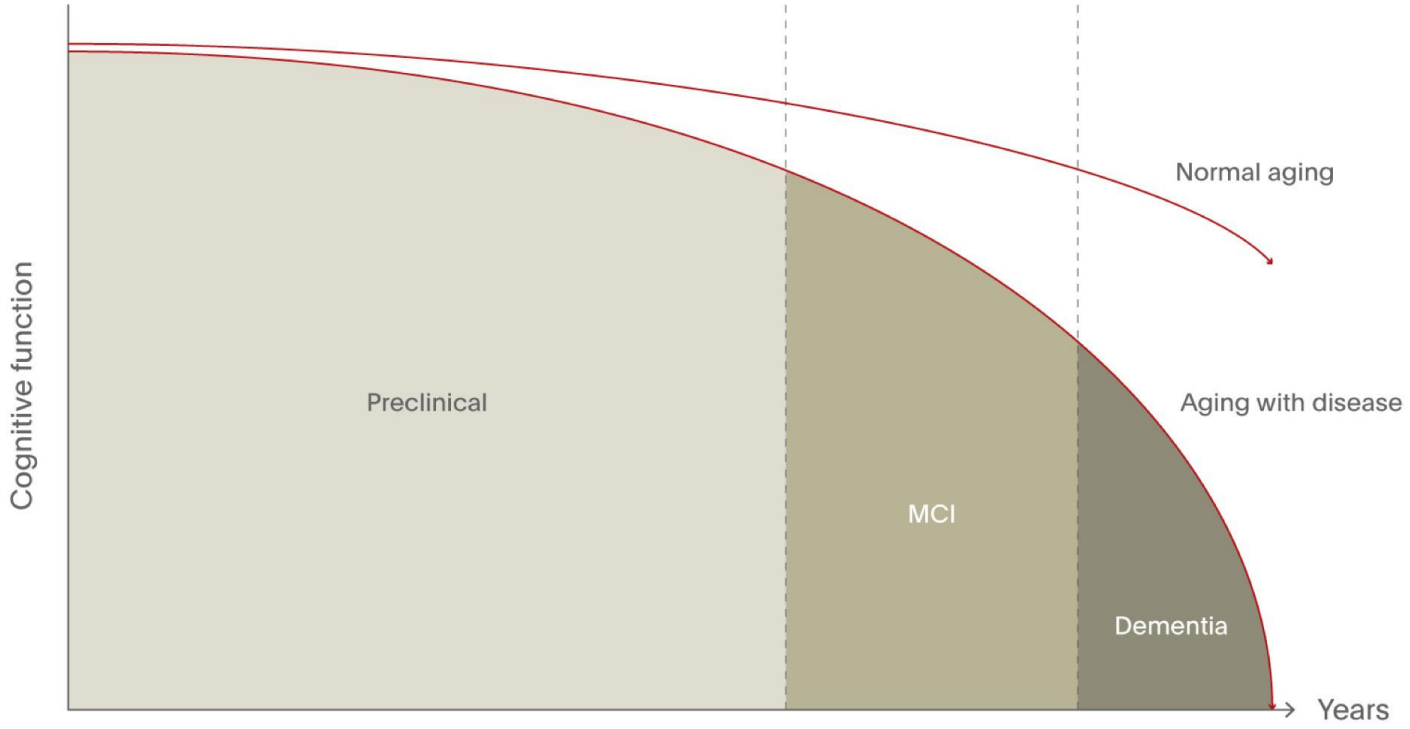


Figure adapted from Villemagne et al. Lancet Neurology. 2013; [1] Baker et al. Alz Dem DADM. 2017; [2] Chetelat et al. Neurology. 2012; [3] Jansen et al. JAMA. 2015

Hypothetical model for the pathological–clinical continuum of Alzheimer's disease



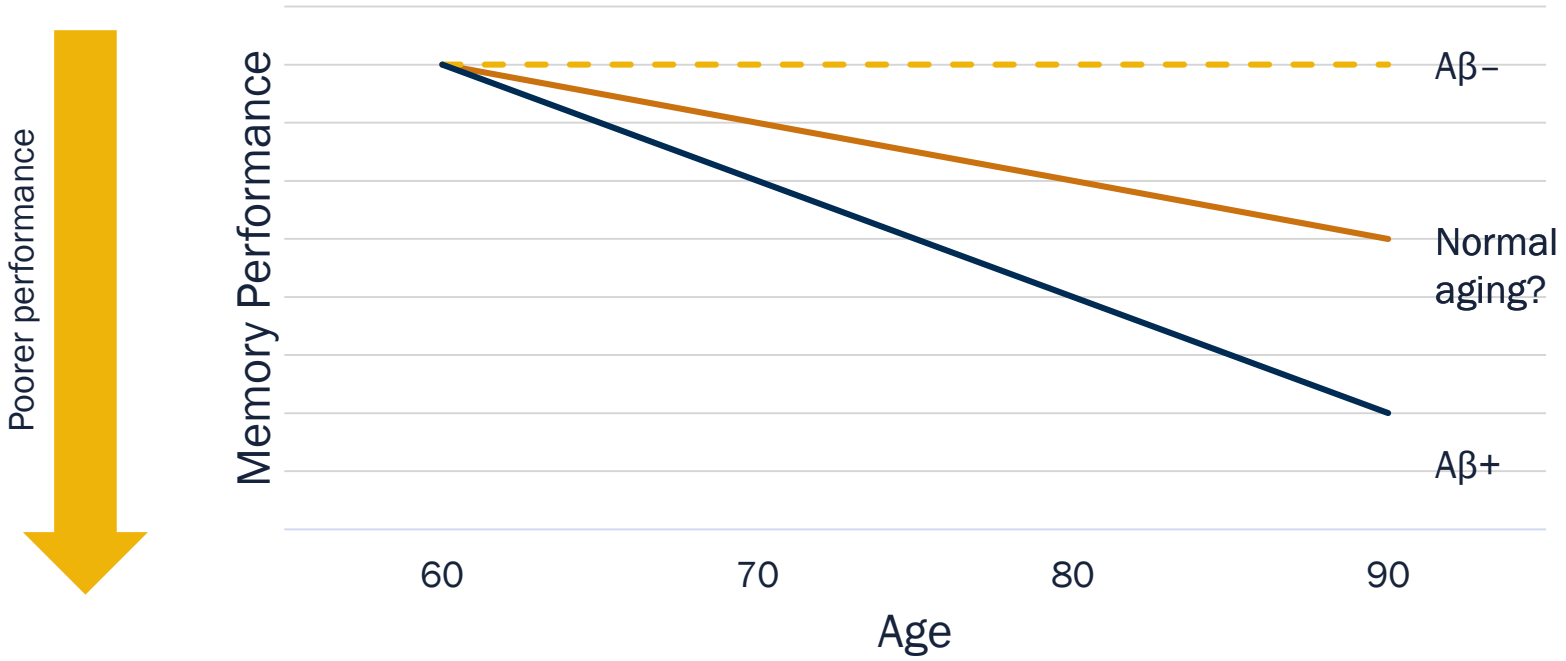
MCI=mild cognitive impairment
Sperling et al. Alzheimers Dement 2011;7(3):280–292

Lundbeck Institute
Campus

Aging

- AD or any cause of dementia is NOT a normal part of aging
- Aging is thought to be associated with declines in cognition and loss of brain volume
  - But not to the same extent as observed in AD
  - BUT MAYBE NOT TRUE

Age-associated cognitive decline



Harrington et al. Neurobiol Aging. 2018

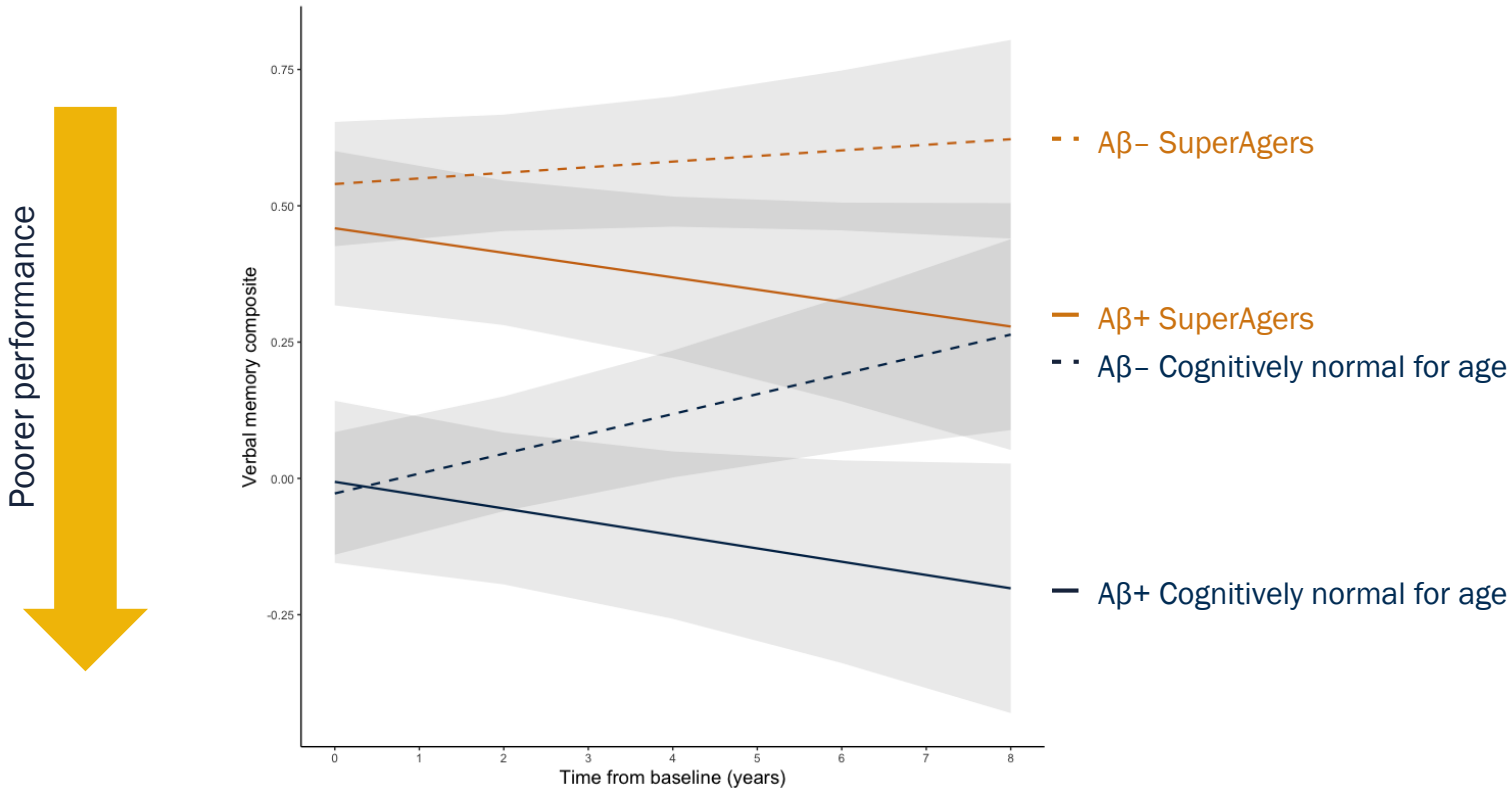
What does successful aging look like?

SuperAgers

- Older adults (≥60) who exhibit **verbal memory** performance equivalent to, or better than, that of individuals **20-30 years younger**, with **no impairment** in any other cognitive domains[1]
- SuperAgers may be able to resist age-associated cognitive decline and neurodegeneration[2,3]
- SuperAging = aging without disease (i.e. normal aging)?

[1] Harrison et al. J Int Neuropsychol Soc. 2012; [2] Cook et al. JAMA. 2017; [3] Rogalski et al. J Cogn Neurosci. 2013

Rate of change is the same for SuperAgers



Dang et al. Arch Clin Neuropsych. 2018

SuperAgers don’t have less Aβ

WERE Aβ+

Key takeaways

- Aβ is a better predictor of future outcomes than being classified as SuperAger
  - Aβ- showed preserved cognition and reduced rates of brain volume loss
  - No difference in rates of change for SuperAgers
- Having exceptionally good memory is not as important to your future as it is to reach old age without accumulating a lot of Aβ

What does this all mean?

- Normal aging is NOT associated with memory loss!
- It is possible to age well without developing signs of AD
- You don’t need to have had a fantastic memory to age well

THANK YOU!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