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**The Role of Beta Amyloid and Tau in Post
Stroke Cognitive Impairment**



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Padang**

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Outlines Introduction

Epidemiology

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Beta Amyloid and Post Stroke Cognitive Impairment

Tau Protein and Post Stroke Cognitive impairment

Conclusion

Introduction

Post stroke cognitive impairment (PSCI)

- failure in any cognitive domain after stroke: executive function; memory; language; visuospatial ability; visuoconstructional ability; or global cognitive function

Post stroke dementia (PSD)

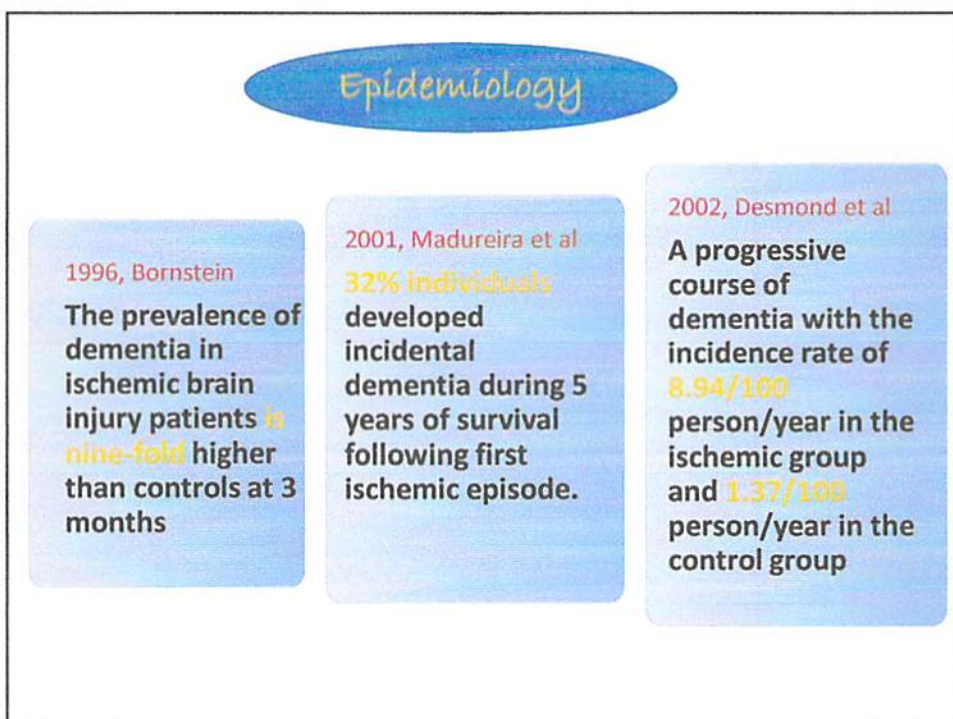
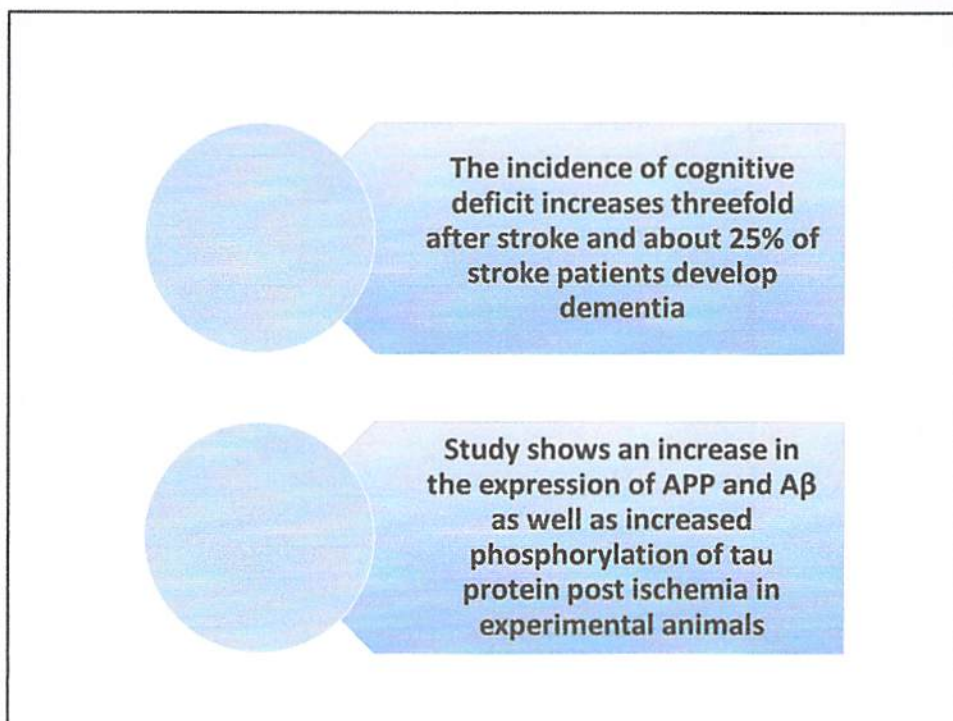
- any dementia occurring after stroke: Vascular dementia; Alzheimer's disease or other degenerative dementia; or mixed dementia.

Both categorized as vascular cognitive impairment (VCI)

Stroke is the leading cause of severe physical disability in the elderly, the second most common cause of dementia

Post-stroke cognitive impairment (PSCI) is defined as a cognitive deficit that develops up to the third month after stroke

Cognitive impairment after stroke is a frequent but neglected consequence compared to other neurological deficits such as sensory or motor impairment



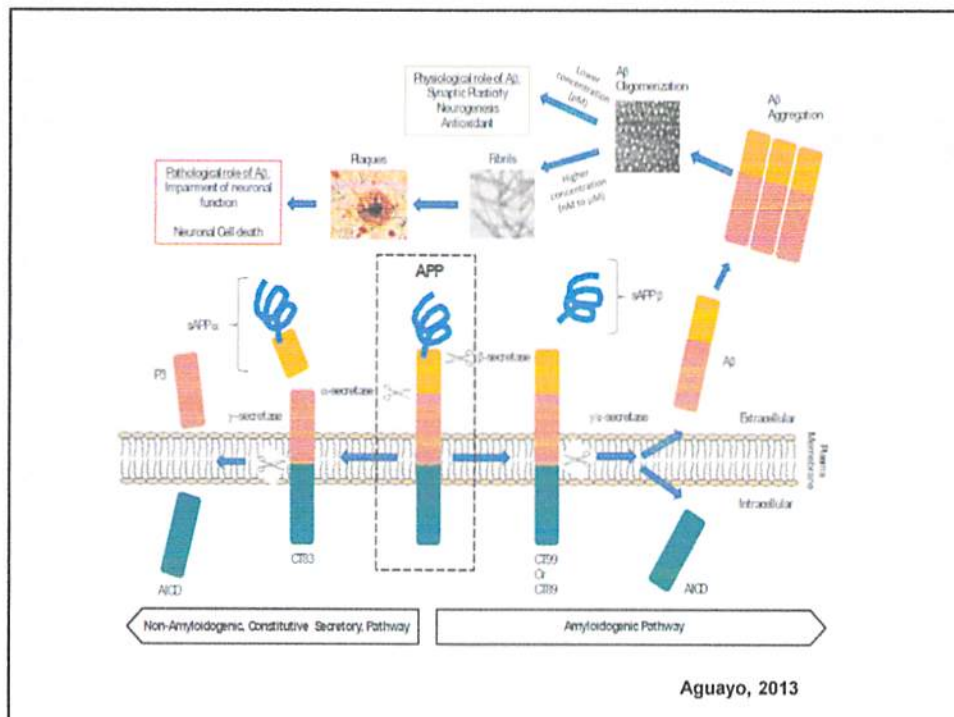
Post Stroke Beta-amyloid

Beta-amyloid

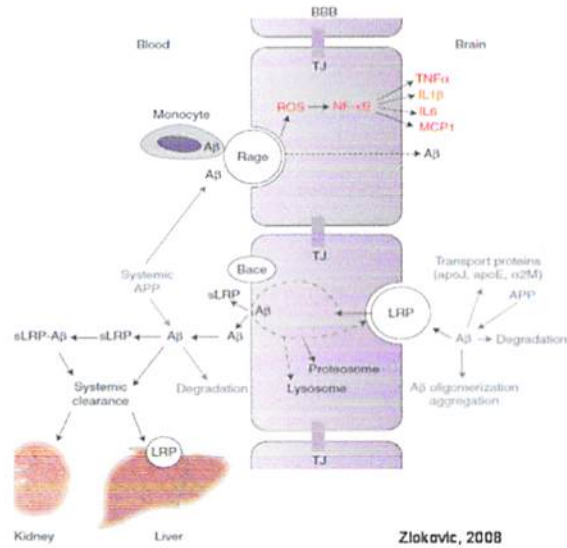
An amyloid that is derived from a larger precursor protein

Different $A\beta$ species exists, but $A\beta_{40}$ is the most abundant (~80-90%), and $A\beta_{42}$ (~5-10%).

$A\beta$ concentration of non-bonded (free) level in the brain is six times higher compared to plasma



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Physiological roles of amyloid-beta

modulation of synaptic function,

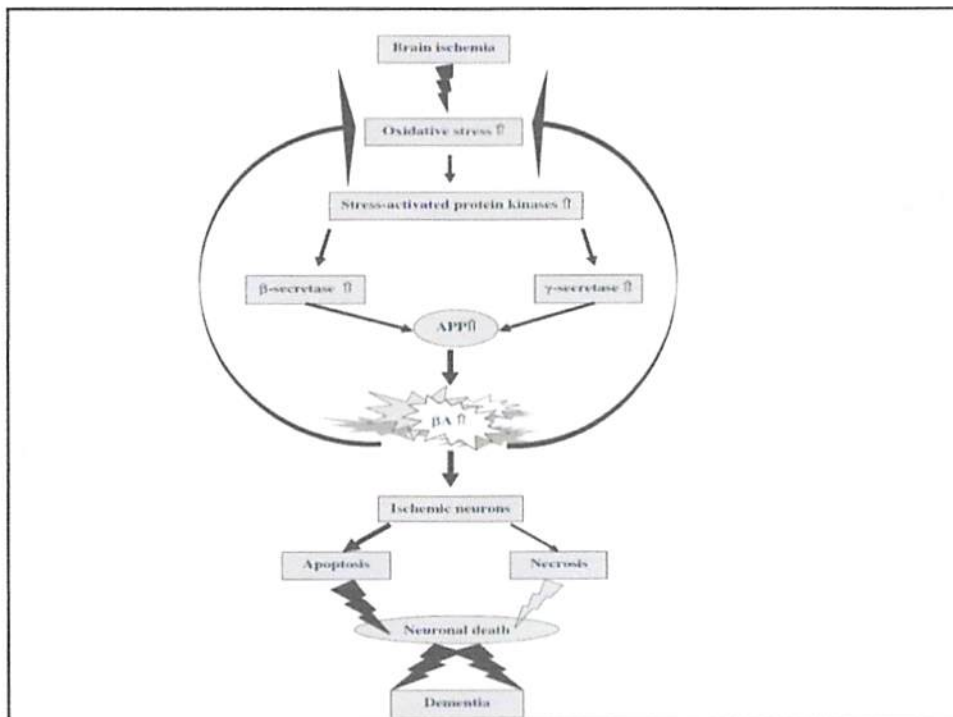
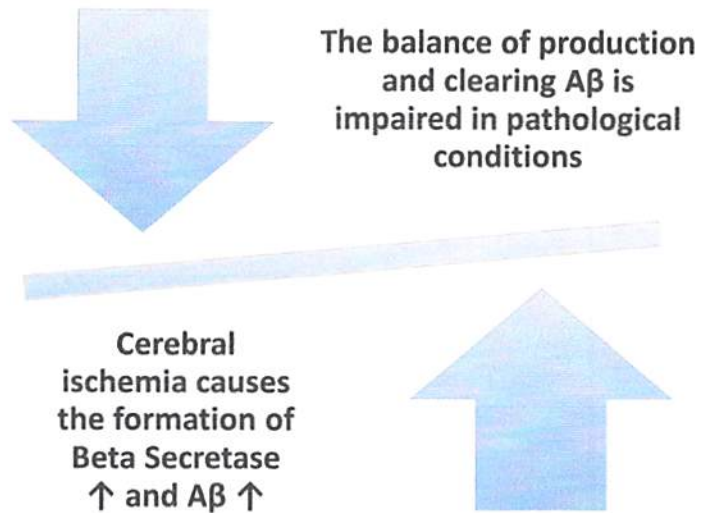
facilitation of neuronal growth and survival

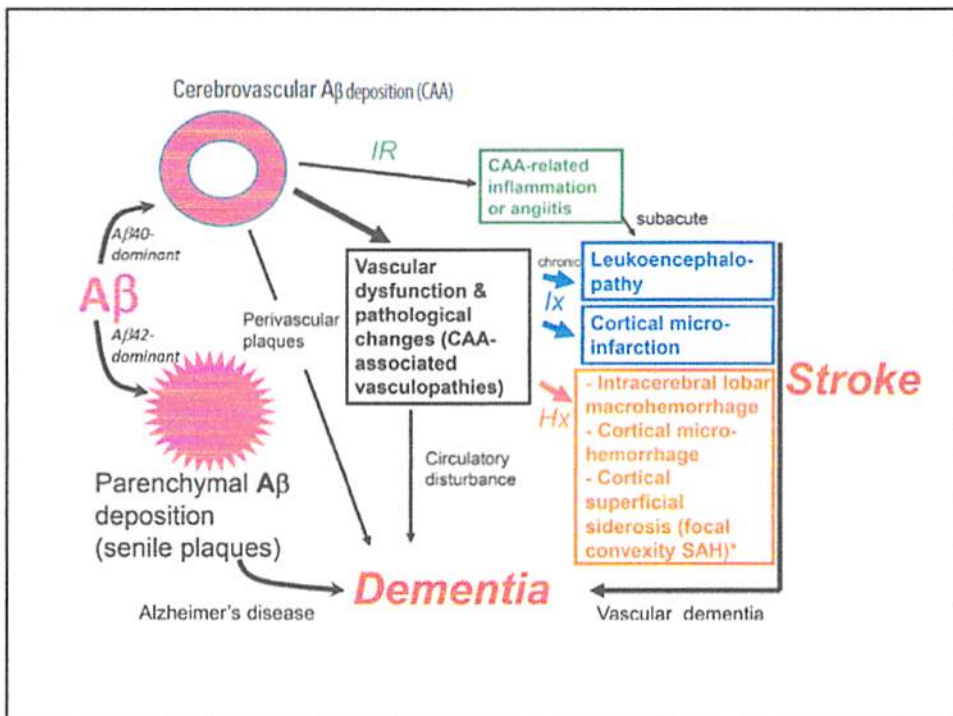
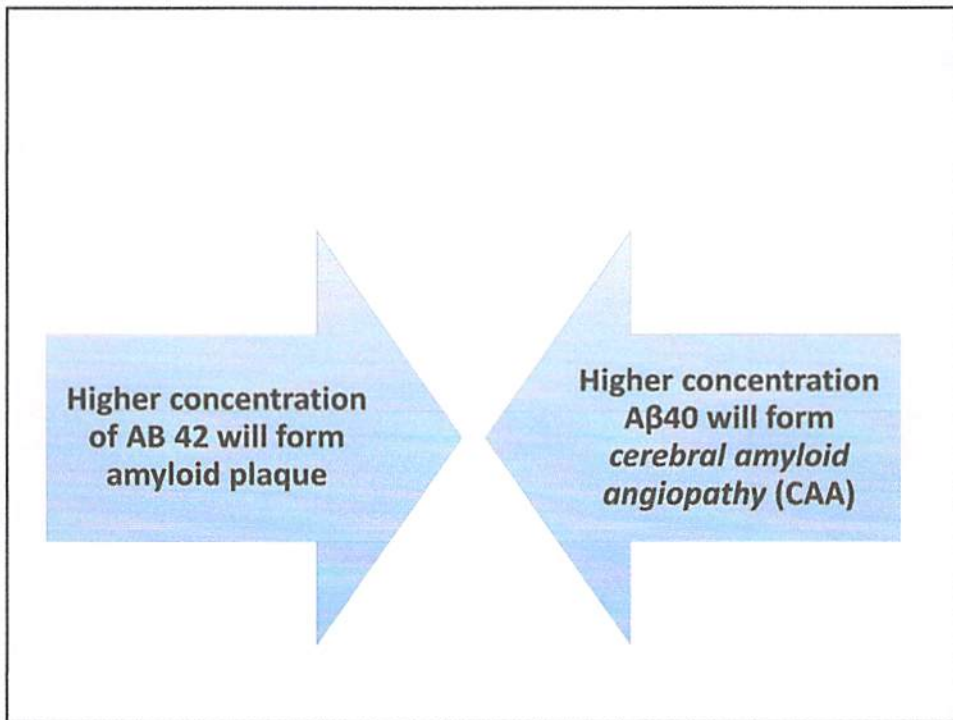
protection against oxidative stress

surveillance against neuroactive compounds, toxins and pathogens

enhancement of synaptic plasticity and memory

cont..





Post Stroke Tau Protein

localized in microtubules (MT) and serves the function of stabilizing microtubules.

related to neuronal process and axonal transport maintenance

Tau protein

In cerebral ischemia, we found elevated phosphorylation of tau protein in cortical neurons with apoptosis signs nearby

cont..

Tapiola T (2009) : low levels of $A\beta_{42}$ and high levels of protein tau in the CSF predict the presence of Alzheimer's pathology in the brain with high accuracy

Beta amyloid causes the damage to synapse plasticity and inhibit long term potentiation (LTP) mediated by Tau protein

Beta Amyloid and Post Stroke Cognitive Impairment

Post stroke cognitive impairment/dementia :
prototype of VCI/VaD

Approximately one third underlying pathogenesis of
post stroke dementia resembles Alzheimer
dementia.

Various vascular risk factors exist as in sporadic AD

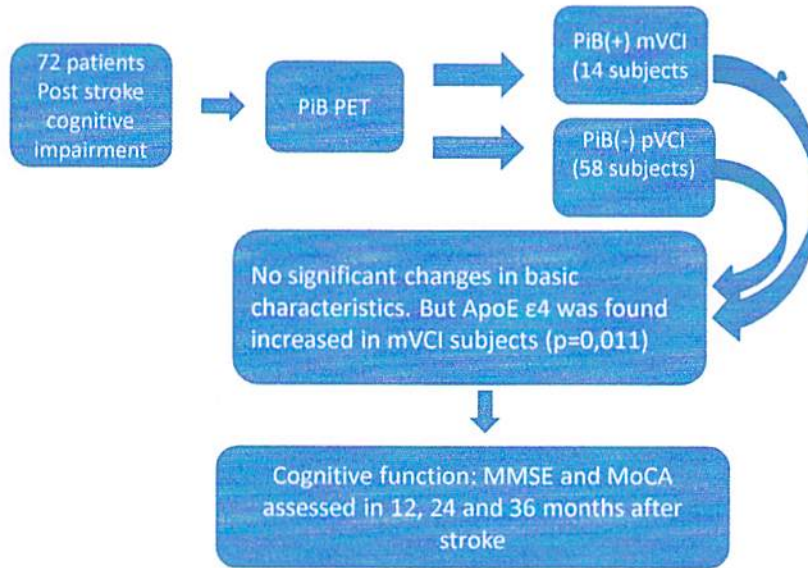
Beta Amyloid and Post Stroke Cognitive Impairment

Andin (2005) : 40% VaD patients was found features of
AD in post mortem biopsy

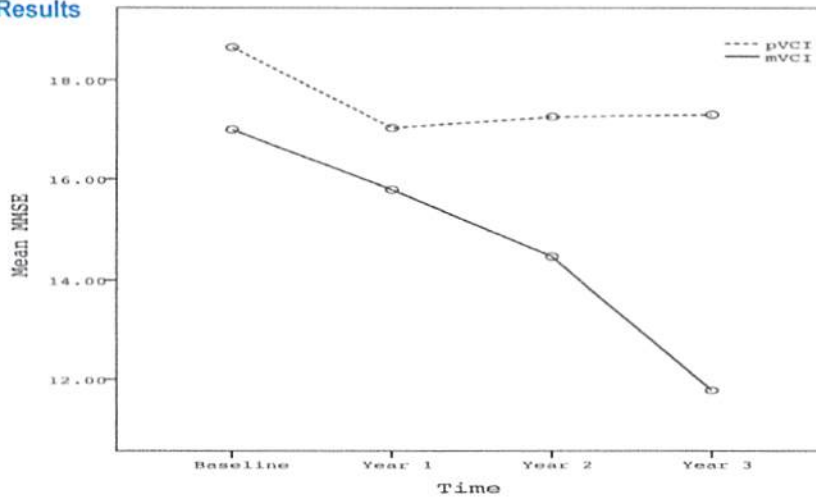
Kalaria (2003) : 43% VaD patients was found deposits
of β -amyloid ($A\beta$) and neurofibrillary tangle (NFT)



Liu W et al (2015) conducted prospective study :

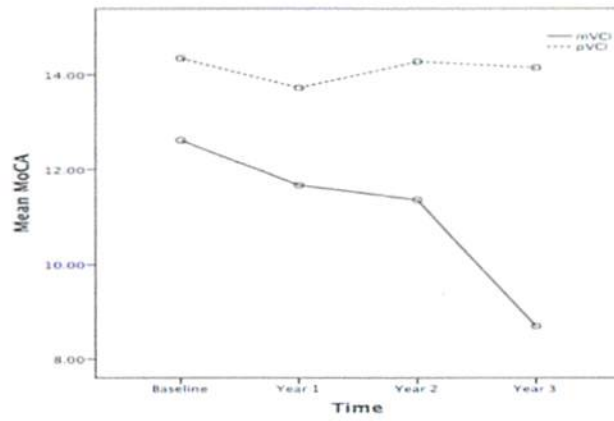


Results

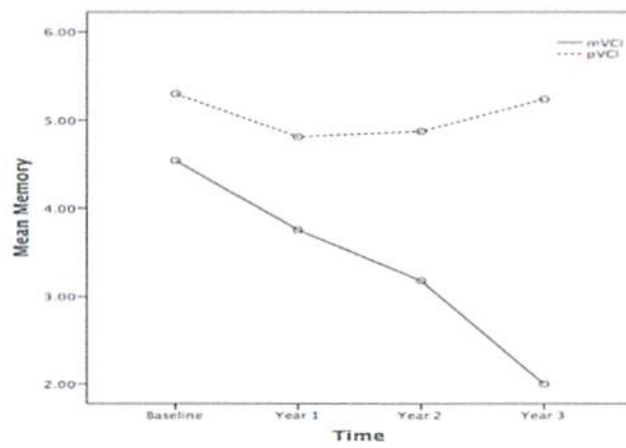


No significant changes of MMSE score found in pVCI group ($p=0,208$)

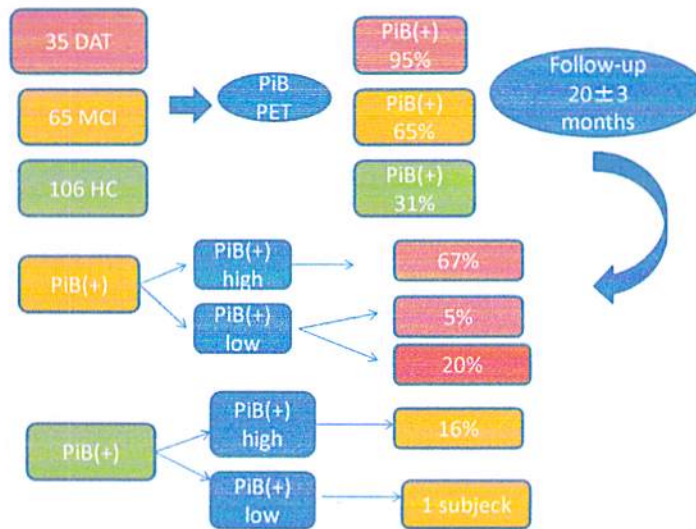
MMSE score in mVCI group (in third year) was significantly lower compared to baseline ($p=0,020$)



The mean MoCA score was found lower in mVCI compared to pVCI.

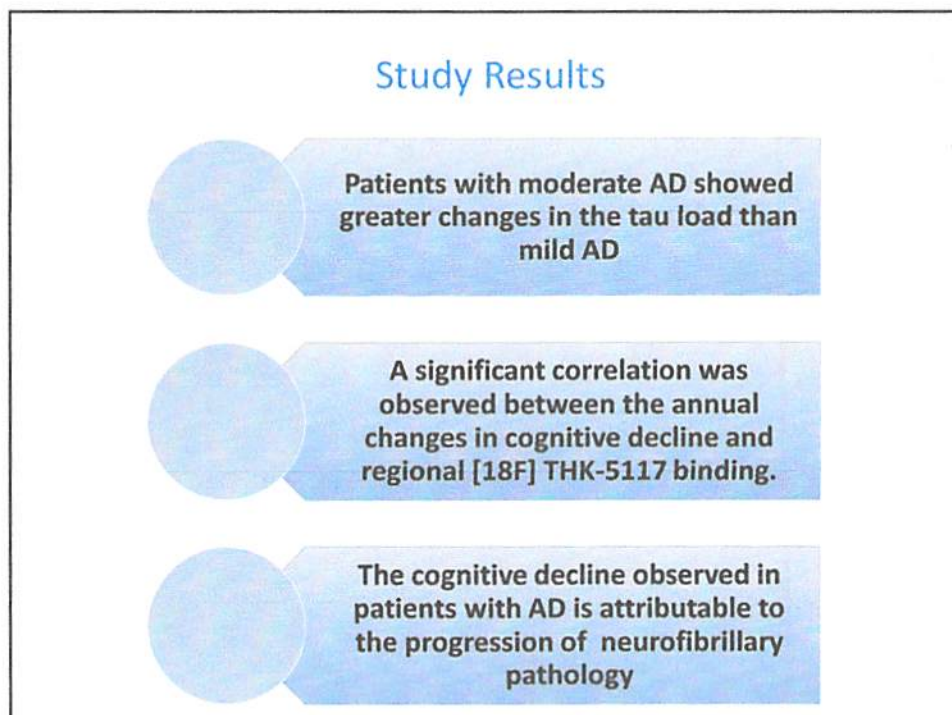
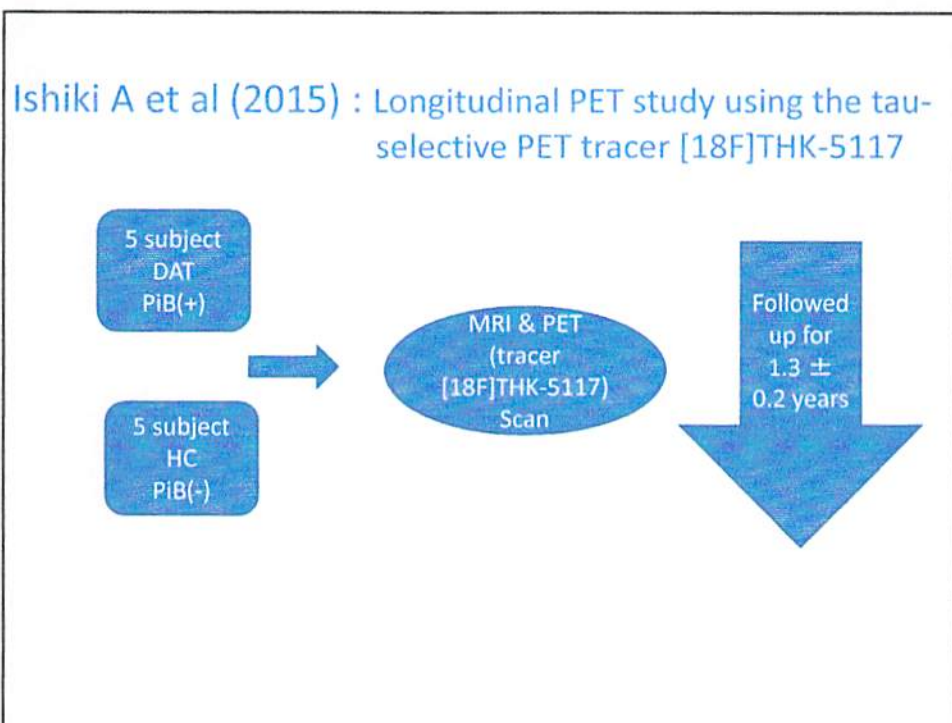


Villemagne VL, et al (2011) : Aβ association to cognitive function and disease progression.



Tau Protein and Post Stroke Cognitive Impairment

- The formation of neurofibrillary tangles is believed to contribute to the neurodegeneration observed in AD
- Strong associations between the neurofibrillary pathology and both neuronal loss and the severity of cognitive impairment (postmortem study)
- PET imaging is expected to be useful for the longitudinal assessment of neurofibrillary pathology in the living brain



Conclusion

- **Poststroke cognitive impairment (PSCI) is prototype of vascular cognitive impairment**
- **Approximately one third underlying pathogenesis of post stroke dementia resembles Alzheimer dementia.**
- **The presence of beta amyloid deposit and Neurofibrillary tangle in the brain of VCI patients may become predictor of developing dementia**

