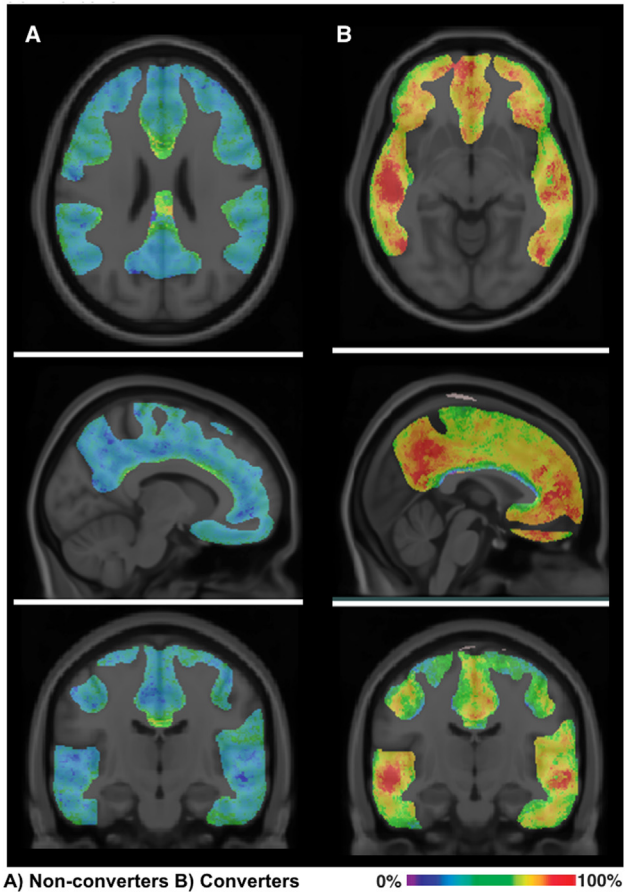

Figure 3. Probabilistic maps of [^{18}F]Florbetapir SUVR positivity at every voxel for MCI Non-converters and converters.

vealed that the analysis of amyloid-PET cut-offs at every voxel might provide important information regarding the patterns of regional Aβ abnormalities associated with the clinical progression to AD.

IC-P-026 PROBABLE CAA AND CLINICAL IMPLICATIONS IN A LARGE MEMORY CLINIC COHORT

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Background: Cerebral amyloid angiopathy (CAA) is associated with cognitive impairment, and especially with Alzheimer's disease. CAA manifests as neuroimaging markers of small vessel disease, and a conspicuous marker for CAA is lobar cerebral microbleeds (CMBs). We aimed to study differences in patients with and without probable-CAA and cognitive impairment in a large memory clinic. **Methods:** A total of 1504 patients undergoing memory investigation were recruited in our study. All patients underwent MR imaging with hemosiderin sensitive sequences and 1039 patients had cerebrospinal fluid (CSF) biomarker analysis. Probable-CAA was classified according to the Boston criteria using microbleeds as hemorrhagic markers. MR images were further analyzed for all markers of small vessel disease including superficial siderosis, white matter hyperintensities (WMH), lacunes and enlarged perivascular spaces. Generalized linear models with appropriate corrections were used to assess data. **Results:** A total of 99 patients (46 Alzheimer disease, 27 mild cognitive impairment, 6 subjective cognitive impairment, 9 vascular dementia, and 11 with other diseases) were classified as having probable-CAA. Patients without probable-CAA had lower CSF amyloid β (Aβ) 42 levels ($P<0.001$); patients with probable-CAA had higher CSF/ serum albumin ratios, reflecting blood brain barrier dysfunction, ($P=0.04$), as well as a tendency for higher total tau and P-tau levels ($P=0.05$). Patients with probable CAA had significantly higher burden of WMH (OR: 2.7, 95CI: 1.6-4.5), cortical superficial siderosis (OR: 6.6, 95CI: 2.3-14.8), lacunes 2.2 (1.3-3.8), and significantly lower amount of deep CMBs (OR: 0.04, 95CI: 0.01 - 0.31). There was no difference in amount of enlarged perivascular spaces, as well as MMSE score between patients with and without probable-CAA. **Conclusions:** Patients with cognitive impairment and probable-CAA likely have higher associated neurodegeneration with their disease, and a higher number of associated small vessel disease markers of CAA.

IC-P-027 AMYLOID-INDUCED MICROGLIAL ACTIVITY IN THALAMOCORTICAL CIRCUITS PREDICTS SUBSEQUENT COGNITIVE DECLINE

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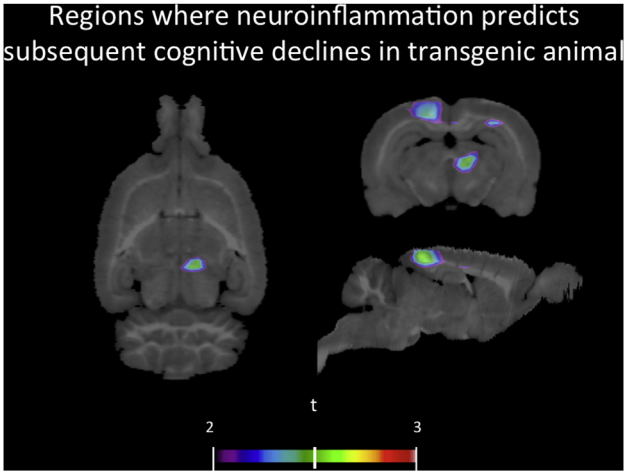
Background: Although the signature properties of Alzheimer's disease (AD), such as the formation of amyloid plaques, activation of inflammatory responses, and hyperphosphorylation have been well studied, the explanation illustrating a clear relationship specifically between neuroinflammation and cognitive impairment has not yet been discovered. Thus in this article, we employed the McGill-R-Thy1-APP transgenic (Tg) rat model to observe for activation of inflammatory responses and the presence of cognitive decline. We hypothesize that early expression of neuroinflammatory signals will lead to future cognitive decline in the amyloid expressing animals. **Methods:** Microglial activation was measured using the ligand [^{18}F]PBR06 for PET, while cognitive performance was measured with the MWM task in 5 wild-type (Wt) and 8 Tg rats at baseline (BL; 11.5 months) and at follow-up (FU; 16 months). PBR-PET images were processed using the cerebellar grey matter as the reference region and cognitive measurement was calculated as the average times of day 3 and 4 of the water maze task. The baseline as well as the change (FU – BL) in the cognitive measurements were correlated at voxel level using "VoxelStats" toolbox to observe for the effect of group and PBR interaction on the change in the water maze performance. The PBR values were corrected for

global cortical values, and t-statistical maps were generated to illustrate the regions of significance. **Results:** The association between baseline levels of cognition and inflammation was greater in the Tg than Wt rats in the right nucleus accumbens, whereas in the opposite was seen in the right inferior colliculus. The association between baseline levels of inflammation and change in cognition at follow-up, several regions including the left retrosplenial cortex, right hippocampus, and the right posterior commissure showed higher decrease in cognition of the Tg animals compared to the Wt (Figure 1). **Conclusions:** At baseline, there is no association between neuroinflammation and cognitive performance; however in more aged rats, baseline levels of PBR is able to predict cognitive decline. The results provide a framework that could potentially be applied in human studies focusing on the detrimental roles of neuroinflammation in AD.



IC-P-028 SLEEP QUALITY IN YOUNG AND MIDDLE AGE-PERIOD IS ASSOCIATED WITH CEREBRAL AMYLOID BURDEN IN COGNITIVELY NORMAL ELDERLY PEOPLE

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Background: Very little is known for the association between lifetime sleep experience and cerebral beta-amyloid protein (Aβ) deposition, which is the core pathological change related to Alzheimer's disease process. This study aimed to investigate the relationship of hours of sleep and sleep quality in young and middle age-period with cerebral Aβ burden in elderly individuals with normal cognition. **Methods:** One hundred and twenty-two cognitively normal old adults (age range: 60-87 years), who participated in the Korean Brain Aging Study for Early Diagnosis and Prediction of Alzheimer's Disease (KBASE), were included. All subjects underwent comprehensive clinical and neuropsychological assessment, ^{11}Clabelled Pittsburgh Compound B (PiB) positron emission tomography (PET). Through structured clinical interview for each participant, mean hours of sleep and sleep quality were assessed for the following age-periods: before 20 years, in their 20-30s, and 40-50s. Current sleep quality was also assessed by using the Pittsburgh Sleep Quality Index (PSQI). Global cerebral Aβ deposition was defined as mean cortical PiB retention of the cortical regions including the frontal, lateral temporal, lateral parietal and precuneus/posterior cingulate cortices. **Results:** The poorer sleep quality in all the three younger age-periods was associated with higher mean cortical PiB retention even after controlling for age, gender, apolipoprotein E e4 status, and Hamilton Depression Rating Scale score. In contrast, mean hours of sleep in any young or middle age-period or current sleep quality measured by the PSQI were not related to mean cortical PiB retention. **Conclusions:** These findings suggest that poorer sleep quality, but not hours of sleep, in young and middle age-period may contribute to increased cerebral amyloid burden in old age.

Table
Correlation between sleep variables and Pittsburgh Compound B (PiB) retention

	Unadjusted		Mutivariable Adjusted*	
	Correlation Coefficient	P Value	Partial Correlation Coefficient	P Value
<20y sleep quality	0.246	0.006	0.224	0.015
<20y sleep duration	-0.050	0.582	-0.030	0.748
20-39y sleep quality	0.175	0.055	0.174	0.060
20-39y sleep duration	-0.002	0.984	-0.013	0.889
40-59y sleep quality	0.203	0.025	0.198	0.032
40-59y sleep duration	-0.058	0.526	-0.085	0.360
Total PSQI	0.010	0.912	0.044	0.637

*Adjusted for age, gender, apolipoprotein E e4 status, and Hamilton Depression Rating Scale score

IC-P-029 POLYMORPHISM IN CYTOCHROME P450 GENE IS ASSOCIATED WITH ALZHEIMER'S PATHOLOGY

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Background: The cytochromes P450 (CYP) are known for their role in metabolizing several endogenous and exogenous substrates. In