education (40%) and referrals. Scans stimulated referrals to develop a family plan of progressive support in 27% and to a psychiatrist in 20%. The most frequent change in care practices was referral to a Alzheimer clinical trial (53%). The diagnosis judged most likely before the scan changed in 27% and there was a 90% increase in diagnostic confidence after the amyloid PET. **Conclusions:** The classification of Alzheimer's pathology with amyloid PET frequently changes provider recommendations that depend upon a specific diagnosis. Changes in recommended non-drug care practices contribute to the value of amyloid PET. Further experience is needed to fully understand the role of amyloid classification technology in clinical practice.

IC-P-008 PREDICTION OF AMYLOID POSITIVITY WITH DEMOGRAPHIC, APOE AND NEUROPSYCHOLOGICAL INFORMATION IN COGNITIVELY NORMAL OLD ADULTS WITH SUBJECTIVE COGNITIVE DECLINE

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Background: There are evidences that subjectively experienced cognitive decline even at the stage of normal performance on cognitive tests is associated with an increased risk for future cognitive decline and Alzheimer's disease (AD) dementia. However, subjective cognitive decline (SCD) is unspecific, and related to various medical, psychiatric, and even cultural conditions. For the purpose of early detection of AD, therefore, identifying the characteristics of SCD at the preclinical stage of AD is very important. We aimed to elucidate the demographic, clinical, neuropsychological, and genetic characteristics associated with beta-amyloid (Aβ) deposition, and to search the best prediction model for Aβ positivity in cognitively normal (CN) adults with SCD. **Methods:** One-hundred thirty two CN elderly with SCD were recruited from the Korean Brain Aging Study for Early Diagnosis and Prediction of Alzheimer's Disease (KBASE). The presence of SCD was defined by the answer for a single question: "Do you feel that your cognitive function has declined compared to past?" All the subjects received comprehensive clinical and neuropsychological assessments, apolipoprotein E (APOE) genotyping, ^{11}C-labelled Pittsburgh Compound B (PiB) –PET imaging. Aβ positivity was determined if the SUVR value was > 1.4 in at least one of the four ROIs **Results:** Univariate analyses showed that Aβ positive group was older and had more APOE e4 carrier than Aβ negative group. In regard of neuropsychological performance, Aβ positive subjects had higher phonemic fluency score than Aβ negative ones. A series of multiple logistic regression analyses showed that the model including age, APOE genotype, and phonemic fluency score together had best predictive ability for Aβ positivity in CN subjects with SCD (prediction accuracy: 87.1%), although the models with each of the three variables or any two of them significantly predict Aβ positivity as well (accuracy: 81.8∼85.6%). **Conclusions:** Our findings suggest that older age, the presence of APOE e4 allele, and higher phonemic fluency increase the likelihood of preclinical AD in cognitively healthy old adults with subjective cognitive complaint. Higher phonemic fluency may be related to compensatory brain activation for Aβ pathology.

Table 2
Finally Selected Logistic Regression Model (model CNG)* for Amyloid positivity Prediction in SCD(+)

Variables	Regression Coefficient	Standard Error	Odd ratios	95% Confidence Interval	*p*
Intercept	-16.44	3.59	0.00		<0.001
Age	0.16	0.04	1.18	1.08-1.28	<0.001
Phonemic fluency	0.09	0.02	1.09	1.04-1.15	<0.001
ApoE4+	2.12	0.64	8.32	2.38-29.04	0.001

Abbreviations: Apo E4+, apolipoprotein ε4 carrier; SCD, subjective cognitive decline.

IC-P-009 OBSTRUCTIVE SLEEP APNEA IS ASSOCIATED WITH LONGITUDINAL INCREASES IN AMYLOID BURDEN IN ELDERLY MILD COGNITIVE IMPAIRMENT INDIVIDUALS

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Table 1
Results Obtained From Logistic Regression Analyses Designed to Select Appropriate Models for Amyloid positivity prediction in SCD(+)

Models	-2LL	χ^2	df	*p*	Diagnostic Accuracy (%)	Significance test for -2LL Difference
One-candidate model						
Model C Age	103.72	15.22	1	<0.001	81.8	
Model S Age at onset of SCD	107.07	11.88	1	0.001		
Model N Phonemic fluency	112.23	6.72	1	0.01	83.3	
Model G APOE4+	110.46	8.49	1	0.003	83.3	
Two-candidate model						
Model CN Age+ Phonemic fluency	92.44	26.51	2	<0.001	84.1	Model CN versus C: p=0.001 Model CN versus N: p=<0.001
Model CG Age+ APOE4+	95.64	23.31	2	<0.001	85.6	Model CG versus C: p=0.004 Model CG versus G: p=<0.001
Model NG Phonemic fluency+ APOE4+	101.22	17.73	2	<0.001	83.3	Model NG versus N: p=0.001 Model NG versus G: p=0.004
Three-candidate model						
Model CNG Age+ Phonemic fluency+ APOE4+	80.97	37.98	3	<0.001	87.1	Model CNG versus CN: p=0.001

Abbreviations: Apo E4+, apolipoprotein ε4 carrier; SCD, subjective cognitive decline.

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Background: Cross sectional analysis has shown an association between OSA severity and Aβ burden using amyloid-PET, globally and regionally in the precuneus among MCI patients. However, whether OSA accelerates longitudinal increases in Aβ burden in MCI patients is presently unclear. **Methods:** Study participants included a total of 798 subjects with a diagnosis of MCI and were a subset of the ADNI cohort (adni.loni.usc.edu). OSA was self-reported and participants were labeled either as OSA+, or OSA−. Aβ burden was determined by florbetapir SUVRs calculated by averaging across the 4 cortical regions and dividing this cortical summary ROI by a composite reference region. Mean and variance of the Aβ data at each time point by OSA status were determined. To test whether OSA is associated with the rate of change in Aβ data longitudinally, SAS PROC MIXED was used to fit the model with randomly varying intercepts and slopes allowing dependence on OSA status. The final model was adjusted for sex, body mass index and CPAP use status since there was no difference between OSA groups for APOE e4 status, age and history of cardiovascular disease. **Results:** At baseline, there was significant variation between subjects in mean Aβ-42 volumes (intercept) (mean SUVR; B = 0.0008, Z-value =11.02, $p < .0001$). A significant variation in the change (slope) in Aβ-42 volumes over time was also seen (mean SUVR; B = 0.0084, Z-value =11.63, $p < .0001$). The covariance between the baseline Aβ-42 level and Aβ-42 volume change over time indicated that SDB subjects experienced a faster increase in brain Aβ-42 volumes over time ($p < .0001$). The rate of change in Aβ-42 deposition also varied significantly across OSA groups over the follow-up period. **Conclusions:** Obstructive Sleep Apnea possibly facilitates longitudinal increases in amyloid burden in elderly Mild Cognitive Impairment individuals. Further research examining mechanisms underlying effects of OSA on the longitudinal increases in Aβ burden is needed.

IC-P-010 SLEEP DISORDERED BREATHING, APOE4 AND β-AMYLOID DEPOSITION IN COGNITIVELY NORMAL ELDERLY

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Background: Sleep Disordered Breathing (SDB) is commonly reported in the elderly, and recent studies in humans and animals describe associations between SDB and Alzheimer disease (AD). ApoE4 allele is considered the most important risk factor for sporadic AD. We examined whether SDB is associated with changes in amyloid burden in a sample of cognitively normal elderly. The interactive effect of SDB*APOE4 on amyloid burden was also examined. **Methods:** Data used were obtained from the ADNI database (adni.loni.usc.edu). Study participants included a total of 516 cognitively normal subjects and were a subset of the ADNI cohort. SDB was self-reported and participants were labeled SDB+, or SDB−. Brain Aβ-42 levels were determined at baseline and follow-up visits. Multi-level mixed effects linear regression models were used to examine the relationship between SDB and Aβ-42 volumes. First, we fit a linear regression model for each participant separately at each time point, and second, we regressed unknown time-specific regression coefficients against time. Our models were adjusted for sex, and body mass index. There was no difference between OSA groups for APOE e4 status, age and history of cardiovascular disease. The interactive effect of SDB*APOE4 on amyloid burden was also examined. **Results:** There was significant variation between subjects in mean Aβ-42 volumes at baseline (intercept) (mean SUVR; B = 0.006, $p > .0001$), as well as significant variation in the change in Aβ-42 volumes over time (slope) (mean SUVR; B = 0.006, $p > .0001$). The covariance between the baseline Aβ-42 level and Aβ-42 volume change over time indicated that SDB subjects experienced a faster increase in brain Aβ-42 volumes over time ($p > .0001$). The interactive effect of SDB*APOE4 on amyloid burden was not significant. **Conclusions:** Among community-dwelling cognitively normal older adults, SDB is associated with greater β-amyloid burden changes over time regardless of APOE4 status. This suggests that clinical interventions aimed at SDB, such as treatment with CPAP or dental appliances, implemented during the early phase in which tissue damage precedes clinical symptoms and neuronal dysfunction, may mitigate the progression of cognitive impairment.

IC-P-011 DETERMINATION OF NEURITIC VERSUS DIFFUSE PLAQUE CONTRIBUTION TO SIGNAL DERIVED FROM CN-FLUTEMETAMOL

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Background: Specificity and sensitivity of Flutemetamol (Vizamyl™) PET are high for detecting neuritic amyloid-beta plaques (NP). Several false-positive results from the Flutemetamol clinical-pathological study, as well as high retention of Flutemetamol in the striatum from AD cases, indicate that this radiotracer may also detect diffuse plaques (DP). The fluorescent derivative of Flutemetamol (CN-Flutemetamol) labels both plaque types in AD brain tissue sections, although NP show brighter fluorescence than DP. The current study explores quantitatively this relation in brain areas affected differentially by NP and DP. **Methods:** Tissue sections from the frontal cortex (FC, variable proportions of NP and DP) and caudate (CD, exclusively DP) from ten AD cases were processed using CN-Flutemetamol and analyzed for plaque load (% area occupied by plaques) and integrated density (a measure that integrates both size and fluorescence intensity of labeled plaques per defined field). A similar approach was used to assess total light output of individual NP and DP imaged using a DSU spinning disk confocal microscope. **Results:** All plaques in both regions were labeled with CN-Flutemetamol. CN-Flutemetamol positive total plaque load was similar in CD and FC (CD = 3.79 ± 0.64; FC = 5.81 ± 1.18; t-test p = 0.15), however, the integrated density in FC was greater than in the CD (CD=5024 ± 490.3; FC=11742 ± 568.2; t-test $p<0.0001$). Confocal analysis yielded similar results with total light output of NP greatly exceeding that of DP. **Conclusions:** For two regions with comparable plaque area coverage, but with different involvement of NP and DP, the region with a preponderance of NP yields greater overall CN-Flutemetamol fluorescence signal when area coverage and signal intensity are calculated as a single value (integrated density). These observations were confirmed by confocal image analysis of individual plaques, and they imply that in PET imaging studies, brain regions with high densities of NP exhibit greatest Flutemetamol retention.