amyloid had poorer longitudinal memory performance than the nonincreasing group. These effects, though subtle, may represent the earliest consequences of amyloid deposition that are detectable prior to the onset of amyloid positivity and any clinically-relevant cognitive dysfunction.

## IC-P-014 PREDICTION OF BETA-AMYLOID POSITIVITY IN MILD COGNITIVE IMPAIRMENT WITH DATA OBTAINED FROM ROUTINE MEMORY CLINIC PRACTICE

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**Background:** Although recent advances in neuroimaging techniques have enabled the detection of in vivo Alzheimer's disease (AD) pathology in human, only a limited number of memory clinics are able to utilize it in clinical practice due to high cost and low accessibility. This study, therefore, aimed to develop prediction models for the beta-amyloid (Aβ) positivity on amyloid positron emission tomography (PET) in mild cognitive impairment (MCI) individuals with data that are routinely obtained in memory clinic setting. **Methods:** Sixty seven MCI patients were included in this study. All subjects received clinical and neuropsychological assessments, laboratory evaluations for blood sample, magnetic resonance imaging, and $^{11}$C-labelled Pittsburgh Compound B (PiB) PET, For the development of Aβ positivity on PiB PET prediction models, all the variables were first categorized into four groups: clinical (C), neuropsychological (N), laboratory (L), and imaging (I) groups. In each group, the variables that showed significant or trend level association with Aβ positivity in univariate analyses were selected for further analyses. The selected variables of each group were combined sequentially in the prediction models, and logit values were calculated. Finally, with the logit values, the receiver operating characteristic (ROC) analyses were performed for each model to calculate the area under the curve (AUC). **Results:** For each group, following variables were selected: Total scores of geriatric depression scale, subjective memory complaint questionnaire, trait anxiety, blessed dementia scale-activities of daily living and history of hypertension for group C; raw scores of word list recall and recognition, and constructional recall for group N; triiodothyronine, high-density lipoprotein cholesterol, erythrocyte sedimentation rate, and APOE 4 positivity for group L; adjusted hippocampal volume for group I. In the ROC analyses, AUC for the prediction models were as follows: 0.793 for group C, 0.894 for combined group C+N, 0.956 for combined group C+N+L, and 0.944 for combined group C+N+L+I. **Conclusions:** The findings suggest that the systematic combinations of data obtained from routine clinical practice may be successfully used to predict Aβ positivity in MCI individuals.

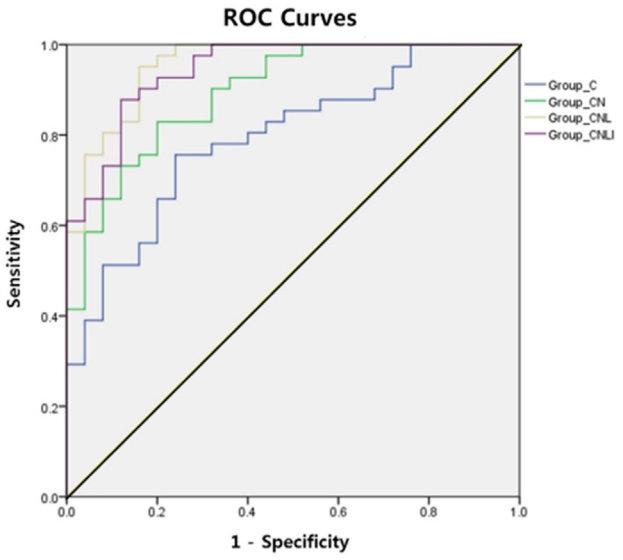


Figure 1. The receiver operating characteristic (ROC) curves of each prediction model for amyloid positivity.

Table 1
Comparison of the area under the ROC curves of each prediction model

| Model | AUC | SD | p | 95% CI |
|---|---|---|---|---|
| Group C | 0.793 | 0.055 | <0.001 | 0.685-0.901 |
| Group C+N | 0.894 | 0.039 | <0.001 | 0.818-0.969 |
| Group C+N+L | 0.956 | 0.023 | <0.001 | 0.910-1.000 |
| Group C+N+L+I | 0.944 | 0.026 | <0.001 | 0.593-0.996 |

Abbreviations: ROC, receiver operating characteristic; AUC, area under the curve; SD, standard deviation; CI, confidence interval.

## IC-P-015 ASSOCIATION OF PAST AND CURRENT BODY MASS INDEX WITH BRAIN AMYLOID DEPOSITION AND NEURODEGENERATION IN COGNITIVELY NORMAL ELDERLY

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**Background:** Both low and high body mass index(BMI) has been associated with cognitive impairment and Alzheimer disease(AD) dementia. Nevertheless, very little information is available for the association between BMI over life course and brain amyloid beta(Aβ) burden and AD-specific neurodegeneration. In this study, we examined the relationship of past and current BMI with in vivo cerebral Aβ deposition and AD-signature region cortical thickness in cognitively normal elderly population. **Methods:** Two-hundred twelve cognitively normal elderly subjects aged

Table 1
Analyses of association between AD biomarker with BMI measure at each age

| | Global Aβ deposition | | | | AD signature cortical thickness | | | |
|---|---|---|---|---|---|---|---|---|
| | B | SE | β | P | B | SE | β | P |
| Current BMI | -0.008 | 0.006 | -0.091 | 0.184 | 0.008 | 0.004 | 0.139 | **0.018** |
| Early adult BMI | -0.011 | 0.006 | -0.118 | **0.082** | 0.007 | 0.004 | 0.108 | **0.063** |
| Mid life BMI | -0.016 | 0.007 | -0.152 | **0.025** | 0.003 | 0.004 | 0.039 | 0.504 |
| Past BMI(20 to 50) | -0.015 | 0.007 | -0.141 | **0.037** | 0.006 | 0.004 | 0.083 | 0.158 |

Abbreviations: BMI=body mass index. All analyses were Adjusted by age, gender, APOE4 carrier status, Vascular risk factors.

60-90, who participated in the Korean Brain Aging Study for Early Diagnosis and Prediction of Alzheimer's Disease (KBASE), were included in this study. All the subjects underwent comprehensive clinical and neuropsychological assessment, 11C-labelled Pittsburgh Compound B (PiB) positron emission tomography (PET), and Brain Magnetic resonance imaging(MRI). Mean cortical thickness values were obtained from AD-signature regions including the entorhinal, inferior temporal, middle temporal, and fusiform gyrus. BMI values for past lifetime periods((i.e., young adulthood: 20-30s and midlife: 40-50s) were calculated from self-recalled body weight and height, and current BMI was calculated from measured body weight and height. Multiple regression analyses were performed controlling age, gender, APOE e4 carrier status, and vascular risk scores. **Results:** For the overall subjects, past BMI, BMI at young adulthood in particular, was negatively associated with global cerebral Aβ deposition, while current BMI was not. In contrast, current BMI showed significant positive association with AD-signature region cortical thickness, while past BMI did not. When subgroup analyses were performed for each gender, very similar relationships between lifetime BMIs and Aβ deposition and cortical thickness were still found for males, whereas no significant association was observed between the variables for females. **Conclusions:** Our results suggest that relatively low past BMI, especially in male, may contribute to increase of cerebral Aβ burden and the risk of AD in late-life, while lower current BMI appears related with emerging neurodegeneration. The findings should be cautiously interpreted considering most of the subjects were in normal range for past and current BMI, and only small proportion of them were in overweight or obese state.

Table 2
Analyses of association between **PiB retention** and BMI at each age and sex group in participant

| | Women | | Men | |
|---|---|---|---|---|
| | β | P value | β | P value |
| Early adult BMI | -0.002 | 0.786 | -0.038 | **0.003** |
| Midlife BMI | 0.003 | 0.673 | -0.028 | **0.009** |
| Past BMI(20 to 50) | 0.001 | 0.944 | -0.036 | **0.003** |
| Current BMI | 0.003 | 0.690 | -0.021 | **0.042** |

BMI=body mass index. All analysis were Adjusted for age, APOE4 carrier status, vascular risk factors.

Table 3
Analyses of association between **AD sig cortical thickness** and BMI at each age and sex group in participant

| | Women | | Men | |
|---|---|---|---|---|
| | β | P value | B | P value |
| Early adult BMI | 0.002 | 0.734 | 0.005 | 0.532 |
| Midlife BMI | 0.003 | 0.600 | 0.014 | **0.022** |
| Past BMI(20 to 50) | 0.003 | 0.621 | 0.012 | **0.098** |
| Current BMI | 0.006 | 0.211 | 0.013 | **0.023** |

BMI=body mass index. All analysis were Adjusted for age, APOE4 carrier status, vascular risk factors.

## IC-P-016 INVESTIGATING THE CLINICAL IMPACT OF [$^{18}$F]FLUTEMETAMOL PET IN A TERTIARY MEMORY CLINIC SETTING IN PATIENTS WITH UNCERTAIN DIAGNOSIS

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**Background:** Since the first application of carbon-11 Pittsburgh Compound-B ([$^{11}$C]PIB) PET more than a decade ago, amyloid PET has been instrumental in advancing the research agenda for Alzheimer's disease (AD). By detecting a core feature of AD pathology, amyloid PET holds potential in clinical settings, particularly given the high rate of misdiagnosis. Clinical studies using recently approved amyloid PET tracers, however, have far been few, with these mainly in highly selected research cohorts. The aim of the present study was thus to investigate the added clinical value of [$^{18}$F]flutemetamol PET in memory clinic patients whose diagnosis remained uncertain following routine clinical work up. **Methods:** 135 patients were included from the Department of Geriatric Medicine, Karolinska University Hospital, Huddinge, Sweden, following referral from primary care physicians. Following standard diagnostic workup, including medical and neurological examination, clinical chemistry (including CSF A$\beta_{1-42}$), rating batteries for depression and neuropsychiatric symptoms, neuropsychological assessment, and structural imaging, the clinical picture remained unclear. [$^{18}$F]flutemetamol PET was thus performed, using a Biograph mCT PET/CT (Siemens/CTI, Knoxville, TN), with the acquisition protocol consisting of a static 20-min scan, 90-min post-injection of 185 MBq. In addition to visual assessment by an experienced nuclear medicine physician, [$^{18}$F]flutemetamol uptake was quantified on a region of interest basis using a fully automated software (Hermes Hybrid BRASS). Diagnoses before and after [$^{18}$F]flutemetamol investigations were reached using a multidisciplinary consensus based approach. **Results:** Based on preliminary results from 61 subjects (38 MCI, 13 AD, 6 dementia NOS, 2 SCI, one FTD, and one DLB), [$^{18}$F]flutemetamol investigations led to a change in diagnosis in 68% of patients. Agreement between visual and quantitative assessment of [$^{18}$F]flutemetamol images was high (89%).