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ALZHEIMER'S IMAGING CONSORTIUM (AIC)
IC-01
TAU IMAGING

IC-01-01 PREDICTORS OF REGIONAL TAU-PET UPTAKE: MAYO CLINIC STUDY OF AGING

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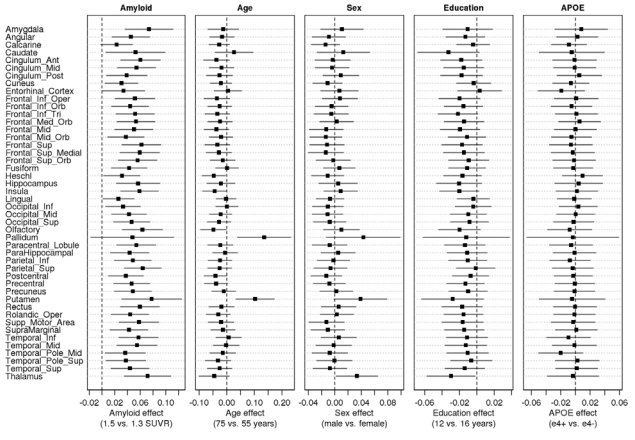
Background: To investigate the effects of age, sex, APOE4, education, and amyloid deposition (via ^{11}C PIB-PET) on the degree of regional tau deposition (via AV1451) in cognitively normal elderly. **Methods:** We identified 168 cognitively normal participants aged 52-94 years in the population-based Mayo Clinic Study of Aging who had undergone APOE4 genotyping, with Tau-PET, and PIB-PET scans available. The subjects identified had a median age of 69, 54% male, 29% APOE4 positive, median education of 16, and median PIB SUVR of 1.34. Regional uptake of Tau-PET was assessed in 46 atlas regions by summarizing the median uptake in each region scaled to the median uptake in the cerebellar crus (SUVR). For each region, we fit a linear regression model with Tau-PET SUVR as the response variable and the following potential predictor variables: global amyloid PET, age, sex, education, and APOE4 genotype. In a preliminary regional correlation analyses of PIB with Tau-PET SUVRs, we found that correlations were driven mainly by global amyloid levels. Therefore, we used global and not regional PIB SUVRs in the models presented here. **Results:** The results are shown in Figure 1 in which each row represents an independent regional model fit and summarizes the estimated mean (95% confidence interval) difference in tau deposition for each covariate: amyloid PET, age, sex, education, and APOE4. Although effect sizes varied across regions, we found strong evidence that higher levels of amyloid were associated with increased levels of tau after accounting for age, sex, education, and APOE ($p<0.01$). Except in the pallidum and putamen ($p<0.01$), age was not independently associated with increased levels of tau. We did not see independent effects of sex, education, or APOE4 ($p>0.01$) in any of the regions. **Conclusions:** Global amyloid burden is predictive of tau deposition in a large number of brain regions after accounting for age, sex, education, and APOE4. Independent effects of age were localized to the pallidum and the putamen where non-specific Tau-PET binding has been observed.



IC-01-02 THE ASSOCIATION BETWEEN AB AND TAU ACCUMULATIONS AND ITS INFLUENCE ON CLINICAL FEATURES IN AGING AND ALZHEIMER'S DISEASE SPECTRUM BRAINS: [^{11}C]PBB3 PET STUDY

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Background: Senile plaques and fibrillary tau inclusions are neuropathological hallmarks of Alzheimer's disease (AD). Recent neuroimaging and neuropathological studies have revealed that proteinopathies represented by amyloid-β (Aβ) and tau accumulations may occur independently and concurrently as exemplified by primary age-related tauopathy and AD, respectively. To elucidate interactions between Aβ and tau accumulations and its influence on clinical features, we performed a cross-sectional positron emission tomography (PET) study in cognitively healthy and impaired individuals. **Methods:** Participants were 20 patients with AD, 17 patients with mild cognitive impairment (MCI), 24 age-matched healthy controls (oHCs) and 10 young HCs. Parametric [^{11}C] PBB3-, [^{11}C]PiB- and [^{18}F]FDG-PET images were genrated for estimating regional Aβ, tau and brain glucose metabolism, respectively, by voxel-based calculation of standardized uptake value ratio (SUVR) to the cerebellum at 30-50 minutes, 50-70 minutes and 30-60 minutes after radiotracer injections, respectively. We also performed three-dimensional T1-weighted MRI and estimated whole grey matter volume for each subject. **Results:** All AD patients, 10 of 17 MCI patients, and 3 of 24 oHCs were classified as PiB(+), and others were classified as PiB(-) by visual assessment of [^{11}C]PiB SUVR images. PBB3(+) tau deposits were observed in medial temporal regions including the hippocampus of a subset of PiB(-) oHCs, and this was associated with subclinical memory declines. Self-expansion of tau deposition to restricted areas without Aβ was also demonstrated in these and PiB(-) MCI subjects. PBB3(+) tau spread to Aβ-rich areas in PiB(+) oHCs and MCI subjects, and was further expanded to widespread neocortical regions in AD patients. In PiB(+) MCI and AD groups, dementia scales