

Table
Changes in the diagnostic confidence between scenarios and over the time.

Variables	T00	T06	T12	T06-T00		T12-T06		T12-T00	
	mean (SD)	mean (SD)	mean (SD)	mean_diff (SD_diff)	Cohen's <i>d</i>	mean_diff (SD_diff)	Cohen's <i>d</i>	mean_diff (SD_diff)	Cohen's <i>d</i>
Befief (0-10)	5.86 (1.47)	6.29 (1.59)	7.00 (2.02)	0.43 (1.35)	<i>0.32</i>	0.71 (1.60)	<i>0.45</i>	1.14 (1.87)	<i>0.61</i>
Scenario 1 (0-100%)	54.29 (31.55)	66.21 (31.85)	55.50 (30.30)	11.93 (31.57)	<i>0.38</i>	-10.71 (26.84)	-0.40	1.21 (38.72)	0.03
Scenario 2 (0-100%)	67.14 (25.17)	55.00 (30.57)	71.07 (29.96)	-12.14 (26.10)	-0.46	16.07 (32.74)	<i>0.49</i>	3.93 (11.45)	<i>0.34</i>
Scenario 3 (0-100%)	23.33 (26.71)	20.48 (23.34)	18.09 (18.06)	-2.86 (33.66)	-0.08	-2.38 (25.19)	-0.09	-5.24 (24.06)	-0.22

Values for the three scenarios refer to percentages. Cohen's *d* is an index of effect size: defined as the difference between two means divided by a standard deviation for the data. Mean_diff is the difference between two time points, SD_diff is calculated with the following formula: $SD_pre * \sqrt{2 * (1 - \rho)}$, where ρ is the correlation coefficient. Values are in italics or bold when the Cohen's *d* is at least 'small' (≥ 0.20): italics for positive difference and bold for negative.

as covariate. Effect Size indices (ES; Cohen's *d* for repeated measures) were also computed. We analyzed the seven dementia experts who both filled out the questionnaire at all time points and enrolled a minimum of 15 patients. **Results:** 'Belief' did not affect the probability of diagnostic change in different scenarios and over time ($p=0.655$). The probability of diagnostic change did not change significantly throughout the study for all scenarios ($p=0.970$). Due to the very small sample size, we considered ES greater than 0.20 ('small'). These were consistent with: i) a trend in increase in the belief on a causal role of AB in AD and ii) a growth of the positive predictive value of the amyloid-PET in 'NonAD-positive' over the whole study (Table). **Conclusions:** Beliefs on the pathogenic role of amyloid-B do not affect the intended clinical use of amyloid-PET in the course of a naturalistic study using amyloid-PET. ES suggested an increase of the belief in a causal role of AB in AD pathogenesis over the study. Moreover, an increased use of the positive predictive value of amyloid-PET emerged after practical usage of amyloid-PET. These results will be compared with the actual use of amyloid-PET by the same physicians.

IC-P-006

REGIONAL BETA-AMYLOID PET COMPARISON ACROSS ATYPICAL AND TYPICAL VARIANTS OF ALZHEIMER'S DISEASE

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Background: Typical dementia of the Alzheimer's type (DAT) presents as a primary amnesic syndrome, and three "atypical" variants are now recognized. Two of these atypical variants include posterior cortical atrophy (PCA), which presents with visual deficits and the logopenic variant of primary progressive aphasia (lvPPA), which presents with primary language disruption, both of which are commonly due to Alzheimer's disease pathology. Here, we compare these Alzheimer's disease variants on the pattern of beta-amyloid deposition on PiB PET, as well as cognitive measures. **Methods:** We matched 27 PCA, 50 LPA, and 77 DAT patients on age, gender, and disease duration as best as possible. Only subjects with positive beta-amyloid deposition on PiB PET, defined using a standardized uptake value ratio (SUVR) of ≥ 1.5 , were included. All voxels in the PiB-PET images were divided by median uptake in cerebellum and transformed into template space. Partial volume correction (PVC) was performed. Voxel-level comparisons were performed across groups both with and without PVC using SPM5. **Results:** were assessed after correction for multiple

comparisons using family-wise error at $p < 0.05$. Subjects also completed memory, language, and visuospatial tests that may have been used to augment DAT and PCA diagnosis, but was not used in diagnosis of LPA. **Results:** Cognitively, DAT subjects had poorer memory than lvPPA and PCA subjects, lvPPA subjects had poorer language scores than DAT and PCA subjects, and PCA had poorer visuospatial scores than lvPPA and DAT subjects. The groups did not differ on global SUVR. Regional distribution of PiB-PET was similarly widespread in each of the three groups, although PCA showed greater uptake in bilateral occipital lobe compared to DAT, and greater uptake in right occipital lobe compared to lvPPA. These findings were observed both with and without PVC. No other regional PiB-PET differences were observed across groups. **Conclusions:** These findings suggest that while beta-amyloid deposition is typically diffuse in Alzheimer's disease variants, regional differences exist as compared to DAT, although this DAT group may represent those with an earlier onset. The occipital lobe is particularly vulnerable to beta-amyloid deposition, as well as neurodegeneration, in PCA.

IC-P-007

BRAIN AMYLOIDOSIS AND COGNITIVE DECLINE IN MCI: 12-MONTH FOLLOW-UP

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Background: Two previous studies demonstrated greater cognitive decline in MCI patients with positive amyloid-PET compared to negative MCI, as assessed by several neuropsychological tests, over 18 (Doraiswamy et al., 2012) and 36 months (Doraiswamy et al., 2014) follow-up. Our aim is to evaluate whether positive amyloid-PET scans are predictive of greater cognitive decline than negative scans over a shorter 12 months period. **Methods:** A naturalistic series of seventy-six MCI patients underwent amyloid-PET with ¹⁸F-Florbetapir (FBP-PET), and homogeneous clinical and neuropsychological multi-domain assessment before FBP-PET and after 12 months (12-FU). For each neuropsychological test we constructed a linear mixed model with scores as the dependent variable, and 'FBP-PET result' (positive or negative) and 'time' (baseline or 12-FU) as main factors. 'Age' and 'education' were included as covariates. Effect size indices (partial Eta-squared, η_p^2) were also computed (0.02, 0.13 and 0.26 referring respectively to small, medium and large Eta-squared). **Results:**