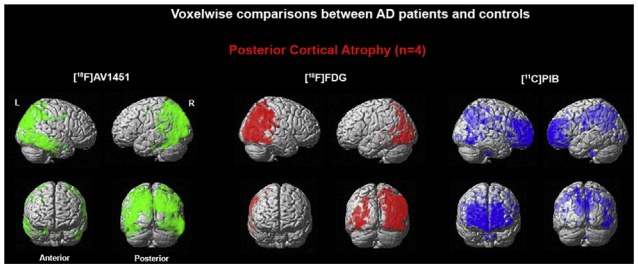

pathology) and [^{18}F]FDG (glucose metabolism) PET. **Methods:** PET scans were performed in 5 posterior cortical atrophy (PCA), 4 logopenic variant primary progressive aphasia (lvPPA), 1 early-onset AD and 2 (memory-predominant) late-onset AD patients (all PIB+) and in 19 cognitively normal controls (Table 1). We created SUVr images for [^{18}F]AV1451 (80-100 minutes, gray matter cerebellum as reference region) and [^{18}F]FDG (30-60 minutes, pons-normalized), and DVR images for [^{11}C]PIB (0-90 minutes, gray matter cerebellum as reference region). We visually assessed [^{18}F]AV1451, [^{11}C]PIB and [^{18}F]FDG uptake patterns in 3 distinct AD variants, and performed voxel-wise contrasts (in SPM) between PCA patients and controls. **Results:** Figure 1 shows asymmetric [^{18}F]AV1451 uptake in parietal, temporal and frontal regions (left>right) in a patient with lvPPA, a classical temporoparietal pattern in a patient with memory-predominant AD, and mainly occipitotemporal and occipitoparietal involvement in a PCA patient. [^{18}F]AV1451 and [^{18}F]FDG appeared strikingly as mirror images, with regions of high [^{18}F] AV1451 uptake corresponding to low [^{18}F]FDG uptake and vice versa, while [^{11}C]PIB binding was observed throughout the association neocortex. Voxelwise contrasts with [^{18}F] AV1451 and [^{18}F]FDG showed that PCA patients significantly differed from controls in clinically affected posterior brain regions, while [^{11}C]PIB binding was greater in both posterior regions and in clinically less affected anterior regions (Figure 2). **Conclusions:** [^{18}F]AV1451 was specifically retained in brain regions closely related to the clinical presentation across distinct AD variants and overlapped substantially with hypometabolic regions in PCA, while [^{11}C]PIB binding was more diffuse and showed less overlap with [^{18}F]FDG uptake. This provides preliminary *in-vivo* evidence that hypometabolism and symptomatology are more closely linked to tau than to Aβ pathology.

IC-02-03 HYPOMETABOLISM OF THE POSTERIOR CINGULATE CORTEX IS NOT RESTRICTED TO ALZHEIMER'S DISEASE

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Background: Hypometabolism of the posterior cingulate cortex (PCC) is associated with Alzheimer's disease (AD). PCC vulnerability, however, could also be present in other neurodegenerative diseases, such as behavioural variant of frontotemporal dementia (bvFTD), and normal aging. The aims of this study were to assess 1) the proportion of AD, bvFTD and cognitive normal subjects (CN) with PCC hypometabolism, and 2) the relationship between PCC metabolism and demographical, neuropsychological, and neurobiological characteristics in AD, bvFTD and CN. **Methods:** We included 33 probable bvFTD patients (Neary criteria, low likelihood of AD pathophysiology based on CSF amyloid-beta$_{1-42}$ >550ng/l), 82 probable AD patients (National Institute on Aging-Alzheimer's Association workgroups core criteria, high likelihood of AD pathophysiology based on CSF tau/amyloid-beta$_{1-42}$ >.52ng/l), and 26 CN (22 subjects with subjective memory complaints and 4 healthy controls, CSF amyloid-beta$_{1-42}$ >550ng/l). Glucose metabolism was assessed using [^{18}F]FDG-PET. Parametric images of standardized uptake value ratios (SUVr) using cerebellar grey matter as reference tissue were generated. First, we defined PCC hypometabolism based on the Receiver Operating Characteristic (ROC) separating AD from CN, to assess the relative prevalence of PCC hypometabolism in AD, bvFTD and CN. Second, we explored relationships between PCC metabolism and demographics (age, sex, and education), Mini-Mental State Examination (MMSE), neuropsychological tests, CSF biomarkers (amyloid-beta$_{1-42}$, tau and phosphorylated tau), and APOE genotype within diagnostic groups using linear regression analyses or ANOVA where appropriate. **Results:** Mean age was 63±7 (AD), 65±8 (bvFTD) and 61±8 (CN) years old. PCC [^{18}F]FDG SUVr was CN > bvFTD > AD. Based on optimal discrimination of AD and CN, the PCC [^{18}F]FDG SUVr cut-off was set at 1.052, resulting in PCC hypometabolism in 78% (AD), 33% (bvFTD), and 23% (CN). PCC [^{18}F]FDG SUVr was associated with age in CN (beta±SE: -.007±.002, p=.002) and bvFTD (beta±SE: -.007±.003, p=.011), not in AD. **Conclusions:** PCC hypometabolism was present in 33% of bvFTD and 23% of CN. PCC [^{18}F]FDG SUVr was associated with age in bvFTD and CN. In the context of diagnostic work-up of dementia, it is important to realize that PCC vulnerability is not restricted to AD, but could be present in bvFTD and normal aging.

IC-02-04 DIRECT COMPARISONS BETWEEN ATROPHY AND HYPOMETABOLISM IN SEMANTIC DEMENTIA AND ALZHEIMER'S DISEASE SUGGEST DISTINCT NEUROPATHOLOGICAL PROCESSES

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Background: Direct comparisons between atrophy and hypometabolism in Alzheimer's disease (AD) have allowed unraveling