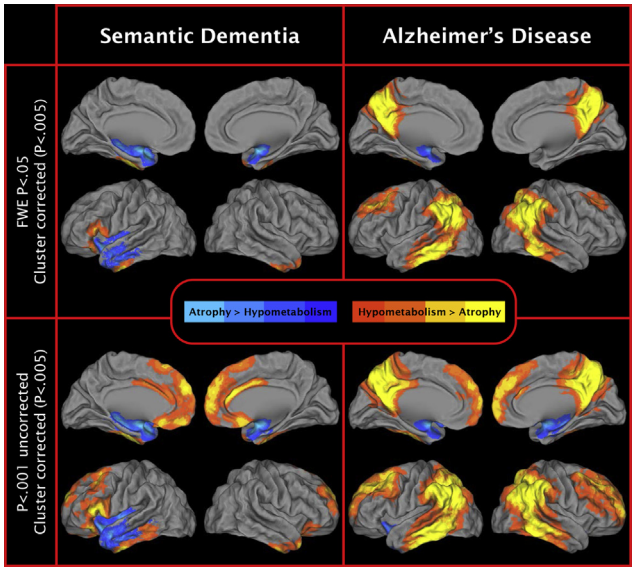
Therefore, the aim of the present study was to compare atrophy *versus* hypometabolism discrepancies in SD to that observed in AD in order to highlight similarities and differences in the pathological processes between both diseases. **Methods:** Sixteen patients with SD, 24 patients with AD and 39 healthy controls matched for age, sex and years of education underwent both structural MRI and 18F-fluorodeoxyglucose PET scans. Images were spatially normalized using dartel in SPM. Age-adjusted Z-score maps were then computed for each patient and each imaging modality using the healthy controls group as a reference. Direct between-modality voxel-wise comparisons were then performed within each patient group. **Results:** Between-modality analyses highlighted more atrophy than hypometabolism in the medial temporal lobe in both SD and AD (FWE $p<.05$). By contrast, more hypometabolism than atrophy was found in extended medial and lateral parietal regions, temporo-parietal and frontal areas in AD, and in much more restricted brain regions in SD, i.e. mainly in the prefrontal regions when using a more liberal threshold ($p<.001$ uncorrected). **Conclusions:** Interestingly, both neurodegenerative disorders were found to be characterized by stronger atrophy than hypometabolism in the medial temporal lobe. However, unlike in AD, the pattern of hypometabolism only slightly differed from the pattern of atrophy in SD. This likely reflects distinct neuropathological processes in both diseases with much less distant effect of neuronal damage in the hippocampal formation in SD. Yet, the excessive prefrontal hypometabolism in SD might as well reflect diaschisis, or the higher sensitivity of 18F-fluorodeoxyglucose PET than structural MRI to detect the underlying neuropathology. Altogether, these findings suggest that the pathological mechanisms affecting the brain might be more homogeneous in SD than in AD.



P2-138 EARLY FRAME OF PIB AND FDG IN AUTOSOMAL DOMINANT ALZHEIMER'S DISEASE: SIMILARITY, DISCREPANCY, AND CLINICAL IMPLICATION

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Background: Alzheimer's disease (AD), the leading cause of dementia in the elderly, can affect individuals in their thirties in autosomal dominant form. The Imaging Core of the Dominantly Inherited Alzheimer Network (DIAN) aims to characterize transition from preclinical to symptomatic disease using imaging biomarkers. Decreases in cerebral glucose metabolism in the parietal lobe are detectable 10 years before the estimated year of symptom onset (EYO) (Benzinger, Blazey et al., 2013) and may represent synaptic dysfunction. In sporadic AD, studies have shown that early perfusion frames of amyloid imaging with [11C]-Pittsburgh Compound B PiB (ePiB) correlate well with glucose metabolism (Rostomian et al., 2011). Here, we evaluated whether ePiB is a reasonable surrogate marker for synaptic dysfunction, in comparison to glucose metabolism hypometabolism, and how ePiB changes with the disease progression. **Methods:** DIAN participants (n=110), including 65 asymptomatic and symptomatic mutation carriers (MC), underwent full dynamic PiB-PET and also had [18F]-fluorodeoxyglucose (FDG) PET and volumetric brain MRI. The MRI was used to register the PET images. A standardized uptake value ratio (SUVR) from MR segmented PiB and FDG regions. An ePiB image with 1-9 min time frames was selected. Voxel-wise spatial correlation between FDG and ePiB was performed for each participant. The mutation and cognitive status were taken into account in the analyses. For each imaging modality, relationship with EYO was evaluated with linear mixed models on specific regions such as inferior parietal and precuneus cortices. **Results:** FDG and ePiB were visually similar and showed high spatial correlation with an average of 0.8 ± 0.04 regardless of the mutation or cognitive status. As we have previously found, the association between FDG and EYO significantly differs between MC and non-carrier groups (p-value<0.001 and p-value<0.01 for inferior parietal and precuneus, respectively). However, these associations were not significant between ePiB and EYO. **Conclusions:** Our findings show that ePiB is strongly correlated with FDG within the same individual. However, ePiB does not display the same sensitivity as FDG to reflect disease progression in this population. Further studies are needed to fully determine the utility of ePiB measurements in clinic.

P2-139 HIPPOCAMPAL MRI TEXTURE IS RELATED TO HIPPOCAMPAL GLUCOSE METABOLISM

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Background: We previously saw that hippocampal magnetic resonance imaging (MRI) texture provided-volume independent information in Alzheimer's disease (AD) diagnosis (AAIC 2012, P1-157) and predicted conversion from mild cognitive impairment (MCI) to AD (AAIC 2013, P3-085). However, it is not known which biological phenomena of the disease process texture reflect. The purpose of this study was to investigate a potential relationship between texture and tau-mediated neuronal injury as reflected by reduced glucose metabolism in FDG-PET. **Methods:** The study dataset consisted of the 215