to produce cortical thickness values. Vertex wise analyses were performed with Qdec comparing cognitively normal (CN) individuals (Clinical Dementia Rating, CDR, 0, N=500) and cognitively impaired (CI) individuals (CDR 0.5, N=145) controlling for APOE genotype, gender, and age and correcting for multiple comparisons ($p < 0.0001$ threshold). Additional analyses compared between CN individuals who remained stable and those that converted (CDR $>$ 0 within two years of MR visit, N=39). **Results:** The cortical thickness clusters (Figure 1) for the CI individuals when compared to the CN individuals showed prominent differences in the temporal and anterior regions of the brain. Additionally, when compared to the CN individuals, the converter individuals when compared to the CN individuals showed differences primarily in the temporal regions (Figure 2). **Conclusions:** OASIS-3 is a valuable resource that may facilitate research in Alzheimer Disease. Previous studies have investigated AD cortical signatures (Dickerson et al., 2009; Wang et al., 2015) as MRI biomarkers. Here we reproduce the cortical thinning pattern for cognitively impaired individuals and additionally identify locations of cortical thinning in CN individuals who later progress to dementia. The converter atrophy pattern may be utilized as a MR screening tool to help identify at-risk individuals and avoid performing PET scans.

P3-440 COMPARING THE CENTILOID SCALE FOR PITTSBURGH COMPOUND B AND FLORBETAPIR IN LONGITUDINAL PET STUDIES OF SPORADIC AD

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Background: Amyloid imaging with Positron Emission Tomography (PET) plays a critical role in research and clinical assessment of Alzheimer's disease (AD). Radiopharmaceutical tracers, such as [^{11}C]Pittsburgh Compound B (PiB) and [^{18}F]florbetapir, were developed to provide *in vivo* quantitative estimates of amyloid in humans as an AD biomarker. However, differences in tracer properties and analysis methods produce significant variability in estimates of amyloid burden. To address this issue, the Centiloid Working Group recommended amyloid SUVRs be converted to a standardized scale called Centiloid[1] for across tracer comparisons. We provide the first comparison of Centiloid values, without and with partial volume correction (PVC), for PiB and florbetapir in longitudinal PET studies of sporadic AD. **Methods:** Baseline and follow-up longitudinal florbetapir (N=26) and PiB (N=54) were obtained from a sporadic AD cohort with high amyloid burden at baseline. SUVRs were calculated between 50-70 minutes post-injection for florbetapir and 30-60 minutes post-injection for PiB with cerebellar grey as a reference and a regional spread function applied for PVC. A 3D sagittal T1-weighted head MR was also acquired to derive regions to average into a global cortical measure of

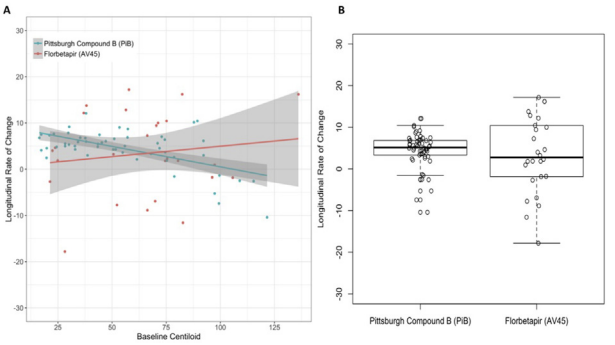

Figure 1. Annualized rate of change of amyloid burden using PiB and florbetapir in the longitudinal sporadic AD cohort. A) As a function of baseline amyloid in Centiloid. Florbetapir showed greater longitudinal variability than PiB. B) Box plot of annualized rate of change measurement for the two tracers. Despite greater variability within the annualized rate of change for florbetapir, both PiB and florbetapir showed comparable annualized rate of change on the Centiloid scale (p=0.55).

amyloid. The average cortical non-PVC and PVC SUVRs were converted to Centiloid for PiB and florbetapir. Annualized rate of change was derived from change in PVC Centiloid over time for each participant from a simple linear model and assessed by paired t-tests. **Results:** Application of PVC reduced overall intra-individual variability and increased the effect sizes of rates of change for both PiB and florbetapir. However, the rates of change were statistically significant only in PiB ($p<.0001$ for both PVC and non-PVC, respectively). Rate of change was not significantly different between the two tracers (p=0.55) for PVC Centiloid but greater variability existed within the florbetapir measurement. **Conclusions:** Florbetapir amyloid measurements in general had higher variability that significantly affected the tracer's ability to detect subtle amyloid burden compared to PiB. Further studies are needed to characterize tracer performance longitudinally. **References:** [1]Klunk WE, Koeppe RA, Price JC, Benzinger TLS, Devous Sr MD, Jagust WJ, et al. The Centiloid Project: Standardizing quantitative amyloid plaque estimation by PET. Alzheimer Dement 2015;11:P1-15.

P3-441 LOGICAL MEMORY DEFICITS ACROSS ALZHEIMER'S DISEASE SPECTRUM ARE ASSOCIATED WITH PATTERNS OF A TAU PROPAGATION PREDICTED BY BRAAK STAGING

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Background: The biological underpinnings of logical memory deficits remain debated in neurological conditions. Although the stages of neurofibrillary tangle (NFT) formation during the course of AD