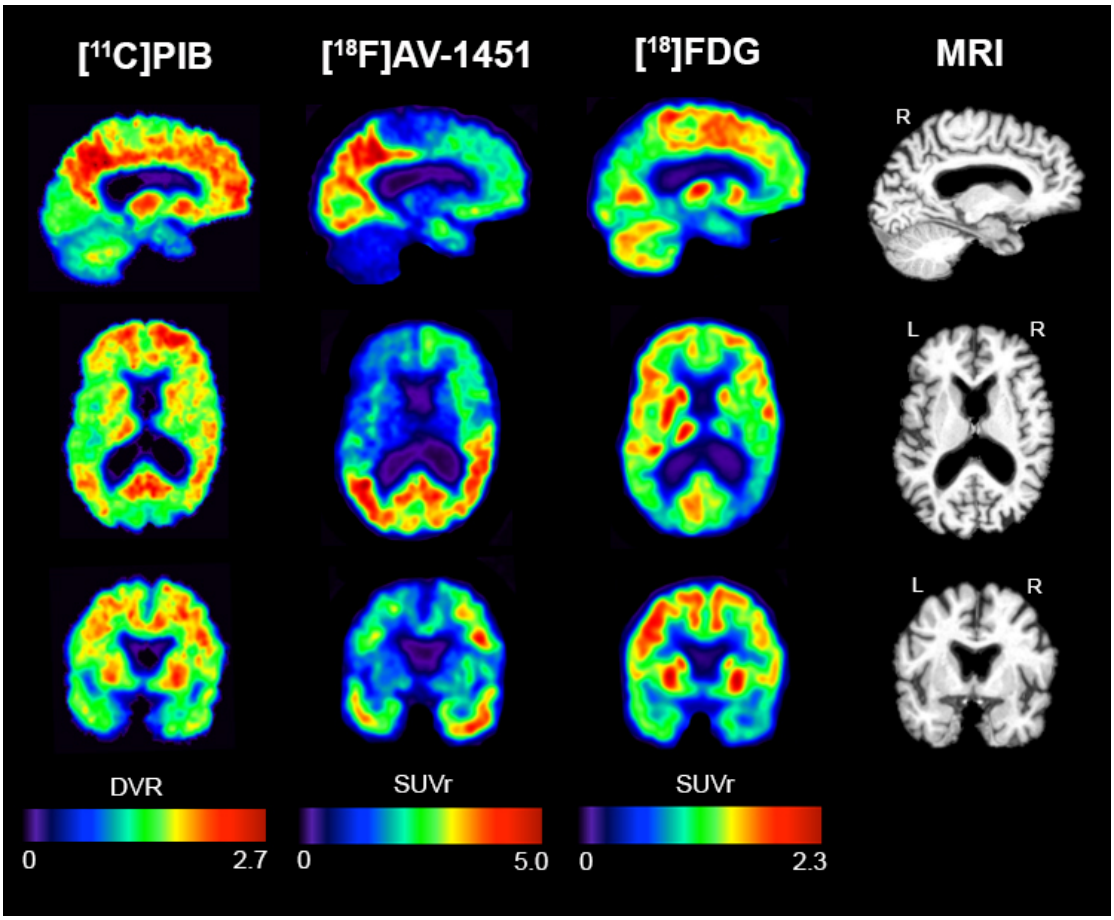

Figure 1. This case study of a patient with posterior cortical atrophy shows shows that AV1451 PET (tau) is distributed in regions that are hypometabolic on FDG PET, while PIB (amyloid) is present diffusely throughout the brain. This suggests that tau is more tightly correlated to clinical symptoms and patterns of neurodegeneration.

From: Ossenkoppele et al. [2015] Annals of Neurology.

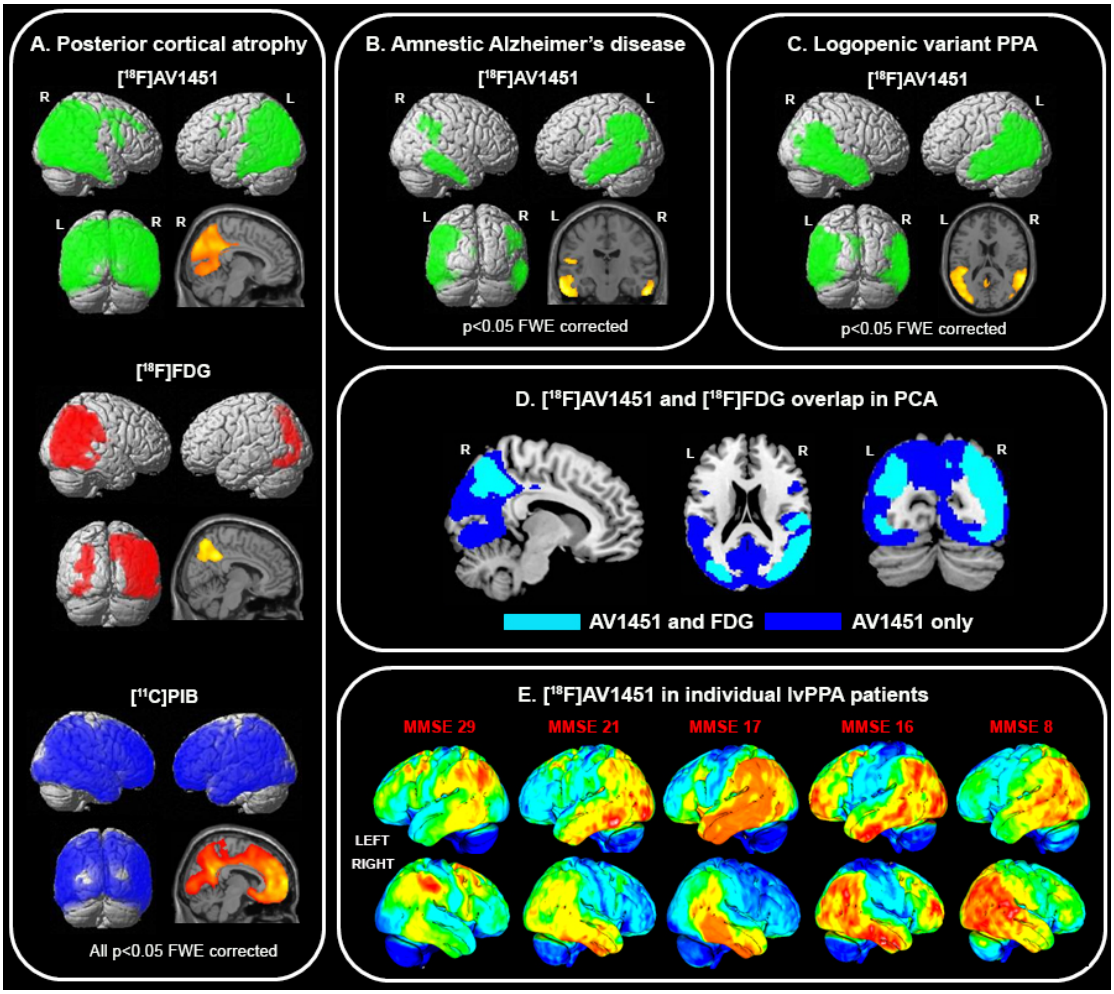

Figure 2. Panel A shows voxelwise differences between patients with posterior cortical atrophy ("the visual variant of AD") and healthy controls for AV1451 (tau), FDG (glucose hypometabolism) and PIB (amyloid). Panel B (amnestic Alzheimer's disease) and C (logopenic variant PPA) show differences in tau PET retention patterns. Panel D indicates that tau and hypometabolism (in cyan) overlap substantially, but there are also several brain regions with abnormal tau but relatively preserved glucose metabolis. Panel E shows tau PET patterns in lvPPA patients as a function of MMSE (a measure of global cognition).

From: Ossenkoppele et al. [2016] Brain.

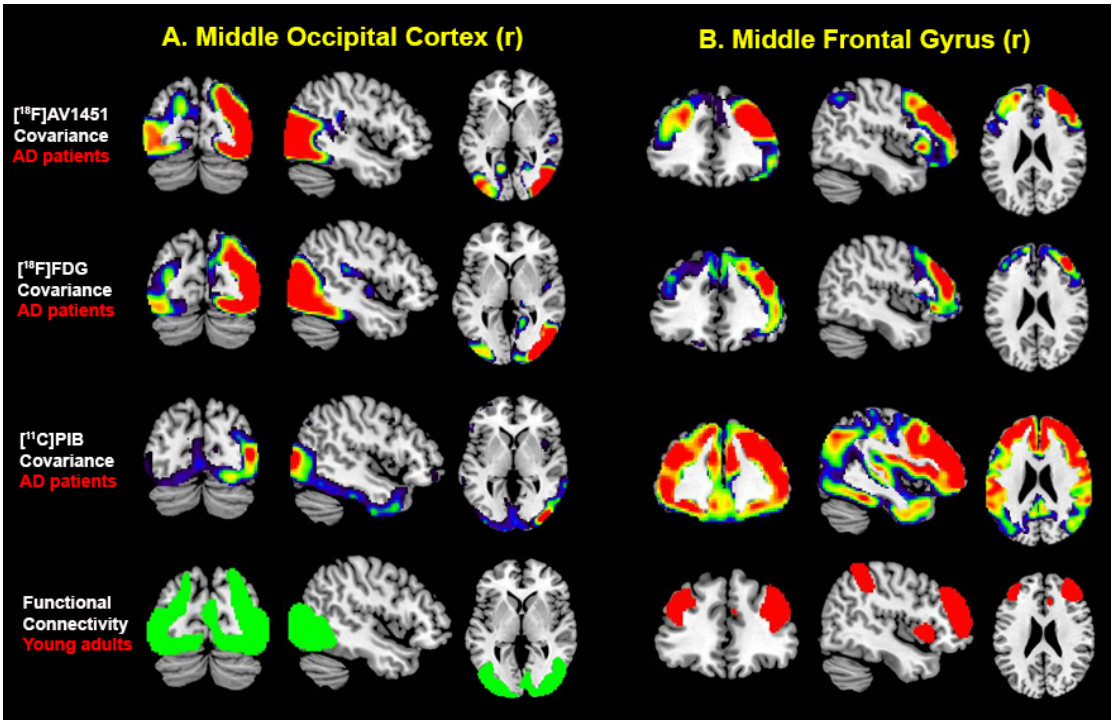

Figure 3. Using different seed regions (middle occipital cortex [A] and middle frontal gyrus [B]) we performed covariance analyses for AV1451, FDG and PIB PET in 36 AD patients and resting-state functional MRI analyses in 1,000 young healthy individuals. Results suggest that tau pathology follows the functional architecture of the healthy brain, while amyloid pathology is more widespread at the clinical stage of the disease.