Supplementary Analyses

AD dementia and prodromal AD versus preclinical AD

The control population may be grouped into those with biomarker signs of Aβ-pathology (i.e. preclinical AD, N=15) and those without biomarker signs of Aβ-pathology (N=15). We calculated AUROCs for ^{18}F-AV-1451 in tau stage I-IV and stage I-V, CSF T-tau and P-tau, hippocampal volume and temporal cortical thickness for preclinical AD versus AD dementia and (separately) versus prodromal AD. See supplementary Figure e-1.

^{18}F-AV-1451 had almost perfect separation for AD dementia versus preclinical AD. The AUROCs were significantly higher for ^{18}F-AV-1451 measures than for CSF T-tau (P=0.00080 for tau stage I-IV; P=0.00096 for tau stage I-V), P-tau (P=0.0032 for tau stage I-IV; P=0.0034 for tau stage I-V) and MRI measures (P=0.00088 for tau stage I-IV versus hippocampal volume; P=0.0096 for tau stage I-IV versus temporal lobe cortical thickness; P=0.0012 for tau stage I-V versus hippocampal volume; P=0.011 for tau stage I-V versus temporal lobe cortical thickness). There were no significant differences in AUROCs between CSF T-tau, P-tau and MRI measures (but a tendency for higher AURUC for P-tau compared to T-tau, P=0.060).

For prodromal AD versus preclinical AD, ^{18}F-AV-1451 in tau stage I-IV (P=0.013) and CSF P-tau (P=0.0088) had significantly higher AUROCs than hippocampal volume, and CSF P-tau had higher AUROC than CSF T-tau (P=0.018).