

Imaging tau pathology in vivo in FTLD: initial experience with [18F] T807 PET

Brad Dickerson¹, Kimiko Domoto-Reilly², Sapolsky Daisy², Michael Stepanovic², Michael Brickhouse², Keith Johnson³,

¹MGH/Harvard Medical School, Charlestown, Massachusetts, United States; ²Massachusetts General Hospital, Charlestown, Massachusetts, United States; ³Massachusetts General Hospital, Boston, Massachusetts, United States.

Background: A critical unmet need for FTLD research, especially therapeutic trials, is the development of biomarkers to distinguish FTLD-tau from FTLD-TDP and other non-tau FTLD pathologies. **Methods:** We are using [18F] T807, a novel PET ligand, to scan a series of patients with FTLD, to date including one MAPT P301L mutation carrier with moderate severity FTD dementia, an asymptomatic carrier of the same mutation, three patients with sporadic mild primary progressive aphasia, and three patients with progressive supranuclear palsy. We analyzed SUVR (cerebellum reference) data to localize and quantify [18F] T807 signal. We also co-registered analyzed [18F] T807 images to MRI images for visualization and calculation of % atrophy relative to controls. **Results:** [18F] T807 signal was elevated in frontal, insular, and anterior temporal cortex in the MAPT carrier with dementia, and colocalized with atrophy. In two non-fluent aphasic patients, [18F] T807 signal was highest in inferior frontal and middle temporal gyri and temporal pole with marked asymmetry, most prominent in the dominant hemisphere, and localized remarkably well with atrophy. The asymptomatic carrier had mildly elevated signal in frontal, insular, and anterior temporal cortex as well as white matter. PSP patients showed elevated brainstem, basal ganglia, thalamic/subthalamic, cerebellar, and frontal signal. **Conclusions:** T807 is a promising new PET ligand for imaging tau pathology in vivo in patients with FTLD.