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FTP tau PET imaging in symptomatic and asymptomatic *MAPT* mutation carriers

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State of the art
PET imaging with tau ligands (e.g., 18F-Flortaucipir, FTP) has established validity to detect paired helical filament tau in Alzheimer's Disease (AD), but this modality is much less useful in detecting the straight filament form of tau seen in Frontotemporal Dementia (FTLD-tau). However, it remains unclear whether such tau PET ligands can detect neuropathology early in patients who are biologically determined to develop FTLD-Tau.
Methodology
To investigate this, we scanned 7 individuals with known *MAPTP301L* mutations with FTP PET imaging. Of these 7, 4 were pre-symptomatic and 3 were symptomatic. These participants were recruited from the Massachusetts General Hospital FTD Family Study. We also compared these individuals to 11 age matched controls.
Results
Consistent with our hypotheses, we found that *MAPTP301L* mutation carriers demonstrated FTP uptake that co-localized to frontotemporal regions which were atrophied, and the magnitude of FTP signal uptake correlated with the magnitude of atrophy in these regions. Lastly, we found that pre-symptomatic *MAPTP301L* mutation carriers demonstrated lower FTP uptake than those with dementia but exhibited higher FTP binding than matched healthy controls.
Conclusion
These results demonstrate that although FTP PET has limited utility in the differential diagnosis of AD versus FTLD, it is a viable biomarker of neurodegeneration in individuals who are expected to develop FTLD-tau. This work will facilitate a better understanding of the pathobiology and evolution of neurodegeneration in FTLD-tau. It will also aid in the development of therapeutic agents by identifying *MAPTP301L* mutation carriers earlier in their disease course.

Conflicts of interest

No interests to disclose