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Neuroinflammation in frontotemporal lobar degeneration: A 11C-PBR28 positron emission tomography study

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State of the art: Neuroinflammation, a hallmark of frontotemporal dementia (FTD), has been sparsely studied *in vivo*. However, neuroinflammation is a potentially important therapeutic target and biomarkers of inflammation would be welcome. To determine the anatomical specificity of a putative biomarker for inflammation, translocator protein 18 kDa (TSPO) imaging, we used 11C-PBR28 PET to measure TSPO levels in patients with the non-fluent/agrammatic (nfvPPA) and semantic (svPPA) variants of primary progressive aphasia and the behavioral variant of FTD (bvFTD).

Methodology: Sixteen nfvPPA patients (10/16 women, age 67±6.3 years), 10 svPPA patients (5/10 women, age 65±7.6 years), 9 bvFTD patients (4/9 women, age 63±8.1 years) and 14 healthy controls (5/10 women, age 68±6.1 years) had 11C-PBR28 PET and MRI. Only two patients had amyloid on PET and neither had an AD-pattern 18F-flortaucipir PET. *VT* values for 11C-PBR28 were calculated with the Logan plot and a metabolite-corrected arterial input function. All images were corrected for partial volume effect. A full factorial analysis was performed on *VT* values between patients and controls at the voxel level.

Results: Compared to controls, increased *VT* values ($p < 0.005$) were found in svPPA (left temporal lobe, anterior right temporal lobe and left insula), nfvPPA (left middle, and inferior frontal gyri, bilateral medial superior frontal gyrus, left putamen and pallidum) and in bvFTD (both frontal lobes, particularly orbitofrontal cortex, anterior temporal lobes, insulae, putamen and pallidum).

Conclusion: The 11C-PBR28 uptake pattern corresponded to anatomical areas known to be involved in nfvPPA, svPPA and bvFTD.

Conflicts of interest

None