IN VITRO CHARACTERISATION OF [18F]-FLORBETABEN (BAY94-9172), AN AB IMAGING RADIOTRACER

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Introduction: Aβ plaques are the major pathological hallmark of Alzheimer’s disease (AD). As their formation precedes disease onset, their non-invasive detection is critical for early diagnosis and therapeutic intervention.

While [11C]-PiB is the most widely used Aβ-PET radiotracer, its usefulness is limited to centers possessing an on-site cyclotron due to the short half-life of [11C] (20mins). Therefore, [18F]-Aβ PET radiotracers with longer half lives (110mins), have been developed. We have recently demonstrated that [18F]-Florbetaben-PET can distinguish AD from frontotemporal lobe dementia (FTLD) or healthy volunteers. Whilst [18F]-Florbetaben-PET retention matched the reported post-mortem distribution of A plaques, the nature of [18F]-Florbetaben binding to other pathological misfolded proteins needs further specification.

Aim: To determine the selectivity of Florbetaben for Aβ plaques.

Methods: Florbetaben binding to human AD, FTLD and dementia with Lewy bodies (DLB) serial brain sections was analysed by autoradiography and histofluorescence microscopy.

Results: Autoradiography data in AD brain sections showed binding of [18F]- and [3H]-Florbetaben to Aβ plaques at PET concentrations, while no binding to tau pathology was observed. Histofluorescence staining of several AD brain regions demonstrated that Florbetaben identified Aβ plaques equally in all regions examined. Moreover, staining did not co-localize with a-synuclein-Lewy bodies or tau pathology in brain sections of DLB and FTLD subjects, respectively.

Conclusion: This study demonstrates that [18F]-Florbetaben-PET retention in AD patients is largely attributable to Aβ burden and not Lewy bodies and/or tau pathology.