Tau, amyloid, and hypometabolism in the logopenic variant of primary progressive aphasia

Figure ^{18}F -FDG, ^{18}F -florbetapir, and ^{18}F -AV-1451 PET superimposed to native MRI

Primary sensory-motor areas (asterisks), as well as the primary visual (striatal cortex) and auditory (Heschl gyrus) regions (arrowheads), have normal metabolism and no tau deposition. Areas with high tau deposition (e.g., inferior parietal lobule, arrows) have decreased metabolism. Avid Radiopharmaceuticals provided the precursor for ^{18}F -AV-1451.

A 57-year-old right-handed woman had an 8-year history of progressive word retrieval and repetition deficits, consistent with the logopenic variant of Alzheimer disease.¹ She was studied using the novel hyperphosphorylated-tau tracer ^{18}F -AV-1451,² in conjunction with metabolic (^{18}F -fluorodeoxyglucose [FDG]) and β -amyloid (^{18}F -florbetapir) PET (figure). Areas with high tau, such as the left inferior parietal lobule, had decreased

metabolism. By contrast, some areas with high amyloid, such as striated cortex, had normal metabolism. This case illustrates tau sparing of primary cortex and provides preliminary in vivo evidence that regional tau is more closely linked to hypometabolism than amyloid density.

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