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AMYLOID DEPOSITION IN BRAIN AGING: CAUSAL AGENT OR INNOCUOUS BY STANDER ?

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PET amyloid imaging has been initially considered as the main tool to investigate the beginning of the AD process in cognitively intact individuals. The percentage of PET-amyloid positive controls is of 6% at age 60 but reaches 50% at age 90 in community-based sample pointing to the fact that amyloid deposition (as amyloid plaque formation) is closely related to aging process. In fact, increased PiB (Pittsburgh Compound B) binding has been reported in almost 20%-30% of cognitively preserved elders mainly in posterior cingulate cortex, precuneus and prefrontal cortex. Compared with amyloid-negative, amyloid-positive controls showed moderate decline in verbal and visual episodic memory over 36 months but no changes were seen in non-memory functions. Most importantly, the absence of amyloid in mild cognitive impairment (MCI) cases is associated with cognitive stability at 36 months. Increased PET-PiB binding is associated with brain atrophy, cortical thinning but also decreased cortical metabolism, aberrant functional connectivity at rest and decreased task-related deactivation of the default mode network. Altogether these data suggest that contrasting with CSF Aß and tau changes that sign a biological diathesis to neuro-degeneration, amyloid positivity in the human brain is present as a part of the aging process representing a critical step preceding the installation of AD pathophysiology. However, not all cases with elevated PET-PiB bindings evolve to AD and several cases develop dementia not necessarily related to amyloid aggregation. Several recent contributions revealed that neurodegeneration takes place without a temporal link with fibrillar amyloid deposits. Alternative but less frequent pathways exist starting from tau deposition with modest Aß pathology.

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