EPAD
European Prevention of
Alzheimer's Dementia Consortium

# Brain Amyloid Pathology and Cognitive Function Alzheimer Disease Without Dementia?

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**If there ever was an exciting time for Alzheimer disease (AD) research, it is now. The discovery of biomarkers beginning 20 years ago, for example, positron emission tomography (PET) tracers that bind to plaques, the core pathological hallmark of the disease, unlocked new research fields. Together with increasing amounts of longitudinal data, it now is possible to study how the disease unfolds. This will transform the way AD is conceptualized, diagnosed, and treated.**

**AD is characterized by aggregated β-amyloid into plaques in the brain. This amyloid pathology can be measured by PET tracers or indirectly by a reduction of the β-amyloid1-42 peptide in cerebrospinal fluid (CSF). Previous research has shown that abnormal amyloid biomarkers are present in up to 50% of cognitively normal older persons.3 The prevalence of amyloid pathology increases with age, and this parallels increases in Alzheimer-type dementia approximately 20 years later. The time lag between amyloid pathology and dementia prevalence suggests a long preclinical stage of the disease, during which pathological events accrue until brain damage is so extensive that cognitive impairment emerges.**

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