



Evaluate Novel Biomarkers of Alzheimer's Disease Pathology Using Advanced Neuroimaging Methods

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DESCRIPTION

Alzheimer's Disease (AD) is a progressive neurodegenerative disorder that affects millions of people worldwide and causes cognitive impairment, dementia, and death. AD is characterized by the accumulation of Amyloid-Beta ($A\beta$) plaques and Neurofibrillary Tangles (NFTs) of hyperphosphorylated tau protein in the brain, as well as synaptic dysfunction, neuroinflammation, and neuronal loss. The diagnosis of AD is currently based on clinical criteria and neuropsychological tests, but these methods are not sensitive or specific enough to detect the disease at its early stages. Therefore, there is a need for novel biomarkers that can reflect the underlying pathological processes of AD and enable early diagnosis, prognosis, and treatment monitoring. Neuroimaging techniques, such as Magnetic Resonance Imaging (MRI), Positron Emission Tomography (PET), and spectroscopy, can provide non-invasive and quantitative measures of brain structure, function, metabolism, and molecular pathology *in vivo*.

Neuroimaging methods for alzheimer's treatment

Amyloid imaging: $A\beta$ plaques are one of the hallmarks of AD and are thought to play a causal role in the disease pathogenesis. Amyloid imaging using PET tracers that bind to $A\beta$ deposits can detect and quantify the extent and distribution of amyloid pathology in the brain. Several amyloid PET tracers have been developed and approved for clinical use, such as Pittsburgh compound B (PiB), florbetapir, florbetaben, and flutemetamol.

Amyloid imaging can distinguish AD patients from healthy controls and other forms of dementia with high accuracy and can also identify individuals with Mild Cognitive Impairment (MCI) who are at risk of developing AD. However, amyloid imaging has some limitations, such as high cost, limited availability, radiation exposure, and variability in tracer binding and interpretation. Moreover, amyloid imaging does not correlate well with cognitive

decline or disease progression, as some individuals may have high amyloid load but no symptoms (preclinical AD) or vice versa (amyloid-negative AD). Therefore, amyloid imaging alone is not sufficient to diagnose or monitor AD and needs to be combined with other biomarkers that reflect other aspects of the disease.

Tau imaging: NFTs of tau protein are another hallmark of AD and are closely associated with neuronal degeneration and cognitive impairment. Tau imaging using PET tracers that bind to tau aggregates can detect and quantify the extent and distribution of tau pathology in the brain. Several tau PET tracers have been developed and are under investigation, such as THK5117, THK5351, AV1451, MK6240, PI2620, and RO948. Tau imaging can differentiate AD patients from healthy controls and other forms of dementia with high accuracy and can also identify individuals with MCI who are likely to progress to AD.

Moreover, tau imaging correlates better with cognitive decline and disease progression than amyloid imaging, as tau pathology spreads along a predictable pattern that matches the Braak staging of AD. However, tau imaging also has some limitations, such as high cost, limited availability, radiation exposure, and variability in tracer binding and interpretation.

Synaptic imaging: Synaptic dysfunction and loss are early events in AD that precede neuronal death and correlate with cognitive impairment. Synaptic imaging using PET tracers that bind to synaptic vesicle proteins or receptors can detect and quantify the extent and distribution of synaptic density in the brain. Several synaptic PET tracers have been developed and are under investigation, such as (11C) UCB-J that binds to synaptic vesicle glycoprotein 2A (SV2A), (11C) SA4503 that binds to sigma-1 receptors, and (11C) ABP688 that binds to Metabotropic Glutamate Receptor 5 (mGluR5). Synaptic imaging can differentiate AD patients from healthy controls and other forms of dementia with high accuracy and can also identify individuals with MCI who are at risk of developing AD.

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Multimodal imaging: It refers to the integration of different neuroimaging techniques and modalities to provide a comprehensive and holistic view of the brain structure, function, metabolism, and molecular pathology *in vivo*.

Amyloid-tau-synaptic imaging: This approach combines amyloid PET, tau PET, and synaptic PET to measure the three main pathological features of AD in the same individual. This approach can improve the accuracy of AD diagnosis and staging and reveal the interactions and associations among the different pathologies.

Amyloid-MRI: This approach combines amyloid PET and structural or functional MRI to measure both the molecular and the macroscopic changes in the brain. This approach can improve the accuracy of AD diagnosis and prognosis and reveal the impact of amyloid pathology on brain atrophy, connectivity, perfusion, and metabolism.

Tau-MRI: This approach combines tau PET and structural or functional MRI to measure both the molecular and the macroscopic changes in the brain. This approach can improve the accuracy of AD diagnosis and prognosis and reveal the impact of tau pathology on brain atrophy, connectivity, perfusion, and metabolism.

Synaptic-MRI: This approach combines synaptic PET and structural or functional MRI to measure both the molecular and the macroscopic changes in the brain. This approach can improve the accuracy of AD diagnosis and prognosis and reveal the impact of synaptic pathology on brain atrophy, connectivity, perfusion, and metabolism.

CONCLUSION

Neuroimaging techniques offer a powerful tool for developing novel biomarkers for AD that can reflect the underlying pathological processes of the disease. However, no single neuroimaging technique or modality can capture the complexity and heterogeneity of AD. Therefore, multimodal neuroimaging approaches that combine different techniques and modalities are needed to provide a comprehensive and holistic view of the brain structure, function, metabolism, and molecular pathology *in vivo*. Multimodal neuroimaging biomarkers can improve the accuracy of AD diagnosis and monitoring and reveal the interactions and associations among the different pathologies. They can also provide insights into the mechanisms and progression of AD and facilitate the development and evaluation of new therapeutic interventions.