

WILL THERE EVER BE A VALID BIOMARKER FOR AD?

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The recent focus on soluble forms of toxicity that may be active long before the formation of brain aggregates implies, however, that A β - or tau-focused curative strategies would be effective only in very early and possibly preclinical stages of Alzheimer's disease (AD) when biological compromise is still avoidable. In last years, AD research provided two main categories of biomarkers. The first concerned markers with good diagnostic specificity that make it possible to differentiate individuals with preclinical and probable AD from individuals with other forms of dementia. The second group's structural and functional neuroimaging as well as biochemical markers that change with disease progression and may even predict the evolution from mild cognitive impairment (MCI) to clinically overt AD. Very recent studies in routine clinical settings suggested that cerebrospinal fluid (CSF) markers (such as phospho tau/A β 42 ratio), hippocampal (or entorhinal cortex) volumes and in some cases amyloid imaging data at baseline might predict accurately the conversion from MCI to clinically overt AD. Perhaps the most important biomarkers are those that could allow for detecting the individual signature of biological vulnerability for AD in healthy elders. These individuals who are cognitively normal, and yet have evidence of AD pathology (i.e. preclinical AD) are the most likely to take profit from future disease modifying/prevention therapies. The thoughtful use of such biomarkers at an individual level will be a major but ethically problematic challenge of AD research in the near future.