Why have we failed to cure AD?

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The most popular target in AD clinical trials has been beta-amyloid. However, while this protein has a central role in early onset AD, particularly in those patients carrying mutations in APP, PSEN1 or PSEN2 genes, there is no convincing evidence that it is involved in the pathogenesis of late-onset AD. Additionally elimination of beta amyloid has not led to clinical improvement or to arrest disease progression. Therefore, attempts should be directed at other targets, including ADOE, inflammation and cardiovascular risk factors.