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The antibody aducanumab reduces A β plaques in Alzheimer's disease

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summary

Alzheimer's disease (AD) is characterized by deposition of amyloid- β (A β) plaques and neurofibrillary tangles in the brain, accompanied by synaptic dysfunction and neurodegeneration. Here authors report the generation of aducanumab, a human monoclonal antibody that selectively targets aggregated A β . In a transgenic mouse model of AD, aducanumab is shown to enter the brain, bind parenchymal A β , and reduce soluble and insoluble A β in a dose-dependent manner. However, the main safety and tolerability findings are amyloid-related imaging abnormalities. These results justify further development of aducanumab for the treatment of AD. If the slowing of clinical decline should be confirmed in ongoing phase 3 clinical trials, it would provide compelling support for the amyloid hypothesis.