

A tangled path: the frustrating search for AD treatment

Just forty years ago, AD was regarded as an inevitable consequence of aging, and hence an untreatable condition. The former director of the Office of Alzheimer Research at NIH recalls that as recently as the early 1990s, concepts like “cure” and “prevention” for AD were “inconceivable.”¹¹

It took decades to finally establish that AD is a disease due to degenerating neurons, and not just part of the aging process. 10 **Beta amyloid** is a protein that occurs naturally in healthy brains. Beta-amyloid is also chemically “sticky” and gradually builds up into plaques.¹²

One of the things we learned about AD is that it is caused by these beta-amyloid plaques and tangles. This led to the “beta-amyloid hypothesis:” To stop AD you need to stop the beta-amyloid clumping.¹³

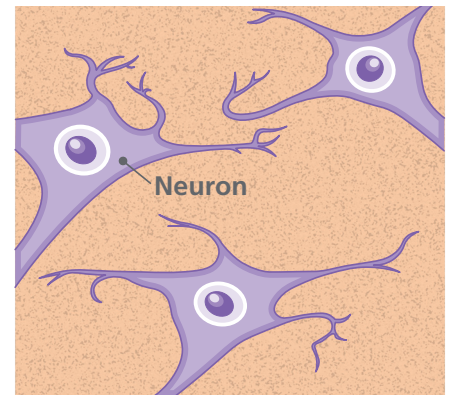
Aducanumab is an engineered antibody (“monoclonal antibody”) that drug developers say can help fight AD by reducing amyloid beta tangles.¹³

The only problem is that despite massive investments of time and money, pharmaceutical firms have been unable to successfully develop disease-modifying treatments for AD using the amyloid hypothesis.¹⁴ So far, at least, this includes aducanumab.

For citations, please see “References” in the main article.

Human brain cells

Normal



Alzheimer's

