

Alzheimer's Disease

Alzheimer's disease is a progressive neurologic disease of the brain that causes damage to neurons - the specialized cells of the nervous system that enable the flow of information, thoughts and memories in the brain. When Alzheimer's disease damages neurons it leads to loss of memory and reasoning, which can affect a person's ability to interact socially or function at work.

Alzheimer's is perhaps the most feared disease in the world because it destroys what makes a person individual. No treatment yet exists to halt the progression of Alzheimer's-related dementia, delay its onset, or better yet prevent it from occurring. Currently available drugs treat the symptoms, but only temporarily.¹ An Alzheimer's treatment that can effectively halt or delay the progression of disease is urgently needed.

Alzheimer's burden

Alzheimer's disease is the most common cause of dementia worldwide.¹ The World Health Organization estimated that 47.5 million people worldwide were living with dementia in 2016, with 7.7 million new cases every year.² Alzheimer's disease may contribute to up to 70% of these cases.²

In the United States, more than 5 million people currently have Alzheimer's disease, and a new case develops every 66 seconds.¹ The annual number of new cases of Alzheimer's and other dementias is projected to double by 2050, representing a critical burden for the world's healthcare systems.¹ If people who have not yet received a formal diagnosis of Alzheimer's are included (e.g., those with mild cognitive impairment who later develop Alzheimer's), then the total number of people affected would be even higher than currently estimated.³

Alzheimer's disease is one of the costliest chronic diseases to society.¹ In 2016, total payments for individuals with Alzheimer's disease and other dementias in America were estimated at \$236 billion.¹ This is expected to increase to over \$1 trillion by 2050.¹

The role of tau tangles

Several theories exist to explain the cause of Alzheimer's disease, and three features of Alzheimer's have attracted the most attention: disturbed cholinergic function, the amyloid cascade, and tau pathologies.

Disruptions in the cholinergic and glutamatergic neurotransmitters formed the basis for the development of current approved treatments for Alzheimer's disease. These treatments, however, are essentially symptomatic and provide only modest and temporary improvements in cognition and global functioning.

The discovery of mutations in the amyloid precursor protein (*APP*) gene led to the amyloid cascade hypothesis.⁴ In this hypothesis, mutations in *APP* have been considered to cause a build-up of extracellular amyloid- β in the brain.⁵ However, while detection of this build-up may be useful for diagnostic purposes, anti-amyloid therapeutic agents have shown disappointing results in clinical trials.^{6,7}

Attention has more recently been focused on the hypothesis that formation of "tau tangles" (more formally described as neurofibrillary tangles formed of paired helical filaments of tau protein sub-units that form inside neurons) leads to neurodegeneration.^{8,9} This pathology begins to develop up to 20 years before clinical symptoms appear.¹⁰ Unlike other hypotheses, the tau mechanism shows a correlation between clinical symptoms and tau pathology. The reduction in the presence of "tau tangles" in the brain is thus a potential focus for therapeutic product development in Alzheimer's disease.¹⁰

TauRx Co-founder and Chairman, Professor Claude Wischik, first found that the tangles linked with Alzheimer's are made of sub-units of tau protein more than 30 years ago. Professor Wischik and his colleagues estimate that about 50% of the U.S. population over age 45 have some form of tau pathology in their brains.¹⁰ This forecast reinforces the importance of developing new treatments to halt and prevent tau tangles from forming and spreading throughout the brain.

References

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