

Failed Alzheimer's test shows in which direction the research should continue

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Disappointing results in clinical Alzheimer's studies discourage doctors and scientists from continuing their research into γ -secretases and a possible treatment against Alzheimer's disease. In the prestigious journal *Cell*, the Alzheimer's expert Bart De Strooper (VIB-KU Leuven) argues that these studies are not pointless, but merely indicate what the next steps should be for the Alzheimer's research. This article - or rather, this message - will be spread at the largest Neuroscience meeting that will take place mid-November in Washington.

Bart De Strooper: "We need to learn from AIDS, cholesterol or cancer research: we can only find a drug to treat Alzheimer's disease if we are willing to learn from the difficulties that we encounter. The current pessimistic mood does not answer the patients' needs."

The γ -secretase Complex

Alzheimer's disease is characterized by abnormal accumulations of a protein (amyloid β ; A β) between the brain cells; the so-called amyloid plaques. Several years ago Bart De Strooper and his team unraveled the structure and the function of the γ -secretase complex, which plays a significant role in the development of these plaques.

Disappointing Results in Clinical Alzheimer's Studies

For many years the γ -secretases were the target for the development of a

drug to treat Alzheimer's disease. It was thought that blocking these γ -secretases would combat the production of the plaques and thereby prevent nerve cells from dying. "Semagacestat" (Eli Lilly) is one of the many examples of candidate drugs that were tested in a clinical setting. The failure of the phase 3 clinical tests (these tests are performed on patients) involving semagacestat resulted in the sudden termination of γ -secretase research. Unfortunately, this effect was not only noticeable in the pharmaceutical industry; the academic world also reacted with disappointment.

Lessons for Future Alzheimer's Research

In the journal *Cell*, Bart De Strooper implores scientists to apply what they have learnt from these studies to future research. Many arguments indicate that $A\beta$ -peptides do play a role in Alzheimer's disease, so it remains very important to test the hypothesis of the amyloid plaques extensively. Furthermore, thorough fundamental research remains essential, considering the fact that the current knowledge gap between the biology, physiology and structural biology of γ -secretases is still very large. New insights and an improved understanding of γ -secretases must form the foundation for a more rational development of a drug.

What is Alzheimer's Disease?

Alzheimer's disease is the most common form of dementia in the developed world. The disease is primarily characterized by a loss of cognitive functions. A loss that can be attributed to the death of [nerve cells](#) in the brain.

Approximately 100,000 people in Flanders currently have dementia. In 2020 this number will increase to 130,000 and in 2050 it will be 200,000. Despite intense efforts and years of research there is currently

no curative treatment. Even a treatment that significantly slows down the condition is not immediately available.

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