

Better understanding of the cause of Alzheimer's disease: New suggestion for a possible treatment

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Alzheimer's disease is the most common form of dementia, affecting over 35 million people worldwide. It is generally assumed that the clumping of beta-amyloid (A β) protein causes neuronal loss in patients. Medication focuses on reducing A β 42, one of the most common proteins and the most harmful. University of Twente PhD student Annelies Vandersteen is refining the current approach. She explains: "The results of my research provide a broader understanding of the processes that lead to Alzheimer's disease and in this way may help to bring about new medication".

The A β protein occurs in the body in various lengths, ranging from 33 to 49 [amino acids](#). The shorter varieties are regarded as 'safe', unlike the longer ones – A β 42 and longer – which are highly aggregating. Current [therapeutic strategy](#) tries to reduce the clumping of A β 42, and its harmful effects, by limiting the release of A β 42. Reducing A β 42 production at the same time results in a rise in A β 38 levels. Vandersteen comments: "One of the findings of my research is that small amounts of A β 38 can in fact increase or temper the clumping and [toxic effects](#) of longer A β proteins. The processes that result in Alzheimer's disease are determined by the whole spectrum of A β proteins. So the picture is far less black and white than has been assumed so far, and less common forms of A β are far less harmless than we thought."

Vandersteen examined the protein mixtures in a laboratory situation. She

devised a series of experiments based on a computer-calculated hypothesis. The behaviour of the various A β proteins and mixtures was studied in detail and described using various biophysical techniques. The influence of the various A β proteins and mixtures on neurons was then studied in a cell culture.

Annelies Vandersteen's PhD research was carried out as part of a triple degree at the University of Twente, the Catholic University of Leuven and the Vrije Universiteit Brussel. The study falls within the work of the MESA+ and MIRA research institutes of the University of Twente, Faculty of Science and Technology, Nanobiophysics Group. The thesis 'Aggregation and toxicity of amyloid-beta peptide in relation to peptide sequence variation' is available on request.

Provided by University of Twente

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