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An investigation into amyloid beta peptide variation as Alzheimer's disease progress

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One of the most prominent hallmarks detected in the brains of patients suffering from Alzheimer's disease is the deposition of amyloidogenic plaques. These plaques are largely composed of the amyloid-beta peptide. It has been demonstrated that, even though these plaques comprise an important and recurring feature for disease, that precursor forms of these plaques, called 'oligomers' or 'protofibrils' more potently affect neuronal functioning. The amyloid-beta peptide arises from cleavage of trans-membrane amyloid precursor protein through cleavage by means of a combination of secretase enzymes. As a result of this enzymatic processing, genetic profile and further modifications, the amyloid beta peptide does not exist as a well-defined species but arises in a variety of truncated and modified forms. For example, it has been reported that, within any one individual, a range of amyloid beta peptides exist varying in length from 34 to 49 amino acids and that multiple otherwise modified forms of this peptide are present in a complex mixture. We have shown that small shifts in the composition of the amyloid beta pool can have significant impact on the aggregation reaction, cellular response and cognitive behavior in animal models. The mechanism by which hence formed variants of the amyloid-beta peptide cause disease still remains elusive. In aim to determine how amyloid beta peptide manifests its pathological effects, we use Fourier transform infrared spectroscopy (FTIR), transmission electron microscopy, mass spectrometry and immunohistochemistry to monitor peptide aggregation.

Biography

Kerensa Broersen completed her Doctorate in the field of protein aggregation at Wageningen University in The Netherlands in 2005. After her Post-doctoral study at the MRC-LMB in the UK, she joined the Free University of Brussels (VUB)/Flanders Institute for Biotechnology (VIB) in 2007. Here, she headed a research team that studied the molecular mechanism of Alzheimer's disease. This led to the discovery of molecular pathways of a number of risk factors that affect Alzheimer's disease pathobiology. Subsequently, she joined the Nanobiophysics Group at the University of Twente/MIRA Institute in The Netherlands as an Assistant Professor investigating further the impact of protein structures on human health with her team.

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