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Amyloid beta in the brain of individuals with Alzheimer's disease

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While there may not be a consensus whether deposition of amyloid beta contributes to Alzheimer's disease or is a consequence of it, there is agreement that something else is promoting the process. Other proteins are often co-deposited with amyloid beta including serum amyloid P component. Recent evidence has suggested that SAP is elevated in Alzheimer's disease. Researchers from Keele University have shown that physiologically-significant concentrations of SAP promote the deposition of amyloid beta under conditions approaching those found in vivo.

The deposition of amyloid beta in the brain of individuals with Alzheimer's disease is the focus of much research into both its cause and treatment.

While there may not be a consensus as to whether the deposition contributes to the disease or is a consequence of the disease, there is agreement that it is not favoured thermodynamically, meaning that something else is promoting the process.

Other proteins are often co-deposited in vivo with amyloid beta and one such protein is serum amyloid P component (or SAP). Recent evidence has suggested that SAP is elevated in Alzheimer's disease and a team of researchers from Keele University in Staffordshire, UK, led by Professor Chris Exley, has shown that physiologically-significant concentrations of SAP promote the deposition of amyloid beta under conditions approaching those found in vivo.

Professor Exley said: "We have shown that SAP is bound by fibrils of amyloid beta and that this interaction stabilises the fibrils over timescales which are physiologically significant. This is the first example of a physiologically significant biomolecule promoting and stabilising the formation of amyloid fibrils of amyloid beta 42 under near-physiological conditions."

The group also found that this property of SAP was enhanced in the presence of aluminium, a metal which has also been shown to be co-deposited with amyloid beta in Alzheimer's disease. There have been recent efforts to reduce the plasma concentration of SAP as a therapy for Alzheimer's disease and the research provides strong evidence that SAP is involved in the deposition of amyloid beta 42 in Alzheimer's disease and that by reducing the plasma concentration of SAP it might also reduce the deposition of amyloid beta. Their observations support serum amyloid P component as a therapeutic target in Alzheimer's disease.

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