# Reciprocal Molecular Interactions between the A$\beta$ Peptide Linked to Alzheimer's Disease and Insulin Linked to Diabetes Mellitus Type II



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Clinical studies indicate diabetes mellitus type II (DM) doubles the risk that a patient will also develop Alzheimer's disease (AD). DM is caused by insulin resistance and a relative lack of active insulin. AD is characterized by the deposition of amyloid $\beta$ (A$\beta$) peptide fibrils. Prior to fibrillating, A$\beta$ forms intermediate, prefibrillar oligomers, which are more cytotoxic than the mature A$\beta$ fibrils. Insulin can also form amyloid fibrils. In vivo studies have revealed that insulin promotes the production of A$\beta$, and that soluble A$\beta$ competes with insulin for the insulin receptor. Here, we report that monomeric insulin interacted with soluble A$\beta$ and that both molecules reciprocally slowed down the aggregation kinetics of the other. Prefibrillar oligomers of A$\beta$ that eventually formed in the presence of insulin were less cytotoxic than A$\beta$ oligomers formed in the absence of insulin. Mature A$\beta$ fibrils induced fibrillation of soluble insulin, but insulin aggregates did not promote A$\beta$ fibrillation. Our study indicates that direct molecular interactions between insulin and A$\beta$ may contribute to the strong link between DM and AD.
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