Study probes interplay of proteins in type 2 diabetes

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A hallmark of age-related diseases such as Parkinson's disease, type 2 diabetes, or Alzheimer's disease is the abnormal clumping of proteins in cells. In people with these conditions, these protein clumps can result in irregular deposits known as amyloids that disrupt normal cell behaviors. A Yale pathologist recently discovered that these interactions can be dramatically reduced in type 2 diabetes when small amounts of neighboring proteins are present.

To understand these interactions at the molecular level, assistant professor and lead author Zachary Levine and his collaborators ran a series of simulations that revealed how amyloids form. They found that a unique protein neighbor, normally encountered elsewhere in the cell, was able to stabilize the amyloids found in type 2 diabetes with very high precision. These interactions were then verified in further experiments, suggesting that the body might regulate amyloid diseases using a cocktail of stabilizing proteins.

This discovery highlights important interactions between amyloid proteins and other small molecules that scientists can mimic using synthetic compounds, said Levine. These observations are not believed to be unique to diabetes, he noted, suggesting that multiple amyloid diseases can be targeted, and potentially treated, in a similar way. The study is published and featured on the cover of the *Journal of the American Chemical Society*

More information: Zachary A. Levine et al. The Mitochondrial Peptide Humanin Targets but Does Not Denature Amyloid Oligomers in Type II Diabetes, *Journal of the American Chemical Society* (2019). DOI: 10.1021/jacs.9b04995

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