The aggregation of the 37-amino acid polypeptide human Islet Amyloid Polypeptide (hIAPP, amylin), as either insoluble amyloid or as small oligomers, appears to play a direct role in the death of pancreatic β-islet cells in type 2 diabetes. While hIAPP is the primary component of type 2 diabetes amyloid, the molecular interactions responsible for this aggregation are not well understood. While it remains unclear how self-assembly of hIAPP leads to the development of disease, recent studies have suggested that the formation of lower order protein aggregates (two to ten self-assembled proteins) leads to cellular toxicity and ultimately to the progression of disease. Preventing the formation of these toxic species may slow, if not prevent entirely, the progression of type 2 diabetes. Our recent results analyzing the aggregation propensity of naturally occurring IAPP variants will be discussed as well as the identification of a series of peptides capable of inhibiting hIAPP aggregation.