In type 2 diabetes, progressive loss and dysfunction of insulin-producing beta cells occurs, in part due to formation of islet amyloid and inflammation within the pancreatic islet. Islet amyloid forms by aggregation of islet amyloid polypeptide (IAPP), a peptide co-secreted with insulin by beta cells. We have recently found that IAPP aggregates are potent inducers of islet inflammation, by activating islet macrophages and inducing expression of pro-inflammatory cytokines. Recent work has provided new insight into the mechanism by which IAPP aggregates lead to macrophage activation and beta cell dysfunction. IAPP-induced inflammation represents a novel therapeutic target in type 2 diabetes.