Programing amylin secretion to slow AD

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Mutations in Aβ and/or in proteins participating in the processing mechanisms were linked to the development of familial AD. Here, we showed that, in addition to Aβ pathology, brains of patients suffering with familial AD have large deposits of amylin, an amyloidogenic hormone co-secreted with insulin. Amylin forms neuritic deposits, co-localizes with Aβ as mixed Aβ-amylin plaques and also accumulates intracellularly in neurons. Ameliorating amylin dyshomeostasis in the periphery reduced AD in a preclinical model.