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Article

Regulation of Amyloid Fibril Formation from Human Islet Amyloid Polypeptide by a Ligand Binding to the Fusion of FK506Ãƒâ¢Ã‚€Ã‚•binding Protein and the InsertionÃƒâ¢Ã‚€Ã‚•inÃƒâ¢Ã‚€Ã‚•Flap Domain

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Abstract

Human islet amyloid polypeptide (hIAPP) is converted to toxic aggregates in typeÃƒÂ¢Ã‚Â€Ã‚Â•2 diabetes (T2D). With the aim of developing a candidate agent regulating hIAPP amyloid fibril formation, we constructed a series of recombinant fusion proteins using a bacterial expression system. FKBPIFÃƒÂ¢Ã‚Â€Ã‚Â•L23ÃƒÂ¢Ã‚Â€Ã‚Â•hIAPP and F36VIFÃƒÂ¢Ã‚Â€Ã‚Â•L23ÃƒÂ¢Ã‚Â€Ã‚Â•hIAPP accelerated amyloid fibril formation. Assays of *cis*/*trans*ÃƒÂ¢Ã‚Â€Ã‚Â•peptidylÃƒÂ¢Ã‚Â€Ã‚Â•prolyl isomerase activity as well as chaperone activity of FKBPIFÃƒÂ¢Ã‚Â€Ã‚Â•L23ÃƒÂ¢Ã‚Â€Ã‚Â•hIAPP fusion proteins suggested that amyloid fibrils were produced because the chaperone activity of FKBPIF in the form of fusion protein was diminished. Moreover, addition of FK506, a binding ligand, slowed down the process of amyloid fibril formation in FKBPIFÃƒÂ¢Ã‚Â€Ã‚Â•L23ÃƒÂ¢Ã‚Â€Ã‚Â•hIAPP fusion protein. Deletion mutant analysis of the insertionÃƒÂ¢Ã‚Â€Ã‚Â•inÃƒÂ¢Ã‚Â€Ã‚Â•flap (IF) domain revealed that the CÃƒÂ¢Ã‚Â€Ã‚Â•terminal half is essential for acceleration of amyloid fibril formation. Based on these observations, we suggested a potential antiÃƒÂ¢Ã‚Â€Ã‚Â•T2D therapeutic strategy by means of a small ligand and fragments of the IF domain inserted in a fusion protein.

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