Cyclopeptides for treating amyloid diseases and their comorbidities

Aggregation and cytotoxicity of Aß, α-synuclein and IAPP (or amylin) are linked to the pathogenesis of Alzheimer's (AD), Parkinson's disease (PD) and type 2 diabetes (T2D). These diseases are major health problems worldwide and have interconnected molecular mechanisms, partly through so-called "cross-seeding" of their amyloid proteins; this may link their pathologies and result in comorbidities and further complications in their treatment.

IP RIGHTS
EP Priority Application
2025

Multi-site & multi-target inhibition plus aggregate remodeling

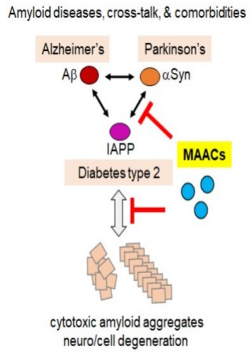
Here, we present MAACs (multifunctional anti-amyloid cyclopeptides), with lead compound 2i, representing the first-in-class small cyclic peptide with potent and multifunctional inhibition of amyloid self-assembly and cross-seeding between AD, T2D, and PD proteins, along with drug-like and diagnostic properties.

01 Strong binding to Aβ42 (AD), IAPP (T2D) and αSyn (PD)

02 Act at nanomolar to low micromolar concentrations, small (~700 Da) and cheap to synthesize

03 Effective BBB crossing in cell models and highly resistant to proteolytic degradation

04 Highly soluble, non-amyloidogenic, and non-toxic up to concentrations far above those required for function

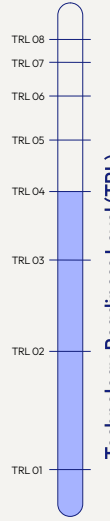


CHALLENGE

Amyloid diseases involve complex, disordered proteins that are difficult to target. Current inhibitors (antibodies, small molecules, peptides) have drawbacks such as low stability, low specificity, poor blood-brain barrier (BBB) permeability, and high production costs. Only a few controversial treatments are available, and no early diagnostic tests exist for amyloid formation.

INNOVATION

A new class of drugs called Multifunctional Anti-Amyloid Cyclopeptides (MAACs), focusing on short, cyclized hexapeptides were developed. These peptides are designed to mimic key AD, T2D, and PD amyloid protein contact points, ensuring high affinity and specificity for these amyloid proteins. The lead MAAC (termed 2i) can inhibit the self-assembly (aggregation) of each of these proteins into toxic forms and to block cross-seeding between these proteins, which otherwise may accelerate disease progression and comorbidity . Moreover, MAACs are able to remodel or disassemble toxic amyloid oligomers and fibrils into non-toxic forms.



01 Basic principles observed · **02** Technology concept formulated · **03** Experimental proof of concept · **04** Technology validated in lab · **05** Technology validated in relevant environment · **06** Technology demonstrated in relevant environment · **07** System prototype demonstrated in operational environment · **08** System complete and qualified

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