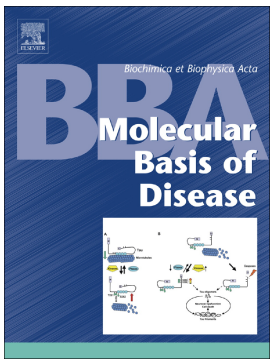

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Identification of prion protein-derived peptides of potential use in Alzheimer's disease therapy

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ABSTRACT

Soluble form of the prion protein (PrP) has been previously shown to interact with amyloid-β (Aβ) peptides, suppressing their fibrillization as well as toxicity, which indicates that this protein may play a protective role in Alzheimer's disease (AD). The shortest known PrP fragment retaining all of these properties corresponds to physiologically generated proteolytic polypeptide PrP23-110/111, called N1. Here we have identified two N1-derived synthetic peptides, encompassing residues 23-50 (PrP23-50) and 90-112 (PrP90-112), which bind to Aβ1-42 protofibrillar oligomers as well as amyloid fibrils. We found that, akin to N1, the abovementioned synthetic peptides not only reduce the initial rate of Aβ fibrillization, but also