

Abstract

S100A8 Interaction with Amyloid- β Peptide Suppresses Its Fibrillation [†]

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1. Introduction

S100A8 protein belongs to the EF-hand family of calcium-binding proteins and is involved in inflammatory processes, immune response, and the pathogenesis of neurodegenerative diseases, including Alzheimer's disease (AD). According to available clinical data, the level of S100A8 is increased in the cerebrospinal fluid of AD patients. Although data in the literature suggest an involvement of S100A8 in the regulation of amyloid- β peptide (A β) metabolism involved in AD progression, the possible interaction between S100A8 and A β remains unexplored. To this end, we aimed to study this aspect and its relevance to A β fibrillation.

2. Methods

The parameters of interaction between recombinant human S100A8 and monomeric recombinant human A β 40/42 were studied using bio-layer interferometry. The A β 40 fibrillation in the absence/presence of S100A8 was monitored using a thioflavin T (ThT) fluorescence assay.

3. Results

Ca²⁺-loaded S100A8 was shown to bind monomeric A β 40/42 with equilibrium dissociation constants of $2.7 \pm 1.0 \mu\text{M}$ and $2.8 \pm 0.7 \mu\text{M}$, respectively. S100A8 dramatically suppressed A β 40 fibrillation, as evidenced by a 12–23-fold decrease in maximum ThT fluorescence intensity.

4. Conclusions

S100A8 interacts with A β 40/42 monomer and inhibits A β 40 fibrillation in vitro, thereby suggesting that S100A8 may be involved in the control of A β deposition in AD.

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