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Co-interaction of Tau and S100A9 Proteins

Neurodegenerative diseases are one of the most common disorders in the world, and despite intensive research, the understanding of these diseases is limited. Alzheimer's disease is the most common neurodegenerative disease, affecting about 50 million people worldwide. In addition to amyloid plaques composed of amyloid-β, neurofibrillary tangles formed from the protein Tau are a hallmark of this disease and other tauopathies. Amyloid beta aggregates (and alpha-synuclein aggregates in Parkinson's disease) have been shown to promote Tau aggregation. It has also been observed that the aggregation of these two peptides involves the pro-inflammatory protein S100A9, whose elevated levels in the brain are recorded after various head injuries. Furthermore, Chronic traumatic encephalopathy registers high levels of Tau aggregates, and the exact reasons for their formation are unknown. Researchers observed that this disease is quite prominent in contact sports players who experience chronic head concussions. There has been some speculation that neuroinflammation could induce Tau pathology; thus, it's feasible that S100A9, as a pro-inflammatory protein, could be at least in part responsible. However, it is strange that little information is available to confirm or rule out the potential of the S100A9 protein or its aggregates to participate directly in Tau aggregation. Therefore, we examined the ability of the S100A9 protein to promote Tau aggregation. We observed that Tau aggregation is dependent on S100A9 aggregate formation. Aggregation kinetics were recorded by fluorescence spectroscopy using the amyloidophilic dye thioflavin T. Atomic force microscopy was performed to analyze the morphology of the formed aggregates, and FTIR spectroscopy was done to analyze secondary structures in the formed aggregates. Cell viability studies have also tentatively shown that higher concentrations of S100A9 aggregates are associated with lower toxicity, which may indicate that the formation of S100A9 aggregates and co-interaction with Tau protein acts as a protective mechanism.

User consent

yes

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