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Amyloid-beta and tau pathology following repetitive mild traumatic brain injury

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Abstract

Neurodegenerative diseases are characterized by distinctive neuropathological alterations, including the cerebral accumulation of misfolded protein aggregates, neuroinflammation, synaptic dysfunction, and neuronal loss, along with behavioral impairments. Traumatic brain injury (TBI) is believed to be an important risk factor for certain neurodegenerative diseases, such as Alzheimer's disease (AD) and chronic traumatic encephalopathy (CTE). TBI represents a ubiquitous problem in the world and could play a major role in the pathogenesis and etiology of AD or CTE later in life. TBI events appear to trigger and exacerbate some of the pathological processes in these diseases, in particular, the formation and accumulation of misfolded protein aggregates composed of amyloid-beta (Aβ) and tau. Here, we describe the relationship between repetitive mild TBI and the development of Aβ and tau pathology in patients affected by AD or CTE on the basis of epidemiological and pathological studies in human cases, and a thorough overview of data obtained in experimental animal models. We also discuss the possibility that TBI may contribute to initiate the formation of misfolded oligomeric species that may subsequently spread the pathology through a prion-like process of seeding of protein misfolding.

Keywords: Alzheimer's disease; Amyloid-beta; Chronic traumatic encephalopathy; Neurofibrillary tangles; Protein misfolding; Repetitive mild traumatic brain injury.