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Tau Protein: All You Need to Know

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Study Description

Tau proteins play a role in the stabilization of microtubules. These proteins are found in abundance in nerve cells, while they are considerably less numerous in oligodendrocytes and astrocytes. Tau proteins are most active in the distal regions of axons, where they maintain microtubules while also providing flexibility. The proteins interact along with tubulin, a globular protein, to stabilize microtubules and assist tubulin assembly in microtubules. Tau proteins achieve their control of microtubule stability through isoforms and phosphorylation. In individuals with Alzheimer's disease (AD), tau protein fibrils are commonly found in the brain. These fibrils interfere with the proper transmission between brain neurons, causing the illness. However, it's not quite obvious what these deposits signify or how they're generated.

Phosphorylation and Isoforms

Tau proteins are found in brain tissue in six distinct isoforms. Their binding domains allow them to be distinguished from one another. Their binding domains allow them to be distinguished from one another. In three of the tau protein isoforms, there are three binding domains, while in three other proteins, there are four. The binding domains are found in the carboxyl-terminal end in the protein. An alternative splicing gene called MAPT produces the tau protein isoforms (microtubule-associated protein tau). They were discovered in 1975 by Marc Kirschner's laboratory at Princeton University. In addition, they are all positively charged, thus they can attach to microtubules that have a negative charge.

Protein kinases, such as PKN, are involved in the phosphorylation of tau protein. Phosphorylation *via* activated PKN disturbs microtubule structure by phosphorylating tau. Helical and straight filaments can be caused by hyperphosphorylation of tau proteins (referred to as neurofibrillary tangles). They contribute to Alzheimer's disease pathogenesis. Alzheimer's pathology shares certain similarities with prion disorders, according to some elements of the pathogenesis. Research demonstrates that all six tau isoforms are commonly observed hyperphosphorylated in paired helical filaments when a brain affected by Alzheimer's disease is examined. It also suggests that tau proteins may be produced through an exosome-based process in Alzheimer's disease. In certain other neurodegenerative disorders, abnormal aggregates enriched in tau isoforms have also been detected.

Key Features

Tau is unique among proteins in that it does not have a definite structure. It is described as "intrinsically disordered" and has the ability to take on a variety of forms. Its intrinsic disorder makes it very difficult to elucidate its structure using otherwise routine and extremely useful studies such as X-ray analysis of molecular structure, due to the great flexibility such an attribute bestows. New active tau proteins were identified to facilitate researchers in their investigation of tau aggregation, which is a distinctive feature of neurodegenerative disorders such as Alzheimer's. Tau aggregation can be induced by tau PFFs that attract monomers to form larger tau fibrils. In thioflavin T tests, active tau monomers combined with active tau PFFs resulted in an increase in fluorescence, indicative of tau fibrillization. While full-length PFFs may be more effective at seeding fibrillization, a mixture of the two can be potentially toxic to neuronal cells. There are several ways to fibrillize tau. The most common way is to use Heparin scaffolding.