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Opinion Article

The Responsibility of Cytoskeleton-Associated Proteins in Brain Biogenesis

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DESCRIPTION

In addition, the Cytoskeleton-associated proteins are a collection of structural proteins that bind to microtubules. Many of them have been found in high concentrations in neurons where their expression is tightly regulated during development, implying that they are involved in neuronal morphogenesis. Several of them have recently had their cDNAs cloned and sequenced, revealing their main structures and allowing genetic modification experiments to determine their roles. Experiments have revealed that CAPSs may be divided into two groups based on the amino acid sequence motifs that they use to bind to tubulin. One of these classes has three known genes, two of which have been expressed in non-neuronal cells and cause microtubule bundling and rearrangement inside the cytosol. The pathway of this reordering is at present dubious. Another notable aspect of these proteins is that several of them are dispersed differently within the neuronal cytoplasm; for example, some variants of CAPS2 are only found in dendrites, whereas CAPS tau is only found in axons in many cases. CAPS2 mRNA, which encodes the protein, is also found in dendrites. This shows that CAPS2 production is controlled at a local level in the dendritic cytoplasm. The molecular mechanism behind protein sorting within neurons is currently unknown.

Dendrites and a long axon form the highly polarised morphology of neurons. Microtubules and Cytoskeleton-Associated Proteins (CAPSs) with distinct architectures are found in both axons and dendrites. CAPS2 is expressed only in dendrites, whereas CAPS2C and tau are prevalent in the axon. However, it is uncertain how CAPS2, CAPS2C, and tau affect the arrangement of microtubule domains in dendrites versus axons. After transfection of complementary DNAs, both CAPS2 and tau stimulate microtubule bundle development in fibroblasts, and Sf9 cells infected with recombinant baculovirus expressing tau extend a long process resembling an axon. CAPS2 and CAPS2C were expressed in Sf9 cells in order to compare their shape and microtubule arrangement to those of Sf9 cells expressing tau.

The range between microtubules varies depending on whether CAPS is expressed: in cells expressing CAPS2, the distance is comparable to that of dendrites, whereas in cells expressing CAPS2C or tau, the distance is comparable to that of axons. CAPS2 and tau, both cytoskeleton-associated proteins, have two functional domains that can be separated. The first is a microtubule-binding site that initiates microtubule assembly; the second is a short C-terminal alpha-helical region that cans crosslink microtubules into dense, stable parallel arrays similar to axons or dendrites using a hydrophobic zipper interaction. As a result, interactions between molecules of the same type can radically reorganise microtubules while also entirely dampening their dynamic capabilities.

To interact with microtubules, F-actin, and intermediate filaments, the neuronal proteins Tau and CAPS2 have homologous C-Terminal MT-Binding Regions (MTBRs). Despite the fact that Tau-MTBR is the main component of pronase-treated Alzheimer's paired helical filaments, Tau and CAPS2 both produce filaments in vitro from disulfide-linked homodimers. The fact that the crucial thiol is located inside a region required for MT interaction raises the question of whether disulfide production prevents Tau-Tau or CAPS2-CAPS2 dimers from attaching to microtubules, causing dimers to divert toward filament formation. Cross-linked Tau and CAPS2 homodimers rapidly induce tubulin polymerization, according to the study, and monomer and dimer affinity for MTs is remarkably identical. As a result, disulfide cross-bridging into homodimers is unlikely to constitute a driving force in Alzheimer's disease filament production.

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