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Moonlighting function of the disordered TPPP/p25 involved in neurodegenerative diseases

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In silico and experimental analysis revealed that a significant part of the products of the human genome are *disordered* proteins which fulfill essential physiological and pathological functions. This moonlighting function is originated from its multifarious macromolecular associations occurring at different intracellular conditions. The disordered neomorphic moonlighting proteins like TPPP/p25 frequently cause undesired protein aggregations, most of them are tightly linked to *neurodegenerative diseases* such as Parkinson's disease (PD) and multiple system atrophy (MSA). TPPP/p25 has extended unstructured segments at the N- and C-terminals straddling a flexible region which includes segments for specific binding of GTP, zinc, α-synuclein and β-amyloid. The physiological function of TPPP/p25 is coupled with the modulation of the dynamics and stability of the microtubule (MT) network by its tubulin polymerization and acetylation promoting as well as MT bundling activities. The TPPP/p25 level is controlled by microRNA and by the proteosoma machinery. In normal brain TPPP/p25 protein is expressed predominantly in the zinc-rich oligodendrocytes (OLGs); it is indispensible for the differentiation of the progenitor cells leading to OLG maturation necessary to the axon ensheathment. The destruction of the myelinization results in the multiple sclerosis (MS) coupled with loss of TPPP/p25-positive OLGs in brain with concomitant enrichment of TPPP/p25 in the liquor. Abnormal co-enrichment of TPPP/p25 with α-synuclein results in synucleinopathies. The absence of TPPP/p25 expression is characteristic for glioma. The moonlighting characteristics of TPPP/p25 originated from its interactions with distinct proteins at physiological or pathological conditions make it powerful diagnostic marker and potential drug target

Biography

JO has completed her PhD (1973) and DSc (1986) from the Institute of (Biochemistry) Enzimology, HAS, Budapest. She is the chief of the Cell Architecture research group from 1989. She was involved in one of the largest Life Science EU projects, BioSim (2005-2011) as a head of a research team and as a governing broad member. Her specialty: biochemistry, structural biology, molecular neurology, system biology. She was invited visiting professor at Universities of USA, Spain and Italy. Editorship: FEBS Letters and IUBMB Life. Her recent research is supported by EU, National Research Foundation as well as Hungarian and German companies. Publication activity: 188 papers with 466 impact factor and 3344 citation; Hirsch index: 30. Her scientific activity was awarded such as Chares Simonyi and Széchényi fellowships, Straub medal and Knight Cross of the Hungarian Republic

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