Synapsin

Synapsins are a family of synaptic vesicle-associated phosphoproteins that play a crucial role in regulating neurotransmitter release at synapses. They are primarily found in nerve terminals and are associated with the reserve pool of synaptic vesicles. Synaptic vesicles store neurotransmitters and their release is a fundamental process in neuronal communication.

There are three main isoforms of synapsin proteins, creatively named synapsin I, synapsin II, and synapsin III. These isoforms are encoded by different genes (SYN1, SYN2, and SYN3, respectively). Each isoform has specific functions and expression patterns in the nervous system.

The primary roles of synapsins include:

Regulation of Neurotransmitter Release: Synapsins tether synaptic vesicles to the cytoskeleton, helping to maintain vesicles in a reserve pool. This regulation influences the availability of vesicles for release during synaptic activity.

Organization of the Reserve Pool: Synapsins contribute to the organization and maintenance of the reserve pool of synaptic vesicles, which can be mobilized to the active zone during increased neuronal activity.

Synaptic Plasticity: Synapsins are implicated in synaptic plasticity, which is the ability of synapses to change their strength over time. This involvement suggests a role in learning and memory processes.

Neuronal Development: During early neuronal development, synapsins are involved in the formation and maturation of synapses.

Studies on synapsins have contributed significantly to our understanding of synaptic transmission and the molecular mechanisms underlying various neurological processes. Abnormalities in synapsin function have been associated with neurological and psychiatric disorders, including epilepsy and schizophrenia. Researchers continue to explore the specific functions and regulatory mechanisms of synapsins to gain insights into the complexities of synaptic function and dysfunction in various conditions.

Shen et al. identified that elevated synapsin 2a (Syn2a) in the infralimbic cortex (IL) to basolateral amygdala (BLA) circuit disrupted presynaptic orchestration, leading to an excitatory/inhibitory imbalance in the BLA region and causing extinction resistance.Overexpression or silencing of Syn2a levels in IL neurons replicated or alleviated behavioral, electrophysiological, and biochemical phenotypes in resistant mice. They further identified the proline-rich domain H in the C-terminal of Syn2a was indispensable for the interaction with synaptogyrin-3 (Syngr3) and demonstrated that disrupting this interaction restored extinction impairments. Molecular docking revealed ritonavir, an FDA-approved HIV drug, could disrupt Syn2a-Syngr3 binding and rescue fear extinction behavior in Syn2a-elevated mice. In summary, aberrant Syn2a elevation and its interaction with Syngr3 at the presynaptic site were crucial in fear extinction resistance, suggesting a potential therapeutic avenue for related disorders [1)].

1)

Shen XY, Zhang J, Huang HZ, Li SD, Zhou L, Wu SP, Tang C, Huang X, Liu ZQ, Guo ZY, Li X, Man HY, Lu YM, Zhu LQ, Liu D. Synapsin 2a/Synaptogyrin-3 interaction regulates fear extinction in mice. J Clin

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