New theory in neuroscience: Common mechanisms in Fragile X and Down syndrome

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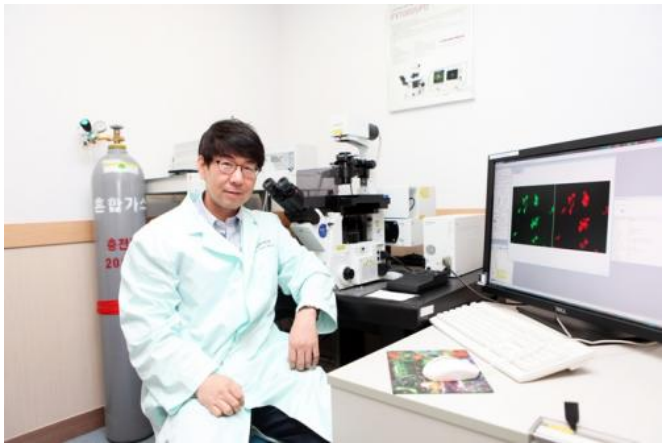

This is Prof. Kyung-Tai Min from UNIST (Ulsan National Institute of Science and Technology). Credit: UNIST

A new common mechanism in Fragile X and Down syndrome has been identified by scientists at Ulsan National Institute of Science and Technology (UNIST), Korea and published in the world leading science journal *Trends in Neurosciences*.

Emerging evidence shows that the regulation of local protein synthesis in dendritic spines plays a crucial role in controlling synaptic morphogenesis and synaptic efficacy. However, scientist do not yet understand how local protein synthesis regulates dendritic spine morphology, a process that is important for learning and memory.

The research team led by Prof. Kyung-Tai Min from UNIST presented evidence that pathways controlled by DSCR1 and FMRP, genes implicated in two of the most common genetic causes of intellectual disabilities – DS and FXS converge to regulate spine morphogenesis, local protein synthesis, and neurotransmission.

Min's research team highlighted the *previous research work published in The EMBO Journal by Prof. Min, showing that some of the proteins altered in Fragile X and Down syndrome are common molecular triggers of intellectual disability in both disorders, DS and FXS.

*Title: DSCR1 interacts with FMRP and is required for spine morphogenesis and local protein synthesis (The EMBO Journal (2012) 31, 3655 – 3666)

They reviewed other genes encoded by chromosome 21 that may regulate dendritic spine morphogenesis and contribute to intellectual disabilities by acting through pathways involving FMRP and DSCR1.

The research work provided an important stepping stone in understanding the multiple roles of DSCR1 in neurons and in identifying a molecule that is closely linked to intellectual disability for both syndromes.

"We will continuously investigate whether reducing FMRP in DS mouse model or elevating DSCR1 in FMRP knockout mice could restore synaptic plasticity, dendritic spine morphogenesis, and local protein synthesis will further advance our understanding of both diseases," said Prof. Min, presenting future research plan.

"Further elucidation of the large functional protein-inter-action network that regulates local protein synthesis, spine morphogenesis, and synaptic transmission may also shed light on overlapping molecular pathways that cause intellectual disabilities in different disorders," added Prof. Min.

More information: Meeting at the crossroads: Common mechanisms in Fragile X and Down

syndrome,
http://dx.doi.org/10.1016/j.tins.2013.08.007

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