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세미나 시리즈

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NMDA receptor dysfunction and sexual dimorphism in mouse models of autism

+2 두번째

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Abstract

Autism spectrum disorders (ASDs) represent a group of neurodevelopmental disorders characterized by impaired social and communication deficits and restricted and repetitive behaviors. ASDs are ~four times more common in males than in females for unknown reasons. Although a large number of ASD-related genetic variations have been identified, only a small number of them have been verified for their causality by approaches including mouse genetics. In addition, neuronal, synaptic, and circuit mechanisms underlying the development of ASDs remain largely unknown. Recently, NMDA receptor dysfunction has been suggested to underlie ASDs, but it remains unclear how general this hypothesis could be and how early the pathophysiology begins during the development of the brain. In addition, little is known about the mechanisms underlying the male preponderance in ASDs even at the level of mouse models of ASD. In this presentation, I will discuss the temporal aspects of NMDA receptor dysfunctions and sexually dimorphic phenotypes in mouse models of ASD.

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