



The bidirectional role of MeCP2 in the brain; a multi layer epigenetic deregulation in MeCP2-associated brain disorders

SEMINAR & VISITING SPEAKER SERIES

DATE

Friday, May 26th, 2017
9:00AM

LOCATION

PX236/238
PsychHealth Building

SPEAKER

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ABSTRACT:

Epigenetic mechanisms control gene expression and brain development without any direct change in the corresponding DNA sequences. Recent discoveries have highlighted the importance of epigenetic mechanisms in brain development, neuroscience, and mental health. MeCP2 is an important epigenetic factor in the brain with a bidirectional functional role that is controlled at multiple levels. While, MeCP2 loss-of-function mutations lead to Rett Syndrome (RTT), its gain-of-function genetic mutations cause MECP2 Duplication Syndrome (MDS) through gene duplication or triplication in the patients. RTT and MDS disorders have overlapping symptoms, and the patients show similar and severe neurological phenotypes that include mental disability, seizures, anxiety, and autism. It is well established that impaired protein translation is a characteristic of human RTT neurons. However, the underlying molecular mechanism of this phenotype is not fully understood. To study the underlying pathobiology of RTT and MDS, my lab investigates the bidirectional role of individual MeCP2 isoforms in controlling fundamental molecular pathways that regulate protein translation. We use a combination of primary neural stem cells, RTT and MDS mouse models, and post-mortem human RTT brain tissues to investigate how molecular deficiencies at the cellular levels lead to compromised brain function in RTT, MDS, and other MeCP2-associated brain disorders including autism.

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