Seminar/Talk

Genetics of epigenetics: relevance for cognitive disorders

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Host: Gaia Novarino

Neurodevelopmental disorders (NDDs), including intellectual disability (ID) and autism spectrum disorders (ASD), are phenotypically and genotypically heterogeneous. The molecular pathways involved in these disorders, however, are rapidly being elucidated by the identification of mutations in genes that cause cognitive impairments in humans. While the identification of disease genes has great benefits for diagnostic and prognostic purposes, for the vast majority of these genes, there is little conceptual knowledge about neurobiological mechanisms that they control at the cellular and network level.
One emerging group of NDDs, named chromatinopathies, are caused by mutations in epigenetic regulators of gene expression. We have recently identified Euchromatin Histone Methyltransferase 1 (EHMT1) as a key modulator in cognition. Heterozygous loss-of-function alleles of this gene cause a specific ID syndrome, denoted Kleefstra syndrome, which is characterized by moderate-severe ID, autism, microcephaly, and dysmorphic features. EHMT1/GLP cooperates with its mammalian paralog EHMT2/G9a and exhibits enzymatic activity in histone 3 lysine 9 mono-and di-methylation (H3K9me1 and H3K9me2), which is known to promote heterochromatic structure and gene repression. Here I will discuss our recent advances towards understanding the contribution of epigenetic regulation of gene expression, through H3K9me2, in learning and memory and specific forms of synaptic plasticity in rodent and hIPSC models.

Thursday, February 23, 2017 02:00pm - 03:00pm
Seminar Room, Lab Building East

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