EpiGenetic Conference BIU
June 2019

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Day 2- 25.6, Plenary II- 09:00-10:30

Is DNA methylation a genomic mechanism for long term adaptation to social environments?

Abstract:

There are well established examples for social environments to have long lasting effects on the phenotype. What are the mechanisms that embed these social signals in the genome to result in stable and long-lasting changes to genomic functions? DNA methylation is a mechanism that marks genes during development and provides identical DNA sequences with different identities. Experiments in rodents demonstrated that low maternal care resulted in changes in DNA methylation in the glucocorticoid receptor gene in the hippocampus, which remained throughout life and altered the life-long behavior of the offspring increasing anxiety and responsivity to stress. We will present data from nonhuman primates and humans indicating that DNA methylation states are altered in response to both prenatal and postnatal stress in multiple tissues; placenta, the immune system and the prefrontal cortex. We propose that the changes in DNA methylation in response to early life adversity are “adaptive genomic” mechanisms that adapt life-long genome programming to the anticipated life-long environment based on signals received during gestation and early life. Social adaptation by epigenetic mechanisms is not limited to early life experiences. Later experiences such as exposure to injury, drugs or trauma might elicit epigenetic alterations that might be playing a similar role in social adaptation to these exposures.