Département de psychiatrie
Centre de neurosciences psychiatriques
Site de Cery
CH-1008 Prilly - Lausanne

Centre de Neurosciences Psychiatriques

CNP SEMINAR

ANNOUNCEMENT

Friday, October 10, 2014, 14:00

"Developmental Stress and Long-Term Psychiatric Outcomes: Shaping Pathology by Gene-Environment and Environment-Environment Interactions"

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Epidemiological research and translational work in animal models suggest that exposure to traumatizing experiences during sensitive periods of postnatal brain maturation can increase the risk of long-term psychiatric disorders. The nature and strength of this association is likely influenced by the genetic background of the affected individuals and/or by interactions with other environmental adversities. To explore the specificity of brain pathology following developmental stress exposure, our research team compares the effects of peripubertal or adolescent stress in multifactorial mouse models that encompass epidemiologically relevant environmental risk factors and specific susceptibility genes implicated in schizophrenia and related disorders. We have recently demonstrated that peripubertal stress exposure induces more severe neuropathological long-term effects in offspring with a history of prenatal immune activation as compared to offspring without such a history. Hence, prenatal immune adversities can function as a "disease primer" that increases the offspring's vulnerability to the detrimental neuronal effects of subsequent stress exposure during peripubertal life. Our ongoing research now reveals that peripubertal stress exposure can similarly interact with rare copy number variation (CNV) in the form of a 15q13.3 microdeletion syndrome. In both cases, we further found that a later application of stress in adolescence did not elicit the interaction with the environmental (prenatal immune activation) or genetic (15q13.3 microdeletion) predisposing factor, suggesting that the precise timing of postnatal stress is a critical determinant of long-term brain pathology in multifactorial disease models. Our findings provide experimental support for the hypothesis that the impact of developmental stress on adult brain functions is strongly influenced by the genetic and environmental contexts in which it occurs. Exposure to peripubertal stress may thus be an important etiological risk factor for long-term psychiatric illness especially in individuals with genetically and/or environmentally driven disease predisposition.

Selected Publications:

1. Sandra Giovanoli et al: Stress in Puberty Unmasks Latent Neuropathological Consequences of Prenatal Immune Activation in Mice. Science 339, 1095 2013; DOI: 10.1126/science. 1228261
2. Kim Fejgin et al: A Mouse Model that Recapitulates Cardinal Features of the 15q13.3 Microdeletion Syndrome Including Schizophrenia- and Epilepsy-Related Alterations. Biol Psychiatry 2014; 76: 128-137.
3. Stéphanie Vuillermot et al: Prenatal Immune Activation Interacts with Genetic N*urr1* Deficiency in the Development of Attentional Impairments. J Neurosci, 2012; 32(2): 436-451

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