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The interaction between stress and maternal VIP on nervous system development in mice: evidence for crucial integrative functions of placenta

VISITING SPEAKER

DATE
Thursday, April 20th, 2023

10:00 AM - 11:00 AM

LOCATION
Apotex Centre 050

Zoom ID: 661 1680 4750
Pass code: 901360

SPEAKER

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BIO

1997 PhD in Molecular and Cellular Biology at the University of Poitiers, France

1997-2001 Postdoctoral fellow at the University of California (Jonsson Cancer Center Foundation fellow)

2001-2004 Research Assistant, MRRC at UCLA

2004-2007 Research Assistant, INSERM U676 (P. Gressens dir.), Robert Debre Pediatric Hospital in Paris (InsermAvenir Fellow)

2007 Associated Professor at Unistra- Team "Neuropeptides and Cerebral Development"

2011 Head of the Joint Masters in Neuroscience (Strasbourg, Basel, Freiburg)

2012 Full professor at Unistra, Team "Molecular Determinants of Pain"

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ABSTRACT

Demonstrated in early 90's, the vasoactive intestinal peptide (VIP) triggers trophic action on the developing embryos with enhanced affinity for the neural tube (Gressens et al, 1993). Thus, in mice, VIP is secreted in high concentration by the placenta as early as embryonic day E7 whilst neural tube of developing embryos starts producing its own VIP later around E10, (Maduna & Lelievre, 2016). In mammals, placenta releases trophic hormones and growth factors and redirects maternal endocrine, immune, and metabolic functions to the embryo's advantage. Conversely, deleterious maternal conditions including stress may alter the whole prenatal growth leading to neurodevelopmental and psychiatric disorders, such as syndromic microcephaly or autistic spectrum disorder. Numerous publications and unpublished data suggested that pregnant females with impaired VIP signaling produce embryos with abnormal neural tube and placenta outgrowth, then give birth to pups with abnormal brain development (smaller brain, autistic-like phenotype and hypersensitivity to pain). In details, these dramatic effects on brain outgrowth are consistent with an embryonic MCPH1-related phenotype (Passemard et al, 2011). This downregulation of MCPH1 expression will affect cell cycle duration and accelerate the neuronal differentiation process. More striking, these deficits were amplified when mothers are stressed during gestation (maternal restraint paradigm). This could be representative of the dual/multiple-hit hypothesis for brain disease, postulating that sequential detrimental events, such as genetic defects and stress, could prime the developing brains and make them highly vulnerable to later challenges triggering unexpected behavioral deficits or psychiatric disorders. The overall aim of these studies is to better understand the link between nervous system development and prenatal stress, and the potential neurotrophic functions that placental factors and VIP may play during these critical periods of neurodevelopment.

OBJECTIVES

1) To quantify the brain size deficit in vasoactive intestinal peptide (VIP)-deficient mice (especially the cortical one) in a more spatio-temporal manner

2) To address the complex question of the (dual) origin of the deficit in VIP

3) Emphasize on the "double hit hypothesis" using maternal restraint" as a model of psychosocial stress

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