FC29-02

IS PRENATAL EXPOSURE TO ALCOHOL A FACTOR WHICH RE-PROGRAM THE BRAIN ?

J.H. Sliwowska
Department of Histology and Embryology, Institute of Zoology, Poznań University of Life Sciences, Poznan, Poland

Introduction: Fetal programming refers to the concept that early environmental factors, including prenatal exposure to stress and drugs, can permanently organize or imprint physiological and behavioural systems and increase vulnerability to disorders such as depression and anxiety later in life.

Aims: Is prenatal exposure to alcohol a factor which re-programs the brain?

Objectives: Effects of prenatal alcohol exposure (PAE) on:

1) the hypothalamus-pituitary-adrenal (HPA) axis;

2) the hypothalamus-pituitary-gonadal (HPG) axis;

3) serotonergic (5-HT) system and

4) adult hippocampal neurogenesis are presented.

Methods: Offspring from prenatal ethanol (PAE), pair-fed (PF) and ad lib-fed control (C) dams are studied across the development or in adulthood. Immunocytochemistry and in situ hybridization techniques are used.

Results: In term of the HPA axis: PAE alters the balance of mineralocorticoids/glucocorticoids (MRs/GRs) receptor levels in the hippocampus of adult females. In the case of the HPG axis: PAE delays puberty and changes hormonal profiles in males and females. PAE also decreases numbers of 5-HT-immunoreactive neurons in the dorsal raphe nucleus of the brainstem in ovariectomized rats and estradiol and progesterone modulate those effects. Finally, in adult PAE males, but not females stress-induced decrease in neurogenesis is altered.

Conclusions: In our animal model PAE re-programs the brain. Effects of PAE are long-lasting, affect HPA and HPG axes, 5-HT system and adult hippocampal neurogenesis and if seen in humans could contribute to increased vulnerability to depression and anxiety.

Founding: NIH/NIAA AA007789, UBC Human Learning Partnership, Bluma Tischler Fellowship and IMPART Fellowship (CIHR grant).