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Editorial

Vitamins Deficiency and Epigenetic Changes of Genes Involved in the Alzheimer's Disease

Saurabh Srivastav*

Department of Physics, Indian Institute of Science, Bangalore-560012,India

Alzheimer's disease (AD) is characterized by progressive neurodegenerative impairment of the central nervous system and is the most prevalent form of dementia. Considering the influence of maternal nutrition on fetal programming, which consequences usually come later in life, we investigated whether maternal vitamin B deficiency during early development alters the offspring expression of genes related to AD etiopathogenesis. Mice dams were submitted to experimental diet one month before and during pregnancy or pregnancy/lactation and, after birth, their offspring were distributed into three groups: control "CT", deficient pregnancy "DP" and deficient pregnancy and lactation "DPL". At postnatal day (PND) 0, a significant decrease of App in females (p=0.007) and App and Bace1 in males (p=0.030 and p=0.040, respectively) was observed when compared to CT group. At PND 28, DPL female presented an increase of App, Bace1 and Ps1 gene expression when compared to CT (p=0.003, p=0.003 and p=0.002, respectively) and DP groups (p=0.017, p=0.005 and p=0.002, respectively). In males at PND 28, a decrease of App and Ps1 was observed in both DP (p=0.012; p=0.001) and DPL (p=0.001; p=0.04) when compared to CT group. No differences were observed in females and males at PND 210. Regarding APP, BACE1 and PS1 protein expression and global DNA methylation pattern, no difference was observed throughout development in female or male offspring. Regarding behavioral evaluations, no changes were observed in the object recognition task, but the DPL males presented lower locomotor activity when compared to DP (p=0.028) and CT (p=0.003) groups. In conclusion, the early exposition to vitamin B deficiency alters the expression of genes related to AD.

***Corresponding to**: Saurabh Srivastav, Department of Physics, Indian Institute of Science, Bangalore-560012,India, E-mail: ssaurabh@iisc.ac.in

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