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Dietary methyl donor deficiency during pregnancy in rats shapes learning and anxiety in offspring.

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Abstract

Two important lines of research have enhanced our understanding of the molecular role of nutrition in influencing behavior. First, exposure to an adverse environment during early life can influence the long-term behavior of the offspring. Second, regulation of the nervous system development and functioning appears to involve epigenetic mechanisms that require a continuous supply of methyl group donors in food. We hypothesized that a maternal diet during pregnancy deficient in methyl donors (MDD) may lead to altered behavior in offspring through permanent changes in hippocampal DNA methylation. We used a rat model of prenatal dietary MDD to test this hypothesis in female offspring as they aged. Prenatal MDD reduced birth weight, litter size, and newborn viability. Aged female offspring of MDD mothers showed increased anxiety and increased learning ability in comparison with control diet group offspring. To explore the role of MDD on epigenetic mechanisms in the brain of adult offspring, we studied expression and methylation of 4 selected genes coding for glucocorticoid receptor, hydroxysteroid dehydrogenase 11 type 2, **neuronatin**, and reelin proteins in the hippocampus. No major group differences in methylation or expression of the studied genes were detected, except for a significant down-regulation of the reelin gene in the MDD female offspring. The prenatal MDD diet caused intrauterine growth restriction, associated with long-term effects on the behavior of the offspring. However, the observed behavioral differences between the MDD and control diet offspring cannot be explained by epigenetic regulation of the specific genes investigated in this study.

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