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Efekty działania alkoholu w okresie prenatalnym w modelu zwierzęcym

Effects of alcohol action in prenatal period in animal model

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Abstract – The developing brain is extremely sensitive to the effects of ethanol. Heavy consumption of ethanol during pregnancy can result as the morphological and neurological changes called the fetal alcohol syndrome (FAS). The growth retardation, facial anomalies and mental retardation in infants born to alcoholic women were observed as a teratogenic outcome of alcohol consumption during pregnancy. Ethanol consumption during gestation can produce long-lasting alterations in neuromodulatory influences on $GABA_A$ receptor-mediated inhibitory neurotransmission in adult offspring. The prenatal ethanol-induced changes may have consequences of differential $GABA_A$ receptor subunit expression.

Rat model of the fetal alcohol exposure (FAE) has been shown behavioral deficits that are linked to electrophysiological changes in the long-term potentiation (LTP). Signal-activated phospholipase C (PLC) and phospholipase A_2 are critical to the induction and maintenance of LTP. Then, alterations of phospholipids metabolism may play a significant role in the LTP deficities observed in FAE offspring.

Number of studies suggest that the hypofunction of the dopaminergic (DA) system may be related to the attention deficits and hyperactivity problems reported in children with fetal alcohol effects or fetal alcohol syndrome. Prenatal ethanol exposure significantly reduced the number of spontaneously active DA neurons in the substantia nigra and ventral tegmental area in 5 month-old male rat's offspring. Further research is ne-

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eded to increase understanding of consequences, risk factors, mechanism, as well as prospects prevention and treatment.

Key words: Fetal alcohol syndrome (FAS), long-term potentiation, ethanol, animal model