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PRENATAL ETHANOL EXPOSURE MODIFIES LOCOMOTOR ACTIVITY AND INDUCES SELECTIVE CHANGES IN MET-ENKEPHALIN CONTENT IN ADOLESCENT RATS

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Prenatal ethanol exposure (PEE) facilitates alcohol acceptance and intake, suggesting that ethanol in utero may increase the probability of drug abuse during adolescence and adulthood. Alcohol reinforcement involves the ethanol-induced activation of opioidergic systems in mesocorticolimbic areas. Changes in opioid neurotransmission may be relevant during ethanol intoxication, as well as in the adaptive neural responses induced by the drug. Some studies have assessed the possible changes in opioidergic systems as a function of ethanol exposure in adolescent animals. However, PEE effects upon locomotive responses elicited by an ethanol challenge and modulation of neurotransmission of opioidergic systems remain to be understood. This work assessed the susceptibility of adolescent rats to prenatal and/or postnatal ethanol exposure in terms of locomotive responses, as well as alcohol-related effects on Methionine-enkephalin (Met-enk) expression in brain areas related to drug reinforcement. Pregnant rats received a daily intragastric administration of ethanol (2 g/kg) or water, during gestational days 17-20. Adolescents at postnatal day 30 (PD30) were tested in a first baseline trial (habituation session) and evaluated in terms of spontaneous activity. Thereafter, animals received an ip injection of vehicle (saline 0.9% w/v) (vehicle session) and were immediately evaluated in terms of activity during 30 min. After this second trial, animals from both prenatal treatments were injected with ethanol (1.0 g/kg ip) or saline, and locomotor activity was immediately assessed for 30 min (drug session). Met-enk content was quantitated by radioimmunoassay in several brain regions: ventral tegmental area [VTA], nucleus accumbens [NAcc], prefrontal cortex [PFC], substantia nigra [SN], caudate-putamen [CP], amygdala, hypothalamus and hippocampus. PEE significantly reduced rearing responses. Ethanol challenge at PD30 decreased horizontal locomotion and showed a tendency to reduce rearings and stereotyped behaviors. PEE increased Met-enk content in the PFC, CP, hypothalamus and hippocampus, but did not alter peptide levels in the amygdala, VTA and NAcc. These findings suggest that PEE selectively modifies behavioral parameters at PD30 and induces specific changes in Met-enk content in regions of the mesocortical and nigrostriatal pathways, the hypothalamus and hippocampus. Prenatal and postnatal ethanol actions on motor activity in adolescents could involve activation of specific neural enkephalinergic pathways.