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"DETERMINANTS OF ALCOHOLISM: BRIDGING THE GAP BETWEEN EPIDEMIOLOGICAL AND BASIC RESEARCH"

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LEARNING EXPERIENCES COMPRISING CENTRAL ETHANOL EXPOSURE IN RAT NEONATES: EFFECTS UPON RESPIRATORY PLASTICITY AND THE BRAIN CATALASE SYSTEM

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Fetal ethanol (EtOH) exposure represents a risk factor for the Sudden Infant Death Syndrome and it's early effects upon respiration also promotes hypoxic ischemic consequences. This study analyzes central ethanol's effects upon breathing plasticity during a stage in the development of the rat equivalent to the 3rd human gestational trimester. The study not only analyzed ethanol's unconditioned breathing effects but also how they are regulated by learning processes. Taking into account that ethanol is primarily metabolized in the brain via the catalase system, we examined the effects of early history with the drug upon the activity of this enzymatic system. During postnatal days 3, 5 and 7 (PDs 3-7) pups either received intracisternal (i.c.) administrations of vehicle or ethanol (300 mg%). They were subsequently exposed to a whole body plethysmograph under normoxia. The apparatus was scented or not with the ethanol odor. The presence of the odorant increased breathing rates. The state of intoxication attenuated the onset of apneas; a phenomenon indicative of an antianxiety effect of the drug given the state of arousal caused by the novel environment, maternal deprivation and the stress of i.c. administrations. At PD9, pups were tested while sober under sequential air conditions (initial-normoxia, hypoxia and recovery-normoxia). Once again the plethysmograh was unscented or contained EtOH odor. Prior experience with the scented chamber associated with EtOH's central effects elicited a conditioned isodirectional response relative to the onset of apneas previously observed during PDs 3-7. Yet, prior history with the drug exacerbated the onset of apneas when pups were defied with hypoxia. Following this test, pups ingested 0.8 g/kg of absolute EtOH and their brains were analyzed to determine catalase activity. Pre-exposure to EtOH's central effects paired with the odor of the drug resulted in heightened enzymatic activity. The results indicate that central EtOH accumulation may exert antianxiety effects that attenuate apneic disruptions but that also has long-lasting effects upon respiratory plasticity under hypoxia. Most importantly, these effects appear to be related with how the brain catalase system reacts to the presence of EtOH in accordance with the nature of prior experiences with the drug.