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INDIVIDUAL DIFFERENCES PREDICTIVE OF ETHANOL-INDUCED REINFORCEMENT AND ETHANOL INTAKE DURING ADOLESCENCE

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Likelihood of alcohol use disorders is greater in adolescence than in other developmental stages. Greater novelty-seeking, enhanced sensitivity to stress or to ethanol-induced appetitive effects, or lessened sensitivity to ethanol-induced sedation may underlie adolescent vulnerability to alcohol-related problems. The present work describes studies assessing susceptibility to ethanol reinforcement and intake in subpopulations of rats that differed in their innate levels of novelty seeking or anxiety patterns, or in their level of response to ethanol. In an early study we found that binge ethanol exposure early on adolescence enhanced ethanol intake and that adolescents exhibiting greater sensibility to the motor activating effects of binge ethanol administration were even more predispose to drink ethanol. Subsequent work indicated that the motor stimulating effect of ethanol and the facilitative effect of ethanol binge exposure on later ethanol intake are greater in adolescent than in adult rats. Moreover, enhanced level of exploration in an open-field at adolescence was associated with greater ethanol-induced conditioned place preference and reduced ethanol-induced conditioned taste aversion. Recent work indicated that adolescents exhibiting reduced exploration of the open arms of an elevated plus maze and higher anxiety response in a light-dark maze drank significantly more ethanol than counterparts with average levels of anxiety. Similar enhancement in ethanol intake was found in adolescents that had been chronically exposed to maternal deprivation. These results suggest that certain subpopulations of adolescents – those with innate or stress-induced high levels of anxiety, exacerbated novelty-seeking response or enhanced sensitivity to ethanol-induced psychomotor effects – may be at greater risk of elevated alcohol intake.

ENVIRONMENTAL TOBACCO SMOKE IN EARLY POSTNATAL PERIOD INDUCES IMPAIRMENT IN MEMORY, SYNAPTOGENESIS AND MYELINATION

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Brain development represents a period of vulnerability and several substances can induce neurotoxicity in this phase. The aim of our group is the study the effects of the environmental tobacco smoke (ETS) exposure during postnatal early brain development. BALB/c mice were exposed to a mixture of central (10%) and lateral (90%) tobacco smoke of the reference cigarettes 3R4F from the 3rd (P3) to the 14th (P14) day of life, twice a day(one hour each exposure at 8 a.m. and 4 p.m.). Memory (Morris water maze), locomotion (open field), anxiety (plus maze), synaptogenesis (synapsin, synaptophysin and BDNF) and axonal myelination (percentage of myelinated fibers in the optic nerve and myelin basic protein (MBP) of cerebellum, brainstem, diencephalon and telenchephalon) were evaluated during infancy, adolescence and adulthood. Our results showed that ETS induced impairment in learning and memory and increased anxiety in all the ages evaluated. ETS also induced impairment in synaptic transmission, by a decrease in synapsin, synaptophysin and BDNF in hippocampus, cerebellum and prefrontal cortex as compared to the control group. The percentage of myelinated fibers in the optic nerve in childhood and MBP levels in telencephalon and brainstem were lower in adolescence exposed to ETS compared to the control group. In cerebellum, there was an increase in MBP levels in infants and a decrease in adults compared to the control group. Taken all together, our results suggest that the exposure to ETS in early postnatal period induces impairment to the brain development. It is noteworthy that these effects are most evident during infancy, however not all effects are reversed in adolescence or even in adulthood.