

IX INTERNATIONAL MEETING OF THE LATIN AMERICAN SOCIETY FOR BIOMEDICAL RESEARCH ON ALCOHOLISM (LASBRA) NOVEMBER 7TH, 8TH AND 9TH, 2019.

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

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by the main public healthcare institutions; and information about sexual and reproductive/non-reproductive rights of women in accordance to our national current laws. We conclude that an effective prevention strategy for alcohol use during pregnancy and breastfeeding should be based on a more complex, integrative view about women's health, including community-based interventions and sexual education contents. Grants: SEDRONAR, convenio Fundación Abriendo Corazones Resol-2018-496-APN-SEDRONAR.

SENSITIZATION TO ETHANOL'S DISRUPTIVE EFFECTS UPON EARLY BREATHING PLASTICITY ASSOCIATED WITH HYPOXIA AND HYPERCAPNIA.

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Ethanol (EtOH) consumption during pregnancy and lactation represents a risk factor related with the Sudden Infant Death Syndrome (SIDS). This phenomenon has promoted research linking prenatal EtOH effects on the respiratory system during early ontogeny. It should also be noted that prolonged episodes of neonatal respiratory depression represent a risk factor in terms of hypoxic-ischemic effects with negative consequences on brain development. In a first study during postnatal day (PD) 9 we analyzed the impact of different doses of EtOH (0.0, 0.75, 1.37 or 2.0 g/kg) upon the respiratory response and the potential psychomotor effects in pup rats pre-exposed or not to 2.0 g/kg of EtOH during PDs 3-7. At PD 9 animals were also subjected to sequential air conditions defined as initial normoxia, hypoxia and recovery normoxia. In a second study we analyzed the blood of animals only exposed to 0.0 or 2.0g/kg of EtOH during PDs 3-9 (not subjected to a hypoxic event). The aim was to examine if mere intoxication with a moderate dose of EtOH is capable of modifying blood metabolic patterns associated with hypoxia or hypercapnia. In the first study during PDs 3-7 EtOH exerted a depressant effect upon breathing frequencies. These animals also showed a progressive sensitization effect relative to the depressant effects of the drug and lesser levels of apneas. At PD 9 dose dependent respiratory depressions were observed when pups were challenged with a hypoxic event. Independently from prior experience with EtOH, drug treatment at PD 9 significantly disrupted respiratory frequencies particularly during the hypoxic and the recovery normoxia phases. Respiratory disorders triggered by these air conditions have been implicated in the pathophysiology of SIDS. These results show that breathing plasticity is disrupted during a critical stage where respiratory alterations may lead to hypoxiaassociated syndromes that endanger brain development. In terms of psychomotor activity, animals exposed to 2.0 g/kg of EtOH at PD 9 showed heightened duration and frequency of grooming. In the second study animals exposed at least one time to EtOH exhibited lower pH and higher CO₂ than animals that were never exposed to EtOH. This results suggest metabolic acidosis probably due to EtOH-related hypercapnia during a vulnerable stage in development relative to SIDS.