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Differential effects of EtOH pre-exposure or acute intoxication on hypoxic ventilatory response, medullary serotonin levels and metabolic parameters in neonate rats

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Acute ethanol-EtOH intoxication during the neonatal period reduces the hypoxic ventilatory response (HVR) and alters serotonin (5-HT) levels in the medullary 5-HT system. Yet, the impact of EtOH pre-exposure (pre-EtOH) on acute intoxication remains unclear. We examined how pre-EtOH and acute EtOH interact to affect HVR, medullary 5-HT, and metabolic responses. Pups were pre-exposed to 2.0 or 0.0 g/kg EtOH (ig) on postnatal days (PD) 3, 5, and 7. On PD9, pups received acute EtOH or vehicle and then were subjected to intermittent (IH) or continuous (CH) hypoxia. Brainstem and trunk blood were collected for 5-HT and metabolic analysis. Acute EtOH reduced HVR in both IH and CH, regardless of pre-EtOH. Apneas were fewer in IH than CH, though EtOH reduced apneas under all conditions. All EtOH exposures decreased 5-HT levels, but significantly in pre-EtOH sober pups. Hypoxia induced alkalosis and HVR-driven hypocapnia, but acute EtOH impaired this response inducing acidosis–hypercapnia -exacerbated in pre-EtOH pups under normoxia (sensitization). Hypoxia also enhanced oxygenation, but acute EtOH attenuated this effect. Notably, pre-EtOH pups under IH showed an adaptive profile, like tolerance. These findings reveal both EtOH exposures disrupt neonatal respiratory function: acute EtOH impairs HVR and metabolic compensation, while pre-EtOH alters 5-HT and metabolic parameters, promoting plasticity processes (sensitization and tolerance) to acute intoxication.