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Double Trouble: Adolescent alcohol exposure in pre and postnatal oxycodone offspring

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Increased risk of oxycodone (oxy) dependency during pregnancy has been linked to maladaptive behaviors and cognitive deficits in exposed offspring. Adding a further layer of complexity is the risk of these offspring to abuse alcohol during adolescence. Currently, there are no studies that have documented associated changes in CNS including molecular, synaptic and behavior outcomes associated with adolescent alcohol exposure in in utero oxy (IUO) and post-natal oxy (PNO) exposed offspring. Our current study aims to fill this important knowledge gap by delineating the underlying mechanisms that further exacerbate CNS and peripheral outcomes in these “two-hit” offspring. Using a preclinical rodent model mimicking, we simulated adolescent binge drinking by treating P28 old animals to a binge equivalent of ethanol or an isovolumetric amount of water from P28-P48, every 48 hours. IUO and PNO animals were consistently lighter than saline controls in both the water and alcohol groups. Additionally, IUO+EtOH rats had a higher blood alcohol concentration than PNO+EtOH and saline+EtOH animals that corroborated with increased inflammation in the livers. Current ongoing studies are focused in understanding if observed peripheral inflammation also leads to higher inflammation in the CNS including decoding synaptic alterations and behavioral deficits in these animals during adulthood.