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Moderate prenatal alcohol exposure alters protein expression of stress and immune factors in the amygdala following early life stress.

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Abstract
Fetal alcohol spectrum disorder (FASD) is the outcome of prenatal alcohol exposure (PAE) that results in cognitive, mood and neuroimmune disorders. PAE negatively impacts the brain stress-response system by dysregulating the expression of stress neuropeptides such as corticotropin releasing hormone (CRH), and neuroimmune factors including interleukin-1beta (IL-1β). Both CRH and IL-1β are suspected to play a role in chronic anxiety and depression. In animal models, studies have characterized the key brain region, the amygdala, in regulating stress and emotion such as anxiety and depression. Prior research demonstrates that stress enhances amygdala activity and alters the expression of neuroimmune factors. However, it is not well understood whether PAE impacts stress responses and neuroimmune factors in the amygdala. Critical brain development continues after birth in mice for several weeks, an early postnatal period in which neurons mature while immune responses are suppressed to prevent disruption of brain maturation. We hypothesized that PAE alters protein levels of CRH and IL-1β in the amygdala following an early life stressor. In this study, pregnant dams voluntarily consumed solutions of saccharine-sweetened 10% alcohol, saccharine-sweetened water, or simply water for 4 hrs/day during gestation. Female and male offspring at postnatal day 10 (PND10) were exposed to maternal separation for 4 hrs. as a stressor, while control mice were not separated. The amygdala was collected immediately thereafter. Results indicate sex differences were observed in the amygdala from PAE but not saccharine control mice. Elevated protein levels of CRH and IL-1β were observed in male offspring independent of stressor exposure. These findings suggest PAE is a risk factor for altered amygdala functioning in early life, which may underlie anxiety and depression in later life. Identifying aberrant factors in key brain regions responsible for regulating mood may eventually elucidate early-life interventions for those individuals with FASD.