

Repeated binge-like ethanol administration during adolescence cause decreased c-fos immunoreactivity in amygdala and arcuate nucleus in adult sprague-dawley rats [La administración repetida de etanol, tipo atracón, durante la adolescencia provoca descenso en la expresión de c-fos en la amígdala y núcleo arqueado de ratas sprague-dawley adultas]

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Binge alcohol drinking during adolescence has been associated with neurotoxicity and increased risk for the development of alcohol use disorders. There is evidence that acute and chronic ethanol administration alters c-fos expression, an indirect index of cellular activity, in different brain regions in adult rats. We evaluate here if a binge-like pattern of ethanol exposure during adolescence has a relevant impact on basal and/or ethanol-stimulated regional c-fos activity during adulthood. For that aim, Sprague- Dawley rats PND 25 were saline pre-treated, (SP group) or binge-ethanol pre-treated (BEP group) for two-consecutive days, at 48-h intervals, over a 14-day period (PND 25 to PND 38). At adult stage (PND 63) and following 25 ethanol-free days, we evaluated c-fos immunoreactivity in response to saline or acute ethanol (1.5 or 3.0 g/kg) in the hypothalamus and amygdala. We found that acute ethanol administration dose-dependently increased c-fos activity in the the Paraventricular nucleus of the hypothalamus (PVN). Interestingly, binge-ethanol exposure during adolescence significantly reduced basal c-fos activity during adulthood in the Central nucleus of the amygdala (CeA) and the Arcuate nucleus of hypothalamus (Arc). We conclude that binge-like ethanol administration during adolescence causes long-term disturbances in basal neural activity in brain areas critically involved with ethanol consumption.

Adolescent

Arcuate nucleus of hypothalamus (Arc)

Binge-like ethanol administration

C-fos

Central nucleus of the amygdala (CeA)

Immunoreactivity

Paraventricular nucleus of the hypothalamus (PVN)

Rats