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The hippocampus is susceptible to the effects of prenatal alcohol exposure (PAE) which can result in persistent cognitive impairments. Although there have been many investigations, deficits in synaptic plasticity remain poorly understood. Previous studies have identified deficits in long-term potentiation (LTP) in the perforant pathway(PP) to dentate gyrus synapses as a robust consequence of PAE. Currently, there are no known, clinically effective pharmacotherapeutic interventions for these deficits. This study sought to investigate the effects of PAE on the pre-synaptic mechanisms involved in LTP. We first investigated differences in extracellular glutamate levels following stimulation of the PP (n=20/PAE = 10). In vivo electrophysiology was conducted along with glutamate biosensor implants in the DG. The fractional amplitude measure revealed less change in the PAE group compared to controls ($p = 0.033$). These observations suggest that deficits in LTP following PAE may be related to alterations in the presynaptic mechanisms associated with LTP. A second experiment examined the effects of two doses of an H3R inverse agonist, SAR152954, on PAE-induced LTP deficits in the dentate gyrus, as a possible agent to reverse those deficits. Rats were given a single injection of SAR152954 (0.1 or 1.0 mg/kg) or saline (vehicle) 30 min prior to the recording session. Saline-treated PAE rats displayed reduced LTP relative to controls ($p = 0.05$). Control rats receiving 0.1 mg/kg SAR152954 displayed decreased LTP relative to VEH. PAE rats receiving 0.1 mg/kg SAR152954 did not show a reversal of PAE-induced deficits ($p > 0.8$). However, the 1.0 mg/kg dose reversed PAE-induced deficits to levels comparable to the control animals ($p > 0.7$) and greater than PAE animals that received either the vehicle or low dose SAR152954 conditions ($p = 0.02$). Together these experiments suggest deficits in the presynaptic mechanisms of LTP and offer a possible pharmacological intervention for those deficits.