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Neonatal serotonin (5-HT) depletion does not disrupt prepulse inhibition of the startle response in rats

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Abstract:
The neurodevelopmental hypothesis of many brain disorders is based on the notion that environmental factors have significant effects on brain maturation. Because serotonin (5-HT) dysfunction in development may be involved in disease etiology, the present investigation assessed the effects of neonatal 5-HT depletion on prepulse inhibition of the startle response (PPI) in rats. Three-day-old Sprague-Dawley rats were pretreated with desipramine (20 mg/kg), followed by an intraventricular injection of the selective 5-HT neurotoxin 5,7-dihydroxytryptamine (5,7-DHT, 70 μg dissolved in 2 μl of 0.1% saline solution in ascorbic acid) on each side. Three months later, the rats' PPI was tested. Despite a severe and permanent decrease (80–100%) in hippocampal, prefrontal and striatal 5-HT levels, treatment with 5,7-DHT caused no disruption of PPI. In contrast to this lack of effect, the 5,7-DHT treatment increased basal startle activity, as measured in response to a 120 dB stimulus. Thus, our results clearly indicate that neonatal 5-HT depletion does not interrupt prepulse inhibition in rats. Studies involving lesions of brain structures or chemical systems run the risk of inducing compensatory changes in brain function, resulting in an amelioration of any deficit. The development of such compensatory mechanisms seems likely in the current study, due to the severe and long-lasting effect of neonatal 5,7-DHT-induced reduction on 5-HT levels.

Key words:
prepulse inhibition, neonatal serotonin depletion, 5,7-DHT, rats