Cadmium exposure in adolescence through adulthood impairs adult hippocampal neurogenesis and cognition in mice

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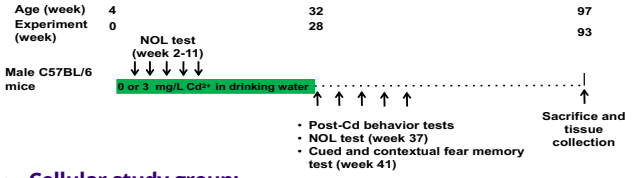
INTRODUCTION

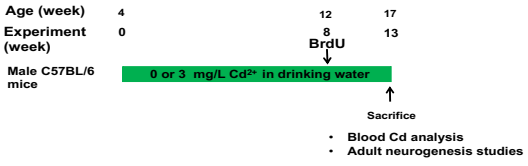
Cadmium (Cd) is a toxic heavy metal of big public health concern. Because of its long half-life in humans, Cd accumulates in body and induces adverse effects in various organs. Cd is suggested to be a neurotoxicant, but the full spectrum and underlying mechanisms of its neurotoxicity are not fully understood. Adolescence represents a critical period of transition to independence during which significant lifestyle changes occur. The rapidly developing immature nervous system during adolescence is especially vulnerable to most toxicants[1]. Studies found that hippocampal neurogenesis, a process that can regulate cognition, is significantly higher during adolescence than adulthood in rodents[2-4]. However, no study to date has investigated the effects of in vivo Cd exposure, starting from juvenile to maturity, on hippocampal neurogenesis and hippocampus-dependent learning and memory Thus, the goal of this study was to characterize the effects of Cd exposure in adolescence through adulthood on cognitive function and hippocampal neurogenesis in mice, and further to determine the critical window for Cd-induced effects on cognition and hippocampal neurogenesis.

METHODS

- **Behavior test group:**

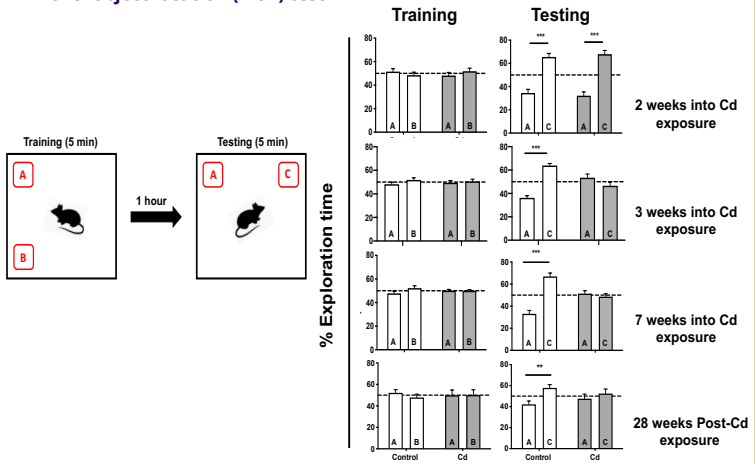


- **Cellular study group:**

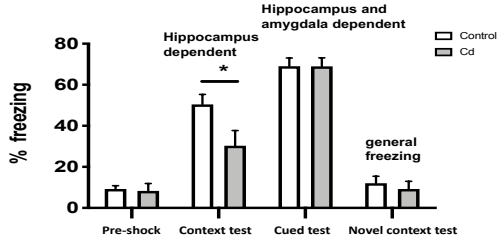


RESULTS

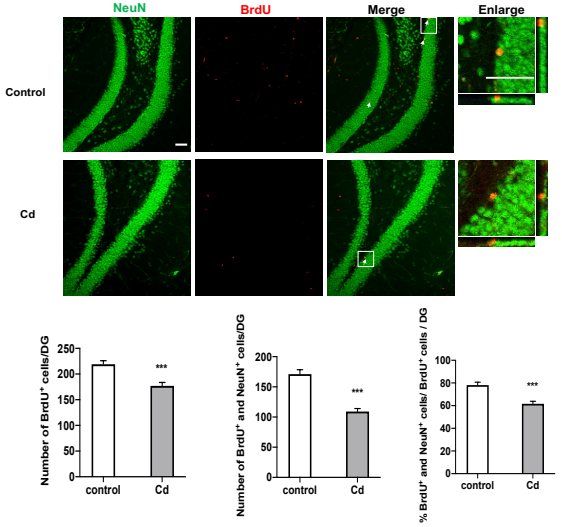
Adolescence through adulthood Cd exposure causes persistent impairment of hippocampus-dependent spatial memory in the novel object location (NOL) test



Adolescence through adulthood Cd exposure causes persistent impairment of hippocampus-dependent contextual fear



Adolescence through adulthood Cd exposure impaired adult hippocampal neurogenesis in mice



CONCLUSION

- **Adolescence through adulthood Cd exposure induced persistent impairments of cognitive functions in mice.**
- **Adolescence through adulthood Cd exposure impaired adult hippocampal neurogenesis in mice.**

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