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Effects of medial prefrontal cortex NMDA NR-1 subunit deletion in adult mice on spatial reference and working memory

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**BACKGROUND**

- Glutamate N-methyl-D-aspartate (NMDA) receptor dysfunction may contribute to cognitive deficits associated with psychiatric illnesses including autism, affective disorders, and schizophrenia.
- The medial prefrontal cortex (mPFC) has been shown to play a role in executive functions including working memory (Kesner & Churchwell, 2011).
- Results of a recent study indicate that early postnatal NMDA NR1 deletion, mostly confined to excitatory neurons in prefrontal and sensory cortices, fails to affect spatial working memory (SWM) as assessed using a spontaneous alternation Y-maze task (Rompala et al., 2013).
- The present study examines the effects of NR1 deletion restricted to the adult mouse mPFC on a more cognitively demanding spatial reference memory (SRM) and SWM 6-arm radial maze (as described in Niewoehner et al., 2007).

**MATERIALS AND METHODS**

**Fig 1.** AAV-Cre or LacZ/aCSF was microinjected into the infralimbic mPFC of adult male floxed NR1 mice (fNR1; JAX, Grin1tm; PN70-90).

**Fig 2.** In situ hybridization analysis revealed loss of NR1 mRNA in the mPFC of Cre-treated mice but not control mice.

**Fig 3.** Mice were habituated and trained on an automated 8-arm radial maze (Med Associates Inc.) adjusted to a 6-arm configuration. Noldus Ethovision XP was used for tracking and data collection. Extra-maze cues were provided to facilitate the use of spatial memory to complete the task.

**REFERENCES**


**DISCUSSION**

- A regionally specific deletion of the NMDA NR1 subunit, induced by local infusion of AAV-Cre into the adult mouse infralimbic mPFC, did not affect acquisition of SRM or SWM, as assessed in a 6-arm radial maze task.
- These results extend earlier demonstrations that SWM assessed using a spontaneous alteration Y-maze task is unaffected by early postnatal NR1 deletions in excitatory neurons of the prefrontal and sensory cortices (Rompala et al., 2013).
- In the present study, increasing cognitive demand by rotating extramaze cues revealed potentiated SRM and SWM deficits following NR1 deletions in the infralimbic mPFC.
- The current data suggest that NR1 deletion in the infralimbic mPFC impairs the ability to modify behavior in the presence of changes in the environment.
- This deficit may reflect an inability for Cre mice to inhibit behavior, as the same mice displayed an increase in premature responding in a 5-choice serial reaction time task (Manning et al., 2014).