Adolescent mouse hippocampal function was impaired by prenatal nicotine exposure

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It is not clear how prenatal nicotine exposure (PNE) may cause cognitive impairment in offspring. In this study, we investigated whether the exposure to nicotine (0.2 mg/mL in drinking water with 2 % sucrose) during E14-P0 impaired hippocampus-dependent learning and memory in adolescence. In the hippocampal CA1 region, the induction and maintenance of N-methyl-D-aspartate (NMDA) receptor dependent long-term potentiation (LTP) was diminished by PNE, whereas the paired-pulse facilitation was not affected. Behaviorally, PNE impaired contextual- but not tone-dependent fear memory in 7- to 8-week-old mice. Both impairments were attenuated by the repeated co-treatment with methyllycaconitine (5 mg/kg s.c.), a nicotinic alpha 7 receptor antagonist. The results suggest that the nicotinic alpha 7 receptor dependent plasticity during embryonic period may be required for NMDA receptor-related long-term memory formation, and that PNE may disrupt this form of plasticity.