CHRONIC COCAINE EXPOSURE DURING ADOLESCENCE IN MICE INDUCES ALTERATIONS IN HIPPOCAMPAL NEUROGENESIS AND SPATIAL MEMORY

Psychopharmacology & addiction

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Objetivos / Objectives

Cocaine is the second most widely consumed illicit drug even at early ages, with a prevalence of 2.9% in Spain in the 14-18 age group. Chronic consumption producesseveral cognitive-behavioral and hippocampal alterations, which can be more dangerous if it begins in adolescence, a sensitive period in brain development. The objective of this work is to study the hippocampal and behavioral consequences of cocaine exposure during adolescence.

Metodología / Methodology

In a first experiment, adolescent male C57BL/6J mice (PND30) received intraperitoneal injections of cocaine (20 mg/kg): one group for 7 consecutive days andanother group for 21 days, both with a control group injected with saline (N=24). On the last day of administration, all mice received bromodeoxyuridine (BrdU) (100 mg/kg,i.p.). One day later, mice were sacrificed, and brain tissue was processed by immunohistochemical procedures to assess adult hippocampal neurogenesis (AHN) (BrdU, DCX and p-H3 markers) and neuroinflammation (Iba1). In a second experiment, a novel place recognition (NPR) test was performed with 21-day administration conditions (N=28).

Resultados y Conclusiones / Results & Conclusions

Cocaine administration for 21 days down-regulates AHN and up-regulates Iba1 expression when the administration was for 7 days. Interestingly, mice that received cocaine for 21 days displayed impairments in the NPR test. Cocaine administration during adolescence causes a deleterious effect on the hippocampus that could be directly related to difficulties in performing spatial memory tasks. These results may beuseful in designing new therapeutic approaches to mitigate the effect of cocaine on developing brains.

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