Stress rapidly reorganizes the glutamate synapse in the prefrontal cortex of cocaine-withdrawn adolescent rats

1. INTRODUCTION

Drug addiction is a major public health issue worldwide characterized by a compulsive drug-seeking and drug-taking behavior. Illicit use of drugs begins and escalates during adolescence, with long-term adverse consequences. The progression from first use to addiction appears to be shorter during adolescence (Clark et al., 1998). The adolescent brain is in a unique state of transition as it undergoes structural and synaptic changes (Crews and Hodge, 2007). The evidence that initiation of drug taking primarily occurs during adolescence suggests a greater addictive potential than at adulthood. The brake against compulsive behaviors is provided by prefrontal cortex that is still developing during adolescence. It is thus possible to hypothesize that if the brake is defective, the chances of risky behaviours become higher. For this reason, adolescence can be considered a crucial period for investigating the development of drug addiction.

The aim of the present work was 1) to investigate the short-term effects of repeated exposure to cocaine during adolescence on the glutamatergic synapse in prefrontal cortex and 2) to evaluate the dynamic response to a challenging event such as an acute stress as a potential indication of coping ability under a challenging condition.

2. MATERIALS AND METHODS

3. RESULTS

GLUTAMATERGIC TRANSMISSION

4. CONCLUSIONS

Stress rapidly reorganizes the glutamate synapse in the prefrontal cortex of cocaine-withdrawn adolescent rats.

Hyper-reactive glutamatergic synapses in mPFC may contribute to explain the hypersensitivity to stress observed in abstinent cocaine users.

Dysregulation of the glutamate homeostasis may contribute to the negative emotional state and stress-induced reinstatement observed in animal models of cocaine abuse.

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