UFR2709, an Antagonist of Nicotinic Acetylcholine Receptors, Delays the Acquisition and Reduces Long-Term Ethanol Intake in Alcohol-Preferring UChB Bibulous Rats

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Abstract

Alcoholism is a worldwide public health problem with high economic cost and which affects health and social behavior. It is estimated that alcoholism kills 3 million people globally, while in Chile it is responsible for around 9 thousand deaths per year. Nicotinic acetylcholine receptors (nAChRs) are ligand-gated ion channels expressed in the central nervous system, and they were suggested to modulate the ethanol mechanism involved in abuse and dependence. Previous work demonstrated a short-term treatment with UFR2709, a nAChRs antagonist, which reduced ethanol intake using a two-bottle free-choice paradigm in University of Chile bibulous (UChB) rats. Here, we present evidence of the UFR2709 efficacy in reducing the acquisition and long-term ethanol consumption. Our results show that UFR2709 (2.5 mg/kg i.p.) reduces the seek behavior and ethanol intake, even when the drug administration was stopped, and induced a reduction in the overall ethanol intake by around 55%. Using naïve UChB bibulous rats, we demonstrate that UFR2709 could delay and reduce the genetically adaptive impulse to seek and drink ethanol and prevent its excessive intake.

Author keywords

nicotinic antagonist; UChB rats; UFR2709; voluntary ethanol intake