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PHARMACOLOGICAL DISRUPTION OF ALCOHOL-RELATED MEMORIES - THERAPEUTIC IMPACT OF THE THEORY OF MEMORY RECONSOLIDATION

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Long-lasting memories that associate environmental stimuli with the effects of alcohol are known to be a main cause of relapse and are a major challenge in the treatment of alcohol addiction. It is reasonable to hypothesize that disrupting consolidated alcohol-related memories might help to prevent relapses. The reconsolidation theory states that a consolidated memory could again become labile and susceptible to disruption by protein synthesis inhibition or NMDA-antagonism after memory retrieval. This has been shown for cocaine- and morphine-associated memories in several recent studies. The aim of our investigations was to examine in an animal model for cue-induced relapse to alcohol-seeking behavior whether the behavioral impact of previously conditioned alcohol associated cues is significantly reduced by blocking the reconsolidation of learned alcohol associations. We show that reconsolidation of alcohol memories is disrupted by post-retrieval ICV-administration of the protein synthesis inhibitor anisomycin. Similarly, post-retrieval i.p.-administration of the NMDA antagonist MK-801 reduced alcohol seeking behavior during the following test day as compared to vehicle treated rats. Pharmacological disruption of reconsolidation of alcohol-associated memories may thus provide a potential therapeutic strategy for the prevention of relapse in alcohol addiction.

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