Involvement of Amygdala on Place Aversion Induced by Naloxone in Single-Dose Morphine Treated Rats

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The neurobiological mechanism underlying the negative motivational component of withdrawal from acute opiate dependence is not well understood. It will benefit the development of appropriate therapies to facilitate opiate abstinence and reduce craving to better understand the mechanisms underlying acute opiate dependence and to determine whether there are dissociation and similarity between the early and fully developed stages of dependence.

In the present study, we examined the influence of c-Fos expression in amygdala in acquisition of conditioned place aversion (CPA) induced by naloxone-precipitated withdrawal from a single morphine exposure 24h before. The effect of microinjection into the central amygdaloid nucleus (CeA) of various kinds of glutamatergic neurotransmission inhibitors was also investigated.

CeA displayed significant increase in c-Fos expression in acquisition of CPA. Furthermore, CPA was attenuated significantly and dose dependently by microinjection into CeA of all glutamatergic neurotransmission inhibitors (NMDA receptor antagonist (+)-5-methyl-10,11-dihydro-5H-dibenzo[*a,d*]cyclo-hepten-5,10-imine maleate (MK-801), AMPA receptor antagonist 1-(4-aminophenyl)4-methyl-7,8-methylenedioxy-5H-2,3-benzodiazepine hydrochloride (GYKI52466), metabotropic glutamate receptor antagonist (±)-α-methyl-4-carboxyphenylglycine (MCPG), and glutamate release inhibitor riluzole).

These results suggest that CeA involves the acquisition of CPA induced by naloxone-precipitated withdrawal from a single morphine exposure, and the function of glutamatergic system projected from amygdala to nucleus accumbens play a facilitative role in formation of morphine dependence.