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Poster presentation

Agmatine synthesis in morphine dependence

Feyza Aricioglu*[1] and Soundar Regunathan[2]

Address: [1]Marmara University, Department of Pharmacology, Turkey and [2]University of Mississippi, USA
* Corresponding author

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Background

Agmatine is an endogenous amine derived from the decarboxylation of arginine decarboxylase. It has been found in various mammalian organs and is thought to act as a neurotransmitter or neuromodulatory agent. When exogenously administered to rodents, agmatine enhances morphine analgesia, blocks tolerance to opioids and attenuates withdrawal syndrome in morphine.

Materials and methods

Rats were implanted with two morphine pellets (75 mg each) or control placebo pellets. After 24 or 48 hours later animals were sacrificed and brain regions were disected. Arginine Decarboxylase (ADC) activiy was measured by using radioimunoassay.

Results

ADC activity was found in ventral tegmental area, hypothalamus, cortex, nucleus accumbens, substantia nigra, locus coeruleus, medulla ans cerebellum, in order of highest to lowest activity. Exposure of rats to morphine for 24 hours significantly increased ADC activity in hypothalamus, cortex, locus coeruleus, nucleus accumbens and cerebellum, compared to placebo control rats. The change in ventral tegmental area, medulla and substantia nigra were not significantly different. When ADC activity was measured after 72 hours, activity was significantly reduced in all regions that showed an increase after 24 hours.

Discussion

The biosythesis of agmatine could be increased in spesific brain regions during the early phase of morphine exposure as a protective mechanism against dependence and that increasing endogenous agmatine may be beneficial in opiate drug abuse.