Poster Abstracts

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Sub-Topics by Disorder:

Addictive Disorders: PM283 – PM333
Childhood & Adolescent Disorders: PM334 – PM357
Schizophrenia: PM358 – PM543

ADDICTIVE DISORDERS: PM283 – PM333

PM283

Agmatine prevents morphine-induced glial activation in rats

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Abstract
Objectives: Opioid addiction is associated with long-term adaptive changes in the brain that involve glial activation. Agmatine is an amine formed by the decarboxylation of l-arginine by the enzyme arginine decarboxylase. It binds to α2-adrenergic and imidazoline receptors, and selectively blocks N-methyl-D-aspartate receptors. Agmatine treatment was reported to have various biological actions. It reduces tolerance to morphine and attenuates behavioral signs of morphine in naloxone-induced abstinence syndrome in vitro and in. This study has been designed to evaluate the effect of agmatine on morphine-induced glial activation in rats.
Methods: Male Wistar Albino rats (190–240 g) were divided into three groups such as Control, Morphine and Morphine+Agmatine. Morphine and Morphine+Agmatine groups were received two morphine pellets containing 75 mg morphine base each that was implanted subcutaneously in the scapular area under light anesthesia. Morphine+Agmatine group received agmatine (40 mg/kg, i.p.) and the injection was repeated after 6 hours. 72 hours later naloxone 2 mg/kg i.p was injected to precipitate withdrawal syndrome, observed for 30 minutes and withdrawal symptoms were recorded. Control group received placebo pellets and only saline injections in the same volume of agmatine. Immunohistochemistry was performed to investigate the expression of glial fibrillary acidic protein (GFAP) as indicator of glial activity and c-fos.
Results: Morphine withdrawal syndrome became more severe as it is repeated. When coadministered with morphine, agmatine significantly attenuated withdrawal symptoms. Immunohistochemistry with GFAP revealed that agmatine significantly decreased morphine-induced over-expression of GFAP and c-fos.
Conclusions: Exogenously applied agmatine can prevent morphine-withdrawal-induced glial reactivity and c-fos. As an endogenous molecule agmatine might be a part of compensatory mechanism within the reward pathway. Therefore further studies are required for better understanding the involving mechanisms in order to develop novel approaches for the morphine addiction strategies.

PM284

Agmatine prevents learning and memory deficit in repeated morphine withdrawal: Is it via glutamatergic system?

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Abstract
Objective: Morphine is important not only because it's known as a most powerful analgesic but also with its potential for addiction. Withdrawal syndrome which occurs with an absence of morphine is a condition that addicts suffer over and over lifelong. We have previously showed that repeated withdrawal syndrome impaired learning-memory. Current study designed to evaluate possible effect of agmatine treatment on impaired learning-memory and if it is related with glutamatergic system.
Material and Method: Wistar Albino rats were divided into groups such as Control, Morphine and Agmatine+Morphine (*n*=8 for each group) and subcutaneously implanted with two pellets, 75 mg morphine base containing/each. 72 hours later naloxone 2 mg/kg i.p was injected to precipitate withdrawal syndrome, observed for 30 minutes. This procedure was repeated by implanting one more pellet each time, repeated 6 times and waited for 3 weeks. Animals received 40 mg/kg agmatine twice daily. Animal were undertaken for Morris water maze and passive avoidance tests. Brains were used to evaluate glutamate immunoreactivity.
Results: Morphine withdrawal syndrome became more severe each time and learning-memory functions were impaired by repeating the syndrome. Agmatine treatment prevented learning-memory deficits in Morris water maze and passive avoidance tests. In hematoxylin-eosin stained slices, dark eosinophilic