عنوان مقاله:

INTERACTION BETWEEN VENTRAL HIPPOCAMPAL GLUTAMATERGIC AND MEDIODORSAL THALAMIC DOPAMINERGIC SYSTEMS IN NICOTINE-INDUCED ANXIOGENIC-LIKE BEHAVIOURS

محل انتشار:

سیزدهمین کنگره بین المللی دانش اعتیاد (سال: 1398)

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خلاصه مقاله:

Background and Aim : Nicotine, contained in tobacco, is one of the most abused drugs which mediate long-lasting alterations in the mesolimbic glutamatergic and dopaminergic rewarding pathways. Furthermore, co-morbidity of tobacco smoking and anxiety disorder has been demonstrated in many literatures. Although, both nicotinic acetylcholine and dopamine receptors are implicated in the behavioural abnormality relevant to anxiety, their interactions in different neural substrate in the mediating anxiety-related behavior has remained elusive. Considering the important role of the mediodorsal thalamus (MD) in emotional behaviors, the involvement of MD dopamine receptors following nicotine exposure in modulating anxiety-like behaviors was investigated in an animal model. Moreover, the blockade of ventral hippocampal (VH) NMDA receptors was used to assess the effect of MD dopaminergic activation on nicotine-induced anxiety. Methods : Male Wistar rats were simultaneously cannulated unilaterally in the MD and the VH with a stereotaxic apparatus. Anxiety behaviours were measured using elevated plus-maze (EPM). The EPM is used as a valuable paradigm to define brain regions which are involved in anxiety and screen pharmacological agents for anxiety treatment. Results : Acute administration of nicotine (0.4-0.6 mg/kg, i.p.) dose-dependently decreased the percentage of open arm time (%OAT), open arm entries (%OAE) as measured by EPM indicating an anxiogenic-like behaviour to nicotine. No changes in locomotor activity were observed at given doses of nicotine. Furthermore, we found that microinjection of apomorphine, a non-selective dopamine receptor agonist, (0.1-0.3 µg/rat) into MD by itself had no effect on the EPM parameters. Interestingly, intra-MD microinjection of the same doses of apomorphine suppressed the anxiogenic-like behaviours induced by nicotine (0.5 mg/kg, i.p.), suggesting a cross-talk between nicotinic and dopaminergic receptors in the anxiety regulation. Intra-VH microinjection of D-AP5, a selective NMDA receptor antagonist (0.3-0.5 µg/rat), reversed the inhibitory effect of apomorphine on nicotine-induced anxiogenic-like behaviours. Conclusion : Taken together, it can be concluded that anxiogenic-like behaviours observed following nicotine administration are partly mediated by mediodorsal thalamus dopamine receptors. The functional interaction between the VH NMDA receptors and the MD dopamine receptors may be involved in the anxiogenic-like effect of nicotine

کلمات کلیدی:
Mediodorsal thalamus; Ventral hippocampus; anxiety; Nicotine; Dopamine and NMDA receptors