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Role of the locus coeruleus in the sleep rebound following two different sleep deprivation methods in the rat.

González MM[1], Valatx JL, Debilly G.

Abstract

The aim of the present study was to assess the involvement of the locus coeruleus in the paradoxical sleep rebound following sleep deprivation in the rat. Animals were sleep-deprived for 10 h before, and after, specific N-(2-chloroethyl)-N-ethyl-2-bromobenzylamine (DSP-4) lesioning of the noradrenergic-locus coeruleus system. Sleep deprivation was produced using either an instrumental (water tank) or pharmacological (methylamphetamine) method. After lesioning, the rats submitted to the instrumental method showed a significant decrease in the paradoxical and slow-wave sleep rebounds (-54% and -78%, respectively), while animals receiving metamphetamine did not. Our results suggest that the noradrenergic system of the locus coeruleus is a relevant component of the sleep rebound mechanisms. However, the extent of involvement is dependent on the sleep deprivation method used.

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Behav Pharmacol. 1998 Dec;9(8):655-62.

Involvement of stress in the sleep rebound mechanism induced by sleep deprivation in the rat: use of alpha-helical CRH (9-41).

González MM[1], Valatx JL.

Abstract

A previous study demonstrated the efficacy of the corticotropin-releasing hormone (CRH) receptor antagonist, alpha-helical CRH (9-41), in blocking the paradoxical sleep increase induced by stress. In the present study, this peptide was used to evaluate the involvement of the stress component of the sleep deprivation, in the paradoxical sleep rebound. Rats were subjected for 10 h to the classical water-tank sleep-deprivation technique and were given, every 2 h throughout the sleep deprivation period, intracerebroventricular injections of either 100 microg/5 microl of alpha-helical CRH (9-41) or vehicle alone. Continuous recordings showed that antagonist treatment decreased the PS rebound, but not the SWS rebound, following sleep deprivation. These findings suggest that, in the water-tank sleep deprivation method, stress, acting via CRH activation, is the main factor inducing the paradoxical sleep rebound.

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Noradrenaline neurotoxin DSP-4 effects on sleep and brain temperature in the rat.

González MM[1], Debilly G, Valatx JL.

Abstract

N-(2-chloroethyl)-N-ethyl-2-bromobenzylamine (DSP-4) has a selective degenerative effect on noradrenergic fibers originating from locus coeruleus (LC) neurons. In the present study, we studied its effect on vigilance states and brain temperature by continuous recordings for periods of 1-5 days and 2-4 weeks following DSP-4 treatment. On the first day, paradoxical sleep duration was significantly decreased (-67%, $P < 0.05$), slow-wave sleep (SWS) duration increased (+16%, $P < 0.05$) up to 48 h after DSP-4 treatment (+8%, $P < 0.05$) and the wake period decreased (-8%, $P < 0.05$). The vigilance states returned to control values 4-5 days later. The brain temperature was decreased during the first night (-2 degrees C) and then recovered the control values. Two and 4 weeks after DSP-4 treatment, paradoxical sleep was still decreased (-18% and -23%, respectively, $P < 0.05$), while SWS was significantly increased only at night during the fourth week (+23%, $P < 0.05$). These results therefore provide evidence for a differential involvement of the noradrenergic LC system in sleep mechanisms depending on the light-dark cycle. Different hypotheses are proposed.

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