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Poster Sessions : Learning and Long-term Memory 1 (P1-j11)
2012/09/18 13:00-14:00

Noradrenergic system is involved in exploratory behavior in decision-making via medial prefrontal cortex

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Electrophysiological and computational studies have suggested that noradrenergic system originating from locus coeruleus (LC) is crucial in assessment of utility of options required in optimal decision-making. It has been proposed that higher discharge activity of LC-noradrenaline neurons should enhance exploratory state to assess the utility of options and to search for an optimal choice in uncertain situation. However, few study empirically examine neural mechanisms of the noradrenergic regulation of exploratory state in decision-making. Therefore, we examined the effects of pharmacological inhibition of noradrenergic system on exploratory state in T-maze decision-making task in rats. In the task, an advantage arm has 3 pellets and disadvantage arm has 1 pellet. To evaluate exploratory state, we recorded vicarious trial-and-error behavior (VTE), which is thought to be attributed to medial prefrontal cortex (mPFC). Noradrenergic system was inhibited with clonidine, α2 noradrenergic receptor agonist. In the first experiment, clonidine (20μg/kg or 50μg/kg) was intraperioneally injected to examine involvement of noradrenergic system for decision-making. In the second experiment, clonidine (10nM/0.5μl) was locally injected into mPFC to examine whether the brain region involved in noradrenergic modulation of decision-making. Experiment 1 showed that clonidine injection impaired the task and decreased VTE. Experiment 2 showed that clonidine injection into mPFC induced impairment of the task and decrease of VTE similar to results of Experiment 1. These data suggest that noradrenergic system can regulate the exploratory state via mPFC, thereby optimize decision-making.

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Poster Sessions : Motivation and Emotion (P2-i07)
2012/09/19 13:00-14:00

Yawning response induced by chemical stimulation of central nucleus of amygdala in rats

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Previously we reported that a stereotyped yawning response, which was characterized by an initial depressor response and an arousal shift in electrocorticogram (ECoG) followed by a single large inspiration with mouth opening, can be evoked by several forms of chemical stimulation of paraventricular nucleus (PVN) of hypothalamus in anesthetized, spontaneously breathing rats. In addition, we have shown that the yawning response is mediated by activation of oxytocin (OT) neurons and corticotropin-releasing factor (CRF) neurons in the PVN. Yawning is often observed in not only the states of boredom and drowsiness but also a stressful situation, in which yawning is also accompanied with anxiety-like behaviors such as freezing, grooming and scratching. This indicates that yawning might be an emotional behavior. Since emotional behavior is known to be mediated by activation of amygdala neurons, it is possible that yawning behavior be meditated by activation of amygdala neurons. We investigated the effect of chemical stimulation of the central nucleus of amygdala (CeA) on yawning responses in anesthetized, spontaneously breathing rats. Yawning response was evaluated by monitoring intercostals electromyogram as an index of inspiratory activity. We also recorded blood pressure and the ECoG to evaluate autonomic function and arousal responses during yawning. Microinjection of L-glutamate into the CeA elicited a stereotyped yawning response. These physiological aspects were associated with a significant increase in activation of c-Fos positive OT and CRF neurons in the PVN. These results indicate that chemical stimulation of CeA induces stereotyped yawning response, which would be mediated through activation of OT and CRF neurons in the PVN.

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Poster Sessions : Sensori-Motor Plasticity (P4-e26)
2012/09/21 11:00-12:00

Constitutive neuronal activation in the mouse brain with 4 weeks of voluntary running

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Physical exercise can improve neuronal plasticity of the hippocampus. Because neuronal activity is a key to promote neuronal plasticity, we have previously examined changes in hippocampal cerebral blood flow, an index of neuronal activity, in rats during treadmill running. We found that blood flow in the hippocampus increases during running in a tetrodotoxin- and NMDA receptor-dependent manners, demonstrating that exercise elicits neuronal activation in the hippocampus. However, it is still uncertain whether chronic physical exercise leads constitutive neuronal activation in the hippocampus. Furthermore, effects of chronic exercise on other brain regions besides the hippocampus are still poorly understood. The objective of the current study was to identify brain regions where constitutively activated by chronic physical exercise. Male C57BL/6 mice were housed with or without running wheel for 4 weeks. ΔFosB was used as a chronic neuronal activity marker. Immunoreactivity of ΔFosB was quantified in 9 brain regions (CA1, CA3, DG of the hippocampus, motor cortex, somatosensory barrel cortex, piriform cortex, striatum, basolateral amygdara, central amygdala) with imaging software (cellSence, Olympus). Voluntary running significantly increased ΔFosB immunopositive structures in the CA1 (360% vs. control), CA3 (343%), DG (322%), motor cortex (162%), and barrel cortex (220%), but not in striatum and amygdala. Interestingly, ΔFosB immunopositive structures in the piriform cortex was significantly decreased with voluntary running (70.3 % vs. control). These results suggest that chronic exercise extensively elicits neuronal activation within the brain, but the effects are not uniform. Further analysis in other brain regions (cerebellum, brain stem) is ongoing.