increase that induced by the infusion of μ-opioid receptor agonist DAMGO in the NAc were significantly suppressed by acute PF_{11} administration.
Conclusions: The present data indicate that PF_{11} inhibits METH-induced dependence and dopaminergic hyperfunction by regulating GABAergic and opioidergic neuronal system in the NAc of mice. And it is proposed that PF_{11} could be a useful compound for the therapeutic treatment of METH dependence.

PM316

Carbachol attenuates excitatory synaptic transmission in cholinergic neurons of the laterodorsal tegmental nucleus

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Abstract
Cholinergic neurons in the laterodorsal tegmental nucleus (LDT) project to the ventral tegmental area (VTA), and changes in the activity of these neurons are thought to affect the activity of VTA dopamine (DA) neurons. We previously reported that in chronically cocaine-administered rats the activity of LDT cholinergic neurons are increased through induction of plastic changes in synaptic transmission and membrane excitability. However, it remains unclear whether acetylcholine (ACh) acts on the LDT cholinergic neurons themselves and modulates their synaptic transmission, and whether such modulatory effects, if any, are affected by chronic cocaine injection. In the present study, we addressed these issues using patch-clamp recordings in rat brain slices. Bath application of carbachol (CCh) significantly reduced the membrane input resistance and the amplitudes of evoked excitatory postsynaptic currents (eEPSCs) without affecting paired pulse ratios. These reductions were suppressed by pretreatment with scopolamine but not mecamylamine, indicating that the effects of CCh are mediated by muscarinic ACh receptors (mAChRs). Intracellular perfusion with GDPβS suppressed the CCh-induced reduction of input resistance but not eEPSC amplitudes, suggesting that the reduction of eEPSC amplitudes is not mediated by a postsynaptic G-protein signaling. Finally, the effects of CCh on eEPSC amplitudes in the LDT cholinergic neurons were not significantly different between the groups repeatedly treated with cocaine or saline. These findings suggest that ACh attenuates excitatory synaptic transmission in the LDT cholinergic neurons through mAChRs, but not through G-protein signaling, and that this effect of CCh is not affected by chronic cocaine administration.

PM317

Characterization of nicotinic neural activities in rat medial and lateral habenula

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Abstract
Most studies have suggested a role for the medial habenula in nicotine addiction but have not assigned these effects to lateral habenula. To demonstrate the distinguished effect of nicotine on habenula between lateral and medial subnuclei, we performed an extracellular recording on various subregions of habenula with nicotine application. We found that whole region of medial habenula presented an identical nicotine-induced neuronal responses which showed a drastic excitation of medial habenula neurons followed by reduction and re-excitation. However, neuronal fields in lateral habenula presented three types of nicotine responses. One type in lateral habenula showed a dramatic excitation followed by reduction and re-excitation against the nicotine application which is similar to the nicotine-induced response in medial habenula. Another type in lateral habenula presented only a reduction of spontaneous neuronal firings for nicotine without excitation. The other type in lateral habenula showed no notable changes after nicotine application. Taken together, our findings demonstrate that lateral habenula as well as medial habenula contributes to alteration of nicotine-induced neural activity. Because the nicotine addiction has not only rewarding properties by the lateral habenula-rostromedial tegmental nucleus regulating dopamine system projection but also aversive properties by the medial habenulo–interpeduncular tract controlling serotonin system projection, the distributional differences in habenula for nicotine-induced neuronal response would be a fascinating approach to comprehend a brain function for the balance between negative and positive reward induced by nicotine. (*supported by NRF-2013R1A1A1057712 and NRF-2014R1A2A2A04007391*)

PM318

Spicy food preference and the HPA axis reactivity to stress in Korean social drinkers

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Abstract
Objectives: Some reports suggest that if individuals prefer spicy foods, they may be vulnerable to stress; thus, their HPA axis reactivity to stress may be abnormal. We investigated the relationship between HPA axis reactivity to stress and spicy food preference in social drinkers.
Methods: The subjects were 40 social drinkers aged above 18 years. They were exposed to stress as cold pressor test and mathematical calculations. Salivary cortisol level was measured before and after the stress and spicy food preference was measured. The subjects were divided into two groups of those who preferred spicy foods (SP, n = 20) and those who less preferred spicy foods (LP, n = 20).
Results: Repeated measures ANOVA on salivary cortisol concentration revealed a significant group by block interaction. Basal and salivary cortisol levels immediately after stress were significantly higher in SP subjects than those in LP subjects. The salivary cortisol level at 80 min after the stress decreased significantly compared to the basal salivary cortisol level in SP subjects. Salivary cortisol level 20 min after the stress increased significantly compared to the basal salivary cortisol level in LP subjects.
Conclusion: HPA axis reactivity to stress in SP subjects was more sensitive than that in LP subjects. These results suggest that HPA axis reactivity in those who prefer spicy foods may be vulnerable to stress.
Key Words: HPA axis · Stress · Spicy food preference · Vulnerability· Salivary cortisol