Methods: Forty-five right-handed patients with PD who met the diagnostic criteria in Structured Clinical Interview for DSM-V and twenty-two healthy control were examined by means of MRI at 3 Tesla. We used the FreeSurfer software package to create a three-dimensional model of the cortical surface for estimating the cortical thickness. In order to test the effect of the gene polymorphism on the brain cortical thickness, we were evaluated their candidate gene polymorphisms of 5-HTTLPR, HTR1A, COMT, BDNF and RGS. We examined between-group differences in cortical thickness using a 2×2 analysis of covariance (ANCOVA) to control age, gender, and education.
Results: We found scores of cortical thickness of PD is significantly lower than those of HC in temporal pole(p=0.000) and insula(p=0.000). Furthermore, analyses of covariance controlling for age, gender and education showed an interaction effects of the genes polymorphisms-by-panic disorder on paralimbic area. 5-HTTLPR rs25531(p=0.011, Lt, temporal pole; p=0.011, Lt, insula), HTR1A rs6295(p=0.007, Lt, temporal pole; p=0.002; Lt, insula; p=0.031, Lt, rostral anterior cingulate), BDNF rs6265 (p=0.012, Lt, temporal pole; p=0.002; Lt, insula), COMT rs4680 (p=0.000, Lt, temporal pole; p=0.001; Lt, insula), RGS2 rs4606(p=0.009, Lt, temporal pole; p=0.009; Lt, insula; p=0.034).
Key words: *Panic Disorder, 5-HTTLPR Polymorphism, Treatment Response*

PS249

White matter microstructural changes are associated with alcohol use in patients with panic disorder

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Abstract
Objective: A close relationship between panic disorder (PD) and alcohol use disorder (AUD) has been suggested. We aimed to investigate alterations in white matter (WM) volume or integrity in patients with PD comorbid with AUD.
Methods: Forty-nine patients with PD, free of comorbid AUD (PD–AUD), and 20 patients with PD comorbid with AUD (PD+AUD) were investigated. All subjects were assessed using the Panic Disorder Severity Scale, Anxiety Sensitivity Inventory-Revised (ASI-R), Beck Depression Inventory, and CAGE questionnaire. Voxel-based morphometry and tract-based spatial statistics were used for imaging analysis.
Results: Increased fractional anisotropy (FA), as well as decreased mean diffusivity and radial diffusivity were observed in multiple WM tracts, including the body and splenium of the corpus callosum and the retrolenticular part of the internal capsule, in the PD+AUD group compared to the PD–AUD group. CAGE scores in the PD+AUD group and ASI-R scores in the PD–AUD group were significantly correlated with FA values for the corpus callosum. No WM volume differences were found.
Conclusions: Our findings revealed microstructural changes in multiple WM tracts, including the corpus callosum and internal capsule, suggesting they could be significant neural correlates of AUD in patients with PD.

PS250

Appearance of atypical cortical rhythm during fear conditioning in rats

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Abstract
Specific electroencephalographic (EEG) activity occur during a certain behavior. To find a specific EEG marker during fear response, EEG was recorded from the frontal and parietal cortices and the hippocampus of Sprague-Dawley rats during and after fear conditioning. Fear response such as freezing behavior was evoked by a tone predicting the occurrence of electric shock through classical aversive conditioning. EEG activity was subjected to power spectrum analysis, and then they were compared to the EEG activity observed during other behavioral states, such as awake still, awake moving, and paralyzed. In awake still, 1.95~2.34 Hz peak was observed in the frontal cortex, while 7.03 Hz peak as well as 1.95~2.34 Hz peak was observed in both the parietal cortex and the hippocampus. In awake moving, there was a prominent 7.81 Hz peak in the hippocampus, though both 1.56 Hz and 7.81 Hz peaks were in all regions. In paralyzed state, both 1.56 Hz and 3.51 Hz peaks observed in all regions. In addition, 6.25 Hz peak observed in the hippocampus. In freezing, a prominent 3.12 Hz peak was observed in the frontal cortex, though both 3.12 Hz and 5.85 Hz peaks were observed in the hippocampus. These observations suggest that the characteristic rhythm could represent functional alteration of the brain region affected by a certain behavioral learning such as fear and could serve as a biomarker of fear.

POST TRAUMATIC STRESS DISORDERS: PS251 – PS267

PS251

Pharmacological suppression of the lateral habenula via the activation of mu opioid receptors ameliorate helpless behaviors in a rodent model of depression

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Abstract
Endogenous opioid systems are involved in stress response and emotion regulatory processes. Stressful events release endogenous opioids and activate three different types of opioid receptors. Among these, μ type opioid receptors (mOR) are G-protein coupled receptors and regulate presynaptic neurotransmitter release probability. mOR deficient mice exhibit reduced anxiety, reduced depressive-like behaviors and decreased corcisoterone responses. However, whether this is due to the compensatory effect of constitutive genetic deficiency of mOR remains to be determined. The lateral habenula (LHb), a brain area involved in depressive disorders show the highest expression of mORs, however mOR in the LHb has not been investigated. To investigate the role of mOR in the LHb, we performed a whole-cell patch clamp recordings. We found that pharmacological mOR activation successfully decreases both excitatory and inhibitory neurotransmission in the LHb, while the net effect of mOR activation was reduction in synaptic transmission. Previous studies observed enhanced activity of the LHb in animal models of depression, thus reducing the activity of the LHb may contribute to reverse helpless behaviors. Surprisingly, mOR-induced decrease in synaptic transmission remains intact in the LHb obtained after exposure to an hour-long restraint plus tail-shock stress, raising the possibility of mOR activation as a pharmacological tool to reduce the activity of abnormally potentiated LHb after stress exposure. The selective activation of mOR in the LHb indeed ameliorate