after controlling for age, sex, depression, and anxiety. Furthermore, CC in low alpha band was positively associated with emotional neglect ($r = .25$, $p = .011$) and negatively associated with posttraumatic growth ($r = -.49$, $p = .024$) in Val/Val individuals.

Conclusion: The results suggest that BDNF could moderate the relationship between childhood maltreatment and resting state EEG-based brain network. Only in the Val/Val group, individuals who experienced more childhood traumatic events showed enhanced CC, GE, and NS in low alpha band.

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P37.03

Low frequency alpha (8–10 Hz) activity correlated with inhibitory behavior

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Objective: Alpha frequency of EEG and the default mode network (DMN) are predominantly activated during resting-state. Also, alpha frequency power is known to be related with inhibitory function. This study investigated the neuropsychological characteristics of alpha band power and alpha DMN at resting state.

Methods: Resting-state EEG, go/nogo ERP/behavioral data, and psychological measures were examined in a total of 101 healthy individuals. Relative alpha (8–12 Hz), low-alpha (8–10 Hz), high-alpha (10–12 Hz) powers were calculated from the resting-state EEG data. Source activations of 25 DMN regions and their global/nodal network measures (clustering coefficient (CC), path length (PL), efficiency, strength, and eigenvector centrality (EC)) were also calculated. Psychological measures included the Gray's behavioral inhibition/behavioral activation scale (BIS/BAS), the Barratt impulsivity scale (BIS), and the Conner's adult ADHD rating scale (CAARS). Individuals were divided into 3 groups (low, middle, high) based on the level of power of each total/low/high-alpha frequency band.

Results: Significant group differences were found in low-alpha frequency power. The high group had significantly higher BIS score (behavior inhibition), significantly higher levels of global/nodal CC, efficiency, and strength, and a significantly lower PL at all region after Bonferroni correction compared to low and middle group. BIS inhibition was positively correlated with global/nodal CC, efficiency, and strength, and negatively correlated with global PL.

Conclusion: Our study revealed that low frequency alpha power is specifically related with inhibitory function. The results also suggest that DMN of low alpha frequency band could be a potential candidate of biological marker of inhibitory function.

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P37.04

Optogenetic manipulation of mural cells evoked regional brain blood flow changes in the deep brain

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A motivated behavior is controlled by the ventral striatum containing circuits. It is expected that the regional brain function is regulated by the regional brain hemodynamics, however, there is no ideal tool to address how the regional brain hemodynamics control the regional circuit function and consequently lead to the behavioral readout. In this study we have developed transgenic mouse tools which enable us to optogenetically manipulate the regional brain blood flow in a freely moving situation. We exploited tetracycline-controllable gene induction system (Tet system) and targeted mural cells by crossing parvalbumin promoter-tTA line and actb knockin tetO-optogene line (tetO-channelrhodopsin [ChR2-YFP] or tetO-photoactivatable adenylate cyclase [PAC-2A-GFP]). In either combination, tTA-mediated gene induction occurred in mural cells but not in parvalbumin-positive interneurons. We characterized tTA-mediated gene induction by immunohistochemistry; we used NG2 (marker for pericytes and smooth muscle cells), laminin alpha 2 (marker for parenchymal basal membrane), and smooth muscle actin (SMA, marker for smooth muscle) antibodies. We identified that all GFP/YFP signals were aligned with laminin alpha 2 signals, indicating that GFP/YFP positive cells were associated with blood vessels. Among them, 30% of GFP positive cells were colabelled with both SMA and NG2, and remaining cells were positive for NG2. We then tested whether the optogenetic manipulation worked. We inserted a laser doppler probe, which was attached an optic fiber, into the ventral striatum and measured regional blood flow levels upon illumination. As a result, ChR2-mediated manipulation induced a regional blood flow decrease and PAC-mediated manipulation induced an increase. Our tools will facilitate researches aiming to address the direct causality between regional hemodynamics and circuit functions in motivated behavior.

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P37.05

Machine-learning-based classification between post-traumatic stress disorder and major depressive disorder using P300 features

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Background: The development of optimal classification criteria for specific mental disorders which share similar symptoms is an important issue for precise diagnosis. We investigated whether

P300 features in both sensor-level and source-level could be effectively used to classify post-traumatic stress disorder (PTSD) and major depressive disorder (MDD).

Method: EEG signals were recorded from fifty-one PTSD patients, 67 MDD patients, and 39 healthy controls (HCs) while performing an auditory oddball task. Amplitude and latency of P300 were evaluated, and the current source analysis of P300 components was conducted using sLORETA. Finally, we classified two groups using machine-learning methods with both sensor- and source-level features. Moreover, we checked the comorbidity effects using the same approaches (PTSD-mono diagnosis (PTSDm, $n = 28$) and PTSD-comorbid diagnosis (PTSDc, $n = 23$)).

Results: PTSD showed significantly reduced P300 amplitudes and prolonged latency compared to HCs and MDD. Moreover, PTSD showed significantly reduced source activities, and the source activities were significantly correlated with symptoms of depression and anxiety. Also, the best classification accuracies at each pair were 80.00% (PTSD-HCs), 67.92% (MDD-HCs), 70.34% (PTSD-MDD), 82.09% (PTSDm-HCs), 71.58% (PTSDm-MDD), 82.56% (PTSDc-HCs), and 76.67% (PTSDc-MDD).

Conclusion: Altered P300 characteristics in sensor- and source-level might be useful biomarkers to discriminate PTSD from MDD and HCs.

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P37.06

Interaction of FKBP5 polymorphism and childhood trauma on brain volume in healthy individuals

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Introduction: Gene–environment interaction influence brain functional and morphologic changes, and these mechanisms are related to greater vulnerability or resilience to stress-related psychopathology. Our study is designed to investigate the contributions of gene with genotype information for 10 FKBP5 polymorphisms (rs9296158, rs3800373, rs1360780, rs9470080, rs4713916, rs4713919, rs6902321, rs56311918, rs3798345 and rs9380528), and their interaction effect with childhood trauma on brain volume in healthy individuals.

Methods: One hundred forty-four healthy volunteers (44 men and 100 women) were genotyped with respect to ten SNPs (rs) of FKBP5 through blood sampling. Participants underwent structural magnetic resonance imaging scan, and psychological assessments such as childhood trauma questionnaire and hospital anxiety and depression scale. A regression analysis, Johnson-Neyman technique, two-way analysis of covariance were conducted.

Results: We found that FKBP5 polymorphism as well as interaction between FKBP5 and childhood trauma predicted volume alteration in regions of left OFC and left MTG. Specifically, larger volume of the left OFC is associated with FKBP5 rs3800373, rs1360780, rs4713916, rs4713919, rs3798345 and with higher CTQ score. AC+CC, CT+TT, AG+AA, AG+AA and CT+TT genotype of each SNP alleviated the effects of childhood trauma on left OFC and was considered as a protective factor. Furthermore, larger volume of the left MTG is associated with FKBP5 rs3800373, rs4713916, rs3798345 and with higher CTQ score. CT+TT, AG+AA and CT+TT genotype of each SNP alleviated the effects of childhood trauma on left MTG and was considered as a protective factor.

Conclusions: FKBP5 has been shown to interact with childhood trauma to predict reactivity of the brain regions (left OFC, left MTG) in healthy participants. Increased volume of these regions may reflect neuroplasticity as a function of compensatory cognitions that help to reduce pathological anxiety.

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P37.07

Identification of potential neuromodulatory targets of stigmasterol through reverse docking integrated network pharmacology approach

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In our previous study, stigmasterol (ST), a plant cholesterol, is demonstrated to promote neuritogenesis, glutamatergic synaptogenesis, and neuronal migration. However, the precise mechanisms that facilitate neuromodulatory effects still remain unclear. In order to provide unique insights, the present studies, therefore, employed reverse pharmacophore mapping together with molecular docking analysis to identify possible new therapeutic targets. In addition, a network pharmacology approach was applied to elucidate the fine details of neuromodulatory mechanisms of ST. In pharmacophore mapping, a total of 209 potential targets were identified, which were refined and validated through molecular docking guided binding energy analysis. As a result, 60 potential targets were considered for the network construction, and subjected for pathway enrichment analysis. The results revealed the significantly binding of ST to the intracellular receptors. Network topology analysis also revealed many hub-bottleneck genes as the dynamic components of the network could be dominated by ST. KEGG pathway analysis indicated that these identified targets were associated with the neuroprotection and brain development. Our study indicates the pharmacological efficacies of ST in multi-target and multi-functional mode for a potential neuroactive compound to improve brain functions and combat neurodegenerative disease.

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P37.08

Serotonin modulates optimal coding of motion envelopes by enhancing neural and behavioral responses

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In order to output appropriate behaviors, sensory systems develop coding strategies that are adapted to highly dynamic natural stimuli. Such adaptation is generally thought to involve feedback projections and neuromodulators. Previous studies have reported that optimized coding of motion envelopes in the weakly electric fish *Apteronotus leptorhynchus* is mediated by glutamatergic