Expression levels of 1,100 transcripts, accounting for approximately 4.5% of analyzed transcripts, were altered in Tsc2(+/-) mice and reversed by rapamycin. These transcripts were enriched in neoplasm formation and inflammation. Regarding the mTOR pathway, the expression level of Tsc2 was reduced to a half of wild-type mice. Conversely, Tsc1 expression level was increased, and was suppressed by rapamycin administration. Gene expressions of Eef2k, Deptor and Ulk1 were increased and all of these increases were also suppressed by rapamycin. Our findings suggested an increased propensity for tumorgenesis and inflammation in Tsc2(+/-) brain.

PT708
Social interaction rescued abnormal mood and attention behaviors caused by acute stress in adolescent mice through ERK1/2 modulation
Ji-Woon Kim, Pyung Hwa Eun, Edison Luck Gonzales, Mee Jung Ko, Hyun Ah Oh, Chan Young Shin
Konkuk university, Republic of Korea

Abstract
Multiple stressors are intertwined with work, school and living conditions. Finding reasonable strategy to cope up with stresses has become an important issue to the current society. Although an inspiring idea, first proposed by Cassel, suggested that the positive social support could alleviate adverse stress responses, the underlying mechanisms are still uncovered. In the present study, we evaluated the stress-buffering effect of social interactions on a molecular signal related to abnormal behaviors induced by an hour of acute restraint stress in ICR mice. Interestingly, one hour of restraint stress induced the activation of ERK1/2, which was reduced in the stress group subjected to social interaction with conspecific mice. We also examined the effects of social interaction on behavioral changes induced by restraint stress, assessed through forced swimming test, and Y-maze test. The abnormal behaviors in the stress group were normalized by the addition of social interaction with conspecific mice. To specify the roles of ERK1/2 in these stress-induced abnormal behaviors, we investigated stress-induced behaviors and ERK1/2 level in prefrontal cortex using ERK1/2 inhibitors, which was reduced to approximately 4.5% of analyzed transcripts, were altered in Tsc2(+/-) mice and reversed by rapamycin. These transcripts were enriched in neoplasm formation and inflammation. Regarding the mTOR pathway, the expression level of Tsc2 was reduced to a half of wild-type mice. Conversely, Tsc1 expression level was increased, and was suppressed by rapamycin administration. Gene expressions of Eef2k, Deptor and Ulk1 were increased and all of these increases were also suppressed by rapamycin. Our findings suggested an increased propensity for tumorgenesis and inflammation in Tsc2(+/-) brain.

PT709
Dopaminergic functions suppress feeding behavior through neuropeptides in the hypothalamus.
Naomi Yonemochi, Hoshi University, Japan

Abstract
Amphetamine and mazindol are known to reduce food intake by affecting the central dopaminergic neurons. However, central dopaminergic functions in the regulation of feeding behavior still remain unclear. The hypothalamus is a key player in the control of food intake. Therefore, it is possible that dopaminergic functions in the hypothalamus regulate feeding behavior. We showed that density of dopamine D1 and D2 receptors was higher in the lateral hypothalamus (LH, hunger center) than in the ventromedial nucleus of hypothalamus (satiety center).

Thus, we investigated the role of lateral hypothalamic dopaminergic functions in feeding behavior of mice. Using in vivo microdialysis, we showed that refeeding significantly increased the dopamine level in the LH after 16-hour fasting. Furthermore, the dopamine D2 receptor agonist SKF 38393 injected into the LH significantly decreased food intake of fasted mice. This decrease was abolished by the dopamine D2 receptor antagonist SCH 23390. Injection of the dopamine D2 receptor agonist quinpirole also decreased food intake and this effect was blocked by the dopamine D2 receptor antagonist l-sulpiride. Since the hypothalamus contains orexigenic neuropeptides such as agouti-related peptide (AgRP), neuropeptide Y (NPY) and orexin and anorexigenic neuropeptides such as α-melanocyte-stimulating hormone (α-MSH), we examined whether dopaminergic functions regulate these neuropeptides. Using RT-PCR, we indicated that SKF 38393 significantly decreased the mRNA levels of AgRP and NPY, whereas quinpirole induced significant reduction in the mRNA level of preproorexin, precursor of orexin, and significantly increase in the mRNA level of proopiomelanocortin, precursor of α-MSH. In conclusion, these results suggest that the stimulation of dopamine D2 receptors inhibits feeding behavior by inhibiting AgRP and NPY neurons and that the stimulation of dopamine D2 receptors inhibits feeding behavior through the inhibition of orexin neurons and the activation of α-MSH neurons.

PT710
Vasopressin increases empathic responding among those high in primary psychopathy
Author/Co-authors
Jung Hwa Han1, Benjamin A. Tabak2, Meghan L. Meyer1, Elizabeth Castle3, Janine M. Dutcher4, Michael R. Irwin2,4,5,6, Matthew D. Lieberman2,3, and Naomi I. Eisenberger7
1Section of Affect and Neuroscience, Yonsei University College of Medicine 2Department of Psychology, University of California – Los Angeles, CA 3Department of Psychology, Princeton University 4Department of Psychiatry and Biobehavioral Sciences, David Geffen School of Medicine, University of California – Los Angeles, CA 5Cousins Center for Psychoneuroimmunology, David Geffen School of Medicine, University of California – Los Angeles, CA 6Semel Institute for Neuroscience, David Geffen School of Medicine, University of California – Los Angeles, CA

Abstract
Individuals with high levels of primary psychopathy, which is associated with a reduced tendency to experience negative affect, tend to show a deficiency in experiencing affective empathy, even though cognitive empathy is left intact. Research on the biological processes underlying empathy has focused on the neuropeptides oxytocin and vasopressin due to their roles in mediating a host of social behaviors. To date, most human research has focused on the social effects of oxytocin with far fewer investigating vasopressin. Although vasopressin has often been associated with aggression in animal research, recent findings in humans suggest that vasopressin may increase prosocial behavior. Using a randomized, double-blind, placebo controlled, between-subjects design, we investigated the main effect of intranasal administration of vasopressin compared to placebo on empathic responses – personal distress and empathic concern – in 83 healthy university students (60 female; Mean Age = 20.84, SD = 2.80). In addition, we investigated the moderating role of psychopathy on the effect of vasopressin on empathic responding.
Results showed no significant main effect of drug condition on empathic responses. However, a significant interaction effect between drug condition and primary psychopathy was found for personal distress and a marginally significant interaction effect was found for empathic concern. Simple effects analyses showed significant negative correlations between primary psychopathy and personal distress as well as empathic concern in the placebo group, but not in the vasopressin group. In addition, among participants with higher levels of primary psychopathy (i.e., +1 SD above the mean) vasopressin increased personal distress and empathic concern compared to placebo.

Results suggest that vasopressin increases emotional arousal and empathic responding in individuals with higher levels of primary psychopathy. This calls for further research on the biological substrates of empathy with focus on vasopressin.

PT711
Molecular analysis of neural action mediated by the antipsychotic agent olanzapine in high glucose exposure
Karu Ikuo1, Kyoysuke Yamanishi2, Sachi Kuuahara-Otani3, Seishi Maeda2, Wen Li1, Yuko Watanebe1, Momoko Yoshida4, Tetsu Hayakawa1, Haruki Okamura1, Hiromichi Yamanishi4 and Hitasho Matsunaga1
1Department of Neuropsychiatry, 2Department of Anatomy and Cell Biology, 3Laboratory of Tumor Immunology and Cell Therapy, and 4Hirakata General Hospital for Developmental Disorders, 2-1-1 Tsudahigashi, Hirakata, Osaka 573-0122, Japan

Abstract
Objectives: Antipsychotic agent, olanzapine was used widely in the treatments for schizophrenia, bipolar disorder, and so on. It was found that neurons, especially its mitochondria exposed to olanzapine were damaged by oxidative stress resulting in induced autophagy, a controlled cellular self-digestion process in human SH-SY5Y neuronal cell line.

Olanzapine was prohibited to patients with diabetes. However, the molecular mechanisms of how olanzapine effects on neurons in high glucose situations remain unknown. The aim of this study is to verify the molecular influence of olanzapine on neurons in high glucose circumstances.

Methods: Human SH-SY5Y neuronal cell line was used in this study, and was grown in the same manner as previously described (Ljubica Vucicevic, et al. Autophagy. 2014). Cells were rested for 24 hours in normal (5 µM) and high glucose (50 µM) medium, and then treated in the same medium with olanzapine (100 µM) for another 24 hours. We performed a comparative analysis of the gene expression profiles using microarrays. We subsequently categorized genes using a web-based bioinformatics analysis tools: network explorer of Ingenuity Pathway Analysis. We then confirmed significant group-differences in mRNA and protein expression levels using qRT-PCR and western blotting.

Results: According to the microarray analysis, several genes which were expressed differentially were picked up focusing on molecules related to cell death and cell protection such as autophagy. Significant differences of these genes were shown by qRT-PCR and western blotting.

Conclusions: These findings supported that olanzapine might mediate the cellular damage and autophagy protects neurons from mitochondrial death by molecular mechanism. Further examination focusing on neural vitality and other function is warranted.

PT712
Functional expression of choline transporter like-protein 1 (CTL1) and CTL2 in human brain microvascular endothelial cells
Beniko Iwao1, Naomi Hara1, Masato Inazu1, Takeshi Inoue1, Yuiko Kawai1, Hiroshi Nishihara1, Tsuyoshi Yamanaka1, Miki Yara1
1Tokyo Medical University, Japan, 2Hokkaido University, Japan

Abstract
Objective: The brain is protected from the rest of body by the blood-brain barrier (BBB) including microvascular endothelial cells. The BBB at the level of the brain microvessel endothelium is the major site of the selective permeability. The central nervous system requires choline to synthesize the neurotransmitter acetylcholine and the membrane phospholipids phosphatidylcholine and sphingomyelin. Therefore, the transport of choline from the blood to the brain through the BBB is a physiologically important process. In this study, we examined the functional characterization of choline transporter in human brain microvascular endothelial cells (hBMECs).

Methods: We examined the [3H]choline uptake into hBMECs. The expression of mRNA and protein of choline transporters was investigated by real-time PCR and Western blotting. The immunohistochemical and immunocytochemical detection was performed to determine the localization of choline transporter in human brain cortex and hBMECs, respectively.

Result: hBMECs was a saturable process that was mediated by a Na+-independent, membrane potential and pH-dependent transport system. Choline uptake was inhibited by various organic cations also interacted with the choline transport system. The cells have two different [3H]choline transport systems. Choline transporter-like protein 1 (CTL1) and CTL2 mRNA were expressed in hBMECs. CTL1 and CTL2 proteins were localized to microvascular endothelial cells in human brain cortical sections. Both CTL1 and CTL2 proteins were expressed on the plasma membrane. CTL2 proteins are mainly expressed in mitochondria.

Conclusion: We conclude that choline is mainly transported via intermediate choline transport system, CTL1 and CTL2 in hBMECs. These transporters are responsible for the uptake of extracellular choline and organic cations. CTL2 participate in choline transport mainly in mitochondria. Choline oxidation occurs in the mitochondria. The function of CTL2 may be associated with the control of choline oxidation.

PT713
Oedipus Complex with Brain Injury
Chia-Lun Tsai
Department of Psychiatry, Hualien Tzu Chi Hospital, Taiwan

Please address correspondence to:
Chia-Lun Tsai, M.D.
Department of Psychiatry, Hualien Tzu-Chi Hospital, 707, Sec. 3, Zongyang Rd, Hualien 970, Taiwan
Tel: 886-3-8561825
E-mail: earthinsea@gmail.com

Abstract
Objectives: Oedipus complex explains the emotions and ideas that concentrates upon a child’s desire to have sexual relations with the parent of the opposite sex. Oedipus complex keeps in the unconscious via dynamic repression, and is usually revealed through psychoanalysis. I report a case who develops Oedipus complex after a severe traumatic brain injury.