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 Eif2k, 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
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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 such as rosuvastatin and SL327. Fascinatingly, these ERK1/2 inhibitors rescued abnormal attention behavior and mood behavior. These results suggest that social interactions could alleviate the acute restraint stress-induced behavioral abnormalities in mice, as well as the overt ERK1/2 activation in the medial prefrontal cortex. Moreover, ERK1/2 might be an important target in social buffering effects against stress.

PT709
Dopaminergic functions suppress feeding behavior through neuropeptides in the hypothalamus.
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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 hypothalumus 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 (satiation 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 D1 receptor agonist quinpirole also decreased food intake and this effect was blocked by the dopamine D1 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 propiomelanocortin, 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 D1 receptors inhibits feeding behavior through the inhibition of orexin neurons and the activation of α-MSH neurons.

PT710
Vasopressin increases emphatic responding among those high in primary psychopathy
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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.