Abstract
The risk of antidepressant treatment versus the risk of major depression itself on the development of the fetus is a long-term subject of debates. Particularly important are possible delayed consequences on mental well-being which can occur later in life. The aim of the present studies was to verify the hypothesis that perinatal exposure to venlafaxine has impact on hormones involved in stress coping as well as on anxiety and depression-like behaviour of adult offspring.

Dams were treated orally with one of three doses of venlafaxine or placebo starting on day 15 of gestation until day 21 post partum. At that time, pups were weaned and caged in groups according to gender. As adult, both male and female offspring were subjected to behavioural tests to measure anxiety and depression-like behaviours. Concentrations of corticosterone and aldosterone were analysed by radioimmunoassay in blood obtained by decapitation under non-stress conditions in the morning.

Plasma corticosterone concentrations were higher in females than in males and they were enhanced in offspring of both genders exposed periatally to venlafaxine (significant main factors gender and treatment by two-way ANOVA). Post-hoc analysis showed that venlafaxine exposure-induced enhancement of plasma corticosterone was particularly strong in females exposed to the lowest (7.5mg/kg) and the highest (75mg/kg) dose. Exposure to venlafaxine resulted also in a mild increase in plasma aldosterone (main factor treatment), particularly in males from the highest dose group. On the other hand, exposure to venlafaxine led to reduced anxiety and depression-like behaviour as shown by several variables in elevated plus maze, light-dark box and forced swim tests.

In conclusion, treatment with venlafaxine during gravidity and lactation may result in positive consequences on signs of anxiety and depression in adult offspring associated with only mild increase in stress hormone levels. *Supported by grants VEGA2/0168/15, VEGA2/0057/15 and partly by APVV-14–0840*

PS114

Effects of Tianeptine on mTOR Signaling in Rat Hippocampal Neurons

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Abstract
Objective: Recent studies have demonstrated that mTOR activation may be related to the antidepressant action. However, the relation between mTOR signaling activation and currently prescribed antidepressants have not been well elucidated. The aim of the present study was to find out whether alterations in mTOR signaling could be observed following treatment with tianeptine under toxic conditions induced by B27 deprivation in rat hippocampal neuronal cultures. Additionally, we investigate whether this drug affect the synaptic proteins, neurite outgrowth and spine density via mTOR signaling.
Methods: Using Western blotting, we measured the phosphorylation levels of mTOR, 4E-BP-1, p70S6K, Akt, and ERK. Changes in BDNF, dendritic outgrowth, spine density, and synaptic proteins (PSD-95, synaptophysin, and GluR1) were measured.
Results: Tianeptine significantly increased the phosphorylation of mTOR, 4E-BP-1, p70S6K, Akt, and ERK. The increase in mTOR phosphorylation was blocked by the PI3K, MEK, and mTOR inhibitors. Tianeptine increased BDNF, dendritic outgrowth, spine density, and synaptic proteins, which were blocked by the mTOR inhibitor.
Conclusions: In this study, we demonstrated that tianeptine activates the mTOR signaling pathway and increases dendritic outgrowth, spine density, and synaptic proteins through mTOR signaling, suggesting that mTOR signaling activation may be related to the antidepressant effects of tianeptine.
Keywords: Depression, Antidepressant drugs, Tianeptine, mTOR signaling, Neuroplasticity

PS115

The Effect of Fluoxetine on Behaviors in Transient Forebrain Ischemic Gerbil

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Abstract
Objective: This study aims to explore the effect of fluoxetine on memory, locomotor and depressive behavior in transient forebrain ischemic model of gerbil.
Methods: The two doses of fluoxetine (10, 40 mg/kg) or vehicle were intraperitoneally administered once 30 min before ischemic surgery in gerbil. Novel object recognition test, spontaneous motor activity, learned helplessness test were performed 4 days, 8 days, or 9 days, respectively, after sham or ischemic surgery.
Results: Fluoxetine treatment (40 mg/kg) significantly reduced recognition memory in sham operated gerbil. However, fluoxetine (10, 40 mg/kg) did not affect ischemia-induced impairment in recognition memory. The treatment of fluoxetine (10, 40 mg/kg) significantly inhibited locomotor hyperactivity induced by transient ischemia even though fluoxetine (40 mg/kg) did not affect spontaneous motor activity in the sham operated gerbils. Fluoxetine did not affect depressive behavior in sham and ischemic gerbils.
Conclusion: The treatment of fluoxetine inhibited ischemia – induced hyperactivity, but did not affect memory and depressive behavior in transient forebrain ischemic gerbils.
Key Words: transient forebrain ischemia, fluoxetine, novel object recognition test, spontaneous motor activity, learned helplessness test

PS116

Desvenlafaxine prevents white matter injury and improves the decreased phosphorylation of rate-limiting enzyme of cholesterol synthesis in a chronic mouse model of depression.

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Abstract
Objective: Serotonin/norepinephrine reuptake inhibitors antidepressants exert their effects by increasing serotonin and