reduction of inflammatory markers. Next PG decreases microglia activation after FCI. Our finding suggests that neurotranscorticoid steroids have a relevant role in the prevention of ischemic outcome in immature brain.

This study was supported by grants GACR P304/12/G069, P304/14/20613S, P303/12/1464, GAUK project No.165115, TACR-TE01020028 and institutional support RVO: 67985823 and CZ.2.16/3.1.00/22197; NPU I(LO) MSMT-3487/2013 (National Program of Sustainability).

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Early Antipsychotic Treatment in Childhood/Adolescent Period has Long-term Effects on Dopamine Receptors of Adult Rat Brains

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Abstract
Background: Prescription/use of antipsychotic drugs (APDs) in children is increasing rapidly worldwide, despite serious limitations in the safety and efficacy of APD use on the developing brain. Whilst APDs are known to have a high affinity for dopamine (DA) receptors to produce therapeutic effects, DA receptors are also involved in critical neurodevelopmental processes. This study investigated the long-term effects of childhood/adolescent APD use on DA receptors in adult brains.

Methods: Male and female rats (n=6/group) were treated with Aripiprazole (1 mg/kg), Olanzapine (1 mg/kg) and Risperidone (0.3 mg/kg), 3 times/day from postnatal day (PD) 22-50. Animals were sacrificed on PD106. Levels of DA D1R and D2R were investigated via quantitative autoradiographic and western blot techniques.

Results: In comparison to controls, D1R protein levels were significantly decreased following Risperidone treatment in the nucleus accumbens (NAc) of male rats (p<0.01), and Aripiprazole treatment in the ventral tegmental area of females (p<0.001). Furthermore, D2R levels were increased in the prefrontal cortex of female rats (p<0.05), with a trend to decrease in the Hippocampus of males (p=0.099).

Trends to significant increases of D1R and D2R bindings were found in female rats. In comparison to controls, Risperidone increased D1R binding in the Hippocampus (p=0.077), whilst Olanzapine increased D2R binding in the NAc (p=0.054).

Conclusions: Long-term changes to D1R and D2R were uncovered following childhood/adolescent APD treatment, indicating the potential long-term effects of APD use on the DA neurotransmitter system during the critical neurodevelopmental window. Furthermore, differing effects of childhood APD treatment on D1R and D2R were found across both genders and APD treatment groups. Further investigations into the neural mechanisms involved for observed differences across drug treatment groups may shed further light on potential chronic effects of APD in the young population. (M. De Santis was supported by an Australian Rotary Health scholarship).

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Treatment of catatonia in autism spectrum disorder: 2 case reports and literature review

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Abstract
Objectives: Catatonia is a severe complication of autism spectrum disorders characterized by increased slowness, difficulty in initiating actions, and increased passivity. The aim of this study is to describe 2 patients diagnosed with autism spectrum disorder who presented with catatonia and to provide a review of literature.

Results: Increasing recognition is being given to catatonic symptoms presenting in children and adolescents with autism spectrum disorder. Recent studies report that high doses of benzodiazepines and the use of electroconvulsive treatment are effective in these conditions.

Case reports: Two cases of high functioning girls diagnosed with autism spectrum disorder are presented. The first case was a 15 year-old girl who presented with freezing in peculiar postures with difficulty initiating movement, slow verbal responses, abnormal repetitive movements, and difficulty crossing demarcation lines. Complete remission of catatonic symptoms was achieved by improvement of family functioning and provision of safe and organized environment along with 6 mg per day of lorazepam. The second case was an 11 year-old girl who also presented with difficulty initiating movement and crossing lines. She achieved partial remission by environmental management and use of ethyl loflazepate, a long-acting benzodiazepine.

Conclusions: The present case reports showed that psychological stress may be a precipitating factor for exacerbation of catatonia in patients with autism spectrum disorder and that catatonia in patients with autism spectrum disorder is not restricted to those with low intelligence. High dose benzodiazepine such as lorazepam and ethyl loflazepate may be effective and well tolerated in treating catatonic symptoms of autism spectrum disorder.

PM338
Investigation of the association of rare single nucleotide variants in methyl-CpG-binding domain protein 5 (MBD5) with phenotypes of autism spectrum disorders and schizophrenia.

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Abstract
The MBD5 gene has been widely cited as a risk gene for neurodevelopmental features. Both partial and complete deletions of MBD5 involving coding and/or non-translated exons are resulted in autism spectrum disorders (ASD), intellectual disability and epilepsy. A significant excess of a rare single nucleotide variant (SNV) in MBD5 coding exon have been detected in ASD patients. The phenotypes observed in patients having disruption of MBD5 include autistic-like symptoms, developmental delay, behavioral problems, repetitive behaviors and seizures. The aim of the present study was to investigate the association between rare MBD5 variants and neuropsychiatric pathogenesis.

A total of 192 ASD (mean age ± SD = 16.3 ± 8.4 years; 77.6% male) and 370 schizophrenia (mean age ± SD, 49.7 ± 14.8 years; 53.0% male) individuals participated. First, we conducted exon-targeted resequencing of MBD5 with next-generation sequencing technology in 562 Japanese patients and detected 12 rare missense variants. We compared phenotypes of patients having these variants with the core characteristics