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EPP0706

Mitochondrial ATP production is impaired in neural stem/progenitor cells derived from olfactory neuroepithelium of patients with schizophrenia

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Introduction: Neural stem/progenitor cells derived from olfactory neuroepithelium (hereafter OE-NS/PCs) are emerging as a viable proxy and a valuable tool for translational studies on severe mental illnesses (SMI). In this respect, the use of OE-NS/PCs as a surrogate cellular model of schizophrenia (SZ) has enabled insights into cell signaling and cell cycle dynamics in this disease.

Objectives: We explored whether mitochondrial dysfunction, which has been already associated with SZ, may have a role in the altered proliferation pattern previously observed in OE-NS/PCs of SZ patients.

Methods: OE-NS/PCs were collected from 20 patients and 20 healthy controls (Hcs) by nasal brushing, cultured in proper medium and expanded. Fresh OE-NS/PCs at passage 3 of both groups underwent BrdU proliferation assays or were frozen for later use. Mitochondrial ATP production was measured in both fresh and thawed OE-NS/PCs by using the ATPlite Luminescence Assay kit.

Results: Fresh OE-NS/PCs of patients grew at a higher rate than those of HCs (M-W U=0; $p<0.001$), whereas the proliferation of thawed OE-NS/PCs of both groups exhibited an opposed pattern (at passage 6, $p=0.002$). Mitochondrial ATP production was significantly lower in OE-NS/PCs of patients than in those of HCs (M-W U=0; $p=0.02$), regardless of freeze-thaw conditions (M-W U=6; $p=0.77$).

Conclusions: Mitochondrial ATP production is negatively affected in OE-NS/PCs of SZ patients as compared to those of HCs. This evidence does not differ in fresh OE-NS/PCs and OE-NS/PCs undergoing freeze-thaw cycles, which instead perturb the proliferation pattern of SZ OE-NS/PCs.

Keywords: cellular models; mitochondria; schizophrenia; translational psychiatry

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Drug-induced metabolic syndrome hasn't associations with 5-HT receptor genes polymorphisms in patients with schizophrenia

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Introduction: Metabolic disturbances are common in patients maintained on neuroleptics. These abnormalities significantly increase the physical comorbidity and mortality rates due to cardiovascular disease. We hypothesized that 5-HT receptor genes polymorphisms have associations with drug-induced metabolic syndrome development in schizophrenic patients.

Objectives: To investigate the role of polymorphic variants of serotonin receptors genes in the development of antipsychotic-induced metabolic syndrome.

Methods: 467 patients with schizophrenia receiving long-term antipsychotic therapy were investigated. The mean age was 40.0 ± 11.6 years. The standard phenol-chloroform method for DNA isolation was used. Genotyping was carried out on eight SNP's of genes HTR1A, HTR2A, HTR3A and HTR2C with the MassARRAY[®] Analyzer 4 (Agena Bioscience[™]) using the set SEQUENOM Consumables iPLEX Gold 96 on the base The Core Facility "Medical Genomics", Tomsk NRMC.

Results: The prevalence of metabolic syndrome was 26.1%. In the study sample, there were significantly more women with metabolic syndrome (56.6%) than men (43.4%) ($p=0.002$). The majority of patients with metabolic disturbances were aged >40 years (62.3%), versus 40.9% in the group without metabolic disorders ($p<0.001$). The duration of the disease was statistically significantly higher in the group of patients with metabolic syndrome ($p=0.003$). We did not find statistically significant associations of polymorphic variants of the studied genes with the development of the drug-induced metabolic syndrome.

Conclusions: Our results do not demonstrate any significant association between allelic variants of serotonin receptor genes and metabolic syndrome in patients with schizophrenia. Conflict of interest. The authors declare no conflict of interest. Supported by Grant of RSF 19-75-10012.

Keywords: Serotonin receptors; Metabolic syndrome; schizophrenia; Genes

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Investigation of the role of polymorphic variants FTO gene in schizophrenia patients with metabolic syndrome

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