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Glycine transporter inhibitor sarcosine changes neuronal and glial parameters in the left dorsolateral prefrontal cortex and glutamatergic parameters in the left hippocampus in stable schizophrenia

D. Strzelecki[1,*], M. Podgórski[2], O. Kałużyńska[1], M. Kotlicka-Antczak[1], O. Gawlik-Kotelnicka[1], A. Gmitrowicz[3], L. Stefańczyk[2], P. Grzelak[2]
[1] *Central Clinical Hospital, Department of Affective and Psychotic Disorders–Medical University of Łódź, Łódź, Poland*
[2] *University Hospital No.1, Department of Radiology–Diagnostic Imaging–Medical University of Łódź, Łódź, Poland*
[3] *Central Clinical Hospital, Department of Adolescent Psychiatry–Medical University of Łódź, Łódź, Poland*
* *Corresponding author.*

Introduction Sarcosine - glycine transporter inhibitor - increases glycine concentration around NMDA (N-methyl-D-aspartate) receptors. Function of the glutamatergic system in the prefrontal cortex and hippocampus is impaired in schizophrenia, which may lead to negative and cognitive symptomatology.
Aims We evaluated the influence of sarcosine therapy on the concentration of metabolites (NAA, N-acetylaspartate; Glx, complex of glutamate, glutamine and γ-aminobutyric acid (GABA); mI, myo-inositol; Cr, creatine; Cho, choline) in the left dorso-lateral prefrontal cortex (DLPFC) and left hippocampus in patients with stable schizophrenia.
Methods Fifty patients with schizophrenia, treated with constant antipsychotics doses, in stable clinical condition were randomly assigned (25 patients in each group) to administration of sarcosine (2 g) or placebo for six months. ^{1}H-NMR spectroscopy (1.5 T) in both localisations and clinical evaluation (PANSS) was performed before and after sarcosine addition.
Results Initially we noted no differences in metabolite concentrations between groups. In the left DLPFC, NAA/Cho, mI/Cr and mI/Cho ratios were significantly higher in the sarcosine than the placebo group after six months. In the sarcosine group, NAA/Cr, NAA/Cho, mI/Cr, mI/Cho ratios also increased compared to baseline values. In the placebo group, only the NAA/Cr ratio increased. In the left hippocampus Glx/Cr and Glx/Cho decreased in sarcosine group at the end of our study.
Conclusions The addition of sarcosine to antipsychotic therapy for six months caused increase of neurons viability (NAA) and neurogilal activity (mI) markers in the left DLPFC and decrease of hyperglutamatergic overstimulation parameters in the left hippocampus with simultaneous improvement of clinical parameters including negative symptoms.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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Selected metabolites of kynurenine pathway and response to antipsychotic treatment in schizophrenia

K. Szymona[1,*], H. Karakuła-Juchnowicz[2], M. Flis[3], T. Kocki[4], A. Urbanska[5], R. Kloc[4], Z. Szymona[6], W. Rosa[7], E.M. Urbańska[4]
[1] *Medical University of Lublin, Mental Health Outpatient Clinic-Children's University Hospital, Lublin, Poland*
[2] *Medical University of Lublin, Department of Clinical Neuropsychiatry Department of Psychiatry- Psychotherapy and Early Intervention, Lublin, Poland*
[3] *Medical University of Lublin, Department of Psychiatry-Psychotherapy and Early Intervention, Lublin, Poland*
[4] *Medical University of Lublin, Laboratory of Cellular and Molecular Pharmacology- Department of Experimental and Clinical Pharmacology, Lublin, Poland*
[5] *Medical University of Lublin, Department of Psychiatry and Psychiatry Rehabilitation, Lublin, Poland*
[6] *PZ Cormay SA, Orphee Group, Lomianki, Poland*
[7] *Lublin University of Technology, Faculty of Fundamentals of Technology- Department of Applied Mathematics, Lublin, Poland*
* *Corresponding author.*

Introduction Deficit of glutamatergic transmission and aberrant function of kynurenine pathway, with disturbed synthesis of glutamate receptors antagonist, kynurenic acid (KYNA) and neurotoxic metabolite of kynurenine, 3-hydroxykynurenine (3-OH-KYN) have been implicated in the pathogenesis of schizophrenia.
Objectives Demonstrated by others higher level of KYNA in the brain may cause relative deficiency of glutamate-mediate transmission with resulting behavioural and cognitive changes.
Aims Search for predictors of satisfactory response to antipsychotic treatment based on the analysis of KYNA and 3-OH-KYN serum levels.
Methods Fifty-three patients with chronic schizophrenia and 46 healthy individuals were enrolled in the study. Quantitative analyses of KYNA and 3-OH-KYN were performed using high-pressure liquid chromatography (HPLC) and electrochemical detection, respectively. Clinical assessments (PANSS, SANS, SAPS) and blood analyses were conducted at 3 time-points: during the active phase of disease, after 4 weeks of modified pharmacotherapy, and after reaching remission.
Results In schizophrenia group, lower levels of KYNA ($P = 0.002$) and non-altered levels of 3-OH-KYN ($p = 0.195$), as compared to control, were detected during active phase of disease. Despite clinical improvement, no significant changes in the level of studied metabolites were observed later on. The initial level of 3-OH-KYN correlated negatively ($r = -0.368$; Spearman's rank) with clinical improvement (negative symptoms) ($P < 0.05$).
Conclusions 1. The peripheral dysregulation of kynurenine pathway metabolites in chronic schizophrenia manifests as relative increase in the ratio between neurotoxic 3-OH-KYN and neuroprotective KYNA. 2. The higher serum level of 3-OH-KYN during relapse of schizophrenia seems to predict poor response to antipsychotic treatment.
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Odors hedonic judgment in patients with schizophrenia. Influence of negative symptoms and β-endorphin levels

M. Urban-Kowalczyk
Medical University of Lodz, Department of Psychotic and Affective Disorders, Lodz, Poland

Introduction The relationship between olfactory and emotional processing is an area of increasing interest in schizophrenia research.
Objectives Olfactory identification deficits are well described in schizophrenia while the results for pleasantness ratings remain unclear.
Aims Evaluation of odor identification and hedonic judgment related to severity of negative symptoms and β-endorphin concentration.
Methods Fifty outpatients with schizophrenia were included in the study: 25 with negative symptoms (PN) and 25 without pre-