T193. THE ROLE OF METACOGNITION ON NEGATIVE SYMPTOMS: A PSYCHOLOGICAL MODEL FOR DIMINISHED EXPRESSION IN SCHIZOPHRENIA

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Background: The resistance of negative symptoms to pharmacologic treatment has spurred interest in understanding the psychological factors that contribute to their formation and persistence. However, little is understood about the psychological processes that reinforce and sustain the negative symptoms domain of diminished expression. Prior research has shown that higher levels of diminished expression relate to deficits in metacognitive capacity. We propose a more complex model in which diminished expression occurs when impairments in metacognitive self-reflectivity, alterations in higher-order language structure, and cognitive deficits interact and thus interfere with persons’ ability to understand and express emotions in ways others can recognize.

Methods: Individuals with schizophrenia-spectrum disorders (N=201) provided personal narratives including their life story and reflections regarding their mental illness and a clinician-rated interview of psychotic symptoms (i.e., Positive and Negative Syndrome Scale; PANSS). Self-reflectivity was measured with the Metacognition Assessment Scale-Abbreviated, and situation models were extracted from participants’ personal narratives via Coh-Metrix 3.0, an automated program that calculates basic and complex language indices. Diminished expression and cognitive symptoms were measured with the PANSS. Structural equation models (SEM) examined whether self-reflectivity mediated the impact of cognitive deficits and situation models on diminished expression.

Results: SEM revealed that self-reflectivity partially mediated the impact of situation models on diminished expression (β = -0.083, p = 0.005, ±95% CI [-0.141, -0.026]) and fully mediated the influence of cognitive symptoms in diminished expression (β = 0.099, p = 0.001, ±95% CI [0.038, 0.160]). Findings persisted after controlling for educational level.

Discussion: This study is the first of its kind to utilize a mediational model including higher-order linguistic structures, cognitive impairment and metacognition to explain diminished expression in psychosis. Results suggest that self-reflectivity, situation models and cognitive symptoms may be useful targets for intervention in efforts to decrease diminished expression.

T194. SUBJECTIVE ANOMALIES OF IMAGINATION AGGREGATE IN SCHIZOPHRENIA-SPECTRUM DISORDERS AND ARE ASSOCIATED WITH DISORDERS OF BASIC SELF

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Background: Imagination is the formation of ideas or images of something known not to be present to the senses. Clinical psychopathology has few notions addressing this domain apart from obsession and rumination. Some classic psychopathological notions such as Jaspers’ concept of pseudohallucination or the pseudo-obsession are relevant to this area. In a recent research project, informed by contemporary philosophy of mind and phenomenology, we have developed novel concepts targeting subjective disturbances of imagination and fantasy life with a focus on the schizophrenia-spectrum. Patients describe a spatialization of images, i.e., stable imagery with an articulated spatial structure being liable to inspection ‘from afar in the mind’ and often undergoing an autonomous development independently of the will of the patient (‘like watching a movie in the head’). Other notions address tacit, non-psychoactive erosions of the demarcation of fantasy life from perception and memory. A broad range of ideations (such as ‘daydreams’, ‘fears’, anticipations, intrusions, paranoid or suicidal ideation) may involve such structural disturbances of experience. Here, we present data from the first, cross-sectional study investigating the distribution of anomalies of imagination in different diagnostic groups and healthy controls as well as their association with positive symptoms, negative symptoms and disorders of basic selfhood.

Methods: The sample (N=81) included in- and outpatients with schizophrenia or another non-affective psychosis (N=32), outpatients with schizotypal disorder (N=15) or other mental illness (N=16) and healthy controls (N=18). The sample was 70% female with mean age 29.9 (SD 6.8; range 18–42) years. Anomalies of imagination were assessed with the Examination of anomalous fantasy and imagination (EAFI), which is an instrument recently developed in our group for a semi-structured interview exploring these experiences. The EAFI has shown very good reliability with average Kappa of 0.84. Disorders of basic self were assessed with the Examination of anomalous self experience (EASE) and positive, negative and general symptoms with the Positive and Negative Syndrome Scale (PANSS).

Results: Anomalies of imagination aggregated significantly (p < 0.000, Kruskall-Wallis test) in the schizophrenia-spectrum disorders compared to other mental illness with no significant difference between schizophrenia and schizotypal disorder. The group of healthy controls very rarely reported these anomalies and scored significantly lower (p < 0.000) than all diagnostic groups. In multivariate linear regression analysis (R2 = 0.66), EAFI score was significantly associated with EASE score (β = 0.62, p < 0.000), PANSS positive (β = 0.34, p = 0.01) and PANSS negative (β = 0.29, p = 0.02), but not PANSS general score (β = -0.29, p = 0.07). More than 79% of the schizophrenia-spectrum patients retrospectively reported the onset of these experiences to adolescence or earlier.

Discussion: The results of this cross-sectional study support that the subjective anomalies of imagination, targeted with the EAFI, are associated with the schizophrenia-spectrum. The association with disorders of basic self, which has been shown to have trait-like characteristics and to predict transition to schizophrenia-spectrum disorders, may reflect that the anomalies of imagination share a common experiential core-structure with self disorders. We suggest that the anomalies of imagination belong to an early onset level of psychopathology in the schizophrenia-spectrum and may have a relevance for differential diagnosis and early detection.
Three patients diagnosed with both schizophrenia spectrum and multiple sclerosis [1]. Immune-related single-nucleotide polymorphisms may support the hypothesis about a common etiology in a subgroup of schizophrenia and multiple sclerosis. Genetic pleiotropy for schizophrenia and other psychotic disorders, and there is a possibility factor for schizophrenia and other psychotic disorders, and there is a possibility for schizophrenia and multiple sclerosis [1].

Background: Multiple sclerosis has been suggested as a potential vulnerability factor for schizophrenia and other psychotic disorders, and there is a hypothesis about a common etiology in a subgroup of schizophrenia and multiple sclerosis [1]. Immune-related single-nucleotide polymorphisms have been associated with schizophrenia and genetic pleiotropy between schizophrenia and multiple sclerosis has been reported, but not between bipolar disorder and multiple sclerosis (at the level of major histocompatibility complex) [2]. As new data about the involvement of genetically-determined immune factors in the susceptibility to schizophrenia appear (e.g., variants of complement factor 4 possibly linked to synaptic pruning during brain development) [3] the interest for finding therapeutic targets within the immune system for psychotic disorders is also increasing.

Methods: Three patients diagnosed with both schizophrenia spectrum disorders (schizophrenia subtype 1, or schizoaffective disorder, depressive type n=1; female, mean age 43.7, with a history of psychotic disorder for at least one year, were monitored during 6 months using Positive and Negative Syndrome Scale (PANSS), Global Assessment of Functioning (GAF), Clinical Global Impressions – Severity (CGI-S), Columbia-Suicide Scale for Schizophrenia (CSSRS), Calgary Depression Scale (CDS), Multiple Schizophrenia Severity Scale (MSSS), and the Extrapyramidal Symptom Rating Scale (ESRS). None of these patients presented other organic or psychiatric co-morbidity, and they were on active treatment for their multiple sclerosis throughout the 6-month duration of psychiatric evaluation. All patients were initiated on a new antipsychotic, because of the lack of efficacy of the previous agents, or due to their lack of therapeutic adherence. A patient was initiated on olanzapine 15 mg/day, while the other two received risperidone 4 mg/day. The antipsychotic doses were flexible during the 6 months of the treatment, with olanzapine between 10 and 20 mg daily, and risperidone between 3–6 mg daily. The initial PANSS mean score was 92.2, with a GAF of 35.3 and a CGI-S of 5.1.

Results: All patients reached the week 24 visit of their evaluation, and the overall tolerability of the antipsychotic treatment was good. All patients had lower PANSS scores at week 24 (the mean decrease was -25.6 points compared to baseline), higher GAF scores (+27.7 points), and lower CGI-S (-2.5 points). CSSRS did not change significantly during the 6 months, the score remained at minimum value, and the CDS scores also remained constantly under 3. ESRS recorded transient increments, but at week 12 they were not significantly increased reported at the baseline values, and no corrective medication was recommended throughout the 6 months for extrapyramidal symptoms. MSSS mean score did not change significantly at week 12 compared to its baseline values.

Discussion: Atypical antipsychotics are efficient and well tolerated in patients with schizophrenia and multiple sclerosis dual diagnosis. The positive effects of atypical antipsychotics maintained during the 6 months of monitoring and they had no significant impact over the multiple sclerosis symptoms.

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