revealed significant group x response interaction (F(1,63)=7.8, p=0.001), with post-hoc independent t-test showing that patients significantly preferred safe over risky options than controls (t=2.6, p<0.05). There were no significant correlations of risky decision-making parameters with symptom ratings and cognitive functions.

Discussion: We extend previous findings of chronic samples to patients with early schizophrenia-spectrum disorder and indicate that suboptimal risky decision-making with risk-aversion preference has also been observed in the early course of illness. Further research is warranted to clarify the longitudinal change of aberrant risk-averse behavioral patterns and its relationship with prospective functional and symptom outcomes.

F63. COGNITIVE CORRELATES OF THE NEGATIVE SYMPTOMS EXPRESSIVE AND EXPERIENTIAL DEFICIT FACTORS IN PSYCHOSIS

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Background: Primary negative symptoms of schizophrenia contribute heavily to functional disability. Treatment of these symptoms continues to be a major unmet need, even when positive symptoms are controlled. Recent factor analyses of negative symptoms using the PANSS and other symptom assessments in patients with schizophrenia have identified two factors of negative symptoms: expressive and experiential deficits. These two factors most likely have very different clinical, neurocognitive and neurobiological correlates. This study examines the clinical and cognitive correlates associated with expressive and experiential deficits in a large cohort of patients with psychosis before and after computerized cognitive remediation.

Methods: This is a secondary data analysis of subjects enrolled in a cognitive remediation program for 12 weeks. One hundred fifty-one subjects age 18 - 55 with a DSM IV-TR diagnosis of schizophrenia, schizoaffective disorder or bipolar disorder were enrolled. Assessments of demographic, psychopathology (PANSS), cognition (MCBB), and daily living skills (UPSA-Brief) were conducted at baseline and endpoint. Exploratory (EFA) and confirmatory (CFA) factor analyses of PANSS items as well as Pearson’s correlations between factors, demographics, MCCB, and UPSA-Brief scores were examined at baseline and endpoint.

Results: EFA baseline PANSS data resulted in the five-factor model of the PANSS with seven items attributed to the Negative Symptom Factor (NSF: N1, blunted affect; N2, emotional withdrawal; N3, poor rapport; N4, passive social withdrawal; N6, lack of spontaneity and flow of conversation; G7, motor retardation; and G16, active social avoidance). CFA of the NSF revealed a two-factor model consisting of an Expressive Deficit (N1, N3, N6, G7), and an Experiential Deficit (N2, N4, and G16). Difference testing comparing the one-factor and two-factor models found that the two-factor model exhibited significantly better fit than the one-factor model (χ2 = 67.117, df= 1, p≤0.001; CFI = 0.92; Tucker–Lewis index TLI = 0.91; root mean square error of approximation RMSEA = 0.040; and Goodness of Fit index GFI = 0.93). There were significant correlations between the Expressive Deficit factor score and cognition: TMT-A (r=−0.259, p=0.001), BACS Symbol coding (r=−0.287, p=0.001), Category Fluency (r=−0.342, p=0.001), Hopkins Verbal Learning Test – revised (HTLV-R) (r=−0.236, p=0.05), Letter Number Sequencing (r=−0.256, p=0.001), and NAB Mazes (r=−0.409, p=0.001). The Expressive Deficit factor was also significantly correlated with the neurocognitive domains of Processing Speed (r=−0.352, p=0.001) and Reasoning/Problem Solving (r=−0.338, p=0.001). There were no significant correlations between either factor and UPSA-Brief or the MCCB cognitive composite. There were no significant correlations for change from baseline to endpoint in negative symptoms.

Discussion: Our results support the negative symptom two-factor model of Expressive Deficit and Experiential Deficit domains. Only the Expressive Deficit factor was associated with baseline deficits in Working Memory, Processing Speed, Reasoning/Problem Solving and Verbal Learning. The association of the Expressive Deficit factor with significant cognitive impairments supports a more profound neurobiological dysfunction in contrast to the Experiential Deficit factor and may represent an important treatment challenge. The relevance of these findings for the treatment of negative symptoms in schizophrenia will be discussed.

F64. DISRUPTION IN WORKING MEMORY GATING OBSERVED IN SCHIZOPHRENIA

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Background: Working memory and cognitive control deficits are hallmarks of schizophrenia. It is not known specifically how gating mechanisms that regulate memory may be disrupted in schizophrenia. Gating mechanisms determine how task-relevant information is selected into working memory while distractors are left out (input gating) and which items stored in working memory are selected for the rule or goal at hand (output gating). The current study investigated whether patients are able to perform the same cognitive control task that is able to dissociate input and output gating processes in a general population, and explored whether schizophrenia patients inappropriately use suboptimal cognitive control strategies (e.g. output gating when input gating can be used).

Methods: Patients (n=5) with schizophrenia or schizoaffective disorder were recruited from the Providence VAMC. Participants completed a computer-based cognitive control task. In this task, participants remembered a target item from a sequence of two items in order to select a response. A context (rule) cued which item was relevant to remember, and was presented first in the stimulus sequence (context first) or last (context last). On selective trials, one item in the trial was relevant. On global trials, both items in the trial were relevant.

Results: Patients were able to complete the task with minor modifications to adjust for ability to understand the task rules. Preliminary results of reaction time data suggest that patients were challenged at increased cognitive load. Patients performed poorly on trials where participants could use only an input gating strategy (selective first). Preliminary data also suggest that performance in patients tended to be slightly worse for selective first trials where the distractor was presented before the relevant item (i.e. on trials where input gating would be required to keep the distractor out of working memory).

Discussion: The current study supports the feasibility of using the cognitive control task selected to investigate gating mechanisms in the schizophrenia patient population. Preliminary data suggest disruption in the ability for patients to optimally use gating strategies and handle cognitive load. Future research will seek to reproduce these preliminary results in a larger sample, as well as compare patient performance to an age-matched control population directly. By understanding how gating mechanisms are disrupted in the patient population, we may be able to better develop therapeutic interventions such as cognitive training strategies to treat cognitive dysfunction in schizophrenia patients.

F65. NETWORK ANALYSIS OF EMPATHY, SCHIZOTYPY AND AFFECTIVE STATES IN A COLLEGE SAMPLE

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