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Research report

# Neurocognitive functioning in a group of offspring genetically at high-risk for schizophrenia in Eastern Turkey

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ABSTRACT

We assessed major cognitive domains in symptom-free children of patients with schizophrenia compared to the healthy children of parents with no psychopathology using neurocognitive tests. We hypothesized that, offspring at high-risk for schizophrenia would have significant impairment in major domains: attention, memory, verbal–linguistic ability and executive functions. Thirty symptom-free children (17-males, 13-females; intelligence quotient = 99.6 ± 13.6; age = 12.69 ± 2.32 and education = 5.8 ± 2.3 years) having a parent diagnosed with schizophrenia and 37 healthy children matched for gender (19-males, 18-females), IQ (106.05 ± 14.70), age (12.48 ± 2.58) and years of education (6.0 ± 2.5) were evaluated. The study group showed significant poor performance in cognitive domains, such as working memory (assessed with Auditory consonant trigram test), focused attention (Stroop test), attention speed (Trail making test), divided attention (Auditory consonant trigram test), executive functions (Wisconsin card sorting test), verbal fluency (Controlled word association test) and declarative memory (Rey verbal learning and Short-term memory test). However, no group differences were detected either on verbal attention (Digit span forward test) or sustained attention (TOVA, a continuous performance task); the latter as consistently reported to be a predictor of schizophrenia. In order to determine the cognitive endophenotype of schizophrenia, it seems more rational to conduct comprehensive evaluation of neurocognitive domains in well-matched groups via using sufficiently challenging tests to detect slight deficits. In addition, longitudinal studies with a larger sample size evaluating neurocognitive functions combined with genetic analysis may provide clues about explaining the genetic background of the disorder within the endophenocognitype concept and serve as new targets for early interventions.



## 1. Introduction

Schizophrenia is a neurobiological disorder, having a multifactorial etiology, including the genetic, neurodevelopmental and environmental influences [43]. In addition to well known negative and positive symptoms, deficits in neurocognitive functioning has come to be regarded as the core component of this disorder [24,43]. These deficits contribute to overall levels of dysfunction and significantly influence the functional outcome [19]. In contrast to significant improvement in positive symptoms after administering both typical [31] and atypical [23] antipsychotic medication, slight improvement in neurocognitive functions indicate that there is a relative independence between neurocognition and positive symptoms [36]. Studies emphasize that evaluating cognitive functioning in schizophrenia may shed light to the pathophysiology and treatment of this disorder.

Functional deficits in neurocognition are not only evident in frank psychosis, but also during the prodromal period of the disorder and high-risk relatives of the patients may also exhibit these deficits, which suggest that there is a genetic base. In this respect, identification of genetically transmitted abnormalities in cognition and information processing that are present even in the symptom-free biological relatives of the patients, has become the scope of interest. However, even if there is a strong support for genetic background, there are rapidly replicating evidences showing the polygenic inheritance of schizophrenia [15] which restricts the genetic studies from making firm conclusions regarding the pathophysiology of the disorder. In addition, it is difficult to find a relationship between the observable phenotype and the genetic background due to the complexity, variability and heterogeneity of clinical symptoms. It has been suggested that it may be possible to find the responsible genes that make symptom-free relatives vulnerable to schizophrenia by identifying latent trait markers [25].

Gottesman and Shields [17] suggested the concept of the endophenotype in the study of neuropsychiatric disorders to clarify classification and diagnosis as well as to foster genetic analysis.

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