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## Positive and negative thought disorder and psychopathology in childhood among subjects with adulthood schizophrenia

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### Abstract

The New York High-Risk Project (NYHRP) is a longitudinal study of offspring of parents with schizophrenia or affective disorder and normal controls. Neuropsychological deficits had been observed at about age 9 in subjects with adulthood schizophrenia. We explored whether in these subjects, early signs of clinical schizophrenia-related symptoms, such as thought disorder or behavioral abnormalities, could also be observed. *Methods:* We rated thought disorder and symptoms from videotaped interviews at age 9, using the Scale for the Assessment of Thought, Language and Communication (TLC), and the Mental Health Assessment Form (MHAF). With factor analyses we examined the structure of the ratings, and from interpretable factors, scales were assembled. MANOVAs were used to examine the effect of parental risk and adulthood psychiatric diagnosis (schizophrenia-related psychosis (SRP), major affective disorder (MAD), no disorder/other (NoDx/other)) as independent variables (IV) on thought disorder and symptoms as dependent variables. *Results:* Global, positive and negative thought disorder, and negative symptoms were significantly higher in subjects with adulthood schizophrenia-related psychosis than both comparison groups. A significant interaction between the two IVs was effective with respect to positive thought disorder. This scale was particularly elevated among subjects with adulthood schizophrenia-related psychosis at parental risk for affective disorder (all of whom had adulthood schizoaffective disorder). *Conclusions:* We were able to show that global, negative and positive thought disorder and negative symptoms were present in subjects with adulthood schizophrenia already at mid-childhood, years before onset of psychosis. Further, we found a particularly high propensity to positive symptoms in subjects with adulthood schizophrenia who have also an affective component in their symptoms. This association, previously reported in acute schizophrenia, was here observed years before the first psychotic episode.

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Since the beginning of scientific and descriptive evaluation of schizophrenia, thought disorder, then

characterized as ‘loosening of associations’, was mentioned as one of the central characteristics of the disorder (Bleuler, 1911). Attempts to systematize the heterogeneous symptoms of schizophrenia, and to determine the disorder’s defining characteristics and their pathophysiological correlates focused on the dimensions positive (presence of abnormal phenom-

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ena) and negative (absence or deficit of normal functions). Thought disorder was first seen as part of the positive symptom complex, but factor analytic studies of symptom ratings suggested that disorganized thought is distinct from positive symptoms such as hallucinations and delusions. Moreover, thought disorder is different from positive and negative symptoms in development and course of the illness (Schultz et al., 1997; Liddle, 1987). The delineation of subtypes and the search for cognitive and biological correlates have remained unclear (O'Leary et al., 2000), and recent discussions have reevaluated a unitary, neo-Bleulerian model of schizophrenia. This model proposes that neurodevelopmental abnormalities result in misconnections of neural circuits, causing a disruption of the fluid coordination of mental activity (named: cognitive dysmetria) which is shared by all subtypes of schizophrenia (Andreasen et al., 1999). Schizophrenia-related phenomena such as negative, positive and disorganized symptoms are seen as the result of cognitive dysmetria, in that disruption of neuronal circuits can occur at various locations and thus create motor, cognitive, and affective symptoms (Middleton and Strick, 2000).

Thought disorder has regained scientific interest in the context of the neurodevelopmental hypothesis (Weinberger, 1995; Asarnow, 1999). The presence of thought disorder already at onset and over the course of the disorder, independent from acute psychotic episodes (Marengo and Harrow, 1997; Schultz et al., 1997; Asarnow, 1999), suggests a connection to the core pathophysiology of schizophrenia. Associations of thought disorder with brain dysfunction or anatomical abnormalities are suggested by an abnormal shift in handedness (Satz and Green, 1999), deficits in verbal fluency and semantic priming (Goldberg et al., 1998; Aloia et al., 1998; Mesure et al., 1998), abnormal activity of *N*-methyl-D-aspartic acid receptors (Adler et al., 1999) and distinct abnormalities in regional cerebral blood flow (Sabri et al., 1997; McGuire et al., 1998) or choroid plexus calcification (Bersani et al., 1999). Familial occurrence of thought disorder, evidenced among first degree relatives of patients with schizophrenia-spectrum disorders (Shenton et al., 1989; Thompson et al., 1997; Arbelle et al., 1997; Docherty and Gordinier, 1999; Kinney et al., 1997) as well as in twin studies (Gambini et al., 1997; Docherty and Gottesman, 2000), indicates a genetic component rather than rearing experiences in families with schiz-

ophrenia. In summary, thought disorder is most likely genetically induced, early and persistent in the course of schizophrenia and related to anatomical and physiological abnormalities, thus, at the very core of the disorder.

We used data of participants of the New York High-Risk Project (NYHRP), a longitudinal study of subjects at risk for schizophrenia, to examine if thought disorder could be detected in individuals with adulthood schizophrenia-related psychoses at about age 9. Evidence of the presence of thought disorder years before the onset of psychosis would confirm that in the development of schizophrenia, thought disorder is different from psychosis, which is increasingly viewed as a sign of exacerbation of mental disorder in general, and not specific to schizophrenia (Tsuang et al., 2000). Previous analyses of the NYHRP have focused on comparisons among parental groups to identify possible indicators of the genetic liability to schizophrenia. Comparisons among adulthood psychiatric outcomes served to investigate precursors or early symptoms in offspring who as adults developed schizophrenia. Offspring at risk for schizophrenia had deficits in attentional, cognitive, and neuromotor measures in childhood compared with offspring of the other parental groups (for a summary, see Erlenmeyer-Kimling et al., 1998), and deficits in social competence and thought disorder at early and mid-adolescence and early adulthood (Dworkin et al., 1990, 1991, 1993). Clinical signs such as negative symptoms and multiple schizotypal features were observed in offspring of parents with schizophrenia at adolescence and early adulthood (Squires-Wheeler et al., 1992, 1997). Comparisons among groups with different adulthood psychiatric outcomes showed that subjects with schizophrenia-related disorders exhibited already at age 9 a tendency to lower IQ (Ott et al., 1998), increased behavior problems (Amminger et al., 1998) and impairments in attention, memory and gross motor skills (Erlenmeyer-Kimling et al., 2000). Some of the analyses (Dworkin et al., 1990; Squires-Wheeler et al., 1992) were based on data rated from videotapes produced at adolescence. For the study presented here, we used videotapes taken at childhood, years before the first psychotic break down, to investigate whether subjects with adulthood schizophrenia-related disorders, who had shown deficits in general functions at that age, also exhibited symptoms more closely related to schizophrenia.

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