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Theory of mind and verbal working memory deficits in parents of autistic children

Sezen Gokcen [a], Emre Bora [b,*], Serpil Erermis [a], Hande Kesikci [a], Cahide Aydin [a]

[a] Department of Child and Adolescent Psychiatry in Ege University School of Medicine, Izmir, Turkey
[b] Melbourne Neuropsychiatry Centre, University of Melbourne, Victoria, Australia

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Abstract

The objective of this study was to investigate the potential values of executive function and social cognition deficits as endophenotypes of autism. While theory of mind (ToM) is generally accepted as a unitary concept, some have suggested that ToM may be separated into two components (mental state reasoning and decoding). In this study, both aspects of ToM and verbal working memory abilities were investigated with relatively demanding tasks. The authors used a neurocognitive battery to compare the executive function and social cognition skills of 76 parents of autistic probands with 41 parents of healthy children. Both groups were matched for IQ, age and gender. Index parents had verbal working memory deficits. They had also low performance on a mental state reasoning task. Index parents had difficulties in reasoning about others' emotions. In contrast to findings in the control group, low performance of mental state reasoning ability was not associated with working memory deficit in index parents. Social cognition and working memory impairments may represent potential endophenotypes, related to an underlying vulnerability for autistic spectrum disorders.

Keywords: Autism; Executive function; Working memory; Theory of mind

1. Introduction

Autistic disorder, Asperger syndrome (AS) and pervasive developmental disorder not otherwise specified are the most common disorders believed to lie along a severity continuum in the spectrum of autistic conditions. These disorders seem to share common genetic susceptibility factors (Pickles et al., 2000). Indeed, these genetic factors can also be manifested in the characteristics of "unaffected" family members of probands with autism. Previous studies have shown that relatives of individuals with autism exhibit higher than normal rates of subtle autism-related personality, social and language features, which are referred to as the "broader autism phenotype" (Bolton et al., 1994; Piven, 2001; Dawson et al., 2002; Constantino et al., 2006).

Despite these findings, no specific genetic factor for autistic disorder has definitely been identified. Heterogeneity and complex phenotypes of psychiatric disorders are among the major challenging issues in genetic studies. A different strategy for overcoming these difficulties is the investigation of pre-behavioural phenotypes that should

* Corresponding author. Melbourne Neuropsychiatry Centre, University of Melbourne, Alan Gilbert Building, NNF level 3, Carlton, Victoria, Australia.
E-mail address: emrebora@hotmail.com (E. Bora).

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also be found in unaffected relatives of patients with psychiatric disorders. These phenotypes are commonly called endophenotypes and include electrophysiological, cognitive and neuroanatomical processes. Since *endophenotypes* are thought to reflect more basic components of complex mental disorders, they may be more closely related to underlying etiological processes than to clinical symptoms. This approach may help to fill the gap in understanding the causal link between complex behavioural manifestation (as in autistic disorder) and genes, and may be essential to discover the genetic susceptibility factors in this disorder (Gottesman and Gould, 2003). Although the endophenotype research for developmental psychopathologies is in its early stages, it holds a promise for clarifying the gene–brain–behaviour pathways for autistic spectrum disorders (Viding and Blakemore, 2007).

Since there is considerable evidence for executive (Ozonoff et al., 1991; Bennetto et al., 1996; Hill, 2004) and social-cognitive dysfunctions (Baron-Cohen et al., 1985) in autistic patients, these cognitive abilities are among the potential endophenotypes of autism. The executive function theory of autism assumes that a form of frontal pathology leading to deficits in working memory (WM) or other abilities such as the inability to shift attention and cognitive inflexibility may be essential in explaining symptoms of the disorder. A limited number of studies have reported deficits in some aspects of executive functions (EF), including cognitive flexibility and spatial working memory in relatives of autistic patients (Ozonoff et al., 1993; Hughes et al., 1997; Piven and Palmer, 1997; Koczat et al., 2002). EF deficits seen in relatives of autistic patients have been most consistently reported with the Tower of Hanoi test (Ozonoff et al., 1993; Hughes et al., 1997; Piven and Palmer, 1997) and to a lesser extent with some cognitive flexibility tasks including the Extradimensional/Intradimensional Shifting Test (Hughes et al., 1997; Wong et al., 2006). Spatial WM deficits seen in relatives of patients with autistic spectrum disorders have also been reported (Koczat et al., 2002). As far as we know, evidence for verbal WM dysfunction in relatives of autistic patients has not been reported yet. Since WM is also considered to be a key component of the other EF abilities, WM deficit may have the potential to explain the relationship between other EF abilities and autism. However, some negative findings in patients with autistic spectrum diagnoses cast doubt on the importance of WM dysfunction as a core feature of the disease (Williams et al., 2005). We believe that these inconsistencies are due to task difficulty; WM tasks that place greater demands on EF may be more suitable to demonstrate WM dysfunction in autism-related conditions.

One well-known area of research in autism involves the theory of mind (ToM) deficit hypothesis (Baron-Cohen et al., 1985). Investigators have proposed that deficits in a component of social cognition may explain many symptoms of autism. Although ToM deficits have been extensively studied in autistic patients, ToM abilities have not been adequately studied in relatives of autistic patients. Furthermore, ToM ability has been investigated as a unitary skill in most of the previous studies. It has been suggested, however, that the ToM concept is composed of two components: mental state decoding (social-perceptual) and mental state reasoning (social-cognitive) (Tager-Flusberg and Sullivan, 2000; Sabbagh, 2004). The mental state decoding component involves the ability to perceive mental states of others based on observable information such as facial expressions or gestures. The mental state reasoning component involves the ability to integrate contextual and historical information about a person (attitudes, knowledge, experiences) to understand behaviour. Previous findings suggest that different aspects of ToM depend on different social brain networks (Sabbagh, 2004). While orbitofrontal cortex and temporal cortex activation may be related to social-perceptual abilities, the medial frontal cortex seems to be critical for the ability to reason about others' mental states.

Since the two aspects of ToM rely on different neural networks, potential endophenotypes of these two aspects should be investigated separately. Several studies have shown that relatives of patients with autistic spectrum disorders seem to fail in the mental state decoding aspect of ToM. While relatives of autistic patients have been reported not to be different from control subjects in basic emotion recognition tasks, Dorris et al. (2004) and Baron-Cohen and Hammer (1997) reported a deficit in one task of social cognition targeting automatic recognition of complex states of mind, called the Eyes task, in parents and siblings of individuals with autistic spectrum disorders. Although the second aspect of ToM, mental state reasoning ability, has been shown to be severely impaired in autistic patients (failure to pass false belief paradigms), this aspect of social cognition has not been adequately studied in relatives of autistic patients. Ozonoff et al. (1993) reported that relatives of autistic patients do not have a deficit in false belief tasks, but this task may be extremely easy for high functioning populations and even for many high functioning autistic/autistic spectrum patients. Several advanced ToM tests have been developed (e.g., the Strange Stories Test; Rajendran and Mitchell, 2007), and these include tasks that may be suitable for research on endophenotypes.

While domain-specific theories suggest that ToM abilities are independent from executive functions,