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Disordered cortical connectivity underlies the executive function deficits in children with autism spectrum disorders

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ABSTRACT

The present study examined the executive function and cortical connectivity of children with autism spectrum disorders (ASD) and investigated whether the executive function deficits exhibited by these children were differentially affected and associated with the cortical connectivity. The present study compared high-functioning (HFA) and low-functioning (LFA) children with typically developing children (TDC) on their executive functions as measured by the Hong Kong List Learning Test, D2 Test of Concentration, Five Point Test, Children's Color Trail Test, Tower of California Test, and Go/No-Go task and neural connectivity as measured by theta coherence in the distributed fronto-parietal network. Thirty-eight children with ASD (19 HFA and 19 LFA) and 28 TDC children, aged 8–17 years, participated voluntarily in the study. The results on executive function showed that the LFA group demonstrated the poorest performance as exhibited by their Executive Composite and individual executive function scores, while the TDC group exhibited the highest. These results have extended the findings of previous studies in demonstrating that HFA and LFA children have significant differences in their degree of executive function deficits. The results on neural connectivity also showed that children with ASD demonstrated a different pattern of electroencephalography (EEG) coherence from TDC children, as demonstrated by the significantly elevated theta coherence in the fronto-parietal network, and that the severity of executive dysfunction between high- and low-functioning children with ASD was found to be associated with the disordered neural connectivity in these children.

What this paper adds?

The present findings have shed light on the neurophysiological underpinnings of the cognitive impairments and behavioral symptoms associated with autism, which will be invaluable for future research into the early identification, assessment, and design of teaching and remedial interventions for young children with autism.

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1. Introduction

Autistic spectrum disorders (ASD) consist of a spectrum of neurodevelopmental disorders that are characterized by disturbances in communication, poor social skills, and restricted/stereotyped behaviors or interests (American Psychiatric Association, 2002). The prevalence of ASD is high, with recent statistics reported by the Centers for Disease Control and Prevention (CDC) at one in 68, making ASD one of the most prevalent childhood developmental disorders. Variability in the degree of language impairment, intellectual ability, and symptom severity was found among individuals with ASD, ranging from severe intellectual disability to isolated cognitive problems, such as stereotypic behavior and difficulty in understanding others feelings (Baron-Cohen, 2001; Dennis et al., 1999; Happe, 1999). High-functioning autism is at one end of the ASD spectrum, with less severe signs and symptoms than other forms of autism. Although these children have average intelligence, school and daily activities can still be challenges of great magnitude. One reason for this is that these children may still show many of the key characteristics of the disorder. They often appear rigid and inflexible, show a strong liking for repetitive behavior and elaborate rituals, and have great difficulty understanding abstract uses of language, such as humor. Particularly detrimental are the presence of restricted and repetitive stereotypic behaviors and uncontrollable temper outbursts over trivial changes in the environment. Although the exact cognitive profile and underlying basis of cognitive processing in autism is not well understood, it has been suggested that fundamental to these cognitive and behavioral problems is a deficiency in executive function (Gilotty, Kenworthy, Sirian, Black, & Wagner, 2002; Ozonoff, 1997).

Executive functions refer to a broadly defined cognitive domain that includes a multidimensional set of abilities required to perform complex behaviors for the attainment of a future goal (Donders, 2002; Nyden, Gillber, Hjelmquist, Heiman, 1999) and are thought to be involved in cognitive processes, such as planning, organization, self-monitoring, cognitive flexibility, and the inhibition of inappropriate actions (Ozonoff et al., 2004). Individuals with ASD have been found to exhibit executive dysfunctions, including disorganized actions and strategies typified by decreased initiative, perseveration, difficulties in forming novel concepts, and the lack of inhibition of inappropriate actions (Bennetto, Pennington & Rogers, 1996; Ozonoff & Jensen, 1999). Neuropsychological studies of executive functions in autism have been inconclusive, however, with some reporting deficits in planning and cognitive flexibility, but not inhibition (Ozonoff & Jensen, 1999), while others have suggested difficulties in response inhibitory control and slow information processing (Schmitz et al., 2006). Some researchers have posited that individuals with ASD may have distinct primary deficits that underlie their executive dysfunctions and have suggested that one of the factors affecting the different cognitive domains of executive functioning in autism is the deficient ability to integrate information across contexts (Klinger, Klinger, & Pohlig, 2000). Specifically, individuals with autism have difficulty learning the relationships between different parts of stimuli, as well as perceiving relationships across multiple experiences, and they tend to compensate for the deficient executive function by memorizing visual details or individual rules from each situation that they encounter (Klinger et al., 2000; Ozonoff et al., 2004). This impaired ability to attend to and integrate information from the environment may explain the reason why children with autism tend to follow routines in precise detail and show great distress over trivial changes in the environment.

Although the exact cognitive profile and the underlying basis of executive dysfunction are uncertain, results from previous studies that reported structural, physiologic, and functional abnormalities in the frontal region of individuals with ASD (Harrison, Demaree, Shenal, & Everhart, 1998; Mundy, 2003; Ozonoff et al., 2004; Rumsey & Ernst, 2000) have widely suggested that the frontal cortex is one of the major brain regions implicated in the cognitive impairments and repetitive stereotypic behaviors commonly observed in the disorder (Schroeter, Zysset, Wahl, & von Cramon, 2004). For example, results from neurobiological studies have revealed abnormal neurobiological processes in the frontal lobes that underlie the cognitive deficits in ASD (Mundy, 2003; Ozonoff et al., 2004; Schmitz et al., 2006). Functional imaging studies have also found altered patterns of activation and glucose metabolism in various areas of the frontal lobes in individuals with ASD during neuropsychological tasks of executive function (Harmony, Alba, Marroquin, & Gonzalez-Frankenberger, 2009; Hazlett et al., 2004; Schmitz et al., 2006). In addition to the frontal lobe, increasing evidence also shows that effective executive functioning involves the integrated action of multiple brain areas of the fronto-parietal network (Osaka et al., 2004; Sauseng, Klimesch, Schabus, & Doppelmayr, 2005). For example, brain imaging studies have shown that the fronto-parietal network is involved in visuospatial working memory (McEvoy, Pellouchoud, Smith, & Gevins, 2001; Oliveri et al., 2001), and the frontal and parietal regions have been found to have increased regional blood flow during non-verbal paired-associated tasks (Klingberg, & Roland, 1998). Given that executive function relies on the frontal cortex and its distributed network to the parietal regions (Babiloni et al., 2004; Osaka et al., 2004), it has been postulated that abnormalities in this neural connectivity may account for the unusual cognitive processing and resultant behavioral symptoms of ASD (Fletcher & Henson, 2001; Rippon, Brock, Brown, & Boucher, 2007). Indeed, diffusion tensor imaging (DTI) studies of cortical connectivity have shown reduced myelin integrity in the ventromedial prefrontal cortex and at the temporo-parietal junctions in individuals with ASD (Barnea-Goraly et al., 2004; Lewis & Elman, 2008). In addition, evidence for disordered neural connectivity in ASD was also found in functional MRI studies that demonstrated that the inhibitory deficit in ASD is associated with decreased synchronization between activated brain areas in the inhibition networks in these individuals (Kana, Keller, Minshew, & Just, 2007). Similarly, electrophysiological studies in children with ASD using coherence, a quantitative electroencephalography (EEG) that measures the level of synchrony or cortical connectivity between brain areas in response to cognitive processes (Coben, Clarke, Hudspeth, & Barry, 2008; Rippon et al., 2007; Thatcher, North, & Biver, 2005), have also provided evidence to suggest disordered connectivity across neural systems in these children (Castelli, Frith, Happe, & Frith, 2002; Just, Cherkassky, Keller, & Minshew, 2004; Luna et al., 2002). Based on the documented executive dysfunctions and the reported neural