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A functional and structural study of emotion and face processing in children with autism

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ABSTRACT

Children with autism exhibit impairment in the processing of socioemotional information. The amygdala, a core structure centrally involved in socioemotional functioning, has been implicated in the neuropathology of autism. We collected structural and functional magnetic resonance images (MRI) in children 8 to 12 years of age with high-functioning autism ($n=12$) and typical development ($n=15$). The functional MRI experiment involved matching facial expressions and people. Volumetric analysis of the amygdala was also performed. The results showed that children with autism exhibited intact emotion matching, while showing diminished activation of the fusiform gyrus (FG) and the amygdala. Conversely, the autism group showed deficits in person matching amidst some FG and variable amygdala activation. No significant between-group differences in the volume of the left or right amygdala were found. There were associations between age, social anxiety and amygdala volume in the children with autism such that smaller volumes were generally associated with more anxiety and younger age. In summary, the data are consistent with abnormalities in circuits involved in emotion and face processing reported in studies of older subjects with autism showing reductions in amygdala activation related to emotion processing and reduced fusiform activation involved in face processing.

1. Introduction

Individuals with autism often demonstrate impaired processing of emotions (Celani et al., 1999; Macdonald et al., 1989), abnormal perception of faces (Adolphs et al., 2001; Ashwin et al., 2006; Baron-Cohen et al., 1999; Critchley et al., 2000; Dalton et al., 2005; Davies et al., 1994; Schultz et al., 2000), increased stress and anxiety (Amaral and Corbett, 2003; Corbett et al., 2006; Corbett et al., 2008; Muris et al., 1998), impaired gaze (Spezio et al., 2007) and impaired judgment of gaze direction and mental state (Courchesne, 1997). Thus, it is not surprising that the amygdala, a brain structure involved in the processing of emotions (Adolphs et al., 2002), novelty (Schwartz et al., 2003), stress (Tsigos and Chrousos, 2002), anxiety (Davis, 1992), eye gaze (Spezio et al., 2007), orienting (Moses et al., 2002; Wright et al., 2003), and empathy (Spinella, 2002; Vollm et al., 2006) would be implicated in the neuropathology of autism.

The amygdala is part of a network of brain regions that form the neural substrate for social cognition, subsequently referred to as the "social brain," which includes the amygdala, orbital frontal cortex (OFC), and the superior temporal sulcus and gyrus (STS/G) (Brothers, 1990). Individuals with autism demonstrate impairment in social cognition that includes the identification of facial expression, face recognition, discrimination of faces, and memory for faces (Adolphs et al., 2001; Adrien et al., 1991; Celani et al., 1999; Green et al., 1995; Hauck et al., 1998; Hobson et al., 1988; Macdonald et al., 1989; Yirmiya et al., 1989), although some studies do not report emotion or face processing deficits (Castelli, 2005; Hadjikhani et al., 2004). The amygdala is also important in acquisition, consolidation, and retrieval of emotional information, especially fear (Adolphs and Tranel, 1999; Aggleton, 2000; Aggleton et al., 1992; Davis, 1992; LeDoux, 1994, 1996; McGaugh et al., 1996).

There is converging evidence implicating the amygdala in the neuropathology of autism from several areas of neuroscience including postmortem (Bauman and Kemper, 1985; Kemper and Bauman, 1998; Schumann and Amaral, 2006), structural magnetic resonance imaging (MRI), and functional MRI (fMRI) studies. Volumetric studies have revealed both increased (Abell et al., 1999; Howard et al., 2000)

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and decreased (Aylward et al., 1999; Pierce and Courchesne, 2000) amygdala volume in subjects with autism. A study of very young children with autism spectrum disorder (ASD), which includes autistic disorder, Asperger syndrome, and pervasive developmental disorder-not otherwise specified (PDD-NOS), showing bilateral amygdalar enlargement found an association between the amygdala and the core symptoms early in development (Sparks et al., 2002). Further, a longitudinal study reported that a larger right amygdala at 3 to 4 years of age was associated with poorer clinical outcome (Munson et al., 2006). Schumann reported amygdalar enlargement in 8-to-12-year-old children with ASD compared with typically developing peers (Schumann et al., 2004). Recently, positive correlations have been reported between amygdala volume and level of anxiety (Juranek et al., 2006) and social impairment (Nacewicz et al., 2006) in ASD. Similar volumetric differences have also been observed in the unaffected siblings of children with autism (Dalton et al., 2007). These data suggest that discrepancies between increased or decreased amygdala volume might be related to the age, level of clinical impairment, and extent of underlying anxiety or stress in the particular sample of subjects studied.

Functional imaging studies have also reported differences in amygdala activity between those with autism and control participants (e.g., Critchley et al., 2000). A recent fMRI study of adult males with ASD reported that activity was abnormal within the "social brain" network, with less activation of the amygdala and the OFC, and increased activity and greater reliance on the superior temporal cortex and anterior cingulate cortex (ACC) during a social perception task (Ashwin et al., 2007). Similarly, individuals with ASD showed significantly less amygdala activation than control subjects during a judgment task (Baron-Cohen et al., 1999). In an emotion matching and labeling task, children with ASD recruited different neural networks and utilized different strategies during the automatic processing of socioemotional information despite relatively unimpaired cognitive assessment of basic emotions (Wang et al., 2004). The familiarity of the stimulus also appears to be an important consideration in activation of the amygdala in adults with autism (Pierce et al., 2004). Additionally, activation in the amygdala and the fusiform gyrus have been shown to be positively associated with the time spent fixating on another's eyes in children with autism (Dalton et al., 2005) and their unaffected siblings (Dalton et al., 2007).

These data suggest that when typical subjects are carrying out tasks that require social evaluation, the amygdala is activated; however, this activation is decreased in individuals with autism. Such findings lend support for the "amygdala theory of autism" proposing that early dysfunction of the amygdala may be responsible, in part, for impairment in socioemotional functioning in autism (Baron-Cohen et al., 2000; Castelli, 2005).

In addition to processing emotion elicited by faces, face processing itself is often impaired in autism (e.g., Critchley et al., 2000; Pierce et al., 2001; Piggot et al., 2004; Schultz et al., 2000; Wang et al., 2004) with some exceptions (Hadjikhani et al., 2004). Face perception is mediated by a distributed cortical network that includes the FG, an extrastriate visual cortical region located in the inferior temporal lobe identified as being selective both for faces (Allison et al., 1994; Haxby et al., 1994; Kanwisher et al., 1997; Kanwisher et al., 1999; McCarthy et al., 1997; Sergent et al., 1992) and for a variety of non-face object classes in which one makes a subordinate level judgment and has obtained a level of perceptual expertise (Gauthier et al., 2000; Kanwisher et al., 1997; Kanwisher et al., 1999).

The aforementioned studies provide the rationale for evaluating the role of the amygdala and fusiform regions in autism using functional and structural MRI. However, many of the studies investigated a heterogeneous sample of individuals across a broad autism spectrum and age span. For the present fMRI study, a homogeneous sample of children with autism and a narrow age range were employed. Due to the mixed results in previous studies of emotion perception (Baron-Cohen et al., 1999; Castelli, 2005; Hobson, 1986; Hobson et al., 1988; Howard et al., 2000), and the rather simple nature of the matching task, it was hypothesized that children with autism would show a comparable performance to the children with typical development. However, as in Schultz et al. (2000), we hypothesized that children with autism would demonstrate more difficulty with face perception. We predicted that the amygdala would show decreased activation to explicit emotion processing. We expected reduced fusiform activation to facial stimuli in autism. In regards to the volume of the amygdala, we predicted that children with autism would show bilateral amygdala enlargement, which would further be correlated with age, anxiety and social functioning.

2. Methods

2.1. Experiment 1: fMRI investigation

2.1.1. Participants

Two groups of children, 8-to-12 years of age, participated in this study: 12 with high-functioning autism and 15 with typical development. The demographic information for the groups is presented in Table 1. Despite the subjects being of average intelligence, independent samples *t*-tests revealed a significant IQ difference between the groups, based on the Wechsler Abbreviated Intelligence Scale (Wechsler, 1999), $t(25) = 4.89$, $P < 0.001$.

Inclusion criteria for all participants consisted of having an IQ > 80, and an absence of fragile X or other serious neurological, psychiatric, or medical conditions. The majority of the diagnostic participants were recruited from the University of California, Davis M.I.N.D. (Medical Investigation of Neurodevelopmental Disorders) Institute Subject Tracking System (STS) and already had a confirmed diagnosis from the Autism Diagnostic Observation Schedule (ADOS) (Lord et al., 1999) and the Autism Diagnostic Interview-Revised (Lord et al., 1994). For children who were not already evaluated ($N = 3$), the following diagnostic procedures were conducted. A strict diagnosis of autistic disorder was based on DSM-IV criteria (American Psychiatric Association, 1994) and established by all of the following: 1) a previous diagnosis of autism by either a psychologist, psychiatrist or behavioral pediatrician with autism expertise, 2) an extensive clinical interview, and 3) confirmation of current autism symptoms by the ADOS Module 3 (Lord et al., 1999). To obtain a more homogeneous sample, only children with autistic disorder were enrolled.

Some research participants responded to announcements placed in various schools, recreational facilities and websites. The University of California, Davis Institutional Review Board (IRB) approved the study. Prior to inclusion, the child's parent completed written informed consent and the child assented to participate in the study.

Table 1
Demographics.

GROUP	*N*	Age	Gender	IQ	SCQ	MASC-T	MASC-SA
Autism	12	9.01 (1.60)	12 Male 0 Female	90.71 (13.82)	22.43 (5.92)	42.57 (17.50)	9.36 (5.95)
Typical	15	9.17 (1.44)	13 Male 2 Female	115.73 (15.76)	3.08 (3.62)	37.73 (10.34)	9.20 (4.42)
	27	$t = -0.34$	$\chi^2 = -0.53$	$t = 4.89$**	$t = -10.15$*	$t = -1.07$	$t = -0.08$

Note: *$P < 0.05$, **$P < 0.01$. IQ = Intelligence Quotient based on the WASI (Wechsler, 1999), broad average range from 85 to 115; SCQ = Social Communication Questionnaire (Rutter et al., 2003), scores > 15 are suggestive of autism; MASC = Multidimensional Anxiety Scale for Children (MASC; March et al., 1997), MASC-T = Total score; MASC-SA = Social Anxiety score.