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Lack of developmental improvement on a face memory task during adolescence in autism

Kirsten O'Hearn¹, Elizabeth Schroer¹, Nancy Minshew^{1,2}, and Beatriz Luna¹

¹Department of Psychiatry, University of Pittsburgh

²Department of Neurology, University of Pittsburgh

Abstract

Autism Spectrum Disorders (ASD) are associated with abnormalities in face memory, which evidence suggests has a protracted development through adolescence. The development of face memory in people with and without ASD, from 9 to 29 years old, was examined using the Cambridge Face Memory Test (CFMT). Results indicate that the developmental improvement evident from adolescence to adulthood typically was not apparent in individuals with ASD. While children and adolescents with ASD performed similarly to typically developing individuals comparable in age and IQ, adults with ASD displayed limitations on the CFMT. The pattern of performance was constant across conditions despite differences in the timing of the presentation and delay. This atypical development in ASD is consistent with the view that the processing of complex visual stimuli continues to develop through adolescence, along with the function and structure of the temporal lobes, but that this process is disrupted in ASD. This result underscores the importance of characterizing adolescent development for understanding ASD, and suggests additional opportunities for intervention.

Keywords

development; developmental disorders; frontal; temporal; social cognition

Introduction

Autism Spectrum Disorders (ASD) affect not just the starting point of development but also how development unfolds (Minshew et al., 2004). Such differences in the course of development may be more striking for skills that have a protracted development through adolescence. The current paper examines the developmental trajectory of one such skill, face memory, in individuals with and without ASD. In this paper, face memory refers to recognizing unfamiliar faces. While this task is complex and engages multiple processes (attention, perception, memory, and matching), it also has high ecological validity, approximating how we interact with faces in the environment. Face memory has been reported to be impaired in high functioning children and adults with ASD (Faja et al., 2009; Scherf et al., 2008a; Wallace

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Corresponding author: Kirsten O'Hearn Laboratory of Neurocognitive Development Western Psychiatric Institute and Clinic Loeffler Building - Office 112 121 Meyran Avenue Pittsburgh, PA 15213 Phone: 412-512-7727 FAX: 412-383-8179 ohearnk@upmc.edu.

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et al., 2008; Wolf et al., 2008), but this deficit is not always evident (Sterling et al., 2008). It is also controversial whether these memory deficits are specific to faces. Impairments in face memory have been reported along with typical, or even better than typical, performance with cars and houses (Wallace et al., 2008; Wolf et al., 2008), but similar impairments have also been reported with non-faces (Behrmann et al., 2006; Scherf et al., 2008a).

Examining the development of face memory provides insight into the neural substrates of ASD, since research now indicates that the brain regions related to face representation mature late and that this maturation is impacted by ASD. Face-related activation in the fusiform face area (FFA) increases from adolescence to adulthood typically, as face memory improves (Golarai et al., 2010). However, activation specific to faces in or near FFA appears both decreased and more posterior in young adolescents with ASD compared to matched controls (Scherf et al., 2010). Structurally, Raznahan and colleagues (2009) report that cortical thinning in temporal regions occurs typically during adolescence but does not happen during adolescence in ASD. This differential development results in increased cortical thickness in adults with ASD compared to controls, could affect face recognition and other high-level visual skills. Similarly, disruption in the development of white matter in the temporal lobe is evident in ASD. A Diffusion Tensor Imaging (DTI) study suggests that white matter tracts in temporal regions continue to mature into adolescence but that these changes do not occur in ASD (Lee et al., 2007). Reduced connectivity between temporal and frontal regions, reported to be late developing typically (on a phonological task; Bitan et al., 2009), also affects face memory in adults with ASD (Koshino et al., 2008).

Thus, limitations in face memory in ASD may not be fully apparent until maturity is reached. To examine this possibility, we used the Cambridge Face Memory Test (CFMT) developed to identify relatively subtle deficits in adults with prosopagnosia (Duchaine & Nakayama, 2006). Individuals with ASD, 9 to 29 years old, individually matched to typically developing individuals in age and IQ, were tested on the CFMT. The CFMT limits the use of non-facial information and feature matching strategies, which may decrease the sensitivity of other standardized face processing tasks. Importantly, the CFMT provides increasing levels of difficulty to control for ceiling and floor effects, which often complicate developmental studies (Crookes & McKone, 2009). The CFMT also exhibits a robust inversion effect (i.e., poorer performance when faces are upside-down), indicating that it tests a skill related to upright faces. One proposal of such a skill is the use of holistic/configural information (perceptual integration of elements into a whole and/or spacing between features), aspects of which may develop late (Diamond & Carey, 1986; Mondloch et al., 2002) and be impaired in ASD (Behrmann et al., 2006).

Given the accumulating evidence of atypical maturation of brain processes in ASD, we hypothesized that, while performance on the CFMT would improve with age typically, it would not in ASD. While the use of the CFMT does not allow us to disentangle exactly *what* is developing late, or whether it is specific to face memory, it seemed likely to be sensitive to late development. Indeed, the typical developmental literature indicates that it would be difficult to pinpoint a particular element of face memory that develops late. While more traditional views suggest that late face development reflects face-specific perceptual skills/expertise coming online (Diamond & Carey, 1986; Mondloch et al., 2002), recent evidence indicates that development of face recognition reflects improvement in general cognitive and/or visual processes, which may impact a range of tasks (Crookes & McKone, 2009). In either case, differences in the development of these skills during adolescence provide insight into the limitations in visual processing and the dysmaturation of brain processes in ASD.

Methods

Participants

Participants included 34 individuals with autism spectrum disorders (ASD) (31 males) from 9 to 29 years old and 34 typically developing control subjects (30 males) matched individually to the participants with ASD on age (± 1 year in children, 2 years in adults) and IQ on the Wechsler Abbreviated Scale of Intelligence (± 15 points) (Table 1). These included children (9-12, $N=8$ /group), adolescents, (13-17, $N=12$ /group), and adults (18-29, $N=14$ /group). Individuals with ASD were recruited through the University of Pittsburgh Autism Center of Excellence Subject Core (HD# 055748). Participants were diagnosed with ASD using the structured research diagnostic instruments, namely the Autism Diagnostic Interview (ADI; Lord et al., 1994) and Autism Diagnostic Observation Schedule-G (ADOS; Lord et al., 1989), with the DSM-IV scoring algorithm. The ADI is a semi-structured interview given to parents of participants with ASD (including adults) asking about early development, communication, social interaction, and behavioral patterns. The ADOS assesses behaviors, including communication, social interaction, and play with objects. These instruments do not explicitly test visual processes but instead focus on the social, communication and repetitive behaviors diagnostic of ASD. Results were confirmed by expert clinical assessment. Individuals with PDD-NOS were excluded, as were those with full-scale IQs < 80 to avoid confounding results with general cognitive deficits. Typically developing participants were recruited through the ACE Subject Core and the Laboratory of Neurocognitive Development. Informed consent and assent was obtained from all participants or their legal guardians prior to the study, which was approved by the Institutional Review Board at University of Pittsburgh.

Stimuli

The stimuli were black and white photographs of young men, with cropped hair and neutral expressions (Figure 1). Six faces were designated as 'targets'. On each test trial, participants saw one target and two distracters. Some distracter faces were repeated, making it possible that both target and distracter faces would be familiar; however, target faces were never used as distracters or vice versa.

Procedure

The procedure began with a practice trial. The participant memorized a target face (Bart Simpson, a cartoon character) from 3 angles and then identified that face from three possible faces. After successfully answering the practice trial, participants completed the 3 conditions. In test trials, participants chose a face as the target by pressing the key corresponding to the number under the face (1, 2, or 3; chance=33.3%).

Condition 1: Same Images—Each of the 6 trials began with a study phase. The target face was presented from 3 angles (left 1/3 profile, front, right 1/3 profile) sequentially, for 3 s each. The test items, consisting of 3 faces, were then presented, one of which was identical to one view of the target face shown during the study phase. Three test trials separately examined encoding of the left 1/3 profile, front, and right 1/3 profile.

Condition 2: Novel images—Difficulty was increased by having participants recognize the faces under novel conditions, a challenge common in the real world. Stimuli in Condition 2 were identical to Condition 1 except that the test items differed from the study items in lighting, pose or both. In the study phase, participants saw all 6 target faces (from the front) for 20 s, reviewing them simultaneously. After this, 30 test trials (5/target face) – each with a target face and two distracter faces – were presented.

Condition 3: Novel images with noise—Condition 3 was identical to Condition 2, except for the addition of noise to the novel images. Gaussian noise was added because noise may require that adults rely more on configural processing, since recognizing the face on the basis of any single feature becomes more difficult with the increased variability (McKone et al., 2001). Participants again viewed all 6 faces from the front for 20 s, after which 24 test trials (4/target face) were presented, each including a novel image of a target face and 2 distracter faces.

Spatial Span

Impaired working memory processes are evident in people with ASD on the spatial span (Luna et al., 2007). To control for contributions of this particular memory impairment to the results, we administered the spatial span from the Cambridge Neuropsychological Test Automated Battery (CANTAB) via a touch-screen computer (Mitsubishi Precise Point 8705). An array of white squares in a random pattern was presented, some of which briefly and sequentially change color and then returned to white; participants remembered the location and the sequence of the squares that changed color. They then touched the squares in the sequence they remembered. The number of squares to be remembered progresses from two to nine, if the participant answers correctly on 1 of 3 trials for a given number.

An analysis of spatial span using an Analysis of Variance (ANOVA), with group (ASD, TD) and age (child, adolescent, adult) as between subjects factors, revealed a main effect of group ($F(1,60)=4.67$, $p=.04$, partial $\eta^2=.07$) and of age ($F(2,60)=3.73$, $p=.03$, partial $\eta^2=.11$) but no interaction (group $\times$ age interaction $F(2,60)=.19$, $p=.83$, partial $\eta^2=.01$). Since spatial span was impaired in ASD but developed at a similar rate to controls, this measure was used as a covariate in the ANOVAs to help mitigate the effects of this working memory difference (See Figure 2 for the data from the spatial span).

Results

Percent correct (Figure 3 and 4) was examined using repeated measures ANOVA with condition (conditions 1, 2, and 3) as a within subject factor, group (TD, ASD) and age (children, adolescents, adults) as between subjects factors, and spatial span as a covariate.¹ This ANOVA revealed main effects of group ($F(1,61)=14.68$, $p<.001$, partial $\eta^2=.19$), condition ($F(2,122)=6.07$, $p=.003$, partial $\eta^2=.09$), and age ($F(2,61)=7.59$, $p=.001$, partial $\eta^2=.20$). People with ASD performed more poorly than controls, performance deteriorated with each progressive condition, and performance improved with age. However, a group $\times$ age interaction ($F(2,61)=4.90$, $p=.01$, partial $\eta^2=.14$) indicated that group differences changed with age. Condition did not interact with other factors (group $\times$ condition, $F(2,122)=.79$, $p=.45$, partial $\eta^2=.01$; age $\times$ group $\times$ condition $F(4,122)=1.56$, $p=.19$, partial $\eta^2=.05$).

ANOVAs were done at each age separately, with condition and group as factors and spatial span as a covariate. In children, there was a main effect of condition ($F(2,26)=3.67$, $p=.04$, partial $\eta^2=.22$), but no main effect of group ($F(1,13)=.49$, $p=.50$, partial $\eta^2=.04$) nor a group $\times$ condition interaction ($F(2,26)=.61$, $p=.55$, partial $\eta^2=.05$). In adolescents, there were no significant effects (condition; $F(2,42)=1.38$, $p=.26$, partial $\eta^2=.06$; group; $F(1,21)=2.18$, $p=.16$, partial $\eta^2=.09$; group $\times$ condition; $F(2,42)=1.21$, $p=.31$, partial $\eta^2=.05$). When children and adolescents were combined, the main effect of group was still not significant ($F(1,37)=2.09$, $p=.16$, partial $\eta^2=.05$). In adults, there was a main effect of group ($F(1,25)=20.46$, $p<.001$, partial $\eta^2=.45$) but no effect of condition ($F(2,50)=2.17$, $p=.13$, partial $\eta^2=.08$) or

¹Spatial span was interpolated from means for a given age/group in 2 individuals who did not take this task due to technical difficulties.

interaction between condition and group ($F(2,50)=1.35$, $p=.27$, partial $\eta^2=.05$). Impaired performance in the group with ASD emerged with adulthood.

Development was examined in each group separately. Typically developing individuals exhibited main effects of condition ($F(2, 62)=139.15$, $p<.001$, partial $\eta^2=.82$) and age ($F(2,31)=16.42$, $p<.001$, partial $\eta^2=.51$), but the interaction between condition and age did not reach significance ($F(4,62)=2.45$, $p=.06$, partial $\eta^2=.14$). Post-hoc analyses (Tamhane's) revealed that adults were better than children ($p<.001$) and adolescents ($p=.002$); children and adolescents did not differ ($p=.15$). In the group with ASD, there was a main effect of condition ($F(2,62)=68.60$, $p<.001$, partial $\eta^2=.70$) but no main effect of age ($F(2,31)=.32$, $p=.73$, partial $\eta^2=.02$; all p 's $>.79$) nor a condition $\times$ age interaction ($F(4,62)=.26$, $p=.90$, partial $\eta^2=.14$).

A similar pattern was evident with correlations examining the association between age (as a continuous factor) and performance: a significant relation in typical participants (Condition 1: $r^2(34)=.56$, $p=.001$; Condition 2: $r^2(34)=.61$, $p<.001$; Condition 3: $r^2(34)=.61$, $p<.001$) that was not evident in participants with ASD (Condition 1: $r^2(34)=-.01$, $p=.96$; Condition 2: $r^2(34)=-.09$, $p=.61$; Condition 3: $r^2(34)=-.09$, $p=.61$). A z-score transformation comparing the correlation coefficients between the two groups found that the relation between age and performance (i.e., the slope) differed between groups in all conditions, with the ASD group showing a flatter slope (all p 's $<.02$).

Discussion

The CMFT task was used to characterize the development of face memory in individuals with and without ASD. Results indicate that performance on this face memory task continues to improve until adulthood typically but not in individuals with ASD similar in age and IQ, leading to impairments in adulthood. The level of deficit documented in the adults with ASD did not differ from that reported in adults with congenital prosopagnosia, while the typically developing adults did not differ from previously reported controls (Table 2: Duchaine & Nakayama, 2006). This result is consistent with evidence showing a lack of maturation in the function and structure of temporal lobe regions in ASD (Lee et al., 2007; Raznahan et al., 2009; Scherf et al., 2010). It is also consistent with behavioral evidence from potentially related visual tasks -- interpreting facial expression (Rump et al., 2009) and configural processing (Scherf et al., 2008b)² -- suggesting that the lack of adolescent development may not be specific to faces, but instead affect visual processes more generally (e.g., configural/holistic processing; visual memory). However, systematic study must be done to identify exactly *what* does not develop in adolescents with ASD, including a careful examination of specific visual and memory components that may be affected.

It is unclear why our results differ from studies reporting impaired face memory in children with ASD with similar samples (Scherf et al., 2008a; Wolf et al., 2008). The current results do not simply reflect floor effects, as children performed above chance, and the pattern was similar across increasing levels of difficulty. One possibility is that, given the difficulty of the CFMT, typical children used immature strategies (e.g., feature-based) resulting in comparable performance between groups at younger ages. Alternatively, the deficits reported previously in children with ASD and the ones documented here in adulthood could have distinct etiologies. For example, deficits specific to faces, or related to how the face is examined, could be evident early in childhood and beyond. However, on the CFMT, individuals with ASD may have been able to compensate for these difference because they had more time, and multiple angles, to

²However, configural processing in a general sense may not be the main reason for these results, since the deficit was not *more* evident in Condition 3, in which additional noise may require participants to rely more heavily on configural information because local information is less reliable (McKone et al., 2001).

examine the target faces, compared to the previous studies that showed differences in childhood (9 s in Condition 1, compared to 250 ms in Scherf et al., 2008a and 1 s in Wolf et al., 2008). In contrast, the differences that emerge in our study may reflect a different source, perhaps a more general developmental difference. Crookes and McKone (2009) argue that measures of explicit memory --like the task used here-- require substantial domain-general development. These limitations in ASD may interact, as early visual experience affects the emergence of configural face processing skills during adolescence (Maurer et al., 2007). While speculative, these possibilities highlight that age may interact with task demands, making differences in ASD more or less evident.

The lack of late improvement is consistent with the view that ASD involves a disrupted developmental trajectory of brain processes (Minshew et al., 2004). Studies have indicated early acceleration of brain growth in infants with ASD, followed by an apparent arrest during early childhood, resulting in normative gray matter volume (Redcay & Courchesne, 2005). Additional measures have identified a second period of dysmaturation; cortical thinning and white matter maturation that occurs typically during adolescence may not occur in ASD (Lee et al., 2007; Raznahan et al., 2009). This leads to abnormalities that are still evident in adulthood in ASD, including reduced white matter integrity that may undermine late developments in functional connectivity (Bitan et al., 2009). This paper reports on the impaired developmental trajectory of visual behavior in ASD *through adolescence*, supporting that the development of brain processes in ASD is atypical into adulthood. The late appearing deficits in face memory in ASD reported here underscore that adolescence may an important period for both study and intervention in ASD.

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ABBREVIATIONS

ADI	Autism Diagnostic Interview
ADOS	Autism Diagnostic Observation Schedule
WASI	Wechsler Abbreviated Scale of Intelligence
CFMT	Cambridge Face Memory Test

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Figure 1.

a) Trial from Condition 1. Participants studied the target from three angles, and then identified it from each angle, one at a time. b) Study stimuli for Conditions 2 and 3. Participants reviewed target faces simultaneously for 20 s. c) Test stimuli for Condition 2, with target faces in a novel pose and/or lighting. d) Test stimuli for Condition 3, with target faces in a novel pose and/or lighting, with added noise.

Figure 2.

a) Spatial span at each age. Dashed lines represent typical development and open circles illustrate individual performance. Solid lines represent development in ASD and solid black circles illustrate individual performance in ASD.

A Performance averaged across conditions

B Performance in Condition 1

C Performance in Condition 2

D Performance in Condition 3

Figure 3.

a) CFMT performance at each age. Dashed lines represent typical development and open circles illustrate individual performance. Solid lines represent development in ASD and solid black circles illustrate individual performance in ASD. b) Condition 1. c) Condition 2. d) Condition 3.

Figure 4.

a) Overall percent correct at each age. Lines represent typical development (dashed lines, open circles) and development in ASD (solid lines, solid circles). b) Condition 1. c) Condition 2. d) Condition 3.

Table 1

Demographic information

Variable	Children						Adolescents						Adults					
	ASD			Control			ASD			Control			ASD			Control		
	M	SD	N	M	SD	N	M	SD	N	M	SD	N	M	SD	N	M	SD	N
N	8		8				12		12				14		14			
Age	11.57	1.11		11.55	1.09		15.35	1.77		15.39	2.02		23.09	4.54		22.73	4.45	
Full scale IQ	100.25	6.69		105.62	9.74		105.08	14.26		105	9.25		107.43	12.51		105.43	10.63	
Verbal IQ	104.25	13.25		104.5	9.26		104.58	12.97		103.75	9.30		106.29	11.22		103.5	10.35	
Performance IQ	96	6.21		105.38	10.72		103.92	15.87		106.42	9.70		106.79	13.42		106.42	11.46	
ADOS																		
Communication	4.67	1.86					4.45	1.13					5.15	1.22				
Social	9.50	1.76					9.09	2.38					9.23	2.17				
Total	14.17	3.31					13.55	2.77					14.03	3.12				

Table 2

CFMT performance (percent correct) in adulthood across studies

<i>N</i>	Duchaine & Nakayama (2006)				Current study			
	Typical Adults		Prosopagnosia		Typical Adults		ASD	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Condition 1	99.0	2.4	85.1	12.3	98.9	2.4	78.6	21.8
Condition 2	79.1	14.4	47.2	13.1	76.2	15.2	47.4	14.2
Condition 3	68.2	16.8	36.5	12.9	67.7	17.5	44.9	15.5
Total	80.4	11.0	50.7	10.4	78.7	12.0	54.4	14.1