Perception of dynamic changes in facial affect and identity in autism

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Despite elegant behavioral descriptions of abnormalities for processing emotional facial expressions and biological motion in autism, identification of the neural mechanisms underlying these abnormalities remains a critical and largely unmet challenge. We compared brain activity with dynamic and static facial expressions in participants with and without high-functioning autism using event-related functional magnetic resonance imaging (fMRI) and three classes of face stimuli—emotion morphs (fearful and angry), identity morphs and static images (fearful, angry and neutral). We observed reduced activity in the amygdala (AMY) and fusiform gyrus (FFG) to dynamic emotional expressions in people with autism. There was also a lack of modulation by dynamic compared with static emotional expressions of social brain regions including the AMY, posterior superior temporal sulcus (STS) region and FFG. We observed equivalent emotion and identity morph-evoked activity in participants with and without autism in a region corresponding to the expected location of the more generally motion-sensitive area MT or V5. We conclude that dysfunctions in key components of the human face processing system including the AMY, FFG and posterior STS region are present in individuals with high-functioning autism, and this dysfunction might contribute to the deficits in processing emotional facial expressions.

Keywords: autism; amygdala; emotion; face processing; fMRI

INTRODUCTION

Autism is a complex neurodevelopmental disorder characterized by severe and pervasive deficits in social functioning and communication as well as restricted, repetitive behaviors and a characteristic developmental course (APA, 2000). Affected individuals differ in the extent to which they demonstrate each of these impairments, but the core disability appears to revolve around social functioning (Kanner, 1943; Waterhouse et al., 1996). The ability to judge emotional expressions and derive other socially relevant information from faces is fundamental to normal reciprocal social interactions and interpersonal communication. Thus, impairments in several aspects of face processing are particularly the striking features of the autism phenotype. For example, children with autism score lower on face recognition tests than do developmentally delayed children (Klin et al., 1999). Individuals with autism do not spontaneously look at faces in the same way as do typically developing individuals: studies of visual scanpaths indicate that they spend less time looking at the eyes (Klin et al., 2002; Pelphrey et al., 2002). Affected individuals also have difficulty identifying emotional characteristics of posed facial expressions (Hobson, 1986a, b; Celani et al., 1999; Adolphs et al., 2001; Pelphrey et al., 2002). The visual scanpaths and emotion recognition deficits in autism are similar to those seen in neurological patients with amygdala (AMY) damage. Both populations are impaired at judging faces displaying fear (Adolphs et al., 2001), and both populations fail to spontaneously examine eyes (Klin et al., 2002; Pelphrey et al., 2002; Adolphs et al., 2005; Dalton et al., 2005).

Functional neuroimaging studies of people with autism have reported abnormal activity in the components of the human face processing system, including the fusiform gyrus (FFG) (Critchley et al., 2000; Schultz et al., 2000; Pierce et al., 2001; Hubl et al., 2003; Pierce et al., 2004; Piggot et al., 2004; Wang et al., 2004; Dalton et al., 2005), posterior superior temporal sulcus (STS) region (Castelli et al., 2002; Pelphrey et al., 2005) and AMY (Baron-Cohen et al., 1999; Critchley et al., 2000; Ogai et al., 2003; Wang et al., 2004; Dalton et al., 2005). All but one study (Dalton et al., 2005), has reported AMY hypoactivation. This neural network is hypothesized to be important for linking the structural encoding of facial features with emotional expressions and biological motion and is part of the neural circuitry underlying the broader ‘social brain’ (Brothers, 1990). However, one limitation of
the existing neuroimaging literature on face processing in autism is the common use of static, posed snapshots of faces. These stimuli might be less effective in eliciting physiological reactions and do not contain the dynamic facial information typically displayed in human social interactions. An important function of facial expressions is the social communication of changes in affective states. The dynamic perception of expressive features in healthy adults differentially recruits specialized processing resources in regions of the social brain, including the FFG, AMY and STS (LaBar et al., 2003; Sato et al., 2004), perhaps to direct appropriate actions in response to the observed sequences in facial motion.

Dynamic information may be integral to the mental representation of faces (Freyd, 1987). Behavioral studies have shown that motion information contributes to facial affects perception in development (Soken and Pick, 1992; Soken and Pick, 1999) and facilitates expression and identity judgments in healthy adults (Hess et al., 1992; Lander et al., 1999; Lander et al., 2004) as well as in blindsight (de Gelder et al., 1999), prosopagnosia (Humphreys et al., 1993) and mental retardation (Norwood et al., 1999). Individuals with autism show deficits in processing biological motion from point-light displays (Blake et al., 2003). However, they appear to benefit from slow dynamic information when categorizing emotional expressions (Gepner et al., 2001).

In the present study, we sought to characterize the neural circuitry associated with the dynamic perception of facial affect and identity in individuals with and without autism. Building upon a prior study in neurologically normal adults (LaBar et al., 2003), we compared brain activity with dynamic and static facial expressions in adolescents and adults with and without high-functioning autism using an event-related functional magnetic resonance imaging (fMRI) design and three classes of face stimuli—emotion morphs, identity morphs and static images. Static images depicted prototypical fearful, angry and neutral expressions. Identity morphs depicted shifts from one person’s face to another, always with neutral expressions. Emotion morphs depicted expression changes from neutral to fear or anger, creating the illusion that the actor was ‘getting scared’ or ‘getting angry’ in real time. Based on our previous fMRI studies of face processing and biological motion, and the existing fMRI literature in autism, we hypothesized that the AMY would show hypoactivation to the emotion morphs in participants with autism. We further predicted that the AMY, STS and FFG would not show differential activation to the emotion morphs compared with the static emotions, perhaps reflecting a lack of modulation of social brain structures by motion during processing of emotional expressions in autism. In contrast, we hypothesized that both participant groups would show activation to the dynamic stimuli relative to the static images in visual motion area MT/V5. Importantly, this design allowed us to demonstrate both intact and impaired regional activation patterns in different brain structures in autism, thus enabling a specification of impairments at the neural circuit level.

**MATERIALS AND METHODS**

**Participants**

Eight right-handed participants with high-functioning autism (two females, six males; 24.5 ± 11.5 years; age range = 17.9–50.3 years) were recruited through the North Carolina Studies to Advance Autism Research and Treatment (STAAF) Center Clinical Core. Participants or their parents consented to a protocol approved by the Duke University Human Investigation Committee. Diagnoses were made by the North Carolina STAAF Center’s Clinical Core and were based on a history of a clinical diagnosis of autism, parental interview [Autism Diagnostic Interview-Revised (ADI-R); (Lord et al., 1994)] and proband assessment [Autism Diagnostic Observation Schedule (ADOS); (Lord et al., 2000)]. All subjects met cut-off criteria for autism based on their ADI-R and ADOS scores. The average (s.d.s; ranges in parentheses) intelligence quotient (IQ) scores for the autism group were: full scale = 113 (11; 83–128), verbal = 115 (14; 87–130), performance = 108 (15; 82–123). Average (s.d.s in parentheses) ADI-R algorithmic scores were 21(8) for Social Interaction, 15(5) for Communication–Verbal, 10(4) for Communication–Nonverbal, 6(3) for Restricted-Repetitive-Stereotyped Behaviors and 3(2) for Onset. For the comparison group, eight right-handed, neurologically normal participants (two females, six males; 24.1 ± 5.6 years; age range: 18.1–32.2 years) screened against major psychiatric illness, developmental disability and neurological problems were recruited from the community. The average IQ scores for the comparison group were: full scale = 120 (9; 106–132), verbal = 118 (10; 108–132), performance = 118 (9; 104–127). Analysis of variance (ANOVA) procedures confirmed that the two participant groups did not differ significantly in age or IQ.

**Stimulus development**

The stimuli used in this study were identical to those used in a previous study by members of our group (LaBar et al., 2003). Pictures were selected from the Ekman series of faces to be universally representative of fearful, angry and neutral emotions (Ekman and Friesen, 1976; Matsumoto and Ekman, 1989). Digital reproductions of the three classes of faces were used as static stimuli. The dynamic emotional stimuli were created by morphing a static neutral image with a static emotional image of the same actor throughout increasing emotional intensity. For dynamic identity stimuli, one actor’s static neutral face was morphed with that of another actor of the same gender and ethnicity. Each actor in the image set was used in the dynamic emotion, dynamic identity, static emotion and static neutral conditions; a subset of the same actors posed both fear and anger displays.
An ovoid mask was used to crop out extraneous information that could influence perception (e.g., hair, neck, ears). All of the faces chosen had a full frontal orientation to ensure that there were no changes in viewpoint. For each image, the luminance and contrast were normalized. Then, the faces were all given a uniform, mid-gray background. Morphs were created using MorphMan 2000 software (STOIK, Moscow, Russia). Approximately 150 fiducial markers were placed on each digital source image in the morph pair and individually matched to corresponding points on the target image. The markers were more densely distributed in areas that are most relevant for the detection of changes in emotion such as the eyes, mouth and corrugator and obicularis oculi muscles (Ekman and Friesen, 1978; Bassili, 1979). Morphs were presented at a rate of 30 frames/s, consistent with previous research (Thornton and Kourtzi, 2002; LaBar et al., 2003). A total duration of 1700 ms was chosen to approximate the timing of natural changes in facial affect observed in videotapes (Gepner et al., 2001). Of this, 1500 ms consisted of the transition from the first to the second face, with 43 frames interpolated between the two images to provide a smooth transformation. For the last 200 ms, the final morph frame remained visible. Morphs were saved in .avi format and displayed as movie clips. For the static displays, one still portrait was shown for 1700 ms. Figure 1 illustrates four frames of a neutral-to-fear morph along with sample static images.

**Experimental design**

Participants viewed 36 unique exemplars of each of four stimulus categories: static neutral, static emotional, dynamic neutral (identity morph) and dynamic emotional (emotion morph). Half of the emotional stimuli represented fear and half represented anger. Each exemplar was presented twice during the course of the experiment (total = 72 stimuli from each category). Stimuli were presented in a pseudorandom event-related design, participant to the constraint that no more than two exemplars of each category were presented in a row to avoid mood induction effects. Faces were separated by a central fixation cross. The intertrial interval varied between 12 and 15 s (mean 13.5 s) to allow hemodynamic and psychophysiological responses to return to baseline levels between stimulus presentations (Figure 1). The scanning session was divided into eight runs of 8 min 24 s duration. Run order was counterbalanced across participants, and no stimuli were repeated within each half-session of four runs. Stimulus presentation was controlled by CIGAL software (Voyvodic, 1999). To ensure attention to the stimuli, participants performed a simple perceptual monitoring task that was unrelated to our questions of interest. Specifically, using their right hand, they pressed a button to indicate when a face appeared on the screen. One example of each kind of stimulus was shown to the participants prior to entering the magnet to familiarize them with the stimuli.

**Imaging parameters**

Scanning was performed on a General Electric Healthcare Technologies (Waukesha, Wisconsin, USA) 1.5 Tesla LX NVi MRI scanner system equipped with 41 mT/m gradients. A quadrature birdcage radio frequency head coil was used for transmission and reception (General Electric, Waukesha, Wisconsin, USA). Sixty-eight axial images were acquired using a 3D fast SPGR sequence (TR = 500 ms; TE = 20 ms; FOV = 24 cm; image matrix = 256²; voxel size = 0.9375 × 0.9375 × 2 mm). Functional images were acquired using a gradient-recalled inward spiral pulse sequence (Guo and Song, 2003) sensitive to blood-oxygenation level-dependent (BOLD) contrast (TR = 1500 ms; TE = 30 ms; FOV = 24 cm; image matrix = 64²; α = 90°; voxel size = 3.75 × 3.75 × 4 mm; 34 axial slices). These functional imaging parameters allowed whole-brain coverage and the spiral imaging protocol facilitated recovery of signal from anterior ventral temporal and other cortical areas that can be highly susceptible to artifact (LaBar et al., 2001; Fig. 1 Depiction of experimental paradigm. Examples of static angry and neutral expressions and four frames of a fear morph are depicted. ISI, interstimulus interval.)
Wang et al., 2005), while also providing good sensitivity to changes in BOLD contrast.

**Image preprocessing**

Image preprocessing was performed using SPM 99 (Wellcome Department of Cognitive Neurology, London, UK) and custom MATLAB (Mathworks, Natick, Massachusetts, USA) scripts. The 16 participants did not have greater than a 3 mm deviation in the center of mass in the x-, y-, or z-dimensions. The temporally realigned and motion-corrected scans were normalized to the Montréal Neurologic Institute (MNI) template. The functional data were high-pass filtered and spatially smoothed with an 8 mm Gaussian kernel. These normalized and smoothed data were used in the analysis procedures described subsequently.

**Data analysis**

The primary analysis consisted of hypothesis-driven random-effects assessments of group differences in response to the conditions (i.e. main effects of group) and the generation of contrast maps presenting differences among the conditions at the peak of the hemodynamic response (HDR) within each group. Our analyses further focused on identifying predicted group differences in activation maps resulting from these contrasts (i.e. group x condition interactions). Analyses consisted of the following steps: (i) Epochs synchronized to the trial onsets (i.e. appearance of the face stimulus) and containing two images preceding (−3.0 s) and seven images following (10.5 s) the onset of the stimulus events were extracted from the continuous time series of image volumes. Epochs were segregated and averaged by condition. Across-participants average time course volumes were then computed for each stimulus condition. The average BOLD-intensity values were converted to percent signal change relative to the prestimulus baseline. (ii) For each participant, a t-statistic for each voxel on pairwise comparisons of interest and between conditions was computed. This contrast analysis was based on time course data for each condition (averaged over single trial repetitions of each condition), and the contrast was computed using the average of the three time points around the expected peak amplitude (6–9 s after stimulus onset). This resulted in a measure of the difference between the two conditions and provided a 3D statistical parametric map for each participant. The t-statistic value at a given voxel represented an estimate of the effect size of the difference between the two epoch average activity waveforms within a time window encompassing the expected peak amplitudes of the hemodynamic response. (iii) The individual participant t-statistic maps were then used in random-effects analyses across participants. Within each participant group, t-values (one derived from each participant) for each voxel in the MNI common space were tested for a significant difference from zero using a one-sample t-test. This random-effects analysis provided two whole-brain normalized maps (one for participants with autism and one for participants without autism) of significance values for each contrast. Lower P-values indicated a larger difference between a voxel’s waveform at the expected peak for each pair of conditions. The threshold for significance was set at a voxelwise uncorrected P<0.05 (two-tailed) and a spatial extent of >4 functional voxels (Forman et al., 1995). (iv) We then identified functional regions of interest within each group and compared activation patterns at the expected peak of the HDRs in the two groups using ANOVAs. Clusters of activity showing significant group x condition interactions (P<0.05) of >4 functional voxels in spatial extent were considered differentially responsive to task demands between groups. In addition to the analyses directed at identifying hypothesized group x condition interactions, we conducted analyses to identify areas of group differences in activation levels within a single condition (e.g. an independent samples t-test to identify regions of differential activation to emotion morphs).

To reduce the number of statistical comparisons and consequently the false positive rate, the results of the random-effects analyses of group differences and differences between conditions computed earlier were restricted to only those voxels in which a significant HDR was evoked by any of the conditions. For this analysis, we thresholded our activation at a false discovery rate (FDR) (Genovese et al., 2002) of 0.05 (t(7) > 3.29, for participants with autism; t(7) > 3.43, for the participants without autism). The voxels with significant HDRs were identified separately for each group in the following steps: (i) The single-trial epochs for each participant were averaged separately for each condition, and the average BOLD-intensity signal values for each voxel within the averaged epochs were converted to percent signal change relative to the prestimulus baseline. (ii) The time waveforms for each voxel were correlated with a canonical reference waveform and t-statistics were calculated for the correlation coefficients for each voxel. This procedure provided a whole-brain t-statistic map in MNI space for each of the trial types. (iii) The t-statistic maps for each participant and for each trial type were used to calculate an average t-map for the union of all trial types across participants. We then identified active voxels as those that surpassed the FDR threshold. (iv) The difference t-statistic maps computed in the random-effects analyses described earlier were masked by the results of step 3. The mask consisted of the union of the average t-maps from the two groups. Thus, the differences in HDR amplitude between conditions and within each participant group were only evaluated for those voxels in which at least one condition evoked a significant HDR in either or both participant groups.

**RESULTS**

Analyses focused on four brain regions (the AMY, STS, FFG and MT/V5) and two statistical activation contrasts...
Group differences in responses to dynamic facial expressions of emotion

We first identified group differences in activation to the emotional (anger and fear) morphs relative to a fixation baseline. As illustrated in Figure 2A, this analysis revealed hypoactivation of the right AMY in participants with autism relative to neurotypical participants, $t(14) = 2.99, P < 0.01$. The HDR waveforms from this region are presented by group in Figure 2B. In addition to this difference in the response of the AMY to dynamic facial expression of emotion, we observed other regions of neurotypical > autism activity localized to the left and right middle temporal gyri, left medial frontal gyrus, right superior frontal gyrus and left fusiform gyrus. The MNI stereotaxic coordinates of the centers of activation within clusters of neurotypical > autism voxels are presented in Table 1.

We next examined group differences in responses to emotional face morphs (fear and anger combined) as compared with static images of faces displaying fearful and angry expressions. This contrast allowed us to identify activity evoked during emotion processing in key regions of interest including the AMY, STS and FFG. We could then determine whether activity in each of these regions of the social brain was modulated by accompanying motion in individuals with autism. As illustrated in Figure 3A, in the comparison group of participants without autism, the contrast of dynamic emotional faces compared with static emotional faces strongly activated the right AMY, right posterior STS, bilateral FFG and a bilateral area posterior and inferior to the STS region localized to the left and right middle and inferior occipital and temporal gyri. The location of this last region corresponds to the expected location of the human analogue of visual motion area MT/V5 (Dumoulin et al., 2000). These results replicated the findings of LaBar et al. (2003) in their study of neurologically normal individuals.

The pattern of emotion morph > static emotion activity was markedly different in individuals with autism (Figure 3B). In this group, only a region of activation localized to the left inferior and middle occipital gyri (area MT/V5) exhibited greater activity for emotional face morphs compared with static emotional expressions. We did not observe significant clusters of emotion morph > emotion static activity in the AMY, FFG or posterior STS region in

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<td>Superior frontal gyrus</td>
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<td>Fusiform gyrus</td>
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<td>Middle temporal gyrus</td>
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$N_{vox},$ number of voxels in the ROI. The $x,$ $y$ and $z$ refer to the stereotaxic MNI coordinates of the center of activation within an ROI. R, right hemisphere; L, left hemisphere; BA, Broadman’s Area. The threshold for significance of the clusters reported here was set at a voxelwise uncorrected $P < 0.05$ (two-tailed) and a spatial extent of four functional voxels.

Table 1 Summary of observed regions of neurotypical > autism activation to emotional morphs

Fig. 2 Results from a random-effects analysis comparing participants with autism with participants without autism. (A) Red-to-yellow colormap indicates regions of significant neurotypical > autism activity evoked during observation of emotional morphs. The map is thresholded at a voxelwise uncorrected $P < 0.05$ (two-tailed) and a spatial extent of four contiguous voxels. (B) Time courses of the average BOLD signal change from the right hemisphere AMY region of neurotypical > autism activation. Bars indicate s.e.m.
These participants. The stereotaxic coordinates of the centers of activation within the displayed clusters of dynamic emotion > static emotion voxels identified in this random-effects analysis are presented by participant group in Table 2.

The average percent signal change at peak from clusters of dynamic emotion > static emotion activity in the right AMY, STS and FFG is displayed by condition and participant group in panels A–C of Figure 4. In each of these regions, participants without autism exhibited modulation of responses to emotional facial expressions by motion with more activity for dynamic compared with static facial expressions. In contrast, responses in these same regions were not modulated by motion in participants with autism. However, it is noteworthy that significant hemodynamic responses were observed in both groups of participants across the three brain regions, indicating that both groups of participants were attending to the stimuli.

Repeated-measures ANOVAs on the BOLD percentage signal change values at the peak of the hemodynamic response confirmed the predicted group (neurotypical vs autism) × condition (dynamic emotion vs static emotion) interactions for the right AMY [F(1,14) = 7.72, P < 0.01, η² = 0.36], right FFG [F(1,14) = 13.38, P < 0.01, η² = 0.49], and right posterior STS [F(1,14) = 7.27, P < 0.01, η² = 0.34]. As illustrated in Figure 4, the significant group × condition interactions in the AMY, STS and FFG indicated a lack of modulation by motion in these regions in our participants with autism. In contrast to the three aforementioned regions of the social brain, a region of activity corresponding to the expected location of visual motion area MT/V5 (Figure 2B) did not exhibit a significant group × condition interaction [F(1,14) = 0.18, P > 0.50, η² = 0.01]. This region responded more strongly to dynamic emotions than to static emotions in both groups (Figure 4D).

Next, to examine the specificity of the significant group × condition interactions described earlier for emotional morphs, we compared the responses within the AMY, FFG, STS and MT/V5 to static neutral faces and identity morphs. Repeated-measures ANOVAs on the BOLD percentage signal change values at the expected peak (6 s–9 s) of the hemodynamic response indicated no significant group (neurotypical vs autism) × condition (identity morphs vs static neutral faces) interactions for the right AMY [F(1,14) = 1.89, P > 0.05, η² = 0.12], right FFG [F(1,14) = 0.45, P > 0.05, η² = 0.03], or right posterior STS [F(1,14) = 2.74, P > 0.05, η² = 0.16], or MT/V5 [F(1,14) = 0.04, P > 0.05, η² = 0.003]. These findings indicate that the modulatory influences of face motion observed here is specific to facial expressions of emotion and not related to motion more generally.

Finally, to facilitate comparisons with prior studies that employed static facial expressions of fear and/or anger, we compared the BOLD responses to static expressions of
emotion within the right hemisphere AMY, FFG and STS ROIs. The results of this analysis revealed no differences in the right AMY $[t(14) = 0.43, P > 0.05]$ and FFG $[t(14) = -0.06, P > 0.05]$. In the right STS, participants with autism responded more strongly to the static facial expressions of emotion ($M = 0.10$ vs $0.04\%$) than did neurotypical comparison participants $t(14) = 2.87, P < 0.05$.

**DISCUSSION**

During observation of dynamic and static facial expressions of emotion, neurologically normal participants and participants with autism activated the AMY, FFG and STS in response to viewing dynamic facial expressions of emotion. However, only the neurologically normal group exhibited activity that differentiated dynamic and static facial expressions of emotion. Area MT/V5, a region of the brain that is not a core component of the human face processing system, was normally sensitive to the motion present in the dynamic facial expressions in both groups of participants. In addition, the AMY and FFG were less active in participants with autism relative to neurologically normal participants during the viewing of dynamic facial expressions of fear and anger. These results provide novel insights into dysfunctional brain mechanisms in autism that underlie detection of change in emotional states in other individuals as signaled by facial displays.

**The amygdala**

Our results regarding AMY dysfunction in individuals with autism are generally consistent with those of several prior functional neuroimaging studies. For example, Baron-Cohen et al. (1999) explored the ability of the participants to judge the mental or emotional state of a person in a photograph that only showed the eyes. Participants with autism exhibited reduced activity in the left AMY relative to neurologically normal participants. Critchley et al. (2000) compared neurologically normal individuals to those with autism on two versions of a task involving emotional faces, one requiring judgments of the expressions (an explicit emotion processing task) and the other requiring gender identification (an implicit emotion processing task). Participants with autism showed hypoactivation of the left AMY relative to neurologically normal participants during the implicit task. Wang et al. (2004) examined the modulation of AMY activity in children and adolescents with and without autism when viewing faces in order to label the emotions or match the expressions. In the participants without autism, the right and left AMY were modulated by task demands (labeling > matching); however, this was not true for those participants with autism. Ashwin et al. (2006) found that typically developing adults showed greater activation in the left AMY and left orbitofrontal cortex, while high-functioning adults with autism spectrum disorders showed greater activation in the anterior cingulate and superior temporal gyrus in response to emotionally expressive faces. Furthermore, the typically developing group but not participants with autism showed varying responses in the AMY to varying intensities of fearful expression. In the present study, we have confirmed prior

**Fig. 4** Bar graphs reflect average percent signal change and standard error by condition and group at expected peak (6–9 s post-stimulus onset) for the right hemisphere (A) amygdala, (B) fusiform gyrus, (C) superior temporal sulcus and (D) MT/V5. The error bars indicate s.e.m.s.
reports of hypoactivation of the AMY in autism in response to facial expressions of emotion, and have further demonstrated that that the AMY’s activity is not enhanced by faces containing dynamic information relative to static snapshots of the same faces in individuals with autism. However, in contrast to prior studies, we did not observe group differences in the AMY for static displays. It may be that the presence of dynamic morphs minimized the emotional impact of the static images such that the difference between individuals with and without autism was not significant.

**The superior temporal sulcus region**

The present finding regarding a lack of modulation of activity in the STS is consistent with a recent fMRI study from our group (Pelphrey et al., 2005) that implicated the STS region in the eye gaze processing deficits that are key features of the autism phenotype (e.g. Mundy et al., 1986; Dawson et al., 1998). In that study, participants watched as a virtual actor looked towards a checkerboard that appeared in her field of view (on congruent trials), confirming the participants expectation regarding what the actor ‘ought to do’ in this situation; or she looked towards empty space (incongruent trials), violating the participant’s expectation. Consistent with prior studies of neurologically normal adults (Pelphrey et al., 2003) and typically developing 7- to 11-year-old children (Mosconi et al., 2005), ‘errors’ (incongruent trials) evoked more activity in the right STS and other social brain regions, indicating a strong modulatory effect of context in neurologically normal participants. The same brain regions were activated during observation of gaze shift in the participants with autism, but these regions did not respond differentially to the congruent and incongruent trials, indicating that activity in the STS region was not modulated by the context of the perceived gaze shift. The present results are also consistent with findings from Castelli and colleagues (2002), who reported reduced activity in the STS region during a task involving the attribution of intentions to moving geometric figures. These prior findings, combined with the results of the current study strongly suggest a role for the STS region as a brain mechanism underlying social dysfunction in autism.

**The fusiform gyrus**

Schultz et al. (2000) compared the FFG response to faces and non-face objects in people with autism and Asperger’s syndrome. They found that there was less face-evoked activation in this region and that areas typically recruited for non-face object perception were used for face perception in these individuals. Since this original study, at least four other fMRI studies have replicated the finding of fusiform gyrus hypoactivation (Critchley et al., 2000; Pierce et al., 2001; Hubl et al., 2003; Pierce et al., 2004). However, these findings have been contested because individuals with autism do not look at faces in the same manner as do typically developing individuals (Klin et al., 2002; Pelphrey et al., 2002), and thus abnormal neural activity in autism during face processing could be due, at least in part, to poor information acquisition during eye scanning. To address this issue, Hadjikhani et al. (2004) added fixation points to the center of images of faces and objects and determined that people with autism exhibited normal FFG activation under that condition. Dalton et al. (2005) monitored eye movements during fMRI and found a strong positive correlation between the number and length of gaze fixations on the eyes of faces and the FFG response in individuals with autism.

We did not directly compare the brain’s response to faces and another object category. Therefore, we were not able to isolate the face-responsive portion of the FFG (i.e. the ‘fusiform face area’). We also did not control for eye movements, although the robust activations in the network of brain regions known to be involved in face processing indicates that both groups of participants attended to the faces during the scan. Nonetheless, we did find a lack of modulation in the FFG by the motion involved in the dynamic expressions. This finding is consistent with hypotheses concerning dysfunction in the bidirectional pathways connecting the FFG with the STS and the AMY (Schultz et al., 2000; Dawson et al., 2002; Schultz, 2005). Similarly, Bird and colleagues (2006) examined whether individuals with autism exhibit attentional modulation for face vs. non-social stimuli. Participants with autism and typically developing participants viewed pairs of face and house stimuli, with one of the pairs randomly assigned to attended locations and the other to unattended locations. Responses to houses were modulated by attention in both the groups, but only the typically developing participants demonstrated attentional modulation of face-selective regions. Thus, the participants with autism demonstrated a lack of attentional modulation which was particularly evident for the faces. Analyses of effective connectivity indicated that these results were due to a failure of attention to modulate connectivity between the fusiform gyrus and earlier visual cortical regions.

**CONCLUSION**

This study used a paradigm that involved, for the first time in autism research, dynamic changes in facial displays of emotional states. In the search for the neural basis of face processing deficits in autism it is critically important to use dynamic stimuli because such stimuli could yield a better experimental model of social brain deficits in autism as compared with static faces. This is so because dynamic stimuli are more ecologically valid, given that dynamic information processing is a key to social interaction. Our findings revealed that three key components of the human face processing system and the broader social brain—the AMY, STS and FFG—were dysfunctional in autism. Importantly, this dysfunction was reflected both in
hypoactivation of the FFG and AMY (neurotypical > autism) and in a lack of enhancement of activity in the STS, AMY and FFG by faces containing dynamic information relative to static snapshots of the same emotional expressive faces. These effects were specific to components of the social brain and were not observed in regions outside of the human face processing system (e.g. area MT/VS). Thus, we observed a dissociable pattern of intact activity in an earlier visual area vs. impaired processing in social brain areas. We conclude that dysfunctions in key components of the human face processing system including the AMY, FFG and posterior STS region are present in individuals with high-functioning autism, and this dysfunction might contribute to the deficits in processing emotional facial expressions.

Conflict of Interest
None declared

REFERENCES

Neuroimaging affect perception in autism


