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Successful Face Recognition is Associated with Increased Prefrontal Cortex Activation in Autism Spectrum Disorder

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Abstract

This study examines whether deficits in visual information processing in ASD can be offset by the recruitment of brain structures involved in selective attention. During functional MRI, 12 children with ASD and 19 control participants completed a selective attention one-back task in which images of faces and houses were superimposed. When attending to faces, the ASD group showed increased activation relative to control participants within multiple prefrontal cortex areas, including dorsolateral prefrontal cortex (DLPFC). DLPFC activation in ASD was associated with increased response times for faces. These data suggest that prefrontal cortex activation may represent a compensatory mechanism for diminished visual information processing abilities.

Keywords

autism; functional MRI; selective attention; facial information processing; dorsolateral prefrontal cortex

Introduction

Social skill deficits are a cardinal feature of Autism-spectrum disorders (ASD), stemming at least in part from difficulties in processing facial information (for review see Harms, Martin, & Wallace, 2010; Tanaka et al., In press; Wolf et al., 2008). These difficulties have been associated with abnormalities in visual information processing structures – namely, a portion of fusiform gyrus (FG) called the fusiform face area (FFA). FG and other temporal lobe deficits are now widely viewed as part the etiology of ASD (Dawson, Webb, & McPartland, 2005; Dziobek, Bahnemann, Convit, & Heekeren, 2010; Schultz, 2005). We are nevertheless only beginning to understand how deficits in facial information processing *develop* among

individuals with ASD. One possibility is that faces are less salient or intrinsically rewarding to individuals with ASD (Chevallier, Kohls, Troiani, Brodtkin, & Schultz, 2012; Kohls et al., 2012); these bottom-up affective signals normally serve to enhance perceptual processes (i.e., Vuilleumier & Pourtois, 2007).

Much of the developmental literature on FFA deficits in ASD has focused on interactions between FG and other temporal lobe structures (including amygdala). However, it has become increasingly clear that putatively basic visual information processing modules such as FG are under the influence of frontally mediated attentional control mechanisms. Selective attention not only shapes visual cortex “online” during object recognition, but may also contribute to the long-term development of category specificity within visual cortex (Beck & Kastner, 2009; Fuster, Bauer, & Jervey, 1985; Gregoriou, Gotts, Zhou, & Desimone, 2009; Miller, Vytlačil, Fegen, Pradhan, & D'Esposito, 2011). These findings complicate FG accounts of facial information processing deficits in ASD by raising the possibility that these deficits are related to problems in top-down attentional resource allocation as well as the bottom-up influence of social and emotional salience. There have been few studies to date that test this hypothesis.

An important test of any visual information processing theory of ASD is whether it can account for the striking profile of deficits *and strengths* frequently observed in this population. Many individuals with ASD show relative strengths in specific visual processing domains, particularly those focused on local rather than global visual properties (see Dakin & Frith, 2005). Much of the research on visual information processes in ASD has centered on the development of theoretical models that can explain why both strengths and weaknesses are observed (for example, Weak Central Coherence Theory; Frith & Happé, 1994; Frith, 1989). The present study examines whether selective may be leveraged by individuals with ASD to compensate for weaknesses in visual information processing. Specifically, selective attention may be one mechanism whereby the individuals with ASD offset their relative emphasis on the processing of local features at the expense of global percepts.

At first glance, the notion that ASD is associated with preserved or even enhanced selective attention mechanisms may seem somewhat paradoxical – ASD is often associated with *deficits* in selective attention and other executive functions (Belmonte & Yurgelun-Todd, 2003; Hill, 2004; Koshino et al., 2005, 2008). The literature on this topic is complex, with considerable variability in the type and severity of attentional deficits reported across individuals and studies (for considerations of this topic see Just, Cherkassky, Keller, Kana, & Minshew, 2007; Kenworthy, Yerys, Anthony, & Wallace, 2008; Koshino et al., 2005; Ozonoff & Strayer, 2001; Remington, Swettenham, Campbell, & Coleman, 2009). In fact, the variability in ASD findings is sometimes referenced as a critique of executive function as a construct (Kenworthy et al., 2008). Ultimately, findings of selective attention deficits do not preclude the possibility that cognitive control structures can facilitate successful visual information processing (e.g., face processing) in ASD, when necessitated by task demands. Few existing studies have examined this possibility.

Most of the studies to date using face paradigms with selective attention manipulations (including some in ASD) have focused on the modulation of visual cortex activation – not on selective attention regions themselves (i.e., prefrontal cortex [PFC]). For example, O'Craven and colleagues (O'Craven, Downing, & Kanwisher, 1999) used a paradigm where face and non-face (house) pictures were superimposed on one another (see Fig. 1). In their task, a non-clinical sample completed a one-back working memory task, indicating whether the face or the house they perceived was the same as the one from the previous trial. The key manipulation was one of selective attention; although all trials involved the simultaneous presentation of faces and houses, participants were asked to focus either on the faces or the houses when performing the task. O'Craven and colleagues found increased FG activation when participants responded to faces within the overlaid face/house stimuli.

The central hypothesis of the present study was that individuals with ASD require increased recruitment of frontal control structures to disambiguate a face from a complex visual display. We tested this hypothesis via O'Craven et al.'s (1999) basic paradigm – a one-back task where participants had to attend to either the face or the house within a stimulus where the two were superimposed (Fig. 1). PFC is home to a constellation of functional regions thought to implement executive functions and cognitive control (for recent reviews see (Banich et al., 2009; Niendam et al., 2012). Of primary interest were group differences in dorsolateral prefrontal cortex (DLPFC) and anterior cingulate cortex (ACC), as these regions implement aspects of executive function related to selective attention. DLPFC implements executive functions related to the maintenance of information in working memory (Dolcos & McCarthy, 2006; MacDonald, Cohen, Stenger, & Carter, 2000). Furthermore, it has been shown to “tune” posterior areas (including visual cortex areas) that are task-relevant (i.e., Banich et al., 2000; Milham, Banich, & Barad, 2003). This aspect of DLPFC function is consistent with the notion that individuals with ASD may require the upregulation of visual cortex structures to accomplish face processing tasks. Anterior cingulate cortex, on the other hand, is widely held to play a role in the resolution of response conflict (Banich et al., 2009; Milham et al., 2001, 2003). These two structures are thought to work together to meet the demands of selective attention when stimuli and responses are ambiguous, as in O'Craven task used here.

Method

Participants—12 individuals with ASD and 19 typically developing controls (TDC) participated in this study. Participants with an autism spectrum disorder were diagnosed according to DSM-IV Criteria following Collaborative Programs of Excellence in Autism (CPEA) guidelines, including the Autism Diagnostic Interview – Revised (ADI-R; Lord, Rutter, & Le Couteur, 1994) and the Autism Diagnostic Observation Schedule – Generic (ADOS-G; Lord et al., 2000). Five participants in the ASD group carried autism diagnoses, 4 carried Asperger Disorder diagnoses, and 3 carried PDD-NOS diagnoses. Diagnostic assessment was administered by a trained clinician with more than five years experience working with individuals on the autism spectrum. See Table 1 for sample characteristics. Participants matched for age between diagnostic groups (ASD = 13.41 ± 4.15 years, TDC = 13.36 ± 3.49 years; $t(29) = .03$, n.s.). Full-scale IQ (FSIQ) was measured for all but one

participant (from the TDC group) using either the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999), the Wechsler Intelligence Scale for Children, 3rd Edition (WISC-III; Wechsler, 2003), the Wechsler Adult Intelligence Scale, 3rd Edition (WAIS-III; Wechsler, 1997), or the Differential Abilities Scale (DAS; Elliot, 2007). Groups were matched in terms of FSIQ (ASD = 108.83 ± 14.78 , TDC = 114.83 ± 10.77 ; $t(28) = 1.28$, *n.s.*). For those cases in which a subject had undergone clinical IQ testing in the last year, this measure was not re-administered but rather the previous scores were utilized. Despite being matched on both chronological age and FSIQ, the ASD group had significantly lower face recognition scores than controls (ASD = 39.42 ± 4.01 , TDC = 43.33 ± 3.20 ; $t(28) = 2.97$, $p < .01$), as measured by the Benton Face Recognition Test (BFRT; Benton, 1994). All participants had normal or corrected-to-normal vision and gave written, informed consent (from parents and/or legal guardians) as dictated by protocols approved by the Institutional Review Board at the Yale University School of Medicine.

Experimental Paradigm—Face stimuli were taken from Endl and Colleagues (Endl et al., 1998). Images were front-view, grayscale, and modified to remove all peripheral features (ears, hair, etc.). Although earlier versions of this paradigm used neutral faces, the superposition of the house over neutral faces gave them a disembodied quality that could be perceived as mildly negative. In order to avoid possible group differences in this effect, faces with a mild happy expression were used for all trials (Fig. 1). House stimuli consisted of pictures taken from neighborhoods surrounding the Yale University campus. No stimuli were repeated during the experiment (except within “same” pairs).

Participants performed a one-back working memory task based on the design of O’Craven and colleagues (O’Craven et al., 1999). Seventy faces and 70 houses were included in the fMRI task. Faces were superimposed on houses, so that both images were simultaneously visible within the same space. Participants provided “same” or “different” responses via button press using the index fingers of the left and right hand.

During fMRI scanning, participants completed a single run of 8 alternating attend-face (AF) and attend-house (AH) blocks (4 AF-AH cycles, separated by 12 s inter-block intervals). Block order (i.e., starting with AF or AH) was counterbalanced between subjects. At the start of a block, participants were prompted with a single word (either FACE or HOUSE, displayed for 2000 ms) to base their identity discrimination on either faces or houses. Blocks lasted 28 seconds and consisted of 10 face/house composite images (each image subtending approximately 8° of visual angle), presented for 1400 ms with a 1400 ms inter-stimulus interval (participants could respond at any point during the stimulus or inter-stimulus interval). Each block of 10 images contained 5 same and 5 different trials in a pseudorandom order. The entire task took 5.6 minutes

fMRI Data Acquisition—MRI data were acquired on a Siemens Trio 3T scanner (Malvern, PA) using a standard quadrature head coil. Functional images were collected using a T2*-weighted gradient-echo echo-planar image (EPI) sequence providing full head coverage (TR = 2320ms, TE = 25ms, flip angle = 60°, FOV = 225 × 225, 3.5mm isotropic voxels [no gap], 40 axial slices, oblique axial prescription parallel to AC-PC plane). Stimuli were back-projected using E-Prime presentation software (Psychology Software Tools Inc.,

Pittsburgh, PA) onto a translucent screen, viewed via a coil-mounted periscopic prism system. Participants' behavioral responses were collected via fiber optic button box, with two possible responses for both AF and AH conditions ('same' or 'different' identities). T1-weighted 2D anatomical images with the same slice prescription as the EPI data (TR = 300ms, TE = 2.43ms, flip angle = 60°), and a T1-weighted 3D anatomical MPRAGE volume (TR = 2530ms, TE = 3.66ms, TI = 1100ms, flip angle = 7°, 1mm isotropic voxels), were acquired during the same session, for standard-space image registration.

FMRI data reduction and analysis

FMRI data processing and analysis was conducted using BrainVoyager QX v1.10 (Brain Innovation, Maastricht, The Netherlands). The first four volumes of each functional run were discarded to allow for signal stabilization. Functional data were motion corrected, temporally filtered (to remove linear trends), and spatially filtered (FWHM = 4 mm). Data from two participants (1 ASD, 1 TDC) displaying excessive motion at the end of their respective runs were shortened to include only the first three AF-AH cycles (i.e., 6 blocks). Several subjects (5 ASD, 3 TDC) moved consistently enough throughout the course of the run to warrant exclusion from data analysis, leaving 31 subjects (12 ASD, 19 TDC) in the final sample. After these exclusions, per-participant estimates of six motion parameters (translation and rotation in the x, y, and z dimensions) were calculated using the maximum difference in motion between each volume and the one preceding it (i.e., framewise displacement). Average translation displacements were below .5 for both groups, and rotation displacements were below .5 degrees, with no significant differences between groups for any of the six parameters.

Statistical maps were generated for each time series via per-voxel GLM. The GLM included explanatory variables coding attend-face (AF) and attend-house (AH) blocks (convolved with a double gamma function to better approximate the hemodynamic response). Resulting parameter estimate (PE) maps were used as dependent variables in group-level mixed-model GLMs examining the effects of task (faces versus houses) and diagnostic group (TDC versus ASD). Statistical maps were registered to standard (Talairach) space using BrainVoyager's manual registration procedures. Family-wise error (FWE) was maintained at $p < .05$ via the application of a per-voxel threshold ($p < .005$) and a cluster size threshold of 5 (this threshold/size combination was determined via Monte Carlo simulation using the program AlphaSim; Ward, 2006).

Behavioral Performance Analyses—Performance data were examined in the context of mixed model ANOVAs including factors representing Condition (AF and AH) and Group. Pearson correlations were used to test the relationship between task performance data (response time and accuracy) and percent signal change (PSC) averaged within a right DLPFC region of interest (ROI) that was shown to be significantly more active for the ASD group when contrasting AF and AH conditions (this ROI is described below; the same exact ROI was used to extract data from both groups). Percent signal change was calculated relative to the average signal during the rest period immediately before task blocks.

Results

Behavioral Performance—Participants were significantly more accurate in discriminating houses than faces, $F(1,29) = 12.232$, $p = .002$ (see Table 1 for cell means). There was no significant main effect of Group, $F(1,29) = 0.225$, $n.s.$, nor was there a significant Condition $\times$ Group interaction, $F(1,29) = .133$, $n.s.$. An equivalent 2×2 (Condition $\times$ Group) ANOVA predicting reaction time showed a statistical trend towards slower responses for AF trials, $F(1,29) = 2.999$, $p = .094$, but no Group main effect or Condition $\times$ Group interaction, $F_s(1,29) = .139$ and 1.037 , respectively, both $n.s.$

Functional MRI Data—The ASD group showed greater activation relative to TDCs for the AF > AH contrast in multiple areas of frontal cortex (i.e., a Group $\times$ Condition interaction; see Table 2 for details regarding significant activation clusters). Among these frontal areas right were middle and superior frontal gyri corresponding to DLPFC (Fig. 2) – areas that have been widely implicated in executive control (Banich et al., 2009; Rajkowska & Goldman-Rakic, 1995). Simple effects tests of Condition within each group (Fig. 2) indicated that this interaction was driven by increased activation in the ASD group for the AF compared to the AH condition, $t(11) = 2.426$, $p = .034$, and the opposite pattern (AH > AF) for the TDC group, $t(11) = 3.310$, $p = .004$. Examining the simple effect of Group within Condition, the ASD group showed significantly increased activation in this area (relative to TDCs) for faces, $t(29) = 1.999$, $p = .055$, but not houses, $t(29) = .082$, $n.s.$ This overall pattern indicates that the ASD group required enhanced selective attention to identify the faces within the visual display, whereas the TDC group required enhanced selective attention to suppress the face percept and focus on houses (hence the increased activation in the TDC group for AH relative to AF). Increased activation was also observed in lateral portions of ventral anterior cingulate cortex, an area associated with the maintenance of attention when confronted with distracting information (Milham et al., 2003).

Across participants, the contrasts of AF > AH yielded significant activation in right fusiform gyrus (see Fig. 1; peak coordinate = 40,-38,-16, $t(30) = 3.35$, $p = .001$). However, the two groups did not significantly differ in this effect. No significant Group or Condition effects were observed in left fusiform gyrus.

Although not of a priori interest in this study, number of areas outside of frontal cortex were also significantly more active for ASD participants for the AF > AH contrast (see Table 2 for coordinates). Significant clusters were observed bilaterally in the inferior parietal lobule and supramarginal gyrus. In the right hemisphere, activation was observed in right superior parietal lobule. In the left hemisphere, activation was observed in precuneus, supramarginal and angular gyri. No significant differences between groups were observed within FG for the AF > AH contrast, or for either condition > rest.

There were no FWE-corrected areas where TDC participants showed greater activation than ASD participants for the AF > AH contrast.

Correlations with Behavioral Performance—In order to examine further the relationship between increased frontal activation in attentional resource allocation, Pearson

correlations were run between DLPFC PEs (AF > Rest, averaged for voxels within group-defined ROI) and response time. In the ASD group, faster response times were associated with increased activation within this ROI, $r = -.61$, $p = .033$ (illustrated in Fig 2). This same correlation was not significant in the TDC group, $r = -.16$, *n.s.* Neither the ASD nor the TDC groups showed a significant correlation between activity within the right DLPFC ROI and accuracy for faces ($r = .29$ for ASD and $.32$ for TDC, both *n.s.*).

Discussion

The present findings indicate that ASD is associated with increased recruitment of cognitive control structures during a facial recognition task requiring selective attention. This pattern was clearest for right DLPFC, where the ASD group showed increased activity when attending to faces, but the TDC showed the opposite pattern (increased DLPFC activity for attend-house). Furthermore, increased activation was associated with enhanced task performance in the ASD group (but not the TDC group), as reflected by increased response time. These data suggest that in typical development, enhanced selective attention is required to disregard the percept of a face in a complex visual display – but in ASD, selective attention is instead required to *process* the face percept, as required by task demands.

The pattern of group means indicated increased right DLPFC recruitment in ASD (compared to TDC) for faces only (i.e., not the attend-house condition; see Fig 2). This suggests that, although the TDC and ASD groups performed comparably on this task, discriminating faces was particularly demanding of attentional resources in the ASD group. This finding is particularly noteworthy, as individuals with ASD have been shown to underactivate portions of PFC during n-back tasks (i.e., Koshino et al., 2005, 2008). The present task differs from prior n-back tasks in using ambiguous visual stimuli that pose particular visual information processing challenges for individuals with ASD. Furthermore, the absence of a significant increase in right DLPFC activation during the attend-face condition (relative to attend-house) in the TDC group indicates that a significant working memory load was required by the ASD group only, despite the use of a 1-back response task. This pattern is consistent with the notion that faces are processed rapidly and efficiently by most individuals, but less so in ASD.

The possibility that aspects of selective attention can be leveraged to compensate for face processing deficits in ASD has significant implications for how we understand and treat the disorder. Despite the common co-occurrence of attentional deficits in ASD (Gadow, DeVincent, & Pomeroy, 2006; Gargaro, Rinehart, Bradshaw, Tonge, & Sheppard, 2011; Leyfer et al., 2006), many individuals on the spectrum are able to maintain a seemingly impenetrable focus on idiosyncratic topics of interest (American Psychiatric Association, 2013). The idea that this type of attention can be refocused on social information is one of the premises behind recent game- and video-based interventions for face processing in ASD (Baron-Cohen, Golan, & Ashwin, 2009; Tanaka et al., 2010; Wolf et al., 2008). The present data suggest a plausible neurobiological mechanism for training-based improvement in social information processing – one related to selective attention and DLPFC.

This study is among the first to report increased frontal activation in ASD during a face processing task. This is somewhat surprising when one considers the large number of face tasks used in ASD samples. There are a few elements of the present study design that may have been important in eliciting increased frontal activation in ASD. First and foremost, this study examined facial recognition in the context of distracting visual information (i.e., face and non-face stimuli appearing simultaneously on the screen). Participants were therefore required to disregard distracting visual information every time a stimulus was presented. This design element plays directly to a key role of DLPFC (Banich et al., 2009).

Nevertheless, Bird et al. (2006) did not report increased frontal activation in ASD using a different paradigm that also presented face and non-face stimuli simultaneously. When juxtaposing the present paradigm with theirs, another key difference emerges – in the present task, face and non-face stimuli were directly superimposed on one another, whereas in their task, face and non-face stimuli were adjacent. Although preliminary, it seems likely that the superposition of face and non-face stimuli in the present task required more PFC-mediated selective attention resources (a within-subject comparison of these two paradigms in a future study would speak more directly to this hypothesis).

The present findings coincide with multiple reports of *decreased* PFC function in ASD, along with decades of neuropsychology research on abnormal executive function in this population (Hill, 2004; Pennington & Ozonoff, 1996). It is important to note that the increased DLPFC activation observed in this study does not challenge the notion of executive function deficits in ASD. Rather, the present DLPFC finding suggests that, for some individuals with ASD, selective attention mechanisms may be called upon under circumstances where they are not generally needed by typically developing individuals. In other words, executive function deficits in ASD may not always manifest as a broad deficiency present under all tasks and circumstances, but rather, as a reallocation of these resources.

It is also important to note that the paradigm used in this study was not designed to elicit global differences in executive function, as both the primary and control conditions (i.e., attend-face and attend-house) necessitated some working memory (i.e., a one-back task). Recent research on frontal lobe function in ASD shows variable results, with some executive function tasks eliciting normal or increased activation patterns (Dichter, Felder, & Bodfish, 2009; Gilbert, Bird, Brindley, Frith, & Burgess, 2008). Findings for DLPFC in fact parallel those of Dichter and colleagues (Dichter et al., 2009), who reported increased activation of dorsal anterior cingulate and medial prefrontal cortices in ASD during a target detection task.

The absence of group differences in FG add to this compensatory account of face processing in ASD, as well as informing ongoing considerations of the precise role of this structure as an endophenotype of ASD. In particular, the absence of FG differences between groups raises the following question: what exactly are the parameters surrounding the presence or absence of FG differences across face processing tasks? The present data suggest that certain types of selective attention may ultimately mitigate what might otherwise appear to be FG deficits in ASD. Although the precise mechanism behind this result clearly warrants detailed

investigation, a compensatory account appears to fit best with the findings from this study. In short, when presented with a task that requires enhanced selective attention to faces (and, conversely, the filtering of competing visual information), individuals with ASD may be able to up-regulate visual information processing systems to the point that activation in visual cortex is comparable to typically developing individuals. Future studies could test this hypothesis more directly by developing tasks where selective attention, and the presence of distracting information, are modulated parametrically.

These findings on DLPFC and FG would seem to point directly to examinations of brain connectivity. The top-down modulation of visual cortex is presently a topic of considerable interest (Bar et al., 2006; Brassen, Gamer, Rose, & Büchel, 2010; Furey et al., 2006; Herrington, Nymberg, & Schultz, 2011; Herrington, Taylor, Grupe, Curby, & Schultz, 2011; Luks & Simpson, 2004; Miyashita & Hayashi, 2000). Furthermore, connectivity deficits are increasingly implicated in ASD (Minshew & Williams, 2007). Connectivity analyses were not conducted with the present data because of the absence of significant differences in FG activation between groups (i.e., there was little variance between groups that may have been captured by connectivity models; see Smith et al., 2011, for a consideration of the importance of accurate localization in connectivity analyses). Whether FG activation is modulated by frontal attention structures in ASD remains an open and important question.

The compensatory account advocated here presumes that individuals with ASD required more selective attention resources than the TDC group to complete the same task. Correlations between behavioral performance and DLPFC activation speak to this compensatory account, as does the diminished performance of the ASD group on the Benton Facial Recognition Task (BFRT). However, a few caveats should be noted. First, right DLPFC activation did not correlate significantly with accuracy. This may indicate that accuracy is less sensitive than response time to individual differences in information processing (this is the case for many paradigms involving rapid responses). It is nevertheless possible that areas other than right DLPFC are more sensitive to accuracy in this task. The second caveat is the absence of correlation between DLPFC activation and BFRT results. This underscores the considerable variability among facial recognition tasks in terms of selective attention and visual information processing demands. For example, like many face tasks deployed in fMRI studies of ASD, the BFRT involves the matching of faces that are presented simultaneously (rather than sequentially, as in the present task). Last, the two tasks were not fully matched on accuracy – participants were significantly more accurate when attending to houses versus faces. However, this effect did not differ by group, suggesting that both groups responded equally well to the difference in condition-wise task demands.

In summary, findings from the present study suggest that individuals with ASD show increased activation of frontal control structures when selectively attending to faces. The data point not only to group differences in DLPFC function, but also to the relationship between DLPFC and individual differences in facial recognition performance. An individual difference perspective seems particularly appropriate when relating these findings to what is already known about abnormal PFC function, and attentional deficits, in ASD. It is possible that those individuals with ASD who are able to leverage selective attention capacities are

those that would benefit most from interventions to enhance face processing skills (Tanaka et al., 2010; Wolf et al., 2008). This is an important question that can be best addressed by future studies combining brain imaging and behavioral interventions for social information processing deficits.

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Fig. 1.

Overlay Task. Panel A: participants completed a one-back, same/different identity discrimination task. Participants were prompted before each block to attend to either the faces or houses within the composite image. Blocks lasted for 28 seconds and consisted of 10 trials (10 images displayed for 1400 ms, with 1400 ms interstimulus interval). Blocks were separated by 12-second rest periods. The task consisted of a single run of 8 blocks (4 for each active condition). Panel B: Increased right fusiform gyrus activation (circled in green) for all participants when attending to faces (versus houses), family-wise error-corrected $p < .05$ (see Method section).

Fig. 2.

DLPFC activation in ASD when attending to faces. Panel A: increased right DLPFC activation in ASD (circled in green; peak = 27,14,49). The cluster was significant at $p < .05$ (corrected), encompassing 526 voxels. The image is shown from above, with a portion of PFC cut away. Panel B: pattern of means for the DLPFC cluster. Panel C: correlation between average parameter estimate within the cluster and response time for faces in the Overlay Task (ASD group only; $r = -.61$, $p = .033$). Percent signal change was calculated relative to the average signal during the rest period immediately before task blocks.

Table 1
Participant Characteristics

CHARACTERISTIC	ASD	TDC	P
Number of Subjects	12	19	
DSM-IV Diagnosis	Autism Disorder: 5 Aspergers Syndrome: 4 PDD-NOS: 3	N/A N/A N/A	
Chronological Age $\pm$ SD (yrs)	13.4 $\pm$ 4.2	13.4 $\pm$ 3.5	0.97
Full-Scale IQ $\pm$ SD	108.8 $\pm$ 14.8	114.8 $\pm$ 10.8	0.24
Benton Face Recognition $\pm$ SD	39.4 $\pm$ 4.0	43.3 $\pm$ 3.2	0.01
ADOS Score	Social: 8.8 $\pm$ 2.4 Communication: 4.6 $\pm$ 2.1 Total: 13.4 $\pm$ 4.1	N/A N/A N/A	
Attend Face: Accuracy (SD)	81% (.09%)	81% (.10%)	
Attend Face: Response Time (SD)	1078 (148)	1042 (175)	
Attend House: Accuracy (SD)	86% (.10%)	88% (.08%)	
Attend House: Response Time (SD)	1035 (118)	1031 (149)	

Note. DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, 4th Edition; PDD-NOS = Pervasive Developmental Disorder-Not Otherwise Specified; SD = Standard Deviation; ADOS = Autism Diagnostic Observation Schedule. Response times in milliseconds.

Table 2
Overlay Task Activation (ASD > TDC, Attend Face > Attend House)

Region	Hemisphere	Talairach	Size (Voxels)	Peak T
Inferior Parietal Lobule	Right	(36,-49,34)	2256	4.80
Middle/Medial Frontal Gyrus	Right	(15,47,7)	949	4.64
Superior/Middle Frontal Gyrus	Right	(27,14,49)	526	5.02
Superior Parietal Lobule	Right	(30,-52,55)	501	4.10
Supramarginal Gyrus	Right	(54,-40,37)	464	4.31
Genu of Corpus Callosum	Bilateral	(0,17,4)	306	3.85
Anterior Cingulate Cortex	Left	(-18,38,1)	254	3.47
Putamen/Globus Pallidus/Posterior Thalamus	Bilateral	(21,-16,10)	248	3.65
Angular Gyrus	Left	(-48,-64,37)	238	3.91
Dorsolateral Prefrontal Cortex	Right	(45,20,28)	235	3.52
Precuneus	Left	(-24,-49,31)	223	5.17
Middle Cingulate Gyrus	Left	(-6,-4,49)	223	3.72
Supramarginal Gyrus	Left	(-42,-40,40)	207	4.10
Middle/Medial Frontal Gyrus	Left	(-18,50,7)	201	3.72
Inferior Parietal Lobule	Left	(-27,-55,46)	199	3.17

Note. All clusters were significant at $p < .05$ (corrected). Talairach coordinates represent peak voxels for each cluster.