Robust orienting to protofacial stimuli in autism

Punit Shah¹,², Anne Gaule¹,³, Geoffrey Bird²,⁴, Richard Cook¹*

¹Department of Psychology, City University London
²Social, Genetic and Developmental Psychiatry Centre (MRC), Institute of Psychiatry, Kings College London
³Division of Psychology and Language Sciences, University College London
⁴Institute of Cognitive Neuroscience, University College London

*Corresponding author: Richard.Cook.1@city.ac.uk
Department of Psychology,
City University London,
Whiskin Street
London, EC1R OJD
Tel: +44 20 7040 8644, Fax: +44 20 7040 8877

Key words: face perception, autism, attention, orienting, protoface, sub-cortical, social attention
Newborn infants exhibit a remarkable tendency to orient to faces. This behavior is thought to be mediated by a subcortical mechanism tuned to the protoface stimulus; a face-like configuration comprising three dark areas on a lighter background. When this unique stimulus translates across their visual field, neurotypical infants will change their gaze or head direction to track the protoface [1-3]. Orienting to this low spatial frequency pattern is thought to encourage infants to attend to faces, despite their poor visual acuity [2, 3]. By biasing the input into the newborn’s visual system, this primitive instinct may serve to ‘canalize’ the development of more sophisticated face representation. Leading accounts attribute deficits of face perception associated with Autism Spectrum Disorders (ASD; [4]) to abnormalities within this orienting mechanism. If infants who are later diagnosed with ASD exhibit reduced protoface orienting, this may compromise the emergence of perceptual expertise for faces [5]. Here we report a novel effect that confirms that the protoface stimulus captures adults’ attention via an involuntary, exogenous process (Experiment 1). Contrary to leading developmental accounts of face perception deficits in ASD, we go on to show that this orienting response is intact in autistic individuals (Experiment 2).

Protoface orienting plays a critical role in the development of infants’ face perception and wider socio-cognitive abilities; however, the subcortical mechanism responsible is also thought to influence the behavior of adults [2]. Unlike most other visual stimuli, the protoface remains detectable by adults in continuous flash suppression paradigms, in which the input into one eye is typically rendered unperceivable by a stream of constantly changing input to the other eye [6]. This advantage disappears when the pattern is presented upside-down or in negative polarity. Similarly, when instructed to orient toward stimuli displayed peripherally, adults’ saccadic
reaction times (RTs) to the protoface are faster than to orientation-inverted and polarity-reversed control patterns [7]. The difference between detecting, and orienting to, the protoface is not trivial; only orienting behaviors bias the input into the developing visual system and thereby canalize the emergence of sophisticated face representation. Nevertheless, the orientation and contrast specificity of the detection [6] and instructed orienting [7] effects seen in neurotypical adults resembles closely the exogenous orienting responses seen in neurotypical infants [1]. These effects may therefore depend on a common mechanism, mediated by subcortical structures (amygdala, superior colliculus, pulvinar), that is both present in neonates and persists into adulthood [see 2].

In our first experiment, 25 neurotypical participants completed a novel attentional-cueing paradigm during which they were tasked with indicating, as quickly as possible, whether a target letter (‘W’) appeared in a left or right array (Figure 1; left-top, left-middle). Participants’ RTs (Figure 1; right-middle) were significantly faster \[t(24) = 2.983, p = .006\] when the correct side of the display was cued by presentation of the protoface (congruent trials), than when the protoface cued the incorrect side (incongruent trials). Contrary to the suggestion that orienting may be elicited by top-heavy patterns [8], no congruency effects (all \(p > .090\)) were observed for the protoface shown in negative polarity, or a T-pattern in either positive or negative polarity (Figure 1; right-top). Because the protoface stimulus was task-irrelevant, this result provides the first evidence that adults exhibit involuntary, exogenous orienting; the protoface captures attention despite being unrelated to the instructed task.
Our second experiment compared the performance of 18 adults with an ASD and 18 matched controls (see Supplemental Information) on this attentional cueing procedure. The control group demonstrated the same pattern of results seen in Experiment 1 (Figure 1; left-bottom), with significantly faster RTs \([t(17) = 3.209, p = .005]\) on congruent trials than on incongruent trials. Critically, the ASD group showed the same pattern of results (Figure 1; right-bottom), demonstrating significantly faster RTs \([t(17) = 4.851, p < .001]\) on congruent trials, than on incongruent trials, indicative of robust orienting to the protoface. Neither group showed significant congruency effects for any of the control patterns (all \(p > .14\)). No group differences were seen in the orienting response to the protoface \([t(34) = 1.121, p = .27]\) or to the control patterns [all \(p > .60\)]. No association was observed between autism severity and protoface orienting \([r = .044, p = .86]\).

Leading accounts of the face processing deficits characteristic of ASD propose that faces are less able to capture the attention of autistic individuals because of abnormalities within a subcortical orienting mechanism. If, as a result, infants who later develop autism spend less time looking at faces, they may fail to develop equivalent perceptual expertise for faces, with distal consequences for related socio-cognitive functioning [5]. Contrary to these accounts however, we find that individuals with ASD exhibit entirely typical orienting responses to the protoface; the stimulus thought most effective in recruiting the subcortical orienting mechanism [2]. The present results therefore speak against developmental accounts of ASD that invoke deficits in facial orienting [5]. Instead, this conclusion accords with recent evidence that children with ASD (5-12 years) display broadly age-appropriate orienting to photographs of adult faces [9].
Where observed, the failure of autistic individuals to develop typical perceptual expertise for faces may be better explained by a reduced propensity to maintain facial fixation due to diminished motivation [1, 10]. The maintenance of facial fixation, following initial orienting, is controlled by a voluntary, endogenous process – we can choose to maintain attention or to look away. If individuals with ASD find social stimuli less rewarding, they may exhibit shorter fixations despite robust orienting responses. Shorter fixation durations – particularly if seen during critical periods of development – may reduce the fidelity with which faces are processed, thereby affecting the emergence of perceptual expertise with faces. Nevertheless, while differences in fixation maintenance remain a possibility, the present results indicate that the involuntary, exogenous orienting instinct thought crucial for perceptual and socio-cognitive development is intact in autism.
ACKNOWLEDGEMENTS:

PS was supported by a doctoral studentship from the Medical Research Council (MRC). AG was supported by an award from the Wellcome Trust (WT102339MA).
REFERENCES:


Figure 1: Experimental trials required participants to indicate as quickly as possible, which of two letter arrays presented either side of fixation, contained a target letter (‘W’). Immediately before the onset of the arrays, the protoface and an inverted control pattern were presented at peripheral left and right locations, for 200 msec. Participants responded faster on congruent trials (left-top; protoface cued the correct location), than on incongruent trials (left-middle; protoface cued the incorrect location). Concurrent presentation of the inverted control pattern ensured that cueing effects were not due to low-level stimulus features (e.g., luminance, contrast, edge). In close accordance with infant orienting responses [1], the cueing effect was selective for the protoface; other stimulus combinations (right-top) failed to yield significant congruency effects (right-middle). Contrary to leading accounts of face perception deficits in ASD [5], autistic individuals and matched neurotypical controls exhibited equivalent orienting responses (right-bottom and left-bottom, respectively). Significance at $p < .010$ is denoted by **; Significance at $p < .001$ is denoted by ***. (See also Table S1 in the Supplemental Information.)
Supplemental Information: Robust orienting to protofacial stimuli in autism

Punit Shah, Anne Gaule, Geoffrey Bird, & Richard Cook

Experiment 1

Supplemental Results
The RT data (Table S1) were analyzed using ANOVA with stimulus (protoface, T-pattern), polarity (positive, negative) and congruency (congruent, incongruent) as within-subjects factors. The analysis revealed a significant stimulus × polarity × congruency interaction \([F(1,24) = 8.392, p = .008]\). This interaction reflected the presence of a polarity × congruency interaction for the protoface \([F(1,24) = 11.275, p = .003]\) but not the T-pattern \([F(1,24) = 2.109, p = .159]\). Crucially, RTs were significantly faster when the protoface cued the correct position when shown in positive polarity \([t(24) = 2.983, p = .006]\), whilst no congruency effect was seen for the negative polarity protoface \([t(24) = 1.738, p = .095]\).

Supplemental Experimental Procedures
Twenty-five right-handed adults (8 male) participated in Experiment 1 (\(M_{\text{age}} = 24.88\) years, \(SD_{\text{age}} = 5.67\) years). All participants had normal or corrected-to-normal vision, gave informed consent, were paid for their participation, and were fully debriefed upon task completion. Ethical clearance was granted by the local ethics committee and the study was conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.
The protoface stimulus and control patterns subtended 4° × 3° of visual angle when viewed at a distance of 60 cm. Upright and inverted patterns were presented 12° apart. The aspect-ratio of the ‘T’ element was matched to the spatial extent of the protoface elements. Each letter array comprised 4 letters presented in white Arial font. The seven distractor letters were chosen from ‘A’ ‘E’ ‘F’ ‘H’ ‘K’ ‘L’ ‘M’ ‘N’ ‘V’ ‘X’ ‘T’ ‘Y’ ‘Z’ and presented at randomized locations. The target letter (‘W’) was equally likely to appear at each of the 8 locations in arrays. The arrays were presented 6° apart. The display background was grey (128 on a 0–255 scale); equidistant between black (0) white (255). Experimental programs were written in MATLAB using Psychtoolbox [S1, S2]. Stimuli were presented on a Dell LCD monitor at 60-Hz refresh rate.

Participants completed 6 practice trials before starting the experimental procedure, comprising 320 trials, grouped into 4 blocks of 80 trials. Overall the procedure lasted approximately 20 minutes. Trial type (positive polarity protoface; negative polarity protoface; positive polarity T-pattern; negative polarity T-pattern) was interleaved within each block. Participants were instructed to fixate on the central dot; to disregard all peripheral stimuli; and to respond as quickly as they could without sacrificing accuracy. Participants used the left and right arrow keys to record the array in which the target letter appeared. Reaction times (RTs) were taken to be the interval from the onset of the letter arrays, to the moment the participant responded. Mean RTs for each condition were calculated having excluded trials where participants made errors – where the location of the target letter was misidentified – or took longer than 1600 msecs to respond. In total, 3.46% of data points were lost; 3.07% and 0.39%, due to errors and slow responding, respectively. Analyses were conducted on the resulting RT distributions.
Experiment 2

Supplemental Results

The RT data (Table S1) were analyzed using ANOVA with stimulus (protoface, T-pattern), polarity (positive, negative) and congruency (congruent, incongruent) as within-subjects factors, and group (control, ASD) as a between-subjects factor. Crucially, this analysis revealed no main effect of \( p = .67 \), or interactions with (all \( p > .15 \)), group. Both the control and the participants with ASD showed significant stimulus × polarity × congruency interactions, \( F(1,17) = 8.017, p = .012 \) and \( F(1,17) = 17.431, p = .001 \), respectively. As in Experiment 1, these effects reflected the presence of polarity × congruency interactions for the protoface only, \( F(1,17) = 7.354, p = .015 \) and \( F(1,17) = 18.641, p < .001 \), respectively. The RTs of both the controls \( t(17) = 3.209, p = .005 \) and the participants with ASD \( t(17) = 4.851, p < .001 \) were significantly faster only when the correct location was cued by the protoface shown in positive polarity.

No association was observed between autism severity – as measured by the Autism Diagnostic Observation Schedule—Generic (ADOS-G; [S3]) – and orienting towards the protoface \( r = .044, p = .86 \), the protoface in negative polarity \( r = -.084, p = .74 \), the T-pattern in positive polarity \( r = -.26, p = .30 \), or the T-pattern in negative polarity \( r = -.15, p = .55 \). Similarly, across all participants, no association was observed between autistic traits – as measured by the Autism-Spectrum Quotient (AQ; [S4]) – and orienting towards the protoface \( r = -.015, p = .93 \), the protoface in negative polarity \( r = .079, p = .65 \), the T-pattern in positive polarity \( r = -.067, p = .70 \), or the T-pattern in negative polarity \( r = -.124, p = .47 \).
Comparison of the data from Experiment 1 and Experiment 2 revealed significantly slower RTs across all conditions \([t(58.734) = 2.085, \ p = .041]\) in Experiment 2 \((M = 665.52 \text{ msec}, \ SD = 129.30 \text{ msec})\) than in Experiment 1 \((M = 605.52 \text{ msec}, \ SD = 95.48 \text{ msec})\). This almost certainly reflects the fact the participants in Experiment 2 were older \((M_{\text{age}} = 41.0 \text{ years})\) than in Experiment 1 \((M_{\text{age}} = 24.9 \text{ years})\). Consistent with the widely accepted view that slower RTs are seen in older populations \([e.g., \ S5]\), a significant correlation was observed between age and average RT \((r = .523, \ p < .001)\). Nevertheless, no association was seen between age and the degree of the protoface orienting i) across all 61 participants \([r = -.081, \ p = .54]\), ii) in the 43 typical participants’ \([r = -.169, \ p = .28]\) or iii) in the participants with ASD \([r = -.211, \ p = .40]\). Similarly, no association was seen between mean RT across trials and the degree of the protoface orienting i) across all 61 participants \([r = .087, \ p = .505]\), ii) in the 43 typical participants’ \([r = .073, \ p = .641]\) or iii) in the participants with ASD \([r = -.012, \ p = .96]\).

**Supplemental Experimental Procedures**

Thirty-six right-handed adults with \((n = 18)\) and without autism \((n = 18)\) participated in Experiment 2. Participants with autism spectrum disorders (ASD) were recruited from a database held at the Institute of Cognitive Neuroscience at University College London. All had received independent clinical diagnosis (according to the DSM-IV; [S6]) of an ASD from a clinical practitioner. All participants also met the criteria for autism or autism spectrum disorder on the ADOS-G [S3]. All participants completed the AQ [S4] to measure autistic traits, for which the ASD group scored significantly higher than the control group (see Table S2). Finally, all 36 participants reported normal or corrected-to-normal vision.
The stimuli and procedure used in Experiment 2 were identical to those of Experiment 1. As in
the first experiment, mean RTs were calculated having first excluded errors and responses
exceeding 1600 msec. Overall 3.03% of data points were lost from the control group (2.22% and
0.81% due to errors and slow responding) and 2.22% were lost from the ASD group (1.38% and
0.84% due to errors and slow responding). There were no group differences in the number of
trials omitted from analysis, due to either responding too slowly ($p = .93$) or incorrectly ($p = .21$).
Table S1: Mean RTs (msecs) observed in Experiment 1 & 2. Standard deviations are shown in italics inside parentheses.

<table>
<thead>
<tr>
<th></th>
<th>Protiface</th>
<th>T-Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polarity</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Positive</td>
<td>Negative</td>
</tr>
<tr>
<td>Congruent</td>
<td>598 (103)</td>
<td>609 (100)</td>
</tr>
<tr>
<td>Incongruent</td>
<td>617 (108)</td>
<td>596 (98)</td>
</tr>
</tbody>
</table>

Table S2: Mean Age, Gender, Autism-Spectrum Quotient (AQ) and IQ scores [S7] for the autism spectrum disorders (ASD) group and the matched neurotypical controls. Autism Diagnostic Observational Schedule (ADOS) score and classification details for the ASD group. Standard deviations are shown in italics inside parentheses.

<table>
<thead>
<tr>
<th>ASD</th>
<th>Controls</th>
<th>Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td>Gender</td>
<td>16 Male, 2 Female</td>
<td>16 Male, 2 Female</td>
</tr>
<tr>
<td>Mean Age (Years)</td>
<td>40.72 (11.90)</td>
<td>41.33 (13.45)</td>
</tr>
<tr>
<td>Mean Full-scale IQ</td>
<td>115.39 (10.00)</td>
<td>112.11 (13.59)</td>
</tr>
<tr>
<td>Mean AQ</td>
<td>34.50 (8.84)</td>
<td>15.06 (6.03)</td>
</tr>
<tr>
<td>ADOS Classification</td>
<td>11 Autism, 7 Autism Spectrum</td>
<td>-</td>
</tr>
<tr>
<td>Mean ADOS-G Score</td>
<td>10.22 (2.69)</td>
<td>-</td>
</tr>
</tbody>
</table>

*Note.* ADOS-G score is derived from a diagnostic algorithm [S1] with a higher score representing a higher degree of autism.
Supplemental References


