Absence of Preferential Unconscious Processing of Eye Contact in Adolescents With Autism Spectrum Disorder

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Eye contact plays an essential role in social interaction. Atypical eye contact is a diagnostic and widely reported feature of autism spectrum disorder (ASD). Here, we determined whether altered unconscious visual processing of eye contact might underlie atypical eye contact in ASD. Using continuous flash suppression (CFS), we found that typically developing (TD) adolescents detected faces with a direct gaze faster than faces with an averted gaze, indicating enhanced unconscious processing of eye contact. Critically, adolescents with ASD did not show different durations of perceptual suppression for faces with direct and averted gaze, suggesting that preferential unconscious processing of eye contact is absent in this group. In contrast, in a non-CFS control experiment, both adolescents with ASD and TD adolescents detected faces with a direct gaze faster than those with an averted gaze. Another CFS experiment confirmed that unconscious processing of non-social stimuli is intact for adolescents with ASD. These results suggest that atypical processing of eye contact in individuals with ASD could be related to a weaker initial, unconscious registration of eye contact. *Autism Res* 2014, ●: ●●–●●. © 2014 International Society for Autism Research, Wiley Periodicals, Inc.

Keywords: eye contact; gaze processing; autism spectrum disorder; unconscious processing

Introduction

Eye contact serves a fundamental role in human social interaction. Even newborns (2–5 days old) prefer to look at faces with a direct gaze compared with faces with an averted gaze [e.g. Farroni, Csibra, Simion, & Johnson, 2002]. In human adults, faces with a direct gaze hold visual attention [Senju & Hasegawa, 2005] and are detected faster than faces with an averted gaze in visual search arrays [Senju, Tojo, & Hasegawa, 2005a; Von Grünau & Anston, 1995]. This particular relevance of eye contact is also reflected at the neural level. For example, brain regions that are sensitive to social visual information and involved in social cognition, such as the fusiform face area (FFA), superior temporal sulcus (STS), medial orbitofrontal cortex, medial prefrontal cortex, and amygdala, exhibit stronger responses to faces with a direct gaze than to faces with an averted gaze [for reviews, see Nummenmaa & Calder, 2009; Senju & Johnson, 2009a]. This strong modulatory influence of eye contact on behaviour and neural processing has been termed the “eye contact effect” [Senju & Johnson, 2009a, 2009b]. This eye contact effect has been suggested to involve a rapid subcortical face detection pathway (including the superior colliculus, pulvinar, and amygdala) that modulates neural processing in cortical areas [Johnson, 2005; Senju & Johnson, 2009a]. Dysfunction of subcortical regions or their atypical connectivity with cortical areas might alter or delay the development of social cognition and “social brain” networks, both of which are related to the difficulties observed in autism spectrum disorder [ASD; Johnson, 2005; Schultz, 2005; Senju & Johnson, 2009a, 2009b].

ASD is a developmental disorder characterised by difficulty with social interaction and communication, and restricted and repetitive behaviours [American Psychiatric Association, 2013]. Importantly, atypical behavioural responses to eye contact are one of the main

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characteristics of ASD. Whereas typically developing (TD) individuals process faces making eye contact with priority and show specific behavioural and neural responses to eye contact, there is evidence that individuals with ASD do not treat eye contact specially. For example, in children with ASD, laterally oriented faces making eye contact do not facilitate detection [Senju et al., 2005a; Senju, Yaguchi, Tojo, & Hasegawa, 2003]. Similarly, in children with ASD, eye contact fails to elicit enhanced performance in facial gender discrimination [Pellicano & Macrae, 2009] and facial memory tasks [Zaki & Johnson, 2013]. Finally, while animated faces with direct compared with averted gaze elicit enhanced activation in the social brain network in TD individuals, individuals with ASD show the opposite activation pattern in this brain network [Von dem Hagen, Stoyanova, Rowe, Baron-Cohen, & Calder, 2013].

Other findings, however, indicate that some aspects of the eye contact effect might be preserved in individuals with ASD. For instance, when eyes are presented in isolation [Senju, Kikuchi, Hasegawa, Tojo, & Osanai, 2008; Senju et al., 2005a] or embedded in front-view faces [Senju et al., 2008], even children with ASD show a detection advantage over faces with an averted gaze. In addition, when individuals with ASD are explicitly instructed to fixate the centre or the eyes of the facial stimuli, they show enhanced activation in the fusiform gyrus [Hadjikhani et al., 2004; Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2007], amygdala, and pulvinar [Zürcher et al., 2013]. Children with ASD also showed the same delayed attentional disengagement from faces as TD children when the task required attending to the eyes, while still showing atypical event-related spike potentials [Kikuchi et al., 2011]. Neurophysiological studies also suggest a privileged but different response to eye contact in children with ASD [Grice et al., 2005; Kylläinen & Hietanen, 2006; but see also Senju, Tojo, Yaguchi, & Hasegawa, 2005b]. Together, these results suggest that some kind of eye contact effect might be observed even in individuals with ASD, but its cognitive and neural basis might be different from that of TD individuals.

One possible mechanism underlying this atypical eye contact effect is a reduced function of subcortical areas that play a crucial role in the initial, unconscious registration of eye contact [Johnson, 2005; Senju & Johnson, 2009a]. To test this possibility, we compared the influence of eye contact on the unconscious processes that precede and lead to stimulus awareness between individuals with ASD and TD individuals, by using continuous flash suppression [CFS, Tsuchiya & Koch, 2005]. In a recent study using this CFS technique, a target face with a direct or an averted gaze was presented to one eye while dynamic Mondrian-like masks were flashed into the other eye, suppressing the target from awareness. This technique is different from other techniques measuring unconscious processing such as subliminal brief presentation (e.g. 14 ms) and ordinary binocular rivalry techniques in that the target stimulus is suppressed from awareness for extended periods up to several seconds despite the stimulus being physically input into the retina [e.g. Tsuchiya, Koch, Gilroy, & Blake, 2006]. In TD individuals, the duration until the target face became visible was shorter for faces with a direct gaze than for faces with an averted gaze [Stein, Senju, Peelen, & Sterzer, 2011; Yokoyama, Noguchi, & Kita, 2013], suggesting that eye contact is preferentially processed even before observers are aware of seeing a face [i.e. unconsciously, Jiang, Costello, & He, 2007; Zhou, Jiang, He, & Chen, 2010]. There is evidence that subcortical areas are crucially involved in mediating unconscious stimulus processing [Morris, Ohman, & Dolan, 1998; Williams, Morris, McGlone, Abbott, & Mattingley, 2004] and that these subcortical circuits might be altered or dysfunctional in ASD [Kleinmans et al., 2011]. Thus, this unconscious eye contact effect under CFS may represent a particularly sensitive measure of atypical eye contact processing in individuals with ASD.

In the present study, Experiment 1 (contrast, CFS) was conducted to ensure that individuals with ASD could do the CFS task adequately. We predicted that high-contrast Gabor patches would overcome CFS more quickly than low-contrast Gabor patches in both ASD and TD participants [e.g. Tsuchiya & Koch, 2005]. In Experiment 2 (Gaze, CFS), we investigated whether individuals with ASD would show an atypical unconscious eye contact effect. We predicted that faces with a direct gaze would break CFS suppression faster than faces with an averted gaze in TD participants [Stein et al., 2011], but not in participants with ASD. In Experiment 3 (Gaze, non-CFS), we explored whether the findings in Experiment 2 could be attributed to differences in unconscious processing between the two groups. Therefore, this control experiment was designed to resemble the task conducted in Experiment 2 but did not involve interocular suppression. We predicted that there would be no group difference in Experiment 3.

Material and Methods
Participants
Eighteen adolescents with ASD (12–21 years old, one female) and 18 TD adolescents (12–20 years old, five females) participated in Experiments 1, 2, and 3 (Table 1). Participants with ASD had been previously diagnosed with autistic disorder (n = 11), Asperger syndrome (n = 1), or pervasive developmental disorder without a detailed diagnosis (n = 6) by at least one child psychiatrist or paediatrician according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) [American Psychiatric Association, 1994]. All of the participants’ parents completed the Japanese version of the
Social Communication Questionnaire [SCQ, Rutter, Bailey, & Lord, 2003a] to corroborate their clinical presentation (all participants with ASD above the cut-off point, 15). The SCQ is a 40-item rating scale and its content is based on that of the Autism Diagnostic Interview-Revised [Rutter, Le Couteur, & Lord, 2003b], which is a standardised parent interview. Ten participants with ASD were assessed by the Autism Diagnostic Observation Schedule [ADOS; Lord et al., 2000], which is also a standardised assessment of ASD. These ten participants (M = 24.0, standard deviations [SD] = 4.4) did not differ significantly from the other eight ASD participants (M = 28.0, SD = 6.1) in their SCQ scores (P = .125). All ten participants scored above the cut-off points of ASD on both subscales Communication and Reciprocal Social Interaction (2 and 4, respectively). An abbreviated version of the Japanese Wechsler Intelligence Scale for Children [WISC; Wechsler, 1992; Japanese WISC-III Publication Committee, 1998] or Wechsler Adults Intelligence Scale [WAIS; Wechsler, 1981; Shinagawa, Kobayashi, Fujita, & Maekawa, 1990] was administered to all of the participants to measure their IQ. The WAIS for those greater than 16.9 years old and the WISC for those less than 16.9 years old. There were no significant group differences in IQ, chronological age, or sex ratio (all Ps > .05). All participants had normal or corrected-to-normal visual acuity. Written informed consent was obtained from all participants and from their parents. This study was approved by the Research Ethics Committee of University of Tokyo.

### Apparatus and Stimuli

Participants viewed a pair of dichoptic displays on a 17-inch CRT (cathode ray tube) monitor (1024 × 768 pixel resolution, 60-Hz frame rate) through a custom-built mirror stereoscope. The participant’s head was stabilised by a chin-and-head rest at an effective viewing distance of 50 cm. Stimuli were presented with Matlab (The MathWorks, Natick, MA, USA), using the Cogent 2000 toolbox (www.vislab.ucl.ac.uk/cogent.php) on a desktop PC. Two red frames (9.5° × 9.5°) were displayed side-by-side on the screen against a uniform grey background, such that one frame was visible to each eye. To further support binocular alignment, fusion contours (width 0.7°) consisting of random noise pixels were presented within the red frames. In the centre of each frame, a red fixation dot (0.6° × 0.6°) was displayed. High-contrast coloured Mondrian-like masks (8.2° × 8.2°) were used to suppress the target from conscious awareness.

In Experiment 1 (contrast, CFS), we studied the effect of luminance contrast on suppression durations using vertically and horizontally oriented Gabor patches (2.6° × 2.6°), as target stimuli; half were high contrast (Michelson contrast = 1) and the other half were low contrast (Michelson contrast = 0.37). In Experiments 2 (gaze, CFS) and 3 (gaze, non-CFS), we used face stimuli that were selected to rule out the potential confounding influence of greater eye symmetry present in faces with a direct gaze and straight head direction. We adopted face photographs that were used in a series of previous studies investigating the detection of visible [Senju & Hasegawa, 2005; Senju et al., 2005a] and invisible gaze directions [Stein et al., 2011]. These stimuli were constructed from the same base image depicting a female model with a laterally averted head. Eye regions derived from other photographs of the same person were then superimposed onto the base image and carefully smoothed into this base image. The superimposed eyes were directed maximally either to the left or to the right. This yielded the impression of a direct gaze when eye gaze and head were oriented in opposite directions and the impression of an averted gaze when eye gaze and head were pointing in the same direction. Face stimuli were greyscale and cropped to oval shapes (Fig. 1; 2.7° × 4.0°), and then equalised for global contrast and luminance; after this, the edges of the ovals were blurred into the background. Half of the faces were oriented to the left and half to the right.

### Procedure

In all experiments, trials started with a 1-sec presentation of the red frames, the fusion contours, and the fixation...
target faces (Experiments 2 and 3, direct or averted), two Gabor orientations (Experiment 1, vertical or horizontal), or two head orientations of the target faces (Experiments 2 and 3, left or right) occurred equally often within each experiment.

Design

The mean response times (RTs, seconds) were compared by a two-way mixed analysis of variance (ANOVA) with group (ASD or TD) as the between-participants factor and contrast (high or low) in Experiment 1 or gaze direction (direct or averted) in Experiments 2 and 3 as the within-participant factor. In addition, to directly compare the effect of eye contact on CFS and on the non-CFS control condition between groups, we conducted a three-way mixed ANOVA with the group and gaze direction as factors, along with experiment as an additional within-participant factor (Experiment 2 or 3). Trials with no responses and trials with incorrect responses (Experiment 1, 3.3% and 1.0%; Experiment 2, 0.1% and 1.4%; Experiment 3, 0.1% and 0.7%, respectively) were excluded from the calculation of mean RTs. Trials with RTs of more than 2 SDs above or below the mean of each individual in each condition (Experiment 1, 5.0%; Experiment 2, 3.2%; Experiment 3, 3.9%) were excluded from the RT analysis. The number of no responses and incorrect responses did not differ between groups or conditions in all experiments (all Ps > .05).

Results

Experiment 1 (Contrast, CFS)

An ANOVA revealed a significant main effect of contrast \( F_{11, 34} = 18.77, P < .001, \eta^2_p = .36 \). As predicted, high-contrast Gabor patches \( M = 7.42, \text{standard error of the mean} = 0.37 \) were localised faster than low-contrast Gabor patches (Table 2; \( M = 8.15, \text{SEM} = 0.31 \)). Neither the main effect of group nor the interaction between group and contrast were significant (both Ps > .05).

Comparison of Gaze Effect Under CFS (Experiment 2) and Non-CFS (Experiment 3)

A three-way ANOVA revealed a significant interaction between group, gaze direction, and experiment \( F_{11, 34} = 6.73, P = .014, \eta^2_p = .17 \). We followed up on this interaction effect by conducting separate two-way ANOVAs for Experiments 2 and 3 (see later). There were also significant main effects of gaze direction \( F_{11, 34} = 28.41, P < .001, \eta^2_p = .46 \) and experiment \( F_{11, 34} = 26.45, P < .001, \eta^2_p = .44 \); namely, faces with a direct gaze \( M = 4.66, \text{SEM} = 0.23 \) were localised faster than faces with an averted gaze \( M = 4.95, \text{SEM} = 0.23 \),
and RTs were longer in the CFS experiment (Experiment 2; M = 5.51, SEM = 0.32) than in the non-CFS experiment (Experiment 3; M = 4.10, SEM = 0.19).

**Experiment 2 (Gaze, CFS)**

Under CFS, there was a significant main effect of gaze direction (F_{1, 34} = 8.88, P = .005, η^2 = .21); faces with a direct gaze (M = 5.36, SEM = 0.33) were localised faster than faces with an averted gaze (Fig. 3A; M = 5.65, SEM = 0.32). Importantly, there was also a significant interaction between group and gaze direction (F_{1, 34} = 4.79, P = .036, η^2 = .12). A simple effect analysis revealed a significant main effect of gaze in the TD group (F_{1, 17} = 15.13, P = .001, η^2 = .47) but not in the ASD group (F_{1, 17} = 0.28, P = .604, η^2 = .02). Only TD participants localised faces with a direct gaze (M = 4.91, SEM = 0.43) faster than faces with an averted gaze (M = 5.41, SEM = 0.41). The main effect of group was not significant (P > .05).

**Experiment 3 (Gaze, Non-CFS)**

There were significant main effects of group (F_{1, 34} = 9.14, P = .005, η^2 = .21) and gaze direction (F_{1, 34} = 36.37, P < .001, η^2 = .52). TD participants (M = 3.58, SEM = 0.17) localised the faces faster than participants with ASD (M = 4.63, SEM = 0.21), and faces with a direct gaze (M = 3.96, SEM = 0.27) were generally localised faster than faces with an averted gaze (Fig. 3B; M = 4.25, SEM = 0.28). The interaction between group and gaze direction was not significant (P > .05).

**Comparison of Effect of Contrast (Experiment 1) and Gaze Direction (Experiment 2) Under CFS**

Finally, we directly compared the influence of contrast differences (Experiment 1) and gaze direction (Experiment 2) on detection under CFS between groups. To this end, we compared the difference scores of luminance contrast (RT_{low-contrast} − RT_{high-contrast}) and gaze direction (RT_{direct} − RT_{averted}) by a two-way mixed ANOVA with group (ASD or TD) as the between-participants factor and stimulus (contrast or gaze) as the within-participant factor. We expected to find a group difference only for the gaze effect, but not for the contrast effect. There was a significant main effect of stimulus (F_{1, 34} = 6.64, P = .015, η^2 = .16); the influence of contrast (M = 0.73, SEM = 0.17) on detection under CFS was larger than that of gaze direction (M = 0.29, SEM = 0.10). There was also a trend towards a significant interaction between group and stimulus (F_{1, 34} = 3.51, P = .070, η^2 = .09). A simple effect analysis revealed difference between groups for the gaze effect (F_{1, 34} = 4.79, P = .036, η^2 = .12) but not for the contrast effect (F_{1, 34} = 0.44, P = .511, η^2 = .01). The gaze difference score for the TD group (M = 0.50, SEM = 0.13) was larger than that for the ASD group (M = 0.08, SEM = 0.14). There was also a significant main effect of stimulus in the ASD group (F_{1, 17} = 7.23, P = .016, η^2 = .30) but not in the TD group (F_{1, 17} = 0.39, P = .539,
Only in the ASD group, the difference score of contrast ($M = 0.84$, $SEM = 0.29$) was larger than that of gaze ($M = 0.08$, $SEM = 0.14$). The main effect of group was not significant ($P > .05$).

**Discussion**

The present study investigated unconscious and conscious processing of faces with either a direct gaze or an averted gaze in adolescents with ASD and TD adolescents using CFS. In Experiment 1, we first ensured that adolescents with ASD could do the CFS task adequately. Adolescents with ASD displayed similar overall suppression durations and showed the same well-established advantage of high-contrast over low-contrast Gabor patches as TD adolescents. This suggests that there is no group difference in unconscious processing of basic low-level features under CFS. In the critical experiment (Experiment 2), we compared suppression durations for faces with direct gazes and those with averted gazes. As predicted, in TD adolescents, faces with a direct gaze overcame CFS more quickly than faces with an averted gaze. Critically, this unconscious eye contact effect was not found in adolescents with ASD. Thus, eye contact enhanced unconscious face processing in TD adolescents but not in adolescents with ASD. This suggests that there is no group difference in unconscious processing of basic low-level features under CFS. In the critical experiment (Experiment 2), we compared suppression durations for faces with direct gazes and those with averted gazes. As predicted, in TD adolescents, faces with a direct gaze overcame CFS more quickly than faces with an averted gaze. Critically, this unconscious eye contact effect was not found in adolescents with ASD. Thus, eye contact enhanced unconscious face processing in TD adolescents but not in adolescents with ASD. In contrast, both adolescents with ASD and TD adolescents detected faces with a direct gaze faster than those with an averted gaze when they were presented binocularly in the control non-CFS experiment (Experiment 3). These findings suggest that primarily the initial, unconscious processing of eye contact is atypical in ASD.

The present results suggest that atypical eye contact, which is a diagnostic [American Psychiatric Association, 2013; Lord et al., 2000; Rutter et al., 2003b] and broadly reported [e.g. Clifford, Young, & Williamson, 2007; Von dem Hagen et al., 2013; Werner, Dawson, Munson, & Osterling, 2005] feature of ASD, could be mainly driven by the weaker initial, unconscious registration of eye contact. This weaker initial processing in individuals with ASD may prevent them detecting/experiencing eye contact and developing typical social cognition and social brain network [Senju & Johnson, 2009a, 2009b]. The weaker initial eye contact processing may be related to atypical functioning of the subcortical face detection pathway [Johnson, 2005; Senju & Johnson, 2009a]. Interocular suppression is known to strongly suppress neural activity in visual cortical areas [Pasley, Mayes, & Schultz, 2004; Troiani & Schultz, 2013; Williams et al., 2004]. In contrast, residual responses to suppressed emotional faces in subcortical areas such as the amygdala [Jiang & He, 2006; Pasley et al., 2004; Troiani, Price, & Schultz, 2012; Troiani & Schultz, 2013; Williams et al., 2004], superior colliculus, thalamus [Troiani & Schultz, 2013], and in the STS [Jiang & He, 2006] and the FFA [Troiani et al., 2012]—supposedly cortical target areas of the subcortical route—suggest that the subcortical pathway and its cortical output are fairly robust against interocular suppression. Accordingly, the absence of the unconscious eye contact effect under CFS in individuals with ASD might be based on the atypical functioning of subcortical areas including the amygdala [Dalton et al., 2005; Kleinhans et al., 2008; Swartz, Wiggins, Carrasco, Lord, & Monk, 2013; Zürcher et al., 2013], pulvinar [Zürcher et al., 2013], or superior colliculus [Kleinhans et al., 2011], or atypical functional connectivity between these structures and cortical areas involved in eye contact processing [Senju & Johnson, 2009a, 2009b].

Somewhat surprisingly, eye contact facilitated detection in the conscious (i.e. non-CFS) condition even in participants with ASD. This might appear inconsistent
with studies showing atypical eye contact effects in behavioural measures [Pellicano & Macrae, 2009; Senju et al., 2003, 2005a; Zaki & Johnson, 2013] and neural processing [Senju et al., 2005b; Von dem Hagen et al., 2013] in individuals with ASD. However, other studies also demonstrated faster detection of eye contact [e.g. Senju et al., 2005a, 2008] as well as selective physiological responses to eye contact [e.g. Kylliäinen & Hietanen, 2006; Grice et al., 2005] under some experimental conditions. The present results further highlight the atypical, but not fully impaired, nature of eye contact processing in individuals with ASD. Interestingly, only in the non-CFS Experiment 3 did we find overall longer RTs for individuals with ASD, while there was no such overall group difference in the CFS Experiments 1 and 2. This might indicate that the observed eye contact advantage in conscious processing (i.e. Experiment 3) in the ASD group could be a consequence of additional or alternative processing that TD individuals do not need. However, this overall group difference as well as the absence of eye contact effect under CFS in the ASD group should be interpreted with caution, as the age range of the participants was relatively wide and about half of the participants with ASD (8 out of 18) were not assessed with the standardised ASD assessment (i.e. ADOS). Further studies will be required to identify the specific conditions under which individuals with ASD demonstrate weaker sensitivity to perceived eye contact, which would help provide a better understanding of the mechanisms underlying atypical eye contact processing in individuals with ASD, and help develop more effective modes of intervention and support.

In this study, we demonstrated that, in individuals with ASD, eye contact does not facilitate the detection of faces rendered invisible through interocular suppression. These findings suggest that the initial, unconscious registration of eye contact is attenuated in individuals with ASD. This absence of preferential unconscious processing of eye contact in individuals with ASD might be related to their atypical response to eye contact and might contribute to their atypical social development. A promising avenue for future research is to study how this atypical unconscious eye contact effect is related to the daily social communication and social development of individuals with ASD. As a first investigation of unconscious visual processing in individuals with ASD using CFS, the present study might also offer a new approach to uncover the cognitive basis of developmental and psychiatric disorders.

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References


