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Atypical eye contact in autism: models, mechanisms and development

Atsushi Senju* and Mark H Johnson

Centre for Brain and Cognitive Development, Birkbeck, University of London, Malet Street, London WC1E 7HX, UK.

* Corresponding Author: Fax: +44 207 631 6587; E-mail: a.senju@bbk.ac.uk

Abstract

An atypical pattern of eye contact behaviour is one of the most significant symptoms of Autism Spectrum Disorder (ASD). Recent empirical advances have revealed the developmental, cognitive and neural basis of atypical eye contact behaviour in ASD. We review different models and advance a new ‘fast-track modulator model’. Specifically, we propose that atypical eye contact processing in ASD originates in the lack of influence from a subcortical face and eye contact detection route, which is hypothesized to modulate eye contact processing and guide its emergent specialization during development.

Keywords: Autism Spectrum Disorders; Social Cognition; Social Brain; Gaze; Eye Contact; Development; Amygdala

Contents

1. Introduction
 2. How do typical and atypical brains process eye contact?
 - 2.1. Two variants of the affective arousal model
 - 2.2. The communicative intention detector model
 - 2.3. The fast-track modulator model
 3. Current evidence of atypical eye contact in ASD
 - 3.1. Behavioural studies before mid 1990s
 - 3.2. Contextual modulation of fixation on the eyes
 - 3.3. Individual differences in fixation on the eyes within ASD
 - 3.4. Response to direct vs. averted eye gaze
 - 3.5. Developmental basis of atypical eye contact
 4. Conclusions and future directions
 - 4.1. Summary of the current evidences
 - 4.2. Future directions
- Acknowledgments
References

1. Introduction

Direct gaze signals that the gazer is looking at the perceiver. In many non-human species, direct gaze elicits an aversive response, possibly because it signals threat from a predator or a hostile conspecific (Coss, 1978; Emery, 2000). By contrast, in humans direct gaze is arguably the most important platform for social interaction and communication (Csibra & Gergely, 2006; Kleinke, 1986). Some even claim that the depigmentation of human sclera, which highlights the dark iris against white sclera and makes gaze direction clearly visible from distance, is an evolutionary adaptation for ‘eye communication’, (Kobayashi & Kohshima, 1997, 2001). In addition, because eye gaze can be defined geometrically as the relative rotation of eyes from head, torso or the viewer, the stimulus-variables corresponding to gaze direction can be analysed and experimentally controlled more easily than other variables such as facial expressions (Gibson & Pick, 1963). Based on this functional significance as well as the relative ease for controlled stimulus presentation, eye contact processing has been regarded as a ‘model system’ for studying human social interaction and communication, and thus the topic is of interest to researchers in the field of developmental, social and the cognitive neurosciences.

Human eyes attract attention. Eye-tracking studies have revealed that when looking at others’ faces, adults (Yarbus, 1967) and even infants (Maurer & Salapatek, 1976) preferentially fixate to the eyes than other facial features. Not surprisingly, the information in eye region is critical for varying face processing such as the recognition of identity, age, gender and expression (e.g. Gosselin & Schyns, 2001; Whalen et al., 2004; for a review, see Itier & Batty, 2009). Recent neuropsychological studies have suggested the core role of amygdala in such preferential orienting to the eyes. For example, a patient with bilateral amygdala lesions makes fewer spontaneous fixations on the eyes in the context of face-to-face communication (Spezio et al., 2007c) or during performing a facial expression recognition task (Adolphs et al., 2005). Moreover, Adolphs et al. (2005) demonstrated that when the patient was instructed to fixate to the eyes, the performance of facial expression recognition greatly improves to the typical level.

A recent review of the previous literature revealed that eye contact, or perceived direct gaze, modulates concurrent and/or immediately following cognitive processing and/or behavioural responses, a phenomenon we have termed the “eye contact effect” (Senju & Johnson, 2009). For example, perceived eye contact facilitates the performance of face-related tasks such as gender discrimination (Macrae et al., 2002), recognition of face identity (Hood et al., 2003) and detection of gaze direction (Senju et al., 2003, 2005a). Results from neuroimaging studies also indicate that perceived eye contact modulates the activation of social brain network (defined as the cortical and subcortical structures specialized for the processing of social information, such as fusiform gyrus, superior temporal sulcus, medial prefrontal and orbitofrontal cortex and amygdala; for review, see Senju & Johnson, 2009).

Eye contact processing is also potentially an ideal model system for studying the neural, cognitive and developmental basis of atypical social interaction and communication

in Autism Spectrum Disorders (ASD). This is because an atypical pattern of mutual gaze behaviour, or eye contact, is among the most distinguishable manifestation of the qualitative impairment in social interaction in ASD. Since Kanner's first report (Kanner, 1943, 1944), such atypical pattern of eye contact has been reported and discussed in many clinical and experimental settings, including recent studies using eye-tracking methods (Figure 1, Boraston et al., 2008; Dalton et al., 2005; Pelphrey et al., 2002; Spezio et al., 2007c; Sterling et al., 2008, but see also Dapretto et al., 2006; Rutherford & Towns, 2008; van der Geest et al., 2002). Based on this clinical significance, eye contact is currently included in standardised diagnostic criteria such as DSM-IV-TR (American Psychiatric Association, 2000) and ICD-10 (World Health Organization, 1993). In DSM, it is defined as "*marked impairment in the use of multiple nonverbal behaviours (e.g. eye-to-eye gaze, ...) to regulate social interaction and communication*" (American Psychiatric Association, 2000, p.70). It is also possible that an atypical pattern of eye contact in individuals with ASD is relevant to their difficulty in processing other types of social information, such as identity. For example, several studies have reported that weaker activation of fusiform gyrus during face processing in individuals with ASD could be partly attributed to the reduced spontaneous fixation on the eyes: The duration of spontaneous fixation on the eyes correlates with the level of activation in fusiform gyrus (Dalton et al., 2005) and specific instruction to fixate the eyes results in the typical level of activation in fusiform gyrus (Hadjikhani et al., 2004, 2007) in individuals with ASD. As the spontaneous fixations on the eyes are critical to achieve eye contact, these studies strongly suggest a relation between the capacity for eye contact and the processing of other social information.

In addition to the evidence above, retrospective home video analyses of infants who were later diagnosed with ASD have revealed that atypical patterns of eye contact can be observed within the first year of life, well before the age of diagnosis (Baranek, 1999; Clifford et al., 2007; Maestro et al., 2005; Osterling & Dawson, 1994; Osterling et al., 2002; Werner et al., 2005). The presence of atypical eye contact in early development could potentially hamper a wide range of social learning, as eye contact is known to play a critical role in communicative learning (Csibra & Gergely, 2006). For example, in typical development, preferential orienting to eye contact is present even in newborns (Farroni et al., 2002). Perceived eye contact also facilitate the processing of face identity (Farroni et al., 2007) and communicative facial expression (Grossmann et al., 2008) during the first half year of life. In addition, perceived eye contact also plays a critical role in gaze following (Senju & Csibra, 2008) and the encoding of referential gaze (Senju et al., 2008a). Thus, atypical eye contact processing may also contribute to the atypical gaze following behaviour commonly observed in young children with ASD (Charman, 2003; Loveland & Landry, 1986).

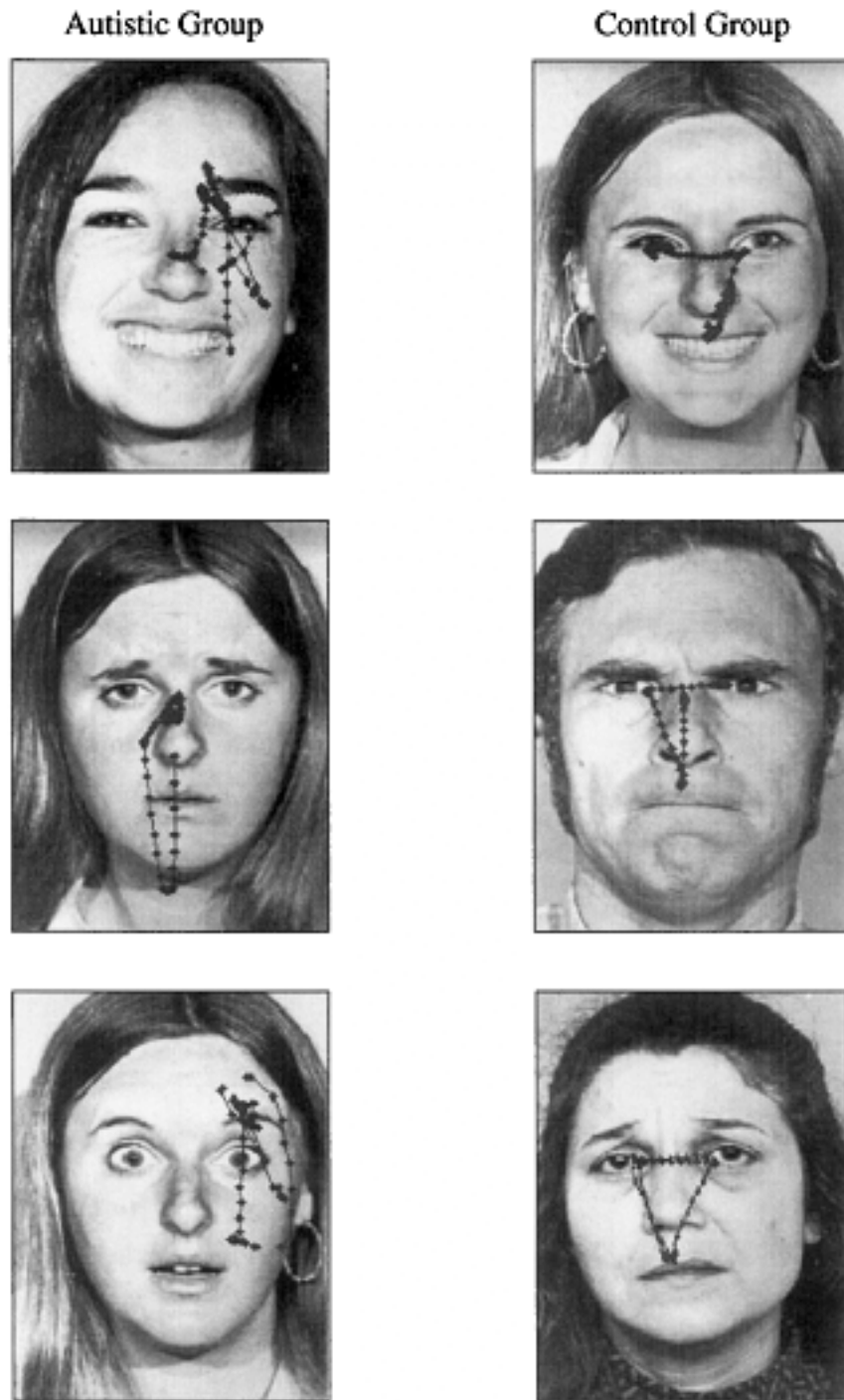


Figure 1. Examples of scanpaths of individuals with autism and typically developed individuals. Reproduced from Pelphrey, K.A. et al., 2002 Visual scanning of faces in autism. *Journal of Autism and Developmental Disorders*, 32, 249-261, with permission.

To date, several models have been proposed to account for the mechanisms underlying, and the development of, atypical eye contact in individuals with ASD. Perhaps not surprisingly, these models closely relate to the different models of the typical development of eye contact processing (Senju & Johnson, 2009). Because ASD is a highly heritable neurodevelopmental disorder (even though its genetic etiology appears to be very complex (Abrahams & Geschwind, 2008; Geschwind, 2008) these ASD models focus on biological susceptibilities, and how they interact with the postnatal environment to affect the emergence of the symptoms.

In this review we will summarize four models of the eye contact effect; two variants of the affective arousal model (the *hyperarousal* model and the *hypoarousal* model), the communicative intention detector model, and the fast-track modulator model. These models clarify some aspects of our understanding of the atypical eye contact observed in ASD, and have relevance for clinical practice. Most critically, these models generate different predictions about aspects of fixation on the eyes, the processing of direct gaze, and its developmental course. Thus, the main aim of the current review is to highlight different models, evaluate these models against the currently available evidence, and to identify the areas where further research is required. We note that there are other review papers that cover cognitive and neural basis of gaze processing in typical development (Frischen et al., 2007; George & Conty, 2008; Itier & Batty, 2009; Senju & Johnson, 2009) and more general gaze processing in ASD (Nation & Penny, 2008). In particular, Buitelaar (1995) published a seminal review paper on the early behavioural studies about atypical eye contact in ASD, but much new evidence has accrued over the past decade.

2. How do typical and atypical brains process eye contact?

2.1. Two variants of the affective arousal model

This model proposes that eye contact directly activates brain arousal systems and thus directly elicits an emotional response. Such emotional arousal is commonly associated with visceral, autonomic and endocrine changes in the body, induced by subcortical structures, particularly the amygdala, and generally activates widespread cortical structures (Adolphs, 2003; Pfaff et al., 2008). In the field of autism research, two distinctive models, the *hyperarousal* model and the *hypoarousal* model, have been developed based on the affective arousal model.

The “hyperarousal model” states that the face and eyes of others are strongly aversive stimuli to individuals with ASD, and thus gaze avoidance is an adaptive response (Corden et al., 2008; Coss, 1978; Dalton et al., 2005; Hutt & Ounsted, 1966; Joseph et al., 2008; Kylliäinen & Hietanen, 2006; Richer & Coss, 1976). Based on this model, some have even made a proposal such as “*Teachers and nurses are recommended not to make efforts to engage autistic children even in friendly eye contact as this provokes more flight behaviour.*” (Richer & Coss, 1976, p.193). Within this model, it is hypothesized that individuals with ASD are in a state of physiological hyperarousal (Corden et al., 2008; Dalton et al., 2005; Hutt & Ounsted, 1966; Joseph et al., 2008; Kylliäinen & Hietanen, 2006) and withdrawal (or anxiety) dominated motivation (E. A. Tinbergen & Tinbergen,

1972; N. Tinbergen & Tinbergen, 1983), which causes them to avoid eye contact. In a recent variant of this idea, Dalton et al. (2005) stated that “*According to this model, face-processing deficits in autism arise from hyperactivation in the central circuitry of emotion that produces heightened sensitivity to social stimuli, leading to characteristic diminished gaze fixation, which in turn results in atypical activation of the fusiform gyrus.* (p. 524)”.

In typical development, the affective arousal model emphasizes the role of the intrinsic reward value of eye contact and its general effect on overall arousal. In typical development, this model hypothesizes that the repeated co-occurrences of eye contact and a wide variety of positive experiences through social interaction attaches the positive reward value to eye contact. Based on this model, the development of ASD can be hypothesized as the failure to form such an association, possibly due to the predominant withdrawal (or anxiety) motivation and/or sustained states of overarousal. Proponents of this model have even claimed that individuals with ASD learn to attach negative valence to eye contact because of its co-occurrences with overly high physiological arousal, which, in turn, causes mal-adaptive learning in a social environment (e.g. Hutt & Ounsted, 1966; E. A. Tinbergen & Tinbergen, 1972).

The hyperarousal model generates several specific predictions. Firstly, individuals with ASD are predicted to actively avoid, rather than passively omit, making eye contact. Such active gaze avoidance should be more prominent in response to perceived eye contact (or direct gaze) than to faces with averted eye gaze. Moreover, active gaze aversion should be present from early infancy, because it is potentially the cause, rather than the consequence, of the atypical development of eye contact. Secondly, fixation on the eyes and/or the presence of eye contact should elicit high physiological arousal, and interfere with the processing of the stimuli which appear concurrently with, and/or immediately following, eye contact.

In addition to the hyperarousal model, other researchers claim that atypical development of eye contact in ASD is based on the “*hypoarousal*”, or hypoactivation of amygdala in early infancy. Specifically, they hypothesized that such hypoactivation in amygdala interferes with attaching positive reward value (Dawson et al., 2005) or emotional saliency (Grelotti et al., 2002) to others during face and eye contact, and thus hampers reinforcement learning about the social environment in general. Within the framework of affective arousal model, the lack of reinforcement learning is hypothesized to later result in the lack of expertise of the social brain network, which fails to learn to efficiently process social stimuli.

This hypoarousal model predicts that individuals with ASD should not show any preference for eye contact from early in development, because of the lack of any attached positive reward value. In addition, either fixation on the eyes or perceived eye contact should have no effect on concurrent behavioural performance, physiological arousal or neural response in the social brain network, because of the lack of developmental expertise to social and communicative environment including eye contact.

2.2. The communicative intention detector model

This model proposes that eye contact directly activates theory-of-mind computation, because it signals the intent to communicate to the perceiver. This model is consistent with the claims that the atypical eye contact observed in individuals with ASD is based on their difficulty in reading others mental states from their eyes (Baron-Cohen, 1995; Baron-Cohen et al., 1997b, 2001a). As an impairment in theory of mind computation is among the most prominent characteristics of ASD (Baron-Cohen et al., 1985; Frith & Frith, 1999), this model has many proponents and is often referred to by researchers investigating atypical gaze processing in ASD (e.g. Baron-Cohen et al., 1997a; Pelphrey & Carter, 2008; Pelphrey et al., 2005; Redcay, 2008).

The communicative intention detector model often involves an assumption about the innate capacity to detect and react to eye contact. For example, Baron-Cohen (1995) proposed the existence of an innate eye direction detector (EDD) module. The function of EDD is to detect eyes and then input to another module that then calculates others mental states, called the theory of mind mechanism (ToMM). Such mechanisms are usually claimed not to require postnatal experience because their function is to guide subsequent learning. Based on this model, individuals with ASD are hypothesized to lack one or both modules. For example, in the specific hypothesis proposed by Baron-Cohen (1995), ASD entails impairment in a shared attention mechanism (SAM), which relays the input from EDD to ToMM. This modular impairment leads to the failure to infer “mentaristic significance of the eyes”, even though the capacity for the decoding of gaze direction (hypothesized to be computed by EDD) may be spared.

The communicative intention detector model predicts that either fixation on the eyes or perceived eye contact should have no effect on concurrent behavioural performance, physiological arousal or neural response in the social brain network, because of defects in the module(s) required to attribute and infer mentalistic significance, including communicative intention, to eye contact. As this model claims that the eye contact effect depends of the inference of communicative intention from eye contact, lack of such inference should lead to the lack of behavioural, physiological or neural response that follows it. In addition, such lack of modulation by eye contact should not change throughout development, as it is generally hypothesized to not be due to learning.

2.3. The fast-track modulator model

We have recently proposed the fast-track modulator model (Figure 2). This model proposes that eye contact processing is mediated by the subcortical face detection pathway (de Gelder et al., 2003; Johnson, 2005b; Johnson & Morton, 1991; LeDoux, 1996; Morris et al., 1999), hypothesized to include superior colliculus (SC), pulvinar and amygdala. The route is fast, operates on low spatial frequency visual information and modulates the cortical processing of social information (Johnson, 2005b), which led LeDoux (1996) to describe it as the “quick & dirty” pathway.

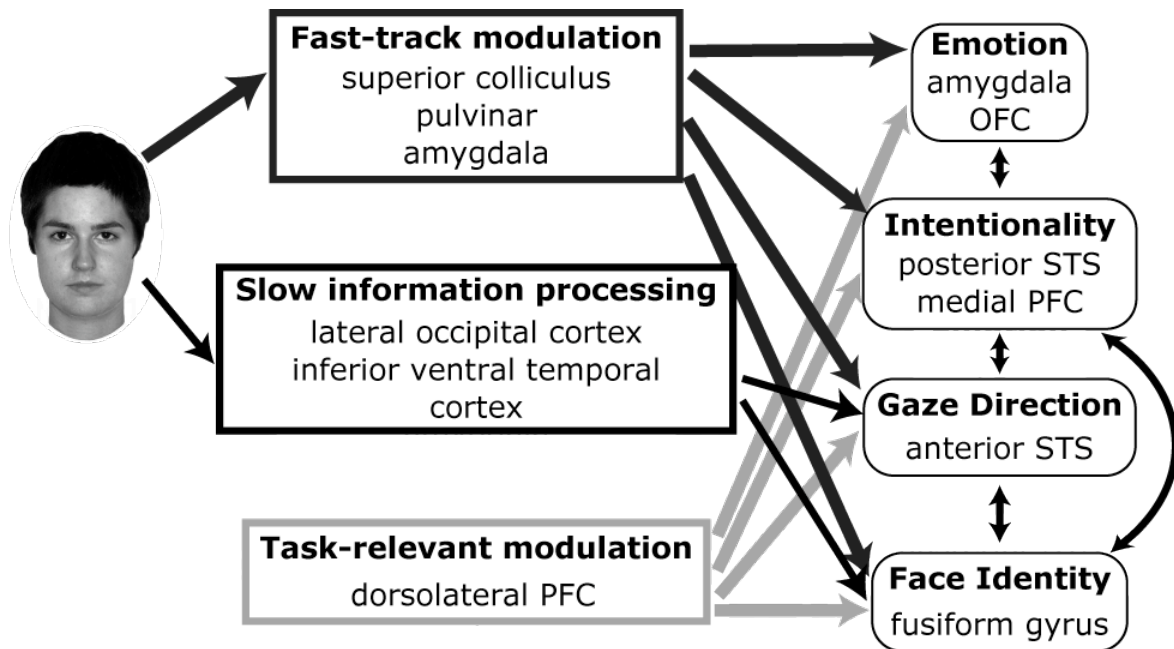


Figure 2. An illustration of fast-track modulator model. Perceived eye contact (upper left) is initially detected by subcortical route, that projects to various regions of social brain network (thick black lines). This signal from subcortical route then interacts with contextual modulation based on the task demands as well as the social context (thick grey lines) to modulate the response of these regions to the following input from a cortical route (thin black lines). These pathways are based on previous analyses on cortical and subcortical face processing, as well as on top-down voluntary attention. Reproduced from Senju, A., Johnson, M.A., in press The eye contact effect: Mechanisms and development. Trends in Cognitive Sciences, 13, 127-134, with permission.

Evidence that the route is fast comes from event-related potential (ERP) and magnetoencephalographic (MEG) studies showing that components associated with a “fast pathway” for face processing can occur at much shorter latencies than those generally associated with the “structural encoding” stage of cortical face processing (such as the N170 and M170) (Bailey et al., 2005). Further, the idea that sub-cortical responses to faces might precede those in the cortex is supported by intra-cranial event-related potentials recorded from epileptic patients with depth electrodes implanted into the amygdala (Krolak-Salmon et al., 2004). These studies support the claim that subcortical face detection pathway shows fast response, which precedes slower cortical face processing pathway.

Evidence that the route processes low spatial frequencies comes from fMRI studies in which the pulvinar, amygdala and SC (which together compose the subcortical face detection pathway) selectively respond to low spatial frequency (LSF) information about faces, and particularly fearful faces (Winston et al., 2003). This sub-cortical route was insensitive to the HSF information about faces that can activate the cortical face processing pathway, such as the fusiform gyrus. Finally, evidence that the sub-cortical route modulates cortical processing comes from several functional imaging studies indicating that the

degree of activation of structures in the sub-cortical route (amygdala, SC and pulvinar) predicts or correlates with the activation of cortical face processing areas, such as fusiform gyrus (George et al., 2001; Kleinhans et al., 2008).

It has been proposed that the sub-cortical route is also responsible for face preferences in newborn infants in whom the cortical visual pathways are only poorly functioning (Johnson, 2005b). Current work is investigating the extent to which the optimal stimuli for eliciting face preferences in newborns are similar to those that maximally activate the adult sub-cortical route (Figure 3) (Farroni et al., 2005; Johnson et al., 1991).



Figure 3. Schematic illumination of the stimuli that might be optimal for activating the hypothesized subcortical route. Such a configuration is optimal for face-detection from a distance, as well as for eye contact detection in close-up. Reproduced from Johnson, M. H. (2005). Subcortical face processing. *Nature Reviews Neuroscience*, 6, 766-774., with permission.

We hypothesize that the combination of this subcortical pathway and contextual modulation given by task demands and social context, implemented as top-down modulation by dorsolateral prefrontal cortex (Banich et al., 2000; Curtis & D'Esposito, 2003), then directly or indirectly modulates key structures involved in the cortical social brain network, such as the fusiform gyrus, STS, medial prefrontal and orbitofrontal cortex (Figure 2). The fast-track modulator model also assumes that infants are born with widespread connections between the subcortical route and cortical structures (Johnson, 2005b). As a consequence, input from eye contact initially activates widespread structures, which combines with architectural bias in cortex (Elman, 1996; Johnson, 2005a) to form specialized connections between a subcortical route activated by eye contact and relevant cortical and subcortical structures during the course of development.

There are several lines of evidence that support this model. Firstly, George et al. (2001) reported that direct gaze increases the functional connectivity, or temporal correlation of regional activity, between amygdala and the fusiform gyrus. This is consistent with the hypothesis in that the amygdala specifically modulates the activation of the fusiform gyrus in response to the perceived eye contact. Secondly, Conty et al. (2007) found that the effect of presence/absence of eye contact in medial prefrontal and orbitofrontal cortex, possibly encoding communicative intention, occurs as early as 150-170 ms after the stimulus onset, possibly preceding in time the response in STS. This

suggests that the mechanism underlying the eye contact effect is fast and occurs before the full and detailed cortical analysis of gaze direction (Calder et al., 2007) and human action (Pelphrey et al., 2004) subserved by STS. Although we cannot fully exclude the possibility that rapid cortical pathways modulated both prefrontal cortex and the STS, it is consistent with the fast-track modulator model in that the subcortical pathway initially detects eye contact, and then subsequently modulates cortical processing. The stimuli that best activate the putative subcortical face processing route are consistent with the idea that this route can support the detection of eye contact also. This is possibly because the pattern of phase contrast that gives optimal input to the subcortical face detector (Figure 3) also preferentially detect eye contact (i.e. direct gaze) than averted gaze when the face is close-up to the viewer (Gilad et al., 2009; Gliga & Csibra, 2007; Johnson, 2005b; Senju & Johnson, 2009).

According to this model, atypical eye contact in ASD could be caused by either an impairment in the subcortical face and eye contact detecting route, or in the cortical architectural biases that interacts with subcortical input to form specialized connections. As a result, individuals with ASD fail to develop the social brain, a network of cortical and subcortical structures specialized for the processing of social information. In a related argument, Schultz (2005) has also hypothesized that congenital abnormality in the amygdala may impair a subcortical face detection network, which then impairs the development of cortical face processing regions such as fusiform gyrus.

The fast-track modulator model predicts absent or weaker modulation from subcortical structures to the social brain network when they process faces with eye contact. The fast-track modulator model does not exclude the possibility that fixation on the eyes, as well as the presence of eye contact, could affect the cortical activation. However these effects are predicted to be atypical, less specialized and different from the eye contact effect in typically developing individuals. Moreover, the fast-track modulator model predicts that the impairment in face and eye contact processing in ASD should be more pronounced when the stimuli only contains low spatial frequency information, or when it is presented very briefly and/or to the peripheral vision.

3. Current evidence of atypical eye contact in ASD

By mid 1990's, most of the studies on atypical eye contact in ASD assessed behaviour in naturalistic or experimental settings. More recently, advances in experimental techniques have led to further studies investigating the neural and cognitive mechanism underlying atypical eye contact processing in ASD. These techniques include eye-tracking, skin conductance response (SCR), electroencephalography (EEG), magnetoencephalography (MEG), structural and functional MRI, as well as refined assessments of behaviour. These techniques have been used either in isolation, or in combination, or with more traditional methods such as clinical evaluation based on parental interview and behavioural observation, and/or self-reported personality traits measured by questionnaires. Moreover, recent advances in prospective developmental studies with infants at high-risk for ASD are beginning to provide a valuable data on the early

development of eye contact processing in ASD. In this section, we first summarize the data reported by the mid 1990's, and then turn to review the more recent studies with advanced techniques. We will examine the extent to which data support each of the three models presented earlier.

3.1. Behavioural studies before mid 1990s

Most of the older studies tested predictions of the hyperarousal model, or whether children with ASD show 'gaze avoidance' in either naturalistic or controlled environments. Initial empirical support for the presence of gaze avoidance came from behavioural studies, which reported that children with autism spend less time looking at a human face in experimental settings (e.g. Hutt & Ounsted, 1966), particularly when the eyes in the face are visible (e.g. Coss, 1978; Richer & Coss, 1976). However, as other concurrent studies failed to replicate gaze avoidance (e.g. Churchill & Bryson, 1972; O'Connor & Hermelin, 1967), many further studies were conducted. Finally, nearly 30 years after the publication of initial reports, Buitelaar (1995) reviewed 11 controlled behaviour observation studies of eye contact behaviour published between 1966 and 1994, and did not find consistent evidence of gaze avoidance in autism. In this seminal review paper, he concluded that "... *in behaviour observation studies autistics demonstrated an absent visual reciprocity and other qualitative differences in social gaze, but not a universal and predominant pattern of gaze avoidance*" (Buitelaar, 1995, p.338). Although it was not denied that gaze avoidance might occur in some individuals with ASD, Buitelaar argued that it was a secondary phenomenon and not a primary cause of autistic pathology. Thus, the results obtained from behavioural observation seem inconsistent with the hyperarousal model. However, these results are consistent with the hypoarousal model, the communicative intention detector model and the fast-track modulator model, all of which hypothesized that individuals with ASD simply *omit*, rather than actively *avoid*, eye contact.

3.2. Contextual modulation of fixation on the eyes

Some of eye-tracking studies have revealed that individuals with ASD fixate others eyes less than typically developing individuals do whether the face is with direct gaze (Boraston et al., 2008; Dalton et al., 2005; Pelphrey et al., 2002; Spezio et al., 2007c) or looking away from the viewer (Klin et al., 2002; Riby & Hancock, 2008, 2009), but other studies failed to replicate (Dapretto et al., 2006; Fletcher-Watson et al., 2009; Rutherford & Towns, 2008; van der Geest et al., 2002) or reported mixed results (Neumann et al., 2006; Speer et al., 2007). Such inconsistencies may result from the differences in task demands and/or the characteristics of stimuli used. In general, reduced fixations on the eyes is most prominent with complex and cognitively demanding face stimuli, e.g. obscuring faces with 'Bubbles' masks (Neumann et al., 2006; Spezio et al., 2007b) or by using dynamic videotape stimuli, including conversations (Klin et al., 2002; Riby & Hancock, 2009; Speer et al., 2007). Several behavioural studies also report that individuals with ASD rely less on the upper part of the face when they process faces (Joseph & Tanaka, 2003; Langdell, 1978; Riby et al., 2009; Rutherford et al., 2007; Spezio et al., 2007a).

These results could be equally well explained by all four models, and thus do not help us discriminate between the models. According to the hyperarousal model, individuals with ASD actively avoid eye contact under increased cognitive demand, as it causes negative arousal and distracts their task-relevant processing. Proponents of the communicative intention model could claim that lack of fixation on the eyes is a compensatory strategy to acquire more information from others' mouth, as they have modular impairment in decoding information from the eyes. For example, Klin et al. (2002) reported that in individuals with ASD greater fixation on the mouth region predicts higher levels of social adaptation and lower levels of autistic social impairment. In contrast, both the hypoarousal model and the fast-track modulator model would encourage the view that individuals with ASD are more readily attracted by visually salient features such as the speaking mouth when it is moving, because their fixation is not adequately guided to eye contact.

3.3. Individual differences in fixation on the eyes within ASD

Several structural and functional MRI studies have revealed relationships between fixation on the eyes and the structure, connectivity and functioning of amygdala. Firstly, Dalton et al. (2005) conducted concurrent recording of fMRI and eye-tracking, and found that the activation of amygdala, as well as fusiform gyrus, correlated with the amount of fixation on the eyes in adolescents and adults with ASD, but not in controls. Participants with ASD who spent a longer time looking at the eyes of face stimuli elicited larger amygdala activation. A later study from the same group reported that the volume of the amygdala also relates to fixation on the eyes in ASD: The larger the amygdala is, the longer they fixated to the eyes of the stimuli (Nacewicz et al., 2006). The authors of these studies claim that the correlation between the structure and function of the amygdala and duration of fixation on the eyes suggests that prolonged fixation on the eyes causes negatively valenced hyperarousal in individuals with ASD (hyperarousal model). In a similar line of research, Kleinhans et al. (2008) reported weaker functional connectivity between amygdala and fusiform gyrus in ASD participants when they process faces compared with neurotypical controls. Kleinhans et al. (2008) also reported in ASD a negative correlation between the level of functional connectivity between amygdala and fusiform gyrus, and the clinical severity measured by ADOS social score and ADI-R social score.

These studies clearly demonstrate the atypical fixation on the eyes and/or face processing in individuals with ASD, and the involvement of the amygdala. At first sight these results could be taken as evidence for the hyperarousal model. However, we argue that these results are not, in fact, consistent with the hyperarousal model for the following reasons. First of all, the involvement of amygdala does not necessarily mean that eye contact elicits a negative valence in individuals with ASD. The amygdala does not only process affectively negative stimuli, but a far wider range of stimuli including positive value ones (Adolphs, 2008; Sergerie et al., 2008). Secondly, Dalton et al. (2005) reported that the amount of fixation on the eyes is positively, not negatively, correlated with the level of activation in fusiform gyrus. Hadjikhani et al. (2004, 2007) also reported that

explicit instruction to fixate on the eyes enhances activation of fusiform gyrus in ASD. These findings are inconsistent with the affective arousal model, which predicts that increased fixation on the eyes should interfere with other aspects of face processing because of negatively valenced hyperarousal. According to the fast track modulator model the amygdala is a critical part of face and eye contact processing (Johnson, 2005b; Senju & Johnson, 2009). Thus, the MRI data described above are consistent with this model. In particular, the weaker functional connectivity between amygdala and fusiform gyrus in ASD (Kleinmans et al., 2008) supports the fast-track modulator model, as it predicts less specialized and less functional modulation from amygdala to fusiform gyrus. Moreover, Hadjikhani et al. (2007) reported that even when individuals with ASD fixate to the eyes, they still show hypoactivation in the social brain network beyond fusiform gyrus, such as STS, inferior frontal cortex and right amygdala. These observations are consistent with the fast-track modulator model, which predicts that the effect of fixation on the eyes on the social brain network is less specialized and less functional in ASD. We argue that the MRI data just described are not consistent with the communicative intention detector model or the hypoarousal model, both of which should predict amygdala hypoactivity in ASD regardless of fixation. This is because in the hypoarousal model, hypoactivation of amygdala is hypothesized to cause the atypical processing of social stimuli. In the communicative intention detector model, the amygdala is hypothesized to be a part of the theory-of-mind network (Baron-Cohen et al., 2000), which is impaired in ASD and therefore fails to respond to the relevant social stimuli.

In addition, Corden et al. (2008) recorded eye movement during a face processing task, and administered a questionnaire about social phobia and anxiety to the same participants with ASD. These authors found that the amount of time fixated on the eyes positively correlated with accuracy to recognize a fearful face as well as the level of self-reported social anxiety. However, neither the duration of fixation on the eyes nor the level of self-reported social anxiety correlated with the level of clinical manifestation of autistic symptoms as measured by Autism Diagnostic Observation Schedule (Lord et al., 2000) or Autism Quotient (Baron-Cohen et al., 2001b). These results indicate that part of individual differences in the level of fixation on the eyes within ASD could be due to the level of comorbid high anxiety (Hobson et al., 1973; Kunihiro et al., 2006), and not by the level of social and communicative impairment *per se*. Previous studies have demonstrated that non-autistic individuals with high social anxiety fixate less to others' eyes, especially when they process expressive face (Horley et al., 2003, 2004, but see also Wieser et al., 2009).

3.4. Response to direct vs. averted eye gaze

Several studies have compared eye movements, SCR, EEG, MEG or behavioural responses for direct gaze and for averted gaze. The affective arousal model should predict stronger physiological arousal and poorer task performance in response to direct gaze than to averted gaze. This is because perceived eye contact should elicit hyperarousal, that leads to active avoidance of these stimuli and interferes with the task performance. By contrast, the communicative intention detector model predicts no differences between the response

for direct and averted gaze, as it hypothesizes that individuals with ASD lack the mechanism to detect the communicative intention from direct gaze. Similarly, the fast-track modulator model predicts no difference in the task performance between direct and averted gaze conditions. This is because eye contact is hypothesized not to modulate the cortical social brain network due to the lack of top-down modulation from subcortical face detection pathway. The fast-track modulator model does not make any specific prediction about the physiological arousal in response to perceived direct or averted gaze.

Behavioural studies have found that typically developing individuals were more accurate (Senju et al., 2003) and faster (Senju et al., 2005a, 2008b) to detect direct gaze than averted gaze. In contrast, individuals with ASD were equally efficient in detecting direct as well as averted gaze. As a result, individuals with ASD were equally efficient in detecting averted gaze as typically developing individuals, but did not show the facilitative effect of eye contact. Thus, typically developing individuals detected eye contact faster and more efficiently than individuals with ASD (Senju et al., 2003).

Several studies have recorded field potentials on the scalp with either EEG or MEG. Firstly, Grice et al. (2005) recorded EEG while children with ASD and control children were passively observing faces with either direct or averted gaze, and found that in children with ASD, direct gaze elicited larger event-related potential (ERP) over posterior regions than averted gaze. By contrast, the ERPs of control children at this age showed no effect of gaze direction. Secondly, Kylliäinen et al. (2006) recorded MEG while children were discriminating whether two faces, presented sequentially, were the same or different. Results showed that children with ASD displayed a stronger evoked signal over a left posterior region in response to direct gaze. By contrast, typically developing children showed significant evoked signal in a right posterior region in response to averted gaze. Senju et al. (2005b) recorded EEG while children perceived either direct or averted gaze from this stimulus sequence. In contrast to the previously described studies, in this study it was typically developing children who showed a larger ERP amplitude for direct than for averted gaze. By contrast, ERPs of children with ASD were not modulated by the presence of eye contact.

Two studies have recorded SCR, an index of physiological arousal, while individuals with ASD observe faces with either direct or averted gaze. In one of these studies, Kylliäinen and Hietanen (2006) presented looming faces with either direct or averted eye gaze while they were asked to maintain fixation on the faces doing nothing, and found that direct gaze elicits larger amplitude of SCR than averted gaze in ASD, but not in controls. Interestingly, the overall SCR was lower in participants with ASD than in control participants, although this effect did not reach significance. Joseph et al. (2008) conducted a similar experiment, but with static images of face stimuli with either direct or averted eye gaze and participants were asked to remember the faces. The results revealed that participants with ASD show significantly higher amplitudes of SCR than control participants, but their SCR was not affected by the gaze direction of the stimuli. In addition, the latter study found that SCR in response to direct gaze, but not to averted gaze, negatively correlated with the performance on a face recognition task.

Finally, Hernandez et al. (2009) used eye-tracking technique to test whether direct gaze elicits more gaze avoidance than averted gaze. Even though the result replicated previous eye-tracking studies in that individuals with ASD fixated less on the eyes than typically developing individuals, no differences in the duration of fixations were observed between direct and averted gaze.

The physiological data are more consistent with the hyperarousal model than the hypoarousal model, as individuals with ASD showed heightened arousal to direct gaze (Kylliäinen & Hietanen, 2006) or generally heightened arousal for the face, regardless of gaze direction (Joseph et al., 2008). In addition, one could claim that larger ERP or MEG components in ASD when they observe direct gaze (Grice et al., 2005; Kylliäinen et al., 2006) was caused by higher physiological arousal. However, other results are also inconsistent with hyperarousal model. Firstly, an eye-tracking study did not find selective avoidance of direct gaze (Hernandez et al., 2009). In addition, behavioural studies found that individuals with ASD are equally good at processing direct and averted gaze (Senju et al., 2003, 2005a). These findings contradict the hyperarousal model, which predicts that perceived eye contact elicits negatively valenced hyperarousal, which then interferes with face processing. More critically, one ERP study, in which participants were explicitly instructed to attend to the eyes, did not find heightened ERP response for direct gaze in individuals with ASD (Senju et al., 2005b). If the larger ERP response for direct gaze in ASD is caused by heightened arousal, instruction to attend to the eyes should cause an even more enhanced response, which was not the case. Finally, note that SCR is an index of physiological arousal, which is independent of its affective valence (Andreassi, 2000). Thus, further studies will be required to examine whether these heightened arousal in response to direct gaze is really 'negatively valenced'. In summary, the current evidence clearly does not support the hyperarousal model.

Both behavioural and eye-tracking results are consistent with the hypothesis generated from the hypoarousal model, from the communicative intention detector model and from the fast-track modulator model, which all predict that direct gaze does not facilitate, or interact with, face processing in ASD. Results from physiological studies and some of the electrophysiological studies are inconsistent with the communicative intention detector model, which does not predict a heightened response to direct gaze than to averted gaze. In contrast, the fast-track modulator model argues that atypically larger ERP and MEG components for direct gaze in individuals with ASD reflects less specialized and less functional effects on the cortical structures. Interestingly, Grice et al. (2005) also reported that the scalp spatial topography of the ERP components for direct gaze in ASD is more similar to those of typical 4-months-old infants, rather than typically developing children in the same age range. This may be because of the lack of developmental specialization in individuals with ASD, which resulted in widespread, less specialized and less functional cortical responses. Nonetheless, this conclusion is inconsistent with hypoarousal model because this model predicts weaker and more limited cortical activation in children with ASD as well as in young infants, as they should not have developed cortical regions that preferentially respond to eye contact due to the lack of extensive reinforcement learning. In addition, the results in Senju et al. (2005b) are also consistent with the fast-track modulator

model, since they observed that perceived eye contact facilitated *both* behavioural performance and the amplitudes of an ERP component in typically developing individuals, but not in individuals with ASD. As we discussed earlier, the fast-track modulator model does not generate specific predictions about physiological arousal induced by perceived direct gaze in ASD.

3.5. Developmental basis of atypical eye contact

Empirical data about eye contact processing in the early development of ASD is very scarce, mainly due to the fact that the majority of individuals with ASD cannot be reliably diagnosed before the age of 3 years (Cox et al., 1999; Landa, 2008). However, recent studies of the early development of ASD, especially the prospective studies with high-risk infants for ASD, are beginning to add significantly to the data obtained by retrospective studies. For example, Merin et al. (2007) investigated the pattern of face fixation with infants at high-risk for ASD while they engaged in a face-to-face interaction with their caregivers via a closed circuit TV-video system. They did not find any group differences in the duration of gaze aversion from the TV screen, or overall face fixation measured by an eye-tracking system. The only difference observed in this study was that a subgroup of high-risk infants fixated on the mouth more than the eyes. However, a follow-up study revealed that this increased mouth fixation did not predict later diagnosis of ASD in high-risk or in control infants (Young et al., in press). Other studies also failed to find reduced face fixation or increased face aversion in infants at high risk for ASD (Cassel et al., 2007; Yirmiya et al., 2006, for a review, see Elsabbagh & Johnson, 2007). A very recent study by Chawarska and Shic (in press) confirmed these patterns in toddlers diagnosed with autism at the age of 2 years old. In their longitudinal study, 2-year-old children with autism showed similar amount of fixation on the eyes as typically developing children, even though they showed less fixation on the mouth than typically developing children. In contrast, at the age of 4 years old, children diagnosed with autism spent less time looking at the inner parts of the face including eyes, mouth and nose than typically developing children. However, the difference in the amount of fixation on the eyes between groups did not reach significance.

Elsabbagh et al. (2009) recorded EEG from high-risk infants as well as from low-risk control infants while they watched faces with either direct or averted eye gaze, and conducted ERP and time-frequency analysis (TFA). Similarly to Merin et al. (2007), the behavioural measures taken indicated no selective aversion of direct gaze in high-risk infants. In addition, ERP analysis revealed that an early ERP component, which reflects the initial stages of visual processing, appeared faster for direct gaze than for averted gaze in both high-risk and control infants. However, they also found two major group differences in EEG response. Firstly, a later ERP component (P400), which is known to relate to face processing and is strongly influenced by top-down modulation, has a longer latency in response to direct gaze in high-risk infants. Secondly, TFA analysis revealed clearly distinguished and temporally sustained high-frequency oscillatory activity in the gamma-band frequency for direct gaze compared to averted gaze in control infants. In contrast,

high-frequency oscillatory activity in gamma-band for direct gaze compared to averted gaze in high-risk infants was delayed and less persistent. These results suggest that atypical eye contact processing in high-risk infants relate to the top-down modulation (as indicated by the slower P400 latency) and task-relevant synchronization of brain activations (as indicated by the lack of differential gamma-band activation in response to eye contact).

We argue that these three studies support the fast-track modulator model over the other models. Infants at high-risk for ASD do not show any avoidance of the face or for eye contact, and they show sensitivity to direct gaze comparable to control infants, at least in the initial stages of visual processing. However, these high-risk infants show atypical brain responses at later latency ERP components and in differential gamma-band oscillations, which suggests atypical top-down modulation and/or synchronization of neural activities in response to perceived direct gaze. Overall, these results suggest that infants at high-risk for ASD are as sensitive to direct gaze as infants at the low-risk for ASD, but that perceived direct gaze fails to modulate cortical face processing in the same way for high-risk infants.

Note that these studies with high-risk infants for ASD are literally in their ‘infancy’ and have limitations. One of the major limitations at present is that most of these studies have yet to report the follow-up diagnoses of these infants and to examine the relationship between these early behavioural and neurophysiological phenotypes and the later diagnosis of ASD. Such follow-up data will shed light on the characteristics of ASD in early infancy. Until then, these studies only allow conclusion about population differences, possibly reflecting the broader autism phenotype (Baron-Cohen & Hammer, 1997; Dawson et al., 2002), who shares some of the autistic phenotypes but do not fall into the diagnostic criteria for ASD.

4. Conclusions and future directions

4.1. Summary of the current evidences

In the current paper we have outlined four major models of the mechanisms underlying, and the development of, atypical eye contact processing in ASD. The models, in turn, reflect those of the typical development of eye contact processing (Senju & Johnson, 2009). Several inferences can be drawn from the evidence available so far. First, the empirical findings are still inconclusive as to whether fixation on the eyes is reduced in ASD compared to typically developing individuals. The available evidence at present suggests that the reduced fixation on the eyes in ASD is most prominent under conditions of high cognitive demand. In addition, recent developmental studies suggested that such reduced fixation on the eyes may not be present early in development. Moreover, the atypical structure, activation and/or connectivity of the amygdala is involved in atypical face processing, which could contribute to the atypical orienting to the eyes observed in ASD. However, these observations cannot explain how the atypical amygdala functioning relates to the atypical fixation on the eyes. Second, individuals with ASD show sensitivity to perceived eye contact in some of physiological and electrophysiological studies, but these physiological and electrophysiological responses are not reflected in measures such as the speed and accuracy of manual response in face/gaze processing or the spontaneous

fixation of the eyes. The discrepancy between studies suggests that the physiological and neurophysiological responses are less specialized and/or less functional, and do not either facilitate or interfere with the behavioural response to eye contact. Third, the emerging evidence reported from infants at high risk for ASD, as well as from toddlers with ASD, suggests widespread and less specific response to eye contact, but not active avoidance or a complete lack of response. These data from early development appear to support the hypothesis derived from the fast-track modulator model: Atypical eye contact behaviour in ASD is based on the atypical specialization of the social brain network, due to atypical functioning of subcortical face and eye contact detection route, which includes amygdala, and/or its atypical communication with the cortical and subcortical social brain network, from early in development. However, the apparently inconsistent results between different studies, as well as the scarcity of empirical data on young infants at-risk for ASD, precludes us from drawing any definitive conclusions. In the next and the last section, we discuss some of the major questions that require further investigation, and propose potential studies that would help to clarify the nature of atypical eye contact processing in ASD.

4.2. Future directions

First, it is still unclear how the genetic and epigenetic factors contributing to ASD lead to the atypical interaction between subcortical structures, including the amygdala, and other cortical and subcortical structures involved in the social brain network. The fast-track modulator model proposes that atypical development results in the lack of specialization of, and within, the social brain network. Within this framework, impairment in the amygdala and/or its functional communication with other cortical and subcortical structures in early infancy could lead to a lack of selective modulation in response to social stimuli including the face and eyes, which then results in weaker functional specialization of the cortical network that processes social stimuli. In contrast, both versions of the affective arousal model emphasize the role of attached reward value to social stimuli (either negative (the hyperarousal model) or neutral (the hypoarousal model)) and this hampers reinforcement learning about the social environment in general. Further studies with infants with high risk for ASD will be required to assess these theories. For example, it would be important to test whether direct gaze *per se* has a positive reward value in early infancy, and whether it has a neutral or even negative reward value for infants at high risk for ASD.

A second line for future studies will be whether (and/or how) atypical eye contact processing in ASD relates to other, and particularly non-social, symptoms. For example, Jemel et al. (2006) proposed that individuals with ASD have a bias to orient toward the local aspects of the face, which then interferes with their spontaneous face (and possibly eye contact) processing. The same authors also claim that this locally oriented bias is based on an atypical pattern of cortical specialization, which includes overall superior functioning of primary (or posterior) perceptual cortex relative to high-order (or anterior) perceptual cortex (Mottron et al., 2006). Actually, this theory accords well with the fast-track modulator model, as it predicts atypical cortical specialization for social processing throughout the course of development. Further studies will be necessary to investigate the

characteristics of visual processing in infants at high risk for ASD, and how they interact with the development of the social brain network. For example, McCleery et al. (2007) reported higher sensitivity to luminance contrast in high-risk infants for ASD, which suggest atypical sensitivity of magnocellular pathway. As the magnocellular pathway provides the primary input to the subcortical pathway, these authors propose that atypical functioning of magnocellular pathway affects the specialization of the social brain network, as well as other atypical perceptual profile in ASD.

In a third line of investigation it will be important to clarify the developmental basis of active gaze avoidance, which is unlikely to be universal among individuals with ASD, but could be present in a subgroup of ASD. Interestingly, reports of reduced fixation on the eyes are mostly limited to the adolescents or adults with ASD (Dalton et al., 2005; Klin et al., 2002; Neumann et al., 2006; Pelphrey et al., 2002; Spezio et al., 2007c). By contrast, some studies with younger children (van der Geest et al., 2002) or infants (Elsabbagh et al., 2009; Young et al., in press) failed to observe such behaviour. Thus, it is possible that active gaze avoidance is one of the secondary symptoms for some of the individuals with ASD that in most cases appears during adolescence or later, particularly when they have a high level of social anxiety (Corden et al., 2008). Further studies will be required to investigate the developmental course of active gaze avoidance in ASD, and its relations with comorbid symptoms such as social anxiety.

Finally, we believe it will be beneficial to test other predictions derived from the fast-track modulator model. For example, the fast-track modulator model predicts that the atypical response to eye contact in ASD should be most prominent for low spatial frequency stimuli, and when the stimuli are presented briefly and/or to the peripheral vision. As other studies have reported that individuals with ASD have difficulty in recognizing face identity and facial expression from low spatial frequency stimuli (Deruelle et al., 2004, 2008), future studies will be important to examine the effect of eye contact in ASD using low spatial frequency stimuli. Similarly, several studies have reported that individuals with ASD shows difficulties in gaze processing (Wallace et al., 2006) as well as in the processing of facial expression (Clark et al., 2008), when the stimuli were presented only briefly. Thus, it is also possible that the atypical response to eye contact could be most prominent in briefly presented stimuli. In addition, as we have discussed previously (Senju & Johnson, 2009), the eye contact effect is present from early infancy and seems to modulate gaze following behaviour (Senju & Csibra, 2008; Senju et al., 2008a). Thus, it would be critical to examine how and whether the input to the subcortical face detection route and/or its interaction with other parts of social brain network would affect the atypical development of gaze following behaviour in ASD (Charman, 2003; Loveland & Landry, 1986).

Eye contact behaviour is an ideal model system to investigate the mechanisms underlying, and the development of, human social interaction and communication, both in typically developing individuals as well as in individuals with ASD. Advances in this field have revealed the neural, cognitive and developmental basis of atypical social interaction and communication in individuals with ASD. Further studies on eye contact processing in

ASD, based on clear developmental models, will be beneficial for the understanding of the nature of impairment in social interaction and communication in ASD, and may help develop viable intervention and support programs. Moreover, such studies will also shed light on the typical development of eye contact behaviour, as well as the development of human social interaction and communication.

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