The eye contact effect: mechanisms and development

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The eye contact effect: mechanisms and development

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Abstract

The “eye contact effect” is the phenomenon that perceived eye contact with another human face modulates certain aspects of the concurrent and/or immediately following cognitive processing. In addition, functional imaging studies in adults have revealed that eye contact can modulate activity in structures in the social brain network, and developmental studies show evidence for preferential orienting toward, and processing of, faces with direct gaze from early in life. We review different theories of the eye contact effect and advance a ‘fast-track modulator’ model. Specifically, we hypothesize that perceived eye contact is initially detected by a subcortical route, which then modulates the activation of the social brain as it processes the accompanying detailed sensory information.

Keywords: gaze, eye contact, social brain, amygdala, development

Direct gaze signals that the gazer is looking at the perceiver. In many species, the perception of direct gaze elicits an aversive response¹, probably because it is a salient signal for potential threat. In humans, by contrast, eye contact provides a foundation of communication and social interaction²,³. Some researchers argue that the depigmentation of the human sclera, which does not exist in other primate species, has evolved for effective communication and social interaction based on eye contact⁴.

Recent advances in the fields of developmental, social and cognitive neurosciences have revealed a network of structures involved in human social interaction and communication, sometimes termed “the social brain”⁵-⁷. The social brain is the cortical and subcortical network of regions, including ventral and medial prefrontal cortex, superior temporal gyrus, fusiform gyrus, cingulate gyrus and amygdala⁵, that are specialized to process social information such as the face⁸, gaze⁹, biological motion⁹, human action¹⁰, goal-directedness¹⁰, theory of mind¹¹ and empathy¹². Whilst it is commonly agreed that eye contact modulates the development and activation of the social brain network, the precise mechanisms and developmental processes involved remain unclear. In this review we summarize research findings on eye contact processing, before addressing issues about the mechanisms underlying the effects of eye contact on the social brain network and its development.

Eye contact modulates the social brain

Psychological studies have revealed that perceived eye contact modulates cognition and attention. For example, a series of studies adopted visual search tasks to test whether human observers are faster to detect a face¹³,¹⁴ or eyes¹⁵ with direct gaze than those with averted gaze. In these studies, participants are required to judge whether the target image is
present or absent among distracters (that are the same images as targets except for their gaze direction). Results show that participants were faster to detect the presence/absence of the target with direct gaze than those with averted gaze. In addition, direct gaze facilitates other face-related tasks such as gender discrimination \(^{16}\), and the encoding and decoding of identity \(^{17}\). Direct gaze also holds attention \(^{18}\): the detection of peripheral targets becomes slower when participants fixate on a face with direct gaze than that with averted gaze. It has also been reported that a stranger gazing directly at the perceiver increases autonomic arousal in adults \(^{19}\). In the remainder of this paper, we will refer to this general effect of perceived direct gaze as the ‘eye contact effect’. That is, the eye contact effect is defined as the phenomenon that perceived eye contact modulates the concurrent and/or immediately following cognitive processing and/or behavioural response.

Table 1. Neuroimaging studies compared direct vs. averted gaze processing.

<table>
<thead>
<tr>
<th>Study</th>
<th>Method</th>
<th>Task</th>
<th>Orientation</th>
<th>Movement</th>
<th>Expression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calder et al. (^{21})</td>
<td>PET</td>
<td>Eyebrows</td>
<td>Oriented</td>
<td>Static</td>
<td>Neutral</td>
</tr>
<tr>
<td>Conty et al. (^{25})</td>
<td>ERP</td>
<td>Gaze</td>
<td>Front view</td>
<td>Dynamic</td>
<td>Neutral</td>
</tr>
<tr>
<td>Engell &amp; Haxby (^{52})</td>
<td>fMRI</td>
<td>Identity</td>
<td>Front view</td>
<td>Static</td>
<td>Expressive</td>
</tr>
<tr>
<td>George et al. (^{22})</td>
<td>fMRI</td>
<td>Gender</td>
<td>Front view</td>
<td>Static</td>
<td>Neutral</td>
</tr>
<tr>
<td>Hoffman &amp; Haxby (^{81})</td>
<td>fMRI</td>
<td>Passive</td>
<td>Front view</td>
<td>Static</td>
<td>Neutral</td>
</tr>
<tr>
<td>Kampe et al. (^{28})</td>
<td>fMRI</td>
<td>Passive</td>
<td>Front view</td>
<td>Static</td>
<td>Neutral</td>
</tr>
<tr>
<td>Kawashima et al. (^{29})</td>
<td>PET</td>
<td>Gaze</td>
<td>Oriented</td>
<td>Dynamic</td>
<td>Neutral</td>
</tr>
<tr>
<td>Pageler et al. (^{23})</td>
<td>fMRI</td>
<td>Gaze</td>
<td>Front view</td>
<td>Static</td>
<td>Neutral</td>
</tr>
<tr>
<td>Pelphrey et al. (^{26})</td>
<td>fMRI</td>
<td>Gaze</td>
<td>Oriented</td>
<td>Dynamic</td>
<td>Neutral</td>
</tr>
<tr>
<td>Sato et al. (^{30})</td>
<td>fMRI</td>
<td>Gender</td>
<td>Oriented</td>
<td>Dynamic</td>
<td>Neutral</td>
</tr>
<tr>
<td>Schilbach et al. (^{27})</td>
<td>fMRI</td>
<td>Gaze &amp;</td>
<td>Front view</td>
<td>Dynamic</td>
<td>Expressive</td>
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<td></td>
<td></td>
<td>Expression</td>
<td>or Oriented</td>
<td></td>
<td>or Neutral</td>
</tr>
<tr>
<td>Wicker et al. (^{51})</td>
<td>PET</td>
<td>Expression</td>
<td>Front view</td>
<td>Dynamic</td>
<td>Neutral</td>
</tr>
<tr>
<td>Wicker et al. (^{24})</td>
<td>PET</td>
<td>Gaze or</td>
<td>Front view</td>
<td>Dynamic</td>
<td>Expressive</td>
</tr>
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</table>

We summarized the neuroimaging method (event related potential (ERP), functional magnetic resonance imaging (fMRI) or positron emission topography (PET)), the behavioural task performed by the participants during recording (discrimination of eyebrow thickness (Eyebrow), facial expression discrimination (Expression), gaze direction discrimination (Gaze), identity matching (Identity), gender discrimination (Gender) or passive viewing (Passive)), facial orientation (in front view (Front) or laterally oriented (Oriented)), movement of stimulus face (static or dynamic) and facial expression (neutral or expressive). We only included studies that directly compare brain activity between direct and averted gaze processing and that did source localization. Kylliäinen et al. \(^{68}\) was not included in the analysis because they did not find any localized activation that discriminates between direct and averted gaze processing in adult participants.
Figure 1. Cortical and subcortical regions that showed the eye contact effect in more than one study. The Fusiform gyrus (A), particularly in the right hemisphere, shows larger activation for faces with direct gaze than those with averted gaze, when direct gaze accompanied the abrupt onset of static faces \cite{21,22}. Two studies \cite{21,24}, which explicitly instructed participants to actually attend to the eye region, have reported increased activity in
the anterior part of the right STS regions (B) in response to direct gaze. The posterior part of right STS region (C) is also sensitive to the presence/absence of eye contact in two studies which used dynamic stimuli (D) and orbitofrontal cortex (E). In other studies, by contrast, the activation in medial prefrontal cortex was greater for averted gaze than for direct gaze (D’). Finally, two studies reported increased activation in right amygdala (F) in response to direct gaze, based on region-of-interest analyses. This figure is created with Caret software (see: http://brainmap.wustl.edu/caret) and projected on Human PALS-B12 atlas.

Functional neuroimaging has also been used to compare the patterns of brain activation in response to the perception of direct gaze compared to that with averted gaze (Table 1, see also for the review of brain activation in response to the perception of the gaze in general). In reviewing these studies, six regions have been reported to show differential activity between direct and averted gaze in more than one study (Figure 1 and Box 1): fusiform gyrus, anterior and posterior parts of superior temporal sulcus (STS), medial prefrontal and orbitofrontal cortex and amygdala. Taken together, these studies reveal that perceived eye contact (a) enhances the activation of components of the social brain network, but (b) this activation interacts with task demands, as well as the social context, to influence precisely which regions in the social brain network are activated.

How does eye contact activate the social brain?

Currently most of the accounts of the eye contact effect that have been given are either insufficiently detailed to generate testable predictions, or are specific to a particular experimental paradigm. Two general accounts have often been invoked to explain the mechanisms underlying the eye contact effect. In this section, we will summarize these two models and advance a third model that we believe to be at least equally consistent with the majority of the results on the eye contact effect. Although current empirical evidence cannot rule out any of these accounts at this early stage of research, our aim is to highlight differences between models, to identify areas in which empirical evidence could differentiate between them, and hence to stimulate further research.

The affective arousal model. Some have argued that eye contact directly activates brain arousal systems and/or elicits a strong emotional response. This raised arousal or emotional level then influences subsequent perceptual and cognitive processing. Although the neural mechanisms underlying this effect have not been specified, 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. This view accords with the introspective impression of ‘being looked at’, and is consistent with earlier findings that eye contact elevates physiological arousal. In addition, the view is consistent with an integrative model including other aspects of face processing such as expression and attractiveness, and which is based on the detection and evaluation of reward value and reward intensity.

However, we consider that this general account of the eye contact effect has several limitations. Firstly, if the mechanism underlying the eye contact effect is general arousal, the effects should be more widespread and unselective in terms of activation within the cerebral cortex. This prediction does not fit well with the highly selective enhancement of the relevant regions within the social brain network found in previous studies. Secondly, results of recent psychophysiological studies are incongruent with the emotional arousal theory. For example, Kampe and colleagues examined the effect of autonomic arousal on the eye contact effect.
by measuring pupil dilation, which is a reliable index of arousal. Their results, however, revealed that autonomic arousal could not account for the effect of eye contact on the increased activity in medial prefrontal cortex. More recent studies indicated that autonomic arousal to eye contact is restricted to the prolonged presentation of live humans and does not occur in response to static images. Thus, it appears unlikely that the affective arousal model on its own can fully explain the eye contact effect.

The communicative intention detector model. Other researchers have argued that eye contact directly activates theory-of-mind computations or a pedagogy brain system, because it signals the intent to communicate with the perceiver.

Some proponents of the communicative intention detection model have claimed that the computations underlying this function are subserved by specific cortical structures such as medial prefrontal cortex, temporal pole, superior temporal sulcus and/or the temporal parietal junction. Recent studies also suggested that the fusiform gyrus can be a component of this theory-of-mind network. This model is a strong contender to account for the eye contact effect, as the cortical and subcortical structures involved in theory-of-mind computation overlap substantially with the regions relevant to eye contact detection. However, we suggest that the model cannot fully account for the range of observations associated with the eye contact effect, particularly with regard to why only parts of the network, rather than the whole network, are activated for eye contact in each study, and the varying patterns of activation depending on task demands and context.

The first-track modulator model. In order to better explain the range of phenomena associated with the eye contact effect, we propose an alternative fast-track modulator model (Figure 2). This model proposes that the eye contact effect is mediated by the subcortical face detection pathway hypothesized to include the superior colliculus, pulvinar and amygdala. This route is fast, operates on low spatial frequency visual information, and modulates cortical face processing, which led LeDoux to describe it as the “quick & dirty” pathway.

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). 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.

Evidence that the route processes low spatial frequencies comes from fMRI studies in which the pulvinar, amygdala and superior colliculus respond to low spatial frequency (LSF) information about faces, and particularly fearful faces. This sub-cortical route was insensitive to the HSF information about faces that can activate the fusiform cortex. 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.

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. 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 (see Box 2).

Figure 2. An illustration of the fast-track modulator model. Perceived eye contact (upper left) is initially detected by subcortical route, that projects to various regions of social brain network (blue lines). This signal from subcortical route then interacts with contextual modulation based on the task demands as well as the social context (green lines) to modulate the response of these regions to the following input from a cortical route (black lines). These pathways are based on previous analyses on cortical and subcortical face processing, as well as on top-down voluntary attention.

We hypothesize that the combination of this subcortical pathway and contextual modulation given by task demands and social context, which is implemented as a top-down modulation by dorsolateral prefrontal cortex, directly or indirectly modulates key structures involved in the cortical social brain network, such as the fusiform gyrus, STS, medial prefrontal and orbitofrontal cortex.

There are several lines of evidence that support this model. Firstly, George et al. reported that direct gaze increases the functional connectivity, or temporal correlation of regional activity, between the amygdala and the fusiform gyrus. This is consistent with the hypothesis that the amygdala specifically modulates the activation of the fusiform gyrus in response to perceived eye contact. Secondly, Conty et al. found that the effect of presence/absence of eye contact in medial prefrontal and orbitofrontal cortex, possibly encoding communicative intention (Figure 2), occurs as early as 150-170 ms after the stimulus onset, possibly preceding in time the response in the 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 and human action subserved by the STS. Although we cannot fully exclude the possibility that rapid cortical pathways modulated activity in both the prefrontal cortex and the STS (see also Box 5), these findings are 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 (Box 2).

One of the advantages of the fast-track modulator model is that it generates several
specific predictions about the eye contact effect. Firstly, as the putative subcortical route is
activated by low spatial frequency information, the eye contact effect should be dependent on
the presence of mid or low spatial frequency information of eye contact in close proximity,
and should be diminished when only high spatial frequency information is provided.
Secondly, the eye contact effect should be restricted to, or centered on, a specific subset of
cortical and subcortical structures. This contrasts with the prediction of the emotional arousal
model that there are non-specific changes in widespread cortical regions. Thirdly, such fast-
track modulation may compete with, and sometimes even be overcome by, other sources of
modulation, such as top-down attention based on instruction and/or task demands. That is,
in adults task demands may reduce or eliminate the eye contact effect under the influence of
task-relevant attention.

The developmental basis of the eye contact effect
A number of studies have revealed that sensitivity to eye contact is present even in
newborns. Neuroimaging studies have also demonstrated that eye contact
modulates cortical activation in infants as young as 4 months. This suggests that human
infants are equipped with a bias to detect and orient toward faces that make eye contact with
them. Several other studies support the view that the eye contact effect is present from early
in life in humans.

The question remains how the eye contact effect develops in human ontogeny. The
three models presented above suggest different hypotheses on the development of the eye
contact effect. The affective arousal model emphasizes the role of the reward value of eye
contact and its non-specific effect on general arousal. Thus, it is conceivable that the eye
contact effect could emerge as a result of extensive exposure to the co-occurrences of eye
contact and a wide variety of positive experiences through social interaction and
communication, which then attaches reward value to eye contact. In contrast, the
communicative intention detector model often involves the innate capacity to detect and react
to eye contact. For example, Baron-Cohen proposed an innate module called the eye
direction detector (EDD). The function of EDD is to detect eyes and inputs to another module
that then computes mental states, called the theory of mind mechanism (ToMM). Relatedly,
other theorists hypothesize that infants are born with mechanisms to detect ostention (i.e.
manifestation of intention to communicate to the perceiver) from perceived eye contact,
which then sensitizes the perceiver to the following referential communication. Such
systems are generally claimed not to require postnatal experience since their function is to
guide subsequent learning. In contrast, the fast-track modulator model assumes that infants
are born with widespread connections between the subcortical route and cortical structures.
As a consequence, input from perceived eye contact initially activates widespread cortical
structures, which combines with architectural biases to form specialized connections
between the subcortical ‘eye contact detector’ and relevant cortical and subcortical structures
during the course of development.

These three models differ somewhat in the role attributed to the postnatal
environment. The affective arousal model claims that postnatal experience determines the
reward value of eye contact and the general arousal it induces. The communicative intention
detector model assumes that postnatal experience has no effect on the maturation of the
mechanism underlyng the eye contact effect. The fast-track modulator model claims that
postnatal experience interacts with the innate architectural bias to narrow down the initially
widespread effect of eye contact. Due to the paucity of current data from developmental
studies, differentiating between these accounts on the basis of developmental studies will be
important in the future.

Looking forward

As Kleinke 3 noted, “the significance of eyes in human relationship fascinated writers and philosophers (as well as scientists) for centuries” (p. 78, parenthesis inserted by the authors). Great advances in adult functional neuroimaging studies and infant behavioural studies in the last decade have opened the study of the eye contact effect within the field of developmental cognitive neuroscience. These advances and accumulating empirical findings have enabled us to revisit the old question of how eye contact works in human communication. In future studies it will be beneficial to clarify the neural and computational mechanism underlying the eye contact effect, its typical development (Box 3) as well as atypical development in autism spectrum disorder (Box 4) and its functional specialization (see Box 5 for outstanding questions). These studies will lead to a better understanding of the cognitive, neural and developmental basis of human communication and social interaction.

Acknowledgments

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References

communicative signals. *Curr. Biol.* 18, 668-671
Box 1 Brain regions activated in response to direct gaze

Following five regions consistently showed larger activation for direct gaze than for averted gaze in two or more studies.

**Fusiform gyrus (FG).** When direct gaze accompanies the abrupt onset of static faces, the FG shows larger activation for faces with direct gaze than those with averted gaze. However, the effect is absent when an already-present face shifts its gaze toward the participants or when participants are required to attend to the identity of the stimulus faces. These results suggest that the increased fusiform activation relates to the enhanced face encoding at initial detection, but that it can be masked when participants are instructed to attend to face identity.

**Anterior part of the right STS region (aSTS).** Two studies, which explicitly instructed participants to actually attend to the eye region, have reported increased activity in the anterior part of the right aSTS in response to direct gaze. These findings suggest that instructed attention to the eyes, and the presence of eye contact, facilitate the encoding of gaze direction in this region. Similar to FG, instructed attention to gaze direction may mask the eye contact effect in aSTS. (see main text and Figure 2 for task-dependent effects)

**Posterior part of right STS region (pSTS).** This region is also sensitive to the presence/absence of eye contact as demonstrated in three studies that used dynamic stimuli. However, other studies did not find the effect. These results may indicate that perceived eye contact activates right pSTS, but only in the context that the perceiver recognizes it as being genuinely social and/or communicative (see also).

**Medial prefrontal cortex (mPFC) and orbitofrontal cortex (OFC).** Several studies reported greater activation in response to direct gaze in the right mPFC and OFC. Note that three of these studies presented dynamic facial expressions and two of them required participants to decode the intention of the presented face to communicate, which could have influenced the greater activation for direct gaze in these regions. In other studies, by contrast, the activation in mPFC was greater for averted gaze than for direct gaze, even though the focus of activation was slightly posterior to the areas which show greater activation for direct gaze.

**Amygdala.** Finally, three studies reported the effect in amygdala. It is not clear why these three studies found amygdala responses whereas other studies did not find the effect. One possibility is that, since the amygdala is a small structure relative to the cortical areas discussed above, some current neuroimaging methods are not sensitive enough to detect these effects.
Box 2: CONSPEC: A mechanism for eye contact detection?

Newborn infants preferentially orient to facial configurations \(^{53,54}\), as well as to faces with direct gaze \(^{48}\). Farroni et al. \(^{53}\) demonstrated in newborns that their preferences are consistent with the hypothesized CONSPEC \(^{54}\). Specifically, CONSPEC is activated by a stimulus with a configuration of three dark blobs against a lighter background that corresponds to the areas of shadow and reflected light in a naturally lit face (see Figure I). As Gliga and Csibra \(^{55}\) (see also \(^{38}\)) argued, CONSPEC is not only a mechanism for detecting facial configuration with the putative subcortical route, but at close distance may be the best representation to detect eye contact (Figure I). In addition, adult neuroimaging studies also suggested the existence of a subcortical eye contact detection route. For example, Whalen et al. \(^{56}\) demonstrated that the extent of white sclera field surrounding the iris of the eyes regulates amygdala activity, with the wider sclera field associated with fearful faces eliciting greater activation. Interestingly, fearful facial expressions are also known to enhance the activity of the fusiform gyrus in response to the sudden onset of facial stimuli \(^{33}\), just like a face with direct gaze \(^{21-23}\). Adolphs et al. \(^{57}\) also reported that in one patient with bilateral amygdala damage, the impairment in recognizing fearful facial expression is attributable to the lack of spontaneous fixation to the eyes. These studies suggest that the amygdala is involved in the fixation to the eyes, as well as the detection of eye contact.

Thus, we suggest that the rapid detection of eye contact at close proximity is a function shared with face detection, dependent on distance and spatial frequency, by the subcortical face route. Further studies will be required to establish whether these two functions are implemented as a single mechanism, or are dissociable as two separate, but highly interdependent, neural routes.

**Figure I.** 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 with permission from \(^{38}\).
Box 3: The eye contact effect in young infants.

Recent advances in infant studies suggest that perceived eye contact modulates the concurrent and/or immediately following cognitive processing or behavioural response of infants as well as adults. Firstly, Farroni et al. 58 presented faces either with direct gaze or with averted gaze to 4-month-old infants, and tested whether the presence of eye contact affects memory for facial identity as it does in adults. Results revealed that infants in their study discriminated the previously presented face from a novel face only when it had been previously presented with direct gaze. Secondly, Farroni et al. 59 reported that a period of preceding eye contact is required in order for 4-month-old infants to shift their attention toward the direction of another’s gaze. Similarly, Senju and Csibra 60 demonstrated that 6-month-old infants follow adult’s gaze when it is preceded by a period of eye contact, and that they stop following adult’s gaze when eye contact was removed from the stimuli. The results also support the prediction of Perrett and Emery 61 that the detection of eye contact is important for the gaze following. Thirdly, Senju et al. 62 examined whether 9-month-old infants can encode the relationship between gaze direction and the location of an object. In their study, infants observed a scene in which a face always looked toward a peripheral object, or always looked away from it. When the gaze shift was preceded by a period of eye contact, infants discriminated between the gaze shifts congruent to the location of the object and those incongruent to the location of the object, and consistently preferred to look longer to the former than to the latter. However, the removal of a period of eye contact preceding the gaze shift eliminated this preferential looking behaviour. These latter two studies clearly demonstrated that the presence or absence of eye contact modulates the processing of social stimuli that follows it. However, these early manifestations of the eye contact effect should not be taken to imply that it does not change over the course of development. For example, Smith et al. 63 reported that the magnitude of the eye contact effect on the memory for face identity increases over the period of development from 6 to 11 years.
Box 4: Atypical eye contact in individuals with ASD

The development of the eye contact effect may be disrupted in Autism Spectrum Disorders (ASD). ASD is characterized by difficulties in social interaction and communication, as well as narrowed interest. Clinical observations often report atypical patterns of mutual gaze behavior, which can be found early in ontogeny. The results of recent studies on eye contact processing in ASD are mixed. Some report that eye contact does not affect the speed or accuracy of the gaze direction detection or neurophysiological response to the face, but others report that individuals with ASD elicit stronger neurophysiological and physiological responses for direct than for averted gaze. Recent studies have demonstrated that individuals with ASD may respond to the psychophysical properties rather than the eye contact defined by the facial configuration (Figure I). In addition, recent neuroimaging studies with infants at high risk for developing ASD also demonstrated a relative lack of an increased neurophysiological response to eye contact. Thus, atypical response to eye contact effect may be present from very early in development.

The disrupted eye contact effect found in individuals with ASD may result from the structural impairment in sub-cortical structures that underlies the eye contact effect, such as the amygdala and/or the functional connectivity between the amygdala and other structures, or may be the outcome of insufficient opportunity to learn about eye contact, which originated from a lack of social orienting mechanisms or motivation.

Figure I. The eye contact stimuli used in our previous experiments. Children were asked to detect a face with particular gaze direction, which was presented among the distracter faces with different gaze directions. Typically developing children were better at detecting eye contact, and were facilitated when the faces were presented upright (A and B). However, this eye contact effect disappeared when the faces were inverted. The findings suggest that efficient eye contact detection in typically developing children depends on facial configuration or CONSPEC (Box 2). In contrast, children with ASD were better at detecting eye contact when the faces were in front view, regardless of facial orientation (A and C). However, the advantage for eye contact detection was absent when the laterally oriented faces were used as the stimuli (B and D). These studies suggest that efficient eye contact detection in children with ASD relies on psychophysical properties such as bilateral symmetry.
Box 5: Outstanding questions

- Why are some parts of the medial prefrontal cortex activated to direct gaze while other parts are more sensitive to averted gaze? Is there a functional specialization within the medial prefrontal cortex for different gaze directions?
- What is the precise nature of the modulatory mechanism in the eye contact effect? One mechanism could involve simple biasing (e.g. 74), which modulates the thresholds for activation of particular cortical and subcortical structures. Or the modulation may involve more complex top-down facilitation (e.g. 75), in which a rapidly processed partial image creates a coarse representation, which is then back-projected as a “first guess” to guide and modulate input driven processing.
- Does eye contact modulate non-social functions as well as social cognition?
- Is the eye contact effect specific to the visual input of eye contact, or are there other classes of stimuli that can elicit a similar response? For example, infant-directed speech or using someone’s name also indicates an intention to communicate may elicit similar effect as the eye contact effect in some conditions (e.g. 28, 60). It is also possible that other manual behaviours indicate communicative intention in some conditions (e.g. 76).
- Which of the models presented leads to a better understanding of typical development, as well as characterizing the developmental origin of atypical social interaction and communication in individuals with ASD?
- What is the function of the eye contact effect? Is it for reading others’ minds 28 or for communication 2? Although these two theories are not mutually exclusive, they make different predictions regarding which aspect of the event will become salient following a period of eye contact.
- Direct gaze can be a threatening signal under some contexts. Does the eye contact effect also function to detect potential threat?
- To what extent are cortical fast routes 75 involved in the eye contact effect?