Review

The eye contact effect: 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

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 towards, 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.

Introduction

Direct gaze signals that the gazer is looking at the perceiver. In many species, the perception of direct gaze elicits an aversive response [1], probably because it is a salient signal for potential threat. In humans, by contrast, eye contact provides a foundation of communication and social interaction [2,3]. 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 contact [4].

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 (FG), cingulate gyrus and amygdala [5], which are specialized to process social information such as the face [8], gaze [9], biological motion [9], human action [10], goal-directedness [10], theory of mind [11] and empathy [12]. Although 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. Here, 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 face [13,14] or eyes [15] 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 or 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]. Here, we 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'.

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 Ref. [20] 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): FG [21–23], anterior [21,24] and posterior [25–27] parts of superior temporal sulcus (STS), medial prefrontal [21,25,27,28] and orbitofrontal [24,25] cortex and amygdala [24,29,30]. Taken together, these studies reveal that perceived eye contact (i) enhances the activation of components of the social brain network [5,6], but (ii) this activation interacts with task demands, and 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. Here, we 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

Corresponding author: Senju, A. (a.senju@bbk.ac.uk).

1364-6613/$ – see front matter doi:10.1016/j.tics.2008.11.009 Available online 13 February 2009

Table 1. Neuroimaging studies compared direct versus averted gaze processing

Study	Method	Task	Orientation	Movement	Expression
Calder *et al.* [21]	PET	Eyebrows	Oriented	Static	Neutral
Conty *et al.* [25]	ERP	Gaze	Front view or Oriented	Dynamic	Neutral
Engell and Haxby [52]	fMRI	Identity	Front view	Static	Expressive
George *et al.* [22]	fMRI	Gender	Front view or Oriented	Static	Neutral
Hoffman and Haxby [81]	fMRI	Passive	Front view	Static	Neutral
Kampe *et al.* [28]	fMRI	Passive	Front view or Oriented	Static	Neutral
Kawashima *et al.* [29]	PET	Gaze	Oriented	Dynamic	Neutral
Pageler *et al.* [23]	fMRI	Gaze	Front view or Oriented	Static	Neutral
Pelphrey *et al.* [26]	fMRI	Gaze	Oriented	Dynamic	Neutral
Sato *et al.* [30]	fMRI	Gender	Oriented	Dynamic	Expressive or Neutral
Schilbach *et al.* [27]	fMRI	Gaze and Expression	Front view or Oriented	Dynamic	Expressive
Wicker *et al.* [51]	PET	Passive	Front view	Dynamic	Neutral
Wicker *et al.* [24]	PET	Expression or Gaze	Front view	Dynamic	Expressive or Neutral

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 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.

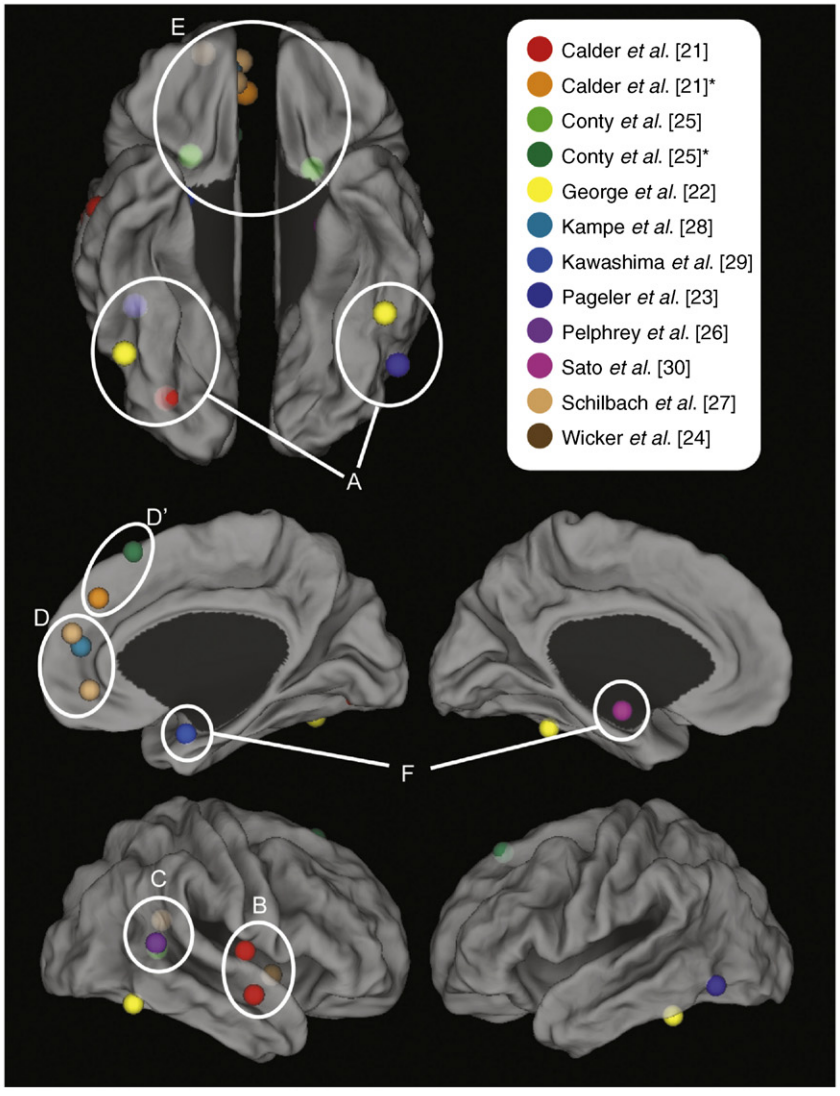

Figure 1. Cortical and subcortical regions that showed the eye contact effect in more than one study. **(A)** The FG, particularly in the right hemisphere shows larger activation for faces with direct gaze than those with averted gaze, when direct gaze accompanied the abrupt onset of static faces [21–23]. Two studies [21,24], which explicitly instructed participants to actually attend to the eye region, have reported increased activity in **(B)** the anterior part of the right STS regions in response to direct gaze. **(C)** The posterior part of right STS region is also sensitive to the presence of eye contact in two studies that used dynamic stimuli [25–27]. Several studies reported greater activation in response to direct gaze in the **(D)** right medial prefrontal cortex [27,28] and **(E)** orbitofrontal cortex [24,25]. In other studies [21,25], 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 **(F)** right [29] and left [30] amygdala in response to direct gaze, based on region-of-interest analyses. This figure is created with Caret software [77] (see: http://brainmap.wustl.edu/caret) and projected on Human PALS-B12 atlas [78]. *; regions showed larger activation for averted gaze.

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
When direct gaze accompanies the abrupt onset of static faces [21–23], 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 towards the participants [24,27,29,30,51] or when participants are required to attend to the identity of the stimulus faces [52]. These results indicate 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
Two studies [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 region (aSTS) in response to direct gaze. These findings indicate that instructed attention to the eyes, and the presence of eye contact, facilitate the encoding of gaze direction in this region [20,47]. Similar to FG [52], instructed attention to gaze direction might mask the eye contact effect in aSTS [22,23,25–27,29]. (See main text and Figure 2 for task-dependent effects.)

Posterior part of right STS region
This region is also sensitive to the presence of eye contact as demonstrated in three studies that used dynamic stimuli [25–27]. However, other studies [24,29,51] did not find the effect. These results indicate that perceived eye contact activates the posterior part of right STS region (pSTS), but only in the context that the perceiver recognizes it as being genuinely social and/or communicative (see also Ref. [20]).

Medial prefrontal cortex and orbitofrontal cortex
Several studies reported greater activation in response to direct gaze in the right medial prefrontal cortex (mPFC) [27,28] and orbitofrontal cortex (OFC) [24,25]. Note that three of these studies [24,25,27] presented dynamic facial expressions and two of them [24,27] 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 [21,25], 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 [24,29,30]. It is not clear why these three studies found amygdala responses whereas other studies did not find the effect [21,23,27,51,52]. One possibility is that because the amygdala is a small structure relative to the cortical areas discussed earlier, some current neuroimaging methods are not sensitive enough to detect these effects.

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 [17] and/or elicits a strong emotional response [29]. 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 activates widespread cortical structures [31,32]. This view accords with the introspective impression of 'being looked at', and is consistent with earlier findings that eye contact elevates physiological arousal [19]. In addition, the view is consistent with an integrative model including other aspects of face processing such as expression [33] and attractiveness [34], 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. First, 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. Second, results of recent psychophysiological studies are incongruent with the emotional arousal theory. For example, Kampe and colleagues [28] 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. Studies that are more recent 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 [35]. Thus, it seems 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 [24,25,27,28] or a pedagogy [2] brain system because it signals the intent to communicate with the perceiver.

Some proponents of the communicative intention detection model [11] have claimed that the computations underlying this function are subserved by specific cortical structures such as medial prefrontal cortex, temporal pole, STS and/or the temporal parietal junction. Recent studies also indicated that the FG can be a component of this theory-of-mind network [36]. 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

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

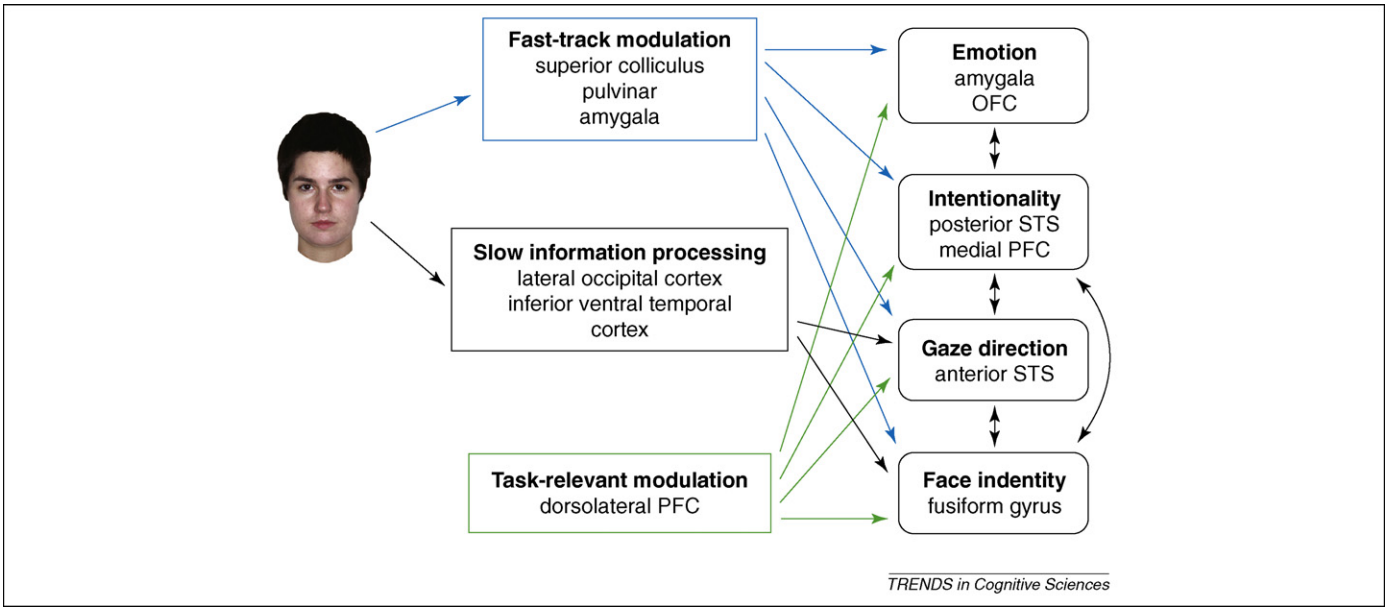

Figure 2. An illustration of the fast-track modulator model. Perceived eye contact (upper left) is initially detected by subcortical route, which projects to various regions of social brain network (blue lines). This signal from subcortical route then interacts with contextual modulation based on the task demands in addition to the social context (green lines) [46,79] 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 [8,80] and subcortical [37,38] face processing and on top-down voluntary attention [46]. Modified, with permission, from Ref. [38].

eye contact effect is mediated by the subcortical face detection pathway [37–41] 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 [38], which led LeDoux [40] to describe it as the 'quick and 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 usually associated with the 'structural encoding' stage of cortical face processing (such as the N170 and M170) [42]. Furthermore, the idea that sub-cortical responses to faces might precede those in the cortex is supported by intra-cranial ERPs recorded from epileptic patients with depth electrodes implanted into the amygdala [43].

Evidence that the route processes low spatial frequencies comes from functional magnetic resonance imaging (fMRI) studies in which the pulvinar, amygdala and superior colliculus respond to low spatial frequency (LSF) information about faces, and particularly fearful faces [44]. This sub-cortical route was insensitive to the high spatial frequency 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 [22,45].

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 [38]. 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).

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 [46], directly or indirectly modulates key structures involved in the cortical social brain network, such as the FG, STS, medial prefrontal and orbitofrontal cortex.

There are several lines of evidence that support this model. First, George *et al.* [22] reported that direct gaze increases the functional connectivity, or temporal correlation of regional activity, between the amygdala and the FG. This is consistent with the hypothesis that the amygdala specifically modulates the activation of the FG in response to perceived eye contact. Second, Conty *et al.* [25] found that the effect of presence or 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 [47] and human action [10] 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, 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).

Box 2. CONSPEC: a mechanism for eye contact detection?

Newborn infants preferentially orient to facial configurations [53,54], in addition to faces with direct gaze [48]. Farroni *et al.* [53] demonstrated in newborns that their preferences are consistent with the hypothesized CONSPEC, a visual mechanism that detects conspecifics [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 (Figure I). As Gliga and Csibra [55] (see also Ref. [38]) argued, CONSPEC is not only a mechanism for detecting facial configuration with the putative sub-cortical route but also at close distance might be the best representation to detect eye contact (Figure I). In addition, adult neuroimaging studies also indicated 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 FG 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 indicate that the amygdala is involved in the fixation to the eyes, in addition to 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.

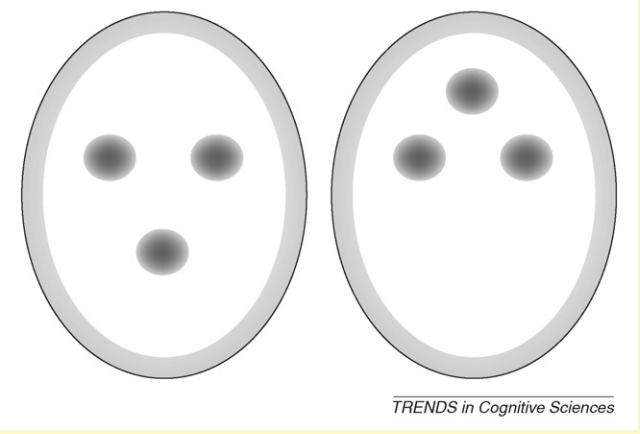

Figure I. Schematic illumination of the stimuli that might be optimal for activating the hypothesized subcortical route. Such a configuration is optimal for face-detection from a distance, in addition to eye contact detection in close-up. Reproduced, with permission, from Ref. [38].

One of the advantages of the fast-track modulator model is that it generates several specific predictions about the eye contact effect. First, 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. Second, the eye contact effect should be restricted to, or centred 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. Third, such fast-track modulation could compete with, and sometimes even be overcome by, other sources of modulation, such as top-down attention based on instruction and/or task demands [46]. That is, in adults, task demands can reduce or eliminate the eye contact effect under the influence of task-relevant attention.

The developmental basis of the eye contact effect

Several studies have revealed that sensitivity to eye contact is present even in newborns [48] (Box 2). Neuroimaging studies have also demonstrated that eye contact modulates cortical activation in infants as young as 4 months of age [48]. This suggests that human infants are equipped with a bias to detect and orient towards 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 (Box 3).

The question remains how the eye contact effect develops in human ontogeny. The three models presented earlier, indicate 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. By contrast, the communicative intention detector model often involves the innate capacity to detect and react to eye contact. For example, Baron-Cohen [49] proposed an innate module called the eye direction detector (EDD). The function of EDD is to

Box 3. The eye contact effect in young infants

Recent advances in infant studies indicate that perceived eye contact modulates the concurrent and/or immediately following cognitive processing or behavioural response of infants and adults. First, 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. Second, Farroni *et al.* [59] reported that a period of preceding eye contact is required for 4-month-old infants to shift their attention towards 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. Third, 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 towards 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 Autism Spectrum Disorder

The development of the eye contact effect might be disrupted in Autism Spectrum Disorder (ASD). ASD is characterized by difficulties in social interaction and communication and narrowed interest. Clinical observations often report atypical patterns of mutual gaze behaviour [64], which can be found early in ontogeny [65]. 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 [13] or neurophysiological response to the face [66], but others report that individuals with ASD elicit stronger neurophysiological [67,68] and physiological [69] responses for direct than for averted gaze. Recent studies [13,14] have demonstrated that individuals with ASD 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 [70]. Thus, atypical response to eye contact effect might be present from very early in development.

The disrupted eye contact effect found in individuals with ASD could result from the structural impairment in sub-cortical structures that underlies the eye contact effect, such as the amygdala [71] and/or the functional connectivity between the amygdala and other structures [45], or could be the outcome of insufficient opportunity to learn about eye contact, which originated from a lack of social orienting mechanisms [72] or motivation [73].

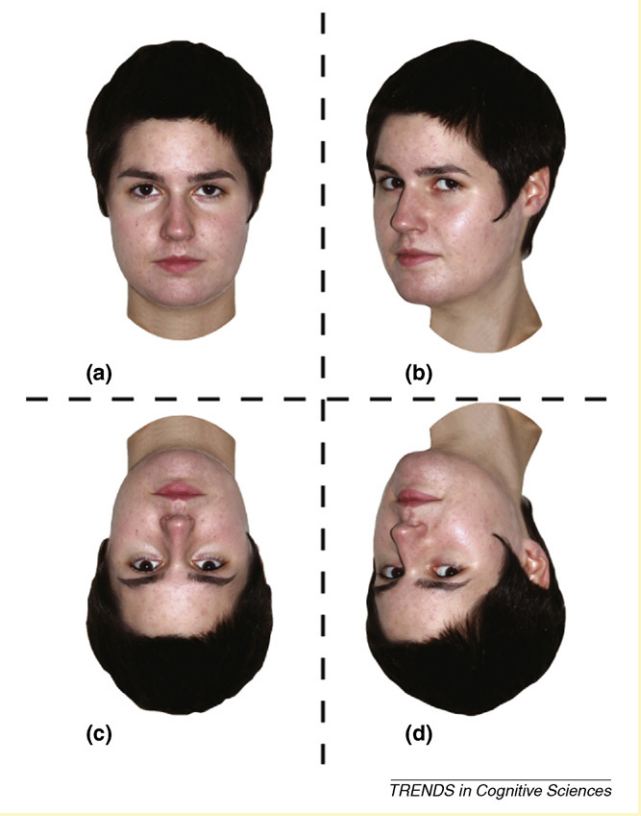

Figure I. The eye contact stimuli used in our previous experiments [13,14]. Children were asked to detect a face with particular gaze direction, which was presented among the distracter faces with different gaze directions. Typically developing children were better at detecting eye contact, and were facilitated when the faces were presented upright (a,b). However, this eye contact effect disappeared when the faces were inverted. The findings indicate that efficient eye contact detection in typically developing children depends on facial configuration or CONSPEC (Box 2). By contrast, children with ASD were better at detecting eye contact when the faces were in front view, regardless of facial orientation (a,c). However, the advantage for eye contact detection was absent when the laterally oriented faces were used as the stimuli (b,d). These studies indicate that efficient eye contact detection in children with ASD relies on psychophysical properties such as bilateral symmetry.

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 [2]. Such systems are usually claimed not to require postnatal experience because their function is to guide subsequent learning. By contrast, the fast-track modulator model assumes that infants are born with widespread connections between the subcortical route and cortical structures [38]. As a consequence, input from perceived eye contact initially activates widespread cortical structures, which combines with architectural biases [50] 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 underlying 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

Box 5. Outstanding questions

- Why are some parts of the medial prefrontal cortex activated to direct gaze, whereas 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. Ref. [74]), which modulates the thresholds for activation of particular cortical and subcortical structures. Alternatively, the modulation could involve more complex top-down facilitation (e.g. Ref. [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 in addition to 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 can elicit similar effect as the eye contact effect in some conditions (e.g. Refs [28,60]). It is also possible that other manual behaviours indicate communicative intention in some conditions (e.g. Ref. [76]).
- Which of the models presented leads to a better understanding of typical development, in addition to 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 after 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?

contact. Because of 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'. 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) and 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.

Acknowledgements

We thank Kathrin Cohen Kadosh, Laurence Conty, Gergely Csibra, Kevin Pelphrey and Bruno Wicker for the help and discussions on earlier version of draft. AS was supported by an ESRC Research Fellowship (RES -063–27–0207), and MHJ was supported by the UK Medical Research Council (G0701484).

References

1 Emery, N.J. (2000) The eyes have it: the neuroethology, function and evolution of social gaze. *Neurosci. Biobehav. Rev.* 24, 581–604
2 Csibra, G. and Gergely, G. (2006) Social learning and social cognition: the case for pedagogy. In *Processes of Change in Brain and Cognitive Development. Attention and Performance* (Vol. 21) Munakata, Y. and Johnson, M.H.,eds In pp. 249–274, Oxford University Press
3 Kleinke, C.L. (1986) Gaze and eye contact: a research review. *Psychol. Bull.* 100, 78–100
4 Kobayashi, H. and Kohshima, S. (1997) Unique morphology of the human eye. *Nature* 387, 767–768
5 Adolphs, R. (2009) The social brain: neural basis of social knowledge. *Annu. Rev. Psychol.* 60, 693–716
6 Brothers, L. (1990) The social brain: a project for integrating primate behavior and neuropsychology in a new domain. *Concepts Neurosci.* 1, 27–51
7 Johnson, M.H. *et al.* (2005) The emergence of the social brain network: evidence from typical and atypical development. *Dev. Psychopathol.* 17, 599–619
8 Haxby, J.V. *et al.* (2000) The distributed human neural system for face perception. *Trends Cogn. Sci.* 4, 223–233
9 Allison, T. *et al.* (2000) Social perception from visual cues: role of the STS region. *Trends Cogn. Sci.* 4, 267–278
10 Pelphrey, K.A. and Morris, J.P. (2006) Brain mechanisms for interpreting the actions of others from biological-motion cues. *Curr. Dir. Psychol. Sci.* 15, 136–140
11 Frith, C.D. and Frith, U. (2006) The neural basis of mentalizing. *Neuron* 50, 531–534
12 Singer, T. (2006) The neuronal basis and ontogeny of empathy and mind reading: review of literature and implications for future research. *Neurosci. Biobehav. Rev.* 30, 855–863
13 Senju, A. *et al.* (2005) Does perceived direct gaze boost detection in adults and children with and without autism? The stare-in-the-crowd effect revisited. *Vis. Cogn.* 12, 1474–1496
14 Senju, A. *et al.* (2008) Is anyone looking at me? Direct gaze detection in children with and without autism. *Brain Cogn.* 67, 127–139
15 Conty, L. *et al.* (2006) Searching for asymmetries in the detection of gaze contact versus averted gaze under different head views: a behavioural study. *Spat. Vis.* 19, 529–545
16 Macrae, C.N. *et al.* (2002) Are you looking at me? Eye gaze and person perception. *Psychol. Sci.* 13, 460–464
17 Hood, B.M. *et al.* (2003) Eye remember you: the effects of gaze direction on face recognition in children and adults. *Dev. Sci.* 6, 67–71
18 Senju, A. and Hasegawa, T. (2005) Direct gaze captures visuospatial attention. *Vis. Cogn.* 12, 127–144
19 Nichols, K.A. and Champness, B.G. (1971) Eye gaze and GSR. *J. Exp. Soc. Psychol.* 7, 623–626
20 Nummenmaa, L. and Calder, A.J. (2009) Neural mechanisms of social attention. *Trends Cogn. Sci.* 13, 135–143
21 Calder, A.J. *et al.* (2002) Reading the mind from eye gaze. *Neuropsychologia* 40, 1129–1138
22 George, N. *et al.* (2001) Seen gaze-direction modulates fusiform activity and its coupling with other brain areas during face processing. *Neuroimage* 13, 1102–1112
23 Pageler, N.M. *et al.* (2003) Effect of head orientation on gaze processing in fusiform gyrus and superior temporal sulcus. *Neuroimage* 20, 318–329
24 Wicker, B. *et al.* (2003) Being the target of another's emotion: a PET study. *Neuropsychologia* 41, 139–146
25 Conty, L. *et al.* (2007) When eye creates the contact! ERP evidence for early dissociation between direct and averted gaze motion processing. *Neuropsychologia* 45, 3024–3037
26 Pelphrey, K.A. *et al.* (2004) When strangers pass: processing of mutual and averted social gaze in the superior temporal sulcus. *Psychol. Sci.* 15, 598–603
27 Schilbach, L. *et al.* (2006) Being with virtual others: neural correlates of social interaction. *Neuropsychologia* 44, 718–730
28 Kampe, K.K. *et al.* (2003) Hey John": signals conveying communicative intention toward the self activate brain regions associated with "mentalizing" regardless of modality. *J. Neurosci.* 23, 5258–5263
29 Kawashima, R. *et al.* (1999) The human amygdala plays an important role in gaze monitoring. A PET study. *Brain* 122, 779–783
30 Sato, W. *et al.* (2004) The amygdala processes the emotional significance of facial expressions: an fMRI investigation using the interaction between expression and face direction. *Neuroimage* 22, 1006–1013
31 Adolphs, R. (2003) Cognitive neuroscience of human social behaviour. *Nat. Rev. Neurosci.* 4, 165–178
32 Pfaff, D. *et al.* (2008) Concepts and mechanisms of generalized central nervous system arousal. *Ann. N. Y. Acad. Sci.* 1129, 11–25
33 Vuilleumier, P. and Pourtois, G. (2007) Distributed and interactive brain mechanisms during emotion face perception: evidence from functional neuroimaging. *Neuropsychologia* 45, 174–194
34 Winston, J.S. *et al.* (2007) Brain systems for assessing facial attractiveness. *Neuropsychologia* 45, 195–206
35 Hietanen, J.K. *et al.* (2008) Seeing direct and averted gaze activates the approach-avoidance motivational brain systems. *Neuropsychologia* 46, 2423–2430
36 Schultz, R.T. *et al.* (2003) The role of the fusiform face area in social cognition: implications for the pathobiology of autism. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* 358, 415–427
37 de Gelder, B. *et al.* (2003) A modulatory role for facial expressions in prosopagnosia. *Proc. Natl. Acad. Sci. USA.* 100, 13105–13110
38 Johnson, M.H. (2005) Subcortical face processing. *Nat. Rev. Neurosci.* 6, 766–774
39 Johnson, M.H. and Morton, J. (1991) *Biology and Cognitive Development.* Blackwell
40 LeDoux, J.E. (1996) *The Emotional Brain.* Simon & Schuster
41 Morris, J.S. *et al.* (1999) A subcortical pathway to the right amygdala mediating "unseen" fear. *Proc. Natl. Acad. Sci. USA.* 96, 1680–1685
42 Bailey, A.J. *et al.* (2005) Abnormal activation of face processing systems at early and intermediate latency in individuals with autism spectrum disorder: a magnetoencephalographic study. *Eur. J. Neurosci.* 21, 2575–2585
43 Krolak-Salmon, P. *et al.* (2004) Early amygdala reaction to fear spreading in occipital, temporal, and frontal cortex: a depth electrode ERP study in human. *Neuron* 42, 665–676
44 Winston, J.S. *et al.* (2003) Effects of low-spatial frequency components of fearful faces on fusiform cortex activity. *Curr. Biol.* 13, 1824–1829

45 Kleinhans, N.M. *et al.* (2008) Abnormal functional connectivity in autism spectrum disorders during face processing. *Brain* 131, 1000–1012
46 Curtis, C.E. and D'Esposito, M. (2003) Persistent activity in the prefrontal cortex during working memory. *Trends Cogn. Sci.* 7, 415–423
47 Calder, A.J. *et al.* (2007) Separate coding of different gaze directions in the superior temporal sulcus and inferior parietal lobule. *Curr. Biol.* 17, 20–25
48 Farroni, T. *et al.* (2002) Eye contact detection in humans from birth. *Proc. Natl. Acad. Sci. USA.* 99, 9602–9605
49 Baron-Cohen, S. (1995) *Mindblindness: An Essay on Autism and Theory of Mind.* MIT Press
50 Johnson, M.H. (2005) *Developmental Cognitive Neuroscience: An Introduction.* (2nd Edn), Blackwell
51 Wicker, B. *et al.* (1998) Brain regions involved in the perception of gaze: a PET study. *Neuroimage* 8, 221–227
52 Engell, A.D. and Haxby, J.V. (2007) Facial expression and gaze-direction in human superior temporal sulcus. *Neuropsychologia* 45, 3234–3241
53 Farroni, T. *et al.* (2005) Newborns' preference for face-relevant stimuli: effects of contrast polarity. *Proc. Natl. Acad. Sci. USA.* 102, 17245–17250
54 Johnson, M.H. *et al.* (1991) Newborns' preferential tracking of face-like stimuli and its subsequent decline. *Cognition* 40, 1–19
55 Gliga, T. and Csibra, G. (2007) Seeing the face through the eyes: a developmental perspective on face expertise. *Prog Brain Res.* 164, 323–339
56 Whalen, P.J. *et al.* (2004) Human amygdala responsivity to masked fearful eye whites. *Science* 306, 2061
57 Adolphs, R. *et al.* (2005) A mechanism for impaired fear recognition after amygdala damage. *Nature* 433, 68–72
58 Farroni, T. *et al.* (2007) Direct gaze modulates face recognition in young infants. *Cognition* 102, 396–404
59 Farroni, T. *et al.* (2003) Infants perceiving and acting on the eyes: tests of an evolutionary hypothesis. *J. Exp. Child Psychol.* 85, 199–212
60 Senju, A. and Csibra, G. (2008) Gaze following in human infants depends on communicative signals. *Curr. Biol.* 18, 668–671
61 Perrett, D.I. and Emery, N.J. (1994) Understanding the intention of others from visual signals – neurophysiological evidence. *Curr. Psychol. Cogn.* 13, 683–694
62 Senju, A. *et al.* (2008) Understanding the referential nature of looking: infants' preference for object-directed gaze. *Cognition* 108, 303–319
63 Smith, A.D. *et al.* (2006) Eye remember you two: gaze direction modulates face recognition in a developmental study. *Dev. Sci.* 9, 465–472
64 American Psychiatric Association (2000) *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)* (4th edn), American Psychiatric Publishing, Inc
65 Maestro, S. *et al.* (2005) How young children treat objects and people: an empirical study of the first year of life in autism. *Child Psychiatry Hum. Dev.* 35, 383–396
66 Senju, A. *et al.* (2005) Deviant gaze processing in children with autism: an ERP study. *Neuropsychologia* 43, 1297–1306
67 Grice, S.J. *et al.* (2005) Neural correlates of eye-gaze detection in young children with autism. *Cortex* 41, 342–353
68 Kylliäinen, A. *et al.* (2006) Face and gaze processing in normally developing children: a magnetoencephalographic study. *Eur. J. Neurosci.* 23, 801–810
69 Kylliäinen, A. and Hietanen, J.K. (2006) Skin conductance responses to another person's gaze in children with autism. *J. Autism Dev. Disord.* 36, 517–525
70 Elsabbagh, M. *et al.* (2009) Neural correlates of eye gaze processing in the infant broader autism phenotype. *Biol. Psychiatry* 65, 31–38
71 Schumann, C.M. *et al.* (2004) The amygdala is enlarged in children but not adolescents with autism; the hippocampus is enlarged at all ages. *J. Neurosci* 24, 6392–6401
72 Schultz, R.T. (2005) Developmental deficits in social perception in autism: the role of the amygdala and fusiform face area. *Int. J. Dev. Neurosci.* 23, 125–141
73 Dawson, G. *et al.* (2005) Understanding the nature of face processing impairment in autism: insights from behavioral and electrophysiological studies. *Dev. Neuropsychol.* 27, 403–424
74 Spratling, M.W. and Johnson, M.H. (2004) A feedback model of visual attention. *J. Cogn. Neurosci.* 16, 219–237
75 Bar, M. and Aminoff, E. (2003) Cortical analysis of visual context. *Neuron* 38, 347–358
76 Cartmill, E.A. and Byrne, R.W. (2007) Orangutans modify their gestural signaling according to their audience's comprehension. *Curr. Biol.* 17, 1345–1348
77 Van Essen, D.C. *et al.* (2001) An integrated software suite for surface-based analyses of cerebral cortex. *J. Am. Med. Inform. Assoc.* 8, 443–459
78 Van Essen, D.C. (2005) A Population-Average, Landmark- and Surface-based (PALS) atlas of human cerebral cortex. *Neuroimage* 28, 635–662
79 Banich, M.T. *et al.* (2000) Prefrontal regions play a predominant role in imposing an attentional 'set': evidence from fMRI. *Brain Res. Cogn* 10, 1–9
80 Palermo, R. and Rhodes, G. (2007) Are you always on my mind? A review of how face perception and attention interact. *Neuropsychologia* 45, 75–92
81 Hoffman, E.A. and Haxby, J.V. (2000) Distinct representations of eye gaze and identity in the distributed human neural system for face perception. *Nat. Neurosci.* 3, 80–84