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Is eye contact the key to the social brain?

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Abstract: Eye contact plays a critical role in many aspects of face processing, including the processing of smiles. We propose that this is achieved by a subcortical route, which is activated by eye contact and modulates the cortical areas involved in social cognition, including the processing of facial expression. This mechanism could be impaired in individuals with autism spectrum disorders.

The Simulation of Smiles (SIMS) model proposed by Niedenthal et al. emphasizes the core role of eye contact, which is hypothesized to trigger embodied simulation of the perceived smile. The authors also speculated that the same mechanism may also mediate the processing of other facial expressions. However, eye contact is known to modulate a far wider range of cognitive processes, such as the encoding of gender, identity, and gaze (Senju & Johnson 2009b). We recently reviewed this phenomenon, which we have termed the “eye contact effect,” and proposed the fast-track modulator (FTM) model to explain its neural and developmental basis (Senju & Johnson 2009b). In this commentary, we present a brief overview of the FTM model and discuss several areas in which the FTM model complements the SIMS model, and thus would facilitate further exploration of the neural, cognitive, and developmental mechanism underlying the effect of eye contact on face processing.

The FTM model proposes that the eye contact effect is mediated by a 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 (Figure 1). Evidence that the route is fast comes from event-related potential and magnetoencephalographic studies showing that components associated with this pathway can occur at shorter latencies than those usually associated with the cortical processing of faces (Bailey et al. 2005). In addition, evidence that the subcortical route modulates cortical processing comes from several functional imaging studies indicating that the degree of activation of structures in the subcortical route (amygdala, superior colliculus, and pulvinar) predicts or correlates with the activation of cortical face processing areas (George et al. 2001, Kleinhans et al. 2008). It has also been proposed that the subcortical route is also responsible for face preference in newborn infants (Johnson 2005) and even in adults (Tomalski et al. 2009). We hypothesized that the combination of this subcortical pathway and contextual modulation given by the task demands and social context directly or indirectly modulates key structures involved in the cortical social brain network.

The FTM model shares several key features with the SIMS model. However, there are several differences between these two models, by which the FTM model expands and broadens the SIMS model. First, the FTM model proposes the neural mechanism linking eye contact and facilitation of cortical face processing, including the embodied simulation. The FTM model proposes that perceived eye contact directly activates a subcortical route, which then modulates the cortical areas involved in different aspects of social cognitive processing. Thus, it is possible to incorporate the SIMS model by arguing that the subcortical route also modulates the motor cortex, which controls mimicry. The FTM model also provides new predictions about the effect of eye contact on the processing of smiles: It should be fast and operate on low spatial frequency visual information.

Second, the FTM model can also provide alternative hypotheses about the mechanism by which eye contact facilitates the processing of smiles. The FTM model hypothesizes that the subcortical route receiving input from perceived eye contact directly modulates the cortical face processing areas. This contrasts with the SIMS model that assumes that eye contact must elicit the embodied simulation first in order to facilitate the processing of smiles. As we discussed earlier, the FTM does not rule out the possibility that the subcortical route mediates the embodied simulation in response to eye contact. However, the FTM also leads us to propose a more parsimonious hypothesis: The subcortical route directly

modulates visual cortical areas, which then facilitates the processing of facial expression, including smiles. For example, the FTM model predicts that eye contact modulates the processing of smiles even when the activation of the motor cortex is experimentally suppressed. By contrast, the SIMS model would not predict that eye contact facilitates the processing of smiles under this condition, because embodied simulation is suppressed.

Third, the FTM model presents a unique perspective on the development of the eye contact effect. In the target article, Niedenthal et al. suggested an interesting hypothesis that the preference for eye contact in infants reflects an evolutionary-based mechanism for triggering embodied simulation, even though they did not discuss how such a mechanism develops. By contrast, the FTM 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 bias to form specific connections between the subcortical “eye contact detector” and relevant cortical and subcortical structures during the course of development. Interestingly, recent studies on the early development of autism spectrum disorder (ASD), showing manifest atypical patterns of eye contact behavior, are consistent with the predictions based on the FTM model. Even though infants and young children with autism show apparently typical eye contact behavior (Chawarska & Shic 2009), neuroimaging studies demonstrate more widespread and nonspecific cortical activation in response to eye contact (Elsabbagh et al. 2009), and behavioral studies demonstrated that eye contact does not facilitate cognitive processing in children with ASD (Senju et al. 2003). These studies suggests that infants and young children with ASD are sensitive to eye contact, but that it fails to modulate cortical face processing in the same specialized way as typically developing children (Senju & Johnson 2009a). Future studies will need to test whether eye contact elicits facial mimicry and affects the processing of smiles in individuals with ASD, especially because current evidence is inconsistent as to whether individuals with ASD show spontaneous facial mimicry (Magnée et al. 2007) or not (McIntosh et al. 2006, Oberman et al. 2009).

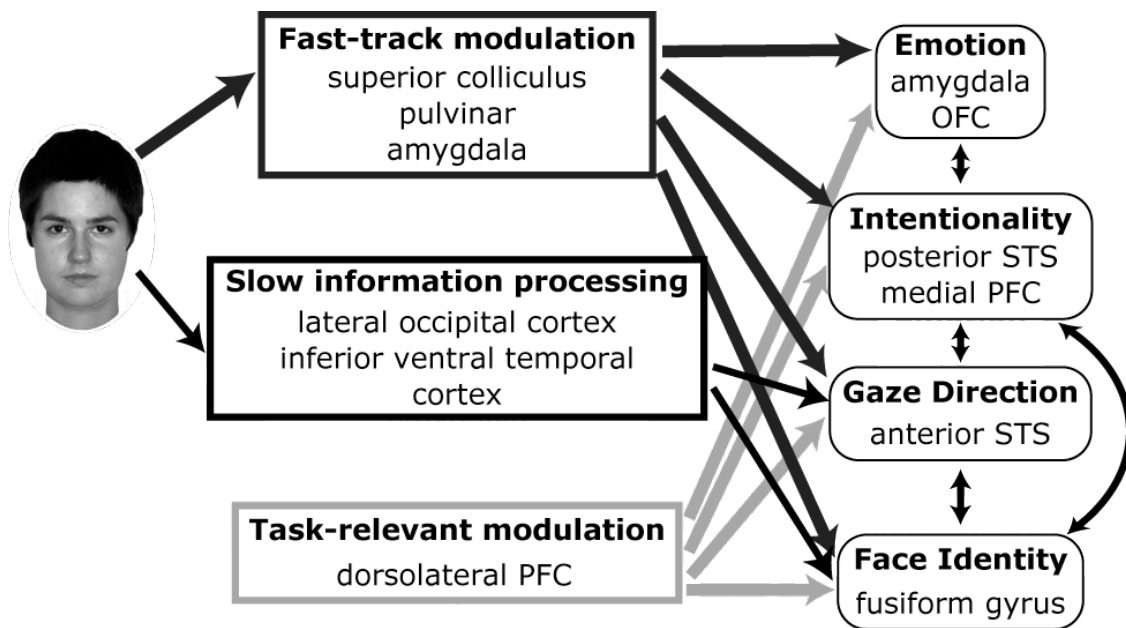
Eye contact plays a critical role in face-to-face communication, and we propose it is the key to adaptively modulate many aspects of social cognition, including the processing of smiles. We hope the areas of overlap and contrast between the SIMS and the FTM models will generate empirical studies, and help further understand the neural, cognitive, and developmental mechanisms underlying human social behavior.

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<CAPT>Figure 1. (Senju et al.) An illustration of the fast-track modulator model (FTM). Perceived eye contact (upper left) is initially detected by a subcortical route that projects to various regions of the social brain network (thick black lines). This signal from the subcortical route then interacts with contextual modulation based on the task demands, as well as the social context (thick gray 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 of cortical and subcortical face processing, as well as on top-down voluntary attention. OFC = orbitofrontal cortex, PFC = prefrontal cortex, STS = superior temporal sulcus. Reproduced with permission from A. Senju & M. H. Johnson (2009b) The eye contact effect: Mechanisms and development. *Trends in Cognitive Sciences* 13:127–34.