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The right place at the right time: Priming facial expressions with emotional face components in developmental visual agnosia

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ABSTRACT

The current study examined the nature of deficits in emotion recognition from facial expressions in case LG, an individual with a rare form of developmental visual agnosia (DVA). LG presents with profoundly impaired recognition of facial expressions, yet the underlying nature of his deficit remains unknown. During typical face processing, normal sighted individuals extract information about expressed emotions from face regions with activity diagnostic for specific emotion categories. Given LG's impairment, we sought to shed light on his emotion perception by examining if priming facial expressions with diagnostic emotional face components would facilitate his recognition of the emotion expressed by the face. LG and control participants matched isolated face components with components appearing in a subsequently presented full-face and then categorized the face's emotion. Critically, the matched components were from regions which were diagnostic or non-diagnostic of the emotion portrayed by the full face. In experiment 1, when the full faces were briefly presented (150 ms), LG's performance was strongly influenced by the diagnosticity of the components: his emotion recognition was boosted within normal limits when diagnostic components were used and was obliterated when non-diagnostic components were used. By contrast, in experiment 2, when the face-exposure duration was extended (2000 ms), the beneficial effect of the diagnostic matching was diminished as was the detrimental effect of the non-diagnostic matching. These data highlight the impact of diagnostic facial features in normal expression recognition and suggest that impaired emotion recognition in DVA results from deficient visual integration across diagnostic face components.

1. Introduction

Developmental visual agnosia (DVA) is characterized by lifelong difficulties with visual recognition in the absence of evident brain lesions, cognitive impairments or low level impaired eyesight (Farah, 1990; Gilaie-Dotan, Perry, Bonneh, Malach, & Bentin, 2009). Individuals with DVA may present with profound deficits in object recognition, impaired visual integration and deficits in processing face identity and face expression (Ariel & Sadeh, 1996; Aviezer et al., 2009). While previous work has demonstrated profound deficits in facial expression recognition in DVA the underlying cause remains unknown. The current study examined the impaired recognition of face-expressed emotions by testing how diagnostic face parts are perceived and integrated in case LG, an individual with a rare form of DVA with a profound visual integration deficiency (Gilaie-Dotan et al., 2009).

Although severely impaired face identification is characteristic to DVA, this rare syndrome differs from classic developmental prosopagnosia (DP) in three important ways. First, from a clinical perspective, individuals with DVA typically present with profound and pervasive visual deficits, which typically include form agnosia, deficient perceptual integration of parts to a meaningful whole, and impaired generalized processing of faces, including gender, emotion, and identity information (Ariel & Sadeh, 1996; Duchaine, Nieminen-von Wendt, New, & Kulomaki, 2003). By contrast accumulating evidence suggests that most individuals with DP do not typically suffer from such pervasive visual deficits. Rather, they often have fairly intact recognition of social and emotional information from faces alongside specific deficits in identity processing (e.g., Dobel, Bolte, Aicher, & Schweinberger, 2007; Duchaine, Jenkins, Germine, & Calder, 2009; Duchaine, Murray, Turner, White, & Garrido, 2009; Duchaine & Nakayama, 2006; Duchaine, Parker, & Nakayama, 2003; Garrido et al., 2009; Humphreys, Avidan, & Behrmann, 2007; Palermo, Willis, Rivolta, Wilson, & Calder, 2010;

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Palermo et al., 2011; Todorov & Duchaine, 2008). While some DPs may have deficits with both face and object processing (Behrmann & Avidan, 2005) most exhibit face-specific deficits with no evidence of impairment in matched tests of object recognition or other types of visual recognition (Bentin, DeGutis, D'Esposito, & Robertson, 2007; Bentin, Deouell, & Soroker, 1999; Duchaine & Nakayama, 2005; Duchaine, Yovel, & Nakayama, 2007). Furthermore, LG, the individual with DVA tested here, displayed profound integration deficits which are uncharacteristic of the typical DP case. We further elaborate on the unique aspects of DVA as apposed to DP when we describe LG's case history.

The second important difference between DVA and DP stems from functional neuroanatomy. A previous study of LG revealed a highly atypical pattern of brain activity to visual stimuli which has not been described in DP. Specifically, Gilaie-Dotan et al. (2009) have shown that in LG, V1 was robustly activated by visual stimuli, and activity in down stream visual areas showed selectivity for houses and places (but not for faces and objects). Yet intriguingly, intermediate visual areas (V2–V4) showed strong deactivation in response to any visual stimulation. Studies in developmental prosopagnosia have yielded inconsistent results with regard to activation in the fusiform gyrus, with some studies showing normal activity (Avidan, Hasson, Malach, & Behrmann, 2005; Hasson, Avidan, Deouell, Bentin, & Malach, 2003) while others did not (Bentin et al., 2007; Van den Stock, van de Riet, Righart, & de Gelder, 2008). Yet, studies with DP have not showed such atypical deactivation of intermediate visual areas as in case LG, further establishing the difference between DP and, at least, the current case of DVA.[1]

Finally, from an epidemiological perspective there is an additional distinction between DP and DVA: with an estimated prevalence of 2% in the general population (Kennerknecht et al., 2006), DP is surprisingly common, while DVA appears to be far rarer and is seldom described in the literature. Consequently, because of the scarcity of DVA cases, little is known about the perceptual mechanisms underlying their deficient face processing in general and their recognition of emotional expressions in particular. To this end, the present study makes an important contribution to a broader understanding of face processing impairments in DVA.

In their original report, Ariel and Sadeh (1996) described LG as poor at recognizing facial expressions; however the procedure and testing stimuli they used were informal. Recent follow up testing with prototypical and standardized facial expressions indicated that LG is still densely impaired at the recognition of facial expressions, e.g., his recognition of anger and disgust hovered around chance level (Aviezer, Hassin, & Bentin, 2011). Yet, while the existence of LG's current impairment is well established, its underlying cause remains unclear.

One possible explanation for LG's deficit may be that he fails to correctly extract and process information from the emotional face components. Individuals with normal vision recognize basic facial expressions by extracting information from specific signals (e.g., nose wrinkling, eye widening, etc.) which are diagnostic of particular emotions (Brosch, Pourtois, & Sander, 2010; Buck, 1994; Ekman, 1993; Schyns, Petro, & Smith, 2007; Smith & Scott, 1997; Smith, Cottrell, Gosselin, & Schyns, 2005; Whalen et al., 2004). Although research has demonstrated holistic characteristics in facial expression perception (Calder, Young, Keane, & Dean, 2000) the components themselves are often sufficient to drive full emotional recognition (Ellison & Massaro, 1997). In this line, Smith et al. (2005) revealed distinct diagnostic fingerprint-regions for each expression category: while the diagnostic region for anger faces entailed the detection of activity in the eyes, the diagnostic region for disgust expressions entailed the detection of activity in the lower, oral-nasal regions. LG, however, has lifelong visual agnosia, and he may have never learned how to successfully process emotional faces. Specifically, he may be impaired at focusing his visual processing on the face regions which entail diagnostic face components and extracting from them the affective information necessary for categorization.

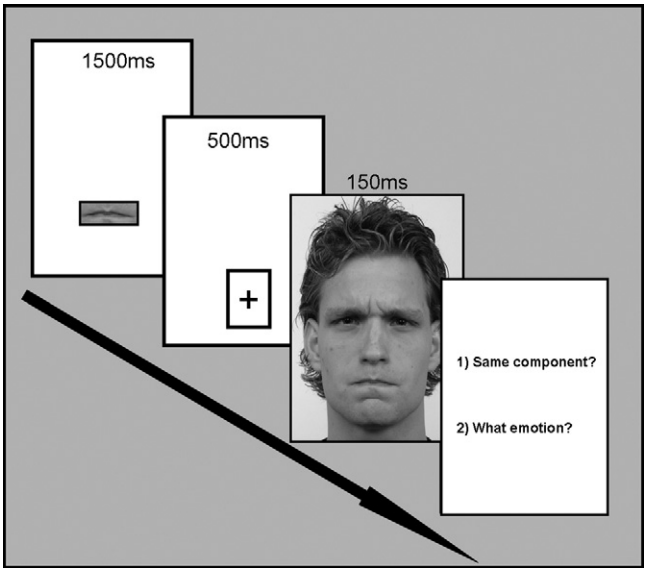

Fig. 1. Outline of an experimental trial. At the first stage a component (mouth or eyes) was shown and was later matched (as same/different) to the corresponding component in the full face. Subsequently, the emotion of the full face was determined. The figure portrays a "same" trial because the isolated mouth is identical to the full-face mouth. The trial is also "non-diagnostic" because the mouth is not diagnostic of facial anger.

Facial expression used with permission from the Radboud Faces Database (Langner et al., 2010).

In order to examine the role of diagnostic-component processing in LG's deficit we devised a *component-matching task* which directs participants to process specific facial components which may facilitate or interfere with the subsequent categorization of a facial expression. As seen in Fig. 1, the task starts with a *matching procedure*: the participant is exposed to an isolated facial component (e.g., a mouth) followed by a briefly presented full face and is asked whether the respective components (i.e., the two mouths) are identical or not. This is followed by a facial-expression judgment procedure in which the participant is asked to report the emotion of the full face.

Critically, the matched component may or may not be diagnostic of the actual emotion expressed by the full face. For example, consider a trial in which an image of a mouth (obtained from an angry face) precedes an image of a full angry face (Fig. 1). The matched feature in this case is non-diagnostic because, as previously described, anger is not recognized from the information in the mouth but rather from information in the eyes. While the mouth region of angry faces may be uninformative for the recognition of anger, it may actually hold information erroneously suggesting other emotions. Hence, forcing participants to match a non-diagnostic component (i.e., a facial component that provides no reliable information about the intentionally posed facial expression) may reduce the subsequent recognition of the facial expression.

Note that the component matching procedure could influence the facially-expressed emotion recognition in two, non-mutually exclusive ways, which were effectively intertwined in our design. First, by enhancing the recognition of emotional information

[1] Since DVA is an extremely rare syndrome and there is no imaging data from other cases, we do not know whether this strange pattern of activations in the visual system is typical to DVA or peculiar to LG.