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Startle modulation in autism: Positive affective stimuli enhance startle response

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ABSTRACT

Behavioral evidence suggests that emotion processing deficits in individuals with autism spectrum disorders (ASD) may occur at the level of basic (early, rapid, automatic) affective processes. Consistently, neurological evidence indicates that key brain areas associated with basic affective processing are atypical in ASD. The current study sought to better specify these deficits by comparing different components of basic affective processing in 14 adolescents and adults with ASD and 14 typical controls matched for age and verbal ability. Participants viewed affective pictures, and their responses were assessed with (i) affective eyeblink startle modulation, an indicator of the brain's aversive motivational system; (ii) facial electromyography, an online indicator of implicit valence appraisal; and (iii) self-report, an indicator of overt valence appraisal. The results show that in contrast to the typical pattern, in which exposure to negative stimuli increases startle whereas exposure to positive stimuli decreases startle, individuals with ASD showed startle potentiation to both positive and negative stimuli. Atypical potentiation during positive stimuli occurred despite individuals with ASD demonstrating appropriate implicit valence appraisals, reflected in their facial EMG responses, and appropriate overt appraisals, reflected in their self-reported ratings of the stimuli. Potentiation of startle to both positive and negative stimuli suggests a disruption in basic affective processes in ASD at the level of the early motivational response. This atypical pattern of responses has implications for understanding social and emotion deficits in ASD and calls for further investigation of basic affective processes.

Clinical experience and research reveal that individuals with autism spectrum disorders (ASD) have a mosaic of social and emotional skills. People with ASD show atypicalities in several areas of social and emotional functioning (Fein, Pennington, Markowitz, Braverman, & Waterhouse, 1986; Hobson, 2005; Kanner, 1943; Rogers & Pennington, 1991; Sigman, Kasari, Kwon, & Yirmiya, 1992; Sigman & Ruskin, 1999). These disruptions include altered responsiveness to social and affective signals, atypical processing of faces, and greater generation of negative emotional expressions (Dawson et al., 2002; Dissanayake, Sigman, & Kasari, 1996; Hobson, 1995, 1999; Kasari & Sigman, 1996; Rieffe, Terwogt, & Stockmann, 2000; Rogers & Pennington, 1991). However, not all areas of social and emotional functioning are disrupted. Individuals with ASD express a full range of emotion, show attachment behaviors, and comprehend a variety of emotional situations (Braverman, Fein, Lucci, & Waterhouse, 1989; Rogers & Pennington, 1991; Sigman et al., 1992; Sigman & Ruskin, 1999). Additionally, some ASD deficits in affective processing may be better attributed to cognitive or language factors (Dawson, Soulieres, Gernsbacher, & Mottron, 2007; Ozonoff, Pennington, & Rogers, 1990).

The above pattern of findings suggests that social and emotional processing in ASD is selectively disrupted, rather than uniformly impaired. This calls for a careful examination of different aspects of affective processing to specify which functions are atypical. The present study contributes to this goal by assessing several components of basic affective processing using physiological and behavioral measures.

Successful affective processing involves several basic components, including assignment of salience to stimuli, determination of positive or negative valence, activation of aversive and appetitive systems which prime approach and avoidance behaviors, and generation of affective experience. These early, rapid, spontaneous, and largely automatic processes help guide behavior, especially in fast, dynamic, online interactions with the environment, such as reciprocal social exchanges (Bradley & Lang, 2007; Cacioppo, Tassinary, & Berntson, 2000; Dawson et al., 2002; Lang, 1995). Some of these processes are so rudimentary that several writers proposed that human infants are biologically prepared to perceive and recipro-

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cally respond to social and emotional information (Balaban, 1995; Meltzoff & Moore, 1995; Stern, 1995; Valenza, Simion, & Cassia, 1996). Consequently, disruptions in these basic skills may contribute to atypical social and emotional development.

Behavioral and neuroscience research suggests that basic affective processes, such as those involved in creating emotional reciprocity, are impaired in autism (Dawson, Webb, & Wijsman, 2005; Dissanayake et al., 1996; Pennington, 2002; Rogers & Pennington, 1991). For example, individuals with ASD are impaired on rapid and spontaneous facial mimicry (McIntosh, Reichmann-Decker, Winkielman, & Wilbarger, 2006; Oberman, Winkielman, & Ramachandran, 2009). They also show undifferentiated and inconsistent rapid facial responses to emotional expressions (Beall, Moody, McIntosh, Hepburn, & Reed, 2008) and demonstrate increased latencies in early event related potential (ERP) brain responses to faces (Webb, Dawson, Bernier, & Panagiotides, 2006; McPartland, Dawson, Webb, Panagiotides, & Carver, 2004; Dawson et al., 2005).

Behavioral evidence of atypicalities in basic affective processes is consistent with the neuropsychological evidence of ASD deficits in basic affective and social brain networks, especially the temporal lobe, the orbital frontal cortex, and the prefrontal cortex (Amaral, Bauman, & Schamann, 2003; Bachevalier, 2000; Baron-Cohen, Ring, Bullmore, Wheelwright, & Williams, 2000; Brothers, 1997; Dalton et al., 2005). In particular, work has focused on the amygdala, a temporal lobe structure involved in processing faces, assignment of affective significance to stimuli, and organization of defensive responses (Aggleton, 2000). Compared to typical peers, in ASD the amygdala shows functional and structural differences (Aylward et al., 1999; Bauman & Kemper, 1985; Bauman & Kemper, 2005) and atypical activation to affective and social stimuli (Baron-Cohen et al., 2000; Critchley et al., 2000; Dalton et al., 2005; Hazenedar et al., 2000; Pierce, Muller, Ambrose, & Courchesne, 2001). In non-human primates, removal of or damage to areas of the temporal lobe, including the amygdala, result in deficits in social and affective functioning that parallel some deficits in ASD (Amaral et al., 2003; Bachevalier & Loveland, 2006).

Although behavioral and neuroscience evidence suggests ASD deficits in basic affective processes, it is not clear which specific components are atypical. To examine this, the current study used three different methods that tap into motivational, valence, and explicit appraisal components: (i) affective startle modulation; (ii) facial electromyography; and (iii) self-reports of stimulus valence. As described in the next section, affective startle modulation measures the influence of the valenced stimuli on the rapid and reflexive responses of the aversive motivational system. Typically, negative stimuli activate the aversive response system (enhancing startle) whereas positive stimuli dampen its activation (reducing startle). In contrast, facial EMG provides an online measure of implicit valence appraisals. Typically, positive stimuli activate facial muscles associated with smiling whereas negative stimuli activate facial muscles associated with frowning. Finally, self-report ratings of stimulus valence tap the overt appraisal of the stimulus as well as the socialized understanding of the stimulus value. Assessing all these three components together helps better specify the typical and atypical operation of basic affective processes that underlie social and emotional functioning in people with ASD.

1. Motivational response system: affective startle modulation

The modulation of a startle response during exposure to affective stimuli has been used extensively to study aversive and appetitive response systems in the brain (Davis, 1997; LeDoux, 2000). This method has several benefits. Responses are not contingent upon motor planning, language, or participants' voluntary actions (Ornitz, Lane, Sugiyama, & de Traversky, 1993). Startle modulation is evident across the lifespan, from infancy through adulthood (Balaban, 1995; McManis, Bradley, Berg, Cuthbert, & Lang, 2001). The method is validated across several clinical populations, including individuals with anxiety disorders and sociopathy (Patrick, Cuthbert, & Lang, 1994; Patrick & Zempolich, 1998).

In typical humans and other animals, negative stimuli prime an aversive motivational response (supporting avoidance behaviors) by enhancing startle magnitude to a sudden loud noise whereas positive stimuli prime an appetitive motivational response (supporting approach behaviors) by dampening the startle responses (Bradley, Cuthbert, & Lang, 1990; Cuthbert, Bradley, & Lang, 1996; Davis, 1997). Importantly, simple startle responses are intact among individuals with ASD (Bernier, Dawson, Panagiotides, & Webb, 2005; McAlonan et al., 2002; Ornitz et al., 1993). Thus, the extent and direction of startle modulation by affect helps assess the functioning of basic motivational systems in people with ASD.

Affective startle modulation is supported by neural systems involved in basic affective processing, such as the amygdala. For example, electrical stimulation of the amygdala directly enhances startle reflex amplitude, while lesions abolish it (Davis, 1997; Lang, 1995). Thus, atypical startle modulation responses in individuals with ASD may point to a disruption in amygdala function.

Despite the value of the affective startle modulation as a probe into the early affective and motivational systems, only one study has examined it in ASD, and it obtained inconclusive results (Salmond, de Haan, Friston, Gadian, & Vargha-Khadem, 2003). Salmond and colleagues compared startle modulation to "nice" and "scary" images in children and adolescents with ASD and typical peers. Unexpectedly, in both typical and ASD groups, startle magnitudes were *smaller* during the negative stimuli than the positive. The typical group results are especially surprising as they run counter to the highly robust finding of startle *enhancement* during negative stimuli in adults. The interpretation of the study is further complicated by the absence of a neutral condition. Without it, it cannot be determined if there was a suppression of startle to the negative stimuli or an elevation of the positive. Still, these results could be interpreted as reflecting the characteristics of their sample and the characteristics of the manipulation. First, the participants were largely young males (13:1 for ASD, >50% of typical controls). Interestingly, previous research found that typical boys (but not typical girls) demonstrated *increased* startle responses with exposure to positive versus neutral stimuli and *suppressed* startle with exposure to negative versus neutral stimuli (McManis et al., 2001). Additional data showed that the typical boys did not demonstrate as strong of arousal activation (skin conductance) or affective responses (corrugator activity) to the negative stimuli as girls or adults. McMannis and colleagues proposed that the results indicate incomplete activation of the defensive response system in the young boys. Second, it is worth pointing out that the Salmond and colleagues study did not control for the arousal value for the stimuli. Thus, assuming the mild nature of researcher-selected images for children, the observed decreased amplitude (reflecting orienting) to negative stimulus would be expected, rather than a defensively related increase found at higher levels of intensity for negative stimuli. As shown in previous research, decreased activation to negative or threat stimuli is also occasionally found in adult males, and also across genders when the stimulus intensity is low (Bradley & Lang, 2007; Cuthbert et al., 1996). Finally, the startle probe in Salmond and colleagues (2003) was set at a significant lower volume than is customary for startle research, which could further weaken activation of the aversive motivation system. In short, the previous research on affective startle modulation in ASD is not conclusive regarding the presence and nature of any atypicalities.