



Altered neurophysiological responses to emotional faces discriminate children with ASD, ADHD and ASD + ADHD



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ARTICLE INFO

Article history:

Received 25 April 2014

Accepted 25 August 2014

Available online 30 August 2014

Keywords:

ASD

ADHD

Comorbidity

Emotion

Event-related potentials (ERP)

ABSTRACT

There are high rates of overlap between autism spectrum disorder (ASD) and attention deficit hyperactivity disorder (ADHD). Emotional impairment in the two disorders, however, has not been directly compared using event-related potentials (ERPs) that are able to measure distinct temporal stages in emotional processing. The N170 and N400 ERP components were measured during presentation of emotional face stimuli to boys with ASD ($n = 19$), ADHD ($n = 18$), comorbid ASD + ADHD ($n = 29$) and typically developing controls ($n = 26$). Subjects with ASD (ASD/ASD + ADHD) displayed reduced N170 amplitude across all stimuli, particularly for fearful versus neutral facial expressions. Conversely, subjects with ADHD (ADHD/ASD + ADHD) demonstrated reduced modulation of N400 amplitude by fearful expressions in parietal scalp regions and happy facial expressions in central scalp regions. These findings indicate a dissociation between disorders on the basis of distinct stages of emotion processing; while children with ASD show alterations at the structural encoding stage, children with ADHD display abnormality at the contextual processing stage. The comorbid ASD + ADHD group presents as an additive condition with the unique deficits of both disorders. This supports the use of objective neural measurement of emotional processing to delineate pathophysiological mechanisms in complex overlapping disorders.

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1. Introduction

Many of the characteristic social impairments of autism spectrum disorder (ASD) are likely to involve the ability to process socio-emotional signals including information from faces (Baron-Cohen, 1995; Baron-Cohen, Tager-Flusberg, & Cohen, 1993), particularly as there is consistent evidence of face processing deficits among these subjects (Dawson, Webb, & McPartland, 2005). Deficits are reported for the recognition of emotional expressions in ASD (Harms, Martin, & Wallace, 2010), with the greatest deficits shown for negative expressions (Ashwin, Chapman, Colle, & Baron-Cohen, 2006; Boraston, Blakemore, Chilvers, & Skuse, 2007; Uljarevic & Hamilton, 2012; Wallace, Coleman, & Bailey, 2008). Recent findings suggest there is substantial co-occurrence between

ASD and attention deficit hyperactivity disorder (ADHD) that can be largely accounted for by overlapping genetic influences (Rommelse, Franke, Geurts, Hartman, & Buitelaar, 2009; Rommelse, Geurts, Franke, Buitelaar, & Hartman, 2011). Although the majority of research in ADHD has focused on the core symptoms of inattention, hyperactivity and impulsivity, there is accumulating evidence to suggest emotional impairment in the disorder (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Uekermann et al., 2010). In the limited studies available, deficits are reported in recognising and identifying emotion both generally (across different emotions) and for specific emotional expressions, as well as understanding their contextual meaning (Dickstein & Castellanos, 2012), although inconsistencies pertaining to methodological differences by age and intellectual ability are apparent.

Similar performance on emotion identification tasks (labelling or matching emotional faces) has been reported in direct comparisons of ASD and ADHD (Buitelaar, Van der Wees, Swaab-Barneveld, & van der Gaag, 1999; Fine, Semrud-Clikeman, Butcher, & Walkowiak, 2008), although some studies demonstrate poorer

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performance in ASD (Downs & Smith, 2004), particularly when controlling for intellectual ability (Dyck, Ferguson, & Shochet, 2001). A comparison of emotional face recognition revealed increased error rates for identification of emotional expressions in ASD + ADHD (for surprise) and ADHD-only (for joy) compared with ASD-only and typically developing controls (Sinzig, Morsch, & Lehmkuhl, 2008). These findings support efforts to uncover the neural correlates of these deficits and their overlap with those demonstrated in ASD. This is of particular importance in elucidating the basis of the co-occurrence of ASD and ADHD, whereby investigation of underlying pathophysiological mechanisms may reveal whether the comorbidity reflects an additive condition or a separate condition with distinct impairments (Rommelse et al., 2011; Taurines et al., 2012). While there is consistent evidence for abnormalities in fronto-striatal-cerebellar and frontoparietal neural circuits involved in cognitive control in ADHD (Cubillo, Halari, Smith, Taylor, & Rubia, 2012; Dickstein, Bannon, Castellanos, & Milham, 2006; Durston, van Belle, & de Zeeuw, 2011), which also show abnormal activation during reward processing (Cubillo et al., 2012), little is known about brain processing during face processing. In contrast, there are several neuroimaging studies that indicate abnormal activation of brain regions subserving social cognition in ASD, with activation differences shown in the frontal gyrus, superior temporal gyrus, inferior parietal lobe and fusiform gyrus during face processing (Gargarò, Rinehart, Bradshaw, Tonge, & Sheppard, 2011; Rommelse et al., 2011; Taurines et al., 2012). Measurement of event-related potentials (ERPs) during emotional face processing enables investigation of the distinct temporal stages (McLoughlin, Kuntsi, Brandeis, & Banaschewski, 2005; Tye, McLoughlin, Kuntsi, & Asherson, 2011), and the utility of ERPs in teasing apart cognitive impairments in and neurophysiological underpinnings of ASD and ADHD has been recently demonstrated (Tye et al., 2013, 2014).

A neuroscientific approach to face processing has recognised specific brain regions and circuits involved in face processing, notably referring to a 'core' system that underlies the early visual processing of faces, and an 'extended' system for more complex processing of face information (Gobbini & Haxby, 2007; Haxby, Hoffman, & Gobbini, 2000). Emotional facial expressions elicit robust neural changes that can be measured using ERPs that are likely to be related to these distinct circuits (Dickstein & Castellanos, 2012; Lang, Bradley, & Cuthbert, 1998). The lateral occipito-temporal face-sensitive N170 component is generally thought to be unaffected by changes in emotional expression (Eimer & Holmes, 2002; Munte et al., 1998), although inversion of faces elicits a larger and delayed N170 (Bentin, Allison, Puce, Perez, & McCarthy, 1996). Accordingly, the N170 is proposed to reflect early automatic structural encoding of faces, which occurs prior to a comparison of these structural descriptions with representations stored in semantic memory (Ashley, Vuilleumier, & Swick, 2004; Bentin & Deouell, 2000; Eimer & Holmes, 2002; Eimer, Holmes, & McGlone, 2003; Holmes, Winston, & Eimer, 2005). The N170 has been associated with brain processing in the lateral occipital-temporal regions, particularly the superior temporal sulcus (STS) and the fusiform gyrus (FG) (Itier & Taylor, 2004; Rossion, Joyce, Cottrell, & Tarr, 2003), thus related to the 'core' face processing system. Accordingly, the FG is involved in the invariant aspects of faces and the STS is involved in early visual processing (Haxby, Hoffman, & Gobbini, 2002). The later centro-parietal N400 component is proposed to be associated with semantic processing to allow contextual and meaning evaluation, supported by its modulation by familiarity and emotional expressions (Balconi & Pozzoli, 2005; Debruille, Pineda, & Renault, 1996; Jemel, Schuller, & Goffaux, 2009; Kutas & Federmeier, 2011; Munte et al., 1998; Olivares, Iglesias, & Rodriguez-Holguin, 2003; Posamentier & Herve, 2003; Schweinberger & Burton, 2003). The N400 has been hypothesised to reflect processing in amygdala-cortical circuitries (Williams et al., 2004) and more generally a

highly distributed neural network (Kutas & Federmeier, 2011), thus related to the 'extended' face processing system (Davidson, 2001; Haxby et al., 2002). The temporal differentiation of these components therefore supports a stage-like model of face processing with expression-independent structural encoding occurring earlier than semantic processing (Bruce & Young, 1986). More recent studies, however, challenge the view that structural encoding is temporally distinct from emotion processing and show modulation of the N170 by emotional expressions, as shown by larger amplitude and longer latency (Batty & Taylor, 2003; Blau, Maurer, Tottenham, & McCandliss, 2007; Caharel, Courty, Bernard, Lalonde, & Rebai, 2005; Campanella, Quinet, Bruyer, Crommelinck, & Guerit, 2002; Pizzagalli et al., 2002). Here, therefore, we focus on (1) the N170, as a large body of work supports it as a face-sensitive component compared to earlier visual processing components (e.g. P1), and (2) the N400, which is a suitable and sensitive late component for this younger age group. In addition, the present task has been specifically validated for measuring the N170 and N400 components (Battaglia et al., 2005, 2007; Bertoletti, Zanoni, Giorda, & Battaglia, 2012; for review of other components, see Eimer & Holmes, 2007) and these components are associated with neural sources known to be abnormally activated in ASD and ADHD (see above).

Abnormal ERP responses to emotional face expressions have been reported in ASD; consistent alterations are demonstrated for the N170 in response to faces alone (Dawson et al., 2002, 2005; Grice et al., 2005; McPartland, Dawson, Webb, Panagiotides, & Carver, 2004; Tye et al., 2013; Webb, Dawson, Bernier, & Panagiotides, 2006), with delayed and reduced N170 components to emotional faces (Batty, Meaux, Wittemeyer, Roge, & Taylor, 2011) and reduced modulation of the N170 by fear compared to typically developing individuals (Wagner, Hirsch, Vogel-Farley, Redcay, & Nelson, 2013). Nevertheless, other studies report differences only in adults (O'Connor, Hamm, & Kirk, 2005) or no differences in ERP components but rather in source localisation activity (Wong, Fung, Chua, & McAlonan, 2008). The limited ERP studies of emotion processing in ADHD show discrepant findings; in adults, reduced modulation of the N170 component particularly for positive emotion is reported in ADHD (Ibanez et al., 2014, 2011), whereas in adolescents with ADHD an enhanced N170 is shown (Williams et al., 2008). The degree to which semantic processing indexed by the N400 is altered in ASD and ADHD is poorly understood, yet given evidence for abnormal processing of emotional faces at later stages of processing in older adolescents and adults, examination of the N400 is warranted in these disorders (e.g. P300, LPP, N300; Dawson, Webb, Carver, Panagiotides, & McPartland, 2004; Singhal et al., 2012; Williams et al., 2008).

Findings to date converge to suggest both individuals with ASD and ADHD demonstrate behavioural and neurophysiological abnormalities in response to emotional face expressions at both early and later stages of processing. The two disorders, however, have not been directly compared and there is no ERP study on emotion processing in children presenting with symptoms of both disorders. The aim of the present study was, therefore, to compare children with comprehensively assessed diagnoses of ASD, ADHD and comorbid ASD + ADHD. Based on the neural models of emotion processing and known neural atypicalities of the disorders reviewed above, we expected (i) both ASD and ADHD groups to show abnormal neurophysiological responses to emotional face expressions; (ii) children with ASD to show specific deficits in processing negative emotional expressions, whereas children with ADHD to show deficits processing both positive and negative emotional expressions; and (iii) children with comorbid ASD + ADHD to show abnormalities of both disorders. In particular, the classification of emotional responses by the two dimensions of "motivational salience" is likely to be informative for examining general versus specific effects of: (1) arousal, referring to the

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