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Callous-unemotional traits moderate executive function in children with ASD and ADHD: A pilot event-related potential study

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ABSTRACT

Attention deficit hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) are associated with varied executive function (EF) difficulties. Callous-unemotional (CU) traits, a proposed antecedent of adult psychopathy, are often associated with intact or enhanced EF. Here we test whether CU traits may therefore modulate EF in ASD and ADHD, in which EF is typically impaired. We collected CU traits and measured event-related potentials (ERPs) that index EF during a cued-continuous performance test (CPT-OX) in boys with ASD, ADHD, comorbid ASD + ADHD and typical controls. We examined attentional orienting at cues (Cue-P3), inhibitory processing at non-targets (NoGo-P3) and conflict monitoring between target and non-target trials (Go-N2 vs. NoGo-N2). In children with ASD, higher CU traits were associated with an enhanced increase in N2 amplitude in NoGo trials compared to Go trials, which suggests relatively superior conflict monitoring and a potential cognitive strength associated with CU traits. The results emphasise the importance of considering the effects of co-occurring traits in the assessment of heterogeneity of EF profiles in neurodevelopmental disorders.

1. Introduction

Autism spectrum disorder (ASD) and attention deficit hyperactivity disorder (ADHD) are two common childhood-onset disorders that show substantial behavioural and genetic overlap (Ronald et al., 2008). Impairments in executive function (EF), behaviours such as planning, online monitoring and working memory, characterise both children with ASD and ADHD, and may underlie some of the behavioural features of the disorders (Happé et al., 2006; Rommelse et al., 2011). Children with ASD often perform poorly on tasks requiring planning and mental flexibility, while children with ADHD consistently demonstrate difficulties inhibiting responses (Geurts et al., 2004; Happé et al., 2006). Event-related potentials (ERPs) which capture distinct underlying neural processes related to these functions, have demonstrated that ASD and ADHD can be dissociated on the basis of their neurophysiological responses during attentional (Tye et al., 2014a) and social cognitive tasks (Tye et al., 2013, 2014b). Specifically, children with ADHD symptoms (both ADHD and comorbid ASD *and* ADHD; ASD + ADHD) demonstrate impairment in response inhibition (reduced NoGo-P3 to non-targets) and attentional orienting (reduced Cue-P3 to cue/warning stimuli), while children with ASD (ASD and ASD + ADHD) show reduced conflict monitoring (reduced N2 enhancement from Go (target) to NoGo (non-target) trials; Tye et al., 2014a), on a cued Continuous Performance Task (CPT-OX). These findings indicate that impaired EF processes are distinct in ASD and ADHD, whereas children with co-occurring ASD + ADHD present as an additive co-occurrence with the unique deficits of both disorders. Still, little is known about the role of other co-occurring traits in moderating EF in ASD and ADHD and their overlap, particularly those that are associated with typical EF. The recent shift toward dimensional over categorical approaches in psychopathology (Cuthbert and Insel, 2013) emphasises the importance of a transdiagnostic approach, assessing traits rather than categorical disorders. Linking neurocognitive markers to dimensions will likely be more informative in terms of understanding the underlying mechanisms.

There has been growing interest in the comorbidity demonstrated between psychopathic tendencies, anti-social behaviour and both ASD and ADHD (Colledge and Blair, 2001; Kadesjö and Gillberg, 2001; Simonoff et al., 2008; Leno et al., 2015). Children with ASD display increased antisocial and aggressive behaviour (Bauminger et al., 2010) and a quarter to a third of individuals have a co-occurring diagnosis of oppositional defiant disorder (ODD) and/or conduct disorder (CD;

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Simonoff et al., 2008; Kaat et al., 2013). These disruptive behaviours tend to have a highly stable and persistent course when left untreated and are associated with a higher rate of dysfunctional outcomes. Follow-up studies of children with ADHD indicate 21% meet criteria for antisocial personality disorder (ASPD) in young adulthood, with the severity of childhood conduct problems as a contributory factor (Fischer et al., 2002). Psychiatric comorbidity in ASD is a major factor contributing to violent offending (Woodbury-Smith et al., 2005; Newman and Ghaziuddin, 2008) and there are consistent associations between ASD traits and psychopathic traits (Soderstrom et al., 2005). A longitudinal study, however, suggested no risk for ASPD in adult patients with a childhood diagnosis of ASD (0%), but an increased risk for those with childhood-onset ADHD (30.9%) and ASD + ADHD (18.5%; Anckarsäter et al., 2006), although ASPD may be more common in pervasive developmental disorder-not otherwise specified (Hofvander et al., 2009). The pathophysiological mechanisms underlying the developmental trajectories to antisocial behaviour and psychopathic traits may be separable in ASD and ADHD.

Recent research has highlighted the role of callous-unemotional (CU) traits, characterised by a lack of guilt, remorse and empathy, in increased risk for persistent antisocial behaviour and adult psychopathy (Barry et al., 2000; Frick et al., 2003; Viding, 2004; Frick and White, 2008; Frick et al., 2014). Individuals with CU traits represent a putative subgroup of antisocial behaviour that show several distinct cognitive and emotional characteristics (Frick et al., 2008). At the behavioural and neural level, CU traits are associated with a selective impairment in affective processing, which can be differentiated from the social cognitive deficits observed in ASD (Jones et al., 2010; Schwenck et al., 2012; Wallace et al., 2012; Lockwood et al., 2013; O'Nions et al., 2014). Accordingly, a “double hit” hypothesis has been proposed whereby individuals with ASD and elevated CU traits exhibit the unique profiles that are independently associated with each disorder rather than being inherently related to the core symptom of ASD (Rogers et al., 2006). In support of distinct causal factors associated with ASD and CU traits, largely independent genetic and environmental influences have been reported for ASD and psychopathic and CU traits (Jones et al., 2009).

While limited research has been conducted specifically investigating EF in individuals with high CU traits, attentional processes and EF have been studied with relation to antisocial behaviour and psychopathic traits (Morgan and Lilienfeld, 2000; Blair, 2005; Blair and Mitchell, 2009). Here we focus on the EF domains indexed in the cued CPT-OX (Tye et al., 2014a). Studies that have assessed response inhibition directly have shown that adults with psychopathy or elevated psychopathic traits tend to make more commission errors than typical adults during Go/No-Go tasks (Lapierre et al., 1995; Munro et al., 2007; Sellbom and Verona, 2007) and show reduced amplitude of the N275 ERP to NoGo stimuli (Kiehl et al., 2000), which suggests an inhibitory deficit. In contrast, another study showed that psychopathic offenders demonstrate the typical increase in the frontal N2 from Go to NoGo trials (Munro et al., 2007), which suggests intact conflict monitoring. This is indirectly supported by typical or better performance by individuals with high psychopathic traits compared to typical controls on attentional set-shifting tasks, such as the Wisconsin Card Sorting Task (Lapierre et al., 1995; Ishikawa et al., 2001; Mitchell et al., 2002) and executive attention tasks that involve conflict or error monitoring (Lapierre et al., 1995; Mitchell et al., 2002; Blair et al., 2006; Bresin et al., 2014). There is no work directly investigating the association between psychopathy and attentional orienting, as measured in Tye et al. (2014a), but there is some evidence for weaker alerting during the Attentional Network Task, indexing preparedness to respond, demonstrated by reduced P1 amplitude (Racer et al., 2011).

There is evidence, therefore, for typical or even superior EF in individuals with high psychopathic traits (Moffitt, 2003; Blair, 2005), which suggests that in some instances CU traits in childhood may confer a advantage through relative cognitive strengths. The potential buffering effect of CU traits has been shown previously, whereby higher verbal intelligence (Loney et al., 1998) and better social problem-solving abilities (Waschbusch et al., 2007) are demonstrated in children with conduct problems and CU traits compared to children with conduct problems alone. In the current study, we examine the moderating effect of CU traits within an ASD/ADHD population that has impaired EF. Previous findings have suggested that individuals with ASD and CU traits or delinquency show impairments in emotion recognition, yet EF is unaffected (Woodbury-Smith et al., 2005; Rogers et al., 2006; Leno et al., 2015). High CU traits in ASD (and ADHD) may therefore offer particular cognitive strengths that are associated with distinct neurophysiological profiles.

The aim of this preliminary study is to provide proof-of-concept that abnormal EF in children with ASD and ADHD is conditional upon the level of CU traits, using the same sample and analyses described in Tye et al. (2014a). Specifically, we investigate whether EF is a relative cognitive strength in individuals with CU traits and ASD/ADHD, and whether this effect differs between ASD and ADHD. We focus here on sensitive ERP markers of EF to enable investigation of covert and distinct information processing stages, selected on the basis of previous findings indicating specificity to ASD or ADHD (Tye et al., 2014a). We investigated (1) associations between CU traits and attentional orienting, inhibitory control and conflict monitoring; and (2) the moderating role of CU traits on the association between ASD, ADHD and ERP-indexed EF.

2. Material and methods

2.1. Sample

The sample from Tye et al. (2014a) was used for these analyses. Nineteen male participants with ASD, 18 with ADHD, 29 with ASD and ADHD, and 26 typically developing controls (TDC) took part in the study. The age range was 8–13 years; there was no significant difference in age across groups (Table 1). All participants were required to have an IQ > 70, normal or corrected-to-normal vision, and not to be taking any medication except for stimulants (6 participants with ADHD, 6 participants with ASD + ADHD), which had to be interrupted 48 h prior to testing sessions. Exclusion criteria included non-fluent English, specific medical disorders, other comorbid psychiatric disorder including conduct disorder (not including ODD), history of traumatic brain injury and a diagnosis of epilepsy.

The participants were recruited from out-patient neurodevelopmental clinics and local parent support groups in southeast London. All participants had a clinical diagnosis made according to ICD-10 criteria (autism, Aspergers syndrome, ADHD combined type) and then underwent systematic and rigorous clinical assessment to confirm pure or comorbid research diagnosis (see Tye et al., 2014a). All cases were initially evaluated with Conners' 3rd Edition Parent Rating Scale short form (Conners, 2008) and Social Communication Questionnaire (SCQ; Rutter et al., 2003). Cases of ASD were diagnosed using the Autism Diagnostic Interview–Revised (ADI-R; modified criteria IMGSAC, 1998) and the Autism Diagnostic Observation Schedule (ADOS-G; Gotham et al., 2007). Cases of ADHD were diagnosed using Parent Account of Childhood Symptoms (PACS; Taylor et al., 1986), which has been extensively used by the IMAGE consortium (Chen et al., 2008). Co-morbid ASD + ADHD cases met full diagnostic criteria for ASD using the ADI-R/ADOS and full diagnostic criteria for ADHD using the PACS. Trained and research-reliable postgraduate researchers carried out the ASD and ADHD research diagnostic assessments. Two additional measures were administered to aid group classification and in-depth assessment where diagnostic classification was unclear: the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997) and Development and Wellbeing Assessment (DAWBA; Goodman et al., 2000). An experienced clinical academic (PB) reviewed the available data and decided on the ‘best estimate’ diagnosis using this multi-measure multi-informant approach, with greater weight given to clinical diagnosis, followed by ADI-R,

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