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A test of the 'parent distortion' hypothesis when assessing generalised anxiety disorder in boys with an autism spectrum disorder

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ABSTRACT

The 'parent distortion' hypothesis regarding assessment of a child's anxiety state was examined in the mothers of 128 boys with an autism spectrum disorder. Mothers' own **generalised anxiety disorder (GAD)** and their ratings of their sons' **GAD** were compared with the boys' self-ratings and the ratings of the boys given by a clinician. Boys' cortisol concentrations were also explored for their association with these three sources of **GAD** ratings. Results indicated that mothers' **GAD** was significantly and directly correlated with the ratings they gave for their sons' but that only mothers who were above-minimally anxious gave ratings of their sons' anxiety that significantly agreed with those from the clinicians. Minimally-anxious mothers appeared to underestimate their sons' anxiety, and these effects generalised to their sons' self-ratings of anxiety. Associations between the boys' cortisol concentrations suggested an interaction between the presence of the diurnal fluctuation in boys' cortisol concentrations and mothers' anxiety states.

1. Introduction

1.1. Parental distortion of child anxiety state

The presence of bias in parents' reports of their child's psychological state has been extensively studied and is sometimes referred to as a "distortion" that arises from the parent's own psychological distress (Richters, 1992, p. 485). Following some early work which focused on the effects of mothers' anxiety states upon the ratings they gave of their children's anxiety (Engel, Rodrique, & Geffken, 1994; Frick, Silverthorn, & Evans, 1994), a review of this field concluded that greater anxiety in mothers was directly associated with them giving more severe ratings for their children's anxiety (de Los Reyes & Kazdin, 2005). However, an indication that this was not a simple relationship came from a recent study which reported an interaction between maternal anxiety and age of the child, with mothers of older children rating their children's anxiety

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higher than the self-ratings given by their children but mothers of younger children giving lower ratings than their children's self-ratings (Niditch & Varela, 2011).

1.2. Assessing anxiety in autism spectrum disorder

Children and adolescents with an autism spectrum disorder (ASD) experience high levels of anxiety (APA, 2013; Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; van Steensel, Bogels, & Perrin, 2011). As well as presenting a clinical challenge for these young people, this comorbidity of ASD and anxiety can potentially confound accurate diagnosis and treatment planning for them, thus making the accurate assessment of anxiety in these young people an issue of significant clinical importance. However, much of the assessment and diagnosis of anxiety relies solely upon parents' assessment of their children's anxiety. For example, in their meta-analysis of 31 studies of anxiety in children with an ASD, van Steensel et al. (2011) were unable to include source of anxiety ratings as a variable because studies "were heavily dominated by parent reports" (p. 307). In the few studies that have examined the agreement in anxiety ratings given by children with an ASD about themselves and those given about them by their parents, the findings are inconclusive as regards parental bias. For example, parents rated their ASD children's anxiety more severely than the children did themselves in several studies of children aged between 7 and 17 years (Blakeley-Smith, Reaven, Ridge, & Hepburn, 2012; Gillott, Furniss, & Walter, 2001; Russell & Sofronoff, 2005; Storch et al., 2012) but another study of children aged between 11 and 17 years found that they rated their anxiety higher than the ratings their parents gave for them (Hurtig et al., 2009). Although some of those studies included interview data from professional clinicians (*e.g.*, Storch et al., 2012), most relied on self-reports from parents and their children. Some of the disagreement between the ratings that parents give for the anxiety state of their child with an ASD compared to the self-ratings from those children may be due to the limitations which these children exhibit in being able to understand the language used in anxiety scales and/or the relevance of such scales for them because most scales are developed on non-ASD samples (Mazefsky, Kao, & Oswald, 2011). While this may suggest that parents' ratings of their child's anxiety might be the most accurate source of information, the possible presence of parental bias in this process also needs to be considered. One way to clarify whether such bias exists and also to provide some insight into the relative accuracy of parents' *vs* children's assessment of the latter's anxiety state is via comparison with external criteria.

1.3. Using expert clinicians and biological indicators of anxiety

1.3.1. Expert clinicians

Using anxiety assessment performed by clinicians could provide a triangulation method for assessing the validity of mothers' ratings of their ASD children's anxiety, the validity of the children's self-ratings of their own anxiety, and the presence of any bias due to mothers' own anxiety (which has received scant attention in children with an ASD). Because such a process goes beyond the comparison of only parents' and their children's ratings of the children's anxiety that has been used in most previous research, it has the potential to clarify the nature of the association between parental anxiety state and their reports on their children's anxiety in young people with an ASD as well as to provide some insights into the relative validity of parents' and children's assessment of the latter's anxiety states.

1.3.2. A biological indicator of anxiety—Cortisol concentrations

Several studies have reported on the significant association between elevated cortisol and clinical anxiety in children (*e.g.*, Greaves-Lord et al., 2007; Lanni, Schup, Simon, & Corbett, 2012). Cortisol collected in saliva 30 min after waking in the morning (called 'waking cortisol'), or the difference between cortisol immediately after waking and 30 min later (referred to as the Cortisol Awaking Response—CAR) may be used as an index of elevated physiological responses to stress or anxiety (Miller, Chen, & Zhou, 2007). Cortisol is released from the adrenal cortex following a cascade of prohormones from the hypothalamus and pituitary. Concentrations of cortisol may fluctuate during the day in response to the diurnal fluctuation (DF) in the hypothalamus–pituitary–adrenal (HPA) axis, with a peak shortly after waking (*i.e.*, 'waking' cortisol), decreasing then to the early evening (Weitzman et al., 1971). However, this DF is not present in all persons and may be disrupted or absent in 17% of some adult samples (Smyth et al., 1997), with day to day variations in the overall range of fluctuation in about 30% of samples (Smyth et al., 1997) and depression of the DF due to a range of factors in up to 50% of some samples (Stone et al., 2001). Additionally, the precise time points of maximum and minimum cortisol concentrations vary across individuals (Smyth et al., 1997).

1.3.3. Cortisol and anxiety in children with an ASD

In a recent review of cortisol in children with an ASD, Taylor and Corbett (2014) noted that proportions of these children are likely to show disruption of their cortisol DF (which then affects the CAR) but that afternoon cortisol concentrations were comparable to those in non-ASD children, potentially arguing for collection of cortisol in the afternoon as well as in the morning. Although (as mentioned above) the association between cortisol and anxiety has been demonstrated for non-ASD children (Greaves-Lord et al., 2007; Lanni et al., 2012), the data regarding cortisol as an indicator of anxiety in children with an ASD is less well-established, possibly due to the confound caused by inconsistency in the DF in children with an ASD. However, at least one previous study did demonstrate a significant association between cortisol and GAD in children with an ASD (Bitsika, Sharpley, Sweeney, & McFarlane, 2014), which was replicated in a further study that investigated the effects of