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Prevalence, structure and correlates of anxiety-depression in boys with an autism spectrum disorder

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ABSTRACT

Background: Comorbidity of anxiety and depression predicts impaired treatment outcomes, poor quality of life and increased suicide risk. No study has reported on a combined measure of anxiety-depression in boys with an Autism Spectrum Disorder.
Aims: To explore the prevalence, underlying factor structure and relationships between anxiety-depression, physiological stress and symptoms of Autism Spectrum Disorder (ASD).
Methods: 150 boys (aged 6–18 years; IQ $M = 94.9$, range = 73–132) with an ASD plus their parents (135 mothers, 15 fathers) completed scales about the boys' anxiety and depression, and the boys provided samples of their saliva in the morning and afternoon. Parents also completed the ASD Behaviour Checklist about the boys' ASD symptoms.
Results: The two sources of ratings were not significantly different for prevalence of anxiety-depression but the factor structures varied between the parents' and boys' responses, with a four-factor solution for the boys' ratings and a three-factor solution for the parents' ratings. There were also differences in the correlations between cortisol and anxiety-depression and between ASD symptoms and anxiety depression across the boys' and parents' data.
Conclusions: Assessment of anxiety and depression comorbidity from parents and from children with an ASD themselves could provide a valuable adjunct datum when diagnosing ASD.

1. Introduction

1.1. Comorbidity of anxiety and depression

Comorbidity of Generalised Anxiety Disorder (GAD) and Major Depressive Disorder (MDD) is associated with impaired improvement following treatment (Coryell et al., 1988; Fava et al., 1997; Gaynes et al., 1999), delayed recovery, poorer quality of life, and increased suicide risk (Brown & Kroenke, 2009; Vollrath & Angst, 1989). Even though they are often

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examined as separate disorders in comorbidity studies, it has been suggested that the distinction between anxiety and depression is artificial and solely due to limitations engendered by a categorical model of disorder (Widiger & Samuel, 2005). For example, GAD and MDD share the common symptom of fatigue (APA, 2013; Zinbarg et al., 1994) and irritability in children (APA, 2013). They may also be causally linked, with elevated GAD during adolescence increasing the risk of developing MDD later in life (Muris, Merckelbach, Schmidt, Gadet, & Bogie, 2001). It may therefore be that studies of comorbidity of anxiety and depression could benefit from consideration of the underlying structure of a combined measure of the two disorders rather than separate measures of GAD and MDD.

1.2. Anxiety and depression in children with an Autism Spectrum Disorder (ASD)

Children with an ASD also often experience comorbid anxiety or depression (Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; Simonoff et al., 2008), which may arise as a response to the experience of having the ASD itself (e.g., from difficulties in communication and socialisation) but can also reciprocally contribute to limitations in trusting others and engaging in social interactions that appear to the child with an ASD to be unpredictable or invasive (APA, 2013). A review of 31 studies of anxiety in children and adolescents with an ASD reported prevalence of 7.5–84.1% for at least one anxiety disorder (van Steensel, Bogels, & Perrin, 2011). This is considerably higher than the prevalence of anxiety among non-ASD children aged 9–16 years (i.e., <2.5%: Costello, Egger, & Angold, 2005). Similar findings have been reported for depression in children with an ASD, with prevalence as high as 30% (Matson & Nebel-Schwalm, 2007) compared to just 3.5% in an Australian sample of 4083 non-ASD males and females between 4 and 17 years of age (Sawyer et al., 2001). In a study of that measured both anxiety and depression in 59 children with an ASD, Kim et al. (2000) reported prevalence of 13.6% for anxiety and 16.9% for depression, well above those described above for non-ASD samples.

Although it is sometimes suggested that GAD may be a part of the diagnosis of ASD itself (Wood & Gadow, 2010), the reporting of significant correlations between the presence of GAD symptoms and biological indicators of GAD (e.g., salivary cortisol) in only some children with an ASD (Taylor & Corbett, 2014) suggests that GAD may be independent of ASD but often comorbid with it. Under the DSM-5 (APA, 2013) definition of GAD, a differential diagnosis may be made if the GAD is "a physiological effect" of ASD. That physiological connection remains to be validated in the ASD population and so the conservative position to take at the moment is that the two diagnoses are independent but often comorbid.

1.3. Correlates of anxiety and depression in children with an ASD

Several previous studies have reported on the association between GAD and/or MDD and various correlates in children with an ASD, including their ASD symptom profile (e.g., Kim et al., 2000; Steensel et al., 2011; Stranga et al., 2012), but there have been no identified reports of the correlations between ASD symptoms and the factors underlying GAD-MDD in these children. As well as being novel research that can extend the literature regarding the correlates of anxiety-depression among these children, such an investigation might assist in the identification of potential therapeutic targets for the treatment of anxiety-depression in young people with an ASD. Similarly, because GAD and MDD are usually linked to prolonged stress and have been associated with elevated salivary cortisol in a range of people (Gillespie & Nemeroff, 2005) including children with an ASD (Bitsika, Sharpley, Andronicos, & Agnew, 2015; Corbett, Schupp, Levine, & Mendoza, 2009), the factors underlying the combined GAD-MDD might also be similarly associated with cortisol concentrations in children with an ASD but these relationships have not previously been reported.

The association between cortisol and GAD/MDD is sometimes explained as an outcome of prolonged activation of the Hypothalamus-Pituitary-Adrenal (HPA) axis to chronic stress, which may be facilitated by genetic disposition and may lead to hyperesponsivity of the HPA axis under stressful environmental conditions, leading to GAD and MDD (Du et al., 2009; Fiocco, Wan, Weekes, Pim, & Lupien, 2006; Gillespie & Nemeroff, 2005; Segerstrom & Miller, 2004). Up-regulation of the HPA axis instigates hypercortisolemia, which can induce organic changes to several brain regions that are associated with cognitive processing of the threat valence of environmental demands and the selection of rational vs emotional responses to those demands (Chrousos, 2009). In this way, organic changes to the brain have been linked to the eventual development of the withdrawal behaviours that underlie depressive symptomatology (Bolling, Kohlenberg, & Parker, 1999; Kanter, Busch, Weeks & Landes, 2008). These organic changes include alterations to the volumes of certain brain regions such as the amygdala (van Eijndhoven et al., 2009), prefrontal cortex (Levin, Heller, Mohanty, Herrington & Miller, 2007) and hippocampus (MacQueen, Yucel, Taylor, Macdonald & Joffe, 2008). There are also alterations to the connectivity of those regions of the brain (e.g., amygdala-prefrontal: de Almeida et al., 2009; amygdala-hippocampus: Fu et al., 2004). Although beyond the scope of this paper, these models provide a hypothetical basis for the association between GAD, MDD and cortisol and argue for the use of salivary cortisol as an indicator of prolonged or chronic stress that is associated with GAD and MDD.

1.4. Self-reports of anxiety and depression

A further variation upon previous studies of anxiety and depression that was included in this study was the collection of those data about the child with an ASD from themselves as well as from their parent. Many previous studies of these disorders in children with an ASD have relied upon the parents' ratings of their child's GAD or MDD, but recent findings indicate that these children are capable of understanding and reporting upon their anxious and depressed states