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EEG and electrodermal activity in girls with Attention-Deficit/Hyperactivity Disorder

F.E. Dupuy [a,b,*], A.R. Clarke [a,b], R.J. Barry [a,b], M. Selikowitz [c], R. McCarthy [c]

[a] Brain & Behaviour Research Institute and the School of Psychology, University of Wollongong, Wollongong 2522, Australia
[b] Centre for Psychophysics, Psychophysiology, and Psychopharmacology, University of Wollongong, Wollongong 2522, Australia
[c] Sydney Developmental Clinic, 6/30 Carrington St., Sydney 2000, Australia

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HIGHLIGHTS

- This is the first study to support hypoarousal in girls with AD/HD.
- A positive correlation found between SCL and alpha in AD/HD girls implies an anomalous arousal mechanism.
- This study also explores intriguing relationships between physiology and symptom behaviours.

ABSTRACT

Objective: This study investigated the Hypoarousal Model of Attention-Deficit/Hyperactivity Disorder (AD/HD) in girls.
Methods: 40 girls with AD/HD and 40 girl controls (aged 7–12 years) had an eyes-closed resting EEG recorded from 19 electrodes and Fourier transformed. Estimates for total power, absolute and relative power in the delta, theta, alpha, beta and gamma frequency bands, and theta/beta ratio were analysed in nine cortical regions. Skin conductance level (SCL) was simultaneously recorded. Regression analyses explored relationships between symptoms and physiology.
Results: Compared with controls, girls with AD/HD had globally elevated relative delta, globally reduced absolute beta, and globally reduced absolute and relative gamma activity. Girls with AD/HD also had lower mean SCL. Inattentive symptoms were predicted by elevated frontal relative delta, reduced SCL, and reduced temporal relative gamma activity, while elevated hyperactive–impulsive symptoms correlated with elevated frontal relative delta activity in both the patient and control groups.
Conclusions: These EEG results are comparable with the limited female AD/HD literature. Girls with AD/HD are hypoaroused, indicated by reduced SCL, and appear to have an anomalous arousal mechanism. Absolute and relative gamma results are similar to previous findings in AD/HD children. Symptom correlations with physiology offer intriguing insights for future research.
Significance: This is the first study to examine CNS arousal exclusively in girls with AD/HD.

1. Introduction

Attention-Deficit/Hyperactivity Disorder (AD/HD) is a neurodevelopmental disorder characterised by inappropriate behaviours of inattention and/or hyperactivity-impulsivity (APA, 2000). The disorder is estimated to affect 3–7% of school children and is diagnosed more often in boys than girls; boy-to-girl ratios range from 3:1 to 9:1 (Gaub and Carlson, 1997; Arcia and Conners, 1998; Hartung and Widiger, 1998; APA, 2000; Rutter et al., 2003; Pastor and Ruben, 2008; Willcutt, 2012). With the preponderance of AD/HD being diagnosed in boys, it is no surprise that our understanding of the disorder is based heavily on research with male cohorts. However, there is increasing interest in sex differences and female profiles that are separate from male profiles (Arnold, 1996; Gaub and Carlson, 1997; Quinn, 2005; Briscoe-Smith and Hinshaw, 2006; Rucklidge, 2010; Dupuy et al., 2013b).

While boys are more likely to have AD/HD, girls with AD/HD are more likely to experience greater levels of peer rejection (Berry et al., 1985; Arnold, 1996) and abuse (Briscoe-Smith and Hinshaw, 2006), and are at a higher risk for psychological problems than boys with AD/HD (Rucklidge and Tannock, 2001). Girls with AD/

* Corresponding author at: Brain & Behaviour Research Institute and the School of Psychology, University of Wollongong, Wollongong 2522, Australia. Tel.: +61 242214458; fax: +61 242214163.
E-mail address: fdupuy@uow.edu.au (F.E. Dupuy).

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HD are also less hyperactive, but more inattentive, and have higher rates of depressive and anxiety disorders than boys with AD/HD (Carlson et al., 1997; Gaub and Carlson, 1997; Gershon, 2002; DuPaul et al., 2006; Quinn, 2005). Quinn (2005) suggested that in place of excess motor activity (the hallmark of hyperactivity), girls with AD/HD will more often display behaviours such as high emotional reactivity and excessive talking. These sex differences suggest that separate male and female profiles offer better understanding of the nature of AD/HD.

The EEG activity of the majority of children with AD/HD is characterised by a deviant baseline cortical pattern (Lansbergen et al., 2011). These children tend to have increased posterior absolute delta activity (Matousek et al., 1984; Clarke et al., 2001a,b), globally elevated absolute and relative theta activity, most often frontally (Satterfield et al., 1972; Chabot and Serfontein, 1996; Lazzaro et al., 1998; Clarke et al., 2002; Barry and Clarke, 2009; Barry et al., 2009b), and reduced relative alpha and relative beta activity (Lazzaro et al., 1998; Clarke et al., 2001a,b, 2011a,b). The gamma frequency band, although not as extensively researched as the four traditional bands, has been found to be significantly reduced in groups of AD/HD children, in both absolute and relative power (Barry et al., 2009a, 2010). A larger, aberrant theta/beta ratio has also been consistently found in AD/HD children (Lubar, 1991; Janzen et al., 1995; Monastra et al., 1999, 2001; Clarke et al., 2001a,b, 2011a,b; Synder and Hall, 2006; Barry et al., 2009b; Lansbergen et al., 2011). It is important to note that these studies are based largely on male-normed data, with minimal, if any, female inclusion.

There have been few studies that have examined testing EEG profiles exclusively in girls with AD/HD (for a review, see Dupuy et al., 2013b). One of the first, Baving et al. (1999), found that 15 AD/HD girls (aged 4–8 years) had greater frontal alpha activation in the left than right hemisphere, suggested to represent a left frontal deficit, compared with aged-matched control girls. Other studies have found that girls with AD/HD, compared with girl controls, had globally elevated absolute delta, theta and total power, greater relative theta and reduced relative beta (Clarke et al., 2001b, 2003, 2007; Dupuy et al., 2011, in press). Although these initial studies are broadly comparable to the male-based AD/HD literature (with an elevation of slow wave activity and reduction of faster wave activity), there are EEG differences between boys and girls with AD/HD. These discrepancies should not be overlooked and separate male and female profiles should be utilized as part of standard EEG-AD/HD research.

Despite our knowledge of characteristic EEG abnormalities within AD/HD, we are yet to fully understand the exact causes of the disorder. It is generally accepted that a dysfunction of the Central Nervous System (CNS) is involved, although the underlying mechanisms are not well known (Fonesca et al., 2013). The hypoarousal model proposes that the CNS is underaroused in children with AD/HD, which in turn causes symptoms of inattention and hyperactivity–impulsivity (Satterfield and Cantwell, 1974). This model has been widely used to explain the seemingly-paradoxical effect that stimulant medications have on AD/HD behaviours. In small doses, psychostimulants act by increasing arousal to normal levels, resulting in improved behaviour. Satterfield et al. (1974) found that stimulant medications improved hyperactivity and raised CNS arousal to a more optimal level. Satterfield et al. (1974) also found that hyperactive children who had low CNS arousal levels (measured by skin conductance level; SCL) also had a negative correlation between their arousal level and the severity of their behaviour: the lower SCL, the greater behavioural disturbances (distractibility and inattention). SCL is an electrodermal measure that reflects output from the sympathetic branch of the autonomic nervous system (Wallis, 1981; Boucsein, 1992; Hoeldtke et al., 1992; El-Sheikh, 2007) and is a reliable marker of physiological arousal (Raskin, 1973; Rosenthal and Allen, 1978; Raine et al., 1990; Barry and Sokolov, 1993; van Lang et al., 2007).

Initial attempts to support the hypoarousal model using electrodermal measures resulted in mixed findings; some supported underarousal in AD/HD (Satterfield and Dawson, 1971; Satterfield et al., 1974) and others found no difference in CNS arousal between AD/HD and non AD/HD subjects (Cohen and Douglas, 1972; Spring et al., 1974; Montagu, 1975). However, this disparity was suggested to be a methodological issue due to the use of Cambridge electrode jelly, which is specifically formulated to increase skin conductance (Montagu, 1975), as the contact medium. Subsequent investigations of the hypoarousal model (with inert contact media) have found more consistent results; AD/HD is associated with significantly lower SCL (Lazzaro et al., 1999; Hermens et al., 2004; Broyd et al., 2005; Barry et al., 2009b, 2012). Again, it is important to note that these studies included only males, except for Barry et al. (2012). Barry et al. (2012) included mixed-sex subject groups (26 males and 10 females), but sex was not included as a factor within their analyses. Hermens et al. (2004, 2005) also included mixed-sex groups of adolescents and adults in their studies of CNS arousal in AD/HD, but their SCL measure reflected SCL changes over time or 'rate of EDA decrement', which is not commonly reported in the literature, making it difficult to draw comparisons with other studies.

There are no published studies that directly explore CNS arousal or the hypoarousal model exclusively in girls with AD/HD. To date, it has been assumed that girls with AD/HD would have lower SCL, as boys with AD/HD appear to have, yet there has been no direct investigation of SCL in girls with AD/HD. The aim of this study is to address this gap and investigate differences in CNS arousal and EEG activity in girls with AD/HD.

2. Methods

2.1. Participants

The study included 80 Caucasian girls aged 7–12 years ($M = 9.43$, SD = 1.73). Of these, 40 were healthy controls and 40 girls were diagnosed with AD/HD (30 were diagnosed with the Inattentive type and 10 were diagnosed with the combined type – AD/HD types are not referred to in this study as our previous research found that girls with the combined and inattentive AD/HD types have indistinct EEG profiles – see Clarke et al., 2003; Dupuy et al., 2011; in press). The clinical participants were selected from patients at a paediatric practice and controls were recruited via the local community. All participants had an IQ score of 80+. Participants had no history of medication use for any psychiatric disorder, and AD/HD participants were tested nil medication.

Inclusion in the AD/HD group was based on clinical assessments made by a paediatrician and a psychologist, and both agreed on the diagnosis. Both clinicians used behavioural observations, a comprehensive history taken from parent(s)/guardian(s), school reports from the past 12 months, and any other relevant reports, to make their diagnoses.

Participants were excluded if they had a history of problematic prenatal, perinatal or neonatal periods, a history of CNS diseases, convulsions or convulsive disorders. They were also excluded if there was evidence of a consciousness disorder, head injury with cerebral symptoms, paroxysmal headaches or tics. Participants were excluded if they met criteria for Conduct Disorder, Oppositional Defiant Disorder, an anxiety or depressive disorder, Asperger's or Tourette's Syndrome.

Controls were included based on clinical interviews with parent(s)/guardian(s), similar to the AD/HD participants, described above. Control subjects scored in the normal range on measures