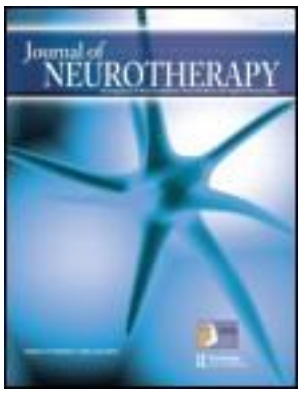

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EEG Activity in Females with Attention-Deficit/ Hyperactivity Disorder

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REVIEW ARTICLE

EEG ACTIVITY IN FEMALES WITH ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

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Although Attention-Deficit/Hyperactivity Disorder (AD/HD) affects millions of females worldwide, our understanding of AD/HD continues to be based heavily on male-dominated research. Significant sex differences reported in the presentation and diagnosis of AD/HD can no longer be ignored; females with AD/HD are different from males with the disorder. Electroencephalography (EEG) is a valuable tool for measuring electro-cortical activity and has been found useful in AD/HD research. Preliminary studies have shown that females with AD/HD have EEG abnormalities different from those found in males with AD/HD. This article reviews the current literature on EEG activity of females with AD/HD and concludes that the lack of comprehensive research draws attention to the necessity for sex-specific EEG research within AD/HD populations.

INTRODUCTION

Attention-Deficit/Hyperactivity Disorder (AD/HD) is one of the most pervasive problems within mental health and results in negative impacts on an individual's developmental, educational, and vocational accomplishments; overall health; and welfare (Baron, 2007). Although AD/HD is one of the most widely researched disorders, until recently few researchers have considered the possibility of a separate female profile of AD/HD distinct from that of males. This has resulted in limited information about gender differences in this disorder. A comprehensive understanding of gender differences in AD/HD has public health and scientific importance for the advancement of women's health (Biederman & Faraone, 2004). This article reviews the current literature on electroencephalography (EEG) activity in AD/HD populations with a specific focus on females and the lack of sex-specific investigations.

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

The *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev. [*DSM–IV–TR*]; American Psychiatric Association, 2000) conceptualizes AD/HD as a developmentally inappropriate and persistent pattern of inattention and/or hyperactivity-impulsivity. Symptoms of inattention include difficulty sustaining attention, organizing, and completing tasks. Children with inattention appear not to listen or do not hear what is said. Symptoms of hyperactivity include fidgetiness and motor activity in excess of what would be expected given the individual's stage of development. Hyperactive children cannot sit still, talk excessively, and

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appear restless. Children with impulsivity have great difficulty with patience and delaying responses; they frequently interrupt and intrude on others. To be diagnosed with AD/HD, children must display significant impairments in at least two environments and symptoms must be present prior to the age of 7 (*DSM–IV–TR*). There are three types of AD/HD: predominantly Inattentive, predominantly Hyperactive-Impulsive, and the Combined type (*DSM–IV–TR*). Although most AD/HD children will have some symptoms of both inattention and hyperactivity-impulsivity (leading to diagnosis of the Combined type if above thresholds in each dimension), some will have only one or the other symptom pattern. The Inattentive type is characterized by a predominant symptom pattern of inattention, and the Hyperactive-Impulsive type demonstrates predominant symptoms of hyperactivity and impulsivity (*DSM–IV–TR*).

Prevalence studies estimate that AD/HD affects 3 to 7% of school children (*DSM–IV–TR*; Pastor & Reuben, 2008). Approximately 70% of AD/HD children will continue to have AD/HD into adolescence (Barkley, Fischer, Edelbrock, & Smallish, 1990) and 30 to 50% will have the disorder into adulthood (Faraone et al., 2000; Weiss & Hechtman, 1993). Within general population samples, the Inattentive type is most prevalent, with an average estimate of 4.5%, whereas the Combined type is estimated at 1.9% and the Hyperactive-Impulsive type at 1.7% (Gaub & Carlson, 1997). However, within clinic populations, the Combined type is 2 to 4 times more prevalent than the other two types of AD/HD (Wolraich, Hannah, Pinnock, Baumgaertel, & Brown, 1996).

There is a general consensus that more boys than girls have AD/HD. Epidemiological studies show boy-to-girl ratios of 3:1, whereas clinic samples have ratios as high as 9:1 (*DSM–IV–TR*; Arcia & Conners, 1998; Gaub & Carlson, 1997; Hartung & Widiger, 1998; Rutter, Capsi, & Moffitt, 2003). In *DSM-IV* (American Psychiatric Association, 1994) field trials, Lahey et al. (1994) found differences in male-to-female ratios between AD/HD types with 7.3:1 for the AD/HD Combined type, 4:1 for the Hyperactive-Impulsive type, and 2.7:1 for the Inattentive type. Several issues are evident in these ratio discrepancies: (a) There is a large difference between AD/HD females in clinic and community samples, (b) there are ratio differences between males and females, and (c) there is variability between AD/HD types.

Berry, Shaywitz, and Shaywitz (1985) suggested that male-to-female ratio discrepancies could be partially explained by selective referrals, as hyperactive boys are more likely than girls to be referred because they exhibit more disruptive behaviors that are deemed troublesome by adults. Girls tend to have lower levels of disruptive behaviors and higher levels of inattentiveness, social impairment, and internalizing symptoms (Biederman et al., 2002; Carlson, Tamm, & Gaub, 1997; Gaub & Carlson, 1997; Gershon, 2002). Two well-cited reviews (Gaub & Carlson, 1997; Gershon, 2002) found that girls with AD/HD tend to show less physical hyperactive symptoms than AD/HD boys and have fewer externalizing behaviours. In place of excess motor activity, females will more often display other symptoms, such as hypertalkativeness and high emotional reactivity (Quinn, 2005). Females with these behaviors are less likely to disrupt classrooms and/or disturb teachers, so they are more likely to be overlooked for diagnosis and subsequent treatment (Berry et al., 1985; Biederman et al., 2002; DuPaul et al., 2006; Gaub & Carlson, 1997; Gershon, 2002; Quinn, 2005; Sciutto & Eisenberg, 2007). If males are more likely than females to receive attention for their AD/HD behaviors, this could help explain why males are diagnosed with AD/HD more than females.

The *DSM* diagnostic criteria for AD/HD are based primarily on research with school-aged boys (McBurnett et al., 1999) and as such are highly likely to skew the inclusion/exclusion of AD/HD females, especially in those who do not stand out with severe, overt behavioural symptoms (Staller & Faraone, 2006). Ohan and Johnston (2005) found that mothers perceived *DSM–IV* AD/HD symptom criteria as descriptive of boys. An AD/HD diagnosis relies heavily on a parent's view of their child's behavior, and if symptom criteria do not correlate with parents' behavior descriptions, it can result in

misdiagnosis. Waschbusch and King (2006) found that using sex-specific norms for AD/HD evaluations led to identification of a group of impaired girls who otherwise would have been missed. The importance of separate standards for rating AD/HD behaviors has been acknowledged (e.g., Conners Rating Scales have gender-specific norms across age groups), yet the *DSM–IV–TR* does not include this factor in their diagnostic criteria (Rucklidge, 2010). As clinicians rely on *DSM* criteria for a clinical diagnosis, a lack of sex-specific norms may also help explain the large discrepancy in prevalence ratios between clinic and community samples.

Girls are twice as likely as boys to have the Inattentive type of AD/HD, and are more likely to have symptoms of inattention, rather than hyperactivity-impulsivity (Biederman & Faraone, 2004). Berry et al. (1985) found that girls with attention deficit disorder (ADD) without hyperactivity were, on average, older at referral than boys, although they exhibited similar impairments. Berry et al. suggested that the clinical course of ADD is influenced by maturational factors that differ between boys and girls. Although the age-of-onset criterion for AD/HD is 7 years, females appear to have a later age of onset, particularly in those with the Inattentive type (Lahey et al., 1994; McBurnett et al., 1999; Quinn, 2005; Wolraich et al., 1996). As symptoms of inattention are less disruptive and overt than those of hyperactivity-impulsivity, the higher prevalence of inattention in girls, relative to boys, could help explain the gender ratio discrepancy between the AD/HD types (Berry et al., 1985; Biederman & Faraone, 2004). Some girls with AD/HD may go undiagnosed due to less disruptive hyperactive and impulsive symptoms than boys, a greater likelihood of having inattentive symptoms, or a later age of symptom onset.

Females with AD/HD are at greater risk of anxiety, depression, self-blame, shame, and low self-esteem than AD/HD males (Kato, Nicholas, Kerivan, & Huffman, 2001; Quinn, 2005; Quinn & Nadeau, 2002). Adolescent females with AD/HD are also at greater risk of smoking, substance abuse, sexual promiscuity, unplanned pregnancy, peer rejection, and social isolation (Berry et al., 1985; Biederman & Faraone, 2004; Quinn, 2005; Quinn & Nadeau, 2002). Oestrogen, a female hormone, has been found to influence the severity of AD/HD symptoms with many females reporting that they become emotionally hyperreactive during certain stages of their menstrual cycle, intensifying their AD/HD symptoms (Quinn & Nadeau, 2002; Ratey, Miller, & Nadeau, 1995).

Differences in the presentation, identification, and diagnosis of AD/HD between genders highlight important issues. Arnold (1996) expressed the importance of distinguishing normal sex differences from those that are attributable to AD/HD pathology. Females normally differ from males; therefore, normative sex differences need to be clearly distinguished from AD/HD-related sex differences (Arnold, 1996). As the Inattentive type is more common in females and may be associated with a later onset of symptoms, there is an emphasis on the need to clarify if the life course of the disorder differs between genders (Arnold, 1996). Yet, despite AD/HD being one of the most researched psychiatric conditions, females continue to remain underrepresented in the literature. Most AD/HD studies have either excluded females to satisfy the desire for homogeneous groups or used disproportionate male/female ratios to reflect the comparatively small number of clinically identified females (Berry et al., 1985).

EEG ACTIVITY IN CHILDREN

EEG measures brain function through the analysis of electrical activity at the scalp that is generated by underlying brain structures (Becker & Holtmann, 2006). Resting EEG activity provides useful information on the background cortical state, indexing substrates of behaviour and cognition (Barry, Clarke, & Johnstone, 2003). Spectral analyses of EEG activity provide amplitude and power measures in the classical frequency bands: delta (<4 Hz), theta (4–7 Hz), alpha (8–12 Hz), beta (13–25 Hz), and gamma (30–80 Hz) and reflect information about the excitability of neural networks and their maturation (Cragg et al., 2011; Nunez & Srinivasan, 2006; Taylor & Baldeweg, 2002). Alpha wave

activity is associated with a relaxed conscious state and will be disrupted with increased mental load; beta wave activity is evident during cognitive processing; and the slower waveforms, delta and theta, are more commonly associated with immaturity and abnormalities (Andreassi, 2000). The gamma frequency band is considered to range from 30 to 80 Hz and has been linked with cognitive functioning (Engel, Fries, & Singer, 2001; Fell, Fernandez, Klaver, Elger, & Fries, 2003; Herrmann, Frund, & Lenz, 2010).

Cortical development is reflected in changes to childhood EEG activity. Generally, there is a predominance of slow-wave delta and theta in infancy, thought to reflect brain immaturity; and this activity increases in frequency with age (Benninger, Matthis, & Scheffner, 1984; Hudspeth & Pribram, 1992; Tye, McLoughlin, Kuntsi, & Asherson, 2011). Maturational EEG studies generally report that with increasing age, low-frequency band (delta and theta) activity decreases and higher frequency band (alpha and beta) activity increases (Cragg et al., 2011; Gasser, Jennen-Steinmetz, Sroka, Verleger, & Mocks, 1988; Gasser, Verleger, Bacher, & Sroka, 1988; Gmehlin et al., 2011; John et al., 1980; Matousek & Petersen, 1973; Matsuura et al., 1985). These changes are believed to begin over posterior brain regions (Clarke, Barry, McCarthy, & Selikowitz, 2001a; Gasser, Jennen-Steinmetz, et al., 1988), followed by the central and frontal regions (Gasser, Jennen-Steinmetz, et al., 1988; Katada, Ozaki, Suzuki, & Suhara, 1981; Matousek & Petersen, 1973). Like alpha and beta, gamma power increases with age, particularly over the frontal regions (Takano & Ogawa, 1998).

Clarke et al. (2001a) used easily interpretable estimates of absolute and relative power to examine developmental EEG changes in 80 healthy children (40 boys and 40 girls, aged 8–12 years). Children showed decreasing absolute delta, relative delta and theta, and increasing relative alpha and beta with increasing age, consistent with the general consensus mentioned above. Clarke et al. (2001a) found that the decreases in delta and theta activity and increases in alpha activity occurred more quickly in the posterior regions than the frontal regions, consistent with Katada et al. (1981); Matousek and Petersen (1973); and Somsen, van't Klooster, van der Molen, van Leeuwen, and Licht (1997). Clarke et al. (2001a) also noted differences in the rates of maturation between the midline sites (Fz, Cz, and Pz) and the two hemispheres. Absolute delta activity and the theta/beta ratio were greater at the midline in younger children, before becoming more equipotential with increasing age, suggesting that EEG maturation occurs faster at the midline than in the hemispheres.

John et al. (1980) presented linear functions to predict the EEG composition for log-transformed relative power as a function of age—research by Ahn, Baird, and Kaye (1980); Alvarez, Valdes, and Pascual (1987); and Clarke et al. (2001a) supported these equations. Others have suggested that developmental changes are due to periodic growth spurts around 6, 10, and 14 years of age (Epstein, 1980; Thatcher, 1991). Although maturational changes are believed to reflect underlying changes in cortico-cortical and cortico-thalamic networks (Lopes da Silva, 1991; Steriade, Gloor, Llinas, Lopes da Silva, & Mesulam, 1990), the exact processes remain unclear. There has been speculation that they may reflect developmental changes in gray and white matter (Whitford et al., 2007) or increases in myelination or axon size (Segalowitz, Santesso, & Jetha, 2010); however, these are not yet fully understood.

Studies of sex differences in the EEG maturation of healthy children have had mixed results. Some report no significant differences (Cohn, Kircher, Emmerson, & Dustman, 1985; Gasser, Jennen-Steinmetz, et al., 1988; Matousek & Petersen, 1973), others suggest that EEG power matures earlier in females (Petersen & Eeg-Olofosson, 1971), and some find that females display an EEG maturational lag (Harmony, Marosi, Diaz de Leon, Becker, & Fernández, 1990). The best supported finding is that maturation during childhood, reflected by increased relative alpha and a lower delta/theta ratio, occurs earlier in males than females (Clarke et al., 2001a; Harmony et al., 1990; Matthis, Scheffner & Benninger, 1980), but females have a greater rate of

maturation (Benninger et al., 1984; Clarke et al., 2001a; Harmony et al., 1990).

Clarke et al. (2001a) found that healthy boys had more absolute theta, relative theta, and relative alpha power activity than healthy girls. With increasing age, absolute delta, theta, and alpha power decreased in girls but remained relatively constant in boys. Barry and Clarke (2009) provided a summary of their EEG research to date and stated that girls (8–12 years) had a more immature EEG profile than boys, evidenced by greater absolute and relative posterior delta, globally elevated absolute and relative theta, and reduced absolute and relative posterior alpha activity. The girls' relative immaturity in the absolute delta and theta bands decreased with increasing age, but posterior absolute alpha continued to show immaturities compared with aged-matched boys. Cragg et al. (2011) mapped EEG maturation in 10-year-olds that were tested three times over a 3-year period. Although not as robust as previous studies, their results supported the findings of earlier maturation in males and a greater rate of development in females. These studies indicate that girls have maturationally delayed EEG activity compared with boys, but this lag disappears by adolescence, suggesting that although male EEG activity matures earlier, females catch-up in adolescence.

EEG ACTIVITY IN AD/HD

The electroencephalogram has an established history within AD/HD—Solomon, Jasper, and Bradley (1937) and Solomon, Bradley, and Jasper (1938) suggested that an underlying cerebral component could explain the high incidence of EEG abnormalities within ''Childhood Behaviour Disorder.'' Favorable EEG and behavioral responses to commonly prescribed medications of the time (e.g., Dilantin and Phenobarbital) reinforced this idea (Cutts & Jasper, 1939; Lindsley & Cutts, 1940; Lindsley & Henry, 1942; Walker & Kirkpatrick, 1947). A clear understanding of EEG maturation and activity in males and females can help divide sex differences that are normally occurring from those that are related to AD/HD and help determine if the life course of AD/HD is different between genders.

Although this is a female-focused review, it is important to describe the current EEG profiles of AD/HD children. Note that these profiles are based on combined groups of males and females (or males alone), and although they should be interpreted with caution, they provide a framework for describing EEG activity within AD/HD. Children (7–13 years) with AD/HD typically show globally elevated slow wave activity (absolute delta and theta) and globally reduced absolute alpha and beta activity compared to children without AD/HD (for overall reviews, see Barry & Clarke, 2009; Barry, Clarke, et al., 2003). These children also have elevated absolute frontal theta activity (Chabot & Serfontein, 1996; Clarke, Barry, Bond, McCarthy, & Selikowitz, 2002; Clarke, Barry, Dupuy, et al., 2011; Lansbergen, Arns, van Dongen-Boomsma, Spronk, & Buitleaar, 2011; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992) and increased posterior absolute delta activity (Clarke, Barry, Dupuy, 2011; Clarke, Barry, McCarthy, & Selikowitz, 2001c, 2001d). Within relative power, AD/HD children commonly show globally elevated relative theta and globally reduced relative alpha and beta activity (Barry & Clarke, 2009; Barry, Clarke, Johnstone, McCarthy, & Selikowitz, 2009; Chabot & Serfontein, 1996; Clarke, Barry, Dupuy, et al., 2011; Clarke et al., 2001b, 2001d, 2011). A larger theta/beta ratio has consistently been found in AD/HD children compared with healthy controls (Barry et al., 2009; Clarke, Barry, Dupuy, et al., 2011; Clarke et al., 2001b, 2001d, 2011; Janzen, Graap, Stephanson, Marshall, & Fitzsimmons, 1995; Lansbergen et al., 2011; Lubar, 1991; Monastra, Lubar, & Linden, 2001; Monastra et al., 1999).

Adolescents with AD/HD continue to have significant impairments in attention, impulsivity and overactivity (Barkley, 1991). Although only a small number of studies have examined EEG activity in AD/HD adolescents (14–17 years), relatively consistent findings have emerged. Generally, adolescents with AD/HD have

elevated absolute theta and absolute alpha activity with a larger theta/beta ratio compared with aged-matched controls (Bresnahan, Anderson, & Barry, 1999; Hermens, Kohn, Clarke, Gordon, & Williams, 2005; Hobbs, Clarke, Barry, McCarthy, & Selikowitz, 2007; Lazzaro et al., 1999; Lazzaro et al., 1998). AD/HD adolescents have also been found to have higher absolute and relative delta compared with aged-matched controls (Bresnahan et al., 1999; Hobbs et al., 2007). Lazzaro et al. (1999) found that AD/HD adolescents had globally elevated absolute and relative alpha1 (8–9 Hz) activity, compared with controls. Although there is variability amongst reports of beta power, most have found reduced relative beta activity in AD/HD adolescents compared with adolescents without AD/HD (Bresnahan et al., 1999; Hobbs et al., 2007; Lazzaro et al., 1999; Lazzaro et al., 1998). A robust continuation of abnormally elevated theta activity in AD/HD adolescents strengthens the notion of AD/HD persisting beyond childhood.

Adults with AD/HD (18 years +) show a similar profile to adolescents. However, caution is urged during interpretation as conditions vary between studies, most notably eyes-open versus eyes-closed baseline EEG conditions; it is well accepted that differences between these two conditions lead to differences in power levels and topography (Barry et al., 2007). Generally, adults with AD/HD have greater absolute and relative delta, absolute and relative theta activity, and a larger theta/beta ratio compared with controls (Bresnahan et al., 1999; Bresnahan & Barry, 2002; Bresnahan, Barry, Clarke, & Johnstone, 2006; Clarke et al., 2008; Hermens et al., 2004). In addition, Koehler et al. (2009) found that adults with AD/HD had greater absolute alpha activity and increased absolute posterior theta power than controls. As with AD/HD adolescents, the beta band varies across studies; some have found AD/HD adults have increased absolute beta activity (Bresnahan & Barry, 2002), and others report reductions in absolute beta activity (Clarke et al., 2008; Hermens et al., 2004), whereas Bresnahan et al. (1999) found absolute beta (in fronto-central regions) to be normal in AD/HD adults during an eyes-open condition.

In relation to AD/HD types, it has generally been reported that the Combined type of AD/HD is associated with greater EEG abnormalities than the Inattentive type of AD/HD, particularly in absolute theta and alpha bands (Barry & Clarke, 2009; Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 1998, 2001b, 2001d). Chabot and Serfontein (1996) investigated EEG differences between ADD children with and without hyperactivity and concluded that the differences between the two clinical groups were in the degree, rather than type, of abnormality. Mann et al. (1992) found that boys with ADD without hyperactivity had increased frontal absolute and relative theta power and a greater decrease in posterior and temporal absolute beta power during sustained attention tasks compared with controls.

Clarke et al. (1998) investigated EEG differences between the Combined and Inattentive types of AD/HD and controls in a mixed group of 60 children (48 boys, 12 girls). The combined type group had greater relative posterior delta and global theta activity and less global relative alpha activity than the inattentive type group. Clarke et al. (2001d) replicated these results with 120 children (96 boys, 24 girls) and also found that the combined group had a greater theta/beta ratio and a reduced theta/alpha ratio than the inattentive group. Clarke et al. (2001b) used equal numbers (40 boys, 40 girls) to compare AD/HD type differences in resting EEG activity. The combined group had greater absolute and relative theta, higher theta/beta and theta/alpha ratios, and less relative alpha than the inattentive group. The widespread significant differences between AD/HD types led Clarke et al. (2001d) to suggest that the two AD/HD types have separate neuroanatomical anomalies.

Although EEG abnormalities have been consistently reported in AD/HD populations, the underlying nature and explanation of these abnormalities remain unclear. However, the use of EEGs has helped shape two models that attempt to explain AD/HD abnormalities; the

first suggests that AD/HD is the result of a maturational lag in central nervous system (CNS) functioning, and the second proposes that a developmental deviation results in AD/HD. Both models have received research attention yet fail to fully explain the underlying nature of AD/HD. It is highly unlikely that one single model can capture and explain the disorder in its totality; nonetheless, these models provide relevant information about the nature of EEG abnormalities in AD/HD.

The maturational lag model suggests that the AD/HD EEG abnormalities are due to a lag, or delay, in cortical development (Kinsbourne, 1973; P. Shaw et al., 2007). According to Kinsbourne (1973), AD/HD children are underdeveloped for their age, and this is reflected in their cortical activity, which is similar to that of a healthy younger child. Support for this model is found in AD/HD symptom criteria, which refer to AD/HD behaviors that are inappropriate for the developmental stage, meaning that they may be found in younger normal children. Also, AD/HD symptoms improve with age—up to 50% of children grow out of AD/HD in adolescence and adulthood (*DSM–IV–TR*; Biederman et al., 1994; Faraone et al., 2000). This suggests that children with AD/HD have a developmental delay compared to normally developing children, and this delay remits by adulthood (Hill & Schoener, 1996).

However, the increasing recognition of AD/HD in adolescents and adults suggests that children do not always grow out of AD/HD. It has been estimated that about 30 to 50% will continue to have AD/HD into adulthood (Faraone et al., 2000; Weiss & Hechtman, 1993). A criticism of the maturational lag model is that it cannot account for adults who continue to have AD/HD. Bresnahan et al. (1999) examined age-related quantitative EEG changes in AD/HD participants aged 6 to 42 years. The results showed that the AD/HD adults had increased absolute delta and theta activity compared to normal controls. Bresnahan and Barry (2002) found that 50 adults with AD/HD had elevated absolute delta, theta, alpha, beta, and total power, and relative theta activity compared to healthy controls. Clarke et al. (2008) also found adult AD/HD EEG profiles similar to AD/HD children in that they also showed increased relative theta and reduced relative beta at midline sites compared to aged-matched controls. These studies have found EEG abnormalities in adults with AD/HD, most notably in absolute delta and theta and relative theta activity, arguing against the maturational lag model.

Lubar (1991) linked low skin conductance level (SCL; an indication of CNS arousal) in hyperactive children (Satterfield & Dawson, 1971) with an EEG study by Jasper, Solomon, and Bradley (1938), who suggested that a shift from a resting to active state resulted in an EEG power shift from dominant theta and alpha activity to dominant beta activity. Lubar (1991) hypothesized that AD/HD children would have increased theta power with a reciprocal reduction in beta activity and that this theta/beta ratio represented CNS hypoarousal. Mann et al. (1992) supported this notion, and the theta/beta ratio became a popular marker for AD/HD. Satterfield, Cantwell, and Satterfield (1974) found that hyperactive children had low CNS arousal levels (measured by SCL) and noted a negative correlation between CNS arousal levels and the severity of the hyperactive child's behavioral disturbances: the lower the SCL, the greater the severity of behavioral disturbances. Satterfield et al. (1974) went on to discover that stimulant medications improved behavior and CNS arousal levels. Increased theta with reciprocal reduced beta power, believed to reflect cortical hypoarousal, has been found consistently across AD/HD groups (Barry & Clarke, 2009; Barry et al., 2009; Clarke et al., 2001d; Clarke, Barry, McCarthy, et al., 2011; Lansbergen et al., 2011; Lazzaro et al., 1999; Lazzaro et al., 1998).

However, Barry et al. (2004) examined the link between the theta/beta ratio and SCL activity in 24 healthy boys. There were no significant differences in either the theta or beta band as a function of arousal level, and there was no difference in the theta/beta ratio between high- and low-aroused children (measured via SCL). Instead, elevated SCL was significantly associated with decreased alpha activity,

particularly in posterior and hemispheric regions (Barry et al., 2004). Barry et al. (2004) proposed that the theta/beta ratio may reflect differences in functionality of the basis of attentional processing. Barry et al. (2009) reinvestigated the link between arousal and the theta/beta ratio in 30 AD/HD boys. The AD/HD boys had lower SCL, elevated absolute theta, and a larger theta/beta ratio with reduced absolute alpha and beta activity compared with controls. Although these results are consistent with previous AD/HD reports, they did not support the notion that theta/beta ratio reflects CNS arousal, as there was no significant correlation between SCL and the theta/beta ratio. Instead, decreased relative alpha was correlated with elevated SCL in both the healthy and AD/HD groups. Barry and colleagues (Barry et al., 2009; Barry et al., 2004) proposed that the theta/beta ratio represents a substrate of activation, particularly in cognitive/attention tasks. Within AD/HD, this ratio signifies an impaired capacity for attentional tasks, a processing deficit, not an arousal deficit (Barry et al., 2009). Labeling EEG patterns in arousal terms needs to be reconsidered, as although the theta/beta ratio is a consistent marker for AD/HD, it does not indicate CNS hypoarousal (Barry et al., 2009).

Clarke, Barry, McCarthy, et al. (2011) correlated EEG abnormalities with core symptoms of AD/HD. In the 60 boys with AD/HD that participated, Clarke, Barry, McCarthy, et al. (2011) correlated three AD/HD subscales of the Conners Rating Scales with anomalous EEG activity. Increased frontal absolute theta activity was significantly correlated with the Inattentive and Total subscales. As elevated frontal theta activity is the most commonly reported EEG abnormality within AD/HD, the fact that it correlated with inattention helps explain why it is found in both the Inattentive and Combined AD/HD types (Clarke, Barry, McCarthy, et al., 2011). The theta/beta ratio correlated with the Hyperactive-Impulsive and Total subscales. This is interesting because Clarke et al. (2001c) had previously found that the theta/beta ratio matured at a faster rate in children with the Combined type of AD/HD and became similar to levels found in children with the Inattentive type of AD/HD by age 12. This was interpreted as indicating that hyperactivity was associated with the theta/beta ratio and the reduction in the degree of abnormality was related to a reduction in hyperactivity (Clarke et al., 2001c).

The use of EEG studies has also helped improve understanding and use of AD/HD medications. Stimulant medications (namely, dexamphetamine and methylphenidate) are widely used in the treatment of AD/HD, with clinically significant benefits found in approximately 80% of treated patients (leaving 20% with nonfavorable responses; Swanson, McBurnett, Wigal, & Pfiffner, 1993; Wilens & Biederman, 1992). Psychostimulants are thought to increase the arousal level of the CNS by stimulating the release, and inhibiting the reuptake, of the dopamine and noradrenalin neurotransmitters (Biederman & Spencer, 1999; Durston, 2003; Lawrence et al., 2005). Early EEG studies on AD/HD medications had problems clearly defining global EEG changes due to medication (Chabot, Orgill, Crawford, Harris, & Serfontein, 1999; Swartwood et al., 1998), and it was speculated that only those who responded positively to medications would show EEG normalization (Loo, Teale, & Reite, 1999).

Generally, in AD/HD children who respond well to psychostimulants, the medications appear to normalize EEG characteristics by decreasing abnormal absolute theta and increasing absolute beta activity levels (Clarke, Barry, Bond, et al., 2002; Clarke, Barry, McCarthy, Selikowitz, & Croft, 2002; 2003b; Loo et al., 1999; Lubar, White, Swartwood, & Swartwood, 1999). Rowe, Robinson, and Gordon (2005) found a reduction of relative theta and alpha power with medication use in a small group of AD/HD male adolescents (12–17 years), trending toward normalization. In a group of 50 AD/HD adults (equal numbers of male and female), medication had similar effects to that found in children and adolescents. Reductions were noted in absolute delta, absolute and relative theta, and total power, with the EEG profiles of AD/HD adults trending toward normalization.

Attention has turned to examining EEG differences between AD/HD patients who respond positively and those who responded negatively to medications. Work by Satterfield and colleagues (Satterfield, Cantwell, Saul, Lesser, & Podsin, 1972, 1973; Satterfield, Saul, Lesser, & Cantwell, 1973) found that those who responded well to stimulant medications had greater EEG abnormalities, particularly in increased absolute delta and theta, compared to poor responders. Satterfield and Cantwell (1974) believed that these results indicated that good medication responders were cortically hypoaroused. Clarke et al. (2002b) supported the notion that good medication responders have greater EEG abnormalities than poor responders; good responders to methylphenidate had higher total power and a larger theta/beta ratio than poor responders. Clarke, Barry, McCarthy, and Selikowitz (2002) also investigated EEG differences between good and poor responders to dexamphetamine, as there is variability among responses to stimulant medications (Chabot & Serfontein, 1996). Good responders to dexamphetamine also showed greater EEG abnormalities compared to poor responders, namely, reduced relative alpha and beta activity and greater posterior total power. Clarke, Barry, McCarthy, and Selikowitz (2002) concluded that good responders to popular psychostimulants appear to have more abnormal EEG profiles than poor responders.

It is important to note that all the EEG literature just reviewed was based on either disproportionate samples of males and females or excluded females altogether. Although it is commonly accepted that males and females are different, conclusions about AD/HD in females continue to be drawn from this male-dominated research. Although there is increasing support for gender specific AD/HD profiles, the inclusion of few females, referral bias, and inappropriate diagnostic criteria hamper efforts to make genuine predictions on how males and females differ, emphasising that sex effects and differences should be part of the standard comparison in studies (Rucklidge, 2010).

EEG ACTIVITY IN FEMALES WITH AD/HD

The literature is scant and mixed on the EEG activity of females with AD/HD. The majority of research in this area included either males alone (e.g., Barry et al., 2009; Clarke, Barry, Bond, et al., 2002; Lansbergen et al., 2011; Lazzaro et al., 1998) or combined groups of males and females (i.e., Chabot & Serfontein, 1996; Clarke et al., 1998; Clarke et al., 2001d; Clarke et al., 2008; Hale et al., 2010). Significant behavioral differences between AD/HD males and females suggest that research that relies heavily on males cannot provide an adequate understanding of AD/HD in females.

Baving, Laucht, and Schmidt (1999) investigated frontal activation patterns in 117 children with AD/HD (aged $4^1/_2$ years and 8 years). Baving et al. found that 15 AD/HD girls (combined age groups) had significantly greater *left* frontal alpha power activation than aged-matched control girls, whereas AD/HD boys had significantly greater *right* frontal alpha power activation than control boys. Baving et al. suggested that the AD/HD girls' enhanced left frontal alpha power corresponds to a left frontal deficit. Although the opposite direction of asymmetry in alpha power activation between AD/HD boys and girls was noted, Baving et al. could not link cognitive or behavioral effects to this observed EEG gender difference. It was suggested that gender-specific lines of development result in differing frontal alpha activation patterns between males and females, but this was not further investigated. Ultimately Baving et al. stressed the importance of gender-specific analyses in AD/HD.

Mentioned earlier, Clarke et al. (2001b) investigated age- and sex-related effects on the EEGs in equal-sized groups of 40 boys and 40 girls with AD/HD. The AD/HD girls (pooled AD/HD types) had greater absolute alpha, greater relative delta and theta activity, more total power, and reduced absolute beta activity than control girls. Between the AD/HD types, the girl combined group had larger theta/alpha and theta/beta ratios than the girl inattentive group, and those of both clinical groups were

significantly greater than girl controls. However, the statistical design did not include a separate, explicit analysis of the EEG activity within AD/HD girls.

Clarke, Barry, McCarthy, Selikowitz, Clarke, et al. (2003) addressed the prior issue and recorded eyes-closed resting EEGs from 100 girls (aged 8–12 years) with AD/HD. The AD/HD girls had greater total power; greater relative theta; and less relative delta, alpha, and beta than age-matched controls. Clarke, Barry, McCarthy, Selikowitz, Clarke, et al. noted the novelty of a *global* deficiency of relative delta activity in AD/HD girls, whereas *excess posterior* relative delta activity had been more commonly found in male and mixed AD/HD groups (Clarke et al., 1998; Clarke et al., 2001b, 2001d; Matousek, Rasmussen, & Gilberg, 1984). Clarke, Barry, McCarthy, Selikowitz, Clarke, et al. (2003) followed on from two male cluster analysis studies (Clarke et al., 2001c; Clarke, et al., 2001d) that investigated the homogeneity of EEG activity in boys with the Combined and Inattentive types of AD/HD. Two distinct EEG profiles emerged, the first characterized by elevated total power and relative theta activity, a larger theta/beta ratio, and a reduction in relative delta and beta activity (thought to represent cortical hypoarousal at the time of publication), and the second characterized by increased relative delta and relative theta and reduced relative alpha and relative beta activity (thought to represent maturational lag). A third cluster, characterised by excess relative beta activity, believed to represent cortical hyperarousal, was found in boys with Combined type AD/HD (Clarke et al., 2001c). The same cluster analysis was done on this group of AD/HD girls (Clarke, Barry, McCarthy, Selikowitz, Clarke, et al., 2003). A two-cluster model emerged, with the majority of AD/HD girls (96%) showing greater relative theta and less relative delta and beta activity. The second cluster, only 4% of the total group, had elevated fronto-central total power; increased relative frontal theta activity; and reduced relative delta, alpha, and beta. Clarke, Barry, McCarthy, Selikowitz, Clarke, et al. believed that the two clusters may represent differing levels of cortical hypoarousal and were not necessarily qualitatively different. The cluster profiles of the female groups were highly homogenous, with the majority fitting into one cluster type, whereas the AD/HD boys were more evenly spread.

A study by Dupuy, Clarke, Barry, McCarthy, and Selikowitz (2011) followed directly from Clarke et al. (2001c) and explicitly analyzed EEG activity between the Combined and Inattentive types of AD/HD within three groups of 30 girls (8–12 years). The AD/HD girls (pooled types) had elevated total power, absolute delta and theta, and reduced relative beta activity compared with controls. Between the clinical groups, the Combined type group had elevated absolute right theta power, greater midline-posterior absolute beta and relative alpha activity, reduced right relative delta activity, reduced left relative theta activity, and reduced relative beta activity within central regions of both hemispheres compared with the Inattentive type group. Although there were topographic differences, there were no significant global differences between the two AD/HD types. Global differences have been found previously in AD/HD males (Clarke et al., 1998; Clarke et al., 2001b, 2001d) and these were so significant that the authors even suggested that the two AD/HD types could be neuroanatomically different. Dupuy et al. (2011) referred to the homogenous nature of EEG activity found in girls with AD/HD (Clarke, Barry, McCarthy, Selikowitz, Clarke, et al., 2003) and suggested that this could explain the discrepancy apparent between the EEG profiles of boys and girls with AD/HD. Dupuy et al. suggested that sex biases in identification, referral, and diagnosis of AD/HD could explain why global differences between clinically selected AD/HD types are so prominent in boys yet elusive in girls.

Girls with AD/HD have shown reduced relative beta activity with a reciprocal increase in relative theta activity compared with healthy girl controls (Clarke et al., 2001b; Clarke, Barry, McCarthy, Selikowitz, Clarke, et al., 2003; Dupuy et al., 2011). This elevated theta/beta ratio, originally thought to be ''cortical

hypoarousal'' and now believed to represent underlying deficits in attentional processing (Barry et al., 2009), is a consistent profile among AD/HD populations, both in males and females. However, unlike boys, AD/HD girls have commonly shown abnormalities in absolute and relative delta and total power, though further replication across populations would be beneficial to support these results.

Whereas it was once assumed that children outgrew AD/HD by puberty, it is now overwhelmingly apparent that many continue to experience AD/HD in adolescence and adulthood. The estimated adult prevalence of AD/HD ranges from 1.2% to 3.2%, even up to 4.4% (Faraone, Biederman, & Mick, 2006; Kessler et al., 2006), depending on selection criteria. An important limitation of the previous adolescent and adult EEG studies is lack of female inclusion; most studies excluded females (Hobbs et al., 2007; Lazzaro et al., 1999; Lazzaro et al., 1998), whereas others had mixed groups (Bresnahan et al., 1999; Hermens et al., 2005). Only one study to date, Hermens et al. (2005), included a statistical analysis of EEG activity in female adolescents; twenty-two AD/HD female adolescents (aged 11–17 years) were part of a larger group of 70 AD/HD adolescents that had an eyes-closed, resting EEG recorded simultaneously with electrodermal activity. Adolescent AD/HD females had elevated midline absolute theta power, decreased posterior absolute beta power with a slight increase of frontal absolute beta, and a larger theta/beta ratio compared with female controls. Hermens et al. (2005) also investigated sex differences and found that although AD/HD male adolescents had greater global absolute theta activity, AD/HD female adolescents had a more specific frontal elevation of absolute theta activity. As reported in adults (Hermens et al., 2004), Hermens et al. (2005) replicated results that AD/HD male adolescents (but not female) had elevated global EEG theta activity, whereas AD/HD female adolescents (but not male) had decreased SCL. Hermens et al. (2005) suggested that differing and distinct core neurobiological deficits underlie the developmental course of AD/HD between males and females. It is important to note that, to date, there have been no published AD/HD studies on EEG activity of female adolescents or adults alone.

Clarke, Barry, McCarthy, Selikowitz, and Johnstone (2007) investigated medication effects on the EEGs of 20 girls with AD/HD (aged 7–12 years). Eyes-closed EEG activity was recorded in girls with AD/HD both on and off medication (either 10 mg of methylphenidate or 5 mg of dexamphetamine). Girls with AD/HD (no medication) had greater total power, absolute delta and theta, and relative theta and reduced relative delta and beta activity compared with controls, similar to previous female EEG results. The EEGs from the medicated AD/HD girls showed a significant reduction in relative theta activity, with no difference from controls remaining in this frequency band, thought to reflect a complete normalisation of relative theta activity (due to medication). Although previously published medication studies found theta activity trended toward normal levels (often not significant; Clarke, Barry, McCarthy, & Selikowitz, 2002; Clarke, Barry, McCarthy, Selikowitz, Brown, et al., 2003), this was the first to find a *complete* normalization of relative theta. Clarke et al. (2007) considered that the EEGs of AD/HD girls may not be as abnormal as those of AD/HD boys, so EEG changes due to medication show greater normalization because they were less aberrant to start with. A second possibility was that the dominant homogenous EEG profile found in AD/HD girls responds better to medication than other EEG profiles (Clarke et al., 2007).

Electrophysiological parameters are ideal for studying higher level cognitive processes, such as attention, response selection, and decision making, which are paramount in the study of AD/HD (McLoughlin, Clarke, Barry, McCarthy, & Selikowitz, 2005). Other aspects of EEG that are often utilized in AD/HD research are event-related potentials (ERPs) and EEG coherence. ERPs result from ongoing EEG that is averaged and time locked to stimulus or response events (Becker & Holtmann, 2006) and are believed to reflect discharges

from large groups of neurons, linked to specific aspects of sensory and cognitive processing (Taylor & Baldeweg, 2002). Although not discussed here, ERPs provide detailed analyses of timing and location of specific aspects cortical information processing (Barry & Clarke, 2009) and have been widely used in AD/HD studies (for a review, see Barry, Johnstone, & Clarke, 2003). Coherence reflects time-locked joint EEG activity in different cortical regions within a particular frequency band (J. Shaw, 1981). Coherence offers valuable information on connectivity, and AD/HD populations have shown marked deviations in collaborative brain activity between different cortical regions (for a review, see Barry & Clarke, 2009). Although ERP and EEG coherence are beyond the scope of this review, they offer highly valuable information on the cortical processing and connectivity within AD/HD for both males and females.

CONCLUSIONS AND FUTURE DIRECTIONS

The majority of AD/HD literature has relied heavily upon school-aged boys (Arnold, 1996) and is frequently applied to girls and women. Mounting evidence of gender disparities in the prevalence, presentation, and detection of AD/HD argues that this male-based literature is not necessarily appropriate for females. It is generally accepted that males and females normally differ, so it is a small step to agree that males and females are also likely differ importantly on aspects of AD/HD.

Electroencephalography is a reliable measure of electro-cortical activity and has reputable history in the study of AD/HD. Although it is accepted that EEG maturation is influenced by gender differences, many AD/HD investigations continue to pool males and females together. The small number of EEG studies of females with AD/HD presented in this review stresses the lack of knowledge on how electrocortical activity is influenced by AD/HD in girls and women. In addition, to date, there are no studies that have investigated EEG activity in adult women with AD/HD. As AD/HD often persists into adulthood, it is important to know if the life course of AD/HD differs between males and females. Although the literature is sparse on adult AD/HD, a few male-based EEG studies have been published in this area, but further research is needed.

The three types of AD/HD, predominantly Inattentive, Hyperactive-Impulsive, and Combined type, represent different manifestations of the disorder, based on prevailing symptoms. Significant EEG differences between the prevalent Inattentive and Combined types have been reported; in boys, global differences were noted in absolute theta, alpha bands, theta/beta, and theta/alpha ratios. However, girls did not produce these apparent global differences. Clinical populations of AD/HD girls have been found to have rather homogenous EEG profiles (Clarke, Barry, McCarthy, Selikowitz, Clarke, et al., 2003; Dupuy et al., 2011) (whereas AD/HD boys appear to be more heterogeneous). The majority of the reviewed studies relied on clinical samples; it would be worthwhile to invest in EEG research within community populations of AD/HD females to determine if reported EEG abnormalities are not merely limited to clinical populations. An insightful comment that evolved from a conference on AD/HD sex differences is that the awareness of female manifestations of AD/HD and sex differences will not only be useful to females but also contribute to a deeper understanding of the disorder in general (Arnold, 1996). We need to build our research around that concept.

REFERENCES

Ahn, H., Baird, H., & Kaye, H. (1980). Developmental equations reflect brain dysfunction. *Science*, *210*, 1259–1262.

Alvarez, A., Valdes, P., & Pascual, R. (1987). EEG developmental equations confirmed for Cuban schoolchildren. *Electroencephalography and Clinical Neurophysiology*, *67*, 330–332.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental*

disorders (4th ed., text rev.). Washington, DC: Author.

Andreassi, J. L. (2000). *Psychophysiology: Human behavior and physiological response* (4th ed.). Mahwah, NJ: Erlbaum.

Arcia, E., & Conners, C. K. (1998). Gender differences in AD/HD? *Journal of Developmental and Behavioral Pediatrics*, *19*, 77–83.

Arnold, L. (1996). Sex differences in ADHD: Conference summary. *Journal of Abnormal Child Psychology*, *24*, 555–569.

Barkley, R. A. (1991). Adolescents with ADHD: Patterns of behavioral adjustment, academic functioning, and treatment utilization. *Journal of the American Academy of Child and Adolescent Psychiatry*, *30*, 752–761.

Barkley, R. A., Fischer, M., Edelbrock, C. S., & Smallish, L. (1990). The adolescent outcome of hyperactive children diagnosed by research criteria, I: An 8-year prospective follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *29*, 546–557.

Baron, I. S. (2007). Attention-Deficit/Hyperactivity Disorder: New challenges for definition, diagnosis, and treatment. *Neuropsychological Reviews*, *17*, 1–3.

Barry, R. J., & Clarke, A. R. (2009). Spontaneous EEG oscillations in children, adolescents, and adults: Typical development, and pathological aspects in relation to AD/HD. *Journal of Psychophysiology*, *23*, 157–173.

Barry, R. J., Clarke, A. R., & Johnstone, S. J. (2003). A review of electrophysiology in attention-deficit/hyperactivity disorder: I. Qualitative and quantitative electroencephalography. *Clinical Neurophysiology*, *114*, 171–183.

Barry, R., Clarke, A., Johnstone, S., Magee, C., & Rushby, J. (2007). EEG differences between eyes-closed and eyes-open resting conditions. *Clinical Neurophysiology*, *118*, 2765–2773.

Barry, R. J., Clarke, A. R., Johnstone, S. J., McCarthy, R., & Selikowitz, M. (2009). Electroencephalogram Θ/β ratio and arousal in Attention-Deficit/Hyperactivity Disorder: Evidence of independent processes. *Biological Psychiatry*, *66*, 398–401.

Barry, R. J., Clarke, A. R., McCarthy, R., Selikowitz, M., Rushby, J. A., & Ploskova, E. (2004). EEG differences in children as a function of resting-state arousal level. *Clinical Neurophysiology*, *115*, 402–408.

Barry, R. J., Johnstone, S. J., & Clarke, A. R. (2003). A review of electrophysiology in attention-deficit/hyperactivity disorder: II. Even-related potentials. *Clinical Neurophysiology*, *114*, 184–198.

Baving, L., Laught, M., & Schmidt, M. H. (1999). Atypical frontal brain activation in ADHD: Preschool and elementary school boys and girls. *Journal of the American Academy of Child and Adolescent Psychiatry*, *38*, 1363–1371.

Becker, K., & Holtmann, M. (2006). Role of electroencephalography in attention-deficit hyperactivity disorder. *Expert Reviews of Neurotherapeutics*, *6*, 731–736.

Benninger, C., Matthis, P., & Scheffner, D. (1984). EEG development of healthy boys and girls. Results of a longitudinal study. *Electroencephalography and Clinical Neurophysiology*, *57*, 1–12.

Berry, C. A., Shaywitz, S. E., & Shaywitz, B. A. (1985). Girls with Attention Deficit Disorder: A silent minority? A report on behavioral and cognitive characteristics. *Pediatrics*, *76*, 801–809.

Biederman, J., & Faraone, S. V. (2004). The Massachusetts General Hospital studies of gender influences of attention-deficit/hyperactivity disorder in youth and relatives. *Psychiatric Clinics of North America*, *27*, 225–232.

Biederman, J., Faraone, S. V., Spencer, T., Wilens, T., Mick, E., & Lapey, K. A. (1994). Gender differences in a sample of adults with Attention Deficit Hyperactivity Disorder. *Psychiatry Research*, *53*, 13–29.

Biederman, J., Mick, E., Faraone, S. V., Braaten, E., Doyle, A., Spencer, T., … Johnson, M. A. (2002). Influence on gender on attention deficit hyperactivity disorder in children clinically referred to a psychiatric clinic. *American Journal of Psychiatry*, *159*, 36–42.

Biederman, J., & Spencer, T. (1999). Attention-deficit/hyperactivity disorder (AD/HD) as

a noradrenergic disorder. *Biological Psychiatry*, *46*, 1234–1242.

Bresnahan, S. M., Anderson, J. W., & Barry, R. J. (1999). Age-related changes in quantitative EEG in Attention-Deficit/Hyperactivity Disorder. *Biological Psychiatry*, *46*, 1690–1697.

Bresnahan, S. M., & Barry, R. J. (2002). Specificity of quantitative EEG analysis in adults with attention deficit hyperactivity disorder. *Psychiatry Research*, *112*, 133–144.

Bresnahan, S. M., Barry, R. J., Clarke, A. R., & Johnstone, S. J. (2006). Quantitative EEG analysis in dexamphetamine-responsive adults with attention-deficit/hyperactivity disorder. *Psychiatry Research*, *141*, 151–159.

Carlson, C. L., Tamm, L., & Gaub, M. (1997). Gender differences in children with ADHD, ODD, and co-occurring ADHD/ODD identified in a school population. *Society for Personality and Social Psychology*, *27*, 1706–1714.

Chabot, R. J., Orgill, A., Crawford, G., Harris, M., & Serfontein, G. (1999). Behavioral and electrophysiologic predictors of treatment response to stimulants in children with attention disorders. *Journal of Child Neurology*, *14*, 343–351.

Chabot, R. J., & Serfontein, G. (1996). Quantitative electroencephalographic profiles of children with attention deficit disorder. *Biological Psychiatry*, *40*, 951–963.

Clarke, A. R., Barry, R. J., Bond, D., McCarthy, R., & Selikowitz, M. (2002). Effects of stimulant medication on the EEG of children with Attention-Deficit/Hyperactivity Disorder. *Psychopharmacology*, *164*, 277–284.

Clarke, A. R., Barry, R. J., Dupuy, F. E., McCarthy, R., Selikowitz, M., & Heaven, P. C. L. (2011). Childhood EEG as a predictor of adult attention-deficit/hyperactivity disorder. *Clinical Neurophysiology*, *122*, 73–80.

Clarke, A. R., Barry, R. J., Heaven, P. C. L., McCarthy, R., Selikowitz, M., & Bryne, M. K. (2008). EEG in adults with Attention-Deficit/Hyperactivity Disorder. *International Journal of Psychophysiology*, *70*, 176–183.

Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (1998). EEG analysis in Attention-Deficit/Hyperactivity Disorder: A comparative study of two subtypes. *Psychiatry Research*, *81*, 19–29.

Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2001a). Age and sex effects in the EEG: Development of the normal child. *Clinical Neurophysiology*, *112*, 806–814.

Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2001b). Age and sex effects in the EEG: Differences in two subtypes of attention-deficit/hyperactivity disorder. *Clinical Neurophysiology*, *112*, 815–826.

Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2001c). EEG-defined subtypes of Attention-Deficit/Hyperactivity Disorder. *Clinical Neurophysiology*, *112*, 2098–2105.

Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2001d). Electroencephalogram differences in two subtypes of Attention-Deficit/Hyperactivity Disorder. *Psychophysiology*, *38*, 212–221.

Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2002). EEG differences between good and poor responders to methylphenidate and dexamphetamine in children with Attention-Deficit/Hyperactivity Disorder. *Clinical Neurophysiology*, *113*, 194–205.

Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2011). Correlations between EEG activity and behavior in children with Attention-Deficit/Hyperactivity Disorder. *Journal of Neurotherapy*, *15*, 193–199.

Clarke, A. R., Barry, R. J., McCarthy, R., Selikowitz, M., Brown, C., & Croft, R. J. (2003). Effects of stimulant medication on the EEG of children with Attention-Deficit/Hyperactivity Disorder predominantly inattentive type. *International Journal of Psychophysiology*, *47*, 129–137.

Clarke, A. R., Barry, R. J., McCarthy, R., Selikowitz, M., Clarke, D. C., & Croft, R. J. (2003). EEG activity in girls with attention-deficit/hyperactivity disorder. *Clinical Neurophysiology*, *114*, 319–328.

Clarke, A. R., Barry, R. J., McCarthy, R., Selikowitz, M., & Croft, R. (2002). EEG differences between good and poor responders to methylphenidate in boys with the inattentive type of attention-deficit/hyperactivity

disorder. *Clinical Neurophysiology*, *113*, 1191–1198.

Clarke, A. R., Barry, R. J., McCarthy, R., Selikowitz, M., & Johnstone, S. J. (2007). Effects of stimulant medications on the EEG of girls with Attention-Deficit/Hyperactivity Disorder. *Clinical Neurophysiology*, *118*, 2700–2708.

Cohn, N., Kircher, J., Emmerson, R., & Dustman, R. (1985). Pattern reversal evoked potentials: Age, sex and hemispheric asymmetry. *Electroencephalography and Clinical Neurophysiology*, *62*, 399–405.

Cragg, L., Kovacevic, N., McIntosh, A. R., Poulsen, C., Martinu, K., Leonard, G., & Paus, T. (2011). Maturation of EEG power spectra in early adolescence: A longitudinal study. *Developmental Science*, *14*, 935–943.

Cutts, K. K., & Jasper, H. H. (1939). Effect of Benzedrine sulphate and Phenobarbital on behavior problem children with abnormal electroencephalogram. *Archives of Neurology and Psychiatry (Chicago)*, *41*, 1138–1145.

DuPaul, G. J., Jitendra, A. K., Tresco, K. E., Vile Junod, R. E., Vople, R. J., & Lutz, J. G. (2006). Children with Attention Deficit Hyperactivity Disorder: Are there gender differences in school functioning? *School Psychology Review*, *35*, 292–308.

Dupuy, F. E., Clarke, A. R., Barry, R, J., McCarthy, R., & Selikowitz, M. (2011). Girls with Attention-Deficit/Hyperactivity Disorder: EEG differences between *DSM-IV* types. *Clinical EEG & Neuroscience*, *42*, 1–5.

Durston, S. (2003). A review of the biological basis of ADHD: What have we learned from imaging studies? *Mental Retardation and Developmental Disabilities*, *9*, 184–195.

Engel, A. K., Fries, P., & Singer, W. (2001). Dynamics predictions: Oscillations and synchrony on top-down processing. *Nature Reviews Neuroscience*, *2*, 704–716.

Epstein, H. (1980). EEG developmental stages. *Developmental Psychobiology*, *13*, 629–631.

Faraone, S. V., Biederman, J., & Mick, E. (2006). The age-dependent decline of attention deficit hyperactivity disorder: A meta-analysis of follow-up studies. *Psychological Medicine*, *36*, 159–165.

Faraone, S. V., Biederman, J., Spencer, T., Wilens, T., Seidman, L. J., Mick, E., & Doyle, A. E. (2000). Attention-Deficit/Hyperactivity Disorder in adults: An overview. *Biological Psychiatry*, *48*, 9–20.

Fell, J., Fernandez, C., Klaver, P., Elger, C. E., & Fries, P. (2003). Is synchronized gamma activity relevant for selective attention? *Brain Research Reviews*, *42*, 265–272.

Gasser, T., Jennen-Steinmetz, C., Sroka, L., Verleger, R., & Mocks, J. (1988). Development of the EEG of school age children and adolescents. II. Topography. *Electroencephalography and Clinical Neurophysiology*, *69*, 100–109.

Gasser, T., Verleger, R., Bacher, P., & Sroka, L. (1988). Development of the EEG of school age children and adolescents. I. Analysis of band power. *Electroencephalography and Clinical Neurophysiology*, *69*, 91–99.

Gaub, M., & Carlson, C. L. (1997). Gender differences in ADHD: A meta-analysis and critical review. *Journal of American Academy of Child and Adolescent Psychiatry*, *36*, 1036–1045.

Gershon, J. (2002). A meta-analytic review of gender differences in ADHD. *Journal of Attention Disorders*, *5*, 143–154.

Gmehlin, D., Thomas, C., Weisbrod, M., Walther, S., Pfuller, U., Resch, F., & Oelkers-Ax, R. (2011). Individual analysis of EEG background-activity within school age: Impact of age and sex within a longitudinal data set. *International Journal of Developmental Neuroscience*, *29*, 163–170.

Hale, T. S., Smalley, S. L., Dang, G., Hanada, G., Macion, J., McCracken, J. T., ... Loo, S. K. (2010). ADHD familial loading and abnormal EEG alpha asymmetry in children with ADHD. *Journal of Psychiatric Research*, *44*, 605–615.

Harmony, T., Marosi, E., Diaz de Leon, A., Becker, J., & Fernández, T. (1990). Effect of sex, psychosocial disadvantages and biological risk factors on EEG maturation. *Electroencephalography and Clinical Neurophysiology*, *75*, 482–491.

Hartung, C. M., & Widiger, T. A. (1998). Gender differences in the diagnosis of mental disorders: Conclusions and controversies

of the *DSM-IV*. *Psychological Bulletin*, *123*, 260–278.

Hermens, D. F., Kohn, M. R., Clarke, S. D., Gordon, E., & Williams, L. M. (2005). Sex differences in adolescent ADHD: Findings from concurrent EEG and EDA. *Clinical Neurophysiology*, *116*, 1455–1463.

Hermens, D. F., Williams, L. M., Lazzaro, I., Whitmont, S., Melkonian, D., & Gordon, E. (2004). Sex differences in adult ADHD: A double dissociation in brain activity and autonomic arousal. *Biological Psychology*, *66*, 221–233.

Herrmann, C. S., Frund, I., & Lenz, D. (2010). Human gamma-band activity: A review on cognitive and behavioral correlates. *Neuroscience and Biobehavioral Reviews*, *34*, 981–992.

Hill, J. & Schoener, E. (1996). Age-dependent decline of attention deficit hyperactivity disorder. *American Journal of Psychiatry*, *153*, 1143–1146.

Hobbs, M. J., Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2007). EEG abnormalities in adolescent males with AD/HD. *Clinical Neurophysiology*, *118*, 363–371.

Hudspeth, W. J., & Pribram, K. H. (1992). Psychophysiological indices of cerebral maturation. *International Journal of Psychophysiology*, *12*, 19–29.

Janzen, T., Graap, K., Stephanson, S., Marshall, W., & Fitzsimmons, G. (1995). Differences in baseline EEG measures for ADD and normally achieving preadolescent males. *Biofeedback and Self-Regulation*, *20*, 65–82.

Jasper, H., Solomon, P., & Bradley, C. (1938). Electroencephalographic analyses of behavior problem children. *American Journal of Psychiatry*, *95*, 641–658.

John, E. R., Ahn, H., Princhep, L., Trepetin, M., Brown, D., & Kaye, H. (1980). Developmental equations of the electroencephalogram. *Science*, *210*, 1255–1258.

Katada, A., Ozaki, H., Suzuki, H., & Suhara, K. (1981). Developmental characteristics of normal and mentally retarded children's EEGs. *Electroencephalography and Clinical Neurophysiology*, *52*, 192–201.

Kato, P. M., Nicholas, M. L., Kerivan, A. S., & Huffman, L. C. (2001). Identifying characteristics of older and younger females with Attention-Deficit/Hyperactivity Disorder. *Developmental and Behavioral Pediatrics*, *22*, 306–314.

Kessler, R. C., Adler, L., Barkley, R., Biederman, J., Conners, C. K., Olga-Demler, M. A., ... Zaslavsky, A. M. (2006). The prevalence and correlates of adult ADHD in the United States: Results from the National Comorbidity Survey Replication. *American Journal of Psychiatry*, *163*, 716–723.

Kinsbourne, M. (1973). Minimal brain dysfunction as a neurodevelopmental lag. *Annals of the New York Academy of Science*, *205*, 268–273.

Koehler, S., Lauer, P., Schreppel, T., Jacob, C., Heine, M., Boreatti-Hummer, A., ... Herrmann, M. J. (2009). Increased EEG power density in alpha and theta bands in adult ADHD patients. *Journal of Neural Transmission*, *116*, 97–104.

Lahey, B. B., Applegate, B., McBurnett, K., Biederman, J., Greenhill, L., Hynd, G. W., ... Shaffer, D. (1994). *DSM-IV* field trials for attention deficit hyperactivity disorder in children and adolescents. *American Journal of Psychiatry*, *151*, 1673–1685.

Lansbergen, M. M., Arns, M., van Dongen-Boomsma, M., Spronk, D., & Buitleaar, J. K. (2011). The increase in theta/beta ratio on resting-state EEG in boys with attention-deficit/hyperactivity disorder in mediated by slow alpha peak frequency. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *35*, 47–52.

Lawrence, C. A., Barry, R. J., Clarke, A. R., Johnstone, S. J., McCarthy, R., Selikowitz, M., & Broyd, S. (2005). Methylphenidate effects in attention-deficit/hyperactivity disorder: Electrodermal and ERP measures during a continuous performance task. *Psychopharmacology*, *183*, 81–91.

Lazzaro, I., Gordon, E., Li, W., Lim, C. L., Plahan, M., Whitmont, S., ... Meares, R. (1999). Simultaneous EEG and EDA measures in adolescent attention deficit

hyperactivity disorder. *International Journal of Psychophysiology*, *34*, 123–134.

Lazzaro, I., Gordon, E., Whitmont, S., Plahn, M., Li, W., Clarke, S., ... Meares, R. (1998). Quantified EEG activity in adolescent attention deficit hyperactivity disorder. *Clinical Electroencephalography*, *29*, 37–42.

Lindsley, D. B., & Cutts, K. K. (1940). Electroencephalograms of constitutionally inferior and behavior problem children. *Archives of Neurology and Psychiatry (Chicago)*, *44*, 1199–1209.

Lindsley, D. B., & Henry, C. E. (1942). The effects of drugs on behaviour and electroencephalograms of children with behavior disorder. *Psychosomatic Medicine*, *4*, 140–149.

Loo, S. K., Teale, P. D., & Reite, M. L. (1999). EEG correlates of Methylphenidate response among children with ADHD: A preliminary report. *Biological Psychiatry*, *45*, 1657–1660.

Lopes da Silva, F. (1991). Neural mechanisms underlying brain waves: From neural membranes to networks. *Electroencephalography and Clinical Neurophysiology*, *79*, 81–93.

Lubar, J. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback and Self-Regulation*, *16*, 201–224.

Lubar, J. F., White, J. N., Swartwood, M. O., & Swartwood, J. N. (1999). Methylphenidate effects on global and complex measures of EEG. *Pediatric Neurology*, *21*, 633–637.

Mann, C., Lubar, J. F., Zimmerman, A., Miller, C., & Muenchen, R. (1992). Quantitative analysis of EEG in boys with attention-deficit hyperactivity disorder: Controlled study with clinical implications. *Pediatric Neurology*, *8*, 30–36.

Matousek, M., & Petersen, I. (1973). Frequency analysis of the EEG in normal children and adolescents. In P. Kellaway & I. Petersen (Eds.), *Automation of clinical electroencephalography* (pp. 75–102). New York, NY: Raven Press.

Matousek, M., Rasmussen, P., & Gilberg, C. (1984). EEG frequency analysis in children with so-called minimal brain dysfunction and related disorders. *Advances in Biological Psychiatry*, *15*, 102–108.

Matsuura, M., Yamamoto, K., Fukuzawa, H., Okubo, H., Uesugi, H., Moriiwa, M., ... Shimazono, Y. (1985). Age development and sex differences of various EEG elements in health children and adults—Quantification wave form recognition method. *Electroencephalography and Clinical Neurophysiology*, *60*, 394–406.

Matthis, P., Scheffner, D., & Benninger, C. (1980). Spectral analysis of the EEG: Comparison of various spectral parameters. *Electroencephalography and Clinical Neurophysiology*, *52*, 218–221.

McBurnett, K., Pfiffner, L. J., Willcutt, E., Tamm, L., Lerner, M., Ottolini, Y. L., & Furmna, M. B. (1999). Experimental cross-validation of DSM-IV types of attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *38*, 17–24.

McLoughlin, G., Clarke, A. R., Barry, R, J., McCarthy, R., & Selikowitz, T. (2005). Electrophysiological parameters in psychiatric research: ADHD. *Neurophysiology*, *4*, 14–18.

Monastra, V., Lubar, J., & Linden, M. (2001). The development of a quantitative electroencephalographic scanning process for attention deficit-hyperactivity disorder: Reliability and validity studies. *Neuropsychology*, *15*, 136–144.

Monastra, V., Lubar, J., Linden, M., VanDeusen, P., Green, G., Wing, W., ... Fenger, T. (1999). Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: An initial validation study. *Neuropsychology*, *13*, 424–433.

Nunez, P. I., & Srinivasan, R. (2006). *Electric fields of the brain: The neurophysics of EEG* (2nd ed.). Oxford, UK: Oxford University Press.

Ohan, J. L., & Johnston, C. (2005). Gender appropriateness of symptom criteria for attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder. *Child Psychiatry and Human Development*, *35*, 359–381.

Pastor, P., & Reuben, C. (2008). Diagnosis of attention deficit hyperactivity disorder and learning disability: United States, 2004–2006. *Vital Health Statistics*, *10*, 237.

Petersen, I., & Eeg-Olofsson, I. (1971). The development of electroencephalogram in normal children from the age of 1 through 15 years. *Neuropadiatrie*, *2*, 247–304.

Quinn, P. O. (2005). Treating adolescent girls with ADHD: Gender-specific issues. *Journal of Clinical Psychology*, *61*, 579–587.

Quinn, P. O., & Nadeau, K. G. (Eds.). (2002). *Gender issues in AD/HD: Research, diagnosis and treatment*. Silver Spring, MD: Advantage Books.

Ratey, J., Miller, A., & Nadeau, K. (1995). Special diagnostic and treatment considerations in women with attention deficit disorder. In K. Nadeau (Ed.), *A comprehensive guide to attention deficit disorder in adults: Research, diagnosis and treatment* (pp. 260–283). New York, NY: Brunner/Mazel.

Rowe, D. L., Robinson, P. A., & Gordon, E. (2005). Stimulant drug action in attention deficit hyperactivity disorder (ADHD): Inference of neurophysiological mechanisms via quantitative modelling. *Clinical Neurophysiology*, *116*, 324–335.

Rucklidge, J. J. (2010). Gender differences in attention-deficit/hyperactivity disorder. *Psychiatric Clinics of North America*, *33*, 357–373.

Rutter, M., Caspi, A., & Moffitt, T. E. (2003). Using sex differences in psychopathology to study causal mechanisms: Unifying issues and research strategies. *Journal of Child Psychology and Psychiatry*, *44*, 1092–1115.

Satterfield, J. H., & Cantwell, D. P. (1974). CNS function and response to methylphenidate in hyperactive children. *Psychopharmacology Bulletin*, *10*, 36–37.

Satterfield, J. H., Cantwell, D. P., & Satterfield, B. T. (1974). Pathophysiology of the hyperactive child syndrome. *Archives of General Psychiatry*, *31*, 839–844.

Satterfield, J. H., Cantwell, D. P., Saul, R., Lesser, M., & Podsin, R. (1972). Physiological studies of the hyperkinetic child: I. *American Journal of Psychiatry*, *128*, 102–108.

Satterfield, J. H., Cantwell, D. P., Saul, R., Lesser, M., & Podsin, R. (1973). Response to stimulant drug treatment in hyperactive children: Predictions from EEG and neurological findings. *Journal of Autism and Child Schizophrenia*, *3*, 36–48.

Satterfield, J. H., & Dawson, M. E. (1971). Electrodermal correlates of hyperactivity in children. *Psychophysiology*, *8*, 191–197.

Satterfield, J. H., Lesser, M., Saul, R., & Cantwell, D. (1973). EEG aspects in the diagnosis and treatment of minimal brain dysfunction. *Annals of the New York Academy of Sciences*, *205*, 274–282.

Sciutto, M. J., & Eisenberg, M. (2007). Evaluating the evidence for and against the overdiagnosis of ADHD. *Journal of Attention Disorders*, *11*, 106–113.

Segalowitz, S. J., Santesso, D. L., & Jetha, M. K. (2010). Electrophysiological changes during adolescence: A review. *Brain and Cognition*, *72*, 86–100.

Shaw, J. (1981). An introduction to the coherence function and its use in EEG signal analysis. *Journal of Medical Engineering & Technology*, *5*, 279–288.

Shaw, P., Eckstrand, K., Sharpe, W., Blumenthal, J., Lerch, J. P., Greenstein, D., . . . Rapoport, J. L. (2007). Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. *Proceedings of the National Academy of Sciences USA*, *104*, 19649–19654.

Solomon, P., Bradley, C., & Jasper, H. H. (1938). Electroencephalographic analyses of behavior problem children. *American Journal of Psychiatry*, *95*, 641–658.

Solomon, P., Jasper, H. H., & Bradley, C. (1937). Studies on behavior problem children. *Archives of Neurology and Psychiatry (Chicago)*, *38*, 1350–1351.

Somsen, R. J. M., van't Klooster, B. J., van der Molen, M. W., van Leeuwen, H. M. P., & Licht, R. (1997). Growth spurts in brain maturation during middle childhood as indexed by EEG power spectra. *Biological Psychology*, *44*, 187–209.

Staller, J., & Faraone, V. (2006). Attention-deficit hyperactivity disorder in girls:

Epidemiology and management. *CNS Drugs*, *20*, 107–123.

Steriade, M., Gloor, P., Llinas, R. R., Lopes da Silva, F. H., & Mesulam, M. M. (1990). Report of IFCN committee on basic mechanisms. Basic mechanisms of cerebral rhythmic activities. *Electroencephalography and Clinical Neurophysiology*, *76*, 481–508.

Swanson, J., McBurnett, K., Wigal, T., & Pfiffner, L. (1993). Effect of stimulant medication on children with attention deficit disorder: A ''review of reviews''. *Exceptional Child*, *60*, 154–162.

Swartwood, M., Swartwood, J., Lubar, J., Timmermann, D., Zimmerman, A., & Muenchen, R. (1998). Methylphenidate effects on EEG, behaviour, and performance in boys with AD/HD. *Pediatric Neurology*, *18*, 244–250.

Takano, T., & Ogawa, T. (1998). Characterization of developmental changes in EEG gamma-band activity during childhood using the autoregressive model. *Pediatrics International*, *40*, 446–52.

Taylor, M. J., & Baldeweg, T. (2002). Application of EEG, ERP and intracranial recordings to the investigation of cognitive functions in children. *Developmental Science*, *5*, 318–334.

Thatcher, R. (1991). Maturation of the human frontal lobes: Physiological evidence for staging. *Developmental Neuropsychology*, *7*, 397–419.

Tye, C., McLoughlin, G., Kuntsi, J., & Asherson, P. (2011). Electrophysiological markers of genetic risk of attention deficit hyperactivity disorder. *Expert Reviews in Molecular Medicine*, *13*, e9.

Walker, C. F., & Kirkpatrick, B. B. (1947). Dilantin treatment for behaviour problems in children with abnormal electroencephalograms. *American Journal of Psychiatry*, *103*, 484–492.

Waschbusch, D. A., & King, S. (2006). Should sex-specific norms be used to assess attention-deficit/hyperactivity disorder or oppositional defiant disorder? *Journal of Consulting and Clinical Psychology*, *74*, 179–185.

Weiss, G., & Hechtman, L. T. (1993). *Hyperactive children grown up: ADHD in children, adolescents and adults* (2nd ed.). New York, NY: Guilford.

Whitford, T. J., Rennie, C. J., Grieve, S. M., Clarke, C. R., Gordon, E., & Williams, L. M. (2007). Brain maturation in adolescents: Concurrent changes in neuroanatomy and neurophysiology. *Human Brain Mapping*, *28*, 228–237.

Wilens, T., & Biederman, J. (1992). The stimulants. *Psychiatric Clinics of North America*, *15*, 191–222.

Wolraich, M. L., Hannah, J. N., Pinnock, T. Y., Baumgaertel, A., & Brown, J. (1996). Comparison of diagnostic criteria for attention deficit hyperactivity disorder in a countrywide sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, *35*, 319–324.