Journal of
Research in Psychopathology
https://doi.org/10.22098/jrp.2024.14500.1222

2024; Vol. 5, No. 18
Pages: 22 - 35

Review Article

Theta/Beta Ratio or not?: A Review Study of Specified QEEG Parameter for Diagnosis of ADHD Presentations

Touraj Hashemi [1], Zeynab Khanjani [2], Majid Mahmoud alilou [3] & Naimeh Mashinchi Abbasi [4*]

1. Ph.D., Professor, Psychology, Faculty of Educational Sciences and Psychology, University of Tabriz, Tabriz, Iran.
2. Ph.D., Professor, Psychology, Faculty of Educational Sciences and Psychology, University of Tabriz, Tabriz, Iran.
3. Ph.D., Professor, Clinical Psychology, Faculty of Educational Sciences and Psychology, University of Tabriz, Tabriz, Iran.
4. Ph.D., Assistant Professor, Postdoc in Cognitive Neuroscience, Faculty of Educational Sciences and Psychology, University of Tabriz, Tabriz, Iran.

Abstract

Attention deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by difficulties in sustaining attention, impulsivity, and hyperactivity According to the DSM-5, three presentations of ADHD are described: combined, predominantly inattentive, and predominantly hyperactive/impulsive. The theta-beta ratio (TBR), also referred to as the inattention index, is defined by increased theta band power (typically 4–7 Hz) and, specifically, increased theta relative to beta band power (typically 13–30 Hz). It has been reported as the most reproducible psychophysiological finding in ADHD. The present study aims to review the literature on QEEG parameters related to ADHD. The design of the study was a systematic review article. Due to increased theta, TBR is reported by many investigators as a consistent characteristic of ADHD. However, it is not a diagnostic measure for all individuals with ADHD. TBR is unnecessary in making the diagnosis for all ADHD presentations. In other words, a review of studies suggests that TBR cannot serve as a comprehensive diagnostic measure for all ADHD subtypes. It should not be generalized to all presentations. Rather, each presentation may have its specific QEEG measure. Therefore, a QEEG spectrum classification of ADHD population is a significant consideration.

Keywords

ADHD
TBR
ADHD Presentations
QEEG
Diagnostic measure

Received: 2024/01/24
Accepted: 2024/07/02
Available Online: 2024/12/01

Introduction

One of the most common childhood disorders is attention deficit-hyperactivity disorder (ADHD) (Movahed et al., 2024). ADHD is a neurodevelopmental disorder (American Psychiatric Association, 2022) that is often managed by pediatricians (McDonald & Jalbert, 2013). As defined in the DSM-5, it occurs before the age of 12 when levels of attention and hyperactivity–impulsivity fall below expected developmental norms in at least two environments (Kim, Lee, Han, Min, Kim, & Lee, 2015b). It is characterized by difficulties in allocating and sustaining attention, impulsivity and hyperactivity (American Psychiatric Association, 2022; Gonen-Yaacovi et al, 2016).

ADHD is associated with impairment in daily activities, academic performance, peer relationships and family functioning (Sawyer, Whaites, Rey, Hazell, Graetz, & Baghurst, 2002; Bastiaansen, Koot, Ferdinand, & Verhulst, 2004; Danckaerts et al., 2010; Arya, Agarwal, Yadav, Kumar Gupta, Agarwal, 2015).

According to the DSM-5, ADHD can be categorized into three presentations: combined, predominantly inattentive, and predominantly hyperactive/impulsive (American Psychiatric Association, 2022).

These presentations are based on different clusters of symptoms within the two core domains of inattention and hyperactivity/impulsivity. Difficulties with inattention or hyperactivity and impulsivity —the core symptoms of ADHD—pose a frequent psychosocial burden. With an early onset during childhood, ADHD is often persisting throughout life. Regarding heterogeneity of ADHD, the most promising treatment approach should be multimodal in nature (Taylor et al., 2004; Swanson et al., 2008; Albrecht, Uebel-von Sandersleben, Gevensleben, & Rothenberger, 2015).

Considering the wide-ranging consequences and prevalence of ADHD, there is surprisingly little data on

Corresponding author: Assistant Professor, Postdoc in Cognitive Neuroscience, Faculty of Educational Sciences and Psychology, University of Tabriz, Tabriz, Iran. E-mail: **n_psychology20@yahoo.com**

Copyright © 2024 by Authors. Published by University of Mohaghegh Ardabili. This work is licensed under a Creative Commons Attribution-NonCommercial 4.0 international license. Non-commercial purposes uses of the work are permitted, provided the original work is properly cited.

the socioeconomic impact of persistent ADHD. The estimated yearly income loss for adults with persistent ADHD in the US is $ 77 billion (Biederman & Faraone, 2006). Data from the USA indicates direct medical costs per adult ADHD patient per year of 2.500 € (Hinnenthal et al., 2005), translating to roughly 46 billion € for all persistent ADHD patients in the EU.

A principal component of ADHD is an increase in distractibility (Douglas, 1983; Thorley, 1984), which has long been regarded as one of the most common symptoms of ADHD (Barkley & Ullman, 1975). It is prominently featured in the inattentive and combined presentations of ADHD as defined by the DSM-5 (APA, 2013; Brace, Kraev, Rostron, Stewart, Overton, & Dommett, 2015).

In the predominantly inattentive presentation of ADHD (ADHD-I), individuals often avoid, dislike, or are reluctant to engage in tasks that require sustained mental effort over an extended period. They are frequently easily distracted, have difficulty maintaining attention on tasks or activities, and often switch from one activity (mental or physical) to another (Nigg, 2006).

According to the DSM-5 criteria, individuals with the predominantly hyperactive/impulsive presentation of ADHD (ADHD-H) often fidgets with or taps hands or feet or squirms in seat, leave seat in situations when remaining seated is expected. Besides, they may frequently run or climb in inappropriate situations and are unable to play or engage in leisure activities quietly. i.e, they act as if "driven by a motor". Furthermore, individuals with hyperactivity and impulsivity often talk excessively; blurt out an answer before questions have been completed as well. They have difficulty waiting their turn. They may also interrupt or intrude on others (APA, 2013).

Moreover, The National Institute of Mental Health (NIMH) serves as the lead agency within the National Institutes of Health (NIH) for ADHD research. Researchers at National Institute of Mental Health (NIMH), have suggested that individuals with hyperactivity are often excessively fidgety, restless, and "on the go." ADHD-H is characterized by maladaptive levels of hyperactivity-impulsivity without significant inattention. Individuals with ADHD-H display excessive movement that is not required to complete a task, such as wriggling their feet and legs, tapping things, rocking while seated, or shifting their posture or position while performing relatively boring tasks**.** They dash around touching or playing with whatever is in sight, or talk incessantly. Sitting still at dinner or during a school lesson or story can be a difficult task.

In addition, impulsivity is a manifestation of dysfunctional behavioral inhibition and is often demonstrated by deficits in motor impulse control reflected by reduced response inhibition (Bari & Robbins, 2013; Feja, Lang, Deppermann, Yüksel, & Wischhof, 2015).

According to DSM-5, impulsive behavior, one of the core diagnostic features of ADHD, is related with a number of negative behaviors such as social/peer difficulties (Gadow et al., 2000), academic difficulties (Merrell & Tymms, 2001), and interrupting others (Marcus, Fox, & Brown, 1982).

Impulsivity is defined as a predisposition toward rapid, unplanned reactions to internal or external stimuli, without consideration of potential negative consequences (Moeller, Barratt, Dougherty, Schmitz, Swann, 2001; Feki, Moalla, Baati, Trigui, Sellami, & Masmoudi, 2016). As defined by the National Institute of Mental Health, individuals displaying impulsivity may impulsively choose to do things that have an immediate but small payoff rather than engage in activities that may take more effort yet provide much greater but delayed rewards. They seem unable to curb their immediate reactions or think before they act and will often blurt out inappropriate comments, display their emotions without restraint, and act without regard for the long-term consequences of their conduct. They often have difficulty participating in tasks that require taking turns. Blurting out answers to questions instead of waiting to be called and flitting from one task to another without finishing are also characteristics of impulsivity.

Individuals with ADHD-C are characterized by poor behavioral inhibition, they have problems with inhibition of prepotent responses that limits their ability to control behavior, poor planning and anticipation, reduced sensitivity to errors, and poor self-regulation (Barkley, 1997; Bahçivan Saydam, Ayvaşik, Alyanak, 2015).

ADHD occurs in about 5% of children and about 2.5% of adult population (American Psychiatric Association, 2022). The disorder impacts a child's academic performance, social competence, occupational choices and personality development, with its negative effects often persisting into adulthood (Faraone et al, 2000).

Further, it imposes an enormous burden on society in terms of psychological dysfunction, adverse vocational outcomes, stress on families, and societal financial costs (Cortese & Castellanos, 2015). Adolescents with ADHD also are at a higher risk for developmentally-specific problems such as delinquency, substance abuse, and risky driving behavior (Charach, Yeung, Climans, & Lillie, 2011; Sibley et al., 2011; Thompson, Molina, Pelham, & Gnagy, 2007).

Professional guidelines describe best practices for diagnosis (American Psychiatric Association, 2013) and treatment (American Academy of Pediatrics, 2011; American Academy of Child and Adolescent Psychiatry, 2007). Combinations of pharmacological and psychological approaches are recommended for its treatment (Cortese et al., 2015).

Relatedly, poor planning and self-regulation deficits may interfere with follow-through on activities requiring effortful control and planning, such as exercise and dietary changes, which promote healthy weight (Cortese &Vincenzi, 2012).

The exact cause of the disorder has not yet been identified; however, it is believed to result from a complex interaction between the neuroanatomical system and neurobiochemistry. Additionally, genetic,

neurodevelopmental, psychosocial, and neurophysiological factors play an important role.

According to the neurodevelopmental theory formulated by Halperin and Schulz (2006) ADHD is characterized by relatively stable non-cortical dysfunctions. This theory is primarily supported by evidence from cognitive investigations showing that ADHD is associated with deficits in so-called lower cognitive mechanisms (Bedard, Trampush, Newcorn, & Halperin, 2010; Halperin, Trampush, Miller, Marks, & Newcorn, 2008).

Del Campo, Muller, and Sahakian (2012) reported that dopamine transporter availability in the striatum of individuals with ADHD is consistently reduced, indicating a problem in dopamine synthesis. In other words, the findings suggest alterations in monoamine transmission, particularly dopaminergic function (Wender, 1973; Brace, Kraev, Rostron, Stewart, Overton, & Dommett, 2015.

Generally, increasing evidence suggests that ADHD is a brain-based disorder (Swanson & Castellanos, 2002). A growing body of research has investigated the possible differences in social, interpersonal and cognitive functions of children and adults with ADHD compared with individuals without ADHD.

Regarding the pattern of cortical brain development in ADHD, Bolea-Alamañac et al (2014) have suggested an 'immaturity hypothesis', where ADHD patients require more time to achieve the same developmental milestones than unaffected subjects.

Although numerous theories have been proposed about the underlying neural basis of ADHD, it is still poorly understood (Biederman, 2005; Brace et al., 2015).

Factors such as type of instruments and methods that are applied for combining information across measures and informants could influence the diagnosis of ADHD, as well (Valo & Tannock, 2010).

Electrophysiological measures were among the first to be used to study brain processes in children with ADHD. Particularly, electroencephalography (EEG) has been applied both in research to describe and quantify the underlying neurophysiology of ADHD and clinically in its assessment and diagnosis (González-Castro, Rodríguez, López, Cueli, & Alvarez, 2013) and treatment of ADHD.

In recent years, the field of EEG has experienced a resurgence. Now we are facing the renascence of EEG. This renascence is driven by the emergence of new methods in human EEG assessment and new experimental findings in animal research which have enabled electrophysiologists to discover that alterations in oscillatory patterns of EEG play a critical role in maintenance of brain functions and consequently may be used as a powerful tool for diagnosis of brain dysfunctions (Buzsaki, 2016).

Electroencephalography (EEG) measures reflect the relationship between intracranial electrical currents and the resulting voltages on the scalp, capturing facets of brain electrical function and processing, such as the activity levels of various brain regions and their responsiveness to stimuli or cognitive tasks (Loo & Barkley, 2005).

Over the last several decades, there has been a considerable amount of research on whether EEG-derived brain wave patterns in patients with ADHD differ from those without ADHD. EEG is typically categorized into 4 frequency ranges, delta (<4 Hz), theta (4-7 Hz), alpha (8-12 Hz), and beta (13-25 Hz).

Accordingly, EEG has significantly contributed to illuminating the neurobiological mechanisms of ADHD and has revealed a high degree of sensitivity in differentiating ADHD from healthy control participants. In fact; quantitative electroencephalography (QEEG) reflects a network's ability to locally synchronize. Such ability to synchronize is related to the integrative capacities of a network and to the characteristics of its inputs. This can be strongly modified by the active state of the brain. Accordingly, developments in digitization and analytical techniques of QEEG technology have significantly enhanced the use of brain electrical activity data in clinical and research settings (Snyder & Hall, 2006). In other words, results have demonstrated that QEEG results can be used to discriminate between children with and without ADHD.

Monastra et al. (1999) also demonstrated the utility of QEEG in the assessment of ADHD.

Recently, a QEEG spectrum classification of ADHD population has been proposed, identifying four main subtypes: subtype I (abnormal increase of delta-theta frequency range centrally or centrally-frontally), subtype II (abnormal increase of frontal midline theta rhythm), subtype III (abnormal increase of beta activity frontally), and subtype IV (excess of alpha activities at posterior, central, or frontal leads). The first and second subtypes are described clinically with inattention, while in the third subtype mainly hyperactivity, impulsivity and social inadaptation are common. The low attention span is also the main complain of children with alpha excess. (Pop-Jordanova, 2012).

EEG plays an important role in evaluating, classifying and monitoring disorders. The EEG is a widely accepted method for evaluating cortical information processing and neurophysiologic changes that occur during unconsciousness and various states of conscious awareness.

Electroencephalography (EEG) was the earliest measure used to systematically examine human brain cortical activity (Loo & Makeig, 2012).

There is currently debate over routine use of QEEG. American Academy of Neurology (AAN) defines QEEG as the mathematical processing of DEEG to highlight specific waveform components, to transform EEGs into a format or domain that elucidates relevant information, or to associate numerical results with EEG data for subsequent review or comparison.

DEEG is defined by AAN as the computer-based paperless acquisition and recording of EEGs, with storage in digital format on electronic media, and waveform display on an electronic monitor or other computer output device (Kanda, Anghinah, Smidth, & Silva, 2009).

QEEG abnormal patterns are regarded as specific indicators of brain dysfunction (Kanda et al., 2009).

It is essential to determine whether children and adolescents with ADHD have underlying neurophysiologic abnormalities that are responsible for their attention-deficit/hyperactivity behavior that can be reliably detected by EEG.

The analysis of electroencephalogram (EEG) signals, as an informative quantitative method, has revealed that EEG abnormalities in children with ADHD (Barry, Johnstone, & Clarke, 2003, Chabot, di Michele, & Prichep, 2005, Loo & Barkley, 2005; Matthis, Scheffner, & Benninger, 1981) may reflect impairments in their cognitive functions (Swartwood MO, Swartwood JN, Lubar JF, Timmermann DL, Zimmerman AW, Muenchen, 1998; Loo & Barkley, 2005; Matthis et al., 1981).

Because inattention is a characteristic feature of nearly all childhood psychiatric disorders, it is often difficult to make a differential diagnosis between ADHD and other disorders with similar presentations, including Autism Spectrum Disorders (ASDs), mood and anxiety disorders, and learning disabilities, Thus, a biologically based diagnostic test or biological marker that is sensitive and specific to ADHD would be of great assistance. Based on the results previously reviewed, EEG measures are considered a promising biomarker for ADHD (Loo & Makeig, 2012).

Quantitative EEG (QEEG) can uncover the underlying neurophysiology associated with the symptoms of Attention Deficit Hyperactivity Disorder (ADHD) and learning disorders, as well as help differentiate these disorders from other comorbidities. Excess slow wave activity is the most common abnormal finding in children with attention disorders with dysfunction in the thalamocortical and/or septal-hippocampal pathways being the most likely contributors. The QEEG can play an important role in the evaluation and treatment of children and adolescents by providing information that leads to better diagnosis and design of Neurotherapy treatment protocols and medication choices. QEEG Neurometrics has been shown in a number of large studies to have high sensitivity and specificity for distinguishing children with attention disorders and/or learning disorders from children without these disorders (Duff, 2002).

In 2013, the US Food and Drug Administration (FDA) approved the Neuropsychiatric EEG-Based ADHD Assessment Aid (NEBA) (Gloss, Varma, Pringsheim, & Nuwer, 2016).

The most consistent findings reported in the literature on ADHD since the introduction of QEEG are increased absolute power in the theta band (Bresnahan, Anderson & Barry, 1999; Chabot & Serfontein, 1996; Clarke, Barry, McCarthy & Selikowitz, 1998; Mann, Lubar, Zimmerman, Miller & Muenchen, 1992) and sometimes increased absolute Delta EEG power (Bresnahan et al., 1999; Kuperman, Johnson, Arndt, Lindgren & Wolraich, 1996; Matsuura et al., 1993).

Studies indicate that children with ADHD can be differentiated from non-ADHD in over 96% of cases based on their QEEG signatures. QEEG helps distinguish the neurological basis of attention deficits caused by ADHD from those associated with other primary psychiatric disorders, such as depression, anxiety, obsessive compulsive disorder, oppositional defiant disorder or psychosis (Duff, 2002).

Studies investigating whether EEG can differentiate ADHD from learning disorders (LD) and other psychiatric conditions have shown that EEG is highly sensitive (93%-97%) and moderately specific (84%-90%) in distinguishing ADHD from LD (Loo & Barkley, 2005).

Quantitative electroencephalogram (QEEG) studies in children with learning disabilities have demonstrated alterations, including increased absolute power in the delta and theta frequency bands (Ahn, Prichep, John, Baird, Trepetin, Kaye, 1980; John, Prichep, Ahn, Easton, Fridman, & Kaye, 1983; Diaz de Leon, Harmony, Marosi, & Becker, 1988), reduction in alpha activity (Harmony et al., 1990) and reduction in alpha and beta activity, and also poor spatial differentiation (Byring, Salmi, Sainio, & Örn,1991).

The AAN, in an Evidence-Based Practice Advisory, concludes that it is highly likely that EEG theta-beta power ratio and EEG frontal beta power correctly identify patients with ADHD, with an accuracy of 89% to 94% compared to clinical evaluations. The AAN recommends that the EEG test should not replac standard clinical evaluations due to the risk of misdiagnosis, which ranges from 6% to 15% when using the theta/beta ratio. There is neither evidence for, nor against the use of theta/beta EEG power ratio either to confirm a diagnosis of ADHD, nor to support further testing. Whether comorbid disorders such as ODD have similar changes in the theta/beta ratios that mimic the reported finding in ADHD is not known.

Review of Literature: QEEG Findings in ADHD

The most common approach in QEEG research has been to measure the absolute and relative power of fixed frequency bands during resting state (eyes-closed [EC] or eyes-open [EO]) conditions (Klimesch, 1999). In this line, the largest focus of research on brain wave patterns in ADHD has been on whether there is increased theta wave activity and an increased theta/beta ratio in ADHD patients.

Lubar (1995) compared QEEG data of children with ADHD to controls and concluded, "Excessive theta activity and lack of beta activity are the primary neurological landmarks of ADHD".

The major QEEG frequency abnormalities seen in ADHD involve an excess of theta and in some cases low alpha (DeBoer & Abercrombie, 1996; Lcarashi et al., 1997; Russel et al., 1995). Moreover, an excess of theta and reduced alpha waves might result from low dopamine levels that may be caused by a hypofunctioning prefrontal cortex (PFC) and/or the nigrostriatal system, via low dopaminergic firing (Simkin, Thatcher, & Lubar, 2014).

Over the last years, EEG research has found group differences between children with or without ADHD (Sanchis et al, 2024). These include increased theta activity (Satterfield et al., 1972; Janzen et al., 1995; Clarke et al., 1998) which occurs primarily in the frontal regions (Mann et al., 1992; Chabot and Serfontein, 1996; Lazzaro et al., 1998), increased posterior delta (Matousek et al., 1984; Clarke et al., 1998) and decreased alpha and beta activity (Dykman et al., 1982; Callaway et al., 1983), also most apparent in the posterior regions (Mann et al., 1992; Clarke et al., 1998; Lazzaro et al., 1998). Increases in the theta/alpha (Matousek et al., 1984; Ucles & Lorente, 1996; Clarke et al., 1998) and theta/beta (Lubar, 1991; Janzen et al., 1995; Clarke et al., 1998; Liu, 2024) ratios have also been found in children with ADHD compared to normal children.

Studies have identified that increased theta/beta ratio (TBR) as a sensitive marker of ADHD (Monastra, Lubar, & Linden, 2001) and found it to correlate strongly with age-related changes in ADHD behavioral symptomatology overtime (Snyder & Hall, 2006). Given the excess of theta and decreased beta activity observed among children with ADHD, it is easy to understand that altering these parameters through treatment would result in improvements in ADHD symptoms (Moreno-García, Delgado-Pardo, Camacho-Vara de Rey, Meneres-Sancho, & Servera-Barceló, 2015).

Theta-beta ratio, which is called inattention index, is calculated using EEG recordings at a single site, Cz, with reference to linked ears. It was found that this index is three times higher in inattentive and combined types of ADHD children aged 6–10 years compared to a control group. Monastra et al. (2001) found that the sensitivity of this index was 86% and its specificity is 98%.

Most media sources have referred to TBR as a first brain test to diagnose children with ADHD (Arns & Gordon, 2014). However, it has not been approved in all studies.

Some researchers have found a positive association between ADHD and higher Theta/Beta ratio (Clarke et al., 2011). Therefore, most NF protocols for ADHD treatment aim to increase faster Beta frequencies, especially SMR, and decrease Theta waves (Moriyama, Polanczyk, Caye, Banaschewski, & Brandeis, & Rohde, 2012).

In theta / beta training, patients are taught to reduce activity in the theta band of the EEG (4–8 Hz) and to increase activity in the beta band (13–20 Hz). In the resting EEG, increased slow wave (theta) activity and/or reduced relative alpha (8–13 Hz) and beta activity was reported in several studies on children with ADHD. Thus, theta/beta training may address an underlying neuronal dysfunction.

Increased theta band power (typically 4-7 Hz) and, in particular, increased theta relative to beta band power (typically 13-30 Hz) has been the most reproducible psychophysiological finding in ADHD (Saad, Kohn, Clarke, Lagopoulos, & Hermens, 2015).

TBR measured at Cz was reported to differentiate reliably between children with ADHD and controls (Snyder, Quintana, Sexson, Knott, Haque, & Reynolds, 2008).

Previous studies have consistently characterized ADHD by an elevation in low frequency activity (i.e., both absolute and relative theta), during resting (EC or EO) conditions, especially when recorded from frontal sites (Barry, Clarke, & Johnstone, 2003; Bresnahan, Anderson, & Barry, 1999; Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 1998; Clarke, Barry, McCarthy, Selikowitz, & Brown, 2002; Dupuy, Clarke, Barry, McCarthy, & Selikowitz, 2013; Loo & Makeig, 2012).

Studies have indicated that an increased theta/beta ratio has 87% sensitivity, 94% specificity, and 89% accuracy for ADHD diagnosis, whereas the rating scale shows an accuracy of 47–58%. (Delorme & Makeig, 2004; Mizuhara, Wang, Kobayashi, & Yamaguchi, 2004).

Table 1. Characteristics of the studies included in the review

Row	study	Main findings	Reference
1	EEG Theta/Beta Ratio in Children With Sleep Disordered Breathing	Children with SDB exhibited worse ADHD symptoms compared to the healthy control. There was no intergroup difference in TBR.	**(Ma et al,2024)**
2	ADHD-AID: Aiding Tool for Detecting Children's Attention Deficit Hyperactivity Disorder via EEG-Based Multi Resolution Analysis and Feature Selection	This study introduced an aiding tool called ADHD-AID that assists physicians by integrating their clinical experience with EEG to achieve accurate detection of ADHD.	**(Attallah,2024)**
3	A novel approach to identify the brain regions that best classify ADHD by means of EEG and deep learning	the Frontal Lobe and the Left Hemisphere provide more significant information detecting individuals with ADHD, than using the entire set of EEG Channels. However, when combining the Temporal, Parietal and Occipital better results were achieved compared with using only the FL and LH subsets. The best performance was obtained using Feature Selection Methods. In the case of the Backward Stepwise Feature Selection method, a combination of 14 EEG channels yielded a 0.8281 $f1$-score. Similarly, using the Forward Stepwise Feature Selection method, a combination of 11 EEG channels yieldeda 0.8271 $f1$-score.	**(Sanchis et al, 2024)**
4	Mobile sleep EEG suggests delayed brain maturation in adolescents with ADHD: A focus on oscillatory spindle frequency	Adolescents with ADHD exhibited lower frequencies of spectral peaks indicating sleep spindle oscillations whereas adolescents not at-risk for ADHD showed lower spectral power in the slow sleep spindle and beta frequency ranges.	**(Vojnits et al, 2024)**
5	Detection and Classification of ADHD Using Deep Learning Based on EEG Signals	The paper objectively analyzes and summarizes obstacles and issues that may be encountered on the research path of utilizing deep learning as a tool for automated ADHD diagnosis, offering recommendations for future development.	**(Liu, 2024)**

6	Comparing executive functions in children with attention deficit hyperactivity disorder with or without reading disability: A resting-state EEG study	The results revealed that across the frontal regions, the comorbid group showed a significant reduction in the left intrahemispheric coherence in the alpha and beta bands. The ADHD-alone group exhibited increased theta and decreased alpha and beta coherence in frontal regions. In the frontoparietal regions, children in the comorbid group showed lower coherence between frontal and parietal networks compared to children without comorbid RD.	**(Tabiee et al, 2023)**
7	Automatic Identification of Children with ADHD from EEG Brain Waves	This study presents a machine learning model for EEG classification, which can identify ADHD children using statistical and time-domain and frequency-domain features of four sub-bands of EEG signals	**(Alim &Imtiaz,2023)**
8	Detection and Classification of ADHD from EEG Signals Using Tunable Q-Factor Wavelet Transform	The statistical analysis showed that the Katz and Higuchi nonlinear feature estimation methods provide potential features that can be classified with high accuracy, sensitivity, and specificity and is suitable for automatic detection of ADHD. The proposed system is capable of accurately distinguishing between ADHD and non-ADHD subjects with a maximum accuracy of 100%.	**(Joy et al, 2022)**
9	EEG for Diagnosis of Adult ADHD: A Systematic Review With Narrative Analysis	Differences in electroencephalogram activity are potentially unique to adult attention deficit hyperactivity disorder populations. The strongest support was observed for elevated levels of both absolute and relative theta power, along with the finding that alpha activity can typically differentiate adult ADHD populations from normative populations.	**(Adamou et al, 2020)**
10	EEG spectral analysis of attention in ADHD: implications for neurofeedback training?	Particularly in the ADHD-C group, higher theta and alpha activity was found with the most prominent effect in the upper-theta/lower-alpha (5.5–10.5 Hz) range. In the ADHD-I group, a significantly higher theta/beta ratio was observed at single electrodes (F3, Fz) and a tendency for a higher theta/beta ratio when considering all electrodes (large effect size). Higher 5.5–10.5 Hz activity was associated with higher reaction time variability with the effect most prominent in the ADHD-C group. A higher theta/beta ratio was associated with higher reaction times, particularly in the ADHD-I group.	**(Heinrich et al, 2014)**
11	investigating the Effectiveness of Exposure to Green Space on Absolute Power of Alpha Wave and Stress Reduction In peopleWith AttentionDeficit Hyperactivity Disorder	changes in the absolute power of the alpha band in two different thinking situations indicate different functional mechanisms of alpha waves in different brain regions.	**Raeisi Sadati et al (2024)**

Method

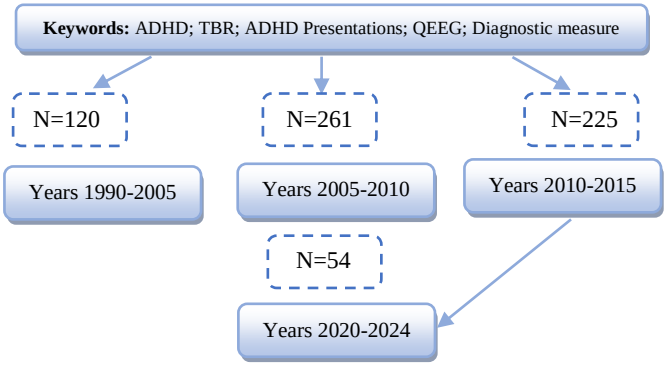
Participants

The common method for conducting systematic review is as follows:

1. Retrieval of published scientific articles. Pre-processing and standardization of collected data in terms of QEEG diagnostic measures for ADHD presentations.
2. Interpretation of results.

Procedure

Data were collected from Google Scholar, Science Direct, covering the period from 1990 to 2024. A search was conducted using the combination of the terms "ADHD, TBR, ADHD Presentations, QEEG, and Diagnostic measure. Ultimately, 660 articles met the entry criteria.



Results

Many EEG studies have reported that ADHD is characterized by an elevated Theta/Beta ratio (TBR).

Findings in QEEG studies, however, are not always consistent. The most recent studies reported an insufficient overall accuracy of 40.3–58% for the theta/beta ratio and 46.8–63% for theta power in distinguishing children with and without ADHD (Liechti et al., 2013; Ogrim, J. Kropotov, K. Hestad, 2012). Heinrich, Busch, Studer, Erbe, Moll and Kratz (2014) highlighted that recent research questions if the major part of children with ADHD are actually characterized by an increased theta/beta ratio in the resting EEG.

Also, one study demonstrated no correlation between QEEG ADHD parameters (theta/beta ratio) and the Coolidge Personality and Neuropsychology Inventory (CPNI) ADHD scale (Coolidge, Starkey, & Cahill, 2007).

Discussion

Studies conducted by Liechti et al (2013) as well as Buyck and Wiersema (2014) did not find significant differences between children with ADHD and typically developing children in any frequency band considered.

Research has revealed that the θ/β ratio demonstrates high efficacy in distinguishing between normal children and those with ADHD. In comparison to the healthy control group, the ADHD group exhibits increased θ wave activity levels, with larger absolute and relative values in the θ band, particularly in the frontal lobe region. Meanwhile, the activity of β waves in the

posterior region decreases, accompanied by smaller absolute and relative values. The θ/β ratio comparison indicates elevated slow-wave activity levels in the EEG signals of the ADHD group. Simultaneously, significant differences are observed in the θ/α ratio between the groups. In the brains of individuals with ADHD, the situation in the α wave band is similar to that in the β wave band, with an average frequency lower than that of the control group and reduced activity. Therefore, the θ/α ratio has a similar effect to the θ/β ratio. Meanwhile, in the low-frequency range, clinical subjects display a higher average frequency in the δ wave band, along with increased δ/θ ratio coefficient values" for conciseness (Liu, 2024).

Hence, θ waves, θ/α, and θ/β ratios can effectively distinguish between ADHD and control groups. Moreover, these three indicators have been found to differ among subtypes in the frontal lobe region, suggesting inherent differences in the neural models among ADHD subtypes (Liu, 2024).

Building on previous research in the field of neurodevelopmental disorders, θ wave activity serves as an electrophysiological marker for many childhood brain function disorders. According to measurement results, in the ADHD-C group, θ wave activity increases from the central to the frontal lobe region, whereas in the ADHD-I group, it decreases. This suggests that the dysfunction in ADHD-C originates from impaired frontal lobe function, while ADHD-I may suffer from either central nervous system dysfunction unrelated to the frontal lobe or different types of frontal lobe dysfunction (Clarke et al, 2001).

TBR, due to increased theta activity, has been reported by many researchers as a consistent characteristic of ADHD. Some groups recommend using the TBR during eyes-opened or eye-closed resting condition as an additional tool for the diagnosis and monitoring of ADHD. However, it is reported that the true functional significance of this measure is still unknown, and an elevated theta activity may represent a nonspecific marker of cortical dysfunction, which is also observed in other disorders such as epilepsy, bipolar disorder, and polysubstance abuse

Furthermore, Arns, Conners, and Kraemer (2012) conducted a meta-analysis on the TBR in ADHD. TBR data during Eyes Open from location Cz were analyzed from children/adolescents 6-18 years of age with and without ADHD. The analysis identified nine studies with a total of 1,253 children/adolescents with ADHD and 517 without ADHD. The grand-mean effect size (ES) for the 6-13 year-olds was.75 and for the 6-18 year-olds was 0.62. However the test for heterogeneity remained significant. The authors concluded that these ESs are misleading and considered an overestimation. Post-hoc analysis revealed a decreasing difference in TBR across years, explained by an increasing TBR for the non-ADHD groups. Consequently, they proposed that excessive TBR cannot be regarded a reliable diagnostic measure for ADHD, however a substantial sub-group of ADHD patients do deviate on this measure and TBR has prognostic value in this sub-group, supporting its use as a prognostic rather than diagnostic measure.

In a study by Poli et al (2014) including 46 patients with ADHD and 68 controls, high-density EEG was recorded from 60 electrodes under resting eyes closed conditions. No significant differences in theta activity were observed between children with and without ADHD. They reported Cohens' D effect size of 0.17 for the TBR between both groups. However, in previous studies, Monastra et al. (1999, 2001) as well as Snyder et al. (2008) the large effect sizes (ES: 1.6–1.8) were reported (Arns & Gordon, 2014).

Accordingly, results of a study by Monastra et al. (1999) suggested significant maturational effects in cortical arousal in the prefrontal cortex accompanied by cortical slowing. The typical pattern demonstrated was excess theta (4-8Hz) and decreased beta (13-21Hz), indicated by an increased theta-beta power ratio in comparison to controls.

Previous studies such as Lubar, Swartwood, Swartwood, and Timmerman (1995) as well as Mann, Lubar, Zimmerman, Miller, and Muenchen (1991) have also obtained similar finding (White, Lubar, & Hutchens, 2000).

It should be noted that EEG profiles in ADHD during cognitive tasks are also important, as most studies have focused on resting EEG.

Hence, an increased TBR cannot currently be considered a reliable diagnostic measure for ADHD. However, based on recent studies, this excess theta and TBR is found in a substantial subgroup of patients with ADHD (25%-40%) and has been demonstrated to be of prognostic value in predicting treatment outcome to stimulant medication and neurofeedback, warranting its application as a prognostic measure rather than a diagnostic measure (Arns, Conners, & Kraemer, 2012).

Russian scientists from the Human Brain Institute in St Petersburg (Kropotov, 2009) showed TBR is a reliable measure only for a subset of the ADHD population. Mapping this index in normal population showed that the location of the maximum of TBR changes significantly with age. For example, the maximum of theta-beta index move from central-parietal location at 7-8 years old children to frontalcentral location in adults. The conclusion was that for better results in discriminating the ADHD population from healthy subjects this index must be measured in different electrode positions depending on age.

As Hagemann et al (2005) have suggested it may be more relevant to consider EEG during task processing. However, it is important to note that resting EEG reflects not only a trait marker but also a state marker.

Heinrich et al. (2014) reported that in contrast to recent resting-EEG studies (e.g., Ogrim, Kropotov, and Hestad, 2012; Liechti et al., 2013), significant differences related to the theta band and the alpha band were observed between children with ADHD and typically developing children: activity in these frequency band was significantly larger in children with

ADHD. In the ADHD-C group, effects were most pronounced in the 5.5–10.5 Hz (upper-theta/lower-alpha) band. Global statistical analysis did not reveal a significant group effect for the theta/beta ratio, i.e., the major part of the children with ADHD was not characterized by an increased theta/beta ratio.

Moreover, Poil et al. (2014) reported higher beta and lower alpha power in adult ADHD combined type group compared with controls; however, this was inconsistent with their observations of ADHD combined type children.

Therefore, specifying EEG measures for each ADHD presentation could be highly beneficial, particularly in designing treatment protocols based on EEG biofeedback.

It is surprising to note that although decreased relative beta activity, increased absolute and relative theta activity, or increased theta/beta ratio has differentiated ADHD from controls (Bresnahan et al, 1999; Bresnahan & Barry, 2002; Clarke, Barry, McCarthy, & Selikowitz, 2002; Lubar, 1991; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992; Monastra et al., 1999), other studies have also indicated increased beta power in ADHD, suggesting that this may reflect a distinct subgroup (e.g., combined presentation with poor IQ) of ADHD (Chabot & Serfontein, 1996; Kuperman, Johnson, Arndt, Lindgren, & Wolraich, 1996).

Heinrich et al (2014) have emphasized that in previous studies; only a single EEG channel was typically used to calculate feedback information in EEG NF training. For theta/beta training in ADHD, most often electrode Cz is considered. In their data, increased upper-theta/lower-alpha activity in the ADHD-C group and a higher theta/beta ratio in the ADHD-I group were not topographically specific, i.e., they were not restricted to or particularly pronounced at a specific electrode. Looking at single electrodes, effects at electrode Cz appeared rather smaller than larger compared to frontal, electrodes (F3, Fz).

Consequently, considering resting EEG, EEG during cognitive tasks, different ADHD presentations, and electrode site selection is of great importance. Not all ADHD subgroups can be characterized by TBR, and electrode Cz can not be considered in theta/beta training in all ADHD subgroups, as well.

Accordingly, frontal midline theta (associated with working memory and cognitive control processes; Jensen & Tesche, 2002; Enriquez-Geppert, Huster, Scharfenort, Mokom, Zimmermann, & Herrmann, 2014) could interfere with the more generalized theta pattern addressed for example in theta/beta training if feedback information is calculated from Cz only.

So, Heinrich et al (2014) stated that a more robust/more specific feedback signal may be obtained if not a single channel but a combination of several electrodes is used. If NF training does not target a topographically specific EEG pattern, the average of a grid of distributed electrodes may be preferable.

The meta-analysis of theta/beta ratio research in ADHD by Arns et al (2012) suggested that the theta/beta ratio may serve as a prognostic measure but not a diagnostic one.

It could be stated that the conventional neurofeedback protocol for reducing inattention and impulsivity, which consists of operant enhancement of beta activity and suppressing theta activity, could not be applied for all ADHD subtypes. Hence, the protocols of neurofeedback should be adjusted for each subgroup based on its specific QEEG measures.

Conclusion

Theta/Beta Ratio (TBR) is not the only measure for Diagnosis of ADHD. Arns and Gordon (2014) proposed that the Theta/Beta ratio is not an unambiguous diagnostic marker in all cases, and suggested that it is unlikely for a single biomarker to differentiate all ADHD patients from controls.

Therefore, review of the studies suggests that EEG plays an important role in evaluating, classifying and following disorders. Accordingly, QEEG abnormal patterns can be regarded as a specific sign of brain dysfunction. Based on the reviewed findings, EEG measures have been considered promising biomarkers for ADHD. QEEG can help reveal the underlying neurophysiology associated with the symptoms of ADHD, and could be useful in differentiating ADHD from other comorbidities. However, QEEG ADHD parameter (theta/beta ratio; TBR) could not be considered as the comprehensive QEEG parameter for all subtypes.

Consequently, it can be proposed that TBR is not necessary for diagnosing all ADHD presentations. In other words, TBR could not be a comprehensive diagnostic measure for all ADHD subtypes. It should not be generalized for all presentations. Rather, each presentation could have its specific QEEG measure.

Therefore, a QEEG spectrum classification of ADHD population would be an important notification. Also, it could have a crucial implication for EEG biofeedback.

One limitation of this research, related to QEEG. Complementary findings in the ERP literature reviewed in the companion paper (Barry et al., 2003) suggest that if selected ERP data were used in concert with the EEG, we could expect improved classification accuracy.

Acknowledgment

The authors would like to acknowledge the following individuals who provided feedback and expert opinions during the development of this paper.

Disclosure Statement

Author declare that they have no conflicts of interest.

ORCID

Touraj Hashemi: https://www.orcid.org/0000-0002-8353-6104

References

AAN. Clinical Practice Guideline Process Manual. Ed. St Paul, MN: Am. *Acad. of Neurology*. PMID: 20876463

Adamou, M., Fullen, T., & Jones, S. L. (2020). EEG for Diagnosis of Adult ADHD: A Systematic Review With Narrative Analysis. Frontiers in psychiatry, 11, 871. https://doi.org/10.3389/fpsyt.2020.00871

Ahn, H., Prichep, L., John, E.R., Baird, H., Trepetin, M., & Kaye, H. (1980). Developmental equations reflect brain dysfunctions. *Science, 210*, 1259-1262. doi: 10.1126/science.7434027.

Albrecht, B., Uebel-von Sandersleben, H., Gevensleben, H., & Rothenberger, A. (2015). Pathophysiology of ADHD and associated problems-starting points for NF interventions? *Frontiers in Human Neuroscience, 9*, 1-14.doi: 10.3389/fnhum.2015.00359

Alim, Anika & Imtiaz, Masudul. (2023). Automatic Identification of Children with ADHD from EEG Brain Waves. Signals. 4. 193-205. doi:10.3390/signals4010010.

American Psychiatric Association. (2000). DSM-IV-TR (American P.). Washington, DC. doi:10.1176/appi.books.9780890423349

American Academy of Child and Adolescent Psychiatry. (2007). Practice parameter for the assessment and treatment of children and adolescents with attention-deficit/hyperactivity disorder. *J Am Acad Child Adolesc Psychiatry,46*, 894-921. doi: 10.1097/chi.0b013e318054e724.

American Academy of Pediatrics (2011). Subcommittee on Attention-Deficit/Hyperactivity Disorder, Steering Committee on Quality Improvement and Management. *ADHD: Clinical practice guideline for the diagnosis, evaluation, and treatment of attention-deficit/hyperactivity disorder in children and adolescents. Pediatrics, 128*, 1007-22. https://doi.org/10.1542/peds.2011-2654

American Psychiatric Association. (2022). *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Washington, DC: APA. https://www.mredscircleoftrust.com/storage/app/media/DSM%205%20TR.pdf

Arya, A., Agarwal,V.,Yadav, S., Kumar Gupta, P., & Agarwal, M. (2015). A study of pathway of care in children and adolescents with attention deficit hyperactivity disorder. *Asian Journal of Psychiatry, 17*, 10-15. doi: 10.1016/j.ajp.2015.07.013. Epub 2015 Aug 6.

Arns, M., Conners, C.K., & Kraemer, H.C. (2012). A decade of EEG theta/beta ratio research in ADHD: A meta-analysis. J. Atten. Disord. http://dx.doi.org/10.1177/1087054712460087

Arns, M., & Gordon, E. (2014). Quantitative EEG (QEEG) in psychiatry: Diagnostic or prognostic use? Clinical Neurophysiology, 125, 1504-1506. DOI: 10.1016/j.clinph.2014.01.014

Attallah O. (2024). ADHD-AID: Aiding Tool for Detecting Children's Attention Deficit Hyperactivity Disorder via EEG-Based Multi-Resolution Analysis and Feature Selection. Biomimetics (Basel, Switzerland), 9(3), 188. https://doi.org/10.3390/biomimetics9030188.

Bahçivan Saydam, R., Ayvaşik, H.B., & Alyanak, B. (2015). Executive functioning in subtypes of Attention Deficit Hyperactivity Disorder. *Arch Neuropsychiatr, 52*, 386-392. doi: 10.5152/npa.2015.8712

Barkley, R.A., & Ullman, D.G. (1975). A comparison of objective measures of activity and distractibility in hyperactive and nonhyperactive children. J Abnorm Child Psychol, 3(3), 231-244. DOI: 10.1007/BF00916753

Barkley R.A. (1997). Behavioral inhibition, sustained attention, and executive function: Constructing a unified theory of ADHD. *Psychol Bull, 121*, 65-94. doi: 10.1037/0033-2909.121.1.65

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.doi:10.1016/S1388-2457 (02)00362-0

Barry, R.J., Johnstone S.J., & Clarke, A.R. (2003). A review of electrophysiology in attention-deficit/hyperactivity disorder: II. Event-related potentials. *Clinical Neurophysiology, 114*, 184-198. doi:10.1016/s1388-2457(02)00363-2

Bastiaansen, D., Koot, H.M., Ferdinand, R.F., & Verhulst, F.C. (2004). Quality of life in children with psychiatric disorders; self-, parent, and clinician report. J. Am. Acad. Child Adolesc. Psychiatry, 43, 221-230. doi: 10.1097/00004583-200402000-00019.

Bedard, A.C., Trampush, J.W., Newcorn, J.H., & Halperin, J.M. (2010). Perceptual and motor inhibition in adolescents/young adults with childhood-diagnosed ADHD. Neuropsychology, 2010, 24, 424–434. doi:10.1037/a0018752

Biederman, J. (2005). Attention-deficit/hyperactivity disorder: a selective overview. Biol Psychiatry, 57(11), 1215-1220. doi: 10.1016/j.biopsych.2004.10.020.

Biederman, J., & Faraone, S. (2006). The effects of attention-deficit hyperactivity disorder on employment and household income. *Medscape General Medicine, 8*, 12. doi: 10.3389/fnmol.2022.925049

Bolea-Alamañac, B., Nutt, D.J., Adamou, M., Asherson, P., Bazire, S., Coghill, D., Heal, D., Müller, U., Nash, J., Santosh, P., Sayal, K., & Sonuga-Barke, E., & Young, S.J. (2014). Evidence-based guidelines for the pharmacological management of attention deficit hyperactivity disorder: Update on recommendations from the British Association for Psychopharmacology. *Journal of Psychopharmacology*, 1-25. doi: 10.1177/0269881113519509

Brace, L.R., Kraev, I., Rostron, C.L., Stewart, M.G., Overton, P.G., & Dommett, E. J. (2015). Altered visual processing in a rodent model of attention-

deficit hyperactivity disorder. *Neuroscience*, *303*, 364-377. doi: 10.1016/j.neuroscience.2015.07.003.

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. doi:10.1016/S0006-3223(99)00042-6

Bresnahan, S.M., & Barry, R.J. (2002). Specificity of quantitative EEG analysis in adults with attention deficit hyperactivity disorder. *Psychiatry Research*, *112*, 133-144. doi: 10.1016/s0165-1781(02)00190-7.

Buyck, I., & Wiersema, J.R. (2014). Resting electroencephalogram inattention deficit hyperactivity disorder: Developmental course and diagnostic value. *Psychiatry Res.* 216, 391-397. doi:10.1016/j.psychres.2013.12.055

Buzsaki, G. (2016). EEG rhythms. *In Quantitative EEG, Event-Related Potentials and neurotherapy*. doi:10.1016/B978-0-12-374512-5.X0001-1

Byring, R., Salmi, T.K., Sainio, K.O., & Örn, H.P. (1991). EEG in children with spelling disabilities. *Electroencephalogr Clin Neurophysiol*, *79*, 247-255. doi: 10.1016/0013-4694(91)90119-o

Callaway. E., Halliday. R., & Naylor, H. (1983). Hyperactive children's event-related potentials fail to support underarousal and maturational-lag theories. *Arch Gen Psychiatry 40*, 1243-1248. doi: 10.1001/archpsyc.1983.01790100089012

Chabot, R.J., & Serfontein, G. (1996). Quantitative electroencephalographic profiles of children with attention deficit disorder. *Biological Psychiatry*, *40*, 951-963. doi: 10.1016/0006-3223(95)00576-5

Chabot, R.J., di Michele, F., & Prichep, L.S. (2005). The role of quantitative electroencephalography in child and adolescent psychiatric disorders. *Child and Adolescent Psychiatric Clinics of North America*,*14*, 21-53. doi: 10.1016/0006-3223(95)00576-5

Charach, A., Yeung, E., Climans, T., & Lillie, E. (2011). Childhood attention-deficit/hyperactivity disorder and future substance use disorders: Comparative meta-analyses. *Journal of the American Academy of Child and Adolescent Psychiatry*, *50*, 9-21. doi: 10.1016/j.jaac.2010.09.019

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. doi:10.1016/s0165-1781(98)00072-9

Clarke, A.R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2002). EEG analysis of children with attention-deficit/hyperactivity disorder and comorbid reading disabilities. *Journal of Learning Disabilities*, *35*, 276-285. https://doi.org/10.1177/002221940203500309

Clarke, A.R., Barry, R.J., McCarthy, R., Selikowitz, M., & Brown, C.R. (2002). EEG evidence for a new conceptualization of attention deficit hyperactivity disorder. *Clinical Neurophysiology*, *113*, 1036-1044. doi:10.1016/S1388-2457(02)00115-3

Clarke, A.R., Barry, R.J., Dupuy, F.E., et al. (2011). Behavioural differences between EEG-defined subgroups of children with Attention-Deficit/Hyperactivity Disorder. *Clin Neurophysiol*, *122*, 1333-1341. doi: 10.1016/j.clinph.2010.12.038

Coolidge FL, Starkey MT, & Cahill BS. (2007). Comparison of a parent-rated DSM-IV measure of attention-deficit/hyperactivity disorder and quantitative EEG parameters in an outpatient sample of children. *J Clin Neurophysiol*, *24*, 348-51. doi: 10.1097/WNP.0b013e318067bcfc

Cortese, S., & Castellanos, F.X. (2015). Attention Deficit/Hyperactivity Disorder. *Neurobiology of Brain Disorders*, 42-58. Doi:10.1016/B978-0-12-398270-4.00004-5

Cortese, S., Ferrin, M., Brandeis, D., Buitelaar, J., Daley, D., Dittmann, R.W., Holtmann, M., Santosh, P., Stevenson, J., Stringaris, A., Zuddas, A., & Sonuga-Barke, E.J.S. (2015). Cognitive training for Attention-Deficit/Hyperactivity Disorder: Meta-analysis of clinical and neuropsychological outcomes from randomized controlled trials. *Journal of the American Academy of Child & Adolescent Psychiatry*, *54* (3), 164-174. doi: 10.1016/j.jaac.2014.12.010

Danckaerts, M., Sonuga-Barke, E.J., Banaschewski, T., Buitelaar, J., Döpfner, M., Hollis, C., Santosh, P., Rothenberger, A., Sergeant, J., Steinhausen, H.C., Taylor, E., Zuddas, A., & Coghill, D. (2010). The quality of life of children with attention deficit/hyperactivity disorder: a systematic review. Eur. Child Adolesc. Psychiatry, 19, 83-105. doi: 10.1007/s00787-009-0046-3

DeBoer, P., & Abercrombie, E.D, (1996). Physiological release of striatal acetylcholine in vivo: modulation by D1 and D2 dopamine receptor subtypes. *J Pharmacol Exp Ther*, *277*, 775-783. PMID: 8627558

Del Campo N, Muller U and Sahakian BJ (2012) Neural and behavioral endophenotypes in ADHD. *Curr Top Behav Neurosci* 11, 65-91. doi: 10.1007/7854_2012_200

Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J Neurosci Methods*,*134*, 9-21. doi: 10.1016/j.jneumeth.2003.10.009

Diaz de Leon, A.E., Harmony, T., Marosi, E., & Becker J. (1988). Effect of different factors on EEG spectral parameters. *Int J Neurosci*,*43*, 123-131. doi: 10.3109/00207458808985789

Douglas, V. (1983). In M,R. (ed) *Developmental Neuropsychiatry*. Guildford Press, New York, pp. 280-329. doi:10.1037/h0085821

Duff, J. (2002). QEEG Neurometrics and differential diagnosis of ADHD. *Behavioural Neurotherapy Clinic*, 1-3. doi:10.1177/1359104502007004023

Dupuy, F.E., Clarke, A.R., Barry, R.J., McCarthy, R., & Selikowitz, M. (2013). EEG differences between the combined and inattentive types of Attention-Deficit/Hyperactivity Disorder in girls: A further investigation. *Clinical EEG and Neuroscience*. Advance online publication. doi:10.1177/1550059413501162

Dykman, R., Holcomb, P., Oglesby, D., & Ackerman, P. (1982). Electrocortical frequencies in hyperactive, learning-disabled, mixed, and normal children. *Biol Psychiatry,17*, 675-685. doi:10.1177/002221949402701002

Enriquez-Geppert, S., Huster, R.J., Scharfenort, R., Mokom, Z.N., Zimmermann, J., & Herrmann, C.S. (2014). Modulation of frontal-midline theta by neurofeedback. *Biol. Psychol.* 95, 59–69. doi: 10.1016/j.biopsycho.2013.02.019

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, *Biol. Psychiatry*, *48*, 9-20. doi: 10.1016/s0006-3223(00)00889-1

Feja, M., Lang, M., Deppermann, L., Yüksel, A., Wischhof, L. (2015). High levels of impulsivity in rats are not accompanied by sensorimotor gating deficits and locomotor hyperactivity. *Behavioural Processes*, 121, 13-20. doi: 10.1016/j.beproc.2015.10.011

Feki, I., Moalla, M., Baati, I., Trigui, D., Sellami, R., & Masmoudi, J. (2016). Impulsivity in bipolar disorders in a Tunisian sample. *Asian Journal of Psychiatry*, *22*, 77–80. doi: 10.1016/j.ajp.2016.05.005

Gadow, K.D., Nolan, E.E., Litcher, L., Carlson, G.A., Panina, N., Golovakha, E., ... Bromet, E.J. (2000). Comparison of attention-deficit/hyperactivity disorder symptom subtypes in Ukranian school children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*(12), 1520–1527. doi:10.1097/00004583-200012000-00014.

Gloss, D., Varma, J.K., Pringsheim, T., & Nuwer, M.R. (2016). Practice advisory: The utility of EEG theta/beta power ratio in ADHD diagnosis. *Neurology*, *87*, 1–5. doi: 10.1212/WNL.0000000000003265

Gonen-Yaacovi, G., Arazi, A., Shahar, N., Karmon, A., Haar, Sh., Meiran, N., & Dinstein, I. (2016). Increased ongoing neural variability in ADHD. *Cortex*, 81 50-63. doi:10.1016/j.cortex.2016.04.010

González-Castro, P., Rodríguez, C., López, A., Cueli, M., & Álvarez, L. (2013). Attention Deficit Hyperactivity Disorder, differential diagnosis with blood oxygenation, beta/theta ratio, and attention measures. *International Journal of Clinical and Health Psychology*, *13*, 101-109. Doi:10.1016/S1697-2600(13)70013-9.

Hagemann, D., Hewig, J., Seifert, J., Naumann, E., & Bartussek, D. (2005). The latent state-trait structure of resting EEG asymmetry: Replication and extension. *Psychophysiology* 42, 740-752. doi:10.1111/j.1469-8986.2005. 00367.x

Halperin, J.M., Trampush, J.W., Miller, C.J., Marks, D.J., & Newcorn, J.H. (2008). Neuropsychological outcome in adolescents/young adults with childhood ADHD: profiles of persisters, remitters and controls. J Child Psychol Psychiatry,49, 958-966. doi:10.1111/j.1469-7610.2008.01926.x

Harmony, T., Hinojosa, G., Marosi E, et al. (1990). Correlation between EEG spectral parameters and an educational evaluation. *Int J Neurosci*, *54*, 145-155. doi: 10.3109/00207459008986630

Heinrich, H., Busch, K., Studer, P., Erbe, K., Moll, G.H., & Kratz, O. (2014). EEG spectral analysis of attention in ADHD: Implications for neurofeedback training? *Frontiers in Human Neuroscience*, *8*, 1-10. doi: 10.3389/fnhum.2014.00611

Hinnenthal, J.A., Perwien, A.R., & Sterling, K.L. (2005). A comparison of service use and costs among adults with ADHD and adults with other chronic diseases. *Psychiatr. Serv*, *56*, 1593-1599. doi: 10.1176/appi.ps.56.12.1593

Janzen, T., Graap, K., Stephanson, S., Marshall, W., & Fitzsimmons, G. (1995). Differences in baseline EEG measures for ADD and normally achieving preadolescent males. *Biofeedback Self-Regul,20*, 65-82. doi:10.1007/BF01712767

Jensen, O., & Tesche, C.D. (2002). Frontal theta activity in humans increases with memory load in a working memory task. *Eur. J. Neurosci.* 15, 1395-1399. doi:10. 1046/j.1460-9568.2002.01975.x

John, E.R., Prichep, L., Ahn, H., Easton, P., Fridman, J., & Kaye, H. (1983). Neurometric evaluation of cognitive dysfunctions and neurological disorders in children. *Progr Neurobiol, 21*, 239-290. doi: 10.1016/0301-0082(83)90014-x

Joy, Rc & George, s.Thomas & Rajan, Albert & Subathra, M. & Sairamya, N. & Prasanna, J. & Mohammed, Mazin & Al-Waisy, Alaa & Jaber, Mustafa & Al-Andoli, Mohammed. (2022). Detection and Classification of ADHD from EEG Signals Using Tunable Q-Factor Wavelet Transform. Journal of Sensors. 2022. 3590973. doi:10.1155/2022/3590973.

Kanda, P.A.M., Anghinah, R., Smidth, M.T., & Silva, J.M. (2009). The clinical use of quantitative EEG in cognitive disorders. *Dementia & Neuropsychologia*, *3*(3), 195-203. doi: 10.1590/S1980-57642009DN30300004

Kim, J.W., Lee, J., Kim, B-N., Kang, T., Min, K.J., Han, D.H., & Lee, Y.S. (2015). Theta-phase gamma-amplitude coupling as a neurophysiological marker of attention deficit/hyperactivity disorder in children. *Neuroscience Letters*, *603*, 25-30. doi:10.1016/j.neulet.2015.07.006

Kim, J.W., Lee, Y.S., Han, D.H., Min, K.J., Kim, D.H., & Lee, CH.W. (2015b). The utility of quantitative electroencephalography and integrated visual and auditory Continuous Performance Test as auxiliary tools for the Attention Deficit Hyperactivity Disorder diagnosis. *Clinical Neurophysiology, 126*, 532-540. doi:10.1016/j.clinph.2014.06.034

Klimesch, W. (1999). EEG alpha and theta oscillations reflect cognitive and memory performance: A review and analysis. *Brain Research Reviews*, *29*, 169-195. doi: 10.1016/s0165-0173(98)00056-3

Kuperman, S., Johnson, B., Arndt, S., Lindgren, S., & Wolraich, M. (1996). Quantitative EEG differences in a nonclinical sample of children with ADHD and

undifferentiated ADD. *Journal of the American Academy of Child & Adolescent Psychiatry*, *35*, 1009-1017. doi:10.1097/00004583-199608000-00011

Lazzaro, I., Gordon, E., Whitmont, S., Plahn, M., Li, W., Clarke, S., Dosen, A., & Meares, R. (1998). Quantified EEG activity in adolescent attention deficit hyperactivity disorder. *Clin Electroenceph,29*, 37-42. doi: 10.1177/155005949802900111

Liechti, M.D., Valko, L., Müller, U.C., Döhnert, M., Drechsler, R., Steinhausen, H.-C., & Brandeis, D. (2013). Diagnostic value of resting electroencephalogram in attention deficit/hyperactivity disorder across the lifespan, *Brain Topogr. 26*, 135–151. doi:: 10.1007/s10548-012-0258-6

Liu, X. (2024). Detection and Classification of ADHD Using Deep Learning Based on EEG Signals. Highlights in Science, Engineering and Technology, 91, 191-199. doi:10.54097/12cjyf16

Loo, S.K,, & Barkley, R.A. (2005). Clinical utility of EEG in attention deficit hyperactivity disorder. *Applied Neuropsychology*, *12*, 64-76. doi: 10.1207/s15324826an1202_2

Loo, S.K., & Makeig, S. (2012). Clinical utility of EEG in attention-deficit/hyperactivity disorder: A research update. *Neurotherapeutics*, *9*, 569-587. doi: 10.1207/s15324826an1202_2

Lubar, J.F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback & Self-Regulation*, *16*(3), 201-225. doi: 10.1007/BF01000016

Lubar, J.F. (1995). Neurofeedback for the management of attention-deficit/ hyperactivity disorder. In Schwartz, M. S. & Associates (Eds.), *Biofeedback: A PractitionersGuide* (2nd ed.) (pp. 493-522). New York: Guilford Press. https://www.guilford.com/excerpts/schwartz.pdf?t=1

Lubar, J.F., Swartwood, M.O., Swartwood, J.N., & Timmermann, D.L. (1996). Quantitative EEG and auditory event-related potentials in the evaluation of attention-deficit/hyperactivity disorder: Effects of methylphenidate and implications for neurofeedback training. [Monograph: Assessment of Attention-Deficit/Hyperactivity Disorders]. *Journal of Psychoeducational Assessment*, 143-204. doi:10.1007/BF01000016

Ma, D., Wu, Y., Wang, C., Zhao, F., Xu, Z., & Ni, X. (2024). Characteristics of ADHD Symptoms and EEG Theta/Beta Ratio in Children With Sleep Disordered Breathing. Clinical EEG and neuroscience, 15500594241234828. Advance online publication. doi:10.1177/15500594241234828

Mann, C.A., Lubar, J.F., Zimmerman, A. W., Miller, C.A., & Muenchen, R.A. (1992). Quantitative analysis of EEG in boys with attention-deficit/hyperactivity disorder: Controlled study with clinical implications. Pediatric Neurology, 8, 30-36. doi:10.1016/0887-8994(92)90049-5

Marcus, S.D., Fox, D., & Brown, D. (1982). Identifying school children with behavior disorders. *Community Mental Health Journal*, *18*(4), 249–256. doi:10.1007/BF00754539.

Matousek, M., Rasmussen, P., & Gilberg, C. (1984). EEG frequency analysis in children with so-called minimal brain dysfunction and related disorders. *Adv Biol Psychiatry*, *15*, 102-108. doi:10.1159/000410508

Matthis, P., Scheffner, D., & Benninger, C. (1981). Spectral analysis of the EEG: Comparison of various spectral parameters. *Electroencephalic Clinical Neurophysiology*, *52*, 218–221. doi:10.1016/0013-4694(81)90171-1

McDonald, D.C., & Jalbert, S.K. (2013). Geographic variation and disparity in stimulant treatment of adults and children in the United States in 2008. *Psychiatr Serv*, 64, 1079-86. PMID: 23912601

Merrell, C., & Tymms, P.B. (2001). Inattention, hyperactivity and impulsiveness: Their impact on academic achievement and progress. *British Journal of Educational Psychology*, *71*(1), 43–56. doi:10.1348/000709901158389

Mizuhara, H., Wang, L.-Q., Kobayashi, K., & Yamaguchi, Y. (2004). A long-range cortical network emerging with theta oscillation in a mental task, Neuroreport 15 (2004) 1233–1238. doi: 10.1097/01.wnr.0000126755.09715.b3

Moeller F.G., Barratt E.S., Dougherty D.M., Schmitz J.M., & Swann A.C. (2001). Psychiatric aspects of impulsivity. *Am. J. Psychiatry*, *158*, 1783-1793. doi:10.1176/appi.ajp.158.11.1783

Monastra, V.J., Lubar, J.F., Linden, M., VanDeusen, P., Green, G., Wing, W., . . . Fenger, T.N. (1999). Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: An initial validation study. *Neuropsychology*, *13*, 424-433. doi:10.1037/0894-4105.13.3.424

Monastra, V.J., Lubar, J.F., & Linden, M. (2001). The development of a quantitative electroencephalographic scanning process for attention deficit-hyperactivity disorder: Reliability and validity studies. *Neuropsychology*, *15*, 136-144. doi: 10.1037//0894-4105.15.1.136

Moreno-García, I., Delgado-Pardo, G., Camacho-Vara de Rey, C., Meneres-Sancho, S., & Servera-Barceló, M. (2015). Neurofeedback, pharmacological treatment and behavioral therapy in hyperactivity: Multilevel analysis of treatment effects on electroencephalography. *International Journal of Clinical and Health Psychology*, *17*, 215-225. doi:10.1016/j.ijchp.2015.04.003

Moriyama, T.S., Polanczyk, G., Caye, A., Banaschewski, T., Brandeis, D., & Rohde, L.A. (2012). Evidence-based information on the clinical use of neurofeedback for ADHD. Neurotherapeutics, 1-11. doi: 10.1007/s13311-012-0136-7

Movahed, J., Amirmajd, M., Ghamari, M., & Pouyamanesh, J. (in press). Comparison of the effectiveness of parent-child relationship therapy and Barkley's parent training of mothers of children with

Attention Deficit-Hyperactivity Disorder in family functioning and parenting stress. *Journal of Research in Psychopathology*, (inpress). doi:10.22098/JRP.2023.12237.1157

Nigg JT. (2006). *What causes ADHD? Understanding what goes wrong and why*. New York: The Guilford Press. https://www.scirp.org/reference/referencespapers?referenceid=3295259

Ogrim, G., Kropotov, J., & Hestad, K. (2012). The quantitative EEG theta/beta ratio in attention deficit/hyperactivity disorder and normal controls: sensitivity, specificity, and behavioral correlates, *Psychiatry Res*, *198*, 482-488. doi:10.1016/j.psychres.2011.12.041

Poil, S.S., Bollmann, S., Ghisleni, C., O'Gorman, R. L., Klaver, P., Ball, J., . . . Michels, L. (2014). Age dependent electroencephalographic changes in attention-deficit/hyperactivity disorder (ADHD). *Clinical Neurophysiology*, *125*, 1626-1638. Retrieved from: doi:10.1016/j. clinph.2013.12.118

Pop-Jordanova, N. (2012). QEEG characteristics and biofeedback modalities in children with ADHD. *Current directions in ADHD and its treatment*, J.M. Norvilitis (Ed.), ISBN: 978-953-307-868-7, InTech, Available from: http://www.intechopen.com/books/current-directions-in-adhd-and-its-treatment/qeegcharacteristics-and-biofeedback-modalities-in-children-with-adhd

Raeisi Sadati, R., Pourbeyrami Hir, Y., & Narimani , M. (in press). Investigating the Effectiveness of Exposure to Green Space on Absolute Power of Alpha Wave and Stress Reduction In peopleWith AttentionDeficit Hyperactivity Disorder. *Journal of Research in Psychopathology* doi: 10.22098/JRP.2024.14911.1230

Russell, V., de Villiers, A., Sagvolden, T., Lamm, M., & Taljaard, J. (1995). Altered dopaminergic function in the prefrontal cortex, nucleus accumbens and caudate-putamen of an animal model of attention-deficit hyperactivity disorder—the spontaneously hypertensive rat. *Brain Res*, *676*, 343-451. doi: 10.1016/0006-8993(95)00135-d

Saad, J.F., Kohn, M.R., Clarke, S., Lagopoulos, J., & Hermens, D.F. (2015). Is the Theta/Beta EEG Marker for ADHD Inherently Flawed? *Journal of Attention Disorders*, 1-12. doi: 10.1177/1087054715578270

Satterfield, J., Cantwell, D., Lesser, M., & Podosin, R. (1972). Physiological studies of the hyperkinetic child: 1. *Am J Psychiatry*, *128*, 103-108. doi: 10.1176/ajp.128.11.1418

Sanchis, J., García-Ponsoda, S., Teruel, M. A., Trujillo, J., & Song, I. Y. (2024). A novel approach to identify the brain regions that best classify ADHD by means of EEG and deep learning. Heliyon, 10(4), e26028. doi:10.1016/j.heliyon.2024.e26028

Sawyer, M.G., Whaites, L., Rey, J.M., Hazell, P.L., Graetz, B.W., & Baghurst, P. (2002). Health-related quality of life of children and adolescents with mental disorders. J. Am. Acad. Child Adolesc. Psychiatry, 41, 530-537. doi:10.1097/00004583-200205000-00010

Sibley, M.H., Pelham,W. E., Molina, B.S., Gnagy, E. M.,Waschbusch, D. A., Biswas, A., et al. (2011). The delinquency outcomes of boys with ADHD with and without comorbidity. *Journal of Abnormal Child Psychology*, *39*, 21-32. doi:10.1007/s10802-010-9443-9

Simkin, D.R., Thatcher, R.W., & Lubar, J. (2014). Quantitative EEG and Neurofeedback in Children and Adolescents Anxiety Disorders, Depressive Disorders, Comorbid Addiction and Attention-deficit/Hyperactivity Disorder, and Brain Injury. *Child Adolesc Psychiatric Clin N Am*, *23*, 427-464. doi10.1016/j.chc.2014.03.001

Snyder, S.M., & Hall, J.R. (2006). A meta-analysis of Quantitative EEG power associated with Attention-Deficit Hyperactivity Disorder. *Journal of Clinical Neurophysiology*, *23* (5), 441-456. doi: 10.1097/01.wnp.0000221363.12503.78

Snyder, S.M., Quintana, H., Sexson, S.B., Knott, P., Haque, A.F., & Reynolds, D.A. (2008). Blinded, multi-center validation of EEG and rating scales in identifying ADHD within a clinical sample. *Psychiatry Res*, *30*, 346-538. doi:10. 1016/j.psychres.2007.05.006

Swartwood, M.O., Swartwood, J.N., Lubar, J.F., Timmermann, D.L., Zimmerman, A.W., & Muenchen, R.A. (1998). Methylphenidate effects on EEG, behavior and performance in boys with ADHD. *Pediatric Neurology*, *18*, 244-50. doi: 10.1016/s0887-8994(97)00205-1

Swanson, J.M., & Castellanos, F.X. (2002). Biological bases of ADHD: Neuroanatomy, genetics, and pathophysiology. Attention-deficit hyperactivity disorder: state of the science, best practices. *Civic Research Institute*. ttps://psycnet.apa.org/record/2003-00785-007

Swanson, J., Arnold, L.E., Kraemer, H., Hechtman, L., Molina, B., Hinshaw, S., et al. (2008). Evidence, interpretation and qualification from multiple reports of long-term outcomes in the multimodal treatment study of children with ADHD (MTA): part I: executive summary. *J. Atten. Disord*. 12, 4-14. doi:10. 1177/1087054708319345

Tabiee, M., Azhdarloo, A., & Azhdarloo, M. (2023). Comparing executive functions in children with attention deficit hyperactivity disorder with or without reading disability: A resting-state EEG study. Brain and behavior, 13(4), e2951. Doi:10.1002/brb3.2951

Taylor, E., Döpfner, M., Sergeant, J., Asherson, P., Banaschewski, T., Buitelaar, J., et al. (2004). European clinical guidelines for hyperkinetic disorder- first upgrade. *Eur. Child Adolesc. Psychiatry* 13 (Suppl. 1), I7-I30.doi:10. 1007/s00787-004-1002-x

Thompson, A.L., Molina, B.S., Pelham,W., & Gnagy, E.M. (2007). Risky driving in adolescents and young adults with childhood ADHD. *Journal of Pediatric Psychology*, *32*, 745-759. doi:10.1093/jpepsy/jsm002

Thorley, G. (1984) Hyperkinetic syndrome of childhood: clinical characteristics. *Br J Psychiatry*, *144*, 16-24. doi:: 10.1192/bjp.144.1.16

Ucles, P., & Lorente, S. (1996). Electrophysiologic measures of delayed maturation in attention-deficit hyperactivity disorder. *J Child Neurol 11*, 155-156. doi:: 10.1177/088307389601100216

Valo, S., & Tannock, R. (2010). Diagnostic instability of DSM-IV ADHD subtypes: Effects of informant source, instrumentation, and methods for combining symptom reports. Journal of Clinical Child and Adolescent Psychology, 39(6), 749-760. doi:10.1080/15374416.2010.517172

Vojnits, B., Magyar, T. Z., Szalárdy, O., Reicher, V., Takács, M., Bunford, N., & Bódizs, R. (2024). Mobile sleep EEG suggests delayed brain maturation in adolescents with ADHD: A focus on oscillatory spindle frequency. Research in developmental disabilities, 146, 104693. doi:10.1016/j.ridd.2024.104693

Wender, P.H. (1973). Some speculations concerning a possible biochemical basis of minimal brain dysfunction. *Ann NY Acad Sci*, *205*, 28-81. doi:: 10.1111/j.1749-6632.1973.tb43159.x

White, JN., Jr, Lubar, J.F., & Hutchens, T.A. (2000). Neuropsychological and QEEG assessment of adult ADHD. Archives of Clinical Neuropsychology 15, 653-850. doi:10.1093/arclin/15.8.689a