ADITUM
Enrich your Research

Neurosurgery and Neurology Research

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 Mahmood alilou [3], & Naimeh Mashinchi Abbasi[4*]
[1]Ph.D., Professor, Psychology, Faculty of Educational Sciences and Psychology, University of Tabriz.
[2]Ph.D., Professor, Psychology, Faculty of Educational Sciences and Psychology, University of Tabriz.
[3]Ph.D., Professor, Clinical Psychology, Faculty of Educational Sciences and Psychology, University of Tabriz.
[4]Ph.D student., Clinical Psychology, Faculty of Educational Sciences and Psychology, University of Tabriz.

Article Info

Received: February 14, 2024
Accepted: February 24, 2024
Published: February 29, 2024

***Corresponding author:** Naimeh Mashinchi Abbasi, Ph.D student., Clinical Psychology, Faculty of Educational Sciences and Psychology, University of Tabriz.

Citation: Hashemi T, Khanjani Z, Majid M alilou, Naimeh M Abbasi, (2024) "Theta/Beta Ratio or Not? A Review Study of Specified QEEG Parameter for Diagnosis of ADHD Presentations". J Neurosurgery and Neurology Research, 6(1). DOI: 10.61148/2836-2829/NNR/048.

Abstract:

Attention deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder. It is characterized by difficulties in allocating and sustaining attention, impulsivity and hyperactivity According to the DSM-5, three presentations of ADHD are described: combined, predominantly inattentive, and predominantly hyperactive/impulsive. Theta-beta ratio (TBR) or inattention index, which is referred to 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. The present study aims to review the literature on QEEG parameters related to ADHD. Due to the increased theta, TBR is reported by many investigators as a consistent characteristic of ADHD. However, it is not the diagnostic measure for all individuals with ADHD. TBR is unnecessary in making the diagnosis for all ADHD presentations

In other words, review of studies suggests that 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.

Key words: ADHD; TBR; ADHD presentations; QEEG; neuropsychology; diagnostic measure

1. Introduction

1.1 Description of ADHD

Attention deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder (American Psychiatric Association, 2013) commonly managed by pediatricians (McDonald & Jalbert, 2013). As defined in the DSM-5, it occurs before the age of 12 when inadequate levels rather than expected developmental levels of attention and hyperactivity–impulsivity appear 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, 2000; Gonen-Yaacovi et al, 2016).

ADHD is associated with impairment in daily activities, academic performance, peer relations 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).

1.2: ADHD: Presentations based on DSM-5

According to the DSM-5, three presentations of ADHD could be described: combined, predominantly inattentive, and predominantly hyperactive/impulsive (American Psychiatric Association, 2013).

These presentations are based on different clusters of symptoms from within the two core symptom domains of inattention and hyperactivity/impulsivity.

Difficulties with inattention or hyperactivity and impulsivity as the core symptoms of ADHD are 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 manifold consequences and the frequency of ADHD, there is a surprising dearth of data on 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 indicate 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) and features in the inattentive and combined presentations of ADHD under DSM-5 (APA, 2013; Brace, Kraev, Rostron, Stewart, Overton, & Dommett, 2015).

As another presentation of ADHD, a person with attention deficit disorder or predominantly inattentive subtype (ADHD-I) often avoids, dislikes, or does not want to do things that take a lot of mental effort for a long period of time. He/she is often easily distracted, usually has trouble keeping attention on tasks or plays activities and frequently switches from one activity (mental or physical) to another (Nigg, 2006).

Based on DSM-5 criteria for the diagnosis of individuals with ADHD-H, they often fidgets with or taps hands or feet or squirms in seat, leave seat in situations when remaining seated is expected. Besides, they often run about or climb in situations where it is inappropriate. They 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. In addition, they interrupt or intrude on others (APA, 2013).

Moreover, The National Institute of Mental Health (NIMH) is the National Institutes of Health's (NIH) lead agency in the research on ADHD. Researchers at National Institute of Mental Health (NIMH), have suggested that individuals expressing hyperactivity in many cases are noted to be excessively fidgety, restless, and “on the go.” ADHD-H is distinguished by maladaptive levels of hyperactivity-impulsivity but not inattention. Individuals with ADHD-H exhibit excessive movement 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 defined as an expression of dysfunctional behavioral inhibition and is often illustrated 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 has been defined as a predisposition toward rapid, unplanned reactions to internal or external stimuli, without regard to the negative consequences (Moeller, Barratt, Dougherty, Schmitz, Swann, 2001; Feki, Moalla, Baati, Trigui, Sellami, & Masmoudi, 2016).

As defined by 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 later 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 characteristics of impulsivity, as well.

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

1.3: ADHD: Prevalence and etiology

ADHD occurs in about 5% of children and about 2.5% of adult population (American Psychiatric Association, 2013). The disorder affects a child’s academic performance, social competence, occupational choices and personality formation, and its negative impacts extend 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 recognized; however, it is thought to be the result of a complex interaction between the neuroanatomical system and neurobiochemistry.

Furthermore, genetic factors, neurodevelopmental factors, psychosocial factors, 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 based on evidence from cognitive investigations showing that ADHD is related to deficits in so- called lower cognitive mechanisms (Bedard, Trampush, Newcorn, & Halperin, 2010; Halperin, Trampush, Miller, Marks, & Newcorn, 2008).

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

Generally, an increased number of findings suggest that ADHD is a disease of the brain (Swanson & Castellanos, 2002). A growing body of research has been 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 many 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, but also clinically, in the assessment, diagnosis (González-Castro, Rodríguez, López, Cueli, & Alvarez, 2013) and treatment of ADHD.

1.3.1: Electroencephalography

During the last few years the situation is slowly changing. Now we are facing the renascence of EEG. This renascence is associated with appearance of new methods in human EEG assessment and new experimental findings in animal research which allowed 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 correspondence between intracranial electrical currents and the resulting voltages on the scalp reflecting certain facets of brain electrical function and processing, such as how electrically active various brain regions are and how responsive they may be to stimuli or during 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 contributed significantly to illuminate 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 the ability of a network 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 (Nazari, 2008). Accordingly, developments in digitization and analytical techniques of QEEG technology have greatly facilitated the use of brain electrical activity data in clinical and research settings (Snyder & Hall, 2006). In other words, results have demonstrated that QEEG results could be used to discriminate between children with and without ADHD.

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

Recently, a QEEG spectrum classification of ADHD population has been proposed defining four main subtypes: I subtype (abnormal increase of delta-theta frequency range centrally or centrally-frontally), II subtype (abnormal increase of frontal midline theta rhythm), III subtype (abnormal increase of beta activity frontally), and IV subtype (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).

1.3.2. Evidence for Utility of QEEG in Diagnosis of ADHD

EEG plays an important role in evaluating, classifying and following disorders. The EEG is a widely accepted method for evaluating cortical information processing and neurophysiologic changes that occur during unconsciousness and varying 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 can be regarded as a specific sign of brain dysfunction (Kanda et al., 2009).

It is critical to learn 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 pathognomonic to nearly all childhood psychiatric disorders, it is often difficult to make differential diagnosis between ADHD and other disorders that could have a similar presentation, 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 have been considered as a promising biomarker for ADHD (Loo & Makeig, 2012).

Quantitative EEG (QEEG) can help reveal the underlying neurophysiology associated with the symptoms of Attention Deficit Hyperactivity Disorder (ADHD) and learning disorders, and help differentiate these disorders from other comorbidities. Excess slow wave activity is the most common abnormal finding in children with attention disorders with the thalamocortical and/or septal-hippocampal pathways most likely to be dysfunctional. The QEEG can play an important role in the evaluation and treatment of these 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 those of increased absolute power in Theta (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 ADHD children can be differentiated from non ADHD in over 96% of cases on the basis of their QEEG signatures. QEEG helps to differentiate the neurological underpinning of attention deficits arising from ADHD, from the neurological underpinning arising from attention deficits associated with other primary psychiatric disorders: depression, anxiety, obsessive compulsive disorder, oppositional defiant disorder or psychosis (Duff, 2002).

Over the last years, EEG research has found group differences between children with or without ADHD. 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) ratios have also been found in children with ADHD compared to normal children.

Studies investigating whether EEG can discriminate among ADHD, learning disorders, and other psychiatric disorders, have indicated that EEG was sensitive (93%-97%) and fairly specific (84%-90%) in differentiating ADHD from LD (Loo & Barkley, 2005).

Quantitative electroencephalogram (QEEG) studies in children with learning disabilities have shown alterations, such as an increase in the absolute power in the delta and theta 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 (accuracy 89% to 94%) as compared to a clinical evaluation. The AAN recommends that the EEG test should not be used in place of a standard clinical evaluation, because of the risks of misdiagnosis of 6-15% 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.

1.4. : QEEG Findings in ADHD
1.4.1 : Theta/Beta Ratio (TBR)

The most common approach of QEEG research has been to determine the absolute and relative power derived from 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 for ADHD children with controls. He 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 low 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).

Studies have indicated that increased theta/beta ratio (TBR) to be a sensitive marker of ADHD (Monastra, Lubar, & Linden, 2001) and correlates 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 by EEG recording at a single place Cz in reference to linked ears. It was found that this index is three times higher in inattentive and combine types of ADHD children at the age of 6-10 years compared with normal group. Monastra et al. (2001) found that the sensitivity of this index was 86% and his specificity is 98%.

Most of the media 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.

Regarding that some authors found a positive association between ADHD and higher Theta/Beta ratio (Clarke et al., 2011). Therefore, most NF protocols for ADHD treatment aim at increasing faster Beta frequencies, especially SMR, and decrease Theta waves (Moriyama, Polanczyk, Caye, Banaschewski, & Brandeis, & Rohde, 2012).

In theta / beta training, patients learn 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).

In previous studies, ADHD has been consistently characterized 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 in indicated an increase in the theta/beta ratio had 87% sensitivity, 94% specificity, and 89% accuracy for ADHD diagnosis, whereas the rating scale exhibited 47–58% accuracy (Delorme & Makeig, 2004; Mizuhara, Wang, Kobayashi, & Yamaguchi, 2004).

1.4.2: Theta/Beta Ratio (TBR) is not the only measure for Diagnosis of ADHD

Findings in QEEG studies 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 discriminating between children with and without ADHD (Liechti et al., 2013; Ogrim, J. Kropotov, K. Hestad, 2012). Heinrich, Busch, Studer, Erbe, Moll and Kratz (2014) have pointed out that recent studies question 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 that there was no correlation between QEEG ADHD parameter (theta/beta ratio) and the Coolidge Personality and Neuropsychology Inventory (CPNI) ADHD scale (Coolidge, Starkey, & Cahill, 2007).

Studies conducted by Liechti et al (2013) as well as Buyck and Wiersema (2014) did not find differences between children with

ADHD and typically developing children in any frequency band considered.

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. Results indicated that nine studies were identified with a total of 1253 children/adolescents with and 517 without ADHD. The grand-mean effect size (ES) for the 6-13 year-olds was 0.75 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 found 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 of ADHD, however a substantial sub-group of ADHD patients do deviate on this measure and TBR has prognostic value in this sub-group, warranting its use as a prognostic measure rather than a 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. Any differences in theta for children with and without ADHD were not obtained. 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, since in most of the studies, resting EEG has been investigated.

Hence, an increased TBR cannot be considered a reliable measure used for the diagnosis of ADHD at this time. 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 good measure only for a part of ADHD population. Mapping this index in normal population showed that the location of the maximum of this index 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 position depending on age.

As Hagemann et al (2005) have suggested it appears to be more relevant to consider the EEG during task processing though it has to be kept in mind that the resting EEG does not only reflect a trait 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 obtained 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 prominent when considering 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, it would be a great idea to consider specified EEG measures for each presentation; especially it would be important in treatment protocols based on EEGbiofeedback.

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/particularly pronounced at a certain electrode. Looking at single electrodes, effects at electrode Cz appeared rather smaller than larger compared to frontal, electrodes (F3, Fz).

Consequently, considering restingEEG, EEG during cognitive task, ADHD different presentations, as well as electrode site are of a great importance. Not all of ADHD subgroups could be

characterized by TBR, and electrode Cz could 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.

2. Conclusion

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

TBR due to increased theta is reported by many investigators as a consistent characteristic of ADHD. Some groups recommend using the TBR during eyes-opened or eye-closed resting condition as an add-on 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 be a nonspecific marker of cortical dysfunction common to other disorders such as epilepsy, bipolar disorder, and polysubstance abuse

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

They have found a strong decline in ES for the TBR across years. They stated that this effect was mainly related to an increase in TBR for control groups and not related to a decrease in TBR for ADHD groups.

Arns and Gordon (2014) have proposed that the Theta/Beta ratio is not an unambiguous diagnostic marker in all cases, and it seems unlikely that a single biomarker can differentiate all ADHD patients from controls.

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.

Therefore, review of the studies suggests that EEG plays an important role in evaluating, clas- sifying and following disorders. Accordingly, QEEG abnormal patterns can be regarded as a specific sign of brain dysfunction. Based on the results previously reviewed, EEG measures have been regarded as a promising biomarker 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 could be proposed that TBR is unnecessary in making the diagnosis for 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.

Acknowledgements: None

References

1. AAN. Clinical Practice Guideline Process Manual. Ed. St Paul, MN: Am. *Acad. of Neurology*
2. Ahn, H., Prichep, L., John, E.R., Baird, H., Trepetin, M., & Kaye, H. (1980). Developmental equations reflect brain dysfunctions. *Science, 210*, 1259-1262.
3. 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.
4. American Psychiatric Association. (2000). DSM-IV-TR (American P.). Washington, DC.
5. 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.
6. 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.
7. American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Washington, DC: APA.
8. 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.
9. 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
10. Arns, M., & Gordon, E. (2014). Quantitative EEG (QEEG) in psychiatry: Diagnostic or prognostic use? *Clinical Neurophysiology, 125*, 1504-1506.
11. Bahçivan Saydam, R., Ayvaşik, H.B., & Alyanak, B. (2015). Executive functioning in subtypes of Attention Deficit Hyperactivity Disorder. *Arch Neuropsychiatr, 52*, 386-392.
12. 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.

13. Barkley R.A. (1997). Behavioral inhibition, sustained attention, and executive function: Constructing a unified theory of ADHD. *Psychol Bull, 121*, 65-94.
14. 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.
15. 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.
16. 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.
17. 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.
18. Biederman, J. (2005). Attention-deficit/hyperactivity disorder: a selective overview. *Biol Psychiatry, 57*(11), 1215-1220.
19. Biederman, J., & Faraone, S. (2006). The effects of attention-deficit hyperactivity disorder on employment and household income. *Medscape General Medicine, 8*, 12.
20. 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.
21. 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.
22. 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.
23. Bresnahan, S.M., & Barry, R.J. (2002). Specificity of quantitative EEG analysis in adults with attention deficit hyperactivity disorder. *Psychiatry Research*, *112*, 133-144.
24. 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
25. Buzsaki, G. (2016). EEG rhythms. *In Quantitative EEG, Event-Related Potentials and neurotherapy.*

26. Byring, R., Salmi, T.K., Sainio, K.O., & Örn, H.P. (1991). EEG in children with spelling disabilities. *Electroencephalogr Clin Neurophysiol, 79*, 247-255.

27. 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.

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

29. 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.

30. 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.

31. 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.

32. 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.

33. 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.

34. 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.

35. Cortese, S., & Vincenzi, B. (2012). Obesity and ADHD: Clinical and neurobiological implications. *Current Topics in Behavioral Neurosciences, 9*, 199–218. http://dx.doi.org/10.1007/7854_2011_154.

36. 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.

37. Cortese, S., & Castellanos, F.X. (2015). Attention Deficit/Hyperactivity Disorder. Neurobiology of Brain Disorders, 42-58.

38. 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.

39. 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.

40. 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.

41. Del Campo N, Muller U and Sahakian BJ (2012) Neural and behavioral endophenotypes in ADHD. *Curr Top Behav Neurosci* 11, 65-91.

42. 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.

43. 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.

44. Douglas, V. (1983). In M,R. (ed) *Developmental Neuropsychiatry*. Guildford Press, New York,pp. 280-329

45. Duff, J. (2002). QEEG Neurometrics and differential diagnosis of ADHD. *Behavioural Neurotherapy Clinic*, 1-3.

46. 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.

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

48. 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.

49. 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.

50. 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.

51. 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.

52. 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.

53. 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

54. 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.

55. 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. http://dx.doi.org/10.1016/S1697-2600(13)70013-9

56. 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

57. 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.

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

59. 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

60. 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.

61. 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.

62. 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.

63. 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.

64. Ikarashi, Y., Takahashi, A., Ishimaru, H., Arai, T., & Maruyama, Y. (1997). Suppression of cholinergic activity via the dopamine D2 receptor in the rat striatum. *Neurochemistry International, 30*, 191-7.

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

66. 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
67. 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.
68. Klimesch, W. (1999). EEG alpha and theta oscillations reflect cognitive and memory performance: A review and analysis. *Brain Research Reviews*, *29*, 169-195.
69. 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:10.1097/00004583-199608000-0001
70. 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.
71. Liechti, M.D., Valko, L., Müller, U.C., D◌̈, hnert, 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.
72. Loo, S.K,, & Barkley, R.A. (2005). Clinical utility of EEG in attention deficit hyperactivity disorder. *Applied Neuropsychology*, *12*, 64-76.
73. Loo, S.K., & Makeig, S. (2012). Clinical utility of EEG in attention-deficit/hyperactivity disorder: A research update. *Neurotherapeutics*, *9*, 569-587.
74. 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.
75. 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.
76. 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.
77. 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.
78. Marcus, S.D., Fox, D., & Brown, D. (1982). Identifying school children with behavior disorders.*Community Mental Health Journal*, *18*(4), 249–256. http://dx.doi.org/10.1007/BF00754539.
79. 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.
80. Matthis, P., Scheffner, D., & Benninger, C. (1981). Spectral analysis of the EEG: Comparison of various spectral parameters. *Electroencephalic Clinical Neurophysiology*, *52*, 218–221.
81. 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.
82. 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. http://dx.doi.org/10.1348/000709901158389
83. 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.
84. 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.
85. 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.
86. 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.
87. 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.
88. 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.
89. Nazari M.A. (2008). Electroencephalographic characteristic of children with attention deficit/hyperactivity disorder and evaluation of the effects of neurofeedback on attentional functions. *Ph.D. dissertation, University of Picardie Jules Verne, Amiens, France*.
90. Nigg JT. (2006). *What causes ADHD? Understanding what goes wrong and why*. New York: The Guilford Press.
91. 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.
92. Onnink, A.M.H., Zwiers, M.P., Hoogman, M., Mostert, J.C., Dammers, J., Kan, C.C., Vasquez, A.A., Schene, A.H., & Buitelaar, J., Franke, B. (2015). Deviant white matter structure in adults with attention-deficit/hyperactivity

disorder points to aberrant myelination and affects neuropsychological performance. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 63*, 14-22.

93. Patros, C.H.G., Alderson, R.M., Kasper, L.J., Tarle, S.J., Lea, S.E., Hudec, K.L. (2016). Choice- impulsivity in children and adolescents with attention-deficit/hyperactivity disorder (ADHD): A meta-analytic review. *Clinical Psychology Review,43*, 162-174.
94. 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.
95. 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,
96. 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.
97. 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.
98. Satterfield, J., Cantwell, D., Lesser, M., & Podosin, R. (1972). Physiological studies of the hyperkinetic child: 1. *Am J Psychiatry*, *128*, 103-108.
99. 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.
100. 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.
101. 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
102. 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.
103. 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.
104. 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.
105. 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*.
106. 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.
107. Visser, S.N., Bitsko, R.H., Danielson, M.L., Ghandour, R.M., Blumberg, S.J., Schieve, L.A., Holbrook, J.R., & Wolraich, M.L., & Cuffe, S.P. (2015). Treatment of Attention Deficit/Hyperactivity Disorder among children with special health care needs. *The Journal of Pediatrics*.
108. 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.
109. 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.
110. Thorley, G. (1984) Hyperkinetic syndrome of childhood: clinical characteristics. *Br J Psychiatry*, *144*, 16-24.
111. Ucles, P., & Lorente, S. (1996). Electrophysiologic measures of delayed maturation in attention- deficit hyperactivity disorder. *J Child Neurol 11*, 155-156.
112. 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.
113. Wender, P.H. (1973). Some speculations concerning a possible biochemical basis of minimal brain dysfunction. *Ann NY Acad Sci*, *205*, 28-81.
114. White, JN., Jr, Lubar, J.F., & Hutchens, T.A. (2000). Neuropsychological and QEEG assessment of adult ADHD. *Archives of Clinical Neuropsychology* 15, 653-850.