Emerging Treatments for Children with Attention-Deficit/Hyperactivity Disorder

by

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Chapter I: Introduction

Overview of ADHD and Rationale for Study

Attention deficit/hyperactivity disorder (ADHD) is the most prevalent neurodevelopmental disorder, affecting approximately 8% of children worldwide (Bunford, Evans, & Wymbs, 2015). ADHD was once considered a disorder of childhood only, but is now known to have a chronic course with symptoms that shift in severity across the lifespan (Barkley, 2015). ADHD presents heterogeneously with symptoms varying in severity and functional impact. For this reason, the American Academy of Pediatrics (AAP) recommends a multi-faceted treatment approach, with academic, family, and social supports provided as necessary (AAP, 2011). The variety of clinical presentations necessitates ongoing research into emerging treatments that may provide additional options for helping children and families struggling with the disorder. In this analytic dissertation I review the origins of ADHD in the historical and scientific literature, and provide a synopsis of the evolution of theory and diagnosis. In addition, I outline current understanding regarding the etiology of ADHD and associated comorbidities. Lastly, I investigate the efficacy of three emerging treatments that may provide additional therapeutic options for children with the disorder: neurofeedback, executive function training, and self-regulation strategies.

The etiology of ADHD is complex with studies showing contributions from multiple sources including a strong genetic link (Banaschewski, Becker, Scherag, Franke, & Coghill, 2010). Other potential contributors include brain injury, trauma/neglect, and exposure to environmental toxins (Barkley, 2015; Karpouzis & Bonello, 2012). MRI studies identify structural brain abnormalities that may contribute to symptoms, as well. Children with ADHD show decreased global brain volume especially in the frontal regions, which may contribute to

difficulties with sustaining attention (Okie, 2006). Likewise, children with the disorder show decreased cortical folding throughout the cerebral cortex, which is associated with impairments in executive function and learning deficits (Wolosin, Richardson, Hennessey, Denckla, & Mostofsky, 2009).

Symptoms of ADHD include a persistent pattern of behaviors stemming from inattention, hyperactivity, and impulsivity (*Diagnostic and Statistical Manual of Mental Disorders* 5th ed.; *DSM-5*; American Psychiatric Association [APA], 2013). Inattention creates impairments in children's ability to organize and complete multi-step tasks, including schoolwork and personal responsibilities. Hyperactivity impacts children's ability to control energy levels, and often presents as restlessness or the inability to control motor activity. Impulsivity presents as behaviors lacking forethought with regard to consequences or the impact on others, such as interrupting, grabbing, or violating the personal space of others (APA, 2013). The symptoms of ADHD impact multiple functional domains (school, family, social) and can range in severity from mild to severe (Tarver, Daley, & Sayal, 2014). Milder versions of the disorder, or *subthreshold ADHD*, may represent either a stable presentation or a developmental period where symptoms decrease in severity (Balázs & Keresztény, 2014). More recently, Barkley (2015) conceptualized ADHD as a disorder of both executive functioning and self-regulation (Barkley, 2015). Executive functioning deficits include difficulties with working memory, cognitive flexibility, and goal-directed behavior (Rapport, Orban, Kofler, & Friedman, 2013). Self-regulation impairments present as difficulty inhibiting emotional and behavioral response, and as externalizing behaviors such as aggression and low frustration tolerance (Sullivan et al., 2015).

Diagnosis of ADHD is based on assessment, which includes direct observation, clinical interview, and the use of instruments such as behavioral rating scales and parent/teacher

questionnaires (AAP, 2011). Clinicians attempt to collect information regarding symptoms in multiple domains because the *DSM* requires that symptoms present before the age of 12 and create functional impairment in more that one environment (APA, 2013). ADHD evaluation can be complicated due to high comorbidity rates with other childhood disorders. Children with ADHD have higher rates of both internalizing (mood, anxiety, depression) and externalizing (conduct, oppositional) disorders (Armstrong, Lycett, Hiscock, Care, & Sciberras, 2015). In addition, these children show increased levels of learning problems, such as dyslexia and communication disorders (Barkley, 2015). Children with ADHD also have higher than average levels of negative health outcomes such as physical injuries, substance use, and childhood obesity (Bunford et al., 2015; Nigg, 2013).

The variety of clinical presentations and comorbidities create challenges with regard to treatment of the disorder (Armstrong et al., 2015). AAP and the Centers for Disease Control and Prevention (CDC) provide treatment guidelines for children ages 4-18 years (AAP, 2011; CDC 2016). Both agencies advise using behavior therapy for children 4-5 years old, with federal drug administration (FDA) approved medication (usually methylphenidate) recommended in cases where behavioral interventions are ineffective (AAP, 2011; CDC, 2016). For older children, the agencies suggest a combination of medication and parent- or teacher-based behavioral therapy, depending on the environment where primary symptoms occur (AAP, 2011; CDC, 2016).

National guidelines provide a general treatment strategy for parents and clinicians, but do not give treatment recommendations for children who cannot tolerate medications or do not have access to behavioral therapy (Sonuga-Barke et al., 2013). Effective treatment of children with ADHD necessitates a multimodal approach with as many options for symptom reduction as possible (Cerrillo-Urbina et al., 2015). Emerging treatments such as neurofeedback, executive

function training, and self-regulation strategies hold the potential for providing additional avenues to increase functioning and improve quality of life for children with this disorder (Chacko et al., 2014; Vollebregt, Dongen-Boomsma, Buitelaar, & Slaats-Willemse, 2014).

Chapter II: Methodology

Data Collection

Because this review serves the dual purpose of presenting both a synopsis of current theory on ADHD (etiology, functional impairments, comorbidities) and a review of three emerging treatments, the research contains a variety of sources. I obtained information on the history and evolution of the disorder from journal articles dating back to 1998 and also from Russell Barkley's 2015 comprehensive, 900-page volume on the history, diagnosis, and treatment of the disorder (Barkley, 2015). I chose articles examining current theory on etiology such as genetics, brain trauma, and neurobiology from a somewhat narrower timeframe (approximately the last 10 years) in order to focus on theory based in current research. Articles investigating the three emerging treatments (neurofeedback, executive function treatments, self-regulation strategies) were mostly published within the last five years.

Inclusion and Exclusion Criteria for Research Articles

Data sources for this study included both qualitative and quantitative reviews presented as theoretical reviews, meta-analyses, and experimental trials (in multiple formats i.e. RCT, wait-list controlled, placebo, sham, etc.). Search databases included PubMed, EBSCO host, Google Scholar, LIRN, CDC, NIMH, and PsycINFO. For articles pertaining to emerging treatments, search filters included only peer-reviewed articles. For diagnostic, assessment, and historical data, inclusion criteria incorporated some non peer-reviewed articles that presented information relevant to the foundations of the disorder. This review cites a total of 85 (mostly peer-

reviewed) articles. I have also referenced four texts by authors whose research contributes relevant theoretical and historical data pertaining to ADHD and the neurobiology of child development, along with five editions of the *DSM*. In addition, this review sites five websites providing clinical guidelines for the treatment of ADHD (AAP, BCIA, CDC, SAMHSA [1], SAMHSA [2]).

Search criteria used the base terms "ADHD" or "attention deficit hyperactivity disorder" with additional limits that followed the content outline e.g. *comorbidity, neurobiology, emerging treatments, etc.* Reference lists of relevant research papers provided additional sources for this review. ADHD studies falling outside the specific scope of this review—emerging treatments for children with ADHD—were not included. This review also excluded studies on specific demographic groups such as adults with ADHD, and specific clinical populations such as runaway teens with ADHD, due to limited generalizability of research results.

Efficacy of Emerging Treatments

Research on emerging therapies for ADHD is limited in quantity for some topics (e.g. executive function treatments). Therefore, studies included in this review contain a variety of experimental approaches with varying levels of methodological rigor. In the articles I surveyed, outcome data were not presented in a consistent format that would allow for direct efficacy comparisons. However, each study did contain a conclusion by the author(s) regarding the effectiveness of the treatment investigated. The most common method for presenting efficacy data was effect size (ES) using Cohen's *d*, with specific values set for small, medium, and large ES correlating to the amount of change produce between pre- and post-test on outcome measures. However ES was not reported in some studies; thus, this review does not include a

comparative analysis of outcome data. Instead, I present a qualitative summary of the rationale, procedures, and reported efficacy of the emerging treatments, with outcome data such as effect size included when available.

Chapter III: Literature Review

History of the Disorder

Early theorists. Symptoms of the core components of ADHD—i.e., inattention, hyperactivity, and impulsivity (including emotional dysregulation)—appear in literature as far back as Shakespeare, who described attentional difficulties, poor impulse control, and hyperactivity in *King Henry VIII* (Shakespeare, trans. 1993). However, the first appearances in actual medical literature do not begin to appear until the late 1700s (Palmer & Finger 2001). A medical textbook initially published anonymously, but later ascribed to German physician Melchior Adam Weikard in 1775, identified both adults and children with the core characteristics of ADHD—inattention, hyperactivity, impulsivity, and lacking in persistence (as cited in Barkley & Peters, 2012). Even in the earliest clinical observations of these symptoms, Weikard seemed to understand the complex etiology of the problem at hand—a complexity that the current research highlights—suggesting that either poor child-rearing or biological predispositions may produce the disorder (as cited in Barkley & Peters, 2012).

Sir Alexander Crichton (1763-1856) also provided some of the first documentations of symptoms of mental disorders, including ADHD (Crichton, 1798). A Scottish physician who practiced in Paris, Stuttgart, and Vienna, Crichton took special interest in cases of various mental illness. In 1798 he published three volumes of observations that approached mental illness from a medical or physiological perspective, a practice that was uncommon at the time. In the second chapter of Book II, titled *On Attention and its Diseases,* Crichton explored the issue of sustained

attention, providing clear definition and noting both the naturally-occurring range of attentional abilities between individuals and within people at differing times (Crichton, 1798). Crichton also noted that inattention did not necessarily mean dysfunction, and pointed out that many factors may cause a disruption in healthy attention, such as stimulating events or one's own volition (Crichton, 1798).

Crichton distilled the product of inattention down to two distinct observations: first, that the person possessed the inability to attend with a degree of constancy to any single object; and second, that this inability to attend arose from what he described as "sensibility in the nerves" (p. 312) which inhibited the individual from consistently attending, and which could be either inborn or the product of disease. Crichton speculated that if the cause were inborn, it would significantly impact the child's education and would be evident very early in his or her life. He also noted the tendency for attention deficits to decrease as the child aged (Crichton, 1798). These early descriptions of the symptoms of inattention and of their etiology and course foreshadowed more recent discoveries regarding ADHD symptoms, their impact on school functioning, and the fact that up to 50% of children outgrow a significant number of their symptoms by adolescence (DuPaul, Weyandt, & Janusis, 2011). Crichton's observations share many similarities with current diagnostic descriptions found in *DSM-5* (APA, 2013). However, Crichton did not specifically address hyperactivity or impulsivity in his writings. He did refer to extreme emotional reactivity, low frustration tolerance, and severe mental restlessness as co-occurring symptoms with the inattention he observed; but he only made reference to this as a product of the mental excitement created by the individual's constant shifts in focus (Crichton, 1798). It is possible that Crichton observed only the inattentive subtype of ADHD, or that he simply did not differentiate issues with attention from hyperactivity and impulsivity as the *DSM*

does today. It is also possible that Crichton observed patients suffering from conditions other than ADHD, such as metabolic issues, epilepsy or head trauma (Lange, Reichl, Lange, Tucha, & Tucha, 2010). In any case, Crichton's observations provide the beginnings of over two centuries of exploration into conditions containing the basic symptomatology of attention deficit/hyperactivity disorder (Crichton, 1798).

A decade before Crichton's death, German physician Heinrich Hoffman created a series of illustrated stories that described symptoms similar to ADHD. Part of Hoffman's clinical training had occurred at the mental hospital in Frankfurt, where he eventually specialized in psychiatry (Lange et al., 2010). Like Crichton, Hoffman broke with the common notion at the time that patients with mental disorders were either possessed or criminal, instead favoring a medical explanation. Hoffman eventually founded a new hospital in Frankfurt that focused on improving the conditions of psychiatric patients. In 1845, he created a series of illustrated stories as a gift to his young son. A well-known publisher heard about the stories and released several of them publicly with great success (Barkley, 2015). One of the stories was the tale of *Fidgety Phil,* a boy who created re-occurring family difficulties due to his inability to sit still and control his behavior at the dinner table. Phil had many of the attributes of modern day ADHD, with symptoms of both inattention and hyperactivity dominating the illustrated scenes, and culminating in Phil falling over in his chair and creating family chaos and parental anger (Hoffman, trans. 1994). Hoffman created a similar story in 1847 titled *Johnny Look-in-the-Air*, which detailed the accounts of a boy so distracted by the clouds in the sky as he walked that he collided with an approaching dog and fell into a river. Hoffman's detailed descriptions of these children, along with his psychiatric training, lend credence to these stories as early descriptions of children with ADHD symptoms (Hoffman, trans. 1994).

In 1902 British pediatrician Sir George Frederic Still delivered a series of lectures that many consider the scientific starting point of the study of ADHD (Still, 1902). In his pediatric work and, later, his professorship in pediatrics at King's College Hospital in London, he led research studies and wrote several medical texts on the topic of pediatric disorders (Baumeister, Henderson, Pow, & Advokat, 2012). In his 1902 Goulstonian Lectures at the Royal College of Physicians of London, he described abnormal psychiatric conditions in children that he thought stemmed from a *defect of moral control* (Still, 1902). Still identified serious deficits in sustaining attention and managing activity level in these children. He also described many of these children as being aggressive, defiant, overly emotional, and showing little ability to inhibit their own behavior (Still, 1902). With regard to behavioral inhibition, Still proposed the inability to delay immediate gratification as the core deficit in these children. He also identified an insensitivity to punishment in these children. Specifically, Still observed that they could be punished for their actions, even harshly, and then re-engage in the same behavior within a short period of time (Still, 1902). This description overlaps with definitions used currently in the *DSM-5* to identify impulsivity, such as children's difficulty with inhibiting behaviors when taking turns, speaking, or handling the belongings of others (APA, 2013). Still's descriptions also appear to connect to deficits in executive function observed in contemporary research, such as challenges with working memory and set shifting, or cognitive flexibility (Rapport et al., 2013).

Still identified another attribute of these children as one of the most commonly observed. He described a heightened passion or emotionality in the children's disposition that left them overly reactive and difficult to control (Still, 1902). He further described this emotional reactivity as a quickness of emotional expression, especially with regard to outbursts of negative

emotion such as anger, rage, hostility, and jealousy; and stated that it was not the expression of the emotion that was problematic, but the failure of the children to be able to control the emotional outbursts (Still, 1902). Still's observations of these children's difficulties managing their emotions supports contemporary understanding of the co-occurrence of deficits in emotional regulation and behavioral inhibition in children with ADHD (Steinberg & Drabick, 2015; Tarver et al., 2014).

Still (1902) described this group of children as possessing a lack of moral consciousness because the children could not constrain their behaviors to act in the best interests of all but, instead, seemed overly invested in their own goals and needs (Still, 1902). Furthermore, he postulated that intellectual impairment produced this defect in the children he observed. Still further theorized that physical disease, such as cerebral tumor, epilepsy, head injury, or other severe afflictions in infancy produced the intellectual impairment (as cited in Baumeister et al., 2012). He also identified some children who appeared to have the moral defect, but who lacked intellectual impairment or history of physical disease (Still, 1902). In these children, he identified symptoms that occurred more in male than in female children (3:1 was his estimate), which correlates roughly with known prevalence ratios of 2:1 for ADHD in current literature (APA, 2013; Still, 1902).

Still observed other correlates as well, such as the tendency for these symptoms to show themselves in early childhood. He noted that most children with the moral defect manifested before the age of seven, which was for many years the diagnostic cut-off in the *DSM* (versions IV-TR and previous) (APA, 2000; Still, 1902). Still also took into account the degree of severity of these children's symptoms, which also overlaps with current diagnostic criteria, as *DSM-5* states that symptoms must be present to a degree maladaptive to the children's functioning

(APA, 2013). In addition, Still noted the importance of factoring children's age into the presentation, which speaks to an understanding of developmental level long before this was a legitimate consideration in conceptualizing children's behaviors (Still, 1902).

Lastly, Still noted a higher incidence of behavior problems in relatives of these children, such as alcoholism, depression, suicide, and criminality. He suggested the likelihood of a biological predisposition in these children rooted in hereditary factors or pre- or perinatal injury (Still, 1902). Indeed, studies published in recent years highlight the heritability of ADHD and support Still's early theories of increased occurrence in immediate family members (Banaschewski et al., 2010; Levy, Hay, & Bennett, 2006; Tarver et al., 2014).

Though George Still's concepts regarding defective moral control continued to refine medical understanding of the foundations of ADHD-like symptomatology, Baumeister et al. argued that his descriptions also closely resemble other childhood disorders, such as conduct disorder or oppositional-defiant disorder (Baumeister et al., 2012). Even so, Still's contributions remain an important step in the history of disruptive symptoms of childhood and their etiology (Martinez-Badía & Martinez-Raga, 2015).

Still was one of the first to recognize that early brain injury may cause children's attentional difficulties. In the period following Still's publications and lectures, Alfred Tredgold (1908) identified similar traits of inattention and low control of willpower or impulse in children. Tredgold furthered Still's observations that behavioral and learning challenges seen in children may stem from early brain damage such as birth defects or perinatal anoxia (Tredgold, 1908). The pandemic of encephalitis in the decade between 1917 and 1928 affected approximately 20 million people, including many children. Many of the children who survived the epidemic showed abnormal behavior described as *post-encephalitic behavior disorder*. Features of this

disorder included personality change, emotional instability, learning difficulties, and cognitive deficits. In addition, medical personnel often observed symptoms of hyperactivity (called *hyperkinesis* at the time), distractibility, and other behavioral challenges (Barkley, 2015; Palmer & Finger, 2001). Like Still, Tredgold noted that the environment often influenced the strength of children's symptoms and that these symptoms appeared to exist in a range from mild to severe, with some children showing no impairment until challenged with the increasing demands of school (Tredgold, 1908). Tredgold and others' studies of postencephalitic children moved the field beyond Still's notion of deficit of moral character toward explanations based on the concepts of brain disease and damage (Barkley, 2015).

Mid-century research. Research in the mid 20th century explored connections between behavioral symptoms in children and possible causes such as disease, birth trauma, toxicity, and head injury (Baumeister et al., 2012). Furthermore, lesion studies in animals revealed an association between damage to the frontal lobes and basal ganglia and disorders of extrapyramidal function (Barkley, 2015). In 1934 Kahn and Cohen proposed the existence of a syndrome called *organic driveness,* which manifested in symptoms of hyperkinesis, inability to voluntarily inhibit behavior, and extreme fluctuation of attention (Kahn & Cohen, 1934). This was the most purely neurological theory to date, and the first to connect ADHD deficits to specific brain regions and to multiple potential etiologies. In the next decade the term *restlessness syndrome* replaced organic driveness, and was eventually called *minimal brain damage* and later *minimal brain dysfunction* in the 1950s and 1960s (Carlew & Zartman, 2017).

The concept of minimal brain dysfunction (MBD) became a central theme in pediatric medicine during this period, with most psychological and behavioral disturbances in children attributed to some type of brain injury—even when no evidence of physiological trauma existed

(Barkley, 2015). By the 1960s institutions such as the National Institute of Neurological Diseases and Blindness began to challenge the notion that brain damage was the primary cause of dysfunction in children. Studies increasingly showed that the category of MBD was far too broad and vague to accurately identify specific disorders in children (Martinez-Badía & Martinez-Raga, 2015). Though inaccurate, the emphasis on purely neurological causes did provide a counter-balance to many of the less scientific theories of etiology at the time, such as the psychoanalytic notion that parental and/or family factors caused these symptoms (Barkley, 2015).

The dissolution of MBD as an explanation for challenging behavior began a clinical shift to an emphasis on identifying more specific behavioral symptoms in children, such as learning disabilities, dyslexia, language disorders, and hyperactivity (Lange et al., 2010). Clinicians identified hyperactivity as the core symptom in overactive and inattentive children and began using the term *hyperactivity syndrome* for this clinical population. Though the etiology of this syndrome still rested in biological causes, pediatric research began to identify additional related symptoms and potential environmental influences (Carlew & Zartman, 2017). Symptoms in addition to hyperactivity included many current functional deficit markers for ADHD, such as poor school performance, oppositional behavior, impulsivity, and poor attention span (AAP, 2011).

Toward a *DSM*-defined disorder. During the 1960s treatment also evolved, with the development of multimodal treatment approaches that mirror contemporary models, including parent counseling, special education services, psychotherapy, medications, and behavior modification (Barkley, 2015). In 1968, the *DSM-II* formally identified hyperactivity and its

associated impairments as *hyperkinetic reaction of childhood disorder*. Although characterized by hyperactivity and distractibility, clinicians did not consider the disorder serious and described it as often remitting by adolescence (APA, 1968).

Research in the mid 1970s revealed that these symptoms often persisted beyond adolescence. In addition, neuropsychologists began to move from a localized model to one that viewed brain regions as functionally interrelated (Carlew & Zartman, 2017). The 1970s saw a shift from a focus on hyperactivity toward an emphasis on deficits in attention. The rationale was that attentional deficits created as many problems for these children as hyperactivity. In addition, research identified that hyperactivity was only present in some of these children, and much of the time with no brain damage present (Barkley, 2015). Diagnosis was complicated further by the fact that several other childhood disorders such as anxiety, mania, and autism also contained symptoms of hyperactivity (Lange et al., 2010).

Hyperactivity soon receded as the central mechanism of the hyperkinetic disorder, as clinical research shifted the decades-long theory that hyperactivity was most often the product of neurological damage. Indeed, many studies identified that hyperactivity may occur with no brain trauma present (Barkley, 2015). In addition, stimulant medication was starting to gain widespread use as an effective method of treatment for problems with focus (Baumeister et al., 2012). This eventually led to a change in diagnostic labeling in the next version of *DSM* (III) in 1980. *DSM-III* renamed hyperkinetic reaction of childhood *attention deficit disorder* (with or without hyperactivity) to reflect the significance of the attentional component (APA, 1980). This definition highlighted the idea that hyperactivity was not the central diagnostic criterion and that,

in fact, the syndrome could occur entirely without it. This re-definition also marked a departure from World Health Organization's *ICD-9* classification, which continued to emphasize hyperactivity as the central disorder (APA, 1980).

In 1987 *DSM-III* underwent revisions and renamed the disorder *attention deficit-hyperactivity disorder* based on evidence from field trials. *DSM-III* combined symptoms of inattention, hyperactivity, and impulsivity into a single symptom list, with specifiers for severity (mild, moderate, or severe). The authors also renamed the subtype ADD without hyperactivity as undifferentiated ADD, with the main diagnostic focus placed on various combinations of the three primary symptoms (APA, 1987; Carlew & Zartman, 2017). The 1994 fourth edition of *DSM* (*DSM-IV*) further refined the diagnosis due to ongoing research showing that children with ADD without hyperactivity differed significantly from those with hyperactivity. Other discoveries also prompted the changes, including the identification of deficits in reinforcement mechanisms, structural abnormalities in the prefrontal-striatal network, genetic contributions to the disorder, and the conclusion that ADHD not only occurred in childhood but often persisted into adolescence and adulthood, as well (APA, 1994; Lange et al., 2010).

Research in the late 1980s and early 1990s prompted more *DSM* changes, including subdividing the single category of ADHD into three subtypes in *DSM-IV*: predominantly inattentive, predominantly hyperactive-impulsive, and a combined type with symptoms from both of the other subtypes (APA, 1994). This new categorization allowed for the re-introduction of the purely inattentive subtype, which clinicians observed to be qualitatively different from the hyperactive-impulsive type—with less aggression and more lethargic, hypoactive, distracted, and academically challenged traits (APA, 1994).

The diagnostic criteria for ADHD changed again in 2013 in *DSM-5* due to expansive research in the areas of psychometrics, molecular biology, neuroimaging, and the cognitive and affective neurosciences (APA, 2013; Carlew & Zartman, 2017). The diagnostic category ADHD NOS split into other specified ADHD and unspecified ADHD, and autism spectrum disorder was no longer an exclusionary diagnosis. *DSM-5* also adjusted the age cutoff from 7 up to 12 years. This allowed adults to look back to symptoms in middle school years to facilitate accurate adult ADHD diagnosis (APA, 2013). In addition, the diagnosis for adults and adolescents 17 and over only required five symptoms instead of the six required for younger children. The three subtypes were also re-worded as *presentations*, which emphasized that the disorder could shift throughout developmental life periods (APA, 2013). Lastly, *DSM-5* added the descriptors *mild, moderate,* and *severe* to convey the number of symptoms a person displayed and how much impact those symptoms had on the person's life. Retention of the 18 core symptoms of the disorder reflected the APA ADHD work group's judgment that the current diagnostic criteria have generally stood the test of time, and are effective at identifying the population who—in spite of a wide range of outcomes—do have neurobiological similarities, genetic correlates, and shared behavioral symptoms (APA, 2013; Epstein & Loren, 2013).

Assessment and Diagnosis

Assessment of the symptoms of ADHD in its many incarnations (sensibility of the nerves, defect of moral control, hyperkinesis, minimal brain dysfunction) came primarily from observation of children's behavior and, in some instances, medical examination for brain injury from disease or trauma (Barkley, 2015). In 1969, C. Keith Conners ushered in a new era in evaluation by developing parent and teacher rating scales to measure levels of hyperactivity in children. Use of these semi-structured scales shifted the assessment process from observational

impressions to quantitative measures of behavioral deviance and severity (Conners, Sitarenios, Parker, & Epstein, 1998). Parents, clinicians, and school personnel used the Conners scales to assess and compare levels of hyperactivity at home and in the classroom. The scales were also useful for clinical research and monitoring the increasing use of stimulant medication. The Conners rating scales enabled the collection of large-scale normative data on the prevalence of a variety of behavioral symptoms in children, and remained the gold standard for assessing inattention and hyperactivity in children for several decades (Barkley, 2015; Conners et al., 1998).

With the reconceptualization of the syndrome into ADD (with or without hyperactivity) in *DSM-III* in 1980, assessment moved from its primary focus on hyperactivity to the inclusion of inattention and impulsivity, as well. As specificity for diagnosis increased regarding age of onset and duration of symptoms, evaluation itself became more specialized in separating ADD from other childhood psychiatric disorders (APA, 1980). At that time, the Conners 10-Item Hyperactivity Index was also measuring symptoms of aggression. This produced samples of children with mixed disorders, and confounded research and clinical diagnoses (Barkley, 2015). During the 1980s and beyond, clinicians developed other scales to measure ADHD symptoms with greater specificity. In the last two decades, the Conners scales were expanded and renamed the Conners Comprehensive Behavior Rating Scales (CBRS) (Conners et al., 1998). The new design provided clinicians with a complete overview of childhood concerns and disorders, including assessment of behavioral, emotional, academic, and social functioning. Currently, the Conners scales guide diagnosis, recommendations for inclusion in special education, treatment planning, and response monitoring. The scales consist of parent, teacher, and self-report forms that cover children ages 6 to 18 years (Epstein & Weiss, 2012). The latest version of the CBRS

conforms to diagnostic criteria in *DSM-5* and is available in both English and Spanish. Both paper and computerized formats are available. There is also a Conners Clinical Index (CI) form, which serves as a quick screening and pre-evaluation instrument. Both the CBRS and CI scales screen for ADHD symptoms with moderate statistical reliability and validity (Chang, Wang, & Tsai, 2016).

Behavior rating scales, comprised of observational checklists of various behaviors, are the most common assessment tools in schools and community settings—largely because they are easy to administer, efficient, and reliable (Barkley, 2015). In the early 1980s, Achenbach and Edelbrock developed the Child Behavior Checklist (CBCL), which gained widespread use due to its comprehensive evaluation of symptoms and rigorously normed construction (Achenbach, 2009). The CBCL is considered a broadband rating scale in that it assesses for a multitude of child psychopathologies including depression, anxiety, conduct issues, and ADHD symptoms (Chang et al., 2016). The CBCL offers both preschool and school age versions most often completed by a parent or close caregiver, in addition to a teacher report form and a youth self-report form that can be filled out by children aged 11 to 18. Scoring is grouped into eight empirically validated syndrome scales, which combine scores for groups of behaviors that typically cluster together. The CBCL has good discriminative ability to identify ADHD in children in all age ranges (Achenbach, 2009; de la Osa, Granero, Trepat, Domenech, & Ezpeleta, 2016).

The Behavior Assessment System for Children, Third Edition (BASC-3) is another broadband assessment scale to initially assess for symptoms associated with ADHD. The scales provide behavioral information for children and youth ages 2 to 21 and take 10 to 30 minutes to complete. There are multiple options for scoring, including manual and web-based

administration (Jarratt, Riccio, & Siekierski, 2005). BASC-3 contains a set of rating scales for teachers, parents, self-report, student observation, and for developmental history. The child and adolescent forms, available in English and Spanish, provide optional behavior intervention guidelines for parents and monitoring tools for school and clinical environments (Heath, Curtis, Fan, & McPherson, 2015).

In addition to the broadband tools for assessing psychopathology in children, scales exist that assess a narrower range of symptoms specific to ADHD. In 2002, Mark Wolraich, MD, developed the Vanderbilt scales at the Oklahoma Health Sciences Center (Wolraich, Lambert, Doffing, Bickman, Simmons, & Worley, 2003). The scales show good reliability and validity across multiple study samples (age, gender, etc.) and reliably identify all subtypes of ADHD as well as oppositional defiant disorder, conduct disorder, and anxiety/depression in children (Epstein & Weiss, 2012). The Vanderbilt rating scales contain 55 items to assess ADHD *symptoms* and *impairment in performance* at home, school, and in social settings. Six subscales help to distinguish the three ADHD subtypes: inattentive, hyperactive/impulsive, and combined (Wolraich et al., 2003).

The Vanderbilt scales ask parents to rate the frequency of children's behaviors on a 0-3 scale with the following parameters: 0: *never*; 1: *occasionally*; 2: *often*; 3: *very often*. A response is considered positive with a score of 2 or 3. The final eight of the 55 questions ask parents to rate children's performance in school and social interactions on a 1-5 scale with the following parameters: 1-2: *problematic;* 3: *average;* 4-5: *above average.* The Vanderbilt scales have the additional advantage that they are in the public domain and, thus, can be accessed by parents and teachers free of charge (Wolraich et al., 2003).

Several additional rating scales also assess for ADHD traits. These scales are normed and show good validity and reliability for identifying symptoms related to the disorder. They include the Disruptive Behavior Disorders Rating Scale (DBDRS) (Prins, Dovis, Ponsioen, Brink, & Van der Oord, 2011), ADHD Rating Scale-IV (ADHD-RS) (Armstrong et al., 2015), ADD-H Comprehensive Teacher's Rating Scale (ACTeRS) (parent and self report also available) (Barkley, 2015), and the Barkley Home and School Situations Questionnaires (HSQ/SSQ) (Barkley, 2015).

The Process of Assessment. Because there is no single behavioral or biological marker for ADHD, assessment requires a multimodal approach to measure not only diagnostic symptoms, but impact on functioning, as well (AAP, 2011). In addition, due to high comorbidity rates with other disorders and genetic factors involved, clinicians must take into account contributions from multiple domains, such as developmental issues, family and peer relationships, and adaptive life skills (Epstein & Weiss, 2012). Evaluations typically begin with a parent interview to collect information about presenting symptoms and functional impairment (Nigg, 2013). The *DSM-5* requires that symptoms must occur in more than one setting for diagnosis of ADHD (APA, 2013). For these reasons, it is necessary to collect information from several sources including observation of the child, parent and teacher interviews, use of assessment scales, physical examination (to rule out possible medical conditions), and additional psychological testing if learning, sensory, or other deficits are suspected (APA, 2013; Barkley, 2015).

Many ADHD scales assess for impairment in additional domains. Scales such as the Vineland Adaptive Behavior Scales (VABS) can assess for deficits in daily living skills, social relationships, and motor functioning (Sparrow, Balla, Cicchetti, & Doll, 2005). The Adaptive

Behavior Assessment System (ABAS) has shown good internal consistency and validity for measurement of communication, leisure, home living, health, and self care skills (Epstein & Weiss, 2012). Quality of life should also be assessed because parent report ratings consistently show lower ratings on quality of life measures including self-esteem, mental health, and emotional functioning for children with ADHD (Armstrong et al., 2015). Quality of life can be assessed through diagnostic interview or psychometric measures such as the Child Health Questionnaire (CHQ)—a 50 or 28 item self- or parent-report measure that encompasses 14 areas of physical and psychosocial functioning in children and adolescents with ADHD (Armstrong et al., 2015). Executive functioning (memory, planning, self-regulation) is also often impaired in children with ADHD. Scales such as the Barkley Deficits in Executive Functioning Scale (BDEFS-CA) or the Behavior Rating Inventory of Executive Functioning (BRIEF) assess for deficits in this area (Barkley, 2015; Rapport et al., 2013).

Although psychometric measures produce statistically valid results, they are not more valuable than the parent interview and the therapist's observation and evaluation. While the parents' and therapist's descriptions of children's behaviors are subjective, nevertheless they provide ecologically valid sources of behavioral information across a range of situations (Barkley, 2015). In addition, parents or other primary caregivers provide initial clues that can lead clinicians to more focused investigations about children's functioning in distinct areas. This provides an indispensible starting point for assessment and assists in developing collaborative treatment goals (Fiks, Mayne, DeBartolo, Power, & Guevara, 2013).

Reliably assessing young children for ADHD is complex due to developmental factors. *DSM-5* (APA, 2013) requires identifiable symptoms before the age of 12, but does not specify a minimum age. According to *DSM,* parents often observe ADHD symptoms in children as

toddlers but, due to large variations in normative behavior, accurate diagnosis is difficult before the age of four (APA, 2013). Rating scales such as the BASC-3 assess children as young as two, but most scales such as the Conners and Vanderbilt scales recommend assessing ages six and above (Sullivan et al., 2015). National guidelines concur with the *DSM-5* position and give treatment recommendations for children starting at four years, in part because cognitive abilities such as executive functioning and reward discounting are not yet sufficiently developed until about that age (AAP, 2011).

Symptomatology

For over two centuries clinicians have observed the current set of symptoms required for diagnosis of ADHD (inattention, hyperactivity, and impulsivity) (Baumeister et al., 2012). Over the decades theories have varied regarding both the etiology of the disorder and which of the observed symptoms deserved the primary focus (Lange et al., 2010). The contemporary conception of ADHD dates from the 1968 *DSM-II*, which introduced hyperkinetic reaction of childhood, with a focus on inattention and hyperactivity but not impulsivity. Since that time, the disorder has undergone several name changes and adjustments in nosology with regard to required symptoms for diagnosis, but the focus on key features of inattention, impulsivity, and hyperactivity have remained (APA, 2013; Barkley, 2015).

DSM-5 requires "a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development" (APA, 2013, p. 59). *DSM-5* also requires that symptoms be present before the age of 12, that they occur across multiple environments, and that they cause significant disruption in social, academic, or occupational functioning (APA, 2013). *DSM-5* offers severity specifiers (mild, moderate, severe), which clinicians use to convey degree

of symptomatology and level of functional impairment. Current *DSM* criteria are similar, but not identical, to requirements for diagnosis in the 10th edition of the International Classification of Diseases (ICD-10) (APA, 2013; Barkley, 2015).

Theories differ with regard to whether the symptoms change through the course of development. In 1798, Crichton observed that inattention was the root cause of difficulties in a specific population of children. He also identified disturbances in attention as varied among children, and even within a single child at different times (Crichton, 1798). In 1902, George Still also noted that in environments, such as school, symptoms were severely disruptive while in other settings they were not noticeable (Still, 1902). This variability is reflected in current understanding that the environment plays an important role in determining the severity of symptoms (Nigg, 2013).

Crichton also noted that symptoms of inattention diminished with age, and postulated that the disorder was limited to childhood (Crichton, 1798). This view pervaded medical thinking until the 1990s, when researchers documented adult ADHD and identified it as a lifelong disorder (Lange et al., 2010). Two recent studies showed that about 50% of children diagnosed with the disorder retain symptoms into adulthood, and that symptoms can improve with age in some cases (DuPaul et al., 2011; Tarver et al., 2014). Symptoms can also shift within the disorder over time due to developmental factors. Children may show various dimensions of the disorder at different points in their life due to the influence of developmental or biological changes (Okie, 2006).

Degree of symptoms and impact on functioning can also shift drastically depending on children's environment. Situational demands that challenge children to sustain attention or control motor activity vary greatly from home to school to social environment and so elicit

different levels of impairment. Attitudes, knowledge, and experience of the person observing the child has additional impact on reported observations that contribute to diagnosis (Epstein & Weiss, 2012).

Subthreshold ADHD. Recent investigations have identified *subthreshold disorders*—conditions with symptoms related to known psychopathologies but in which the severity or number of symptoms do not meet the full criteria for the disorder (Balázs & Keresztény, 2014). Clinicians are now identifying children with subthreshold ADHD, which supports the understanding that ADHD is a dimensional disorder with varying levels of severity. Literature on subthreshold ADHD shows a range of prevalence (0.8 - 23.1%), mainly due to definition and measurement differences in completed studies (Hong et al., 2014). Of those children diagnosed with full-syndrome ADHD that do outgrow symptoms, one-third qualify as having subthreshold ADHD six years later, and one-fifth qualify at the 10 year mark (Balázs & Keresztény, 2014). This is relevant because subthreshold ADHD predicts several comorbidities to the same degree as the full-syndrome disorder, such as alcohol and substance abuse, conduct disorders, and antisocial personality disorders (Balázs & Keresztény, 2014; Hong et al., 2014).

Inattention. Difficulty sustaining attention is the hallmark of the inattentive presentation of ADHD (APA, 2013). Children in this category have significantly shorter attention spans than their developmental peers and have difficulties sustaining focused effort for prolonged periods of time—unless a strong source of stimulation is present. Stimulation most often comes in the form of interest or novelty and may shift significantly with differing environments or tasks (Barkley, 2015). This is often confusing for parents, teachers, and novice clinicians who witness children's inattention varying from extreme to not noticeable (Epstein & Loren, 2013).

Distractibility is another issue for children in this group and differs from inattention in that shifts in focus (distractibility) stem primarily from hypersensitivity to environmental stimuli. This hypersensitivity may represent the neurological need for stimulation, which serves to increase overall brain energy thus improving attention span (Vostal, Lee, & Miller, 2013). Inattention can be difficult to distinguish from distractibility by observation. Children who appear unable to focus on their schoolwork may be highly susceptible to distracting stimuli and, thus, incapable of inhibiting shifts in attention. On the other hand, children's poor task completion may result, instead, from a motivational deficit that creates an inability to persist in tasks that are not immediately reinforcing (Barkley, 2015).

Problems with inattention and distractibility create an array of difficulties in the classroom. Children with these symptoms often have a hard time sustaining effort on academic tasks, and may also be prone to focusing on unnecessary details and environmental sources of stimuli not relevant to their work or goals (Cirelli, Sidener, Reeve, & Reeve, 2016). In addition, these children often demonstrate difficulties with organization, time management, and procrastination (Vostal et al., 2013).

Some research suggests that two distinct subgroups of children are diagnosed as primarily inattentive (Penny, Wachbusch, Klein, Corkum, & Eskes, 2009). One group of children exhibits primary inattention, but also shows a degree of hyperactivity/impulsivity. This group would satisfy the *DSM-5* criteria for inattentive presentation (APA, 2013). A second group of children do not display hyperactive-impulsive symptoms, but their attention deficits are linked to problems with arousal and sluggish cognitive tempo (SCT) (Leopold et al., 2016). Barkley suggests the label *cognitive deficit disorder* (CDD) for this group of children, and identifies

symptoms that revolve mostly around issues related to lack of mental clarity and presence of mind (Barkley, 2015). Teachers and clinicians often describe this subgroup of inattentive children as daydreaming, having trouble staying awake, spacey, easily confused, underactive, lethargic, drowsy, and processing information slowly or inefficiently (Barkley, 2015; Leopold et al., 2016; Penny et al., 2009).

Children with the inattentive ADHD presentation are more likely to struggle with memory deficits than children with the hyperactive/impulsive subtype (Barkley, 2015). With regard to working memory (the ability to hold information long enough to move toward a goal), inattention interferes with task performance because distracted children abandon the immediate task and orient toward the source of distraction (Prins et al., 2011). The theory of "non-optimal energetic state" explains the impact of inattention on working memory (and other cognitive processes) (Prins et al., 2011, p.115). The theory maintains that cortical stimulation is required for attention, which in turn impacts other executive functions dependent on sustained attention. Children struggling with inattention do not maintain optimal cortical energy levels and so may suffer from multiple executive functioning limitations, including working memory deficits (Prins et al., 2011). Inattention is also less likely than hyperactivity or impulsivity to improve with age. Approximately 50% of children diagnosed with ADHD combined presentation outgrow the hyperactive/impulsive symptoms, while inattentive deficits persist into adolescence and adulthood (Epstein & Loren, 2013).

Impulsivity. Tsukayama, Duckworth, and Kim (2013) defined impulsivity as the inability to regulate behavior and emotion in the service of goal attainment. This deficit leads to many self-management challenges for children with ADHD (Bunford et al., 2015). Impulsive children often act and react without considering the impact or consequences of their behavior.

Impulsivity also manifests in impulsive thinking, with impulsive students reporting higher levels of disruptive thought impulses than non-impulsive students (Bunford et al., 2015). Lack of impulse control also impacts learning, and children who are highly impulsive often repeat similar mistakes, even when their errors create negative consequences (Barkley, 2015). Impulsivity also impedes the ability to delay gratification, creates difficulties with patience, and interferes with long-term planning and goal attainment (Sanders & Mazzucchelli, 2013). It also increases risk-taking behaviors, and impulsive children are more prone to accidents and personal injury than other children (Nigg, 2013). Finally, impulsivity creates responses others may view as oppositional. This is due to the speed and intensity (often with negative tone) with which impulsive children react to the demands of others (Nigg, 2013).

Impulsivity may also serve to alleviate negative emotions. Seen in this way, impulsivity is a maladaptive emotion regulation strategy for escaping situations that create frustration or other negative affective states (Bunford et al., 2015). Unfortunately, such children are likely to elicit non-supportive, harsh, or insensitive responses from caregivers, which increases their aggression and impulsivity. This is especially true for children with high approach tendencies, high negative emotionality, or low effortful control (Steinberg & Drabick, 2015). Impulsivity may contribute to childhood obesity through poor regulation of negative affect, which increases the propensity for impulsive children to use food to regulate negative emotional states (Nigg, 2013).

Children who struggle with impulsivity respond too quickly to instructions from others, creating an incomplete understanding of the expectations for a particular setting. This results in impulsive errors and failure to consider consequences associated with particular situations (Barkley, 2015). This is especially problematic in school settings, where impulsive children take

shortcuts in assignments and seek smaller, short-term gratifications over longer-term goals with larger rewards (Tsukayama et al., 2013). Impulsivity in school negatively impacts GPA and teacher ratings of classroom conduct, and corresponds with fewer hours spent studying and more hours spent watching television and playing video games (Tsukayama et al., 2013; Volkow et al., 2011). Situations that require cooperation, sharing, and verbal or physical restraint are problematic for impulsive children, creating a negative impact in the school social domain, where interpersonal impulsivity contributes to peer problems for children (Nigg, 2013).

Hyperactivity. Hyperactivity is another aspect of excessive disinhibition that manifests in developmentally inappropriate levels in children with ADHD. Excess activity exhibits in inappropriate motor activities or vocalizations, as well as unusually paced thinking (Richardson et al., 2015). Excessive motor activities include fidgeting, excessive climbing, pacing, inability to sit still, squirming, and restlessness (APA, 2013). Excessive vocalizations include interrupting, humming, profuse talking, singing to self, and making noises (APA, 2013). These symptoms are often more problematic to those around the child than they are to the child him- or herself. This is because children—especially younger children—have limited self-awareness and experience their activity level as normal to them (Healey & Halperin, 2015).

Traditionally, teachers, parents, and even pediatric professionals approached hyperactivity with skepticism as to whether excess activity level was simply circumstance-related or the product of low effort to self-control (Barkley, 2015). Barkley cited 45 years of studies that objectively document that hyperactive children of both sexes do, indeed, display increased motor and vocal activity regardless of time of day or situation, including while they are sleeping (e.g., bruxism, nightmares, sleep talking, snoring) (Barkley, 2015). Excess activity is

most visible in the classroom, where expectations for regulation of movement are the highest, and where hyperactive children display lower levels of on-task behavior than peers (Cirelli et al., 2016).

Hyperactivity also creates *hyper-reactivity*. Children diagnosed with ADHD are more likely than other children to respond to an event in their surrounding environment, regardless of the nature of the event (Barkley, 2015). The difficulty in discriminating between sources of environmental stimulus is an aspect of both impulsivity and hyperactivity in that it is, at its core, a product of deficiency of inhibition, which creates responses that are both too fast and lacking forethought. The fact that impulsivity and hyperactivity are different aspects of the same inhibitory deficit is one reason they are labeled as part of the same diagnostic presentation (APA, 2013; Bunford et al., 2015). Further, behavioral inhibition underlies self-regulation and executive function (Rapport et al., 2013). Hyperactive children have a more difficult time *down-regulating,* or lowering their activity level after fast-paced, active play. Deficits in self-regulation create higher levels of externalizing behavior such as difficulty regulating emotions and failure to control behavior toward others (Steinberg & Drabick, 2015).

Unlike inattention, hyperactivity may decrease with age (Nigg, 2013). This often happens as children move through the biological changes of the adolescent years. However, this may not represent a true elimination of hyperactive symptoms. Instead, teens and adults with ADHD often describe experiencing a shift in hyperactivity from physical over-activity to mental restlessness as they enter the teen years (Barkley, 2015).

Associated Deficits

ADHD produces an array of deficits related to the core symptoms of inattention, impulsivity, and hyperactivity. According to the neuropsychological model of ADHD, all individuals with ADHD exhibit a primary deficit in inhibition. Those with the hyperactive/ impulsive presentation also exhibit secondary deficits in the regulation of affect, motivation, and arousal; and those with the inattentive presentation exhibit secondary deficits in working memory (Bunford et al., 2015). In addition, ADHD may create developmental and adaptive deficits in daily living, social communication, motor coordination, manual dexterity, expressive and receptive language, learning, and self-perception (Tarver et al., 2014). Both primary and associated deficits related to ADHD create functional impairments in children with the disorder. These impairments vary depending on multiple factors, such as symptom presentation, individual temperament, and family and academic support (Epstein & Weiss, 2012).

Children with ADHD demonstrate significant functional impairment and adverse outcomes. Thirty-three percent of children with the disorder fail to finish high school, compared to the national average of 8.7% (Barkley, 2015). Fifty-two percent of untreated teens with ADHD abuse drugs or alcohol, and having ADHD makes children almost twice as likely to smoke cigarettes (Barkley, 2015). For boys, behavior problems associated with hyperactivity result in higher incidence of arrest—especially if they have conduct disorder issues (CD). Approximately 30% of ADHD/CD boys are convicted of a criminal offense, compared to 8% for controls (Nigg, 2013). The disorder also substantially increases rates for motor vehicle accidents, speeding tickets, suspended and revoked licenses, emergency room visits, and personal injuries (Nigg, 2013).

Children with ADHD demonstrate pragmatic language impairments that impact their social functioning (Barkley, 2015). Pragmatic language skills include a variety of communication abilities that enhance social interactions and facilitate friendships. These skills encompass both verbal and non-verbal aspects of social communication, such as understanding both the spoken message and the non-verbal cues present. Children with ADHD show poorer pragmatic language skills relative to peers across all measures, even after controlling for general receptive and expressive language abilities (Staikova, Gomes, Tartter, McCabe, & Halperin, 2013).

Self-regulation. Several researchers have weighed in on the subject of self-regulation. Epstein and Weiss (2012) described self-regulation as an aspect of executive functioning, referring to children's general ability to regulate their mental activities so as to positively influence performance. Sullivan et al. (2015) defined self-regulation as primarily relating to the regulation of emotion (particularly negative emotion) so as to maintain an appropriate level of affective arousal. However, Siegel (2012) identified the involvement of both the sympathetic and parasympathetic branches of the autonomic nervous system (ANS) as essential components of affect regulation. In this context, emotions are reactions to environmental stimuli, which contain both biological and subjective components (Siegel, 2012). Emotion regulation also involves modifying emotional states (increasing or decreasing) through the modulation of physiological and behavioral processes (Sullivan et al., 2015). Children with ADHD frequently struggle with emotion regulation. Thirty eight percent of children diagnosed with ADHD from community samples displayed deficits in affect regulation (Barkley, 2015). Difficulties with emotion regulation emanate from more general deficits in cognitive and behavioral inhibition.

Both executive and cognitive efforts produce the inhibition of affect and more global self-regulation abilities. Deficits in these processes impact the ability of children with ADHD to manage frustration, impatience, and anger (Bunford et al., 2015).

Children's ability to regulate emotion influences the intensity of their responses and the speed at which they can return to a baseline state. The components of self-regulation that are within children's conscious control are termed *effortful control,* and are the product of intentional response modification such as affective and behavioral suppression and attention shift (Bunford et al., 2015). The aspects of emotional response less under children's control are related to physiology and the activation of the autonomic nervous system (Siegel, 2012). Children with self-regulation deficits (ADHD, bipolar disorder, ODD, etc.) often exhibit both high intensity affective states and a slow return to a baseline state (Bunford et al., 2015); and children with the hyperactive/impulsive presentation show greater deficits in self-regulation than children with inattentive ADHD (Barkley, 2015).

Regulation of affect is not limited to negative states. Though children with ADHD are highly reactive, exhibit low effortful control, have low frustration tolerance, and orient to negative emotionality, they can also be extraverted and display the capacity for over-reactive positive emotions (Bunford et al., 2015). Parents and teachers describe these children as boisterous, rambunctious, or exuberant—traits that cause many of the same problems that negative affect creates, such as poor flexibility and maladaptive social behavior (Bunford et al., 2015). Children's ability to self regulate varies across domains, with some situations eliciting a severely dysregulated emotional response and others allowing for greater emotional control (Steinberg & Drabick, 2015).

For young children, emotion regulation is a bi-directional process, meaning that the behavioral cues that allow for mutual attunement, soothing, and regulation of affect come from both the parent and child (Siegel, 2012). Children with ADHD often have difficulty self-soothing (an aspect of regulation), which creates challenges for parents who become distressed in their inability to calm their children (Sanders & Mazzucchelli, 2013). This is especially challenging for parents who also have some difficulty with self-regulation (Sullivan et al., 2015).

Executive functioning. The literature presents a variety of definitions for *executive functioning*, or *executive functions*. The fact that the nomenclature varies from author to author demonstrates its recent inclusion in neuro-scientific investigations (Barkley, 2015). Research from the last decade shows that executive functioning is a *core deficit* of ADHD (Rapport et al., 2013). Executive functioning (EF) is a general term for higher order cognitive processes that allow for organized, goal directed behavior and novel problem solving in the absence of external direction or structure (Van der Oord, Ponsioen, Geurts, TenBrink, & Prins, 2014). This includes working memory, encompassing several interacting components that hold newly acquired information while integrating relevant stored information (verbal and nonverbal). Executive functions also include cognitive flexibility, planning, organizing, self-monitoring, and inhibitory control such as behavioral and emotional regulation (Van der Oord et al., 2014). Most contemporary models of ADHD show connections between EF deficits and functional impairments in occupational, educational, and interpersonal dimensions (Barkley, 2015; Epstein & Loren, 2013).

Executive functions activate prefrontal cortical regions, such as frontal/pre-frontal cortex, temporal lobe, and basal ganglia (Rapport, et al., 2013). Deeper arousal-regulating mechanisms of the limbic structures also activate the pre-frontal regions and support executive functions

(Bunford et al., 2015). Executive functions work collectively to aid in self-regulation and optimal functioning. For example, the following sequence may occur: a child encounters a frustration, the PFC activates response inhibition to control impulse, working memory retains the event to aid in conscious reflection and planning, cognitive processes counterbalance emotion escalation, and the child chooses a behavioral response based on the best available option. In this example, aspects of executive functioning work together to support response inhibition, self-regulation, reflection, and response flexibility. For children with ADHD, deficits in executive functioning create disruptions in this process and lead to maladaptive behavioral responses (Shuai et al., 2017; Van der Oord et al., 2014).

Children with ADHD display executive function deficiencies such as difficulties with inhibitory control, working memory, delay aversion, slow processing speed, planning, and emotional lability (Barkley, 2015). Deficits in these areas create disruptions in children's learning processes. Children with ADHD have difficulty learning from past mistakes and require higher than average rates of repetition for both academic and social learning (Barkley, 2015). Thus, the actions of children with ADHD may be primarily the product of immediate response (impulsivity) rather than forethought, planning, and reflection on past outcomes (Epstein & Loren, 2013).

Children with ADHD struggle with delaying gratification—another aspect of EF that allows children to defer smaller, immediate rewards in the service of larger, longer-term goals (Banaschewski et al., 2010). This is especially important with regard to academic success, as attainment of larger educational goals requires patience, persistence, and delay of immediate reward. Children with ADHD earn lower grades and lower scores on standardized tests of

academic ability (Tarver et al., 2014). One of the core issues in academic failure for ADHD children stems from poor executive functioning, which appears to play a larger role than hyperactivity or other comorbid behaviors (Tarver et al., 2014).

Even though EF deficits are a prominent aspect of the symptomatology of ADHD, problems with executive function present with substantial heterogeneity among children (Cortese et al., 2015). This is because of the range of factors that can contribute to children's particular ADHD symptom profiles. Comorbidities such as autism, anxiety, and learning disorders impact EF skills (Ayaz, Gökçe, Gümüştaş, & Ayaz, 2014; Steinberg & Drabick, 2015). Likewise, variability in neuropsychological functions, such as working memory and verbal fluency impact EF performance (Shuai et al., 2017). EF deficits appear to be developmentally contiguous; children who present with EF problems in preschool show continued deficits in later childhood and even into adulthood (Rapport et al., 2013).

Social relationships. ADHD frequently creates impairments in social functioning. Historical observations of hyperactive children identified the difficulties these children had playing harmoniously with other children, and the tendency for these children to be unpopular among peers (Lange et al., 2010). Current descriptions of social deficits in children with ADHD continue to highlight similar impairments and provide ongoing evidence that children with the disorder struggle with significant maladaptive social behaviors (APA, 2013).

Social problems for children with ADHD occur in 52-82% of children studied (Tarver et al., 2014). Low levels of emotional control, along with high levels of negative emotionality, create social challenges for children with ADHD. Children with the disorder are more often rejected by their peers and are more often rated as unattractive playmates (Tarver et al., 2014). They also display impairments in accurately recognizing the emotions of others, which impacts

their ability to read social cues appropriately. Such difficulty with social cues stems from inattention, as children with ADHD misread or miss, altogether, the behavioral cues of others due to environmental distractions (Staikova et al., 2013). Additionally, emotional dysregulation creates a negative impact on social relationships. Children with lower attentional control have difficulty attending to positive stimuli; thus, they exhibit dysregulation in the form of withdrawal and internalizing symptoms such as anxiety and depression (Bunford et al., 2015). Externalizing forms of emotional dysregulation also occur socially, with ADHD children exhibiting higher rates of oppositional, negative conduct, and high-risk behaviors (Steinberg & Drabick, 2015).

Children with the combined ADHD presentation show social aggression and intrusive behaviors with others in social situations. This is due to higher rates of social impulsivity which create boundary violations, hypersensitivity to the reactions of others, interrupting, difficulties with turn-taking, and lower frustration tolerance (Barkley, 2015). Children with a primarily inattentive presentation display withdrawn social behavior and internalizing symptoms such as social anxiety and difficulties stemming from poor memory (Armstrong et al., 2015). Each of the ADHD presentations creates multiple peer-related challenges for children; as a result, children with the disorder suffer from social isolation, teasing, and conflict with peers (Steinberg & Drabick, 2015). Because social relationships have such a powerful influence on self-concept for children, peer-related deficits stemming from ADHD frequently contribute to low self-esteem. Many ADHD children develop a *negative filter*—a cognitive coping style which anticipates negative input and prevents them from taking in positive information about themselves, further eroding self esteem (Barkley, 2015). These children also use maladaptive coping strategies such as blame, avoidance of responsibility, and resignation to repeated failures.

Many of these negative coping strategies are a product of their attempts to cope with high levels of interpersonal stress combined with inadequate social support (Nigg, 2013).

The inability to delay gratification also contributes to social difficulties for children with ADHD (Sanders & Mazzucchelli, 2013). Many pro-social skills like sharing, cooperation, taking turns, and keeping promises are based on investments in positive social behavior that contribute to stable social relationships *in the long run.* However, with the primary deficits of inattention and/or impulsivity, many ADHD children are either unable to remain focused on the present social moment or unable to produce the positive behavior needed due to lack of immediate payoff. The result is that peers of ADHD children report feeling devalued, and that ADHD children are frequently labeled as selfish or self-centered and, thus, experience higher rejection by their peers (Staikova et al., 2013).

Family relationships. In the family realm, ADHD contributes to interpersonal conflict and struggles. Children with the disorder frequently push parents to the limits of their abilities with behaviors geared to create stimulation and novelty, such as conflict-seeking and intentional provocation (Heath et al., 2015). Parents also develop negative expectations of their children's behaviors, either developing avoidance behaviors or becoming trapped in escalating conflict patterns (Deault, 2010). ADHD also takes a toll on marriages, with the divorce rate for parents of ADHD children three times that of the general population (Barkley, 2015). Typical emotional response patterns of parents of ADHD children include denial of the disorder, profound grief, excessive guilt, chronic anger, blame, envy of other families, and social isolation of the family unit (Tarver, Daley, & Sayal, 2015). Sibling issues are common, manifesting as conflict and negative feelings between siblings and social embarrassment for non-ADHD children (Deault, 2010). ADHD also creates role imbalances in the family, with diagnosed children typically

filling the role of troublemaker and sibling(s) occupying roles such as the good or perfect child, which serve to alleviate parental stress (Barkley, 2015). Siblings of ADHD children report experiencing embarrassment, anger, resentment, over-responsibility, jealousy over double-standards, and the sense of family life being out of control—all of which make functioning difficult for parents, siblings, and children with ADHD (Barkley, 2015).

ADHD symptoms have a reciprocal influence within the family system: the child's symptoms influence parent and sibling behavior, and characteristics of family functioning impact the afflicted child's clinical symptoms (Nigg, 2013). Seen through a transactional lens, family psychosocial variables such as parental psychopathology, sibling temperament, and the family's cultural and religious norms contribute to a continuous flow of mutual influence between the child with ADHD and family members (both immediate and extended). Family variables thus impact ADHD symptomatology and functional impairment, while the child's symptoms simultaneously impact parent and sibling stress and response (Deault, 2010).

High heritability rates increase the chance that one or both parents struggle with ADHD symptoms, as well. Agha, Zammit, Thapar, and Langley (2012) estimated that up to two-thirds of children with ADHD have at least one parent with a history of the disorder. Parental ADHD problems are linked to greater levels of family conflict and more severe ADHD symptoms in children, with some findings showing maternal ADHD as having greater impact on child and family functioning than paternal symptoms (Agha et al., 2012). The Theule, Wiener, Rogers, and Marton (2011) study found that parents' own levels of ADHD symptoms were the largest predictor of parental stress. Parents with ADHD also have difficulty monitoring the behavior of their children and provide less consistent discipline than parents without ADHD (Agha et al., 2012). In addition, parents with ADHD show higher levels of sensitivity to the difficult

behaviors of their own children (Theule et al., 2011). This suggests that ADHD symptoms in parents impair their resources for coping with their children's ADHD symptoms and lead to increased levels of parenting stress (Theule et al., 2011).

Whether in response to the additional stressors of caring for a child with a neurodevelopmental disability or exacerbated by the parent's own ADHD symptoms, parenting stress creates disruptions in family functioning and contributes to parental psychopathology, including depression (Heath et al., 2015). Up to 50% of children with ADHD have a comorbid mood disorder, and up to 33% have an anxiety disorder; this exacerbates both parent and child ADHD symptoms, adding to greater levels of family stress and higher rates of parent-child conflict (Deault, 2010). Parents of children with ADHD also report significantly lower ratings of parenting self-efficacy than non-ADHD parents; this is linked to lower confidence in parenting skills and less effective parenting practices (Heath et al., 2015).

The pervasive and chronic nature of ADHD creates maladaptive behavior patterns in family members. Adults and siblings are particularly vulnerable to adaptive dysfunction as a result of long term living with ADHD and the repeated reinforcement of negative thoughts and beliefs about the struggling child (Barkley, 2015). High levels of sibling conflict create a negative cycle of mutual dysregulation and reactivity, especially in the presence of comorbid disruptive behavior problems (ODD, conduct issues, etc.) for children with ADHD (Bunford et al., 2015). However, sibling relationships can also serve as a protective buffer for children with ADHD, with siblings acting as an arena for development of social and problem-solving skills and serving as a source of lifetime support for the child (Barkley, 2015).

School functioning. The school setting significantly impacts ADHD symptoms. Children with ADHD show poorer grades, lower scores on standardized tests (on average, 1

standard deviation below mean), and higher grade retention and dropout rates than same-age peers (DuPaul et al., 2011). High comorbidity (approximately 38%) with learning disabilities such as deficits in reading, mathematics, and fine motor skills creates challenges beyond traditional ADHD symptom management (Tarver et al., 2014). Inattention and executive function deficits are the strongest predictors of academic failure, and comorbid disruptive behaviors contribute significantly to teacher-student interpersonal problems (Tarver et al., 2014). Preschool-age children demonstrate behavioral, social, and pre-academic functioning impacted by ADHD deficits; and 70% to 80% of children with ADHD symptoms in preschool will continue to display significant symptoms later in elementary school (Healey & Halperin, 2015).

Inattention and distractibility in the school setting causes multiple challenges with scholastic performance for children with ADHD. Difficulty sustaining attention impacts children's ability to follow teacher instructions, participate in group discussions, and perform consistently on tests (Nigg, 2013). Inattention also impacts executive functions of memory, planning, and organization. This causes students either to procrastinate or to take an inordinate amount of time on class assignments (Rapport et al., 2013). Shifting attention also poses a challenge for these children. ADHD children tend to over-focus on unnecessary details of certain tasks while missing essential elements of others (Cirelli et al., 2016). Issues with attention also create unusual study habits for students with ADHD (Barkley, 2015). Some find concentration difficult except in an environment free from visual and auditory distractions. Other students need a constant flow of stimulation to focus well—such as having TV or music on in the background, or doing their homework on computer vs. paper assignments (Barkley, 2015).

The hyperactivity, impulsivity, and self-regulation deficits associated with ADHD cause other significant school difficulties. Children with ADHD are often restless and have a hard time

sitting still and sustaining attention in traditional seated classrooms (Cirelli et al., 2016). Their high stimulus needs and hyperactivity create disruptions in class through excessive socializing and attempts at engaging others. The ADHD symptoms of restlessness (shaking legs, shifting body postures, etc.) are a source of distraction for other students, impairing both peer and teacher relations with the child (Barkley, 2015). Likewise, impulsivity contributes to disruptive behavior problems in the classroom, with ADHD children blurting out answers, interrupting, and violating the personal space of others (APA, 2013). Poor self-regulation, such as ineffective emotional control, also impacts school success. Children with ADHD may display elevated levels of aggression toward peers and non-compliance with teacher requests (DuPaul et al., 2011).

Dysfunction in motivation and reward processing create academic challenges for children with ADHD. Problems with productivity and organization make completing and turning in assignments on time a particular challenge for this group (Prins et al., 2011). Due to disruptions in the dopamine reward pathway, these children have greater difficulty than peers self-motivating to complete schoolwork—especially if the work is difficult or not naturally rewarding (Prins et al., 2011). Thus, children with ADHD frequently choose immediately gratifying activities like drawing, socializing, or daydreaming rather than focusing on schoolwork (Volkow et al., 2011).

On a positive note, some ADHD children perform very well academically. This is consistent with the broad spectrum of severity and functional impact seen with other ADHD symptoms (Cortese et al., 2015). Variables such the quality of the teacher-student relationship are central determining factors in academic and social adjustment for children with ADHD. Many children report that a teacher's caring attitude, attention, and guidance facilitate their

coping with specific school challenges (Barkley, 2015). Students with ADHD are successful when teachers are patient, understand the disability, collaborate with an interdisciplinary team, and maintain a positive attitude toward the children (Barkley, 2015).

Self-esteem. Difficulties in family, social, and school settings have a significant impact on children's feelings of self-worth. This is especially true if developmental impairments create chronic challenges for these children (Epstein & Weiss, 2012). The impact on self-esteem comes from children internalizing negative messages from adults and peers, which occur in response to the children's disruptive behaviors (Barkley, 2015). Such negative messages from parents and other adults exert influence on children's self-concept and, according to (Nigg, 2013), by the age of 6 or 7, ADHD impairments often begin to erode self-esteem. Poor self-esteem, in turn, interferes with personal relationships and may contribute to later pathology such as increased risk for tobacco use, substance abuse, additional psychiatric disorders, and accidents (Okie, 2006). In addition, parent reports show low self-esteem as a primary contributor to decreased quality of life ratings across psychosocial domains for ADHD children (Epstein & Weiss, 2012).

Although clear evidence exists regarding the negative impact of ADHD on self-concept, some evidence points to the contrary. Specifically, some children with ADHD display a *positive illusory bias* in self-perception (McQuade & Hoza, 2008). In essence, the bias produces elevated levels of positive self-perception for children with ADHD, conflicting with the perceptions of others and resulting in inaccurate assessment by these children of their own competence in various domains (McQuade & Hoza, 2008). Boys experience higher rates of the bias, impacting both their social and academic functioning. The bias reflects these children's misperception of superior performance to peers when, in fact, they demonstrate significantly inferior competence.

Some theorists speculate that the positive illusory bias protects children's self-concept and allows them to better cope with the difficulties they experience. Others see it as a product of deficits in executive function (Barkley, 2015; Epstein & Weiss, 2012).

Comorbidity With Other Disorders

The pervasive nature of ADHD creates significant comorbidity with other disorders (Steinberg & Drabick, 2015). Some appear to be linked developmentally and some manifest behaviorally as a product of the core characteristics of inattention, impulsivity, and hyperactivity (Armstrong et al., 2015). Rates of comorbidity for ADHD children are high, with up to 44% of ADHD children showing at least one other disorder, and 43% showing two or more additional disorders (Steinberg & Drabick, 2015). Comorbidities express themselves in internalizing and externalizing behaviors—both of which can be problematic for children and caregivers. Internalizing symptoms include mood disruptions such as depression, and anxiety disorders such as separation anxiety and specific phobias. Externalizing symptoms include acting out behaviors such as aggression, conduct problems, and ODD (de la Osa et al., 2016). While research such as Armstrong et al. (2015) and Balázs and Keresztény (2014) has established the link between ADHD and comorbid disorders, it remains difficult to predict which children with ADHD will display comorbid conditions and exactly which additional disorders will emerge (Steinberg & Drabick, 2015). Children with ADHD who display significant comorbidity do tend to have a severe and chronic symptom presentation and more negative health outcomes than children with lower levels of comorbidity (Steinberg & Drabick, 2015). These children also evidence higher rates of substance use, delinquency, relationship problems, and academic problems (Nigg, 2013).

Comorbidities appear to be both child-specific and contextually related. Certain children show increased potential for internalizing or externalizing disorders; but many of these additional issues are also activated by environmental influences, such as parenting practices and educational experiences (Nigg, 2013). Given the interplay of both predisposition and environment, a *diverse pathways* model explains the many factors which interact to either create comorbidity or not (Steinberg & Drabick, 2015). The high correlation between ADHD and externalizing disorders such as ODD and CD (30-50%) create significant impairment for children in this sub-group (Armstrong et al., 2015). ADHD/ODD/CD children show increased levels of aggression, destructive behaviors, and impairment in parent-child relations. According to Armstrong et al. (2015), early childhood ODD symptoms are correlated with later CD behaviors, criminal offenses, and substance abuse for children with ADHD. ODD and ADHD typically have their onset by about eight years of age, whereas CD typically presents later in childhood. This suggests that ADHD may predispose children to later CD, or that the two disorders may share common risk factors (Barkley, 2015).

Internalizing disorders such as anxiety co-occur in approximately 33% of children with ADHD, and predict a worse response to clinical treatment and lower scores on measures of academic achievement for these children (Steinberg & Drabick, 2015). The onset of the specific type of anxiety (e.g., separation, specific phobias, social anxiety) varies with age, with separation anxiety being the earliest to show in ADHD samples (Steinberg & Drabick, 2015). As with externalizing comorbidities, a correlation between ADHD and anxiety is present, but the exact relationship is not clear. ADHD may create increased genetic risk for anxiety disorders, or the disorders may develop as a result of children living with the symptoms and circumstances related to ADHD (Armstrong et al., 2015).

Mood disorders also occur in ADHD at higher than average rates, with ADHD children more likely than peers to develop depressive disorders in childhood and adolescence (Tarver et al., 2014). Though depression in particular has a strong genetic risk factor, the fact that depression tends to have a later onset than ADHD suggests that ADHD may be a factor in the development of depression (Steinberg & Drabick, 2015). Additionally, impairment in academic, social, and family functioning from ADHD symptoms may contribute to increased levels of depression for these children (Nigg, 2013).

The core deficit of emotion regulation through a primary disruption in executive inhibition contributes to both internalizing and externalizing comorbidities in ADHD (Rapport et al., 2013). Deficits in self-regulation play a role in comorbid psychopathology, along with contextual factors such as parenting behaviors (Bunford et al., 2015). Parenting behaviors contribute to ADHD children's emotion regulation competence, beginning with emotion socialization when children are young (Bunford et al., 2015). Emotion socialization is promoted by early interactions between parents and children. These include both the overt and nonverbal messages that parents provide for children regarding appropriate emotional expression and response. These parent-child interactions influence children's ability to regulate emotional response, which for ADHD children is already a developmental weakness (Bunford et al., 2015). Thus the early training in self-regulation that ADHD children receive from parents becomes an important contributor to later self-regulation abilities, especially for ADHD children who are susceptible to deficits in this area (Barkley, 2015). Conversely, ADHD children who do not receive supportive emotion regulation experiences in the early years tend to suppress emotion, increasing negative emotional arousal and manifesting in anxiety and emotional outbursts (Steinberg & Drabick, 2015).

Children with ADHD present with other comorbid issues, as well. Approximately 60% to 70% of ADHD children also present with Tourette's syndrome or a comorbid tic disorder (Barkley, 2015). Even children with subthreshold ADHD showed elevated rates of internalizing and externalizing comorbidities (Balázs & Keresztény, 2014). Further, ADHD and autism spectrum disorders (ASD) have many overlapping symptoms, such as impaired communication skills and social interaction problems. Thirty-two percent of boys and 75% of girls diagnosed with ADHD scored within the clinical range for ASD (Ayaz et al., 2014).

Comorbidity rates are high for learning issues, as well, with approximately 38% of ADHD children meeting the criteria for a learning disability (Barkley, 2015). The overlap of ADHD with learning disorders affecting reading, spelling, and mathematics may stem from distinct etiologies that combine in particular cases, along with some degree of genetic contribution (Barkley, 2015). Children with ADHD also have higher rates of bipolar disorder, with 50% of children diagnosed with bipolar I disorder displaying ADHD symptomology such as distractibility, hyperactivity, and severe irritability (Moreno, Laje, Blanco, Jiang, Schmidt, & Offson, 2007).

Comorbidities associated with ADHD have a negative impact on quality of life, with ADHD children showing lower ratings compared to non-ADHD peers, both in childhood and into adolescence (Balázs & Keresztény, 2014). Lower quality of life scores serve as precursors to later depressive episodes and increased functional impairment for children as they age, serving as predictive measures of later psychopathology and as focus areas for preventative measures (Balázs & Keresztény, 2014). The interplay between ADHD and coexisting conditions is

complex and highly variable. For example, treatment of ADHD resolves oppositional defiant disorder in some cases; in others, the treatment improves focus and restlessness with little impact on oppositional behavior (AAP, 2011).

Etiology

Neuroimaging (fMRI, DTI, SPECT, PET) studies and research in molecular genetics, neurology, and neurobiology provide some clarity regarding the etiology of ADHD (Barkley, 2015). There is no lingering debate now that ADHD is a true neurodevelopmental disorder with multiple genetic and environmental etiological factors contributing to symptoms (Lange et al., 2010). Abnormalities in brain structures and neurochemical functions—such as metabolic activity and neurotransmitter deficiencies including dopamine (DA) and norepinephrine (NE)—have been correlated with the disorder (Volkow et al., 2011).

Theories regarding ADHD. Several research-based theories have emerged to explain the origins, functions, and impairments observed in ADHD children. Cognitive theories such as *executive dysfunction theory* posit that structural, biochemical, and functional abnormalities in neural networks lead to the deficits in working memory, focus, self-regulation, and response inhibition commonly seen in the disorder (Vostal et al., 2013). This theory suggests that weaknesses in EF reduce children's ability to effectively organize, problem-solve, and move toward the attainment of selected goals. The cognitive processes lacking or impaired in ADHD children aid in simultaneously maintaining information in working memory, suppressing irrelevant information, and inhibiting responses that are either off task or unnecessary (Rapport et al., 2013). This process allows non-ADHD children to integrate information and analyze reinforcement probabilities to ultimately allow them to select optimal behavioral responses (Prins et al., 2011).

Optimal stimulation theory provides another explanation for the symptoms and impaired functions related to ADHD. This theory suggests that children's physiological arousal needs are responsible for producing the symptoms of ADHD. For instance, impulsivity is a means for seeking sensations needed to increase functioning (Vostal et al., 2013). Similarly, children with ADHD may also receive too much environmental stimulation, which decreases cognitive performance. Seen though the optimal stimulation theory lens, behaviors associated with the disorder serve the purpose of helping the system achieve an optimal state of overall arousal (Prins et al., 2011). This optimal arousal level increases attentional and regulatory capacities for the individual, and is the core purpose for observed behaviors. In children with ADHD, shifts in focus, impulsivity, and hyperactivity all stem from their global needs for maintaining optimal levels of stimulation (Vostal et al., 2013).

Another theory suggests that ADHD emanates from dysfunction in neurological reward/punishment and learning systems. This theory rests on the idea that behavior is dependent on the short- and long-term costs and benefits of behavioral choice (Prins et al., 2011). Neurologically, the improper functioning of brain structures that mediate response contingencies—such as the ventromedial prefrontal cortex—and limbic structures such as the amygdala leaves ADHD children with deficits in monitoring subtleties in reward and punishment probabilities geared to maximize choice benefit (Barkley, 2015). The theory proposes this as the central reason that children with ADHD have trouble learning from their mistakes. This theory suggests that disruptions in reward pathways create negative arousal when ADHD children experience delays in gratification, which may explain the impatience and impulsivity observed (Volkow et al., 2011).

Related to reward pathways, the dopamine transfer deficit (DTD) theory suggests that children with ADHD have dysfunction in their anticipatory dopamine cell firing to cues that predict reward. This dysfunction inhibits maintenance of behaviors once reinforcement is discontinued, thus requiring increased and immediate reinforcement in order to achieve and maintain target behaviors (Tarver et al., 2014). DTD theory also proposes that children with ADHD are less able to build representations of reinforcement history, and are thus more influenced by their immediate environment. This would explain why children with this disorder require increased levels of repetition and external reinforcement for learning (Volkow et al., 2011).

Multiple theories attempt to ascribe known deficits and neurobiological contributors to ADHD, and it is likely that no single theory will ultimately emerge as *correct* (Barkley, 2015). Instead, a growing body of literature suggests that *multiple-pathway* and *multiple-deficit* models are the most comprehensive way to conceptualize the disorder. These models explain ADHD characteristics as the result of combinations of various etiological contributors that result in a variety of heterogeneous symptomatic profiles (Steinberg & Drabick, 2015). This type of conceptualization respects the research and theoretical contributions proposed thus far, while moving away from theoretical competition toward a comprehensive perspective of the disorder (Tarver et al., 2014).

Neurobiological underpinnings. Crichton proposed that symptoms of inattention were the product of abnormalities in brain structure or function due to birth trauma, accident, or disease (Crichton, 1798). Two centuries later, contemporary neuroimaging studies show that ADHD patients have abnormalities in frontal-subcortical-cerebellar systems involved in the

regulation of attention, motor behavior, and inhibition (Lange et al., 2010). Literature suggests that impairments in early brain development (as far back as embryological), such as in the prefrontal-striatal regions, contribute to the disorder (Sullivan et al., 2015).

There is also evidence that early environmental influences such as exposure to toxins or infections increase the risk for ADHD. For instance, brain infections such as meningitis or encephalitis cause toxic inflammation in neural tissues and result in ADHD symptoms (Barkley, 2015). Fetal exposure to environmental toxins, whether from prenatal exposure such as maternal drug or alcohol use or postnatal direct exposure such as pesticides or lead may result in similar symptoms (Karpouzis & Bonello, 2012). Evidence also shows that early brain damage, particularly hypoxic-anoxic types, is associated with ADHD symptoms of inattention and hyperactivity; and that seizures or focal strokes also produce similar symptoms (Barkley, 2015).

Neuroimaging techniques such as MRI have identified several structural abnormalities in children with ADHD. Children with the disorder show reduced overall brain volume and decreased global grey matter volume, especially in the regions forming part of the frontostriatal circuits (Tarver et al., 2014). Children with ADHD show about a 4% reduction in overall brain volume compared to non-ADHD peers (Okie, 2006). These children also show decreased white matter in numerous areas, which suggests impaired communication between affected regions (Lofthouse, Arnold, Hersch, Hurt, & DeBeus, 2012). Additionally, scans identify decreases in the size of anterior right frontal regions and in the caudate nucleus and globus pallidus (Barkley, 2015). Reductions in size of the right frontal region are associated with reduced neurometabolite activity in this area, which corresponds with deficits in sustaining attention (Wolosin et al., 2009). Children with ADHD also have smaller cerebellum size, which is associated with difficulties in executive functioning (Banaschewski et al., 2010). Differences in cortical folding

also create reductions in the size and volume of cerebral areas in ADHD children. Cortical folding serves the purpose of enlarging overall cortical surface area, thus increasing structural and functional capacity of the cerebral cortex. Children with ADHD show decreased folding throughout the cortex, but especially in the right frontal lobe, which is associated with ADHD dysfunction (Wolosin et al., 2009).

Neuroimaging of cerebral blood flow using single-photon emission computerized tomography (SPECT) scans reveal decreased blood flow to the prefrontal regions, most notably in the right frontal area and the pathways that connect this area to the lower limbic regions and the cerebellum (Gunkelman & Johnstone, 2005). Decreased blood flow in these areas is linked to behavioral symptoms and severity in ADHD patients (González-Castro, Cueli, Rodríguez, García, & Álvarez, 2016). Likewise positron emission tomography (PET) scans reveal decreased cerebral glucose metabolism in these same areas, demonstrating the connection between metabolic activity in these regions and ADHD symptom severity (Gunkelman & Johnstone, 2005).

Children with ADHD show other structural abnormalities such as cortical thinning and delay in reaching optimal cortical thickness. These children reach peak levels of cortical thickness a full three years later than typically developing peers (Wolosin et al., 2009). Delays in the development of prefrontal cortical (PFC) areas correspond with deficits in executive functioning and support the theory that, as the prefrontal cortex is largely responsible for executive functions, ADHD is primarily an EF deficit disorder (Tarver et al., 2014). Deficits in the dorsal lateral PFC are also implicated, creating difficulties with sustained attention, impaired short-term memory, and decreased mental speed (Barkley, 2015). Likewise, problems with the inferior orbital cortex are linked to poor impulse control, impaired self-control, and problems

with self-regulation due to this area's connections to the limbic system (Barkley, 2015). In sum, structural abnormalities involving multiple brain regions and pathways contribute to the pathogenesis of ADHD (Tarver et al., 2014). These abnormalities have both environmental and genetic contributors, and research shows that these structural deficits emanate from either genetic mutation or experience, or a combination of the two (Wolosin et al., 2009).

The fact that medications altered levels of dopamine (DA) and norepinephrine (NE) and reduced children's ADHD symptoms established the link between ADHD and specific neurotransmitters (Savill et al., 2015). Neuroimaging research confirmed that stimulant medication increases DA and NE availability in certain brain areas, while decreasing them in others (Volkow et al., 2011). However, drug response does not provide a direct causal connection between neurochemical abnormalities and ADHD; thus, this remains an area of current research (Savill et al., 2015). Results from recent PET scan investigations show compromised function in the brain's dopamine reward pathways in patients with ADHD, further supporting the idea that abnormal levels of both DA and NE are involved in the overall etiology of the disorder (Volkow et al., 2011).

Genetic contributions. Many avenues of research have demonstrated the centrality of genetics in the development and transmission of ADHD, with the literature now showing that ADHD is among the most genetically influenced childhood disorders—with rates similar to that of bipolar and autistic spectrum disorders (Barkley, 2015). Historical accounts presented the first observations of significantly higher rates of ADHD symptoms among immediate family members of affected children (Lange et al., 2010). Subsequent research supported these early observations, showing that parents of children with ADHD are two to eight times more likely to have the disorder, with risk to additional immediate family members at approximately 32%

(Barkley, 2015). Children with ADHD are susceptible not only to inheriting their parents' specific types of ADHD, but also similar externalizing behaviors (Sullivan et al., 2015).

The literature indicates that genetics contribute to specific sub-types of ADHD. For instance, ADHD with conduct disorder (CD) may be a distinct familial subtype of the disorder, with research showing that CD with ADHD significantly clusters in families and shares unique genetic contributions (Banaschewski et al., 2010). Twin studies have provided evidence that manifestation of ADHD requires a greater genetic loading in girls than in boys. This translates to a higher threshold for expression and a higher prevalence in family members needed for the disorder to present in female children (Levy et al., 2006).

Adoption and twin studies provide additional evidence for the heritability of ADHD. Hyperactivity, in particular, presents in levels resembling those found in biological vs. adoptive parents (Barkley, 2015). Likewise, twin studies substantiate that heritability creates a greater concordance for both symptoms of inattention and hyperactivity between monozygotic vs. dizygotic twins (Lange et al., 2010). Some investigations identified variance in ADHD traits as the result of genetic factors in up to 73% of children observed, with increased genetic contribution correlating with severity of symptoms (Levy et al., 2006). Twin and adoption research indicates that heritability is responsible for the majority (70-80%) of variation in ADHD symptoms (Barkley, 2015).

Research in molecular genetics has identified several candidate genes involved in the etiology of ADHD. Genotyping utilizes extraction of DNA from white blood cells and identifies markers, or polymorphisms, which are variances of the DNA gene sequence among individuals (Banaschewski et al., 2010). Marker proximity on the chromosomes impacts gene expression and, ultimately, phenotype. Many of the genes identified thus far are involved in dopaminergic

(DAT, DRD4, DAT5, COMT) and serotonergic systems (5-HTT, HTR1B, SLC6A3) (Levy et al., 2006). Genes related to the noradrenergic system (NET1/SLCA62, ADRA2A, ADRA 2C) and candidate genes related to neurotransmission and neuronal plasticity also contribute to the disorder (Banaschewski et al., 2010). Overall, genetic studies have produced inconsistent findings. Candidate gene associations explain only a small percentage of the genetics of ADHD despite research supporting high heritability rates, highlighting the need for ongoing genetic research relative to ADHD (Banaschewski et al., 2010). Such research suggests that genetic contributions to the disorder are complex with many genes (polygenic etiology) involved in small ways in the expression of the disorder (Deault, 2010). In addition, rather than through single gene mutations the disorder may result, instead, from the accumulation of rare but large deletions and duplications across the genome (Banaschewski et al., 2010). Large-sample investigations that use thousands of genetic scans, such as genomewide association studies (GWAS), reveal additional candidate genes linked to ADHD. These studies look beyond individual gene contributions to sets of genes that create functional networks possibly involved in ADHD etiology (Barkley, 2015).

Epigenetic factors also contribute to familial transmission of ADHD through developmental programming. Rather than direct genetic transmission, this process involves the transmission of genetic susceptibility, which is activated by early experiences and developmental factors (Sullivan et al., 2015). Aspects of temperament, such as activity and effortful control, stem from such epigenetic processes as behavior tendencies originating in the combination of genetic predisposition and early developmental experience (Steinberg & Drabick, 2015). The epigenetic process occurs through modifications of chromatin, which houses the DNA and alters the expression of genes depending on environmental conditions (Sullivan et al., 2015). This

gene x environment model (G x E) is gaining consensus, with many researchers concluding that ADHD is polygenetic with environmental factors interacting in complex ways to contribute to the expression of genetic predisposition (Baumeister et al., 2012). Studies of G x E examine selected genetic markers relative to specific environmental measures, for correlations between genetic expression and psychosocial influences (Barkley, 2015). For example, twin studies by Agha et al. (2012) explored the association between parent antisocial behavior and children's hyperactivity. Results suggested that the parents' impact on children's ADHD symptoms stemmed from both genetic and environmental transmission mechanisms (Agha et al., 2012).

Trauma and environment. While genetic influences appear to be the main contributors to ADHD development, a significant minority of cases (approximately 35%) develop from adverse environmental impacts on neurological development (Barkley, 2015). These cases of secondary ADHD are considered *acquired,* arising from disruptions in development or through some form of traumatic injury (Schachar, Levin, Max, & Purvis, 2004). Birth and pregnancy complications such as longer delivery, fetal distress, toxemia, eclampsia, and low birth weight create increased risk for the disorder, with low birth weight presenting as the strongest contributor (Tarver et al., 2014). Children born pre-term are approximately four times more likely to be diagnosed with inattentive ADHD. The exact mechanism responsible for the correlation of low birth weight and ADHD remains unknown; however, some evidence implicates the impact of fetal growth restriction on brain development (Tarver et al., 2014). Maternal tobacco and alcohol use and higher maternal age also increase the risk for ADHD (Karpouzis & Bonello, 2012; Tarver et al., 2014).

Postnatal insults, such as traumatic brain injury (TBI), are also implicated in ADHD-like symptoms (Schachar et al., 2004). SPECT scans revealed a link between head trauma—

especially trauma resulting in injury to the prefrontal or temporal regions—and ADHD symptoms (Herbert & Esparham, 2017). While the association is clear, there is some difficulty in assigning causation because children with higher levels of activity and impulsivity (ADHD) are at increased risk for having head injuries, and head injuries, themselves, may create ADHD-like symptoms (Nigg, 2013). Approximately 20% of previously unaffected individuals who sustain closed head injuries manifest a syndrome of symptoms called *secondary ADHD (SADHD).* The behavioral symptoms of SADHD, including inattention, restlessness, and impulsivity, mimic the symptoms of primary ADHD (Schachar et al., 2004), suggesting that the causal relationship between head injury and ADHD symptoms is likely bi-directional (Nigg, 2013).

Other sources of brain injury are linked to ADHD, including damage from forceps delivery, falls, sports injuries, physical fights, childhood head banging, and auto accidents (Barkley, 2015; Nigg 2013). Such injuries produce symptoms that mimic ADHD, such as difficulty sustaining attention, memory problems, and decreased energy and motivation (Schachar et al., 2004). Damage to various brain tissues may result from either rotational or shearing effects of traumatic impact, and may involve broken or torn blood vessels at the site of injury, which create bleeding, inflammation, and scarring of the affected tissue (Barkley, 2015). Disrupted blood flow restricts vital oxygen and nutritional supply to affected areas, leaving the tissue without adequate sustenance and inhibiting the removal of cellular waste and toxins that are the product of immune response (Barkley, 2015). Children with no prior history of ADHD who suffer a traumatic brain injury are at least 30% more likely to qualify for an ADHD diagnosis within two years of the injury (Schachar et al., 2004).

Family contributors. Early theories on hyperactivity and inattention focused on psychosocial causal factors such as poor child rearing and poor teaching methods (Lange et al., 2010). Twin study research into psychosocial contributions to ADHD has consistently demonstrated that shared environmental factors such as social class, educational status, home environment, occupation, and parenting practices account for 0-5% of ADHD—a number without statistical significance (Barkley, 2015). Although the origins of ADHD symptoms cannot be explained by social mechanisms separate from established genetic contributors, the relationship between severity of symptoms and psychosocial adversities is clear (Nigg, 2010). This appears especially true for externalizing symptoms associated with ADHD, such as oppositionality and aggression which are influenced by parenting practices, parent mental health, family substance abuse, family violence, sexual/physical/emotional abuse, neglect, lower SES, and parental divorce (Deault, 2010).

ADHD exists as a risk factor for a range of negative outcomes related to family functioning. With ADHD children being at elevated risk for comorbid behavior and mood disorders, academic failure, and impaired social relationships (Balázs & Keresztény, 2014), the symptoms of ADHD also create heightened risk for disturbance in the family system. Correlations between ADHD and problematic family functioning include increased rates of parental psychopathology, family stress, and parent-child conflict (Heath et al., 2015). Deault (2010) found that oppositional and conduct problems—strongly associated with ADHD but not always present in the clinical profile—are associated with the most negative family outcomes (Deault, 2010). Children with challenging temperaments—such as those who are difficult to soothe or those with low positive affect—and difficult behavioral characteristics engender aggressive and coercive parenting practices (Agha et al., 2012). The harsh response by parents

in reaction to children with ADHD further influences children's negative disposition and contributes to the development of additional conduct problems through the process of mutual reinforcement (Tarver et al., 2014).

A complicating element within families is that genetic factors mediate psychosocial influences on the family system, with parents and children sharing both common environmental experiences and heritability, which complicates assigning causation of behaviors strictly to environment (Agha et al., 2012). Family dynamics in response to children's symptoms do impact ADHD, amplifying or reducing symptom severity, but do not initially cause the symptoms (Armstrong et al., 2015). Parent-child interactions serve as the central mechanism in the *developmental transactional* framework, which views ADHD severity in part as a product of family interactions and the quality of the relationships (Steinberg & Drabick, 2015). This framework is not limited to family influence, as it incorporates extra-familial and cultural influences, as well. For instance, the developmental transactional model recognizes that, due to high heritability, children with ADHD are likely living in a system with others possessing the same disorder and/or comorbidities (Steinberg & Drabick, 2015). The genetic and behavioral influences of all participants interact reciprocally to influence one another and amplify risk through epigenetic functions (Barkley, 2015).

Emerging Treatments

Both pharmacological and psychosocial interventions for ADHD have distinct limitations with regard to access, efficacy, and side effects (Rapport et al., 2013; Sonuga-Barke et al., 2013; Tarver et al., 2014). Traditional interventions such as psychostimulant medication and behavioral therapies may not provide long-term improvement for children with ADHD, with the MTA study (1999) documenting continued significant impairment across a range of settings

three to eight years after intensive behavioral and pharmacological treatment (MTA Cooperative Group, 1999; Rapport et al., 2013). Treatment for the disorder is complicated by the heterogeneous presentation of ADHD symptoms and the range of potential comorbidities and functional impairments associated with the disorder (Barkley, 2015).

All of these factors necessitate consideration of individual differences and family circumstance when developing treatment plans (Deault, 2010; Steinberg & Drabick, 2015). Given the heterogeneity involved, alternative treatments offer additional help for children diagnosed with the disorder—especially those for whom pharmacotherapy is either insufficient or not tolerated (Karpouzis & Bonello, 2012; Tarver et al., 2014). Since no single therapy is accepted as completely effective, individualized combinations of evidence-based treatments such as neurofeedback, executive function treatments, and self-regulation strategies may prove to be the most effective way to approach treatment for the disorder (González-Castro et al., 2016).

Neurofeedback. Neurofeedback (NF) is an area of research and treatment based on advances in brainwave mapping utilizing quantitative electroencephalogram (qEEG), which uses measurement of electrical patterns at the surface of the scalp to reflect specific brain activity (Gunkelman & Johnstone, 2005). The EEG fluctuations measured on the scalp represent summed cortical potentials, which arise from the synchronous activation of large neuronal networks. These locally synchronous cortical activities, referred to as EEG source processes, are the targets of EEG biofeedback, also known as neurofeedback (Loo & Makeig, 2012). The earliest approach to imaging cortical activity, EEG has been used for more than 40 years to explore the neurophysiology of attention deficits (Gunkelman & Johnstone, 2005).

Recent years have seen renewed interest in EEG for diagnosis and treatment. This appears related to signal processing advances that allow for higher density EEG recordings and

3-dimensional functional imaging. In addition, increased spatial resolution allows clinicians to capture quick, transient cortical events and changes in cortical tone in specific brain regions (Loo & Makeig, 2012). Current sophisticated programs allow for intensive analysis of data, which leads to increased diagnostic precision and more effective EEG treatment approaches, such as neurofeedback (Loo & Makeig, 2012). Neurofeedback therapy requires specialized training for the clinician, which limits its accessibility. Biofeedback Certification International Alliance (BCIA) certification in neurofeedback is available to individuals with a BA/BS degree in specified health care areas through education, mentoring, and a written exam (BCIA, 2017).

Neurofeedback (NF) reduces symptoms by re-training cortical responses to strengthen deficit areas such as attention and overall brainwave activity (Vollebregt et al., 2014). While neurofeedback is an emerging treatment for ADHD, investigations within the field of child and adolescent psychiatry have explored its efficacy for several decades (Bakhshayesh, Hänsch, Wyschkon, Rezai, & Esser, 2011). For example, NF reduces the severity and frequency of seizure activity and is a commonly used for patients with seizure disorders not well controlled by medications (Loo & Makeig, 2012). The goal of neurotherapy for ADHD is symptom reduction by altering core imbalances in brainwave activity, providing more lasting improvement than can be achieved through medication and behavioral therapy (Gunkelman & Johnstone, 2005). Neurofeedback holds the potential for creating internalized learning with benefits persisting beyond initial training, and does not require ongoing intervention. By contrast, medications that improve ADHD symptoms such as inattention and executive functions do not show reliable efficacy beyond three years and do not create permanent change (Sonuga-Barke et al., 2013).

The procedures of NF vary, but generally entail placing one to three electrodes on areas of the scalp that correspond to specific lobe and hemisphere locations. For example, Cz is the

most common placement location for ADHD NF research, where C refers to the site over the central cortex and z over the top of the head (midline) (Lofthouse et al., 2012). Therapy sessions are typically 30-60 minutes and occur two to three times per week for 20-40 sessions (Loo & Makeig, 2012), recording and processing EEG activity both at rest and during specific tasks. The clinician analyzes changes in cortical processing and compares the recorded data to established norms (Gunkelman & Johnstone, 2005). EEG records cortical activity in selective frequency bands, measured as follows: delta (< 4 Hz) corresponds to slow–wave sleep state; theta (4-7 Hz) corresponds to a drowsy/inattentive state; alpha (8-12 Hz) corresponds to a relaxed/wakeful state; and beta (13-25 Hz) corresponds to an active/attentive state (Lofthouse et al., 2012). Increases in theta activity correspond to decreases in both metabolism and cortical blood flow, and increases in beta activity correlate with increased cortical metabolism (González-Castro et al., 2016). The EEG frequency bands do not occur in isolation, but comprise a range of power spectrum frequencies acting in concert to produce cortical activity in the form of regional changes in amplitude, or power (Loo & Makeig, 2012).

The training of slow cortical potentials (SCPs) is another means to measure and influence cortical activity through neurofeedback. SCPs are electrical changes lasting from several hundred milliseconds to several seconds, and represent task-dependent mobilizations of cortical processing energies (Lofthouse et al., 2012). Negative SCPs represent increased neural excitation and positive SCPs indicate reduction of cortical excitation (i.e., during behavioral inhibition) in the underlying neural networks (Gevensleben et al., 2010). SCP neurofeedback for ADHD attempts to influence SCPs by training children to generate both positive and negative SCPs to help them improve regulation of their cortical energies and improve cortical/behavioral functioning (Gevensleben et al., 2010).

Neurofeedback is based on the general principles of biofeedback and cybernetics, which involve learning self-regulation techniques to modify the electrical behavior of the brain (Gunkelman & Johnstone, 2005). It gives clinicians the ability to view qEEG data while children are engaged in specific activities, such as manipulating controls on a video game (Vollebregt et al., 2014). The therapy also simultaneously attempts to shape power band frequencies by giving positive rewards through the computer game for decreasing theta and increasing beta activity (Loo & Makeig, 2012). The NF process helps children learn to modify the electrical behavior of specific neural networks through exercises that use operant conditioning to increase cortical activity in certain areas and reduce activity in others (Bakhshayesh et al., 2011). Specific NF treatments for ADHD target activity in the frontal lobe regions, responsible for executive functions of sustained attention, memory, and response inhibition (Barkley, 2015).

Current NF treatment takes a video game format, with the children's own amplified minute EEG voltages controlling the computer game (Lofthouse et al., 2012). Over a series of sessions, children learn to influence the content of the game as the clinician adjusts the criteria for reward, which begins to shape children's neural activity patterns. In this respect, the brain's electrical activity is a form of neural behavior, subject to conditioning through electrical re-training (Lofthouse et al., 2012). Factors such as the specific condition being treated and the severity of symptoms impact the number of NF training sessions required (Gunkelman & Johnstone, 2005).

The participant learning curve for neurofeedback is a fifth-order curve, consisting of several distinct phases of learning (Zuberer, Brandeis, & Drechsler, 2015). First is an initial increase in learning, followed by a decrease. Participants experience a second increase in

learning, then an exponential increase toward the end of treatment (Gunkelman & Johnstone, 2005). These fluctuations in the learning process correspond with distinct experiences of participants: First they habituate to the training and experience some success, which provides the initial increase in the curve. Frustration with increased efforts to make more progress corresponds to the subsequent decrease in learning. As participants give up active volitional attempts they allow learning to take place naturally; this surrender contributes to more success, leading to the final exponential increase (Zuberer et al., 2015).

Neurofeedback training often begins with a record of existent brain functioning through a full recording of a child's EEG (Vollebregt et al., 2014). This allows a quantitative analysis and comparison of children's brainwave patterns to similarly aged norms. The comparison to norms from national databases allows the clinician to see which areas deviate from the norm. It also allows them to observe in which direction the deviations proceed—i.e., whether an excess or deficit frequency pattern exists—and the location of the deviation (Gunkelman & Johnstone, 2005). All of this enables the clinician to identify individual patterns of activity, which guide the intervention (Gevensleben et al., 2010).

Evans, Owens, and Bunford (2014) suggest that, rather than within the category of traditional behavior management (BM) interventions, neurofeedback should be conceptualized instead as a training intervention (TI). This is chiefly because treatments such as neurofeedback and cognitive training do not involve manipulation of environmental contingencies to produce behavioral change in a particular setting (Evans, Owens, & Bunford, 2014). Instead, NF produces change by improving a child's skill set and/or through reinforcement in the controlled setting, which then generalizes to larger environments (Zuberer et al., 2015). An advantage of TIs is that such treatments do not rely on adults in home and school environments to implement

modified contingencies with consistency and accuracy. Instead, the interventions rely on the unique training sessions to produce sustainable results for children in multiple settings (Gevensleben et al., 2010). Another advantage of TI interventions is the potential for greater treatment integrity—with no secondary adults such as parents or teachers applying the particular strategy (Evans et al., 2014). Evans et al. (2014) reviewed the growing literature on training interventions and concluded that neurofeedback met criteria for a level 3 (possibly efficacious) evidence-based treatment (Evans et al., 2014). Other studies (Gevensleben et al., 2010; Zuberer et al., 2015) support this conclusion, highlighting the potential for NF to provide effective treatment for ADHD symptoms in children.

Children with ADHD show abnormal EEG patterns, which suggests that the neurobiological difference is likely related to the etiology of the disorder (Barkley, 2015). The frequency bands of greatest interest in ADHD research are theta, alpha, and beta, either in isolation or in comparison to other bands (Loo & Makeig, 2012). The most common EEG differences in ADHD children show as increased fronto-central theta band activity, which contributes to reduced arousal or cortical slowing at the resting state (Lofthouse et al., 2012). Children with ADHD show an average of 32% excess theta band activity compared to control groups.

Children with ADHD also display decreased beta activity and higher theta-to-beta power ratios during rest, compared to non-ADHD controls (Barkley, 2015). Beta band activity is associated with mental activity and concentration; thus, decreased beta activity or higher theta-to-beta power ratio in the fronto-central area during resting condition signals lower resting arousal activity (Zuberer et al., 2015). Theta/beta ratio also negatively correlates to average reaction time, with those diagnosed with ADHD showing both shorter and faster reaction times

(Loo & Makeig, 2012). This may reflect the difficulty children with ADHD have with impulsivity, in which negotiating the speed-accuracy tradeoff—where accuracy is improved if processing speed can be slowed—is particularly challenging. Reductions in theta activity are associated with decreased ADHD symptomatology (Gevensleben et al., 2010). Barkley (2015) also pointed out that significant heterogeneity exists in individual levels of theta band activity among children; consequently, theta activity is not an accurate diagnostic measure for ADHD but is, instead, likely a neurological component of the disorder (Barkley, 2015).

Methylphenidate has the ability to increase alpha frequencies in frontal and occipital areas and to increase beta frequencies in almost all cortical areas (Lofthouse et al., 2012). Frequency increases in frontal beta activity are positively correlated with improvement on continuous performance tests (CPT) and on parent ratings of improved attention and behavior (Bakhshayesh et al., 2011). The idea that brainwave activity can be shaped through both medication and conscious efforts forms the theoretical foundations for exploration of NF in the treatment of ADHD (Lofthouse et al., 2012).

Neurofeedback research findings. Several studies showed positive results for NF treatment for children with ADHD (Bakhshayesh et al., 2011; Gevensleben et al., 2009; González-Castro et al., 2016; Liechti et al., 2010). Though these studies shared some common characteristics, most of the studies in this group demonstrated more differences than similarities. All of the children in the treatment groups had previously been diagnosed with ADHD, had IQs > 80, and were a mix of girls and boys between the 6 and 14 years of age. In addition, all of the studies attempted to impact theta/beta ratios, reducing theta/increasing beta, to improve ADHD symptoms on outcome measures (Bakhshayesh et al., 2011; Gevensleben et al., 2009; González-Castro et al., 2016; Liechti et al., 2010). Gevensleben et al. (2009) additionally targeted NF

training to improve symptoms in comparison to attentional skills training (AST). Additionally, all of the cited studies used an outcome measure consisting of a parent and/or teacher rating scale, with several studies including additional measures. For example, Bakhshayesh et al. (2011) included psychological measures including tests of attention and continuous performance; and González-Castro et al. (2016) assessed children with ADHD through symptom measurement (EDAH parent scale), the Test of Variables of Attention (TOVA), and assessment of activation of qEEG through the theta/beta ratio, with electrodes placed at the Cz and left prefrontal area.

Sample size varied significantly among the studies that produced positive results. The study with the largest sample size included 131 children (González-Castro et al., 2016) and investigated the efficacy of NF against both pharmacological treatment and NF and medication combined. Gonzalez-Castro et al. (2016) separated the children into four groups: the control group (children who received no NF treatment or medication), NF group, medication group, and combined group. The authors concluded that the treatment groups did show improved cortical activation, better symptom control, and improved executive functioning, with the combined group demonstrating more benefit in all measured areas. Analysis indicated no significant differences between pharmacological and NF interventions on cortical activation, demonstrating that both treatments had similar effects (González-Castro et al., 2016). In comparison, Gevensleben et al. (2009) used a smaller sample of 94 children, achieving a medium (0.6) effect size at a confidence interval of $p < .05$.

In contrast, Bakhshayesh et al. (2011) used a much smaller sample size (N = 35) and achieved medium (0.5) to large (0.8) effect sizes when the data were analyzed by calculating repeated measures ANOVA with significance set at $p < .05$ (Bakhshayesh et al., 2011). Liechti et al. (2010) also achieved positive results in their small sample study ($N = 5$). This study was

also narrower in scope than the others because of the authors' specific interest in targeting certain cortical regions in an attempt to balance theta/beta brainwave ratios in the anterior cingulate cortex (ACC) (Liechti et al., 2010). Results showed significance in both linear regression for the group mean and improved ADHD symptoms according to parent ratings. The authors determined that the NF treatment produced decreased theta/beta ratios in the ACC across training sessions. This reflected an increase in attentive resting EEG and improvement in clinical symptoms (Liechti et al., 2010).

Design varied significantly between the cited studies as well, which may have affected outcome results. Bakhshayesh et al. (2011) completed a single-blind, randomized, controlled study of two matched biofeedback variants (Bakhshayesh et al., 2011). The authors compared the effects of EEG neurofeedback aimed at theta/beta ratio reduction to EMG biofeedback on primary ADHD symptoms. The Bakhshayesh et al. (2011) study design attempted to control for confounding variables and produce greater clarity on efficacy than previous studies. The authors used randomized group assignment with EMG biofeedback as placebo due to its similarity to NF, and treated all 35 children in the study for 30 sessions (Bakhshayesh et al., 2011). Liechti et al. (2010) also targeted theta/beta ratios, but compared the EEG neurofeedback training to the children's initial resting EEG conditions to show that the NF training produced decreased theta/beta ratios, with linear regression showing significance for the group mean (Liechti et al., 2010). These results showed that NF treatment focused on the ACC produced increases in attentive resting EEG, which helped normalize theta/beta ratios and improve ADHD symptoms (Liechti et al., 2010).

Following CONSORT guidelines for randomized trials, Gevensleben et al. (2009) also employed a randomized, controlled design to minimize differences between treatment

procedures and to blind parents to children's treatment condition. However, rather than using EMG biofeedback as placebo, Gevensleben et al. compared treatment group results to computerized attention skills training (AST) (Gevensleben et al., 2009). The Gevensleben et al. study (2009) used the SAM (Self-Regulation and Attention Management) specific NF system for the treatment group.

Gevensleben et al. was the only study producing positive results that had included a follow-up study in its design (Gevensleben et al., 2009). In 2010, Gevensleben et al. completed a six-month follow up of the same group of children (n = 61, reduced from the original 94 due to attrition) to determine if the NF training effects remained stable over time (Gevensleben et al., 2010). After six months, 50% of the children in the NF group showed at least 25% reduction in the primary outcome measure (ADHD rating scale). Results showed a medium effect size on the primary outcome measure, and small to medium effects on additional symptoms such as completing homework and personal conduct (Gevensleben et al., 2010). The authors concluded that NF treatment effects were sustained at follow-up, and that the effects of the NF group were superior to those of the control group (Gevensleben et al., 2010).

The González-Castro et al. (2016) study used a different NF treatment system, utilizing the EEG Spectrum program to provide NF treatment, with digitized samples taken at a rate of 256 times per second and separated into various frequency bands (González-Castro et al., 2016). Participating children played a video game that attempted to modify brainwave amplitudes at each frequency band through their responses to goals set by the trainer. Outcomes measured differences in the four treatment groups, with post-treatment data scrutinized with multivariate analysis of variance (MANOVA) and covariance (MANCOVA). The design of this study, however, did not include random group assignment or blinding of participants as the

Bakhshayesh et al. (2011) and Gevensleben et al. (2009) studies had. Instead, the parents in the González-Castro et al. (2016) study assigned their children to specific treatment groups according to their own preferences. Also, the control group did not receive a placebo or sham intervention (González-Castro et al., 2016). Due to the design limitations of the study, González-Castro et al. could not recommend NF as a stand-alone intervention. Instead, the authors concluded that the positive outcomes of this study indicated that NF treatment should be considered one part of a multimodal treatment plan for children with ADHD (González-Castro et al., 2016).

Another aspect of group design that differed among studies was the number of NF treatment sessions. Children in both the 2009 Gevensleben et al. and 2016 González-Castro et al. studies participated in 36 NF sessions, although the length of sessions varied (25-30 minutes, and 15 minutes, respectively) (Gevensleben et al., 2010; González-Castro et al., 2016). In contrast, children in the Liechti et al. study (2010) completed 18 treatment sessions, and children in the Bakhshayesh et al. study (2011) participated in 30 sessions.

The number of children taking stimulant medication also varied among these several studies. The González-Castro et al. (2016) study included comparisons of medicated and non-medicated groups in their NF trial in order to control for the impact of medication on NF treatment outcomes. The other studies, however, did not control for medication, except to ask parents not to change medications or dosages during the treatment phase. Inclusion of medication may have produced nonspecific treatment effects in these studies (Bakhshayesh et al., 2011; Gevensleben et al., 2009; Gevensleben et al., 2010; Liechti et al., 2010).

More recent investigations into NF treatment for ADHD in children have not shown similar promise. Several authors (Lofthouse et al., 2012; Loo & Makeig, 2012; Sonuga-Barke et

al., 2013) completed multi-study reviews of NF trials with a variety of treatment designs. The reviews attempted to attain more accurate results than previous studies by analyzing factors that may have contributed to the validity of study outcomes (Lofthouse et al., 2012; Loo & Makeig, 2012; Sonuga-Barke et al., 2013). Both Lofthouse et al. (2012) and Loo & Makeig (2012) focused on the type of control group, as this provided the comparative data to the treatment group outcome. Lofthouse et al. (2012) and Loo & Makeig (2012) examined outcomes of studies using wait-list (WLC), sham, and active treatments such as CBT/cognitive training as control groups (Lofthouse et al., 2012; Loo & Makeig, 2012).

Lofthouse et al. (2012) analyzed all 14 available published randomized trials on NF treatment, starting with the first documented randomized study of NF for children with ADHD by Fine et al. in 1994. The authors followed the Wilkinson Task Force recommendations on statistical inference to calculate effect size (ES) based on post-treatment means and standard deviations of the treatment and control groups (0.2 – 0.4 = small ES; 0.5 – 0.8 = medium ES; > 0.8 = large ES) (Lofthouse et al., 2012). All of the studies utilized NF targeting theta/beta frequencies, with average NF treatment of 46 minutes in duration at 2.5 times per week. Mean treatment ES for all outcome measures was 0.69 (medium ES). Specific ES for ADHD measures were 0.69; ES for inattention was 0.79; and ES for hyperactivity was 0.71—all of which fell within the medium ES range (Lofthouse et al., 2012).

Loo and Makeig (2012) separated the studies in their review by type of control group, first examining three wait-list control studies (Heinrich, Gevensleben, Freisleder, Moll, & Rothenberger, 2004; Levesque, Beauregard, & Mensour, 2006; Linden, Habib, & Radojevic, 1996) comparing NF treatment groups to groups of children who did not receive the treatment. The WLC studies controlled for maturation, practice effects, and the developmental course of the

disorder, but did not provide blinding for parents or experimenters (Loo & Makeig, 2012). Findings of the WLC studies suggested a positive effect of NF training on post- vs. pre-treatment measures. However, results were complicated by small sample sizes and statistical errors found in both the Levesque et al. (2006) and Linden et al. (1996) studies (Loo & Makeig, 2012).

Vollebregt et al. (2014) also conducted a systematic review of existing studies on the effects of both frequency NF and slow cortical potential (SCP) NF on children with ADHD. Ten randomized controlled trial (RCT) studies met inclusion criteria, although their designs were heterogeneous with differences in sample size, control conditions, level of blindness, and medication use (Vollebregt et al., 2014). Similar to the Loo & Makeig (2012) review, Vollebregt et al., noted significant methodological limitations such as small sample sizes and differences in neurocognitive measures that made meta-analysis impossible. Still, Vollebregt et al. concluded that the majority of studies they surveyed failed to show positive effects for either type of NF on neurocognitive functioning in children diagnosed with ADHD (Vollebregt et al., 2014).

Loo & Makeig (2012), Lofthouse et al. (2012), and Lansbergen, van Dongen-Boomsma, Slaats-Willemse, and Buitelaar (2010) reviewed studies utilizing sham controls. Sham NF treatments incorporate single- or double-blind placebo control in the form of identical treatment protocol for participants, but with participants receiving brain signal feedback that is not their own (Zuberer et al., 2015). The Loo & Makeig (2012) review concluded that studies using sham controls did not produce any significant changes in secondary measures such as resting EEG or on neuropsychological measures of executive function. The authors, however, drew similar conclusions as the Vollebregt et al. (2014) and Lansbergen et al. (2010) studies regarding feasibility—specifically, that it is possible to carry out rigorous, blinded, placebo studies of NF efficacy for ADHD children (Loo & Makeig, 2012). Lofthouse et al. (2012) also cautioned that

their overall mean ES (for studies in their review) using sham conditions (0.69) may have been inaccurate. This is because most of the studies they reviewed that claimed to use sham conditions did not actually use true sham-NF control conditions (a triple-blind exact replication of the treatment NF condition) (Lofthouse et al., 2012). The authors also cautioned that sham-NF treatments may actually give participating children reinforcing EEG feedback during random, inadvertently correct bandwidth responses (Lofthouse et al., 2012). In addition, trainers usually told the participating children that the feedback (correct or random) was dependent on whether the children had been paying attention (Zuberer et al., 2015). Lofthouse et al. (2012) cautioned that such factors may reinforce participating children's attentive behavior in NF studies, regardless of the NF feedback they receive.

Loo and Makeig (2012) surveyed five studies that compared NF to other active treatments such as CBT, group therapy, and computerized attention training. They concluded that none of the studies demonstrated significant differences between NF and comparison treatments in teacher-provided ratings of cognitive functioning at post-treatment assessment. A complication of these studies, however, was that the comparison treatments had not been established as effective for treating ADHD symptoms. The authors concluded that NF was only as effective as non-established ADHD treatments (Loo & Makeig, 2012). Lofthouse et al. (2012) identified an additional difficulty comparing NF studies—significant variations in methods for delivering the NF treatment, such as a wide variety of technology and inconsistent electrode placement on the children (Lofthouse et al., 2012).

Responding to an increasing body of research showing correlations between behavioral symptoms of ADHD and impairments in underlying neurocognitive functioning (Rapport et al., 2013), the Vollebregt et al. (2014) study focused on attention, working memory, time processing,

and executive function. In addition, most studies to date had used behavioral outcome measures in NF-ADHD research, so the authors wanted to know if NF treatment might show specific improvements in executive function (Vollebregt et al., 2014). This focus on executive function was also present in the studies Loo and Makeig (2012) reviewed, comparing NF to computerized attention training in an attempt to improve the attentional aspect of executive functioning.

However, rather than completing a multi-study review, Vollebregt et al. (2014) conducted a double-blind, placebo-controlled, randomized study of the efficacy of NF on cognitive functioning in children diagnosed with ADHD. The study included children 8-14 years of age, with a sample size of $N = 41$ (Vollebregt et al., 2014). The children in the study were stratified by age, medication use, and electrophysiological state of arousal; and, except for the NF therapist, all participants (parents, teachers, and researchers) were blind to treatment. EEG training attempted to normalize power within specific frequency bands unique to each child's EEG abnormality (i.e., hyper- vs. hypo-arousal). Clinicians gave positive reinforcement when a child's beta activity remained increased, or when theta frequencies remained low (Vollebregt et al., 2014).

All children in the study underwent neurocognitive assessments pre- and post-treatment, including Sustained Attention Dots Task (SA-DOTS), Visuo-Spatial Sequencing (VSS), WISC-III Digit Span, Time Production Task, and others. The authors conducted statistical analysis using the SPSS statistical program, with a significance level set at $p < .05$, testing each variable at both the individual and group level using ANCOVA and RCI indexes (Vollebregt et al., 2014). The Vollebregt et al. (2014) study design shared similarities with Lansbergen et al. (2010) who also conducted a single NF study using such rigorous methodology as randomization, double blind construction, and a placebo NF treatment for the control group. Lansbergen et al. (2010)

came to similar conclusions as Vollebregt et al. (2014)—specifically, that NF treatment for ADHD children did not produce benefits greater than placebo treatment (Lansbergen et al., 2010; Vollebregt et al., 2014).

The Vollebregt et al. (2014) study produced similar results as a meta-analysis completed by Sonuga-Barke et al. (2013). Both concluded that NF treatment for ADHD children was not effective; however, their efficacy conclusions came from different focal points in the studies. Vollebregt et al. (2014) suspected that the lack of neurocognitive gains in their treatment group resulted both because NF treatment was ineffective for ADHD, and because neurocognitive treatment effects may take longer to manifest and, thus, may not have been measurable at the time of post-test. In addition, Volgebregt et al. based their results on a select group of neurocognitive tests which, although chosen for their sensitivity to attentional issues, may have created unreliable outcome data. Additionally, their study did not separate sub-types of ADHD and was limited to an all-Caucasian sample, limiting generalizability beyond this sample (Vollebregt et al., 2014).

In contrast, Sonuga-Barke et al. (2013) based their conclusions on findings regarding participant blinding, a principal limitation consistently identified in the Lofthouse et al. (2012) multi-study review. Lofthouse et al. noted that the technology required to provide trainer blinding, while also allowing the trainer to adjust the neurofeedback, was likely not available for most of the studies in their review. The authors estimated that only 57% of studies in their review adopted some type of blinding design that attempted to control for participant, rater, and NF trainer expectancies (Lofthouse et al., 2012). Six of the studies in the Lofthouse review had used no blinding for the control group, with only four studies in their review providing a triple

blind construction (children, informants, trainers) (Lofthouse et al., 2012). The Sonuga-Barke et al. (2013) review found similar blinding issues in the NF studies they surveyed.

Due to the limitations that a lack of blinding may produce, the Sonuga-Barke et al. analysis attempted to determine the effects of participant blinding on ADHD NF studies. The authors conducted a two-level analysis that allowed for comparison of more recent *probably blinded* (n = 4) and older *non-blinded* studies (n = 4) (Sonuga-Barke et al., 2013). The first level of analysis used the score from an unblinded rater who was close to the therapeutic setting. The rating was based on the trial's original outcome measure and was termed the *most proximal assessment* (Sonuga-Barke et al., 2013, p. 276).

The second analysis involved trials conducted either in blinded conditions (such as a placebo-controlled trial) or by an adult who was unlikely to be aware of treatment group assignment (Sonuga-Barke et al., 2013). The second analysis explored potential bias of the person responsible for the *most proximal assessment* (usually a parent or teacher), who may have had a personal interest or investment in the outcome of the treatment (Sonuga-Barke et al., 2013). Initially, Sonuga-Barke et al. achieved results similar to Gevensleben et al. (2010), with individual effect sizes and significant treatment effects observed for the *most proximal* assessments (Sonuga-Barke et al., 2013). However, results did not show statistical significance (ES = 0.30) when calculated for *probably blinded* assessments, which is consistent with both the Vollebregt et al. (2014) and Loo and Makeig (2012) outcomes.

Sonuga-Barke et al. (2013) concluded that expectations of the parents and teachers responding to the most proximal assessments likely created the increased effect size—specifically, that parents'/teachers' responses were inflated due to their investment in the success of the treatment for their children (Sonuga-Barke et al., 2013). Zuberer et al. (2015) cautioned

that, in addition to expectancy effects, outcome bias may occur due to potential learning and adaptation during NF training sessions across time. It is worth noting that, although the effect size fell below the significance threshold for *probably blinded* studies in the Sonuga-Barke et al. (2013) study, the standardized mean difference for NF was higher than for traditional behavioral interventions (parent training, child/parent training, teacher/parent training), which suggested some clinical significance (Sonuga-Barke et al., 2013).

Lansbergen et al. (2010) also controlled for participant blinding and found no indication that NF therapy for ADHD children produced gains over sham treatment. Unlike the Sonuga-Barke et al. (2013) review, the Lansbergen et al. study was a single trial with a small sample size ($N = 15$) (Lansbergen et al., 2010). Despite the small sample, the study's rigorous methodology utilized a double-blind, randomized, placebo-controlled construction examining the effects of NF on ADHD symptoms in children ages 8-15 years (Lansbergen et al., 2010). The children were randomly assigned to either NF or placebo NF groups, and participated in 30 sessions of therapy. The children were assessed after 6, 10, 20, and 30 sessions for treatment effects and severity of ADHD symptoms (Lansbergen et al., 2010). Results found no significant treatment effects; however, the authors concluded that the small sample size may have been a limiting factor ($n = 8$ experimental group; $n = 6$ control group) (Lansbergen et al., 2010).

Lofthouse et al. (2012) came to similar conclusions as Sonuga-Barke et al. (2013), determining that although evidence of efficacy of NF treatment for pediatric ADHD was increasing, the literature was not yet conclusive (Lofthouse et al., 2012; Sonuga-Barke et al., 2013). Both Lofthouse et al. (2012) and Sonuga-Barke et al. (2013) recommended consideration of NF for pediatric ADHD as *probably efficacious* until large, multisite, triple-blind, sham-controlled RCTs could provide consistent evidence for a higher level of efficacy. Loo & Makeig

(2012) echoed this sentiment by concluding that NF is not effective enough as a stand-alone treatment but, instead, should be an adjunct treatment as part of a multimodal approach including psychoeducation, medication, and psychosocial interventions (Loo & Makeig, 2012). On the other hand, Lansbergen et al. (2010) found no evidence that NF treatment was more effective than placebo and, therefore, could not recommend this therapy until further research was conducted.

Executive function treatments. Deficits in executive functioning (EF) constitute a core area of impairment for children diagnosed with ADHD, contributing to many of the behavioral, cognitive, and interpersonal difficulties these children encounter (Barkley, 2015). While the use of stimulant medications typically shows moderate to large magnitude improvements in both externalizing symptoms and some aspects of cognitive functioning—for example, regulation of attention and response speed—they are much less effective at improving measures of executive functions such as working memory, inhibition, and set shifting (Rapport et al., 2013). This is a serious limitation of medication use, as lasting change requires improvement in both task performance and cognitive functioning (Barkley, 2015). Even though stimulant medications impact core structures related to both executive and non-executive function, they may not ameliorate the structural issues—such as delays in cortical thickness—which may contribute to executive function deficits (Wolosin et al., 2009).

Behavioral treatments for ADHD, which rest on the application of learning principles and reinforcement contingencies based on operant conditioning, do help children with ADHD improve behaviors while the reinforcement is occurring (Gunkelman & Johnstone, 2005). However, like stimulant medication, meta-analysis of studies has not confirmed sustained maintenance of behavior change in long-term follow-ups—especially after the positive or

negative reinforcement is withdrawn (Sonuga-Barke et al., 2013). Barkley (2015) summarized that current evidence-based therapies provide, at best, effective short-term reductions in negative behaviors and functional impairments, but provide little effect on EF deficits. Barkley suggested exploration into novel treatments targeting higher-order EFs, such as working memory (WM), behavioral inhibition, and set shifting (Barkley, 2015).

Tarver et al. (2014) noted growing interest in the development of novel interventions targeting some of the neuropsychological impairments thought to underlie ADHD. One goal of such treatments is to improve executive functions through training and extended practice, which may improve cognitive processes and result in consequent improvement in ADHD symptoms (Rapport et al., 2013). Barkley (2015) referred to the potential progress made through EF training as producing *near-transfer* and *far-transfer* skill effects. Near-transfer effects are skill generalizations that expand skills learned in EF training to similar real-world situations. Far-transfer effects generalize more widely to cognition and behaviors children can use in less similar circumstances (Barkley, 2015). Barkley reported that far-transfer effects are the most important improvements to look for in EF training, as they show stable improvement for children beyond the laboratory setting (Barkley, 2015). Some studies show efficacy for EF training. For instance, CBT therapy treatments that specifically target executive dysfunction in children, such as time management and organization training for school, have demonstrated moderate to large effect sizes (Epstein & Weiss, 2012). However, research evidence is mixed, with other studies suggesting that programs seeking to improve neuropsychological functioning do not necessarily improve ADHD symptoms (Tarver et al., 2014).

Executive function treatment research findings. Studies evaluating executive function (EF) treatments vary with regard to study design and outcomes achieved. Several studies show

promising evidence that targeted EF treatments can improve specific skills for children with ADHD (Beck, Hanson, Puffenberger, Benninger, & Benninger, 2010; Memarmoghaddam, Torbati, Sohrabi, Mashhadi, & Kashi, 2016; Prins et al., 2011; Shuai et al., 2017). However, other studies did not produce significant results for treatment of the same skill areas (Bikic, Leckman, Lindschou, Christensen, & Dalsgaard, 2015; Chacko et al., 2014; Rapport et al., 2013). A potential contributing factor to the mixed results may be that, although the studies focused on common aspects of executive functioning (response inhibition, set-shifting/cognitive flexibility, working memory), most of the studies only explored the efficacy of specific EF treatments, such as the ACTIVATE or Cogmed Working Memory Training (CWMT) programs (Bikic, 2016; Chacko et al., 2014).

Four studies (Cortese et al., 2015; Evans et al., 2014; Rapport et al., 2013; Sonuga-Barke et al., 2013) completed meta-analyses focused on methodology in an attempt to clarify the validity of previous EF studies. Evans et al. (2014) explored the efficacy of cognitive training for children with ADHD. The authors reviewed articles using evidence-based criteria (EBT) to evaluate the methodology employed in each study (Evans et al., 2014). Having found only two studies that met all five EBT evaluation criteria, Evans et al. (2014) concluded that cognitive training produced mixed results with regard to improving symptoms related to executive functioning. For this reason, the authors recommended that cognitive training be considered an experimental treatment per EBT criteria (Evans et al., 2014).

The Sonuga-Barke et al. (2013) review also explored the efficacy of cognitive training, investigating three trials focused on attention training and three that attempted to improve working memory. The authors identified significant treatment effects (similar to their NF study) for the *most proximal assessment,* the outcome measure used by the rater closest to the

therapeutic setting (usually parent or teacher). They also calculated treatment effects for *probably blinded* trials, where an individual likely blind to the treatment reported on the outcome measure (Sonuga-Barke et al., 2013). Unlike the Evans et al. (2014) findings, Sonuga-Barke et al. (2013) found significant treatment effects, but only for *most proximal* assessments. However, when the authors analyzed *probably blinded* assessments, the standardized mean difference dropped by 0.40, rendering the results insignificant for both cognitive treatments (Sonuga-Barke et al., 2013). The authors concluded that the drop in significance was likely due to bias introduced by the *most proximal* assessment rater, and that the *probably blinded* assessment was the most objective and valid measure of the treatments' efficacy (Sonuga-Barke et al., 2013). The authors also found significant heterogeneity of effects in both types of treatment assessments; and sensitivity analysis suggested that the inclusion of two trials with high levels of ADHD medication use may have impacted the analysis (Sonuga-Barke et al., 2013). Cortese et al. (2015) also observed that the use of medication in EF studies did, indeed, have an impact on study outcomes. In addition, even with their results showing little treatment effect, Sonuga-Barke et al. (2013) noted that the standardized mean differences for the cognitive training interventions were higher than those for traditional behavior treatments. This suggests that these treatments are worth continued investigation in properly powered future studies (Sonuga-Barke et al., 2013).

The Cortese et al. (2015) meta-analysis investigated randomized controlled trials to explore the efficacy of cognitive EF training for children with ADHD. The authors attempted to update the analysis of the Sonuga-Barke et al. (2013) and Rapport et al. (2013) investigations; and to obtain more accurate data by including only RCTs, and by looking at outcome ratings from those most proximal to the child (usually parents) and those likely blind to treatment

conditions (Cortese et al., 2015). Inclusion criteria incorporated only RCT interventions aimed at training cognitive functions, only published articles, inclusion of children between 3 and 18 years of age with an ADHD diagnosis (any subtype), and control conditions including treatment as usual, wait list, and active/placebo/sham (Cortese et al., 2015). Of the 695 records screened, 15 trials met the entry criteria, with six focusing on working memory (WM) training, four on attention training, two on combined WM and attention training, two on inhibition and WM training, and one on general EF training (Cortese et al., 2015). The number of trials in this review more than doubled the Sonuga-Barke et al. (2013) analysis—which had found only six trials meeting their inclusion criteria. Outcome measures for the Cortese et al. (2015) review were similar to Rapport et al. (2013) and included ratings of ADHD symptoms, parent ratings of executive functioning using the BRIEF inventory, standardized measures of reading and math abilities, measures of verbal and visual WM, inhibition, and attention. Statistical analysis included SDM calculation and combination using the inverse variance method (Cortese et al., 2015).

The results of the Cortese et al. (2015) analysis for the effect of cognitive training on ADHD symptoms followed findings of both the Sonuga-Barke et al. (2013) and Rapport et al. (2013) reviews. The authors found that outcomes from the most proximal raters produced significant effects on inattention (but not hyperactivity or impulsivity) and on total ADHD symptoms with a multi-process training approach (Cortese et al., 2015). However, when the authors limited the analysis to probably blinded raters, the effect sizes were only marginally significant. Furthermore, when they restricted the analysis to trials not involving concurrent medication use, the effects on ADHD symptoms were not found to be significant for either most proximal or probably blinded assessments (Cortese et al., 2015). Analysis of improvements in

neuropsychological functioning produced significant effects from cognitive training on WM, but not on inhibition or attention. This corresponded to results of the Pins et al. (2011) study, which had also produced significant WM improvements through cognitive training using a video game format. However, the small-to-moderate significance the 2015 Cortese et al. trials achieved on the BRIEF ratings for executive functioning came solely from ratings completed by persons most proximal to the child. Furthermore, cognitive training did not produce significant effects in reading or arithmetic domains of academic functioning (Cortese et al., 2015).

The Cortese et al. conclusions echoed that of the Sonuga-Barke et al. (2013) study, in finding little support for cognitive training as a primary treatment for children with ADHD (Cortese et al., 2015). Ratings from persons considered most proximal to the child did produce significant effects on ADHD symptoms, but the effects became insignificant when analysis included individuals probably blind to treatment (Cortese et al., 2015). There was significant improvement from cognitive training on tests of both visual and verbal WM, but these effects did not generalize to academic outcomes. The Cortese et al. study (2015) reached similar conclusions as Sonuga-Barke et al. (2013) and Rapport et al. (2013), namely that meta-analysis demonstrated limited evidence for the efficacy of cognitive training for children with ADHD, except in the narrow area of WM functioning, which was limited to training efficacy in specific settings (Cortese et al., 2015).

Though the Rapport et al. (2013) meta-analysis produced similar results as Cortese et al. (2015) and Sonuga-Barke et al. (2013) reviews, the Rapport et al. design was more limited in scope. Rapport et al. (2013) only investigated studies utilizing the *facilitative training* (FIT programs), designed to promote the development of attention and executive functions through extensive training and practice using a computer program (Rapport et al., 2013). Rapport et al.

examined 25 studies of FIT cognitive training which focused on working memory (WM), set shifting, inhibition, and attention for children with ADHD. The authors calculated effect sizes using Cohen's *d*, with confidence intervals set at 95% based on Comprehensive Meta-Analysis criteria (Rapport et al., 2013). Between group statistics included each group's sample size and *t* or *p* values, group means, and the comparison of *p* values or reported effect sizes converted to Cohen's *d* (Rapport et al., 2013).

Similar to the Beck et al. (2010) and Sonuga-Barke et al. (2013) reviews, the 2013 Rapport et al. analysis evaluated whether cognitive training produced improvement in both cognitive and behavioral areas for children with ADHD. The review first identified WM and sustained attention as the central executive processes that contribute to functional deficits in children with ADHD (Rapport et al., 2013). Like Cortese et al. (2015) the Rapport et al. study also looked at both near- and far transfer effects for the programs, concluding that they produced moderate improvement for near transfer measures of cognitive performance for FIT programs targeting WM (Rapport et al., 2013). The analysis found no significant near transfer improvement in FIT programs targeting a combination of EFs, such as inattention and WM (Rapport et al., 2013). FIT programs targeting set-shifting (cognitive flexibility) or attentional processes produced no significant improvements through cognitive training (Rapport et al., 2013). The authors also calculated far transfer effects, with results showing that FIT training produced no significant improvements in children's academic or behavioral ratings. This corresponded to outcomes achieved by both Cortese et al. (2015) and Sonuga-Barke et al. (2013). These results contrast with the positive far transfer effects reported by nearly three-fourths of the original studies of the Rapport et al. review, which Rapport et al. (2013) had concluded were likely due to practice and expectancy effects rather than significant change.

In sum, the Rapport et al. (2013) review indicated that previous claims that the FIT programs produced improved academic achievement, improved cognitive skills, and reduced symptomatology in children with ADHD was not supported by empirical meta-analysis (Rapport et al., 2013). Authors of all these various multi-study reviews identified serious methodological limitations in past studies of EF treatment for children with ADHD. More accurate study results of future research will accrue through consideration of issues such as targeted aspects of EF, participant blinding, and calculations used for near- and far transfer effects (Cortese et al., 2015; Evans et al., 2014; Rapport et al., 2013; Sonuga-Barke et al., 2013).

Chacko et al. (2014) and two Bikic studies (Bikic et al., 2015; Bikic, 2016) produced similar single-trial conclusions as the multi-study reviews. All three studies incorporated RCT methodology and had a focus on executive function skills, though the Chacko et al. study focused more narrowly on improvements in working memory (Chacko et al., 2014). The treatment interventions for the studies differed also, with the Chacko et al. study utilizing the Cogmed Working Memory Training (CWMT) program, which consisted of a five-week computer-based training program designed to improve working memory (WM) capacity by targeting two areas of working memory: Storage, and storage plus processing/manipulation aspects of both verbal and non-verbal working memory (Chacko et al., 2014). The CWMT program, however, used an individualized video game-type of interface that automatically adjusted the difficulty level of the intervention to each specific child depending on his or her performance. The program also provided contingent reinforcement in the form of small rewards for completion of the training week (Chacko et al., 2014).

In contrast, Bikic et al. (2015) created an initial study protocol, and later followed up with two treatment trials that utilized the ACTIVATE cognitive training program (Bikic et al., 2015;

Bikic, 2016). The ACTIVATE program consisted of a combination of physical exercise and computer training that utilized a program developed to teach cognitive skills to children with deficits in executive functioning. The training targeted skill development in set-shifting, sequence memorization, categorization, and general thinking strategies (Bikic et al., 2015). Study design differed between Chacko et al. (2014) and the Bikic studies as well. Bikic et al. (2015) started with an 8 week multicenter randomized trial geared to determine the efficacy of cognitive computer training for children with ADHD. The authors used the ACTIVATE program as the treatment condition and compared it with treatment as usual. The authors designed the trial to test the hypothesis that cognitive computer training may be a treatment option for promoting neural development in children with ADHD and thus may be effective in reducing symptoms related to cognitive dysfunction (Bikic et al., 2015). They based the study on the concept that *cognitive rehabilitation* may hold the potential to impact neural reorganization through external stimulation and training. The authors speculated that this may be especially true for children, whose activity-dependent synaptic plasticity peaks by around age 7 (Bikic et al., 2015).

Bikic (2016) followed up the initial study protocol with two randomized, controlled trials involving cognitive training for ADHD children. The first trial was a pilot study of 18 adolescents with ADHD who were randomly assigned to either active or placebo treatment. The children participated in a 7 week cognitive training program, which included evaluation at baseline and post-treatment (Bikic, 2016). The second trial included 70 randomly assigned children who received either an intervention targeting cognitive functions or treatment as usual. This trial lasted 8 weeks with outcomes assessed at both 12- and 24-months (Bikic, 2016). The outcome measures used for both trials differed from the Chacko et al. (2014) study as they used

the Cambridge Neurocognitive Automated Battery (CANTAB) and measures of behavioral symptoms pre- and post-treatment (Bikic, 2016). The first study was exploratory and helped the author focus on sustained attention as the primary outcome measure in the larger, second trial (Bikic, 2016).

The Chacko et al. (2014) trial also used a randomized, double-blind community sample of English-speaking children ages 7-11 diagnosed with ADHD who had Internet access at home (Chacko et al., 2014). Exclusion criteria included other developmental or psychiatric disorders, and FSIQ < 80. The Chacko et al. (2014) treatment consisted of five CWMT training sessions per week, 30-45 minutes per session, for a total of five weeks. This was somewhat shorter than Bikic (2016), whose larger trial lasted eight weeks and included one- and two-year follow-ups.

Control conditions differed between the two studies, as well. While Bikic (2016) utilized treatment as usual control, Chacko et al. (2014) incorporated an identical CWMT placebo condition that replicated procedures and training aides but did not scaffold the difficulty level to the children's performance. Outcome measures also differed between the studies, with Bikic (2016) using the CANTAB assessment while Chacko et al. (2014) included parent and teacher reports of ADHD symptoms using the Disruptive Behavior Disorders Rating Scale, the Automatic Working Memory Assessment, and the Wise Range Achievement Test 4. The Chacko el al. study used Cohen's *d* effect size to assess the difference in improvement between the experimental and control conditions (Chacko et al., 2014).

Results for the Bikic (2016) and Chacko et al. (2014) studies failed to show that either executive function treatment was effective for ADHD children. The Chacko et al. CWMT training did not result in significant improvements compared to placebo on parent- or teacher-rated ADHD symptom reports, objective measures of ADHD symptoms, measures of

verbal/non-verbal WM, or academic achievement (Chacko et al., 2014). CWMT treatment did result in improvements on measures of simple WM (storage), such as digit recall. The authors, however, expected these near-transfer effects as the outcome measures were very similar to the training tasks the children had already experienced (Chacko et al., 2014). The authors concluded that CWMT had no significant effect on non-trained WM outcome measures requiring generalization of skills to more complex areas of WM, such as those involving both storage and processing/manipulation (Chacko et al., 2014). The findings of this study led the authors to suggest that CWMT should not be used as a treatment for pediatric ADHD (Chacko et al., 2014). Limitations of this study included difficulty in obtaining equivalence of therapeutic components of CWMT and the placebo, and the possibility that parental expectancy effects may have influenced outcomes. In addition, the study did not include long-term follow up (as Bikic, 2016 did), which may have produced benefits in areas of academics that only occur after longer periods of treatment (Chacko et al., 2014).

Similarly, the first Bikic trial found no significant differences between pre- and post-group cognitive or ADHD symptom measures after the treatment intervention (Bikic, 2016). The second trial also found no significant differences in either primary or secondary measures post-treatment, which indicated that, as rated by parents and teachers, the treatment did not have significant effects on ADHD symptoms or executive functions (Bikic, 2016). The trial also found no significant improvement in sustained attention for the treatment group; however, there was improvement in the area of planning ability for specific subgroups of the treatment group (Bikic, 2016). The Bikic study came to similar conclusions as the Chacko et al. (2014) study—that cognitive training produced no significant beneficial effects for ADHD children. However, the Bikic (2016) results did indicate that cognitive training might be beneficial for specific

subgroups, such as older individuals with ADHD or children with the inattentive subtype. Bikic (2016) noted that future studies may provide additional insights into the use of cognitive training for specific ADHD populations.

Several studies (Prins et al., 2011; Shuai et al., 2017) produced positive results for executive function treatments. The focal points of these studies varied, with some focusing primarily on working memory, and some addressing a broader range of cognitive skills. Both Prins et al. (2011) and Beck et al. (2010) targeted WM training for children with ADHD. The sample sizes were similar in the two studies (52 and 51 respectively); but the age ranges differed, with the Prins et al. (2011) study including children ages 7-12 years and the Beck et al. (2010) study targeting a broader age-range of 7-17 years.

Outcome measures differed between the studies, as well. The Prins et al. (2011) study utilized the Corsi Block-Tapping Test which assessed the capacity of visuo-spatial short-term memory and working memory. The test involved repeating sequences of block tapping in specific order, the number of blocks increasing with each success. Clinicians scored the test by the number of blocks the children were able to correctly tap in sequence from memory (Prins et al., 2011). The Prins et al. (2011) study also used an exit questionnaire for the children to assess their level of interest in the program.

In contrast, the Beck et al. (2010) study used several other outcome measures to assess both behaviors and EF deficits before and after treatment. Outcome measures for their study consisted of the number of inattentive symptoms observed from the *DSM-IV-TR* list, the BRIEF Parent Form, and the Conners Rating Scale-Revised (Beck et al., 2010). Both the Prins et al. (2011) and Beck et al. (2010) studies included children taking medication for ADHD—although the Prins et al. study included only children taking short-acting stimulant medication and

required that parents not administer medication on days of training. Sixty-one percent of the children in the Beck et al. (2010) study took stimulant medication during the training. The authors allowed the children to take medication during treatments, but requested that the parents make no medication changes during the trial period (Beck et al., 2010).

Beck et al. (2010) assigned children in the study to either the experimental or waitlist control group by alternately choosing from an alphabetical list of all participants. Parents and teachers filled out outcome measures before treatment and at the 1- and 4-month follow-up dates. The training program consisted of a computer-based program completed in each child's home under the supervision of a parent. The training took place over 25 sessions within a 6-week period (Beck et al., 2010). The exercises included verbal and visuo-spatial tasks such as presenting objects in a sequence and requiring the children to reproduce the sequence. Data analysis compared experimental and control groups on all outcome measures through analysis of variance. The authors calculated Cohen's *d* effect sizes for baseline and post-treatment change, and evaluated statistically significant results using chi-squared analysis (Beck et al., 2010).

The design of the Prins et al. (2011) study differed somewhat from the Beck et al. (2010) trial in that, in the Prins et al. (2011) study, both treatment and control groups participated in WM training. However, children in the treatment group received a different WM training format consisting of WM training through a novel video game format. The study used this design to determine whether the video game format would produce increased motivation and better training performance for ADHD children (Prins et al., 2011). The authors' hypothesis was that the video game format of their WM intervention would produce more stimulation and, therefore, better WM results for ADHD children who are typically more dependent on stimulation for executive functioning (Prins et al., 2011).

Results for the Beck et al. (2010) study indicated moderate to strong effect sizes (0.76 – 1.49) on measures of inattention, which indicated that WM training had a beneficial effect on reducing parent-reported inattentive symptoms. Results also showed significant improvement on measures of executive functioning at both post-treatment and follow-up (Beck et al., 2010). The authors concluded that, per parent report, executive functioning performance could be improved through intense and prolonged training (Beck et al., 2010). Important limitations of the study included the fact that the parents in this study were not blind to treatment, which may have produced expectancy effects and bias in their reporting. In addition, unlike the Prins et al. (2011) trial, Beck et al. did not achieve true randomization with the use of alternating group assignment from the alphabetical list. Finally, Beck et al. (2010) used only parent and teacher reports, which were subject to potential subjective bias, as outcome measures.

Likewise, the Prins et al. (2011) study also produced positive results, with children who used the WM training that contained the video game element showing increased memory span from pre- to post-test using a 2 x 2 ANOVA for repeated measures of interaction effect between treatment conditions and time (Prins et al., 2011). Results showed that the WM training with game condition produced more impact on working memory than traditional WM training when measured by CBTT. Despite positive results, the question remained regarding which specific elements of the gaming format produced the increased motivation and enhanced performance. Prins et al. (2011) recommended that this question serve as a primary focus in follow-up studies utilizing the video game format to investigate EF programs for ADHD children.

Like the Prins et al. (2011) study, a 2014 study by Van der Oord et al. also utilized a video game format that adjusted the difficulty of the training according to children's performance (Van der Oord et al., 2014). Both studies also used rewarding game content in an

attempt to increase performance and enhance motivation. However, the Van der Oord et al. (2014) study differed by targeting improvement not solely in WM, but in three areas of executive functioning: WM capacity, response inhibition, and cognitive flexibility (Van der Oord et al., 2014). The authors conducted a pilot study investigating the efficacy of a 5- to 6-week intensive EF training for children with ADHD. In this study, a video game trained the children to improve executive functions over a sequence of sessions. The study used a sample of 56 children between the ages of 8-12 who had a FSIQ over 80 with a *DSM* diagnosis of ADHD. The design of the Van der Oord et al. (2014) study shared several similarities with the Beck et al. (2010) study. Both studies randomly assigned the children to treatment or wait-list conditions, with stratification for gender and use of medication. They also both used the Behavior Rating Inventory of Executive Functioning (BRIEF) as the outcome measure, and the number of training sessions, number of weeks, and treatment session length also generally matched (Beck et al., 2010; Van der Oord et al., 2014).

Van der Oord et al. (2014) examined trial results based on improvements in performance on inhibition, cognitive flexibility, and WM from beginning to end of treatment (Van der Oord et al., 2014). Paired *t* tests showed significant improvement from start to end of training for both the cognitive flexibility and inhibition training. The WM training also demonstrated significant improvement, except for one level, which was inconclusive due to technical issues. The authors also assessed long-term effects using a within-group ANOVA with ES size set at < 0.06 = small, 0.06 - 0.14 = medium, > 0.14 = large. Long-term effect sizes were medium to large with regard to improvements in executive functioning and behavioral improvement (Van der Oord et al., 2014). Furthermore, outcomes for the subsample of children treated with methylphenidate did

not differ significantly from the larger sample of children in the study, suggesting the treatment was effective for both medicated and non-medicated children (Van der Oord et al., 2014).

The Van der Oord et al. study shared limitations with Beck et al. (2010), including using a wait-list (vs. alternate training) control that may have produced nonspecific treatment effects. The fact that this study also included training in three EF areas (WM, inhibition, and cognitive flexibility) also introduced the possibility that a combined treatment trial may have produced different results than training for specific EF areas (Van der Oord et al., 2014).

Shuai et al. (2017) explored the efficacy, feasibility, and acceptability of a treatment program that, like Van der Oord et al. (2014), focused on multiple executive functioning components for children with ADHD. The Shuai et al. (2017) intervention targeted EF deficits of working memory, inhibition, set shifting, planning and organizing, and emotional regulation. The study included 44 school-aged children diagnosed with ADHD (variety of subtypes), who participated with their parents in 12 EF training sessions during a 12-week period. The authors compared results from the treatment group to a group of 88 health control (HC) children who matched the ADHD group in age and IQ (Shuai et al., 2017). The Shuai et al. (2017) study shared some similarities with the Chacko et al. (2014) study, both utilizing parent report outcome measures such as Conners parent rating scale and Behavior Rating Inventory of Executive Function (BRIEF) in combination with neuropsychological measures such as the Stroop color and word test. Both studies also used a double-blind design for administration and scoring of assessments (Chacko et al., 2014; Shuai et al., 2017).

Shuai et al. (2017) taught the children executive skills and then motivated them to practice until the skills became habitual. They also provided the parents with psychoeducation regarding ADHD and how best to support skill acquisition to ensure the children completed the

daily EF homework assignments (Shuai et al., 2017). At the conclusion of the trial, the authors performed statistical analysis using $p < .05$ for significance through comparison of baseline and post-training outcomes. By the end of the trial, the children in the treatment group had improved significantly on all EF tests (Shuai et al., 2017), with regard to processing speed, inhibition, set shifting, and WM skills. In addition, they showed decreased ADHD symptoms and better behavioral self-management, with decreased rule violations and better self-control as reported by parents (Shuai et al., 2017). This corresponds with the Van der Oord et al. (2014) study, which had also produced improvements in multiple EF skills and ADHD behaviors. Since the Shuai et al. (2017) study produced significant positive results, the authors hypothesized that their focus on a range of EF skills vs. single skill training created improvements beyond those of previous studies, such as Chacko et al. (2014) (Shuai et al., 2017). The authors concluded that training multiple EF skills at the same time was a more effective strategy for improving overall EF functioning (Shuai et al., 2017).

The Shuai et al. study (2017) also included a high level of parent involvement and practice in real life situations, such as completing assignments at home. These factors marked a stark design difference from other EF studies that did not emphasize real world practice and parent participation as part of the treatment construction (Beck et al., 2010; Van der Oord et al., 2014). Shuai et al. (2017) speculated that both parent involvement and practice in the home setting intensified the dose of intervention, and promoted integration and generalization of learned skills. Like Bikic (2016), Shuai et al. (2017) also addressed the issue of feasibility, concluding that their intervention was feasible to administer and had high acceptability and parent satisfaction (Shuai et al., 2017).

Limitations of the Shuai et al. (2017) study included the fact that five of the children were taking medication during the trial. In addition, use of parent reports for ADHD symptoms may have added subjectivity and did not include teacher feedback. The authors recommended further EF investigations utilizing a RCT design, with larger samples and outcome ratings that would include feedback from teachers blind to treatment groups (Shuai et al., 2017).

Lastly, Memarmoghaddam et al. (2016) conducted a trial with an EF intervention that shared a commonality with the Bikic (2016) ACTIVATE EF program—the use of physical exercise to improve executive functions. Memarmoghaddam et al. (2016) identified a growing body of literature suggesting that physical activity and exercise may produce beneficial changes in EF in ADHD children through the stimulation of neurobiological processes. Specifically, the authors noted that physical activity could exert a positive impact on both cognitive and behavioral inhibition in children with the disorder. Several aspects of EF, including self-regulation, working memory, internalization of speech, attention, and hyperactivity/impulsivity influence behavioral inhibition (Rapport et al., 2013). Memarmoghaddam et al. (2016) also noted that previous research by Dishman et al. (2006) and Hillman, Erickson, and Kramer (2008) had determined that exercise increased neuroadaptation and cerebral blood flow, and facilitated the release of catecholamines (dopamine, epinephrine, norepinephrine)—all of which play a role in ADHD symptomatology (Memarmoghaddam et al., 2016).

Memarmoghaddam et al. constructed the study to test their hypothesis that an exercise program specifically designed to impact EF in children with ADHD may increase aspects of cognitive functioning, including behavioral inhibition (Memarmoghaddam et al., 2016). On a voluntary basis, the study recruited boys between the ages of 7-11 with a formal ADHD diagnosis. Similar to Prins et al. (2011), their study excluded children taking medication, as one

of their goals was to determine how much pure effect exercise exerted on the children's functioning. Other exclusion criteria consisted of IQ < 70, and children with comorbid developmental disorders and health problems (Memarmoghaddam et al., 2016).

Treatment assignment and outcome measures of this study shared some similarities with other EF trials, using random treatment and control group assignment and cognitive performance measures including the Stroop test (Chacko et al., 2014; Shuai et al., 2017). Two outcome measures unique to the Memarmoghaddam et al. study were the additions of the Go-No-Go test to measure behavioral inhibition and the HRR heart rate monitoring system to identify the intensity of specific exercises in the trial (Memarmoghaddam et al., 2016). The authors selected the exercises to promote inhibition of response and behavior in an effort to improve the children's EF abilities. The total treatment sessions (29) were similar in number to other EF studies; however, the 90-minute exercise session length was unique—other EF treatments averaging 30 minutes (Memarmoghaddam et al., 2016). The sessions included various organized aerobic and sports exercises supervised by physical education professors. The authors collected pre- and post-treatment Stroop and Go-No-Go measures for both treatment and control groups, and compared them with a significance level set at $p < .05$ (Memarmoghaddam et al., 2016).

Study results showed that the selected exercise program had a significant effect on both outcome measures. The authors theorized that an important component of this study—responsible for producing a greater effect than that of previous studies—was its focus on exercise intensity, which may have increased the physiological response and subsequent EF performance (Memarmoghaddam et al., 2016). Limitations of the study included the fact that all of the study participants were boys and that, as in the Shuai et al. (2017) study, the children were

heterogeneous in terms of ADHD sub-type. Additionally, none of the participants were taking ADHD medications, which may have impacted performance (Memarmoghaddam et al., 2016).

Self-regulation strategies. Barkley (2015) asserted that research literature in past decades had overlooked the central role of self-regulation in ADHD. He posited that de-emphasis of this trait in favor of the three main ADHD symptoms of inattention, hyperactivity, and impulsivity (as outlined in *DSM-5*), and its subsequent omission from diagnostic descriptions, has contributed to the lack of extensive research in this area (Barkley, 2015). Another reason is that self-regulation is less observable as a distinct feature and, thus, more difficult to quantify (APA, 2013; Bunford et al., 2015). Barkley (2015) argued that the exclusion of self-regulation from diagnostic criteria was a product of incomplete scientific nosology. In fact, many authors involved in ADHD research support the idea that deficits in self-regulation are a core aspect of the disorder and need to be included in ADHD research and diagnosis (Bunford et al., 2015; Epstein & Weiss, 2012; Steinberg & Drabick, 2015; Sullivan et al., 2015; Tarver et al., 2014).

Sanders and Mazzucchelli (2013) pointed to the capacity for self-regulation, as measured in early childhood (as young as 3 years), to predict a wide variety of indices related to social competence and overall wellbeing that persist into adulthood. The authors identified studies linking regulatory abilities to positive outcomes in academic achievement, income, physical health, interpersonal relationships, and mental health (Sanders & Mazzucchelli, 2013). Specific to ADHD, self-regulation plays an important role in the delay of gratification, modulation of attention and motor behavior, and development of social adjustment (Healey & Halperin, 2015). In addition, parental self-regulation is a contributing factor, especially for parents of children with developmental delays. Parental self-regulation is a multi-component process that involves

elements of self-management, self-efficacy, problem-solving, personal agency, and self-sufficiency (Sanders & Mazzucchelli, 2013). Together, these skill areas increase parents' own ability to regulate emotions and behaviors, ultimately benefiting children struggling with regulatory control (Herbert & Esparham, 2017).

Children with ADHD are approximately 11 times more likely to display disruptive behaviors that represent impairment in self-regulation (Steinberg & Drabick, 2015). Difficulty regulating both affect and behavior contributes to externalizing behaviors and predisposes ADHD children to higher rates of oppositional defiant disorder (ODD) and conduct disorder (CD) (Tarver et al., 2014). Deficits in self-regulation also contribute to elevated rates of mood disorders in children with ADHD; and children with the disorder are more likely than non-ADHD peers to develop depression later in childhood and adolescence (Armstrong et al., 2015). Self-regulation also interacts with executive functions related to working memory and cognitive and behavioral response inhibition. Response inhibition allows children to control emotions, motivation, arousal and, ultimately, many of their behaviors (Barkley, 2015). The central role of self-regulation in the emotional and behavioral functioning of children with ADHD makes it an appropriate and necessary area of ongoing investigation. Emerging research (Barkley, 2015; Bunford et al., 2015; Steinberg & Drabick, 2015) explores strategies for increasing children's regulatory capacities with the goal that improvement in self-regulation will benefit overall ADHD symptomatology.

Self-regulation strategy research findings. Findings from studies designed to improve regulatory skills for ADHD children have shown promise. Studies consisted of a mix of multi-study reviews and individual trials, and utilized a variety of treatment designs and outcome measures. Similar to the Shuai et al. (2017) EF study, several self-regulation studies also

included high parent involvement, which may have contributed to positive outcomes (Herbert & Esparham, 2017; Sanders & Mazzucchelli, 2013).

Van der Oord et al. (2012) completed a study that included high parent involvement and evaluated the effectiveness of mindfulness training for a group of 22 children with ADHD. The purpose of the trial was to examine the efficacy of mindfulness training for both children with the disorder and their parents. The rationale for this was that ADHD symptoms for children are affected by parenting practices, and research thus far had demonstrated efficacy ($d = 0.42 – 0.50$) for mindfulness training in adult populations (Barkley, 2015). Van der Oord et al. (2012) pointed to the high heritability rates associated with ADHD, making it likely that parents of ADHD children also need to learn self-regulation techniques (Van der Oord et al., 2012). The hypothesis of the study was that mindfulness training would reduce both children's and parents' inattention and impulsivity/hyperactivity and, for the parents specifically, would reduce stress and over-reactivity (Van der Oord et al., 2012).

Haydicky et al. (2015) likewise emphasized the importance of parent involvement in helping children build regulation skills. In contrast with Van der Oord et al. (2012), the Haydicky et al. (2015) study focused less on ADHD symptoms for parents and children, and more on the high levels of stress between children with ADHD and their parents. The rationale for this was that parental stress is associated with high levels of parent-child conflict, poor psychological adjustment for children, and high levels of ADHD symptoms (Haydicky et al., 2015).

The Van der Oord et al. (2012) study included children between the ages of 8-12, with an IQ over 80, with a diagnosis of ADHD (and no comorbid developmental disorders). Children taking medication were accepted but changes in dose or medications were not allowed during

treatment phase. The study used a quasi-experimental waitlist control (n = 11), with those children waiting an average of 9 weeks before start of treatment. The study included pretest, posttest, and an 8-week follow up (Van der Oord et al., 2012). Measures included Disruptive Behavior Disorder Rating Scale (DBDRS), Parenting Stress Index (PSI), the Parenting Scale (PS), Mindfulness Attention and Awareness Scale (MAAS), and the ADHD Rating Scale (ARS). The authors conducted treatment for the children in highly structured small groups that met 8 times per week for 90 minutes each session. Sessions followed the precepts of mindful child training (MC) which focused on self-awareness, self-control, and mindfulness in difficult situations. Parents participated in a parallel training program and were taught mindful parenting (MP) techniques including presence, acceptance, and self-care (Van der Oord et al., 2012).

Van der Oord et al. used a token reward system similar to the one used by Haydicky et al. (2015) to enhance motivation and participation in the program (Van der Oord et al., 2012). In addition, the authors gave parents and children homework forms and a CD with mindfulness exercises to practice at home. Trainers with extensive experience in mindfulness delivered all of the treatment (Van der Oord et al., 2012). The authors analyzed outcome data using multilevel modeling with four coefficients (waitlist, pre-test, post-test, and follow-up test). They then evaluated treatment effects with *t*-tests at a significance level of .05 (Van der Oord et al., 2012). Results showed a large reduction in inattention (ES = 0.80) and a medium reduction in hyperactivity/impulsivity (ES = 0.56) from pre- to post-test. The study found small but significant reductions in the same symptoms in the parents (ES = 0.36 & 0.48 respectively). In addition, ratings for mindfulness among the parent group increased slightly (ES = 0.28) (Van der Oord et al., 2012).

Though the Van der Oord et al. (2012) study shared a similar focus on mindfulness and parent involvement with Haydicky et al. (2015), their study designs differed substantially. Haydicky et al. (2015) used an adaptation of mindfulness-based cognitive therapy (MBCT) on a group of children and their parents to ascertain whether the intervention had an impact on ADHD symptoms, functional impairment, family functioning, and parental stress (Haydicky et al., 2015). The mindfulness exercises in the study focused on two aspects of self-regulation: regulation of attention through deliberate focus, and regulation of emotion through the development of acceptance and non-reactivity. The authors described this type of mindfulness as an aspect of metacognition, an EF skill involving consciously monitoring cognitive and emotional processes (Haydicky et al., 2015). Unlike Van der Oord et al. (2012), Haydicky did not use a waitlist control or complete post treatment follow-up measures. In addition, the Haydicky study used different rating scales to measure outcomes. The Van der Oord et al. (2012) and Haydicky et al. (2015) studies, however, did share the common element of a token or point-based reward system to increase motivation in participating children (Haydicky et al., 2015; Van der Oord et al., 2012).

The Haydicky et al. (2015) study included 18 children (boys and girls) between the ages of 13-18, and 17 parents. The children all had a diagnosis of ADHD (mixed subtypes) and an IQ > 85 on the WAIS. Sixty-one percent of the children were taking ADHD medication upon enrollment in the program, and 78% reported a comorbid diagnosis. Exclusion criteria included children with ASD, youth with severe behavioral problems, and children living outside of the home—i.e., in residential treatment (Haydicky et al., 2015). The program consisted of a month-long baseline period that allowed for differentiation of treatment effects from placebo or other factors. The children and parents participated in eight sessions of the MYmind program, a

mindfulness-based, manualized group treatment program for children with ADHD and their parents. MYmind aims to foster mindfulness in daily life activities and improve coping with ADHD symptoms, emotions, and family stress (Haydicky et al., 2015).

Parents and children received psychoeducation about mindfulness in initial sessions through videos, discussions, and home exercises outlined on an instructional CD. Program facilitators attended a 12-week mindfulness course and practiced mindfulness meditation regularly (Haydicky et al., 2015). The authors collected data at four time points: baseline, pre-test, post-test, and 6-week follow-up. Outcome measures consisted of Conners parent report and self-report scales, Revised Child Anxiety and Depression Scale (RCADS), the Stress Index for Parents of Adolescents (SIPA), Family Assessment Device (FAD), Acceptance and Action Questionnaire (AAQ), and the Interpersonal Mindfulness in Parenting Scale (IM-P). Statistical analysis included assessing effect size (ES) using Cohen's *d*, with values near 0.2 = small, 0.5 = medium, and over 0.8 = large (Haydicky et al., 2015).

Both Van der Oord et al. (2012) and Haydicky et al. (2015) achieved positive study results, with Van der Oord et al. (2012) pre- to follow-up tests revealing significant reductions in ADHD symptoms, with small to large ES. Teacher ratings were not significant, except for a small ES (0.39) for inattention (Van der Oord et al., 2012). Results of the Haydicky et al. (2015) study indicated that MYmind treatment was associated with reductions in conduct problems and inattentiveness for participating children. This study also showed improvements in peer relations for the children and increased mindfulness and decreased stress for participating parents (Haydicky et al., 2015). Their treatment evaluation also demonstrated that the program was feasible in cross-cultural contexts, and that the MYmind program could help children with ADHD improve symptoms. The authors observed medium/large (d = 0.62) ES for improved

inattention, (d = 0.70) ES for decreased externalizing behaviors, (d = 1.07) for improvement in peer relations, and (d = 1.01) for improvement in externalizing problems. In addition, parents reported significant improvements (d = 0.81) in parenting stress and mindful parenting. Paired t –tests also indicated that improvements for both the children and parents were maintained at 6 week follow-up (Haydicky et al., 2015).

Van der Oord et al. (2012) came to similar conclusions as Haydicky et al.—that mindfulness training produced reductions in ADHD symptoms on parent ratings comparable to traditional behavioral parent training (Haydicky et al., 2015; Van der Oord et al., 2012). Furthermore, the significant reduction in parents' ADHD symptoms, parenting stress, and over-reactivity proved especially useful, as parent participation in therapy is often inconsistent due to the parents' own symptoms. Both Haydicky et al. (2015) and Van der Oord et al. (2012) recommended that research continue on treatments simultaneously helping ADHD children and parents with better coping skills.

The Van der Oord et al. (2012) and Haydicky et al. (2015) studies shared some common design limitations, including that the mindfulness training required trainers experienced in both mindfulness practice and the MC/MP or MyMind programs. In addition, the sample sizes were fairly small, parents in both studies were not blind to treatment, and the studies did not employ active randomized control conditions. Due to these limitations, the authors for both studies stated that non-specific treatment factors or expectancy effects may have affected the outcomes (Haydicky et al., 2015; Van der Oord et al., 2012).

In contrast to the individual trials, both Herbert and Esparham (2017) and Sanders and Mazzucchelli (2013) completed systematic reviews of programs designed to facilitate regulatory skills for children with ADHD. Each study, however, focused on different regulatory treatments

with Herbert and Esparham (2017) evaluating the efficacy of yoga, Tai Chi, physical activity, and mindfulness, while Sanders and Mazzucchelli (2013) evaluated studies utilizing the Positive Parenting Program (PPP) to help parents develop regulatory skills. The Herbert and Esparham (2017) study reviewed literature on mind-body therapy techniques that may affect ADHD symptoms for children, with the rationale that children with ADHD need interventions that are easy to implement and possess few side effects. The authors included aspects of self-regulation, such as the regulation of affect, motivation, and arousal in their study due to the central role self-regulation plays in the behavioral and executive impairments associated with ADHD (Herbert & Esparham, 2017). The focus on regulation as a component of broader EF skills is consistent with the Haydicky et al. (2015) assertion that regulatory skills are the product of metacognition, which allows for self-monitoring and, thus, self-regulation.

One such activity is the practice of yoga, which aims to integrate the body and mind—providing improvements in self-control, attention, awareness, and adaptive skills. In addition, for ADHD children, yoga includes aspects of exercise and meditation, which the authors suspected may decrease hyperactivity and inattention (Herbert & Esparham, 2017). The relatively few studies on the topic have methodological flaws such as small sample sizes, no randomization, and do not use control groups (Haydicky et al., 2015; Van der Oord et al., 2012). Nonetheless, Herbert and Esparham (2017) documented significant benefit from yoga practices for this population. The authors observed up to 35% reduction in symptoms from pre- to post-test on measures of ADHD symptoms for both medicated and non-medicated children. In addition, parent ratings showed improvements in self-esteem, sleep, anxiety, and parents' own ability to handle their children's behaviors (Herbert & Esparham, 2017).

The authors also found moderate effect sizes (ES = 0.77) for improved on-task behavior in school when students participated in several weeks of yoga training (Herbert & Esparham, 2017). Yoga practice also proved effective when it included breathing and relaxation techniques to improve focus and decrease hyperactivity. The authors postulated that positive effects from yoga participation stemmed from improvements in mood, body awareness, and breathing (Herbert & Esparham, 2017). The symptomatic improvement Herbert & Esparham (2017) observed paralleled Van der Oord et al. (2012), who had found that helping children practice increased self-awareness led to improvements in attention and behavioral control. Despite methodological limitations, Herbert and Esparham (2017) concluded that yoga had a positive effect on self-regulation and ADHD symptoms (Herbert & Esparham, 2017).

Herbert and Esparham (2017) also reviewed studies exploring the efficacy of Tai Chi, which consists of slow movements and coordinated balance and breathing exercises. The authors speculated that the actions of Tai Chi might reduce ADHD symptoms and anxiety, similar to the intervention targeted in the Haydicky et al. (2015) study. Herbert and Esparham concluded that outcome measures such as the Conners rating scales showed that at least two 30-minute Tai Chi sessions per week for a minimum of five weeks significantly decreased anxiety, daydreaming, hyperactivity, and negative conduct (Herbert & Esparham, 2017). In addition, Tai Chi increased emotional regulation significantly, with improved scores persisting at two-week follow-up (Herbert & Esparham, 2017). The authors hypothesized that the positive impact on aspects of executive functioning for children with ADHD may stem from the focused attention and regulation of physiology that Tai Chi facilitates. These outcomes regarding improved EF regulatory functions through physiological exertion and control are consistent with the Memarmoghaddam et al. (2016) findings that physical movement increased both cognitive and

behavioral inhibition in children with ADHD. Herbert and Esparham (2017) concluded that Tai Chi may have beneficial effects as an adjunct therapy for children with ADHD (Herbert & Esparham, 2017).

Physical activity was also central to the Herbert and Esparham (2017) meta-analyses surveying the impact of exercise on the executive and regulatory capacities of children with ADHD. The authors based their inquiry on the fact that physical exercise is known to increase cerebral blood flow in the prefrontal regions and to regulate dopamine function, both of which are implicated in ADHD impairment (Herbert & Esparham, 2017). The authors found that physical activity increased EF/regulation capacities with an effect size of 0.53 (Herbert & Esparham, 2017). Children participating in martial arts, which also involves high levels of physical activity, also showed significant effect sizes (0.88, $p < .05$) for improvement in executive and regulatory functions (Haydicky et al., 2015). This supports the Herbert and Esparham (2017) findings on increased EF regulation through forms of physical exertion. As in other self-regulation studies, both authors concluded that evidence for the effect of physical activity on self-regulation was limited in quantity and quality, but that this research showed promise in helping decrease regulatory symptoms (Haydicky et al., 2015; Herbert & Esparham, 2017).

The Herbert and Esparham (2017) multi-study review also looked at studies exploring mindfulness and meditation as adjunctive treatments for children with ADHD. Corroborating the findings of Van der Oord et al. (2012), the authors concluded that mindfulness and meditation practice created significant improvements in symptoms of inattention involving alerting, orienting, and conflict-monitoring in children with ADHD (Herbert & Esparham, 2017). Meditation also created measurable changes in brain-wave activity for participants in one study

comparing meditation to non-meditative relaxation for 20-minute intervals. Results showed significant increases in alpha and theta EEG activity in all brain regions for the meditation group. The authors found that, since abnormal brainwave activity had been linked to ADHD in children, meditation served as a useful mind-body therapy for children with the disorder (Herbert & Esparham, 2017). This stance was supported by studies by Loo & Makeig (2012) and González-Castro et al. (2016) who stated that imbalances in theta/beta brainwave patterns were indeed linked to both behavioral and EF deficits in children with ADHD.

Herbert and Esparham's extensive 2017 analysis of self-regulation interventions found that emerging mind-body therapies do, indeed, show evidence of efficacy in the treatment of ADHD symptoms in children, including enhancing self-regulation. The authors concurred with Barkley (2015) that advantages of these techniques include the lack of side effects, minimal cost, ease of practice at home or school, and guidance through videos and online resources (Barkley, 2015; Herbert & Esparham, 2017). Limitations of the Herbert and Esparham (2017) studies were similar to Haydicky et al. (2015) and included small sample sizes, lack of control groups, and subjective parent/teacher reporting for outcomes.

Sanders and Mazzucchelli (2013) also conducted a multi-study review, but unlike the Herbert and Esparham (2017) study they focused solely on parenting programs that evidenced efficacy in promoting self-regulation. The authors identified the Positive Parenting Program (PPP) as one such intervention that proved effective in improving children's regulatory capacities. The program targeted parent regulation as a central goal, which Bunford et al. highlighted as an important focus for improving family dynamics (Bunford et al., 2015; Sanders & Mazzucchelli, 2013). The PPP program uses a range of strategies to enhance children's self-regulation skills by helping parents model and apply techniques to their own lives. Some of the

working points of the program include modeling for parents how to manage situations in non-judgmental ways and helping them focus on solving problems as they arise. Other areas of focus include helping reinforce and generalize regulation skills across tasks and settings, formulating plans for challenging events, encouraging self-evaluation, and fading support as parents increase their ability to regulate their own behavior more successfully (Sanders & Mazzucchelli, 2013).

The Sanders and Mazzucchelli (2013) study produced positive results and the authors concluded that together these parent-focused regulation strategies promoted more adaptive parenting responses and encouraged better self-regulation for both parents and their children (Sanders & Mazzucchelli, 2013). This study shared a limitation with several of the studies in the Herbert and Esparham (2017) review, namely that authors provided qualitative description of improvements in self-regulation compared to controls on several measures without providing statistical data to support the outcomes (Herbert & Esparham, 2017; Sanders & Mazzucchelli, 2013).

One self-regulation study differed significantly from the others with regard to the age of the children receiving the intervention. Healey and Halperin (2015) focused on the early development of regulatory capacities to show how primary deficits in regulation contribute to ADHD symptoms and executive challenges. This focus is consistent with the Memarmoghaddam et al. (2016) EF study highlighting the association of poor self-regulation with deficits in executive functioning. Healy and Halperin expanded their intervention, though, to target a broader range of regulation skills with the rationale that deficits in self-regulation include cognitive, emotional, and behavioral aspects of inhibition (Healey & Halperin, 2015).

This supports conclusions by Steinberg & Drabick (2015) who stated that regulation of emotion begins in early childhood as the product of an integration of cognitive, motor, and executive inhibitory controls.

Healey and Halperin (2015) devised an early intervention program titled Enhancing Neurobehavioral Gains with the Aid of Games and Exercise (ENGAGE) (Healey & Halperin, 2015). In this novel study, Healy and Halperin targeted behavioral, emotional, and cognitive aspects of self-regulation, with an emphasis on development of internal self-regulation through active interactions with others (Healey & Halperin, 2015). This approach shared similarities with self-regulation studies that included high parent involvement and practice with others (most often parents) to master regulatory skills (Haydicky et al., 2015; Van der Oord et al., 2012). The Healey and Halperin (2015) program used variations of well-known children's games, adapting them to promote aspects of self-regulation. Behavioral regulation games included musical statues, paced skipping, and Simon Says. Emotion regulation activities included relaxation training and deep breathing; games such as puzzles, cups memory, and the "copy me" game taught the children attentional control (Healey & Halperin, 2015, p. 469).

The Healey and Halperin study involved a group (N = 25) of children (boys and girls) aged three to four years, identified with early ADHD-like symptoms and described generally as *difficult to manage* with regard to self-regulation. The children's parents also participated in the study. The authors hypothesized that targeting developmentally young children with an intervention aimed at multiple aspects of self-regulation, with parent involvement, would produce increases in behavioral regulation and cognitive control as measured on outcome ratings (Healey & Halperin, 2015). Inclusion criteria incorporated parent ratings of disruptive behavior and hyperactivity. Exclusion criteria shared similarities with several other regulation studies,

such as IQ < 80, a pervasive developmental disorder, or children taking systemic medication for a chronic medical condition (Chacko et al., 2014; Healey & Halperin, 2015; Van der Oord et al., 2012). The program consisted of five weeks of parents and children attending weekly 90-minute groups, where they learned new games and activities targeting aspects of regulation. The authors also asked parents to play the games with their children for 30 minutes each day at home (Healey & Halperin, 2015). The authors administered pre- and post-treatment outcome measures that included monitoring of behavioral symptoms (hyperactivity, aggression, attention) through the use of the BASC-2 at six intervals throughout treatment (pre-treatment, post-treatment, and 1, 3, 6, and 12 month follow-up). Neurocognitive assessment included Stanford Binet (SB-5) and NEPSY-2 for evaluation of overall functioning and motor and inhibitory control (Healey & Halperin, 2015). Similar to the Bikic (2016) and Beck et al. (2010) studies, a secondary analysis at follow-up intervals sought to determine the durability of gains. The study authors used a control group of typically developing children to account for practice effects. The control group children received identical testing as the experimental group, but without treatment intervention. Healey & Halperin (2015) calculated effect sizes using analysis of variance (ANOVA) for parent behavioral ratings at the six time points. Paired-sample *t*-tests compared pre- and post-intervention scores on children's neurocognitive functioning.

Results showed significant improvements in the children's self-regulation abilities after the 5-week treatment program, as measured by parent ratings. Similar to Van der Oord et al. (2012), the children maintained gains throughout follow-up, but for a significantly longer period. While the Van der Oord et al. (2012) study demonstrated regulatory effects at eight-week follow up, the Healey & Halperin (2015) trial showed significant improvements up to 12 months after treatment. Furthermore, the children participating in the ENGAGE program showed improved

working memory and inhibitory control after five weeks of treatment. In the neurocognitive domain, treatment produced improvements in both working memory and visuomotor precision (Healey & Halperin, 2015).

This study shared a similar limitation of time restrictions with several other regulation studies, which may have limited treatment gains (Haydicky et al., 2015; Healey & Halperin, 2015; Herbert & Esparham, 2017). Healy and Halperin noted that this is especially true for neurocognitive skills, which take longer to develop in young children, and may be the reason the program produced fewer gains in the neurocognitive domain (Healey & Halperin, 2015). Chacko et al. (2014) shared a similar observation that neurocognitive gains may be slower to integrate and so may not be visible in early outcome measures. Additionally, Healey and Halperin (2015) based their results on parent reports, which are prone to expectancy effects, and the trial also did not have a matched control group. Still, the authors shared similar conclusions with Sanders & Mazzucchelli (2013), that their self-regulation intervention was as effective as behavioral management programs used to treat disruptive and hyperactive children. Furthermore, the authors noted that the ENGAGE program also produced neurocognitive improvements comparable to other interventions promoting growth in these areas for young children (Healey & Halperin, 2015).

Chapter IV: Discussion

Efficacy of Emerging Treatments

Neurofeedback. There is clear evidence that children with ADHD display abnormal EEG patterns, most often with increased theta wave activity and decreased beta wave activity (Loo & Makeig, 2012). Stimulant medications increase overall beta frequencies and reduce ADHD symptoms, and NF studies show that NF treatment also holds the potential to reshape

brainwave activity through operant conditioning biofeedback techniques (Bakhshayesh et al., 2011; Liechti et al., 2010). The aim of neurofeedback training is to balance brainwave patterns to improve attention and behavioral control—similar to ADHD medications, but without the negative side effects (Lofthouse et al., 2012).

The reviews of neurofeedback (NF) studies showed mixed results—with older, less methodologically rigorous trials showing more promise, and newer RCTs showing less efficacy. Evans et al. (2014) reviewed the literature on training interventions and concluded that neurofeedback met criteria for a level 3 (possibly efficacious) evidence-based treatment (EBT) (Evans et al., 2014). The five EBT levels that summarize both research and clinical consensus on the efficacy of therapeutic treatments are: 1- well established, 2-probably efficacious, 3-possibly efficacious, 4- experimental treatment, 5- no evidence of efficacy (Evans et al., 2014; SAMHSA, 2017). Studies such as the Gevensleben et al. (2009) trial concluded that NF treatment for children with ADHD may be efficacious due to medium effect sizes (0.06) at six-month follow-up (Gevensleben et al., 2009). Other studies, such as Bakhshayesh et al. (2011), also found evidence of improved parent and teacher symptom ratings following NF treatment. The study produced medium to large ES (0.5 – 0.8) and the authors concluded that NF treatment had a significant impact on reducing beta/theta ratios, corresponding with decreased ADHD symptoms. The study by Liechti et al. (2010) reached similar conclusions—that NF treatment for children in the study produced both increased resting EEG activity and behavioral improvements on parent rating scales.

Results from studies included in this dissertation, however, were not uniformly positive, with recent reviews—such as Loo and Makeig's 2012 critical review of aspects of experimental methodology (i.e., types of controls)—finding a lack of broad support for the efficacy of NF

treatment. The authors concurred with other studies that NF treatment appeared to be as effective as other treatments (e.g., CBT) that do not yet have an evidence base for application to children with ADHD (Loo & Makeig, 2012). Since experimental methodology has important implications for reliability and generalizability of results, recent reviews such as the Lofthouse et al. (2012) study focused on outcomes from studies that were well-constructed, utilizing triple blinding and large sample sizes. Their findings were positive, but conclusive only for probable efficacy due to methodological weaknesses in many of the studies in their survey. Zuberer et al. (2015) also discussed the importance of NF studies using quality-controlled NF instruments with uniform algorithms to produce consistent and reliable results.

Blinding during treatment appears to be an additional significant factor influencing study results. Several studies reported low confidence in even moderate effect sizes due to the potential for bias from expectations or investment in the treatment by outcome raters and practitioners (Sonuga-Barke et al., 2013; Vollebregt et al., 2014). When Sonuga-Barke et al. (2013) adjusted for blinding in treatment assessment, the ES decreased to 0.30 and lost statistical significance. Likewise, the study and systematic review by Vollebregt et al. (2014), which followed RCT protocol, concluded that their own study and the majority of the studies they reviewed failed to show significant improvement from NF treatment.

Vollebregt et al. (2014) also made a distinction between types of cortical arousal among children with ADHD. The authors noted that most NF protocols targeted the most common frequency profile for children with the disorder—those with excess theta activity that are considered cortically "underaroused" (Vollebregt et al., 2014, p. 461). However, Loo & Makeig (2012) suggested that, similar to cognitive and behavioral symptoms, significant heterogeneity likely exists with regard to children's individual EEG profiles. For example, a minority of

children with ADHD display an overaroused frequency pattern, with high levels of beta activity and lower than average levels of alpha and theta frequencies (Bakhshayesh et al., 2011). Indeed, the majority of studies I surveyed for this review targeted the underaroused profile, with the goal of increasing beta and decreasing theta activity (González-Castro et al., 2016; Liechti et al., 2010; Lofthouse et al., 2012; Vollebregt et al., 2014). The fact that most studies appear to target the underaroused profile may leave a small but significant subgroup of ADHD children understudied in NF research, which then limits generalizability of NF study results to the broader population of children with ADHD.

Another inconsistency among the NF studies I surveyed was that several of the trials did not include transfer conditions in their methodology (Bakhshayesh et al., 2011; González-Castro et al., 2016). Transfer conditions assess subjects' ability to regulate their own brainwave activity without the presence of immediate training feedback. Zuberer et al. (2015) emphasized that including transfer trials in NF studies added ecological validity to standard feedback conditions because transfer trials show whether subjects are able to sustain brainwave changes beyond the immediate feedback conditions. Lack of transfer conditions makes it unclear whether positive NF results can be generalized beyond treatment conditions that provide immediate performance feedback (Loo & Makeig, 2012).

Consensus places neurofeedback as an emerging treatment that is probably efficacious for children with ADHD. Mixed results appear to be the product of study limitations and potential influence from uncontrolled factors such as parent expectancy. Still, some encouraging data shows that NF treatment may be an important adjunct treatment in situations where trained practitioners and required technology are accessible (Bakhshayesh et al., 2011; Lofthouse et al.,

2012). As more rigorous studies are completed, a more consistent outcome presentation may emerge with clearer results regarding the efficacy of neurofeedback for childhood ADHD.

Executive function treatments. Treatments targeting executive functioning (EF) are gaining in number as consensus grows regarding the centrality of executive deficits in ADHD (Barkley, 2015). Deficits in working memory, set shifting (cognitive flexibility), inhibition, and planning/organizing are some of the main areas of impairment in children with ADHD (Memarmoghaddam et al., 2016). Evidence is emerging suggesting that programs targeting these specific skills may improve functioning for children with the disorder (Rapport et al., 2013). Several authors question whether skills attained through EF treatments can produce far-transfer effects, gains persisting both beyond the timeframe of training sessions, and beyond treatment conditions (Barkley, 2015; Rapport et al., 2013).

Studies exploring the efficacy of EF treatments produced variable results, showing a range of effect sizes from insignificant to large (Epstein & Weiss, 2012; Tarver et al., 2014). This may be, in part, due to the fact that executive functions comprise a range of skills likely corresponding to a variety of underlying neuropsychological processes (Rapport et al., 2013). Various EF treatments target highly specific aspects of executive functioning and, thus, may produce efficacy data only for that skill area. Working memory (WM) is one area of EF training that produced moderate to strong effect sizes (0.76 – 1.49) in studies that targeted both WM alone (Beck et al., 2010) and as part of a program designed to improve a range of executive functions (Van der Oord et al., 2014). The success of both the single WM and more general WM/EF skills training may stem from the common use of computer/video game format to deliver the training, as video electronics provide cortical stimulation and may enhance cognitive

performance for children with ADHD (Barkley, 2015). The Beck et al. (2010) and Van der Oord et al. (2014) programs also asked parents to encourage their children to complete the training exercises. Volkow et al. (2011) identified motivational deficits in children with ADHD (likely due to low dopamine levels) so parent encouragement may have impacted the children's motivation and thus performance on EF training tasks.

Additional training programs were developed to help improve EF skills for ADHD children. Meta-analysis of multiple studies using the FIT program to create improvements in the four main areas of EF (WM, set shifting, inhibition, attention) did not show promising results (Rapport et al., 2013). Likewise, the Chacko et al. (2014) exploration of the CWMT working memory program for ADHD children produced similar non-significant results—with the exception of some near-transfer WM storage effects. Both Rapport et al. and Chacko et al. identified the lack of generalization of EF skills to more complex tasks as one of the main reasons the study results were not significant. This lack of generalization may be the reason both studies found some benefit from the EF programs but did not find that the skills learned generalized enough to significantly lower scores on parent and teacher ratings for academic performance and ADHD symptoms (Chacko et al., 2014; Rapport et al., 2013).

The Sonuga-Barke et al. (2013) meta-analysis showed initial positive results for EF training, but the authors suspected that parent non-blinding might have had a significant effect on outcome measures in the studies they reviewed. When the authors accounted for effects produced by the subjectivity of non-blinded raters (parents), treatment effects dropped from significant to insignificant levels (Sonuga-Barke et al., 2013). Cortese et al. (2015) confirmed the Sonuga-Barke et al. (2013) results with a similar study that accounted for whether outcome ratings were from the most proximal source (parent)—who may have had an investment in the

outcome—or a blinded rater. Cortese et al. (2015) observed a similar drop in effect when the studies were controlled for rater blinding, and concluded that—except for setting-specific WM improvements—EF training did not produce significant effects.

Results of these studies highlight the need for rater blinding in ADHD research to delineate effects produced by the treatment itself from ratings that contain bias due to the investment of the rater. The Cortese et al. and Sonuga-Barke et al. EF studies also identified a lack of generalization beyond the training setting, which corresponds to similar concerns in the Chacko et al. study (Chacko et al., 2014; Cortese et al., 2015; Sonuga-Barke et al., 2013). The lack of generalization may stem from a number of variables including the specific working memory task used in the study. Some WM tasks (simple, sequential, span) are more prone to training effects than others (spatial, verbal) and so may produce stronger treatment results (Chacko et al., 2014). In addition, there is the issue of whether the EF training task difficulty levels were adapted to the individual abilities of the children in the studies. Van der Oord et al. emphasized that adjusting the task difficulty, especially in WM training, was essential for improving EF skills through training (Van der Oord et al., 2014). Both the Cortese et al. (2015) and Sonuga-Barke et al. (2013) meta-analyses noted that some of the studies adjusted task difficulty while others did not. This factor may also have contributed to the strength of EF training results, especially with regard to working memory training (Cortese et al., 2015; Sonuga-Barke et al., 2013).

Recent research has continued to produce mixed results, with studies such as Bikic (2016) finding little improvement in EF following cognitive training for ADHD children. Still others, such as Shuai et al. (2017), did find significant improvements in all EF categories, as well as better behavior as reported by parents. Shuai et al. speculated that the difference between

theirs and previous studies on EF treatment may have been their targeting of a range of EF skills instead of one specific EF deficit. However, previous reviews such as the Rapport et al. (2013) meta-analysis of studies targeting multiple EF domains showed no significant efficacy. A more plausible explanation may be that the Shuai et al. (2017) study employed the additional elements of high parent involvement and real-life practice of skills in the home environment, which may have helped produce more generalizable, far-transfer effects. As is the case with many emerging treatment studies, lack of consistency in experimental methods for EF trials—i.e., rater blinding, control for practice effects, skills targeted, etc.—appear to have an impact on whether or not treatment outcomes have the power to reach beyond near-transfer effects and produce generalizable results. Consensus among studies appears to show little support for EF treatments as a stand-alone therapy, and places EF treatments for children with ADHD in the EBT category of possibly efficacious (Rapport et al., 2013; Shuai et al., 2017). Clearly, more studies with rigorous methodology, especially for the tracking of far-transfer outcomes, is needed to clarify whether EF is an effective ADHD treatment.

Self-regulation strategies. Strategies for promoting self-regulation in children with ADHD may take several forms, as self-regulation is a skill that, like executive functioning, involves several types of abilities (Sanders & Mazzucchelli, 2013). The most common aspect of self-regulation is the ability to regulate emotional response; however, other aspects of self-regulation also fall under the general heading, such as the ability to modulate one's energy, arousal, and behavioral responses (Barkley, 2015). This multi-faceted definition impacts the survey of literature on the topic, as studies may focus on only one aspect of regulation. In addition, self-regulation has only recently appeared in the nosology of ADHD definition and treatment, and so lags behind in the amount of empirical research available. In this report I

explored the efficacy of mindfulness therapy, mind-body practices, physical activity, Positive Parenting Program (PPP), and the ENGAGE program as strategies for improving self-regulation in children with ADHD.

Mindfulness is associated with moderate (d = 0.42 – 0.50) efficacy for promoting positive health benefits for individuals (Barkley, 2015). The Haydicky et al. (2015) and Van der Oord et al. (2012) studies confirmed that these results were possible for children struggling with ADHD symptoms, including difficulties with self-regulation. The authors observed significant improvements in ADHD symptoms after mindfulness training, with the highest reductions in the areas of peer relations and externalizing problems (Haydicky et al., 2015; Van der Oord et al., 2012). In addition, Haydicky et al. (2015) also found that the treatment reduced parental stress. This is important because ADHD creates increased risk for parent-child conflict though a bi-directional process between the child's symptoms and the parent's reactivity to those symptoms (Sullivan et al., 2015). Treatment gains were sustained at 6-week follow-up, which suggest that far-transfer effects may be possible for this intervention (Haydicky et al., 2015). Of note, the Haydicky et al. (2015) study did not find significant benefits in the realm of general family functioning, even though parents reported decreased stress levels. This may indicate that reductions in externalizing behaviors for children with ADHD benefit them more in the social realm than in family life. It is also possible that the treatment length was sufficient to produce social improvements for these children but was not long enough to impact more entrenched family functioning dynamics (Haydicky et al., 2015).

Yoga, an intervention similar to mindfulness, encourages participants to practice increased self/body awareness while simultaneously engaging concentration. Herbert and Esparham (2017) found similar efficacy outcomes to mindfulness in their review of yoga studies

for children with ADHD. The benefits of yoga extend beyond basic ADHD symptoms, as the authors' analysis also showed significant (d = 0.77) improvements in the children's ability to stay on task with schoolwork. The same authors also found efficacy for Tai Chi adapted for children with ADHD, with reductions observed in ADHD symptoms and anxiety (Herbert & Esparham, 2017). Tai Chi is similar to yoga in that it promotes self-regulation through body control paired with concentration. This may provide not only a physical release for hyperactivity but also a way to promote regulation of impulses and physical behavior while improving concentration. Similarly, the Herbert and Esparham (2017) analysis showed a significant but less robust effect size for physical activity programs for children with ADHD. This may be due to the fact that—unlike yoga, martial arts, or Tai Chi—exercise programs do not equally engage concentration and awareness, which may be a key factor in promoting executive inhibition and overall regulation (Herbert & Esparham, 2017).

Early childhood programs geared toward promoting self-regulation for children with ADHD also show significant efficacy. Programs such as the Positive Parenting Program and the ENGAGE program encourage multiple aspects of self-regulation, including strategies for both children and parents (Healey & Halperin, 2015; Sanders & Mazzucchelli, 2013). Parent regulation is especially important in helping children with limited self-regulation capacities, as the influence of parent arousal has potentially more impact on children with ADHD than on other children (Bunford et al., 2015). In addition, early intervention programs that encourage parents to adjust their responses to the regulatory cues of their children promote an adaptable parenting style, which is associated with more positive, nurturing parenting practices (Sanders & Mazzucchelli, 2013).

Programs supporting self-regulation for young children are especially important given that longitudinal studies show that the majority of self-regulation abilities develop in the first three years of life and impact long-term developmental outcomes for children (Sanders & Mazzucchelli, 2013). Regulatory abilities also impact young children's success in preschool—a time when they are laying the groundwork for important social and academic skills (Healey & Halperin, 2015). Siegel (2012) outlines the importance of young children mastering "arousal recovery" (p. 260) as part of more general regulation skills. Arousal recovery refers to parents helping children regulate the physiological processes of emotional flooding and return to a calmer and more organized state (Siegel, 2012). This aligns with early childhood programs, such as the Positive Parenting Program, that emphasize helping young children with arousal regulating techniques that facilitate emotional recovery (Sanders & Mazzucchelli, 2013).

Limitations of Studies

Studies investigating emerging treatments for ADHD contain many limitations that impact confidence in their conclusions. Foremost, the number of studies available on specific treatments is limited; thus, few study replications are available. A limited pool of studies necessitates the inclusion of some trials with poor methodology in order to aggregate outcome data from multiple studies. Most of the studies have methodological limitations that may cast doubt on the strength of their results. For example, many studies on treatments for children with ADHD rely on outcome data provided by parents and teachers (through parent/teacher rating scales). These data are subjective and may contain expectancy effects due to the parent or teacher's investment in a positive outcome for the children (Gevensleben et al., 2010). Tied to this is the issue of participant blinding, especially with regard to raters. If a parent knows which treatment group a child is assigned to, the chances increase that the parent's own bias may

influence the outcome ratings. In fact, Sonuga-Barke et al. (2013) observed that outcome significance dropped substantially when they accounted for rater blindness in both neurofeedback and executive function studies. Other methodological flaws possibly contributing non-specific effects to studies were practice effects, concomitant use of medications and other treatments during trials, lack of control group, lack of randomization, and small sample sizes—all of which impact the ability to generalize results. With regard to controls, Lofthouse et al. (2012) stated that there may be ethical limitations with regard to studies that use sham- or placebo-controls, and caution that use of these controls may violate ethical standards by withholding the best possible treatment for children.

Other limitations stem not from study methodology but from the feasibility of the treatment, itself. For example, neurofeedback requires specialized training for the practitioner, a learning curve for the patient, and a significant amount of equipment—from computers with NF programs to electrodes and EEG equipment (Gunkelman & Johnstone, 2005; Loo & Makeig, 2012). EF treatments also rely heavily on technology—requiring the use of specialized computer programs to facilitate the cognitive training exercises and to process outcome data (Van der Oord et al., 2014). Even many of the non-technical self-regulation strategies such as Tai Chi, yoga, and meditation require specific training. Practitioners may need months or years of personal training and practice to develop mastery before they are ready to guide others effectively through these interventions (Herbert & Esparham, 2017).

Chapter V: Conclusion

ADHD is a complex disorder with multiple contributing factors and many areas of functional impact. Although a solid foundation of research exists and much is known about the etiology and options for treatment, many questions remain unanswered. One such question

concerns the benefits and efficacy of emerging treatments for children with ADHD. Three emerging treatments—neurofeedback, executive function training, and self-regulation strategies—may hold potential for helping treat deficits associated with the disorder. All of these treatments hold promise for children diagnosed with attention-deficit/hyperactivity disorder. However, efficacy levels among these treatments were mixed, with general consensus that none of the treatments were strong enough to be used as a stand-alone therapy (Barkley, 2015; Herbert & Esparham, 2017; Lofthouse et al., 2012).

Treatments such as neurofeedback and EF training (with high parent involvement and in-home practice) fall into the probably efficacious range of evidence-based treatments and are useful as one part of a multi-faceted treatment approach (González-Castro et al., 2016; Shuai et al., 2017). Some of the most encouraging and consistent results came from studies on self-regulation strategies. Mindfulness, yoga, Tai Chi, and exercise programs have shown significant positive effects on ADHD symptoms (Healey & Halperin, 2015; Herbert & Esparham, 2017). However, research in these areas is limited and some of the methodology is flawed; thus, rigorous additional studies might confirm their utility with this population.

Directions for Future Research

Future research should focus on far-transfer effects for neurofeedback and EF trainings, as substantial differences in outcomes exists when analysis is separated between most proximal (parents) and probably blinded (teachers, clinicians) outcome raters (Cortese et al., 2015; Sonuga-Barke et al., 2013). In addition, future studies must use ecologically valid training approaches to increase far-transfer effects that generalize and produce integrated and long-term changes for ADHD children (Cortese et al., 2015). Studies will also attain better treatment

results by targeting a broader range of neuropsychological deficits, rather than focusing solely on one or two areas of training—such as working memory and sustained attention (Cortese et al., 2015; Shuai et al., 2017).

Future research must develop studies with methodological strength, such as utilizing full blinding, randomization, control groups, and adequate sample size. Combining outcome data from parent/teacher reports—which may be significantly subjective—with standardized neuropsychological measures would also help support the validity of outcomes (Beck et al., 2010). Additionally useful would be studies differentiating between children with specific sub-types of ADHD, as current available studies over-represent children with the hyperactive/impulsive presentation due to their higher rates of disruptive behavior (Barkley, 2015). For example, future NF studies could tailor specific neurofeedback protocols for each ADHD subtype's established qEEG patterns. This would allow researchers to find the most effective target frequencies for each ADHD subtype and develop subtype-specific NF treatment interventions (Zuberer et al., 2015).

Several authors have recommended research in specific areas to broaden the knowledge base—and, thus, the availability—of treatments for children with ADHD. To that end, Balázs and Keresztény (2014) and Hong et al. (2014) recommended that future research focus not only on children with full-syndrome ADHD, but on subthreshold ADHD, as well. They asserted that, due to both the prevalence rates and clinical impairment of children with this level of ADHD, treatment research should include this population. Balázs and Keresztény (2014) speculated that subthreshold ADHD may someday replace the "unspecified" categories in *DSM,* as research continues to show that ADHD is not a categorical disorder but, instead, a dimensional one. If this is true, then specific treatment strategies for subthreshold ADHD should be investigated, as

well. In addition, treatment studies are needed for younger children with ADHD symptoms, as less research is available on possible effective strategies for this population (Healey & Halperin, 2015; Tarver et al., 2014).

Banaschewski et al. (2010) highlighted the need for research into the specific genetic components involved in ADHD. While the evidence for heritability is strong, the exact mechanism for genetic transmission needs more investigation. Barkley (2015) also pointed to developments in epigenetic research showing the impact of experience—first on the genome, and later, on the phenotype—of ADHD. He identified the need for continued studies on G X E to identify environmental contributions to the expression of ADHD genetics (Barkley, 2015). Tarver et al. (2014) also advocated for future research to identify how environmental factors heighten risk for ADHD—specifically in those with genetic vulnerability. Likewise, Sullivan et al. (2015) recommended investigations into sex-specific methods of transmission (fathers to sons, etc.), and the interactions between family risk and early exposure to deficits in nutrition, maternal stress, and early caregiving practices.

The impact of ADHD on self-regulation is another area ripe for research. Deficits in self-regulation are now established as central to ADHD, but many of the underlying processes are still not well understood (Barkley, 2015). For instance, it remains unknown whether self-regulation deficits represent *top down* fronto-limbic dysfunction, such as failure of the lateral and medial PFC and anterior cingulate to regulate lower level limbic activity. Alternately, affect regulation deficits may stem from a *bottom up* process, such as over-activation of limbic structures (amygdala, hippocampus, ventral striatum) (Bunford et al., 2015; Siegel, 2012).

Research into cultural influences on the expression, diagnosis, and treatment of ADHD is another area in need of attention (AAP, 2011). Barkley (2015) advised that outcome measures

be normed for the particular country of residence, with study sampling representing the ethnic backgrounds of the actual population so as not to over-diagnose minority children through use of data normed on dominant groups. Barkley (2015) emphasized that the expectations of individual cultures and the consistency of cultural parenting practices influence the threshold for deviance (and diagnosis), in addition to the goals and treatment preferences of families seeking help for ADHD. Therefore, such factors must be considered in future studies and treatment programs (Fiks et al., 2013).

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