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**Non-pharmacological Interventions for Cognitive Difficulties in ADHD: A Systematic
Review and Meta-Analysis**

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Non-pharmacological interventions for cognitive difficulties in ADHD: A systematic review and meta-analysis

ABSTRACT

Attention deficit hyperactivity disorder (ADHD) is the most common neurodevelopmental disorder in children and is associated with significant risk of educational failure, interpersonal problems, mental illness, and delinquency. Despite a number of comparative and comprehensive reviews on the effects of ADHD treatments on ADHD core symptoms, evidence synthesizing the effects of ADHD interventions on cognitive difficulties is limited. In this meta-analysis, the neuropsychological effects of non-pharmacological interventions for ADHD were examined across studies published between 1980 and 2017. Data were extracted from studies that used objective cognitive measures (either computerized or pencil-and-paper), and multiple meta-analyses were conducted to compare the effectiveness across these interventions. Publication bias was assessed, as well as quality of the evidence, using Cochrane risk of bias tool for randomized control trials studies. Our final meta-analysis included 18 studies with interventions that were categorized into four categories: neurofeedback, cognitive-behavioral therapy, cognitive training, and physical exercises. Physical exercises demonstrated the highest average effect size (Morris $d = 0.93$). A further evaluation of cognitive functions yielded 49 effect sizes for the five categories, including attention, inhibition, flexibility, and working memory. Analyses demonstrated a homogenous, medium to large, effect size of improvement across interventions, with inhibition demonstrating the largest average effect size (Morris $d = 0.685$). This study highlights the positive effect of psychological interventions on ADHD cognitive symptomology and supports the inclusion of non-pharmacological interventions in conjunction with the commonly used pharmacological treatments.

Keywords: ADHD; executive function; inhibition; meta-analysis; cognitive impairment

1. Introduction

Attention deficit hyperactivity disorder (ADHD) is considered the most common neurodevelopmental disorder in children (American Psychiatric Association, 2013; Goldman et al., 1998; Polanczyk et al., 2007). The diagnosis of ADHD is three times more common in boys than in girls (9.2% vs. 3.0%;(Guevara & Stein, 2001). Although most frequently diagnosed during the school years, ADHD affects individuals across the lifespan (Polanczyk et al., 2007). In the long term, ADHD is associated with a significant risk of educational failure, interpersonal problems, mental illness, and delinquency (Biederman et al., 2006). Given that the global prevalence rate of ADHD is around 5% (Polanczyk et al., 2007; Wittchen et al., 2011), it creates a substantial burden on families, as well as on health, social care, and criminal justice systems (W. E. Pelham, Foster, & Robb, 2007). Etiology of ADHD is complex and multidimensional, combining genetic, psychosocial, and environmental factors (Faraone, 2000; Faraone & Biederman, 2005).

Traditionally, the primary neurocognitive deficits of ADHD were considered to be impulsivity, hyperactivity, and inattention (Halperin et al., 1990). This has been supported by several theoretical models (Willcutt, 2015), hypothesizing that ADHD may arise from dysfunctional responses to reward/punishment contingencies (Luman, Oosterlaan, & Sergeant, 2005), pronounced aversion to the experience of delay (E. J. S. Sonuga-Barke, Taylor, Sembi, & Smith, 1992), increased intraindividual variability in response time due to attentional fluctuation (Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003), and overall slow cognitive processing speed (McGrath et al., 2011). However, several authors have focused on a primary deficit in the executive function (EF) domain, as patients tend to display deficits in attentional and strategic flexibility, often fail to monitor and inhibit behavior effectively, and often display lower planning abilities and working memory (Alloway & Passolunghi, 2011; Barkley, 1997; Biederman et al., 2008; Brocki, Randall,

Bohlin, & Kerns, 2008; Castellanos & Tannock, 2002; Pfiffner et al., 2018). Executive functions are a collection of higher level capacities that enable flexibility in goal-directed behavior (Welsh & Pennington, 1988), including working memory, response inhibition, and set shifting (Miyake et al., 2000). Anatomically, executive functions have been primarily localized to the prefrontal cortex and secondarily to the temporal, parietal, and limbic lobes and the striatum (Roth & Saykin, 2004).

Epidemiologic studies report that as much as 50% of referrals to child mental health clinics are for assessment and treatment of ADHD (Salomone et al., 2015). In response to this demand, a plethora of measures have been designed to assess aspects of the triad of symptoms comprising ADHD (i.e., inattention, hyperactivity, and impulsivity; see Table 1). Most commonly, these symptoms are assessed using subjective measures aimed at the behavioral manifestations of the disorder, involving different procedures across stages of development, such as parent and teacher rating scales in children (Barkley, 1991) and self-report scales in adults (Kessler et al., 2005). It is generally accepted that parent and teacher rating scales are reliable and valid components of ADHD assessments (McGough & Barkley, 2004). Less commonly used (Dulcan, 1998) but rather more objective are laboratory measures, which measure the cognitive manifestations of the disorder and purport to distinguish between clinical and non-clinical cases (Barkley, 1991; Forbes, 1998; Greenberg & Waldmant, 1993; Gualtieri & Johnson, 2005). Despite the availability of a range of computerized tests, most children are diagnosed based on clinician-determined behavioral observations from parents or teachers, which could potentially lead to inaccurate diagnosis. This may be due to the lack of clarity in behavioral descriptions, the accessibility of a recognized medicinal treatment upon diagnosis, and the overarching label of ADHD, which can be used to encompass aggression, irritability, and learning difficulties, not readily fitting into other diagnostic categories (Batstra, Nieweg, & Hadders-Algra, 2014). On the other

hand, laboratory measures, such as the various continuous performance tasks, have been argued to have low ecological validity and suffer from poor sensitivity and specificity in diagnosing ADHD (Berger, Slobodin, & Cassuto, 2017).

INSERT TABLE 1 AROUND HERE

Upon diagnosis, psychostimulant medications are generally the first line of treatment for ADHD (Pliszka, 2007; Wolraich, Brown, & Brown, 2011) acting as a moderator, most often resulting in substantial symptom reduction (Cortese et al., 2018), although parents can be reticent to give their children medication due to fears over stigma, side-effects, or the long-term effects of treatment (Muris et al., 2018). Side effects may include insomnia, lack of appetite, and headache (Newcorn, Stein, & Cooper, 2010), and there are few studies on the effects of prolonged use of ADHD medications (Kociancic, Reed, & Findling, 2004). It has recently been reported that there were no long-term cognitive benefits of medication (e.g., reaction time or verbal working memory) after six years when participants were unmedicated at the time of testing (Schworen et al., 2018). However, greater improvements were observed when behavioral interventions, such as parent management training, were used prior to medication (William E. Pelham et al., 2016).

Although psychiatric guidelines note that treatment by medication alone is often not sufficient and recommend various social skills training and behavioral interventions, there are few guidelines as to what these non-pharmaceutical interventions should comprise (Sadock, Sadock, Kaplan, & Sadock, 2009; Young & Myanthi Amarasinghe, 2010). Parenting books contain suggestions and guidelines for cognitive training and parenting skills (Grohol, 2018), psychiatric manuals focus on the range of neuropsychiatric medications available (American Psychiatric Association, 2013; Sadock et al., 2009), and psychological guidelines emphasize a multi-systematic approach combining psychostimulant medication with parenting, school, and child psychoeducation to promote rule following and self-regulation skills (Carr, 2015).

Currently there are no clear guidelines to differentiate among available interventions and inform clinicians who choose to recommend non-pharmacological treatments.

There are various non-pharmacological treatments that have been used alongside or instead of pharmacological treatments (Sharma, Gerbarg, & Brown, 2015). Dietary interventions have been shown to have some small improvement in ADHD symptomatology (Edmund J.S. Sonuga-Barke et al., 2013), with Omega 3 demonstrating small improvements in the emotional lability and oppositional behavior associated with ADHD (Cooper et al., 2016). Meditation-based interventions, such as mindfulness and yoga, are widely practiced, in order to improve both mental and physical health. However, there is substantial variability in the methodology of the interventions and which ADHD deficits they are design to target, and as a result no definitive conclusions have been made regarding the efficiency of such treatments (Evans et al., 2018). School-based and summer program interventions (including cognitive behavioral therapy [CBT], contingency management, and academic interventions) are considered “first-line” treatment approaches and have been demonstrated to improve academic performance (DuPaul & Eckert, 1997), although interventions, such as parental training (Lee, Niew, Yang, Chen, & Lin, 2012) and music therapy, (Maloy & Peterson, 2015) have demonstrated little long-term effects. There appears to be some support for neuro-feedback interventions in improving cognitive and self-control symptoms associated with ADHD (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009; Hodgson, Hutchinson, & Denson, 2014; Van Doren et al., 2019), as well as for non-invasive brain stimulations (such as tDCS, and TMS) in improving cognition and clinical symptoms of inattention and impulsivity (Westwood, Radua, & Rubia, 2019). Although fewer in number, there have also been a number of studies demonstrating similar improvements following physical exercise (Cerrillo-Urbina et al., 2015; Neudecker, Mewes, Reimers, & Woll, 2019), CBT, and cognitive training programs (Knouse, Teller, & Brooks, 2017).

Several systematic reviews and meta-analyses have discussed the effects of non-pharmacological interventions in ADHD (e.g., Arns et al., 2009; Fabiano et al., 2009). However, interpreting these reports, specifically in relation to the impact on core ADHD symptoms, is complicated by the inclusion of trials using nonrandomized designs, non-ADHD samples, and predominately the lack of objective cognitive measures. Furthermore, estimates of efficacy are often based on assessments made by individuals who are likely to be aware of study allocation, which may inflate effect sizes (Jadad & Enkin, 2007). The current study addresses these limitations and endeavors to lower the variability across the field by limiting the methodology to those studies using objective cognitive measures. While recognizing the importance of other outcomes (e.g., oppositional symptoms) as treatment targets for patients with ADHD, analyses of such measures were not viable in this study because of the variance and subjective nature of methodologies. The present analyses aimed to address two major research questions: (1) which leading non-pharmaceutical intervention for ADHD's cognitive symptomology is most effective? and (2) which cognitive symptoms are most amenable to change? To answer these questions, multiple meta-analyses were performed, using data retrieved from the relevant studies. Regarding the first question, we hypothesized there might not be any major preference for one intervention over the other as all interventions share a common therapeutic effect resulting in cognitive improvement. This effect might be stronger and larger than any effect of a specific intervention. As for the second question, we hypothesized that, as the cognitive symptoms are more complex, involving increasing number of cognitive process, the amenability to change should increase.

Given the range of research on single treatments and cognitive factors, studies were divided into four categories of behavioral and psychological treatments on the five key cognitive functions known to be most negatively affected in individuals with ADHD. As the

amount of intervention papers measuring cognitive functions was limited, we decided to include a wider participant age range (children, adolescents, and adults), taking into account the possible variability of results. The four intervention domains included neurofeedback, using the visualization of brain activity to teach patients to increase attention and impulse control (Arns & Strehl, 2013); physical activity, including a range of aerobic exercise forms (Cerrillo-Urbina et al., 2015); cognitive training, employing adaptive schedules that are hypothesized to strengthen ADHD-deficient neuropsychological processes (e.g., working memory) (Markomichali, Pavlina, Donnelly, Nick and Sonuga-Barke, 2009); and CBT, employing skills incorporated into CBT approaches for ADHD, ranging from organization, planning, and time management skills to cognitive reappraisal strategies and mindfulness meditation skills (Lee et al., 2012). Although behavioral interventions (i.e., behavioral parent training and classroom behavioral interventions) have demonstrated some efficacy in treating ADHD, they were not included in this meta-analysis, as none of these studies included cognitive tasks as outcome measures. Furthermore, analyses covered five measures of cognitive domains: mental flexibility, the ability to switch between and hold multiple concepts (Scott, 1962); inhibition, the ability to tune out and avoid stimuli or actions that are irrelevant to the task/process at hand and override a strong internal predisposition (Macleod, 2007); attention, the behavioral and cognitive process of selectively concentrating on a discrete aspect of information (Anderson, 2005); and working memory, the function responsible for temporarily holding information available for processing (Miyake & Shah, 1999). We also created a fifth category of higher executive functions (HE) to incorporate the most complex cognitive functions of planning and reasoning (Diamond, 2013).

2. Materials and methods

2.1. Literature search and selection criteria

This study design is appropriate for summarizing and synthesizing research evidence to inform policy and practice by integrating results from several independent primary studies that are combinable (Cook, Mulrow, & Haynes, 1997). The development of this study protocol, the conduct and design, and the reporting of results was in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-analyses Protocol (PRISMA-P), (Moher, Liberati, Tetzlaff, & Altman, 2009; Shamseer et al., 2015) guidelines.

This study is comprised of a number of stages: (1) literature identification, (2) literature screening, (3) literature selection based on the specified eligibility criteria, yielding (4) a set of studies included in the present meta-analysis. The initial stage encompassed a wide literature search for papers published from January 1980 until December 2017. We used the PsycNET search engines to search for the terms *intervention AND adhd* within the abstract of relevant literature. To control for publication bias, we searched Google Scholar for additional articles that may have been missed on the initial search. We also included the additional *cognitive* and *executive functions* search terms. Literature was also sourced from previously published literature reviews including the aforementioned 2014 (Hodgson et al., 2014), 2016 (Cortese et al., 2016; Tan, Pooley, & Speelman, 2016), 2015 (Battagliese et al., 2015; Cerrillo-Urbina et al., 2015), 2009 (Fabiano et al., 2009) literature reviews. All searches were conducted in English with foreign language papers discarded. Following initial extraction, it was clear that interventions could be divided into four key categories: Neurofeedback, Physical exercise, CBT, and Cognitive training. Other interventions did not contain sufficient number of studies that conformed to our strict inclusion criteria (namely neuropsychological pre- and post-intervention testing) or were similar enough to be encompassed in a pre-existing category (i.e., biofeedback included within the category of neurofeedback).

2.2. Inclusion criteria

1. The study intervention must be tested on participants fulfilling Diagnostic and Statistical Manual of Mental Disorders (DSM) or International Statistical Classification of Diseases and Related Health Problems (ICD) criterion for ADHD. All editions of the manuals for diagnosis were included (American Psychiatric Association, 2013a; World Health Organisation, 2012).
2. The study must examine the effect of one of the four aforementioned interventions on cognitive functions (i.e., measured by objective neuropsychological measures).
3. Effect of the intervention must be evaluated with a validated cognitive or behavioral measurement (i.e., computerized or pen and paper).
4. The study analysis must include data or statistical information which could be used to generate an effect size (d-value). In cases where this was not available the authors were contacted by email to attempt to receive the relevant values.
5. The study must contain a control group or comparison group including ADHD participants. To this end, we included both randomized and non-randomized control trials, as the number of papers with randomized control trials were limited, given potential ethical concerns in randomly denying pharmacological treatment.
6. The study must contain pre- and post-intervention measures.

Our strict inclusion criteria addressed several issues. First, we only included studies that enabled us to generate an appropriate calculation of the experimental design of interest: intervention studies with pre-post-control group design for control pre-test differences. Second, we only included studies with a clinical diagnosis of ADHD, to ensure as clinically homogenous population as possible. Last, we included only studies with cognitive measures, gathered by validated neurocognitive tests, in order to assess the efficacy of interventions using objective measures.

2.3. Exclusion criteria

We did not include studies evaluating the effect of intervention on physiological or neurological functions which were unrelated to neuropsychological functioning (such as depression, fitness or vision). Nor did we include studies evaluating subjective or observer reports.

From the initial search terms, prior to filtering for inclusion criteria, 808 studies in English were retrieved. Duplicates and studies in foreign languages were removed (157), before narrowing down to the four categories of interventions studied (367 removed). The broadness of the search terms allowed us to ensure that all relevant studies were identified, even if they did not necessarily include *cognitive assessment* as a keyword or within the abstract (250 removed). Thirty-four publications which reviewed the neuropsychological effects of interventions were identified and carefully reviewed. The majority of the eliminated studies did not have an ADHD control group or did not use neuropsychological measures, rather relying on self or observer reports of behavior and symptomology. Other reasons for exclusion included inability to access relevant data or calculated effect sizes (such as Drechsler et al., 2007; van der Donk, Hiemstra-Beernink, Tjeenk-Kalff, van der Leij, & Lindauer, 2015; Verret, Guay, Berthiaume, Gardiner, & Béliveau, 2012). Eighteen studies were deemed to fulfil all inclusion and exclusion criteria (from unique publications). One study (Virta et al., 2010) had two intervention groups and an additional control group, thus the data from the control group was included twice (with just half the stated sample size). From each study, we calculated and included all significant and non-significant results. Ten studies were included from analysis of previous meta-analyses and the rest were from further searches. Asterixis in the reference section are used to identify studies included in the meta-analyses and Figure 1 provides an overview of the search process.

INSERT FIGURE 1 AROUND HERE

2.4 Quality assessment

Two reviewers (BL and AH) assessed the quality of data in included studies. We used the National Institute of Health (NIH) quality assessment tools (NIH, 2014). The NIH tool was preferred because it is more comprehensive and thus enables an exhaustive assessment of quality of included studies. Additional methodological appraisal of each eligible RCT study followed the Cochrane Handbook for Systematic Reviews of Interventions Version 5.1.0.

2.5. Data coding

Statistical data including means, standard deviations, and number of subjects were extracted from each study for pre- and post- intervention results in both groups. The results of each neuropsychological test were categorized according to which neuropsychological function they tested. Tests that were commonly used and had frequent literature references, we categorized according to the recognized cognitive function. In tests not commonly used, we relied on the authors' categorization. The categories of cognitive function studied included Attention, Flexibility, Inhibition, Working Memory, and the final category of Higher Executive Function, which included planning and reasoning.

Only studies including group means and standard deviations of neuropsychological scores were included. In cases where there were several reported scores for various measures for one cognitive function in an individual study, we extracted all data but chose to use the most commonly cited test for the most relevant measure (based on the authors' intensive review of the literature). Thus, for each published study it was possible to have 5 effect sizes. Finally, if there were multiple effect sizes for a specific function and they were derived from equally relevant tests, we used the highest effect size reflecting the most robust effect. The following variables were included in a coding sheet: study identification number, first author, year of publication, number of total participants, number of clinical and non-clinical

participants, name of intervention one, name of intervention two or control, number of participants in each group, whether the participants took medication during the trial and testing, the mean age and age range of participants, language of testing, and which cognitive tests were used for each cognitive category. We initially created a coding sheet with all tests and results, even if there were multiple test results for a single category, but following the method detailed above we created a final coding sheet including one pre-intervention score (M and SD) and one post-intervention score (M and SD) for each cognitive function tested within a single study. All studies were coded and the data extracted independently by the first two authors. Inter-rater reliability was 100%. All data was extracted from papers, and if there was a lack of sufficient information to calculate effect sizes, the authors were contacted (for standard deviations).

2.6. *Statistical analyses*

Effect size calculations were made using the Psychometrica program (Lenhard, 2017) and then entered into a programmed excel sheet to analyse effect sizes according to Morris calculations (Morris, 2008). This approach allows inclusion of intervention studies containing groups with uneven sample sizes and varying pre-test means and standard deviations, thus enabling to weight the estimation of the population effect size. Effect size calculation was based on the recommended formula: mean pre- to post-treatment change minus the mean pre- to post-treatment control group change divided by the pooled pretest standard deviation with a bias adjustment (Morris, 2008). Studies with insufficient and missing data were excluded from the meta-analysis. Given the heterogeneity of ADHD assessments, sample characteristics, and implementation of treatments within domains in the included studies, we chose a priori to use random-effects models (Field & Gillett, 2010).

Effect sizes were also calculated according to Rosenthal (1991). In order to assess homogeneity/heterogeneity, we examined the data using Q test (Sanchez-Meca & Fulgencio, 1997; Shadish & Haddock, 1994) and I^2 (Higgins & Thompson, 2002). Accordingly, if the Q value is not significant, then the effect sizes were considered homogeneous, and the mean effect size was considered the best estimation for the data. However, if the Q is significant, moderators should be suggested, as the effect sizes were considered to be heterogeneous.

3. Results

Our final meta-analysis included 18 studies and 19 key interventions, which yielded 49 effect sizes for the five categories of cognitive functions. Initially the cognitive function most susceptible to the intervention was entered into a meta-analysis comprising all the published studies. The results were not deemed homogenous, generating a Q value of 28.57, ($p > 0.05$) and $I^2 = 61.5$, which reflects the presence of a moderator or several moderators. A potential moderator was deemed to be medication. While the majority of studies included both medicated and unmedicated participants, 4 studies solely included unmedicated participants. When studies were separated into two meta-analyses reviewing medicated and un-medicated participants, a homogenous effect size was generated for both. As can be seen in Table 2, un-medicated participants improved significantly in cognitive functions, with an effect size of 0.67 generated ($Q=0.05$, $p<0.05$, $I^2=0$, 95% CIL= -3.76, 4.84). Studies with medicated participants (see Table 3) also generated a homogeneous effect size of 0.68 ($Q=0.05$, $p<0.05$, $I^2=0$, 95% CIL= -3.54, 5.05), indicating only a slightly higher effect of non-pharmaceutical treatments when combined with medication. The homogenous findings indicated that there was no need to search for further moderators. A funnel plot of included studies did not show any asymmetry, an indication that significant publication bias was unlikely (see Figure 2).

INSERT FIGURE 2 AROUND HERE

Following the understanding that non-pharmaceutical treatments were moderately to largely effective both with and without the accompaniment of medication, the second stage of the current study was to determine which intervention was most successful and whether each intervention type could be categorized as having a homogenic, significant effect on cognitive functions. All intervention categories demonstrated homogeneous and significant results (see Figure 3). Physical exercise interventions demonstrated the highest effect size of 0.93, (df= 4, 95% CIL=0.48, 1.45; see Table 4), and studies were deemed to be homogenous, (Q=8.16, $p < 0.05$, $I^2 = 50.98$). Cognitive training interventions demonstrated the lowest effect size of 0.45 (df= 1, 95% CIL= -0.08, 0.89; see Table 5) and were deemed homogenous (Q=0.05, $p < 0.05$, $I^2 = 0$), although given that only two studies were included, this finding should be interpreted with caution. CBT interventions demonstrated a moderate effect size of 0.70 (df=2, 95% CIL=0.24, 1.21; see Table 6), and studies were deemed to be homogeneous (Q=1.087, $p < 0.05$, $I^2 = 0$). Neurofeedback likewise demonstrated a moderate effect size of 0.61 (df= 5, 95% CIL=-3.77, 4.82; see Table 7), and studies were deemed to be homogenous (Q=2.377, $p < 0.05$, $I^2 = 0$).

INSERT FIGURE 3 AROUND HERE

Following comparison of the efficacy across interventions, the third and final stage was to investigate which cognitive function could be classified as most affected by interventions, regardless of intervention type, and whether each cognitive function could be classified as having a homogeneous effect of improvement (see Figure 4). Meta-analyses run on cognitive function generated uniquely homogeneous results, other than the category of higher executive function, in which five scores were analyzed with a resulting Q value of 14.63 ($p > 0.05$) and $I^2 = 72.65$.

INSERT FIGURE 4 AROUND HERE

Inhibition demonstrated the greatest effect size, 0.69, (df=13, 95% CIL= 0.16, 1.13; see Table 8), which was homogeneous across interventions ($Q=17.35$, $p<0.05$, $I^2=25.07$). Working memory demonstrated the lowest effect size, 0.4 (df= 9, 95% CIL=0.45, 0.55; see Table 9), although it was still homogenous ($Q=9.951$, $p<0.05$, $I^2=10.56$). The 14 effect sizes garnered from studies measuring attention revealed an average effect size of 0.41 (df=13 95% CIL= -0.08, 0.84; see Table 10) and homogeneity ($Q=8.81$, $p<0.05$, $I^2=46.4$), indicating a small to medium effect size. A similar effect size was also demonstrated on measures of on flexibility 0.6, (df=6, 95% CIL= -3.64, 4.96; see Table 11), which demonstrated homogeneity ($Q=1.21$, $p<0.05$, $I^2=0$).

INSERT TABLES 2-11 AROUND HERE

4. Discussion

This is the first meta-analysis which collated the most prominent behavioral and cognitive interventions for ADHD and examined their effect on cognitive symptomatology. Whereas other methodically sound meta-analyses generally focused on the subjective patient, parent, or teacher-observed symptomatology, we sought to investigate the effect of non-pharmacological treatments on objective neuropsychological outcomes. We focused entirely on cognitive symptomatology using a range of participant populations (children, adolescents, and adults) and included both those who were taking and not taking medications. We were particularly interested in whether the separate functions were differentially affected by interventions and which cognitive function was most amenable to change.

Notably, there was only slight increased improvement when medication was combined with a non-pharmacological intervention. Studies that included only non-medicated participants still produced a homogeneous moderate to large effect size. This finding highlights the added benefit of a combined medication and non-pharmacological intervention

regime but also suggests the possibility of demonstrating improvement in cognitive function with a non-pharmacological intervention alone.

Of the cognitive and behavioral interventions studied, physical exercise, regardless of type, was found to be the most effective in targeting and reducing cognitive symptoms of ADHD. This was followed by CBT, neuro and biofeedback, and lastly cognitive training. All interventions showed moderate to large effect sizes, indicating their success in reducing cognitive symptoms, as compared to control or less effective interventions.

When all the interventions were considered, inhibition and flexibility were the cognitive functions most affected. They demonstrated the ability to undergo a significant change following the interventions and thus could be considered the most malleable (Stuss & Knight, 2009). Attention and working memory were both found to have moderate effect sizes. These findings accord with theories of executive function. Specifically, the functions with the highest effect sizes were those considered higher cognitive functions, or executive functions, as opposed to basic attention and working memory functions which are less complex and could be categorized as lower cognitive functions (Diamond, 2013; Luria, 1973).

Whereas various brain areas demonstrate plasticity when exercised or after prolonged lack of usage, the pre-frontal cortex, where executive functions have been predominantly localized, appears to develop in animals simply when exposed to complex environments (Stuss & Knight, 2009). Given that the pre-frontal cortex is the last region in the brain to mature, it has the greatest potential for malleability (Hsu, Novick, & Jaeggi, 2014). Thus, although executive functions and the prefrontal cortex are highly susceptible to physiological, psychological, or social stress, these regions are also most susceptible to change (Diamond, 2013). It appears that this susceptibility to change makes higher executive functions most amenable to training and most generalizable to additional executive functions. Compared

with working memory training, inhibitory control training has been demonstrated to have had a greater and more generalizable effect than higher reasoning (Maraver, Bajo, & Gomez-Ariza, 2016). It is thought that whereas working memory requires ‘holding’ information and forms a basis for higher executive functions, inhibition requires ‘working’ with the information, thus capitalizing on active learning processes and enhancing malleability (Diamond, 2013). Inhibition’s increasingly demonstrated malleability and susceptibility to training has led researchers to consider whether it in fact undergoes an automatization (Spierer, Chavan, & Manuel, 2013). According to this theory, training inhibitory control is much like training to ride a bicycle, practicing over and over again perfects balance, coordination, and movement, eventually becoming an automated process.

Despite demonstrating the malleability of inhibitory control, working memory training is far more widely accessible and studied (Goldstein & Naglieri, 2014; Séguin, Nagin, Assaad, & Tremblay, 2004; Shinaver, Entwistle, & Söderqvist, 2014). When higher executive functions, such as inhibition, are targeted in a cognitive training program, they are targeted as part of a more general executive function training program (Diamond, 2012). Although training inhibition and flexibility in a specialized cognitive training program may not be the most effective improvement method, as our research suggests, given the susceptibility to change and the known benefit of usage to promote development (Diamond, 2006, 2012; Diamond & Lee, 2011; Goldstein & Naglieri, 2014), interventions that train and target inhibitory control and flexibility appear to be useful.

The present analyses have found physical exercise to be the most effective intervention for improving the examined cognitive functions. Although physical exercise, such as team or racket sports, appears most effective when it directly challenges executive functions, it may also improve cognition, albeit to a lower degree, when it simply provides aerobic stimulation. Acute bouts of intense aerobic exercise have been shown to improve

executive functions due to neurotransmitter modifications which relate to central executive tasks (McMorris, Tomporowski, & Audiffren, 2009). Changes in blood flow to the prefrontal cortex and prioritized resource allocation, which favors executive functions, have been found to occur during physical exercise (Hillman, Snook, & Jerome, 2003). Thus, in ADHD population, where the impairment is argued to be largely executive (Willcutt, 2015), physical exercise may be especially beneficial for improving executive functions. Improvements in these cognitive functioning might decrease ADHD-related impairments of attentional and strategic flexibility, inhibition, planning, and working memory.

The current findings also have implications for the diagnostic criteria of ADHD. Detecting substantial improvements in executive functions is consistent with the view that impairments in at least some subsamples of individuals with ADHD are predominantly executive in nature (Willcutt, 2015) and thus most effectively targeted using interventions geared toward executive deficits. Accordingly, it is possible that neurocognitive tests assessing executive functioning could be used to support and refine the diagnostic process. This could potentially aid the low ecological validity and sensitivity of continuous performance tasks (Berger, Slobodin, & Cassuto, 2017), while also adding valid objective measures to diagnosis, thereby reducing excessive reliance on subjective, commonly used, clinician-determined behavioral observations from parents, teachers, and physicians (Batstra, Nieweg, & Hadders-Algra, 2014).

A number of limitations in the present study need to be acknowledged. Given the small number of studies that conformed to our strict inclusion criteria, we did not separate studies according to age or gender of the sample studied. These potential moderators should be evaluated in future studies to examine whether there is a critical age cut off or gender preference for cognitive change among ADHD patients. Another limitation of the study was the dearth of ADHD studies that clearly indicated whether the participants were taking any

medications. The majority of studies included mixed groups of participants who were taking stimulant medication during the intervention and testing period.

It should also be noted that there were many quality intervention studies that were not included in the present analyses due to their lack of computerized or written neuropsychological tests. Although inclusion of all intervention studies for ADHD would have provided a much richer database for analyses, it would have been difficult to isolate the effects of these interventions on cognitive functions, which was the primary focus of this study. It is also important to note that this study did not examine whether alternative, non-pharmacological, interventions were more or less effective than medication, but rather focused on their use as a complementary or alternative treatment. As previously indicated, there were few studies that separated and defined medicated and unmedicated participants. Therefore, further research would be required comparing randomized clinical trials while differentiating between medicated and non-medicated participants. It is also important to acknowledge that the significant effects found in this study are limited to laboratory tasks. Therefore, future research should evaluate clinical assessments and interventions conducted outside the lab.

5. Conclusion

The present study has demonstrated that behavioral interventions can be successful in improving cognitive symptomatology of ADHD. These findings highlight the importance for diagnosing physicians in the field of ADHD to become familiar with the range of alternative treatments available for the neuropsychological symptoms of the disorder. It would be important to include neuropsychological testing in the diagnostic process in order to identify key cognitive deficits, and, in line with the personalized medicine approach, the alternative treatment should be chosen based on the intervention shown to improve the specific deficits.

According to this study, although all behavioral interventions designed to aid ADHD seem to have a positive effect on cognitive symptomology, physical exercise, especially aerobic exercise that included targeting executive functions, appears to be the most effective. Based on these findings, it could prove fruitful to integrate complex sports, such as ball sports, martial arts, and physical exercise that involve flexibility and inhibition of impulsive behaviors into ADHD treatment regimen.

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Table 1: Sample of some commonly used ADHD diagnostic tools

Test Name	Type of test	Symptoms measured/ subscales	Age Range	Advantages	Disadvantages
Conners' Rating Scale (Conners et al., 1999)	Parent, Teacher and Self report questionnaires	hyperactivity/ impulsivity & emotional lability	6-18 (parent/ teacher) 8-18 (self-report)	Quick to fill in. Well accepted validity and reliability	Subjective report
Conners' Adult ADHD Rating Scale–Self- Report: Long Version (CAARS; Conners et al., 1999)	Self-report	DSM-IV ADHD symptoms	18+	Quick to fill in. Well accepted validity and reliability.	Subjective report
Behavior Rating Inventory of Executive Function (BRIEF; Gioia, Isquith, Guy, & Kenworthy, 2000; Gioia et al., 2003)	Parent, Teacher and Self report questionnaires	Executive functions including Inhibit, Shift, Emotional Control, Self- Monitor, Initiate, Working Memory, Plan/Organize, Task Monitor, & Organization of Materials	5-18 18+	Includes Inconsistency, Infrequency, and Negativity validity scales. Measures all Executive Functions.	Takes a long time to fill in (10-15 mins)
Conners' Continuous	Computerized	Overall index of	8+	Widely used computerized test for	Requires phonological

Performance Test (Conners et al., 2000)	continuous performance task	attention problems and inattentiveness, impulsivity, sustained attention, and vigilance		ADHD. Overall good reliability and sensitivity to ADHD. Large normative sample	skills. Has been challenged with a lack of ecological validity (McGee, Clark, Psychology, & 2000, n.d.). Requires laboratory testing. Relatively expensive.
MOXO-CPT (Berger & Goldzweig, 2010)	Computerized continuous performance task	Attention, impulsivity, hyperactivity & timeliness	6+	Incorporates auditory and visual distractors. Sensitive to motor versus impulsivity difficulties (Berger et al., 2017) Does not require literacy ability.	Requires laboratory testing. Relatively expensive.
Test of Variables of Attention, TOVA (Greenberg & Waldmant, 1993)	Computerized continuous performance task	Response time variability (consistency), response time (speed), commissions (impulsivity), omissions (focus and vigilance) and total ADHD score	4+	Does not require literacy ability. Validated from a young age. Commonly used computerized measure of attention in ADHD (Manor et al., 2008). Use of micro-switch to generate accurate reaction times.	Requires laboratory testing. Relatively expensive.

Trail Making Test, Part A (Reitan & Wolfson, n.d.)	Paper pencil test of attention	Time taken part A, B, B-A	9-89	Most frequently used measure of attention (Rabin, Barr, & Burton, 2005). Cheap and easy to use with no added equipment.	No clinical cut-off points. Not as sensitive as computerized testing (Tombaugh, 2004). Relies highly on tester ratings
Wisconsin Card Sorting Test (WCST) (Heaton, Chelune, Talley, Kay, & Curtis, 1993)	Card or computer based test of executive function	Planning, flexibility and impulsivity scores	6-89	Easy to use and available in various modalities. Wide range of normative data. Applicable to real world difficulties.	Not specific to ADHD rather general executive functions

Table 2: Effect sizes and methodology of studies with non-medicated participants

Author	Age range or mean (number participants)	Duration of Intervention	Intervention 1	Intervention 2/ Control	Meds	Measures with Sig. Improvement in Clinical symptomatology	Cognitive test	Cognitive function	Effect size
(Choi, Han, Kang, Jung, & Renshaw, 2015)	13-18 (25)	90 min. sessions, 3 times a week for 6 weeks	Physical Exercise-aerobic exercise	Control	no	ADHD sym.	WCST preservative error	Flexibility	0.87
(Miranda, Presentación, Siegenthaler, & Jara, 2013)	7-10 (42)	45 min sessions. 16 sessions, over course of 10 weeks	CBT	Control	no	Parent rating: ADHD sym. Teacher rating: ADHD sym.	Sentences and digit span combined	Inhibition	0.99
(Klingberg et al., 2005)	7-12 (53)	40 minutes per day for 25 days (avg.)	Cognitive Training	Control	no	Parent rating: Conners and ADHD symptoms	Stroop time	Inhibition	0.38
(Geladé et al., 2017)	7-13 (76)	45 min. 3 times a week, for 10-12 weeks	Neurofeedback	Physical Activity	no	None	Stop signal commission	Inhibition	0.28

(Prins, Dovis, Ponsioen, ten Brink, & van der Oord, 2011)	9.47 (51)	35-50 min. sessions one a week for 4 weeks	Cognitive Training game	Cognitive training normal	no	n/a	Corsi Visuospatial WM	Working memory	0.82
							Mean effect size		0.67

Table 3: Effect sizes and methodology of studies with medicated participants

Author	Age range or mean (number participants)	Duration of Intervention	Intervention 1	Intervention 2/ Control	Meds	Measures with Sig. Improvement of Clinical symptomatology	Cognitive test	Cognitive function	Effect size
(Bakhshayesh, Hänsch, Wyschkon, Rezai, & Esser, 2011)	4-16 (35)	30 min. sessions, 2-3 times a week for 10-15 weeks (30 sessions total)	Neurofeedback	Biofeedback	both	Parents rating: ADHD sym. Teacher rating: ADHD sym.	CPT Commission errors	Inhibition	0.65
(Beauregard & Lévesque, 2006)	8-12 (20)	60 min. sessions, 3 times a week for 13 weeks (40 sessions total)	Neurofeedback	Control	both	n/a	Stroop (natural trials mean score)	Inhibition	0.93
(Drechsler et al., 2007)	9-13 (30)	90 min. sessions, once-twice a week for 12-15 weeks (total 15 sessions)	Neurofeedback	CBT	both	Parent rating: ADHD sym., BREIF. Teacher rating: BRIEF	Go No Go commission	Inhibition	0.65
(Heinrich, Gevensleben, Freisleder, Moll, & Rothenberger,	7-13 (22)	50 min. sessions for 3 weeks (total 25 sessions)	Neurofeedback	Control	both	Parent rating: ADHD sym.	CPT impulsive errors	Inhibition	0.74

2004)									
(Steiner, Sheldrick, Gotthelf, & Perrin, 2011)	12.4 (20)	45 min. training, twice a week for 4 months	Neurofeedback	Cognitive Training	both	Parent rating: Conners, BASC, Student rating: Conners	CPT response control	Inhibition	0.39
(Yu-Kai Chang, Hung, Huang, Hatfield, & Hung, 2014)	5-10 (27)	90 min session, twice a week for 8 weeks	Physical Exercise-aquatic exercise	Control	both	n/a	Go No Go accuracy	Inhibition	0.72
(Y.-K. Chang, Liu, Yu, & Lee, 2012)	8-15 (40)	30 min. session	Physical Exercise-aerobic exercise	Control	n/a	n/a	WCST non preservative error	Flexibility	0.73
(Gapin, Labban, Bohall, Wooten, & Chang, 2015)	18-25 (20)	40 min. session	Physical Exercise-aerobic exercise	Physical Exercise	yes	n/a	Digit span mean	Inhibition	0.42
(Pan et al., 2016)	6-12 (32)	70 min. training, twice a week for 12 weeks	Physical Exercise-	Control	both	n/a	Stroop word-color	Inhibition	1.92

			racket sport						
(Miranda M. J.; Soriano, M., 2002)	8-9 (50)	2 sessions a month (each 3 hour long) for 4 months	CBT	Control	n/a	Parent rating: ADHD sym., EPC Teacher rating: ADHD sym., Conners, School Problem Inventory, Self-Control	Stroop interference	Inhibition	0.57
(Virta et al., 2010)	21-50 (15)	60 min, once a week, for 10 weeks	CBT	Control	both	Self ratings: BADDS		Flexibility	0.51
(Menezes, Dias, Trevisan, Carreiro, & Seabra, 2015)	8-13 (18)	60 min. meetings, twice a week for 8 months	Cognitive Training	Control	both	None	Stroop interference	Inhibition	0.5
(Salomone et al., 2015)	18-50 (37)	2 training sessions and home practice for 5 weeks	Cognitive Training	Control	both	Self rating: Conners, BDI, GSES	Hotel task (attempted tasks)	Attention	0.1
							Mean effect size		0.68

Journal Pre-proof

Table 4: Effect sizes and methodology of physical exercise studies

Author	Age range or mean (number participants)	Duration of Intervention	Intervention 1	Intervention 2/ Control	Meds	Cognitive test	Cognitive function	Effect size
(Yu-Kai Chang et al., 2014)	5-10 (27)	90 min session, twice a week for 8 weeks	Physical Exercise-aquatic exercise	Control	both	Go No Go accuracy	Inhibition	0.72
(Y.-K. Chang et al., 2012)	8-15 (40)	30 min. session	Physical Exercise-aerobic exercise	Control	n/a	WCST non-preservative error	Flexibility	0.73
(Choi et al., 2015)	13-18 (25)	90 min. sessions, 3 times a week for 6 weeks	Physical Exercise-aerobic exercise	Control	no	WCST preservative error	Flexibility	0.87
(Gapin et al., 2015)	18-25 (20)	40 min. session	Physical Exercise-aerobic exercise	Physical Exercise	yes	Digit span mean	Inhibition	0.42
(Pan et al., 2016)	6-12 (32)	70 min. training, twice a week for 12 weeks	Physical Exercise-racket sport	Control	both	Stroop word-color	Inhibition	1.92
						Mean effect size		0.93

Journal Pre-proof

Table 5: Effect sizes and methodology of Cognitive training studies

Author	Age range or mean (number participants)	Duration of Intervention	Intervention 1	Intervention 2/ Control	Meds	Cognitive test	Cognitive function	Effect size
(Klingberg et al., 2005)	7-12 (53)	40 minutes per day for 25 days (avg.)	Cognitive Training	Control	no	Stroop time	Inhibition	0.38
(Menezes et al., 2015)	8-13 (18)	60 min. meetings, twice a week for 8 months	Cognitive Training	Control	both	Stroop interference	Inhibition	0.5
(Salomone et al., 2015)	18-50 (37)	2 training sessions and home practice for 5 weeks	Cognitive Training	Control	both	Hotel task (attempted tasks)	Attention	0.1
(Prins et al., 2011)	9.47 (51)	35-50 min. sessions one a week for 4 weeks	Cognitive Training game	Cognitive training normal	no	Corsi Visuospatial WM	Working memory	0.82
						Mean effect size		0.45

Table 6: Effect sizes and methodology of Cognitive behavioral therapy studies

Author	Age range or mean (number participants)	Duration of Intervention	Intervention 1	Intervention 2/ Control	Meds	Cognitive test	Cognitive function	Effect size
(Miranda M. J.; Soriano, M., 2002)	8-9 (50)	2 sessions a month (each 3 hour long) for 4 months	CBT	Control	n/a	Stroop interference	Inhibition	0.57
(Virta et al., 2010)	21-50 (15)	60 min, once a week, for 10 weeks	CBT	Control	both	CNVS	Flexibility	0.51
(Miranda et al., 2013)	7-10 (42)	45 min sessions. 16 sessions, over course of 10 weeks	CBT	Control	no	Sentences and digit span combined	Inhibition	0.99
						Mean effect size		0.69

Table 7: Effect sizes and methodology of neuro-feedback studies

Author	Age range or mean (number participants)	Duration of Intervention	Intervention 1	Intervention 2/ Control	Meds	Cognitive test	Cognitive function	Effect size
(Bakhshayesh et al., 2011)	4-16 (35)	30 min. sessions, 2-3 times a week for 10-15 weeks (30 sessions in total)	Neurofeedback	Biofeedback	both	CPT Commission errors	Inhibition	0.65
(Beauregard & Lévesque, 2006)	8-12 (20)	60 min. sessions, 3 times a week for 13 weeks (40 sessions total)	Neurofeedback	Control	both	Stroop (natural trials mean score)	Inhibition	0.93
(Drechsler et al., 2007)	9-13 (30)	90 min. sessions, once-twice a week for 12-15 weeks (total 15 sessions)	Neurofeedback	CBT	both	Go No Go commission	Inhibition	0.65
(Heinrich et al., 2004)	7-13 (22)	50 min. sessions for 3 weeks (total 25 sessions)	Neurofeedback	Control	both	CPT impulsive errors	Inhibition	0.74

(Steiner et al., 2011)	12.4 (20)	45 min. training, twice a week for 4 months	Neurofeedback	Cognitive Training	both	CPT response control	Inhibition	0.39
(Geladé et al., 2017)	7-13 (76)	45 min. 3 times a week, for 10-12 weeks	Neurofeedback	Physical Activity-aerobic exercise	no	Stop signal commission	Inhibition	0.28
						Mean effect size		0.61

Table 8: Effect sizes and methodology of inhibition studies

Author	Age range or mean (number participants)	Duration of Intervention	Intervention 1	Intervention 2/ Control	Meds	Cognitive test	Effect size
Bakhshayesh et al., 2011	4-16 (35)	30 min. sessions, 2-3 times a week for 10-15 weeks (30 sessions in total)	Neurofeedback	Biofeedback	both	CPT Commission errors	0.65
Beauregard & Lévesque, 2006	8-12 (20)	60 min. sessions, 3 times a week for 13 weeks (40 sessions total)	Neurofeedback	Control	both	Go No Go accuracy	0.93
(Yu-Kai Chang et al., 2014)	5-10 (27)	90 min session, twice a week for 8 weeks	Physical Exercise-aquatic exercise	Control	both	Go No Go accuracy	0.72
(Y.-K. Chang et al., 2012)	8-15 (40)	30 min. session	Physical Exercise-aerobic exercise	Control	n/a	Stroop inference	0.45
Drechsler et al., 2007	9-13 (30)	90 min. sessions, once-twice a week for	Neurofeedback	CBT	both	Go No Go commission	0.65

		12-15 weeks (total 15 sessions)					
(Gapin et al., 2015)	18-25 (20)	40 min. session	Physical Exercise-aerobic exercise	Physical Exercise	yes	Stroop color-word	0.42
Heinrich et al., 2004)	7-13 (22)	50 min. sessions for 3 weeks (total 25 sessions)	Neurofeedback	Control	both	CPT impulsive errors	0.74
(Klingberg et al., 2005)	7-12 (53)	40 minutes per day for 25 days (avg.)	Cognitive Training	Control	No	Stroop time	0.38
(Menezes et al., 2015)	8-13 (18)	60 min. meetings, twice a week for 8 months	Cognitive Training	Control	Both	Stroop interference	0.5
(Miranda M. J.; Soriano, M., 2002)	8-9 (50)	2 sessions a month (each 3 hour long) for 4 months	CBT	Control	n/a	Stroop interference	0.57
Steiner et al., 2011	12.4 (20)	45 min. training, twice a week for 4 months	Neurofeedback	Cognitive Training	Both	CPT response control	0.39

(Pan et al., 2016)	6-12 (32)	70 min. training, twice a week for 12 weeks	Physical Exercise-racket sport	Control	both	Stroop word-color	1.92
Geladé et al., 2017	7-13 (76)	45 min. 3 times a week, for 10-12 weeks	Neurofeedback	Physical Activity-aerobic exercise	no	Stop signal commission	0.28
(Miranda et al., 2013)	7-10 (42)	45 min sessions. 16 sessions, over course of 10 weeks	CBT	Control	no	Stroop interference	0.99
						Mean effect size	0.69

Table 9: Effect sizes and methodology of working memory studies

Author	Age range or mean (number participants)	Duration of Intervention	Intervention 1	Intervention 2/ Control	Meds	Cognitive test	Effect size
Beauregard & Lévesque,	8-12 (20)	60 min. sessions, 3 times a week for 13	Neurofeedback	Control	both	Digit span mean	0.22

2006		weeks (40 sessions total)					
Gapin et al., 2015	18-25 (20)	40 min. session	Physical Exercise-aerobic exercise	Physical Exercise	Yes	Digit span mean	0.72
Klingberg	7-12 (53)	40 minutes per day for 25 days (avg.)	Cognitive Training	Control	no	Digit span mean	0.61
Menezes et al., 2015	8-13 (18)	60 min. meetings, twice a week for 8 months	Cognitive Training	Control	both	Auditory WM mean	0.13
Miranda M. J.; Soriano, M., 2002	8-9 (50)	2 sessions a month (each 3 hour long) for 4 months	CBT	Control	no	Digit span mean	0.12
Virta et al., 2010(a)	21-50 (15)	60 min, once a week, for 10 weeks	CBT	Control	both	CNVS memory	0.12
Virta et al., 2010 (b)	21-50 (14)	60 min, once a week, for 10 weeks	Cognitive training	Control	both	CNVS memory	0.3
Geladé et al.,	7-13 (76)	45 min. 3 times a	Neurofeedback	Physical Activity-aerobic	no	Visuospatial WM	0.94

2017		week, for 10-12 weeks		exercise		backwards	
Miranda et al., 2013)	7-10 (42)	45 min sessions. 16 sessions, over course of 10 weeks	CBT	Control	no	Sentences and digit span combined	0.05
(Prins et al., 2011)	9.47 (51)	35-50 min. sessions one a week for 4 weeks	Cognitive Training game	Cognitive training normal	no	Corsi Visuospatial WM	0.82
						Mean effect size	0.4

Table 10: Effect sizes and methodology of attention studies

Author	Age range or mean (number participants)	Duration of Intervention	Intervention 1	Intervention 2/ Control	Meds	Cognitive test	Effect size
Bakhshayesh et al., 2011	4-16 (35)	30 min. sessions, 2-3 times a week for 10-15 weeks (30 sessions in total)	Neurofeedback	Biofeedback	both	Paper Pencil Cancellation	1.02
Beauregard & Lévesque, 2006)	8-12 (20)	60 min. sessions, 3 times a week for 13 weeks (40 sessions total)	Neurofeedback	Control	both	Stroop (natural trials mean score)	0.69
Y.-K. Chang et al., 2012	8-15 (40)	30 min. session	Physical Exercise-aerobic exercise	Control	n/a	Stroop (color)	0.1
Drechsler et al., 2007	9-13 (30)	90 min. sessions, once-twice a week for 12-15 weeks (total 15 sessions)	Neurofeedback	CBT	both	Paper Paper cancellation	0
Gapin et al.,	18-25 (20)	40 min. session	Physical Exercise-	Physical Exercise	yes	Stroop (color)	0.56

2015			aerobic exercise				
Heinrich et al., 2004)	7-13 (22)	50 min. sessions for 3 weeks (total 25 sessions)	Neurofeedback	Control	both	CPT (no of hits)	0.19
Menezes et al., 2015	8-13 (18)	60 min. meetings, twice a week for 8 months	Cognitive Training	Control	both	Stroop (naming)	0.49
Miranda M. J.; Soriano, M., 2002	8-9 (50)	2 sessions a month (each 3 hour long) for 4 months	CBT	Control	n/a	Stroop (naming)	0.49
Steiner et al., 2011	12.4 (20)	45 min. training, twice a week for 4 months	Neurofeedback	Cognitive Training	both	CPT (attention)	0.59
Virta et al., 2010 (a)	21-50 (15)	60 min, once a week, for 10 weeks	CBT	Control	both	CNVS (reaction time)	0.43
Virta et al., 2010 (b)	21-50 (14)	60 min, once a week, for 10 weeks	Cognitive Training	Control	both	CNVS (reaction time)	0.09
Geladé et al.,	7-13 (76)	45 min. 3 times a	Neurofeedback	Physical Activity-aerobic	no	Oddball (reaction time)	0.25

2017		week, for 10-12 weeks		exercise			
(Salomone et al., 2015)	18-50 (37)	2 training sessions and home practice for 5 weeks	Cognitive Training	Control	both	Hotel task (attempted tasks)	0.1
Miranda et al., 2013)	7-10 (42)	45 min sessions. 16 sessions, over course of 10 weeks	CBT	Control	no	CPT (omissions & commissions)	0.77
						Mean effect size	0.41

Table 11: Effect sizes and methodology of flexibility studies

Author	Age range or mean (number participants)	Duration of Intervention	Intervention 1	Intervention 2/ Control	Meds	Cognitive test	Effect size
Y.-K. Chang et al., 2012	8-15 (40)	30 min. session	Physical Exercise-aerobic exercise	Control	n/a	WCST non preservative error	0.73
(Choi et al., 2015)	13-18 (25)	90 min. sessions, 3 times a week for 6 weeks	Physical Exercise-aerobic exercise	Control	no	WCST preservative error	0.87
Menezes et al., 2015	8-13 (18)	60 min. meetings, twice a week for 8 months	Cognitive Training	Control	both	WCST Total errors	0.49
Pan et al., 2016	7-12 (60)	70 min. training, twice a week for 12 weeks	Physical Exercise-racket sport	Control	both	WCST Total correct	0.58
Virta et al., 2010 (a)	21-50 (15)	60 min, once a week, for 10 weeks	CBT	Control	both	CNVS-cognitive flexibility	0.51
Virta et al.,	21-50 (14)	60 min, once a	Cognitive training	Control	both	CNVS-cognitive flexibility	0.25

2010 (b)		week, for 10 weeks					
Miranda et al., 2013)	7-10 (42)	45 min sessions. 16 sessions, over course of 10 weeks	CBT	Control	no	WCST Preservative errors	0.78
						Mean effect size	0.6

Figure captions

1. Flow chart displaying the literature search process according to PRISMA guidelines

(Moher et al. 2009).

2. Funnel plot showing effect sizes for non-pharmacological treatments delineated by sample size.

3. A forest plot displaying the effect sizes and confidence intervals of the various interventions on executive functions.

4. A forest plot displaying the effect sizes and confidence intervals of executive functions included in the meta-analyses.

Figure 1

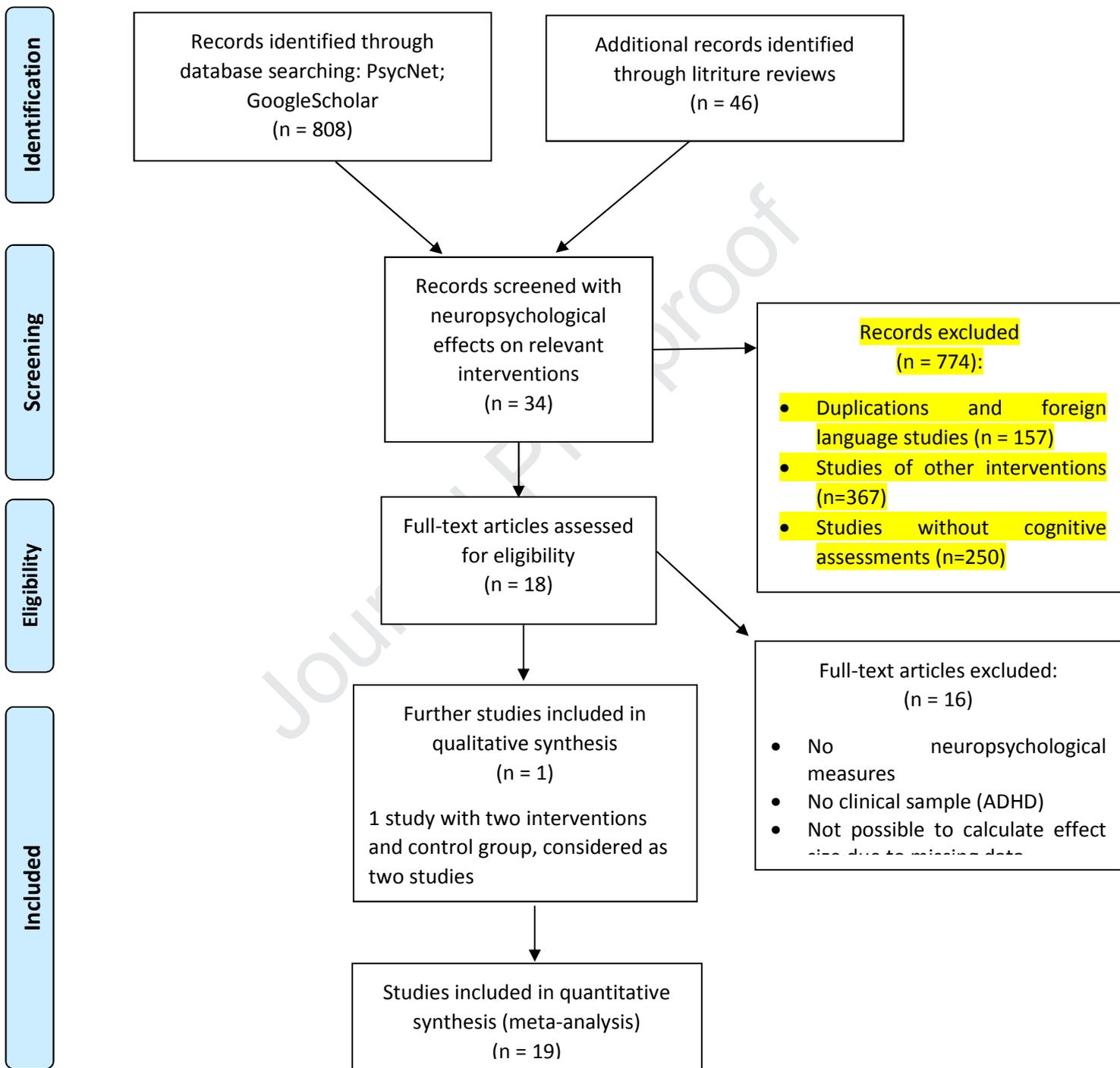


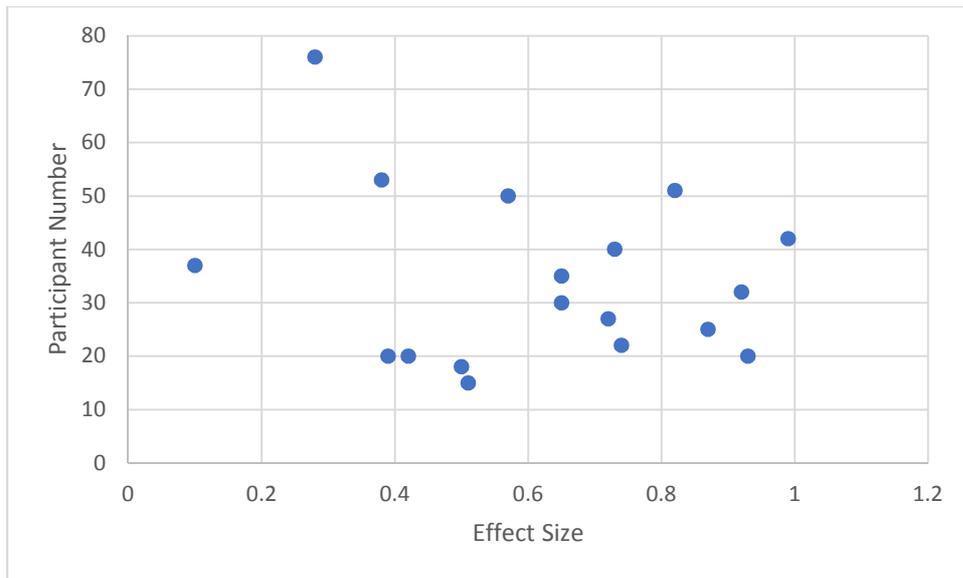
Figure 2

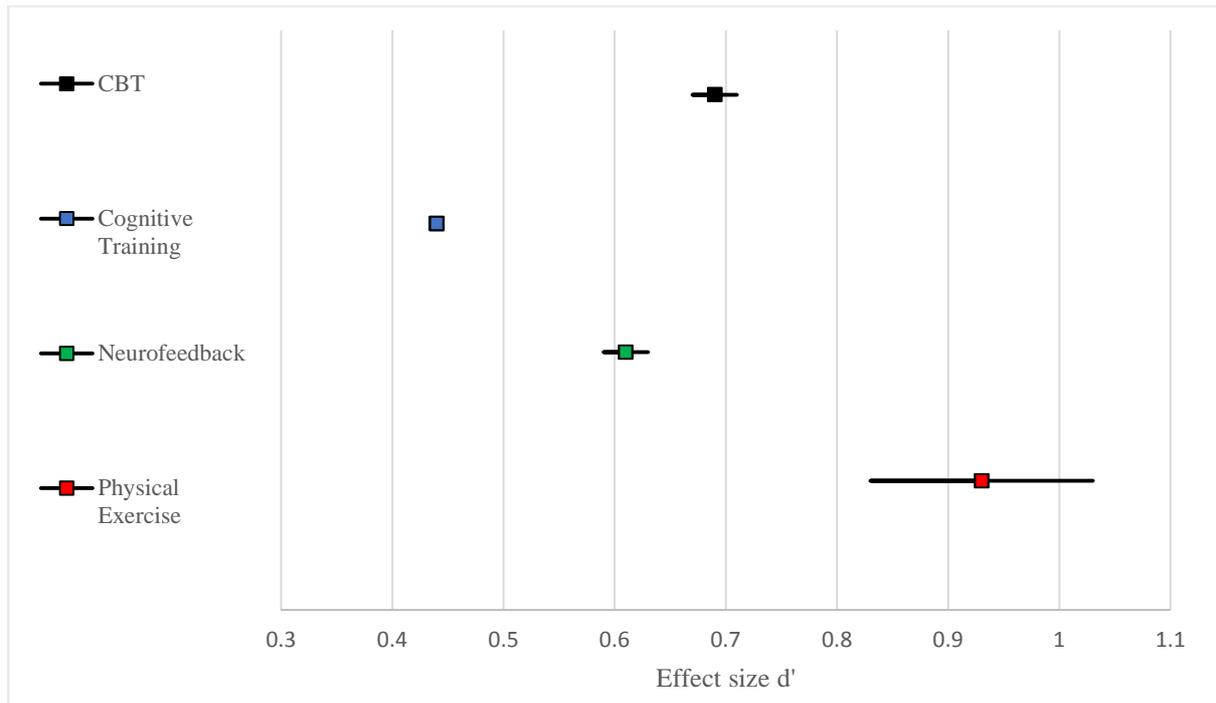
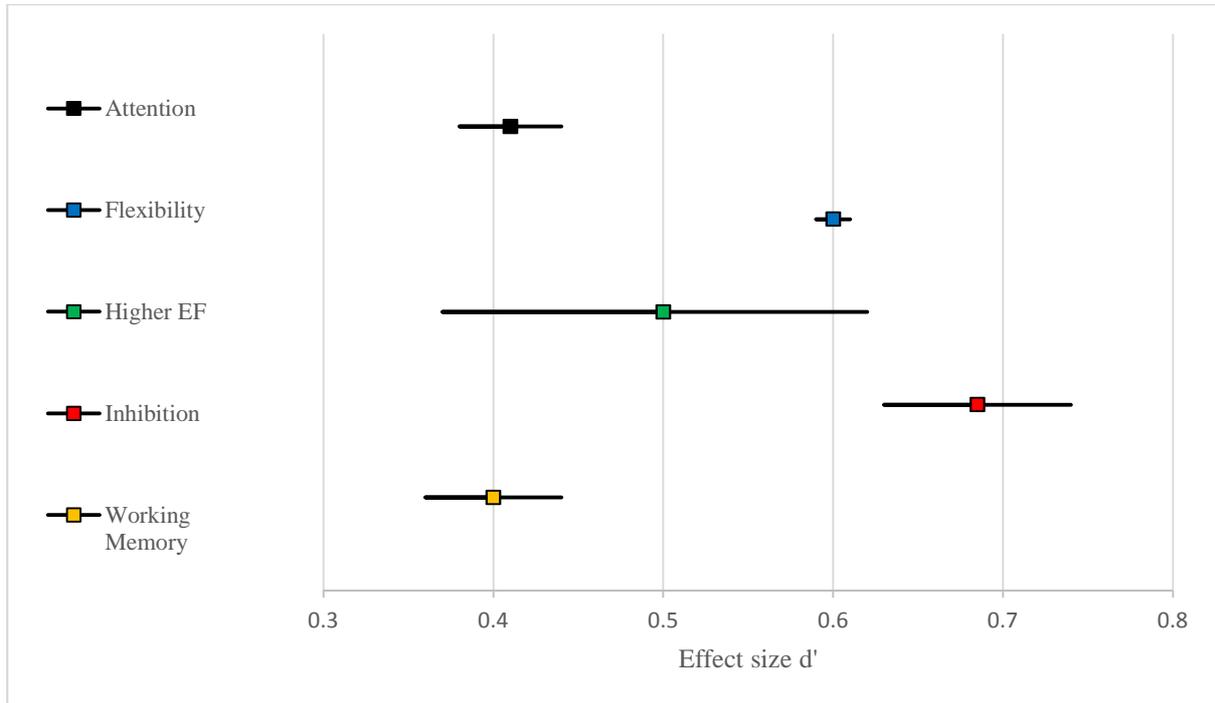
Figure 3

Figure 4



Highlights

- This meta-analysis examined non-pharmacological interventions for ADHD.
- Data were extracted from studies that used objective cognitive measures.
- Physical exercises demonstrated the highest average effect size.
- Findings highlight the positive effect of psychological interventions on ADHD.

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Conflict of interest

All authors declare that they have no conflicts of interest arising from this manuscript.

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