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The Neurocognitive Profile of Attention-Deficit/Hyperactivity Disorder: A Review of Meta-Analyses

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Abstract

Objective: Numerous meta-analyses have summarized studies comparing the neurocognitive performance of individuals with attention-deficit/hyperactivity disorder (ADHD) to that of healthy controls.
Method: The present study is a systematic review and quantitative summary of those meta-analyses that aimed to determine the extent to which individuals with ADHD differ cognitively from typically developing controls.
Results: Of 253 standardized mean differences (SMDs) drawn from 34 meta-analyses, 244 (96%) were positive, indicating better neurocognitive performance in the control group than the ADHD group. The mean effect size was .45 (*SD* = .27). Unweighted means of SMDs for neurocognitive domains ranged from .35 (set shifting) to .54 (working memory). When weighted by the number of studies aggregated, they ranged from .35 (set shifting) to .66 (reaction time variability). Neurocognitive domains with mean effects over .50 included working memory (.54), reaction time variability (.53), response inhibition (.52), intelligence/achievement (.51), and planning/organization (.51). When weighted by number of aggregated studies, the domains with mean effects over .50 were reaction time variability (.66), intelligence/achievement (.60), vigilance (.56), working memory (.54), and response inhibition (.52). Age moderated the relationship between ADHD diagnosis and neurocognitive functioning, with greater between-groups differences among children and adults than among adolescents. Funding also moderated this relationship: meta-analyses that received drug funding found larger effect sizes than those without drug funding.
Conclusions: The evidence suggests that ADHD is associated with substantial deficits across a variety of neurocognitive domains. This is the most in-depth review of the neurocognitive functioning of people with ADHD to date.

Keywords: ADHD; Meta-analysis; Executive functions

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a psychiatric disorder characterized by symptoms of inattention, hyperactivity, and impulsivity that affects an estimated 5% of children and 2.5% of adults across the globe (American Psychiatric Association [APA], 2013). Roughly 15% of individuals diagnosed with ADHD as children continue to meet full criteria at age 25, though 65% are in partial remission (Faraone, Biederman, & Mick, 2006). Attention-deficit/hyperactivity disorder has been associated with academic problems throughout childhood (National Institutes of Health Consensus Development Conference, 2000), persisting into adulthood (Frazier, Youngstrom, Glutting, & Watkins, 2007). There is a high comorbidity rate among children with other externalizing disorders, such as oppositional defiant disorder and conduct disorder (APA, 2013; Barkley, 1997). In adolescence and adulthood, individuals diagnosed with ADHD have been found to have more problems at work, home, and in interpersonal relationships (Barkley, 1997; National Institutes of Health Consensus Development Conference, 2000).

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A number of studies have compared the performance of individuals with ADHD to that of healthy controls on various neurocognitive measures, to determine whether differences in a particular neurocognitive domain would distinguish individuals with ADHD from their typically developing peers. There have been so many such studies that more than 30 meta-analyses have been conducted over the past two decades compiling these results, producing a wide range of effect sizes across neurocognitive domains and meta-analyses. The aim of the present study was to provide a systematic review and quantitative summary of those meta-analyses.

Theoretical Models of Attention-Deficit/Hyperactivity Disorder

Many theoretical models have been proposed to explain the neurocognitive mechanisms underlying ADHD. *Pennington and Ozonoff (1996), among the first to devise a theoretical model for the etiology of ADHD, posited that the attentional problems and impulsive behavior observed in persons with ADHD arose because of deficits in executive functioning. They observed that some of the symptoms of ADHD resembled those of patients with frontal lobe lesions, especially in the prefrontal cortex, and hypothesized that decreased functioning in the prefrontal cortex leads to executive functioning deficits in individuals with ADHD. This hypothesis set the stage for subsequent models.

Perhaps the most widely cited theoretical model of the etiology of ADHD is Barkley's (1997) model of executive deficits. This model posits that the central deficit in ADHD is in response inhibition (frequently called "behavioral inhibition" by Barkley). Barkley proposed that response inhibition is comprised of three interrelated processes: (i) the ability to inhibit an automatic response or a response likely to result in immediate reinforcement (a "pre-potent response"), (ii) the ability to delay a response or pause a response that has already been initiated, and (iii) the ability to remain focused on the response in question and not be distracted by competing stimuli ("interference control"). According to Barkley, impairment in response inhibition directly disrupts four executive processes: working memory, the ability to hold information in conscious awareness and manipulate it; self-regulation, the ability to control one's emotions, motivation, and arousal to achieve goals; internalization of speech, one's internal monologue that allows for more complex reasoning and reflection; and reconstitution, the analysis and synthesis of verbal and behavioral information, allowing for the understanding and production of increasingly more complex language and behaviors. Disruption of these processes in turn leads to problems executing complex actions and goal-directed behaviors, specifically, problems with motor control, fluency, and syntax.

Several theories focus on the differences between the inattentive and combined inattentive-hyperactive/impulsive presentations of ADHD. Diamond (2005), for example, proposed that the inattentive presentation is a disorder of working memory, whereas the combined type stems from problems with response inhibition. Another popular theory has proposed that the inattentive type is associated with disruptions of the "cool" executive function pathway and the hyperactive type with the "hot" executive function pathway (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006). The "cool" pathway regulates tasks that do not require or elicit affective involvement, including abstract tasks such as maintaining attention, an ability that can be evaluated via continuous performance tasks that are commonly used to assess for ADHD. The "hot" pathway, on the other hand, is used to modulate responses to affective tasks, usually involving rewards and motivation (Castellanos et al., 2006; Rubia, 2011). This theory therefore suggests that the inattentive disorder has more to do with problems processing cognitive tasks, the hyperactive/impulsive disorder with emotional stimuli.

Still other models view all three subtypes of ADHD as arising from deficits in both cognitive control and affect regulation (Nigg & Casey, 2005; Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003). These models propose that ADHD symptoms arise from problems with response inhibition and other executive functions, as well as with the abilities to delay gratification and to become aroused and devote energy to a task. Another model posits that the deficits seen in individuals with ADHD are not due to problems with executive functions, but are rather the result of an inability to turn off the default mode network, the pattern of brain activation that occurs at rest, when engaging in a task (Sonuga-Barke & Castellanos, 2007). The majority of these models predict that individuals with ADHD (or that individuals of each ADHD subtype) will have distinct neurocognitive profiles that should be identifiable through neuropsychological assessment.

Meta-Analysis and Meta-Meta-Analysis

More than 30 meta-analyses have quantitatively summarized a large number of studies examining the neurocognitive functioning of individuals with ADHD compared to healthy controls. These meta-analyses have addressed a variety of goals; for example, comparing social cognition among ADHD, autistic, and control groups (Bora & Pantelis, 2016); comparing decision-making in ADHD adults to healthy peers (Mowinckel, Pedersen, Eilertsen, & Biele, 2015); and comparing neurocognition of ADHD and controls by age and gender (*Bálint et al., 2009). They have used a variety of statistical strategies, and

have drawn varying conclusions about the size of the difference in neurocognitive functioning between the two populations. Mean differences have varied greatly across meta-analyses, ranging from −.32 to 1.41.

The availability of a large number of meta-analyses creates an opportunity to make a summative evaluation of the estimated size of the difference between the two populations, at least as captured by the meta-analytic literature. This summative evaluation, sometimes referred to as a meta-meta-analysis or an umbrella review (Cafri, Kromry, & Brannick, 2010; Dragioti, Karathanos, Gerdle, & Evangelou, 2017), can also be used to evaluate other factors as predictors of outcomes in meta-analyses. Examination of moderator effects is only possible for variables that have been reported by a reasonable subset of the sampled meta-analyses. This requirement allowed for evaluation of the following potential moderators:

(1) The number of analyses aggregated in each mean effect size estimate.
(2) The number of participants across the aggregated studies.
(3) Effect size heterogeneity.
(4) Neurocognitive domain. Based on Barkley's (1997) model, we hypothesized that the largest discrepancy would be associated with tasks gauging response inhibition, with slightly smaller between-groups differences in working memory and other executive functions.
(5) Mean age of participants.
(6) Gender distribution of participants.
(7) Publication bias.
(8) Drug funding.
(9) Percent of samples aggregated with small sample sizes.

Other potential moderators were considered, including ADHD subtype and whether participants had been treated with medication, but there was insufficient reporting for these variables.

Materials and Methods

Literature Search

Literature searches were conducted using the PsycINFO and MEDLINE databases from inception until March 2016. The keywords used included *meta-analysis* and variations of *ADHD* paired with *neuropsych**, *cognitive test*, or any term from a comprehensive list of the neurocognitive tests often used in ADHD research (see Appendix A for the full set of search terms). Additional sources were located by searching through references in identified meta-analyses. The search produced 206 unique sources, which were reviewed by a team of raters, consisting of the first author and doctoral students in clinical psychology whom the first author trained.

Inclusion Criteria

All studies included in the review were English-language meta-analyses published in a peer-reviewed academic journal. Other criteria involved reporting at least one standardized mean difference (SMD) statistic across a set of studies comparing a group diagnosed with ADHD to a group of typically developing controls on at least one neurocognitive measure, defined as a behavioral test assessing a neurocognitive domain. Reviews focusing exclusively on rating scales, neuroimaging measures, movement detection, and/or electroencephalographic data were omitted.

Studies reporting any of three SMD statistics were included (McGrath & Meyer, 2006): Cohen's d uses a pooled variance estimate that is unweighted by the group sizes; Hedge's g instead uses differential weighting of the two groups when group sizes are unequal; finally, δ attempts to correct for a small bias in g as an estimator of the population SMD. Based on the mathematics involved, there is no reason to expect any difference between d and g on average; δ should be slightly larger than g, though the difference declines with larger sample sizes.

In order to achieve a large enough sample of meta-analyses, we did not specify as part of our inclusion criteria specific methods individual studies must have used to diagnose ADHD or to recruit participants for the control group. Only nine of the 34 meta-analyses we ended up reviewing specified a particular method of ADHD diagnoses as an inclusion criterion. Six required that DSM-III, -III-R, -IV, -IV-TR, or ICD-10 criteria be followed; two that questionnaires be used; and one that both a clinical interview and a behavior rating scale be used. Of the remaining 25 meta-analyses, 18 provided information about

the various methods their sampled studies used to diagnose ADHD, and seven made no mention of how ADHD was diagnosed in the sampled studies.

Of the 206 unique references identified, 15 were eliminated because they did not come from peer-reviewed journals, and nine were excluded because they were not in English. The remaining 182 studies were each evaluated by two independent raters to determine if they met inclusion criteria. In cases of rater disagreement, a third rater made the final determination of eligibility. One hundred forty-six articles were excluded because they did not report an SMD that compared the performance of an ADHD group to that of a healthy control group on a neurocognitive measure. Two other articles were later excluded during data extraction because the effect sizes reported could not be converted to *d*, *g*, or *δ*. The final sample consisted of 34 studies that reported 253 summative effect sizes, for a mean of 7.03 per study (range = 1–92). See Fig. 1 for the study flow diagram.

Data Extraction

The following information was extracted for each of the summative SMDs evaluated: number of participants sampled, number of studies aggregated, *p*-values associated with the SMD, analyses performed to determine heterogeneity among sampled studies, measure used, sample age group (i.e., children, adolescents, adults, or a combination of these groups), mean age of the sample, gender composition of ADHD sample, methods used to detect publication bias, whether the authors of the meta-analysis received funding from drug companies, and percent of studies sampled with a total sample size less than or equal to 50. The following variables were also collected, but not enough information was reported in the sampled meta-analyses to analyze in this review: comorbid conditions included in sampled studies, ADHD subtypes included in sampled studies, methods used by sampled studies to diagnose ADHD, and use of a random- or fixed-effects meta-analytic model.

The first author, in consultation with a clinical neuropsychologist and a neuropsychological researcher, categorized each measure into neurocognitive domains. In this way, each summative SMD was associated with one primary neurocognitive domain, used for later analysis.

Results

The distribution of summative SMDs from the 34 meta-analyses is presented in Fig. 2. The SMDs were 96% (244 of 253) positive, indicating that the control group consistently demonstrated better neurocognitive performance than the

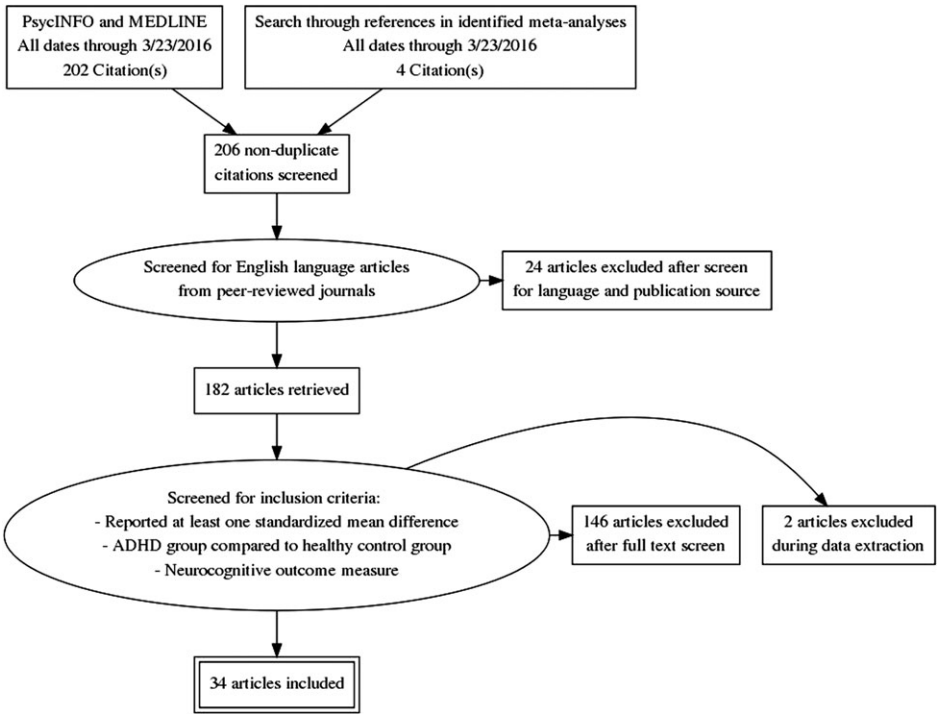

Fig. 1. Study flow diagram.

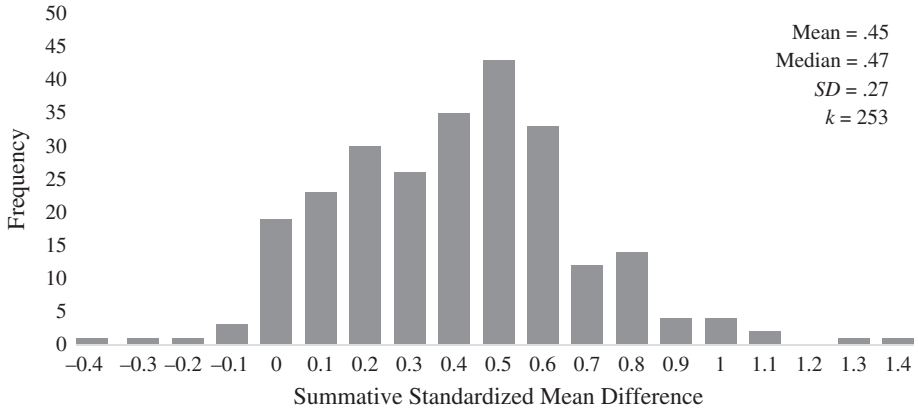

Fig. 2. Distribution of summative standardized mean differences (SMDs).

Table 1. Distribution of *p*-values

p-value	Frequency
< .01	100
.01–.049	13
.05–.099	2
≥ .10	21

ADHD group. Of these, 81% of the SMDs were ≥ .20 (k = 205), 45% were ≥ .50 (114), and 10% were ≥ .80 (26). The distribution was fairly symmetrical about the mean of .45 (SD = .27) and median of .47, with skewness of .26 and kurtosis of .54.

One hundred thirty-six of the summative SMDs from 28 studies included enough information to calculate a *p*-value associated with the effect size. Of these, 83% (113) were significant at $p < .05$. A distribution of *p*-values is shown in Table 1.

A preliminary analysis was conducted to determine if the effect size statistic used by meta-analyses impacted the mean SMD. It was not always obvious which effect size statistic was used in each study. Though g and d are mathematically distinct statistics (McGrath & Meyer, 2006), they are equal unless group sizes differ, and in practice the symbol d is often used for either. For example, *Homack and Riccio (2004) and *Romine and colleagues (2004) referred to their SMD as d but explicitly indicated the use of weighted variance estimates, while *Pennington and Ozonoff (1996) used unweighted variances. In most other cases, it was unclear whether d or g was being computed, so these were combined. Across the 253 effect sizes, 223 were based on Cohen's d or Hedge's g and 30 on the bias-corrected estimator δ. Those studies that reported Cohen's d or Hedges' g had a mean of .45 and median of .47, while those that used the bias-corrected estimator had a mean of .44 and a median of .52. An independent samples t-test revealed no significant differences in SMD based on effect size statistic, $t(251) = .21$, $p = .83$. Therefore, in subsequent analyses all three SMD statistics were combined.

Number of Aggregated Studies

Though each included article had to have at least one SMD that represented an aggregate across a series of analyses, in some cases the number of aggregated analyses was quite small. Sixty-six percent of the summative SMDs were calculated from the results of fewer than 10 studies. This finding is skewed by one meta-analysis (*Hervey, Epstein, & Curry, 2004), in which 51 of 92 SMDs were based on a single analysis. To evaluate the impact of these small-scale aggregates, the mean effect size was computed after weighting by the number of mean differences aggregated in the SMD estimate. One article (*Hasson & Fine, 2012) did not provide information about the number of aggregated analyses, so the two summative SMDs from that meta-analysis were excluded from this calculation. The mean weighted by number of studies was .50 across the remaining 251 summative effects. The frequency distribution of number of analyses aggregated is shown in Fig. 3, along with the unweighted overall mean of those effect sizes within each interval. With one exception, the mean SMD consistently fell between .40 and .60, no matter the number of analyses included in the calculation of the summative SMD.

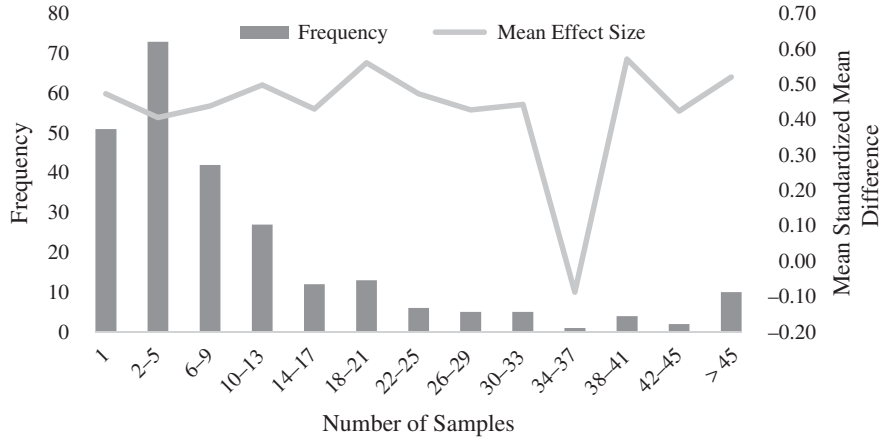

Fig. 3. Frequency and unweighted mean standardized mean difference (SMD) by number of samples.

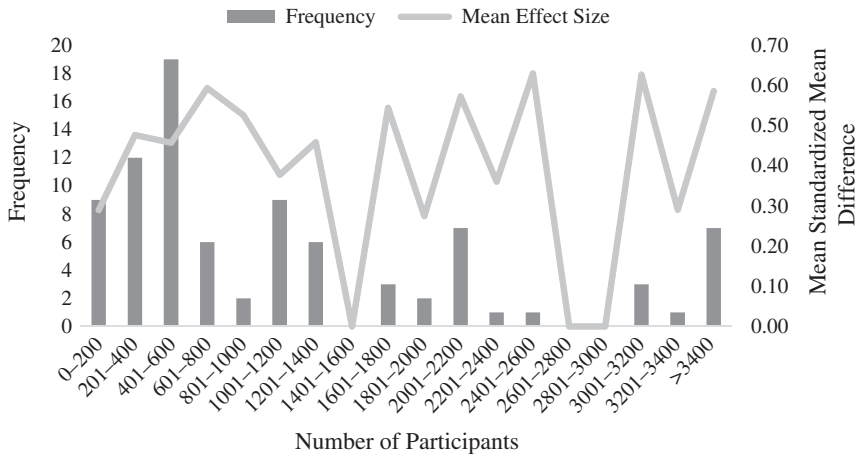

Fig. 4. Frequency and mean standardized mean difference (SMD) by number of participants.

Number of Participants

The number of participants included in the calculation of summative SMDs ranged from 136 to 21,804. Forty-five percent of the summative SMDs involved fewer than 600 participants. The mean weighted by number of participants was .56 for the 88 summative SMDs in which the authors provided the total number of participants across aggregated studies. Figure 4 depicts the frequency of summative SMDs and overall unweighted mean SMDs across the distribution of number of participants. Unweighted mean SMDs ranged from .28 to .63 and did not vary by number of participants in a discernable pattern.

Heterogeneity

Eighty-nine SMDs from 21 meta-analyses provided sufficient information to calculate I^2, an estimate of the percentage of variation across studies due to heterogeneity (Higgins & Thompson, 2002). Figure 5 depicts the distribution of I^2 values and the mean unweighted SMD associated with each I^2 value. Forty percent of studies (36) had I^2 values above 50%, which is considered "substantial" heterogeneity (Deeks, Higgins, & Altman, 2011).

Neurocognitive Domains

Above we summarized the meta-analytic data across all neurocognitive domains to quantify the overall difference in neurocognitive performance between individuals with ADHD and their healthy peers. Here we performed similar analyses within each neurocognitive domain in order to estimate the differences in performance in more specific areas. Supplementary

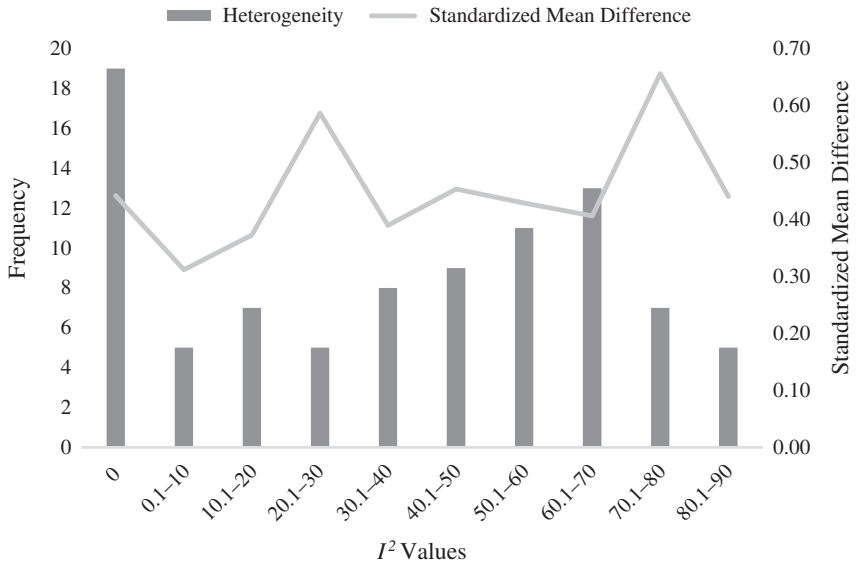

Fig. 5. Mean standardized mean difference (SMD) by heterogeneity.

Table 2. Frequency and mean standardized mean difference (SMD) by neurocognitive domain

Neurocognitive domain	Frequency	*k*	Mean *d*/*δ*	*SD*	Mean *d*/*δ* weighted by *k*
Decision-making	13	192	0.36	0.28	0.41
Fluency	8	50	0.49	0.25	0.49
Intelligence/achievement	13	339	0.51	0.27	0.60
Memory	15	71	0.44	0.29	0.49
Planning/organization	21	37	0.51	0.35	0.49
Reaction time	45	497	0.38	0.26	0.35
Reaction time variability	16	433	0.53	0.32	0.66
Response inhibition	35	438	0.52	0.28	0.52
Selective attention	12	126	0.46	0.24	0.46
Set shifting	24	260	0.35	0.26	0.35
Vigilance	20	282	0.48	0.23	0.56
Working memory	19	156	0.54	0.24	0.54
Other	12	186	0.40	0.19	0.48

Note: *k* = Total number of studies aggregated. Some studies included analyses of more than one neurocognitive domain.

Table S1 lists the mean summative SMD, the total number of participants, and the number of studies aggregated for each meta-analysis, organized by neurocognitive domain. Most summative SMDs reported on a single neurocognitive measure, and some measures were reported in multiple meta-analyses (e.g., continuous performance test omission errors).

Table 2 presents the number of summative SMDs, the unweighted mean SMD, and the mean SMD weighted by number of aggregated analyses for each neurocognitive domain. Because so few meta-analyses reported the number of participants aggregated for each summative SMD, mean SMD weighted by number of participants is not provided. Domain means unweighted by the number of analyses ranged from .35, for set shifting, to .54, for working memory. Weighted mean SMDs ranged from .35, for set shifting, to .66, for reaction time variability. The domains with unweighted mean effects over .50 were working memory (.54), reaction time variability (.53), response inhibition (.52), intelligence/achievement (.51), and planning/organization (.51). When weighted by number of aggregated studies, the domains with mean effects over .50 were reaction time variability (.66), intelligence/achievement (.60), vigilance (.56), working memory (.54), and response inhibition (.52).

Age

Mean age could be calculated for 62 SMD aggregates from 15 meta-analyses. Figure 6 depicts the distribution of mean SMDs by mean age. A U-shaped curve is evident, with larger SMDs observed in children (around .50 in each age group) and adults (varying between .52 and .65). Smaller SMDs were evident in adolescents and emerging adults, with the lowest SMDs

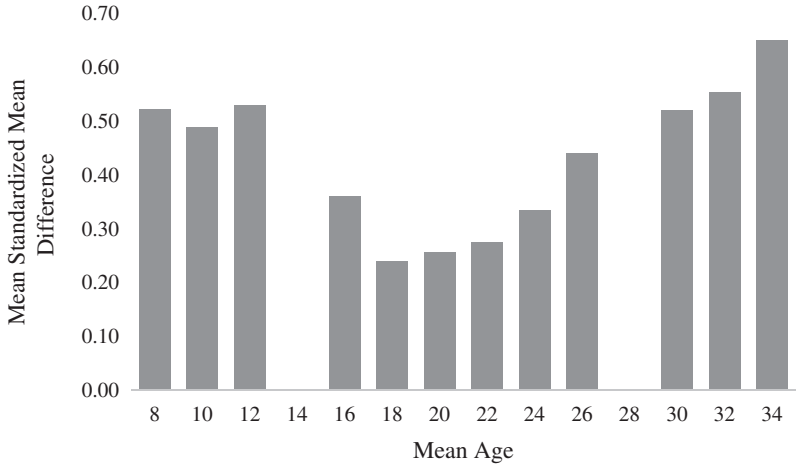

Fig. 6. Mean standardized mean difference (SMD) by mean age.

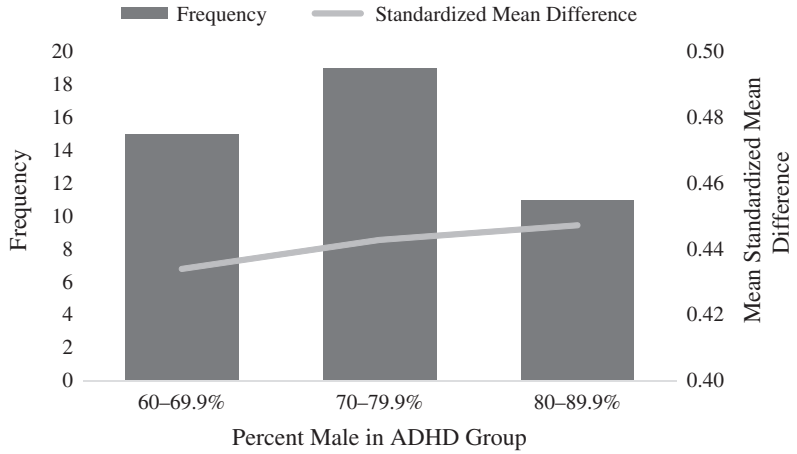

Fig. 7. Mean standardized mean difference (SMD) by percent male in attention-deficit/hyperactivity disorder (ADHD) group.

(below .30) in 18- to 24-year-olds. There was a relationship between mean age and number of participants involved in a SMD, with 85% of participants coming from samples in which the mean age fell between eight and 14 years. Nevertheless, weighting by total number of participants and the number of analyses aggregated had little impact on the overall pattern, suggesting this quadratic relationshipis not an artifact of the fact that there were far more participants who were less than 14 years old (84,717) than participants who were 14 or older (14,754).

Percentage of Males in the Attention-Deficit/Hyperactivity Disorder Group

Because most ADHD research is conducted on predominantly male samples, we evaluated whether gender affected overall mean SMD. Thirteen meta-analyses that reported 48 summative SMDs provided enough information to calculate the percentage of participants in the combined ADHD group that were male. *Hasson and Fine (2012) reported two summative SMDs: one compared the performance of a group of all ADHD males to healthy males, and the other compared the performance of all ADHD females to healthy females. In all other studies where it was possible to compute the percent of males in the ADHD group, this value seemed to represent the percent of males in a convenience sample of ADHD sufferers. The *Hasson and Fine (2012) samples were, therefore, excluded from the gender analysis. In every other case, males represented 60%–90% of the ADHD sample. The unweighted overall mean SMDs did not differ by gender makeup. The frequency and mean SMDs by gender makeup are shown in Fig. 7. The mean SMD consistently fell between .43 and .45, regardless of the percentage of males included in the ADHD group.

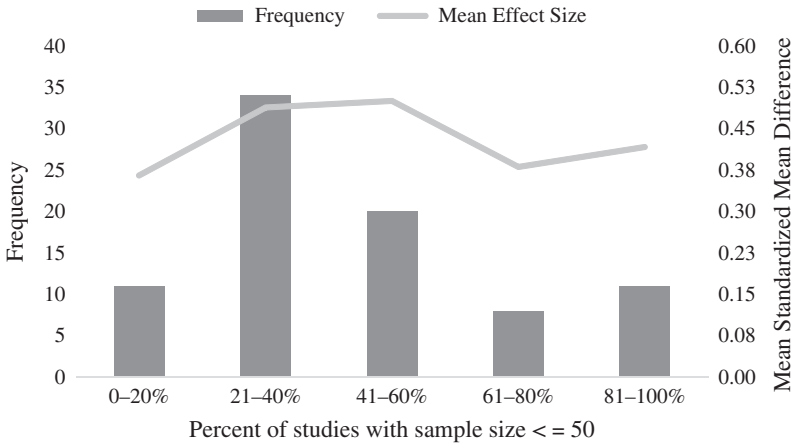

Fig. 8. Frequency and mean standardized mean difference (SMD) by percent of studies with samples of 50 or fewer.

Combination of Moderators

Regression analysis was used to explore the extent to which the above moderators uniquely predicted aggregated SMD. A model with number of SMDs aggregated, number of participants, age, and percentage of ADHD males predicting SMD was significant, $F(5, 43) = 2.66$, $p < .05$, $R^2 = .26$. Age, which was entered into the model as a quadratic variable, emerged as the only unique predictor of SMD; however, the regression coefficient was quite small, $B < .01$, $SE < .01$, $p = .01$.

Publication Bias

Eighty-five of the summative SMDs came from meta-analyses that reported methods used to control for publication bias (e.g., fail-safe *N*, funnel plot); the other 168 SMDs were reported in meta-analyses that did not mention publication bias or report procedures to control for it. The overall mean SMDs were very similar: .44 for those meta-analyses that did control for publication bias versus .46 for those that did not. We specifically wanted to look at the use of Egger's test to control for publication bias, but too few studies included information about this test. Nine SMDs from six meta-analyses reported a value for Egger's test, two of which were significant at $p < .1$, indicating publication bias.

Drug Funding

One hundred eleven summative SMDs came from 26 meta-analyses in which information about funding of the meta-analysis was reported. The unweighted overall mean SMD from meta-analyses that received drug funding was .53 ($k = 23$), compared with .43 ($k = 88$) from meta-analyses whose authors did not receive drug funding.

Percentage of Studies with Small Samples

The mean of SMDs can be biased upward if it is based on small sample sizes; however, this small sample bias is insignificant once samples consist of 50 or more participants (Lipsey & Wilson, 1993). We therefore calculated the percent of studies comprising each summative SMD that had samples of 50 participants or fewer. There was enough information to do so for 84 summative SMDs from 25 meta-analyses. The frequency and overall unweighted mean SMD across percentages of samples of 50 or fewer subjects are displayed in Fig. 8. Mean SMD does not appear to have been meaningfully affected by small sample bias.

Discussion

The present study reviewed 34 published meta-analyses with 253 summative SMDs comparing the neurocognitive performance of individuals with ADHD to that of healthy controls. The summative SMDs were overwhelmingly positive (96%), indicating that the performance of the control group was consistently better than that of the ADHD group across studies and

neurocognitive domains. The unweighted mean SMD, averaged across all studies, was .45. When weighted by the number of aggregated studies, the mean SMD was .50, and when weighted by total number of participants, it was .56. Although these effect sizes are considered medium-sized according to the benchmarks established by Cohen (1988), they are considerably smaller than those found for between-groups difference in behavioral symptoms of ADHD as defined by the DSM (i.e., observed or self-reported symptoms of inattention and/or hyperactivity and impulsivity). One of the meta-analyses reviewed in this study reported that effect sizes for behavioral differences between ADHD and healthy control groups ranged from d = 2.5 to 4.0 (*Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). This range is much larger than that for the SMDs reported in the meta-analyses we reviewed, d = [−.32, 1.41]. It is not surprising that there would be larger between-groups differences in behavioral symptoms of ADHD in light of the fact that these groups were selected based on these same behavioral symptoms. Nevertheless, this range provides a sense of scale; the mean SMD of .45 found in this review is more than five times smaller than the smallest effect size looking at behavioral differences. This indicates that not all individuals with reported or observed behavioral symptoms of ADHD have neurocognitive deficits, or that the neurocognitive deficits of many individuals with ADHD are comparatively small.

The overall mean SMD was quadratically related to age, with larger differences in neurocognitive performance in children and adults with ADHD compared to healthy controls and smaller between-groups differences in adolescents and emerging adults. This was an unexpected result, and while we can offer some hypotheses as to why this result has emerged, we cannot pretend to understand why adolescents and emerging adults have demonstrated fewer neurocognitive deficits. One hypothesis is that this is a result of the high remission rate of ADHD. According to a meta-analysis of ADHD follow-up studies, only about 15% of individuals continue to meet full DSM-IV criteria for ADHD by age 25, though 65% continue to meet criteria for ADHD in partial remission (Faraone et al., 2006). It may be that those individuals who will eventually achieve full remission still meet behavioral criteria for ADHD in adolescence and emerging adulthood, but their neurocognitive performance is closer to that of their same-aged peers. By age 25, however, about 1/3 of individuals who had ADHD in childhood have achieved full remission and are not included in the participant pool for research studies; therefore, one would expect greater differences in neurocognitive performance between healthy controls and this group of adults who still meet criteria for ADHD than would be found during adolescence. This hypothesis could be tested with a longitudinal study comparing the neurocognitive performance of individuals with ADHD to that of healthy controls from early adolescence (around age 12) into adulthood (age 30 and above). If this hypothesis is true, one would expect to see a linear relationship between age and differences in neurocognitive performance when comparing the original cohorts, defined by the presence or absence of ADHD symptoms around age 12. Between-groups differences would decrease in adolescence and early adulthood and continue into adulthood. A different pattern would emerge if the ADHD group were only comprised by currently symptomatic individuals. If only those individuals who continue to report ADHD symptoms are compared to the healthy controls, one would expect to see the same quadratic relationship observed in this review.

Another hypothesis that might explain why adolescents and emerging adults have demonstrated comparatively fewer neurocognitive deficits is related to the widespread neurocognitive changes that occur in all individuals during this period. An increasing amount of research is identifying adolescence and emerging adulthood as a sensitive period in the development of the brain (Fuhrmann, Knoll, & Blakemore, 2015). The increased neuroplasticity of this developmental period may affect the neurocognitive performance of all individuals in this age group, thus decreasing observed between-groups differences. Greater noise during this time period might be dampening our ability to detect a particular signal. It may also be that the neuroplasticity of this period somehow contributes to a decrease in neurocognitive deficits in individuals with ADHD, either directly or by means that may be uncovered by future research.

Mean SMD also differed based on whether meta-analysis authors received drug money: the mean effect size from studies that received funding was .53, compared with a mean SMD of .43 for those studies that received no drug funding. The tendency for studies funded by the pharmaceutical industry to report larger treatment effects for medications is well-documented in experimental studies (Lundh, Lexchin, Mintzes, Schroll, & Bero, 2017), and bias in favor of medications has also been demonstrated in systematic reviews and meta-analyses (Ebrahim, Bance, Athale, Malachowski, & Ioannidis, 2016; Ioannidis, 2016). The current finding suggests estimates of the size of deficits associated with ADHD are also larger when industry funding is involved.

Mean SMD differed somewhat by neurocognitive domain. After weighting by number of aggregated studies, reaction time variability (.66) and intelligence/achievement (.60) emerged as the domains with the largest mean SMDs, and set shifting and reaction time as the domains with the smallest (.35). The relatively large effect size of reaction time variability, most commonly assessed by measuring reaction time standard deviation on tasks like the stop signal task (SST) and continuous performance test (CPT), most strongly supports the default mode model (Sonuga-Barke & Castellanos, 2007). According to this model, an inability to switch from a resting to an active cognitive state would result in greater variability in performance on attentional tasks. *Huang-Pollock and colleagues are also proponents of this default mode model, and in interpreting the results

of their 2012 meta-analysis, emphasized that many of the deficits observed in individuals with ADHD can be attributed to slower and more variable rates of processing information, an artifact of difficulty switching from the default mode to an active cognitive state.

The next largest weighted mean SMD was found in the domain of intelligence and achievement. This result is difficult to interpret because, unlike most of the other domains, intelligence and achievement are global estimates of overall neurocognitive functioning, as opposed to a measure of (supposedly) molar neurocognitive abilities. As a result, intelligence and achievement are highly correlated with all other neurocognitive domains. It is, therefore, not surprising that large differences would be found in this domain, since any neurocognitive deficits would impact intelligence and achievement. It may also be, however, that many of the differences observed between individuals with ADHD and their healthy peers in other domains are artifacts of differences in intelligence and achievement. Although many ADHD studies exclude individuals with an IQ less than 80, few control for IQ scores or test for moderation.

Interestingly, this analysis of neurocognitive domains does not lend strong support to any of the top-down theoretical models of ADHD. Barkley's (1997) model posited that response inhibition is the central neurocognitive function affected in ADHD, but the weighted mean SMD for response inhibition, .52, was almost identical to the weighted mean SMD across all neurocognitive domains, .50, and was not the largest effect observed. Although response inhibition seems to be impaired in individuals with ADHD compared to healthy controls, the evidence does not support the theory that this is the central deficit of the disorder.

There are some inherent flaws in the way that response inhibition is assessed. CPT commission errors and SST stop signal reaction time (SSRT) are the neuropsychological tests most often used to assess response inhibition, and there is a long history of debate as to the extent that these variables are actually measuring that construct. Corkum and Siegel (1993) reviewed the extant literature on visual CPT paradigms and identified the large number of variables that are often not controlled for or even mentioned in studies using the CPT, all of which can affect test results. Most importantly, as relates to this study, they identified several variables intrinsic to each CPT paradigm that can affect test results, such as whether discrimination is successive or simultaneous, the duration of exposure of the stimuli, the amount of time between presentations of stimuli, the duration of the entire task, and the target probability. Likewise, *Huang-Pollock, Karalunas, Tam, and Moore (2012) stressed the importance of using CPT paradigms with < 50% target probability, excluding two of the most common paradigms, the Conners' CPT and the Sustained Attention to Response Task. The greater the target probability, they explained, the more likely the participant will make a commission error, whereas the smaller the target probability, the more likely he or she will make an omission error.

Many, but not all, of the meta-analyses we reviewed did not control for CPT paradigmatic variables like target probability, and this review combined all versions of the CPT indiscriminately, not only with each other, but with other measures, like SSRT. This almost certainly affected the mean SMD. It is important to note, however, that the mean SMDs of all other neurocognitive domains were similarly calculated, and all of the results reported here are heterogeneous. This was a deliberate choice in order to identify larger patterns in the vast literature. It is possible, though unlikely, that the heterogeneity of the response inhibition measures disproportionately affected the mean SMD thereof. It may be that the inclusion of CPT paradigms with higher target probability deflated the mean SMD, because these paradigms increase the likelihood of commission errors for all participants, making it more difficult to distinguish ADHD from control. It also may be that commission errors committed by both healthy and ADHD participants increase proportionally on paradigms with higher target probability paradigms, in which case mean SMDs would be comparable. This is a question that future studies might evaluate.

As for SSRT, *Lijffijt and colleagues (2005) critiqued previous analyses of SSRT, stating that this variable is too strongly correlated with reaction time. They posited that stop signal delay, which is used to calculate the theoretical SSRT, is a purer measure of response inhibition. They hypothesized that differences in SSRT performance indicate that individuals with ADHD have slower reaction time than heathy controls, as opposed to impaired response inhibition. The results of the present study overwhelmingly refute this hypothesis. Reaction time had the lowest weighted mean SMD. Moreover, *Lijffijt et al.'s (2005) hypothesis would suggest weaker differences in response inhibition, not the other way around.

The mean SMD we reported for response inhibition is a best estimate, given the heterogeneity and controversy inherent in the studies that have measured this construct. It is one of the most often researched constructs in the literature we reviewed, with results from 438 individual studies comprising this composite. The mean SMD we found (.52) was the same, whether unweighted or weighted by number of aggregate studies. Elements of Barkley's (1997) theory are borne out by these results: it is clear that response inhibition is impaired in individuals with ADHD, as is working memory, another key domain implicated by Barkley. Nevertheless, if response inhibition were the central deficit of ADHD, and all other functions were secondary to that deficit, one would expect a larger mean SMD in this domain, especially as compared with the others.

More recent integrative models have hypothesized that ADHD is a disorder of both top-down and bottom-up processes. According to these theories, executive functions, including working memory and attention, and decision-making factors, such

as motivation and rewards, are implicated in ADHD. While there is certainly evidence that most executive functions are impaired in ADHD, the weighted mean SMD of the decision-making domain was much smaller than that of most of the other domains (.41). Although this decision-making appears to be affected by ADHD, the results offer weak evidence that it is a central deficit of the disorder.

Several theories distinguish between inattentive and combined ADHD presentations, hypothesizing that the inattentive presentation is a disorder of executive functions, whereas the combined presentation is an affective one that manifests itself in decision-making and reward learning. Unfortunately, most of the meta-analyses reviewed did not report the number of participants who were diagnosed as inattentive, hyperactive/impulsive, or combined type ADHD; therefore, it was not possible to analyze whether mean SMDs of each neurocognitive domain differed by ADHD presentation. Future studies should evaluate whether neurocognitive performance does indeed differ by ADHD type.

The clinical implications of these analyses are provided with caution. When assessing for ADHD, no one test should be given too much credence, as evidenced by the large standard deviations of the mean SMDs across all domains. Although group differences are found in all domains, individual performances vary greatly, and no one measure can be said to differentiate individuals with ADHD from their typically developing peers. That being said, it would behoove clinicians to emphasize measures of reaction time variability, intelligence and achievement, vigilance, working memory, and response inhibition more in their evaluations, and reaction time, set shifting, and decision-making less. Given the relatively large mean SMD of reaction time variability as compared with the other domains, clinicians may choose to give the most weight to measures within this domain.

There were several limitations to this review. This study was only able to summarize the information provided by the meta-analyses reviewed. Fewer than half of the summative SMDs, for example, were accompanied by information about the total number of participants from all aggregated studies. Additionally, specific information about ADHD subtypes, methods of diagnosing ADHD, and comorbid conditions was not readily available for most summative SMDs, and these very important potential moderators could not be evaluated in the present analyses.

In some instances, the SMDs reported in individual research studies were included in more than one meta-analysis reviewed here, leading to some overlap in the studies aggregated and possibly affecting the resulting mean SMDs calculated in this review. It was not deemed reasonably feasible to control for this duplication; therefore, this remains a limitation to the present study.

For the purposes of the present study, summative SMDs were categorized by neurocognitive domain. Most measures, however, do not uniquely assess for a single neurocognitive function; multiple neurocognitive processes are involved in almost every task and every measure. For each summative SMD, the first author, a clinical neuropsychologist, and a neuropsychological researcher determined the primary neuropsychological domain being assessed. Nevertheless, there was a good deal of subjectivity involved in the process, which may have affected the results. Moreover, the fact that multiple neurocognitive processes are involved in each task likely has resulted in shared variance among the mean SMDs reported in this review. This last limitation is not unique to the present review, but rather common to all research that uses behavioral tests in an attempt to measure a single neurocognitive construct.

Conclusions

The present review is the first to aggregate meta-analyses comparing the neurocognitive performance of individuals with ADHD to that of healthy controls and is the most in-depth evaluation of the neurocognitive profile of ADHD to date. The meta-analyses reviewed consistently found that typically developing individuals outperformed their peers with ADHD. Between-groups differences were larger in children and adults and smaller in adolescents. Additionally, meta-analyses that received drug funding found larger effect sizes than those without drug funding. Individuals with ADHD had the greatest deficits relative to healthy controls in the neurocognitive domains of reaction time variability, intelligence/achievement, vigilance, working memory, and response inhibition. These results lend support to the default mode model, which posits that deficits in ADHD arise because the brain has difficulty switching from rest mode to an active mode.

Supplementary Material

Supplementary material is available at *Archives of Clinical Neuropsychology* online.

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Conflict of Interest

None declared.

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Appendix A: Search Terms for Review of Meta-Analyses

Meta-analysis AND (ADHD OR attention deficit disorder OR attention deficit hyperactivity disorder OR attention-deficit/hyperactivity disorder) AND (neuropsych* OR cognitive test* OR go no go OR go/no-go OR stopping OR saccade OR conflict motor task OR inhibition OR continuous performance test OR Conners OR CPT OR stop signal* OR SST OR SSRT OR delayed oculomotor response* OR matching familiar figures* OR trailmaking* OR trail making* OR sustained attention to response task OR letter cancellation* OR paced auditory serial addition* OR PASAT OR shifting sets* OR stroop OR color-word OR 3RT OR finger tapping OR digit span* OR target orientation test OR Wechsler OR Woodcock OR digit symbol OR GDS* OR TOAD OR logical memory* OR reading span OR 2-back OR n-back OR 1-back OR auditory verbal learning* OR letter-number sequencing OR letter number sequencing OR cognitive activation* OR dichotic memory* OR whole report* OR continuous word recognition* OR number-letter OR visual reproduction* OR CANTAB OR spatial span* OR corsi block tapping* OR LAMB OR Rey* OR Rey-Osterreith* OR complex figure* OR visual memory span* OR simon game OR simon task OR design memory OR counting span OR sentence span OR CML OR numbers backward OR finger windows OR arithmetic OR auditory consonant trigrams OR mazes OR Tower of Hanoi OR Tower of London OR Delis-Kaplan OR Porteus Mazes OR progressive planning* OR Wisconsin card sort* OR number/letter sequencing OR number recall OR dot test OR spatial memory OR word order OR phonological memory test OR digit recall OR numbers forward OR missing digit OR California verbal learning test OR Kimura recurring figures test OR letter fluency OR category fluency OR Controlled Oral Word Association Test OR COWAT OR animal fluency* OR cookie theft test OR design fluency OR similarities OR information OR vocabulary OR reinforcement task OR Eriksen Flanker* OR attention network test OR fast track OR stop change task OR complex visual memory search OR visual orienting and detection task OR test of variables of attention OR TOVA OR flicker OR Delayed SRT OR CRT visual attention OR KITAP OR anterior network test OR perceptual discrimination task OR probabilistic learning OR divided attention OR focused attention OR 0-back OR n-back OR Sternberg task OR visual search task OR word matching task OR tracking task OR reversal task OR bilateral responding task OR response time OR SRT monetary incentive task OR SCAT OR motor inhibition OR cue-detection task OR Go-change task OR ETC)

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References marked with an asterisk indicate studies included in the meta-analysis

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