Attention-Deficit/Hyperactivity Disorder and Monetary Delay Discounting: A Meta-Analysis of Case-Control Studies

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Abstract

Background—A growing number of studies have investigated delay discounting, a behavioral economic index of impulsivity, and its relevance to attention deficit hyperactivity disorder (ADHD), but with mixed findings. The current meta-analysis synthesizes the literature on the relationship between monetary delay discounting and ADHD in studies using case-control designs. Specifically, the objectives were: 1) to characterize the aggregated differences in monetary delay discounting between individuals with ADHD (cases) and controls in studies using categorical case-control designs; 2) to examine potential differences based on sample age (<18 vs. >18), reward outcome (real vs. hypothetical), and prevalence of conduct disorder and oppositional defiant disorder in the sample; and 3) to evaluate potential small-study (publication) bias in the literature.

Methods—From 567 candidate articles, 21 independent investigations yielded 25 case-control comparisons (total N=3,913). Random effects meta-analysis was conducted using Cohen's d as the common effect size. Publication bias was evaluated using fail-safe N, Begg-Mazumdar and Egger tests, and metaregression of publication year and effect size.

Results—Across studies, a statistically significant difference of medium magnitude effect size was present for the case-control comparisons (d=0.43; $p < 10^{-15}$). No significant differences based on sample age, reward outcome, or comorbid status was detected. Minimal heterogeneity and evidence of publication bias was present.

Conclusions—These findings provide robust evidence that delay discounting is significantly elevated among individuals with ADHD compared to controls. Gaps in the literature and the importance of characterizing the neural and genetic bases of this relationship are discussed.

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Keywords

Attention deficit hyperactivity disorder; delay discounting; impulsivity; intertemporal choice; decision making; meta-analysis

Introduction

Since the earliest clinical descriptions of attention deficit hyperactivity disorder (ADHD), high levels of impulsivity have been described as a cardinal feature of the disorder (1). However, although impulsivity can be broadly defined as a person's capacity to inhibit or regulate arising impulses, it is operationalized in a variety of different ways (2; 3), including both self-report questionnaires and behavioral tasks. Importantly, these diverse measures are often not highly intercorrelated, undermining the notion of a single underlying process. A recent meta-analysis of the interrelationships among self-report and behavioral task measures of impulsivity indicated little overlap between the two domains (4). Recent factor analyses suggest that when multiple measures of impulsivity are examined concurrently, latent aggregations of measures emerge (5-7), suggesting that impulsivity appears to be a psychological genus that subsumes a number of species.

One discrete form of impulsivity is delay discounting, reflecting how much a person devalues a reward based on its delay in time. Delay discounting is an index of impulsivity from behavioral economics, a hybrid discipline that integrates principles and methods from psychology and economics to study choice behavior. It is commonly measured using decision making tasks that pose a variety of decisions, such as "Would you rather have $40 today or $100 in a month?," with systematic variation of smaller immediate rewards and delays in time, while keeping the larger delayed reward consistent. Then, across an array of smaller (discounted) immediate rewards and future delays, an individual's overall devaluation of the larger delayed reward can be generated. Often, this is via examination of where an individual switches preferences from smaller-sooner to large-later rewards and then using those points of indifference across delays to model the individual's temporal discounting function. A widely used model uses a hyperbolic function, $v_d = V/(1+kd)$ (8), where v_d is the discounted value of the delayed reward, V is the objective value of the delayed amount, d is the delay duration, and k is the derived parameter that characterizes the degree of future reward discounting. Alternatively, the 'switchpoints' can be used to generate an individual's discounting curve and area under the curve (AUC) can be used as a measure of future discounting (9). Of note, larger k values reflect more precipitous devaluation of future rewards (reflecting a larger denominator in the equation), whereas the opposite is true for AUC values (reflecting a smaller space beneath the curve). Common to all methods is that if a delayed reward loses value more rapidly, the individual is considered more impulsive. Of note, delay discounting tasks are related to delay of gratification paradigms (10), such as the "marshmallow test" (11), but differ insofar as the latter typically have a real-time component in which participants can alter their preference at any point, incorporating an element of *in vivo* temptation.

There has been longstanding experimental and theoretical interest in steep discounting of delayed rewards as a feature of ADHD. Empirically, early studies used simple choice tasks and found support for intertemporal choice deficits among children with ADHD (12; 13). Subsequently, studies using iterative behavioral tasks systematically examining preferences for monetary rewards have revealed similar patterns in children, adolescents, and adults (14-18). Theoretically, steep delay discounting is considered a hallmark deficit of ADHD (19-21), akin to deficits in response inhibition and sustained attention(22; 23). The underlying deficit has been theorized to be hypoactivity in mesocortical dopamine neurotransmission based on preclinical and functional magnetic resonance imaging (fMRI) studies (19; 24), although characterizing these processes remains an active area of inquiry. In addition, steep temporal discounting may be a cause of the high comorbidity between ADHD and substance use disorders (SUDs) (25; 26). For example, individuals with ADHD have, depending on the drug type, a twofold-to-eightfold higher prevalence of substance use disorder (25). Conversely, in a large recent study of treatment-seeking individuals with SUDs, over 40% screened positive for ADHD (27). This substantial overlap suggests some level of common etiological causality, one form of which may be delay discounting. Behavioral studies have found evidence of more precipitous temporal discounting in individuals with alcohol use disorders (28), nicotine dependence (29), cocaine dependence (30), and opioid dependence (31) compared to matched control participants. Thus, steep discounting of delayed rewards may be a common risk process in ADHD and addiction.

As a proliferation of studies on delay discounting and ADHD has emerged over the last five years, several have not reported significant associations (32; 33), suggesting the link may be weaker or more ambiguous than initially believed. Furthermore, the accumulating literature has not been systematically examined to characterize overall patterns of findings and possible bias. This was the focus of the current meta-analysis. Specifically, the present study had three aims: 1) to characterize the relationship between monetary delay discounting and ADHD in previously published case-control comparisons; 2) to examine three potential moderators of effects, namely, sample age (<18 vs. >18), reward outcome (real vs. hypothetical), and prevalence of conduct disorder/ oppositional defiant disorder in the sample; and 3) to investigate the presence of small-study bias, reflecting the probability of publication bias.

Methods and Materials

Meta-analysis sample

The initial inclusion criterion was any peer-reviewed published study or unpublished dissertation reporting comparisons of delay discounting between a group meeting ADHD diagnostic criteria and a control group. To minimize substantial methodological variability, studies were restricted to delay discounting of monetary rewards (e.g., simple choice tasks using golden donkeys, spaceships, etc., were excluded) and studies of probability discounting and social discounting were excluded. Studies were identified via a literature search using the PubMed and PsyclNFO databases as of September 25, 2015. The specific Boolean terms entered were ("discounting" OR "delay of gratification") AND ("attention" OR "ADHD"). The search term "ADHD" is automatically expanded to include "attention

deficit disorder with hyperactivity" OR ("attention" AND "deficit" AND "disorder" AND "hyperactivity"). A total of 567 records were generated, of which 422 were unique after eliminating duplicates from the two databases, and 49 were relevant. Full-text reviews were conducted on the relevant studies, yielding 21 viable studies with 25 distinct comparisons. Records were excluded if they used rodent models, were review papers, or if they did not collect data on either ADHD or delay discounting. Two of the relevant studies reported overlapping data. After contacting the authors, only the more extensive of these two papers was included. The meta-analysis was performed on the 25 viable comparisons of delay discounting and ADHD. Meta-analysis of continuous associations was considered but ultimately not pursued because of a paucity of studies (k = 3). A PRISMA flow diagram that is consistent with meta-analyses guidelines is provided in Figure 1 (34).

Sample characteristics

Effect sizes reflecting differences between an ADHD-positive group and a control group were available for 21 of the 422 uniquely identified articles, yielding 25 comparisons. Fifteen of these relevant comparisons reported statistically significant differences in DD performance between ADHD and control groups and ten reported non-significant differences. Three individual papers provided results for delay discounting at multiple magnitudes. All reported comparisons were included in order to maximize representation of the literature, but a follow-up analysis included a single meta-analyzed effect size from studies using multiple measures.

Individual study characteristics are available in Table 1 and illustrate the wide variation of study protocol. Sample sizes range considerably, from N=36 to N=1,298 (total N=3,913), with an average sample size of 157. The average age within studies ranged from 7.9 to 36.9 years old, with a total sample average age of 21.0 and median study age of 16.0. The delayed reward for various discounting tasks were primarily for hypothetical outcomes, ranging from $0.10 to $5000, with an average of $380 and a median of $42.50. The vast majority of studies used k or AUC as the index of discounting, but one study reported individual points of indifference and one reported impulsive choice ratio and both included an aggregated F statistic for a group contrast; the latter was used as an overall index of discounting.

Meta-analytic approach

Both fixed and random effects meta-analytic approaches were considered, but a random effects approach was selected as the primary method in light of methodological heterogeneity. The effect size used in the meta-analysis of case-control studies was Cohen's d (35). This value was either identified directly from the publication or generated from reported statistics. Effect sizes generated using AUC were inverted to be consistent with those generated using k values. In three viable studies, sufficient information to generate effect sizes was not provided. The authors of these studies were contacted and all were able to supply the necessary data. A fixed effects approach was applied as a follow-up strategy to characterize heterogeneity of effect size. Cochran's Q and I^2 are two common indices that determine heterogeneity of effect size. Cochran's Q statistic reflects the sum of square differences among the individual weighted study effects and the overall mean. Q tests the

significance of heterogeneity using a χ^2 test. I^2 reflects the percentage of study effect size variation that is explained by true heterogeneity. Values of I^2 25% indicate low heterogeneity, ~ 50% indicate moderate heterogeneity, and 75% indicate high heterogeneity across studies (36). Following the analysis of all included studies, a 'jackknife' analysis systematically excluding each effect size was conducted. In addition, because active ADHD medication could suppress observed differences between groups, a follow-up analysis was conducted excluding studies including medicated participants with no washout period. Participant average age (below 18 versus above 18), task outcome (hypothetical versus actual), and percentage of comorbid conduct disorder and conduct disorder or oppositional defiant disorder were examined as moderators. For the latter, studies were coded based on the reported prevalence of the comorbidities, with absent information coded as zero. Age and task outcome were tested using the Q statistic associated with the between groups difference in a mixed effects analysis. Percent comorbidity was examined using meta-regression. Four indices were used to evaluate publication bias: the classic fail-safe N approach; examination of the funnel plots of sample size and effect size via the two-tailed Begg-Mazumdar test (37), measuring the interdependence of variance and effect size; and the one-tailed Egger's test (38), measuring asymmetry of the funnel plot away from negative findings (assuming no publication bias for positive findings); and meta-regression to examine the relationship between year of publication and effect size. Overall evidence for bias was based on consideration of all four indices. Using Duval and Tweedie's trim and fill approach (39) adjusted estimates of effect size were generated based on imputed unpublished studies. Meta-analytic computations were conducted using Comprehensive Meta-analysis 2.0 (40). The methods of this meta-analysis conformed to the preferred reporting items for systematic reviews and meta-analysis (PRISMA) standards (34).

Results

Primary Meta-Analysis Findings

Overall, the difference between groups was of medium effect size ($d = 0.43$) and highly statistically significant ($p < 10^{-15}$). The forest plot of the aggregated effect is presented in Figure 2. Although a random effects model was the primary analytic strategy, a fixed effects analysis was conducted to characterize heterogeneity, which was not significantly present across the sample of studies ($Q = 25.91$, $p = .36$; $I^2 = 7.56$), meaning that both models produce virtually identical results. Re-running the primary analysis and systematically excluding each study generated very similar effect sizes and significance levels ($ds = .42$-.45, all $ps < 10^{-15}$), suggesting a limited influence of any single study on the results. Re-running the primary analysis with a consolidation of effect sizes from studies using multiple measures revealed a similar effect size ($d = 0.50$, $p < 10^{-15}$), suggesting a modest influence. The majority of studies indicated that participants had been abstinent from medication for at least 48hrs, were medication naïve, or did not discuss medication status. However, after removing three studies from the analysis that indicated participants had been medicated during the study, the effect-size increased slightly ($d = 0.45$, $p < 10^{-15}$).

Relationship to Age, Task Outcome, and Comorbidity with Conduct Disorder/Oppositional Defiant Disorder

Although there was no significant heterogeneity, the studies differed noticeably in the age of the samples (Table 1) and were further examined by studies using child/adolescent participants (i.e., mean age <18) and adult participants (i.e., mean age >18). In studies that focused on children and adolescents with ADHD, the difference between groups was again highly statistically significant and of medium effect size ($d = .47$; $p < 10^{-12}$). Similarly, in studies that used adult participants, the difference was also statistically significant and the effect size was of medium magnitude ($d = 0.40$; $p < 10^{-11}$), albeit somewhat smaller. However, the difference in effect size was not statistically significant ($Q = .69$, $p = .41$). In addition, a meta-regression of age and effect size was non-significant (slope = $-.004$, $p = .25$). Thus, the magnitude of difference in delay discounting between individuals with ADHD and controls appears to be generally consistent across age.

With regard to tasks using hypothetical versus actual rewards, tasks using hypothetical outcomes exhibited somewhat larger effect sizes ($d = 0.44$, $p < 10^{-15}$ versus $d = 0.41$; $p < 0.03$), but the difference was not statistically significant ($Q = 0.016$, $p = 0.90$). However, it is notable that there were many more effect sizes for tasks with hypothetical outcomes than actual outcomes (k=20 versus k=5; Table 1).

The meta-regression analysis of percentage of participants with conduct disorder was nonsignificant (slope = .84, $p = .17$), as was the meta-regression of percentage of participants with conduct disorder and/or oppositional defiant disorder (slope = $-.05$, $p = .72$).

Publication Bias

For the primary meta-analysis findings, none of the four indices indicated small-study bias. The classic fail-safe N indicated that there would need to be 676 unpublished studies to raise the p-value to above the threshold for statistical significance. With regard to the funnel plot (Figure 3), Kendall's $\tau = 0.12$ ($p = 0.40$) and the Egger's test intercept = 0.43 ($p = 0.53$), do not suggest publication bias. Finally, a meta-regression indicated no statistically significant relationship between year of publication and effect size (slope = 0.011; $p = 0.29$). Duval and Tweedie's trim and fill method (39) indicated the possibility of four unpublished studies and, if they were included in the meta-analysis, then the effect size would change from 0.43 to 0.40 (Figure 3).

Discussion

The primary aim of the present meta-analysis was to quantitatively aggregate the findings across published studies comparing monetary delay discounting in individuals with ADHD to healthy control individuals. The results provide very strong evidence of significantly higher discounting of future rewards in ADHD individuals. Indeed, the level of statistical significance observed ($p < 10^{-15}$) would meet the highest evidentiary thresholds in statistics. In terms of the magnitude of the difference, the aggregated effect size was of medium magnitude, reflecting approximately half a standard deviation. Additional analyses

suggested that there was relatively little heterogeneity of effect size and no single study had a substantial effect on the aggregated effect size. Notably, approximately half of the studies focused on children and the other half focused on adults, but neither categorical nor continuous analyses suggested that the effect sizes were systematically different by age. Similarly, no significant difference was present between studies using tasks with hypothetical outcomes and those using actual outcomes, although the lopsided numbers of studies makes this conclusion somewhat more tentative. Meta-regressions of the relationship effect size and percent of participants with conduct disorder and oppositional defiant disorder did not suggest systematic differences, although caution should be applied to this finding because many of the included studies did not fully characterize the sample for these conditions. For the primary meta-analysis, a further goal of the meta-analysis was to characterize the potential for publication bias in the literature on delay discounting and ADHD. In this case, the bias indicators suggest a large number of null findings would need to be in the 'file-drawer' (41) to render the overall conclusion null and that the interrelationships between effect size magnitude and variance or year of publication are not suggestive of small study bias. Of course, it is worth noting that these are imperfect measures, but the consistent absence of evidence for publication bias is supportive of the overall conclusions. Collectively, these findings suggest that elevated monetary delay discounting is a robust distinguishing feature of ADHD in studies using case-control designs and that this is consistent in the context of various collateral variables.

These findings have a number of potentially important implications. To start, a clear implication is the need to understand how the observed difference is instantiated neurally. In general, fMRI studies have identified several domains of abnormal functionality in ADHD, including reward, attention, inhibition, timing, and default mode activity (42-48). Individuals with ADHD have been found to exhibit reduced ventral striatal activity in response to reward, opposite to their healthy control counterparts (47; 48). Scheres *et al.* suggest that neural hypo-responsiveness to anticipated reward may result in compensatory increases of reward-seeking behavior - a key characteristic of ADHD and an elemental process within delay discounting decision making (47). Indeed, studies of delay discounting using fMRI have directly implicated the ventral striatum in delay discounting, among a number of other regions, including subunits of prefrontal cortex, posterior cingulate cortex, anterior insula, and anterior cingulate (24; 49). Among these active regions, multiple accounts for both integrated and opposing relationships have been proposed, although the data do not support any account definitively at this point (24). However, the regions implicated in neuroimaging studies, particularly the ventral striatum and prefrontal cortex, are highly compatible with findings from preclinical studies using animal models of delay discounting (50; 51).

Directly speaking to this question for ADHD, a small number of studies have examined the neural correlates of delay discounting in individuals with ADHD compared to controls using fMRI. In a pediatric sample, boys with ADHD exhibited significantly less activation in inferior prefrontal cortex, orbitofrontal cortex, and inferior parietal lobule compared to controls during a contrast of larger delayed choices minus smaller immediate choices (52). In another pediatric study, three clinical groups - individuals with ADHD, individuals with autism spectrum disorder, and individuals with both - were contrasted with each other and control participants (53). In this case, the comorbid group exhibited the most pronounced

differences in terms of activation, specifically in areas of ventromedial prefrontal cortex, anterior cingulate, inferior frontal cortex and ventral striatum, among others. Interestingly, the ADHD-only group exhibited some of these differences but also exhibited more robust associations between inferior frontal cortex activity and temporal discounting functions. One study has investigated the neural correlates of delay discounting in adults with ADHD versus matched controls, reporting reduced ventral striatal activity during both choices for immediate rewards and delayed rewards (54), which is consistent with the hypoactive reward system hypothesis. In addition, Plichta et al. (54) detected greater activity in the dorsal caudate and amygdala in ADHD participants during delayed reward choices and, in turn, this activity was associated with ADHD symptoms, a pattern that may reflect delay aversion in ADHD.

Notably, these findings are highly compatible with a meta-analysis of monetary delay discounting in studies of drug addiction (55). That study similarly found evidence of a medium effect size difference in delay discounting between addiction criterion groups and control participants. Interestingly, most studies on delay discounting and addiction do not report ADHD status or symptoms, raising the question of whether observed elevations are in part a function of unidentified ADHD. This is necessarily speculation, but the results of this meta-analysis provide further support for the need to understand the intersection of ADHD and addictive disorders. It is also notable that similar general patterns of striatal hypoactivity are seen in substance users (56), indicating the possibility that reduced ventral striatal activity may be a shared trait between the two populations. In addition, fMRI studies on delay discounting in individuals with SUDs compared to control participants, have revealed similar findings to those on ADHD. For example, alcohol misuse has been implicated with distinct patterns of neural activity in subunits of the prefrontal cortex, parietal cortex, anterior cingulate and anterior insula, among others, during delay discounting decision making (57-59). Future imaging studies have the potential to disentangle commonalities and differences between individuals with addictive disorders and ADHD at an even finer level.

Finally, a more oblique implication of these findings is that they provide further support for studies investigating delay discounting in efforts to understand genetic influences on ADHD. There is extensive evidence of the heritability of ADHD (42), including family, twin, and adoption studies (60-62). However, attempts to determine the molecular basis for genetic influences have been less successful than anticipated, potentially because of the heterogeneity of the diagnosis. Endophenotypes, or genetically-influenced mechanistic variables that are putatively simpler than clinical phenotypes (63; 64), are hoped to inform psychiatric genetics by providing a simpler genetic architecture, leading to more robust and reliable associations, and revealing the etiological processes that confer risk and protection. With regard to delay discounting, there is evidence that delay discounting preferences are heritable from studies using both non-human animals (65; 66) and twin cohorts (67; 68). In addition, a small number of molecular genetic studies have implicated dopamine- related polymorphisms and delay discounting (58; 69-72) and one has investigated this question among individuals with ADHD (73). Paloyelis et al. (73) found evidence that rs4680 genotype in *COMT* and *DAT1* haplotype predicted discounting rates in a case-control study of adolescents with ADHD. Specifically, A-allele *COMT* homozygotes exhibited higher discounting of the future across both groups, and an interaction was present between *DAT1*

haplotype status and diagnosis such that the group without ADHD who were carriers of the *DAT1* risk haplotype exhibited discounting levels that were similarly steep to the group with ADHD. These links to dopamine-related loci are consistent with preclinical studies that implicate dopamine neurotransmission with delay discounting. Of note, however, serotonin and norepinephrine also appear to play important roles (50; 51; 74) and two studies have found associations between polymorphisms in serotonin and noradrenergic genes and delay discounting preferences (75; 76). Although genetic studies are at an early stage, the current findings support the need for further attempts to examine whether delay discounting can be used to understand one pathway for genetic influences on ADHD.

Despite generally robust findings, it is important to note that the current study does not address a number of issues. Most studies did not make a distinction between ADHD subtypes and it is important to note that, when treated as different groups, some studies have reported that ADHD subtypes exhibit significantly different results for delay discounting (77) and Scheres et al. reported that symptoms of these different ADHD subtypes correlated with delay discounting differently (78). Future studies will be needed to tease out potentially meaningful differences by ADHD subtype. In addition, due to the small number of studies identified, this meta-analysis could not address continuous relationships between delay discounting and ADHD symptoms. Clearly, there is a need for more research examining this relationship and when more studies are present, it will be important to determine if parallel findings are present or whether any systematic differences emerge (e.g., larger effect sizes in continuous studies). Finally, from a methodological standpoint, the study focused on monetary delay discounting tasks, not simple choice tasks that use points or other outcomes and substantially different procedures (e.g., 71). These tasks have revealed important findings in fractionating elemental deficits in ADHD (80) but were considered qualitatively different from the typical monetary delay discounting tasks. Thus, the findings cannot speak to that domain of the literature.

These considerations notwithstanding, the current meta-analysis nonetheless provides robust validation for steep discounting of future monetary rewards as a feature of individuals with ADHD, with minimal heterogeneity of effect size or publication bias. As such, these findings support further focus on understanding the role of delay discounting in the etiology of ADHD in general, and the dissection of the neural and genetic underpinnings of this relationship in particular.

Acknowledgments

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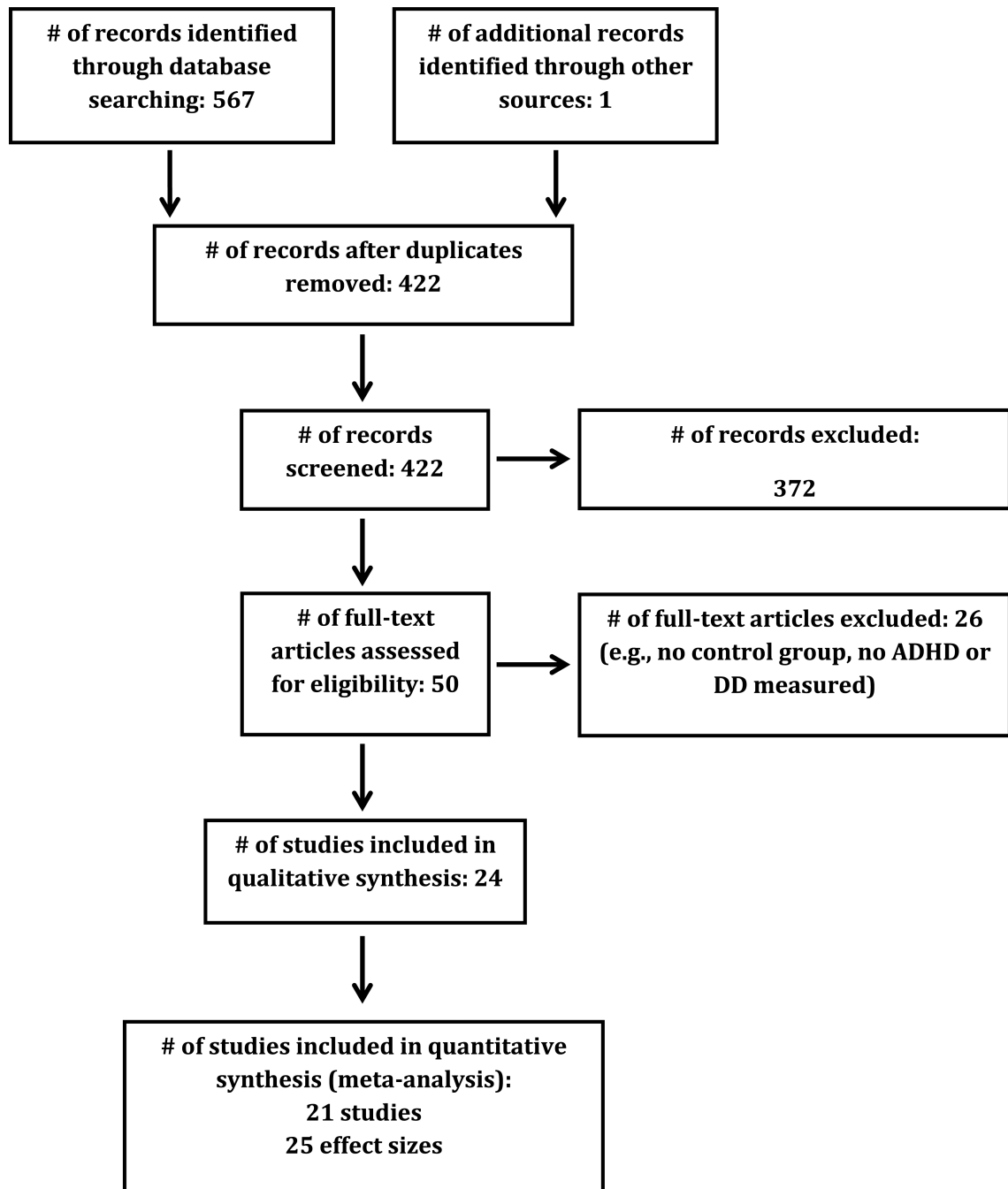
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              │  articles assessed   │───────▶│ (e.g., no control group, no ADHD or │
              │  for eligibility: 50 │        │        DD measured)          │
              └─────────────────────┘        └──────────────────────────────┘
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              │ # of studies included in │
              │ qualitative synthesis: 24│
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        │           25 effect sizes                │
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Figure 1.
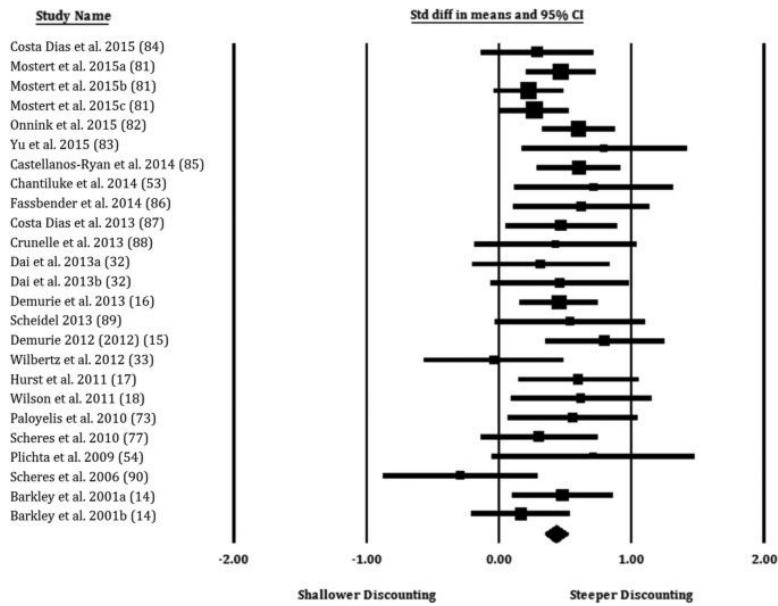Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) inclusion flow diagram.

Figure 2.
Forest plot providing effect sizes (standard differences in means; *d*) and 95% confidence intervals by study for comparisons of individuals with ADHD to control individuals. Effect size symbols are proportional to study sample size. Effects to the right of zero reflect greater delay discounting (steeper devaluation of future rewards) in the ADHD group compared to the control group. Study subscripts refer to multiple comparisons within the same study; numbers in parentheses refer to the references.
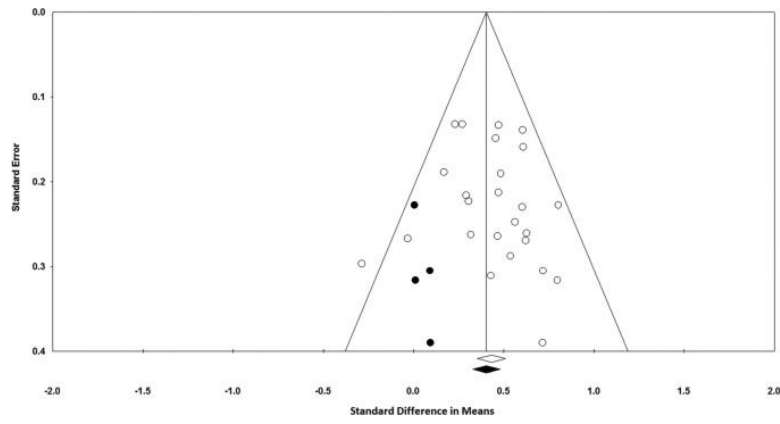
Figure 3.
Funnel plot of the relationship between effect sizes (d) and standard errors (open circles), with imputation of unpublished studies (filled circles) using Duval and Tweedie's trim and fill approach.

Table 1

All case-control effect sizes (*k*=24), listed in order of recency.

Study	Mean Age	Group Ns (ADHD vs. Control)	ADHD Comorbidities	Stimulant Medication Status	Reward Type	Delayed Amount	Temporal Delays	Discounting Index
Mostert et al. 2015a (81)	35.93	109 vs. 123	1 or more comorbidity	76% medicated, no abstinence	Hypothetical	€10	NA	*k*
Mostert et al. 2015b (81)	35.93	133 vs. 132	1 or more comorbidity	76% medicated, no abstinence	Hypothetical	€30	NA	*k*
Mostert et al. 2015c (81)	35.93	133 vs. 132	1 or more comorbidity	76% medicated, no abstinence	Hypothetical	€100	NA	*k*
Onnink et al. 2015 (82)	35.55	107 vs. 109	49% 1+ MDD episodes	60% medicated, no abstinence	Hypothetical	€100	2, 30, 180, 365, and 730 days	*k*
Yu et al. 2015 (83)	8.93	19 vs. 25	None	None	Real	$ 0.10	0, 5, 10, 20, 30, and 60 secs	AUC
Costa Dias et al. 2015 (84)	9.28	36 vs. 54	39% 1+ comorbid diagnosis	42% medicated, abstinence of 5 half-lives (24-48hr)	Hypothetical	$ 10	7, 30, 90, and 180 days	*k*
Castellanos-Ryan et al. 2014 (85)	16.00	41 vs. 1257	27% CD	NA	Hypothetical	$ 55†	7-186 days (23 unique delays)	*k*
Chantiluke et al. 2014 (53)	14.79	31 vs. 18	42% ASD	50% medicated, 48hr abstinence	Hypothetical	£ 100	1 wk, 1 mo, and 1 yr	*k*
Fassbender et al. 2014 (86)	17.99	25 vs. 40	None	None	Real	$ 33	7, 14, 21, 28, 35, 42, 49, and 56 days	*k*
Dai et al. 2013a (32)	33.20	31 vs. 28	Significantly higher rate of MDD and SP	NA	Hypothetical	NZ$ 50	1 and 6 mos, 1, 3, 5, and 10 yrs	AUC
Dai et al. 2013b (32)	33.20	31 vs. 28	Significantly higher rate of MDD and SP	NA	Hypothetical	NZ$ 5000	1 and 6 mos, 1, 3, 5, and 10 yrs	AUC
Costa Dias et al. 2013 (87)	9.34	35 vs. 64	None	Abstinence of 5 half-lives (24-48hr)	Hypothetical	$ 10	1, 7, 30, 90, 180, and 365 days	*k*
Crunelle et al. 2013 (88)	32.98	28 vs. 17	39% cocaine dependence	Medication naive	Hypothetical	NA	5 days, 1 and 3 mos, 1, 3 and 10 yrs	*k*
Demurie et al. 2013 (16)	11.82	72 vs. 130	None	24hr abstinence	Hypothetical	€30	1 and 2 days, 1 and 2 wks	AUC
Scheidel 2013 (89)	26.46	25 vs. 25	None	100% medicated, no abstinence	Hypothetical	$ 1000	1 and 3 days, 1 wk, 6mos, and 5 yrs	AUC
Demurie et al. 2012 (15)	12.30	38 vs. 46	11% ODD/CD	79% medicated, 24hr abstinence	Hypothetical	€30	1 and 2 days, 1 and 2 wks	AUC
Wilbertz et al. 2012 (33)	36.91	28 vs. 28	25% current diagnosis, 32% lifetime	two months abstinence	Hypothetical	€200	1, 3, 9, 24, 60, 120, and 240 mos	*k*

Study	Mean Age	Group Ns (ADHD vs. Control)	ADHD Comorbidities	Stimulant Medication Status	Reward Type	Delayed Amount	Temporal Delays	Discounting Index
Wilson et al. 2011 (18)	7.93	27 vs. 31	26% ODD	30% medicated, 24–48hr abstinence	Hypothetical	$ 10	7, 30, 90, and 180 days	k
Hurst et al. 2011 (17)	20.00	21 vs. 218	None	NA	Hypothetical	$ 1000	1 and 2 wks, 1 and 6 mos, 1, 3, and 10 yrs	AUC
Scheres et al. 2010 (77)	11.81	45 vs. 37	None	Unmedicated	Real	$ 0.10	5, 10, 20, 30 and 60 secs	AUC
Paloyelis et al. 2010 (73)	15.42	36 vs. 32	None	72% medicated, 48hr abstinence	Hypothetical	£ 17 †	1, 2, 7, 14, 30, 60, 120, and 180 days	AUC
Plichta et al. 2009 (54)	23.45	14 vs. 14	None	50% medicated, 96hr abstinence	Real	€60	0, 2, and 4 wks	ICR
Scheres et al. 2006 (90)	11.42	22 vs. 24	14% psychotic or PDD symptoms	81% medicated, 36% abstinent	Real	$ 0.10	0, 5, 10, 20, and 30 secs	AUC
Barkley et al. 2001a (14)	14.73	101 vs. 39	100% ODD	58% medicated, 24hr abstinence	Hypothetical	$ 100	1 mo, 1, 5, and 10 yrs	POI
Barkley et al. 2001b (14)	14.73	101 vs. 39	100% ODD	58% medicated, 24hr abstinence	Hypothetical	$ 1000	1 mo, 1, 5, and 10 yrs	POI

Notes: Hypothetical = hypothetical delay discounting task; NA = not available; k = hyperbolic discounting function; MDD = major depressive disorder; Real = real delay discounting task; secs = seconds; AUC = area under the curve; hr = hour; CD = conduct disorder

† indicates a mean value; ASD = autism spectrum disorder; wk = week; yr = year; SP = Social Phobia; mo = month; ODD = oppositional defiant disorder; ICR = intertemporal choice ratio; PDD = pervasive developmental disorder; POI = points of indifference