The interaction between reinforcement and inhibitory control in ADHD: A review and research guidelines

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HIGHLIGHTS

• Qualitative and quantitative approaches were used to review reinforcement × inhibition interactions in children and adolescents with ADHD and controls.
• Meta-analyses show that reinforcement normalized inhibition in children and adolescents with ADHD to the baseline level of controls.
• Potential factors contributing to the reinforcement effects include the reinforcement schedules and the type of reinforcement.
• Future behavioral and neuroimaging research will help elucidate this interaction further.

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ABSTRACT

The majority of studies which have aimed to identify cognitive and motivational factors at play in ADHD have investigated cognitive-control processes and reinforcement effects in isolation. Notably, in recent years, the interaction between these two processes has been increasingly examined. Here, we aimed to provide a comprehensive and critical review of the behavioral and functional neuroimaging studies that have investigated reinforcement effects on inhibitory control in ADHD. The findings of our meta-analyses show that reinforcement can normalize inhibitory control in children and adolescents with ADHD to the baseline level of controls. Furthermore, the data suggests that inhibitory control may improve to a larger extent in youth with ADHD compared with controls, as a function of reinforcement. Based on (1) this review and meta-analyses, (2) functional neuroimaging studies in healthy populations, and (3) existing ADHD and neurobiological models of dual processes, we propose specific guidelines for future research, which are anticipated to further elucidate processes underlying impulsive behavior associated with ADHD.

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Contents

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1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a common child and adolescent developmental disorder with prevalence rates of 5–10% (Scahill & Schwab-Stone, 2000). ADHD is mainly characterized by age-inappropriately high levels of inattention and/or impulsivity and hyperactivity. Three symptomatic subtypes are currently distinguished: ADHD-inattentive subtype, ADHD-hyperactive/impulsive subtype, and ADHD-combined subtype (American Psychiatric Association, 2013).

Theoretical causal models of ADHD have suggested that deficits in inhibitory control lead to secondary impairments in other cognitive-control functions, resulting in inattention, hyperactivity, and impulsivity (Barkley, 1997). This theory has triggered a burgeoning literature of behavioral and functional magnetic resonance imaging (fMRI) studies. Findings indicate that ADHD is indeed associated with poor cognitive control, particularly inhibitory control as measured with the stop task (Chamberlain et al., 2011; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005; Lipszyc & Schachar, 2010; Logan, Schachar, & Tannock, 1997; Logan & Sergeant, 1998; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Generally, individuals with ADHD have slower stop signal reaction times (SSRT), suggesting that it takes them longer to inhibit prepotent responses. The inhibitory deficit in ADHD is associated with both structural and functional abnormalities in fronto-striatal and fronto-parietal neural circuitries often revealing hypoactivation in prefrontal areas during stop or go/no-go tasks as compared to typical populations (Castellanos & Proal, 2012; Cortese et al., 2012; De La Fuente, Xia, Branch, & Li, 2013; Hart, Radua, Nakao, Mataix-Cols, & Rubia, 2013; Paloyelis, Mehta, Kuntsi, & Asherson, 2007; Valera, Faraone, Murray, & Seidman, 2007; Valera & Seidman, 2005). Despite the established relation between ADHD and cognitive control deficits, meta-analyses have shown that effect sizes are small to moderate: poor inhibitory control is typical of only 50% of all patients with ADHD (Logan & Sergeant, 1998; van Mourik, Oosterlaan, & Sergeant, 2005; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Willcutt et al., 2005).

In addition to impairments in cognitive control, altered reinforcement sensitivity is considered one of the important deficits in ADHD (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Castellanos & Tannock, 2002; Douglas & Parry, 1994; Sonuga-Barke, 2002, 2003, 2005; Sonuga-Barke & Fairchild, 2012; Tripp & Wickens, 2012). Reinforcement sensitivity may be defined as a tendency that varies across individuals and is often measured with questionnaires. As such, it is assumed that higher levels of reward sensitivity will be correlated with larger benefits in task performance when this is reinforced with rewards (Luman, van Meel, Oosterlaan & Geurts, 2012). Importantly, Fosco, Hawk, Rosch, and Bubnik (2015) tested this assumption

and demonstrated that higher questionnaire-based reward sensitivity is indeed associated with larger increases in task performance when it is rewarded compared to when it is not rewarded. Research using questionnaire-based measures of reinforcement sensitivity has demonstrated that indeed, children and adolescents with ADHD are more sensitive to rewards than controls (Fosco et al., 2015; Luman et al., 2012). However, in experimental research, findings are less conclusive: while there is some evidence that the positive effects of reinforcers on task performance are stronger in those with ADHD than controls, psychophysiological research suggests decreased reinforcement sensitivity at that level of analysis in individuals with ADHD (see Luman et al. (2005) for a review).

A more consistent finding of altered reinforcement sensitivity in ADHD is a relatively strong preference for small immediate rewards compared to larger delayed rewards (Antrop et al., 2006; Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001; Bitsakou, Psychogiou, Thompson, & Sonuga-Barke, 2009; Demurie, Roeyers, Baeyens, & Sonuga-Barke, 2012; Luman et al., 2005; Marco et al., 2009; Scheres, Tontsch, Thoeny, & Kaczkurkin, 2010; Sergeant, 2000; Shiels et al., 2009; Solanto et al., 2001; Sonuga-Barke, Taylor, Sembi, & Smith, 1992; Tripp & Alsop, 2001; Wilson, Mitchell, Musser, Schmitt, & Nigg, 2011 but also see Plichta et al., 2009; Scheres et al., 2006; Solanto et al., 2007; Wilbertz et al., 2013). In terms of brain activation during choices between small immediate and larger delayed rewards, only two studies have been published so far (Plichta et al., 2009; Rubia, Halari, Christakou, & Taylor, 2009). Plichta et al. (2009) showed ventral striatal hypoactivation in adults with ADHD while deciding between smaller sooner and larger later reward options. Additionally, they reported hyperactivation of the dorsal caudate nucleus and amygdala in those with ADHD when the soon option was not immediate. These findings suggest reduced neural reward processing in individuals with ADHD, and are consistent with the delay aversion theory (Sonuga-Barke et al., 1992). The second study (Fabiano et al., 2009) found that preferences for small immediate rewards were correlated with hyperactivity symptoms. When contrasting delayed vs. immediate choices, adults with ADHD showed hypoactivation in orbital and inferior prefrontal cortices, putamen, thalamus, inferior parietal lobe, posterior cingulate/precuneus, and cerebellum. Given the involvement of these regions in other processes including temporal processes (e.g. temporal discounting), inhibition, and attention, Rubia, Halari, Christakou et al. (2009) suggested that a combination of such skills and abilities is needed to wait for large delayed rewards, and that these may be compromised in ADHD.

Secondly, a recent meta-analysis (Plichta & Scheres, 2014; but see von Rhein et al., 2015; Plichta & Scheres, 2014) of functional MRI studies has revealed medium-sized hypoactivation of the ventral striatum during anticipation of potential monetary reward in adolescents and adults with ADHD as measured with the monetary incentive delay

(MID) task (Knutson, Westdorp, Kaiser, & Hommer, 2000). In contrast, there are very few fMRI studies on reward outcome in relation to ADHD (Paloyelis, Mehta, Faraone, Asherson, & Kuntsi, 2012; Strohle et al., 2008; Wilbertz et al., 2012). These findings are interesting though mixed regarding the role of ventral striatum and orbitofrontal cortex during reward outcome in individuals (adolescents and adults) with ADHD. More research is needed to determine the relation between ADHD and reward outcome processing at different developmental stages, and studies examining both anticipation and outcome of rewards within the same paradigm and sample are of particular interest (see von Rhein et al., 2015).

Taken together, although ADHD is marked by heterogeneity and multiple mechanisms are involved, there is accumulating evidence supporting the notion of altered reinforcement effects in ADHD. Additionally, a link between ADHD and inhibitory control deficits had already been established. The importance of both processes in ADHD is reflected in the roles both play in behavioral interventions: desired behaviors (including inhibitory control) are often trained by the use of reinforcers (Antshel et al., 2011; Daley et al., 2014; DuPaul & Stoner, 2014; Fabiano et al., 2009). Together, altered reinforcement effects and inhibitory control deficits may form one of the fundamental mechanisms for the diversity of ADHD symptoms.

1.1. Reinforcement and inhibitory control integration

Inhibitory control and reinforcement effects can be studied in isolation as has been done in the majority of the studies described above. However, goal directed behavior often involves aiming to achieve a positive outcome (reward) or avoiding a negative outcome (e.g. punishment) in daily life situations. Therefore, reinforcement may be expected to play a vital role in inhibitory control, requiring adequate integration of these two functions in order to serve appropriate goal directed behavior. For example, a child may be required to pay attention during a school lecture and inhibit the temptation to talk to friends during to get better grades. In this example, reward (getting good grades) increases the likelihood of someone to demonstrate cognitive control (in this case, inhibition of interacting with friends). Thus, one way in which reinforcement can interact with cognitive control is that incentives lead to behavioral improvements (ameliorating effects). Another, much less frequently studied manner in which reinforcement and inhibitory control interact is in the opposite way, i.e., reinforcement may impair inhibitory control. Specifically, stimuli or responses to stimuli that have resulted in a reward or the avoidance of punishment will be harder to avoid or inhibit than (responses to) stimuli that have not been associated with reinforcement. For example, a child may find it more difficult to stop being the class clown if his/her behavior resulted in the approval of peers than if it did not. The impairing effects of reinforcement or motivational significance on cognitive control and its neural correlates have been classified as "hot" forms of cognitive control by Metcalfe and Mischel (1999) and later fine-tuned by Zelazo and Müller (2002). This is in contrast to a "cool" form of cognitive control which is purely abstract, such as measured by the stop task (Logan et al., 1997).

Given that reinforcement plays an important role in inhibitory control in various ways, it is encouraging that the combination of inhibitory control and reinforcement has been increasingly incorporated in theoretical models of multiple causal mechanisms of ADHD (Castellanos & Tannock, 2002; Nigg, 2003; Nigg & Casey, 2005; Nigg et al., 2005; Sagvolden, Johansen, Aase, & Russell, 2005; Sonuga-Barke, Bitsakou, & Thompson, 2010; Sonuga-Barke, 2002, 2003, 2005). Specifically, the dual-pathway model suggests an impaired dorsolateral cortical-striatal brain circuitry to be associated with (cool) cognitive control impairments, whereas dysfunctions in the medial and orbital prefrontal-ventral striatal circuits are linked to altered reinforcement sensitivity. Although earlier versions of this model primarily viewed these pathways as independent, more recently, Sonuga-Barke, Sergeant, Nigg,

and Willcutt (2008) proposed that cognitive control and reinforcement interact, despite the fact that strong preferences for immediate rewards and poor cognitive control are distinguishable to some extent in their relation to ADHD (Solanto et al., 2001). Thus, ADHD has progressively come to be viewed as a multi-systemic disorder with diverse neuropsychological profiles (i.e. altered reinforcement effects, and/or inhibitory/cognitive control deficits) (Nigg & Casey, 2005; Sonuga-Barke et al., 2010; de Zeeuw, Weusten, van Dijk, van Belle, & Durston, 2012).

Nonetheless, empirical studies examining the conjunction of reinforcement and inhibitory control pathways in ADHD are still sparse. Although a number of behavioral studies have examined the ameliorating effects of reinforcement on inhibition, impairing effects of reinforcement on inhibitory control have only been investigated in ADHD in one study (Wodka et al., 2007). Additionally, functional neuroimaging studies on the integration of reinforcement and cognitive control have only just started and are limited to the ameliorating effects of reinforcement on cognitive control. We argue that more research is needed in which the integration between these two processes is examined in individuals with ADHD, because more variance in the symptoms of ADHD may be explained when measuring the conjunction of these two important functions than when measuring each in isolation.

2. Approach

While other executive functions are impaired in ADHD as well, inhibitory control deficits have been shown to most robustly differentiate individuals with ADHD from controls (e.g., Willcutt et al., 2005). For this reason, as well as for the sake of brevity, the focus of this review will be on the integration of inhibitory control and altered reinforcement effects. We will (a) provide a comprehensive qualitative review of behavioral studies in which the interaction between these was measured. We will also report a quantitative meta-analysis, which is based on a subset of these studies. (b) This will be followed by a description of relevant functional neuroimaging studies. (c) We will provide brief descriptions of 3 relevant ADHD models and 3 relevant neurobiological models. For each of these three sections, we will critically discuss the current state of knowledge, and suggest directions for future research. Finally, we will address specificity issues. In particular we will discuss disorder specificity and cognitive control domain (i.e. working memory and sustained attention) specificity.

Although we acknowledge that reinforcement shares some overlap with emotion (Chiew & Braver, 2011), and that it is important to study interactions between emotion and cognitive control in individuals with ADHD, in this review we will primarily focus on the effects of reinforcement, defined as the effects of incentivizing action or inhibition, and its interaction with inhibitory control.

For the purpose of this review, we used the following key words to find potentially relevant articles: ADHD/attention deficit hyperactivity disorder, reinforcement/motivation/reward, inhibition/inhibitory control/stop signal task/go no-go task. These key words were entered into the search engines PubMed, Web of Science and Google Scholar. Studies using non-clinical ADHD samples, animal studies, and non-English articles were excluded. Seventeen studies were included for this review, and met the following criteria:

- Inclusion of individuals with ADHD according to ICD-10, DSM-III-R, DSM-IV, DSM-IV(-TR) or DSM-V and a typical control group
- Use of go/no-go or stop tasks in at least two conditions (monetary reinforcement and control)
- Publication year between 1995 and 2015

We also performed a meta-analysis for a subset ($n = 10$) of the 17 studies, which met the additional inclusion criteria below:

- The control condition did not include monetary reinforcement, or the amounts were less than in the reinforcement condition

Table 1
Overview of empirical studies on reinforcement and inhibitory control in ADHD, not included in the meta-analysis.

Study	Subjects	Age	Task	Reinforcement manipulation	Dependent variables	Type of reward	Interaction effect	Main effects
Liddle et al. (2011)	18 ADHD-C 18 Controls	9–15	Go/no-go	CC no reward: – RC1 low reward: • Correct hit 1 point • Correct inhibition 1 point • False alarm −1 point RC2 high reward: • Correct hit 1 point • Correct inhibition 5 points • False alarm −5 points	• Default mode network deactivation • Overall inhibition rate • D prime (degree of co maximization of speed and inhibition) bias to inhibit (degree to which the balance between go and inhibition shifts to inhibition)	Points (not specified further)	ns	Main effects of group: • D prime controls > ADHD • Misses controls < ADHD Main effects of condition: • Inhibition low < high • D prime low < high • Bias-to-inhibit low < high
Wodka et al. (2007)	59 ADHD 84 Controls	7–16	Go/no-go	CC no reward: no feedback CC2 no reward: cognitively demanding go/no-go (1 back go/no-go) RC1 reward + punish: • Hit +1 point • Hit RT < 350 ms +2 points • False alarms −2 points	• False alarm rate • Miss • Hit RT • RT standard deviation	Points exchangeable for money	ns	Main effects group: no reward: • False alarms ADHD > controls • RT standard deviation ADHD > controls No reward cognitively demanding: • False alarms ADHD > controls Reward + punish: • Miss ADHD > controls • False alarm ADHD > controls • RT standard deviation ADHD > controls Main effects of condition: • False alarms cognitively demanding > no reward • False alarms reward + punish > no reward ns for false alarms cognitively demanding versus reward + punish
Michel et al. (2005)	20 ADHD 20 controls	7–12	Adjusted stop task	CC no reward: • Response speed feedback RC1: • Correct inhibition +15 cents • False alarm −15 cents • Response speed feedback	• SSRT • RT hit	Money	ns	Main effect group: • SSRT ADHD > controls Main effect of condition: • SSRT reward < no reward
Stevens et al. (2002)	76 ADHD 76 Controls	7–12	Stop task	CC no reward: no feedback RC1 reward: • Hit +5ct • Correct inhibition +5ct	• SSRT • RT hit • RT standard deviation • Probability of responding given a signal	Money	ns	Main effects group: • SSRT ADHD > controls • RT standard deviation ADHD > controls • Probability of responding given a signal ADHD > controls Main effects of condition: • SSRT reward > no reward • RT standard deviation reward < no reward • Probability of responding given signal reward >

Table 1 (*continued*)

Study	Subjects	Age	Task	Reinforcement manipulation	Dependent variables	Type of reward	Interaction effect	Main effects
Slusarek et al. (2001)	33 ADHD-C 33 Controls 33 Other diagnoses	6–14	Adjusted stop task	CC no reward: – RC1 low reward + punish: • False alarm −1 point • Correct inhibition +1 point RC2 high reward + punish: • False alarm 50% −1 point 50% −5 points • Correct inhibition +1 point	• SSRT • Probability of inhibition • RT	Points (not exchangeable for external rewards to relate the incentives to internal rather than external motivation)	Significant: SSRT and probability of inhibition in ADHD improved during high reward + punish condition compared to low reward + punish but controls and other diagnoses group did not	no reward Main effects group: • Probability of inhibition ADHD < controls and other diagnoses • SSRT ADHD > controls and other diagnoses Main effects of condition: –
Oosterlaan and Sergeant (1998)	14 ADHD 21 Controls 14 Disruptive 14 Anxious	7–13	Stop task	CC no reward: – RC1 reward: • Correct inhibition +1 point and written feedback "Good" RC1 punish: • False alarms −1 point and written feedback "Wrong"	• SSRT • Probability of inhibition • False alarms • RT • RT standard deviation	Points for presents	ns	Main effects group when comparing ADHD to controls only: • SSRT ADHD > controls • Probability of inhibition ADHD < controls • RT ADHD > controls • RT standard deviation ADHD > controls Main effects of condition: –
Iaboni et al. (1995)	19 ADHD 17 Controls	8–13	Go/no-go	CC no reward: – RC1 reward + punish: • Hit +5 • False alarms −5 RC2 reward + punish: • Miss −5 • Correct inhibition +5 RC3 punish: • Miss −5 • False alarms −5 RC4 reward: • Hit +5 • Correct inhibition +5 In all conditions: Written feedback • "You win" • "You lose" Auditory tone • Correct response high tone • Incorrect low tone	• Omission • False alarms • RT	• Money • Verbal feedback • Auditory tone	ns for inhibition measures significant for response execution: RT during punish < RT during reward in ADHD. In controls, RT during punish = RT during reward	Main effects group: • False alarms ADHD > controls • Hit ADHD > controls Main effects of condition: • Miss rate reward < all other conditions • Hit reward > reward + punish

CC = control condition; RC = reinforcement condition; SSD = stop signal delay; SSRT = stop signal reaction time.

– Studies had to report the means and standard deviations for the SSRT (stop signal tasks), or false alarm rate (go/no-go tasks) and the reaction time (RT) for response execution during go trials, as well as their sample sizes. If the data were not numerically reported, or transformed into a different type of dependent variable, the authors were contacted with a request for numerical details

– Studies reported unique data (not overlapping with samples from other studies).

2.1. Selection of conditions and subgroups

Studies that compared multiple reinforcement conditions to one or multiple control conditions, or vice versa, would lead to non-independent effect sizes when the same sample is used. To avoid non-independent effect sizes we selected the conditions that best matched our study selection criteria. This was done for the study by Kohls, Herpertz-Dahlmann, and Konrad (2009) where we selected the condition using monetary reinforcement over social reinforcement. In the study by Desman, Petermann, and Hampel (2008) the reward only condition was selected from the three types of reinforcement conditions. Finally, one study compared two groups of ADHD subtypes to a control group (Huang-Pollock, Mikami, Pfiffner, & McBurnett, 2007). We collapsed the data from the two ADHD subgroups. This was done because the other studies included in our meta-analysis did not distinguish or select a specific subgroup, and because ADHD subtypes are not stable across the lifespan (APA, 2013). Based on the above-mentioned inclusion criteria, we identified 10 studies eligible for the meta-analysis. An additional 7 studies also investigated reinforcement effects on response inhibition but could not be included in the meta-analysis (see Table 1). Five of these studies did not report the required data to compute the necessary effect sizes and/or the study authors did not respond to our request for data, or no contact information was available. Two of these studies could not be included due to the lack of a control condition (Oosterlaan & Sergeant, 1998) or by aiming to hamper inhibition (Wodka et al., 2007). Therefore, we will first report on the methodological details of all 17 studies, followed by a qualitative summary and discussion of the findings. Additionally, we will report and discuss findings of a meta analysis of studies that met the inclusion criteria (see Table 2).

2.2. Data analysis

In order to determine whether the effect of reward on inhibition differed across groups, we computed Cohen's d_{av} effect size for within subjects effects (Lakens, 2013) for each study and each group separately. Cohen's d_{av} was calculated by dividing the mean difference of the control and reinforcement conditions by the mean standard deviation of the control and reinforcement conditions. Secondly, we also focused on the between group difference, because it gave insight into ADHD-control differences in inhibitory control without and with reinforcement. To this end, we calculated Cohen's d effect size for between subjects effects (Cohen, 1988) for each study and each condition separately. Cohen's d was calculated by dividing the mean difference between ADHD and control groups by the pooled standard deviation.

Based on the effect sizes and sample sizes, the weighted mean effect sizes were calculated. For the within subjects effect of reward, these were calculated for each group separately. For the between subjects effect (i.e., group difference in inhibition), these were calculated for control conditions and reinforcement conditions separately. The weighted mean effect size (Hedge's g) and the 95% confidence interval were computed according to the random effects procedure described in Hedges and Olkin (1985). In addition, Q and I^2 were calculated. Q reflects weighted sums-of-squares to quantify the variability that is due to heterogeneity of study findings. I^2 is a similar statistic but adjusts for the small number of studies and small sample sizes within studies. When influential outliers were detected, results were reported with and without the influential outlier (Viechtbauer & Cheung, 2010). There were two influential outliers (Sinopoli, Schachar, & Dennis, 2011; Uebel et al., 2010) that greatly altered the Hedge's g effect sizes.

3. Comprehensive review: behavioral ADHD studies of reinforcement effects on inhibitory control

3.1. A comparison of methodological details

All studies in this review included non-medicated, clinically diagnosed children and adolescents with ADHD and healthy control groups in the age range of 6–18 years. To our knowledge, there were no studies with adult ADHD groups. The main dependent variables for response inhibition are SSRT for the stop task, and false alarm rate (proportion of no-go trials to which a participant responded) for the go/no-go task. As for response execution, mean reaction time (RT) and RT variability are the main measures for both tasks. Reinforcement manipulations can be segregated into reinforcement of inhibitory control, i.e., behavior on no-go/stop trials (false alarms or correct inhibition) and reinforcement of response execution, i.e., behavior on go trials (omissions or hits). Frequently, studies aimed at improving inhibitory control by reinforcing correct inhibition on no-go/stop trials (e.g. Groom et al., 2010; Kohls et al., 2009; Rosch et al., 2015; Scheres, Oosterlaan, & Sergeant, 2001; Sinopoli et al., 2011; Uebel et al., 2010). In most of these studies, the reinforcement condition consisted of a combination of rewarding correct inhibition and punishing false alarms (Desman et al., 2008; Groom et al., 2010; Iaboni, Douglas, & Baker, 1995; Liddle et al., 2011; Michel, Kerns, & Mateer, 2005; Shanahan, Pennington, & Willcutt, 2008; Slusarek, Velling, Bunk, & Eggers, 2001; Uebel et al., 2010; Wodka et al., 2007). Ten studies included rewarding hits on go trials in addition to reinforcing inhibitory control, in order to maintain the pre-potency of response execution, an essential characteristic of these tasks (Desman et al., 2008; Groom et al., 2010; Huang-Pollock et al., 2007; Iaboni et al., 1995; Rosch et al., 2015; Shanahan et al., 2008; Slusarek et al., 2001; Stevens, Quittner, Zuckerman, & Moore, 2002; Uebel et al., 2010; Wodka et al., 2007). In one study, the researchers explicitly aimed at rewarding hits more strongly than correct inhibitions, in order to evoke impulsivity. They hypothesized that this would have a detrimental effect on inhibitory control, especially in the ADHD group (Wodka et al., 2007).

Not only did studies differ in the behavior that was reinforced as described above, but also other task aspects differed. The design of four studies enabled measurement of the unique effects of reward versus punishment (Desman et al., 2008; Iaboni et al., 1995; Groom et al., 2010; Oosterlaan & Sergeant, 1998). Four studies compared the effects of high versus low reward (Groom et al., 2010; Huang-Pollock et al., 2007; Liddle et al., 2011; Slusarek et al., 2001) on inhibition. While most studies used tangible incentives (money amounts, points exchangeable for money or gifts), two studies used points only (Liddle et al., 2011; Slusarek et al., 2001). In four studies, the monetary rewards were combined with written feedback, which appeared on screen (Iaboni et al., 1995; Konrad, Gauggel, Manz, & Schöll, 2000; Oosterlaan & Sergeant, 1998) or positive verbal feedback by the experimenter (i.e., social feedback) (Scheres et al., 2001). Finally, in one study, the effects of monetary rewards on inhibition were directly compared with the effects of social rewards, namely pictures of exuberant facial expressions (Kohls et al., 2009).

Additionally, the control conditions to which the reinforcement condition was compared differed across studies. For example, while most studies did not use any feedback in the control condition, one study reported the use of informative written feedback ("good work") after correct inhibition in the control condition of the task (Sinopoli et al., 2011). Another used an informative-feedback control condition in addition to a completely neutral control condition in which no feedback was provided (Desman et al., 2008). Finally, two studies

Table 2
Overview of empirical studies included in meta-analyses.

Study	Subjects	Age	Task	Reinforcement manipulation	Dependent variables	Type of reward	Interaction effect	Main effects
Rosch et al. (2015)	26 ADHD 33 Controls	9–12	Stop task	CC no reward: uninformative feedback RC1 reward: • Hit fast +5 points • Hit slow +2 points • Correct inhibition preceded by fast go response +15 points • Correct inhibition preceded by slow go response +6 points	• SSRT • Percent inhibition • Percent accuracy • RT hit • RT variability • Mean SSD	Points for gifts	Significant: During CC ADHD slower SSRT compared to controls but during RC the ADHD group improved more in SSRTs than controls, thereby reaching performance level of controls during CC.	Main effects of group: • SSRT ADHD > controls • percent inhibition ADHD < controls • RT ADHD > controls • SSRT no reward > reward Main effects of condition: SSRT no reward > reward
Sinopoli et al. (2011)	19 ADHD 9 secondary ADHD 40 TBI 44 Control	7–17	Task 1: stop task Task 2: stop task with fixed SSD = 0 (simultaneous with go stimulus onset)	CC no reward: • Correct inhibition written feedback "good work!" RC1 low reward: • Correct inhibition +2 points RC2 high reward: • Correct inhibition +10 points	• Percent inhibition • SSRT • RT • RT variability • Hits	Points for gifts or money	ns	Main effects of group: • SSRT ADHD > controls • RT SD ADHD > controls • Hit rate ADHD < controls Main effects of condition: Task 1: • SSRT no reward > low reward > high reward Task 2: • SSRT no reward > low reward and high reward
Groom et al. (2010)	28 ADHD 28 Controls	9–15	Go/no-go	CC no reward: – RC1 low reward: • Correct hit 1 point • Miss −1 point • Correct inhibition 1 point • False alarm −1 point RC2 high reward: • Correct hit 1 point • Miss −1 point • Correct inhibition 5 point RC3 high punish • Correct hit 1 point • Miss −1 point • False alarm −5 point	• RT [cap] – floor differential (staircase tracked response deadline on go trials – initial response deadline based on practice trials) • N2 (ERP measure) • P3 (ERP measure)	Points (not specified further)	ns	Main effects of group: • P3 controls >ADHD • Main effects of condition: • RT [cap] – floor differential • Low > high reward and response cost • P3 low > high reward and response cost • N2 high reward >response cost
Uebel et al. (2010)	205 ADHD 53 Controls 173 unaffected siblings	6–18	Slow event rate go/no-go	CC no reward: no feedback RC1 reward + punish: • False alarm −5 points • Correct inhibition +1 point • Hit +1 point • Miss −1 point	• RT • RT variability • Miss rate • False alarm rate	Points for small prizes	Significant: • RT reward + punish < no feedback for ADHD group only • Miss rate reward + punish < no feedback, this effect was largest in the ADHD group	Main effects of group: • RT variability controls < ADHD • false alarm rate controls < ADHD Main effects of condition: • RT variability reward + punish < no feedback • Miss rate reward + punish < no feedback
Kohls et al. (2009)	16 ADHD 16 Controls	8–13	Go/no-go	CC no reward: Uninformative feedback (mosaic picture after no-go stimuli) RC1 social: • Correct inhibition happy face picture • False alarm neutral face picture RC2 monetary: • Correct inhibition wallet 50ct picture • False alarm empty wallet picture	• False alarm rate • RT hit • RT false alarm	• Pictures of happy/neutral faces • Money (each child was told to have won 3 euros)	Significant: • False alarm rate decreased during social reward versus no reward. This effect was stronger in ADHD compared to HC • RT hits during monetary reward was slower in ADHD group but faster in HC	Main effects of group: – Main effects of condition: • False alarm rate social and monetary < no reward

Desman et al. (2008)	Study1: 19 ADHD-C 19 Controls Study2: 6 ADHD-I 6 ADHD-C 6 Controls	8–12	Go/no-go	CC no reward: no feedback RC1 auditory: • Hit high sound • Correct inhibition high sound • Miss low sound • False alarm low sound RC2 reward: • Hit +5ct • Correct inhibition +5ct RC3 punish: • Miss −5ct • False alarm −5ct RC4 reward + punish: • Hit +5ct • Correct inhibition +5ct • Miss −5ct • False alarm −5ct	• False alarm rate • Miss rate • RT hit • Heart rate • Skin conductance response (study1)	• Sound • Money	Study 1: ns Study 2: ns	Study 1: Main effects of group: • False alarm rate ADHD > controls • Miss rate ADHD > controls Main effects of condition: • False alarm rate no reward > auditory, reward, punish, reward + punish • HR no reward, auditory, punish < reward, reward + punish • Skin conductance response no reward < reward, punish, reward + punish Study 2: Main effects of group: • False alarm rate ADHD-C > ADHD-I and controls • RT ADHD-I > controls and ADHD-C • RT standard deviation ADHD-I > controls and ADHD-C • Main effects of condition: • Miss rate no reward > reward
Shanahan et al. (2008)	25 ADHD 30 Controls	8–18	Stop task	CC no reward: no feedback RC1 reward + punish: • Hit +10ct • Miss rate −10ct • Correct inhibition +25ct • False alarm −25ct	• SSRT • RT • RT standard deviation • Errors	Money (each child was told to have won $20)	ns for inhibition measures significant group × incentive interaction on RT: controls were slower during reward + punish compared to no reward. For ADHD, RT was equal between conditions.	Main effects of group: • SSRT ADHD > controls • RT standard deviation ADHD > controls • False alarms ADHD > controls Main effects of condition: –
Huang-Pollock et al. (2007)	33 ADHD-I 23 ADHD-C 36 Controls	7–12	Stop task	CC no reward: – RC1 low reward: • Hit = 1point • Correct inhibition = 2 point RC2 high reward: • Hit = 1 point • Correct inhibition = 10 points	• RT hits • RT standard deviation • Error • Miss • False alarms • SSRT	Points for toys	Significant three way interaction of order × reward × group: In control and ADHD-C group false alarms decreased during high reward and this effect was larger when the high reward condition came after low reward. For ADHD-I false alarms decreased only when high reward was presented after the low reward condition. When high reward was first, the ADHD group showed increased false alarms in the low reward condition.	Main effects group: • SSRT ADHD > controls (ADHD-I > controls and ADHD-C). Main effects of condition: • RT high reward < low reward • False alarms high reward < low reward • Miss rate high reward > low reward
Scheres et al. (2001)	24 ADHD 27ADHD + ODD/CD 21ODD + CD 41 Controls	7–12	Stop task	CC no reward: No feedback RC1 reward: • Correct inhibition +100 points and verbal feedback by experimenter "good!"	• SSRT • % correct inhibition • RT • RT standard deviation • Hit rate	Points for presents	ns for inhibition measures significant interaction on response execution: RT reward > no reward. This effect was stronger in ADHD + ODD/CD compared to controls. Trend towards same effect in pure ADHD group but not ODD/CD	Main effects group: • % Correct inhibition ODD/CD and ADHD + ODD/CD> controls • RT ADHD > controls • RT standard deviation ADHD > controls Main effects of condition: • SSRT no reward > reward • RT no reward < reward • Hit rate no reward < reward
Konrad et al. (2000)	21 ADHD 27 TBI 16 Controls	8–12	Stop task	CC no reward: No feedback RC1 reward: • Correct inhibition +1 point and positive verbal feedback	• SSRT • RT	Points for toys/sweets	Significant: During no reward, ADHD slower SSRT compared to controls but during the reward condition both groups show equal SSRTs	Main effects group: SSRT ADHD > controls Main effects of condition: • SSRT no reward > reward

CC = control condition; RC = reinforcement condition; SSD = stop signal delay; SSRT = stop signal reaction time.

used uninformative feedback in the control condition (e.g., abstract figure) presented after both successful and unsuccessful no-go trials (Kohls et al., 2009; Rosch et al., 2015). One study only used a control condition in which only response speed feedback was provided (Michel et al., 2005). Similarly, one study added a cognitively more demanding inhibition task as an extra control condition to address difficulty effects (Wodka et al., 2007). Thus, important to note is that the studies reviewed here differ in the conditions that are compared to one another, and that the majority of studies failed to control for factors such as difficulty, feedback or salience.

3.2. Qualitative review and discussion of the findings

In terms of main effects of reinforcement manipulations, we can conclude that (1a) Reinforcement conditions led to improvement on at least one of the measures for task performance in 14 out of 16 studies (88%) independent of group. This main effect of reinforcement was not detected in the studies by Shanahan et al. (2008) and Slusarek et al. (2001). The study by Oosterlaan and Sergeant (1998) was not included in this comparison as they only compared rewarding correct inhibition to punishing false alarms. (1b) Reinforcing response inhibition, as compared to control conditions, led to an improvement in inhibitory performance on stop/no-go trials in 10 out of 15 studies (67%; Desman et al., 2008; Huang-Pollock et al., 2007; Kohls et al., 2009; Konrad et al., 2000; Liddle et al., 2011; Michel et al., 2005; Rosch et al., 2015; Scheres et al., 2001; Sinopoli et al., 2011; Stevens et al., 2002). In addition to Oosterlaan and Sergeant (1998), Wodka et al. (2007) was not included in this comparison as they mainly aimed to reinforce go performance. (1c) Reinforcing inhibition while not reinforcing response execution led to (strategic) slowing of RT on go trials in one of the two studies using such an approach (Scheres et al., 2001), and not in the study by Konrad et al. (2000); (1d) Reinforcing response execution (rewarding hits or punishing omissions) led to an improvement in performance on go trials in 7 out of 9 studies (78%): faster RT (Groom et al., 2010; Huang-Pollock et al., 2007; Uebel et al., 2010), lower RT variability (Stevens et al., 2002; Uebel et al., 2010), or fewer omissions (Desman et al., 2008; Iaboni et al., 1995; Uebel et al., 2010, although one study reported the opposite effect Huang-Pollock et al., 2007).

With regard to group differences it was observed that (2a) Individuals with ADHD showed poor inhibitory control relative to healthy controls in 14 out of 17 studies (82%), irrespective of reinforcement manipulation (Desman et al., 2008; Huang-Pollock et al., 2007; Iaboni et al., 1995; Konrad et al., 2000; Liddle et al., 2011; Michel et al., 2005; Oosterlaan & Sergeant, 1998; Rosch et al., 2015; Shanahan et al., 2008; Sinopoli et al., 2011; Slusarek et al., 2001; Stevens et al., 2002; Wodka et al., 2007; Uebel et al., 2010). Four did not find main group effects for inhibition (Groom et al., 2010; Kohls et al., 2009; Konrad et al., 2000; Scheres et al., 2001). (2b) Regardless of reinforcement manipulation, response execution was affected in ADHD in 10 out of 17 studies (59%): in particular, higher RT variability was reported in 6 studies out of 15 (33%) (Rosch et al., 2015; Shanahan et al., 2008; Sinopoli et al., 2011; Stevens et al., 2002; Uebel et al., 2010; Wodka et al., 2007), consistent with previous findings (e.g. Castellanos et al., 2005). In some cases slower RTs were reported (Michel et al., 2005; Oosterlaan & Sergeant, 1998; Rosch et al., 2015; Scheres et al., 2001). More omissions were reported in 4 studies (24%; Desman et al., 2008; Liddle et al., 2011; Sinopoli et al., 2011; Wodka et al., 2007), but see Iaboni et al. (1995) for an exception.

(3) Contrary to our expectations, significant interactions between reinforcement condition and group for inhibition measures (SSRT or false alarms) were found in a minority of studies ($n = 4$; 24%; Kohls et al., 2009; Konrad et al., 2000; Rosch et al., 2015; Slusarek et al., 2001). This suggests that, based on statistical significance, there was no strong evidence for reinforcement manipulations to affect the performance of individuals with ADHD differentially from healthy controls.

One potential explanation is the widely acknowledged heterogeneity of symptoms in ADHD. For example, for the interaction between inhibition and reinforcement to be altered, both might need to be inflicted in the same individuals. Indeed, these systems can also operate quite independently (Robbins, Gillan, Smith, de Wit, & Ersche, 2012). Another general explanation is the fact that interaction effects require relatively strong power (i.e., larger sample sizes) to be detected. Although a significant interaction was shown in only a minority of studies, the direction of this interaction was consistent. Therefore, it is unlikely that these were chance findings, and we will focus on examining potential factors that may have contributed to this interaction effect.

Two factors that turned out not to distinguish between the four studies that did report an interaction and the remaining studies are power and room for improvement. First, we examined whether these four studies had more power to detect interactions, either because of relatively large group sizes, or by relatively large number of trials per condition. Group size is not a likely candidate, since these varied between 16 and 33 for the 4 studies that did find and interaction, while for the remaining studies group sizes varied between 9 and 84. As for number of trials per condition, the number of stop/no-go trials varied between 24 and 60 in the 4 studies. For the other studies, the number of trials per condition was comparable: between 20 and 54. A second possibility is that inhibition deficits in the ADHD groups during the control conditions were present in these 4 studies. In other words, significant interactions could be dependent on the presence of room for improvement in the ADHD group (Fosco et al., 2015). While Kohls et al. (2009) reported a lack of group difference for false alarm rate during the control condition and still found a significant interaction, the other three studies reporting significant interactions (75%) all demonstrated inhibition deficits in the ADHD group (suggesting room for improvement). Out of the 13 remaining studies, there was room for improvement in the ADHD group in 11 studies (85%) (Desman et al., 2008; Huang-Pollock et al., 2007; Iaboni et al., 1995; Liddle et al., 2011; Michel et al., 2005; Oosterlaan & Sergeant, 1998; Shanahan et al., 2008; Sinopoli et al., 2011; Stevens et al., 2002; Uebel et al., 2010; Wodka et al., 2007). Thus, it is unlikely that the significant interactions as reported in these 4 studies were driven by relatively large power, or by relatively large room for improvement in the ADHD groups.

Two factors that may have contributed to the significant interactions include contingency management and nature of the reinforcer. As for contingency management, most studies that reported significant interactions reinforced inhibitory control, while not reinforcing response execution (Konrad et al. (2000), Kohls et al. (2009), and Slusarek et al. (2001) with the exception of Rosch et al. (2015)). Conversely, the majority of studies that did not report a significant interaction reinforced both inhibitory control and response execution (Desman et al., 2008; Groom et al., 2010; Huang-Pollock et al., 2007; Iaboni et al., 1995; Liddle et al., 2011; Shanahan et al., 2008; Slusarek et al., 2001; Stevens et al., 2002; Uebel et al., 2010; Wodka et al., 2007). It is possible, yet speculative, that by selectively reinforcing inhibition, a bias towards inhibition was created and participants did not need to make trade-offs between inhibiting and executing responses. As a result, participants may have developed a (strategic) style in which they favored inhibition over execution. In order to maximize reward, this strategic adaptation might have been applied to a larger extent by the ADHD group than controls, leading to an interaction effect. Interestingly, the group by reinforcement interaction effect for RT as reported by Scheres et al. (2010) and Kohls et al. (2009) may be a reflection of such a "waiting for the stop signal" strategy. In this context, the study by Rosch et al. (2015) was the only one reporting a significant interaction while also reinforcing both stopping and going. Clearly, the reported interaction was not a result of strategic slowing in the ADHD group, as all participants responded faster in the reinforcement condition than in the no reinforcement condition. Possibly, the very carefully designed reinforcement manipulation turned out to be optimal for individuals with ADHD:

slow and fast hits were rewarded with 2 and 5 cents respectively, while correct inhibition preceded by slow and fast go responses were rewarded with 6 and 15 points respectively. This is the only study in which (a) reward magnitude varies as a function of preceding response prepotency, and (b) there is one amount (15 cents) that stands out compared to the other amounts. It is possible that this design is what has worked particularly well for those with ADHD. For example, Tripp and Wickens (2008) suggested that individuals with ADHD show increased effects to unexpected rewards compared with controls. The 15 cents in this design could be viewed as unexpected, because it is much larger than the other amounts. Therefore, more research is needed in which the effects of different reinforcement schedules on inhibition are directly compared with one another.

As for nature of the reinforcer, two out of the four studies that reported a significant interaction used non-tangible reinforcers (Kohls et al., 2009; Scheres et al., 2001). This is contrary to the majority of studies in which the points earned were exchangeable for tangible rewards (e.g. candy, money, or toys). In the study by Slusarek et al. (2001), the points were not representative of any external reward. Interestingly, the study by Kohls et al. (2009) also used alternative (non-tangible) reinforcers as they included one condition with social rewards (pictures of happy faces) in addition to a condition with monetary rewards. Unexpectedly, they found that in comparison to the control condition in which uninformative feedback (mosaic pictures after successful and unsuccessful no-go trials) were used, social rewards led to a significantly larger improvement of inhibition in the ADHD group than in the healthy control group, while monetary reward did not. Other research has also suggested that social rewards lead to greater improvement in interference control (Geurts, Luman, & Van Meel, 2008) and memory performance (Krauel et al., 2007) in individuals with ADHD than healthy controls. Therefore, it is an important question for future research to examine further whether inhibitory control may be ameliorated more easily in individuals with ADHD by using non-tangible forms of reinforcement, specifically social rewards such as happy faces (but also see Demurie, Roeyers, Baeyens, & Sonuga-Barke, 2011).

Similarly, only one out of the four studies that reported a significant interaction (Slusarek et al., 2001) used a combination of rewarding correct inhibition and punishing false alarms, while the remaining three studies only rewarded successful inhibition. Therefore, it is possible yet speculative that reinforcement effects on inhibitory control may be especially strong in individuals with ADHD when rewards are used in the absence of punishments. Future research is needed in order to address this possibility.

Finally, based on these studies, we identified 3 other factors that may potentially be relevant for future studies on the interaction between reinforcement and inhibition in ADHD: (1) order effects; (2) the type of control condition that is used; and (3) ameliorating versus impairing effects of reinforcement on inhibitory control. (1) Although order effects may not have been examined in all experiments, or they were examined but did not exist (e.g., Rosch et al., 2015), two studies did report reinforcement by order interactions, some of which interacted with group (Huang-Pollock et al., 2007; Slusarek et al., 2001). Huang-Pollock et al. (2007) demonstrated that in contrast to healthy controls and individuals with ADHD-combined type, those with ADHD-inattentive type had higher false alarm rates in the second block specifically when low followed high reward (Huang-Pollock et al., 2007). This suggests that participants of the inattentive subtype may have perceived the low reward condition as demotivating when it followed the high reward condition. In the study by Shanahan et al. (2008), participants performed more poorly (slower RT, more errors on go trials, and slower SSRTs) in the reinforcement condition compared to the control condition, but this effect only showed when the reinforcement condition followed the control condition. Together, these findings indicate a context dependency of reinforcement effects that may affect the ADHD group more strongly than the healthy controls. Therefore, future research needs to not only counterbalance conditions, but also analyze order effects in order to obtain more insight into the role of context on reinforcement by inhibitory control interactions.

(2) In the majority of studies reviewed here, reinforcement conditions were contrasted with neutral control conditions in which no feedback at all was used. One limitation of neutral control conditions is that it remains unclear to what extent differences between reinforcement and control conditions can be attributed to the reinforcement per se, or to other related aspects of the reinforcement such as salience, level of information about the accuracy of the response, or difficulty level. To address this, Desman et al. (2008) added an extra control condition in which informative auditory feedback was provided. They found the effects of auditory feedback to be comparable to the reinforced conditions. This suggests that the effect of the reinforcement condition may be due to the informative feedback aspect of the reinforcement rather than due to the reward itself (but see Desman et al., 2008). In sum, we encourage the addition of extra control conditions such as described here as a very useful tool that can aid in the interpretation of reinforcement effects on inhibitory control, and task performance in general.

(3) The main methods employed to investigate the interaction between reinforcement and inhibitory control in ADHD has been by delivering reward after successful inhibition with the goal of ameliorating inhibitory control. However, studies using a design which assesses impairing effects of reinforcement on inhibitory control are lacking, despite important theoretical contributions in the area of cognitive control-reinforcement interactions (Barkley, 1997; Castellanos et al., 2006; Sergeant, 2005; Sergeant, Geurts, & Oosterlaan, 2002; Sinopoli et al., 2011), and in spite of compelling models that show that reward pathways have bottom-up effects on cognitive control (Casey, Jones, & Hare, 2008; Haber, 2003; Sonuga-Barke et al., 2008). Wodka et al. (2007) are the only ones so far who used a go/no-go task, which aimed at creating a bias towards response execution by rewarding hits with 1 point, and fast hits (<350 ms) with 2 points, and by not reinforcing correct inhibition. False alarms, which occur relatively infrequently, resulted in the loss of 2 points. The idea was that participants would emphasize response execution because of the extra incentive for responding fast, and because of the 3:1 ratio of go versus no-go trials. Therefore, this study was unique in that it hypothesized that inhibition would deteriorate in the reinforcement condition, especially in individuals with ADHD, due to the emphasis on/bias to response execution. As hypothesized, children committed more false alarms in the reinforcement condition than the neutral condition, but no significant group by condition interaction was found. Potentially, future studies with different reinforcement schedules such as including larger reward magnitudes, will. We feel that this sort of design is still a very useful one to pursue because of high ecological validity (see also Padmala & Pessoa, 2010): in daily life, individuals with ADHD typically need to inhibit responses that lead to positive outcomes (e.g., resisting the temptation to go and play, in favor of doing homework).

3.3. Quantitative review of a subset of studies and discussion of the findings

Ten studies were eligible for the meta-analysis (Table 2). There were two highly influential outliers (Uebel et al., 2010 and Sinopoli et al., 2011), which enlarged the overall effect sizes. Results are therefore presented both with and without these two studies. When excluding the influential outliers the total sample size was 484 (ADHD $n = 210$; controls $n = 274$, age-range was 7–18 years). For the effect of reinforcement within groups, the meta-analysis demonstrated that reinforcement improved response inhibition in both groups. The weighted mean effect size was medium in the control group ($g = .36$, 95% CI [0.31, 0.40]), and large in the ADHD group ($g = .52$, 95% CI [0.42, 0.60]). The results also show that the reinforcement-induced improvement was larger for the ADHD group than the control group (.52 versus .36; see Table 3 for weights and effect sizes per study). Heterogeneity was small for both the control group ($Q = 1.55$) and the ADHD group ($Q = 5.66$). For both groups, I^2 fell below zero, which translates to an

Table 3
Effect sizes

Study	Weight %	Cohen's d_{av} controls	Cohen's d_{av} ADHD
Rosch et al. (2015)	13.75	0.97	0.89
Groom et al. (2010)	11.89	0.37	0.61
Kohls et al. (2009)	7.46	1.23	1.92
Desman et al. (2008)	8.86	0.27	0.14
Shanahan et al. (2008)	12.82	−0.35	−0.20
Huang-Pollock et al. (2007)	21.45	0.19	0.10
Scheres et al. (2001)	15.15	0.19	0.23
Konrad et al. (2000)	8.62	0.32	0.86

Effect sizes and weights for each study, excluding influential outliers.
CC = control condition; RC = reinforcement condition; SSRT = stop signal reaction time;
FA = false alarm rate; Cohen's dav indicates the within subjects effect size.

I^2 value of 0% (Higgins, Thompson, Deeks, & Altman, 2003). When including the influential outliers, the total sample size was 724 (ADHD $n = 353$; controls $n = 371$; age range 6–18 years). The age range was 7 to 18 years. The weighted mean effect size was large in the control group ($g = 1.01$, 95% CI [0.55, 1.47]), and large in the ADHD group ($g = 1.36$, 95% CI [−0.47, 3.18]). Heterogeneity was large for both the control group ($Q = 42.70$, $I^2 = 78.92$) and the ADHD group ($Q = 50.12$, $I^2 = 82.04$).

In line with previous meta-analyses (e.g., Willcutt et al., 2005) and inhibition deficit models of ADHD (e.g., Barkley, 1997), the between-subjects meta-analysis excluding the two outliers demonstrated that the weighted mean effect size of the group difference in the control condition was large ($g = 0.52$, 95% CI [0.48, 0.57]; Fig. 1A): individuals with ADHD performed more poorly than controls in terms of inhibitory control. In reinforcement conditions, the group difference was similar ($g = .49$, 95% CI [0.45, 0.53]; Fig. 1B). This indicates that in reinforcement conditions, the ADHD group did not reach the same inhibition performance as the control group reached during reinforcement. Heterogeneity assessment indicated low heterogeneity in the control condition ($Q = 5.46$) and in the reinforcement condition ($Q = 1.77$). For both conditions, I^2 fell below zero, indicating strong homogeneity of the study findings. When including the influential outliers between group differences in the control condition was large and heterogeneity high ($g = 1.33$, 95% CI [1.31, 1.36], $Q = 27.12$, $I^2 = 66.81$). For the reinforcement condition group differences were

also large but slightly decreased ($g = 1.04$, 95% CI [0.62, 1.47], $Q = 41.12$, $I^2 = 78.17$).

Finally, when excluding the two outliers and comparing inhibitory control of the ADHD group during reinforcement with inhibitory control of the control group during control conditions, the confidence intervals contained zero, indicating that the group differences were no longer significant ($g = 0.08$, 95% CI [−0.03, 0.19], $Q = 0.05$, $I^2 < 0$; Fig. 1C). This shows that during reinforcement, individuals with ADHD normalized to the "baseline" level of controls. This finding was similar when including the influential outliers).

In summary, when observing studies separately there was no consistent evidence for reinforcement manipulations to affect the performance of individuals with ADHD differentially from controls. However, when quantifying the studies in meta-analyses a pattern emerged indicating that reinforcement ameliorates response inhibition in individuals with ADHD more than in controls and may normalize their performance to the baseline level of controls. Given the very low heterogeneity of the meta-analyses when excluding two influential outliers, it is not likely that systematic differences between studies account for heterogeneity. Most studies used a similar age range and IQ thresholds. Taken together, these findings endorse the notion that incorporating reinforcement schedules in ADHD treatment is effective (e.g., Fabiano et al., 2009), and is commonly recommended in combination with medication (e.g., Fabiano et al., 2007).

Based on the above overview, we suggest that future research focuses on the following questions: "Under which circumstances does reinforcement improve inhibitory control to a larger extent in individuals with ADHD relative to controls?", and "Which individual factors contribute to these effects?". As promising factors to focus on, we suggest varying contingency managements (e.g., reward magnitudes), and using various types of rewards in addition to monetary ones, especially social rewards. Furthermore, we recommend that order effects are carefully considered, and that additional control conditions (such as feedback but no reward) are included. Additionally, studying detrimental effects of reinforcement on inhibitory control may increase ecological validity in certain respects and might capture more of the heterogeneous behaviors as observed in those with ADHD. Finally, adding a separate baseline assessment to the typical design of comparing reinforcement with neutral conditions has proven to be useful too: Fosco et al. (2015) demonstrated that the significant condition (reinforcement/neutral) by group interaction was not simply due to
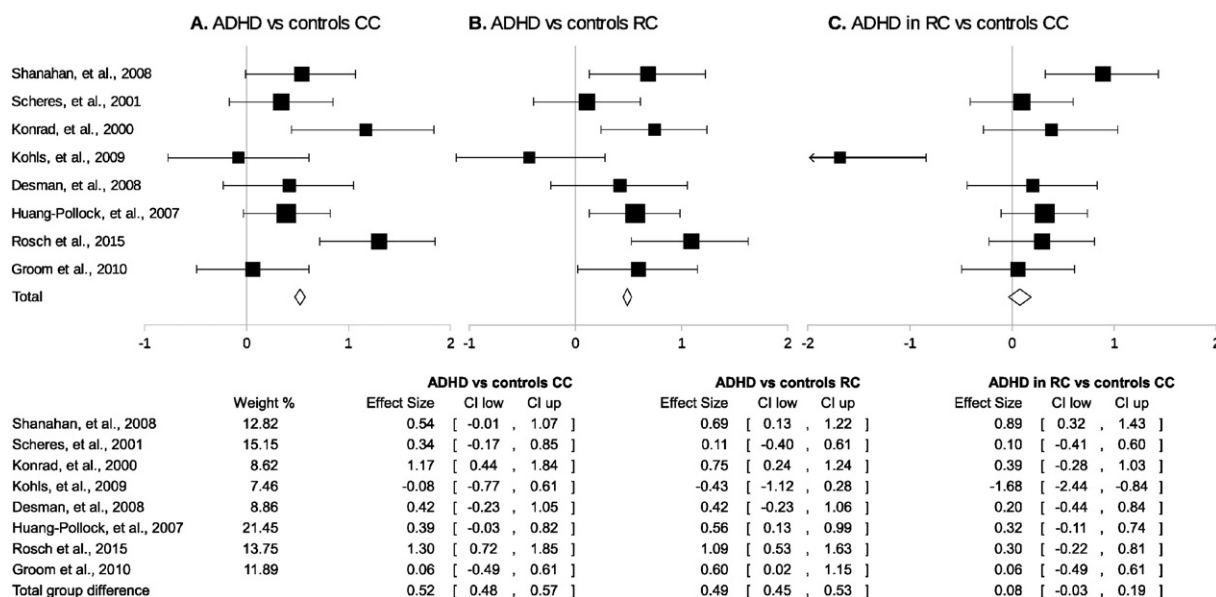

		ADHD vs controls CC			ADHD vs controls RC			ADHD in RC vs controls CC		
	Weight %	Effect Size	CI low	CI up	Effect Size	CI low	CI up	Effect Size	CI low	CI up
Shanahan, et al., 2008	12.82	0.54	[−0.01 , 1.07]		0.69	[0.13 , 1.22]		0.89	[0.32 , 1.43]	
Scheres, et al., 2001	15.15	0.34	[−0.17 , 0.85]		0.11	[−0.40 , 0.61]		0.10	[−0.41 , 0.60]	
Konrad, et al., 2000	8.62	1.17	[0.44 , 1.84]		0.75	[0.24 , 1.24]		0.39	[−0.28 , 1.03]	
Kohls, et al., 2009	7.46	−0.08	[−0.77 , 0.61]		−0.43	[−1.12 , 0.28]		−1.68	[−2.44 , −0.84]	
Desman, et al., 2008	8.86	0.42	[−0.23 , 1.05]		0.42	[−0.23 , 1.06]		0.20	[−0.44 , 0.84]	
Huang-Pollock, et al., 2007	21.45	0.39	[−0.03 , 0.82]		0.56	[0.13 , 0.99]		0.32	[−0.11 , 0.74]	
Rosch et al., 2015	13.75	1.30	[0.72 , 1.85]		1.09	[0.53 , 1.63]		0.30	[−0.22 , 0.81]	
Groom et al., 2010	11.89	0.06	[−0.49 , 0.61]		0.60	[0.02 , 1.15]		0.06	[−0.49 , 0.61]	
Total group difference		0.52	[0.48 , 0.57]		0.49	[0.45 , 0.53]		0.08	[−0.03 , 0.19]	

Fig. 1. Results from the group comparison meta-analyses. CC = control condition, RC = reinforcement condition. $I^2 = 0\%$.

children with ADHD having more room for improvement in baseline inhibition. Rather, the larger effect of reward on inhibition in children with ADHD relative to controls was associated with greater sensitivity to reward as measured with questionnaires. Therefore, including baseline assessments and measures of individual differences in reward sensitivity is a very useful addition for future studies.

4. Functional neuroimaging research

Neuroimaging techniques can be applied to provide insight into the neural correlates associated with reinforcement and inhibition interactions. However, to our knowledge, there are no functional neuroimaging studies on how reward affects inhibition directly in ADHD. Nonetheless, there are two studies in which such effects were addressed indirectly, the findings of which will be discussed here. Secondly, in order to set up guidelines for future functional neuroimaging research in individuals with ADHD, we will discuss a number of relevant functional neuroimaging studies on the interaction between reinforcement/motivation and inhibitory control in non-ADHD samples, as these may guide future research directions in the field of ADHD.

4.1. Functional neuroimaging in ADHD populations

Neuroimaging techniques can be applied to provide insight into the neural correlates associated with reinforcement and inhibition interactions in individuals with ADHD. Liddle et al. (2011) revealed that children with ADHD show a higher motivational threshold (i.e., require more reinforcement) to normalize default mode network deactivation during a reinforced go/no-go task (Liddle et al., 2011). The default mode network is active during resting state and deactivates during engagement in a cognitive task (Greicius, Krasnow, Reiss, & Menon, 2003). Furthermore, Groom et al. (2010) demonstrated that in children with ADHD, event-related potentials associated with inhibition and attention are reduced in a non-reinforced condition of a go/no-go task and normalized to the baseline of the control group during reinforcement (Groom et al., 2010). Others have focused on investigating task-independent resting state functional connectivity in individuals with ADHD (see for reviews Castellanos & Proal, 2012; De La Fuente et al., 2013). Particularly relevant to reinforcement and cognitive control interaction studies is the finding that in children with ADHD, the superior parietal cortex (involved in attention processing) shows lower connectivity with the orbitofrontal cortex and dorsal precuneus while the orbitofrontal cortex shows stronger connectivity with the ventral striatum, suggesting alterations in the interaction between (attention) control and reinforcement processing networks (Tomasi & Volkow, 2012). Although these studies are promising, neuroimaging research on how reinforcement affects inhibition directly in ADHD is lacking. Therefore, designs that turned out to be effective in improving inhibition to a larger extent in ADHD than controls (e.g., Rosch et al., 2015) could be used in combination with fMRI research in order to gain insight into the neural correlates of these effects. Additionally, we will discuss a number of relevant neuroimaging studies on the interaction between reinforcement/motivation and inhibitory control in non-ADHD samples that might also help in guiding future research directions in the field of ADHD.

4.2. Functional neuroimaging in healthy populations

One example of a functional neuroimaging study design that is applicable to the study of reinforcement–inhibition interactions in ADHD are fMRI studies that make use of emotional go/no-go tasks. These tasks are suitable for examining the neural correlates of social reinforcement effects on inhibitory control and may be particularly relevant here because social reward could be powerful for individuals with ADHD (see before). Additionally, these tasks allow for measurement of both the ameliorating and detrimental effects of social reward

on inhibition within the same task (ameliorating effects: sad/angry no-go trials; detrimental effects: happy no-go trials). A developmental fMRI study with typically developing participants revealed that adolescents show hyper-responsivity of the ventral striatum when viewing happy faces compared to the other age groups (Somerville, Hare, & Casey, 2010). Functional connectivity analyses further revealed that adolescents showed co-activation of ventral striatum and dorsal striatum, and of dorsal striatum and right inferior frontal gyrus, a region strongly implicated in inhibition (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003). These are relevant regions of interest, because they have also been shown to play an important role in ADHD (e.g., Cortese et al., 2012). Similarly, in healthy adults, a stop task in which a bias to response execution was created by rewarding fast and accurate response execution (Padmala & Pessoa, 2010). Activity in regions including inferior frontal cortex and putamen decreased during the reward condition as compared to the neutral condition. It is an empirical question how reinforcement manipulations as used in this study will affect inhibitory control in individuals with ADHD, both at the behavioral and the neural level. One may hypothesize that rewarding response execution will exacerbate already present inhibition deficits and associated hypo-activation in prefrontal areas in individuals with ADHD. Clearly, paradigms such as the ones described here in combination with connectivity analyses would be an excellent starting point when examining impairing effects of reinforcement on inhibitory control in individuals with ADHD. Thirdly, reinforced anti-saccade tasks (e.g., Geier, Terwilliger, Teslovich, Velanova, & Luna, 2010; Padmanabhan, Geier, Ordaz, Teslovich, & Luna, 2011) offer a promising approach because these allow for investigation of neural activity during the incentive cue, response preparation, and response phase separately. This can help elucidating the effects of reinforcement on differential aspects of inhibition. Finally, in order to determine whether reinforcement effects and inhibitory control are truly integrated at the neural level, the type of analyses as well as the specific task designs used in fMRI studies can be a crucial tool. Reinforcement and inhibitory control would be truly integrated when certain brain regions activate uniquely in response to reinforcement, others uniquely to inhibition, and yet others uniquely to their interaction (Gray, 2004). Clever designs such as the ones developed by Gray, Braver, and Raichle (2002) could be applied to inhibition tasks in individuals with ADHD in order to test the hypothesis that ADHD-control differences in interaction-related brain activation will be present.

5. Theoretical models: implications for studying the integration of reinforcement and cognitive control in ADHD

In this section we will briefly describe 3 theoretical models of ADHD and some of their implications for future behavioral work on inhibition–reinforcement interactions in ADHD. Next, we will briefly describe 3 neurobiological models on the integration of reinforcement and cognitive control, and how these may aid in the formulation of neural hypotheses for future functional neuroimaging ADHD studies.

5.1. Reinforcement models of ADHD

5.1.1. Dynamic developmental theory

The dynamic developmental theory (DDT) by Sagvolden et al. (2005) hypothesizes that altered meso-limbic dopamine responsivity is associated with ADHD, causing a steeper decrease of the power of reinforcement over time. This is thought to result in relatively strong preferences for small immediate rewards (Sonuga-Barke et al., 1992), and relatively weak impact of the omission of reinforcement on behavior (slower extinction). For the study of inhibition–reinforcement interactions in ADHD, one would predict that effects of reward on inhibition would need to be delivered immediately after the successful inhibition, in order for these to work optimally. Future research could implement designs in which the time between successful inhibition and reward

varies. If timing is indeed crucial, as suggested here, then one would expect that the weakening of reward effects as a function of delay would be stronger in individuals with ADHD than controls. If, on the other hand, reward is more important in individuals with ADHD than timing of the reward, then these weakening effects would be comparable for both groups. One study did compare effects of immediate versus delayed reward on inhibition in a small sample of children with ADHD and controls (Michel et al., 2005). Unfortunately, the task manipulation did not work (i.e., small and immediate rewards had comparable effects), as noted by the authors possibly due to too short delays and the use of fixed condition orders. Future research could study this further by implementing the helpful suggestions as made by Michel and colleagues. Finally, the DDT also predicts that the positive effects of reinforcers on inhibition should be maintained longer in those with ADHD than controls after omitting them. Future research can test this hypothesis.

5.1.2. Dopamine transfer deficit theory

The dopamine transfer deficit (DTD) theory (Tripp & Wickens, 2008) proposes that ADHD is associated with altered anticipatory firing of dopamine cells to cues that predict reinforcement. Based on the seminal work by Schultz, Dayan, and Montague (1997), this theory assumes that when an unconditioned stimulus (the reward) repeatedly follows a conditioned stimulus (the predictor), dopamine neurons not only fire in response to reward delivery, but also begin firing in response to the predictor. Once the reward-predictor association has been established, these cells fire only in response to the predictor. According to the DTD theory, the transfer in firing from reward outcome to reward predictor does not, or only partially take place in individuals with ADHD. As a result, a predictor will not be as powerful in triggering reward anticipation in those with ADHD, and once a reward is delivered, it may seem more unexpected to those with ADHD than controls. Applying this to reinforcement–inhibition interactions, one can imagine that for participants with ADHD, a reward that is delivered after successful inhibition may be relatively unexpected, as they may not learn the association between stop-trial and possible reward optimally. Therefore, in order to make the reward more powerful for individuals with ADHD, it could help to use a reinforcement condition in which the information about potential reward is already included in the stimulus display (e.g., one could use background colors to distinguish reinforcement from neutral blocks or trials, or the stimulus itself could be accompanied by a $ sign, etc.). Additionally, reward magnitudes could be varied around a mean as to increase unexpectedness, which may have more impact for those with ADHD.

5.1.3. Unifying theory

Barkley's unifying theory (1997) predicted that individuals with ADHD have a decreased capacity to induce drive or motivational states, and that as a result, those with ADHD will have a larger dependency on external reinforcers that influence motivation than controls. Although the evidence in this review seems to support this notion (individuals with ADHD, while being reinforced, normalized to controls' "baseline" performance), it is still unclear to what extent individuals with ADHD may improve inhibitory control when intrinsic motivation is supported (Ryan & Deci, 2000). It would be very interesting to directly compare the effects of external reinforcers on inhibition with intrinsic motivation in individuals with ADHD and healthy controls.

5.2. Neurobiological models of reinforcement-cognitive control integration

5.2.1. Non-reciprocal loops

Animal work has revealed dopamine dependent neuroanatomical constructs of the integration of reward and cognitive control. These are comprised of nonreciprocal frontostriato-nigral networks in which information is channeled from ventromedial frontostriatal structures (implicated in reward processing) to more dorsolateral frontostriatal circuits (implicated in cognitive control) to motor-related frontostriatal loops (Haber, 2003). This model proposes a bottom-up hierarchy of

how reward influences cognitive control, which in turn regulates motor output. The implications of Haber's model are highly applicable to ADHD models and have been suggested to help elucidate the neural correlates of the heterogeneous symptoms (Castellanos et al., 2006). Specifically, ventromedial-ventrostriatal as well as medial and orbital prefrontal cortex dysfunction may account for "hot" motivational impairment, in turn affecting goal-directed behavior mediated by the dorsal striatum-dorsolateral prefrontal cortex loop and motor behavior in ADHD-combined type. On the other hand, intact reinforcement processing areas, but compromised dorsolateral prefrontal cortex and motor areas may account for "cool" cognitive control impairments, which may be more strongly associated with ADHD-inattentive symptoms (Castellanos et al., 2006). Therefore, this model provides a framework in which future fMRI research on reinforcement–inhibitory control interactions in ADHD may be embedded. For example, fMRI may be applied to recruit these pathways by using hot and cool conditions within inhibitory control tasks and study the relation between pathway activation and the symptom domains inattention and hyperactivity–impulsivity. Additionally, this framework may be used in designing fMRI studies that assess both ameliorating and impairing effects of motivation on inhibitory control (see Aarts, van Holstein, & Cools, 2011).

5.3. Dual competition framework

Pessoa (2009) argues that distinct subcomponents of cognitive control (e.g. inhibition, shifting and updating) compete with one another for attention resources. A similar competition takes place in the visual cortex for sensory representation. The dual competition framework proposes reinforcement, motivation and emotion to direct information flow of cognitive control and perception. Concretely, the model states that reinforcement, motivation, or affect recruit the anterior cingulate cortex (ACC) via its connections with the amygdala, ventral striatum and orbitofrontal cortex (Pessoa, 2009; Pessoa, Padmala, Kenzer, & Bauer, 2012). The ACC directs attention towards motivationally salient stimuli (reinforced stimuli) through connections with the inferior frontal gyrus and dorsolateral prefrontal cortex. Reinforced stimuli become enhanced in their perceptual representation. These enhanced representations receive increased attention (for example to their spatial location). The competition-aspect comes into play when highly reinforced or motivational events occur. During such events, attentional resources are made available for processing of stimuli with high motivational significance while detracting/depleting resources from other cognitive control components.

Based on the dual competition framework, an interesting target brain area for further research is the ACC. In individuals with ADHD, this brain area has been found to be decreased in volume (Makris et al., 2007), under-activated during cognitive control tasks (Bush et al., 1999; Tamm, Menon, Ringel, & Reiss, 2004), and to have an abnormal functional connectivity with the default mode network (Sun et al., 2012). From the dual competition framework, it follows that impaired ACC functioning potentially leads to inappropriate regulation of motivational significance in ADHD. For example, strongly reinforced stimuli may detract attention from cognitive control more in those with ADHD. One direction for further research that can be derived from this model is to target the anterior cingulate cortex and its connectivity with the ventral striatum in ADHD. However, caution is warranted, as ACC function is highly complex and only few studies have explicitly aimed at exploring the role of the anterior cingulate cortex in ADHD (Bledsoe, Semrud-Clikeman, & Pliszka, 2013; Fan, Gau, & Chou, 2014; Sun et al., 2012).

5.4. Dual processing

Dual processing models (Casey et al., 2008) argue that striatal reward processing areas drive impulsive, immediate rewarding actions, whereas a prefrontal control system regulates behavior in favor of long-

term goals (Metcalfe & Mischel, 1999; Zelazo & Müller, 2002). The interaction between these systems is thought to drive behavior. However, both systems show different developmental patterns. That is, subcortical, reinforcement processing structures, such as the striatum, are shown to mature earlier compared to the prefrontal cortex, resulting in an imbalance between cognitive control and reinforcement processes particularly in adolescence (e.g. Giedd, 2004; Gogtay et al., 2004; Mills, Goddings, Clasen, Giedd, & Blakemore, 2014; Tamnes et al., 2010). Specifically, the immaturity of the prefrontal cortex, combined with a relatively early maturing limbic system, is linked to the increase in risk taking and reward-sensitivity that is observed in adolescence, compared to childhood and adulthood (Casey et al., 2008; Somerville et al., 2010). Extensions of this model highlight that developmental neuroimaging studies do not fully support a simple model of frontal cortical immaturity (Crone & Dahl, 2012; Pfeifer & Allen, 2012). For instance, it is proposed that neurodevelopmental changes in the interactions between motivational and control-systems may lead to less automatic and more flexible cognitive control allocation in adolescence.

According to the dual processing model, this imbalance in prefrontal and subcortical structures is most prevalent in adolescence, leading to typical adolescent behaviors, among which heightened impulsivity (Casey et al., 2008). As ADHD is partly characterized by heightened levels of impulsivity, as well as by relatively weak cognitive control and altered reward sensitivity, it is relevant to study the balance in maturity between the prefrontal cortex and the limbic system in relation to the interaction between cognitive control and reinforcement effects in individuals with ADHD, using designs similar to the ones as used in the adolescent neurodevelopmental literature. Evidently, because symptom persistence of ADHD seems to be sensitive to developmental changes (individuals with a childhood diagnosis of ADHD show 15% full and 65% partial remission in adulthood, Faraone, Biederman, & Mick, 2006), taking on a developmental perspective is therefore informative of the developmental changes associated with the disorder. Neuroimaging studies in healthy controls have shown volume decreases in the caudate with age while in ADHD no age-related changes were reported (Castellanos et al., 1994). Besides structural development of the striatal and frontal cortex as separate entities, deviant functional and structural frontostriatal connectivity have been implicated in ADHD (for a review see Liston, Cohen, Teslovich, Levenson, & Casey, 2011). However, longitudinal research is needed to adequately investigate the potential role of an imbalanced striatum and prefrontal cortex development and their connectivity, in hot forms of cognitive control in ADHD.

6. Specificity

6.1. Disorder specificity

It should be noted that hot cognitive control abnormalities in ADHD may be associated with comorbid behavioral disorders such as oppositional defiant disorder and conduct disorder. This notion arises from studies that have employed a rewarded continuous performance task (a rewarded sustained attention task) combined with fMRI (Rubia, Smith et al., 2009). Here, the rewarded target trials were contrasted against the neutral target trials and abnormal activation of orbital frontal areas was only found in patients with pure conduct disorder, but not in patients with pure ADHD. In another study, adults with persisting ADHD symptoms showed hypoactivation in the right ventromedial prefrontal cortex/orbitofrontal cortex and in the right medial and superial frontal cortices compared to controls (Cubillo, Halari, Smith, Taylor, & Rubia, 2012). Post-hoc analyses illustrated that abnormal activity in VMPFC/orbitofrontal cortex was only found in those patients with comorbid conduct disorder. Only one other study focused on the effects of motivation on cognitive control in individuals with ADHD and another clinical group, namely those with autism spectrum

disorders (Geurts et al., 2008). Interestingly, the results indicated that participants with ADHD benefited more from social motivation than those with autism spectrum disorder on an interference control task. However, when directly comparing monetary to social rewards, Demurie et al. (2011) found that children with ADHD had similar reaction times to healthy controls during a monetary reward condition, but children with ADHD were relatively slow during the social reward condition. The autism spectrum disorder group, on the other hand, was slower than healthy controls in both reward conditions, although this group difference was most pronounced in the social reward condition. In sum, the question of specificity clearly warrants further investigation: more research is needed with multiple clinical groups before we can draw conclusions as to what extent unique motivation–cognitive control interactions are related to ADHD, or more commonly observed in other (comorbid) clinical conditions.

6.2. Cognitive control domain specificity

The causal model of ADHD by Barkley (1997) proposes that difficulties in inhibitory control form a core deficit, which in turn leads to secondary impairments in other cognitive-control domains, giving rise to ADHD symptoms. Additionally, in the meta-analytic review by Willcutt et al. (2005), it is shown that response inhibition yields one of the most consistent group differences between ADHD and controls (82% of all studies) of cognitive control domains. Based on these grounds, this review's main focus is on response inhibition. However, other domains of cognitive control are also compromised in ADHD. Specifically, working memory, sustained attention (vigilance) and planning yield similar effect sizes as response inhibition (Willcutt et al., 2005; 2008). Reinforcement effects on working memory and sustained attention have been addressed in a number of studies. Generally, the studies on working memory found that children with ADHD improved more relative to controls when reinforcers were administered (Dovis, Van der Oord, Wiers, & Prins, 2013, 2012, 2015; Strand et al., 2012). For sustained attention, such an altered effect of reinforcement in the ADHD group was absent (Rubia, Smith, & Taylor, 2007; Rubia, Smith et al., 2009; Rubia, Halari, Cubillo et al., 2009 but see Bubnik, Hawk, Pelham, Waxmonsky, & Rosch, 2015 for an exception). Based on these studies, it will be interesting to directly compare reinforcement effects on inhibitory control and working memory/sustained attention within the same individuals, in order to address the question whether those with ADHD are generally more sensitive to rewards (if this is the case, the ADHD group would improve to a larger extent than controls under reinforcement on all cognitive control tasks), or whether the unique ADHD-related improvement under reinforcement is specifically related to one of the domains.

6.3. Task specificity: detrimental effects of reward

In this review, we have highlighted that when response execution is associated with potential rewards in an inhibition task (stop signal task), this can lead to decrements in inhibitory control, as has been shown in healthy adults. We have suggested that it is highly relevant for the ecological validity of inhibitory control studies in ADHD to investigate the detrimental effects of reinforcement on inhibitory control aside from the positive effects. It may be argued that such detrimental consequences of rewards on inhibitory control are also measured in temporal discounting and risky decision tasks. Temporal discounting refers to the decrease of the subjective value of a reward as the delay to obtain that reward increases. In temporal discounting tasks participants choose between an immediate small reward and a larger but delayed reward, and thus the tendency to select the immediate reward needs to be inhibited in favor of the larger delayed reward when someone wants to maximize their gains. Temporal discounting has been shown to be relatively strong in those with ADHD, indicating a stronger preference for immediate rewards in ADHD groups than

controls (Sonuga-Barke et al., 2008; Sonuga-Barke et al., 2010). Risk-taking paradigms typically consist of choices between options with a different probability of reward. For instance, a risk-taking paradigm may present the choice between a 'safe' option (i.e., a small sure reward) and a 'risky' option (i.e., a gamble with a certain probability of obtaining a larger reward or a negative outcome). A recent review by Groen and colleagues (2013) revealed that children and adolescents with ADHD displayed more risky behaviour than controls in 50% of the studies. Both temporal discounting and risk taking studies are thus in support of the idea that the presence of rewards may impair inhibition, especially in ADHD groups. It should be noted, however, that temporal discounting and risk taking are distinct constructs from motor inhibition as measured with stop or go/no-go tasks (e.g., Bari & Robbins, 2013; Robbins et al 2012). Typically, for temporal discounting the relationship with motor inhibition is absent, including in ADHD populations (Solanto et al., 2001; Sonuga-Barke et al., 2010). Also, the role of inhibitory control in risk-taking is inconsistent (e.g. Brevers et al., 2012; de Water et al., 2014; Geurts et al., 2006). In sum, future investigations of detrimental effects of rewards on motor response inhibition specifically are encouraged.

7. Conclusion

Reinforcement and cognitive control interactions reflect daily life conditions and this interplay is increasingly studied in typically developing populations as well as in psychiatric disorders. The current review and meta-analyses demonstrated that youth with ADHD benefited more from reinforcement contingencies than healthy controls on inhibitory control tasks. Meta-analyses further demonstrated that youth with ADHD may normalize inhibitory control during reinforcement to the baseline performance level of controls. These findings endorse the use of reinforcement schedules as ADHD treatment.

We recommend future behavioral research to focus on under which circumstances this interaction takes place (e.g., varying reinforcement schedules including magnitude and expectancy and reward type), which individual differences contribute to these interactions (e.g., questionnaire-based reward sensitivity) and direct comparison of the effects of external reinforcers versus intrinsic motivation. Furthermore, special attention needs to be given to extra control conditions and order effects, as well as detrimental effects of reward on inhibitory control.

Neuroimaging can complement behavioral studies, because altered neural responses to both inhibitory control and reward are clearly associated with ADHD. FMRI studies could start out by using designs that have proven to be effective in showing group by condition interactions. Neurobiological models can be used to identify certain regions (e.g., ACC) or networks (e.g., ventromedial and dorsolateral fronto-striatal circuitries) of interest while studying this relevant interaction in individuals with ADHD.

Author disclosure

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