Cogn Affect Behav Neurosci (2014) 14:698–714
DOI 10.3758/s13415-014-0298-3

Impaired reward processing by anterior cingulate cortex in children with attention deficit hyperactivity disorder

Akina Umemoto · Carmen N. Lukie · Kimberly A. Kerns · Ulrich Müller · Clay B. Holroyd

Published online: 30 May 2014

Abstract Decades of research have examined the neurocognitive mechanisms of cognitive control, but the motivational factors underlying task selection and performance remain to be elucidated. We recently proposed that anterior cingulate cortex (ACC) utilizes reward prediction error signals carried by the midbrain dopamine system to learn the value of tasks according to the principles of hierarchical reinforcement learning. According to this position, disruption of the ACC–dopamine interface can disrupt the selection and execution of extended, task-related behaviors. To investigate this issue, we recorded the event-related brain potential (ERP) from children with attention deficit hyperactivity disorder (ADHD), which is strongly associated with ACC–dopamine dysfunction, and from typically developing children while they navigated a simple "virtual T-maze" to find rewards. Depending on the condition, the feedback stimuli on each trial indicated that the children earned or failed to earn either money or points. We found that the reward positivity, an ERP component proposed to index the impact of dopamine-related reward signals on ACC, was significantly larger with money feedback than with points feedback for the children with ADHD, but not for the typically developing children. These results suggest that disruption of the ACC–dopamine interface may underlie the impairments in motivational control observed in childhood ADHD.

Keywords Reinforcement learning · Motivation · Cognitive control · ADHD

Experimental psychologists are well familiar with "good" participants who happily engage with their assigned task to the best of their ability despite little or no compensation, as well as participants who appear disinclined to perform the same task irrespective of the incentives offered. What determines subjects' motivation for participating in and completing an experiment? Research over the past two decades has highlighted the critical role of dorsolateral prefrontal cortex (DLPFC) in executing such goal-directed, effortful behaviors (Stuss & Knight, 2013). In particular, DLPFC is said to apply *top-down* control over the execution of "task sets" (Cohen, Dunbar, & McClelland, 1990; Miller & Cohen, 2001), or the "configuration of cognitive processes that is actively maintained for subsequent task performance" (Sakai, 2008, p. 219). This proposal was originally formalized in a computational model based on principles of parallel distributed processing in which "task demand units" in prefrontal cortex facilitate processing along stimulus–response pathways responsible for task execution (Cohen et al., 1990), and elucidated by subsequent studies that examined how DLPFC regulates the degree of top-down control according to evolving task dynamics (Botvinick, Braver, Barch, Carter, & Cohen, 2001). Nevertheless, the success of this theoretical framework notwithstanding, it remains to be determined how DLPFC selects what task to execute in the first place, as well as how much control to exert over the task once selected.

Recent developments in reinforcement learning theory may provide an answer to this question. The principles of hierarchical reinforcement learning (HRL) extend standard approaches to reinforcement learning by representing complex sequences of behaviors at higher levels of temporal abstraction (Botvinick, 2012). These abstract behaviors, called

Electronic supplementary material The online version of this article (doi:10.3758/s13415-014-0298-3) contains supplementary material, which is available to authorized users.

A. Umemoto · C. N. Lukie · K. A. Kerns · U. Müller · C. B. Holroyd
University of Victoria, Victoria, British Columbia, Canada

A. Umemoto (✉)
Department of Psychology, University of Victoria, 3800 Finnerty Road, Victoria, British Columbia V8N 1M5, Canada
e-mail: aumemoto@uvic.ca

options, are learned about and manipulated as units, which enhances computational efficiency for problems characterized by hierarchical structure. On the basis of similarities between the concepts of options and task sets, Botvinick, Niv, and Barto (2009) suggested that DLPFC may be responsible for option selection and maintenance. Alternatively, we have developed this proposal further by suggesting that a different brain structure for cognitive control—anterior cingulate cortex (ACC)—subserves this role (Holroyd & Yeung, 2012; see also Shenhav, Botvinick, & Cohen, 2013). According to this account, ACC learns the value for the task at hand by utilizing reward prediction error signals carried by the midbrain dopamine (DA) system, which encodes the discrepancy between the expected and actual reward outcomes (Schultz, 2002; Schultz, Dayan, & Montague, 1997) for the purpose of reinforcement learning and decision making (Montague, Hyman, & Cohen, 2004). Subsequently, ACC selects tasks for execution on the basis of their learned value and biases DLPFC activity to enforce top-down control over the selected task. This proposal, which unifies a wide body of literature on control, reward processing, and decision making (reviewed in Holroyd & Yeung, 2012; see also Holroyd, 2013; Holroyd & McClure, 2014), suggests that disruptions to the ACC–DA system should impair motivation of task-appropriate behaviors.

Attention deficit hyperactivity disorder (ADHD) provides a model for investigating these behavioral and neurocognitive issues. One of the most common neurodevelopmental disorders in childhood, ADHD is characterized by a persistent pattern of age-inappropriate inattention, hyperactivity, and/or impulsivity that is estimated to occur in approximately 3 %–7 % of school-age children (American Psychiatric Association, 2013). Although both genetic and environmental factors are believed to contribute to ADHD, its etiology and neurobiological basis remain unclear (Swanson et al., 2007). Impairments in cognitive control related to inhibition, attention, and working memory are commonly reported (Barkley, 1997; Chamberlain et al., 2011; Nigg, 2005, 2006; Quay, 1997; Schachar & Logan, 1990; Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003; Sonuga-Barke, 2002, 2003; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Children with ADHD also show various behavioral impairments associated with reward processing. For example, they process the costs and benefits of ongoing events differently from typically developing children, which gives rise to altered reinforcement learning and to motivational deficits (Luman, Oosterlaan & Sergeant 2005; see Luman, Tripp, & Scheres, 2010, for a review). Children with ADHD appear atypically sensitive to rewards (C. L. Carlson & Tamm, 2000; McInerney & Kerns, 2003), especially to the consistency of reward delivery (Douglas & Parry, 1994) and to recent rewards, which strongly impacts their behavior, due to a failure to integrate their previous reward history over time (Sonuga-Barke, Taylor, Sembi, & Smith, 1992; Tripp & Alsop, 1999, 2001). They are also relatively insensitive to changing rates of reinforcement (Kollins, Lane, & Shapiro, 1997) and to increases in penalty sizes (Luman, Oosterlaan, Knol, & Sergeant, 2008). Further evidence suggests that externally provided motivators such as monetary incentives, tokens, and social rewards tend to normalize their impairments in cognitive control (Dovis, Van der Oord, Wiers, & Prins, 2012; Kohls, Herpertz-Dahlmann, & Konrad, 2009; Konrad, Gauggel, Manz, & Schöll, 2000; Shiels et al., 2008), perhaps because salient incentives tend to invigorate their otherwise diminished response to abstract rewards.

At a neural level, ADHD is strongly associated with abnormal function of both the ACC and the DA system (but see also Nigg & Casey, 2005; Sonuga-Barke, 2002, 2003). Dysfunctions within the frontal, cingulate, and striatal regions are well-documented, highlighting in particular structural and functional abnormalities in ACC (Bush, 2009; see Bush, 2011, for a review) and striatum (Plitcha & Scheres, 2014). For example, ACC is hypoactive in children with ADHD when they are engaged in tasks that demand cognitive control (e.g., Konrad, Neufang, Hanisch, Fink, & Herpertz-Dahlmann, 2006; Rubia et al., 2010; Rubia et al., 2008), possibly due to impaired utilization of reinforcement–action contingencies for motivating task-appropriate behaviors (Bush, 2009). Further, administration of psychostimulants such as methylphenidate (e.g., Ritalin, Concerta), which increases extrasynaptic DA levels by blocking DA reuptake in the striatum (see Cragg & Rice, 2004, for a review; Kuczenski & Segal, 1975; Wilens, 2008), normalizes ACC hypoactivity (Bush, 2009), and improves the behavioral and cognitive symptoms of ADHD (Arnsten, 2006; Rubia et al., 2009; Wilens, 2008). Stimulant medications are also suggested to normalize the size of ACC, which is otherwise smaller in medication-naïve children with ADHD (Semrud-Clikeman, Pliszka, Bledsoe, & Lancaster 2012; Semrud-Clikeman, Pliszka, Lancaster, & Liotti, 2006).

Converging lines of evidence also point to altered processing by the midbrain dopamine system, which is believed to code for when events are "better" or "worse" than expected, as phasic increases and decreases from baseline activity, respectively (Schultz et al., 1997). Several recent theories have proposed that the behavioral impairments observed in children with ADHD stem from the functional consequences of disrupting these phasic signals. For example, a prominent DA-related theory of ADHD, the dynamic developmental theory (DDT), holds that DA hypofunctioning results in impaired learning of behavior–outcome associations, producing a short and steep delay-of-reinforcement gradient and poor behavioral extinction (Sagvolden, Johansen, Aase, & Russell, 2005). A similar hypothesis by Tripp and Wickens (2009) proposes that the DA system fires normally in response to

rewards, but that these phasic signals are not effectively transferred back to cues and actions that predict future reward delivery, resulting in impaired anticipatory cue learning. Other theories propose exaggeratedly large positive and negative reward prediction errors (Grace, 2001), abnormally small (Volkow, Wang, Fowler, & Ding, 2005) or large (Seeman & Madras, 2002) positive prediction errors, or impaired cortical–subcortical circuits involving the DA pathway (Frank, Santamaria, O'Reilly, & Willcutt, 2007; Nigg & Casey, 2005; Sonuga-Barke, 2002, 2003). Cockburn and Holroyd (2010) investigated a range of these hypotheses by systematically manipulating the size of positive and negative phasic DA signals in a computational simulation of performance on a reinforcement learning task. They found that simulations with asymmetrically larger phasic DA increases (positive signals) relative to phasic DA decreases (negative signals) accounted best for the behavior of children with ADHD relative to typically developing children. Finally, Silvetti, Wiersema, Sonuga-Barke, and Verguts (2013) reported that simulated disruption to midbrain DA signals gives rise to disrupted probabilistic learning, steep temporal discounting, and impaired performance under a partial-reward schedule as observed in ADHD. They concluded that the reduced transmission of midbrain DA signals to ACC results in the motivational deficits associated with ADHD.

Biological considerations also point to DA-system dysfunction. Although no single gene determines whether an individual will exhibit the symptoms of ADHD, several DA-related genes weakly predict increased risk for ADHD (Banaschewski, Becker, Scherag, Franke, & Coghill, 2010). Furthermore, a commonly studied rat model of ADHD is characterized by increased expression of a gene that codes for the dopamine transporter (DAT) in DA-innervated areas (Sagvolden & Johansen, 2012), and administration of methylphenidate counteracts the synaptic effects of this increase by blocking DA reuptake by DATs (Roessner et al., 2010). Polymorphisms in the dopamine D4 receptor gene (DRD4), which is expressed abundantly in prefrontal cortex (PFC) and ACC, have also been associated with increased risk for ADHD (Faraone, Doyle, Mick, & Biederman, 2001; Li, Sham, Owen, & He, 2006; Swanson et al., 2000).

These converging lines of evidence suggest that disruption of the ACC–DA interface may underlie the abnormalities in reinforcement learning and motivational control observed in ADHD. To investigate this possibility, we had previously examined the reward positivity, also known as the *feedback error-related negativity* or *feedback-related negativity*, in children with ADHD (Holroyd, Baker, Kerns, & Müller, 2008a; cf. van Meel, Heslenfeld, Oosterlaan, Luman, & Sergeant, 2011; van Meel, Oosterlaan, Heslenfeld, & Sergeant, 2005). The reward positivity is a component of the event-related brain potential (ERP) that is elicited in reinforcement learning and guessing tasks by unexpected positive feedback stimuli.[1] This ERP component is distributed over fronto-central areas of the scalp and appears to index the impact of midbrain DA reward signals on ACC (Holroyd & Coles, 2002; see Walsh & Anderson, 2012, for a review; but see also J. M. Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011; Foti, Weinberg, Dien, & Hajcak, 2011). In the previous study (Holroyd et al., 2008a), the electroencephalogram (EEG) was recorded from children with ADHD and typically developing children as they navigated a virtual T-maze from a first-person perspective to find monetary rewards (Baker & Holroyd, 2009). On each trial, the children selected between the left and the right alleys of the maze by pressing one of two corresponding buttons, and were presented with a feedback stimulus indicating 5 cents or 0 cents (represented by images of an apple and an orange) presented at the end of the selected alley. Halfway through the experiment, the participants were physically given their accumulated rewards. We found that neither the amplitude of the reward positivity nor of any other ERP components differed between the groups. However, when the data were averaged separately before and after the midway payment, the reward positivity amplitude was larger for the children with ADHD following the payment than before the payment. We interpreted this result as suggesting that relative to typically developing children, children with ADHD are abnormally sensitive to motivationally salient rewards—such as the physical presence of money—relative to more abstract rewards such as positive and negative feedback. In particular, motivationally salient rewards may normalize an otherwise *insensitive* response to abstract rewards in children with ADHD. These results point to a disturbance of the ACC–DA interface in childhood ADHD.

Nevertheless, these findings are qualified by some methodological concerns about the study. First, because the main result was not predicted a priori, it may have been a statistical anomaly. Second, the study did not rule out the possibility that the larger reward positivity observed in children with ADHD after they were rewarded in the second half of the experiment, relative to before they were rewarded in the first half of the experiment, was due to time on task rather than to reward salience. Third, although the diagnoses for children with ADHD and for the typically developing controls were confirmed by administration of a diagnostic questionnaire to their parents and caregivers, this screening procedure would be

[1] Although the negative deflection in the ERP elicited by negative performance feedback is often associated in the literature specifically with errors, more recent evidence has suggested that the negative deflection is actually the N200 ERP component, which is elicited by unexpected, task-relevant events in general (Holroyd, 2004; Holroyd, Pakzad-Vaezi, & Krigolson, 2008b). According to this view, unexpected positive feedback stimuli would elicit phasic increases in dopamine that would suppress the N200, giving rise to the reward positivity (Foti, Weinberg, Dien, & Hajcak, 2011; Holroyd et al., 2008b).

improved with independent clinical interviews. Finally, participants' intelligence quotient (IQ) scores were not measured, leaving open the possibility that the results might reflect group differences in IQ rather than the disorder itself (see also van Meel et al., 2011).

Here, we examined the relationship between the reward positivity and ADHD by replicating and extending the previous study. We recruited a new sample of typically developing children and children with combined subtype ADHD from local school districts. Most children in the ADHD group were previously diagnosed with ADHD by qualified healthcare professionals (e.g., registered psychologists, pediatric psychiatrists or neurologists, and pediatricians),[2] whereas the control group excluded children with a previous diagnosis of ADHD. The diagnoses were then independently confirmed by doctorate-level students from the Department of Psychology clinical program using the Diagnostic Interview for Children and Adolescents (DICA-IV; Multi-Health Systems Inc.) and were further supported by parent and teacher ratings on the Conners-3 short versions (Multi-Health Systems Inc.).We also assessed IQ levels as well as the presence of common comorbid disorders, including oppositional defiant disorder (ODD), conduct disorder (CD), generalized anxiety disorder (GAD), depression, and learning disability. All children performed two conditions of the T-Maze task (Baker & Holroyd, 2009), the order of which was counterbalanced across participants: In a *points* condition, the feedback stimuli represented abstract points, and in a *money* condition, the feedback stimuli indicated that actual money would be awarded to the participants at the end of the experiment. After every block of trials in the money condition, children were awarded their accumulated earnings in Canadian nickels added to a clear glass jar placed on a desk beside the computer screen. We predicted that the children with ADHD would exhibit larger reward positivities in the money condition than in the points condition, irrespective of condition order, whereas the typically developing children would show comparably sized reward positivities across the two conditions.

Method

Participants

Participants 8 to 13 years of age were recruited from three school districts from and around Victoria by way of newsletters sent home from school, flyers posted on the campus of the University of Victoria, its surrounding areas, and in the offices of local pediatricians, and advertisements in local parent magazines. The effect size observed in our previous study (Cohen's $d = 0.73$; Holroyd et al., 2008a) indicated about 108 participants would be needed to attain a statistical power >.95.[3] All children were first screened via a phone conversation with their parents or caregivers; to be included in the study, all of the children with ADHD were previously diagnosed by a qualified healthcare practitioner (e.g., by a registered psychologist, pediatric psychiatrist, neurologist, or pediatrician; see note 2 for the exceptions). Children who had neurological or psychiatric disorders other than ADHD and its associated comorbid disorders as reported by parents or caregivers were excluded from the study.

After being informed that they would have a total of two or three appointments, depending on the outcome of the first session, the families of eligible children were invited to the first appointment, which took place in our laboratory in a standard testing room for children and a private interview room for parents or caregivers. The Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) was administered to each child participant to obtain the verbal, performance, and full-scale IQ scores, as well as the reading and mathematics sections of the Wechsler Individual Achievement Test-II (WIAT-II; Psychological Corp., 2002), by either an experienced psychometrician or an advanced graduate student (minimum master-level assessment training) in the Clinical Psychology program. In parallel, a doctorate-level graduate student in the program administered the Diagnostic Interview for Children and Adolescents (DICA; Reich, 2000) to the child's parent(s) or primary caregiver(s). This interview was audio recorded and the diagnosis was subsequently confirmed by a blind interrater. Parents also completed a short version of the Conners Rating Scale Revised (Multi-Health Systems Inc.), as well as the Behaviour Rating Inventory of Executive Function (BRIEF; Gioia, Isquith, Guy, & Kenworthy, 2000). Finally, the Conners and BRIEF questionnaires designed for teachers were sent home with the parents during the second session, to be given to and completed by each child's teacher. For the purpose of clinical evaluation and diagnosis, children prescribed with medication were asked to take it as usual prior to the first session, which lasted approximately 2.5 h.

To be included in the ADHD group, children were required to meet the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) criteria for the ADHD-combined subtype from both the primary and secondary raters. Children prescribed ADHD-related medications were not excluded. None of the children presenting as typically developing (the control group) met the criteria for a diagnosis of ADHD or

[2] We included two children who were not officially diagnosed with ADHD by qualified health care professionals, as well as two additional children whose ADHD diagnoses were made by family physicians—however, the presence of ADHD-combined type in these participants was confirmed through our own clinical diagnostic interview procedure.

[3] We initially ran approximately 40 participants and analyzed their data for the purpose of a thesis defense, at which point it was decided to collect a second wave of participants.

were on any psychiatric medications; five children in the control group were excluded due to their Conners behavioral scores falling in the clinical range (see the Results section, below). All children were required to have an IQ above 85. Children were excluded if they exhibited evidence of (1) neurological or psychiatric disorders other than ADHD, as assessed during the initial phone screening or during the diagnostic interview (e.g., autism, Tourette's disorder, fetal alcohol syndrome, or seizure/epilepsy, excluding the comorbid disorders associated with ADHD); (2) a history of head injuries or concussions; or (3) uncorrected visual or hearing impairments. Because ADHD is a complex, heterogeneous disorder that often co-occurs with other externalizing and internalizing disorders (i.e., comorbid disorders), the presence of the following comorbid disorders was also assessed with the DICA: ODD, CD, GAD, and a major depressive episode (MDE) past or present. Learning disorders (LD) were identified by comparing the math and reading WIAT-II scores separately with the full-scale WASI IQ scores; a discrepancy equal to or larger than two standard deviations (≥30) between the full-scale WASI IQ scores and the WIAT-II scores was considered a LD. The comorbid disorders, LD, and ADHD-related medication status were utilized to control for additional sources of variance in the data as appropriate for the statistical analyses. Each family's socioeconomic status (i.e., gross annual household incomes) was also obtained for the participants.

During a second session on a different day, children performed a simple reinforcement-based task (the T-maze task) while ongoing EEG was recorded (Baker & Holroyd, 2009). They subsequently performed a child-friendly version of the Iowa Gambling Task (Bechara, Damasio, Damasio, & Anderson, 1994; Bechara, Tranel, & Damasio, 2000), the results of which are not reported here. All children diagnosed with ADHD were required to withdraw from any ADHD-related medications 24 h prior to this second session (21 children with ADHD were taking stimulant medications, and two children with ADHD were taking other mediations[4] for the disorder). The second session lasted on average 1.5 h. A subset of these children were later invited back to participate in a third, final session that involved another reinforcement-based behavioral task called the *probabilistic selection* task (Frank, Seeberger, & O'Reilly, 2004), the results of which are reported in the supplementary materials, as well as a sequence of memory-related tasks that will be reported elsewhere (Talbot & Kerns, in press).

All sessions took place in our laboratories on the University of Victoria campus. All participants except one completed each appointment on separate weekends; most children completed the second appointment within a few months of completing the first. For each session, parents received $10 CAN compensation for their time and reimbursement for any parking costs and transportation fees. One family recruited from outside the greater Victoria area was compensated for room and board expenses. Children received a small toy following each session and earned approximately $5 CAN for their performance on the T-Maze task (see below). Written assent and consent were provided by the parents and children prior to participation. The experiment was approved by the human subjects review board at the University of Victoria and was conducted in accordance with the ethical standards prescribed in the 1964 Declaration of Helsinki.

Procedure

During the second, ERP session, children were seated comfortably in front of a computer monitor in a dimly lit electromagnetically shielded room. The T-Maze task requires participants to move in a T-shaped "virtual maze" by turning right or left toward either arm of the maze to find rewards at the end of the selected arm (Fig. 1; Baker & Holroyd, 2009). At the start of each block of trials, participants were provided with an overview of the maze (Fig. 1, top three panels). On every trial, an initial screen image displayed the maze stem for 1,000 ms (Fig. 1, lower left panel) followed by a green double arrow at the end of the alley (Fig. 1, lower left-middle panel) that served as a prompt for participants to choose which way they wanted to turn. The arrow was presented until participants responded by pressing either a left or a right button on a stimulus–response box (Psychology Software Tools, Pittsburgh, PA), corresponding to the left and right alleys, respectively. When a response was made, an image of the selected alley appeared for 500 ms, followed by an image of either an apple or an orange for 1,000 ms, together with the selected alley. Participants engaged in both "money" and "points" reward conditions of the task, the order of which was counterbalanced across subjects. In the money condition, participants were told that a particular fruit (e.g., an apple) indicated that they had earned a 5-cent reward, whereas a different fruit image (e.g., an orange) indicated that they had earned no money on that trial. In the points condition, these same images indicated 5-point versus 0-point outcomes, consistent with the reward mappings used in the money condition (e.g., if the orange stimulus represented 5 cents in the money condition, then it also represented 5 points in the points condition). The mapping of stimuli across the reward conditions was counterbalanced across participants. Participants were told to navigate the maze so as to maximize their reward/points earnings. Unbeknownst to the participants, the feedback type was delivered at random with 50 % probability. The feedback image was followed by a blank screen delay for 1,000 ms, and then the next trial began.

Both the points and money conditions of the T-maze task were composed of four blocks of 50 trials each. Rest periods

[4] Clonidine and Strattera

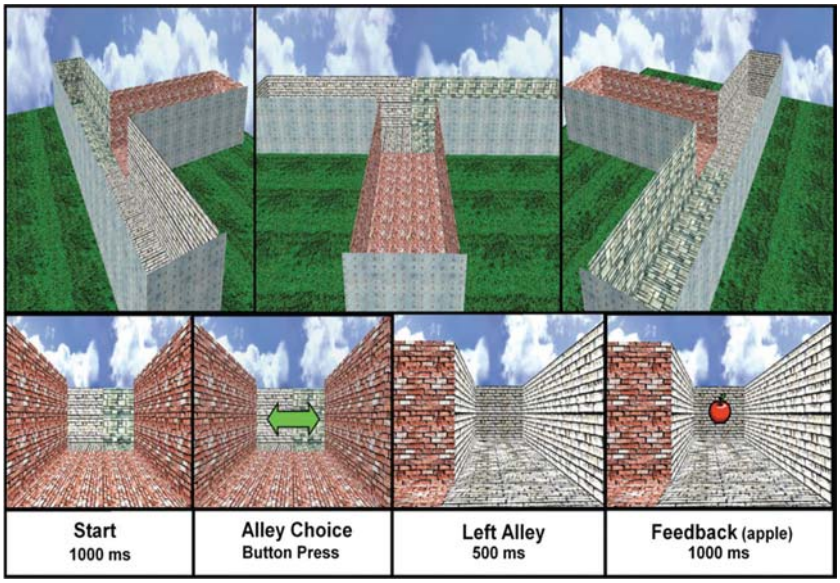

Fig. 1 Top: View of the T-maze from above, as seen from three different angles. Bottom: Example sequence of events and associated timings in each trial, extending from the left to the right panel. The right panel depicts an example image of reward feedback (apple). The bottom line shows stimulus durations; the double arrow remained visible until the buttonpress. (Note that the double arrow is enlarged here for the purpose of illustration.)

were provided between blocks, the durations of which were controlled by the participants. During the money condition, participants' accumulated winnings were physically provided to them during the rest period following each block by adding the money to a clear glass jar placed on a table in front of them, enabling them to visually see their earnings. Participants were told that all the money that they earned would be theirs to take home at the end of the experiment. Participants were not told about the second condition until the first condition was completed. All of the participants were encouraged to respond quickly to keep them engaged in the task, especially if they showed boredom or frustration.

ERP data acquisition and analysis

During the T-Maze task, participant EEGs were recorded from 19 electrode sites using BrainVision Recorder Software (Brainproducts, GmbH, Munich, Germany). Signals were acquired using sintered Ag/AgCl ring electrodes mounted in a fitted nylon cap with a standard 10–20 layout and referenced to a common ground. The horizontal electrooculogram (EOG) was recorded from the external canthi of both eyes, and the vertical EOG was recorded from the suborbit of the right eye and electrode channel Fp2 for the purpose of artifact correction. Interelectrode impedances were kept below 15 kΩ, and two electrodes were placed on the right and left mastoids. During recording all activity was referenced to an overall average. The EEG data were sampled at a rate of 250 Hz and amplified by low-noise electrode differential amplifiers with a frequency response of DC 0.017–67.5 Hz (90-dB octave roll-off).

Postprocessing and data visualization were performed using the Brain Vision Analyzer software (Brainproducts, GmbH). The EEG data were filtered through a phase-shift-free Butterworth filter with a passband of 0.10–20 Hz. An 800-ms epoch of data extending from 200 ms prior to feedback stimulus onset to 600 ms following the stimulus was extracted from the continuous EEG for analysis. Ocular artifacts were corrected using the eye movement correction algorithm described by Gratton, Coles, and Donchin (1983). The EEG data were re-referenced to linked mastoid electrodes and baseline corrected by subtracting from each sample the average activity recorded at that electrode during the 200-ms interval preceding stimulus onset. Trials with muscular and other artifacts were discarded when the maximum change in voltage at any channel exceeded 200 μV across each 200 ms, or 50 μV across one sample. The EEG data were then resegmented by condition (reward or no-reward).

The single-trial EEG time-locked to feedback type (reward or no reward) was averaged for each electrode and participant, to create ERPs separately across points and money conditions. The reward positivity was measured at electrode site FCz, where it typically reaches maximum amplitude (Holroyd & Krigolson, 2007; Miltner, Braun, & Coles, 1997). The reward positivity was extracted for each participant by subtracting the average ERPs associated with reward from those associated with no-reward feedback (Holroyd & Coles, 2002; Holroyd & Krigolson, 2007). Reward positivity amplitude was then determined by averaging the value of the difference wave from

200 to 400 ms following feedback onset. Values for the "raw" ERPs were also determined by averaging the ERPs separately for reward and no-reward feedback trials during the same time window.

Both ERP and behavioral measures were analyzed using the Statistical Package for the Social Sciences (SPSS 17.0, IBM, Armonk, NY). Standard errors of the means are reported along with the means in all analyses, in parentheses. Cohen's *d* was calculated on the basis of the pooled raw score standard deviations in the denominator for both the paired and unpaired *t* tests.

Results

Of the 111 participants who completed the ERP session, five children in the control group were excluded from the final ERP analysis because of inattention and hyperactivity/impulsiveness scores on the Conners questionnaire for parents that exceeded threshold (*t* score > 70), and the data for one participant were missing, yielding data for a total of 105 participants.

Demographic information

Table 1 presents the participants' demographic information. Although the small difference (7 points) on the WASI full-scale IQ measure between the typically developing children and children with ADHD was statistically significant ($p = .01$), no significant effects of sex, age, or IQ emerged in the electrophysiological measures.[5] The family gross annual household incomes for the participants ($N = 102$; 57 control) were coded on a scale of 1 to 5 (1 = *below $20,000*, 2 = *$20,000–$40,000*, 3 = *$40,000–$60,000*, 4 = *$60,000–$80,000*, and 5 = *over $80,000*); the mean incomes were similar between the typically developing children (4 ± 0.1) and the children with ADHD (3.7 ± 0.2), with close to half the families ($N = 45$) having over $80,000 incomes. Diagnoses were confirmed by the Conners scores reported by parents and caregivers, which revealed inattention levels that were significantly higher for the ADHD group (82.8 ± 1.6) than for the control group (47.6 ± 0.9), $t(95) = -20.83$, $p < .01$, and hyperactivity/impulsivity levels that were significantly higher for the ADHD group (85.3 ± 2.5) than for the control group (49.6 ± 1.0), $t(95) = -14.79$, $p < .01$. These same measures, as reported by the teachers (80 % return rates), supported the parental reports: The Conners scores for the ADHD group ($n = 28$) were significantly higher than those for the control group ($n = 49$) for both inattention (control, 52 ± 1.6; ADHD, 71.7 ± 1.4), $t(75) = -8.36$, $p < .01$, and hyperactivity/impulsivity (control, 54.4 ± 2.3; ADHD, 80.2 ± 3.1), $t(75) = -6.74$, $p < .01$.We also examined the subset of reports by teachers who confirmed that the children with ADHD were not taking ADHD-related medications while in their care, which yielded comparable results ($n = 12$) [inattention levels: control, 52 ± 1.6; ADHD, 75.3 ± 3.2; $t(59) = -6.52$, $p < .01$; hyperactivity/impulsivity levels: control, 54.4 ± 2.3; ADHD, 82.3 ± 4.0; $t(59) = -5.56$, $p < .01$].

Behavioral performance in the T-maze task

The reaction time (RT) data for the T-maze task were analyzed for 93 children (54 controls and 39 with ADHD); the data of 12 children were excluded due to missing behavioral data (from either one or both reward conditions). A three-way mixed-design analysis of variance (ANOVA) on RTs with Group (control, ADHD) and Order (money condition first group, points condition first group) as between-subjects factors, and Time on Task (Time 1 = task performed first, Time 2 = task performed second) as a within-subjects factor revealed a main effect of group, with the control group (683 ± 52 ms) being significantly faster to respond than the ADHD group (895 ± 60 ms), $F(1, 89) = 7.15$, $p = .01$, $\eta_p^2 = .07$ (Fig. 2). A main effect of time on task was marginally significant (Time 1, 823 ± 41 ms; Time 2, 754 ± 48 ms), $p = .08$. The interaction of time on task and order was significant, $F(1, 89) = 12.19$, $p < .01$, $\eta_p^2 = .12$, showing that the money condition was performed faster than the points condition, irrespective of condition order. No other effects were significant.

Next, we examined whether participants adjusted their behavior in response to feedback by comparing RTs when they failed to receive reward on the immediately preceding trials with when they received reward. A mixed-design ANOVA on RTs with Group (control, ADHD) and Feedback Type on the Previous Trial (reward, no reward) as factors revealed a significant main effect of group, with the control group (683 ± 50 ms) performing significantly faster than the ADHD group (896 ± 59 ms), $F(1, 91) = 7.6$, $p = .01$, $\eta_p^2 = .08$. The same analysis on the RT data separately for the money condition revealed only a significant main effect of group (control, 633 ± 48 ms; ADHD, 800 ± 57 ms), $F(1, 91) = 5$, $p = .03$, $\eta_p^2 = .05$ (Fig. 3). For the points condition, the analysis revealed significant main effects of group (control, 732 ± 64 ms; ADHD, 993 ± 75 ms), $F(1, 91) = 7$, $p = .01$, $\eta_p^2 = .07$, and of feedback, such that when participants had failed to

[5] The analyses we conducted were as follows: The continuous variables (age and IQ) were separately correlated with the dependent variables (DVs): overall reward positivity (i.e., the average across the money and points conditions) and the difference in reward positivity amplitudes between the two conditions. This enabled us to examine whether age and IQ correlated with reward positivity amplitude in general or showed different effects, depending on the condition (e.g., whether higher IQs correlated with a larger reward positivity to points feedback or with larger reward positivity to money feedback). Because age and IQ were not correlated with the DVs, we did not use either of these variables as a covariate in subsequent statistical analyses. Similarly, for the categorical variable (sex), a simple univariate analysis of variance was performed on the overall reward positivity amplitude and on the difference in reward positivity amplitude between the two conditions. Again, because sex did not interact with either DV, it was not used as a covariate.

Table 1 Participant demographic information for typically developing children (controls) and children with attention deficit hyperactivity disorder (ADHD)

	Controls			ADHD		
	Mean	*SD*	Range	Mean	*SD*	Range
	n = 58			*n* = 47		
Sex (M:F)	35:23			34:13		
Age (years)	9.91	1.57	8–13	10.02	1.65	8–13
	n = 56			*n* = 41		
FSIQ (standard score)	119	13.14	91–153	113	12.92	83–143
Inattention (*t* score)	47.57	6.59	39–63	82.76	10.03	58–105
Hyperactivity/impulsivity (*t* score)	49.55	7.67	40–69	85.34	15.76	48–116
	Number of Participants			Number of Participants		
Learning disability (Reading:Math)	3:5			4:13		
Medication (current)	0			23		
ODD	2			30		
CD	0			3		
MDE (both present and past)	2			5		
GAD	1			3		

The full-scale intelligence quotient (FSIQ) is based on the WASI full score. Inattention and hyperactivity/impulsivity scores are based on the Conners scale. Learning disability was estimated on the basis of the WASI full scale and WIAT-II scores separately for the reading and math scores (excluding five children in the control group, who were identified with learning disorders and who were involved in a language immersion or English-as-a-second-language program at the time of experimental testing). ODD = oppositional defiant disorder, CD = conduct disorder, MDE = major depressive episode (present and past), GAD = general anxiety disorder

receive points on the previous trial they responded more slowly on the current trial (900 ± 57 ms) than when they had received points on the previous trial (825 ± 48 ms), $F(1, 91) = 3.8$, $p = .05$, $\eta_p^2 = .04$. The interaction of group and previous feedback was not significant.

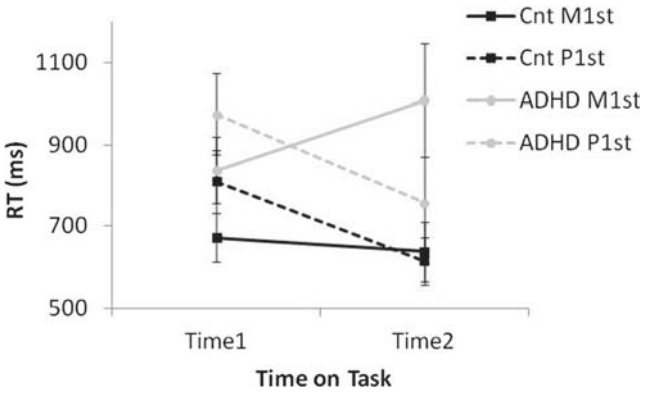

Fig. 2 Reaction times (RTs) for the task that was performed first (Time 1) and second (Time 2), shown separately for typically developing children (Cnt, black) and children with attention deficit hyperactivity disorder (ADHD, gray), which are divided further into children who performed the money condition first, followed by the points condition (M1st), and children who performed the points condition first, followed by the money condition (P1st). The *y*-axis indicates RT in milliseconds. Cnt M1st = children in the control group who performed the money condition first; ADHD M1st = children with ADHD who performed the money condition first; Cnt P1st = children in the control group who performed the points condition first; ADHD P1st = children with ADHD who performed the points condition first. For instance, Time 1 shows RTs for the money condition for the Cnt M1st group, and for the points condition for the Cnt P1st group. Error bars indicate standard errors of the means.

Electrophysiological results

The reward positivity was maximal at channel FCz for both the control and ADHD groups, averaged across money and points conditions and separately for the two conditions (see

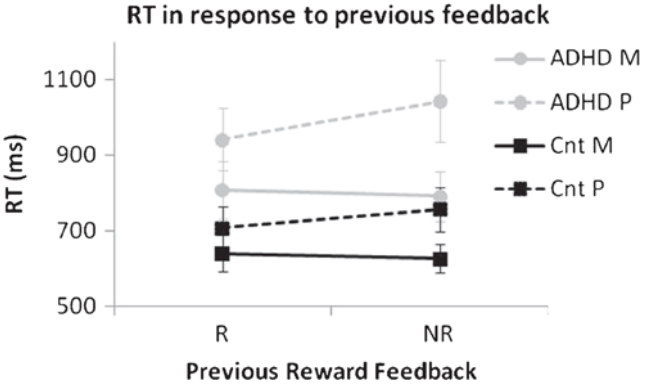

Fig. 3 Reaction times (RTs) on the current trials in response to the feedback received on the previous trials for the control group (Cnt) and the attention deficit hyperactivity disorder group (ADHD). ADHD M = the money condition for the ADHD group; ADHD P = points condition for the ADHD group; Cnt M = money condition for the control group; Cnt P = points condition for the control group. The *x*-axis indicates whether the previous trial provided reward (R, left) or no reward (NR, right). Error bars indicate standard errors of the means.

Figs. 4d–f)—except for the points condition for the ADHD group, in which the component was maximal at channel Cz but was not statistically different from FCz ($p > .05$) (Fig. 4f, right column). Inspection of the ERPs in Fig. 4(a–c) reveals a sequence of negative, positive, and negative deflections in the raw ERP occurring at approximately 150, 200, and 300 ms postfeedback, respectively, consistent with previous observations of feedback ERPs in this age group (Holroyd, Baker, et al., 2008a; Lukie, Montazer-Hojat, & Holroyd, in press).

On the basis of our previous study (Holroyd, Baker, et al., 2008a), our a priori prediction was that reward positivity amplitude would be larger in the money condition than in the points condition for the ADHD group, but not for the control group. A paired t test revealed that reward positivity amplitude was significantly larger in the money condition ($-5.7 \pm 0.8\,\mu V$) than in the points condition ($-3.9 \pm 0.7\,\mu V$) for the ADHD group, $t(46) = -2.6$, $p = .01$, Cohen's $d = 0.36$, whereas reward positivity amplitudes for the control group were similar between the two conditions ($p = .58$), confirming the prediction. Furthermore, when comparing across groups, reward positivity amplitudes in the money condition were similar between groups ($p = .4$), whereas reward positivity amplitudes in the points condition were significantly reduced for the ADHD group ($-3.9 \pm 0.7\,\mu V$) as compared to the control group ($-6.2 \pm 0.4\,\mu V$), $t(103) = -3.0$, $p < .01$, Cohen's $d = 0.58$ (Fig. 5). These results confirm that children with ADHD exhibit reduced reward positivity amplitudes to feedback indicating points, relative to feedback indicating monetary rewards, and that the difference in reward positivity amplitudes between children with ADHD and typically developing

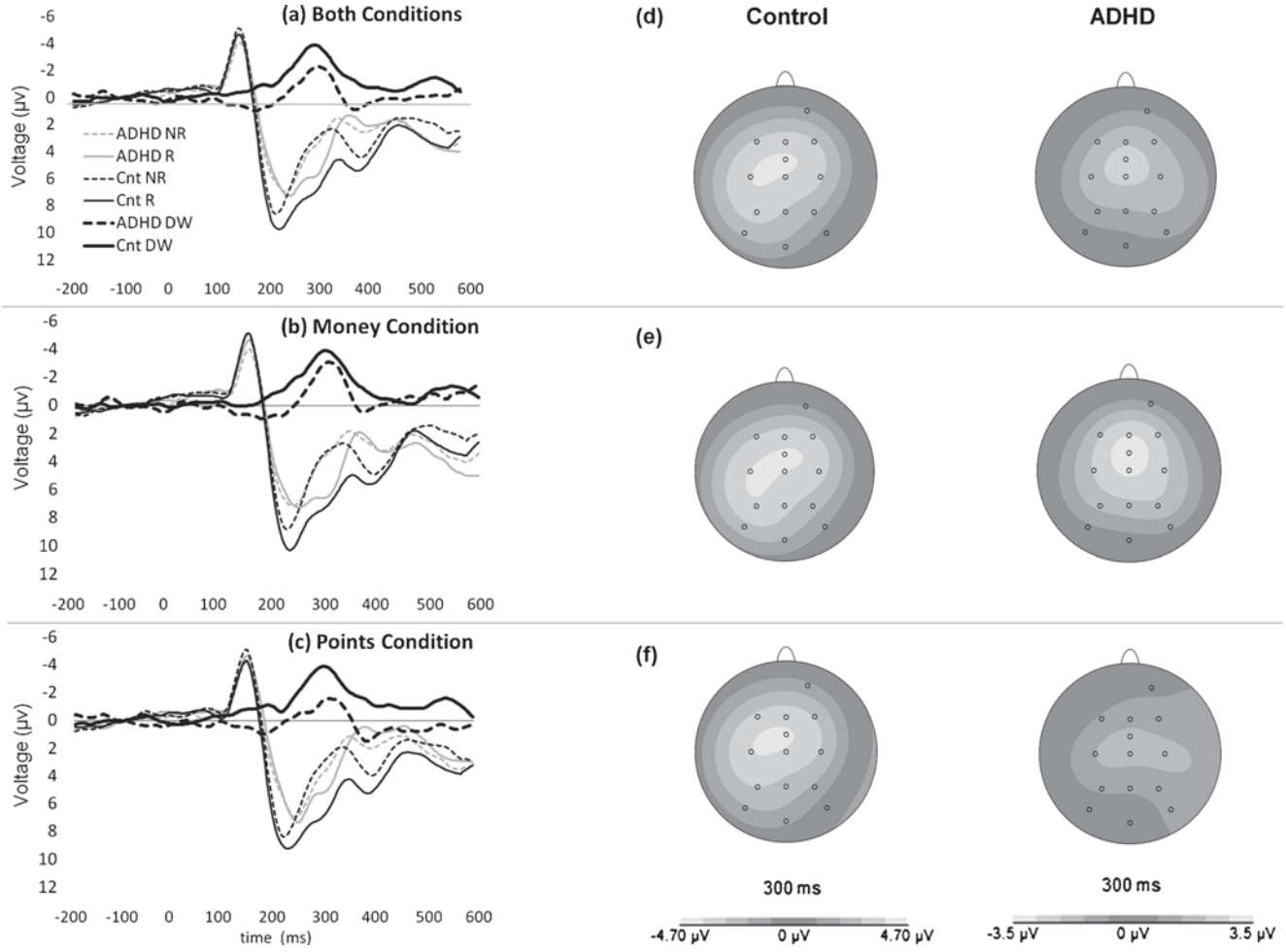

Fig. 4 Event-related brain potentials (ERPs) elicited by reward and no-reward feedback stimuli and associated scalp distributions for the children with attention deficit hyperactivity disorder (ADHD) and the typically developing children. Left column: ERPs recorded at channel FCz averaged (a) across the money and points conditions, (b) for the money condition only, and (c) for the points condition only. Difference waves are shown in thick black and denoted by ADHD DW and Cnt DW for the ADHD and control groups, respectively. ADHD NR and ADHD R correspond to the ERPs for the no-reward and reward conditions, respectively, for the ADHD group. Cnt NR and Cnt R correspond to the ERPs for the no-reward and reward conditions, respectively, for the control group. 0 on the x-axis corresponds to time of feedback onset, and negative is plotted up by convention. Middle and right columns: Scalp voltage maps associated with the peak values of the difference waves at 300 ms following feedback onset (d) across both reward conditions, (e) for the money condition alone, and (f) for the points condition alone, provided separately for the control (middle column) and ADHD (right column) groups.

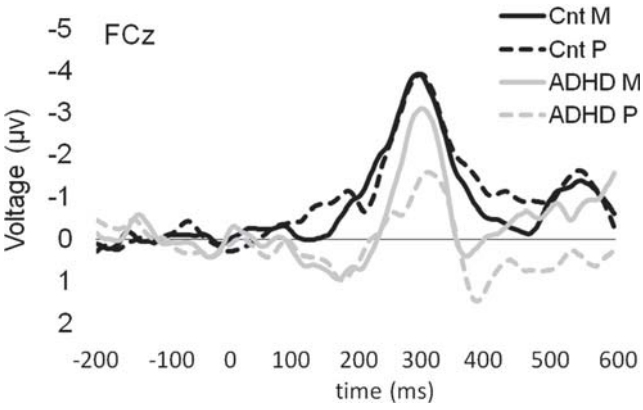

Fig. 5 Reward positivity amplitudes for the money (solid lines) and points (dashed lines) conditions for the control (black lines) and the attention deficit hyperactivity disorder (ADHD; gray lines) groups, measured at channel FCz. Negative is plotted up by convention. 0 on the *x*-axis corresponds to time of feedback onset.

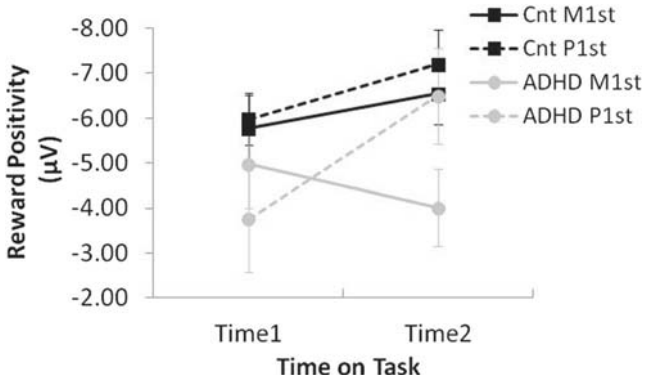

Fig. 6 Reward positivity amplitudes evaluated at channel FCz for the task that was performed first (Time 1) and second (Time 2), separately for the children with and without attention deficit hyperactivity disorder (ADHD), for children who performed the money condition first followed by the points condition (M1st), and for children who performed the points condition first followed by the money condition (P1st). The *y*-axis indicates reward positivity amplitude. Cnt M1st = children in the control group who performed the money condition first; ADHD M1st = children with ADHD who performed the money condition first; Cnt P1st = children in the control group who performed the points condition first; ADHD P1st = children with ADHD who performed the points condition first. For instance, Time 1 shows the reward positivity amplitude for the money condition for the Cnt M1st group, but for the points condition for the Cnt P1st group. Negative is plotted up by convention. Error bars indicate standard errors of the means.

children is mainly due to the smaller reward positivity associated with points feedback for the children with ADHD.

The results were further analyzed with an exploratory three-way mixed-design ANOVA on reward positivity amplitude with Group (control, ADHD) and Order (money condition first group, points condition first group) as between-subjects factors, and Time on Task (Time 1 = task performed first, Time 2 = task performed second) as a within-subjects factor (Fig. 6). The main effects of group (control, –6.4 ± 0.5 μV; ADHD, –4.8 ± 0.5 μV), $F(1, 101) = 4.7$, $p = .03$, $\eta_p^2 = .04$, and of time on task (Time 1, –5.1 ± 0.4 μV; Time 2, –6.1 ± 0.4 μV), $F(1, 101) = 4.3$, $p = .04$, $\eta_p^2 = .04$, were statistically significant. In addition, the interaction of time on task and order was significant, $F(1, 101) = 5.3$, $p = .02$, $\eta_p^2 = .05$, and the three-way interaction of group, order, and time on task was marginally significant, $F(1, 101) = 3.3$, $p = .07$, $\eta_p^2 = .03$. No other effects were significant. To explore this three-way interaction, we conducted separate mixed-design ANOVAs with order and time on task for the two groups, revealing only a marginally significant effect of time on task for the control group (Time 2 > Time 1, $p = .09$), and a significant interaction of order and time on task for the ADHD group, $F(1, 45) = 6.8$, $p = .01$, $\eta_p^2 = .13$. This result indicates that both money and points feedback stimuli elicited a relatively large reward positivity in typically developing children, whereas money feedback stimuli but not points feedback stimuli, irrespective of task order, elicited a relatively large reward positivity in children with ADHD.

For exploratory purposes, we also investigated whether the "raw" ERPs to the positive and negative reward feedback were different for the two groups. A mixed-design ANOVA on the ERP mean amplitudes associated with positive and negative feedback with Group (control, ADHD) and Feedback (reward, no reward) as factors, collapsing across conditions (money, points), revealed a significant main effect of feedback, with the reward feedback ERP (5.7 ± 0.6 μV) being more positive than the no-reward feedback ERP (4.3 ± 0.5 μV), $F(1, 103) = 27.4$, $p < .01$, $\eta_p^2 = .21$, no significant main effect of group, and a significant interaction of group and feedback, $F(1, 103) = 8.5$, $p < .01$, $\eta_p^2 = .08$ (Fig. 4a). Separate *t* tests on the reward and no-reward feedback ERPs (collapsing across money and points) revealed that the interaction was driven mainly by the reward feedback ERP, which showed a trend toward being more positive for the control group (6.8 ± 0.8 μV) than for the ADHD group (4.5 ± 0.9 μV), $t(103) = 1.9$, $p = .06$, Cohen's $d = 0.38$, as opposed to the no-reward feedback ERPs, which were similar between groups, $p = .5$.

We followed up with separate exploratory analyses for the money and points conditions. A mixed-design ANOVA on ERP amplitudes in the money condition with Group (control, ADHD) and Feedback (reward, no reward) as factors (Fig. 4b) revealed a significant main effect of feedback (reward, 6.0 ± 0.6 μV; no reward, 4.5 ± 0.6 μV), $F(1, 103) = 21.5$, $p < .01$, $\eta_p^2 = .17$, no main effect of group, and a marginally significant interaction of group and feedback ($p = .08$). The same analysis for the points condition (Fig. 4c) revealed a significant main effect of feedback (reward, 5.3 ± 0.6 μV; no reward, 4.1 ± 0.5 μV), $F(1, 103) = 14.2$, $p < .01$, $\eta_p^2 = .12$, no main effect of group, and a significant interaction of group and feedback, $F(1, 103) = 8.4$, $p = .01$, $\eta_p^2 = .08$. Follow-up *t* tests revealed that this significant interaction was driven by the reward feedback ERPs being more positive for the control group (6.6 ± 0.8 μV) than for the ADHD group (4.0 ± 0.9 μV),

$t(103) = 2.2$, $p = .03$, Cohen's $d = 0.43$, rather than by the no-reward feedback ERPs. These results indicate that neural activity associated with receiving reward, but not with failing to receive one, appeared to drive the group difference, especially for the points condition.

Correlations between RTs and reward positivity amplitude

We also examined whether reward positivity amplitude correlated with RTs within and across groups. RTs and reward positivity amplitude were inversely correlated across participants across groups (larger reward positivities were associated with faster RTs) in the points condition ($N = 93$, Pearson's $r = .33$, $p < .01$) but not in the money condition ($p > .4$). When the two groups were analyzed separately, the correlation between RT and reward positivity amplitude in the points condition remained significant for the control group ($N = 54$, Pearson's $r = .33$, $p = .01$) and showed a trend for the ADHD group ($N = 39$, Pearson's $r = .24$, $p = .13$), indicating that the overall correlation was not driven entirely by the control group.

Correlations between ADHD symptoms and reward positivity amplitude

To examine whether variance in ADHD symptomatology was predictive of reward positivity amplitude, we correlated the parents' ratings on the Conners Scales of Inattention and Hyperactivity/Impulsiveness with reward positivity amplitudes in the money and points conditions. The data of eight children were excluded from this analysis: The Conners scores from two children were missing (one control), and scores from six children (one control) yielded "probably invalid" positive or negative impressions (indicating that parental/caregiver reports of their children's behavior were extremely skewed relative to normal). Therefore, a total of 97 children (56 control) were included in this correlation analysis. Inattention and hyperactivity/impulsivity scores were significantly positively correlated with each other (Pearson's $r = .79$, $p < .01$) and with reward positivity amplitude in the points condition (inattention, Pearson's $r = .21$, $p = .04$; hyperactivity/impulsivity, Pearson's $r = .29$, $p < .01$), but not in the money condition, indicating that higher inattention and hyperactivity/impulsivity levels are associated with reduced reward positivity amplitudes to points feedback. To explore these results further, for each participant we computed the difference in reward positivity amplitudes between the money and points conditions, yielding a measure of the relative difference in reward positivity amplitudes across conditions, and correlated this measure with ADHD symptom levels (Fig. 7). This analysis revealed that whereas the inattention scores were not correlated with the difference in reward positivity amplitude across conditions ($p = .14$), the hyperactivity/impulsivity scores were negatively correlated with the difference in reward positivity amplitude (Pearson's $r = –.23$, $p = .03$): Higher hyperactivity/impulsivity levels were associated with larger (more negative) reward positivity amplitudes in the money condition than in the points condition. When restricted to the data of children with ADHD, the correlations of the hyperactivity/impulsivity scores with reward positivity amplitude in the points condition (Pearson's $r = .29$, $p = .07$) and with the difference in reward positivity amplitudes between the money and points conditions (Pearson's $r = –.24$, $p = .14$) were marginally statistically significant, indicating that the correlations were not entirely driven by the overall group difference. These results are in line with the previous finding that the group difference in reward positivity amplitudes was driven by the points condition, but not by the money condition.

Medication status, ODD, and LD effects on reward positivity amplitude

We checked whether the group difference observed in reward positivity amplitudes was due to the medication status of the ADHD group. Out of the 47 children diagnosed with ADHD, 18 reported never having taken medications for their diagnosis (*never-medicated group*), 23 reported being currently on medications (*medicated group*), and six reported a history of taking ADHD-related medications. Separate *t* tests on overall reward positivity amplitudes (collapsing across the money and points conditions), reward positivity amplitude in the money condition, and reward positivity amplitude in the points condition for the medicated versus the never-medicated groups revealed no significant effects of medication.

A comparable analysis of the effect of ODD diagnosis on reward positivity amplitudes for the children with ADHD (17 children not diagnosed with ODD vs. 30 diagnosed; only two children in the control group were diagnosed with ODD) revealed no significant effect of ODD diagnosis on overall reward positivity amplitude, reward positivity amplitude in the money condition, or reward positivity amplitude in the points condition.

We also found a significant effect of reading LD on overall reward positivity amplitude, such that the reward positivity amplitude was reduced in children with reading LD ($n = 7$) as compared to children without reading LD ($n = 98$) ($p = .05$). Note, however, that we also found an interaction between reading LD and group, indicating that the reduction in reward positivity amplitude associated with reading LD for the ADHD group was unlikely to be due only to LD. Because the sample sizes of the children with reading LDs in this study were very small (three children with ADHD and four typically developing children), the possible impact of reading LD on the reward positivity awaits further research. Nevertheless, as a check we reanalyzed our a priori predictions excluding the

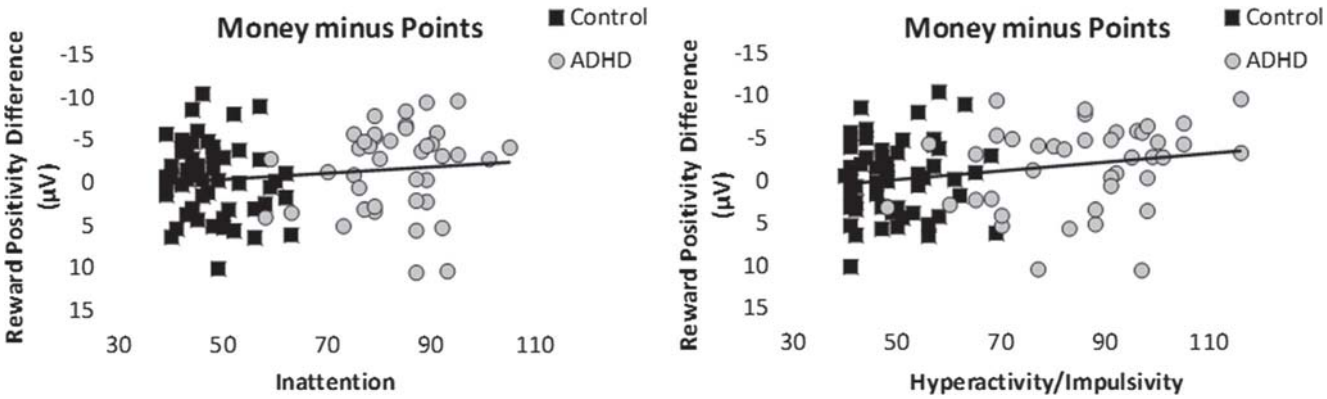

Fig. 7 Correlations between the inattention (left panel) and hyperactivity/impulsivity (right panel) scores, as measured by the Conners behavioral rating index for parents and the difference in reward positivity amplitudes measured at channel FCz between the money and points conditions. More-negative values on the *y*-axis indicate that the reward positivity to money was larger (more negative) than that to points, whereas more-positive values indicate that the reward positivity to points was larger than that to money. The black squares represent the data of the typically developing children, and shaded circles represent the data of the children with attention deficit hyperactivity disorder (ADHD).

data of these seven children, and the findings remained statistically significant ($p < .05$).

Discussion

We have recently proposed that the ACC functions to motivate sequences of goal-directed actions (Holroyd & Yeung, 2012). This theory, which is supported by a wealth of neuroimaging, neurophysiological, and lesion data in both humans and nonhuman animals (Holroyd & McClure, 2014; Holroyd & Yeung, 2012), is based on the proposal that a neural mechanism for task selection learns task values according to the principles of HRL (Botvinick et al., 2009). By this view, ACC determines task values on the basis of reward signals carried there by the midbrain dopamine system (Holroyd & Coles, 2002; Holroyd & Yeung, 2012), selects tasks on the basis of these learned values, and directs DLPFC to apply the appropriate level of top-down control needed to support the desired level of performance (Umemoto & Holroyd, 2014). This theoretical framework holds ACC responsible for executing extended behaviors such as whether and how enthusiastically a participant decides to take part in a psychology experiment (Holroyd & Yeung, 2012). Consistent with this proposal, substantial empirical evidence (Bush, 2009, 2011; but see also Nigg & Casey, 2005; Sonuga-Barke, 2002, 2003) and the results of computational simulations (Cockburn & Holroyd, 2010; Silvetti et al., 2013) suggest that impairments related to control, motivation, and reinforcement learning in ADHD result partly from disruption of the ACC–DA system. These observations converge on a suggestion that studies of the reward positivity, a putative index of the reinforcement learning function mediated by the ACC–DA interface (Holroyd & Coles, 2002; Walsh & Anderson, 2012; but see J. M. Carlson et al., 2011; Foti et al., 2011), can provide insight into the deficits in motivational control associated with childhood ADHD.

Here we found that when the reward positivity was averaged across reward conditions, the reward positivity amplitude for children with ADHD was smaller than that for typically developing children, which in that age group is comparable to that for adults (Lukie et al., in press). Furthermore, consistent with our predictions, when averaged separately according to the money and points conditions, the sizes of reward positivity were comparable between the two reward conditions for the control group, whereas the reward positivity amplitude in the points condition was significantly smaller than that in the money condition for the ADHD group (although the interaction between group and condition was not statistically significant). Furthermore, the sizes of the reward positivity in the money condition were also comparable between groups, whereas the reward positivity amplitude in the points condition was significantly reduced for the ADHD group relative to the control group. These results were unrelated to the participants' IQ levels, current medication status, presence of comorbid disorders (i.e., ODD), or learning disabilities. Furthermore, reward positivity amplitude was positively correlated with parent and caregiver ratings of both inattention and hyperactivity/impulsivity levels for the points condition, but not for the money condition, suggesting that increasing severity of ADHD symptoms are associated with decreased sensitivity to the acquisition of points.

These results indicate that—whereas both abstract, nonsalient rewards, such as feedback indicating points, and motivationally salient rewards, such as feedback indicating monetary gains, elicit a relatively large reward positivity in typically developing children—only salient rewards, but not abstract rewards, elicit a relatively large reward positivity in children with ADHD. In our previous study, we concluded that children with ADHD are more sensitive to salient than to abstract rewards (Holroyd et al., 2008a). Our present results

corroborate the previous findings and further indicate that the salient rewards "normalize" a relative insensitivity to abstract rewards (since the latter rather than the former was what actually differed between groups). That said, we emphasize that we have only investigated two points along the subjective value functions for the two groups (e.g., Trepel, Fox, & Poldrack, 2005), and it remains possible that especially prized rewards (such as an exciting toy) could elicit even *larger* reward positivities in children with ADHD relative to typically developing children. Such flexibility in reward valuations is suggested by their sometimes elevated behavioral sensitivity to rewards, since the motivational effect of reinforcement is larger for children with ADHD than for typically developing children (C. L. Carlson & Tamm, 2000; Luman, Oosterlaan, & Sergeant, 2005; McInerney & Kerns, 2003). Social approval also appears to be a more salient reinforcer for children with ADHD than for typically developing children (e.g., Kohls et al., 2009), although not always (Demurie, Roeyers, Baeyens, & Sonuga-Barke, 2011).

Such flexibility in reward valuation also appears to be at play in special populations of adults. For example, the reward positivity amplitude elicited by monetary reward is reduced in substance-dependent individuals relative to controls (Baker, Stockwell, Barnes, Haesevoets, & Holroyd, 2014; Baker, Stockwell, Barnes, & Holroyd, 2011), and this insensitivity is normalized by feedback stimuli indicating acquisition of drug rewards (viz. cigarette puffs; Wood, Baker, & Holroyd, 2013). Problem gamblers in a guessing task also produce especially large reward positivities to feedback indicating monetary gains, which presumably for them is relatively potent (Hewig et al., 2010). Likewise, high temporal discounters produce larger reward positivities to immediate rewards than do low temporal discounters (Cherniawsky & Holroyd, 2013).

The terms "abstract," "salient," "motivation," and so on are themselves imprecise and are sources of confusion. For example, it might be suggested that monetary rewards are more "immediate" than points rewards, rather than more salient per se. Arguing against this possibility, participants saw abstract feedback stimuli at the time of reward presentation (i.e., images of an apple and an orange) in *both* of the reward conditions. The monetary rewards were indeed more immediate than points feedback, since physical coins were presented to the participant following each block of trials in the former case, but not in the latter. However, the points feedback also elicited a reward positivity (albeit reduced in size), despite the fact that the children understood that no physical winnings would *ever* actually be delivered. In our view, the terms "abstract" and "salient" characterize this dimension of the feedback stimuli more accurately than do "immediate" and "delayed."

As well, the fact that children with ADHD responded more strongly to salient rewards is also compatible with the idea that children with ADHD need more reinforcement than do controls to achieve equivalent levels of performance (Slusarek, Velling, Bunk, & Eggers, 2001; cf. Demurie et al., 2011). We suggest that the larger reward positivity to monetary rewards reflects the role of ACC in "motivating" performance. Larger reward positivity amplitudes were also associated with faster RTs, but only in the points condition (because reward positivity amplitudes were uniformly large across participants in the money condition). These observations suggest a common process underlying both fast responses and large reward positivities during high task engagement: For the less-motivated participants, the slow responses and smaller reward positivities in the points condition were normalized by the motivating effect of monetary rewards in the money condition. Although other accounts are possible, our suggestion is in line with the fact that the children with ADHD were slower to respond overall than were the typically developing children, consistent with previous reports (Leth-Steensen, King Elbaz, & Douglas, 2000) and with observations that performance incentives can decrease RTs in this population (e.g., Andreou et al., 2007; Kuntsi, Wood, Van Der Meere, & Asherson, 2009).

As we discussed in the introduction, several neurobiological theories of ADHD have proposed that the motivational deficits associated with the disorder stem from underlying disruption to DA system function. In particular, Cockburn and Holroyd (2010) compared the predictions of several theories by systematically manipulating the sizes of the positive and negative phasic DA signals in a computational simulation to evaluate the effects of disrupted DA processing on reinforcement learning. Their simulations suggested that, relative to that of typically developing children, the reinforcement system in children with ADHD responds disproportionately more strongly to rewards than to nonrewards[6] (though both signals may be either larger or smaller, relative to those of typically developing children). We also explored this prediction by separately averaging the ERPs to reward and no-reward feedback and found that whereas the ERP responses to no reward were relatively constant across groups, the ERP responses to positive outcomes were strongly modulated by the saliency of the reward for children with ADHD: Abstract rewards such as points generated a relatively weak neural response, and salient rewards such as money normalized this outcome, producing a stronger response. This observation seems to counter the prediction of Cockburn and Holroyd (2010), which was that the positive reward signal should be larger for children with ADHD than for typically developing children when—as occurred in the present experiment—the negative reward signal is the same size across groups.

[6] Tentative support for this proposal is provided by an analysis of the probabilistic selection task results (see the supplementary online materials), which suggests that more children with ADHD exhibited a bias to learning from positive feedback than a bias to learning from negative feedback, whereas the typically developing children were about equally divided between positive and negative learners.

However, in line with our discussion above, it is also possible that the rewards in that study sampled an even more valuable part of the value function for both groups. We speculate that especially rewarding outcomes, like a desirable toy, could increase the amplitude of the reward positivity even further. In this case, the difference between positive and negative feedback would be larger for the children with ADHD than for the typically developing children.

It is also likely that other mechanisms besides reward processing are involved in the underlying dysfunction of ADHD. For example, the dual-pathway model holds that at least two independent, DA-related neural pathways contribute to the disorder (Sonuga-Barke, 2002, 2003), and an integrative theory involving multiple neural pathways emphasizes PFC dysregulation (Nigg & Casey, 2005). Although the present study does not directly speak to other pathways involved, future research should consider the neural mechanisms underlying different ADHD subtypes (i.e., inattentive, hyperactive/impulsive, and combined), which appear to constitute separate disorders with distinct impairments (e.g., Barkley, 1997; Milich, Balentine, & Lynam, 2001).

Our present results are generally consistent with the findings of our previous study (Holroyd et al., 2008a). In that study, children performed the same simple pseudo-reinforcement-learning task utilized here, wherein they navigated through a virtual maze to find monetary rewards. But unlike our present study, in which the conditions were counterbalanced, in the previous study the accumulated monetary rewards were placed conspicuously on the table midway through the experiment for all of the participants. Children with ADHD showed a statistically significant increase in reward positivity amplitude following the midway monetary payment, whereas typically developing children showed a nonsignificant decrease in reward positivity amplitude, resulting in a significant interaction of group and time (before vs. after the midway payment).

Nevertheless, this finding was qualified by the fact that it was not predicted a priori, increasing the probability that it was a statistical fluke. In the present study, we addressed this concern by replicating the finding, and further improved on the previous study design in several ways. First, we manipulated the saliency of the rewards such that all of the children performed the task in two counterbalanced conditions that differed in reward saliency: In the "points" condition, the reward feedback indicated a gain of 5 points and no-reward feedback 0 points, whereas in the "money" condition, the reward feedback indicated a gain of 5 cents and no-reward feedback 0 cents. Second, we conducted independent diagnostic interviews to confirm the diagnosis of ADHD for children with the disorder, both to exclude children from the control group who might have had the disorder and to assess the presence of common comorbid disorders such as ODD and CD. Finally, for all of the children we assessed the severity of ADHD-related symptoms, learning disabilities, and IQ levels. In contrast to the previous results, however, the typically developing children in the present study showed an increased reward positivity amplitude in the second half of the experiment relative to the first half of the experiment, irrespective of the order of the reward conditions. This discrepancy may have been due to the small sample size of the control group in the previous study ($N = 14$) as compared to the present study ($N = 58$), or perhaps to the length of the experiment, which was twice as long in the present as in the previous study. This discrepancy would need to be examined in future investigations.

Our results are generally consistent with the existing literature on altered midbrain DA function (Levy, 1991; Luman et al., 2005; Luman et al., 2010; Swanson et al., 2000; Swanson et al., 2007; see Tripp & Wickens, 2009, for a review), atypical ACC structure and function (Bush, 2009, 2011), and abnormal behavioral sensitivity to rewards (C. L. Carlson & Tamm, 2000; Luman et al., 2005; McInerney & Kerns, 2003) in children with ADHD as compared to typically developing children. The findings dovetail with previous research showing that motivationally salient rewards can normalize the impaired cognitive functions associated with ADHD (Dovis et al., 2012; Kohls et al., 2009; Konrad et al., 2000; Rubia et al., 2009; Shiels et al., 2008), and with the proposal that ACC is responsible for motivating task selection and execution (Holroyd & Yeung, 2012). According to this view, an impaired ability to learn and use task values for motivating task-relevant behaviors due to disruption of the ACC–DA interface, as suggested by the abnormal reward positivity, may underlie the motivational and control deficits observed in childhood ADHD (Bush, 2009; Silvetti et al., 2013).

Author note All authors are affiliated with the Department of Psychology, University of Victoria. This research was supported by Canadian Institutes of Health Research Operating Grant No. 86467. We thank Julie Chang, Erin Eadie, Laurie Fitzgerald, Jeff Frazer, Sarah Hutchinson, Julie Irwin, Celine Koryzma, Jennifer MacSween, Kate Randall, Karley Talbot, and Emanuela Yeung for their assistance in this project.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Association.

Andreou, P., Neale, B. M., Chen, W. A. I., Christiansen, H., Gabriels, I., Heise, A., & Kuntsi, J. (2007). Reaction time performance in ADHD: Improvement under fast-incentive condition and familial effects. *Psychological Medicine, 37,* 1703–1715.

Arnsten, A. F. (2006). Stimulants: Therapeutic actions in ADHD. *Neuropsychopharmacology, 31,* 2376–2383. doi:10.1038/sj.npp.1301164

Baker, T. E., & Holroyd, C. B. (2009). Which way do I go? Neural activation in response to feedback and spatial processing in a virtual T-maze. *Cerebral Cortex, 19,* 1708–1722.

Baker, T. E., Stockwell, T., Barnes, G., Haesevoets, R., & Holroyd, C. B. (2014). *Top-down versus bottoms-up! Intermediate phenotypes for cognitive control and personality mediate the expression of dopamine genes in addiction*. Manuscript submitted for publication.

Baker, T. E., Stockwell, T., Barnes, G., & Holroyd, C. B. (2011). Individual differences in substance dependence: At the intersection of brain, behavior and cognition. *Addiction Biology, 16,* 458–466.

Banaschewski, T., Becker, K., Scherag, S., Franke, B., & Coghill, D. (2010). Molecular genetics of attention-deficit/hyperactivity disorder: An overview. *European Child and Adolescent Psychiatry, 19,* 237–257.

Barkley, R. A. (1997). *ADHD and the nature of self-control*. New York, NY: Guilford Press.

Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition, 50,* 7–15. doi:10.1016/0010-0277(94)90018-3

Bechara, A., Tranel, D., & Damasio, H. (2000). Characterization of the decision-making deficit of patients with ventromedial prefrontal cortex lesions. *Brain, 123,* 2189–2202.

Botvinick, M. M. (2012). Hierarchical reinforcement learning and decision making. *Current Opinion in Neurobiology, 22,* 956–962. doi:10.1016/j.conb.2012.05.008

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108,* 624–652. doi:10.1037/0033-295X.108.3.624

Botvinick, M. M., Niv, Y., & Barto, A. C. (2009). Hierarchically organized behavior and its neural foundations: A reinforcement learning perspective. *Cognition, 113,* 262–280.

Bush, G. (2009). Dorsal anterior midcingulate cortex: Roles in normal cognition and disruption in attention-deficit/hyperactivity disorder. In B. A. Vogt (Ed.), *Cingulate neurobiology and disease* (pp. 245–274). Oxford, UK: Oxford University Press.

Bush, G. (2011). Cingulate, frontal, and parietal cortical dysfunction in attention-deficit/hyperactivity disorder. *Biological Psychiatry, 69,* 1160–1167.

Carlson, J. M., Foti, D., Mujica-Parodi, L. R., Harmon-Jones, E., & Hajcak, G. (2011). Ventral striatal and medial prefrontal BOLD activation is correlated with reward- related electrocortical activity: A combined ERP and fMRI study. *NeuroImage, 57,* 1608–1616.

Carlson, C. L., & Tamm, L. (2000). Responsiveness of children with attention deficit-hyperactivity disorder to reward and response cost: Differential impact on performance and motivation. *Journal of Consulting and Clinical Psychology, 68,* 73–83.

Chamberlain, S. R., Robbins, T. W., Winder-Rhodes, S., Müller, U., Sahakian, B. J., Blackwell, A. D., & Barnett, J. H. (2011). Translational approaches to frontostriatal dysfunction in attention-deficit/hyperactivity disorder using a computerized neuropsychological battery. *Biological Psychiatry, 69,* 1192–1203.

Cherniawsky, A. S., & Holroyd, C. B. (2013). High temporal discounters overvalue immediate rewards rather than undervalue future rewards: An event-related brain potential study. *Cognitive, Affective, & Behavioral Neuroscience, 13,* 36–45. doi:10.3758/s13415-012-0122-x

Cockburn, J., & Holroyd, C. B. (2010). Focus on the positive: Computational simulations implicate asymmetrical reward prediction error signals in childhood attention-deficit/hyperactivity disorder. *Brain Research, 1365,* 18–34. doi:10.1016/j.brainres.2010.09.065

Cohen, J. D., Dunbar, K., & McClelland, J. L. (1990). On the control of automatic processes: A parallel distributed processing account of the Stroop effect. *Psychological Review, 97,* 332–361. doi:10.1037/0033-295X.97.3.332

Cragg, S. J., & Rice, M. E. (2004). Dancing path the DAT at the DA synapse. *Trends in Neurosciences, 27,* 270–277.

Demurie, E., Roeyers, H., Baeyens, D., & Sonuga–Barke, E. (2011). Common alterations in sensitivity to type but not amount of reward in ADHD and autism spectrum disorders. *Journal of Child Psychology and Psychiatry, 52,* 1164–1173.

Douglas, V. I., & Parry, P. A. (1994). Effects of reward and nonreward on frustration and attention in attention deficit disorder. *Journal of Abnormal Child Psychology, 22,* 281–301.

Dovis, S., Van der Oord, S., Wiers, R. W., & Prins, P. J. M. (2012). Can motivation normalize working memory and task persistence in children with attention-deficit/hyperactivity disorder? The effects of money and computer-gaming. *Journal of Abnormal Child Psychology, 40,* 669–681.

Faraone, S. V., Doyle, A. E., Mick, E., & Biederman, J. (2001). Hierarchical control over effortful behavior by rodent medial frontal cortex: A computational model. *American Journal of Psychiatry, 158,* 1052–1057.

Foti, D., Weinberg, A., Dien, J., & Hajcak, G. (2011). Event–related potential activity in the basal ganglia differentiates rewards from nonrewards: Temporospatial principal components analysis and source localization of the feedback negativity. *Human Brain Mapping, 32,* 2207–2216.

Frank, M. J., Santamaria, A., O'Reilly, R. C., & Willcutt, E. (2007). Testing computational models of dopamine and noradrenaline dysfunction in attention deficit/hyperactivity disorder. *Neuropsychopharmacology, 32,* 1583–1599.

Frank, M. J., Seeberger, L. C., & O'Reilly, R. C. (2004). By carrot or by stick: Cognitive reinforcement learning in Parkinsonism. *Science, 306,* 1940–1943. doi:10.1126/science.1102941

Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2000). *Behavior rating inventory of executive function*. Odessa, FL: Psychological Assessment Resources.

Grace, A. A. (2001). Psychostimulant actions on dopamine and limbic system function: Relevance to the pathophysiology and treatment of ADHD. In M. V. Solanto, A. F. Torrance Arnsten, & F. X. Castellanos (Eds.), *Stimulant drugs and ADHD: Basic and clinical neuroscience* (pp. 134–157). New York, NY: Oxford University Press.

Gratton, G., Coles, M. G. H., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology, 55,* 468–484. doi:10.1016/0013-4694(83)90135-9

Hewig, J., Kretschmer, N., Trippe, R. H., Hecht, H., Coles, M. G. H., Holroyd, C. B., & Miltner, W. H. R. (2010). Hypersensitivity to reward in problem gamblers. *Biological Psychiatry, 67,* 781–783. doi:10.1016/j.biopsych.2009.11.009

Holroyd, C. B. (2004). A note on the oddball N200 and the feedback ERN. In M. Ullsperger & M. Falkenstein (Eds.), Errors, Conflicts, and the Brain: Current Opinions on Performance Monitoring. Leipzig: MPI of Cognitive Neuroscience.

Holroyd, C. B. (2013). Theories of anterior cingulate cortex function: Opportunity cost. *Behavioral and Brain Sciences, 36,* 693–694. doi:10.1017/S0140525X13001052

Holroyd, C. B., Baker, T. E., Kerns, K. A., & Müller, U. (2008a). Electrophysiological evidence of atypical motivation and reward processing in children with attention-deficit hyperactivity disorder. *Neuropsychologia, 46,* 2234–2242. doi:10.1016/j.neuropsychologia.2008.02.011

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review, 109,* 679–709. doi:10.1037/0033-295X.109.4.679

Holroyd, C. B., & Krigolson, O. E. (2007). Reward prediction error signals associated with a modified time estimation task. *Psychophysiology, 44,* 913–917. doi:10.1111/j.1469-8986.2007.00561.x

Holroyd, C. B., & McClure, S. M. (2014). *Hierarchical control over effortful behavior by rodent medial frontal cortex: A computational model*. Manuscript submitted for publication.

Holroyd, C. B., Pakzad–Vaezi, K. L., & Krigolson, O. E. (2008b). The feedback correct–related positivity: Sensitivity of the event–related brain potential to unexpected positive feedback. *Psychophysiology, 45,* 688–697. doi:10.1111/j.1469-8986.2008.00668.x

Holroyd, C. B., & Yeung, N. (2012). Motivation of extended behaviors by anterior cingulate cortex. *Trends in Cognitive Sciences, 16,* 122–128. doi:10.1016/j.tics.2011.12.008

Kohls, G., Herpertz-Dahlmann, B., & Konrad, K. (2009). Hyperresponsiveness to social rewards in children and adolescents with attention-deficit/hyperactivity disorder (ADHD). *Behavioral and Brain Functions, 5,* 20. doi:10.1186/1744-9081-5-20

Kollins, S. H., Lane, S. D., & Shapiro, S. K. (1997). Experimental analysis of childhood psychopathology: A laboratory matching analysis of the behavior of children with attention-deficit hyperactivity disorder. *Psychological Record, 47,* 25–44.

Konrad, K., Gauggel, S., Manz, A., & Schöll, M. (2000). Lack of inhibition: A motivational deficit in children with Attention Deficit/Hyperactivity Disorder and children with traumatic brain injury. *Child Neuropsychology, 6,* 286–296.

Konrad, K., Neufang, S., Hanisch, C., Fink, G. R., & Herpertz-Dahlmann, B. (2006). Dysfunctional attentional networks in children with attention deficit/hyperactivity disorder: Evidence from an event-related functional magnetic resonance imaging study. *Biological Psychiatry, 59,* 643–651.

Kuczenski, R., & Segal, D. S. (1975). Differential effects of D- and L-amphetamine and methylphenidate on rat striatal dopamine biosynthesis. *European Journal of Pharmacology, 30,* 244–251.

Kuntsi, J., Wood, A. C., Van Der Meere, J., & Asherson, P. (2009). Why cognitive performance in ADHD may not reveal true potential: Findings from a large population-based sample. *Journal of the International Neuropsychological Society, 15,* 570.

Leth-Steensen, C., King Elbaz, Z., & Douglas, V. I. (2000). Mean response times, variability, and skew in the responding of ADHD children: A response time distributional approach. *Acta Psychologica, 104,* 167–190.

Levy, F. (1991). The dopamine theory of attention deficit hyperactivity disorder (ADHD). *Australian and New Zealand Journal of Psychiatry, 25,* 277–283.

Li, D., Sham, P. C., Owen, M. J., & He, L. (2006). Meta-analysis shows significant association between dopamine system genes and attention deficit hyperactivity disorder (ADHD). *Human Molecular Genetics, 15,* 2276–2284. doi:10.1093/hmg/ddl152

Lukie, C. N., Montazer-Hojat, S., & Holroyd, C. B. (in press). Developmental changes in the reward positivity: An electrophysiological trajectory of reward processing. *Developmental Cognitive Neuroscience.*

Luman, M., Oosterlaan, J., Knol, D. L., & Sergeant, J. A. (2008). Decision-making in ADHD: Sensitive to frequency but blind to the magnitude of penalty? *Journal of Child Psychology and Psychiatry, 49,* 712–722.

Luman, M., Oosterlaan, J., & Sergeant, J. A. (2005). The impact of reinforcement contingencies on AD/HD: A review and theoretical appraisal. *Clinical Psychology Review, 25,* 183–213. doi:10.1016/j.cpr.2004.11.001

Luman, M., Tripp, G., & Scheres, A. (2010). Identifying the neurobiology of altered reinforcement sensitivity in ADHD: A review and research agenda. *Neuroscience & Biobehavioral Reviews, 34,* 744–754.

McInerney, R. J., & Kerns, K. A. (2003). Time reproduction in children with ADHD: Motivation matters. *Child Neuropsychology, 9,* 91–108.

Milich, R., Balentine, A. C., & Lynam, D. R. (2001). ADHD combined type and ADHD predominantly inattentive type are distinct and unrelated disorders. *Clinical Psychology: Science and Practice, 8,* 463–488.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24,* 167–202. doi:10.1146/annurev.neuro.24.1.167

Miltner, W. H. R., Braun, C. H., & Coles, M. G. H. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a "generic" neural system for error detection. *Journal of Cognitive Neuroscience, 9,* 788–798. doi:10.1162/jocn.1997.9.6.788

Montague, P. R., Hyman, S. E., & Cohen, J. D. (2004). Computational roles for dopamine in behavioural control. *Nature, 431,* 760–767.

Nigg, J. T. (2005). Neuropsychologic theory and findings in attention-deficit/hyperactivity disorder: The state of the field and salient challenges for the coming decade. *Biological Psychiatry, 57,* 1424–1435.

Nigg, J. T. (2006). *What causes ADHD? Understanding what goes wrong and why.* New York, NY: Guildford Press.

Nigg, J. T., & Casey, B. J. (2005). An integrative theory of attention-deficit/hyperactivity disorder based on the cognitive and affective neurosciences. *Development and Psychopathology, 17,* 785–806.

Plitcha, M. M., & Scheres, A. (2014). Ventral–striatal responsiveness during reward anticipation in ADHD and its relation to trait impulsivity in the healthy population: A meta-analytic review of the fMRI literature. *Neuroscience & Biobehavioral Reviews, 38,* 125–134. doi:10.1016/j.neubiorev.2013.07.012

Psychological Corporation. (2002). *Wechsler individual achievement test second edition examiner's manual*. San Antonio, TX: Psychological Corporation.

Quay, H. C. (1997). Inhibition and attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 25,* 7–13.

Reich, W. (2000). Diagnostic Interview for Children and Adolescents (DICA). *Journal of the American Academy of Child and Adolescent Psychiatry, 39,* 59–66.

Roessner, V., Sagvolden, T., DasBanerjee, T., Middleton, F. A., Faraone, S. V., Walaas, S. I., & Bock, N. (2010). Methylphenidate normalizes elevated dopamine transporter densities in an animal model of the attention-deficit/hyperactivity disorder combined type, but not to the same extent in one of the attention-deficit/hyperactivity disorder inattentive type. *Neuroscience, 67,* 1183–1191.

Rubia, K., Cubillo, A., Smoth, A. B., Woodley, J., Heyman, I., & Brammer, M. J. (2010). Disorder-specific dysfunction in right inferior prefrontal cortex during two inhibition tasks in boys with attention-deficit hyperactivity disorder compared to boys with obsessive-compulsive disorder. *Human Brain Mapping, 31,* 287–299.

Rubia, K., Halari, R., Cubillo, A., Mohamman, A.-M., Brammer, M., & Taylor, E. (2009). Methylphenidate normalises activation and functional connectivity deficits in attention and motivation networks in medication-naïve children with ADHD during a rewarded continuous performance task. *Neuropharmacology, 57,* 640–652.

Rubia, K., Halari, R., Smith, A. B., Mohammed, M., Scott, S., Giampietro, V., & Brammer, M. J. (2008). Dissociated functional brain abnormalities of inhibition in boys with pure conduct disorder and in boys with pure attention deficit hyperactivity disorder. *American Journal of Psychiatry, 165,* 889–897. doi:10.1176/appi.ajp.2008.07071084

Sagvolden, T., & Johansen, E. B. (2012). Rat models of ADHD. In C. Stanford & R. Tannock (Eds.), *Behavioral neuroscience of attention deficit hyperactivity disorder and its treatment* (pp. 301–315). Heidelberg, Germany: Springer.

Sagvolden, T., Johansen, E. B., Aase, H., & Russell, V. A. (2005). Adynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behavioral and Brain Sciences, 28,* 397–468.

Sakai, K. (2008). Task set and prefrontal cortex. *Review of Neuroscience, 31,* 219–245.

Schachar, R., & Logan, G. D. (1990). Impulsivity and inhibitory control in normal development and childhood psychopathology. *Developmental Psychology, 26,* 710–720.

Schultz, W. (2002). Getting formal with dopamine and reward. *Neuron, 36,* 241–263.

Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science, 275,* 1593–1599.

Seeman, P., & Madras, B. (2002). Methylphenidate elevates resting dopamine which lowers the impulse-triggered release of dopamine: A hypothesis. *Behavioural Brain Research, 130,* 79–83.

Semrud-Clikeman, M., Pliszka, S. R., Bledsoe, J., & Lancaster, J. (2012). Volumetric MRI differences in treatment naïve and chronically treated adolescents with ADHD-combined type. *Journal of Attention Disorders*. doi:10.1177/1087054712443158

Semrud-Clikeman, M., Pliszka, S. R., Lancaster, J., & Liotti, M. (2006). Volumetric MRI differences in treatment-naive vs. chronically treated children with ADHD. *Neurology, 67,* 1023–1027.

Sergeant, J. A., Geurts, H., Huijbregts, S., Scheres, A., & Oosterlaan, J. (2003). The top and the bottom of ADHD: A neuropsychological perspective. *Neuroscience & Biobehavioral Reviews, 27,* 583–592.

Shenhav, A., Botvinick, M. M., & Cohen, J. D. (2013). The expected value of control: An integrative theory of anterior cingulate cortex function. *Neuron, 79,* 217–240.

Shiels, K., Hawk, L. W., Lysczek, C. L., Tannock, R., Pelham, W. E., Jr., Spencer, S. V., & Waschbusch, D. A. (2008). The effects of incentives on visual–spatial working memory in children with attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology, 36,* 903–913.

Silvetti, M., Wiersema, J. R., Sonuga-Barke, E., & Verguts, T. (2013). Deficient reinforcement learning in medial frontal cortex as a model of dopamine-related motivational deficits in ADHD. *Neural Networks, 46,* 199–209. doi:10.1016/j.neunet.2013.05.008

Slusarek, M., Velling, S., Bunk, D., & Eggers, C. (2001). Motivational effects on inhibitory control in children with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 40,* 355–363.

Sonuga-Barke, E. J. S. (2002). Psychological heterogeneity in AD/HD—A dual pathway model of behavior and cognition. *Behavioral Brain Research, 130,* 29–36.

Sonuga-Barke, E. J. S. (2003). The dual pathway model of AD/HD: An elaboration of neuro-developmental characteristics. *Neuroscience & Biobehavioral Reviews, 27,* 593–604.

Sonuga-Barke, E. J. S., Taylor, E., Sembi, S., & Smith, S. (1992). Hyperactivity and delay aversion—I. The effect of delay on choice. *Journal of Child Psychology and Psychiatry, 33,* 387–398.

Stuss, D. T., & Knight, R. T. (2013). *Principles of frontal lobe function*. New York, NY: Oxford University Press.

Swanson, J. M., Flodman, P., Kennedy, J., Spence, M. A., Moyzis, R., Schuck, S., & Posner, M. (2000). Dopamine genes and ADHD. *Neuroscience and Biobehavioral Reviews, 24,* 21–25.

Swanson, J. M., Kinsbourne, M., Nigg, J., Lanphear, B., Stefanatos, G. A., Volkow, N., & Wadhwa, P. D. (2007). Etiologic subtypes of attention-deficit/hyperactivity disorder: Brain imaging, molecular genetic and environmental factors and the dopamine hypothesis. *Neuropsychological Review, 17,* 39–59.

Trepel, C., Fox, C. R., & Poldrack, R. A. (2005). Prospect theory on the brain? Toward a cognitive neuroscience of decision under risk. *Cognitive Brain Research, 23,* 34–50.

Tripp, G., & Alsop, B. (1999). Sensitivity to reward frequency in boys with attention deficit hyperactivity disorder. *Journal of Clinical Child Psychology, 28,* 366–375.

Tripp, G., & Alsop, B. (2001). Sensitivity to reward delay in children with attention deficit hyperactivity disorder (ADHD). *Journal of Child Psychology and Psychiatry, 42,* 691–698.

Tripp, G., & Wickens, J. R. (2009). Neurobiology of ADHD. *Neuropharmacology, 57,* 579–589. doi:10.1016/j.neuropharm.2009.07.026

Umemoto, A., & Holroyd, C. B. (2014). *Task-specific effects of reward on task switching*. Manuscript submitted for publication.

van Meel, C. S., Heslenfeld, D. J., Oosterlaan, J., Luman, M., & Sergeant, J. A. (2011). ERP associated with monitoring and evaluation of monetary reward and punishment in children with ADHD. *Journal of Child Psychology and Psychiatry, 52,* 942–953.

van Meel, C. S., Oosterlaan, J., Heslenfeld, D. J., & Sergeant, J. A. (2005). Telling good from bad news: ADHD differentially affects processing of positive and negative feedback during guessing. *Neuropsychologia, 43,* 1946–1954.

Volkow, N. D., Wang, G. J., Fowler, J. S., & Ding, Y. S. (2005). Imaging the effects of methylphenidate on brain dopamine: New model on its therapeutic actions for attention-deficit/hyperactivity disorder. *Biological Psychiatry, 57,* 1410–1415.

Walsh, M. M., & Anderson, J. R. (2012). Learning from experience: Event-related potential correlates of reward processing, neural adaptation, and behavioral choice. *Neuroscience & Biobehavioral Reviews, 36,* 1870–1884. doi:10.1016/j.neubiorev.2012.05.008

Wechsler, D. (1999). *Wechsler Abbreviated Scale of Intelligence (WASI)*. San Antonio, TX: Harcourt Brace, Psychological Corp.

Wilens, T. E. (2008). Effects of methylphenidate on the catecholaminergic system in attention-deficit/hyperactivity disorder. *Journal of Clinical Psychopharmacology, 28,* S46–S53.

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: A meta-analytic review. *Biological Psychiatry, 57,* 1336–1346.

Wood, J. M., Baker, T. E., & Holroyd, C. B. (2013, May). *Relative reward valuation in individuals who smoke as revealed by the reward positivity*. Poster session presented at the North West Cognition and Memory Fifteenth Annual Meeting, Surrey, BC.