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Ventral Striatal Prediction Error and Reward Expectancy during a Monetary Reward Task in Youth with ADHD versus Typically Developing Youth

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Abstract

Reward processing abnormalities have been observed in individuals with attention deficit/hyperactivity disorder (ADHD) in both behavioral and neuroimaging studies. Models of reinforcement learning in healthy individuals have laid the foundation for neurobiological theories addressing reward processing in ADHD. In healthy individuals, dopamine responses in ventral striatum (VS) gradually shift from actual rewards received (prediction error [PE]), toward cues which reliably predict such rewards (reward expectancy [RE]). Drawing on these observations, two theories posit that either low striatal dopamine (dynamic developmental theory) or failed signal shifts per se (dopamine transfer deficit theory) are behind reward-processing deficits in individuals with ADHD. However, the predicted signal abnormalities have not been examined directly. Forty-two participants with ADHD and 56 typically developing (TD) controls participated in a functional magnetic resonance imaging (fMRI) reward paradigm examining whole-task and temporal-change measures of PE and RE. Results showed that, contrary to theoretical predictions, the groups did not differ in either an overall measure of RE, or a composite index of PE-RE signal shifts. Furthermore, while overall PE activity was higher in the ADHD group (partly supporting the dopamine transfer deficit theory, which allows for high PE), observed decreases over time were similar between the groups (which was unexpected). Exploratory dimensional analyses showed that while a positive linear relationship between hyperactive/impulsive symptoms and RE was present in the full group, a quadratic (inverse U-shape) model better explained this relation in a sub-sample with currently-diagnosed ADHD, possibly supporting a model of downregulation due to higher symptoms. Finally, there were no significant associations between the index of temporal signal shifts and symptoms, or between overall PE and symptoms. In sum, results do not support the dynamic developmental theory, and only partially support the dopamine transfer deficit theory. Additionally, results suggest that overall signals, rather than dynamic changes, are better able to differentiate ADHD whether at the group level (PE) or at the individual level (RE). Increased PE in ADHD also suggests that immediate rewards (versus delayed reward anticipation cues) may be a useful strategy for interventions.

Layman's Abstract

People with attention deficit/hyperactivity disorder (ADHD) show differences in the way they react to rewards such as food or money. When a healthy person first receives a reward, a chemical named dopamine is released deep in the brain. Over time though, dopamine gradually stops firing in response to the reward, but rather starts firing to clues indicating that the reward is coming. Two theories claim that in people with ADHD the problem is either low levels of dopamine (dynamic developmental theory) or a high dopamine response to rewards which doesn't shift to clues (dopamine transfer deficit theory). However, no studies have directly looked at this change in firing over time in people with ADHD. Forty-two young people with ADHD and 56 healthy young people participated in a functional brain scan. Brain activity was measured in a part of the brain called the ventral striatum while participants saw clues that they may win money, and while they actually won money. Contrary to what the theories predicted, the brain response did not differ between the groups during clues that a reward was coming, nor in terms of how much the brain activity changed over time. Also, while activity related to receiving the money was higher in the ADHD group (partly supporting the dopamine transfer deficit theory), the decrease over time was similar in both groups (which was not expected). When we looked at both groups together, we found that people with more hyperactive/impulsive symptoms tended to have higher brain activity during reward clues. However, we did not find any relationships between brain activity changes over time and symptoms, or between activity while receiving money and symptoms. In summary, the results did not support the dynamic developmental theory, and only partly supported the dopamine transfer deficit theory. Additionally, the results suggest that activity across the whole task, rather than changes within the task, are better able to differentiate people with ADHD versus healthy people. Finally, the higher activity in people with ADHD while receiving rewards, suggests that immediately rewarding desired behavior may help young people with ADHD.

Introduction

Attention deficit/hyperactivity disorder (ADHD) is a common, highly heritable neurodevelopmental disorder characterized by difficulties with attention, hyperactivity/impulsivity, or a combination of these. Common consequences of poor attention can include losing things, failing to follow instructions, or not finishing difficult tasks, while consequences of hyperactivity/impulsivity can be seen in fidgeting, excessive talking, or frequent interrupting (American Psychiatric Association, 2013). ADHD prevalence estimates vary widely (0.2% to 34%), possibly due to sampling, assessment instruments, and informant differences, with a meta-analysis of 175 studies estimating a pooled prevalence of 7.2% worldwide (Thomas et al., 2015). Children with ADHD have high rates of comorbid disorders, and are at an increased risk for serious negative outcomes in adolescence and adulthood, including poor academic achievement, unemployment, substance use disorders, and premature death (Dalsgaard et al., 2015; Franke et al., 2018; Fredriksen et al., 2014). Considering both the high prevalence and high risk that ADHD poses, it is vital to gain a deeper understanding of this disorder and its neurobiological underpinnings in order to inform the further development of effective interventions.

Early neuroimaging studies of ADHD focused on neural correlates of executive functions, under the assumption that these represented core deficits (Barkley, 1997). Indeed, studies probing inhibitory, attentional, and working memory processes in individuals with ADHD compared to typically developing (TD) controls found alterations in fronto-striatal, fronto-parietal, and fronto-cerebellar networks (for a review, see Rubia, 2018). However, behavioral studies have also revealed abnormal motivational processes in ADHD, such as a preference for smaller, immediate rewards over larger delayed ones (for meta-analytic reviews, see Jackson & Mackillop, 2016; Patros et al., 2016). These findings spurred a growing body of neuroimaging literature highlighting aberrant reward processing as an additional deficit in this disorder, along with theoretical frameworks attempting to explain this deficit (Plichta & Scheres, 2014; Rubia, 2018). A consistent neuroimaging finding has been that, relative to healthy controls, there is ventral striatum (VS) hypoactivation during *anticipation* of reward in both children (Scheres et al., 2007; van Hulst et al., 2017) and adults with ADHD (Hoogman et al., 2011; Plichta et al., 2009; Ströhle et al., 2008). While findings regarding reward *receipt* itself have been less consistent, several studies have reported increased VS activation during reward receipt in youth and adults with ADHD compared to healthy controls (Furukawa et al., 2014; Paloyelis et al., 2012; Von Rhein et al., 2015).

Theories attempting to explain reward processing abnormalities in ADHD have been greatly influenced by normative reinforcement learning models. It is known that neural responses to rewarding stimuli are mediated by tonic (constant), and phasic (burst-like) dopaminergic firing (Schultz,

2016). A series of experiments in non-human primates revealed that initially, phasic bursts of mesolimbic dopaminergic activity are associated with novel or unexpected rewards, described as a type of 'prediction error' (PE) due to the mismatch between expectation and novelty or surprise (Schultz, 1998). However, as learning occurs, dopamine firing gradually shifts to the earliest cues which can reliably predict such reinforcers, a phase known as 'reward expectancy' (RE; Holland & Gallagher, 2004). This pattern of shifting has been described by the temporal difference model, alluding to the importance that timing plays in updating predictions as learning proceeds. In line with the animal literature, neuroimaging studies in healthy human adults and adolescents have shown prominent activation of dopamine-rich ventral striatal areas (and to a lesser extent, insula and ventromedial prefrontal cortex) in response to anticipation and receipt of rewards (for meta-analyses see Sescousse et al., 2013; Silverman et al., 2015). Furthermore, seminal work by O'Doherty and colleagues (2003) and recent meta-analyses (Chase, Kumar, et al., 2015; Garrison et al., 2013) have confirmed that PE signals are closely tracked in the human VS.

Based on these insights, as well as observations of altered reward-related decision making in ADHD, a number of neurobiological theories of abnormal neural reward processing in ADHD have been proposed. The dynamic developmental theory (Sagvolden et al., 2005) suggests that low tonic levels of dopamine in individuals with ADHD lead to overall weaker phasic reward activation and a slower transfer to predictive cues in VS, resulting in weaker reinforcement. In contrast, the dopamine transfer deficit theory (Tripp & Wickens, 2008) posits that phasic dopamine actually fires normally during initial reward receipt, yet fails to meaningfully transfer to the reward cue, requiring more immediate reinforcers. A prediction that both theories hold in common is that striatal hypoactivation will be observed in individuals with ADHD during reward anticipation (specifically RE) due to either low dopamine firing, or a failed dopaminergic shift from receipt to anticipation. Neuroimaging studies in ADHD cited above have indeed shown such hypoactivation, lending support to both theories. Another prediction in common is a smaller magnitude of shift in activation between initial and later stages, whether due to low dopamine limiting shifting through a floor effect (dynamic developmental theory) or due to a failure to shift per se (dopamine transfer deficit theory). To our knowledge, however, this prediction has not been tested in the existing literature. Finally, the two theories actually diverge regarding predictions of neural responses to reward receipt (and more specifically, PE): according to the dynamic developmental theory, one would expect striatal hypoactivation during reward receipt due to low dopamine levels, while according to the dopamine transfer deficit theory, one would expect normative or even enhanced activation during this phase. As mentioned above, while some studies have found no group differences, a few have found striatal hyperactivation to reward receipt in ADHD versus control participants, lending support to the dopamine transfer deficit theory. Indeed, Furukawa

et al. (2014) explicitly interpreted the VS reward receipt hyperactivation they found as supportive of this theory.

Studies on neural reward processing in ADHD have generally relied on simple condition contrasts typically used in fMRI. Only one study that we know of has specifically tested a parametrically-modulated prediction error model in reward processing (Hauser et al., 2014), finding deficient PE activity in medial prefrontal cortex in youth with ADHD compared to controls. Importantly, this study did not focus on VS activity, which is a region more closely associated with PE/RE models in the normative reward processing literature, nor did it look at RE. Additionally, to our knowledge, no previous study with ADHD participants has probed temporal shifts in striatal activity between initial and later stages within the same task—an analysis that could produce more direct evidence for the theories previously discussed.

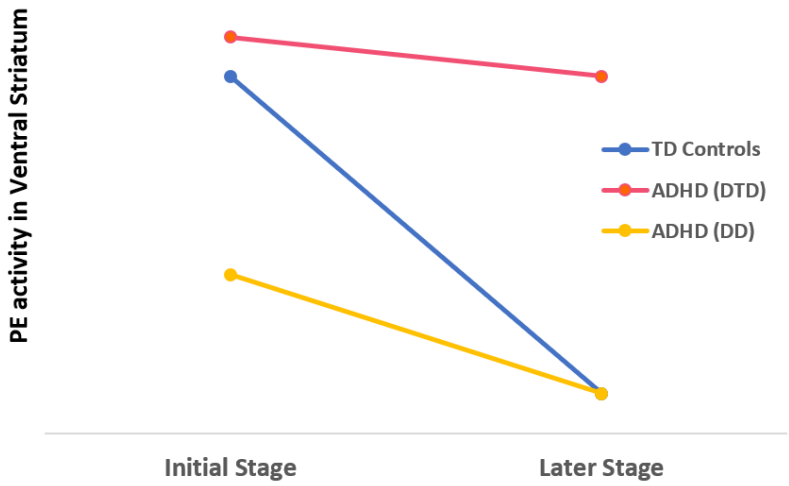
In an attempt to account for the temporal dynamics of RE and PE, Greenberg, et al. (2020) divided a monetary reward task in two halves, allowing for a direct comparison between initial and later stages of reward processing. Using this approach, they were able to link the temporal dynamics of reward processing with clinical response to pharmacological treatment response in their target population of depressed individuals. In earlier studies (Chase, et al., 2015; Greenberg, et al., 2015), they also found the expected signal shifts (decreasing PE and increasing RE over time) in healthy controls, but less so in patients. Applying a similar split-task approach with an ADHD population would allow for testing the dynamic developmental and dopamine transfer deficit theories in a more direct way. Deriving greater specificity about the components of reward processing that are impaired (outcome-related PE vs anticipation-related RE or both) could lead to more tailored reinforcement-based interventions for youth with ADHD, shifting intervention focus more toward enhanced anticipatory cues, toward primary reinforcers, or both, as the case may be.

The main aim of the present study is to test the two aforementioned theories by comparing striatal PE and RE activity during initial vs later stages of a monetary reward fMRI task in adolescents with ADHD versus TD controls. Our first hypothesis, in line with both theories and based on earlier findings, is that an overall anticipation-related RE signal in the VS will be reduced in ADHD versus TD participants. Second, we hypothesize that compared to TD youth, participants with ADHD will show a smaller PE *decrease* between initial and later stages, and a smaller RE *increase* between initial and later stages, resulting in a small composite index of change. Again, this pattern would be consistent with both low dopamine (dynamic developmental theory), and with failed signal shifting (dopamine transfer deficit theory). Third, based on previous studies of VS hyperactivation to reward receipt in ADHD, which supports the dopamine transfer deficit theory, we expect that the reward receipt-related PE signal in VS will be similar or stronger in ADHD versus TD youth in the first part of the task, and that

any signal difference between the groups would be larger in the second part of the task (as PE remains high in ADHD, yet diminishes in TD youth). In contrast, the dynamic developmental theory predicts a lower initial PE signal in youth with ADHD, which due to floor effects, diminishes slightly to match levels in TD youth in the second part. Differences between the groups under this theory would therefore be larger in the first, rather than the latter part (see Figure 1 for a visual illustration).

Figure 1

Hypothetical PE signal changes over time according to two theories of ADHD



Abbreviations. PE = prediction error; TD = typically developing; ADHD = Attention deficit / hyperactivity disorder; DTD = dopamine transfer deficit theory; DD = dynamic developmental theory.

Finally, as a secondary aim of the study, we explored potential associations between ADHD symptom severity and neural reward signals. While much of the existing ADHD literature has relied on discrete diagnostic group categories, recent work has adopted a more dimensional approach, seeking to uncover neural correlates of symptoms or phenotypic dimensions across clinical and non-clinical groups (see Coghill & Sonuga-Barke, 2012). While studies in healthy participants have shown positive correlations between VS activation to reward anticipation and reward impulsivity measures, studies in ADHD have tended to show negative correlations, in line with VS hypoactivation (for a review, see Plichta & Scheres, 2014). It is possible that symptom measures and reward impulsivity measures are capturing different aspects of the reward process. Alternatively, Plichta and Scheres (2014) propose that VS activity in relation to reward sensitivity/ADHD symptom scales might follow an inverted U-shape. Such a relationship would explain the positive relationship among healthy controls, yet negative relationship among participants with ADHD. In this context, exploring symptom-neural relationships using the more granular PE-RE approach, while testing for this kind of non-linear relationship, may yield more reliable results.

Methods

Design

This study employed an fMRI monetary reward task in a sample that was partially derived from an earlier study on risk-taking in male adolescents with ADHD (Dekkers et al., 2020). The study was approved by the medical ethical committee of the Leiden University Medical Center (protocol P16.013). The main independent variables for this study were: within-trial reward phase (RE coupled to anticipation, and PE coupled to outcome), time point within task (initial stage tied to run 1, vs later stage tied to run 2), group affiliation (ADHD vs TD controls), and ADHD symptom severity. The main dependent variables were: RE- and PE-related blood oxygen-level dependent (BOLD) signal in VS, and a reward-index measuring RE and PE shifts over time.

Participants

After enrolling participants who consented to be followed up from a previous sample, 53 male youths with ADHD and 63 TD controls were recruited for the current fMRI study (for details see Dekkers et al., 2020; and preregistration at https://osf.io/mz3d2/). Recruitment was limited to males due to power concerns for hormone assays in the original study. Inclusion criteria for both groups consisted of age between 13-23 years old, estimated IQ >= 80, and fluency in spoken and written Dutch. Dyslexia for the ADHD group was allowed if participants were able to read short sentences within 5 seconds. ADHD status was defined as meeting a previous (lifetime) diagnosis of ADHD by a licensed psychologist or psychiatrist. Current ADHD diagnosis (all subtypes) was based on a semi-structured interview (DISC-IV; Shaffer et al., 2000) administered to one of the parents/caretakers; however, remitters who no longer met DISC-IV criteria were also included in the ADHD group (*n* = 10; 24%). The DISC-IV was adapted to reflect DSM 5 changes where necessary. IQ was derived from the WISC/WAIS subtests: Similarities (Verbal) and Block Design (Performance), and groups were matched for age and IQ.

TD participants were excluded if they had a lifetime diagnosis of ADHD, oppositional defiant disorder or conduct disorder. Youth were excluded if they were on atomoxetine, clonidine, or anti-psychotic medications. Neuroimaging data were not available for 3 youths with ADHD (*n* = 2, technical problems, *n* = 1 not scanned due to anxiety) and for 3 TD youth (*n* = 1 safety concerns, *n* = 1 incidental finding, *n* = 1 language disorder). Following preregistered cutoffs, a total of 9 participants (*n* = 6 ADHD, *n* = 3 TD) were excluded for excessive head motion (>15% volumes >0.5mm framewise displacement), while 1 participant (with ADHD) was excluded for > 15% missed trials. Additionally, 3 participants (*n* =

1 ADHD, *n* = 2 TD) were excluded for severe scanner-induced artifacts. Final excluded (*n* = 11) and included (*n* = 42) participants with ADHD did not differ significantly on age (*p* = .18), IQ (*p* = .86), or total self-reported ADHD symptoms (*p* = .61). In all, our final sample consisted of 98 participants; 42 youth with ADHD, and 56 TD youth (see Table 1).

Table 1

Descriptive summary of the final sample of TD and ADHD Youth (including remitters)

	TD (*n* = 56)	ADHD (*n* = 42)	Statistic
	Mean (SD) Range		
Age	17.96 (2.26) 13.62 - 23.43	17.69 (2.26) 14.35 - 22.90	*t*(96) = -0.59, *p* = .560
WISC/WAIS estimated IQ	106.63 (12.05) 80 - 125	108.07 (13.33) 80 - 138	*t*(96) = 0.56, *p* = .578
DISC ADHD presentation (% of all ADHD)	- - - -	Combined: 7 (17%) Inattentive: 25 (59%) Hyper/Impulsive: 0 (0%) Remission: 10 (24%)	
ADHD-SR total score	**3.13 (3.03)** **0 - 13**	**8.57 (4.98)** **1 - 18**	***t*(96) = 6.70, *p* < .001**
ADHD-SR impulsivity/ hyperactivity score	**1.57 (1.46)** **0 - 6**	**3.88 (2.70)** **0 - 9**	***t*(96) = 5.43, *p* < .001**

Abbreviations. WISC = Wechsler Intelligence Scale for Children (WISC-V-NL). WAIS = Wechsler Intelligence Scale for Adults (WAIS-III). DISC = Diagnostic Interview Schedule (DISC-IV). ADHD-SR = Dutch version of ADHD DSM-IV rating scale.

Since our sample contained ADHD remitters whose inclusion with the active ADHD sample might have reduced power to detect group differences, we decided to exclude these participants from group comparisons for maximal differentiation. Based on the same rationale, we excluded participants with symptom outlier scores within each group (< 1st quartile - 1.5 * interquartile range, or > 3rd quartile + 1.5 * Interquartile range), as excessively low or high scores raised the possibility that those participants did not belong to our target populations. Using this cutoff, 4 TD youth with high outlier ADHD self-report scores were excluded. However, for the dimensional analyses, all subjects with usable data (including remitters) were included since the relationship between symptom scores and neural activity was assessed directly.

Of the 32 patients with a current DISC diagnosis, 25 (78%) had the inattentive subtype, 7 (22%) had the combined subtyped, and none had the hyperactive/impulsive subtype. Comorbidity among youth with ADHD, as reported by parents, included dyslexia (17%), autism spectrum disorders (10%), depression (2%), and oppositional defiant disorder (2%), for a total of 31% of the ADHD sample. Participants on psychostimulants (52%) were asked to refrain from using methylphenidate for 24 hours, and dextroamphetamine for 48 hours to reach complete wash-out (Wong & Stevens, 2012).

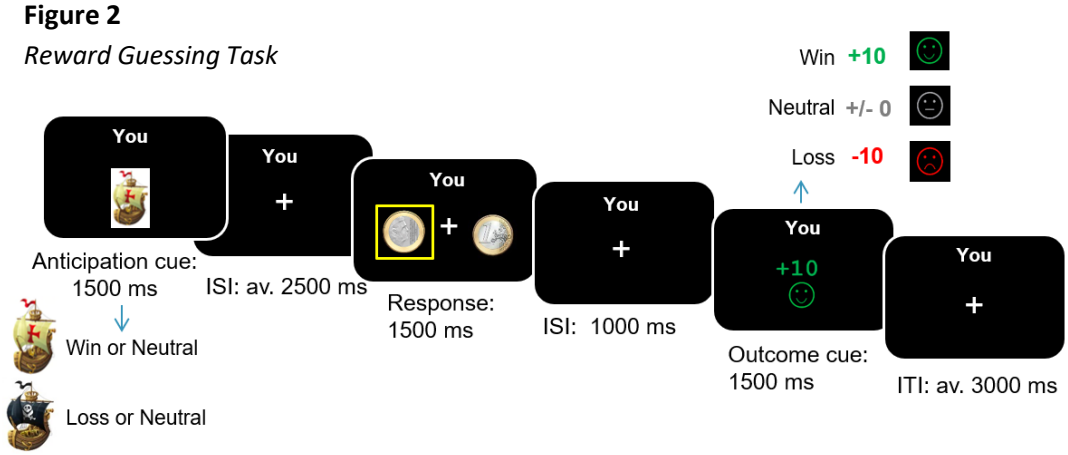
Experimental Task and Measures

fMRI Reward Task

Figure 2 depicts the reward guessing task used in this study. Participants were presented with a visual cue (either a regular ship or a pirate ship) in anticipation of a potential monetary gain or potential monetary loss. After a jittered delay, they were shown a heads and tails face of a €1-coin side by side, and were instructed to choose one of the coins through a button press with their index or ring finger. Participants were told that correct guesses would result in winning 10 points for the reward trials (or not losing any points in the loss trials), and incorrect guesses would result in winning 0 points for the reward trials (or losing 10 points for the loss trials). Unbeknownst to participants, trials were set so that wins and no-wins (or losses and no-losses) were presented in randomized order. After a fixed delay, a feedback stimulus appeared indicating points won ("+10" in green, with a smiley face), points neither won nor lost ("+0" or "-0" in grey, with a neutral face), or points lost ("-10" in red, with a sad face). Finally, a jittered inter-trial interval was included as a baseline.

Figure 2

Reward Guessing Task



A visual schematic of the reward guessing task. Participants are given a cue indicating a possible win trial or possible loss trial. After choosing a heads or tails coin, they are given feedback indicating a win or no-win for the possible win condition, or a loss or no-loss for the possible loss condition.

The task consisted of two runs, with 20 trials in the first run and 16 in the second run (trials were split up this way for practical reasons). Reward and loss cues were presented semi-randomly within the two runs (with no more than 3 consecutive trial types), and each run contained an equal number of possible reward and possible loss trials. In addition to the solo reward task (indicated by the "You" label), participants played a social reward task, which was identical to the solo task except that participants were playing on behalf of a best friend (indicated by their friend's name). Each run consisted of a social and solo block, and order of blocks within runs were counterbalanced across participants. Although a future study aims to compare these conditions, for our research question, the social and solo blocks were collapsed for improved power. In order to assess initial versus later stages of reward processing, the first and second runs were used to represent these respective stages. Since the number of trial types per block was fixed, no behavioral outcomes were measured.

The main task-related independent variables were two parametric modulators: RE and PE, coupled to the anticipation and outcome conditions of each trial, respectively. RE represents the probability of winning or losing 10 points, with a value of +0.5 for the possible-win trials, and -0.5 for the possible-loss trials. PE represents the difference between the expected probability and the actual outcome, with a value of +0.5 for a win after a possible-win trial, -0.5 for a no-win following a possible-win trial, -0.5 for a loss following a possible-loss trial, and +0.5 for a no-loss after a possible-loss trial.

To quantify the transfer of PE to RE, we calculated a 'reward-index' (Greenberg et al., 2020), composed of striatal Δ PE signal (PE run 1 - PE run 2), plus striatal Δ RE (RE run 2 - RE run1). It is assumed that in healthy participants, the marked shift between PE to RE activity in VS over the course of a task would result in relatively high Δ values for each component, as both change over time. When summed together, they would thus result in an overall high reward-index. In contrast, either overall low PE and RE signals limiting the range of shifts across time points (in line with the dynamic developmental theory), or high PE signals and low RE signals remaining static across time points (in line with the dopamine transfer deficit theory), would both result in decreased Δ values for each component, and therefore a lower reward-index.

fMRI Data Acquisition

Neuroimaging data was collected using a 3T Achieva Philips MRI scanner (Philips Medical Systems, Best, the Netherlands) at Leiden University Medical Center with a standard head coil. Anatomical T1 weighted whole brain 3D images were acquired for each subject (FFE pulse sequence; 155 continuous slices; repetition time [TR] = 7.9 ms; echo time [TE] = 3.5 ms; flip angle [FA] = 8°; field of view [FOV] = 249.9mm x 195.8mm x 170.5mm; voxel size = 1.1mm x 1.1mm x 1.1mm; reconstruction

matrix = 240 x 240). Whole brain T2* weighted echo planar images with a BOLD contrast were acquired (EPI; 38 interleaved slices; TR = 2200 ms; TE = 30 ms; FA = 80°; FOV = 220mm x 220mm x 114.7mm; voxel size = 2.75mm x 2.75mm x 2.75mm; reconstruction matrix = 80 x 80), comprising 208 volumes for the first run and 168 volumes for the second run. Additionally, a B0 nonuniformity fieldmap was acquired for later distortion correction.

Individual Differences in Symptom Severity

ADHD symptom severity was measured with the Dutch-language self-report DSM-IV ADHD rating scale (ADHD-SR; Kooij et al., 2005). The scale consists of 26 questions based on the set of 18 DSM-IV ADHD symptoms (some items correspond to different aspects of the same symptom, resulting in more items than symptoms). Participants rated each item on a scale from "0" (never) to "3" (very often), with scores of 2 or 3 coded as 1 point, indicating the presence of that specific symptom. For symptoms with multiple items, a score of 2 or 3 on any one of its corresponding items was considered as endorsing that symptom, following the scoring guide. As such, final scores ranged from 0 to 18 possible ADHD symptoms, with higher scores indicating greater overall symptomatology. The subscale measuring only hyperactivity/impulsivity symptoms was a subset of items matching the DSM-IV criteria for hyperactivity/impulsivity symptoms, and relied on the same scoring algorithm, resulting in possible scores of 0 to 9, with higher scores reflecting more symptoms. Reliability and external validity have been shown to be adequate (all Cronbach's alpha's > .7 for inattentive, hyperactive, and impulsive sub-scores; significant correlation shown with self-rated psychological disorder and impairment; Kooj et al., 2005).

Procedure

All participants and their parents (for minors < 16 years) signed an informed consent form prior to the start of the study. Before the scan session, youth were trained to reduce head motion on a mock scanner, and given instructions and a short practice with the task. Participants then completed the scan session, which involved a T1-weighted anatomical scan, a fieldmap of B0 inhomogeneities, two BOLD tasks (only the reward task was analyzed), a DTI sequence, and resting state acquisition if time allowed. Self-report questionnaires were filled out in Qualtrics after the scan during the same session. The total experimental session took approximately 3 hours. Parent-report questionnaires were filled out by the parents during a home visit in which a DISC interview was also carried out. Although participants were told that monetary compensation would depend on their performance, all youths

received a fixed monetary compensation in Euros for their participation. Parents received 10 euros to complete a set of questionnaires, and 10 euros for participating in the DISC-interview (the latter for the ADHD group only).

Data Processing and Analysis

Preprocessing

fMRI data was visually inspected for quality after running an automated quality control tool used to calculate head motion and signal outliers (mriqc; Esteban et al., 2017). Participants were excluded if they showed excessive motion or severe scanner artifacts (see exclusions in the *Participants* section). Preprocessing was performed using a validated pipeline (fMRIprep; Esteban et al., 2019). Briefly, T1-weighted anatomical images were corrected for intensity non-uniformity and skull stripped before performing brain tissue segmentation. Images were then normalized to standard Montreal Neurological Institute (MNI) space through nonlinear registration of the reference T1 weighted image and an ICBM-152 template (Fonov et al., 2009). BOLD images in each run were skull-stripped, and susceptibility distortion correction was applied to a BOLD reference based on the acquired fieldmaps. The BOLD reference image was then co-registered to the T1 weighted reference using boundary-based registration in FSL (https://fsl.fmrib.ox.ac.uk/). Head motion parameters with respect to the BOLD reference were then estimated using FSL MCFLIRT, and slice-time correction was applied using AFNI (https://afni.nimh.nih.gov/). All volumes were then resampled into native space to apply a single composite transform from the previous steps. Framewise displacement motion values, mean signal from cerebrospinal fluid and white matter tissue, and separate binary regressors for each timepoint where the framewise displacement was > 0.5mm, were calculated in this step. Finally, all volumes were resampled to standard MNI space, and a gaussian smoothing kernel of 8mm at full-width half-maximum was applied.

First-level Analyses

For each participant, we constructed a fixed-effect general linear model in SPM12 (http://www.fil.ion.ucl.ac.uk/spm). Since parametric modulators require a matching unmodulated regressor, a regressor containing all onsets and durations for the anticipation cue (regardless of possible win or loss) was input into the model, and the modulator RE described in the *measures* section was paired to each onset. Similarly, a regressor containing onsets and durations for all the outcome

events (regardless of win, loss, or neutral outcomes) was input into the model and the modulator PE was paired to each onset as described above. As previously mentioned, solo and peer trials were combined to increase power. Additionally, an unmodulated regressor with the onsets and durations for each coin guess (representing participant responses) was also included to fully model the experimental space. All other intra- and inter-trial intervals were left as implicit baselines in the model, and a separate constant term was included for each run to account for mean differences. Finally, the following nuisance regressors were included: a regressor for omitted trials (with onset at trial start and duration spanning the entire trial), 6 motion parameters from the realignment (3 translation and 3 rotation axes) along with their first order derivatives, mean signal in cerebrospinal fluid and white matter masks along with their first order derivatives, a regressor for framewise displacement, and a variable number of regressors for each volume in which framewise displacement was greater than 0.5mm. Regressors of interest were convolved onto a standard hemodynamic response function (HRF) to model underlying neural activation, and high-pass filtered with discrete cosine basis functions at 128s cut-off to remove scanner drift. Serial autocorrelations were modeled using a first-order autoregressor process.

Second-level Analyses

After running first level models, we extracted parameter estimates from our VS region of interest for each participant. In order to avoid biased estimates, we used an independent functional mask of left (x = -12, y = 8, z = -4) and right (x = 12, y =14, z = -6) VS derived from a meta-analysis of reward activation to similar tasks in healthy youth (Silverman et al., 2015). An 8mm sphere matching the smoothing kernel was centered on each of the coordinates in order to extract and average the parameter estimates of each dependent variable of interest within a sphere and between the two spheres to create an overall measure of VS activity. In order to test our first hypothesis, RE values were extracted and compared between the two groups (ADHD vs controls) in SPSS with a one-way ANCOVA, covarying for age and mean framewise displacement. Statistical assumptions were checked for all ANCOVA variables, including visual inspection using P-P plots examining within-group distributions, inspection for outliers > 3 standard deviations, homogeneity of variance between groups using Levene's test, and homogeneity of regression slopes by examining scatterplots. Unless otherwise specified, all assumptions were met.

To evaluate reward-index differences between ADHD vs. TD participants, we extracted PE and RE signals (i.e. beta values from a sphere mask centered in VS) separately for the first and second runs of the task. A composite reward-index was constructed by first deriving Δ values between the runs as described above, and then summing these together (Δ PE + Δ RE). This index was then entered in a

one-way ANCOVA comparing ADHD and TD controls, with age and mean framewise displacement as covariates of no interest.

To test our third hypothesis, we ran a 2x2 mixed ANCOVA on PE, with time point (run 1, run 2) as the within-subjects factor and group (ADHD, TD) as the between-subjects factor. We included age and mean framewise displacement as covariates of no-interest in the model. Since the dopamine transfer deficit theory predicts normative initial PE signals in ADHD which remain constant (in contrast to decreasing signal in controls), a significant interaction such that differences between PE signal among the groups are greater in the second run, would support this hypothesis. Note that this analysis is more specific than that of our second hypothesis: while small reward indices could be due to either overall low PE/RE signals restricting Δ's *or* to constant PE/RE signals across the task restricting Δ's, a specific test comparing PE in run 1 and run 2 (with run 1, but not run 2 being equal or greater) was necessary.

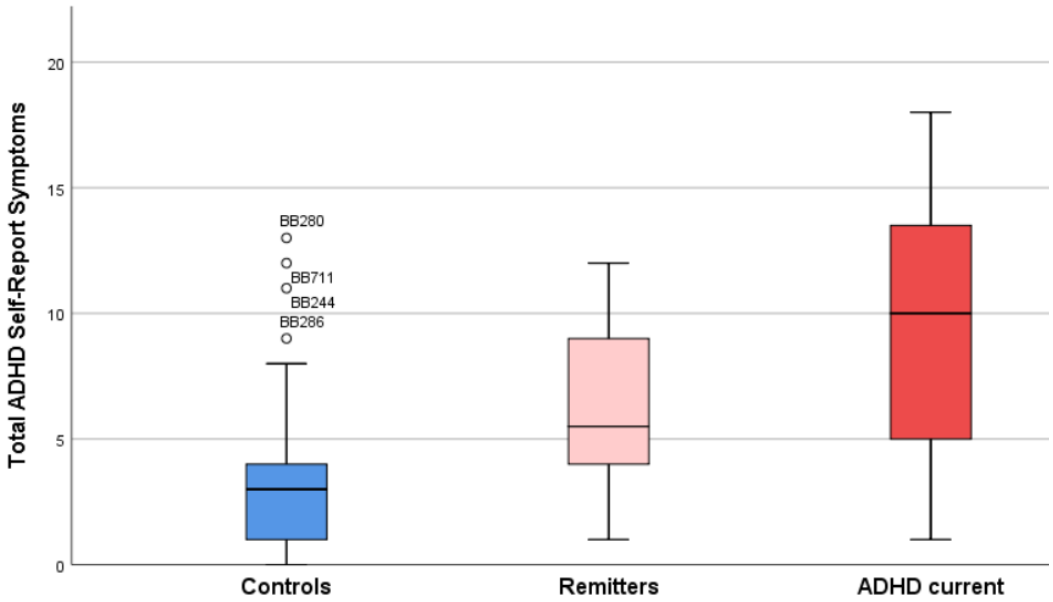
Finally, for exploratory individual differences analyses, we included the entire sample, as relationships between symptoms and neural activity were tested cross-diagnostically. We ran three main multiple regression models. In the first model, RE across the entire task was the outcome variable, with self-report ADHD hyperactivity/impulsivity symptoms as the predictor, and age, mean framewise displacement, and medication status (on/off medications), as regressors of no interest. As a variant of this model, we also tested a quadratic term for hyperactivity/impulsivity symptoms, as suggested by (Plichta & Scheres, 2014). In the second main model, the reward-index was entered as the outcome variable, with total self-report ADHD symptoms as the predictor, and age, mean framewise displacement, and medication status, as regressors of no interest. Finally, mean PE signal was entered as the outcome variable, with self-report ADHD hyperactivity/impulsivity symptoms as the predictor of interest, and age, mean framewise displacement, and medication status as nuisance regressors. The hyperactivity/impulsivity subscale was used in the regressions predicting overall RE and PE to more closely align with the studies reviewed in Plichta and Scheres (2014), which focused on scales related to impulsivity, rather than inattention. Total symptom scores were used for the reward-index regression as the dynamic aspect make it a novel framework which would benefit from an initial probe of overall symptoms. Statistical assumptions for regressions were checked, including multicollinearity among predictors (variance inflation factor < 10), normality of residuals and homoscedasticity through visual inspection, and no residual outliers (standardized residuals < 3 standard deviations). Unless otherwise stated, all assumptions were met.

Results

Group analyses were restricted to the ADHD subgroup with an active SCID diagnosis (no remitters) and the TD controls with outliers excluded, as this more closely followed our aim of maximizing group differences by comparing currently clinical vs non-clinical groups (see Figure 3 for boxplots comparing the three groups before exclusions). Thus, the main group analyses were based on n = 52 TD youth and n = 32 youth with an active ADHD diagnosis (total *n* = 84).

Figure 3

Boxplots of Total ADHD Self-Report Symptoms by Group



Note: boxplots shown for illustrative purposes, are for all participants before excluding remitters and control outliers. Shaded areas represent the interquartile range, with horizontal lines indicating the median, and error bars extend out to the furthest data point within 1.5 * IQR in both directions.

RE across Entire Task

To evaluate potential differences in anticipation-related RE activity in VS between ADHD versus TD participants, a one-way ANCOVA was run on mean extracted RE betas. Results showed no significant main effect of group, $F(1,80) = 0.11$, $p = .74$, partial $\eta 2 = .001$ (TD adjusted $M = 0.06$, $SE = 0.02$; ADHD adjusted $M = 0.07$, $SE = 0.03$).

Reward-index

Second, to evaluate potential differences in RE and PE signal shifting between the groups, a one-way ANCOVA was run comparing the two groups on the computed reward-index from extracted

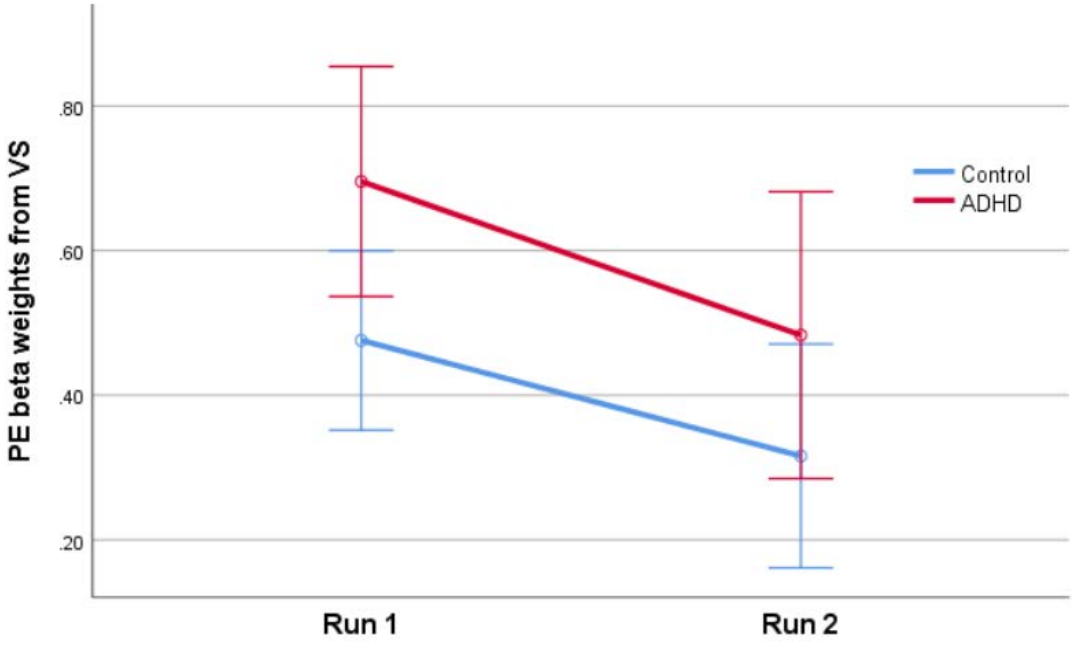
VS values. Results showed no significant main effect of group, $F(1,80) = 0.11$, $p = .75$, partial η2 = .001 (TD adjusted $M = 0.09$, $SE = 0.10$; ADHD adjusted $M = 0.14$, $SE = 0.12$).

PE by Runs

Third, a mixed 2x2 ANCOVA on VS-extracted PE values with time point (run 1, run 2) as the within-subjects factor, and group (ADHD, TD controls) as the between-subjects factor, showed that there was no significant interaction between time point and group, $F(1,80) = 0.14$, $p = .71$, partial η2 = .002. However, there was a significant decrease between values in the first and second run across all subjects, $F(1,80) = 7.42$, $p = .008$, partial η2 = .09 (run 1 adjusted $M = 0.59$, $SE = 0.05$; run 2 adjusted $M = 0.40$, $SE = 0.06$), as well as a significantly greater PE activation across both runs for the ADHD group, $F(1,80) = 4.38$, $p = .039$, partial η2 = .052 (TD adjusted $M = 0.40$, $SE = 0.06$; ADHD adjusted $M = 0.60$, $SE = 0.07$; see Figure 4).

Figure 4

PE beta values extracted from VS for ADHD and TD participants as a function of task run



Abbreviations. PE = Prediction Error. VS = Ventral Striatum.
Notes. Points represent estimated marginal means. Bars represent 95% confidence intervals. Covariates appearing in the model are evaluated at the following values: Age at Scan = 17.8, Mean Framewise Displacement across both runs = 0.135

Individual Differences

For exploratory dimensional analyses, we included the entire sample with available data (n = 97 due to missing symptom data for one participant), since the focus was on cross-diagnostic individual differences rather than group comparisons. A dimensional symptom approach for RE, Reward Index and PE was applied, mirroring the order of the group analyses.

ADHD Symptoms and RE

First, we ran a regression analysis to test whether self-reported ADHD hyperactivity/impulsivity sub-scores could significantly predict RE activity in VS, while accounting for possible confounds. Results showed that the model was a significant predictor of VS RE, $F(4,93) = 2.92$, $p = .025$, adj. $R^2 = .07$ (see Table 2). The variable of hyperactive/impulsive symptoms was a significant positive predictor of RE, β = .27, $t(93) = 2.55$, $p = .012$, while medication status was a significant negative predictor of RE, β = -.28, $t(93) = 2.48$, $p = .015$. Case-wise diagnostics revealed that two participants had standardized residual values above 3 standard deviations. Removing those two participants resulted in a similar pattern of findings.

Table 2.

Multiple regression results for Ventral Striatum Reward Expectancy

VS RE	*B*	95% CI for *B*		*SE B*	β	*t*	*p*	R^2
		LL	*UL*					
Model								.11
Constant	0.092	-0.174	0.359	0.134		0.69	.493	
Hyper/Impulsive ADHD Symptoms	**0.015**	**0.003**	**0.028**	**0.006**	**.27**	**2.55**	**.012**	
Age	-0.006	-0.019	0.006	0.006	-.11	-1.01	.316	
Mean framewise displacement	0.468	-0.196	1.133	0.335	.15	1.40	.165	
Medication	**-0.089**	**-0.161**	**-0.018**	**0.036**	**-.28**	**-2.48**	**.015**	

Note. VS = ventral striatum; RE = reward expectancy; *B* = unstandardized regression coefficient; CI = confidence interval; *LL* = lower limit; *UL* = upper limit; *SE B* = standard error of the coefficient; β = standardized coefficient; R^2 = coefficient of determination; Adj. R^2 = adjusted R^2.

Additionally, we also tested a quadratic regression of hyperactive/impulsive symptoms and VS RE, as suggested by Plichta and Scheres (2014), to potentially reconcile prior findings of opposite associations between impulsivity and reward anticipation in healthy controls (positive) and participants with ADHD (negative). Adding a mean-centered quadratic term for hyperactive/impulsive symptoms to the simple linear model with VS RE activity as the outcome variable increased the R square from .052 to .077, but this was not a significant R square change: linear model, $F(1,95) = 5.13$, $p = .026$, $R^2 = .052$; model with linear and quadratic terms, $F(2,95) = 2.58$, $p = .112$, $R^2 = .077$; R^2 Change = .026, $F(1,95) = 2.58$, $p = .112$. Since the inverted-U-shape model in Plichta and Scheres (2014) was proposed based on studies using an active diagnosis in the ADHD group, we also ran this regression excluding remitters. Results showed that the linear model by itself was no longer significant, $F(1,82) = 2.45$, $p = .121$, $R^2 = .029$, while the model with the quadratic term was a significant predictor of VS RE, $F(1,81) = 4.58$, $p = .035$, $R^2 = .081$, adj. $R^2 = .058$; R^2 Change = .052, $F(1,81) = 3.57$, $p = .033$, with the quadratic term significantly predicting VS RE (see Table 3 and Figure 5).

Table 3.

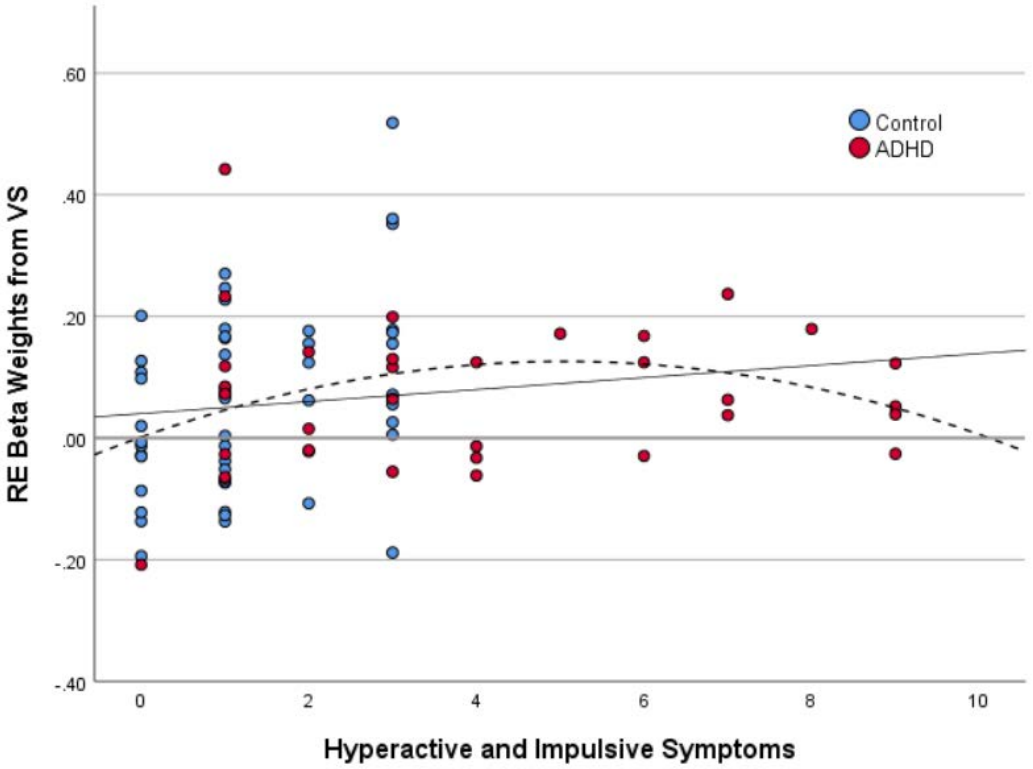
Regression results for a Linear versus Quadratic Model of Ventral Striatum Reward Expectancy

VS RE	*B*	95% CI for *B*		*SE B*	β	*t*	*p*	R^2
		LL	*UL*					
Model 1 (Linear)								.03
Constant	**0.066**	**0.036**	**0.095**	**0.015**		**4.39**	**< .001**	
Hyper/Impulsive ADHD Symptoms	0.010	-0.003	0.022	0.006	.17	1.57	.121	
Model 2 (Linear and Quadratic)								.08
Constant	**0.096**	**0.055**	**0.136**	**0.020**		**4.71**	**< .001**	
Hyper/Impulsive ADHD Symptoms	**0.024**	**0.006**	**0.042**	**0.009**	**.42**	**2.66**	**.009**	
Hyper/Impulsive ADHD Symptoms Squared	**-0.005**	**-0.009**	**0.000**	**0.002**	**-.34**	**-2.14**	**.035**	

Note. VS = ventral striatum; RE = reward expectancy; *B* = unstandardized regression coefficient; CI = confidence interval; *LL* = lower limit; *UL* = upper limit; *SE B* = standard error of the coefficient; β = standardized coefficient; R^2 = coefficient of determination; Adj. R^2 = adjusted R^2.

Figure 5

Linear and Quadratic Fit of Hyperactive/Impulsive Symptoms versus Reward Expectancy in right VS



Abbreviations. VS = ventral striatum; RE = reward expectancy

ADHD Symptoms and the Reward Index

Next, we ran a regression analysis to examine whether ADHD self-report scores could predict reward-index activation in left and right VS, while covarying for possible confounds. The multiple regression model was not a statistically significant predictor of VS reward-index, $F(4,93) = 0.90$, $p = .47$, $R^2 = .037$. None of the predictor beta coefficients were statistically significant (all p's > .2). One participant had a standardized residual lower than 3 standard deviations. Excluding that participant resulted in similar results.

ADHD Symptoms and PE

Finally, we ran a regression analysis to examine whether ADHD hyperactivity/impulsivity symptoms could predict VS PE activity, while accounting for possible confounds. Results showed that while the regression model significantly predicted VS PE, $F(4,93) = 2.63$, $p = .039$, $R^2 = .10$, adj. $R^2 = .06$. hyperactive/impulsive symptoms were not a significant individual predictor (see table 3).

Table 3.

Multiple regression results for Ventral Striatum Prediction Error

VS RE	*B*	95% CI for *B*		*SE B*	β	*t*	*p*	R^2
		LL	*UL*					
Model								.10
Constant	1.543	0.702	2.384	0.423		3.64	.000	
Hyper/Impulsive ADHD Symptoms	0.035	-0.003	0.073	0.019	.20	1.85	.067	
Age	**-0.052**	**-0.092**	**-0.012**	**0.020**	**-.28**	**-2.61**	**.010**	
Mean framewise displacement	-1.719	-3.814	0.375	1.055	-.17	-1.63	.106	
Medication	-0.063	-0.288	0.162	0.113	-.06	-0.56	.579	

Note. VS = ventral striatum; PE = prediction error; *B* = unstandardized regression coefficient; CI = confidence interval; *LL* = lower limit; *UL* = upper limit; *SE B* = standard error of the coefficient; β = standardized coefficient; R^2 = coefficient of determination; Adj. R^2 = adjusted R^2.

Discussion

In the present study, our main goal was to test two neurobiological theories of reward processing abnormalities in ADHD, using an fMRI reward guessing task. The dynamic developmental theory suggests that reward processing abnormalities are due to low striatal dopamine, with hypoactivation during anticipation and receipt of rewards, and restricted shifts between the two, due to floor effects. In contrast, the dopamine transfer deficit theory suggests that while initial dopamine response to receipt of rewards is normative or enhanced, it remains high over time, while anticipatory reward activity remains low. In line with both theories, we predicted that youth with ADHD would show reduced striatal activity during reward anticipation, and reduced striatal activity shift between reward receipt and anticipation, compared to typically developing controls. Additionally, we expected that solely in line with the dopamine transfer deficit theory, reward receipt activation would remain high in youth with ADHD across the task, while decreasing in healthy youth over time. To our knowledge, this was the first study to use a reinforcement learning approach examining reward receipt-linked prediction error signals, and reward anticipation-linked reward expectancy signals in

striatum in this population. Furthermore, none of the existing studies have analyzed changes in these signals within the same task – a dynamic process which is both predicted by normative reward processing theories, and theorized to be deficient in individuals with ADHD (Sagvolden et al., 2005; Tripp & Wickens, 2008).

First, contrary to expectations, our results showed no significant differences in reward anticipation-linked striatal RE activity between youth with ADHD and healthy controls. One possible explanation is that, despite excluding ADHD remitters, participants in the ADHD sample may have had symptoms which were too low, resulting in overlap with the control group, thus making it harder to detect small group differences. Indeed, 14 out of 32 currently diagnosed youth (44%) reported symptoms low enough to be considered within the normal range. Additionally, four TD participants had high outlier scores, and though these were removed from group analyses, others with somewhat elevated scores within the IQR * 1.5 range were included. Task design could have also played a role. Most previous studies showing anticipatory striatal hypoactivation in ADHD were based on the monetary incentive delay task, where participants must respond quickly enough to a cue in order to win (or avoid losing) a reward (Hoogman et al., 2011). It is plausible that the increased attentional demand and greater engagement more strongly recruits VS, especially during the anticipation phase, resulting in greater power to detect group differences. Additionally, the inclusion of a peer condition in our task could have diminished power to detect group differences if there was differential VS involvement in self-, versus peer-conditions. Future analyses focusing on this condition will help elucidate the role that social perspective may play in reward processing.

Second, contrary to expectations, we found no differences between the two groups in the magnitude of signal shift between reward receipt-linked PE and anticipation-linked RE, measured through a composite index of change. Again, it is possible that symptom overlap obscured group differences, especially if signal change differences between the groups were subtle. A different aspect of task design could have also impacted measures of within-task signal change. The reward guessing task in studies by Greenberg et al. (2015, 2020) was a slow event-related task which included a 9-second baseline at the end of each trial, from which the last 3 seconds were sampled as an explicit baseline. In contrast, the task used in this study was a fast event-related design, with an implicit baseline. Although explicit baselines are usually discouraged (Pernet, 2014), modeling one in a slow event-related design (with enough time for the hemodynamic response to return to baseline) could hypothetically provide a more stable contrast when comparing the same condition over time within the same task. A final possibility, however, is that contrary to both theories, PE and RE signals truly do change in a similar manner in both healthy controls and youth with ADHD. Indeed, when specifically looking at PE, we found a signal decrease between the first and second run of the task across all participants (suggesting that our task was indeed able to pick up on dynamic aspects of PE (in the

expected normative direction), and that this decrease was similar between youth with ADHD and healthy controls.

Third, despite finding a similar PE signal decrease between the groups (i.e. no time point x group interaction), we found a main effect of PE, such that youth with ADHD had an overall higher PE activation across both time points versus healthy youth. A higher overall PE signal is more compatible with the dopamine transfer deficit theory than the dynamic developmental theory, since the latter would predict *lower* reward receipt activity in ADHD across both time points. Additionally, higher activation during PE in ADHD participants is also consistent with previous studies reporting increased activation to reward receipt in this population (Furukawa et al., 2014; Paloyelis et al., 2012; Von Rhein et al., 2015). However, the observed PE decrease over time in ADHD participants does not support the persistently high PE signal predicted by the dynamic dopamine theory. Collectively, our group findings only partially support the dynamic dopamine theory, suggesting that an overall higher reward receipt signal, rather than altered signal shifts, are better able to differentiate ADHD compared to healthy controls.

Our first dimensional analysis revealed that hyperactive-impulsive symptoms positively predicted overall RE values across the combined samples, in contrast to the RE group comparison failing to show differences. While this supports the validity of a dimensional approach, it is also possible that the previously-mentioned symptom overlap between the groups could have favored individual versus group differences. Interestingly, the positive relationship found is more in line with studies showing a positive association between VS reward activity and measures of impulsivity in healthy participants (e.g. Abler et al., 2006; Forbes et al., 2009; Hoogman et al., 2011), whereas studies with ADHD participants have tended to show a negative association (e.g. Carmona et al., 2012; Scheres et al., 2007; Ströhle et al., 2008). In order to explain the discrepant literature, Plichta and Scheres (2014) proposed that measures of impulsivity may have a quadratic, inverted U-shaped relationship with anticipatory VS activity, such that VS activity initially increases with impulsivity in healthy controls, but after downregulation in response to overstimulation, it falls in individuals with ADHD. Since our full sample included more healthy controls and ADHD youth with relatively low symptom scores, it is possible the positive linear relationship on the left side of a putative inverted U-shape may have predominated. Additionally, most of the studies reviewed by Plichta and Scheres (2014) only included participants with currently-diagnosed ADHD, making it possible that including remitters biases the relationship to the left side. Indeed, while a linear relationship better explained the neural-symptom relationship in our full sample, excluding remitters resulted in a model with a quadratic term offering a better fit. An important caveat to this interpretation is that effect sizes were small, and the exploratory nature of our analysis require further studies which can specifically recruit more differentiated yet similarly sized samples for this type of analysis

In contrast to RE results, a categorical approach proved superior to a dimensional one in examining PE differences. Though the direction of the effect was in line with at least one previous study showing a positive association between VS reward receipt activation and impulsive symptoms among individuals with ADHD (Furukawa et al., 2014), it was not a significant relationship. While a larger sample size may have been needed, it is also possible that PE activity is less susceptible to individual differences, and may rather represent a more general trait in ADHD. Studies have previously shown increased reward receipt-related striatal activity in participants with ADHD (Furukawa et al., 2014; Paloyelis et al., 2012; Von Rhein et al., 2015), a finding which better comports with the dopamine transfer deficit theory. Importantly, such a finding suggests that interventions focusing on immediate rewards to shape behavior in individuals with ADHD may prove more effective than interventions using cues or delayed rewards, which may vary according to the individual.

Finally, unlike the overall RE signal measure which showed dimensional relations, there was no significant association between ADHD symptom scores and the dynamic reward-index. As such, this result again failed to support the importance of dynamic aspects. Taken together with the findings of the group analyses, it seems likely that dynamic PE and RE changes are not good markers of ADHD diagnosis or symptoms, calling into question the full validity of the theories tested.

Strengths, Limitations and Future Directions

This was the first study to examine reward processing in ADHD using a temporal difference model with intra-task time point comparisons in fMRI. As such, it allowed us to more directly test two neurobiological theories of reward processing abnormalities, accounting for signal changes within the same task. Despite these strengths, the current study had some limitations. First, the overlap in symptom scores – even when excluding remitters – raises the possibility that the samples were suboptimal for detecting group differences. Both low symptoms among ADHD youth, and some high scores among TD youth could have decreased power to detect differences. Future studies could therefore try to recruit ADHD participants with higher symptom severity and enforce symptom cut-offs for TD participants. Second, although the task we used is based on a well validated card guessing paradigm (Delgado, 2007), its design differs from the task (Monetary Incentive Delay task) that has most commonly been used to probe reward processing in ADHD. Furthermore, our task included an additional “peer” condition where the participant was told that reward won or lost for those trials will go to their friend. While this allows for elucidating the role of social cognition in reward, for the purposes of the current analyses it may have reduced power. Future studies could use the reinforcement learning approach with the Monetary Incentive Delay task to examine whether these findings replicate. Also, while our study was intentionally focused on VS, future studies could examine PE and RE processes in other regions, such as anterior cingulate, medial prefrontal areas, and insula,

as well as functional connectivity to elucidate the interplay between brain regions over time. Finally, generalizability could be improved by exploring these differences in groups including females. And while our study collapsed all ADHD subtypes, future studies could explore overall and dynamic differences in each subtype, provided sufficiently large sample sizes are recruited.

In summary, the current study examined reward anticipation- and receipt-related signals to test two neurobiological theories of reward processing in ADHD. Findings revealed that while there were no group differences in dynamic changes predicted by the theories, a higher receipt-related PE signal in youth with ADHD was more supportive of the dopamine transfer deficit theory than the dynamic developmental theory. Dimensional analyses revealed a positive linear relationship between symptoms and reward anticipation signals; however, when excluding remitters, a quadratic inverted U-shape model was favored, with lower activity at the extremes. Taken together, results suggest that dynamic ventral striatal reward signal changes may not be key features of ADHD. Rather, overall striatal activation during anticipation may be associated with individual symptoms, while ADHD youth as a group may be biased towards higher reward receipt activation. These results, if confirmed, would favor interventions targeting immediate rewards in shaping impulsive behavior, rather than reward cues which may vary across individuals.

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