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Disrupted Expected Value Signaling in Youth With Disruptive Behavior Disorders to Environmental Reinforcers

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Abstract

Objective—Youth with disruptive behavior disorders (DBD), including conduct disorder (CD) and oppositional defiant disorder (ODD), have difficulties in reinforcement-based decision making, the neural basis of which is poorly understood. Studies examining decision making in youth with DBD have revealed reduced reward responses within the ventromedial prefrontal cortex/orbitofrontal cortex (vmPFC/OFC), increased responses to unexpected punishment within the vmPFC and striatum, and reduced use of expected value information in the anterior insula cortex and dorsal anterior cingulate cortex during the avoidance of suboptimal choices. Previous work has used only monetary reinforcement. The current study examined whether dysfunction in youth with DBD during decision making extended to environmental reinforcers.

Method—A total of 30 youth (15 healthy youth and 15 youth with DBD) completed a novel reinforcement-learning paradigm using environmental reinforcers (physical threat images, e.g., striking snake image; contamination threat images, e.g., rotting food; appetitive images, e.g., puppies) while undergoing functional magnetic resonance imaging (fMRI).

Results—Behaviorally, healthy youth were significantly more likely to avoid physical threat, but not contamination threat, stimuli than youth with DBD. Imaging results revealed that youth with DBD showed significantly reduced use of expected value information in the bilateral caudate, thalamus, and posterior cingulate cortex during the avoidance of suboptimal responses.

Conclusions—The current data suggest that youth with DBD show deficits to environmental reinforcers similar to the deficits seen to monetary reinforcers. Importantly, this deficit was unrelated to callous-unemotional (CU) traits, suggesting that caudate impairment may be a common deficit across youth with DBD.

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Keywords

disruptive behavior; conduct disorder; decision making; expected value; environmental reinforcers

Youth with disruptive behavior disorders DBD, including conduct disorder (CD) and oppositional defiant disorder (ODD), show increased aggression and antisocial behavior.¹ Moreover, prognosis is poor, with approximately half presenting with severe pathology in adulthood.^{2,3} In addition to antisocial behavior, youth with DBD have difficulties in reinforcement-based decision making, the neural bases of which are only beginning to be understood.⁴⁻⁹

Youth with DBD show reduced reward responses within the ventromedial prefrontal cortex/orbitofrontal cortex (vmPFC/OFC),^{5,8,10} and increased responses to unexpected punishment within the vmPFC and striatum.^{5,7} A recent model-based functional magnetic resonance imaging (fMRI) study revealed specific neuro-computational impairments.⁶ Thus, both prediction error (PE) signaling and the representation of the expected value (EV) of a choice are disturbed in youth with DBD. Typically, healthy individuals show increased striatal responses to positive PE (greater reward than expected).¹¹ Youth with DBD showed a reduction in this signaling relative to comparison youth.⁶ In addition, during response selection, healthy individuals typically show increases in vmPFC activity as a function of EV (i.e., increasing vmPFC activity the more that the choice is associated with expected reward).^{11,12} Youth with DBD have shown a reduction in this signaling relative to comparison youth.⁶ Finally, when attempting to avoid suboptimal choices, healthy individuals typically show increases in the anterior insula cortex (AIC), caudate, and dorsal anterior cingulate cortex (dACC).^{13,14} These regions are involved in changing current responding (i.e., if the individual is about to avoid responding to a desirable stimulus), which can occur as a function of EV.^{15,16} Youth with DBD showed a reduction in EV signaling within AIC and caudate during avoidance responses.⁶

Previous studies of decision making in youth with DBD have used monetary reinforcement. The goal of the current study was to determine whether dysfunction in PE and EV signaling in youth with DBD would extend to environmental reinforcement. This is an interesting issue, as aversive environmental reinforcement is exposure to a noxious stimulus, whereas aversive reinforcement during monetary reinforcement is the removal of a positive stimulus. This study involved a novel decision-making paradigm in which participants chose whether to enter doors that might lead to rewarding (appetitive images) or aversive consequences (images of physical or contamination threats). Importantly, the current study used computational model-based fMRI, which allows for testing how, not simply where, a function is conducted (i.e., whether EV and PE computations are intact).¹⁷ Regressors for the choice phase were weighted by EV (the degree to which the door previously predicted outcome), and those for the feedback-phase were weighted by PE (the difference between predicted and received outcome), according to learning theory.¹⁸ We predicted that, during decision making (choice phase), youth with DBD relative to healthy youth would show the following: reduced modulation of activity within vmPFC by reward-related EV when making optimal choices (i.e., when choosing the door associated with appetitive images);

and reduced modulation of activity within AIC, dACC, and caudate by threat-related EV in youth with DBD when making suboptimal choices (i.e., when not choosing the door associated with appetitive images or when choosing the doors associated with aversive images).

During the feedback phase, we predicted that youth with DBD would show, relative to healthy youth, both reduced positive PE modulation to appetitive outcomes and increased negative PE modulation to aversive outcomes in vmPFC and caudate.

METHOD

Participants

A total of 34 youth participated: 15 youth with DBD and 15 healthy comparison (HC) youth, 10 to 17 years of age (Table 1). Four additional youth underwent scanning (2 youth with DBD and 2 HC youth) but were excluded, as they opened all doors and thus prevented modeling of nonchoice behavior. Youth were recruited from the community through advertising and referrals from area mental health practitioners. A statement of informed assent and consent was obtained from participating children/adolescents and parents. This study was approved by the National Institute of Mental Health Institutional Review Board.

Exclusion criteria were pervasive developmental disorder, Tourette's syndrome, lifetime history of psychosis, depression, bipolar disorder, generalized, social, or separation anxiety disorder, posttraumatic stress disorder (PTSD), neurologic disorder, history of head trauma, history of substance abuse, and IQ of <70. All youth and parents completed Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS)¹⁹ assessments conducted by a doctoral-level clinician as part of a comprehensive psychiatric and psychological assessment. The K-SADS has demonstrated good validity and interrater reliability ($\kappa > 0.75$ for all diagnoses).¹⁹ The K-SADS assesses for substance abuse and substance dependence; however, because of exclusion criteria, no participants in either group met criteria for these diagnoses. IQ was assessed with the Wechsler Abbreviated Scale of Intelligence (2-subtest form). Youth meeting K-SADS criteria for CD or ODD were included in the DBD group, whereas comparison subjects did not meet criteria for any K-SAD diagnosis. In addition, parents completed the Inventory of Callous-Unemotional Traits (ICU),²⁰ a measure of CU traits.

Study Measures

Inventory of Callous-Unemotional Traits—The ICU is a 24-item parent-report scale designed to assess CU traits in youth. The ICU was derived from the CU scale of the Antisocial Process Screening Device²¹ that has been widely used in various youth samples. The construct validity of the ICU has been supported in community and juvenile justice samples.²²

Doors Task—Participants were presented with a novel task in which they were shown doors of 3 colors followed by images. The images were selected from the International Affective Picture System (IAPS)²³ and consisted of 48 physical threat (e.g., snake poised to strike), 48 contamination threat (e.g., feces-covered toilet), and 84 appetitive images (e.g.,

puppy) spread over six 4.5-minute runs. Images were matched for arousal across all 3 categories (described in Supplement 1, available online).

Before undergoing scanning, participants completed a practice “learning” condition in which the different doors were consistently paired with stimuli from the different categories (e.g., green door–physical threat picture; purple door–contamination threat picture; see Figure 1). This relationship of door color to feedback type remained constant for that participant across the practice session and the 6 fMRI runs. Although the relationship remained constant across trials, the relationship was probabilistic such that, for each door color, 60% of trials would result in 1 feedback type. The association of door color to feedback type was randomized across participants, as were trials within runs. However, for 40% of trials, the choice of door engendered an unexpected feedback type (i.e., for 40% of total trials, choice of the door associated with appetitive images resulted in physical or contamination threat images).

Trials involved 2 phases. During the choice phase, participants were presented with an image of a closed door (2000 milliseconds), and they chose to open or not to open the door via a button press. In 50% of trials, the door would open regardless of participant choice (participant choice was recorded for all trials regardless of choice efficacy). This phase preceded a 1,000- to 3,000-millisecond randomly jittered fixation period. Next, an open or closed door was presented for 500 milliseconds, depending on whether the participant would see an image (regardless of participant choice). Then, during the 2,000-millisecond feedback phase, either feedback (appetitive, physical threat, or contamination threat images) was presented, or, if the participant chose not to open the door in a trial in which participant response was considered, a blank screen was presented. A second 1,000- to 3,000-millisecond randomly jittered interval preceded the next trial.

Using participants’ behavioral data, including practice trial data, a learning curve was modeled and used to establish EV and PE. The EV for each door type was initially set to 0. The PE was then calculated based on the feedback (F), which was coded 1 (feedback image was consistent with the majority of images paired with that door; e.g., passing through the threat door led to a threatening image) or 0 (feedback image was inconsistent with the majority of images paired with that door; e.g., passing through the threat door led to an appetitive image) with the following formula:

$$PE_{(t)} = F_{(t)} - EV_{(t)}$$

where the PE for the current trial_(t) equaled the feedback value for the current trial minus the EV for the current trial. EV was calculated by the following formula:

$$EV_{(t)} = EV_{(t-1)} + (\alpha \times PE_{(t-1)})$$

where the EV of the current trial (t) equaled the EV of the previous trial (t-1) plus the PE of the previous trial multiplied by the learning rate (α), which was set at 0.1 following previous research.²⁴

MRI Parameters

Participants underwent scanning using a 1.5-T GE Signa scanner. A total of 118 functional images per run were taken with a gradient echo planar imaging (EPI) sequence (repetition time = 2,300 milliseconds; echo time = 30 milliseconds; matrix = 64×64 ; flip angle = 90° ; flip angle; field of view = 24 cm). Whole-brain coverage was obtained with 31 contiguous axial slices (thickness = 4 mm; in-plane resolution = 3.75×3.75 mm). A high-resolution anatomical scan (3-dimensional spoiled gradient recalled acquisition in a steady state; repetition time = 9 milliseconds; echo time = 2.872 milliseconds; field of view = 24 cm; flip angle = 20° ; axial slices = 124; thickness = 1.5 mm; matrix = 256×256) in register with the EPI data set was obtained covering the whole brain.

Imaging Data Preprocessing

Imaging data were preprocessed and analyzed in AFNI.²⁵ At the individual level, functional images from the first 6 repetitions, collected before equilibrium magnetization was reached, were discarded. Functional images from the 6 time series were motion corrected and spatially smoothed with a 6-mm full-width-at-half-maximum gaussian filter. The time series were normalized by dividing the signal intensity of a voxel at each point by the mean signal intensity of that voxel for each run and multiplying the result by 100. Resultant regression coefficients represented a percentage of signal change from the mean.

Nineteen task regressors were modeled. Nine indicator functions were modeled: doors opened (appetitive, contamination threat, physical threat), doors not opened (appetitive, contamination threat, physical threat), and feedback (appetitive, contamination threat, physical threat). These 9 regressors modeled the average response to door choices for each door type and feedback types. An additional 6 regressors were parametric modulator functions that multiplied the average response to the choice-phase regressors (doors opened and doors not opened: appetitive, contamination threat, physical threat) by EV. The 3 final regressors were parametric modulator functions that multiplied the average response to the feedback types (appetitive, contamination threat, physical threat) by PE. The 19th regressor modeled trials in which no response was recorded. No contrasts were modeled at this level. All regressors were created by convolving the train of stimulus events with a gamma variate hemodynamic response function to account for the slow hemodynamic response. The participants' anatomical scans were then individually registered to the Talairach and Tournoux atlas²⁶; studies have shown that normalization of brain volumes from age 7 to 8 years onward does not introduce major age-related distortions in localization or time course of the blood-oxygen-level-dependent (BOLD) signal in event-related fMRI.²⁷ The individuals' functional EPI data were then registered to their Talairach anatomical scan. Linear regression modeling was performed using the 19 regressors described above plus regressors to model a first-order baseline drift function. This produced a modulated and nonmodulated β coefficient and associated t statistic for each voxel and regressor.

fMRI Data Analysis

At the group level, results from the parametric modulator regressors were used. These 9 regressors were entered into 4 separate analyses of variance (ANOVAs) using 3dMVM in AFNI; for example, a 2 (group: DBD, HC) $\times$ 2 (door type: physical, appetitive) $\times$ 2 (choice:

open, not open) repeated-measures ANOVA was constructed. Importantly, 3dMVM is capable of modeling within-subject contrasts (door type and choice) and between-group (group) contrasts simultaneously. 3dMVM appropriately pools the variance for between-groups relative to within-subject contrasts. Following previous work^{9,28} and in the interest of conserving power, separate ANOVAs were conducted to compare physical threat stimuli to appetitive stimuli and contamination threat stimuli to appetitive stimuli. Initial thresholding was set at $p = .005$ with an extant threshold of 10 voxels, a combination recommended for an optimal balance between type I and type II errors.²⁹ Post hoc analyses of significant main effects and interactions were assessed with planned t tests within SPSS 19.0. As youth with DBD had significantly lower IQs than the HC youth (Results and Table 1), all analyses were repeated including IQ as a covariate. For all of the regions reported below, the introduction of the IQ covariate did not remove the significant interaction with group. In addition, these analyses were rerun in an IQ-matched sample of 22 youth (11 youth with DBD). Finally, because of reports of CU traits modulating neural response in youth with DBD, the relationship between CU traits and modulated activation in regions showing significant interactions of interest was examined.

RESULTS

The HC and DBD groups did not differ significantly on age, racial, or gender breakdown (Table 1), nor were there significant group difference in the numbers of censored TRs because of excessive movement ($t = 1.54, p = .14$). The HC group did have a significantly higher IQ [$t(28) = 3.438, p = .002$], and medications could not be withheld during scanning for 3 youth with DBD.

Behavioral Results

HC youth approached appetitive doors at a rate greater than chance ($t = 3.176, p = .007$), although they did not avoid either physical ($t = 0.413, p = .686$) or contamination threat doors ($t = 1.169, p = .262$) at rates greater than chance. Youth with DBD did not show any differences in their choices to open or not open any type of doors ($t = 0.223-1.529, p = 0.819-0.146$). A 2 (group: DBD or HC) $\times$ 3 (door type: physical threat, contamination threat, appetitive) repeated-measures ANOVA was conducted on percentage of trials during which participants chose to enter doors (Table 2). No main effect of group ($F_{2,27} = 0.074, p = .787$) or door type was observed ($F_{2,27} = 2.604, p = .092$). However, there was a significant group-by-door type interaction effect ($F_{2,27} = 3.835, p = .034$). Although there were no significant group differences for physical threat, contamination threat, or appetitive door choices (appetitive: $t = 1.45, p = .157$; physical threat: $t = 1.26, p = .231$; contamination threat: $t = 0.565, p = .577$), healthy youth were significantly less likely than youth with DBD to choose to enter physical threat relative to appetitive doors [$t(28) = -2.774, p = .010$], but not relative to contamination threat doors [$t(28) = -0.775, p = .455$]. In short, the group-by-door type interaction was influenced by both the appetitive and physical threat door choice behavior. A 2 (group: DBD or HC) $\times$ 3 (door type: physical threat, contamination threat, appetitive) repeated-measures ANOVA conducted on response latencies did not yield a significant effect of door type ($F_{2,27} = 1.468, p = .248$), group ($F_{2,27} = 2.446, p = .129$), or

group-by-door type interaction ($F_{2,27} = 0.543, p = .587$). IQ was not a significant covariate for either analysis.

fMRI Results

The current study sought to determine whether youth with DBD show aberrant sensitivity to environmental reinforcement EV and PE information in the context of a decision-making task. To conserve power,^{9,28} and given the behavioral results, separate ANOVAs were conducted to compare physical threat stimuli to appetitive stimuli and contamination threat stimuli to appetitive stimuli. Our first 2 analyses, two 2 (group: DBD, HC) $\times$ 2 (door type: physical or contamination threat, appetitive) $\times$ 2 (choice: open, not open) repeated-measures ANOVAs, were conducted on the choice-phase data modulated by EV. The second 2 analyses, two 2 (group: DBD, HC) $\times$ 2 (feedback type: physical or contamination threat image, appetitive image) repeated-measures ANOVAs, were conducted on the feedback-phase data modulated by PE. The key interactions with respect to our predictions for the choice phase (group-by-door type-by-choice) and the key interactions with respect to our predictions for feedback (group-by-feedback type) are described in the following results to provide tests of our a priori hypotheses. Complete results are presented in the Supplement 1, available online.

Choice Data Modulated by EV–Physical Threat

Group-by-Door Type-by-Choice Interaction—Regions showing a significant group (DBD, HC)–by-door type (physical threat, appetitive)–by-choice (open, not open) interaction included the bilateral caudate, a bilateral region of the thalamus/posterior cingulate cortex (PCC), a medial region of the thalamus and bilateral parahippocampal gyrus (Table 3, Figure 2). In these regions, healthy youth showed a greater differential in modulated response relative to youth with DBD when not choosing appetitive stimuli relative to choosing appetitive doors; that is, healthy youth showed an increased use of EV information within these regions when choosing suboptimally ($t = 2.172\text{--}2.754, p = .010\text{--}.044$), although this difference fell to trend levels in the right parahippocampal gyrus [$t(28) = 1.981, p = .057$]. Similarly, healthy youth showed a greater differential in modulated response within these regions, relative to youth with DBD, when not choosing relative to choosing physical threat doors, that is, when they were choosing suboptimally [$t = 2.220\text{--}2.765, p = 0.010\text{--}.035$], although this difference fell to trend levels in the right caudate [$t(28) = 1.883, p = .070$] and in the bilateral parahippocampal gyrus [$t = 1.951\text{--}1.792, p = .061\text{--}0.084$]. Given our a priori interest in AIC and dACC, we examined these regions with a region-of-interest (ROI) analysis using ROIs from previous work (small-volume correction initial threshold $p = .02, \kappa > 22.7$).³⁰ The same pattern of modulated activation was observed within the dACC ROI ($x,y,z: 4,28,37; \kappa = 48$); however, activation observed in left AIC did not survive small volume correction ($x,y,z: -37,-4,-7; \kappa = 6$).

Feedback Data Modulated by PE–Physical Threat

Group-by-Feedback Type Interaction—Regions showing a group (DBD, HC)–by-feedback type (physical threat, appetitive) interaction included right inferior parietal cortex (Table 3). In this region, healthy youth showed increased activation as a function of positive

PE relative to youth with DBD [$t(28) = 3.871, p = .001$] and increased deactivation as a function of negative PE relative to youth with DBD [$t(28) = 2.585, p = .015$].

Choice Data Modulated by EV–Contamination Threat

No regions of activation survived correction for multiple comparisons in the group-by-door type-by-choice interaction.

Feedback Data Modulated by PE–Contamination Threat

No regions of activation survived correction for multiple comparisons in the group-feedback type interaction.

Potential Confounds

Given the significant group IQ differences, the physical threat analyses were re-run in an IQ-matched sample of 11 youth with DBD and 11 HC youth [$t(20) = 1.397, p = .178$]. Given the lack of contamination threat findings in the initial analyses, these analyses were not re-examined. The preceding effects of interest remained with proximal activations in the same brain regions in both choice and feedback phases (Table S1, available online). However, in the choice-phase analysis, the right caudate and left caudate results were seen only at slightly more lenient thresholds ($p = .0054$ and $.0095$, respectively).

To account for possible effects of medication and comorbid attention-deficit/hyperactivity disorder (ADHD), the physical threat analyses were also re-conducted. The analyses were re-run excluding 3 youth with DBD whose medications could not be withheld during scanning and 7 youth with ADHD. In both cases, the effects of interest remained (Tables S2 and S3, available online).

Symptom Severity and Neural Response

The relationship between CU traits and the differential in modulated response when not choosing, relative to choosing to open appetitive and physical threat doors in regions showing a significant group-by-door type-by-choice interaction was examined. No significant relationship between CU traits and neural response was observed. Given the relatively small numbers, we conducted power analyses at the observed effect sizes to determine the sample sizes needed to reveal a significant relationship. This revealed sample sizes of between 85 (relationship between CU traits and PCC differential response when choosing relative to avoiding physical threat doors) and 5,103 would be needed to reveal statistical significance for these effect sizes.

DISCUSSION

This study sought to determine whether youth with DBD would show aberrant sensitivity to EV and PE information in the context of a decision-making task using environmental reinforcers. There were 4 main findings. First, decision-making deficits in youth with DBD relative to HC youth were observed using environmental reinforcers. Second, youth with DBD showed reduced modulation by EV information when choosing to open doors associated with threat and when choosing not to open doors associated with appetitive

stimuli in the caudate and thalamus. Third, significant differences in sensitivity to EV and PE information between the DBD and HC groups were observed only with respect to physical threat stimuli and not to contamination threat stimuli. Fourth, increased signaling as a function of positive PE and decreased signaling as a function of negative PE were observed in inferior parietal cortex during feedback.

Considerable work has shown impaired decision making in youth with DBD, especially those with CU traits, using monetary reinforcers.^{4,5,7,8,10} This study is the first to use environmental reinforcers. This study revealed that healthy youth were significantly less likely to open physical threat relative to appetitive doors than youth with DBD. However, the DBD group was as likely to avoid contamination threat doors relative to appetitive doors as healthy youth. Interestingly, different neural systems have been implicated in processing physical relative to contamination threats.³¹ Specifically, physical threats are associated with responses within the amygdala, whereas contamination threats are associated with responses within the insula.³¹ This is consistent with considerable data that suggest that youth with DBD show abnormal amygdala response to fear expressions,^{9,28,32} but that have generally not found insula dysfunction in youth with DBD^{7,8} (but see White *et al.*⁶).

In this study, youth with DBD showed reduced modulation by EV information when choosing to open doors associated with threat and when choosing not to open doors associated with appetitive stimuli in the caudate and thalamus. Moreover, at a less stringent threshold, the same pattern was seen in the dmFC/dACC, although not in the AIC. Although this finding must be interpreted cautiously because of its failure to survive correction for multiple comparisons at the a priori initial threshold,²⁹ this is interesting, as previous work has implicated the dACC, caudate, and thalamus (and AIC) in the avoidance of suboptimal choices.^{13,14} Furthermore, in our previous work caudate, the dACC and AIC showed reduced modulation by EV information during avoidance responses in youth with DBD.⁶ In other words, across the current study and our previous work,⁶ we have seen a reduced capacity for youth with DBD to use EV information to guide behavior away from poor decisions (i.e., not choosing appetitive doors and choosing threat doors). This is, of course, consistent with clinical observations of youth with DBD persistently violating rules and social norms despite multiple disciplinary interventions (e.g., suspension from school, incarceration).

It should be noted that 2 predictions were not confirmed by the current data. First, considerable work demonstrates that vmPFC shows modulation by reward EV when participants make choices.^{11,12} In contrast, youth with DBD have shown reduced responses to reward.⁵⁻⁷ However, in the current study, there was no indication of reduced modulation by EV within the vmPFC in the youth with DBD. This likely represents specific parameters of the current paradigm, as no modulation of vmPFC was seen in the main effect of choice, even when healthy participants were analyzed separately. Future work is needed to determine whether this represents the use of environmental reinforcers, type II error, or other factors.

Second, a prediction regarding PE was not confirmed by the data. Although youth with DBD did show reduced modulation of activity by PE information, this was seen within the

right inferior parietal lobule (iPL) rather than the striatum, as predicted. Although the iPL has been implicated in PE signaling,³³ this is less seen than striatal modulation by PE information.²⁴ Currently, it is unclear whether the lack of differences in striatal modulation as a function of PE reflects the current paradigm (the healthy youth showed no modulation by PE within these regions during feedback when studied independently), the use of environmental reinforcers, or type II error.

Notably, no relationship between dysfunction in decision-making paradigms and CU traits (e.g., lack of guilt and empathy) was observed. This is consistent with previous work that has found dysfunction during decision making in youth with DBD without regard for CU traits,⁵ and with our own previous work directly testing this association.^{6,34} This is in striking contrast to recent work demonstrating an association between CU traits and amygdala response to distress/fear cues.^{9,28,32,35} Further work is needed to determine whether the level of CU traits is associated only with empathy impairment and not decision-making impairment, and whether behavioral manifestations can be identified that are associated with dysfunction in the use of EV information (and in PE information in previous work⁶) observed here. However, it is important to remember that this null finding may reflect a lack of sufficient power to reveal a relationship between CU traits and computational dysfunction during decision making. The issue deserves greater attention from studies involving larger numbers, and limited conclusions should be drawn from these data.

There are 7 caveats to consider with respect to the current data. First, despite high rates of comorbid ADHD within the DBD group, an ADHD comparison group was not included in this study, as previous work had indicated that youth with ADHD do not present with the pathophysiology found in youth with DBD.^{7,9,10} Further mitigating this limitation, the subsequent analysis excluding youth with ADHD revealed proximal activations for the interactions and main effects. Second, medications for 3 youth with DBD could not be withheld during scanning. Mitigating this limitation, the subsequent ANOVA excluding these participants again identified activation in proximal regions. Third, the current study used environmental reinforcers, unlike previous research, which has used monetary reinforcers. Without directly comparing monetary and environmental reinforcers, the current study cannot identify the influence of this different class of reinforcer. Fourth, the DBD and HC groups significantly differed in terms of IQ. Mitigating this limitation, the results of the initial analysis survive post hoc analysis of covariance with covarying IQ. In addition, subsequent analyses with an IQ-matched sample identified significant effects in proximal regions. Fifth, continuous measures of psychopathology and substance abuse were not collected. Therefore, it is not possible to examine the influence of subclinical psychopathology here. Sixth, differences between volitional versus forced viewing of stimuli were not examined. Consequently, the impact of these differences cannot be assessed here. Finally, the initial threshold used in the current study is less stringent than some recommendations, potentially increasing the likelihood of type I error.

In summary, youth with DBD showed reduced modulation by EV information in response to negative expectancies in regions including the caudate and thalamus (and dACC at subthreshold levels) during a decision-making task using environmental, as opposed to

monetary, reinforcers. Importantly, these impairments were unrelated to levels of CU traits. The current data suggest that caudate dysfunction in DBD may represent a shared impairment irrespective of levels of CU traits. With the advent of the CU specifier in *DSM-5*, understanding the differences and commonalities between youth high and low on CU traits will become even more clinically relevant.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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FIGURE 1.

Doors task. The subject was required to indicate, via button press, whether to open the door or not. Note: Door color was probabilistically associated with contamination threat, physical threat, or appetitive reinforcers. Upward pointing hand denotes participant response.

FIGURE 2.

Diagnosis-by-door type-by-choice interaction in bilateral caudate in 15 youth with disruptive behavior disorders (DBD) and 15 healthy youth. Note: Healthy youth showed a greater differential in modulated response relative to youth with disruptive behavior disorders (DBD) when not choosing appetitive stimuli relative to choosing appetitive doors, and healthy youth showed a greater differential in modulated response within these regions, relative to youth with DBD, when not choosing relative to choosing physical threat doors. Healthy youth showed an increased use of expected value (EV) information within these regions when choosing suboptimally. *Significant group differences were observed in bilateral caudate during the choice phase.

TABLE 1
Characteristics of Youth With Disruptive Behavior Disorders (DBD) and Healthy Comparison Youth

Characteristic	Youth With DBD (n = 15)		DSM-IV Diagnoses		HC Youth (n = 15)	
	Mean	SD	Mean	SD	Mean	SD
Age (y)	14.36	1.98	14.04	2.30		
IQ ^a	93.47*	10.12	106.6*	10.80		
ICU	31.71***	11.28	14.53***	6.52		
	n	%	n	%		
Gender (female)	4	26.67	5	33.33		
DSM-IV Diagnoses	n	%	n	%		
CD	11	73.33	0	0		
ODD	4	26.67	0	0		
ADHD	7	46.67	0	0		

Note: ADHD = attention-deficit/hyperactivity disorder; CD = conduct disorder; HC= healthy comparison; ICU = Inventory of Callous-Unemotional Traits; ODD = oppositional defiant disorder.

^a Assessed with the Wechsler Abbreviated Scale of Intelligence (2-subtest form).

* $p < .05$;

*** $p < .001$.

TABLE 2

Task Performance by Group

	<u>Youth With Disruptive Behavior Disorders</u>		<u>Healthy Youth</u>	
	Mean	SD	Mean	SD
Percentage of doors that participants chose to open				
Door type				
Contamination threat	51.0	17.1	54.3	14.1
Physical threat	56.2	15.5	47.7	22.0
Appetitive	54.4	17.9	63.5	16.5
Difference in percentage of doors that participants chose to open				
Appetitive – contamination threat	3.4	26.5	9.3	14.8
Appetitive – physical threat	-1.8*	9.2	15.9*	22.9
Response latency				
Contamination threat	609.57	105.04	670.45	114.95
Physical threat	608.90	94.24	672.83	126.63
Appetitive	593.67	99.16	674.99	155.81
Difference in response latency				
Appetitive – contamination threat	-15.89	46.33	4.55	57.90
Appetitive – physical threat	-15.23	53.03	2.17	69.15

Note:

*
 $p < .05$.

TABLE 3

Brain Regions Demonstrating Differential Modulated Blood-Oxygen-Level-Dependent (BOLD) Responses During Task Performance in Choice Phase (Modulation by Expected Value) and Feedback Phase (Modulation by Prediction Error) in 15 Youth With Disruptive Behavior Disorders and 15 Healthy Comparison Youth.

Region ^a	Coordinate of Peak Activation ^b						p	Voxels
	Left/Right	BA	x	y	z	F (df = 1,38)		
Diagnosis-by-Door Type-by-Choice								
Caudate	Right		7.5	7.5	17.5	15.90	.0007	29
Caudate	Left		-4.5	7.5	20.5	15.07	.0009	12
Posterior cingulate cortex/thalamus	Left		-10.5	-31.5	11.5	18.54	.0002	115
Thalamus	Right		1.5	-13.5	11.5	16.10	.0004	112
Parahippocampal gyrus	Left		-28.5	-34.5	5.5	11.98	.0017	11
Parahippocampal gyrus	Right		28.5	-40.5	2.5	14.16	.0008	10
Diagnosis-by-Feedback Type								
Inferior parietal cortex	Right	40	52.5	-49.5	47.5	14.17	.0008	11
Lingual gyrus	Left		-22.5	-55.5	2.5	18.51	.0002	10

Note: BA = Brodmann areas; df = degrees of freedom.

^a According to Talairach Daemon Atlas (<http://www.nitrc.org/projects/tal-daemon/>).

^b Based on Talairach Daemon Atlas standard brain template.