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Neural response to monetary loss among youth with disruptive behavior disorders and callous-unemotional traits in the ABCD study

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ABSTRACT

Etiological models highlight reduced punishment sensitivity as a core risk factor for disruptive behavior disorders (DBD) and callous-unemotional (CU) traits. The current study examined neural sensitivity to the anticipation and receipt of loss, one key aspect of punishment sensitivity, among youth with DBD, comparing those with and without CU traits. Data were obtained from the Adolescent Brain and Cognitive Development (ABCD)[SM] Study (N = 11,874; Mage = 9.51; 48% female). Loss-related fMRI activity during the monetary incentive delay task was examined across 16 empirically-derived a priori brain regions (e.g., striatum, amygdala, insula, anterior cingulate cortex, medial prefrontal cortex) and compared across the following groups: (1) typically developing (n = 693); (2) DBD (n = 995), subdivided into those (3) with CU traits (DBD + CU, n = 198), and (4) without CU traits (DBD-only, n = 276). Latent variable modeling was also employed to examine network-level activity. There were no significant between-group differences in brain activity to loss anticipation or receipt. Null findings were confirmed with and without covariates, using alternative grouping approaches, and in dimensional models. Network-level analyses also demonstrated comparable activity across groups during loss anticipation and receipt. Findings suggest that differences in punishment sensitivity among youth with DBD are unrelated to loss anticipation or receipt. More precise characterizations of other aspects punishment sensitivity are needed to understand risk for DBD and CU traits.

Disruptive Behavior Disorders (DBD), including Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD), are among the most common childhood psychiatric disorders (Kazdin, 2010). Symptoms of DBD include acts of defiance, aggression, and the violation of others' rights, and are associated with increased risk for suicide, substance use, and incarceration (Brent, 1995), lower academic achievement and poor interpersonal functioning (Loeber et al., 1998), and greater likelihood of psychiatric disorders in adulthood (Frick and Viding, 2009; Schaeffer et al., 2003). These disorders represent a substantial public health burden (Rivenbark et al., 2018) and there is an urgent need to elucidate DBD risk factors to inform intervention.

1. DBD and loss sensitivity

Etiological models highlight reduced punishment sensitivity as a core risk factor for DBD, due to its impact on associative learning processes that motivate behavior change in response to negative consequences (Lykken, 1995; Fowles, 1980; Blair, 2001). Broadly, the study of punishment sensitivity focuses on individual differences in sensitivity to negative outcomes, like loss of money or points (Byrd et al., 2014; Lutz and Widmer, 2014). Decades of behavioral research suggest that youth with DBD are less likely to alter their behavior in response to negative outcomes, including monetary loss (see Byrd et al., 2014 for review). However, studies have not differentiated between distinct phases of loss

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processing (Murray et al., 2018; Byrd et al., 2014). Thus, it is unclear whether youth with DBD are less sensitive to impending loss (i.e., anticipation), rendering them less motivated to avoid it, or less sensitive to the receipt of loss, impeding their ability to associate behavior with negative consequences. Given that treatments for DBD encourage consistent implementation of negative consequences, like loss of privileges (Kaminski et al., 2008; Kazdin, 2010), and report only modest effect sizes with limited long-term effectiveness (Hawes et al., 2014), a more nuanced understanding of the neural mechanisms underlying sensitivity to loss anticipation and receipt could inform tailored intervention strategies.

2. Neural response to loss in youth with DBD

Investigating neural responses to loss anticipation versus receipt has the potential to clarify individual differences in punishment sensitivty among youth with DBD. Among healthy populations, studies have identified limbic (e.g., striatum, amygdala) and prefrontal (e.g., insula, anterior cingulate cortex [ACC], medial prefrontal cortex [mPFC]) regions that are important in the processing of negative outcomes, specifically monetary loss (Oldham et al., 2018; Delgado et al., 2000; Knutson et al., 2000; Dugré et al., 2018). While aberrant loss processing in these regions are implicated in other psychopathologies, including mood and anxiety disorders (Knutson et al., 2008) and substance abuse (Bjork et al., 2008), few studies have explored this mechanism among youth with DBD. Those that have rarely differentiate between loss anticipation and receipt (see Cohn et al., 2015; Huang et al., 2019 for exceptions), and findings have been mixed, with some studies showing reduced amygdala activation to loss among youth with DBD (Byrd et al., 2018; Cohn et al., 2015; Huang et al., 2019) and others reporting no associations between neural sensitivity to loss and DBD (Bjork et al., 2010).

Several gaps in the literature could explain these inconsistent findings. First, there is heterogeneity among DBD youth (Frick and Ellis, 1999), including evidence for an etiologically-distinct subgroup with co-occuring callous-unemotional (CU) traits (DBD + CU youth; e.g., lack of empathy and guilt; Waller et al., 2020b) who show more severe and persistent DBD symptoms (Byrd et al., 2012; Pardini et al., 2018). Notably, DBD + CU youth appear to be characterized by reduced punishment sensitivity (Byrd et al., 2014), which has been linked to reduced responsiveness to standard interventions for DBD (Hawes and Dadds, 2005; Haas et al., 2011). Although recent studies show no associations between CU traits and neural activation to the anticipation or receipt of loss (Cohn et al., 2015; Huang et al., 2019; Byrd et al., 2018), conclusions are limited by the use of small, predominantly male samples, with reduced power to detect potential group differences. Second, previous research exploring neural responses to loss among youth with DBD has focused almost exclusively on adolescence, which heralds increases in impulsivity, risk-taking, and substance abuse, as well as concomitant changes in the neural circuitry underlying loss processing (Bjork and Pardini, 2015). Investigating neural responses to the loss anticipation and receipt prior to adolescence, and the onset of more serious forms of psychopathology, can enhance our understanding of developmental trajectories of DBD by further clarifying when these process go awry. Finally, prior research has focused solely on loss processing in individual regions of interest (ROIs) among youth with DBD despite research suggesting these regions operate within an overarching neural network (Knutson et al., 2000; Dugré et al., 2018). Expanding this work to examine whether the shared coactivation among regions belonging to the same higher-order network explains brain-behavior associations (see Cooper et al., 2019) can further elucidate the neuroetiology of DBD.

3. Current study

To advance our understanding of punishment sensitivity as a risk mechanism for DBD and CU traits, the current study utilized a large sample from the landmark Adolescent Brain and Cognitive Development (ABCD)[SM] Study. We focused on a key aspect of punishment sensitivity – loss processing – given its well-documented associations with other forms of psychopathology (e.g., Knutson et al., 2008; Bjork et al., 2008) and its specific relevance to etiological and intervention models of DBD (Byrd et al., 2014). We examined group differences in neural sensitivity during the anticipation and receipt of monetary loss between: (1) DBD versus typically-developing (TD); (2a) DBD-only versus TD; (2b) DBD + CU versus TD; and (2c) DBD-only versus DBD + CU, and tested whether sex moderated these associations. We also explored the moderating effects of anxiety given its known associations with punishment sensitivity (Gray, 1975). Lastly, we used latent variable modeling to examine network-level activity to loss anticipation and receipt. Across all analyses, we focused on 16 cortical and subcortical ROIs (Fig. 1, Table S1) that have been consistently identified in fMRI meta-analyses of loss processing and DBD (see Alegria et al., 2016; Oldham et al., 2018; Dugré et al., 2018). We hypothesized that, relative to TD youth, youth with DBD would exhibit reduced activity across limbic and prefrontal regions to loss anticipation and receipt, and that these deficits would be most pronounced in DBD + CU youth. Moreover, we expect reduced network-level activity to loss anticipation and receipt among DBD youth relative to TD youth, with the most pronounced deficits among DBD + CU youth.

4. Methods

4.1. Participants

Participants were drawn from the ongoing longitudinal ABCD Study® with data from the annual 3.0 data release (https://data-archive.nimh.nih.gov/abcd). The ABCD Study recruited 11,874 healthy children, aged 9–10 years, to be followed into early adulthood, and the current study focuses on 1,688 children within four phenotypically-narrow groups. Participants across 21 study sites were primarily recruited through public and private elementary schools with sampling approaches intended to yield a final sample that approximated national sociodemographic characteristics (Garavan et al., 2018; Compton et al., 2019). Institutional review boards at participating universities approved all study procedures. Participants and their legal guardian provided written assent and consent to participate.

4.2. Measures

4.2.1. Disruptive behavior disorders (DBD)

DBD were classified using the Child Behavior Checklist (CBCL; Achenbach and Ruffle, 2000) DSM-5 oriented scales and the Schedule for Affective Disorders and Schizophrenia for school-age children (K-SADS-PL DSM-5; Kaufman et al., 1996). Parents completed the self-administered computerized version of each of these measures.

4.2.2. Callous-unemotional (CU) traits

We classified CU traits using a 4-item measure derived and validated in prior studies using ABCD data (Hawes et al., 2019; Waller et al., 2020a), which included one item from the parent-report CBCL ("lack of guilt after misbehaving") and three [reverse-scored] items from the parent-report Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997) (e.g., "is helpful if someone is hurt or upset"). This measure showed strong psychometric properties, including evidence of discriminant and convergent validity, measurement invariance, and independent replication (Hawes et al., 2019). Finally, we derived *maximum a posteriori* (MAP) scale scores that provided person-specific CU traits factor scores (see Supplementary Materials).

4.2.3. Potential moderators

Sex (0 = male; 1 = female) and the CBCL DSM-5 Anxiety Problems subscale were examined as potential moderators.

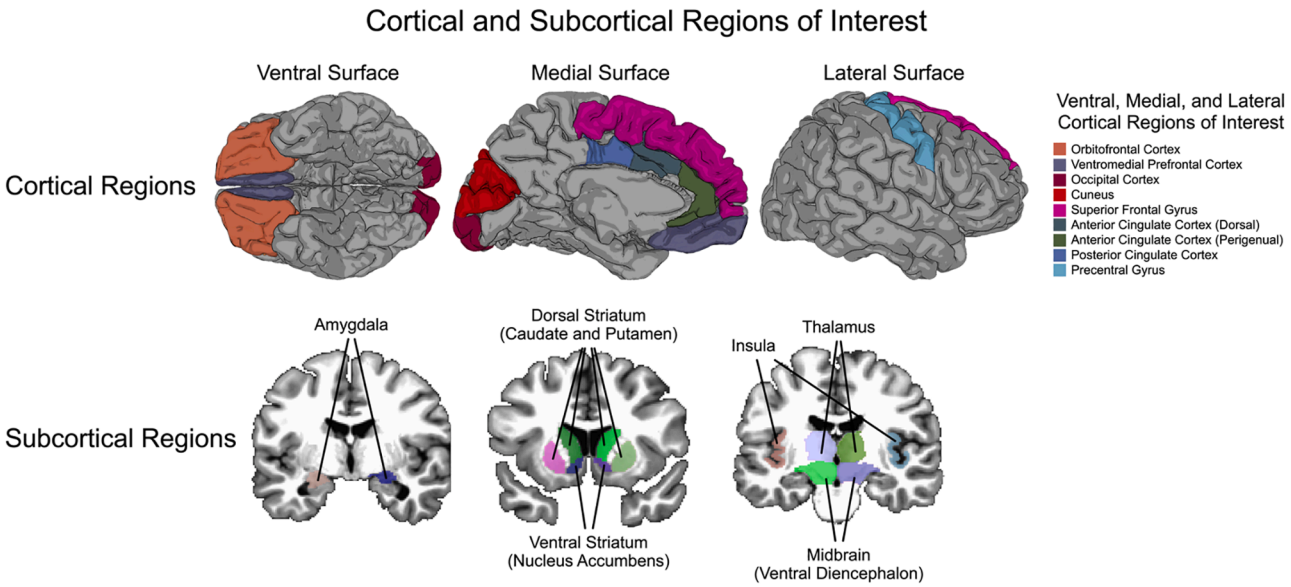


Fig. 1. Study regions of interest for loss sensitivity in children with disruptive behavior disorders and callous-unemotional traits.

4.2.4. Potential confounders

To account for any confounding effects of attentional, cognitive, or emotional difficulties that are comorbid with DBD, all analyses included CBCL DSM-5 attention-deficit/hyperactivity and internalizing problems subscales as covariates. We also covaried for sex, age, race/ethnicity, and parental education.

4.2.5. Group classification

We classified the presence of DBD based on youth scoring at or above the borderline clinical range (i.e., T-scores $\geq$ 67) on either the CBCL's DSM-oriented conduct problem *or* oppositional defiant problems scale, or receiving a K-SADS ODD or CD diagnosis (n = 995). Youth with DBD were further categorized based on CU traits. The DBD + CU group had high CU traits based on a conjunction of summed scores $\geq$ 4 on the summed CU traits measure *and* CU MAP scores $\geq$ 90th percentile (DBD + CU, n = 198) (see Supplementary Methods). To maximize phenotypic differences between groups, youth in the DBD-only group had summed scores of zero on the CU traits measure (n = 276). Typically developing youth were those with T-scores = 50 across all CBCL DSM-Oriented and Syndrome scales *and* summed scores of zero for CU traits (n = 693) (Table 1).

4.3. Imaging measures

4.3.1. Monetary incentive delay task

A version of the Monetary Incentive Delay (MID) task was used to measure brain activation during anticipation and receipt of three conditions (Casey et al., 2018): win ($0.20 or $5), loss (-$0.20 or -$5), or no incentive ($0). Participants saw a cue (pink circle/yellow square/blue triangle) at the beginning of trials indicating the valence (win/loss/no incentive) and amount of money at stake ($0/$0.20/$5). Cue presentation (2000 ms) was followed by a jittered anticipatory delay (1500–4000 ms). A black target shape (same shape as the previously presented cue) was then shown and participants could gain money or avoid losing money by pressing a response button while the target was onscreen. The time the target was onscreen was dynamically manipulated to maintain a 60% success rate. After a short response window, feedback was provided (2000 ms). For the anticipation phase, participants received 40 reward trials, 40 loss trials, and 20 neutral trials. For the outcome phase, the adaptive algorithm, on average, yielded 24 "positive" feedback trials (e.g., loss avoidance) and 16 "negative" feedback trials (e.g. loss receipt) (Casey et al., 2018). Participants completed two task runs each lasting approximately 5.5 min. We focused on two primary contrasts: (1) anticipation of large loss versus no incentive[2] and (2) loss receipt versus loss avoidance.

4.3.2. Image preprocessing and calculation of ROI data

The ABCD Data Analysis and Informatics Center (DAIC) performed centralized processing and analysis, leveraging validated methods used in other large-scale studies (see Supplementary Methods and Hagler et al., 2018; Casey et al., 2018). In brief, we analyzed parcellated cortical and subcortical regions already derived from cortical surface reconstruction and subcortical segmentation performed using FreeSurfer v5.3.0 (Fischl, 2012). Estimates of task-related activation were computed at the individual subject-level using a general linear model (GLM) implemented in AFNI's 3dDeconvolve and were released as contrast beta weights (Cox, 1996). For contrasts of interest, average GLM beta coefficients were computed separately for the two task runs and then averaged across runs. Brain activity was examined in 16 cortical and subcortical ROIs determined *a priori* (Fig. 1, Table S1). (see Supplementary Methods for additional information).

4.4. Analytic strategy

First, we used multinomial logistic regression to examine differences in loss-related brain activity within *a priori* ROIs (Fig. 1, Table S1) comparing: (1) DBD versus TD; (2a) DBD-only versus TD; (2b) DBD + CU versus TD; and (2c) DBD-only versus DBD + CU, while controlling for covariates. We also examined sex and anxiety[3] as potential moderators. Supplementary analyses were conducted to further explore study associations, including different covariate sets, alternative grouping approaches (i.e., more and less stringent), and zero-inflated dimensional (vs. group-based) models. Second, we tested a latent factor model to supplement the ROI analyses and explore group differences at a network-level (Fig. 2). We evaluated how all regions contributed to a hypothesized network during specific task phases and assessed whether the network could be modeled similarly across groups. We used

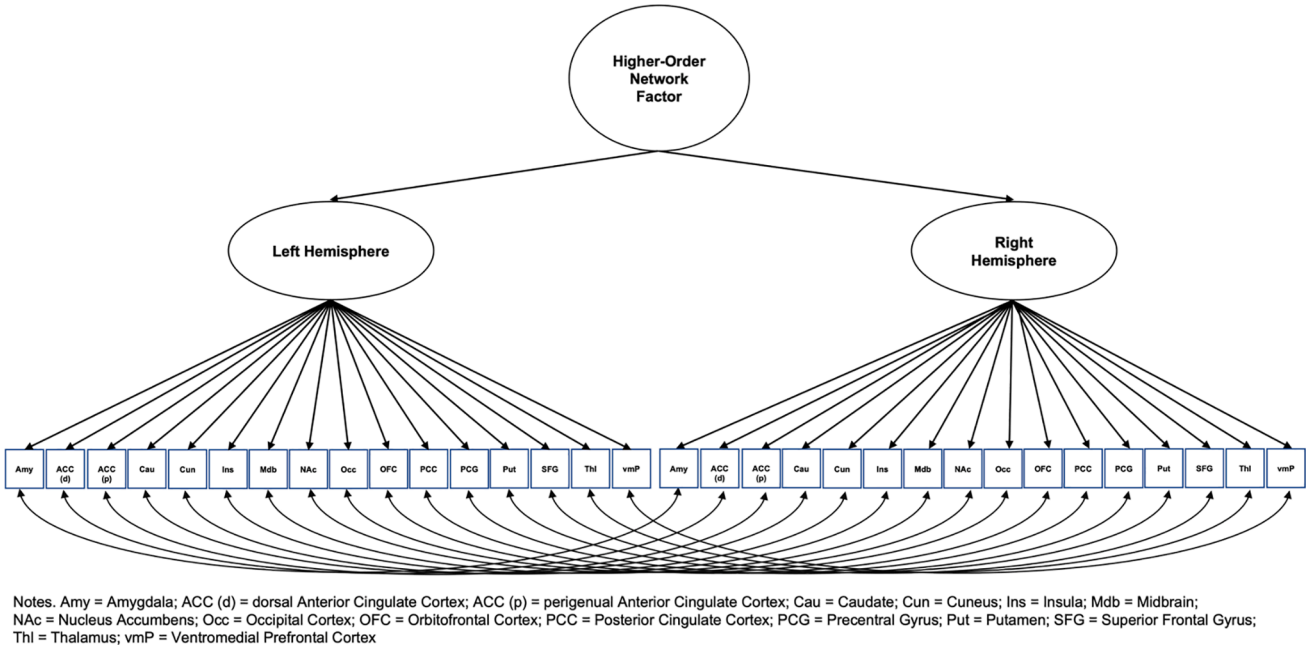
[2] In post-hoc analyses, we also explored the following two anticipation contrasts: (1) anticipation of small loss versus no incentive and (2) anticipation of large loss versus anticipation of small loss.

[3] The internalizing subscale was not included as a covariate when examining the moderating effect of anxiety.

Table 1
Descriptive statistics for all study variables.

	Diagnostic Group							
	DBD + CU		DBD-only		TD		Total	
	M/%	*SD*	*M*/%	*SD*	*M*/%	*SD*	*M*/%	*SD*
Demographic Variables								
Age	9.51[a]	0.50	9.49[a]	0.50	9.51[a]	0.50	9.49	0.49
Sex (% Male)	67%[a]	–	57%[a]	–	41%[b]	–	51%	–
Race/Ethnicity								
% Black	19%[a]	–	13%[a]	–	15%[a]	–	15%	–
% White	53%[a]	–	56%[a]	–	51%[a]	–	54%	–
% Hispanic/Latinx	8%[a]	–	11%[a, b]	–	14%[b]	–	13%	–
% Other	18%[a]	–	19%[a]	–	18%[a]	–	18%	–
Parental Education	16.42[a]	2.79	16.79[a]	2.22	16.74[a]	2.72	16.54	2.65
Diagnostic Criteria								
CBCL Conduct Problems	66.77[a]	7.71	55.11[b]	6.12	50.00[c]	0.00	56.22	8.36
CBCL Oppositional Defiant Problems	65.45[a]	7.88	57.73[b]	6.74	50.00[c]	0.00	56.46	8.11
CBCL Anxiety Problems	58.27[a]	9.11	58.11[a]	9.29	50.00[b]	0.00	53.46	6.12
CBCL Internalizing Problems	58.89[a]	11.35	56.04[a]	11.12	50.00[b]	0.00	52.26	9.51
CBCL Attention/Hyperactivity Problems	61.28[a]	8.34	55.80[b]	7.18	50.00[c]	0.00	54.89	7.36
K-SADS CD Diagnosis	35%[a]	–	16%[b]	–	0%[c]	–	11%	–
K-SADS ODD Diagnosis	42%[a]	–	77%[b]	–	0%[c]	–	37%	–
K-SADS ADHD Diagnosis	38%[a]	–	19%[b]	–	0%[c]	–	8%	–
CU traits Summed Score	4.83[a]	1.07	0.00[b]	0.00	0.00[b]	0.00	1.17	1.71
CU traits MAP Score	2.01[a]	0.42	−0.33[b]	0.16	−0.39[c]	0.16	0.02	0.92

Note. [a, b, c] Groups with matching superscripts do not differ significantly from each other on the corresponding variable. Groups with different superscripts indicate a significant difference between those groups on that variable. DBD+CU = High DBD/High CU Traits; DBD-only = High DBD/Low CU Traits; TD = Typically Developing. ADHD=attention deficit hyperactivity disorder; CBCL=Child Behavior Checklist: CD = conduct disorder; CU = callous-unemotional; K-SADS= Schedule for Affective Disorders and Schizophrenia for school-age children; MAP= *maximum a posteriori*.



Notes. Amy = Amygdala; ACC (d) = dorsal Anterior Cingulate Cortex; ACC (p) = perigenual Anterior Cingulate Cortex; Cau = Caudate; Cun = Cuneus; Ins = Insula; Mdb = Midbrain; NAc = Nucleus Accumbens; Occ = Occipital Cortex; OFC = Orbitofrontal Cortex; PCC = Posterior Cingulate Cortex; PCG = Precentral Gyrus; Put = Putamen; SFG = Superior Frontal Gyrus; Thl = Thalamus; vmP = Ventromedial Prefrontal Cortex

Fig. 2. Higher-order latent network factor in a study of loss sensitivity in children with disruptive behavior disorders and callous-unemotional traits.

measurement invariance testing (via the DIFFTEST procedure in MPlus 7; Muthén and Muthén, 1998–2012) to evaluate: (1) the network factor model across group (i.e., configural invariance), and (2) whether each region contributed to the higher-order network equivalently across groups (i.e., metric and scalar invariance) (see Supplementary Methods). We also tested for group differences in mean activity at the network-level, made more advantageous by modeling latent variables free of measurement error. All analyses were conducted with MPlus 7 (Muthén and Muthén, 1998–2012) using maximum likelihood estimation with robust standard errors (MLR) and a Monte Carlo numerical integration algorithm. Complex sampling and recruitment procedures for the ABCD Study were accounted for using CLUSTER correction (i.e., for sibling pairs) and stratification sampling (i.e., study site, scanner type) procedures (Muthén and Muthén, 1998–2012). Study outliers were winsorized at values +/-2.5 times the mean. Outlier cases did not exceed 5% for any region. To avoid Type 1 error inflation resulting from multiple comparisons, we applied the Benjamini-Yekutieli correction (Benjamini et al., 2006), with a false discovery rate of 5%.

5. Results

Descriptive statistics showed groups to be equivalent on

demographic variables, except for a lower proportional representation of female and Hispanic/Latinx children in the DBD groups (Table 1). There were no between-group differences on MID task performance (e.g., mean reaction time; Table S2).

5.1. Loss anticipation and receipt in individual regions of interest

Results from multinomial logistic regressions are shown in Table 2. No significant group differences were identified in any ROIs for loss anticipation[4] nor loss receipt and there were no significant interactions with sex or anxiety. Findings did not significantly differ when covariates were removed (Table S4).[5]

5.2. Network-Level responses using latent network factor models

Group differences in network-level responses were examined separately for loss anticipation and receipt. Model fit was in the acceptable range for all groups (Table S6), suggesting that a higher-order network was present across groups for both task phases.

Multigroup invariance testing revealed no significant differences in model fit between groups (Table S7), suggesting that the shared co-activation among study ROIs fit the hypothesized higher-order network model and that this shared co-activitation was the same (i.e., invariant) across groups. There were no differences in the mean-level network factor between groups (Table S8).

6. Discussion

Despite the central role of reduced punishment sensitivity in etiological models of DBD and CU traits (Lykken, 1995; Fowles, 1980), research characterizing the neural underpinnings of punishment sensitivity among DBD youth is sparse. We focused on brain activity during an important aspect of punishment sensitivity, loss processing, given prior literature showing its association with various forms of psychopathology (Knutson et al., 2008; Bjork et al., 2008) and its centrality to etiological and intervention models for DBD (Byrd et al., 2014). We examined brain activity during the anticipation and receipt of monetary loss during a key developmental period in late-childhood, leveraging the ABCD Study to derive phenotypically narrow groups in the largest DBD-focused loss processing fMRI study to date. Contrary to hypotheses, youth with DBD (irrespective of CU traits) did not differ from TD youth in neural responses to loss anticipation or receipt. Null findings were confirmed across several group-based and continuous analyses that examined activation within empirically-derived *a priori* ROIs. Moreover, we established a higher-order latent network factor that showed consistent activity patterns across groups, further highlighting that punishment sensitivity, operationalized as sensitivity to monetary loss, did not vary as a function of DBD or CU traits.

While the null findings appear to stand in contrast to long-standing theoretical and behavioral studies (Byrd et al., 2014), they offer clarity about the role of punishment sensitivity in DBD. The MID task separates loss and reward trials, and in doing so, isolates loss anticipation and loss receipt by inducing expectancies (i.e., youth know which cues signal potential loss and they believe they can avoid it). Our null findings suggest that, when examined in isolation, the motivation to avoid loss (anticipation) and receipt of *unexpected* loss may be intact among youth with DBD. Differences in punishment sensitivity may instead emerge in the context of competing rewards and punishments, when youth must learn by trial and error which behavior leads to negative consequences and adapt their behavior accordingly. This interpretation is consistent with evidence for deficits in processing prediction errors among DBD youth (Blair et al., 2018; White et al., 2016; White et al., 2013), which may underlie difficulties in shifting behavior when punishment avoidance requires overriding a previously-rewarded response. Indeed, behavioral and neuroimaging studies in youth with DBD show performance deficits and differences in neural processing during paradigms that require behavioral modification in the context of competing reward and punishment (e.g., passive avoidance, response reversal; Byrd et al., 2014; Blair et al., 2018). It is also possible that these individual differences are related to difficulties allocating attention to punishment that is less salient (Patterson and Newman, 1993) and/or enhanced sensitivity to reward, as was previously demonstrated in this cohort (Hawes et al., 2020). Taken together, findings from our large-scale investigation represent a critical step towards systematically addressing these questions, and highlight the need for continued work in this area.

An alternative explanation for our null findings center on the use of monetary loss, which may not be particularly salient in late childhood (Helfinstein et al., 2013). While the removal of an appetitive stimulus (e.g., monetary loss) is a negative outcome that *can* elicit negative feelings similar to the presentation of an aversive outcome (e.g., shock), the latter may more reliably produce neural activity and render stronger effects on behavior (Delgado et al., 2006). Related, reduced sensitivity to an aversive outcome (e.g., shock) may better characterize youth with DBD, particularly DBD + CU youth (Byrd et al., 2014). Additionally, research is needed to characterize neural responses during *other* aspects of punishment sensitivity, including associating cues with stimuli (i.e., classical conditioning) and associating behavior with consequences (i.e., instrumental conditioning), which may be impaired in youth with DBD and have important implications for etiological and intervention models. While the MID incorporates aspects of both associative learning processes, contingencies are fixed, limiting opportunities to assess learning. Systematic manipulation of these learning processes are needed to enhance our understanding of how punishment sensitivity goes awry among DBD youth with and without CU traits.

Results should be considered in the context of several limitations. First, the cross-sectional design precludes any examination of potential changes in punishment sensitivity over time; follow-up studies that utilize future waves of ABCD Study data are therefore paramount. Second, while we found no evidence that sex moderated our outcomes, the sample age (9–10 years old) could predate many of the sex-based differences in brain-behavior associations that emerge during adolescence following pubertal development (Kaczkurkin et al., 2019). Third, while the ROIs fit a higher-order network factor model, the model represents a simplified version, which likely encompasses an expanded set of brain regions (Oldham et al., 2018; Delgado et al., 2000; Knutson et al., 2000; Dugré et al., 2018). Finally, information about clinical services was not assessed.

In sum, youth with DBD (irrespective of CU traits) showed similar patterns of brain activity to the anticipation and receipt of loss relative to TD youth. Results were confirmed across group-based and continuous analyses that examined activation within empirically-derived ROIs, as well as in analyses that modeled broader network-level activation in a higher-order latent factor. Findings advance our understanding of punishment sensitivity in youth with DBD and suggest that well-documented behavioral differences are unrelated to the anticipation or receipt of loss. Future neuroimaging studies that consider how other aspects of punishment sensitivity increase risk for DBD and CU traits are needed, as this has important implications for intervention.

[4] Analyses examining potential differences in the intensity or magnitude of loss anticipation (i.e., small loss vs. no incentive and large loss vs. small loss) showed no group differences in activation for any of the ROIs (**Table S3**).

[5] Analyses utilizing alternative grouping approaches (i.e., both more and less stringent; **Table S4**), different covariate sets (**Table S4**), and continuous DBD and CU traits scores (i.e., zero-inflated models; see **Table S5**) showed no significant associations between DBD or CU traits and activation in any of the individual ROIs.

Table 2
Odds ratios comparing activation during loss anticipation and loss receipt across study groups.

	Planned Group Comparisons							
	Loss Anticipation				Loss Receipt			
	TD vs Overall ^DBD	TD vs ^DBD + CU	DBD-only vs. ^DBD + CU	TD vs. ^DBD-only	TD vs Overall ^DBD	TD vs. ^DBD + CU	DBD-only vs. ^DBD + CU	TD vs. ^DBD-only
Left Hemisphere								
Amygdala	0.94	1.09	1.03	1.06	0.86	0.81	0.89	0.90
dACC (dorsal)	0.95	1.04	1.06	0.98	0.99	1.14	1.09	1.03
pACC (perigenual)	0.87	1.10	1.04	1.05	0.84	0.73	0.94	0.76
Caudate	0.78	0.97	1.06	0.92	0.98	1.13	1.14	0.99
Cuneus	0.94	0.84	0.88	0.95	0.99	0.92	0.99	0.93
Insula	0.88	1.06	1.07	0.99	1.22	1.05	0.77	1.35
Midbrain	0.86	1.30	1.18	1.10	0.84	1.04	1.03	1.01
Nucleus Accumbens	0.99	1.11	0.97	1.14	0.89	0.90	0.99	0.91
Occipital Cortex	0.69	0.67	1.00	0.67	0.81	0.93	0.95	0.97
Orbitofrontal Cortex	0.65	0.66	0.99	0.66	1.10	0.95	0.80	1.19
Posterior Cingulate Cortex	0.91	1.03	1.10	0.94	1.14	1.10	0.82	1.33
Precentral Gyrus	0.99	1.11	1.00	1.11	0.87	0.74	0.85	0.86
Putamen	0.92	0.78	0.94	0.83	0.96	1.06	1.07	0.99
Superior Frontal Gyrus	0.87	0.95	0.97	0.97	1.25	1.48	1.10	1.34
Thalamus	0.76	0.73	0.91	0.80	0.81	1.08	1.13	0.95
vmPFC	0.67	0.73	1.02	0.71	0.96	0.86	0.85	1.00
Right Hemisphere								
Amygdala	0.96	1.09	1.05	1.03	1.15	1.21	0.89	1.35
dACC (dorsal)	0.92	1.15	1.06	1.07	0.75	0.78	1.04	0.74
pACC (perigenual)	1.04	1.04	1.07	0.96	0.84	1.04	1.03	1.01
Caudate	0.99	1.11	1.00	1.11	0.72	0.60	0.80	0.74
Cuneus	0.67	0.83	1.13	0.73	0.97	0.82	0.89	0.91
Insula	0.71	0.96	1.12	0.85	1.22	1.34	0.97	1.38
Midbrain	0.96	1.06	1.07	0.99	0.87	0.73	0.81	0.90
Nucleus Accumbens	0.86	1.30	1.18	1.10	1.06	1.10	0.96	1.13
Occipital Cortex	0.77	0.92	1.15	0.80	0.62	0.52	1.05	0.50
Orbitofrontal Cortex	0.76	0.69	0.91	0.76	1.25	1.48	1.10	1.34
Posterior Cingulate Cortex	0.92	0.97	1.01	0.95	0.95	0.74	0.77	0.95
Precentral Gyrus	0.99	1.14	1.09	1.03	0.87	0.85	0.87	0.98
Putamen	0.79	0.91	1.05	0.86	0.91	0.93	0.94	0.99
Superior Frontal Gyrus	0.86	0.81	0.89	0.90	1.15	1.16	1.06	1.09
Thalamus	0.64	0.74	0.90	0.82	0.99	0.80	0.84	0.95
vmPFC	1.08	1.34	1.12	1.18	0.87	0.74	0.85	0.86

Note. DBD = DBD overall; DBD + CU = High DBD/High CU Traits; DBD-only = High DBD/Low CU Traits; TD = Typically Developing; ACC = Anterior Cingulate Cortex; vmPFC = ventromedial prefrontal cortex, ^ = Reference group. Odds ratios are reported relative to the reference group. *O.R.'s* > 1 indicate increased activation among the non-reference group relative to the reference group (i.e., decreased activation in the reference group). *O.R.'s* < 1 indicate decreased activation among the non-reference group relative to the reference group (i.e., greater activation among the reference group).

CRediT authorship contribution statement

Amy L. Byrd: Conceptualization, Methodology, Writing – original draft, Writing - review & editing. **Samuel W. Hawes:** Conceptualization, Methodology, Formal analysis, Visualization, Writing - review & editing. **Rebecca Waller:** Conceptualization, Methodology, Writing - review & editing. **Mauricio R. Delgado:** Writing - review & editing. **Matthew T. Sutherland:** Writing - review & editing. **Anthony S. Dick:** Writing - review & editing. **Elisa M. Trucco:** Writing - review & editing. **Michael C. Riedel:** Writing - review & editing. **Ileana Pacheco-Colón:** Writing - review & editing. **Angela R. Laird:** Supervision, Project administration, Funding acquisition. **Raul Gonzalez:** Supervision, Project administration, Funding acquisition.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

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