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Antisocial behavior is associated with reduced frontoparietal activity to loss in a population-based sample of adolescents

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Original Article

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Abstract

Background. Adolescent antisocial behavior (AB) is a public health concern due to the high financial and social costs of AB on victims and perpetrators. Neural systems involved in reward and loss processing are thought to contribute to AB. However, investigations into these processes are limited: few have considered anticipatory and consummatory components of reward, response to loss, nor whether associations with AB may vary by level of callous-unemotional (CU) traits.

Methods. A population-based community sample of 128 predominantly low-income youth (mean age = 15.9 years; 42% male) completed a monetary incentive delay task during fMRI. A multi-informant, multi-method latent variable approach was used to test associations between AB and neural response to reward and loss anticipation and outcome and whether CU traits moderated these associations.

Results. AB was not associated with neural response to reward but was associated with reduced frontoparietal activity during loss outcomes. This association was moderated by CU traits such that individuals with higher levels of AB and CU traits had the largest reductions in frontoparietal activity. Co-occurring AB and CU traits were also associated with increased precuneus response during loss anticipation.

Conclusions. Findings indicate that AB is associated with reduced activity in brain regions involved in cognitive control, attention, and behavior modification during negative outcomes. Moreover, these reductions are most pronounced in youth with co-occurring CU traits. These findings have implications for understanding why adolescents involved in AB continue these behaviors despite severe negative consequences (e.g. incarceration).

Introduction

Antisocial behavior (AB) refers to acts of aggression, rule-breaking, and delinquency and is a significant public health concern due to the often chronic trajectory of AB and substantial long-term costs for perpetrators, victims of AB, and to society (Foster & Jones, 2005). Dysfunctional reinforcement processing is thought to be central to the development and persistence of AB across development (Blair, 2015; Fowles, 1980). In order to elucidate the biological processes that contribute to the persistent, maladaptive, and reward-driven behaviors that characterize AB, a growing body of research has reported links between AB and brain reactivity during reward and loss processing (Blair, Veroude, & Buitelaar, 2018; Byrd, Loeber, & Pardini, 2014; Murray, Waller, & Hyde, 2018).

Reward and loss processing are complex constructs that contain multiple components, including anticipation of, and hedonic responses to, reward and loss (Berridge & Robinson, 2003). These basic processes form the backbone of more complex reinforcement-based cognition, such as learning and decision-making. A common paradigm for studying the basic neural response to reward and loss is the monetary incentive delay task (MID; Knutson, Fong, Adams, Varner, & Hommer, 2001). Meta-analyses of the MID have found that reward and loss *anticipation* recruit similar brain regions, including the dorsal striatum, ventral striatum (VS), amygdala, insula, and supplementary motor cortex. Reward *receipt* recruits the VS, posterior cingulate, amygdala, and orbitofrontal cortex (Dugré, Dumais, Bitar, & Potvin, 2018; Oldham et al., 2018), whereas loss receipt recruits the dorsal striatum and medial prefrontal cortex (Dugré et al., 2018). In concert, these regions make up the reward processing circuitry that allows individuals to direct attention towards salient stimuli,

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evaluate potential rewards and consequences, and engage in behavior that maximizes reward and minimizes punishment.

Disruptions in several reinforcement processes are thought to contribute to persistent AB. For example, early etiologic models identified a preference for immediate, often risky rewards in individuals with AB (Quay, 1993). However, more recent models posit that a reduced responsivity to reward in brain regions associated with value-based decision making can lead to maladaptive, aggressive behavior (Blair et al., 2018). From these theoretical models, several studies have examined the neural mechanisms that underlie disrupted reward processing in youth AB (for reviews, see Blair et al., 2018; Byrd et al., 2014). These studies have generally demonstrated that youth with AB display reduced neural reactivity to reward, including during the reward receipt phase of the MID (Cohn et al., 2015), during risky-decision making (Crowley et al., 2010), and during passive avoidance learning (Finger et al., 2011; White et al., 2013). A recent study using the Adolescent Brain and Cognitive Development (ABCD) dataset found that children with disruptive behavior disorders (DBD) displayed reduced frontostriatal reactivity during reward anticipation and increased frontostriatal activity during reward receipt relative to typically developing youth (Hawes et al., 2021). However, a meta-analysis found no differences in reward-related brain reactivity between subjects with high AB compared to controls (Dugré et al., 2020).

Individuals who engage in persistent AB also appear to have disrupted punishment processing (Blair et al., 2018; Fowles, 1980), which may, in turn, impact their ability to modulate behavior in response to negative outcomes (Byrd et al., 2014). Several studies have investigated links between conduct problems and brain function in the context of punishment/loss-related decision making. For example, youth with conduct problems showed an impaired representation of expected value in frontoinsular regions when making avoidance choices (White et al., 2016), and increased striatal response to negative outcomes on passive avoidance tasks (White et al., 2013). In tasks probing brain responses to simple monetary loss, findings have been more mixed. Some studies have reported that conduct problems are linked to reduced amygdala activity to loss in clinical (Byrd, Hawes, Burke, Loeber, & Pardini, 2018), and community (Huang et al., 2019) samples, whereas others reported increased amygdala activity to loss in youth with persistent DBD (Cohn et al., 2015). However, a recent analysis of the very large ABCD dataset found no significant differences in loss-related brain responses in children with and without DBD (Byrd et al., 2021).

There are several possible explanations for the seemingly conflicting findings regarding the relationship between AB and reward/loss processing. First, AB is a heterogeneous construct with subgroups that have potentially different etiologies. Callous-unemotional (CU) traits–which include low empathy, remorse, and interpersonal affect–delineate a group of youth high on AB, with a more stable and severe pattern of AB, and different behavioral and biological correlates (Frick, Ray, Thornton, & Kahn, 2014). In some cases, links between AB and reward/loss processing were not impacted by the presence of CU traits in clinical (Byrd et al., 2018) and community (Murray, Shaw, Forbes, & Hyde, 2017) samples. However, others found that CU traits were related to increased (Hawes et al., 2021) or decreased (Cohn et al., 2015) amygdala activity during reward outcomes. Thus, it is still unclear whether CU traits are uniquely associated with reinforcement-related brain function and whether the presence of CU traits impacts links between AB and brain reactivity to reward and/or loss.

A second factor potentially contributing to conflicting findings is the considerable methodological heterogeneity across neuroimaging investigations of reinforcement processing. Reward and loss processing include distinct phases (e.g. anticipation, consumption, learning), which have unique patterns of neural activation across a network of corticolimbic brain regions (Berridge & Robinson, 2003; Knutson & Greer, 2008). Thus, studies that employ tasks such as the MID may show different associations with AB than studies that use reinforcement learning or decision-making tasks. Even within the MID, studies have demonstrated distinct patterns of brain activity to anticipation *v.* receipt of reward in youth and adult AB or healthy samples, which vary on psychopathic traits (Buckholtz et al., 2010; Hawes et al., 2021; Murray et al., 2018). Finally, few studies have examined associations between youth AB and brain reactivity during the anticipation and receipt of monetary loss (Byrd et al., 2021; Cohn et al., 2015), making it difficult to isolate specific sources of dysfunction in AB within the broader punishment sensitivity construct. Thus, studies that examine links between AB and the anticipation and receipt of reward/loss have the potential to identify specific mechanisms of neural dysfunction in AB.

Finally, most neuroimaging research on AB is conducted using either relatively small groups of youth with severe AB or samples of very healthy participants who reside near major research universities. Both sampling strategies have their respective strengths and limitations, either by examining neural response to reward in extreme groups, thus not capturing the dimensional nature of AB (Krueger, Markon, Patrick, Benning, & Kramer, 2007), or by recruiting non-representative, advantaged, and very healthy community samples, which may limit reliability and generalizability (e.g. across a range of racial/ethnic groups and socioeconomic status) (Button et al., 2013; Chiao & Cheon, 2010). With regard to the study of AB, it is especially important to recruit participants from underrepresented and underserved groups (e.g. African Americans) due to these groups' disproportionate exposure to systemic racism, which leads to greater exposure to low-income, dangerous neighborhoods, and places youth at greater risk for AB (Leventhal & Brooks-Gunn, 2000). Moreover, youth of color are more likely to experience harsher consequences for risky behavior than non-minority youth (e.g. incarceration) (Burch, 2015). Though the longitudinal ABCD study marks a significant step towards larger samples which include youth across the entire spectrum of AB and with greater representation of youth from different contexts and identities (and with a stronger sampling strategy than many studies), current analyses of this cohort are focused on baseline data (ages 9–10) and do not capture adolescence, a critical developmental period when AB becomes more common and severe (Shaw, Hyde, & Brennan, 2012). Questions of generalizability and replicability of neuroimaging findings to broader, more representative samples highlight the critical importance of examining brain-behavior relationships in well-sampled cohorts that include substantial representation of participants of color (Falk et al., 2013).

Current study

The current study seeks to fill gaps in the previous literature by investigating associations between AB and neural activity during both anticipatory and consummatory phases of reward and loss processing in a sample of primarily low-income adolescents, the

majority of which identify as youth of color. In addition to examining associations to neural response to both reward and loss, the study examines whether CU traits moderate associations between AB and neural activity. We hypothesized that AB would be associated with decreased VS response during reward anticipation (Hawes et al., 2021; Murray et al., 2017) and that CU traits would not be associated with reward-related reactivity (Murray et al., 2018). We hypothesized that AB would be associated with neural response to loss and that CU traits would moderate this association; however, given the limited and mixed research on neural response to monetary loss, we did not make specific directional hypotheses. We conducted targeted analyses in the VS region of interest (ROI), a key hub of the reward/loss processing circuitry and conducted exploratory whole-brain analyses to characterize links between AB, CU traits, and brain function in this unique sample.

Method

Participants

The Study of Adolescent Neural Development (SAND) included 237 youth who participated in the longitudinal Fragile Families and Child Wellbeing Study (FFCWS; Reichman, Teitler, Garfinkel, and McLanahan, 2001). The FFCWS is a population-based sample of infants born in hospitals in twenty US cities (population > 200 000) between 1998 and 2000, oversampled 3:1 for non-marital births. Based on this sampling frame, the resulting sample was enriched for families with lower-income and substantial representation of families of color. Families in the FFCWS completed interview-based assessments when target youth were 1, 3, 5, 9, 15 years old. SAND consists of youth born in three of the twenty cities: Detroit, MI, Toledo, OH, and Chicago, IL. Of the 123 youth with usable fMRI data in the present analyses (online Supplementary Table S1), the average age was 15.9 years (range 15.0–17.6); 59% were female; 76% identified as Black/African American, 12% identified as white/European American, and 41.5% had a family income <$25 000/year (online Supplementary Table S2). Youth also completed self-report questionnaires and a psychiatric interview. Primary caregivers completed questionnaires and a psychiatric interview about themselves and their child. The University of Michigan institutional review board approved all procedures.

Measures

Monetary incentive delay task

Participants completed a point-based, modified version of the MID task during fMRI (Murray, Lopez-Duran, Mitchell, Monk, & Hyde, 2020). The task consisted of two 45-trial, 9.4-min runs. Trials consisted of a cue indicating the trial type (potential win, loss, or neutral), then a variable fixation crosshair delay, then a target (white square), followed by a jittered inter-trial interval. Participants responded to the target with their right index finger to win or avoid losing points. Task difficulty was adjusted so that each participant successfully responded to ~50% of the trials. The task produces a robust response in the extended corticolimbic circuit and is able to separate the anticipation and receipt of reward and loss via additional jitter (2–4 s) between trials (Murray et al., 2020).

Antisocial behavior

We used several indicators to create a multi-informant, multi-method dimensional measure of AB as done previously in this sample (Dotterer et al., 2020). AB was assessed using (a) total score from the youth-reported Self-Report of Delinquency (Elliott, Huizinga, & Ageton, 1985), (b) rule-breaking, and (c) aggression subscales from the parent-reported Child Behavior Checklist (CBCL; Achenbach, 1991), and (d) combined lifetime CD and oppositional defiant disorder (ODD) symptom counts from the clinician-rated Schedule for Affective Disorders and Schizophrenia for School-Age Children diagnostic interview (K-SADS; Kaufman et al., 1997). The measures were modestly-to-strongly correlated (range, $r = 0.30–0.81$, all $ps < 0.001$). To create a dimensional measure of AB, we used confirmatory factor analysis (CFA) in Mplus (Version 7.3; Muthén & Muthén, 2014) with maximum likelihood estimation with robust standard errors. Scale loadings on the latent AB factor were moderate-to-high ($\beta = 0.39–0.93$, $p < 0.001$; online Supplementary Table S3).

Assessment of callous-unemotional traits

We used several indicators to create a multi-informant, multi-method dimensional measure of CU traits (Dotterer et al., 2020), (a) parent-reported and (b) youth-reported Inventory of Callous-Unemotional Traits (Frick, 2004), and (c) clinician-rated total lifetime symptom counts from the Michigan Addendum to the K-SADS (Walker et al., 2020), which consisted of items derived from the Clinical Assessment of Prosocial Emotions (CAPE; Frick, 2016). The measures were modestly correlated ($r = 0.13–0.35$, all $ps < 0.05$). A latent factor was created in Mplus (Muthén & Muthén, 2014) using CFA with full information maximum likelihood estimation with robust standard errors. Scale loadings were moderate-to-high ($\beta = 0.35–0.86$, $p < 0.001$; online Supplementary Table S4).

Other variables included as covariates

To account for potential demographic or developmental effects, we included the following covariates: (a) parent-reported annual family income, (b) age (months), (c) gender, (d) pubertal development (Petersen, Crockett, Richards, & Boxer, 1988), (e) self-reported race, a social construct, used here to address differences in exposures to personal and systemic racism (coded dichotomously into all other reported race/ethnicities *v.* non-Hispanic white/European to account for youth likely *v.* unlikely to experience marginalization), and (f) standard scores from the Peabody Picture Vocabulary Test (Dunn & Dunn, 1997), administered at the age 9 FFCWS visit, were used to control for intellectual ability.

Analysis

Bold fMRI acquisition and pre-processing

Youth were scanned with a GE Discovery MR750 3 T MRI scanner with an 8-channel head coil. T1-weighted gradient-echo images were taken before the functional scans (TR/TE = 9.0/1.8 ms; TI = 500 ms; flip angle = 15°; FOV = 26 cm; slice thickness = 1.4 mm; 256 × 256 matrix; 40 slices). Functional T2*-weighted BOLD images were acquired using a reverse spiral sequence with interleaved contiguous axial 3 mm slices (TR/TE = 2000/30 ms; flip angle = 90; FOV = 22 cm) aligned with the AC-PC plane. Functional images were positioned to maximize limbic coverage. An auto-shimming procedure was conducted to reduce field inhomogeneity. The standard pre-processing procedure from the University of Michigan fMRI Center was applied, including removing outliers from the raw *k*-space data, image reconstruction, fieldmap correction, and slice-timing correction. Using SPM12 (http://www.fil.ion.ucl.ac.uk/spm/), high-resolution

anatomical images were re-oriented to the AC-PC plane, gray matter segmented, and functional images were realigned, co-registered, normalized, and smoothed with an 8 mm FWHM Gaussian filter.

Artifact detection Tools (ART) software (http://www.nitrc.org/projects/artifact_detect/) was used to identify motion outliers (>2 mm movement or 3.5° rotation) that were removed in the statistical model via spike regression. Due to our focus on the VS, participants were only included in analyses if there was a minimum of 70% coverage in the VS ROI. Two spheres of 10 mm radius were created in WFU PickAtlas Tool v3.0.5 around the Montreal Neurological Institute (MNI) coordinates $x = \pm 12$, $y = 12$, and $z = -10$ to encompass the bilateral VS (Murray et al., 2017). Data were also visually inspected for signal drop out (particularly in frontostriatal reward circuitry). Four subjects were excluded for having several large, or many small movement artifacts, despite having fewer than 5% outlier scans identified with ART. Analyses including these subjects are similar to those reported in the main text (online Supplementary Table S5).

In addition to testing associations between AB and reward- and loss-related neural response in the VS ROI using small volume correction, we also conducted whole-brain analyses (masked to include only gray matter). 3dClustSim (Cox, Chen, Glen, Reynolds, & Taylor, 2017) was used for multiple comparison correction using a Monte Carlo simulation to achieve a correction of $p < 0.05$. Spatial autocorrelation (ACF) values for a random 10% of the sample were calculated from the first-level model residuals using 3dFWHMx in AFNI. ACF values (mean values: 0.512, 6.737, 12.656) were used in 3dClustSim to estimate the noise smoothness using a Gaussian plus mono-exponential function. We used a voxel-wise correction of $p < 0.001$. The resulting cluster thresholds were $k = 3$ for the VS ROI and $k = 81$ for whole-brain analyses.

Neural response to reward and loss in AB

Neuroimaging analyses were conducted in SPM12. Separate multiple regression analyses of AB and CU traits as predictors were examined in contrasts allowing for the separation of anticipation and consumption phases: (1) Reward Anticipation > Reward Win (2) Loss Anticipation > Loss Outcome. Contrasts of (3) Reward Win > No-Win and (4) Loss Outcome > No-Loss were also tested to examine the neural response to successful and unsuccessful reward and loss outcomes. We also examined whether CU traits moderated associations between AB and neural response.

We present the results of models that include the primary predictor (i.e. AB or CU traits) and covariates, and for the moderation analyses, the AB × CU interaction term, the main effects of AB and CU traits, and covariates. We also ensured that regression assumptions were met, including residual normality and homoscedasticity. Analyses of the zero-order associations (e.g. AB without covariates) and analyses accounting for shared variance of AB and CU traits (e.g. AB controlling for CU traits) were highly similar (online Supplementary Table S6).

Results

Is AB related to neural response to reward?

AB was not associated with neural reactivity to Reward Anticipation > Reward Outcome, nor Reward Win > No Win contrasts.

Is AB related to neural response to loss?

AB was associated with several large clusters of reduced reactivity during Loss Outcome > No-Loss (Table 1, Fig. 1), including the bilateral inferior, middle, and superior frontal gyri, inferior and superior parietal lobules, precentral gyrus, cerebellum, fusiform and lingual gyri, and superior occipital gyri. AB was not associated with reactivity to Loss Anticipation > Loss Outcome, or with loss-related reactivity in the VS ROI.

Are CU traits related to neural response to reward?

CU traits were not associated with neural reactivity to Reward Anticipation > Reward Outcome, nor Reward Win > No Win contrasts.

Are CU traits related to neural response to loss?

CU traits were associated with reduced neural response to Loss Outcome > No Loss in the middle temporal gyrus and inferior and middle frontal gyri (Table 1, Fig. 2). These regions overlapped with the AB analysis and did not survive when controlling for AB (online Supplementary Table S6). CU traits were not associated with Loss Anticipation > Loss Outcome, or loss-related reactivity in the VS ROI.

Do CU traits moderate the neural response to reward and loss in AB?

There was a significant interaction between AB and CU Traits for both loss-related analyses. Specifically, the interaction of AB and CU traits predicted increased activity in the right precuneus/angular gyrus during Loss Anticipation > Loss Outcome, and this effect was driven by a reduced response in those with low CU traits (Table 1, Fig. 3).

The interaction of AB and CU traits predicted reduced frontoparietal reactivity to Loss Outcome > No Loss such that the pattern of reduced activation was strongest for those with high levels of CU traits (Table 1, Fig. 1*c*). Frontoparietal clusters largely overlapped with the AB analysis.

Exploratory examination of aggression v. rule-breaking

Because AB is a heterogeneous construct (Frick & Viding, 2009), we wanted to examine whether the results were driven by aggression or rule-breaking subscales of the CBCL, despite them being highly correlated. We found that results were similar for aggression and rule-breaking. However, when we controlled for their overlap, findings appeared to be driven by rule-breaking (online Supplementary Table S7).

Discussion

The current study characterized associations between AB, CU traits, and reward- and loss-related brain reactivity in a population-based sample of youth. We improved upon previous research by using a task designed to measure neural responses to anticipation *and* receipt of reward *and* loss and examining whether CU traits impacted findings using a multi-method multi-informant latent variable approach. Contrary to hypotheses, AB and CU traits were not associated with neural reward processing. Instead, AB and CU traits were associated with differences in

Table 1. Neural reactivity during reward and loss processing in antisocial behavior and Callous Unemotional traits

	t	Cluster size	MNI coordinates	Brain region
Reward Anticipation > Reward Outcome				
No significant clusters				
Reward Win > No Win				
No significant clusters				
Loss Anticipation > Loss Outcome				
AB × CU Traits	4.82	430	30 −64 40	Precuneus, angular gyrus
Loss Outcome > No Loss				
AB	−6.02	3551	−18 −66 56	Superior and inferior parietal lobule
	−5.89	1234	−44 12 26	Inferior and middle frontal gyri
	−5.37	857	48 38 12	Inferior and middle frontal gyri
	−5.21	231	22 −6 48	Precentral gyrus, superior frontal gyrus
	−4.81	206	2 40 38	Superior medial frontal gyrus
	−4.56	123	−8 −38 6	Posterior cingulate, white matter, hippocampus
	−4.30	440	−22 −68 −16	Cerebellum, fusiform gyrus, lingual gyrus, white matter
CU Traits	−5.07	150	−42 −64 8	Middle temporal gyrus
	−4.51	479	−44 18 40	Middle frontal gyrus
	−4.22	140	36 8 34	Inferior frontal gyrus
AB × CU Traits	−6.80	2893	24 −58 62	Superior parietal lobe, precuneus, angular gyrus
	−6.57	1240	−50 10 28	Inferior frontal gyrus, precentral gyrus
	−5.92	538	−30 −48 40	Inferior and superior parietal lobe
	−5.59	217	56 −62 −2	Inferior temporal lobe, fusiform gyrus
	−5.23	533	46 4 22	Inferior frontal gyrus, inferior frontal operculum
	−5.06	316	50 34 14	Inferior and middle frontal gyri
	−4.77	129	−22 18 14	Caudate, white matter
	−4.31	445	0 36 14	Anterior cingulate, superior middle frontal gyrus

Note: Associations between AB factor scores, CU traits factor scores, and neural response to reward and loss. All models included age, gender, pubertal status, annual family income, self-reported race, and intellectual ability as covariates and were significant at $p < 0.001$ and $\alpha = 0.05$., cluster threshold $k = 81$. Regression assumptions were met, including residual normality and homoscedasticity. Supplemental analyses of zero-order associations and analyses controlling for the overlap between AB and CU traits were highly similar (online Supplementary Table S6).

neural loss processing. AB was associated with decreased reactivity across a widespread frontoparietal network during loss outcomes, and this effect was strongest for youth with higher levels of CU traits. Finally, although AB and CU traits were not independently linked to neural response during loss anticipation, the interaction of AB and CU traits was associated with greater precuneus activity during loss anticipation. Our findings provide support for loss-processing as an important target for continued research to inform our understanding of AB. Moreover, they underscore the importance of investigating the unique and moderating effects of CU traits in youth who engage in AB and the decomposition of anticipation *v.* receipt of reward and loss.

AB was associated with reduced frontoparietal activation during loss outcomes. Additionally, we found that this pattern of findings seemed most closely associated with rule-breaking (as opposed to aggression), an important point and avenue for future research. The regions of reduced activation, including the inferior and superior parietal lobes and inferior and middle frontal gyri, correspond with key nodes of the frontoparietal control network (Seeley et al., 2007). This network is engaged during tasks requiring behavioral and cognitive control, and components of this network have distinct connections with the default mode network and dorsal attention network (Dixon et al., 2018). Prior work has found that oppositional traits were linked to reduced resting-state functional connectivity in frontoparietal networks youth with ADHD (Lin, Tseng, Lai, Matsuo, & Gau, 2015), and a meta-analysis found that AB was linked to reduced activity to punishment in the precentral gyrus (Dugré et al., 2020), which spatially overlaps with our findings. Finally, our results show spatial overlap with meta-analytical findings of reduced resting-state functional connectivity in AB, particularly between the dorsomedial PFC and superior parietal lobule (Dugré & Potvin, 2021). Thus, our results may suggest that individuals with AB have dysfunctional processing of punishment in regions associated with cognitive control and attention. Indeed, conduct problems have been related to reduced recruitment of frontoinsular regions involved in representing expected value when making loss-related decisions (White et al., 2016), and reduced frontal and parietal activity during response inhibition tasks (for review, see Blair et al., 2018). While speculative, it is possible that reduced frontoparietal activity to loss may impair reinforcement learning and lead to abnormal representations of expected risk/reward in more complex decision-making and learning tasks.

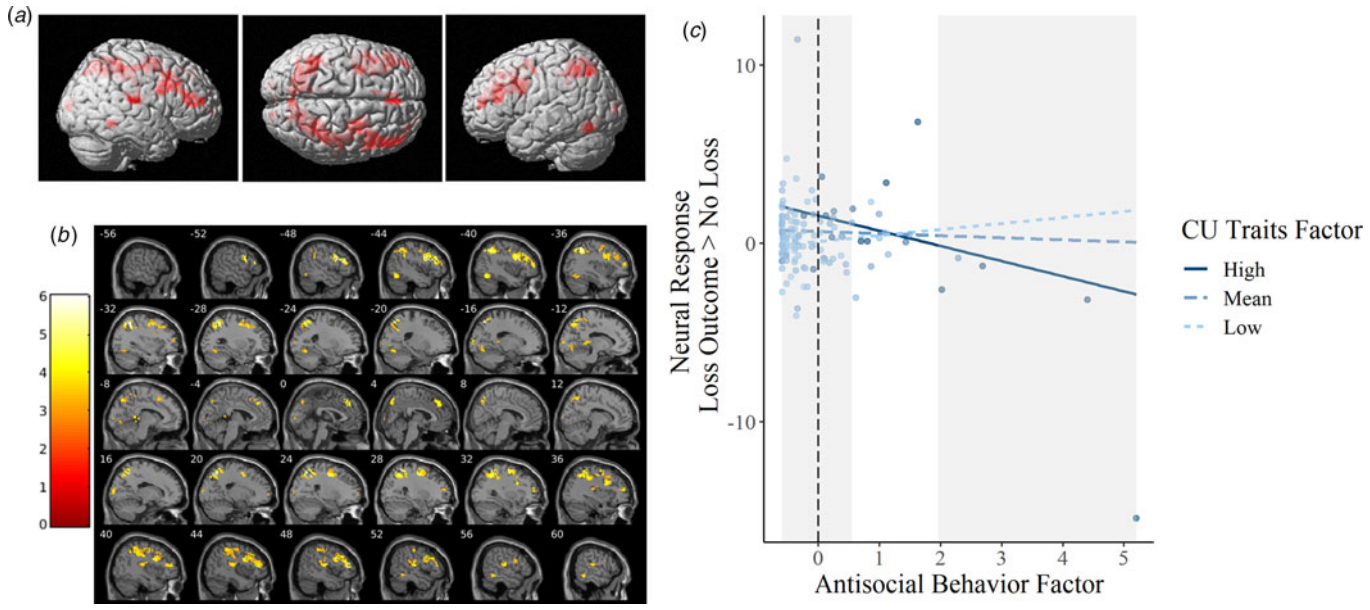

Fig. 1. Callous-unemotional traits moderate associations between antisocial behavior and frontoparietal activation during Loss Outcome. (*a*) Cortical surface rendering of the association between AB and neural response to Loss Outcome > No Loss. (*b*). Multi-slice activation map of the association between AB and neural response to Loss Outcome > No Loss. (*c*) Association between AB and neural response to Loss Outcome > No Loss as a function of CU traits. Mean cluster beta-weights were extracted at from the middle/inferior frontal gyrus cluster ($t = -5.89$, $k = 1234$, MNI: $x = -44$, $y = 12$, $z = 26$). Simple slopes plotted at mean, high, and low levels CU traits. High CU traits were coded as+ 1 s.d. from the CU traits factor mean, whereas low was coded as the lower bound of the CU traits factor due to there being no subjects 1 s.d. below the mean. The gray area indicates the level of AB at which the association is significant (AB factor score < 0.55 and >1.96).

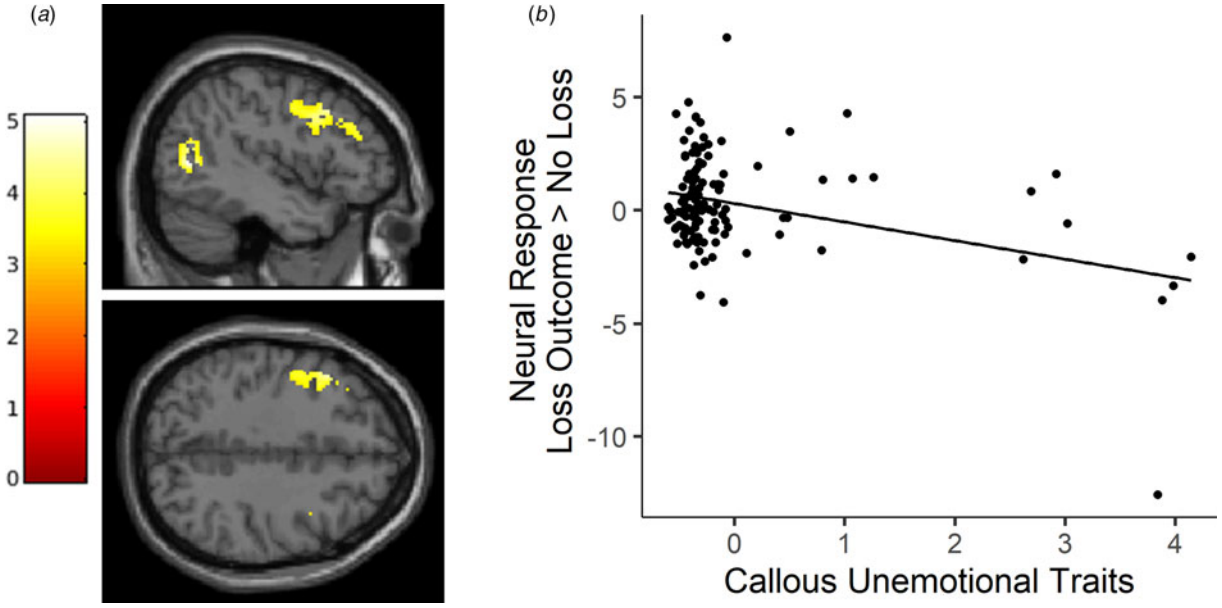

Fig. 2. Callous-unemotional Traits are associated with reduced middle frontal gyrus activity during Loss Outcome. (*a*) The CU traits factor score was associated with reduced response in the middle frontal gyrus during Loss Outcome > No Loss (centered at peak voxel MNI: $x = -44$, $y = 18$, $z = 40$; $t = -4.51$, $k = 479$). (*b*) Scatterplot of association between CU traits and neural response to Loss Outcome > No Loss.

The association between AB and reduced reactivity to loss was most pronounced in youth with higher CU traits. This finding is consistent with behavioral research indicating that individuals with AB, particularly those with CU traits (Byrd et al., 2014; Frick et al., 2014), may have impaired punishment sensitivity and have difficulty modulating responses in the context of competing reward and punishment (Newman & Kosson, 1986). Notably, our findings conflict with a recent study of loss-related brain reactivity in the ABCD study, which reported no significant differences between children with DBD (irrespective of CU traits) and typically developing children (Byrd et al., 2021). It is possible that frontoparietal dysfunction to loss identified here is more evident during adolescence, given the significant neurodevelopment during this period (Spear, 2011) which may impact reinforcement

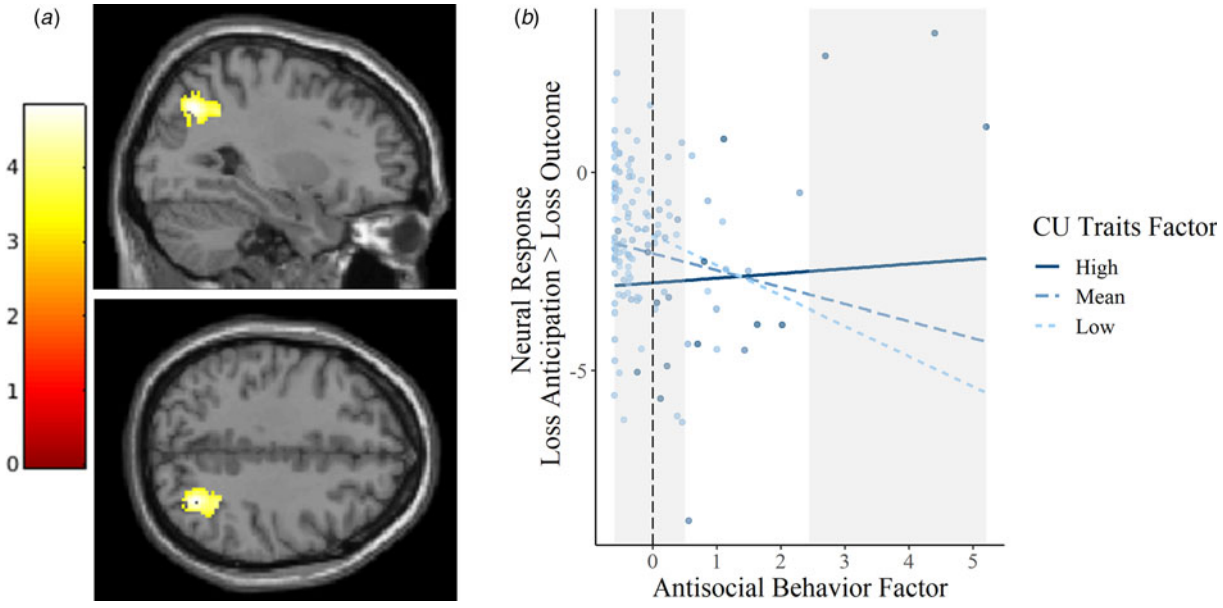

Fig. 3. Callous-unemotional traits moderate associations between antisocial behavior precuneus activation during Loss Anticipation. (*a*) CU traits significantly moderated the association between AB and neural response in the precuneus/angular gyrus during Loss Anticipation > Loss Outcome (centered at peak voxel MNI: $x = 30$, $y = -64$, $z = 40$; $t = 4.82$, $k = 430$). (*b*) Association between AB and neural response to Loss Anticipation > Loss Outcome as a function of CU traits. Mean cluster beta-weights were extracted at from precuneus/angular gyrus cluster ($t = 4.82$, $k = 430$, MNI: $x = 30$, $y = -64$, $z = 40$). Simple slopes plotted at mean, high, and low levels CU traits. High CU traits were coded as +1 s.d. from the CU traits factor mean, whereas low was coded as the lower bound of the CU traits factor due to there being no subjects 1 s.d. below the mean. The gray area indicates the level of AB at which the association is significant (AB factor score < 0.50 and >2.44).

processing, regulation, and AB (Steinberg et al., 2018). Longitudinal studies will help to characterize potential changes in punishment sensitivity across development and their relationship with AB.

There was also a significant interaction of AB and CU traits during loss anticipation. AB was associated with increased precuneus and angular gyrus reactivity during loss anticipation in those with higher levels of CU traits, but reduced reactivity in those with lower levels of CU traits. The precuneus and angular gyrus have multiple functions, including attention, multisensory integration, and mentalization (Cavanna & Trimble, 2006; Seghier, 2013). They are also nodes of the default mode network, which is typically downregulated during goal-directed activity (Buckner, Andrews-Hanna, & Schacter, 2008). Previous research found that individuals with psychopathic traits had attenuated down-regulation of the default mode network (including the precuneus) during a go/no-go task (Freeman et al., 2015), and reduced resting-state coupling between the default mode network and attention networks (including between the precuneus and PFC) in AB (Tang, Jiang, Liao, Wang, & Luo, 2013). Our findings may suggest that during loss anticipation, youth high on AB and CU traits have altered activity in regions previously shown to deactivate during goal-directed tasks, which may impair their ability to appropriately allocate attention to guide behavior.

It is surprising that we found no associations between AB or CU traits and neural response to reward, given previous literature linking these traits to reward-related neural dysfunction, particularly in the VS (Byrd et al., 2014; Hawes et al., 2021; Murray et al., 2018). Moreover, it is important to note that the findings we did have in other regions were exploratory in nature. However, a meta-analysis of youth and adults did not find significant associations between neural reward function and AB (Dugré et al., 2020), though the authors emphasized caution given the small number of studies in this area. Although we found a reduced frontoparietal activity to loss, we did not find associations between AB or CU traits in other regions implicated in loss processing, such as the dorsal striatum or amygdala (Dugré et al., 2018), at our stringent whole-brain correction. Future studies may want to examine these regions using small volume correction, or with larger samples to increase power (though see, Byrd et al., 2021).

The results of the current study should be considered in the context of several limitations. First, to avoid potential confounding effects in our primarily low-income sample, the MID task provided point-based incentives (instead of money, which may be valued differentially depending on family income). Point rewards may not provide the same motivational salience as monetary rewards. Second, we did not record participant MID responses that occurred outside of the response window. Thus, we were unable to distinguish too-slow responses from non-responses. We addressed this issue by carefully inspecting the data and excluding subjects with inconsistent responding. Nevertheless, we are encouraged that the task main effects (Murray et al., 2020) are consistent with prior meta-analyses of the MID (Dugré et al., 2018; Oldham et al., 2018). Third, intelligence was not assessed at the current study wave. To address this issue, we used a measure of intellectual ability from the age 9 assessment as a covariate, which did not impact the findings. Finally, the population-based sampling strategy and dimensional approach for assessing AB and CU traits is consistent with dimensional conceptualizations of psychopathology (Insel et al., 2010). However, although several participants ($n = 10$) had a lifetime diagnosis of CD and/or ODD, the subsample was too small for case-control analyses. Our findings may not generalize to samples with more severe AB and require replication in clinical/forensic samples to determine whether the current associations exist linearly across a wider range of AB and CU traits.

Conclusion

In a well-sampled cohort of adolescents from medium-sized US cities and primarily low-income environments, AB was associated with decreased frontoparietal response to loss, with results being more pronounced in youth with higher levels of CU traits. However, AB was not associated with reward-related neural response. These results demonstrate that AB is associated with reduced recruitment of regions involved in attention and executive control in response to negative outcomes, which may explain why antisocial youth persist in harmful, risky behaviors despite severe consequences. Improving our understanding of the role of reward and loss-processing in AB and CU traits is key for improving the treatment and prevention of this costly mental illness.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0033291722000307

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Conflict of interest. None.

Ethical standards. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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