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Reward-Related Neural Correlates of Antisocial Behavior and Callous–Unemotional Traits in Young Men

Laura Murray, Daniel S. Shaw, Erika E. Forbes, and Luke W. Hyde

ABSTRACT

BACKGROUND: Individuals involved in antisocial behavior often engage in excessive reward-driven behavior even in the face of severe punishments, including incarceration. However, the neural mechanisms of reward processing in antisocial behavior have not been examined while considering the heterogeneity of antisocial behavior and specific phases of reward and loss processing. In this study, we investigated the relationship among antisocial behavior, callous–unemotional (CU) traits, and neural activity during the anticipation and receipt of rewards and losses.

METHODS: A community sample of 144 low-income, racially diverse urban male individuals at risk for antisocial behavior completed self-report measures, a clinical interview, and a functional magnetic resonance imaging scan at 20 years of age. Neural response during the anticipation and receipt of monetary rewards and losses was linked to antisocial behavior and CU traits using a priori ventral striatum region of interest analyses and exploratory whole-brain analyses.

RESULTS: Antisocial behavior, but not CU traits, was related to less ventral striatum response during reward anticipation. There were no significant relationships between neural reactivity and antisocial behavior or CU traits during reward or loss outcomes. Antisocial behavior was also related to less ventrolateral prefrontal cortex reactivity during reward and loss anticipation.

CONCLUSIONS: These findings support a hyporeactivity model of reward and loss anticipation in antisocial behavior. Lower striatal reactivity to cues of reward and lower prefrontal regulatory recruitment during reward and loss anticipation may contribute to maladaptive reward-related behavior found in antisocial behavior.

Keywords: Antisocial behavior, Callous–unemotional, fMRI, Loss, Reward, Ventral striatum

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Antisocial behavior (AB), which includes aggression and rule breaking, is the cornerstone of the diagnoses of conduct disorder in youths and antisocial personality disorder (APD) in adults (1). AB is an important public health concern because of the large financial and emotional costs to perpetrators, victims, and society (2). Recently, neuroimaging research has focused on connecting emotional deficits seen in AB, such as abnormal fear processing, to altered function in limbic and prefrontal neurocircuitry (3,4). However, individuals high on AB also show marked behavioral differences in response to reward (5). For example, they perseverate on previously rewarded but now punished behaviors, engage in greater risk taking, and are less sensitive to punishments and losses (5–9). Improved understanding of the neural bases of these behavioral deficits is key to understanding the etiology of AB and informing biologically based assessment and treatment.

Reward Processing in AB

The few studies investigating reward-related neural activity in relation to AB focus on the ventral striatum (VS), a region active during reward evaluation, anticipation, and receipt (10). These studies have yielded conflicting results. Two studies have linked AB to greater reward-related VS reactivity in youths (11) and healthy adults (12), consistent with studies of substance abusers who show neural hypersensitivity to highly valued rewards (i.e., drug cues) (13–15). These studies suggest that individuals high on externalizing/AB may be hypersensitive to rewards, leading to reward-dominant behavior. Conversely, two other studies have linked AB to less reward reactivity in youths with persistent disruptive behavior disorders (16) and in undergraduates (17). This pattern of hyporeactivity to rewards parallels the lower VS reactivity found in those with attention-deficit/hyperactivity disorder (ADHD) (18,19) and in substance users when responding to nondrug rewards. This pattern is hypothesized to drive maladaptive reward-seeking behavior via attempts to normalize reward-related neural reactivity by pursuing progressively more intense rewards (20–22). Based on the conflicting findings for AB, research is needed to identify the extent to which those engaged in AB may be better characterized by hypersensitivity versus hyposensitivity to highly valued cues (i.e., monetary reward).

One potential explanation for the heterogeneity of findings of reward-related neural functioning in AB may be the failure of

previous studies to discriminate between phases of reward and loss processing. Human and animal research demonstrates that reward anticipation and receipt have dissociable neural networks (14,23) and may be differentially implicated in AB and externalizing disorders (11,12,16). Thus, neuroimaging studies of AB that discriminate between anticipation and receipt of reward are needed.

Beyond reward-related reactivity in the VS, a broader literature on decision making and learning suggests that AB is linked to dysfunction in prefrontal regions during tasks that tap emotion regulation, affective decision making, and learning (i.e., orbitofrontal cortex/ventromedial prefrontal cortex [PFC]) (24–28) as well as affective responses to reward (i.e., ventrolateral PFC [vlPFC]) (29,30). In Blair's model of AB, impairment in prefrontal functioning that leads to deficits in cognitive control and reward-dominant behavior is central to the etiology of broad disinhibited externalizing behaviors, including AB, ADHD, and substance use (31). These studies and theoretical models suggest that beyond the VS, reward-related processing is likely to elicit AB-related differences in the medial and lateral PFC (32).

Dimensions of AB

Beyond the need to separately examine phases of reward, little existing research has examined whether reward-related neural activity may differentiate different types of AB (12,17). Research examining dimensions of AB and the callous–unemotional (CU) traits prominent in adult psychopaths and youths diagnosed with the DSM-5 "limited prosocial emotions" specifier to conduct disorder (1) has demonstrated divergent relationships among AB, CU traits, and emotion-related amygdala reactivity (33,34). These studies suggest that dimensions of AB and CU traits should be examined separately in relation to neural reactivity. In one of the few studies parsing AB versus CU trait dimensions in relation to reward-related neural reactivity, we showed that antisocial, but not affective, components of psychopathy were associated with reduced reward-related VS reactivity (17). However, that study used a sample of healthy college students, highlighting the need for studies of those with a greater range of AB.

Current Study

The current study aimed to elucidate the reward-related neural underpinnings of AB in a diverse community sample enriched for AB by sampling young men who were raised in low-income urban environments. We examined the impact of the phase of reward (i.e., anticipation vs. receipt/outcome) on the association between neural reactivity and AB while leveraging multimethod assessment of AB through self-report, diagnostic interview, and official report as well as self-reports of CU traits. Finally, we examined these questions at the transition to adulthood when serious AB peaks and when youths transition to more independence and the adult legal system.

Based on previous findings (5), we hypothesized that AB would be related to greater VS reactivity during reward anticipation but not during reward outcome. Because of the lack of concern about performance that often characterizes individuals high on CU traits (1), we hypothesized that CU traits would be related to decreased VS reactivity during reward outcome, reflecting reduced sensitivity to the receipt of rewards.

METHODS AND MATERIALS

Participants

Participants were part of the Pitt Mother and Child Project, an ongoing longitudinal study of 310 low-income boys and their families recruited in 1991 and 1992 from Allegheny County Women, Infant, and Children Nutritional Supplement Clinics when boys were between 6 and 17 months old (35). This community sample was at high sociodemographic risk for AB based on being male, urban, and primarily low income (at initial recruitment, per capita income was $241 per month). The sample was also racially diverse (i.e., 53.5% European American and 36.0% African American of those included at 20 years of age). Target children and their mothers were seen almost yearly from age 1.5 to 20 years in the laboratory and/or home with assessments that included questionnaires, a psychiatric interview, and (at age 20) a functional magnetic resonance imaging (fMRI) scan. Participants were reimbursed after each assessment, and all procedures were approved by the University of Pittsburgh Institutional Review Board. Retention rates were high at each time point, with behavioral and fMRI data on 186 participants at 20 years of age (35,36). After excluding for motion, task, and signal-related errors, 144 men had usable fMRI data (Supplemental Table S1).

Measures

Self-Report Measures. AB was assessed using the 53-item Self-Report of Antisocial Behavior Questionnaire (37). Items probing alcohol and drug use were removed to reduce the possibility that substance use could explain any potential findings. The remaining 41 items were summed to form a dimensional measure of AB ($\alpha = .84$). CU traits were measured using a sum of 5 items from the CU factor of the Antisocial Process Screening Device (38) as described previously in this sample ($\alpha = .58$) (39).

Interview Measures. APD was assessed by trained interviewers using the Structured Clinical Interview for DSM-IV for Axis II personality disorders (40). Cases approaching diagnosis were reviewed by a licensed clinical psychologist (39). As reported previously, at 17 years of age, 35 of 250 participants (14%) met diagnostic criteria for conduct disorder, and at 20 years of age, 34 of 254 participants (13%) met criteria for APD (39). In the current sample, 8% ($n = 11$) met criteria for APD. Thus, rates of diagnosis were above national prevalence estimates (i.e., APD rate is 5.5% in male individuals) (41) but below forensic/clinical samples, consistent with an at-risk community sample (42). Based on research emphasizing the dimensional nature of AB (43,44), for the current analyses APD symptoms were summed to create a dimensional measure of AB. For covariates, we used the Structured Clinical Interview for DSM-IV (45) to assess for lifetime symptom counts of major depressive episode, generalized anxiety disorder, and substance use disorders.

Court Records. Records of adult violent charges (e.g., homicide, arson, sexual assault) were collected using the Pennsylvania state public court records website. These