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A Systematic Review Examining the Link Between Psychopathic Personality Traits, Antisocial Behavior, and Neural Reactivity During Reward and Loss Processing

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Abstract

Antisocial Behavior (AB) has a tremendous societal cost, motivating investigation of the mechanisms that cause individuals to engage and persist in AB. Recent theories of AB emphasize the role of reward-related neural processes in the etiology of severe and chronic forms of AB, including antisocial personality disorder and psychopathy. However, no systematic reviews have evaluated the hypothesis that reward-related neural dysfunction is an etiologic factor in AB in adult samples. Moreover, it is unclear whether AB is linked to a hyper- or hyposensitive reward system and whether AB is related to neural sensitivity to losses. Thus, the current systematic review examined whether AB (including antisocial personality disorder) and psychopathic traits are related to neural reactivity during reward processing, loss processing, or both. Our review identified seven task-based functional MRI or functional connectivity studies that examined associations between neural response to reward and loss, and dimensional and categorical measures of adult AB and/or psychopathy. Across studies, there was evidence that AB is associated with variability in neural functioning during both reward and loss processing. In particular, impulsive-antisocial traits appeared to be specifically associated with hypersensitivity in the ventral striatum during the anticipation, but not the receipt, of rewards.

Keywords

antisocial; fMRI; loss; psychopathy; reward

Antisocial behavior (AB), including physical, verbal, and sexual aggression, risk-taking behaviors, and theft, confers a tremendous cost to victims, families, and society (McCollister, French, & Fang, 2010). AB is associated with trait impulsivity, emotion reactivity, and is a core defining feature of the diagnosis of antisocial personality disorder

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(APD) in the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2013). Psychopathy is a related personality disorder that captures individuals who engage in AB and have extreme personality traits, including callousness, manipulativeness, irresponsibility, and shallow affect (Hare, 2003). Consistent with the theoretical structure of psychopathy as measuring both AB and personality traits, the most commonly used measure of psychopathy, the Psychopathy Checklist–Revised has two factors: Factor 1 assesses affective-interpersonal personality features (e.g., shallow affect, lack of remorse and empathy, grandiosity, and lying), and Factor 2 assesses impulsive-antisocial behaviors (e.g., lack of long-term goals, criminality, and history of serious AB; Hare, 2003). Although most individuals who meet criteria for psychopathy also meet criteria for APD, those with APD often display high levels of AB (captured by Factor 2), but do not have Factor 1 personality traits associated with psychopathy (Hare, 2003). Thus, a large group of individuals engage in serious AB with many of these individuals meeting criteria for APD, whereas a much smaller group of individuals engage in AB and also show signs of serious personality dysfunction and meet criteria for psychopathy.

Theories emphasize the role of affective, reward, and impulsivity deficits in the etiology of AB and psychopathy (Blair, 2015). A long history of behavioral research has established that altered reward-related behavior is critical to the emergence and persistence of AB and related emotional and personality features (Newman & Kosson, 1986). In recent years, neuroimaging techniques (e.g., functional magnetic imaging; fMRI) have enabled researchers to probe the neural mechanisms underlying AB and psychopathy. Consistent with behavioral studies, neuroimaging studies that have examined reward-related neural circuitry have linked AB to dysfunction in frontostriatal reward circuitry, and deficits in reward-related frontostriatal function are now central to etiological theories of chronic AB, as well as psychopathy (Blair, 2015). However, this literature has not been systematically reviewed. The primary goal of this article was to systematically review studies that had examined whether differences in neural response to reward and loss were related to AB and psychopathic personality traits.

Disrupted Behavioral Responsivity to Reward and Loss in AB

A long history of behavioral research has examined how AB is related to differences in reward-related behavior. Although AB likely arises from the complex interaction of deficits in disinhibition, socioemotional processing, and reward-dominant behavior, much of the *persistence* of AB may be linked to deficits in responding to and then learning from reward and loss. For example, those engaging in chronic AB often persist in illegal or risky behaviors despite the potential for severe punishment (e.g., incarceration), which implies that the systems responsible for reward valuation, loss processing, and learning may be disrupted in those who engage and persist in AB (Newman & Kosson, 1986). Early research on AB drew on Gray's behavioral activation system (BAS; approach to reward) and behavioral inhibition system (BIS; inhibiting behaviors that result in aversive outcomes; Gray, 1970). For example, Quay (1993) suggested that AB develops from an overactive BAS, leading to reward-dominant behavior (Quay, 1993). In contrast, others suggested that AB emerged from an *underactive* BAS, with reward seeking being a way to "normalize" an underactive system (Cloninger, 1987). These two positions leave open the question of

whether individuals high on AB have over- or underactive initial responses to rewards. Key theories have also hypothesized that AB emerges from a weak BIS (i.e., insensitivity to punishment; Fowles, 1980; Lykken, 1995), and more recent research suggests that AB arises from both an overactive BAS and an underactive BIS (Hoppenbrouwers, Neumann, Lewis, & Johansson, 2015). This research emphasizes the importance of reward systems in the etiology of AB, the need to examine both reward and loss in AB, and the need to examine whether these reward systems are over-versus underactive.

In support of some of these theories, behavioral research suggests that individuals engaging in chronic and severe AB may be hypersensitive to rewards and insensitive to losses. Adults engaging in severe AB continue pursuing rewards even as contingencies change and result in loss, as exemplified in research using response reversal tasks (Budhani, Richell, & Blair, 2006) and passive avoidance tasks (Newman & Kosson, 1986). More broadly, those high on externalizing psychopathology (including AB, impulsivity, and substance use) are hypersensitive to motivationally salient information, such as rewards, threat, and drug cues (Baskin-Sommers & Newman, 2013). Together, behavioral research supports the notion that both AB and broader forms of externalizing psychopathology are characterized by hypersensitivity to reward cues. Notably, however, behavioral research is limited in the extent to which it can measure individual differences in immediate reward response and thus cannot identify distinct phases of reward processing, including the anticipation versus receipt of reward. Extensive work with both human and nonhuman animal models suggests that the anticipation and consumption of rewards have dissociable frontostriatal circuits (Berridge & Robinson, 2003; Buckholtz et al., 2010). This line of research has greatly aided our understanding of the development and maintenance of substance use disorders (Robinson & Berridge, 2001), which are highly prevalent among individuals engaging in AB. Together, this prior research highlights the need to separately examine neural responses during anticipating versus receiving rewards in those with AB. Understanding these neural mechanisms can elucidate specific deficits in AB and, by extension, may identify targets for intervention (e.g., focus on motivational processes vs. consequences of behavior or pharmacological treatments that target mesolimbic hyper/hyporeactivity).

Brain Regions Implicated in Reward Response in AB

The most widely studied anatomical structure involved in reward processing is the ventral striatum (VS), which contains the nucleus accumbens and is involved in the valuation, anticipation, and consumption of rewards (Haber & Knutson, 2010; Knutson, Westdorp, Kaiser, & Hommer, 2000; Richards, Plate, & Ernst, 2013). Although the VS has been a primary target for fMRI studies of reward processing in AB and psychopathy using a region of interest approach, the VS receives input from other structures in the mesocorticolimbic dopamine circuit, including the midbrain, anterior cingulate (ACC), amygdala, insula, midbrain, and prefrontal cortex (PFC), and projects to the PFC via the thalamus (Haber & Knutson, 2010; Richards et al., 2013). Areas of the PFC involved in reward processing include the orbitofrontal cortex (OFC) and dorsolateral PFC, which are involved in reward representation, executive control, and decision-making (Haber & Knutson, 2010; O'Doherty, 2007; Öngür & Price, 2000). A recent meta-analysis found that the anticipation of both rewards and losses recruits the striatum, insula, and thalamus, whereas the OFC was

uniquely recruited during reward consumption (Oldham et al., 2018). Although frontostriatal circuitry is likely important to the etiology of AB, particularly to the reward-related behavioral deficits, no systematic work has reviewed the existing literature on this topic in adults to examine the extent to which neural differences are specific to the VS or are more broadly distributed across frontostriatal circuitry (or even more broadly in the brain).

Is AB Associated With Neural Hyper- or Hyposensitivity to Reward?

Much of the research examining neural mechanisms of reward processing in AB has been conducted on adolescent samples and has been reviewed elsewhere (Blair, 2015; Byrd, Loeber, & Pardini, 2014). These reviews highlight the complexity of associations between AB and neural response to reward, with some studies reporting frontostriatal *hyper*sensitivity but others reporting frontostriatal *hypo*sensitivity among individuals high on AB (Blair, 2015; Byrd et al., 2014). These divergent findings may be due to considerable methodological heterogeneity in the type of reward and/or loss investigated, suggesting that a more fine-grained approach is needed. Complex reinforcement behaviors (e.g., risky decision-making and delay discounting) comprise several processes not limited to risk/reward valuation, regulation, anticipation, and consumption (Ernst, Romeo, & Andersen, 2009), making it difficult to distill the source of dysfunction. Identifying links between personality and basic reward-related processes is a key step before examining more complex forms of reward processing. Another factor limiting conclusions is that the frontostriatal circuit undergoes rapid change during adolescence (Ernst et al., 2009), meaning that adolescent findings may not generalize to adults. A focus on adults is important because brain and behavior (particularly impulsivity and risk-taking) are more stable during adulthood (Steinberg, 2005), and studying AB in adulthood can identify individuals with the most severe and chronic AB trajectories (Hyde et al., 2016).

Subtypes of AB: Do AB and Psychopathy Differ on Neural Response to Reward/Loss?

Although individuals high on psychopathy share some features with individuals high on AB only, neural functioning appears to differ among individuals with AB and psychopathy. For example, dimensional measures of AB versus psychopathy have been linked to divergent patterns of neural activation in response to socioemotional stimuli (Hyde et al., 2016). In addition, research suggests that targeting neurobehavioral deficits that are unique to those with broader forms of externalizing psychopathology versus psychopathy may be an effective treatment strategy (Baskin-Sommers, Curtin, & Newman, 2015). Structural imaging also suggests that affective-interpersonal versus impulsive-antisocial features of psychopathy are linked to divergent patterns of striatal volume, with the impulsive-antisocial features being linked to greater volume in the VS (Glenn, Raine, Yaralian, & Yang, 2010). Functional neuroimaging research found that impulsive-antisocial, but not affective-interpersonal, features of psychopathy were linked to greater VS response to reward cues and dopamine release in the nucleus accumbens (Buckholtz et al., 2010). Thus, in reviewing the reward-related neural correlates of AB and psychopathic personality traits in adults, it is critical to systematically assess the extent to which these neural correlates are specific to

broader AB versus psychopathy (vs. the affective-interpersonal vs. impulsive-antisocial components of psychopathy).

Present Review

The primary goal of this systematic review was to examine the extent to which AB and psychopathy are associated with monetary reward- and loss-related neural activity, whether the findings imply a hyper- versus hyposensitive neural reward system, and the extent to which results are specific to AB with or without psychopathic traits. As prior studies have found differences in response to rewards versus losses and anticipation versus consumption of reward/loss, we examined whether these outcomes and phases affected findings. Finally, because reward is a complex construct that features a host of multidimensional processes, we focused on fMRI studies that isolated responses to specific monetary rewards and losses in simple tasks such as the monetary incentive delay (MID) task (Knutson et al., 2000). By examining studies using similar fMRI tasks, we aimed to provide clearer conclusions. However, as a follow-up analysis, we also considered studies that examined broader, more complex reward processes or recruited samples that had various comorbidities to help draw conclusions about the specificity of any reward or loss processing deficits.

Method

Study Selection

Online searches were performed in the databases Medline (PubMed), EMBASE, PsycINFO, and Web of Science on November 13th, 2016. We used search terms relating to AB and psychopathic personality traits, focusing on a wide range of terms to capture similar constructs across domains, including criminology (e.g., crime and violence) and psychiatry (e.g., APD, psychopathy, and aggression). Thus, the search was sensitive to studies that assessed the clinical diagnosis of adult APD and psychopathy, as well as continuous measures of AB and psychopathic traits. We combined these terms with those assessing functional neuroimaging and reward and loss processing (see Table S1 in the online supplemental materials for search terms used).

Exclusion Criteria

Due to our focus on neural response to simple monetary reward and loss, we excluded studies that investigated other forms of reward and loss processing, as those typically included processes like learning and decision-making, and studies in which participants did not receive concrete rewards or punishments (e.g., neural response to punishing others). Because of our focus on reward processing and AB specifically, we excluded studies that had recruited subjects based on drug or alcohol abuse to reduce the possibility that substance use could be driving associations (and given that substance use disorders are common and are often present in individuals who are not engaged in serious AB; American Psychiatric Association, 2013). We also excluded studies that had recruited participants based on a clinical disorder other than APD or psychopathy, including pathological gambling, attention deficit hyperactivity disorder, pedophilia, and other personality disorders. Finally, because we were interested in neural mechanisms of reward processing in adult AB, we excluded

studies with a mean sample age of [H11021]18 years. Consistent with published recommendations for systematic review protocols, we focused on a narrow set of criteria that would enable us to address a specific research question (Moher et al., 2015, p. 3). This approach meant that our results were focused on links between basic reward and loss processing and AB and psychopathy without a myriad of confounds in sampling and task design.

Results

Retrieved Studies

The flow of studies through the review screening is summarized in Figure S1 in the online supplemental materials. We identified 699 publications, of which 237 were duplicate articles. After removing duplicates, we screened 462 reports, 446 of which were excluded based on their title or abstract. The full texts of the remaining 16 reports were screened in detail; nine were excluded (Table S2 in the online supplemental materials). Seven publications met our inclusion criteria (Table 1). We focus our presentation of the results on the findings from these seven studies (Table 2). However, because of the low number of identified studies, we broadened our study pool to confirm findings in studies that had assessed AB/psychopathy in samples recruited for psychopathologies that are often comorbid with AB (e.g., substance abuse, attention deficit hyperactivity disorder, and cluster B personality disorders), and in studies that assessed other components of reward and loss processing, including reward learning and social reward processing (i.e., rewarding and punishing others). Applying these expanded criteria, we included nine additional studies (Tables S3 & S4 in the online supplemental materials), which we reference in the Results and Discussion in relation to the findings of the seven studies that met our original stringent criteria.

Sample, Methodological, and Analytic Features of Included Studies

Sample.—Of the seven included studies, three investigated relationships using community or undergraduate samples (Bjork, Chen, & Hommer, 2012; Buckholtz et al., 2010; Carré, Hyde, Neumann, Viding, & Hariri, 2013). Four studies examined forensic samples, including two that compared incarcerated offenders with healthy controls (Geurts, von Borries, Volman, Bulten, & Cools, 2016; Völlm et al., 2010), one that compared psychopathic and nonpsychopathic inmates (Pujara, Motzkin, Newman, Kiehl, & Koenigs, 2014), and one that compared psychopathic and nonpsychopathic inmates with a third, healthy control group (Gregory et al., 2015). All studies of community/undergraduate samples, but none of the studies of forensic samples, included women.

Measurement approach.—Two studies examined associations using a case-control approach (e.g., APD/psychopathy vs. healthy controls; Geurts et al., 2016; Völlm et al., 2010). Three studies examined associations dimensionally (Bjork et al., 2012; Buckholtz et al., 2010; Carré et al., 2013), and two studies adopted both dimensional and case-control approaches (Gregory et al., 2015; Pujara et al., 2014).

Type and measure of AB.—Four studies assessed whether different facets of psychopathy (e.g., impulsive-antisocial vs. affective-interpersonal traits) were related to neural reactivity during reward processing (Bjork et al., 2012; Buckholtz et al., 2010; Carré et al., 2013; Geurts et al., 2016). Within studies of forensic samples, two used the Structured Clinical Interview for *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* Axis II personality disorders (SCID-II) and the Psychopathy Checklist–Revised (PCL-R) to identify offenders with APD with and without co-occurring psychopathy (Gregory et al., 2015; Pujara et al., 2014), one study assessed psychopathy via the PCL-R but did not measure APD (Geurts et al., 2016), and one study assessed APD via the SCID-II but did not measure psychopathy (Völlm et al., 2010). Four studies used the PCL-R (Hare, 2003) or a self-report that resulted in comparisons between affective-interpersonal and antisocial-lifestyle facets, and three studies used the Psychopathic Personality Inventory (Lilienfeld, Widows, & Staff, 2005), which resulted in comparisons between fearless-dominance and impulsive-antisocial subtypes of psychopathic traits. Thus, most studies distinguished between Factor 1 and Factor 2 of psychopathy (Hare, 2003).

Type of reward/loss.—All but one of the seven studies (Gregory et al., 2015) used a monetary reward task. Five studies investigated reward processing only (Bjork et al., 2012; Buckholtz et al., 2010; Carré et al., 2013; Geurts et al., 2016; Völlm et al., 2010), and two studies investigated both reward and loss processing (Gregory et al., 2015; Pujara et al., 2014). In terms of the specific phase of reward and/or loss processing, two studies investigated reward anticipation only (Bjork et al., 2012; Geurts et al., 2016), two investigated reward and loss consumption only (Gregory et al., 2015; Pujara et al., 2014), one investigated reward anticipation and consumption (Buckholtz et al., 2010), and two investigated reward response using a blocked design that did not separate anticipation and consumption (Carré et al., 2013; Völlm et al., 2010).

Findings From fMRI Studies of Reward/Loss Processing in AB

First, we present the results of studies that reported findings in the VS either using a region of interest (ROI) and/or whole-brain analysis. Second, we discuss studies that reported findings in other regions using a whole brain approach. Within these groupings, we separate results based on type (i.e., reward vs. loss), and when possible, phase (i.e., anticipation vs. consumption of reward/loss). To examine the generalizability of the results to more complex forms of reward processing and broader definitions of AB, we also discuss findings from the nine studies in our expanded sample (Tables S3 & S4 in the online supplemental materials) at the end of each subsection.

Ventral Striatal Associations Between Reward and AB: ROI and Whole-Brain

Anticipation of reward.—Four studies reported links between AB and psychopathy and VS activity during reward processing. First, Bjork and colleagues (2012) found that psychopathic traits were related to greater VS activity in a sample of healthy controls during a modified MID task. In supplemental analyses, the impulsive-antisociality, but not fearless-dominance, factor of psychopathy was linked to greater VS activity. Second, using a MID task in a community sample, Buckholtz and colleagues (2010) found that the impulsive-antisociality, but not the fearless-dominance, factor of psychopathy was linked to greater VS

activity during reward anticipation. This effect remained significant when controlling for general impulsivity. Third, Geurts and colleagues (2016) found that during reward anticipation, psychopathic inmates and noninmates high on impulsive-antisociality had increased VS reactivity compared with noninmates low on impulsive-antisociality. Finally, Carré and colleagues (2013) investigated a sample of undergraduates and used the Self-Report of Psychopathy to break apart the antisocial-lifestyle factor into two correlated facets. They found that the lifestyle facet of psychopathy was negatively correlated with VS activity to reward (no separation of reward phases), whereas the antisocial facet of psychopathy was positively correlated with VS activity. When controlling for self-reported impulsivity, the negative VS association with the lifestyle facet of psychopathy remained, but the positive association with the antisocial facet became a trend. Together, these four studies suggest that greater VS reactivity during reward anticipation may be specific to those high on general forms of AB but not those with the affective-interpersonal psychopathic traits, with two studies contradicting each other on whether trait impulsivity may (Carré et al., 2013) or may not (Buckholtz et al., 2010) explain these associations.

Consumption of reward.—Of the three studies that isolated reward consumption, none found significant associations between psychopathy and/or APD and VS reactivity (Buckholtz et al., 2010; Gregory et al., 2015; Pujara et al., 2014).

Expanded sample.—One out of the six studies that investigated reward processing found links between AB and VS response to reward. Cohn and colleagues (2015) found that individuals with persistent disruptive behavior disorders, versus controls and those who had desisted, had reduced VS response during the consumption, but not anticipation of rewards.

Ventral Striatal Associations Between Loss and AB: ROI and Whole-Brain

Loss processing.—Only two studies investigated neural response to loss processing. Gregory and colleagues (2015) found no links between APD or psychopathy and loss-related VS activity. In contrast, Pujara and colleagues (2014) found that compared with controls, psychopaths had reduced VS reactivity to loss receipt compared with neutral trials (Pujara et al., 2014). Thus, though there was some suggestion that those high on psychopathy have reduced VS reactivity during the receipt of loss, the literature is not well-developed enough to inform a conclusion.

Expanded sample.—Six of the nine studies identified with our expanded criteria investigated loss processing (broadly defined). However, none of these identified studies reported associations between AB or psychopathic traits and VS response to loss.

Other Whole-Brain Associations Between Reward Reactivity and AB

Reward anticipation and consumption: Prefrontal cortex.—Three studies examined correlates of reward in regions outside of the VS and reported that APD or psychopathy were related to PFC reactivity to reward (Bjork et al., 2012; Geurts et al., 2016; Völlm et al., 2010). First, compared with controls, inmates with APD had increased right OFC activity to reward (Völlm et al., 2010). However, the main effects of the blocked task indicated that the task did not activate hypothesized reward-related brain regions, including

the ventral and medial PFC, ACC, amygdala, striatum, and midbrain. Second, during the anticipation of passively received rewards, Bjork and colleagues (2012) found that psychopathic traits among a community sample were positively associated with anterior medial PFC reactivity. Finally, in the only study to investigate functional connectivity (i.e., correlated brain activity between brain regions), Geurts and colleagues (2016) reported that psychopathic inmates had increased functional connectivity between the VS and the dorsomedial PFC compared with noninmates. These findings suggest that APD and psychopathic traits may be linked to greater medial PFC and OFC reactivity to reward.

Reward anticipation and consumption: Other brain regions.—Völlm and colleagues (2010) reported that compared with controls, inmates with APD had increased pregenual ACC reactivity during reward anticipation. Bjork and colleagues (2012) reported that impulsive-antisocial traits were linked to increased dorsal ACC reactivity during reward anticipation. Although the activation reported in these two studies does not show anatomical overlap, the findings suggest that individuals with APD or impulsive-antisocial psychopathic traits have greater reactivity in the ACC broadly during reward anticipation. Finally, Gregory and colleagues (2015) reported that those with APD and psychopathy had reduced reactivity to reward in regions of the temporal lobe (right superior temporal gyrus and anterior middle temporal gyrus) compared with individuals with APD without psychopathy and healthy controls.

Expanded sample.—With regard to impulsive, emotionally reactive forms of AB, studies reported decreased medial prefrontal cortex (mPFC) or OFC reactivity to rewards in individuals with intermittent explosive disorder (Gan et al., 2016), individuals with high AB and alcohol use (Oberlin et al., 2012), and patients with APD and/or borderline personality disorder (Völlm et al., 2007). In a study of risky decision-making in high and low emotionally reactive APD, inmates with emotionally reactive APD had increased cerebellum and occipital gyrus activity when anticipating risky, uncertain rewards, whereas inmates with APD and Factor 1 psychopathic traits had decreased ACC activity (Prehn et al., 2013). Two studies reported that Callous-Unemotional traits were associated with decreased mPFC (Veroude et al., 2016) and amygdala (Cohn et al., 2015) reactivity to reward.

Other Whole-Brain Associations Between Loss Reactivity and AB

Loss anticipation and consumption: Prefrontal cortex.—Two of the identified studies investigated neural response to loss. However, neither of these studies reported associations between AB and prefrontal activity.

Loss anticipation and consumption: Other brain regions.—In the only study to report whole-brain findings for loss processing, Gregory and colleagues (2015) found that inmates with APD and psychopathy had greater anterior insula, posterior cingulate, and precuneus activity to punished reversal errors versus inmates with APD without psychopathy. Psychopathy scores were also positively correlated with posterior cingulate activity to punished reversal errors.

Expanded sample.—Two studies in the expanded sample reported associations between AB and neural response to loss, including increased amygdala reactivity in those with persistent disruptive behavior disorders (Cohn et al., 2015) and greater mPFC and ACC and less lateral PFC reactivity in patients with APD and/or borderline personality disorder (Völlm et al., 2007). In contrast, two studies reported associations between psychopathic traits and loss, including decreased ACC response in those with APD and Factor 1 psychopathic traits (Prehn et al., 2013) and reduced OFC/ACC reactivity when accepting of unfair monetary offers (Vieira et al., 2014).

Discussion

A long history of theories has emphasized the importance of reward processing in the etiology of AB and psychopathy (Blair, 2015; Byrd et al., 2014; Fowles, 1980; Newman & Kosson, 1986; Quay, 1993). This systematic review represents a key step that advances our understanding of the relationships between AB, psychopathy, and reward- and loss-related neural functioning. We found that AB and the impulsive-antisocial components of psychopathy were linked to greater VS and PFC reactivity during reward anticipation. However, given the small number of studies, a clear implication from this review is the need for more studies to explore the specific patterns of reward-related functioning that might be differentially associated with AB versus psychopathy. Indeed, the small number of studies and the small sample sizes within many of these studies is not only an issue for interpreting individual findings, but for our review as a whole. Keeping these limitations in mind, we outline the main findings from the review and focus on specific gaps in the literature that can be addressed in future work.

Is AB Associated With Neural Differences During Reward Processing in the VS?

We reviewed seven studies that investigated neural mechanisms of reward processing in AB and psychopathic traits. Together, these studies provide evidence that AB and psychopathy are related to differences in neural reactivity during reward processing. The most consistent finding (four of seven studies) was a positive association between AB, impulsive-antisocial psychopathic traits, and VS reactivity (or VS-PFC functional connectivity) during reward processing (Bjork et al., 2012; Buckholtz et al., 2010; Carré et al., 2013; Geurts et al., 2016). Moreover, these findings were mostly for the VS response to the anticipation, rather than receipt, of reward and were most consistently related to APD in forensic samples or the antisocial facet of psychopathy in healthy/community samples. However, only three studies investigated reward consumption, and only one investigated both reward anticipation and consumption, highlighting the importance of replication before strong conclusions can be drawn.

Nevertheless, animal and human research suggests that the anticipation (i.e., “wanting”) of rewards is distinct from the consumption (i.e., “liking”) of rewards. An imbalance between wanting and liking of rewards may drive other externalizing behaviors (i.e., substance use) by driving reward motivation despite lack of enjoyment when consuming the reward (Robinson & Berridge, 2001). Applying this perspective to the current findings, individuals high on impulsive-antisocial traits may seek risky rewards due to greater anticipatory

"wanting" of rewards, rather than experiencing excessive pleasure (i.e., "liking") upon receiving rewards. Behavioral research in youth and adults suggests that individuals engaging in AB show heightened risk-taking and continue to pursue rewards despite the possibility of punishment (Budhani et al., 2006; Byrd et al., 2014), supporting the notion of an imbalance between motivational and consummatory responses to reward in chronic AB, which may impede appropriate behavior change.

At the same time, these findings were not replicated in three studies in our expanded sample that had investigated reward anticipation (see Table S4 in the online supplemental materials). Thus, the link between impulsive-antisocial psychopathic traits and reward anticipation may be specific to tasks that tap simple, isolated types of reward, including the MID task, as opposed to tasks that probe risky decision-making or social reward and punishment. Alternatively, given that three of the four core studies that reported this association were studies of healthy controls, it may be that relatively healthy individuals with impulsive-antisocial traits differ from individuals with more severe forms of AB (including APD) in their neural response to reward. The comparison with the broader set of studies also highlighted differences in how AB was measured or conceptualized, as these studies included intermittent explosive disorder (Gan et al., 2016) and APD co-occurring with borderline personality disorder (Völlm et al., 2007), as opposed to a "pure" AB phenotype within incarcerated samples (Gregory et al., 2015). In sum, impulsive-antisocial psychopathic traits were somewhat consistently linked to greater VS response during reward anticipation, though this finding was largely confined to healthy or community samples and to simple monetary reward tasks rather than more complex forms of reward processing.

Is AB Associated With Neural Differences During Reward Processing in the PFC?

Beyond VS reactivity, several studies found that APD or self-reported impulsive-antisocial traits were related to greater OFC and medial PFC reactivity, and VS-dorsomedial PFC connectivity during reward processing (Bjork et al., 2012; Geurts et al., 2016; Völlm et al., 2010). As many studies did not conduct whole-brain or PFC ROI analyses, it is difficult to know how consistent these findings are across samples (of the three studies that examined the whole brain, two reported findings in the PFC). Increased mPFC reactivity has been linked to heightened emotional arousal and expected value signaling to reward cues (Marsh, Blair, Vythilingam, Busis, & Blair, 2007; O'Doherty, 2007). Together, these findings could imply broad reward-related frontostriatal hyperactivity in those high on AB. At the same time, this conclusion was not supported by the studies from the expanded sample. In fact, four (of seven) of these studies found that reduced PFC reactivity during reward processing was related to AB. As before, these differences may have arisen from considerable sample and task differences, underscoring the need for studies to examine neural reactivity during paradigms that separate reward/loss anticipation from receipt in samples that allow for the isolation of specific components of AB.

In addition to greater PFC reactivity, impulsive-antisocial traits and APD were associated, in at least one study, with increased neural reactivity to rewards in frontal regions broadly associated with emotional salience and reward-based decision-making, including the pregenual (Völlm et al., 2010) and dorsal (Bjork et al., 2012) ACC. Furthermore, during

risky decision-making, individuals with APD and psychopathy had *reduced* ACC activity (Prehn et al., 2013), suggesting that ACC response may differentiate those high on APD versus those high on APD and psychopathy. The dorsal ACC is linked to multiple functions related to error monitoring, attention, action selection, and reward anticipation (Botvinick, Cohen, & Carter, 2004; Knutson et al., 2000), whereas the pregenual ACC is linked to emotional learning and reward preference (Öngür & Price, 2000). Together these results suggest that APD and impulsive-antisocial traits may be linked to greater reward sensitivity within brain regions that support error monitoring, attention, and reward preference. Relatedly, the results of the current review suggest that Factor 1 psychopathy is largely unrelated to frontostriatal functioning during reward processing. Thus, dysfunction in reward-related neural circuits may be a mechanism that contributes to AB more broadly, rather than one that is specific to psychopathy.

Is AB Associated With Neural Differences in Loss Processing?

Both studies that investigated neural responses during loss processing reported significant associations (Gregory et al., 2015; Pujara et al., 2014). These studies found that psychopathy was linked to greater reactivity in several regions, including the posterior cingulate, precuneus, and insula during error-related loss receipt (Gregory et al., 2015), and decreased VS reactivity during loss anticipation (Pujara et al., 2014). It is challenging to interpret these findings given that each was unique to only one study. Nevertheless, it is noteworthy that increased reactivity in the posterior cingulate and precuneus, key nodes of the default mode network, have also been linked to higher AB during tasks probing affective processing (Raine & Yang, 2006) and behavior modification in the context of reward (Pearson, Heilbronner, Barack, Hayden, & Platt, 2011). Within the broader pool of studies, APD and borderline personality disorder were associated with increased medial PFC and ACC reactivity and reduced lateral PFC reactivity to punishment (Völlm et al., 2007). However, psychopathy was related to reduced reactivity in limbic and frontocortical circuitry when receiving unfair monetary offers or losing money during social tasks such as ultimatum or dictator games (Osumi et al., 2012; Prehn et al., 2013; Vieira et al., 2014). Together, findings from across the full, expanded pool of 16 studies suggest that individuals with more reactive forms of AB (e.g., Factor 2 psychopathy, comorbid borderline personality disorder, and APD) show greater neural responsivity to punishing outcomes, whereas those with interpersonal-affective deficits (e.g., Factor 1 psychopathy) show reduced reactivity to punishment, particularly punishment with a social component. These findings generally support behavioral research that suggests that individuals high on impulsive, antisocial traits are emotionally hyperreactive to salient information (greater BAS), whereas individuals high on affective psychopathic traits are less reactive to negative stimuli (lower BIS; Baskin-Sommers & Newman, 2013; Hoppenbrouwers et al., 2015).

Future Directions

Although our review helps to shed some light on the neural correlates of AB during reward and loss processing, the relatively small literature and heterogeneity between studies highlight several important limitations of the current literature and future directions.

Task design.—Overall, we found that AB and psychopathy were linked to dysfunction in reward and loss processing. However, the small number of included studies makes it challenging to draw strong conclusions, especially given significant methodological heterogeneity across studies. For example, no studies in the core sample, and only one study in the expanded sample, investigated links between AB and/or psychopathy and neural reactivity while separating both phase and valence within a single design. Behavioral research suggests that investigating reward and loss in a single design can help to elucidate complex relationships. For example, among youth, psychopathic traits were related to deficits during a response reversal task, which were driven by a failure to shift away from reward-dominant responses following punishment rather than by a reduced tendency to learn contingencies or to continue to respond to rewarded stimuli (Budhani & Blair, 2005). In addition, few studies investigated associations between AB or psychopathy and loss processing, emphasizing this area as a critical target for future research. As such, future research is needed to investigate how specific phases of reward and loss processing are linked to dysfunction in AB versus psychopathy, which could help identify specific biomarkers of AB and externalizing behavior versus psychopathy and inform individualized treatments based on these biomarkers (for a discussion of this type of approach see, Brazil, van Dongen, Maes, Mars, & Baskin-Sommers, 2016).

Expanding our inclusion criteria to include nine additional studies that had examined broader forms of reward and loss processing did not help to inform clearer conclusions. Indeed, these broader studies further highlighted the difficulty inherent in classifying or developing task conditions as “rewarding”. For example, studies within our expanded pool used tasks that required participants to give electric shocks or rewards to in-group and out-group members (Molenberghs et al., 2014) and that explored neural responsivity to unfair monetary offers in social ultimatum or dictator games (Osumi et al., 2012; Vieira et al., 2014). It is unlikely that these tasks are comparable with those focused on simple monetary loss within nonsocial contexts (i.e., Is punishing others with an electric shock rewarding or punishing? Are unfair offers truly “punishing” in the same way as directly losing money?). Nevertheless, results from the expanded pool of studies highlight the complexity of reward and loss processing and suggest that AB may be associated with dysfunction across multiple kinds of reward, learning, and social processes.

Data analysis approach.—The review also highlights the need to move beyond VS region of interest investigations and to instead investigate associations across the whole brain. Findings from included studies that adopted whole-brain analytic approaches suggested that AB may be related to dysfunction in several limbic and cortical regions during reward and loss processing. However, given the small number of studies, these findings need to be replicated.

Sample.—Studies in the review differed significantly in both type of AB (measures of behaviors such as AB vs. measures of traits such as psychopathic vs. components of the psychopathy construct such as impulsive-antisociality) and severity of AB (community vs. forensic). This sample heterogeneity highlights several areas to be addressed in future research. First, three of the four studies that reported positive associations between

impulsive-antisocial psychopathic traits and reward sensitivity were in community samples, and one was in both incarcerated and community samples, suggesting that the findings were consistent across a range of severity. However, it is also possible that in healthy samples, impulsive-antisocial psychopathic traits may tap trait impulsivity rather than AB specifically. Similarly, our expanded study pool included individuals with a range of externalizing disorders, which share some phenotypic overlap with severe AB (e.g., impulsivity and emotion dysregulation), but may also exhibit some unique neural correlates (Rubia et al., 2009). Two studies did control for self-reported impulsivity, with one reporting that links between reward and psychopathic traits remained significant, whereas the other reported that some associations weakened to a trend. Thus, there is some evidence supporting the notion that AB may have unique patterns of neural response to reward beyond trait-level impulsivity. Finally, a limitation of our review is our narrow definition of AB as a dimension of severe AB, APD, and psychopathic traits. There may be important subtypes within the larger construct of AB, including proactive and reactive aggression, violent and nonviolent AB, aggression versus rule breaking, and AB in the context of substance use, and these subgroups have largely gone uninvestigated in neuroimaging studies. Future research should probe whether relationships between AB and reward processing are unique to AB, or are shared across a range of externalizing psychopathologies, or whether they may be specific to certain AB phenotypes.

Second, due to challenges associated with recruiting and conducting neuroimaging research among individuals with severe AB, who are often incarcerated or in forensic settings, sample sizes of included studies tended to be small (e.g., range [H11005] 12–25). Thus, many studies included in the review may have been underpowered to detect effects (i.e., Type II errors) and/or may have reported effects that will not replicate (i.e., Type I error; Button et al., 2013). At the same time, three studies examined healthy control samples with a larger range of sample sizes (range [H11005] 24–171). Such samples are easier to recruit, but findings may not be generalizable to those engaging in more severe AB. In our review, several of the relationships between psychopathic traits and neural activation were found in studies of these traits in healthy controls. This is a major limitation, as it is unclear whether these relationships are linear across trait severity or whether these relationships differ in healthy and clinical groups. This limitation highlights the importance of future studies testing associations between AB and neural response to reward using dimensional measures and samples that include a wide range of AB from very low to those with extensive arrest records and diagnoses of APD.

Replicability.—Consistent with this point, as is well-documented in behavioral and neuroimaging research, psychology and clinical neuroscience are facing a replicability crisis (Abram & DeYoung, 2017; Button et al., 2013; Eklund, Nichols, & Knutsson, 2016; Tackett et al., 2017). The typically small sample sizes, low power, large number of exploratory analyses over many voxels, and significant sample heterogeneity that plagues the majority of the neuroimaging research in psychopathy/AB, and clinical science more broadly, limits the ability to draw conclusions (Abram & DeYoung, 2017). Our review is dependent on the studies conducted. As these studies suffer from small samples, their findings may capitalize on chance findings, and aggregating many small findings may only magnify this problem.

That is, without replicable studies in a review, the review itself may suffer from the same weakness of the studies within (particularly given our small study sample size of 7). Thus, our findings should be seen as preliminary and help guide future research with larger samples.

Finally, even with *large* samples, our understanding of true neural mechanisms of psychopathology may be impacted by using *unrepresentative* samples (e.g., racial, socioeconomic, and cultural bias in sampling; Falk et al., 2013). For example, one of our studies examined undergraduates, and several studies used exclusively male samples, which are unlikely to generalize to the general population, female samples, and samples at higher risk of AB. These limitations in the field of clinical neuroscience highlight the importance of encouraging large-scale collaboration and replication efforts, representative sampling methods, making data publicly available in order to address the issue of replicability in our field (Tackett et al., 2017).

Conclusion

This systematic review explored the relationship between AB and neural reactivity during reward and loss processing in adults. Findings suggest that AB and psychopathic traits are linked to neural dysfunction during both reward and loss processing. In the studies that separated the factors of psychopathy, it appeared that Factor 2 (i.e., impulsive-antisocial) psychopathy and APD were both related to *increased* reactivity in the VS during reward *anticipation*. This greater VS response appeared to be specific to the anticipation, rather than consumption, of rewards. Finally, AB and psychopathy were related to reactivity in regions beyond the VS during reward and loss processing, including the cingulate and PFC, highlighting the need for future studies to adopt both whole-brain and ROI approaches to study neural dysfunction in AB. The results of this review are consistent with the theory in the field (Blair, 2015), emphasizing that reward-related neural differences are related broadly to AB, rather than specifically to psychopathy.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

References

Abram SV, & DeYoung CG (2017). Using personality neuroscience to study personality disorder. Personality Disorders: Theory, Research, and Treatment, 8, 2–13. 10.1037/per0000195

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (DSM–5), Fifth Edition. Washington, DC: Author.

Baskin-Sommers AR, Curtin JJ, & Newman JP (2015). Altering the cognitive-affective dysfunctions of psychopathic and externalizing of-fender subtypes with cognitive remediation. Clinical Psychological Science, 3, 45–57. 10.1177/2167702614560744 [PubMed: 25977843]

Baskin-Sommers AR, & Newman JP (2013). Differentiating the cognition-emotion interactions that characterize psychopathy versus externalizing In Robinson MD, Watkins E, & Harmon-Jones E (Eds.), Handbook of cognition and emotion (pp. 501–520). New York, NY: Guilford Press.

Berridge KC, & Robinson TE (2003). Parsing reward. Trends in Neurosciences, 26, 507–513. 10.1016/S0166-2236(03)00233-9 [PubMed: 12948663]

Bjork JM, Chen G, & Hommer DW (2012). Psychopathic tendencies and mesolimbic recruitment by cues for instrumental and passively obtained rewards. Biological Psychology, 89, 408–415. 10.1016/j.biopsycho.2011.12.003 [PubMed: 22178441]

Blair RJR (2015). Psychopathic traits from an RDoC perspective. Current Opinion in eurobiology, 30, 79–84. 10.1016/j.conb.2014.09.011

Botvinick MM, Cohen JD, & Carter CS (2004). Conflict monitoring and anterior cingulate cortex: An update. Trends in Cognitive Sciences, 8, 539–546. 10.1016/j.tics.2004.10.003 [PubMed: 15556023]

Brazil IA, van Dongen JD, Maes JH, Mars RB, & Baskin-Sommers AR (2016). Classification and treatment of antisocial individuals: From behavior to biocognition. Neuroscience and Biobehavioral Reviews, 91, 259–277. 10.1016/j.neubiorev.2016.10.010 [PubMed: 27760372]

Buckholtz JW, Treadway MT, Cowan RL, Woodward ND, Benning SD, Li R, … Zald DH (2010). Mesolimbic dopamine reward system hypersensitivity in individuals with psychopathic traits. Nature Neuroscience, 13, 419–421. 10.1038/nn.2510 [PubMed: 20228805]

Budhani S, & Blair RJR (2005). Response reversal and children with psychopathic tendencies: Success is a function of salience of contingency change. Journal of Child Psychology and Psychiatry, 46, 972–981. 10.1111/j.1469-7610.2004.00398.x [PubMed: 16109000]

Budhani S, Richell RA, & Blair RJR (2006). Impaired reversal but intact acquisition: Probabilistic response reversal deficits in adult individuals with psychopathy. Journal of Abnormal Psychology, 115, 552–558. 10.1037/0021-843X.115.3.552 [PubMed: 16866595]

Button KS, Ioannidis JP, Mokrysz C, Nosek BA, Flint J, Robinson ES, & Munafò MR (2013). Power failure: Why small sample size undermines the reliability of neuroscience. Nature Reviews Neuroscience, 14, 365–376. 10.1038/nrn3475 [PubMed: 23571845]

Byrd AL, Loeber R, & Pardini DA (2014). Antisocial behavior, psychopathic features and abnormalities in reward and punishment processing in youth. Clinical Child and Family Psychology Review, 17, 125–156. 10.1007/s10567-013-0159-6 [PubMed: 24357109]

Carré JM, Hyde LW, Neumann CS, Viding E, & Hariri AR (2013). The neural signatures of distinct psychopathic traits. Social Neuroscience, 8, 122–135. 10.1080/17470919.2012.703623 [PubMed: 22775289]

Cloninger CR (1987). A systematic method for clinical description and classification of personality variants. A proposal. Archives of General Psychiatry, 44, 573–588. 10.1001/archpsyc.1987.01800180093014 [PubMed: 3579504]

Cohn MD, Veltman DJ, Pape LE, van Lith K, Vermeiren RR, van den Brink W, … Popma A (2015). Incentive processing in persistent disruptive behavior and psychopathic traits: A functional magnetic resonance imaging study in adolescents. Biological Psychiatry, 78, 615–624. 10.1016/j.biopsych.2014.08.017 [PubMed: 25497690]

Eklund A, Nichols TE, & Knutsson H (2016). Cluster failure: Why fMRI inferences for spatial extent have inflated false-positive rates. Proceedings of the National Academy of Sciences of the United States of America, 113, 7900–7905. 10.1073/pnas.1602413113 [PubMed: 27357684]

Ernst M, Romeo RD, & Andersen SL (2009). Neurobiology of the development of motivated behaviors in adolescence: A window into a neural systems model. Pharmacology, Biochemistry, and Behavior, 93, 199–211. 10.1016/j.pbb.2008.12.013

Falk EB, Hyde LW, Mitchell C, Faul J, Gonzalez R, Heitzeg MM, … Schulenberg J (2013). What is a representative brain? Neuroscience meets population science. Proceedings of the National Academy of Sciences of the United States of America, 110, 17615–17622. 10.1073/pnas.1310134110 [PubMed: 24151336]

Fowles DC (1980). The three arousal model: Implications of gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. Psychophysiology, 17, 87–104. 10.1111/j.1469-8986.1980.tb00117.x [PubMed: 6103567]

Gan G, Preston-Campbell RN, Moeller SJ, Steinberg JL, Lane SD, Maloney T, … Alia-Klein N (2016). Reward vs. retaliation— the role of the mesocorticolimbic salience network in human reactive aggression. Frontiers in Behavioral Neuroscience, 10, 179 10.3389/fnbeh.2016.00179 [PubMed: 27729852]

Geurts DE, von Borries K, Volman I, Bulten BHE, Cools R, & Verkes RJ (2016). Neural connectivity during reward expectation dissociates psychopathic criminals from non-criminal individuals with

high impulsive/antisocial psychopathic traits. Social Cognitive and Affective Neuroscience, 11, 1326–1334. 10.1093/scan/nsw040 [PubMed: 27217111]

Glenn AL, Raine A, Yaralian PS, & Yang Y (2010). Increased volume of the striatum in psychopathic individuals. Biological Psychiatry, 67, 52–58. 10.1016/j.biopsych.2009.06.018 [PubMed: 19683706]

Gray JA (1970). The psychophysiological basis of introversion-extraversion. Behaviour Research and Therapy, 8, 249–266. 10.1016/0005-7967(70)90069-0 [PubMed: 5470377]

Gregory S, Blair RJ, Ffytche D, Simmons A, Kumari V, Hodgins S, & Blackwood N (2015). Punishment and psychopathy: A case-control functional MRI investigation of reinforcement learning in violent antisocial personality disordered men. The Lancet Psychiatry, 2, 153–160. 10.1016/S2215-0366(14)00071-6 [PubMed: 26359751]

Haber SN, & Knutson B (2010). The reward circuit: Linking primate anatomy and human imaging. Neuropsychopharmacology, 35, 4–26. 10.1038/npp.2009.129 [PubMed: 19812543]

Hare RD (2003). The Hare Psychopathy Checklist-Revised (2nd ed.). Toronto, Ontario, Canada: Multi-Health Systems.

Hoppenbrouwers SS, Neumann CS, Lewis J, & Johansson P (2015). A latent variable analysis of the Psychopathy Checklist-Revised and behavioral inhibition system/behavioral activation system factors in North American and Swedish offenders. Personality Disorders: Theory, Research, and Treatment, 6, 251–260. 10.1037/per0000115

Hyde LW, Shaw DS, Murray L, Gard A, Hariri AR, & Forbes EE (2016). Dissecting the role of amygdala reactivity in antisocial behavior in a sample of young, low-income, urban men. Clinical Psychological Science, 4, 527–544. 10.1177/2167702615614511 [PubMed: 27429865]

Knutson B, Westdorp A, Kaiser E, & Hommer D (2000). FMRI visualization of brain activity during a monetary incentive delay task. NeuroImage, 12, 20–27. 10.1006/nimg.2000.0593 [PubMed: 10875899]

Lilienfeld SO, Widows MR, & Staff P (2005). Psychopathic Personality InventoryTM-Revised [SOI]. Social Influence, 61, 97.

Lykken DT (1995). The antisocial personalities. Hillsdale, NJ: Erlbaum.

Marsh AA, Blair KS, Vythilingam M, Busis S, & Blair RJ (2007). Response options and expectations of reward in decision-making: The differential roles of dorsal and rostral anterior cingulate cortex. NeuroImage, 35, 979–988. 10.1016/j.neuroimage.2006.11.044 [PubMed: 17292631]

McCollister KE, French MT, & Fang H (2010). The cost of crime to society: New crime-specific estimates for policy and program evaluation. Drug and Alcohol Dependence, 108, 98–109. 10.1016/j.drugalcdep.2009.12.002 [PubMed: 20071107]

Moher D, Shamseer L, Clarke M, Ghersi D, Liberati A, Petticrew M, … the PRISMA-P Group. (2015). Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. Systematic Reviews, 4, 1 10.1186/2046-4053-4-1 [PubMed: 25554246]

Molenberghs P, Bosworth R, Nott Z, Louis WR, Smith JR, Amiot CE, … Decety J (2014). The influence of group membership and individual differences in psychopathy and perspective taking on neural responses when punishing and rewarding others. Human Brain Mapping, 35, 4989–4999. 10.1002/hbm.22527 [PubMed: 24753026]

Newman JP, & Kosson DS (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. Journal of Abnormal Psychology, 95, 252–256. 10.1037/0021-843X.95.3.252 [PubMed: 3745647]

Oberlin BG, Dzemidzic M, Bragulat V, Lehigh CA, Talavage T, O'Connor SJ, & Kareken DA (2012). Limbic responses to reward cues correlate with antisocial trait density in heavy drinkers. NeuroImage, 60, 644–652. 10.1016/j.neuroimage.2011.12.043 [PubMed: 22227139]

O'Doherty JP (2007). Lights, camembert, action! The role of human orbitofrontal cortex in encoding stimuli, rewards, and choices. Annals of the New York Academy of Sciences, 1121, 254–272. 10.1196/annals.1401.036 [PubMed: 17872386]

Oldham S, Murawski C, Fornito A, Youssef G, Yücel M, & Lorenzetti V (2018). The anticipation and outcome phases of reward and loss processing: A neuroimaging meta-analysis of the monetary incentive delay task. Human Brain Mapping. Advance online publication. 10.1002/hbm.24184

Öngür D, & Price JL (2000). The organization of networks within the orbital and medial prefrontal cortex of rats, monkeys and humans. Cerebral Cortex, 10, 206–219. 10.1093/cercor/10.3.206 [PubMed: 10731217]

Osumi T, Nakao T, Kasuya Y, Shinoda J, Yamada J, & Ohira H (2012). Amygdala dysfunction attenuates frustration-induced aggression in psychopathic individuals in a non-criminal population. Journal of Affective Disorders, 142, 331–338. 10.1016/j.jad.2012.05.012 [PubMed: 22840629]

Pearson JM, Heilbronner SR, Barack DL, Hayden BY, & Platt ML (2011). Posterior cingulate cortex: Adapting behavior to a changing world. Trends in Cognitive Sciences, 15, 143–151. 10.1016/j.tics.2011.02.002 [PubMed: 21420893]

Prehn K, Schlagenhauf F, Schulze L, Berger C, Vohs K, Fleischer M, … Herpertz SC (2013). Neural correlates of risk taking in violent criminal offenders characterized by emotional hypo- and hyper-reactivity. Social Neuroscience, 8, 136–147. 10.1080/17470919.2012.686923 [PubMed: 22747189]

Pujara M, Motzkin JC, Newman JP, Kiehl KA, & Koenigs M (2014). Neural correlates of reward and loss sensitivity in psychopathy. Social Cognitive and Affective Neuroscience, 9, 794–801. 10.1093/scan/nst054 [PubMed: 23552079]

Quay HC (1993). The psychobiology of undersocialized aggressive conduct disorder: A theoretical perspective. Development and Psychopathology, 5, 165–180. 10.1017/S0954579400004326

Raine A, & Yang Y (2006). Neural foundations to moral reasoning and antisocial behavior. Social Cognitive and Affective Neuroscience, 1, 203–213. 10.1093/scan/nsl033 [PubMed: 18985107]

Richards JM, Plate RC, & Ernst M (2013). A systematic review of fMRI reward paradigms used in studies of adolescents vs. adults: The impact of task design and implications for understanding neurodevelopment. Neuroscience and Biobehavioral Reviews, 37, 976–991. 10.1016/j.neubiorev.2013.03.004 [PubMed: 23518270]

Robinson TE, & Berridge KC (2001). Incentive-sensitization and addiction. Addiction, 96, 103–114. 10.1046/j.1360-0443.2001.9611038.x [PubMed: 11177523]

Rubia K, Smith AB, Halari R, Matsukura F, Mohammad M, Taylor E, & Brammer MJ (2009). Disorder-specific dissociation of orbitofrontal dysfunction in boys with pure conduct disorder during reward and ventrolateral prefrontal dysfunction in boys with pure ADHD during sustained attention. The American Journal of Psychiatry, 166, 83–94. 10.1176/appi.ajp.2008.08020212 [PubMed: 18829871]

Steinberg L (2005). Cognitive and affective development in adolescence. Trends in Cognitive Sciences, 9, 69–74. 10.1016/j.tics.2004.12.005 [PubMed: 15668099]

Tackett JL, Lilienfeld SO, Patrick CJ, Johnson SL, Krueger RF, Miller JD, … Shrout PE (2017). It's time to broaden the replicability conversation: Thoughts for and from clinical psychological science. Perspectives on Psychological Science, 12, 742–756. 10.1177/1745691617690042 [PubMed: 28972844]

Veroude K, von Rhein D, Chauvin RJ, van Dongen EV, Mennes MJ, Franke B, … Buitelaar JK (2016). The link between callous-unemotional traits and neural mechanisms of reward processing: An fMRI study. Psychiatry Research: Neuroimaging, 255, 75–80. 10.1016/j.pscychresns.2016.08.005 [PubMed: 27564545]

Vieira JB, Almeida PR, Ferreira-Santos F, Barbosa F, Marques-Teixeira J, & Marsh AA (2014). Distinct neural activation patterns underlie economic decisions in high and low psychopathy scorers. Social Cognitive and Affective Neuroscience, 9, 1099–1107. 10.1093/scan/nst093 [PubMed: 23748499]

Völlm B, Richardson P, McKie S, Elliott R, Dolan M, & Deakin B (2007). Neuronal correlates of reward and loss in Cluster B personality disorders: A functional magnetic resonance imaging study. Psychiatry Research: Neuroimaging, 156, 151–167. 10.1016/j.pscychresns.2007.04.008

Völlm B, Richardson P, McKie S, Reniers R, Elliott R, Anderson IM, … Deakin B (2010). Neuronal correlates and serotonergic modulation of behavioural inhibition and reward in healthy and antisocial individuals. Journal of Psychiatric Research, 44, 123–131. 10.1016/j.jpsychires.2009.07.005 [PubMed: 19683258]

Table 1

Summary of Findings of Reward and Loss Processing in Antisocial Behavior/Psychopathy in Community and Clinical Samples

Study	Sample	Measure	Task/Reward	Type/Phase	Reward contrast	Results	Loss contrast	Results	Summary
					Community/Healthy samples				
Bjork, Chen, & Hommer, 2012	*n* = 31 (18M)	Self-Report PPI	Modified MID (response-dependent and response-independent)	Reward	Reward > Neutral	All Trials:	N/A	N/A	PPI scores positively linked to VS, dorsal ACC, and mesial PFC activity during reward anticipation. Activation for PPI-IA but not PPI-FD mirrored findings for PPI-total.
			Monetary	Anticipation		PPI total: ↑ VS and dorsal ACC Response-dependent: PPI total: ↑ right VS (r = .38, p <.05) and left VS (r = .52, p <.01) PPI-IA: ↑ right VS and dorsal ACC PPI-FD: ns Response-independent: PPI total: ↑ left VS (r = .31, p <.10; trend); ↑ mesial PFC (r = .65, p < .001)			
Buckholtz et al., 2010	*n* = 24 (8M)	Self-Report PPI	MID	Reward	Reward Anticipation > Neutral	PPI-IA: ↑ right VS (r = .63, p = .001)	N/A	N/A	PPI-IA linked to increased VS activity during reward anticipation. Remained significant when controlling for impulsivity.
			Monetary	Anticipation and receipt	Reward Receipt > Neutral	PPI-FD: ns PPI-IA: ns PPI-FD: ns			
Carré, Hyde, Neumann, Viding, &	*n* = 171 (68 M)	Self-Report SRP-SF	Card guessing game	Reward	Reward > Loss	SRP-Lifestyle: ↓ left VS (ß = −.24, p < .05)	N/A	N/A	Lifestyle facet of psychopathy related to decreased VS activity to reward. Antisocial facet of psychopathy

Study	Sample	Measure	Task/Reward	Type/Phase	Reward contrast	Results	Loss contrast	Results	Summary
Hariri, 2013									related to increased VS activity to reward, but not when controlling for impulsivity
			Monetary	Blocked design		SRP-Antisocial: ↑ left VS (ß =.24, $p < .05$)			
					Reward > Loss, controlling for impulsivity	SRP-Lifestyle: ↓ left VS and (trend) right VS			
						SRP-Antisocial: ↑ left VS (trend)			
					Reward > Loss, gender analyses	Men: ns			
						Women: SRP-Affective: ↑ right VS, $B = .014$ (006), $p = .015$; SRP-Lifestyle: ↓ right VS (trend), $B = -009$ (.005), $p = .06$, and left VS, $B = -.010$ (.005), $p = .03$.			
					Inpatient/Incarcerated samples				
Geurts et al., 2016	Total $n = 34$ (34M)	Interview	MID	Reward	Reward Anticipation > No Reward Anticipation	Psychopaths + Controls high on IA vs. Controls low on IA: ↑ VS ($T = 3.30$, $p = .011$ small volume; $T = 5.31$, $p = .049$ whole brain)	N/A	N/A	PPI-IA related to increased VS activity during reward anticipation, independent of criminality. Psychopaths had increased VS–dmPFC functional connectivity vs. Controls.
	Incarcerated psychopaths ($n = 14$)	PCL-R	Monetary	Anticipation		Psychopaths vs. Controls high on IA: ns			High PPI-FD controls had greater periaqueductal gray activity during reward anticipation vs. psychopaths.
	Healthy controls ($n = 20$)	Self-Report PPI: IA and FD scales				Group × Reward expectancy interaction in the periaqueductal gray driven by greater reward reactivity in high FD controls compared with the psychopath group			
Gregory et al., 2015	Total $n = 50$ (50M)	Interview	Probabilistic response reversal	Reward and Loss	Reward > Punished reversal error	APD + P vs. APD-P: ↓ right STG extending to anterior MTG ($Z = 3.68$, $p = .039$)	Punished reversal	APD + P vs. APD-P: ↑ PCC, precuneus ($Z = 3.84$, $p = .001$) and	Offenders with APD and psychopathy had increased activation in

Study	Sample	Measure	Task/Reward	Type/Phase	Reward contrast	Results	Loss contrast	Results	Summary
							error > Reward	right anterior insula (Z = 3.16, p = .044)	cingulate, insula and precuneus to loss, and decreased STG activation to rewards. PCL-R scores positively correlated with posterior cingulate reactivity during punished reversal errors
	Violent offenders with APD and psychopathy (n = 12)	SCID-II	Points	Receipt		APD + P vs. Controls: ↓ right STG extending to anterior MTG (Z = 3.76. p = .019)		APD + P vs. Controls: ↑ PCC and precuneus (Z = 3.47, p = .011)	
	Violent offenders with APD only (n = 20)	PCL-R				APD-P vs. Controls: ns		APD-P vs. Controls: ns	
	Healthy nonoffender controls (n = 18)					APD-P: ↑ PCC and STG		APD-P: ns	
						Controls: ↑ PCC and STG		Controls: ↑ inferior parietal lobe	
						APD + P: ns		APD + P: ↑ inferior parietal lobe and PCC	
Pujara et al., 2014	Prison inmates n = 41: 18 psychopaths and 23 nonps ychopaths (41M)	Interview	Probabilistic slot machine game	Reward and Loss	Reward > Neutral	Between groups: ns	Loss > Neutral	Between groups: ns	Psychopathy severity related to increased VS activity to Reward > Loss, driven mainly by negative correlation to Loss > Neutral
		PCL-R	Monetary	Receipt	Reward > Loss	Within groups:		Within groups:	
						Nonpsychopaths: ns		Nonpsychopaths: ns	
						Psychopaths: ns		Psychopaths: ↓ VS (r = −.61, p = .007)	
						Within groups:			
						Nonpsychopaths: ns			
						Psychopaths: ↑ left VS (r = .74, p = .0004)			
Völlm et al., 2010	Total n = 57 (57M)	Interview	Reward receipt task	Reward	Reward > No reward	APD vs. Controls: increased in right OFC (Z =3.38) and	N/A		Offenders with APD had increased right

Study	Sample	Measure	Task/Reward	Type/Phase	Reward contrast	Results	Loss contrast	Results	Summary
						pregenual cortex (Z = 3.31) $p < .001$ uncorrected			OFC and pregenual cortex activity to rewards compared with controls
	Incarcerated offenders with APD (n = 25) Healthy Controls (n = 32)	SCID-II	Monetary	Blocked design		Main effect of the task was not significant in hypothesized reward-related brain regions.			

Note. M_ male; ACC _ anterior cingulate cortex; APD _ antisocial personality disorder; dmPFC _ dorsomedial prefrontal cortex; FD _ fearless dominance; IA _ impulsive-antisocial; MID _monetary incentive delay task; MTG _ middle temporal gyrus; OFC _ orbitofrontal cortex; PCC _ posterior cingulate cortex; PCL-R _ Psychopathy Checklist–Revised; PPI _ Psychopathic Personality Inventory; PFC _ prefrontal cortex; SCID-II _ Structured Clinical Interview for *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*; SRP-SF _ Self-Report of Psychopathy–Short Form; SRP-Affective _ Self-Report of Psychopathy Affective Facet; SRP-Antisocial _ Self-Report of Psychopathy Antisocial Facet; SRP-Lifestyle _ Self-Report of Psychopathy Lifestyle Facet; STG _ superior temporal gyrus; VS _ ventral striatum.

Table 2

Summary of Results

Valence	Anticipation	Consumption	General
		Ventral striatum	
Reward	↑ (Bjork et al., 2012; Buckholtz et al., 2010; Geurts et al., 2016)		↑ (Carré et al., 2013)
			↓ (Carré et al., 2013)
Loss		↓ (Pujara et al., 2014)	
		Whole-brain	
Reward	↑ dACC (Bjork et al., 2012)		↑ rOFC (Völlm et al., 2010)
	↑ mPFC (Bjork et al., 2012)		↑ pregenual ACC (Völlm et al., 2010)
	↑ VS–dmPFC connectivity (Geurts et al., 2016)		
	↑ PAG (Geurts et al., 2016)		
Loss		↑ Cingulate (Gregory et al., 2015)	
		↑ Insula (Gregory et al., 2015)	
		↑ Precuneus (Gregory et al., 2015)	

Note. ACC [H11005] anterior cingulate cortex; dACC [H11005] dorsal anterior cingulate cortex; dmPFC [H11005] dorsomedial prefrontal cortex; mPFC [H11005] mesial prefrontal cortex; PAG [H11005] periaqueductal gray; rOFC [H11005] right orbitofrontal cortex.