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Antisocial Behavior, Psychopathic Features and Abnormalities in Reward and Punishment Processing in Youth

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Abstract

A better understanding of what leads youth to initially engage in antisocial behavior (ASB) and more importantly persist with such behaviors into adulthood has significant implications for prevention and intervention efforts. A considerable number of studies using behavioral and neuroimaging techniques have investigated abnormalities in reward and punishment processing as potential causal mechanisms underlying ASB. However, this literature has yet to be critically evaluated, and there are no comprehensive reviews that systematically examine and synthesize these findings. The goal of the present review is twofold. The first aim is to examine the extent to which youth with ASB are characterized by abnormalities in (1) reward processing; (2) punishment processing; or (3) both reward and punishment processing. The second aim is to evaluate whether aberrant reward and/or punishment processing is specific to or most pronounced in a subgroup of antisocial youth with psychopathic features. Studies utilizing behavioral methods are first reviewed, followed by studies using functional magnetic resonance imaging. An integration of theory and research across multiple levels of analysis is presented in order to provide a more comprehensive understanding of reward and punishment processing in antisocial youth. Findings are discussed in terms of developmental and contextual considerations, proposed future directions and implications for intervention.

Keywords

Antisocial behavior; Psychopathy; Callous–unemotional; Reward; Punishment; Youth

Many youth engage in some form of antisocial behavior (ASB) in adolescence (ASB; Elliot et al. 1985), and a small subset will continue engaging in ASB into adulthood (Loeber et al. 2008; Moffitt et al. 2002). In the United States alone, over 2 million youth are arrested

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annually (Puzzanchera 2009). Given the negative impact these behaviors have on the youth themselves, the victims of their crimes and society as a whole, there has been a strong emphasis on understanding the mechanisms underlying ASB. A better understanding of what leads youth to engage in ASB has significant implications for prevention and intervention efforts. The aim of the present review is first, to evaluate the extent to which abnormalities in reward and punishment processing may underlie ASB in youth and second, to examine whether these abnormalities are characteristic of a more homogenous subgroup of antisocial youth with psychopathic features who demonstrate severe and protracted forms of ASB.

Theories on the etiology of ASB have emphasized a variety of environmental and biological factors, recognizing that the development of ASB is likely the result of a complex interplay between child, family and social factors. However, given the active role individuals play in shaping their social environments (Patterson et al. 1992) and evidence suggesting that ASB has a significant heritable component (Viding et al. 2008), one prominent area of research has focused on further elucidating child-specific mechanisms. Historically, abnormalities in reward/punishment processing have been conceptualized as one potential causal mechanism (e.g., Newman and Wallace 1993), with the adult literature consistently demonstrating that deficiencies in reward and punishment responsivity are associated with severe and chronic adult offending (e.g., Fowles 2000; Newman and Lorenz 2002). Moreover, the processing of reward and punishment has profound implications for associative and contingency learning, which serve as the foundation for many aspects of behavioral, social and emotional development (Akers 1998; Kochanska 1994).

Gray's reinforcement sensitivity theory (1981, 1987) provides one of the most cited frameworks for understanding reward and punishment processing in humans. Gray proposed the existence of two systems: one which increases activity and initiates goal-directed behavior in response to reward, while the other serves to inhibit action and avoid aversive stimuli or punishment. Modifications of this theory have focused on associations between ASB and primary deficits in one or both of these systems. Drawing upon these theories, considerable behavioral and, more recently, neurobiological work has been devoted to better understand the extent to which abnormalities in reward and/or punishment processing characterize antisocial youth. Moreover, empirical work has attempted to pinpoint whether these abnormalities may help to delineate a subset of youth, characterized by psychopathic features, who are at heightened risk for severe and chronic ASB. However, while recent work in this area has provided a comprehensive overview of these processing systems in antisocial youth (Matthys et al. 2012a, b; Patrick et al. 2012), empirical studies have yet to be systematically evaluated, and at present, there are no reviews that seek to integrate and interpret these findings.

Along these lines, the current review seeks to accomplish several aims. The first is to examine the extent to which antisocial youth are characterized by abnormalities in (1) reward processing; (2) punishment processing; or (3) both reward and punishment processing. The second is to evaluate whether aberrant reward and/or punishment processing is specific to or most pronounced in a subgroup of antisocial youth with psychopathic features. To meet these goals, this review will first focus on extensive behavioral research

that has examined reward/punishment processing in youth exhibiting ASB. Next, to highlight the complexities of these processes and examine potential deficits in the underlying neural circuitry, neurobiological theory and research using functional magnetic resonance imaging (fMRI) will be reviewed. Lastly, this review will integrate theory and research across multiple levels of analysis to provide a more comprehensive understanding of reward and punishment processing in antisocial youth.

Defining Terms

Antisocial Behavior

ASB refers to actions or attitudes that violate societal norms and/or the personal and property rights of others (American Psychiatric Association 2000). This includes oppositional defiant disorder (ODD), defined as early behaviors that tend to be more irritable and defiant in nature, and conduct disorder (CD), defined as more severe behaviors such as aggression toward people and animals, destruction of property and theft or deceitfulness. ASB is also defined using broadband behavioral measures (e.g., externalizing behaviors), often referred to as conduct problems (CP) and delinquency (e.g., illegal acts). While these constructs include behaviors that are largely consistent with DSM-IV symptoms of CD/ODD, there is still notable variability in type and severity. The current review seeks to focus on more severe forms of ASB and as such, less attention will be given to behaviors that reflect early manifestations of irritability and defiance.

Subtypes

Given the noted heterogeneity that exists among youth exhibiting ASB (Frick and White 2008) and empirical work suggesting many youth desist from ASB by adulthood (Farrington 1986; Laub and Sampson 2001), researchers have proposed various subtyping schemes in hopes of delineating a more homogenous subgroup of youth at risk for exhibiting persistent ASB. For example, distinctions have been made between early (i.e., childhood) versus late (i.e., adolescent) onset ASB. Specifically, early manifestations of ASB have been linked to a prolonged, stable course of problem behaviors while adolescent onset is thought to be more normative and transient, reflecting a ‘maturity gap’ and contextual influences (Moffitt 1993). More recently, considerable focus has been directed toward the presence of psychopathic features in youth, as research has consistently shown these characteristics to be associated with severe and protracted engagement in ASB (e.g., Byrd et al. 2012; Frick and White 2008; Pardini and Loeber 2008). These features are comprised of a constellation of interpersonal (e.g., superficial charm, manipulative and deceitful behavior), affective (e.g., lack of empathy and remorse) and behavioral (e.g., impulsivity, irresponsibility) characteristics, though some researchers have focused exclusively on the affective dimension (i.e., callous–unemotional (CU) traits). These features are particularly relevant because they are not adequately represented among current definitions of ASB, with their estimated prevalence ranging from 10 to 46 % in community samples to 21–59 % in clinic samples of antisocial youth (see Pardini and Frick 2013). Moreover, these features are thought to be indicative of distinct causal processes that lead to a heightened proclivity for persistent ASB (Dadds et al. 2005; Frick et al. 2000; Pardini 2006). Specifically, research suggests that one potential etiological pathway may be related to a reward-dominant response style and

deficient punishment processing that increases risk for engagement in ASB (Frick et al. 2003; Frick and Marsee 2006; Pardini 2006). As such, the current review seeks to investigate the extent to which abnormalities in reward and punishment processing are specific to or most pronounced in a subset of antisocial youth characterized by psychopathic features.

Reward and Punishment Processing and Antisocial Behavior

Historically, reward and punishment processing has been conceptualized in terms of the behavioral activation system (BAS) and the behavioral inhibition system (BIS; Gray 1981, 1987). These systems serve to facilitate the redirection of attention to relevant environmental stimuli and initiate subsequent behavioral responses. Specifically, the BAS serves to increase activity and initiate goal-directed behavior in response to reward, while the BIS functions to inhibit goal-directed action and avoid negative, painful, or threatening outcomes in the presence of aversive stimuli or punishment. Gray (1981, 1987) viewed variations in the functioning of the BAS as an index of reward sensitivity, with heightened levels of BAS activation resulting in increased propensity to engage in reward-directed behavior. Conversely, the BIS is sensitive to cues of punishment, with heightened activation in this system manifesting behaviorally as increased avoidance behaviors and a greater proneness to feelings of anxiety. Lower BIS reactivity is thought to be associated with insensitivity to punishment/threat and reduced anxiety, leading to increased reward-seeking behaviors.

Extensions and modifications of this theory have focused on associations between ASB and primary deficits in one or both of these systems. Quay (1993) was one of the first to extend Gray's (1981, 1987) theory to manifestations of ASB in youth, suggesting that these behaviors stem from an overactive reward system (BAS). Specifically, Quay (1993) proposed that the excessive BAS activity seen in antisocial youth results in a reward-dominant response style that predominates over the BIS and leads to persistent approach or reward-seeking behaviors. Others proposed that ASB instead reflects an inherently underactive reward system that facilitates sensation seeking (Cloninger 1987; Zuckerman 1996). This theory suggests that a chronically underactive reward system results in an aversive physiological state, requiring individuals to seek stimulation (e.g., antisocial behavior) in order to increase arousal and alleviate an unpleasant internal condition.

In contrast to a central focus on reward, researchers have also posited a primary deficit in punishment processing, where ASB is reflective of a hypoactive inhibition system and insensitivity to cues of punishment (Fowles 1980; Lykken 1995). This theory suggests that individuals with reduced BIS reactivity experience little negative arousal in response to punishment and consequently have difficulty learning from cues indicative of punishment. Due primarily to a notable reduction in conditioned anxiety/fear of punishment, these individuals demonstrate difficulties inhibiting reward-seeking behaviors (e.g., ASB) even when these behaviors result in punishment. Incorporating aspects of both reward and punishment systems, Newman and colleagues (Patterson and Newman 1993; Wallace and New-man 2008) proposed that ASB can be attributed to deficits in the modulation of both systems, specifically when the avoidance of punishment requires overriding or suspending a dominant reward response. This theory posits that a failure to allocate attention to

punishment cues that may be less salient in situations involving competing reward- or goal-driven behavior underlies chronic forms of ASB.

In light of the heterogeneity that exists among individuals with ASB, researchers have also examined BAS/BIS function in subgroups of antisocial youth with psychopathic features. This body of research takes a more fine-grained approach and posits differential associations between BAS/BIS and variants of psychopathy. While the assessment of these variants, often referred to as primary versus secondary psychopathy, is somewhat controversial, research suggests that certain characteristics may serve to parse differences in etiology within subgroups of individuals with psychopathy (Kimonis et al. 2012; Kimonis et al. 2011; Poythress et al. 2010). Moreover, these characteristics have been linked to BAS/BIS functioning and are thus particularly relevant for the current review. One distinction focuses on the presence (or absence) of high levels of anxiety (Karpman 1941; Skeem et al. 2003). Specifically, psychopathy in the absence of high anxiety is believed to be characterized by hypoactive BIS reactivity that drives chronic ASB by fostering punishment insensitivity; however, the BAS is believed to be relatively intact (Lykken 1995; Newman et al. 2005). The absence of high anxiety has been linked to the 'core' interpersonal and affective features of the disorder (e.g., CU traits), which are believed to reflect an inherent underlying deficit in emotionality (Cleckley 1941). Conversely, psychopathic individuals with high anxiety are characterized by an overactive BIS and BAS that manifests as emotion dysregulation (i.e., increased anxiety, anger) and high levels of impulsivity and ASB (Lykken 1995; Newman et al. 2005). Throughout the current review, distinctions based on (1) level of anxiety and (2) the presence of 'core' psychopathic features (i.e., CU traits) will be highlighted with regard to interpretation of findings among studies investigating youth with psychopathic features.

Models of Contingency Learning and Antisocial Behavior

Classical and operant conditioning are two fundamental models of contingency learning that provide a framework for understanding how aberrant reward/punishment processing may give rise to the development and persistence of ASB in youth.

Classical Conditioning

Classical conditioning involves learned associations between reflexive responses and antecedent stimuli or events (Pavlov 1927). This process is achieved through the pairing of a stimulus of significance (unconditioned stimulus; UCS) with another stimulus or cue (conditioned stimulus; CS). The UCS evokes a specific reflexive response (unconditioned response; UCR) that is eventually transferred to the CS through continued pairing of the two stimuli. Thus, a new association is formed between the CS and the UCR, resulting in a conditioned response (CR) that can be induced by the CS alone. Many researchers have applied models of classical conditioning to the engagement and persistence of ASB (e.g., Damasio 1994; Kochanska 1994). Overall, these theoretical models emphasize the importance of reflexive emotional responses to punishment and suggest that individual differences in punishment sensitivity may have cascading effects that ultimately increase the likelihood of engaging in ASB. For example, Kochanska (1994) emphasized the role of classical conditioning in conscience development, suggesting that the internalization of

social norms is facilitated by the negative emotional arousal a child experiences when being disciplined or punished for wrongdoing. Eventually, this emotional arousal becomes paired with the act of wrongdoing, and through repeated disciplinary interactions, youth likely become conditioned to experience increases in negative arousal when contemplating or engaging in misconduct. Importantly, this occurs even in the absence of an authority figure and is a key feature of guilt and remorse. Youth who are particularly insensitive to punishment, as evidenced by low anxiety and low temperamental fearfulness, experience relatively little negative emotion when punished for rule-breaking behavior and thus fail to establish conditioned associations between disciplinary interactions and emotional arousal. These youth may be less likely to encode parental messages about the acceptability of behaviors, which could hinder the internalization of moral beliefs that guide prosocial behavior. Research suggests that punishment insensitivity is linked to reduced concern for the suffering of others (Young et al. 1999) and less guilt following transgressions (Rothbart et al. 1994), characteristics that increase the likelihood that one will engage in ASB. Along these lines, Blair (2004) proposed that antisocial individuals, particularly those with psychopathic features, fail to encode the emotional components of reward and punishment and are thus unable to acquire conditioned associations. For example, distress cues in others, such as sad or fearful expressions that occur following an act of their own wrongdoing, fail to elicit negative emotional arousal and in turn impede the acquisition of important affective associations. This is believed to have downstream consequences and hinder the development of empathy and remorse, resulting in a heightened risk for engagement and persistence of ASB.

Operant Conditioning

Operant conditioning refers to the learning and modification of non-reflexive, voluntary behaviors through contingent stimuli or feedback (i.e., reward/reinforcement or punishment). It is founded on the principle that behaviors that are reinforced or rewarded are strengthened and perpetuated, while punished behaviors are weakened and diminished (Skinner 1969). However, factors such as consistency, proportion and immediacy mitigate the impact that rewards/punishments have on behavior. Specifically, a consistent, proportionally relevant consequence that occurs immediately following a target behavior will be most likely to influence subsequent behavior. Social learning theories of ASB rely heavily on operant principles, suggesting that parents may unintentionally reinforce ASB while simultaneously failing to reinforce prosocial behaviors (Patterson et al. 1992). For example, Patterson et al. (1992) focused on a coercive cycle that exists in parent–child interactions, whereby parents respond to ASB with inconsistent and harsh punishment techniques that serve to escalate the severity and frequency of problem behaviors over time. Accordingly, a central component of parenting interventions is to break this coercive cycle by shifting behavioral contingencies and teaching parents to positively reinforce prosocial behaviors, while setting clear and consistent consequences for disruptive behaviors (Kazdin 2003; Serketich and Dumas 1996). It is possible that these negative parenting behaviors, such as harsh and inconsistent discipline, may be particularly detrimental for youth with deficits in reward/punishment processing and could serve to exacerbate ASB (see Dadds and Salmon 2003; Matthys et al. 2012b). Along these lines, research suggests that youth with increased reward-seeking and punishment insensitivity respond well to the reward-based components of parent training

(e.g., praise, token reinforcement) yet appear insensitive to the disciplinary components of treatment (e.g., time-out; Hawes and Dadds 2005). This suggests that deficits in reward/punishment processing not only help us to understand the development of ASB but are also particularly important with regard to tailoring intervention techniques to achieve optimal effectiveness.

Researchers have also explored ways in which peers function to reinforce and maintain ASB, suggesting that deviant peer interactions reinforce and perpetuate ASB (i.e., deviancy training; Dishion et al. 1996; Patterson et al. 2000). Specifically, association with deviant peers has been consistently linked to higher levels of risk taking and ASB over time (Rusby et al. 2005), with this association being most pronounced during adolescence (Dishion et al. 2004). The presence of deviant social influences may be particularly prominent for those youth with a reward-dominant response style and decreased sensitivity to punishment. For example, an adolescent who is hypersensitive to reward and unconcerned with potential punishment may be more likely to engage in ASB if it results in positive reinforcement from peers. Thus, these deficits may make youth more vulnerable to negative social influences.

Developmental Considerations

Adolescence marks a transition period noted for drastic physical, psychological and social development, and most pertinent to the current review are marked normative increases in risk-taking and reward-seeking behaviors that are thought to reflect still-developing neurobiological systems (Steinberg and Morris 2001). Specifically, research documents a heightened neural sensitivity to reward when comparing typically developing adolescents to adults (Ernst et al. 2005; Galvan et al. 2006) and consistently demonstrates underdeveloped regulatory regions in adolescents (Fareri et al. 2008; Luna et al. 2001). This coupled with decades of research showing that the prevalence of antisocial behavior rises and peaks during adolescence and then greatly dissipates for most individuals by adulthood (e.g., Farrington 1986) further underscores the need to consider atypical processes from a baseline of typical development (Cummings et al. 2000; Sroufe and Rutter 1984). Along these lines, manifestations of ASB and psychopathic features also change over time, specifically with regard to frequency and severity (Edens et al. 2001; Hinshaw and Lee 2003). Consequently, research on the mechanisms underlying these behaviors at various points in development may have very different implications, and efforts to maintain a more developmentally homogenous sample are particularly important with regard to interpretation. Thus, the present review aims to narrow the developmental window to that of late childhood and adolescence and considers potential developmental differences in reported findings.

Summary

While considerable empirical work has evaluated the extent to which antisocial youth manifest abnormalities in reward and/or punishment processing, a systematic review of this literature is necessary to examine several core theoretical issues. This involves the extent to which antisocial youth are characterized by a primary deficit in reward processing, a primary deficit in punishment processing, or deficits in the modulation of both reward and punishment processing. Second, there has not been a critical evaluation of the degree to

which these deficits may be specific to a subset of youth characterized by psychopathic features and most at risk for persistent ASB.

Inclusion/Exclusion Criteria

Peer-reviewed, English language studies published online through December 2012 were located from (a) reference lists of prior narrative reviews and individual studies and (b) major publication databases (e.g., PubMed) using the following key words: antisocial behavior, conduct disorder/problems, delinquency, externalizing behavior, aggression, youth, psychopathy, CU traits, reward, punishment, learning, conditioning, fMRI and adolescence. The results of the literature search were filtered according to the following criteria:

Construct

In an attempt to focus on more severe ASB, only those studies that assessed CD symptoms, conduct problems and delinquency were included. Because ODD symptoms are conceptualized as early age-inappropriate behaviors and thus less severe (American Psychiatric Association 2000), studies defining ASB as ODD were only included when ODD symptoms were combined with CD symptoms. In addition, any studies assessing psychopathic features among youth with ASB were included. Due to the limited number of studies in this area, the current review included studies regardless of their measure of psychopathic features, though distinctions are made between ‘psychopathic features’ (i.e., interpersonal, affective, behavioral, antisocial facets) and ‘CU traits.’

Age

In light of the developmental considerations outlined above, the current review attempted to narrow the developmental window to include late childhood (age 10–12) and adolescence (age 13–17). Of note, wide age ranges were common (i.e., 6–18 years), resulting in the inclusion of some youth younger than 10 and older than 17.

Study Design

Only studies utilizing behavioral tasks that included the administration of reward and/or punishment were included in this review. This was done in attempt to examine reward and punishment processing in vivo and under experimental manipulation. While several studies have been conducted using self-report of reward and punishment sensitivity (e.g., Pardini and Byrd 2012; Pardini et al. 2003), these were not included here due to a potential lack of insight into these more automatic processes. Studies utilizing both cross-sectional and longitudinal design were included in order to maximize the number of studies reviewed.

Behavioral Research

Extensive behavioral work has attempted to disentangle the contributions of reward and punishment processing in antisocial youth using a variety of behavioral paradigms including (a) risk taking; (b) psychophysiological; (c) passive avoidance; and (d) response reversal. Each of these areas is reviewed below. A brief description of the task is provided followed

by a critical evaluation of findings in (a) antisocial youth and (b) antisocial youth with psychopathic features.

Risk-Taking Tasks

Research examining 'risk taking,' or behavior in response to immediate reward despite potential punishment, has used the Iowa Gambling Task (IGT; Bechara et al. 1994), the Risky Choice Task (RCT; Rogers et al. 2003) and the Balloon Analogue Risk Task (BART; Lejuez et al. 2002). In both the IGT and RCT, youth are given the choice between 2 or more stimuli (e.g., deck of cards). Selection of one deck (e.g., 'risky deck'), results in a higher magnitude of reward (e.g., +$1,000) or an even larger magnitude of punishment (e.g., −$1,250), while the other deck results in a lower magnitude of reward (e.g., $500) or a lower magnitude of punishment (e.g., −$250) and ultimately yields the greatest reward over time. Conversely, the BART has no fixed probability of reward/punishment, and almost all responses are rewarded. Across multiple trials, participants accumulate money by pressing a button that inflates a balloon and are able to transfer their money into a permanent bank at any time. However, the balloon 'pops' after a preset number of button presses, resulting in a loss of all money.

Antisocial Youth: Late Childhood—Only one study that investigated risk taking in late childhood was identified (Table 1). This study utilized the BART and found self-reported ASB to be unrelated to the level of risk taking both concurrently and longitudinally in a racially diverse community sample of males and females (MacPherson et al. 2010). While youth in this study were assessed for behaviors that are consistent with CD symptoms and delinquency (e.g., stealing, fighting, etc.), more minor behaviors (e.g., not wearing a seatbelt) were also included, and thus, the severity of ASB may be less comparable.

Antisocial Youth: Adolescence—Four studies examining risk taking in adolescents with ASB were identified (Table 1). First, a community sample of males characterized by either early-onset or adolescent-onset CD were found to exhibit significantly more risk taking than healthy controls; however, there were no differences between CD groups (Fairchild et al. 2009). Second, Miura (2009) compared risk taking in youth convicted of a violent crime to that of youth convicted of a non-violent crime. Both groups selected the risky deck at a rate greater than chance, though there was no control group and it is unclear whether risk taking in these groups differed from that of a normative sample. In another offender sample, Syngelaki et al. (2009) found male offenders to engage in more risk taking than healthy controls and this was driven by a strong tendency to make risky decisions following small rewards though no differences were seen following large rewards. A fourth study found that institutionalized males and females with ASB and substance dependence were significantly more likely to make risky decisions than healthy controls (Schutter et al. 2011). Additionally, antisocial youth with substance dependence exhibited greater risk taking than antisocial youth without substance dependence.

Antisocial Youth with Psychopathic Features: Adolescence—All four studies examining risk taking in antisocial youth with psychopathic features focused on predominately male adolescent samples (Table 1); however, two of these studies had wide

age ranges (i.e., ages 9–17) and included youth in late childhood (Blair et al. 2001; Marini and Stickle 2010). First, Blair et al. (2001) found that antisocial youth with high psychopathic features chose risky decks significantly more often than antisocial youth with low psychopathic features. Moreover, analyses examining changes in deck selection across all trials found antisocial youth with high psychopathic features chose the risky deck at a consistently higher rate. This was most notable at the end of the task (40 vs. 25 % in healthy controls), suggesting a failure to learn to avoid disadvantageous decks. Fairchild et al. (2009) also found psychopathic features to be associated with greater risk taking in a community sample of youth with CD, further suggesting these abnormalities may be most pronounced within this subgroup of antisocial youth. Two final studies examined CU traits and associations with risk taking using the BART. In a sample of offenders, there was no association between CU traits and overall risk taking. After controlling for sensation seeking and impulsivity, CU traits were associated with *less* risk taking following reward (i.e., the number of button presses following a non-popped balloon; Marini and Stickle 2010). Similarly, Centifanti and Modecki (2013) examined the association between CU traits, risk taking and peer influence in a large community sample and found no association between CU traits and overall risk taking after accounting for gender and peer influence. Moreover, CU traits were associated with *less* risk taking following reward. However, when in the presence of peers, those youth with high CU traits responded more quickly following punishment than youth with low CU traits.

Summary—Taken together, research suggests that adolescent youth with ASB exhibit an increased tendency to pursue large, immediate rewards despite the possibility of punishment. Interestingly, no differences in risk-taking behavior were seen within groups of antisocial adolescents when subtypes were based on the presence of severe violence ASB (Miura 2009) or age of onset (Fairchild et al. 2009), though there was some suggestion that the presence of substance dependence may be linked to increased risk taking in antisocial youth (Schutter et al. 2011). Moreover, among antisocial youth, increased psychopathic features (broadly defined) were associated with higher levels of risk taking (Blair et al. 2001; Fairchild et al. 2009), providing some evidence for the notion that these deficits may be more pronounced within this subgroup. However, those studies examining CU traits specifically found no significant differences in overall risk taking (Centifanti and Modecki 2013; Marini and Stickle 2010), suggesting that reward sensitivity and heightened risk taking may be less specific to the core affective features of psychopathy. It is also important to note that those studies assessing CU traits utilized the BART, which differs from other risk-taking tasks in a number of ways (Bishara et al. 2009). Namely, there is no fixed probability of reward/punishment, limiting opportunities for learning. Moreover, the BART isolates response to reward by reinforcing the majority of responses. This suggests that antisocial youth with CU traits show no impairment on tasks that isolate reward function, while antisocial youth with psychopathic features (broadly defined) show more consistent deficits on risk-taking tasks that necessitate the inhibition of reward-seeking behaviors in the face of probable punishment.

In sum, researchers conclude that antisocial youth, particularly those with psychopathic features (broadly defined), are characterized by increased risk taking and a reward-dominant

response style that may reflect abnormalities in reward responsivity. However, these findings should be considered with several factors in mind. First, the inherent presence of reward and punishment in these paradigms makes it difficult to specify the underlying mechanism, as abnormalities in reward and/or punishment processing may be responsible for these effects. Second, only two of the positive studies included a control sample (Fairchild et al. 2009; Schutter et al. 2011), making it unclear whether or not these deficits are specific to or most pronounced in youth exhibiting ASB or instead indicative of more normative increases in risk taking among adolescents (Centers for Disease Control & Prevention 2006). As such, the inclusion of healthy controls is particularly important. Lastly, while null findings were linked to the BART task, negative studies were also characterized by samples of youth in late childhood (MacPherson et al. 2010; Marini and Stickle 2010). This is particularly relevant given the neurodevelopmental changes that occur during the transition into adolescence (Steinberg 2008) and may suggest later emergence of these deficits.

Psychophysiological Tasks

Empirical paradigms utilizing psychophysiological measures examine reflexive responses to reward and punishment, although the majority of studies focus on individual differences in responsivity to inherently aversive stimuli (e.g., loud tones). Two measures have been utilized to examine autonomic responsivity to reward/punishment in youth with ASB: (1) skin conductance response (SCR) which refers to changes in electrical conductivity of the skin and (2) eye-blink startle, a measure that records individual differences in the eye-blink reflex (Mazur 2006). These measures have been assessed in three different experimental designs (1) uncued: responsivity to randomly presented aversive stimuli; (2) cued: responsivity to cue-stimulus contingencies in aversive conditioning paradigms; and (3) task: responsivity during a task that includes reward and/or punishment.[1]

Antisocial Youth: Late Childhood—Three studies examined physiological responsivity in late childhood (Table 3). Two studies utilized uncued designs and one used a task design. First, van Goozen et al. (2004) found males and females with CD/ODD to show reduced eye-blink startle to uncued aversive tones compared to healthy controls. Additionally, Herpertz et al. (2001) found males with CD and attention deficit hyperactivity disorder (ADHD) to show reduced SCR to uncued aversive tones and more rapid habituation relative to youth with ADHD alone and healthy controls. However, in the same sample, no group differences emerged in eye-blink startle for the same uncued tone when presented during emotionally valenced slides, though it was speculated that habituation effects might have played a role. Lastly, Matthys et al. (2004) examined differences in physiological responsivity during a response reversal task that included the administration of reward and punishment. Youth with CD/ODD demonstrated reduced SCR relative to healthy controls; however, it is unclear whether or not this is specific to reward or punishment as SCR were averaged across all trials.

[1]While extensive research has examined baseline levels of autonomic responsivity (for review see Lorber 2004; Raine 1996), this is beyond the scope of the current review which focuses specifically on responsivity to reward/punishment.

Antisocial Youth: Adolescence—Seven studies examined psychophysiological responsivity in adolescent males with ASB (Table 2), with five studies using a cued experimental design, one using an uncued design and one using a task design. In the first study of its kind, Raine and Venables (1981) found youth exhibiting ASB to show reduced SCR to cues signaling impending punishment. In a follow-up study, these same youth were classified into three groups (i.e., persisters, desisters, non-delinquents) based on official record of conviction up to age 29 (Raine et al. 1996). Youth with persistent patterns of delinquency exhibited reduced SCR to punishment cues relative to youth who desisted from delinquency by adulthood, even after accounting for age, socioeconomic status, neighborhood and level of academic functioning. However, groups did not differ on level of responsivity to the cued aversive stimulus, suggesting that youth with persistent ASB may have particular difficulties learning cue-stimulus contingencies. In a similar study, Loeber et al. (2007) classified community-recruited youth into the same three groups (i.e., persisters, desisters, non-delinquents) based on their engagement in moderate/serious delinquency across four developmental time periods (age range = 7–20). Both delinquent groups showed reduced SCR to cues of impending punishment relative to non-delinquents; however, persisters and desisters did not differ in their level of responsivity. Additionally, all groups had similar levels of responsivity to the cued aversive stimulus. Fairchild et al. (2008) also found youth with CD, regardless of age of onset, to show reduced SCR to punishment cues relative to healthy controls, despite being able to identify the cue-stimulus contingency in a post-task questionnaire. Moreover, all groups demonstrated similar levels of responsivity to the cued aversive stimulus. Replicating these results, a recent study found a group of offenders to demonstrate poorer responsivity to cues of impending punishment relative to healthy controls, while SCR to the cued aversive stimulus did not differ from healthy controls (Syngelaki et al. 2013).

One additional investigation examined differences in responsivity to uncued, randomly presented aversive tones. Youth with CD, irrespective of their age of onset, evidenced reduced eye-blink startle to aversive tones relative to healthy controls (Fairchild et al. 2009). Lastly, Beauchaine et al. (2001) examined SCR during performance of a repetitive motor task in which rewards (e.g., money) were administered and removed across trials. Youth with CD/ADHD did not differ from youth with ADHD alone or healthy controls in their levels of responsivity when they failed to receive expected reward.

Antisocial Youth with Psychopathic Features: Adolescence—Only one study investigated psychophysiological responsiveness in males with psychopathic features (Table 2). In an at-risk community sample of males, youth with high psychopathic features demonstrated reduced SCR to both cues of impending punishment and the cued aversive stimulus relative to youth with low psychopathic features (Fung et al. 2005). When participants were re-classified as ‘high delinquents’ (i.e., moderate or serious theft/violence) and ‘low delinquents’ (i.e., no delinquency or minor delinquency), the same findings emerged, suggesting this was not specific to the presence of psychopathic features. Furthermore, while a greater proportion of ‘high delinquents’ had psychopathic features that exceeded the 80th percentile, no significant differences in SCR emerged between those delinquent youth with high versus low psychopathic features.

Summary—Empirical work investigating a primary deficit in punishment processing among youth with ASB demonstrates consistent results across both late childhood and adolescence. Youth exhibiting ASB tend to show SCR to uncued aversive stimuli indicating that they are less aroused when punished (Fairchild et al. 2008; Herpertz et al. 2001; van Goozen et al. 2004). Antisocial youth also show reduced responsiveness to cues signaling impending punishment (Fairchild et al. 2008; Loeber et al. 2007; Raine and Venables 1981; Raine et al. 1996; Syngelaki et al. 2013), suggesting a deficit in the acquisition of cue-stimulus associations. Taken together, this suggests that youth with ASB experience relatively little physiological arousal when punished and this hinders their ability to form associations with cues of impending punishment. This is consistent with Kochanska's (1994, 1997) theoretical model, which posits that youth with punishment insensitivity may fail to establish conditioned associations between disciplinary action and wrongdoing, increasing their likelihood of engaging in ASB. At the same time, there is some evidence to suggest that antisocial youth can cognitively identify cue-punishment pairing in a post-task questionnaire (Fairchild et al. 2008), indicating that they may be cognitively aware of these contingencies but exhibit deficits in the affective component of conditioning. However, youth with ASB did not show deficits in physiological responsivity to cued aversive stimuli relative to healthy controls (Fairchild et al. 2008; Loeber et al. 2007; Raine et al. 1996; Syngelaki et al. 2013). It is possible that this occurs because youth are less likely to experience significant arousal to predictable aversive stimuli regardless of their level of ASB. Lastly, while some longitudinal work indicates that punishment insensitivity may help to explain persistent ASB (Raine et al. 1996), Loeber et al. (2007) found these deficits did not prospectively distinguish between desisting and persisting offenders.

In light of the evidence presented above, it is important to consider the one study that failed to find a reduced responsivity to punishment (Beauchaine et al. 2001). The task design differs significantly from the other tasks reviewed in this section in that punishment was characterized as a failure to receive reward (vs. presentation of an aversive stimulus) and may be more related to error monitoring as opposed to primary punishment processing. These processes may index different underlying neural circuitries and in turn differentially impact autonomic responsivity (see below for a description of neural processing systems).

Finally, despite long-standing theory that punishment insensitivity is characteristic of individuals with psychopathic features, the only study that attempted to examine this empirically found no differences in responsivity among delinquent youth with high versus low psychopathic features (Fung et al. 2005). Given the consistent findings described above, one may conclude that these deficits are not specific to this subgroup of antisocial youth. At the same time, it is important to note that research in this area is limited and Fung et al. (2005) failed to differentiate between variants of psychopathy as indexed by level of anxiety or the presence of core psychopathic features (i.e., CU traits). Failure to address these key distinctions could contribute to null findings, as these features are differentially associated with BAS/BIS function and, therefore, with varying degrees of punishment sensitivity.

Passive Avoidance Tasks

Passive avoidance tasks, such as the avoidance loss of reward paradigm (PALR; Newman et al. 1985), incorporate aspects of both reward and punishment and require participants to learn by trial and error. In this task, responses to half of the stimuli result in reward (e.g., money), while responses to the other half of the stimuli result in punishment (e.g., loss of money). Over repeated trials, participants learn to respond to cues of reward (e.g., touch a card) and inhibit responses to cues of punishment (e.g., refrain from touching a card). Dependent measures include passive avoidance or commission errors (responses to cues of punishment) and omission errors (lack of responses to cues of reward). Three different conditions have been utilized: (1) mixed (i.e., positive stimuli are rewarded, negative stimuli are punished, with no consequences for omission); (2) reward only (i.e., participants are rewarded for responding to positive stimuli and for correct passive avoidance); and (3) punishment only (i.e., participants are punished for negative stimuli and omission errors).

Antisocial Youth: Adolescence—The two empirical investigations using the PALR task in adolescent youth with ASB produced conflicting findings (Table 3). In a clinical/community sample of males and females, participants completed mixed and punishment only conditions. CD symptoms were significantly associated with more passive avoidance errors in the mixed incentive condition. However, CD symptoms were unrelated to performance in the punishment only condition (Hartung et al. 2002), suggesting youth were able to inhibit responses to negative stimuli when the possibility of reward was removed. Somewhat contrary to these findings, a longitudinal investigation in a community sample of at-risk males and females found CD symptoms to be unrelated to performance regardless of condition (i.e., mixed, reward only, punishment only) (Castellanos-Ryan et al. 2011).

Antisocial Youth with Psychopathic Features: Adolescence—Three studies examining passive avoidance learning in adolescent males with psychopathic features were identified (Table 3). First, Newman et al. (1985) examined performance in mixed and punishment only conditions among clinically referred youth with heightened psychopathic features and ‘high’ versus ‘low’ anxiety. Youth with high psychopathic features and low anxiety committed more passive avoidance errors in the mixed incentive condition relative to high psychopathic youth with high anxiety and non-psychopathic youth with low anxiety. However, in the reward only condition, no significant performance differences were found between groups. A more recent study found youth with high psychopathic features and low anxiety to show more passive avoidance errors during a mixed condition when compared to youth with low levels of psychopathic features and low anxiety (Vitale et al. 2005). However, there were no differences between youth with high psychopathic features and high versus low anxiety. A final study utilized a mixed incentive condition and found no significant differences in passive avoidance errors between groups of incarcerated youth with high versus low psychopathic features, though the role of anxiety was not examined (Scerbo et al. 1990). Nonetheless, youth with high psychopathic features committed fewer omission errors and responded to more reward stimuli relative to youth with low psychopathic features.

Summary—Research examining passive avoidance learning in adolescent youth with ASB is limited and inconsistent. While results from one study suggest that deficits in antisocial youth may be most evident in the presence of competing rewards and punishment (Hartung et al. 2002), one of the few longitudinal investigations found ASB to be unrelated to task performance whether reward and punishment were presented simultaneously or alone (Castellanos-Ryan et al. 2011). This may be related to differences in the severity of ASB between clinical and community samples, respectively, or differences in study design (cross-sectional vs. longitudinal). It is also possible that deficits in passive avoidance learning (i.e., learning by trial error in the presence of competing reward and punishment) are less characteristic of antisocial youth as a whole. Along these lines, adolescent youth with ASB and psychopathic features demonstrated the most pronounced deficits when required to inhibit a rewarded response in the face of potential punishment. This was evidenced by a failure to avoid punished stimuli in the context of competing reward (Newman and Kosson 1986; Vitale et al. 2005) and an increased responsivity to rewarded stimuli (Scerbo et al. 1990). Moreover, when antisocial youth with psychopathic features were sufficiently motivated by cues of reward in the absence of punishment (i.e., reward only condition) deficits in passive avoidance learning were eliminated (Newman and Kosson 1986), suggesting that abnormalities may be specific to punishment processing particularly when in the presence of competing reward. Along these lines, psychopathic features were only associated with deficits in passive avoidance learning in the absence of high anxiety (Newman et al. 1985; Vitale et al. 2005), which is hypothesized to be indicative of a hypoactive BIS and reduced punishment sensitivity (Gray 1987). Importantly, the only study that failed to find differences in passive avoidance errors did not account for level of anxiety (Scerbo et al. 1990), which is believed to parse etiological heterogeneity among individuals with psychopathic features. In sum, findings appear consistent with the idea that antisocial youth with psychopathic features demonstrate the most pronounced difficulties when they are required to inhibit a prepotent reward response in the face of impending punishment (Newman and Wallace 1993), with some suggestion that this may be driven by punishment sensitivity.

Response Reversal Tasks

Like passive avoidance tasks, response reversal paradigms (Daughtery and Quay 1991; Newman et al. 1987) include reward and punishment, and require participants to learn by trial and error. However, these paradigms vary the probability of contingent reward/punishment throughout the task, requiring participants to adjust their performance. Typically, participants start with a 90 % probability of receiving a reward (e.g., money) and a 10 % chance of receiving punishment (e.g., loss of money). After every 10 stimuli, the probability changes by 10 % until there is 0 % probability of reward and 100 % probability of punishment. Participants are instructed that they can stop playing the game at any time and collect the money they have accumulated, with poorer performance reflecting a greater number of played stimuli.

Antisocial Youth: Late Childhood—As depicted in Table 4, seven studies utilizing response reversal paradigms were identified in late childhood. All but one of these samples were male and many also included youth in middle childhood. First, a community sample of

youth with CP showed poorer performance than youth with increased anxiety and healthy controls (Daughtery and Quay 1991). Similarly, in a clinical sample of youth, those with CD demonstrated poor performance relative to healthy controls (Fonseca and Yule 1995). Matthys et al. (1998, 2004) replicated these results in two samples of antisocial youth between the ages of 7 and 12, finding youth with CD/ODD to show poorer response reversal learning relative to healthy controls. Additionally, youth with CD/ODD responded more quickly following punishment, but showed no differences in response times following reward (Matthys et al. 2004). Two studies by O'Brien et al. (1994, 1996) also found youth with ASB to show poor performance on response reversal tasks, and group differences were related to the presence of anxiety symptoms. In the first study, significant differences in task performance between youth with CD/ODD and healthy controls emerged only after controlling for co-occurring anxiety symptoms (O'Brien et al. 1994). Echoing these findings, O'Brien and Frick (1996) found youth with CD/ODD to respond to significantly more stimuli than youth with CD/ODD and a comorbid anxiety disorder. Moreover, while CD/ODD youth performed significantly worse than healthy controls, CD/ODD youth with an anxiety disorder did not.

Lastly, while cross-sectional studies have consistently found antisocial youth to show poorer performance on response reversal tasks, the only known longitudinal study failed to show performance differences among youth assessed prospectively for delinquency (White et al. 1994). Youth from an at-risk community sample were classified as 'non-delinquents,' 'other delinquents' and 'stable delinquents' based on severity of delinquent behavior at ages 10 and 12–13. While delinquency at age 10 was significantly associated with poorer task performance, these differences did not differentiate youth who exhibited persistent versus more transient delinquency across time.

Antisocial Youth: Adolescence—Two studies of predominately males examined performance on a response reversal task in adolescent youth with ASB; however, it should be noted that both of these samples consisted of youth from a wide age range (Table 4). First, Shapiro et al. (1988) found that clinically referred youth with elevated CP performed significantly worse, as indexed by continued responding despite increasing punishment, than youth with low CP. A second study found an offender sample was more likely to continue responding despite increasing punishment when compared to healthy controls (Fonseca and Yule 1995).

Antisocial Youth with Psychopathic Features: Late Childhood—One study examined response reversal learning among antisocial youth with psychopathic features in late childhood (Table 4). In a sample of clinically referred youth with CD/ODD and community-recruited healthy controls, O'Brien and Frick (1996) grouped youth based on level of CU traits and the presence of an anxiety disorder. Youth with increased CU traits and no anxiety disorder demonstrated poorer performance relative to youth with CU traits and a co-occurring anxiety disorder; however, those youth with CU traits and a co-occurring anxiety disorder did not differ from healthy controls. Moreover, when non-anxious youth with CU traits were divided into those with and without CD/ODD, groups did not

significantly differ from each other, suggesting that results were not driven by co-occurring CD/ODD.

Antisocial Youth with Psychopathic Features: Adolescence—Three studies using response reversal paradigms have been conducted in adolescent youth with psychopathic features (Table 4). Frick et al. (2003) found community-recruited youth with CD who scored in the upper quartile on a measure of CU traits exhibited continued responding despite increasing punishment relative to youth with increased CD symptoms and low CU traits and youth with low levels of both CD symptoms and CU traits. Moreover, there were no differences in task performance between youth with CU traits and high versus low CD symptoms, suggesting that these deficits are uniquely associated with the affective features of psychopathy. Fisher and Blair (1998) found youth characterized by high psychopathic features had poorer performance relative to youth with low psychopathic features. Utilizing a larger sample of participants, total and subscale (i.e., CU, impulsive/CD) scores were examined continuously and all measures of psychopathic features were significantly associated with poorer performance. However, in contrast to Frick et al. (2003), after accounting for overlap between subscales, only associations between performance and impulsive/CD problems remained significant. Lastly, Budhani and Blair (2005) used a variation of the response reversal paradigm to examine these deficits more closely. In this task, contingencies of reward punishment ranged from 100–0 to 70–30. Once participants learned which stimulus was rewarded, the contingency was reversed. Youth with increased psychopathic features had more errors following contingency reversal. In addition, a greater percentage of youth characterized by high psychopathic features failed to *initially* learn which stimulus resulted most often in reward and this was specific to the 70–30 contingency condition. This suggests that youth with psychopathic features not only have deficits modulating responses to contingency change but also display difficulties in initial encoding when reinforcement/punishment is more inconsistent.

Summary—With one exception, empirical work examining response reversal learning consistently found youth with ASB to show marked difficulty inhibiting a prepotent reward response in the face of increasing punishment. This was replicated in samples of youth in late childhood and adolescence from a variety of community (Daughtery and Quay 1991), clinical (Fonseca and Yule 1995; Matthys et al. 2004; Matthys et al. 1998; O'Brien and Frick 1996) and offender samples (Fonseca and Yule 1995; O'Brien et al. 1994). In addition, antisocial youth with psychopathic features showed the most pronounced deficits on response reversal tasks (Budhani and Blair 2005; Fisher and Blair 1998), with some evidence that CU traits in particular may be driving this association (Frick et al. 2003; O'Brien and Frick 1996). Along these lines, one study found that, irrespective of CD/ODD, the presence CU traits was indicative of difficulties inhibiting a previously rewarded response despite escalating punishment (O'Brien and Frick 1996). Moreover, there was further support for the notion that processing deficits are linked to decreased anxiety (O'Brien and Frick 1996; O'Brien et al. 1994). As summarized above, the presence of anxiety is believed to be indicative of increased responsivity to punishment cues (Gray 1987); conversely, the absence of anxiety among antisocial youth reflects insensitivity to punishment and potential difficulties inhibiting reward-focused action. In line with this

theory, reduced anxiety appears to be an important marker among antisocial youth, particularly those with psychopathic features.

Of note, the one study that produced null findings utilized a longitudinal design. Consistent with theory suggesting that early engagement of ASB is reflective of neurocognitive abnormalities (Moffitt 1993), significant performance differences were associated with increased frequency of delinquency engagement at age 10. However, these abnormalities were unrelated to the persistence of delinquency, which may reflect heterogeneity in the etiology of these behaviors even at an early age.

Overall Summary

Primary Deficit in Reward Processing

Results show a higher propensity for reward-seeking behavior among adolescents with ASB (Fairchild et al. 2009; Miura 2009; Schutter et al. 2011); however, this strong affinity for immediate reward despite a high probability of punishment was not seen in late childhood (MacPherson et al. 2010). While research suggests aberrant reward processing in adolescents with ASB, future research is needed to address several important issues. First, it remains unclear whether the observed increase in reward-seeking behaviors is explained by a primary dysfunction in reward processing. The majority of tasks reviewed here incorporate aspects of *both* reward and punishment, precluding firm conclusions about underlying mechanisms. Moreover, those studies that focused predominantly on reward (i.e., BART, PALR: reward only) failed to find performance deficits in antisocial youth (Castellanos-Ryan et al. 2011; MacPherson et al. 2010). Thus, future work should investigate reward processing among youth with ASB, with careful consideration of task selection and experimental design to ensure successful isolation and investigation of reward responsivity. Second, a more in depth examination of reward processing in children is warranted. Given differences in findings based on developmental age and the lack of control groups in some positive studies, it is possible that these deficits reflect more normative increases in risk-taking and reward-seeking behaviors during adolescence.

Primary Deficit in Punishment Processing

Empirical work consistently found antisocial youth to show punishment insensitivity in both late childhood and adolescence. Specifically, youth with ASB showed reduced autonomic responsivity to uncued punishment (e.g., Fairchild et al. 2008; Herpertz et al. 2001) and cues of impending punishment (e.g., Loeber et al. 2007; Raine et al. 1996), suggesting a relative lack of arousal to punishment that impedes the development of conditioned associations. This is in line with theories suggesting that a reduction in reflexive emotional responsivity to punishment or distress inhibits the ability to establish conditioned associations and increases risk for engagement in ASB (Blair 2004; Kochanska 1994). Findings were specific to primary punishment (i.e., aversive tones) and thus congruent with classical conditioning learning models. While limited in number, studies that examined other forms of punishment (i.e., loss of money) found no differences in responsivity (Castellanos-Ryan et al. 2011; Hartung et al. 2002). As such, future research may seek to further investigate potential deficits in processing various forms of punishment as these differences may have important

treatment implications. Lastly, while some longitudinal work indicates that punishment insensitivity may help to explain persistent ASB (Raine et al. 1996), other studies did not find these deficits to prospectively distinguish desisting and persisting adolescent offenders (Loeber et al. 2007). As such, more emphasis on prospective longitudinal designs that assess potential changes in processing as well as whether these deficiencies help to inform our understanding of divergent trajectories of ASB (i.e., desisting versus persisting) is needed.

Deficits in Reward and Punishment Processing

Antisocial youth consistently show performance deficits in the face of competing reward and punishment, and this is evident across a number of different tasks (e.g., PALR, response reversal, IGT, RCT). Results were most reliable and thoroughly investigated with regard to response reversal learning, which highlights difficulties inhibiting a dominant response to reward despite increasing punishment and trouble responding to changing contingencies (Fonseca and Yule 1995, Matthys et al. 1998, 2004). Moreover, several studies found the presence of heightened anxiety to be a marker of better performance (Daughtery and Quay 1991), and in some instances, anxiety served to differentiate subgroups of antisocial youth, such that reduced anxiety characterized those with the poorest performance (O'Brien and Frick 1996; O'Brien et al. 1994). In light of consistent deficits on tasks that examine punishment sensitivity, it may be that deficient punishment processing is driving performance differences on these tasks. However, it is also possible that youth with ASB evidence greater difficulty when cognitive demands are high, and they are required to shift attention to less salient cues of punishment in the face of dominant reward-focused action, consistent with Newman's response modulation hypothesis (Patterson and Newman 1993). As future research continues to investigate these processes, it will be important to delineate whether performance deficits are specific to punishment insensitivity, difficulties modulating attention in the presence of competing reward/punishment or a combination of the two.

Reward and Punishment Processing in Antisocial Youth with Psychopathic Features

Research examining reward and punishment processing in youth with psychopathic features is relatively limited and somewhat inconsistent. While antisocial youth with psychopathic features (broadly defined) have been shown to exhibit increased risk-taking, indicative of a reward-dominant response style, other studies have found that the core affective features of psychopathy (i.e., CU traits) are not associated with primary deficits in reward processing (Centifanti and Modecki 2013; Marini and Stickle 2010). Moreover, studies examining reward processing in psychopathic youth with reduced anxiety also failed to show significant deficits when comparing performance to psychopathic youth with heightened anxiety (Newman and Kosson 1986). This suggests that psychopathic youth characterized by reduced anxiety and core psychopathic features (i.e., CU traits) may show punishment insensitivity and hypoactive BIS reactivity, rather than dysfunction in the BAS. However, research examining a primary deficit in punishment processing failed to show deficiencies specific to youth psychopathic features (broadly defined). This area of research includes only one study, and null findings are arguably related to a failure to account for heightened anxiety and/or the presence of CU traits. Along these lines, studies that investigated deficits in the presence of both reward and punishment processing consistently found antisocial youth with psychopathic features to show the poorest performance (e.g., Newman and

Kosson 1986). These deficits were uniquely associated with the affective features of psychopathy (i.e., CU traits; Frick et al. 2003) and even more specifically, the absence of high anxiety (O'Brien and Frick 1996; Vitale et al. 2005). However, studies investigating primary deficits in reward or punishment processing remain rare and often failed to delineate between variants of psychopathy. As such, future research examining aberrant reward and punishment processing in youth exhibiting antisocial behavior should seek to account for levels of anxiety and examine associations with dimensions of psychopathy that may be differentially related to differential BIS/BAS functioning.

Limitations

Taken as a whole, it appears that youth with ASB exhibit abnormalities in reward and punishment processing, with some suggestion that deficits may be more pronounced in antisocial youth with core affective psychopathic features and reduced levels of anxiety. While the behavioral literature has increased our understanding of associations between reward/punishment processing and ASB, there are several limitations. First, these studies are limited in their ability to completely disentangle the mechanisms underlying reward and punishment processing. For example, while behavioral tasks may identify a reward-dominant response style, it is difficult to determine whether hyper-active or hypoactive reactivity to cue or receipt of reward underlies this pattern of behavior. In addition, many of the behavioral paradigms discussed above incorporate aspects of both reward and punishment, making it difficult to discern unique association between deficits in these systems and ASB. Moreover, behavioral studies rarely acknowledge the complexities of reward and punishment processing, as each of these mechanisms are comprised of multiple phases of learning (Ernst et al. 2006), and aberrant reward/punishment processing could result from abnormalities at one or multiple phases. For example, abnormalities may exist during (1) encoding, defined as the initial processing of a stimulus; (2) acquisition, the process of associative learning that occurs with the repeated pairing of two stimuli or stimuli and response; or (3) extinction, the removal of an expected stimulus (Balsam et al. 2010). A failure to evaluate these mechanisms as nuanced processes drastically limits our understanding of their unique contributions to the etiology of ASB.

Recent advances in neuroscience have the potential to address several of these limitations. Specifically, researchers have identified neural circuitries associated with reward and punishment processing and have linked specific neural substrates to distinct phases of processing. An overview of these circuitries follows along with a review of emerging neuroimaging studies examining reward/punishment processing in youth with ASB.

Reward and Punishment Processing: Neural Circuitries

In his original theory, Gray (1981, 1987) attempted to delineate two distinct sets of neurobiological substrates that subserve reward (BAS) and punishment (BIS) processing. Building upon this theory and decades of research on animal models, emerging neuroimaging work has helped to refine and enhance our understanding of reward and punishment processing systems in the human brain. While a comprehensive examination of the complex circuitry is beyond the scope of this review, there are various subcortical and

cortical regions that have been consistently implicated as significant components of these multifaceted networks. As such, a parsimonious overview of these key nodes will be discussed in terms of three neural systems, mirroring the triadic model (Ernst et al. 2006): (1) reward/approach; (2) punishment/avoidance; and (3) regulatory. Although these circuits are discussed here in isolation, it is important to note that there is considerable overlap between systems as well as extensive functional connections that include both direct and indirect projections (Carmichael and Price 1995). In addition, other neurobiological systems, such as dopaminergic and serotonergic neurotransmitter systems, are intricately associated with the neural circuitry discussed below. Specifically, regions associated with reward processing are rich in dopamine, and research indicates that the release of dopamine is directly related to reinforcement learning (Schultz 2002, 2007). Serotonin is heavily implicated in punishment/avoidance circuitry, and empirical work suggests that serotonin functioning is associated with reactivity to perceived aversive stimuli and punishment sensitivity. Due to the limited scope of the current review, this literature is not reviewed here; for a more thorough discussion of these neurotransmitters, broader functioning and associations with ASB see Matthys et al. (2012a, b).

Reward/Approach System

Extensive research on the reward system has implicated regions of the basal ganglia, namely the striatum, in addition to higher-order cortical areas included within the frontal cortex (discussed below). The striatum is comprised of two anatomically and functionally distinct regions: the ventral and dorsal striatum (VS and DS, respectively; Delgado 2007). The VS contains the nucleus accumbens (NAcc) and receives input from a variety of regions including the ventral tegmental area (VTA), a region implicated in the initial detection or encoding of a reward stimulus (Fields et al. 2007) and the amygdala, an area associated with the emotional valence of reward (Cardinal et al. 2002). Given these afferent connections, it is not surprising that the NAcc has been shown to play an important role in the anticipation or prediction of reward (Knutson and Cooper 2005) and affective aspects of reward processing (Cardinal et al. 2002). The DS contains the caudate and putamen, two areas that have been consistently linked to learning reward-response contingencies (Balleine et al. 2007). Research has shown these regions to be involved in both the acquisition and extinction phases of learning. Specifically, the putamen plays an important role in the coding of reward-action associations, while the caudate is engaged in reward-prediction errors or the comparison of actual versus predicted rewards (O'Doherty et al. 2004).

Punishment/Avoidance System

Decades of empirical work have identified the amygdala as central to punishment processing, namely aversive conditioning (LeDoux 2003). The amygdala can be divided into two anatomically and functionally distinct components, including the basolateral complex and the central nucleus (Swanson 2003). In serial processing models, the basolateral complex is linked to processing sensory and contextual CS and has been shown to be particularly important in the acquisition of US–CS associations (Balleine and Killcross 2006; LeDoux 2003). This area has reciprocal projections with various regions of the prefrontal cortex, facilitating its influence on complex behaviors, and extensive connections with the VS and central nucleus (Everitt et al. 2003). Propagation of this information to the

central nucleus serves to mediate the CR through projections to the hypothalamus, midbrain reticular formation and brainstem, areas that are associated with behavioral and autonomic responses (Kapp et al. 1992). It is also important to note that the amygdala has been implicated in instrumental or operant conditioning, with the basolateral complex and the central nucleus playing important roles in the emotional valence and value of stimuli, thus influencing motivation (Balleine et al. 2007; Cardinal et al. 2002). Long-standing theories on ASB and psychopathy have proposed that amygdala dysfunction (and impaired communication with higher-order regulatory regions) underlies the development and persistence of ASB (Blair et al. 2005). More recently, the differential amygdala activation model (DAAM) suggests that the basolateral complex is underactive in individuals with ASB and psychopathy and results in a failure to process important information related to the value of stimulus, leading to an inability to form US–CS associations (Moul et al. 2012).

Regulatory System

Several regions of the prefrontal cortex have been implicated in reward and punishment processing, namely the orbital frontal cortex (OFC), ventral medial prefrontal cortex (vmPFC) and the anterior cingulate cortex (ACC). These regions are noted for their involvement in higher-order processing and are extensively enervated by direct and indirect projections from subcortical regions linked to reward (i.e., striatum) and punishment (i.e., amygdala) circuitries (Cardinal et al. 2002; Rogers et al. 2004). The OFC has been implicated in outcome evaluation, pattern detection and decision making and functions to shape behavior according to the estimated value of actions associated with reward and punishment contingencies (Cardinal et al. 2002). Medial and lateral regions of the OFC respond to various primary and secondary rewards and punishments, respectively, and have been implicated in the representational value or magnitude of reward/punishment (Elliott et al. 2000). Like the OFC, the vmPFC is involved in outcome evaluation, pattern detection and decision making (Clark et al. 2004). Moreover, given extensive connections with the amygdala, the vmPFC is thought to play an integral role in emotional learning (Damasio 1994). Lastly, the ACC has been linked to error monitoring, with evidence suggesting that it not only detects and monitors errors but also serves to initiate action in response to error detection via connections with the motor system (Rogers et al. 2004). The rostral-ventral portions of the ACC in particular have been linked to reinforcement and emotional learning and reward preference, with strong connections to the OFC, vmPFC and striatum (Öngür and Price 2000). Various theories on ASB have implicated these regions in conceptualizations of ASB, suggesting that a dysfunction in these regulatory mechanisms results in heightened levels of ASB (e.g., Blair 2004; Damasio 1994).

Neuroimaging Research

While various techniques have been used to examine the underlying neurobiology of reward and punishment responsivity, the focus of the current review is fMRI due to its ability to evaluate neural activation during reward and punishment processing. This technique noninvasively quantifies changes in blood-oxygenation-level-dependent (BOLD) response, an assessment of the ratio between oxygenated and deoxygenated blood in the brain that is thought to reflect neural activation. Functional MRI is based on the assumption that

increases in neuronal activity in a particular area of the brain lead to increases in cerebral blood flow in the same brain areas. Thus, task-specific changes in neural activity can be identified with relatively good temporal and spatial resolution thereby linking the components of a given task to regions of the brain in order to localize specific functions (Buxton 2002; Toga and Mazziotta 2002).

As was evident in the behavioral literature, emerging fMRI research in antisocial youth uses a variety of different paradigms to assess reward and punishment processing. These tasks are briefly described below and fMRI studies are evaluated in (a) antisocial youth and (b) antisocial youth with psychopathic features.

Antisocial Youth: Adolescence

Three studies examined the neural processing of reward and punishment in adolescent samples, though two of these studies had wide age ranges and also included some youth in late childhood (see Table 5). First, Rubia et al. (2009) utilized a rewarded continuous performance task to examine differences between responsivity to receipt of reward versus non-reward among males with early-onset CD, ADHD and healthy controls. When comparing activation to reward versus non-reward, youth with ASB showed less activation to receipt of reward in the lateral and medial OFC relative to youth with ADHD and healthy controls. In a second study, youth completed a monetary incentive task and were rewarded during every other block of trials (i.e., every 10 trials). No differences were seen in response to receipt of reward. However, during trials of non-reward, males with CD and ADHD showed reduced activation in the ACC while simultaneously showing increased activation in the striatum relative to healthy controls (Gatzke-Kopp et al. 2009). Differences in the neural response were specific to omission of expected reward (i.e., error monitoring, extinction), suggesting ASB may be linked to a failure to process changes in reward contingencies. Lastly, a community sample of predominately males completed a similar monetary incentive task (Bjork et al. 2010). When comparing rewarded responses to non-rewarded responses, youth with ASB showed greater activation to receipt of reward in the striatum and vmPFC relative to healthy controls.

Antisocial Youth with Psychopathic Features: Adolescence

Two studies examined reward and punishment processing in subgroups of antisocial adolescents with increased psychopathic features, and both of these studies examined samples of predominately adolescent males (see Table 5). Finger et al. (2008) examined the BOLD response in youth with CD/ODD and psychopathic features, youth with ADHD, and healthy controls during a response reversal task. Youth with CD/ODD and psychopathic features demonstrated increased activation in the caudate and vmPFC to punished errors, though no differences were seen in responsivity to reward. This increase in activation was also associated with a continuous measure of CU traits. Somewhat contrary to these findings, a later study by Finger et al. (2011) used a passive avoidance task and found CD/ODD youth with psychopathic features to demonstrate reduced responsivity in the OFC to rewarded trials relative to healthy controls, with no notable differences in response to the absence of expected reward (i.e., error monitoring, extinction) or in response to punished errors. The authors also investigated differences in processing during early versus late trials

and found youth with ASB and psychopathic features showed decreased activation in the caudate and OFC in early versus late trials relative to healthy controls, which can be interpreted as potential deficits in acquisition. Lastly, there was a main effect of group, such that across all trials, youth with ASB and psychopathic features showed decreased activation in the prefrontal cortex, caudate and amygdala relative to healthy controls.

Summary

Among antisocial youth, aberrant processing was consistently found in areas associated with reward and regulatory function and less frequently shown in regions implicated in punishment processing (i.e., amygdala), which may reflect the type of task and nature of the punishment administered (i.e., failure to receive reward versus presentation of an aversive stimulus). While some studies provide support for reduced neural reactivity to reward in antisocial youth (Rubia et al. 2009), others suggest the youth with ASB may experience increased neural reactivity to reward (Bjork et al. 2010) as evidenced by increased activation in the striatum and OFC. At the same time, there is some indication that ASB may also be associated with dysfunctional neural processing of contingency changes, particularly those involving a failure to receive an expected reward (Gatzke-Kopp et al. 2009). This involves higher-order regulatory areas (i.e., OFC, ACC) that play a role in outcome evaluation, representational value of reward/punishment and error monitoring (Elliott et al. 2000).

Studies examining these processes in youth with psychopathic features also produced mixed findings. A more recent study by Finger et al. (2011) found youth with ASB to evidence decreased activation across trials in prefrontal regulatory regions and subcortical regions including the caudate and amygdala. While one study found that antisocial youth with psychopathic features showed decreased activation across all trials and specifically a reduced responsivity to receipt of reward during a passive avoidance task (Finger et al. 2011), another found no differences in reward sensitivity during a response reversal task (Finger et al. 2008). Instead, the latter study found increased activation to punished response reversal errors (i.e., failure to receive an expected reward), suggesting deficits specific to error monitoring and difficulties processing contingency change. Discrepancies between studies may be task specific as passive avoidance paradigms do not involve contingency change, and thus, the meaning of punished errors differs across tasks. Regardless, it is important to note that, contrary to behavioral studies that evaluated antisocial youth with high versus low psychopathic features, these studies *only* examined antisocial youth with high psychopathic features in comparison with healthy controls. They did not include non-psychopathic antisocial youth, and thus, conclusions about the extent to which these findings are specific to or most pronounced in the presence of psychopathic features are limited. Moreover, the studies reviewed here focused on psychopathic features (broadly defined). In light of behavioral work demonstrating that core psychopathic features (i.e., CU traits) and the absence of high anxiety may be associated with the most pronounced deficits, future work may aim to stratify groups based on these characteristics.

Discussion

As summarized above, behavioral research examining a primary deficit in reward processing in youth with ASB is inconclusive, though studies investigating this hypothesis were notably limited. At the same time, neuroimaging research suggests that youth with ASB may be characterized by dysfunctional reward processing; however, it is unclear whether this is best conceptualized as a hyper-versus hypo-reactivity to reward, as preliminary studies have produced conflicting results. Nonetheless, these findings suggest that aberrant reward processing may go undetected in laboratory-based behavioral studies investigating reward responsivity and highlights the need for continued neuroimaging work in this area. Neuroimaging studies demonstrate abnormal processing in the amygdala, striatum and high-order regulatory regions like the OFC and vmPFC, consistent with postulations by Blair (2004). More recently, research in this area has examined indices of dopaminergic functioning in antisocial youth, and genetic studies have shown associations between variation in the gene encoding for the dopamine D4 receptor (DRD4) and aggressive behaviors and conduct problems as early as age 4 (Holmes et al. 2002; Schmidt et al. 2002). Other work has linked ASB and variation in catecholomine-O-methyltranferase (COMT) and monoamine oxidase A (MAOA), both of which are associated with regulating levels of catecholamines-like dopamine in the brain (Matthys et al. 2012a). Taken together, this provides strong support for continued research into the mechanisms underlying deficits in reward processing and reinforcement learning seen in youth with ASB.

Research examining punishment processing in ASB youth is more definitive, with behavioral studies finding youth with ASB to have difficulty encoding unexpected punishment and acquiring cue-stimulus associations. This was evident in late childhood and adolescence, and recent work suggests that these deficits are present as early as 3 years of age and serve to predict criminal offending in adulthood (Gao et al. 2010). Consistent with Blair (2004), this provides evidence for inherent deficits in emotional arousal to punishment that hinders the development of conditioned associations between cues of impending punishment and distress. This has been linked to amygdala dysfunction, and there is growing research documenting abnormal amygdala activity in antisocial youth, particularly those with heightened psychopathic features, during passive avoidance learning (Finger et al. 2011) and emotion processing tasks (e.g., Jones et al. 2009; Marsh et al. 2008). In addition, studies have shown differences in gray matter volume of the amygdala among youth with ASB (De Brito et al. 2009; Huebner et al. 2008; Sterzer et al. 2007) and adults with a history of early-onset ASB and psychopathic features (Pardini et al. 2013).

Moul et al. (2012) offer a more nuanced theory on amygdala dysfunction and the deficits in aversive conditioning seen in antisocial and psychopathic populations. Specifically, this model posits underactivity that is specific to the basolateral amygdala, resulting in problems with the modulation of attention to important features of the cue stimulus as opposed to dysfunction in the central amygdala that is linked to responsivity to the aversive stimulus. This is more consistent with Newman's response modulation hypothesis (1993) and focuses on an inability to allocate attention to less salient cues in the environment. This theory draws support from the adult literature and suggests that psychopathic individuals maintain normative autonomic responses to unconditioned aversive stimuli (e.g., Flor et al. 2002).

Moreover, recent studies demonstrate that directed attention to cues of impending punishment can mitigate diminished conditioned responses (Newman et al. 2010). However, data from the current review suggests that in addition to aberrant responsivity to cues of impending punishment, antisocial youth demonstrate reduced responsivity to uncued aversive stimuli, indicative of abnormalities in central amygdala function and more consistent with Blair (2004). While this discrepancy could be explained by a failure to account for psychopathic features in the studies reviewed here, it also possible that both deficient emotional arousal and difficulties modulating attention play a role in the development of ASB, with the former developmentally preceding the latter.

Also in line with Newman and Wallace (1993) response modulation hypothesis, youth exhibiting ASB showed consistent deficits on tasks that incorporated competing reward and punishment (e.g., response reversal, passive avoidance) and these deficits were particularly pronounced among antisocial youth with core affective psychopathic features (e.g., CU traits) and reduced levels of anxiety. Neuroimaging studies using similar tasks found antisocial youth with psychopathic features to show abnormalities in the processing of punished errors (i.e., failure to receive expected reward), suggesting this may be the driving force behind behavioral differences (Finger et al. 2008). Thus, observed differences appear to be more consistent with deficits in error monitoring and difficulties processing contingency change, as evidenced by aberrant processing in the striatum and prefrontal regulatory regions (e.g., Gatzke-Kopp et al. 2009; Rubia et al. 2009). At the same time, there is some suggestion that abnormal processing is most prominent in early versus late trials, when encoding and acquisition is thought to be occurring (Finger et al. 2011), prior to an established dominant response set. This suggests that in addition to difficulties modulating attention to less salient cues of punishment in the context of a dominant reward set, there may also be deficits in the initial encoding of stimuli and learning contingencies.

Individual Differences and Contextual Considerations

In addition to delineating subgroups of antisocial youth by the presence of psychopathic features, individual differences in level of anxiety evidenced significant associations with reward and punishment processing. This is consistent with research suggesting that low levels of anxiety may distinguish a distinct etiology among psychopathic individuals (Kimonis et al. 2011, 2012; Poythress et al. 2010). Moreover, low levels of anxiety are linked to reduced BIS activation and punishment insensitivity that is believed to hinder the internalization of moral beliefs and the development of empathy and guilt (Kochanska 1994). Also noteworthy, ADHD and substance abuse have been linked to perseverative, impulsive reward-seeking behaviors, and given the high degree of comorbidity between ASB, ADHD and substance use, it may be particularly important to consider the extent to which mechanisms underlying these disorders differ (Hinshaw and Lee 2003; Krueger et al. 2002). Lastly, little attention has been given to the role of gender, though there are notable differences in the prevalence and trajectories of ASB in males and females (see Brennan and Shaw 2013). While many of the studies reviewed here included both males and females, researchers have yet to compare potential differences in the association between reward/punishment processing and ASB in gender-specific samples.

It is also important to consider the extent to which contextual influences may serve to moderate associations between aberrant processing of reward/punishment and ASB. As summarized above, harsh and inconsistent parenting is thought to inadvertently reinforce ASB (Patterson et al. 1992) and may contribute to the development of psychopathic features (Waller et al. 2013). This type of parenting combined with an insensitivity to punishment and difficulties inhibiting a reward-dominant response style may be particularly detrimental. Moreover, research suggests that parent–child interactions are bidirectional and as such may have cascading effects that serve to further entrain aberrant reward and punishment processing (Dodge and Pettit 2003; Pardini et al. 2008; Sameroff 2000). Lastly, youth with deficits in reward/punishment processing may be particularly vulnerable to deviant social influences, and emerging work reviewed here suggests differences in responsivity to reward in the presence of peers (Centifanti and Modecki 2013). As such, further investigation of these contextual factors as potential moderators of the associations between reward/punishment processing deficits and ASB may be particularly informative.

Developmental Considerations

Developmental considerations have been highlighted throughout this review. Most notable is the normative maturation of the reward and punishment processing systems. Given that the sharp increase in the prevalence and frequency of ASB in adolescence has been linked to an imbalance of early developing subcortical areas and underdeveloped regulatory frontal regions (Fareri et al. 2008; Steinberg and Morris 2001), it is essential that our understanding of abnormalities in reward/punishment processing be framed within the context of normative development. Along these lines, it is important to note that some youth do not engage in ASB while many of those who do, desist from ASB in late adolescence or early adulthood (Loeber et al. 2008). This raises several issues. First, the engagement in ASB in some youth may merely reflect normative maturational processes, and performance deficits and neurobiological differences may be, at least in part, reflective of such. At the same time, it is plausible that ASB in other youth may be related to slower maturational processes or reflective of preexisting abnormalities. This is consistent with research documenting structural differences in antisocial individuals within regions reward/punishment processing, such as the amygdala, striatum and prefrontal regulatory regions (e.g., De Brito et al. 2009; Glenn et al. 2010) and suggests that early investigations of these processes may be particularly informative. Thus, heterogeneity within trajectories of ASB may reflect slower developmental maturation, maturation gone awry, or pre-existing structural abnormalities and/or deficient functional connectivity.

Implications for Intervention

Multifaceted, multimodal intervention efforts have been shown to be particularly effective for youth with ASB (Kazdin 2010), and emerging evidence suggests that antisocial youth with core affective psychopathic features respond favorably to intervention (Waller et al. 2013). These interventions are founded in learning theory, and empirical work demonstrates the success of parent management training coupled with child-focused cognitive-behavioral therapy and problem-solving skills training (Eyberg et al. 2008; Kazdin et al. 1992; Webster-Stratton et al. 2004). These programs often teach children to become more attentive to the

positive and negative consequences of their actions and teach strategies that aim to interrupt perseverative, reward-focused action by helping youth to develop a socially appropriate plan. These types of interventions may be particularly effective for youth demonstrating deficits in reward/punishment processing. Moreover, parents are taught to engage in more consistent contingency management strategies to eliminate problem behaviors and increase prosocial responses by emphasizing reward-based strategies for reinforcing children's positive behavior while ignoring negative behaviors. Early intervention designed to teach parents to more stringently manage behavioral contingencies may be particularly useful in reducing ASB among youth with abnormalities in reward and punishment processing. However, while intervention efforts based in social learning theory demonstrate success, effect sizes are still small to moderate (see Matthys et al. 2012b). This may be related to the 'one size fits all' approach and a failure to assess and treat child-specific deficits at an individual level. Given the noted heterogeneity that exists among youth with ASB, efforts to tailor interventions to meet child-specific needs may increase the effectiveness of learning-based interventions. For example, treatment for youth with primary deficits in reward processing may focus on increasing the use of praise and reward systems that facilitate the reinforcement of positive, prosocial behaviors, while intervention for youth with punishment insensitivity may emphasize the importance of clear, consistent and proportionally relevant consequences (see Dadds and Salmon 2003).

Limitations and Future Directions

The research reviewed here furthers our understanding of reward/punishment processing as potential mechanisms underlying ASB; however, it is almost exclusively behavioral. While an examination of the underlying neurobiology has potential to further elucidate the mechanisms subserving behavioral differences, there is currently a dearth of fMRI studies in youth with ASB. Moreover, given the complexity of the learning process (i.e., encoding, acquisition, extinction) and the implicated neural circuitries, conclusions about the extent to which there are abnormalities at one or multiple phases of processing may be difficult to obtain. While intricate tasks assessing passive avoidance and response reversal learning have provided fairly consistent behavioral results, neuroimaging findings may be difficult to interpret without a strong understanding of basic reward and punishment processing at initial encoding. As such, future research may want to take a bottom-up approach, focusing first on a single phase of processing (i.e., receipt of reward or punishment) which would help lay the foundation for a more comprehensive understanding of these complex processes in antisocial youth. Second, the current literature is limited by an inconsistent evaluation of important subgroups. Less than half of the studies reviewed here examined the role of psychopathic features, and those that did varied greatly in the measurement of these features (i.e., psychopathy (broadly defined) vs. CU traits). Moreover, there was a failure to account for level of anxiety, and this likely contributed to mixed findings. Future work should aim to assess these characteristics and symptoms associated with ADHD (i.e., impulsivity, inattention) and substance use. Importantly, this work should account for high and low levels of these characteristics *within* populations of antisocial youth and always compare function to that of healthy controls.

It is also important to consider the extent to which abnormalities in reward/punishment processing are associated with persistent trajectories of ASB. While one longitudinal study found deficits in reward/punishment processing to characterize youth with persistent ASB (Raine et al. 1996), all other longitudinal investigations reviewed here failed to find predictive effects (e.g., Castellanos-Ryan et al. 2011; MacPherson et al. 2010; White et al. 1994). Though this may be related to the length of follow-up or the measure/classification of ASB, it is also possible that cross-sectional differences, while associated with concurrent engagement in ASB, tell us little about future ASB. At the same time, antisocial adults, particularly those with psychopathic features, show similar performance deficits on the same behavioral tasks reviewed here (e.g., Budhani et al. 2006; Mitchell et al. 2002; Newman et al. 1990) and emerging evidence documents abnormalities in reward/punishment circuitry that mirror findings in youth (e.g., Mitchell et al. 2006; Völlm et al. 2010), suggesting these deficits may be a stable mechanism underlying the persistence of ASB. Future research employing longitudinal, prospective designs would allow for a better understanding of stability and predictive utility as well as potential changes in these mechanisms during developmentally distinct time periods. Given the inclusion of samples with wide age ranges and a focus on adolescence, future research should aim to examine these processes using repeated assessments across childhood and the transition to adolescence. In doing so, a focus on pubertal timing as opposed to chronological age is necessary in line with research suggesting that pubertal hormones play an important role in neurobiological maturation (Steinberg 2008, 2010).

Lastly, there are likely individual differences in the severity of dysfunction within these processing systems. While the studies reviewed here focus on group level differences, it may be important to utilize person-centered approaches that examine within individual variability across time. In doing so, a more thorough examination of potential moderators is needed. Specifically, the contextual factors discussed above (i.e., parenting, peers) likely influence reward/punishment processing systems and certainly play a role in the development of ASB and psychopathic features. As such, research investigating whether these factors impact reward/punishment processing as mechanisms underlying ASB is warranted.

Summary

While there are undoubtedly myriad etiological pathways to ASB, the current review suggests that youth with ASB appear to be characterized by punishment insensitivity and significant difficulties suspending reward-seeking behaviors despite a high probability of punishment. Moreover, there is some evidence to support the notion that these deficits are most pronounced in antisocial youth with psychopathic features, specifically those characterized by reduced anxiety and the presence of core affective features (i.e., CU traits). However, at present, the mechanisms underlying these associations have yet to be fully elucidated due in large part to the complexity of learning processes and the implicated neural circuitry. Future research may seek a bottom-up approach paying particular attention to the limitations of the current studies reviewed (e.g., small sample sizes, broad age ranges, failure to differentiate between youth with ASB and variants of psychopathy). Ultimately, understanding how these mechanisms go awry and to what extent individual difference characteristics (e.g., anxiety) and contextual factors (e.g., parenting, peers) may moderate

their effects on the engagement in ASB will be particularly important for further refining interventions. Specifically, such information may aid in the modification of these processing systems over time and/or the adaption of compensatory coping strategies that serve to reduce ASB.

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Table 1

Risk-taking tasks in antisocial youth and antisocial youth with psychopathic features

Study	Sample (Race)	Age range	Male (%)	Groups	Group classification	Control variables	Task	Results
Antisocial Youth: Late Childhood								
MacPherson et al. (2010)[a]	Community 34 % African American; 14 % Latino/Other	9 & 13	53	277 youth	YRBS (C) (summed # of risk taking behaviors)	Age; Gender	BART	NS associations with risk taking
Antisocial Youth: Adolescence								
Fairchild et al. (2009)	Community 8 % Non-white	14–18	100	38 EO-CD 34 AO-CD 84 HC	K-SADS (P/C) (DSM-IV criteria)	IQ; SES; Race; Substance use	RCT	**EO-CD = AO-CD > HC: greater risk taking**
Miura (2009)	Offender 100 % Asian	*m* = 13.1	100	142 violent 167 non-violent	Official record (all met DSM-IV criteria for CD)	IQ	IGT	NS group differences in risk taking **Risk taking > chance in both groups**
Syngelaki et al. (2009)	Offender versus Community	12–18	100	103 offenders 84 HC	Official record K-SADS (P/C) (HC no diagnosis)	IQ	RCT	**Offenders > HC: greater risk taking**
Schutter et al. (2011)	Offender versus Community	15–20	75	25 CD/ODD +SD 28 CD/ODD 99 HC	DISC (C) (DSM-IV criteria)	Age; IQ; ADHD	IGT	**CD/ODD +SD > CD/ODD = HC: greater risk taking**
Psychopathic Features: Adolescence								
Blair et al. (2001)	Clinical 50 % African American	9–17	100	20 HI PSY 23 LO PSY	PSD (T) (> 25; < 20)	Age; IQ	IGT	**HI PSY > LO PSY: greater risk-taking**
Fairchild et al. (2009)	Community 7 % Non-white	14–18	100	72 CD youth (groups unknown)	YPI (C) (continuous and group: 2.5 cutoff)	IQ; SES; Race; Substance use	RCT	**PSY associated with greater risk-taking** NS group differences in risk-taking
Marini and Stickle (2010)	Offender 14 % Non-white	11–17	70	148 youth	ICU (CU traits) APSD (IMP) (P/T/C) (continuous)	Gender; Sensation seeking; Impulsivity	BART	NS associations with risk-taking **CU traits associated with *less* risk taking following reward**
Centifanti and Modecki (2013)	Community 25 % Pakistani; 11 % Non-white	16–20	48	675 youth	ICU (C) (CU traits)	Gender; Peer presence	BART	NS associations with risk-taking **CU traits associated with *less* risk taking following reward**

Significant effects are listed in bold ($p < .05$)

EO-CD early-onset conduct disorder, *AO-CD* adolescent-onset conduct disorder, *HC* healthy controls, *SD* substance dependence, *PSY* psychopathic features, *CD* conduct disorder, *DSM-IV* Diagnostic and Statistical Manual-IV, *KSADS* Kiddie-Schedule for Affective Disorders and Schizophrenia, *YRBS* Youth Risk Behavior Survey, *PSD* Psychopathy Screening Device, *YPI* Youth Psychopathic Inventory,

ICU Inventory for Callous–Unemotional Traits, *APSD* Antisocial Personality Screening Device, *CU* callous–unemotional, *IMP* impulsivity, *P* parent-report, *T* teacher-report, *C* child-report, *SES* socioeconomic status, *IGT* Iowa Gambling Task, *RCT* Risky Choice Task, *BART* Balloon Analogue Risk Task, *NS* non-significant

[a] Longitudinal study

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Table 2

Psychophysiological tasks in antisocial youth and antisocial youth with psychopathic features

Study	Sample (Race)	Age range	Male (%)	Groups	Group classification	Control variables	Measure (design)	Results
Antisocial Youth: Late Childhood								
Herpertz et al. (2001)	Clinical versus community Race unspecified	8–13	100	26 CD/ADHD 21 ADHD 21 HC	DISYPS (P) (DSM-IV criteria)		SCR (uncued) Eye-blink (uncued)	**UCS: CD/ADHD < ADHD = HC** UCS: NS group differences
van Goozen et al. (2004)	Clinical versus community Race unspecified	7–12	61	21 CD/ODD 33 HC	DISC (P) (DSM-IV criteria) CBCL (P) (continuous)	Gender; ANX/DEP	Eye-blink (uncued)	**UCS: CD/ODD < HC**
Matthys et al. (2004)	Clinical versus community Race unspecified	7–12	100	19 CD/ODD 20 HC	Measure unknown (DSM-IV criteria) CBLC (P) (continuous)		SCR (task)	**CD/ODD < HC during reward/punishment task**
Antisocial Youth: Adolescence								
Raine and Venables (1981)	Community Race unspecified	14–16	100	101 youth	SR measures (C) BPC (T) (continuous)	SES	SCR (cued)	**CS: Higher ASB associated with lower SCR** UCS: NS association with ASB
Raine et al. (1996)[a]	Community Race unspecified	14–16 & 29	100	17 persisters 17 desisters 17 non-del.	SR measures (C) BPC (T) Official record (conviction)	Age; SES; Neighborhood Academic functioning	SCR (cued)	**CS: Persisters < desisters** UCS: NS group differences
Beauchaine et al. (2001)	Community Race unspecified	12–17	100	20 CD/ADHD 17ADHD 22 HC	ASI (P) (DSM-IV criteria) CBCL (P) (≥95th percentile)		SCR (task)	NS group differences following removal of reward
Loeber et al. (2007)[a]	Community 57 % African American	7–20	100	Persisters Desisters Non-del.	SRD (C) YASR (C) (moderate/serious delinquency)		SCR (cued)	**CS: Persiters = Desisters < Non-Del.** UCS: NS group differences
Fairchild et al. (2008)	Clinical versus community 3 % African American; 8 % Mixed/Other	14–18	100	43 EO-CD 28 AO-CD 54 HC	KSAD-S (P/C) (DSM-IV criteria)	Age; IQ; SES; ADHD	SCR (cued) Eye-blink (uncued)	**CS: EO-CD = AO-CD < HC** UCS: NS group differences UCS: EO-CD = AO-CD < HC
Syngelaki et al. (2013)	Offender versus community Race unspecified	12–18	100	40 Offenders 40 HC	Official record	Age; IQ	SCR (cued)	**CS: Offender < HC** UCS: NS group differences
Psychopathic Features: Adolescence								
Fung et al. (2005)	Community 57 % African American	16	100	65 HI PSY 65 LO PSY 35 PSY/DEL	CPS (C) (20th & 80th percentiles) CPS (C)	IQ; SES; ADHD	SCR (cued) SCR (cued)	**CS: HI PSY < LO PSY** **UCS: HI PSY < LO PSY**

Study	Sample (Race)	Age range	Male (%)	Groups	Group classification	Control variables	Measure (design)	Results
				16 NONPSY/DEL	SRD (C) CBCL (P/T)			CS: NS group differences UCS: NS group differences

Significant effects are listed in bold ($p < .05$)

CD conduct disorder, *EO-CD* early-onset conduct disorder, *AO-CD* adolescent-onset conduct disorder, *ODD* oppositional defiant disorder, *ADHD* attention deficit hyperactivity disorder, *HC* healthy controls, *PSY* psychopathic features, *DSM-IV* Diagnostic and Statistical Manual-IV, *ASI* Adolescent Symptom Inventory, *CBCL* child behavior checklist, *DISYPS* Diagnostic System for Mental Disorders in Children and Adolescents, *DISC* Diagnostic Interview Schedule for Children, *KSADS* Kiddie-Schedule for Affective Disorders and Schizophrenia, *SR* Self-Report, *CPS* Child Psychopathy Scale, *P* parent-report, *C* child-report, *T* teacher-report, *ANX* anxiety, *DEP* depression, *SES* socioeconomic status, *SCR* skin conductance response, *NS* non-significant

[a]Longitudinal study

Table 3

Passive avoidance tasks in antisocial youth and antisocial youth with psychopathic features

Study	Sample (Race)	Age range	Male (%)	Groups	Group classification	Control variables	Design	Results
Antisocial Youth: Adolescence								
Hartung et al. (2002)	Clinical/community 90 % Caucasian	13–18	53	172 youth	DSM-IV DBD Checklist (P/C) (continuous)	IQ; Reading level; Parental education	Mixed; Punishment only	**CD associated with passive avoidance errors** NS association with passive avoidance
Castellanos-Ryan et al. (2011)[a]	Community 38 % African American; 15 % South Asian; 13 % Mixed/ Other	14–16	45	76 youth	CD sx from multiple measures (C) (continuous)		Mixed; Reward only; Punishment only	NS association with passive avoidance errors
Psychopathic Features: Adolescence								
Newman and Kosson (1986)	Clinical 100 % Caucasian	14–18	100	11 HI PSY/LO ANX 34 HI PSY./HI ANX 31 LO PSY/LO ANX	Psychopathic Deviate Scale (C) Welsh Anxiety Scale (C) (median split)		Mixed; Reward only	**HI PSY/LO ANX > HI PSY/HI ANX = LO PSY/LO ANX: passive avoidance errors** NS group differences in passive avoidance
Scerbo et al. (1990)	Offender 55 % African American 22 % Hispanic	13–18	100	24 HI PSY 16 LO PSY	RPBC/SRD IMP/SRP (C) (group unknown)		Mixed	NS group differences in passive avoidance **HI PSY < LO PSY: commission errors**
Vitale et al. (2005)	Community 83 % Caucasian	16	100	25 HI PSY/LO ANX 51 HI PSY/HI ANX 30 LO PSY/HI ANX 54 LO PSY/LO ANX	APSD (C) Welch Anxiety Scale (C) (median split)		Mixed	**HI PYS/LO ANX > LO PSY/LO ANX: passive avoidance errors**

Significant effects are listed in bold ($p < .05$)

ANX anxiety, *HI* high, *LO* low, *PSY* psychopathic features, *CD* conduct disorder, *sx* symptoms, *RBPC* Revised Behavior Problem Checklist, *SRD* Self-Report of Delinquency, *DSM-IV* Diagnostic and Statistical Manual-IV, *IMP* impulsivity, *SRP* Self-Report of Psychopathy, *APSD* Antisocial Personality Screening Device, *P* parent-report, *C* child-report, *NS* non-significant

[a] Longitudinal Study

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Table 4

Response reversal tasks in antisocial youth and antisocial youth with psychopathic features

Study	Sample (Race)	Age range	Male (%)	Groups	Group classification	Control variables	Results
Antisocial Youth: Late Childhood							
Daughtery and Quay (1991)	Community 66 % Hispanic; 8 % African American	8–12	50	10 CP 10 CP/ATT 9 ATT 9 ANX 14 NC	RBPC (T) (1 SD above mean)	Attention problems; Anxiety	**CP = CP/ATT < ANX = NC: poorer response reversal**
O'Brien et al. (1994)	Clinical/offender versus community 42 % African American	9–13	100	21 BD 22 HC	DBD scale (P/T) (DSM-III-R criteria) CBCL (P/T) (2 SD above mean)	ADHD; Anxiety	**BD < HC: poorer response reversal ONLY AFTER accounting for anxiety symptoms**
White et al. (1994)[a]	Community 54 % African American	10 & 13	100	71 non-delin. 314 delin. 25 stable delin.	SRA (C) SRD (C) (frequency/seriousness)		NS group differences Age 10, higher frequency of delinquency associated with poor response reversal
Fonseca and Yule (1995)	Clinical versus community Race unspecified	7–11	100	27 CD 26 HC	ICD-9 (DSM-III-R criteria) Rutter Questionnaire (T) (cutoff score)	Age; IQ	**CD < HC: poorer response reversal**
O'Brien and Frick (1996)	Clinical versus community 73 % Caucasian	6–13	100	9 CD/ODD 25 CD/ODD/ANX 18 ADHD 40 HC	DISC (C/P/T) (DSM-III-R criteria)	ADHD sx	**CD/ODD < ALL: poorer response reversal**
Matthys et al. (1998)	Clinical versus community Race unspecified	8–11	100	11 CD 10 CD/ADHD 31 HC	CBCL (P)		**CD/ADHD < CD < HC: poorer response reversal**
Matthys et al. (2004)	Clinical versus community Race unspecified	7–12	100	19 CD/ODD 20 HC	Measure unknown (DSM-IV criteria) CBCL (P) (continuous)		**CD/ODD < HC: poorer response reversal** **CD/ODD < HC: RT after punishment** CD/ODD = HC: RT after reward
Antisocial Youth: Adolescence							
Fonseca and Yule (1995)	Offender versus community Race unspecified	11–15	100	44 Delin 20 HC	Official Record	Age; IQ	**Delin < HC: poorer response reversal**
Shapiro et al. (1988)	Clinical 29 % African American	7–18	75	9 HI CP 10 LO CP	RBPC (T) (*T* score ≥ 55)	Attention problems	**HI CP < LO CP: poorer response reversal**
Psychopathic Features: Late Childhood							
O'Brien and Frick (1996)	Clinical versus community 73 % Caucasian	6–13	100	29 HI CU/LO ANX 37 HI CU/HI ANX 46 LO CU/LO ANX	PSD: CU (P/T) (1 SD above mean) DISC (P/T/C) (DSM-III-R criteria)	ADHD sx	**HI CU/LO ANX < ALL: poorer response reversal**

Study	Sample (Race)	Age range	Male (%)	Groups	Group classification	Control variables	Results
Psychopathic Features: Adolescence							
Fisher and Blair (1998)	Clinical Race unspecified	11–15	100	8 HI PSY 9 LO PSY	PSD (T) (highest & lowest scorers)		**HI PSY < LO PSY: poorer response reversal**
		9–16	100	39 youth	PSD (T): CU & I/CP (continuous)	IQ	**PSY associated with poorer response reversal**
Frick et al. (2003)	Community 21 % minority	10–14	53	25 HI CD/HI CU 23 HI CD/LO CU 25 LO CD/HI CU 25 LO CD/LO CU	CSI; DISC (P/T) (DSM-IV symptoms) APSD: CU (P/T) (upper quartile/ below mean)		**HI CU < LO CU: poorer response reversal**
Budhani and Blair (2005)	Clinical 7 % African American	9–17	100	19 HI PSY 22 LO PSY	APSD (≥ 25 vs. ≤ 15)	ADHD	**HI PSY < LO PSY: poorer response reversal**

Significant effects are listed in bold ($p < .05$)

CP conduct problems, *ATT* attention problems, *ANX* anxiety, *HC* normal control, *BD* behavior disorder, *Delin* delinquent, *CD* conduct disorder, *ODD* oppositional defiant disorder, *ADHD* attention deficit hyperactivity disorder, *HI* high, *LO* low, *PSY* psychopathic features, *RBPC* Revised Behavior Problem Checklist, *SD* standard deviation, *DBD* disruptive behavior disorder, *DSM-III-R* Diagnostic and Statistical Manual-III-Revised, *CBCL* Child Behavior Checklist, *SRA* Self-Report of Antisocial Behavior, *SRD* Self-Report of Delinquency, *DSM-IV* Diagnostic and Statistical Manual-IV, *KSADS* Kiddie-Schedule for Affective Disorders and Schizophrenia, *YRBS* Youth Risk Behavior Survey, *ICD-9* International Classification of Disease-9, *DISC* Diagnostic Interview Schedule for Children, *PSD* Psychopathy Screening Device, *CSI* Child Symptom Inventory, *APSD* Antisocial Personality Screening Device, *P* parent-report, *T* teacher-report, *C* child-report, *sx* symptoms, *NS* non-significant

[a]Longitudinal study

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Table 5

Neuroimaging studies of reward/punishment processing in antisocial youth and antisocial youth with psychopathic features

Study	Sample (Race)	Age range	Male (%)	Groups	Group classification	Control variables	Task (design)	Contrast	Results	MNI Coordinates (*x*, *y*, *z*)
Antisocial Youth: Adolescence										
Rubia et al. (2009)	Clinical/community Race unspecified	9–16	100	14 EO-CD 18 ADHD 16 HC	MDSI (DSM-IV criteria) SDQ (threshold)	Age; IQ	Reward CPT (event)	Reward versus non-reward	**EO-CD < ADHD = HC:** R lateral and medial OFC (BA 47)	**35, 41, −11 (R)**
Gatzke-Kopp et al. (2009)	Clinical/community 21 % Non-white	*m* = 13.3	100	19 CD/ADHD 11 HC	ASI (DSM-IV criteria) CBC-L (98th percentile)	CD sx	Monetary incentive (block)	Reward versus fixation	CD/ADHD = HC:	
								Non-reward versus fixation	R/L striatum	
									CD/ADHD < HC:	
									R/L ACC	**6, 36, 26 (R)** **−8, 40, 18 (L)**
									CD/ADHD > HC:	
									R/L caudate	**14, 22, 0 (R)** **−10, 2, 8 (L)**
									R/L putamen	**30, 16, 2 (R)** **−20, 20, 2 (L)**
Bjork et al. (2010)	Community Race unspecified	13–17	75	12 DBD 12 HC	DISC (DSM-IV criteria) CBC-L (continuous)	Age	Monetary incentive (event)	Reward versus non-reward	**DBD > HC:**	
									R/L mesial frontal cortex	**4, 50, −11 (R)** **4, 39, −4 (L)**
									R/L NAcc	**13, 15, −7 (R)** **−10, 14, −7 (L)**
Psychopathic Features: Adolescence										
Finger et al. (2008)	Clinical/community Race unspecified	10–17	66	14 PSY/CD/ODD 14 ADHD 14 HC	APSD (P) PCL-YV (total score > 20) KSADS (P/C) (DSM-IV criteria)	Age; Gender; IQ	Response reversal (event)	Punished RR errors versus rewarded responses	**PSY/CD/ODD > ADHD, HC:**	
									R/L medial frontal gyrus (BA 10)	**23, 47, 12 (R)** **−17, 48, 8 (L)**
									PSY/CD/ODD > HC:	
									R caudate	**25, −25, 27 (R)**

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Study	Sample (Race)	Age range	Male (%)	Groups	Group classification	Control variables	Task (design)	Contrast	Results	MNI Coordinates (*x*, *y*, *z*)
Finger et al. (2011)	Clinical/community Race unspecified	*m* = 13.7	60	15 PSY/CD/ODD 15 HC	APSD (P) PCL-YV (total score > 20) KSADS (P/C) (DSM-IV criteria)	Age; IQ	Passive avoidance (event)	All trials	**PSY/CD/ODD < HC:**	
									R medial frontal gyrus (BA 10)	**11, 27, 43 (R)**
									L middle frontal gyrus (BA 8)	**−35, −72, 45 (L)**
									R amygdala	**20, −10, −26 (R)**
									L caudate	**−8, 7, 16 (L)**
								Rewarded responses versus punished errors Early versus late trials	**PSY/CD/ODD < HC:**	
									R OFC (BA 10)	**23, 36, −13 (R)**
									L middle frontal gyrus (BA 8)	**−2, 16, 13 (L)**
									PSY/CD/ODD < HC:	
									R OFC (BA 11)	**5, 64, −8 (R)**
									L caudate	**−44, 12, 49 (L)**

Significant effects are listed in bold ($p < .05$)

CD conduct disorder, *EO-CD* early-onset conduct disorder, *ADHD* attention deficit hyperactivity disorder, *HC* healthy controls, *DBD* disruptive behavior disorder, *PSY* psychopathic features, *MDSI* Maudsley Diagnostic Structural Interview, *DSM-IV* Diagnostic and Statistical Manual-IV, *SDQ* Strengths and Difficulties Questionnaire, *ASI* Adolescent Symptom Inventory, *CBCL* Child Behavior Checklist, *DICA* Diagnostic Interview for Children and Adolescents, *APSD* Antisocial Process Screening Device, *PCL-YV* Psychopathy Checklist-Youth Version, *KSADS* Kiddie-Schedule for Affective Disorders and Schizophrenia, *P* parent-report, *C* child-report, *CPT* Continuous Performance Task, *R* right, *OFC* orbitofrontal cortex, *BA* Brodmann area, *L* left, *ACC* anterior cingulate cortex, *NAcc* nucleus accumbens, *MNI* Montreal Neurological Institute

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