Original article

# Dysfunctional neurocognition in individuals with clinically significant psychopathic traits

Robert James R. Blair, PhD

The main goal of this review is to consider the main forms of dysfunctional neurocognition seen in individuals with clinically significant psychopathic traits (ie, reduced guilt/empathy and increased impulsive/antisocial behavior). A secondary goal is to examine the extent to which these forms of dysfunction are seen in both adults with psychopathic traits and adolescents with clinically significant antisocial behavior that may also involve callous-unemotional traits (reduced guilt/empathy). The two main forms of neurocognition considered are emotional responding (to distress/pain cues and emotional stimuli more generally) and reward-related processing. Highly related forms of neurocognition, the response to drug cues and moral judgments, are also discussed. It is concluded that dysfunction in emotional responsiveness and moral judgments confers risk for aggression across adolescence and into adulthood. However, reduced reward-related processing, including to drug cues, is only consistently found in adolescents with clinically significant antisocial behavior, not adults with psychopathy.



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## Introduction

The term *psychopathy* characterizes an increased risk for antisocial behavior coupled with pronounced emotional deficits reflecting reduced guilt, remorse, and empathy.[1,2] In children this emotional component is typically referred to as callous-unemotional (CU) traits.[1] Children and youth with CU traits are at notably increased risk for meeting criteria for psychopathy as adults.[3] Psychopathic traits are a source of considerable concern as they are associated with particularly heightened levels of aggression that may be less amenable to treatment than other factors increasing the risk for violence.[1,2] The goal of this review is to consider the main neurocognitive impairments seen in individuals with clinically significant psychopathic traits. Given the necessity for brevity, several sections of the literature will not be considered: First, there will be no consideration of data from healthy participants who vary in levels of psychopathic traits. While it is useful to know the extent to which a form of pathology associated with specific symptoms is seen in healthy individuals, it is probably unwise to assume that data from healthy individuals is informative for understanding individuals with a clinically significant condition. The clearest example of the necessity for caution is provided by the literature relating reward responsiveness to impulsiveness (a core symptom of Attention Deficit-Hyperactivity Disorder [ADHD]). In a critical meta-analytic review of the literature,[4] the authors concluded that, while increased stri-

**Author affiliations:** Center for Neurobehavioral Research, Boys Town National Research Hospital, Boys Town, Nebraska, US. **Address for correspondence:** Susan and George Haddix Endowed Chair in Neurobehavioral Research, Center for Neurobehavioral Research, Boys Town National Research Hospital, 14100 Crawford Street, Boys Town, NE 68010, US. (email: James.blair@boystown.org)

atal responsiveness to reward was associated with increased impulsiveness in healthy individuals, it was associated with decreased impulsiveness in patients with ADHD. Given the evidence presented by this review, it would be unwise to make claims regarding the functional impairment underpinning clinical ADHD, and potentially other conditions like psychopathic traits, on the basis of data obtained from healthy individuals.

Second, neither data from structural imaging nor resting-state studies will be considered. Reviews of this literature are available elsewhere. The data are clearly informative regarding the pathophysiology of the disorder. However, the focus on this paper is on neurocognition. Any functional interpretation of structural imaging or resting state data is inevitably reverse inferencing – not based on an experimental manipulation of a functional process.

Dysfunction in emotional responsiveness and moral judgments confers risk for aggression across adolescence and into adulthood

This review will consider the data from studies with both children and adults. Indeed, this review will address the extent to which the literature is consistent across adolescent and adult populations. However, it is necessary to note some concern here. The functional magnetic resonance imaging (fMRI) literature with adolescent samples has typically considered youth with *DSM* diagnoses of Conduct Disorder (CD) or Oppositional Defiant Disorders, relative to comparison youth,[5,6] and then sometimes examined the modulation of blood oxygen level-dependent (BOLD) responses by psychopathic or callous/unemotional traits.[7,8] In contrast, the literature with adults has typically ignored psychiatric diagnostic status (including that of the adult homologue of CD, antisocial personality disorder) in favor of examining forensic populations either differing by group according to their Psychopathy Checklist Revised (PCL-R) score[9,10] or via examination of severity of functional impairments as a function of *level* of psychopathic traits as indexed by the Hare Psychopathy Checklist, Revised (PCL-R).[11,12] As such, this review might be better conceptualized as addressing the extent to which specific impairments in neurocognition are associated with clinically significant levels of antisocial behavior/psychopathic traits. Of course, this type of focus is more compatible with the more recent push to generally consider psychiatry in terms of forms of pathology giving rise to symptom groups rather than considering the pathology found in individuals showing specific clusters of behavioral symptoms.[13]

The review will concentrate on two related main foci of the fMRI literature on clinically significant psychiatric traits, emotional and reward processing, and implications of these functions for responding to drug cues and moral judgements. Emotional/reward processing implicates a series of highly interconnected regions including the amygdala, striatum, anterior insula, anterior cingulate/dorsomedial, rostromedial, ventromedial frontal and posterior cingulate cortices *(Figure 1)*. These regions in turn have a web of connections with other brain areas. The amygdala and striatum are particularly important for forms of reinforcement-based learning. Anterior insula and dorsomedial frontal cortices are particularly important for selecting responses/avoiding/inhibiting responses as a function of expected value information and other cues. Rostromedial, ventromedial frontal, and posterior cingulate cortices are particularly important for representing the value (and possibly with respect to rostromedial frontal cortex, in particular, maintaining the value) of response choices.

Attention-based views, dominant in the 1980s and 1990s, and still very prominent today,[14] will receive less attention but will be briefly considered.

## Emotional processing

### Emotional facial expressions

Emotional expressions have a communicative function: they both modulate ongoing behavior and allow the rapid transmission of valence information regarding objects and actions.[15] This is seen, for example, in social referencing where the observer learns the value of a stimulus based on another individual's emotional reaction to it (eg, the negative value of a novel threat because the caregiver shows fear towards it). There has been a considerable amount of work that has investigated the processing of emotional facial expressions in adolescent and adult populations with psychopathic traits. This is related to suggestions that psychopathic traits might particularly relate to a reduced

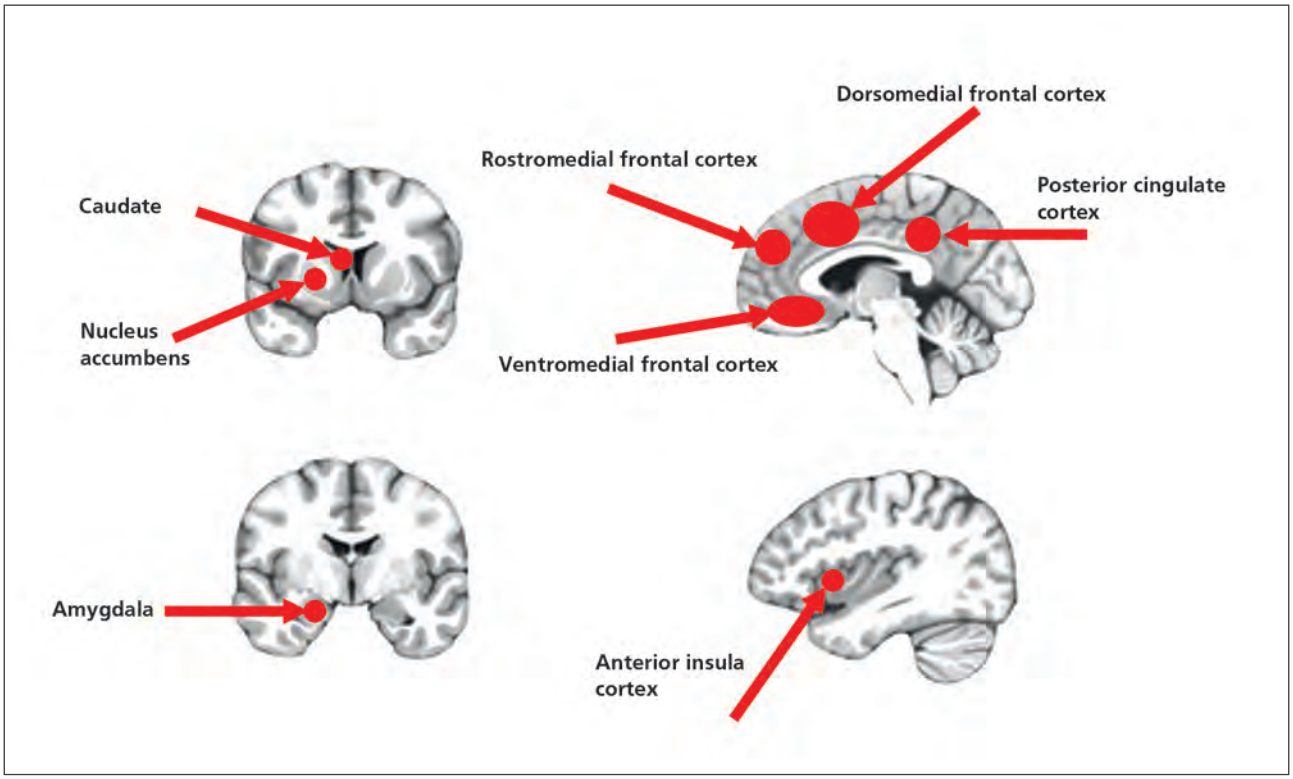


**Figure 1. Regions implicated in emotional and reward-related processing.** These regions have been implicated as critical for specific functional processes and selectively interact to allow specific functional processes to occur. Studies have reported dysfunctional responses within these regions in adolescents with conduct problems and adults with psychopathy during emotional and reward-related processing.

response to the distress of others.[15] According to this view, such a reduced response should be associated with reduced learning to avoid actions that harm other individuals (the individual finds the "punishment" of the other individual's distress less aversive) and thus reduced avoidance of the commission of actions that might harm other individuals.[15]

Face stimuli are processed by a series of neural regions including those related to emotional processing listed above as well as temporal cortical regions (eg, fusiform and superior temporal cortex) that are particularly involved in processing faces relative to inanimate objects.[16] Many of these regions (eg, the amygdala, anterior insula, and fusiform cortex) show stronger responses to emotional relative to neutral faces.[16] Moreover, there are indications of regional specification regarding particular emotional expressions; the amygdala appears particularly responsive to fearful, sad, and happy expressions but not angry and disgusted expressions, while anterior insula cortex is particularly responsive to disgust and anger expressions.[16]

Youth and adults with conduct problems, particularly those with psychopathic or CU traits, have been reported to show deficits in expression recognition, perhaps particularly for fearful, sad, and happy expressions, though this is debated.[17,18] The impaired recognition of fearfulness and sadness is pervasive, applying also to vocal tones and body postures.

Youth with conduct problems, particularly when marked with psychopathic or CU traits, consistently show reduced responses to facial expression stimuli (particularly fearful and sad expressions) in emotion/face processing regions, including the amygdala.[5,7,19-22] In a particularly interesting study, and consistent with theory,[15] Lozier and colleagues revealed that the positive relationship between CU traits

and aggression was mediated by the reduced responsiveness of the amygdala to the distress of other individuals.[20] Work with adults with psychopathy also reports similarly reduced BOLD responses in affect/face processing regions to facial expressions relative to comparison adults.[9,11,23] However, only one of these studies specifically reported reduced amygdala responses.[23]

In summary, the existing literature relatively reliably indicates reduced responsiveness to facial expressions, particularly distress cues, in children and adults with conduct problems that may be particularly marked in those with psychopathic traits. The regions implicated across studies are not always consistent–and the reduced amygdala responsiveness is seen much more in the work with adolescents than that with adults–but the basic finding of reduced neural responsiveness appears robust.

**Pain stimuli**

The facial expressions of another individual in pain can also be considered a distress cue. However, many studies examining responsiveness to the pain of another individual in individuals with psychopathic/CU traits have often used visual stimuli depicting painful events (a hand caught in a slamming door) rather than facial *expressions* of pain. These are depictions of events associated with another individual's pain, and require either interpretation or some association with the aversiveness of such events in the viewer's past.

A series of studies have identified a "pain matrix"; a network of brain regions that respond to the sight of another individual in pain. These regions include the neural regions related to emotional processing listed above as well as supplementary motor area (for a meta-analytic review of this literature, see ref 24). The activation of the supplementary motor area is interesting as it probably reflects activity relating to the association of the visual image with comparable events in the observer's past.

It has been known for some time that individuals with psychopathy show reduced emotional (autonomic) responses to the sight of other individuals in apparent pain.[25] Recent fMRI studies have examined the neural basis of this dysfunction in youth with conduct problems.[26-28] These studies have reported that observing others in pain was associated with *reduced* activity within rostral medial/anterior cingulate cortex, the amygdala and insula cortex in this population.[26-29] Work with adults with psychopathy has revealed that all of these regions are also associated with reduced responsiveness in this population as a function of level of psychopathy.[10,12,30] For two of the studies (one child, one adult), asking the participants to imagine the events were happening to another were *particularly* associated with reduced activity as a function of psychopathic traits.[12,26] However, it should be noted that a third study found psychopathy effects only when participants were *not* asked to "feel with the receiving (50%) or the approaching (50%) hand" (they were present under passive viewing conditions).[10]

In summary, the existing literature relatively reliably indicates reduced responsiveness to pain stimuli in children and adults with conduct problems, particularly as a function of psychopathic traits. Studies have relatively reliably identified reduced responding within dorsomedial and anterior insula cortices and, though less often, the amygdala.

**Emotional stimuli generally and the role of attention**

In addition to the above evidence of reduced responding to emotional expressions and pain stimuli, there is some work indicating that responding during aversive conditioning and to emotional stimuli may be generally compromised in youth with callous and unemotional traits and adults with psychopathy. With respect to aversive conditioning, behavioral work has indicated that this is reduced in adults with psychopathy relative to comparison adults (ie, reduced differentiation in autonomic responding between the stimulus associated with the punishment and the one not associated with the punishment)[31] though findings in adolescents have been rather more mixed.[32,33] fMRI work has indicated that CU traits in youth and psychopathy in adults are associated with reduced amygdala and anterior cingulate responding.[33,34] With respect to emotional responding, work has indicated that both youth with CU traits[35] and adults with psychopathic traits[36,37] show reduced responses to negative pictures within the amygdala, ventromedial frontal cortex, and other emotion-associated regions relative to comparison individuals.

The latter two studies[36,37] together with other work[10,12,26] are interesting because they indicate that attentional manipulations can, under certain circumstances, influence the extent to which individuals with psychopathic traits show reduced neural responses to emotional stimuli. Most of the studies

reviewed above, indicating reduced responsiveness, have used passive viewing conditions, or conditions where the participant is engaged in a low attentional load task such as gender identification. However, two studies revealed that any reduced responding to emotional images in adults with psychopathy was effectively "normalized" when the participants were explicitly asked to attend to the emotional content of the picture and classify the picture as emotional or nonemotional[37] or enhance their emotional responding.[36] A third study reported that this also occurred if participants were encouraged to "empathize" with the actors in a video.[10] However, it should be noted that two further studies reported, in contrast, that the association with psychopathy and reduced responding to pain stimuli was *most marked* during the empathy for others condition.[12,26]

These findings are of interest given views that psychopathy might reflect a problem in attention rather than emotion.[14] The attention-based views effectively suggest that individuals with psychopathy attend to other features of the stimulus array than the emotional ones and thus show weaker responses to the emotional stimuli. Clearly, such a view is compatible with several of the above findings[10,36,37] though inconsistent with others.[12,26] Of course, an alternative speculation is that individuals with psychopathy do show reduced responding to emotional stimuli. However, if the intensity of this stimulus is sufficiently heightened, via an attentional manipulation that increases the emotional stimulus' representational strength, group differences are reduced (because the individuals with lower psychopathic traits reach an asymptote level in responding). This latter view is also compatible with the absence of findings, indicating that individuals with higher psychopathic traits show heightened recruitment of regions implicated in top-down attention during passive viewing and other task manipulations.

## Reward responsiveness

The second main focus of the fMRI literature on clinically significant psychiatric traits considered here is on reward responsiveness. There is a considerable animal and human literature on regions involved in responding to reward. An adequate review of this literature is beyond the scope of the current paper (see instead ref 38). However, the regions typically implicated are those previously considered with respect to emotional processing; eg, the amygdala, striatum, anterior insula, anterior cingulate/dorsomedial, rostromedial, ventromedial frontal, and posterior cingulate cortices *(Figure 1)*.

Two possibilities might be considered with respect to the impact of reward on antisocial behavior: First, excessive reward responses to objects in the immediate environment might increase impulsive behavior towards these objects, including aggression; or Second, reduced reward sensitivity/responsiveness, particularly within regions critical for the representation of long-term goals, should result in an individual who makes poorer decisions (response choices will be less well guided by goal-modifiable reward expectations). Such an individual is also more likely to be impulsive and more likely to become frustrated and aggressive as a function of their frustration.[39]

Very little work supports the suggestion of increased reward responsiveness in adolescents with conduct problems/CU traits. One study reported increased striatal responsiveness to reward in a small sample of youth with externalizing difficulties relative to comparison youth[40] while a second found that within a group of adolescents with conduct problems, increasing CU traits were associated with increased striatal responses to watching another win reward–though CU traits did not relate to reward responding when the participant won a reward (ie, there were no indications of heightened responsiveness for reward for the self[41]). In contrast, a series of studies have reported that youth with conduct problems, some of whom also had elevated psychopathic traits, show *reduced* neural responsiveness to reward/reward omissions within striatum and ventromedial prefrontal cortex.[42-46] However, most of this literature has not indicated a relationship between reward responsiveness and CU/psychopathic traits in particular. The only exception to this is a report of an inverse relationship between rostro- and ventromedial responses during reward anticipation and level of CU traits in a large adolescent sample.[47]

The literature with adults is less clearcut. Three studies have reported that psychopathy, or antisocial personality disorder,[48] were associated with increased reward responsiveness.[48-50] These studies reported that: (i) psychopathy was associated with stronger subjective value-related activity within the nucleus accumbens during inter-temporal choice[49]; (ii) individuals with antisocial personality disorder show increased responses in right orbitofrontal and subgenual cingulate

cortices to the receipt of reward[48]; and (iii) individuals with psychopathy show increased nucleus accumbens responses during reward anticipation.[50] However, it is notable that this last result held only if the individuals with psychopathy were compared against healthy participants who scored below the healthy participant sample median for impulsivity on a personality measure. There were no group differences between those with psychopathy and healthy participants who scored above the healthy participant median for impulsiveness. Two further studies reported no group differences in reward responsiveness.[51,52] Pujara and colleagues found no significant relationship between psychopathy and nucleus accumbens response to reward relative to neutral reinforcement.[51] However, they showed a significant inverse relationship between psychopathy and loss relative to neutral reinforcement. Gregory and colleagues,[52] similar to an earlier study with youth with psychopathic tendencies using the same reversal learning task,[53] observed a failure in adults with psychopathy to suppress responding within posterior cingulate and insula cortex to unexpected punishment. Finally, an additional study reported diminished responding within rostral anterior cingulate cortex during high, relative to low uncertainty, choice conditions in a decision-making task.[54]

In summary, the literature relatively clearly indicates that adolescents with conduct problems show reduced reward responsiveness, though whether this relates to severity of CU traits is less certain. Currently, the literature with respect to adults with psychopathy is currently equivocal.

## Drug cues

One further way to examine the relationship between reward responsiveness and psychopathy is by examining the relationship between psychopathy and neural responsiveness to drug cues. Substance abuse is associated with a heightened response to drug reward cues and a diminished response to non-drug rewards within regions including dorsomedial frontal, anterior cingulate and anterior insula cortices, the amygdala and striatum (for a review of this literature, see ref 55). This is thought to reflect a learning-based adaptation to the very high levels of dopamine released when the substance is abused such that cues anticipating this release become highly rewarding. Other rewards are associated with relatively weaker dopamine responses and thus become less rewarding.[55]

If psychopathy is associated with heightened reward responsiveness, one might predict that this learning would occur more rapidly and underlie the emergence of the substance abuse disorders which are often comorbid in this population.[56] Individuals with psychopathy should show heightened responsiveness to drug cues. Alternatively, if psychopathy is associated with reduced reward responsiveness, one might predict that individuals with psychopathy should show reduced responsiveness to drug cues. The high comorbidity of psychopathic traits with substance abuse disorders would reflect dysfunction in systems representing anticipated rewards and punishments associated with their decision-making impairments.[39]

Currently, only three studies have examined this issue. Two, one in an adult[57] and the other in an adolescent[58] sample, are clearly consistent with the second position. Both studies reported a negative correlation between psychopathic traits and neural response to drug versus neutral images within anterior cingulate cortex, amygdala, and striatum.[57,58] The results of the third study were somewhat more complex.[56] In contrast to the previous two studies, this study reported that psychopathic traits were *positively* correlated with responsiveness to drug relative to food cues within the right anterior insula cortex and the left amygdala. However, *only* individuals with *lower* psychopathic traits showed an increasing differentiation in their response to drug vs food cues as a function of duration of drug use. This was seen within left dorsomedial prefrontal and right anterior insula cortex and striatum. Instead, individuals with higher psychopathic traits showed a decreasing differentiation in their drug vs food cues response within these regions. In short, this third study indicated a *heightened* differentiation in individuals with psychopathy in response to drug versus food cues within the right anterior insula cortex and the left amygdala but that this differentiation was *decreased* as a function of length of substance abuse within left dorsomedial prefrontal and right anterior insula cortex and striatum as a function of psychopathy level.

In summary, the literature on responsiveness to drug cues has not clarified the situation with respect to reward responsiveness and psychopathy in adults. The Vincent et al study[58] is consistent with the previous work with adolescent samples indicating reduced reward responsiveness as a function of conduct disorder/psychopathic traits.[42-46] The results with adults are again relatively inconsistent.[56,57]

**Moral judgments**

Moral judgments involve both emotional responses to the emotional content of the moral/immoral action and decision-making on the basis of this content. Youth with conduct problems and CU traits and adults with psychopathy are compromised in at least some forms of moral judgments (for a more detailed review of the behavioral literature, see ref 59). While they typically show no difficulty in recognizing which acts are transgressions,[60,61] they: (i) distinguish less in their permissibility judgments between acts that harm others relative to those that simply cause social disorder in the absence of rules[60]; (ii) endorse less such harm-based norms though their endorsement of social-disorder based rules remains intact[62]; and (iii) are more likely to allow actions that indirectly harm another.[63]

Moral judgments involve the recruitment of the regions depicted in *Figure 1* (for a meta-analytic review of the moral judgment/fMRI literature, see ref 64). Consistent with the behavioral findings, the fMRI literature has relatively consistently documented in studies using a variety of paradigms that youth and adults with CU/ psychopathic traits show reduced responding within these regions during moral judgment tasks relative to comparison individuals.[8,65-67]

## Conclusions

The two main goals of this review were to examine the main neurocognitive impairments seen in individuals with clinically significant psychopathic traits and the extent to which they were seen in adolescent and adult samples. Of course, the immediate concern, particularly with this second aim, is the potential differences in the populations identified in the research on adolescents relative to the research on adults. As noted above, the adolescent literature typically starts with the *DSM* diagnosis of conduct disorder and then may examine the influence of CU traits. The adult literature typically starts with the psychopathy checklist and may, on the basis of this, examine relationships with particular psychopathic factors (ie, the emotional factor 1 and the more antisocial/ impulsive behavior factor 2). Importantly, though, despite these concerns it should by now be clear that the adolescent and adult literature, at least with respect to emotional responding, are consistent with one another. A population, seen in adolescents and adults, marked by high levels of aggression and disrupted empathy/guilt are associated with weaker responding to facial distress cues, indications of pain (perhaps particularly when these are represented as occurring to another individual) and emotional stimuli in the regions depicted in *Figure 1*. Moreover, a putative functional process thought to be reliant on this emotional processing, particularly appropriate responding to the distress others, moral judgment is disrupted in this population. This form of reasoning impairment likely increases the risk that these individuals will engage in antisocial behavior that harms other individuals.

There is inconsistency though with respect to the literature on reward processing. The studies with adolescents indicate that a population marked by high levels of aggression is associated with reduced reward responsiveness and that this is echoed in a relatively reduced responsiveness to drug cues. In contrast, the adult literature is relatively evenly divided between studies reporting hyper- and hypo-reward responsiveness (including to drug cues) in adults with psychopathic traits. The reasons for this inconsistency are unclear. It could represent differences in the type of individual identified in the studies with adolescents relative to those with adults. But that would suggest that there is a population of highly aggressive adolescents who are marked by hyper-reward responsiveness. Yet, none have been found. There could be developmental effects. It is notable that when psychiatric comorbidities are examined, the rates of ADHD in the adolescent samples are typically high (>60%[42]). As noted above, ADHD is marked by reduced reward responsiveness relative to comparison adolescents.[4] It is unclear the extent to which the pathophysiology of ADHD is ameliorated by adulthood, and much of the adult literature has not involved full psychiatric assessments of the participant samples. But it remains possible that psychopathy in adulthood is not comorbid with ADHD and perhaps the findings of increased reward responsiveness reflect the relationship between increased reward responsiveness and impulsive reward-seeking behavior seen in healthy participants.[4] Indeed, the one adult study examining the relationship of reward responsiveness found that increased reward responsiveness was only seen relative to low (not high) impulsive healthy participants[50]; ie, even though the study reported hyper-reward responsiveness this responsiveness was within the healthy range.

In conclusion, this review highlights two forms of dysfunctional neurocognition that incur risks of psychopathology. The first concerns reduced emotional responsiveness and the implications of this for empathy, moral judgments and immoral behavior. This form of neurocognitive dysfunc-

tion confers risk for aggression across adolescents and into adulthood. The second concerns reduced reward responsiveness. This is deficient in adolescents with significant conduct problems and probably exaggerates their behavioral difficulties as a function of poor decision-making. It is likely deficient in at least some adults with conduct problems but how pervasive this is the case and whether there is any relationship with psychopathy is currently unclear. ■

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