CHAPTER 21

The Development and Neural Bases of Psychopathy

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In this chapter, we address three main issues, the first being the nature of psychopathy. Specifically, we describe the defining features of the disorder and indicate how the construct differs from that of *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, text revision (DSM-IV-TR; American Psychiatric Association, 2000) diagnoses of antisocial personality disorder (APD) and the pediatric counterpart to this disorder, conduct disorder (CD). Second, we address the origins of psychopathy, giving particular consideration to evidence regarding whether psychopathy has a genetic or social basis. Third, we consider the neurocognitive basis of psychopathy, describing both areas of relative consensus and points of contention within the literature.

What Is Psychopathy?

Cleckley (1976) provided the foundation for the modern conceptualization of psychopathy in his book *The Mask of Sanity*. His description was used by Robert Hare to develop the original Psychopathy Checklist (PCL; Hare, 1980), which was first revised in 1991 (the PCL-R; Hare, 1991) and then again in 2003 (Hare, 2003). The PCL-R is an empirically determined, formalized tool for the assessment of psychopathy in adults. Comparable measures have been developed for assessment of children and adolescents—the PCL: Youth Version (Forth, Kosson, & Hare, 2004; Kosson, Cyterski, Steuerwald, Neumann, & Walker-Matthews, 2002) and the Antisocial Process Screening Device (Frick & Hare, 2001).

Psychopathy involves two core components: emotional dysfunction and antisocial behavior. These components have been identified through factor analysis (Frick, 1995; Frick, O'Brien, Wootton, & McBurnett, 1994; Harpur, Hakstian, & Hare, 1988; Hart, Forth, & Hare, 1990; Hobson & Shine, 1998). The emotional dysfunction involves "callous and unemotional" (CU) traits, such as reduced guilt and empathy, as well as reduced attachment to significant others. The antisocial behavior component involves a predisposition to antisocial behavior from an early age. Some recent work has challenged the two-factor description of psychopathy in favor of three-factor (Cooke & Michie, 2001; Frick & Hare, 2001) or four-factor (Williams, Paulhus, & Hare, 2007) solutions. However, it should be noted that both three- and four-factor solutions effectively involve the subdivision of the original emotional dysfunction into two subcategories: the CU dimension (concentrating on the lack of guilt, empathy, or attachment to significant others) and a narcissism dimension (concentrating on grandiose feelings of self-worth) (Cooke & Michie, 2001; Frick & Hare, 2001).

Given its reliance on antisocial behavior, the construct of psychopathy overlaps with the DSM-IV-TR diagnoses of both childhood CD and adult APD. However, only a subset of these diagnosed with either CD or APD meet criteria for psychopathy. In adult forensic samples, only approximately 25% of individuals who warrant a diagnosis of APD meet criteria for psychopathy (Hare, 2003). The major difference between psychopathy and the DSM-IV-TR diagnoses of CD and APD is that the latter focus on the antisocial behavior and do not consider its potential etiology. In contrast, the construct of psychopathy emphasizes the specific form of emotional dysfunction thought to underlie the emergence of the antisocial behavior (Blair, Mitchell, & Blair, 2005).

The failure of the DSM-IV-TR to consider the causes of CD and APD is regrettable. Many different factors may increase an individual's risk for displaying antisocial behaviors such as aggression (see Blair et al., 2005); as a result, individuals with wholly different pathophysiologies are being given these diagnoses. Thus the antisocial behavior of some children with CD and adults with APD is associated with the reduced emotional responsiveness of psychopathy, whereas that of others is associated with exaggerated emotional responsiveness or otherwise dysregulated emotional responding.

Its focus on etiology renders psychopathy a more useful classification. For example, studies have shown the predictive power of scores on the PCL-R with respect to recidivism (Hare, Clark, Grann, & Thornton, 2000; Hart, Kropp, & Hare, 1988; Kawasaki et al., 2001). Variance among individuals in CU traits (the affective component of psychopathy) predicts receptivity to parenting strategies, with heightened levels of CU traits being associated with less receptivity to standard parenting strategies (Oxford, Cavell, & Hughes, 2003; Wootton, Frick, Shelton, & Silverthorn, 1997). Children's CU traits also predict their responsiveness to behavioral parent-training intervention (Hawes & Dadds, 2005).

A defining behavioral feature of psychopathy is an increased risk for "instrumental" aggression (Cornell et al., 1996; Williamson, Hare, & Wong, 1987). Instrumental aggression (also referred to as "proactive aggression") is purposeful and goal-directed; the aggression is used instrumentally to achieve a specific desired goal (Berkowitz, 1993). This contrasts with "reactive" aggression (also referred to as "affective" or "impulsive" aggression), which is not goal-directed, but is triggered by a frustrating or threatening event and is frequently associated with anger. Many conditions increase the risk for reactive aggression—for example, lesions including the orbital frontal cortex (OFC) (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Grafman, Schwab, Warden, Pridgen, & Brown, 1996), childhood bipolar disorder (Leibenluft, Blair, Charney, & Pine, 2003), posttraumatic stress disorder (Calhoun

et al., 2002), intermittent explosive disorder (Coccaro, 1998), or borderline personality disorder (Siever, Torgersen, Gunderson, Livesley, & Kendler, 2002). The only condition known to increase the risk for instrumental (and reactive) aggression is psychopathy (Cornell et al., 1996; Williamson et al., 1987).

What Are the Origins of Psychopathy?

Genetic Factors

Many studies have implied that antisocial behavior is heritable (for a review, see Rhee & Waldman, 2002). It is improbable that genes code for antisocial behaviors directly, however. Instead, genes may influence those systems that increase an individual's risk for antisocial behavior.

In the section above, we have described psychopathy as an emotional disorder marked by reduced guilt and empathy, and associated with an increased risk for antisocial behavior. Is there a genetic basis to psychopathy? Several recent studies have investigated this issue and concluded that it is heritable and under moderate environmental influence (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005; Viding, Blair, Moffitt, & Plomin, 2005). Moreover, whereas antisocial behavior in the presence of elevated levels of CU traits is strongly heritable (81%), heritability of antisocial behavior without CU traits is only moderate (30%) (Viding et al., 2005).

Although behavioral genetic work indicates a genetic contribution to the development of psychopathy, the molecular genetics of psychopathy are currently unknown. Suggestive data are provided by recent investigations of the influence of specific genetic polymorphisms on neural and behavioral responding. These specific genetic polymorphisms appear to have implications for the responsiveness of the amygdala, a structure that appears dysfunctional in psychopathy (see below). For example, several studies have reported that individuals who are homozygous for the long allele of the serotonin transporter gene promoter (5-HTTLPR) show significantly reduced amygdala responding to emotional expressions, relative to those who have the short-form polymorphism of 5-HTTLPR (Hariri et al., 2002). In addition, such individuals show behavioral impairment on some emotional learning tasks reliant on the amygdala (Finger et al., 2007). This is not to suggest that long-allele homozygosity causes psychopathy, but rather that the serotonin transporter gene may be one of several genes that have polymorphisms associated with decreased emotional and amygdala responsiveness. The basic genetic risk for psychopathy may emerge in individuals who have several independent polymorphisms that predispose them toward reduced emotional and amygdala responsiveness.

Environmental Factors

An increased risk for aggression, and potentially for psychopathy, has been associated with a variety of environmental causes. In this section we will consider one environmental cause—exposure to extreme threats.

An individual is at increased risk for aggression following exposure to extreme threat, whether it is violence in the home or neighborhood (Miller, Wasserman, Neugebauer, Gorman-Smith, & Kamboukos, 1999; Schwab-Stone et al., 1999), or physical or sexual abuse (Dodge, Pettit, Bates, & Valente, 1995; Farrington & Loeber, 2000). However, these data do

not necessarily imply that this environmental factor increases the risk for the development of psychopathy.

Considerable animal work has shown that prolonged threats and stress lead to long term potentiation of the neural and neurochemical systems that respond to threat; in other words, the individual becomes more responsive to aversive stimuli (Bremner & Vermetten, 2001; King, 1999). Moreover, traumatic exposure in humans, such as exposure to violence in the home or neighborhood, increases the risk for mood and anxiety disorders, which are typically associated with increased emotional responsiveness rather than with the decreased emotional responsiveness seen in psychopathy (Charney, 2003; Gorman-Smith & Tolan, 1998; Schwab-Stone et al., 1999).

Because exposure to extreme threats is associated with an increase in emotional responsiveness rather than a decrease, it is unlikely that it causes psychopathy to develop. Why then does it increase the risk for aggression? The answer to this question probably relates to the type of aggression being displayed. For example, exposure to threat as a consequence of abusive parenting or other trauma is associated with an increased risk for *reactive* aggression (Dodge et al., 1995), such that the individual is more likely to respond with aggression when provoked by threatening or frustrating events.

The mammalian response to threat is gradated. At low levels of danger from a distant threat, animals tend to freeze. As the danger level increases and the threat draws closer, animals attempt to escape. At the highest level of danger, when the threat is very close and escape is no longer possible, reactive aggression is displayed (Blanchard, Blanchard, & Takahashi, 1977). This gradated response is mediated by a basic threat system that runs from medial amygdaloidal areas downward, largely via the stria terminalis to the medial hypothalamus, and from there to the dorsal half of the periaqueductal gray (Gregg & Siegel, 2001; Panksepp, 1998). This system is regulated by OFC, medial frontal cortex, and ventrolateral frontal cortex (Blair, 2004).

The responsiveness of the neural threat system can be potentiated by prior exposure to threatening or stressful events (Charney, 2003), and the behavioral response to threat (whether freezing, fleeing, or fighting) is determined by the responsiveness of the system. If the system has been potentiated by prior threat exposure, an individual will escalate his or her response to the threat more rapidly than another individual who has not had that prior exposure. In other words, we believe that exposure to extreme threat or stress increases the risk for *reactive* aggression, because it increases the underlying responsiveness of the system and therefore makes the individual more likely to escalate his or her response to threats.

What Is the Neurocognitive Basis of Psychopathy?

Various accounts have been offered to explain psychopathy. Some exist primarily at the neural level (Gorenstein, 1982; Kiehl, 2006), and others primarily at the cognitive level (Blair, 1995; Frick & Marsee, 2006; Hartung, Milich, Lynam, & Martin, 2002; Lykken, 1995); still others are cognitive neuroscience positions, which consider the cognitive functions mediated by particular neural regions (Blair, 2001; Damasio, 1994; Patrick, 1994; Raine, 2002). The accounts have included the frontal lobe hypothesis (Gorenstein, 1982), the somatic marker hypothesis (Damasio, 1994), the amygdala-based hypotheses (Blair, 2001; Patrick, 1994), fear dysfunction accounts (Frick & Marsee, 2006; Lykken, 1995; Patrick, 1994), the paralimbic

hypothesis (Kiehl, 2006), the response set modulation hypothesis, models based on impulse control problems (Hartung et al., 2002), and the violence inhibition mechanism and integrated emotion systems accounts (Blair, 2001, 2006).

Some positions can be considered to have been superseded (Blair, 1995; Gorenstein, 1982; Raine, 2002). That is, some early positions appeared to suggest that there is general frontal lobe impairment in psychopathy (Gorenstein, 1982; cf. Raine, 2002). However, because the frontal lobes account for nearly 50% of the total cerebral cortex, such positions were highly underspecified. Moreover, neuropsychological work demonstrated that functions mediated by the dorsolateral and dorsomedial frontal cortex are intact in individuals with psychopathy, and that only functions mediated by the OFC, ventromedial frontal cortex, and possibly inferior frontal cortex appear compromised (Blair et al., 2006b; LaPierre, Braun, & Hodgins, 1995; Mitchell, Colledge, Leonard, & Blair, 2002). For a review of this literature and criticisms of these positions, see Blair et al. (2005).

Blair's (1995) original violence inhibition mechanism model faced difficulty accounting for data indicating more general problems in individuals with psychopathy in the processing of punishment information (e.g., Patrick, 1994). This has led to a revision and expansion of the model at both the cognitive and neural levels (i.e., the integrated emotion systems model; Blair, 2004, 2006).

Although some accounts have been superseded, exciting progress has been made in the field of psychopathy over the past 10 years. This progress has permitted refinements to be made to the frontal lobe and violence inhibition mechanism positions. A specific form of frontal lobe dysfunction does appear to be a main component of the pathophysiology of psychopathy. An emphasis on the importance of an appropriate response to the distress of others (as in the violence inhibition mechanism model) remains an important part of a developmental account of the disorder. Moreover, the general refinement and increased specification of positions such as these has resulted in increased consensus among the various positions emerging. We briefly describe the points of (rough) consensus below.

Consensus at the Neural Level

1. *At the anatomical level, amygdala dysfunction is a core feature of psychopathy.* This position was first proposed by Patrick (1994) and has been considerably extended and revised by Blair (e.g., 2001, 2006). The amygdala is one of the systems considered impaired within Kiehl's (2006) paralimbic hypothesis, and although neither Lykken nor Frick makes extensive reference to the structure, the impairments described by both are highly consistent with an amygdalocentric view (Frick & Marsee, 2006; Lykken, 1995).

Core deficits seen in psychopathy are in aversive conditioning (Birbaumer et al., 2005; Lykken, 1957), the augmentation of the startle reflex by visual threat primes (Levenston, Patrick, Bradley, & Lang, 2000; Patrick, Bradley, & Lang, 1993), passive avoidance learning (Newman & Kosson, 1986), and the recognition of fearful expressions (Blair, Colledge, Murray, & Mitchell, 2001). These impairments are all seen following amygdala lesions (for a review of the literature, see Blair, 2006). Moreover, recent neuroimaging work with adult forensic populations has consistently indicated that individuals with psychopathy show reduced amygdala responses to emotional words in the context of emotional memory paradigms (Kiehl et al., 2001) and during aversive conditioning (Birbaumer et al., 2005). Work with subclinical populations has similarly found that individuals with psychopathic traits show reduced amygdala responses to emotional expressions (Gordon, Baird, & End, 2004)

and less amygdala differentiation in responding when making the choice to cooperate versus defect in a prisoner's dilemma paradigm (Rilling et al., 2007).

One important position that does not regard amygdala dysfunction as important for the development of psychopathy is the somatic marker hypothesis of Damasio (1994). Damasio (1994) and colleagues propose that psychopathy may be a developmental consequence of early damage to the OFC. However, this view has been extensively criticized (Blair, 2004; Blair & Cipolotti, 2000). Importantly, none of the core impairments seen in psychopathy are found following OFC lesions. They are, however, found following lesions of the amygdala (for extended discussions of these data, see Blair, 2004, 2006).

2. *It is necessary to consider OFC functioning when considering psychopathy.* While Damasio's viewpoint that psychopathy is a developmental consequence of early OFC damage is untenable, given data that the core impairments seen in psychopathy are not found following OFC lesions (see Blair, 2004, 2006), this does not imply that OFC activity is irrelevant to the understanding of psychopathy. Both Blair (2004) and Kiehl (2006) consider atypical OFC activity to contribute to the pathophysiology of psychopathy. However, other dominant theoretical viewpoints remain unspecified at the anatomical level (Frick & Marsee, 2006; Lykken, 1995) or have remained agnostic (Patrick, 1994).

Kiehl's (2006) paralimbic hypothesis is primarily a model at the neural level. There is considerable discussion of structures that may be impaired, but rather less consideration of their functional contribution. In contrast, Blair's cognitive neuroscience position, the integrated emotion systems model (Blair, 2004, 2006), follows Gallagher, Schoenbaum, and their colleagues regarding the function of the OFC and its relationship with the amygdala. The basic suggestion is that the amygdala feeds forward reinforcement information associated with stimuli to medial OFC, which then represents this outcome information, allowing decision making in regard to behavior such as approach and avoidance (Gallagher, McMahan, & Schoenbaum, 1999; Schoenbaum, Nugent, Saddoris, & Setlow, 2002; Schoenbaum, Setlow, Saddoris, & Gallagher, 2003). This suggests that even if there is not primary pathology within the OFC, individuals with psychopathy should show anomalous medial OFC activity in the context of tasks that activate the amygdala. This is exactly what is seen. In Kiehl et al.'s (2001) study of emotional memory, individuals with psychopathy showed not only reduced amygdala responses to the emotional words, but also reduced rostral anterior cingulate cortex/medial OFC activation. In addition to showing reduced amygdala activity, individuals with psychopathy also exhibited medial OFC activity during aversive conditioning (Birbaumer et al., 2005), as well as less medial OFC differentiation in responding when making cooperation versus defection choices in the prisoner's dilemma paradigm (Rilling et al., 2007).

- *Point of contention: The functions of many neural systems are compromised in psychopathy.* According to Kiehl's (2006) paralimbic hypothesis, individuals with psychopathy face disruption of the neural systems that make up what he terms the "paralimbic system" (i.e., the OFC, insula, anterior and posterior cingulate cortex, amygdala, parahippocampal gyrus, and anterior superior temporal gyrus). This is in notable contrast to Blair's position, which states that although the functioning of regions beyond the amygdala and medial OFC may be anomalous under certain conditions, this is due to deficient input from the amygdala and medial OFC rather than to direct dysfunction of the systems themselves.

In some respects, Kiehl's position is likely to be correct. As noted above, data are emerging to indicate a strong genetic contribution to psychopathy (Blonigen et al., 2005; Viding et

al., 2005). It is likely that any genetic contribution will affect the development of structures beyond the amygdala and OFC.

In other respect, Kiehl's position is clearly incorrect. The position uses a neurological approach to understand a psychiatric condition. Kiehl's position implies that the functions of all the regions making up the paralimbic system are compromised under a wide range of circumstances. But available data suggest that this is not the case. For example, some social cognition functions reliant on amygdala function, such as the making of trustworthiness judgments or mental state judgments from eye information, appear spared in psychopathy (Richell et al., 2003; Richell et al., 2005). Similarly, the crucial role of dorsal anterior cingulate cortex in the resolution of response conflict appears spared in psychopathy (Blair et al., 2006b; Hiatt, Schmitt, & Newman, 2004). The problem with Kiehl's position is its neurological approach to this psychiatric condition (i.e., a reference specifically to dysfunctional neural structures). It is unlikely that psychiatric conditions should be explained with reference to dysfunction within particular neural structures, rather than dysfunction in functional systems that rely on multiple structures (and where other functional systems reliant on the same neural structures may be intact).

Consensus at the Cognitive Level

3. *Psychopathy is an emotional disorder.* This is part of the clinical description, and almost all positions agree on this point (Blair, 2001, 2006; Damasio, 1994; Frick & Marsee, 2006; Kiehl, 2006; Lykken, 1995; Patrick, 1994).

4. *Psychopathy reflects impaired processing of reinforcement information.* Almost all positions also agree on this general statement (Blair, 2001, 2006; Damasio, 1994; Frick & Marsee, 2006; Kiehl, 2006; Lykken, 1995; Patrick, 1994). Some of the very earliest positions on psychopathy were variants of this type of position (Hare, 1970; Lykken, 1957).

There are some disagreements in the details, however. The first is whether the impairment in reinforcement processing reflects impaired *learning* on the basis of reinforcement information or the impaired *use* of reinforcement information. Most positions on psychopathy have assumed that the problem reflects an inability to learn from reinforcement information (Blair, 2001, 2006; Frick & Marsee, 2006; Kiehl, 2006; Lykken, 1995; Patrick, 1994). However, Damasio's (1994) viewpoint on psychopathy assumes that it reflects the impaired use of prior learning. Within his model, the OFC is described as responding to somatic marker information to allow decision making. Certain data strongly refute this position, however. For example, individuals with psychopathy show impairment in aversive conditioning (Birbaumer et al., 2005; Lykken, 1957). According to the Damasio model, this process does not require somatic marker information, but rather allows the formation of somatic markers that the OFC can process (Bechara, Damasio, Damasio, & Lee, 1999). Indeed, the data on aversive conditioning in individuals with psychopathy formed the basis of some of the earliest criticism of the somatic marker hypothesis of psychopathy (Blair, 2001).

The second disagreement pertains to whether psychopathy is characterized by dysfunction in processing punishment information. This view is shared by most of the models (Frick & Marsee, 2006; Kiehl, 2006; Lykken, 1995; Patrick, 1994). However, it faces two serious challenges:

a. There are many forms of punishment processing (i.e., partially independent neurocognitive systems process punishment information), and not all are equivalently impaired

in psychopathy. For example, social threats such as another's angry expression are arguably processed by different systems from those that respond to fearful expressions (Blair, 2004). Individuals with psychopathy show no impairment in the processing of angry expressions, but considerable difficulty with processing fearful expressions (Blair et al., 2004a).

Similarly, the use of punishment in stimulus–reinforcement learning (i.e., the ability to form stimulus–punishment associations) is partially separable from the ability to use punishment information in the context of stimulus–response associations (Baxter & Murray, 2002). For example, in the classic passive avoidance learning paradigm, the individual learns that responding to some stimuli gives rise to reward, while responding to others gives rise to punishment. This paradigm can be solved through stimulus–reinforcement associations: Stimulus–reward associations guide the individual toward the good stimuli, while stimulus–punishment associations guide the individual away from the bad stimuli. Individuals with psychopathy show significant impairment in passive avoidance learning and other stimulus–reinforcement learning paradigms (Blair, Leonard, Morton, & Blair, 2006a; Blair et al., 2004b; Newman & Kosson, 1986). However, a modification of the passive learning paradigm can change it to a stimulus–*response* learning task. This occurs if the individual learns that responding to some stimuli gives rise to reward, while *not* responding to gives rise to reward (an alternative version of this modification involves punishment for responding to some and punishment for *not* responding to others). In these variants, there are no good or bad stimuli; whether a stimulus is good (or bad) depends on how the individual responds to that stimulus. Individuals with psychopathy do not show significant impairment on such conditional learning variants of passive avoidance paradigms or on other stimulus–response paradigms, such as object discrimination (Mitchell et al., 2002, 2006a; Newman, Patterson, Howland, & Nichols, 1990);

b. Individuals with psychopathy are impaired in the processing not only of punishment information, but also of reward information (Blair et al., 2006a; Mitchell, Richell, Leonard, & Blair, 2006b; Verona, Patrick, Curtin, Bradley, & Lang, 2004). For example, individuals with psychopathy show impairment in deciding between two objects associated with different levels of reward on the differential reward punishment task (albeit less impairment than when deciding between two objects associated with different levels of punishment) (Blair et al., 2006a). Similarly, individuals with psychopathy show less interference by positive as well as negative emotional distractors on an emotional interruption task (Mitchell et al., 2006b). In addition, individuals with psychopathy show reduced autonomic activity to positive as well as negative emotionally evocative noises (Verona et al., 2004).

One way to reconcile these potentially contradictory findings (not all forms of punishment processing are impaired, and not only punishment processing but also reward processing is impaired) is by reference to the neural level (Blair, 2006). The amygdala is less involved in processing some threats than others. For example, the amygdala shows considerably less responsiveness to angry expressions than to fearful expressions (Whalen, Shin, McInerney, & Rauch, 1998). Similarly, the amygdala is not necessary for the processing of punishment information in the context of stimulus–response tasks, though it is necessary for the formation and use of stimulus–reinforcement associations (Baxter & Murray, 2002). Finally, the amygdala is involved in the processing not only of punishment information, but also of reward information (Baxter & Murray, 2002). In short, the data suggest that psychopathy should be characterized not as an impairment in punishment processing, but as an impairment in those forms of punishment processing mediated by the amygdala.

5. *The impairment in the processing of reinforcement, particularly the reinforcement of others' distress, underlies the difficulties in the socialization of individuals with psychopathy.* Almost all positions agree that the impairment seen in psychopathy interferes with their socialization (Blair, 2001, 2006; Frick & Marsee, 2006; Lykken, 1995; Patrick, 1994). Direct evidence that psychopathy is associated with disruption in the ability to be socialized was first obtained by Paul Frick and colleagues (Wootton, Frick, Shelton, & Silverthorn, 1997). They showed that although parenting type has a significant impact on healthy children (who show increased conduct problems following ineffective parenting), it does not have a significant impact on children who show high levels of the CU traits that constitute the emotional component of psychopathy. This result has been subsequently replicated (Oxford et al., 2003).

Very early viewpoints on psychopathy considered that socialization would be best achieved through the use of punishment-based learning strategies; these positions were based on the idea that psychopathy is due to reduced sensitivity to punishment (Trasler, 1978). The problem with these views is that punishment-based socialization strategies are not particularly effective with respect to socialization (Brody & Shaffer, 1982). This is not surprising, because conditioning theory suggests that the stimulus associated with punishment will be the one that best predicts the punishment. This is unlikely to be the transgression committed at variable lengths in the past, and far more likely to be the individual who actually delivers the punishment.

More recent work has stressed the importance of empathic responses. Work has shown that empathy induction techniques, such as focusing the transgressor's attention on the victim's distress, are effective in socialization (Brody & Shaffer, 1982; Hoffman, 1970). There are considerable data suggesting that the processing of the victim's distress is impaired in individuals with psychopathy (Aniskiewicz, 1979; Blair, Jones, Clark, & Smith, 1997; Blair et al., 2004a; Dolan & Fullam, 2006).

Views That Disagree with the Consensus

Although there is considerable consensus regarding the five points raised above, two positions do not share this consensus: Newman's response set modulation hypothesis and Lynam's inhibition account (Hartung et al., 2002; Whiteside & Lynam, 2001). Both views have their origins in Gray's (1971) behavior inhibition system model, despite the attacks on this model at both the anatomical level (functions ascribed to the septum and hippocampus have been shown to be reliant on the amygdala; LeDoux, 2000) and the cognitive level (functions ascribed to a unitary system, the behavior inhibition system, have been shown to be dissociable; Blair, 2004).

Response set modulation has been defined as the rapid and relatively automatic (i.e., non-effortful or involuntary) shift of attention from the effortful organization and implementation of goal-directed behavior to its evaluation (Newman, 1998; Patterson & Newman, 1993). The impulsivity associated with psychopathy (Miller, Flory, Lynam, & Leukefeld, 2003; Whiteside & Lynam, 2001) has been conceptualized as a lack of premeditation and perseverance. Within this account, this lack of premeditation reflects the inability to inhibit previously rewarded behavior when presented with changing contingencies (Whiteside & Lynam, 2001), whereas the lack of perseverance may be related to disorders that involve the inability to ignore distracting stimuli or to remain focused on a particular task (Whiteside & Lynam, 2001).

Both the response set modulation and impulsivity positions disagree with all the points of consensus outlined above. Neither regards the amygdala or OFC as central to the pathophysiology of psychopathy. Moreover, although both positions acknowledge the existence of emotional deficits in individuals with psychopathy, they do not consider psychopathy to be a primarily emotional disorder. Instead, they consider emotional deficits to be secondary to problems in response set modulation or impulsivity. In addition, they do not consider problems in processing reinforcement, social or otherwise, to be central to the disorder or to explain the difficulties with socialization. Rather, both consider the problem primarily one of attention: Either the individual attends primarily to reward information and cannot shift attention to punishment information (i.e., is incapable of response set modulation; Newman, 1998); or, alternatively, the individual is not attending at all but has been distracted (i.e., exhibits impulsivity; Miller et al., 2003).

It should be noted that such domain-general accounts face considerable difficulties with the psychopathy literature, however. For example, these positions should predict that individuals with psychopathy would be impaired on a broad range of tasks, because many tasks can be considered to involve response set modulation or attention—for example, the intradimensional/extradimensional (ID/ED) and spatial alteration/object alteration (SA/OA) tasks. In these tasks, there are two principal measures. The first of these is the number of response reversal errors/object reversals; these errors occur when subjects fail to change their response from one object to another following a change in reinforcement contingency. The second measure is the number of ED errors/spatial reversals; these errors occur when subject fail to change their response from one semantic category to another (shapes to lines; ED) or from one spatial location to another (SA).

Response/object reversal, ED shifting, and SA all appear to require the inhibition of a previously rewarded behavior/response modulation. Thus, according to an inhibition or response set modulation account, individuals with psychopathy should show impairment not only in response/object reversal, but also in ED shifting and SA. Yet they do not. While individuals with psychopathy do show impairment in response/object reversal, they show no significant difficulty with ED shifting/SA (Blair et al., 2006a; Mitchell et al., 2002). This would be more problematic if the consensus view also had difficulty explaining these data. However, it does not. The functional integrity of the OFC and ventromedial frontal cortex is necessary for successful performance on response reversal and OA tasks (Dias, Robbins, & Roberts, 1996; Freedman, Black, Ebert, & Binns, 1998). As we have argued above, individuals with psychopathy show OFC and ventromedial frontal cortex dysfunction; thus their impairment on response reversal and OA tasks is to be expected. The functional integrity of dorsolateral frontal cortex is necessary for successful performance on ED shifting and SA tasks (Dias et al., 1996; Freedman et al., 1998). As we have argued above, individuals with psychopathy do not appear to have dorsolateral frontal cortex dysfunction; thus their lack of impairment on ED shifting and SA tasks is to be expected.

Conclusions

Individuals with psychopathy represent a subset of individuals with CD/APD whose pathological development is related to a specific form of emotional dysfunction: CU traits. Behaviorally, individuals with psychopathy are marked by an increased risk for reactive *and* instrumental aggression. The increased risk for instrumental aggression is noteworthy because it

is not seen in any other psychiatric condition, and its presence suggests that the pathology of psychopathy interferes with appropriate socialization. The psychopathy construct has been extremely successful in predicting long-term prognosis, particularly in adult samples. But perhaps the greatest importance of this construct for child psychiatrists is that it identifies a sample of children who are unlikely to benefit from standard treatment strategies for children with CD or oppositional defiant disorder (Hawes & Dadds, 2005).

Behavioral genetic work indicates that CU traits are strongly heritable, and that the antisocial behavior of children with these traits is under considerable genetic influence. However, the corresponding molecular work remains in its infancy. Social factors clearly influence the behavioral manifestation of CU traits. They are likely to have a considerable influence on an individual's motives, such as whether he or she needs to offend in order to gain resources or whether a range of alternative strategies is available.

With the exception of the response set modulation and impulsivity positions, a relatively good consensus exists within the field with respect to several main issues. Given this consensus, it is unsurprising that both the response set modulation and impulsivity positions face considerable difficulty accounting for much of the literature. Most positions agree, or at least do not explicitly disagree, on the involvement of the amygdala and OFC in the pathophysiology of psychopathy. Similarly, all positions agree that the disorder is an emotional disorder, and that problems in the processing of social and nonsocial reinforcement are central to the disorder. These emotional impairments are thought to interfere with socialization and to allow the development of the disorder.

There are, of course, points of disagreement within this consensus. A main one concerns the extent of the neural dysfunction, with Kiehl proposing that an extensive array of systems may be impaired in the disorder, and Blair arguing for a far more limited number (the amygdala and ventromedial prefrontal cortex). Moreover, according to Blair, even within those neural regions, the impairment may be confined to some specific functional capacities but leave others intact. It is to be hoped that the triangulation of neuroimaging and neuropsychological data will be able to settle this point of contention.

A secondary point of disagreement concerns the nature of the impairment in reinforcement processing. Many positions have suggested that the impairment is in the processing of punishment information. However, there is growing evidence that this is an inaccurate characterization. The use of punishment information in the context of learning stimulus–response associations appears intact in individuals with psychopathy. Moreover, individuals with psychopathy appear to show problems in using reward information in some contexts. It is possible that the impairment in psychopathy is in the use of reinforcement information in the context of stimulus–reinforcement association formation. A particularly important reinforcement with respect to socialization is, of course, the distress of victims.

Importantly, this growing consensus has useful clinical implications. If psychopathy is understood as a form of emotional disorder, albeit one marked by reduced rather than increased emotional responsiveness, it is possible to consider potential treatment interventions. Many emotional disorders are being treated relatively successfully. These treatments usually involve the suppression of emotional responding; however, such treatments provide interesting suggestions with respect to psychopathy. For example, agents that reduce noradrenergic and, consequently, amygdala activity appear useful in treating posttraumatic stress disorder (Strawn & Geracioti, 2008). Perhaps psychopathy could be treated by agents that increase noradrenergic activity. Given that psychopathy is currently regarded as untreatable, such possibilities provide hope for the future.

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