Cognition–Emotion Interactions in Psychopathy: Implications for Theory and Practice

Arielle R. Baskin-Sommers

University of Wisconsin-Madison

Joseph P. Newman

University of Wisconsin–Madison

Cognitive and affective neuroscientists have made substantial progress specifying mechanisms underlying diverse processes, such as decision making and self-regulation. Increasingly, they also find that cognitive and affective processes are inextricably related and frequently interact to influence behavior (Bishop, Jenkins, & Lawrence, 2007; Davis & Whalen, 2001; Lang, Bradley, & Cuthbert, 1997; Pessoa, McKenna, Gutierrez, & Ungerleider, 2002; Phelps, 2006). Thus, it is not surprising that experimental psychopathologists are integrating these advances and adopting theoretical and methodological approaches that incorporate the complex relationships between cognition and emotion. This is evident in recent research on psychopathy.

Psychopathy is a common and severe psychopathological disorder affecting approximately 1% of the general population and 25% of incarcerated male offenders (Hare, 2006; Neumann & Hare, 2008). Despite psychopathic individuals' good intelligence and an absence of Axis I psychopathology (aside from substance abuse; Hart & Hare, 1989), they display an inability to form genuine relationships with parents, teachers, friends, or lovers; limited and superficial affective processing especially with respect to anticipatory anxiety and remorse; an impulsive behavioral style involving a general failure to evaluate anticipated actions and inhibit the inappropriate ones; and a chronic antisocial lifestyle that entails great costs to society as well as for the affected individual (e.g., incarceration). While both affective and behavioral characteristics are important elements of psychopathy, the affective deficits have traditionally been considered the root cause of the psychopath's problems.

Affective deficits in psychopathy have most often been understood in the context of the low-fear model (Lykken, 1957). However, this traditional view tends to

undervalue the role that cognitive-affective and cortical-subcortical brain interactions have in modulating the etiological and phenotypic manifestation of psychopathy. Thus, more recent theoretical and empirical models of psychopathy attempt to integrate cognitive and affective patterns and their influence on prototypic psychopathic behavior. These models vary in the classification of the controlling (i.e., underlying) variable, but all consider the interactive effects of cognition and emotion on behavior. For example, Blair's violence-inhibition-mechanism-deficit hypothesis (VIM; Blair, 1995) and and Kiehl's paralimbic dysfunction hypothesis (Kiehl, 2006) still regard affective functioning as the primary deficit in psychopathy, but to varying degrees consider the role of cortical and cognitive effects in the disinhibition of psychopaths. Alternatively, Newman's response modulation hypothesis (RMH) suggests that attention is the controlling variable, which in turn interferes with emotion processing. The differences among these models might appear subtle, but they are important in terms of understanding the effects of cognition and emotion on the psychopath's decision-making and self-regulation capabilities, and they have significant implications for the legal system.

Theoretical Perspectives on Psychopathy

In 1957, Lykken proposed that psychopaths were inherently fearless and that their fear deficit interferes with their ability to inhibit inappropriate (i.e., punished) responses (i.e., passive avoidance learning; see also Lykken, 1995). As such, psychopaths display poor fear conditioning (Lykken, 1957), minimal autonomic arousal (i.e., electrodermal response) in anticipation of aversive events (e.g., loud noises, electric shocks; Hare, 1978), problems learning to inhibit punished responses (Newman & Kosson, 1986), and a lack of startle potentiation while viewing unpleasant versus neutral pictures (Patrick, Bradley, & Lang, 1993). Moreover, preliminary neuroimaging evidence suggests that psychopaths display less amygdala (i.e., a brain region once believed to underlie fear processing) activation than controls during aversive conditioning, moral decision making, social cooperation, and memory for emotionally salient words (Birbaumer, Veit, Lotze, et al., 2005; Glenn, Raine, & Schug, 2009; Kiehl et al., 2001; Rilling et al., 2007). However, results from imaging studies focused on the amygdala are equivocal. Other research indicates that the amygdala is hyperreactive when psychopaths view certain emotionally salient scenes (Muller et al., 2003). Developments in neuroscience indicate that the function of the amygdala is more complex than just fear processing and likely plays a significant role in attention and in detecting relevance (Pessoa & Adolphs, 2010; Sander, Grafman, & Zalla, 2003). Furthermore, there is increasing evidence that a much broader range of brain activation deficiencies than just the amygdala are present in psychopaths (Kiehl, 2006). These results suggest that the psychopath's deficit may be more complex than localizing it to the amygdala and fear processing.

While the low-fear model highlights the central role of the amygdala in the psychopath's insensitivity to punishment, Blair's VIM hypothesis suggests that a

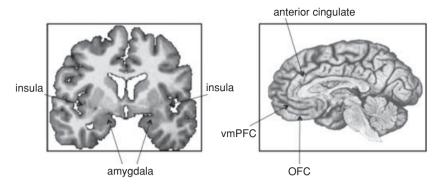


Figure 4.1 Depiction of key brain regions with psychopathy-related dysfunction. OFC = orbital frontal cortex; and vmPFC = ventral medial prefrontal cortex.

failure of fear processing results in poor autonomic arousal and the disinhibition of behavior. According to Blair, the emotion deficit of psychopaths results from a dysfunctional interaction between the amygdala and fear processing with cortical structures (e.g. the orbital frontal cortex [OFC] and ventral medial prefrontal cortex [vmPFC]) and the cognitive processing of nonverbal communications of distress (Blair *et al.*, 1995) (see Figure 4.1).

As a result of amygdala dysfunction, the vmPFC receives fractured information about reinforcement expectancies, an important component of adaptive decision making (Blair, 2008). In addition to the neural abnormalities in the amygdala cited above, reduced activation also has been found in the vmPFC in response to emotional words in emotion memory paradigms (Kiehl *et al.*, 2001) and during aversive conditioning (Birbaumer *et al.*, 2005). Furthermore, a number of laboratory paradigms have demonstrated vmPFC-related deficits in psychopathy. For example, psychopaths demonstrate deficits in reversal learning (Budhani, Richell, & Blair, 2006; Hornak *et al.*, 2004) and in gambling tasks (Bechara, Damasio, Tranel, & Damasio, 1997; Mitchell, Colledge, Leonard, & Blair, 2002; cf. Schmitt, Brinkley, & Newman, 1999).

Nonetheless, much of the support for VIM comes not from work with psychopaths, but from research on patients with lesions to the vmPFC. These individuals are described as having "pseudo-psychopathy" and show a constellation of behaviors similar to those of psychopaths, such as lack of empathy, irresponsibility, and poor decision making (Blumer & Benson, 1975). Moreover, patients with early-onset lesions (e.g. before age 2) exhibit antisocial psychopathic behaviors later

¹ Blair (2006) developed the integrated emotion system (IES) model as an extension of the VIM and low-fear models. Essentially, the IES is a neurocognitive model that suggests a fundamental impairment in the stimulus reinforcement associated with generating affective representations. It identifies key brain regions implicated in this impaired process, such as the amygdala, vmPFC, and OFC. This model is highly similar to the VIM, in terms of both its neural function and empirical support. However, given the practical emphasis in this chapter, we refer primarily to the VIM due to its intimate association with aggression and morality.

in life, such as minor theft, physical assaults, sexual promiscuity, and pathological lying (Anderson *et al.*, 2009). Subsequent clinical and laboratory studies continue to highlight similarities between psychopaths and vmPFC lesion patients (Damasio, Tranel, & Damasio, 1990; Koenigs & Tranel, 2006; Koenigs, Krupke, & Newman, 2010). Moreover, Koenigs *et al.* (2010) recently reported that a subgroup of psychopathic individuals (i.e., primary low-anxious psychopaths) performed similarly to vmPFC lesion patients in Ultimatum and Dictator decision-making games. Specifically, both primary psychopaths and vmPFC lesion patients accepted fewer unfair offers in the Ultimatum game and offered lower amounts to others in the Dictator game. These results support the purported connection between psychopathy and vmPFC dysfunction. Despite this evidence, though, the extent to which other neural systems are also implicated in psychopathy remains an active focus of research.

Kiehl's paralimbic hypothesis implicates a considerably larger variety of brain structures including the amygdala, insula, orbital frontal cortex, ventral striatum, anterior and posterior cingulate, superior temporal cortex and the hippocampus (see Figure 4.1) (Kiehl, 2006). These brain regions are involved in various cognitive and affective processes, such as emotion identification and generation, error monitoring, cognitive control, processing the saliency of stimuli, and attention. Imaging studies directly examining psychopathy suggest that brain regions in the paralimbic and limbic regions are hypo-functioning during language, attention and orienting, and affective processing tasks (Kiehl, 2006; Kiehl *et al.*, 2004).

Similar to Blair's work, research supporting Kiehl's hypothesis clearly receives its strongest support from studies carried out with lesion patients and indirect associations to psychopathy. For example, lesions of the anterior cingulate lead to perseveration (Mesulam, 2000), emotional apathy (Mesulam, 2000), and response inhibition abnormalities (Degos, da Fonseca, Gray, & Cesaro, 1993; Tekin & Cummings, 2002). Correspondingly, psychopathy is associated with perseveration (see review by Newman, 1998), apathy (Cleckley, 1976), and response inhibition abnormalities (Kiehl, Smith, Hare, & Liddle, 2000). Additionally, patients with temporal lobe damage (i.e., amygdala and superior temporal gyrus) show aberrant patterns of psychophysiological brain responses, specifically a late negative event-related potential (ERP) commonly elicited by salient or potentially meaningful stimuli. Psychophysiological studies with psychopaths demonstrate the same pattern of aberrant (i.e., larger) late negative potentials. Kiehl and colleagues (2004) suggest that this pattern in psychopaths reflects difficulties using cognition and in orienting attention to process meaningful information (Kiehl, Hare, McDonald, & Brink, 1999). Given the pervasive abnormalities in brain functioning and performance on tasks highlighted by Kiehl's hypothesis, an interesting observation is that the psychopath's disinhibition is largely influenced by a dysfunction in information processing more generally, rather than simply an inability to experience emotions. Therefore, understanding the role that specific components of information processing, such as attention, have on the psychopathy-related disinhibition is essential.

According to the RMH, attention plays a crucial role in moderating the affective and self-regulatory deficits associated with psychopathy. Response modulation

involves the "temporary suspension of a dominant response set and a brief concurrent shift of attention from the organization and implementation of goal-directed responding to its evaluation" (Patterson & Newman, 1993, p. 717). In the absence of normal response modulation, an individual is prone to ignore crucial contextual information needed to evaluate one's behavior and exercise adaptive self-regulation (MacCoon *et al.*, 2004; Newman, 1998). Thus, psychopaths are oblivious to potentially meaningful peripheral information because they fail to reallocate attention while engaged in goal-directed behavior. This difficulty balancing demands to process goal-directed and peripheral information creates a bias whereby psychopaths are unresponsive to information unless it is a central aspect of their goal-directed focus of attention.

An important implication of the RMH is that the emotion deficit of psychopathic individuals varies as a function of attentional focus. A recent experiment by Newman, Curtin, Bertsch, and Baskin-Sommers (2010) involving fear potentiated startle (FPS) provides striking support for this hypothesis. Of note, existing evidence suggests that FPS is generated via the amygdala (Davis, Falls, Campeau, & Kim, 1993; Grillon, Ameli, Goddard, Woods, & Davis, 1994). The task used in this study required participants to view and categorize letter stimuli that could also be used to predict the administration of electric shocks. Instructions engaged either a goal-directed focus on threat-relevant information (i.e., the color that predicted electric shocks) or an alternative, threat-irrelevant dimension of the letter stimuli (i.e., upper or lower case of the letter or its match or mismatch in a two-back task). The results provided no evidence of a psychopathy-related deficit in FPS under conditions that focused attention on the threat-relevant dimension. However, psychopathy scores were significantly and inversely related to FPS under conditions that required participants to focus on a threat-irrelevant dimension of stimuli (i.e., when threat cues were peripheral). Although the results from Newman *et al.* (2010) provided some of the strongest evidence to date that the fear deficit of psychopaths is moderated by attention, the study did not specify the attentional mechanism underlying this effect. Baskin-Sommers, Curtin, and Newman (2011) specified this attentional-mediated abnormality in a new sample of offenders by measuring FPS in four conditions that crossed attentional focus (threat versus alternative focus) with early versus late presentation of goal-relevant cues. First, the authors replicated the key findings reported by Newman et al. (2010): that psychopaths' deficit in FPS was virtually nonexistent under conditions that focused attention on the threat-relevant dimension of the experimental stimuli (i.e., threat-focus conditions), but was pronounced when threat-relevant cues were peripheral to their primary focus of attention (i.e., alternative-focus conditions). Second, the psychopathic deficit in FPS was only apparent in the early alternative focus condition, in which threat cues were presented after the alternative goal-directed focus was already established. These results corroborate the idea that attention moderates the fearlessness of psychopathic individuals and, moreover, implicate an early attention bottleneck as a proximal mechanism for deficient response modulation in psychopathy (see Newman & Baskin-Sommers, 2011, for discussion of the bottleneck; and see Figure 4.2).

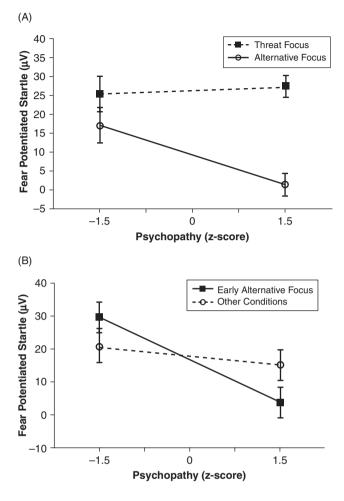


Figure 4.2 Fear-potentiated startle (FPS) as a function of psychopathy ($\pm 1.5~SD$ from the mean) and condition. Panel A: In Newman et~al. (2010), focus of attention significantly moderated the psychopathy effect on FPS. Prisoners high on psychopathy displayed significantly lower FPS than prisoners low on psychopathy in the alternative-focus conditions. High- and low-psychopathy prisoners displayed comparable FPS in the threat-focus condition. Panel B: In Baskin-Sommers et~al. (2011), condition significantly moderated the psychopathy effect on FPS. Prisoners high on psychopathy compared to those low on psychopathy displayed significantly lower FPS in the early-alternative-focus condition, but comparable FPS in the other three conditions.

There is equally clear evidence that the inhibitory deficit considered as the earliest evidence supporting the low-fear model of psychopathy is also moderated by attention. Using a Go/No-Go learning task, Newman and Kosson (1986) examined passive avoidance (i.e., learning from punishment) under reward-and-punishment and punishment-only conditions. When participants were focused on avoiding punishment (punishment-only), there were no group differences in passive avoidance. However, when punishment was peripheral to the primary focus of

earning rewards (reward-and-punishment), psychopaths committed significantly more passive avoidance errors than controls. Thus, psychopaths' deficit in passive avoidance learning, like their FPS deficits, is moderated by their focus of attention (see also Newman, Patterson, Howland, & Nichols, 1990).

The recent evidence for an attention bottleneck that curtails emotion processing is compelling. However, as noted above, others have proposed that psychopaths are less likely to redirect attention to emotion cues because a fundamental amygdalamediated emotion deficit undermines their motivation or capacity to do so (Blair & Mitchell, 2009; Lykken, 1995). While such perspectives acknowledge the importance of attention–emotion interactions, they attribute the attentional abnormalities to a fundamental emotion deficit. In light of these alternative proposals, it is important to consider the substantial evidence demonstrating that psychopaths display similar attentional abnormalities on laboratory tasks involving motivationally neutral information.

In standard versions of the color–word and number Stroop tasks, participants first perceive the conflicting elements and must then reprioritize attention to the appropriate element of the display (MacLeod, 1998). Thus, the quality of one's response depends on the ability to resolve the conflict prior to making a response (i.e., executive functions, such as cognitive control; Botvinick, Braver, Barch, Carter, & Cohen, 2001). Under such conditions, psychopathic and nonpsychopathic individuals show comparable levels of interference (Blair et al., 2006; Hiatt, Schmitt, & Newman, 2004; Smith, Arnett, & Newman, 1992). Conversely, on Stroop-like tasks that facilitate early selection (i.e., attention bottleneck) of goal-relevant information by spatially or temporally separating the incongruent elements of the display, psychopathic individuals display significantly less interference than nonpsychopathic individuals; and paralleling the findings from the classic Stroop design nonpsychopathic individual displayed a significant level of interference (Hiatt et al., 2004; Mitchell et al., 2006, Newman, Schmitt, & Voss, 1997; Vitale, Brinkley, Hiatt, & Newman, 2007). Essentially, for psychopaths the bottleneck is most effective when information is presented in a manner that is visually (i.e., different locations or features) separable, as is the case with the latter Stroop tasks. This early attention bottleneck effectively blocks the processing of conflicting information and reduces the salience of the conflict. Therefore, in certain contexts, psychopathic individuals can effectively screen out the distraction, whereas nonpsychopathic individuals "answer the call for processing" and are influenced by the conflict regardless of experimental context.

Corroborating this interpretation of the Stroop data, Zeier, Maxwell, and Newman (2009) used a modified Erikson flanker task with an attentional cuing manipulation to examine whether an early attention bottleneck is a crucial factor differentiating sensitivity to response conflict in psychopathic individuals. On some trials, pretrial cuing was used so that participants could orient attention to the location of the task-relevant target before the target and distracting flanker stimuli were presented. On other trials, the pretrial cues directed attention to both the target and distractor locations. Whereas psychopathic participants displayed significantly less interference than controls in the former condition, they displayed nonsignificantly more interference in the latter condition. Combined, these studies show that psychopathic

participants are significantly less sensitive to information if it is peripheral to a preestablished focus of goal-directed behavior. Moreover, the fact that this abnormality applies to affectively neutral as well as affectively significant peripheral information implicates an early attention bottleneck that undermines the processing of goalincongruent cues regardless of affective significance (see also Hiatt *et al.*, 2004; Jutai & Hare, 1983; Mitchell *et al.*, 2006; Vitale *et al.*, 2007).

Notably though, to date, no imaging studies have directly examined the processes implicated in response modulation. Thus, unlike other psychopathy models, there currently is no direct neural basis for the RMH. However, many of the brain regions identified by VIM and the paralimbic hypothesis are known to be important in attention (e.g. vmPFC and ACC) and directly impact the functioning of the amygdala and other emotion-related brain regions (e.g. insula and nucleus accumbens).² The combination of studies presented provides strong evidence that psychopathic individuals are characterized by an abnormal cognition-emotion interaction, specifically guided by an abnormal early attention bottleneck that effectively precludes response inhibition, conflict monitoring, affective processing, and self-regulation. In light of reliable evidence that attention moderates the inhibitory (Zeier et al., 2009), affective (Baskin-Sommers et al., 2011, Newman et al., 2010), and self-regulation (Newman & Kosson, 1986) deficits associated with psychopathy, we believe that it is crucial for future research to further specify the cognitive-affective networks responsible for these deficits in psychopathic individuals. Further exploring cognition-emotion interactions has potentially crucial implications for understanding the types of behaviors commonly displayed by psychopaths, how those behaviors differ from other disinhibitory groups, and other practical issues related to the legal processing and treatment of psychopaths.

Practical Issues in Psychopathy

cognition—emotion interactions establish patterns for thinking, feeling, and acting. These are central aspects that figure prominently in poor inhibition and antisocial behavior. An understanding of the functioning and consequence of these interactions provides a nuanced view of the source of disinhibited behavior and has important implications for key legal issues. In this section we will discuss issues related to aggression, judicial practice (e.g. culpability and post-incarceration release), and treatment.

Aggression

To illustrate the relevance of cognition—emotion interactions in the context of real-world behaviors, we will consider instrumental aggression versus reactive aggression.

² The neural structures listed here follow from what has been reported in existing psychopathy-related imaging studies and are not necessarily the neural regions responsible for response modulation/attention bottleneck. Proposals for the neural underpinnings of response modulation are outlined in Newman & Baskin-Sommers (2011).

Simply stated, psychopaths are distinguished by their tendency to display instrumental aggression, whereas other disinhibited individuals (e.g. antisocial personality disorder, conduct disorder, and trait impulsivity) are prone to reactive aggression (Blair, 2001; Glenn & Raine, 2009; Sprague & Verona, 2010). Although these types of aggression are not mutually exclusive, it is the differential susceptibility that distinguishes between these types of individuals and highlights the unique contribution of cognition–emotion interactions to psychopathy.

On the one hand, instrumental aggression is deliberate and goal directed, and, as already indicated, is typically linked to psychopathy (Blair, 2001; Cornell *et al.*, 1996). Instrumental aggression is premeditated, suggesting a conscious perception of goals, and is not typically preceded by a burst of emotional reactivity. Specifically with regard to psychopathy, its association with instrumental aggression also plays out in terms of their patterns of criminal behavior (Glenn & Raine, 2009). Psychopathic offenders are much more likely to commit a violent crime based on the motivation for material gain than nonpsychopathic offenders (45.2% versus 14.6%; Williamson, Hare, & Wong, 1987). They are twice as likely as nonpsychopathic offenders to commit instrumental (premeditated) homicides; indeed, 93.3% of homicides committed by psychopaths compared to 48% of nonpsychopaths are instrumental (Woodworth & Porter, 2002).

Case example: A landlord threatened to evict a man from his apartment for not paying rent for 6 months straight. The man could not pay, so he decided to beat and tie up the landlord. As the man was leaving, he told the landlord he was catching the bus to Iowa and issued a final threat. The man was arrested at the bus station.

Some explanations for instrumental aggression suggest that it is a function of poor socialization and an inability to recognize or experience emotions. However, the abnormal cognitive-emotional interaction (i.e., early attention bottleneck) identified by Baskin-Sommers *et al.* (2011) may also impact instrumental aggression. Inhibitory emotion cues are generally peripheral to one's primary goal (e.g. attaining respect, money, or drugs, robbing a bank). An early attention bottleneck would be expected to preclude the processing of emotions when psychopathic individuals are engaged in goal-directed aggression. Thus, from the point of view of the RMH, psychopathic individuals do not engage in aggression because of innate callousness, but they are callously oblivious to information that is not directly and immediately related to their goal. The case example presented above highlights the lack of perspective this type of attention abnormality can create. Not only did the man believe that the best way to handle late rent was to assault the landlord, but also he was so focused on getting out of paying the rent that he told the landlord where he was going to escape.

On the other hand, reactive aggression is often in response to a frustration or threat (e.g., in response to an insult, or in the context of a heated argument). It is often motivated by the situational context.

Case example: Jason started going off about Paul's baby mama. He was calling her a slut and told Paul that the baby wasn't his. All of a sudden Paul "couldn't take it anymore" and just went off on Jason. He took a pipe that was on the ground and whacked Jason across the face. When Paul fell to the ground, Jason kept on wailing on him and screaming, "Don't talk about my baby mama."

Models for reactive aggression assume roles for higher-order cognitive functions (e.g. cognitive control) and emotion systems (Blair, 2001; Dougherty *et al.*, 2004). In essence, the combination of poor inhibitory control and an inability to process and manage emotions effectively, particularly frustration, results in the disinhibited expression of aggressive responses (Davidson, Putnam, & Larson, 2000). Brain regions, such as the orbitofrontal cortex (OFC) and the amygdala, identified by Kiehl's paralimbic hypothesis and Blair's VIM, primarily contribute to reactive aggression. Two functions of the OFC include management of reward expectations (and the frustration that coincides when reward is expected but not received) and social cognition (Blair, 2003). As noted, individuals with lesions to this region are regarded as having "pseudopsychopathy" and display reactive aggression, but have not been associated with instrumental aggression (Blair, 2007; Glenn & Raine, 2009). Thus, in the case example presented above, it is possible that Paul has an impairment in the amygdala and/or OFC region that interferes with his management of frustration and executive thinking.

This characterization based on types of aggression highlights the unique motivational and behavioral style that distinguishes psychopaths from other offenders. Psychopaths' cognition—emotion interaction yields an obliviousness that induces a narrowed focus and a tendency to act on premeditated and motivated aggression. Other disinhibited individuals, who primarily engage in reactive aggression, are characterized by a cognition—emotion interaction that results in a heightened state of arousal, a failure of inhibitory or cognitive control, and a tendency toward volatile and reckless aggression. Understanding the differences between these two types of cognition—emotion interactions, as they relate to aggression and other behaviors, is important for thinking about legal issues as they implicate different pathways and motivations for disinhibition.

Judicial Practice: Culpability and Post-incarceration Release

In light of evidence presented in this chapter regarding the cognitive and affective mechanisms that give rise to psychopathy, it should be clear that the constellation of features that comprise psychopathy makes these individuals particularly difficult to handle within the legal system. As already discussed, psychopaths rarely learn from punishment or experience (i.e., passive avoidance learning). The RMH suggests that the psychopathy-related attention deficit reduces online conflict processing, specifically of peripheral and secondary information (e.g., see the Stroop findings in this chapter). This attentional style precludes the reallocation of attention to important information and subsequent reflection. Thus, if psychopaths do not learn from the consequences of their behavior and are known to have neural dysfunction, then pertinent questions related to culpability arise.

According to the penal codes of most states *culpability* defines a person's actions as "purposeful," "knowing," "reckless," or "negligent" (Fabian, 2010). Culpability is also usually linked to the capacity to control behavior (Siegel & Douard, in press). Frequently, this is demonstrated by the capacity to appreciate the criminality of the offense and is assessed using neuropsychological testing. With regard to psychopathy, the issue of culpability is complex. On the one hand, there are known neural dysfunctions associated with psychopathy. These dysfunctions occur in regions of the brain that are fundamental to adaptive behavior. Additionally, evidence suggests that psychopaths don't learn from punishment or the consequences of their behavior, engage in self-defeating behavior, and thus have a deficit in decision-making and regulatory capabilities.

On the other hand, psychopathic offenders rarely display deficits in the neuropsychological testing geared toward assessing capacity and cognitive dysfunction (Blair et al., 2006; Hart, Forth, & Hare, 1990; Hiatt et al., 2004; Munro et al., 2007; Smith et al., 1992). In fact, psychopathic offenders are generally characterized as having "good intelligence" (Cleckley, 1976) and suffer no impairment of consciousness (i.e., knowing right from wrong; Schoop & Slain, 2000). Additionally, empirical evidence related to the RMH suggests that the psychopathy-related deficit in inhibitory learning and emotion responding can appear and disappear depending on the context (e.g., see Newman et al., 2010, and Baskin-Sommers et al., 2011, FPS findings). That is, it is possible for psychopaths to regulate, but it may be more difficult for them to do so in demanding contexts. Lastly, the imaging data within the field of psychopathy have not provided a clear consensus on the neurological root of the disorder; nor have they specified the dysfunction. Functional imaging data associate psychopathy with abnormal, but not necessarily diminished, activity in all four lobes of the cortex (frontal, temporal, parietal, and occipital), as well as several subcortical structures (e.g., amygdala and hippocampus). Given these issues, at this time there is no definitive evidence that individuals identified as psychopaths are in some way precluded from culpability. However, in order to make headway on this issue, both the legal and psychological communities first need to be clearer on what constitutes evidence for culpability (e.g. abnormality on a scan, evidence of trauma). Moreover, as theory-driven neuroimaging research grows, the specificity of a potential neural dysfunction and its relationship to criminal capacity and behavior will increase; providing the justice system with a more systematic understanding of how cognition-emotion interactions influence culpability.

Aside from trial-related issues, the specific traits of psychopaths also raise concerns for post-incarceration release. Psychopaths comprise 15–25% of the prison population, commit more than twice as many crimes as other offenders, and are approximately five times more likely to reoffend than nonpsychopathic offenders. Not only are psychopaths highly recidivistic (Cima-Knijff & Raine, 2009; Hare & Neumann, 2009), but also they are particularly prone to violent recidivism (Tengstrom, Grann, Langstrom, & Kullgren, 2000). In a study by Quinsey, Rice, and Harris (1995), 80% of the psychopathic offenders committed multiple violent crimes over a 6-year period post-release. Thus, how should the justice system manage these offenders? In order to answer this question, two potential avenues for further research and exploration exist: one pertaining to the use of civil commitment, and another involving deficit-targeted treatment (reviewed in the "Treatment" section of this chapter).

Civil commitment refers to the post-incarceration institutionalization of people who are mentally ill or have a mental abnormality that substantially reduces their capacity for self-control. The process of civil commitment involves various risk assessments, treatment, and socialization skill-building strategies. Its guiding principles are related to issues of dangerousness and risk of recidivism. Overall, civil commitment provides an alternative to release for those who consistently demonstrate that they don't function appropriately within societal guidelines; but it is not without controversy. Those selected for civil commitment have already served their time, but are deemed too high risk to reintegrate into the community. Essentially, civilly committed individuals are forced to serve more time than their sentence, albeit not in a prison but in a treatment facility. To date, civil commitment primarily has been applied to sexual offenders (of which 25% are diagnosed as psychopaths; Jackson, 2008). However, the tenets of civil commitment (e.g. to reduce recidivism and identify high-risk individuals) appear to be applicable to a broader group of psychopaths. Psychopaths are at a high risk for violence (Harris, Rice, & Quinsey, 1993) and recidivism (Cima-Knijff & Raine, 2009; Hare & Neumann, 2009), often fail to learn from their behavior, and have deficits in decision making; thus, an option of civil commitment to prevent these individuals from reintegrating into society post-release may be a useful strategy. One criticism of this is that civil commitment would just delay the inevitable release of the psychopath. However, research suggests that psychopathy, particularly the impulsive and antisocial components of psychopathy, decreases with age (Harpur & Hare, 1994). Therefore monitoring these individuals for an extended period of time might naturally diminish the behavioral manifestation of their syndrome. Another important issue, though, is that during civil commitment there is a strong focus on treatment. This focus on treatment is a potential shortcoming of the application of civil commitment to psychopathic individuals, as many treatments targeting psychopathy frequently have been in vain (Hare & Neumann, 2009; see Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003, for a discussion of treatment utility based on psychopathic subtypes).

Treatment

To date, many of the canonical behavioral and cognitive treatments have proven ineffective with psychopaths. According to Hare (2006), "Some of the most popular prison treatment and socialization programs may actually make psychopaths worse than they were before ... group therapy and insight oriented programs help psychopaths develop better ways of manipulating, deceiving and using people but do little to help them understand themselves" (p. 717). Supporting this notion, not only are psychopaths more likely to reoffend, but also after treatment they reoffend at a greater rate and more violently than nontreated psychopaths (Hughes, Hughes, Hollin, & Champion 1997; Ogloff, Wong, & Greenwood, 1990; O'Neil, Lidz, & Heilbrun, 2003; Rice, Harris, & Cormier, 1992). In 2009, Hare and Neumann stated that "unlike most other offenders, people with psychopathy appear to suffer little personal distress, see little wrong with their attitudes and behavior, and seek treatment only when it is in their best interests to do so, such as when seeking probation or parole" (p. 798). This characterization of the psychopath's intentions with regard to treatment is commonly held by treatment providers, who typically consider psychopaths to be untreatable. Nonetheless, with advancing knowledge regarding the cognition-emotion interactions that undermine the psychopaths' ability to selfregulate, new treatment options are on the horizon (Hare & Neumann; 2009; Skeem et al., 2003; Wallace, Schmitt, Vitale, & Newman, 2000; Wallace, Vitale, & Newman,

Among those treatment possibilities being explored currently is that of cognitive remediation. *Cognitive remediation* refers to an approach that trains the individual in particular cognitive skills, such as paying attention to contextual cues, applying working memory, and sustained attention (Klingberg, 2010; Wykes & van der Gaag, 2001). In healthy adults, Klingberg and colleagues have shown that working memory training not only improves overall working memory capacity, but also changes the functioning of dopamine neurotransmission and brain plasticity (McNab *et al.*, 2009). Research on disorders with known cognitive abnormalities, such as attention-deficit hyperactivity disorder and schizophrenia, has started to assess the efficacy of cognitive remediation as a treatment strategy (Stevenson, Whitmont, Bornholt, Livesey, & Stevenson; 2002; Wykes *et al.*, 2003). For example, the application of working memory training has demonstrated a durable improvement in memory (Wykes *et al.*, 2007).

Given the attentional abnormalities associated with psychopathy, particularly those highlighted by the RMH, it may be possible to develop cognitive remediation treatments that target the specific cognitive deficits of psychopaths. Psychopathic individuals are oblivious to inhibitory and punishment cues (Newman & Kosson, 1986) that contraindicate ongoing goal-directed behavior (i.e., mismatch information; Hiatt *et al.*, 2004), and emotional information that modulates responding in others (Baskin-Sommers *et al.*, 2011; Newman *et al.*, 2010). Despite increasing evidence that these problems are linked to abnormal neurological and biochemical

functioning (e.g., Blair *et al.*, 2006; Buckholtz *et al.*, 2010; Kiehl, 2006), the psychopath's failure to attend to contextual information may nevertheless be conceptualized as specific skills deficits. Thus, they may also improve with explicit practice and skill building, and such change may even be reflected in brain-related measures (as seen in Klingberg *et al.*, 2009). Along these lines, Newman and collaborators have designed cognitive interventions that are believed to target the specific cognitive, affective, decision-making, and self-regulation deficits associated with psychopathy. The potential advantage of such a treatment is that it is based on an etiological theory that targets deficits uniquely associated with psychopathy rather than assuming that these individuals function like other criminals.

Conclusion

In sum, this chapter provides evidence that psychopathy is associated with unique cognition-emotion interactions that guide behavior. Many of the theoretical models presented in the first section provide a framework for psychopathy that recognizes the inextricable link between cognition-emotion and behavior. Blair's VIM model works to specify the emotional roots of psychopathy, whereas Newman's RMH focuses on specifying the attentional abnormalities. Importantly, these models and Kiehl's paralimbic hypothesis are not mutually exclusive. Some of the brain structures identified by Blair and Kiehl may be very important for the attentional functions identified by Newman and colleagues. However, more work is needed to specify the nature of these cognition-emotion interactions and characterize the primary neural underpinnings associated with them. In order to draw more definitive conclusions about cognition-emotion interactions and their relation to legal issues, future research will need to be carefully designed to parse and specify the capabilities and motivations of psychopaths. As research further clarifies the neural and behavioral patterns in psychopathy, specific recommendations for the justice system (e.g., culpability) and treatment (e.g., cognitive remediation) will naturally emerge.

References

- Baskin-Sommers, A. R., Curtin, J. J., & Newman, J. P. (2011). Specifying the attentional selection that moderates the fearlessness of psychopathic offenders. *Psychological Science* 22(2), 226–234.
- Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy, *Science*, 275, 1293–1295.
- Birbaumer, N., Veit, R., Lotze, M., Erb, M., Christiane, H., Grodd, W., & Flor, H. (2005). Fear conditioning in psychopathy: A functional magnetic resonance imaging study. *Archives of General Psychiatry*, *62*, 799–805.
- Bishop, S. J., Jenkins, R., & Lawrence, A. D. (2007). Neural processing of fearful faces: Effects of anxiety are gated by perceptual capacity limitations. *Cerebral Cortex*, *17*, 595–603.
- Blair, R. J. R. (1995). A cognitive developmental approach to morality: Investigating the psychopathy. *Cognition*, *57*, 1–29.

- Blair, R. J. R. (2001). Neuro-cognitive models of aggression, the antisocial personality disorders, and psychopathy. *Journal of Neurology, Neurosurgery, & Psychiatry*, 71, 727–731.
- Blair, R. J. R. (2003). Neurobiological basis of psychopathy. *British Journal of Psychiatry*, 182, 5–7.
- Blair, R. J. R. (2006) Subcortical brain systems in psychopathy: The amygdala and associated structures. In C. J. Patrick (Ed.), *Handbook of psychopathy* (pp. 296–312). New York: Guilford Press.
- Blair, R. J. (2007). Dysfunctions of medial and lateral orbitofrontal cortex in psychopathy. Annals of the New York Academy of Sciences, 1121, 461–479.
- Blair, R. J. (2008). The amygdala and ventromedial prefrontal cortex: Functional contributions and dysfunction in psychopathy. *Philosophical Transactions of the Royal Society: Biological Sciences*, 363, 2557–2565.
- Blair, R. J. R., & Mitchell, D. V. G. (2009). Psychopathy, attention and emotion. *Psychological Medicine*, 39, 543–555.
- Blair, K. S., Newman, C., Mitchell, D. G., Richell, R. A., Leonard, A., Morton, J., & Blair, R. J. (2006). Differentiating among prefrontal substrates in psychopathy: Neuropsychological test findings. *Neuropsychology*, 20, 153–165.
- Blair, R. J. R., Sellars, C., Strickland, I., Clark, F., Williams, A. O., Smith, M., & Jones, L. (1995). Emotion attributions in the psychopath. *Personality and Individual Differences*, 19, 431–437.
- Blumer, D., & Benson, D. F. (1975). Personality changes with frontal and temporal lesions. In D. F. Benson & D. Blumer (Eds.), *Psychiatric aspects of neurological disease*. New York: Stratton.
- Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., Cohen, & J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, *108*, 624–652.
- Buckholtz, J. W., Treadway, M. T., Cowan, R., Benning, S. D., Li, R., Ansari, M. S., & Zald, D. H. (2010). Mesolimbic dopamine reward system hypersensitivity in individuals with psychopathic traits. *Nature Neuroscience*, *13*, 419–421.
- Budhani, S., Richell, R. A., & Blair, R. J. (2006) Impaired reversal but intact acquisition: Probabilistic response reversal deficits in adult individuals with psychopathy. *Journal of Abnormal Psychology*, 115, 552–558.
- Cima-Knijff, M. J., & Raine, A. (2009). Distinct characteristics of psychopathy relate to different subtypes of aggression. *Personality and Individual Differences*, 47, 835–840.
- Cleckley, H. (1976). The mask of sanity (5th ed.). St. Louis, MO: Mosby.
- Cornell, D. G., Warren, J., Hawk, G., Stafford, E., Oram, G., & Pine, D. (1996). Psychopathy in instrumental and reactive violent offenders. *Journal of Consulting and Clinical Psychology*, 64, 783–790.
- Damasio, A. R., Tranel, D., & Damasio, H. (1990). Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli. *Behavioural Brain Research*, 41, 81–94.
- Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation: A possible prelude to violence. *Science*, *289*, 591–594.
- Davis, M., Falls, W. A., Campeau, S., & Kim, M. (1993). Fear-potentiated startle: A neural and pharmacological analysis. *Behavioural Brain Research*, *58*, 175–198.
- Davis, M., & Whalen, P. (2001). The amygdala: Vigilance and emotion. *Molecular Psychiatry*, 6, 13–34.

- Degos, J. D., da Fonseca, N., Gray, F., & Cesaro, P. (1993). Severe frontal syndrome associated with infarcts of the left anterior cingulate gyrus and the head of the right caudate nucleus: A clinico-pathological case. *Brain*, *116*, 1541–1548.
- Dougherty, D. D., Rauch, S. L., Deckersbach, T., Marci, C., Loh, R., Shin, L. M., . . . Fava, M. (2004). Ventromedial prefrontal cortex and amygdala dysfunction during an anger induction positron emission tomography study in patients with major depressive disorder with anger attacks. *Archives of General Psychiatry*, *61*, 795–804.
- Fabian, J. M. (2010). Neuropsychological and neurological correlates in violent and homicidal offenders: A legal and neuroscience perspective. *Aggression and Violent Behavior*, 15, 209–223.
- Glenn, A. L., & Raine, A. (2009). Psychopathy and instrumental aggression: Evolutionary, neurobiological, and legal perspectives. *International Journal of Law & Psychiatry*, 32, 253–258.
- Glenn, A. L., Raine, A., & Schug, R. A. (2009). The neural correlates of moral decision-making in psychopathy. *Molecular Psychiatry*, *14*, 5–6.
- Grillon, C., Ameli, R., Goddard, A., Woods, S. W., & Davis, M. (1994). Baseline and fear-potentiated startle in panic disorder patients. *Biological Psychiatry*, *35*, 431–439.
- Hare, R. D. (1978). Electrodermal and cardiovascular correlates of psychopathy. In R. D. Hare & D. Schalling (Eds.), *Psychopathic behavior: Approaches to research* (pp. 107–143). Chichester, UK: John Wiley & Sons.
- Hare, R. D. (2006). Psychopathy: A clinical and forensic overview. Psychiatric Clinics of North America, 29, 709–724.
- Hare, R. D. (1996). Psychopathy: A clinical construct whose time has come. *Criminal Justice and Behavior*, 23, 25–54.
- Hare, R. D., & Neumann, C. S. (2009). Psychopathy: Assessment and forensic implications. *Canadian Journal of Psychiatry*, 54, 791–802.
- Harpur, T. J., & Hare, R. D. (1994). Assessment of psychopathy as a function of age. *Journal of Abnormal Psychology*, 103, 604–609.
- Harris, G. T., Rice, M. E., & Quinsey, V. L. (1993). Violent recidivism of mentally disordered offenders: The development of statistical prediction instrument. *Criminal Justice and Behavior*, 20, 315–335.
- Hart, S. D., Forth, A. E., & Hare, R. D. (1990). Performance of criminal psychopaths on selected neuropsychological tests. *Journal of Abnormal Psychology*, 99, 374–379.
- Hart, S. D., & Hare, R. D. (1989). Discriminant validity of the Psychopathy Checklist in a forensic psychiatric population. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, 1, 211–218.
- Hiatt, K. D., Schmitt, W. A., & Newman, J. P. (2004). Stroop tasks reveal abnormal selective attention in psychopathic offenders, *Neuropsychology*, *18*, 50–59.
- Hornak, J., O'Doherty, J., Bramham, J., Rolls, E. T., Morris, R. G., Bullock, P. R., & Polkey, C. E. (2004). Reward-related reversal learning after surgical excisions in orbito-frontal or dorsolateral prefrontal cortex in humans. *Journal of Cognitive Neuroscience*, 16, 463–478.
- Hughes, G., Hughes, T., Hollin, C., & Champion, H. (1997). First-stage evaluation of a treatment programme for personality disordered offenders. *Journal of Forensic Psychiatry*, 8, 515–527.
- Jutai, J., & Hare, R. D. (1983). Psychopathy and selective attention during performance of a complex perceptual-motor task. *Psychophysiology*, *20*, 146–151.

- Kiehl, K. A. (2006). A cognitive neuroscience perspective on psychopathy: Evidence for paralimbic system dysfunction. *Psychiatry Research*, 142, 107–128.
- Kiehl, K. A., Hare, R. D., McDonald, J. J., & Brink, J. (1999). Semantic and affective processing in psychopaths: An event-related potential study. *Psychophysiology*, *36*, 765–774.
- Kiehl, K. A., Smith, A. M., Hare, R. D., & Liddle, P. F. (2000). An event-related potential investigation of response inhibition in schizophrenia and psychopathy. *Biological Psychiatry*, 48(3), 210–221.
- Kiehl, K. A., Smith, A. M., Hare, R. D., Mendrek, A., Forster, B. B., Brink, J., & Liddle, P. F. (2001). Limbic abnormalities in affective processing by criminal psychopaths as revealed by functional magnetic resonance imaging. *Biological Psychiatry*, 50, 677–684.
- Kiehl, K., Smith A., Mendrek A., Forster B., Hare R., & Liddle, P. (2004) Temporal lobe abnormalities in semantic processing by criminal psychopaths as revealed by functional magnetic resonance imaging. *Psychiatry Research: Neuroimaging*, 130, 27–42.
- Klingberg, T., & McNab, F. (2009). Working memory remediation and the D1 receptor. *American Journal of Psychiatry*, 166(5), 515–516.
- Koenigs, M., Kruepke, M., & Newman, J. P. (2010). Economic decision-making in psychopathy: A comparison with ventromedial prefrontal lesion patients. *Neuropsychologia*, 48, 2198–2204.
- Koenigs, M., & Tranel, D. (2006). Pseudopsychopathy: A perspective from cognitive neuroscience. In D. H. Zald & S. L. Rauch (Eds.), *The orbitofrontal cortex* (pp. 597–619). New York: Oxford University Press.
- Klingberg, T. (2010). Training and plasticity of working memory. *Trends in Cognitive Science*, 14, 317–324.
- Lang, P. J., Bradley, M. M., & Cuthbert, M. M. (1997). Motivated attention: Affect, activation and action. In P. J. Lang, R. F. Simons, & M. T. Balaban (Eds.), Attention and orienting: Sensory and motivational processes (pp. 97–135). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Lykken, D. T. (1957). A study of anxiety in the sociopathic personality. *Journal of Abnormal and Social Psychology*, *55*, 6–10.
- Lykken, D. T. (1995). *The antisocial personalities*. Hillsdale, NJ: Lawrence Erlbaum Associates. MacCoon, D. G., Wallace, J. F., & Newman, J. P. (2004). Self-regulation: The context-appropriate allocation of attentional capacity to dominant and non-dominant cues. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of self-regulation: Research, theory, and applications* (pp. 422–446). New York: Guilford.
- MacLeod, C. M. (1998). Training on integrated versus separated Stroop tasks: The progression of interference and facilitation. *Memory & Cognition*, 26, 201–211.
- McNab, F., Varrone, A., Farde, L., Jucaite, A., Bystritsky, P., Forssberg, H., & Klingberg, T. (2009). Changes in cortical dopamine D1 receptor binding associated with cognitive training. *Science*, *323*, 800–802.
- Mesulam, M. (2000). Attentional networks, confusional states and neglect syndromes. In M. Mesulam (Ed.), *Principles of behavioral and cognitive neurology* (pp. 174–256). New York: Oxford University Press.
- Mitchell, D. G., Colledge, E., Leonard, A., & Blair, R. J. (2002). Risky decisions and response reversal: Is there evidence of orbitofrontal cortex dysfunction in psychopathic individuals? *Neuropsychologia*, 40, 2013–2022.
- Muller, J. L., Sommer, M., Wagner, V., Lange, K., Taschler, H., Roder, C. H., *et al.* (2003). Abnormalities in emotion processing within cortical and subcortical regions in

- criminal psychopaths: Evidence from a functional magnetic resonance imaging study using pictures with emotional content. *Biological Psychiatry*, *54*, 152–162.
- Munro, G. E. S., Dywan, J., Harris, G. T., Mckee, S., Unsal, A., & Segalowitz, S. J. (2007). ERN varies with degree of psychopathy in an emotion discrimination task. *Biological Psychology*, 76, 31–42.
- Neumann, C. S., & Hare, R. D. (2008). Psychopathic traits in a large community sample: Links to violence, alcohol use, and intelligence. *Journal of Consulting and Clinical Psychology*, 76, 893–899.
- Newman, J. P. (1998). Psychopathic behavior: An information processing perspective. In D. J. Cooke, R. D. Hare, & A. Forth (Eds.), *Psychopathy: Theory, research and implications for society* (pp. 81–104). Dordrecht, The Netherlands: Kluwer Academic Publishers.
- Newman, J. P., & Baskin-Sommers, A. R. (2011). Early selective attention abnormalities in psychopathy: Implications for self-regulation. In M. Poser (Ed.). *Cognitive neuroscience of attention* (pp. 421–440). New York: Guilford.
- Newman, J. P., Curtin, J. J., Bertsch, J. D., & Baskin-Sommers, A. R. (2010). Attention moderates the fearlessness of psychopathic offenders. *Biological Psychiatry*, *67*, 66–70.
- Newman, J. P., & Kosson, D. S. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of Abnormal Psychology*, 95, 257–263.
- Newman, J. P., Patterson, C. M., Howland, E. W., & Nichols, S. L. (1990). Passive avoidance in psychopaths: The effects of reward. *Personality and Individual Differences*, 11, 1101–1114.
- Newman, J. P., Schmitt, W. A., & Voss, W. (1997). The impact of motivationally neutral cues on psychopathic individuals: Assessing the generality of the response modulation hypothesis. *Journal of Abnormal Psychology*, *106*, 563–575.
- Ogloff, J., Wong, S., & Greenwood, A. (1990). Treating criminal psychopaths in a therapeutic community program. *Behavioral Sciences & the Law, 8*, 181–190.
- O'Neil, M., Lidz, V., & Heilbrun, K. (2003). Adolescents with psychopathic characteristics in a substance abusing cohort: Treatment process and outcomes. *Law and Human Behavior*, 27, 299–313.
- Patrick, C. J., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath: Startle reflex modulation. *Journal of Abnormal Psychology*, 102, 82–92.
- Patterson, C. M., & Newman, J. P. (1993). Reflectivity and learning from aversive events: Toward a psychological mechanism for the syndromes of disinhibition. *Psychological Review*, 100, 716–736.
- Pessoa, L., & Adolphs, R. (2010). Emotion processing and the amygdala: From a "low road" to "many roads" of evaluating biological significance. *Nature Reviews Neuroscience*, 11, 773–782.
- Pessoa, L., McKenna, M., Gutierrez, E., & Ungerleider, L. G. (2002). Neural processing of emotional faces requires attention. *Proceedings of the National Academy of Sciences, USA*, 98, 683–687.
- Phelps, E. A. (2006). Emotion and cognition: Insights from studies of the human amygdala. *Annual Review of Psychology*, 24, 27–53.
- Quinsey, V. L., Rice, M. E., & Harris, G. T. (1995). Actuarial prediction of sexual recidivism. *Journal of Interpersonal Violence*, *10*, 85–105.
- Rice, M., Harris, G., & Cormier, C. (1992). An evaluation of a maximum security therapeutic community for psychopaths and other mentally disordered offenders. *Law and Human Behavior*, *16*, 399–412.
- Rilling, J. K., Glenn, A. L., Jairam, M. R., Pagnoni, G., Goldsmith, D. R., Elfenbein, H. A., & Lilienfeld, S. O. (2007). Neural correlates of social cooperation

- and non-cooperation as a function of psychopathy. *Biological Psychiatry*, 61, 1260–1271.
- Sander, D., Grafman, J., & Zalla, T. (2003). The human amygdala: An evolved system for relevance detection. *Reviews in the Neurosciences*, 14, 303–316.
- Schmitt, W. A., Brinkley, C. A., & Newman, J. P. (1999). Testing Damasio's somatic marker hypothesis with psychopathic individuals: Risk takers or risk averse? *Journal of Abnormal Psychology*, *108*, 538–543.
- Siegel, A., & Douard, J. (in press). Who's flying the plane: Serotonin levels, aggression and free will. *International Journal of Law and Psychiatry*.
- Skeem, J. L, Poythress, N., Edens, J. F., Lilienfled, S. O., & Cale, E. M. (2003). Psychopathic personality or personalities? Exploring potential variants of psychopathy and their implications for risk assessment. *Aggression and Violent Behavior*, *8*, 513–546.
- Smith, S. S., Arnett, P. A., & Newman, J. P. (1992). Neuropsychological differentiation of psychopathic and nonpsychopathic criminal offenders. *Personality and Individual Differences*, 13, 1233–1245.
- Sprague, J., & Verona, E. (2010). Emotional conditions disrupt behavioral control among individuals with dysregulated personality traits. *Journal of Abnormal Psychology*, 119, 409–419.
- Stevenson, C. S., Whitmont, S., Bornholt, L., Livesey, D., & Stevenson, R. J. (2002). A cognitive remediation programme for adults with attention deficit hyperactivity disorder. *Australian and New Zealand Journal of Psychiatry*, *36*, 610–616.
- Tekin, S., & Cummings, J. (2002). Frontal-subcortical neuronal circuits and clinical neuropsychiatry: An update. *Journal of Psychosomatic Research*, *53*, 647–654.
- Tengstrom, A., Grann, M., Langstrom, N., & Kullgren, G. (2000). Psychopathy (PCL-R) as a predictor of violent recidivism among criminal offenders with schizophrenia. *Law and Human Behavior*, 24, 45–58.
- Vitale, J. E., Brinkley, C. A., Hiatt, K. D., & Newman, J. P. (2007). Abnormal selective attention in psychopathic female offenders. *Neuropsychology*, *21*, 301–312.
- Wallace, J. F., Schmitt, W. A., Vitale, J. E., & Newman, J. P. (2000). Information processing deficiencies and psychopathy: Implications for diagnosis and treatment. In C. Gacono (Ed.), *The clinical and forensic assessment of psychopathy: A practitioner's guide* (pp. 87–109). Mahwah, NJ: Lawrence Erlbaum Associates.
- Wallace, J. F., Vitale, J. E., & Newman, J. P. (1999). Response modulation deficits: Implications for the diagnosis and treatment of psychopathy. *Journal of Cognitive Psychotherapy*, 13, 55–70.
- Woodworth, M., & Porter, S. (2002). In cold blood: Characteristics of criminal homicides as a function of psychopathy. *Journal of Abnormal Psychology*, 111, 436–445.
- Wykes, T., Reeder, C., Landau, S., Everitt, B., Knapp, M., Patel, A., & Romeo, R. (2007). Cognitive remediation therapy in schizophrenia: Randomised controlled trial. *British Journal of Psychiatry*, 190, 421–427.
- Wykes, T., Reeder, C., Williams, C., Corner, J., Rice, C., & Everitt, B. (2003). Are the effects of cognitive remediation therapy (CRT) durable? Results from an exploratory trial in schizophrenia. *Schizophrenia Research*, *61*, 163–174.
- Wykes, T., & Van der Gaag, M. (2001). Is time to develop a new cognitive therapy for psychosis? Cognitive remediation therapy. *Clinical Psychological Review*, *21*, 1227–1238.
- Zeier, J. D., Maxwell, J. S., & Newman, J. P. (2009). Attention moderates the processing of inhibitory information in primary psychopathy. *Journal of Abnormal Psychology*, 118, 554–563.