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Criminal Justice and Behavior 2009; 36; 1241

DOI: 10.1177/0093854809343119

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THE CRIMINOLOGY OF THE AMYGDALA

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A central part of the brain's limbic system, the amygdala is crucial for emotional learning, aversive conditioning, and response to fear and other emotions. Although the amygdala is a vibrant area of study in the neurosciences, it is virtually ignored in criminology. Here, we review the anatomical location, connectivity, and functions of the amygdala, explore its role in James Blair and colleagues' recently advanced theory of psychopathy, explicate amygdala abnormalities in diverse populations, and explore genetics research relating to amygdala functioning. Because of its role in the regulation of fear and other emotional memory and response, the amygdala is importantly related to psychopathy; callous-unemotional traits; and the vibrant, neuroscience-based investigations of the etiology of antisocial behavior.

Keywords: amygdala; psychopathy; limbic system; fear; callous-unemotional traits; neural substrates

In a classic article in *Criminal Justice and Behavior*, Hare (1996) introduced the construct of psychopathy to a criminal justice and criminological audience. Although it had been studied for nearly two centuries in the fields of medicine, psychiatry, and psychology, the personality disorder was relatively understudied within criminology despite its clear links to the study of crime. Today, the study of psychopathy has proliferated and it is among the most investigated constructs in psychological studies of antisocial behavior. The current study aspires to do the same by introducing the amygdala, which is a brain region in the limbic system that is involved in emotional learning, aversive conditioning, and response to fear and other emotions. Like the construct of psychopathy, we believe the amygdala is crucial to the scientific understanding of antisocial behavior.

Although it has been studied scientifically for nearly two centuries, the amygdala has rarely been an object of criminological study. For instance, according to the National Criminal Justice Reference Service, the amygdala has been the subject of just one criminological study (Pontius & LeMay, 2003). This is unfortunate because the amygdala is extensively studied in other fields and is implicated in a prominent theory of psychopathy (J. Blair, Mitchell, & Blair, 2005) and likely has much to offer criminology (DeLisi, Wright, Vaughn, & Beaver, 2009). Here, we review the anatomical location, connectivity, and functions of the amygdala, explores its role in R. J. R. Blair's theory of psychopathy, explicate amygdala abnormalities in diverse populations, and explore genetics research of the amygdala. Because of its role in the regulation of fear and other emotional memory and response, the amygdala is importantly related to psychopathy, callous-unemotional (CU) traits, and the vibrant, neuroscience-based investigations of the etiology of antisocial behavior.

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CRIMINAL JUSTICE AND BEHAVIOR, Vol. 36 No. 11, November 2009 1241-1252

DOI: 10.1177/0093854809343119

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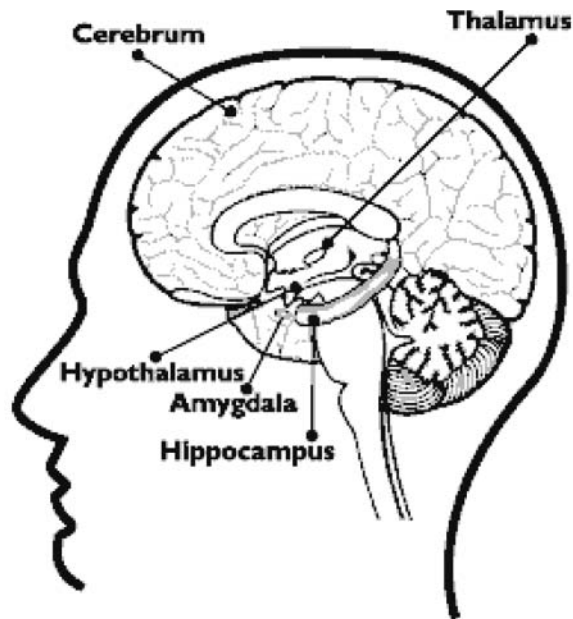


Figure 1: The Amygdala

ANATOMICAL OVERVIEW AND FUNCTIONS

Latin for *almond*, the amygdala is an almond-shaped structure located in the medial temporal lobes (see Figure 1). It is bilaterally located, thus one amygdalae exists in the right temporal lobe and one amygdalae exists in the left temporal lobe. Like most brain regions, the amygdala is not a single, coherent mass but instead is composed of multiple subareas or nuclei. Thus, the almond-shaped structure that is conventionally understood as the amygdala is the basal nucleus. The amygdala is often divided into three primary nuclear areas (which of course can be subdivided). These are the lateral (basolateral) nuclei, central nucleus, and medial (corticomedial) nuclei (Clark, Boutros, & Mendez, 2008). Various subdivisions of the amygdala are in use, and it is commonly but variously referred to as the basolateral amygdala, amygdaloid complex, or simply, amygdala, in the research literature (R. J. R. Blair, 2008a; Davis, 1992; Davis & Whalen, 2001; LeDoux, 1996, 2002; Phelps, 2006).

Along with other brain structures including the hippocampus, septum, basal ganglia, and others, the amygdala is part of the limbic system, which regulates the expression of emotion and emotional memory. As shown in Figure 2, the amygdala is importantly linked to other brain regions and is responsible for multiple and diverse physiological responses to emotional cues such as fear. As Davis and Whalen (2001) succinctly suggested, "A stimulus that predicts an aversive outcome will change neural transmission in the amygdala to produce the somatic, autonomic, and endocrine signs of fear, as well as increased attention to that stimulus" (p. 13). In addition to its connectivity with other regions, the amygdala is also related to neurotransmitter systems, which themselves are importantly related to

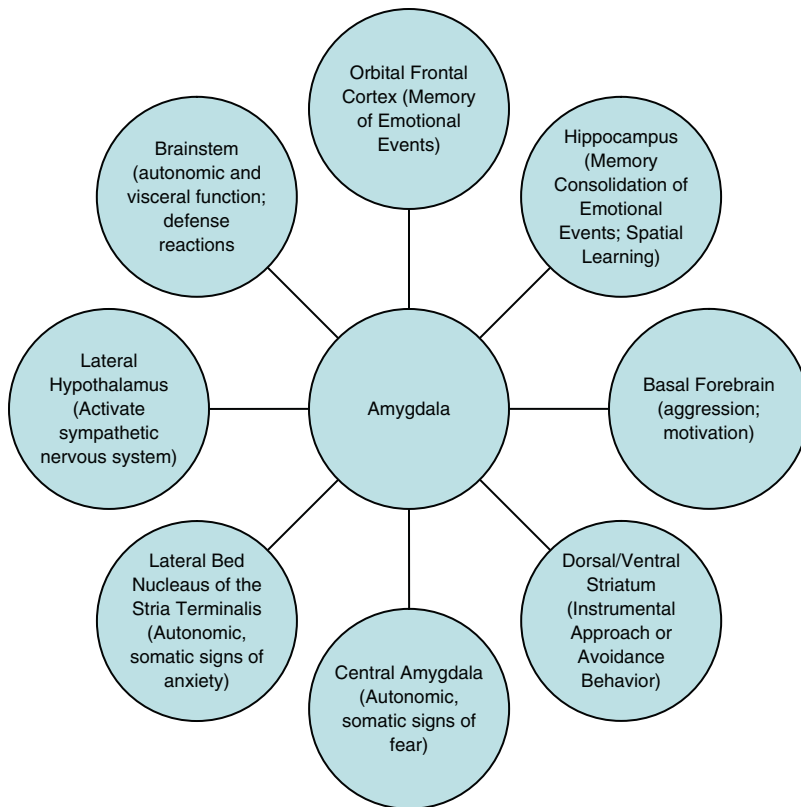


Figure 2: Connectivity (with functions) to the Amygdala

behavior. For instance, the information that flows through the amygdala is modulated by several neurotransmitter systems. Norepinephrine, dopamine, serotonin, and acetylcholine released in the amygdala influences how excitatory and inhibitory neurons interact and these neurotransmitters are released throughout the forebrain, including within the amygdala (LeDoux, 2007). In sum, the amygdala is important not only for its independent functions but also its relationship to other diverse neural functions within and beyond the limbic system (Damasio, 1994; Phelps, 2006).

Without question, the amygdala is the key brain structure mediating defensive behavior in stages of fear and anxiety, is involved in instrumental learning and aversive conditioning, and is activated in responses to fearful and sad facial expressions (Adolphs, Gosselin, Buchanan, Tranel, Schyns, & Damasio, 2005; Adolphs, Tranel, Damasio, & Damasio, 1994, 1995; R. J. R. Blair, 2004; Cacioppo & Berntson, 2005; Delgado, Nearing, LeDoux, & Phelps, 2008; Herry et al., 2008; LeDoux, 2000; Morris, Öhman, & Dolan, 1998). In their seminal studies, Adolphs et al. (1994, 1995) found that persons with bilateral amygdala damage were unable to recognize fearful facial expressions even though they were able to recognize facial identity. Similarly, Adolphs, Tranel, and Damasio (1998) compared social judgments based on stranger facial expressions among three subjects with complete bilateral amygdala damage. Compared to controls, the impaired group was unable to form appropriate social judgments of the most unapproachable and untrustworthy people.

Although it is best known for its role in processing fear, the amygdala has also been implicated in emotional states associated with aggressive, maternal, sexual, and eating behaviors. For instance, R. J. R. Blair, Morris, Frith, Perrett, and Dolan (1999) found increased activity in the left amygdala in response to sad expressions similar to those in response to fear. Interestingly, no amygdala response was found in response to anger. It has been suggested that the amygdala is differentially responsive to fear and anger because fear is an uncertain emotion that requires greater arousal whereas anger is more certain. For instance, an angry face connotes clearly that the person with an angry expression is upset. On the other hand, a person with a fearful face does not readily convey what stimulus is causing the fear. Whalen and his colleagues (Davis & Whalen, 2001; Whalen, 1998; Whalen et al., 2001; Whalen, Shin, Somerville, McLean, & Kim, 2002) suggested that the amygdala is involved in vigilance and resolving ambiguity, which would explain its greater reactivity to fear versus anger.

The amygdala has been shown to process motivationally relevant information in accordance with current processing goals—a process known as affective flexibility, and researchers have found that the amygdala processes negative information less flexibly than positive information (Cunningham, Van Bavel, & Johnsen, 2008; Whalen et al., 2004). Other research has found that oxytocin reduces amygdala activation and modulates the coupling of amygdala–brain stem activity implicated in autonomic and behavioral manifestations of fear (Kirsch et al., 2005). In short, the amygdala processes the emotional significance of external stimuli, interacts with the hippocampus (where emotional memories are stored), and interacts with cognitive functions in the orbitofrontal cortex to respond to the stimuli (Bechara, Damasio, & Damasio, 2000; Hein & Singer, 2008; LeDoux, 2007; Phelps, 2004; Whalen, 2007).

It is important to note that fear is a multifaceted construct that is assessed by multiple tasks (see Adolphs, 2008). For instance, fear expression is assessed by physiological tasks, such as startle reflexes, freezing, and facial expressions. Fear experience is inferred from fear expressions and is self-reported. Innate fear is assessed by biologically basic sensory stimuli. Fear detection is assessed by reaction time to fear stimuli. Fear discrimination and classification are gauged by accuracy in distinguishing fear from other stimuli. In other words, fear is a complex phenomenon and one with multiple neural substrates.

In a landmark study, Davidson, Putnam, and Larson (2000) implicated the amygdala as an important brain structure involved in emotion regulation and violence. They suggested that

the role of the amygdala in impulsive aggression is complex. Individual behaviors that connote threat (e.g., staring eyes, threatening vocalization, lunging posture) are conveyed to the lateral nucleus of the amygdala, which then projects to the basal nuclei, and it is there that information about the social context derived from OFC (orbitofrontal cortex) projections is integrated with the perceptual information. Behavioral responses can then be initiated via projections from the basal nuclei to various cortical zones, and physiological responses can be produced via projections from the basal nuclei to the central nucleus and then to the hypothalamus and brainstem. *Too much or too little activation of the amygdala may give rise to either excessive negative affect or decreased sensitivity to social cues that regulate emotion, respectively.* (p. 594, italics added, references omitted)

Indeed, scholars noticed that the physiological effects of amygdala dysfunction were consistent with manifestations of psychopathic personality (see R. J. R. Blair, 1995, 1997, 1999; Patrick, 1994; Raine, 1993; van Goozen, Snoek, Matthys, van Rossum, & van

Engeland, 2004). To illustrate, R. J. R. Blair, Budhani, Colledge, and Scott (2005) explored the ability of boys with psychopathic tendencies to process auditory affect. Subjects were presented with neutral words spoken with intonations conveying happiness, disgust, anger, sadness, and fear and asked to identify the emotions of the speaker based on prosody. Compared to controls, they found that psychopathic boys presented with impairment for the recognition of fearful vocal affect. In a study of startle reflex, van Goozen and her colleagues (2004) compared startle reflexes of 21 behaviorally disordered or psychopathic children and 33 normal controls to a series of 27 slides of positive, neutral, and negative stimuli. The startle-elicited blinks of behaviorally disordered children were significantly lower for all categories of slides, and the more antisocial the children were, the lower their startle responses were during unpleasant states. Taken together, amygdala dysfunction melds with the core emotional deficiencies demonstrated by persons with psychopathic personality. This relationship is the basis of what we refer to as Blair's amygdala theory of psychopathy, which is examined next.

BLAIR'S AMYGDALA THEORY OF PSYCHOPATHY

The amygdala is thus involved in all the processes that, when impaired, give rise to the functional impairments shown by individuals with psychopathy. It is therefore suggested that amygdala dysfunction is one of the core neural systems implicated in the pathology of psychopathy. (R. J. R. Blair, 2003, p. 5)

This quotation captures the essence of the amygdala dysfunction theory of psychopathy advanced by James Blair and his colleagues (R. J. R. Blair, 1995, 1997, 1999, 2005, 2008a, 2008b; R. J. R. Blair & Mitchell, 2009; J. Blair, Mitchell, & Blair, 2005). According to Blair, psychopathic persons show reduced neural response to threatening stimuli, reduced aversive conditioning, reduced emotional responses in anticipation of punishment, reduced emotional responses to imagined threatening events, and reduced startle reflex to aversive stimuli. All of these impairments are consistent with general amygdala dysfunction. Moreover, the amygdala is responsible for two capacities that are necessary for successful socialization. First, people must be capable of an aversive emotional response to distress or fear in other people so that they can—and this is the second capacity—internalize “right” and “wrong.” Psychopathic persons are impaired on both fronts, evidenced by reduced autonomic responses to distress cues of others and impaired fear recognition (R. J. R. Blair, 2007).

This means that individuals with amygdala dysfunction and psychopathic personalities are impaired in their ability to process the fear and sadness of their victims. They are unable to empathize because the amygdala is necessary for the formation of stimulus-response and stimulus-reinforcement associations (particularly aversive-oriented learning). According to R. J. R. Blair (2006),

The temperamental variable of fearfulness, related to the ease with which the child can be socialized, can be understood as an index of the integrity of the amygdala. While fear conditioning is not necessarily important in socialization, it is argued that the amygdala's response to the fear and sadness of victims during empathy induction is crucial for socialization. For normal moral socialization to occur, the developing child needs to associate harmful transgressions with the punishment of the distress of the victim. However, individuals with psychopathy are

less able to form these associations and so are at greater risk to choose antisocial behavioral options. (p. 307, references omitted)

Blair's theory is a fascinating attempt to link neural dysfunction in a particular brain region with the physiological and behavioral manifestations of psychopathic personality. To be sure, Blair's theory is significantly more complex than the current presentation and involves brain regions beyond the amygdala, such as the ventromedial prefrontal cortex (see R. J. R. Blair, 2004, 2006, 2008a; J. Blair et al., 2005). However, the amygdala forms the core of his theory and we focused our discussion on it for the purpose of this study. Next, we explore two areas of research that amplify the criminological salience of the amygdala: amygdala abnormalities in populations diverse in antisocial behavior and CU traits.

AMYGDALA ABNORMALITIES IN DIVERSE POPULATIONS

It is recognized that multiple neurocognitive impairments differentiate pathological offenders from nonpathological offenders (Crowe & Blair, 2008; DeLisi, 2005, 2009b; Moffitt, 1993; Raine et al., 2005; van Goozen & Fairchild, 2008; Vaughn & DeLisi, 2008), and diverse investigators have studied amygdala abnormalities as potentially a source of this variation. For example, using healthy respondents and positron-emission tomography measures of neural activity, Morris et al. (1996) reported greater activity in the left amygdala in response to fearful as opposed to happy expressions. Amygdala activity increased with increasing fearfulness and decreased with increasing happiness providing direct evidence that the amygdala processes the emotional valence of faces, particularly fearful expressions (also see Adolphs et al., 1995, 2005, 1994; Davis & Whalen, 2001). It is suggested that amygdala dysfunction would contribute to an inability of fear conditioning which in turn would increase impulsivity and risk taking—two important correlates of behavioral control (Barkley, 1997; Gottfredson & Hirschi, 1990). Using data from 20 male college students, Gordon, Baird, and End (2004) found that those scoring high on the Psychopathic Personality Inventory (Lilienfeld & Andrews, 1996) used a different pattern of neural activity in response to tasks that required affective processing. Whereas nonpsychopathic males used their amygdala, more psychopathic respondents primarily used their right dorsolateral prefrontal cortex, which is associated with working memory functioning.

In a comparative study of spousal abusers and nonabusers, Lee, Chan, and Raine (2008) produced functional brain imaging (fMRI) data that show increased activation in the right amygdala when responding to aggressive words, suggesting that “batterers have inadequate prefrontal resources to exercise top-down regulatory control over the excessive limbic activation generated by negative stimuli” (p. 656). On the other hand, not all imaging studies reported amygdala differences between offenders and nonoffenders. For example, Tiihonen et al. (2008) compared regional brain volumes of 26 persistently violent offenders with antisocial personality disorder and substance dependence and 25 healthy male controls and found no amygdala differences (for a review, see Raine & Yang, 2006).

Marsh et al. (2008) conducted an fMRI study of 36 children, 12 with CU traits and conduct disorder or oppositional defiant disorder, 12 with attention-deficit hyperactivity disorder, and 12 healthy comparison subjects. Relative to healthy children and those diagnosed with attention-deficit hyperactivity disorder, youths with CU traits

displayed reduced amygdala activation while processing fearful expressions but not neutral or angry expressions. Symptom severity in the CU group was negatively correlated with connectivity between the amygdala and ventromedial prefrontal cortex. Others have also found amygdala abnormalities in children with conduct disorder (Vloet, Konrad, Huebner, Herpertz, & Herpertz-Dahlmann, 2008).

Decety, Michalska, Akitsuki, and Lahey (2009) recently executed the first functional neuroimaging investigation of brain responses to pain empathy-eliciting stimuli in adolescents with conduct disorder. Their findings were startling. Youths with conduct disorder exhibited less amygdala/prefrontal coupling when watching pain inflicted by another. Prior research suggests that compared to nonpsychopathic inmates, psychopaths have brain abnormalities in the paralimbic system generally that are similar to those evinced by patients with amygdala damage (Kiehl, 2006; Kiehl, Bates, Laurens, Hare, & Liddle, 2006). In his review of paralimbic system dysfunction, Kiehl (2006) noted that removal of the anterior temporal lobe appears to alleviate psychopathic and antisocial symptoms in patients, including reduced violence and hostility, increased empathy, and increased warmth in social relationships. Moreover, elective amygdalotomies, which have been performed on patients with severe aggressive disorders, result in reduced aggressive behaviors and restored emotional control. In short, neuropsychiatric studies are increasingly showing that the amygdala plays an important part in the development of an empathic brain, and when dysfunction is present, a brain that fails to demonstrate empathy (see Decety & Moriguchi, 2007).

Further study of amygdala abnormalities could provide links between psychiatric diagnoses and extreme forms of criminal behavior, such as sexual murder and serial murder. For instance, Briken, Habermann, Berner, and Hill (2005) examined the prevalence of brain abnormalities in a sample ($n = 166$) of sexual murderers and found that 30% of the homicide offenders had brain abnormalities. Many of these offenders also had severe psychiatric diagnoses. For instance, 50% of sexual murderers with brain abnormalities presented with sadistic personality disorder. Because the hallmark of sexual sadism is the infliction of pain and suffering for the gratification of the offender, it makes sense that amygdala dysfunction plays some role in aggravated homicide offending (Briken et al., 2005; Hill, Habermann, Berner, & Briken, 2007).

CU TRAITS

In recent years, criminologists have established that one of the most pernicious risk factors for serious antisocial behavior is CU traits (see Frick & White, 2008). For instance, Stickle, Kirkpatrick, and Brush (in press) assessed CU traits vis-à-vis other risk factors for aggression based on data from a sample of 150 confined adolescents. They found that CU traits contributed unique and strong associations with aggressive behaviors withstanding the competing effects of onset of antisocial behavior, impulsivity, aggression beliefs, and other controls. Across studies, children and adolescents with CU traits display a temperament characterized by deficits in their emotional arousal to fear and distress in others and abnormalities in their responses to cues of punishment and danger (Pardini, 2006). In other words, CU traits comprise the hallmark of fledgling psychopathy which—according to Blair's theory—is suggestive of amygdala dysfunction. Indeed, according to Frick and White (2008),

These temperamental characteristics could provide clues to distinct neural mechanisms that . . . could help explain the genetic diathesis of their antisocial behavior. . . . These specific emotional and cognitive deficits could implicate deficits in amygdala functioning and related neural activity. (p. 366)

Recently, Jones, Laurens, Herba, Barker, and Viding (2009) conducted an fMRI of 17 boys with conduct problems and elevated levels of CU traits and 13 comparison boys of equivalent age and intelligence. To emphasize, healthy amygdala functioning involves increased activation when shown fearful as opposed to neutral faces. Jones and her colleagues found that relative to the comparison group boys with conduct problems and CU traits manifested reduced right amygdala activity to fearful faces (left amygdala activity was found). They concluded that the neural substrate of emotional impairment associated with CU antisocial behavior was already present in boys as young as age 11.

Behavioral genetics researchers have convincingly shown that CU traits are strongly heritable. For instance, Viding, Blair, Moffitt, and Plomin (2005) studied 3,687 twin pairs and found that 67% of variation in extreme CU traits among 7-year-old children was genetic in etiology. For extreme antisocial behaviors in 7-year-olds with psychopathic tendencies, genes accounted for 81% of the variation. Other studies found that 71% of conduct problems in boys and 77% in girls were attributable to genes (Viding, Frick, & Plomin, 2007) and that 80% of variance in twins with CU traits and antisocial behavior was heritable (Larsson, Viding, & Plomin, 2008).

If CU traits are strongly heritable—and they are—then the next step is to identify measured genes that are associated with these traits. Indeed, one of the foremost scientific frontiers of the current era centers on finding candidate genes for violent and antisocial behaviors that are located in specific brain regions (DeLisi, 2009a; Plomin & Asbury, 2005; Viding & Frith, 2006). Several candidate genes affecting amygdala functioning have been researched, including the tryptophan hydroxylase-2 gene (TPH₂; Brown et al., 2005), neuropeptide Y gene (NPY; Hariri & Weinberger, 2009), the dopamine catabolic enzyme catechol-O-methyltransferase (COMT Val 158Met; Montag et al., 2008), and monoamine oxidase A (MAOA; Meyer-Lindenberg et al., 2006). For instance, Meyer-Lindenberg and colleagues (2006) studied the impact of a common functional MAOA polymorphism on brain structure and function assessed with fMRI data. They found that the low expression MAOA polymorphism predicted pronounced limbic volume reductions and hyperresponsive amygdala activity during emotional arousal. Montag and colleagues (2008) reported evidence that the Val 158Met polymorphism in the COMT gene is associated with increased reactivity to fearful stimuli, which confers greater susceptibility to anxiety disorders. Based on these findings, there should be a negative relationship between this polymorphism and psychopathy.

The greatest amount of molecular genetics research on the amygdala has centered on the serotonin transporter gene, which has been shown to have robust effects on amygdala function (Hariri et al., 2005). For instance, Hariri et al. (2002) explored the serotonin transporter gene (SLC6A4) and amygdala functioning and found that those with one or two copies of the short allele (5-HTT) promoter polymorphism exhibited greater amygdala neuronal activity and thus greater fear- and anxiety-related behaviors. Similarly, Hariri and Holmes (2006) concluded that genetic variations in 5-HTT function affect both the structure and function of key limbic pathways that regulate the brain's capacity for effectively dealing with stress and affective conditions. Carriers of the short allele of the serotonin transporter

gene have also been found to reduce gray matter volume in the amygdala (Pezawas et al., 2005). This is consistent with research that finds reduced fear and internalizing disorders among antisocial persons with serotonin hypofunction (Seo, Patrick, & Kennealy, 2008). Indeed, a recent meta-analysis supported the association between the serotonin transporter gene and amygdala activation (Munafò, Brown, & Hariri, 2008). In sum, greater study of the amygdala can direct gene hunters to candidate genes for endophenotypes associated with antisocial behavior.

DISCUSSION

Evidence is converging that neural substrates play an important role in explaining variations in antisocial behavior (Wright, Tibbetts, & Daigle, 2008). For instance, Raine and Yang (2006) concluded that

there are some similarities between the neural system underlying moral decision making in normal individuals, and brain mechanisms thought to be impaired in delinquent, criminal, violent, and psychopathic populations. We suggest that this is not a chance association but instead represents a neural insight into the etiology of antisocial behavior. (p. 203)

To illustrate, much research has explored differences for criminal behavior that was perpetrated under different emotional states, such as instrumental (cool or affective) versus reactive (hot or hostile) states (R. J. R. Blair & Mitchell, 2009; Hare, 1996; Kiehl, 2006; Meloy, 2006; Weinshenker & Siegel, 2002). Although this strict dichotomy is likely mixed, there are theoretical reasons to link instrumental forms of violence to amygdala dysfunction. Future criminological research and theorizing would benefit from approaches that link criminal behavior and its immediate milieu with important underlying neural substrates, such as the amygdala. This intersection of brain and behavior can then be fruitfully tied to genetics research and the study of environmental risk pathogens across time.

Finally, we hope that this review not only innervates research in criminology but also criminal justice settings. All practitioners can attest to the reality that correctional treatments and treatment modalities are differentially successful across the offender population. But even among offenders who are generally matched in terms of their risk factors, there are differences in amenability and responsiveness to treatment. Wilson's (1993) ambitious general theory of morality implicated the limbic system as partly responsible for ensuring that "animal impulses are kept in reasonable check by social ones" (p. 133). Without mentioning it, Wilson was implicating the amygdala. And a greater understanding of this brain region can shed meaningful light on understanding the etiology of antisocial behavior and perhaps why some offenders are successful at desisting from crime and others are not.

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