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Psychopathy & Aggression: When Paralimbic Dysfunction Leads to Violence

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Abstract

Psychopaths can be alarmingly violent, both in the frequency with which they engage in violence and the gratuitous extent of their violent acts. Indeed, one principal utility of the clinical construct of psychopathy is in predicting future violent behavior in criminal offenders. Aggression is a complex construct that intersects psychopathy at many levels. This chapter provides a review of psychopathy as a clinical construct including the most prominent cognitive and neurobiological models which serve to account for its pathophysiology. We then describe how the brain abnormalities implicated in psychopathy may lead to diverse behavioral outcomes, which can include aggression in its many forms.

Keywords

Psychopathy; Violence; Impulsive Aggression; Premeditated Aggression

Introduction

Many psychiatric disorders and neuropsychological conditions may be linked to aggressive behavior and violent acts. Among these, psychopathy deserves special attention as this condition is strongly associated with persistent aggressive traits and violent behavior throughout the lifespan. This chapter describes how neural abnormalities associated with psychopaths' emotional dysfunction and personality traits may also be linked to aggressive tendencies in some contexts; however, several important qualifications must be made with regard to this relationship, particularly with respect to how psychopathy and aggression are defined. Psychopaths are often mischaracterized in the popular media, promulgating misconceptions about this psychiatric disorder, which has been well-defined in clinical circles for decades. Furthermore, aggression comes in many forms ranging from drunken bar brawls to calculated homicide, and in some conceptualizations even includes non-violent actions. In order to achieve a more sophisticated understanding of the relationships between psychopathy and aggression, it will be necessary to review some essential background which has contributed to our contemporary understanding of both constructs. In doing so, this will

help to clarify the pathophysiology underlying discrete patterns of aggressive behavior among psychopaths, a population that may indeed be particularly susceptible to violent behavior.

Essential Background: Conceptualization of Psychopathy

Descriptions of particularly unscrupulous individuals, who fail to learn from punishment and who habitually violate the rights of others for personal gain, have been well documented throughout history and across cultures (Murphy 1976). However, notions that such behavior may constitute a mental deficiency rather than an evil nature gained traction only relatively recently, around the early 19th century. This period marked the infancy of our understanding of abnormal psychology, such that practitioners were only beginning to understand the organic relationship between the brain and individual differences in thoughts and behavior. The clinician Pinel documented the notion of *madness without delirium*, in which individuals retained most outwardly observable faculties such as intelligence and a grasp on reality, while still suffering deficiencies in their character, such as emotions or moral judgment (Pinel 1806). Prichard (1835) extended this notion to a condition he called *moral insanity*, in which a specific and essentially isolated deficit in moral behavior was recognized. These early conceptualizations provided a starting point for our understanding of a variety of disorders, including psychopathy.

As the concept of the *psychopathic personality* gained prominence in the early 20th century, there remained a need for some descriptive consolidation to unify the construct and bolster its clinical utility. While the construct has continued to evolve, our modern clinical understanding of the psychopath is conceptually founded on Cleckley's (1941) authoritative delineation of 16 core diagnostic traits which characterize the disorder. His case studies paint a vivid picture of the psychopath suffering from an inability to experience empathy, guilt or remorse, and as one who often fails to exercise sound judgment and lacks the basic foundation for socialized, moral behavior. As such, psychopaths show a profound disregard for the rights and well-being of others. At the same time, psychopaths are ordinarily not delusional or psychotic, and they generally perform at normal to high levels of intelligence. This combination of personality traits may be recognized at varying levels among healthy, relatively high functioning members of society; however, the full clinical manifestation of these traits occurs in less than 1% of the general population (Hare 2003). Individuals who meet clinical criteria for psychopathy tend to fail to achieve normal academic success, frequently lose their jobs or quit without justification, suffer through multiple divorces, and may be socially ostracized due to others' perception of their chronic imprudent choices. As such, psychopathy represents a maladaptive condition severely impairing decision-making, moral judgment, and social functioning.

While a diverse collection of personality traits characterize psychopathy—and indeed define it clinically—contemporary neuroscience research suggests these traits develop from abnormalities in brain structure and function that bias fundamental processes of learning and behavior in measurable ways. As we delineate relationships between psychopathy and aggression, a repeated theme in this chapter will be that certain specific behavioral manifestations of a neuropsychiatric disorder, such as the manifestation of aggressive

behavior in psychopathy, are often context-specific and highly variable due to a wide range of developmental influences and etiological factors. A primary focus on neurobiological factors capable of influencing patterns of learning and motivation can provide a more stable reference, and so this chapter will emphasize these features of psychopathy prior to discussions of aggressive outcomes. First, it will be necessary to understand how psychopathy is measured, clinically and for the purposes of empirical research.

Measuring Psychopathy

Research examining psychopathy and its associated behavioral outcomes requires an appeal to some standard measure of the construct. Most studies have relied on assessment tools specifically developed to account for the range of traits defined by Cleckley (1941) in his case studies. Of these tools, Hare's Psychopathy Checklist (PCL; Hare, 1980), now in a revised form (PCL-R; Hare, 1993; 2003), has emerged as the most widely used and well-recognized measure of psychopathy. It is a semi-structured interview paired with a review of institutional files for collateral information, and is designed to be carried out by trained assessors to evaluate the overall presence and severity of 20 items, each graded from zero to two. The recommended clinical cutoff for a diagnosis of psychopathy is 30 out of a maximum 40 points on the scale, although researchers have sometimes used different cutoffs in studies of the underlying biology.

Items on the PCL-R fall into two correlated dimensions, or factors, representing emotional symptoms (Factor 1) and socially deviant behaviors (Factor 2), respectively (Harpur et al. 1988; Hare et al. 1990). This two-factor conceptualization of psychopathy aligns with theoretical treatments of the construct which refer to emotional deficits as the core, precipitating features that drive the development of certain behavioral manifestations including antisocial tendencies (Harpur et al. 1989). There have been recent suggestions for more complex factor solutions for the PCL-R (Cooke and Michie 2001; Hare 2003) nonetheless, each of these alternative solutions have elements which distinguish socially deviant behavior from emotional abnormalities and personality features, and this distinction remains by far the most widely referenced in terms of its correspondence to theoretical constructs of personality as well as etiological and neuropsychological antecedents.

Other measures of psychopathy have been developed for specific purposes such as measuring psychopathic traits in non-incarcerated samples (Levenson et al. 1995; Lilienfeld and Widows 2005) and youth with psychopathic traits (Forth et al. 2003; Frick and Hare 2001). Analyses of these scales generally support the two-factor conceptualization of psychopathy (for a detailed review of the measurement issues in psychopathy, see (Hare and Neumann 2009). Each measure has particular advantages and limitations, and each of these measures has pushed research forward in helpful ways. In addition to refining the clinical construct of psychopathy, the development of the PCL-R and its derivatives has aided the growth of a sound body of empirical literature, including a better understanding of the relationship between psychopathy and aggressive behavior. Indeed, the more accurately one measures a psychological construct (i.e., psychopathy) the more likely one will find stable neurobiological correlates of that construct.

Psychopathy as a Psychiatric Diagnosis

While the construct of psychopathy was intended to fall under the DSM-IV-TR (APA, 2000) categorization of *Antisocial Personality Disorder* (ASPD), it is a much belabored point among researchers and clinicians that it is not well accounted for by the ASPD diagnostic criteria (Cunningham and Reidy 1998; Hare et al. 1991). Early versions of the DSM had utilized a different classification system which included categories such as *sociopathic personality disturbance*; however, this was not specifically synonymous with our modern concept of psychopathy. ASPD was first introduced in DSM-III (APA, 1980), which remains in the current version (DSM 5). However, the current diagnostic criteria miss the mark when assessing classic psychopathic symptomatology, as ASPD focuses heavily on criminal behavior and social deviance, while largely ignoring the affective elements considered central to psychopathy (Blackburn 2007). Due to the generality of the symptoms, over 4% of the general public will meet criteria for ASPD (APA, 2000), but less than 1% meet criteria for psychopathy using the Hare Psychopathy Checklist-Revised (Hare 2003). In forensic samples, where ASPD diagnoses may occur at rates near 80% or higher, the best estimates for rates of psychopathy are 15–25% on average (Hare 2003; Hare et al. 1991). Despite the necessary distinction between ASPD and psychopathy as assessed by the Hare PCL-R, antisocial behavior and aggression remain among the most visible characteristics of psychopathy. It must simply be recognized that persistent criminal behavior *per se* is not sufficient for an accurate diagnosis of psychopathy, which requires the presence of the core emotional deficits. Still, antisocial behavior, but not necessarily criminal behavior, is a critical feature of the disorder.

Longstanding efforts to study non-incarcerated and “successful” psychopaths persist (Babiak and Hare 2006; Gao and Raine 2010; Widom 1977); however, what may seem like a distinct subtype may likely be differentiated as individuals with subclinical levels of a few psychopathic traits. Nevertheless, psychopathic traits, even at subclinical levels, may be related to poor outcomes, including aggressive behaviors. Going forward in our examination of aggression in psychopaths, we should recognize it as a situationally-complex behavior which can arise from a more fundamental influence of neurobiology on learning, motivation, and personality traits which characterize psychopaths.

Predicting Violent Outcomes

Psychopathy, as measured by the PCL-R, has long been documented as a robust indicator in forensic settings of how likely one is to reoffend, (Porter et al. 2001; Salekin et al. 1996), and it is a particularly strong predictor of future violent recidivism (Cornell et al. 1996; Harris et al. 1991; Porter et al. 2009). Indeed, research has indicated that within one year of release from prison psychopaths are about 4–6 times more likely to commit another violent crime than are non-psychopaths (Hemphill et al. 1998). Estimates are fairly consistent across studies: within 10 years after release over 70% of psychopaths (with a history of violence) commit another violent offense, and 20 year follow-ups indicate that as many as 90% of psychopaths (with a history of violence) will be re-arrested for violent crimes. In contrast, the average non-psychopathic violent offender appears to have a risk for violent recidivism around 40% (Hare et al. 2000; Harris et al. 1991; Hemphill et al. 1998; Rice and Harris

1997). Furthermore, analyses of the actual crimes committed by psychopaths suggest a heightened severity and gratuitous nature to their violent acts (Porter et al. 2003).

These same trends are evident in youth. An accumulating body of literature has provided strong evidence indicating that carefully defined aspects of psychopathic traits, typically referred to as *callous and unemotional traits* when assessed in youth, are apparent at a young age and are persistent across the lifespan (Lynam et al. 2007). These traits are often accounted for by assessment tools designed for behaviorally disordered youth (Forth et al. 2003; Frick and Hare 2001). The combination of persistent behavioral problems (i.e., conduct disorder) and callous, unemotional traits in youth is associated with persistent patterns of severe aggression, and is a particularly strong predictor of future violent offending (Frick et al. 2003; Vitacco and Vincent 2006).

Within forensic samples the PCL-R/PCL-YV reliably predicts violent outcomes, but it should be noted that not all psychopaths are necessarily violent (Cleckley, 1976). Violence should not be seen as a unique distinguishing quality of psychopaths, yet among the neural deficits underlying psychopathy, some may contribute to increased risk for violent behavior in various contexts. Examining the wide variety and motivations for aggressive acts and the various neural systems which support them can thus provide us with an informative framework to discuss the specific neural deficits associated with psychopathy.

Varieties of Aggression

It is easy to recognize variability in aggressive behavior among humans and other animals. Indeed, many have felt the impulse to react violently toward someone who provokes them, and some of us may have even acted upon these impulses—such instincts are not necessarily pathological. It is quite another thing to stalk a potential victim for days, devising a malicious plan to harm someone else for their own benefit. The latter requires a host of violations of ordinary social rules, which are not simply overwhelmed in a momentary fit of rage, but rather, are wholly disregarded in a prolonged plan to achieve some self-serving goal at another's expense. The heterogeneity inherent in violent behavior has prompted the establishment of a variety of categorizations, the most prominent one distinguishing between impulsive and premeditated aggression (McEllistrem 2004).

Impulsive aggression is characterized as a reactive, emotional response to some immediate provocation or frustration. In contrast, *premeditated aggression* results from more complex motivating factors and is largely characterized by predatory actions serving some instrumental purpose. Each of these concepts can be referred to by several alternative terms such as *reactive* versus *proactive* aggression (see elsewhere in this volume), but the conceptual divisions are essentially the same. Factor analyses of reported aggressive acts support the distinction between impulsive and premeditated aggression (Barratt et al. 1999), and some evidence suggests that an individual's aggressive behavior can be characterized as aligning predominantly toward one or the other mode of aggressive tendencies (Stanford et al. 2003). It is also increasingly recognized that aggression need not be strictly defined by physical altercations, but can be complex social actions sometimes referred to as *indirect* or *relational* aggression (Archer and Coyne 2005), and which may include general hostility, bullying, manipulation, and intimidation.

It has sometimes been suggested that psychopaths predominantly commit predatory, instrumental violence (Williamson et al. 1987; Woodworth and Porter 2002) but it is important to recognize that psychopaths also commit impulsive violent acts (Blair 2010) and some have suggested that impulsive risk-taking is a strong motivating factor in psychopaths' aggressive behavior, and is in itself reinforcing (Porter et al. 2000). But this feature is not unique to psychopathy. Impulsive aggression and behavioral disinhibition are prevalent among many psychiatric disorders including post-traumatic stress disorder (Silva et al., 2001), depression (Fava, 1998), bipolar disorder (Garno, Gunawardane, & Goldberg, 2008), intermittent explosive disorder (Coccaro, 2000), temporal lobe epilepsy (Tebartz van Elst et al., 2000), Alzheimer's disease (Lai et al. 2003), dementia (Haller et al., 1989), and substance abuse (Boles & Miotto, 2003).

It may be more appropriate to consider instrumental forms of aggression a more distinguishing characteristic of psychopathy, particularly among violent offenders (Cornell et al. 1996). Impulsive violence demonstrates a lack of behavioral control consistent with antisocial features of psychopathy, whereas premeditated aggression corresponds to more complex motivational patterns reflecting impaired socialization, lack of empathy, and abnormal moral reasoning. Both impulsive and premeditated violence represent a lack of deliberation or concern for potential consequences, but rather than being limited to acute lapses in behavioral control, for psychopaths this also impinges on long-term planning and goal-oriented behavior. In non-incarcerated samples, conceivably less prone to physical violence, these deficits remain apparent, represented by forms of indirect, relational aggression (Czar et al. 2011; Warren and Clabour 2009).

As we have noted, complex behavioral outcomes like aggression, violence, and specific forms of criminality can be highly variable and context-specific. As an alternative to examining discrete behaviors directly, it can be more enlightening to focus on personality traits and motivational styles which bias behavior in specific ways and are more proximally related to the neural systems which influence them. As we examine neurobiological models of psychopathy, it will become clear that specific neural deficits differentially influence distinct features of psychopathy; and in turn, these may uniquely account for the occurrence of distinct forms of aggression.

Neurobiological Models of Psychopathy

Early recognition of abnormal psychophysiological features in psychopaths have contributed to a large body of research supporting neurobiological models of psychopathy, most of which place a strong emphasis on accounting for a form of emotional dysfunction germane to the construct. Some of the first physiological evidence for these models demonstrated that psychopaths fail to show appropriate autonomic responses in conditioning paradigms (Hare and Quinn 1971; Lykken 1957), and in anticipation of aversive stimuli (Hare 1968, 1982; Hare et al. 1978). This trend has been replicated in many different contexts, as psychopaths show relatively subdued physiological responses to emotionally evocative stimuli including sounds (Verona et al. 2004), emotional words (Williamson et al. 1991), and disturbing images (Herpertz et al. 2001). Generally speaking, the most widely replicated effects in these early studies seem to demonstrate a failure in psychopaths to form associations

between specific stimuli and potential harm, which in healthy individuals evokes a physiological cascade of events preparing the body and mind for threatening circumstances (Lang and Davis 2006; Lang et al. 2000)

A prominent extension of these findings came with the recognition that psychopaths fail to demonstrate the ordinary potentiation of startle reflexes under the influence of negative emotional contexts (Patrick et al. 1993). That is, where healthy individuals react with increased startle responses when they are primed by aversive pictures or an otherwise anxiety-provoking state, psychopaths' startle responses remain relatively constant, or even decrease in these circumstances. This rather specific feature of psychopathy has become one of the most consistently replicated physiological features of the disorder, having been documented in a wide range of samples and contexts (Anderson and Stanford 2012; Anderson et al. 2011; Benning et al. 2005; Justus and Finn 2007; Levenston et al. 2000; Patrick 1994; Sutton et al. 2002; Vanman et al. 2003).

The neural architecture supporting the potentiation of startle reflexes has been well described, and we understand that amygdala activation is directly responsible for moderating the magnitude of the startle reflex through its connections with the *caudal pontine reticular nucleus* in the brainstem (Davis 1992; Kim and Davis 1993). The demonstration that affective priming of this startle reflex was impaired in psychopaths provided strong early support for the notion that psychopaths suffered contextual deficits in amygdala activation, and thereby added support to the *Low Fear Hypothesis* of psychopathy (Lykken 1995). This model holds that psychopathy's deficits are due to a specific reduction in the brain's response to potentially threatening features of the environment. This could theoretically account for many of psychopathy's features inasmuch as this emotional reaction is an important endogenous signal for us to inhibit certain behaviors and avoid situations which might harm us. Recognizing and/or effectively avoiding potential consequences is certainly an area in which psychopaths show serious deficits; however, several other models ostensibly account for a wider range of deficits associated with psychopathy.

An alternative model emphasizes that our behavior is influenced by the proficiency with which we attend to a broad range of stimuli and allocate cognitive resources appropriately. Incorporating these elements, Newman and colleagues have proposed the *Response Modulation Hypothesis*, which suggests that the specific deficit psychopaths have is in using motivationally relevant cues to shift attention and thereby regulate behavior, but are otherwise able to process emotional stimuli at normal levels (Newman and Lorenz 2003). A subtle consequence of this demonstrated in several empirical studies is that psychopaths are capable of adjusting their behavior appropriately when their primary goal is to do so, or when punishment is the only incentive (Newman and Kosson 1986; Newman et al. 1987; Newman et al. 1990). However, in ordinary circumstances with conflicting rewards and punishments, psychopaths are impaired in shifting their attention to accommodate complex reinforcement contingencies. If one indeed had a specific cognitive deficit of this nature, it is reasonable to suspect that one consequence might be increased incidence of aggression if it satisfies a specific goal, while conflicting, alternative considerations of potential consequences remain unattended or unimportant.

It has been suspected for some time that portions of the prefrontal cortex may also be impaired in psychopaths. Prefrontal injuries have been demonstrated to result in insensitivity to future consequences (Bechara et al. 1994) and also interrupt implementation of advantageous decision-making (Bechara et al. 1997). In fact, some accounts of psychopathy have invoked the *Somatic Marker Hypothesis*, drawing directly from neuropsychological evidence that the orbitofrontal cortex is essential for online evaluation of previously encoded emotional associations (Damasio 1994). The ventromedial/ orbitofrontal region of the prefrontal cortex shares extensive reciprocal connections with the amygdala through the uncinate fasciculus. Several reports exist of damage to the orbitofrontal cortex leading to the inception of rash, aggressive behavior accompanied by disinhibited, impulsive traits (Harlow 1993; Meyers et al. 1992; Cato et al. 2004). The consequences of injury to this relatively vulnerable region are conspicuous enough to have spawned the terms *pseudo-psychopathy* or *acquired sociopathy* (Blumer and Benson 1975; Damasio 1994). However, such injuries, particularly those occurring later in life, do not result in the full spectrum of behavioral styles and core personality features of psychopathy. In order to account for traits such as shallow affect and the unique variety of aggressive tendencies typical among psychopaths, including a prevalence of premeditated-instrumental aggression, one must appreciate a wider-reaching, more complex network of brain regions which work together to integrate affective information into several cognitive processes.

With the recent addition of functional neuroimaging to the list of available measures for studying these phenomena, this literature has expanded and allowed for additional specificity in identifying compromised regions of the brain in psychopathy. In reviewing this literature, however, one should understand that examining brain dysfunction using neuroimaging techniques is a delicate process which depends as much upon properly controlling behavioral aspects of a given task as it does on the technical details for adequately modeling brain activity with advanced methods such as functional magnetic resonance imaging (fMRI). To demonstrate an abnormality in brain activity, one must carry out a specific cognitive task and show differentiable patterns of neural activation which are dependent upon a trait or category for which a theoretical basis exists for different functions. This introduces uncertainty at many levels including the appropriateness of trait measurements, efficacy of the task being implemented, and a host of statistical issues which cannot be addressed fully here. Nevertheless, progress is being made, and the accumulating literature has revealed some remarkable consistency in identifying abnormal brain regions in psychopaths (for a full review see Anderson & Kiehl, 2012).

Two prominent, contemporary neurobiological models of psychopathy which have, from their origins, integrated contemporary neuroimaging are proffered by Blair (2006) and Kiehl (2006). These models share a number of attributes but also have some important differences. These models implicate components of the limbic system—a contiguous network of brain regions which support emotional processing (including fear) and the integration of affective information into more general behavioral regulation (Papez 1937). Blair's model has emphasized dysfunction in the amygdala resulting in deleterious consequences on reinforcement learning, and attributes some of the functional disturbances in other regions such as the orbitofrontal cortex to hierarchical effects of amygdala dysfunction (Blair 2007).

Kiehl's paralimbic dysfunction model integrates evidence for more widely distributed abnormalities in the brains of psychopaths, appearing throughout an extended *paralimbic* network of brain regions, defined by cytoarchitectonic maps as an integrated system (Brodmann 1994), providing a transition from subcortical structures to higher neocortical regions (Kiehl 2006).

The most recent neuroimaging research investigating the concept of psychopathy have largely supported these models, consistently implicating several functional brain regions related to basic emotional processing and higher order integration of emotion into online cognitive processing. Dysfunction in the amygdala and ventromedial/orbitofrontal cortex are the most widely reported in studies of psychopathy, and they likely represent the most robust, easily observed abnormalities in function. Kiehl and colleagues (2001) were the first to report amygdala dysfunction in criminal psychopaths using fMRI, demonstrating reduced activity there when comparing emotional and non-emotional words. Failure to engage the amygdala and orbitofrontal cortex during tasks which require aversive conditioning (i.e. learning to associate a specific behavior with punishment) has also been consistently associated with psychopathy (Birbaumer et al. 2005; Veit et al. 2002). Reduced activity in these same regions has been associated with psychopathy in a wide variety of other tasks including the *prisoners dilemma* (Rilling et al. 2007), viewing pictures of facial affect (Gordon et al. 2004; Dolan and Fullam 2009), evaluating pictures depicting moral violations (Harenski et al. 2010), and viewing aversive photographic stimuli (Harenski et al. 2009), each of which require a form of emotional processing either directly, or in making task-related decisions.

Beyond the amygdala and orbitofrontal cortex, several other brain areas show evident dysfunction; however, due to the higher order processing and likely more specialized function of these regions, evident abnormalities there may be more difficult to demonstrate and more highly contextual. In Kiehl and colleagues' (2001) initial report of reduced amygdala function, they also reported reduced activity in the hippocampal formation, parahippocampal gyrus, ventral striatum, and in the anterior and posterior cingulate cortex. In their *prisoner's dilemma* task, (Rilling et al. 2007) also reported that psychopathy scores were associated with low activity in the anterior cingulate during trials when individuals defected against their partner in crime. Likewise, Birbaumer et al. (2005) and Veit et al. (2002) have demonstrated lower anterior cingulate activity and lower insula activity in psychopaths during aversive conditioning. Abnormally low activity in the right temporal pole of psychopaths has also been reported during an emotion-modulated cognitive task (Müller et al. 2008). Many brain regions rely on contextual emotional information to facilitate specific cognitive processes, and to such a degree that emotional processing areas of the brain are impaired in psychopaths, these diverse domains of brain activity will also show contextual deficits related to reductions in the availability of these systematic responses. Furthermore, these extensive deficits are apparent at a young age with brain imaging findings in youth with psychopathic traits (i.e. conduct disorder combined with callous-unemotional traits) largely mirroring the findings from adults described above (Finger et al. 2008; Finger et al. 2011; Jones et al. 2009; Marsh et al. 2008).

Several reports investigating anatomical features of the brain, independent of function, suggest that psychopaths' brain abnormalities are not limited to patterns of neural activation; rather, morphological brain differences accompanying psychopathic traits mirror the functional deficits noted above in primary and paralimbic regions of the brain. Reduced gray matter volumes have been reported in psychopaths' amygdala and orbitofrontal cortex and cingulate cortex (Boccardi et al. 2011; Yang and Raine 2009; Yang et al. 2010). Others have reported psychopathy-related tissue reductions in the anterior, superior temporal regions (Müller et al. 2008; Yang et al. 2011) and the insula (de Oliveira-Souza et al. 2008). In a large-scale investigation of nearly 300 adult incarcerated participants, psychopathy scores were associated with tissue reductions in the amygdala, orbitofrontal cortex, posterior cingulate, parahippocampal region, and the temporal pole (Ermer et al. 2012), and these diffuse structural differences were largely replicated in a sample of juveniles with psychopathic traits (Ermer et al. 2013).

Given the variety of brain areas implicated in psychopathy and the heterogeneity of some behavioral manifestations of psychopathy, some have preferred to itemize specific features of the disorder, relating them to more discrete neural systems. When this approach is taken, it is most common to adhere to the two factor conceptualization of psychopathy, differentiating emotional symptoms from behavioral traits and developmental course. Recent attempts to consolidate this literature point out that abnormalities in the structure and function of prefrontal cortical areas are very commonly associated with reactive aggression, impulsivity, and antisocial characteristics (Wahlund and Kristiansson 2009; Yang and Raine 2009; Bufkin and Luttrell 2005), while temporal-limbic abnormalities are more consistently associated with affective, interpersonal facets (Blair 2008; Kiehl 2006). Furthermore, in contrast to prevalent findings that psychopaths present with reduced activity in brain areas related to emotional processing, those whose aggressive acts are primarily characterized as impulsive often show *hyper* activity in the amygdala during emotional primes (Bufkin and Luttrell 2005; Coccaro et al. 2007; Herpertz et al. 2008). The relevance of these accounts is in distinguishing psychopathy from other forms of disinhibitory psychopathology, which further underscores the involvement of widespread paralimbic circuitry, predisposing psychopaths to both reactive aggression and instrumental aggression.

Etiologies of Violence in the Psychopath

Diverse etiological factors have been implicated in guiding developmental trajectories in psychopathy, impacting the manifestation of a variety of psychological and behavioral outcomes. This has contributed to descriptions of various subtypes of the construct, a prominent one referring to *primary* and *secondary* psychopaths (Karpman 1941)—a distinction originally intended to differentiate between those with inborn deficits and those who acquired these traits through poor social rearing. The field having progressed beyond a simplistic *nature vs. nurture* dichotomy, the term secondary psychopath now generally refers to those with higher levels of anxiety (Lykken 1957; Skeem et al. 2007), more prone to reactionary aggression (Patrick and Zempolich 1998) and impaired prefrontal executive function (Brower and Price 2001; Dolan and Park 2002; Ross et al. 2007). In contrast, primary psychopaths are characterized as less anxious, demonstrating low reactivity to stress and punishment cues (Hare 1982; Verona et al. 2004), normal to high executive function

(Dolan 2012; Ross et al. 2007), and whose aggressive tendencies are more typically premeditated (Falkenbach et al. 2008; Patrick and Zempolich 1998).

Among the contemporary models of psychopathy described above, it should be clear that there is a general consensus recognizing functional deficits with deleterious effects on the utility of contextual emotional information in the management of ongoing behavior. This basic deficit impacts essential cognitive functions such as aversive conditioning (Birbaumer et al. 2005), passive avoidance learning (Blair et al. 2004), and ultimately advantageous decision making when negative consequences should be a relevant consideration (Newman et al. 1987). What remains to be discussed is the extension of these deficits to aggressive behavior. Due to the prevalence of violence among psychopaths, particularly among those who are incarcerated, any model attempting to thoroughly address the development of psychopathic traits and precipitating behavior must make some account for their aggression-prone nature. Furthermore, any such model would benefit from an account of both impulsive and premeditated varieties of aggression. While many have addressed the occurrence of impulsive aggression, relatively fewer models make explicit accounts for the incidence of premeditated aggression.

Impulsive Aggression

Impulsive aggression is in some ways easier to account for in neurobiological models of behavior. In its essential form, impulsive aggression need not be considered pathological in that rapid defensive and aggressive responses are an adaptive feature of our biology, preserved through evolution. Models for impulsive aggression rely heavily on animal research engaging the mammalian basic threat circuit, which connects the medial hypothalamus and periaqueductal gray matter (Gregg and Siegel 2001). Electrical stimulation of this circuit in animals produces an immediate *defensive rage* response. The amygdala, hippocampus, cingulate gyrus, and prefrontal cortex each send projections to this circuit and can modulate the intensity of this response. In humans, abnormalities in the ventromedial, orbitofrontal cortex have often been associated with un-moderated reactive aggression, conceivably through decreased inhibitory input to this basic threat circuit (Bufkin and Luttrell 2005). Indeed, in contrast to psychopaths' prototypical reduction in primary limbic activity, individuals characterized chiefly by reactive, impulsive aggression demonstrate relative hyperactivity in the amygdala, paired with reduced regulating activity in the ventromedial prefrontal cortex (Meyer-Lindenberg et al. 2006; Coccaro et al. 2007; Raine et al. 1998).

Gene by environment developmental models have been proposed as one etiology for these behavioral tendencies (Buckholtz and Meyer-Lindenberg 2008; Meyer-Lindenberg et al. 2006); however, it remains clear that this frontal executive system is also particularly vulnerable to focal brain injuries, which carry rather informative consequences (Harlow 1993; Meyers et al. 1992; Cato et al. 2004). These injuries have been strongly associated with impulsivity (Berlin et al. 2004) short-sighted decision-making (Bechara et al. 2000), and increases in both verbal and physical aggression (Grafman et al. 1996). These consequences represent a form of behavioral disinhibition which can be seen in psychopaths; however, they do not account for the full spectrum of psychopathic traits, and

in terms of aggressive actions, fall more conspicuously under the impulsive-reactive variety. It is reasonable, then, to account for these specific outcomes in psychopathy by appealing to the local impairments recognized in ventromedial regions of the brain by recent neuroimaging studies; however, as described in the previous section, psychopaths' brain abnormalities are not isolated to this region.

Instrumental Aggression

In contrast to the abrupt and ballistic nature of impulsive aggression, instrumental aggression requires planning and enduring cognitive processes which progress uninhibited over longer periods of time. It is not limited by a unitary affective reaction, and thus may be seen as a more profound representation of a failure in governing one's behavior. Premeditated aggression exhibits a futility in what should be a veritable concert of cognitive processes aimed at preventing the behavior including the exercise of such constructs as moral judgment, empathy, and socialization. It is perhaps better represented as an abnormal set of motivational principles, which is missing any impactful consideration for ordinary social rules or the well-being of others. On the other hand, instrumental forms of aggression need not necessarily be considered pathological either. Given a socially acceptable impetus, such actions may be considered well-founded. What is immediately obvious, however, is that the rightness or wrongness of this action inherently depends on an evaluation of goals.

This form of aggression is less tractable to extant neurobiological models, as its occurrence is more difficult to reduce to a relatively simple brain system. Animal models have demonstrated that differentiable neural circuits from those accounting for impulsive aggression are responsible for this behavior. However, the behavior instigated in these models represents a species-specific form of predation, which again remains a rudimentary analog of the complex behavior we see in humans. The central amygdala connects to ventral periaqueductal gray matter through the lateral hypothalamus, and electrical stimulation of this circuit will instigate stereotyped predation behavior in mammals (Gregg and Siegel 2001). For example, a resting cat will rise, stealthily circle an anesthetized rodent (which it was previously ignoring), and deliver a single calculated strike at the prey. The degree to which this stereotyped behavior effectively represents human predatory aggression is certainly debatable, but the fact that even this simple example of differentiable behavior is governed by a distinct neural circuit is an important consideration. Certainly the motivating factors behind human instrumental aggression can take a more complex form than what we see in the cat. Rather than simply serving an instinct to hunt and eat, human predatory aggression may serve purposes as diverse and complicated as revenge, sexual gratification, monetary reward, interpersonal dominance, or even to achieve a political goal. Ultimately the diversity of such behavior in humans is not succinctly accounted for by any single neurobiological system; however, a more tractable means of considering these influences may be to examine brain deficiencies associated with the chronic disregard for the rights and well-being of others in pursuit of personal goals.

The goal here is not to universally account for all forms of premeditated aggression, but rather to describe what brain systems, that have been demonstrated to be dysfunctional in psychopaths, might influence their increased propensity for premeditated aggression, thus

distinguishing their behavior from more common forms of disinhibitory psychopathology. Blair explicitly consolidated neurobiological accounts of psychopathy into a specific model accounting for violent behavior in psychopaths and called it the *violence inhibition mechanism* (VIM) model (Blair 2001). Proposed as an explanation for the occurrence of increased instrumental aggression, VIM described a means by which proper socialization is hindered when ordinary reactions to observing other humans in pain (an aversive unconditioned stimulus) might fail in psychopaths burdened by faulty emotional responses. This model has since been expanded into the *integrated emotional systems* (IES) model (Blair 2006), which accounts for a wider variety of dysfunction related to disrupted subcortical systems in psychopathy extending to expanded paralimbic regions of the brain.

Blair maintains that impulsive, reactive aggression in psychopaths can be largely accounted for by impaired regulation of subcortical and brainstem threat circuitry by the dysfunctional frontal executive system. In contrast, the model proposes that instrumental violence and a great deal of premeditated, antisocial behavior in general are likely elevated in psychopaths due to disruption of critical mechanisms responsible for socialization. In accounting for this disruption in socialization, Blair highlights the involvement of the amygdala in the formation of critical associations, particularly *conditioned stimulus-unconditioned response* associations and *stimulus-reinforcement* associations—processes which facilitate advantageous decision-making through emotional responses. To illustrate, a healthy child finds witnessing the pain of others aversive, and eventually thoughts of acts which cause pain to others become aversive by association. When contextual cues are associated with aversive or rewarding emotional states, a kind of memory trace is established which is capable of biasing future behavior to pursue or avoid similar outcomes when one re-encounters those same contextual cues. And so, a disruption of this type of emotional learning interrupts a great deal of the socialization process as it sabotages the efficacy of direct punishment and associated aversive mental states in guiding the planning of future behavioral patterns.

Alternative Considerations

As we have already seen, there is certainly a great deal of evidence which suggests that systems governing emotion-related cognition are often dysfunctional in psychopaths, particularly the amygdala. Where Blair's model potentially falls short in accounting for premeditated violence in psychopaths is in the over-specification of one relatively limited system supporting socialization. It is somewhat difficult to rely on neuropsychological evidence to differentiate specific deficits resulting from amygdala damage because isolated, focal damage to this deep tissue is very rare. In contrast to prefrontal brain injuries, reports exploring effects of amygdala lesions point to more subtle and diverse deficits including impaired fear conditioning and emotional episodic memory (Phelps et al. 1998), impaired recognition of facial emotion (Adolphs et al. 1999), and failures in theory of mind reasoning (Stone et al. 2003), which appears to be dependent upon the age at which damage occurs (Shaw et al. 2004). Conspicuously absent from these reports are indications of acquired psychopathic features or increased instrumental aggression.

Socialization is a rather broad term—likely too broad to be monopolized by one anatomical nucleus or even a single neural system. It implies, among other things, an appropriate valuation of one's place among others in society and the appreciation of certain expectations of behavior. Certainly, the formation of stimulus-reinforcement learning is a necessary component of this process (Gao et al. 2009), but many aspects of socialization are also accounted for by behavioral control, delay of gratification, foresight, a balanced appreciation of rewards and consequences, and empathy for other human beings. Another consideration is that socialization depends greatly on the mores and folkways of our communities, biased by one's immediate environment and cultural circumstances. One might suspect, then, that the socialization process can be sabotaged by dysfunction in any of a large number of neural systems, or as Karpman (1941) and others have long suggested, even by a destructive rearing environment.

Dysfunction or abnormal development in a large number of brain regions beyond the amygdala can be detrimental to the socialization process, and potentially instigate behavioral tendencies toward instrumental aggression. When orbitofrontal injuries occur very early in life, the lingering and pervasive behavioral consequences suggest a disruption of the normal socialization processes (Anderson et al. 2000; Anderson and Moore 1995; Taylor and Alden 1997; Williams and Mateer 1992). The anterior cingulate cortex also has important roles in integrating emotion and cognitive responses in error monitoring (Bush et al. 2000) and focal brain damage here may instigate disinhibition, hostility, and difficulty with conflict monitoring and cognitive control (Hornak et al. 2003; di Pellegrino et al. 2007). Anterior portions of the temporal cortex, or temporal pole, have been implicated in highly integrative processes involving emotional and social functions including theory of mind, and damage here can produce a host of symptoms ranging from diminished empathy and hostility to inappropriate behaviors similar to what is seen in Kluver-Bucy syndrome (Olson et al. 2007). So, while the amygdala certainly plays a role in the formation of associations important for socialization, a wider yet highly integrated network of brain areas relies on these associations to effectively implement behavior which we call well-socialized.

Finally, a lingering consideration is, as Cleckley (1941) and others (Patrick 2006) have suggested, violence, per se, is not an essential quality of psychopaths. If we consider psychopathy to be primarily a disorder of personality—even one largely influenced by neurobiological factors—then discrete behavioral consequences such as violence will be a highly idiosyncratic manifestation of how one's cognitive function biases their behavior within a specific environment. A more fundamental quality of psychopaths may simply be that they fail to stop and consider the consequences of their behavior on self and others. Heilbrun (1982) provided evidence that cognitive ability may be a significant moderator of violent outcomes in psychopaths. Furthermore, in examining incarcerated samples we are more likely to observe those whose psychopathic traits have manifested in behavior aligning with more conspicuous forms of crime, readily enforced by police and courts, such as assault and homicide. It is conceivable that psychopaths who benefit from additional resources, or from environments more protected from legal intervention, may be less likely to use physical violence in pursuit of their specific goals. However, these arguments remain largely speculative, and deserve further attention in future research.

Where it seems the extant literature in psychopathy has been particularly informative is in defining aberrant neurocognitive processes that accompany abnormalities in specific brain systems. The subsequent process of extrapolating very specific behavioral consequences such as violence and antisociality from this brain data is more prone to speculation, as higher-order behavior is influenced by such a large number of physiological and environmental variables. While several models exist for the development of psychopathy, many features of these models are ultimately complementary (van Honk and Schutter 2006), and research efforts should continue to explore more precisely the impact recognizable neural deficits have on the way the brain acquires and utilizes information, the way this biases behavior in specific ways, and the varying etiological mechanisms responsible for these neural deficits.

Summary and Conclusion

Psychopathy is a disorder that can manifest itself in diverse ways, but which at its core, precipitates from deficits in emotional processing that impair cognitive functions involved in the

development of empathy, moral judgment, and sensitivity to future consequences. A common trait among psychopaths is impulsivity and lack of behavioral controls, which may prompt instances of reactive aggression; however, the complex effects of psychopathy's core emotional deficits may also instigate patterns of instrumental aggression, considered by some to distinguish psychopathy from other forms of disinhibitory psychopathology. There remains some debate about whether violence is a common feature of psychopathy *per se* or if it represents a particular manifestation of psychopathy in an etiological subset of psychopaths; however, available empirical data does not specifically address this, and this suggestion ultimately has little impact on the established recognition of essential neurocognitive deficits which influence the development of psychopathy's definitive personality traits.

Accumulating evidence supports the notion that psychopaths present with abnormal—usually reduced—activity in a closely related network of brain regions responsible for integrating emotional responses into higher cognitive functions. Among the most consistently implicated brain areas are the amygdala, orbitofrontal/ventromedial prefrontal cortex, anterior and posterior cingulate, insula, and anterior-superior temporal cortex. Each of these regions has been demonstrated, through neuroimaging or neuropsychological evidence, to be an integral part of a complex system for utilizing emotional information in behavior management and socialization. The disruption of either the initial encoding of critical associations with emotional responses or the failure of higher-order, integrative cognitive processes accessing these associations could effectively disrupt cognitive processes involved in socialization. Furthermore, these functional abnormalities may result from diverse etiological factors including genetic vulnerabilities, early damage to developing neural systems, and inadequate rearing environments.

To the degree that psychopaths are prone to impulsive, reactive aggression, this is likely related to dysfunction in online regulation of basic threat circuitry and impulse control. High

rates of predatory, instrumental aggression, conversely, are more representative of psychopathy's core affective deficits, and likely present as a demonstration of complex failures in socialization, abnormalities in motivational influences, and skewed moral judgments. Psychopaths are characteristically self-centered and generally unable to gauge of the impact of their behavior on others, while retaining the intelligence and observational acuity necessary to effectively manipulate and exploit those who are close to them. In some instances this can take the form of violence.

At the time of this writing, and very likely as you are reading this, public attention has again been drawn to a recent act of brutality. It is, at once, astonishing and familiar. When we encounter these instances of irrational violence, we are forced to remember similar instances that came before it, and we become more sensitive to the aggression that surrounds us every day. Violence and aggression are—it often seems unfortunately—part of our nature, preserved and adapted through evolution. It remains a robust part of our behavior, psychology, and neural architecture. As such, it is also vulnerable to all which can go wrong with the complex physiological systems which govern it. We have made substantial progress in psychology and neuroscience, identifying various forms of aggression and the neural systems which support them, and we continue to make progress understanding both the utility of these complex systems in adapting to our surroundings and the pathological outcomes when things go wrong. On occasions when we are forced to examine the tragic consequences of pathological aggression, we ought to be all the more motivated as students and scientists to understand its occurrence in such a way that we are better equipped to intervene and prevent it in the future.

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