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Psychopathy: Developmental perspectives and their implications for treatment

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Abstract. Psychopathy is a mental disorder marked by deficient emotional responses, lack of empathy, and poor behavioral controls, commonly resulting in persistent antisocial deviance and criminal behavior. Accumulating research suggests that psychopathy follows a developmental trajectory with strong genetic influences, and which precipitates deleterious effects on widespread functional networks, particularly within paralimbic regions of the brain. While traditional therapeutic interventions commonly administered in prisons and forensic institutions have been notoriously ineffective at combating these outcomes, alternative strategies informed by an understanding of these specific neuropsychological obstacles to healthy development, and which target younger individuals with nascent symptoms of psychopathy are more promising. Here we review recent neurobehavioral and neuroimaging literature that informs our understanding of the brain systems compromised in psychopathy, and apply these data to a broader understanding of its developmental course, ultimately promoting more proactive intervention strategies profiting from adaptive neuroplasticity in youth.

Keywords: Psychopathy, antisocial behavior, conduct disorder, callous unemotional traits, treatment

1. Establishing the construct of psychopathy

Psychopathy is a disorder characterized in part by shallow emotional responses, lack of empathy, impulsivity, and an increased likelihood for antisocial behavior (Cleckley, 1941; Hare, 1996). Psychopaths are responsible for an inordinate proportion of crime committed (Kiehl & Hoffman, 2011), and their conning, manipulative interpersonal style typically has a broad, destructive impact on the individuals' life, work, and relationships. A great deal of research suggests that the core, precipitating features of psychopathy are developmental in nature, with relatively persistent traits becoming apparent before the age of 10; furthermore, it seems these traits are accompanied by significant genetic risk factors (Viding et al., 2005, 2008). This notion has profound implications, not the least of which suggesting that neurocognitive peculiarities can hijack the development of our moral sensibility. It further suggests a basis for the failure of traditional remedial interventions on those with seemingly intractable behavioral problems ranging from conduct disorder in youth to the adult criminal psychopath. Sufficient knowledge of the neurobiological correlates of psychopathy has accumulated such that it may inform the development of new and better strategies for managing the specific deficits responsible for this altered developmental trajectory. The purpose of the present report is to review the most current neuropsychology and neuroimaging research informing our knowledge of psychopathy, noting how these data support existing neurobiological models for the disorder, with particular attention to how this information can inform better treatment and intervention strategies.

Our modern assessment and conceptualization of psychopathy has been largely based on Cleckley's

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(1941) classification of specific traits which often occur together in such individuals who, while lacking a basis for moral sensibility, retain mainstream psychological faculties such as general intelligence and memory. The construct of *psychopathy* was already common in psychiatric parlance prior to Cleckley's practice, and the recognition of viciously unscrupulous characters that lacked most other outward signs of mental deficiency has been pervasive across time and cultures. The psychiatrist Philippe Pinel (1806) used the phrase *manie sans délire* (madness without delirium) to describe this disorder over 200 years ago, but the sophistication with which we define psychopathy has advanced a great deal since then. For this we owe a debt to those who developed reliable measures for operationalizing these traits, particularly Hare's Psychopathy Checklist (PCL), now in its revised form (PCL-R; Hare, 2003), which remains the most widely used psychopathy assessment tool for institutionalized samples. Reliable measurement of the construct instigated an escalating number of investigations dedicated to defining psychopathy in more empirical ways. As such, this disorder can now be described in fairly specific neurobiological terms, which includes dysfunction in parts of the brain responsible for utilizing emotional responses, such as responding to cues indicating potential for punishment, in the modification of ongoing behavior (reviewed in depth below).

2. Refining the construct of psychopathy as a key to its etiology

In examining etiological factors contributing to psychopathy, it is perhaps important to address what is now a relatively common notion, that there may be more than one relevant developmental trajectory which contributes to psychopathic traits. That is, to the degree that psychopathy is characterized by recognizable behavioral outcomes, there are likely several distinct routes to severe antisocial behavior. An influential position on the matter was taken by Karpman (1941), who suggested that *primary psychopathy* was the consequence of an intrinsic, idiopathic deficit—what we may now consider to be genetic influences—and *secondary psychopathy* was the result of indirect factors (e.g., trauma exposure) with the behavioral consequences of each appearing quite similar, with subtle differences. Lykken (1995) mirrored this distinction invoking the term *sociopathy* in reference to those whose deficits were predicated by environmental factors such as incompetent parents and impoverished rearing environments, which would hinder proper socialization. This distinction has evolved somewhat in recent years, and rather than adhering to such strict divisions of etiology, it is often suggested that primary psychopaths are characterized by lower anxiety, general poverty of emotional expression, and tend to commit crimes which are fundamentally instrumental in nature; conversely, secondary psychopaths are more anxious, showing more emotional volatility, and commit more impulsive, reactionary crimes (Skeem et al., 2007).

So, while it may have been tempting in the past to make strident claims regarding what ultimately amounted to a *nature vs. nurture* distinction, the field has largely advanced beyond this, recognizing the improbability for one's genes or environment to play a solitary role in any given psychological outcome; rather, both will contribute significantly (see Viding, 2004). The relevant distinctions that have evolved from this initial dichotomy are perhaps better accounted for by unique neurobiological substrates for subtly different varieties of antisocial behavior and elements of personality. For instance, some early accounts of this distinction were made primarily on the basis of anxiety. Referring to primary psychopaths as *low-anxious* psychopaths and the secondary variety as *high-anxious* psychopaths, several reports supported this distinction on the basis of reactivity and arousal to stress (for a review see Newman & Brinkley, 1997). Fowles (1980) invoked Gray's (1975) neurocognitive model of the behavioral inhibition system (BIS) and behavioral activation system (BAS) suggesting that primary psychopaths have a deficient BIS, and secondary psychopaths have an overactive BAS. Still others have accounted for this distinction based on the nature of an individual's criminal activity, citing evidence that those committing primarily instrumental-predatory offenses and those committing more impulsive-reactionary offenses have dysfunction in differentiable neural systems (e.g. Raine et al., 1998; Houston et al., 2003).

Regardless of the specific taxonomy or nomenclature applied, a distinction clearly needs to be made. Those who might be characterized as secondary psychopaths, referring to highly-anxious individuals (Skeem et al., 2007) prone to reactionary-impulsive aggression (Patrick & Zempolich, 1998) and impaired prefrontal-executive function (Brower & Price, 2001; Dolan & Park, 2002; Ross et al., 2007), fit reasonably well into the current DSM-IV-TR classification of

antisocial personality disorder. Along with prefrontal impairments, these traits have often been associated with exaggerated subcortical/limbic activity (for review see Bufkin & Luttrell, 2005). In contrast, those who might be characterized as primary psychopaths are not well accounted for by DSM antisocial personality disorder, which largely ignores the core emotional deficits and personality features that Cleckley (1941) emphasized. These individuals classically present with low reactivity to stress and punishment cues (Hare, 1982; Lykken, 1957; Verona et al., 2004), more premeditated acts of violence (Cornell et al., 1996; Patrick & Zempolich, 1998), and normal to high executive functioning. Indeed Dolan (2011) recently reported that while criminal offenders with antisocial personality disorder showed specific cognitive impairments compared to healthy controls, there were no significant associations between psychopathic traits and executive impairment in these same individuals. Ross et al. (2007) found significant, but opposite relationships between executive dysfunction and primary and secondary psychopathy, with primary symptoms predicting higher overall executive functioning. Furthermore, in contrast to the exaggerated subcortical activity reported in impulsive, violent individuals (e.g. Raine et al., 1998), most evidence suggests that psychopaths have global reductions in subcortical-paralimbic circuits (discussed in detail below), which has very important implications for the unique developmental trajectories of these differentiable disorders. And so, it should be clear that while antisocial deviance features prominently in the developmental outcomes of primary psychopathic deficits, a criminal record *per se*, is neither necessary nor sufficient for the classification of psychopathy, as exhibited by the ostensive *successful psychopath* who either refrains from conventional criminal activity or at least avoids getting caught (See Gao & Raine, 2010).

Dissociating antisocial behavior from the emotional deficits, which are heralded as the core features of psychopathy, has been a longstanding pattern in most accounts of the disorder, and one that is reflected in assessment tools aimed at operationalizing the construct. For instance, the PCL-R has had a few different factor structures suggested over the years. Two (Harpur et al., 1988), three (Cooke & Michie, 2001), and four factor (Hare, 2003) models each account for antisocial behavior and emotional deficits separately. Other measures designed to operationalize psychopathic traits as defined by Cleckley have also produced two similar factors (e.g. Benning et al., 2003). Despite some subtle variation in factor elements between various analyses and measures, the concept of two factors fits well with larger theoretical models of the psychopathy construct, and thus references to a two factor conceptualization far outnumber the rest. These elements are popularly referred to as *factor 1* elements of psychopathy, *emotional dysfunction*, or *primary facets* of psychopathy—related, but not to be confused with *primary psychopathy* as Karpman described it—and *factor 2* elements, *impulsive-antisocial* characteristics, or *secondary facets*. Recent attempts to account for these two broad factors separately have appeared in cognitive neuroscience literature, and have been moderately successful. These factors have often accounted for unique psychophysiological features of psychopathy as has been commonly demonstrated with deficient acoustic startle modulation, which regularly is attributed solely to factor 1 elements of psychopathy (e.g. Anderson et al., 2011; Patrick et al., 1993; Vanman et al., 2003). With respect to developmental trajectories, Taylor et al. (2003) reported evidence for the genetic independence of these two major trait dimensions of psychopathy.

3. Psychopathy as a developmental disorder contributing to antisocial deviance

Psychopathy is a construct that has traditionally been restrictively applied to adults (Viljoen et al., 2010), mainly because the label is strongly associated with antisociality and also carries with it connotations of intractable deviance and incorrigibility. While only about 1% of the adult general adult population would be classified as such by Hare's Psychopathy Checklist-Revised, psychopaths make up around 20% of the prison population in North America (Hare, 2003). Above and beyond criminal activity, psychopaths are particularly prone to violence, demonstrating increased aggressive behavior and committing a greater number of violent attacks than non-psychopaths (Salekin et al., 1996).

Psychopathy is also a strong predictor of how likely one is to re-offend after release from prison (Hart et al., 1988; Porter et al., 2001), and it is a particularly strong predictor of violent recidivism (Cornell et al., 1996; Harris et al., 1991; Porter et al., 2009). Within one year of release psychopaths are about three times more likely to recidivate than non-psychopaths,

and four times more likely to violently recidivate (Hemphill et al., 1998). Indeed, after 10 years, 77% of psychopaths had committed a violent offense compared to 40% of the sample in a large follow-up assessment (Harris et al., 1991). Non-psychopathic offenders' violent recidivism rates appear to plateau at about 40%; however, after 20 years, it was reported that 90% of psychopaths had committed another violent crime (Rice & Harris, 1997). Furthermore, these trends remain consistent outside North America, generalizing across a variety of cultures (Hare et al., 2000).

Extending the construct of psychopathy downward into youth raises a number of important concerns. Indeed, certain perceived psychopathic traits in youth may simply be a consequence of immature behavioral controls, which usually improve with time and guidance. Further, some oppose the application of such a label due to its implications for a kind of rigid deterministic fatalism. However, an accumulating literature has provided rather strong evidence indicating that carefully defined aspects of psychopathy are apparent at a young age and are remarkably persistent across the lifespan (Lynam et al., 2007). Further evidence indicates significant genetic influences promoting the development of these traits (Viding et al., 2005). Mirroring the divergent etiological patterns noted above, there are many potential causes for behavioral disruptions in youth; but among those with conduct disorder, the most reliable and distinctive extension of psychopathy into this younger age bracket appears to be callous-unemotional traits (Frick, 2009; Frick & White, 2008).

The concept of callous-unemotional traits extends naturally to the primary emotional deficits underlying psychopathy, whereas poor behavioral control and even some social deviance are likely more natural developmental stages that some youth must grow through. These emotional deficits which have been established as a clear risk factor for adult psychopathy can, by themselves, be considered an etiological mechanism which undermines this natural developmental process by interfering with the formation of associations between disadvantageous behavior and negative affective states. Essentially, juveniles with impaired emotional responses have insufficient endogenous cues for learning to avoid conduct likely to result in punishment, embarrassment, and ostracization. Furthermore, they may lack a functional mechanism for the development of empathy.

Examining behavioral patterns among conduct-disordered youth with callous-unemotional traits, it is apparent that this combination of traits is associated with persistent patterns of severe aggression (Frick et al., 2003; Vitacco & Vincent, 2006), and is a particularly strong predictor of future violent offending (Kruh et al., 2005; Vitacco & Vincent, 2006). Forms of violence are also a differentiating factor in juveniles, with callous-unemotional youth committing more premeditated, instrumental offenses (Frick & Marsee, 2006). Juveniles with identifiable psychopathic traits demonstrate similar recidivism rates as adults. For instance, Gretton et al. (2001) noted psychopathy's predictive utility in both violent and non-violent recidivism, reporting over the course of an average of 55 months, about half of those with psychopathic traits had re-offended within the first six months (double that of comparisons), with this proportion increasing to about 70% over 5 years. Vincent et al. (2008) reported similar estimates, with recidivism rates of about 65% for those high in callous/unemotional traits with conduct disorder.

Accumulating research makes it clear that many of the personality traits evident in adult psychopaths are recognizable in youth and adolescents. As will be described below, these similarities also extend to neuropsychological, neuroanatomical and functional imaging outcomes, supporting a biological basis for the generalization of the construct to youth as a developmental disorder. Given existing neurobiological models of psychopathy, the structural and functional deficits apparent in the brains of psychopaths tell us a great deal about the kinds of learning that are impaired in the disorder, and this should ultimately be taken into account as we consider treatment and intervention strategies.

4. Neurobiological models of psychopathy

As mentioned above, the core distinguishing deficits which set psychopathy apart from the more prevalent antisocial personality disorder (and conduct disorder in youth) are emotional in nature, ultimately serving one's capacity to feel (or appreciate) remorse or shame and use cues of potential punishment or loss while governing ongoing behavior. The integration of basic emotional responses into monitoring and governing behavior through pursuit of reward and avoidance of punishment are primary motivational features that support a healthy, adaptive lifestyle—and these are in some way impaired in the psychopath. Many developmental,

cognitive, and neurobiological models of psychopathy have been proposed, and they generally have much in common in that they emphasize some abnormalities in the integration of emotional response into behavior—essentially the ability to recognize potentially aversive situations and avoid them. An early account of this is Lykken's (1995) *Low Fear Hypothesis*, which suggests that psychopaths have a subdued fear response—something that ordinarily promotes avoidance of dangerous, painful, or embarrassing situations. In terms of Damasio's (1994) *Somatic Marker Hypothesis*, it has been suggested that psychopaths have a diminished ability to utilize somatic emotional cues for the purposes of anticipating and avoiding punishment. An alternative cognitive model of psychopathy is Newman's *Response Modulation Hypothesis*, which suggests that psychopaths have a more specific deficit shifting attention to non-dominant cues, including emotional cues which capture attention automatically in healthy individuals (e.g. Newman et al., 1997).

In recent years, advances in technology have promoted an explosion of neuroimaging literature, and investigations of psychopathy have not been ignored in this movement. The accumulating data from both structural and functional neuroimaging reports have contributed to the development of two prominent neurobiological theories of psychopathy (Blair, 2006; Kiehl, 2006), which account for specific abnormalities that have been demonstrated using contemporary neuroimaging technology. These models, too, emphasize abnormalities in brain areas important for incorporating emotional information into higher order cognitive processes, namely limbic and paralimbic networks.

Blair's and Kiehl's models share a number of attributes but also have some important differences. Blair's model has primarily emphasized dysfunction in the amygdala, a primary limbic structure located bilaterally, anterior to the hippocampus in the medial temporal lobe. The amygdala is integral in forming associations between environmental cues and affective states and the activation of basic threat circuits; therefore, dysfunction there carries the consequences of failure to use affective cues to influence behavior. Blair (2007) acknowledges hierarchical effects this deficit has on other functional circuits which rely on input from the amygdala, such as the ventromedial prefrontal cortex for ongoing monitoring of behavior against established reinforcement expectancies. Kiehl's model extends this even further, accounting for a wider array of abnormalities that are apparent in the extant neuroimaging literature of psychopathy (reviewed below). These deficits go beyond basic emotional responses represented at the amygdala, and apparently impact subtle aspects of higher order processes as well. This may reflect alternative, compensatory, processes adopted by psychopaths to get along in a world without the ability to rely on essential emotional responses for support. However the degree to which these effects are indeed hierarchical, resulting from primary deficits in one brain region, such as the amygdala, or whether genetic and environmental factors produce direct effects on a wider range of functional brain regions has yet to be determined. In terms of neural development and plasticity, it is at least reasonable to suspect that these functional brain abnormalities are likely to develop over time, as a lifetime of repeated failures to integrate these intrinsic affective cues into adaptive responses governing behavior accumulate and slowly contribute to an abnormal functional organization of the brain.

5. Neuroimaging in psychopathy: A brief review

As noted above, Kiehl's paralimbic model of psychopathy accounts for abnormalities in a variety of brain structures which make up a tightly interconnected network supporting the integration of basic, sub-cortical emotional responses into high order cognitive processes (Brodmann, 1994; Mesulam, 2000). As such, psychopaths present with abnormalities in the ventral-medial areas of the prefrontal cortex including orbitofrontal cortex, in addition to core limbic structures such as the amygdala and hippocampus, and surrounding paralimbic regions such as the parahippocampal gyrus, insula, cingulate cortex, and anterior temporal cortex (temporal pole). While most of this data has been accumulated using adult samples, the smaller body of work examining these effects in youth with psychopathic traits is generally supportive of the same conclusions. Here we briefly examine this information organized by neuroanatomical regions. For a more detailed review of these data, see Anderson & Kiehl (2011).

5.1. Prefrontal cortex

The prefrontal cortex has long been suspected for its role in the development of psychopathic behavior,

due in part to a phenomenon sometimes referred to as pseudo-psychopathy, or acquired sociopathy which can result from focal damage to areas of the prefrontal cortex (Blumer & Benson, 1975; Damasio, 1994). Such damage, particularly to the ventromedial/orbitofrontal region, has occasionally been known to cause disinhibited, impulsive behavior (e.g. Meyers, Berman, Scheibel & Hayman, 1992; Cato, Delis, Abildskov & Bigler, 2004) and in experimental settings has been demonstrated to interrupt implementation of advantageous decision-making in game-scenarios with changing rules (Bechara et al., 1997); however, it should be clear that these symptoms do not account for the full spectrum of deficits recognized in psychopaths. In line with developmental perspectives of psychopathy, the most destructive consequences are actually apparent when such damage has occurred very early in life (Anderson et al., 2000), often resulting in severe and persistent deficits in decision-making, emotional volatility, and social maladjustment throughout the lifespan.

Neuroimaging investigations of psychopathy have often noted deficits in the orbitofrontal/ventromedial prefrontal cortex. Reductions in orbitofrontal gray matter have been consistently reported when comparing psychopaths to non-psychopaths (e.g. Boccardi et al., 2011; Tiihonen et al., 2008; de Oliveira-Souza, 2008), along with volume reductions in the most anterior frontopolar regions of the prefrontal cortex (Tiihonen et al., 2008; de Oliveira-Souza, 2008). It has also been reported that cortical thickness in the orbitofrontal region of psychopaths is inversely related to response perseveration—a classic behavioral correlate of psychopathy (Yang et al., 2011). These structural abnormalities closely parallel functional differences which have also been reported in prefrontal regions. For example, psychopaths have demonstrated a failure to engage the orbitofrontal cortex during tasks which require aversive conditioning, i.e. learning to associate a specific behavior with punishment (Birbaumer et al., 2005; Veit et al., 2002). Reduced activity in this region has also been associated with psychopathic traits in a wide variety of other tasks including a *prisoners dilemma* task (Rilling et al., 2007), viewing pictures of facial affect (Gordon et al., 2004), viewing pictures depicting moral violations (Harenski et al., 2010), and during an *Emotional Simon* paradigm, which requires the integration of emotional information into ongoing behavioral outcomes (Müller et al., 2008). Furthermore, these patterns are evident from an early age. Adolescents with callous-unemotional traits and conduct problems also exhibit reduced ventromedial/orbitofrontal activity during reinforcement learning (Finger et al., 2008, 2011).

5.2. Amygdala

The amygdala has also featured prominently in theories of psychopathy due to its role in forming stimulus-reinforcement associations, conditioned fear responses, and the initiation of affective states (Davis, 1997; Davis & Whalen, 2001). Damage to the amygdala indeed prevents the acquisition of conditioned autonomic responses (Bechara et al., 1999). Adolphs and colleagues have reported several effects of amygdala damage including impaired declarative memory for emotional information (Adolphs et al., 1997), and impaired recognition of negative facial emotions (Adolphs et al., 1999). More recently, it has been demonstrated that certain forms of social learning are dependent upon amygdala function. For instance, Shaw et al. (2004) reported that damage to the amygdala early in life interrupts development of *theory of mind* reasoning; that is, the ability to entertain another person's point of view or state of being. However, damage to the amygdala later in life does not result in similar impairments. This might suggest that psychopaths' apparent disregard for others' states of being might derive in part from early neuropsychological deficits in the amygdala, allowing for development of self-centered motivational patterns in the absence of a full spectrum of representational emotional states.

In related research, Shamay-Tsoory et al. (2010) investigated theory of mind deficits in criminal psychopaths and found them to be specifically impaired in affective theory of mind and not cognitive theory of mind—a dissociation which underscores the emotional deficits associated with amygdala-orbitofrontal cortex dysfunction in psychopaths.

Recent neuroimaging data have strongly implicated the involvement of the amygdala in psychopathy-related deficits. In a large-scale investigation involving nearly 300 incarcerated subjects, Ermer et al. (2011) reported reduced volumes in the amygdala, along with several other regions discussed further below. Yang et al. (2010) reported that volume reductions in both the prefrontal cortex and the amygdala were more pronounced in psychopaths with criminal convictions compared to both controls and "successful" psychopaths. In another report, volume reductions in

the amygdala were more reliably associated with the affective/interpersonal "primary" facets of psychopathy rather than the impulsive/antisocial facets (Yang et al., 2009). Task-related differences in brain activity parallel these structural findings. Kiehl et al., (2001) were the first to report amygdala dysfunction in criminal psychopaths using fMRI, demonstrating reduced activity there when comparing emotional and non-emotional words. Amygdala deficits in psychopathy have also been demonstrated during aversive conditioning (Birbaumer et al., 2005; Rilling et al., 2007; Veit et al., 2002), when viewing pictures depicting moral violations (Harenski et al., 2010), viewing pictures of facial affect (Gordon et al., 2004), viewing generally aversive photographic stimuli (Harenski et al., 2009), and when viewing fearful faces (Dolan & Fullam, 2009). Many of these reports are the same as those indicating lower prefrontal activity in psychopaths, and this likely speaks to the extensive connections between the amygdala and prefrontal cortex. Building on the pattern noted above, youth with callous/unemotional traits and conduct disorder also show lower amygdala activity when engaged in a passive avoidance learning (Finger et al., 2011) and viewing fearful faces (Jones et al., 2009). This result suggests that the disruption in affective processing evident in adults is a deficit which begins early in life, having persistent effects into adulthood.

5.3. Paralimbic and additional structures

In addition to the amygdala and prefrontal cortex, several other brain regions serve functions that are disrupted in psychopathy. For example, damage to the anterior cingulate has similar consequences as damage to the orbitofrontal cortex, i.e. disinhibition, hostility, and difficulty with conflict monitoring and cognitive control (Hornak, 2003; di Pellegrino et al., 2007). The posterior cingulate is involved the evaluation of emotional significance and self-reflective thought (Johnson et al., 2002; Maddock, 1999). Activity in anterior portions of the temporal cortex has been associated with complex social and emotional processing, including *theory of mind* reasoning and facial recognition, and damage here again mimics effects of orbitofrontal damage including unstable mood and irritability (Glosser et al., 2000; Jones et al., 2010; Olson et al., 2007; Weller et al., 2009).

Apparent abnormalities in the brains of psychopaths also extend into these regions. Reduced gray matter volumes have indeed been found in psychopaths' cingulate cortex and other paralimbic structures (Boccardi et al., 2011). Others have reported psychopathy related tissue reductions in the temporal pole (Müller et al., 2008) and the insula (de Oliveira-Souza et al., 2008). Using the largest sample of its kind, to date, Ermer et al. (2011) demonstrated that, in addition to the amygdala and orbitofrontal cortex, psychopathy was associated with tissue reductions in the posterior cingulate, parahippocampal region, and the temporal pole. Again, functional abnormalities mirror these structural abnormalities. Kiehl et al. (2001) revealed widespread activation differences in psychopaths which included reduced activity in the parahippocampal gyrus, anterior cingulate, and posterior cingulate, and ventral striatum. Rilling et al. (2007) reported that psychopathy scores were associated with low activity in the anterior cingulate during defection in the *prisoners dilemma* task, and Birbaumer et al. (2005) demonstrated lower anterior cingulate activity in psychopaths during aversive conditioning. Likewise, Veit et al. (2002) reported reduced activity in the insula and anterior cingulate while psychopaths engaged in aversive conditioning. Abnormally low activity in the right temporal pole of psychopaths has also been reported during an emotion-modulated Simon paradigm (Müller et al., 2008) and when comparing abstract and concrete words (Kiehl et al., 2004). Furthermore, some of these effects remain apparent in youths with psychopathic traits, such as abnormal function in the insula (Finger et al., 2011), the cingulate cortex (Marsh et al., 2008), and the parahippocampal gyrus (Finger et al., 2011).

It seems reasonable to suspect that some of the psychopathy related deficits apparent in paralimbic regions and extended, higher-order networks could be the direct result of a persistent lack of input from primary limbic structures; however, reduced activity in higher-order paralimbic structures does not always correspond to reduced primary limbic activity. For instance, Müller et al. (2003) utilized a simple task, viewing pictures with varied emotional content, and reported a wide range of differences between psychopaths and controls in activity throughout paralimbic structures, which included relatively increased amygdala and insular cortex activity during negative picture-viewing, but relative decreased activation in parts of the anterior cingulate and parahippocampal gyrus. Outcomes like this help to emphasize that activity in this circuit is not limited to a linear,

feed-forward pattern of influence. Complex connectivity and reciprocal influences assure communication in both directions. Therefore, it is also reasonable to suspect that the long-term consequences of psychopathy impact the efficacy of both bottom-up and top-down cognitive processes—both sensory-driven, feed-forward information processing, as well as regulative feedback from higher structures. What may ultimately be necessary to clarify these relationships is the development of detailed path models of functional connectivity, with an emphasis on how these paths are altered over time in the developing psychopathic brain.

6. Consequences and implications for treatment

The abnormalities in brain structure and function described above have severe consequences on both cognition and behavior, which can have devastating effects on one's ability to thrive in a social environment. Among the most significant of these, from a social perspective, is the toll psychopaths take on society through antisocial activity, as evidenced by the high rates of criminal behavior and remarkable rates of recidivism. As illustrated above, these patterns of delinquency are persistent from a young age, and are a conspicuous cause for concern that the developmental nature of psychopathy may place even the very young on a trajectory for incorrigible antisocial deviance. Evidence suggests, however, that such a bleak outlook may only apply when traditional intervention strategies are implemented, and even so, often belatedly, well into adulthood. In fact, alternative strategies which incorporate knowledge of psychopaths' impaired forms of social reasoning have proven to be more effective, particularly when applied in younger offenders.

Reported success rates of traditional rehabilitative intervention strategies, even within the general incarcerated population have been relatively modest (see MacKenzie, 1997). Many early reports indicated largely unsuccessful outcomes in correctional treatment (Lipton et al., 1975), an effect mirrored in juvenile offenders (e.g. Whitehead & Lab, 1989). More recently, however, others have determined that specific targeted treatments are more effective in particular categories of offenders and contexts (e.g. Andrews et al., 1990, 1994), suggesting that our best efforts may be in tailoring intervention efforts to specific groups. While this idea may have spawned selective optimism in rehabilitative efforts, an element of psychopathy which has contributed to its enigmatic reputation is that it is notoriously resistant to treatment. Indeed, therapeutic intervention and rehabilitation strategies with adult psychopaths have very often proven to be ineffective and occasionally even counterproductive (e.g. Hughes et al., 1997; Ogloff et al., 1990; Rice, Harris & Cormier, 1992; Seto & Barbaree, 1999; but see also Barbaree, 2005), leading to a generally pessimistic view among many experts as to the potential for improved outcomes among psychopaths.

A major hurdle in applying remedial interventions to the most treatment-resistant criminal offenders is that psychopaths do not believe that there is anything wrong with them; and in fact, they generally have an inflated sense of self-worth and see themselves as superior to those around them. Psychopaths are, therefore, unlikely to approach treatment efforts with any genuine commitment or desire to change; but rather, they may only use it as an opportunity to gain insight for their own manipulative strategies, including potential exploitation of administers of therapy (Hare, 1999). Indeed, psychopaths have been reported to perform better in day-to-day treatment operations and are more likely to achieve conditional release, while still re-offending at higher rates than the average parolee (Porter, Brinke & Wilson, 2009). This mirrors Seto and Barbaree's (1999) findings that inmates scoring high for psychopathy, who also demonstrated good performance in treatment efforts, had the highest rates of recidivism among all groups.

Considering the perspective of psychopathy as a developmental disorder, insofar as the associated traits and behaviors are evidently ingrained and reinforced through years of learning from a very young age, it seems rather unlikely that any traditional psychotherapeutic strategy would be capable of eliminating these traits from an uncooperative adult, who is unmotivated to change. Recognition of this fact along with mounting evidence of poor treatment outcomes have led to strong advocation for the identification of incarcerated psychopaths and directed implementation of distinct strategies which target behavioral control rather than empathy, temperament, or other cognitive factors ordinarily addressed in traditional therapeutic settings (Wong & Hare, 2001). Such strategy is likely to be more practically effective, such that effort isn't futilely directed toward changing the nature of one's well-established character. Where targeted treatments tailored for specific groups of offenders are

promisingly effective, it seems that for adult criminal psychopaths, the best strategy might be to focus on minimizing the harm they cause others by reinforcing specific behavioral patterns and self-control. That is to say, if psychopaths are uncooperative in therapy, poor at reacting to aversive cues, and are relatively insensitive to punishment, a more effective means of motivating adaptive behaviors might be to promote such behavior with measured rewards.

So, rather than promoting a defeatist attitude, we should recognize that alternative strategies may be more effective. Furthermore, successful interventions might be more likely at an earlier developmental stage when the focused reinforcement of socially adaptive behaviors is likely to have a more robust impact on the developing personality and behavioral habits of the fledgling psychopath. Recent efforts toward this end have shown some promising results. For instance, an ambitious treatment program has been designed and implemented at the Mendota Juvenile Treatment Center (MJTC) in Madison, Wisconsin, which employs intensive one-on-one therapeutic attention, several hours a day, for a minimum of six months (Caldewll & Van Rybroek, 2001). To be sure, the costs of such a program are high—the clinical staff to patient ratio at MJTC is about double that required at a standard treatment facility—but the outcomes have been positive. Reports have indicated that this intensive treatment protocol may cut violent recidivism rates in half, compared to juveniles receiving treatment as usual, e.g. standard group therapy sessions (Caldwell & Van Rybroek, 2001, 2005; Caldwel, Skeem & Van Rybroek, 2006). While these outcomes are much better than those for typically-treated adult psychopathic offenders, the administrators of this program still recognize that the outcomes have been best among juveniles with low to moderate levels of psychopathic traits, and when they have been treated for longer than one year (Caldwell et al., 2007).

This raises the question: is treatment focused even on juvenile offenders occurring early enough, or does the occurrence of severe antisocial behavior in youth indicate that the developmental trajectory of psychopathy has already succumbed to its own momentum? The age of the typical juvenile offender in treatment is between 13 and 17 years; however, investigations into the developmental antecedents of adult psychopathy have indicated the emergence of persistent psychopathic traits as early as 3 years old, and consistently earlier that 10 years old (Glenn et al., 2007; Viding et al., 2005, 2008). Insomuch as these nascent psychopathic traits represent stable patterns of attitudes and behaviors that continue into adulthood, this evidence is a strong indication that true remedial efforts may be more effective when implemented *much* earlier, prior to the onset of severe antisocial behavior in the teenage years.

7. Treating psychopathy as a neuropsychological condition

While overwhelming evidence suggests that specific neurobiological deficits undergird the development of this disorder, identifying the personality traits associated with psychopathy is still the most effective means of diagnosing it in most contexts. These traits are essentially remote, but conspicuous clues to physical/functional abnormalities in the brain, which follow a developmental trajectory that is likely more plastic in earlier stages of development. In this way, perspectives on intervention may be informed by strategies implemented in cases of traumatic brain injury (TBI) in youth, particularly injury of the prefrontal cortex. The similarities between these conditions, in fact, are more than superficial. Long term consequences of TBI vary based on the neural systems affected and the degree of plasticity inherent in those systems when injury occurs. The prefrontal cortex is only a portion of the entire paralimbic system that is affected in psychopathy; however, it is a particularly vulnerable portion considering its location and size. As noted above, when injury to this region occurs, persistent behavioral problems often result and even worsen over time, particularly when TBI occurs in very young children (Anderson et al., 2000; Anderson & Moore, 2001; Taylor & Alden, 1997). In this cohort, social and behavioral problems are the most common, persistent consequence, as opposed to intellectual and cognitive functioning (Eslinger et al., 1997; Taylor & Alden, 1997; Williams & Mateer, 1992).

With targeted attention to specific modes of development, evidence suggests these features are plastic enough to be impacted by intensive, focused treatment, and recovery or dramatically improved outcomes can occur. For example, Feeney and Ylvisaker (2003, 2006) have described successful implementation of cognitive-behavioral rehabilitation efforts in individuals who, following TBI at very young ages, exhibited increasing behavior problems including aggressive outbursts. Such behavioral problems

improved dramatically over time with focused efforts on positive behavioral supports along with identification and regulation of cognitive antecedents to aggressive outbursts. Unlike traditional treatment strategies in forensic settings, which may amount to a very limited scope of effort over a set number of hours per week, successful interventions in cases of early TBI are often implemented in a more pervasive context in the child's rearing environment. These efforts are guided by clinicians, but largely implemented by teachers, parents, and others with persistent, direct contact with the child in a variety of contexts (Ylvisaker et al., 2005). Furthermore, recent efforts at longitudinal tracking of individual progress in regaining functional neural activity following early prefrontal brain injury have demonstrated plastic reorganization and adaptation of functional circuits using fMRI (e.g. Thompson et al., 2009); however, as is a common theme in this literature, prognosis is often improved when TBI occurs in younger patients (Payne & Lomber, 2001), suggesting higher degrees of plasticity and therefore more successful compensatory re-organization of functional circuits in the brain.

Reports like these are promising indications that organic brain dysfunction is accessible to intervention strategies that are informed by an understanding of the neuropsychological obstacles to healthy development; however, such strategies have not been wholly integrated into treatment efforts combating the developmental course of psychopathy. In order to determine whether such positive effects might generalize to juveniles demonstrating behavioral indications of emerging psychopathy, it will be necessary to carry out rigorous investigations of changes in functional circuitry over the course of reasonably successful intervention efforts—such as those reported from Caldwell's group at MJTC. More informative, still, would be longitudinal investigations of youth demonstrating psychopathic traits prior to onset of severe antisocial behavior, assessing development of functional circuitry and behavioral outcomes in varying treatment conditions. The relatively recent development of functional brain imaging and its application in forensic settings has provided us with a useful tool for assessing the efficacy of such treatment and intervention strategies in new ways, and the technology is advancing rapidly. Improvements in fMRI acquisition and analysis are even providing new opportunities for innovative treatment strategies, such as real-time imaging analysis and its application in brain-computer interfacing. An exciting development in this arena has been the development of bio-feedback interfaces with fMRI implemented as an aid for effortful self-regulation of localized brain activity, which might be useful in focused therapeutic settings aimed at developing healthy functional activity in paralimbic networks of those most at risk for developing psychopathy (Sitaram et al., 2007; Weiskopf et al., 2004).

8. Summary and conclusion

Psychopathy is a developmental disorder that manifests itself as a set of core personality traits which allow one to disregard the rights of others in pursuit of impulsive, self-serving goals. Its development is undergirded by a neuropathology which distinguishes it from more typical antisocial deviance, presenting with hypofunctioning of paralimbic circuits in the brain, which ordinarily support the integration of affective information into cognitive processes governing ongoing behavior. Due to these apparent paralimbic deficits, psychopaths may have difficulty forming stimulus-punishment associations and are therefore poor at engaging in adaptive behaviors which conflict with other primary motivations. A second possibility is that if these associations are effectively formed, these deficits may render one unable to draw on these associations in hypothetical future-planning. In either case, the ultimate behavioral outcome may appear the same, and it remains possible that global reduction in paralimbic activity impacts both processes, or that distinct etiological routes may contribute uniquely to similar effective outcomes.

The developmental trajectory of psychopathy apparently begins very early, adversely impacting one's management of reward-punishment contingencies and one's ability to establish adaptive social habits, very often resulting in patterns of antisocial deviance. Early indications of this developmental trajectory include the presence of callous-unemotional traits combined with conduct problems and deviance in youth, and these apparently become more intractable as the pattern extends into adulthood. Traditional strategies aimed at remedial intervention in adults with psychopathy have not been successful, and have sometimes contributed to higher rates of recidivism. It has been suggested here that the developmental nature of psychopathy involves behaviors and motivational styles that are deeply ingrained in one's personality by adulthood, but

which remain more plastic and susceptible to focused intervention in younger ages. Effective intervention might require very early recognition of nascent psychopathic traits, despite concerns regarding relative stability of these traits and the stigma of incorrigibility associated with the label.

If we are to respond appropriately to the mounting evidence which shows that psychopathy is accompanied by structural and functional deficits in the brain, this requires adopting alternative strategies more focused on promoting adaptive re-organization of functional circuits that allow for more successful social adjustment. Furthermore, it is clear that such strategies are most successful in a younger population in which greater neuroplasticity may support these efforts. As such, these efforts might mirror successful interventions in TBI in youth, for which strategies integrating positive behavioral reinforcement and the deliberate aid of those in perpetual contact with the child have yielded particularly beneficial outcomes. A proper assessment of the efficacy of this technique would, however, require focused longitudinal studies documenting adaptive changes in brain circuitry using functional imaging techniques. If such techniques are demonstrated to be successful, it would help to confirm developmental flexibility in the outcomes of this disorder and provide a more optimistic outlook for those who are neuropsychologically impaired in their ability to acquire key social implements such as conscience, empathy, and moral reasoning.

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