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The Development of Severe and Chronic Violence Among Youth: The Role of Psychopathic Traits and Reward Processing

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Abstract

Psychopathic traits are a manifestation of a personality pathology that comprises a core affective-interpersonal dysfunction (callous-unemotional traits) and an impulsive-antisocial behavioral component. Of particular importance, psychopathic traits are associated with the perpetration of some of the most severe acts of violence, and they appear to indicate a subset of youth at risk for earlier onset, greater frequency, and persistence of violent offending. Although these youth represent a minority of the population, they commit a significant proportion of the violence in the general community. In our review, we highlight evidence of a unique neurobiological predisposition that underlies the core affective deficits and describe contemporary accounts for the developmental processes leading to the antisocial behavior associated with psychopathy. Current evidence suggests that, for this subset of youth, the structure and function of neural circuitry supporting emotion processing, reward learning, decision making, and the development of emotion related to empathy may be crucial to understanding why they are at risk for violence. In particular, a reward dominant pattern of neurobehavioral conditioning may explain how these youth progress to some of the most severe and persistent forms of violence. However, this pattern of conditioning may also be essential to the primary prevention of such deleterious behavior. We suspect that effective strategies to prevent such violence may ultimately be informed by understanding these affective and motivational mechanisms.

Keywords

Psychopathy; Psychopathic traits; Callous-unemotional traits; Violence; Aggression

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Introduction

The Centers for Disease Control and Prevention (CDC) defines youth violence as occurring among persons between 10 and 24 years old, although patterns of violence can begin very early in childhood [1]. Youth violence is a ubiquitous social problem that ranks among the leading causes of death for this age group and generates a cost to society that surpasses $17 billion annually [1–3]. In the United States, youth represent 35% of all homicide victims and approximately 50% of all homicide perpetrators [4]. However, homicide statistics alone do not sufficiently capture the severe acts of violence among youth that may result in serious injury such as physical and sexual assaults. For instance, approximately 25% of high school students report having been in a physical fight, and 18% report carrying a weapon [5]. Yet, it is important to recognize that, while many youth will engage in violence at some point in their life, most will never be violent. It is, in fact, a minority of youth who commit a large proportion of the violence, especially the most severe acts of violence. For example, in a seminal study conducted in Philadelphia, Wolfgang and colleagues [6] found that 6% of boys from a group of 10,000 were responsible for approximately 70% of all murders, rapes, and aggravated assaults. In a second cohort of 13,000 from the same city, the authors found that 7% of adolescents perpetrated 60% of murders, 75% of rapes, and 65% of aggravated assaults [7]. In a contemporary, nationally representative sample of 19,000 adolescents, Vaughn, Salas-Wright, Delisi, and Maynard [8] found that fewer than 5% of adolescents were responsible for approximately 30% of the most severe violent crimes.

From a violence prevention and reduction perspective, focusing on this minority of violent youth would appear to proffer a substantial reduction in rates of extreme acts of violence at collective levels [8–10]. This process requires that we identify those contributory factors with the greatest impact on the development and maintenance of these severe acts of violence among this minority of youth. One characteristic that has been proposed as a marker for severe and chronic violence is the construct of psychopathy and its likely adolescent precursor: callous-unemotional (CU) traits. Theorists have long argued that psychopathy traits may be an indicator for the early identification of violent and chronic offenders [8, 9, 11]. And indeed, data support the contention that psychopathy is associated with some of the most severe and chronic acts of violence across the lifespan (see “Psychopathy and Violence” section below).

In the current paper, we argue that youth manifesting psychopathic traits have biological predispositions starting at a very early age; (1) these predispositions create a skewed pattern of neurobehavioral conditioning that underlies the development of their severe and chronic violence; and (2) this same neurobehavioral conditioning mechanism offers the best opportunity to prevent the violence it engenders before its onset. In doing so, we provide the reader with a background on the association between psychopathy and acts of severe violence, developmental processes and underlying mechanisms that predispose this subset of youth to such acts of violence, and the implications for treatment and prevention strategies. This is not intended to be a comprehensive review of psychopathy, its association to violence, or the neurobiology that underlies it. Readers who want a comprehensive review of this construct beyond its relevance to violence are referred to Blair [12], Patrick [13], and Glenn and Raine [14].

Psychopathy

Psychopathy in Adults

In adult populations, psychopathy has traditionally been conceptualized as a set of interpersonal, affective, and behavioral features that include superficial charm, social poise, dishonesty, grandiosity, guiltlessness, callousness, promiscuous sexual behavior, and poor impulse control [15–18]. It has been argued that central among these core characteristics are an affective deficit and lack of empathy that predispose these individuals to antisocial behavior [18, 19]. Although Cleckley's [15] conceptualization did not restrict psychopathy to violent and criminal personalities (e.g., he included doctors, lawyers, businessman under his rubric), psychopathy is widely associated with antisocial behaviors [16, 20].[1] As one might expect, psychopathy is commonly found in forensic and clinical settings. However, growing data indicate that psychopathy manifests along a dimension of severity in adults and adolescents alike indicating the presence of psychopathic traits in the general population as well [24–27].

Psychopathy is often mistakenly thought to be synonymous with Antisocial Personality Disorder (ASPD) as defined by the American Psychiatric Association's (2013) *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) and its precursors [28]. Generally, less than one quarter of individuals with ASPD meet criteria for psychopathy as assessed by clinical diagnostic measures [16]. The conflation of these constructs is common and has arisen, in part, due to the ever-changing conceptualization and terminology related to the construct in the DSM iterations [28]. Nevertheless, ASPD generally reflects a list of socially deviant and criminal behaviors while ignoring the crucial affective-interpersonal components of psychopathy. Research indicates that ASPD correlates only moderately with psychopathy as assessed by clinical diagnostic measures [28] and provides less clinical utility than psychopathy [29]. The two conditions likely have substantially disparate implications for violent and criminal behavior [16, 30].

Psychopathic Traits in Youth

The precursor of ASPD in adults is Conduct Disorder (CD) in adolescents. Much like its adult counterpart, CD has generally overlooked the callous affect and interpersonal features that distinguish psychopathy. However, a body of empirical evidence suggests that psychopathic characteristics in children and adolescents manifest similarly to those of adults, comprising both the affective-interpersonal traits—commonly referred to as callous-unemotional (CU) traits in adolescents—and the impulsive-antisocial features [31–34]. In particular, the CU traits that mirror the affective-interpersonal dysfunction of psychopathy seen in adults, may be reliably assessed and distinguished from associated behavioral disorders (i.e., CD, Oppositional Defiant Disorder) in youth [33, 35]. It is perhaps for this reason, that the latest iteration of the DSM includes a "limited prosocial emotions" specifier for diagnoses of CD. This specifier identifies patterns in emotional and interpersonal functioning across multiple settings and relationships and is intended to denote youth with a

[1]There has been significant debate about the role of criminality in the construct of psychopathy. It is beyond the scope of this paper to review these issues here and interested readers are referred elsewhere [21–23] for a more detailed overview. Despite this debate it is generally recognized that violence is certainly a correlate if not a consequence of psychopathy [20].

more serious pattern of behavior. There is a commonly accepted distinction between youth who evince severe CD behaviors early in childhood and those who demonstrate onset in adolescence [36]. Relative to the adolescent onset group, the childhood-onset group demonstrates a more persistent and severe pattern of violent offending well into adulthood [32, 33]. The CU features associated with psychopathy appear to provide a pertinent demarcation between these two groups. Importantly, whereas a diagnosis of CD is unlikely at an early developmental age [37], CU traits can be observed and measured at a very early age prior to typical age of clinical diagnoses [38–45]. Moreover, these traits are generally stable across adolescence and into adulthood, as is the association between these traits and acts of severe violence [10]. Thus, psychopathic traits, especially those of callous affect, appear to designate a particularly violent type of juvenile who offends earlier, with greater severity, chronicity, and diversity in type of violence and victim [32, 46, 47].[2]

Psychopathy and Violence

Psychopathy predisposes individuals to acts of violence with greater frequency, severity, and chronicity and has been established as a risk factor for violence across college, community, psychiatric, adult, and adolescent populations [20, 49]. Psychopathy is associated with some of the most severe forms of violence including homicides that are instrumental, dispassionate, premeditated, sadistic, and gratuitously violent [50–55]. Moreover, psychopathic men commit a disproportionate number of sexual homicides [52]. Experts estimate that psychopaths make up 1% or less of the population, yet commit a very large proportion of the violent crime, even when compared to other violent offenders [17, 56]. For example, Coid and Yang [24] reported in their epidemiological evaluation of violence in the general population of Great Britain that individuals with high levels of psychopathic traits represented 0.7% of the population but had a population attributable risk for violence of 17.5% over a 5 year period.[3]

Pertinently, the literature indicates that adolescents evincing high levels of psychopathic traits, similar to highly psychopathic adults, are more likely to engage in instrumental violence compared to their low psychopathy counterparts [20, 57]. This distinction in type of aggression is important because instrumental violence tends to be a less common and more pathological form of violence which characterizes perpetrators at elevated risk for severe and chronic violence [20, 58, 59]. And indeed, youth with these traits appear to be at risk to commit some of the most severe acts of violence. Cope and colleagues [60] found that incarcerated adolescents who had committed murder scored significantly higher in psychopathic traits than incarcerated adolescents who had not murdered. In this sample, the CU features of psychopathy appeared to most strongly distinguish youth who had committed homicide, evincing a moderately large effect size ($d = 0.87$) [60].[4] Agar [57] found that

[2]There exist several disparate methods of assessing psychopathic traits in youth including clinician rated instruments, teacher report, parent report, and self-report instruments. Importantly, there are a number of critical issues related to the convergence (or lack of convergence) among these alternative assessment methods [48]. Although a number of measures have been validated for research purposes, expert rater devices employing clinician interview and file review are most widely used for clinical and forensic decision making purposes.

[3]The population attributable risk indicates the number (or proportion) of cases (i.e., violent incidents) that would not occur in a population if the factor (i.e., psychopathy) were eliminated. In other words, it indicates the percentage of violence that would be prevented.

among youth homicide offenders, psychopathy was associated with a higher degree of instrumentality, sexual violence, and gratuitous violence (i.e., “excessive violence beyond the level necessary to accomplish the homicide and/or caused the victim unnecessary pain and suffering”). Flexon and Meldrum [61] assessed the influence of psychopathic traits on serious violent delinquency after controlling for a number of risk factors established in the criminological literature (e.g., parenting, peers, self-control). When all covariates were entered into a regression model, the CU components of psychopathy proved to be one of the strongest predictors of violence.

In addition to severity, youth high in psychopathic traits demonstrate frequent and persistent violent offending. Prospective data suggest that the behaviors associated with psychopathy in adolescence, in particular violence and criminal offending, are stable across time and into adulthood even after controlling for numerous pertinent risk factors. For example, Lynam et al. [62] showed that psychopathic traits at age 13 predicted a variety of criminal arrests at age 18 and 26. Salekin [63] found that psychopathy was predictive of both violent and general recidivism from adolescence into adulthood even after controlling for numerous risk factors associated with criminal offending. Vincent et al. [64] found that youth scoring high on the interpersonal, affective, and impulsive features of psychopathic traits were twice as likely to commit future violent crimes (i.e., robbery, physical and sexual assault, homicide) compared to youth scoring low on all or a subset of psychopathic traits. Additionally, these youth scoring on the continuum of psychopathy traits reoffended with violence at a significantly faster rate.[5] Gretton et al. [68] investigated recidivism among a sample of adolescent sex offenders over an average period of 55 months. Using retrospectively scored psychopathy indices, they found that youth high in psychopathic traits were three times more likely to recidivate for both violent and sexually violent recidivism. Gretton et al. [69] used a similar procedure in a sample of adjudicated adolescents to demonstrate increased risk for violence over a 10 year period, even after controlling for relevant CD behaviors, historical violence, and age of onset for offending. In both samples, youth with psychopathic traits reoffended with violence and sexual violence at significantly faster rates than their low psychopathy counterparts [62, 68]. Vaughn and DeLisi [11] assessed the influence of psychopathic traits and other psychopathologies on being a “career-criminal” offender (i.e., high frequency offender). These authors found psychopathic traits to be the strongest personality factors linked to being a career offender with a significantly greater history of violent offending, $d = 1.93$ [11, 70]. Furthermore, youth scoring high on psychopathic traits started offending, had their first contact with law enforcement, and first contact with the juvenile court system at a significantly younger age [70].

[4]Cope et al. [60] reported Pearson effect sizes ($r = .40$). We converted to Cohen’s d using the formula: $d=\sqrt{\frac{4r^2}{1-r^2}}$.

[5]In another study with a longer follow-up period, Vincent et al. [65] reported that predictive power was primarily due to the behavioral features of psychopathic traits leading the authors to speculate that the predictive utility of the CU features may be greatest at younger ages and diminish post adolescence. In a related vein, Stockdale et al. [66] found that all of the psychopathy facets significantly predicted violent youth recidivism but when looking at adult recidivism the interpersonal and affective traits were not significantly associated. However, it may not be unexpected that the behavioral features would become a stronger predictor of violence as youth age and accumulate violent perpetrations [67]. This would create a scenario of criterion contamination where violence assessed as part of the behavioral features of psychopathy would have more covariance with violent behavior assessed as the criterion than would the CU features. Thus, this covariance does not negate the potential etiological role CU traits may play in the development of violence [67].

Development of Psychopathic Traits

Recent research using twin studies, molecular genetics, and neuroimaging has made substantial progress in identifying etiological factors for psychopathy including both genetic and environmental influences [14, 71] (see Viding and McCrory [34] for a review). Evidence from children and adolescents with CU traits and antisocial behavior (AB) combined indicates psychopathic traits are at least moderately heritable and modestly influenced by environmental factors [34, 71].[6] Twin studies have linked CU traits with AB by illustrating a strong genetic correlation (the degree to which traits may share a gene) between CU traits and AB [72, 73] and instrumental aggression [74]. Additionally, a number of family and parenting factors have been linked to psychopathic traits across a 40 year longitudinal investigation [75]. Likewise, poor nutrition, toxic exposures, head injuries, substance use/abuse, maternal gestational behaviors among other factors are all associated with the kinds of functional and structural brain anomalies often seen in individuals with pronounced psychopathic traits [31, 76]. Moreover, efforts to explicate the etiology of psychopathy have begun to specify familial and environmental factors that interact with genetic and biologic risk to promote development of the underlying dysfunctions [77]. Given that CU traits may stem from a combination of environmental influences and biological factors, it is important to consider how potential neurobiological abnormalities associated with CU traits may underlie cognitive and emotional dysfunctions that lead to antisocial behavior and violence.

A growing body of structural and functional magnetic resonance imaging (MRI and *f*MRI) research has identified anomalies in a variety of brain regions including the amygdala, caudate nucleus, anterior cingulate cortex (ACC), orbitofrontal cortex (OFC), dorsolateral prefrontal cortex (dlPFC), and hippocampus that are associated with psychopathic traits [14, 17, 31, 74, 78–82]. Of course it is possible that, in some cases, the neurologic dysfunctions associated with psychopathy may be a consequence rather than a precursor of psychopathy [31, 76]. However, there are a few reasons to suspect the neurobiology associated with psychopathy is the etiology rather than the sequelae of such traits. First, evidence that youth with CU traits exhibit structural abnormalities similar to those of psychopathic adults [31, 83, 84] lends support to the speculation that there is a neurodevelopmental basis for the core interpersonal-affective dysfunction in psychopathy [85]. For example, adolescents with psychopathic traits show decreased gray matter volume in a number of brain regions associated with emotion processing including OFC, bilateral temporal poles, and posterior cingulate cortex [86]. Also, similar to adults with psychopathy [81], evidence suggests youth with CU traits show increased caudate nucleus and ventral striatal gray matter volume [84].[7]

Second, Raine et al. [88] found evidence that psychopathy may be linked to structural brain abnormality reflective of prenatal neural mal-development. These authors assessed the presence of cavum septum pellucidum (CSP), a marker for fetal neural mal-development, and its relation to psychopathy and criminal offending in a non-forensic community sample. Initial analyses indicated that the group with presence of the CSP was higher on

[6]It bears mentioning that as of yet, no genetic studies have been conducted with adult populations assessed and diagnosed as psychopathic via the PCL measures (i.e., scoring above 30).

[7]Koenigs [87] offers some cautions for comparing imaging data from child/adolescent populations to adult populations—even among healthy populations.

psychopathy, and, after controlling for confounding psychopathologies, the relationship between CSP and psychopathy became substantially stronger. Additionally, there was a main effect indicating that the group with presence of CSP scored higher on aggression compared to the group without CSP. White and colleagues [89] replicated these results in a sample of adolescents finding that a large CSP was associated with both increased levels of psychopathic traits and proactive aggression compared to healthy youth. Thus, the presence of this marker for congenital neural mal-development may be associated not only with psychopathic traits, but also with the heightened levels of aggression and violent behavior that is associated with these traits. Taken together with research identifying similar dysfunction between youth and adults with CU/psychopathy traits, these findings lend support to the contention that there is a neurodevelopmental component to psychopathy.

Notably, White and colleagues [89] expanded upon their findings to show that ODD/CD youth with the presence of CSP did not differ on level of psychopathy traits or proactive aggression from ODD/CD youth without the presence of CSP. This finding indicates that CSP is not pathognomonic of psychopathy and violence, and it highlights that there are likely multiple developmental pathways to the manifestation of psychopathic traits [90]. These findings are important as they highlight the potential contribution of environmental and experiential influences to shape the development and manifestation of psychopathy [14, 71, 76, 77]. This interaction of developmental factors has important implications for prevention of psychopathy and associated violence that we discuss later.

Development of Psychopathic Violence

The Role of Deficits in Punishment Processing

For certain youth demonstrating psychopathic traits, biological and congenital predispositions for aberrant brain activity in regions that support emotion, cognition, and decision-making may predispose these youth to a pernicious form of neurobehavioral conditioning that engenders increasingly severe acts of violence. Moreover, the socialization processes for individuals with these predispositions may innately reinforce the use of violent and aggressive behavior. Several theorists have argued that the central feature of psychopathy is a deep-seated emotional deficit that not only restricts an individual from experiencing certain emotions (i.e., sadness and fear: [91]), but also prevents processing or responding to aversive cues such as threat or punishment. Consistent with this theory, psychopathic adults exhibit reduced autonomic responses to aversive stimuli [18, 92–94], and under various conditions exhibit problems learning to inhibit punished responses [95–100], corroborating the notion that a failure to appropriately respond to punishment interferes with the ability to regulate behavior.

Theoretical accounts of psychopathy and findings from functional neuroimaging have mutually informed proposed biological mechanisms that may predispose psychopathic adults to severe antisocial behavior and violence. As work conducted among youth with psychopathic traits continues to grow [101–103], studies reveal a pattern of hypo-responsivity in the amygdala, ventromedial PFC, OFC and striatum that is associated with aberrant emotion processing, empathy, and learning. In an effort to identify potential neurobiological markers of psychopathy that may lead to violence, Blair [19, 31] has

integrated theoretical and neurocognitive accounts of psychopathic dysfunction that implicate several key brain regions. More specifically, Blair [31] outlines two impairments that are prevalent among youth with psychopathic traits: the first involving reinforcement learning and decision making gives rise to the second which involves impaired emotional empathy. It is important to note that each of these processes implicate a number of brain regions and may respectively recruit overlapping regions. However, the specific aspects of neural dysfunction that youth and adults with psychopathic traits exhibit in comparison with healthy controls has helped to provide further clarity to these broader cognitive impairments.

One of the impairments linked to psychopathic traits, and in particular, to CU traits, is impaired reinforcement learning; from this perspective, abnormal classical and operant conditioning lead to a selective increase in antisocial and violent behavior. Part of Blair's [31] model argues that because the amygdala is critically involved in the formation of connections between conditioned stimuli and unconditioned responses (classical conditioning), as well as between conditioned responses and reinforcement contingencies (operant conditioning), it is the major substrate for the development and maintenance of violence. Abnormal responsivity in amygdala undermines stimulus-reinforcement associations because it sends aberrant information to other brain regions that are involved in processing reinforcement expectations such as the ventromedial prefrontal cortex (vmPFC), rostral insular cortex, and OFC. Notably, all of these regions have dense anatomical connections to the amygdala, with strong reciprocal connections in the basolateral subdivision of the amygdala [104–106]. Deficient stimulus-reinforcement processing is said to occur due to impaired integrated functioning (functional connectivity) in amygdala and OFC among youth and adults with psychopathic traits [96, 102, 103]. The evidence for reduced responsiveness of the amygdala to aversive stimuli during aversive conditioning in individuals with psychopathic traits [92, 93, 107, 108] also points to a role for the amygdala in impaired reinforcement learning (which also has implications for empathy). The instinctively punishing distress cues emitted by the victims of violent acts (e.g., facial expressions and vocalizations of fear, distress, pain, etc.) would fail to dissuade children with this sort of neural dysfunction from acting aggressively. Indeed, data indicate that children exhibiting a high degree of psychopathic traits (in particular CU traits), much like their adult counterparts, are less behaviorally responsive to and less able to learn from punishment cues under many conditions [31, 32, 107].[8] Presumably, neurobiological abnormalities in amygdala and OFC compromise reinforcement learning and prevent children with psychopathic traits from learning associations between the negative emotional responses of others and their own aggressive behaviors.

In addition to impaired reinforcement learning, functional brain abnormalities associated with psychopathic traits affect responsivity to emotional cues. Reduced responsivity to emotional cues, in turn, disrupts normal empathic socialization. In contrast to cognitive empathy that involves understanding the thoughts and intentions of others, emotional

[8]Recent studies suggest that it is under specific conditions involving competing contingencies or task demands that individuals with psychopathic traits demonstrate deficient amygdala response to noxious stimuli or punishment cues [109]. Because functioning in the real world almost always presents competing contingencies, there are likely many conditions in which psychopathic traits predict a reduced neural response to punishment cues.

empathy involves recognizing and processing emotional expressions or emotional states of other individuals [31, 110].[9] Emotional empathy functions to enable the observer to perceive and translate emotional cues communicated by others (such as facial expressions, vocal tones or physical postures). Signifying that the basic foundation of emotional empathy is compromised, youth with psychopathic traits exhibit poor recognition of emotional cues [112] and diminished autonomic and amygdala activity in response to fear, [10] sadness, happiness or pain in others [102, 103, 120, 121]. These results illustrate that youth with psychopathic traits show reduced responsivity to happiness, and more importantly, distress cues in others [122]. Moreover, similar investigations demonstrate that this is a cross-modal impairment, such that youth (and adults) with psychopathic traits have difficulty recognizing vocal, facial and body postures that signal emotional expression and distress [19, 123–125]. Of note, these deficits in emotional empathy are particularly linked to the CU component of psychopathy [31].

To the extent that psychopathic individuals have reduced capacity to register distress in others, they are far less likely to understand the emotional consequences for others of their own behavior. Also, consistent with reduced emotional empathy, youth with psychopathic traits show diminished responses in brain regions associated with emotional responding to another's pain (such as amygdala and rostral cingulate cortex) when viewing images of pain-inducing injuries [101, 126–128], and report they are less concerned that aggression will cause suffering to victims [129]. Notably, this impairment does not apply to all emotions, as youth and adults with psychopathic traits exhibit typical responses to expressions of anger and disgust [112]. In addition to deficient emotion processing, youth with CU traits exhibit reduced activity in the vmPFC, ACC, and anterior insula when using emotion cues to predict the behavior of others [101, 128]. Given that successful social cooperation and behavioral regulation depends on the ability to recognize and interpret cues from others, evidence of dysfunction across several of these critical underlying neurobehavioral markers hinders the normal development of empathy and highlights a compelling association between CU traits and violent behavior.

[9]In contrast to impaired emotional empathy, youth with CU traits show no impairments in cognitive empathy which is the ability to understand the intentions or beliefs of others in order to predict their behavior [31]. However, Brook and Kosson [111] reported impairments on a laboratory measures of cognitive empathy among adult psychopaths.

[10]Evidence suggestive of a selective emotion processing deficit in combination with diminished amygdala activity has strengthened theories that psychopathy is associated with a "fear deficit". However, the underlying emotion deficit in psychopathy and accompanying neurocognitive dysfunction has been the subject of a widely studied and controversial debate in the field of psychopathy research. Although it is generally agreed that psychopathy and CU traits are associated with a lack of empathy, there is ongoing discussion as to whether the emotion processing deficits linked to reduced amygdala response in psychopathy are absolute or moderated by attention [109, 113, 114]. Consistent with the former interpretation, there is evidence that children with CU traits exhibit reduced activation in amygdala in response to fearful facial expressions [102, 103] and affective Theory of Mind scenarios (e.g., imagining the emotional states of others) [101]. Consistent with the latter interpretation, instructing psychopathic youth and adults to attend to important features of emotional cues (such as the eyes in fear expressions) ameliorate fear recognition deficits and diminished amygdala response [115–118]. In support of both of these theories, a recent neuroimaging study reported that youth with psychopathic traits show reduced recruitment of brain regions in the dorsal endogenous attention network in response to fearful facial expressions [115]. These findings corroborate integrated neurobiological theories of psychopathy that implicate both amygdala dysfunction and downstream attention processing [119]. Even among controversies regarding whether psychopathy is marked by a distinct impairment in processing fear or whether attention modulates the manner in which individuals respond to emotion cues, there is clear evidence that psychopathy is associated with marked dysfunction in emotion-relevant brain circuitry and the ability to demonstrate emotional empathy towards others.

The Interaction of Positive Reinforcement and Punishment

The deficits in punishment processing may further be exacerbated by the function of reward processing (i.e., positive reinforcement)[11] in youth with psychopathy traits. Aggression and violence can be an effective tactic to obtain natural and subjective rewards (e.g., food, sex, money, drugs, etc.). In the absence of punishers for such behavior, the successful attainment of reward would serve to positively reinforce such tactics [130, 131]. This of course assumes that the neural reward systems in youth with psychopathic traits are functionally intact. But the data on reward functioning and psychopathy, at first glance, are hard to reconcile. Among adults, some data suggest psychopathy may be associated with hyper-responsivity to reward in the ventral striatum and ACC. For example, the striatum, which is associated with impulsive and reward dominant behavior, may be significantly enlarged in adults with a high degree of psychopathic traits [81, 133]. Additionally, some have found increased activation in the ventral striatum and ACC suggesting hyper-responsivity to reward [107, 108]. However, these data are from non-psychopathic community participants and the findings are limited to the impulsive behavioral components of psychopathy rather than the affective/interpersonal facets. Blair et al. [95] found diminished punishment processing but intact reward processing in psychopathic men but later reported impairments in both punishment and reward processing in a different sample of psychopathic men [134]. Notably, the data on reward processing among youth is somewhat more consistent but limited. A study using a passive avoidance task (i.e., participants learn to respond to a rewarded stimulus and not to respond to a punished stimulus) demonstrated that youth with CU traits showed more behavioral commission errors to stimuli associated with punishment and showed less OFC responsivity to reward [135]. Likewise, Marini and Stickle [136] found an inverse correlation between the CU traits of incarcerated youth reward responsivity on a laboratory task. Centifanti and Modecki [137] reported a similar finding among a large sample of high-school students.

However, considering the diminished neural and behavioral responses to punishment in psychopathy, it may not be that persons with these traits are necessarily hyper-responsive to reward compared to non-psychopathic persons [99]. Instead, it may be that the ratio of their neurological reward sensitivity to their punishment sensitivity (or lack thereof) is greater. That is, in the absence of punishment sensitivity, normal or even blunted reward sensitivity would still result in a net reward (i.e., positive reinforcement). In fact, Pujara et al. [138] found that the severity of psychopathy in adults was associated with the ratio of reward to punishment sensitivity when measured via activation in the ventral striatum as well as when measured via self-report surveys.

Glenn et al. [139] reported a similar reward dominant functioning of the endocrine system wherein, a high basal testosterone to low cortisol reactivity ratio was positively correlated with the degree of psychopathy. Cortisol, a hormone released as part of the hypothalamic–pituitary–adrenal (HPA) axis in response to stressful events or cues, acts upon the amygdala

[11]Reward has often been thought to reflect a hedonic state or conscious state of pleasure. However, competing theories suggest that the exact nature of reward at the neurochemical level is not clear and may represent motivation, wanting, liking, or strictly reflect positive reinforcement defined as increased probability of behavior occurring in the future [130–132]. We use the term reward to reflect reinforcement (i.e., increases the likelihood of the behavior occurring in future) without suggestion of the means by which it does so (i.e., motivation, wanting, liking, anhedonia, etc.).

to potentiate fear and sensitize individuals to punishment. Conversely, testosterone, which is secreted within the hypothalamic–pituitary–gonadal (HPG) axis, is associated with reward seeking behavior. Testosterone inhibits the function of HPA axis and associated autonomic systems, reducing sensitivity to punishment. In contrast, cortisol is thought to increase sensitivity to fear through suppression of the hypothalamic–pituitary–gonadal (HPG) axis [140]. Notably, this hormonal imbalance has long been associated with violent and aggressive behavior [140].

Additionally, behavioral data with youth appear to support this theory of reward dominant reinforcement. Hawes and Dadds [141] reported that boys with elevated levels of psychopathic traits were less responsive to punishment methods of parenting and displayed less negative affect in response to punishment than did boys low in psychopathic traits. Yet, the groups did not differ in response to reward-based parenting methods. Pardini et al. [142] found that psychopathic traits were associated with an increased focus on the benefits of aggression and a decreased focus on the consequences. Similarly, laboratory paradigms suggest children with psychopathic traits assign greater focus and value to reward outcomes than punishment outcomes [32].

Importantly, CU youth and psychopathic adults demonstrate another critical deficit in reinforcement learning: reversal learning [31, 96, 143, 144]. In reversal learning tasks, individuals first learn to respond to a specific stimulus to obtain a reward, but then the reinforcement contingency changes (correct responses are no longer rewarded) so that individuals must learn to respond to a different stimulus to earn the reward. Such reversal learning tasks also utilize punishment for incorrect responses during the initial learning phase and following contingency reversal phase. In other words, responses that were initially rewarded are now punished following the contingency reversal. Finger et al. [96] reported that children with CU traits showed increased activity in vmPFC and decreased activity in caudate following unexpected punishments for a previously rewarded response (demonstrating vmPFC and caudate activation in the opposite direction to that of control youth). In other words, youth with CU traits were locked into their initially rewarded behavioral response, even after that response was no longer rewarded and was now punished. In reference to violent behavior of youth with psychopathy traits, this would mean that the initial aggressive behaviors committed in early childhood would be reinforced by their reward dominant conditioning patterns, and this behavior would persist even when the consequences for such behavior intensified and the rewards dissipated. [12]

Understanding these general mechanisms of reward dominant processing in the brain may be critical to understanding why many youth with psychopathic traits progress to the perpetration of severe acts of violence with great frequency. When a behavior is reinforced there is an associated neurochemical response in dopaminergic neurons in the ventral tegmental area (VTA) and nucleus accumbens (NAcc), regions of the brain associated with reward processing [131]. This contiguous neurochemical response positively reinforces the

[12]A complete review of the empirical literature on cognitive dysfunction in psychopathy is beyond the scope of this review; however, there is also substantial evidence of deterioration in cognitive efficiency under approach motivation conditions [29, 145] and for specific impairments in processing peripheral stimuli under approach motivation conditions [99, 109] which are likely to further reduce the ability to modify approach behavior based upon punishment cues.

behavior which increases the probability of reoccurrence [130–132, 146]. Violence and aggression, given a deficient experience of innate punishers (i.e., distress cues from the victim), are very effective tactics for attaining those things deemed desirable. Therefore, when young children with psychopathic predispositions see another child with something they want (e.g., a coveted toy or food), they may use aggression to obtain the desired object. Given their reduced experience of punishment from distress cues under such conditions, they will only be rewarded, both by the tangible gain stimulating pleasure and a dopaminergic response in the reward circuitry of the brain.

The effect of dopamine activation following a reinforced behavior is to stamp in the memory traces of the stimulus and the response association [130, 146]. Therefore, with repetition of the reinforcement, the association between violence and tangible reward becomes more locked in. Rewarded behaviors tend to persist and increase over time, and the behavioral sequences associated with reward become over-learned [146]. These behavioral sequences become well-rehearsed and automatized. Moreover, with repetition, the dopamine response transfers from the actual reward to predictive cues associated with reward [130, 131, 146, 147]. For example, neuroimaging studies indicate that, with repetition, brain activation in dopaminergic areas increases in response to cues that signal opportunities to perform rewarded responses [148]. As such, predictive cues of reward (e.g., a peer with a coveted possession, a sexually desirable individual, someone withdrawing cash from an ATM, etc.) can activate these now well-rehearsed behavioral sequences previously used to obtain reward (i.e., violence) via dopamine stimulation in the VTA [131, 146]. Thus, in this scenario, a youth with psychopathic traits possessing this reward-dominant neural conditioning pattern would begin to experience a dopamine response in association to the act associated with the attainment of reward (i.e., violence). Therefore, the act of violence itself may come to elicit a dopamine response and, as a result, may become intrinsically reinforcing [149, 150]. Animal models support contentions that increased frequency and severity of aggression may result from temporally contiguous increases in dopaminergic activity which serve to reinforce and strengthen violent behavior [132, 149, 151, 152]. Thus, this deleterious reinforcement cycle would indicate that CU youth would become violent more frequently and with greater severity as they engage in violence: violence begets violence. This process could explain why psychopathic individuals persist in responding when an initially rewarded behavior is no longer extrinsically rewarded [96, 144].

Pertinently, acts of violence that occur concomitantly with substance use may be increasingly reinforced because enhanced dopamine neurotransmission increases incentive or motivational effects of reward cues [131]. Nearly all forms of drugs work to stimulate dopamine release and increase the strength of excitatory synapses on dopamine neurons of the VTA [132, 146]. This mechanism is germane because psychopathy is associated with pathological substance use and abuse [16, 17]. Consequently, it seems feasible that youth with these traits would be more likely to engage in violence while intoxicated by illicit substances. Similarly, sexual acts stimulate dopamine release [132, 146] and when paired with acts of violence, may have significantly greater reinforcement properties. Therefore, the relationship between sexual violence and dopaminergic reinforcement is particularly relevant as it relates to psychopathy, because it is associated with disproportionate perpetration of sexual violence, including sexual homicides [52, 57, 153]. Thus, through

interplay of operant and classical conditioning processes, these acts of violence perpetrated by highly psychopathic individuals may be more strongly reinforced. This may explain psychopathy's unique association to the use of violence for secondary gain, that is, instrumental violence [20]. Furthermore, under these imbalanced conditioning circumstances, reinforcement would be likely to transfer from the primary reward to the actual act of violence. In other words, violence would become self-rewarding and consequently self-perpetuating. Indeed, psychopathy has been associated with the derivation of pleasure from violence and the use of gratuitous violence in multiple samples [52–54, 154]. Foulkes et al. [155] reported positive correlations between all facets of psychopathy and enjoyment of callous treatment of others in two diverse samples. Neuroimaging studies with youth and adults with psychopathic traits report increased activity in the ventral striatum in response to viewing others in pain [126] or imagining others in pain [129] suggesting that psychopathic individuals not only show reduced empathy for others in pain, but may derive some form of pleasure or reward from seeing others suffer. This activation of ventral striatum in response to others' pain appears to be linked solely to the callous affect and interpersonal components of psychopathy [127, 156]. In sum, reward plays a role in violent behavior, and increasing evidence of the association between impaired emotion processing and reinforcement learning among individuals with psychopathic traits highlights a potential target for treatment and intervention.

Implications and Conclusions

In our review, we have highlighted evidence of a unique biological predisposition that underlies the core affective deficits and developmental processes engendering the violence associated with psychopathy. Contemporary evidence suggests that, for this subset of youth, the structure and function of neural circuitry supporting emotion processing, reward learning, decision making, and the development of emotion related to empathy may be crucial to understanding why they are at risk for some of the most severe and persistent forms of violence, and how to prevent it.

Importantly, there is warranted ethical concern about the possible consequences and stigma of falsely identifying youth as future violent offenders. Therefore, identified indicators of youth at risk for severe violence must have an established empirical base and clearly delineated developmental processes that engender such violence so that we may develop and implement the most effective and ethical prevention and treatment strategies for the individual and society as a whole. Psychopathy has long been linked to social deviance and acts of violence in adult populations. In children and adolescents, psychopathic traits, in particular the CU features, appear to indicate a subset of youth at remarkable risk to start offending early and persist significantly longer than their peer counterparts. Moreover, youth with CU traits progress to commit some of the most severe acts of violence with greater frequency and persistence [32, 46, 70]. Of course, it is a minority of youth that perpetrate such extreme violence [6, 7]. Likewise, it is a minority of youth that demonstrate significant elevations of psychopathic traits [64, 157]. Although continued empirical investigation is necessary, evidence suggests these two groups of youth significantly overlap [9, 11, 46]. Considering the substantial proportion of population levels of violence for which they may

be responsible [6–8, 11], we suspect focusing violence prevention efforts within this minority population could yield substantial reductions in overall rates of violence [9, 24].

Above, we have laid out the evidence suggesting that youth with psychopathic traits, in particular CU traits, appear to have specific neurobehavioral patterns of conditioning that yield aberrant socialization and inherently reinforce their use of aggressive and violent behavior [17, 19, 31]. Moreover, this imbalance in reinforcement may predispose these youth to a virulent cycle wherein violence becomes intrinsically rewarding and propagates increasingly severe and frequent acts of such violence [7, 50, 57, 60]. Yet, this deleterious conditioning cycle may actually proffer the strategies to prevent their violence. Considering these youths' unique predispositions for conditioning of reward relative to punishment, we suspect violence prevention and reduction strategies that focus on heavily positively reinforcing prosocial behaviors may be most effective [34]. For example, the Mendota Juvenile Treatment Center is one of the only programs that have shown to reliably reduce rates of violence by youth with elevated psychopathic traits (see Reidy et al. [49] for a review of treatment studies). This program at the MJTC reduces focus on sanctions and instead implements a token-economy-like program that inherently positively reinforces prosocial behavior [158]. Notably, Caldwell and Van Rybroek [158] note that the MJTC appears to have the most pronounced effect on the most serious violent offending: whereas youth in the comparison group committed 16 homicides during the multiple year follow-up period, youth treated at the MJTC committed none. In fact, evidence suggests that the MJTC may not only influence reductions in violence but may also be associated with reduction in psychopathy traits themselves [159].

In a related vein, programs that incorporate parenting strategies of positively reinforcing prosocial behaviors and decreasing the use of punishment tactics may potentially reduce psychopathic traits in youth [160, 161]. While these particular programs require further evaluation for their efficacy to reduce the actual violence associated with psychopathic traits, they may offer promising strategies to curb such behavior among these youth. Adapting programs like the MJTC to develop primary prevention strategies for callous-unemotional youth may prove to be a most effective method for preventing their aggression including the most severe acts of violence. The goal of primary prevention, to prevent violence before its onset, generally requires implementing prevention strategies at a young age [155]. Early evidence suggests the CU features of psychopathy traits can be reliably identified in young children as early as 3 years old [38–45].[13] And, these traits identified at 3 years, predict elevated and stable aggressive behavior including instrumental aggression [38, 39]. This is important because it substantiates both the early onset of aggressive behavior and the consequent need to start primary prevention very early. But more importantly, it demonstrates we can feasibly develop indicated[14] primary prevention strategies tailored to youth with psychopathic traits and reward dominant processing to be implemented as early

[13]We should reiterate at this point the heterogeneity among measures of psychopathic traits in youth [48]. Although a number of studies have measured CU traits in children under the age of 6 [38, 41–45], the nascent state of this literature indicates the need for further refinement before such assessment methods could be put into practice.

[14]In terms of youth violence prevention, universal programs are those administered to everyone within a defined population regardless of risk; selected programs are directed to a population who is at-risk for youth violence but has yet to engage in violent behavior; and indicated programs are those that target individuals who show early signs of engaging in violence [162].

as preschool age. In fact, it is likely that all youth would benefit from such a prevention strategy and thus could be developed as a universal primary prevention program. But the very predispositions that make youth with psychopathic traits prone to severe and chronic violence (i.e., reward dominant reinforcement) may also make them the most responsive to this form of primary prevention. Evidence suggests youth with psychopathic traits tend to persist in behaviors that were initially rewarded even after they are no longer rewarded [96]. Thus, it seems that locking in prosocial behavior with intense and pervasive positive reinforcement starting at a very early age may inoculate youth with psychopathic traits against the development of the antisocial and violent behavior that would otherwise be reinforced. It bears mentioning that although there are a number of "natural" rewards that will directly act on neurotransmission (e.g., food, drugs, sex), generally speaking, what constitutes reward is highly subjective and will differ by individual. For example, evidence suggests that the affective deficits of psychopathy are related with deriving less pleasure from social relationships but positively correlated with sexual relationships and treating others callously [155]. In other words, children and adolescents with CU traits will be less likely to respond to social rewards and may require some alternative method of reinforcing their behavior. Thus, positively reinforcing prosocial behaviors will require identifying the subjective reinforces specific to each youth.

Importantly, although we have emphasized the bio-developmental component of psychopathy, it is necessary to reiterate that genetic studies demonstrate heritability only explains about 50% of variation in the expression of psychopathic traits [34] indicating that environmental factors play substantial role in the development of psychopathy and its consequences [163]. Environmental factors and life experiences will interact with genetic predispositions to alter the manner in which genes and behaviors are ultimately expressed [77]. But to be clear, we refer here to interactions of epigenetic form and not the statistical interactions of gene and environment. Epigenetics is the process whereby environmental processes alter gene expression without altering DNA [164–166]. Such interactions are inherent to our argument for primary prevention strategies that shape prosocial behavior through extensive positive reinforcement.

Cultivating environments to have an epigenetic impact would likely require a multi-level social-ecological approach encompassing parenting, family, and school based intervention, potentially even community based strategies. While this is seemingly a high resource investment, there is reason to suspect the returns would far exceed the investments. For example, each injurious assault that is prevented translates to a savings of $7,166–$165,389 [167] depending on level of care necessary.[15] Each homicide prevented equates to a savings of more than $1.5 million [167]. And, these estimates include only medical costs and lost wages. They do not address savings to the criminal justice system. Caldwell, Vitacco, & Van Rybroek [168] reported that the MJTC yielded a return of $7 for every $1 invested on these youth with extensive violence histories. Fagan and Catalano [169] found that resource intensive prevention strategies on average had a return of $4 to $5 for every $1 invested.

[15]These numbers are updated to 2016 dollars from 2010 dollars using the Consumer Price Index.

Importantly, these estimates are conservative as they don't account for the prevention of lifetime offending and perhaps, more importantly, the suffering of the victims.

At this point we must make explicit that we are not arguing that the presence of psychopathic traits is pathognomonic of violence. Much as psychopathy traits manifest on a continuum of severity [26, 27], so too will the degree of reward dominant reinforcement and neurobiological dysfunction each child demonstrates. These varying levels of function and dysfunction will interact with the multitude of different experiential factors and additional personality factors of each youth. Hence, not every youth presenting with psychopathic traits will progress to the intrinsically rewarding form of violence we have described herein. Indeed, there is reason to believe that some youth with these traits will desist or never become violent [170, 171]. It is commonly believed that there may be subtypes of psychopathy with disparate associations to violence as well as different etiologies and outcomes [172, 173]. In a related vein, we do not suggest that the markers and pathways we identify represent the only pathways to the expression of psychopathy and its associated violence. Based in part on the principle of equifinality, which asserts that there may be multiple developmental trajectories that lead to the same pathological outcome [90], we suspect there are a number of interactions among biologic predispositions, environmental factors, and social experiences that all may result in high levels of psychopathic traits, as there are for all types of psychopathology [1, 31, 90]. Likewise, there are likely a number of causal factors and pathways to severe violence perpetrated by youth. We believe this point is critical because understanding these developmental pathways will allow us ultimately to alter them.

Youth violence represents a substantial burden to society contributing nearly half of all homicides in the U.S. and costing society billions of dollars each year [1, 4]. Acts of extreme violence, although committed by a minority of youth, are prevalent, costly to society, and merit attention by researchers and practitioners. Consequently, reducing population levels of violence ultimately means we must prevent the onset of violence in youth [1]. Understanding those developmental processes that lead some youth to perpetrate acts of severe violence may offer us evidence of effective strategies and critical time periods to intervene. Identifying psychopathic traits in youth and altering their expression may be one of the most critical avenues on which to intervene.

Summary

In this paper, we have laid out several arguments with evidence to support our claims. First, although it is only a minority of youth who will evolve into violent individuals, it is, in fact, only a minority of youth that commit the majority of violence, especially the most severe acts of violence. This has significant implications for prevention purposes as it suggests focusing on a small group of youth may proffer the greatest reductions in collective levels of societal violence. Second, psychopathy traits (commonly referred to as callous-unemotional traits in youth) are one potential marker of this minority of youth that will progress to violence. Third, herein, we have presented evidence of an underlying neurobehavioral conditioning pattern that shapes the developmental experiences of these youth starting from a very early age. In particular, these youth who demonstrate a reward-dominant pattern of

conditioning may become inherently reinforced for committing the most severe acts of violence. Fourth, we believe that this neurobehavioral conditioning process holds the key to ultimately preventing these and other youths' violence and shaping potentially prosocial outcomes. Finally, we want to stress that we do not argue these traits are pathognomonic of violence and youth should not be labeled or stigmatized as such. There are a myriad of factors that interact to ultimately shape the developmental outcomes of all individuals.

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