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Biological explanations of criminal behavior

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ABSTRACT

There is a growing literature on biological explanations of antisocial and criminal behavior. This paper provides a selective review of three specific biological factors – psychophysiology (with the focus on blunted heart rate and skin conductance), brain mechanisms (with a focus on structural and functional aberrations of the prefrontal cortex, amygdala, and striatum), and genetics (with an emphasis on gene-environment and gene-gene interactions). Overall, understanding the role of biology in antisocial and criminal behavior may help increase the explanatory power of current research and theories, as well as inform policy and treatment options.

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A growing body of literature has indicated the importance of considering neurobiological factors in the etiology of antisocial and criminal behavior. Behaviors, including criminality, are the result of complex, reciprocally influential interactions between an individual's biology, psychology, and the social environment (Focquaert, 2018). As research progresses, the misconception that biology can predetermine criminality is being rectified. Elucidating the biological underpinnings of criminal behavior and broader, related outcomes such as antisocial behavior can provide insights into relevant etiological mechanisms. This selective review discusses three biological factors that have been examined in relation to antisocial and criminal behavior: psychophysiology, brain, and genetics.

Psychophysiology

Psychophysiology, or the levels of arousal within individuals, has become an important biological explanation for antisocial and criminal behavior. Two common psychophysiological measures are heart rate and skin conductance (i.e. sweat rate). Both capture autonomic nervous system functioning; skin conductance reflects sympathetic nervous system functioning while heart rate reflects both sympathetic and parasympathetic nervous system activity. Blunted autonomic functioning has been associated with increased antisocial behavior, including violence (Baker et al., 2009; Choy, Farrington, & Raine, 2015; Gao, Raine, Venables, Dawson, & Mednick, 2010; Portnoy & Farrington, 2015).

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Longitudinal studies have found low resting heart rate in adolescence to be associated with increased risk for criminality in adulthood (Latvala, Kuja-Halkola, Almqvist, Larsson, & Lichtenstein, 2015; Raine, Venables, & Williams, 1990). However, there is likely a positive feedback loop whereby blunted autonomic functioning may lead to increased antisocial/criminal behavior, which in turn may reinforce disrupted physiological activity. For example, males and females who exhibited high rates of proactive aggression (an instrumental, predatory form of aggression elicited to obtain a goal or reward) in early adolescence were found to have poorer skin conductance fear conditioning in late adolescence (Gao, Tuvblad, Schell, Baker, & Raine, 2015; Vitiello & Stoff, 1997).

Theories have been proposed to explain how blunted autonomic functioning could increase antisociality. The fearlessness hypothesis suggests that antisocial individuals, due to their blunted autonomic functioning, are not deterred from criminal behavior because they do not experience appropriate physiological responses to risky or stressful situations nor potential aversive consequences (Portnoy et al., 2014; Raine, 2002). Alternatively, the sensation-seeking hypothesis suggests that blunted psychophysiology is an uncomfortable state of being, and in order to achieve homeostasis, individuals engage in antisocial behavior to raise their arousal levels (Portnoy et al., 2014; Raine, 2002).

Another mechanism that could connect disrupted autonomic functioning to antisocial behavior is the failure to cognitively associate physiology responses with emotional states. Appropriately linking autonomic conditions to emotional states is important in socialization processes such as fear conditioning, which is thought to contribute to the development of a conscience. The somatic marker hypothesis (Bechara & Damasio, 2005) suggests that 'somatic markers' (e.g. sweaty palms) may reflect emotional states (e.g. anxiety) that can inform decision-making processes. Impairments in autonomic functioning could lead to risky or inappropriate behavior if individuals are unable to experience or label somatic changes and connect them to relevant emotional experiences. Indeed, psychopathic individuals exhibit somatic aphasia (i.e. the inaccurate identification and recognition of one's bodily state; Gao, Raine, & Schug, 2012). Moreover, blunted autonomic functioning impairs emotional intelligence, subsequently increasing psychopathic traits (Ling, Raine, Gao, & Schug, 2018a). Impaired autonomic functioning and reduced emotional intelligence may impede the treatment of psychopathy (Polaschek & Skeem, 2018) and disrupt development of moral emotions such as shame, guilt, and empathy (Eisenberg, 2000). Such moral dysfunction, a strong characteristic of psychopaths, may contribute to their disproportionate impact on the criminal justice system (Kiehl & Hoffman, 2011).

While there is evidence that antisocial/criminal individuals typically exhibit abnormal psychophysiological functioning, it is important to acknowledge that there are different antisocial/criminal subtypes, and they may not share the same deficits. Whereas individuals who are high on proactive aggression may be more likely to exhibit blunted autonomic functioning, individuals who are high on reactive aggression (an affective form of aggression that is elicited as a response to perceived provocation) may be more likely to exhibit hyperactive autonomic functioning (Hubbard, McAuliffe, Morrow, & Romano, 2010; Vitiello & Stoff, 1997). This may have implications for different types of offenders, with elevated autonomic functioning presenting in reactively aggressive individuals who engage in impulsive crimes and blunted autonomic functioning presenting in proactively aggressive offenders engaging in more premediated crimes. Similarly, psychopaths

who are 'unsuccessful' (i.e. convicted criminal psychopaths) exhibit reduced heart rate during stress while those who are 'successful' (i.e. non-convicted criminal psychopaths) exhibit autonomic functioning similar to non-psychopathic controls (Ishikawa, Raine, Lencz, Bihrle, & LaCasse, 2001). Despite differences among subgroups, dysfunctional autonomic functioning generally remains a reasonably well-replicated and robust correlate of antisocial and criminal behavior.

Brain

There has been increasing interest in the role of the brain in antisocial/criminal behavior. In general, research suggests that antisocial/criminal individuals tend to exhibit reduced brain volumes as well as impaired functioning and connectivity in key areas related to executive functions (Alvarez & Emory, 2006; Meijers, Harte, Meynen, & Cuijpers, 2017; Morgan & Lilienfeld, 2000), emotion regulation (Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Eisenberg, 2000), decision-making (Coutlee & Huettel, 2012; Yechiam et al., 2008), and morality (Raine & Yang, 2006) while also exhibiting increased volumes and functional abnormalities in reward regions of the brain (Glenn & Yang, 2012; Korponay et al., 2017). These prefrontal and subcortical regions that have been implicated in antisocial/criminal behavior are the selective focus of this review.

Prefrontal cortex

Conventional criminal behavior has typically been associated with prefrontal cortex (PFC) structural aberrations and functional impairments (Brower & Price, 2001; Yang & Raine, 2009). The PFC is considered the seat of higher-level cognitive processes such as decision-making, attention, emotion regulation, impulse control, and moral reasoning (Sapolsky, 2004). In healthy adults, larger prefrontal structures have been associated with better executive functioning (Yuan & Raz, 2014). However, structural deficits and functional impairments of the PFC have been observed in antisocial and criminal individuals, suggesting that PFC aberrations may underlie some of the observed behaviors.

While many studies on brain differences related to criminal behavior have consisted of correlational analyses, lesion studies have provided some insight into causal neural mechanisms of antisocial/criminal behavior. The most well-known example of the effects of prefrontal lobe lesions is the case of Phineas Gage, who was reported to have a dramatic personality change after an iron rod was shot through his skull and damaged his left and right prefrontal cortices (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994; Harlow, 1848, 1868). Empirical studies suggest that prefrontal lesions acquired earlier in life disrupt moral and social development (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Taber-Thomas et al., 2014). A study of 17 patients who developed criminal behavior following a brain lesion documented that while these lesions were in different locations, they were all connected functionally to regions activated by moral decision-making (Darby, Horn, Cushman, & Fox, 2018), suggesting that disruption of a neuromoral network is associated with criminality. Nevertheless, while lesion studies have implicated specific brain regions in various psychological processes such as moral development, generalizability is limited because of the heterogeneity of lesion characteristics, as well as subjects' characteristics that may moderate the behavioral effects of the lesion.

In recent years, non-invasive neural interventions such as transcranial magnetic stimulation and transcranial electric stimulation have been used to manipulate activity within the brain to provide more direct causal evidence of the functions of specific brain regions with regard to behavior. These techniques involve subthreshold modulation of neuronal resting membrane potential (Nitsche & Paulus, 2000; Woods et al., 2016). Using transcranial electric stimulation, upregulation of the PFC has been found to decrease criminal intentions and increase perceptions of moral wrongfulness of aggressive acts (Choy, Raine, & Hamilton, 2018), providing support for the causal influence of the PFC on criminal behavior.

Importantly, there is evidence of heterogeneity within criminal subgroups. Successful psychopaths and white-collar offenders do not seem to display these prefrontal deficits (Raine et al., 2012; Yang et al., 2005). While unsuccessful psychopaths exhibit reduced PFC gray matter volume compared to successful psychopaths and non-offender controls, there are no prefrontal gray matter volume differences between successful psychopaths and non-offender controls (Yang et al., 2005). Similarly, while prefrontal volume deficits have been found in conventional criminals (i.e. blue-collar offenders), white-collar offenders do not exhibit frontal lobe reductions (Brower & Price, 2001; Ling et al., 2018b; Raine et al., 2012) and in fact may exhibit increased executive functioning compared to blue-collar controls (Raine et al., 2012). Lastly, antisocial offenders with psychopathy exhibited reduced gray matter volumes in the prefrontal and temporal poles compared to antisocial offenders without psychopathy and non-offenders (Gregory et al., 2012). It is therefore important to acknowledge that there are various types of antisocial and criminal behavior that may have different neurobiological etiologies.

Amygdala

The amygdala is an important brain region that has been implicated in emotional processes such as recognition of facial and auditory expressions of emotion, especially for negative emotions such as fear (Fine & Blair, 2000; Murphy, Nimmo-Smith, & Lawrence, 2003; Sergerie, Chochol, & Armony, 2008). Normative amygdala functioning has been thought to be key in the development of fear conditioning (Knight, Smith, Cheng, Stein, & Helmstetter, 2004; LaBar, Gatenby, Gore, LeDoux, & Phelps, 1998; Maren, 2001), and appropriate integration of the amygdala and PFC has been argued to underlie the development of morality (Blair, 2007). The amygdala is thought to be involved in stimulus-reinforcement learning that associates actions that harm others with the aversive reinforcement of the victims' distress and in recognizing threat cues that typically deter individuals from risky behavior. However, amygdala maldevelopment can lead to a diminished ability to recognize distress or threat cues; disrupting the stimulus-reinforcement learning that discourages antisocial/criminal behavior (Blair, 2007; Sterzer, 2010). Indeed, while reduced amygdala volume in adulthood has been associated with increased aggressive and psychopathic characteristics from childhood to early adulthood, it is also associated with increased risk for future antisocial and psychopathic behavior (Pardini, Raine, Erickson, & Loeber, 2014).

Although the amygdala has been implicated in criminal behavior, there may be important differences between subtypes of offenders. Whereas psychopathic antisocial individuals may be more likely to exhibit cold, calculating forms of aggression, non-psychopathic antisocial individuals may be more likely to engage in impulsive, emotionally-reactive aggression (Glenn & Raine, 2014). Research suggests the former may exhibit amygdala

hypoactivity and the latter, amygdala hyperactivity (Raine, 2018a). Indeed, violent offenders have been found to exhibit increased amygdala reactivity in response to provocations (da Cunha-Bang et al., 2017). Spousal abusers have also been found to exhibit increased amygdala activation when responding to aggressive words compared to non-abusers (Lee, Chan, & Raine, 2008). In a community sample of healthy adults, psychopathy scores were negatively related to amygdala reactivity while antisocial personality disorder scores were positively associated with amygdala reactivity after adjusting for overlapping variance between psychopathy and antisocial personality disorder (Hyde, Byrd, Votruba-Brzal, Hariri, & Manuck, 2014). Nevertheless, more research is needed to determine whether the presence of callous–unemotional traits (e.g. lack of guilt; Lozier, Cardinale, VanMeter, & Marsh, 2014; Viding et al., 2012) or severity of antisocial behavioral traits (Dotterer, Hyde, Swartz, Hariri, & Williamson, 2017; Hyde et al., 2016) are most relevant to the observed amygdala hypo-reactivity.

Striatum

The striatum has recently garnered more attention as a region that could be implicated in the etiology of criminal behavior because of its involvement in reward and emotional processing (Davidson & Irwin, 1999; Glenn & Yang, 2012). Dysfunction in the striatum has been hypothesized to be a neural mechanism that underlies the impulsive/antisocial behavior of criminals. Indeed, individuals with higher impulsive/antisocial personality traits have been found to exhibit increased activity in the striatum (Bjork, Chen, & Hommer, 2012; Buckholtz et al., 2010; Geurts et al., 2016). Psychopathic individuals, compared to non-psychopathic individuals, demonstrate a 9.6% increase in striatal volumes (Glenn, Raine, Yaralian, & Yang, 2010). Moreover, striatal enlargement and abnormal functional connectivity of the striatum has specifically been associated with the impulsive/antisocial dimension of psychopathy (Korponay et al., 2017), suggesting this dimension of psychopathy is related to reward processes (Hare, 2017).

While much of the literature on striatal abnormalities in antisocial individuals has focused on psychopathic individuals, there is some evidence that offenders in general exhibit striatal abnormalities. Increased volume (Schiffer et al., 2011) and increased reactivity to provocations (da Cunha-Bang et al., 2017) have both been found in violent offenders as compared to non-offendersMoreover, weak cortico-striatal connectivity has been associated with increased frequency of criminal convictions (Hosking et al., 2017). In contrast, one study found reduced striatal activity to be associated with antisocial behavior (Murray, Shaw, Forbes, & Hyde, 2017). While more research is needed, current literature suggests that striatal deviations are linked to criminal behavior. One important consideration for future studies is to determine a consistent operationalization for the striatum, as some studies examine the *dorsal striatum* (i.e. putamen and caudate; Yang et al., 2015), others assess the *corpus striatum* (i.e. putamen, caudate, and globus pallidus; Glenn et al., 2010), and still others analyze the role of the *ventral striatum* (i.e. nucleus accumbens and olfactory tubercle; Glenn & Yang, 2012) in relation to antisocial/criminal behavior.

The neuromoral theory of antisocial behavior

Abnormalities in brain regions other than the PFC, amygdala, and striatum are also associated with antisocial behavior. The neuromoral theory of antisocial behavior, first proposed

by Raine and Yang (2006), argued that the diverse brain regions impaired in offenders overlap significantly with brain regions involved in moral decision-making. A recent update of this theory (Raine, 2018b) argues that key areas implicated in both moral decision-making and the spectrum of antisocial behaviors include frontopolar, medial, and ventral PFC regions, and the anterior cingulate, amygdala, insula, superior temporal gyrus, and angular gyrus/temporoparietal junction. It was further hypothesized that different manifestations of antisocial behavior exist on a spectrum of neuromoral dysfunction, with primary psychopathy, proactive aggression, and life-course persistent offending being more affected, and secondary psychopathy, reactive aggression, and crimes involving drugs relatively less affected. Whether the striatum is part of the neural circuit involved in moral decision-making is currently unclear, making its inclusion in the neuromoral model debatable. Despite limitations, the neuromoral model provides a way of understanding how impairments to different brain regions can converge on one concept – impaired morality – that is a common core to many different forms of antisocial behaviors.

One implication of the model is that significant impairment to the neuromoral circuit could constitute diminished criminal responsibility. Given the importance of a fully developed emotional moral capacity for lawful behavior, moral responsibility would appear to require intactness of neuromoral circuity. To argue that the brain basis to moral thinking and feeling are compromised in an offender comes dangerously close to challenging moral responsibility, a concept which in itself may be just a short step removed from criminal responsibility.

Genetics

There is increasing evidence for a genetic basis of antisocial/criminal behavior. Behavioral genetic studies of twins and adoptees have been advantageous because such designs can differentiate the effects of genetics and environment within the context of explaining variance within a population (Glenn & Raine, 2014). Additionally, a variety of psychological and psychiatric constructs associated with antisociality/criminality, such as intelligence, personality, and mental health disorders, have been found to be heritable (Baker, Bezdjian, & Raine, 2006). While individual study estimates vary, meta-analyses have suggested the level of heritability of antisocial behavior is approximately 40–60% (Raine, 2013). Shared environmental factors have been estimated to explain approximately 11–14% of the variance in antisocial/criminal behavior and non-shared environmental influences approximately 31–37% (Ferguson, 2010; Gard, Dotterer, & Hyde, 2019). However, the heritability of antisocial/criminal behaviors vary in part based upon the specific behaviors examined (Burt, 2009; Gard et al., 2019).

Inspired by prominent theories of the neurobiology of aggression, there have been several candidate genes implicated in the serotonergic and catecholaminergic neurobiological systems that have been examined in relation to antisocial/criminal behavior (Tiihonen et al., 2015). However, a meta-analysis of genetic variants related to antisocial/criminal behavior yielded null results at the 5% significance level (Vassos, Collier, & Fazel, 2014). Nevertheless, genes do not operate in isolation, thus it is important to consider the context in which genes are activated.

Gene-environment (G x E) interactions have garnered increasing attention over the years, as these can increase risk for antisocial behavior and/or produce epigenetic

changes within individuals. Longitudinal studies and meta-analyses have documented the moderating effect of the monoamine oxidase A (MAOA) gene on the relationship between maltreatment and antisocial behaviors, with the maltreatment-antisocial behavior relationship being stronger for individuals with low MAOA than high MAOA (Byrd & Manuck, 2014; Caspi et al., 2002; Fergusson, Boden, & Horwood, 2011; Kim-Cohen et al., 2006). Similarly, in a large study of African-American females, having the A1 allele of the DRD2 gene or a criminal father did not individually predict antisocial outcomes, but having both factors increased risk for serious delinquency, violent delinquency, and police contacts (Delisi, Beaver, Vaughn, & Wright, 2009). This type of G x E interaction reflects how genotypes can influence individuals' sensitivity to environmental stressors. However, there may be important subgroup differences to consider when examining genetic risk for criminal behavior. For example, low-MAOA has been associated with higher risk for violent crime in incarcerated Caucasian offenders but not incarcerated non-Caucasian offenders (Stetler et al., 2014). Additionally, high-MAOA may protect abused and neglected Caucasians from increased risk of becoming violent or antisocial, but this buffering effect was not found for abused and neglected non-Caucasians (Widom & Brzustowicz, 2006). Thus, while the MAOA gene has been associated with antisocial/criminal behavior, there are still nuances of this relationship that should be considered (Goldman & Rosser, 2014).

Another way in which G x E interactions manifest themselves is when environmental stressors result in epigenetic changes, thus becoming embedded in biology that result in long-term symptomatic consequences. For example, females exposed to childhood sex abuse have exhibited alterations in the methylation of the *5HTT* promoter region, which in turn has been linked to subsequent antisocial personality disorder symptoms (Beach, Brody, Todorov, Gunter, & Philibert, 2011). There has been a growing body of work on such epigenetic mechanisms involved in the biological embedding of early life stressors and transgenerational trauma (Kellermann, 2013; Provencal & Binder, 2015). Thus, just as biological mechanisms can influence environmental responses, environmental stressors can affect biological expressions.

While genes may interact with the environment to produce antisocial/criminal outcomes, they can also interact with other genes. There is evidence that dopamine genes DRD2 and DRD4 may interact to increase criminogenic risk (Beaver et al., 2007; Boutwell et al., 2014). The effect of the 7-repeat allele DRD4 is strengthened in the presence of the A1 allele of DRD2, and has been associated with increased odds of committing major theft, burglary, gang fighting, and conduct disorder (Beaver et al., 2007; Boutwell et al., 2014). However, there is some evidence that DRD2 and DRD4 do not significantly affect delinquency abstention for females (Boutwell & Beaver, 2008). Thus there may be demographic differences that moderate the effect of genetic interactions on various antisocial outcomes (Dick, Adkins, & Kuo, 2016; Ficks & Waldman, 2014; Rhee & Waldman, 2002; Salvatore & Dick, 2018), and such differences warrant further research.

Interactions between biological factors

Importantly, biological correlates of antisocial and criminal behavior are inextricably linked in dynamical systems, in which certain processes influence others through feedback loops. While a detailed summary is beyond the scope of this review, some interactions between

biological mechanisms are briefly illustrated here. Within the brain, the PFC and amygdala have reciprocal connections, with the PFC often conceptualized as monitoring and regulating amygdala activity (Gillespie, Brzozowski, & Mitchell, 2018). Disruption of PFC-amygdala connectivity has been linked to increased antisocial/criminal behavior, typically thought to be due to the impaired top-down regulation of amygdala functioning by the PFC. Similarly, the brain and autonomic functioning are linked (Critchley, 2005; Wager et al., 2009); output from the brain can generate changes in autonomic functioning by affecting the hypothalamic–pituitary–adrenal axis, but autonomic functions also provide input to the brain that is essential for influencing behavioral judgments and maintaining coordinated regulation of bodily functions (Critchley, 2005). While not comprehensive, these examples illustrate that biological systems work together to produce behavior.

Implications

While biological processes can contribute to antisocial/criminal behavior, these do not guarantee negative outcomes. Considering that many of the aforementioned biological risk factors are significantly influenced by social environment, interventions in multiple spheres may help mitigate biological risks for antisocial behavior.

With regard to psychophysiological correlates of antisocial behavior, research suggests differential profiles of arousal impairment depending on the type of antisocial behavior (Hubbard et al., 2010; Vitiello & Stoff, 1997). Treatments designed to address the issues associated with psychophysiological differences are typically behavioral in nature, targeted at associated symptoms. Studies of mindfulness have suggested its utility in improving autonomic functioning (Delgado-Pastor, Perakakis, Subramanya, Telles, & Vila, 2013) and emotion regulation (Umbach, Raine, & Leonard, 2018), which may better help individuals with reactive aggression and hyperarousal. Hypo-arousal has been associated with impaired emotional intelligence (Ling et al., 2018a), but emotional intelligence training programs have shown some promise in reducing aggression and increasing empathy among adolescents and increasing emotional intelligence among adults (Castillo, Salguero, Fernandez-Berrocal, & Balluerka, 2013; Hodzic, Scharfen, Ropoll, Holling, & Zenasni, 2018), and in reducing recidivism (Megreya, 2015; Sharma, Prakash, Sengar, Chaudhury, & Singh, 2015).

Regarding healthy neurodevelopment, research has supported a number of areas to target. Poor nutrition, both in utero and in early childhood, have been associated with negative and criminal outcomes (Neugebauer, Hoek, & Susser, 1999). Deficits of omega-3 fatty acids have been linked with impaired neurocognition and externalizing behavior (Liu & Raine, 2006; McNamara & Carlson, 2006). The opposite relationship is also supported; increased intake of omega-3 fatty acids has been associated with a variety of positive physical and mental health outcomes (Ruxton, Reed, Simpson, & Millington, 2004), increased brain volume in regions related to memory and emotion regulation (Conklin et al., 2007), and reduction in behavioral problems in children (Raine, Portnoy, Liu, Mahoomed, & Hibbeln, 2015). Studies examining the effect of nutritional supplements have suggested that reducing the amount of sugar consumed by offenders can significantly reduce offending during incarceration (Gesch, Hammond, Hampson, Eves, & Crowder, 2002; Schoenthaler, 1983). Thus, nutritional programs show some promise in reducing antisocial and criminal behavior.

A healthy social environment is also crucial for normative brain development and function. Early adversity and childhood maltreatment have been identified as significant risk factors for both neurobiological and behavioral problems (Mehta et al., 2009; Teicher et al., 2003; Tottenham et al., 2011). A review of maltreatment prevention programs supports the efficacy of nurse-family partnerships and programs that integrate early preschool with parent resources in reducing childhood maltreatment (Reynolds, Mathieson, & Topitzes, 2009). Promoting healthy brain development in utero and in crucial neurodevelopmental periods is likely to reduce externalizing behaviors, as well as other psychopathology.

Knowing that the social context could help to buffer biological risks is promising because it suggests that changing an individual's environment could mitigate biological criminogenic risk. Rather than providing a reductionist and deterministic perspective of the etiology of criminal behavior, incorporating biological factors in explanations of antisocial/criminal behaviors can highlight the plasticity of the human genome (Walsh & Yun, 2014). They can also provide a more holistic understanding of the etiologies of such behavior. For example, sex differences in heart rate have been found to partially explain the gender gap in crime (Choy, Raine, Venables, & Farrington, 2017). Social interventions that aim to provide an enriched environment can be beneficial for all, but may be particularly important for individuals at higher biological risk for antisocial behavior. While biological explanations of antisocial and criminal behavior are growing, they are best thought of as complementary to current research and theories, and a potential new avenue to target with treatment options.

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