

Antisocial Personality Disorders

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Abstract

Neuroscience research is beginning to uncover significant neurobiological impairments in antisocial, violent, and aggressive groups. The neurophysiologic basis of antisocial behavior is complex – many structures have been implicated, each of which may be related to antisocial behavior in different ways. Research in social neuroscience is helping us to better understand the role of many of these regions in normal social behavior, and thus why abnormality would result in a disruption of appropriate social behavior. This chapter highlights neuroscience data on antisocial individuals and provides interpretation based on the knowledge that has been gained in recent years in the field of social neuroscience.

Keywords: antisocial personality disorder, conduct disorder, empathy, moral decision-making, prefrontal cortex, amygdala, violence, aggression,

Antisocial Personality

Antisocial personality disorder (APD), as outlined in the DSM-IV, is a categorization of individuals who consistently fail to conform to social norms and display criminal or antisocial behavior. Studies have estimated that approximately 75% of the prison population has APD (Hare, 1991). The construct of APD is very heterogeneous; since antisocial behavior is broadly defined, individuals may vary greatly on the type and severity of antisocial behavior they exhibit. For example, a common distinction in the type of antisocial behavior is between “reactive” and “proactive” forms of aggression. Reactive or “hot” aggression is aggression in response to a perceived threat or frustration. Proactive or “cold” aggression is planned, purposeful aggression used to achieve a goal (e.g. to obtain possessions). Psychopathic individuals fall into the category of individuals with APD who engage in high rates of proactive aggression, though most engage in reactive aggression as well. Psychopathy is a more specific type of antisocial personality and is discussed in chapter 59 and therefore will not be included in the present review. Although some studies have focused on more specific types of antisocial behavior (e.g., Raine et al., 1998), studies commonly define antisocial or violent groups in a broad sense. As such, neuroscience findings appear to implicate a wide range of brain regions and mechanisms. However, it is likely that there are multiple biological pathways that lead to antisocial behavior and that not all antisocial individuals demonstrate the same brain abnormalities.

The major childhood precursor to antisocial personality disorder is conduct disorder (CD) (Lahey et al., 2005). Conduct disorder is described as a longstanding pattern of violations of rules and laws, including aggressive behavior, manipulateness, deceitfulness, theft, forced sex, bullying, running away from home, and property destruction. Conduct disorder is typically diagnosed in older children and adolescents. Antisocial behavior in younger children may be diagnosed as oppositional defiant disorder (ODD), which involves persistently hostile, defiant, and disruptive behavior, as well as low frustration tolerance, occurring outside of the normal range of behavior. In the present chapter, we will focus on studies that implement these clinical definitions of antisocial personality in adults and youth, as well as studies of criminal, delinquent, or violent groups.

Social Neuroscience and Antisocial Personalities

There is strong evidence suggesting that brain abnormalities, whether developmental or as a result of injury, may serve as precursors to antisocial behavior. Paralleling the increasing use of brain imaging techniques to examine brain functioning during normal social processing, many studies have begun using brain imaging to identify the brain regions that are disrupted in antisocial individuals. The knowledge gained by social neuroscience studies has been crucially important in our ability to interpret how deficits in specific brain regions may lead to antisocial behavior. Research on constructs such as empathy, emotion regulation, response inhibition, and moral decision-making has begun to illuminate the neurobiological underpinnings of these processes, thus helping to explain why disruptions to regions involved in these processes could potentially result in disrupted social behavior. However, these processes are also extremely complex and interrelated, and there are likely a large number of potential sources of disruption that could lead to antisocial behavior.

Perhaps the most complex and highest-level construct that is directly relevant to antisocial behavior is moral decision-making. Several studies in social neuroscience have begun to examine the brain regions that are involved when people contemplate moral dilemmas (e.g. Greene et al., 2001) or make moral judgments (e.g. Moll, Oliveira-Sousa et al., 2002; Oliveira-Sousa & Moll, 2000). It has been argued that regions important in moral decision-making and the processes that underlie it, may play an especially central role in the development of antisocial personality (Raine & Yang, 2006). Moral decision-making relies on several underlying processes, including the experience of empathy and guilt, theory-of-mind, and decision-making capabilities. Each of these processes in turn require further capabilities; for example, empathy itself is a complex construct that requires affective sharing between the self and other, self awareness, mental flexibility, and emotion regulation (Decety & Moriguchi, 2007), all of which are still quite complex and have been shown to recruit multiple brain regions. Thus, it is possible that the impairments in higher-order construct of moral decision-making observed in antisocial individuals may result from disruption in any number of different underlying processes. Furthermore, disruptions of different processes may lead to different forms of antisocial behavior.

As an example, reduced empathy is a common feature of antisocial individuals. This reduction in empathy may result from disruption to one or more of the relatively lower-level processes that underlie it. In some individuals, the deficit may come from a reduced ability to share other people's emotional state, primarily in the domains of sadness and fear, which has been found to be associated with activity in the amygdala (Blair, 2007; Blair et al., 2001). Such deficits may result in increased proactive, unprovoked aggression. In contrast, other individuals may exhibit deficits in the ability to self-regulate emotions, as many antisocial individuals perform poorly on tests of executive functioning (Morgan & Lilienfeld, 2000) and inhibitory control (Vollm et al., 2004). This may be due to impairments in orbitofrontal or dorsolateral prefrontal regions. As a result, individuals may be prone to engage in more reactive aggression.

Despite the enormous complexity of studying the neurobiology of antisocial behavior, research has identified several regions which appear to be consistently implicated. These regions are primarily in the prefrontal and temporal cortices. The following will provide a review of several of the regions implicated in antisocial behavior along with the insights gained from social neuroscience about why these regions are likely involved.

The Neural Basis of Antisocial Personality

The best-replicated abnormality across a wide range of antisocial groups and across different imaging methodologies is in the prefrontal cortex. Within the frontal lobe, both structural and functional abnormalities have been observed. Using PET, reduced frontal functioning has been observed in impulsive aggressive individuals (New et al., 2002), murderers (Raine et al., 1997), and violent psychiatric patients (Volkow et al., 1995). Using SPECT, reduced blood flow in the prefrontal cortex has been observed in violent offenders (Soderstrom et al., 2000) and alcoholics with APD (Kuruoglu et al., 1996). Structurally, Raine et al. (2000) found individuals with APD show an 11% reduction in gray matter volume in the prefrontal cortex compared with controls. Additional studies have further localized these abnormalities to specific areas of the prefrontal cortex. Laakso et al. (2002) reported reduced gray matter volume in dorsolateral, orbitofrontal, and medial prefrontal cortex in alcoholics with APD compared to controls. In a recent study using voxel-based morphometry, Tiihonen et al. (2008) found reduced gray matter in the frontopolar and orbitofrontal cortex bilaterally in persistently violent offenders. Regional cortical thinning has been observed in the ventromedial prefrontal cortex in violent individuals with APD (Narayan et al., 2007). A functional imaging study found reduced activity in the orbitofrontal cortex of antisocial individuals during inhibitory control (Vollm et al., 2004). Within the prefrontal cortex, the medial and orbitofrontal/ventromedial regions appear to be the most commonly implicated.

Studies from social neuroscience have provided insight into the functions of regions within the prefrontal cortex and thus why they might be involved in antisocial behavior. The orbitofrontal/ventromedial region is thought to play a role in affective theory of mind (Shamay-Tsoory et al., 2005), processing reward and punishment information (Rolls, 2000), inhibiting responses (Aron et al., 2004; Vollm et al., 2006), and regulating emotions (Ochsner et al., 2005). Unsurprisingly, the orbitofrontal cortex is also important in moral decision-making (Borg et al., 2006; Moll, Oliveira-Souza et al., 2002; Oliveira-Souza & Moll, 2000). It has been suggested that dysfunction in the orbitofrontal region results in poor response inhibition (Aron et al., 2004) and poor decision-making (Bechara, 2004). This also applies to the moral domain, as recent studies of patients with damage to the orbitofrontal / ventromedial prefrontal cortex have demonstrated impairments in moral decision-making (Ciaramelli et al., 2007; Koenigs et al., 2007). Additional studies of patients with brain injury are discussed in Box 58.1.

Box 58.1. Lesion Studies

Studies of patients with damage to particular brain regions have helped to further confirm the importance of some of the brain regions implicated in antisocial behavior. Lesion studies have shown that damage to the orbitofrontal/ventromedial prefrontal cortex produces personality traits and behaviors strikingly similar to those observed in APD (Damasio, 1994). Individuals with lesions in this region display significant rule breaking, lying, impulsivity, failure to hold jobs, failure to plan for the future or form goals, and financial irresponsibility. They are described as lacking empathy, guilt, remorse, and fear, and are unconcerned with their behavioral transgressions. They also show disturbances in moral behavior and decision-making. In moral decisions involving highly conflicting considerations of aggregate welfare versus emotionally aversive behaviors (i.e. smothering one's baby to save a group of people), patients with ventromedial prefrontal cortex damage demonstrate an abnormally utilitarian pattern of judgments compared to controls (Koenigs et al., 2007). It is suggested that the ventromedial prefrontal cortex is crucial for the generation of emotional responsiveness to the aversive acts. While the patients show intact explicit knowledge of social and moral norms, their moral decisions are not guided by emotion to the same degree as normal controls.

Lesion studies have found that individuals who incur brain damage to the ventromedial prefrontal cortex very early in life have even more pronounced antisocial traits. Anderson et al. (1999) found that patients who incurred damage before the age of 16 months developed irresponsible and criminal behavior, abusive behavior towards others, and a lack of empathy or remorse. These antisocial characteristics and behaviors were more severe than those observed in patients who suffered damage in adulthood. It has been suggested that intact functioning of the ventromedial prefrontal cortex early in life is important for moral development (Anderson, 1999). When this region is damaged early on, the process of moral socialization may be disrupted. Indeed, the study found that these individuals demonstrated an immature stage of moral reasoning.

Individuals with lesions to the amygdala have also exhibited some of the same impairments that are observed in antisocial individuals, including impairments in aversive conditioning (Bechara et al., 1999), augmentation of the startle reflex to visual threat primes (Angrilli et al., 1996), and recognizing fearful facial expressions (Adolphs, 2002). However, patients with lesions to the amygdala do not closely resemble individuals with antisocial personality. This may be because amygdala abnormalities in antisocial individuals are not as widespread or severe as those of lesion patients.

Antisocial behavior has also been observed in individuals with frontotemporal dementia (FTD) (Mendez, 2006), a neurodegenerative disorder that affects the frontal lobes, temporal lobes, or both. The transgression of social norms is a core feature of FTD; patients engage in behaviors such as stealing, shoplifting, inappropriate sexual behavior, physical violence, and poor financial decision-making (Mendez et al., 2005). Such a demonstration of pervasive disregard for social and moral standards closely resembles that of individuals with APD, and suggests that impairment in the prefrontal and temporal cortices likely play a significant role in antisocial behavior.

The medial prefrontal cortex has been implicated in the prosocial emotions of guilt, embarrassment, and compassion (Moll et al., 2007; Takahashi et al., 2004), the cognitive appraisal of emotion (Ochsner et al., 2002), and in self reflection (Gusnard et al., 2001). This region has also been implicated in moral judgment (Greene et al., 2001) as well as in the regulation of moral emotions (Harenski & Hamann, 2006). Impaired functioning in the medial region of the prefrontal cortex may lead to disruptions in one or more of these processes that are important in appropriate social behavior and moral judgment.

Finally, within the prefrontal cortex, several studies have found the dorsolateral region to be impaired. In contrast to the orbitofrontal and medial prefrontal regions, which play a large role in emotion and moral decision-making, the dorsolateral prefrontal cortex is likely associated with antisocial behavior because of its role in executive functions. The dorsolateral prefrontal cortex is involved in processes such as planning and organization (Smith & Jonides, 1999), attentional set shifting and cognitive flexibility (Dias et al., 1996), cognitive reappraisal of emotional experience (Ochsner et al., 2002), and response perseveration (Lombardi et al., 1999). Thus, dysfunction in the dorsolateral prefrontal cortex may impair planning and other executive functions (Smith & Jonides, 1999) that may predispose to outcomes such as occupational failure and hence low income, repetition of maladaptive antisocial responses, or a failure to consider alternative strategies to resolve conflict. In antisocial groups, reduced gray matter volume of the dorsolateral prefrontal cortex has been observed in alcoholics with antisocial personalities (Laakso et al., 2002), and reduced bloodflow has been found in aggressive patients (Hirono et al., 2000). Abnormal dorsolateral prefrontal cortex functioning has been observed in two fMRI studies of individuals with APD (Schneider et al., 2000; Vollm et al., 2004).

In addition to the prefrontal cortex, there is substantial evidence for structural and functional impairments in the amygdala in antisocial groups. Reduced volume of the amygdala has been reported in violent offenders (Tiihonen et al., 2000). Functional asymmetries of the amygdala have been observed in murderers (Raine et al., 1997), showing reduced left and increased right amygdala activity. However, two studies have reported *increased* amygdala activation in antisocial individuals while viewing negative visual content (Muller et al., 2003) and during aversive conditioning (Schneider et al., 2000). Deficits have also been observed in the adjacent temporal cortex in antisocial individuals. Volume reductions in the temporal lobe have been observed in patients with APD (Barkataki et al., 2006) and impulsive-aggressive personality-disordered patients (Dolan et al., 2002). Reduced metabolism in the temporal cortex has been observed in violent patients (Seidenwurm et al., 1997; Volkow et al., 1995) and reduced blood flow has been observed in aggressive patients (Hirono et al., 2000) and violent offenders (Soderstrom et al., 2000). Functional impairments in the temporal lobe have been shown in aggressive patients (Amen et al., 1996; Volkow & Tancredi, 1987) and in violent offenders (Raine et al., 2001).

The disruption of amygdala functioning may interfere with processes that have been found to be important to normal socialization and social behavior. Amygdala dysfunction impairs classical conditioning (LeDoux, 2000) which is hypothesized to form the basis of conscience and the anticipatory fear that normally deters individuals from committing antisocial acts (Blair, 2004). More specifically, the amygdala is necessary for the formation of stimulus-reinforcement associations, which are necessary for an individual to learn to associate their harmful actions with the pain and distress of others, thus facilitating empathy for victims and discouraging antisocial behavior (Blair, 2006). It is also involved in the production of emotional states (Phillips et al., 2003), and enhancing attention to emotional stimuli, such as facial expressions of emotion (Adolphs et al., 1999). Finally, the amygdala has been identified as a region important in moral judgment (Greene et al., 2004), the experience of moral emotions (Moll, Oliveira-Souza et al., 2002), and has also been found to respond during one's own moral violations (Berthoz et al., 2006).

There are two additional regions that are commonly implicated in moral decision-making that have also been associated with antisocial behavior – the angular gyrus (posterior superior temporal gyrus) and the posterior cingulate. Deficits in the angular gyrus have been observed in murderers (Raine et al., 1997) and in impulsive, violent criminals (Soderstrom et al., 2000). Reduced volume of the posterior cingulate has been observed in persistently violent offenders (Tiihonen et al., 2008). The angular gyrus is implicated in the experience of guilt and embarrassment (Takahashi et al., 2004) which are secondary emotions motivating rule-breaking individuals to desist from future antisocial behaviors. It has also been found to be involved in reasoning about social contracts (Fiddick et al., 2005). The posterior cingulate is involved in self-referencing (Ochsner et al., 2005) and reflecting on one's duties and obligations (Johnson et al., 2006). Both the posterior cingulate and angular gyrus are involved in aspects of social cognition that are important to moral decision-making, and have been found to be active in studies of moral judgment (Greene et al., 2004; Greene et al., 2001).

There are additional areas that have been implicated in antisocial behavior that have not commonly been associated with moral judgment. For example, the functional integrity of the hippocampus has been found to be abnormal in murderers (Raine et al., 1997), and in violent offenders (Soderstrom et al., 2000). It has been suggested that abnormalities

in this region may reflect disrupted neurodevelopmental processes (Raine et al., 2004). The hippocampus is also important in the retrieval of emotional memories and is involved in contextual fear conditioning (Fanselow, 2000; LeDoux, 1998). Thus, hippocampal impairments may disrupt learning in the social context rendering antisocial individuals insensitive to environmental cues of future punishment.

In sum, numerous brain regions involved in social processes have been implicated in antisocial behavior. Given the heterogeneity of the categorization of antisocial individuals, it is likely that different brain impairments underlie different forms of antisocial personality. However, a core feature of antisocial personality disorders is immoral behavior. This may result from a disruption to one or more brain regions underlying moral decision-making, including the medial prefrontal and orbitofrontal cortices, amygdala, angular gyrus, and posterior cingulate. As highlighted in a recent review (Raine & Yang, 2006), the regions frequently activated in moral decision-making tasks demonstrate significant overlap with the brain regions structurally and functionally compromised in antisocial populations. Figure 1 depicts the regions commonly implicated in moral decision-making studies, along with those commonly implicated in antisocial populations. By juxtaposing these two sets of empirical data, the commonalities become apparent. This overlap may give rise to the hypothesis that some of the brain impairments observed in antisocial individuals disrupt moral emotions and decision-making, in turn predisposing to antisocial behavior (Raine & Yang, 2006).

Antisocial Personality in Youth

Childhood antisocial behavior is an especially important area of study because it can give insight into the developmental pathways that lead to long-term antisocial behavior. Although brain imaging methods in youth were previously limited due to potential hazards of administering radioactive isotopes or ionizing radiation, the development of MRI techniques have allowed for the extension of brain imaging studies to youth. Studies of youth with conduct disorder have produced results that are largely similar to those in antisocial adults, suggesting that the brain impairments observed in adults likely exist at an early age. However, some inconsistencies in findings do exist.

The first structural MRI study of conduct disordered youth did not produce significant results (Bussing et al., 2002); however, this study was limited by a small sample size of seven individuals. Later, Kruesi et al. (2004) found that youth with conduct disorder and a history of ADHD demonstrated significantly reduced volumes of the temporal lobes; volumes of the prefrontal cortex also tended to be smaller in subjects with CD, but results did not reach statistical significance. In a recent study using voxel-based morphometry, Huebner et al. (2008) found that boys with conduct disorder, most of whom had comorbid ADHD, demonstrated a 6% decrease in overall gray matter. Specific reductions were observed in the orbitofrontal cortex and temporal lobes, including the hippocampus and amygdala. Symptoms of conduct disorder correlated primarily with reduced gray matter in limbic brain regions.

Several functional MRI studies have demonstrated reduced activity in the amygdala of youth with conduct disorder. Sterzer et al. (2005) found reduced activation in the amygdala in aggressive children with conduct disorder while viewing negative emotional pictures. Jones et al. (2009) found that boys with conduct problems and callous-unemotional traits demonstrated reduced activity in the amygdala when viewing fearful faces compared to control participants. Similarly, Marsh et al. (2008) found that children with callous-unemotional traits demonstrate reduced amygdala activity to fearful facial expressions, but not to neutral or angry expressions. Furthermore, these children demonstrated reduced connectivity between the amygdala and ventromedial prefrontal cortex; the severity of symptoms in the callous-unemotional traits groups was found to be negatively correlated with the degree of connectivity between these regions. It is suggested that the connectivity between these regions is important because it allows for input from the amygdala to guide behavioral selection processes in the ventromedial prefrontal cortex.

Additional regions that have demonstrated reduced functioning fMRI studies of youth with conduct disorder include the orbitofrontal cortex, insula, hippocampus, and anterior cingulate during a rewarded continuous performance task (Rubia et al., 2009), and the posterior cingulate and temporal-parietal regions during an inhibition task (Rubia et al., 2008). Reduced activity in the medial and orbitofrontal prefrontal cortex and temporo-parietal junction has been observed in adolescents with conduct disorder when viewing scenes of pain being intentionally inflicted on another individual (Decety et al., 2009). Adolescents with CD also exhibited less amygdala/prefrontal coupling when perceiving others in pain, which may reflect impairment in the ability to regulate emotions.

However, some discrepancies remain. Herpertz et al. (2008) found *increased* left-sided amygdala activity in boys with conduct disorder when viewing negative pictures, and no evidence of reduced functioning in orbitofrontal, anterior

cingulate, or insular cortices. Similarly Decety et al. (2009) found greater activity in the amygdala and temporal pole in adolescents with aggressive conduct disorder compared to healthy adolescents when perceiving other individuals in pain. It is hypothesized that this activation may reflect an aroused state of enjoyment or excitement at viewing others in pain.

For the most part, findings from neuroimaging studies in antisocial youth tend to parallel those of adult antisocial individuals. This suggests that brain abnormalities likely exist early in life and thus affect socialization.

Conclusion and Implications

The findings of brain abnormalities in antisocial youth and adults raises an intriguing forensic question. There is little doubt that most criminal and delinquent individuals *know* the difference between right and wrong – the question is whether they have the *feeling* of what is right and wrong. Moral decision-making is viewed as heavily predicated on affect (Greene & Haidt, 2002; Moll et al., 2005). This “moral feeling”, based on the functioning of the moral neural circuit, is thought to translate the cognitive recognition that an act is immoral into behavioral inhibition; in normal individuals, these emotional experiences inhibit aggressive impulses (Davidson et al., 2000). This system may function less well in antisocial individuals. This issue raises the question, if a criminal offender has disruption to this neural circuitry, are they fully accountable for their immoral behavior? This issue is further discussed in Box 58.2.

Box 58.2. Neuroethical Implications

The use of neuroscience to explore the biological mechanisms that may motivate antisocial behavior in some individuals raises important questions about the implications of this new knowledge for society, the law, and civil liberties. Such research is of prime consideration in the newly developed field of “neuroethics” (see Chapter XX – Martha Farah). One issue concerns the use of neuroscience evidence in the legal system. Interestingly, such evidence has the potential to be used to make two very different cases. The first argument may be that if early biological factors beyond the individual’s control result in brain dysfunction that increases the probability of engaging in antisocial behavior, it seems reasonable to consider such evidence as grounds for mitigation of punishment. If an individual demonstrates brain abnormalities that impair moral decision-making, are they really to blame for their immoral behavior? This argument poses significant threat to the concepts of individual accountability and free will, upon which the legal system is based. An alternative argument may be that if an individual demonstrates biological risk factors for antisocial behavior, it may be grounds for the *exacerbation* of punishment, as it may indicate increased likelihood of reoffending. This argument has the potential to raise significant civil liberties issues by opening the door to the use of prediction tools to contain or punish crime-prone individuals before offenses have occurred. It also carries with it the risk of harm caused by labeling or wrongly identifying individuals as susceptible to antisocial behavior.

Although these are important points of consideration, it should be noted that the ability to quantify neurobiological impairments in the absence of obvious brain injury is currently very limited. The neurobiological studies of antisocial behavior involve examining differences at the group level – the findings represent the average of many individuals and do not suggest that all antisocial individuals exhibit a particular abnormality. This type of information should be made clear by scientists taking part in legal cases in order to ensure that brain imaging evidence is not over-interpreted in these settings. At some point in the future it may be possible to build up normative datasets so that significant differences in brain structure or functioning could be quantifiable, increasing the potential for use in courts. In this case, evidence of neurobiological impairment is probably best treated similarly to evidence from other biological, psychological, or psychosocial sources. It should be taken into consideration as one of many factors that may increase an individual’s risk for criminal behavior, but should not be over-interpreted as representing a causal, one-to-one relationship with behavior.

Despite an exponential increase in brain imaging research on antisocial populations implicating multiple brain systems, neuroscience research on this important social and clinical construct is far from complete. With the continual development of imaging techniques, as well as unique paradigms from social neuroscience, our understanding of the neurobiological bases of antisocial personality will become more sophisticated. The application of neuroscience methods to the study of antisocial personality has the potential to lead to new approaches for treatment by providing an understanding of the mechanisms that underlie the development of antisocial personalities. Given the heterogeneous nature of the disorder, it is likely that different biological risk factors underlie different manifestations of the disorder. Although the neurobiological impairments currently seem widespread, it is likely that different biological risk factors may lead to antisocial behavior in different ways. In the future, it may be possible to develop individualized treatments that target specific neurobiological risk factors.

Advances in this field will also need to take increasing cognizance of the environmental context within which neurobiological predispositions give expression to antisocial behavior. There is initial evidence that environmental factors may moderate brain-violence relationships. Surprisingly, integrative biosocial research in this imaging field is almost non-existent. Another future direction will be the delineation of the specific genes that give rise to the brain impairments found in antisocial groups. For example, a common polymorphism in the MAOA gene has been implicated in antisocial behavior (Caspi et al., 2002), and in males this same polymorphism is associated with an 8% reduction in the volume of the amygdala, anterior cingulate, and orbitofrontal cortex (Meyer-Lindenberg et al., 2006), structures compromised in antisocial individuals. Given the evidence that 90% of the variability in prefrontal gray volume is attributable to genetics (Thompson et al., 2001), a “genes-to-brain-to-antisocial behavior” approach is likely to increasingly provide an important conceptual framework for future empirical research of this important societal problem (Raine, 2008).

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