Neural foundations to moral reasoning and antisocial behavior

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Neural foundations to moral reasoning and antisocial behavior

Abstract
A common feature of the antisocial, rule-breaking behavior that is central to criminal, violent and psychopathic individuals is the failure to follow moral guidelines. This review summarizes key findings from brain imaging research on both antisocial behavior and moral reasoning, and integrates these findings into a neural moral model of antisocial behavior. Key areas found to be functionally or structurally impaired in antisocial populations include dorsal and ventral regions of the prefrontal cortex (PFC), amygdala, hippocampus, angular gyrus, anterior cingulate and temporal cortex. Regions most commonly activated in moral judgment tasks consist of the polar/medial and ventral PFC, amygdala, angular gyrus and posterior cingulate. It is hypothesized that the rule-breaking behavior common to antisocial, violent and psychopathic individuals is in part due to impairments in some of the structures (dorsal and ventral PFC, amygdala and angular gyrus) subserving moral cognition and emotion. Impairments to the emotional component that comprises the feeling of what is moral is viewed as the primary deficit in antisocials, although some disruption to the cognitive and cognitive-emotional components of morality (particularly self-referential thinking and emotion regulation) cannot be ruled out. While this neurobiological predisposition is likely only one of several biosocial processes involved in the etiology of antisocial behavior, it raises significant moral issues for the legal system and neuroethics.

Keywords
antisocial, psychopathy, moral, prefrontal, temporal

Comments

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A common feature of the antisocial, rule-breaking behavior that is central to criminal, violent and psychopathic individuals is the failure to follow moral guidelines. This review summarizes key findings from brain imaging research on both antisocial behavior and moral reasoning, and integrates these findings into a neural moral model of antisocial behavior. Key areas found to be functionally or structurally impaired in antisocial populations include dorsal and ventral regions of the prefrontal cortex (PFC), amygdala, hippocampus, angular gyrus, anterior cingulate and temporal cortex. Regions most commonly activated in moral judgment tasks consist of the polar/medial and ventral PFC, amygdala, angular gyrus and posterior cingulate. It is hypothesized that the rule-breaking behavior common to antisocial, violent and psychopathic individuals is in part due to impairments in some of the structures (dorsal and ventral PFC, amygdala and angular gyrus) subserving moral cognition and emotion. Impairments to the emotional component that comprises the feeling of what is moral is viewed as the primary deficit in antisocials, although some disruption to the cognitive and emotional-emotional components of morality (particularly self-other relational thinking and emotion regulation) cannot be ruled out. While this neurobiological predisposition is likely only one of several biobehavioral processes involved in the etiology of antisocial behavior, it reflects significant moral issues for the legal system and neuroethics.

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INTRODUCTION

The burgeoning field of social neuroscience is beginning to provide important insights into the neural mechanisms that underlie the cognitive and affective processes that guide social behavior in everyday life. One particularly important subset within this area that has significant sociopolitical implications concerns the neural basis to antisocial behavior. The prevailing view that will be developed here is that there are some similarities between the neural systems underlying moral decision-making in normal individuals, and brain mechanisms that are also involved in both antisocial and violent and psychopathic populations. We suggest that this is not a chance association but instead represents a neural insight into the etiology of antisocial behavior.

A basic tenet underlying this thesis is that despite some differences in the constructs of childhood conduct disorder, antisocial personality disorder (APD), violence and psychopathy, a critically important common denominator to all is the failure to conform to the commonly accepted norms of society. Clearly, not all 'illegal' behavior is illegal, and not all features of a psychopathic personality concern immorality; there is more to antisocial behavior than a breakdown in the neural networks subserving moral thought and feeling. Nevertheless, the overlap between morality and antisocial disorders is substantial, and it is

regarded here that this is partly accounted for by disruption to neural systems common to both.

A general overview of studies of the neural structures implicated in those with persistent and significant antisocial behavior will first be outlined. The developing knowledge base on neural mechanisms underpinning moral judgment will then be presented, with emphasis on the behavioural correlates of moral decision-making. Differences and similarities between moral and antisocial neural correlates will be delineated, and a hypothesis that the rule-breaking behavior common to both affective and cognitive components of morality may predispose to the rule-breaking that is central to antisocial disorders.

IMAGING FINDINGS ON ANTSOCIAL, VIOLENT AND PSYCHOPATHIC GROUPS

Despite the increasing evidence for neurological impairment in antisocial individuals, very few structural and functional brain imaging studies have been conducted specifically on the recognised medical disorder for antisocial behavior, i.e. APD. For this reason, the following review will include brain imaging studies on antisocial, violent and psychopathic populations as well as those with APD. While those groups make up different populations, the key thesis of this study is that disruption to the neural systems underlying moral thinking and feeling gives rise to an 'antisocial tendency' (rule-breaking behavior), a key common feature to the pathological condition. We will focus on the structural and functional imaging findings based on anatomical structures that form frontal lobe (orbitofrontal cortex, OFC), dorsal prefrontal cortex (DLPFC), temporal lobe (superior temporal gyrus, amygdala/hippocampus) and other brain areas (parietal lobe/parietoangular/cingulate cortex).

Frontal lobe

Global prefrontal abnormalities. Prefrontal impairments are, perhaps, the best-replicated finding in the imaging literature on antisocial behavior. Reduced prefrontal glucose metabolism has been observed in murderers compared with normal controls (Raine et al., 1994), while acts of aggressive impulsive behavior have been associated with reduced metabolism in the orbitofrontal, anterior medial frontal, and left anterior frontal regions (Goyer et al., 1994). Using single photon emission computed tomography (SPECT), several studies have found significant correlations between reduced frontal blood flow and increased antisocial, aggressive behaviors (Oder et al., 1992; Kurzglo et al., 1996; Gerras et al., 1998; Soderstrom et al., 2000, 2002).

These functional impairments are paralleled by some evidence for structural prefrontal impairments. Three anatomical MRI (aMRI) studies have found significantly reduced prefrontal gray matter in antisocial and psychopathic individuals (Raine et al., 2000; Yang et al., 2005) and aggressive patients with temporal lobe epilepsy (Wellka et al., 2000). Although there is also one null finding (Dolan et al., 2002). Similar findings were reported by Kruesi et al., 2004 where a large (16%) reduction in prefrontal gray volume was found in children diagnosed with conduct disorder. However, because the sample size (N = 10), this finding was not statistically significant. Although these findings suggest widespread prefrontal deficits in APD, new evidence has begun to accumulate providing additional evidence indicating that the abnormality in APD may be localized to the orbitofrontal and dorsolateral prefrontal regions.

 Orbitofrontal cortex (OFC). Neurological research has indicated that individuals with orbitofrontal lesions are typically disinhibited, impulsive and uncaring with the consequences of their behavior. (Bolla et al., 1994; Brewer and Price, 2001). However, only two aMRI studies to date show reduced gray matter volumes in the OFC in antisocial individuals (Laakson et al., 2002; Yang et al., 2006a). Regarding function, two positron emission tomography (PET) studies have found reduced glucose metabolism in the OFC and medial PFC in impulsive patients and aggressive children (Johans et al., 2001; Sayer et al., 1999). Similar findings were reported to another two PET studies showing significant negative correlations between aggression and metabolism rate in the OFC and medial frontal cortex (Goyer et al., 1994; Pietrini et al., 2000). In addition, several aMRI studies have shown abnormal OFC activation in impulsive individuals with dorsal medial frontal cortex inhibition (Horn et al., 2003) and in APD patients during both inhibitory control (Vollm et al., 2004) and face-recognizing tasks (Drummond et al., 2005).

Dorsolateral Prefrontal Cortex (DLPFC). Patient studies have shown that damage to the DLPFC classically leads to problems in planning, attention, shift, decision-making and perseverative responding (Menes et al., 2002; Gomez-Beldarrain et al., 2004). However, only one structural study to date has assessed prefrontal subregions, finding reduced gray matter volumes in the left DLPFC and the right OFC in alcoholics with antisocial personalities compared with controls (Laakso et al., 2002). Regarding functional studies, reduced metabolism in the DLPFC has been found in aggressive patients (Hirano et al., 2000) and also aggressive children with epilepsy (Johans et al., 2001). In addition, two fMRI studies have observed abnormally DLPFC functioning in APD patients during both an emotional task (Schneider et al., 2003) and also in an inhibition task (Vollm et al., 2004).

Temporal lobe

The temporal lobe is the second major brain area traditionally associated with antisocial and aggressive behavior (Raine, 1993). Widespread abnormalities have been reported in several studies. Regarding structure, Kruesi et al. (2004) found a significant association between early-onset conduct disorder (without substance abuse comorbidity) and smaller temporal gray matter volumes. Similar findings of reduced temporal lobe volume have been reported in both incarcerated psychopaths (Dolan et al., 2002) and APD patients (Gainotti et al., 2003). Although functional imaging of SPET has documented reduced temporal lobe functioning in aggressive patients (Volkow and Taurelli, 1987; Amen et al., 1996). Another SPET study found significant negative correlations between psychopathy and temporal perfusion, particularly left temporal blood flow (Soderstrom et al., 2001). One fMRI study on violent offenders found reduced functioning in the temporal cortex compared with non-aggressive controls (Raine et al., 2001). There are also some failures to observe temporal lobe functional impairments; one PE T study of violent offenders failed to observe reduced temporal lobe glucose metabolism (Raine et al., 1997a).

These findings are beginning to confirm global structural and functional temporal lobe abnormalities, although results are not entirely consistent. As mentioned, some studies have attempted to localize abnormalities to the middle, anterior inferior, superior and medial temporal (amygdala and hippocampus) regions.

Middle temporal gyrus. One PET study found reduced bilateral metabolism in the middle temporal gyrus in aggressive children with temporal lobe epilepsy (Johans et al., 2001). A recent SPET study also revealed reduced blood flow in the right middle temporal gyrus in APD patients (Cothath et al., 2005).

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Anterior inferior temporal cortex. Using PET, Wong et al. (1997a) found reduced metabolism in the inferior temporal cortex in two separate groups of violent patients. Similar findings were reported in a SPECT study suggesting a significant reduction in regional blood flow in the left anterior inferior temporal cortex (Hirano et al., 2003).

Superior temporal gyrus. One study has revealed activation deficits in antisocial and psychopathic individuals localized to the right posterior superior temporal gyrus in a semantic processing task (Kuhl et al., 2004). Conversely, a second study observed reduced activation in the left hemisphere superior temporal gyrus in a memory task (Kumari et al., 2006).

Middle temporal cortex. In functional terms, two PET and one SPECT studies have found abnormal glucose metabolism in the middle temporal cortex. Vollhow et al. (1995) found significantly reduced metabolism in the middle temporal cortex in violent offenders (Vollhow et al., 1997a; Tlibonien et al., 2000) and psychopathic individuals (Yang et al., 2006b). Conversely, no volumetric differences have been found in aggressive patients with temporal lobe epilepsy (Trimbble and Van Ellef, 1995; Van Ellef, 2000), although the latter study also reported a significantly higher rate of amygdala atrophy (20%) in aggressive patients.

In terms of functioning, abnormalities in the amygdala have been found in one PET study one magnetic resonance spectroscopy (MRS) study, and several fMRI studies. In the PET study, Raine et al. (1997a) found abnormal functional asymmetries in murderers, showing lower left and increased right amygdala functioning. Critchley et al. (2002) found that in patients with mild retraction to have reduced metabolism in the right amygdala-hippocampal complex compared with non-violent controls. Somewhat surprisingly, two fMRI studies have reported increased amygdala activation in antisocial individuals while viewing negative visual content (Muller et al., 2003) and during an inverse conditioning task (Schaider et al., 2000). In contrast, reduced activation in the amygdala during the processing of affective stimuli has been found in criminal psychopaths (Kiehl et al., 2001), psychopathic individuals (Vett et al., 2002; Birbaumer et al., 2005), and adolescents with conduct disorders (Steeves et al., 2005).

Summary and Interpretation of Imaging Findings in Antisocial Populations

A fairly sizable imaging literature has now been built up on functional and (to a lesser extent) structural brain abnormalities in diverse antisocial groups, although for some structures such as the amygdala and hippocampus, the results are mixed.

Hippocampus

Hippocampal imaging studies have examined the functional integrity of this region and found abnormalities in murderers (Raine et al., 1998), criminal psychopaths (Kiehl et al., 2001) and violent offenders (Soderstroem et al., 2000), violent offenders with APD (Laakso et al., 2000), violent psychiatric patients (Kumari et al., 2006; Barkatsios et al., 2008) and antisocial alcoholics (Laakso et al., 2001).

Other Brain Areas

Several structural and functional imaging studies have suggested that the parietal lobe (particularly the angular gyrus) and anterior/posterior cingulate gyrus may also be compromised in antisocial groups.

Parietal Lobe. Regarding function, reduced metabolism has been found in the superior parietal cortex in aggressive patients (Hirano et al., 2000), murderers (Raine et al., 1997a) and individuals with impulsive personality disorders (Steeves et al., 1999). However, no structural MRI study has been conducted to date showing structural impairments in individuals with APD.

Only two studies to our knowledge have assessed functioning in the angular gyrus in individuals with APD. Both found significantly reduced activation, but in different hemispheres. Raine et al. (1997a) in a PET study found that murderers have reduced glucose metabolism in the left angular gyrus. Using SPECT, Soderstroem et al. (2000) found a significant blood flow reduction in the right angular gyrus in impulsive violent criminals.

Anterior and Posterior Cingulate. Several fMRI studies have provided evidence showing functional impairments in the anterior and posterior cingulate cortex (PCC) in APDs. Five fMRI studies have shown reduced activation in the anterior cingulate cortex (ACC) in criminal psychopaths during an effective memory task (Kiehl et al., 2001), APD patients during a working memory task (Kumari et al., 2006), psychopaths during a free-associating condition (Birbaumer et al., 2005), and conduct disorder patients during viewing of negative emotional pictures (Steeves et al., 2000b). In addition, abnormal ACC activation was noted in one study that reported in APD patients during a response inhibition task (Vollhow et al., 2004). With regard to the PCC, two functional imaging studies have shown PCC reduced activation in this region in criminal psychopaths (Kiehl et al., 2001) and aggressive patients (New et al., 2002).

Neurological Studies of Antisocial Behavior

Neurological studies of patients suffering trauma to discrete brain regions have provided invaluable data on neural mechanisms predisposing to antisocial behavior. An inevitable limitation of imaging findings is that they are correlational, while, in contrast, the natural accidents that comprise neurological findings bear more directly on causality. Intriguingly, findings from these studies converge with evidence from brain imaging studies, particularly with respect to the PFC.

Two key prefrontal neurological systems that bear on antisocial behavior have been delineated on the basis of the timing of neurological damage, and give rise to slightly different antisocial outcomes. 'Acquired sociopathy', in which accidental damage occurs to the ventromedial PFC in adulthood, has been shown to result in pseudo-psychopathic, disinhibited, antisocial behavior (Damasio et al., 1990; Damasio, 1994), together with bad decision-making and reduced antisocial skin conductance (see also Friston et al., 1996) and a tendency to comply with the demands of others. In contrast, the development of antisocial behavior is associated with lesions to the ventrolateral PFC, with the result that impulsive, antisocial behavior is characterized by the absence of social and moral considerations, and by a failure to make or implement plans. In this context, our research indicates that these two neurological systems provide the basis for the two different forms of antisocial behavior. However, despite the evidence, our research is not the first or only to have suggested that neurological differences may be associated with the development of antisocial behavior. Indeed, the idea that there may be a neurological basis for antisocial behavior has been a popular one, and it has been debated for many years.

Neural Basis to Moral Judgments

Behavior that breaks the moral guidelines set down by society is a fundamental feature of antisocial disorders, and almost all of these guidelines are related to the maintenance of social order. The nature of this break is not always clear, but in some cases it is clear that the behavior is not motivated by a desire to harm others, but rather to gain some personal advantage. In these cases, the behavior is often referred to as antisocial. In other cases, the behavior is motivated by a desire to harm others, and this behavior is often referred to as criminal. In all cases, the behavior is motivated by a desire to achieve some personal goal, and this goal is often related to the maintenance of social order. In this context, the behavior is often referred to as antisocial or criminal.

Neural basis to moral judgments refers to the neurological processes that underlie the ability to make moral judgments. These processes are thought to involve the prefrontal cortex, which is involved in the evaluation of moral situations. The prefrontal cortex is also involved in the regulation of behavior, and this is thought to be important in the ability to make moral judgments. The relationship between the prefrontal cortex and moral judgment is thought to be mediated by the ventromedial prefrontal cortex, which is involved in the evaluation of social outcomes. The ventromedial prefrontal cortex is thought to be involved in the ability to make moral judgments because it is involved in the evaluation of social outcomes, and this is thought to be important in the ability to make moral judgments.

In conclusion, our research indicates that the prefrontal cortex is involved in the ability to make moral judgments. This involvement is thought to be mediated by the ventromedial prefrontal cortex, which is involved in the evaluation of social outcomes. The relationship between the prefrontal cortex and moral judgment is thought to be mediated by the ventromedial prefrontal cortex, which is involved in the evaluation of social outcomes. In this context, the ability to make moral judgments is thought to be related to the functioning of the ventromedial prefrontal cortex, which is involved in the evaluation of social outcomes.
Recent imaging research is beginning to identify which cerebral areas are activated when subjects perform tasks involving moral consultations. This section provides a brief overview of what we have learnt so far about brain mechanisms subsuming moral decision-making from 12 DMRI studies. As will be seen, a wide variety of tasks have been used asking somewhat different questions on the neural correlates of morality. Despite this diversity, a number of key brain areas appear to be a common denominator for moral information-processing.

Regions consistently activated—medial PFC, angular gyrus and ventral PFC

The ground-breaking study in this area (Greene et al., 2001) focused on the specific differences between making judgments (i.e. 'appropriate' or 'inappropriate') on 'moral personal dilemmas' (e.g. throwing a person out of a sinking life-boat to save others), and 'moral impersonal dilemmas' (e.g. keeping money found in a lost wallet). Moral dilemmas involving a personal component, compared with both impersonal moral dilemmas and nonmoral dilemmas, activated the medial frontal gyrus (BA 9 and 10), the posterior cingulate (BA 23) and both left and right angular gyr (BA 39). This initial study suggested that these structures play a crucial role in the emotional processes that influence personal moral decision-making. Studies since 2001 have confirmed the importance of the mediodorsal PFC, angular gyrus and posterior cingulate in processing moral stimuli, and at the same time implicated a further structure—the ventral PFC. The medial PFC (and in some tasks, BA 10) is activated by passive viewing of pictures depicting moral versus nonmoral scenes (Greene et al., 2001; Moll et al., 2002a; Harenski and Hamann, 2006), making judgments about moral versus nonmoral sentences (Oliva-vetra et al., 2005; Heekeren et al., 2006), and reading moral versus nonmoral descriptions (Moll et al., 2002b; Herenstein and Hamann, 2006). In addition, Moll et al. (2002b) found activation in the region of the superior temporal sulcus bordering the angular gyrus when responding to unpleasant moral versus unpleasant nonmoral statements. The weight of support for angular gyrus involvement in moral tasks, in association with the posterior superior temporal sulcus, is consequently as strong as that for medial PFC involvement.

One additional brain region that could not be imaged in the initial study by Greene et al. (2001) due to susceptibility artefact, yet which is being increasingly implicated in moral judgment tasks, is the ventral PFC. This region encompasses the DPC and the gyrus rectus (also broadly termed ventrolateral and ventromedial, respectively). Activation in this region has been found during passive viewing of pictures depicting moral vs. nonmoral violations (Moll et al., 2002a; right orbitofrontal), responding to unpleasant moral vs. unpleasant nonmoral statements (Moll et al., 2002b; gyrus rectus and orbitofrontal), passive viewing of morally disgusting vs. nonmorally disgusting statements (Moll et al., 2002b; left orbitofrontal), and semantic decision-making (Moll et al., 2002b; left orbitofrontal), moral decision-making (Moll et al., 2002b; orbitofrontal), moral decision-making (Moll et al., 2002b; left orbitofrontal), and semantic decision-making (Moll et al., 2002b; right orbitofrontal), moral decision-making (Moll et al., 2002b; left orbitofrontal), and semantic decision-making (Moll et al., 2002b; right orbitofrontal), moral decision-making (Moll et al., 2002b; left orbitofrontal), and semantic decision-making (Moll et al., 2002b; right orbitofrontal), moral decision-making (Moll et al., 2002b; left orbitofrontal), and semantic decision-making (Moll et al., 2002b; right orbitofrontal). 

Regions less consistently activated—posterior cingulate, amygdala and temporal pole

While the polar/medial PFC, ventral PFC and angular gyrus encapsulates areas with the strongest evidence for activation during moral tasks, three other regions also need to be considered. Activation of the posterior cingulate has been observed during moral personal versus impersonal judgments (Harenski and Hamann, 2006). In addition, Greene et al. (2004) observed consistent activation of the posterior cingulate across all three of their experimental moral decision-making conditions (personal vs. impersonal, difficult vs. easy and utilitarianism vs. non-utilitarianism). Several studies have also reported amygdala activity (Moll et al., 2002a; Herenstein and Hamann, 2006; Loo et al., 2006). The temporal pole has also been activated in some studies (Oliva-vetra et al., 2005; Moll et al., 2002b; Herenstein and Hamann, 2006; Loo et al., 2006). It is likely that some form of our moral values are stored in a deep evolutionary history where emotions—cognitions—constituted the driving force of moral action. The interesting success of early hominids was largely predicated on reciprocation and sharing social structure. Nevertheless, selfishness (taking but not giving resources) can constitute a competing evolutionary stable strategy that has to be kept in check for the survival of the species. Negative moral emotions likely evolved to counteract the breakdown of social conventions. Moral feelings of indignation, disdain, disgust and contempt can give rise to the strongest emotions of outrage and vengeance that then give rise to ostracization of the child from the social group, injury or even death. At this level, morality is largely emotion-driven, relatively automatic, and has little or no higher cognitive control component in early hominids. As hominid society became more complex, higher-order cognitive processes likely became increasingly important for both dealing with more complex moral dilemmas, and for regulating the expression of moral emotions.

Despite the evolution of social mechanisms to deter antisocial 'cheating' behavior, it has been argued that antisocial behavior is an evolutionary stable strategy—a pre-programmed behavioral approach that maximizes reproductive fitness. Psychopathy has been viewed as the full expression of this 'cheating' strategy (Fagan, 1993). At low base rates within the populations, psychopaths can be successful in extracting resources from other individuals before moving on to other social groups to avoid the consequences of moral rule-breaking. Psychopathic traits of supravalid charm, egocentrism, manipulation, pathological lying and deception, promiscuous sexual behavior, lack of remorse and guilt, superficial relationships and general parasitic lifestyle are viewed as key components of this evolutionary stable strategy that has been observed in both humans and nonhumans (Babiak and Hare, 1995). It is possible that psychopaths adopt a transient, unstable, 'stimulation-seeking' lifestyle, moving from one place to another to avoid ultimate detection—an essential attribute to the moral machinery that would otherwise be meted out to them.

In this evolutionary context, an essential component of this successful cheating strategy must be a gene mechanism that lacks a core moral sense. One way to create such a cheating mechanism would be to engineer individuals lacking the moral circuitry essential for moral feelings and behavior. One prediction generated by this model is that antisocial and psychopathic individuals would manifest impairments in the brain mechanism requisite for morals, particularly those neural processes critical to the
Neural bases of morality and antisocial behavior may be implicated in antisocial, psychopathic behavior.

CONCLUSIONS AND SUMMARY

In summary, we have argued the following:

(i) brain regions implicated in antisocial, violent, and psychopathic populations include both dorsal and ventral regions of the PFC, amygdala, hippocampus, angular gyrus, anterior cingulate, and temporal cortex including the superior temporal gyrus,

(ii) regions activated during moral decision-making in normal individuals include the polar medial PFC, ventral PFC, angular gyrus, amygdala, and posterior cingulate,

(iii) brain areas associated with both moral reasoning and antisocial behavior significantly overlap,

(iv) the rule-breaking, immoral behavior of antisocial and psychopathic individuals may in part be due to impairments in those brain regions subserving moral cognition and emotion,

(v) while impairments to the moral emotional system may be primary in antisocials, disruption of moral cognitive and cognitive-emotional systems are also possible.

Fig. 1 A schematic diagram of brain regions implicated in antisocial behavior (red), areas associated with both moral reasoning and antisocial behavior (green), and regions common to both antisocial behavior and moral decision-making (yellow).

A. Raine and Y. Yang
Neural bases of morality and antisocialThis neuro-moral theory of antisocial, violent and psychopathic behavior is regarded as provisional. The precise loci of the likely multiple neural deficits in antisocial groups (particularly within the large regions comprising prefrontal and temporal cortex) remains to be delineated, while an understanding of the neural basis to moral decision-making is clearly still in its infancy. For example, there are hints that temporal pole impairments activate different neural systems underlying moral decision-making, respectively, yet, limitations in the evidence to date precluded inclusion in Figure 1. A compromised moral circuit will be only one of multiple etiological processes ultimately found to predispose to the complex constructs of antisocial/aggressive/psychopathic behavior. Intersections between neural and social risk factors for antisocial behavior cannot be ignored (Raine et al., 1997b; Caspi et al., 2002), raising yet another layer of complexity to a full elucidation of antisocial disorders. Despite these caveats, we believe that a neural moral hypothesis of antisocial disorders is worthy of examination in future studies. Neuroscientists and laypeople alike are beginning to raise important questions about the implications of new neuroscience knowledge for society, the law, and civil liberties (Moore, 2004), leading to the beginning of a new sub-discipline of “neuroethics” (Farah, 2004). Psychopathology may not be “morally insane” in any strict legal sense as they are capable of distinguishing right from wrong, but if they lack the capacity for the feeling of what is moral due to neurobiological impairments beyond their control, are they fully responsible for their criminal behavior? If not, what are the implications for punishment and our concepts of both justice and retribution? This challenging question that lies at the interface of law, neuroscience and neuroethics, begs for further enlightenment from future systematic imaging research on both morality-processing and antisocial behavior.

Conflict of InterestNone declared.

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