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Review article

The neuromoral theory of antisocial, violent, and psychopathic behavior

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ABSTRACT

The neuromoral theory of antisocial behaviors argues that impairment to the neural circuitry underlying morality provides a common foundation for antisocial, violent, and psychopathic behavior in children, adolescents, and adults. This article reviews new findings in two research fields since this theory was first proposed: brain mechanisms underlying moral decision-making, and brain systems subserving antisocial behaviors. The neuromoral theory is updated to take into account new empirical findings. Key areas implicated in both moral decision-making and the spectrum of antisocial behaviors include fronto-polar, medial, and ventral prefrontal cortical regions, and the anterior cingulate, amygdala, superior temporal gyrus, and angular gyrus / temporoparietal junction. It is hypothesized that different manifestations of antisocial behavior are characterized by differing degrees 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 by neuromoral dysfunction. Limitations of the current model, social contextual factors, neural remediation interventions, ascertaining whether the affective or cognitive component of empathy is most implicated, and directions for future research are outlined. One forensic implication of the model is that significant impairment to the neuromoral circuit could constitute diminished criminal responsibility.

1. Introduction

This review aims to address one of the most important questions in forensic clinical psychology and psychiatry which is rarely if ever raised, let alone answered: *What is the most important neurobiological process that underlies so many different forms of antisocial, aggressive, violent, conduct-disordered, and psychopathic criminal behaviors?* It is argued here that functional impairment to the neural circuit underlying morality is, in varying degrees, a common denominator to this wide spectrum of antisocial disorders across the lifespan.

This article begins by outlining the original neuromoral theory of antisocial behavior (Raine and Yang, 2006) and its empirical foundations. It then reviews new MRI findings over the past thirteen years since the neuromoral theory was first outlined in two research fields: brain mechanisms underlying moral decision-making, and brain systems subserving antisocial behaviors. Four different lines of evidence bearing on the question of whether this model has been supported or refuted are then outlined, together with a revision of the neuromoral model. It then moves into the challenging question of the extent to which different manifestations of antisocial, violent, psychopathic, and criminal behaviors are differentially predicated on neuromoral dysfunction. Finally, this extended neuromoral model is discussed within the contexts of childhood socialization, treatment, and the criminal justice system, together with directions for future research.

2. The moral brain and antisocial behavior

A pioneering brain imaging study of moral decision-making at the turn of the century documented greater activation of the medial and polar prefrontal cortex, posterior cingulate, and the angular gyrus to personal moral dilemmas compared to impersonal dilemmas (Greene et al., 2001). This lead to more extended fMRI studies which in addition to confirming these findings, further implicated the ventromedial prefrontal cortex (Hutcherson et al., 2015). Reviews of the functional neuroanatomy of antisocial, violent, and psychopathic behavior were also implicating similar brain areas (Raine, 2013).

Thirteen years ago a review of these two fields synthesized findings and for the first time generated the hypothesis that there was a common neural denominator to both antisocial behavior and moral decision-making, based on functional imaging and neurological studies (Raine and Yang, 2006). With respect to antisocial behavior, it was argued that key areas found to be functionally or structurally impaired in antisocial populations included dorsal and ventral regions of the prefrontal cortex, the amygdala, hippocampus, angular gyrus, posterior

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cingulate, and subregions of the temporal cortex including anterior and superior gyri. For moral decision making, it was suggested that research up to that point in time implicated a neural circuit consisting of polar, medial, and ventral regions of the prefrontal cortex, the superior temporal sulcus, and the angular gyrus, with initial findings additionally implicating the temporal pole and the amygdala.

Juxtaposing these two sets of findings, it was proposed that there is substantial overlap between brain mechanisms implicated in antisocial / psychopathic behavior on the one hand, and moral decision-making on the other (Raine and Yang, 2006). Regions argued to be common to antisociality and morality included polar, medial, and ventral prefrontal cortices, the amygdala, the superior temporal gyrus, and the angular gyrus. Based on these findings, it was hypothesized from a casual standpoint that impairments to these regions predisposed to impairments in morality which in turn predisposed to antisocial behavior.

3. Recent findings on the neural correlates of morality and antisociality

To what extent has this neuromoral theory of antisocial behavior been supported or refuted? Since 2006 different lines of research have investigated this proposal on the confluence of the neural correlates of morality and antisociality. Taken together, findings from more recent research have broadly supported the initial model, but have also provided new insights that require modification to the model. These lines of research will now be outlined, followed by a revision of the neuromoral model.

At a neurological level one innovative lesion study provides significant support for the neuromoral model of criminal behavior (Darby et al., 2018). Seventeen cases were identified where brain lesions were followed in time by antisocial / criminal behavior, with the most common lesion site being to the ventral prefrontal cortex (nine cases). While these brain lesions covered disparate parts of the brain, using lesion network mapping the authors documented that all 17 connected to the same neural network, consisting of the orbitofrontal cortex, the medial prefrontal cortex, and the anterior temporal cortex - broadly consistent with prior work on the neuroanatomical correlates of antisocial behavior (Raine and Yang, 2006). Furthermore, 16 of the 17 lesion sites were connected to the ventromedial prefrontal cortex and the nucleus accumbens. They then documented that this criminality-associated connectivity pattern overlapped with the functional network commonly associated with moral-decision-making. Specifically, lesion-locations in the 17 antisocial cases overlapped with moral decision-making areas in the ventromedial prefrontal cortex, inferior frontal gyrus, and temporal cortex (see Fig. 4a, Darby et al., 2018). Findings were replicated in a sample of 23 lesion cases in which the temporal link between onset of lesion and later criminal behavior was uncertain. Furthermore, this pattern of findings was not observed for four other neuropsychiatric conditions. These results are broadly consistent with the neuromoral theory of antisocial behavior (Raine and Yang, 2006), although there are differences which shall be returned to later.

A second line of evidence consists of MRI studies examining whether psychopathic populations (who represent the extreme of the antisocial continuum) show functional impairments in brain areas common to antisocial behavior and morality when either making moral decisions or viewing morally-relevant stimuli. The first study in this area documented not just that highly psychopathic individuals show reduced amygdala activation when contemplating moral decisions, but also that those scoring high on the interpersonal factor of psychopathy (manipulation, conning, superficiality and deceitfulness) additionally showed reduced activation in the medial prefrontal cortex, posterior cingulate and angular gyrus (Glenn et al., 2009). These initial findings received support from a later study which also documented a breakdown in the neural circuit subserving moral decision-making in psychopaths, documenting lower activation in the medial prefrontal cortex and posterior cingulate (Pujol et al., 2012). Reduced amygdala activation has also been highlighted in psychopaths when judging the moral acceptability of fear-evoking statements (Marsh and Cardinale, 2014), and also in psychopathic and antisocial youth during a moral judgement implicit association task (Marsh et al., 2011). These fMRI findings on non-neurological cases place a focus on the amygdala as a key region in the neuromoral theory, in contrast to the lesion study of Darby et al., (2018).

A third line of evidence involves studies presenting morally distressful stimuli to psychopaths and which observed reduced activation in different components of the neuromoral circuit. Findings include reduced activation in psychopaths in ventromedial prefrontal and anterior temporal cortices (Harenski et al., 2010), the temporal-parietal junction (Harenski et al., 2014), dorsolateral prefrontal cortex and caudate (Yoder et al., 2015), anterior cingulate (Fede et al., 2016), and amygdala (Harenski et al., 2009). These findings are in relatively broad agreement with the original neuromoral model, but additionally implicate an area of the striatum (caudate).

A fourth line concerns decreased coupling of neuromoral brain regions during moral paradigms in psychopathic and antisocial individuals. These findings include decreased coupling of the right amygdala and the temporoparietal junction with the anterior cingulate, anterior insula, striatum, and ventromedial prefrontal cortex (Yoder et al., 2015), and also reduced coupling of the amygdala and the orbitofrontal cortex (Marsh et al., 2011). These findings are again in broad agreement with the neuromoral model, assuming extension of the angular gyrus into the temporoparietal junction.

Taken together, these four related but different sources of evidence provide, with some exceptions, support for the original neuromoral theory of antisocial behavior that posits that brain mechanism dysfunctional in antisocial, violent, and psychopathic individuals in part map onto the neural circuit underling morality, and the proposition that impaired morality mediates the brain – antisocial relationship (Raine and Yang, 2006).

4. Revisions to the neuromoral theory of antisocial behavior

Despite broad support for the neuromoral model of antisocial behavior, there are discrepancies in findings since the development of this model which require resolution. Regarding the functional imaging studies on morality and antisocial behavior (research lines two-four above), while almost all studies identify dysfunctional brain regions in antisocial populations which are lying on the moral neural circuit, most individual studies do not implicate all of these regions, but rather implicate one (e.g. amygdala; Marsh and Cardinale, 2014) or two regions (Yoder et al., 2015). A possible explanation may be that different antisocial populations (youth, adult, psychopathic, criminal) may present with neural dysfunction to somewhat different regions of the morality network, all of which can contribute at some level to moral dysfunction and antisocial behavior.

Furthermore, while almost all of the brain areas implicated in the above studies were documented in the neuromoral model (Raine and Yang, 2006), some were not. These include the nucleus accumbens and inferior frontal gyrus in the lesion study (Darby et al., 2018), and the striatum in one brain imaging study (Yoder et al., 2015). Initial brain imaging findings on abnormalities in the striatum (caudate, putamen, globus pallidus, nucleus accumbens) in psychopaths did not begin until almost a decade ago (Glenn et al., 2010) and have led to increased attention to this region in reward-based theories of antisocial and psychopathic behavior (Blair, 2013; Raine, 2018). One review of the neural basis to moral decision-making has also implicated the striatum (Fumagalli and Priori, 2012), while the nucleus accumbens has been implicated in both criminal behavior and moral circuitry (Darby et al., 2018). In addition, reduced activation of the caudate has also been observed in psychopaths to morally distressful stimuli (Yoder et al., 2015). This would suggest future candidacy of the striatum in

neuromoral theory of antisociality. Nevertheless, recent meta-analyses of morality have not directly implicated the striatum (Garrigan et al., 2017; Eres et al., 2018; Han, 2017) which may make its inclusion in the model at the current time debatable.

A limitation of the original neuromoral theory in the light of research since 2006 is that the anterior cingulate was implicated in the neural-antisocial circuit but not in the neuromoral circuit. One recent meta-analysis has however identified the anterior cingulate as part of the neuromoral circuit (Boccia et al., 2017), while two further meta-analyses also include the cingulate in this circuit (Garrigan et al., 2017; Han, 2017). A fourth recent meta-analysis does not however (Eres et al., 2018), and the anterior cingulate was not featured in the lesion network study of criminality (Darby et al., 2018). This mismatch must be placed in the context that although findings from lesion studies yield valuable insights, they inevitably cannot be taken as a direct parallel to individual differences in brain functioning in the larger population. Several meta-analyses have however confirmed structural and functional abnormalities in the anterior cingulate in youth with disruptive behavior disorders (Gavita et al., 2012), and antisocial adults (Yang and Raine, 2009), providing further support for the anterior cingulate as a possible common denominator to antisociality and morality.

A final limitation in the original model is that the insula was not included as research findings on its role in antisocial behavior and morality were at that time sparse. Since 2006 an increasing body of research has implicated this structure in antisocial behavior. Recent meta-analytic reviews of brain imaging findings on antisocial populations have all found evidence at some level to implicate abnormalities in the insula (Alegria et al., 2016; Aoki et al., 2014; Noordermeer et al., 2017). Findings implicating the insula in morality is somewhat less persuasive, with some meta-analyses including this region as part of the neural moral network (Boccia et al., 2017; Eres et al., 2018) while others do not (Garrigan et al., 2017; Han, 2017). On balance however, there does seem to be sufficient evidence to at least provisionally consider the insula as playing a role in both antisocial behavior and moral decision-making.

Based on these considerations, a revised neuro-model of antisocial behavior is presented in Fig. 1. The insula and anterior cingulate have been added as areas common to both antisociality and morality. The striatum (caudate, putamen, globus pallidus, nucleus accumbens – only the caudate and putamen are illustrated) is currently added as an area that is specific to antisocial behavior, although as noted above there is increasing support for its role in both antisocial and moral processing that may require future revision. The angular gyrus remains as a common area, but should be taken more widely to represent the associated temporoparietal junction.

It is likely that this updated model will still require future revisions for several reasons. First, it is agnostic as to laterality effects because while they have been reported in individual studies, there is no clear consistency in findings. Second, despite the growth in brain imaging research in both of these areas and the consequent publication of meta-analyses, there are still a surprising number of inconsistencies in the field that preclude firm conclusions. Third, as already noted, some areas of the striatum have been implicated in both antisociality and morality, making it a candidate for further inclusion in the common network. Despite these caveats, there is growing consensus that fronto-temporal regions are primary regions of consideration. Specifically, some of the best-supported candidates for commonality between morality and antisociality include the medial prefrontal, ventral prefrontal, and temporal lobe regions that include anterior, superior, temporoparietal, and amygdala sub-regions.

5. Forms of antisocial behavior and the neuromoral brain

While the immoral brain can be broadly construed as a common denominator to many forms of antisocial behavior, there are likely subtle differences in neuromoral impairment between different manifestations of offending. This section provides an initial and very provisional analysis of what forms of antisocial behavior may be most – and least – associated with the neuromoral theory of antisociality on the basis of both structural and functional brain imaging.

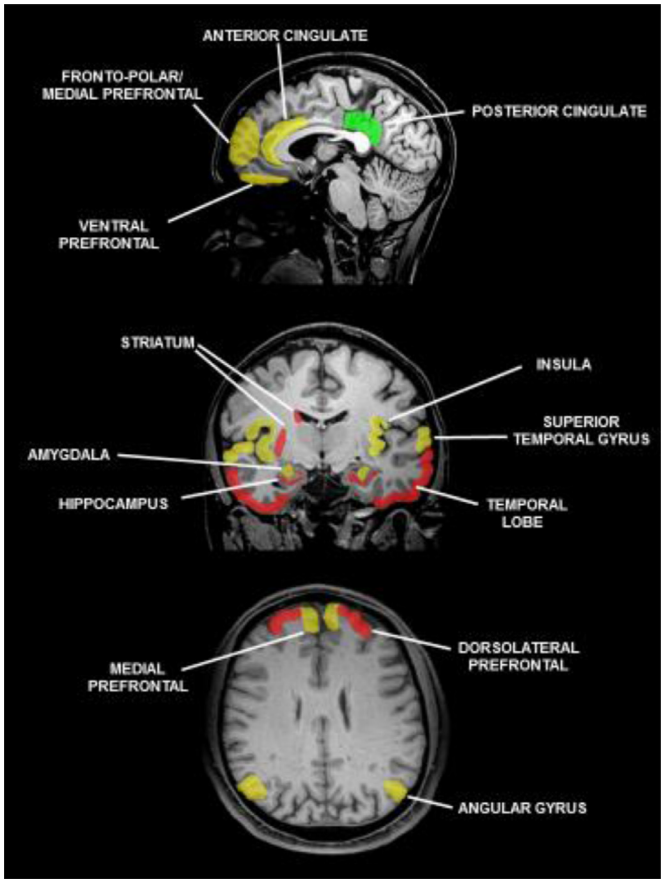

Fig. 1. The revised neuromoral model illustrating brain regions impaired only in antisocial groups (red), activated only in moral decision-making (green), and regions common to both antisocial behavior and moral decision-making (yellow).(For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 1 outlines the key components of this neuromoral – offending classification. Most offenders are hypothesized to have some level of

Table 1
The neuromoral-offending classification in which different processes and offense types are subsumed under mild or high levels of neuromoral impairment. CU, callous-unemotional; DBD, disruptive behavior disorder; ODD, oppositional defiant disorder; CD, conduct disorder; APD, Antisocial Personality Disorder.

	Level of neuromoral impairment	
	Mild - Moderate	*Moderate - High*
Autonomic nervous system	Heightened	Reduced
Neurocognitive functioning	Moderate impairment	Mild impairment
Personality	Impulsive	Planful
Development		
Life-course	Adolescent limited	Life-course
DBD diagnosis	ODD	CD
CD specifier (CU)	Absent	Present
Adult diagnosis	non-APD	APD
Antisocial manifestations		
Psychopathy	Secondary	Primary
Aggression	Reactive	Proactive
Violence	Domestic homicide	Mass / Serial killer
White-gray-blue	White collar / Gray collar	Blue collar
Sex offending	Pedophilia	Rape
Drug addiction	Present	Absent

neuromoral structural / functional impairment (fronto-polar, medial prefrontal, anterior cingulate, insula, superior temporal gyrus, amygdala, angular gyrus) which differs only in degree, with some having moderate to high levels of impairment (e.g. primary psychopaths) while others have only moderate levels (e.g. secondary psychopaths) or quite minimal levels (reactive aggression). As such, the model argues for differences in degree rather than in kind, and with dysfunction to some brain areas (e.g. prefrontal) being implicated in varying degrees in many antisocial manifestations.

At the level of the autonomic nervous system (ANS), reduced ANS functioning including reduced anticipatory fear, reduced reactivity to stress and empathy-inducing stimuli, and low physiological arousal, are anticipated to go alongside those antisocial sub-types characterized by high neuromoral impairment. In contrast, higher levels of ANS responsivity are hypothesized to characterize those with *less* neuromoral impairment, in part due to enhanced (and not diminished) amygdala functioning. At the personality level, those with only mild neuromoral impairment are hypothesized to be more deficient in impulse control, whereas those with more neuromoral impairment are viewed as lacking this risk factor and instead demonstrating more regulated, planful behavior with more complete recognition of the negative consequences to their victims.

From a developmental perspective, those with mild neuromoral impairment are viewed as being more likely to lie on the more common adolescent-limited antisocial pathway which is more dominated by peer influences, in contrast to a much less common sub-type with more significant neuromoral impairment who are more likely to be life-course persistent (Moffitt, 2018). At a diagnostic level, children with relatively significant neuromoral dysfunction are viewed to be on the path for more serious conduct disorder and as having the lack of prosocial emotions (callous-unemotional) DSM 5 specifier in adolescence, and antisocial personality disorder in adulthood. Children with mild neuromoral dysfunction are less likely to be characterized by callous-unemotional traits and more likely to have the milder and relatively less serious diagnosis of oppositional defiant disorder, with a reduced likelihood of manifesting full antisocial personality disorder in adulthood.

Turning to psychopathy, aggression / violence, and criminal subtypes, one of the firmer predictions of this model is that those with a stronger neuromoral impairment will more likely be predisposed to primary psychopathy. Such individuals possess the core, classic psychopathic features that includes blunted affect and who have been historically viewed as “morally insane” (Pritchard, 1837), while psychopathy in general has been empirically linked to impaired moral reasoning (Blair et al., 2005). Alternatively, weaker neuromoral impairment is suggested in secondary psychopaths who are more characterized by heightened anxiety and stress reactivity, impulsivity, reactive aggression, and substance abuse (Patrick, 2018).

Regarding aggression, a key distinction is that the mild neuromoral impairment group are hypothesized to display reactive aggression, in contrast to the proactive aggression displayed by those with more significant neuromoral impairment. This is predicated on the notion that poor emotion regulation, limited impulse control, and enhanced reactivity to mildly provocative emotional stimuli are instead viewed as more salient predispositions for reactive aggression than neuromoral impairment (Lickley and Sebastian, 2018), whereas an impaired moral sense has greater etiological relevance in the context of more proactive, predatory, and planned use of aggression that requires a stronger immoral indifference to the well-being of victims (Kempes et al., 2005).

At the more serious level of homicide, impaired neuromoral circuitry is hypothesized in serial killers who have a callous disregard for their victims, and to a somewhat lesser extent in mass killers who are instead more characterized by mental illness and paranoid thinking (Dutton et al., 2013; Stone, 2015). In contrast, those perpetrating domestic homicide are more likely to have more severe mental illness (psychosis) and less likely to have prior convictions and antisocial personality disorder compared to perpetrators of homicide outside the home (Hanlon et al., 2016). Furthermore, spouse abusers have been characterized by *enhanced* (not reduced) amygdala responsivity (Lee et al., 2008), as well as increased activation in the anterior cingulate, insula, and middle prefrontal cortex to emotional stimuli (Bueso-Izquierdo et al., 2016), which is not consistent with reduced activation of the neuromoral circuit.

A distinction is also proposed between white-collar crime and blue-collar crime. Blue-collar (street) crime that involves interpersonal confrontation with the victim is viewed to be driven in part by blunted feelings of wrongfulness and impaired moral judgement (Stams et al., 2006). In contrast, white collar crime is hypothesized to involve a reduced degree of neuromoral impairment as, with some exceptions, it is more impersonal, involving institutions rather than individuals as victims. Furthermore, white-collar criminals have been found to have neurocognitive *superiorities*, including enhanced executive functions, enhanced psychophysiological attention and arousal, and *increased* (not decreased) cortical gray matter thickness in the ventromedial prefrontal cortex, inferior frontal gyrus, and the temporal-parietal junction (Raine et al., 2012), brain areas implicated in morality. Furthermore, gray-collar criminals (those who perpetrate both white-collar and blue-collar crime) have been found to show *increased* volume of the superior frontal gyrus and anterior cingulate, unlike blue-collar offenders (Ling et al., 2018).

Regarding sex offending, neuromoral dysfunction is tentatively viewed to be somewhat less characteristic of pedophiles where instead strong sexual attraction to prepubescent children is argued to be characterized by risk factors of sexual abuse and in utero neurodevelopmental perturbations (American Psychiatric Association, 2013). In contrast, those who rape their victim despite that victim's distress and suffering have been found in one diffusion tensor imaging study to have impairments in brain mechanisms subserving moral decision-making (Chen et al., 2016). Finally, offenders whose crimes are embedded in substance use disorders are thought to have impairments more centered on cortico-striatal pathways (Kravitz et al., 2015), although some disruption to neuromoral circuitry cannot be fully discounted.

6. Conclusions, remaining questions, and future directions

The neuromoral theory of antisocial behaviors constitutes a functional neuroanatomical model which posits that a foundational cause of disparate antisocial behaviors resides in dysfunction to a network of brain areas providing the infrastructure for moral behavior. As a causal model, its core proposition is that dysfunction to one or more areas of the neuromoral circuit results in impairment to feeling, thinking, and behaving in a moral way which in turn lays the foundation for antisocial, violent, and psychopathic behavior. At the same time there are critical questions that require answers. What are the remaining gaps in the neuromoral literature that need to be filled? How can neuromoral dysfunction be remediated? And what are the broader forensic implications with respect to the criminal justice system?

One gap that is conspicuous by its absence is the role of mental illness. Are antisocial individuals who are comorbid for significant mental illness characterized by relatively mild or relatively significant neuromoral dysfunction? At one level, mental illness in itself can have a direct pathway to antisocial behaviors (e.g. paranoid ideation predisposing to reactive aggression), and yet antisocial behaviors and psychopathology may well have commonalities in dysfunctional brain networks. Future research on the question of whether or not the presence of mental illness moderates antisocial brain – moral brain relationships can shed light on this important clinical issue.

Another lacuna in our knowledge is that empirically there is little if any research on which subtypes of antisocial behavior are more, or less, predicated on appropriate neuromoral functioning. As such, this aspect of the model is much more speculative and underdeveloped. This is a major gap in our knowledge that is sorely in need of redress. Are

secondary psychopaths and reactively aggressive individuals really less impaired in their neuromoral circuitry than primary psychopaths and proactively aggressive individuals?

An equally important void is the lack of both research on children and longitudinal research. While the neuromoral theory has implications for conduct disorder and oppositional defiant disorder (see Table 1), most of the extant imaging research is based on adults. Research on children with disruptive behavior disorders is sorely needed. From the standpoint of longitudinal research an important unresolved question is whether early impairments to the neuromoral circuit predict later offending. Because such research is easier to recommend than to undertake, a beginning program of research could target early neurocognitive or psychophysiological proxies for sub-components of the neural moral circuit. As one example, autonomic fear conditioning is predicated on the amygdala, insula, anterior cingulate, and orbitofrontal cortex (Gao et al., 2010). Taking autonomic fear conditioning as a proxy for functioning in these neuromoral regions, poor fear conditioning at age 3 has been shown to predict to adult criminal offending 20 years later, providing psychophysiological support for the neuromoral theory (Gao et al., 2010). As such, initial developmental tests of the neuromoral model can be made in the absence of brain imaging methodology by judicious choice of proxies.

One Achilles heel in the neuromoral theory, at least in its current form, is that it is agnostic to social circumstances. One can imagine a child with intact neuromoral circuitry, but who is brought up in a deprived neighborhood with antisocial parents and negative peer influences. Will not that child become quickly acculturated into an immoral way of living, in much the same way that the Artful Dodger in Charles Dicken's *Oliver Twist* became adeptly acculturated into an antisocial way of life in Fagin's kitchen? How many ostensibly good citizens in Nazi Germany, despite normal neuromoral circuitry, became acculturated into the immorality of Jewish oppression due to social influences? Social neuroscience research on how early social forces both moderate and influence the immoral brain – antisocial relationship could help address this question.

Drilling down to the constitutional elements of morality, which component is more impaired in offenders – the cognitive or the emotional? This is a central question that remains open to empirical evaluation. The neuromoral theory has argued that the emotional *feeling* of what is morally wrong constitutes the primary deficit, with cognitive components of morality being secondary (Raine and Yang, 2006). In contrast, research on brain lesion cases suggests that emotional components such as empathy are not involved, whereas cognitive components of morality such as theory of mind and reward-based decision making are involved (Darby et al., 2018). These two competing predictions on the primacy of affective versus cognitive attributes of morality in relation to the neuromoral theory requires further resolution.

A compelling question, assuming that the premise of the neuromoral theory is correct, concerns how we are to judiciously alter the immoral brain to promote moral behavior in the future. Brain interventions for antisocial behavior are extraordinarily rare and yet potentially represent a promising direction for future preventive efforts. Preliminary experimental evidence has shown that individuals randomized into transcranial direct current stimulation of the prefrontal cortex not only reduced their intent to commit both physical and sexual violent criminal acts at a later day, but also enhanced their cognitive assessment of how immoral such criminal acts are (Choy et al., 2018). Mediation analyses supported a causal model flowing from prefrontal upregulation to enhanced moral thinking to reduced criminal intent – with intention viewed as a prelude for criminal behavior. This very initial study both provides a tentative example of how neuromodulation of brain abnormalities could potentially be addressed, and also exemplifies how experimental research can test the neuromoral model by selectively manipulating some of the neural areas constituting the neuromoral circuit.

The latter part of this review has speculatively focused on the question of which sub-forms of offending are most impacted by neuromoral dysfunction. Yet perhaps the deeper and more profound question posed by the neuromoral theory ultimately resides in how we are to judiciously dispose of offenders who plead that they are not morally responsible for their actions due to significant impairment of their neuromoral circuitry, caused beyond their control. The “moral insanity” of primary psychopaths may not be a legal defense in that cognitively they know right from wrong, and because our legal concept of responsibility is founded on cognitive capacity (Morse, 2008). Nevertheless, emotion informs decision-making (Damasio, 1994). If psychopaths lack the *feeling* of what is moral due, for example, to amygdala impairment (Yang et al., 2009), and if such impairment to their moral sense is not of their making, can we morally hold them fully responsible for their immoral actions? If we cannot, what are the implications for punishment and our concepts of both justice and retribution?

This thorny neurolegal issue potentially represents a more significant implication of the neuromoral theory. Should impairment to the neuromoral circuit be recognized as a significant mitigating factor in criminal trials given the importance of a fully developed emotional moral capacity for lawful behavior? Moral responsibility would seem to require intactness of neuromoral circuity. It may be one thing to argue that reduced prefrontal functioning in a defendant predisposed them to reduced impulse control, poor emotion regulation, and hence violent behavior. However, emotion regulation and impulse control are removed from the legal concept of responsibility. In contrast, to argue that the brain basis to *moral* thinking and feeling are compromised in a violent offender comes dangerously close to challenging moral responsibility, a concept which in itself is just a short step removed from criminal responsibility. Future enlightenment from neuroimaging research on both morality and antisociality, combined with further cross-disciplinary neurolegal discussion on this issue, could help resolve questions stemming from the neuromoral model that are central to forensic psychology and psychiatry.

Finally, grand theories of criminal behavior which claim for one predominating cause must be humbled by the empirical reality that offending is a complex jigsaw puzzle made up of many different causal pieces that do not fit neatly together. The neuromoral theory is but one piece, consisting at best of a limited number of inter-related neural elements in a much larger cortical and subcortical space. It is however a testable model that generates clear predictions and raises a significant question on criminal responsibility that requires resolution. Whether this theoretical perspective on disparate antisocial behaviors can be translated into meaningful empirical advances that can be instantiated into future clinical and legal practice remains to be seen.

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References

Alegria, A.A., Radua, J., Rubia, K., 2016. Meta-analysis of fMRI studies of disruptive behavior disorders. Am. J. Psychiatry 173, 1119–1130.

American Psychiatric Association, 2013. Diagnostic and Statistical Manual of Mental Disorders (5th ed.), 5th ed. American Psychiatric Association, Washington, DC.

Aoki, Y., Inokuchi, R., Nakao, T., Yamasue, H., 2014. Neural bases of antisocial behavior: a voxel-based meta-analysis. Soc. Cognit. Affect. Neurosci. 9, 1223–1231.

Blair, R.J.R., 2013. The neurobiology of psychopathic traits in youths. Nat. Rev. Neurosci. 14, 786–799.

Blair, R.J.R., Mitchell, D., Blair, K., 2005. The Psychopath: Emotion and the Brain Blackwell. Oxford.

Boccia, M., Dacquino, C., Piccardi, L., Cordellieri, P., Guariglia, C., Ferlazzo, F., Ferracuti, S., Giannini, A.M., 2017. Neural foundation of human moral reasoning: an ALE meta-analysis about the role of personal perspective. Brain Imaging Behav. 11, 278–292.

Bueso-Izquierdo, N., Verdejo-Roman, J., Contreras-Rodriguez, O., Carmona-Perera, M.,

Perez-Garcia, M., Hidalgo-Ruzzante, N., 2016. Are batterers different from other criminals? An fMRI study. Soc. Cognit. Affect. Neurosci. 11, 852–862.
Chen, C.Y., Raine, A., Chou, K.H., Chen, I.Y., Hung, D., Lin, C.P., 2016. Abnormal white matter integrity in rapists as indicated by diffusion tensor imaging. BMC Neurosci. 17.
Choy, O., Raine, A., Hamilton, R.H., 2018. Stimulation of the prefrontal cortex reduces intentions to commit aggression: a randomized, double-blind, placebo-controlled, stratified, parallel-group trial. J. Neurosci. 38, 6505–6512.
Damasio, A., 1994. Descartes' error: Emotion, reason, and the human brain GP Putnam's Sons, New York.
Darby, R.R., Horn, A., Cushman, F., Fox, M.D., 2018. Lesion network localization of criminal behavior. Proc. Natl. Acad. Sci. 115, 601–606.
Dutton, D.G., White, K.R., Fogarty, D., 2013. Paranoid thinking in mass shooters. Aggression Violent Behav. 18, 548–553.
Eres, R., Louis, W.R., Molenberghs, P., 2018. Common and distinct neural networks involved in fMRI studies investigating morality: an ALE meta-analysis. Soc. neurosci. 13, 384–398.
Fede, S.J., Borg, J.S., Nyalakanti, P.K., Harenski, C.L., Cope, L.M., Sinnott-Armstrong, W., Koenigs, M., Calhoun, V.D., Kiehl, K.A., 2016. Distinct neuronal patterns of positive and negative moral processing in psychopathy. Cognit. Affect. Behav. Neurosci. 16, 1074–1085.
Fumagalli, M., Priori, A., 2012. Functional and clinical neuroanatomy of morality. Brain 135, 2006–2021.
Gao, Y., Raine, A., Venables, P.H., Dawson, M.E., Mednick, S.A., 2010. Association of poor childhood fear conditioning and adult crime. Am. J. Psychiatry 167, 56–60.
Garrigan, B., Adlam, A.L.R., Langdon, P.E., 2017. The neural correlates of moral decision-making: a systematic review and meta-analysis of moral evaluations and response decision judgements (vol 108, pg 88, 2016). Brain Cognit. 111, 104–106.
Gavita, O.A., Capris, D., Bolno, J., David, D., 2012. Anterior cingulate cortex findings in child disruptive behavior disorders. A meta-analysis. Aggression Violent Behav. 17, 507–513.
Glenn, A.L., Raine, A., Schug, R.A., 2009. The neural correlates of moral decision-making in psychopathy. Mol. Psychiatry 14, 5–6.
Glenn, A.L., Raine, A., Yaralian, P.S., Yang, Y.L., 2010. Increased volume of the striatum in psychopathic individuals. Biol. Psychiatry 67, 52–58.
Greene, J.D., Sommerville, R.B., Nystrom, L.E., Darley, J.M., Cohen, J.D., 2001. An fMRI investigation of emotional engagement in moral judgment. Science 293, 2105–2108.
Han, H., 2017. Neural correlates of moral sensitivity and moral judgment associated with brain circuitries of selfhood: a meta-analysis. J. Moral Educ. 46, 97–113.
Hanlon, R.E., Brook, M., Demery, J.A., Cunningham, M.D., 2016. Domestic homicide: neuropsychological profiles of murderers who kill family members and intimate partners. J. Forensic Sci. 61, S163–S170.
Harenski, C.L., Edwards, B.G., Harenski, K.A., Kiehl, K.A., 2014. Neural correlates of moral and non-moral emotion in female psychopathy. Front. Hum. Neurosci. 8. https://doi.org/10.3389/fnhum.2014.00741.
Harenski, C.L., Harenski, K.A., Shane, M.S., Kiehl, K.A., 2010. Aberrant neural processing of moral violations in criminal psychopaths. J. Abnorm. Psychol. 119, 863–874.
Harenski, C.L., Kim, S.H., Hamann, S., 2009. Neuroticism and psychopathy predict brain activation during moral and nonmoral emotion regulation. Cognit. Affect. Behav. Neurosci. 9, 1–15.
Hutcherson, C.A., Montaser-Kouhsari, L., Woodward, J., Rangel, A., 2015. Emotional and utilitarian appraisals of moral dilemmas are encoded in separate areas and integrated in ventromedial prefrontal cortex. J. Neurosci. 35, 12593–12605.
Kempes, M., Matthys, W., de Vries, H., van Engeland, H., 2005. Reactive and proactive aggression in children - a review of theory, findings and the relevance for child and adolescent psychiatry. Eur. Child Adolesc. Psychiatry 14, 11–19.
Kravitz, A.V., Tomasi, D., LeBlanc, K.H., Baler, R., Volkow, N.D., Bonci, A., Ferre, S., 2015. Cortico-striatal circuits: novel therapeutic targets for substance use disorders. Brain Res.. 1628, 186–198.
Lee, T.M.C., Chan, S.C., Raine, A., 2008. Strong limbic and weak frontal activation to aggressive stimuli in spouse abusers. Mol. Psychiatry 13, 655–656.
Lickley, R.A., Sebastian, C.L., 2018. The neural basis of reactive aggression and its development in adolescence. Psychol. Crime Law 24, 313–333.
Ling, S.C., Raine, A., Portnoy, J., Yang, Y., Schug, Ho, R.M.H., 2018. Increased frontal lobe volume as a neural correlate of gray-collar offending. J. Res. Crime Delinquency. https://doi.org/10.1177/0022427818802337.
Marsh, A.A., Cardinale, E.M., 2014. When psychopathy impairs moral judgments: neural responses during judgments about causing fear. Soc. Cognit. Affect. Neurosci. 9, 3–11.
Marsh, A.A., Finger, E.C., Fowler, K.A., Jurkowitz, I.T., Schechter, J.C., Yu, H.H., Pine, D.S., Blair, R., 2011. Reduced amygdala-orbitofrontal connectivity during moral judgments in youths with disruptive behavior disorders and psychopathic traits. Psychiatry Res.-Neuroimaging 194, 279–286.
Moffitt, T.E., 2018. Male antisocial behaviour in adolescence and beyond. Nat. Hum. Behav. 2, 177–186.
Morse, S.J., 2008. Psychopathy and Criminal Responsibility. Neuroethics 1, 205–212.
Noordermeer, S.D.S., Luman, M., Greven, C.U., Veroude, K., Faraone, S.V., Hartman, C.A., Hoekstra, P.J., Franke, B., Buitelaar, J.K., Heslenfeld, D.J., Oosterlaan, J., 2017. Structural brain abnormalities of attention-deficit/hyperactivity disorder with oppositional defiant disorder. Biol. Psychiatry 82, 642–650.
Patrick, C.J., 2018. Cognitive and emotional processing in psychopathy. In: Patrick, C.J. (Ed.), The Handbook of Psychopathy. Guilford, New York, pp. 422–456.
Pritchard, J.C., 1837. A Treatise on Insanity and Other Disorders Affecting the Mind Carey and Hart. Philadelphia.
Pujol, J., Batalla, I., Contreras-Rodriguez, O., Harrison, B.J., Pera, V., Hernandez-Ribas, R., Real, E., Bosa, L., Soriano-Mas, C., Deus, J., Lopez-Sola, M., Pifarre, J., Menchon, J.M., Cardoner, N., 2012. Breakdown in the brain network subserving moral judgment in criminal psychopathy. Soc.Cognit. Affect. Neurosci. 7, 917–923.
Raine, A., 2013. The Anatomy of Violence: The Biological Roots of Crime Pantheon Books. New York.
Raine, A., 2018. Antisocial Personality as a Neurodevelopmental Disorder. Annu. Rev. Clin. Psychol. 14 (14), 259–289.
Raine, A., Laufer, W.S., Yang, Y.L., Narr, K.L., Thompson, P., Toga, A.W., 2012. Increased executive functioning, attention, and cortical thickness in white-collar criminals. Hum. Brain Mapp. 33, 2932–2940.
Raine, A., Yang, Y., 2006. Neural foundations to moral reasoning and antisocial behavior. Soc., Cognit. Affect. Neurosci. 1, 203–213.
Stams, G.J., Brugman, D., Dekovic, M., van Rosmalen, L., van der Laan, P., Gibbs, J.C., 2006. The moral judgment of juvenile delinquents: a meta-analysis. J. Abnorm. Child Psychol. 34, 697–713.
Stone, M.H., 2015. Mass murder, mental illness, and men. Violence Gender 2, 51–86.
Yang, Y.L., Raine, A., 2009. Prefrontal structural and functional brain imaging findings in antisocial, violent, and psychopathic individuals: a meta-analysis. Psychiatry Res.-Neuroimaging 174, 81–88.
Yang, Y.L., Raine, A., Narr, K.L., Colletti, P., Toga, A.W., 2009. Localization of deformations within the amygdala in individuals with psychopathy. Arch. Gen. Psychiatry 66, 986–994.
Yoder, K.J., Harenski, C., Kiehl, K.A., Decety, J., 2015. Neural networks underlying implicit and explicit moral evaluations in psychopathy. Transl. Psychiatry 5. https://doi.org/10.1038/tp.2015.117.