

Forensic neurobiology underlying violent criminal behavior

Amy Du Beau, PhD

Matanuska Forensic Science
Palmer, Alaska 99645 USA

© 2018, Amy Du Beau

ISBN 978-1-5342-0416-4
Glasstree Academic Publishing

This work is licensed under a Standard Copyright License. All rights reserved.

Abstract: Violent criminal behavior may be a sequela of functionally and structurally compromised prefrontal and corticolimbic cortices. These anatomically distinct yet functionally integrated regions of the human brain confer qualities of moral sensibility and intentionality of action. Criminal behavior leading to conviction necessitates the commission of a prohibited act, *actus reus*, coincidentally occurring with a guilty state of mind, *mens rea*. Sentencing determinations markedly differ for those who intentionally violate compared to reckless acts and such outcomes can be critically life-impactful. However, making inferential assessments about an aggressor's mental state can be a challenging task for legal experts. This meta-analysis reviews how the functional somatotopy of brain regions associated with aggression can be forensically assessed to contextualize violent criminal behavior to facilitate legal processes. Because brain scans have diagnostic credibility, by extension, they are increasingly becoming persuasive forensic evidence. A centralized neuroimaging database may emerge as a game-change for legal processes. The intercalated framework of neurolaw uniquely offers great power to elucidate criminological factors within the statute.

Contents

1. Introduction: Neurobiology underlying violent criminal behavior	3
Figure 1: Brain regions	6
2. Neurolaw and the role of neuroimaging	7
3. Aggression	9
4. Psychopathology	11
5. Reactive versus instrumental psychopathological aggression.....	14
6. Psychopathology versus antisocial personality disorder	16
7. Antisocial personality disorder	17
8. Other neuropsychological disorders and acquired pseudopsychopathology	19
8.1 Traumatic Brain Injury	21
8.2 Intellectual disability	22
9. Intentionality and <i>mens rea</i>	23
10. Forensic implications	26
11. Conclusion.....	28
Acknowledgement	29
References	29

Abbreviations: American Psychiatric Association, APA; Antisocial Personality Disorder, ASPD; Computerized Axial Tomography, CAT; Deoxyribonucleic Acid, DNA; Diagnostic and Statistical Manual of Mental Disorders, DSM; Electroencephalogram, EEG; Frontotemporal Lobe Degeneration, FTD; Functional Magnetic Resonance Imaging, fMRI; Intellectual Disability, ID; Positron Emission Tomography, PET; Traumatic Brain Injury, TBI

Keywords: Aggression, Neuroanatomy, Neuroimaging, Neurolaw, Neuroscience, Psychopathy

1. Introduction: Neurobiology underlying violent criminal behavior

Anatomically distinct yet functionally integrated prefrontal and corticolimbic regions of the human brain underlie the expression of identifiable behaviors. The prefrontal cortex uniquely distinguishes adult humans from other animals, conferring qualities of reasoning, mental abstraction and reflective awareness of our own thought processes. The evolutionarily ancient limbic system, responsible for emotional expression, is highly phylogenetically conserved as compared to the relatively modern frontal cortex. What happens should our neural precocity go awry? Evidence overwhelmingly supports the notion that dysfunction of both prefrontal and corticolimbic structures can intersect with an array of violent behaviors.

Criminal behavior leading to a conviction necessitates the commission of an unlawful act, *actus reus*, guilty act, coincidentally occurring by a state of mind, *mens rea*, guilty mind, that implies accountability for the action. Investigating how these implicated neuroanatomical regions interact is at the crux of understanding how compromised behavior ultimately result in criminal violence. Case studies of criminals having neuropsychological and/or cognitive disorders or injury provide provocative insights into violent behavior which may be a sequela of functionally and/or structurally compromised brain regions. Functional neuroimaging, which gives quantitative perspective to the neurology underlying criminal actions, is increasingly becoming standard evidence in controversial criminal cases (Kumarasamy 2014).

Our developed prefrontal cortex confers conscientiousness, affording accountability for our actions. Over two hundred years ago, French physician Philippe Pinel recognized rare individuals who exhibited deviant behavior yet had no indication of any apparent cognitive disorder such as hallucinations, “*manie sans délire*.” Historically, case studies dating back to 1835 have reported the onset of antisocial personality traits after frontal lobe injury, notably the famous case of Phineas Gage who survived albeit with drastic personality changes after his frontal lobe was accidentally ablated with an iron rod. Frontal lobe injury is associated with compromised axonal

projections to distal limbic regions that are involved in 'primitive impulses' (Grafman *et al.*, 1996). Resultantly, dysfunction in prefrontal domains may influence social perception, self-control, judgment, decision-making processes and normative morality.

Functional connectivity mapping revealed a pattern of diminished gray matter involving prefrontal cortices and limbic-paralimbic regions coupled with altered connections in the dorsal frontal lobe in psychopathic subjects, suggestive of a weakened link between emotional and cognitive domains (Contreras-Rodriguez *et al.*, 2015). Classical studies using positron emission tomography (PET) to investigate functional abnormalities associated with aggression in murderers, particularly those who plead guilty by reason of insanity, found reduced cerebral glucose metabolism in prefrontal regions, including anterior medial prefrontal and lateral prefrontal cortices (Raine *et al.*, 1997). Medial prefrontal cortices are involved in self-reflection and rumination; critical attributes that foster social emotions such as empathy, guilt and embarrassment (Qin & Northoff 2011). Misallocated recruitment of prefrontal and subcortical structures may characterize individuals who commit affective acts of violence. Assessing the somatotopy of these structures may yield valuable forensic information in conjunction with an existing behavioral profile.

Neurobiology addresses morality discriminately. Corticolimbic domains cumulatively associated with conscientiousness, specifically the amygdala and medial prefrontal cortices have been implicated in non-clinical studies of moral judgments (Harenski *et al.*, 2014). Normative morality is the universally recognized modicum of social behavior, regardless of any given group or cultural affiliation, that is based on fairness, reciprocity, treating others as we wish to be treated; 'do no harm' and basic human compassion. In contrast, descriptive morality refers to conduct expectations held by particular cohesive societal groups that ensure right and wrong; accepting common customs and following agreed upon rules and laws (Mendez 2009).

Neurobiology is principally concerned with normative morality. This meta-analysis generally

addresses the neural substrate underlying violations of normative morality, *malum in se*, while recognizing the invariable overlap with descriptive morality. Aberrant neural connectivity and/or signalling within and between prefrontal structures necessitate a predisposition to depreciated conscientious awareness, *mens rea*, but may not be sufficient for commencing an act of violence, *actus reus*, without subcortical recruitment of limbic regions. Inferences about such mental states motivating criminal acts are firmly ensconced in the law.

This meta-analysis is not intended to be an exhaustive description of specific neuroanatomical regions and their functionality *per se* but rather to characterize the dysfunction of structures pertinent to the concomitant expression of violent criminal behaviors. Using the PubMed National Center for Biotechnology Information database, current literature was reviewed, focusing on research articles addressing the neurobiology underlying criminality with emphasis on psychopathology and pseudo-psychopathology.

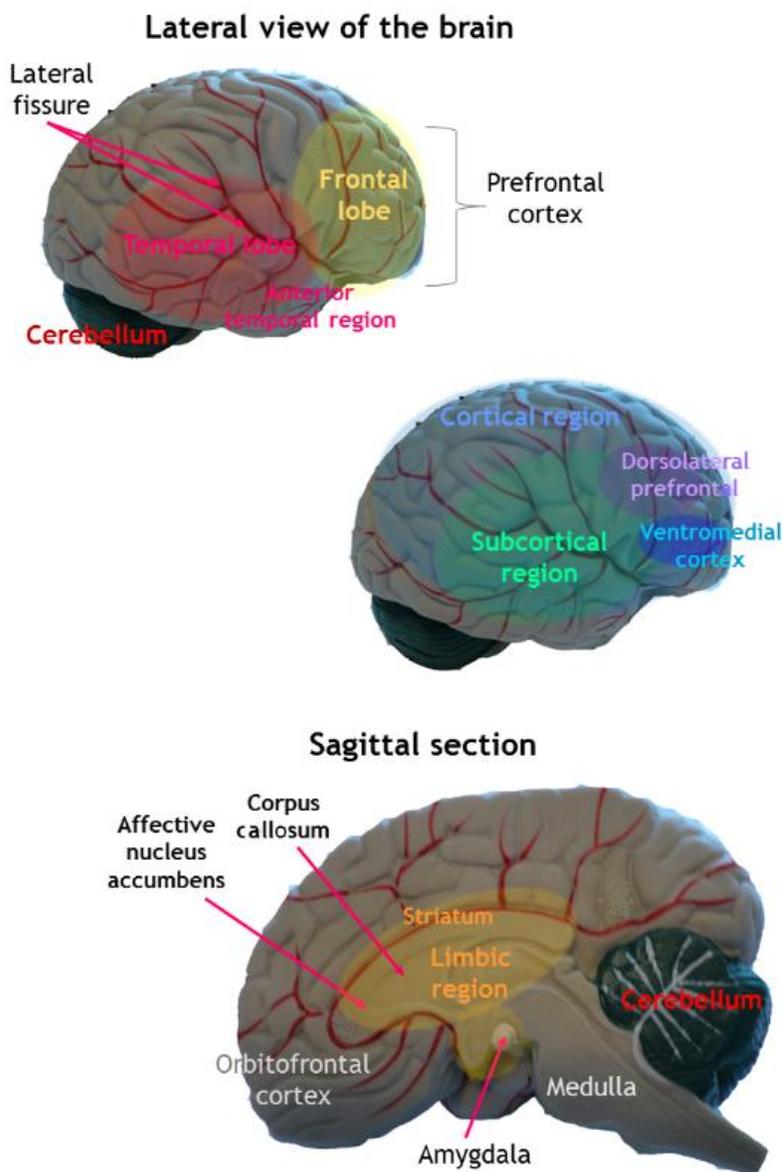


Figure 1: Brain regions

To illustrate the neuroanatomy associated with criminality, a standard acrylic anatomical brain model (Classic Brain Model, Lexington, SC USA) was digitally photographed at zoom 1 (Sony Cybershot, 12.1 Megapixels). Images were enhanced, cropped, sized, shaded and labelled using Adobe Photoshop Elements 13 Expert software (Adobe Systems Incorporated, version 13.1, 2014). For anatomical context, please refer to this figure (above) throughout this meta-analysis.

Anatomically distinct regions of the human brain are identified by pastel shading and corresponding colored text. The sagittal section divides the brain into left and right hemispheres. Medial regions are closer to the midline whereas lateral regions are closer to the skull. Ventral refers to towards the front whereas dorsal regions approach the cerebellum/medulla.

2. Neurolaw and the role of neuroimaging

Neurolaw is the burgeoning field integrating neuroscience as applied to law, drawing ideas from criminology, neurobiology, physiology, psychology and sociology. An outstanding issue is the extent to which criminal punishment can modify behavior precipitated by neural abnormalities. Sentencing based upon modifiability i.e., punishment decisions based on neuroplasticity, the brain's ability to change itself, is proposed by neuroscientist David Eagleman (Eagleman 2011). In cases where prefrontal and/or corticolimbic dysfunction putatively associated with violent acts are presented as criminal defense, the sentencing would entail psychiatric institutionalization for palliative treatment rather than prison. So neurobiological forensic evidence for determinations of guilt or innocence might be especially helpful during pretrial or sentencing determinations (Farahany 2016). Neuroscience is beginning to address novel perspectives regarding violent criminal behavior that can be a game-changer for legal processes. Criminal lawyers might question whether defendants, witnesses or jurors are lying or accurately recalling the truth. Polygraphic evidence contextualized by understanding underlying psychophysical mechanisms emerges as a powerfully persuasive investigative tool. Tempering this argument, the reliability of lie detection has always been controversial, the fundamental issue being conditions under which such 'mind reading' techniques could be coercive or taken out of context; polygraphic results are only as credible as their interpreters. In general, deception is associated with greater activation of the prefrontal and/or anterior cingulate cortices, whereas truthfulness is typically not associated with any greater activity of any cerebral region (Jiang *et al.*, 2016). Regardless of debate, truth verification technology is already underway e.g., the 'No Lie MRI' technique (Greely 2013; Pardo 2013).

Because brain scans have diagnostic credibility, by extension, they are becoming standard evidence in pivotal legal trials beyond the experimental or medical context to substantiate criminal behavior at the neuroanatomical level. The advent of functional and structural

neuroimaging techniques, such as PET, electroencephalography (EEG), computerized axial tomography scan (CAT) and functional magnetic resonance imaging (fMRI) affords sensitive detection of regional brain dysfunction with precision and accuracy that can otherwise elude conventional psychological assessment. Forensic brain scans presented as evidence can better elucidate a psychiatric diagnosis to facilitate an insanity defense or mitigate legal outcomes (Presidential Commission 2015).

The John Hinckley trial was the first criminal defense to use neuroscientific imaging. Hinckley shot United States President Ronald Reagan and three others in 1981. Psychiatrist David Bear diagnosed Hinckley with severe schizophrenia and depression. In defense, Bear presented CAT scans of Hinckley's brain in court, revealing that Hinckley's cortical sulci, the lateral fissures, were significantly wider and deeper than typical (Kelkar 2016). (Excessive neural pruning during adolescence and early adulthood is an etiology of schizophrenia). Former United States President Barack Obama's bioethics commission stated that neuroscience is currently used in a quarter of capital cases and that percentage is rising rapidly (Presidential Commission 2015).

While neurological abnormalities detected by imaging technology cannot ascribe any coincident violent behavior to an alleged crime, results may be diagnostically suggestive. Brain scans are taken after the alleged crime, so a drawback potentially exploited by prosecutors is that investigators cannot know with definitive certainty whether the revealed neurological aberration exactly coincided with the commission of the violent criminal act in question, *actus reus*. Relying on images of functional neuroanatomy to reveal morality may be arguably presumptuous. While neuroimaging can definitively diagnose e.g., a tumor or lesion, such scans cannot implicate behavioral outcomes with predictable certainty. However, fMRI analysis has been recently used in the context of the law, including predicting psychopathy (Vilares *et al.*, 2017). Brain scans are increasingly becoming critical arbitrators in legal settings.

3. Aggression

Aggression is conventionally defined as any threatening or physically assaultive behavior intended to harm another (Coccaro *et al.*, 2011). Violence means actions that inflict physical harm in violation of normative social constructs. These overlapping definitions may be used interchangeably in this meta-analysis. Aggressive behaviors could have been evolutionarily adaptive if we imagine ancient humans competing for requisite resources such as food, territory or even mates in times of scarcity. In our modern society, such aggressive traits can become counterproductive, violating our consensual ethical standards i.e., descriptive morality, *malum prohibitum*, and inflicting harm onto others i.e., normative morality, *malum in se*.

Based on data from neuroimaging studies, while psychopathic aggressors display different neural functioning, they are often effectively able to use compensatory mechanisms in basic cognitive tasks (Freeman *et al.*, 2015). So gross aberrations of affect and behavior may be disguised since cognitive, motor and sensory functioning still remain relatively intact. Animal studies identify various midbrain structures that underlie aggression e.g., medial preoptic area, lateral septum, anterior and ventromedial hypothalamus periaqueductal gray and bed nucleus of stria terminalis (Nelson & Trainor 2007). In human subjects, neuroimaging data increasingly implicate analogous anomalies in corticolimbic circuits associated with aggressive behavior (Coccaro *et al.*, 2011).

Orbitomedial prefrontal cortices have repeatedly been demonstrated to exert inhibitory control over explosive aggression (Brower & Price 2001; Coccaro *et al.*, 2011; Duffy & Campbell 1994; León-Carrión & Ramos 2003). Deficits in this region are also specifically implicated in flawed decision-making, suggesting a link between these two behavioral functions (Coccaro *et al.*, 2011). Ventromedial cortices appear to be recruited when aggressive urges are suppressed rather than enacted (Patrick 2008). Dysfunction within both ventromedial cortices and amygdalar regions render psychopaths relatively insular to the aversive consequences of moral transgression and thus less likely to avoid committing them (Harenski *et al.*, 2014). Imaginal

anger is associated with enhanced activation of the left orbitofrontal cortex, right affective nucleus accumbens and bilateral anterior temporal regions (Bufkin & Luttrell 2005). Early case reports link orbitofrontal EEG spiking to violent hallucinations and assaultive behavior (Fornazzari *et al.*, 1992). And orbitofrontal, ventromedial and dorsolateral prefrontal dysfunction contribute to violent behaviors in different ways.

The ventromedial and anterior cingulate cortices mediate an array of social and affective decision-making functions, and deficits may further contribute to violent behaviors (Koenigs 2012). Orbitofrontal and ventromedial cortices appear to function in concert to refine complex decision making processes; subtleties that appear to help understand punishment contingencies. The orbitofrontal cortex was found to equalize the value of competing outcomes so that the value of differing rewards can be compared (Montague & Berns 2002; Schoenbaum & Roesch 2005) (whereas the ventromedial cortex plays a key role in representing the value of goal-directed outcomes and options (Grabenhorst & Rolls 2001; O'Doherty 2011)).

Deeper in the brain, the anterior cingulate-orbitofrontal region is responsible for assigning emotional valence to social stimuli (Shackman *et al.*, 2011). Anatomically embedded within the corticobasal ganglia, the anterior cingulate cortex belongs to the reward/incentive circuit linked to emotional processing (Haber & Behrens 2014). Neural threat circuitry includes the amygdala, hypothalamus and dorsal periaqueductal gray matter (Gregg & Siegel 2001; Pement 2013) and these corticolimbic regions may be regulated by frontal regions e.g., orbital, medial and ventrolateral frontal cortices (Blair 2004; Pement 2013). If the frontal lobe exerts executive control over this threat circuitry, then such deficits may impair threat response regulation and even hinder the function of proximal and distal regions receiving their axonal projections, increasing the likelihood of unforeseen and potentially problematic behavior. Psychopathy related behavioral disorders are consistently correlated with dysfunction of orbitofrontal-limbic

structures, which are associated with somatic reactions to emotion, behavioral planning and responsibility-taking (Del Casale *et al.*, 2015).

4. Psychopathology

Psychopaths are responsible for an inordinate proportion of violent crime (Anderson & Kiehl 2014) with their behavior consistently correlated with dysfunction of orbitofrontal-limbic and paralimbic structures (Del Casale *et al.*, 2015; Koenigs 2012), regions critical for coordinating cognitive and affective functions. Modern neuroscientific research is advancing rapidly, reifying psychopathy with practical ramifications for how the law regards psychopathic criminality.

Psychopaths are typified by their lack of normative 'moral emotions,' guilt and empathy, which contribute to criminality and callous disregard for harming others (Harenski *et al.*, 2014).

An estimated 1% of the general populace meets the criteria for psychopathy and psychopaths constitute 15 - 22% of the prison population and commit over 50% more criminal offenses than non-psychopathic prisoners (Hare 1996; 1999). Psychopathy is a strong predictor of violent recidivism (Cornell *et al.*, 1996; Harris & Rice 1991; Porter *et al.*, 2009). Compared to non-psychopaths, psychopaths showed a higher risk for incarceration (20-25 times) and violent recidivism (4-8 times) coupled with resistance to rehabilitative treatment (Kiehl 2014). The Diagnostic and Statistical Manual of Mental Disorders (DSM) uses a polythetic set of criteria to characterize all personality disorders. The American Psychiatric Association (APA) recognizes psychopathy as a constellation of personality traits having stochastic etiologies. The classical diagnosis of psychopathy as historically characterized by psychiatrist Hervey Cleckley (Cleckley 1941) as well as modern models (Lynam *et al.*, 2011; Patrick *et al.*, 2009) and the Psychopathy Checklist-Revised (Hare 2003) have a controversial relationship with the DSM-V. Regardless of classification, psychopathy is a well-established personality disorder with robust clinical,

neuroscientific and forensic application. Frontal structures such as the posterior orbitomedial cortex exhibit rich reciprocal connections with the amygdala (Ghashghaei & Barbas 2002; Ghashghaei *et al.*, 2007) that may serve to regulate output of amygdalar nuclei (Ghashghaei & Barbas 2002). There is evidence of greater prefrontal and amygdalar structural deficits in 'unsuccessful' psychopaths, which may predispose them to impaired behavioral control and decision-making, thus making them more prone to convictions (Yang *et al.*, 2010). Psychopaths were also found to have thinner anterior temporal cortices bilaterally as well as thinner cortices in the left insula and right inferior frontal gyrus compared to healthy controls (Ly *et al.*, 2012). Such dysfunction of ventromedial prefrontal and amygdalar structures may contribute to impaired moral socialization (Birbaumer *et al.*, 2005; Blair 2008; Harenski *et al.*, 2014).

The recognition of happy, sad and fearful emotional expressions was observed to be deficit in psychopathic subjects. In subsequent brain imaging analysis, psychopaths with better recognition of these facial emotional expressions showed higher volumes in prefrontal structures, the somatosensory cortex, anterior insula, cingulate cortex and posterior lobe of the cerebellum (Pera-Guardiola *et al.*, 2016). Studies further exploring anatomical correlates may be useful for elucidating neuro-functional evidence gleaned from psychiatric research.

Emotional face morphing tasks may be useful for distinguishing subtle emotive impairments in psychopathic subjects (Pera-Guardiola *et al.*, 2016). In sum, such research directives are suggestive of neuroarchitectural distinctions between psychopathic subjects based on their acuity in recognizing and empathically characterizing emotive facial expressions.

Amygdala are responsible for the manifold processing of nuanced emotional expression and reinforcement of reward/punishment contingencies in tandem with the adjacent ventromedial prefrontal structures. Inextricably linked with ventromedial prefrontal cortices, amygdala relay important stimulus information (Price 2003; Shoenbaum & Roesch 2005) associated with the rapid detection of threat and initiation of response (Coccaro *et al.*, 2011). Amygdala are

associated with aversive or fear conditioning, instrumental learning (reward) and retrieval of socially relevant knowledge, such as facial trust-worthiness and approachability (Mendez 2009). Studies of youths with psychopathic tendencies relative to controls found compromised functional connectivity between amygdala and ventromedial prefrontal cortices (Marsh *et al.*, 2008). Incarcerated adult male psychopaths showed a similar etiology, with reduced engagement of anterior temporal cortices and amygdala shown during the commission of moral judgments (Harenski *et al.*, 2014). If the frontal lobe and amygdala are implicated in psychopathy, then ostensibly tracts allowing communication between these two broad regions may show developmental defects (Pemment 2013; Craig *et al.*, 2009).

The hippocampus underlies memory formation linked with autonomic nervous system responses. Functional imaging studies revealed exaggerated asymmetry of hippocampi in the brains of unsuccessful psychopaths, specifically that the right anterior hippocampi were bigger than the left as compared to healthy controls (Raine *et al.*, 2004). Further, asymmetry of both hippocampi and amygdala were found in murderers compared with control subjects (Patrick 2008; Raine *et al.*, 1997). Resultant poor memory consolidation would invariably contribute to their inability to make sound social cognitive judgments (Pemment 2013; Raine *et al.*, 2004).

Findings from a recent psychiatric study comparing psychopathic criminals to non-criminals having high and low levels of impulsive traits suggest that overt criminality is not necessarily characterized by abnormal reward expectation, but rather by enhanced communication between striatal regions involved in reward i.e., amygdala, nucleus accumbens, ventral palladium, coupled with frontal brain regions (Geurts *et al.*, 2016). The amygdala relays stimulus reinforcement learning information to the orbitofrontal cortex, allowing good decision making to occur. Regional deficits may account for why psychopaths may struggle with forming stimulus-punishment associations and are poor at engaging in adaptive behaviors that conflict with other

primary motivators (Anderson & Kiehl 2014). Cumulative findings suggest that 'gut reactions' to threat miscues may be a learned response reinforced by misappropriated reward expectation. Classical neuropsychological studies showed that psychopathic individuals may be impaired in both behavioral extinction and reversal learning (Budhani & Blair 2005; Budhani *et al.*, 2006). Recruitment of adjacent corticolimbic structures (amygdala, insula, orbitomedial prefrontal cortex) were abolished in psychopaths, suggesting fundamental deficits in learning about punishing consequences (Birbaumer *et al.*, 2005; Veit *et al.*, 2002). Such insensitivity to aversive stimulation may render psychopaths relatively impervious to punishment contingencies. An outstanding question is how learned behaviors may potentiate psychopathy.

5. Reactive versus instrumental psychopathological aggression

Forensic typology makes a distinction between instrumental and reactive aggression. Instrumental aggressors use purposeful, cunning, controlled tactics e.g., intimidation or coercion of a rival, physical incapacitation or stalking (Coccaro *et al.*, 2011). Conversely, generalized rage is a hallmark of reactive aggression resulting in an impulsive act of violence without regard for consequences (Blair 2008; Raine *et al.*, 1998). Reactive aggression, uniquely typified by situationally provoked impromptu anger without antecedent deliberation, is associated with abnormal emotional regulation (Coccaro *et al.*, 2011).

So what is the neurobiological distinction? Deviations in frontal, temporal and anterior cingulate brain regions have been found in subjects who reactively aggress (Patrick 2008), suggesting relatively widespread neural deficits compared to instrumental aggressors. Neural scans of reactive murderers have demonstrated significantly lower prefrontal metabolic activity compared with controls, whereas frontal metabolism in instrumental murderers resembled controls (Brower & Price 2001). Based on convergent neuroimaging studies, instrumental

aggressors are typified by a decreased response in both amygdalar and orbitofrontal cortices when faced with emotionally evocative stimuli; a functionally 'cool brain'. In contrast, individuals who present with an increased risk for reactive, but not instrumental, aggression show increased response in both amygdalar and orbitofrontal cortices when exposed to such stimuli; a functionally 'hot brain'.

Compared with non-psychopathic prisoners, psychopathic prisoners showed less deactivation in the posterior medial cortex during externally focused tasks. These findings suggest a potential biomarker underlying key features associated with psychopathy e.g., excessive self-focus and diminished empathy. Further, posterior medial cortex dysfunction was found to relate specifically to instrumental aggressors, suggesting that a failure to inhibit this region during externally focused tasks may be specifically linked to affective/interpersonal deficits associated with criminality (Freeman *et al.*, 2015).

While deficits in amygdalar functioning were found in the brains of instrumental psychopaths, the difference in amygdalar volume was slight; statistically insignificant as compared to control group (Anckarsäter *et al.*, 2007). Enhanced amygdalar response may better characterize reactive aggression, whereas blunted amygdalar responses were found to typify psychopathic subjects prone to instrumental aggression (Coccaro *et al.*, 2011). If both frontal lobe and amygdalar dysfunction are a prequel to aggression, then we might also expect deficits in connectivity between these regions.

There are conflicting hypotheses relating psychopathy to violent aggression. Studies of conduct disorders and delinquency reveal that psychopathy confers an increased risk for both instrumental and reactive aggression (Frick *et al.*, 2003). However, other research concludes that psychopathy is the only psychiatric condition implicated in increasing the risk of instrumental aggression (Blair 2008; Patrick 2008; Porter *et al.*, 2009). Various emotional

conditions e.g., post-traumatic stress disorder, childhood bipolar disorder, etc. may be a precursor to reactive aggression (Blair *et al.*, 2005) but not necessarily instrumental aggression.

At the neuroanatomical level, reactive murderers may differ from instrumental murderers.

Should all violent criminals be treated equally? Courts must use their best discretion in giving special credence to those who display compromised neural functioning. Regardless of the etiology underlying aggression, subjects who exhibit core affective features of psychopathy may be considered distinct from other types of violent offenders. In sum, psychopathy may be necessary for the commission of violent instrumental aggression. However, psychopathy may be a sufficient, but not necessary impetus for reactive aggression.

6. Psychopathology versus antisocial personality disorder

Diagnostically, psychopathy overlaps with antisocial personality disorder (ASPD), although these related conditions may not be synonymous (de Oliveira-Souza *et al.*, 2008; Pemment 2013). Is there a demarcation between instrumental psychopathy and reactive aggression demonstrated by ASPD subjects? Corroborative studies reveal that when presented with emotionally evocative stimuli, amygdalar responses were increased for reactive but not for instrumental aggressors, suggesting that reactive aggressors may be primed to respond strongly to threatening stimuli. In contrast, other psychopathic subjects show decreased amygdalar and orbitofrontal cortex responses to this stimuli (Blair 2010). Psychopathy is thought to involve a deficit in negative emotions such anxiety and fear. Psychopaths may differ from other antisocial aggressors in subcortical brain structures that mediate basic emotional processes (Patrick 2008).

High impulsivity is a diagnostic hallmark of ASPD according to the DSM-V. ASPD was especially characterized by increased rapid-response impulsivity (Dougherty *et al.*, 2005) while aspects of impulsivity related to reward delay or attention appear relatively intact (Swann *et al.*, 2010).

The neural threat circuitry (amygdala, hypothalamus, dorsal half of periaqueductal gray) is potentiated in reactive aggression (Gregg & Siegel 2001). The greater the activity in this circuit, the greater the chance for reactive aggression. Conversely, other psychopaths appear to show deficits in this response, demonstrating impairment in tasks that rely on functional amygdala (Blair 2010).

Neurodevelopmental abnormalities in the corpus callosum have been implicated in ASPD (Pement 2013). Neural pruning of the corpus callosum occurs normally in childhood, refining neural communicative signalling in prefrontal cortices. The corpus callosum was discovered to be 23% larger in volume in subjects with ASPD than the control group. However, no such distinction was observed in psychopaths (Raine *et al.*, 2003). The prefrontal gray matter in the ASPD brain was reduced by 11% in comparison to control groups (Raine *et al.*, 2000) and this important finding was replicated in other studies (Yang *et al.*, 2010). Further, the relationship between impulsive reactive aggression and reduced frontal lobe volume has been observed in those with ASPD (Laakso *et al.*, 2002; Raine *et al.*, 2000).

Subjects with ASPD showed thinner cortices with larger surface area in various brain regions, specifically the bilateral superior frontal gyrus, orbitofrontal and triangularis, insula cortex, precuneus, middle frontal gyrus, and middle temporal gyrus (Jiang *et al.*, 2016). Such neuroarchitectural defects may account for the uncontrolled and callous behavioral characteristics that define ASPD, and these biomarkers may help characterize the pathomechanism underlying ASPD.

7. Antisocial personality disorder

High proportions of personality disorders ($\leq 30\%$) are reported by adults who have suffered TBI (van Reckum *et al.*, 1996; Grafman *et al.*, 1996) with psychiatric comorbidity ($\leq 44\%$) (Hibbard *et*

al., 1998) and heavy alcohol use (37-57%) (Kolasowsky-Hayner *et al.*, 1999) being contributing factors. A close link between ASPD and criminal behavior is affirmed by the report that 47% of male prisoners are diagnosed with ASPD (Fazel & Danesh 2002; Jiang *et al.*, 2013).

Concurrent abnormalities in both frontal and temporal lobes were found in a study of ASPD offenders (Jiang *et al.*, 2016), which may increase the risk of aggression as compared to dysfunction in each anatomical region independently (Potegal 2012). Dysfunction in both frontal and temporal regions may confer a predisposition to antisocial behavior and, specifically, hypoactivity in these anatomical regions may be linked to the commission of severe violent crimes (Anckarsäter *et al.*, 2007).

Converging lines of evidence confirm that prefrontal cortical thickness to surface area ratios are altered in ASPD brains, with direct implications for impulsivity, described as a core feature of ASPD according to the DSM-V (Dougherty *et al.*, 2005; Jiang *et al.*, 2016), specifically rapid-response impulsivity. Notably, results showed that orbitofrontal cortices extending to pars orbitalis and pars triangularis had significantly reduced thickness in ASPD brains (Ogilvie *et al.*, 2011). A recent study investigating ratio parameters found that the percentage of successfully inhibited responses was significantly lower for ASPD subjects, suggestive of an impaired response inhibition in ASPD subjects (Jiang *et al.*, 2016). While aspects of impulsivity about reward-delay or attention appear relatively intact in ASPD, substantial deficits in rapid-response impulsivity were discovered (Swann *et al.*, 2010).

Corroborative neuroimaging and neuropsychological data indicate that limbic regions are compromised in psychopathic individuals. Both the volume and gyrification of amygdala and striatal structures i.e., putamen and pallidum, were found to be reduced in violently aggressive adults and youths with conduct disorders (Blair 2008; Wallace *et al.*, 2014). Diminished amygdalar activation in response to noxious odors was discovered in subjects with ASPD as

compared to controls during aversive conditioning (Schneider *et al.*, 2000). Such disregard for unpleasant or disgusting stimuli; misperception of discomfort and personal physical harm may be accounted for by deficits specifically in the affective nucleus accumbens (Bufkin & Luttrell 2005).

8. Other neuropsychological disorders and acquired pseudopsychopathology

The insanity defense, or pleading not guilty by reason of insanity, has been used since the historic M’Naghten case (1843) to exculpate those who lack the mental capacity to know what they do, or to know that what they are doing is wrong. Since functional neuroanatomy may ultimately determine behavior, a factor in *mens rea*, the concept of the insanity defense has logically been extended to include other neuropathic disorders (Loyd v Whittley 1992). Studies reveal that nearly 66% of murderers have neurological diagnoses e.g., brain injury, intellectual disability, epilepsy, dementia, etc. (Candini *et al.*, 2017). Neuroimaging and EEG results gleaned from murderers pleading not guilty by reason of insanity and in violent psychiatric inpatients have shown substantial hypometabolism and hypoperfusion in corticolimbic regions (Critchley *et al.*, 2000; de Oliveira-Souza *et al.*, 2008; Hoptman *et al.*, 2011; Raine *et al.*, 1998).

Acquired pseudopsychopathic disorders have been linked to damage to orbitofrontal cortices (Pement 2013) and/or ventromedial or ventrolateral frontal regions via either trauma or disease. Dorsolateral prefrontal cortices may be involved with deceptive behavior and lying, inhibiting the normal ‘default’ propensity towards truth-telling (Karton & Bachmann 2011). Dorsolateral dysfunction may distinguish those having comorbid fetal or birth related brain injury, attentional disorders, substance misuse and antisocial conduct (Pennington & Ozonoff 1996). Hypoactivity in ventromedial regions can alter social moral behavior, resulting in an

inability to consider future consequences, potentiating risky behavior even when other viable options are presented (Brower & Price 2001; Bufkin & Luttrell 2005).

Ventromedial prefrontal cortices mediate physiological reactions associated with accountability for moral violations (Marazziti *et al.*, 2013). Recent focal lesions in this region can alter normative moral sensibilities (Tranel *et al.*, 2002) while the awareness of descriptive morality i.e., rules and moral conventions (Koenigs & Tranel 1994) appear to stay intact. Patients with ventromedial prefrontal cortical focal lesions showed no or little autonomic responses e.g., heart rate, skin conductance, pupillary reactivity, piloerection, sweating, etc. when presented with socially evocative stimuli (Marazziti *et al.*, 2013; Tranel 1994). These patients appear phony and can act manipulatively, demonstrating callous disregard towards their victims. In particular, approximately 50% of patients with frontotemporal lobe degeneration (FTD) present with sociopathic behaviors. FTD is a non-Alzheimer's degenerative dementia and affected neural regions are intricately connected to limbic cortices. In contrast to Alzheimer's dementia, FTD is characterized by violations of previously acquired descriptive morality and patients exhibit indifference to punitive consequences (Marazziti *et al.*, 2013; Neary *et al.*, 1998).

The expression of anger is suggested to be the single most important factor associated with violent behavior (Coid 2013) and several studies have revealed a relationship between anger, impulsivity and aggression (Birkley & Eckhardt 2015; Rubio-Garay 2016). Anger can lead to violent acts, especially when coupled with impulsivity and emotional dysregulation. These behavioral characteristics are observed in various psychopathological conditions, such as substance abuse, mood disorders, posttraumatic stress disorder and intermittent explosive disorder (Candini *et al.*, 2017). Imaginal anger is associated with enhanced activation of the left orbitofrontal cortex, right affective nucleus accumbens and bilateral anterior temporal regions (Bufkin & Luttrell 2005). Early case reports link orbitofrontal EEG spiking to violent hallucinations and assaultive behavior (Fornazzari *et al.*, 1992). Violent criminals can

misinterpret elements of ordinary situations e.g., regard a trivial slight as a threat, overreact to provocative stimuli and resultantly make poor social choices and behave inappropriately. Corticolimbic deficits may be the neurobiological substrate potentiating such emotional dysregulation.

Severe mental disorders such as schizophrenia are commonly linked to an increased risk of violent behavior accelerated with substance and/or alcohol misuse (Candini *et al.*, 2017; Iozzino *et al.*, 2017). Rates of schizophrenia with comorbid personality disorders are reported to range broadly from 4.5% to 100% (Candini *et al.*, 2017). The heterogeneity of assessment techniques, methodologies and study settings may account for this great variability (Newton-Howes *et al.*, 2008). Disentangling the correlation between personality traits and schizophrenia can be confounding since symptoms tend to overlap (Candini *et al.*, 2017). Nevertheless, a dual diagnosis of schizophrenia and personality disorder is definitively associated with increased risk of violent aggression (Bo *et al.*, 2013). Bipolar disorder also is linked to increased risk of violent behavior (Candini *et al.*, 2017; Volavka 2013). ASPD increased recidivism of violence when coupled with other mental disorders (Shepherd *et al.*, 2016) and identifying such comorbidity may be predictive of the risk of future criminality.

8.1 Traumatic Brain Injury

There is evidence at the epidemiological level that traumatic brain disorder (TBI) is linked with psychiatric disorders, criminality [105] (Timonen *et al.*, 2002) and pseudopsychopathy (Anderson & Kiehl 2014). For patients with TBI, those with impulsive aggression post-injury most often had pre-morbid antisocial behaviors (Greve *et al.*, 2001), suggesting that TBI may further disinhibit those already prone to violence. Additionally, the compound risk of incurring TBI was reported to be four-fold in a subgroup of mentally disordered males with co-existing criminality (Timonen

et al., 2002). There may be a cyclical link between TBI and crime as an epiphenomenon; underlying demographic variables that influence both TBI and criminal behavior (León-Carrión & Ramos 2003) i.e., delinquents may be prone to engage in risky lifestyle choices that predispose them to incurring a TBI.

Cumulative findings suggest that practitioners working with mentally ill individuals who have experienced a TBI should be proactively aware of increased risk for delinquency and violence. Because the temporal lobes are anatomically situated in direct contact with the floor of the skull, they are especially prone to traumatic injury. Such contusions may result from movement of the brain inside the skull when struck directly by an object or from arrested forward motion of the head, producing a counter-coup injury (Diaz 1995; Elliot 1982). Prompt and comprehensive medical treatment of TBI may allay the occurrence of violent crime (Sarapata *et al.*, 1998).

8.2 Intellectual disability

Common characteristics of those with intellectual disability (ID) place them at greater likelihood of contact with the criminal justice system (Olley 2013), particularly risking both victimization and perpetuation of violent and/or sexual offenses (Nixon 2017). Those with ID may appear in court for various reasons, both civil e.g., guardianship or custodial matters, services, etc., and criminal, which vary widely in gravity from minor offenses e.g., shoplifting or disruptive behavior, to felonies. In such cases, the fallacious argument has been posited that an ID diagnosis requires showing that the defendant's deficits were caused by low intelligence. However, such a causal link is not required by either the APA or the American Association on Intellectual and Development Disabilities (Olley 2013). Knowledgeable agency may not be

equivalent to *ipso facto* intention, and a legal gray zone emerges when intellectual or cognitive deficits are subtle.

Compared to typically developing peers, those with ID may not progress through developmental stages of moral reasoning as quickly and sophisticated stages may be unattainable. Moral development is associated with cognitive abilities such as abstract reasoning, planning and decentration, etc. that are invariably impacted by ID. Moral judgment in those with ID is suggested to be curvilinear (Langdon *et al.*, 2010). That is, the lowest level of moral judgment may be protective against criminality, conferring unquestioning obedience of authorities and the law. But for those with borderline ID, reasons for moral decisions are dominated by gratification of their own immediate needs, placing them at great risk for delinquent behavior (Van Vugt *et al.*, 2011).

The commission of a violent act without competent awareness by the perpetrator is considered exempt from punishment as there is no recognizable guilty intent. More stringent punishments are mandated for those who violate within a state of knowledge, compared with merely a state of recklessness.

9. Intentionality and *mens rea*

Criminal conviction is contingent upon evidence beyond a reasonable doubt that commission of a prohibited act, *actus reus*, coincides with a statutorily defined mental state, *mens rea*. Existing research suggests that law enforcement officials and jurors have trouble distinguishing between the motives of defendants who violate under reckless pretenses from those who act with a knowledgeable state of intentionality, and ensuing discrepancies can generate a great deal of doubt and dispute. We judge intentional attempts to cause harm to someone, even if the attempt fails, with more blameworthy gravity than harm inflicted inadvertently or recklessly

(Gan *et al.*, 2016). Ascribing just and equitable treatment of probative mental states implies two directional challenges: conceptual and inferential. Concepts of mental states can effectively assign valence to the defendant's responsibility, culpability and punishment. Comparatively, inferential evaluations try to make logical inferences of mental states from behavior and circumstantial evidence. And making such inferences with a reliable degree of accuracy remains a formidable task. Within the law, the concept of intentionality has many synonyms, referred to as voluntarily, purposely, knowingly or willfully, etc. However, in practice, these all mean the same in legal proceedings.

The brain is posited to have an innate 'moral grammar' (Mikhail 2007). Evidence gleaned from neuroimaging studies suggest there is a neuroanatomical substrate to moral agency (Mendez 2009) and such evaluations using neuroscientific technology can determine on which side of the legally defined boundary a defendant's mental state lies. Structures at the bilateral superior temporal-parietal junction are associated with moral decisions (Vilares *et al.*, 2017). The ventromedial prefrontal cortex, particularly the right hemisphere, may confer moral cognition supported by input from the adjacent orbitofrontal, ventrolateral, dorsolateral cortices and the amygdala. Disorders to the right ventromedial prefrontal cortex e.g., focal lesions or frontotemporal dementia, disrupt moral emotions and decision making processes (Greene *et al.*, 2004). fMRI studies found that activation of the right ventromedial prefrontal cortex coincides with tasks requiring explicit moral judgments and empathy (Greene *et al.*, 2004; Mendez 2009).

Where is the statutorily defined demarcation between knowledgeable intentionality and recklessness? The dorsomedial prefrontal cortex was found to be more involved in knowledgeable mental states than in recklessness (Vilares *et al.*, 2012). Research investigating whether attributes of intentionality are associated with different brain regions than recklessness found that the anterior insula was most predictive of knowledgeable states of intentionality (Vilares *et al.*, 2017). (Anatomically, the insular cortex lies deep within the folds of the lateral

sulci, richly bi-directionally innervated with limbic and frontal regions). This finding is congruent with previous experiments implicating the role of the anterior insula with representations of risk and uncertainty (Singer *et al.*, 2009; Vilares *et al.*, 2012) and previous studies suggest that this region plays a predominant role in uncertainty stochastic of innervation of the reward circuit (Preuschoff *et al.*, 2006) i.e., mesolimbic structures comprised of the ventral tegmental area, nucleus accumbens, ventral striatum and basal ganglia. Recent imaging experiments found there are communication differences between these mesolimbic structures associated with reward and motivation in both non-criminals and criminals with psychopathic traits (Geurts *et al.*, 2016). In sum, psychopathic brains are highly attenuated to the expectation of reward. An especially strong focus on reward coupled with impulsivity may be linked to the tendency to offend.

The ability to integrate intention with the outcome of a given action is a critical crux of sophisticated moral judgment. While moral emotions such as empathy are assumed to be lacking upon commission of an offense, immature moral judgment specifically has been shown to be most strongly related to delinquent behavior (Van Vugt *et al.*, 2011). Investigating the time course of integration between antecedent intention and the certainty of the resultant outcome may reveal processing stages in moral judgment, and findings suggest a temporal sequence of neural activation. Recent studies using event-related potentials indicate that the right temporal-parietal junction appears to be especially active during both initial and late moral integration processing (Gan *et al.*, 2016).

Based on corroborative clinical experience, researchers report that psychopaths can have at least normal intelligence, and, somewhat counterintuitively, even a normal capacity to make moral judgments. Rather, their actual behavior reflects volitional amorality (Tassy *et al.*, 2013). Such observations might explain why some studies report that psychopathic subjects acknowledge descriptive and normative morality, demonstrating the ability to make moral

judgments, yet fail to act in accordance with them, illustrating a 'moral hypocrisy' (Batson *et al.*, 1997).

10. Forensic implications

Provocative philosophical questions arise if we consider that our prefrontal cortex might serve as a filter to mask inherently basal violent aggression. Because violent criminals may lack the aptitude for accountability and insight into the breadth of their behavioral deficits, conventional rehabilitative measures may be rendered moot. What are the implications for criminal law?

Consideration for a suspect's mental state is requisite to fair legal justice. After all, an act of violence committed by a rampaging wild animal, sleepwalker or an individual in a profoundly psychotic state, for instance, is considered exempt from punishment as there is no recognizable guilty intent. A legal gray zone emerges when expert testimony presents less overt forensic evidence of a violent criminal's compromised neurology. The intercalated framework of neurolaw uniquely offers great power to describe and clarify criminological factors.

The scientific method involves iteratively testing hypotheses using various techniques, and this systematic approach can be at odds with how the legal system operates. Legal processes are concerned with the doctrine of precedent, *stare decisis*, and judges need to make definitive decisions based on conclusive evidence. To define the scope of neurolaw, as per the federal rule of evidence, expert testimony needs only to be helpful, relevant and reliable to the trier and impactful to jurors. As per the Daubert ruling (1993), the United States Supreme Court stipulates that the trial judge's own discretion determines whether to admit expert evidence into their court. If a scientific expert's testimony is grounded in scientific methodology, then their knowledgeable expertise is deemed admissible, in accordance with Daubert's Rule 702 (Dixon & Gill 2001). Organizing and centralizing compiled neuroimaging data could streamline

efficiency for investigative processes. Since functional and structural neuroimaging has become more commonplace, its role as statutorily viable evidence are valuable in detecting regional brain dysfunction that can otherwise elude conventional psychological assessment.

Centralized biometrics might be a means to reduce rates of recidivism (Riley 2005) as violent offenses, and individual mitigating circumstances, can be better tracked and retrieved to optimize investigative profiling. Analogously, forensic deoxyribonucleic acid analysis (DNA) is routinely used to solve modern crimes. In North America, data compiled by the Canadian National DNA Data Bank and the United States' Combined DNA Index System have proved to be an invaluable resource for investigators. For physical evidentiary samples such as latent prints, the Integrated Automated Fingerprint Identification System and Western Identification Network are well-established databases of identified known prints. By extension, establishing an integrated forensic neuroimaging database may emerge as a game-changer in terms of how violent crime investigations are conducted.

Should all violent criminals be treated equally? Cumulative psychiatric and neuroimaging findings imply that neuroarchitectural deficits associated with psychopathy hijack the development of such morality (Anderson & Kiehl 2014), so conventional punishment contingencies may be effectively moot. Therapeutic law encourages apology for *malum in se* crimes by granting broader protection from admissibility in liability determinations, plus affording restoration for the victim (Shuman 2000). Sincere apology hold substantive psychological and societal meaning, necessitating acknowledgment of the grievous consequence of the offending act and attributing culpability with expression of remorse. Neuropsychiatric researchers found that psychopathic subjects were unable to make sincere apologies for their offenses, representing a fundamental gap in moral understanding (Ayob & Thornton 2014). Further findings report that psychopathic inmates have the highest rates of recidivism compared to peer inmates without psychopathy (Seto & Barbaree 1999).

Based on cumulative evidence presented in this meta-analysis, psychopathic criminals are refractory to change relative to violent criminals without psychopathy and consequently need to be regarded separately from other violent offenders. On the upside, if psychopathy is ruled out for other aggressors, then therapeutic services and resources, which are often limited, may be better allocated for their rehabilitation. Therapeutic techniques to enhance neuroplasticity emerge as a potential direction. Advances in interventions at the genomic level, developmentally and/or epidemiologically, are worthy areas of research. Neuroscience is beginning to address novel perspectives regarding violent criminal behavior that will invariably impact future legal processes.

11. Conclusion

This meta-analysis highlights how the functional somatotopy of prefrontal and corticolimbic regions can be forensically assessed to characterize violent criminal behavior in legal settings. Neuroscientific evidence can be used to critically arbitrate criminal cases, even determining sentencing outcomes and assessing the feasibility of rehabilitative measures based on neuroimaging results in conjunction with behavioral profiles. This meta-analysis distinguishes between instrumental and reactive aggression at the neuroanatomical level, comparing and contrasting differences with other neuropsychological disorders and pseudo-psychopathy. Criminal intentionality is explored within the context of neurolaw. Psychopathic criminals are relatively refractory to rehabilitative treatment compared to non-psychopathic criminals, underscoring the need for alternate options for the psychopathic population. This meta-analysis proposes a centralized neuroimaging database to streamline biometric information to optimize legal investigation, with respect to privacy and bioethical constraints. The need for advances in individualized techniques to treat criminal aggression emerge as an important future research directive.

Acknowledgement

Many thanks to Dr John Riley, Professor of Criminology at University of Alaska Anchorage, Department of Sociology, for sharing his perspective.

References

- Anckarsäter, H., Piechnik, S., Tullberg, M., Ziegelitz, D., Sörman, M., Bjellvi, J., et al., 2007. Persistent regional frontotemporal hypoactivity in violent offenders at follow-up. *Psychiatry Res.* 156(1), 87-90.
- Anderson, N.E., Kiehl, K.A., 2014. Psychopathy: Developmental perspectives and their implications for treatment. *Res Neurol Neurosci.* 32(1), 103-117.
- Ayob, G., Thornton, T., 2014. Psychopathy: what apology making tells us about moral agency. *Theor Med Bioeth.* 35, 17-29.
- Batson, C.D., Kobryniewicz, D., Dinnerstein, J.L., Kampf, H.C., Wilson, A.D., 1997. In a very different voice: Unmasking moral hypocrisy. *J Pers Soc Psychol.* 72(6), 1335-1348.
- Birbaumer, N., Veit, R., Lotze, M., Erb, M., Hermann, C., Grodd, W., et al., 2005. Deficient fear conditioning in psychopathy: A functional magnetic resonance imaging study. *Arch Gen Psychiatry.* 62, 799-805.
- Birkley, E.L., Eckhardt, C.I., 2015. Anger, hostility, internalizing negative emotions, and intimate partner violence perpetration: A meta-analytic review. *Clin Psychol Rev.* 37, 40-56.
- Blair, R.J.R., 2004. The roles of orbital frontal cortex in the modulation of antisocial behavior. *Brain Cognition.* 55(1), 198-208.
- Blair, R.J.R., 2008. The amygdala and ventromedial prefrontal cortex: functional contributions and dysfunction in psychopathy. *Philos Trans R Soc Lond B Biol Sci.* 363, 2557-2565.
- Blair, R.J.R., 2010. Neuroimaging of psychopathy and antisocial behavior: A targeted review. *Curr Psych Rep.* 12(1), 76-82.
- Blair, R.J.R., Mitchell, D.G.V., Blair, K.S., 2005. *The psychopath: emotion and the brain.* Blackwell Ed. Oxford UK.
- Bo, S., Abu-Akel, A., Kongerslev, M., Haahr, U.H., Simonsen, E., 2013. The role of co-morbid personality pathology in predicting self-reported aggression in patients with schizophrenia. *Comp Psychiatry.* 54, 423-431.
- Brower, M.C., Price, B.H., 2001. Neuropsychiatry of frontal lobe dysfunction in violent and criminal behaviour: a critical review. *J Neurol Neurosurg Psychiatry.* 71, 720-726.
- Budhani, S., Blair, R.J.R., 2005. Response reversal and children with psychopathic tendencies: success is a function of salience of contingency change. *J Child Psychol Psychiatry.* 46, 972-981.
- Budhani, S., Richell, R., Blair, R.J.R., 2006. Impaired reversal but intact acquisition: Probabilistic response reversal deficits in adult individuals with psychopathy. *J Abnorm Psychol.* 115, 552-558.
- Bufkin, J., Luttrell, V., 2005. Neuroimaging studies of aggressive and violent behavior: Current findings and implications for criminology and criminal defense. *Trauma, Violence Abus.* 6, 176-191.

- Candini, V., Ghisi, M., Bottesi, G., Ferrari, C., Bulgari, V., Iozzino, L., et al., 2017. Personality, Schizophrenia, and Violence: A Longitudinal Study. *J Pers Disord.* 1-17.
- Cleckley, H., 1941. *The mask of sanity.* St. Louis, MO. Mosby.
- Coccaro, E.F., Sripada, C.S., Yanowitch, R.N., Phan, L., 2011. Corticolimbic function in impulsive aggressive behavior. *Bio Psych.* 69(12), 1153-1159.
- Coid, J.W., Ullrich, S., Kallis, C., Keers, R., Barker, D., et al., 2013. The relationship between delusions and violence: Findings from the east London first episode psychosis study. *JAMA Psychiatry.* 70(5), 465-471.
- Contreras-Rodríguez, O., Pujol, J., Harrison, B.J., Soriano-Mas, C., Deus, J., López-Solà, M., et al., 2015. Functional Connectivity Bias in the Prefrontal Cortex of Psychopaths. *Biological Psychiatry.* 78, 647-655.
- Cornell, D., Warren, J., Hawk, G., Stafford, E., Oram, G., Pine, D., 1996. Psychopathy in instrumental and reactive violent offenders. *J Consult Clin Psych.* 64, 783-790.
- Craig, M.C., Catani, M., Deeley, Q., Latham, R., Daly, E., Kanaan, R., et al., 2009. Altered connections on the road to psychopathy. *Mol Psychiatry.* 14, 946.
- Critchley, H.D., Simmons, A., Daly, E.M., Russell, A., van Amelsvoort, T., Robertson, D.M., et al., 2000. Prefrontal and medial temporal correlates of repetitive violence to self and others. *Biol Psychiatry.* 47(10), 928-934.
- de Oliveira-Souza, R., Hare, R.D., Bramati, I.E., Garrido, G.J., Azevedo Ignácio, F., Tovar-Moll, F., et al., 2008. Psychopathy as a disorder of the moral brain: Fronto-temporo-limbic grey matter reductions demonstrated by voxel-based morphometry. *NeuroImage.* 40, 1202-1213.
- Del Casale, A., Kotzalidis, G.D., Rapinesi, C., Di Pietro, S., Alessi, M.C., et al., 2015. Functional Neuroimaging in Psychopathy. *Neuropsychobiol.* 72, 97-117.
- Diaz, F.G., 1995. Traumatic brain injury and criminal behaviour. *Med Law.* 14, 131-140.
- Dixon, L., Gill, B., 2001. Changes in the standards for admitting expert evidence in federal civil cases since the Daubert decision. RAND Institute for Civil Justice.
- Dougherty, D.M., Mathias, C.W., Marsh, D.M., 2005. Laboratory behavioral measures of impulsivity. *Behav Res Methods.* 37, 82-90.
- Duffy, J., Campbell III, J., 1994. The regional prefrontal syndromes: a theoretical and clinical overview. *Clin Neurosci.* 6, 379-387.
- Eagleman, D., 2011. *Incognito: The Secret Lives of the Brain.*
- Elliot, F., 1982. Findings in adult minimal brain dysfunction and the dyscontrol syndrome. *J Nerv Ment Dis.* 170, 680-687.
- Farahany, N.A., 2016. Neuroscience and behavioral genetics in US criminal law: an empirical analysis. *J Law Biosci.* 2(3), 485-509.
- Fazel, S., Danesh, J., 2002. Serious mental disorder in 23,000 prisoners: a systematic review of 62 surveys. *Lancet.* 359, 545-550.
- Fornazzari, L., Farcnik, K., Smith, I., Heasman, G., Ichise, M., 1992. Violent visual hallucinations and aggression in frontal lobe dysfunction: clinical manifestations of deep orbitofrontal foci. *J Neuropsychiatry Clin Neurosci.* 4, 42-44.

- Freeman, S.M., Clewett, D.V., Bennett, C.M., Kiehl, K.A., Gazzaniga, M.S., Miller, M.B., 2015. The posteromedial region of the default mode network shows attenuated task-induced deactivation in psychopathic prisoner. *Neuropsychol.* 29, 493-500.
- Frick, P., Cornell, A., Barry, C., Bodin, D., Dane. H., 2003. Callous-Unemotional Traits and Conduct Problems in the Prediction of Conduct Problem Severity, Aggression, and Self-Report of Delinquency. *J Abnorm Child Psychol.* 31(4), 457-470.
- Gan, T., Lu, X., Li, W., Gui, D., Tang, H., Mai, X., et al., 2016. Temporal dynamics of the integration of intention and outcome in harmful and helpful moral judgment. *Front Psychol.* 6, 2022.
- Geurts, D.E.M., von Borries, K., Volman, I., Bulten, B.H., Cools, R., Verkes, R.J., 2016. Neural connectivity during reward expectation dissociates psychopathic criminals from non-criminal individuals with high impulsive/antisocial psychopathic traits. *Soc Cogn Affect Neurosci.* 11(8), 1326-1334.
- Ghashghaei, H.T., Barbas, H., 2002. Pathways for emotion: interactions of prefrontal and anterior temporal pathways in the amygdala of the rhesus monkey. *Neuroscience.* 115(4), 1261-1279.
- Ghashghaei, H.T., Hilgetag, C.C., Barbas, H., 2007. Sequence of information processing for emotions based on the anatomic dialogue between prefrontal cortex and amygdala. *Neuroimage.* 34(3), 905-923.
- Grabenhorst, F., Rolls, E.T., 2011. Value, pleasure and choice in the ventral prefrontal cortex. *Cogn Sci.* 15, 56-67.
- Grafman, J., Schwab, K., Warden, D., Pridgen, A., Brown, H., Salazar, A., 1996. Frontal lobe injuries, violence, and aggression: a report of the Vietnam Head Injury Study. *Neurology.* 46, 1231-1238.
- Greely, H.T., 2013. Mind reading, neuroscience, and the law. doi:10.1093/acprof:oso/9780199859177.003.0005
- Greene, J.D., Nystrom, L.E., Engell, A.D., Darley, J.M., Cohen, J.D., 2004. The neural bases of cognitive conflict and control in moral judgment. *Neuron.* 44, 389-400.
- Gregg, T.R., Siegel, A., 2001. Brain structures and neurotransmitters regulating aggression in cats: implications for human aggression. *Prog Neuro-Psychopharmacol Biol Psychiatry.* 25(1), 91-140.
- Greve, K., Sherwin, L., Stanford, M., Mathias, C., Love, J., Ramzinski, P., 2001. Personality and neurocognitive correlates of impulsive aggression in long-term survivors of severe traumatic brain injury. *Brain Injury.* 15, 255-262.
- Haber, S.N., Behrens, T., 2014. The Neural Network Underlying Incentive-Based Learning: Implications for Interpreting Circuit Disruptions in Psychiatric Disorders. *Neuron.* 83, 1019-1039.
- Hare, R., 1996. Psychopathy: a clinical Construct whose time has come. *Crim Justice Behav.* 23(1), 25-54.
- Hare, R., 1999. Psychopathy as a Risk Factor for Violence. *Psychiatric Quarterly.* 70(3), 181-197.
- Hare, R., 2003. Psychopathy Checklist Revised (PCL-R): Technical manual.
- Harenski, C.L., Edwards, B.G., Harenski, K.A., Kiehl, K.A., Barbey, A.K., Decety, J., et al., 2014. Neural correlates of moral and non-moral emotion in female psychopathy. *Front Hum Neurosci.* doi.org/10.3389/fnhum.2014.00741

- Harenski, C.L., Harenski, K.A., Kiehl, K.A., 2014. Neural processing of moral violations among incarcerated adolescents with psychopathic traits. *Dev Cogn Neurosci.* 10, 181-189.
- Harris, G., Rice, M., 1991. Psychopathy and violent recidivism. *Law Human Behav.* 15, 625-637.
- Hibbard, M., Uysal, S., Kepler, K., Bogdany, J., Silver, J., 1998. Axis I psychopathology in individuals with traumatic brain injury. *J Head Trauma Rehabil.* 13, 24-39.
- Hoptman, M.J., Antonius, D., Kline, N., 2011. Neuroimaging correlates of aggression in schizophrenia: an update HHS Public Access. *Curr Opin Psychiatry.* 24, 100-106.
- Iozzino, L., Ferrari, C., Large, M., Nielsens, O., de Girolamo, G., 2017. Prevalence and risk factors of violence by psychiatric acute inpatients: A systematic review and metaanalysis. *PLoS one.* doi.org/10.1371/journal.pone.0128536.
- Jiang, W., Li, G., Liu, H., Shi, F., Wang, T., Shen, C., et al., 2016. Reduced cortical thickness and increased surface area in antisocial personality disorder. *Neuroscience.* 337, 143-152.
- Jiang, W., Liu, H., Liao, J., Ma, X., Rong, P., Tang, Y., et al., 2013. A functional MRI study of deception among offenders with antisocial personality disorders. *Neuroscience.* 244, 90-98.
- Karton, I., Bachmann, T., 2011. Effect of prefrontal transcranial magnetic stimulation on spontaneous truth-telling. *Behav Brain Res.* 225, 209-214.
- Kelkar, K., 2016. Can a brain scan uncover your morals? British Neuroscience Association.
- Kiehl, K, Hoffman, M., 2014. The criminal psychopath: history, neuroscience, treatment, and economics. *Jurimetrics.* 51, 355-397.
- Koenigs, M., 2012. The role of prefrontal cortex in psychopathy. *Rev Neurosci.* 23(3).
- Koenigs, M., Tranel, D., 2007. Irrational Economic Decision-Making after Ventromedial Prefrontal Damage: Evidence from the Ultimatum Game. *J Neurosci.* 27(4), 951-956.
- Kolalowsky-Hayner, S., Gourley III, E., Kreutzer, J., Marwitz, J., Cifu, D., McKinely, W, 1999. Pre-injury and substance abuse among persons with brain injury and persons with spinal cord injury. *Brain Injury.* 13, 571-581.
- Kumarasamy, J., 2014. Neurolaw: The Potential and Pitfalls of a Newly Developing Field. *McGill Journal of Law and Health.*
- Laakso, M.P., Gunning-Dixon, F., Vaurio, O., Repo-Tiihonen, E., Soininen, H., Tiihonen, J., 2002. Prefrontal volumes in habitually violent subjects with antisocial personality disorder and type 2 alcoholism. *Psychiatry Res Neuroim.* 114, 95-102.
- Langdon, P.E., Clare, I.C.H., Murphy, G.H., 2010. Developing an understanding of the literature relating to the moral development of people with intellectual disabilities. *Dev Rev.* 30, 273-293.
- León-Carrión, J., Ramos, F., 2003. Blows to the head during development can predispose to violent criminal behaviour: rehabilitation of consequences of head injury is a measure for crime prevention. *Brain Injury.* 17(3), 207-216.
- Loyd v Whittle, 5th Cir., 1992. 977 F2d:149.
- Ly, M., Motzkin, J.C., Philippi, C.L., Kirk, G.R., Newman, J.P., Kiehl, K.A., et al., 2012. Cortical Thinning in Psychopathy. *Am J Psychiatry.* 169(7), 743-749.

- Lynam, D.R., Gaughan, E., Miller, J.D., Miller, D., Mullins-Sweat, S., Widiger, T.A., 2011. Assessing the basic traits associated with psychopathy: Development and validation of the Elemental Psychopathy Assessment. *Psychological Assessment*. 23, 108-124.
- Marazziti, D., Baroni, S., Landi, P., Ceresoli, D., Dell'Osso, L., 2013. The neurobiology of moral sense: facts or hypotheses? *Ann Gen Psychiatry*. 12(1), 6.
- Marsh, A., Finger, E., Mitchelle, D., Reid, M., Sims, C., Kosson, D., et al., 2008. Reduced Amygdala Response to Fearful Expressions in Children and Adolescents With Callous-Unemotional Traits and Disruptive Behavior Disorders. *Am J Psychiatry*. 165(5),166-712.
- Mendez, M.F., 2009. The neurobiology of moral behavior: review and neuropsychiatric implications. *CNS Spectr*. 14, 608-620.
- Mikhail, J., 2007. Universal Moral Grammar: Theory, Evidence, and the Future. *Trends Cogn Sci*.11(4), 143-152.
- Montague, P.R., Berns, G.S., 2002. Neural economics and the biological substrates of valuation. *Neuron*. 36, 265-284.
- Neary, D., Snowden, J.S., Gustafson, L., Passant, U., Stuss, D., Black, S., et al., 1998. Frontotemporal lobar degeneration. *Neurology*. 51, 1546-1554.
- Nelson, R.J., Trainor, B.C., 2007. Neural mechanisms of aggression. *Nat Rev Neurosci*. 8, 536-546.
- Newton-Howes, G., Tyrer, P., North, B., Yang, M., 2008. The prevalence of personality disorder in schizophrenia and psychotic disorders: systematic review of rates and explanatory modelling. *Psychol Med*. 38, 1075-1082.
- Nixon, M., 2017. Estimating the risk of crime & victimization in people with intellectual disability: a data-linkage study. *Soc Psychiatry Psychiatr Epidemiol*. 52, 617-626.
- O'Doherty, J.P., 2011. Contributions of the ventromedial prefrontal cortex to goal-directed action selection. *Ann N Y Acad Sci*. 1239, 118-1129.
- Ogilvie, J.M., Stewart, A.L., Chan, R.C.K., Shum, D.H.K., 2011. Neuropsychological measures of executive function and antisocial behavior: a meta-analysis. *Criminol*. 49(4), 1063-1107.
- Olley, J.G., 2013. Definition of Intellectual Disability in Criminal Court Cases. *Intellect Dev Disabil*. 51, 117-121.
- Pardo, M.S., 2013. *Minds, Brains, and Law: The Conceptual Foundations of Law and Neuroscience*.
- Patrick, C.J., 2008. Psychophysiological correlates of aggression and violence: an integrative review. *Phil Trans R Soc B*. 363, 2543-2555.
- Patrick, C.J., Fowles, D.C., Krueger, R.F., 2009. Triarchic conceptualization of psychopathy: Developmental origins of disinhibition, boldness, and meanness. *Dev Psychopathol*. 21, 913-938.
- Pemment, J., 2013. The neurobiology of antisocial personality disorder: The quest for rehabilitation and treatment. *Aggression Viol Behav*. 18(1), 79-82.
- Pennington, B., Ozonoff, S., 1996. Executive functions and developmental psychopathology. *J Child Psychol Psychiatry*. 37, 51-87.
- Pera-Guardiola, V., Contreras-Rodríguez, O., Batalla, I., Kosson, D., Menchón, J.M., Pifarré, J., et al., 2016. Brain Structural Correlates of Emotion Recognition in Psychopaths. *PLoS one*. 11.

- Porter, S., Brinke, L., Wilson, K., 2009. Crime Profiles and conditional release performance of psychopathic and non-psychopathic sexual offenders. *Legal Criminol Psych.* 14, 109-111.
- Potegal, M., 2012. Temporal and frontal lobe initiation and regulation of the top-down escalation of anger and aggression. *Behav Brain Res.* 231(2), 386-395.
- Presidential commission for the study of bioethical issues, March 2015. *Gray Matters: Topics at the Intersection of Neuroscience, Ethics, and Society.*
- Preuschoff, K., Bossaerts, P., Quartz, S.R., 2006. Neural Differentiation of Expected Reward and Risk in Human Subcortical Structures. *Neuron.* 51, 381-390.
- Price, J.L., 2003. Comparative Aspects of Amygdala Connectivity. *Ann Rev NY Acad Sci.* 985, 50-58.
- Qin, P., Northoff, G., 2011. How is our self related to midline regions and the default-mode network? *Neuroimage.* 57, 1221-1233.
- Raine, A., Buchsbaum, M., Lacasse, L., 1997. Brain abnormalities in murderers indicated by positron emission tomography. *Biological Psychiatry.* 42, 495-508.
- Raine, A., Ishikawa, S.S., Arce, E., Lencz, T., Knuth, K.H., Bihrlle, S., et al., 2004. Hippocampal structural asymmetry in unsuccessful psychopaths. *Biol Psychiatry.* 55(2), 185-191.
- Raine, A., Lencz, T., Bihrlle, S., LaCasse, L., Colletti, P., 2000. Reduced prefrontal gray matter volume and reduced autonomic activity in antisocial personality disorder. *Arch Gen Psychiatry.* 57(2), 119-127.
- Raine, A., Lencz, T., Taylor, K., Hellige, J.B., Bihrie, S. et al., 2003. Corpus callosum abnormalities in psychopathic antisocial individuals. *Arch Gen Psychiatry.* 60(11), 1134-1142.
- Raine, A., Meloy, J.R., Bihrlle, S., Stoddard, J., LaCasse, L., Buchsbaum, M.S., 1998. Reduced prefrontal and increased subcortical brain functioning assessed using positron emission tomography in predatory and affective murderers. *Behav Sci Law.* 16(3), 319-332.
- Riley J., 2005. Review essay: But they all come back. *Alaska Justice Forum.* 22.
- Rubio-Garay, F., Carrasco, M.A., Amor, P.J., 2016. Aggression, anger and hostility: Evaluation of moral disengagement as a mediational process. *SJoP.* 57(2), 129-135.
- Sarapata, M., Hermann, D., Johnson, T., Aycocock, R., 1998. The role of head injury in cognitive functioning, emotional adjustment and criminal behaviour. *Brain Injury.* 12, 821-842.
- Schneider, F., Habel, U., Kessler, C., Posse, S., Grodd, W., Müller-Gärtner, H.W., 2000. Functional imaging of conditioned aversive emotional responses in antisocial personality disorder. *Neuropsychobiol.* 42, 192-201.
- Schoenbaum, G., Roesch, M., 2005. Orbitofrontal cortex, associative learning, and expectancies. *Neuron.* 47, 633-636.
- Seto, M.C., Barbaree, H.E., 1999. Psychopathy, Treatment Behavior, and Sex Offender Recidivism. *J Interpers Viol.* 14, 1235-1248.
- Shackman, A.J., Salomon, T.V., Slagter, H.A., Fox, A.S., Winter, J.J., Davidson, R.J., 2011. The integration of negative affect, pain and cognitive control in the cingulate cortex. *Nat Rev Neurosci.* 12, 154-167.

- Shepherd, S.M., Campbell, R.E., Ogloff, J.R.P., 2016. Psychopathy, Antisocial Personality Disorder, and Reconviction in an Australian Sample of Forensic Patients. *Int J Offender Ther Comp Criminol.* 62(3), 609-628.
- Shuman, D.W., 2000. The Role of Apology in Tort Law. *Judicature.* 83(4), 180-189.
- Singer, T., Critchley, H.D., Preuschoff, K., 2009. A common role of insula in feelings, empathy and uncertainty. *Trends Cogn Sci.* 13, 334-340.
- Swann, A.C., Lijffijt, M., Lane, S., Steinberg, J., Moeller, F., 2010. Trait Impulsivity and Response Inhibition in Antisocial Personality Disorder. *J Psychiatr Res.* 43, 1057-1063.
- Tassy, S., Deruelle, C., Mancini, J., Leistedt, S., Wicker, B., 2013. High levels of psychopathic traits alters moral choice but not moral judgment. *Front Hum Neurosci.* doi: 10.3389/fnhum.2013.00229
- Timonen, M., Miettunen, J., Hakko, H., Zitting, P., Veijola, J., von Wendt, L., et al., 2002. The association of preceding traumatic brain injury with mental disorders, alcoholism and criminality: the Northern Finland 1966 Birth Cohort Study. *Psychiatry Res.* 113, 217-226.
- Tranel, D., 1994. Acquired sociopathy: the development of sociopathic behavior following focal brain damage. *Prog Exp Pers Psychopathol Res.* 285-311.
- Tranel, D., Bechara, A., Denburg, N.L., 2002. Asymmetric Functional Roles of Right and Left Ventromedial Prefrontal Cortices in Social Conduct, Decision-Making, and Emotional Processing. *Cortex.* 38, 589-612.
- van Reckum, R., Bolago, I., Finlayson, M., Garner, S., Links, P., 1996. Psychiatric disorders after traumatic brain injury. *Brain injury.* 10, 319-327.
- Van Vugt, E., Gibbs, J., Stams, G.J., Bijleveld, C., Hendriks, J., van der Laan, P., 2011. Moral Development and Recidivism: A Meta-Analysis. *Int J Offender Ther Comp Criminology.* 55(8), 1234-1250.
- Veit, R., Flor, H., Erb, M., Hermann, C., Lotze, M., Grodd, W., 2002. Brain circuits involved in emotional learning in antisocial behavior and social phobia in humans. *Neurosci Lett.* 28, 233-236.
- Vilares, I., Howard, J.D., Fernandes, H.L., Gottfried, J.A., Kording, K.P., 2012. Differential Representations of Prior and Likelihood Uncertainty in the Human Brain. *Current Biol.* 22, 1641-1648.
- Vilares, I., Wesley, M., Ahn, W.Y., Bonnie, R., Hoffman, M., Jones, O.D., et al., 2017. Predicting the knowledge-recklessness distinction in the human brain. *PNAS.* 114(12), 3222-3227.
- Volavka, J., 2013. Violence in schizophrenia and bipolar disorder. *Psychiatria Danubina.* 25:24-33.
- Wallace, G.L., White, S., Robustelli, B., Sinclair, S., Hwang, S., Martin, A., et al., 2014. Cortical and Subcortical Abnormalities in Youths with Conduct Disorder and Elevated Callous-Unemotional Traits. *J Am Acad Child Adolesc Psychiatry.* 53(4), 456-465.
- Yang, Y., Raine, A., Colletti, P., Toga, A.W., Narr, K.L., 2010. Morphological alterations in the prefrontal cortex and the amygdala in unsuccessful psychopaths. *J Abnormal Psychol.* 119(3), 546-554.