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# Aggression and Violent Behavior



## Neuropsychological and neurological correlates in violent and homicidal offenders: A legal and neuroscience perspective

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### ABSTRACT

Violence and murder have their roots in biological, psychological, and sociological factors. This article will focus on one specific element of the biological aspects of violence and murder; specifically; neurological and neuropsychological aspects. The author will provide a literature review contrasting structural brain abnormalities and dysfunction (neuropathology) and brain–behavior (neuropsychological) relational attributes to violence, aggression, and homicidal behavior in particular. After reviewing the literature, the author will address how these brain related structural and functional correlates to violence are utilized in court proceedings. Specifically, the article questions how expert witnesses can integrate neurological and especially neuropsychological data to address psycholegal issues, such as mitigation, freewill, and moral culpability, especially within death penalty and murder cases. The author provides recommendations for the practicing forensic neuropsychologist evaluating homicide cases.

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## 1. Biopsychosocial risk factors and violence

The origins of criminal violent and homicidal behavior have their etiological lineage within a biopsychosocial framework (Brennan & Raine, 1997; Buikhuizen, 1988; Eysenck, 1964; Fabian, 2009; Giancola, 1995; Moffitt, 1993a,b). Violence and murder stem from a multitude of factors including social learning (Akers, 1985; Bandura, 1973), frustration-induced violence (Berkowitz, 1962), cognitive and moral development (Arbuthnot, Gordon, & Jurkovic, 1987; Chandler & Moran, 1990; Raine, 1993), mental and neurological disorder (Raine, 2002a,b; Pincus & Tucker, 2003; Swanson et al., 1990), environmental factors; such as socioeconomic variables (Durant, Cadenhead, Pendergrast, Slavens, & Linder, 1994), violent victimization (Patchin et al., 2006), family dynamics and dysfunction (Haapasalo & Pokela, 1999; McCord, 1991; Widom, 1989), genetics (Hutchings & Mednick, 1975), and substance abuse (White, Loeber, Stouthamer-Loeber, & Farrington, 1999).

A trend in the literature has been to offer an integrative analysis of these causative factors to violence. The U.S. Department of Justice, Office of Justice Programs, and Office of Juvenile Justice and Delinquency Prevention (OJJDP), (2004) that address the study of violence, have exposed research revealing an interactive and cumulative effect of the following risk domains: *individual, family, school, peers, and community* (Fabian, 2009; Hawkins, Herrenkohl, Farrington, Brewer, & Catalano, 2000; Masten & Garmezy, 1985) with the etiology of violence.

For example, consider an individual whose mother used alcohol and crack cocaine and was subject to domestic violence and a lack of prenatal care during her pregnancy. This individual has a history of individual characteristics, such as Attention Deficit Hyperactivity Disorder (ADHD) and substance abuse, and experienced family issues including a broken home where his mother and father were not married, there was physical abuse and neglect, and he witnessed parental domestic violence. Further, the mother was not committed to his education, and he had a history of learning disabilities, grade failures, and truancy. While seeking peer acceptance and dealing with his negative home life as an adolescent, he was exposed to peer and community risk factors in that he became a member of a gang, used substances with his peers, witnessed multiple friends murdered, he was shot and purchased a gun for self-protection, displayed conduct problems, and was beaten repeatedly over the head with a pipe to the loss of consciousness. These risk factors do not operate in isolation. Rather they have a cumulative additive effect for some type of negative outcome, such as psychopathology, violence and criminality, substance abuse disorder, homelessness, and unemployment (Fabian, 2009; Freedman & Hemenway, 2000; Moses, 1999).

In particular to serious violence, some of these risk factors have been found to have a cumulative causative effect on homicidal behavior (Fabian, 2009; Freedman & Hemenway, 2000; Loeber & Pardini, 2005). Death row statistics divulge that these inmates have histories of the following risk factors (Cunningham & Vigen, 2002; Fabian, 2003, 2009; Lewis, Pincus, Feldman, & Bard, 1986).

- 1) Intellectual deficiency (IQ's around 80 indicating borderline range of intelligence)
- 1) Low commitment to school and poor academic success
- 2) Head injury, neurological injury, and organic brain impairment
- 3) Neuropsychological and cognitive deficit
- 4) Psychiatric disorders, primarily schizophrenia and affective disorders
- 5) History of familial family abuse/neglect
- 6) Parental substance abuse
- 7) Family separation
- 8) History of substance dependence.

Our discussion will focus on the neurological and neuropsychological correlative research findings with violence and homicide.

## 2. Environmental factors breeding neuropsychological impairment

Before discussing the empirical literature reviewing the neurobiological/structural and brain-behavior correlates to violence, it is critical to highlight etiological risk factors that breed neuropathology and cognitive dysfunction. More often than not, defendants charged with homicide for example, have been exposed to various environmental factors that leave them at risk to generate cognitive, neuropsychological, and organic brain impairment which can lead to later violent behavior. Some relevant risk factors include the following: (Brennan, Grekin, Mortensen, & Mednick, 2002; Conser, Rivara, Barnoski, & Emanuel, 1997; DeMuth & Brown, 2004; Fergusson, Horwood & Lynskey, 1993; Gibson & Tibbetts, 1998, 2000; Green, Gesten, Greenwald, & Salcedo, 2008, Harrison & Sidebottom, 2008):

- 1) Young maternal age during pregnancy
- 2) Maternal alcohol, nicotine, and drug use during pregnancy and during labor
- 3) Infant testing drug positive at birth
- 4) Poor maternal diet and medical care during pregnancy
- 5) Maternal depression during pregnancy
- 6) Fetal maldevelopment, minor physical abnormalities, and Fetal Alcohol Syndrome
- 7) Low birth weight
- 8) Pregnancy and birth complications
- 9) Parental criminality and substance abuse
- 10) Domestic violence to mother during pregnancy
- 11) Poor offspring nutrition and medical care
- 12) Exposure to parental physical abuse and emotional neglect
- 13) Exposure to housing instability and deplorable home conditions
- 14) Exposure to toxins, lead, parasites, and infection
- 15) Poor socioeconomic conditions
- 16) Deficient parental and offspring education
- 17) Substance abuse and dependence history; brain dysfunction is more common among substance users. Substance abusers are more likely to have pre-existing neurological conditions and deal with conditions by use of substances
- 18) Experience of violent victimization with possible exposure to head injury and symptoms of Posttraumatic Stress Disorder (PTSD)
- 19) Low heart rate at birth.

While we know that the cumulative effect of biopsychosocial risk factors increases the likelihood of violence, we also must consider the preceding risk factors that specifically spawn organic neuropathology and neuropsychological impairment as having a unique cumulative effect on one's functional behavior and potential likelihood for violence. Most importantly, we know that the additive quality of both neurobiological and environmental factors place an individual at greatest risk for negative outcome (Raine, Buchsbaum, & LaCasse, 1997; Raine, 2002a,b).

## 3. Affective/hostile/reactive versus instrumental/predatory violence

Before focusing on the association between neurological dysfunction and neuropsychology with violence, the reader must be cognizant of the bimodal classification scheme of aggression and violence that can be associated with homicidal violence.

The bimodal classification includes considering aggression along a continuum ranging from predatory to affective defense behavior in animals (Blair, 2002; Flynn, 1976; Siegel & Pott, 1988; Siegel & Brutus, 1990). When applied to humans, the bimodal categories include instrumental and predatory aggression versus defensive affective, reactive, and hostile aggression (Cornell et al., 1996; Kingsbury, Lambert, & Hendricks, 1997; Meloy, 2000).

When considering the former instrumental type, an individual's murderous rage may be fueled by a cold-blooded premeditated act with the utilization of violence as an instrument to an achieved objective and goal (i.e., money, sex, drug trafficking territory). In contrast, the "hot-blooded" offender reacts to a stressful situation in which the goal is reducing the threat and the autonomic arousal in an instinctive manner. The violence is immediate, impulsive, emotional, and reactive rather than planned and premeditated. Table 1 illustrates Meloy's (2006) bimodal classification of affective and predatory violence.

This categorization of violence is important to note not only based on behavioral components (i.e., violence is due to emotion versus achieving a goal), but also brain structure and functioning (Raine, Meloy et al., 1998; Raine, Stoddard et al., 1998; Weinschenker & Siegel, 2002). Reactive and instrumental violence are likely mediated by different neural architectures (Blair, 2002). Specifically, affective aggression has been shown to be related to lower left and right prefrontal functioning, high right hemisphere subcortical functioning, and lower right hemisphere prefrontal/subcortical functioning (Raine, Meloy et al., 1998; Raine, Stoddard et al., 1998). In essence, reactive aggression is related to a fight response to a threat mediated by subcortical systems (Panksepp, 1998).

Blair, Mitchell and Blair (2005) have created a dysfunctional regulation hypothesis in which there is a basic threat system that mediates reactive aggression and there are areas of the frontal cortex that regulate the system. Individuals at risk for this dysfunction have heightened threats to circuitry sensitivity as a result of prior exposure to significant environmental threats, and as a result of innate biological predispositions. These individuals are at risk for reduced regulation of threat circuitry due to disturbance of orbital and medial frontal cortex regions and serotonergic abnormalities.

Other data suggest that homicide frequently occurs because the individual responds to provocation with violent aggression that is out of proportion to the instigating stimulus. The tendency for this process may be due to damage in the medial hypothalamic areas of the brain responsible for modulating defensive aggression (Albert, Walsh & Jonik, 1993).

In contrast, instrumental aggression is goal-directed motor behavior and is likely to involve the same cortical neural systems as any other goal-directed motor program (Blair, 2002). These neural systems include the temporal cortex and striatal and premotor cortical neurons to implement the actual behavior. The amygdala and ventral orbitofrontal cortex are crucially involved in learning that a particular behavior is the correct one to utilize to achieve a goal (Murray, Bussey, & Wise, 2000). If the amygdala provides a reward signal following the instrumental aggression, the individual will be more likely to be instrumentally aggressive in the future (Blair, 2002).

Instrumental violent offenders are more likely to have higher levels of psychopathy (Cornell et al., 1996). Research has revealed prefrontal cortical functioning of predatory murderers to be normal as compared to the impaired affective murderer groups (Raine, Meloy et al., 1998; Raine, Stoddard et al., 1998).

**Table 1**  
Forensic criteria for determining affective or predatory violence (Meloy, 2006).

Affective violence	Predatory violence
1. Intense autonomic arousal	1. Minimal or absent autonomic arousal
2. Subjective experience of emotion	2. No conscious emotion
3. Reactive and immediate violence	3. Planned or purposeful
4. Internal or external perceived threat	4. No imminent perceived threat
5. Goal is threat reduction	5. Variable goals
6. Possible displacement of target	6. No displacement of target
7. Time-limited behavioral sequence	7. No time-limited sequence
8. Preceded by public posturing	8. Preceded by private ritual
9. Primarily emotional/defensive	9. Primarily cognitive/attack
10. Heightened and diffuse awareness	10. Heightened focused awareness

#### 4. Structural and functional neuroimaging, neuropathology, and violence: Frontal lobe dysfunction

The fields of neurology and neuropsychology have revealed correlative research that associates certain brain structural and functional impairment with violence. Our discussion will begin with the study of brain structure and neuroimaging.

When considering neurological correlates to violence, there has been discussion pertaining to the neuroanatomical locus and areas of the brain with violence and aggression (i.e., frontal lobes and prefrontal cortex, temporal lobes, amygdala and limbic system) (Blake, Pincus, & Buckner, 1995; Giancola, 1995; McAllister & Price, 1987; Pincus & Tucker, 2003; Siegel, 2005). These areas and, in particular, the prefrontal cortex, the anterior cingulate cortex, the posterior right hemisphere, the insular cortex, and subcortical structures, such as the amygdala, hippocampus, and thalamus, are known to regulate emotion (Bufkin & Luttrell, 2005). It is hypothesized that the subcortical structures that regulate emotion (i.e., amygdala) are under the direct regulatory control of the prefrontal cortex (Seo, Patrick, & Kennealy, 2008). Impairments within the prefrontal brain regions may in part cause a biological vulnerability to impulsive aggression by limiting the capacity to inhibit subcortical emotional centers. Accordingly, murderers whose crimes are considered affective in nature promoted by situational rage are more likely to display reduced activity in the prefrontal cortex and increased activity in the basal ganglia and limbic system and amygdala (Amen, Stubblefield, & Carmichael, 1996), midbrain, hippocampus, and thalamus (Raine, Meloy et al., 1998; Raine, Stoddard et al., 1998). These and other neuroanatomical areas are hypothesized to experience neurocognitive dysfunction in their relationship to violence and aggression (Pincus & Tucker, 2003). However, no study has reliably demonstrated that prefrontal dysfunction is predictive of violent crime; therefore longitudinal studies are recommended (Bufkin & Luttrell, 2005).

The structural components of neurocognitive dysfunction or neuropathology, can be readily assessed by the neurologist or neuroscientist within the field of structural and functional neuroimaging (Roth, Koven, & Pendergrass, 2008). Brain imaging techniques include, but are not limited to, Computed Tomography (CT), Magnetic Resonance Imaging (MRI), Functional Magnetic Resonance (fMRI), Positron Emission Tomography (PET), Single Proton Emission Computerized Tomography (SPECT), and Electroencephalography (EEG).

We will first focus our discussion on the frontal lobes and prefrontal cortex. Brain imaging, neurological, and neuropsychological research all suggest that damage or dysfunction to the prefrontal cortex is a significant predisposition to antisocial and violent behavior (Langevin, Ben-Aron, Wortzman, Dickey, & Handy 1987; Martell, 1996; Raine, 2002a,b). In a review of the literature examining frontal lobe dysfunction and violence, Brower and Price (2001) found cumulative evidence from neuroimaging studies associating increased aggression and violence and reduced prefrontal cortical size or activity. The authors commented that the increased risk of violence seems less than widely assumed when considering frontal lobe dysfunction. Rather, evidence is most significant for an association between focal prefrontal damage and an impulsive subtype of aggressive behavior rather than premeditated and predatory aggression.

In a review of neuroimaging research, Bufkin and Luttrell (2005) found that 100% of SPECT and PET studies reported deficits in prefrontal functioning (frontal lobe deficits) in violent, aggressive, and antisocial groups. Analysis of specific regions in the medial prefrontal cortex revealed that individuals who were aggressive and or violent had significantly lower prefrontal activity in the orbitofrontal (4 of 10 studies), anterior medial cortex (5 of 10 studies), medial frontal cortex (2 of 10 studies) and or superior frontal cortex (1 of 10 studies). The MRI studies (2 of 4 studies) reported decreased gray matter volume in

prefrontal or frontal regions and (1 of 4 studies) nonspecific white matter abnormalities not localized to the frontal cortex.

Two regions of the prefrontal cortex, ventromedial and orbito-frontal cortexes, have been associated with the understanding and processing of information, communication, understanding others' reactions, abstracting and reasoning, controlling impulses/stopping behavior/emotional regulation, using knowledge to regulate behavior, persisting with appropriate behavior, appreciating the impact of behaviors onto others (empathy), and manipulating learned and stored information when making decisions (Brower & Price, 2001).

Dysfunction in the orbitomedial region of the prefrontal cortex has been shown to impair control over anger and impulsive aggression (Davidson, Putnam, & Larson, 2000; Lapierre, Braun, & Hodgins, 1995). Research has revealed that patients with early onset ventromedial lesions experience difficulties anticipating future consequences, delaying gratification, and engaging in risky behavior. In addition, they have defective autonomic reactions to punishment contingencies (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Bechara, Damasio, Damasio, & Anderson, 1994).

Decreases in prefrontal brain activity and increases in subcortical activity have been associated with antisocial behaviors. Criminal groups have higher rates of EEG abnormalities than controls (Yaralian & Raine, 2000). These abnormalities include in particular cortical underarousal and fronto-temporal abnormalities (Raine, 2002a,b). An MRI brain volumetric study found that subjects with antisocial personality disorder showed more violent crimes, more psychopathic traits, and reduced overall prefrontal gray matter volume (Raine, Lencz, Bihrl, Lacasse, & Colletti, 2000). Forensic psychiatric patients assessed with PET brain scans revealed decreased frontal cortical blood flow or metabolism associated with repetitive and purposeless violent behavior (Volkow & Tancredi, 1987; Volkow, Tancredi, Grant et al., 1995).

Research has divulged abnormal frontal EEG activity and diminished frontal related potentials correlating with antisocial personality disorder and a history of aggression (Bars, Heyrend, Simpson, & Munger, 2001; Bauer, O'Connor, & Hesselbrock, 1994; O'Connor, Bauer, Tasman & Hesselbrock, 1994). Frontal lobe lesions have also been the best predictor of involvement in a violent act among inpatients on a neuropsychiatric unit (Heinrichs, 1989). A study of 333 prisoners referred for evaluation after being charged with a violent crime assessed EEG findings to habitual physical aggression or explosive rages (Williams, 1969). About 57% of habitually aggressive subjects had EEG abnormalities (62% frontal) compared to about 12% of other subjects who had committed a single isolated violent act. Similarly, psychiatric inpatients who were more persistently violent were found to have more significant frontal lobe impairment than transiently violent patients (Krakowski & Czobor, 1997).

When considering the specific forensic population of murderers, one study of 31 murderers referred for mitigation evaluations revealed that 64.5% showed some physical evidence of frontal dysfunction (Blake et al., 1995). In another study of 41 defendants charged with murder or manslaughter assessing neurobiology related to mitigation, murderers as a group revealed significant bilateral prefrontal metabolic decreases during a frontal lobe activation task (Raine et al., 1997). The authors found reduced glucose metabolism in other areas including superior parietal gyrus, left angular gyrus and corpus callosum, and they showed abnormal asymmetries (left hemisphere lower than right) in the amygdala, thalamus, and medial temporal lobe.

In another study with these same defendants, they were separated into affective versus predatory types and results indicated that the affective types had significantly lower prefrontal metabolic activity relative to subcortical functioning as compared to the predatory types (Raine, Meloy et al., 1998). The predatory murderers had excessively higher right subcortical activity. Finally, Raine, Meloy et al. (1998), Raine, Stoddard et al. (1998) found that only those subjects lacking histories of psychosocial deprivation had significantly lower overall prefrontal metabolic rates. They reasoned that the group who

experienced psychosocial deprivation would be violent due to environmental factors rather than brain abnormalities.

As we have seen, the literature has designated some connection between frontal lobe dysfunction, aggression, and homicide. While it has been theorized that the prefrontal cortex regulates one's emotional functioning, including the amygdala and temporal lobe regions, we will turn to the discussion of what relationship these latter regions have with violence.

## 5. Temporal lobe and limbic system dysfunction and violence

The temporal lobes and limbic system, the amygdala in particular, are structures that, if damaged, can impact the processing of unprovoked or exaggerated anger, memory and intellectual impairment, behavioral dyscontrol, receptive language impairment, and the regulation of responses to cues that connote threat. The amygdala in particular which is located bilaterally within the temporal lobe, functions as the core of the limbic system. While functioning as the low-order autonomic neural processing center, it is grounded in the regulation of immediate emotional impulses (impulsivity). It is hypothesized that impairment in these areas is related to violence (Albert et al., 1993; Devinsky & Bear, 1984).

Bufkin and Luttrell (2005) found that 70% of the SPECT and PET studies they reviewed included temporal lobe dysfunction in aggressive and or violent groups, with reductions in left temporal lobe activity in 6 of 7 studies. When considering the medial-temporal lobe, which includes subcortical circuits such as the amygdala, hippocampus, and basal ganglia, subcortical dysfunction characterized individuals who were aggressive and or violent in 4 of 7 studies. Of these studies, 3 of the 4 found excessive subcortical activity in individuals who were aggressive and or violent, specifically on the right side compared to the left and 25% found diminished subcortical activity in general. Six of the six MRI studies reviewed reported temporal irregularities including asymmetrical gray patterns in the temporal-parietal region, decrease in anterior-inferior temporal lobe volume including the amygdala-hippocampal region or adjacent areas, and increases in left temporal lobe volume, or pathologies specific to the amygdala (Bufkin & Luttrell, 2005).

Excessive right subcortical activity or abnormal temporal lobe structure is common in patients with a history of intense violent behavior, similar to those with intermittent explosive disorder (Elst et al., 2000; Volkow et al., 1995), and in murderers pleading not guilty by reason of insanity (Raine, Meloy et al., 1998; Raine, Stoddard et al., 1998).

Scientific evidence has indicated a link between abnormal EEG's and homicide offenders, many with evidence of temporal lobe epilepsy (Hill & Pond, 1952; Langevin et al., 1988). Other research has suggested that violent homicidal offenders, who had suffered severe child abuse, are more prone to display reduced right hemisphere functioning, specifically in the right temporal cortex (Raine, 2002a,b). Further, Electroencephalography (EEG) and Positron Emission Tomography (PET) assessment in 14 murderers revealed EEG results indicating significant increases in slow-wave activity in the temporal, but not frontal lobe in contrast to prior PET findings that showed reduced prefrontal, but not temporal, glucose metabolism (Gatzke-Copp et al., 2001). Thus, some murderers have structural deficits in both the frontal and temporal lobes.

## 6. Prevalence of brain dysfunction/neuropathology in specific offender populations

While we have focused on structural and functional neuroimaging in offenders, there are also data regarding the prevalence of brain dysfunction and neuropathology within various criminal and forensic populations (Martell, 1996). Along these lines, head injuries, tumors, and other insults and injuries to the brain in some cases may produce epilepsy and have been associated with violence, aggression and

murder (Langevin et al., 1988; Lewis, Shanok, Grant, & Ritvo, 1983). Critically to this discussion, it is well established that victims of head injury are over-represented in certain sections of the population (Miller, 2002). Specifically, young adult males from socially deprived backgrounds are most likely to suffer significant head injuries which are the same group prone to engage in juvenile delinquency and adult criminality (Jennett & MacMillan, 1981; Richardson, 2000).

Martell (1996) commented that violent behavior is known to occur with certain brain disorders, including head injury, seizure disorder, cognitive impairment, and neurological abnormalities. Hafner and Boker (1982) studied 533 mentally disordered offenders in a forensic mental health system over a 10-year period and found that 33.6% of the patients had a diagnosis reflecting organic cerebral impairment including 12.7% with mental retardation; 8% with Late-Acquired Brain Damage; 7.5% with Cerebral Atrophy, and 5.4% with Epilepsy. Martell (1992) studied 50 maximum security forensic psychiatric inpatients and found that 66% of the cases had multiple indicators of potential brain dysfunction. Many had a history of severe head injury with loss of consciousness (22%), evidenced of cognitive impairment (18%), abnormal neurological findings (75%), and abnormal neuropsychological or neurodiagnostic findings (32%). Patients with a diagnosis or history suggesting organic brain impairment were significantly more likely to have been indicted for violent criminal charges.

Table 2 indicates that various offender groups have a significant chance of experiencing a history of head injury (Miller, 2002).

When considering murderers, Blake et al. (1995) found that 10% of their murderer population had suffered head injuries as compared to 67% (death row sample) in the study by Lewis, Pincus, Feldman, Jackson and Bard (1986). The lowest figures in Table 2 are in excess of the data epidemiological research suggests is the prevalence of past head injury in the general population (1–2%) (Jennett & MacMillan, 1981).

Taking into consideration psychopathic offenders, this group has been said to have more serious and lengthy histories of diverse criminal offending, engage in predatory rather than impulsive and affective violence, lack remorse for their offenses, have juvenile criminal records, and experience deficits in conscience development including traits of empathy and remorse (Hare, 1999; Hart & Hare, 1997; Salekin, Rogers, & Sewell, 1996; Serin, 1991; Serin, Peters, & Barbaree, 1990; Porter et al., 2003; Woodworth & Porter, 2003).

Neuroimaging studies have revealed brain differences in psychopathic individuals without specifically associating these deficits with violence specifically (Intrator, Hare, Strizke, & Brichtswein, 1997; Muller et al., 2003; Soderstrom et al., 2000). However, other research suggests that psychopaths have various neural-cognitive structures that may lead to their destructive and violent acts (Blair et al., 2005).

Pridmore, Chambers and McArthur (2005) collected five structural and 15 functional neuroimaging studies assessing psychopathic

offenders. Structural studies revealed decreased prefrontal gray matter, decreased posterior hippocampal volume, and increased callosal white matter. Functional studies suggested reduced perfusion and metabolism in the frontal and temporal lobes. Impairments in functioning were found in frontal and temporal lobe structures during classical conditioning and response inhibition tasks, and in the processing of words and pictures. Similarly, Weber et al. (2008) found, in their review of neuroimaging studies, that psychopaths have exaggerated structural hippocampal asymmetry, gray matter loss in the right superior temporal gyrus, and amygdala volume loss. In another study, Laasko et al. (2001) assessed a group of habitually violent male prisoners diagnosed with antisocial personality disorder and found a strong negative correlation between psychopathy scores and the volume of the posterior half of the hippocampus on both sides of the brain. Laasko et al. (2002) studied the same group and found there were no differences between this group and controls in MRI data assessing the total prefrontal, prefrontal white, and cortical volumes.

In contrast, Raine et al. (2003) studied groups of psychopathic and non-psychopathic individuals from the community measuring the corpus callosum (volume of white matter, thickness and length, using MRI) and found that psychopathic individuals had statistically significant (23% larger) callosal white matter volume with a reduction in thickness but an increase in length.

As mentioned above, dysfunction in the frontal lobe has been found to be related to antisocial and criminal behavior. Three sets of data support this argument including data from patients with lesions of frontal cortex, data from neuropsychological studies of individuals with antisocial behavior, and neuroimaging data (Blair et al., 2005). While psychopaths are known to engage in predatory violence, increased levels of reactive violence have been shown in patients with lesions in the orbital (ventral) and medial frontal cortex, but not the dorsolateral cortex (Anderson et al., 1999; Damasio, 1994).

Speculations within the literature have suggested that psychopaths may present with weak or unusual lateralization of language function. Also, they may have fewer hemisphere resources for processing language than do normal individuals, commonly referred to as the left hemisphere activation hypothesis (Hare & Jutai, 1988).

One final area of neurocognitive accounts of psychopathy includes dysfunction in the amygdala leading to impairment in emotional learning (Blair, 2001; Patrick, 1994). The amygdala is the anterior portion of the temporal lobe that is the critical region for processing emotion, and can be referred to as the “emotional brain.” Blair et al. (2005) suggests that the amygdala influences the behavioral expression of basic emotional reactions and primes the subcortical basic threat response system. The psychopathic individual's amygdala dysfunction ultimately compromises their ability to properly modulate affect and fear, respond to stimulus–punishment associations, and incorporate a moral socialization system.

**Table 2**

Prevalence of head injury in offender groups (Miller, 2002).

Study	N	Nature of group	Rate (%)
Bach-y-Rita and Veno (1974)	62	Habitually violent offenders	61
Blake et al. (1995)	31	Murderers	10
DelBello (1999)	25	Sexual offenders	36
Frierson, Schwartz-Watts, Morgan, & Malone (1998)	54	Murderers	24
Gibbens, Pond, & Stafford-Clark (1959)	72	Severely psychopathic criminals	40
Lewis et al. (1986)	15	Convicts on “death row”	67
Lewis et al. (1988)	14	Convicts on “death row” sentenced as juveniles	58
Lumsden, Chesterman, & Hill (1998)	97	Consecutive admissions to UK special hospital	42
Martell (1992)	50	Inmates in maximum security hospital for offenders	22

## 7. Neuropsychological functional impairment and violence

In contrast to structural assessment of the brain, neuropsychology is the assessment of cognitive functioning and brain–behavior relationships (Meier, 1974; Lerner, 2008) including the specific examination of functional cognitive ability, dysfunction, and impairment. The evidence of neuropsychological and cognitive impairment manifests itself in global areas of neuropsychological functioning including:

- 1) Executive functioning
- 2) Intelligence
- 3) Memory
- 4) Visuospatial construction
- 5) Attention
- 6) Language
- 7) Academic achievement
- 8) Motor coordination.

Three domains of cognitive impairment have been associated with violent behavior including executive functions, verbal abilities, and abnormalities in cerebral dominance, namely right hemisphere dominance over left-language abilities (Raine & Buchsbaum, 1996; Séguin, J. R., Pihl, R. O., Harden, P. W., Tremblay, R. E., Boulerice, B., 1995). We will focus primarily on executive and verbal functioning as it relates to antisocial behavior and violence.

Two divisions of the frontal lobes are responsible for different executive functions. The dorsolateral (prefrontal cortex) is associated with cognitive functions including language, working memory, and selective and sustained attention. The ventral and polar frontal cortex assists in regulating emotions, self-awareness, decision-making and social awareness. The following list highlights global frontal lobe brain-behavior responsibilities that potentially have an impact on the outcome of criminality and violence:

- Attention and concentration,
- Understanding, processing, and communicating information,
- Planning, organizing, and initiating thoughts and behavior,
- Understanding others' reactions,
- Abstracting and reasoning,
- Controlling impulses/stopping behavior/emotional regulation,
- Inhibiting unsuccessfully, inappropriate, or impulsive behaviors,
- Using knowledge to regulate behavior,
- Behavioral flexibility to changing contingencies,
- Modulating behavior in light of expected consequences,
- Distraction from persisting with appropriate behavior,
- Lacking appreciation of impact of behaviors onto others,
- Manipulation of learned and stored information when making decisions.

One question that remains is whether executive functioning impairments and other cognitive deficits for that matter, are empirically related to aggression, violence, and murder. If temporal or frontal lobe dysfunction is related to violence, then should we assume that neuropsychological testing results assessing these brain regions will be deficient? Additionally, neuropsychological studies do not always make distinctions between frontal lobe and other areas because neuropsychological tests are limited in the extent of their brain region sensitivity, with some tests being sensitive to impairment localized to one brain area (Raine & Buchsbaum, 1996).

We know that frontal lobe impairment does not always lead to violence or aggression; rather, frontal lobe impairments vary considerably from one another (Hart & Jacobs, 1993). Similarly, temporal lobe deficits and impairments in the limbic system are also not always associated with violent acts (Golden, Jackson, Peterson-Rohne, & Gontkovsky).

However, Raine (2002a,b) and Ishikawa and Raine (2002) imply that impairments in the dorsolateral region are involved in cognitive flexibility and response perseveration, which may be related to recidivistic antisocial and violent behavior which can be perceived as perseverative, unmodifiable behavior in the face of a repeatedly punished response.

Various studies inform us of frontal lobe dysfunction by means of neuropsychological testing. When considering neuropsychological assessment of offenders, Morgan and Lilienfeld (2000) performed a meta-analytic review of the relationship between antisocial behavior and executive functioning in neuropsychological assessment. Their results inform us that antisocial groups performed .62 standard deviations worse on executive functioning tests than comparison groups; however, evidence for the specificity of these deficits relative to impairments on other neuropsychological tasks was inconsistent. Yeudall and Fromm-Auch (1979) found that, on the Halstead Reitan neuropsychological battery, a violent offender group had significantly more anterior (including frontal lobe) neuropsychological dysfunction than a normal group. In an extensive neuropsychological investigation of aggressive criminals, Yeudall and Flor-Henry (1975) reported that 76% of the subjects had cognitive dysfunction in the

frontal and temporal regions of the brain, and 79% of these showed fronto-temporal abnormalities lateralized to the left hemisphere. Yeudall, Fromm-Auch and Davies (1982) obtained similar results for a group of delinquents including anterior cerebral dysfunction that was greater in the nondominant right than dominant hemisphere.

When considering various criminal offender groups and neuroimaging assessment, Yeudall (1977) reported abnormal Halstead Reitan findings in 90% of his sample including 94% of homicidal offenders and 87% of assaulters. Comparably, Langevin et al. (1988) examined neuropsychological functioning between homicidal, violent, and nonviolent male offenders. They found that 33% of the murders were significantly impaired on sections of the Halstead Reitan and 21% were significantly impaired on the Luria Nebraska, whereas the nonviolent samples were not significantly impaired. A study of murderers by Blake et al. (1995) found not only evidence of frontal lobe and temporal lobe dysfunction, but evidence of neuropsychological abnormalities in all subjects.

Taking into account forensic psychiatric patients, Sreenivasan et al. (2000) administered neuropsychological tests to violent non-criminally responsible offenders and mentally ill prisoners. They found that both groups displayed a pattern of lowered functioning in key cognitive areas. Both groups demonstrated cognitive rigidity, one aspect of orbitofrontal deficit and a potential factor in impulsive aggression. Foster, Hillbrand, and Silverstein (1993) found that male forensic psychiatric patients who performed poorly on the neuropsychological measures (i.e., impairments in processing of sensory schemata, ability to switch cognitive sets, and aprosodia in anger detection) exhibited a higher frequency and severity of aggression during their hospitalizations.

In contrast, the only study comparing premeditated violent aggressive individuals to non-aggressive controls reported no differences on a variety of neuropsychological tests except for a single subscale of the Wisconsin Card Sorting Test where the premeditated group exhibited greater failure to maintain set than controls (Stanford, Houston, Villemarette-Pittman, & Greve, 2003).

Research on brain dysfunction in offenders including juvenile delinquents has focused on intellectual functioning which will be explained further below (Tarter, Hegedus, Winsen & Alterman, 1984). Raine et al. (2005) found that life course persistent and adolescent limited offenders were particularly impaired on spatial and memory functions independent of abuse, head injury, and ADHD. Murderers have a history of lower IQ scores and poor problem solving abilities (Holcomb & Adams, 1983; Holcomb, Adams, Ponder & Anderson, 1984). Violent offenders are more likely to have deficits in verbal and full scale IQ scores (Valliant, Asu, Cooper, & Mammola, 1984).

Several studies suggest that habitually violent offenders with conduct disorder or antisocial personality disorder exhibit impairments in a broad range of executive and memory functions (Dolan, 1994; Moffitt & Henry, 1989). When considering more intense neuropsychological testing, Golden, Hammeke and Purisch (1980) found that violent offenders were significantly more impaired on all of the Luria Nebraska Neuropsychological Battery-Form I summary scales. Seventy-three percent of the brain damaged group committed violent crimes versus only 28% of the non-brain damaged group. The violent group had impaired performance on complex tasks requiring integration of sensory information from the auditory, visual, and somesthetic processing systems, and were deficient in abilities to create, plan, organize, and execute goal-directed behavior. Sustained attention and concentration were also impaired in the violent offender group (Golden et al., 1980).

In another study using the Luria Nebraska, violent crimes were found in 73% of subjects classified as brain damaged compared to 28% of those classified as normal (Bryant, Scott, Golden & Tori, 1984). A further study using the Luria Battery revealed impairment in functioning for the violent group in tasks measuring temporal rather than frontal lobe dysfunction (Brickman, McManus, Grapentine, & Alessi, 1984). Deckel, Hesselbrock and Bauer (1996) observed that

poor scores on Luria motor tasks and Porteus Maze test were associated with diagnosis of antisocial personality disorder, whereas the Wisconsin Card Sorting Test, Trail-Making Test, and the Control Oral Word Association Test were not.

When considering the performance of criminal psychopaths on neuropsychological testing, Hart, Forth and Hare (1990) studied adult male prisoners. They found there was no difference in performance on neuropsychological tests (Visual Retention Test, Auditory-Verbal Learning Test, Trail-Making Test, Visual Organization Test, Wechsler Adult Intelligence Scale-Revised, Wide Range Achievement Test) between offenders with low, medium, and high psychopathic traits. The overall prevalence of test-specific and global neuropsychological impairment was low; and results did not support any traditional brain-damage explanations of psychopathy.

Conversely, some studies of psychopaths have examined deficits in frontal lobe functioning finding response perseveration deficits (Gorenstein, 1982; Newman, Patterson, & Kosson, 1987). Day and Wong (1996) found psychopaths to display perceptual asymmetries and visuospatial deficits. Other studies suggest prototypical psychopathy is associated with deficits in ventromedial prefrontal function rather than dorsolateral prefrontal impairments. Psychopaths have been found to experience impairment similar to those patients with amygdala lesions, and are deficient in examinations measuring orbitofrontal–ventromedial skills (Lapierre et al., 1995). Specifically, they are prone to lack skills requiring verbal mediation, concept integration, anticipating consequences of actions, and utilizing feedback from behaviors to modify maladaptive response patterns (Miller, 1987).

Dolan and Park (2002) reported that subjects with antisocial personality disorder displayed impairments on dorsolateral frontal executive function tasks of planning ability and set shifting. They also displayed deficit in dorsolateral prefrontal Go/NoGo tasks and in visual memory tasks. Fedora and Fedora (1983) found greater impairment of dominant left hemisphere function among criminal psychopaths on the Halsted Reitan. Lapierre et al. (1995) noted that psychopaths were significantly impaired on all orbitofrontal–ventromedial tasks including Go/NoGo and a maze task, but scored similarly to nonpsychopaths on tasks related to dorsolateral frontal cortex functioning.

Recently, there has been momentous theoretical focus proposing psychopathy as a disorder of the paralimbic system indicating deficits in language processing, attention, and orienting processes, and processing and regulation of affect and emotion (Kiehl, 2006). In particular, the latest studies assessing affective processing of words by psychopaths (Kiehl, Smith, Hare et al., 2001) have revealed that their processing of affective stimuli was associated with less limbic amygdala/hippocampal formation, parahippocampal gyrus, ventral striatum and anterior posterior cingulate activation, and overactivity in the bilateral fronto-temporal cortex. While psychopaths fail to exhibit evoked response potentials differentiation of word types, such as affective versus neutral words (Kiehl, Hare, McDonald & Brink, 1999; Williamson, Harpur, & Hare, 1991), they exhibit a large and atypical centro-frontal negative going potential (Kiehl et al., 1999). Kiehl et al. (2004) studied criminal psychopaths processing of abstract and concrete words and found that they were slow to respond to both types of words. In addition, they failed to show a difference in activation of the right anterior temporal gyrus when processing both types. Consequently, psychopathy might be associated with dysfunction of the right hemisphere during the processing of abstract material.

The recent study by Debrito and Viding (2009) was the first to assess neuropsychological functioning with groups of violent psychopaths with antisocial personality disorder, violent antisocial personality disordered individuals without psychopathy, and healthy non-offender volunteers. The results indicate that both offender groups exhibited impairments in neuropsychological testing assessing cognitive functioning pertaining to amygdala, orbitofrontal and dorso-

lateral prefrontal cortex regions. The healthy control group did not display any impairments. There were no statistically significant differences between the performance of the violent psychopath group and the violent non-psychopath antisocial personality group.

The research is unclear and inconsistent regarding the prevalence of neuropathology in psychopathic offenders and its association to violence. Psychopaths may not be characterized as having damage to the frontal cortex. Rather, they may have damage to other areas such as the orbitofrontal cortex (Raine & Buchsbaum, 1996) and limbic system (Kiehl, 2006) which may result in personality changes rather than the type of cognitive impairments measured by traditional frontal lobe neuropsychological tests.

## 8. Developmental cognitive dysfunction, conduct disorder, and adult criminality

While we have discussed scientific neuroimaging data that has associated aggression and violence especially with individuals with frontal lobe dysfunction (i.e., orbitofrontal and dorsolateral prefrontal dysfunction), researchers have characterized another group of offenders who experience episodic aggressive dyscontrol rooted in “developmental deviance” (Elliott, 1990). This group of violent individuals may have isolative or cumulative features of fetal or birth related brain injury, developmental learning disorders, attention deficit hyperactivity disorder, substance misuse, and antisocial personality disorder (Kandel, 1992; Pennington & Ozonoff, 1996). This group may experience minimal brain dysfunction and neurological “soft signs” and executive functioning deficits. Essentially, “the most certain conclusion that might be drawn from the adult literature is that *persistent adult criminals* show the same general impairments on IQ tests and neuropsychological test batteries as do juvenile delinquents.” (Moffitt & Lynam, 1994). Prominently, poor neuropsychological status predicts male offending before age 13 and persistent criminality in adolescence and adulthood (Moffitt, Lynam & Silva, 1994).

Accordingly, we must describe the developmental pathways to neurocognitive and neuropsychological dysfunction in offenders. For example, amygdala dysfunction is closely related to deregulated emotions; conduct-disordered children display similar structural aberrations of fronto-limbic structures to adults with antisocial behavior (Vloet, Konrad, Huebner, Herpertz, & Herpertz-Kahlmann, 2008). Adolescents diagnosed with conduct disorder often manifest cognitive impairments characteristic of frontal lobe dysfunctions of adults with brain damage (Lueger & Gill, 1990).

Research has revealed that perhaps the most common characteristics of delinquency and adult criminal behavior is low IQ, especially verbal IQ deficit, as those with verbal shortage often rely on physical and emotional modes of self-expression (Lynam, Moffitt & Stouthamer-Loeber, 1993). Intelligence and IQ is a measure of neuropsychological health, and represents executive functioning including sustained attention, concentration, social judgment, language processing, abstract reasoning, planning, and initiating purposeful behavior (Moffitt & Lynam, 1994). Normal auditory-verbal memory and verbal abstract reasoning skills influence the success of a child's socialization, and are essential to the development of self-control for inhibiting childhood behaviors (Moffitt, 1993a,b). Low IQ scores, especially verbal IQ, are indicative of impulsive judgment, weak language processing, poor memory, and failure to synchronize visual information with motor actions (Lezak, 1988). It is hypothesized by Moffitt (1993a,b) that life course persistent delinquents begin their antisocial behaviors early in childhood because they develop subtle neuropsychological dysfunctions which disrupt normal development of language, memory, and self-control.

While IQ has been suggested to be related to violence, Moffitt, Lynam, & Silva (1994) assessed whether global neuropsychological performance (IQ, motor coordination, auditory-verbal learning, visuospatial perception, and executive functioning) were associated with criminality. While they

found that verbal skills and verbal memory abilities were most robustly related to delinquency, neuropsychological scores at age 13 predicted delinquency at age 18.

Although verbal deficits are significantly related to delinquency, and to some degree adult criminality, the clinical diagnosis of attention deficit hyperactivity disorder (ADHD), a condition of attentional and prefrontal lobe executive dysfunction, is also significantly related to delinquency and early aggression (Lilienfeld & Waldman, 1990).

The concept of attention is the base of cognitive activity. Low cortical arousal and impairment in selective attention, poor planning ability, information acquisition, storage and retrieval of knowledge, and a history of learning disorders, are all related to functional attention (Hurt & Naglieri, 1992). Low autonomic activity manifested by stimulation seeking, perceptual motor impairments, restlessness, psychomotor impulsivity, and fearlessness predisposes one to aggression and criminality and is related to symptomatology of ADHD.

Nearly 55% of delinquents qualify for ADHD (Zagar et al., 1989). In fact, delinquents suffering from ADHD are at significant greater risk for adult criminality than those without ADHD (Farrington, 1990). Juvenile delinquents and adult offenders who suffer from neuropathological attention deficits have difficulties selectively ignoring or attending to salient competing stimuli in receptive and expressive attention tasks. Juveniles with ADHD are more likely to have a number of legal difficulties, carry a weapon, and engage in serious physical aggression (Lilienfeld & Waldman, 1990).

When considering a developmental association between ADHD and adult crime, adult violent offenders experience similar brain-behavior deficits and function poorly on assessments of executive functioning as delinquents with chronic histories of aggression. The association of antisocial behavior with impaired performance on executive functioning tasks may be due to individuals with ADHD presenting with antisocial behavior. ADHD may be a risk factor for impairment that leads to antisocial behavior even if the pathology associated with ADHD itself does not lead to aggression (Blair et al., 2005).

A marked number of adult inmates suffer from ADHD symptoms as children and continue to have these traits in adulthood (Eyestone & Howell, 1994). Vitelli (1996) studied the prevalence of conduct disorder and ADHD in adult maximum security inmates and found that 63% of the group qualified for childhood conduct disorder as youth and 41% qualified for ADHD during childhood. Lie (1992) suggested that conduct disorder was a better predictor of subsequent development of adult criminality than ADHD. However, children who had both conduct disorder and ADHD were at a higher risk than children with conduct disorder alone. Children with both conditions tend to be arrested at an earlier age and have more total charges than children with conduct disorder alone (Moffitt, 1990).

When considering psychopathic offenders, some argue that they share attributes to those with deficits in prefrontal lobe functioning and ADHD in particular (Morgan & Lilienfeld, 2000). Common characteristics of psychopaths and youth with ADHD include low frustration tolerance, difficulty delaying gratification, antisocial behavior, and poor planning and judgment (Lilienfeld & Waldman, 1990). Lynam (1996) proposed that individuals with both ADHD and child conduct problems are “fledgling psychopaths” suggesting that children experiencing both ADHD and conduct disorder are characterized by significant neuropsychological, executive, and information processing deficits associated with adult psychopathy.

When considering the preceding information, neuropsychological impairment especially in the areas of verbal IQ, language processing, attention, and executive functioning skills, may be related to the onset and consistency of offending behaviors (Teichner & Golden, 2000). It should be emphasized that diagnoses of criminality including Conduct Disorder and Antisocial Personality Disorder, express a developmental aspect of criminality. Some of the symptoms of these disorders may be explained, in part, by neuropsychological/neurological impairments (recklessness, impulsivity, appreciating consequences, aggression, fear conditioning to

punishment, and regulation of arousal) rather than by conduct or personality diagnoses. Additionally, the comorbidity of conduct disorder and ADHD, for example, is quite high, as about 50% of those delinquents qualifying for conduct disorder also suffer from ADHD (Gittelman, Mannuzza, Shenker, & Bonagura, 1985; O’Shaughnessy, 1992). Further, those defendants with a history of juvenile delinquency and conduct disorder together are much more likely to display developmental criminality and violence than those with just one disorder.

While we know that there are correlative links between poor verbal skills and ADHD with criminality, the exact processes that associate the two are less clear.

## 9. Neuroscience, freewill, and moral culpability

To this point, we have discussed the neurological and neuropsychological correlates of violent and homicidal behavior leaving many thoughtful questions. How does a forensic clinician apply this information to forensic mental health issues? For example, psychiatric disorders, such as schizophrenia and bipolar disorder, may impair one’s thoughts and behaviors and interfere with reality contact, rational thought, and volitional behavior. These disorders are the foundation of a legal insanity defense that negates a defendant’s criminal responsibility. Similarly, how do the fields of neurology and neuropsychology lead, respond to, or integrate with legal questions? Consider for example whether neurocognitive impairment, as assessed by structural imaging of frontal lobe lesions, prior diagnosis of ADHD and learning disability, and/or neuropsychological functional testing indicating low IQ and executive functioning deficits that affect one’s thoughts and behaviors, can be used as mitigating one’s lack of freewill and behavioral control, ultimately speaking to moral culpability.

Human behavior, and violence, aggression, and murder can be considered from a dichotomous image of biopsychosocial determinism versus freewill and conscious choice and responsibility over one’s behaviors (Fabian, 2009). Consider that defense attorneys take a deterministic stance of moral culpability, and will consider their murder defendant’s behavior as being shaped by their living in a broken home, being abused, having their mother prostitute within the home, and being exposed to gangs and drugs while being shot three times in the community. On the other hand, the concept of freewill is not a legal excusing condition, nor is it a criterion for any clinical diagnostic category (Morse, 2007). There are few data in the field of psychology and neuroscience that suggest that most people most of the time are not conscious and intentional creatures who act for reasons (Morse, 2007). Accordingly, the prosecutor in a capital case will rely on a simplistic analysis of human and criminal behavior, including the defendant as being plagued with chronic antisocial behavior, juvenile delinquency, violence, antisocial personality disorder, psychopathy and always having complete freewill over his behaviors without paying heed to biopsychosocial risk factors affecting their life.

From a psycholegal forensic mental health perspective, the psychologist evaluating a capital defendant and an attorney defending him in a mitigation legal proceeding, must emphasize the mitigating elements that place a defendant at risk for homicidal behavior, and ask the jury whether the defendant is “death-worthy” and morally blameworthy enough for execution based upon some factors beyond his control. Some of these factors relevant to this article are based on innate and/or environmentally caused neuropsychological impairment that may in part affect the defendant’s ability to make choices and to exert complete control over his behavior.

## 10. Cognitive dysfunction and freewill

As previously highlighted, two areas of the brain, (amygdala and the prefrontal cortex) interplay and impact one’s decision-making and willpower (Burns & Bechara, 2007). The amygdala neural system is impulsive and based on immediate emotional responses and



prospects of an option. It is engaged in regulating emotional situations requiring a rapid response and can be described as a “low-order” regulator of emotional reactions arising from autonomic processes. In contrast, the frontal lobes are a reflective neural circuit signaling consequences of the future prospects of an option. Once the lower order reactions are made, “higher order” emotional responses are monitored by the ventromedial prefrontal cortex which is driven by thoughts and reflection requiring thinking, reasoning and more conscious awareness. It is proposed that willful behavior is the product of a healthy interaction between these two separate and interactive neural circuits and an impaired process may have its roots in a hyperactive impulsive amygdala system that overhauls a dysfunctional reflective cortex system.

When considering self-control and cognitive dysfunction, there is an association between the combination of both executive functioning and verbal ability with violence. The impairment in executive functioning reflects a deficit in organizing several parameters simultaneously, distinguishing complex rules, planning and anticipating future consequences of choices, behaviors, and actions, and reasoning abstractly in order to solve interpersonal and social dilemmas (Séguin et al., 1995). The ability to reflect and reason appropriately in impaired defendants may be overwhelmed when they are in a motivational situation that requires a more adaptive social response (Patterson & Newman, 1993).

The issue of freewill and human violent behavior may be dissected within types of aggression, such as impulsive and reactive aggression versus premeditated and predatory violence. There may be, on average, more empirical data associating neuropathological dysfunction with the former than the latter type of violence (Houston et al., 2003). In particular, affective defensive and reactive violence resembles episodic dyscontrol (Weinschenker & Siegel, 2002). This type of reactive rage assumes the presence of excessive neuronal discharges from limbic structures to subcortical regions such as the hypothalamus and brainstem. It is hypothesized that aggressive individuals have an inability to regulate negative emotions in situations where they or others are vulnerable (Davidson et al., 2000). The inability to regulate this negative emotion, and in particular, reactive violence, stems from the capacity of the prefrontal cortex to inhibit emotional activation arising from subcortical structures. Impaired regulatory control of the prefrontal cortex may lead to excessive negative emotional reaction and violence (Seo et al., 2008). The argument of volitional impairments affecting a psychopath's freewill as to their premeditated violent acts is less well received at this time and further investigation is needed to explore this area.

## 11. Substance abuse and freewill

Research from the National Institute on Drug Abuse suggests that half of all violent episodes in the United States occur when the victim and or offender are under the influence of acute intoxication. Substance abuse likely plays a role in two-thirds of violent crimes, 62% assault; 68% manslaughter; 54% murder or attempted murder (NIAAA, 1990). Death row inmates report significant histories of substance abuse and dependence in the community as well as being under the influence of substances during their homicidal offenses (Cunningham & Vigen, 2002).

A defendant's freewill and control and inhibitions over behavior are also affected by conditions of substance intoxication, abuse, dependence, and addiction. The biological mechanisms in humans imply that in the thrust of addiction, individuals do not have freewill to behave otherwise (Dilts, 2000). The brain depends on dopaminergic tracts to drive biologically preferred behavior. Substances that stimulate the dopaminergic system such as cocaine, ultimately become biological imperatives to the addicted organism. Addicting substances become a life and death essential that can be resisted only with great effort. Ultimately one's freewill must toil against a brain

giving signals that to go without the addicting substances is to run great risk. Addictive substances essentially “hijack a biological system designed to reward biologically necessary behavior, and instead compel addictive behavior as a biological imperative” (Dilts, 2000). Source: *Models of the Mind: A Framework for Biopsychosocial Psychiatry*. Stephen L. Dilts. Routledge 2000.

Notably to the substance of this paper, individuals with mental illness and or neuropathological and cognitive impairment are more likely to abuse substances and the combination of cognitive dysfunction and substance abuse place an individual at greater risk for violence (Fishbein, 2000; Langevin, Wortzman, Dickey, Wright, & Handy, 1988). Theoretically, substance abuse, criminality, and violence have all been linked to a neurobiological entity, the ventromedial prefrontal cortex. Research has indicated that executive dysfunction, such as deficits in decision-making, cognitive flexibility, and response inhibition, are associated in substance abuse in some individuals (Dolan, Bechara, & Nathan, 2008).

The link between brain functional impairment, drug abuse, and violence, is mediated by altered cognitive capacities, such as attention, concentration, verbal ability, abstract reasoning, problem solving, planning, and goal-oriented behaviors (Mirsky & Siegel, 1994). Moreover, structural impairments, such as abnormalities in the way the brain metabolizes glucose, are similar between violent offenders and substance users (Stapelton et al., 1995). Alcohol abuse and brain damage may have a synergistic effect on the disinhibition of behavior together predisposing individuals with developmental or acquired brain defects toward aggression (Elliott, 1992; Miller, 1990). These findings may lend themselves to a further connection between the etiology of violence with causative roots of cognitive impairment and substance abuse.

While most violent offenses that are associated with substance use are based on voluntary intoxication of the defendant, the addictive quality of substances and the pre-existing conditions of the defendant (i.e., history of mental illness and neuropsychological impairment) are related to increase use of alcohol and drugs and heightened inability to control this use (Fabian, 2007). Further, impulsive violence shares many of the same biological components as substance abuse given their comorbidity and similarities in behavioral dimensions.

Importantly, there is a developmental association between substance abuse and violence (Wagner, 1996; White et al., 1999). The frequency and severity of substance use for adolescents are related to the frequency of violence. Among juveniles who engage in both delinquency and substance abuse, delinquent behavior, including aggression, developmentally precedes their initiation of drug and alcohol use (Elliott & Huizinga, 1989; White, Loeber, Stouthamer-Loeber, & Farrington, 1990). Early drug use predicts later violence and violence also predicts later drug and alcohol use. Additionally, those offenders with developmental cognitive deficits are more susceptible to abusing substances.

In summation, the presence of both substance abuse disorders and neurocognitive pathology places an individual at greater risk to experience problems with reason, judgment, attention, planning, and other executive tasks that are linked with aggression and behavioral impulsivity.

## 12. Neuroscience and the law: Recommendations for lawyers and forensic neuropsychologists practicing within the criminal justice system

In this section, we will attempt to address how neuroscience can be applied within the criminal justice system, specifically in its connection with violence at mitigation in capital death penalty sentencing proceedings.

Every courtroom is a laboratory of human nature where jurists clinically and legally question our memory, behavior, emotion, sanity, and sense of responsibility and culpability (Hotz, 2009). Forensic neurology and forensic neuropsychology are the applications of neurology and neuropsychology to address legal questions. Forensic

neuropsychologists are utilized more often than neurologists in the courtroom based on the need for assessment of a defendant's functional capacity. A neuropsychologist, for example, can assess for cognitive functioning via neuropsychological testing instruments and use these data, in part, to address a legal question such as whether a defendant's brain damage is an excuse or mitigating factor to explain his criminal behavior.

When considering human behavior and criminal behavior, in particular, contemporary forensic psychology and psychiatry base its concepts of responsibility and blame upon free and voluntary decision-making as a psychosocially determined process (Witzel, Walter, Bogerts, & Northoff, 2008). However, recent advances in neuroscience reveal that decision-making is not merely a psychosocially determine process, but a biopsychosocial determined neuropsychological process (Witzel et al., 2008).

When considering mitigation at capital sentencing, such evidence can include anything about the defendant's background, character, and the nature of the offense, for example (Lockett v. Ohio, 1978). This evidence does not give rise to a criminal defense. Rather, it assists in describing "the defendant's homicidal acts and prior violent/criminal behavior in a humanly understandable light given his past history, unique characteristics affecting his development, and exposure to heightened risk factors and deficits in protective/mediating factors" (Fabian, 2009). Mitigation evidence assists the trier of fact in assessing the defendant's moral culpability, blameworthiness, and appropriate punishment for a crime(s).

In Alabama for example, the legislature has outlined some of the following mitigating circumstances in death penalty cases:

- (1) The capital offense was committed while the defendant was under the influence of extreme mental or emotional disturbance;
- (2) The capacity of the defendant to appreciate the criminality of his conduct or to conform his conduct to the requirements of law was substantially impaired.

The neuroscience expert can address, in part, these two areas through the assessment of neuropsychological structure and function. Further, the expert must address potential risk factors, such as the earlier mentioned environmental risk factors that may breed neuropsychological dysfunction and how they may impact behavior and lead to violence. Finally, the forensic neuropsychological expert must investigate the issue of resiliency pertaining to the balance and integration between risk factors, protective factors, coping skills, and innate neurological and neuropsychological deficits, the latter affecting one's resourcefulness, problem solving skills, and appreciation of consequences, for example (Fabian, 2009).

The criminal defense attorney who finds him/herself representing a defendant who he suspects or knows to have a history of neuropathology and cognitive dysfunction must enlist the assistance of other team members (e.g., psychologist competent in neuropsychological assessment, psychiatrist, neurologist) to assist in the investigation and examination of his/her client. Data the attorney should investigate regarding potential neurocognitive disorder include:

- 1) Past/present diagnosis of organic brain disorder
- 2) Past psychological, neuropsychological, and neurological diagnostic assessment data including abnormal neurological/cognitive/neuropsychological findings
- 3) History of traumatic brain injury and/or severe head injury with loss of consciousness (documented or self-reported by defendant)
- 4) History of seizure activity
- 5) Evidence of cognitive impairment and low IQ
- 6) Collateral information concerning risk factors affecting cognitive development, neuropathology and neuropsychological functioning
- 7) History of ADHD during childhood and adolescence.

Despite the research that associates structural brain deficits and violence, many capital defendants referred for neuropsychological assessment may not have a well-documented history of head injuries or structural impairment. In fact, most offenders will not have a history of brain imaging studies revealing structural deficits, but rather have evidence of global cognitive impairment and some neuropathology and cognitive dysfunction. Even if they are examined with structural imaging techniques, such as MRI, EEG, and CAT scan during the pretrial phase, results may not divulge evidence of impairment. Essentially, cognitive dysfunction can be lost in a structure that appears normal via neuroimaging data.

Despite this lack of structural data, this author contends that many offenders may still have "underdeveloped" brains marked by neuropathology and organic brain disorder conditions that have no known etiological source. It should be emphasized in capital cases where a history of childhood abuse is common, this early childhood trauma may interfere with normal brain development. Physical abuse, neglect, and lack of attachment will affect neural circuitry (especially the orbitofrontal cortex, anterior cingulate cortex, and the amygdala) responsible for physiological, emotional, psychological, and social development (Heide & Solomon, 2006). Trauma is likely related to an individual having difficulties accessing higher cortical centers in the brain which are needed for one to think logically, formulate decisions, regulate affect and relate to other people appropriately. Fittingly, when biological and social factors are grouped and violence and criminality are the outcome, the presence of both risk factors exponentially increases the likelihood of such negative outcome (Raine, 2002a,b).

A common capital case scenario includes the defendant with a history of learning disorders, ADHD, conduct disorder, adolescent and adult substance abuse, and antisocial personality disorder diagnosis in adulthood. This group may have evidenced episodic violent behavior rooted in "developmental deviance" manifested by "attention deficit disorder and minimal brain dysfunction," and associated with "neurological soft signs and executive function deficits" without a history of documented neuroimaging highlighting neurological disorder (Elliott, 1990; White et al., 1999; Widom, 1989). In other words, the defendant may have a history of cognitive brain dysfunction that falls short of major brain impairment. However, these deficits have in some ways affected his thinking and behavior and are linked to antisocial behavior, aggression, and even homicidal behavior. Critical to this point is the premise that neuropsychological impairment is developmental in nature.

Unless a murder defendant with neuropsychological impairment has a documented history of head injury and then resultant functional impairment, we must assume to some degree that those who display functional deficits without known documented histories have experienced a developmental process of neurocognitive dysfunction/organicity. Accordingly, Raine (2006) have found that life course persistent offenders are more likely to have experienced deficits in neuropsychological testing and these impairments are not attributed to a history of head injuries, ADHD, or environmental factors. In essence, these deficits may play a significant role in molding antisocial behavior rather than being a consequence of a condition, such as ADHD. Therefore, these offenders' deficits are not easily explained by known conditions and they may not be free of long-lasting cognitive impairment. Therefore, it is critical to formally and thoroughly assess a chronic antisocial offender's neuropsychological functioning.

Given the limited financial resources in many U.S. jurisdictions even in capital cases, and courts' fear of pursuing unwarranted investigative "fishing expeditions," neurologists may not be requested for neuroimaging data in "soft sign" cases. Rather, a neurological evaluation with neuroimaging may be best utilized if the defendant

has a documented history of organic brain disorder, seizure disorder, or a history of prior abnormal neurological findings.

The use of brain imaging in homicide case deserves a brief discussion. While U.S. courts have routinely admitted neuroimaging data (CT and MRI) as proof of brain trauma and neuropathology, they have been more reluctant to admit PET, SPECT, and fMRI imaging especially when extrapolating this evidence to a violent or homicidal act (Moriarty, 2008). Specifically, neuroimaging has potential use in criminal trials concerning areas of insanity, diminished capacity, and mitigation for evidence.

Criminal cases throughout the U.S. have addressed neuroimaging in capital cases. In *People v. Protsman*, the defendant utilized an expert to communicate PET scan evidence to the jury to mitigate the degree of murder. The expert testified to frontal lobe damage of a traumatic brain injury. In rebuttal, the state's expert testified that PET scan data are not generally accepted in the medical field for diagnosing or evaluating head trauma or predicting behavioral patterns. The California Court of Appeals agreed with the state expert that the defense's utilization of the PET scan images were not supported by the neurology and brain imaging community. Sources Moriarty, J. (2008). Flickering admissibility: Neuroimaging evidence in the U.S. Courts. Behavioral Sciences and the Law, 26, 29–49. *People v. Protsman*, 105 Cal. Rptr. 2d 819 (Cal. App. 4th Dist. 2001).

State supreme courts have honored the importance of neuroimaging examinations in capital cases. In *State v. Reid* (2006), the Tennessee Supreme Court accepted the trial court's admission of a PET scan in which the expert testified that the results indicated shrinkage and atrophy of the left temporal lobe of the defendant's brain. In *State v. Hoskins* (1999) the Supreme Court of Florida ruled that the trial court erred in disallowing defense experts to conduct a PET scan of the defendant. Whether structural imaging data will be regularly admissible and persuasive or probative evidence for that matter in assessing criminal responsibility, mens rea, and mitigation is yet to be seen on a consistent basis (Feigenson, 2006).

While the courts are contemplating the admissibility and relevance of neuroimaging in its connection or nexus to violent acts, recent research indicates that neuroimaging and neuropsychological testing data can affect mock juror decision-making. Gurley and Marcus (2008) assessed the effects of neuroimaging and traumatic brain injury on insanity defenses. Mock jurors were presented with descriptions of violent crimes and asked to determine whether the perpetrators should be found not guilty by reason of insanity. Some participants received a description of the crime along with expert testimony that the perpetrator was psychopathic. Other participants received the description of the crime plus one of the following (a) evidence that the perpetrator has a history of brain trauma, or (b) brain images suggesting damage to the frontal lobes. Results suggest that the percentage of participants who judged that the perpetrator was not guilty by reason of insanity was higher when accompanied by a brain image (37%), by testimony concerning brain injury (43%), or by both (50%), than they were when participants received neither (22%) source.

In contrast, critical to the forensic examination is the focus on cognitive function examined in neuropsychological assessment. Even with refinements and enhancement in neuroimaging, forensic neuropsychological and neuropsychiatric evaluations must address the interplay between biopsychosocial factors to develop a full understanding of the mental disorders and their association with violence (Felthous & Saß, 2008). Brain-behavior relationships, as measured in neuropsychological assessment, are directly connected with decision-making and moral judgment when defining the domain of criminal responsibility and blame (Witzel et al., 2008).

Fittingly, given our knowledge that the majority of murder defendants have a history of some neuropsychological impairment (Blake et al., 1995; Cunningham & Vigen, 2002; Lewis et al., 1986), neuropsychological testing beyond IQ assessment is recommended.

Intellectual assessment is the core assessment modality as it in many ways guides further examination in cognitive areas, (i.e., verbal and language skills, attention, and visuospatial abilities). However, further neuropsychological testing in the areas speculated to have impaired functional capacity will provide insight into not only abilities the tests measure (i.e., planning, problem solving, and judgment), but perhaps also to specific brain regions where there might be traces of organicity and damage. The forensic neuropsychological assessment of violent offenders should include an evaluation of executive functioning, particularly in cases involving recurrent impulsive aggression (Brower & Price, 2001).

Examinations of capital defendants will often yield impairments, primarily relevant to verbal IQ and executive functioning deficits which have been associated with impulsivity, problems with planning and judgment, inability to appreciate consequences of behaviors, deficits in learning appropriate responses, abstract reasoning, and understanding social cues for example (Henry & Moffit, 1997). Executive functioning impairment detrimentally affects one's abilities to generate alternative socially acceptable and adaptive behavioral responses and to execute a sequence of responses necessary to avoid aggressive and stressful interactions. These deficits may very well be associated with antisocial behavior, aggression, and homicidal behavior.

The neuropsychologist expert must not conclude that poor performance on a test or tests directly relates to the causative factors of the homicidal act. Brain structure and function data can be perceived as one factor among many that predisposes one to an increased probability of a type of behavior or influence on behavior (Yang, Glenn & Raine, 2008). For example, evidence of child abuse and its relationship as influencing one to adult violence is often used in court as mitigation. This evidence does not imply the existence of a one-to-one causal relationship; and similarly, neuroscientific evidence should be handled the same way.

The expert witness presenting neuropsychological evidence must be descriptive and provide insight into the defendant's general cognitive functioning, such as planning, judgment, abstract reasoning, and ability to use knowledge to regulate behavior. However, the expert must embrace the fact that no single test can infer a causal connection between something as complex as a single homicidal act, for example, to brain insults of any type. All testing instruments and technology has its limitations that affect the ability of legal and mental health professionals to infer causal relationships (Tancredi & Brodie, 2007). Only then can the jury consider whether the defendant's level of functioning as compared to the general population is so impaired as to consider sparing his life.

The capital defense attorney and his expert must attempt to educate the jury about what the neuropsychological testing and assessment means in the context of the defendant's cognitive functioning in light of the defendant's history (i.e., environmental background affecting neuropsychological development, prior neuropsychological testing and neuroimaging data), and the commission of the homicide if possible.

When addressing the latter issue, the U.S. Supreme Court holding in *Tennard v. Dretke* (2004) informs us that death penalty mitigation need not be a nexus to the homicide; however, capital jurors wish for an explanation of the violence (Fabian, 2006). Accordingly, the forensic neuropsychologist's ultimate role in death penalty mitigation may be to educate the jury on the presence of neuropsychological functioning and explain these results in light of the capital defendant's ability to form intent to commit the crime, plan his behavior, use knowledge to regulate behavior, inhibit unsuccessful behaviors, and appreciate consequences and risks to his behavior.

By utilizing the research data outlined throughout this paper, the criminal defense team in a capital case may consider environmental risk factors that produce cognitive dysfunction and similarly address developmental deviance and developmental neuropathology,

distinguish between types of violence (affective versus predatory), and present neuropsychological functional assessments and neuroimaging data when indicated.

When considering homicidal violence, the forensic expert must be knowledgeable about the research and literature distinguishing affective versus predatory violence. The literature proposes that reactive aggression and rage may be not only situationally based, but also in part due to reduced activity in the prefrontal cortex and increased activity in the basal ganglia and limbic system (Amen et al., 1996; Raine et al., 1998). It is hypothesized that violence and aggression may have its origin in an impulsive amygdala system that overhauls a dysfunctional reflective prefrontal cortex system (Sapolsky, 2004). This imbalance resulting in reactive aggression could be considered aggression without freewill (Blair, 2007).

The forensic examiner must be cognizant that the age when any damage to the prefrontal cortex occurs is critical to one's development. Specifically, damage any time after the adolescent years causes an adult to have markedly impulsive behavior and little foresight for assessing future consequences when in an emotionally charged situation (Sapolsky, 2004). In contrast, when damage occurs at earlier ages, executive function is impaired and the impulsivity takes on a global and destructive nature which may be in part causative of psychopathy and premeditated aggression.

Importantly to assessing life course persistent antisocial and violent offenders versus adolescent limited offenders, the former group may more likely experience early damage or dysfunction to the prefrontal cortex, and this may lead to information overload during adolescence resulting in less regulatory control and further life long antisocial behavior (Raine, 2002a,b). Others with an intact but late maturing prefrontal cortex may be antisocial during childhood and adolescence. However, further maturation of the frontal lobes in early adulthood may lead to a cessation of antisocial behavior. Raine (2002a,b) argues that social and executive function demands of late adolescence superimpose the late developing prefrontal cortex leading to prefrontal dysfunction and a lack of inhibitory control over aggression that peaks at this age.

When advancing the issue of age further, the U.S. Supreme Court recognized in *Roper v. Simmons* (2005) the differences between mentally retarded adults and juvenile delinquents charged with murder. The Court considered the American Psychological Association's amicus brief providing expert opinions addressing recent MRI research on adolescent brain function suggesting that the brain continues to develop through young adulthood in areas that affect adolescent decision-making and behavioral regulation. The amicus brief considered characteristics of adolescents and their relationship to aggression and violence including less mature decision-making, impulsivity, risk-taking, peer orientation, and temporal perspective, the extent to which long term and short term consequences are taken into account (APA, 2004).

Although executing juveniles in capital proceedings is now unconstitutional, neuropsychological assessment of juveniles germane to mitigation at sentencing in non-capital homicide case is still essential. Further, and important to the substance of this article, the assessment of developmental neuropsychological deficits in adult defendants is critical in capital proceedings, as many of these offenders have suffered from a history of cognitive impairment through childhood and adolescence.

In summary, the connection between neurological and neuropsychological impairment and aggression and violence is notable, and the background histories of many murder defendants breed impairments in these areas. These cognitive impairments, coupled with other biopsychosocial risk factors, may be linked to an individual's capacity to inhibit and control their behavior. Accordingly, some capital defendants may lack the inherent freewill of human behavior due to a shortage in their neural circuitry resources, marked cognitive deficits, and stressful and threatening environmental situations.

Neuropsychology must inform criminology. The substance of mitigation and the resolve of moral culpability, punishment, and blame lie within the argument of human behavior and the dichotomy of freewill versus determinism. Mitigation can be considered as a cumulative interaction between biopsychosocial factors (Fabian, 2009; Freedman & Hemenway, 2000; Loeber & Pardini, 2005), such as paternal substance abuse during pregnancy, abuse, neglect, violent victimization, ADHD, verbal deficit, foster home placement, and a lack of treatment and intervention efforts.

In the spirit of this article, mitigation can also be considered cumulative evidence within one unique sphere, that of neuropsychological and neurological dysfunction. As one author said marvelously, "To understand is not to forgive or to do nothing; whereas you do not ponder whether to forgive a car that, because of the problems with its brakes, has injured someone, you nevertheless protect society from it... but although it may seem dehumanizing to medicalize people into being broken cars, it can still be vastly more human than moralizing them into being sinners" (Sapolsky, 2004).

While it is critical to integrate neuropsychological and neurological findings that are present in a case to the defendant's behavior at the time of the offense, the goal of the forensic neuropsychologist is a thorough explanation to the trier of fact rather than an excuse (Reynolds, Price & Niland, 2003).

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