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Structural Brain Abnormalities in Aggression and Violent Behavior

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Abstract

The purpose of this review is to synthesize the existing research on the structural brain abnormalities among perpetrators of violence. While previous work has documented the functional brain correlates of aggressive behavior, insufficient attention has been paid to structural brain damage and abnormalities associated with violence perpetration. Thus, the present review consists of three domains that have been examined in the context of violence perpetration, namely, head injury studies, lesion studies, and structural neuroimaging. Findings from head-injury studies suggest that brain damage is associated with difficulties in decision making and affect regulation. Lesion studies suggest that lesions in the prefrontal, temporal and associated limbic structures are particularly important, while imaging studies emphasize the orbitofrontal cortex as an area of influence. Overall conclusions include strong evidence from all three domains that damage or reductions in frontal areas of the brain and connected regions are associated with violence and aggression. Discussion of the clinical implications of existing findings, and present suggestions for future studies is included.

Structural Brain Abnormalities in Aggression and Violent Behavior

1. Introduction

The extant literature suggests that there is an association between brain damage (such as traumatic brain injury or lesions) and interpersonal violence and aggression (Farrer, Frost, & Hedges, 2012; Pinto et al., 2010). For example, as many as 70% of patients with traumatic brain injury (TBI: defined as a resulting injury that results from a violent blow or jolt to the head or body or from an object penetrating the skull; Mayo Clinic, 2014), display considerable irritability and aggression post-injury and cause significant distress to their families (McKinlay et al., 1981). As a number of researchers have outlined the functional neurological correlates and risk factors for interpersonal violence (e.g., Ali & Naylor, 2013; Howard, 2012; Pinto et al., 2010), our review attempts to provide the first comprehensive detailing of structural disruptions (i.e. brain injury), which are often accidental and incidentally-obtained in nature. Existing research from head injury and lesion studies will be discussed with the hope of elucidating structural changes that may predispose individuals to violence and aggression. With this aim, we will subsequently suggest areas that warrant further study with combined structural and functional imaging methods.

Before proceeding, it is important to note the inconsistencies surrounding the assessment (Norlander & Eckhardt, 2005) and conceptual overlap in the terms *aggression* and *violence* that can contribute to confusion in interpreting results. While violence is often conceptualized as an aggressive act, many aggressive acts are not violent (e.g., do not cause harm; Anderson & Bushman, 2002). Further, an act of violence may not be aggressive, in that the harm was accidental or an act of defense. In collecting articles for the present review, we found that definitions of aggression and violence often implied that the constructs were indistinguishable. Herein, violence refers to behaviors that inflict physical harm. Conversely, aggression can refer to both affect/personality and violent behavior (e.g., irritability or frustration versus infliction of physical harm). Thus, our review proceeds with the knowledge that trait aggressiveness and violent behavior are correlated but not identical (see O'Leary, Slep, & O'Leary, 2007), and that violence perpetration can occur without trait aggression, and vice-versa.

Included articles were focused on scientific works containing male adult (18 or older) samples of perpetrators of intimate partner violence (IPV; any physical, sexual, or psychological harm occurring between current or former intimate partners) as well as general violence and aggression. Studies containing clinical populations, such as those selected exclusively containing a comorbid psychiatric diagnosis or medical conditions were excluded from the present review. As alcohol use is frequently associated with both partner and general violence, studies containing populations with comorbid alcohol problems were included. Literature searches were conducted on PsychINFO, Google Scholar, and PubMed for relevant articles. The search terms for head injury and lesion studies were *head injury*, *traumatic brain injury*, *intracranial injury*, *lesion*, *brain damage*, and *frontal lobe injury* combined with the terms *partner violence*, *partner abuse*, and *domestic violence*. For the review on imaging techniques, we limited our discussion to studies not included in the above sections, and used the terms *structural imaging*, *neuroimaging*, *MRI*, and *brain abnormality*. Search terms from each section were also combined with the terms *general violence*, *violence*, *violent crime*, and *aggression* for the integrated discussion.

2.1 Head Injury

The occurrence of head injury was first linked to partner violence nearly three decades ago when Rosenbaum and Hoge (1989) found that closed head injury was present in 61.3% of partner violence perpetrators (closed head injury refers to cases of injury in which there is no open wound or gash). With a larger sample, Rosenbaum and colleagues (1994) later replicated the findings with similar prevalence rates of head injury among male spouse abusers (51%). This rate was elevated when compared to the rates of head injury in non-violent maritally-discordant (25%) and maritally-satisfied (16%) men, all of whom were matched on demographic variables. Further, head injury was a unique predictor of IPV perpetration. The findings of elevated rates of head injury among male abusers compared to healthy controls have since been widely replicated (Cohen, Rosenbaum, Kane, Warnken, & Benjamin, 1999; Cohen et al., 2003; Marsh & Martinovich, 2006). However, one study comparing a head-injured group with a group of orthopedically (spine, joint, and muscle) injured men on the occurrence of IPV found no differences in rates of IPV between groups based on injury type (Warnken et al., 1994). Though there were no

differences in perpetration of IPV, head-injured men reported more factors that may predispose them to physical violence than did the orthopedically injured group (Warnken et al., 1994). For example, head-injured men reported increased loss of temper and control, more difficulty communicating verbally, arguing with others, and more verbal abuse when compared to the orthopedically injured men.

Prevalence, as well as severity of head injury and abuse has been examined across studies. Farrer, Frost, and Hedges (2012) conducted a meta-analysis to examine the association between traumatic brain injury and violence perpetration using six studies and over 222 perpetrators of IPV. The authors found that the weighted average of TBI among IPV perpetrators (53.6%) was significantly higher than that of the general population-based prevalence of TBI in men (38.5% of men by age 25; McKinlay et al., 2008). Additionally, Turkstra, Jones, and Toler (2003) compared 20 African American men convicted of domestic violence to men without criminal convictions matched for age, race, and socioeconomic status and found that while more than half of the participants in both groups had experienced TBI, the IPV perpetrator group had significantly more *severe* damage (as determined by a health history questionnaire).

In clinical populations, patients have demonstrated changes in both behavior and temperament. One study conducted with TBI patients found that 11% had increases in aggression and agitation following injury that were not explained by pre-injury characteristics (Brooke, Questad, Patterson, & Bashak, 1992). Importantly, agitation and frustration place stress on both caregivers and workers; these symptoms are often inappropriately treated with physical or chemical restraints that paradoxically increase frustration for patients (Alderman, 2003). Brooke and colleagues (1992) also observed restlessness (behavior interfering with staff or requiring staff action but less severe than aggression or agitation) in 35% of the patients, and suggested that for some, restlessness may be a normative experience of recovery. However, one study found that 31% of caregivers (mostly spouses) for individuals who sustained TBI reported moderate or severe “aggressiveness” toward themselves by 2 years post-injury (Hall et al., 1994). Further, caregivers reported more complaints with head-injured persons’ behaviors over time; specifically, they noted fatigue, slowness, and forgetfulness, as well as increasingly severe temper outbursts, anxiety, and self-centeredness over time. A similar study found that head injured patients as a group had higher reported anger and extreme violence scores than normal controls, and

relatives of the patients reported considerable distress with patient behavior (Grafman et al., 1996). Thus, in the immediate time following head injury roughly 1/3 of head-injured individuals display changes in temperament that produce disruptions for caregivers and attending staff. For both clinical and nonclinical populations, suboptimal treatment of TBI-related changes in function (e.g., personality, cognition, impulse-control, and behavior), may present a substantial obstacle to recovery (Kim, 2002).

Changes in aggression and violence perpetration have also been studied multiple years after the occurrence of head injury. A study of 300 inpatients at a brain injury center found that at any given period postdischarge (6, 24, and 60 months post-injury), 25% of individuals with moderate to severe TBI exhibited aggressive behavior (Baguley, Cooper, & Felmingham, 2006). In one study conducted with a large cohort of 520 head-injured patients from the Vietnam Head Injury Study, over one-fifth of veterans reported increases in violent behavior nearly 15 years after the initial trauma (Schwab, Grafman, Salazar, & Kraft, 1993). Proximal changes have also been explored by Tateno, Jorge, and Robinson (2003) who found elevated rates (33.7%) of aggression 6 months after injury among trauma patients. Aggressive behavior was also associated with the presence of major depression, poor premorbid¹ social functioning, history of substance abuse, and specific damage to the frontal lobes. Elevated rates of aggression were found in several additional studies (Alderman, 2003; Baguley, Cooper, & Felmingham, 2006; Kim, 2002), and the deficits in self-regulation of anger may reflect broader difficulties associated with post-concussion syndrome (Stevens, 1982).

Head injury is also associated with broad risk for criminal behavior among both violent and nonviolent individuals. One such study conducted by Diamond and colleagues (2007) screened 107 male and 118 female offenders from 6 federal prison sites across 3 prison security levels for traumatic brain injury and found that 88% of prisoners experienced at least one head injury incident. Another study conducted with a cohort of prisoners in New Zealand found that 86.4% of all offenders, both violent and nonviolent, had experienced at least one significant head injury, and 56.7% reported experiencing multiple significant head injuries (Barnfield & Leathem, 1998a; Barnfield & Learthem, 1998b). A meta-analysis including 15 studies (as well as the aforementioned studies in this paragraph) found that among all types

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¹ Pre-injury

of male offenders, the estimated prevalence of TBI was 64.41% (95% CI: 53.3 to 75.53; Shiroma, Ferguson, & Pickelsimer, 2012). León-Carrión and Ramos (2003) found similarly elevated frequencies of lifetime experience of multiple blows to the head between violent (61.1%) and non-violent (46.2%) offender groups. Elevated rates of head injury are also observed in community settings. Among offenders in community rehabilitation groups, half of nonviolent convicted felons reported head injury with co-occurring cognitive and emotional impairment, and 83% reported that their head injury occurred prior to their first encounter with law enforcement (Sarapata, Harmann, Johnson, & Aycock, 1998). Additionally, Martell (1992) conducted a study on maximum-security forensic inpatients and found that 22% had a history of severe head injury with loss of consciousness, and patients with brain impairment were more likely to be indicted for violent criminal charges.

The experience of head injury alone cannot fully explain the occurrence of violence, and researchers should examine functional impairment immediately following trauma. Though few studies began their focus on the association between head injury and aggressive behavior *prior* to the occurrence of TBI, several studies have highlighted the functional correlates of head injury that may predispose brain-injured individuals to violence, suggesting that executive functioning deficits may be the mediator through which violence occurs after TBI. Marsh and Martinovich (2006) explored domestic violence in the context of executive dysfunction and found that among perpetrators receiving treatment for partner abuse, individuals who experienced TBI had lower-IQs and poorer performance on verbal response initiation and suppression tasks, as well as non-verbal tasks such as visuospatial sequencing. Walling and colleagues (2012) examined the relationship of IPV to head injury, executive functioning, and intelligence in 102 male perpetrators and 62 nonaggressive men, and found that the most severely aggressive IPV perpetrators had significantly lower scores on a measure of verbal intelligence, suggesting impaired executive functioning.

Further, Brower and Price (2001) reviewed the impact of executive dysfunction on violent and criminal behavior across 18 studies and on a variety of neuropsychological tests. The authors found that executive dysfunction was generally associated with aggression, and in particular, focal orbitofrontal injury was significantly associated with increased aggression. As well, the authors noted that deficits in

executive function may increase the odds of future aggression, although no study has been conducted on TBI patients to test whether specific patterns of frontal damage predicts future violence. Though the direction of these effects cannot yet be determined, taken together, these findings suggest that head injury may predispose individuals to violent behavior, perhaps as a consequence of resulting deficits in executive function.

2.2 Summary

Overall, these findings suggest that head injury can lead to deficits in decision-making ability and regulation of affect, which may influence violent behavior. Though these deficits in functioning may directly produce aggression, it is also likely that observed aggression may be exacerbated by deficits in premorbid function. For example, Tateno and colleagues noted a number of potential premorbid risk factors of aggression following head injury, including poor social functioning and chronic substance use, suggesting a dual influence of premorbid vulnerability and head injury in aggressive outcomes. Additionally, changes in personality, cognition, and behavioral functioning often occur after head injury and increase over time. Specifically, authors noted increases in restlessness, agitation and anxiety, fatigue, slowness, forgetfulness, and temper outbursts over time (Brooke et al., 1992; Hall et al., 1994). Moreover, inadequate strategies to treating TBI-related symptoms may negatively impact recovery and increase an individual's propensity towards aggressive behavior.

However, there are important limitations of head injury studies, including a general lack of prospective work as well as inadequate controls in many studies for developmental risk factors such as prior history of violence, socioeconomic status, stability of employment, and substance use or abuse (Brower and Price, 2001). As Pinto and colleagues (2010) noted, despite the elevated rates of head injury (between 40-60%) among IPV perpetrators, it is unclear whether head injury directly influences partner violence perpetration because of the long intervals between injury and assessment. For example, while Rosenbaum et al (1994) found an association of head injury and IPV, a large percentage of the men had experienced their head injury at least fifteen years prior to testing. Additionally, individuals tend to report head injuries that led to medical attention, and subconcussive head injuries are not taken into account in these studies. Further, the association between TBI and IPV may be confounded by alcohol use, history of

aggression, and retrospective methods of studying head injury (Farrer, Frost, & Hedges, 2012). Prospective studies are needed to elucidate the factors that influence aggression in both the acute and chronic stages of head injury recovery.

Despite these limitations, there appears to be a significant impact of head injury on violent behavior, as demonstrated in the increased prevalence among offender groups found in both meta-analyses (e.g., Farrer, Frost, and Hedges, 2012) and studies using matched controls for age, race, and socioeconomic status (e.g., Turkstra, Jones, and Toler, 2003). However, we cannot make conclusions about the specific regions of activity that mediate violent behavior because damage in the instance of head injury is often widespread or a combination of acute versus permanent effects. Consequently, lesion studies may offer a more targeted opportunity to understand the specific functional correlates of violence perpetration and increases in aggression.

3.1 Lesion Studies and Frontal Lobe Deficits

A lesion is any abnormality or damage in the tissue of the brain that results from disease or trauma. While head injury is often a precipitant of brain lesions, we make a semi-artificial distinction in that lesions often result in confirmed loss of focal (specific) regions, and includes multiple reasons for abnormalities beyond trauma (both accident and illness). Several studies have demonstrated links between the occurrence of focal lesions and changes in trait aggressiveness. Perhaps the most famous case is that of Phineas Gage, an individual who sustained massive damage to his frontal lobes, resulting in an inability to uphold workplace responsibility and an increasingly short temper (Harlow, 1869). Since then, several authors have provided evidence that frontal lesions (specifically the dorsolateral prefrontal cortex, ventromedial prefrontal cortex, orbitofrontal cortex, and medial frontal cortex) are associated with anger, irritability, and violence (Blair, 2003; Blair & Cipolotti, 2000; Damasio et al., 1994; Grafman et al., 1996; Seguin, 2009). Specifically, Brower and Price (2001) reviewed studies of frontal lobe function in aggressive and antisocial subjects and found strong evidence for a link between both broad frontal lobe dysfunction and focal orbitofrontal injury and increased violent behavior. Bechara, Damasio, and Damasio (2000) found that patients with inferior frontal lobe damage have an impaired ability to appreciate the future consequences of their actions, a risk factor of impulsive violence. Additionally,

Anderson and colleagues (1999) studied two adults who obtained focal damage to their prefrontal cortices before 16 months of age, one due to accident and the other as a product of tumor resection. Similar to individuals with prefrontal cortex damage obtained in adulthood (Shallice & Burgess, 1991), as adults, the two cases had remarkably poor social functioning compared to normal cognitive abilities, and deficits included failure to understand punishment contingencies. However, the early-onset subjects were more severely impulsive, and more physically and verbally violent against persons and property (Anderson et al., 1999).

Interestingly though, there is evidence that right ventromedial prefrontal cortex (VMPFC) might play a unique role in decision-making and emotional processing. Tranel, Bechara, and Denburg (2002) conducted a study comparing individuals with focal, stable unilateral lesions to the right or left amygdala, and found that the right-damaged individuals exhibited marked disturbances in social and interpersonal behavior and had reduced anticipatory skin conductance during a gambling task involving risk and reward decisions. Additionally, most of the right VMPFC lesioned individuals met criteria for “acquired sociopathy,” a combination of critical deficits in understanding and responding to punishment, inability to learn from previous mistakes, and blunted negative emotional affect. The left VMPFC lesion group showed markedly different results. Specifically, they performed normally during experimental tasks, having normal emotional processing, social and interpersonal behavior, and relatively unchanged personal traits compared to premorbid functioning. The findings of the right VMPFC-damaged group can be placed in context with the “somatic marker” hypothesis proposed by the same research group (Damasio, Everitt, & Bishop, 1996; Damasio, Tranel, & Damasio, 1991), which asserts that right VMPFC lesions result in deficits because of an inability to recruit emotionally salient information when making decisions with some uncertain outcome, producing a tendency to favor immediate rewards.

These observed deficits, as well as the propensity for obtaining damage to frontal areas after head injury illustrate the importance of frontal areas in modulating aggression and violence. However, frontal lesions do not systematically produce aggression or violence (Séguin, 2009), and developmental background is important because lesions, like head injury, are often “acquired,” or caused by events after birth rather than by a genetic or congenital disorder, and disrupt otherwise normal functioning.

Séguin(2009) reviewed the associations between frontal lobe lesions and violent behavior, and argued that while frontal lesions do not directly cause increases in aggression and violent behavior, they often produce marked impairment in psychosocial functioning, emotional decision making, and attending to informational cues that may increase the propensity towards aggression and violence under conditions of frustration.

Another set of areas that have received attention in studies of violence and aggression are the temporal lobes. Tonkonogy (1991) performed computerized tomography (CT) and magnetic resonance imaging (MRI) scanning on 23 patients with organic mental syndromes and found that 36% of patients who exhibited violent behavior had focal lesions in the anterior-inferior temporal lobe. The author asserted that damage to this area might produce violence in combination with damage to the adjacent amygdala, and subsequent stimulation of preserved limbic structures through mechanisms such as kindling². Additionally, one study compared individuals with histories of extremely violent behavior to healthy control subjects and found that while structural MRI scans of the violent individuals yielded normal results, PET scans showed metabolic deficiencies in the temporal lobes (Seidenwurm, Pounds, Globus, & Valk, 1997). Thus, through these examples there are emerging links to underlying temporal dysfunction and aggressive behavior.

3.3 Summary

Previous studies have supported an association between focal (confined) lesions and aggressive and violent behavior. Overall, the findings from head injury and lesion studies of aggression and violence suggest that the mechanisms of impairment point to specific regions of the brain, including: prefrontal and connected subcortical areas, temporal areas and their associated limbic structures, and generalized or extensive damage across regions of the brain (Golden, Jackson, Rohne, & Gontkovsky, 1996). Séguin (2009) reviewed the associations between frontal lobe lesions and aggression, arguing that lesioned individuals show poor performance on cognitive tasks that assess reward and punishment. It is theorized that this may occur because of disruptions in the limbic circuits, which disrupt emotional processing, thereby leading a lesioned individual to make a decision without appropriate emotional input, and thus

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² a seizure that occurs and is subsequently made more likely after a different brain event

increase the likelihood of conflict escalation and physical violence. These results also highlight the potential for equifinality in that damage to any of multiple areas may produce the same outcome of physical violence and aggression.

Deficits in emotion regulation and executive control might produce violence after provocation, such as those observed in early lesion patients (Anderson et al., 1999) who fail to both control their emotional expression and understand the impact of their behavior. Lesions can also produce profound impairment later in life, and produce marked personality changes that disrupt otherwise healthy social and interpersonal behavior (Bechara, Damasio, & Damasio, 2000; Damasio et al., 1994; Harlow, 1869), which suggests that the studied individuals were not developmentally more likely to be violent. From these studies, as well as those included on head injuries, there is notable evidence that “acquired” injuries to frontal areas of the brain are associated with a greater propensity towards violent behavior. Moreover, as Anderson and colleagues (1999) observed, there is also evidence that prefrontal lesions occurring earlier in life produce more marked and widespread impairment than those occurring at later ages, perhaps because they additionally affect an individual’s developmental trajectory.

It is also important to note that lesions to the frontal areas of the brain do not singularly produce violence, but rather create deficits in core executive processes such as emotion regulation (Damasio, Tranel, & Damasio, 1991), decision making (Tranel, Bechara, & Denburg, 2002), social functioning, and understanding punishment contingencies (Bechara, Damasio, & Damasio, 2000; Shallice & Burgess, 1991) that can inherently increase the risk for violence perpetration. Moreover, we are limited in our conclusions about the brain regions of activity associated with violence and aggression from lesion studies alone, as individuals with lesions in isolated regions are preferred as study participants, and it is unclear whether or not lesions affecting multiple areas would produce more frequent or more severe violence. Accordingly, results from head injury and observations from lesion studies should be examined in the context of neuroimaging of individuals without lesions to better identify the brain regions associated with violent behavior.

4.1 Imaging Studies

In beginning this section, it is important to note that the correlates of violent behavior observed in neuroimaging studies may have different developmental origins than the individuals observed in studies of traumatic brain injury. For example, genetic variants such as the functional polymorphisms in monoamine oxidase A (MAOA) and the serotonin transporter (5-HTTT) influence individuals from the early stages of life and have been linked to limbic system abnormalities in aggressive individuals (Pavlov, Chistiakov, & Chekhonin, 2012). Since these genetic risk factors lead to such profound developmental alterations in neurocircuitry and can additionally influence the environment that an individual experiences, it is unclear if these genetic variants partially mediate the influence of head injury in violent behavior. Thus, to address this ambiguity, we proceed to consider the structural abnormalities associated with human aggression, with the caveat that they are likely a more complex amalgamation of vulnerabilities than focal brain injuries.

There is some evidence that the presence of observable brain abnormalities is associated with violent behavior (Hoptman, 2003). While imaging studies conducted with partner-violent samples alone are considerably limited, structural abnormalities have been studied in abusive individuals with comorbid alcohol dependence. Zhang and colleagues (2013) used volumetric magnetic resonance imaging (MRI) to compare volumes of structural components of the prefrontal cortex and 6 subcortical structures among three groups of men: partner violence perpetrators with alcohol dependence (IPV-AD), non-violent alcohol-dependent men, and a group of healthy controls. When the selected brain areas were compared, no differences were found in cerebrospinal fluid, gray matter, white matter, total brain volume, or intracranial volume for any groups. Interestingly, the IPV-AD group had smaller right amygdalae (both raw volume and adjusted) than both the nonviolent ADs and healthy controls. However, partner violence was not necessarily explained by structural differences alone, and the authors found that IPV-ADs started drinking earlier than the NV-AD men, and had greater prevalence of comorbid personality disorders.

Regarding general violence and aggression, Aigner and colleagues (2000) used MRI scans coded for presence or absence of brain abnormalities to examine high security prisoners, excluding those with neurological disease, psychotic disorder diagnosis, or severe organic mental disorders. The authors found notable differences between the highly violent and low violent offenders: 65.5% of the high violent group

had identifiable MRI abnormalities compared to only 16.6% of the low violent group. There is also evidence that general reductions in tissue volume in frontal areas are linked to trait aggression and antisociality (Raine et al., 2000), a finding supported by observations with lesion patients (e.g., Brower & Price, 2001).

Other structural abnormalities have been explored extensively due to their broad connections to functions hypothesized to be related to violence. More specifically, because the amygdala is associated with emotionally-influenced memory (Adolphs, Tranel, Damasio, & Damasio, 1994; Phelps, 2004) and fear conditioning (Phillips & LeDoux, 1992), it has been an area of interest for violence researchers. Reduced amygdala volume has consistently been implicated in the development of trait aggressiveness and perpetration of violence. In one sample of 56 adult men with varying histories of violence recruited from a larger sample of 500 subjects studied in first grade, Pardini and colleagues (2014) found that individuals with lower amygdala volume exhibited higher levels of aggression and psychopathic features from childhood to adulthood. Low amygdala volume also predicted adult perpetration of violence and levels of aggression in a 3-year period, even after controlling for childhood levels of aggression, violence, and psychopathic traits. Moreover, there is additional evidence among community samples that low amygdala volume is connected to lifetime history of aggression. One study conducted using morphometric brain scans in 20 volunteers found that high lifetime aggression scores (as measured by the Lifetime History of Aggression Assessment, LHA; Coccaro et al., 1997) were associated with 16%-18% smaller amygdala volumes, and the authors found a highly significant negative correlation between right ($r = -0.650, p = 0.003$) and left ($r = -0.630, p = 0.007$) amygdala volume and trait aggression (Matthies et al., 2012). These results suggest that lower amygdala volume is associated with trait aggression, antisociality, and sensation-seeking, all of which may indicate the presence of functional impairments in fearlessness and arousal regulation (Raine et al., 2000). Despite the number of studies showing differences in amygdala volumes in aggressive individuals, one study did not find differences in volumes of the amygdala between offenders and healthy men. However, this study conducted by Tiihonen and colleagues (2008) did find evidence for a wider array of structural abnormality. The researchers compared the brain anatomy of repeat violent offenders with antisocial personality disorder (ASPD) and comorbid alcohol

dependence to a comparison group of healthy men. Only one of the 26 violent offenders had a history of head injury, a finding that is somewhat unusual in and of itself as reflected in the research reviewed herein, and none had a diagnosis of a mental disorder beyond ASPD or a behavioral disorder caused by previous damage. The authors found evidence for widespread abnormalities in the offender group, such that the offenders had larger white matter volumes in the occipital and parietal lobes, and left cerebellum. The volumes differences were even greater between the healthy men and the subgroup of offenders with diagnoses of psychopathy. The offenders also had reduced gray matter volume in the postcentral gyri, frontopolar cortex, and orbitofrontal cortex as compared with healthy men, and the reductions were greater for the psychopathy group. Similar results were also obtained in two studies of adolescent boys with conduct disorder (Kruesi et al., 2004; Sterzer et al., 2005). The authors interpreted these differences in the context of functional imaging studies, and suggested that the frontal cortex, and orbitofrontal cortex specifically, are critical for the regulation of violent and aggressive behavior (as Grafman et al., 1996 also reports), and that malfunctioning in the area produces antisocial behavior. As well, the larger regional volumes in posterior areas were proposed as a sign of early neurodevelopmental abnormalities that may have jointly influenced stable antisocial behavior and proclivity to substance abuse.

Researchers have also attempted to identify differences between alterations in gray matter volume associated with antisocial behavior and those of lifelong substance abuse in order to identify regions of specificity. In one study conducted in Germany, male participants were recruited from prisons, hospitals and communities and underwent structural magnetic resonance imaging (Schiffer et al., 2011). When the researchers compared violent offenders to non-offenders, they found that violent offenders had larger gray matter in the amygdala bilaterally, the left nucleus accumbens, and the right caudate and less gray matter in the left insula. These results are potentially conflicting with previous findings of reduced gray matter volumes in the amygdala (Pardini et al., 2014; Zhang et al., 2013). However, the authors argued that previous studies did not attempt to disentangle the structural brain correlates of substance use and violence, and suggest that the structural alterations associated with violent behavior may be increased gray matter volume in the mesolimbic reward regions, whereas those associated with substance use are smaller gray matter volumes in the OFC, VMPFC, and premotor cortex. While the attempt to distinguish

between substance use disorders and repeated violence is a promising concept, the findings of Schiffer and colleagues (2011) have yet to be replicated.

Researchers have also utilized the results from lesions studies to examine the potential loci of dysfunction for violent psychiatric patients. Best, Williams, and Coccaro (2002) examined the orbital/medial prefrontal cortex circuit (OMPCC) and its connection to the amygdala after observations of increased impulsive aggression from individuals with acquired lesions (i.e. Anderson et al., 1999; Davidson, Putnam, & Larson, 2000). The authors hypothesized that individuals with a diagnosis of Intermittent Explosive Disorder (a psychological disorder characterized by impulsive aggression) would perform poorly on tasks sensitive to lesions in the OMPCC. Indeed, the authors found support for their hypothesis, as IED patients demonstrated marked impairment on decision making, facial emotion recognition, and odor identification. Conversely, the IED patients showed preserved ability on control tasks measuring other areas of function such as working memory, which is thought to be mediated by the dorsolateral prefrontal cortex and posterior association cortices. Moreover, as the IED patients displayed a negative bias towards neutral emotional faces during recognition task, these results may explain the hallmark facet of IED of aggression that is out of proportion to provocation. Taken in context with lesion studies of the OMPCC (Anderson et al., 1999), these findings suggest that disruption to the circuit can result in deficits in decision making ability and emotion recognition, which may often lead to violence and aggressive behavior. Importantly, these findings also illustrate the potential for multiple developmental pathways to produce a similar outcome of behavioral and cognitive deficits.

Bufkin and Luttrell (2005) reviewed neuroimaging studies of aggressive and violent behavior in the broader context of emotion regulation. Although the authors did not distinguish between medical and criminal samples, their review of 17 studies had several notable findings. In particular, the authors found support for the complexity of neural circuitry underlying regulation of negative affect. The authors noted that violent offenders exhibited deficits in parietal cortex, which has a role in the integration and interpretation of sensory information (both visual and auditory; Kalat, 2001). As this connected system is critical in linking sensory experiences with emotion, the authors hypothesized that dysfunction may lead to aggressive reactions in ambiguous situations (Hirano et al., 2000; as cited by Bufkin & Luttrell, 2005).

These findings gain support from a study conducted by Soderstrom and colleagues (2000) who investigated 21 offenders convicted of impulsive violent crimes and examined both functional and structural abnormality. The MRI results revealed no corresponding structural damage compared to a matched group of healthy controls. However, functional neuroimaging techniques revealed reduced cerebral blood flow in the right angular gyrus (a portion of the parietal lobe) and right medial temporal gyrus, bilaterally in the hippocampus, and in the left white frontal matter. Additionally, the offenders had increased cerebral blood flow in the parietal association cortex. This evidence illustrates the theory that structure and function may not be correlated in a given group (Mills & Raine, 1994) in that individuals may have functional anomalies in absence of structural deficits.

4.3 Summary

Overall, this evidence suggests that the orbitofrontal cortex seems to be critical in predisposing individuals to violence (Best, Williams, & Coccaro, 2002; Corvo & Johnson, 2013; Raine et al., 2000; Séguin, 2004; Tiihonen et al., 2008). In addition, the finding of reduced gray matter in this area gains support from the results of early-onset lesion studies (Anderson et al., 1999), in which the patients had similar impairments in social functioning and decision-making ability. There is also evidence that severity of physical violence and degree of brain abnormality covary (Aigner et al., 2000), and general reductions in tissue volume are associated with aggression (Raine et al., 2000; Tiihonen et al., 2008). Further, taken with the findings reviewed previously herein, the sample characteristics of one study (Tiihonen et al., 2008) conducted with offenders without diagnosed personality disorders and low occurrence of head injury suggest that widespread abnormalities in gray matter tissue volume may represent one marker for violence that has separate a separate etiological course than other “acquired” risk factors such as forms of isolated brain damage, though they demonstrably produce similar impairment.

There is also extensive evidence that low amygdala volumes correlate with violence perpetration. The only structural neuroimaging study conducted with partner offenders specifically found smaller right amygdala volumes compared to both healthy controls and a group of nonviolent alcohol dependent men. Smaller amygdala volumes were also found in community samples (Matthies et al., 2012) as well as in

studies of aggressive individuals from childhood to adulthood (Pardini et al., 2014). Raine and colleagues (2000) found that arousal deficits commonly found in offenders are associated with traits of fearlessness, antisociality, and sensation seeking, which may be related to amygdala abnormalities. There is some evidence that does not support the association between low amygdala activity and violence, as one study found the reverse result in that larger gray matter volumes were associated with aggression (Schiffer et al., 2011) and another found no differences between violent and nonviolent groups (Tiihonen et al., 2008). These results may be explained by the concept of equifinality- the end state of violence perpetration may be produced by a number of etiologically separate vulnerabilities. However, theoretical explanations of amygdala damage suggest that functional disruption may operate in tandem with prefrontal areas. As Best and colleagues (2002) asserted and as previously observed deficits in lesion patients support, damage to the integrity of the orbital/medial prefrontal cortex circuit (OMPCC), which includes sections of the prefrontal cortex and the amygdala, may predispose individuals to aggression and violent behavior. In turn, since this functional system is responsible for projecting inhibitory responses from the prefrontal cortex to the amygdala, damage to either area may secondarily produce violence due to insufficient response inhibition.

5. Conclusions and Recommendations

Over two decades ago, Mills and Raine (1994) reviewed 20 brain imaging studies conducted on violent and sexual offending individuals and presented two seemingly alternative positions, one that frontal lobe dysfunction was associated with violent offending and temporal lobe dysfunction was associated with sexual offending. While the scope of this review is limited to violent offending and thus is unable to shed light on this second proposition about sexual offending, Mills and Raine (1994)'s second position is worth discussing. The authors argued that temporal or frontal areas may predispose to offending, with the type of offending likely being determined by non-biological factors. Though the authors compared different modes of violence (violent, sexual, or both) than the present review, this hypothesis seems to be supported for partner violence, as well as general violence and aggression. As Corvo and Johnson (2013) argued, and as the heterogeneity of regions of abnormality observed in

neuroimaging studies support, violence can emerge from a variety of multiple pathways, with exposure or biological risk interacting with resiliency or mediating environmental factors and combine to produce violent behavior. In this context, partner violence and general violence may emerge from similar vulnerabilities, and the mediating environmental factors may determine the context of pathological expression.

Overall, the evidence presented herein mirrors results from functional imaging approaches exploring regions of connectivity, and suggests that violence can involve some form of disruption of the fronto-temporolimbic circuits (Best, Williams, & Coccaro, 2002; Davidson, Putnam, & Larson, 2000; Hoptman, 2003). Importantly, as Best, Williams, and Coccaro (2002) note, the functional difference between a developmentally shaped abnormality observed in neuroimaging studies (e.g., smaller right amygdala volume) compared to acquired damage obtained from lesions or head injury can be profound, as developmental differences are slower changes that often allow for increased time for neural reorganization. As observed in the influential work by Anderson and colleagues (1999), abrupt damage to the prefrontal region may result in severe and chronic social maladjustment despite intact cognitive abilities. Importantly, the relative heterogeneity of regions of abnormality obtained in neuroimaging results compared to lesion and head injury studies highlights a potential increased threshold for damage needed to produce aggressive behavior. In other words, it is possible that in the case of developmental abnormalities obtained in neuroimaging studies, more widespread (or similarly profound) damage is needed to result in the same behavioral manifestation of violence perpetration.

Considering the potential equifinality of violence, this review has elucidated the general structural vulnerabilities that may predispose an individual to violent behavior. There is strong evidence from head injury, lesion, and imaging studies that damage or reductions in frontal areas of the brain and connected regions are associated with violence and aggression. Similar correlates were also obtained in Yang and Raine (2010)'s meta-analytic review of combined functional and structural imaging studies of individuals with antisocial, psychopathic, or aggressive traits. However, as our review was limited to studies of violent individuals without comorbid medical diagnoses, this review presents the first evidence of the structural correlates of violent behavior per se. As our conclusions present similar brain regions

associated with violent behavior as those found among individuals with social conduct deficits (Damasio, Tranel, & Damasio, 1990), decision making and emotional processing(Tranel, Bechara, & Denburg, 2002), substance abuse (Goldstein & Volkow, 2002), and traits such as antisociality and psychopathy (Yang & Raine, 2010), it is possible that prefrontal dysfunction can elicit a broad range of behavioral and temperamental difficulties, with the specific manifestations determined by non-neurological environmental or genetic factors.

Importantly, our review has elucidated the role of lesion studies in pinpointing potential loci of interest for violence and aggression. From these studies, it also seems possible that highly charged emotional situations can impair otherwise normal problem solving skills. Davidson, Putnam, and Larson (2000) posited that damage resulting in violent behavior could stem from disrupted communication in the circuit involving the orbitofrontal cortex and interconnected structures (including the anterior cingulate, and the amygdala), and in turn lead to an inability to constrain reactions to negative emotional behavior. This result was also supported by Best, Williams, and Coccaro (2002)'s work with patients with Intermittent Explosive Disorder (IED), a form of psychopathology characterized by impulsive aggression. Further, Best and colleagues (2002)' theory of disruption to the orbitofrontal cortex and interconnected structures such as the amygdala find support in a number of head injury, lesion, and neuroimaging studies of violence and aggression (Brower & Price, 2001; Grafman et al., 1996; Pardini et al., 2014; Raine et al., 2000; Tiihonen et al., 2008; Tranel, Bechara, & Denburg, 2002; Zhang et al., 2013). In turn, as several studies focused on specific regions of dysfunction within the prefrontal cortex and connected structures, it seems that damage to any portion of this interconnected circuit may increase an individual's propensity towards aggression.

One limitation of the present review is a lack of attention to the functional networks that encompass the aforementioned that contribute to violent and aggressive behavior (Corvo & Johnson, 2013). However, we believe that before cause-effect relationships can be determined between structural damage and functional impairment, more work needs to be done with developmentally at-risk individuals (i.e. those with lesions or TBI) to determine if the dysfunctional neural networks that produce violent behavior are the same as for those who have more of a developmental risk. Both genetic risk and

environmental factors are important in the exploration of factors that lead to violence, and one study of 2,925 adult twins found evidence for a 2-factor common pathway model, with heritability for aggressive behavior as assessed with the Lifetime History of Aggression questionnaire (LHA; Coccaro et al., 1997) ranging from .37 to .57 and nonshared environmental effects ranging from .43 to .65 (Yeh, Coccaro, & Jacobson, 2010). Thus, symptom onset, severity, and course may be different for individuals with acquired versus developmentally-shaped abnormalities, and even among twins both genetics and the environment are considerable determinants in the propensity for lifetime aggression. Future studies that combine structural and functional imaging are needed on those populations as well as offender groups in particular to determine if regions of the brain previously linked to aggressive behavior are functionally specific.

The present review presents a number of implications for future studies of brain abnormalities and violence. There is a critical need for longitudinal assessment of acute head injury, beginning with initial hospitalization, in order to understand whether acute or long-term changes in functioning following damage are more important for predicting subsequent violent or aggressive behavior (Bowman, 1997). Importantly, acquired brain injuries produce neurological changes in affected individuals that in turn place new limits on physical and psychological functioning post-injury (e.g., Alderman, 2003; Kim, 2002). Such changes can produce a new set of challenges for brain-injured persons that can be a source of frustration in individuals, whether or not they were aggressive pre-injury. As we have yet to fully characterize these changes over time, theoretical models of violence (two-subtype model, partner-only vs. generally violent) should be incorporated into imaging studies and studies of individuals with focal brain damage to determine if profiles of violence are neurofunctionally distinct. Imaging techniques such as diffusion tensor imaging (DTI) that have the potential to capture disruption of neural axons can further elucidate the interplay between these regions of function, and should be a focus for future studies. Further, conceptual issues that have been discussed historically still apply to brain imaging studies, and neuroimaging studies should integrate data on social and cognitive risk factors, accounting for recidivism, and exploring comorbidity with substance use and among psychiatric populations (Mills & Raine, 1994).

In addition to the need for prospective studies of head injury and longitudinal assessment of brain abnormalities and violence, future studies should consider potential gender differences in brain and behavior outcomes after TBI or brain lesions. Many of the studies included in the present review were conducted with only male populations, or did not investigate gender differences in their samples. For example, out of the 20 studies of head injury reviewed herein, only 7 included women in their samples. Further, only 4 studies included in this review examined potential gender differences, and only 2 conducted statistical comparisons. Both studies (Baguley, Cooper, & Felmingham, 2006; Tateno, Jorge, & Robinson, 2003) found no relation between gender and aggressive behavior. Additionally, Shiroma, Ferguson, and Pickelsimer (2012) created pooled prevalence estimates across samples and found similar rates of TBI across male (64.41%) and female (69.98%) offenders. However, authors have noted that the similarities in rates of head injury and severity of aggressive behavior may be due to the underrepresentation of women in TBI research (Baguley, Cooper, & Felmingham, 2006). Another study conducted by Diamond and colleagues (2007) noted that more women reported head injury by assault than men (46.6% vs. 27% of all injuries), and men reported losing consciousness for longer than women following head injury. Despite finding associations between lifetime history of head injury and multiple measures of cognitive and behavioral functioning, the authors did not test for differential effects by gender. Thus, though previous research has highlighted *some* gender differences in type of head injury, researchers have yet to accurately compare the association of head injury and aggression in male and female samples.

There are several important limitations to the current trend of adopting a gender-insensitive approach. In particular, it is unclear whether brain dysfunction is associated with the same patterns of aggression and violence in both men and women. It is also unclear whether men and women have the same likelihood of exhibiting aggressive behavior after similar brain injury or with the same pattern of structural deficits. These differences can offer important targets for intervention, and should be fully explored in future research.

Last, this information presented in this review also has implications for clinicians and emergency responders. As Stern (2004) noted, traumatic brain injury is both an effect and cause of violence and

abuse, and should be properly identified by medical professionals. Proper screening for physical abuse, especially when it occurs to children, is needed to limit secondary damage and prevent recurrence.

Unfortunately, many victims ultimately become abusers themselves because so many cases of physical abuse are missed during head injury evaluation (Stern, 2004). Emergency room screenings of head trauma should include questionnaires for both aggression and victimization to better inform subsequent medical and therapeutic attention. As there is evidence that anger management programs following TBI are effective in reducing both trait and state anger (Walker et al., 2010), proper screening and referral can be effective in reducing violent behavior for individuals with brain injury.

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