FRONTAL LOBE DYSFUNCTION AND AGGRESSION: CONCEPTUAL ISSUES AND RESEARCH FINDINGS

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ABSTRACT. The notion that there is a relationship between frontal lobe damage and aggression has been part of clinical lore for more than 50 years. However, although there is evidence for an association between general brain dysfunction and aggressive behavior, much of the evidence for a specific relationship between frontal lobe dysfunction and aggression has consisted of case reports. This article begins with a discussion of normal frontal lobe (i.e., executive) functions, followed by a description of the types of problems that result from frontal lobe damage. Conceptualization of the ways in which these frontal lobe deficiencies may be related to violence are offered, and relevant research regarding this relationship is reviewed.

KEY WORDS. Aggression, executive functions, frontal lobes, brain damage, violence

THERE IS COMPELLING EVIDENCE of an association between brain dysfunction and aggressive behavior. Reports indicate that as many as 70% of patients with traumatic brain injury have displayed sufficient irritability and aggression to cause significant distress to their families (McKinlay et al., 1981). Even among psychiatric patients with personality disorders not known to have sustained a brain injury, the occurrence of neurological “soft-signs” (e.g., involuntary movements or sensory-perceptual aberrations in the absence of gross neurological damage) has been related to aggressivity (Stein et al., 1993). Based on their daily experiences, mental health and rehabilitation professionals are likely to perceive a relationship between poor emotional and behavioral control (ranging from irritability to indiscriminate violence) and brain damage.

These clinical findings and perceptions, coupled with the seemingly “senseless” nature of much criminal violence, have prompted research into the possible brain-dysfunction underpinnings of violent criminal behavior. One study demonstrated that neuropsychological measures outperformed personality measures in distinguishing 40 violent from 40 nonviolent male delinquents, correctly classifying 95% of these offenders (Spellacy, 1978).

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However, although the violent delinquents obtained lower cognitive scores, it is unclear whether this was the result of brain damage. Most of the violent offenders obtained scores in the low average to average ranges on many of the tests administered—scores that would be obtained by many individuals leading normal and adaptive lives.

Nevertheless, similarly suggestive findings have been reported by other investigators. For example, Brickman, McManus, Grapentine, and Alessi (1984) found neuropsychological abnormalities to be pervasive among offenders tested in residential settings (see also Hurwitz, Bibace, Wolff, & Rowbotham, 1972). Similarly, Yeudall and colleagues (cited in Brickman et al., 1984), in a series of studies of offenders, reported a high frequency of frontal and temporal abnormalities coupled with a disproportionate history of head injuries, blackouts, and unconsciousness. In addition, Krynicki (1978) found greater similarities between assaultive delinquents and organic brain syndrome patients than between assaultive delinquents and their nonassaultive delinquent peers. Frontal electroencephalogram (EEG) abnormalities, visual-motor perseveration, short-term memory deficits, and more prevalent ambidexterity were distinguishing features among the violent. Berman and Seigal (1976) have also reported neuropsychological abnormalities in delinquent and criminal populations.

Among the most striking of findings are those of Lewis and her colleagues in studies of death row inmates. All 15 death row inmates examined by Lewis et al. (1986) had histories of severe head trauma. Five displayed major neurologic impairment, while seven others demonstrated neurological soft signs. Similar findings were reported for juveniles condemned to death in the United States (Lewis et al., 1988).

Spellacy (1978) has argued that violence in these populations is just one of a variety of behaviors that reflect poor impulse control, along with such behaviors as immature attention-seeking, ego-centeredness, and careless automobile driving. While a tendency to behave impulsively across a variety of situations could be interpreted as evidence of brain compromise, it may also be simply attributed to personality pathology. Demonstrations of a high level of brain compromise among offenders does not prove a causal connection between brain dysfunction and criminality in general, or violent behavior in particular. There are, however, theoretical reasons for predicting such a connection.

**TEMPORAL LOBE DAMAGE, EPILEPSY, AND AGGRESSION**

Much attention has been devoted to the possible role of damage to a phylogenetically ancient system in the brain responsible for emotional experience, the limbic system, in the genesis of seemingly uncontrollable violence. The limbic system is the source of drives critical to survival behavior, such as seeking food, sex, and preparing for fight or flight in response to threats or competition for life-sustaining resources. Elements of the system (the amygdaloid complex and hippocampus) reside in the temporal lobes, where damage may result in a condition known as temporal lobe epilepsy (TLE). Some researchers link TLE to intensified feelings of anger or irritability (Bear et al., 1985).

The ideas that seizures cause uncontrollable violence may be particularly attractive to some individuals facing charges for violent offenses (and their lawyers). The appeal is accentuated by the association between temporal lobe damage and memory impairment; events during a seizure may not be recalled by the individual, allowing for plausible denial of recall for an episode of violence. However, relatively few violent episodes occur during ictal episodes (seizures) per se (Silver & Yudofsky, 1987). And the overall relationship between aggression and epilepsy, regardless of the locus of seizure activity in the brain, is controversial (Sbordone, 1993; Silver & Yudofsky, 1987).
Frontal Lobe Violence

The strong interest in limbic damage as an explanatory mechanism partially reflects the intuitive connection between a primitive system running amok and apparently senseless violence. However, aggressive and violent behavior may also, on occasion, be explained with reference to the brain structure at the other end of the phylogenetic pole—the prefrontal cortex.

The Prefrontal Cortex

In *Homo sapiens*, the prefrontal cortex is highly developed and responsible for many of the most advanced and “human-like” of abilities. The frontal lobes play a primary role in the planning, initiation, integration and implementation of complex behavioral acts that are labeled as “executive” by neuropsychologists (Hall, 1993; Lezak, 1995). Both the biological substrata and their complex mental and behavioral products are relatively fragile.

This article outlines ways in which prefrontal and executive impairment may, theoretically, be related to violence. We begin with a discussion of normal executive functions, followed by descriptions of the types of problems that result from damage to the frontal lobes. We then explore ways in which these deficiencies may be conceptually related to violence, and review relevant research regarding this relationship.

WHAT DO THE FRONTAL LOBES DO?

Given the contributions of the frontal lobes to the most human-like of qualities, the neuropsychological literature is replete with rich—often poetic—descriptions of frontal activities. The following categorization attempts to capture the essential elements of the orchestration of conduct by the frontal lobes. The divisions employed are inevitably somewhat arbitrary, as the functions described are seamless, reciprocal, and highly integrated in the normal state.

Directing and Maintaining Higher-Level Attention

Luria viewed the frontal lobes as the directing and controlling source of the brain—the place where awareness is translated into action (Hall, 1993). They are key in regulating the active state of the organism (Hecaen & Albert, 1975), in deciding what to attend to (Golden, Jackson, Peterson-Rohne, & Gontkovsky, 1996), and in sustaining or switching attention when desirable.

Correlating Internal and External Information

The frontal cortex is extensively connected to the limbic system as well as to cortical association regions, thereby receiving both emotional and higher-order information (Nauta, 1971). Material from all sources (e.g., internal and external, conscious and unconscious, memory storage and visceral arousal centers) is integrated into ongoing activity (Lezak, 1995) so that behavior can be modulated to satisfy drives within the constraints of the internal and external environments (Golden, Jackson, Peterson-Rohne, & Gontkovsky, 1996).

Generating Intentions, Plans, and Programming Activity

Based upon a constant monitoring of internal needs and external demands and possibilities, the frontal lobes control the essential elements of intentions (Hecaen & Albert, 1975). Planning and programming of complex activity is required to enact these intentions, and to provide continuity and coherence of behavior across time (Golden et al., 1996). Anticipation, sequencing, and foresight are related notions.
**Initiation, Monitoring, and Adapting Behavior**

Even the most rudimentary of intentions requires translation into action, including simply starting or stopping specific behaviors. Plans must be implemented and progress toward goals monitored so that ongoing adjustments can be made.

**Common Symptoms and Deficits Associated with Frontal Lobe Damage**

Damage to the frontal systems results in consequences that are diverse, multifaceted, and often catastrophic. The resulting deficits can include apathy; emotional lability; anticipating, planning, and sequencing deficits; deficiencies in initiating behavior; deficits in monitoring behavior; problems in shifting, adapting, and stopping behavior; and deficient abstract reasoning. The first two of these categories (i.e., apathy and emotional lability) pertain principally to personality and emotion, whereas the latter refer to behavioral production and management. Again, these taxonomic distinctions are arbitrary and the deficiencies they denote are intricately interrelated.

**Apathy.** Prefrontal lobe damage frequently produces deficits in motivation or “drive,” resulting in inertia and apathy (Hall, 1993; Kwentus, Hart, Peck, & Kornstein, 1985; Stuss & Benson, 1984) sometimes labeled “pseudodepression” (Kwentus et al., 1985). Indifference and shallowness are key features, resulting in a reduced capacity to feel or express the normal range of human emotions. A loss of interest in social interactions, when coupled with a lack of concern regarding the consequences of social behavior, can result in “lewdness with a loss of social graces, (and) inattention to appearance and hygiene” (Silver & Yudofsky, 1987). Problems in maintaining focus contribute to these difficulties.

**Emotional lability.** Marked emotional instability is another common sequelae. Irritability may quickly give way to euphoria, and increased extraversion is frequently reported (Kandel & Freed, 1989). Victims of prefrontal injury may behave in a childlike and selfish manner, resulting in features often labeled “pseudosociopathic” (Kwentus et al., 1985). Individuals with frontal lobe impairment may be intrusive, boisterous, speak loudly with free use of profanities, and engage in elevated risk-taking, indiscriminate eating, and unrestrained drinking (Silver & Yudofsky, 1987). In short, rudimentary drives and emotions are poorly regulated and the individual’s behavior is highly impulsive. Dyscontrol of rage may result (Silver & Yudofsky).

The apparent contradiction between these first two deficit categories (apathy and lability) underscores the complex role that the frontal lobes play in integrating personality. Attempts have been made to distinguish subtypes of personality change on the basis of precise location of brain damage. The relevance of these distinctions to many, if not most, cases is questionable; although relatively “pure” subtypes of personality change may exist, frontal lobe damage is often diffuse, or widely distributed, and the resulting personality changes are often mixed, varied, and many times appear contradictory (e.g., apathy combined with disinhibition). Further complicating matters is the likelihood that frontal lobe damage exacerbates pre-existing traits, such as disorderliness or argumentativeness in some individuals (Silver & Yudofsky, 1987).

**Anticipating, planning, and sequencing deficits.** The prefrontal cortex engages in temporally oriented programming to accomplish tasks. Damage, therefore, often results in disorganization and impaired problem-solving (Hall, 1993; Stuss & Benson, 1984), and a
Deficiencies in initiating behavior. Even when the individual has the capacity to plan, deficits in behavioral initiation may result in the commonly observed breakdown between stated intentions and actual behavior (Lezak, 1995). Patients display “diminished spontaneity and initiative, reduced productivity, and diminished verbal output” (Lezak, 1995).

Deficits in monitoring behavior. Frontally injured individuals often lack the ability to adequately appraise progress toward intended goals. They may fail to recognize their mistakes and their impact on others, and fail to properly evaluate social situations (Lezak, 1995; Stuss & Benson, 1984). Concentration deficits and an impaired ability to maintain mental set (i.e., to keep a goal and requisite steps in mind) further contribute to deficient self-monitoring.

Problems in shifting, adapting, and stopping behavior. Individuals with frontal lobe impairment often experience difficulty adapting to changing circumstances, even when their ability to monitor is intact and they intend to be flexible in their response. Such individuals often get locked into mental or behavioral patterns, resulting in perseveration (i.e., maladaptive repetition or persistence of a behavior beyond its useful span). Implicated in these difficulties is the inability to use knowledge to regulate or rapidly adjust behavior based on external cues (Kandel & Freed, 1989; Stuss & Benson, 1984).

Deficient abstract reasoning. Individuals with frontal lobe impairment often display a concrete attitude in which events are taken at their face value, and the individual is unable to separate him/herself from the surroundings (Lezak, 1995). Damage to the dorsolateral frontal lobes may be particularly likely to impair abstraction (Hall, 1993), resulting in a reduced capacity to utilize language, symbols, and logic (Silver & Yudofsky, 1987).

HOW MIGHT FRONTAL LOBE DEFICITS CONTRIBUTE TO VIOLENCE?

According to clinical lore, frontal lobe damage results in “frontal lobe syndrome”, a component of which is the inability to control anger (e.g., Silver & Yudofsky, 1987). Although problems of disinhibition are implicated, there are many ways in which frontal lobe deficiency may heighten the risk for aggressive conduct. Perhaps most salient are defects in planning (leading to careless or inappropriate behaviors), rigidity or difficulty modifying behavior, and interpersonal inappropriateness (including a lack of awareness of one’s effect on others, or of their emotional state).

Deficits in Inhibitory Control

The inability to inhibit inappropriate or exaggerated responses may have a particularly salient influence in propelling one toward violent responding. Frontal lobe damage may result in an inability to maintain emotional equilibrium, and an inability to control the behavioral expression of mood changes (Golden et al., 1996); the usual capacity to mediate between intellect and emotion breaks down (Hall, 1993).

Grafman et al. (1996) argue that in the normal brain “when schema-like knowledge (which would include rules of social behavior), stored in the frontal lobes, is activated,
it leads to an inhibition of more primitive reactions (e.g., violent or aggressive behavior) to environmental provocation” (p. 1231). They further state:

Knowledge stored in the prefrontal cortex plays a managerial role over behavior and takes the form of mental models, thematic understanding, plans, and social rules ... [enabling engagement] in an extended series of behaviors that have an overall theme or goal, rather than simply reacting to provocations or demands of the environment by expressing their internal raw emotion. (p. 1237)

Hence, if the relevant “knowledge” contained in the frontal lobes is less accessible due to damage or dysfunction, the inhibitory benefit of such information is diminished and inappropriate behaviors are more likely to emerge.

Thus, individuals with frontal lobe impairment may experience heightened and exaggerated emotional responses to events, have difficulty inhibiting these responses, and respond in accord with these emotions in an exaggerated or inappropriate fashion.

**Deficits in Planning**

Deficits in the ability to plan may result in inappropriate or self-defeating behavior. A lessened capacity to self-correct, learn, and think flexibly (Heinrichs, 1989) will be particularly handicapping in situations lacking clear rules and structure. The inability to generate a suitable response may exacerbate frustration and the tendency to reflexive emotional responding.

**Rigidity**

In a sense, reliance on reflexive responding suggests a form of behavioral rigidity, but the rigidity associated with frontal lobe impairment also takes on another form—that of overly persistent or perseverative responding. Lezak (1995) defines perseveration as “the continuation of a response after it is no longer appropriate” (p. 160), a problem Hall (1993) argues could be a contributing factor in some instances of aggravated assault or murder. Even planned violence may shift to disorganized and perseverative behavior, wherein an attack is continued beyond the point of resistance on the part of the victim.

**Interpersonal Inappropriateness**

To the extent that an individual with frontal lobe impairment demonstrates deficient interpersonal sensitivity, his/her propensity for inconsiderate (at least) and violent (at worst) behavior will be enhanced. Lezak (1995) notes that “Frontal damage can be suspected in those ... whose affective or empathic capacity is muted” (p. 185). An inability to empathize with the plight of the victim may exacerbate violent behavior.

Inconsiderate behaviors will provoke angry reactions in others, heightening the potential for violence (Golden et al., 1996; Kandel & Freed, 1989). Hall (1993), for example, notes that the tendency for individuals with frontal lobe impairment to be “stimulus-bound” can result in a tendency to gaze inappropriately at others, which, if interpreted as staring, may provoke a confrontation. The frontally impaired individual may fail to recognize signals that others are becoming angered and will fail to adjust his/her behavior. Other potentially annoying behaviors are alluded to in Stuss and Benson’s (1984) description of the “frontal lobe personality,” which exhibits “unrestrained and tactless behavior; mood changes including jocularity and bawdy, puerile joking (‘Witzelsucht’); blunted feelings; callous unconcern; boastfulness; and grandiose, obstinate, and childishly egocentric behavior” (p. 19).
The irritability and other personality changes frequently exhibited by brain-injured patients (e.g., Brooks, Campsie, Symington, Beattie, & McKinlay, 1987) place a heavy burden on significant others. When a significant other expresses frustration, a pattern of interaction may ensue in which confrontation quickly escalates due to the impaired emotional modulation of the injured individual.

Confrontations will also be worsened by the cognitive limitations of frontally impaired individuals. Inattention and confusion proneness may frustrate others (Hall, 1993), as will perseverative vocalizations or behaviors. Abstract-thinking deficiencies, resulting in an inability to conceptually remove oneself from a situation, further the potential for violence (Hall, 1993).

Finally, the limitations and reduced adaptive behaviors of the frontally impaired enhance their interpersonal vulnerability, increasing the likelihood that they will be ridiculed and provoked by others (Heinrichs, 1989).

In summary, individuals with frontal lobe impairment have problems modulating emotional reactions and inhibiting impulses, planning appropriate responses, and switching behavior when necessary. On the one hand, they tend to become distracted from persisting with appropriate behavior (or lose control over it), while on the other, they persist with behaviors when no longer appropriate. They may fail to demonstrate appropriate concern for, or awareness of, their effect on others, and may behave in socially inappropriate ways, eliciting angry reactions in others that they are ill-equipped to handle. They are likely to be deficient in thinking through or enacting alternatives to violence when conflict occurs, exacerbating their propensity to disinhibited responding.

**IS THERE EMPIRICAL EVIDENCE THAT FRONTAL LOBE DAMAGE IS ASSOCIATED WITH VIOLENCE?**

The notion that there is a relation between frontal lobe damage and rage has been part of clinical lore for more than 50 years (e.g., Goldstein, 1944). Historically, much of the evidence for this association has been based on case reports. Yudofsky, Williams, and Gorman (1981), for example, discussed a case in which a 63-year-old male, struck by an exploding truck tire, sustained diffuse bilateral prefrontal cortical damage. Following a 3-week coma, he was dangerously combative between 4 and 10 times per day. Despite the dramatic nature of such reports, relatively few studies have investigated the specific relationship between frontal lobe deficits (rather than global indices of brain impairment) and aggression.

Studies of the relations between neurological functioning and behavior generally yield one of three types of information. In between-group designs, individuals (usually inmates) are categorized as violent or nonviolent, and the resulting groups are compared with respect to various indices of neurological status. Alternatively, neurologically impaired and intact groups are compared with respect to incidences of aggression. In within-individual comparisons, the incidence of aggression is examined in individuals before and after a traumatic brain injury. In the third type, correlational comparisons, neurological functioning, and aggressive behavior are quantified, and the statistical relationship between the variables examined.

**Between-Group Comparisons**

In an important study, Grafman et al. (1996) examined the relationship between frontal lobe lesions and aggression in 279 veterans who had sustained penetrating head injuries.
during the Vietnam war. These patients were compared to 57 non-brain-injured veteran controls matched by age, education, and time served in Vietnam. Computed tomography (CT) scan results determined the locus of injury. Both the veteran and a friend or family member provided information regarding postinjury aggression and violence. The brain-injured veterans were reported by family and friends to be more aggressive than the non-brain-injured veterans. In particular, veterans with ventromedial frontal lobe lesions were reported to be more aggressive than non-brain-injured controls, and, importantly, were more aggressive than veterans with lesions elsewhere in the brain.

Bryant, Scott, Golden, and Tori (1984) examined the relation between neuropsychological functioning and violent behavior in 110 inmates. All participants were classified as brain-damaged or not on the basis of scores obtained on the Luria-Nebraska Neuropsychological Battery (Golden, Hammmeke, & Purisch, 1980). Violent crimes were reported to have been committed by 73% of the brain-damaged group and 28% of the non-brain-damaged group. Specifically germane to the issue of frontal involvement was the finding that the violent group demonstrated deficits in “the ability to create, plan, organize, and execute goal directed behaviors” (p. 324).

Mills and Raine (1994) reviewed studies that used neuroimaging techniques to examine brain structure (i.e., CT, magnetic resonance imaging [MRI]) and function (i.e., positron emission tomography [PET], regional cerebral blood flow [RCBF]) differences between violent and nonviolent offenders, and studies that used these techniques to examine the incidence of neurologic impairment within samples of violent offenders. The authors concluded that two alternative positions are supported by the available findings. The first postulates that frontal dysfunction may be associated with violent offending, temporal lobe dysfunction with sexual offending, and fronto-temporal dysfunction with violent sexual offending. The alternative position holds that anterior brain dysfunction (frontal, temporal, and fronto-temporal) may represent a general predisposition to offending, irrespective of the specific location of the dysfunction, and that the specific nature of the offense (e.g., violent, sexual, or violent and sexual) may be determined primarily by nonbiological factors, such as life history and personality.

**Within Individual Comparisons**

A common method for examining the sequelae of brain injury involves asking the patient, and/or a friend or family members, about the behavioral and affective changes that followed the injury. Although a number of studies have established aggression as a sequela of traumatic brain injury in general (e.g., Brooks et al., 1987), these studies have tended not to separate individuals who have sustained frontal lobe damage from others whose injuries are localized elsewhere in the brain.

**Correlational Comparisons**

Heinrichs (1989) gathered information regarding the association between neurological, psychiatric, and demographic variables and violent incidents within an institutional context. In a sample of 45 neuropsychiatric inpatients, frontal lesions (confirmed through CT scans and quantified as present or absent, rather than as exclusively frontal and exclusively nonfrontal) were the best single predictor of violent incidents over a 2-year period, accounting for 11% of the variance, whereas cerebral damage in general did not predict violent incidents.
Conclusions Based on a Review of the Evidence

Given the paucity of studies examining the specific link between frontal lobe impairment and aggression, it is not surprising that previous reviewers have drawn varying and highly tentative conclusions. Miller (1994) concluded that “aggressiveness can occur as a feature of frontal lobe damage due to brain injury . . . this includes a lower threshold for aggressive behavior” (p. 93). In contrast, Kandel and Freed (1989) reviewed three studies and concluded that, due to methodological problems (e.g., lack of adequate control groups), “the evidence for the association between specifically violent criminal behavior and frontal-lobe dysfunction is weak at best,” even though two studies found “violent criminals to evidence significantly more frontal-lobe dysfunction” (p. 410).

Although these two sets of authors examined nonoverlapping subsets of the limited research, the differences in their conclusions appear to stem less from differences in the studies reviewed than from differences in the respective authors’ judgments regarding the adequacy of the evidence. Additional well-designed research is obviously desirable. Nevertheless, the sparse information available, consistent with clinical lore, suggests that frontal lobe impairment is associated with aggressive tendencies. The strength of this relationship, and particularizing factors (e.g., exact location of damage, severity of injury, premorbid behavior) leading to the expression of aggression in some but not other individuals with frontal lobe impairment remain undetermined.

EXECUTIVE DEFICITS AND AGGRESSION IN NORMAL SAMPLES

Somewhat ironically, some of the most compelling evidence regarding the association between frontal lobe functioning and aggression may emerge from studies of individuals with no known history of brain injury. The determination of a relationship between executive weaknesses and aggression within noninjured populations would suggest an association between “subclinical” frontal lobe deficits (coupled with otherwise intact neuropsychological functioning) and a heightened propensity toward aggression. In one respect, research within a normal population provides a stringent test of this association: The variability of scores on variables is likely to be relatively restricted, resulting in attenuation of correlations and increasing the probability of null results.

Sequin, Pihl, Harden, and Tremblay (1995) examined the relations between cognitive-neuropsychological functioning and physical aggression in a sample of adolescent boys who, together with their parents and teachers, had participated in a series of annual assessments. The boys’ level of physical aggression was assessed at ages 6, 10, 11, and 12, and they were classified as stable aggressive, unstable aggressive, and nonaggressive. At the age of 13, and again at 14, the boys participated in neuropsychological testing, which generated four ability factors: Verbal Learning, Incidental Spatial Learning, Tactile-Lateral Ability, and Executive Functions. The relations between scores on each of the four neuropsychological performance factors and aggression were examined while controlling for social disadvantage (i.e., family adversity) and anxiety. Significant main effects were found only on the Executive Functions factor, with both aggressive groups scoring significantly lower (indicating specific executive weakness) than the nonaggressive group.

Giancola and Zeichner (1994) examined the relationship between frontal-lobe functioning and aggression in a community sample of young men (age range, 18–32). Subjects with a past or present problem with alcohol, drugs, or learning disability were excluded, as were those with a history of psychiatric problems or head injury. The investigators used a modified version of the Taylor reaction-time paradigm in which electric shocks
were received from and delivered to a fictitious opponent during completion of a competitive task under low- and high-provocation conditions. High provocation involved the delivery of high-intensity shocks by the opponent, and aggression on the part of the participant was determined by the duration and intensity of shocks delivered to this opponent. A significant association was found between aggression and poor performance on one putative test of frontal lobe functioning, the Conditional Association Task, but not another (the Self-Ordered Pointing Task).

Lau, Pihl, and Peterson (1995) examined the relation between provocation, alcohol intoxication, frontal lobe functions, and aggressive behavior among a community sample of physically and psychologically healthy nonalcoholic male social drinkers. Individuals receiving medical treatment that contraindicated alcohol consumption, who had sustained a brain injury, or were familiar with psychological experimentation were excluded. Participants were selected from the upper and lower quartiles of a ranking of performance on two tests of frontal lobe functioning. Participants were randomly assigned to alcohol and no-alcohol conditions, and each man’s pain threshold for electric shock was determined.

Participants then participated in a competitive reaction-time task with a fictitious opponent under conditions of varying provocation, and delivered shocks to the opponent if the opponent lost the trial and received shocks from the opponent if the participant lost the trial. Aggression was operationalized as the intensity of shock delivered to the opponent. The results revealed that “shock intensity significantly increased as a main effect of provocation, alcohol intoxication, and lower cognitive (i.e., executive) performance. Furthermore, provocation interacted significantly with test performance such that individuals in the lower cognitive performance quartile responded to increased provocation with heightened aggression.” (p. 150). Controlling for IQ did not substantially alter the results.

Taken together, these studies provide evidence of a relationship between executive weaknesses and aggression in normal subjects. That such an association is established under conditions where no known brain-injuries have occurred (and the deficits are likely primarily “subclinical” in expression) suggests that aggressive impulses are sensitive to even relatively minimal levels of dysfunction or executive weaknesses, in at least some individuals.

CONCLUSION

Clinical lore has asserted, and the available empirical evidence appears to corroborate, that brain damage in general, and frontal lobe damage in particular, is associated with an increased propensity for aggression and violence. That an association between executive functioning weaknesses and aggression can be demonstrated within samples of individuals with no known history of brain compromise serves to underscore the sensitivity of aggressive impulses to even relatively minimal impairment. Given the pressing nature of violence as a societal concern, it is surprising to find that there has been a relative paucity of research in this area. Any conclusions drawn from the available literature must be tempered by that observation.

REFERENCES


