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Orbitofrontal correlates of aggression and impulsivity in psychiatric patients

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Abstract

The association between orbital frontal cortex (OFC) volume and aggression and impulsivity was investigated among a heterogeneous group of non-psychotic psychiatric clients. Fifteen non-psychotic subjects from two different psychiatric clinics (New England Medical Center and Lemuel Shattuck Hospital) with a variety of diagnoses were sequentially referred for magnetic resonance imaging (MRI) for clinical purposes. This convenience sample, clinically stable at the time of evaluation, received a standardized psychiatric diagnostic interview, aggression and impulsivity psychometrics (Barratt Impulsivity, Lifetime History of Aggression, and Buss-Perry Aggression scales), and an MRI protocol with image analysis. OFC gray matter volume, total as well as left and right, was significantly and positively associated with motor impulsivity. OFC asymmetry was associated with aggression, though total, left, and right OFC volume measurements were not. For subjects without affective disorder, there was a strong and positive association of the OFC to motor and no-planning subscales of the Barratt Impulsivity Scale. For subjects with affective disorder, there was a strong association of OFC asymmetry to both of the aggression psychometrics. Consistent with expectation, results are suggestive of OFC involvement in the neural circuitry of impulsivity and aggression. The findings suggest a dissociation of the role of the OFC in relation to aggression and impulsivity, such that the OFC may play a part in the regulation of aggressive behavior and a generative role in impulsive behavior.

Keywords: Magnetic resonance imaging; Personality; Barratt Impulsivity Scale

1. Introduction

The association between mental illness and aggression has long been established (Eronen et al., 1998), particularly when major mental illness combines with substance abuse (Soyka, 2000). The interaction of

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multiple, complex biological factors, such as brain lesions, neurophysiologic dysfunction and the contribution of neurochemical systems, are posited to influence the neurobiological basis of aggressive behavior (Eichelman and Hartwig, 1996; Kavoussi et al., 1997). Neuroanatomical models of brain circuitry involved in aggression consistently implicate an interconnected network of regions that includes the frontal lobes and the amygdala along with other brain areas. Many of these models agree on the importance of prefrontal cortical dysfunction (Blair, 2001; Brower and Price, 2001; Davidson et al., 2000; Giancola, 1995; Pietrini et al., 2000; Volavka, 1999), although they may differ as to the localization of dysfunction within that large and complex region of the brain. In a number of these models, dysfunction of the dorsolateral prefrontal cortex (DLPFC) creates a pathway to violence by way of executive dysfunction and consequent problems of meeting occupational/social/academic expectations (Brower and Price, 2001; Giancola, 1995). Impulsivity, acting before thinking, can be considered a form of executive dysfunction in these models, along with other phenomena such as cognitive rigidity (difficulty weighing alternatives) or the inability to generate strategies. Alternatively, it has been posited that dysfunction of the orbitofrontal cortex (OFC) creates a pathway to aggression via emotional dysregulation (Blair, 2001; Giancola, 1995), functional deactivation (Pietrini et al., 2000), impulsivity (Brower and Price, 2001), or a low threshold for activation of negative affect (Davidson et al., 2000). Volavka (1999), incorporating environmental influences, takes a somewhat broader view of a neurobiological typing for violence, which includes the temporal lobe. He proposes two propensities toward violence: one via a genetic predisposition to prefrontal dysfunction (without further localization) leading to impulsive violence, while in another sub-type, an abnormal rearing environment leads to violence via decreased temporal lobe volume.

The involvement of the prefrontal cortex in aggressive and/or impulsive behavior finds support from recent neuroimaging studies. Raine et al. (1997) compared 41 murderers pleading not guilty by reason of insanity to 41 age and sex-matched controls on regional PET activation. They found lower activation in the prefrontal, superior parietal, and angular gyrus regions and no differences in temporal lobe activation. Higher occipital lobe activation was encountered among the murderers during a sustained attention task. Soderstrom et al. (2000) compared violent offenders to normal controls on head SPECT and found reduced perfusion bilaterally in the hippocampus, the left frontal white matter, and the right angular gyrus and mediotemporal region. Research on normal controls also supports an association between the frontal lobe and aggression and may suggest some degree of lateralization as well. For example, Pietrini et al. (2000) found that specific deactivation of the left and right medial frontal gyri was associated with cognitive restraint during experimentally induced aggression scenarios. This area was also described as Brodmann's area 10. During a scenario involving unrestrained aggression, peak deactivation was seen in the left medial frontal gyrus, also described as BA 11. Comparing experimental conditions, functional deactivation of BA 10/11 was strongest when subjects were instructed to express, rather than inhibit, aggressive behavior.

The use of emotion-induction paradigms with psychiatric patients has also led to recent advances in understanding the neurobiology of aggression. Recently, Dougherty et al. (2004) found 10 patients with major depression who were prone to anger attacks had significantly less PET-derived activation in the left ventromedial prefrontal cortex (VMPFC) than either 10 normal controls or 10 major depressive controls not prone to anger attacks. Invoking the neural circuitry of aggression, an association was found between amygdaloid and VMPFC activation. The authors concluded the pathophysiology of major depression with and without anger attacks may be different.

Grafman et al. (1996) explored the relation between penetrating head wounds and aggressive/violent behavior among Vietnam War Veterans and found that veterans with ventromedial-frontal lesions obtained significantly higher scores on a measure of aggressiveness and violence in comparison to veterans with other lesions. In a population characterized by its tendencies to aggression, Raine et al. (2000) found individuals diagnosed with anti-social personality disorder had lower prefrontal gray matter volume (but not white matter) in comparison to psychiatric, substance abuse, and community-based controls. Note Raine's work took the PFC as a whole without reference to subdivisions. Tonkonogy (1991) selected 23 chronic psychiatric patients from a larger group of 87 patients with suspected organic mental disorder. Tonkonogy found lesions of the anterior and inferior temporal lobe were more common in violent patients, and lesions of frontal, parietal or superior temporal lobe were more common in non-violent patients. As pointed out previously, in some aggression neurobiology models, impulsivity is considered a critical factor moderating the connection between OFC dysfunction and aggression (Brower and Price, 2001).

Interestingly, a recent review finds some support for functional localization of inhibition to the right inferior