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Prefrontal volumes in habitually violent subjects with antisocial personality disorder and type 2 alcoholism

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Abstract

Pathology of the prefrontal cortices has been suggested to be a part of neural networks underlying deviant behavioral patterns. Recently, reduced overall prefrontal cortical volumes have been proposed in subjects with antisocial personality disorder (ASP). It is not known whether there are specific patterns of volume loss within the prefrontal regions. Nor is it known if there are correlations between the prefrontal volumes and degree of psychopathology. In this study, total prefrontal, prefrontal white, and cortical (dorsolateral, orbitofrontal, medial frontal) prefrontal volumes were measured from magnetic resonance images in 24 non-psychotic, violent male subjects who had a diagnosis of ASP in combination with type 2 alcoholism, and 33 age-matched control males. The degree of psychopathy in the ASP subjects was assessed using the Psychopathy Checklist-Revised (PCL-R). Compared with the controls, the ASP subjects had significantly smaller volumes of all three cortical regions on the left, but this significance disappeared after controlling for differences in education and duration of alcoholism. For the dorsolateral and orbitofrontal cortices, only duration of alcoholism was significantly associated with the observed volume deficit, and for the medial frontal cortex it was the difference in education. Thus, the observed volume deficits in this sample were related more to alcoholism or differences in education rather than to the diagnosis of ASP. Moreover, no significant correlations between any of the volumes and the degree of psychopathy were found. © 2002 Elsevier Science Ireland Ltd. All rights reserved.

Keywords: Alcohol; Antisocial personality; Homicide; Imaging; MRI; Prefrontal; Sociopathy; Violence

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1. Introduction

The frontal lobes, particularly the prefrontal cortices, have been proposed as the prime candidate for a cerebral substrate of 'the social man'. Since the famous case of Phineas Gage, lesion studies have linked ventromedial (i.e. orbitofrontal and medial prefrontal) cortical damage to impairment in social behavior and proper decision-making (Damasio, 1994). Studies of small samples of individuals with childhood lesions of the ventromedial prefrontal cortex have suggested that such lesions sustained at an early age may lead to impairment of moral and social reasoning and even to violent antisocial behavior in late adolescence and young adulthood (Anderson et al., 1999; Price et al., 1990). Although the roots of antisocial personality can often be traced to childhood (Hamparin et al., 1978), the vast majority of antisocial persons do not have a history of gross prefrontal pathology. Yet, in accord with lesion studies, perfusion imaging studies of violent antisocial persons, with or without psychiatric comorbidity, have rather consistently revealed decreased prefrontal metabolism in comparison with non-violent normal and psychiatric control subjects (Raine et al., 1997; Soderstrom et al., 2000; Volkow et al., 1995). Moreover, a recent magnetic resonance spectroscopy (MRS) study proposed decreased levels of *N*-acetyl aspartate (NAA) and creatine in the right medial prefrontal region in mentally retarded individuals with a history of repetitive aggressive behavior (Critchley et al., 2000). While both these approaches have their merits in detecting pathologies, their neuroanatomical specificity is limited, as neither method can readily distinguish whether the observed deficit is due to neural dysfunction or neural loss. Volumetric measures obtained from high-resolution structural magnetic resonance imaging (MRI) scans, while not free of limitations, can contribute to resolution of this dilemma by revealing regional shrinkage, which can be taken as a reflection of neural loss.

Recently, the first evidence linking smaller volume of the prefrontal cortex with antisocial personality disorder (ASP) was provided by Raine et al. (2000). However, in that study, the authors suggested that ASP is associated with reduction in

the prefrontal cortical volume without examining the issue of regional specificity. Given the functional heterogeneity between different parts of the prefrontal cortices, knowledge of deficits in specific locations would be of further interest, in order to better understand the pathology of the neural networks underlying ASP. Moreover, it remains unknown as to whether there are any correlations between the degree of psychopathy and the prefrontal cortices in terms of a structure–(mal)function relationship. Thus, the aims of this study were to replicate and expand the findings of Raine et al. (2000) in two major aspects. First, we attempted to specify the location of the volume loss and to establish whether ASP is differentially associated with circumscribed prefrontal regions. Second, we examined the possibility of a dose–response relationship between the severity of psychopathy in the ASP group and the degree of prefrontal volume differences. To this end, we assessed whether or not regional or total frontal volumes correlate with the degree of psychopathy in subjects with ASP, by using the Psychopathy Checklist-Revised (PCL-R) (Hare, 1991).

2. Methods

In this study, a total of 57 male subjects were studied (Table 1). Of these, 33 were control subjects, and 24 were violent offenders, with diagnoses of antisocial personality disorder (ASP) and type 2 alcoholism. The study was approved by the local research ethics committee.

2.1. Control subjects

The control subjects consisted of hospital staff, students, and their relatives or spouses. They were healthy, with no history of somatic, psychiatric or neurological disorders, or substance abuse. The controls had obtained a mean of 14 ± 2 years of education.

2.2. The ASP group

The ASP subjects were recruited for the study from a pretrial forensic psychiatric evaluation that lasted from 4 to 8 weeks, in a security ward of a

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