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Positron emission tomography in female patients with Borderline personality disorder

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Abstract

The pathology of Borderline personality disorder (BPD) is poorly understood and its biological basis remains largely unknown. One functional brain imaging study using [¹⁸F]Deoxyglucose-PET previously reported frontal and prefrontal hypometabolism. We studied brain metabolism at baseline in 12 medication-free female patients with BPD without current substance abuse or depression and 12 healthy female controls by [¹⁸F]Deoxyglucose-PET and statistical parametric mapping. We found significant frontal and prefrontal hypermetabolism in patients with BPD relative to controls as well as significant hypometabolism in the hippocampus and cuneus. This study demonstrated limbic and prefrontal dysfunction under resting conditions in patients with BPD by FDG-PET. Dysfunction in this network of brain regions, which has been implicated in the regulation of emotion, may underlie symptoms of BPD.

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1. Introduction

Borderline personality disorder (BPD) is a persistent and severe mental disorder characterized by patterns of instable interpersonal relationships, self-image, affects, and marked impulsivity that begins in early adulthood and is present in a variety of different contexts throughout adult life (American Psychiatric Association, 2000). Most researchers currently agree that a dysfunction of the emotional regulation system is a core component of the disorder (Linehan, 1993; Silk, 2000; Corrigan et al., 2000; Herpertz et al., 1999; Stiglmayr et al., 2001). Currently, the etiology of BPD is poorly understood and its biological basis remains largely

unknown. Preclinical research has revealed a network of regions involved in emotional regulation including prefrontal cortex, hippocampus, and amygdala (Davidson et al., 1999). The amygdala play a decisive role in the regulation of fear (Davis, 2001) and the hippocampus is involved in fear responses to the context of a stressful situation (Phillips and Le Doux 1992). Prefrontal cortex also regulates emotion and stress responses, including impulse control, inhibition of responses to external stimuli, and extinction of fear responses.

The crucial function of these brain regions in the expression and modulation of emotion and impulsivity in both animals and humans has led to the hypothesis that dysfunctions in these regions may underlie some of the psychopathological symptoms seen in patients with BPD. One functional brain imaging study employing [¹⁸F]Deoxyglucose positron emission tomography (FDG-PET) under resting conditions revealed decreased metabolism in premotor and prefrontal areas, the anterior part of the cingulate cortex, and the thalamic, caudate and

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lenticular nuclei, in BPD patients as compared to controls (De la Fuente et al., 1997). In a pilot fenfluramine challenge study in five BPD patients and eight controls, Soloff et al. (2000) found greater FDG uptake in response to fenfluramine in medial and orbital regions of right prefrontal cortex (area 10), left middle and superior temporal gyri, left parietal lobe, and left caudate body in the control participants. There were no areas in which patients with BPD had greater relative regional uptake than controls. Herpertz et al. (2001) found elevated fMRI BOLD signals in the amygdala and prefrontal cortex of patients with BPD but not of controls during the presentation of emotionally aversive pictures of the International Affective Pictures System (IAPS).

In the present study, we compared brain metabolism in patients with BPD in comparison to normal controls using FDG-PET under resting conditions. Since there is only one published study to date investigating baseline brain metabolism in BPD, we aimed at replicating the results of De la Fuente et al. (1997). Based on the pre-clinical studies and preliminary PET studies in BPD cited above we hypothesized alterations in function in prefrontal cortex, anterior cingulate, and hippocampus in BPD.

2. Methods

2.1. Patients

Twelve female patients fulfilling DSM-IV as well as DIB-R (Zanarini et al., 1989a) criteria for BPD (score ≥ 8) were recruited at the Department of Psychiatry and Psychotherapy/University of Freiburg Medical School. All patients had been referred to participate in an inpatient treatment program for BPD. All patients had been free of psychotropic medication for at least 4 weeks. Diagnosis of BPD was assessed using the appropriate segment of the Structured Clinical Interview for DSM-IV Personality Disorders (First et al., 1995). Axis I disorders were assessed by use of the structured clinical interview for DSM-IV Axis I Disorders (Spitzer et al., 1995) or by use of the Mini-International Neuropsychiatric Interview (MINI; Sheehan et al., 1998). Patients with a lifetime diagnosis of schizophrenia, bipolar I disorder, alcohol or drug abuse within the past 6 months, or current anorexia or major depression were excluded. Four patients had current comorbid dysthymia, two panic disorder, two agoraphobia, three social phobia, three obsessive-compulsive disorder, three posttraumatic stress disorder (PTSD), and two bulimia. In addition to the interview, depression was self-assessed by the patients using Beck's Depression Inventory (Beck et al., 1961). Only patients who had successfully finished regular schooling in the German system were included.

PET measurements in patients with BPD were compared to 12 healthy female controls who were recruited by newspaper advertisement. The controls were free of lifetime psychiatric disorders and reported no psychiatric disorders in their first degree relatives. Mean age in the patient group was 25 ± 4 years (range 18–32 years), mean age in the control group was 30 ± 9 years (range 18–39 years). There was no significant group difference in mean age between patients and controls (two tailed *t*-test; $P=0.131$). Written informed consent was obtained before participation in the study. The experiments were performed in accordance with the Helsinki Declaration of 1975. The protocol was approved by the local Ethical Committees.

2.2. Positron emission tomography

The PET procedure was performed according to previously defined standards (Juengling et al., 1999; Juengling et al., 2000). In brief, the patients were allowed to rest for at least 10 min before injection of 200 ± 20 MBq 18-FDG and during the uptake period for another 20 min in an acoustically isolated and dimmed room. The patients were then transferred to the scanner (Siemens CTI ECAT EXACT tomograph, 10.8 cm FOV, 6.8 mm FWHM), where the patients' heads were positioned according to the orbitomeatal line. The control group was measured on the same type and model of the named PET-scanner at a later time point.

Image acquisition was started 30 min after injection. Six dynamic frames of 5 min duration each were acquired. Images were then reconstructed using filtered back-projection by Shepp-Logan filter (cut-off 0.35 cycles/pixel). Attenuation correction was performed using the standard mathematical algorithm implemented in ECAT software. The dynamic frames were then checked for motion artifacts and summed up to generate a single dataset of 31 transaxial planes.

Table 1
Hypometabolic areas in patients with BPD as compared to controls^a

Anatomical structure	Hemisphere	<i>x y z</i> – Talairach coordinates ^b (center)	Maximum Z-score	Extent of difference to NDB (%)
Hippocampus	Left	32 –10–16	5.14	–10.7%
Cuneus (BA 19)	Left	30 –67 20	6.13	–14.4%

^a Given are the anatomical structure, the putative areas of Brodman, the Talairach coordinates of the localization of the maximum Z-score of each cluster (in *x*-, *y*-, *z*-direction) and the values of the maximum Z-scores and percentage of difference in regional activity as compared to the normal data base.

^b Image orientation is according to radiological convention, i.e. negative *x*-coordinates correspond to the right hemisphere.

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