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Reduced glucose metabolism in temporo-parietal cortices of women with borderline personality disorder

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Abstract

Individuals with borderline personality disorder (BPD) and posttraumatic stress disorder (PTSD) often experience dissociative symptoms. Evidence is increasing that stress-related hyperglutamatergic states may contribute to dissociative symptoms and neurodegeneration in temporo-parietal cortical areas. Seventeen young women with BPD who had been exposed to severe childhood physical/sexual abuse and presented with pronounced dissociative symptoms underwent ¹⁸fluoro-2-deoxyglucose positron emission tomography (FDG-PET). Nine healthy, matched volunteers served as comparison subjects. Borderline subjects displayed reduced FDG uptake (as analyzed by SPM) in the right temporal pole/anterior fusiform gyrus and in the left precuneus and posterior cingulate cortex. Impaired memory performance among borderline subjects was significantly correlated with metabolic activity in ventromedial and lateral temporal cortices. Our results demonstrate regional hypometabolism in temporal and medial parietal cortical regions known to be involved in episodic memory consolidation and retrieval. Currently, the precuneus/posterior cingulate cortex is modeled as part of a network of tonically active brain regions that continuously gather information about the world around and within us [Gusnard, D.A., Raichle, M.E., 2001. Searching for a baseline: functional imaging and the resting human brain. *Nature Reviews Neuroscience* 2, 685-694.]. Decreased resting metabolic rate of these regions may reflect dissociative symptoms and possibly also identity disturbances and interpersonal difficulties of individuals with BPD.

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1. Introduction

Borderline personality disorder (BPD) is defined as an intermediate level of personality organization that is considered to occupy a borderline area between

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neurosis and psychosis (Kernberg, 1967). Stress-related dissociative symptoms and psychotic features occur in about 75% of individuals with BPD (Skodol et al., 2002) and in about 40% of individuals with posttraumatic stress disorder (PTSD) (Bremner et al., 1992; David et al., 1999). However, dissociative symptoms in BPD and PTSD and their possible relationship to impaired brain mechanisms have received only limited systematic investigation.

Research so far has focused on size reductions of the amygdala and hippocampus in individuals with BPD who had been exposed to childhood physical or sexual abuse (Driessen et al., 2000; Schmahl et al., 2003a; Tebartz van Elst et al., 2003; Rüscher et al., 2003; Brambilla et al., 2004; Irle et al., 2005). Functional imaging studies showed that persons with BPD display pronounced prefrontal dysfunction (De la Fuente et al., 1997; Soloff et al., 2000, 2003; Juen-gling et al., 2003; New et al., 2004) and enhanced prefrontal and amygdala activation in response to emotional or traumatic stimuli (Herpetz et al., 2001, Donegan et al., 2003; Schmahl et al., 2003b; Driessen et al., 2004). Dysfunction in prefrontal regions (Brodmann areas 9–12) is suggested to be implicated in the deficits of persons with BPD to regulate emotional behavior (Soloff et al., 2003; New et al., 2004).

Studies investigating the structural and functional neural correlates of dissociative symptoms in BPD are lacking. However, research on individuals with epilepsy of the temporal or parietal lobes has consistently demonstrated that abnormal EEG activity, seizures, or brain stimulation of the temporal or parietal cortices are associated with dissociative states (Halgren et al., 1978; Mesulam, 1981; Gloor et al., 1982; Salanova et al., 1995; Blanke et al., 2002). Stimulation of the parietal cortex typically leads to somatosensory aura or disturbed perceptions of the body (Salanova et al., 1995; Blanke et al., 2002). Lesion studies provide evidence that the parietal cortex is engaged in the generation and maintenance of an internal (sensorimotor) representation of the body (Sirigu et al., 1996; Berlucchi and Aglioti, 1997; Wolpert et al., 1998).

Increasing evidence suggests that glutamatergic dysfunction represents an important part in the pathophysiology of dissociative states. The subanesthetic application of the *N*-methyl-D-aspartate (NMDA) antagonist ketamine is known to produce dissociative

symptoms in humans (Krystal et al., 1994). The proposition has been put forward that NMDA receptor hypofunction might cause excitotoxic limbic (i.e. hippocampal) and temporo-parietal cortical neurodegeneration (Olney and Farber, 1995). Evidence is increasing that stress-related hyperglutamatergic states may also contribute to dissociative symptoms and neural toxicity (i.e., hippocampal degeneration) in individuals who have been exposed to traumatic stress (Chambers et al., 1999). A recent study found that burn victims with enduring ketamine application in the posttraumatic state showed significantly stronger PTSD symptoms than burn victims without such treatment, and more severe PTSD symptoms of subjects with ketamine treatment were related to smaller hippocampal size (Winter and Irle, 2004).

In a previous investigation (Irle et al., 2005), we found reduced size of the hippocampus and parietal cortex in a sample of women with BPD who had been exposed to severe childhood sexual and physical abuse. All subjects presented with pronounced dissociative symptoms. In the present investigation, the brain glucose metabolism (by use of ¹⁸fluoro-2-deoxyglucose, FDG) of 17 women with BPD (who were also included in the report of Irle et al., 2005) was compared with that of a healthy matched control group (*n*=9). The goals of our study were 1) to investigate whether brain glucose metabolism is selectively reduced in temporo-parietal cortices of BPD subjects presenting with pronounced dissociative symptoms and 2) to investigate whether temporo-parietal metabolic changes are related to clinical symptoms and to memory deficits of BPD subjects.

2. Methods

2.1. Subjects

2.1.1. Subjects with borderline personality disorder

The sample comprised 17 young female in-patients with the diagnosis of borderline personality disorder (BPD) according to the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* (American Psychiatric Association, 1994) consecutively admitted to the Psychiatric State Hospital of Lower Saxony, Göttingen, Germany (Table 1). The hospital has a

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