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Pain experience related to self-injury in eating disorder patients

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Abstract

A reduced pain sensitivity has been found in eating disorder (ED) patients. These patients often show various types of self-injurious behaviors (SIB) which some experience as painful, while others report analgesia during episodes of SIB. How can we explain these differences? We studied female ED patients ($N=185$) of whom 84 had admitted some form of SIB in their recent history. The presence/absence of pain report during SIB was not significantly related to the type of ED. The longer the history of SIB, the less pain was reported. Ratings of dissociation and traumatic experiences were higher in patients who did not experience pain while injuring themselves. We discuss several biopsychosocial explanations for these findings with suggestions for future research.

Keywords: Anorexia nervosa; Bulimia nervosa; Pain sensitivity; Self-injury

1. Introduction

We define self-injury or self-injurious behavior (SIB) as the deliberate, direct destruction or alteration of body tissue, without conscious suicidal intent, but resulting in injury severe enough for tissue damage to occur (Gratz, 2003). Examples of SIB are self-cutting, burning, hitting and scratching. High rates of SIB have been described in eating disorder (ED) patients, particularly in bulimics (e.g., Claes, Vandereycken, & Vertommen, 2003; Favaro & Santonastaso, 1998; Welch & Fairburn, 1996). At least one-half of individuals with borderline personality disorder (BPD) who engage in SIB such as superficial cutting report that they typically do not feel pain during episodes of self-injury (Leibenluft, Gardner, & Cowdry, 1987). In the ED patients we have studied previously (Claes et al., 2003), no pain during SIB has been reported by 38.9% of those scratching themselves, 16.7% of the self-bruising patients, and 33.3% of the cutters. Patients who did not feel pain during SIB reported more dissociative experiences, but the difference was only statistically significant for the act of cutting.

Russ et al. (1992) addressed the question of whether the intensity of pain experience during SIB is related to pain sensitivity. They studied a group of 22 BPD patients showing SIB with or without reported pain experience,

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and compared them to a control group of normal females ($N=6$). The group reporting no pain during SIB had significantly lower pain ratings during a cold-pressor test than the others. Interestingly, only those patients who were obviously pain-insensitive also showed a decrease in negative emotions (e.g., depression, anxiety, and anger) and an increase in positive emotions (e.g., vigor) after the cold pain stimulation. Also of interest, Russ, Roth, Kakuma, Harrison, and Hull (1993) and Russ, Shearin, Clarkin, Harrison, and Hull (1993) reported that the absence of pain during episodes of SIB in women with BPD is related to higher levels of anxiety, depression, dissociation, impulsiveness, trauma symptoms, and suicide attempts, as compared with a group of pain-sensitive patients with BPD (Bohus et al., 2000). Stress-induced analgesia in patients with BPD and patients with dissociative disorder has been explained as a component of conditioned defensive reactions following early inescapable stress or trauma (Nijenhuis, Spinhoven, van Dyck, van der Hart, & Vanderlinden, 1997). However, data of Bohus et al. (2000) do not fully support this appealing hypothesis. Under conditions of calmness, BPD patients did not differ from healthy control subjects in experiencing dissociative features, but nevertheless revealed a highly significantly increased pain threshold. Thus, these findings give some support to the hypothesis of a state-independent neurosensory alteration which may become accentuated when the patient experiences stress or stress-related dissociative features. The underlying neurobiological mechanisms remain to be elucidated. There is some evidence that alterations of the opioid system might play a role (Bohus et al., 1999), but this suggestion is controversial (Russ, Roth et al., 1993).

Before discussing our research questions we will briefly review the results of pain research in ED patients. In comparison to normal controls, patients with anorexia nervosa (AN), bulimia nervosa (BN), or binge eating disorder (BED) have elevated thresholds to thermally (Lautenbacher, Pauls, Strian, Pirke, & Krieg, 1990; Lautenbacher, Pauls, Strian, Pirke, & Krieg, 1991) and mechanically induced pain (Faris et al., 1992; Raymond et al., 1995), but they do not exhibit similar alterations in their sensitivity to non-noxious stimuli, such as warmth, cold vibration, or touch (de Zwaan, Biener, Bach, Wiesnagrotzki, & Stacher, 1996). The various types of ED do not differ from each other with respect to pain thresholds (Lautenbachter, Pauls et al., 1991; de Zwaan et al., 1996; Raymond et al., 1995). Lautenbachter and Krieg (1994) discussed several explanations for the difference in pain threshold in ED patients compared to normal controls.

1. The core hypothesis of the reduced pain sensitivity being opioid-mediated was found to be rather unlikely, since a normalization of the increased pain thresholds could not be achieved by administering the opiate antagonist naloxone (Lautenbacher et al., 1990).
2. A second hypothesis, that the reduced pain sensitivity is a consequence of general somatosensory deficits due to a subclinical malnutrition neuropathy, also had to be rejected since other sensory modalities, such as warmth, cold, and vibration sensitivity, were either not affected or showed only minor impairments (Pauls, Lautenbacher, Strian, Pirke, & Krieg, 1991).
3. A third possible explanation for the change in pain sensitivity was that the fasting state by itself would be enough to cause the reduced pain sensitivity (Pirke, Pahl, Schweiger, & Warnhoff, 1985). However, a 3-week 1000 kcal diet did not influence pain sensitivity in healthy volunteers even though the diet induced a fasting state comparable to that found in ED patients (Lautenbacher, Barth et al., 1991).
4. It can also be postulated that the short-term metabolic and endocrine adjustment reactions to excessive changes in food intake are sufficient to produce decreased pain sensitivity. Such an assumption is supported by the finding that a considerable percentage of chronic dieters, who, like ED patients, shift frequently between reduced and excessive eating, show increased pain thresholds (Krieg, Roscher, Strian, Pirke, & Lautenbacher, 1993).
5. The decreased pain sensation in ED patients can also be associated with symptoms specific to ED, such as bingeing and purging. This relates specifically to abnormalities in vagal functioning. It has been suggested by Halmi and Sunday (1991) that abnormal satiety responses in BN patients as well as bingeing/purging behaviors (Faris et al., 1998) may be mediated by high levels of afferent vagal stimulation. Elevated afferent vagal activation may also stimulate descending pain inhibitory pathways, leading to an elevation in somatosensory pain thresholds (Raymond et al., 1999). Therefore, dysregulated vagal control in BN could be accompanied by decreased nociceptive responsivity (Faris et al., 1992). This hypothesis is supported by the temporary decrease among BN patients of pressure pain threshold immediately after an episode of bingeing and purging, and its elevation during the intervals between consecutive episodes (Faris et al., 1998). Furthermore, BN patients do not report greater pain