Psychoneuroendocrinology (2005) **30**, 996–1002

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Psychobiological perspectives on somatoform disorders

Winfried Rief[a,b,*], Arthur J. Barsky[a]

[a]*Brigham and Women's Hospital/Harvard Medical School, 75 Francis Street, Boston, MA, USA*
[b]*University of Marburg, Department of Clinical Psychology and Psychotherapy, Gutenbergstrasse 18, D-35032 Marburg, Germany*

Received 17 November 2004; received in revised form 7 March 2005; accepted 7 March 2005

KEYWORDS
Somatoform;
Immunology;
Amino acids;
Psychophysiology;
Neuroimaging

Summary Common physical symptoms such as abdominal pain, headache, back pain and dizziness play a major role for the health care system. Existing models for the development and maintenance of these symptoms emphasize a vicious circle with cognitive-perceptual, behavioral, and psychobiological components. In this manuscript, we present examples of psychobiological factors that might contribute to somatoform disorders. We emphasize that somatoform symptoms are not strictly mental events, but are associated with a diversity of biological processes. The possible role of the endocrine and immune system, amino acids and neurotransmitters, but also physiological activation and cerebral activity is exemplified. These approaches are categorized using a model of perception and filtering of bodily signals. Studies are needed that combine the investigation of different biological systems with assessments of psychological variables in longitudinal trials, but also experimental investigations in humans examining the interaction of behavior changes, biological variations, and body perception are still rare.

Abdominal pain, bloating, dizziness, chest pain, pelvic pain, intolerance of food, palpitations or joint pains are common symptoms and typical reasons for doctor visits. Some of these symptoms have base rates of more than 30% in the general population (Rief et al., 2001a). Although the probability of remittance is substantial for individual symptoms, many affected people have multiple symptoms which tend to be persistent (Kroenke and Mangelsdorff, 1989). The health care impact of these syndromes is substantial (Smith et al., 1986; Barsky et al., 2001). A diversity of diagnoses and labels have been suggested for these complaints, e.g. unexplained physical symptoms, subjective health complaints, as well as fibromyalgia, chronic fatigue syndrome, or multiple chemical sensitivity syndrome show associations with these complaints. Therefore, we will refer to these disorders with the term 'somatoform-associated disorders'. However, the focus of this article will be on research using the

* Corresponding author. Tel.: +49 6421 28 23657; fax: +49 6421 28 28904.
E-mail address: rief@staff.uni-marburg.de (W. Rief).

0306-4530/$ - see front matter © 2005 Elsevier Ltd. All rights reserved.
doi:10.1016/j.psyneuen.2005.03.018

DSM-IV concept of somatoform disorders. The most expensive subgroup of patients with somatoform disorders are those with multiple and persisting complaints. DSM-IV suggests the diagnosis somatization disorder for patients with at least eight symptoms from four body sites; a total list of 33 somatic symptoms is suggested to be typical for somatization disorder. We will refer to the list of these 33 symptoms as 'somatoform symptoms'. As the criteria for somatization disorder are very exclusive and do not represent the majority of patients with multiple somatic complaints, subsyndromal classification for these patients have been suggested. In most of our studies, we included patients with at least eight symptoms from the list of 33 somatoform symptoms, but not necessarily fulfilling all criteria for somatization disorder. We will use the term 'somatization syndrome' for this subgroup. This implies that not all patients with multiple physical complaints or with subjective health complaints are included in this subgroup, but only those with bodily symptoms from the somatization disorder symptom list, which cannot be attributed to typical medical conditions. While somatization disorder and somatization syndrome are sub-diagnoses of the general category 'somatoform disorders', this general category also includes other diagnoses such as hypochondriasis, conversion disorder, pain disorder, or body dysmorphic disorder.

Most models of somatoform symptoms emphasize the interaction of cognitive and perceptual processes with behavioral, affective, and biological changes. Although there is evidence that all of these features contribute to the perception of physical complaints, somatoform disorders are frequently misunderstood as mere cognitive-attributional phenomenon. In this article, we will therefore review the literature suggesting that biological factors play a role in the development and maintenance of somatoform symptoms. As space limitations do not allow a comprehensive literature review, we will present just a few examples highlighting biological components of the disorder. These findings will be discussed in light of the cognitive activation theory and sensitization model published in this journal (Ursin and Eriksen, 2004). Finally, we will conclude with suggestions for further psychobiological research.

DSM-IV somatoform disorders are characterized by bodily complaints that cannot be completely attributed to known physical conditions. However, as already outlined elsewhere (Sharpe and Bass, 1992), this does not mean that these symptoms are without physiological covariates. Although there may not be evidence of discrete organic pathology, demonstrable physiological changes can still increase the risk of development and maintenance of physical complaints.

In many of the studies included, the specificity of the findings is unclear, since anxiety, traumatization and depression can also be associated with biological changes, and frequently occur in conjunction with somatoform symptoms.

1. Somatoform symptoms are not 'unfounded'—Some biological correlates

1.1. Autonomic physiological arousal

Somatoform symptoms could in theory result from heightened physiological activity. Increased physiological activation increases the likelihood of perception and misattribution of bodily signals. Although it is evident that perception is substantially influenced by psychological factors, physiological activation of variables such as heart rate or others could still play an independent role (Wientjes and Grossman, 1994). Empirical investigation of this basic hypothesis is less frequent than expected. In Pennebaker's model (Pennebaker, 1982), the perception of physical symptoms is determined by the intensity of the interoceptive signal itself divided by the intensity of external stimulation ('distraction'). This model would suggest a direct relationship between the intensity of physiological signals and the severity of somatoform symptoms. Physiological hyperreactivity would therefore be a risk factor for the development of physical symptoms.

In a study by our group (Rief and Auer, 2001), the psychophysiological reactivity of patients with multiple somatoform symptoms was assessed during relaxation and mental distress. As mental stressor, the span of apprehension test which requires continuous attentional processing was used. This is a choice-reaction time task with visual stimuli including differing numbers of distracting elements. For most physiological signals included in this study (such as muscular reactivity, electrodermal responses, peripheral circulation), no significant differences between healthy controls and patients with somatization syndrome were found. In healthy controls, the change from attention tasks to rest periods was associated with a substantial decrease in heart rate activity ('recovery response'). This reduction of physiological activity after mentally distressing tasks was not found in patients with somatization syndrome. This effect was not determined by depression or anxiety.