



Biological sensitisation and psychological amplification: Gateways to subjective health complaints and somatoform disorders

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Received 6 October 2004; accepted 28 January 2005

KEYWORDS

Subjective health complaints;
Somatoform disorders;
Biological sensitization;
Psychological amplification;
Cognitive behavioural therapy

Summary Subjective health complaints without known physical pathology are common and might develop into somatoform disorders. Normalization of neuroticism and anxiety in patients with duodenal ulcer disease after a relapse-free state is acquired and maintained is an indication that biological factors influence psychological factors. Recent research has shown that psychological and behavioural changes, on the other hand, have the capacity to change the brain. Visceral hyperalgesia is an example of biological sensitisation, but the abnormal sensory perception can be normalised by hypnotherapy. The dual-etiology hypothesis of functional somatic syndromes implies that in some patients with somatoform disorders there is a predominant biological etiology, whereas in others there is a predominant psychological etiology. The theory is supported by recent research, and may result in better handling of patients. Cognitive factors like catastrophising amplifies subjective physical symptoms and emotions effect the perception of them. In preventive health care the following slogans are proposed as antidotes preventing subjective health complaints form developing into somatoform disorders: Do not listen to your body's signals! Do not trust your feelings! Do not trust your thoughts!
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1. Introduction

Subjective health complaints (SHC) without known physical pathology range from purely normal physiological sensations, experienced by most people, to disabling symptoms, classified as Somatoform disorders (WHO, 1992). Surveys on

the general population document that SHC are common in industrialised, Western societies, as well as in people living under primitive conditions (Eriksen et al., 2004; Verbrugge and Ascione, 1987). Musculoskeletal symptoms are experienced by 30% (Cunningham and Kelsy, 1984) and significant fatigue by more than 20% of a normal population (Pawlikowska et al., 1994). The decision to seek medical help is influenced by symptom severity, anxiety, psychological morbidity, life event stress,

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personality, abuse and abnormal illness attitudes and beliefs (Shapiro and Olden, 2000; Koloski et al., 2001). The etiopathogenesis of SHC is complex, and the role played by genetics, biological sensitization and psychosocial factors probably differs between individuals and within groups of people given a diagnosis because of symptom clusters. Sensitization, defined as a central nervous process in which neuronal sensitization may be the neurobiological substrate, has been proposed as the biological substrate for intolerable SHC, constituting somatoform disorders (Eriksen and Ursin, 2002). The term includes biological sensitization, for example visceral hyperalgesia, and psychological sensitization, when hypervigilance to a specific SHC leads to amplification of the symptom, as in hypochondriasis.

The aim of this article is to review some recent publications that give new insights into the interaction between biological and psychological factors in SHC and somatoform disorders, with special emphasis on functional gastrointestinal syndromes (FGS). The discussion focuses on the consequences these findings could have on the communication between the medical community and the public.

2. Biological factors influence psychological processes—and vice versa

The invention of positron emission tomography (PET) scan and functional magnetic resonance imaging (fMRI) of the brain has shown that the interaction between psychological factors and organic brain characteristics is a two-way route. Psychiatric disorders, like major depression, phobias and obsessive-compulsive disorder, are characterised by regional brain metabolic changes, which are changed when the patients are in remission (Holthoff et al., 2004). These changes can be initiated by pharmacological interventions, but also by purely psychological influences, like cognitive behaviour therapy (CBT) (Schwartz et al., 1996). fMRI has demonstrated that when major depressive disorder is treated by antidepressants, response-specific regional changes occur in the brain. PET-studies of patients with unipolar depression have shown that also CBT modulates the functioning of specific sites in limbic and cortical regions (Goldapple et al., 2004). Paquette et al. (2003) used fMRI to study the effect of cognitive-behavioural therapy on neuronal correlates of spider phobia. Regional brain activity while watching a film depicting spiders was registered in

13 normal control subjects and 12 female patients, before and after successful treatment. Indeed, the dysfunctional neural activation in the right dorso-lateral prefrontal cortex, the bilateral parahippocampal gyrus and associative visual cortex before treatment was normalised afterwards. The activation found in patients was not found in the controls or when the patients were subject to neutral film excerpts of butterflies in nature.

The interaction of biological and psychological factors is also illustrated by our finding that neuroticism and anxiety are normalised in patients with duodenal ulcer (DU) when a relapse-free state is acquired and maintained (Wilhelmsen and Berstad, 2004). When the patients were infected by *Helicobacter pylori* (*H. pylori*) and had a relapsing disease, their scores of neuroticism and anxiety were higher than normal controls. The gradual reduction to normal levels of psychological distress, abdominal and other subjective somatic symptoms, started when the patients became relapse-free after effective triple-treatment eradicated the bacteria. This led us to conclude that the distress of having relapsing ulcers had negative, secondary psychological consequences; and even traits considered to be stable personality characteristics, like neuroticism, are subject to change. The relationship between life-event stress and *H. pylori* infection was highlighted in a study of gastric ulcer after an earthquake in Japan in 1995 (Matsushima et al., 1999). It was found that 83.1% of the patients who developed gastric ulcer after the earthquake were infected, indicating that *H. pylori* is a potent risk factor enhancing ulcer development when a person is subject to environmental stress.

3. A dual-etiology hypothesis

Functional somatic syndromes are overlapping and co-occurring (Barsky and Borus, 1999). In FGS, for example, irritable bowel syndrome (IBS), there is a high comorbidity with other FGS, as well as with other somatoform disorders, like fibromyalgia, chronic fatigue syndrome and temporomandibular joint disorder. An old fashioned dualistic approach, implying that a condition is either organic or psychological, is overly simplistic, and of no help in our understanding of and communication with patients. A systematic review of the comorbidity in patients with (IBS) led the authors to propose a new dual-etiology hypothesis (Whitehead et al., 2002). This theory implies that in some patients IBS has a predominantly psychological etiology, whereas in others it has a predominantly biological etiology.

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