Shorter communication

The catastrophic misinterpretation of physiological distress

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Abstract

Cognitive theories of panic disorder suggest that the catastrophic misinterpretation of bodily sensations is the trigger for a panic attack. A challenge to cognitive theories is the suggestion that dyspnea (shortness of breath) is central to the development of panic and that negative cognitions are by-products of panic. To examine these seemingly contradictory theoretical perspectives, the present study investigated panic symptomatology in a sample of patients with chronic shortness of breath (i.e. pulmonary patients). Past studies have shown an increased prevalence of panic in pulmonary patients, a finding that may be useful in elucidating panic etiology. The current sample of pulmonary patients (N = 28) confirmed previous reports of high prevalence rates of panic in this population. Based on self-report of panic symptomatology, a total of nine patients (32%) met DSM-IV criteria that were consistent with panic disorder. Multivariate comparison of participants with and without panic symptomatology revealed that panickers had significantly higher levels of anxiety, depression and agoraphobic cognitions. However, these groups showed no significant differences on physiological measures of pulmonary functioning. The authors conclude that dyspnea alone is inadequate in predicting panic development. High levels of panic symptomatology in pulmonary samples may reflect increased opportunities for these patients to misinterpret bodily sensations and, in particular, their pulmonary symptoms. © 1999 Elsevier Science Ltd. All rights reserved.

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1. Introduction

The cognitive model of panic proposed by Clark (1986) has been extremely influential in the empirical study of panic. According to this model, panic results from the catastrophic misinterpretation of bodily or mental sensations. Specifically, in individuals with panic disorder, sensations commonly associated with anxiety are interpreted as much more dangerous than is actually the case. Not only are these sensations interpreted as dangerous, they are also thought to signal immediate danger (Clark, 1988). For example, an individual may experience breathlessness, heart palpitations, dizziness or other sensations associated with anxiety. These triggers lead to a state of increased apprehension, which in turn leads to an increase of anxiety provoked bodily sensations. These additional sensations are then interpreted in a catastrophic manner. This process builds upon itself with increasingly greater levels of anxiety (Craske & Barlow, 1993). The result is a vicious cycle of sensations, apprehensions and catastrophic thoughts that eventually culminate in a panic attack.

Anticipatory anxiety, a fear of additional panic attacks, may predispose a person to future panic attacks. The model of Clark (1986) accounts for panic attacks that occur with anticipatory anxiety by suggesting that the anxiety leads to an increased focus on bodily functioning, thus triggering the panic cycle. A hallmark of panic, however, is that panic attacks frequently occur unexpectedly or ‘out of the blue’. According to the cognitive theory, attacks that occur without anticipatory anxiety are triggered by bodily sensations from an emotional state other than anxiety.

Cox (1996) expanded upon Clark’s cognitive model by suggesting an interactional view of panic etiology. He theorized that an individual’s susceptibility to the development of panic is determined by the interaction of a trait and congruent trigger. The trait, a cognitive disposition (e.g. state anxiety) representing specific, ideographic vulnerabilities, may involve beliefs associated with bodily symptoms as well as concerns about other areas (e.g. social evaluation). Each individual’s cognitive disposition will interact only with certain triggering stimuli. Cox suggests that, although the trigger may be external or internal, it must connect in a ‘meaningful’ way with the trait. For instance, the belief that dyspnea has negative health consequences will be triggered most often by shortness of breath and not by feelings of dizziness or other symptoms often associated with panic. The subjective interpretation of the trigger is clearly important. Ultimately, the individual must accept or reject the association between the trait and trigger. Once an association has been made, the person may then develop catastrophic cognitions and continue on to develop a panic attack.

While this cognitive theory of panic is presently very popular, it is not without its critics. Some more persistent challenges to cognitive models come from biomedical models that stress the importance of respiratory distress in the development of panic.

1.1. Dyspnea–fear theory

Ley (1989, 1994) proposed the dyspnea–fear theory of panic, in which he suggested that hyperventilatory panic attacks arise from an innate emotional response to severe dyspnea or shortness of breath. In this model, fear experienced in the context of breathlessness is given
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