

Invited commentary

## Challenges to conventional thinking about mind and body

The notion that physical symptoms of sane individuals may sometimes bear little or no relationship to measurable physiological functioning is counterintuitive to most of us, whether we are lay people, psychologists, physicians or even psychosomatic experts. Such an idea profoundly violates nearly everyone's ordinary view of reality and challenges our sense of safety in the world: If we cannot trust those filtered cortical transmissions telling us that something is happening to our body, what can we believe? If our bodily perceptions are so error-prone, how do we know when we ought to be truly alarmed by somatic symptoms? If we possess a nervous or fearful disposition, does this mean we are doomed to 'invent' or 'amplify' physical symptoms and are even more unable than less anxious types to distinguish between genuine physiological alterations and misperceptions?

Precisely, these kinds of issues are raised by the research in this issue by Houtveen et al. The authors report that the extent of self-rated somatic symptoms during mental stress and CO<sub>2</sub> rebreathing was generally unrelated to a variety of cardiovascular, autonomic and ventilatory measures, but it was, on the other hand, strongly and positively associated with trait anxiety and a history of physical complaints. These findings correspond to a number of studies that have found no exaggerated physiological reactivity to relevant laboratory stressors among individuals marked by a heightened proclivity to physical complaints and/or chronic anxiety. However, it remains unclear why still other investigations of anxiety accompanied by extreme physical symptoms, as in panic disorder, sometimes do find concurrent evidence of atypical physiological levels or reactivity.

Do these other findings represent real distinctions between normal individuals and patients with distinct types of anxiety disorder, as suggested by differences in laboratory studies of cardiovascular autonomic control between panic patients and normal controls (e.g., Ref. [1]), or by Klein's suffocation alarm theory of panic disorder [2] in which impaired central regulation of respiration is proposed as a primary cause of the disorder? Or could it merely be that most studies have taken place not in the real world, but in a laboratory environment, often without sufficient time for subjects to adapt themselves to the unfamiliar setting and physiological measurement devices. One might reasonably assume that particularly under non-adapted conditions, chronically anxious patients may display greater laboratory physiological responses than

nonanxious controls, but that these variations may not necessarily reflect real-life, neurophysiological differences between the groups, as so often suggested in the literature. Regarding panic disorder, such laboratory specificity of response is supported by several studies: For example, Stein and Asmundson [3,4] found no autonomic or cardiovascular differences during rest or challenge between panic patients and controls after they employed an extended acclimatization phase. An ambulatory study of panic disorder patients and healthy controls also found no differences in cardiac activity between groups under real-life conditions [5]. Still other research reviewed by Houtveen et al. indicates that even under typical laboratory conditions, highly relevant stressors, such as public speaking, often produce no exaggerated physiological reactions among social phobics, although somatic symptom reporting and anxiety are greatly elevated. Furthermore, even when we consider those publications where differences between patient and control groups are reported, there are always large proportions of the patient groups who produce no atypical physiological baseline levels or reactions to stressors, although subjective reports of physical symptoms and anxiety are consistently elevated among these patients. When such diverse evidence is considered, incongruence between physiology and experience of anxiety and somatic complaints seems very credible. But how can we understand this lack of concordance between physiology and symptomatology?

In general, the literature has primarily focused upon the cognitive-behavioral explanation that chronically anxious individuals manifest a tendency toward increased attention to and amplification of bodily symptoms related to consequences of negative, often catastrophic appraisal of somatic sensations. However, equally plausible is the possibility that states of anxiety actually alter the central nervous system circuitry so that visceral perceptions are, indeed, more unpleasant or painful. Subsequent negative appraisal and aversive responses to bodily sensation may be epiphenomenal to this altered central processing of somatic sensation. Increasing support for this second hypothesis has been gathered by imaging and pharmacological studies in recent years, especially by investigations examining relations between pain and anxiety. Interactions between serotonergic and endorphinic systems, and chemical-perceptual mapping of cortical regions, provide evidence of central links between anxiety and interoceptive

experience [6,7]. Also, emotion-related limbic functioning has been tied to central sensory up-regulation [8], and specific hippocampal networks that exacerbate pain under anxiety-provoking conditions have been identified [9]. These are just a few examples of the growing body of evidence that indicate that the experience of unpleasant or painful bodily sensations does, in fact, reflect *accurate perception* during anxious times, despite the lack of heightened peripheral physiological activity.

Should such an intimate connectedness of emotion and somatic experience continue to be supported by future investigation, we may need to rethink current ideas about mind–body relations. At least in terms of interpreting subjective experience of psyche and soma, it may be useful to reexamine pre-Cartesian, even ancient Buddhist views of an inseparable mind/body, in which emotional state (among other things) centrally conditions our every perception. For example, the simple wisdom: “When an ordinary person is contacted by a painful feeling, he feels two feelings — a bodily one and a mental one” [10]. In this light, one may gain courage to attempt to answer the questions posed at the beginning of this commentary: Unpleasant somatic symptoms may always tell us something is awry, but only sometimes is peripheral physiological functioning the problem. Paying close and impartial attention to our bodies during emotional states may also teach us that we store much of our emotional dysphoria in our bodily sensations, even if these sensations are mainly located at the top of our heads: We ought not to forget that physical symptoms constitute major diagnostic criteria for almost all clinical psychiatric disorders, and these symptoms are rarely accompanied by convincing evidence of actual physiological dysfunction. Studies like that of Houtveen et al. force us to step out of our perhaps dated scientific paradigms and consider other ways of understanding the embodied mind.

## References

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