

The Misidentified Threat: When the Brain Cannot Read the Body

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The proposition we defend

Fear can originate from a disagreement between bodily signals and the brain's interpretation of them. We argue that some recurrent anxiety states are disorders of interoceptive inference: benign or ordinary peripheral signals acquire exaggerated threat significance, provoking autonomic changes that make the next signal even harder to interpret calmly.

An organism can experience fear without an external threat when internal bodily signals are interpreted as dangerous. The new synthesis asks whether altered interoceptive precision, autonomic variability and learned predictions can lock a person into recurrent physiological alarm even when the peripheral signal is benign.

Why the mechanism deserves to be taken seriously

Baroreceptors, chemoreceptors, vagal afferents and spinal visceral pathways report internal state to the brainstem, insula and related networks. Predictions about body sensations influence what the person notices and how they respond. Hyperventilation, palpitations or gastrointestinal sensations can be both consequences and triggers of fear, creating a loop between appraisal and autonomic output.

The causal argument

The brain does not passively read heart rate, gut movement and breathing. It estimates what those inputs signify in the context of prior learning, uncertainty and current physiological state. When sensory precision is low or threat priors are unusually strong, normal fluctuations can be classified as emergencies. The resulting sympathetic activation increases pulse, muscle tension and ventilation, furnishing more intense sensory input and confirming the original interpretation. This is a mechanistic feedback model involving actual circulation and neural prediction—not a claim that symptoms are invented.

Causal architecture under closer examination

The interoceptive system does not simply receive objective measurements and announce their meaning. It combines uncertain bodily input with expectations about what that input usually signifies. Someone who has experienced frightening palpitations may assign unusually high danger to future normal cardiovascular variation. This can initiate a cascade of sympathetic activity, ventilation changes, muscle tension and attentional narrowing. At that point the body is genuinely aroused, even though the external or internal threat may have been misclassified. A mechanistic model should specify three stages: the initial sensory fluctuation, the inferred threat and the physiological amplification triggered by that inference. If the original fluctuation was pathological—arrhythmia, low blood glucose, orthostatic hypotension—the first stage needs conventional medical treatment. In a benign-input case, a learned predictive process may instead be maintaining alarm. The theory therefore demands better physiological measurement, not less. It rejects both the idea that the body has no role in anxiety and the idea that every intense bodily sensation proves dangerous disease.

A concrete physiological case

A patient experiences palpitations when rising from a chair and develops intense anticipatory fear. For one person, orthostatic measurements may reveal significant hypotension or tachycardia requiring medical assessment; for another, comparable sensations arise without dangerous cardiovascular change and are followed by escalating threat appraisal. Only the second pattern clearly supports a primary interpretation loop, and even then the investigation should include medication, endocrine and cardiopulmonary causes.

Original conceptual illustration

A harmless increase in heart rate can become alarming to someone who interprets it as impending collapse; that fear may increase adrenergic output and magnify the sensation. But a genuine arrhythmia can produce a similar subjective experience. The investigative priority is to determine which physiology is present and whether prediction about the sensation contributes additional amplification. The model must be capable of recognizing both possibilities.

What established physiology already supports

Interoceptive processing and autonomic feedback are established topics in affective neuroscience. What this thesis adds is a testable dynamic claim: excessive threat precision precedes and amplifies physiological alarm, then learning stabilizes the loop. A useful account must predict why one interoceptive signal triggers panic while similar bodily signals in another setting do not.

The strongest counterargument—and the answer

The main rival is a genuine occult physiological threat. Dismissing palpitations, dizziness or dyspnea as “misread signals” without evaluation is unsafe. Conversely, finding one benign physiological fluctuation does not prove a primary anxiety disorder. We must demonstrate that the interpreted threat exceeds the observed bodily danger and that modification of inference changes the trajectory.

Cardiac arrhythmias, thyroid disease, hypoglycemia, pulmonary disease and medication effects may produce genuine dangerous bodily signals. A predictive-processing framework should never be used to psychologize symptoms before appropriate differential diagnosis. Nor does every sensation arise from inaccurate perception.

The discriminating experiment

Use tasks that experimentally vary interoceptive uncertainty while recording cardiovascular and respiratory responses. Compare prediction error, reported threat and objective physiology over repeated trials. The theory predicts that shifts in learned expectation can reduce alarm even when the benign peripheral stimulus is held approximately constant. It also predicts failure where unrecognized physiological instability remains the primary driver.

One could compare symptom reports with objective physiology across controlled, clinically safe conditions and determine whether perception remains disproportionate after known medical causes are addressed. The model gains force if individual prediction errors prospectively forecast symptom escalation; it weakens if symptoms consistently correspond to identifiable untreated pathology.

Decisive comparisons and limits of inference

A discriminating experiment could vary expectations while delivering a safe, standardized bodily stimulus. Participants might receive accurate information about a transient heart-rate change or a different framing designed to alter perceived threat without changing the underlying stimulus magnitude. Researchers could then measure anticipatory physiology, reported fear and subsequent bodily amplification. The most important endpoint is the interaction between prior expectation and measured response. If threat appraisal changes the physiological loop at equal peripheral input, the inference mechanism has causal force. If the response tracks only genuine

cardiovascular instability, the predictive-processing account may be secondary or irrelevant. Studies also need to test persistence: does repeated benign exposure change future alarm thresholds? Does a person continue to expect catastrophe despite repeated safe outcomes, suggesting exceptionally sticky priors? Such research could clarify why some individuals develop recurrent panic around ordinary sensations while others do not. It would not erase real dysautonomia, endocrine disease or trauma-related symptoms. The bold argument is that a nervous system can become trapped in misinterpreting its own operating signals; that mistaken classification can then create physiological consequences that confirm the original belief.

A second prediction from the original model

The key prediction is not simply that anxious people perceive their bodies differently. It is that a measurable mismatch between physiological state and perceived threat predicts recurrent symptoms and that reducing the mismatch changes both interpretation and function.

Why the hypothesis matters if correct

The model's key feature is amplification of a real response from an inaccurate interpretation. A small benign bodily fluctuation is assigned dangerous meaning; sympathetic and respiratory activity then alter the body; the new sensations appear to confirm the original threat estimate. This loop predicts that expectation and sensory precision will influence the eventual magnitude of the physiological response, not just a person's verbal report. A strong experiment could hold a safe peripheral stimulus reasonably constant while varying what participants expect it to mean and measure autonomic amplification. If there is no effect of prior expectation and only objective physiological instability predicts alarm, the interpretation theory is less useful for that subgroup. This gives a direct way to distinguish an interoceptive disorder from a missed cardiac, endocrine or neurological disease. The larger implication is that bodily medicine and psychological medicine cannot always be separated. A misclassification in perception can initiate an objective, self-reinforcing physiological event, while a genuinely dangerous bodily event can be wrongly dismissed if the model is applied carelessly.

The clinical and conceptual consequence

The thesis challenges the line between bodily disease and emotion. A false alarm is still a real bodily event once initiated. Successful intervention may require correcting both the signal's physiological source and the brain's threat model rather than choosing one side of that false divide.

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Source bibliography: verification pending

- Seth AK, Critchley HD. Extending predictive processing to the body: emotion as interoceptive inference. Behav Brain Sci. 2013.
- McEwen BS. Stress, adaptation, and disease: Allostasis and allostatic load. Ann N Y Acad Sci. 1998.

Editorial and clinical boundaries

This paper advances a causal thesis and identifies observations that would support or defeat it. It does not establish a new diagnosis or treatment, and it should not be used to discontinue medically indicated care. Bibliographic entries inherited from the original edition have not all been independently verified.