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Somatic Marker Theory-Based Integrated Model of Bottom-up Psychosomatic Symptom Formation from Body to Symptoms

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Abstract: The mechanisms underlying the development of clinical psychosomatic symptoms have long been predominantly studied through a 'top-down' cognitive-emotional pathway. However, a large number of patients presenting with somatic discomfort as their chief complaint, which cannot be explained by organic pathology, exhibit the opposite process: abnormally amplified somatic sensations, lacking effective emotional markers, gradually solidify into persistent psychosomatic symptoms. Building upon a systematic review of somatic marking theory, interoception theory and emotional construction theory, this paper proposes an integrative bottom-up theoretical model that divides the development of psychosomatic symptoms into four stages: perceptual abnormality, marking deficiency, integrative dysfunction and symptom consolidation. Finally, the practical implications of the model are discussed in terms of clinical differentiation, doctor-patient communication and psychotherapeutic strategies.

1. Introduction

Psychosomatic symptoms refer to clinical phenomena triggered or exacerbated by psychosocial factors, yet primarily manifested as physical discomfort. Typical examples include functional gastrointestinal disorders, chronic non-specific pain, and cardiovascular reactions during panic attacks [1]. In clinical practice, patients with such conditions often seek repeated consultations across various departments in general hospitals, yet no clear evidence of organic pathology is found. Regarding the psychological mechanisms underlying such phenomena, there has long been an implicit 'top-down' bias.

The so-called 'top-down' bias refers to the tendency among researchers to assume that emotional or cognitive evaluations first occur at the level of the cerebral cortex, subsequently influencing peripheral organs via the hypothalamic-pituitary-adrenal axis or the autonomic nervous system, ultimately resulting in somatic symptoms. For example, the classic cognitive model of anxiety posits that an overestimation of threats leads to heightened physiological arousal [2]; the Health Belief Model, meanwhile, emphasises that patients' catastrophic cognitions regarding their illness amplify negative interpretations of normal bodily signals [3]. This perspective has undoubtedly garnered substantial empirical support and forms the theoretical cornerstone of cognitive

behavioural therapy. However, clinical observations reveal a relatively overlooked fact: for a large number of patients, it is not that 'worry' precedes the onset of somatic symptoms, but rather that they first experience an indescribable, vague sense of physical abnormality—only subsequently developing anxiety about this sensation and engaging in repeated medical consultations [4]. In other words, chronologically, the abnormal variation in bodily sensations precedes cognitive evaluation, rather than the reverse. This potential 'bottom-up' pathway has not yet been systematically elaborated within existing mainstream psychosomatic theoretical frameworks. The aim of this paper is precisely to address this gap. Using the classic somatic marker theory as a framework, we will integrate the latest advances in interoception theory and emotion construction theory to construct a theoretical model of the 'bottom-up' mechanism underlying the formation of psychosomatic symptoms.

2. Theoretical Review

2.1 The Theory of Bodily Markers

The theory of bodily markers was proposed by neuroscientist Damasio in 1994 to explain how emotions play a role in decision-making [5]. The core tenet of this theory is that when individuals face complex situations, bodily states associated with past emotional experiences (such as changes in heart rate, muscle tension, and visceral sensations) are reactivated to guide behavioural choices through a 'marking' mechanism. Subsequent research has shown that these somatic markers can influence responses both consciously and unconsciously [6].

The most significant contribution of the somatic marker theory to mind-body research lies in its explicit recognition that somatic states themselves constitute signals of informational value, rather than merely passively accepting cognitive regulation. However, the theory is primarily used to explain decision-making and risk-taking behaviour, and has rarely been systematically applied to the field of psychosomatic symptom formation. Damasio focuses primarily on the adaptive functions of 'normal marking', whilst devoting little attention to 'how abnormal marking leads to pathological symptoms' [7].

2.2 The Theory of Interoception

Internal sensation refers to the process of sensing, interpreting and integrating the body's internal states. Craig's model of the internal sensation cortex suggests that physiological signals from the whole body are projected via the spinal cord–brainstem–thalamus pathway to the insular cortex, forming neural representations of internal sensation [8]. In recent years, predictive coding theory has been introduced into research on internal sensation, emphasising that the brain continuously generates predictions of bodily states and compares these predictions with actual incoming signals: the smaller the prediction error, the more stable the internal sensation; conversely, this may give rise to a subjective 'sense of bodily discomfort' [9].

The association between interoception theory and psychosomatic symptoms has received preliminary empirical support. For example, patients with functional somatic syndromes (such as irritable bowel syndrome and fibromyalgia) often exhibit higher interoceptive sensitivity, but simultaneously lower interoceptive accuracy [10]. However, the interoception theory focuses primarily on perceptual-level processing and has not yet fully explained 'how an abnormal interoceptive signal ultimately solidifies into a stable psychosomatic symptom'.

2.3 The Theory of Emotional Construction

The theory of emotional construction, proposed by Barrett, emphasises that emotions are the result of a joint construction by the core affective system and the conceptual system [11]. This theory posits that changes in interoception constitute the neural basis of core affect (the valence-arousal dimension), whilst individuals rely on acquired emotional concepts to categorise these core affective experiences into specific emotional states. Subsequent research has further revealed that the availability of emotional concepts directly influences an individual's recognition and expression of bodily sensations [12].

Applying the constructivist theory of emotion to psychosomatic symptoms leads to an important inference: when an individual lacks available emotional concepts to interpret abnormal bodily signals, these signals are not experienced as a specific emotion, but rather as a 'nameless sense of discomfort'. Such bodily abnormalities lacking clear emotional attribution are precisely the hallmark of the 'somatisation' phenomenon observed in clinical practice [13].

In summary, the three theoretical frameworks contribute respectively: somatic markers—the informational value that endows sensations with orienting meaning; interoception—the explanation of the generation and abnormal amplification of sensory signals; and the theory of emotional construction—the explanation of the process of classifying and naming signals. However, the task of integrating these into a longitudinal 'bottom-up' pathological pathway remains unaddressed.

3. Construction of the Core Model: The Four Stages of the Bottom-Up Pathway

Drawing on the aforementioned theoretical resources, we propose a four-stage model of the bottom-up pathway underlying the formation of psychosomatic symptoms. The model's fundamental assumption is that not all psychosomatic symptoms originate from top-down cognitive-emotional influences; a significant proportion of symptoms begin with abnormalities in interoceptive signals at the peripheral or central levels, which, following dysregulation in the marking and integration stages, ultimately solidify into clinical symptoms.

3.1 Stage One: Perceptual Abnormalities

This stage begins with abnormal deviations in the intensity, frequency, or quality of somatic sensory signals. Possible sources include: increased visceral sensitivity [14], weakened sensory gating in interoceptive pathways [15], or the failure of interoceptive predictive models [9]. At this stage, the individual subjectively experiences a sense that 'something is wrong', but has not yet assigned a specific name or emotional valence to this sensation. In most healthy individuals, transient sensations of bodily abnormality resolve spontaneously, whereas susceptible individuals may progress to the next stage.

3.2 Stage Two: Lack of Labelling

When abnormal bodily signals persist or recur, the brain must identify and categorise them. Under healthy conditions, the signal is matched against an existing repertoire of emotional bodily labels [5]. However, in individuals susceptible to psychosomatic symptoms, two patterns of labelling deficit may emerge: under-labelling (where the signal fails to match any existing somatic label, becoming an 'unnamed bodily sensation') and mislabelling (where the signal is erroneously assigned to an irrelevant label category, such as labelling an interoceptive alert as 'a problem with the heart') [16]. The labelling deficit stage is a central switching point in the bottom-up pathway and aligns closely with the clinical phenomenon of alexithymia [17].

3.3 Stage Three: Integration Dysfunction

Building upon the labelling deficit, abnormal somatic signals cannot be successfully integrated into the individual's overall self-perception and situational interpretation framework. This stage involves two key integration systems: temporal integration (the inability to link current abnormalities with past similar experiences, with each instance feeling like a 'completely new catastrophe') and situational integration (difficulty attributing bodily sensations to external or psychological causes, with a tendency to attribute them to organic disease) [18]. The consequence of integration dysregulation is that bodily signals become a persistent, threatening focus requiring constant attention. This corresponds to the psychological state in which patients repeatedly seek medical attention and undergo various tests, yet are unable to accept the explanation that 'everything is fine' [19].

3.4 Stage Four: Symptom Consolidation

Following repeated cycles through the first three stages, abnormal bodily perception – lack of labelling – integration dysfunction form a self-reinforcing positive feedback loop [20]. Ultimately, this process solidifies into stable, identifiable 'psychosomatic symptoms'. The symptom consolidation stage involves changes in neuroplasticity: functional connectivity in brain regions such as the insula, anterior cingulate cortex and prefrontal cortex is reorganised, making bottom-up pathways the default, automated processing mode [21]. At this stage, symptoms can persist independently even after the initial organic trigger has disappeared.

4. Distinctions and Connections with Existing Models

4.1 Complementarity with Cognitive Appraisal Theory

The classic Lazarus cognitive appraisal theory posits that individuals first conduct primary and secondary appraisals of stimuli [2]. The present model suggests that, within the bottom-up pathway, the occurrence of appraisal depends on a prerequisite—namely, that the somatic sensation has already been preliminarily labelled as an object with clear meaning. If this labelling is absent, cognitive appraisal loses its material foundation. Consequently, this model serves as a supplement to the 'pre-cognitive conditions' of the cognitive appraisal theory.

4.2 Two Sides of the Same Coin with the Theory of Emotion Construction

Barrett's theory of emotion construction emphasises that emotions are the product of the interaction between changes in interoception and the conceptual system [11]. The model presented in this paper essentially constitutes a concretisation of this perspective within the context of psychosomatic pathology: we have clearly distinguished between 'changes in internal sensations' (Stage 1), 'concept/marker matching' (Stage 2), and 'integration and meaning construction' (Stage 3), and proposed a cumulative pathological pathway. It can be said that this model represents an extension of the theory of emotion construction towards the concept of 'failed construction'.

4.3 Application and Extension of the Theory of Somatic Marking

Damasio's theory of somatic markers primarily explains adaptive decision-making [5]. We have transplanted its core concept—the 'marker'—from the domain of decision-making to that of symptom formation, and have distinguished between two pathological modes: 'marker deficiency'

and ‘marker error’. This transplantation is valid because, whether in decision-making or symptom interpretation, individuals face the common task of ‘attributing meaning to the current state to guide action’.

4.4 The Nested Relationship with the Endo-Predictive Coding Model

Within the endo-predictive coding framework, symptoms are considered to arise from a mismatch between highly accurate prior beliefs and incoming signals [9]. Our model describes in greater detail what occurs following this ‘mismatch’: if the brain lacks adequate marking resources to update predictions, the mismatch is not only not corrected but actually intensifies the abnormal focus on somatic signals.

5. Clinical Implications

5.1 Reinterpreting the Experiences of Patients with “No Organic Lesions”

Clinically, patients often complain, “The doctor says there’s nothing wrong with me, but I really feel terrible.” From the perspective of this model, the patient’s feelings are genuine—their phase of perceptual abnormality has already been triggered. Therefore, doctors should reframe “no organic disease” as “current examinations have not revealed any tissue damage, but we still need to pay attention to how your bodily sensations are being processed by the brain” [1][19]. This phrasing neither denies the patient’s subjective experience nor closes the door to psychological intervention.

5.2 Label-assisted strategies for doctor-patient communication

Stage Two (marker deficiency) suggests that providing appropriate linguistic markers for patients’ vague physical discomfort is, in itself, a form of treatment. For example, when a patient says, “I have an indescribable discomfort in my stomach,” the doctor can ask about specific sensory characteristics (such as a burning sensation, churning sensation, or feeling of pressure). This helps patients link vague signals to a repertoire of bodily markers [16][17].

5.3 Tiered Psychotherapy Recommendations

Based on the four stages of this model, psychological interventions can be targeted more precisely: Perceptual Anomaly Dominance: Introsensory awareness training and introsensory exposure therapy are recommended [23]. Labelling Deficit Dominance: Emotional labelling training and somatic labelling reconstruction are recommended [17]. Integration Dysfunction Dominance: Cognitive reappraisal and situational attribution training are recommended [18]. Symptom Consolidation Stage: Interventions based on inhibitory learning and functional reintegration should be introduced [22].

6. Limitations and Outlook

6.1 Limitations of the Model

Idealisation of stage division. Real psychological processes do not progress strictly through four linear stages but are often cyclical [20]. The model provides a simplified treatment of peripheral factors. It focuses on central processing mechanisms, with insufficient integration of peripheral sources such as the immune system, endocrine system, and gut microbiota [14].The model offers inadequate explanation of individual differences. Why do some individuals tend towards bottom-up

pathways, whilst others favour top-down pathways? This may be related to the body schema library formed during early attachment relationships [7].There is a lack of analysis regarding gender and culture [13].

6.2 Directions for Future Research

First, neuroimaging validation: use resting-state functional connectivity and task-based fMRI to examine brain activation patterns corresponding to the four stages [21]. Second, developmental and longitudinal studies: does repeated exposure to unmarked bodily distress during childhood permanently alter the connection patterns between interoception and marking [4]? Third, validation of intervention targets: Design micro-intervention experiments to manipulate each of the four stages separately and observe the impact on symptom reports [23]. Fourth, cross-diagnostic applications: Test the model's applicability to panic attacks, eating disorders, and other conditions [1][10].

7. Conclusion

Starting from genuine clinical dilemmas and using the theory of somatic marking as a framework, this paper integrates the theory of interoception with the theory of emotional construction to construct a four-stage, bottom-up pathway model of the formation of psychosomatic symptoms. This model is not intended to deny classical top-down cognitive mechanisms, but rather to fill a long-neglected dimension. For researchers with backgrounds in both medicine and psychology, this model can effectively bridge the gap between 'the doctor's listening' and 'psychological theory-building'. We sincerely hope that this theory will inspire further empirical testing and ultimately help patients who are 'tormented by suffering despite no identifiable medical cause' to receive more empathetic understanding and more precise interventions.

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