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Psychosomatic concepts in nociplastic pain: The case of burning mouth syndrome

We read with interest the recent perspective proposing that burning mouth syndrome (BMS) be reconsidered as a nociplastic pain disorder, given the growing evidence of both peripheral nerve dysfunction and central sensitization.¹ We largely concur that BMS, highlighting its dual etiology involving both peripheral nerve dysfunction and central sensitization, accurately reflects these underlying mechanisms.

However, we urge caution in broadly applying the term “nociplastic.” Overextending this concept in clinical settings may lead to diagnostic ambiguity and confusion regarding treatment strategies. As noted by Kitahara, the risk lies in conflating varied pain mechanisms under a single label without sufficient stratification of individual patient presentations.² Rigorous criteria and clarity of terminology are paramount to prevent misdiagnosis and to tailor interventions more effectively.

Our group has emphasized that pain arises from a continuous interplay of peripheral and central processes, as highlighted by Bushnell and colleagues.³ Pain is not merely the result of ascending nociceptive signals; it also reflects the dynamic descending modulation from higher brain centers, including the prefrontal cortex and anterior cingulate cortex. These regions can either amplify or suppress pain signals, shaping an individual’s pain experience. In chronic pain states like BMS, adaptive changes occur not only at peripheral nociceptors but also throughout the brain, suggesting that peripheral and central mechanisms intersect in a mutually reinforcing manner. Consequently, BMS cannot be reduced to a simple dichotomy of peripheral versus central mechanisms. Rather, its pathophysiology appears to be dynamically modulated at multiple levels. Recognizing this complex feedback loop underscores the importance of an integrative approach when diagnosing and managing BMS.

Regarding BMS as an oral psychosomatic disorder makes it evident that psychosocial factors are deeply woven into the pathophysiology of chronic pain. Kitahara underscores the need for a biopsychosocial model—biological,

psychological, and social elements interact to influence both the onset and persistence of chronic pain.² Toyofuku similarly advocates for management approaches that address psychosocial dimensions alongside biomedical interventions—so-called psychosomatic approaches.⁴ Notably, the concept of nociplastic pain—emphasizing alterations in pain processing rather than straightforward tissue damage—closely aligns with psychosomatic principles, as both acknowledge that psychological stressors, emotional states, and social contexts significantly shape how pain is experienced. Such comprehensive, multidisciplinary care has the potential to improve patients’ quality of life by addressing stress, mood, and interpersonal factors that often exacerbate or maintain chronic pain. Incorporating psychological support, patient education, and social resources can be particularly beneficial in chronic or treatment-resistant cases.

In summary, we support the authors’ perspective that BMS embodies features of both peripheral and central sensitization, in alignment with nociplastic pain principles. Nevertheless, the term “nociplastic” must be applied judiciously to avoid oversimplification. Future investigations should integrate a biopsychosocial framework and clarify diagnostic benchmarks, ensuring that patient-centered, individualized strategies guide the management of BMS. By combining precise diagnostic criteria with holistic care, clinicians can more effectively address the multifaceted nature of this chronic pain disorder.

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

Acknowledgments

There is no funding for this study.

<https://doi.org/10.1016/j.jds.2025.04.020>

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Received 17 April 2025

Final revision received 19 April 2025

Available online 30 April 2025