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Letter to the editor

The enigma of burning mouth syndrome: Insights into dual pain mechanisms

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Burning mouth syndrome (BMS) is a complex condition characterized by chronic orofacial pain without apparent organic cause, however the exact etiology remains elusive. Recent findings suggest presence of dual pain mechanisms within BMS, emanating from both peripheral and central pathways.

Zhang et al.[1] indicate that peripheral pain in BMS may stem from a localized inflammatory response, characterized by heightened pro-inflammatory cytokines, notably interleukin-1β (IL-1β) and interleukin-8 (IL-8). Furthermore, transient receptor potential (TRP) channels (TRPV1 and TRPA1),[2] expressed on nociceptive nerve fibers, potentially assume a pivotal role in perceiving thermal and chemical pain. Neuropeptides, such as substance P and calcitonin gene-related peptide (CGRP), actively contribute to peripheral sensitization in BMS, nurturing neurogenic inflammation and amplifying pain sensitivity.

Concurrently, elevated levels of tumor necrosis factor-alpha (TNF-α) in saliva, particularly in BMS patients with concurrent anxiety and depression, indicate a central role for neuroinflammation. Central sensitization, a characteristic feature of chronic pain, may arise from the complex interplay between TNF-α and other cytokines, fostering a state of heightened pain perception, encompassing hyperalgesia and allodynia.[3] Hormonally, correlation between psychological distress and BMS pain underscores the influence of stress-induced hormonal fluctuations, encompassing dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and cortisol secretion.

Remarkably, central sensitization in BMS may employ distinct molecular pathways that operate independently of endogenous pain modulation, potentially contributing to development of chronic pain. Nevertheless, it is crucial to acknowledge that not all BMS patients exhibit both peripheral and central mechanisms in tandem. Further research is imperative to unravel the nuances underlying these conditions.

Declaration of competing interest

The author has no conflicts of interest relevant to this article.

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References

1. Zhang Y, Ye S, Zhang Y, et al. Potential salivary and serum biomarkers for burning mouth syndrome and their relationship with anxiety/depression. *J Dent Sci* 2023 (in press).

https://doi.org/10.1016/j.jds.2024.01.015

2. Seol SH, Chung G. Estrogen-dependent regulation of transient receptor potential vanilloid 1 (TRPV1) and P2X purinoceptor 3 (P2X3): implication in burning mouth syndrome. *J Dent Sci* 2022; 17:8—13.
3. Gremeau-Richard C, Pionchon P, Mulliez A, Dualé C, Dallel R. Enhanced pain facilitation rather than impaired pain inhibition in burning mouth syndrome female patients. *J Headache Pain* 2022;23: 143.

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