

Stomatodynia—Burning Issues

Stomatodynia, also known as burning mouth syndrome (BMS), is a relatively common yet poorly characterized intraoral pain condition that especially occurs in postmenopausal women.¹⁻⁴ It manifests as a spontaneous burning pain that is generally constant and felt in the tongue, hard palate and/or lips, but there are usually no visible signs of any mucosal lesions, inflammation, or disease. It is frequently accompanied by impaired taste, oral dysesthesia, and a sense of xerostomia, and often coexists with other chronic pain conditions such as fibromyalgia and temporomandibular disorders.

Several etiological factors have been proposed, and include systemic factors such as certain nutritional deficiencies and hormonal dysfunction, local factors such as denture irritation and yeast infection, as well as psychogenic factors since many patients have high levels of anxiety or depression. None of these particular proposals have stood the test of time, and BMS remains an enigma. Nonetheless, progress has been made over the past two decades in defining the clinical and psychophysical features of the condition, including documentation that it may manifest alterations in pain, taste, or other sensory parameters, and most recently there have been reports of neuropathy affecting small-diameter oral nerve fibers in BMS patients (see ref 5) lending support to earlier concepts (see ref 2) that BMS may reflect a neuropathic pain condition. But what causes these neuropathic changes in the oral nerve fibers and the sensory parameters? Such a conundrum, coupled with several lines of evidence that altered levels of steroids may represent a predisposing factor for BMS, have led to the hypothesis outlined by Drs Woda, Dao, and Grémeau-Richard in the Focus Article⁵ in this issue of the *Journal of Orofacial Pain*. They posit that the interaction of adrenal, gonadal, and neuroactive steroids may induce neuropathic changes in small-diameter oral nerve fibers that result in the symptoms of BMS. The hypothesis proposes that chronic anxiety or posttraumatic stress triggers a disruption of the production of adrenal steroids followed by a decreased production of neuroactive steroids that is further aggravated by the marked fall in gonadal steroids that occurs around menopause or in aged individuals.

Woda et al in their Focus Article and Zakrzewska,⁶ Eliav and Nasri-Heir,⁷ and El-Etr⁸ in their Critical Commentaries highlight several questions that arise

from this hypothesis, eg, why are the symptoms manifested only in the oral cavity as a result of this cascade of events, why is it that not every postmenopausal woman with a major stress or anxiety disorder develops stomatodynia, and why is hormonal replacement therapy a relatively ineffective treatment? The four articles, and the Authors' Response,⁹ collectively note several research directions that can address these intriguing questions, as well as new approaches that could be tested for the management of this orofacial pain condition. Thus, like any hypothesis "worth its salt," the hypothesis by Woda et al is based on a plausible framework derived from several lines of investigation, proposes a variety of etiologic pathways that can be tested by a number of research avenues, and raises several questions that also are testable and that may lead to an improved understanding and management of this vexing condition.



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References

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