Steroid Dysregulation and Stomatodynia (Burning Mouth Syndrome)

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After many years of research, burning mouth syndrome (BMS) remains an enigma: the pathophysiology is not defined, there is no gold standard for diagnosis, and the treatment provided is not sufficient. Concepts such as that suggested in the Focus Article by Woda et al[1] may serve as a foundation for advanced research that may finally shed light on this pathology.

Before addressing the topic of this Focus Article, we would like to support the authors' decision to abstain from using the term burning mouth syndrome: the condition is definitely not a syndrome. The term stomatodynia used by the authors can serve as a suitable alternative, although some would prefer burning mouth disease.

The possible association between stomatodynia and hormonal levels is not a new theory. The fact that this condition is more common in women, particularly following menopause, triggered many clinicians to recognize this link. Surprisingly, only a limited number of studies, with inconsistent findings, addressed this issue. As a result, despite the logical correlation, this association has never been entirely proven. The Focus Article by Woda et al sharpens this connection and gives theoretical background to this intuitively suggested mechanism. However, several links are still missing and definitive research data are still needed, before this theory could grow to be the definitive mechanism for stomatodynia.

The major shortfall in the hypothesis may be the fact that the condition is limited to the oral cavity, while steroid dysregulation is a generalized condition. The authors are aware of this problem and suggest a possible relation to local diminution in neuroactive steroids level. As these hormones are locally active, this can indeed be an explanation. Nevertheless, the same question may rise again: why only in the oral cavity and not in other organs? It has been suggested that local nerve damage plays an important role in stomatodynia's pathophysiology.[2–4] It is plausible that this damage induces reduction in neuroactive steroids secretion in the affected site. Even so, the local nerve hypofunction theory is not in line with systemic steroid failure as the first and major cause initiating stomatodynia, as suggested by the authors.

Another point that needs to be clarified with the theory is hormonal replacement therapy for the treatment of oral discomfort or stomatodynia. While local administration of hormones has some alleviating effect,[5–7] systemic replacement therapy is reported as ineffective.[8,9] The effective local treatment can support the neuroactive steroid theory suggested by the authors. The ineffectiveness of systemic replacement therapy may be related to treatment onset. It is likely that the damage was already established and that earlier treatment could have been more efficient. There is no doubt that more research is necessary to address this issue.

Steroids play a major role in the physiology of many organ systems. Steroid depletion, therefore, should have a huge impact on many systems and should not be limited to the oral cavity only. Equally, due to the complexity and the various pathways that steroids act upon, steroid depletion can trigger oral burning sensation via additional mechanisms (in addition to depression and direct effect on the nerve), eg, altered glucose levels or damage to small blood vessels.

Further research should question whether steroid depletion in stomatodynia patients affects other organs as well. Another question to address is whether stomatodynia is more common among patients suffering from other conditions related to low steroid levels, eg, Addison disease.

The correlation between depression and stress disorders on one hand, and chronic pain on the other, is well known and has been studied extensively.[10–12] Stomatodynia is not unique; it is a chronic pain condition that may trigger or be triggered by the above-mentioned pathologies.[12,13] This, of course, does not rule out the possibility that the series of events suggested by the authors (steroids depletion/depression/neuropathic pain) does occur in stomatodynia patients.

The theory suggested by Woda et al[1] is interesting and may be applied to stomatodynia or other chronic pain conditions. Although the process hypothesized cannot be the sole mechanism, it definitely can be a major contributing factor, at least in a number of patients suffering from oral discomfort.

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