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MiniReview

# Brain-Heart Axis in Burning Mouth Syndrome: A Neurobiological Framework

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## Abstract

Burning mouth syndrome is a chronic nociplastic pain disorder characterized by a persistent intraoral burning sensation. While traditionally viewed as a localized or psychogenic condition, recent evidence suggests a systemic dysregulation of the brain-heart axis. This review synthesizes findings on autonomic nervous system impairment, evidenced by reduced heart rate variability and altered QTc intervals, and links these to hyperactive salience networks and hypoactive default mode networks. We discuss molecular mechanisms involving dopamine D2 and NMDA receptors, including drug-induced pathways (SSRIs/ACE-Is), and propose novel roles for glial-mediated neuroinflammation and Piezo-channel mechanosensing under conditions of increased intracranial pressure. This framework advocates for multimodal therapies targeting the interplay between central neural circuits and peripheral cardiovascular feedback.

**Keywords:** burning mouth syndrome, autonomic nervous system, central sensitization, pain catastrophizing, brain-heart axis, salience network, default mode network

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## 1 INTRODUCTION

Like many chronic pain disorders, burning mouth syndrome (BMS) is driven by central sensitization[1], where increased excitability of the central nervous system (CNS) amplifies pain signals. This results in a persistent burning sensation even in the absence of peripheral stimulation[1]. This neuroplasticity, or the nervous system's ability to reorganize itself, is one of the underlying factors behind the chronic nature of pain. BMS frequently co-occurs with psychiatric disorders such as depression and anxiety, suggesting a shared neurobiological basis[2]. Clinically, BMS predominantly affects postmenopausal women, with a prevalence ranging from 0.7% to 5% of the general population. It is characterized by a "triad" of symptoms: oral mucosal pain, dysgeusia (taste alteration), and xerostomia (subjective dry mouth), often following a circadian rhythm where pain intensifies throughout the day. The brain's "pain matrix" and areas responsible for emotion regulation, such as the amygdala and anterior cingulate cortex, are deeply interconnected[3]. Chronic pain acts as a stressor, potentially dysregulating these circuits and leading to mood disorders. Conversely, anxiety and depression may lower an individual's pain threshold, exacerbating BMS symptoms and creating a harmful feedback loop. This interaction between pain and psychological factors is mediated by the autonomic nervous system (ANS), which in turn can affect cardiac function[4]. Therefore, this paper provides a narrative review from the perspective of the relationship between the brain and heart in chronic pain as one way to explore the pathogenesis of BMS.

## 2 BMS: A CHRONIC OROFACIAL PAIN DISORDER

The pathogenesis of BMS is categorized into peripheral and central types. Peripheral BMS involves small-fiber neuropathy of the trigeminal nerve, while central BMS is linked to deficient endogenous pain-inhibitory systems. Key risk factors include female sex, menopause, vitamin

deficiencies (B12, folic acid), endocrine disorders (diabetes, thyroid dysfunction), and psychological stressors. BMS is formally defined as a chronic intraoral burning or dysesthetic sensation that occurs daily for more than two hours for more than three months, in the absence of an identifiable causative dental or medical lesion. It represents a form of chronic orofacial pain with a complex, often multifactorial etiology. A 2024 review highlights BMS as a significant contributor to the global burden of chronic orofacial pain, underscoring its impact on quality of life and the need for robust neurobiological models that extend beyond a purely localized or psychogenic explanation[5]. This recognition of BMS within the broader context of chronic orofacial pain strengthens the argument for a systemic model, such as the brain-heart axis (BHA), to fully understand its pathogenesis.

## 3 METHODS

The search for relevant articles was conducted across major academic databases, including PubMed, Scopus, and Google Scholar. The search terms were strategically chosen to capture the key concepts of the review. These included a combination of terms such as "burning mouth syndrome," "BMS," "autonomic nervous system," "ANS," "heart rate variability," "HRV," "central sensitization," and "chronic pain." Boolean operators (AND, OR) and phrase searching were used to refine the search and ensure comprehensive coverage.

Articles were selected based on their relevance to the core themes of the review: the link between BMS and autonomic dysfunction, the role of large-scale brain networks, and parallels with other chronic pain disorders. Priority was given to primary research articles, including clinical studies, neuroimaging studies, and reviews, that were published in peer-reviewed journals. The timeframe was not strictly limited, but a focus was placed on research published in the last 15 years to ensure the review reflects the most current understanding of the topic.

Exclusion criteria included: (1) Studies not published in English; (2) Editorials, letters, and opinion pieces that did not present original data; (3) Studies primarily focused on animal models without direct clinical relevance; and (4) Papers that addressed chronic pain but did not discuss a direct link to the ANS or large-scale brain networks.

From the selected articles, key findings were extracted, including study designs, participant demographics, methodologies (e.g., HRV analysis, neuroimaging), results, and conclusions. The synthesis process involved identifying common themes and contrasting findings across the literature. This has enabled us to construct a coherent narrative linking seemingly disparate research fields, from physiological markers of autonomic function to psychological factors and large-scale brain networks, and to build a comprehensive neurobiological model of BMS. By using this systematic yet flexible approach, the narrative review was able to integrate a diverse body of evidence to provide a holistic perspective on BMS's pathophysiology and its relationship to other chronic pain disorders.

## 4 AUTONOMIC DYSFUNCTION AND THE BRAIN-HEART AXIS IN BMS

Autonomic dysfunction, a core feature of BMS and other chronic pain disorders, is driven by a complex interplay between the brain's neural circuits and the heart's function. The brain's salience network and default mode network (DMN), influenced by maladaptive psychological factors and neurotransmitter imbalances, can disrupt the balance of the ANS, ultimately affecting heart rate and rhythm[6]. This results in a state of chronic sympathetic over-activation or parasympathetic withdrawal, creating a vicious cycle of pain and physiological dysregulation.

The brain and heart are in constant communication, with the ANS acting as the primary mediator[7]. The ANS has two branches: the sympathetic nervous system, responsible for the "fight-or-flight" response, and the parasympathetic nervous system, which promotes "rest-and-digest" functions. In a healthy state, these two branches are balanced, regulating heart function and maintaining heart rate variability (HRV)[8]. A high HRV indicates a flexible, healthy ANS, while a low HRV suggests an inability to adapt to changing demands, a key characteristic of autonomic dysfunction.

In BMS, this delicate balance is disrupted. Studies show that patients with BMS have significantly lower HRV and altered exhalation/inspiration ratios, indicating a global impairment of both sympathetic and parasympathetic systems[9]. BMS is associated with autonomic abnormalities, and therapeutic interventions such as stellate ganglion near-infrared irradiation have been shown to correct this dysregulation[10]. This systemic effect on heart function, measurable through electrocardiogram (ECG) and HRV analysis, is not unique to BMS. It's also a hallmark of other chronic pain conditions like fibromyalgia[11] and myofascial pain syndrome[12]. These conditions, like BMS, often present with an increased sympathetic response and a decreased parasympathetic response, leading to symptoms that parallel those of postural orthostatic tachycardia syndrome[13].

## 5 CARDIAC REPOLARIZATION AND THE QTc INTERVAL AS A MARKER OF SYMPATHETIC TONE

The QT interval, a measurement on the ECG, reflects the duration of ventricular depolarization and subsequent repolarization. Crucially, it serves as a sensitive, non-invasive indicator of ANS tone because the ANS directly controls both heart rate and the speed of cardiac repolarization. Increased sympathetic activity (the "fight-or-flight" response) tends to shorten the repolarization time, which

can affect the QT interval, while increased parasympathetic vagal activity slows the heart and can lengthen it. Thus, the corrected QT interval (QTc) is influenced by the precise balance between these two ANS branches. For instance, conditions characterized by a high sympathetic drive, like acute ischemic stroke, often exhibit a prolonged QTc interval. In the context of BMS, studies have observed that the hyperactive salience network activity, exacerbated by pain catastrophizing, can intensify sympathetic activation, leading to a shortening of the QT and QTc intervals[14]. This paradoxical shortening, rather than prolongation, suggests a profound dysregulation in the adrenergic signaling pathways specific to BMS, highlighting the QTc interval's value as a biomarker for sympathetic tone imbalance within the BHA.

## 6 BRAIN NETWORKS AND THE VICIOUS CYCLE

The root of this autonomic dysregulation lies in large-scale brain networks. The salience network, which alerts the body to important internal and external stimuli, becomes hyperactive in chronic pain[15]. This persistent "threat" signal drives the sympathetic nervous system into a continuous state of arousal, essentially keeping the body in a prolonged "fight-or-flight" mode. This hyperactivation is exacerbated by maladaptive psychological responses, such as pain catastrophizing, where patients focus excessively on and magnify their pain[16]. This cognitive distress can intensify the salience network's activity, further increasing sympathetic activation and shortening the QT and QTc intervals on an ECG in patients with BMS, which are markers of sympathetic tone[16].

In contrast, the DMN, which is active during periods of rest and is linked to the parasympathetic nervous system, shows reduced activity in BMS patients[17]. This diminished DMN function directly contributes to the decreased parasympathetic tone and low HRV observed in these individuals. The overall effect is a perpetual imbalance: a hyperactive salience network driving sympathetic arousal, coupled with a hypoactive DMN failing to promote parasympathetic rest.

## 7 DEEPENING THE BHA MECHANISM: PARALLELS WITH OTHER NEUROLOGICAL AND CARDIAC DISORDERS

The BHA involves a complex network of neural, hormonal, and inflammatory pathways that allow the brain and heart to communicate bidirectionally, critical for maintaining both cardiovascular function and brain health[7].

### 7.1 Insight from Takotsubo Syndrome

The mechanism of BHA dysfunction in BMS may share common pathways with other stress-induced disorders, notably Takotsubo Syndrome (TTS), also known as 'broken heart syndrome.' Both BMS and TTS are strongly associated with intense psychological stress and show a higher prevalence in middle-aged and postmenopausal women. While BMS is chronic orofacial pain and TTS is transient cardiac dysfunction, both conditions are hypothesized to be mediated by the autonomic and endocrine systems' response to stress. In TTS, excessive release of catecholamines due to stress triggers temporary left ventricular dysfunction. Crucially, the insular cortex (Ic) is a pivotal component of the central ANS and plays a central role in sympathovagal balance. Research suggests that the BHA in TTS, particularly in postmenopausal women, involves a sex hormone-specific neuroanatomy where decreased female sex hormones may alter functional cerebral asymmetry and lead to autonomic instability, with the Ic acting as a key moderator[18]. This parallel suggests that a similar dysregulation of central autonomic control centers, like the insula, possibly influenced by hormonal changes, may contribute to the autonomic dysfunction and chronic pain manifestation in BMS.

## 8 NEUROTRANSMITTER DYSREGULATION AND THE DOPAMINE LINK

This network-level dysfunction is underpinned by changes at the neurotransmitter and receptor level[19]. The N-methyl-D-aspartate receptor, a type of glutamate receptor, is a key player. Its overactivation leads to central sensitization, where the nervous system becomes more sensitive to pain signals[20]. This process contributes to the hyperactive state of the salience network and perpetuates sympathetic arousal.

Crucially, dopamine D2 receptors are also implicated. Given the observed similarities between BMS and Parkinson's disease[9], a condition characterized by dopamine deficiency, it is believed that a dysfunction in dopaminergic systems disrupts the brain's natural pain-inhibitory pathways. This breakdown of central inhibitory control, driven by impaired D2 receptor function, contributes to the chronic pain state. This link is further supported by cases of drug-induced BMS. Medications like selective serotonin reuptake inhibitors (SSRIs)[20] and angiotensin-converting enzyme inhibitors (ACE-Is)[21], despite their different pharmacological effects, both reduce basal ganglia dopamine. Both SSRIs and ACE-Is are the two main classes of drugs that induce BMS, and they both cross the blood-brain barrier to exert their pharmacological effects in the brain. The cortico-basal ganglia loop regulates the output of the dopaminergic neurotransmission to the cortex via the thalamus. As shown in Figure 1, SSRIs inhibit dopamine release at D2 receptors in the striatum by presynaptic inhibition, blocking the indirect pathway and decreasing dopaminergic output from substantia nigra pars reticulata (SNPR), potentially causing pain. In contrast, ACE-Is inhibit the enzymatic degradation of substance P, which activates D1 receptors and decreases dopaminergic output from SNPR via the direct pathway, also potentially causing pain. Thus, both SSRIs and ACE-Is have the potential to decrease

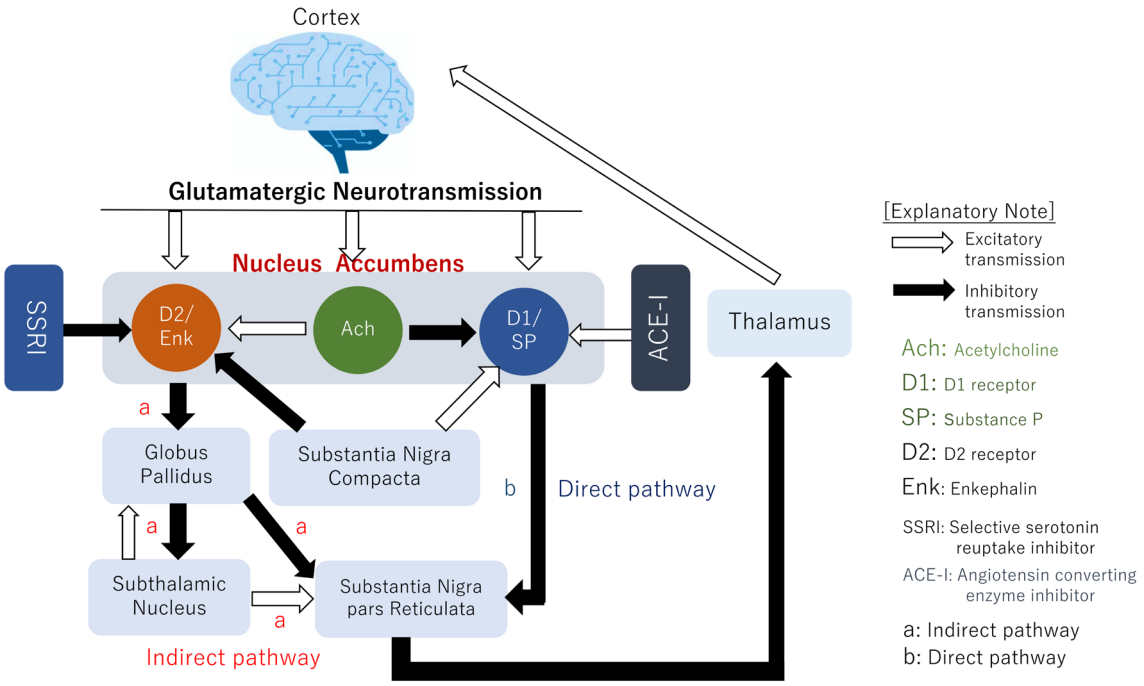


**Figure 1. The Cortico-Basal Ganglia Loop and Drug-Induced BMS. Schematic representation of the direct and indirect pathways.** (a) SSRIs enhance serotonergic inhibition of dopamine release at D2 receptors in the striatum (indirect pathway), leading to decreased output from the SNPR. (b) ACE-Is inhibit the degradation of substance P, which over-activates D1 receptors (direct pathway), similarly leading to decreased dopaminergic output. Both mechanisms converge on a reduction of central pain-inhibitory signaling.

dopaminergic output in the cortico-basal ganglia loop[22]. This model is supported by clinical observations where the withdrawal of offending drugs often leads to a gradual remission of symptoms, suggesting a reversible functional blockade of D2 receptors. Consequently, low-dose D2 receptor agonists have been hypothesized as a treatment; however, their clinical use remains limited by side-effect profiles, emphasizing the need for highly selective ligands. A study utilizing positron emission tomography to investigate dopamine D1 and D2 receptor function in the striatum of BMS patients discovered that D1 receptors remained unaltered, while D2 receptor function was diminished, and endogenous dopamine levels were lowered[23].

## 9 THE ROLE OF GLIA IN BHA-MEDIATED INFLAMMATION

A rapidly evolving area of BHA research is the role of glial cells—traditionally viewed as mere support cells—in mediating inflammation and dysfunction in both the brain and the heart. The BHA, and its associated pathologies, is increasingly recognized as being driven by a pro-inflammatory state in both the CNS and the peripheral nervous system. In the brain, microglial activation contributes to central sensitization and pain chronification. In the heart, the intrinsic cardiac nervous system is a complex network of neurons and cardiac glial cells[24]. These cardiac glia cells, which represent a heterogeneous cell population, are now understood to contribute significantly to the autonomic control of the healthy heart and, crucially, to the pathological changes in a diseased heart[24]. The bidirectional communication mediated by inflammatory molecules, or cytokines, can be amplified by these glia cells in both organs. Therefore, the hypothesis arises that a common mechanism of BMS pathogenesis involves glial-mediated neuroinflammation in the brain contributing to central sensitization, while simultaneously, the dysfunctional autonomic outflow from the brain (the BHA) may trigger or be modulated by pathological changes in the heart's own glial cells, perpetuating the systemic dysregulation. The bridge between central microglial activation and peripheral cardiac glia is likely built by circulating pro-inflammatory cytokines (e.g., TNF-α, IL-6). In this "systemic neuro-inflammation" model, a "leaky" blood-brain barrier under stress may allow central signals to influence the heart's intrinsic nervous system, and vice versa.

## 10 INTRACRANIAL PRESSURE, PIEZOELECTRICITY, AND THE BHA IN BMS

A key emerging concept in the pathogenesis of BMS is the potential role of cerebrospinal fluid (CSF) dynamic disturbances and increased intracranial pressure (ICP). A recent case-control study reported a significantly higher prevalence of neuroimaging signs indicative of impaired intracranial pressure control, or idiopathic intracranial hypertension (IIH), in BMS patients compared to healthy controls, including a high prevalence of empty sella, enlargement of the optic nerve sheath, and dural sinus stenosis/hypoplasia[25]. This finding suggests that BMS may be, in part, a chronic neurological disorder linked to CSF dynamic disturbances. Elevated ICP can lead to a decrease

in the clearance of the glymphatic system and can compress cranial nerves, such as the trigeminal nerve, resulting in chronic cranial pain[26].

This mechanism links directly to the BHA via the mechanical pathway of communication. Recent work emphasizes the importance of mechanoreceptors in the BHA, particularly those expressing mechanosensitive Piezo protein channels, which relay crucial information about blood pressure and mechanical strain to the central nervous system[27]. The brain, including the central autonomic network, integrates signals from these mechanoreceptors, which detect pressure changes in the heart and blood vessels. In the context of elevated ICP, the mechanical forces transmitted through the meningeal and vascular structures of the brain and skull — potentially including the trigeminal nerve — could be sensed by these Piezo channels. Therefore, it is plausible that IIH-related increased pressure and mechanical strain could dysregulate the Piezo-mediated feedback loop of the BHA, intensifying the central sensitization and pain signals felt in the mouth. This perspective provides a novel, mechanical-physiological framework for understanding the neurological underpinnings of BMS that goes beyond simple network dysfunction. While pharmacological modulation of Piezo channels is in its infancy, compounds like GsMTx4 (a peptide blocker) are being studied in mechanotransduction research. If ICP-related mechanical strain is a driver of BMS, these channels represent a frontier for targeted "mechanomedicine."

## 11 LIMITATIONS

As a narrative review, this paper is subject to several limitations inherent to the methodology. Unlike a systematic review, which follows a rigid protocol to minimize bias, a narrative review relies on the author's expertise and judgment to select and synthesize the literature.

**Selection Bias:** Despite the structured search strategy, there is a risk of selection bias, where the author may have inadvertently prioritized studies that align with a particular viewpoint while overlooking or de-emphasizing contradictory evidence.

**Lack of Quantitative Synthesis:** The nature of a narrative review does not allow for a quantitative synthesis, such as a meta-analysis. Consequently, it cannot provide a statistical measure of the effect sizes or a definitive conclusion about the overall strength of the evidence.

**Heterogeneity of Studies:** The included studies vary widely in methodology, sample size, and patient populations. Integrating these diverse findings into a single coherent narrative is challenging and can obscure important nuances or inconsistencies in the data.

**Incomplete Scope:** Although a comprehensive search was performed, it is possible that some relevant literature, particularly studies published in non-English languages or in less-indexed databases, may have been missed.

These limitations highlight that the conclusions of this review should be viewed as a framework for understanding BMS, rather than as statistically proven definitive statements. The main value of this paper lies in its ability to integrate diverse research streams and build a coherent, hypothesis-generating model of the BHA for future research into the pathogenesis of BMS as nociplastic pain[28].

## 12 CONCLUSION

BMS is a systemic nociplastic disorder rooted in BHA dysregulation. This framework integrates autonomic instability, large-scale brain network imbalance, and dopaminergic/glial dysfunction. Recognizing BMS as more than a localized sensation is essential for developing multimodal strategies that address both the "brain" and the "heart" of the pain.

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Not applicable.

## Ethical Statement

Not applicable.

## Conflicts of Interest

The author declared no conflict of interest.

## Data Availability

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

## Author Contribution

Nagamine T solely carried out all aspects of this work.

## Abbreviation List

ACE-Is, Angiotensin-converting enzyme inhibitors
ANS, Autonomic nervous system
BHA, Brain-heart axis
BMS, Burning mouth syndrome
CNS, Central nervous system
CSF, Cerebrospinal fluid
DMN, Default mode network
ECG, Electrocardiogram
HRV, Heart rate variability
Ic, Insular cortex
ICP, Increased intracranial pressure
IIH, Idiopathic intracranial hypertension
QTc, Corrected QT interval
SNPR, Substantia nigra pars reticulata
SSRIs, Selective serotonin reuptake inhibitors
TTS, Takotsubo Syndrome

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