

When Pain Outruns Pathology: Toward a Bidirectional Model of Illness in Burning Mouth Syndrome

Diana Cassi ¹, Geraldo Aki Takanami de Oliveira ², Carlo Galli ²  and Marco Meleti ^{3,*}

¹ U.O di Odontostomatologia, Azienda Ospedaliero-Universitaria di Parma, 43126 Parma, Italy; dcassi@ao.pr.it

² Department of Medicine and Surgery, University of Parma, Via Volturmo 39, 43126 Parma, Italy; geraldoaki.takanamideooliveira@studenti.unipr.it (G.A.T.d.O.); carlo.galli@unipr.it (C.G.)

³ Department of Medicine and Surgery, Dental School, University of Parma, Via Gramsci 14, 43126 Parma, Italy

* Correspondence: marco.meleti@unipr.it

Abstract

Burning Mouth Syndrome (BMS) is a chronic orofacial pain condition characterized by persistent burning sensations in the absence of consistently identifiable pathology. This mismatch between severe symptoms and inconclusive findings challenges conventional diagnostic reasoning, which presumes that subjective complaints reflect underlying biological lesions—leading to repeated investigations, diagnostic delay, and uncertainty about symptom legitimacy. This paper uses BMS as a paradigmatic case to question a core assumption of clinical reasoning: that objective pathology constitutes primary evidence. In this Perspective, drawing on phenomenology and medical anthropology, we argue that when pathology is indeterminate, illness—the lived experience of suffering—must be treated as primary clinical evidence rather than as a secondary expression of disease. We propose a bidirectional conceptual framework in which physiological processes, psychological states, and lived experience interact dynamically, with symptoms actively participating in the maintenance of the condition. This framework explains persistent diagnostic uncertainty, heterogeneous treatment responses, and the clinical significance of validation and meaning-making. Reframing BMS in this way supports earlier recognition, reduces reliance on exclusion-based strategies, and justifies multimodal interventions addressing both biological and experiential dimensions of pain. More broadly, it suggests that clinical reasoning must expand to incorporate lived experience as indispensable evidence wherever pathology is indeterminate.

Keywords: burning mouth syndrome; phenomenology; illness and disease; lived body; medical anthropology; patient-centered care



Academic Editors: Hessam Nowzari and Omar Kujan

Received: 16 June 2026

Revised: 21 July 2026

Accepted: 28 July 2026

Published: 3 August 2026

Copyright: © 2026 by the authors.

Licensee MDPI, Basel, Switzerland.

This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC BY\)](https://creativecommons.org/licenses/by/4.0/) license.

1. Introduction—When Pain Outruns Pathology

Burning Mouth Syndrome (BMS) is a chronic orofacial pain condition characterized by persistent burning sensations, dysgeusia, and paresthesia in the absence of consistent or identifiable pathology [1,2]. Despite advances in neurobiological understanding, many patients present with normal clinical findings, unremarkable imaging, and inconclusive laboratory results. This mismatch between severe symptoms and limited objective evidence poses a recurrent challenge in clinical practice. Patients often undergo repeated investigations and diagnostic delay, and may encounter uncertainty regarding the legitimacy of their symptoms, while clinicians face difficulty integrating subjective reports with established diagnostic frameworks [3]. BMS therefore exemplifies a broader class of conditions in which suffering is evident but cannot be readily anchored to a single identifiable lesion.

This difficulty reflects a deeper tension within clinical reasoning. Although contemporary medicine increasingly incorporates biopsychosocial models, diagnostic validity continues to be closely associated with demonstrable pathology, while subjective experience is often treated as secondary or confirmatory [4]. The distinction between disease—objective dysfunction—and illness—the lived experience of disruption—therefore remains clinically consequential [5]. In conditions such as BMS, where pathology is indeterminate, this hierarchy becomes unstable: suffering persists even as its biological correlate remains uncertain. This raises a fundamental question for oral medicine: how should symptoms be interpreted when they cannot be securely linked to a discrete pathological process?

At the same time, contemporary research complicates any simple opposition between the presence and absence of disease. BMS is now understood to involve a heterogeneous mix of peripheral, central, and psychological contributors, none of which, individually or in combination, fully accounts for the severity and structure of the reported experience; even the terminology remains unsettled, with recent proposals to replace “syndrome” with “disorder” reflecting unresolved questions about whether BMS is a unified pathological entity or a heterogeneous experiential condition [6]. Section 2 reviews this evidence, and the diagnostic and therapeutic uncertainty it documents, in full. The point relevant here is narrower: these tensions reveal not only a diagnostic challenge but a deeper epistemic problem—how to interpret suffering when it cannot be securely anchored to a single identifiable lesion.

The present article builds on this evidence but pursues an aim that is theoretical and conceptual: it examines how existing evidence on BMS should be interpreted when severe and structured suffering persists despite indeterminate pathology. The clinical literature reviewed above thus provides the evidential background for the argument, while the proposed bidirectional model represents a hypothesis-generating interpretation of that evidence. Throughout, we distinguish evidence-based clinical premises—diagnostic criteria, exclusion of secondary causes, neuropathic and nociplastic mechanisms, psychological comorbidity, and heterogeneous treatment responses—from the conceptual claim that lived illness should acquire primary evidential status when pathology remains indeterminate.

This paper argues that BMS does more than exemplify a gap between disease and illness; it functions as a critical test case that exposes a limitation in the epistemic structure of clinical reasoning itself. Biomedical practice typically treats objective pathology as primary evidence and subjective experience as its derivative expression [7]. Yet where pathology remains indeterminate, suffering is not secondary or provisional but constitutes the primary and irreducible evidence that something is wrong [8]. This entails an inversion of the conventional evidential hierarchy: rather than deriving its legitimacy from pathology, lived experience precedes and guides the search for disease. The inversion does not deny the relevance of biological mechanisms, nor does it imply that patients’ causal interpretations of their symptoms are automatically correct or that differential diagnosis should be suspended. The claim is narrower: the reality of suffering should not depend on prior pathological confirmation, even though its causes and meanings remain open to clinical interpretation.

To articulate this shift, we draw on phenomenology, which approaches embodiment not as an object of measurement but as the lived medium through which the world is experienced [9]. From this perspective, BMS can be understood as a condition in which pain reorganizes perception, action, and social engagement, extending beyond local sensory disturbance. By tracing how this experience unfolds, we show that illness is an active dimension of the condition, potentially shaping biological, behavioral, and relational dynamics. This framework also supports a non-linear view of causality, in which experiential and physiological processes reciprocally influence one another.

The aim of the analysis is therefore both theoretical and practical. Conceptually, it reframes BMS as a condition that challenges lesion-based models of evidence and foregrounds the epistemic role of illness in diagnostic reasoning. Clinically, it shows that validation of patient experience performs a necessary epistemic role in diagnosis when illness functions as primary evidence. The proposal does not replace current diagnostic algorithms for BMS; it specifies how clinical reasoning should proceed once those algorithms have reasonably excluded common secondary causes, at the point where persistent burning, altered taste, xerostomia-like complaints, and disruption of daily life remain clinically evident despite the absence of mucosal lesions or explanatory laboratory findings. The approach does not propose new phenomenological or anthropological concepts but repositions established ones within clinical reasoning, to redefine what counts as evidence, how causation is understood, how diagnosis proceeds, and how management is initiated once primary BMS is recognized.

Methodological Approach

The aim of this Perspective is to examine a recurrent problem in clinical reasoning: how Burning Mouth Syndrome should be understood when severe and persistent symptoms coexist with limited or indeterminate objective findings. The approach was interpretive and integrative rather than empirical and developed the proposed framework in four steps.

We first identified the central problem: the mismatch between persistent oral burning, dysgeusia, xerostomia-like complaints, and functional disruption on the one hand, and the frequent absence of explanatory mucosal, laboratory, or imaging findings on the other. We then grounded this problem in a selective, narrative appraisal of the current BMS literature, prioritizing recent systematic reviews, meta-analyses, and large cohort studies on diagnosis, mechanisms, diagnostic delay, and treatment, supplemented by individual studies addressing specific gaps. This is not a systematic review: no formal search protocol, risk-of-bias assessment, or quantitative synthesis was undertaken, and the literature was used to define the problem and keep the proposed model compatible with contemporary evidence rather than to estimate pooled effects.

We then interpreted this problem through established concepts from phenomenology and medical anthropology—the distinction between disease and illness, between the objective and the lived body, and the role of illness narratives in clinical encounters—used not as alternative empirical mechanisms but as tools for clarifying what counts as evidence when pathology is indeterminate. Finally, we synthesized these clinical and conceptual materials into a bidirectional model in which physiological processes, psychological states, and lived experience are treated as reciprocally interacting dimensions of BMS, and derived its practical implications by asking how the model changes diagnostic reasoning, validation, communication, treatment planning, and follow-up. Because the approach is conceptual, its claims are explanatory and hypothesis-generating rather than estimates of efficacy or diagnostic accuracy; the framework's limitations are set out in Section 6.4.

2. The Current Evidence Base: What Clinical Research Establishes and Leaves Unresolved in BMS

2.1. Definition, Classification, and Epidemiology

Burning mouth syndrome is consistently defined across current classification systems as chronic intraoral burning or dysaesthesia in the absence of an identifiable local or systemic cause, but the operational criteria used to reach that diagnosis differ across schemes. The International Classification of Headache Disorders, 3rd edition (ICHD-3) requires daily burning pain for more than two hours per day, persisting for more than three months, together with a normal-appearing mucosa and unremarkable clinical examination [10]. The

International Classification of Orofacial Pain (ICOP) adopts a comparable exclusion-based definition and further subclassifies BMS according to the presence or absence of somatosensory changes [11]. ICD-11, by contrast, situates BMS within the broader category of chronic primary pain, framing it explicitly as multifactorial and biopsychosocial rather than as a diagnosis reached by exclusion alone [12].

This classificatory divergence is not merely terminological. A systematic review of disease definitions used in BMS randomized controlled trials found substantial heterogeneity in inclusion and exclusion criteria across studies, raising the possibility that some trial populations captured overlapping or misclassified conditions [13]. Criteria are, in other words, still actively contested rather than settled—a fact with direct consequences for how prevalence and treatment-response findings should be read.

Epidemiologically, BMS is uncommon but clinically significant: a systematic review and meta-analysis estimated a pooled prevalence of approximately 1.73% in the general population, rising to 7.72% in clinical settings [1]. A subsequent systematic review and meta-analysis applying International Headache Society classification criteria characterized the typical clinical profile as predominantly affecting women in their sixth and seventh decades, most frequently involving burning of the tongue, and commonly co-occurring with anxiety and depression [14].

Altogether, these findings support three points that bear directly on the argument developed below. First, BMS continues, in practice, to be handled largely as a diagnosis of exclusion [6,15]. Second, diagnostic criteria are actively contested and revised, but no single criterion set has achieved both broad validation and clinical consensus. Third, and most consequential for what follows, the literature offers epidemiological description and criteria revision, but no validated account of how much exclusionary workup is sufficient before a positive diagnosis is warranted. This is not a gap in how the literature has been read here; it is a gap in the literature itself, and it is a gap the argument in Section 6 takes up directly.

2.2. Proposed Mechanisms

Peripheral, central, and psychological mechanisms have each accumulated supporting evidence, though each explains only part of the clinical picture. At the peripheral level, a systematic review of eight studies found tongue-biopsy evidence of reduced epithelial nerve fiber density, ranging from 30% to 60% below control values, alongside altered expression of TRPV1, NGF, and P2X3 nociceptive markers, consistent with small-fiber neuropathy in a substantial subset of patients [16]. A subsequent systematic review of fourteen studies (214 BMS patients, 130 controls) using GRADE-based quality assessment corroborated reduced nerve fiber density and nociceptive-marker overexpression, while noting considerable variability across studies and that no single histological finding is yet established as diagnostic [17].

At the central level, altered trigeminal modulation and subtle structural and functional brain changes have been reported, indicating that the absence of overt peripheral lesions does not exclude biological involvement [15]. A 2025 systematic review comparing neuroimaging findings across chronic primary orofacial pain subtypes under ICD-11 classification found that BMS shares central neuroplastic features, including altered functional connectivity and grey matter change, with temporomandibular disorder—suggesting a mechanism common to conditions grouped as chronic primary pain, rather than one specific to BMS [18].

Psychological and stress-related factors show a comparably consistent but non-specific association. A systematic review and meta-analysis identified anxiety and depression as the most frequent psychological comorbidities in BMS [19], and a subsequent meta-

analysis found that BMS patients score higher on stress measures and show some biomarker differences relative to controls, while cautioning that these biomarkers are not specific to BMS [20]. The direction of this association is genuinely unresolved rather than merely under-studied: a nationwide cohort study found that prior depression, anxiety, and bipolar disorder were associated with increased subsequent risk of developing BMS [21], while a separate cohort found that a BMS diagnosis was itself associated with increased subsequent risk of depression, anxiety, dementia, and Parkinson disease [22]. Read together, these two cohorts provide direct longitudinal evidence for reciprocal influence between psychological state and BMS rather than support for causation running in a single direction.

Thus, the peripheral, central, and psychological literatures each identify real, replicated findings, but none—individually or combined—accounts for why symptom severity and structure so often exceed what the corresponding biological or psychological measures alone predict. This explanatory residue is what the framework proposed in Section 6 is built to address: not by supplying a further causal mechanism, but by treating the residue itself as clinically significant.

2.3. *Diagnosis and Diagnostic Delay*

Current diagnostic algorithms for BMS proceed by staged exclusion: ruling out oral infections, nutritional deficiencies, hormonal and systemic disease, and iatrogenic causes before a diagnosis of primary BMS is reached [15]. This logic is reflected in the classification systems reviewed above and reinforced by an international Delphi consensus recommending which secondary causes should be systematically evaluated before a positive diagnosis is considered [6].

The clinical cost of this exclusion-based pathway is well documented. In a cohort of 500 Italian patients, the mean diagnostic delay was 29.71 months, patients consulted an average of 2.61 practitioners before diagnosis, and the mean number of misdiagnoses was 3.54, most commonly nonspecific stomatitis, candidiasis, and gastroesophageal reflux; longer delay was independently predicted by higher age, lower education, the presence of dysgeusia, and a prior history of psychiatric illness [23]. These figures indicate not an occasional diagnostic difficulty but a structural feature of how BMS is currently managed.

The field has recently begun to address part of this problem directly. A 2026 narrative review proposes a “minimum sufficient” diagnostic workup and a stepwise, phenotype-guided algorithm intended to reduce diagnostic delay and low-value testing [24]. This is a genuine advance on the efficiency of the exclusionary pathway itself: it specifies which tests to order, in what sequence, and when further testing ceases to add diagnostic value. It does not, however, address a distinct and prior question: once exclusion is complete, what evidential status should the patient’s reported experience itself carry in reaching a positive diagnosis? A workup can be minimized in sequence and content without settling whether, or on what basis, sustained and structured suffering should count as sufficient grounds for diagnostic closure in the absence of a positive biological finding. This is the specific gap the framework proposed in Section 6 addresses: not how to exclude more efficiently, but what should count as evidence once exclusion has been reasonably completed.

2.4. *Treatment Evidence*

Treatment evidence for BMS remains heterogeneous across modalities and outcome measures. Two systematic reviews conducted under the World Workshop on Oral Medicine VII initiative assessed, respectively, topical and systemic pharmacological interventions: the topical-interventions review found short-term benefit for selected agents based on pain-scale change, but noted few trials per agent, high heterogeneity, and low overall certainty of evidence [25]; the systemic-pharmacotherapy review, covering interventions including

clonazepam, alpha-lipoic acid, and antidepressants, found no single intervention with consistent superiority across trials [26]. A subsequent network meta-analysis attempting to rank treatments directly encountered a sparse evidence network with limited comparative power, constraining confidence in any resulting hierarchy [27]. A broader systematic review of BMS treatment trials identified favorable signals for cognitive behavioral therapy, clonazepam, capsaicin, and photobiomodulation, but again noted substantial heterogeneity in interventions and limited long-term follow-up [28].

Table 1 summarizes these and the other systematic reviews and meta-analyses discussed across this section, together with the certainty or principal limitation each reports.

The treatment literature thus supports two conclusions relevant to the argument developed below. First, no intervention has demonstrated consistent superiority, and current guidance defaults, by necessity rather than by positive evidence, toward individualized multimodal care. Second, the comparability of this evidence is itself limited: a companion WWOM VII review of outcome reporting found that standardized pain-outcome domains recommended by the IMMPACT consensus were inconsistently reported across BMS randomized trials, with meaningful gaps even after the consensus was published [29]. This is not a call for more trials of the same kind; it is a specific limitation bearing on how confidently any treatment recommendation—including the multimodal, communication-inclusive approach argued for in Section 6—can currently be grounded in trial evidence alone.

Table 1. Recent systematic reviews and meta-analyses on Burning Mouth Syndrome, by evidence domain.

Source	Year	Design	Focus	Principal Finding	Certainty/Main Limitation
Headache Classification Committee [10]	2018	Classification	Diagnostic criteria (ICHD-3)	Defines the exclusion-based diagnostic standard	Classification criteria rather than empirical validation
ICOP, 1st edition [11]	2020	Classification	Diagnostic criteria and subtyping	Adds somatosensory-based subtyping	Classification criteria rather than empirical validation
Ariyawardana et al. [13]	2019	SR	Disease definitions used in BMS RCTs	Identified substantial heterogeneity in inclusion and exclusion criteria	Restricted to RCTs published through 2017
Wu et al. [1]	2022	SR + MA	Prevalence	Pooled prevalence was approximately 1.73% in the general population and 7.72% in clinical populations	Marked epidemiological heterogeneity
de Lima-Souza et al. [14]	2024	SR + MA	Clinical and epidemiological profile	Typical profile comprised women in the sixth to seventh decades of life, most commonly reporting tongue burning	Evidence was largely descriptive and based on cross-sectional studies
Kouri et al. [16]	2024	SR	Small-fibre neuropathy	Reduced nerve-fibre density was reported in approximately 30–60% of patients across eight studies	Variable methodology and small samples
Porporatti et al. [17]	2025	SR	Oral-tissue histology	Reported reduced nerve-fibre density and overexpression of nociceptive markers	Fourteen studies with considerable methodological variability
Fischer et al. [18]	2025	SR	Neuroimaging in BMS and TMD	Identified central neuroplastic changes shared by BMS and TMD	Findings may reflect mechanisms common to chronic orofacial pain rather than BMS-specific changes
Galli et al. [19]	2017	SR + MA	Psychological factors	Anxiety and depression were the most frequently reported psychological comorbidities	Direction of association was not established
Porporatti et al. [20]	2023	MA	Stress measures and biomarkers	Found higher perceived-stress scores and some differences in stress-related biomarkers	Biomarkers were non-specific
Adamo et al. [23]	2024	Cohort (<i>n</i> = 500)	Diagnostic delay	Mean diagnostic delay was 29.71 months, with 3.54 misdiagnoses per patient	Single-centre study involving a referral population
Tan et al. [28]	2022	SR	Treatment efficacy	Reported favourable signals for CBT, clonazepam, capsaicin, and photobiomodulation	Heterogeneous interventions and limited follow-up
Farag et al. [26]	2023	SR + MA	Systemic pharmacotherapy	No intervention was consistently superior across comparisons	Overall certainty was low
Goncalves et al. [25]	2023	SR	Topical interventions	Selected topical agents showed short-term benefit	Few trials per intervention and short follow-up
Alvarenga-Brant et al. [27]	2023	SR + NMA	Comparative treatment ranking	No robust comparative treatment hierarchy could be established	Sparse evidence network and constraints on transitivity
Farag et al. [29]	2019	SR	Outcome-domain reporting	IMMPACT outcome domains were inconsistently reported	Underreporting persisted even after publication of consensus recommendations

3. When Pathology Falls Silent: The Limits of Lesion-Based Reasoning

While often used interchangeably in everyday language, the terms disease and illness refer to distinct dimensions of human suffering [30]. Disease denotes dysfunction at the level of biological processes, whereas illness designates the lived, experiential disruption of being unwell. Although contemporary medicine increasingly acknowledges their interplay, clinical reasoning continues to operate within an epistemic hierarchy that privileges the former as primary evidence, with the latter often treated as its expression or consequence [4].

3.1. Disease as Object: The Biomedical Approach to the Body

Biomedicine approaches the patient by seeking disease—an objective, reproducible alteration in bodily structure or function. Kleinman defines disease as “*the malfunctioning or maladaptation of biologic and psychophysiologic processes*” [31], a disruption that can be detected, measured, and classified. In this framework, the body is rendered as an analyzable system whose deviations can be localized, whether in the form of infection, metabolic imbalance, genetic mutation, or structural lesion [32]. Canguilhem’s account of the pathological as a break in the organism’s normative continuity further grounds this orientation [33].

The strength of this model lies in its capacity to isolate discrete, actionable problems from the complexity of lived experience [34]. Clinical encounters are therefore structured around the transformation of patient narratives into diagnostically relevant data: physical findings, imaging results, and laboratory values that can anchor a disease label [35]. Through this process, biomedicine achieves remarkable explanatory and therapeutic power. Yet this transformation also entails a selective reduction, in which elements of experience that do not readily map onto measurable variables may recede from clinical attention [36].

3.2. Illness as Experience: The Lived Body in Clinical Context

From the patient’s perspective, however, what is at stake is not disease as such but illness—the embodied, meaning-laden experience of disruption [37]. Kleinman describes illness as “*the human experience of sickness*”, shaped not only by sensory symptoms but also by emotional, relational, and cultural contexts [38]. Pain, fatigue, fear, and loss of capacity are not isolated data points but elements of a transformed world. While disease may be localized within an organ system, illness unfolds across the entirety of a life [39]. Cassell’s formulation captures this asymmetry succinctly: illness is what the patient feels when they seek care, whereas disease is what is identified through clinical reasoning, what the patient takes home [40].

Disease and illness seldom map neatly onto each other (Figure 1) [41]. A patient may harbor disease without illness, as in silent hypertension or an occult carcinoma; the pathological abnormality exists, yet lived health remains undisturbed [42]. Conversely—and central to the argument here—a person may suffer profound illness despite the absence of an identifiable disease [43]. Chronic pain syndromes, chronic fatigue, and Burning Mouth Syndrome exemplify this latter condition, in which suffering is unmistakable while diagnostic markers remain inconclusive. In such cases, the absence of demonstrable disease does not diminish the reality of illness, yet it often destabilizes its epistemic status within clinical practice [44].

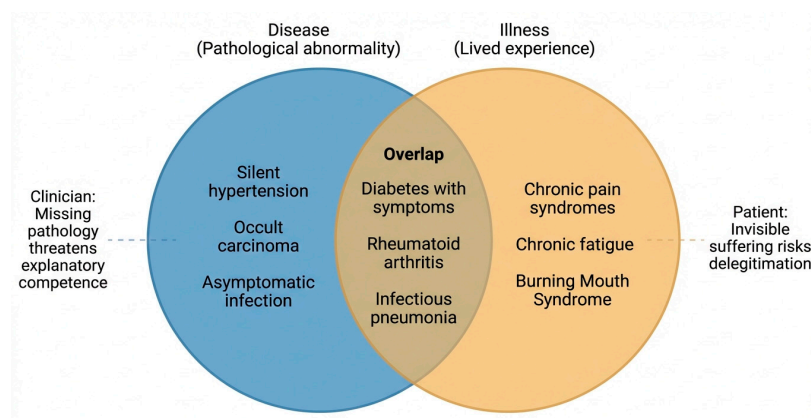


Figure 1. Disease (objectively identifiable pathology) and illness (lived experience of suffering) do not map onto one another, as Burning Mouth Syndrome illustrates.

3.3. From Narrative to Data: What the Clinic Loses in Translation

Patients do not present symptoms as isolated variables. They present them as narratives that connect bodily sensations with personal meaning, social consequences, and expectations about the future [45]. Kleinman's notion of the illness narrative highlights this interpretive work: patients try to make suffering intelligible within the context of their lives [38]. These narratives connect physical discomfort to broader concerns—work, relationships, identity, and the possibility of a livable future [46]. Cultural scripts supply further threads—collective ideas about blame, stigma, and what suffering signifies [47]. Thus, a patient may liken her torment to a pebble dropped in a pool, its ripples widening [48], a powerful image that captures how a single sensory locus can unsettle every concentric ring of daily existence.

Yet the ordinary clinical encounter often requires a rapid act of translation, and translation inevitably entails loss [49]. Charged with differential diagnosis, the clinician parses the story for data points that might anchor a disease label [50]. What is lived as an all-encompassing disruption—such as the dread of eating due to oral burning—may be reformulated as, e.g., “*pain exacerbated by acidic intake*”, while existential uncertainty may be reduced to a numerical score on a Visual Analog Scale [51].

Such reduction is not malpractice; it is built into the epistemic grammar of biomedicine, and it is most often a necessity [52]. Yet each abstraction widens the experiential gap between patient and practitioner by stripping the narrative of its existential stakes [53]. As Toombs observed, this divergence can lead clinicians and patients to talk at cross purposes, each operating within a different horizon of meaning [54]. It is precisely within this gap that conditions such as BMS become difficult to interpret, as the absence of stable pathological markers leaves illness without its usual biomedical anchor.

In this context, the distinction between disease and illness has direct epistemic consequences. When illness cannot be securely tied to demonstrable pathology, its status within clinical reasoning becomes unstable. The following section addresses this instability by examining how lived experience, rather than functioning only as a secondary indicator of disease, may in certain cases constitute the primary evidence that calls for explanation.

4. A Different Kind of Evidence: The Clinical Relevance of Lived Experience

Understanding illness that resists pathological verification requires a shift from the third-person perspective of biomedical observation to the first-person horizon of lived experience. Phenomenology provides both the method and conceptual framework for this shift [55]. Rather than explaining symptoms through pre-existing theoretical models, it begins from how experience is given, describing illness as it appears to the person who

lives it [56]. Contemporary scholars such as Havi Carel and S. Kay Toombs have shown how this approach clarifies the predicament of illness, which is irreducibly first-personal, saturating perception, action, and meaning [54,57].

4.1. *The Lived and Objective Body in Clinical Practice*

A phenomenological approach to illness pivots on the distinction between *Körper* and *Leib* in the German tradition [58,59]. The *Körper* is the anatomical body—the layered aggregate of cells, tissues, and organs that lends itself to weighing, imaging, and dissecting. It is the body to which physiology, biochemistry, and surgical technique address their questions; its logic is quantitative and exterior [60]. The *Leib*, by contrast, is the body lived from within: the center of perception, agency, and affect, the tacit medium through which the world is disclosed. Merleau-Ponty notes that, in health, the *Leib* remains largely transparent—one does not calculate finger trajectories when lifting a cup; one simply reaches, propelled by an unspoken “I can” [61]. In this primordial intimacy, the body is not an object we happen to own; it is the very ground of selfhood [62].

This distinction is not merely descriptive; it is epistemically consequential. Clinical medicine primarily engages with the *Körper*, whereas illness unfolds at the level of the *Leib*. When these two dimensions align, this dual perspective is unproblematic. However, when no clear pathological correlate can be identified—as in BMS—the clinical focus on the objective body encounters its limits, while the lived body remains saturated with disruption.

While the accuracy of any particular perception may be questioned, the fact that experience is occurring cannot be doubted [61]. Husserl calls this reflective suspension of naïve assumptions the *epoché*—a bracketing of the world’s factual status that nevertheless leaves the indubitable fact of appearance untouched [63,64].

Transposed into clinical terms, this yields an asymmetry: disease, as an inferred or measured entity, may be uncertain or absent; illness, as lived experience, is immediately given. This does not make the report incorrigible. What reaches the clinic is not the experience itself but an account of it, shaped by language, memory, and circumstance. Such accounts can be imprecise, revised, or mistaken in their attributions. The phenomenological point is narrower and more secure: that something is being undergone is given to the one who undergoes it, even when its cause is unknown and its description imperfect. Questioning the accuracy and interpretation of reports is legitimate. The problem arises when suffering itself is treated as provisional until confirmed by a lesion. This reverses the proper order. Illness here counts as evidence in its own right that something has gone awry, evidence whose reality stands even when its organic correlate, and the patient’s own account of it, remain open to revision.

This point is especially important in conditions such as psychosis or functional neurological disorder. In these presentations, symptom meaning, agency, or attribution may require particularly careful assessment [65]. In such cases, validation does not mean confirming the patient’s explanatory model [31], nor does diagnostic caution mean denying the reality of suffering. Rather, the experience is taken seriously as evidence that something requires clinical understanding, while causal interpretation remains open to assessment, revision, and differential diagnosis [66,67].

From a phenomenological perspective, illness is not a collection of symptoms but a transformation of one’s entire mode of *being-in-the-world* [68,69]. Bodily functions that normally operate in the background become obtrusive [70]. The lived body—the *Leib*—moves from invisible ground to obstinate foreground, fracturing the tacit harmony that ordinarily unites organism and environment [69].

This shift is not merely local but global. Pain folds attention back onto the self, compressing temporal horizons and distorting spatial relations; mundane tasks swell with effort, futures contract, social worlds recede. By articulating these transformations, phenomenology reveals that seemingly disparate complaints form a coherent experiential structure.

4.2. *Disruption of Embodiment and Everyday Life*

Chronic illness can intensify this transformation into a form of bodily alienation. The body, once experienced as part of oneself, becomes an object that resists control—a source of unpredictability and constraint [71]. This shift from lived body to object body is accompanied by a broader disruption of the *Lebenswelt*, the background of taken-for-granted meanings that organize daily life [69,72]. For patients with BMS, this disruption is tangible, reorganizing daily routines around vigilance and discomfort rather than remaining confined to the oral cavity [57,73].

4.3. *Patients as Interpreters of Their Own Symptoms*

A further consequence of this transformation is that patients become involuntary analysts of their own experience [74]. Classical phenomenology involves a deliberate suspension of habitual assumptions in order to examine how experience is structured [64]. Chronic illness imposes a comparable process involuntarily. As Carel observes, illness generates a “*radical, violent philosophical motivation*”, forcing sustained attention to the body and its alterations [57].

The healthy individual lives through the body with minimal reflection. By contrast, the person with chronic illness is compelled to monitor, interpret, and anticipate bodily signals. Kleinman’s description of the patient as “*their own interpreter of bodily signs*” captures this shift [38]. Patients with BMS may meticulously track pain levels throughout the day, experiment to identify foods or situations that trigger flare-ups, and struggle to find words for a sensation that is simultaneously burning, tingling, and metallic. In doing so, they engage in a practical and urgent form of phenomenological investigation. They are forced to attend to, describe, and attempt to make sense of their own *Erlebnis*, their own lived experience, in Husserl’s terminology [64], with an intensity that the healthy person can scarcely imagine. Patients are not a passive repository of symptoms; they are active interpreters and the foremost experts on the phenomenology of their own suffering.

5. BMS as Lived Illness: What Patient Experience Reveals

A phenomenological lens does more than enrich the description of Burning Mouth Syndrome; it clarifies its epistemic significance. Rather than being confined to the biomedical impasse of an undetectable lesion, BMS can be understood as a condition in which illness is fully manifest despite indeterminate pathology [75]. Patient accounts, though diverse in detail, reveal a consistent experiential structure. First, they describe a radical estrangement from a bodily region once taken for granted: the mouth becomes an alien terrain, at once intimate and hostile [76]. Second, this disturbance extends beyond the local sensation, reshaping the broader lifeworld, as detailed in Section 5.2 [77]. Third, sufferers embark on an exhausting quest for legitimacy, compelled to defend the reality of their torment in clinical settings where validation is often tied to visible pathology [23]. These features indicate that BMS is not a vague complaint awaiting biological confirmation but a coherent form of illness whose structure is immediately accessible in lived experience. It is precisely this accessibility—rather than the presence of a lesion—that establishes its clinical reality.

5.1. *The Mouth Becomes Other*

The mouth occupies a distinctive position in embodied experience, serving as a site of nourishment, communication, and intimacy [61]. In health, it functions transparently within the lived body; attention flows through it rather than toward it [78]. BMS disrupts this transparency. Patients describe sensations of burning, scalding, or chemical alteration—“*a tongue on fire*”, “*a mouth scalded with boiling water*” [79–81]. These descriptions do not merely indicate increased sensory intensity but signal a transformation in the mode of embodiment. The mouth shifts from lived subject to intrusive object, from *Leib* to *Körper* [82]. What was once a tacit medium of engagement becomes an explicit focus of attention, demanding constant monitoring and constraining spontaneous action. As one patient reports, the pain “*takes on a life of its own*” [79], capturing the sense in which a part of the body becomes experientially autonomous. This shift has epistemic consequences. The absence of visible pathology does not diminish the reality of this transformation; on the contrary, it highlights that the condition is given most directly at the level of lived experience. BMS thus provides a concrete instance in which the condition is most directly accessible through lived experience, even when pathological correlates remain indeterminate [2].

5.2. *Impact on Time, Social Life, and Identity*

The disruption initiated at the level of the body extends to the entire structure of experience. Time, in particular, is reorganized around pain. Many patients report a daily rhythm in which symptoms intensify over the course of the day, shifting attention away from chosen activities toward the anticipation and management of discomfort [83]. The future, once open and project-oriented, becomes constrained by uncertainty and fear, and daily life may come to feel like endurance [84]. Social and relational dimensions are similarly affected. Food, a central medium of pleasure and social bonding, becomes a potential trigger; meals are approached with caution rather than enjoyment [85]. Conversation itself may become effortful or painful, leading to withdrawal from social interaction and increasing isolation [86]. Over time, these changes can reshape the sense of self. Patients describe diminished quality of life, emotional distress, and a progressive identification with the condition itself, as if the illness colonizes identity [87,88]. These transformations are not secondary psychological reactions but integral components of the illness. They demonstrate that BMS is not confined to a local sensory disturbance but reorganizes temporality, sociality, and selfhood. In this way, it confirms the phenomenological claim that illness is a global modification of being-in-the-world.

5.3. *Proving Pain: The Quest for Legitimacy*

The epistemic tension at the core of BMS becomes most visible in the clinical encounter. Patients whose lives have been profoundly altered by persistent pain often confront a series of normal findings: intact mucosa, unremarkable imaging, and laboratory values within range [89]. What clinicians interpret as the successful exclusion of pathology may be experienced by patients as a form of negation, in which the absence of measurable abnormalities casts doubt on the legitimacy of their suffering.

Yet the sensations themselves—burning, dysgeusia, paresthesia—are not hypothetical constructs awaiting validation. For the person who experiences them, they are directly given realities. Sharpe and Greco describe such phenomena as *selective realities*, experiences that demand attention because they disrupt the organism’s coherence [90]. Their evidential force lies not in their correlation with a lesion but in their capacity to reorganize lived experience.

In this context, the assumption that symptoms derive their legitimacy from an underlying anatomical substrate becomes untenable. Pain is encountered first as experience, not

as a laboratory finding. BMS therefore exposes a structural limitation in clinical reasoning: when pathology is indeterminate, illness does not lose epistemic status but becomes the primary evidence that calls for explanation.

The structural tensions described above are not merely theoretical abstractions; they are recurrent features of clinical practice. The following vignette is a composite constructed for illustration from features reported in the published clinical literature [3,15]; it does not describe an identifiable individual. It shows how BMS typically unfolds within contemporary care pathways.

A 56-year-old woman presents with an eighteen-month history of daily oral burning described as “like chili,” affecting the anterior tongue and hard palate. The discomfort intensifies over the day and is partially relieved by chewing. She reports dry mouth despite normal salivary flow, a persistent metallic taste, intermittent tingling, and associated headaches. Clinical examination reveals healthy mucosa; imaging and laboratory investigations are unremarkable.

Over the following months, she consults multiple specialists. Each encounter generates further investigations and empirical treatments—antifungals, proton-pump inhibitors, occlusal adjustments—without sustained benefit. The absence of identifiable pathology leads to repeated reassurance, yet the symptoms persist. At times, she disengages from care, uncertain whether the problem lies in her body or in its interpretation.

After exclusion of secondary causes, a diagnosis of primary BMS is established. Management proceeds through pharmacological and behavioral strategies, yielding partial but meaningful improvement. At follow-up, the pain remains but is experienced as more intelligible and less overwhelming.

This trajectory reveals the coexistence of two distinct orientations. Within a biomedical framework, the absence of pathology prolongs investigation and limits explanatory clarity. Within a phenomenological framework, the central transformation lies in how the patient’s experience is interpreted, validated, and integrated into a coherent account. The shift does not eliminate symptoms but alters their meaning and their place within the patient’s life.

The divergence between these orientations is not merely rhetorical but structural. They reflect different assumptions about what counts as evidence, how symptoms are interpreted, and what constitutes a therapeutic goal. BMS thus serves as a paradigmatic case in which illness, rather than functioning as a derivative sign of disease, becomes the primary phenomenon through which the condition is both experienced and known.

To clarify how phenomenological analysis complements rather than replaces conventional clinical reasoning, Table 2 compares the dimensions foregrounded by the clinical–biomedical and lived-experience perspectives on BMS.

Table 2. Burning Mouth Syndrome from biomedical and phenomenological perspectives. The table contrasts the objective-body perspective of conventional clinical reasoning with the lived-body perspective foregrounded by phenomenological analysis.

Dimension	Clinical–Biomedical Perspective (<i>Körper</i>)	Phenomenological Perspective (<i>Leib</i>)
Primary object of attention	The clinical pattern of oral burning or dysaesthesia, associated symptoms, possible biological mechanisms, and the exclusion of relevant local or systemic causes.	The way oral burning is experienced from within and how it alters perception, agency, attention, and engagement with everyday life.
Characterization of symptoms	Burning pain, dysgeusia, xerostomia-like complaints, and paraesthesia characterized according to location, duration, intensity, temporal pattern, examination findings, and relevant investigations.	Experiences such as “a mouth on fire”, estrangement from the mouth, constant bodily vigilance, unpredictability, and the sense that pain has acquired a life of its own.

Table 2. *Cont.*

Dimension	Clinical–Biomedical Perspective (<i>Körper</i>)	Phenomenological Perspective (<i>Leib</i>)
Diagnostic contribution	Recognition of the established clinical pattern in clinically normal mucosa after focused and proportionate exclusion of relevant secondary causes.	The patient’s structured account provides evidence of the reality, severity, and functional consequences of the illness, supporting clinical understanding and diagnostic communication.
Understanding of the body	The mouth is examined as a system of mucosa, nerves, salivary function, sensory pathways, and other measurable structures or processes.	The mouth is ordinarily a tacit medium of eating, speaking, intimacy, and social participation but becomes obtrusive, estranged, and difficult to inhabit.
Consequences for everyday life	Functional impairment, psychological distress, quality of life, sleep, diet, and treatment response can be assessed through clinical history and standardized measures.	Pain reorganizes temporality, eating, communication, social relationships, self-understanding, and expectations about the future.

6. Rethinking Causality in BMS: From Linear Models to Bidirectional Interaction

The argument that follows rests on a set of evidence-based clinical premises: BMS is a chronic oral pain condition characterized by persistent burning or dysesthetic symptoms in clinically normal mucosa; diagnosis requires exclusion of secondary local, systemic, medication-related, and mucosal causes; current evidence supports multifactorial neuropathic and nociplastic mechanisms; psychological distress and comorbidity are frequent but do not by themselves establish psychogenic causation; and available treatments show variable, often partial benefit [15]. The conceptual claim developed here is distinct from these premises: once they are accepted, BMS exposes a limitation in clinical reasoning, because when pathology remains indeterminate the structured illness experience must itself function as primary clinical evidence. This is a philosophical and clinical interpretation of the existing evidence, not an empirical finding produced by the present article.

6.1. Situating the Framework Relative to Existing Models

The relevant comparison for assessing novelty is not the classical, lesion-focused biomedical model [91,92], which contemporary pain medicine has already moved well beyond, but the family of frameworks now used to handle pain that resists a demonstrable lesion. Chief among these is the ICD-11 category of chronic primary pain, which is diagnosed when pain has persisted for more than three months and is associated with significant distress or functional disability, without requiring that the pain be reducible to, or fully explained by, an identified biological cause [93]. On this point, the framework proposed here is not staking new ground: the claim that a condition can be diagnostically legitimate without a demonstrated lesion is already established in mainstream classification, and the account developed in this article should not be read as the first to escape lesion-centered reasoning.

Three further accounts sit closer to the present argument and require more careful differentiation. Classical biopsychosocial models, as discussed below, treat biological, psychological, and social factors as separate contributions whose effects are summed, with biological dysfunction retained as the entity to be explained [92]; the present account differs by treating experience as constitutive of the condition rather than as one input among others (Section 6.2). Nociplastic and central-sensitization accounts, increasingly used to explain chronic primary pain conditions including BMS, locate the relevant change in altered central processing and treat experience as the resulting output of that change—a readout of a process that remains, in explanatory terms, confined to the nervous system [94]. Predictive-processing accounts of pain go further in giving experience an active computational role, modelling pain as the product of Bayesian inference over noisy bodily and contextual

cues rather than as a passive readout [95]; even so, the inference in question is standardly modelled as occurring within the organism's own predictive architecture, and the account does not extend the relevant explanatory system beyond the individual nervous system.

Closest to the present account are enactive treatments of illness, which hold that health and illness are brought forth through an organism's ongoing engagement with its environment rather than residing in either pole alone [96]. The framework proposed here shares this general orientation but makes a narrower and more specific claim than an enactive account of illness in general: not simply that illness is relational rather than located in the organism alone, but that structured, sustained lived experience should carry positive evidential weight in clinical diagnosis, and that validation and narrative reframing should be understood as interventions on the condition itself rather than as adjuncts to biological treatment. This is a clinical-epistemic claim about diagnostic and therapeutic reasoning, not only an ontological claim about what illness is.

Read against this literature, the contribution of this article is best stated narrowly. The current evidence base, reviewed in Section 2, establishes that BMS is frequently diagnosed by exclusion and that diagnostic delay is substantial. Existing BMS frameworks do not generally make explicit what positive evidential weight should be assigned to structured lived experience once a proportionate exclusion of secondary causes has been completed (Section 2.3). It is this specific, unresolved question, rather than the general claim that pain can exist without a lesion or that illness is relational, that the bidirectional account developed below is intended to address. Longitudinal evidence for reciprocal influence between psychological state and BMS onset (Section 2.2) offers empirical, rather than only phenomenological, support for treating this relation as genuinely bidirectional rather than reducible to a single direction of causation.

6.2. From Linear Models to Bidirectional Interaction

BMS does more than expose a diagnostic blind spot; it reveals a limitation in the causal grammar that continues to shape much clinical reasoning. Classical models of disease are typically organized around a linear sequence: an identifiable lesion or dysfunction initiates a cascade of physiological changes, which then generate symptoms. Within this framework, disease holds ontological and explanatory primacy, while illness appears as its downstream manifestation. BMS disrupts this sequence. Here, suffering is fully present and clinically consequential, while its putative biological origin remains indeterminate. This asymmetry does not simply signal missing knowledge; it calls into question whether a strictly linear model of causality is sufficient to account for such conditions.

Models that treat subjective experience as secondary or derivative therefore become inadequate. When pathology cannot be identified, symptoms risk being reinterpreted as provisional, psychogenic, or of lesser ontological status; however, in BMS, the persistence, structure, and impact of the experience resist such demotion. Illness does not await biological confirmation but constitutes the phenomenon any adequate causal model must explain. Empirical findings already point toward a more complex landscape: BMS is frequently associated with anxiety, depression, and significant life stressors [97], and patient narratives often situate onset within contexts of bereavement, caregiving strain, or social disruption. Attempts to impose a unidirectional interpretation prove unstable—if distress precedes pain, the condition is read as psychological, while if pain precedes distress, the emotional sequelae are treated as secondary [98]—because chronic pain can sensitize neural pathways and heighten vigilance while sustained distress modulates immune function and nociceptive thresholds, amplifying the experience of pain [99]. As discussed in Section 6.1, this additive structure works where a lesion or measurable dysfunction anchors the disorder. It loses traction precisely where BMS sits: with no biological term to anchor,

the biopsychosocial reading defaults to an idiopathic core surrounded by psychological comorbidity, reinstating the hierarchy it set out to overcome.

The account proposed here differs in three respects that bear directly on practice. First, it treats lived experience not as one variable acting on the disorder but as the level at which the disorder is individuated at all. In the absence of a lesion, the structured experience of burning, estrangement, and disrupted everyday life is what makes the condition a recognizable entity rather than a residue left after exclusion. This licenses positive diagnosis on experiential grounds once major secondary causes are reasonably excluded, and it predicts that diagnostic confidence should not increase with the accumulation of negative tests. This is consistent with the persistence of uncertainty and diagnostic delay reported under exclusion-based pathways, where repeated normal findings rarely produce closure for either clinician or patient.

Second, because biological and experiential aspects are treated as dimensions of one process rather than separate causes, the question of which is primary does not arise. The recurrent instability of attempts to classify BMS as either psychogenic or neuropathic [98] follows from this: the dispute is not unresolved for want of data but ill-posed, since the two designate aspects of a single condition rather than competing origins.

Third, validation and narrative reframing are not supportive additions to a biological treatment but interventions on the same loop that sustains the symptom; on this account they would be expected to bear on the pain itself, and not only on coping or quality of life, a prediction that remains to be tested directly. This expectation is not merely speculative. Controlled and observational studies of cognitive and psychotherapeutic interventions in BMS report reductions in pain intensity rather than in distress alone [100,101], and the quality of clinical communication and validation has been shown to modulate pain through expectation and the therapeutic relationship [102]. The framework treats such effects as consistent with, rather than incidental to, the hypothesis that experience participates in the mechanism that maintains the condition. This view extends prior work conceptualizing disease as an interface between lifeworld and scientific abstraction [103], and aligns with enactive accounts in which health and illness are brought forth through an organism's engagement with its world rather than residing in either pole alone [96].

As distinguished in Section 6.1, the difference from nociplastic accounts is sharpest where biology is indeterminate, as in BMS, and narrows where a discrete lesion is present: the claim is not that lived experience is constitutive of every condition, but that it becomes the decisive evidential and explanatory level when no lesion is available to carry that role. Used in this sense, "bidirectional" denotes reciprocal causation within an integrated system rather than parallel influences acting on a shared substrate.

Downward influences, in which affective and cognitive states reshape biological processes, are, we suggest, at least as plausible as upward mechanisms [90], and phenomenology grounds this view by conceiving the human being as an integrated, sense-making whole rather than a juxtaposition of mind and body [104]. Once illness is recognized as primary evidence rather than a derivative expression of disease, causal explanation must account for its active role. The relationship between pain and distress in BMS, for example, is better understood not as a sequence but as a self-reinforcing loop (Figure 2). In this loop, life stressors, altered neuroimmune regulation, burning pain, vigilance, distress, sympathetic activation, and increased nociceptive gain are understood as mutually reinforcing processes rather than as isolated causes. Life events such as bereavement or chronic stress may recalibrate neuroimmune and autonomic processes, contributing to the onset of burning sensations (downward influence). The persistence of pain—especially when it lacks clear validation—can in turn generate heightened vigilance, anxiety, and

depressive states, further modulating physiological responses and sustaining the condition (upward influence).

From this perspective, illness without lesion may involve distributed rather than localized causation [90]. The absence of a discrete lesion does not imply that the condition is uncaused or unreal, but that causality is distributed rather than localized, with the system as a whole—physiological processes, experiential states, and social contexts—becoming the site of dysfunction [42]. This does not negate contemporary neuropathic or nociplastic accounts of BMS [83], nor does it replace them with an alternative mechanism; it suggests they may be incomplete when they treat lived experience as an epiphenomenon rather than a constitutive element of the condition. So construed, the model clarifies several otherwise puzzling features of BMS: why exclusion-based strategies often fail to resolve patient uncertainty, why therapeutic responses are partial and heterogeneous, and why interventions addressing meaning, validation, and coping can exert measurable effects alongside pharmacological treatment. This distributed etiology is what dissolves the neuropathic-versus-psychogenic opposition noted above, rather than adding a third causal candidate to it. Clinical care therefore cannot be oriented toward identifying a single causal origin but must address the network of interactions through which the condition is sustained, so that multimodal approaches—combining pharmacological modulation, psychological support, and social intervention—emerge not as pragmatic compromises but as theoretically grounded responses.

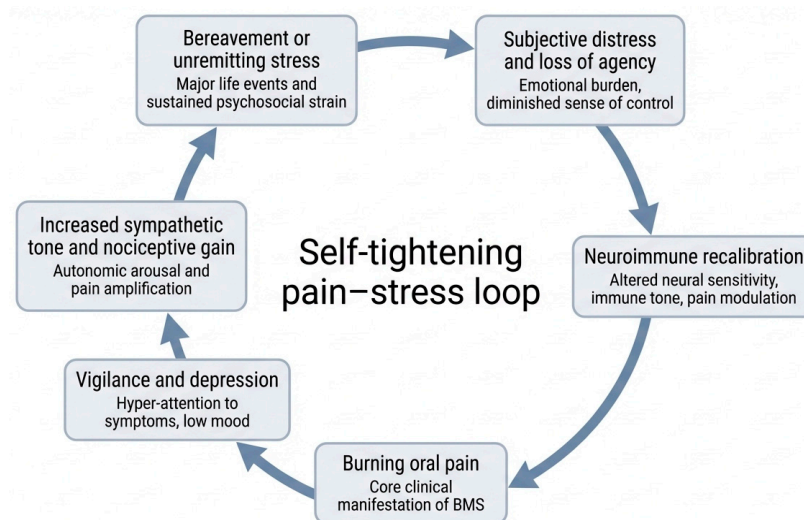


Figure 2. The bidirectional pain–stress loop in Burning Mouth Syndrome, in which physiological, experiential, and behavioral factors reciprocally reinforce one another.

6.3. Clinical Implications: Diagnosis, Validation, and Management

At the diagnostic level, the framework redirects clinical effort rather than relaxing diagnostic rigor. Current approaches identify persistent oral burning or dysesthetic pain in clinically normal mucosa and exclude secondary causes that may produce similar symptoms; this exclusion should be focused rather than indefinite, comprising careful history-taking, medication review, oral examination for mucosal disease, screening for candidiasis where clinically indicated, assessment of salivary function or xerostomia-related complaints, and relevant hematological and metabolic testing for deficiencies or systemic conditions known to produce oral burning [14].

Once these common and clinically actionable causes have been reasonably excluded, the diagnostic task should change: the absence of pathology should no longer function as a reason to keep searching for a hidden lesion, but as the threshold at which primary

BMS can be positively recognized on the basis of its clinical and experiential pattern. This threshold is a focused and proportionate assessment of major local, systemic, medication-related, mucosal, and metabolic causes according to the patient's presentation, risk factors, comorbidities, and local standards of care—not exhaustive exclusion of every theoretical abnormality. At that point, persistent burning, dysgeusia, xerostomia-like complaints, daily fluctuation, distress, and the patient's structured account of disruption become evidence for diagnosis rather than residual complaints awaiting validation. The proposed epistemic shift therefore enters clinical decision-making precisely at the transition from secondary-cause exclusion to positive diagnosis.

Validation acquires a defined place in this pathway. It does not mean endorsing every causal explanation offered by the patient, nor abandoning diagnostic caution; it means explicitly acknowledging that the pain is real, clinically significant, and worthy of treatment even when visible mucosal lesions or laboratory abnormalities are absent. This allows the patient to move from defending the legitimacy of symptoms to engaging in treatment [102].

Privileging subjective experience is an epistemic correction, not a diagnostic shortcut. It presupposes the distinction drawn earlier between the reality of suffering, which deserves recognition; the patient's account of that suffering, which may be partial, metaphorical, or shaped by distress; and causal interpretations of symptoms, which remain open to clinical assessment. In practice, this requires two concurrent lines of reasoning: on one track, the patient's pain and disruption are treated as clinically real and relevant from the outset; on the other, differential diagnosis, red-flag assessment, medication review, oral examination, and appropriate laboratory or specialist investigations remain active until common and actionable secondary causes have been reasonably excluded. Diagnostic closure should be documented as a proportionate clinical judgment rather than as a claim that every possible abnormality has been excluded, and the diagnosis should remain revisable if new mucosal signs, systemic symptoms, neurological findings, atypical progression, or unexpected treatment responses emerge. These safeguards allow lived experience to function as primary evidence without turning subjective reports into unchallengeable diagnostic conclusions.

At the therapeutic level, the framework reorders clinical priorities. Rather than sequencing treatments strictly according to a presumed single underlying mechanism, it supports early integration of multimodal strategies, including pharmacological pain modulation, management of oral symptoms, patient education, behavioral or psychological support where appropriate, and attention to stress, sleep, diet, and functional limitation [27]. These are not competing alternatives between biological and psychological care but complementary responses to a condition sustained across biological, experiential, and relational dimensions, so that validation and narrative engagement act within the same bidirectional system as physiological processes rather than as adjuncts to it.

Finally, the model also affects follow-up. Clinical review should assess not only pain intensity but also symptom predictability, distress, functional interference, coping, treatment expectations, and the patient's understanding of the diagnosis. By relinquishing the expectation of a single linear cause, clinicians can better accommodate the complexity of BMS while preserving diagnostic rigor. The practical value of the framework lies in clarifying decisions that already occur in BMS care—when to stop open-ended exclusion, when to name the condition, how to explain it without delegitimizing the patient's experience, and how to initiate multimodal management—rather than in replacing clinical judgment or existing diagnostic protocols.

6.4. Limitations of the Conceptual Framework

Several limitations should be acknowledged. As noted in Section 1, this article does not report new empirical data; its contribution is conceptual, and the limitations below follow directly from that scope. It relies on a selective integration of clinical literature on BMS with concepts from phenomenology and medical anthropology, and does not include the exhaustive search strategy, risk-of-bias assessment, or quantitative synthesis expected of a systematic review; the argument should accordingly be read as interpretive rather than as a comprehensive appraisal of all available evidence.

BMS is also clinically heterogeneous. Patients may differ in the relative contribution of peripheral neuropathic mechanisms, central nociplastic changes, psychological distress, systemic comorbidities, medication effects, salivary complaints, and life-world disruption [105]. The proposed bidirectional model is intended to accommodate this heterogeneity, but it should not be taken to imply that the same causal configuration applies to every patient: in some cases, biological mechanisms may be more prominent; in others, distress, vigilance, meaning, or social disruption may play a larger role.

Privileging lived experience as primary clinical evidence carries a risk of misunderstanding if separated from the diagnostic safeguards specified in Section 6.3. Its aim is to prevent the delegitimation of suffering while maintaining diagnostic rigor.

The practical implications proposed here require empirical testing. Future studies should assess whether earlier diagnostic closure, explicit validation, narrative reframing, and integrated multimodal care improve outcomes such as pain intensity, distress, functional limitation, coping, treatment adherence, diagnostic satisfaction, and patient–clinician communication. These limitations do not undermine the value of the framework but define its proper scope: a conceptual model intended to clarify clinical reasoning and generate testable hypotheses for future BMS research.

7. Conclusions: Clinical Implications of Treating Illness as Primary Evidence

The analysis of Burning Mouth Syndrome brings a persistent tension in contemporary medicine into focus: the tendency to equate clinical reality with demonstrable pathology, even where suffering is unmistakable and lesions remain indeterminate. In such cases, the challenge is not merely diagnostic but epistemic. As Tesio and Buzzoni have argued, the biological–clinical split reflects a deeper difficulty in integrating measurable dysfunction with lived experience [4]. BMS makes this difficulty explicit by presenting a condition in which illness is fully manifest while its pathological correlate remains elusive.

This paper has argued that such cases require medicine’s epistemic hierarchy to be reordered, in which illness is recognized as primary evidence and pathology as one—though not exclusive—mode of its explanation. Illness is not a derivative shadow cast by disease but a primary mode through which disorder is disclosed: structured, coherent, and directly accessible in experience, even when biological mechanisms remain uncertain [96]. BMS exemplifies this inversion. It transforms embodiment, reorganizes temporality and sociality, and generates a sustained crisis of legitimacy, yet does so with sufficient regularity to constitute a recognizable clinical entity. The patient’s account is therefore not a defective proxy for an unseen lesion but a primary source of evidence for lived suffering, requiring interpretation with the same rigor as laboratory findings [106]. As Kleinman observed, to focus exclusively on disease while neglecting illness is to leave the central reality of suffering unaddressed [38].

This epistemic shift has direct clinical implications. If illness is treated as primary evidence rather than as a provisional indicator, validation is no longer an ethical supplement but a justified clinical response. Listening without dismissiveness, naming the pain as real, and acknowledging its impact are not ancillary gestures but integral components of

diagnosis [107]. In conditions such as BMS, where exclusion-based strategies often fail to provide resolution, this reorientation can alter the trajectory of care, allowing patients to move from defensive justification toward forms of understanding and adaptation. As accounts of chronic pain suggest, this shift may mark the beginning of healing even in the absence of cure [108,109]. The contribution lies in identifying where lived experience is not one factor among others but the level at which conditions such as BMS are individuated and sustained, and in drawing out what this entails for diagnosis and treatment when no lesion is available to anchor them.

At a broader level, this perspective calls for a rebalancing of medical education and practice. While biomedical training equips clinicians to detect and manage pathology, it often leaves the capacity to interpret illness as lived experience underdeveloped. Integrating narrative competence and phenomenological sensitivity into training is therefore not an optional humanistic addition, but a necessary extension of clinical reasoning. Educational research supports the value of incorporating illness narratives, reflective practices, and relational skills into curricula, both to preserve empathy and to enhance interpretive capacity [110–117].

This distinction between cure and healing is not a concession to failure but a redefinition of therapeutic success, one that acknowledges the complexity of lived illness [118]. Taking lived experience seriously alongside laboratory evidence broadens medicine's scientific and clinical foundation, provided that validation remains coupled to appropriate diagnostic safeguards (Section 6.3). BMS demonstrates that when pathology cannot fully account for symptoms, illness itself becomes the most reliable point of departure for clinical understanding and care. In such contexts, diagnostic reasoning must extend beyond the search for identifiable lesions to include the structured reality of the patient's experience.

This shift does not replace biological investigation but repositions it within a broader interpretive framework in which subjective and objective evidence are integrated. In this setting, the clinician is not only a diagnostician but also an interpreter of symptoms and a partner in making the patient's condition intelligible, and where possible, manageable within everyday life. BMS thus serves not only as a clinical challenge but as a conceptual probe that exposes the limits of lesion-based medicine and compels a broader, non-reductive account of evidence, causality, and care.

Author Contributions: Conceptualization, C.G.; investigation, C.G., D.C. and G.A.T.d.O.; writing—original draft preparation, C.G. and D.C.; writing—review and editing, C.G., M.M., D.C. and G.A.T.d.O.; visualization, D.C. and G.A.T.d.O.; supervision, M.M. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: No new data were created in the present work.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Wu, S.; Zhang, W.; Yan, J.; Noma, N.; Young, A.; Yan, Z. Worldwide Prevalence Estimates of Burning Mouth Syndrome: A Systematic Review and Meta-analysis. *Oral Dis.* **2022**, *28*, 1431–1440. [[CrossRef](#)] [[PubMed](#)]
2. Adamo, D.; Spagnuolo, G. Burning Mouth Syndrome: An Overview and Future Perspectives. *Int. J. Environ. Res. Public Health* **2022**, *20*, 682. [[CrossRef](#)] [[PubMed](#)]
3. Martins Almeida, N.L.; Oliveira, G.M.; Santos, C.F.; Lara, V.S.; Cardoso, C.L. Challenges of Diagnosis and Management of Burning Mouth Syndrome: A Literature Review. *Curr. Aging Sci.* **2025**, *18*, 102–119. [[CrossRef](#)] [[PubMed](#)]

4. Tesio, L.; Buzzoni, M. The Illness-Disease Dichotomy and the Biological-Clinical Splitting of Medicine. *Med. Humanit.* **2021**, *47*, 507–512. [[CrossRef](#)] [[PubMed](#)]
5. Kleinman, A.; Eisenberg, L.; Good, B. Culture, Illness, and Care: Clinical Lessons from Anthropologic and Cross-Cultural Research. *Ann. Intern. Med.* **1978**, *88*, 251–258. [[CrossRef](#)] [[PubMed](#)]
6. Chmieliauskaite, M.; Stelson, E.A.; Epstein, J.B.; Klasser, G.D.; Farag, A.; Carey, B.; Albuquerque, R.; Mejia, L.; Ariyawardana, A.; Nasri-Heir, C.; et al. Consensus Agreement to Rename Burning Mouth Syndrome and Improve International Classification of Diseases-11 Disease Criteria: An International Delphi Study. *Pain* **2021**, *162*, 2548–2557. [[CrossRef](#)] [[PubMed](#)]
7. Sullivan, M. The New Subjective Medicine: Taking the Patient's Point of View on Health Care and Health. *Soc. Sci. Med.* **2003**, *56*, 1595–1604. [[CrossRef](#)] [[PubMed](#)]
8. Sensky, T. Suffering. *Int. J. Integr. Care* **2010**, *10*, e024. [[CrossRef](#)] [[PubMed](#)]
9. Jackson, C.; Vaughan, D.R.; Brown, L. Discovering Lived Experiences through Descriptive Phenomenology. *Int. J. Contemp. Hosp. Manag.* **2018**, *30*, 3309–3325. [[CrossRef](#)]
10. Olesen, J. International Classification of Headache Disorders. *Lancet Neurol.* **2018**, *17*, 396–397. [[CrossRef](#)] [[PubMed](#)]
11. International Classification of Orofacial Pain, 1st Edition (ICOP). *Cephalalgia* **2020**, *40*, 129–221. [[CrossRef](#)] [[PubMed](#)]
12. World Health Organization. *Icd-11 for Mortality and Morbidity Statistics-Burning Mouth Syndrome*; World Health Organization: Geneva, Switzerland, 2020.
13. Ariyawardana, A.; Chmieliauskaite, M.; Farag, A.M.; Albuquerque, R.; Forssell, H.; Nasri-Heir, C.; Klasser, G.D.; Sardella, A.; Mignogna, M.D.; Ingram, M.; et al. World Workshop on Oral Medicine VII: Burning Mouth Syndrome: A Systematic Review of Disease Definitions and Diagnostic Criteria Utilized in Randomized Clinical Trials. *Oral Dis.* **2019**, *25*, 141–156. [[CrossRef](#)] [[PubMed](#)]
14. de Lima-Souza, R.A.; Pérez-de-Oliveira, M.E.; Normando, A.G.C.; Louredo, B.V.R.; Mariano, F.V.; Farag, A.M.; Santos-Silva, A.R. Clinical and Epidemiological Profile of Burning Mouth Syndrome Patients Following the International Headache Society Classification: A Systematic Review and Meta-Analysis. *Oral Surg. Oral Med. Oral Pathol. Oral Radiol.* **2024**, *137*, 119–135. [[CrossRef](#)] [[PubMed](#)]
15. Canfora, F.; Calabria, E.; Armogida, N.G.; Ottaviani, G.; Mignogna, M.D.; Spagnuolo, G.; Adamo, D. Burning Mouth Syndrome: Updates on Pathogenesis and Diagnostic Algorithms. *J. Oral Facial Pain Headache* **2025**, *39*, 1–30. [[CrossRef](#)] [[PubMed](#)]
16. Kouri, M.; Adamo, D.; Vardas, E.; Georgaki, M.; Canfora, F.; Mignogna, M.D.; Nikitakis, N. Small Fiber Neuropathy in Burning Mouth Syndrome: A Systematic Review. *Int. J. Mol. Sci.* **2024**, *25*, 11442. [[CrossRef](#)] [[PubMed](#)]
17. Porporatti, A.L.; Schroder, Â.G.D.; Lebel, A.; Moreau, N.; Réaux-Le Goazigo, A.; Taihi, I.; Rittner, H.; Renton, T.; Boucher, Y. Morpho-Histo-Cytological Evaluation of Oral Tissues in Burning Mouth Syndrome: A Systematic Review. *J. Oral Rehabil.* **2025**, *52*, 2449–2480. [[CrossRef](#)] [[PubMed](#)]
18. Fischer, S.; Tsoumpas, C.; Chana, P.; Feltbower, R.G.; Aggarwal, V.R. Neuroimaging Signatures of Temporomandibular Disorder and Burning Mouth Syndrome: A Systematic Review. *Dent. J.* **2025**, *13*, 340. [[CrossRef](#)] [[PubMed](#)]
19. Galli, F.; Lodi, G.; Sardella, A.; Vegni, E. Role of Psychological Factors in Burning Mouth Syndrome: A Systematic Review and Meta-Analysis. *Cephalalgia* **2017**, *37*, 265–277. [[CrossRef](#)] [[PubMed](#)]
20. Porporatti, A.L.; Schroder, Â.G.D.; Lebel, A.; Moreau, N.; Misery, L.; Alajbeg, I.; Braud, A.; Boucher, Y. Is Burning Mouth Syndrome Associated with Stress? A Meta-analysis. *J. Oral Rehabil.* **2023**, *50*, 1279–1315. [[CrossRef](#)] [[PubMed](#)]
21. Lee, S.J.; Kim, C.; Yu, H.; Kim, D.-K. Relationship of Depression, Anxiety, and Bipolar Disease with Burning Mouth Syndrome: A Nationwide Cohort Study. *Int. J. Environ. Res. Public Health* **2023**, *20*, 3391. [[CrossRef](#)] [[PubMed](#)]
22. Kim, J.-Y.; Kim, Y.S.; Ko, I.; Kim, D.-K. Association between Burning Mouth Syndrome and the Development of Depression, Anxiety, Dementia, and Parkinson Disease. *JAMA Otolaryngol.–Head Neck Surg.* **2020**, *146*, 561–569. [[CrossRef](#)] [[PubMed](#)]
23. Adamo, D.; Calabria, E.; Canfora, F.; Coppola, N.; Pecoraro, G.; D'Aniello, L.; Aria, M.; Mignogna, M.D.; Leuci, S. Burning Mouth Syndrome: Analysis of Diagnostic Delay in 500 Patients. *Oral Dis.* **2024**, *30*, 1543–1554. [[CrossRef](#)] [[PubMed](#)]
24. Oh, C.-K.; Ju, H.-M.; Jeong, S.-H.; Ahn, Y.-W.; Jeon, H.-M.; Ok, S.-M. Burning Mouth Syndrome 2026: From “Diagnosis of Exclusion” to a Structured Diagnostic Algorithm (Narrative Review). *J. Oral Med. Pain* **2026**, *51*, 20–28. [[CrossRef](#)]
25. Goncalves, S.; Carey, B.; Farag, A.M.; Kuten-Shorrer, M.; Natto, Z.S.; Ariyawardana, A.; Mejia, L.M.; Chmieliauskaite, M.; Miller, C.S.; Ingram, M.; et al. WWOM VII: Effectiveness of Topical Interventions in the Management of Burning Mouth Syndrome: A Systematic Review. *Oral Dis.* **2023**, *29*, 3016–3033. [[CrossRef](#)] [[PubMed](#)]
26. Farag, A.M.; Kuten-Shorrer, M.; Natto, Z.; Ariyawardana, A.; Mejia, L.M.; Albuquerque, R.; Carey, B.; Chmieliauskaite, M.; Miller, C.S.; Ingram, M.; et al. WWOM VII: Effectiveness of Systemic Pharmacotherapeutic Interventions in the Management of BMS: A Systematic Review and Meta-analysis. *Oral Dis.* **2023**, *29*, 343–368. [[CrossRef](#)] [[PubMed](#)]
27. Alvarenga-Brant, R.; Costa, F.O.; Mattos-Pereira, G.; Esteves-Lima, R.P.; Belém, F.V.; Lai, H.; Ge, L.; Gomez, R.S.; Martins, C.C. Treatments for Burning Mouth Syndrome: A Network Meta-Analysis. *J. Dent. Res.* **2023**, *102*, 135–145. [[CrossRef](#)] [[PubMed](#)]
28. Tan, H.L.; Smith, J.G.; Hoffmann, J.; Renton, T. A Systematic Review of Treatment for Patients with Burning Mouth Syndrome. *Cephalalgia* **2022**, *42*, 128–161. [[CrossRef](#)] [[PubMed](#)]

29. Farag, A.M.; Albuquerque, R.; Ariyawardana, A.; Chmieliauskaite, M.; Forssell, H.; Nasri-Heir, C.; Klasser, G.D.; Sardella, A.; Mignogna, M.D.; Ingram, M.; et al. World Workshop in Oral Medicine VII: Reporting of IMMPACT-recommended Outcome Domains in Randomized Controlled Trials of Burning Mouth Syndrome: A Systematic Review. *Oral Dis.* **2019**, *25*, 122–140. [[CrossRef](#)] [[PubMed](#)]
30. Eisenberg, L. Disease and Illness Distinctions between Professional and Popular Ideas of Sickness. *Cult. Med. Psychiatry* **1977**, *1*, 9–23. [[CrossRef](#)] [[PubMed](#)]
31. Kleinman, A. Concepts and a Model for the Comparison of Medical Systems as Cultural Systems. *Soc. Sci. Med. B* **1978**, *12*, 85–93. [[CrossRef](#)] [[PubMed](#)]
32. Galadí, J.A. The Mind-Body Problem: An Overview of Proposed Solutions. In *The Theory of Mind Under Scrutiny: Psychopathology, Neuroscience, Philosophy of Mind and Artificial Intelligence*; Springer: Cham, Switzerland, 2024; pp. 435–467.
33. Canguilhem, G. *Le Normal et Le Pathologique*; Quadrige (Paris. 1981); Presses Universitaires de France: Paris, France, 2013; ISBN 9782130619505.
34. Thessen, A.E.; Bogdan, P.; Patterson, D.J.; Casey, T.M.; Hinojo-Hinojo, C.; de Lange, O.; Haendel, M.A. From Reductionism to Reintegration: Solving Society’s Most Pressing Problems Requires Building Bridges between Data Types across the Life Sciences. *PLoS Biol.* **2021**, *19*, e3001129. [[CrossRef](#)] [[PubMed](#)]
35. Forsey, J.; Ng, S.; Rowland, P.; Freeman, R.; Li, C.; Woods, N.N. The Basic Science of Patient–Physician Communication: A Critical Scoping Review. *Acad. Med.* **2021**, *96*, S109–S118. [[CrossRef](#)] [[PubMed](#)]
36. Davis, J.E.; González, A.M. *To Fix or to Heal: Patient Care, Public Health, and the Limits of Biomedicine*; NYU Press: New York, NY, USA, 2016; Volume 3, ISBN 1479809586.
37. Egnew, T.R. Suffering, Meaning, and Healing: Challenges of Contemporary Medicine. *Ann. Fam. Med.* **2009**, *7*, 170–175. [[CrossRef](#)] [[PubMed](#)]
38. Kleinman, A. *The Illness Narratives: Suffering, Healing, and the Human Condition*; Hachette: London, UK, 2020; ISBN 154167460X.
39. Öhman, M.; Söderberg, S.; Lundman, B. Hovering Between Suffering and Enduring: The Meaning of Living with Serious Chronic Illness. *Qual. Health Res.* **2003**, *13*, 528–542. [[CrossRef](#)] [[PubMed](#)]
40. Cassell, E.J. *The Healer’s Art: A New Approach to the Doctor–Patient Relationship*; MIT Press: Cambridge, MA, USA, 1976.
41. Jennings, D. The Confusion Between Disease and Illness in Clinical Medicine. *CMAJ Can. Med. Assoc. J.* **1986**, *135*, 865–870.
42. Hahn, R.A. Rethinking “Illness” and “Disease”. *J. Dev. Soc.* **1984**, *18*, 1. [[CrossRef](#)] [[PubMed](#)]
43. Hofmann, B. On the Triad Disease, Illness and Sickness. *J. Med. Philos.* **2002**, *27*, 651–673. [[CrossRef](#)] [[PubMed](#)]
44. Ware, N.C. Suffering and the Social Construction of Illness: The Delegitimation of Illness Experience in Chronic Fatigue Syndrome. *Med. Anthropol. Q.* **1992**, *6*, 347–361. [[CrossRef](#)]
45. Hall, J.M.; Powell, J. Understanding the Person through Narrative. *Nurs. Res. Pract.* **2011**, *2011*, 293837. [[CrossRef](#)] [[PubMed](#)]
46. Bury, M. Illness Narratives: Fact or Fiction? *Sociol. Health Illn.* **2001**, *23*, 263–285. [[CrossRef](#)]
47. Paik, Y.-G.; Sung, J. Learning about Social Suffering through Illness Narrative: Possibilities and Challenges. *BMC Med. Educ.* **2025**, *25*, 978. [[CrossRef](#)] [[PubMed](#)]
48. Fine, Z.D. The Ripples, Post-Pebble: Phenomenological Explorations of Disability through Narrative. *J. Med. Person* **2015**, *13*, 82–90. [[CrossRef](#)]
49. Kerosuo, H. Lost in Translation: A Patient-centred Experience of Unintegrated Care. *Int. J. Public Sect. Manag.* **2010**, *23*, 372–380. [[CrossRef](#)]
50. Baba, W.A. Gaze and Medical Diagnosis: A Historical Account of Concepts in Biomedicine. *J. Res. Hist. Med.* **2021**, *10*, 147–156.
51. Seidlein, A.-H.; Salloch, S. Illness and Disease: An Empirical-Ethical Viewpoint. *BMC Med. Ethics* **2019**, *20*, 5. [[CrossRef](#)] [[PubMed](#)]
52. Keating, P.; Cambrosio, A. Does Biomedicine Entail the Successful Reduction of Pathology to Biology? *Perspect. Biol. Med.* **2004**, *47*, 357–371. [[CrossRef](#)] [[PubMed](#)]
53. Derksen, F.; Olde Hartman, T.; Bensing, J.; Lagro-Janssen, A. Empathy in General Practice—The Gap between Wishes and Reality: Comparing the Views of Patients and Physicians. *Fam. Pract.* **2018**, *35*, 203–208. [[PubMed](#)]
54. Toombs, S.K. The Meaning of Illness: A Phenomenological Approach to the Patient–Physician Relationship. *J. Med. Philos.* **1987**, *12*, 219–240. [[CrossRef](#)] [[PubMed](#)]
55. Gallagher, S. What Is Phenomenology? In *Phenomenology*; Gallagher, S., Ed.; Springer International Publishing: Cham, Switzerland, 2022; pp. 1–10, ISBN 978-3-031-11586-8.
56. Hill, C.O. Husserl and Phenomenology, Experience and Essence. In *Phenomenology and Existentialism in the Twentieth Century: Book One New Waves of Philosophical Inspirations*; Tymieniecka, A.-T., Ed.; Springer: Dordrecht, The Netherlands, 2009; pp. 9–22, ISBN 978-90-481-2725-2.
57. Carel, H. Pathology as a Phenomenological Tool. *Cont. Philos. Rev.* **2021**, *54*, 201–217. [[CrossRef](#)] [[PubMed](#)]
58. Gschwandtner, C.M. Körper, Leib, Gemüt, Seele, Geist: Conceptions of the Self in Early Phenomenology. In *Gerda Walther’s Phenomenology of Sociality, Psychology, and Religion*; Springer: Cham, Switzerland, 2018; pp. 85–99.

59. Husserl, E. *Ideas Pertaining to a Pure Phenomenology and to a Phenomenological Philosophy*; Second Book: Studies in the Phenomenology of Constitution; Kluwer: Dordrecht, The Netherlands, 1989.
60. Slatman, J. The Körper-Leib Distinction. In *50 Concepts for a Critical Phenomenology*; Northwestern University Press: Evanston, IL, USA, 2019; pp. 203–209.
61. Merleau-Ponty, M.; Smith, C. *Phenomenology of Perception*; Routledge: London, UK, 2015; Volume 26.
62. Carman, T. The Body in Husserl and Merleau-Ponty. *Philos. Top.* **1999**, *27*, 205–226. [[CrossRef](#)]
63. Walsh, R.D. Husserl's Epoche as Method and Truth. *Auslegung J. Philos.* **1988**, *14*, 211–223.
64. Husserl, E. *Ideas Pertaining to a Pure Phenomenology and to a Phenomenological Philosophy*; First Book: General Introduction to a Pure Phenomenology; Springer: Dordrecht, The Netherlands, 1982.
65. Bovey, M.; Hosny, N.; Dutray, F.; Heim, E. Trauma-Related Cultural Concepts of Distress: A Systematic Review of Qualitative Literature from the Middle East and North Africa, and Sub-Saharan Africa. *SSM-Ment. Health* **2025**, *7*, 100402. [[CrossRef](#)]
66. Chatterjee, P.; Tsai, T.C.; Jha, A.K. Delivering Value by Focusing on Patient Experience. *Am. J. Manag. Care* **2015**, *21*, 735–737. [[PubMed](#)]
67. McClimans, L.M. *Patient-Centered Measurement: Ethics, Epistemology, and Dialogue in Contemporary Medicine*; Oxford University Press: Oxford, UK, 2024; ISBN 0197572073.
68. Bredlau, S. Illness as a Phenomenon of Being-in-the-World with Others: Plato's Charmides, Kleinman and Merleau-Ponty. *Med. Humanit.* **2021**, *47*, 20–26. [[CrossRef](#)] [[PubMed](#)]
69. Svenaeus, F. Illness as Unhomelike Being-in-the-World: Heidegger and the Phenomenology of Medicine. *Med. Health Care Philos.* **2011**, *14*, 333–343. [[CrossRef](#)] [[PubMed](#)]
70. Charmaz, K.; Rosenfeld, D. Reflections of the Body, Images of Self: Visibility and Invisibility in Chronic Illness and Disability. In *Body/Embodiment*; Routledge: London, UK, 2016; pp. 35–49.
71. Ahlén, R. Illness as Unhomelike Being-in-the-World? Phenomenology and Medical Practice. *Med. Health Care Philos.* **2011**, *14*, 323–331. [[CrossRef](#)] [[PubMed](#)]
72. Husserl, E. *The Crisis of European Sciences and Transcendental Phenomenology: An Introduction to Phenomenological Philosophy*; Northwestern University Press: Evanston, IL, USA, 1970.
73. Husserl, E. *The Phenomenology of Internal Time-Consciousness*; Heidegger, M., Ed.; Indiana University Press: Bloomington, IN, USA, 1964.
74. Michael, S.R. Integrating Chronic Illness into One's Life: A Phenomenological Inquiry. *J. Holist. Nurs.* **1996**, *14*, 251–267. [[PubMed](#)]
75. Hakeberg, M.; Hallberg, L.R.; Berggren, U. Burning Mouth Syndrome: Experiences from the Perspective of Female Patients. *Eur. J. Oral Sci.* **2003**, *111*, 305–311. [[CrossRef](#)] [[PubMed](#)]
76. Svenaeus, F. The Phenomenology of Chronic Pain: Embodiment and Alienation. *Cont. Philos. Rev.* **2015**, *48*, 107–122. [[CrossRef](#)]
77. Nilsen, G.; Elstad, I. Temporal Experiences of Persistent Pain. Patients' Narratives from Meetings with Health Care Providers. *Int. J. Qual. Stud. Health Well-Being* **2009**, *4*, 51–61. [[CrossRef](#)]
78. Leder, D. *The Absent Body*; University of Chicago Press: Chicago, IL, USA, 1990; ISBN 0226470008.
79. Aherne, S.; Trace, A.; Curtin, S.; McCreary, C. Women's Experiences of Living with Burning Mouth Syndrome: An Interpretative Phenomenological Analysis. *Adv. Dent. Oral Health* **2017**, *5*, 555664. [[CrossRef](#)]
80. López-Jornet, P.; Collado, Y.; Zambudio, A.; Pons-Fuster, E.; Castillo Felipe, C.; Tvarijonavičiute, A. Chemosensory Function in Burning Mouth Syndrome a Comparative Cross-Sectional Study. *Nutrients* **2021**, *13*, 722. [[CrossRef](#)] [[PubMed](#)]
81. Suarez, P.; Clark, G.T. Burning Mouth Syndrome: An Update on Diagnosis and Treatment Methods. *J. Calif. Dent. Assoc.* **2006**, *34*, 611–622. [[CrossRef](#)]
82. Toombs, S.K. Illness and the Paradigm of Lived Body. *Theor. Med.* **1988**, *9*, 201–226. [[CrossRef](#)] [[PubMed](#)]
83. Russo, M.; Crafa, P.; Guglielmetti, S.; Franzoni, L.; Fiore, W.; Di Mario, F. Burning Mouth Syndrome Etiology: A Narrative Review. *J. Gastrointest. Liver Dis.* **2022**, *31*, 223–228. [[CrossRef](#)] [[PubMed](#)]
84. Wade, B.L. The Meaning of Chronic Pain: A Phenomenological Analysis. *S. Afr. J. Physiother.* **2003**, *59*, a196. [[CrossRef](#)]
85. Tan, H.L.; Renton, T. Burning Mouth Syndrome: An Update. *Cephalalgia Rep.* **2020**, *3*, 2515816320970143. [[CrossRef](#)]
86. Karayannis, N.V.; Baumann, I.; Sturgeon, J.A.; Melloh, M.; Mackey, S.C. The Impact of Social Isolation on Pain Interference: A Longitudinal Study. *Ann. Behav. Med.* **2019**, *53*, 65–74. [[CrossRef](#)] [[PubMed](#)]
87. Nunes, D.A.P.; Gomes, J.F.; Neto, F.; de Matos, D.M. Chronic Pain and Language: A Topic Modelling Approach to Personal Pain Descriptions. *arXiv* **2021**, arXiv:2109.00402.
88. Jiang, Y.; Shi, Z.; Liu, A. Experiences of Social Isolation in Older Adults with Chronic Diseases: A Qualitative Study Based on Patients' Perspectives. *Healthcare* **2025**, *13*, 902. [[CrossRef](#)] [[PubMed](#)]
89. Canfora, F.; Calabria, E.; Spagnuolo, G.; Coppola, N.; Armogida, N.G.; Mazzaccara, C.; Solari, D.; D'Aniello, L.; Aria, M.; Pecoraro, G.; et al. Salivary Complaints in Burning Mouth Syndrome: A Cross Sectional Study on 500 Patients. *J. Clin. Med.* **2023**, *12*, 5561. [[CrossRef](#)] [[PubMed](#)]

90. Sharpe, M.; Greco, M. Chronic Fatigue Syndrome and an Illness-Focused Approach to Care: Controversy, Morality and Paradox. *Med. Humanit.* **2019**, *45*, 183–187. [[CrossRef](#)] [[PubMed](#)]
91. Engel, G.L. The Need for a New Medical Model: A Challenge for Biomedicine. *Science* **1977**, *196*, 129–136. [[CrossRef](#)] [[PubMed](#)]
92. Bolton, D.; Gillett, G. *The Biopsychosocial Model of Health and Disease: New Philosophical and Scientific Developments*; Palgrave Macmillan: Cham, Switzerland, 2019.
93. Nicholas, M.; Vlaeyen, J.W.S.; Rief, W.; Barke, A.; Aziz, Q.; Benoliel, R.; Cohen, M.; Evers, S.; Giamberardino, M.A.; Goebel, A.; et al. The IASP Classification of Chronic Pain for ICD-11: Chronic Primary Pain. *Pain* **2019**, *160*, 28–37. [[CrossRef](#)] [[PubMed](#)]
94. Pinto-Pardo, N.; Rodriguez-Zaninovic, M.P. Redefining Burning Mouth Syndrome: A Nociceptive Pain Disorder? *J. Dent. Sci.* **2024**, *20*, 1382–1383. [[CrossRef](#)] [[PubMed](#)]
95. Tabor, A.; Thacker, M.A.; Moseley, G.L.; Körding, K.P. Pain: A Statistical Account. *PLoS Comput. Biol.* **2017**, *13*, e1005142. [[CrossRef](#)] [[PubMed](#)]
96. Svenaeus, F. Health and Illness as Enacted Phenomena. *Topoi* **2022**, *41*, 373–382. [[CrossRef](#)] [[PubMed](#)]
97. Cohen, S.; Janicki-Deverts, D.; Miller, G.E. Psychological Stress and Disease. *JAMA* **2007**, *298*, 1685–1687. [[CrossRef](#)] [[PubMed](#)]
98. Vieira, W.F.; Coelho, D.R.A.; Litwiler, S.T.; McEachern, K.M.; Clancy, J.A.; Morales-Quezada, L.; Cassano, P. Neuropathic Pain, Mood, and Stress-Related Disorders: A Literature Review of Comorbidity and Co-Pathogenesis. *Neurosci. Biobehav. Rev.* **2024**, *161*, 105673. [[CrossRef](#)] [[PubMed](#)]
99. Chapman, C.R.; Tuckett, R.P.; Song, C.W. Pain and Stress in a Systems Perspective: Reciprocal Neural, Endocrine, and Immune Interactions. *J. Pain* **2008**, *9*, 122–145. [[CrossRef](#)] [[PubMed](#)]
100. Bergdahl, J.; Anneroth, G.; Ferris, H. Cognitive Therapy in the Treatment of Patients with Resistant Burning Mouth Syndrome: A Controlled Study. *J. Oral Pathol. Med.* **1995**, *24*, 213–215. [[CrossRef](#)] [[PubMed](#)]
101. Miziara, I.D.; Araújo Filho, B.C.; Oliveira, R.; dos Santos, R.M.R. Group Psychotherapy: An Additional Approach to Burning Mouth Syndrome. *J. Psychosom. Res.* **2009**, *67*, 443–448. [[CrossRef](#)] [[PubMed](#)]
102. Benedetti, F. Placebo and the New Physiology of the Doctor-Patient Relationship. *Physiol. Rev.* **2013**, *93*, 1207–1246. [[CrossRef](#)] [[PubMed](#)]
103. Galli, C. Threshold of Understanding: Disease as the Interface of Lifeworld and Science. *Med. Health Care Philos.* **2025**, *29*, 67–73. [[CrossRef](#)] [[PubMed](#)]
104. Buzzoni, M.; Tesio, L.; Stuart, M.T. Holism and Reductionism in the Illness/Disease Debate. In *From Electrons to Elephants and Elections: Exploring the Role of Content and Context*; Wuppuluri, S., Stewart, I., Eds.; Springer International Publishing: Cham, Switzerland, 2022; pp. 743–778, ISBN 978-3-030-92192-7.
105. Orliaguet, M.; Misery, L. Neuropathic and Psychogenic Components of Burning Mouth Syndrome: A Systematic Review. *Biomolecules* **2021**, *11*, 1237. [[CrossRef](#)] [[PubMed](#)]
106. Salana, K.; Maty, S.; Hage, R. A Whole Other Story: Interpreting Narrative Medicine. *Glob. Adv. Health Med.* **2021**, *10*, 1–6. [[CrossRef](#)] [[PubMed](#)]
107. Mudiyanse, R.M. Empathy for Patient Centeredness and Patient Empowerment. *J. Gen. Pract.* **2016**, *4*, 224. [[CrossRef](#)]
108. Charon, R. *Narrative Medicine: Honoring the Stories of Illness*; Oxford University Press: Oxford, UK, 2008; ISBN 0195340221.
109. Cassell, E.J. The Nature of Suffering and the Goals of Medicine. *Loss Grief Care* **1998**, *8*, 129–142. [[CrossRef](#)]
110. Kumagai, A.K. A Conceptual Framework for the Use of Illness Narratives in Medical Education. *Acad. Med.* **2008**, *83*, 653–658. [[CrossRef](#)] [[PubMed](#)]
111. Holmes, S.M. Learning Language, Un/Learning Empathy in Medical School. *Cult. Med. Psychiatry* **2025**, *49*, 40–64. [[CrossRef](#)] [[PubMed](#)]
112. Brodahl, K.Ø.; Storøy, H.-L.E.; Finset, A.; Pedersen, R. Medical Students' Experiences When Empathizing with Patients' Emotional Issues during a Medical Interview—A Qualitative Study. *BMC Med. Educ.* **2022**, *22*, 145. [[CrossRef](#)] [[PubMed](#)]
113. Le, A.; Miller, K.; McMullin, J. From Particularities to Context: Refining Our Thinking on Illness Narratives. *AMA J. Ethics* **2017**, *19*, 304–311. [[CrossRef](#)] [[PubMed](#)]
114. Jones, D.S. A Complete Medical Education Includes the Arts and Humanities. *AMA J. Ethics* **2014**, *16*, 636–641. [[CrossRef](#)]
115. Shield, R.R.; Tong, I.; Tomas, M.; Besdine, R.W. Teaching Communication and Compassionate Care Skills: An Innovative Curriculum for Pre-Clerkship Medical Students. *Med. Teach.* **2011**, *33*, e408–e416. [[CrossRef](#)] [[PubMed](#)]
116. Egnew, T.R. A Narrative Approach to Healing Chronic Illness. *Ann. Fam. Med.* **2018**, *16*, 160–165. [[CrossRef](#)] [[PubMed](#)]
117. Lindsey, E. The Gift of Healing in Chronic Illness/Disability. *J. Holist. Nurs.* **1995**, *13*, 287–305. [[CrossRef](#)] [[PubMed](#)]
118. Leder, D.; Aho, K. Transformational Healing: The Phenomenology of Fulfilment in the Face of Adversity. *Med. Humanit.* **2025**, *51*, 59–66. [[CrossRef](#)] [[PubMed](#)]

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.