

Concise Review

Clinical Practice Guidelines on the Diagnosis and Treatment of Halitosis



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ABSTRACT

With growing awareness of oral health and rising social expectations, the management of halitosis has attracted increasing attention, as fresh breath is essential for social etiquette and personal image. Due to the diverse origins and varying clinical manifestations of halitosis, treatment approaches differ significantly. Recent advancements have facilitated the development of more refined diagnostic tools and therapeutic methods. Based on current clinical evidence and practice, a consensus framework has been established to guide the management of halitosis. This consensus systematically outlines the prevalence and classification of halitosis, reviews its aetiology and pathogenesis, and details evidence-based diagnostic methods. Notably, it proposes tailored treatment strategies aligned with specific etiological factors. The goal is to promote the standardization, precision, and optimization of halitosis diagnosis and treatment in clinical practice.

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Introduction

Halitosis (oral malodour) is a clinical condition characterized by the unpleasant breath odour originating from the oral or nasal cavity during exhalation, which is etiologically classified as genuine or delusional halitosis. It originates from intraoral sources (predominantly microorganisms in tongue coating and periodontal tissues) or extraoral causes (such as gastrointestinal disorders). Clinically, key diagnostic methods include: organoleptic assessment, gas chromatography (GC), and sulphide monitoring. Given its multifactorial nature, effective treatment mandates precise etiological diagnosis followed by targeted interventions. These may encompass oral hygiene maintenance, periodontal therapy, pharmacological treatment, and management of systemic conditions. Heightened public health awareness has driven advances in diagnostic and therapeutic approaches.

We first systematically developed a comprehensive theoretical framework covering the epidemiology, classification, etiological mechanisms, diagnostic procedures, and treatment strategies for halitosis. Building on this foundation, we conducted an extensive review and integration of recent cutting-edge research findings from both domestic and international sources, allowing us to incorporate several breakthrough diagnostic technologies and innovative therapeutic approaches. At the same time, we deeply synthesized the valuable practical experience accumulated by frontline clinicians through long-term patient management and transformed these insights into a draft guideline with high clinical applicability. On this basis, we were honoured to invite numerous authoritative experts in the field of dentistry

across China to form a steering committee. Through multiple rounds of rigorous review and refinement, we continuously incorporated high-level academic feedback. Ultimately, these efforts resulted in a clinical guideline that represents the current professional consensus and embodies both forward-looking vision and practical value. It provides clinicians with evidence-based, standardized guidance for diagnostic and therapeutic consistency.

Epidemiology of halitosis

Extensive research on halitosis prevalence reveals significant variation across populations. Specific studies illustrate this: a 2006 Chinese epidemiological survey of 2,000 individuals aged 15 to 65 found a prevalence of 27.5%,¹ while 2010 American Dental Association data indicated up to 50% affected Americans.² An Italian clinical trial in 2015 reported 53.51%.³ By 2020, a review estimated a global prevalence between 15% and 60%,⁴ consistent with later findings: a 2022 New Zealand cross-sectional study (372 young adults) detected 31.2%,⁵ and another epidemiological study in the same year documented a range of 2.4% to 78%⁶ (Table). This substantial methodological heterogeneity underscores the lack of standardized diagnostic criteria. Additionally, this variability is influenced by several factors, including the geographical location of study participants, their pretest and testing conditions, the sophistication of diagnostic technology and equipment used, and clinicians' proficiency in both theoretical principles and clinical skills related to halitosis diagnosis and management. This variability hinders the collection and comparison of

Table – Epidemiology of halitosis.

Y	Country	Study	Sample size	Age range (y)	Diagnostic method	Reported prevalence
2006	China	Liu et al ¹	2000	15-65	An epidemiological survey	27.5%
2010	USA	Settineri et al ²	1052	15-65	American Dental Association data	50%
2015	Italy	Aimetti et al ³	744	20-75	A cross-sectional study	53.51%
2022	New Zealand	Yu et al ⁵	372	18-30	A cross-sectional study	31.2%

epidemiological data across different countries and impedes clinical diagnosis and treatment improvements. Therefore, establishing a unified and standardized diagnostic and treatment guideline for halitosis is urgently needed. Further large-scale, multiregional studies are also essential to fully understand its prevalence.

Classification of halitosis

Genuine halitosis

Physiological halitosis

Physiological halitosis originates primarily from bacterial putrefaction on the posterior tongue. Contributing factors include hunger, recent consumption of pungent foods (eg, garlic, onions, alcohol), and hormonal changes during menstruation.

Pathological halitosis

Pathological halitosis is classified as intraoral or extraoral, caused by oral or nonoral factors, respectively.^{6,7} Intraoral causes include tongue coating microbiota, periodontitis, odontogenic infections, Sjögren's syndrome, oral mucosal diseases, and malignancies. Extraoral causes encompass gastrointestinal disorders, respiratory diseases, endocrine disturbances, and medications.

Delusional halitosis

Pseudohalitosis

Pseudohalitosis refers to a patient's persistent belief in having halitosis that is not detectable by others. This belief typically resolves following dental consultation and oral hygiene instruction.

Halitophobia

Halitophobia describes a psychological condition where patients maintain a belief in having halitosis despite clinical evidence to the contrary. They persist in self-reporting halitosis even after receiving a professional diagnosis and treatment.

Aetiology and pathogenesis of halitosis

Intraoral causes

Intraoral sources are responsible for 80% to 90% of all halitosis cases, encompassing periodontal diseases, tongue coating, odontogenic diseases, mucosal disorders, and oral cancers.⁸ The characteristic olfactory profile of intraoral halitosis is dominated by volatile sulphur compounds (VSCs), primarily hydrogen sulphide (H_2S), methyl mercaptan (CH_3SH), and dimethyl sulphide ($(CH_3)_2S$). Trace amounts of other gases, including dimethyl disulphide (CH_3SSCH_3) and carbon disulphide (CS_2), have also been identified.⁹ These VSCs originate from the bacterial degradation of sulphur-containing amino acids by specific

oral microorganisms.⁴ Notable VSC-producing bacteria include *Porphyromonas*, *Tannerella forsythia*, *Fusobacterium*, *Prevotella*, *Peptostreptococcus*, *Eubacterium*, *Selenomonas*, *Leptotrichia*, *Bacteroides*, *Actinomyces*, and *Halobacterium*.⁴

Periodontal diseases

Studies confirm that patients with periodontal diseases exhibit elevated VSCs levels compared to healthy individuals,¹⁰ with VSCs concentrations directly proportional to disease severity.¹¹ Crucially, these elevated VSC levels exacerbate periodontal inflammation through a multistep mechanism. Anaerobic bacteria in dental plaque activate host immune responses, initiating periodontal inflammation and subsequent tissue destruction. During this process, both bacterial and host proteases degrade periodontal tissue proteins (eg, collagen). Subsequent proteolytic breakdown releases sulphur-containing amino acids (eg, cysteine, methionine) and iron ions. These substrates are central to a vicious cycle: (1) The amino acids serve as direct precursors for bacterial VSCs production; (2) iron ions act as essential nutrients, promoting bacterial proliferation and protease secretion. This amplifies periodontal tissue destruction and further VSC generation.¹⁰ Thus, periodontitis and halitosis form a self-perpetuating cycle of mutual exacerbation. Bacterial composition analysis reveals a distinct difference between periodontal diseases patients with halitosis and periodontally healthy individuals.⁴ Key alterations include, at the genus level, significantly elevated abundances of *Tannerella*, *Selenomonas*, *Bacteroides*, *Filifactor*, and *Acidobacteria*. At the phylum level, there is a marked increase in *Desulfobacterota* and *Synergistetes*. Furthermore, the periodontal pathogens 'red complex' has been identified to produce H_2S , CH_3SH , and CH_3SCH_3 VSCs directly contributing to halitosis.¹²

Tongue coating

Tongue coating manifests as a thin, whitish layer covering the dorsal tongue surface, typically thinner anteriorly and thicker posteriorly. Its composition includes desquamated epithelial cells, immune cells, food debris, saliva components, bacteria, and fungi. Tongue coating-associated malodour originates from two primary sources: odoriferous food debris itself, and VSCs generated via microbial proteolytic degradation, eg, by *Prevotella intermedia* (*P. intermedia*) and *Treponema denticola*.^{4,13}

Regular oral hygiene and adequate salivation preserve the coating's typical distribution, thickness, colouration, and composition. Conversely, factors like poor oral hygiene, Sjögren's syndrome, or microbial infections can alter the coating's characteristics. This manifests pathologically as thickened, discoloured (yellowish), or black hairy tongue formation, significantly promoting halitosis.

Odontogenic factors

It includes dead pulp teeth, pulp exposure, poorly contoured prostheses, dry sockets, dentition crowding, pericoronitis, fixed orthodontic appliance, peri-implantitis, etc.⁶ These regions are particularly difficult to clean, leading to the accumulation of food debris. This, in turn, serves as a nutrient source for the proliferation of oral bacteria, such as

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Fusobacterium nucleatum (*F. nucleatum*) and *Porphyromonas gingivalis* (*P. gingivalis*). These bacteria can anaerobically metabolize the accumulated organic substrates, including food debris and shed epithelial cells. This metabolic process is facilitated by the cysteine desulphurylase enzyme system, which contributes to the degradation of sulphur-containing amino acids, such as methionine and cysteine, into VSCs. The subsequent release of these VSCs can contribute to developing halitosis.¹²

Oral mucosal diseases

Oral mucosal lesions like recurrent ulcers can cause necrosis, ulceration, and pseudomembrane formation. This compromised mucosa fosters the accumulation of pathogenic microorganisms and the subsequent degradation of organic matter, leading to VSC production. Furthermore, infectious causes – including bacterial, fungal (eg, *Candida albicans*),¹⁴ and viral (eg, Herpes simplex virus) infections – similarly induce organic matter breakdown and thus halitosis.^{15,16}

Oral cancer

Oral malignancies cause local mucosal necrosis due to compromised blood supply.¹⁷ Additionally, persistent mechanical friction contributes to mucosal ulceration and bleeding. In the context of diminished local and systemic immunity in cancer patients,¹⁸ this damaged oral microenvironment promotes extensive colonization of anaerobic bacteria within the biofilm covering the lesions. These bacteria decompose necrotic tissue and other organic matter, generating VSCs and contributing to halitosis.¹⁹

Others

- Overnight denture wear: Micropores form during acrylic resin polymerization and adsorb salivary proteins via hydrophobic interactions.²⁰ Coupled with nocturnal hyposalivation, which impairs oral self-cleaning, this creates ideal conditions for anaerobic bacterial proliferation and odour production.²¹
- Starvation: Periods of starvation lead to a significant increase in $(\text{CH}_3)_2\text{S}$ level and promote proliferation of oral bacteria such as *Megasphaera*, *Dialister*, *Prevotella*, *Bifidobacterium*, *Treponema*, and *Selenomonas* – species strongly implicated in the genesis and exacerbation of halitosis.²²

Extraoral pathologies

Approximately 2% of halitosis originates from gastrointestinal, metabolic, or endocrine disorders, such as Zenker's diverticulum, inflammatory bowel disease (IBD), *Helicobacter pylori* (*Hp*) infection, and gastroesophageal reflux. Conversely, an additional 8% arises from respiratory or otorhinolaryngological conditions like sinusitis, tonsillitis, and pulmonary diseases.⁸ Other contributing factors include specific medications, tobacco use, chronic psychological stress, and potential genetic predispositions.

Gastrointestinal disorders

- Zenker diverticulum, achalasia, pyloric stenosis, oesophageal and gastric cancer: Gastrointestinal disorders such as Zenker diverticulum, achalasia, pyloric stenosis, oesophageal cancer, and gastric cancer promote halitosis through gastric content retention.²³ The resulting acidic environment stimulates gram-negative anaerobic bacteria to decompose organic material, producing VSCs²⁴ – a significant contributor to malodour.²
- IBD: IBD correlates with a higher prevalence of halitosis, partly attributable to concomitant oral manifestations such as recurrent aphthous ulcers, generalized gingival hyperplasia, periodontitis, and Sjögren's syndrome. These conditions facilitate colonization by VSCs-producing pathogenic bacteria, driving malodour development.²⁵ Additionally, IBD patients exhale distinctive volatile organic compounds (VOCs), which may originate from elevated oxidative stress during inflammatory flares.²⁶ This oxidative stress increases intestinal permeability, enabling VOCs to translocate across the mucosa into systemic circulation. Following hematogenous transfer, VOCs undergo pulmonary gas exchange and are ultimately excreted via breath.^{27,28}
- Infection with *Hp*: *Hp* infection contributes to halitosis through several mechanisms. *Hp* colonizes significant quantities in the saliva, heavy tongue coating, and supra-gingival and subgingival plaque of periodontitis patients. Critically, it can metabolically degrade sulphur-containing amino acids to produce H_2S and CH_3SH .^{25,29,30} Meanwhile, the gastritis or gastric ulcers it induces can lead to gastrointestinal dysfunction, further exacerbating the halitosis.³¹ Supporting this etiological role, studies demonstrate significant improvement in halitosis among *Hp*-positive patients following successful eradication therapy.³²
- Gastroesophageal reflux: Multiple systematic reviews and analyses indicate that patients with gastroesophageal reflux have significantly poorer oral health compared to healthy controls, which is strongly correlated with elevated levels of VSCs in their breath.^{31,33} Gastroesophageal reflux contributes to halitosis through both local and systemic mechanisms. The refluxate damages the oesophageal mucosa, triggering inflammation and epithelial cell detachment. This inflammatory cascade activates enzymatic pathways (eg, cystathionine γ -lyase) that metabolize sulphur-containing amino acids into VSCs, primarily H_2S and CH_3SH . These VSCs may subsequently enter the oral cavity directly during reflux episodes, or diffuse into systemic circulation and undergo pulmonary excretion via alveolar gas exchange, or be secreted into the oral cavity via saliva. Collectively, these pathways contribute significantly to halitosis.³⁴

Respiratory diseases

- Sinusitis: Acute sinusitis typically resolves with appropriate antibiotic therapy that can reduce anaerobic bacterial loads and suppress VSC production. Conversely, halitosis is predominantly attributed to chronic sinusitis, while a frequently underdiagnosed condition, chronic sinusitis pathogenesis involves persistent upper respiratory infections, chronic inflammatory mucosal disorders, and structural nasal deformities.⁶

- **Tonsillitis:** Anaerobic bacterial breakdown of desquamated cells within tonsillar crypts generates sulphur compounds. Inadequate cryptic hygiene promotes the accumulation of bacteria, food debris, and necrotic material, potentially leading to tonsillolith formation and elevating halitosis risk by up to 10-fold.^{6,35}
- **Pulmonary diseases:** Malodorous pulmonary conditions, including anaerobic lung abscesses, necrotic pneumonia, bronchogenic carcinoma, chronic obstructive pulmonary disease, and tuberculosis, share a common mechanism as follows: anaerobic bacteria proliferate within these lesions and degrade necrotic tissues, producing VSCs that are directly excreted into the oral cavity via exhalation, resulting in persistent halitosis.^{6,36-38}

Metabolic and endocrine disorders

Diabetes mellitus, renal failure, and liver cirrhosis/failure are recognized causes of halitosis. Volatile compounds produced by these metabolic and endocrine disorders enter the bloodstream, are exchanged in the lungs during respiration, and are exhaled, resulting in malodorous breath.³⁹⁻⁴³

- **Diabetes mellitus:** Hyperglycaemia can lead to metabolic changes in oral bacteria, while xerostomia exacerbates microbial dysbiosis, resulting in the production of more VSCs and an increase in malodour. Additionally, in diabetic patients, reduced glucose utilization and oxidation often stimulate fat breakdown and fatty acid oxidation, leading to increased production of ketone bodies.⁴⁴ When production exceeds extrahepatic tissue utilization, blood ketone body concentrations rise. These ketones enter the respiratory system via the bloodstream and are exhaled through the lungs, imparting a characteristic odour resembling rotten apples – specifically, ketoacidosis.⁴⁵
- **Renal failure:** Impaired renal function reduces urea nitrogen (BUN) clearance, elevating its blood concentration. Urea diffuses from the blood into saliva and the intestines, where bacterial degradation produces ammonia. This ammonia is absorbed back into the blood and subsequently excreted via respiration, producing a characteristic ammonia-based odour.⁴⁶
- **Fish odour syndrome:** Trimethylaminuria, also known as fish odour syndrome, is an autosomal recessive disorder characterized by trimethylamine (TMA) accumulation, which results in a foul, fish-like odour in breath, urine, and sweat. Deficient hepatic flavin-containing monooxygenase 3 (FMO3) activity impairs oxidation of TMA to odourless TMA N-oxide, leading to unmetabolized TMA accumulation in bodily fluids and the characteristic malodour.^{45,47}

Medications

Numerous medications can induce halitosis through distinct mechanisms. Agents that suppress salivary flow (eg, anticholinergics, antihistamines, antihypertensives, diuretics, anaesthetics) diminish the self-cleaning capacity of oral cavity. Saliva rinses away debris and bacteria while containing antimicrobial components like lysozyme and immunoglobulins. Reduced salivary secretion weakens these effects, promoting

bacterial proliferation. These bacteria then decompose sulphur-containing amino acids within food debris and necrotic tissues, releasing VSCs responsible for malodour.^{46,48}

A second mechanism involves sulphur-containing medications themselves (eg, penicillins, cephalosporins, sulphonamides, aminothiols, PX-12, omeprazole). During metabolism, these drugs can produce VSCs directly, contributing to malodorous breath.

Additionally, other classes of drugs, including steroids, bisphosphonates, fish oil, selenium, and vitamin E, have been associated with halitosis, although their precise mechanisms remain unclear and require further investigation.^{46,49}

Others

- **Smoking:** Smoking positively correlates with both the incidence and severity of halitosis. It reduces normal oral microorganism quantity, increases VSCs-producing bacteria, disrupts subgingival microbial ecology, and accelerates biofilm formation. Furthermore, smoking exacerbates malodour by reducing saliva secretion.⁵⁰
- **Psychological stress:** Psychological stress also correlates with malodour. Studies using hydrogen/methane and nitric oxide breath tests found significantly elevated dimethyl sulphide levels in individuals experiencing high stress.⁵¹
- **Gene:** According to a cross-sectional study, gingival fibroblasts of the AVI/AVI genotype showed weaker inhibition of anaerobic bacteria and higher baseline VSCs levels, as well as notable enrichment of *P. intermedia*, compared to the non-AVI/AVI genotype. Crucially, VSC levels remained unchanged in AVI/AVI individuals despite identical periodontal treatment and tongue cleaning. These findings indicate that hTAS2R38 polymorphisms modulate oral microbiota, influencing both halitosis prevalence and treatment outcomes.⁵²

Diagnostic techniques of halitosis

Comprehensive medical history collection and oral examination facilitate identifying the origin of halitosis. But the definitive diagnosis of halitosis needs to be verified by professional techniques (Figure 1).

Organoleptic method and GC

Currently, the primary clinical approaches for monitoring halitosis are the organoleptic method and GC.⁵⁰ The organoleptic method requires a physician to directly assess the patient's exhaled breath by smelling it and assigning an odour intensity score. Due to its low cost and direct results, this method remains the diagnostic gold standard for halitosis, despite its inherent subjectivity.

GC analyses exhaled breath by separating VSCs components, specifically quantifying hydrogen sulphide, methyl mercaptan, and dimethyl sulphide concentrations. This technique offers higher sensitivity, better specificity, and more objective results than the organoleptic method, eliminating its subjectivity. Furthermore, the quantitative nature of GC data enables tracking treatment efficacy in systemically

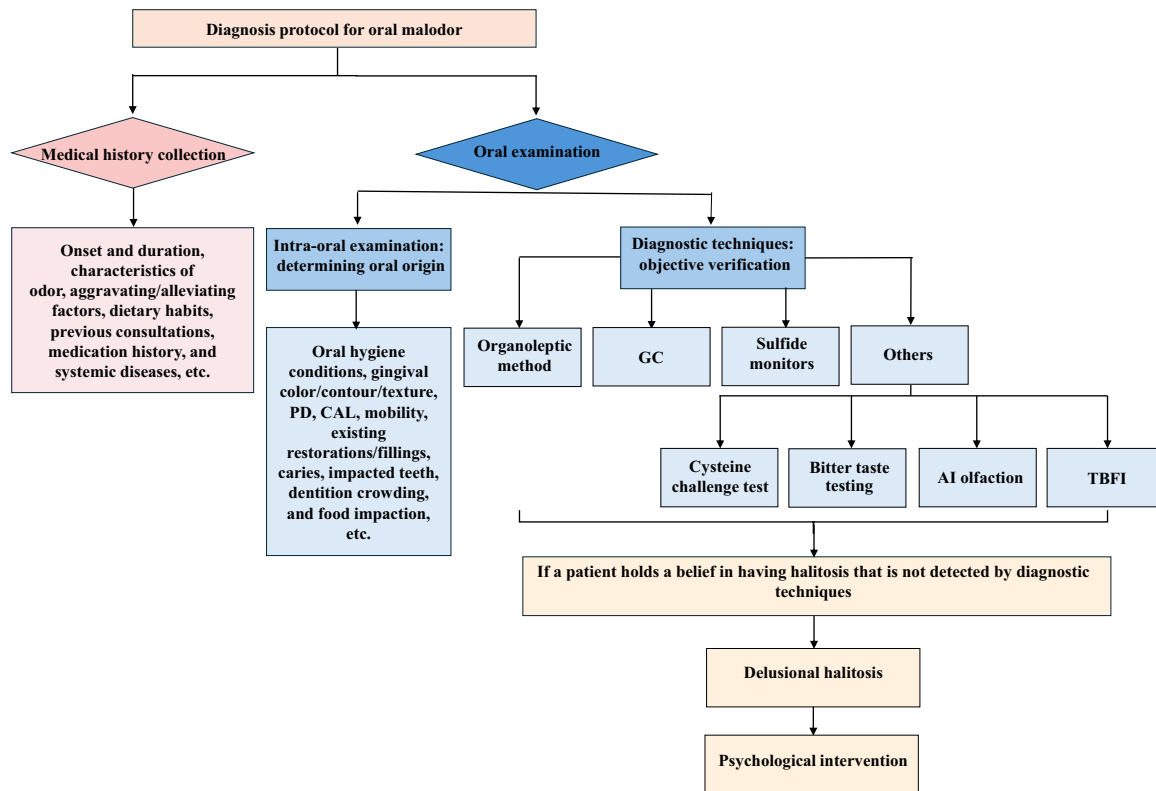


Fig. 1 – Diagnosis protocol for halitosis. AI, artificially intelligent; CAL, clinical attachment loss; GC, gas chromatography; PD, probing depth; TBFI, tongue biofilm fluorescence index.

treated patients. However, GC has higher costs and requires greater technical expertise and instrument sensitivity.⁵³⁻⁵⁵

Sulphide monitors

Portable sulphide monitors (eg, Halimeter, OralChroma, Breathtron) enable rapid clinical screening and assessment of malodour levels. However, their inability to qualitatively and quantitatively analyse exhaled gas components limits diagnostic precision, often yielding inconclusive results.⁵⁶

Cysteine challenge test

The cysteine challenge test serves not only to measure halitosis at a single time point but also to identify patients' susceptibility to developing chronic malodour.⁵⁶

Bitter taste testing

Recent research identified specialized chemosensory cells in murine gingival tissue expressing bitter taste receptors and signalling pathways. These cells correlated with shifts in oral microbiota composition and the severity of periodontal destruction in mice.⁵⁷ Human studies show bitter taste sensitivity varies by genotype. Nontasters (AVI/AVI) exhibit reduced bitter taste sensitivity compared to supertasters (PAV/PAV) and tasters (PAV/AVI). AVI/AVI individuals demonstrate weaker inhibition of anaerobic bacteria, elevated VSCs levels, and poorer responses to periodontal therapy and tongue cleaning for malodour control. These findings suggest bitter taste testing holds promise for screening malodour

susceptibility and aiding diagnosis, though clinical translation requires further validation.⁵⁵

Artificially intelligent (AI) olfaction

In recent years, AI has been widely applied across various specialties in dentistry.⁵⁸ Driven by rapid AI advancements, AI olfaction synergizes with VSCs monitors to analyse intra- and extrahalitosis. The method boasts high sensitivity, rapid reactivity, and can also assist in the analysis of patient case data, thus achieving high diagnostic efficiency. Key technologies enabling AI olfaction include exhaled breath analysis, E-nose, Odorant receptors, electrochemical sensors, and piezoelectric sensors, which facilitate AI-driven monitoring and diagnosis of halitosis.^{59,60}

Tongue biofilm fluorescence index (TBFI)

TBFI is an objective scoring system leveraging bacterial biofilm fluorescence to quantify tongue coating. The method captures dual-mode images of visible light photographs and biofilm fluorescence imaging of the dorsal tongue surface, then calculates fluorescence intensity and coverage area using standardized metrics.⁶¹ Statistical analyses reveal that elevated TBFI values are correlated with higher fluorescence intensity, greater biofilm coverage, and increased VSC levels. These findings demonstrate that TBFI can be a reliable diagnostic indicator for halitosis.

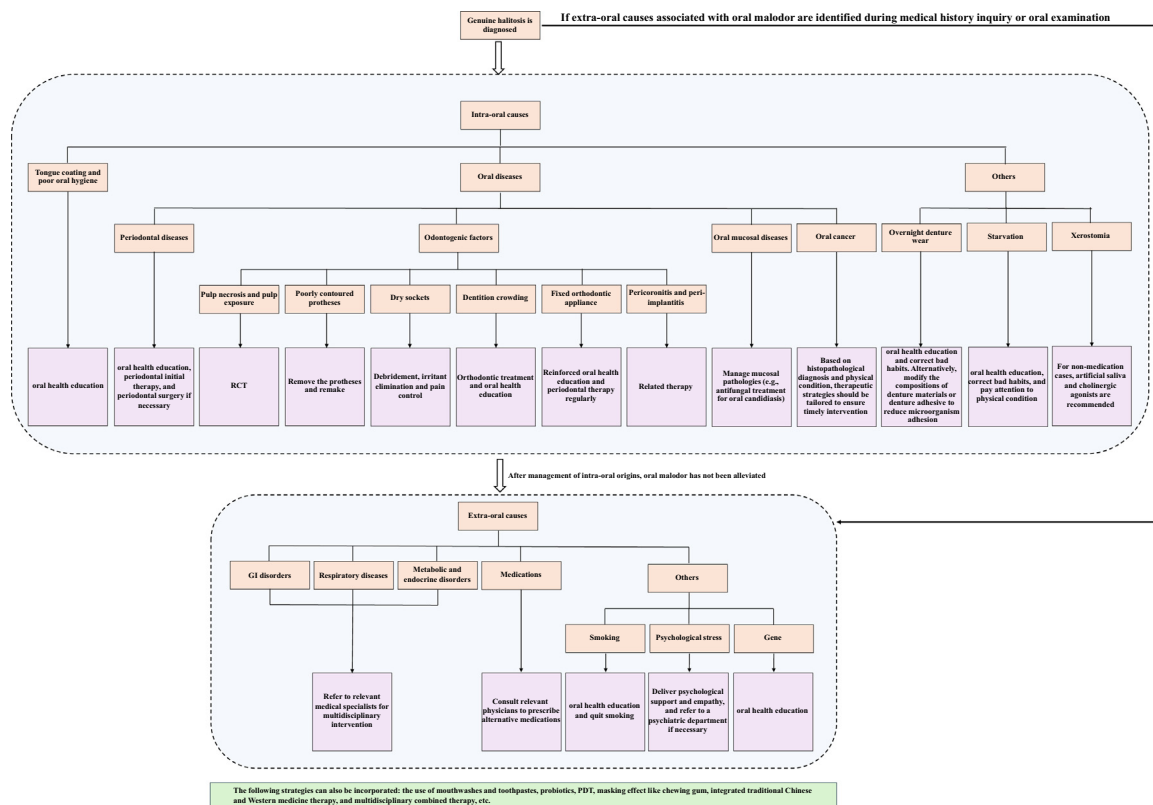


Fig. 2 – Treatment protocol for genuine halitosis. GI, gastrointestinal; PDT, laser therapy and photodynamic therapy; RCT, root canal therapy.

Prophylaxis and therapy of halitosis

Genuine halitosis

Genuine halitosis signals underlying pathology requiring clinical intervention. Key management principles include identifying causative factors precisely, implementing targeted therapy, and using symptomatic treatment as adjunctive support (Figure 2).

Etiological therapies

Oral health education. Patients with poor oral hygiene should receive oral health education to improve their self-care practices, irrespective of the presence of oral diseases. It has been confirmed that a combined regimen of tooth brushing, mouth rinsing, and tongue cleaning proves most effective in reducing halitosis.⁵⁵ Within a certain range (frequency of brushing ≤ 3 times/d), increased frequency of brushing correlates with lower levels of VSCs.⁶² In contrast, mouth rinsing exhibited comparable efficacy to brushing, and the enhancement in halitosis was more substantial with prolonged duration and higher frequency. Furthermore, compared to toothbrushes, tongue scrapers have been proven to be more effective in reducing the levels of VSCs, achieving a decrease in VSCs production by approximately 75%.⁶³ Therefore, to fundamentally reduce the occurrence and progression of halitosis, we propose the following structured oral hygiene maintenance plan. For daily care, patients are advised to brush their teeth at least twice a day and incorporate daily use of dental floss and

a tongue scraper. In terms of patient education, visual materials (such as animated diagrams illustrating the causes of halitosis) should be used to educate patients that malodour commonly originates from the microbial activity in periodontal pockets and on the tongue coating, while also addressing common misconceptions. Step-by-step illustrated brochures should be distributed to guide patients through self-administered oral hygiene routines. Additionally, personalized follow-up plans should be established. For instance, under normal circumstances, maintenance appointments are recommended every 6 to 12 months following initial treatment. By implementing the above measures, patient compliance is expected to improve significantly, thereby effectively reducing the recurrence rate of halitosis.

Periodontal initial therapy. Periodontal diseases are the primary cause of halitosis, and comprehensive periodontal therapy should be provided for the affected patients. Periodontal pockets and subgingival plaque in periodontitis patients harbour substantial anaerobic bacteria, including VSCs-producing species (*P. gingivalis*, *P. intermedia*, and *F. nucleatum*). Periodontal initial therapy comprising oral health education, supragingival scaling, subgingival scaling, and root planning reduces halitosis by decreasing periodontal pathogen load, altering oral microbiota composition, and suppressing VSCs-producing microorganisms. Research demonstrates direct mechanistic links between post-therapy reductions of *F. nucleatum*, *Capnocytophaga gingivalis*, *Campylobacter showae*, and diminished VSCs production, confirmed through subgingival plaque microbiome analysis.⁶⁴ Both quadrant-based SRP

and full-mouth SRP significantly reduce VSCs levels and improve malodour in chronic periodontitis patients.⁶²

Treatment of other oral diseases and conditions. For dental caries, it is essential to remove the decayed tissue and restore the tooth. In cases of pulp necrosis or exposure, root canal therapy is imperative. Meanwhile, poorly contoured prostheses should be replaced with clinically appropriate alternatives. As for dry sockets, the following management strategies should be employed: debridement, irritant elimination, and pain control. Patients with dentition crowding require oral health education and orthodontic intervention when clinically indicated. Additionally, conditions such as pericoronitis and peri-implantitis require appropriate therapy (rinsing, scaling, etc.). In the cases of oral mucosal diseases, tailored treatment to the diagnosis should be provided, such as antifungal treatment for oral candidiasis. In terms of oral cancer, customized strategies (including surgery, radiation therapy, and chemotherapy) should be deployed based on histopathological diagnosis and physical condition for timely intervention.

Multidisciplinary combined therapy. While most halitosis stems from intraoral sources, ineffective local therapy warrants investigation of extraoral causes for definitive diagnosis and targeted management. Related management requires comprehensive history-taking and physical examination during clinical consultation, followed by referral to relevant medical specialists for multidisciplinary intervention to address the underlying aetiology.

Additional adjunctive therapies for halitosis.

- Chemical agent:

Mouthwashes: Mouthwashes containing chlorhexidine, triclosan, cetylpyridinium chloride, or zinc are commonly used to manage halitosis. These agents operate via distinct mechanisms; chlorhexidine, triclosan, and cetylpyridinium chloride exert bactericidal effects that inhibit VSCs production, while zinc neutralizes sulphides through chemical binding.⁶ Among these agents, chlorhexidine demonstrates the highest efficacy in reducing VSCs, establishing it as the gold-standard antimalodour mouthwash. However, it carries risks of tooth staining and transient taste disturbances.⁶³ Zinc-containing mouthwashes, conversely, may impair wound healing postperiodontal surgery due to zinc's cytotoxic properties.⁶⁵

Toothpaste: Clinical trials demonstrate that toothpastes containing stannous fluoride, zinc, or triclosan effectively reduce halitosis. Additionally, formulations with ϵ -poly-L-lysine (ϵ -PL) or functional peptides modulate oral microbiota diversity to inhibit malodour formation.⁶⁶

- For xerostomia-induced halitosis: For medication-induced xerostomia, the physician should be consulted to prescribe alternative medications. For nonmedication cases, artificial saliva substitutes are recommended. Severe cases may require cholinergic agonists (eg, pilocarpine, cevimeline hydrochloride) for symptomatic relief. Additionally,

patients should increase water intake and avoid caffeinated beverages to minimize xerostomia aggravation.⁶

- Probiotics: Probiotics, particularly lactic acid bacteria (eg, *Lactobacillus* and *Bifidobacterium*), reduce halitosis by inhibiting pathogens, maintaining oral-systemic homeostasis, and modulating immune responses.⁶⁷ Notably, *Streptococcus salivarius* K12 – a pioneer colonizer abundant in non-malodour individuals,^{68,69} significantly reduces VSCs through suppressing VSCs-producing bacteria activity to lower measurable VSC levels.^{62,70-72}
- Laser therapy and photodynamic therapy (PDT): Laser therapy utilizes high-energy lasers (eg, erbium-doped yttrium aluminium garnet laser, erbium, chromium: yttrium scandium gallium garnet laser, and neodymium-doped yttrium aluminium garnet laser) to achieve sterilization and tissue cleansing through thermal vaporization, concurrently reducing halitosis. Lopes et al⁷³ found that this therapy significantly reduced VSCs concentration and populations of VSC-producing anaerobic bacteria, with the bacterial reduction demonstrating more immediate and longer-lasting effects post-treatment. Another study reported immediate alleviation of halitosis following laser therapy, with effects persisting throughout a 3-month observation period.⁷⁴ PDT employs specific light wavelengths to activate photosensitizers, generating bactericidal reactive oxygen species via photochemical reactions. For halitosis treatment, a 660 nm wavelength with methylene blue as the photosensitizer is common, though *Bixa orellana* activated at 395 to 480 nm has also proven effective.⁷⁴
- Integrated traditional Chinese and Western medicine therapy: Research indicates that combining Traditional Chinese Medicine (TCM) with Western medicine is significantly more effective than Western medicine alone, demonstrating faster and more potent short-term efficacy.^{73,74} TCM attributes halitosis to dampness, heat, or fire stagnation in the mouth, stomach, liver, and spleen – interconnected imbalances addressed through whole-body systemic regulation. Consistent with this holistic ('Wholism') theory, orally administered TCM requires systemic absorption to exert therapeutic effects. In contrast, periodontal therapy directly reduces oral VSC levels and anaerobic bacteria immediately.⁷⁵ Thus, integrating TCM and Western medicine yields optimal halitosis management outcomes, with complementary application of both approaches recommended for maximum efficacy.
- Modify the compositions of denture materials or denture adhesive: Modifying denture material or adhesive composition may be beneficial in cases of halitosis associated with prolonged denture use. For example, polymethyl methacrylate dentures incorporating silver nanoparticles and graphene demonstrated reduced *Candida albicans* adhesion and significantly decreased malodour.^{76,77} This design leverages silver nanoparticles' potent antimicrobial action and graphene's dual benefits of enhanced mechanical strength and lower water absorption. Alternatively, denture adhesives containing sulphide-binding metal cations (eg, sodium-magnesium-zinc or calcium-zinc copolymers in PVM/MA adhesives) can directly mitigate malodour.⁷⁸
- Natural extracts:

Honey: An experimental study demonstrated honey's significant potential for treating oral diseases, including dental caries, gingivitis, and halitosis.⁷⁹ The mechanism involves diluted honey activating glucose oxidase, generating hydrogen peroxide (H₂O₂) via an enzymatic reaction. This exerts potent antibacterial effects that reduce VSC-producing bacteria populations, thereby alleviating halitosis.

Green tea: *P. gingivalis* degrades sulphur-containing amino acids, producing VSCs that exacerbate halitosis.⁶² Green tea demonstrates efficacy comparable to 0.2% chlorhexidine in reducing plaque accumulation and VSC levels. This effect involves bactericidal catechins that decrease pathogenic bacterial populations and inhibit *P. gingivalis* adhesion to oral epithelium.^{80,81}

Apple polyphenol extract: Apple polyphenol extract combats halitosis by disrupting periodontopathogens (*P. gingivalis*, *P. intermedia*, and *F. nucleatum*). Treatment induces cell membrane irregularities, wall-membrane separation, and bacterial death, thereby reducing populations of VSCs-producing bacteria.⁸²

Phyllanthus emblica fruit extract (PE): PE inhibits growth and aggregation of VSCs-producing bacteria (eg, *P. gingivalis*, *F. nucleatum*) in a dose-dependent manner, with efficacy increasing at higher concentrations.⁸³

Symptomatic therapy. Mint tablets, chewing gum, and breath fresheners are common personal remedies for halitosis, valued for their pleasant scent, social acceptability, convenient access, and ease of use without venue constraints. However, these products primarily mask odours rather than address underlying causes. Their effects are also transient, requiring frequent and prolonged use that can lead to significant ongoing expense.⁶

Delusional halitosis

Delusional halitosis is fundamentally psychological, requiring psychological intervention and support. Clinical management must assess patient sensitivity and concern level, employ empathetic communication (verbal and nonverbal), and demonstrate respect while providing reassurance and support. As dental practitioners typically cannot manage halitophobia alone, psychiatric referral is essential for comprehensive psychological assessment, diagnostic evaluation, and tailored therapy.

Conclusions and expectations

This guideline comprehensively reviews the epidemiology, classification, aetiology, pathogenesis, diagnosis, and treatment of halitosis to establish evidence-based clinical guidelines. Briefly, halitosis is categorized into genuine halitosis and delusional halitosis. The aetiology and pathogenesis can be classified into two primary sources: intraoral causes, primarily tongue coating and periodontal diseases; and extraoral causes, predominantly triggered by gastrointestinal or respiratory disorders. The diagnostic methods employed encompass organoleptic measurement, GC, sulphide

monitor, bitter taste testing, and AI olfaction. The therapeutic interventions include periodontal initial therapy, psychological support and empathy, daily oral hygiene, chemotherapeutic agents, probiotics, PDT, masking effect, psychological intervention, integrated traditional Chinese and western medicine therapy, and multidisciplinary combined therapy. Despite the advent of novel diagnostic and therapeutic technologies in recent years and the gradual enhancement of their efficacy, the clinical management of halitosis still confronts multiple challenges: the intricacy and heterogeneity of etiological mechanisms, the absence of standardization and accessibility of existing diagnostic techniques, the necessity to optimize the precision and sustainability of treatment, along with the disparity between patients' demand for medical treatment and medical resource allocation.

Based on a systematic understanding of the classification and complex aetiology of halitosis, the consensus integrated both widely used clinical diagnostic and therapeutic methods, as well as recent advanced and novel diagnostic approaches (such as bitter taste testing, AI olfaction) and emerging treatment modalities (such as laser therapy, PDT, and natural extracts). By employing holistic thinking, we established precise, interdisciplinary diagnostic and treatment pathways tailored to different etiological sources. This consensus helps address the current lack of systematic and standardized clinical management frameworks for halitosis and provides a meaningful reference for improving diagnostic consistency and optimizing clinical treatment strategies.

Author contributions

Conceptualization and investigation: X.D.Z. and S.H.G. Supervision: X.D.Z. and S.H.G. Original draft: X.D.S. and X.X.C. Review and editing: X.D.S., X.X.C., C.X.L., Z.W., B.C., J.L.S., Y. L., A.M.S., H.M.G., Y.Y., H.R.L., M.D., J.Z., W.Y.K., J.H.L., B.J.M., F.H.Y., Y.P.P., F.M.C., Y.X., Q.X.L., L.Z., Y.S., Z.C.S., Y.L., Z.G.C., J.X., C.J.Z., A.L., J.S., Z.M.W., L.J.L., X.Y.R., S.L.X., D.D.M., Y.F.X., G.X.Z., L.J.Z., J.L., F.C., X.P., X.J.L., P.H.D., J.J.X., K.L., A.L., L.L., B. M.T., Y.C., X.L., Q.F., J.D., C.Q.Y., G.D., H.G.Z., K.Q.P., X.J.W., C. Q.Q., G.H.R., M.L., X.Y.S., S.G., R.Z., L.L., S.L.L., T.T., Q.C., H.W., D.Y.W., C.F.Z., S.H.G., and X.D.Z.

Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this article.

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