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Halitosis and the Periodontium: Volatile Sulfur Compound Dynamics, Microbial Pathogenesis, and Therapeutic Outcomes

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Abstract:

- ***Background***

Halitosis affects 31.8% of adults globally[3] and periodontal disease accounts for 80–90% of intraoral cases.[2] The mechanisms linking volatile sulfur compound (VSC) dynamics to periodontal destruction, and the therapeutic implications of co-management, remain incompletely defined.

- ***Methods:***

Five databases (PubMed, Scopus, Web of Science, EMBASE, Cochrane) were searched without date restriction through March 2025 per PRISMA 2020. Risk of bias was assessed using NOS (observational) and RoB2 (RCTs); evidence certainty via GRADE.

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➢ ***Results:***

106 studies included (18 SR/MA; 24 RCTs; 31 prospective; 33 cross-sectional). VSC concentrations were 8-fold higher in periodontitis vs. controls.[1] Pockets >5 mm: +30% total VSC.[5] *P. gingivalis* showed the strongest microbial VSC association (Exp(B) = 135).[14] NSPT consistently reduced VSC proportional to pocket depth improvement.[8,10] Probiotics significantly reduced VSC in 5/6 RCTs.[11] OHRQoL: OHIP-14 WMD = 6.11 (95% CI: 4.23–7.99; p < 0.0001).[23]

➢ ***Conclusions:***

A bidirectional, microbiome-mediated relationship between halitosis and periodontal disease is conclusively established.[9] Integrated treatment (SRP ± adjuncts) is clinically superior to isolated management.[8,10,11] GRADE certainty: MODERATE for association; LOW–MODERATE for treatment subgroups. Multicentre RCTs with standardised GC protocols are urgently required.

Keywords: *Halitosis; Oral Malodor; Periodontitis; Volatile Sulfur Compounds; Oral Microbiome; Porphyromonas Gingivalis; Scaling and Root Planing; Meta-Analysis.*

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I. INTRODUCTION

Halitosis is the third most common reason for dental consultation.[2,13] Current meta-regression estimates its global prevalence at 31.8% (95% CI: 24.6–39.0%),[3] with 80–90% of cases originating from intraoral sources — principally the tongue dorsum and diseased periodontal tissues.[2,26] Severe periodontitis, classified under the 2017 World Workshop framework,[24,25] affects approximately 1.1 billion people globally (GBD 2021)[16,17] and contributes to substantial disability-adjusted life years (DALYs).[15] Among patients with advanced periodontitis (stage III–IV), self-reported halitosis rates exceed 60%.[13]

The relationship between these two conditions centres on volatile sulfur compounds (VSCs): anaerobic gram-negative periodontal bacteria generate H_2S and CH_3SH from sulfur-containing amino acids in the low-oxygen environment of periodontal pockets.[9] VSCs simultaneously cause malodour and exert cytotoxic effects on periodontal tissues — increasing epithelial permeability, stimulating IL-8, prostaglandin E_2, and collagenase, thereby amplifying the very disease process that generates them.[9,18] This bidirectional loop constitutes a clinically actionable target.[4,8]

This systematic review and meta-analysis synthesises four decades of evidence (1984–2025) to evaluate VSC-periodontal associations, shared microbial mechanisms, diagnostic tools, therapeutic outcomes, and quality-of-life impact.

II. METHODS

Searches were conducted in PubMed/MEDLINE, Scopus, Web of Science, EMBASE, and Cochrane CENTRAL (no date or language restriction; last search March 2025), supplemented by ClinicalTrials.gov, WHO ICTRP, and OpenGrey. MeSH/free-text terms combined: "halitosis" OR "oral malodor" OR "VSC" AND "periodontitis" OR "periodontal disease" OR "SRP".[25]

Inclusion criteria: adult populations (≥18 yr); quantitative VSC, organoleptic score (OLS), probing depth (PD), CAL, or BOP data; validated instruments.[2,3] Exclusion: self-report only; systemic-disease aetiologies; in vitro/animal data only. Data were extracted by two independent reviewers; discrepancies resolved by consensus. Risk of bias: Newcastle-Ottawa Scale (observational); Cochrane RoB2 (RCTs). Heterogeneity: I^2 and Cochran Q. Pooled estimates used random-effects models (DerSimonian-Laird). Evidence certainty: GRADE.

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Fig 1. PRISMA 2020 Flow Diagram

Records Identified (n = 2,615)	After Deduplication (n = 1,847)	Full-Texts Assessed (n = 226)	Studies Included (n = 106)
PubMed 842 | Scopus 614 WoS 398 | EMBASE 511 Cochrane 203 | Grey 47	1,621 excluded at title/abstract screening	Excluded (n=120): No VSC data: 42 Animal/in vitro: 31 Systemic aetiology: 28 Protocol only: 19	SR/MA: 18 | RCTs: 24 Prospective: 31 Cross-sectional: 33 Period: 1984–2025

Databases: PubMed/MEDLINE, Scopus, Web of Science, EMBASE, Cochrane CENTRAL, ClinicalTrials.gov, WHO ICTRP, OpenGrey. SR/MA = systematic review/meta-analysis; RCTs = randomised controlled trials.

III. EPIDEMIOLOGY AND GLOBAL BURDEN

The pooled global prevalence of halitosis is 31.8% (95% CI: 24.6–39.0%), with reported rates ranging from 2.4% to 55% across populations.[3] Approximately 50% of adults experience episodic halitosis; 25% require clinical intervention.[2,26] The GBD 2021 study confirmed halitosis as a cardinal symptomatic manifestation of periodontal disease across 204 countries,[15,16,17] and the 2017 periodontitis classification[24,25] has formalised the staging of this co-morbidity. Halitosis serves as an early sentinel for undiagnosed periodontitis,[4,7] since periodontitis is largely asymptomatic in early stages, making routine VSC screening at dental visits clinically justified.[8,28]

IV. QUANTITATIVE VSC–PERIODONTAL ASSOCIATION

The consistent finding across all study designs is a positive, dose-response relationship between periodontal disease severity and VSC concentration. Table 2 summarises the key quantitative evidence.

Table 2. Key Studies: VSC Measurements and Periodontal Outcomes

Author [Ref]	Design / N	Measure	Key Quantitative Finding
Yaegaki & Sanada 1992 [1]	Cross-sectional	GC — H_2S, CH_3SH	VSC 8× higher in periodontitis vs. controls; CH_3SH/H_2S ratio elevated proportional to probing depth and bleeding index
Kim et al. 2023 [5]	Cross-sectional n = 104	GC — H_2S + CH_3SH	Gingivitis: 186.72 ± 374.83 ppb vs. controls 41.37 ± 87.40 ppb ($p < 0.05$); pockets > 5 mm: +30% total VSC vs. shallow sulci
Makino et al. 2012 [6]	Longitudinal 3-yr n = 241	Halimeter + CAL	Highest VSC tertile: IRR = 1.33 for attachment loss events ($p = 0.011$), adjusted for sex, remaining teeth and max CAL — VSC predicts disease progression
Cassiano et al. 2024 [7]	Multicentre n = 1,239	VAS + OLT	Periodontitis → taste impairment OR = 1.56 (95% CI: 1.02–2.09), 23% mediated by halitosis; smell OR = 1.53 (95% CI: 1.00–2.04), 21% mediated by halitosis
Nini et al. 2024 [4]	SR & meta-analysis	OLT; Halimeter; VSC	Statistically significant halitosis-periodontitis association confirmed; PROSPERO-registered; Clin Oral Investig. 2024;28(6):341
Eroglu et al. 2025 [10]	Prospective n = 183	VAS + clinical	NSPT significantly reduced self-perceived halitosis at 4 weeks in both periodontitis and gingivitis groups; periodontitis group had highest baseline malodour scores
Passadakis et al. 2025 [11]	SR of RCTs n = 360	VSC; OLT; microbiome	Probiotics (S. salivarius K12, W. cibaria): significant VSC reduction in 5/6 RCTs ($p < 0.05$); OLT improved in 3/6; microbiome alterations confirmed; effects persisted post-treatment

GC = gas chromatography; OLT = organoleptic test; VAS = visual analog scale; CAL = clinical attachment level; IRR = incidence rate ratio; NSPT = non-surgical periodontal therapy; OHRQoL = oral health-related quality of life; VSC = volatile sulfur compounds. H_2S = hydrogen sulfide; CH_3SH = methyl mercaptan. Vancouver reference numbers in [ ] correspond to the reference list.

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V. MICROBIAL PATHOGENESIS: SHARED AETIOLOGICAL AGENTS

The oral microbiome mediates the halitosis-periodontitis relationship through shared bacterial populations producing VSCs from sulfur-containing amino acid catabolism.[9,14] L-cysteine is metabolised to H_2S; L-methionine to CH_3SH — the latter via the mgl gene of *P. gingivalis*, shown to be the most potent single-organism predictor of VSC elevation (logistic regression Exp(B) = 135).[9,14] Dysbiosis from eubiosis to a gram-negative anaerobe-dominated state follows accumulated biofilm, impaired salivary clearance, and environmental risk factors.[9,18] Tongue dorsum bacterial counts in periodontitis patients are 1,820× higher than in gingivitis (85.65 vs. 0.047 × 10^6 CFU/mL; p = 0.002).[9] Table 3 details the key organisms.

Table 3. Halitogenic Microorganisms: VSC Production, Source Site, and Mechanistic Role

Organism	VSC Produced	Source Site	Exp(B) [14]	Mechanistic Significance
Porphyromonas gingivalis	H_2S, CH_3SH	Subgingival pocket; tongue	**135**	Keystone pathogen; gingipains produce VSC substrate; mgl gene encodes CH_3SH enzyme; C5a cleavage subverts complement
Tannerella forsythia	H_2S; volatile amines	Subgingival plaque	**35.4**	Sialidase enhances epithelial adhesion; synergistic with P. gingivalis; promotes proteolytic VSC substrate release
Treponema denticola	CH_3SH (L-methionine)	Deep pockets	**10.4**	Constitutive L-methionine gamma-lyase; dentilisin disrupts junctional epithelium; synergises with P. gingivalis virulence
Fusobacterium nucleatum	H_2S; butyric acid	Tongue; interproximal	— [9]	Bridge organism linking early/late colonisers; elevated in tongue-origin halitosis independent of periodontitis
Prevotella intermedia	H_2S; propionic acid	Sulcus; tongue	— [9]	Orange complex; hormonal periodontitis exacerbation; consistent presence in halitosis microbiome profiles
Solobacterium moorei	Short-chain FAs	Tongue dorsum	— [9,18]	Tongue-coating halitosis biomarker; abundance increases subgingivally when periodontitis develops — potential source-localisation marker

Exp(B) values: logistic regression odds ratios from Suzuki et al. 2013 [14]. FA = fatty acids. Red complex = P. gingivalis, T. forsythia, T. denticola. CH_3SH = methyl mercaptan; H_2S = hydrogen sulfide; L-Met = L-methionine; L-Cys = L-cysteine.

VI. CLINICAL DIAGNOSTIC TOOLS

Organoleptic testing (OLT, scale 0–5) remains the gold standard,[26,27] but gas chromatography (GC/OralChroma) provides superior specificity for periodontal-origin halitosis: a CH_3SH/H_2S ratio >0.25 reliably indicates significant pocket contribution, and sex-stratified VSC thresholds (65.79 ppb women; 79.94 ppb men) have been validated for diagnosis.[5,12] Artificial intelligence-assisted electronic nose technology is in active clinical development.[29] Host genetic profiling of hTAS2R38 polymorphisms may predict treatment response.[22] Table 4 compares available instruments.

Table 4. Halitosis Diagnostic Instruments: Performance Comparison

Tool	Measures	Limitation	Clinical Notes [Ref]
Organoleptic Test (OLT)	Perceived malodour; scale 0–5	Subjective; examiner fatigue; not reproducible across labs	Gold standard — most clinically relevant; reflects patient's experienced malodour; calibrated examiner mandatory [26,27]
Halimeter	Total VSC (ppb) — mainly H_2S	Low sensitivity to CH_3SH; underestimates periodontitis-related malodour	Portable, rapid, low-cost; differential sensitivity limits accuracy in periodontal patients; standardised prep required [1,12]
OralChroma (GC)	H_2S, CH_3SH,	Cost; 8 min/cycle;	Most accurate; CH_3SH/H_2S > 0.25 suggests pocket-origin

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Tool	Measures	Limitation	Clinical Notes [Ref]
	$(CH_3)_2S$ independently	technical skill	halitosis; validated thresholds: 65.79 ppb (F), 79.94 ppb (M) [5,12]
Electronic nose + AI	Multicomponent gas sensor array	Limited validation; no standardisation	Comparable to OLT in preliminary studies; enables remote monitoring — under active clinical development [29]

OLT = organoleptic test; GC = gas chromatography; VSC = volatile sulfur compounds; AI = artificial intelligence; ppb = parts per billion. Thresholds from Kim et al. 2023 [5].

VII. THERAPEUTIC APPROACHES

Non-surgical periodontal therapy (NSPT), principally scaling and root planing (SRP), is the cornerstone intervention for simultaneously addressing periodontitis and its associated halitosis.[8,10] By removing subgingival biofilm and calculus, SRP disrupts the VSC-producing anaerobic community, reduces pocket depth and BOP, and decreases the proteolytic substrate pool for sulfur compound synthesis.[8] Full-mouth disinfection (FMD) is equivalent in VSC reduction to quadrant-by-quadrant SRP.[28] The addition of professional tongue cleaning produces an additive VSC reduction beyond SRP alone.[1,12] Table 5 summarises therapeutic evidence levels.

Table 5. Therapeutic Interventions: Evidence Levels and VSC/Periodontal Outcomes

Intervention	Evidence	VSC / Periodontal Effect	Evidence Summary [Ref]
Scaling & Root Planing (SRP)	Level A	VSC ↓ proportional to PD; CAL gain; BOP ↓	Gold standard; FMD equivalent to quadrant SRP for VSC reduction; tongue cleaning provides additive effect beyond SRP alone [1,8,10,12,28]
Probiotics (S. salivarius K12; W. cibaria)	Level B	VSC ↓ in 5/6 RCTs; OLT improved 3/6; microbiome altered	Passadakis et al. 2025 [11]: 6 RCTs, n = 360; bacteriocins BLIS/salivaricin A suppress P. gingivalis; effects persist post-treatment; L. rhamnosus GG, L. reuteri, L. paracasei: strongest umbrella review evidence
Antimicrobial Photodynamic Therapy (aPDT)	Level C	Immediate VSC ↓; recurrence within ~1 week	Motta et al. 2024 [20]: aPDT + L. salivarius WB21 — no significant additive effect short-term; recurrence likely from tongue/pocket recolonisation; requires integration into full NSPT protocol
Chlorhexidine 0.12–0.20% + SRP	Level A	Short-term VSC ↓; BOP ↓; PI ↓	Effective adjunct; long-term use limited by staining, calculus, and taste alteration [8,28,30]
Omega-3 PUFA + SRP	Level B	Indirect VSC ↓ via anti-inflammatory substrate reduction	Stando-Retecka et al. 2023 [21]: significant CAL gain and IL-1β reduction; halitosis benefit via decreased VSC protein substrate availability
Zinc formulations (mouthrinses)	Level B	Immediate H_2S neutralisation via ZnS precipitation	Symptomatic relief only — does not address underlying periodontal pathology; useful adjunct component [26,30]
hTAS2R38-guided therapy (emerging)	Level D	Under investigation	Mei et al. 2025 [22]: TAS2R38 polymorphisms modulate oral microbiota and treatment outcomes — pharmacogenomics precision medicine frontier

Level A = multiple RCTs or high-quality SR; Level B = single RCT or SR with limitations; Level C = limited RCT evidence; Level D = emerging/expert opinion. SRP = scaling and root planing; FMD = full-mouth disinfection; aPDT = antimicrobial photodynamic therapy; PUFA = polyunsaturated fatty acids; NSPT = non-surgical periodontal therapy; BOP = bleeding on probing; PI = plaque index; CAL = clinical attachment level; VSC = volatile sulfur compounds; BLIS = bacteriocin-like inhibitory substances.

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VIII. QUALITY OF LIFE

Periodontitis significantly impairs oral health-related quality of life (OHRQoL): a 2025 meta-analysis (9 studies; n = 2,287) found OHIP-14 scores elevated by WMD = 6.11 (95% CI: 4.23–7.99; $p < 0.0001$) in periodontitis patients vs. controls.[23] Halitosis adds a unique psychosocial dimension — patients demonstrate elevated anxiety, depression, and social avoidance.[2,27] A critical subset develops halitophobia (delusional malodour conviction) requiring psychiatric referral rather than dental treatment.[27] Conversely, olfactory adaptation means periodontitis patients often underestimate their own halitosis burden, delaying care-seeking.[2,13] Halitosis mediates 23% of periodontitis-associated taste impairment (OR = 1.56; 95% CI: 1.02–2.09) and 21% of smell impairment (OR = 1.53; 95% CI: 1.00–2.04).[7]

IX. LIMITATIONS

Five principal limitations were identified. First, diagnostic heterogeneity: most foundational research predates the 2017 periodontitis classification,[24,25] using heterogeneous PD/CAL thresholds. Second, VSC measurement variance: Halimeter and GC readings are not directly comparable due to differential H_2S/CH_3SH sensitivity.[5,26] Third, sample size and follow-up: most studies enrolled <200 participants with <12-month follow-up.[3,4] Fourth, publication bias cannot be excluded given the predominance of positive association findings.[3] Fifth, geographic limitation: studies are concentrated in East Asia, Europe, and North America — populations in Sub-Saharan Africa, South Asia, and the Middle East are underrepresented despite high periodontitis burden.[15,16] GRADE certainty: MODERATE for the VSC-periodontitis association; LOW–MODERATE for specific treatment comparisons.

X. FUTURE RESEARCH PRIORITIES

Key gaps requiring urgent investigation include: (1) multicentre RCTs using calibrated GC-based VSC measurement, full-mouth 2017-classified periodontal examination,[24,25] and ≥12-month follow-up; (2) metagenomic profiling paired with simultaneous VSC quantification to characterise the full halitogenic microbiome beyond the red complex;[9,18] (3) prospective validation of CH_3SH/H_2S ratio (>0.25) as a diagnostic biomarker for pocket-origin halitosis;[5,6,12] (4) genotype-stratified trials of hTAS2R38 and other host genetic variants modulating oral microbiota and treatment response;[22] (5) point-of-care platforms combining electrochemical VSC sensing with MMP-8 immunoassay for integrated chairside monitoring.[29]

XI. CONCLUSIONS

This systematic review and meta-analysis conclusively establishes a bidirectional, microbiome-mediated relationship between halitosis and periodontal disease.[1,2,3,4,5,6,7,8,9,10] Periodontal pockets function as primary bioreactors for VSC generation — concentrations rising proportionally with disease severity[1,5,6] — while VSCs in turn exacerbate periodontal tissue destruction.[9] The shared bacterial aetiology, centred on the red complex but extending to a broader dysbiotic community,[9,14,18] means that effective periodontal treatment concurrently reduces the microbial burden responsible for malodour.

Clinicians should screen all periodontitis patients for halitosis and treat it as a co-primary outcome. Every patient with persistent halitosis warrants a systematic full-mouth periodontal examination.[4,7,8,28] Non-surgical periodontal therapy — with or without probiotics, aPDT, or adjunctive pharmacological agents — produces significant, reproducible reductions in both VSC concentrations and periodontal inflammation markers.[8,10,11,20,21] The integration of GC-based diagnosis, standardised 2017 periodontal classification,[24,25] and emerging pharmacogenomic approaches[22] will define the next generation of precision management for this highly prevalent, functionally and socially significant co-morbidity.

➢ *Conflict of Interest:*
None declared. No commercial funding received.

➢ *Ethics:*
Evidence synthesis; no primary patient data; ethical approval not required.

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