CJDR Caspian Journal of Dental Research
Caspian Journal of Dental Research
e-ISSN: 2322-2395 p-ISSN: 2251-9890
Babol University Of Medical Sciences

Herbal Agents For The Prevention Of Alveolar Osteitis Following Tooth Extraction: A Narrative Review

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Article type
Review Article

Received: 12 Jun 2026
Revised: 2 Aug 2026
Accepted: 12 Oct 2026
Pub. online: 24 Nov 2026

ABSTRACT

Introduction: Alveolar Osteitis (AO) affects up to 30% of surgical third molar cases. Chlorhexidine gluconate carries risks of cytotoxicity, anaphylaxis, and resistance. This review evaluates herbal preventive alternatives.

Materials & Methods: A narrative review was conducted using PubMed/MEDLINE and Google Scholar (January 2015 – May 2026); the search was limited to these two databases, a stated limitation of this review. Twenty-one sources were included: 14 clinical trials, 4 in vivo animal studies, and 3 pharmacological background references covering honey, Aloe vera, propolis, Nigella sativa, and Curcuma longa.

Results: No herbal agent prevented alveolar osteitis in an adequately powered trial. A propolis formulation produced 0% alveolar osteitis versus 23.1% with placebo; significance was not reached. Honey reduced postoperative pain; its effect on incidence was inconclusive. Acemannan improved socket healing in one small trial; prevention efficacy could not be evaluated due to zero events in both groups. Nigella sativa demonstrated treatment efficacy in two independent RCTs. The only in vivo prevention study found no preventive effect.

Conclusion: The evidence base is limited by underpowering, formulation heterogeneity, absent pharmacokinetic data, and frequent conflation of the treatment–prevention distinction. Pharmacological rationale for herbal AO prevention is established, but proven preventive efficacy in adequately powered trials is lacking. In this narrative appraisal, propolis is identified as the most justifiable candidate for a powered prevention trial, and Nigella sativa warrants prevention-focused evaluation. Standardized formulations and socket-specific pharmacokinetic data are prerequisites for clinical translation.

Keywords: Dry Socket; Phytotherapy; Tooth Extraction.

Cite this article: Ahmed Sh. Herbal Agents For The Prevention Of Alveolar Osteitis Following Tooth Extraction: A Narrative Review. Caspian J Dent Res. 2026; 15: 68-90.

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Publisher: Babol University of Medical Sciences

Introduction

Clinical significance of alveolar osteitis

Alveolar Osteitis (AO), colloquially termed dry socket, represents one of the most common and clinically significant postoperative complications in oral and maxillofacial surgery. First described by Crawford in 1896, AO is defined by the onset of severe

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progressive pain within the extraction socket between the first and third postoperative days, accompanied by partial or complete disintegration of the intra-alveolar blood clot, with or without halitosis [1, 2].

The reported incidence varies considerably, ranging from 0.49% following routine extractions to as high as 30% following surgical removal of impacted mandibular third molars [2, 3]. The mandibular posterior region is disproportionately affected due to dense cortical bone, reduced vascularity, and limited blood supply [4]. AO substantially impairs quality of life, necessitates unplanned return visits, and prolongs the overall treatment episode by seven to fourteen days [2].

Pathophysiology and intellectual lineage of the fibrinolysis hypothesis

The fibrinolysis hypothesis — first articulated by Birn in 1972 and 1973 — remains the dominant framework for understanding AO pathogenesis [5, 6]. Birn proposed a dual-pathway model: direct trauma activating tissue plasminogen activators in alveolar bone, and indirect bacterial invasion triggering plasminogen-mediated fibrinolysis. Cardoso et al. (2010) codified this model and added the contribution of vascular insufficiency [7]. Mamoun (2018) further refined it by emphasizing the role of compromised local blood supply in posterior mandibular extractions [4].

This consensus was challenged by a 2019 metagenomic case-control study. Before this, conventional culture-based microbiology had generally identified predominantly commensal oral flora at AO sites, a finding that was used to question the bacterial invasion pathway. Aguilar-Durán et al. [8], using next-generation sequencing, demonstrated a distinct polymicrobial dysbiosis in AO patients — identifying 151 species, of which 55 were found exclusively in AO patients, including Prevotella nanceiensis and Treponema maltophilum. Conventional culture-based approaches detect only a fraction of oral microbial taxa compared to culture-independent sequencing methods [39], and the metagenomic methodology is therefore technically superior in this respect. However, this remains a single preliminary case-control study that has not yet been independently replicated; the dysbiotic pattern it describes should be regarded as a hypothesis-generating observation rather than established pathophysiology, pending confirmatory independent studies.

Limitations of conventional prevention and the CHX lineage

Chlorhexidine gluconate (CHX) became established as the primary evidence-based preventive agent following Taberner-Vallverdu et al.'s 2017 systematic review [9]. Daly et al.'s 2022 Cochrane review — encompassing 49 RCTs and 6,771 participants — quantified this effect: CHX rinse reduces AO odds by 62% (OR 0.38; 95% CI 0.25–0.58) and CHX gel by 58% (OR 0.44; 95% CI 0.27–0.71) versus placebo [10].

However, Ghosh et al.'s 2022 scoping review challenged CHX's status as the uncontested standard, citing documented anaphylaxis risk, cytotoxic effects on oral fibroblasts, and contribution to global antimicrobial resistance [3]. These two reviews are not contradictory: Daly's Cochrane review demonstrates efficacy; Ghosh's scoping review documents safety

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limitations. Together they define the clinical space that herbal alternatives must occupy — agents that match CHX's preventive efficacy without its safety and resistance liabilities.

Antibiotic prophylaxis has been found to lack statistically significant evidence for AO prevention and is increasingly discouraged under antimicrobial stewardship imperatives [10]. Obtundant dressings such as Alvogyl are employed primarily for treatment, not prevention [10].

The case for herbal alternatives

Herbal and plant-derived preparations possess properties intrinsically relevant to AO pathogenesis: broad-spectrum antimicrobial activity against oral anaerobes; anti-inflammatory effects mediated through COX and NF-κB inhibition; antioxidant capacity; and properties that promote wound healing and soft tissue regeneration [11]. Many agents demonstrate favourable biocompatibility, minimal systemic toxicity, and low likelihood of inducing microbial resistance.

Honey generates hydrogen peroxide, maintains low pH, and contains flavonoids with antimicrobial and anti-inflammatory activity [12]. Propolis — rich in caffeic acid phenethyl ester (CAPE) and flavonoids — has potent activity against oral pathogens and regenerative potential in socket healing models [13]. Nigella sativa oil, containing thymoquinone (TQ), has been evaluated in randomized trials for dry socket treatment [14, 15]. Aloe vera (acemannan), Curcuma longa (curcumin), and clove oil have similarly demonstrated relevant pharmacological properties [11]; clove oil is not reviewed as an independent candidate agent in this manuscript — see *Section Mixed herbal agents and general studies* for the rationale.

No review identified in the search conducted for this manuscript specifically addresses herbal prophylaxis for AO prevention. Siriwattanadom et al. (2026) [11] reviewed herbal agents exclusively for AO treatment — not prevention. Daly et al. (2022) [10] addressed general local interventions for AO management without focusing on herbal prophylaxis. Neither review addresses the prevention-specific question that is the focus of the present manuscript.

Aim and objectives

The aim of this narrative literature review was to evaluate evidence on herbal and phytotherapeutic agents for the prevention of alveolar osteitis following tooth extraction. Specific objectives are: [1] to identify and characterise herbal agents with documented or potential preventive efficacy against AO; [2] to compare their preventive effects against conventional agents in terms of AO incidence, pain outcomes, and wound healing; [3] to summarize the pharmacological mechanisms underlying each agent's biological activity relevant to AO prevention; [4] to narratively describe key methodological characteristics and limitations of included studies, including sample size, study design, and outcome reporting; and [5] to identify current evidence gaps and provide directions for future research.

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Search Strategy and Literature Selection

Study design

This study was conducted as a narrative literature review with reference to the Scale for the Assessment of Narrative Review Articles (SANRA) [41] reporting framework. A narrative design was selected given the substantial heterogeneity in herbal agent formulations, concentrations, delivery systems, and outcome measures across available studies, which precluded meaningful quantitative data pooling. To improve reproducibility and transparency beyond what is typical for purely descriptive narrative reviews, structured reporting elements were incorporated — including explicit search strings, defined date limits, stated inclusion and exclusion criteria, and a description of the study selection process. These elements are intended to document the scope and boundaries of the literature surveyed, not to constitute systematic review methodology. No protocol was pre-registered, no dual independent screening was performed, and no formal risk-of-bias tool was applied; the review therefore does not meet the criteria for a systematic review and is not presented as one.

Databases searched

A literature search was conducted across two electronic databases: PubMed/MEDLINE and Google Scholar. PubMed was selected as the primary database for its comprehensive indexing of peer-reviewed biomedical and dental clinical literature. Google Scholar was used as a supplementary source (915 results; the first 200 were screened by relevance ranking). The author acknowledges that restricting the primary search to two databases is a significant limitation. Scopus, EMBASE, Web of Science, and the Cochrane Central Register of Controlled Trials (CENTRAL) were not searched; studies indexed exclusively in these databases without concurrent PubMed coverage may not have been captured. To partially compensate, reference lists of all included articles and related systematic reviews were manually screened for additional eligible publications, and forward citation tracking was performed using Google Scholar “Cited by”.

Search terms

The following MeSH terms and free-text keywords were used in combination for PubMed/MEDLINE: (“alveolar osteitis”[MeSH] OR “alveolar osteitis”[tiab] OR “dry socket”[tiab] OR “fibrinolytic alveolitis”[tiab]) AND (“herbal”[tiab] OR “plant extract”[tiab] OR “phytotherapy”[tiab] OR “natural product”[tiab] OR “honey”[tiab] OR “propolis”[tiab] OR “Nigella sativa”[tiab] OR “black seed”[tiab] OR “thymoquinone”[tiab] OR “Aloe vera”[tiab] OR “acemannan”[tiab] OR “Curcuma longa”[tiab] OR “curcumin”[tiab] OR “turmeric”[tiab] OR “neem”[tiab] OR “green tea”[tiab]) AND (“prevention”[tiab] OR “prophylaxis”[tiab] OR “post-extraction”[tiab] OR “tooth extraction”[tiab] OR “socket healing”[tiab]). An adapted free-text version was used for Google Scholar. Date range: January 2015 – May 2026. Searches were executed in May 2026.

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Inclusion and exclusion criteria

Inclusion criteria: studies published January 2015 – May 2026; RCTs, CCTs, in vivo studies, systematic reviews, narrative reviews; herbal or plant-derived agent used prophylactically or applied at the time of extraction; at least one quantifiable outcome (AO incidence, pain score, or healing index); human studies preferred, with in vitro/in vivo animal studies included for pharmacological characterization; English full-text available. Exclusion criteria: studies published before January 2015; studies evaluating treatment of established AO only (excluded unless providing pharmacological characterization relevant to prevention); studies unrelated to tooth extraction or post-extraction healing; herbal agents used exclusively as tooth storage media, periodontal adjuncts, or in non-extraction contexts; case reports, editorials, conference abstracts, letters to the editor, duplicate publications; retracted publications (two honey studies excluded on confirmation of retraction via PubMed retraction notices). Note: nine references were retained purely as foundational pharmacological/mechanistic background citations, providing biological rationale rather than original evidence addressing the review question: Molan 1992 [12] (honey), Bankova et al. 2000 [13] (propolis), Adams et al. 2009 [16] (manuka honey), Jittapiromsak et al. 2010 [22] (acemannan), Surjushe et al. 2008 [23] (aloe vera), Orsolic 2010 [29] (propolis), Darakhshan et al. 2015 [33] (thymoquinone), Aggarwal et al. 2007 [35] (curcumin), and Dewhirst et al. 2010 [39] (oral microbiome). These nine references are explicitly excluded from the 21-study included evidence base (Table 1) and from the study-selection flow (Figure 1); they are cited only in the "Pharmacological background" subsections of *Section Review of Literature* to explain proposed mechanisms of action, and are not used to draw any conclusion regarding clinical efficacy, prevention, or treatment.

Study selection and exclusion rationale

Literature screening was conducted in two sequential phases. In Phase 1, titles and abstracts of all retrieved records were screened against the inclusion and exclusion criteria independently by the author. In Phase 2, full texts of all potentially eligible studies were retrieved and assessed. Studies were excluded at the full-text stage for the following reasons: focus on treatment of established AO without any prevention component; herbal agent used in a non-extraction dental context; no quantifiable clinical outcome reported; or study design not meeting inclusion criteria. Two publications confirmed as retracted were excluded from the corpus regardless of their findings.

Data extraction

Data were extracted from included studies using a standardized form capturing: first author and year; country; study design; sample size; herbal agent and active component; formulation and concentration; comparator; application method; follow-up duration; primary outcome (AO incidence); secondary outcomes (pain VAS/NRS, healing index, and adverse effects); and key conclusions. For in vivo animal studies, molecular outcome measures were additionally recorded (RANKL, OPG, VEGF, FGF2, collagen, NF-κB expression).

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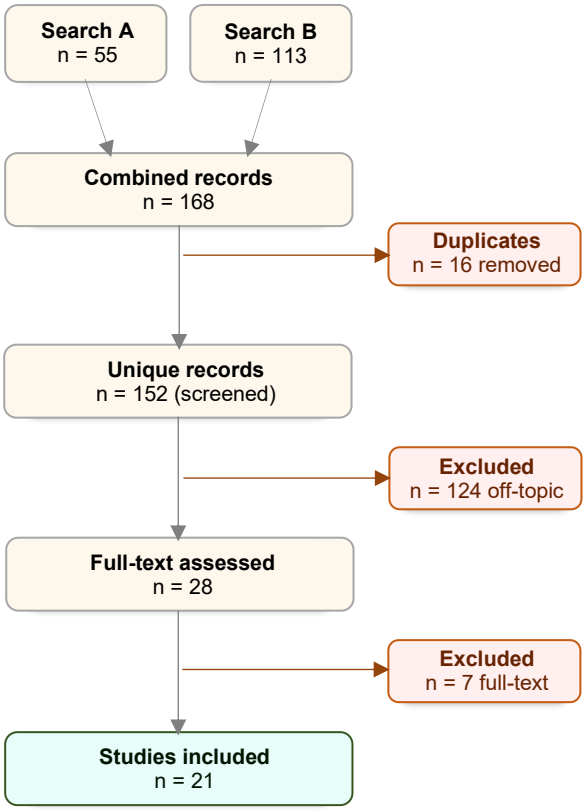


Figure 1. Study selection flow (narrative review — not a PRISMA systematic-review diagram).

Quality assessment

A formal risk of bias assessment using standardized tools was not undertaken, as the objective of this narrative review was evidence synthesis rather than systematic evaluation. Methodological quality was appraised narratively from the full-text reports where available, with key limitations described in *Section Limitations* of the included literature. Without a formal certainty-of-evidence assessment (such as GRADE), the certainty of evidence cannot be graded; however, based on the universal underpowering, formulation heterogeneity, and absent pharmacokinetic data identified in this review, the author's narrative reading of the included studies suggests a weak and inconclusive evidence base for AO prevention across the agents reviewed, though this impression has not been derived through a formal evidence-grading process.

Studies included

Two complementary PubMed/MEDLINE searches were conducted: Search A ("alveolar osteitis"/"dry socket" terms AND herbal/phytotherapeutic terms; n = 55) and Search B ("tooth extraction"/"socket" terms AND herbal/phytotherapeutic terms; n = 113), yielding 168 combined records. After removal of 16 duplicates, 152 unique records were screened at the title/abstract level. Of these, 124 were excluded as topically irrelevant (e.g., studies of unrelated herbal pharmacology, non-dental applications, or plant chemistry unconnected to oral surgery), leaving 28 records for full-text assessment. Seven were excluded at full-text stage for the reasons detailed in *Section Study selection and exclusion rationale*, yielding the

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21 studies included in the primary evidence corpus: 14 randomized or controlled clinical trials, 4 in vivo animal studies, and 3 secondary references included for pharmacological context. A supplementary Google Scholar search (915 results; first 200 screened by relevance) and citation snowballing were used to identify the remaining background pathophysiology and foundational pharmacology references cited elsewhere in this review; these searches did not surface additional primary prevention/treatment studies beyond the 21 identified through the PubMed searches above. The distribution by herbal agent was: honey (6 studies), Aloe vera/acemannan (5 studies), propolis (3 studies), Nigella sativa (3 studies), Curcuma longa/turmeric (2 studies), and mixed/general herbal studies (2 studies). All included studies are presented in Table 1.

Table 1. Data extraction summary — all 21 included studies, with evidence-category classification.

#	Author / Year	Country	Design	N	Agent & Form	Comparator	Key Outcome	AO Finding	Evidence category
Honey (H1–H6)									
H1	de la Puente Dongo et al. 2022	Brazil	SR + meta-analysis	Multiple	Honey (various)	Placebo / no tx	Honey effective for prevention and treatment of AO	Effective (pooled) — methodological limitations noted	Mixed prevention/treatment SR (background)
H2	de Souza et al. 2024	Brazil	SR + meta-analysis	5 RCTs; 330 pts	Intra-alveolar honey	Placebo	Pain: MD −1.05 (day 2); MD −0.97 (day 5); analgesic use lower (MD −4.77)	AO incidence: inconclusive	Prevention
H3	Abu-Mostafa et al. 2019	Saudi Arabia	RCT parallel	100 pts	Natural honey (intra-alveolar)	CHX 0.12% / placebo	Both honey and CHX reduced AO vs placebo; no significant difference between active groups	Reduced vs placebo	Prevention
H4	Soni et al. 2016	India	Pre-post study	50 pts (established AO)	Natural honey dressing	None (pre-post)	Pain reduced day 5 ($p<0.001$); CRP fell 44.1%; granulation tissue by day 7	N/A — treatment study	Treatment-only (background)
H5	Onuoha et al. 2023	Nigeria	RCT	97 pts	Manuka honey (intra-alveolar)	No treatment	Lower complication frequency; earlier socket healing	Reduced complications	Prevention
H6	Famili et al. 2025	Iran	RCT parallel	36 pts	Honey + clove + eugenol + Iris germanica + triamcinolone	Alvogyl	Lower pain at 30 & 60 min ($p<0.001$)	N/A — treatment; combination design	Treatment-only (background)
Aloe vera / Acemannan (AV1–AV5)									
AV1	Ajmera et al. 2024	India	RCT 3-arm	150 pts	Aloe vera mouthwash	CHX 0.12% / saline	AO incidence not significantly different; CHX better for wound dehiscence ($p=0.013$)	Comparable to CHX for AO incidence	Prevention
AV2	Vazirabadi et al. 2025	Iran	DB split-mouth RCT	30 pts	Aloe vera nanoparticle hemostatic sponge	Plain gelatin sponge	Reduced dry socket; lower pain at 48 h and 72 h	Reduced — nanoparticle delivery critical	Prevention

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#	Author / Year	Country	Design	N	Agent & Form	Comparator	Key Outcome	AO Finding	Evidence category
AV3	Srivastava et al. 2024	India	RCT	NR	Aloe vera + bromelain in gelatin sponge	Plain gelatin sponge	Pain reduced days 1, 3, 7 ($p<0.05$); no significant difference in healing index	Not reported	Non-AO endpoint (background)
AV4	Vu et al. 2021	Thailand / Vietnam	RCT 12-month	35 pts / 43 sockets	Acemannan sponge 50 mg	Spontaneous clotting	Greater socket volume reduction at 3, 6, 12 months ($p<0.05$); zero AO all groups	Zero AO — low baseline risk	Prevention
AV5	Zandian et al. 2025	Iran	In vivo (rat)	30 rats	Acemannan extract	Suturing only	Bone resorption 0.76 mm vs 1.09 mm ($p=0.008$)	N/A — animal model	Animal model (mechanistic)
Propolis (P1–P3)									
P1	González-Serrano et al. 2021	Spain	DB split-mouth RCT (pilot)	13 pts / 26 extractions	Propolis 2% + Vit C + Vit E gel; 3×/day ×7 days	Placebo gel	0% AO test vs 23.1% placebo ($p=0.25$, NS); pain VAS lower ($p=0.007$)	0% vs 23.1% — did NOT reach significance ($p=0.25$)	Prevention
P2	Lisbona-González et al. 2021	Spain	Split-mouth study (periodontal disease patients)	60 pts / 120 extractions	Propolis paste 2%	CHX 0.2% / control	90% complete healing day 3 in propolis paste vs 13.4% control	AO not primary endpoint; findings not generalizable beyond periodontal disease patients	Non-AO endpoint (background)
P3	Kresnoadi et al. 2020	Indonesia	In vivo (guinea pig)	56 Cavia cobaya	Propolis + bovine bone graft + PEG	PEG / BBG+PEG	Highest FGF2, VEGF, osteoblast expression in propolis+BBG+PEG group	N/A — animal model	Animal model (mechanistic)
Nigella sativa / Black seed (NS1–NS3)									
NS1	Alabdullah et al. 2023	Syria	RCT	36 pts / 40 sockets	Nigella sativa oil + Gelfoam	Eugenol + Gelfoam	Superior soft tissue healing and reduced inflammation at day 7 ($p<0.05$)	N/A — treatment study	Treatment-only (background)
NS2	Khan et al. 2022	Saudi Arabia	DB RCT 3-arm	60 pts	Nigella sativa powder + oil mixture	Alvogyl / control	Better pain relief than Alvogyl at all timepoints; fewer dressing changes	N/A — treatment study	Treatment-only (background)
NS3	Kresnoadi et al. 2023	Indonesia	In vivo (guinea pig)	56 Cavia cobaya	Nigella sativa + bovine bone graft	NS alone / BBG alone / control	Reduced RANKL and RANKL/OPG ratio; increased OPG at days 7 and 14 ($p<0.05$)	N/A — animal model	Animal model (mechanistic)
Curcuma longa / Turmeric (T1–T2)									
T1	Lone et al. 2018	India	RCT	178 pts	Turmeric paste + mustard oil	ZOE dressing	Faster pain reduction and wound healing vs ZOE ($p<0.05$); no side effects	N/A — treatment study	Treatment-only (background)

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#	Author / Year	Country	Design	N	Agent & Form	Comparator	Key Outcome	AO Finding	Evidence category
T2	Pandya et al. 2023	India	Split-mouth RCT	21 pts	Curcuma longa gel (prophylactic)	Placebo gel	Higher healing scores days 3 & 7 ($p \leq 0.05$); lower pain day 7; zero AO both groups	Zero AO both groups — low baseline risk	Prevention
Mixed / General Herbal (M1–M2)									
M1	Burdurlu et al. 2021	Türkiye	In vivo (rat)	21 rats	Medicinal herbal extract (undefined)	Gelatin sponge / control	No significant AO difference ($p>0.05$); higher osteopontin ($p<0.01$)	AO not prevented — null result	Animal model (prevention-tested)
M2	Siriwattanadom et al. 2026	Thailand	Narrative review	8 clinical articles	Clove, turmeric, aloe vera, black cumin, olive oil	Various	Significant therapeutic properties for AO treatment; standardized protocols lacking	Review of treatment	Treatment-only (background)

AO = alveolar osteitis; VAS = visual analogue scale; RCT = randomized controlled trial; DB = double-blind; CHX = chlorhexidine; ZOE = zinc oxide eugenol; BBG = bovine bone graft; NS = not significant; NR = not reported; N/A = not applicable. Zero AO incidence in Vu 2021 (AV4) and Pandya 2023 (T2) likely reflects low baseline AO risk rather than proven preventive efficacy. Evidence category values: Prevention = study addresses AO prevention outcomes; Treatment-only (background) = evaluates established/treated AO, retained for pharmacological context only; Animal model (mechanistic) = in vivo animal study providing mechanistic evidence; Non-AO endpoint (background) = clinical study whose primary outcome was not AO incidence/prevention; Mixed prevention/treatment SR (background) = systematic review pooling both prevention and treatment data.

Analytical Framework and Outcome Measures

Pharmacological evaluation framework

Each herbal agent was evaluated against three pharmacological domains relevant to AO prevention: [1] antimicrobial activity — spectrum of action against oral anaerobes, facultative organisms, and biofilm-forming pathogens implicated in socket contamination; [2] anti-inflammatory activity — inhibition of pro-inflammatory mediators including COX-1/COX-2, IL-1β, TNF-α, and NF-κB; and [3] wound healing and regenerative activity — promotion of granulation tissue, fibroblast proliferation, angiogenesis, and bone remodeling.

Primary outcome measures

Where reported in included studies, the following primary outcome measures were extracted: incidence of alveolar osteitis — defined using Blum's (2002) diagnostic criteria: post-extraction pain developing days 1–3 with partial or complete clot loss, with or without halitosis; VAS or NRS pain scores at standard postoperative timepoints (days 1, 2, 3, 5, 7); wound healing index — using the Landry-Turnbull-Howley Index or equivalent widely used scoring instrument; and analgesic consumption as a surrogate pain endpoint.

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Secondary and molecular outcome measures

For in vivo animal studies, molecular markers of bone remodeling were extracted where reported: RANKL and OPG expression and RANKL/OPG ratio; VEGF and FGF2 as markers of angiogenesis; type I collagen synthesis; and NF-κB, TLR2, TNF-α expression as markers of inflammatory pathway activation.

Separation of prevention and treatment evidence

Because the stated objective of this review is to evaluate preventive efficacy, included studies were classified by evidence category (prevention, treatment-only, mechanistic/animal, or non-AO endpoint; see Table 1) at the point of data extraction. Within *Section Review of Literature*, evidence for each agent is presented in clearly separated prevention and treatment subsections. Treatment-only studies are retained exclusively for pharmacological and mechanistic context; they are not pooled with, weighted alongside, or used to support any conclusion regarding preventive efficacy in *Sections Discussion*, *Future Directions*, or *Conclusion*.

Review of Literature

Twenty-one studies were included (14 RCTs/CCTs, 4 in vivo animal studies, 3 secondary references included for pharmacological context). Studies are presented by herbal agent. Of the 41 references cited in this manuscript, 21 constitute the primary included evidence base (Table 1); 9 are foundational pharmacological background citations excluded from that evidence base *(Section Inclusion and exclusion criteria)*; and the remainder comprise general etiology/pathophysiology literature and methodological citations (including the SANRA framework [41]). Consistent with the narrative design of this review *(Section Study design)*, the appraisals of relative evidence strength presented in this and the following section reflect the author's qualitative synthesis of the literature, not a formal evidence grading, ranking, or certainty-of-evidence system such as GRADE.

Honey

Pharmacological background (mechanistic context — not part of the included evidence base)

Honey's biological activity derives from multiple simultaneous mechanisms: high osmolarity; hydrogen peroxide generation through glucose oxidase activity; low pH (3.2–4.5); and flavonoids including quercetin and caffeic acid with anti-inflammatory and antioxidant activity [12]. Manuka honey additionally contains methylglyoxal (MGO), with potent non-peroxide antimicrobial activity particularly effective against Gram-positive oral organisms [16].

Prevention-relevant evidence

Abu-Mostafa et al. (2019) [19] found honey comparable to CHX 0.12% in reducing AO incidence versus placebo in a parallel-arm RCT. A 2023 RCT of Manuka honey found lower complication frequency and earlier healing versus no treatment [20]. de Souza et al. (2024) [18]

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conducted a rigorous meta-analysis of five RCTs (330 participants) using the Cochrane Risk of Bias tool version 2 and the GRADE framework, finding honey significantly reduced pain at day 2 (MD: −1.05; 95% CI −2 to −0.1) and day 5 (MD: −0.97; 95% CI −1.97 to −0.03) and analgesic consumption (MD: −4.77; 95% CI −6.73 to −2.81); critically, the effect on AO incidence itself was inconclusive. In this narrative appraisal, the 2024 review is judged in this narrative appraisal, the 2024 review is judged methodologically superior to the pooled analysis below and is treated as the more reliable evidence source on this point.

In summary: honey demonstrates consistent pain reduction benefit. Its preventive effect on AO incidence is statistically inconclusive per the best available meta-analytic evidence.

Treatment evidence (background only)

de la Puente Dongo et al. (2022) [17] concluded honey is effective for both prevention and treatment of AO; however, this review pooled prevention and treatment studies as equivalent outcomes and used less rigorous bias assessment, and is therefore not relied upon for the prevention-specific conclusions of this review. Famili et al. (2025) [21] used honey as one of five components in a novel dressing tested against established dry socket — outcomes cannot be attributed to honey specifically and this study is retained for background only.

Aloe vera (acemannan)

Pharmacological background (mechanistic context — not part of the included evidence base)

The principal bioactive compound of Aloe vera is acemannan — a high-molecular-weight acetylated mannose polysaccharide that stimulates macrophage activation, promotes fibroblast proliferation and collagen synthesis, induces alkaline phosphatase activity and mineralization in dental pulp cells, and has demonstrated osteoinductive properties in vitro and in vivo [22]. Additional Aloe vera compounds — anthraquinones, saponins, vitamins C and E — contribute antimicrobial and anti-inflammatory activity [23].

Prevention-relevant evidence

Ajmera et al. (2024) [24] conducted the largest aloe vera study in the corpus (n=150, three arms) comparing aloe vera mouthwash, CHX 0.12%, and normal saline. AO incidence did not significantly differ across groups; CHX outperformed aloe vera for wound dehiscence (p=0.013) and pain at day 7. Vazirabadi et al. (2025) [25] tested aloe vera as a nanoparticle-embedded hemostatic sponge (n=30, split-mouth) and found reduced dry socket occurrence and lower pain at 48 and 72 hours versus plain gelatin sponge. These two studies are not contradictory — they demonstrate that the delivery system is a primary determinant of aloe vera's clinical efficacy. Vu et al. (2021) [27] demonstrated in a rigorous 12-month RCT (n=35 patients; 43 sockets) that a 50 mg acemannan sponge produced significantly greater socket volume reduction at 3, 6, and 12 months versus spontaneous clotting ($p<0.05$), with zero AO cases in all groups. This zero-event outcome renders the study incapable of evaluating AO prevention: if baseline AO incidence is 1–2% in general extraction patients, a sample of this

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size cannot meaningfully detect a preventive effect, regardless of the intervention; the zero AO finding should not be interpreted as evidence of prevention.

In this narrative appraisal, aloe vera evidence appears strongest for nanoparticle-delivered intra-alveolar acemannan; mouthwash delivery appears pharmacologically insufficient for meaningful socket tissue contact.

Treatment / non-AO-endpoint evidence (background only)

Srivastava et al. (2024) [26] combined aloe vera with bromelain in a gelatin sponge, finding pain reduction ($p<0.05$) but no significant improvement in healing index; AO incidence was not reported, so this study is retained for background only and not used to draw prevention conclusions.

Mechanistic/animal evidence

Zandian et al. (2025) [28] confirmed acemannan significantly reduced marginal bone resorption in rat sockets (0.76 vs 1.09 mm; $p=0.008$).

Propolis

Pharmacological background (mechanistic context — not part of the included evidence base)

Propolis is a resinous bee product whose activity is primarily attributed to flavonoids and caffeic acid phenethyl ester (CAPE), which demonstrate broad-spectrum antimicrobial activity against oral pathogens including Streptococcus mutans, Porphyromonas gingivalis, and Fusobacterium nucleatum [13]. CAPE inhibits NF-κB activation, suppressing the inflammatory cascade at the transcriptional level [29]. Propolis also stimulates VEGF and FGF2 expression in bone healing models [30].

Prevention-relevant evidence

González-Serrano et al. (2021) [31] is the most directly relevant prevention study in this entire corpus. This double-blind split-mouth pilot RCT (n=13; 26 extractions) randomized patients to a compound gel containing 2% propolis extract, nanovitamin C, and nanovitamin E — applied three times daily for seven days — versus placebo. An important limitation is that the intervention was a multi-component formulation; the 0% AO outcome cannot be attributed to propolis in isolation, as the independent contributions of nanovitamin C and nanovitamin E cannot be disaggregated from these data. Zero AO cases were recorded in the test group versus 23.1% in placebo; this difference did not reach statistical significance ($p=0.25$). Pain VAS was significantly lower in the test group ($p=0.007$), and no adverse effects occurred. This result is best interpreted as a clinically interesting preliminary signal rather than evidence of proven preventive efficacy — with only 13 patients, the study was substantially underpowered for its primary endpoint.

In this narrative appraisal, propolis appears to hold the strongest direct prevention signal among all agents reviewed, based on the González-Serrano et al. pilot data alone; an adequately powered prevention RCT is identified as the most pressing research need in this

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field. This ranking reflects the author's appraisal of the pilot signal; acemannan nanoparticles and Nigella sativa are alternative candidates whose prioritization could equally be defended on pharmacological grounds.

Treatment / non-AO-endpoint evidence (background only)

Lisbona-González et al. (2021) [32] found 90% complete socket healing at day 3 in the propolis paste group versus 13.4% in controls across 120 extractions; this study was conducted exclusively in periodontal disease patients, AO was not the primary endpoint, and these findings should not be generalized to routine extraction populations without further evidence.

Mechanistic/animal evidence

Kresnoadi et al. (2020) [30] demonstrated in a guinea pig model that propolis combined with bovine bone graft produced the highest FGF2, VEGF, and osteoblast expression at days 3 and 7 ($p<0.001$); this study used propolis only in combination with bone graft, so molecular outcomes cannot be attributed to propolis alone.

Nigella sativa (black seed)

Pharmacological background (mechanistic context — not part of the included evidence base)

Nigella sativa's principal bioactive, thymoquinone (TQ), has characterized antimicrobial activity against a broad spectrum of oral pathogens, anti-inflammatory effects through COX-2 inhibition and NF-κB suppression, analgesic properties, and stimulation of bone formation through modulation of the RANKL/OPG axis [33].

Prevention-relevant evidence

No study in this corpus evaluated Nigella sativa in a prevention design; all available clinical evidence for this agent is treatment-only (see below).

Treatment evidence (background only)

Alabdullah et al. (2023) [14] compared Nigella sativa oil versus eugenol for dry socket treatment (n=40 sockets), finding statistically superior healing and inflammation reduction at day 7 ($p<0.05$). Khan et al. (2022) [15] found Nigella sativa powder and oil mixture provided significantly better pain relief than Alvogyl at all measured time points, with fewer dressing changes required. Both are treatment studies. In this narrative appraisal, Nigella sativa appears to have the most consistent treatment evidence in this corpus, with two independent RCTs from different countries demonstrating superior pain relief and soft tissue healing — however, both studies evaluated established dry socket, and neither has a prevention arm. Nigella sativa warrants evaluation in a future prevention-focused RCT, with propolis taking priority given its existing pilot prevention data.

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Mechanistic/animal evidence

Kresnoadi et al. (2023) [34] demonstrated in guinea pig models that Nigella sativa combined with bovine bone graft reduced RANKL/OPG ratio and increased OPG at days 7 and 14 ($p<0.05$).

Curcuma longa (turmeric)

Pharmacological background (mechanistic context — not part of the included evidence base)

Curcumin exerts anti-inflammatory activity through COX-2, LOX, and NF-κB pathway inhibition, demonstrates broad-spectrum antimicrobial activity against oral pathogens including Streptococcus mutans and Candida species, and promotes wound healing through TGF-β stimulation and collagen synthesis [35].

Prevention-relevant evidence

Pandya et al. (2023) [37] applied Curcuma longa gel prophylactically in 21 patients (split-mouth), finding higher healing scores at days 3 and 7 ($p\leq0.05$) and lower pain at day 7. Zero dry socket cases were observed in both groups. With only 21 participants and no AO events in either arm, this study was incapable of evaluating AO prevention due to insufficient event rates; it cannot contribute meaningfully to conclusions about preventive efficacy regardless of the direction of its findings on secondary outcomes.

Treatment evidence (background only)

Lone et al. (2018) [36] evaluated turmeric paste with mustard oil in 178 patients with established dry socket versus ZOE — the largest single-agent herbal RCT in the corpus. Turmeric produced faster pain reduction and wound healing ($p<0.05$) with no side effects.

Mixed herbal agents and general studies

Prevention-relevant / mechanistic evidence

Burdurlu et al. (2021) [38] tested an undefined medicinal herbal extract (MHE) in a rat AO model (n=21). AO signs were observed in all groups with no statistically significant difference ($p>0.05$); the MHE showed higher osteopontin expression ($p<0.01$). This is the only in vivo study in the corpus that explicitly tested AO prevention with a herbal agent and found no preventive effect; this null result is noted and should be considered alongside positive clinical signals when interpreting the evidence base.

Treatment evidence (background only)

Siriwattanadom et al. (2026) [11] reviewed 8 clinical articles on clove, turmeric, aloe vera, black cumin, and olive oil for AO treatment, confirming anti-inflammatory, antibacterial, and wound-healing properties while explicitly calling for standardized protocols.

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Discussion

Overview of findings

This review identified 21 included studies evaluating herbal agents for AO prevention and socket healing. The central finding is that no herbal agent has been proven to prevent alveolar osteitis in an adequately powered randomized controlled trial. This is a finding of insufficient evidence, not inefficacy — the distinction matters considerably for clinical practice and future research design.

Strength of prevention evidence by agent

Propolis shows the most direct prevention signal in this narrative appraisal: González-Serrano et al. [31] reported zero AO versus 23.1% in placebo. The primary outcome did not reach statistical significance (p=0.25; n=13). This result is appropriately interpreted as a preliminary signal requiring confirmation in an adequately powered trial. A further limitation is that the intervention was a compound formulation (propolis extract plus nanovitamin C plus nanovitamin E); the 0% AO finding cannot be attributed exclusively to propolis, and a future prevention RCT should test a propolis-only formulation to isolate its independent contribution.

Aloe vera as a nanoparticle hemostatic sponge (Vazirabadi et al., 2025 [25]) reduced dry socket occurrence. However, Ajmera et al. (2024 [24]) — the largest aloe vera study, n=150 — found mouthwash delivery comparable to CHX for AO incidence but inferior on secondary wound outcomes. The contrast is a delivery system variable, not a pharmacological contradiction.

Honey's preventive efficacy remains the most contested finding in the corpus. de la Puente Dongo (2022) [17] concludes effectiveness; de Souza (2024) [18] concludes inconclusiveness for AO incidence. In this narrative appraisal, the 2024 review is judged methodologically superior, and its conclusion in the author's narrative assessment should be treated as the current best evidence position.

The only in vivo study in this corpus that directly tested AO prevention with a herbal agent and found no effect — Burdurlu et al. (2021) [38] — should be given proportionate weight in any interpretation of this evidence base. The possibility of publication bias favouring positive results cannot be excluded, and this should be borne in mind when assessing the overall direction of the literature.

The treatment-prevention conflation problem

As detailed in *Section Separation of prevention and treatment evidence* and Table 1, prevention and treatment evidence were classified and presented separately throughout this review to prevent this conflation from affecting the conclusions drawn. The majority of included studies evaluated herbal agents in the treatment of established AO, not its prevention. Thirteen of 21 studies enrolled patients after AO had developed. Extrapolating treatment efficacy to prevention is pharmacologically plausible but clinically unvalidated. The conditions under which a herbal agent must work prophylactically — clot stabilisation,

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microbial contamination prevention, fibrinolysis modulation before AO develops — are categorically different from treating an already established, exposed, infected socket.

Contradictions and methodological tensions

The most theoretically significant tension in this evidence base concerns the microbial etiology of AO. Prior culture-based microbiological assessments had identified predominantly commensal oral flora at AO sites, casting doubt on the bacterial invasion pathway. This was directly challenged by a metagenomic case-control study: Aguilar-Durán et al. (2019) [8], using next-generation sequencing, demonstrated a distinct polymicrobial dysbiosis in AO patients — identifying 151 species in AO patients, 55 of which were exclusive to the AO group, including Prevotella nanceiensis and Treponema maltophilum. The metagenomic methodology is technically superior to conventional culture-based approaches in its breadth of microbial detection [39]; however, this is a single preliminary case-control study and replication is lacking. The dysbiotic pattern it describes is therefore best treated as a hypothesis-generating finding. If the dysbiosis hypothesis is confirmed by future studies, antimicrobial herbal agents may target the right pathway. However, if fibrinolytic activity dominates over bacterial contamination, the antimicrobial selection rationale underpinning the majority of herbal agents in this corpus may be targeting a secondary rather than primary causal pathway. This remains unresolved and represents a fundamental uncertainty that limits interpretation of the entire evidence base.

The concurrent 2022 publications by Daly et al. [10] (CHX rinse reduces AO odds by 62% and CHX gel by 58%) and Ghosh et al. [3] (CHX carries anaphylaxis risk and resistance concerns) are not contradictory — they address efficacy and safety respectively and are both simultaneously correct. Their coexistence defines the clinical gap that herbal alternatives must fill.

The standardization problem

Every review in this corpus identifies non-standardized formulations as the primary obstacle. Across 14 clinical RCTs, honey was applied as natural honey, manuka honey, and intra-alveolar dressing at unspecified concentrations. Propolis ranged from 2% gel to 2% paste to 2% mouthwash — three vehicles delivering very different local concentrations. Without pharmaceutical-grade standardized formulations, results cannot be pooled, compared, or translated into clinical guidelines.

Untested assumptions underlying the evidence base

The following assumptions are shared by the majority of included studies but are never explicitly tested. First, all studies assume that the absence of reported adverse events constitutes safety evidence. No study used a validated adverse event reporting scale as a primary endpoint; none screened systematically for hypersensitivity to bee-derived products or plant-derived compounds; only one study followed patients beyond four weeks [27].

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Second, all studies attribute observed clinical outcomes to the named herbal agent without isolating active compounds. If honey's benefit derives from osmotic pressure rather than flavonoids, any hyperosmotic agent would work equivalently.

Third, thirteen of the 21 studies were conducted exclusively in mandibular third molar extraction patients. All draw conclusions about tooth extraction broadly. Third molars carry 30% baseline AO risk versus 0.5–3% for routine extractions. An intervention validated in third molar patients cannot be assumed to confer equivalent benefit in general dental extraction settings without direct evidence.

Fourth, not all studies stated the diagnostic criteria used for AO, creating classification heterogeneity that makes cross-study incidence comparisons unreliable.

Fifth, no study measured local herbal compound concentration in socket tissue. Bioavailability is universally assumed. A mouthwash contacting the socket for 30 seconds is not delivering a pharmacologically active dose to socket tissue.

Methodological critique

RCTs dominate this corpus at 50%, but split-mouth designs, used by seven RCTs, halve required sample size while introducing carry-over risk — systemic absorption or contralateral spread of herbal agents — that none of the included studies formally assessed.

More significantly, two categories of study are completely absent from this corpus: pharmacokinetic studies and microbiome-profiling studies. No study has measured local herbal compound concentration in human socket tissue at any time point after topical application. No study has used metagenomic sequencing to evaluate whether any herbal agent corrects the microbial dysbiosis associated with AO.

Comparison with conventional agents

Against the Cochrane evidence base for CHX — OR 0.38–0.44 at moderate certainty from 49 RCTs and 6,771 participants [10] — no herbal agent in this corpus provides comparable evidence quality. The best herbal evidence is drawn from underpowered single-centre pilots with narrative appraisal consistently indicating a weak and inconclusive evidence base. This asymmetry reflects not pharmacological inferiority but research investment asymmetry: the CHX evidence base was built over two decades; the herbal evidence base consists predominantly of single-centre pilots.

Limitations

Limitations of the included literature

Thirteen of 21 studies enrolled fewer than 50 patients per arm — insufficient to detect a statistically significant difference in a low-prevalence outcome such as AO incidence. No two studies used identical herbal agent preparations, concentrations, or delivery vehicles, precluding quantitative synthesis. Not all studies explicitly stated the diagnostic criteria used for AO. The majority of included studies evaluated herbal agents as treatments for established

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AO. No study measured local herbal compound concentration in socket tissue. Most studies followed patients for seven to fourteen days only.

Limitations of this review

As a narrative review, this study is subject to selection bias compared to a pre-registered systematic review with dual independent screening. The literature search was restricted to PubMed/MEDLINE as the primary structured database, with Google Scholar used supplementarily. Scopus, EMBASE, Web of Science, and CENTRAL were not searched. Quantitative meta-analysis was not performed. Two publications confirmed as retracted were excluded; both evaluated honey as a post-extraction intervention, and their exclusion does not materially alter the conclusions of this review, as the remaining honey evidence base (six studies including two meta-analyses) is independently sufficient to support the findings reported. Grey literature and unpublished trials were not systematically sought, introducing potential publication bias toward positive findings. A further structural limitation inherent to narrative review methodology is that evidence is not formally weighted by sample size, risk of bias, or study design quality: within the narrative structure, a 13-patient pilot study and a 330-patient meta-analysis can appear to contribute equally to a conclusion, even though they should not. Evidence should therefore be interpreted with consideration of study size and methodological rigour, with larger, well-designed studies given proportionately greater weight.

Future Directions

Adequately powered multicentre prevention RCTs

An adequately powered multicentre double-blind RCT testing AO prevention with propolis gel (2% standardized extract) is identified as the highest priority, based on the pilot prevention data of González-Serrano et al. [31]. Formal sample size calculation should be undertaken during trial design using locally applicable baseline AO incidence estimates, a pre-specified minimum clinically important difference, and standard power parameters. Nigella sativa oil is identified as a strong second candidate based on its consistent treatment RCT evidence. Pre-registration on ClinicalTrials.gov or ISRCTN is mandatory.

Standardized pharmaceutical-grade herbal formulations

Before clinical trials can be compared, standardized preparations must be developed: active compound content quantified by high-performance liquid chromatography (HPLC); extraction method, solvent, and concentration specified; stability testing conducted; and delivery vehicle specified.

Pharmacokinetic studies in extraction sockets

Socket pharmacokinetic studies are needed to establish local concentration of each herbal bioactive in socket tissue at 24, 48, and 72 hours post-application; the minimum inhibitory

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concentration for AO-relevant oral pathogens in the socket environment; and the optimal delivery vehicle for sustained socket contact.

Microbiome-guided precision prevention

Should the polymicrobial dysbiosis identified by Aguilar-Durán et al. [8] be confirmed by independent replication, a logical next step would be to investigate whether pre-extraction oral microbiome profiling can identify patients at elevated AO risk and enable targeted herbal antimicrobial intervention — transforming population-level prophylaxis into a precision medicine approach. This research direction is contingent on replication of the Aguilar-Durán findings and is not justified by the current single preliminary case-control study alone.

Formal pharmacovigilance and safety assessment

A dedicated pharmacovigilance study for topically applied herbal agents in oral surgery is needed: systematic pre-operative screening for bee-product allergy (propolis, honey), plant latex allergy (aloe vera), and drug interactions (thymoquinone with anticoagulants); adverse event reporting using Common Terminology Criteria for Adverse Events version 5.0 (CTCAE v5.0) as primary endpoint; minimum 90-day follow-up; and sample sizes sufficient to detect adverse events with 2% incidence at 80% power.

Conclusion

This narrative literature review evaluated 21 included studies examining herbal and phytotherapeutic agents for the prevention of alveolar osteitis following tooth extraction, encompassing randomized controlled trials, in vivo animal studies, and systematic reviews published between 2015 and 2026.

Several conclusions can be drawn from the evidence reviewed. The pharmacological rationale for herbal agents in AO prevention is well established: each agent possesses characterized antimicrobial, anti-inflammatory, or wound-healing properties directly relevant to AO pathogenesis. No serious adverse events were reported in the included studies; however, absence of reporting should not be equated with absence of adverse events, and formal safety evidence remains sparse — systematic pharmacovigilance data for topically applied herbal agents in oral surgery are currently lacking. In the treatment of established dry socket, Nigella sativa, honey, and turmeric demonstrate efficacy comparable to or superior to eugenol-based dressings in multiple independent RCTs.

What cannot be concluded is equally important: no herbal agent has been proven to prevent alveolar osteitis in an adequately powered human randomized controlled trial. The closest available evidence — zero AO versus 23.1% with propolis gel — did not reach statistical significance ($p=0.25$; $n=13$). Honey's preventive effect on AO incidence is inconclusive per the best available meta-analysis. Every prevention-focused study in this corpus was underpowered for its primary endpoint.

The central limitation of this field is not pharmacological plausibility — it is the absence of adequately powered, standardized, pre-registered multicentre prevention trials. Based on the author's appraisal of the available pilot data, propolis is considered the most justifiable

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candidate for a powered prevention RCT; Nigella sativa is considered a strong second candidate on the basis of consistent treatment-phase evidence. These represent the author's research prioritization judgement, not an evidence-derived efficacy ranking, and alternative prioritizations are defensible.

Conflict of interest

The author declares no conflict of interest.

Funding

This research did not receive any specific funding.

Ethical approval

Ethical approval was not required. This study is a review of previously published data. No patient data were directly collected or analyzed.

Informed consent

This article does not contain any studies involving human subjects.

Authors' contributions

Conceptualization, Methodology, Investigation, Literature search, Data acquisition, Manuscript preparation, Writing — original draft, Writing — review and editing. The author accepts responsibility as guarantor for the integrity of this work as a whole, from inception to published article.

AI Disclosure

An artificial intelligence language model was used for language editing and grammatical refinement during the preparation of this manuscript. The author takes full responsibility for the scientific content, accuracy of all statements, integrity of the data synthesis, and conclusions presented. AI assistance was limited to language and did not contribute to study design, data extraction, interpretation, or any intellectual component of this work.

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