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Mapping Mechanistic and Biological Evidence of *Ganoderma lucidum*-derived Bioactives in Periodontal Inflammation: A Scoping Review

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ABSTRACT

Objective: Periodontal inflammation is a host-mediated condition characterized by dysregulated immune and oxidative responses, prompting increasing interest in natural bioactives compounds with potential host-modulatory properties. *Ganoderma lucidum* has been widely studied for its anti-inflammatory and antioxidant activities; however, evidence relating specifically to periodontal inflammation remains limited and fragmented. This study aimed to conduct a scoping review of the available experimental literature on the biological and mechanistic roles of *Ganoderma lucidum*-derived bioactives in periodontal inflammation. **Material and methods:** Electronic searches were conducted in PubMed, Scopus, and Web of Science to identify English-language studies published between 2020 and 2025. Original preclinical experimental studies evaluating *Ganoderma lucidum* or its bioactives fractions in periodontal or gingival inflammation models were included. Data were descriptively summarized to outline the experimental models used, types of extracts, dosages applied, inflammatory and oxidative outcomes assessed, and the molecular pathways reported. **Results:** Four studies met the inclusion criteria. Three used *in vivo* rodent models, while one combined inflamed human gingival fibroblasts in vitro with a murine ligature-induced periodontitis model. Investigated preparations included polysaccharide extracts, ethanolic extracts, spore-oil fractions, and crude extracts. Across studies,

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administration was associated with reduced inflammatory mediators, including tumour necrosis factor-α, interleukin-1β, prostaglandin E2, matrix metalloproteinase-1, and interleukin-8, while one study reported increased interleukin-10. Some studies also reported reduced oxidative stress markers and lower alveolar bone loss. Mechanistic evidence was limited; one study suggested JNK-related signalling involvement, whereas NF-κB-related mechanisms were discussed more indirectly. **Conclusions:** The available evidence suggests that *G. lucidum*-derived preparations may be associated with anti-inflammatory, antioxidative, and tissue-protective effects in experimental models of periodontal inflammation. However, the evidence base remains limited, heterogeneous, and restricted to short-term preclinical studies. Further research incorporating standardized extract characterization, mechanistic validation, and human clinical evaluation is needed to clarify the translational relevance of these findings.

1. INTRODUCTION

Periodontitis is a chronic, multifactorial inflammatory condition marked by the gradual destruction of the tooth-supporting tissues, including the gingiva, periodontal ligament, cementum, and alveolar bone (Pihlstrom et al., 2005; Kinane et al., 2017). While the disease is initially triggered by a dysbiotic subgingival biofilm, its progression is driven predominantly by an exaggerated and poorly regulated host immune response, which sustains inflammation and accelerates tissue breakdown. Increased levels of pro-inflammatory mediators—such as tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6)—along with matrix metalloproteinases (MMPs), play a central role in extracellular matrix degradation and alveolar bone resorption (Hajishengallis & Chavakis, 2021; Könönen et al., 2019).

Oxidative stress represents an important biological link between microbial challenge and host-mediated tissue damage in periodontitis (Baltacioglu et al., 2014; Chapple & Matthews, 2007). Excessive generation of reactive oxygen species (ROS) during inflammation can disrupt redox balance, damage cellular macromolecules, and further amplify inflammatory signalling. Among the pathways frequently discussed in this context are nuclear factor kappa B (NF-κB), which regulates the expression of pro-inflammatory genes, and nuclear factor erythroid 2-related factor 2 (Nrf2), which contributes to antioxidant and cytoprotective responses through downstream targets such as heme oxygenase-1 (HO-1) (Suzuki & Yamamoto, 2015). Together, dysregulated inflammatory and oxidative pathways are thought to contribute to the persistence and progression of periodontal inflammation.

In this context, host-modulation therapy has emerged as an important adjunct to mechanical periodontal treatment (Preshaw, 2018). Pharmacologic agents such as subantimicrobial-dose doxycycline and nonsteroidal anti-inflammatory drugs can partially attenuate inflammatory mediator activity; however, their long-term use is limited by adverse effects and narrow molecular specificity. These limitations have stimulated interest in natural bioactives compounds capable of exerting multi-target immunomodulatory and antioxidant effects with favorable safety profiles.

Among such candidates, *Ganoderma lucidum* (Reishi or Lingzhi) is a medicinal mushroom traditionally used in East Asian medicine and increasingly investigated for its pharmacological properties. Its bioactive constituents—including triterpenoids, polysaccharides, spore-oil fractions, and immunomodulatory proteins—have been associated with anti-inflammatory, antioxidant, and immunomodulatory activities in various experimental systems (Ren et al., 2025). In the context of periodontal disease, modulation of the NRF2/KEAP1 pathway has been investigated as a potential

mechanism through which phytotherapeutic compounds may enhance antioxidant defense and counteract oxidative stress (Tossetta et al., 2024). However, whether *G. lucidum*-derived bioactives exert their effects in periodontal tissues specifically through NRF2/KEAP1 signalling remains insufficiently established.

Despite a growing body of literature describing the systemic anti-inflammatory properties of *Ganoderma lucidum,* its application in periodontal inflammation has received comparatively limited and fragmented attention. Existing reviews of natural products in periodontitis often address broad classes of plant-derived compounds without specific synthesis of fungal bioactives, while reviews focusing on *G. lucidum* predominantly emphasize non-oral disease models. To date, no scoping review has systematically mapped the extent, nature, and mechanistic characteristics of evidence relating *G. Lucidum* to periodontal inflammation.

A scoping review was considered more appropriate than a systematic review because the currently available literature is small, methodologically heterogeneous, and oriented toward mapping experimental models, extract types, outcomes, and proposed mechanisms rather than estimating intervention efficacy. This approach also adds value beyond narrative reviews by using a transparent, structured framework to identify the extent and nature of periodontal-specific evidence on *Ganoderma lucidum* and to highlight gaps relevant to translational periodontology.

Accordingly, this scoping review aimed to map contemporary experimental evidence (2020–2025) on *Ganoderma lucidum* and its derivatives in models relevant to periodontal inflammation. The specific objectives were to: (1) characterize the types of experimental models and *G. lucidum* preparations investigated; (2) summarize reported inflammatory, oxidative, molecular, and tissue-level outcomes; and (3) identify the principal mechanistic pathways suggested within the included studies. By mapping this evidence, the review seeks to clarify the current scope and limitations of the available preclinical literature, highlight important research gaps, and inform future translational and clinical research in periodontology.

2. MATERIAL AND METHODS

2.1 Review design and framework

This scoping review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) guidelines and followed the methodological framework proposed by Arksey and O'Malley (2005), with refinements by Levac et al. (2010). A scoping approach was selected because the purpose of the review was to map the extent, nature, and characteristics of a limited and heterogeneous evidence base rather than to evaluate intervention effectiveness, perform meta-analysis, or generate pooled estimates.

2.2 Research questions

The review was guided by the following research questions:

(i) What experimental models have been used to investigate *Ganoderma lucidum*–derived preparations in periodontal or gingival inflammation?
(ii) What types of *Ganoderma lucidum* extracts or bioactives fractions have been evaluated?
(iii) What inflammatory, oxidative, and tissue-level outcomes have been reported?
(iv) What molecular pathways have been described in relation to host-modulatory effects?

2.3 Search strategy

A comprehensive literature search was conducted in PubMed, Scopus, and Web of Science to identify relevant studies published between January 2020 and September 2025. The search strategy combined

controlled vocabulary where available, such as Medical Subject Headings (MeSH) in PubMed, with free-text keywords, synonyms, and truncated terms related to *Ganoderma lucidum* and periodontal inflammation. Intervention-related terms included "*Ganoderma lucidum*," "Reishi," "Lingzhi," "Reishi mushroom," and "Lingzhi mushroom." Disease- and model-related terms included "periodontitis," "periodontal disease," "gingivitis," "gingival inflammation," "oral inflammation," periodont*, gingiv*, "*Porphyromonas gingivalis*," "alveolar bone loss," and "ligature-induced periodontitis," as appropriate for each database. Terms were combined using Boolean operators and adapted according to the syntax and search fields of each database. Searches were limited to peer-reviewed English-language articles. The 2020-2025 timeframe was selected to map recent evidence on biological and mechanistic trends in periodontal models, whereas earlier non-periodontal literature was considered only as supporting background in the discussion. Reference lists of included articles were also screened manually to identify additional eligible studies.

2.4 Eligibility criteria

Studies were included if they met the following criteria: (i) original experimental research (in vivo or in vitro); (ii) evaluation of *Ganoderma lucidum* or its bioactives components; (iii) use of periodontal or gingival inflammation models; and (iv) reporting of inflammatory, oxidative, or mechanistic outcomes. Reviews, commentaries, clinical case reports, conference abstracts, and studies unrelated to oral or periodontal inflammation were excluded.

2.5 Study selection

Following removal of duplicates, titles and abstracts were independently screened by two reviewers. Full-text assessment was performed for potentially eligible articles. Disagreements were resolved through discussion until consensus was reached. The study selection process is summarized using a PRISMA-ScR flow diagram.

2.6 Data charting and synthesis

Data were charted using a predefined extraction form, capturing information on study design, experimental model, *Ganoderma lucidum* preparation, dosage and route of administration, outcome measures, and reported molecular pathways. Consistent with PRISMA-ScR recommendations, no formal risk-of-bias or methodological quality assessment was undertaken, as the purpose of the review was to map available evidence rather than critically appraise intervention efficacy. Extracted data were synthesized narratively and organized thematically according to mechanistic domains.

3.0 RESULTS

3.1 Study selection

The database search identified 28 records, including 9 from PubMed, 14 from Scopus, and 5 from Web of Science. After removal of duplicates, 18 unique records remained. Application of the predefined publication date range (2020–2025) resulted in the exclusion of 6 records prior to title and abstract screening, leaving 12 records for further assessment. Following title and abstract review, 8 records were excluded, and the remaining 4 studies met the eligibility criteria and were included in the final synthesis. The study selection process is illustrated in Figure 1.

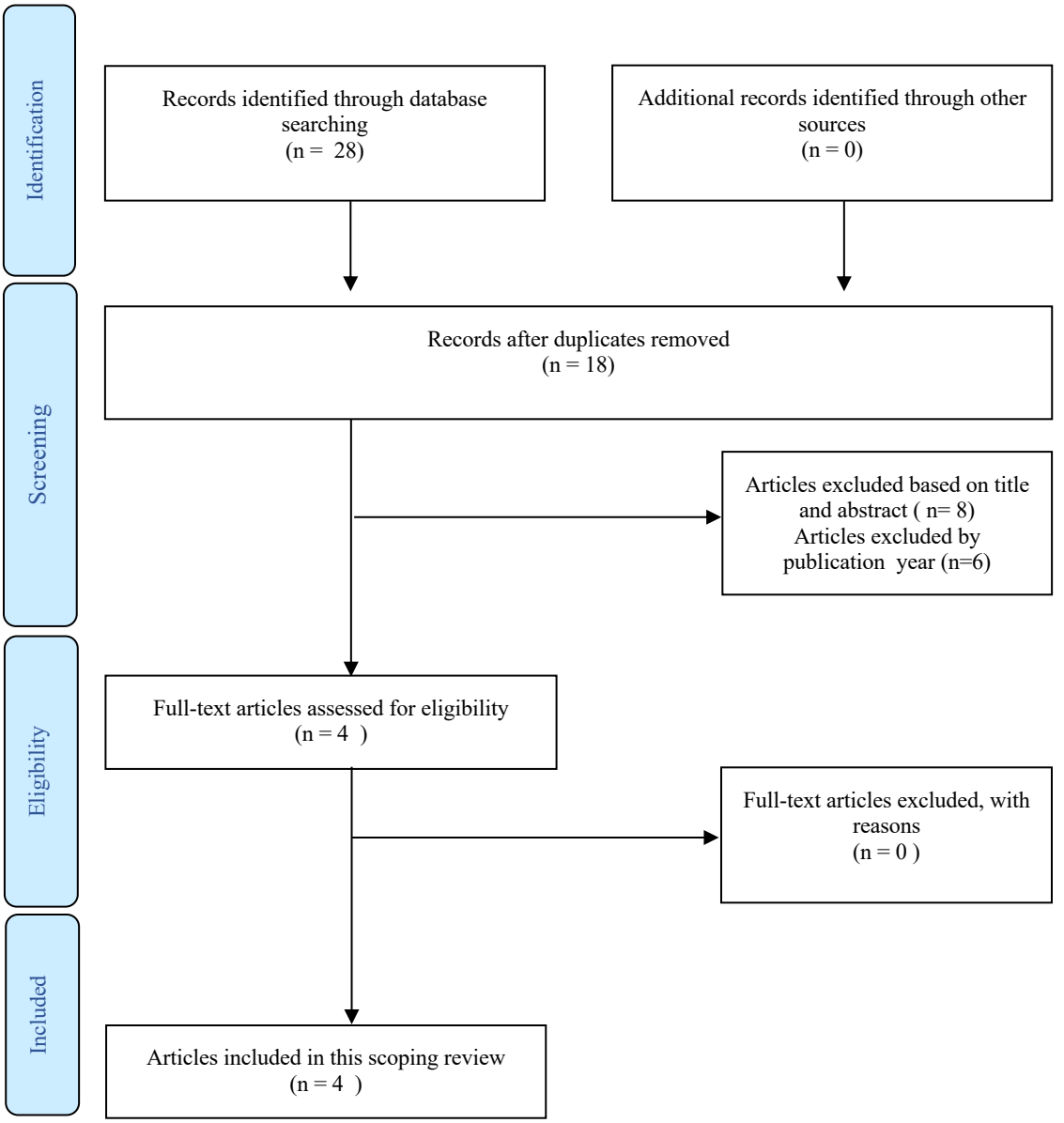


Fig. 1. PRISMA-ScR diagram of the search and study selection

Table 1. Direct experimental evidence (2020–2025) of *Ganoderma lucidum* effects on periodontal or gingival inflammation

No	Author (Year)	Experimental model	*G. lucidum* preparation	Dose and route	Key biological outcomes
1	Chen et al. (2023) [13]	Ligature-induced periodontitis in mice	Polysaccharide extract (CGLP)	50 mg/kg – 100 mg/kg BW daily, oral	Decreased IL-1β and TNF-α, increased IL-10, and reduced alveolar bone loss
2	Hossain et al. (2024) [14]	Ligature-induced periodontitis in rats	Ethanol extract	300 mg/kg BW daily, oral	Decreased gingival TNF-α and LPO levels, inhibited oxidative stress/lipid peroxidation, and showed antioxidant activity
3	Kim et al. (2024) [15]	In vitro inflamed human gingival fibroblasts and in vivo ligature-induced murine periodontitis in mice	Spore-oil extract (GLSO)	In vitro: 200, 400, and 800 μg/mL; In vivo: 6 mg/kg body weight, intraperitoneal injection	Reduced MMP-1 and IL-8 expression, decreased MMP activity, inhibited JNK signaling, and reduced periodontal inflammation and alveolar bone loss
4	Andini et al. (2024) [16]	*P. gingivalis* -induced periodontitis in rats	*Ganoderma lucidum* ethanolic extract	5, 10, and 20 mg/kg BW	Reduced gingival TNF-α and PGE_2 levels in periodontitis rats in a dose-dependent manner

3.2 Characteristics of included studies

All four included studies used preclinical experimental designs to investigate the effects of *Ganoderma lucidum* on periodontal or gingival inflammation. Three studies employed in vivo rodent models, while one combined inflamed human gingival fibroblasts in vitro with a murine ligature-induced periodontitis model. Two studies utilized ligature-induced periodontitis models, one employed a *Porphyromonas gingivalis*-induced periodontitis model, and one described a rat periodontitis model more generally. These experimental models reproduced key features of periodontal disease, including increased inflammatory mediators, oxidative stress, matrix degradation, and alveolar bone loss.

The duration of interventions was short-term across all four studies, generally ranging from 7 days to 2 weeks. *Ganoderma lucidum* preparations varied and included polysaccharide extracts, ethanolic extracts, spore-oil fractions, and crude extracts, as summarized in Table 1. Routes of administration also differed, including oral administration, oral gavage, and intraperitoneal injection, while the in vitro study applied the extract directly to cultured cells. Animal doses ranged from 5 to 300 mg/kg body weight, and in vitro

concentrations ranged from 200 to 800 μg/mL. The current evidence base therefore remains limited, short-term, and methodologically heterogeneous.

3.3 Reported inflammatory and oxidative outcomes

Across the four included studies, *Ganoderma lucidum* preparations were consistently associated with reductions in inflammatory mediators linked to periodontal tissue breakdown. Reported outcomes included decreases in tumour necrosis factor-α (TNF-α), interleukin-1β (IL-1β), prostaglandin E_2 (PGE_2), matrix metalloproteinase-1 (MMP-1), and interleukin-8 (IL-8), measured in gingival tissue, periodontal tissue, or inflamed gingival fibroblasts (Andini et al., 2024; Chen et al., 2023; Kim et al., 2024). One study also reported increased interleukin-10 (IL-10), suggesting a shift toward an anti-inflammatory response (Chen et al., 2023). In addition to these molecular changes, several studies described reduced alveolar bone loss and attenuation of periodontal inflammation in the treated groups (Chen et al., 2023; Kim et al., 2024).

Oxidative outcomes were reported less consistently but still suggested a potential antioxidative effect. One included study showed reduced lipid peroxide (LPO) levels in gingival tissue, inhibition of in vitro oxidative stress, and free radical-scavenging activity following *G. lucidum* administration (Hossain et al., 2024). Mechanistic evidence, however, was limited. Among the included studies, only Kim et al. (2024) directly examined intracellular signalling and found inhibition of c-Jun N-terminal kinase (JNK) phosphorylation, whereas nuclear factor kappa B (NF-κB) signalling was not significantly altered. The remaining studies discussed pathway-related mechanisms only indirectly and did not directly confirm specific intracellular targets. Overall, the findings point toward anti-inflammatory and, to a lesser extent, antioxidative effects of *G. lucidum* in experimental periodontal models, but these observations remain limited to short-term preclinical studies and should therefore be interpreted cautiously.

3.4 Reported molecular pathways

Reported molecular pathway findings were limited and were not examined uniformly across the included studies. Among the four studies, the clearest mechanistic evidence came from Kim et al. (2024), who showed that *Ganoderma lucidum* spore-oil extract suppressed phosphorylation of c-Jun N-terminal kinase (JNK) in inflamed human gingival fibroblasts, with accompanying reductions in MMP-1 and IL-8 expression. In contrast, NF-κB signalling was evaluated in that study but was not significantly affected, suggesting that the anti-inflammatory effect was more closely related to JNK pathway inhibition than to NF-κB modulation.

For the remaining studies, pathway-level interpretation was more indirect. Chen et al. (2023) reported reductions in IL-1β and TNF-α together with increased IL-10, but did not directly assess intracellular signalling pathways. Andini et al. (2024) discussed NF-κB-related mechanisms based on previous literature, although their own experimental findings were limited to reductions in TNF-α and PGE_2. Hossain et al. (2024) likewise reported inflammatory and oxidative changes without direct pathway analysis. Overall, the current evidence suggests possible involvement of JNK-related signalling, whereas the roles of other pathways, including NF-κB, remain proposed rather than confirmed within the included evidence base.

3.5 Histological and tissue-level findings

Histological and tissue-level findings from the included studies generally supported the reported anti-inflammatory associations of *Ganoderma lucidum* in experimental periodontal models. In the ligature-induced models, treatment was associated with reduced periodontal inflammation and lower alveolar bone loss compared with untreated controls, based on micro-computed tomography, histological observation,

and immunohistochemical assessment (Chen et al., 2023; Kim et al., 2024). These findings included better preservation of alveolar bone architecture, lower cementoenamel junction–alveolar bone crest distances, and reduced expression of inflammatory markers in periodontal tissues.

However, tissue-level outcomes were not assessed uniformly across all four included studies. In the *Porphyromonas gingivalis*-induced rat model, the reported findings were limited mainly to reduced gingival TNF-α and PGE_2 levels rather than detailed histological evaluation (Andini et al., 2024). Similarly, Hossain et al. (2024) focused primarily on inflammatory and oxidative biochemical markers. Therefore, while the available tissue-level findings are supportive, they remain limited and should be interpreted cautiously within the context of short-term preclinical evidence.

4.0 DISCUSSION

This scoping review mapped experimental studies published between 2020 and 2025 that examined *Ganoderma lucidum*-derived preparations in models relevant to periodontal inflammation. Although only four eligible studies were identified, all of which were preclinical, the available evidence still provides a useful early picture of the biological responses reported in this area. In line with the exploratory nature of a scoping review, the findings are discussed descriptively, with emphasis on reported inflammatory, oxidative, molecular, and tissue-level outcomes. These observations may be of interest in the context of host-modulation strategies in clinical periodontology, but they should be interpreted cautiously given the limited and heterogeneous nature of the current evidence.

4.1 Inflammatory responses in periodontal models

Across the included studies, administration of *G. lucidum*-derived preparations was associated with lower levels of several inflammatory mediators linked to periodontal tissue breakdown, including tumour necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), prostaglandin E_2 (PGE_2), matrix metalloproteinase-1 (MMP-1), and interleukin-8 (IL-8) (Andini et al., 2024; Chen et al., 2023; Kim et al., 2024). One study also reported increased interleukin-10 (IL-10), suggesting a possible shift toward a more anti-inflammatory profile (Chen et al., 2023). These mediators are closely linked to periodontal tissue destruction through their roles in inflammatory cell recruitment, matrix degradation, and bone resorption (Kinane et al., 2017; Könönen et al., 2019). In addition, some included studies reported reduced periodontal inflammation and lower alveolar bone loss in treated groups, supporting the biological relevance of these inflammatory changes at the tissue level (Chen et al., 2023; Kim et al., 2024).

At the molecular level, the available mechanistic evidence was limited. Kim et al. (2024) provided the clearest pathway-related finding by reporting that *G. lucidum* spore-oil extract was associated with suppression of c-Jun N-terminal kinase (JNK) phosphorylation in inflamed human gingival fibroblasts, together with reduced MMP-1 and IL-8 expression. In contrast, nuclear factor kappa B (NF-κB), signalling was assessed in the same study but was not significantly altered. In the remaining included studies, NF-κB related mechanisms were discussed mainly as biologically plausible explanations based on prior literature rather than as directly measured outcomes. Although NF-κB is an important regulator of inflammatory gene expression in periodontal disease (Chapple & Matthews, 2007), its involvement remains plausible, but within the present review it should be interpreted as a suggested mechanism rather than a confirmed molecular target.

4.2 Oxidative stress modulation

Oxidative findings were reported less consistently than inflammatory outcomes, but the available evidence suggested a possible antioxidative association of *Ganoderma lucidum* in periodontal models. Hossain et al. (2024) reported lower lipid peroxide (LPO) levels in gingival tissue following chronic oral administration of *G. lucidum* extract in periodontitis model rats, together with inhibition of in vitro oxidative stress and free radical-scavenging activity. These findings suggest that antioxidative effects may contribute, at least in part, to the biological responses observed in some periodontal models.

This interpretation is biologically plausible because oxidative stress is known to contribute to periodontal tissue injury by amplifying inflammatory signalling and promoting connective tissue and alveolar bone destruction (Baltacioglu et al., 2014; Chapple & Matthews, 2007). However, direct mechanistic validation of antioxidant signalling pathways was not performed consistently across the included studies. Therefore, the current evidence supports only a tentative association between *G. lucidum* and attenuation of oxidative stress in experimental periodontal settings.

4.3 Bioactives preparations and experimental heterogeneity

The included studies investigated several different *G. lucidum* preparations, including polysaccharide extracts, ethanolic extracts, spore-oil fractions, and crude extracts. Across these studies, treatment was associated with broadly favourable biological responses, including reduced inflammatory mediator expression, lower oxidative stress markers, and attenuation of tissue destruction (Andini et al., 2024; Chen et al., 2023; Hossain et al., 2024; Kim et al., 2024). However, it was not possible to determine whether one type of preparation was consistently more effective than another, because the extraction methods, experimental models, doses, routes of administration, and outcome measures varied substantially.

This heterogeneity limits direct comparison across studies and requires cautious interpretation of preparation-specific effects. In addition, variability in extract preparation and the limited chemical characterisation reported across some studies may reduce reproducibility and hinder meaningful comparison between different *G. lucidum* preparations. Taken together, the available literature suggests potential biological activity across different *G. lucidum* preparations, but the evidence remains too heterogeneous to support conclusions regarding the relative efficacy of individual extract types.

4.4 Tissue-level outcomes and translational relevance

At the tissue level, the available evidence suggested that *G. lucidum* treatment was associated with preservation of periodontal structures in some experimental models. In the ligature-induced studies, treated groups showed reduced periodontal inflammation and lower alveolar bone loss compared with untreated controls, based on micro-computed tomography, histological observation, and immunohistochemical findings (Chen et al., 2023; Kim et al., 2024). These observations are broadly consistent with the reported reductions in inflammatory mediators and support the biological plausibility of tissue-level protection in experimental periodontal inflammation.

From a clinical periodontology perspective, these findings are of interest because contemporary periodontal therapy increasingly considers host modulation as a potential adjunct to conventional mechanical treatment, particularly in patients with persistent inflammatory burden or increased risk of tissue breakdown (Könönen et al., 2019). Nevertheless, all included evidence was derived from short-term preclinical models. Although such models reproduce selected features of periodontal inflammation, they do not fully reflect the chronicity, microbial complexity, and host variability of human periodontitis (Könönen et al., 2019). Therefore, the reported tissue-level findings should be interpreted as preliminary experimental observations rather than indicators of clinical efficacy.

4.5 Research gaps and future directions

Several important gaps currently limit translation of *G. lucidum* research to periodontal applications. First, the evidence base remains very small, with only four included studies identified, all of which were preclinical. Second, substantial heterogeneity in extract preparation, dosing regimens, routes of administration, and outcome measures limits cross-study comparison (Andini et al., 2024; Chen et al., 2023; Hossain et al., 2024; Kim et al., 2024). Third, variability in extract preparation and limited chemical characterisation across the available studies make it difficult to determine which specific bioactive constituents may be responsible for the observed biological effects. Greater standardisation of *G. lucidum* preparations and more detailed chemical characterisation are therefore needed to improve reproducibility and facilitate future mechanistic and translational research.

For clinical periodontology, future research should move beyond descriptive animal findings toward translational models that better reflect human disease, including standardized preclinical protocols, mechanistic validation, and eventually early-phase clinical studies. Such work would help determine whether *G. lucidum*-derived bioactives have realistic potential as adjunctive host-modulatory agents alongside scaling and root planing or other established periodontal therapies.

Importantly, no randomised controlled trials evaluating *G. lucidum* as an adjunct to periodontal therapy were identified. Future research should prioritise translational studies and early-phase clinical trials assessing safety, tolerability, and modulation of relevant inflammatory biomarkers, in line with contemporary host-modulation paradigms in periodontology (Preshaw, 2018).

5. CONCLUSIONS

This scoping review mapped recent experimental evidence on *Ganoderma lucidum*-derived preparations in models relevant to periodontal inflammation. The available studies reported associations between *G. lucidum* administration and reduced inflammatory mediator levels, including TNF-α, IL-1β, PGE_2, MMP-1, and IL-8, with one study also reporting increased IL-10. Some studies additionally reported lower oxidative stress markers and reduced alveolar bone loss, suggesting possible anti-inflammatory, antioxidative, and tissue-protective associations in experimental periodontal settings. At the mechanistic level, evidence was limited, although one included study suggested involvement of JNK-related signalling, while NF-κB related mechanisms were discussed more indirectly and were not consistently confirmed across the included studies.

Despite these findings, the current evidence base remains small, heterogeneous, and restricted to short-term preclinical studies. Important gaps persist in relation to extract standardisation, chemical characterisation, mechanistic validation and clinical evaluation. Therefore, the present findings should be interpreted as preliminary experimental observations rather than evidence of clinical efficacy. Further rigorous translational research and well-designed human studies are needed before *G. lucidum*-based interventions can be meaningfully considered within periodontal therapy frameworks.

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CONFLICT OF INTEREST STATEMENT

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

AUTHORS' CONTRIBUTIONS

All authors participated in designing the study. NIAR conducted the literature search and data extraction. All authors contributed to the data analysis and synthesis of data. All authors reviewed and critically contributed to the drafting of the manuscript. All authors have approved the final version of the manuscript.

DECLARATION OF GENERATIVE AI IN THE WRITING PROCESS

During the preparation of this work, the authors used ChatGPT (OpenAI) to assist with language refinement, improving readability, grammar, and academic writing style. Following the use of this tool, the authors critically reviewed, edited, and verified all content and take full responsibility for the accuracy, originality, and integrity of the manuscript.

DATA AVAILABILITY

As this study is a scoping review, no new datasets were generated. All data analysed during this study were extracted from published articles included in the review. The data supporting the findings are presented within this article, and additional information is available from the corresponding author upon reasonable request.

ETHICS STATEMENT

This study is a scoping review of previously published literature and did not involve human participants, animal subjects, or the collection of primary data. Therefore, ethics committee approval and informed consent were not required.

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5. APPENDIX

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