



# Green Tea Catechins in Periodontal Therapy: A Literature Review of Efficacy as an Adjunct to Initial Periodontal Treatment

Imane Sabili, Nadia Ausalah Taoufik, Ihsane Benyahya

Faculty of Mohammed VI Dentistry, Mohammed VI University of Health Sciences (UM6SS), Casablanca, Morocco

Email: imanesabili@outlook.fr

**How to cite this paper:** Sabili, I., Taoufik, N.A. and Benyahya, I. (2026) Green Tea Catechins in Periodontal Therapy: A Literature Review of Efficacy as an Adjunct to Initial Periodontal Treatment. *Open Access Library Journal*, **13**: e15145. <https://doi.org/10.4236/oalib.1115145>

**Received:** March 11, 2026

**Accepted:** April 11, 2026

**Published:** April 14, 2026

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## Abstract

**Background:** Periodontitis is a chronic inflammatory disease initiated by pathogenic biofilms, leading to the progressive destruction of periodontal supporting tissues. Green tea catechins, particularly epigallocatechin-3-gallate (EGCG), have emerged as promising therapeutic agents due to their potent antioxidant, anti-inflammatory, and antimicrobial properties. **Objective:** This review aims to evaluate the clinical and biological efficacy of green tea catechins as an adjunct to scaling and root planing (SRP) in the management of chronic periodontitis. **Methods:** A narrative synthesis was conducted focusing on 7 pivotal articles that detail various local delivery systems, including chips, gels, strips, and dentifrices. These core studies provide the evidence base for assessing changes in Probing Pocket Depth (PPD), Clinical Attachment Level (CAL), and subgingival microbial flora. **Results:** The adjunctive use of green tea catechins significantly improved clinical parameters compared to SRP alone. Quantitative evidence showed a substantial reduction in PPD with a standardized mean difference (SMD) of 1.02 and a significant gain in CAL with an SMD of 0.58. Biologically, EGCG exerted strong bactericidal effects against *Porphyromonas gingivalis* at a concentration of 0.5 mg/mL and effectively modulated the host response by inhibiting collagenase activity and suppressing pro-inflammatory cytokines. **Conclusion:** Green tea catechins represent an effective, biocompatible, and safe adjunct to non-surgical periodontal therapy. Their dual ability to target bacterial pathogens while simultaneously modulating host inflammatory pathways offers a significant clinical advantage over mechanical therapy alone.

## Subject Areas

Global Health

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## Keywords

Green Tea, Catechins, EGCG, Periodontitis, Scaling and Root Planing, Adjunctive Therapy

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## 1. Introduction

Periodontal diseases, characterized by chronic inflammation of the tooth-supporting tissues, remain a significant public health concern due to their high prevalence and potential impact on systemic well-being [1]. The British Society of Periodontology defines this condition as a chronic inflammatory disease of bacterial etiology affecting the supporting tissues around the teeth, caused by an imbalance between oral biofilms and the host's immune response [1]. Conventional non-surgical periodontal therapy, primarily scaling and root planing, aims to disrupt this biofilm and halt disease progression, yet complete pathogen elimination is often unattainable due to the complex anatomy of periodontal pockets and bacterial recolonization [1]. Consequently, the integration of adjunctive agents has been explored to enhance therapeutic outcomes and mitigate the risk of disease recurrence, although current guidelines regarding local antimicrobials remain cautious due to the low quality of existing evidence and the potential for adverse effects associated with long-term chemical use [1]. In light of these limitations, there is a growing interest in identifying natural alternatives that possess antimicrobial and anti-inflammatory properties without inducing bacterial resistance or other significant adverse reactions [1]. Green tea, derived from the *\*Camellia sinensis\** plant, has emerged as a promising candidate in this regard, containing high concentrations of polyphenolic catechins that exhibit potent biological activities relevant to periodontal management [2]. Among these bioactive compounds, epigallocatechin-3-gallate is the most abundant and therapeutically active constituent, contributing significantly to green tea's capacity to inhibit bacterial adhesion, modulate inflammatory pathways, and reduce oxidative stress within the periodontal microenvironment [2]. This review aims to critically evaluate the current evidence regarding the incorporation of green tea catechins into initial periodontal therapy, and specifically to assess their efficacy as an adjunct to scaling and root planing in improving clinical parameters and modulating the host response [3] [4].

## 2. Background of Periodontal Disease

Periodontal disease encompasses a spectrum of pathological conditions affecting the periodontium, including gingivitis, periodontitis, and necrotizing periodontal diseases, which are initiated by microbial dysbiosis and perpetuated by the host's inflammatory immune response [1]. The complex interplay between pathogenic microorganisms and host defense mechanisms results in the progressive destruction of connective tissue attachment and alveolar bone, which characterizes the pathogenesis of periodontitis [5].

Current clinical protocols follow the EFP S3 Level Clinical Practice Guideline, which recommends a stepwise approach starting with the control of local and systemic risk factors, followed by subgingival instrumentation with or without adjunctive therapies [6]. Green tea catechins are evaluated within this second step as potential enhancers of “pocket closure”, defined as PPD < 4 mm [6] [7].

## 2.1. Etiology and Pathogenesis

The initiation and progression of periodontal destruction are fundamentally driven by a shift from a symbiotic microbial community to a dysbiotic biofilm dominated by gram-negative anaerobic bacteria, such as \*Porphyromonas gingivalis\*, \*Treponema denticola\*, and \*Tannerella forsythia\*, which collectively trigger a chronic host inflammatory response [1]. These pathogenic microorganisms release virulence factors, such as lipopolysaccharides and proteases, which stimulate host immune cells to release pro-inflammatory cytokines and matrix metalloproteinases, ultimately leading to the degradation of collagen fibers and resorption of alveolar bone [1] [8]. This dysregulated inflammatory cascade is further exacerbated by the overproduction of reactive oxygen species, which overwhelms the local antioxidant defense mechanisms and amplifies tissue damage through oxidative stress [1] [3]. Specifically, polymorphonuclear leucocytes generate reactive oxygen species as a primary defense mechanism against bacterial pathogens, yet excessive production induces lipid and protein oxidation that contributes to the breakdown of periodontal tissues [1]. Furthermore, the sustained release of pro-inflammatory mediators, such as interleukin-1 beta and tumor necrosis factor-alpha, not only perpetuates local inflammation but also stimulates osteoclastic activity, thereby facilitating the resorption of alveolar bone characteristic of advanced periodontitis [1]. The clinical manifestation of this destructive process includes the formation of periodontal pockets, clinical attachment loss, gingival bleeding, and increased tooth mobility, which collectively compromise the functional integrity of the dentition [1] [9]. The primary therapeutic objective in managing periodontitis is to suppress the dysbiotic microbial challenge and control the destructive host inflammatory response to prevent further attachment loss and maintain the functional stability of the dentition.

## 2.2. Current Periodontal Treatment Modalities

The management of periodontitis relies primarily on mechanical debridement to disrupt the subgingival biofilm and reduce the bacterial load, thereby creating an environment conducive to healing and tissue repair. Scaling and root planing constitute the cornerstone of non-surgical therapy, effectively removing supra- and sub-gingival calculus and bacterial endotoxins from root surfaces to facilitate the re-establishment of a healthy periodontal attachment apparatus [10]. Despite the established efficacy of mechanical debridement in reducing inflammation and probing depths, these procedures often fail to restore the natural balance of the oral microbiota or completely eliminate pathogenic bacteria from

deep pockets, leading to potential recolonization and disease recurrence [11]. To address these limitations, various adjunctive therapies have been proposed, including local and systemic antimicrobials, host modulation therapy, and the use of natural antioxidants, which are gaining attention for their ability to target the oxidative stress mechanisms that play a predominant role in tissue destruction [12] [13].

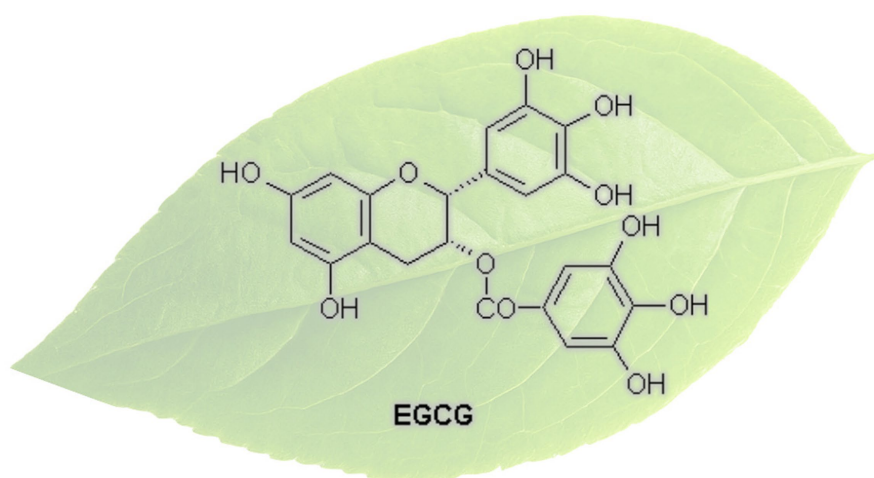
### 3. Green Tea Catechins: Chemical Composition and Pharmacological Properties

#### 3.1. Chemistry of Green Tea Catechins

Green tea, derived from the leaves of *Camellia sinensis*, is characterized by a high concentration of polyphenolic compounds known as catechins, which constitute approximately 30% to 42% of the leaf's dry weight [2] [14]. These compounds belong to the flavan-3-ol class and are primarily responsible for the plant's therapeutic properties [15]. The four major monomeric catechins found in green tea are:

- (-)-Epigallocatechin-3-gallate
- (-)-Epigallocatechin
- (-)-Epicatechin-3-gallate
- (-)-Epicatechin [2] [14]

Among these, EGCG is the most abundant and biologically active component, accounting for 50% to 80% of the total catechin content [14] [15]. Chemically, catechins are characterized by a di- or tri-hydroxyl group substitution on the B-ring and a meta-5,7-dihydroxy substitution on the A-ring (Figure 1) [2]. The presence of the galloyl moiety in EGCG and ECG significantly enhances their antioxidant and iron-chelating capabilities, which are crucial for neutralizing reactive oxygen species in the inflamed periodontal environment [16] [17]. Furthermore, the unique structure of EGCG allows it to bind to bacterial cell membranes and inhibit the activity of essential enzymes, such as collagenase and proteinases, which are key drivers of periodontal tissue destruction [3] [18].



**Figure 1.** Chemical structures of EGCG.

### 3.2. Pharmacological Properties Relevant to Periodontal Health

The antioxidant capacity of green tea catechins is particularly noteworthy, as their free radical scavenging activity has been reported to be significantly more potent than that of conventional antioxidants like vitamins C and E [16]. This superior bioactivity is attributed to the high density of hydroxyl groups on the catechin structure, which facilitates the neutralization of reactive oxygen species and the chelation of transition metal ions involved in oxidative reactions [16]. By mitigating oxidative stress, these compounds protect periodontal tissues from lipid peroxidation and protein degradation, thereby preserving the structural integrity of the gingiva and supporting alveolar bone [16]. In addition to their antioxidant potential, green tea catechins exhibit broad-spectrum antimicrobial activity against periodontal pathogens, including both gram-positive and gram-negative bacteria, as well as various fungi and viruses associated with oral infections [15] [14]. Epigallocatechin gallate constitutes the most effective antibacterial component among these catechins and has become the primary focus of contemporary research [15]. Specifically, EGCG has been shown to inhibit bone resorption induced by lipopolysaccharides by either suppressing the production of interleukin-1 beta or directly impeding osteoclastogenesis [19]. Additionally, it has been discovered that EGCG can reduce periapical lesions by inhibiting the expression of Cysteine-rich protein 61 in osteoblasts [19]. In the field of human dentistry, EGCG has been shown to enhance host conditions in periodontitis and periapical lesions through its bactericidal impact on periodontal pathogens and its inhibitory effect on cytokine production within gingival fibroblasts or osteoblasts [19]. EGCG exhibits antioxidant properties by minimizing oxygen radicals and stabilizing compounds [19]. Furthermore, its antitumor effects have been highlighted, elucidating how EGCG inhibits tumor cell growth and induces apoptosis through various cellular pathways, such as regulating the cell cycle, gene expression, signal transduction, and transcription factors [19]. These multifaceted interactions underscore the potential of EGCG to serve as a therapeutic agent capable of modulating the host response and altering the disease trajectory at a molecular level [3] [8].

## 4. Methodology

The methodology employed for this article is a narrative review of the literature. While a structured search was conducted to identify relevant studies, the analysis is primarily qualitative and narrative.

We conducted a comprehensive search across electronic databases, including PubMed, MEDLINE, Scopus, and Web of Science, to locate peer-reviewed articles published in English without restriction on publication date. The search strategy used a combination of Medical Subject Headings and free-text terms, including “green tea”, “*Camellia sinensis*”, “catechins”, “epigallocatechin gallate”, “periodontitis”, “periodontal disease”, “gingivitis”, “scaling and root planing”, and “adjunctive therapy”, to capture a broad spectrum of clinical evidence.

### 4.1. Search Strategy

We applied Boolean operators to refine the search results and ensure the retrieval of studies specifically addressing the intersection of green tea polyphenols and periodontal therapy outcomes. The search syntax was tailored to each database interface to maximize sensitivity, and the reference lists of retrieved articles were manually screened to identify additional relevant studies that may have been missed by the initial electronic query.

### 4.2. Selection Criteria

We considered studies eligible for inclusion if they investigated the effects of green tea catechins—whether administered as local delivery systems, mouthwashes, dentifrices, or dietary supplements—on clinical periodontal parameters in human subjects. To ensure a focused analysis of adjunctive efficacy, we prioritized randomized controlled trials and comparative clinical studies, while we excluded case reports, *in vitro* studies, and animal experiments from the primary selection process. Additionally, we eliminated articles lacking sufficient data on clinical outcomes or those focusing solely on general health parameters unrelated to periodontal status to maintain the relevance of the review.

The 7 primary studies were specifically retained because they provided longitudinal human clinical data (3 - 6 months) using standardized PPD and CAL metrics.

### 4.3. Data Extraction

A narrative synthesis was conducted to integrate the findings from the included studies. This review does not perform a new meta-analysis; instead, it contextualizes existing high-level evidence, such as the SMD values of 1.02 for PPD and 0.58 for CAL reported by Mazur *et al.* [14]. This narrative approach is necessitated by the significant heterogeneity in the dosages and formulations used in current periodontal research [1] [20].

Key data regarding study design, sample characteristics, intervention protocols, and clinical outcomes were systematically extracted from the selected full-text articles using a standardized form to ensure consistency and accuracy. The extracted variables included the type of catechin formulation used, such as gels or mouthrinses, as well as the specific dosage, frequency of application, and duration of the follow-up period [20]. Clinical outcome measures, specifically probing pocket depth, clinical attachment level, bleeding on probing, plaque index, and gingival index, were recorded to facilitate a quantitative comparison of treatment efficacy across the included studies [14] [20].

Due to the heterogeneity of the included protocols, a qualitative narrative synthesis was conducted. This approach was selected to rigorously distinguish between findings derived from laboratory models and direct clinical evidence from human randomized controlled trials [1] [14].

#### 4.4. Assessment of Risks of Bias Related to Article Selection

Protocol-related biases stem from the choice of language: Only articles in English were included, which may have excluded studies published in other languages.

### 5. Results

#### 5.1. Study Selection and Characteristics

Following the selection criteria, 7 pivotal clinical studies focusing on human subjects were included for primary analysis. These studies investigate various delivery formats including local chips, gels, and systemic beverages. To ensure a comprehensive review, these primary findings are further contextualized using larger meta-analytical data from Mazur *et al.* [14] and supportive mechanistic research [17] [21].

While the broader literature, notably the meta-analysis by Mazur *et al.*, compiles data from 15 clinical trials involving a total of 870 participants to establish high-level statistical evidence—reporting a standardized mean difference of 1.02 for probing pocket depth—this review provides a detailed analysis of the 7 key references that illustrate the diversity of current therapeutic protocols [14].

The characteristics of these included studies, summarized in **Table 1**, reveal a variety of administration modalities, including:

- **Green tea catechin-loaded strips** [18].
- **Biodegradable sustained-release chips** [3].
- **Topical EGCG gels concentrated at 1%** [22].
- **Direct delivery via ultrasonic scaler tips** [8].
- **Catechin-enriched dentifrices** [16].
- **Systemic green tea beverages** as an adjunct to mechanical therapy [1].
- **Locally manufactured subgingival gels** [1].

**Table 1.** Characteristics of included primary clinical studies (n = 7).

First Author, Year	Study Design	Intervention	Participants	Main Outcomes
Hattarki <i>et al.</i> , 2013 [18]	Clinico-Microbiological	Catechin Strips	20	Significant reduction in Plaque Index and Gingival Index at 5 weeks [18].
Kudva <i>et al.</i> , 2011 [3]	Clinical Trial	Biodegradable Chip	20	Inhibition of collagenase; significant reduction in anaerobic periodontal pathogens [3].
Wang <i>et al.</i> , 2021 [8]	Split-mouth RCT	EGCG via Scaler Tip	22	Effective biofilm disruption at 0.5 mg/mL; significant decrease in IL-6 and IL-8 levels [8].
Zeng <i>et al.</i> , 2022 [22]	Randomized Clinical Trial	Local EGCG Gel (1%)	20	Stable and significant PPD reduction and CAL gain maintained over 12 weeks [22].
Maruyama <i>et al.</i> , 2010 [16]	Clinical Study	Catechin Dentifrice	15	Suppression of gingival oxidative stress and reduction in periodontal inflammation [16].
Chopra <i>et al.</i> [1]	Double-blind RCT	Green Tea Beverage	120	Large-scale evidence: significant PPD reduction (P < 0.001) compared to control [1].
Nagate <i>et al.</i> [1]	Clinical Trial	Local Green Tea Gel	Pilot cohort	Improvement in both PD and CAL at 6 months post-initial therapy [1].

Follow-up periods across these clinical trials range from 21 days to 12 weeks (3 months), with some studies reporting long-term clinical benefits at 6 months, allowing for the evaluation of both the immediate inflammatory response and the stability of clinical attachment gains [1] [3] [22].

## 5.2. Supportive Mechanisms

These mechanisms, though promising, are based on *in vitro* studies and animal models that provide a theoretical framework rather than direct clinical proof [1]. Laboratory assays indicate that green tea catechins, particularly EGCG, inhibit the growth of periodontal pathogens like *Prevotella intermedia* and block enzymes responsible for collagen degradation [1] [16].

These findings represent laboratory-based research that explains the biological pathways through which green tea might impact oral health. They serve as a theoretical foundation rather than direct evidence of patient recovery.

- **Pathogen Inhibition:** *In vitro* studies have suggested that green tea catechins can inhibit the growth of specific periodontal pathogens, such as *Prevotella intermedia* [1].
- **Tissue Protection:** Laboratory evidence suggests that these catechins may help prevent the destruction of periodontal tissues by inhibiting the enzymes responsible for collagen breakdown [1].
- **Antimicrobial Potential:** Laboratory assays indicate that green tea vehicles, such as mouth rinses, can demonstrate a negative effect on the colony-forming units of bacteria like *Streptococcus mutans* and *Lactobacillus*, supporting its role as an antimicrobial agent [14]. The efficacy of EGCG is highly concentration-dependent. Research indicates that a Minimum Inhibitory Concentration of 1.0 mg/mL is required to achieve direct bacteriostatic effects against major periodontal pathogens such as *Porphyromonas gingivalis* and *Prevotella* species [22]. However, lower concentrations of 0.25 to 0.5 mg/mL are sufficient for anti-adhesive and biofilm-disruptive actions, effectively preventing these bacteria from attaching to host cells and inducing bacterial aggregation for easier removal [21] [22].

These *in vitro* findings establish the biological basis for the efficacy of EGCG, specifically identifying the 0.5 mg/mL threshold for effective biofilm disruption [8]. The integration of these mechanistic thresholds with their corresponding clinical outcomes is synthesized in **Table 2**.

## 5.3. Direct Clinical Proof

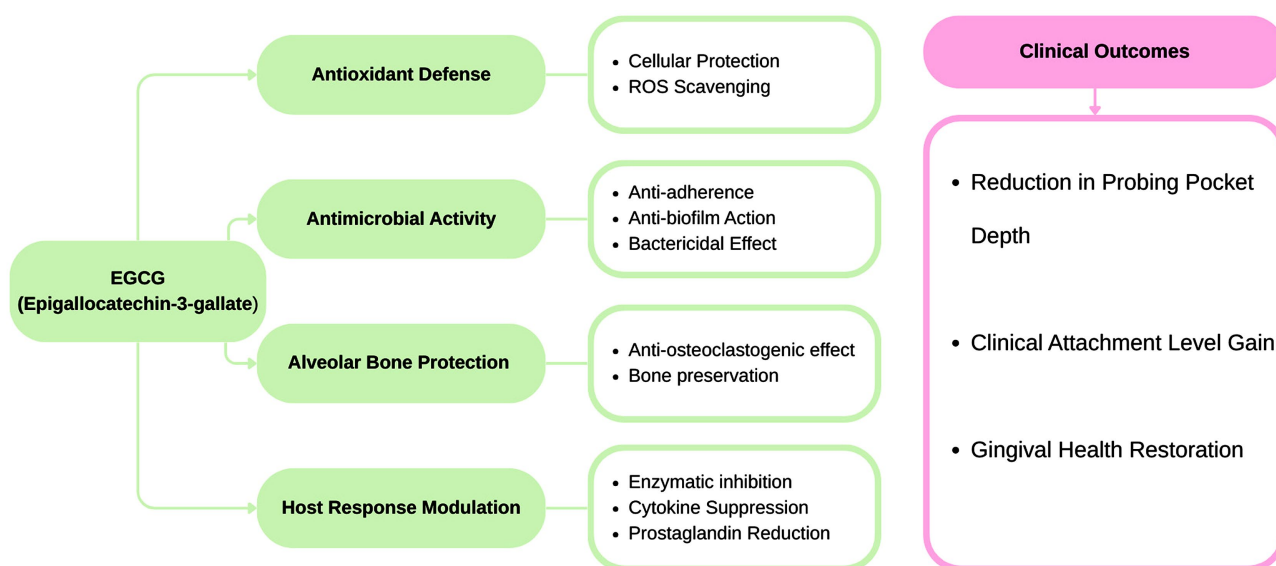
The clinical efficacy of green tea catechins is supported by a growing body of human randomized controlled trials. As synthesized in **Figure 2**, the therapeutic success observed in these trials is the result of multifaceted biological actions including biofilm disruption and host modulation that translate into measurable patient benefits [8] [21].

The quantitative impact of these interventions has been established by the

meta-analysis of Mazur *et al.*, which reports a large positive effect on Probing Pocket Depth (SMD = 1.02) and a medium positive effect on Clinical Attachment Level (SMD = 0.58) [14]. These findings are further detailed in **Table 2** and supported by individual trials, such as the double-blind RCT by Chopra *et al.* (n = 120), which demonstrated statistically significant improvements ( $P < 0.001$ ) over a 3-month period [1].

**Table 2.** Synthesis of mechanistic thresholds and clinical outcomes of EGCG.

Category	Source Type	Mechanism of Action	Threshold/Concentration
Mechanistic	In Vitro	<b>Bacteriostatic Effect</b>	<b>1.0 mg/mL</b> [22] [23]
Mechanistic	In Vitro	<b>Biofilm Disruption</b>	<b>0.5 mg/mL</b> [8] [21]
Mechanistic	In Vitro	<b>Metabolic Suppression</b>	<b>0.32–0.65 mg/mL</b> [21]
<b>Clinical</b>	Human RCTs	<b>PPD/CAL Gain</b>	$P < 0.001$ in human cohorts [1] [14] <b>SMD = 1.02</b> and <b>0.58</b> as reported by Mazur <i>et al.</i> [14]



**Figure 2.** Multifaceted therapeutic mechanisms of EGCG in periodontal health.

These findings are derived from randomized controlled trials and systematic reviews involving human subjects, providing verifiable evidence of clinical improvements in periodontal disease.

- **Clinical Parameter Improvements:** A systematic meta-analysis of human studies found that green tea has a large positive effect on reducing Probing Pocket Depth and a medium positive effect on Clinical Attachment Level [14].
- **Significant Treatment Outcomes:** In a double-blind, placebo-controlled RCT involving 120 human participants (aged 20 - 50), the adjunctive use of green tea yielded statistically significant improvements ( $P < 0.001$ ) in both PPD and CAL over a 3-month period [1]. It is important to note that while these clinical

gains are statistically significant, they are often derived from trials with small sample sizes, typically ranging from 15 to 22 participants [1] [18]. Such cohort sizes suggest that while the outcomes are positive, they require further validation through larger multi-center studies [14].

- **Long-Term Stability:** Human trials have shown that the results at six months significantly favor the use of green tea in improving periodontal health compared to control groups receiving standard scaling and root planing alone [1].
- **Evidence Constraints:** While clinical gains are documented, the strength of the evidence is tempered by factors such as small sample sizes in certain trials (e.g.,  $n = 10$  to  $12$ ) and potential allocation biases in studies where randomization was performed by simple methods like a coin toss [1].

### 5.3.1. Periodontal Pocket Depth and Attachment Level

The most significant impact of green tea catechins reported in the analyzed literature is the reduction of probing pocket depth. Meta-analysis results indicate a substantial positive effect with a standardized mean difference (*SMD*) of 1.02 [14]. Furthermore, clinical attachment level showed a significant improvement, with an *SMD* of 0.58 [14]. Among our 7 pivotal studies, clinical trials using local delivery systems—such as the biodegradable chips evaluated by Kudva *et al.*—confirmed that these clinical improvements are detectable as early as 21 days post-application [3].

### 5.3.2. Gingival Inflammation and Plaque Control

The administration of green tea catechins, whether through local delivery or topical application, resulted in a significant decrease in inflammatory markers across the selected trials [1] [16].

- **Gingival and Plaque Indices:** Adjunctive therapy showed superior efficacy in reducing both the Gingival Index and Plaque Index compared to placebo groups [14]. For instance, Hattarki *et al.* reported a significant reduction in PI ( $0.90 \pm 0.55$ ) and GI ( $1.15 \pm 0.58$ ) by the fifth week of treatment [18].
- **Bleeding on Probing:** A marked reduction in bleeding sites was consistently documented, reflecting a decrease in subgingival inflammation and improved tissue health [14] [18].

### 5.3.3. Comparison with Chemical Adjuncts

While green tea catechins demonstrated higher efficacy than placebos or triclosan-based formulations, their impact on GI and PI was slightly lower than that of chlorhexidine in some meta-analytical subgroups [14]. However, the 7 pivotal studies highlight that green tea formulations offer the advantage of high biocompatibility and a lower risk of side effects, such as tooth staining, which are commonly associated with long-term CHX use [4] [8]. The therapeutic benefits remained stable throughout follow-up periods extending up to 12 weeks in recent randomized clinical trials [12].

## 6. Discussion

### 6.1. Interpretation of Findings

The results of this narrative review confirm that green tea catechins, particularly epigallocatechin-3-gallate, are highly effective adjuncts to scaling and root planing in the management of periodontitis [15] [22]. The clinical significance of these findings is primarily highlighted by the meta-analytical data reported by Mazur *et al.*, which demonstrates a large positive effect size for probing pocket depth reduction (SMD = 1.02; 95% CI: 0.45 - 1.59) and a medium positive effect size for clinical attachment level improvement (SMD = 0.58; 95% CI: -0.49 - 1.65) compared to SRP alone [14]. It is important to emphasize that the quantitative results discussed in this section represent a narrative interpretation of synthesized literature rather than a primary statistical pooling of data [14]. This allows for a more nuanced discussion of how adjuncts like green tea fit into the broader clinical framework of the EFP S3 Level Guidelines, which emphasize a stepwise approach toward achieving “pocket closure” (PPD < 4 mm) through both mechanical and adjunctive means [6] [7].

The observed clinical gains are largely attributed to the dual chemical and biological actions of catechins. Microbiologically, EGCG exhibits a clear concentration-dependent efficacy: research indicates that a Minimum Inhibitory Concentration of 1.0 mg/mL is required to achieve direct bacteriostatic effects against major periodontal pathogens, such as *Porphyromonas gingivalis* and *Prevotella* species [18] [22]. However, lower concentrations ranging from 0.25 to 0.5 mg/mL are sufficient for anti-adhesive and biofilm-disruptive actions, effectively preventing these bacteria from attaching to host cells and inducing bacterial aggregation for easier removal [19] [21]. This dual threshold—1.0 mg/mL for growth inhibition and 0.5 mg/mL for biofilm disruption—is a critical finding for clinical application, suggesting that even if a sustained 1.0 mg/mL concentration is challenging to maintain with simple mouthwashes, the lower threshold for neutralizing pathogen virulence remains achievable and therapeutic [21] [22].

Simultaneously, these compounds modulate the host response by suppressing pro-inflammatory cytokines and neutralizing reactive oxygen species, thereby protecting the periodontal ligament and alveolar bone from oxidative damage [16]-[18]. The antioxidant properties of catechins help stabilize the gingival environment, which further enhances the mechanical effects of subgingival debridement [3] [16]. While the clinical improvements are statistically significant ( $P < 0.001$ ), the narrative synthesis suggests that these results are often derived from trials with relatively small sample sizes, highlighting the need for continued large-scale research to confirm these promising trends [1] [18].

### 6.2. Clinical Applicability

In terms of clinical applicability, the most feasible delivery formats for routine non-surgical periodontal care include green tea-based mouthwashes, dentifrices,

and local drug delivery systems such as subgingival gels [1] [16]. While mouthwashes offer high patient compliance for daily maintenance, professional local delivery systems are particularly effective for targeting deep pockets where higher concentrations of EGCG are required for bacteriostatic action [3] [18]. However, the significant heterogeneity in therapeutic dosages, varying formulations, and disparate follow-up durations (ranging from 3 to 6 months) across the literature currently prevents the recommendation of a single, standardized clinical protocol [14] [20]. Until multi-center longitudinal trials establish a gold-standard concentration and delivery frequency, clinicians should view green tea as a personalized adjunct rather than a replacement for conventional mechanical therapy [1] [24].

### 6.3. Comparison with Other Adjunctive Therapies

When compared to standard chemical adjuncts, green tea catechins offer a favorable safety profile [14]. While chlorhexidine remains a gold standard for plaque control, evidence suggests that green tea formulations can achieve comparable results in gingival index reduction without the risk of tooth staining or taste alteration [4] [14]. While some studies show that CHX may be slightly more effective in reducing the plaque index, green tea catechins provide superior biocompatibility, promoting faster healing of the connective tissue through the inhibition of collagenase activity [14] [18]. This suggests that green tea is not only a natural alternative but a biologically active therapeutic agent that supports tissue regeneration [22].

### 6.4. Limitations of Current Research

Despite the positive clinical outcomes observed, several limitations must be addressed to translate these findings into routine clinical practice.

The primary concern regarding both the 7 pivotal studies and the broader literature is the significant heterogeneity in delivery systems—ranging from local chips and gels to catechin-loaded strips and specialized scaler tips—which complicates the establishment of a standardized clinical protocol [8] [14] [20]. Furthermore, many of the core clinical trials analyzed in this review feature relatively small sample sizes, often involving only 15 to 22 participants, which may limit the statistical power and generalizability of the reported benefits [3] [8] [16].

Another critical limitation is the relatively short follow-up duration across the studies. Most trials monitored patients for periods ranging from 21 days to 12 weeks, providing insufficient data on the long-term stability of clinical attachment gains or the permanence of subgingival microbial shifts [3] [22]. Additionally, the lack of standardized EGCG concentrations across different green tea formulations makes it difficult to define a definitive therapeutic dose for adjunctive treatment [14]. Finally, current research often lacks ethnic diversity, with many trials conducted in specific geographic regions, which may limit the applicability of the findings to a global patient population [1].

Although this review highlights the potential of green tea as a therapeutic ad-

junct, several methodological limitations in the current literature must be addressed. First, significant heterogeneity exists across delivery systems (e.g., mouthwashes versus local gels), which complicates the establishment of a standardized therapeutic dosage [14] [20]. Second, there is a potential for measurement bias in several trials due to a lack of documented detail regarding the blinding of clinical examiners [1]. Finally, most human studies are limited to a 3-to-6-month follow-up period, leaving the long-term stability of the reported clinical attachment gains uncertain [1] [24].

## 7. Conclusion and Future Directions

In conclusion, this narrative review confirms that green tea catechins, particularly epigallocatechin-3-gallate, represent a promising therapeutic adjunct to scaling and root planing in the management of periodontitis [14] [16]. The synthesized evidence highlights a dual efficacy: a direct antimicrobial action, with a bacteriostatic threshold established at 1.0 mg/mL, and a capacity to disrupt periodontal biofilms at concentrations as low as 0.25 to 0.5 mg/mL [8] [21]. Clinically, these biochemical properties translate into significant improvements in periodontal parameters, notably a substantial reduction in probing pocket depth (SMD = 1.02) and clinical attachment level gain (SMD = 0.58) compared to mechanical debridement alone [14]. The adjunctive use of these catechins aligns with the “pocket closure” strategy (PPD < 4 mm) advocated by the EFP S3 Level Clinical Practice Guideline [6] [7].

Despite these encouraging results, the heterogeneity in dosages, formulations, and follow-up durations in the current literature still prevents the recommendation of a universal clinical protocol [1] [20]. Future research directions must focus on conducting long-term, multicentric clinical trials to identify the optimal delivery mode—specifically comparing local-release gels to therapeutic mouthwashes—and to standardize therapeutic concentrations [4] [14]. Furthermore, in-depth studies on the long-term stability of catechins in the oral environment and their specific impact on the global periodontal microbiome will be essential to definitively validate their role as a standard of adjunctive care in non-surgical periodontal therapy [16]-[18].

## Conflicts of Interest

The authors declare no conflicts of interest.

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