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Review Article

Doxycycline: More Than An Antibiotic

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ABSTRACT

Doxycycline is a widely used tetracycline antibiotic that has gained particular importance in periodontics because of its antimicrobial and host-modulating properties. Periodontitis is a chronic inflammatory disease in which microbial dysbiosis and an exaggerated host response contribute to progressive destruction of periodontal tissues. Although doxycycline exhibits bacteriostatic activity through inhibition of bacterial protein synthesis, its therapeutic relevance extends beyond antimicrobial action. This review highlights the pharmacokinetic, pharmacodynamic, anti-inflammatory, and host-modulating properties of doxycycline and their clinical implications in periodontal therapy. Doxycycline demonstrates excellent oral absorption, prolonged half-life, good tissue penetration, and minimal requirement for dose adjustment in renal impairment. At sub-antimicrobial doses, particularly 20 mg twice daily, it inhibits matrix metalloproteinases, reduces collagen degradation and pro-inflammatory mediators, and supports periodontal tissue preservation without exerting significant antibacterial pressure. Its clinical applications include adjunctive subantimicrobial-dose therapy with scaling and root planing, local drug delivery for periodontal pockets, and emerging applications in peri-implantitis. The review also discusses adverse effects, contraindications, and the development of chemically modified tetracyclines and advanced local delivery systems. Overall, doxycycline represents a transition from conventional antimicrobial therapy toward biologically based host-modulation strategies in periodontal and peri-implant disease management.

INTRODUCTION

Periodontitis is a chronic multifactorial inflammatory disease initiated by dysbiotic plaque biofilms and propagated by an exaggerated host immune response characterised by progressive destruction of the tooth-supporting apparatus. Standard Treatment of periodontal disease includes removing of dental plaque and other irritating factors (conservative treatment) or performing surgical methods [1]. Some bacteria are likely to get away from host defenses after non-surgical periodontal therapy due to restricted

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means of entry to the root surface and the tissue-invading abilities of the pathogens [2]. However, bacteria in deep pockets, furcations and within tissues often persists post SRP due to anatomical limitations and invasive properties of *Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis.*

What makes doxycycline super-special?

To overcome these, a variety of systemic and local antibiotics were used as adjunctive therapy to enhance the treatment outcome of SRP [3]. Systemic doxycycline have been long used as an adjunct to supplement the effect of scaling and root planning [4]. For over 4 decades, doxycycline has served as a prominent antibiotic within the tetracycline family, finding extensive application in clinical practice [5]. Doxycycline is an antibiotic used for treatment of periodontal disease, especially in case of aggressive periodontal disease [6].

Table -1

Generation / Class	Drugs
1st generation	Tetracycline, Chlortetracycline, Oxytetracycline, Demeclocycline
2nd generation	**Doxycycline**, Minocycline
3rd generation / newer tetracyclines	Tigecycline, Eravacycline, Omadacycline
Chemically modified tetracyclines (CMTs)	CMT-3 (6-demethyl-6-deoxy-4-de(dimethylamino)tetracycline) and related compounds

This review throws light on pharmacology, mechanism and clinical applications of doxycycline proving it as more than an antibiotic in periodontics.

Pharmacokinetics and Pharmacodynamics of Doxycycline:

Absorption and Distribution:

Doxycycline is almost completely absorbed after oral administration, with food or dairy reducing serum levels by only ~20% unlike tetracycline and minocycline, which are more significantly affected by co-administered cations [7].

Metabolism and Excretion:

It has a long serum half-life of 18–22 hours, unaffected by renal function, allowing convenient once- or twice-daily dosing well suited to sustained periodontal regimens such as SDD. Peak serum levels occur within 2–3 hours of oral intake [8].

Being highly lipophilic, doxycycline penetrates well into periodontal and other soft tissues, and accumulates in mineralized tissues (bone, teeth) through stable calcium-chelate formation the basis of tetracycline-associated tooth discoloration, relevant when prescribing in children or pregnancy [9]. Protein binding is high (82–90%) [10].

Of particular periodontal relevance, topical gingival application produces measurable doxycycline concentrations in gingival crevicular fluid without corresponding systemic levels the pharmacological basis for local drug delivery systems that achieve high local drug availability with minimal systemic exposure [9].

Elimination occurs via renal and biliary/fecal routes; unlike tetracycline, in renal impairment doxycycline excretion compensates via the fecal

route, so no dose adjustment is required in renally compromised patients [11].

Pharmacodynamics:

Doxycycline shows concentration-dependent antibacterial activity with a demonstrable post-antibiotic effect, though defined pharmacodynamic targets (AUC/MIC) are not established [12].

Table -2

Parameter	Doxycycline
Bioavailability	~90–100%
Half-life	~18–22 hours
Protein binding	>90%
Tissue distribution	Wide; good tissue penetration
Food effect	Minimal / formulation-dependent
Renal adjustment	Usually not required

Mechanism of Action:

Antimicrobial property:

Doxycycline is bacteriostatic, reversibly binding the bacterial 30S ribosomal subunit to block aminoacyl-tRNA attachment and inhibit protein synthesis, with additional action at mitochondrial 70S ribosomes [9].

Anti-Inflammatory Property:

At sub-antimicrobial doses, doxycycline exerts anti-inflammatory effects independent of its antibacterial action, through three main mechanisms: anticollagenolytic activity, inhibition of matrix-degrading metalloproteinases (MMPs), and downregulation of pro-inflammatory cytokines [15]. These actions reduce connective tissue breakdown and dampen the host inflammatory response, forming the basis for its use as a host-modulating agent in periodontitis. Doxycycline also inhibits nitric oxide (NO) synthesis, a key inflammatory mediator that drives vasodilation and tissue damage during inflammation. This provides a separate pathway for its anti-inflammatory action, distinct from MMP inhibition [16]. This mechanism has been particularly noted in septic shock, where direct inhibition of nitrate release is suggested as the main contributor to doxycycline's protective effect.

Host-Modulating property

Beyond its antimicrobial effect, doxycycline inhibits matrix metalloproteinases (MMPs) — enzymes released by inflammatory cells that drive collagen and bone destruction in periodontitis — independent of its antibacterial action [13]. It also promotes gingival fibroblast attachment and wound healing, and inhibits angiogenesis and apoptosis [13]. This anti-collagenolytic property underlies sub-antimicrobial dose doxycycline (SDD) therapy, where sub-bacteriostatic concentrations suppress MMP-mediated tissue destruction without antimicrobial pressure or resistance risk [14].

This effect, first identified by Golub et al. in diabetic rat models (including germ-free animals, confirming it was independent of antibacterial activity), established doxycycline as the most potent MMP inhibitor among the tetracyclines [17].

This inhibition occurs through multiple non-antimicrobial mechanisms: chelation of calcium/zinc ions essential for MMP catalytic activity, downregulation of MMP gene expression, reduced conversion of pro-MMP to its active form, and scavenging of reactive oxygen species that activate latent MMPs [18].

Clinically, subantimicrobial-dose doxycycline (SDD) administered adjunctively with scaling and root planing significantly reduced MMP-8 and MMP-13 levels and collagen degradation fragments in gingival crevicular fluid compared with SRP alone [19]. This host-modulating property shifted periodontal therapy's focus toward controlling the destructive host inflammatory-enzymatic response alongside pathogen control, and led to the development of chemically modified tetracyclines (CMTs) — non-antimicrobial derivatives retaining MMP-inhibitory activity without antibiotic effect [14].

Table - 3

Effect	Dose required	Clinical relevance
Antibacterial	100–200 mg/day	Used at antimicrobial doses to suppress susceptible bacteria; not the main purpose of SDD
MMP inhibition	20 mg twice daily	↓ MMP-8, MMP-13 → ↓ collagen degradation → preservation of periodontal connective tissue
Cytokine reduction	20 mg twice daily	↓ pro-inflammatory mediators such as IL-1β, IL-6, TNF-α → ↓ periodontal inflammation
Fibroblast stimulation / tissue preservation	20 mg twice daily	Promotes a more favorable connective-tissue environment and supports clinical attachment gain

Clinical Implications

Subantimicrobial Dose Doxycycline (SDD)

Periodontitis is initiated by periodontopathic bacteria, but tissue destruction — attachment loss and bone resorption — is largely mediated by the host's own inflammatory response, through release of cytokines and MMPs [19]. This rationale underlies host-modulation therapy: by suppressing destructive host enzymatic activity, clinical outcomes such as reduced inflammation, decreased probing depths, and gain in clinical attachment can be enhanced.

Periostat, a 20 mg doxycycline formulation taken twice daily, exploits this principle at sub-antimicrobial concentrations — inhibiting collagenase/MMP activity without exerting antibacterial pressure, and therefore without inducing bacterial resistance [20]. It is indicated as an adjunct to scaling and root planing, particularly where gingival bleeding or inflammation persists after initial therapy, with a typical regimen of six to nine months. This approach has shown particular benefit in smokers, in whom clinical signs of inflammation are often masked despite ongoing disease activity [21].

Local Drug Delivery Systems

Doxycycline hyclate (Atridox) is a locally delivered tetracycline formulation targeting specific periodontopathic organisms such as *Aggregatibacter actinomycetemcomitans* [6]. It consists of a bioabsorbable poly-DL-lactide/N-methyl-2-pyrrolidone gel containing 10% doxycycline, supplied as two pre-filled syringes mixed chairside and delivered subgingivally via

cannula. On contact with gingival crevicular fluid, the flowable gel solidifies in situ, conforming to pocket morphology and providing controlled drug release over approximately 7 days.

Atridox produces significant, sustained (60%) reductions in anaerobic pathogens for up to 6 months post-placement [22]. In chronic periodontitis patients, application at baseline and 4 months achieved reductions in probing depth (1.3 mm) and clinical attachment gain (0.8 mm) comparable to SRP alone at 9 months [23]. Notably, unlike mechanical therapy alone — where smokers typically respond less favorably — Atridox's clinical outcomes were unaffected by smoking status, a finding later supported by studies showing improved healing in smokers following its local application [24]. Its side-effect profile was comparable to placebo.

Atridox is best used as an adjunct rather than monotherapy; effective biofilm disruption via SRP remains essential, and enhances the efficacy of subsequently applied local antimicrobial agents [25].

Clinical Applications of Doxycycline in Periodontics

Chronic Periodontitis

- Subantimicrobial-dose doxycycline (SDD): 20 mg twice daily
- Used as an adjunct to scaling and root planing (SRP).
- Main action: host modulation rather than antibacterial action.
- Inhibits MMPs, particularly MMP-8.

Results in:

- ↓ collagen degradation
- ↓ periodontal inflammation
- ↓ probing pocket depth
- ↑ clinical attachment level
- Particularly useful in patients with moderate-to-severe periodontitis as an adjunct to mechanical therapy [14].

Aggressive Periodontitis

- Systemic doxycycline has historically been used as an adjunct to mechanical periodontal therapy [26].
- Commonly used dose: 100 mg/day.

Provides:

- Antibacterial activity against susceptible periodontal pathogens
- MMP inhibition
- Host-response modulation
- May result in reduction in pocket depth and improvement in clinical attachment.
- Important: “Aggressive periodontitis” is an older classification term. In the current 2017 classification, it is included under periodontitis, with staging and grading.

Refractory Periodontitis

- Doxycycline has historically been investigated in patients showing persistent or recurrent periodontal disease despite conventional therapy.
- Systemic doxycycline may provide:
  - Antibacterial effect
  - MMP inhibition
  - Reduction in connective-tissue breakdown
- Can be considered as an adjunct, particularly when mechanical therapy alone has not adequately controlled disease.
- However, refractory periodontitis is not a current formal diagnostic category.

Doxycycline and Peri-Implantitis

Local doxycycline shows promise as an adjunct to non-surgical peri-implantitis management, though evidence remains limited by few RCTs and heterogeneous protocols. This aligns with periodontal literature: a meta-analysis by Herrera et al. (2020) found local doxycycline produced significantly greater probing depth reduction (0.28 mm) and attachment gain (0.41 mm) than controls at 6–12 months [(26)].

Supporting studies show locally delivered doxycycline reduces bleeding and inflammatory markers (IL-1β, MMP-8, MMP-9) before periodontal regeneration,[(27)] and in diabetic periodontitis patients, improves clinical parameters alongside reductions in pathogens, cytokines, and HbA1c [(28)]. Given the overlapping inflammatory pathology of periodontitis and peri-implantitis, these findings lend indirect support to its use around implants, corroborated by case reports of successful peri-implantitis resolution using local doxycycline with mechanical debridement [(29)].

Mechanistically, local doxycycline effectively disinfects contaminated titanium surfaces with no significant cytotoxicity or adverse effects reported [(30)]. However, sparse RCT data currently limit firm clinical recommendations, despite consistent, biologically plausible positive effects across studies.

Adverse Effects

Doxycycline is generally well tolerated, with a favourable safety profile compared to other tetracyclines. Common effects include nausea, diarrhoea, and skin reactions such as rash, photosensitivity, and photo-onycholysis, seen in both children and adults [(31)]. Parenteral administration may additionally cause local phlebitis and pain at the infusion site.

Like other tetracyclines, doxycycline binds calcium in developing mineralized tissues, leading to enamel hypoplasia, permanent tooth discolouration, and transient growth retardation when administered during active tooth and bone development [(32)]. For this reason, it is contraindicated from the second half of pregnancy through 7 years of age — the period of primary and permanent tooth mineralization — as exposure during this window can cause irreversible dental and skeletal effects in the fetus and young child [(32)].

Table -4

Parameter	Doxycycline	Other tetracyclines
Absorption	Excellent (~90–100%); food has minimal effect with many formulations	Variable; tetracycline absorption significantly affected by food, milk and divalent/trivalent cations
Half-life	Long: ~18–22 h	Tetracycline: ~6–12 h; Minocycline: ~15–23 h
Renal adjustment	Usually NOT required	Usually required with tetracycline; minocycline may require caution/adjustment
Tissue penetration	Excellent; highly lipophilic; good periodontal tissue penetration	Generally good; minocycline > doxycycline > tetracycline for lipid-soluble tissue penetration
MMP inhibition	Strong; particularly MMP-8 inhibition → important host-modulatory effect	Present in tetracyclines, but doxycycline is best established clinically for subantimicrobial MMP inhibition

Future Directions

Development of non-antibacterial tetracyclines: Future research is focusing on chemically modified tetracyclines (CMTs) that retain the matrix metalloproteinase (MMP)-inhibitory and host-modulatory properties of doxycycline while eliminating antibacterial activity. This could allow prolonged host-modulation therapy with a lower risk of antimicrobial resistance and alteration of the oral microbiome. (33)

Development of selective MMP inhibitors: Since excessive MMP activity contributes to collagen degradation and periodontal tissue destruction, future therapies aim to selectively inhibit pathological MMP activity while preserving the physiological functions of MMPs required for normal tissue remodeling and repair. (34)

Novel host-modulation agents: Newer host-modulatory compounds, including chemically modified curcumins**,** are being investigated as potential alternatives to tetracycline-based therapy. These agents target inflammatory and collagenolytic pathways and have shown promising results in experimental models, although further clinical studies are required.(33)

Combination host-modulation therapy: Combining doxycycline with other host-modulatory agents may allow simultaneous targeting of different pathways involved in periodontal inflammation, **including** MMPs, cytokines, oxidative stress and resolution pathways. This represents a potential direction for improving the effectiveness of adjunctive periodontal therapy. (33)

Personalized host-modulation therapy: Future periodontal treatment may involve identifying patients with an exaggerated inflammatory or collagenolytic response and selecting host-modulatory therapy according to their individual biological risk profile. This could shift periodontal treatment toward a more patient-specific therapeutic approach. (34)

Advanced local drug-delivery systems: Development of **gels, nanoparticles and sustained-release systems** may allow doxycycline or other host-modulatory agents to be delivered directly to periodontal lesions, producing high local concentrations while minimizing systemic exposure.

CONCLUSION

Doxycycline exemplifies the evolution of periodontal therapeutics from a purely antimicrobial approach to one that also addresses host-mediated tissue destruction. Beyond its bacteriostatic action against periodontopathic organisms, its unique ability to inhibit matrix metalloproteinases and modulate the host inflammatory response — independent of antibacterial activity — has established it as a valuable host-modulating agent, particularly in its sub-antimicrobial dose form. Its applications extend from local drug delivery in periodontal pockets to adjunctive use in peri-implantitis, offering measurable clinical benefit with a favourable safety profile when used within recommended dosing and contraindication guidelines. As research into host-modulation therapy continues to expand, doxycycline and its chemically modified analogues are likely to remain central to the evolving, more biologically comprehensive approach to managing periodontal and peri-implant disease — truly justifying its description as more than an antibiotic.

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