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Quorum Quenching as an Ecological Modulator of Periodontal Biofilms

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ABSTRACT:

Background: Periodontal disease is driven by dysbiotic biofilms characterized by altered microbial functions rather than mere pathogen overgrowth. Quorum sensing (QS) enables coordinated microbial behavior that sustains dysbiosis, while quorum quenching (QQ) has emerged as a strategy to disrupt microbial communication without bactericidal effects. Existing reviews primarily address QS inhibition broadly or focus on cariogenic biofilms, leaving a critical gap regarding the ecological implications of QQ in periodontal biofilms.

Objective: This scoping review aimed to map current evidence on quorum quenching strategies targeting periodontal bacteria, with a specific focus on their role in modulating biofilm ecology and restoring microbial balance.

Methods: A scoping review was conducted following PRISMA ScR guidelines. Searches were performed in PubMed, Scopus, and Web of Science for studies published between 2016 and 2025 investigating quorum quenching mechanisms in periodontal bacteria or periodontal biofilm models. Data were charted and synthesized descriptively.

Results: Five studies met the inclusion criteria. Quorum quenching strategies included enzymatic degradation of signaling molecules, inhibition of signal synthesis or reception, and natural compound based inhibitors. Most studies targeted interspecies signaling pathways and demonstrated reduced biofilm maturation, attenuation of virulence-associated functions, and partial restoration of symbiotic microbial behavior without eliminating commensals.

Conclusion: Quorum quenching represents a novel ecology based approach for periodontal biofilm management by reprogramming microbial communication rather than eradicating bacteria. This perspective highlights quorum quenching as a promising adjunctive strategy for restoring periodontal biofilm homeostasis

KEYWORDS: antivirulence therapy, dysbiosis, oral microbiome, periodontal biofilm, quorum quenching.

INTRODUCTION

Periodontal disease is increasingly recognized as a consequence of ecological dysregulation within polymicrobial biofilms rather than a classical infectious disease caused by specific pathogens. The shift from symbiosis to dysbiosis involves coordinated changes in microbial metabolism, virulence expression, and host and microbe interactions, ultimately leading to chronic inflammation and tissue destruction.[1,2] This transition involves functional shifts in microbial metabolism, virulence expression, and host microbe interactions.[3]

Central to this ecological shift is quorum sensing (QS), a bacterial communication system that enables population level regulation of gene expression. In periodontal biofilms, quorum sensing facilitates interspecies cooperation, biofilm maturation, and collective stress adaptation.[4,5] Quorum sensing, particularly AI-2 mediated interspecies signaling is a fundamental ecological regulator of the transition from symbiosis to dysbiosis in periodontal biofilms. By coordinating community wide metabolic activity, virulence expression, host and microbe interactions, QS drives functional dysbiosis without reliance on single-pathogen overgrowth.[6] Disruption of QS has therefore emerged as a therapeutic target.

Quorum quenching (QQ) refers to mechanisms that interfere with QS by degrading signaling molecules, inhibiting their synthesis, or blocking signal receptor interactions. Unlike antibiotics, QQ aims to attenuate pathogenic behavior without killing bacteria, thereby minimizing selective pressure for antimicrobial resistance and preserving beneficial microbial interactions.[7,8] Many

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of the *in vitro* studies presented in this review have been performed on monospecies biofilms, particularly with respect to dental caries, where *S. mutans* biofilms predominate. Periodontal QQ research is based on *P. gingivalis* or a consortium of bacteria including, amongst others, *P. gingivalis*, *T. forsythia* and *F. nucleatum*.[9]

Although QS inhibition has been widely explored, existing reviews tend to focus on molecular mechanisms or general anti biofilm effects. Notably absent is a synthesis that conceptualizes QQ as a tool for ecological modulation of periodontal biofilms. This scoping review addresses this gap by mapping how QQ strategies influence biofilm ecology, symbiosis, and dysbiosis in periodontal systems.

METHODS

This scoping review followed the PRISMA Extension for Scoping Reviews (PRISMA-ScR) guidelines.[10] Systematic searches were conducted in PubMed, Scopus, and Web of Science using combinations of the keywords: *quorum quenching*, *quorum sensing inhibition*, *periodontal bacteria*, *periodontal biofilm*, and *oral dysbiosis*.

Studies were included were published between 2016 and 2025, investigated quorum quenching mechanisms or agents, targeted periodontal bacteria or periodontal biofilm models, original research articles or mechanistic reviews in English. Studies focused exclusively on cariogenic biofilms or non oral systems were excluded. Extracted data included publication year, QQ strategy, targeted QS system, microbial context, experimental model, and ecological outcomes. Data were synthesized narratively.

RESULTS

A total of five studies met the inclusion criteria. Most studies employed in vitro multispecies biofilm models, while a smaller subset used animal models. Clinical studies were scarce.

Tabel 1. Data extracted from included studies

No	Author Year	QQ Strategy	Targeted QS Pathway	Model	Ecological Outcome
1	Polizzi et al. (2022)[11]	Natural QS inhibitors	AI-2	In vitro biofilm	Reduced virulence without bactericidal effects
2	Hashim et al. (2024)[12]	Polysaccharide based QQ	Interspecies QS	Subgingival biofilm	Suppressed biofilm maturation
3	Zhao et al. (2025)[13]	Enzymatic quorum quenching	Multiple QS signals	Polymicrobial biofilm	Disrupted pathogenic cooperation
4	Nagi et al. (2023)[4]	QS signal interference	AI-2	Multispecies biofilm	Reduced host inflammatory signaling
5	Alum et al. (2025)[14]	Plant-derived inhibitors	QS receptors	In vitro	Partial restoration of symbiotic

Across the included studies, QQ approaches were grouped into several major categories, including natural QS inhibitors, plant derived inhibitors, polysaccharide based QQ**,** enzymatic quorum quenching, and QS signal interference. The most frequently targeted pathways involved interspecies quorum sensing, particularly AI-2 associated signaling, reflecting the importance of community level communication in periodontal biofilm ecology.

Overall, QQ interventions were consistently associated with ecological outcomes indicative of biofilm modulation rather than eradication. Reported effects included reduced virulence expression without bactericidal activity, suppression of biofilm maturation, disruption of pathogenic cooperation in polymicrobial consortia, attenuation of host inflammatory signaling, and partial restoration toward a more symbiotic community profile.

DISCUSSION

This scoping review highlights quorum quenching (QQ) as a promising ecological approach to modulate periodontal biofilms, moving beyond traditional antimicrobial strategies. Quorum sensing (QS) is a conserved bacterial communication

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mechanism that regulates biofilm formation, virulence expression, and community behaviors in polymicrobial ecosystems, including the oral microbiome. QS signals such as autoinducer-2 (AI-2) serve as *universal interspecies language* that coordinates collective behavior's across diverse oral taxa, impacting biofilm structure and resilience in periodontal niches.[9,13]

QQ strategies that disrupt signaling pathways, degrade signaling molecules, or competitively inhibit receptors offer ecological advantages by attenuating pathogenic behavior without applying strong bactericidal pressures. Such anti virulence effects are attractive because they may reduce selective forces that drive resistance a major concern with conventional antiseptics and antibiotics in periodontal therapy.[4,15]

For example, the quorum quenching enzyme Aii20J has been shown to modulate in vitro periodontal biofilm formation by interfering with AHL like signaling without affecting total cell viability, indicating targeted disruption rather than broad spectrum killing.[4]

Periodontal disease progression is tightly linked to dysbiosis, where shifts from a symbiotic to a pathogenic consortium are driven by ecological pressures and host inflammation. QS mechanisms contribute to this transition by coordinating expression of virulence determinants, stress responses, and metabolic adaptability in biofilm communities.[9,15]

Elevated AI-2 levels have been associated with heightened periodontal inflammation and alveolar bone loss, suggesting QS signaling directly influences disease severity and host response.[16,17]

The ecological impact of QQ extends to host immune modulation. QS molecules themselves can interact with host immune pathways, influencing macrophage polarization and pro-inflammatory signaling cascades relevant to periodontal tissue destruction.[18] This highlights that QS interference may benefit periodontal health via both microbial coordination disruption and indirect modulation of host inflammatory responses.

Different QQ modalities produce varied ecological outcomes. Natural QS inhibitors derived from plant based compounds (e.g.,coumarins) have been demonstrated to inhibit biofilm maturation and AI-2 activity, while enzymatic and material based QQ approaches showed differential effects on community structure and virulence expression.[19]

Despite promising preclinical data, translational evidence remains limited. The majority of studies use in vitro biofilm models with short exposure periods and heterogeneous endpoints. Standardized frameworks for assessing QQ efficacy, including longitudinal community dynamics, host inflammatory readouts, and resilience to ecological disruptions are urgently needed to facilitate comparison and clinical translation.[15]

Future research should integrate high-resolution community profiling, systems level host response analyses, and well controlled clinical trials to validate QQ's potential as an adjunct in periodontal therapy. By shifting the therapeutic focus toward ecological modulation rather than microbial eradication, QQ may complement mechanical debridement and host modulating therapies, contributing to sustained periodontal health.[9,15]

CONCLUSION

Quorum quenching represents a novel, ecology based approach for managing periodontal biofilms by modulating microbial communication rather than eradicating bacteria. Evidence from the last decade indicates that QQ can attenuate dysbiotic behaviors while preserving symbiotic microbial functions. Framing quorum quenching as an ecological reprogramming strategy offers new perspectives for periodontal therapy and highlights the need for translational and clinical investigations.

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