REVIEW ARTICLE

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Bacterial Adhesion on Orthodontic Bracket Surfaces: Mechanisms, Contributing Factors, and Preventive Approaches Using Nano Particles and Herbal Extract

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Fixed orthodontic appliances are helpful in achieving optimal tooth positioning; however, their use imposes surfaces that are prone to bacterial adhesion and biofilm development. Biofilm formation can be detrimental to one's oral hygiene as it may lead to caries, gingivitis, or decalcification of enamel. We focus on the phenomenon of bacterial colonization on orthodontic brackets and discuss how some surface features like roughness, surface energy, and hydrophobicity promote the adhesion of microorganisms. Some dominant microorganisms involved in biofilm development, which later on are reinforced by the secretion of extracellular polymeric substances (EPS), are *Staphylococcus aureus* and *Streptococcus mutans*. This paper also analyzes the bacterial attachment using different approaches like microbial culture, quantitative PCR (qPCR), or scanning electron microscopy (SEM) and discusses their pros and cons. To reduce bacterial adhesion, preventive methods such as antibacterial coatings (e.g. AgNPs and TiO_2) and mouth antiseptics (e.g. chlorhexidine and chamomile extract) are proposed. Herbal extract especially Chamomile extract is equally as effective as chlorhexidine in the reduction of *S. aureus* colonies making it a better option due to its low side effects. New developments such as smart brackets with nanoparticle coating, and therapies using probiotics, are proving useful in reshaping oral bacteria by targeting pathogenic bacteria. Additional work is necessary on clinical trials over extended periods, standardizing the use of probiotics, and creating multifunctional coatings designed to strategically improve oral health during orthodontics treatment. This review highlights the need for enhanced interdisciplinary efforts to address the issues of bacterial adhesion and patient care in orthodontics.

Keywords: bacterial adhesion, orthodontic brackets, biofilm, nanoparticles, herbal extracts, oral hygiene

Introduction

Most orthodontic treatments are performed using fixed appliances attached directly to the teeth. Fixed orthodontic appliances are necessary for accurately positioning teeth. Brackets, arch wires, and additional components forming the fixed appliance mediate tooth movement, which occurs at the tooth-bracket interface.[1] Fixed orthodontic appliances are bonded to teeth through chemical or micromechanical adhesion, allowing orthodontists to achieve a much wider range of tooth movement compared to removable appliances.[2] The main components of fixed appliances (brackets, bands, ligatures, and arch wires) can reduce the physiological self-cleaning mechanisms by the tongue or cheeks, increase bacterial plaque retention, and alter the qualitative and quantitative bacterial population. In vivo studies show that elastomeric ligatures are less susceptible to plaque

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adhesion compared to stainless steel metallic ligatures.[3] Modern orthodontic brackets are made of three different materials: metal brackets (stainless steel, titanium, and cobalt chromium), plastic brackets, and ceramic brackets (monocrystalline and polycrystalline). Each material has its own benefits and limitations. Ideally, bracket materials should possess qualities such as biocompatibility in the oral environment, low cost, high modulus of elasticity, high corrosion resistance, non-magnetic properties, minimal friction during wire-bracket interaction, appropriate strength and hardness, resistance to staining and discoloration in the oral environment, resistance to plaque deposition, and aesthetic appeal. However, no contemporary material meets all these criteria.[4]

Orthodontic brackets and fixed appliances create new areas for plaque accumulation, promoting bacterial growth and inflammation. Fixed appliances contribute to increased oral microorganisms and, along with factors like diet, hygiene, and microbial acids, alter the oral microbiota.[5] Proper oral hygiene plays a crucial role in maintaining dental, gum, and overall supporting structure health during and after orthodontic treatment. Effective oral hygiene during orthodontic treatment includes brushing and cleaning teeth at least twice daily.[6,7] Additionally, antimicrobial mouthwashes can help reduce harmful bacterial growth. Periodontal health has been shown to improve permanently with stricter oral hygiene practices (brushing and supragingival scaling).[8]

Result and Disscusion

Bacterial Adhesion and Biofilm Formation on Orthodontic Surfaces

Microbial biofilms play a significant role in the development of oral diseases such as cavities and gum disease. Dental plaque (oral biofilm) is a complex mixture of microbes in a self-produced matrix that adheres primarily to teeth or gum tissue. Bacteria adhere to enamel by binding to the protein-rich pellicle. The oral microbiome, which includes up to 700 species of aerobic and anaerobic bacteria, can be resistant to various antimicrobials. Biofilms can also form on foreign objects like orthodontic appliances, which are more prone to bacterial buildup.[9]

Orthodontic appliances provide new surfaces suitable for oral bacterial colonization and biofilm formation, which can compromise oral hygiene and lead to enamel demineralization during treatment.[10] Bracket adhesion alters the tooth surface both physically and chemically. Demineralized enamel encourages the adhesion and growth of *Streptococcus mutans*. Polymer surfaces, such as those in composite resins used for bracket bonding, strongly support microbial adhesion and growth.[11] Organic acids from bacteria like *Streptococcus mutans* and *Streptococcus sobrinus* contribute to enamel demineralization, making bacterial adhesion to orthodontic materials a critical factor in this process during treatment.[12]

Areas around brackets accumulate more plaque than occlusal areas because these regions are most disrupted by arch wires and ligatures, making natural self-cleansing impossible. Difficult and inadequate oral hygiene, along with reduced ability to use dental floss, leads to plaque buildup and changes in oral flora, potentially causing gingivitis, enamel white spot lesions (WSL), and cavities.[13-15]

Adhesion is the first stage of biofilm development and involves complex interactions between bacteria and surface-specific factors. Free-floating bacteria move toward surfaces due to gravity, random motion, and possibly external agents aiding mobility. Initially, they adhere reversibly, then transition to stable, irreversible adhesion. This process is driven by non-specific forces, including long-range van der Waals forces, electrostatic interactions, and short-range acid-base interactions.[16,17] Surface properties like surface energy, hydrophobicity, roughness, and texture play a significant role in bacterial adhesion.[15, 18-20] As shown in **Table 1**, The process of bacterial colonization on tooth surfaces occurs in three main stages. The first stage is initial adhesion, where bacteria approach the tooth surface and attach temporarily through physicochemical interactions like van der Waals forces and electrostatic interactions. Next, in the irreversible adhesion stage, bacteria form stronger bonds with the tooth surface through specific adhesins and begin producing extracellular polymeric substances (EPS), which play a crucial role in biofilm formation.[21] In the third stage, biofilm maturation, bacteria proliferate and form complex three-dimensional structures protected by the EPS matrix. This structure provides protection against antimicrobial

agents and the host immune system, allowing bacteria to survive in unfavourable environmental conditions.[22]

The orthodontic biomaterials in the wires had the greatest attraction for and concentration in SS and NiTi wires. *S. aureus* exhibited the highest concentration, and *C. albicans* displayed the lowest concentration.[14] The non-shedding surfaces, including teeth, dentures, and dental implants, are prone to biofilm formation, which can lead to significant complications such as dental caries, periodontitis, and dental implant failure. The inflammation induced by biofilm on dentures, mucosal surfaces, and acrylic resins may modify local pH, nutrient availability, protein composition, and gingival crevicular fluid flow, subsequently altering microflora composition and potentially promoting unique bacterial adhesion patterns.[15] Oral health is seriously threatened by bacterial colonization of dental biomaterials, which also causes biofilm-related problems such infections, inflammation, and material deterioration.[20] Complex interactions between biomaterial characteristics like surface energy, roughness, and hydrophobicity and surface microbial components (such adhesins and fimbriae) govern the adhesion process.[16] In addition to stabilizing the biofilm, extracellular polymeric substances (EPS) create a barrier that protects it from the host immune system and antimicrobial agents.[23] To limit bacterial colonization and its clinical effects, effective solutions must be developed, which requires an understanding of these pathways.

The salivary provides attachment sites for pioneer bacteria like *Streptococcus sanguinis* and S*treptococcus gordonii.* Meanwhile, EPS is a key component of the biofilm matrix, composed of polysaccharides, proteins, and extracellular DNA. EPS plays a vital role in aiding bacterial adhesion to surfaces, stabilizing biofilm structure, and providing protection against environmental stresses, including antibiotics and host immune responses.[23] The biofilm matrix is essential for the initial attachment and adhesion strength of the biofilm to surfaces; it provides mechanical stability and acts as a barrier to antibacterial agents. Consequently, targeting the extracellular polymeric substances (EPS) may be a promising antibacterial approach. However, it is important to note that the composition of EPS varies depending on the microbial species involved, making it inherently heterogeneous, and is also influenced by environmental factors such as nutrient availability.[24]

Incorporating antibacterial agents into dental restorative materials can help combat biofilm formation by disrupting the EPS network and interfering with quorum sensing, especially through the gradual release of these agents over time.[25] Natural examples demonstrating such antibacterial properties include lotus leaves, shark skin, gecko skin, and cicada wings. Nonetheless, the formation of a protein pellicle on surfaces can reduce these effects by limiting direct contact between the antibacterial agents and the biofilm.[24]

Oral Bacteria Associated with Orthodontic Biofilms

As shown in **Table 2**, Several bacterial species are known to play significant roles in colonization and biofilm formation in the oral cavity. *Streptococcus mutans*, a Gram-positive bacterium, is the primary cause of dental cavities. It produces EPS through the enzyme glucosyltransferase, enabling the formation of strong biofilms resistant to acidic conditions.[22] Additionally, some *Lactobacillus* species can produce EPS that supports biofilm formation. Interestingly, certain *Lactobacillus* strains show probiotic potential by inhibiting *S. mutans* growth and reducing biofilm formation through biosurfactant production.[26] *Staphylococcus aureus*,

Table 1. Mechanisms of bacterial adhesion on orthodontic brackets

Stage	Process	Key Factors Involved
Initial Adhesion	Reversible attachment via physicochemical interactions	Van der Waals forces, electrostatic interactions
Irreversible Adhesion	Bacterial adhesins and EPS production strengthen attachment	Fimbriae, surface proteins, EPS matrix
Biofilm Maturation	3D structure formation with protective EPS	Quorum sensing, microbial cooperation

Table 2. Common oral bacteria in orthodontic biofilms

Bacterium	Role	Clinical Impact
Streptococcus mutans	Primary caries pathogen; produces EPS	Enamel demineralization, white spot lesions
Staphylococcus aureus	Forms antibiotic-resistant biofilms	Implant/bracket infections, gingivitis
Lactobacillus sp.	Acid-producing; some strains are probiotic	Caries progression or inhibition (species-dependent)

though better known as a nosocomial pathogen, can also form biofilms on tooth and implant surfaces, contributing to chronic infections that are difficult to treat due to antibiotic resistance.[21] Various biomaterials are employed in dentistry for a range of purposes. The development of biofilms on dental biomaterial surfaces is affected by factors such as surface roughness, surface energy, topography, and composition. *Streptococcus mutans* is the primary bacteria found on the surface of restorations, and its presence leads to an increase in surface roughness from 0.1 to 1 µm over the course of one month of biofilm buildup. To minimize biofilm formation and associated infections, it is recommended that the surface roughness of implants and other dental biomaterials be maintained below a threshold of 0.2 µm.[27]

Methods for Assessing Bacterial Adhesion

As summarized in **Table 3**, to analyzes the bacterial attachment using different approaches like microbial culture, quantitative PCR (qPCR), or scanning electron microscopy (SEM). Microbial culture method is simple, requires no advanced equipment, and can be applied to a wide range of bacteria. However, it has limitations, such as low sensitivity for hard-to-culture or biofilm-forming bacteria and the inability to distinguish between live and dead bacteria.[28] The biofilm model designs range from basic to advanced depending on the research objectives. Agar plate and microtiter assays are microbial culture models conducted in closed systems, which are inexpensive and easy to handle. In contrast, open-system microbial culture models are more complex and produce biofilms that more closely resemble natural dental plaque. The choice of model for a biofilm study is determined by factors such as growth conditions, the specific characteristics needed for the biofilm, and the goals of the investigation. [29]

The qPCR technique aids in clarifying the signalling pathways that pathogens utilize to initiate and grow biofilms. By examining the expression levels of genes involved in quorum sensing, adhesion, and extracellular matrix formation, researchers gain insights into the molecular processes that drive biofilm development. For instance, genes like Luxs, which play a key role in bacterial communication within biofilms, can be analyzed using qPCR to assess their activity under various environmental conditions or in the presence of antimicrobial substances. This information is crucial for identifying potential targets for intervention and understanding how environmental factors and interactions within the food matrix affect biofilm behaviour in real-world situations.[30]

qPCR is used to analyse gene expression related to bacterial adhesion and biofilm formation. The process involves RNA extraction from samples, cDNA synthesis, and amplification using primers specific to target genes. This method is highly sensitive and specific, allowing evaluation of gene expression under various environmental conditions. However, qPCR requires specialized equipment and reagents and cannot provide visual or morphological information about bacterial location on surfaces.[30]

SEM allows direct visualization of bacterial morphology and distribution on surfaces with high resolution. The process involves fixing samples with glutaraldehyde, dehydrating with ethanol, coating with gold, and observing under an electron microscope. SEM provides clear images of bacterial structure and quantity on surfaces and can help differentiate species based on cell shape. However, its drawbacks include complex sample preparation and the inability to observe live samples.[32]

The primary drawback of SEM involves the invasive process of preparing specimens. Due to the vacuum environment, organic samples must undergo initial fixation and dehydration. Additionally, non-conductive specimens need to be coated with a conductive material to prevent electron buildup. Conventional SEM images are two-

Table 3. Methods to assess bacterial adhesion

Method	Advantages	Limitations	Ref
Microbial Culture	Low cost, broad applicability	Cannot distinguish live/dead cells	[29]
Quantitative PCR (qPCR)	Detects specific bacterial genes	No morphological data; requires expertise	[30]
Scanning Electron Microscopy (SEM)	High-resolution imaging	Destructive sample preparation	[32,33]

dimensional, making it difficult to assess the depth of objects; however, acquiring three-dimensional information is achievable through digital reconstruction techniques.[33]

Preventive Approaches for Controlling Bacterial Adhesion

To reduce bacterial adhesion, preventive methods such as antibacterial coatings (e.g. AgNPs and TiO_2) and mouth antiseptics (e.g. chlorhexidine and chamomile extract) are proposed. In recent years, attention has increasingly focused on modifying orthodontic brackets, arch wires, and aligners through surface coatings. Materials like metal oxides, metals, and organic compounds have been used to create antibacterial coatings, reducing reliance on oral hygiene maintenance, decreasing surface roughness and bacterial adhesion, and improving the biocompatibility of orthodontic appliances. Several relevant reviews have been published, particularly introducing the application of antibacterial nanoparticles in orthodontic materials.[34]

Reactive oxygen species (ROS) cause oxidative stress. Nanoparticles (NPs) react with oxygen molecules to produce various ROS types. For example, titanium dioxide is a semiconductor activated by photons with wavelengths below about 385 nm (UVA), which undergo photocatalysis to allow free electron movement in the conduction band. Oxygen and water can capture charges and generate reactive oxygen species like superoxide anions and hydroxyl radicals, damaging cell walls and plasma membranes, leading to cell content leakage and bacterial death. Additionally, the generated ROS can diffuse into bacteria, attacking proteins and inhibiting certain plasmid activities that maintain normal bacterial morphology and physiological processes.[35]

Various metals and their compounds have been coated on fixed orthodontic appliances, including silver (Ag), titanium dioxide (TiO_2), zinc oxide (ZnO), zirconium oxide (ZrO_2), and titanium nitride (TN). TiO_2 is the most studied and used. Silver nanoparticles (Ag NPs) have become common in dentistry and orthodontics due to their high antibacterial activity. Research shows that without altering the physical and chemical properties of archwires, Ag NP-coated archwires significantly reduce the presence of *S. aureus* and *S. mutans*.[36] Lysozyme specifically degrades the cell walls of microorganisms without harming other tissues, and it has been applied to modify archwires with antibacterial properties. The composite arch wires (CAWs) coated with 40 g/L of lysozyme exhibited the most potent antibacterial activity against *S. aureus*.[34]

Bacterial plaque can adhere to materials used in fixed orthodontic treatment. Several studies report chemical control using mouthwashes to reduce bacterial strains. Bacterial plaque not only affects plaque buildup and cavity formation but also increases pathogenic colonies in biofilms directly linked to gingival and periodontal issues during months-long orthodontic treatment.[37] Herbal remedies that block quorum-sensing and EPS synthesis, including as neem (*Azadirachta indica*), green tea (EGCG), and chamomile (α-bisabolol), enhance these effects. In addition to improving antibacterial activity (**Table 4**), these synergistic strategies lower the likelihood of resistance, providing a long-term substitute for conventional antiseptics like chlorhexidine.[38]

Chlorhexidine (CHX) is generally biocompatible and used orally as an antiseptic mouthwash by dental practitioners and the public to prevent plaque and bacterial biofilm buildup.[39] CHX mouthwash is the most potent chemotherapeutic agent and gold standard for reducing *S. mutans* and oral biofilms. In dentistry, CHX products are available by prescription and include formulations like mouthwash, gel, chips, and varnish.[40] In dentistry, 0.12–0.2% CHX can be used pre- or post-surgery (if brushing is possible) or long-term (CHX chips). The recommended dose for maximum effect per use is 18–20 mg.[39]

Bacterial walls are negatively charged, and CHX is positively charged, creating mutual attraction. On bacterial

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Table 4. Comparative overview of antibacterial strategies in orthodontics

Method	Mechanism of Action	Applications in Orthodontics	Advantages	Limitations/Side Effects	Ref
Antibacterial Coatings (Silver Nanoparticles & TiO_2)	- Generates ROS (e.g., superoxide anions, hydroxyl radicals), damaging bacterial cell walls. - Disrupts bacterial proteins and plasmid activities. - Ag NPs exhibit strong antibacterial effects.	- Coating on brackets, archwires, aligners. - Reduces adhesion of S. mutans , S. aureus . - Lysozyme-coated archwires inhibit bacterial growth.	- Reduces biofilm formation. - Improves biocompatibility. - Long-lasting antibacterial effect.	- Potential cytotoxicity at high concentrations. - TiO_2 requires UV activation. - Cost and coating durability concerns	[34,35, 36, 46]
Antimicrobial Mouthwashes (Chlorhexidine - CHX)	- Cationic CHX binds to negatively charged bacterial walls, causing cell lysis. - Inhibits enzymes, leaks cytoplasmic components. - Prolonged release (8–12 hrs).	- 0.12–0.2% CHX mouthwash/gel for plaque control. - Prevents gingivitis during orthodontic treatment.	- Gold standard for antibacterial efficacy. - Broad-spectrum (antifungal, antiviral).	- Tooth staining, metallic taste. - Mucosal irritation. - Bacterial resistance over time	[37, 40, 41]
Herbal Mouthwashes (Chamomile Extract)	- α-Bisabolol (in chamomile oil) inhibits biofilm growth. - Apigenin disrupts bacterial nutrient transport. - Anti-inflammatory (blocks COX/LOX pathways).	- Alternative to CHX for reducing plaque and gingival bleeding. - Used in natural antibacterial formulations.	- Fewer side effects than CHX. - Antioxidant & anti-inflammatory benefits.	- Weaker immediate effect than CHX. - Limited long-term clinical studies.	[43, 44, 45]

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walls, phosphate-containing molecules strongly adsorb to CHX. CHX penetrates bacterial walls, damages cells, inhibits membrane-associated enzymes, and causes leakage of low-molecular-weight cytoplasmic components like potassium ions. This is a reversible bacteriostatic stage, but if CHX remains, antimicrobial action continues to the bactericidal stage, where cytoplasmic coagulation and precipitation occur, forming complexes like ATP and nucleic acids.[37]

Oral surfaces are negatively charged, so cationic CHX adheres well, effectively disrupting bacterial adhesion. Strong binding to oral surfaces allows prolonged retention in the mouth. CHX shows prolonged action and can be released over 8–12 hours in the mouth. CHX has antifungal, antimicrobial (anaerobic and aerobic), and antiviral (RNA, DNA, and lipophilic enveloped viruses) activity.[37,41]

Orthodontic appliance components that tend to retain and accumulate plaque, bacteria, or food debris can trigger gingivitis.[42] Chlorhexidine 0.2% mouthwash, as a common chemical antibacterial agent for gingivitis therapy, still has side effects like tooth staining, taste alterations (metallic taste), oral mucosa irritation, bacterial resistance, and corrosion of metal fixed orthodontic appliances.[40]

Research on alternative therapeutic antibacterial agents from natural sources with minimal side effects, such as plants, is ongoing. One herbal remedy with potential as an antibacterial agent is chamomile. Chamomile flowers (*Matricaria chamomilla* L.), native to Southern and Eastern Europe, contain essential oils, phenolics, flavonoids (apigenin, quercetin, patuletin, luteolin), and glycosides.[43]

The main component of chamomile flower essential oil is α-bisabolol, which has anti-inflammatory effects by inhibiting phospholipase A, cyclooxygenase, and lipoxygenase pathways. Additionally, α-bisabolol has been shown to be a key part of chamomile essential oil's antibacterial activity, inhibiting pathogenic bacteria and damaging cell membranes, thus inhibiting biofilm growth.[44,45]

Matricaria chamomilla L. flower extract can reduce dental biofilm (plaque) accumulation and gingival bleeding in gingivitis patients, likely due to the antibacterial and anti-inflammatory activity of phenolic compounds in chamomile, particularly the flavonoid apigenin.[45] The antibacterial mechanism of apigenin against S*taphylococcus aureus* involves hydroxyl groups in the flavonoid structure, altering organic components and nutrient transport, ultimately causing toxic effects on bacteria.[43]

New developments such as smart brackets with nanoparticle coating, and therapies using probiotics, are proving useful in reshaping oral bacteria by targeting pathogenic bacteria. Smart brackets with antimicrobial release and probiotic use or microbiome modification strategies are recent research on bacterial adhesion on orthodontic bracket surfaces. Orthodontic treatment with fixed appliances often poses oral hygiene challenges due to increased colonization of pathogenic bacteria like *Streptococcus mutans*. To address this, smart brackets coated with antimicrobial materials like silver nanoparticles, titanium dioxide (TiO_2), and zinc oxide (ZnO) have been developed. These materials are designed to locally suppress microorganism growth without causing systemic resistance as with antibiotics.[46-48]

Bracket modification with silver nanoparticles can significantly inhibit *S. mutans* and *S. sobrinus* adhesion in vitro.[46] Additionally, nitrogen-doped titanium dioxide coatings significantly reduce bacterial colony counts within 90 days. TiO_2 has greater antibacterial and anti-adhesion effects compared to ZnO.[47,48] Combinations like Ag-ZnO, CuO-ZnO, and Ag-CuO in orthodontic brackets have shown enhanced effects in reducing *S. mutans* counts, with CuO-ZnO exhibiting the highest antibacterial activity.[49]

Probiotics as adjuvant agents in maintaining oral health during orthodontic treatment have gained widespread attention. Probiotics balance the oral microbiome, reduce pathogenic bacteria, and promote beneficial bacterial colonization. A systematic review and meta-analysis show that probiotics significantly improve periodontal treatment outcomes when used alongside conventional therapy.[50]

Consuming probiotic yogurt for six weeks reduces *S. mutans* and *Lactobacillus* counts and improves plaque and gingival indices in orthodontic patients.[51] These results align with studies using Lactobacillus reuteri tablets, which, within 14 days, reduce plaque index, papillary bleeding, and periodontopathogen Porphyromonas pasteri counts.[52] Probiotic use during orthodontic treatment may reduce gingivitis and cavity risks, though results depend on probiotic type and duration.[53]

Conclusion

Conventional small-sized, simply designed brackets can minimize bacterial colonization but still pose S. aureus retention risks, which can also be mitigated with 100%

chamomile flower extract, proven to have bactericidal effects. A solution made entirely from chamomile flower extract could serve as a different antibacterial treatment for people using fixed orthodontic appliances, showing effectiveness like that of 0. 2% chlorhexidine mouthwash in decreasing *S. aureus* populations on orthodontic brackets. Chamomile extract should be developed as an alternative antibacterial agent against *S. aureus* to support oral hygiene maintenance in fixed orthodontic appliance users. Future research suggestions include long-term clinical trials to assess human efficacy and safety, biocompatibility evaluations to avoid local or systemic nanoparticle side effects, studies on coating durability under oral conditions like pH and friction, and multifunctional coating development.

Authors' Contribution

S: Conceptualization, methodology, literature search, data curation, formal analysis, and writing—original draft. N: Investigation, validation, and writing—review and editing. FS: Supervision and writing—review and editing. All authors have read and approved the final version of the manuscript.

Conflict of Interest

The authors declare no competing interests.

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