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Oral Biofilm and Oral Disease, a Narrative Review

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Abstract

Background: Stagnated Oral Biofilm OB) is responsible for most oral pathology. Stagnation induces change n the microbes that allow dental decay and gum disease. ***Aim:*** This contribution appraises various OB's and their derived morbidity, caries and periodontitis. ***Discussion:*** Fundamental changes of OB that start caries and gum disease, and methods of OB control are reviewed. ***Conclusion:*** Not all biofilms are the same. Because undisturbed OB causes caries and gum disease OB removal is essential for prevention of tooth-decay and oral soft tissue morbidity.

Introduction

Humans are born with sterile mouths which rapidly become colonized after birth with resident microbes. Colonies and plaques of mixed bacteria inhabit human mouths for the rest of the persons life. The oral microbiota settles on all surfaces in the mouth, on both hard and soft tissues. The soft tissues include all the oral mucosae, hard- and soft-palate, the lining cheek alveolar mucosae, the dorsum and ventral of the tongue, the tonsillar fauces and upper pharynx, the floor of the mouth and linings of the tongues. Oral-biofilm ***(O-B) exists in planktonic or sessile forms***. While saliva distributes the planktonic microbes to all the soft tissues mentioned, the sessile forms adhere mainly to the teeth, by adhering microbial colonies which grow in the occlusal pits and fissures and other areas of diminished saliva irrigation. Biofilms are surface-associated communities, whereas saliva contains planktonic or dispersed organisms. Shedding mucosal surfaces and non-shedding tooth surfaces support different ecologies. Sessile areas are loci of microbial stagnation, and are located inter-proximally and cervically around all erupted teeth. Because the microbes tend to grow in amorphous plaques, the colloquial term used interchangeably for oral biofilm is referred to as 'Plaque.'

Abbreviations

OB =Oral Biofilm: TBT =Tooth Brushing Technique.

Aim

This narrative appraisal reviews and focuses on the Oral-Biome, its progress, development, and indicates why the two major clinical manifestation of derived deleterious effects on teeth, namely caries and gum disease, obtains, and also summarizes why some harmful systemic conditions implicates OB.

An internet searches were done on Google Scholar, CiteFactor, Academic Resource Index (ResearchBib), Scilit (Scientific Literature), PubMed, and Research Gate, with key words, oral biofilm, oral hygiene, tooth brushing, caries, gum diseases, periodontitis and systemic diseases and Oral Biome. The authors discretionary judgments selected relevant fundamental publications relevant to this narrative appraisal.

Composition and Maturation of Oral Biofilms

Not all O-B's are the same. Oral biofilms are ionic dynamic multispecies communities embedded in an extracellular matrix. They may include bacteria, OB acts as an ionic exchange gradient in which activity is increased with increased acidic ions. Because OB is not easily visible most people are unaware or ignorant about the potential pathogenicity of OB.

O-B starts as a pilot ecosystem after one day of stagnation: Generally the early O-B consists of non-motile, Gram-Positive bacteria, which are mainly aerobic, produce acids, enzymes, exotoxins, and form bacterial plaques as colonies bonded together with extra-cellular polysaccharides, formed by microbial glucosyl-transferases using carbohydrates, (mono-saccharides, fructose, glucose, galactose, and di-saccharides, sucrose, maltose, lactose). Early colonization is spatially and temporarily available, and although not totally uniform as described, the bacteria causing decalcification and subsequent caries are a selection of acidogenic and aciduric communities, that thrive on frequent fermentable carbohydrate nutrients. These bacteria are proficient at lowering plaque p-H for tooth demineralization, mainly by decalcification, and producing microcavities that are the start of tooth decay. The putative bacteria include: Streptococci and Actinomyces, Staphylococci, Fusobacteria, Lactobacilli, and Leptothricia, Rothia dentocariosa [3-6].

Intermediate O-B forms over a period of between 2 to 14 days stagnation. The pilot-OB transforms to a mix of existing motile and non-motile microbes, combined with Gram-negative and Gram-positive species of bacteria, which produce endotoxins. These microbes induce soft tissue inflammation. A main constituent is the 'corn-cob' bacterium like Corynebacterium matruchotii but also Streptococcus mutans, and Streptococcus gordonii. Coren-cob structures are spatially organized consortia of duplicated Streptococcus species [3, 4].

After 14 days a mature climax-OB shows mainly anaerobic, motile, Gram-Negative Bacteria, that produce endotoxins, enzymes (like collagenases), various antigens, and mitogens that cause inflammation, soft tissue disruption and bone resorption. All these results in a chronic, painless progressive destruction of dento-gingival fibers, the periodontal ligament attachments from tooth to bone, and the eventual loss of the tooth. Periodontitis will develop in a host susceptible when a dysbiosis obtains, such as severe periodontitis when associated with Herpes viral infection. The microbes involved include: *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans*, *Prevotella intermedia*, *Tanarella forsythia*, *Campylobacter rectus*, *Fusobacterium nucleatum* and *various spirochaetes* [5, 6].

Pathogenic Mechanisms in Dental Caries and Periodontal Disease. Modes of attack on teeth and gums

The microbial constituents change when allowed to stagnate, and all stagnated O-B have three major outcomes on human tissues. Dental caries, periodontitis and calculus all of which have similar starts but different distinct pathological pathways. Calculus is calcified plaque that retains OB.

First on the morbidity of dental hard tissues: Gram-positive microbes like Streptococcus mutans, adhere to tooth surfaces have the ability to make acid to decalcify dental hydroxy-apatite at the critical pH of decalcification; the critical pH is not fixed and is moderated by the amount of calcium, phosphate, fluoride, saliva in and on the tooth. Starting at pH-6.6 and consistently and reliably at pH5.5 or below demineralization will occur. Micro-cavitation starts and may be remineralized, be worn away as erosion, or persist as an enlarging cavity in which the demineralized material is progressively attacked by the microbes leading to the formation of tooth caries. Caries forms in locations of stagnation like in the pits and fissures on the occlusal surfaces, interdentally under undisturbed inter-dental contact points, and on cervical margins. Contemporary clinical detection for diagnosis of dental caries is by visual-tactile assessment, with a ball-ended probe and judicious validated radiography [5-8]. See figure 1.

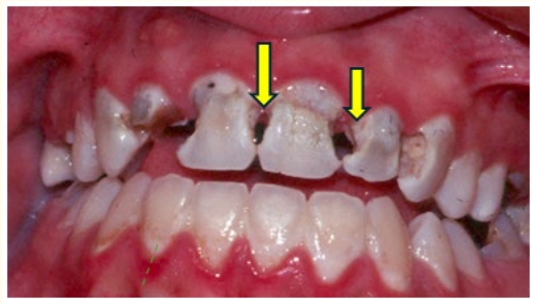

Figure 1: This shows interproximal tooth decay formation (yellow arrows) on the upper incisors, and stagnated O-B accumulation on the cervical margins of the teeth. The gingivae show signs of inflammation and the decay forms with after OB causes demineralization on the interdental areas of the upper canines and incisors.

Second, stagnated O-B can be calcified to form calculus, or tartar. This happens when a continuous supply of saliva allows alkaline precipitation of calcium into the O-B, and calculus tends to form on the lingual side of lower incisors from being opposite the openings of the sub-mandibular, and sublingual salivary glands, as does calculus form on the buccal side of upper molars being opposite the opening of the parotid salivary glands. Supragingival calculus mineralizes mainly from saliva, whereas subgingival calculus is influenced by gingival crevicular fluid [24]. Figure 2 and Figure 3.

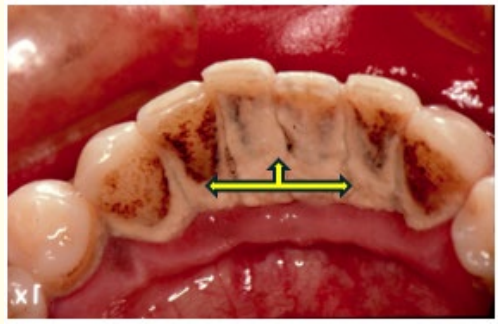

Figure 2: The lingual view of calculus (Arrow) accumulation on the anterior mandibular teeth. Calculus is mineralized OB and can have a loose or strong bond to tooth surfaces. The brown stain may be associated with extrinsic staining from diet, tobacco or other sources. Calculus grows over time, deriving its calcium and other ions from the saliva. Calculus can vary in hardness and color, may include microbes, and attaches to the tooth surface both above and below the gum margins.

Third stagnation also changes the ecosystem from a pilot and intermediate ecosystem to a climax community O-B that produces toxins, acids, enzymes, antigens and mitogens which slowly and painlessly destroys the soft tissues as gingivitis or periodontitis. Periodontitis may be mild, moderate or severe, and is now classified by Type, Stage and Grade. Figure 4, Figure 5 and Figure 6. All periodontitis may start as gingivitis, but most gingivitis does not progress to periodontitis. Progression depends on host susceptibility and risk modifiers like smoking, diabetes, immune-status or viral infections. Gingivitis is localized to the gingival sulcus, whereas periodontitis destroys the attaching periodontal ligament that attaches the tooth-root to the bony alveolar bones in the maxilla and mandible. Gingivitis is managed with individualized self-performed plaque control, and by regular professional debridement [9-12].

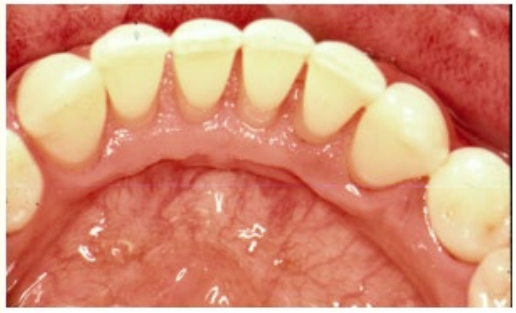

Figure 3: The same set of teeth as in Figure 2, but with removal of the adhering calculus. Hand sickle scalers and ultra- sonic instruments were used to remove the calculus, followed by manual scaling and polishing with rotatory brush and prophylaxis paste.

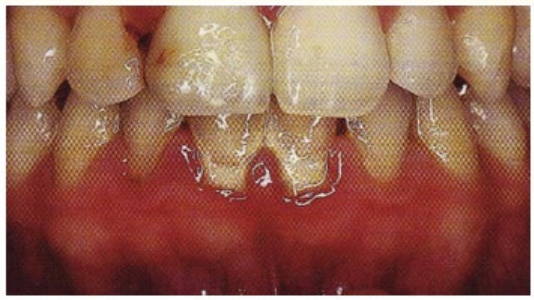

Figure 4: Teeth showing O-B and gingivitis. There was bleeding on probing between all the teeth shown. There is marginal gingival oedema, colorchanges, swelling , inflammation and probing depths that do not exceed 3mm.

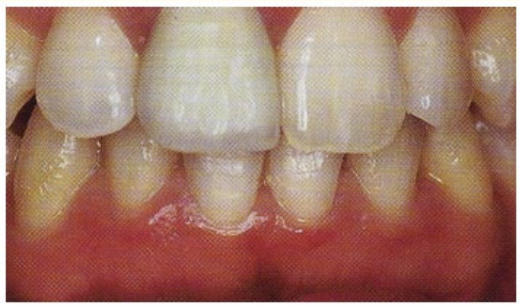

Figure 5: The same mouth as in Figure 4 after periodontal prophylactic debridemet with scaling (removal of extrinsic tooth material, (calculus, O-B, stain, debris), polishing and stringent individualized self-performed plaque control with oral hygiene practices.

Periodontitis is so named once the inflammtory and desutructive process derived from O-B causes lossof periodontal ligament attachment to alveolar bone. Different types of periodontitis exist, namely Necrotizing, ulceromembranous ginivitis, Chronic periodontitis, and Aggressive periodontitis. The disease is assessed by stage from Stage-I, to Stage-IV, and Graded as A (slow), B (moderate) and C (Rapid). Different Stages and Grades may be present simultaneously in the same mouth. Precise clinical signs and presentaion variables (recession, mobility, graded probing depths [over 3mm], bleeding on probing, furcal-involvements, tooth vitality etc) are described elsewhere [9-11]. Ignored and unchecked the disease pogresses until the teeth are lost. Periodontitis is identified by charcteristic clinical presentation with gingival bleeding on probing, attachment and bone loss patterns, excluding non-periodontic causes. Clinical findings are supplemented by full mouth radiograhy, but radiography does not reveal OB, but possibly mineralized calculus, which is calcified OB [9-11].

The intact relatively healthy cervical dento-gingival fibres stop at pocket-probing depths of 3mm, and any loss beyondf these fibres involved the periodontal ligament, and called periodontitis. Total loss of attachment includes the sum of measures of recession and pocket-probing depth.

Periodontitis is typified by bacterial-associated, host-interceded inflammation that renders a loss of periodontal attachment. The pathophysiology of the disease induces inflammatory biochemical pathways, that results in activation of host-derived enzymes (like proteinases/collagenases) which destroy the marginal periodontal ligament fibers, with simultaneous apical migration of the junctional epithelium, that allows apical spread of OB down the root surface by microbially-implicated host-mediated inflammation and renders a loss of periodontal attachment. The pathophysiology of periodontitis is characterized with reactive biochemical molecular pathways, that moderate activation of host-derived proteinases which induce destruction of marginal periodontal ligament fibers, apical migration of the junctional epithelium, and facilitates apical spread of OB along the root surface [10-11]. Figure 6-A & 6-B.

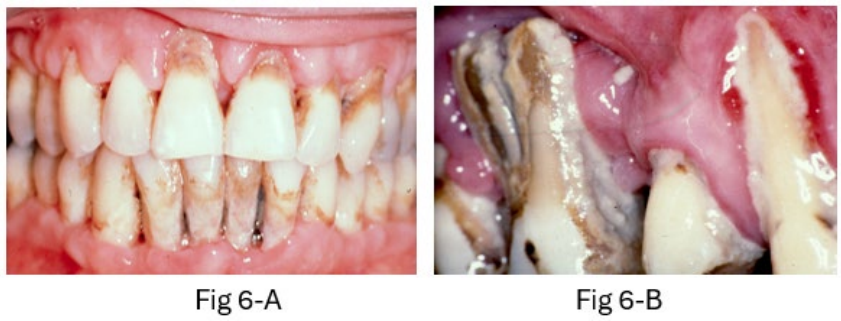

Figure 6A: Clinical presentation of Periodontitis in an adult. Note OB accumulationalong the gingival margins, and apical recession of the gum margins with root exposrue. ***Figure 6-B:*** Advanced progressive destruction from staganted O-B. Stage-IV and Grade-C. Tissue breakdown is substantially host mediated response to dysbiotic OB. The bacterial plaques destroy the peiodontal ligament and supporting alveolar bone. The roots become exposed, and the adjacent gingiva becomes inflamed leading to soft tissue necrosis, with eventual loss of the affected teeth.

Periodontitis is measured by losses of attatchment mainly by measuring probing pocket depths clinically; periodontitis has been assessed with a PSR score (Periodontal Score Record/Rating) derived from mild (pocket-probing depths 3-5mm), moderate (pocket-probing depths 5-7 mm) or severe (pocket-probing depths 7+mm). Currently (2026) Priodontitis is assessed and classified as to its Form, Stage and Grade, with attachment loss, pocket probing from the cemento-enamel junction to the periodontal ligament attachment to the tooth-root [9-13].

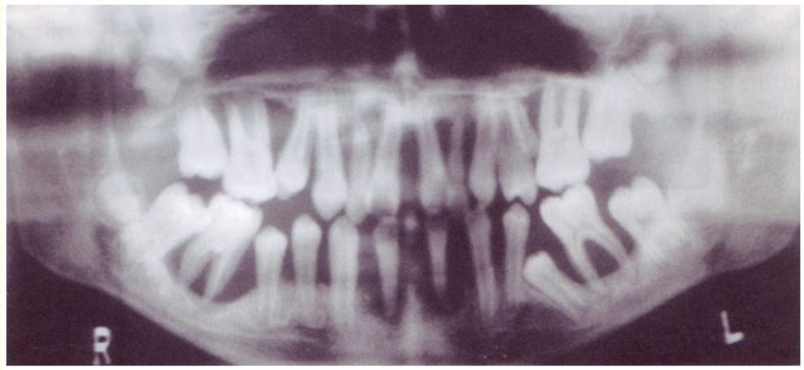

Figure 7: A pan-oral radiograph of a case of Aggressive Peiofontitis in an 18 year-old male teen-ager, at Stage-IV, and Grade-C, Note the extensive loss of alveolar bone especially around the molars and incisors. OB does is rarely visible in radiaodgraphs.

Periodontitis is treated with detoxifying supra and sub-gingival instrumentation and conditioning; root-plaining (removal of infected or affected intrinsic tooth material from microbes, including cementum, dentine or rarely enamel). Therapy may be supplemented by use of anti-microbials and/or courses of antibiotics. Selective muco-gingival surgery for resective or regenerative purposes, to eliminate residual pockets and to re-establish stable periodontal ligament to alveolar supporting bone is determined by risk assessment for success. Without regular maintenance, monitoring and effective oral hygiene, treated cases tend to recur. Periodontal treatment demands disciplined home-care oral-hygiene support and implementation by the patient, and regular professional attendances for exam, remotivating, monitoring and maintenance [10-13]. Figures 8-A with 8-B, and figure 9-A and 9-B

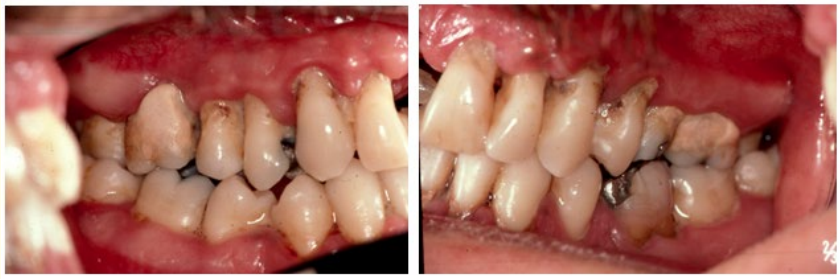

Figure 8-A and Figure 8-B: This shows the dentition of a dentate mouth with chronic periodontitis, Stages III and IV, Grade 3. Figure 8-A on the left buccal view, and Figure 8-B on the right buccal view. The clinical records revealed bleeding on probing, varying levels of mobility, positive thru-and-thru furcal involvements on #17 and #28 (FDI tooth numbering system) with radiographic evidence of alveolar bone loss on all teeth. There is O-B on the cervical and interproximal areas, with calculus formation. Probing depth ranged between 4->7mm +; (Radiographs are not shown as this review focuses on OB mainly and this case is presented solely for illustration of OB morbidity).

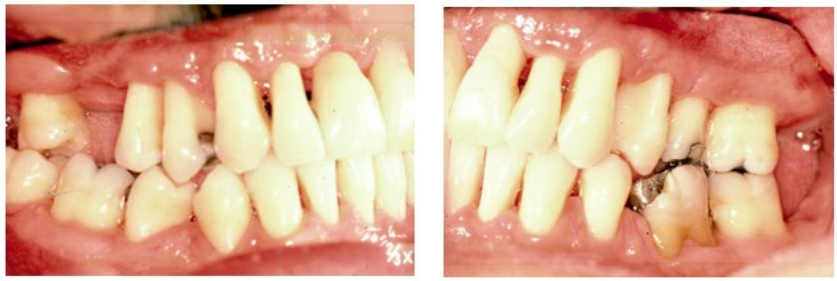

Fig 9-A Fig 9-B

Figure 9-A and 9-B: The same mouth six months post-operative as in Figure 8. Note tooth #16 and # 17 (FDI tooth numbering systemy) were were deemed unslvagable and removed. The gingiva are not inflamed; Oral hygiene for plaque control is near perfect. Strict home-care oral hygiene was successful and full mucogingival surgery was done. The residual pocketing was minimal (all below 3mm), and the teeth appear longer after healing with healthy gingival attachment post-operatively. This dentition and gingiva are stable; with prescribed regular (3-4 monthly) professional and daily home-care oral-hygiene removal of OB, recurrence is unlikely and should serve the patient well [9-13].

Discussion

The commonest, ubiquitous and most ancient disease affecting human mouths is tooth decay and gum disease. Tooth decay was found in skulls of archaeological humans dating back to neolithic times. Caries and gum disease are still prevalent in all societies in the 21st century. The pragmatic, biological and clinical premise is made, that because stagnated OB is cause-related to development of both dental caries and gum disease, total OB removal or its' disruption, will prevent the initiation of the diseases [18].

Prevention of periodontitis derives mainly from poor oral hygiene affecting predisposed subjects to the disease. People with compromised immunity are particularly vulnerable to developing periodontitis, but the precipitating factor in these cases is frequently neglected oral hygiene and stagnating OB. Systemic immunity from viral infections (like Herpes or HIV), can allow aggressive periodontitis to develop [18].

With caries, other multiple different influences impact development of tooth decay. Pre- eruption and post eruption dietary factors influence decay formation. Diet, access to care and/or informed advice, absence of sugar in the diet, fluoride exposure, salivary function, smoking, diabetes and other social influencing determinants all function on the prevalence of onset of decay. Different social values mores habits and practices in communities all impact decay development. For example, the ubiquitous sugar treats rewards for all children in the United Kingdom, combined with poor oral hygiene education, resulted in an epidemic of tooth decay from 1946 to 1956. The prevalence of tooth decay was significantly moderated by the introduction of comprehensive UK National Health Dental services after 1946, World-War II. In most advance societies (those with contemporary health care systems, and education facilities that finish with high-school Standard Ten) personal hygiene is taught with various methods for OB removal are introduced and advocated. Oral hygiene helps enormously to moderate both caries and periodontal disease, and while OB pathogenesis is the major putative factor, there are many unknown factors impacting these oral morbidities [5, 6, 9, 17, 18].

Brushing, and the ***use of dental floss*** and tooth picks are the most common; but oral irrigation and modifying the enamel critical pH are also major approaches used.

To remove or control OB, ***various brushing techniques*** have been described: Horizontal-Scrub; Vertical Leonard; Physiologic/ Smith-Bell; Circular/Fones; Rolling Stroke; Charters' Modified Rolling stroke; Stillman's Modified Rolling stroke; Modified Bass Rolling stroke; and Circular scrub. These techniques are all partially effective but not evidence equal; twice daily brushing with fluoride tooth

paste is effective, but personalized individual instruction to maximize OB control is desirable, and diligent removal of OB is more important than the technique uses [12, 14, 17, 18].

Battery/electric powered toothbrushes have been introduced for OB control. Figure 10.

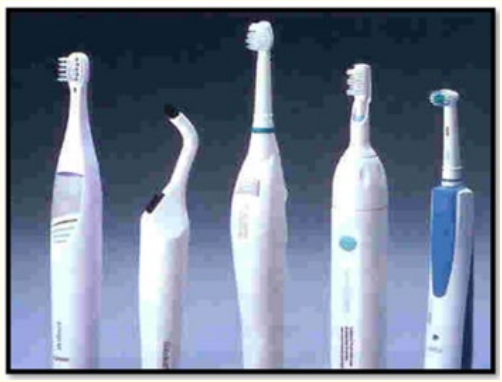

Figure 10: Examples of popular rechargeable power tooth brushes. Many types of vibrating and/or rotating brush-types of battery assisted tooth brushes are available. The handles are enlarged for the batteries and facilitate holding grips.

Oral irrigators to remove OB are available. The Water-Pik or Interplak are examples of these devices. Powered oral liquid irrigation is an adjunct for brushing and individualized interdental cleaning for fixed prostheses [15, 16]. Figure 11.

Figure 11: An example of an Oral irrigator. Oral irrigators are used to remove OB. The apparatus has a tank for the liquid and a hand-held narrow, mobile hollow small tube-like device, that spurts the liquid onto focused areas to remove OB. The sprayed water pressure is changeable with a pressure setting scale. The ejected stream of sprayed liquid accumulates in the mouth and is expectorated [23].

Interdental flossing is used to disrupt interproximal OB. Dental-floss is traditionally and ubiquitously advised as a regular part of home-care oral hygiene. Embrasure anatomy, the patients' dexterity and preference for other interdental brushes all can influence the success of interproximal OB removal. Evidence for consistent clinical success is varied [15, 16, 23]. Figure 12.

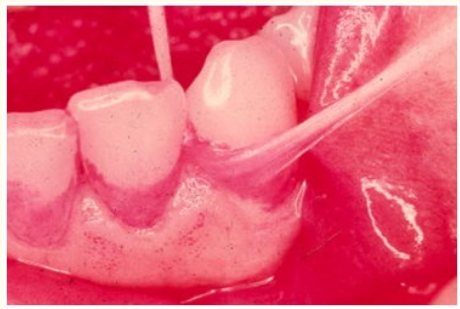

Figure 12: Demonstration of dental-floss use in an interproximal area. Use of dental floss successfully disrupts and removes OB from interproximal areas.

Exposing OB Plaque

Plaque control is sustained by risk-based professional assessment, (advice, motivation and prophylaxis procedures), with skilled regular home care. Sessile OB is virtually colorless on teeth in the mouth. Without plaque disclosure OB is not easily identified for removal. To show up sessile OB, the bacterial plaques can be stained with a plaque-disclosing agent, like erythrosine or toluidine blue. It shows up supra-gingival OB, as the dye, that is absorbed and concentrated in the plaque, yet far less absorbed by the tooth. Plaque disclosing agents do not characterize sub-gingival OB. Figure 13.

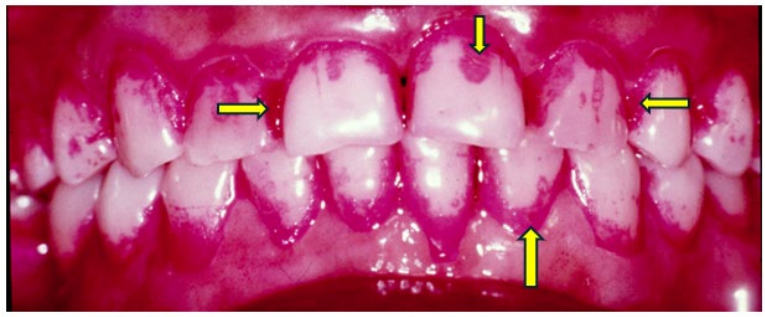

Figure 13: OB-disclosed with dye on all the teeth. Note how the sessile OB accumulates in those areas which allow stagnation, namely the interdental area and cervical regions. Arrows. Not shown are the pits and fissures on occlusal surfaces.

Care of the full dentition involves multimodal care and oral hygiene instructions, practices which include professional prophylaxis of scaling, polishing, fissure sealants and topical application of fluoride.) and home care by the patient using TBT brushing, floss and irrigation. Professional visits depend on clinical risk-based judgement calls of relapse, and is not uniform; some patients may need to attend once a year, yet others with advanced gum disease, every few months. Yet, there is no best method for OB removal, but success depends on the proficiency used rather than the method. Use of fluoride toothpaste, effective brushing, with adherence to individualized interdental cleaning techniques, strongly influence positive outcomes [14-17].

Use of Eufluoride at 1ppm (One part per million; 1mg/Liter) in potable community drinking water produces CaF (Calcium-Fluoride) which ionizes at a much lower critical pH than non- fluoridated tooth hydroxyapatite. The recommended 0.7 mg/L is the current USA recommended guideline, and is jurisdiction-specific. The critical pH for decalcification of non-fluoridated hydroxyapatite dental

crystals can start at pH 6.5 and definitely at pH 5. Consequently, any acidogenic diet is less liable to start decalcification of teeth with CaF integrated into the hydroxyapatite dental hard tissues than in non-fluoridated dentitions. The reduced anticaries effects are topical through reduced demineralization and enhanced remineralization. Many epidemiological studies reaffirm that potable fluoridated municipal drinking water significantly reduces the prevalence of caries [24-27].

Oral-Systemic Considerations and Concluding remarks

Oral morbidity from biofilm dysbiosis may contribute to host-mediated tissue injury. OB not only may sustain microbes which are capable of attacking teeth, but also can harbor microbes which possibly impact the development of serious systemic disease remote from the mouth [15].

For example, Helicobacter pylori may survive orally and can seed the gastric contents of vulnerable people to develop gastric ulcers [28].

Also, those who have suffered Rheumatic fever and have compromised cardiac valves, may be susceptible to Bacterial Valvular Endocarditis from seeding oral microbes like Viridans group of Streptococci that are introduced into the blood stream during any dental operative procedures that result in oral bleeding [28-30].

People may be susceptible to septic shock, if their endotoxin level is supplemented from existing concomitant chronic periodontitis [31].

The resident OB keeps the ecosystem in balance, in that it inhibits overgrowth of oral fungi like Candida albicans. Often, in some patients taking antibiotics, they disrupt their oral ecosystem and gut biome, and fungal overgrowth as Candidiasis manifests. Another manifestation of a disrupted OB is the development of a black hairy tongue. Although these conditions have multiple aetiologies, disruption of the OB is involved with both [32-33].

Conclusion

Irrespective of which oral hygiene device is used for home care, due diligence to scrupulous oral hygiene home-care cleaning increases long-term success. O-B control with home care and supportive periodontal-care, with risk appropriate periods, for selected professional prophylaxis will improve oral health benefits, and decrease any unwanted morbidity derived from poor oral hygiene [34].

Authors statement

Conflict of Interest

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

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None.

Data Availability Statement

Not applicable.

Ethical Statement

The project did not meet the definition of human subject research under the purview of the IRB according to academic regulations and consequently is exempt.

Informed Consent Statement

Informed consent was taken for this study.

Authors' Contributions

The author is the sole writer and accepts full responsibility for the content.

References

1. Dominguez-Bello MG., et al. "Delivery mode shapes the acquisition and structure of the initial microbiota across multiple body habitats in newborns". Proceedings of the National Academy of Sciences of the United States of America 107.26 (2010): 11971-11975.
2. Marsh PD. "Microbial ecology of dental plaque and its significance in health and disease". Advances in Dental Research 8.2 (1994): 263-271.
3. Kolenbrander PE., et al. "Oral multispecies biofilm development and the key role of cell-cell distance". Nature Reviews Microbiology 8.7 (2010): 471-480.
4. Mark Welch JL., et al. "Biogeography of a human oral microbiome at the micron scale". Proceedings of the National Academy of Sciences of the United States of America 113.6 (2016): E791-E800.
5. Touyz LZG. "The Pathophysiology of Oral Biofilms and its relation to Initial Gum Disease and Caries". Journal of Dental and Oral Disorders Therapy 5.4 (2017): 1-6.
6. Takahashi N and Nyvad B. "The role of bacteria in the caries process: ecological perspectives". Journal of Dental Research 90.3 (2011): 294-303.
7. Pitts NB., et al. "Dental caries". Nature Reviews Disease Primers 3 (2017): 17030.
8. Dawes C. "What is the critical pH and why does a tooth dissolve in acid?". Journal of the Canadian Dental Association 69.11 (2003): 722-724.
9. Pitts NB and Ekstrand KR. "International Caries Detection and Assessment System (ICDAS) and its International Caries Classification and Management System (ICCMS): methods for staging the caries process and enabling dentists to manage caries". Community Dentistry and Oral Epidemiology 41.1 (2013): e41-e52.
10. Chapple ILC., et al. "Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium: consensus report of workgroup 1 of the 2017 World Workshop". Journal of Clinical Periodontology 45.Suppl 20 (2018): S68-S77.
11. Papapanou PN., et al. "Periodontitis: consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions". Journal of Clinical Periodontology 45.Suppl 20 (2018): S162-S170.
12. Tonetti MS, Greenwell H and Kornman KS. "Staging and grading of periodontitis: framework and proposal of a new classification and case definition". Journal of Clinical Periodontology 45.Suppl 20 (2018): S149-S161.
13. Sanz M., et al. "Treatment of stage I-III periodontitis: the EFP S3 level clinical practice guideline". Journal of Clinical Periodontology 47.Suppl 22 (2020): 4-60.
14. Herrera D., et al. "Treatment of stage IV periodontitis: the EFP S3 level clinical practice guideline". Journal of Clinical Periodontology 49.Suppl 24 (2022): 4-71.
15. Kant K., et al. "A literature review on periodontitis and systemic diseases". International Journal of Periodontology and Implantology 8.2 (2023): 94-96.
16. Yaacob M., et al. "Powered versus manual toothbrushing for oral health". Cochrane Database of Systematic Reviews 2014.6 (2014): CD002281.
17. Worthington HV., et al. "Home use of interdental cleaning devices, in addition to toothbrushing, for preventing and controlling periodontal diseases and dental caries". Cochrane Database of Systematic Reviews 4.4 (2019): CD012018.
18. Sälzer S., et al. "Efficacy of inter-dental mechanical plaque control in managing gingivitis - a meta-review". Journal of Clinical Periodontology 42.Suppl 16 (2015): S92-S105.

19. Touyz LZG. "After Centuries of Dentistry. Prevention Remains Best". Scientific Archives of Dental Sciences 7.5 (2024): 06-11.
20. Jepsen S., et al. "Prevention and control of dental caries and periodontal diseases at individual and population level: consensus report of group 3 of the joint EFP/ORCA workshop". Journal of Clinical Periodontology 44.Suppl 18 (2017): S85-S93.
21. Kamra S., et al. "Oral Hygiene Instructions With Plaque-Disclosing Agents to Improve Self-Performed Dental Plaque Control: A Case Report". Cureus 16.10 (2024): e72205.
22. Touyz LZG. "What method is best for removal of Oral Biofilm? A commentary". PriMera Scientific Medicine and Public Health 9.1 (2026): 13-17.
23. Centrix. "Expose tablet and applicator". Centrix Dental (2026). https://www.centrixdental.com/
24. Mayo Clinic Staff. "Dental floss vs. water flosser: Which is better?". Mayo Clinic (2025).
25. US Department of Health and Human Services Federal Panel on Community Water Fluoridation. "U.S. Public Health Service recommendation for fluoride concentration in drinking water for the prevention of dental caries". Public Health Reports 130.4 (2015): 318-331.
26. Iheozor-Ejiofor Z., et al. "Water fluoridation for the prevention of dental caries". Cochrane Database of Systematic Reviews 10.10 (2024): CD010856.
27. Touyz LZG and Nassani LM. "Does fluoridated municipal water reduce prevalence of tooth caries? Fluoridated water DMFT compared with Non-fluoridated DMFT in the greater Montreal area. A clinical epidemiological study". World Journal of Pharmacy and Pharmaceutical Sciences 8.6 (2019): 15-25.
28. Touyz LZG and Nassani LM. "Affirmation: Fluoridated water reduces caries in children". Journal of Pediatric Dentistry and Hygiene 1.1 (2019): 1003.
29. Wilson WR., et al. "Prevention of viridans group streptococcal infective endocarditis: a scientific statement from the American Heart Association". Circulation 143.20 (2021): e963-e978.
30. Sanz M., et al. "Periodontitis and cardiovascular diseases: consensus report". Journal of Clinical Periodontology 47.3 (2020): 268-288.
31. Touyz LZG. "Cardiovascular Disease and Periodontitis". Clinical Dentistry 8.9 (2007): 17-24.
32. López-Valverde N., et al. "Possible association of periodontal diseases with Helicobacter pylori gastric infection: a systematic review and meta-analysis". Frontiers in Medicine 9 (2022): 822194.
33. Touyz LZ. "Periodontitis contributes to initiation, progress and aggravation of septic shock; a feasible hypothesis". Medical Hypotheses 81.4 (2013): 650-652.
34. Vila T., et al. "Oral candidiasis: a disease of opportunity". Journal of Fungi 6.1 (2020): 15.
35. Arweiler NB and Netuschil L. "The oral microbiota". Advances in Experimental Medicine and Biology 902 (2016): 45-60.
36. Touyz LZG. "After Centuries of Dentistry. Prevention Remains Best". Scientific Archives of Dental Sciences 7.5 (2024): 06-11.

Addenda on source references

1. Kamra S., et al. "Oral Hygiene Instructions with Plaque-Disclosing Agents to Improve Self-Performed Dental Plaque Control: A Case Report". Cureus 16.10 (2024): e72205.
2. Centrix. "Expose Tablet and Applicator". Centrix Dental (2026). https://www.centrixdental.com/
3. Mayo Clinic Staff. "Dental floss vs. water flosser: Which is better?". Mayo Clinic (2025).
4. Vibhute A and Vandana KL. "The effectiveness of manual versus powered toothbrushes for plaque removal and gingival health: A meta-analysis". Journal of Indian Society of Periodontology 16.2 (2012): 156-160.
5. Toothbrush Express. "Stimudent Tooth Picks". Toothbrush Express (2025). https://www.toothbrushexpress.com/