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PR

Caries and Periodontal Disease Risk Assessment and Management

Understanding bacterial diseases of the teeth and supporting structures in adults

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ABSTRACT

Increasing rates of decay and periodontal disease began with the cultivation of grains in the Neolithic, but it was the tremendous upsurge in consumption of refined carbohydrates in the Industrial Revolution that caused epidemic levels of these diseases to erupt. In the middle of the 20th century, pathogenic bacterial biofilms were identified as the cause of both diseases. Identification of the environmental risks that cause oral biofilms to become pathogenic and the management of those risks is the most appropriate means currently available to treat caries and periodontal disease.

LEARNING OBJECTIVES

- Describe biofilms, their general structure, and their role in the development of caries and periodontal disease
- Explain how environmental risks modify biofilms to create caries and periodontal disease
- Evaluate how conservative management of the oral environment successfully decreases the risks of caries and periodontal disease

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Caries and periodontal disease were minor concerns for early hominids.[1] In the late Neolithic Period, grain cultivation and the consequent rise in carbohydrate ingestion resulted in a considerable increase in both diseases. It wasn't until the mid-19th century Industrial Revolution, however, that efficient procurement and refinement of sugars and grains resulted in enormous increases in consumption of these fermentable carbohydrates. Substantial alteration in oral environments ensued, causing the eruption of epidemic levels of caries and periodontal disease that, in many parts of the world, still continues today.[2] This major dietary change moved the nascent dental profession from surgical treatments by mostly apprenticeship-trained barber-surgeons toward a more scientific education in university-affiliated dental schools. By the latter part of the 19th century, Willoughby Miller[3] and GV Black[4] began investigating bacteria as the causes of both decay and periodontal disease. Black recognized the need for vigorous tooth brushing and developed his classic cavity preparation designs into "self-cleansing" areas,[4] and JM Riggs of Connecticut published his treatise on oral hygiene, regular "scraping" of the roots along with co-treatments of, interestingly, myrrh as an antibacterial.[5]

Biofilms

In 1676, van Leeuwenhoek scraped "scruff" from his teeth, examined it under one of his first microscopes, and saw strange "animalcules" scurrying about. A bit more than 200 years later, Black[4] renamed the "scruff" *plaque*. Plaque has become more fully understood

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today as a *biofilm*: complex colonies of differing forms of microscopic life living together within a sticky film.[6]

Biofilms are extraordinarily complex multispecies aggregations of bacteria, fungi, and occasionally admixed viruses that "stick" to any wet surface and/or to each other within a self-produced "gluey" polysaccharide-polymeric matrix. These multispecies communities of sheets and clumps make up approximately 50% of the world's overall biomass.[6] Within humans, 97% of DNA is biofilm in origin; only 3% is actually human.[7]

Biofilms, like all living things, are crafted by their environments.[8] Most environments result in primarily commensal or mutualistic biofilm organizations. Though many biofilms are health encouraging, others can become dysbiotic and pathogenic.[9] Dysbiotic biofilms in the oral cavity often result from changes in local nutritional and/or overall host metabolic environments.

Cooperation or pathogenicity occurs through a communication processes called *quorum sensing*.[10] Quorum sensing is like voting. Intraspecies-specific, hormone-like "votes" are produced by individual bacteria within a colony to let others know how strong the colony has become through reproduction. Once a certain quorum vote is reached, the genomic functions and/or structure of the species changes, becoming more organized and more flexible. Additionally, all bacterial species possess a second, chemically different interspecies voting system. Interspecies votes tell other species they may be in an environment to become allies—or stronger allies—with others. Such "alliances" cooperate to modify the composition of the biofilm for good or ill. It is only within the past 30 years that recognition of how complex environmental stressors existing within and around human beings can change harmless or beneficial oral biofilms into pathogenic entities that can cause caries and chronic periodontal disease.[9] It is estimated that 70% to 80% of chronic diseases are the result of pathogenic biofilms.[11]

Caries Development

Healthy oral cavity biofilms live in slightly acidic to neutral pH environments (ie, between ~6.5 and 7.0).[12] However, consuming high levels of fermentable carbohydrates, sugars in any form, and sticky, highly refined flours leads to acidic metabolic byproducts from acidophilic bacteria that considerably lower this "normal" pH. Thus the biofilm's acidophilic bacterial populations continue to increase in the ever-intensifying acidic environment through intra- and interspecies quorum sensing. As an example, quorum sensing may facilitate their strategic reposition within the biofilm for better metabolic cooperation or nutrition. The combined acid byproducts of this interspecies cooperation further lower pH and dysbiotic, pathogenic biofilms form, ultimately causing dental caries.[9]

Another major cause of increased salivary acidity is reduction in the amount of saliva. The resulting xerostomia, or "dry mouth," means a proportional decrease in salivary buffering proteins, thereby increasing acid levels. Autoimmune diseases, many chronic diseases and conditions, and many medications—both prescribed and over the counter (OTC)—are the major causes of xerostomia.[13] This environmental change benefits population increases of acidophilic bacteria at the expense of more neutral pH-tolerating species.

Periodontal Disease Development

The first description of periodontal disease was made in 1757 in France by Etienne Bourdet, when he linked bone loss, gingival inflammation, and loosening teeth with

TABLE 1

Caries Risk Factors

LOW TO MODERATE

- No current clinical or radiographic decay within 3 years
- Good to excellent oral hygiene
- Female gender (possible hormonal connection)

HIGH

- Poor oral hygiene
- Xerostomia
- Chronic emotional stress
- Diet high in carbohydrates/sugars/acids, sticky foods
- Caries incidence within 3 years (past and present radiographic and clinical evidence, incipient/demineralization)
- Deep pits/fissures
- Gingival recession, enamel or dentin erosion
- Orthodontics, both removable and traditional
- 60 years of age or older
- Acid reflux/GERD
- Recreational drug use, frequent alcohol consumption
- Smoking and other tobacco product use
- Non-fluoridated drinking water or use of diet supplements
- Extensive prosthodontic or restorative therapy, removable or fixed partial dentures, esthetic restorations
- Current or recurrent caries that extends to the dentino-enamel junction or just beyond on one or more teeth
- Infectious contact with another who is at high/extreme caries risk
- Family history (possibly genetic)

VERY HIGH TO EXTREME

- Current or recurrent caries that extends at least halfway to the pulp on multiple surfaces and in multiple teeth
- Severe xerostomia
- Chemotherapy/head and neck radiation
- Chronic systemic or idiopathic ailments/diseases, compromised immune system
- Diabetes (type 1), kidney diseases, Sjögren's syndrome, or similar diseases

GERD = gastroesophageal reflux disease. Adapted from references 18 through 20.

the deepening of the periodontal sulcus.[14] Various treatments have been advocated to control periodontal disease, but only recently has its complex genesis become clearer by understanding biofilm interactions with each other and their host environments.[15] This information provided the basis for therapies and treatments to manage dysbiotic environments, as opposed to surgical interventions that attempt only to eliminate periodontal disease's signs and symptoms.

The Role of Subgingival Biofilms

Subgingival biofilm formation is exceptionally complex. Many bacterial species form biofilms adhering to tooth and tissue pellicles and some to each other, but some other biofilm bacteria cannot attach to others or to pellicles. They may, however, coaggregate with bacteria that can attach to pellicles or other species through quorum sensing. Coaggregation is analogous to bridging, such as in a situation where two people who cannot hold hands with each other can "bridge" via a third person who holds hands with both.[16]

As undisrupted, mostly aerobic, supragingival plaque colonies grow in amount and density, internal biofilm pH increases in the slightly oxygen-depleted depths, allowing formation of facultative anaerobic microcolonies. Multiple-species supragingival biofilms can generate tissue-irritating metabolites, many of which are tissue toxic, and cause gingivitis. With undisrupted biofilms, inflammatory gingival swelling deepens the periodontal sulcus and a more oxygen-depleted environment forms in its depths.

Facultative aerobes within the supragingival biofilm bridge with other facultative aerobes, forming more anaerobic periodontal pockets, and they then coaggregate with increasing types of obligate anaerobes. Subgingival dysbiotic biofilms are developed and the host inflammatory response goes into high gear, producing the mix of immune proteins, dead epithelial cells, and other proteomic materials upon which increasingly pathogenic anaerobic bacterial colonies feed. A more chemically basic, more anaerobic, and higher-temperature playing field continues to develop where the most aggressive periodontal pathogens are better able to gain dominance. Accelerating inflammation and host response lead subgingival pathogenic biofilms to continually destroy the periodontium.[16]

Risk Assessment

Pathogenicity only occurs as a result of dysbiotic biofilms.[12] A myriad of oral environmental changes encourage dysbiotic biofilm formation: xerostomia, diet, social habits, inadequate hygiene, and/or host chronic diseases, to name only a few. Risk identification and evaluation provide information on which well-researched risk management strategies will be developed to positively change the course of caries and periodontal disease.

Risk assessment begins with medical, dental, social, and family histories to uncover past and present systemic risks for oral environmental biofilms. Intraoral and radiographic evaluations help identify present and past conditions or diseases. Together, these data help uncover the environmental stressors that have created and will continue to create caries and periodontal disease.

Both history and examination data for decay and periodontal disease should be recorded within specifically constructed forms to allow for more efficient risk assessment and diagnosis. Such forms facilitate risk management and treatment plans to manage dysbiotic biofilms and their damaging results. They can be created for the particular needs of each practice by using the following information.

CaMBRA, **Ca**ries **M**anagement **B**y **R**isk **A**ssessment, is a widely used and well-understood tool for risk assessment and management of dental caries.[17] Table 1[18-20] shows modified CaMBRA risk factors[13] arranged from low to extreme. Periodontal disease risk factors are presented in Table 2.[19-21]

Determining risk levels for caries and periodontal disease is not formulaic but should be determined by the experience and knowledge of the dentist and/or hygienist, preferably in

TABLE 2

Periodontal Disease Risk Factors

LOW TO MODERATE

- Good oral hygiene
- Pockets <4.0 mm with slight to no gingival inflammation or bleeding
- Minimal to moderate calculus
- Minimal malocclusion or malaligned teeth

MODERATE TO HIGH

- Poor oral hygiene
- Greater than 4.0 mm pocketing, either locally or generally
- Gingival inflammation/bleeding either spontaneously or with minimal stimulation
- Continued bleeding after scaling and root planing and establishment of good oral hygiene
- Tobacco or cannabis smoking or any use of tobacco products
- Older than 60 years of age
- Hormonal changes (puberty, pregnancy, menopause)
- Missing teeth (due to damage, gum disease, decay, etc)
- Emotional stress
- Extensive prosthodontic or restorative therapy, removable or fixed partial dentures, esthetic restorations
- Infectious contact with another who has periodontal disease
- Family history (possibly genetic)
- Chemotherapy
- Chronic systemic or idiopathic inflammatory diseases or conditions

EXTREME

- History of chronic periodontal disease/infections (includes clinical and/or radiographic evidence of bone loss or furcation involvement)
- Head and neck radiation

Adapted from references 19 through 21.

consultation with each other.[22] The following can serve as a helpful guide in determining risk level, however:

- Low/moderate risk—patients with none of the high-risk factors
- High risk—one or more risks in this category
- Extreme risk—one or more risks in this category

Caries Risk Management

A summary of caries management techniques by risk level is presented in Table 3. The main products and strategies are explained in more detail below.

Encouraging Plaque Control

Historically, one of the major tools for risk management of caries has been plaque control. Many products and techniques for plaque control exist, but demonstration and personalization of technique is as important as the devices or products used. The techniques a patient feels most comfortable with should be continually encouraged or modified at all visits. Positive behavioral reinforcement from all clinical staff members is very important.

Products that assist with plaque control include electronic brushes, whether rotating or sonic; water irrigation devices; and interdental plaque control products such as floss and flossing aids and other interdental floss disruption devices deemed appropriate by professional staff.

Modifying Tooth Surface Chemistry

Calcium and phosphate ion interchange between tooth structure and saliva constantly occurs, but within highly acidic saliva, ionic movement is greater from tooth structure into saliva than in the opposite direction. Effective formulations of fluoride and calcium phosphate varnishes and toothpastes will remineralize tooth surfaces and, in doing so, also create a microsurface of more acid-resistant fluorapatite. Fluorapatite surfaces have lower surface energies, making bacterial attachment more difficult.[23]

The combination of fluoride/calcium phosphate toothpastes and varnishes is a highly effective strategy for changing both tooth surface and oral environment in managing caries risk.[24] Products for enhancing tooth surfaces against acid dissolution include fluoride/calcium phosphate varnishes, fluoride/calcium phosphate toothpastes, and calcium phosphate pastes and rinses.

TABLE 3

Caries Management by Risk Level

LOW

- Fluoridated toothpastes
- Low sugar/sticky carbohydrate diet
- 6-month to 1-year hygiene recall visits
- Use of plaque control products as appropriate

MODERATE

- Apply fluoride/calcium phosphate varnishes at quarterly or semiannual hygiene recall visits
- Dispense or prescribe fluoride/calcium phosphate toothpastes
- Low sugar/sticky carbohydrate diet
- Use of plaque-control products as appropriate

HIGH

- Apply fluoride/calcium phosphate varnishes and toothpastes at quarterly hygiene recall visits
- Dispense or prescribe fluoride/calcium phosphate toothpastes
- Low sugar/sticky carbohydrate diet
- Use of plaque-control products as appropriate
- Baking soda rinses/tooth brushing

VERY HIGH TO EXTREME

- Apply fluoride/calcium phosphate varnishes at monthly/bimonthly to quarterly hygiene recall
- Dispense or prescribe fluoride/calcium phosphate toothpastes supplemented by calcium phosphate pastes and rinses
- Baking soda rinses/tooth brushing
- Insist on low sugar/sticky carbohydrate diet
- Use of plaque-control products as appropriate

Modifying Xerostomia

Xerostomia is very difficult to manage. There are more than 600 medications that result in various levels of xerostomia.[13] Many medications causing dry mouth are not within the control of the dentist to change, as many are prescribed by other health care professionals. Communication with other health care professionals with suggestions to change medications or initiate health-producing strategies to eliminate medications is strongly recommended. Patients also self-prescribe OTC medications that cause dry mouth. Communication with patients about OTC medications is very important and will often cause them to change their strategies for managing problems for which they have self-medicated. In addition, xerostomia can also be a common side effect of many chronic conditions or disorders.

No current oral products can easily change or modify xerostomia as a caries risk. However, there are some products available that contain calcium, phosphate, and pH-buffering ingredients that may play a role in the reduction of caries risk. They would, however, be most likely beneficial adjunctively in extreme xerostomic/extreme caries risk cases only based on product chemical interaction potential, cost, and patient compliance issues.

Altering Acidogenic Biofilm Development

Developing a more neutral salivary pH may be achieved through dietary changes that reduce easily fermentable sticky foods and sugars. As stated before, this is a difficult change to make. It requires a structured plan for dietary counseling that is beyond the scope of this article. However, urging patients to modify their diet is exceptionally helpful.

Strategies for reducing acidic saliva include using a baking soda rinse multiple times daily, especially after any food consumption, and

brushing with baking soda combined with fluoride/calcium phosphate toothpastes.

Using Sealants

Sealants and Class V restorations are effective in caries management both for children and adults, especially in patients who have deep pits and fissures,[25] but they should be done only after remineralization has been initiated through the use of fluoride varnishes and toothpastes.

Periodontal Disease Risk Management

Risk reduction of periodontal disease should include strategies to restore biofilms to commensalism or preferably mutualism. As with caries, many risk factors for periodontal disease are difficult to change or cannot be changed. A summary of strategies is presented in Table 4.

Diagnosis of Active Disease

The first step in treatment of periodontal disease is to diagnose the problem, possibly using bacterial testing. Subgingival biofilm sampling for testing of pathogens should be performed at initial examination if the diagnosis of active periodontal disease is made. However, if signs or symptoms of infection continue to be present, testing also may be done following home care instruction review, or during re-evaluation following scaling/root planing, surgical treatments, or other treatments or therapies.[26] Performing microbial testing through an oral microbiology lab is especially helpful in the identification of specific periodontal disease–causing bacteria and their susceptibility and/or resistance to certain antibiotics to help determine antibiotic options for their control.[27]

Plaque Control

Plaque control in periodontal disease uses essentially the same techniques as for caries, with the strongly advised addition of an oral irrigator to access pockets at any depth but especially at 4.0 mm or greater. Removing supragingival plaque does not mean subgingival plaque is disrupted or removed. Evidence suggests that subgingival irrigation is a very worthwhile tool for achieving control of subgingival biofilms.[28]

TABLE 4

Periodontal Disease Management by Risk Level

LOW TO MODERATE

- Encourage low sugar/sticky carbohydrate diet
- Praise current hygiene habits and review compliance and oral hygiene options
- At 6-month or 1 year hygiene recall visits for assessment of periodontal disease emergence based on home care compliance and quantity of calculus

HIGH

- Recommend water irrigator and electronic/sonic toothbrush
- Evaluate oral hygiene compliance
- Bacterial testing if chronic inflammation and/or bleeding on stimulation persists
- If microbial testing is positive, antibiotic therapy with inclusion of scaling and root planing in the middle of the antibiotic therapy
- Recall at 3-4 months to assess signs and symptoms, especially inflammation and bleeding
- Retest if signs and symptoms persist

VERY HIGH TO EXTREME

- The above with an initial monthly recall/re-evaluation
- Repeat testing and treatments as needed
- Localized or general surgical intervention
- Possible extractions if local signs or symptoms re-emerge in combination with microbial testing and possible systemic antibiotic therapy

Nonsurgical Periodontal Therapy

Antimicrobial agents in the form of rinses, irrigations, or locally placed antibiotics should perhaps be avoided as they might actually result in dysbiosis of a biofilm or in antibiotic resistance. In addition, pathogenic bacteria may become integrated into periodontal or granulation tissue or attached to root surfaces in the form of persister cells (cells that become dormant until advantageous environmental conditions return for reproduction).[29]

Interestingly, in Europe and South Asia, the treatment of aggressive periodontitis has for many years involved combinations of systemic antibiotics, mainly metronidazole and amoxicillin. The combination of antibiotic therapy with intra-pocket debridement has been shown to be more successful than conventional periodontal surgery, scaling and root planing alone, local antibiotic therapy alone, or systemic antibiotic combinations alone.[26] Better success with combined systemic antibiotics and intra-pocket debridement/scaling and root planing therapy may in part be the result of reducing the chance that pathogenic persister cells reach reproduction levels.

Probiotics

Because many biofilms are host mutualistic and necessary for healthy function, it is critical to respect and not destroy all microbes in the interest of treating any particular diseases. If antibiotics are prescribed for periodontal therapy, an added twice a day probiotic supplemental regimen, either OTC or prescribed, is very strongly suggested during the antibiotic therapy.[30] Probiotics for treatment and prevention of periodontal disease and caries are currently being studied.[31]

Conclusions

Risk assessment/management in biofilm control in caries and periodontal disease is an excellent approach to manage and treat both diseases. Risk levels and management strategies developed during consultation by both dentist and hygienist is very strongly advised. Continual patient communication on risks and their management enhances treatment and therapy outcomes and may encourage a positive and continuing referral base.[13] A great amount of research over many years has substantiated the effectiveness of conservative therapies and treatments in managing dysbiotic and pathogenic biofilms that cause caries and periodontal disease.

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1. What are biofilms?
   - A. Acid byproducts from carbohydrate consumption
   - B. Multispecies aggregations of bacteria and fungi within a self-produced sticky matrix
   - C. A simple, single-species colony of bacteria living together
   - D. A type of ribonucleic acid

2. What percentage of chronic diseases is estimated to result from pathogenic biofilms?
   - A. 30% to 40%
   - B. 50% to 60%
   - C. 70% to 80%
   - D. More than 90%

3. Which of the following factors contribute to an increase in the acidity of the oral environment?
   - A. Consuming high levels of fermentable carbohydrates
   - B. Consuming sugars in any form
   - C. Reduced saliva due to xerostomia
   - D. All of the above

4. Coaggregation is:
   - A. an intraspecies-specific, hormone-like "voting" system.
   - B. a way that bacterial species can indirectly attach to one another.
   - C. another term for host response in the oral cavity.
   - D. when bacterial species bind directly to each other to form biofilm.

5. What type of biofilm is the only cause of pathogenicity?
   - A. Dysbiotic
   - B. Commensal
   - C. Mutualistic
   - D. Cooperative

6. Risk assessment begins with which of the following?
   - A. Medical history
   - B. Dental history
   - C. Family history
   - D. All of the above

7. Strategies for management of caries may include:
   - A. plaque control, scaling and root planing, sealants.
   - B. fluoride and calcium phosphate varnishes and antibiotics.
   - C. plaque control, use of varnishes, addressing xerostomia.
   - D. more frequent patient recall, bacterial testing.

8. For patients with deep pits and fissures, sealants may be used after:
   - A. remineralization via fluoride varnishes and toothpastes.
   - B. a regimen of baking soda rinses.
   - C. a round of systemic antibiotics.
   - D. all permanent teeth have erupted.

9. According to the article, the first step in treating periodontal disease is:
   - A. diagnosis.
   - B. patient education emphasizing home care.
   - C. use of antimicrobial agents.
   - D. use of systemic antibiotics.

10. Managing periodontal disease with locally placed antibiotics:
    - A. has shown promising results in Europe and South Asia.
    - B. can result in dysbiosis of a biofilm or antibiotic resistance.
    - C. should be combined with debridement for best results.
    - D. has been shown to be more successful than conventional surgery.

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LAST 4 DIGITS OF SSN ☐☐☐☐

ADA Number ☐☐☐☐☐☐☐☐☐☐

AGD Number ☐☐☐☐☐☐☐☐

The Month and Day (not year) **of Birth. Example, February 23 is 02/23** **Month/Date of Birth** _____

NAME _____

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PROGRAM EVALUATION

Please circle your level of agreement with the following statements.
(4 = Strongly Agree; 0 = Strongly Disagree)

1. Clarity of objectives	4	3	2	1	0
2. Usefulness of the content	4	3	2	1	0
3. Benefit to your clinical practice	4	3	2	1	0
4. Usefulness of the references	4	3	2	1	0
5. Quality of the written presentation	4	3	2	1	0
6. Quality of the illustrations:	4	3	2	1	0
7. Clarity of review questions	4	3	2	1	0
8. Relevance of review questions	4	3	2	1	0

9. Did this lesson achieve its educational objectives? Yes No

10. Did this article present new information? Yes No

11. How much time did it take you to complete this lesson? _____ min