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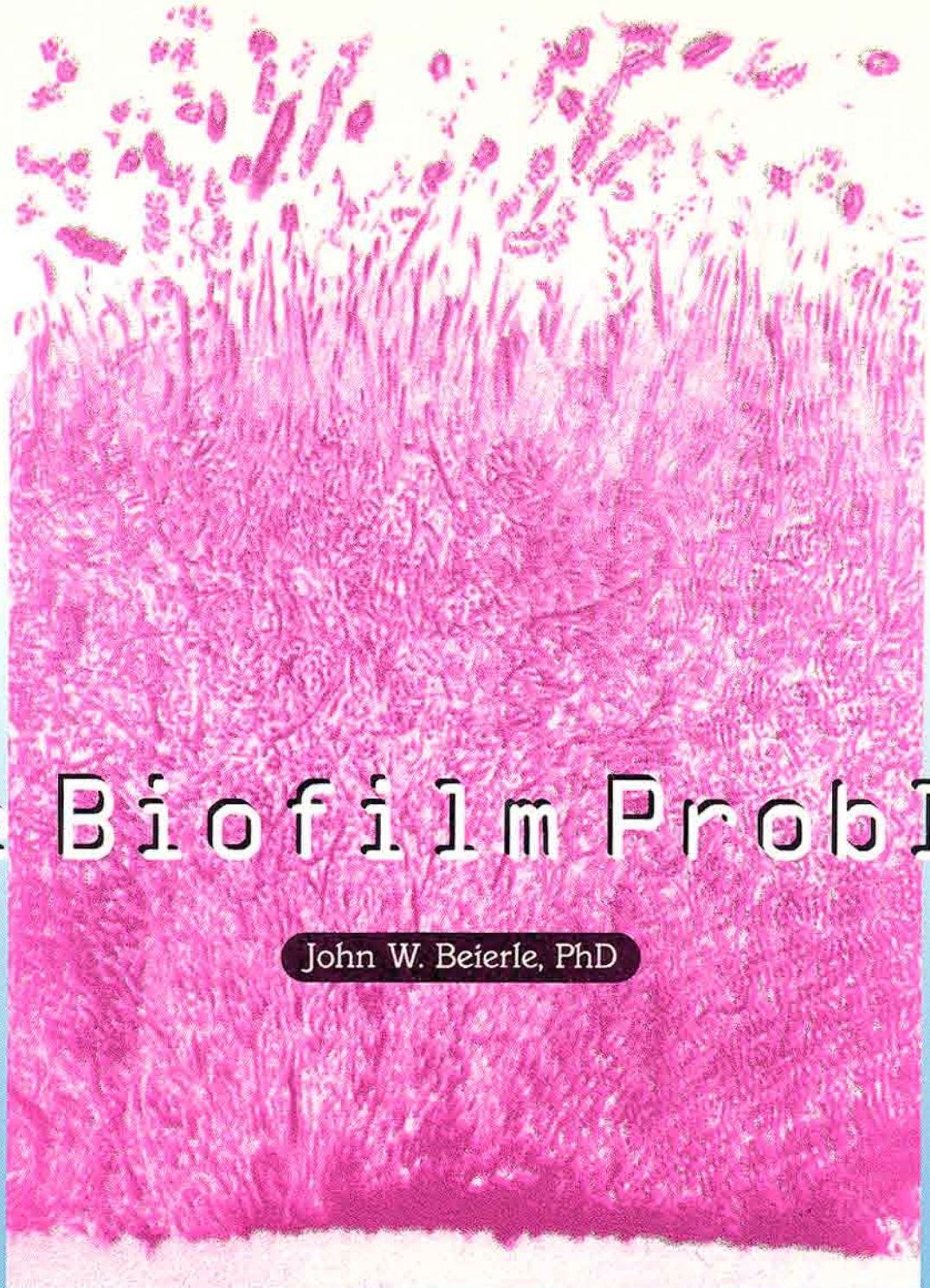
Periodontitis

Waterlines

Plaque



The Biofilm Problem

John W. Beierle, PhD

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DEPARTMENTS

FEATURES

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The Right Time...At Last

Steven A. Gold, DDS

The March 2001 CDA Board of Trustees meeting witnessed a landmark decision. Trustees passed a resolution outlining support for licensure by credential in the state of California. This would bring us one step closer to true "freedom of movement" for all licensed dentists. While there is some doubt as to whether Chief Joseph was referring to dentists in the United States, there is no doubt that it is the right time for licensure by credential to become a reality.

For those unfamiliar with the issue, licensure by credential really means licensure without further examination. If implemented in the state of California, it would mean that dentists with a license in good standing in another state could obtain a license to practice in California without taking the state board exam. Similarly, dentists licensed in California could, without further examination, obtain licenses in other states that recognize licensure by credential. The desire would be to eventually have reciprocity between all states, and the adoption of licensure by credential in California would certainly be an important step toward that end.

Currently, more than two-thirds of all states recognize some form of licensure by credential.

The dental profession in California historically has been opposed to this concept, citing that a state's right to maintain the standard of care by controlling dental licensure should not be compromised by reciprocating agreements to recognize and award licenses to dentists from other states. The premises of this argument are that the dental licensing exam is an absolute measure of competency and that the quality of dentistry is better in California than in other states. This antiquated belief of superiority is not unlike Ptolemy's conclusion that the universe revolved around the earth. For while it is obvious that there exist differences in techniques taught in dental schools across the country, there is no evidence that dentistry done in California serves its public any better than dentistry done elsewhere in the country.

However, many feel the real reason for such opposition to licensure by credential is not the noble cause of upholding the standard of care but rather fear -- fear of adverse economic consequences by a massive influx of dentists into California should the state board exam be waived for dentists licensed elsewhere. Do we really believe that people everywhere so envy the weather and lifestyle in California that they would uproot any ties to their communities and families to live here? If so, then why haven't other professions that don't have a state licensing examination been inundated by such an economically disastrous westward migration?

The fact is we do live in a more mobile society today than we did 50 or even 15 years ago. Dental students and young dentists unsure of where they will practice

are concerned about the prospect of having to take multiple examinations to prove competency and receive licensure. Perhaps the group that is most unfairly affected by current licensing restrictions is specialists. Consider the case of an oral surgeon from Texas who has been practicing for 20 years with an impeccable record and standing in his community and profession. Should he desire to relocate to California, he would have to prove his competence on, among other procedures, a class II amalgam, which he likely has not done since entering his oral surgery residency. Assuming he passes the licensure exam, he would then have to promise not to do any of these amalgams so that he may ethically announce his specialty as an oral surgeon. Now compare this situation to a practicing neurosurgeon, who is not required to take a clinical examination to practice in California. Why is it that we let someone do brain surgery in this state without further examination? Is dentistry more complicated?

Licensure by credential will become a reality in California. Assemblyman Sam Aanestad, as many of you know, is the only dentist in the state legislature. He is sponsoring legislation, Assembly Bill 1428, that will make licensure by credential California law, and he has promised he will get this legislation passed. He is not only a CDA member but also a champion for the dental profession and is maintaining close communication with CDA leaders on this issue, all of which will ensure that dentistry's best interest is represented in the new law.

It is the right time for licensure by credential. The majority of CDA dentists say they want it, as reflected by the action of the Board of Trustees and a similar resolution passed by the House of Delegates. Perhaps the strongest argument for pursuing licensure by credential at this time is that if dentistry doesn't act to guide this legislation, there are other individuals and groups that will do it for us; and they may do so in a design that is unfavorable to the dental profession.

We support Sam Aanestad as he works to make licensure by credential a reality in California. After all, it's not just a matter of obtaining a dental license. It's about doing what is best for the profession. It's about freedom to live and practice where we want. And, it's about time.

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Dentists Decry Soda in Schools

By Debra Belt

Soft drink machines are becoming a part of the landscape on middle and high school campuses across the country, and soda reigns as the beverage of choice among the nation's young.

And while some of the possible detrimental health effects are still in dispute, the negative effect on dental health is not.

Since 1998, soft drink availability in schools has increased fivefold with

240 school districts in 31 states approving exclusive "pouring rights" contracts with soda companies. At the same time, the U.S. Department of Agriculture reports unprecedented rates of consumption among kids and cites statistics such as 56 percent of 8-year-olds drinking a soda per day and one-third of teenage boys drinking at least three cans of soda a day.

Fizzing along with the carbonated beverage phenomenon is debate about its health effects. Public health organizations, parents, consumer groups, and industry officials are facing off in discussions about the sugar, caffeine, and caloric content of the 450 varieties of soda on the market and their effect on developing bones, teeth, minds, and bodies.

Lawmakers in California and other states are targeting the easy access to soda on campus, and last year the ADA declared its opposition to contracts that influence consumption of soft drinks in schools. Meanwhile, the National Soft Drink Association recently staged a fly-in to lobby Congress about the "proper perspective" on soft drinks in schools.

The jury is still out on whether soft drinks contribute to obesity, caffeine dependence, and bone weakening. But as The Washington Post recently reported, there is one health effect that even the soft drink industry won't dispute. That, of course, would be the health effect dentists are familiar with.

"I learned years ago about what soda, especially when sipped over an extended period of time, can do to teeth," says William Comport, DDS, who has operated a private practice in San Jose for 31 years. "Early in my career, I treated a young man who sipped Dr. Pepper all day long while he worked pressing clothes at a dry cleaning business. He was ready for dentures at 27."

Comport notes that this was a worst-case scenario and that the key relationship between soda and tooth decay is how the drink is consumed. "If a soft drink is consumed fairly quickly and with a meal, it's not a problem," Comport says. "The problem comes in when a soda is sipped over time, and sugar and acid keeps getting reintroduced into the mouth."

Dental professionals concur on the one-two punch soda can deliver to teeth.

"The problem with soft drinks is twofold," says Simon Morris, DDS, a pediatric dentist in Los Gatos. "The sugar in soft drinks sets up the normal acid attack by the bacteria, but the acidic nature of the soda can lower the pH of the plaque even further."

Information recently released from the Ohio Dental Association pointed out that acid begins to dissolve tooth enamel in only 20 minutes.

Dentists also note that soft drinks appear to be more popular with teenagers than with younger children.

"Parents have more control over what younger children eat and drink," says Randall Wiley, DDS, a pediatric dentist with offices in Concord and Danville. "The trouble with soft drinks comes in with social drinking and sipping. It's very important to limit the amount of soda and limit the duration of drinking time. The last thing you want to see is a teenager sipping a 48-ounce soda over three hours while studying."

Information on soft drink sales in schools coincides with the trends dentists point out. Soft drinks sales are concentrated in middle and high school campuses targeting the nation's teenagers and their combined $141 billion spending power. Current law also states that vending machines can't be turned on until after the final lunch period, encouraging social soda drinking.

The consensus of dentists, health care professionals, and advocacy groups is that soft drinks are an offering of easily accessible sugar and empty calories that substitutes for more-nutritional options.

"I'm concerned in general about nutrition in the schools," Wiley says. "From a nutritional standpoint, the availability of soft drinks in schools is not a good idea."

Public pressure on this issue has humbled Coca-Cola to agree to stop signing lucrative, exclusive agreements with pubic schools and limit the availability of its products on campuses. The cola giant also says it will add more juices, bottled water, and sugar-free drinks to its 100,000 campus vending machines. Whether other soft-drink makers will follow suit has yet to be seen.

Coping With Market Volatility

By Marios P. Gregoriou

Periods of increased stock volatility, when securities prices tend to sharply rise or fall within a relatively short period of time, make many investors understandably uncertain. Investors who are currently in the market or are considering entering have probably been wondering about the best course of action to take during periods of unsettled market activity.

Don't Overreact

It's somewhat of a cliche to say that two emotions -- fear and greed -- are the driving forces behind a good deal of stock market activity. During a bull market, as share prices rise, some investors develop a false sense of confidence regarding future price levels and believe that nothing short of a catastrophe will stop the continuing upward trend. In their zeal, they erroneously project their short-term gains into an uncertain and long-term future.

On the other side of this coin lies the disappointment that may set in whenever market values start to drop within a relatively short period. During these bear markets, some investors overreact and

begin imagining a loss of their nest eggs due to lower share prices. They may even begin selling their holdings in the fear that prices may fall even further.

It's important, however, for individual investors to view market volatility in its proper perspective. Swings in stock market prices, even those lasting a few months or years, generally should not be allowed to disrupt a long-term investment strategy. Why? It's simple. Historically speaking, long-term investing has tended to smooth out many of the fits and starts that can cause investors so much short-term discomfort.

Focus on Long-Term Objectives

Setting a middle course, one that avoids both bullish euphoria and bearish despair, can help individual investors keep their long-term financial objectives in sight. A focus on long-term objectives also helps avoid the temptation of trying to predict what the financial markets will do tomorrow, next week, or next month. Long-term investors realize that even investment professionals cannot always accurately predict short-term market movements.

Adopting a long-term investment philosophy also helps guard against overreacting to business stories that appear in the newspapers or other media. Regardless of whether such news is generally thought to be "good" or "bad," an investor should always consult with a financial adviser to evaluate the potential impact of these developments on his or her overall investment plan.

Review Strategy Periodically

Reviewing one's financial strategy at least yearly is yet another way of coping with market volatility. As one does the review, he or she should make sure the investment plan takes into account his or her age and investment timeline, as well as financial resources and tolerance for risk.

At least for the foreseeable future, occasional spells of stock market volatility are probably unavoidable. However, following a long-term financial plan can help an investor weather the storm.

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Caries at Age 6 Tied to Snack Habits at 3

Sugary snack habits at age 3 can lead to increased caries by age 6, regardless of oral hygiene habits, according to an article in Dentistry and Oral Epidemiology. Children 3 years old who had candy and juice more than once a week and who also had tarter were almost twice as likely to have caries by age 6, as opposed to those who had sweets no more than once a week, according to the study of 135 Finnish children by Dr. Sara Karjalainen and colleagues from the University of Turku.

About 19 percent of the subjects had one to four carious lesions at age 6 and 10 percent had five or more.

Toothbrushing habits did not differ between children who developed lesions and those who did not.

"Candy and sweet drinks contain sucrose and therefore both should be used reasonably -- definitely not on a daily basis or even worse, several times a day," Karjalainen says.

Sjögren Booklet Available for Patient Education

A new comprehensive booklet on Sjögren's syndrome for the public is available from the National Institute of Arthritis and Musculoskeletal and Skin Diseases, part of the National Institutes of Health.

Questions and Answers About Sjögren's Syndrome includes information about symptoms, diagnosis, the types of doctors to see, treatment, and ongoing research. It also includes practical information on living with Sjögren's syndrome, such as tips on oral hygiene and eye care, ways patients can protect their voices, and medicines with side effects that can contribute to dryness of the mouth. The booklet ends with a list of professional, voluntary, and government organizations with information relevant to some aspect of the disease.

The booklet is available free online at www.nih.gov/niams/healthinfo/sjogrens/ or by writing NIAMS Information Clearinghouse, NIAMS/NIH, 1 AMS Circle, Bethesda, MD 20892-3675.

Fauchard Foundation Offering Grants

The Foundation of the Pierre Fauchard Academy is seeking applications for program grants to be awarded in 2001.

The grants are aimed at volunteer dental clinics and other programs offering specific services to needy patients in the United States and abroad.

"In the year 2000, we awarded $306,480, and we fully expect to exceed that amount in the coming years," says Robert Shira, DDS, president of the Foundation.

More than $1.5 million has been awarded since the program's inception five years ago.

To submit a grant application for 2001, please contact Shig Ryan Kishi, DDS, executive director, PFA Foundation, 1441 Avocado Ave., #508, Newport Beach, CA 92660-7704, or fax to (949) 721-9146, or e-mail to fpfa@aol.com. The deadline for applications is June 1, 2001.

Museum Re-Opens Modern Dental Office Exhibition

The Dr. Samuel D. Harris National Museum of Dentistry has updated and re-opened its Modern Dental Office Exhibition, which shows the striking contrast between the dental office environments

British Patients Can See Medical Records Online

In a pilot program, patients at two medical practices in Britain will be able to see their medical records online.

"Electronic patient records will help to put patients in control of their own health and health care," says Health Minister Gisela Stuart.

The pilot program is an initial step in Britain's National Health Service plan to ensure that everyone in England and Wales will have online access to their own health records by 2004.

"Patients will be able to look at their medical records prior to seeing their general practitioner, ensuring that they are fully informed about their health and have time to think of the questions they wish to ask," Stuart says. "This will make consultations faster and clearer as both the doctor and the patient will have access to the same information prior to consultation."

Each time patients access their records, their identity would be checked by a smart mouse that reads index fingerprints. Patients would also need passwords to access their files.

"Rather than Trust me I'm the doctor,' the focus should be Trust me, I'm the patient,'" says Dr. Cecilia Pyper, from the Bury Knowle Health Centre in Oxford, one of the medical practices participating in the pilot program. "Offering patients access to information is like offering a currency that enables them to form more equal partnerships with health professionals."

of the 19th and 21st centuries.

As visitors walk through the museum's permanent exhibition, 32 Terrific Teeth, they can see how the innovative equipment on display in the Modern Dental Office Exhibition contrasts with early American dental offices. On display are the technological and ergonomic breakthroughs in dentist and patient comfort that dental offices have undergone since the time of the itinerant dentists and G.V. Black in the 1800s.

Where the modern office stresses seated ergonomic comfort, early dental offices were designed for the dentist to stand while treating patients. The itinerant office featured the standard wooden chair customary in hotels, and early American chairs used leather or plush materials and were difficult to clean. When Black practiced, natural light was used, so most dental offices featured large windows and the chairs were turned to face the south, if possible, so light could be used all day.

Dental offices of the 1800s usually featured a wooden table for the itinerant dentist or an ornate wood and glass wardrobe-like cabinet that required the dentist to stand when retrieving dental tools. In addition, X-ray machines have advanced since their introduction in 1896 when patients had to sit still with film in their mouth for 25 minutes until the rays could pass through the film.

The National Museum of Dentistry opened its doors five years ago. Exhibits include a tower of dental chairs, antique extraction instruments and early dental tools, and George Washington's not-so-wooden dentures.

The Modern Dental Office was updated with loans of dental equipment from the DentalEZ group. The G.V. Black dental office exhibit is on loan from the Smithsonian Institution.

The National Museum of Dentistry is in Baltimore, Md., and can be reached at (410) 706-0600 or at www.dentalmuseum.org.

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An Introduction to Biofilms

JOHN W. BEIERLE, PHD

AUTHOR

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The issue of dental waterlines has raised many questions about office safety, effective treatment of biofilms, and the means by which to achieve those solutions. Even further, the issue of office safety has been challenged by the question of whether the waterline biofilm problem exists at a significant level or is merely a tempest in a teapot. In some instances, the dental practitioner feels that the "system" is leaning on him or her personally for singling out on what is, in fact, a universal problem. How common, then, are waterline biofilm problems? Are biofilms established in systems other than dental waterlines?

The Universality of Biofilms

Biofilms are universal. They coat the walls of swimming pools, line the bottom of boats, and live in medical and dental devices and even hospital showerheads. Waterlines in virtually all standard plumbing eventually get a buildup of microbial masses, which we call biofilms. The scientific community is now recognizing the fact that biofilms are a potential problem in many areas of medicine, biology, and public life. In medicine, hip joint replacements are coated with biofilms, as are indwelling catheters, kidney dialysis machines, and numerous other devices.

An intent of this issue of the *Journal of the California Dental Association* is to bring the worldwide problem into focus in relation to dentistry's specific niche in the biofilm world. Simply stated, a biofilm is an colonized mass of bacteria attached to some solid or tissue surface. That surface may be dental enamel, a water pipe, or any waterline. The biofilm may even be a slimy mass attached to an instrument panel in a space vessel, which would take the biofilm problem beyond the Earth's surface. Dentistry also has biofilms below dentino-enamel junctions, in a process we call periodontitis. The coatings on our tongues, such as found in candidiasis, are also biofilm in nature. Bacteria don't care where they form biofilms; they just want to colonize for survival and protection.

Bacteria have a few key ways to attach to a surface. Pili, short structural appendages connected to the cell wall, can help attach bacteria to a variety of surfaces. Another attachment method is the glycocalyx, a protein-polysaccharide coating that was once called the slime layer. The slime layer is a loose, amorphous mass of carbohydrate-based material secreted by bacteria; it was considered unimportant until the past few decades. This material is critical to the attachment of bacteria to surfaces

and has a variety of other functions as well. Once a biofilm forms, a number of amazing things begin to happen. Bacteria start chemically signaling each other by a process known as quorum sensing. The bacteria then send other signals to each other, which, in turn, starts a process known as signaling. Signaling induces bacteria to start behaving not as planktonic free single cells, but rather like multicellular systems acting in concert. Resistance to drugs and chemicals, the initiations of rapid cell division, mucin production, and toxin production are all part of the signaling process that make biofilms extremely well-protected. We in the health professions inherit the problem of dealing with this biomass.

Why do bacteria need to colonize? To survive. There is indeed safety in numbers as any animal flock or school of fish knows. It is estimated that many bacteria live in biofilms, much as we live in cities. Antony van Leeuwenhoek, the first microscopist, initially saw bacteria in dental plaque, making him the first oral microbiologist and biofilm experimenter back in the 1600s. It has taken a long time for humankind to begin to appreciate a bacterium's "lifestyle."

So What Is a Dental Waterline Biofilm?

A biofilm is a mass of microorganisms coating some solid object, be it organic or inorganic. The microbes secrete a mucinous glue, which allows them to stick to a surface. Other microbes then attach to the adherent ones and, before long a mass of bacteria occur, which appears to be a sticky layer, often called a slime layer. They form in dental waterline units, and originally the bacteria come from the city water supply. We are allowed to receive up to 500 colony forming units per milliliter of water considered drinkable or potable. We are not allowed to have coliforms (enteric microbes such as E. coli) in our water at all. Are there some potentially pathogenic microbes found in safe city (or potable) water? Yes, Pseudomonas lives in normal water supplies, as does Legionella capable of causing Legionnaires disease. Various molds also live in drinking water. These microbes are normal inhabitants of water.

Are there other microbes that may be harmful? Yes, microbes found in the mouth or blood from the oral cavity may be sucked back into the dental waterlines; and those oral microbes may join the natural waterline biofilm colonies already established there. Every time the water in the units is used, sections of the biofilm mass release into the lines and then into patients' mouths. The harmless water microbes are not the problems. A build-up of human pathogenic bacteria could be a problem, especially to the young and old, the infirmed, or the medically compromised. The medically compromised includes patients undergoing chemotherapy or radiation therapy or who are immunosuppressed. Organ transplant recipients are one example of such a patient. The AIDS or HIV-positive patient also falls into this category. If these people are assaulted by high levels of microbes through waterlines, the portals of entry of ingestion and respiratory route infection, or even blood-borne transfer, are now introduced into the equation. For these reasons, as well as esthetic ones of maintaining a clean practice environment, clean waterlines are important. Other concerns include the patient's perception of the problem and adverse publicity from the media.

The public view is often that if one dentist has a problem, all dentists have the problem. Adverse publicity through the media can also make life difficult in the realm of public relations. A proactive approach to a problem is often the best approach. Education of the profession on issues is essential to the solution. Education of the public and the media is also crucial.

We must remember that we are not alone with biofilms in dentistry, for the entire world is covered with microbes. Dental biofilms include plaque, calculus, periodontal spaces, the tongue, gingiva, and epithelial linings of the oral cavity. The entire gastrointestinal tract is, in fact, a subway of massive adherent biofilms containing billions of microbes clinging to tissue linings and to each other. The entire elementary tract is indeed a continuous organ-microorganism system.

Dental Operatory Waterlines

The question of the level of contamination in dental waterlines has prompted a variety of studies and propelled development of a series of engineering controls to deal with the problem. Purging of lines between patients and after periods of inactivity ranging from hours to weeks has demonstrated that the build-up of biofilms is rapid and enormously high in colony-forming units. Purging can temporarily reduce this buildup in cell numbers, but the problem persists because of rapid microbial cell division and repopulation.

In instances where one uses an independent water reservoir and a pump system, one can often find high numbers of bacteria still remaining in the waterline. Usually, the solution is easy and dependent on the so-called clean water source. In one instance, it was subsequently learned that the water source was a water cooler's reservoir. The reservoir itself was contaminated with more than 250,000 cfu/ml, and a biofilm had formed in the interior of the water cooler. Staff members using sipper cups obtained drinking water by placing the sipper mouth to the cooler spigot, thereby inoculating the bottle reservoir. Water bottles of one-gallon size obtained from markets and labeled "drinking water" or "distilled water" are often used for clean water sources. The presence of colony forming bacteria are almost always detected in low numbers, e.g., 2-14/ml. The water in bottles is very clean but not sterile.

If we choose to utilize a bottled water system, we should exercise care in the water source we use and change the bottled water often enough to avoid

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microbial buildup over longer periods. Sterile water is difficult to keep sterile; but without nutrients, microbes grow slowly. Nutrients may enter the system via connecting tubing and backflow-delivering quantities of saliva and blood as a nutrient source, along with the associated microbes derived from dental unit sources. Frequent changeover of water is essential, as is the cleaning and sterilization of tubing associated with our waterlines. We also have a tendency to forget we can sterilize our lines and water in our own sterilizers, which, in fact, are checked weekly for efficacy. Anecdotal reports trickle in raising the issue of corrosion by water containing added bleach. People have a tendency to do too much of a good thing. If a little bleach is OK, then a whole lot should be better. Bleach is a full disinfectant at 1:100 dilution (0.05 percent) of a 5 percent solution.

Water filtration systems are another means of providing a clean water supply. The cost of filter changes and the potential buildup of biofilms in lines leading to or after the filter are possibilities.

Perhaps the major issue with dental waterlines concerns people with compromised immune systems. Old, young, cancer, HIV-positive, organ transplant recipient, chemotherapy, and radiation therapy patients all have potential problems from a microbial assault. Normal water supplies routinely harbor common water microbes. These microbes are not selectively screened out of the indigent population of microbes in our city water supplies.

A British study has revealed that dental surgical suites in England have the same problems as their American counterparts. Their studies have revealed that oral microbial species such as Moraxella and Flarobacterium often end up in dental waterline biofilms. The British studies also revealed that airline biofilms existed and could contain Streptococci, Candida, Lactobacillus and Legionella. Their ultimate finding was that 95 percent of the British dental waterlines exceeded potable water limits versus 83 percent failure for American Dental Association recommendations (accepted British loads were less than 100 cfu/ml and ADA loads were 200 cfu/ml).

Obviously, dental unit waterlines are a worldwide problem, and equally as obvious is that the poorly understood nature of biofilms will not instantly yield any one-step answers. We must first acknowledge the problem as a worldwide one and not view the issue as a personal affront. It took the microbes a few billion years to get this far, the field of microbiology is about 125 years old. In the past decade, we have made substantial progress.

The Purpose of This Issue

To assist the practicing clinician, we have assembled this issue to highlight some, but not all, of the problem confronting the dental and medical world regarding biofilms. Periodontal disease, an issue to us all, is approached from different, yet similar avenues by Dr. Casey Chen at the University of Southern California and Dr. Wenyuan Shi at the University of California at Los Angeles. Dr. Shi also ties together medical-dental problems of common concern. Elinor Pulcini presents a more technical side of the biofilm problem to illustrate the complexity of microbes in a biomass. She outlines many of the events occurring in the world of biofilms. The understanding of the mechanisms underlying the formation, maintenance, and repair of biofilms will eventually provide solutions to the problem. Ms. Pulcini is a member of the Montana State University Center for Biofilm Engineering in Bozeman, Mont. The center is arguably the leading biofilm research institute in the world. It is hoped that this issue of the CDA Journal will help to build on the base of information available to the dental community and aid in the understanding and solutions to a universal problem.

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Biofilms: Sensing and Signaling

ELINOR DELANCEY PULCINI

ABSTRACT Biofilms are a community of surface-attached microorganisms that can have far-reaching effects. Biofilms are costly to industry and affect human health in a variety of ways. Research is only now beginning to discern the complexities of biofilm formation.

AUTHOR

Elinor deLancey Pulcini is a PhD candidate at the Center for Biofilm Engineering at Montana State University. Prior to that, she was head of the Science Department and a science instructor at Bigfork High School in Montana.

The problem of bacterial contamination of dental waterlines is an excellent illustration of a basic precept in biofilm science: Biofilms are the preferred mode of growth for most bacteria. Existence as a biofilm provides bacteria with a protective environment that effectively prevents attack by antimicrobials, biocides, and even immunologic factors. Biofilms are costly for industry due to their biofouling potential, which can cause a pressure drop or product degradation.1 The detachment of biofilms has been implicated in the contamination of food and household products during manufacturing and processing. Biofilms are also associated with public health issues beyond the problem of dental waterline contamination. For example, biofilms in drinking water systems may act as a reservoir for potential pathogens.2 In the human body, there is a direct relationship between the presence and severity of dental plaque biofilm and an increase in the potential of suffering a heart attack.3 Despite the growing body of research into biofilm formation, relatively little is known about the metabolism and physiology of biofilm bacteria.4

Antony van Leeuwenhoek could be considered one of the first biofilm researchers when, in the late 1600s, he scraped dental plaque from his mouth and looked at it with his microscope. In 1943, ZoBell published a study of the affinity of marine bacteria for attaching to surfaces.

However, it was not until the 1970s that research in the formation of biofilms really started. Two assumptions were pervasive in early biofilm research: that biofilm bacteria and planktonic or free-floating bacteria are the same and that biofilms were relatively simple systems of homogeneous slime. The more-traditional microbiological methodologies of plating and broth culturing of bacteria have, until recently, warped the view of how bacteria really live and survive in the environment. Improvements in technology have allowed biofilm scientists to prove otherwise.5

Current Research

When a bacterial cell comes in contact with a surface, it may or may not stick immediately. The confocal scanning laser microscope allows for the visual examination of biofilms in real time with minimal preparation. Using the confocal scanning laser microscope, individual cells of Pseudomonas aeruginosa containing a genetic insert called green fluorescent protein were followed as they attached to a surface. The green fluorescent protein genes, which come from jellyfish, cause the cells to fluoresce, allowing for the visualization of bacteria without the use fixatives or stains that kill the cells. Results indicated that some bacteria will permanently attach to the surface while others will attach briefly and then move on to another position.6 During this time of initial adhesion, there are a number of changes taking place within the bacterial cell. Bacteria that are dividing at the rate of minutes in culture will stop dividing for hours when first attached to a surface.6 During this time, there are numerous changes occurring as that bacterial cell makes the transition from a planktonic to a biofilm cell. Eventually, the biofilm bacterial cell will be metabolically and physiologically very different from its planktonic counterpart to the point that there may even exist what is now termed the biofilm phenotype.7

Attached bacteria produce an exopolysaccharide matrix that can act as a protective polymer for the cells embedded within. As the biofilm grows and thickens, it begins to develop into a heterogeneous matrix interspersed with channels that allow nutrients and oxygen to penetrate into the depths of even the thickest biofilms. Researchers have shown that the cells within the biofilm matrix exhibit differences in physiology depending on their location. This concept of spatial heterogeneity within a biofilm has been applied to oxygen limitations (from aerobic to anaerobic), pH, nutrients, and rates of growth.8-10 Within a thick biofilm, there are various microniches that allow for numerous types of metabolic processes to take place. Dental plaque is an excellent example of the complexity of microorganisms that can exist within a biofilm with a range of metabolic capabilities.10

The development of a biofilm appears to be a very effective survival strategy for bacteria. The cells within the biofilm exhibit an increased resistance to biocides and antimicrobials in comparison to planktonic cells. A number of hypotheses have been put forth to attempt to explain this phenomenon. In some cases, there is a limitation to the penetration of the antimicrobials into the biofilm matrix. Since cells within the matrix are living at different physiologic states, the rate of uptake into the cell of the antimicrobial can be affected. The exopolysaccharide of the biofilm matrix may provide a physical barrier to the penetration of antimicrobials.11 The differences in bacterial cell physiology within the biofilm will reduce the susceptibility of cells to some antimicrobials such as growth-dependent antibiotics.12 However, diffusion and growth limitations alone may not account for the entire decrease in susceptibility to antimicrobials seen in biofilm cells. A study of the effects of antibiotics on Klebsiella pneumoniae biofilms grown on microporous polycarbonate membranes showed that ampicillin, unable to penetrate the biofilm matrix, cannot kill K. pneumoniae biofilm cells. In contrast, ciprofloxacin was shown to be able to diffuse through the K. pneumoniae biofilm in as little as 20 minutes. However, K. pneumoniae cells were resistant to ciprofloxacin at even 10 times its established minimal inhibitory concentration.13 This suggests that the genetic changes the planktonic bacterium undergoes as it becomes a biofilm cell may somehow also affect its susceptibility to various antimicrobials.

Ongoing research at the Center for Biofilm Engineering at Montana State University in Bozeman has been working to delineate the changes that occur in P. aeruginosa during initial attachment. Proteomics involves the analysis of differentially expressed (induced or repressed) proteins and allows researchers to analyze the protein expression of an organism at a particular point in time or under a particular condition. Results indicate that cells of P. aeruginosa attaching to a surface begin to express changes in their protein profiles (when compared to planktonic cells) in as little as 10 minutes after inoculation. These changes in protein expression continue during the time of the experiments (three hours).14 These differences in protein expression during initial adhesion indicate physiologic changes are taking place within cells as they attach to a surface.

As the biofilm develops, bacterial cells within the matrix will release chemical signals. These signal molecules may enable the bacterial colonies to develop the characteristics of a more mature biofilm. A number of bacterial species, both gram-positive and gram-negative, use these chemical signal molecules to coordinate activity.15 The action of these signal molecules relies on a process called quorum sensing. In quorum sensing, the ability of the molecule to cause an action is dependent on its concentration within the environment. That concentration can increase only when there is a sufficient number of bacterial cells producing that particular signal. Probably some of the best-known quorum sensing systems are

found in marine bacteria of the genus Vibrio. Species of this bacterial genus symbiotically colonize the light organs of certain fish or squid and will emit luminosity only when the population density has reached sufficient quorum density numbers.16

The cell-to-cell signaling systems of P. aeruginosa have been extensively studied as a model for quorum sensing during biofilm development by gram-negative bacteria. Mutant strains of P. aeruginosa deficient in one of the quorum sensing systems (lasR) have been shown to produce biofilms that lack the towers and channels often seen in P. aeruginosa biofilms. In addition, these mutant biofilms lack the resistance to treatment by sodium dodecyl sulfate seen in wild-type biofilms.17 Recently, researchers have isolated quorum-sensing molecules produced by P. aeruginosa from the sputum of cystic fibrosis patients, suggesting that this is a biofilm disease of the lungs.18

Research into the cell-to-cell signaling capabilities of gram-positive biofilm-forming bacteria has also been ongoing. Mutants of Streptococcus gordonii, a gram-positive oral bacterium that initiates the formation of dental plaque, were assayed for defective biofilm formation. In this particular study, nine mutants shown to have defects in genes of known function could not form biofilms. One of the genes identified, ComD, is a known component of the cell-to-cell signaling system in gram-positive bacteria.19

Conclusion

The majority of bacteria in the environment are found attached to surfaces rather than as unicellular, freely suspended planktonic cells. Biofilms are found in almost every environmental system studied and in nearly every industrial and medical setting where microbial contamination is a problem. Dental water lines can provide just the environment conducive to biofilm growth. The quality of dental water is obviously critical. To successfully minimize contamination, it is important to understand the physiology and metabolism of biofilm bacteria. That bacteria do not usually live in the environment in suspensions of single cells has significant ramifications both for the relevance of how most bacterial species are studied and for the treatment options utilized for biofilm control.

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Bacterial Biofilm and Dentistry

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ABSTRACT Bacterial biofilms are ubiquitous in nature. Recent studies have demonstrated many unique qualities previously unknown to bacteria and have yielded new insights into relevant dental issues.

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A biofilm is commonly accepted as a community of bacteria that is adherent to a particular surface. These communities have been observed in nature since the early days of microbiology. Dating back to the mid 17th century, Antony van Leeuwenhoek was making observations about the various organisms he witnessed in tooth plaque. For the many decades following Leeuwenhoek's initial observations, biofilms were typically a phenomenon studied by environmental microbiologists, who usually studied them in the context of various aquatic and marine habitats. By the 1970s, pioneers in the field were beginning to recognize that biofilms were more than an aberration seen in certain river beds, but were actually a mode of bacterial survival common to many different environments.1 In some instances, biofilm bacteria were recovered from the most unthinkable of locations. For example, a report in 1998 described the isolation of several sulfate-reducing and acid-producing species of bacteria from the storage basin for spent nuclear fuels. These communities persisted in extremely nutrient poor deionized water under constant stress from α–, β-, and γ-emitting radioactive material.2 By the 1990s, it was very clear that sessile biofilm communities were the dominant populations of bacteria inhabiting every sampled (and imaginable) aquatic environment.3

Bacterial biofilms are exceptionally well-organized and typically form mushroom-like structures.3 Within the

biofilm communities are numerous water channels that deliver nutrients and remove toxic waste products. These structures are so well-ordered that they have been compared to higher levels of eukaryotic organization.4

Mechanisms for Formation

Attachment of bacteria to a particular surface has long been associated with biofilm growth,5 but the steps leading to the biofilm development process have only recently been elucidated. A layer of exopolysaccharide typically surrounds wild-type free-floating bacterial cells (called planktonic cells). These provide the first mechanisms of potential attachment to a surface or tissue.6 Once the planktonic cells have sensed that they have attached to a surface, they quickly respond by expressing a new set of genes that enable them to form biofilms. Mutagenesis experiments in different biofilm-producing bacteria have shown many common themes in the production of biofilms. As might be expected, genes involved with the production of exopolysaccharide are a common requirement for normal biofilm development. In a recent mutagenesis study on Vibrio cholerae,7 it was shown that exopolysaccharide-defective mutants failed to form normal biofilms. It also appears that some sort of motility and/or adhesion apparatus is required for biofilm formation.7-10 This makes sense given that motility can help bacteria get close to a surface, while adhesion apparatus such as pili enable bacteria to attach to the surface.

A less-obvious biofilm-forming mechanism involves bacterial cell signaling. In Pseudomonas aeruginosa, a mutant in the lasI gene produced flat, undifferentiated biofilms, which were also much more susceptible to treatment with biocide.11 The lasI gene is a critical factor associated with intercellular signaling in P. aeruginosa. This provided the first proof that biofilm production is actually a signaling event involved in regulating genes responsible for proper biofilm development. More recently, it has also been shown in the oral bacterium Streptococcus gordonii that mutations in its intercellular signaling system disrupt proper biofilm development.10

Physiological Differences

It is only recently that microbiologists have begun to treat biofilm bacteria as distinct entities from their planktonic counterparts.6 Recent studies show that biofilm formation induces many phenotypic changes in bacteria, and large portions of the entire gene expression profile are affected. A screen for differential expression of genes in Escherichia coli found that 38 percent of its genes had changes in expression as a consequence of biofilm development.12 In P. aeruginosa, biofilm cells were found to have 30 percent to 40 percent of their cell envelope proteins expressed in only the biofilm phenotype.6 The metabolic capacity of biofilm bacteria also undergoes a dramatic change, with bacterial doubling time being markedly increased.13 Cells in these communities also have an extraordinary ability to modulate their metabolic needs based upon their particular niche within the biofilm.14

It has been demonstrated that bacteria within a biofilm have a strong propensity to share genetic material as well. In an experiment measuring genetic material exchange through conjugation within a biofilm between E. coli and Alcaligenes eutrophus, the rates of conjugation were observed to be as much as a 1,000-fold higher than those obtained from typical plating methods.15 Certainly, this presents a scenario that is ripe for rapid and efficient adaptation to any number of environmental challenges. Taken together, it becomes increasingly clear why there has been such a strong selective pressure for biofilm growth in many different species of bacteria. Biofilms afford bacteria a protected community in which to grow, allow for great flexibility in metabolic needs, and facilitate rapid adaptation to an unimaginable number of environments.

Persistent Bacterial Infection

It has only been within the past decade that much attention has been directed toward the study of biofilms. This delay in attention was largely due to the misconception that pathogenic processes are a phenomenon associated with planktonic bacteria. Certainly, this was a bias that was firmly established in the days of the golden age of microbiology. Without any perceived clinical relevance, biofilms were simply left to be studied more as an academic discipline. Ironically, improvements in medical technology created new niches that were exceptionally well-suited for biofilm growth. With the advent of implanted medical devices and a growing population that was immunocompromised, there began an increasing trend in patients to become afflicted with baffling chronic infections.6,16 What was even more puzzling for doctors was the fact that treatment by conventional methods such as antibiotic therapy would yield only temporary relief of symptoms. The same infections would inevitably recur in a seemingly endless cycle of treatment and reinfection. To complicate matters further, when organisms were cultured from patients and plated as planktonic cells, they were typically found to be readily susceptible to conventional therapy.

Costerton recounted a case in which a patient was stricken with a recurrent bacteremia. The patient was treated for three weeks using 16g of cloxacillin per day. Inevitably, the treatment regimen would eliminate the bacteremia. However, upon termination of antibiotic therapy, there would be another cycle of infection. It was known that the patient had an old pacemaker, and therefore it was decided that the device would be removed. Upon removal, it was discovered that the pacemaker contained a thick biofilm of Staphylococcus aureus.17 The intense antibiotic therapy provided very little challenge to the cells growing on the device, and certainly the patient's own immune system was not capable of clearing

the infection in the protective biofilm.

This type of scenario was increasingly becoming more common as various medical devices such as catheters and heart valves were implanted into patients. Also, many different chronic infections of tissues within the body were discovered to be biofilm-related as well.18 In vitro biofilm experiments readily demonstrated the remarkable capability of biofilm bacteria to resist antibacterial therapies. In a comparison of planktonic P. aeruginosa with its biofilm counterpart, it was found that a treatment regimen of 50 µg/ml of tobramycin for eight hours was certain overkill for the planktonic bacteria. However, when the biofilm phenotype P. aeruginosa was given 1,000 µg/ml for an even longer time period, there was no significant reduction in biofilm viability.17

If the body is viewed as just one of many aquatic environments inhabited by biofilms, it makes sense that they are so resistant to different treatments. In the body, bacteria are able to employ the same mechanisms that protect them from microbial predation and chemical antagonism in the wild. In fact, it is commonly held that about 1,000 to 1,500 times more of specific antimicrobial agents are required to treat a biofilm-living bacteria as opposed to its planktonic form.6

Certain conditions such as cystic fibrosis are known to be biofilm-mediated diseases. In this particular disease, the immune system is unable to clear a biofilm infection, and the host's own immune system mechanisms are the cause of major tissue damage. This is largely a result of immune complexes accumulating on the outside of the biofilm.18

What properties permit such exquisite resistance to otherwise harsh environments? There are several explanations that are probably all factors contributing to the success of biofilm bacteria. The first is more of a physical limitation. Biofilms are surrounded by a thick, viscous layer of exopolysaccharide that makes penetration of antibacterial agents very difficult for a variety of reasons.19,20 The exopolysaccharide is analogous to a bullet-proof vest. If the bullets are not able to effectively penetrate the vest, then no real damage will occur.

Another resistance mechanism of biofilm bacteria stems from their metabolic capabilities. As was mentioned before, bacteria in the biofilm can tailor their metabolic needs based upon their own niche within the biofilm. Consequently, not all the bacteria in the biofilm are going to be particularly metabolically active. So, assuming an agent such as an antibiotic can actually penetrate the biofilm in any appreciable quantity, it may not be very effective against a metabolically inactive bacterium.18 In fact, many antibiotics are directed at some stage of bacterial metabolism.

Finally, there is some speculation that part of the biofilm developmental process includes differentiation into a phenotypically resistant organism. This is distinct from the metabolic changes that take place in response to nutrient availability. This theory is still largely speculative, but there has been some evidence suggesting a possible phenotypic resistance to tobramycin in the bacteria of younger biofilms.19

Nosocomial Infection through Medical Devices

In the hospital setting, it has been well-documented that biofilm bacterial species are a common source of nosocomial infection.17,18 This is not surprising given the extraordinary potential for adaptability seen in biofilm bacteria. In hospitals, there has been continued difficulties with the pathogens P. aeruginosa, Legionella pneumophila, and nontuberculosis Mycobacteria species, with P. aeruginosa alone accounting for 9 percent to 11 percent of all reported nosocomial infections in the United States.16 These bacteria are found primarily as environmental species, but all are well-suited for growth wherever there is water and a supply of nutrients.

Even with the constant attempts at cleanliness and sterility, there will always be some minute niche that will be overlooked or not properly sanitized. For instance, in 1991, the University of Wisconsin reported a 36 percent increase in nosocomial upper gastrointestinal infections following endoscopy at its hospital.21 Subsequent investigative culturing of the endoscopes revealed heavy contamination with gram-negative bacilli, mainly P. aeruginosa. Further investigation proved that the source of the contamination was actually the automated endoscope washer -- the machine designed to clean and disinfect endoscopes after usage. The machine was new and routinely cleaned and disinfected according to manufacturer's protocol.

Dental Plaque

While much effort has been devoted to the study of medically relevant single-species biofilms, one of the most commonly observed multispecies biofilms in humans has gone largely overlooked. This is dental plaque, which in many ways is proving to be a useful model for the study of multispecies biofilm interactions. While much of the knowledge gained as a result of single species biofilm research is also applicable to multispecies biofilms, there are many complexities that make multispecies biofilms unique.

An interesting phenomenon of oral bacteria is their tendency to consistently associate with a defined set of partners. When this association occurs in suspension, it is referred to as coaggregation, while a similar association in a biofilm setting is referred to as coadhesion.22 The ability of oral bacteria to associate with each other in a specific, defined manner becomes very important in the development of a dental biofilm. It appears that the formation of a multispecies dental biofilm is a process that occurs in discrete steps with certain groups of bacteria joining the biofilm at specific stages of biofilm development.

Many of the different Streptococcus species constitute the typical early

colonizers of the tooth surface. This is generally thought to occur via saliva-specific receptors on the surface of oral Streptococcus species.23 Fusobacterium nucleatum constitutes the next major colonizer of the dental biofilm through direct interactions with the early colonizing Streptococcus species. F. nucleatum is generally thought to be the microbial "glue" that can coaggregate with numerous oral bacteria and allow for many other late colonizers to join in the advanced stages of plaque development.22 Interestingly, F. nucleatum seems to be extremely important in the survival of different obligate anaerobic oral bacteria in both the planktonic and biofilm environments during aeration.24 It is an intriguing possibility that in a dental plaque this function could be exploited to bridge the gap between the oxygen-tolerant facultative anaerobes and the much less tolerant obligate anaerobes.

Indeed, there are numerous examples of complex interdependency among the different species in dental plaque. More defined interactions have been elucidated in numerous examples of metabolic cooperation between oral bacteria, and this cooperativity seems to be intimately associated with specific coaggregations and coadhesions.22 One of the reasons this may occur is for metabolic efficiency of the biofilm community as a whole. For instance, many oral Streptococcus species produce fermentable carbohydrates as a product of host serum glycoprotein degradation.25 These carbohydrates can then be fermented by other neighboring species of oral bacteria.22

Other examples of metabolic communication may be even more fundamental. The ScaA lipoprotein in S. gordonii functions both to scavenge for Mn+2 ions under limiting conditions and to act as a specific adhesin for various other oral bacteria. It has been proposed that ScaA may provide a source of Mn+2 for different bacteria unable to scavenge the divalent ion, and that this cooperativitity may be mediated through specific coaggregations utilizing ScaA.26 It would seem that the community aspects of multispecies biofilms are much more intricate than those of single species. In nature, it is thought that the majority of biofilms exist in a multispecies setting27 and therefore, future work on dental biofilms will likely yield new insights into the convoluted metabolic and signaling events occurring in other multispecies biofilms.

Dental Unit Water System

Many investigations have revealed numerous issues of safety concerning equipment, personnel, and patients in dental clinics. While the epidemiological data establishing the link between dental treatment and biofilm-related nosocomial infection is weak, there are reported cases of infection due to treatment.28 In particular, biofilms present an ever-growing concern due to their prevalence in dental unit water systems. While there seems to be little direct evidence suggesting dental unit water system biofilms as sources of nosocomial infection, there is a general concern that they may serve as potential reservoirs of infection, especially in the elderly and immunocompromised.16

Groups such as the American Dental Association Council on Scientific Affairs point out that the public should expect the highest standards of safety and sanitation from the modern dentist regardless of risk.29 For these reasons, in 1995 the ADA set forth a goal for dental unit water systems to deliver water with less than 200 colony-forming units/ml of unfiltered output water.29 In essence, the goal was to effectively remove biofilms from dental unit water systems by the year 2000. It is worthy of note however, that having less than 200 cfu/ml can underestimate the number of bacterial counts in a water sample, simply due to the fact that only a very small fraction of cells in a sample are able to form colonies on agar plates.30 In any case, this goal was to be the voluntary standard of acceptable cleanliness and was to be achieved through a combination of research into waterline-related issues as well as through a conscious effort to inform practitioners. In the five years since the statement, there has been a strong response from industry, and a wide array of products made specifically for dental units are available. These fall into one of four categories: independent water systems, chemical treatment methods, point-of-use filters, or sterile water delivery systems.29 The ADA Council on Scientific Affairs evaluates many such products submitted for its Seal of Acceptance Program.29

Despite the plethora of available products, some investigations into dental unit water systems in the clinical setting have yielded dismal results. In a recently published study in England, 55 water and tube samples were taken from actual dental surgeries. The aim was to assess relative cleanliness under real-use conditions as well to compare contamination from different types of dental unit water systems. In 95 percent of the samples taken, counts were higher than the ADA-recommended less than 200 cfu/ml standard. Of the 55 surgeries, waterline samples had an average microbial load of 2,900 cfu/ml, while air rotor waterlines contained an average of 3,300 cfu/ml. The surfaces of the waterlines contained an average biofilm coverage of 43 percent, while the corresponding air line value was 5.2 percent. Oral streptococci were identified in 7 percent of the samples, thus raising the possibility that some antiretraction devices may be inadequate. It is interesting to note that the authors were not able to show to statistical significance that there was any difference in microbial contamination between different types of dental unit water systems.30 This contradicts the belief some dentists may have that certain units are inherently cleaner than others.

It is not surprising that sampling efforts of dental unit water systems have produced numbers much higher than recommended values. Despite the best intentions of many dentists, there is

likely to be the assumption that typical decontamination protocols will be sufficient to provide clean water to their patients. When dealing with planktonic bacteria, surely these efforts would be sufficient. However, biofilm-dwelling bacteria present a unique situation that defies many of the classical microbiology dogmas.

Furthermore, it is an exceptional challenge to keep a closed system like the dental unit completely free of biofilm formation. Once a biofilm has been established, it is difficult to remove from the system. Indeed, biofilm removal has been a major issue plaguing other industries for years. It seems the most effective way of removing a biofilm is simply by using brute force and mechanically disrupting it. This is the same reason that scaling and root planning is so effective and why people benefit from brushing their teeth every day. In a closed system like a dental unit, mechanical disruption is not a time- and cost-effective method of biofilm removal. However, numerous recommendations have been made to help ameliorate the problem. Both the Centers for Disease Control and Prevention and ADA recommend the simplest of these.29 They advise dentists to flush their waterlines for two to three minutes before treating the first patient of the day in an effort to eliminate suspended bacteria in the water. It was also proposed that dentists flush their lines for about 30 seconds between patients, which helps to remove any bacteria that may have entered the waterlines during treatment.31,32 This should also prevent cross-contaminating bacteria between patients.

Numerous studies have been conducted to evaluate the effectiveness of accepted decontamination methods. High concentrations of disinfectants (such as formalin) have been shown to be effective at killing biofilm bacteria, however, they are largely ineffective at removing the biofilm matrix from the attached surface. In fact, there is some evidence they may even impede further cleaning efforts.33 Given the extreme toxicity of such compounds, there is a large concern that the biofilm will serve as a reservoir of toxic disinfectant that can be transmitted to the patient. In some instances, disinfection is achieved through cleaning waterlines with enzyme detergents.33 This can effectively remove the biofilm matrix from tubing, however, it should be noted that this can release many bacteria from the biofilm into suspension. In the previously mentioned study of 55 dental unit water systems, five of the units were recently decontaminated and all showed higher levels of bacterial contamination in the water samples.30 Direct sampling of the biofilms on the dental unit water systems tubing did confirm the presence of fewer biofilm embedded bacteria, however. Therefore, careful flushing of the waterlines is advisable if such a cleaning strategy is to be used because there is likely to be a transient worsening of water quality.

The most extensively investigated option is the periodic treatment of waterlines with a 1:10 solution of sodium hypochlorite.29 A recent study evaluated the effectiveness of such a treatment and concluded that weekly treatments with 1:10 NaOCl combined with the use of chlorinated water was sufficient to retard biofilm production and maintain the ADA recommended less than 200 cfu/ml.34 Hypochlorite-based bleach has also been demonstrated to aid in the removal of biofilms.33 It is not known whether such a treatment regimen has a detrimental effect on oral tissue or various dental procedures.34

Clearly, there are numerous options dentists may employ to achieve better dental unit water systems water quality. As has been demonstrated before, it should not be assumed that any device or treatment protocol is going to perform to expectations. Consider the scenario presented with the University of Wisconsin automated endoscope washer. Therefore, the ADA Council on Scientific Affairs has wisely suggested a routine sampling of waterline quality for patient safety as well as preventing dentist liability. They advise water samples to be taken before routine disinfection in order to assess maximum potential exposure.29 There are waterline testing kits available that are both cost-effective and easy to use. Their efficacy was demonstrated in a study that found a strong correlation between kit results and those obtained from laboratory methods.35 Given the present lack of definitive options, this may be the optimal strategy to monitor the quality of water to which patients and staff are subjected.

Summary

Bacteria form complex communities (called biofilm) on particular surfaces. Bacteria in biofilm survive better and exhibit stronger resistance to various environmental factors than do planktonic cells. This unique physiological status causes persistent bacterial infection or contamination of medical devices. Similarly, biofilm is also responsible for various oral bacterial diseases and contamination of dental unit water systems. The dental field has much to gain from molecular investigations of biofilm production. Novel methods of prevention and treatment of tooth decay and periodontal disease are inevitable outcomes from the biofilm research under way in many laboratories throughout the world. Future research will also undoubtedly yield new biofilm-resistant materials and/or safe chemical additives that disrupt biofilm formation. However, it is unlikely that scientists and practitioners will ever again take sanitation for granted, given the relentless persistence of biofilm bacteria.

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Periodontitis as a Biofilm Infection

Casey Chen, DDS, PhD

ABSTRACT Microbial biofilms are a major concern of infections associated with implantable medical devices as well as with many non-implant related chronic infectious diseases in humans. Dental plaque is also a biofilm. Dental plaque is probably one of the best characterized biofilms occurring on the surface of human tissues. This article will examine the impact of biofilm research on concepts of microbial etiology and the treatment implications for periodontitis.

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Biofilm formation has become a focus of intense research in recent years. Biofilms are the predominant mode of existence of most bacteria in nature.1 A biofilm can be defined as a community of bacteria embedded in exopolysaccharide that adheres onto an inert or living surface. The ubiquitous biofilms are found on the surfaces of rocks, oil and water pipelines, sewage treatment systems, plants, animals and humans. Microbial biofilms are a major concern of infections associated with implantable medical devices as well as with many non-implant related chronic infectious diseases in humans.2

Dental plaque is also a biofilm. Dental plaque is probably one of the best characterized biofilms occurring on the surface of human tissues. From the perspective of conducting scientific research, dental plaque has the advantages of being universally present on tooth surfaces, being easily accessible, and containing sufficient complexity for researchers to examine the basic principle of biofilm formation. Historically, the studies of dental plaque and the studies of biofilms have had different emphases. The time seems ripe for examining the impact of biofilm research on concepts of microbial etiology and the treatment modalities of periodontitis.

Teleology of Biofilms

The terms sessile and planktonic are often used to distinguish the two modes of existence of bacteria. Sessile bacteria refer to the attached bacteria in biofilms.

Planktonic bacteria are free-floating single-cell bacteria. Many microbial species exist predominantly as sessile bacteria but release small numbers of planktonic cells for dispersion. The question of why bacteria form biofilms may have many answers (speculations). Several possible reasons are described below.

Homeostasis. Immobilized biofilms offer some semblance of stability for bacteria with regard to nutrient availability, temperature, mineral concentration, oxygen concentration, and pH. In a relatively stable environment, sessile bacteria need not waste energy in reacting to the constantly changing conditions that would be encountered by free-floating planktonic cells.

Growth and metabolic dependence. Synergistic relationships have been observed among different bacterial species forming biofilms.3 A bacterial species may create a favorable environment, e.g., an anoxic condition, to support the growth of other organisms, e.g., obligate anaerobic bacteria. The presence of one bacterial species may provide additional adherence sites for other bacteria through coaggregation.4,5 Different bacteria in biofilms may be metabolically dependent on each other. For example, vitamin K produced by certain Prevotella species is used by subgingival Porphyromonas gingivalis for growth.6

Protection. Biofilms can protect sessile bacteria from harmful substances. Exopolysaccharide appears to be an essential component of biofilms. An important function of exopolysaccharide is the protection of the sessile bacteria from antibiotics and bactericides.7-9

Diversification as a survival strategy. Bacteria of the same species localized in different parts of the biofilm are in different physiologic states and express different phenotypes. The phenotype diversification may help the survival of the bacteria if the environment changes suddenly. Bacteria may also acquire new genetic traits to increase diversities. Genetic exchange has been shown to occur in biofilms.10 Close proximity of different bacterial species in biofilms may facilitate genetic exchange and provide bacteria opportunities for acquisition of new genetic traits.

Selected Characteristics of Microbial Biofilms

A thorough discussion of biofilm characteristics can be found in several excellent reviews of this topic.1-3 The following review of selected features of biofilms is intended to provide a framework for the subsequent discussion of dental plaque as a biofilm.

Microscopic structural characteristics. The study of biofilm structure used to be performed predominantly by electron microscopy. A major drawback of electron microscopy studies is the need to dehydrate samples, which may distort the spatial relationship among bacteria in biofilms. Also, the structure of the loose, highly hydrated exopolysaccharide in biofilms is not usually revealed by electron microscopy. As a consequence, such studies may show biofilms to be a collection of random aggregates of bacteria without distinct features.

With the advent of the digital imaging devices such as confocal scanning laser microscopy and the use of various nontoxic fluorescent probes, fully hydrated bacterial biofilms can be observed in situ.11 Biofilms display distinct architectural structures that consist of a variable distribution of cells and cell aggregates, the associated exopolysaccharide, void spaces, and water channels.1,3 The existence of void spaces and water channels allows the diffusion of nutrients and waste products. The detailed structures of biofilms vary among different bacterial species and also are dependent on growth conditions.

The microscopic study of biofilms has entered an exciting era in which different technologies converge to give a more realistic picture of biofilms. For example, fluorescent in situ hybridization utilizes specific fluorescence-tagged DNA probes to localize individual bacterial species in biofilms.12 The combination of fluorescent in situ hybridization and confocal scanning laser microscopy may be used to map the location of target bacterial species within a mixed-species biofilm in reconstructed three-dimensional images.

Cell-to-cell communication. Bacteria can coordinate with each other and act as a group when a critical density of bacterial cells is reached. Research on quorum sensing systems showed that the vast majority of gram-negative bacteria utilize N-acyl homoserine lactones as signaling molecules.13,14 The signaling molecules are produced at a low level by each bacterium and are freely diffusable through bacterial envelopes. When the N-acyl homoserine lactones are accumulated and reach a critical threshold value (due to higher cell densities), the molecule triggers and activates a transcriptional activator that in turn induces the expression of target genes. Quorum sensing systems have been shown to play a role in biofilm formation.15 P. aeruginosa has two quorum sensing systems, the lasR-lasI and the RhlR-RhlI systems. Specific inactivation of the lasR-lasI system allowed the mutant to adhere to a glass surface, but rendered the mutant unable to form a mature, structured, thick, and biocide-resistant biofilm.15 There is evidence that the N-acyl homoserine lactones are produced by P. aeruginosa biofilms in vivo.16 The involvement of quorum sensing systems in biofilm formation seems logical. Genes involved in biofilm formation, which requires increase of cell density and cell-to-cell contact, may be useless if only few cells in planktonic phase exist.

Resistance to antimicrobial agents. Sessile bacteria exhibit several hundred- to thousand-fold higher resistance to antimicrobial agents in comparison to planktonic cells of the same species.2,7 Several hypotheses have been proposed to explain the mechanisms of the increased resistance.2,7-9 Exopolysaccharide may retard the diffusion of certain antibiotics and biocides. Some of the sessile bacteria are slow-growing or in a dormant state

due to nutrient limitations. The starved cells are metabolically inactive and may be more resistant to antibiotics. It is also speculated that some sessile bacteria display a distinct biofilm phenotype that is inherently resistant to antimicrobial agents. The expression of this resistance phenotype is a result of a programmed response to growth in a biofilm and is not related to the starvation or the metabolic activity of the bacteria. The resistance phenotype persists even after the sessile cells are removed from biofilms and grown in enriched media.

Table 1. Consensus Periodontal Pathogens[38]

Strong Evidence	Moderate Evidence	Initial Evidence
Actinobacillus actinomycetemcomitans	Campylobacter rectus	Eikenella corrodens
Porphyromonas gingivalis	Eubacterium nodatum	enteric rods
Bacteroides forsythus	Fusobacterium nucleatum	Pseudomonas
	Prevotella intermedia/ nigrescens	Selenomonas
	Peptostreptococcus micros	Staphylococcus
	Streptococcus intermedius-complex	yeasts
	Treponema denticola	
	Spirochetes	

Microbial Etiology of Periodontitis

Periodontitis is a chronic bacterial infection that affects supporting structures of the teeth, including gingivae, periodontal ligament, and alveolar bone. The disease is one of the most common bacterial infections among humans.17 A tremendous effort has been directed toward identifying the bacterial causative agents of periodontitis and understanding their pathogenic mechanisms.18 It should be remembered that in scientific research, answers are often influenced by the way in which the questions are framed. In the following discussion, the conventional viewpoint of microbial periodontal etiology is presented.

Conceptual framework of infectious diseases. To understand infectious diseases, it is easier to use as a model an acute infectious disease in which a single pathogen is the sole cause. A good example may be primary syphilis, in which the causative agent, Treponema pallidum, invades the genitalia of the infected individual and causes a localized lesion called chancre. The disease occurs within days (i.e., acute) of infection by T. pallidum. No other bacterial species are involved in the pathogenesis of syphilis. Nor does biofilm formation play a role in the infections. Once the role of T. pallidum in syphilis is defined, subsequent studies may be devoted to examining the detailed pathogenic mechanisms. Attention may focus on identifying the bacterial virulence factors, the host immune response, and how each parameter modulates the disease progression. In general, medical science has a relatively good grasp on the pathogenesis of single-pathogen infectious diseases and is largely successful in treating this type of acute bacterial infection with antimicrobial therapy. However, there are other bacterial infectious diseases operating under different principles.

Consensus list of periodontal pathogens. The causal relationship between oral bacteria and periodontitis is difficult to determine for a number of reasons.19 Briefly, more than 500 taxa of oral bacteria may be cultivable from gingival crevices.20 There may be several hundred more noncultivable bacteria in gingival crevices as well. These noncultivable bacteria may be detected and characterized by polymerase chain reaction cloning and sequencing of their 16S rRNA genes.21,22 The sheer numbers of different gingival bacteria needed to be evaluated may overwhelm anyone trying to decide whether a bacterium, or a consortium of bacteria, is the cause of periodontitis. Furthermore, periodontitis is typically, but not always, a slowly progressing disease. There is a time gap between the initial infection by the periodontal pathogens and the clinical manifestation of the disease. The causal relationship between the bacteria and the disease becomes obscure. Also, many oral bacteria are considered commensal organisms due to their low pathogenicity and frequent occurrence in healthy individuals. Yet, the commensal bacteria may cause periodontitis under the "right" condition such as poor oral hygiene, poor host immune response, or deepened periodontal pockets. It is difficult to determine when commensal organisms play the role of nonpathogens and when they play the role of causative agents in the disease. Finally, it would have been relatively easy to substantiate the etiologic role of a putative periodontal pathogen if the bacteria could be selectively eradicated by periodontal therapy. However, periodontal therapy is usually accompanied by a major change in subgingival microbiota. The etiologic role of bacteria cannot be inferred from the clinical outcomes of periodontal treatment. In spite of these difficulties, one of the most remarkable achievements in periodontal research is the delineation of the role of a number of specific bacterial species in periodontitis. This is due in part due to advancements in anaerobic culture techniques, bacterial taxonomy, and molecular identification and characterization of subgingival

Table 2. Partial List of Human Infections Involving Biofilms[2]

Infections	Biofilm Property
Dental caries	Mutans streptococci, actinomycetes
Periodontitis	Subgingival dental plaque
Otitis media	Nontypeable Haemophilus influenzae
Native valve endocarditis	Viridans group streptococci
Cystic fibrosis pneumonia	Pseudomonas aeruginosa and Burkholderia cepacia
Infections of implantable devices	
Contact lens	*P. aeruginosa* and gram-positive cocci
Urinary catheter cystitis	*E. coli* and other gram-negative rods
IUDs	*Actinomyces israelii*
Central venous catheters	*S. epidermidis*
Orthopedic devices	*S. aureus and S. epidermidis*

bacteria. It is worth noting that the following consensus list of important periodontal pathogens was derived using a set of modified Koch's postulates as proposed by Socransky.23 A periodontal pathogen should possess the following characteristics:

- **Association with disease.** The bacterium should be present at high levels in diseased individuals and either absent or present in lower levels in healthy controls.
- **Elimination of the organism.** Elimination or suppression of the organism should result in the arrest of the disease.
- **Host response.** Increased or decreased host immune response to a specific bacterium is suggestive of a significant role of the bacterium in disease.
- **Animal pathogenicity.** The pathogenicity of the bacterium could be inferred from the ability of the bacterium to cause disease in experimental animals.
- **Mechanisms of pathogenicity.** The bacterium should possess characteristics that could contribute to the pathogenesis of periodontal disease.

The resultant list of periodontal pathogens24 is shown in **Table 1**. The bacteria are categorized based on the strength of the evidence compiled from numerous clinical and microbiological studies.

Periodontitis as a Biofilm Infection

The modified Koch's postulates, the resultant list of periodontal pathogens, and the implied concept of microbial etiology of periodontitis are influenced by the conceptual framework of single-pathogen acute infections caused by planktonic bacterial cells. However, periodontitis is more similar to bacterial biofilm infections than to acute infections. A list of selected biofilm infections in humans is listed in **Table 2**.

Biofilm infections share some common features.2 A critical early step of the disease involves the forming of biofilms on inert surfaces or living tissues. Commensal bacteria are frequently involved in biofilm infections. Clinically, biofilm infections are slow to progress and difficult to treat. The bacteria in biofilm infections are frequently resistant to antimicrobial agents that are effective against planktonic bacteria. The bacteria and the infections relapse after the cessation of the drug therapy. The host immune response is ineffective against biofilm infections and may even be harmful to the host. All these characteristics of biofilm infections apply to periodontitis.

The view of periodontitis as a biofilm infection does not change the importance of the consensus periodontal pathogens. It does modify the view of microbial etiology and may have some influence over the treatment modality of periodontitis. Dental plaque may be viewed as being a primitive multicellular organism. Some part (readers: periodontal pathogens) of this multicellular organism causes great damage to periodontal tissue. Other parts of the primitive multicellular organism may be essential for the survival of the entire organism. The goal of periodontal therapy will remain the elimination of periodontal pathogens. Nevertheless, one may choose to target the weaknesses of the primitive multicellular dental plaque (which may not be the pathogens) to achieve the goal.

Dental plaque is a ubiquitous structure formed on the surface of oral tissues by oral bacteria.25,26 Dental plaque is made up predominantly of bacterial cells but also includes other minor components such as bacterial enzymes, bacterial metabolic products, host salivary proteins, immunoglobulins IgG and IgA, and complement components. This paper accepts a priori that dental plaque is a biofilm. Nevertheless, it will be helpful to review the supporting evidence for dental plaque as a biofilm. The following discussion begins with a description of the orderly process of plaque formation, followed by a brief review of the biofilm features of dental plaque.

Orderly process of plaque formation. More details have been learned about the formation of dental plaque than any other naturally formed biofilm. Plaque formation is an extremely complex process. Several excellent reviews are available for interested readers.6,26,27 Only a brief description of the process is provided here. Immediately after removal of bacteria on the tooth surface by prophylaxis, a ubiquitous layer of dental pellicles is formed on the tooth surface.

The early bacterial colonizers, mostly facultative gram-positive streptococci and Actinomyces species, adhere to the dental pellicles on the tooth surface. Following the adherence of early colonizers, the plaque increases its cell numbers mainly by bacterial growth. The early colonizers provide a variety of niches for the adherence and growth of late bacterial colonizers. The plaque continues to increase in thickness by both adherence and bacterial growth. The microbial composition of plaque gradually becomes more diversified and includes an increasing number of gram-negative bacteria and obligate anaerobic organisms. The inter-species and inter-generic bacterial coaggregations play a significant role in the plaque maturation process.4,5,28 After several days to two to three weeks, the plaque reaches its full potential and becomes a mature bacterial community.

The orderly process of plaque formation suggests specific interactions. Many bacteria express pili (fimbriae), which are proteinacious hair-like structures projecting from the bacterial surface. Bacteria may also express nonfimbrial adhesins on the cell envelope. Both types of adhesins mediate the attachment of the bacteria to receptors in dental pellicles or on the surface of other bacteria. For example, Actinomyces naeslundii expresses two types of pili, type 1 pili mediate the attachment to dental pellicles and type 2 pili bind streptococci.29 The specific interactions are also found between salivary components of dental pellicles and the early colonizers. α-amylase binds Streptococcus gordonii.26 The salivary protein statherin binds Actinomyces viscosus.26 Proline-rich proteins also mediate the adherence of a number of actinomyces and streptococci.26 It is important to recognize that the specific interactions in the plaque formation may provide some opportunities to disrupt the formation process.

Microscopic structural characteristics of dental plaque. Electron microscopy studies have some drawbacks in identifying the structural characteristic of biofilms. Nevertheless, it is worth revisiting the seminal work of these type of studies of dental plaque by Listgarten.30,31

With electron microscopy, the mature supragingival plaque appeared as a layer of dense and predominantly filamentous organisms adhering to the enamel surface (Figures 1 and 2). The filamentous organisms were long and oriented with their longitudinal axis perpendicular to the tooth surface. The bacterial cells were held together by extracellular matrix. The surface of the plaque contained distinctive corncob formations indicative of interbacterial species coaggregation (**Figure 3**). The subgingival plaque is a natural extension of supragingival plaque. There was a gradual change from the dense, thicker, predominantly filamentous supragingival plaque, to the thinner, less densely packed, and less organized subgingival plaque (Figures 4 and 5). The adhering layer of the subgingival plaque contained short filamentous bacteria. The surface of the subgingival adherent layer was covered with distinctive bacteria comprising many flagellated bacteria and spirochetes. The surface of the subgingival adherent layer also contained bristle brush and test-tube brush formations (**Figure 6**). The presence of exopolysaccharide in subgingival plaque was also evident.

The supra- and subgingival plaque shown in electron micrographs appear to be more compact than the single-species biofilms examined by confocal scanning laser microscopy in vitro. The lack of void spaces and water channels may be an artifact due to the collapse of bacterial aggregate from dehydration during the sample preparation or a result of a higher nutrient contents in gingival crevices supporting the growth of a higher density of bacteria. Also, the presence of numerous different bacterial species, each favoring a different ecological niche, may reduce the voids in the biofilms. Nevertheless, it is clear that dental plaque exhibits an orderly structure. The distribution of different bacterial morphotypes in dental plaque shows discernible patterns.

More recently, confocal scanning laser microscopy has been employed to examine the structure of natural dental plaque. Wood and colleagues32 examined the architecture of 4-day-old dental plaque formed in volunteers wearing a device attached to the buccal surface of molars. The device contained an enamel substrate to allow for plaque formation. The confocal scanning laser microscopy results showed a highly heterogeneous distribution of bacterial mass. There were void spaces throughout the plaque, and some appeared to open to the surface of the plaque, allowing exchange of fluids. Large bacterial masses resembling mushroom structures surrounded by open spaces and channels were also observed.

These microscopic structural studies presented above support the concept that supra- and subgingival plaque are biofilms. The naturally formed dental plaque shows more complexity than in vitro biofilms of single or limited species, and it may have different structural characteristics. The studies also suggest that specific interactions were involved in plaque formation, as would be expected from biofilms.

Cell-to-cell communication in dental plaque. It is reasonable to assume that bacteria in dental plaque may utilize certain cell-to-cell communication systems in order to coordinate their behaviors. Although direct evidence for bacterial communication in plaque is lacking, cell-density-dependent behaviors of oral bacteria have been demonstrated in naturally formed subgingival plaque on the tooth surface. Bloomquist and colleagues33 examined bacterial growth patterns of the plaque formed from two to 24 hours on enamel pieces placed on the tooth surfaces of healthy volunteers. Following rapid adherence of oral bacteria onto the enamel surface to a density of 2.5 to 6.3x105 cells/mm, there was a period of relatively slow cell growth. A rapid burst of cell growth occurred when the cell density reached the level of 2.5 to 4 x

106/mm, and the growth rate declined at densities beyond 6.3 x 106 cells per mm2. It was postulated that the cell-density-dependent burst of cell proliferation was a demonstration of cell-to-cell communication. The mechanism of this postulated cell-to-cell communication was not known. There may be a tremendous difference in behavior between young plaque and mature plaque. Therefore the issue of biofilm behavior of dental plaque is not settled by the previous studies.

Antimicrobial resistance of bacteria in dental plaque. A limited number of in vitro studies showed that oral streptococci in biofilms were more resistant to chlorhexidine in biofilms than planktonic cells.34,35 The resistance phenotype of sessile subgingival bacteria may also be inferred from clinical studies of adjunct systemic antibiotics therapy for periodontitis.36-38 Although antibiotic therapy resulted in a proportional increase of subgingival bacteria resistant to the corresponding drugs, the antibiotics did not eliminate all susceptible subgingival bacteria. While there are many plausible explanations, these findings may be explained by the higher resistance of sessile bacteria than planktonic cells to antibiotics.

Treatment Implications

The concept of microbial etiology of periodontitis has undergone a tremendous change from nonspecific plaque hypothesis in which the quantity of the plaque is considered critical, to the specific plaque hypothesis in which a limited number of periodontal bacterial species (i.e., the quality) are recognized as the cause of the disease. Still, the available treatment modalities remain essentially nonspecific. Plaque removal, commonly achieved by scaling and root planing, is an important step in periodontal therapy. Periodontal osseous surgery eliminates deep pockets to allow effective plaque removal regiments (i.e., brushing and flossing). Various chemical agents with

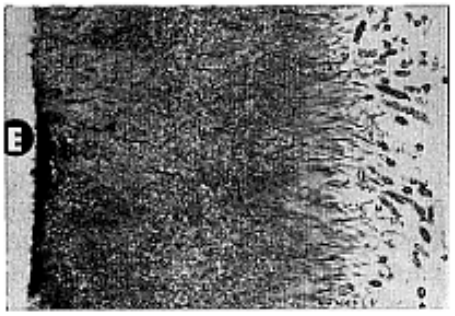

Figure 1. Supragingival plaque of a periodontitis patient. The enamel surface (E) is on the right side of the micrograph. A dense layer of bacteria, mostly filamentous, adheres to the enamel. Corncob formations are visible on the surface of the plaque. 850x magnification. From Listgarten,30 used with permission.

Figure 2. Magnified view (1,500x) of the adherent bacterial layer in Figure 1. From Listgarten,30 used with permission.

Figure 3. Magnified view (1,500x) of corncob formations on the surface of the adherent bacterial layer in Figure 1. From Listgarten,30 used with permission.

Figure 4. Subgingival plaque of a periodontitis patient. The cementum surface (C) is on the right side of the micrograph. Bacteria adhere to the cementum surface appear to be less dense and less filamentous. The test-tube brush formations are found on the surface of the adherent bacterial layer. 600x magnification. From Listgarten,30 used with permission.

Figure 5. Magnified view (2,000x) of the surface of the adherent bacterial layer in Figure 4. MC: Mammalian cells, mostly macrophages and neutrophils, adhere to the bacterial layer. From Listgarten,30 used with permission.

Figure 6. Magnified view (1,500x) of the test-tube brush formations. From Listgarten,30 used with permission.

broad spectrum of antimicrobial activities are used for oral rinsing and subgingival irrigation. Even systemic or local delivery of antibiotics, which markedly modify the composition of the subgingival microbiota, can be considered a nonspecific therapy. Eradication of selective, specific periodontal pathogens has always been the "unspoken" or "unachievable" goal of periodontal therapy.

Biofilm research is at an infancy stage and has not resulted in a noticeable change in periodontal treatment modalities but will have an impact to the future periodontal treatment. **Table 3** provides an outline of clinical implications based on the biological properties of biofilms. It should be noted that the goal of eradicating periodontal pathogens and the importance of microbial diagnosis will remain unchanged. What will likely change is how these periodontal pathogens are controlled.

The first four biofilm properties listed in **Table 3** suggest the idea that it may be possible to interfere with the plaque formation process and/or weaken the plaque structure. It could be done by killing key bacterial members of the plaque, by blocking the specific interactions between bacterial and host molecules, by disrupting cell-to-cell communication, and by attacking non-cellular structural components of the biofilms with chemical agents. In particular, the idea that the disruption of bacterial cell-to-cell communications may interfere with biofilm formation is the most promising and exciting area of research. It may one day be possible to use subgingival irrigation solutions containing inhibitors of the bacterial communication pathways to interfere with plaque formation. The fifth biofilm property suggests that antimicrobial agents alone are often ineffective in treating periodontitis without concomitant plaque removal by mechanical means. Biofilm research has further identified additional means to help remove biofilms. For example, electric current applied to biofilm has been shown to reduce the resistance of biofilm bacteria to antimicrobial agents.39 The last biofilm property suggests that vaccination may not be a good strategy of periodontal therapy. It may even suggest that elevated immune responses to bacterial biofilms may be potentially harmful to the host. Modulating host immune response to biofilms may reduce tissue damages in disease.

Table 3. Clinical Implications of Periodontal Therapy From the Perspective of Periodontitis as a Biofilm Infection

Biofilm Property	Clinical Implication
1. Biofilms behave as a living community or a primitive multicellular organism.	Changing part of the biofilms may influence the survival of the target periodontal pathogens. Certain key members of the dental plaque community may be relatively easy to remove (the weak links). The removal of these key members may result in a collapse of the dental plaque community which can no longer support bacterial pathogenic species.
2. Biofilm formation involves an orderly process.	Plaque formation may be modified by interfering with the specific interactions involved in this process. For example, antagonists of bacterial adhesins may be used to prevent the target bacteria from becoming a member of the subgingival plaque.
3. Biofilm formation requires cell-to-cell communication and coordinated behavior among individual bacterial members.	The obstruction of the cell-to-cell communication may disrupt plaque formation. It seems possible that inter-bacterial communications may be blocked by the use of antagonists of the signal molecules.
4. Biofilms contain structural characteristics which are not present in planktonic cells.	Some of the structural components of biofilms may be a good target of periodontal therapy. For example, exopolysaccharide is an integral structural component of the biofilm and also offers protective functions for bacteria. Removal of exopolysaccharide by chemical agents may weaken biofilm structural integrity and sensitize the bacteria to antimicrobial agents.
5. Sessile bacteria are resistant to antimicrobial agents.	Antimicrobial agents are not a substitute for thorough scaling and root planing. Removal of mature plaque remains a critical step of periodontal therapy.
6. Biofilm infections are resistant to host immune response	Vaccination against periodontal pathogens may not be a good strategy for the prevention or treatment of periodontitis.

Conclusion

Biofilms are the preferred mode of growth for many bacteria in nature, including periodontal pathogens. Although early periodontal microbiologists did not use the term "biofilms" to describe dental plaque, they clearly noted the distinct structure of the plaque, the orderly process, and the specific interactions involved in plaque formation. These are now considered salient features of biofilms.

The previous focus of the periodontal microbiology research has been in identifying and classifying subgingival bacteria and delineating the causal relationship of the bacteria and periodontitis. The consensus list of periodontal pathogens represents a remarkable achievement of dental research. The next breakthrough will likely result from the convergence of different disciplines of biofilms research. Ultimately, it may be learned how individual periodontal pathogens cause periodontitis, how the dental plaque

behaves as a community, what is the most effective means to disrupt the plaque formation, and what methods could be used to convert pathogenic plaque to one compatible with oral health.

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Educating Patients With the Speed-Sell

Robert E. Horseman, DDS

Man goes into a store to buy a tie. He emerges 90 minutes later wearing the expression of a stunned mullet. His sales receipt indicates he was sold a complete wardrobe -- a three-piece suit, shirt, socks, underwear and cufflinks. He re-enters the store. Forgot the tie.

Woman goes in to purchase a new handbag. Two hours later, she has matching pumps, lingerie, and a darling frock with mix-and-match accessories to die for. And a hat -- no, two hats -- plus some cologne, body lotion and appropriate jewelry.

What does this tell us? Are these people victims of their own feebleminded-ness? Exploitation by avaricious sales-clerks? No, of course not! They have been *educated*. The education has been done altruistically by people with specialized knowledge of what the customer needs.

It is a win-win proposition. The education is in the consumer's best interest, because frequently the customer doesn't know what he needs. What he wants is subject to whimsy. What he needs is *guidance*. In providing that guidance -- that education -- the store wins, incidentally, by making a tidy profit.

Or maybe not so incidentally. This has been SOP in the retail world since Day One. What is depressing is how long it has taken dentistry to recognize how pitifully inadequate our attempts to educate our patients have been. We've been dedicating our efforts into explaining what they need. How many patients want what they need? Why not education based on want rather than need, the marketing mavens ask. Seems to work for everybody from Tiffany & Co. to Burger King.

Imagine this scenario if you can: Patient comes in for a prophy, that's all. She wants her teeth cleaned, she needs her teeth cleaned; wants and needs neatly balanced. Cost: (she thinks) about $50. One hour later, she has had her teeth cleaned; had impressions made for tooth-whitening splints; and had her shopping bag filled with a tongue scraper, a home hygiene maintenance kit consisting of fluoride rinses, anti-halitosis agents with a volatile sulfur measuring device, two kinds of floss, assorted vitamins, whitening splints, a month's supply of bleaching materials, a shade guide to confirm her bleaching progress, and a handful of referral cards to hand out to her friends. She is wearing the expression of a stunned mullet. Cost: about $500 (for the stuff -- the expression is free), but she has been *educated*, and the cost of education can sometimes be a little high as parents of college kids can affirm.

The above scenario, according to brochures, fliers and product report maga-

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zines deluging our desk, is becoming more common as forward-looking dentists seek innovative ways to educate their patients with the avowed purpose of improving their oral well-being.

In other professions, this is called the "speed-sell." One would think that long experience with used car and aluminum siding salesmen would inure people to some extent from blandishments of this nature. But it is sometimes difficult for the consumer to tell where the education leaves off and the speed-sell begins, so closely and skillfully are they interwoven. If the ostensible purpose of the message is to improve or safeguard his health, it's hard for the patient/consumer to argue with the messenger.

That's why a customer will drive away in his new car with $10,000 worth of leather eight-way power seats and dealer-enhanced pin striping he really didn't know he needed. That's why one dentist can insist that 100 percent of his patients receive the bleaching procedure as a part of their treatment plan and another has a hygienist so adept at speed-selling that he had to inaugurate an intricate extended payment plan to handle the $20,000 extra a month she generates.

Is any of this unethical by any stretch of imagination? Well, hardly, if you consider that an educated patient is better prepared to make intelligent choices. After all, nobody held a gun to his head. Perhaps it all depends on the curriculum and who is doing the educating.

Maybe our comfort level with high-powered marketing will increase with time. Shoot, even the general acceptance of global warming and presidential perjury took a while.