Dental Academy of Continuing Education™

EARN **3 CE CREDITS**

This course was written for dentists, dental hygienists, and assistants.

Pathways to Gingivitis Control with Stabilized Stannous Fluoride: A Novel Discovery

A Peer-Reviewed Publication

Written by Sherri M. Lukes, RDH, MS, FAADH

PUBLICATION DATE:	JULY 2018
EXPIRATION DATE:	JUNE 2021

PennWell®

SUPPLEMENT TO PENNWELL PUBLICATIONS

EARN
3 CE
CREDITS

Pathways to Gingivitis Control with Stabilized Stannous Fluoride: A Novel Discovery

EDUCATIONAL OBJECTIVES

1. Identify risk factors associated with gingivitis.
2. Explain the mechanisms of action by which antimicrobials reduce gingivitis.
3. Define the mechanism by which stannous fluoride reduces the inflammatory response.
4. Discuss implications for patient care.

ABSTRACT

Gingivitis continues to be prevalent, with nine out of ten adult Americans exhibiting symptoms of mild or greater severity. Its occurrence and severity are influenced by multiple factors, including the bacterial composition of plaque biofilm and the host response. Antimicrobial agents are often recommended for gingivitis patients to reduce the quantity of bacteria in the biofilm and/or inhibit bacterial metabolism. New research has shown stannous fluoride—the only fluoride with antimicrobial properties—also improves gingival health by reducing the toxicity of plaque, even in the gingival sulcus, through interference with the host response. Specifically, stannous fluoride binds to the bacterial endotoxins and prevents their interaction with gingival tissue receptors associated with inflammation. This mechanism is important as it supports the use of stabilized stannous fluoride dentifrice not only in patients with existing gingival bleeding and inflammation, but also in patients who may be susceptible to it.

Dental Academy of Continuing Education™

Go Green, Go Online to take your course
www.DentalAcademyofCE.com
QUICK ACCESS CODE 15318

This educational activity was made possible through an unrestricted grant from Procter & Gamble.

This course was written for dentists, dental hygienists and assistants, from novice to skilled.

Educational Methods: This course is a self-instructional journal and web activity.

Provider Disclosure: PennWell does not have a leadership position or a commercial interest in any products or services discussed or shared in this educational activity nor with the commercial supporter.

Requirements for Successful Completion: To obtain 3 CE credits for this educational activity you must pay the required fee, review the material, complete the course evaluation and obtain a score of at least 70%.

CE Planner Disclosure: Heather Hodges, CE Coordinator does not have a leadership or commercial interest with products or services discussed in this educational activity. Heather can be reached at hhodges@pennwell.com

Educational Disclaimer: Completing a single continuing education course does not provide enough information to result in the participant being an expert in the field related to the course topic. It is a combination of many educational courses and clinical experience that allows the participant to develop skills and expertise.

Image Authenticity Statement: The images in this educational activity have not been altered.

Scientific Integrity Statement: Information shared in this CE course is developed from clinical research and represents the most current information available from evidence based dentistry.

Known Benefits and Limitations of the Data: The information presented in this educational activity is derived from the data and information contained in reference section. The research data is extensive and provides direct benefit to the patient and improvements in oral health.

Registration: The cost of this CE course is $59.00 for 3 CE credits.

Cancellation/Refund Policy: Any participant who is not 100% satisfied with this course can request a full refund by contacting PennWell in writing.

Academy of General Dentistry PACE Program Approval for Continuing Education

PennWell designates this activity for 3 continuing educational credits.

Dental Board of California: Provider 4527, course registration number CA#03-4527-15318
"This course meets the Dental Board of California's requirements for 3 units of continuing education."

The PennWell Corporation is designated as an Approved PACE Program Provider by the Academy of General Dentistry. The formal continuing dental education programs of this program provider are accepted by the AGD for Fellowship, Mastership and membership maintenance credit. Approval does not imply acceptance by a state or provincial board of dentistry or AGD endorsement. The current term of approval extends from (11/1/2015) to (10/31/2019) Provider ID# 320452.

ADA CERP® | Continuing Education Recognition Program

PennWell is an ADA CERP recognized provider

ADA CERP is a service of the American Dental Association to assist dental professionals in identifying quality providers of continuing dental education. ADA CERP does not approve or endorse individual courses or instructors, nor does it imply acceptance of credit hours by boards of dentistry.

Concerns or complaints about a CE provider may be directed to the provider or to ADA CERP at www.ada.org/goto/cerp.

PennWell® dental CE digest™

GINGIVITIS: IMPACT AND AWARENESS

All dental professionals are aware that gingivitis—an inflammatory periodontitis precursor—is characterized by red, edematous gingival tissues that bleed upon provocation (Figure 1). This extremely common condition is estimated to affect 50% to 90% of adolescents and adults globally.[1]

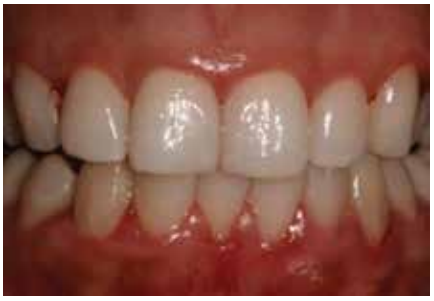


Figure 1. The cardinal signs of gingivitis are red, edematous, bleeding gums. (Image courtesy of dentalcare.com)

Given the wide accessibility of health information, it is surprising that many individuals seem unaware or unconcerned about the symptoms of gingivitis. In a systematic review of six studies involving almost 8,000 adults, substantial knowledge gaps were found in gingivitis/periodontitis disease awareness (80%); etiology (75%); related risks (71%); and signs and symptoms, risk factors, and treatment (50% each).[2] The questions "*What causes gum disease?*"; "*If your gums bleed, does this worry you?*"; and "*If your gums bleed, what do you do about it?*" revealed high unawareness in one trial, while elsewhere respondents thought bleeding gums while brushing was normal, and tooth loss was a natural part of aging or a fate decided at birth.[2]

The clinical assessment of the gingiva includes an evaluation of the color, consistency, and contour of the tissues for signs of inflammation. These more subjective parameters may be missed by a patient, whereas bleeding when expectorating after brushing is harder to overlook. In-office bleeding upon probing is also an objective disease measure, and the outcome can be shared as a springboard for discussing gingivitis causes and treatment strategies, along with the risks of disease progression with inaction.

GINGIVITIS ETIOLOGY AND RISK FACTORS

Löe and colleagues' ground-breaking induced gingivitis experiments in the 1960s illustrated how gingival tissues became inflamed and bled within days after cessation of oral hygiene, but were restored to health with the resumption of mechanical plaque removal.[3,4] Subsequent research has consistently confirmed that undisturbed plaque can produce deleterious effects on the gingiva and promote periodontitis in susceptible individuals.[5-7] This occurs because the plaque biofilm in the gingival sulcus harbors microbes that produce inflammatory by-products, and becomes increasingly virulent if left to proliferate (Figure 2).

As plaque matures, anaerobic gram-negative bacteria predominate in the subgingival microbiota, with species such as *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tannerella forsythia*, *Campylobacter rectus*, and *Aggregatibacter actinomycetemcomitans* colonizing the deposits.[7-9] Eventually, via their direct release of toxins, lipopolysaccharides (LPSs), or enzymes—or through the indirect triggering of the host's inflammatory systems—the plaque pathogens can instigate tissue destruction.[10] Additionally, lipoteichoic acid (LTA) contained in mature supragingival plaque's gram-positive bacteria is thought to contribute to acute infection and inflammatory actions.[11] The end result of these pathogenic processes is the characteristic constellation of clinical gingivitis indicators that follow: loss of gingival stippling; edema; erythema; and bleeding upon probing.[12]

Although periodontitis is always preceded by gingivitis, not all gingivitis progresses to periodontal disease. Research related to causality is ongoing to investigate how plaque-specific virility and individual host response—working individually or collectively—influence which patients will see their gingivitis evolve into a more serious condition. It has been recognized for some time that hormonal surges (e.g., adolescence, pregnancy),[13] certain medications (e.g., anticoagulants),[14] and systemic disease (e.g., uncontrolled diabetes)[15] can promote the symptoms of gingivitis. Behavioral practices such as smoking can also be contributing factors.[16] But in the absence of these well-documented examples, individual genetic factors that may explain the variation of response severity to similar levels and pathogenicity of plaque continue to be explored.

ANTIMICROBIAL REDUCTION OF GINGIVITIS

While it is theoretically possible to remove all supragingival plaque daily with skilled tooth brushing and interdental cleaning, there is abundant evidence that few adults achieve this goal, with many spending insufficient time and effort on oral hygiene.[17-19] Accordingly, expecting all patients to be gingivitis free following professional brushing and flossing instruction is not realistic, as evidenced by the high gingivitis prevalence.[1] Additionally, there appears to be a subset of individuals whose health status and/or unique host factors render them more reactive to lesser amounts of undisturbed plaque than typically cause adversity in others.[20-22] In the same way that the classic set of heart disease factors (smoking, diabetes, dyslipidemia, hypertension) does not explain all heart attacks,[23] plaque *quantity* is not the determining factor in the severity of every case of gingivitis.

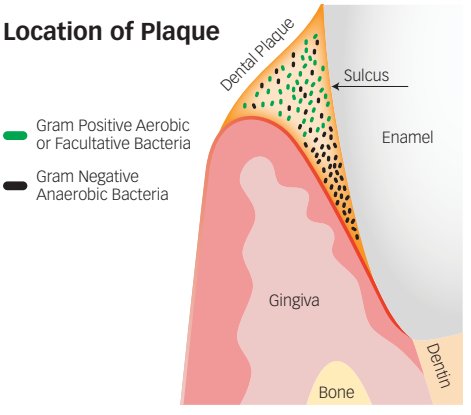


Figure 2. The virulence of plaque is related to factors such as biofilm maturity and location.

Antimicrobial dentifrices and mouth rinses (chemotherapeutics) for use in tandem with mechanical plaque removal were developed with the goal of boosting the results of the typical oral hygiene session by targeting plaque, and thus gingivitis. The concept is straightforward: Because tooth brushing—regardless of its inefficiency or frequency—is an almost universal practice in developed countries, substituting an antimicrobial toothpaste for a regular fluoride toothpaste is a very simple means of helping to prevent and treat gum disease. (Adding a mouth rinse can represent an additional step for patients not previously using one, so equal compliance is uncertain.)

Historically, antimicrobial oral health products have been utilized both to reduce the quantity of plaque and to decrease the virulence of unremoved plaque. Antimicrobial agents may possess one or both of the following attributes: bactericidal (kills microbes) and bacteriostatic (impedes bacterial growth and metabolism). Commonly used antimicrobial agents in oral rinses and/or dentifrices include chlorhexidine, cetylpyridinium chloride, triclosan, and stannous fluoride. The effectiveness of these antimicrobials varies and is dependent upon factors such as their substantivity (retention in the oral cavity), strength, and successful formulation.

Chlorhexidine

Chlorhexidine—available as a 0.12% chlorhexidine gluconate prescription-only mouth rinse—has long been used in the management of gingivitis and periodontitis. Chlorhexidine has broad-spectrum antibacterial properties, with its primary mechanism of action being bactericidal by membrane disruption; secondarily it inhibits glycosidic and proteolytic enzymes produced by bacteria.[24] Chlorhexidine has proven oral substantivity and reduces pellicle formation and inhibits plaque regrowth.[25] Two separate systematic reviews collectively reviewing 24 clinical trials found statistically significant antiplaque and antigingivitis benefits with chlorhexidine use.[26,27] Patient compliance, however, is often compromised by taste and staining potential. Chlorhexidine incorporation into a dentifrice has been limited due to formulation difficulties related to ingredient compatibility, esthetics, and the dosage that would be required for effectiveness.

Cetylpyridinium Chloride

Like chlorhexidine, a mouth rinse is the customary vehicle for cetylpyridinium chloride (CPC). Highly substantive and bioavailable when properly formulated, the broad-spectrum antimicrobial CPC works primarily via lysis of organism cell walls, disruption of cell metabolism, and inhibition of cell growth.[28] Numerous laboratory and ***in vivo*** clinical trials have demonstrated plaque-inhibiting and gingival health benefits of CPC.[29-31] Alcohol-free CPC rinses are widely available since many patients do not like the burning sensation associated with alcohol. Successful formulation of CPC oral products continues to require expertise for assurance of clinical effectiveness, and patients must be amenable to assimilating a mouth rinse step into their routine.

Triclosan

Some dentifrices with gingival health indications contain the broad-spectrum antibacterial phenolic agent triclosan. Triclosan targets both gram-negative and gram-positive bacteria and blocks fatty acid biosynthesis in the cytoplasmic membranes to facilitate cell death.[28,32] A copolymer (polyvinylmethylether/maleic acid—"Gantrez") is generally necessary for inclusion in 0.3% triclosan dentifrices to bolster antiplaque/antigingivitis effectiveness by increasing oral retention.[33] In a large review and meta-analysis of clinical trials assessing a triclosan/Gantrez toothpaste versus control dentifrices, the authors concluded, "This review presents moderate-quality evidence that toothpastes containing triclosan/copolymer, in addition to fluoride, reduce plaque, gingival inflammation and gingival bleeding, when compared with fluoride toothpastes without triclosan/copolymer. These reductions may or may not be clinically important and are evident regardless of initial plaque and gingivitis levels, or whether a baseline oral prophylaxis had taken place."[34] While triclosan has a lesser antimicrobial benefit than chlorhexidine, it offers more favourable esthetics and greater formulation compatibility with other dentifrice ingredients.[33]

Stannous Fluoride

Stannous fluoride (SnF_2) is the only anticaries agent that is also an antimicrobial. As the active ingredient in the first toothpaste to receive the American Dental Association's Seal of Acceptance, it continues to be in wide use today due to its unique ability to address multiple oral health indications, including plaque and gingivitis control.[35,36] Stannous fluoride reduces bacterial growth, bacterial adhesion, and the production of acids and other metabolic toxins that contribute to gingivitis. Furthermore, it has notable substantivity, which promotes long-lasting bacteriostatic and bactericidal properties.[37,38] A body of controlled clinical trials of varying study designs and durations have demonstrated superior antiplaque and antigingivitis efficacy for stannous fluoride dentifrices compared to both placebo and other antimicrobial control dentifrices.[35,36,39-44] Formulation expertise is critical for optimum bioavailability of stannous fluoride, efficacy, and esthetics.[36,39-44] First-generation stannous fluoride dentifrices could cause staining in some, but the current leading marketed multibenefit 0.454% stannous fluoride dentifrices actually are extrinsic whitening pastes that have been fully stabilized to allow stannous fluoride to coexist with cosmetic and other oral health therapeutic ingredients.[45]

STANNOUS FLUORIDE AND INFLAMMATION

Stabilized SnF_2's plaque control properties were already well-known, but dentifrice researchers wanted to explore a disproportionate gingivitis benefit/plaque benefit ratio that had been observed in many clinical trials. For example, Gerlach & Amini reported the results of a three-month study where participants with preexisting gingival bleeding were randomly assigned to twice daily unsupervised tooth brushing with either a 0.454% stabilized stannous fluoride dentifrice or a regular fluoride toothpaste.[46] After three months, the SnF_2 group averaged 74% fewer

bleeding sites relative to baseline (compared to 2% for the placebo group) (Figure 3). Interestingly, however, reduction in plaque quantity in clinical studies of this 0.454% SnF_2 dentifrice averaged 27.2%[47,48] (Figure 4).

What might explain the strikingly larger SnF_2 gingivitis reduction compared to the typical reduction in plaque quantity? To provide an answer, innovative research has led to a fresh understanding of how stannous fluoride goes beyond antibacterial plaque regrowth reduction effects to also modulate plaque toxicity through its impact on the process of gingival inflammation.[49,50]

Figure 3. In 99 adults, SnF_2 dentifrice produced up to 74% significantly less bleeding sites versus baseline or the control (P<0.001). Nearly one half of SnF_2 users completed with one or no bleeding sites.

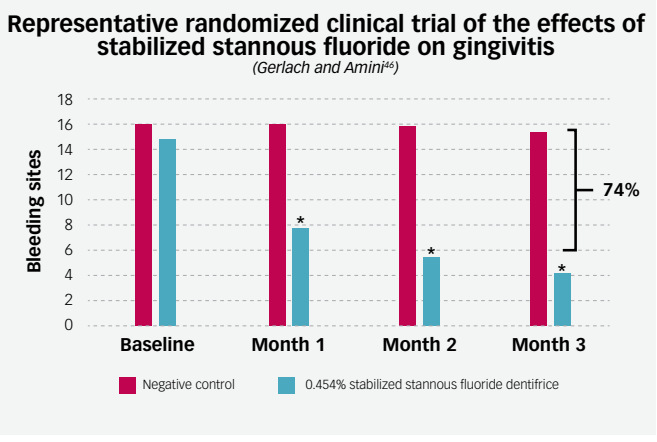
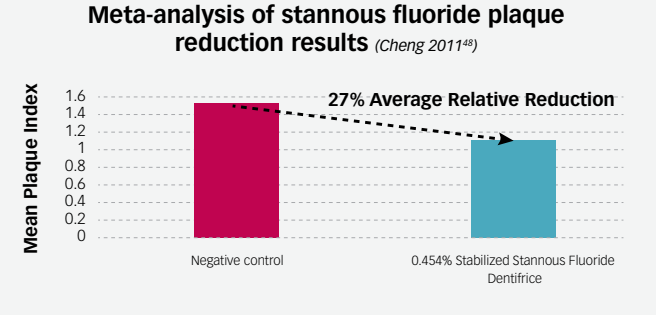


Figure 4. A meta-analysis of three 4-day plaque model clinical crossover trials found that SnF_2 dentifrice provided 27% significantly greater plaque reduction compared to the negative control toothpaste (P<0.0001).



Inflammation is a biological defense mechanism that occurs in response to injury or irritants, such as bacteria. In an attempt to protect against tissue damage and initiate healing, the immune system activates a complex process whereby inflammatory mediators are released to destroy or neutralize the insult, causing the recognizable symptoms of acute inflammation. The early steps of the inflammatory response can be summarized as follows:

Recognition

The body's tissues contain receptors—sentinel cells—that are pattern recognition receptors (PRR). They encounter and identify the patterns of potential invaders that are uniquely different from the host, e.g., pathogen-associated molecular patterns (PAMPs).[51]

Recruitment

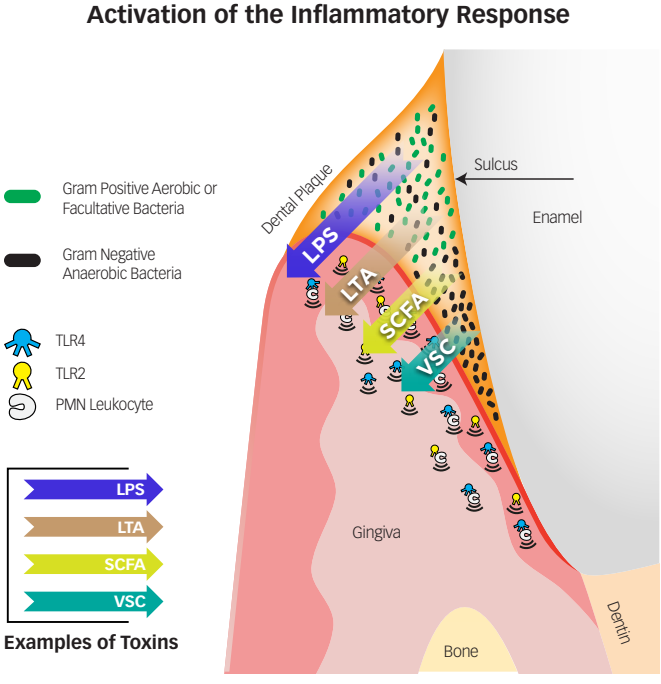
PRRs summon the host's inflammatory mediators (e.g., cytokines), resulting in cellular/vascular permeability changes that promote the inflammatory response at the affected site.[51,52]

Gingival Inflammation

In the gingival tissues, current understanding holds that the tissue components (gingival fibroblasts, epithelia, endothelia, periodontal ligament fibroblasts) contain sentinel PRR cells called toll-like receptors (TLRs) that are scanning for the presence of bacterial pathogens, like those found in plaque biofilm. TLRs are also found in the individual's resident inflammatory cells (e.g., neutrophils, macrophages), where they stand on guard if needed to recruit cells to fight the pathogens and are activated when bacteria begin to invade.[11,47,49,50,53]

More specifically, the PAMPs of the pathogens in the gingival sulcus—the bacterial endotoxins lipopolysaccharide (LPS) and lipoteichoic acid (LTA)—are recognized as harmful by the body's TLRs (TLR4, TLR2), which bind with them and spur the TLRs to activate the inflammatory response. As illustrated in Figure 5, in the recruitment phase, activation

Figure 5. Plaque bacteria in the sulcus produce toxic metabolites that stimulate host receptors (TLRs) to recruit other cells to begin the responsive inflammatory process.



of TLRs stimulates the secretion of cytokine and other effector molecules, leading to the cascade of actions that causes recognizable symptoms of inflammation. Additionally, metabolites are produced by the aerobic and anaerobic pathogens that heighten the response of the TLRs and thus exacerbate the inflammation, inhibit tissue repair, and potentially increase tissue permeability: namely enzymes such as gingipains and collagenase that are synthesized directly by bacteria, and saliva and/or diet-derived short chain fatty acids/carboxylic acids, volatile sulfur-hydrogen sulfide, methylmercaptan, and amines/amides.[11,47,49,50,53]

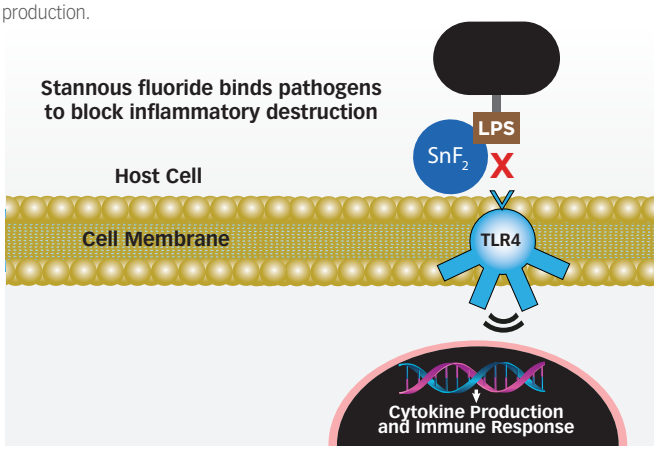
With this understanding of the proinflammatory actions of plaque bacteria, researchers questioned how oral antimicrobials might be modulating the toxicity of plaque. Previous research had assessed the *quantity* of plaque microbes; however, this did not appear to correlate directly to plaque toxicity, given that different pathogens produce different amounts of toxic metabolites, and these also vary in LPS quantity. The binding to LPS/LTA of oral care cationic antimicrobials such as stannous fluoride had not previously been investigated. Therefore, contemporary research using novel methodology was developed to explore this potential additional mechanism of action in reducing plaque virulence. Table 1 summarizes the findings.

Table 1: Key Conclusions of SnF_2 Plaque Toxicity Laboratory Studies[49,50]

STANNOUS FLUORIDE:
• **BINDS** to toxins (e.g., lipopolysaccharides)
• **BLOCKS** endotoxins' reactivity with toll-like receptors
• **BLUNTS** inflammatory process to reduce/prevent gingivitis

The results of these studies collectively revealed an anti-inflammatory mechanism whereby stannous fluoride binds the plaque bacterial endotoxins and diffuses their virulence, effectively shutting down the host inflammation response (Figure 6).

Figure 6. Stannous fluoride has been shown to block the antigen reactivity of plaque pathogens (LPS, LTA) via toll-like receptors to effectively deactivate cytokine production.



Like a sturdy child-safety lock on a cabinet full of hazardous cleaners that blocks the cabinet handles to prevent a child from opening the doors to defend against harm and injury, stannous fluoride serves as the "lock" that renders the "cabinet" (i.e., LPS/LTA and the inflammatory cascade they trigger) less harmful, reducing the potential for damage (i.e., gingivitis, periodontitis) (Figure 7).

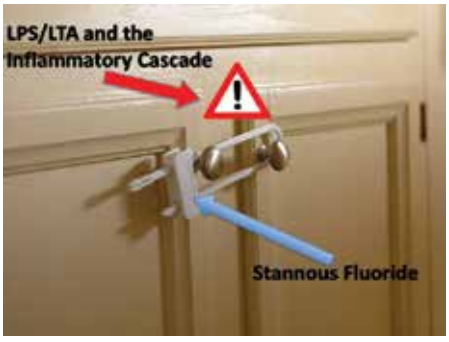


Figure 7. Stannous fluoride works similarly to a "lock" to bind plaque endotoxins and thus prevent the interaction with the host toll-like receptors.

Clinical Research Evidence

Stannous fluoride has an extensive body of research across several decades supporting its gingival health clinical benefits, and previously these were believed to emanate solely from long-lasting bacterial inhibition and bacterial kill properties and acid suppression. New evidence illuminating the mechanisms by which SnF_2 reduces plaque virulence and inflammation was discussed above. Randomized and controlled clinical trials with additional toxicity measurements have confirmed these effects.

Klukowska et al. reported on a four-week trial with high bleeding and low bleeding cohorts of subjects.[54] Both groups brushed twice daily with a stabilized 0.454% stannous fluoride dentifrice. The results showed significant reductions in gingival bleeding (42%-53%) at week four for both the diseased and healthy cohorts. Analysis of subgingival plaque sampling demonstrated that following the use of the SnF_2 dentifrice, there was markedly reduced LPS/LTA dye activity, toll-like receptor activity, and cytokine expression in both groups. Salivary lavage testing of morning wake-up plaque samples additionally demonstrated significant suppression of short chain carboxylic acid toxins in both cohorts.[54,55] Important study findings on how SnF_2 therapy modulated the gingival responses of both the high and low bleeding/gingivitis cohorts are shown in Table 2.

Table 2: Subgingival Plaque in High and Low Gingival Bleeders

4-WEEK TRIAL OF 0.454% SNF_2 DENTIFRICE (KLUKOWSKA 2017)[54]
• Stannous fluoride suppressed toxicity in all sampled subgingival sites in both the high and low bleeding/gingivitis cohorts.
• SnF_2 benefits could be seen in both diseased sites as well as sites not yet showing signs of disease.

These clinical gingival health improvements confirmed the findings from the previous *in vitro*[49,50] and clinical research[56] where SnF_2 significantly decreased plaque toxicity and decreased the inflammatory response. Notably, the SnF_2 benefits of blocking the reactivity of plaque LPS/LTA were observed not only in the diseased sites, but also in the sites termed "healthy," which were not yet displaying the measurable signs of inflammation. This indicates that this stabilized fluoride dentifrice can not only treat disease, but can also prevent the development of disease in patients not exhibiting clinical signs of gingivitis.

Subgingival plaque sampling is not a common measure in OTC dentifrice studies, and its inclusion in these antigingivitis stabilized SnF_2 dentifrice trials allowed for some new insights about SnF_2's impact on the modulation of pathogenicity and inflammation in the sulcus, where disease is known to begin. Subgingival penetration and retention of stabilized stannous fluoride during tooth brushing was confirmed in a recent two-week clinical investigation in subjects with gingivitis.[57] At 30 minutes postbrushing and 12 hours postbrushing, significant levels of tin—a stannous fluoride marker—were detected in gingival crevicular fluid at sampling sites 2 to 4 mm in depth.

PATIENT CARE IMPLICATIONS

Gingival Health Strategies

Dental professionals can develop strategies to assist patients in achieving health beyond the prophylaxis recall visit.

Strategy No. 1: Assess Individual Risk and Causation

Inflamed tissues and gingival bleeding are easily observed during the initial patient visual assessment, periodontal probing, and debridement procedures. A review of the medical history may occasionally uncover contributing factors that require more meticulous plaque control. More often, where undisturbed supragingival plaque is present and/or the patient confirms a lack of thorough home care, it can be assumed that better oral hygiene should result in decreased gingivitis. But if home care is relatively good, it is possible there is heightened sensitivity to plaque toxicity or a greater plaque composition virulence, i.e., the "high responder" patient.[58] Assigning causality and forecasting disease outcomes based on individual predispositions or analyzing plaque toxicity for high responders is not currently possible for the office-based clinician.

Strategy No. 2: Instruct in Mechanical Plaque Removal

An effective mechanical home oral hygiene regimen is always the first line of defense against gingivitis. Patients have a wide range of toothbrush options, including popular electric toothbrushes, which can enhance motivation and brushing effectiveness. Oscillating-rotating power brushes, for example, have some short-term advantages for plaque and gingivitis reduction relative to side-to-side electric brush technologies, according to a Cochrane Group systematic review and meta-analysis.[59] Optimal brushing technique, longer duration, and angled bristle designs have been linked with more plaque removal.[60-62] Even under optimal conditions, however, some plaque is generally left behind after brushing, particularly in more difficult-to-access areas.[18-20,63-65]

Strategy No. 3: Recommend an Effective Adjunctive Chemotherapeutic Product

Dental professionals are in a uniquely advantageous position to assess which oral health products would offer patients the greatest benefits, and to make specific product recommendations to the patient accordingly. In one survey, dental hygienist respondents ranked their top two criteria for making toothpaste recommendations as follows: 1) provided an important patient benefit; and 2) had claims that were substantiated with peer-reviewed published research.[66] Undoubtedly, assisting the patient in choosing among the multitude of OTC offerings in the toothpaste aisle to select a dentifrice best suited for their needs with solid clinical evidence is an invaluable service, as few patients will access or evaluate the supporting research, or lack thereof.

Antimicrobial dentifrices provide patients with existing gingivitis an actionable means of reducing disease. Based on the research among high and low bleeder groups, stabilized stannous fluoride dentifrice gives those with relatively healthy tissues an insurance policy of sorts: with the simple switch to a properly formulated stannous fluoride dentifrice, patients receive antiplaque and antigingivitis benefits every time they brush their teeth. These benefits can even extend until the next brushing, with use of this highly substantive antimicrobial.[38]

Using a seatbelt each time one drives, despite the low likelihood of being involved in an accident and suffering harm, is nearly universally considered a wise, low-effort, forward-thinking means of preserving health and life and reducing expenditures. The utility of preventive and public health interventions is often evaluated by assessing whether the investment in efforts and/or cost are offset by the resulting benefits (e.g., health improvement, less medical spending long term). Applying this paradigm to the regular use of a SnF_2 antimicrobial toothpaste in lieu of a "regular" dentifrice, the documented gingival health benefits established by a myriad of research have been thoroughly documented. The following questions could be considered:

Is it economical? Since virtually everyone currently uses toothpaste, there's likely no appreciable cost increase.

Is it easy to adopt? Patients need only switch toothpastes. The adult usage instructions for an antimicrobial paste are generally similar to those of a regular fluoridated paste. The standard professional consensus is to advise twice daily tooth brushing, striving for two minutes per session.

Are there cosmetic benefits? When correctly formulated, the new generation of stabilized stannous fluoride dentifrices with sodium hexametaphosphate, hydrogen peroxide (2-step process) or zinc citrate has been shown not only to be associated with less stain, including interproximally, but also to remove existing stain (whiter teeth).[36,67,68]

Are there additional advantages? Leading marketed dentifrices have either sodium fluoride, sodium monofluorophosphate, or stannous fluoride as the active fluoride agent; all three have established anticaries efficacy.[69] Stannous fluoride is the only fluoride to also provide multiple clinically proven therapeutic and cosmetic benefits, including protec-

tion against plaque, gingivitis, dentinal hypersensitivity, enamel erosion, and breath malodor.[36,45] Additionally, the bacteriostatic/bactericidal properties of a stannous fluoride dentifrice make it ideal for use with dental implants because stannous fluoride kills bacteria and inhibits biofilm formation, of utmost importance in dental implant maintenance. When combined in an all-in-one, stabilized, highly bioavailable toothpaste formulation with an antitartar agent and stain protection/whitening ingredients, the result is a unique multibenefit dentifrice that both targets existing gingivitis and prevents against new disease.

Given the ease of implementation and lack of disadvantages, dental professionals should consider clinically proven stabilized stannous fluoride dentifrice in treatment planning for patients who have, or are susceptible to, gingivitis.

CONCLUSION

Multiple published studies support the ***in vitro*** and clinical efficacy of stabilized stannous fluoride across broad therapeutic and cosmetic indications. New research has revealed additional multimodal actions for this combination antimicrobial/anticaries fluoride: beyond stannous fluoride's proven bactericidal/bacteriostatic actions, it also modulates the virulence of sulcular plaque by binding with pathogenic endotoxins to effectively block the host inflammatory cascade at its initiation point. As a result, the regular use of a chemotherapeutic, stabilized stannous fluoride dentifrice can benefit both patients with active gingivitis as well as those who may be prone to developing it based on their oral hygiene acumen and/or unique individual host factors.

ACKNOWLEDGEMENTS

Shelly Campbell, RDH, MPH, provided writing assistance and Procter & Gamble provided funding for the development of this CE course.

REFERENCES

1. Albandar JM, Rams TE. Global epidemiology of periodontal diseases. *Periodontol 2000* 2002;29:7-10.
2. Varella-Centelles P, Diz-Iglesias P, Estany-Gestal A, Seonae-Romero JM, Bugarin-Gonzáles R, Seonane J. Periodontitis awareness amongst the general public: A critical systematic review to identify gaps of knowledge. *J Periodontol* 2016;87:403-441.
3. Löe H, Theilade E, Jensen SB. Experimental gingivitis in man. *J Periodontol* 1965;36:177–187.
4. Theilade E, Wright WH, Jensen SB, Löe H. Experimental gingivitis in man II. A longitudinal and bacteriological investigation. *J Periodont Res* 1966;1:1-13.
5. Page RC. The etiology and pathogenesis of periodontitis. *Compend Contin Educ Dent* 2002;23(suppl 5):111-4.
6. Schlätzle M, Faddy MJ, Cullinan MP, Seymour GJ, Lang NP, Bürgin W, Anerud A, Boysen H, Löe H. The clinical course of chronic periodontitis: V. Predictive factors in periodontal disease. *J Clin Periodontol* 2009;36:365-371.
7. Lang NP. Commentary: bacteria play a critical role in the etiology of periodontal disease. *J Periodontol* 2014;85:211–213.
8. Savitt ED, Socransky SS. Distribution of certain subgingival microbial species in selected periodontal conditions. *J Periodontal Res* 1984;19:111-123.
9. Lovegrove JM. Dental plaque revisited: bacteria associated with periodontal disease. *J N Z Soc Periodontol* 2004;87:7-21.
10. Lang NP, Schlätzle M, Löe H. Gingivitis as a risk factor in periodontal disease. *J Clin Periodontol* 2009;36:3-8.
11. Huggins T, Haught JC, Xie S, Tansky CS, Klukowska M, Miner MC, White DJ. Quantitation of endotoxin and lipoteichoic acid virulence using toll receptor report gene. *Am J Dent* 2016;29:321-327.
12. Mariotta A, Hefti AF. Defining periodontal health. *BMC Oral Health* 2015;15 Supple 1:S6.
13. Markou E, Eleana B, Lazaros T, Antonios K. The influence of sex steroid hormones on gingiva of women. *Open Dent J* 2009;3:114-119.
14. Heong FL. Heart medications & oral health. National Dental Centre Singapore. November 2014; Accessed 2/6/18. https://www.ndcs.com.sg/Newsroom/MediaCoverage/Pages/HeartMedicationsOralHealth.aspx
15. Daniel R, Gokulanathan S, Shanmugasundaram N, Lakshmigandhan M, Kavin T. Diabetes and periodontal disease. *Journal of Pharmacy & Bioallied Sciences* 2012;4(Suppl 2):S280-S282.
16. Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. *J Periodontol* 2000;71(5):743–751.
17. McCracken G, Janssen J, Heasman L, Stacey F, Steen N, deJager M, Heasman P. Assessing adherence with toothbrushing instructions using a data logger toothbrush. Br Dent J 2005;198:29-32.
18. Bakdash B. Current patterns of oral hygiene product use and practices. *Periodontol 2000* 1995;8:11–43.
19. Tedesco LA. Behavioral research related to oral hygiene practices: a new century model of oral health promotion. *Periodontol 2000* 1995;8:15-23.
20. Lie MA, Danser MM, van der Weijden GA, Timmerman MF, de Graaff J, van der Velden U. Oral microbiota in participants with a weak or strong response in experimental gingivitis. *J Clin Periodontol* 1995;22:642-647.
21. Tatakis DN, Tromelli L. Modulation of clinical expression of plaque-induced gingivitis. I. Background review and rationale. *J Clin Periodontol* 2004;31:229-238.
22. Trombelli L, Farina R. A review of factors influencing the incidence and severity of plaque-induced gingivitis. *Minerva Stomatol* 2013;62:207-234.
23. Glick M. The Oral-Systemic Health Connection: A Guide to Patient Care. 1st edition. Quintessence Pub Co;2014.
24. Santos A. Evidence-based control of plaque and gingivitis. *J Clin Periodontol* 2003;30(Suppl 5):13-16.
25. McBain AJ, Bartolo RG, Catrenich CE, Charbonneau D, Ledder RG, Gilbert P. Effects of a chlorhexidine gluconate-containing mouthwash on the vitality and antimicrobial susceptibility of in vitro oral bacterial ecosystems. *Appl Environ Microbiol* 2003;69:4770-4776.
26. Gunsolley JC. A meta-analysis of six-month studies of antiplaque and antigingivitis agents. *J Am Dent Assoc* 2006;137:1649–1657.
27. Van Strydonck DA, Slot DE, Van der Welden U, Van der Weijden F. Effect of a chlorhexidine mouthrinse on plaque, gingival inflammation and staining in gingivitis patients: A systematic review. *J Clin Periodontol* 2012;39:1042-1055.
28. Scheie AA. Models of action of currently known chemical antiplaque agents other than chlorhexidine. *J Dent Res* 1989;68:1609-1616.
29. White DJ. An alcohol-free therapeutic mouthrinse with cetylpyridinium chloride (CPC) – the latest advance in preventive care: Crest Pro-Health Rinse. Am J Dent 2005;18, Spec No:3A-8A.
30. García-Godoy F, Klukowska MA, Zhang YH, Anastasia K, Cheng R, Gabbard M, Coggan J, White DJ. Comparative bioavailability and antimicrobial activity of cetylpyridinium chloride mouthrinses in vitro and in vivo. *Am J Dent* 2014;27:185-190.
31. Cheng R. Breath and plaque prevention with cetylpyridinium chloride rinses: clinical meta-analysis. *J Dent Res* 2014;93 (Sp Is A): (Abstract 573).
32. Regös J, Hitz HR. Investigations on the mode of action of triclosan, a broad spectrum antimicrobial agent. *Zentralbl Bakteriol Orig A* 1974;226:390-401.
33. Blinkhorn A, Bartold PM, Cullinan MP, Madden TE, Marshall RI, Raphael SL, Seymour GJ. Is there a role for triclosan/copolymer toothpaste in the management of periodontal disease? *Br Dent J* 2009;207(3):117-125.
34. Riley P, Lamont T. Triclosan/copolymer containing dentifrices for oral health. Cochrane Database Syst Rev. Dec 5;(12):CD010514. Doi: 10.1002/14651858.CD010514.pub2.
35. Sensabaugh C, Sagel ME. Stannous fluoride dentifrice with sodium hexametaphosphate: review of laboratory, clinical and practice-based data. *J Dent Hyg* 2009;83:70-78. Review.
36. Baig A, He T. A novel dentifrice technology for advanced oral health protection: A review of technical and clinical data. *Compend Contin Educ Dent* 2005:26(9 Suppl) 1):4-11.

37. Otten MP, Busscher HJ, Abbas F, van der Mei HC, van Hoogmoed CG. Plaque-left-behind after brushing: intra-oral reservoir for antibacterial toothpaste ingredients. *Clin Oral Investig* 2012;16:1435-1442.
38. Ramji N, Baig A, Lawless MA, Saletta L, Suszcynsky-Meister E, Coggan J. Sustained antibacterial actions of a new stabilized stannous fluoride dentifrice containing sodium hexametaphosphate. *Compend Contin Educ Dent* 2005;26(9 Suppl 1):19-38.
39. Friesen L, Goyal CR, Qaqish JG, He T, Eusebio R, Zsiska M, Farmer T, Schneiderman E. Comparative antiplaque effect of two antimicrobial dentifrices: Laboratory and clinical evaluations. *J Clin Dent* 2017;28(Spec Iss B):B6-11.
40. He T, Eusebio R, Goyal CR, Qaqish JG. Assessment of the effects of a novel stabilized stannous fluoride dentifrice on gingivitis in a two-month positive-controlled clinical study. *J Clin Dent* 2017;28(Spec Iss B):B12-16.
41. Mankodi S, Bartizek RD, Winston JL, Biesbrock AR, McClanahan SF, He T. Anti-gingivitis efficacy of a stabilized 0.454% stannous fluoride/sodium hexametaphosphate dentifrice: a controlled six-month clinical trial. *J Clin Periodontol* 2005;32:75-80.
42. He T, Barker ML, Biesbrock AR, Eynon H, Milleman JL, Milleman KR, Putt MS, Wintergerst AM. Digital plaque imaging evaluation of a stabilized stannous fluoride dentifrice compared with a triclosan/copolymer dentifrice. *Am J Dent* 2013;26:303-306.
43. Sharma N, He T, Barker ML, Biesbrock AR. Plaque control evaluation of a stabilized stannous fluoride dentifrice compared to a triclosan dentifrice in a six-week trial. *J Clin Dent* 2013;24:31-6.
44. Mallatt M, Mankodi S, Bauroth K, Bsoul SA, Bartizek RD, He T. A controlled 6-month clinical trial to study the effects of a stannous fluoride dentifrice on gingivitis. *J Clin Periodontol* 2007;34:762-767.
45. He T, Farrell S. The case for stabilized stannous fluoride dentifrice: An advanced formula designed for patient preference. *J Clin Dent* 2017;28(Spec Iss B):B1-B5.
46. Gerlach RW, Amini P. Randomized controlled trial of 0.454% stannous fluoride dentifrice to treat gingival bleeding. *Compend Contin Educ Dent* 2012:33:134-136,138.
47. White DJ. Advantages of using antimicrobial toothpastes. New understandings regarding their effects on dental plaque virulence in gum disease. Catapult Education. 9/14/2017; Accessed 1/15/18. http://www.catapulteducation.com/courses/anti-microbial-t
48. Cheng R. Meta-analysis of clinical anti-plaque effectiveness for 0.454% stannous fluoride dentifrices. *J Dent Res* 2011;90(Spec Iss A): Abstract 1318.
49. Haught JC, Xie S, Circello B, Tansky CS, Khambe D, Sun Y, Lin Y, Sreekrishna K, Klukowska M, Huggins T, White DJ. Lipopolysaccharide and lipoteichoic acid binding by antimicrobials used in oral care formulations. *Am J Dent* 2016;29:328-332.
50. Haught C, Xie S, Circello B, Tansky CS, Khambe D, Klukowska M, Huggins T, White DJ. Lipopolysaccharide and lipoteichoic acid virulence deactivation by stannous fluoride. *J Clin Dent* 2016;27:84-89.
51. Kolaczkowska E, Kubes P. Neutrophil recruitment and function in health and inflammation. *Nat Rev Immunol* 2013;13:159-175.
52. Kumar V, Cotran RS, Robbin SL. Robbins Basic Pathology. Philadelphia, PA: Saunders; 2003.
53. Hans M, Hans VM. Toll-like receptors and their dual role in periodontitis: a review. *J Oral Sci* 2011;53:263-271.
54. Klukowska M, Haught JC, Xie S, Circello B, Tansky CS, Khambe D, Huggins T, White DJ. Clinical effect of stabilized stannous fluoride dentifrice in reducing plaque microbial virulence I: Microbiological and receptor cell findings. *J Clin Dent* 2017;28:16-26.
55. Cannon M, Khambe, Klukowska M, Ramsey D, Miner M, Huggins T, White DJ. Clinical effects of stabilized stannous fluoride dentifrice in reducing plaque microbial virulence II: Metabonomic changes. *J Clin Dent* 2018; 29:1-12.
56. Klukowska M, Goyal CR, Khambe D, Cannon M, Miner M, Gurich N, Circello B, Huggins T, Barker ML, Furnich C, Conde E, Hoke P, Haught C, Xie S, White DJ. Response of chronic gingivitis to hygiene therapy and experimental gingivitis. Clinical, microbiological and metabonomic changes. *Am J Dent* 2015;28:273-284.
57. Klukowska M, Ramsey D, Combs C, Rattanaudompol U, Haven C, McClenathan D, Ramji N, Milleman J, Milleman K. A clinical trial to assess subgingival penetration and retention of stannous fluoride. Retrieved Feb 6, 2018 from: https://www.dentalcare.com/-/media/dentalcareus/research/pdf/other/gumdetoxifyretentionfinal%20pdf.pdf?la=en&v=1
58. Trombelli L, Tatakis DN, Scapoli C, Bottega S, Orlandini E, Tosi M. Modulation of clinical expression of plaque-induced gingivitis. II. Identification of "high-responder" and "low-responder subjects. *J Clin Periodontol* 2004:31:239-252.
59. Deacon SA, Glenny A-M, Deery C, Robinson PG, Heanue M, Walmsley AD, et al. Different powered toothbrushes for plaque control and gingival health. Cochrane Database of Systematic Reviews 2010, Issue 12. Art. No.: CD004971. DOI: 10.1002/14651858.CD004971.pub2.
60. Creeth J, Zero D, Mau M, Bosma ML, Butler A. The effect of dentifrice quantity and toothbrushing behavior on oral delivery and retention of fluoride in vivo. *Int Dent J* 2013:63(Suppl 2):14-24.
61. Cugini MA, Warren PR. The Oral-B CrossAction manual toothbrush: A 5-year literature review. *J Can Dent Assoc* 2006;72:323a-323k.
62. Slot DE, Wiggelinkhuizen L, Rosema NAM, Van der Weijden GA. The efficacy of manual toothbrushes following a brushing exercise: a systematic review. *Int J Dent Hyg* 2012;10:187-197.
63. Weinstein P, Milgrom P, Melnick S, Beach B, Spadafora A. How effective is oral hygiene instruction? Results after 6 and 24 weeks. *J Public Health Dent* 1989;49:32-38.
64. Stewart JE, Wolfe GR. The retention of newly-acquired brushing and flossing skills. *J Clin Periodontol* 1989;15:331-332.
65. Furuichi Y, Lindhe J, Ramberg P, Volpe AR. Patterns of de novo plaque formation in the human dentition. *J Clin Periodontol* 1992;19:423-33.
66. Hovliaras CA, Panagakos FS. Making product recommendations to patients. Hygienetown Magazine. 211;June 2010. Accessed 1/15/2018. http://www.hygienetown.com/Hygienetown/Article.aspx?aid=2750
67. Friesen L, He T, Eusebio R. Extrinsic stain removal efficacy of a 0.454% stannous fluoride dentifrice. *J Dent Res* 2017;96 (Spec Iss A): Abstract 1941.
68. Terezhalmy GT, Biesbrock AR, Farrell S, Barker ML, Bartizek RD. Tooth whitening through the removal of extrinsic stain with two sodium hexametaphosphate-containing whitening dentifrices. *Am J Dent* 2007;20:309-314.
69. Stookey GH. Are all fluoride dentifrices the same? In Wei SHY (ed.) Clinical Uses of Fluoride. Philadelphia: Lea @ Febiger, 1985, 105-131.

AUTHOR PROFILE

Sherri M. Lukes, RDH, MS, FAADH, has been a dental hygienist for 37 years. She holds advanced degrees in education. She is associate professor emerita, Southern Illinois University, where she taught oral pathology, public health, and multicultural dental hygiene. Research was concentrated in migrant oral health, pathology, and public health issues, resulting in multiple peer-reviewed publications. She is an approved speaker of and holds a pathology fellowship in the American Academy of Dental Hygiene and is a past president of the Illinois Dental Hygienists' Association. Honors include community service, research, and teacher of the year awards while at SIU, IFLOSS Coalition/Illinois Department of Public Health Oral Health Champion Award, and the Sunstar/RDH Award of Distinction.

AUTHOR DISCLOSURE

Sherri M. Lukes, RDH, MS, FAADH *has done consulting work with Procter & Gamble, the sponsors of the unrestricted educational grant for this course. The author of this course receives an honorarium.*

ONLINE COMPLETION

QUICK ACCESS CODE 15318

Use this page to review the questions and answers. Return to **www.DentalAcademyOfCE.com** and sign in. If you have not previously purchased the program select it from the "Online Courses" listing and complete the online purchase. Once purchased the exam will be added to your Archives page where a Take Exam link will be provided. Click on the "Take Exam" link, complete all the program questions and submit your answers. An immediate grade report will be provided and upon receiving a passing grade your "Verification Form" will be provided immediately for viewing and/or printing. Verification Forms can be viewed and/or printed anytime in the future by returning to the site, sign in and return to your Archives Page.

QUESTIONS

1. Most patients are likely to be aware of:
a. color changes in their gingival margins.
b. the signs and symptoms of periodontitis.
c. their individual periodontitis risk profile.
d. bleeding upon expectorating after brushing.

2. Which of the following statements is true?
a. Periodontitis is preceded by gingivitis.
b. Gingivitis always eventually progresses to periodontitis.
c. The quantity of unremoved plaque directly correlates with gingivitis risk.
d. Gingivitis prevalence is decreasing globally and is now uncommon in developed countries.

3. Undisturbed plaque can cause gingivitis because:
a. as a mechanical irritant, it promotes exfoliation of the gingiva.
b. plaque microbes produce inflammatory by-products that initiate tissue destruction.
c. research has shown its presence reduces patient motivation to brush regularly.
d. *Strep mutans* in plaque acid erodes capillaries, causing erythema and bleeding.

4. Which of the following may have an impact upon gingivitis?
a. Tobacco use
b. Pregnancy
c. Systemic conditions
d. All of the above

5. The endotoxins lipopolysaccharide (LPS) and lipoteichoic acid (LTA) are released by:
a. plaque bacteria.
b. cell membranes of the periodontal ligament.
c. salivary enzymes.
d. none of the above.

6. Certain individuals may be more reactive to lesser amounts of undisturbed plaque than others, likely due to:
a. diet.
b. unique host factors.
c. frequency of dental prophylaxes.
d. all of the above.

7. Antimicrobial dentifrices and mouth rinses are referred to as chemotherapeutics because they:
a. fight or kill plaque bacteria that cause disease.
b. meet standards for caries prevention established by the ADA.
c. were initially used in oncology treatment.
d. a and c.

8. Mechanisms of action for an antimicrobial agent may include:
a. inhibition of plaque metabolism.
b. bactericidal activity.
c. bacteriostatic activity.
d. all of the above.

9. Which of the following is not commonly used as an oral chemotherapeutic?
a. Cetylpyridinium chloride
b. Potassium nitrate
c. Triclosan
d. Stannous fluoride

10. Which of the following statement(s) is/are true of chlorhexidine?
a. Patient objections include staining and taste.
b. It has fewer clinically shown gingival health benefits relative to triclosan.
c. Its primary mechanism of action is bactericidal.
d. a and c.

11. Which of the following statements is correct about triclosan?
a. It has been associated with calculus build-up.
b. In dentifrice, it is formulated with a copolymer to increase oral retention.
c. Both a and b.
d. None of the above.

12. Which of the following statement(s) about stannous fluoride is/are correct?
a. It is both an anticaries and an antimicrobial agent.
b. It has well-established substantivity.
c. It was first incorporated into a dentifrice in the early 2000s.
d. a and b.

13. To ensure stannous fluoride's optimum bioavailability and esthetics:
a. dentifrice formulation expertise is critical.
b. a mouth rinse is the preferred vehicle.
c. it must be combined with a copolymer.
d. none of the above.

14. _____ is a biological defense mechanism that occurs in response to injury or irritants, e.g., bacteria.
a. Regeneration
b. Alveolar bone loss
c. Inflammation
d. Decalcification

15. Pattern recognition receptors (PRRs) are considered _____ cells, because they encounter and identify the patterns of potential invaders that are uniquely different from the host.
a. osmotic
b. carrier
c. transport
d. sentinel

16. Which of the following is an example of a PRR in the gingival tissues and resident inflammatory cells, scanning for bacterial pathogens?
a. Pathogen associated molecular patterns (PAMPs)
b. Toll-like receptors (TLRs)
c. Lipopolysaccharides (LPS)
d. All of the above

17. In the gingival sulcus, pathogen PAMPs (endotoxins LPS and LTA) are recognized as harmful by the body's TLRs, which in turn:
a. initiate the fight-or flight reaction.
b. spur the TLRs to activate the inflammatory response.
c. directly attack and eliminate the PAMPs.
d. none of the above.

18. The host inflammatory "cascade of actions" stimulated via TLRs includes the secretion of which of the following?
a. Cytokines
b. *P. gingivalis*
c. PAMPs
d. a and c

19. Characteristic clinical signs of inflammation in the gingival tissues include:
a. edema.
b. erythema.
c. altered gingival margin contours.
d. all of the above.

20. The body of clinical research on the average gingival health benefits of a stabilized stannous fluoride dentifrice has shown:
a. plaque quantity reduction generally closely correlated with the magnitude of gingivitis/bleeding site reduction.
b. gingivitis/bleeding site reduction generally significantly exceeded plaque quantity reduction.
c. no significant reduction in gingivitis/bleeding sites was seen.
d. none of the above.

21. Research shows that stannous fluoride has a newly discovered anti-inflammatory mechanism, whereby it _____ plaque bacteria endotoxins and weakens their virulence, to _____ the host inflammation response.
a. binds/block
b. deactivates/heighten
c. activates/begin signaling
d. none of the above

22. With the use of stannous fluoride, the reactivity of toll-like receptors to PAMPs (e.g. LPS) is:
a. increased.
b. reduced.
c. not affected.
d. none of the above.

23. Using a child-safety device in cabinet preventive analogy, stannous fluoride is similar to which of the following?
a. The LTS/LTA endotoxins inside the cabinet
b. A child's prying hand opening the cabinet
c. The lock that reduces the risk of the cabinet being opened
d. a and b

24. Stannous fluoride can both prevent and reduce gingivitis through which of the following means?
a. Bactericidal actions
b. Plaque acid suppression
c. Anti-inflammatory mechanisms
d. All of the above

25. In a four-week stannous fluoride trial (Klukowska et al.) with high and low bleeding site cohorts:
a. there was a significant reduction in the number of bleeding sites in the high but not the low bleeders cohort.
b. benefits were observed not only in diseased sites, but also in sites not yet displaying measurable signs of disease.
c. a and b.
d. none of the above.

26. New research on stannous fluoride's ability to modulate the pathogenicity of plaque in the gingival sulcus shows a key factor may be its _____ and _____ in the gingival crevicular fluid.
a. supragingival coverage/absorption
b. subgingival penetration/retention
c. subgingival insolvency/impermeability
d. none of the above

27. Plaque bacterial endotoxins PAMPs (e.g., LPS) triggering an inflammatory response are a threat because they:
a. have been found in periodontitis patients' gingiva and root surfaces.
b. have been shown to damage alveolar bone.
c. are conclusively linked with adverse periodontal outcomes.
d. all of the above.

28. If a patient's home care is believed to be good but gingivitis is present (i.e., a "high responder" patient):
a. the patient is likely mispresenting their oral hygiene practices because plaque quantity is always predictive of disease severity.
b. a heightened sensitivity to plaque toxicity or greater plaque virulence may be a factor.
c. an in-office diagnostic test can identify the host factors that are contributing to the gingival response.
d. none of the above.

29. Clinical research shows chemotherapeutic, antimicrobial, stannous fluoride dentifrices can benefit the following patient group(s):
a. Those with widespread bleeding sites
b. Those currently showing few observable signs of gingivitis (susceptible)
c. a and b
d. None of the above

30. Which of the following is a reason to recommend a stannous fluoride antimicrobial dentifrice?
a. It offers other therapeutic benefits.
b. It does not require a significant change in the normal oral hygiene routine.
c. Contemporary formulations offer cosmetic benefits.
d. All of the above.

NOTES

PUBLICATION DATE:	JULY 2018
EXPIRATION DATE:	JUNE 2021

ANSWER SHEET

Pathways to Gingivitis Control with Stabilized Stannous Fluoride: A Novel Discovery

Name: Title: Specialty:

Address: E-mail:

City: State: ZIP: Country:

Telephone: Home ( ) Office ( )

Lic. Renewal Date: AGD Member ID:

Requirements for successful completion of the course and to obtain dental continuing education credits: 1) Read the entire course. 2) Complete all information above. 3) Complete answer sheets in either pen or pencil. 4) Mark only one answer for each question. 5) A score of 70% on this test will earn you 3 CE credits. 6) Complete the Course Evaluation below. 7) Make check payable to PennWell Corp. **For Questions Call 800-633-1681**

EDUCATIONAL OBJECTIVES

1. Identify risk factors associated with gingivitis.
2. Explain the mechanisms of action by which antimicrobials reduce gingivitis.
3. Define the mechanism by which stannous fluoride reduces the inflammatory response.
4. Discuss implications for patient care.

COURSE EVALUATION

1. Were the individual course objectives met?

Objective #1: Yes No Objective #2: Yes No

Objective #3: Yes No Objective #4: Yes No

Please evaluate this course by responding to the following statements, using a scale of Excellent = 5 to Poor = 0.

2. To what extent were the course objectives accomplished overall?	5	4	3	2	1	0
3. Please rate your personal mastery of the course objectives.	5	4	3	2	1	0
4. How would you rate the objectives and educational methods?	5	4	3	2	1	0
5. How do you rate the author's grasp of the topic?	5	4	3	2	1	0
6. Please rate the instructor's effectiveness.	5	4	3	2	1	0
7. Was the overall administration of the course effective?	5	4	3	2	1	0
8. Please rate the usefulness and clinical applicability of this course.	5	4	3	2	1	0
9. Please rate the usefulness of the supplemental webliography.	5	4	3	2	1	0

10. Do you feel that the references were adequate? Yes No

11. Would you participate in a similar program on a different topic? Yes No

12. If any of the continuing education questions were unclear or ambiguous, please list them.

_____

13. Was there any subject matter you found confusing? Please describe.

_____

14. How long did it take you to complete this course?

_____

15. What additional continuing dental education topics would you like to see?

_____

If not taking online, mail completed answer sheet to

PennWell Corp.
Attn: Dental Division,
1421 S. Sheridan Rd., Tulsa, OK, 74112
or fax to: 918-212-9037

For IMMEDIATE results,
go to www.DentalAcademyOfCE.com to take tests online.

QUICK ACCESS CODE 15318

Answer sheets can be faxed with credit card payment to 918-212-9037.

☐ Payment of $59.00 is enclosed.
(Checks and credit cards are accepted.)

If paying by credit card, please complete the following: ☐MC ☐Visa ☐AmEx ☐Discover

Acct. Number: _____

Exp. Date: _____

Charges on your statement will show up as PennWell

1.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	16.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
2.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	17.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
3.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	21.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	22.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	23.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	24.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	25.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	26.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	27.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	28.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	29.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	30.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

AGD Code 016

PLEASE PHOTOCOPY ANSWER SHEET FOR ADDITIONAL PARTICIPANTS.

COURSE EVALUATION and PARTICIPANT FEEDBACK
We encourage participant feedback pertaining to all courses. Please be sure to complete the survey included with the course. Please e-mail all questions to: hhodges@pennwell.com.

INSTRUCTIONS
All questions should have only one answer. Grading of this examination is done manually. Participants will receive confirmation of passing by receipt of a verification form. Verification of Participation forms will be mailed within two weeks after taking an examination.

COURSE CREDITS/COST
All participants scoring at least 70% on the examination will receive a verification form verifying 3 CE credits. The formal continuing education program of this sponsor is accepted by the AGD for Fellowship/Mastership credit. Please contact PennWell for current term of acceptance. Participants are urged to contact their state dental boards for continuing education requirements. PennWell is a California Provider. The California Provider number is 4527. The cost for courses ranges from $20.00 to $110.00.

PROVIDER INFORMATION
PennWell is an ADA CERP Recognized Provider. ADA CERP is a service of the American Dental association to assist dental professionals in identifying quality providers of continuing dental education. ADA CERP does not approve or endorse individual courses or instructors, nor does it imply acceptance of credit hours by boards of dentistry.

Concerns or complaints about a CE Provider may be directed to the provider or to ADA CERP at www.ada.org/cotocerp/

The PennWell Corporation is designated as an Approved PACE Program Provider by the Academy of General Dentistry. The formal continuing dental education programs of this program provider are accepted by the AGD for Fellowship, Mastership and membership maintenance credit. Approval does not imply acceptance by a state or provincial board of dentistry or AGD endorsement. The current term of approval extends from (11/1/2015) to (10/31/2019) Provider ID# 320452

RECORD KEEPING
PennWell maintains records of your successful completion of any exam for a minimum of six years. Please contact our offices for a copy of your continuing education credits report. This report, which will list all credits earned to date, will be generated and mailed to you within five business days of receipt.

Completing a single continuing education course does not provide enough information to give the participant the feeling that s/he is an expert in the field related to the course topic. It is a combination of many educational courses and clinical experience that allows the participant to develop skills and expertise.

CANCELLATION/REFUND POLICY
Any participant who is not 100% satisfied with this course can request a full refund by contacting PennWell in writing.

IMAGE AUTHENTICITY
The images provided and included in this course have not been altered.