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

A Peer-Reviewed Publication

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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.

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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, lipopolysac-

charides (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.

Location of Plaque

Dental Plaque
Sulcus
Enamel

— Gram Positive Aerobic or Facultative Bacteria

— Gram Negative Anaerobic Bacteria
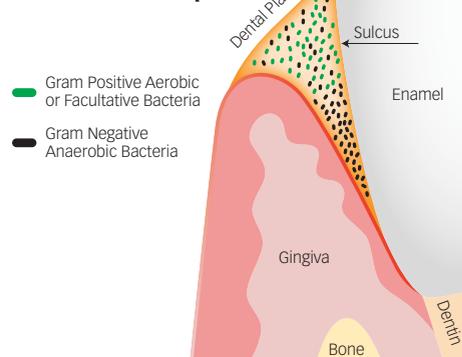
Gingiva

Dentin

Bone

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.

Representative randomized clinical trial of the effects of stabilized stannous fluoride on gingivitis
(Gerlach and Amini[46])
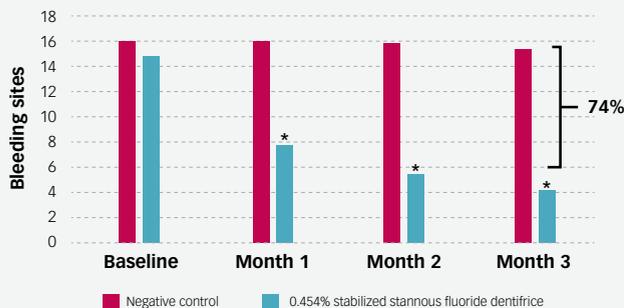
Figure 4. A meta-analysis of three 4-day plaque model clinical crossover trials found that SnF$_2$ dentifrice provided 27% significantly greater plaque reduction compared to the negative control toothpaste (P<0.0001).

Meta-analysis of stannous fluoride plaque reduction results *(Cheng 2011[48])*
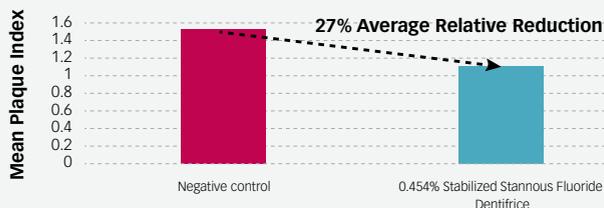
Inflammation is a biological defense mechanism that occurs in response to injury or irritants, such as bacteria. In an attempt to protect against tissue damage and initiate healing, the immune system activates a complex process whereby inflammatory mediators are released to destroy or neutralize the 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.

Activation of the Inflammatory Response
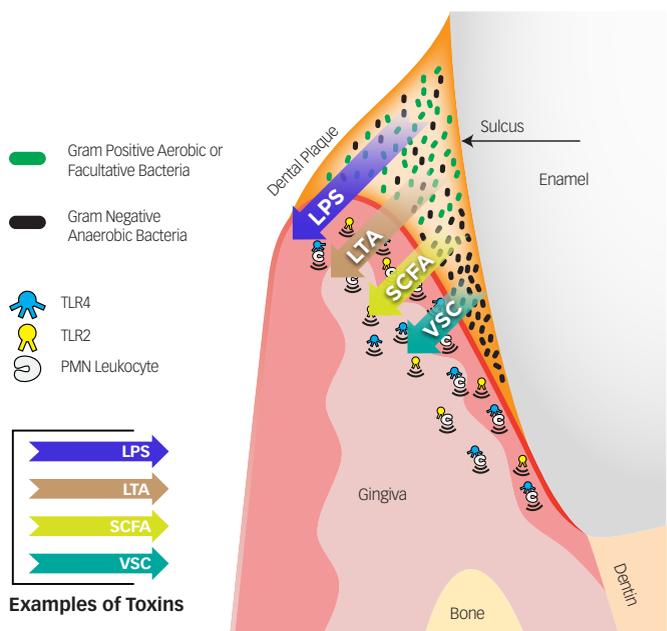
of TLRs stimulates the secretion of cytokine and other effector molecules, leading to the cascade of actions that causes recognizable symptoms of inflammation. Additionally, metabolites are produced by the aerobic and anaerobic pathogens that heighten the response of the TLRs and thus exacerbate the 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.
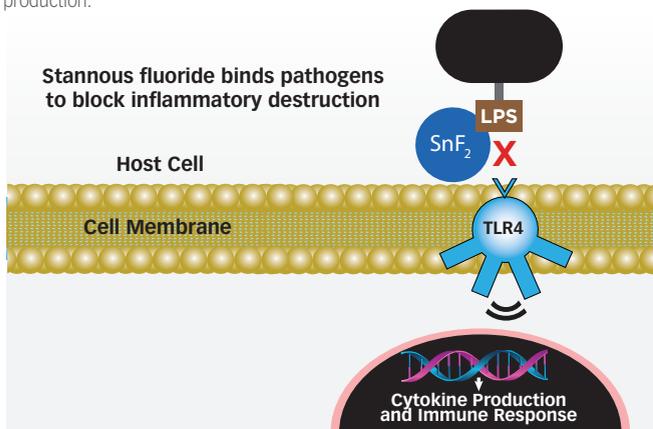
Like a sturdy child-safety lock on a cabinet full of hazardous cleaners that blocks the cabinet handles to prevent a child from opening the doors to defend against harm and injury, stannous fluoride serves as the "lock" that renders the "cabinet" (i.e., LPS/LTA and the inflammatory cascade they trigger) 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

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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

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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

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

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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

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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

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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.

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