American Dental Hygienists' Association

Journal of Dental Hygiene

The American Dental Hygienists' Association

February 2013 Volume 87 Number 1

Journal of Dental Hygiene

VOLUME 87 • NUMBER 1 • FEBRUARY 2013

STATEMENT OF PURPOSE

The *Journal of Dental Hygiene* is the refereed, scientific publication of the American Dental Hygienists' Association. It promotes the publication of original research related to the profession, the education, and the practice of dental hygiene. The journal supports the development and dissemination of a dental hygiene body of knowledge through scientific inquiry in basic, applied, and clinical research.

SUBSCRIPTIONS

The *Journal of Dental Hygiene* is published quarterly online by the American Dental Hygienists' Association, 444 N. Michigan Avenue, Chicago, IL 60611. Subscription rates for nonmembers are one year, $45.

SUBMISSIONS

Please submit manuscripts for possible publication in the *Journal of Dental Hygiene* to JoshS@adha.net.

Inside

Journal of Dental Hygiene
Vol. 87 • No. 1 • February 2013

Features

Editorial

Rebecca S. Wilder, RDH, BS, MS

Looking Towards Celebrating the 100th and Beyond!

Recently I spent time at ADHA central office with our staff editor, Josh Snyder, thumbing through issues of the *Journal of Dental Hygiene*. The reason is that we are preparing for a special edition of the *Journal* to celebrate the 100th anniversary of our wonderful profession that will be distributed at the ADHA CLL/Annual Session in Boston this June. It was an educational experience for me because I was unaware of much of our rich history. In the coming months, Access Magazine and the *Journal* will highlight our history and show how far we have come in many areas and also what is yet to be achieved.

As I read many of the articles and editorials that have been published, reviewed the meetings and minutes of constituent and national meetings, I was so impressed by all of the hard work and dedication that has gone into making our profession what it is today. One of the pictures that was displayed in one of the journals from the 70s/80s contained a picture of one of my dental hygiene mentors, Mrs. Margaret Cain, who held a leadership position in the ADHA. It brought back so many memories of the mentoring she provided to me. Not only was she a stickler for perfection in the clinical setting, she was the epitome of a dedicated professional. She was soft spoken and kind, she held the highest of standards and expected each student to be a professional at all times. She is most likely the reason that I pursued a higher degree in dental hygiene.

There were many other dental hygienists displayed in those journals who dedicated a good part of their lives to the association and the *Journal of Dental Hygiene*. They were goal oriented, intelligent, assertive women who were not afraid to advance the profession. Was it always easy… absolutely not! But they persevered and we all need to thank them!

We have seen many changes over the course of 100 years, from the way dental hygienists dress, to the equipment we use, to the research evidence we have available to us to make the best decisions about care for our patients, to the products available to us to promote the best oral health care. As we move towards the next 100 years, one thing will be certain – change will occur in our profession. More Americans will receive oral health care than ever before. Dental hygienists will have a prominent role in the delivery of oral care and to the overall care that patients receive. We will work more closely with our medical, nursing and allied health colleagues to assess, diagnose and refer patients to the appropriate providers. The science base in dental hygiene will expand as we further build our unique body of knowledge. And we will be valued as an essential part of the health care team for patients.

What role will you have in the dental hygiene profession as we work towards the next 100 years? Let me provide some ideas for you: set short and long term goals for your professional growth, adopt a mentor, be a mentor, run for a component or constituent office, take public speaking classes so you are more confident in front of an audience, obtain a higher degree, collaborate and educate your medical colleagues about the importance of oral health. Do something to make your profession all it can be! Call on the ADHA to help you!

See you in Boston!

Sincerely,

Rebecca Wilder, RDH, BS, MS
Editor–in–Chief, Journal of Dental Hygiene

Linking Research to Clinical Practice

Probiotics and Oral Health

Denise M. Bowen, RDH, MS

The purpose of Linking Research to Clinical Practice is to present evidence based information to clinical dental hygienists so that they can make informed decisions regarding patient treatment and recommendations. Each issue will feature a different topic area of importance to clinical dental hygienists with A BOTTOM LINE to translate the research findings into clinical application.

Twetman S, Keller MK. Probiotics for caries prevention and control. *Adv Dent Res*. **2012;28(2):98-102.**

Objective: Modulation of the microbiota for restoring and maintaining health is a growing issue in medical science. A search for relevant clinical trials on the use of probiotic bacteria as a potential and clinically applicable anti-caries measure was performed.

Methods: According to predetermined criteria, papers were selected and key data on study design, sample size, intervention, duration and results were extracted.

Results: Two animal and 19 human studies were retrieved. Most studies were short-term and restricted to microbiological endpoints, and only 3 human studies reported a caries endpoint. A high degree of heterogeneity among the included investigations hampered the analysis. Significant reductions of mutans streptococci in saliva or plaque following daily intake of probiotic lactobacilli or bifidobacteria were reported in 12 out of 19 papers, whereas 3 reported an increase of lactobacilli. Three caries trials in preschool children and the elderly demonstrated prevented fractions between 21 and 75% following regular intakes of milk supplemented with *L. rhamnosus*. No adverse effects or potential risks were reported.

Conclusions: The currently available literature does not exclude the possibility that probiotic bacteria can interfere with the oral biofilm, but any clinical recommendation would be premature. Large-scale clinical studies with orally derived specific anti-caries candidates are still lacking.

Commentary

An increased interest in use of probiotics to foster oral health has been fueled by the marketing of new products, consumer interest in possible preventive and health maintenance benefits, and research to investigate accuracy of claims and effectiveness in oral health care. Concern about the development of resistant strains to antibiotics is also a factor leading to the emergence of new approaches to combating bacterial infections. A joint statement by the United Nations Food and Agricultural Organization and the World Health Organization included the most recent definition of probiotics as, "Live microorganisms which when administered in adequate amounts confer a health benefit on the host."[1] Unfortunately, the term has been used loosely, not always applied to live bacteria in adequate doses to benefit health or to those bacterial species and genera that have been shown scientifically to confer a health benefit. Studies to validate contents and effectiveness of various probiotics are needed. Because dental caries and periodontal disease are caused by different bacteria, different probiotics might be needed to combat those oral diseases.

Regulation of probiotics by the Food and Drug Administration differs from that of antimicrobials, dentifrices or mouthrinses that make therapeutic claims (e.g. anti-gingivitis or anti-caries). The regulation of claims made for probiotics depends upon their intended use and categorization, such as a dietary supplement, so the burden of proof falls with the FDA after marketing of the product. In the case of other therapeutic oral health products, the burden of proof is the responsibility of the sponsor of the product, so research results

are needed to substantiate claims before marketing. This difference requires the oral health professional's diligence in considering the evidence related to the claims made regarding the effectiveness of probiotics in prevention and treatment of oral diseases or maintenance of oral health.

Studies of clinical oral health benefits of probiotics are uncommon. This study focused on the use of probiotic bacteria as an anti-caries measure. It is a review of the literature related specifically to that purpose; therefore, the authors established criteria to guide what published research articles would be included. They decided to include both in vitro (laboratory) and in vivo (live humans) studies with any caries-related outcome measure, such as reductions in mutans streptococci or lactobacilli, known etiologic species in caries, in saliva or oral biofilm or reduced clinical caries in subjects over time. This literature review cannot be classified as a systematic review, the highest level of evidence, because it did not formally evaluate quality of studies included or limit its inclusion criteria to the highest quality of research – randomized clinical trials (RCTs). For that reason, there was a great deal of variability in research findings included in this review. Two animal and 19 human studies were found, most of which were short-term and restricted to microbiological endpoints. Only 3 of the 19 human studies reported a caries endpoint. A reduction in microbes associated with caries cannot be assumed to result in reduced clinical caries unless caries are evaluated clinically over time. Caries clinical trials generally are at least 3 years in length due to the time it takes to develop new carious lesions that can be measured clinically. These longitudinal RCTs are expensive to conduct, so related outcome measures are studied first to test whether there is promise for a particular intervention such as probiotics.

Results showed statistically significant reductions of mutans streptococci (MS) in saliva or plaque at the end of probiotic use reported in 12 of the 19 human studies, of which only 1 was conducted longer than 3 weeks. Probiotics tested included *L. rhamnosus GG*, Lactobacilli mix, bifidobacteria, *L. reuteri* (2 strains) and *L. rhamnosus* LB21, and combinations thereof. Regrowth of caries etiologic bacteria after probiotic usage was not measured in any of these studies. Interestingly, the authors note, lactobacilli only was reported to have increased in 3 studies after daily intake of the probiotic, lactobacilli. The mode of delivery, for example various dairy products versus tablets or lozenges, did not seem to impact the findings. In addition to being short term, most of the studies also had small sample sizes and did not control and/or define dosages used. Dosages of specific strains of probiotics needed to have beneficial health effects are critical for effectiveness. These weaknesses in study design make definitive conclusions impossible.

If one focuses on the 3 RCTs that were identified in this literature review, findings and clinical endpoints are similar. All investigated strains of *L. rhamnsous* (GG or LB21) that were delivered in milk. One study showed no statistically significant reduction in caries in preschool children after 7 months. The other study of early childhood caries (ECC) showed a significant reduction in ECC after use of milk supplemented with *L. rhamnsous* and 2.5 ppm fluoride, but the effects of the 2 interventions could not be separated, so it is unknown whether the probiotic, fluoride or both affected the outcome of reduced caries. The third study evaluated the effects of probiotics and fluoride on root caries in 4 groups of elderly adults. Findings indicated root caries reversal in all groups compared to the control group with the greatest effect in the probiotic/fluoride group. Perhaps the probiotic bacteria can be considered as an adjunct to fluoride in prevention and control of the caries process, although further study is needed before such a claim can be accurately made with dental hygiene clients who inquire about using probiotics to prevent dental caries. None of the studies reported significant side effects of the probiotics studied. As the authors indicated, there is nothing in the literature to negate the possibility that probiotic bacteria can interfere with the oral biofilm, but any clinical recommendation would be premature. Large-scale RCTs with specific candidates for anti-caries probiotics are lacking.

van Essche M, Loozen G, Godts C, et al. Bacterial antagonism against periodontopathogens. J Periodontol. 2012. [Epub ahead of print].

Background: The aim of the current study was to compare the prevalence of commensal bacteria, with beneficial properties, for healthy and diseased individuals, and additionally to examine the inhibitory effect of some commercial dietary probiotics on periodontopathogens comparing this inhibitory effect with that of orally derived beneficial bacteria.

Methods: Subgingival plaque samples from 35 patients (healthy and periodontitis patients) were analyzed. Growth inhibition of the periodontal pathogens *Porphyromonas gingivalis*, *Prevotella intermedia*, *Fusobacterium nucleatum* and *Aggre-*

gatibacter actinomycetemcomitans was examined using the agar overlay technique and agar well diffusion method. The quantification of the inhibitory effect was checked with the agar well diffusion method.

Results: Using the agar overlay technique the prevalence of strains antagonistic towards *P. gingivalis*, *A. actinomycetemcomitans* and *F. nucleatum* was found to be higher in healthy individuals than in individuals suffering from periodontitis. This could not be validated by the agar well diffusion assay. Compared with the antagonistic activity of the isolated strains, the probiotic strains overall showed a stronger inhibition of the periodontal pathogens.

Conclusion: It was shown that some oral bacteria can cause antagonism towards periodontopathogens and these observations underline the therapeutic potential of applications that stimulate oral health by the application of beneficial effector strains.

Commentary

In periodontal diseases, there is an increase in the quantity of plaque and a shift in bacterial composition towards requisite anaerobic and proteolytic bacteria, many of which are Gram–negative. The host damage that occurs during the disease process is caused by the combined activities of subgingival biofilms and the host responses to these diverse bacterial inhabitants. Limited knowledge is available regarding the effect of probiotics on biofilm-related periodontitis. The oral microbiota is complex and dental biofilms are considered to be difficult therapeutic targets. The current view on the etiology of plaque-related periodontal inflammation considers 3 factors that determine whether disease will develop: a susceptible host, the presence of pathogenic species and the reduction or absence of supposed beneficial bacteria.[2] The complexity of the etiology, initiation and progression of inflammatory periodontal disease lies at the root of the failure of many previous approaches to eradicate or definitively control the disease, such as local and systemic antimicrobial therapies. Oral probiotics represent a current approach to combat periodontal pathogens by introducing "so called" beneficial bacteria that may have the ability to prevent colonization of pathogenic bacteria in the oral biofilm.

The purpose of this study was two–fold. The first aim was to compare the prevalence of commensal bacteria, with beneficial properties, for healthy and diseased individuals. Commensal bacteria have a symbiotic relationship in which one species is benefited while the other is unaffected. These researchers wanted to know how many of these bacteria with beneficial properties were present in individuals with periodontal health in comparison to those with periodontal disease. Previous research has shown that periodontally healthy sites have greater numbers of endogenous beneficial species than diseased sites. The second aim was to examine the inhibitory effect of selected commercial dietary probiotics on periodontopathogens by comparing it with that of orally–derived beneficial bacteria. In other words, the goal was to evaluate if dietary probiotics available on the market inhibited or hindered periodontopathogens, pathogenic bacteria identified as capable of producing periodontal disease, and compare those products to beneficial bacteria derived from the oral cavity. Commensal bacteria have been shown to have a beneficial effect on the host response and the growth and colonization of periodontal pathogens in plaque biofilm.

The most common probiotic strains belong to the genera Lactobacillus and Bifidobacterium; however, probiotic strains have been isolated from several species within each of these genera. The lactobacillus species from which probiotic strains have been isolated include *L. acidophilus*, *L. johnsonii*, *L. casei*, *L. rhamnosus*, *L. gasseri* and *L. reuteri*. Similarly, the bifidobacterium strains include *B. bifidum*, *B. longum* and *B. infantis*.[2] Dietary lactobacillus strains are most commonly found in milk products such as yogurt, fermented milk (e.g. kefur, buttermilk, acidophilus milk) or cheese with active cultures. Bifidobacterium is also found in fermented milk products, as well as fermented teas, such as kombucha, and cultured vegetables like sauerkraut. The lactobacillus strains tested in this study included *L. fermentum* 8900 LMG, *L. casei* Shirota YACULT, *L. casei* Actemel, *L. casei* ACTT-393, *L. paracasei* L 07-21, *L. rhamnosus* Hansen 1968 and *L. rhamnosus* GG.

Subgingival plaque samples were taken from 35 patients (healthy and periodontitis patients) and analyzed for growth inhibition of periodontal pathogens (i.e. *Porphyromonas gingivalis*, *Prevotella intermedia*, *Fusobacterium nucleatum* and *Aggregatibacter actinomycetemcomitans*). Each sample was examined using the agar overlay technique which allows for allows for production of homogeneous bacteria within a thin layer of agar across the surface of an agar plate and the agar well diffusion method to determine the sensitivity of the microbes to the probiotic. The extent of the inhibitory effect also was checked with the agar well diffusion method. Results of the agar over-

lay test showed that the prevalence of isolated strains antagonistic to the periodontal pathogens was greater in samples from healthy individuals; however, this effect could not be verified through the agar well diffusion method. The inhibitory effect of the probiotic strains was greater than the antagonistic effect of the isolated strains indicating that "beneficial" oral bacteria can cause antagonism towards periodontopathogens.

The authors explained that, theoretically, restoring reduced numbers of beneficial bacteria via probiotics might be of interest in the treatment of plaque-related periodontal diseases. Probiotics might not only suppress the emergence of endogenous pathogens (within the host) or prevent the superinfection with exogenous pathogens (from external sources), they might also protect us through the promotion of a beneficial host response. Some oral bacteria act as antagonists to periodontopathogens and inhibit their growth. Probiotics can, easily and with little side effects, reduce the level of indigenous oral microbes, thus they can provide more sites for colonization by probiotic bacteria. This mechanism of action is similar to gastrointestinal and urogenital applications, and these similarities represent an interesting advance in knowledge related to oral healthcare.[2]

Although these findings contribute toward an understanding of the potential inhibitory effect of probiotics, the role of beneficial bacteria in preventing the emergence of pathogenic species and oral health remains unknown. There is a need for additional research to clarify the role of the oral beneficial microbiota, to identify beneficial bacteria and to provide a foundation for large-scale studies on the usefulness of probiotics to maintain or improve oral health. In the meantime, it is premature to inform our patients that probiotics can prevent or cure periodontal disease.

The Bottom Line

There has been a rapid increase of studies published in the literature about probiotics and oral health within the past decade. Clinicians and consumers are encouraged to continue to read new research findings to determine the exact species, dosages and delivery mechanisms that are effective in prevention and control of oral diseases such as dental caries and periodontal disease.

Each of these studies examined the effect of probiotics on oral health, specifically dental caries and periodontal disease. Probiotics have the potential to offer a new mechanism for prevention of these oral diseases by boosting the beneficial oral immune response and by interfering with the growth and colonization of pathogens. Results add to the body of knowledge about probiotics in the prevention and treatment of these oral diseases; however, they do not provide evidence of the effectiveness of probiotics in combating dental caries or periodontal disease.

Based on the findings of these 2 studies, the following conclusions can be drawn:

- Probiotics have been shown to have a positive effect on the oral immune response and inhibition of pathogens associated with periodontal disease.
- Because probiotics seem to affect the colonization of periodontal pathogens, it is logical to assume their potential lies in the regrowth of plaque following its removal by self-care or professional therapy rather than with decreasing the effects of established periodontopathogens in oral biofilm.
- Future large-scale clinical studies are needed to make clinical recommendations for probiotics as anti-caries agents. Probiotic bacteria might be considered as an adjunct to fluoride in prevention and control of the caries process, although further study is needed before such a claim can be accurately made with dental hygiene clients who inquire about using probiotics to prevent dental caries. Certainly, the use of probiotics in lieu of fluoride therapy should be discouraged.
- Some oral probiotics on the market might make exaggerated claims, and these claims are not monitored by the Federal Trade Commission for probiotics as they are for other dental therapeutic products like dentifrices and mouthrinses containing fluoride or antimicrobials. As a result, dental hygienists need to read research related to the benefits of probiotics in relation to oral health care.
- Probiotics are safe for use by our patients when used as instructed as these studies and others have shown no significant side effects.

Summary

Dental hygienists are preventive professionals responsible for advising their patients and the public about the effects of oral care products and natural interventions. The recent increase in consumer and professional interest in the potential effects of probiotics on oral and systemic health further emphasizes the relationship between oral and systemic health, especially as related to the host immune response and growth of pathogens

in the oral biofilm. Probiotics may reduce the colonization of oral bacteria, similarly to their effect in the gastrointestinal tract, but such an effect would most likely have an impact for regrowth of bacteria after self-care, dental hygiene care, nonsurgical or surgical periodontal therapy rather than with biofilm that is firmly established. Clinical recommendations for probiotics as anti-caries or as periodontal disease therapeutic agents are premature.

Denise M. Bowen, RDH, MS, is Professor Emerita in Dental Hygiene at Idaho State University. She has served as a consultant to dental industry, as well as numerous government, university and private organizations and presently is a member of the National Advisory Panel for the National Center for Dental Hygiene Research in the U.S. She has served as Chair of the American Dental Hygienists' Association Council on Research and Chair of the Research Committee for the Institute for Oral Health and has received national awards for excellence in dental hygiene.

References

1. Food and Agriculture Oranization, World Health Organization. Health and Nutritional Properties of Probiotics in Food Including Powder Milk with Live Lactic Acid Bacteria. World Health Organization [Internet]. 2001 October. Available from: http://www.who.int/foodsafety/publications/fs_management/en/probiotics.pdf

2. Teughels W, Van Essche M, Sliepen I, Quirynen M. Probiotics and oral healthcare. *Periodontol 2000*. 2008;48:111–147.

Antiseptic Mouth Rinses: An Update on Comparative Effectiveness, Risks and Recommendations

Diane Osso, RDH, MS; Nehal Kanani, RDH, BS

Abstract

Purpose: Antiseptic mouth rinses are widely recommended and marketed to improve oral health. This article summarizes current studies on the comparative effectiveness of selected antiseptic mouth rinses in controlling plaque and gingivitis, as well as risks associated with daily exposure, including salivary flow rate, oral cancer and wear of composite restorations.

Methods: Electronic database searches were conducted using Google Scholar and PubMed to identify articles comparing the effectiveness of 4 commercially marketed antiseptic mouth rinses differing in active ingredients (0.12% chlorhexidine gluconate, essential oils (menthol, thymol and eucalyptol) and methyl salicylate, 0.7% cetylpyridinium chloride and 20% aloe vera gel) for controlling plaque and gingivitis. Criteria for inclusion included controlled clinical trials and systematic reviews appearing in English language publications evaluating the comparative effectiveness of the mouth rinses in controlling plaque and gingivitis, as well as risks associated with daily usage.

Results: The majority of studies have shown mouth rinses containing chlorhexidine gluconate or essential oils and methyl salicylate provide clinically significant anti–gingivitis and anti–plaque benefits. Cetylpyridinium chloride has been found to provide only limited clinical benefits compared to inactive control mouth rinse. Inadequate evidence is available to evaluate the clinical effectiveness of aloe vera gel. Chlorhexidine, essential oils and cetylpyridinium have been found to be safe. However, limited data are available on the effects of the mouth rinse on wear patterns of dental restorations. Studies reviewed reported no significant difference in salivary flow rate related to alcohol based mouth rinse.

Conclusion: Research supports the effectiveness of antiseptic mouth rinses in reducing plaque and gingivitis as an adjunct to home care. Insufficient evidence is available to support the claim that oral antiseptics can reduce the risk of developing periodontitis or the rate of progression of periodontitis.

Keywords: Mouth rinse, anti–plaque, anti–gingivitis, xerostomia, oral cancer, composite restorations, essential oils, chlorhexidine gluconate, cetylpyridinium chloride

This study supports the NDHRA priority area, **Health Promotion/Disease Prevention:** Investigate the effectiveness of oral self–care behaviors that prevent or reduce oral diseases among all age, social and cultural groups.

Introduction

Dental plaque is the primary etiology for chronic gingivitis, which typically develops within 10 to 21 days in the absence of plaque control. Approximately 50% of the population over the age of 30 has some form of gingivitis.[1] Although mechanical plaque control can be an effective strategy for preventing the progression of periodontal diseases, most individuals do not adequately brush their teeth, and only 11 to 51% of the population admits to using dental floss or some type of inter–dental cleaning device on a daily basis.[1] The daily use of an effective antiseptic mouth rinse is generally considered a simple strategy most patients can easily incorporate into their home care routine.

A relatively high degree of motivation, manual dexterity and compliance in oral hygiene regime are required to achieve the level of oral hygiene necessary to control bacterial plaque formation. The hard tissues of the teeth are not the only surfaces that plaque will colonize. The oral mucosa and the specialized mucosa of the tongue constitute about 80% of the remaining oral surfaces colonized by plaque biofilm.[1] These surfaces serve as reservoirs for pathogenic bacteria, which can re–colonize on the teeth.

Using an antiseptic mouth rinse to supplement mechanical plaque removal can produce an antimicrobial effect throughout the mouth.[2] Chemical agents in a mouth rinse should be effective at modifying the microbiota by selectively eliminating pathogens without negatively

impacting the normal flora that may result in an overgrowth of pathogenic organisms.[3] Evidence shows that the long–term twice daily use of 0.12% chlorhexidine gluconate (Peridex®; 3 M ESPE, Minneapolis, Minn) and essential oils and methyl salicylate (Listerine®; McNeil–PPC, Inc, Skillman, NJ), both anti–plaque and anti–gingivitis mouth rinses approved by the Council on Dental Therapeutics of the American Dental Association (ADA), do not have a negative effect on the oral microbial flora.[4]

Since 1931 the ADA has been placing its "Seal of Acceptance" on oral homecare dentifrice and mouth rinse. To earn its seal, the ADA requires 2 positive clinical trials lasting 6 months in duration, with an intermediate evaluation at 3 months, evaluating the product's efficacy, safety of the chemical agents and patient compliance.[5] Generally, agents or drugs must also receive approval by the FDA in order to be marketed in the U.S. The ADA evaluates the product itself, but the FDA evaluates the products' individual active ingredients to determine if they are recognized as safe, effective and not misbranded. All of the products included in this review have been approved by the FDA. Currently, formulation containing essential oils and methyl salicylate is the only mouth rinse that has earned the ADA seal of acceptance to be effective against plaque and gingivitis. Chlorhexidine products had previously earned the ADA seal of acceptance, but recent changes to the ADA seal program have phased out all prescription products.[4]

Methods and Materials

The purpose of this systematic review was to address the following focused question: What is the effectiveness of commercial antiseptic mouth rinses in controlling plaque and gingivitis? A secondary focused question was: What are the risks associated with daily use of antiseptic mouth rinses? The latter question targeted the effects antiseptic mouth rinses have on salivary flow rate, oral cancer and wear of composite restorations. Electronic database searches were conducted using Google Scholar and Pub Med to identify articles published between 2007 and April 2011 that compared the effectiveness of 4 commercially–marketed antiseptic mouth rinses: chlorhexidine gluconate 0.12%, essential oils and methyl salicylate, cetylpyridinium chloride 0.7% (Crest Pro Health®; Proctor & Gamble, Cincinnati, Ohio) and 20% aloe vera gel (Natural Dentist® Healthy Gums Daily Oral Rinse®, Caldwell Consumer Health, LLC, Blue Bell, Pa) for controlling plaque and gingivitis. Table I provides an overview of the 4 antiseptic mouth rinse formulations reviewed. Criteria for inclusion included controlled clinical trials and systematic reviews appearing in English language publications providing data on comparative effectiveness in controlling plaque and gingivitis, as well as risks, including salivary flow rate, oral cancer and wear of composite restorations. Selected studies reference list were screened for additional papers.

Table II provides a list of key words used in the search strategy. Eligibility criteria included peer reviewed journals, controlled clinical trials, randomized controlled clinical trials and/or longitudinal studies. Abstracts were screened for relevancy to the focus question in order to be considered. Hundreds of articles were screened and 42 were chosen that met the inclusion criteria. Full text papers were reviewed independently by the authors for inclusion in the study.

Results and Discussion

Patients rely on dental professionals to recommend products that will benefit their oral health. Substantivity determines a product's effectiveness. It is the length of time the ingredients remain active after they are applied to the area of treatment, absorption to the available soft tissues and the subsequent slow release into the saliva. The longer the product's active ingredients remain in the oral cavity the greater the products effectiveness.[6]

Saliva is continually refreshed, rinsing away the active ingredients of mouth rinse. But plaque remaining after mechanical cleaning absorbs mouth rinse antimicrobials, serving as a reservoir to prolong the product's substantivity. Plaque most frequently remains in fissures, interproximal spaces and at the gingival margin where antimicrobial activity is needed most. This theory does not promote incomplete oral hygiene, but does reduce the negative effects of plaque left behind and reinforces the benefits of mouth rinse use in patients with poor plaque control.[7]

Dental professionals should be recommending antiseptic mouth rinses that have extended substantivity, however, consideration for the patient's taste preference, history of alcoholism, religious beliefs and/or their severity of periodontal disease must be considered when making a recommendation. There are many studies comparing the effectiveness of 0.12% chlorhexidine gluconate, essential oils and methyl salicylate, 0.7% cetylpyridinium chloride and 20% aloe vera gel in controlling plaque and gingivitis. Studies included used one active agent group that was compared against a placebo and/or vehicle control groups. The outcome for comparisons as-

Table I: Popular OTC and RX Mouth Rinses

Product	Crest Prohealth®	Peridex®	The Natural Dentist Healthy Gums®	Listerine®
	Cetylpyridinium Chloride	Chlorhexidine Gluconate	Herbal	Essential Oils
	No Alcohol	Contains Alcohol	No Alcohol	Contains Alcohol
Ingredients	Active ingredients: • Cetylpyridinium Chloride (0.07%) Inactive ingredients: • water, glycerin, flavor, poloxamer 407, sodium saccharin, methyl paraben, propyl paraben, propolyne glycol, blue 1, 6 yellow and green 3	Active ingredients: • Chlorhexidine Gluconate 0.12% Inactive ingredients: • water, 11.6% alcohol, glycerin, PEG–40 sorbitan diisostearate, flavor, sodium saccharin, coloring	Active ingredients: • Aloe Vera 20% Inactive ingredients: • Purified Water, Vegetable Glycerin, Echinacea, Goldenseal, Calendula, Citric Acid, Grapefruit Seed Extract, natural flavors, poloxamar 407, vitamin B12	Active ingredients: • Eucalyptol 0.092%, Menthol 0.042%, Methyl Salicylate 0.060%, Thymol 0.064% Inactive ingredients: • water, alcohol 21.6%, sorbitol solution, flavoring, poloxamer 407, benzoic acid, sodium saccharin, sodium benzoate, F D & C green #3
Suggested Use	Twice daily rinse for 30 seconds with 2/3 fl. oz and spit	After brushing and thoroughly rinsing with water, rinse with ½ fl. oz for 30 seconds	Twice daily rinse for 30 seconds with ½ fl. oz and spit	Twice daily rinse for 30 seconds with 2/3 fl. oz and spit
Adverse Effects	Surface–level brown tooth discoloration, ulcerations and burning	Staining of oral surfaces, an increase in calculus formation and an alteration in taste perception	Mouth irritation	Burning, caustic injury, gingival pain, mucosal sloughing, glossitis, black hairy tongue, candiddiasis
Efficacy Claims	CPC interacts with bacterial membrane and dissolves it, effectively fighting plaque, gingivitis and bad breath for up to 12 hours.	Effective FDA approved gingivitis care.	Oils help prevent and reduce plaque and gingivitis, cleansing, soothing, & breath freshener	Kills germs on contact, prevents and reduces plaque and gingivitis, freshens breath, kills germ between teeth
ADA Approved	No	No	No	Yes
Website	www.crest.com	www.3M.com	www.revivepersonalproducts.com	www.listerine.com
Cost	33.8 oz/$6.99	RX only 16oz/$22.00	16.9 oz/$6.99	50 oz/$5.30

sessed test subjects for gingivitis by the plaque index (PI), gingival index (GI) and/or bleeding on probing (BOP). The results of these studies are reviewed below.

Chlorhexidine Gluconate 0.12% Mouth Rinse

Chlorhexidine gluconate is the most effective antiseptic mouth rinse available today. Chlorhexidine tightly binds to tooth structure, oral tissues and dental plaque and releases slowly, resulting in 8 to 12 hour substantivity.[8] Side effects, such as brown staining, calculus formation and temporary loss of taste, limit the long term use of this product.[9] The mechanisms of action for this mouth rinse are rupturing of the bacterial cell membrane resulting in cell death and inhibiting pellicle formation and plaque colonization. Chlorhexidine has been shown to penetrate dental plaque biofilm killing pathogens. Due to the reduced effectiveness caused by positively charged

dentifrice ingredients interacting with chlorhexidine, it is recommended to rinse 30 minutes after tooth brushing.[10]

Chlorhexidine gluconate can be alcohol or non–alcohol based. The most commonly prescribed chlorhexidine product (Peridex®) contains alcohol. Chlorhexidine mouth rinse products are available by prescription only, which limits patient accessibility. Side effects are a concern and should be discussed with the patient before prescribing so that risk versus benefit can be evaluated. This product is typically recommended to patients with moderate to severe periodontal disease when short term plaque control is critical and for post–operative procedures. Rarely is chlorhexidine used on a long term basis as a home care adjunct.[11]

Seven studies were reviewed comparing the effectiveness of chlorhexidine, essential oils and aloe vera gel formulations.[9,11–16] Of those, 4 found chlorhexidine to be superior to both essential oils and aloe vera gel,[9,13,14,16] 2 found no significant difference between chlorhexidine and essential oils[11,12] and 1 found no significant difference between chlorhexidine and aloe vera gel.[15] In Gunsolley's 2006 meta–analysis of 6 month randomized clinical trials, all 7 studies reviewed agreed that chlorhexidine was more effective in reducing plaque and gingival inflammation than mouth rinses containing essential oils.[17] Although studies consistently find chlorhexidine gluconate provides the greatest anti–plaque and anti–gingivitis benefits available today, the negative side effects associated with long term use and limited availability (prescription only) may decrease patient compliance and/or the frequency of professional recommendation.[11]

Essential Oils and Methyl Salicylate Mouth Rinse

Essential Oils refer to over the counter antiseptic mouth rinse containing 2 phenol related essential oils, thymol and eucalyptol mixed with menthol and methyl salicylate in a hydro–alcoholic vehicle. It is the antiseptic mouth rinse with the longest history, dating back to the nineteenth century. Most essential oils contain alcohol (as a solvent) at a concentration of approximately 22%, which is contraindicated for young children and patients who are immune–compromised, have mucositis, a history of alcohol abuse and/or undergoing radiation therapy for head and neck cancer.[18]

The mechanisms of action for this antiseptic mouth rinse formulation are two–fold: rupturing of the bacterial cell membrane resulting in cell death and preventing bacterial aggregation and recolonization, thus decreasing plaque mass. It has been demonstrated that essential oils can penetrate dental plaque biofilm killing pathogens even in interproximal spaces.[18] Because of its diffusion into the biofilm, essential oils have substantive activity extending several hours beyond the rinsing period. It is the only mouth rinse available today that is approved by the ADA for chemotherapeutic control of supragingival plaque and gingivitis.[5]

Table II: List of Key Words Used in Search

Key Words	Number of Articles Found
Cetylpyridinium chloride mouth rinse	69
Crest Pro Health® mouth rinse	13
Chlorhexidine gluconate mouth rinse	39
Peridex® mouth rinse	96
Essential oils mouth rinse	56
Listerine® mouth rinse	238
Herbal mouth rinse	12
Healthy Dentist® mouth rinse	7
Anti–gingivitis mouth rinse	4
Anti–plaque mouth rinse	18
Gingivitis clinical studies and mouth rinse	69
Gingivitis clinical trials and mouth rinse	134
Dry mouth and alcohol containing mouth rinse	14
Xerostomia and alcohol containing mouth rinse	13
Salivary flow and alcohol containing mouth rinse	11
Bioavailability and alcohol containing mouth rinse	6
Substantivity and mouth rinse	8
Alcohol mouth rinse and oral cancer	23
Dental restorations and alcohol containing mouth rinse	36
Dental composites and alcohol containing mouth rinse	55

Ten studies were reviewed comparing the effectiveness of essential oils, chlorhexidine, cetylpyridinium and aloe vera gel formulations.[3,11–16,19–21] Of these, 3 studies found chlorhexidine superior to both essential oils and aloe vera gel,[13,14,16] 3 studies comparing essential oils and cetylpyridinium found no difference,[3,19,21] 2 studies testing essential oils and chlorhexidine found no difference,[11,12] 1 study found essential oils better than cetylpyridinium[20] and 1 study found aloe vera gel superior to essential oils

and chlorhexidine.[15] In the 2006 meta–analysis, 20 studies reviewed claimed chlorhexidine to be 40% more effective in reducing plaque and gingival inflammation than mouth rinses containing essential oils.[17] One author speculated that the burning sensation when rinsing with essential oils may contribute to decreased rinsing time, diminishing its effectiveness.[22] Searching review of the literature suggests that essential oil mouth rinse continues to test well when compared to therapeutic mouth rinse other than chlorhexidine.

Cetylpyridinium Chloride 0.7% Mouth Rinse

Cetylpyridinium Chloride is a quaternary ammonium compound that has antiseptic properties. It is a broad spectrum antimicrobial agent which has proven effective for preventing supragingival plaque formation and reducing gingivitis.[22] Similar to other antiseptic mouth rinses, cetylpyridinium ruptures the bacterial cell membrane, causing leakage of intracellular material and ultimately cell death. Cetylpyridinium has been shown to alter bacterial metabolism and growth. The chemical binds to both tooth structure and dental plaque biofilm producing substantive action for up to 6 hours after rinsing.[23] Like chlorhexidine, cetylpyridinium rinse may be adversely affected by ingredients found in dentifrice. Rinsing with water prior to use or waiting 30 minutes after brushing is recommended. Side effects are similar to chlorhexidine, but less severe. Cetylpyridinium is cleared from the mouth more rapidly than chlorhexidine, which explains the lower efficacy for this compound.[24]

Five studies were reviewed comparing the effectiveness of cetylpyridinium, chlorhexidine and essential oil formulations.[5,12,19–21] Of these, 3 studies found no difference between essential oils and cetylpyridinium,[5,19,21] 1 study favored essential oils over cetylpyridinium[20] and 1 found both essential oils and chlorhexidine better than cetylpyridinium in reducing plaque and gingival inflammation.[12] Seven studies reviewed in the 2006 meta–analysis showed inconsistent results because cetylpyridinium chloride concentrations varied between 4.5 to 7%. Although the 6 month results were promising for the non–alcohol–based product, more long–term studies are recommended to establish a greater level of evidence comparable to the evidence available for chlorhexidine and essential oils mouth rinse agents.[17] To date, Crest Pro–Health® has not earned the ADA seal of approval.[25]

Aloe Vera Gel 20% Mouth Rinse

Natural, organic and herbal products are gaining popularity among today's more educated consumers. Aloe vera gel antiseptic mouth rinses are appealing because they do not contain alcohol, artificial preservatives or artificial colors and flavors. Most herbal rinses claim only to kill bad breath germs. Although one manufacturer (Natural Dentist®) claims anti–plaque and anti–gingivitis effectiveness, there is limited research data supporting this claim.[13–16,26]

Echinacea, goldenseal and grape fruit seed extract are 3 active ingredients in antiseptic aloe vera gel mouth rinse that exhibit anti–inflammatory and anti–fungal therapeutic effects.[14] The mechanism of action for these herbal extracts is disruption of the bacterial membrane and release of the cytoplasmic contents, within 15 minutes after rinsing.[26] Research by Kaim et al indicates antiseptic aloe vera gel mouth rinse significantly reduces salivary aerobic, microaerophilic and anaerobic bacteria for up to 2 hours.[16] The exact substantivity is still unclear – more research is needed to determine this.[9]

Four studies were reviewed comparing the effectiveness of aloe vera gel, chlorhexidine and essential oil formulations.[9,14–16] Two in vitro studies produced conflicting results, with 1 study finding aloe vera gel to be significantly better then chlorhexidine and essential oils.[15] The second study found chlorhexidine to be better than aloe vera gel and aloe vera gel to be better than essential oils.[14] Two in vivo studies were conducted on a small number of participants. The larger of these, with 63 randomly assigned participants, found chlorhexidine to be significantly better than aloe vera gel.[9] In the smaller study, 20 volunteers participated, with results favoring chlorhexidine as most effective, followed by aloe vera gel and essential oils, respectively, in reducing plaque and gingival inflammation.[16] There is limited research available to support recommending aloe vera gel over other antiseptic mouth rinse to control gingival diseases.

A secondary focus question was: "What are the risks associated with daily use of antiseptic mouth rinses?" This question targeted the effects antiseptic mouth rinses has on salivary flow rate, oral cancer and wear of composite restorations. The results of the literature review are summarized below.

Alcohol Containing Mouth Rinse and Salivary Flow Rate

Many antiseptic mouth rinse products contain alcohol (ethanol) to keep flavoring agents and active ingredients in solution and biologically active.[4] A number list alcohol as an active ingredient, claiming antiseptic and/or germicidal properties. In order to be considered an effective germicide, alcohol concentrations should range between 50 to 70%. Even the highest alcohol concentration available today

(26.9%) is well below the level necessary for alcohol to be considered an effective antiseptic.[16] Alcohol–based mouth rinse has been linked to desiccation of the oral mucosal membrane. Many dental care providers have a misconception that alcohol in mouth rinse negatively affects the flow rate of saliva and/or the salivary pH in patients that already have xerostomia.[4]

Xerostomia is well–defined as a reduction or absence of saliva in the mouth, a subjective perception of dry mouth.[27] Most often, xerostomia is a side effect of certain medications, radiation therapy for oral pharyngeal cancer and/or systemic diseases like diabetes and Sjogren's syndrome. With the recent increase in these compounding factors, dry mouth is a major concern for today's dental provider. Dental diseases such as caries, gingivitis and periodontitis are all exacerbated with low salivary flow.[28] All 3 research studies reviewed that focused on the effects of salivary flow and alcohol–based mouth rinses reported no significant difference in salivary flow rate or salivary pH related to the use of alcohol based mouth rinse.[27–29] Any perception of dry mouth immediately following rinsing is of short duration. Therefore, alcohol containing products can be recommended to most patients.

Alcohol Containing Mouth Rinse and Oral Cancer

Oral and oropharyngeal cancers are considered the sixth most common cancers in the world.[30] According to the National Cancer Institute, "the age–adjusted incidence rate was 10.4 per 100,000 men and women per year," and "the age–adjusted death rate was 2.5 per 100,000 men and women per year" based on cases from 2003 to 2007.[31] In the past 3 decades, 9 epidemiologic studies have been conducted on the relationship between alcohol containing mouthwash (ACM) use and the risk for oropharyngeal cancer.[32] Of these only 3 reported positive, but weak findings.

Rinsing with an ACM is considered low alcohol exposure when used according to manufacturer directions: 0.5 fl oz of 25% alcohol twice daily for 30 seconds. This type of exposure is equivalent to the consumption of 1 to 2 alcoholic beverages per day, which would most likely not increase the risk for oral cancer. Mouth rinse use is known to be higher among drinkers and smokers. It is difficult to eliminate the confounding effects of these variables in research studies. The mechanism by which alcoholic beverages may induce human oral cancer is related to the ingestion, topical exposure and/or solvent action that enhances absorption of tobacco and other carcinogens into the tissues.[32]

The International Agency for Research on Cancer has identified the long term habitual consumption of alcoholic beverages can greatly increase the risk for oral cancers.[33] Commercial mouth rinse contains pharmaceutical grade ethanol alcohol, which has not been found as a carcinogen. Alcoholic beverages contain chemicals and additives, such as urethane, which is a known carcinogen.[34] Current evidence strongly suggests that ACM use does not increase the risk for oral cancer.[27,30,32–35] Research must meet certain criteria to establish a causal relationship between ACM and oral cancer that would be acceptable to the scientific community. Shortcomings in study design are blamed for the failure of studies to date supporting a connection between oral cancer and alcohol containing mouth rinses.[33]

Mouth Rinse and Composite Restoration Wear

Antiseptic mouth rinse active ingredients and/or low pH may affect the hardness, gloss, color and wear of composite restorations.[36] Of the mouth rinses reviewed, chlorhexidine and aloe vera gel are less acidic (5 to 7 pH)[25,37] than essential oils and cetylpyridinium (3.8 to 4.8 pH).[37,38] During bacterial acid attacks, enamel subsurface dissolution occurs at this same pH range of 3.8 to 4.8.[39] These facts suggest that essential oils and cetylpyridinium products could have a negative effect on restorations due to low pH, especially in patients who use these products excessively.

Five recent studies evaluating the effects of antiseptic mouth rinse on composite restorations found that mouth rinses containing alcohol have a greater effect than non alcohol formulations, deducing that alcohol may cause composite wear.[36,37,40–42] Aesthetics have become a top priority for patients, evidenced by the popularity of bleaching procedures both in office and at home treatments. One study evaluating the effects of alcohol containing mouth rinse on composite resins that had been subjected to prior bleaching found that all tested rinses had a statistically significant negative effect on surface hardness, gloss and color of the restorations.[41]

Variables that influence the effect of antiseptic mouth rinse on a composite restoration are: age of the restoration, material composition and surface roughness.[37] With the aging population of America, it is important to consider the effects these products could have on our geriatric patients. Other negative effects may depend on in vivo factors that cannot be replicated in vitro. Research studying the effects of antiseptic mouth rinse on composite restorative materials is limited. Due to the constant influx of new restorative materials, routine assessment and testing is recommended.[41]

Conclusion

Gingivitis and periodontitis are among the most prevalent infections afflicting humans, making it essential for dental professionals to include risk assessment and disease management in patients' treatment plans to insure a favorable outcome. Risk factors for periodontitis to be considered include pathogen burden (specific microbes), systemic factors (diabetes, HPV, medications, etc.) behavioral habits (tobacco use, home care, etc.) and local factors (tooth proximity, faulty restorations, etc.).[43]

Although research supports the effectiveness of antiseptic mouth rinse as adjunctive therapy to reduce plaque and gingivitis, patients must be advised that these products have little effect on periodontitis. Studies have found that agents used in rinsing can only reach 21% of a 1 to 6 mm periodontal pocket.[9] Therefore, recommending the use of anti–plaque and anti–gingivitis antiseptic mouth rinse can be considered only as an adjunct for helping our patients control gingival diseases.

Strong evidence exists supporting the effectiveness of daily antiseptic mouth rinse used as an adjunct to mechanical plaque control to reduce or control plaque and gingivitis.[17] Chlorhexidine gluconate 0.12% is the most effective mouth rinse available today, but side effects should be considered.[8] ADA approved essential oils and methyl salicylate are very effective in controlling gingival disease, with less side effects than chlorhexidine.[5] Cetylpyridinium chloride 0.7% and 20% aloe vera gel do not test as well as chlorhexidine or essential oils, but may be an option for certain patients. Health professionals should continually review products and evaluate their effectiveness based on evidence before making a recommendation to their patients.

Diane R. Osso, RDH, MS, is a full-time faculty member at the Community College of Denver Dental Hygiene Program. Nehal Kanani, RDH, BS, is a degree completion student at the University of Mayland's Department of Periodontics

References

1. Barnett ML. The rationale for the daily use of an antimicrobial mouth rinse. *J Am Dent Assoc*. 2006;137(Suppl):16S–21S.

2. Mager DL, Ximenez–Fyvie LA, Haffajee AD, Socransky SS. Distribution of selected bacterial species on intraoral surfaces. *J Clin Periodontol*. 2003;30(7):644–654.

3. Albert-Kiszely A, Pjetursson BE, Salvi GE, et al. Comparison of the effects of cetylpyridinium chloride with an essential oil mouth rinse on dental plaque and gingivitis – a six month randomized controlled clinical trial. *J Clin Periodontol*. 2007;34(8):658–667.

4. DePaola LG, Spolarich AE. Safety and Efficacy of Antimicrobial Mouth rinses in Clinical Practice. *J Dent Hyg*. 2007;13–25.

5. Acceptance Program Guidelines. Chemotherapeutic Products for Control of Gingivitis. American Dental Association [Internet]. 2008 July [cited 2010 November 5]. Available from: http://www.ada.org/sections/scienceAndResearch/pdfs/guide_chemo_ging.pdf

6. Dental Glossary [Internet]. [cited 2010 November 5]. Available from: http://www.dentalglossary.net/definition/2405–Substantivity

7. Otten MP, Busscher HJ, van der Mei HC, Abbas F, van Hoogmoed CG. Retention of Antimicrobial Activity in Plaque and Saliva following Mouth rinse Use in vivo. *Caries Res*. 2010;44(5):459–464.

8. Zanatta FB, Antoniazzi RP, Rösing CK. The Effect of 0.12% Chlorhexidine Gluconate Rinsing on Previously Plaque–Free and Plaque–Covered Surfaces: A Randomized, Controlled Clinical Trial. *J Periodontol*. 2007;78(11):2127–2134.

9. Southern EN, McCombs GB, Tolle SL, Marinak K. The Comparative Effects of 0.12% Chlorhexidine and Herbal Oral Rinse on Dental Plaque–Induced Gingivitis. *J Dent Hyg*. 2006;80(1):1–3.

10. Foster JS, Pan PC, Kolenbrander PE. Effects of antimicrobial agents on oral biofilms in a saliva conditioned flow–cell. *Biofilms*. 2004;1(1):5–12.

11. Charles CH, Mostler KM, Bartels LL, Mankodi SM. Comparative anti–plaque and anti–gingivitis effectiveness of chlorhexidine and an essential oil mouth rinse: 6 month clinical trial. *J of Clin Periodontol*. 2004;31(10):878–884.

12. Pan PC, Harper S, Ricci–Nittel D, Lux R, Shi W. In-vitro evidence for efficacy of antimicrobial mouthrinses. *J Dent.* 2010;38(Suppl 1):S16–S20.

13. Roberts C, Murray L, Veiga N, Teles R, Martin L, Socransky SS, Haffajee AD. Comparison of the Clinical and Microbiological Effects of Four Rinses. The Forsyth Institution [Internet]. 2008. Available from: http://www.revivepersonalproducts.com/media/image/invivo_092608.pdf

14. Haffajee AD, Yaskell T, Socransky SS. Antimicrobial effectiveness of an herbal mouth rinse compared with an essential oil and a chlorhexidine mouth rinse. *J Am Dent Asooc*. 2008;139(5):606–611.

15. Kaim JM, Gultz J, Do L, Scherer W. An In Vitro Investigation of the Antimicrobial Activity of an Herbal Mouth rinse. *J Clin Dent*. 1998;9(2):46–48.

16. Gultz J, Kaim JM, DeLeo J 4th, Scherer W. An In Vivo Comparison of the Antimicrobial Activities of Three Mouth rinses. *J Clin Dent*. 1998;9(2):43–45.

17. Gunsolley JC. A meta–analysis of six–month studies of antiplaque and antigingivitis agents. *J Am Dent Assoc*. 2006;137(12):1679–1656.

18. Stoeken JE, Paraskevas S, Van der Weijden GA. The Long–Term Effect of Mouth rinse Containing Essential Oils on Dental Plaque and Gingivitis: A Systematic Review. *J Periodontol*. 2007;78(7):1218–1228.

19. Albert-Kiszely A, Pjetursson BE, Salvi GE, et al. Comparison of the effects of cetylpyridinium chloride with an essential oil mouth rinse on dental plaque and gingivitis– a six month randomized controlled clinical trial. *J Clin Periodontol*. 2007;34(8):658–667.

20. Amini P, Araujo MW, Wu MM, Charles CA, Sharma NC. Comparative anti–plaque and anti–gingivitis efficacy of three antiseptic mouth rinses: a two week randomized clinical trial. *Braz Oral Res*. 2009;23(3);319-325.

21. Witt JJ, Walters P, Bsoul S, Gibb R, Dunavent J, Putt M. Comparative clinical trial of two antigingivitis mouthrinses. *Am J Dent*. 2005;18:15A–17A.

22. Blenman TV, Morrison KL, Tsau GJ, Medina AL, Gerlach RW. Practice implications with an alcohol–free, 0.07% cetylpyridinium chloride mouth rinse. *Am J Dent*. 2005;18:29A–34A.

23. Elworthy A, Greenman J, Doherty FM, Newcombe RG, Addy M. The substantivity of a number of oral hygiene products determined by the duration of effects on salivary bacteria. *J Periodontol*. 1996;67(6):572–576.

24. Haps S, Slot DE, Berchier CE, Van der Weijden GA. The effect of cetylpyridinium chloride mouth rinses as adjuncts to tooth brushing on plaque and parameters on gingival inflammation: a systematic review. *Int J Dent Hyg*. 2008;6(4):290–303.

25. ADA Seal Product List. American Dental Association [Internet]. [cited 2011 October 11]. Available from: http://www.ada.org/adasealproducts.aspx

26. Scherer W, Gultz J, Lee SS, Kaim J. The Ability of an Herbal Mouthrinse to Reduce Gingival Bleeding. *J Clin Dent*. 1998;9(4):97–100.

27. Silverman S Jr., Wilder R. Antimicrobial mouth rinse as a part of a comprehensive oral care regimen: Safety and compliance factors. *J Am Dent Assoc*. 2006;137(11 S):22S–26S.

28. Kerr AR, Katz RW, Ship JA. A comparison of the effects of 2 commercially available nonprescription mouth rinses on salivary flow rates and xerostomia. *Quintessence Int*. 2007;38(8):e440–e447.

29. Cortelli SC, Cortelli JR, Holzhausen M, et al. Essential oils in one stage full–mouth disinfection: double–blind, randomized clinical trial of long–term clinical, microbial and salivary effects. *J Clin Periodontol*. 2009;36(4):333–342.

30. Warnakulasuriya S. Causes of oral cancer – an appraisal of controversies. *Br Dent J*. 2009;207(10):471–475.

31. SEER Stat Fact Sheets: Oral Cavity and Pharynx. National Cancer Institute [Internet]. 2010 [cited 2010 September 24]. Available from: http://seer.cancer.gov/statfacts/html/oralcav.html

32. Cole P, Rodu B, Mathisen A. Alcohol–containing mouthwash and oropharyngeal cancer: A review of the epidemiology. *J Am Dent Assoc*. 2003;134(8):1079–1087.

33. Science brief on alcohol–containing mouth rinses and oral cancer. American Dental Association [Internet]. 2009 [cited 2010 September 11]. Available from: http://www.ada.org/sections/professionalResources/pdfs/topics_cancer_brief_mouthrinses.pdf

34. Food and Drug Administration. Oral health care drug products for over–the–counter human use: anti–gingivitis/anti–plaque drug products; Establishment of a monograph; proposed rules. *Fed Regist*. 2003;68:32232–32287.

35. Winn DM, Blot WJ, McLaughlin JK, et al. Mouthwash Use and Oral Conditions in the Risk of Oral and Pharyngeal Cancer. *Cancer Res*. 1991;51(11):3044–3047.

36. Yap AU, Tan BW, Tay LC, Chang KM, Loy TK, Mok BY. Effect of mouthrinses on microhardness and wear of composite and compomer restoratives. *Oper Dent*. 2003;28(6):740–746.

37. Cavalcanti AN, Mitsui FH, Ambrosano GM, Mathias P, Marchi GM. Effect of different mouthrinses on Knoop hardness of a restorative composite. *Am J Dent*. 2005;18(6):337–340.

38. Riley L. Crest Pro health – Contact Us. Crest [Internet]. [cited 2010 December 10]. Available from: http://www.crest.com/contact–crest.aspx

39. Collins FM, Florman M. Topical Fluoride: Mechanism of action. Fluoride Guide. The Academy of Dental Therapeutics and Stomatology® [Internet]. 2010. Available from: http://www.ineedce.com/courses/1850/PDF/1003cei_fluoride%20guide_Web_1.pdf

40. Diab M, Zaazou MH, Mubarak EH, Olaa MI. Effect of Five Commercial Mouth rinses on the micro hardness and Color Stability of Two Resin Composite Restorative Materials. *Australian J Basic Applied Sci*. 2007;1(4):667–674.

41. Gurgan S, Yalcin Cakir F. The Effect of Three Different Mouth rinses on the Surfaces Hardness, Gloss and Colour Change of Bleached Nano Composite Resins. *Eur J Prosthodont Restor Dent*. 2008;16(3):104–108.

42. de Carvalho Rocha AC, Araujo de Lima CS, da Silva Santos MC, Resende Montes MAJ. Evaluation of Surface Roughness of a Nanofill Resin Composite after Simulated Brushing and Immersion in Mouth rinses, Alcohol and Water. *Materials Res*. 2010;13(1):77–80.

43. Lamster IB. Antimicrobial mouth rinses and the management of periodontal disease. *J Am Dent Assoc*. 2006;137(11S):5S–9S.

Chronic Inflammatory Gingival Enlargement Associated with Orthodontic Therapy – A Case Report

Tanya Jadhav, MDS; K Mahalinga Bhat, MDS; G Subraya Bhat, MDS; Jothi M Varghese, MDS

Abstract

Purpose: Gingival enlargement, also synonymous with the terms gingival hyperplasia or hypertrophy, is defined as an abnormal overgrowth of gingival tissues. A case of a 19–year–old male presenting with maxillary and mandibular chronic inflammatory gingival enlargement associated with prolonged orthodontic therapy is reported here. Surgical therapy was carried out to provide a good aesthetic outcome. No recurrence was reported at the end of 1 year. The importance of patient motivation and compliance during and after therapy as a critical factor in the success of treatment has also been highlighted through this case report.

Keywords: Gingival enlargement, chronic inflammation, orthodontic therapy, compliance, motivation

This study supports the NDHRA priority area, **Clinical Dental Hygiene Care:** Assess the use of evidence–based treatment recommendations in dental hygiene practice.

Introduction

Gingival enlargement, a globally accepted terminology for an increase in the size of the gingiva, is a general feature of gingival diseases. It is a multifactorial condition that develops in response to various stimuli and interactions between the host and the environment. It may be plaque–induced or associated with systemic hormonal disturbances. It also occurs as a manifestation associated with several blood dyscrasias, such as leukemia, thrombocytopenia or thrombocytopathy. A rare variant, idiopathic gingival fibromatosis, with a familial inheritance, has also been reported.[1] Based on the extent and severity, these enlargements may lead to functional disturbances like altered speech, difficulty in mastication and aesthetic and psychological problems.

Inflammatory gingival enlargement may be categorized as acute or chronic, wherein chronic changes are much more common.[1] The ability to perform oral hygiene measures is compromised in some patients with gingival enlargements, which may be further complicated by the presence of prosthesis and fixed orthodontic appliances. This may lead to more inflammation and further plaque accumulation perpetuating this vicious cycle. Thus, there is a transformation of the gingival sulcus into a periodontal pocket creating an area where plaque removal becomes impossible.

One of the most important determinants of treatment outcomes is patient compliance. The willingness to perform adequate oral hygiene measures and receive timely periodic recalls and treatment are deemed essential for a successful outcome. The therapeutic approaches related to gingival enlargement are based on the underlying etiology and the subsequent changes it manifests on the tissues. The prime treatment modalities involve obtaining a detailed medical history and non–surgical periodontal therapy, followed by surgical excision to retain esthetical and functional demands.

This case report presents a case of chronic gingival enlargement associated with prolonged orthodontic therapy.

Case Report

A 19–year–old male patient reported to the Department of Periodontology, Manipal College of Dental Sciences, Manipal, India. The patient complained of swelling of the upper and lower gums in the front tooth region. The patient had noticed the swelling 3 years prior and reported that it had not increased in size since then. He also complained of bleeding from the gums while brushing. The patient revealed that he had undergone incomplete orthodontic treatment which was initiated 6 years prior. There was no other relevant medical, dental or family history.

Consistent with the history of incomplete orthodontic treatment, intraoral inspection revealed orthodontic molar bands and brackets on all teeth except the maxillary left central incisor. On clinical examination, marginal and papillary gingiva ap-

peared red and enlarged in the maxillary and mandibular arches, which was more prominent in the anterior sextants and also more pronounced on the right side as compared to the left (Figures 1–3). Further soft tissue assessment revealed soft and edematous consistency and bleeding on probing on all teeth.

A treatment plan consisting of initial periodontal therapy followed by a gingivectomy procedure was planned to improve aesthetics and function. The initial periodontal therapy comprising supragingival and subgingival scaling was performed. Oral hygiene instructions were given and the use of chlorhexidine mouthwash (0.2% Clohex™, Dr. Reddy's Laboratories Ltd., India) twice a day for one week was advised. At the next visit, in spite of use of the prescribed medicated mouthwash, the gingival enlargement did not show considerable reduction in size, but the tissues appeared to be firm in consistency. At this stage, radiographs were taken and complete blood count investigations (RBC, WBC and platelet counts, ESR, bleeding time, clotting time, prothrombin time) were carried out (Figure 4).

Results

These investigations were non–contributory. An internal bevel gingivectomy was performed for the maxillary sextant. The excised tissue was sent for histopathological examination. Following this, the patient failed to report for subsequent recall appointments.

The histopathological examination revealed a hyperplastic parakeratinized epithelium overlying inflamed connective tissue. The underlying stromal tissue showed numerous proliferating young fibroblasts admixed with focal aggregates of chronic inflammatory cells. Few fibroblasts appeared stellate, with numerous nuclei distributed in a collagenized stroma. At places the stromal tissue exhibited myxoid degeneration. A histopathological diagnosis suggestive of inflammatory fibrous hyperplasia was given (Figure 5).

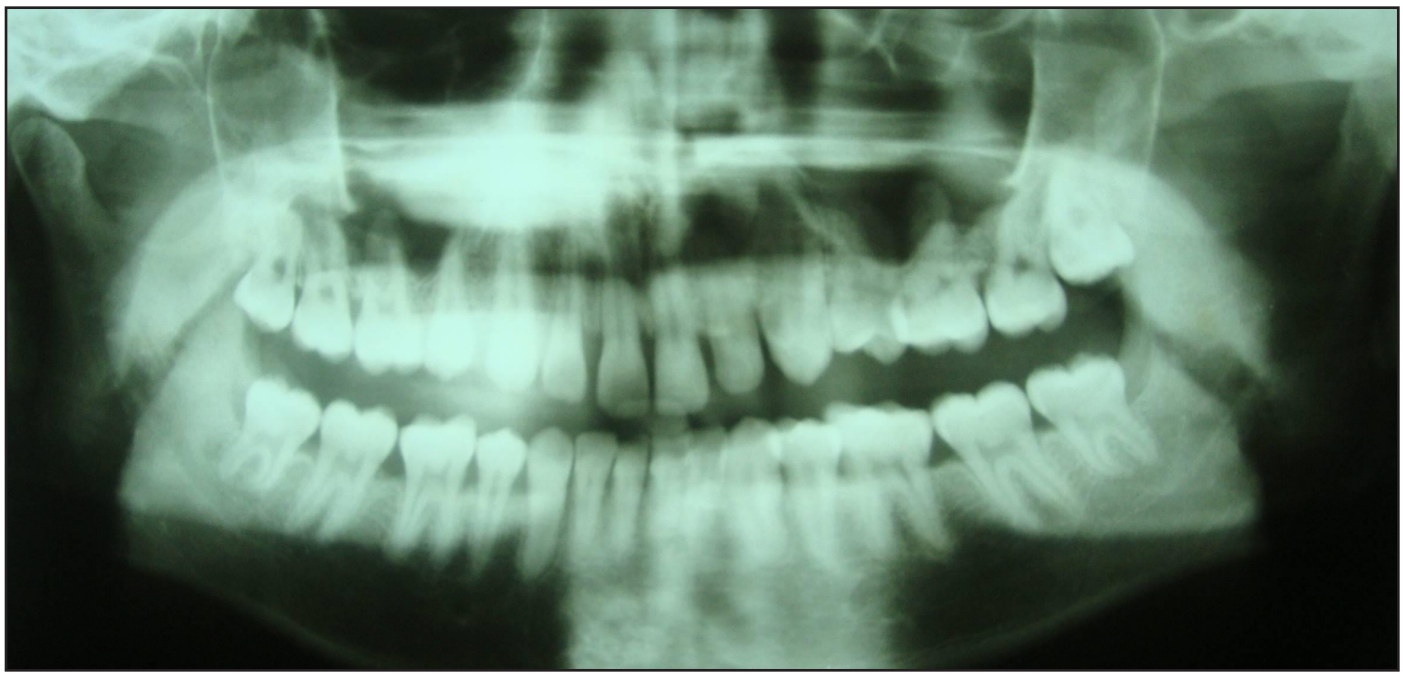
One year later, the patient reported back to the clinic. At this stage, the patient also expressed the unwillingness to continue the orthodontic therapy. Intraoral examination revealed that the maxillary surgical site had healed satisfactorily. There was no recurrence of the gingival enlargement in the maxillary anterior sextant (Figure 6). However, enlargements in the untreated areas persisted. Initial periodontal therapy was performed again and oral hygiene instructions were reinforced. To further improve plaque control measures, the orthodontic appliances were removed at this

Figure 1: Intra–oral pre–operative right lateral view

Figure 2: Intra–oral pre–operative frontal view

Figure 3: Intra–oral pre–operative left lateral view

Figure 4: Orthopantomograph



stage by the orthodontist. The patient was also counseled regarding the importance of follow up and maintenance with special emphasis on motivation.

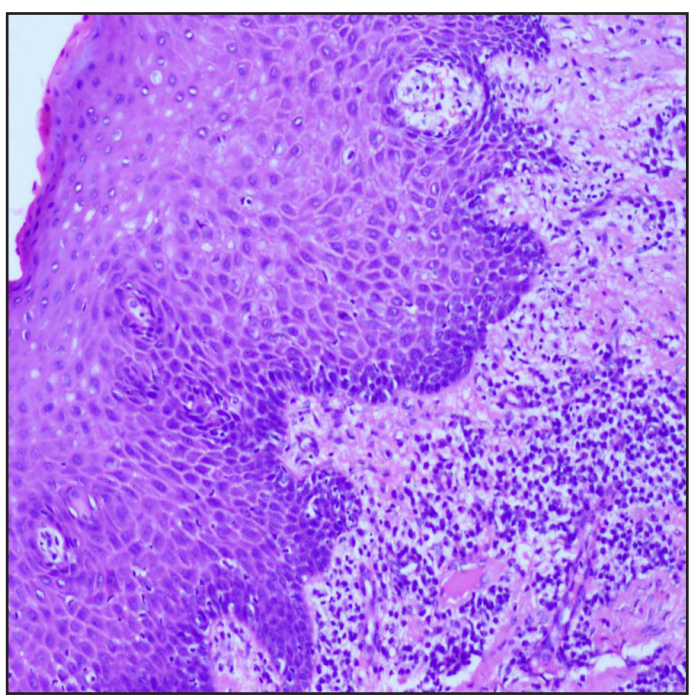
Following this, gingivectomy was performed in the mandibular anterior sextant (Figure 7) and maxillary right posterior sextant at different scheduled appointments. Then the patient was reviewed and healing was found to be satisfactory.

Discussion

Gingival overgrowth varies from mild enlargement of isolated interdental papillae to segmental or uniform and marked enlargement affecting 1 or both of the jaws with a diverse etiopathogenesis.[2]

Here, we report a case of chronic inflammatory gingival enlargement. These enlargements are often associated with a long–standing bacterial plaque accumulation. Regular professional oral prophylaxis and good patient compliance are required in the management of such cases. In this case, patient compliance was lacking as evidenced by the history of incomplete orthodontic treatment and the failure to report for regular recall appointments. Also, the presence of the appliances may have further compromised the maintenance of adequate oral hygiene. This reflects the importance of patient education, motivation and compliance during and after dental treatment. Reinforcement of effective oral hygiene is essential, since patients have a tendency to revert to their original behavior. The patient must be placed into a maintenance schedule to preserve a healthy dentition.

Figure 5: Histological section showing hyperplastic parakeratinized epithelium with fibro–collagenous connective tissue with chronic inflammatory cells (H&E 10X)



Consequently, it was noticed that once the appliances were removed and oral hygiene instructions were reinforced, the patient was able to maintain good oral hygiene. A study by Sallum et al showed significant impact of orthodontic ap-

Figure 6: Maxillary arch 12 months postoperative view after gingivectomy

Figure 7: Mandibular arch 3 months postoperative view after gingivectomy

pliance removal and professional prophylaxis on periodontal health.[3]

The patient was recommended to undergo complete blood investigations to rule out underlying systemic disease and allergies. Some authors have reported a possible allergic reaction to orthodontic metal which may cause gingival enlargement. Allergic contact stomatitis by dental metals, particularly nickel, has shown to cause gingival hyperplasia. Özkaya et al reported 2 cases with nickel–induced oral mucosal hyperplasia.[4] Although extremely rare, a hyperplastic form has also been reported in single cases from nickel in dental appliances[5,6] and from gold and palladium in a dental clasp.[7]

Orthodontic treatment–induced gingival overgrowth shows a specific fibrous and thickened gingival appearance, different from fragile gingiva with marginal gingival redness, which is seen in allergic or inflammatory gingival lesions. Histologically, inflammatory gingival hyperplasia is mainly observed as an increase and thickening of mature collagen bundles in the connective tissue stroma. Microscopic appearance of fibroblasts in the connective tissue stroma and chronic inflammatory cell component is suggestive of non–specific gingival enlargement. Fibrous gingival enlargements associated with fixed orthodontic appliances seem to be transitory, and it is generally thought that enlargement resolves after orthodontic therapy.[1] However, there are also studies reporting that this resolution is not complete.[8,9]

When chronic inflammatory gingival enlargements include a significant fibrotic component that does not resolve completely after initial periodontal therapy or does not meet the aesthetic and functional demands of the patient, surgical removal is the treatment of choice. The most widely employed surgical approaches for the treatment of gingival enlargements is gingivectomy or the flap technique.

Conclusion

This report helps to highlight the importance of patient motivation and patient compliance in treatment planning. Oral hygiene education supplemented with positive motivation should be started at the initial stages of the treatment strategy in order to obtain predictable outcomes. At each recall visit, the patient should be notified about their ongoing dental condition and the effects of risk factors like poor oral hygiene, smoking and deleterious habits on the existing oral state. Even though revolutionary advances have taken place in dental specialties, these 2 factors still play a critical role in the success of a therapeutic program. An effective communication is, thus, vital in motivating and educating patients about their dental problems. As a consequence, successful treatment outcome is believed to relate to 2 sides of the same coin, necessitating the combined efforts of both the patient and the clinician.

Tanya Jadhav, MDS, is involved in Private practice at Pune, Maharashtra-India. K Mahalinga Bhat, MDS, is a professor at the Department of Periodontics, Manipal College of Dental Sciences. G Subraya Bhat, MDS, is the Head of Department at the Department of Periodontics, Manipal College of Dental Sciences. Jothi M Varghese, MDS, is an associate professor at the Department of Periodontics, Manipal College of Dental Sciences.

References

1. Carranza FA, Hogan EL. Gingival enlargement. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA. Carranza's *Clinical Periodontology. 11th ed.* Philadelphia, Penn: W.B. Saunders Company; 2006:373–390.

2. Tiwana PS, Kok IJ, Stoker DS, Cooper LF. Facial distortion secondary to idiopathic gingival hyperplasia: surgical management and oral reconstruction with endosseous implants. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2005;100(2):153–157.

3. Sallum EJ, Nouer DF, Klein MI, et al. Clinical and microbiologic changes after removal of orthodontic appliances. *Am J Orthod Dentofacial Orthop*. 2004;126(3):363–366.

4. Özkaya E, Babuna G. Two cases with nickel–induced oral mucosal hyperplasia: A rare clinical form of allergic contact stomatitis? *Dermatol Online J*. 2011;17(3):12.

5. Kalkwarf KL. Allergic gingival reaction to esthetic crowns. *Quintessence Int Dent Dig*. 1984;15(7):741–745.

6. Counts AL, Miller MA, Khakhria ML, Strange S. Nickel allergy associated with a transpalatal arch appliance. *J Orofac Orthop*. 2002;63(6):509–515.

7. Kabashima K, Miyachi Y. Gingival hyperplasia due to metal allergy. *Clin Exp Dermatol*. 2005;30(1):88–89.

8. Ramadan AAF. Effect of nickel and chromium on gingival tissues during orthodontic treatment: a longitudinal study. *World J Orthod*. 2004;5(3):230–234, 235.

9. Gursoy UK, Sokucu O, Uitto VJ, et al. The role of nickel accumulation and epithelial cell proliferation in orthodontic treatment–induced gingival overgrowth. *Eur J Orthod*. 2007;29(6):555–558.

Dental Hygienists' Role in Practice Based Research: PEARL Network Evaluation

Ashley C. Grill, RDH, BSDH, MPH; Joanne Johnson, RDH; Damon Collie, BS, MSHS; Van P. Thompson, DDS, PhD; Ronald G. Craig, DMD, PhD; Frederick A. Curro, DMD, PhD

Abstract

Purpose: The goal of this paper is to evaluate the PEARL Network's satisfaction with training/support and assess the relationship between practice research coordinators (PRCs) involvement and study participation.

Methods: At the PEARL Network 2011 Annual Meeting, an evaluation form was completed by practitioner–investigators and PRCs who attended the annual meeting. Results from the paper evaluation form were entered into an Excel database, and analyzed using the statistical analysis software SPSS. The bivariate correlation test, Pearson Correlation, was conducted, and results were considered significant if $p<0.05$.

Results: During a program evaluation among 84 network respondents, a positive correlation ($p=0.004$) was found between the number of PRCs and the number of studies in which a site participates. In addition, there was a positive correlation between satisfaction with the training, support and involvement of PRCs in organizing study activities ($p=0.008$). There was also positive correlation between satisfaction with training/support and the number of PRCs utilized by the office ($p=0.039$).

Conclusion: Practice research coordinators are key members of the research team, and they are important to conducting clinical studies in everyday practice.

Keywords: Practice Based Research, Dental Hygiene Research

This study supports the NDHRA priority area, **Health Services Research:** Evaluate strategies that position and gain recognition of dental hygienists as a primary care providers in the health care delivery system.

Introduction

Practice based research networks (PBRNs) are designed to answer questions about everyday practice, and they have the potential to change and improve the practice of dentistry. Office based research requires the participation of the dentist, termed the Practitioner–Investigator (P–I) and a staff member designated as the practice research coordinator (PRC), who is often a dental hygienist. The Practitioners Engaged in Applied Research and Learning (PEARL) Network defines a PBRN as "collaboration between an academic health science center(s) and community practitioners conducting primarily clinical studies of mutual interest that would benefit and enhance patient care, delivery, cost, and health care policy."[1] PBRNs require an infrastructure to conduct studies and include: P–Is, PRCs, clinical research associates (CRAs), a data coordinating center, personnel to analyze results and administrators. PEARL can serve as the basis of an infrastructure to support "big science."[2] The vision of big science is that by pooling resources researchers can learn more together than from independent observations outside of organized science. An example of big science outside of health care is the large Hadron Collider near Geneva, Switzerland organized by the European Organization for Nuclear Research (CERN), which involve large numbers of investigators from many countries.[3] The advantage of big science in dentistry is that it allows researchers to evaluate practice and procedures systematically.

The Role of the PBRN in Dentistry

The PEARL Network is ideally positioned to evaluate and disseminate precise and accurate definitions related to diagnosis codes, disease states and risk factors for use in dentistry. "This may have the potential to create real–time evaluation of new advancements in medications, products, and procedures in dentistry that are relevant, practical, and applicable to everyday practice."[4] The PBRN initiative has the potential to impact the future of dentistry in many ways: it increases the knowledge base of the profession, it provides a place to find answers to questions related to clinical care, it creates a resource for providers to continue learning throughout their career and it builds connections between providers to enhance professional development. Providers report a sense of ownership of the results because they reported the data first hand.[5] PEARL provides the opportunity to increase the adoption of knowledge and transfer of information into practice thereby closing the translational gap.

The Role of the PRC in a PBRN

In many ways PRCs are essential to the success of practice based research teams, and their contributions are recognized. Dental hygienists are formally educated members of the dental team. They are well suited to present the informed consent prior to treatment, and can interpret and translate the protocol for the patient, once trained in the principles of good clinical practice. Good clinical practice "is an international ethical and scientific quality standard for designing, conducting, recording, and reporting trials that involve the participation of human subjects."[6] Some of the common strengths of PRCs include organizational expertise, communication skills and attention to detail. Dental hygienists who engage in clinical research may experience career growth and professional development. Some PRCs in the PEARL Network have commented anecdotally about greater job satisfaction through participation in research and contributing to the knowledge base of the profession. Learning skills needed to conduct standard of care studies in accordance with good clinical practice also prepares dental hygienists as teachers or educators,[7] or as research industry professionals. Through participation, PEARL Network research studies provide Network dental sites with a method to objectively measure and benchmark what is happening in the office. An example is the PEARL analgesic study and communication discrepancies reported between patients and providers in that study.[8–10] The PEARL Network found in the analgesic study that there was significant variation between the providers documented recommendation and patients perceptions for analgesics used for pain control.

Comparative Effectiveness Research (CER) and the PBRN

In oral health research, the PBRN provides the opportunity to conduct comparative effectiveness research. This type of research compares patient outcomes for various treatments and procedures looking at effectiveness, efficiency and cost data. Some policy makers believe comparative effectiveness research may have the potential to align payments with evidence based care.[11] The PEARL Network shares values with other well intentioned international health research organizations. PEARL "provide(s) answers to the complex and difficult questions that decision makers face when designing policies that affect health and health care."[12] In 2005, a major investment in the future of dentistry was made by the National Institute of Health's National Institute of Dental and Craniofacial Research (NIH/NIDCR) with the development of the PBRN program for dentistry.[13] A 7 year award was given to initiate 3 PBRNs with a focus on oral health. Starting in 2012 there will be a single PBRN in dentistry, the National Network. The NIDCR would like to grow the organization significantly during the next 7 year phase of this initiative.[14]

The PEARL Network

The PEARL Network's administrative headquarters are located at New York University in New York, and are comprised of 3 cores: the protocol development and training core, information dissemination core and the recruitment, retention and clinical operations core. The clinical operations department is staffed with a team of CRAs who work to ensure compliance under good clinical practice requirements and data integrity. The PEARL Network strongly recommends each site have a PRC. The next iteration of the grant defines PBRN P–Is as dentists, dental hygienists and other dental professionals who are engaged in the daily practice of dentistry.[15]

The goal of this paper is to evaluate a PBRN, the PEARL Network's satisfaction with training/support and assess the relationship between PRCs involvement and study participation.

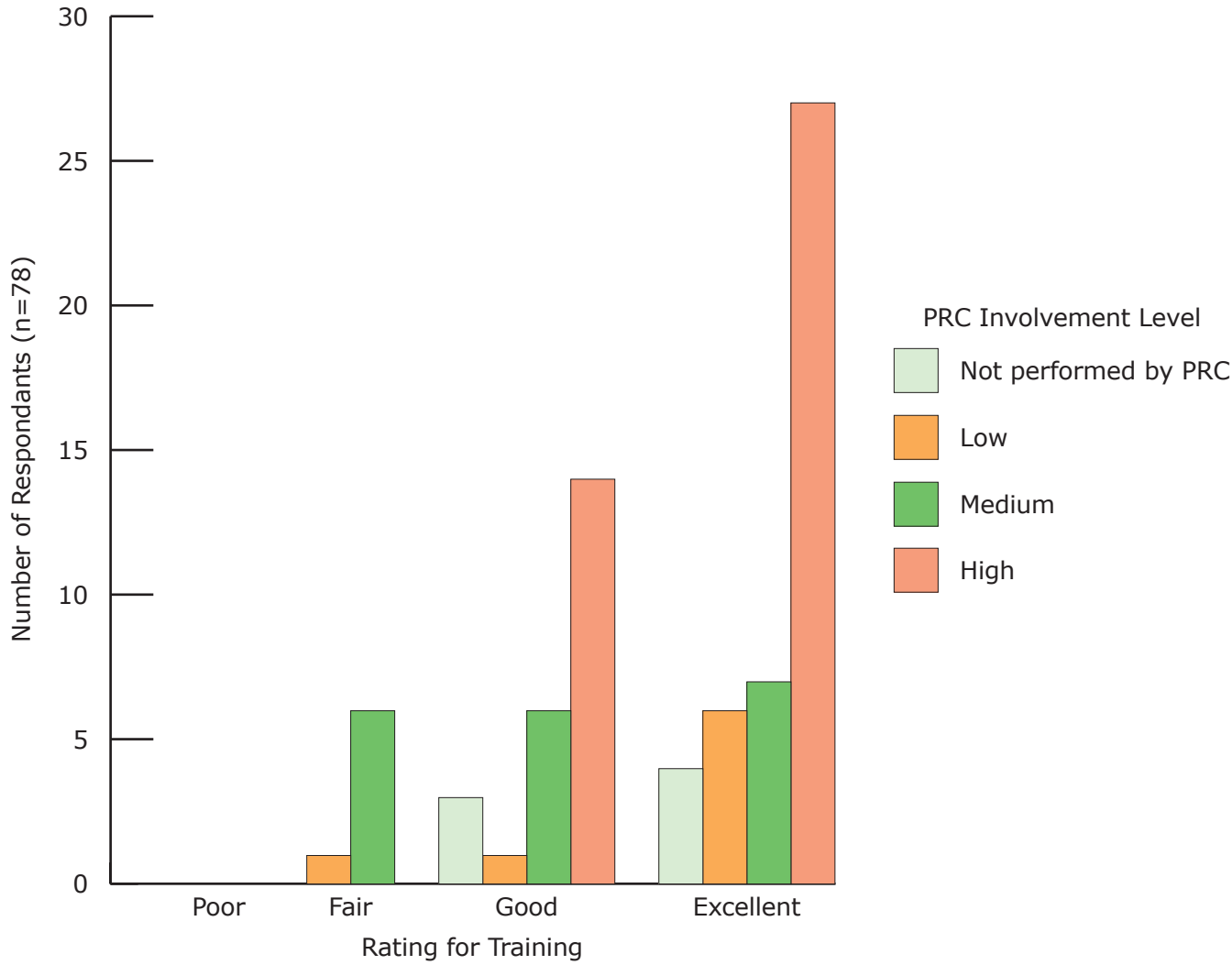
Methods and Materials

At the PEARL Network 2011 Annual Meeting held in New York, an evaluation form was completed by 130 network respondents (P–Is and PRCs who attended the meeting). The form was developed by PEARL Network staff and was not tested prior to being administered. Of the network respondents, there were 74 P-Is and 56 PRCs. The evaluation form asked questions about how involved PRCs are in coordinating study activities, how many PRCs each site utilizes and the satisfaction with training and support. The evaluation form also measured how many clinical studies the respondent participated in. The PEARL Network Program Evaluation was not classified as human subject research, because it was not a systematic investigation and no identifying personal information was collected. Results from the paper evaluation form were entered into an excel database, and analyzed using the statistical analysis software SPSS. The bivariate correlation test, Pearson Correlation, was conducted, and results were considered significant if $p<0.05$.

Results

Eighty four participants completed the evaluation and returned it to the Network staff. The evaluation form did not differentiate between P–Is and PRCs, or ask respondents their role in the dental team. The network respondents answered positively to the overall evaluation of how satisfied they are with the training and support they have received from

Figure 1: PEARL Network PRC Involvement and Evaluation of Training



the PEARL Network throughout their participation, and they reported positive levels of satisfaction with using PEARL's electronic data capture system, Advantage EDCSM.

A statistically significant correlation (p=0.004) was found between the number of PRCs at a site and the number of studies in which the site participated. The level of involvement of the PRCs in coordinating study activities was rated: 0=not applicable (not performed by a PRC), 1=low, 2=medium, 3=high and the number of studies participated in captured. Ratings for PRC satisfaction with training and Advantage EDCSM were: 1=poor, 2=fair, 3=good and 4=excellent. In addition, there was a correlation between satisfaction with the training/support and the involvement of PRCs in organizing study activities (p=0.008), and a correlation between satisfaction with training/support and the number of PRCs utilized by the office (p=0.039). From our interaction with the PRCs during monthly PRC calls, the CRA team expected to see a relationship between the number of PRCs and overall satisfaction. Feedback from CRAs supports that when the practice has motivated people to help conduct research it is easier for the office to participate. PRCs have also reported a sense of pride with certification, and a sense of accomplishment when the CITI tutorial is completed. Another reported benefit of participation is that providers feel more connected to the results, and report that they have a greater sense of buy in because the results are generated in their practice.

Figure 1 shows the frequency number of respondents who rated the PRC involvement (not performed by PRC, low, medium or high) by the rating for the training (poor, fair, good or excellent). There was a statistically significant positive correlation between the rating of training and the level of PRC involvement (p=0.008). This was another relationship that was identified by the CRAs during the PRC teleconference calls. The CRA team observed that offices with increased PRC personnel participated

more actively in the network. PEARL has a limited number of member dentists who have taken on the role of the research team. The CRA team has observed the offices ability to participate and enroll patients in multiple studies is dependent upon the participation and interest of the PRCs.

Discussion

To date, the studies in the PEARL Network have addressed issues that improve the evidence basis for patient care, such as providing real world outcomes data for dentin caries activity,[16] root canal therapy at 3 to 5 years post treatment,[17–21] reporting the risk factors for osteonecrosis of the jaw[22] and presenting data about analgesic use effectiveness.[8–10] In addition, the studies strive to improve patient centered care, by providing dentistry with a better understanding of the oral health impact of dental disease and treatment procedures on the patient's quality of life of patients.[23] All patients who enroll in a study complete the Oral Health Impact Profile.[24] Currently, the Network is preparing multiple publications related to the studies described above.

The results indicate that participation in the PEARL Network and the satisfaction with network training/support correlates with the number of PRCs at a dental PBRN site. This would suggest that the PBRN has a positive effect on dental practices with support staff, and that the ADA model for optimal efficiency in a dental practice corresponds to that of a dental practice based research site. We posit that additional PRCs provide a support mechanism at dental practices engaged in clinical research, and they have the ability of learning from one another, thereby supplementing the training provided by PEARL CRAs. More PRCs may reduce the burden of participation, possibly by distributing the workload between multiple individuals. In addition, provider satisfaction with communication and dissemination efforts should be evaluated, and the satisfaction during professional development can be made possible and facilitated through the Network. As the network grows, both national and regional differences in responses to the network evaluation should be analyzed to enhance dental care, facilitate quality, cost effectiveness and the ultimate goal of improving health and well–being.

Conclusion

Just as dental hygienists are key members of a dental practice, PRCs are key members of the PEARL Network PBRN clinical research team. The evaluation demonstrated the positive relationship between PRC involvement when conducting clinical studies in our dental PBRN. We found a correlation between the number of PRCs at a site and the number of studies in which a site participates. Further, the number of PRCs involved in organizing research activities at the site was found to be related to satisfaction with the training and support systems implemented by the PEARL administrative and clinical operative team. Future evaluations will look at additional information about how different types of providers (P–Is and PRCs) differ in their response to the evaluation forms.

Ashley Grill, RDH, BSDH, MPH, is a clinical assistant professor of dental hygiene at the New York City College of Technology. Joanne M. Johnson, RDH, is an associate in evidence based dentistry at the DentaQuest Oral Health Center. Damon Collie, BS, MSHS, is the project manager of the Data Coordinating Center for the PEARL Network at the EMMES Cooperation. Van P. Thompson, DDS, PhD, is the PEARL Network Director of Protocol Development and Training, Professor; Chairperson Biomaterials, King's College London Dental Institute. Ronald G. Craig, DMD, PhD, is the PEARL Network Director of Information Dissemination, Associate Professor; Basic Sciences and Craniofacial Biology, Department of Periodontology and Implant Dentistry, New York University College of Dentistry. Frederick A. Curro, DMD, PhD, is the PEARL Network Director of Recruitment, Retention and Operations, Director of Regulatory Affairs, Bluestone Center for Clinical Research, New York University College of Dentistry.

References

1. Curro FA, Thompson VP, Grill A, Vena D, Terracio L, Naftolin F. Practice Based Research Networks Impacting Periodontal Care. J *Periodontol*. 2012. [Epub ahead of print].

2. Bertolami C. Practice Based Research Networks. P–I breakout session. PEARL Network Annual Meeting. NYU College of Dentistry, 2011 March. Palisades, NY.

3. Large Hadron Collider. CERN [Internet]. 2008 [cited 2011 September 9]. Available from: http://user.web.cern.ch/public/en/LHC/LHC-en.html

4. Grill AC, Alai S, Bray KK, Collie DM, Branson BG, Thompson VP, Curro FA. Assessing PEARL PBRN clinical study results with public use data sets and common data elements for comparative effectiveness research. *J Am Dent Assoc*. In press.

5. Craig RG, Curro FA, Thompson VP. Dr. Jeannette Abboud–Niemczyk: Going the extra mile for patients. PERAL Network [Internet]. 2010. Available from: http://www.pearlnetwork.org/newsletters/winter2011.pdf

6. Guideline for good clinical practice. International Conference on Harmonisation [Internet]. 1996 [cited 2010 October 26]. Available from: http://www.ich.org/fileadmin/Public_Web_Site/ICH_Products/Guidelines/Efficacy/E6_R1/Step4/E6_R1__Guideline.pdf

7. Curro FA, Grill AC, Thompson VP, et al. Advantages of the Dental Practice–Based Research Network Initiative and Its Role in Dental Education. *J Dent Educ*. 2011;75(8):1053–1060.

8. Wong YJ, Craig RG, Thompson VP, Wu J, Vena D, Curro FA. Analgesic use and patient–reported effectiveness: PEARL interim results. IADR [Internet]. 2009 [cited 2011 September 9]. Available from: http://iadr.confex.com/iadr/2009miami/webprogram/Paper120637.html

9. Keenan J, Wong Y, Craig R, Thompson V, Wu J, Vena D. Practice–based research assessment of analgesic effectiveness: PEARL Interim findings. IADR [Internet]. 2010 [cited 2011 September 9]. Available from: http://iadr.confex.com/iadr/2010barce/webprogramcd/Paper139572.html

10. Hudson K, Bryan H, Thompson V, Vena D, Craig R, Wu J, Collie D, Curro F. Analgesics prescribed/recommended for coded dental procedures: PEARL practitioner profiles. IADR [Internet]. 2011 [cited 2011 September 9]. Available from: http://iadr.confex.com/iadr/2011sandiego/webprogram/Paper149964.html

11. Keckley PH, Frink BB. Comparative Effectiveness: A Strategic Perspective on What It Is and What It May Mean for the United States. *J Health Life Sci Law*. 2009;3(1):53–90.

12. Quinn M. eviDent shares experiences with US dental researchers. ADAVB Newsletter [Internet]. 2011 June. Available from: http://www.evident.net.au/LinkClick.aspx?fileticket=Ycupijp85rw%3D&tabid=166&mid=734&language=en-US

13. NIDCR Awards Grants for New Practice-Based Initiative. NIDCR [Internet]. 2005 March 31. Available from: http://www.nidcr.nih.gov/Research/ResearchResults/NewsReleases/ArchivedNewsReleases/NRY2005/PR03312005.htm

14. DeNucci, D. The Future: NIDCR Sponsored Practice–Based Research Networks. P–I breakout session. PEARL Network Annual Meeting. 2011 March. NYU College of Dentistry, Palisades, NY.

15. National Dental Practice–Based Research Network Limited Competition (U19). NIH [Internet]. 2010 November [cited 2011 September 9]. Available from: http://grants.nih.gov/grants/guide/rfa-files/RFA-DE-12-002.html

16. Lehmann M, Veitz-Keenan A, Matthews AG, et al. Dentin caries activity in early occlusal lesions selected to receive operative treatment: findings from the Practitioners Engaged in Applied Research and Learning (PEARL) Network. *J Am Dent Assoc*. 2012;143(4):377-385.

17. Bernstein S, Horowitz A, Craig RG, et al. Endodontic treatment and restoration outcomes in general practice. IADR [Internet]. 2008 [cited 2011 September 9]. Available from: http://iadr.confex.com/iadr/2008Toronto/techprogram/abstract_107982.htm

18. Man M, Pliszczak J, Craig RG, et al. General practice endodontic treatment and restorative findings. IADR [Internet]. 2009 [cited 2011 September 9]. Available from: http://iadr.confex.com/iadr/2009miami/webprogram/Paper118285.html

19. Spielman H, Schaffer S, Craig R, Thompson V, Vena D. Single tooth endodontic and restorative treatment outcomes: PEARL interim findings. IADR [Internet]. 2010 [cited 2011 September 9]. Available from: http://iadr.confex.com/iadr/2010barce/webprogramschedule/Paper139466.html

20. Horowitz A, Barna J, Craig R, et al. Outcomes of endodontic therapy in general practice: PEARL Network findings. IADR [Internet]. 2011 [cited 2011 September 9]. Available from: http://iadr.confex.com/iadr/2011sandiego/webprogram/Paper150047.html

21. Bernstein SD, Horowitz AJ, Man M, et al. Outcomes of endodontic therapy in general practice: a study by the Practitioners Engaged in Applied Research and Learning Network. *J Am Dent Assoc*. 2012;143(5):478-487.

22. Barasch A, Cunha-Cruz J, Curro FA, et al. Risk factors for osteonecrosis of the jaws: a case-control study from the CONDOR dental PBRN. *J Dent Res*. 2011;90(2):439–444.

23. Matthews AG, Botello–Harbaum M, Vena D, et al. Hypersensitivity and oral health–related quality of life: PEARL Network findings. IADR [Internet]. 2011 [cited 2011 September 9]. Available from: http://iadr.confex.com/iadr/2011sandiego/webprogram/Paper150110.html

24. Slade GD. Derivation and validation of a short–form oral health impact profile. *Community Dent Oral Epidemiol*. 1997;25(4):284–290.

Preliminary Findings on the Correlation of Saliva pH, Buffering Capacity, Flow, Consistency and Streptococcus mutans in Relation to Cigarette Smoking

Marsha A. Voelker, CDA, RDH, MS; Melanie Simmer–Beck, RDH, MS; Molly Cole, RDH, BS; Erin Keeven, RDH, BS; Daniel Tira, PhD

Abstract

Purpose: The purpose of this preliminary study was to examine the relationship of caries risk, salivary buffering capacity, salivary pH, salivary quality (flow, consistency) and levels of Streptococcus mutans in relation to cigarette smoking.

Methods: This clinical trial consisted of 53 volunteer patients receiving care in a university based dental hygiene clinic. Participants completed a questionnaire specific to their social history in regards to tobacco use, oral health and dietary history. Measurements of unstimulated saliva were collected followed by collection of stimulated saliva samples. These samples were used to measure salivary pH, buffering capacity and Streptococcus mutans levels.

Results: The subject's smoking status was significantly associated with caries risk (p= 0.001), with 25% of the variability of caries risk attributed to smoking. The smoking status was significantly associated with buffering capacity (p=0.025), with 9% of the variability of buffering status attributed to the smoking. Associations between smoking status and salivary pH were not statistically significant. The subject's caries risk was significantly associated with buffering capacity (p= 0.001), with 25% of the variability of caries risk attributed to the buffering capacity. The subject's caries risk was significantly associated with salivary pH (p= 0.031), with 9% of the variability of caries risk attributed to the salivary pH. The Streptococcus mutans test showed no statistical significance (p>0.05) possibly due to the number and low variance in the subjects.

Conclusion: A relationship between caries risk and smoking, buffering capacity and smoking, and stimulated salivary pH and smoking were concluded. No significance difference (p>0.05) between caries risk and salivary pH, salivary quality and smoking, S. mutans and smoking were noted from the preliminary results.

Keywords: saliva testing, caries risk, pH, S. mutans, buffering capacity

This study supports the NDHRA priority area, **Clinical Dental Hygiene Care:** Investigate how dental hygienists identify patients who are at–risk for oral disease.

Introduction

Dental caries, a transmissible infectious disease of microbial origin, is mediated by modifiable risk factors. As a result, caries risk assessment is becoming the standard of care. In April of 2002, a consensus conference was held on the topic of Caries Management by Risk Assessment (CAMBRA).[1] During the conference, an expert panel created a caries risk assessment tool based upon current literature regarding caries risk factors (disease indicators–bacterial tests, risk factors, protective factors–oral health regimen, supplements and saliva quantity and ability to buffer).[1] This tool evaluates 9 risk factors (biological predisposing factors):[2,3]

- Medium or high Streptococcus mutans (S. mutans) and Lactobacilli counts
- Visible heavy plaque biofilm on teeth
- Frequent snacking between meals
- Deep pits and fissures
- Recreational drug use
- Inadequate salivary flow by observation or measurement
- Saliva–reducing factors
- Exposed roots
- Orthodontic appliances

The tool also assesses saliva in terms of pH (stimulated and unstimulated), consistency and buffering capacity as risk factors for dental caries. The caries risk tool ultimately assists dental professionals in determining low, moderate, high or extreme high caries risk. One factor not recognized as a risk factor for caries, in the CAMBRA model, is cigarette smoking.

There is reasonable evidence that cigarette smoking increases individual's risk for developing caries,[4–11] leading some oral health providers to modify

the CAMBRA tool to include cigarette smoking as a risk factor.[12] Several studies examined the relationship between early childhood caries and parental smoking and concluded there is an association between environmental tobacco smoke and risk of caries among children and adolescents.[3,9,13,14] Studies in young adults revealed an association between cigarette smoking and tooth loss resulting from dental caries and plaque scores, and decayed, missing, filled teeth (DMFT) scores were significantly higher in smokers than non–smokers.[5,9] Bartoloni examined dental caries in Air Force personnel and reported tobacco use had an elevated risk of developing caries.[15] Iida used 1999 to 2004 data from the National Health and Nutrition Examination to examine the oral health status of U.S. women of childbearing age and concluded current smoking was a strong independent risk factor for untreated caries, periodontitis and, to a lesser extent, decayed, missing, filled surfaces (DMFS), and the odds of having poor oral health among previous smokers was slightly higher than in women who had never smoked.[7] Lastly, Aguilar–Zinser examined the relationship of smoking of professional truck drivers and reported that, as the number of cigarettes increased, so did the number of large caries.[16] These findings were statistically significant.[4] Collectively, the evidence suggests smoking is a possible risk factor for caries.

Most of the aforementioned studies examined tobacco use in a narrow group of subjects already at moderate to high risk for developing caries, such as the elderly,[8] the U.S. Air Force,[15] professional truck drivers in Mexico[16] and women of childbearing age.[7] Several of the authors used tooth loss, decayed, missing and filled (DMF), DMFS and/or DMFT as the dependent variable,[4–9] which is problematic because the point in time when tooth loss or decay occurred cannot be established. Additionally, the severity of periodontal disease was not documented. Therefore, one cannot assume a casual association between smoking and tooth loss, DMF, DMFS and/or DMFT. Prior studies were conducted primarily outside of the U.S. and do not adequately control for external variables that could influence the development of caries.[4,6,17] Only 2 recent studies were conducted in the U.S.[7,15] Fluoride status was not documented in any of the studies.[4–11,15] Additionally, several authors concluded that caries risk status was influenced by co–founding factors such as the socioeconomic status of the subjects,[9,15] poor oral hygiene in smokers,[5,9,15] younger subjects placing less value on general health,[15] mal–distribution of smokers[4,6] and having Medicaid or no insurance.[7] The pH of saliva has been cited as a likely variable affecting caries risk, reporting an increase of pH while smoking and decrease after smoking.[4,17,18] Over long periods of time, smokers have a lower pH in stimulated saliva.[19] Buffering capacity was also found to be lower in smokers.[20,21]

The literature is lacking studies in the U.S. that examine smoking in relation to caries using biologic dependent variables while controlling for co–founding factors. The purpose of this preliminary study was to examine the relationship of caries risk, salivary buffering capacity, salivary pH, salivary quality (flow rate and consistency) and Streptococcus mutans (bacteria associated with dental caries) in relation to cigarette smoking in a sample of adults that had limited co–founding factors.

Methods and Materials

This study was approved by the University of Missouri–Kansas City (UMKC) Adult Health Science institutional review board. This cross–sectional clinical trial used a convenience sample of 53 patients of record seeking dental hygiene care at UMKC School of Dentistry. All 53 subjects voluntarily chose to participate in the study. A total of 77% of subjects were female and 23% were male. The sample was intentionally homogenous to minimize the effects of co–founding variables. None of the subjects were taking medications, had a systemic disease or had undergone radiation treatments that would alter their salivary function. Demographics of the sample are illustrated in Table I. Smoking status in relation to age and insurance status is described in Table II. Smoking status in relation to plaque index and caries protective factors is described in Table III.

Data regarding each subject's medical history and dental history was assembled from the electronic patient record. Additional data was collected through a written questionnaire focusing on smoking status, the Oral Health Related Quality of Life questionnaire[22] and CAMBRA tool. Smoking status was classified as: current smokers, previous smokers, nonsmokers and second hand smoke exposure. The presence of caries was determined during the dental exam by 1 dentist throughout the study. Data was collected by calibrated dental hygiene student clinicians during scheduled clinic sessions as part of the dental hygiene process of care. Saliva quality was determined by examining salivary flow and consistency. Figure 1 outlines the saliva collection procedures. The Saliva–Check Buffer system (GC America, Inc., Alsip, Il) was used to measure stimulated and unstimulated saliva. The Saliva–Check Buffer system was packaged with the following: pH paper strip, measuring cup, dropper, wax and buffer test strip. Saliva–Check Mutans (GC America, Inc., Alsip, Il) was used to measure the presence of S. mutans. The Saliva–Check Mutans system was packaged with the following: wax, dropper, mixing container, reagent 1 and 2, and mutans

Table I: Demographics (Ethnicity, Age, Insurance Status and Smoking Status)

Ethnicity	Percentage	Average Age	Health Insurance	Dental Insurance	Current Smoker	Past Smoker	Second–Hand Smoke	Non–Smoker
Caucasian	89%	39	70%	53%	11.3%	3.8%	1.9%	74%
African American	3.8%	48	0%	0%	0%	0%	0%	3.8%
Hispanic	1.9%	57	0%	1.9%	0%	0%	0%	1.9%
Asian	1.9%	22	1.9%	1.9%	0%	0%	0%	1.9%
American Indian	1.9%	21	1.9%	0%	0%	0%	0%	1.9%
Hawaiian	1.9%	28	0%	0%	0%	0%	0%	1.9%
Overall	100%	36	73.8	56.8%	11.3%	3.8%	1.9%	83%

Table II: Smoking Status in Relation to Age and Insurance

Smoking Status	Average Age	Private Health Insurance*	Medicaid	Medicare	None	Private Dental Insurance*	Medicaid Dental	None
Current Smoker	37	4%	0%	0%	0%	4%	0%	0%
Past Smoker	36	6%	0%	0%	2%	4%	0%	2%
Second–Hand Smoker	49	15%	2%	0%	0%	8%	0%	11%
Non–Smoker	36	49%	0%	0%	13%	38%	0%	26%

Table III: Smoking Status in Relationship to Average PI and Daily Fluoride Exposure and Daily Xylitol Exposure

Smoking Status	Avgerage PI	Fluoride Exposure Toothpaste	Fluoride Exposure Mouthrinse	Lives in Fluoridated Community	Xylitol x4 daily either mints or gum
Current Smoker	13%	4%	2%	2%	0%
Past Smoker	25%	11%	2%	9%	4%
Second–Hand Smoker	37%	19%	11%	19%	0%
Non–Smoker	21%	64%	32%	62%	4%

test device. Accuracy of the Saliva–Check Buffer system was established by using a calibrated electronic pH meter. Both measurements of pH were comparable, therefore validating the Saliva–Check Buffer system.

Data was entered into an Excel Spreadsheet and converted to SPSS. Data was analyzed by using the Spearman RHO correlation coefficient. The independent variable was the smoking status. The present study used buffering capacity, salivary pH, flow rate, consistency and Streptococcus mutans as the dependent variables, which provides an accurate picture of the relationship between smoking and caries risk.

Results

Table IV outlines the buffering capacity, salivary pH and salivary quality in terms of individual smoking statuses. Regardless of a subjects smoking status, the pH of stimulated and uanstimulated saliva remained within the healthy range of 6.8 to 7.8.

Figure 1: Saliva Testing Steps

Data revealed nearly all of the non–smokers had normal or high resting flow rates. Data revealed the saliva consistency and saliva quantity was very similar between smoking statuses. There was a variance in buffering capacity depending on the subject's smoking status with smokers having between very low to low status where the non–smokers were between low and normal status.

Table V describes the relationship of smoking status with caries risk, buffering capacity and stimulated saliva pH. Data revealed that the subject's smoking status was significantly associated with caries risk (p= 0.001), with 25% of the variability of caries risk attributed to smoking. The smoking status was significantly associated with buffering capacity (p=0.025), with 9% of the variability of buffering status attributed to the smoking. The smoking status and salivary pH were not statistically significant (p>0.05).

Table VI describes the relationship of caries risk with buffering capacity and salivary pH. Data revealed the subject's caries risk was significantly associated with the buffering capacity (p= 0.001), with 25% of the variability of caries risk attributed to the buffering capacity. The subject's caries risk was significantly associated with salivary pH (p= 0.031), with 9% of the variability of caries risk attributed to the salivary pH. The Streptococcus mutans test showed no statistical significance (p>0.05).

Discussion

This appears to be the first study that examines the relationship between biological variables and smoking. The means by which tobacco modifies the

Table IV: Percentage of salivary quality, buffering capacity and average salivary pH per smoking status

Smoking Status	Resting Flow Rate (%)			Saliva Consistency (%)			Salivary pH
	Low	Normal	High	Residues	Increased Viscosity	Normal Viscosity	Un–stimulated
Current Smoker	7.5	0	3.8	0	5.7	5.7	6.8
Past Smoker	0	3.8	0	0	1.9	1.9	6.8
Second–Hand	0	1.9	0	0	1.9	0	7.6
Non–Smoker	7.5	45.2	26.4	3.8	42	36	7.0
Overall	15	50.9	30.2	3.8	51.2	43.6	7.1
Smoking Status	Saliva Quantity (%)			Buffering Capacity (%)			Salivary pH
	Low	Normal	High	Residues	Increased Viscosity	Normal Viscosity	Stimulated
Current Smoker	0	3.8	7.5	1.9	9.4	0	7.5
Past–Smoker	0	0	3.8	0	1.9	1.9	7.7
Second–Hand	0	1.9	0	0	1.9	0	7.4
Non–Smoker	1.9	5.7	74	3.8	38	40	7.6
Overall	1.9	11.4	85.3	5.7	51.2	41.9	7.6

Table V: Smoking Status in Relationship with Caries Risk, Buffering Capacity and Stimulated Salivary pH

Relationship	Spearman Rank Order (RHO)
Caries Risk	p=0.001*
Buffering Capacity	p=0.025*
Salivary pH	p=0.065

*=statistically significant

Table VI: Caries Risk in Relationship with Buffering Capacity and Salivary pH

Relationship	Spearman Rank Order (RHO)
Buffering Capacity	p=0.001*
Salivary pH	p=0.031*

*=statistically significant

caries process and its relationship with availability of saliva in the mouth is still unclear.[4,23] Some studies have suggested tobacco leads to transient decline in the availability of saliva in the mouth,[4,24] while other studies show that salivary flow actually increases during tobacco use.[4,17,25] Saliva pH changes have been cited as variables for modifying caries risk.[4] Reports suggest that pH transiently increases while smoking and decreases after smoking, but in some cases it stays at lower levels.[4,17] Liede et al indicated that tobacco smokers implicated in dental/oral conditions, such as increased Lactobacilli[4,26,27] or Candida albicans and Streptococcus mutans,[4,23,25] demonstrated reduced buffering capacity.[4] The preliminary results from the present study revealed a relationship between caries risk and smoking as well as caries risk and buffering capacity.

The validity and reliability of caries assessment tools evaluating pH and buffering capacity of saliva has been well established.[28,29] The validity and reliability of chairside Saliva–Check mutans test has not been well established. This study agreed with previous studies regarding the validity and reliability of the Saliva–Check buffer pH readings by comparing with an electronic pH meter. Omori examined the detectability and operability of chairside bacteria testing kits and reported difficulties in accuracy regarding order of measure of the accurate number of bacteria and S. mutans.[30] The present study revealed Saliva–Check Mutans system not being statistically significant (p>0.05). In fact, all data looked exactly the same (all negative results or the device did not indicate negative or positive for increase number of S. mutans).[4] Investigators question whether or not the test was functioning properly. Further testing to determine the validity and reliability of this chairside test should be conducted by using a standard microbial lab test.

The present study accounted for the protective factors (fluoride and xylitol) and contributing factors

(medications, radiation therapy and systemic disease) where other studies have not included this in the data collection.[4,6,8,9,15] Fluoride exposure included toothpaste, mouthwash or living in a fluoridated community. The majority of the subjects exposed to fluoride were the non–smokers, and 4% of the subjects who used xylitol at least 4 times daily were past smokers and non–smokers. Non–smokers had a higher percentage of fluoride exposure compared to current and past smokers, either via toothpaste, mouthwash or living in a fluoridated community.

Regarding contributing factors, none of the subjects in the present study reported taking medications, systemic disease or undergoing radiation therapy that would alter salivary function; therefore, the results from the study were not affected by predisposed xerostomia. This could have impacted the results of the study if subjects did have these contributing factors that resulted in xerostomia due to caries risk for the subjects would be extremely high.[2,31–33] A low percentage of non–smoking subjects in the present study reported experiencing dry mouth. None of the subjects who smoked reported experiencing dry mouth. Other components to consider as risk factors for caries include diet, poor oral hygiene care, genetics and socioeconomic status.[15,32,34]

The present study included plaque index data that revealed smokers had a lower plaque index percentage than non–smokers. This leads the investigators to believe that caries risk was not influenced by homecare. Bartoloni et al suggests caries risk status is probably influenced by the socioeconomic status.[15] Graves and Stamm stated that socioeconomic status had a strong influence on the tendency of populations to seek care, with socioeconomic status inversely related to caries experience.[15,35] The present study reported a lower percentage of current and past smokers had dental insurance which may be a factor whether a patient who smokes seeks dental care and the impact of the subjects overall oral condition (Table I, II). Future studies with a larger sample size should account for protective and contributing factors as well as oral hygiene regimens.

No significance ($p>0.05$) between caries risk and the salivary pH were noted from the preliminary results, which we expected due to the small sample size and large number of non–smokers. There have been studies regarding tobacco effects on caries risk, but the data collected from these studies have not utilized salivary pH as part of the assessment tools for determining caries risk.[15] This maybe the first study that has collected data regarding salivary pH in relation to smoking and caries risk and if this study had a greater population of smokers the salivary pH may have been of statistical significance.

Determining the subjects smoking status through self–report was a limitation of this study. The investigators have no way of knowing whether or not subjects provided an accurate reflection of their daily habits. Continine is a major metabolite of nicotine and often used to measure the extent of tobacco use and the exposure to the environmental tobacco smoke. Utilization of the detection limit of 0.05 ng/mL in serum continine would have assisted in determining the status between exposure and non–exposure to tobacco smoke.[7,36] Future studies should measure serum cotinine to assess smoking status. Another limitation to the study was using a convenience sampling where the majority of the subjects were Caucasian non–smokers. Further studies need to be conducted to encompass a larger subject pool that control for diverse subject population that smoke.

Dental caries is a complex, dynamic, multifactorial process and many factors (disease, risk, protective, contributing) are to be considered when determining a patient's risk factor for caries.[2,31–33] There should be strong consideration to include smoking as one of the factors when conducting a CAMBRA due to the evidence presented within various studies which indicates smoking has an effect on the oral cavity.[6–9,15]

Conclusion

Within the limitations of the preliminary study on the relationship of buffering capacity, salivary pH, salivary quality and S. mutans in relation to cigarette smoking, the following can be concluded:

- A relationship exists between caries risk and smoking.
- A relationship exists between buffering capacity and smoking.
- A relationship exists between stimulated salivary pH and smoking.
- There is not a relationship between salivary quality and smoking.
- There is not a relationship between smoking status and S. mutans.

Marsha A. Voelker, CDA, RDH, MS, is an assistant professor at the Division of Dental Hygiene University of Missouri–Kansas City School of Dentistry. Melanie Simmer–Beck, RDH, MS, is an associate professor at the Division of Dental Hygiene University of Missouri–Kansas City School of Dentistry. Molly E. Cole, RDH, BS, is a graduate of the Division of Dental Hygiene University of Missouri–Kansas City School of Dentistry. Erin Keeven, RDH, BS, is a graduate of the Division of Dental Hygiene University of Missouri–Kansas City School of Dentistry. Daniel Tira, PhD, is a retired professor from the University of Missouri–Kansas City School of Dentistry.

References

1. Doméjean-Orliaguet S, Gansky SA, Featherstone JD. Caries risk assessment in an educational environment. *J Dent Educ*. 2006;70(12):1346–1354.

2. Featherstone JD. The science and practice of caries prevention. *J Am Dent Assoc*. 2000;131(7):887–899.

3. Aligne CA, Moss ME, Auinger P, Weitzman M. Association of pediatric dental caries with passive smoking. *JAMA*. 2003;289(10):1258–1264.

4. Aguilar-Zinser V, Irigoyen ME, Rivera G, Maupomé G, Sánchez-Pérez L, Velázquez C. Cigarette smoking and dental caries among professional truck drivers in Mexico. *Caries Res*. 2008;42(4):255–262.

5. Al–Habashneh R, Al–Omari MA, Taani DQ. Smoking and caries experience in subjects with various form of periodontal diseases from a teaching hospital clinic. *Int J Dent Hyg*. 2009;7(1):55–61.

6. Axelsson P, Paulander J, Lindhe J. Relationship between smoking and dental status in 35–, 50–, 65–, and 75–year–old individuals. *J Clin Periodontol*. 1998;25(4):297–305.

7. Iida H, Kumar JV, Kopycka–Kedzierawski DT, Billings RJ. Effect of tobacco smoke on the oral health of U.S. women of childbearing age. *J Public Health Dent*. 2009l;69(4):231–241.

8. Jette AM, Feldman HA, Tennstedt SL. Tobacco use: a modifiable risk factor for dental disease among the elderly. *Am J Public Health*. 1993;83(9):1271–1276.

9. Ojima M, Hanioka T, Tanaka K, Aoyama H. Cigarette smoking and tooth loss experience among young adults: a national record linkage study. *BMC Public Health*. 2007;7:313.

10. Tada A, Hanada N. Sexual differences in smoking behaviour and dental caries experience in young adults. *Public Health*. 2002;116(6):341–346.

11. Zitterbart PA, Matranga LF, Christen AG, Park KK, Potter RH. Association between cigarette smoking and the prevalence of dental caries in adult males. *Gen Dent*. 1990;38(6):426–431.

12. Roberts DR, Maragliano P, Chapman R. Put the plan into action: how to implement a successful and efficient caries prevention program. Dimensions. 2011;9(6):70–73.

13. Ayo–Yusuf OA, Reddy PS, van Wyk PJ, van den Borne BW. Household smoking as a risk indicator for caries in adolescents' permanent teeth. *J Adolesc Health*. 2007;41(3):309–311.

14. Shenkin JD, Broffitt B, Levy SM, Warren JJ. The association between environmental tobacco smoke and primary tooth caries. *J Public Health Dent*. 2004;64(3):184–186.

15. Bartoloni JA, Chao SY, Martin GC, Caron GA. Dental caries risk in the U.S. air force. *J Am Dent Assoc*. 2006;137(11):1582–1591.

16. Kreth J, Zhu L, Merritt J, Shi W, Qi F. Role of sucrose in the fitness of streptococcus mutans. *Oral Microbiol Immunol*. 2008;23(3):213–219.

17. Bayraktar G, Kazancioglu R, Bozfakioglu S, Ecder T, Yildiz A, Ark E. Stimulated salivary flow rate in chronic hemodialysis patients. *Nephron*. 2002;91(2):210–214.

18. Kenney EB, Saxe SR, Bowles RD. The effect of cigarette smoking on anaerobiosis in the oral cavity. *J Periodontol*. 1975;46(2):82–85.

19. Parvinen T. Stimulated salivary flow rate, pH and lactobacillus and yeast concentrations in non–smokers and smokers. *Scand J Dent Res*. 1984;92(4):315–318.

20. Heintze U. Secretion rate, buffer effect and number of lactobacilli and streptococcus mutans of whole saliva of cigarette smokers and nonsmokers. *Scand J Dent Res*. 1984;92(4):294–301.

21. Johnson NW, Bain CA. Tobacco and oral disease. EU–working group on tobacco and oral health. *Br Dent J*. 2000;189(4):200–206.

22. Gadbury–Amyot CC, Williams KB, Krust–Bray K, Manne D, Collins P. Validity and reliability of the oral health–related quality of life instrument for dental hygiene. *J Dent Hyg*. 1999;73(3):126–134.

23. Fenoll-Palomares C, Muñoz Montagud JV, Sanchiz V, et al. Unstimulated salivary flow rate, pH and buffer capacity of saliva in healthy volunteers. *Rev Esp Enferm Dig*. 2004;96(11):773–783.

24. Edgerton M, Koshlukova SE, Araujo MW, Patel RC, Dong J, Bruenn JA. Salivary histatin 5 and human neutrophil defensin 1 kill Candida albicans via shared pathways. *Antimicrob Agents Chemother*. 2000;44(12):3310–3316.

25. Van Nieuw Amerongen A, Bolscher JG, Veerman EC. Salivary proteins: protective and diagnostic value in cariology? *Caries Res*. 2004;38(3):247–253.

26. Liede KE, Haukka JK, Hietanen JH, Mattila MH, Rönkä H, Sorsa T. The association between smoking cessation and periodontal status and salivary proteinase levels. *J Periodontol*. 1999;70(11):1361–1368.

27. Anttila SS, Knuuttila ML, Sakki TK. Depressive symptoms favor abundant growth of salivary lactobacilli. *Psyschosom Med*. 1999;61(4):508–512.

28. Ericson D, Bratthall D. Simplified method to estimate salivary buffer capacity. *Scand J Dent Res*. 1989;97(5):405–407.

29. Rothen M, Cunha-Cruz J, Mancl L, et al. Inter–examiner reliability of salivary diagnostic tests in a practice–based research network. *J Dent Hyg*. 2011;85(2):143–150.

30. Ohmori K, Harda C, Takao A, Momoi Y. Comparison of 4 salivary test kits in detecting mutans streptococci. *Ha no Ushoku Chiryo deno Saikingakuteki Apurochi to Saisei Chiryo Heisei*. 2006:107–113.

31. Featherstone JD. The continuum of dental caries––evidence for a dynamic disease process. *J Dent Res*. 2004;83(Spec No C):C39–C42.

32. Featherstone JD, Domejean–Orliaguet S, Jenson L, Wolff M, Young DA. Caries risk assessment in practice for age 6 through adult. *J Calif Dent Assoc*. 2007;35(10):703–707, 710–713.

33. Featherstone JD. Remineralization, the natural caries repair process––the need for new approaches. *Adv Dent Res*. 2009;21(1):4–7.

34. Young DA, Featherstone JD. Implementing caries risk assessment and clinical interventions. *Dent Clin North Am*. 2010;54(3):495–505.

35. Graves RC, Stamm JW. Oral health status in the united states: prevalence of dental caries. *J Dent Educ*. 1985;49(6):341–353.

36. National Center for Health Statistics Centers for Disease Control and Prevention. NHANES 2001–2002 data release. Documentation for laboratory results. CDC [Internet]. 2007 May [cited 2011 December 14]. Available from: http://www.cdc.gov/nchs/data/nhanes/nhanes_01_02/l06_b_doc.pdf

How Do Diet and Body Mass Index Impact Dental Caries in Hispanic Elementary School Children?

Mary Creske, DrPH; Naomi Modeste, DrPH; Joyce Hopp, PhD; Sujatha Rajaram, PhD; David Cort, PhD

Abstract

Purpose: The purpose of this observational study was to examine the association between body mass index and dental caries in Hispanic children. The research evaluated the influences of obesity, diet, parent education level, family acculturation, tooth brushing habits and gender as predictors of childhood caries.

Methods: One examiner visually screened 177 third grade students from 3 elementary schools located in southern California's Coachella Valley. The children were screened for number of decayed, missing and filled teeth (DMFT). Height, weight, age and gender determined their body mass index. Primary caregivers completed a 30–point questionnaire for each participant. Multivariate analyses accessed the association between childhood dental caries and weight status and the influences of the measured variables.

Results: Results indicate that those in the obese category had a statistically significant lower rate of DMFT than did children in the healthy weight category. Overweight children showed a higher DMFT than healthy weight children but the results were not statistically significant. Covariates that significantly influenced this association were diet and socioeconomic status.

Conclusion: Results from this study provide oral health professionals with baseline data and literature to support development of preventive programs for this population that concurrently address both obesity and oral health issues in scope and design.

Keywords: dental caries, body mass index, diet, socioeconomic status

This study supports the NDHRA priority area, **Health Promotion/Disease Prevention:** How diversity among populations impacts the promotion of oral health and preventive behaviors.

Introduction

Childhood dental caries is a serious health problem. Although largely preventable, dental caries remains the most common chronic disease of children ages 6 to 11, with 41% of children in this age group experiencing decay in their primary (deciduous) teeth. Estimates are that 1 out of every 4 missed days of school are due to dental pain, predominantly caused by dental caries. Dental caries is harmful to children's growth, development and academic performance.[1]

According to the 2007 to 2008 National Health and Nutrition Examination Survey (NHANES), using measured heights and weights, 17% of children and adolescents ages 2 to 19 years were obese, and an additional 31.6% were overweight.[2] Since 1980, the number of overweight children ages 6 to 11 has doubled, and the number of overweight adolescents has tripled. Childhood obesity is currently the most prevalent nutritional condition of children in the U.S.[3]

Research results are contradictory regarding the association of childhood obesity and dental caries. This study attempted to clarify the association in a group of children from a geographic area that experiences a higher risk of both conditions.

Dental caries can lead to tooth loss, dental pain, infection and, in rare instances, death.[4–6] Childhood obesity can lead to increased risk of diseases such as type–2 diabetes and heart disease.[7] The burden both financially and physically for children experiencing dental caries and obesity places them at risk for a compromised quality of life.[8]

Children from families with an income below 199% of the federal poverty level are 3–times more likely to have their dental needs unmet than children from families 200% or above the federal poverty level. Estimates indicate that children lose approximately 52 million school hours each

year due to dental problems. Obesity and dental caries can both negatively affect a child's quality of life and ability to succeed in school.[9–11]

Research supports an association between ethnicity, obesity prevalence and dental caries experience. Data demonstrates that Hispanic children show a higher prevalence of dental caries and obesity as compared to their Caucasian counterparts.[12–14]

Both obesity and dental caries are linked to diet, making it important for studies of both conditions to assess diet. Snacking between meals, drinks containing high–fructose corn syrup or sucrose, and consumption of high–carbohydrate foods are associated with both an increase in dental caries and an increase in obesity.[15]

Women with lower educational levels are more likely to consume diets high in fat and carbohydrates.[16] These dietary choices are identified as risk factors for dental caries and increased body mass index (BMI). Women with a higher educational level make dietary choices containing more fruits and vegetables. These dietary choices are considered healthier for prevention of both dental caries and obesity in themselves and their children.[16]

In a review article by Vartanian et al, the authors evaluated the results of the effects of soft drink consumption on nutrition and health from 88 studies of subjects with varying ages.[17] Findings suggest that decreasing soft drink consumption would lower disease burden and weight gain. The results also indicate that the ingestion of fructose leads to a higher weight gain than ingestion of glucose. As a means of prevention of both dental caries and obesity, the authors suggest eliminating sugar–sweetened beverages along with paying attention to overall caloric intake.

Prior studies are unclear regarding an association between obesity and dental caries. Some research indicates a positive association,[15,18,19] while other research indicates a negative association between obesity and dental caries.[20] Still other research indicates no association between obesity and dental caries,[21–23] and some researchers report no association for younger children and a negative association for older children.[24] This study adds evidence to clarify these conflicting results.

The objective of this study was to examine the possible relationship between childhood dental caries and childhood obesity. This demographic population was chosen because previous research indicates it is a population with tendencies to exceed the average numbers of both childhood dental caries and obesity. The influence of other factors in this population, such as diet, gender, family acculturation and parents' education level and perceptions regarding oral health, were explored as covariates.

Methods and Materials

This observational study was conducted in Riverside County's Coachella Valley, located in southern California. The participants were all third grade students from 3 randomly selected elementary schools in the Coachella Valley Unified School District.[25] The residents of this geographical area are predominantly of Hispanic descent and of a low socioeconomic status (SES). Many of the residents are not fluent in English. All written materials to parents and students were provided in both English and Spanish. A total of 177 children (68 male and 109 female) were screened for decayed, missing and filled teeth (DMFT) and their height and weight were measured and BMI for age calculated. All third grade students were included in the study if they provided signed consent, parental signed consent, completed the questionnaire and were of Hispanic descent.

The participating child's parent or primary caregiver completed a 30–point questionnaire with questions ranging from dietary and tooth brushing habits to parents' perceptions about their child's weight status and dental caries rate. The questionnaire was available in both English and Spanish and pre–tested by 10 families with a translated, back–translated method, a valid and reliable tool for translation in cross–cultural research as shown in Brislin's model. Parents were also asked about their education level, family eligibility for the free or reduced–fee school lunch program and linguistic ability. Eligibility for the free or reduced–fee school lunch program was used as a proxy for determining SES. Linguistic acculturation as determined by the self–reported ability of the parent to speak, understand, read and write in English was used as a proxy for acculturation level.[26]

For consistency, 1 California registered dental hygienist performed all of the dental screenings. This was a non–invasive visual screening done with the use of a mouth mirror and illumination with Orascoptic's Zeon light (Orascoptic, Middleton, Wis.) attached to dental loupes. This screening determined the number of DMFT. No dental x–rays were used. Because this is an age group with mixed dentition, an attempt was made to

count, as missing, only those teeth that had been extracted due to decay. DMFT (restored with any method including stainless steel crowns) were included for a total count of teeth with decay experience. No distinction was made between primary and permanent teeth. In addition to the dental screening children were measured for height (cm) and weight (kg) in light clothing without shoes by 1 examiner. The gender and age were recorded for each participant.

The BMI of each participant was determined by entering the child's age, gender, height and weight into the BMI calculating tool provided by the Center for Disease Control and Prevention.[27]

The BMI and percentile standing for each of the participants were determined using this method: A number (0 to 3) was assigned correlating the percentile with the weight status of each participant. For purposes of this study, the children with a BMI that placed them in the underweight category were coded "0," the children classified as normal weight were coded "1," the children classified as overweight were coded "2" and the children classified as obese were coded "3."

Survey questions were designed to determine dietary habits, e.g. the number of snacks and number of high carbohydrate foods and drinks consumed per day. As a proxy for socioeconomic questions such as income, families who qualified for the free or reduced–fee school lunch program were considered of a low SES. Family acculturation level was determined by the self–reported ability of the parents to speak, understand, read and write in English. They were ranked from low acculturation to high acculturation level depending on their self–reported linguistic ability. Tooth brushing habits were measured as the number of times per week the child brushed before bedtime, as reported by the parent.

Statistical tests were run for 2 research questions with the dependent variable being the number of DMFT. The main independent variables of interest were BMI and diet. The covariates were gender, tooth brushing habits, SES and family acculturation. This was an observational study with quantitative data collected at 1 observation. The research questions were:

1. Is there an association between BMI and the number of teeth with dental carious lesions, and how is that association affected by the presence of other control variables, e.g. family SES and linguistic acculturation, parents' level of education, self–efficacy and perceptions regarding oral health and child's tooth brushing habits and gender?
2. Is there an association between diet and number of teeth with dental carious lesions and how is that association affected by the presence of other control variables, e.g. family SES and linguistic acculturation, parents' level of education, self–efficacy and perceptions regarding oral health and child's tooth brushing habits and gender?

A sample size was calculated through the use of the software G*Power based on a Poisson regression that models the total number of DMFT. The main independent variable of interest was assumed to be dichotomous with a 1:1 ratio. Other covariates were assumed to have R2 of 0.2 with the dependent variable. The base rate (the mean number of DMFT) was set to 4. For the desired effect size, relative rate of 1.4 (or 0.71 in the opposite direction) was used. With alpha of 0.05 (2–tailed) and power of 80%, this yielded a sample size of 74 observations. Initially, 300 students were asked to participate in the study, with the expectation of a 40% failure to provide consent, permission or complete the questionnaire. One hundred and seventy–seven participants qualified for inclusion in the study.

Data were analyzed using the SPSS v17. A double–entry method was used to make certain that data entry was accurate. Missing data were estimated using the Amos full information maximum likelihood analysis or estimating the missing values from the current data. Frequencies and percentages were performed on all categorical data and descriptive statistics performed for continuous data. Analysis was run with the use of negative binomial regression.

Results

Tables I and II show the results of frequencies and distributions of the demographic and behavioral data. In Table I, we see that 62% (n=109) of the child participants were female. The questionnaire was completed by mothers of participants 81% (n=144) of the time. Of the 177 families completing the questionnaire, 88.7% (157) qualified for the free or reduced–fee lunch program, placing their families in a low socioeconomic category. Respondents self–reported that 14.7% could not speak, understand, read or write in English (this is indicated by those answering "0" under acculturation). Thirty–six percent of the parents stated that they could speak, understand, read and write "very well" in English (this is indicated by those who answered 12 un-

Table I: Demographic Frequencies and Distributions of the Survey Respondents (n=177)

	n	%
Student's Gender		
Male	68	38.4
Female	109	61.6
Parent's Educational Level		
No school	5	2.0
Elementary only	35	19.7
Attended High School	45	25.4
Graduated HS	51	28.8
Some college	24	13.5
College grad	13	7.3
Graduate School	6	3.3
Family Eligibility for Free Lunch? (SES)		
No	20	11.3
Yes	157	88.7
Parental Acculturation		
0	26	14.6
1	14	7.9
2	3	1.7
3	3	1.7
4	20	11.3
5	4	2.3
6	9	5.0
7	6	3.4
8	20	11.3
9	4	2.3
10	1	0.6
11	3	1.7
12	64	36.2
Child's Weight Status		
Underweight	4	2.3
Healthy weight	92	52.0
Overweight	34	19.2
Obese	47	26.5
Child's Decayed, Missing, Filled Teeth		
0	41	23.2
1	13	7.3
2	17	9.6
3	18	10.2
4	23	13
5	11	6.2
6	11	6.2
7	11	6.2
8	13	7.3
9	5	2.8
10	6	3.4
11	6	3.4
12	1	0.6
17	1	0.6

Table II: Behavioral Frequencies and Distributions of the Survey Respondents (n=177)

	n	%
Last dental exam		
Never	2	1.1
6 months or <	101	57.1
12 months or <	56	31.6
24 months or <	18	10.2
Could not get care		
No	135	76.3
Yes	42	23.7
Nights brush per week		
0	20	11.3
1	15	8.5
2	14	7.9
3	22	12.4
4	31	17.5
5	75	42.4
Diet (number of carbs/day)		
4 or <	46	25.99
>4 <8	89	50.28
8<10	24	13.56
10>	18	10.17
Think most have caries		
No	46	26
Yes	131	74
Think overweight		
Strongly agree	35	19.8
Agree	57	32.2
Disagree	48	27.1
Strongly disagree	37	20.9
Think child has caries		
Don't know	30	16.9
No	60	33.9
Few	75	42.4
Many	37	6.8
Worried child overweight		
Strongly Agree	28	15.8
Agree	47	26.5
Disagree	64	36.2
Strongly Disagree	38	21.5
Worried child has caries		
Strongly Agree	35	19.8
Agree	57	32.2
Disagree	48	27.1
Strongly Disagree	37	20.9
Dental tx needs		
None	108	61
See DDS soon	49	27.7
Urgent care needed	20	11.3

der acculturation). The self–report acculturation questions were asked to determine the influence of acculturation of the family into the American diet and customs as opposed to those who have retained their cultural dietary habits.

Approximately 77% of the study participants had experienced dental caries. The national average for children of this age group is 41%. The demographic statistics also reveal that 26.6% of the study participants were classified as obese and 19.2% were classified as overweight. The national average for this age group is 17% obese and 31.6% overweight. There are more children in this study in the obese category than the national average, but the total percent of obese and overweight in this study is 45.8%, slightly less than the national average of 48.6%.

Results of the behavioral data showed that 41.8% of the children were overdue for a dental exam, and 24% of the parents responded that they felt they could not get the dental care that their child needed. They listed affordability as the reason in 30% of the cases. Forty–two percent of the parents reported that their child brushed before bed at least 5 times per week. Of the parents surveyed, 74% thought that a majority of children develop dental caries, while 26% felt they did not. In another response, 49% of the parents surveyed thought that their child had dental caries and 51% weren't sure or did not think their child had caries. These 2 variables were used as a measure of perceived seriousness and susceptibility and were answered in a dichotomous response (yes/no). Dental screening results indicated that a total of 37% of the participants needed to see a dentist soon. Of those, 11% needed urgent care.

Table III: Negative Binomial Model for Weight (n=136)

		95% Conf. Interval		
	OR	Lower	Upper	p–value
Weight status				
Healthy weight	1.000	–	–	–
Overweight	1.111	0.765	1.612	0.58
Obese	0.683	0.479	0.975	0.04
Gender				
Male	1.043	0.774	1.407	0.78
Female	1.000	–	–	–
Education				
Elementary or none	1.353	0.901	2.030	0.15
Attended/Graduated HS	1.000	–	–	–
Attended/Graduated College	1.019	0.698	1.487	0.92
Eligible for Free Lunch (SES)				
No	1.000	–	–	–
Yes	1.944	1.171	3.228	0.01
Acculturation				
(as continuous)	0.984	0.947	1.022	0.40
Last Exam				
<= 6 months	1.000	–	–	–
>6 months	0.920	0.680	1.244	0.59
Could not get care				
No	1.000	–	–	–
Yes	0.917	0.645	1.303	0.63
Night Brush per week				
(as continuous)	0.976	0.894	1.065	0.58
No. Carb Drinks				
(as continuous)	0.993	0.887	1.111	0.90
No. Carb Foods				
(as continuous)	1.011	0.966	1.060	0.63

Principal Findings

When analyzing the association of weight status with DMFT (Table III), results indicate that children from the obese category were less likely to have dental caries (OR=0.68, 95% CI (0.48, 0.98)) than children in the normal weight category, and this was statistically significant (p=0.04). The results also indicate that although there was a positive association between dental caries in the children in the overweight category in this study population, it was not significant (OR 1.11, 95% CI (0.77, 1.61)). These results are similar to those of Marshall et al, who found that children at risk of being overweight were more likely to experience dental caries than those who were obese when using healthy weight children as the control.[28]

The above results hold true when placing the demographic variables of gender, parent's education, SES and acculturation, and the behavioral variables of diet, tooth brushing habits, availability of dental care and frequency of dental visits into the model. SES has the only statistically significant effect on the association (OR=1.94, 95% CI (1.17, 3.23), p=0.01).

Table IV reports the association between diet

(number of carbohydrate–containing foods and drinks per day) and number of dental caries. Results indicate that there is a statistically significant association between diet and DMFT (OR=1.00, 95% CI (1.00, 1.13), p=0.04). When adding demographic and behavioral variables into the model, the association between diet and DMFT (OR=1.00, 95% CI (0.99, 1.12)) is no longer significant. SES remains a significant covariate in the association throughout the model (OR=1.93, CI (1.16, 3.21), p=0.01).

Table V shows the mean number of dental caries by categorical variables used in the model. The Kruskal–Wallis test for significance yields significant results for weight status (p=0.01), and using the Mann–Whitney test for significance yields significant results for SES (p=0.01).

Table IV: Negative Binomial Model for Diet

		95% Conf. Interval		
	OR	Lower	Upper	p–value
Diet				
	1	1.003	1.127	0.04
Diet with covariates				
	1	0.944	1.115	0.08
Gender				
Male	1	0.757	1.351	0.94
Female	1.046	0.772	1.417	0.78
Education				
Elementary or none	1.371	0.911	2.062	1.13
Attended/Graduated HS	1	–	–	–
Attended/Graduated College	0.957	0.656	1.397	0.92
Eligible for Free Lunch (SES)				
No	1	–	–	–
Yes	1.929	1.159	3.212	0.01
Acculturation				
(as continuous)	0.991	0.953	1.03	0.06
Last Exam				
<= 6 months	1	–	–	–
>6 months	0.89	0.657	1.206	0.45
Could not get care				
No	1	–	–	–
Yes	0.883	0.629	1.257	0.49
Night Brush per week				
(as continuous)	0.975	0.893	1.064	0.57

Discussion

Childhood dental caries and obesity are 2 of the most common afflictions affecting the health and quality of life of children. Results of this study indicate that childhood obesity and dental caries are common in our study population, especially in children from low–socioeconomic families. Diet plays a significant role in both conditions. The results of this study also indicate that, in this population, childhood dental caries and obesity coexist but are not necessarily associated. It is important for educators to note that the research indicates that when parents were asked to rate their child's weight status their answers correlated with the child's actual weight, however, they significantly underestimated the child's weight. Thus parents may be aware that their child is overweight but their perception of the seriousness of the problem was not accurate.

Gibson et al found that the strength of association between social class and dental caries experience was twice that of the association between tooth brushing and dental caries experience.[29] They also found that the association between social class and dental caries experience was nearly 3 times greater than the association between sugar consumption and dental caries. These results would lead to the assumption that lower SES is the variable with the greatest strength of association with increased dental caries. The results from this study indicate similar results, and are in agreement with Harris et al, who suggest that more longitudinal studies and those with validated measures of dietary and oral hygiene habits are needed to better understand this association.[30]

Health professionals should explore methods to improve access to appropriate foods and increase dietary education for low SES families to decrease the risk of both conditions. Future studies should include standardized measurements of the risk factors for both obesity and dental caries. Optimal study designs should be longitudinal to better assess the predictors of both conditions.

In his call to action regarding oral health, former U.S. Surgeon General David Satcher stated that there are profound and consequential oral health disparities within the U.S. population, and that scientific research is key to further reduc-

tion in the burden of diseases and disorders that affect the face, mouth and teeth.[31]

The geographic area of this study was primarily rural, low–socioeconomic, Hispanic and a designated health professional shortage area by the Department of Health and Human Services. This area was chosen because the population had many of the risk factors for both childhood obesity and dental caries. Our demographic results show that this population experiences a much higher prevalence of both obesity and dental caries than the national average. Eliminating disparities and improving access to care are vital in providing fair and equal preventive and educational information to those at highest risk. Information gathered in this research can be useful as support for further study, baseline data for those residing in the area and program implementation aimed at reducing both of these widespread, chronic childhood diseases through education and preventive program implementation. Inconsistencies in measurement and analysis may be factors in the confusing results of previous studies. The development of validated instruments is imperative for future studies.

Table V: Mean Number of Caries by Categorical Variables Used in the Model (n=138)

	n	Mean	SD	p–value
Weight status				0.01
Normal	96	4.2	3.4	
Overweight	34	4.8	3.5	
Obese	47	2.8	3.3	
Gender				0.82
Female	109	3.8	3.3	
Male	68	4.1	3.7	
Education				0.30
Elementary or none	38	4.8	4.0	
Attended/Graduated HS	96	3.7	3.2	
Attended/Graduated College	43	3.5	3.3	
Eligible for Free Lunch (SES)				0.01
No	20	2.2	2.4	
Yes	157	4.2	3.5	
Last Exam				0.49
<= 6 months	101	4.0	3.4	
>6 months	76	3.8	3.6	
Could not get care				0.58
No	135	4.0	3.5	
Yes	42	3.6	3.2	

a: Kruskal–Wallis Test
b: Mann–Whitney Test

Conclusion

Information from the literature indicates that both childhood obesity and childhood dental caries are complicated disease processes. As a means of decreasing the prevalence of both diseases it would be effective to strengthen and improve the knowledge of the health and educational workforce, families, legislators and other key players.

Mary J. Creske, RDH, RDHAP, DrPH, is an assistant professor of dental hygiene at Loma Linda University. Naomi N. Modeste, DrPH, is Professor of Health Promotion and Education at Loma Linda University, School of Public Health, Loma Linda, California. Joyce Hopp, PhD, MPH, is Distinguished Professor Emeritus of Health Promotion and Education at Loma Linda University, School of Public Health, Loma Linda, California. David Cort, PhD, is an Assistant Professor of Sociology at the University of Massachusetts, Amherst, Massachusetts. Sujatha Rajaram, PhD, is an Associate Professor of Nutrition at Loma Linda University, School of Public Health, Loma Linda, California.

References

1. U.S. Department of Health and Human Services. Take care of your child's teeth. Healthfinder.gov [Internet]. [cited 2010 May 9]. Available from: http://www.healthfinder.gov/prevention/ViewTopic.aspx?topicID=64&areaID=1

2. Singh GK, Siahpush M, Kogan MD. Rising social inequalities in US childhood obesity, 2003–2007. *Ann Epidemiol*. 2010;20(1):40–52.

3. Hong L, Ahmed A, McCunniff M, Overman P, Mathew M. Obesity and dental caries in children aged 2–6 years in the United States: National Health and Nutrition Examination Survey 1999–2002. *J Public Health Dent*. 2008;68(4):227–233.

4. Broder HL. Children's oral health–related quality of life. *Community Dent Oral Epidemiol*. 2007;35(Suppl 1):5–7.

5. Locker D, Allen F. What do measures of 'oral health–related quality of life' measure? *Community Dent Oral Epidemiol*. 2007;35(6):401–411.

6. Strauss RS. Childhood obesity and self–esteem. *Pediatrics*. 2000;105(1):e15.

7. Freedman DS, Mei Z, Srinivasan SR, Berenson GS, Dietz WH. Cardiovascular risk factors and excess adiposity among overweight children and adolescents: The Bogalusa Study. *J Pediatr*. 2007:150(1);12–17.

8. John J, Wenig CM, Wolfenstetter SB. Recent economic findings on childhood obesity: cost–of–illness and cost–effectiveness of interventions. *Curr Opin Clin Nutr Metab Care*. 2010;13(3):305–313.

9. Casamassimo PS, Thikkurissy S, Edelstein BL, Maiorini E. Beyond the dmft. The human and economic cost of early childhood caries. *J Am Dent Assoc*. 2009;140(6):650–657.

10. Filstrup SL, Briskie D, da Fonseca M, Lawrence L, Wandera A, Inglehart MR. Early childhood caries and quality of life: child and parent perspectives. *Pediatr Dent*. 2003;25(5):431–439.

11. Flegal KM, Ogden CL, Yanovski JA, et al. High adiposity and high body mass index–for–age in US children and adolescents overall and by race–ethnic group. *Am J Clin Nutr*. 2010;91(4):1020–1026.

12. Huntington NL, Kim IJ, Hughes CV. Caries–risk factors for Hispanic children affected by early childhood caries. *Pediatr Dent*. 2002;24(6):536–542.

13. Shiboski CH, Gansky SA, Ramos-Gomez F, Ngo L, Isman R, Pollick HF. The association of early childhood caries and race/ethnicity among California preschool children. *J Public Health Dent*. 2003;63(1):38–46.

14. Taveras EM, Gillman MW, Kleinman K, Rich–Edwards JW, Rifas–Shima SL. Racial/ethnic differences in early–life risk factors for childhood obesity. *Pediatrics*. 2010;125(4);686–695.

15. Sharma A, Hegde AM. Relationships between body mass index, caries experience and dietary preferences in children. *J Clin Pediatr Dent*. 2009;34(1):49–52.

16. Barker M, Lawrence W, Woadden J, Crozier SR, Skinner TC. Women of lower educational attainment have lower food involvement and eat less fruit and vegetables. *Appetite*. 2008;50(2–3):464–468.

17. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: A systematic review and meta–analysis. *Am J Public Health*. 2007;97(4):667–675.

18. Bailleul-Forestier I, Lopes K, Souames M, Azoguy-Levy S, Frelut ML, Boy-Lefevre ML. Caries experience in a severely obese adolescent population. *Int J Pediatr Dent*. 2007;17(5):358–363.

19. Willerhausen B, Blettner M, Kasaj A, Hohenfellner K. Association between body mass index and dental health in 1,290 children of elementary schools in a German city. *Clin Oral Investig*. 2007;11(3):195–200.

20. Sánchez-Pérez L, Irigoyen ME, Zepeda M. Dental caries, tooth eruption timing and obesity: a longitudinal study in a group of Mexican schoolchildren. *Acta Odontol Scand*. 2010;68(1):57–64.

21. Granville-Garcia AF, de Menezes VA, de Lira PI, Ferreira JM, Leite-Cavalcanti A. Obesity and dental caries among preschool children in Brazil. *Rev Salud Publica (Bogota)*. 2008;10(5):778–795.

22. Juárez-López ML, Villa-Ramos A. Caries prevalence in preschool children with overweight and obesity. *Rev Invest Clin*. 2010;62(2):115–120.

23. Pinto A, Kim S, Wadenya R, Rosenberg H. Is there an association between weight and dental caries among pediatric patients in an urban dental school? A correlation study. *J Dent Edu*. 2007;71(11):1435–1440.

24. Kopycka-Kedzierawski DT, Auinger P, Billings RJ, Weitzman M. Caries status and overweight in 2– to 18–year–old US children: findings from national surveys. *Community Dent Oral Epidemiol*. 2008;36(2):157–167.

25. School district and demographic information. Coachella Valley Unified School District [Internet]. [cited 2010 May 9]. Available from: http://www.coachella.k12.ca.us/demographics/

26. Cort DA. What happened to familial acculturation? *Ethnic Racial Studies*. 2010;33(2):313–335.

27. BMI Percentile Calculator for Child and Teen Metric Version. Centers for Disease Control and Prevention [Internet]. [cited 2011 February 10]. Available from: http://apps.nccd.cdc.gov/dnpabmi/Calculator.aspx?CalculatorType=Metric

28. Marshall TA, Eichenberger-Gilmore JM, Broffitt BA, Warren JJ, Levy SM. Dental caries and childhood obesity: roles of diet and socioeconomic status. *Community Dent Oral Epidemiol*. 2007;35(6):449–458.

29. Gibson S, Williams S. Dental caries in preschool children: associations with social class, toothbrushing habit and consumption of sugars and sugar-containing foods. Further analysis of data from the National Diet and Nutrition Survey of children aged 1.5-4.5 years. *Caries Res*. 1999;33(2):101–113.

30. Harris R, Nicoll AD, Adair PM, Pine CM. Risk factors for dental caries in young children: a systematic review of the literature. *Community Dent Health*. 2004;21(Suppl 1):71–85.

31. Peck SB. The crisis in children's dental health: A silent epidemic. Testimony before the Public Health Subcommittee on the US Senate Committee on Health Education, Labor and Pensions. ADHA [Internet]. 2002 [cited 2010 September 20]. Available from: http://www.adha.org/profissues/children_oral_health/testimony.htm

An Innovative HIV Training Program for Dental Hygiene Students

Periza Zaninovic, DDS; Zuhair S. Natto, BDS, MBA, MPH, DrPH(c); Rodney L. Turner, DDS; Gerardo A. Toledo DDS, MA; Graciela G. Duran, RDH; James R. Trott, DDS, MPH, MS; Thomas C. Rogers, DDS, MPH, MA

Abstract

Purpose: Patients with HIV/AIDS deserve to be treated with compassion and receive comprehensive care by their dentist and dental hygienists. Previous studies have shown that many dental and dental hygiene students had negative attitudes towards treating such individuals. This article addresses the effectiveness of a program that trains dental hygiene students in the issues of treating this population. All pre–doctoral students at the Loma Linda University School of Dentistry are required to receive this training. Dental hygiene students complete a pre–session survey during their first year and a post–session survey as they complete the training during their second year. The survey questionnaire was administered during the period of 2003 to 2009, during which 197 students completed responses to the post–session survey. Five questions in the survey address self–evaluation of knowledge, attitudes, confidence in the efficacy of Universal Precautions and Post–Exposure Prophylaxis (PEP) following blood borne exposures. This study reports on 5 overlapping 2 year testing cycles and shows significant shifts in all 5 areas surveyed. The most significant gains were in "Familiarity with PEP" and "Confidence in the Efficacy of PEP." These data support the usefulness of an HIV program in preparing future dental hygienists to deliver appropriate care to persons living with HIV/AIDS.

Keywords: HIV/AIDS, community–based education, dental hygiene education, blood borne exposures

This study supports the NDHRA priority area,

XXX:

XXX.

Introduction

HIV/AIDS first emerged as a recognized disease in 1981. No one could have predicted how this disease would evolve over the next 3 decades, the impact it would have worldwide and the enduring difficulties associated with preventing and treating it. The disease remains a great challenge to public health and human rights worldwide. In the heavily impacted developing countries, it is so devastating that it can impact the fabric of society and national security.[1] Due to the advances and accessibility of anti–retroviral therapy, HIV/AIDS is no longer considered an immediate death sentence, and is now being viewed in the wealthier developed countries as a chronic illness.[2] However, both a cure and effective vaccines remain elusive, and the successes of anti–retroviral therapy have also led to complacency, particularly among populations who are at risk of acquiring the disease.[1] A pressing problem in the developed countries lies in the fact that, although people with HIV are living longer, the number of new infections has not been reduced.[3]

The estimated number of new cases per capita in the U.S., according to the Center for Disease Control and Prevention (CDC) 2005 to 2008 surveillance report, went from 37,000 to 42,000 based on better and more complete reporting of new HIV infections.[3] The report further stated that this incidence rate has been roughly stable since the early 2000s.[1] Studies have shown that about 19.3% of HIV infected medical patients in the U.S. have unmet dental needs that have not been treated in the previous 6 months.[4] This number is much higher in states without dental benefits, where unmet dental needs may reach as high as 31.5%.[4] The highest recorded unmet need of 43% occurs predominantly among lower income African American women.[5] Formicola et al found that disparities exist in health care with respect to minority groups, and that bias and stereotypic beliefs held by providers may contribute to the disparities.[6] Dental professionals provide a unique role that can help alleviate pain and infection, which may increase the quality of life and ultimately impact the course of the disease.[4] This points out the need for knowledgeable and dedicated dental communities, who not only assist in early diagnosis, but also treat these HIV/AIDS patients in a compassionate, comprehensive manner to improve the lives of

those who are living with the disease and ultimately help the reduction of new infections with early detection.

There is not a great deal of scientific literature regarding the effects of educational efforts on the attitudes and beliefs of dental and dental hygiene students with respect to treating HIV–positive patients. Previous studies in the field of dental education suggested that male dental students had significantly stronger negative attitudes towards patients at risk for HIV/AIDS than female students.[7] The findings also pointed out a lack of knowledge which was significant due to the fact that it could "translate into a potential risk both for the patients and providers."[7] In this same study, the students suggested including case studies, discussion groups and possibly supervised clinical rotations to improve their curriculum. A more recent study demonstrated that dental students' knowledge of an HIV–seropositive status, and perceived responsibility of the patients for contracting HIV, could be predictive of negative attitudes towards the treatment of these patients.[8] In this study, Seacat et al recommended interaction between dental care providers and persons living with HIV/AIDS (PLWHA) that intermingled the classroom and clinical experience, as a mandatory component in the dental curriculum.[8] Other data also showed that negative attitudes and discrimination continues beyond the 4 year dental program and that dentists in postgraduate programs can also have a negative bias towards PLWHA and also toward homosexuals.[9]

In a study by Rohn et al that reviewed the social and psychological concerns that impede delivery of care to HIV–positive patients in the dental education arena, it was found that fear of status disclosure to health care workers among PLWHA was a significant barrier in access to care.[10] Their findings suggested ways to improve students' attitudes to reduce prejudicial or discriminatory behaviors, which in turn might improve patient confidentiality. These recommendations included:

- Inviting HIV–positive individuals to talk with students and to share their perspectives as patients
- Have faculty model appropriate ways of interacting with patients and discussion confidential information
- Provide role–playing opportunities for students as they start seeing patients in the clinic, so they have the chance to apply what is learned in the classroom

These, along with other recommendations, were made to prepare students to ultimately "enhance access to health care."[10]

In a study involving dental students and their comfort level in treating vulnerable populations and future willingness to treat, only 47.4% expressed comfort in treating HIV/AIDS patients, while 17.1% expressed willingness to treat PLWHA in the future.[11] Most of the students did not have any experience with seropositive patients, and only 22.7% had some experience. Generally, prior experience, such as community–based clinical experiences, had a positive impact on the comfort level of the students and, in some instances, translated into future willingness to treat vulnerable populations.[12,13] Mulligan et al also recognized the importance of providing continuing dental education on HIV after graduation that covers oral pathology, medical issues, medications, psychological issues, legal and ethical implications, risk assessment and OSHA principals.[14]

There are a few studies that specifically address the concerns of dental hygienists or dental hygiene students with respect to HIV/AIDS. King et al mailed a survey to practicing dental hygienists in the U.S. and received 856 responses.[15] A majority of respondents (53.9%) felt that treating patients with HIV/AIDS increased their personal risk, and 63.5% reported always using extra precautions with known HIV/AIDS patients. A total of 38% believed that double–gloving was appropriate when treating this population and 25.4% indicated that different sterilization and disinfection methods were necessary. The authors concluded that the attitudes and practices reported by many of the respondents suggest a lack of understanding of the concepts of infection control and standard precautions. They also recommended that dental hygiene students should have multiple experiences providing treatment for persons with infectious diseases such as HIV/AIDS.[15]

Cohen et al conducted a one–time survey of dental hygiene students to determine student attitudes toward persons with 2 different diseases (AIDS and leukemia) and sexual preferences (heterosexual and homosexual).[16] The survey indicated no bias toward homosexuals but found there was bias towards persons with AIDS. Giulani et al surveyed practicing dental hygienists in Italy and found that 5.9% of respondents indicated they had denied treatment based on patients' HIV status, and 80% of these respondents did so due to fear of getting the disease themselves.[17] Those who refused treatment reported a lower use of personal protective equipment, particularly eyewear, than those who did not deny treatment. These authors cited an older survey of dental health care workers from the pre–anti–retroviral therapy era, which found that willingness to treat HIV–infected per-

sons was proportional to the individuals' practice of more thorough infection control procedures.[18]

Dental Partnership Grant under the Ryan White Program

The Health Resources and Services Administration (HRSA) acknowledged the need for improved training of dental and dental hygiene students in the care of individuals with HIV. In 2002, HRSA announced a grant opportunity for funds under Part F of the Ryan White CARE Act. This grant established the Community–Based Dental Partnership Program in which dental education institutions were chosen to partner with community–based dental providers to train students in community clinical settings where dental care is provided to an HIV–positive clientele.[13] The Loma Linda University School of Dentistry partnered in the program with the Social Action Community Health System (SACHS) that operates a low–cost community dental clinic in nearby San Bernardino and has a large HIV–positive clientele. The Program and Application Guidance document for the grant indicated that grantees would have to "develop innovative curriculum design, quality improvement programs and program assessment methods."[19]

Development of the Loma Linda University School of Dentistry Program

The original faculty of the HIV and the Dentist program at Loma Linda University School of Dentistry reviewed the existing curriculum content of those pre–doctoral courses that addressed HIV disease to better understand what was actually being taught elsewhere in the predoctoral dental and dental hygiene programs. The intent was to build on what was already being taught and to minimize presentation of redundant material. The faculty also attended the continuing education program of the Pacific AIDS Education Training Center at the University of Southern California (USC) School of Dentistry. In reviewing the literature for this course, it was noted that participation in the USC program resulted in significantly changed HIV–related knowledge, attitudes and beliefs among course participants, as well as enhanced commitment to infection control and screening for risk behaviors and presence of HIV infection.[20] During this course, the Loma Linda University School of Dentistry faculty were able to interact with HIV–positive patients in a clinical setting, along with observing a role–playing experience that demonstrated possible ways of interacting with such patients.

In the Loma Linda program, there was an extensive effort to develop an innovative curriculum that involved faculty, dentists and staff at SACHS, and the community advisory group that was formed to give input on the program. Loma Linda University established this program as a required rotation for all predoctoral students during their senior year. Completion is a requirement for graduation. The program is conducted entirely at the SACHS clinic and has both clinical and didactic components. The didactic component includes lectures, discussion of cases, interaction with staff dentists, interviews with patients, role playing to illustrate possible responses to various clinical situations and viewing a video that was produced specifically for this program. The clinical component involves students providing dental care to the HIV–positive clientele under the supervision of faculty. During program development, it was decided to train the students in small groups (5 to 7 students) over 2 half–day periods. The students in each group spent a total of 8 hours during 1 week periods in the HIV training program at the community clinic so that every student could have direct interaction with HIV–positive patients. With this scheduling, it requires most of the academic year to train all of the dental and dental hygiene students each year. The curriculum for the HIV and the Dentist program is presented in Table I.

In the development of the HIV and the Dentist curriculum, the authors took advantage of the close proximity of the School of Dentistry and the SACHS clinic (3 miles). This allowed students to attend a 4 hour session in the morning at SACHS and still be able to attend the 4 hour afternoon session at Loma Linda University on the same day. The brevity of each student's training experience (8 hours) and the inability to predict the patient–care experiences that would be available each week contributed to the decision to consider the patient–care component as service learning. The authors also decided to assess program outcomes by means of pre–session and post–session surveys to determine if attitudinal shifts and self–reported gains in knowledge were occurring with respect to treating HIV–positive individuals. Rubin stated that there is evidence that service learning experiences help "develop cultural literacy, improve citizenship, enhance personal growth and foster a concern for social problems."[21]

The aim of this program is to help students at the Loma Linda University School of Dentistry manage the oral health care needs of persons with HIV infection. The purpose of this article is to discuss the impact the training program had on these students regarding the issues related to treating patients with HIV/AIDS. This discussion presents

Table I: HIV and the Dentist Curriculum at the Loma Linda University School of Dentistry

Session I	
Didactic Component	• HIV Origin and Epidemiology • Immune System • Understanding the HIV (Pathogenesis and Pharmacology) • Scope of Dental Treatment
Behavioral Component	• Video and role–play with students
Patient Interview	• Patient scheduled by SACHS staff (mediated by faculty)
Didactic Component	• HCV/HIV coinfection, overview of opportunistic infections • HIV/AIDS: Progression, transmission and clinical manifestations (Clinical Photos)
Session II	
Didactic Component	• Universal Precautions • HIV Post–Exposure Prophylaxis for health care workers • Dentistry, HIV, and the Law
Clinical Component:	• Patients scheduled by SACHS staff
Survey	
Wrap up	

the results of pre– and post–session surveys of dental hygiene students administered over a 6 year period. These surveys focused on attitudes towards the HIV–positive clientele, comfort with treating this group, confidence in the effectiveness of Universal Precautions and Post–Exposure Prophylaxis (PEP) following blood– borne exposures, and self–assessment of understanding the issues involved.

Methods and Materials

Assessment of program effectiveness has been measured since the program's inception by means of pre– and post–session surveys of the student participants. The survey questions were developed with the assistance of staff from the Behavioral Health program at SACHS. The pre–session survey is completed by dental hygiene students during the spring quarter of their first year in the Medically Compromised Patients course. The post–session survey is completed by the same students at the end of their second and final session each week as they complete their rotations during the second year. The surveys contained 5 statements regarding HIV general knowledge, attitudes towards the HIV–positive clientele, comfort with treating this group, confidence in the effectiveness of Universal Precautions and PEP following blood borne exposures, and self–assessment of understanding of the issues involved.

Analysis occurred with 6 years of pre– and post–session survey results (composed of 5 overlapping 2 year cycles). During 2003 to 2009, the surveys were distributed to 197 dental hygiene students, with 172 (87.31%) completing the post–session survey. Slight modifications were made to the surveys during the 6 year period, but general content remained the same. For example, at the end of the survey there was a place for student comments. The most frequent comment made was that students wanted to have more time with patients. The next most frequent comment was that there was some overlap of information from previous courses. A few changes were made in the course as a response to those comments cited above to eliminate unplanned redundancies. There was also a small reduction of didactic material presented to allow for more clinical time during the second day of the rotation. The range of student responses did not change substantially from year to year during the 6 year period of the study.

Statistical Analysis

Statements used in the surveys and the student responses during the study period are summarized in Table II. All statistical analysis was done using SAS 9.2. Those dental hygiene students who did not complete post–training questionnaires were excluded from the analysis. Descriptive statistics were generated, including means. The normality distributions were examined using histograms and Kolmogorov–Smirnov normality tests. Given the nature of the data, the non–parametric Wilcoxon Signed–Rank Test was performed between data recorded in pre–session and post–session surveys. A p–value of <0.05 was considered statistically significant. The data was compared on a 6–point Likert Scale ranging from "none" to "high" in all 5 areas surveyed as displayed in the header of Table II. The overall change in means was compared to generate the final result.

Table II: Percentages of dental hygiene student responses to the statements about general knowledge and treatment of HIV patients

Statement	Session	None	Insufficient	Marginal	Acceptable	Sufficient	High	Mean	p–value
General knowledge about HIV infection	Pre	0.00	4.57	29.44	32.49	29.90	6.60	4.02	
	Post	0.00	0.00	4.65	31.98	29.07	34.3	4.99	
	Change							24.13	<0.0001
Feel competent about treating an HIV positive patient	Pre	0.00	9.23	30.26	28.21	17.95	14.36	4.04	
	Post	0.00	0.00	1.16	19.08	32.95	46.82	5.44	
	Change							34.65	<0.0001
Familiarity with the PEP (Post–Exposure Prophylaxis)	Pre	25.13	22.05	30.26	13.33	4.62	4.62	2.65	
	Post	0.00	4.05	5.20	26.59	32.95	31.21	4.88	
	Change							84.51	<0.0001
Feel confident about the efficacy of PEP	Pre	17.53	16.49	34.02	16.49	11.34	4.12	3.02	
	Post	0.00	1.88	3.13	17.50	21.25	56.25	5.41	
	Change							79.14	<0.0001
Feel comfortable with effectiveness of universal precautions in the dental office	Pre	0.51	8.63	20.30	26.40	22.84	21.32	4.32	
	Post	0.00	0.00	1.78	10.65	23.08	64.5	5.80	
	Change							34.26	<0.0001

Results

The Wilcoxon Signed–Rank Test revealed positive changes in all the categories that were highly statistically significant at a p–value of 0.0001 (Table II). The categories included "general knowledge about HIV infection" where there was a 24.1% change in the positive direction (pre–session mean equaled 4.02, and post–session mean equaled 4.99). The category "feeling competent in treating HIV positive patients" had a change of 34.7% (4.04, 5.44). The category of "familiarity with PEP" had a change of 84.5% (2.65, 4.88). The category of "confidence in the efficacy of PEP after an exposure to a contaminated dental instrument" had a change of 79.1% (3.02, 5.41). The final category addressed the "level of comfort with effectiveness of Universal Precautions in the dental office with reference to HIV infection," and had a positive change of 34.3% (4.32, 5.80). The overall change in all the areas about the knowledge and treatment of HIV patients was in a positive direction, with the most convincing results seen in the area of familiarity with PEP and confidence about the efficacy of PEP following a blood borne exposure. A graphic presentation of a comparison of the means of the pre– and post–session responses to the 5 survey items is presented in Figure 1.

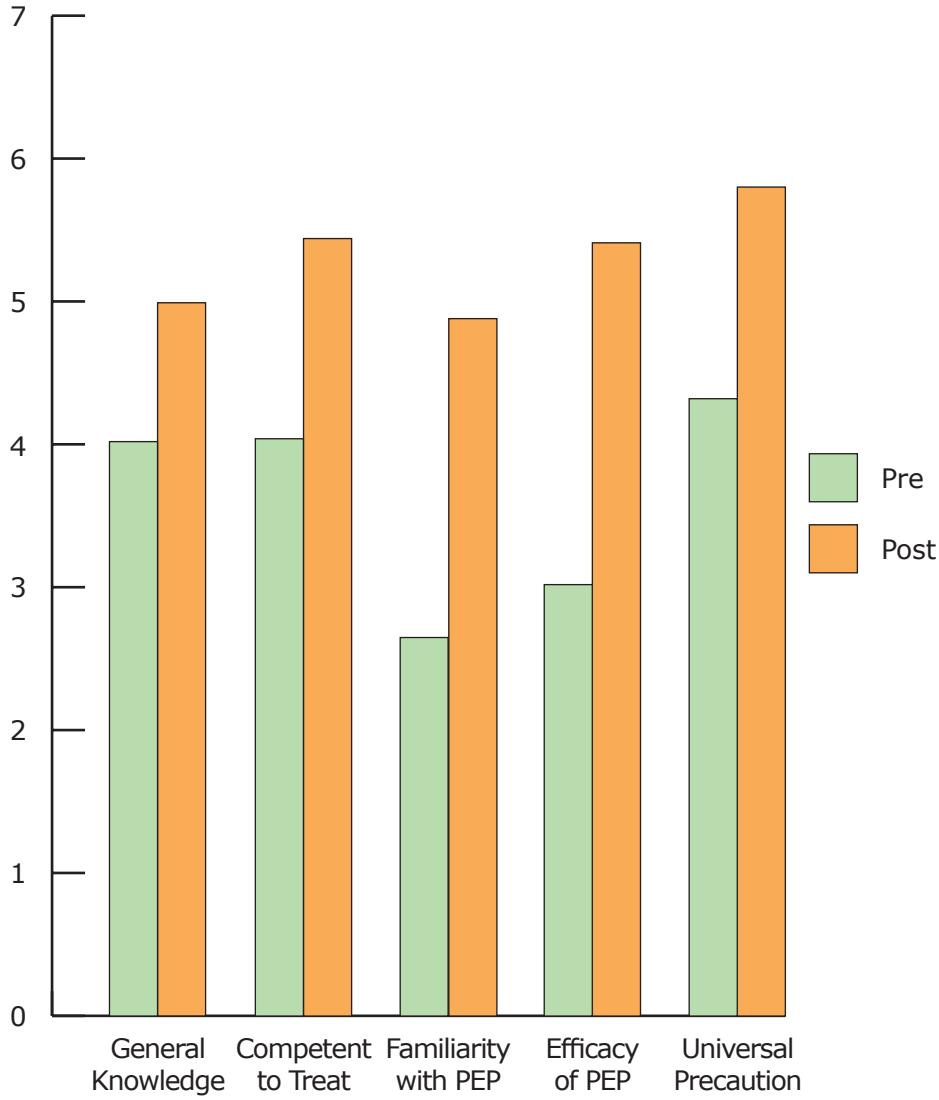
Discussion

A goal of the HIV and the dentist program was to improve the students' knowledge, attitudes and beliefs regarding the provision of dental treatment to PLWHA. Significant changes were observed in all 5 areas surveyed as reported in Table II. The observed changes are taken as evidence that student competence in dealing with HIV–positive patients has been improved by this program. It is also evident from Figure 1 that the differences in the pre– and post–session responses were substantially different in the 2 responses involving PEP as compared to the other 3 areas. The most likely reason for this is that the 3 items with the least pre– and post–session change are those areas to which the students had more substantial curricular exposure prior to the HIV and the Dentist training experience. Due to the fact that the second year dental hygiene students have already been introduced to HIV–related topics in the school curriculum, we expected the students to already have some HIV knowledge, resulting in less of a measurable difference in the post–test data regarding general knowledge of HIV infection. This was also the case for feeling competent to treat HIV–positive patients and belief in the efficacy of Universal Precautions. The other 2 items, which addressed familiarity with PEP and confidence in the efficacy of PEP, had been minimally addressed elsewhere in the curriculum, and these were the areas where the greatest changes were observed in the positive direction. All areas surveyed had a significant p–value of <0.0001.

One of the predictable findings of this program has been that, after completion of the externship, an increased number of students felt competent

treating an HIV–seropositive patient. The data in Table II show that 60.1% of the first year dental hygiene students already expressed a feeling of competence to treat HIV–positive patients in the pre–session surveys. The expression of competence to treat rose to 98.9% in the post–session surveys. Part of this increase in confidence may be attributable to the additional clinical experience of the second year students (as compared to their first year status when completing the pre–session survey). The remainder of the increase is most likely attributable to the HIV training program. One of the unique features of this program that may have increased confidence has been the personal interview of an HIV positive patient by the students. This degree of personal interaction may have been the major factor in the most frequently observed comment in the post–session surveys in which students requested more patient contact time.

Figure 1: The Means of Pre– and Post–Session Responses to the 5 Survey Items



The most significant change was in the area of PEP. The "Familiarity with PEP" protocol had the greatest statistical change in the desired direction at 84.5%. Confidence in the efficacy of PEP after a blood–borne exposure had the second highest positive change at 79.1%. A likely explanation for these findings is that the HIV and the Dentist rotation is the only place in the dental hygiene curriculum where PEP is described in detail. Students show a high level of interest in strategies to protect themselves from acquiring an infectious disease like HIV due to occupational exposure. In addition, magnitude of the change may also be related to the fact that PEP was the last topic reviewed prior to the treatment of the patients during the second training session. Students were always encouraged to ask questions or make comments, and PEP has been one of the subjects where there has been high student participation in the form of questions and comments.

A less predictable finding was in reference to the increased confidence of Universal Precautions in relation to preventing transmission of HIV, with a 34.3% change in the response mean. There was a brief review of Universal Precautions since the students had already been exposed to the subject multiple times in the Loma Linda University School of Dentistry curriculum, yet there was still a highly significant change in a positive direction in this survey item. The categories of "Effectiveness of Universal Precautions," "General Knowledge of HIV Infection" and "Feel Competent about Treating an HIV Positive Patient" had the most significant shift in the 2 lowest categories (None and Insufficient) from the pre– to the post–session surveys. All students expressed at least a marginal comfort level in these 3 areas after the externship. A possible future study could be to survey those students that participated in the

training program after graduation, to determine if the positive outcomes observed in the study translated into an increased willingness to treat HIV patients. Additional questions in such a study could include reflecting on the importance of this rotation in their pre–doctoral training and whether it inspired them to continue to learn about treatment issues regarding people with HIV disease and other medically compromising illnesses.

Future modifications to the HIV and the dentist program could include training dental and dental hygiene students to conduct rapid HIV testing in the dental setting. An estimated 1.1 million persons in the U.S. are living with HIV/AIDS, and an estimated 24 to 27% of these people are undiagnosed and/or unaware of their HIV–positive status.[22] Patients who become aware of their HIV diagnosis are more likely to reduce high risk behavior.[23] It is estimated that approximately 25% of HIV–positive patients are responsible for 55% of the new cases, and most of these individuals are in the unaware category.[23] Dental professionals are an untapped resource to access patients for HIV testing. Knowledge of a positive sero–status can help with early access to treatment, along with ultimately reducing further transmission.[24] A study was conducted in a dental clinic to see how patients would respond to oral HIV testing in the dental setting. In that study, 73% of patients were willing to undergo HIV testing, and 37% actually preferred their dentist above any other provider.[25] The CDC launched the Advancing HIV Prevention initiative in 2003, which allows testing to be conducted in a variety of settings, aimed at increasing the early diagnosis of HIV positive people.[26] A separate study also determined that one–third of dental educators would consider offering rapid oral HIV testing in their clinics.[27]

Conclusion

The pre– and post–session survey data gathered over a 6 year period demonstrated that the program was successful in fulfilling the original goals of HRSA to impart a social context for health care and a greater understanding of the health needs of the HIV–positive population. The most significant changes in student knowledge were in the areas of PEP, followed by "Familiarity with PEP" protocol. Future studies may investigate teaching dental hygienists and dental students to conduct rapid HIV testing the dental setting.

Periza Zaninovic, DDS, is employed at the Mendocino Community Health Clinic, Ukiah, California. Zuhair S. Natto, BDS, MBA, MPH, DrPH (candidate), is currently a resident in the graduate program in Periodontics at Tufts University, Boston, Massachusetts, and is also a Lecturer in Community Dentistry, School of Dentistry, King Abdulaziz University, Saudi Arabia. Rodney Turner, DDS, is employed as a community dentist at the SAC Health System in San Bernardino, California. Gerardo Toledo, DDS, MA, is employed at Gerardo Toledo DDS – practice limited to Endodontics, Redlands, California. Graciela Duran, RDH, is employed at the Loma Linda University School of Dentistry. James Trott, DDS, MPH, MS, is employed at the Loma Linda University School of Dentistry. Thomas Rogers, DDS, MPH, MA, is employed at the Loma Linda University School of Dentistry.

Disclosure

This project was supported by a Community–Based Dental Partnership Grant issued under Part F of the Ryan White Program by the Federal Health Resources and Services Agency (HRSA) HIV/AIDS Bureau, Grant number H65HA00004.

References

1. Stine GJ. AIDS UPDATE 2009: An Annual Overview of Acquired Immune Deficiency Syndrome. Columbus: McGraw–Hill; 2009.

2. Pierret J. Everyday life with AIDS/HIV: surveys in the social sciences. *Soc Sci Med*. 2000;50(11):1589–1598.

3. Centers for Disease Control and Prevention. HIV/AIDS Statistics and Surveillance. CDC Division of HIV/AIDS Statistics. 2008.

4. Marcus M, Freed JR, Coulter ID, et al. Perceived unmet need for oral treatment among a national population of HIV–positive medical patients: social and clinical correlates. *Am J Public Health*. 2000;90(7):1059–1063.

5. Shiboski CH, Palacio H, Neuhaus JM, Greenblatt RM. Dental care access and use among HIV–infected women. *Am J Public Health*. 1999;89(6):834–839.

6. Formicola A, Stavisky J, Lewy, M. Cultural Competency: Dentistry and Medicine Learning from One Another. *J Dent Educ*. 2003;67(8):869–875.

7. Seacat JP, Inglehart MR. Education about treating patients with HIV infections/AIDS: the student perspective. *J Dent Educ*. 2003;67(6):630–640.

8. Seacat JD, Litt MD, Daniels AS. Dental students treating patients living with HIV/AIDS: the influence of attitudes and HIV knowledge. *J Dent Educ*. 2009;73(4):437–444.

9. Cohen LA, Romberg E, Grace EG, Barnes DM. Attitudes of advanced dental education students toward individuals with AIDS. *J Dent Educ*. 2005;69(8):896–900.

10. Rohn EJ, Sankar A, Hoelscher DC, Luborsky M, Parise MH. How do social–psychological concerns impede the delivery of care to people with HIV? Issues for dental education. *J Dent Educ*. 2006;70(10):1038–1042.

11. Kuthy RA, McQuistan MR, Riniker KJ, Heller KE, Qian F. Students' comfort level in treating vulnerable populations and future willingness to treat: results prior to extramural participation. *J Dent Educ*. 2005;69(12):1307–1314.

12. Kuthy RA, Heller KE, Riniker KJ, McQuistan MR, Qian F. Students' opinions about treating vulnerable populations immediately after completing community–based clinical experiences. *J Dent Educ*. 2007;71(5):646–654.

13. Mofidi M, Gambrell A. Community–based dental partnerships: improving access to dental care for persons living with HIV/AIDS. *J Dent Educ*. 2009;73(11):1247–1259.

14. Mulligan R, Lemme SA. HIV risk assessment: building on dentistry's history of promoting health. *J Calif Dent Assoc*. 2001;29(2):149–154.

15. King TB, Muzzin KB. A national survey of dental hygienists' infection control attitudes and practices. *J Dent Hyg*. 2005;79(2):8.

16. Cohen L, Romberg E, Dixon DA, Grace EG. Attitudes of dental hygiene students toward individuals with AIDS. *J Dent Educ*. 2005;69(2):266–269.

17. Giuliani M, Tumbarello M, Marino M, et al. Dental hygienists behavior towards HIV–positive patients in highly active antiretroviral therapy era: a pilit survey. *Int J Dent Hyg*. 2011;9(3):204–210.

18. Gerbert B, Badner V, Maguire B. AIDS and dental practice. *J Public Health Dent*. 1988;48(2):68–73.

19. Health Resources and Services Administration (HRSA). The HIV/AIDS Program: Part F Community–Based Dental Partnership Program. U.S. Department of Health and Human Services. 2002.

20. Mulligan R, Seirawan H, Galligan J, Lemme S. The effect of an HIV/AIDS educational program on the knowledge, attitudes, and behaviors of dental professionals. *J Dent Educ*. 2006;70(8):857–868.

21. Rubin RW. Developing Cultural Competence and Social Responsibility in Preclinical Dental Students. *J Dent Educ*. 2004;68(4):460–467.

22. Centers for Disease Control and Prevention. HIV/AIDS Surveillance Report 2005–2008. CDC. 2008.

23. Marks G, Crepaz N, Senterfitt JW, Janssen RS. Meta–analysis of high–risk sexual behavior in persons aware and unaware they are infected with HIV in the United States: implications for HIV prevention programs. *J Acquir Immune Defic Syndr*. 2005;39(4):446–453.

24. Vernillo AT, Caplan AL. Routine HIV testing in dental practice: can we cross the Rubicon? *J Dent Educ*. 2007;71(12):1534–1539.

25. Dietz CA, Ablah E, Reznik D, Robbins DK. Patients' attitudes about rapid oral HIV screening in an urban, free dental clinic. *AIDS Patient Care STDS*. 2008;22(3):205–212.

26. Bowles KE, Clark HA, Tai E, et al. Implementing rapid HIV testing in outreach and community settings: results from an advancing HIV prevention demonstration project conducted in seven U.S. cities. *Public Health Rep*. 2008;123(Suppl 3):78–85.

27. Patton LL, Santos VA, McKaig RG, Shugars DC, Strauss RP. Education in HIV risk screening, counseling, testing, and referral: survey of U.S. dental schools. *J Dent Educ*. 2002;66(10):1169–1177.