Disclosures

Brian Nový, DDS
Executive Director
DentaQuest Oral Health Center

Dr. Nový has previously received financial compensation as a speaker from the following companies: GC America, Air Techniques, Xlear, Philips, Voco, SDI, Triodent, Shofu, CariFree, Solution Reach.
Who Caries?

Brian B. Nový, DDS
Executive Director, DentaQuest Oral Health Center
Dental professionals strongly cautioned

<table>
<thead>
<tr>
<th>PG-13</th>
<th>Dental professionals strongly cautioned</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Some material may be inappropriate for those expecting the standard lecture</td>
</tr>
</tbody>
</table>

This lecture has been rated PG-13.

The lecture contains moderate language, references to God, drug content, peril, violence, lewdness, violations of the law, drama, and tangential stories…
In the interest of transparency...

This lecture IS NOT sponsored

Previous lectures have been sponsored by:
GC America, Xlear, Solution Reach, Air Techniques, CariFree,
Philips, Ivoclar, Triodent, Shofu, SDI, VOCO and GSK

Adjunct Associate Professor LLUSD
with research funding from Oragenics and others above

Director of Practice Improvement, DentaQuest Institute

Photos within this presentation were taken with my iPhone, a Canon G6, or Shofu's Eye Special II.
Some pictures were obtained from the internet. Images may have been altered for contrast, brightness, and gamma...that's it.
Here are the current course handouts. Please note: There is no evidence based protocol for treating caries. I suggest reading JADA 2011;142(9) 1065-1071 for some of the most current science and expert opinion on the topic of medically managing tooth decay. Peace be with you and "Go see Ghost Town..." -Brian

References

Twelve Thin

Material Science

Something Motivational

The Fifth Handout
“It is paradoxical that the hardest of all tissues is so susceptible to lesions in living humans, while it is the most indestructible in the dead.”
Meet Derek
• “Caries Research” has been published for forty-five years
• Dentists have tried to stop this disease for more than one hundred years
• Caries is the most prevalent disease in the world
• S. mutans is the second most popular organism in the scientific literature!
DIVERSE FACTORS OF DENTAL CARIES
Clinical observations often show that dental caries is not necessarily active in unclean mouths. It has been shown by a number of investigators that caries can be reduced without changing habits of oral hygiene. This indicates that some force, such as systemic conditions, is a counterbalancing factor in the welfare of the teeth. Dental destruction seems to be dependent on some deficiency or excess of dietary elements, systemic disturbances, or hereditary taint. Any of these factors may affect the natural protection of the teeth (saliva and dental pulp),
As a Fellow in the American College of Dentists

“...it is not enough for the dentist to rely on [a speaker’s] presentation about a product’s safety and efficacy. The dentist has an independent obligation to inquire into the truth and accuracy of such claims and verify that they are founded on accepted scientific knowledge or research...”
There's more to it than impact factors...

- Systematic Reviews
- Randomized Controlled Trials
- Cohort Studies
- Case Control Studies
- Case Reports
- Expert Opinion
- Common Sense

Brian Nový's opinion
**Randomization**

Increases initial quality

**Outcome**

Critical

Important

Not

Summary of findings & estimate of effect for each outcome

**Grade recommendations**

- For or against (direction) ±
- Strong or conditional/weak (strength)

By considering balance of:

- Quality of evidence
- Balance benefits/harms
- Values and preferences

Revise if necessary by considering:

- Resource use (cost)

**Guideline**

Formulate Recommendations (†, ♦, ©...):

- "The panel recommends that ... should..."
- "The panel suggests that ... should..."
- "The panel suggests to not ...
- "The panel recommends to not..."
sealants for preventing and arresting pit-and-fissure occlusal caries in primary and permanent molars

A systematic review of randomized controlled trials - a report of the American Dental Association and the American Academy of Pediatric Dentistry

ABSTRACT

Background. National Health and Nutrition Examination Survey 2011-2012 data indicated that, in the United States, nearly one-fourth of children and over one-half of adolescents experienced dental caries in their permanent teeth. The purpose of this review was to summarize the available clinical evidence regarding the effect of dental sealants for the prevention and management of pit-and-fissure occlusal carious lesions in primary and permanent molars, compared with a control without sealants, with fluoride varnishes, or with other head-to-head comparisons.

Type of Studies Reviewed. The authors included parallel and split-mouth randomized controlled trials that included at least 2 years of follow-up, which they identified using MEDLINE (via PubMed), Embase, LILACS, the Cochrane Central Register of Controlled Trials, and registers of ongoing trials. Pairs of reviewers independently conducted the selection of studies, data extraction, and risk of bias assessments.
DECAY (caries)
Figure 5. Ecological shifts associated with microbial adaptation and selection. MS: mutans streptococci; non-MS, no mutans streptococci (oral streptococci except mutans streptococci); PG: Porphyromonas gingivalis. Modified, combined, and extended from Takahashi (2005) and Takahashi and Nyvad (2008).
The Caries Balance / Imbalance

Disease Indicators
- White Spots
- Restorations < 3 yrs
- Enamel Lesions
- Cavities / Dentin

Risk Factors
- Bad Bacteria
- Absence of Saliva
- Dietary Habits (poor)

Protective Factors
- Saliva & Sealants
- Antibacterials
- Fluoride / Ca²⁺ / PO₄³⁻
- Effective Diet
- Risk-based Reassessment

(Caries Disease Progression) (Health)
It’s incredibly complex and yet our “treatments” only replace missing tooth structure.

What would life be like if we had as many treatments for caries as we have dentin bonding agents?
Caries is NOT a fluoride deficiency!
Does this patient have caries?
Some things shouldn't be poked.
Remineralization of Caries Lesions Extending into Dentin

INTRODUCTION

In the past four decades, remineralization of the dental hard tissues has been at the subject of numerous studies. Key investigations which have prompted comprehensive studies on the mechanisms of mineral deposition and its implications for caries prevention were those by Kiousis et al. (1965) and Blacker and Drisko (1966) in the early 1960s. There is now a preponderance of data to show that early white-spot enamel lesions are remineralized when brought into contact with saliva (for review, see Dowd, 1999). Laboratory studies have revealed that remineralization occurs by crystal growth of the apatite crystallites which were partly dissolved during caries attacks (Ten Cate et al., 1981). After initial attention to the more fundamental aspects, clinical studies and in vivo model experiments were undertaken to assess the magnitude and frequency of the “caries reversals” in vivo. Modern diagnostic methods facilitate the quantitative analysis of the remineralization state of early enamel lesions and have been used to measure remineralization in vivo (Ahl-Kluteet al., 1998). Even with more crude diagnostic methods (such as radiographs), caries reversals have been reported (Baleshock et al., 1998).

Decay of the root surface has also been the focus of caries research. Again, investigations into non-restorative repair have shown that the remineralization and arrest of root dentin lesions should be a concern in caries prevention (Nyvad and Fejerskov, 1986). The dentin tissue differs from enamel because of its smaller crystallites and considerably larger volume of organic components. Several authors have reported that collagen neither contributes to nor interferes with the remineralization of dentinal lesions (Kisewei Ten Cate, 1991; Fisherman, 1994). The organic matrix does not serve as a matrix for apatite precipitation unless in the presence of specific proteins (Lassig and Linde, 1995).

Given the observation that dentin can also be remineralized, the question may be raised whether coronal lesions extending into the dentin can be remineralized. This study was aimed at investigating the remineralization of lesions extending through enamel into dentin, the relative efficacy of enamel and dentin remineralization, and the possible effects of agents which either stimulate (fluoride) or inhibit (arbitrarily) crystal growth.

MATERIALS & METHODS

The study was performed on sections which were covered with a protective layer, except for the thin outer enamel surface. This facilitated the non-destructive monitoring of changes inside the specimen—both enamel and dentin—by microdensitometric analysis.

Preparation of the Single Sections

Plano-parallel sections approximately 100 µm thick were cut perpendicular to the surfaces of bovine incisors and were totally covered with a bonding agent, Scotchbond Multi-purpose (3M, St. Paul, MN, USA). The sections were then embedded in a thin envelope of Araldite® (CIBA-GEIGY Plastics, Amherst, The Netherlands). After 3 days’ setting of the Araldite, the outer enamel surface of each section was cut off parallel to and at a distance of about 200 µm from the...
Figure 2. Average relative remineralization, with time of remineralization, for the five experimental groups in the four zones of interest: A, outer enamel, B, inner enamel, C, outer dentin, and D, inner dentin (n = 5 per group). Markers indicate the five experimental groups: control (●), 1000 ppm fluoride (▲), 1 ppm fluoride (○), MHBP (□), and combination (■).
"How would you chart the distal of #9?"

Is it progressing?
Is it arrested?
Has it reached the dentin?
Should it be restored?
In order to measure remineralization, you need a new way to classify lesions.

["sorry Dr. Black"]
Sealants for preventing and arresting pit-and-fissure occlusal caries in primary and permanent molars

A systematic review of randomized controlled trials—a report of the American Dental Association and the American Academy of Pediatric Dentistry

John T. Wright, DDS, MS; Malavika P. Tampi, MPH; Laurel Graham, MLS; Cameron Estrich, MPH; James J. Crall, DDS, MS, ScD; Margherita Fontana, DDS, PhD; E. Jane Gillette, DDS; Brian B. Nový, DDS; Vineet Dhar, BDS, MDS, PhD; Kevin Donly, DDS, MS; Edmond R. Hewlett, DDS; Rocio B. Quinonez, DMD, MS, MPH; Jeffrey Chaffin, DDS, MPH, MBA, MHA; Matt Crespin, MPH, RDH; Timothy Iafolla, DMD, MPH; Mark D. Siegal, DDS, MPH; Alonso Carrasco-Labra, DDS, MSc, PhD(c)

ABSTRACT

Background. National Health and Nutrition Examination Survey 2011-2012 data indicated that, in the United States, nearly one-fourth of children and over one-half of adolescents experienced dental caries in their permanent teeth. The purpose of this review was to summarize the available clinical evidence regarding the effect of dental sealants for the prevention and management of pit-and-fissure occlusal carious lesions in primary and permanent molars, compared with a control without sealants, with fluoride varnishes, or with other head-to-head comparisons.

Type of Studies Reviewed. The authors included parallel and split-mouth randomized controlled trials that included at least 2 years of follow-up, which they identified using MEDLINE (via PubMed), Embase, LILACS, the Cochrane Central Register of Controlled Trials, and registers of ongoing trials. Pairs of reviewers independently conducted the selection of studies, data extraction, risk of bias assessments, and quality of the evidence assessments by using the Grading of Rec-
Heterogeneity: Not applicable
Test for overall effect: $z = 0.86$ ($P = .39$)

### J. 1.1 No caries

<table>
<thead>
<tr>
<th>Study</th>
<th>Effect Size</th>
<th>95% CI</th>
<th>Number of Teeth</th>
<th>Caries Incidence</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arrow and Riordan, 1995</td>
<td>-1.7</td>
<td>-0.451</td>
<td>412</td>
<td>12.6%</td>
<td>0.18 (0.08-0.44)</td>
</tr>
<tr>
<td>Pardi and Colleagues, 2005</td>
<td>-0.6</td>
<td>0.671</td>
<td>97</td>
<td>10.6%</td>
<td>0.55 (0.15-2.04)</td>
</tr>
<tr>
<td>Amin, 2008</td>
<td>0.178</td>
<td>1.249</td>
<td>24</td>
<td>6.2%</td>
<td>1.19 (0.10-13.82)</td>
</tr>
<tr>
<td>Baseggio and Colleagues, 2010</td>
<td>0.941</td>
<td>0.171</td>
<td>628</td>
<td>14.5%</td>
<td>2.56 (1.83-3.58)</td>
</tr>
<tr>
<td>Antonson and Colleagues, 2012</td>
<td>-1.444</td>
<td>1.605</td>
<td>27</td>
<td>4.5%</td>
<td>0.24 (0.01-5.48)</td>
</tr>
<tr>
<td>Dhar and Chen, 2012</td>
<td>-0.936</td>
<td>0.721</td>
<td>50</td>
<td>10.1%</td>
<td>0.39 (0.10-1.61)</td>
</tr>
<tr>
<td>Chen and Li, 2013</td>
<td>0</td>
<td>0.601</td>
<td>75</td>
<td>11.2%</td>
<td>1.00 (0.31-3.25)</td>
</tr>
<tr>
<td>Guler and Yilmaz, 2013</td>
<td>-0.883</td>
<td>0.422</td>
<td>68</td>
<td>12.8%</td>
<td>0.41 (0.18-0.95)</td>
</tr>
<tr>
<td>Haznedaroglu and Colleagues, 2016</td>
<td>0.06</td>
<td>1.424</td>
<td>64</td>
<td>5.3%</td>
<td>1.08 (0.07-17.58)</td>
</tr>
</tbody>
</table>

**Subtotal (95% CI)**

<table>
<thead>
<tr>
<th>Effect Size</th>
<th>95% CI</th>
<th>Number of Teeth</th>
<th>Caries Incidence</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>-0.96</td>
<td>0.22</td>
<td>1,445</td>
<td>1,562</td>
<td>87.8%</td>
</tr>
</tbody>
</table>

Heterogeneity: $I^2 = 1.33; X^2 = 47.77, P < .00001; f = 830$

Test for overall effect: $z = 0.99$ ($P = .32$)

### J. 1.3 Mixed

<table>
<thead>
<tr>
<th>Study</th>
<th>Effect Size</th>
<th>95% CI</th>
<th>Number of Teeth</th>
<th>Caries Incidence</th>
<th>95% CI</th>
</tr>
</thead>
</table>

**Subtotal (95% CI)**

<table>
<thead>
<tr>
<th>Effect Size</th>
<th>95% CI</th>
<th>Number of Teeth</th>
<th>Caries Incidence</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
<td>Not estimable</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Heterogeneity: Not applicable
Test for overall effect: Not applicable

**Total (95% CI)**

<table>
<thead>
<tr>
<th>Effect Size</th>
<th>95% CI</th>
<th>Number of Teeth</th>
<th>Caries Incidence</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>-0.96</td>
<td>0.22</td>
<td>2,727</td>
<td>2,014</td>
<td>100.0%</td>
</tr>
</tbody>
</table>

Heterogeneity: $I^2 = 1.10; X^2 = 47.84, P < .00001; f = 81$

Test for overall effect: $z = 0.85$ ($P = .39$)

Test for subgroup differences: $X^2 = 1.69, P = .19; f = 40.9$

---

**Figure 7.** Forest plot of comparison 3. Overall: Glass ionomer sealants versus resin-based sealants, outcome: 3.1 caries incidence (2-3 years). CI: confidence interval. IV: Inverse-variance. SE: Standard error.
<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Log (Odds Ratio)</th>
<th>SE</th>
<th>Glass Ionomer Total</th>
<th>Resin-Based Total</th>
<th>Weight</th>
<th>Odds Ratio IV, Random, 95% CI</th>
<th>Odds Ratio IV, Random, 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.2.1 Mixed</td>
<td>-1.152</td>
<td>0.862</td>
<td>21</td>
<td>28</td>
<td>34.0%</td>
<td>0.32 (0.06-1.71)</td>
<td></td>
</tr>
<tr>
<td>Subtotal (95%/0 CI)</td>
<td>21</td>
<td>28</td>
<td>100.0%</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heterogeneity: Not applicable</td>
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</tr>
<tr>
<td>Test for overall eff·ed: z = 1.34 (P = .18)</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>I.2 Mixed</td>
<td>0.152</td>
<td>0.619</td>
<td>56</td>
<td>56</td>
<td>66.0%</td>
<td>0.41 (0.12-1.37)</td>
<td></td>
</tr>
<tr>
<td>Subtotal (95%/0 CI)</td>
<td>56</td>
<td>56</td>
<td>100.0%</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heterogeneity: Not applicable</td>
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</tr>
<tr>
<td>Test for overall eff·ed: z = 1.45 (P = .15)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Total (95% CI)</td>
<td>61</td>
<td>84</td>
<td>100.0%</td>
<td></td>
<td></td>
<td>0.37 (0.14-1.00)</td>
<td></td>
</tr>
<tr>
<td>Heterogeneity: C=0.00; $X^2=0.06, P=.81; r =0$%</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test for overall eff·ed: z = 1.96 (P = .05)</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test for subgroup differences: X =0.06, P=.81; r = 0%</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

eFigure 8: Forest plot of comparison. 3. overall: Glass ionomer sealants versus resin-based sealants, outcome: 3.2 caries incidence (4-7 years). CI: confidence interval. IV: Inverse-variance. SE: Standard error.
The American Dental Association Caries Classification System for Clinical Practice

A report of the American Dental Association Council on Scientific Affairs

Douglas A. Young, DDS, DMD, MEA, MD, Brian S. Novy, DDS, Gregory G. Zeller, DDS, MS, Robert Hale, DDS, Thomas C. Hart, DDS, PhD, Edmond L. Trueblood, DDS, MSD, American Dental Association Council on Scientific Affairs

**ADA Caries Classification System.**

<table>
<thead>
<tr>
<th>ADA CARIES CLASSIFICATION SYSTEM</th>
<th>Clinical Presentation</th>
<th>Other Lesions</th>
<th>Appearance of Occlusal Surfaces (Pit and Fissure)*</th>
<th>Accessible Smooth Surfaces, Including Cervical and Root*</th>
<th>Radiographic Presentation of the Approximal Surface*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sound</td>
<td>No clinically detectable lesion. Dental hard tissue appears normal in color, translucency, and gloss.</td>
<td>None</td>
<td>Unlikely</td>
<td>E0*</td>
<td>No radiolucency</td>
</tr>
<tr>
<td>Initial</td>
<td>Earliest clinically detectable lesion compatible with mild demineralization. Lesion limited to enamel or to shallow demineralization of dentin/enamel. Mild lesions are detectable only after drying. When established and active, lesions may be white or brown and enamel has lost its normal gloss.</td>
<td>Visually noncavitated</td>
<td>Established, early cavitated, shallow cavitation, microcavitation</td>
<td>E1*</td>
<td>Radiolucency may extend to the dentin-enamel junction or outer one-third of the dentin. Note: radiographs are not reliable for mild occlusal lesions.</td>
</tr>
<tr>
<td>Moderate</td>
<td>Visible signs of enamel breakdown or signs that the dentin is moderately demineralized.</td>
<td>Unlikely</td>
<td>Unlikely</td>
<td>E2*</td>
<td>Radiolucency extends into the middle one-third of the dentin.</td>
</tr>
<tr>
<td>Advanced</td>
<td>Enamel is fully cavitated and dentin is exposed. Dentin lesion is deeply/ severely demineralized.</td>
<td>Possible</td>
<td>Present</td>
<td>E3*</td>
<td>Radiolucency extends into the inner one-third of the dentin.</td>
</tr>
</tbody>
</table>

Dental caries remains a common chronic disease and, if not treated, it may progress until the tooth is destroyed. Despite advances in restorative materials and the implementation of various preventive approaches, more than 80% of adults in the United States have experienced at least one dental caries episode before age 30 years of age.

Dental caries is a multifactorial disease involving many complex risk and protective factors. The clinical presentation of caries disease is a caries lesion; the severity of the disease and of individual caries lesions is the result of complex personal, biological, behavioral, and environmental factors. Some factors are protective, such as the presence of fluoride in the biofilm, whereas others lead to hard tissue destruction, such as a lower plaque pH. Caries risk assessment is the organized process of evaluating these protective and pathogenic factors and provides the foundation for selecting treatment interventions.

The dental profession continues to implement a more integrative, noninvasive, therapeutic model to prevent, treat, and reverse caries lesions, particularly in the early stages. Despite progress, the profession still faces challenges in caries lesion detection and management.

This article has an accompanying online continuing education activity available at http://jada.org/ce. Copyright © 2015 American Dental Association. All rights reserved.
Sound  Initial  Moderate  Advanced

SEAL  RESTORE
“Dentists should [aim] for lesion arrest / inactivation to preserve dental hard tissues, avoid initiation of the restorative cycle and retain the tooth for as long as possible (strong recommendation)”
Indigenous microflora protect the host from exogenous pathogens by stimulating a vigorous immune response.
“...agents designed to produce a more favorable ecologic balance may ultimately be the most effective in controlling caries...”
Caries Risk Assessment works because of the identification of risk factors, patient behavior change, & risk-appropriate recare.
Transition to Caries Risk Conversations!
Begin by asking yourself...

- Is caries active...
- Is there one lesion?
  - An initial lesion
  - A moderate lesion
  - An advanced lesion
- Are there more than six filled surfaces?
Then it’s time for motivational interviewing…

- Where do you live?
- What’s your favorite candy?
- What’s next to your bed?
The inherent issue with CRA...

“How many times a day do you eat or consume: hard candies, sticky candies, cough drops, throat lozenges, cookies, cakes, pastries, doughnuts, fig newtons, tortilla chips, potato chips, Cheetos, crackers, bread, jams, jellies, marmalades, preserves, fruit juice, soda, pop, sweet tea, diet soda, or any other food that causes cavities?”
Correlations of oral bacterial arginine and urea catabolism with caries experience


Background: Alkaline generation by oral bacteria plays a key role in plaque pH homeostasis and may be a major determinant in the development of dental caries. To determine if the capacity of oral samples to produce ammonia from arginine or urea was related to caries experience, the arginine deiminase system (ADS) and urease activity in saliva and dental plaque samples were measured in 45 adult subjects.

Methods: The subjects were divided into three groups according to caries status: 13 non-caries (CF) individuals (dental, missing, and filled teeth = 0; 21 caries-active (CA) individuals (dental = 0 and 4); and 11 caries-experienced (CE) individuals (dental teeth = 0, missing and filled teeth > 2). Real-time polymerase chain reaction was used to quantify the proportion of certain acid- or alkaline-producing organisms in the samples.

Results: The amount of ammonia generated from the test substrates by plaque samples was generally higher than that produced by saliva samples in all groups. Significantly higher levels of sublingual ADS activity and plaque urease activity were observed in CF subjects compared to CA subjects (P = 0.0096 and P = 0.014, respectively). The proportions of Streptococcus mutans from saliva and dental plaque of CA subjects were significantly higher than those from the CF group (P = 0.0137 and P = 0.0032, respectively). In the CA group, there was an inverse relationship between urease activity and the levels of S. mutans (P = 0.0001).

Conclusion: This study supports the theory that increased caries risk is associated with reduced alkaline-generating capacity of the bacteria colonizing the oral cavity, providing compelling evidence for further understanding of oral alkaline-generating bacteria in health and disease.

Oroalgic bacteria that colonize the teeth form dental plaque, a biofilm community that is in dynamic equilibrium with host defense and is generally compatible with the integrity of the host tissues (13, 16, 47). The reactions from oral health to oral disease, such as dental caries and periodontal disease, is characterized by compositional and metabolic changes in the substrates of oral biofilm (16, 65). In the case of Caries, frequent acidification of dental plaque favors the emergence of an acidoforesis and acidoinduction (acidic) microflora, central for maintaining streptococci and Leuconostoc spp. that are capable of rapidly forming dietary carbohydrates and lowering the pH to an extent where significant amounts of tooth demineralization can occur (12, 15, 19, 63, 64). Although production of acid by dental plaque is the direct cause of dental caries, its microbicore that increases in the proportions of acidic organisms appears to occur at the expense of species that are less acidogenic and generally associated with dental health (1, 10, 12, 13, 24), including Streptococcus anginosus and Streptococcus gordonii. Some of the low acidogenic organisms associated with dental health derive protection from plaque acidification by hydrolyzing urea or arginine to ammonia, both by expressing various enzymes or by the arginine deiminase system (ADS).
Eat your spinach and fruit;
Drink water and milk;
Snack on nuts and cheese!
Toothpaste FOAM can be powerful!

Oh great...
There is no greater public health achievement than water fluoridation!
Starting with ONE patient...

- Have a caries risk conversation then complete a CRA
- The patient’s risk is high or low
- Move through moderate risk to demonstrate a decrease in risk
- Attempt risk based recare intervals…
After a century and a half of neglect, disease control programs are finally gaining respectability as bona fide tools of dentistry. These seemingly new programs are incredibly similar to those introduced by Pannly in 1815. His well-grounded clinical belief that flossing between the teeth would prevent more decay than all the brushes and powders in the world never caught on. Without scientific evidence for its decision, dentistry shrugged off the use of floss for interproximal cleaning and became enamored with the impotent "brush after meals" approach to controlling disease. The terrible price paid by mankind for this nineteenth century mistaken decision is only beginning to be appreciated by dentists throughout the world.
It is a privilege to treat another human...
If you don’t enjoy it, you’re not doing it correctly.