Effect of time and duration of sorbitol gum chewing on plaque acidogenicity

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Abstract

Recent data indicate that salivary stimulation by chewing sugarless gum after snacks or meals can reduce the acidogenic potential of foods significantly. The purpose of this study was to determine the optimal initiation time and duration of post-snack salivary stimulation to obtain the maximum benefits of chewing sorbitol gum on reducing the acidogenic potential of starch-containing snacks. An indwelling plaque pH telemetry system was used on five adults in a randomized block design with four starch-containing snacks—pretzels, potato chips, granola bars, and corn chips. Results indicated that salivary stimulation caused by chewing sorbitol gum initiated after 5 min rather than waiting 15 min significantly reduced the acidogenic challenge induced by the snack foods. This study indicates that when the recommendation to chew sugarless gum following food ingestion is used as an adjunct in caries prevention, it should start within 5 min after food ingestion—the sooner the gum chewing is initiated the better—and should continue for at least 15 min to obtain the maximum benefits. (Pediatr Dent 15:197-202, 1993)

Introduction

A fermentable carbohydrate's concentration and the duration of its presence in the mouth determine the extent and time of the resulting drop in plaque pH.\(^1\) Mastication and gustation stimulate salivary flow, which affects, to varying degrees, the plaque pH responses resulting from fermentable carbohydrates.\(^2\)\(^-\)\(^4\) Chewing a peppermint-flavored, sorbitol-containing gum for 10 min has been shown to significantly increase the rate of salivary flow, pH, and buffering capacity of saliva.\(^5\) After eating snacks containing sucrose, the time during which the interproximal plaque pH remains below the critical level (i.e., pH at which dissolution of dental enamel occurs faster than remineralization) is shortened significantly by chewing sugarless gum.\(^6\)\(^-\)\(^7\) The effect of chewing sugarless gum on reversing the pH drop caused by sugar-containing snacks and on maintaining a nonharmful resting plaque pH for a sustained period after snack ingestion has been reviewed thoroughly.\(^8\) This review indicated that the results varied depending upon the experimental methods used. For both 5- and 10-min chewing periods, a sorbitol-containing gum and paraffin chewing were effective in altering the interproximal plaque pH after eating a jelly doughnut.\(^9\) Chewing sorbitol-containing gum for 10 min after ingesting predominantly sucrose-containing foods was also effective in neutralizing acid produced in interproximal plaque.\(^7\) Chewing gum containing sucrose caused a depression of plaque pH, while the pH rose to 7.5 when a "sugarless" gum was chewed.\(^8\)\(^-\)\(^11\)

These findings prompted us to examine some variables that could moderate the plaque pH phenomenon. In previous studies in our laboratory, the plaque pH response to starch foods had been somewhat more difficult to mediate than the response to sucrose foods.\(^12\) Because starch in foods may be important in contributing to cariogenicity\(^12\)\(^-\)\(^14\) and less responsive to increased salivary flow, we decided to use starch snacks in this study. The purpose of this study was to determine the effects on the plaque pH response to snack foods of: 1) the time interval between snack food ingestion and chewing a sorbitol gum, and 2) the duration of gum chewing. The plaque pH responses were measured by an interproximal indwelling, glass pH electrode telemetry system.

Methods and materials

The four starch-containing snack foods used as challenges in this study were pretzels, potato chips, corn chips, and granola bars. These snacks have been shown to promote a significant plaque pH response in previous animal and laboratory studies.\(^12\)\(^-\)\(^16\) The snacks were purchased locally and were provided to subjects in 10-g portions. After the nature of the procedure, possible discomforts, and risks had been fully explained, an informed consent was obtained from all participants. The selection criteria for the five panelists involved in this study were based on the considerations listed in the San Antonio consensus reports.\(^17\) Mean age of panelists participating in this study was 37.8 years with an age range from 25 to 46 years. Their average dental caries history in terms of DMFT and DMFS was 11.0 and 22.2, respectively.

An appliance containing an interproximal wire-telemetry pH sensor was prepared for a missing mandibular molar. The description of the indwelling plaque pH telemetry system has been reported previously.\(^12\)\(^-\)\(^18\) The participants were asked to refrain from oral hygiene for three days while wearing their prostheses and to fast for a minimum of 12 hr immediately prior to the morning of the test.
Daily Sequential Procedures

Wire-telemetry Connection
Pretest Rinse and Stabilization
Monitoring Resting Plaque pH Response
Test Snack Ingestion for 2-Minutes

Monitor Plaque pH for Remainder of 2 Hours (Baseline Session)

Monitor Plaque pH for Either 15 Minutes (Test 1) or 5 Minutes (Test 2)

Sorbitol Gum Chewing Either for 10 Minutes (Test 1) or 15 Minutes (Test 2)

Monitor Plaque pH for Remainder of 2 Hours (Gum Chewing Session)

pH calibration with two reference buffers (pH 7 and pH 4)

Fig 1. Flow chart.

The study design and test procedures are illustrated as a flow chart in Fig 1. The test began by connecting the indwelling electrode system to the wire telemetry system, a pretest rinsing with warm tapwater, stabilizing plaque pH response, and recording the baseline resting plaque pH for a 5-min period.

In this series of tests, the panelists ingested the designated test foods according to a randomized block design. The subjects were asked to chew one of the snack foods for 2 min while distributing the bolus evenly throughout their mouths and then to swallow. The participants were instructed that they should continue their normal swallowing behavior during the test. The plaque pH responses were monitored continuously for 2 hr, with pH values (millivolts) collected every 0.3 sec; each three consecutive millivolt readings were averaged and stored on a computer disk. After eating all four test foods, the panelists repeated the test following the same randomized block design except that, after the plaque response to the food was monitored for 15 min, they chewed a sorbitol-containing gum (Trident™, Warner-Lambert Co., Morris Plains, NJ) for 10 min (Test 1). At a later date, each panelist repeated the two regimens with the plaque pH response period after eating recorded for only 5 min before the gum was chewed for a 15-min period (Test 2).

At the end of each test session, panelists were asked to rinse thoroughly twice with a total of 50 ml tap water. A reference buffer solution (pH 7.0) was administered topically to the interdental space and the stabilized electrode response (mV/pH) was recorded. Panelists were asked again to rinse thoroughly with 50 ml tap water and the electrode response to a second reference pH buffer (pH 4.0) was recorded. The millivolt readings of these two reference buffers were used to transform the millivolt readings from the preceding test into pH values.

For data analyses, the following parameters were evaluated statistically: 1) resting baseline pH; 2) minimum plaque pH; 3) maximum plaque pH drop (baseline resting pH minus minimum pH attained); and 4) the integrated area below plaque pH of 5.5 during the test period. The area of the curve under pH 5.5 was derived using a computer-processed geometric integration and was defined as the area enclosed by the pH response on the X-axis (time) and a straight line across pH 5.5 on the Y-axis (pH scale); this value represents the hydrogen ion activity under the critical pH 5.5 as function of time. Means and standard errors for each test regimen (corresponding data points on the individual response curves) were calculated. A composite curve of the means, plus or minus one standard error, was constructed for each test regimen. Since baseline data (no gum chewing and resting pH) were obtained prior to each experimental period and were not significantly different between the two tests, they were
Table 1. Area of plaque pH curve (pH/time) under 5.5

<table>
<thead>
<tr>
<th>Test Snack</th>
<th>Baseline (No Gum)</th>
<th>Gum Chewing</th>
<th>Chewing Impact (% Reduction)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test 1</td>
<td>Test 2</td>
<td>Test 1</td>
</tr>
<tr>
<td>Pretzels</td>
<td>6995 ± 1735†</td>
<td>2559 ± 881</td>
<td>197 ± 165†</td>
</tr>
<tr>
<td>Potato chips</td>
<td>9822 1039</td>
<td>1583 836</td>
<td>539 537§</td>
</tr>
<tr>
<td>Granola bar</td>
<td>10447 1844</td>
<td>3710 2209</td>
<td>292 292</td>
</tr>
<tr>
<td>Corn chips</td>
<td>12069 ± 1220</td>
<td>7395 3171</td>
<td>4142 ± 1665</td>
</tr>
</tbody>
</table>

Test 1 — 5 min baseline pH; 2 min snack ingestion; 15 min wait; 20 min gum chewing; and 88 min monitor. Test 2 — 5 min baseline pH; 2 min snack ingestion; 5 min wait; 15 min gum chewing; and 93 min monitor.

† Mean ± SEM (n = 5).

§ Underlined values do not differ significantly (P > 0.05) as determined by Newman-Keuls tests.

Table 2. The effect of gum chewing on minimum plaque pH

<table>
<thead>
<tr>
<th>Test Snack</th>
<th>No Gum</th>
<th>Gum Chewing Regimen</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test 1</td>
<td>Test 2</td>
</tr>
<tr>
<td>Pretzels</td>
<td>4.53 ± 0.10†</td>
<td>4.89 ± 0.11†</td>
</tr>
<tr>
<td>Potato chips</td>
<td>4.43 0.05</td>
<td>4.90 0.17</td>
</tr>
<tr>
<td>Granola bars</td>
<td>4.48 0.25</td>
<td>4.90 0.22</td>
</tr>
<tr>
<td>Corn chips</td>
<td>4.01 ± 0.07†</td>
<td>4.48 ± 0.22</td>
</tr>
</tbody>
</table>

Test 1 — 5 min baseline pH; 2 min snack ingestion; 15 min wait; 10 min gum chewing; and 88 min monitor. Test 2 — 5 min baseline pH; 2 min snack ingestion; 5 min wait; 15 min gum chewing; and 93 min monitor.

† Mean ± SEM (n = 5).

Pediatric Dentistry: May/June, 1993~ Volume 15, Number 3 199
gum for 10 min starting 15 min after snack ingestion (Test 1) significantly reduced the area under pH 5.5 to 1583 units while the 15-min gum chewing period following a 5-min delay (Test 2) further reduced the integrated area below pH 5.5 to 559 units. Both of these reductions in response of gum chewing were significantly different from the baseline response without gum chewing.

The composite plaque pH response after eating a granola bar is illustrated in the third row (Fig 2). Again, the baseline plaque pH fell slowly as a function of time following snack ingestion (no gum), but by chewing gum for 10 min beginning 15 min after snack ingestion (Test 1), the critical area in plaque pH response below pH 5.5 was reduced significantly from the baseline area of 10,447 to 3710 units. Chewing sorbitol gum for 15 min beginning only 5 min after snack ingestion (Test 2) also significantly reduced the area under the critical pH 5.5 by approximately 98% from baseline, i.e., from 10,447 to 292 units (Table 1).

The composite baseline plaque pH response after eating corn chips is shown in the fourth series of graphs (Fig 2) and illustrates a slow fall of plaque pH as a function of time with a rise beginning near the end of the 2-hr test period. When the corn chips were followed 15 min later by chewing sorbitol gum for 10 min, a numerical (but not statistically significant) reduction in the area units of plaque pH below 5.5, i.e., from 12,069 to 7395, was observed. Chewing sugarless gum for 15 min beginning only 5 min after ingesting the snack food further reduced the critical area below pH 5.5 from an average baseline (no gum) area of 12,069 units to 4142 units.

Table 2 compares the minimum plaque pH after the two different gum-chewing regimens and the no-gum regimen for each of the snack foods. The resting (pretest) plaque pH values (not presented) were not significantly different among groups, ranging from averages of 6.9 to 7.0. Only the potato chip group had a significantly higher minimum pH following the shorter gum chewing session (Test 1); although the minimum pH values observed with the other three snack foods were elevated, the change in pH was not statistically significant. When the longer (15-min) gum-chewing time was used (Test 2), three of the four pH minimum values were raised significantly; only the increase observed in the corn chips regimen was not statistically significant.

Table 3 summarizes data regarding the maximum plaque pH drop from baseline resting plaque pH after eating the different snack foods and chewing gum regimens. Test regimen 1 resulted in a significant reduction in the plaque pH drop with only two of the snack foods (pretzels and potato chips), although numerical improvements were observed in all snack food regimens. Test regimen 2, however, provided a significant reduction in plaque pH drop from all four snack foods. In addition, test regimen 2 was significantly more effective than test regimen 1 in two of the four groups (pretzels and granola bars).

**Discussion**

It is apparent from this study that the stimulation of salivary flow by chewing sorbitol gum modifies the plaque pH responses to starch-containing snacks. Results also showed that the sooner and the longer sorbitol gum was chewed after eating, the greater the level of protection from acid attack. Rapid return of the plaque pH to resting pH levels may be attributed to an increase in salivary flow rate that could alter the food clearance from the mouth and/or an increase the salivary buffering capacity.

The pH-lowering effect of fermentable carbohydrates has been shown to be prevented when the flow of saliva is stimulated by chewing a palatable inert material such as paraffin wax. Quite recently it has been shown that the decrease in plaque acidity is due to a combination of increased salivary flow and increased buffering capacity attributable to salivary bicarbonate.

Although the critical pH at which enamel dissolution begins in the plaque environment is currently a subject of discussion, this level was considered to be 5.5 in our study. Harper et al. and Edgar and Geddes reviewed available information from the consensus conference and concluded that the critical pH varies among individuals and among sites within an individual. However, the critical pH has been defined as the hydrogen ion concentration at which any particular saliva stops being saturated with calcium and phosphate. The critical pH in the plaque environment varies according to the calcium and phosphate concentration in the plaque, but it is usually about 5.5.

Clinical and in situ studies have indicated a significant

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**Table 3. The effect of sorbitol gum chewing on maximum plaque pH drop (baseline resting pH — minimum pH attained)**

<table>
<thead>
<tr>
<th>Test Snack</th>
<th>Baseline (No Gum)</th>
<th>Gum Chewing Regimen Test 1</th>
<th>Gum Chewing Regimen Test 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretzels</td>
<td>2.41 ± 0.08 *</td>
<td>2.05 ± 0.08</td>
<td>1.29 ± 0.24</td>
</tr>
<tr>
<td>Potato chips</td>
<td>2.47 + 0.07</td>
<td>2.01 + 0.15</td>
<td>1.31 + 0.31</td>
</tr>
<tr>
<td>Granola bars</td>
<td>2.50 + 0.25</td>
<td>2.08 + 0.22</td>
<td>1.14 + 0.17</td>
</tr>
<tr>
<td>Corn chips</td>
<td>2.94 + 0.08</td>
<td>2.47 + 0.22</td>
<td>2.12 + 0.30</td>
</tr>
</tbody>
</table>

Test 1 — 5 min baseline pH; 2 min snack ingestion; 15 min wait; 10 min gum chewing; and 88 min monitor. Test 2 — 5 min baseline pH; 2 min snack ingestion; 5 min wait; 15 min gum chewing; and 93 min monitor.

* Mean ± SEM (n = 5).
† Underlined values do not differ significantly (P > 0.05) as determined by Newman-Keuls tests.
anticaries benefit from chewing sugarless gum. Chewing sorbitol gum significantly enhanced remineralization when used over a long period of time. These effects presumably were due to the stimulation of salivary flow and subsequent inhibition of enamel demineralization when the gum was chewed immediately after meals and/or snacks. Fast-flowing saliva is alkaline and contains adequate concentrations of minerals for enamel remineralization. Chewing sugarless gum is, therefore, very likely to favor enamel remineralization.

This study clearly confirmed these previously observed clinical and in situ benefits of chewing sorbitol gum after meals or snacks. A chewing regimen including a sorbitol gum chewed 5 min after snack food ingestion was more effective in reducing the acidogenicity of interdental plaque than a 10-min chewing regimen that began 15 min after food ingestion. The increased salivary flow induced by chewing gum causes a reversal of plaque pH and appears to neutralize the deleterious effect of acids produced by the challenge of fermentable carbohydrate snack foods.

In retrospect, it is unfortunate that the study design in the present investigation did not keep one of the two variables (chewing time or length of delay after snack ingestion before gum chewing) constant. As a result it is impossible to determine the relative importance of these two variables. With every snack food, the test regimen 2 resulted in a greater reduction in the foods' acidogenicity. However, the study design does not permit us to determine whether this is due to a reduced time period between snack food ingestion and initiation of gum chewing, i.e., 5 min vs 15 min, or a longer chewing period, i.e., 15 min vs 10 min, in the test regimen 2. It is quite likely that both factors contributed to the overall impact, but further studies are required to define the relative importance of these variables.

It is apparent that chewing sorbitol gum sooner and for a longer period of time after food ingestion reversed the plaque pH drop more effectively than did waiting a longer time period and chewing for shorter duration. The results of our study suggest that the hypothesized effect of increased salivary flow resulting in possible remineralization, increased oral clearance and increased plaque acid buffering. This indicates that chewing of sorbitol gum after eating starch-containing snacks is an alternative to toothbrushing in formulating dietary counseling procedures, but only if the latter is not possible. However, gum chewing should not replace toothbrushing. If chewing gum is recommended, the patient should be encouraged to use a sugarless gum within 5 min of eating and to continue chewing for at least 15 min. It is emphasized that this recommendation is based on acidogenicity, which is measured with an indwelling plaque pH telemetry system, and not actual cariogenicity, although these parameters are strongly correlated.

Conclusion

Decreasing the time between eating and sorbitol gum chewing along with an increased duration of the chewing period produced significant benefits in the control of acidogenicity of dental plaque after exposure to starch-containing snacks.

This study was supported by the Warner-Lambert company. The authors thank Dr. Ann J. Dunipace for her valuable help in preparing this manuscript.

17. Schachtele CF, Abelton D, Edgar WM, et al: Human plaque acid-
Treating lead poisoning in children may improve their IQ

**Three million kids have lead levels high enough to affect intelligence**

Lowering blood lead levels in moderately lead-poisoned children may help their intelligence over time, according to a study published in this week's *Journal of the American Medical Association*.

"The results suggest an association between decreases in blood lead level and cognitive improvements in moderately lead-poisoned children," writes Holly A. Ruff, PhD, from the Department of Medicine, Albert Einstein College of Medicine, Bronx, N.Y., with colleagues.

The authors report on 154 previously untreated children who had blood lead levels between 25 and 55 mg/dL of blood (10 mg/dL can affect intelligence and development).

The children ranged from 13 to 87 months old; 58% were Hispanic, 37% were black; and 57% were boys. Data taken at study initiation suggest the children were "disadvantaged and at risk for developmental delays."

Medical interventions were chelation therapy (treatment to rid the body of lead) and/or iron supplements if the child was iron deficient. Children received one or both of these therapies. All children had home inspection to eliminate exposure to lead-based paint.

"In the short term (7 weeks), changes in blood lead levels were not related to changes in cognitive scores," they report. "In the long term (6 months), however, changes in performance were significantly related to changes in blood lead levels...The standardized score increased 1 point for every 3 mg/dL in blood lead levels."

They note: "It is not impossible that some unmeasured variable caused independent but parallel changes in [blood lead] level and cognitive performance, and we must remain cautious in making casual attributions. These data are consistent, however, with the presence of an association between cognitive changes and changes in lead levels."