

Raisins and Oral Health

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Abstract: Traditionally, raisins have been thought to promote dental caries due to their suspected “stickiness” and sugar content. Current research identifies some evidence contrary to traditional thought, suggesting that raisins may not contribute to dental caries. This article reviews new findings with regards to raisins and the 3 conditions that are thought to contribute to the formation of dental caries; low oral pH, adherence of food to teeth, and biofilm (bacterial) behavior. The studies reviewed concluded that raisin: consumption alone does not drop oral pH below the threshold that contributes to enamel dissolution, do not remain on the teeth longer than other foods, and contain a variety of antioxidants that inhibit *Streptococcus Mutans*, bacteria that is a primary cause of dental caries. Further research in this area should be considered.

Keywords: adenosine triphosphate, antioxidant, ATP, biofilm, bioluminescence, dental caries, pH, raisins

Introduction

Raisins have a long-standing reputation as a food that promotes dental caries (cavities). The suspected mechanism was raisins' adherence to the teeth and high sugar content. Studies conducted in the 1950s to 1990s found that raisins may promote dental caries and their consumption has been discouraged ever since. For example, in a study on rats, raisins were among test foods that have a moderate to high cariogenic (cavity causing) potential; therefore, raisins should not be “used indiscriminately as a frequent snack without appropriate oral hygiene” (Mundorff and others 1990, p 352). Past studies also show some correlation between increased dental caries risk among children who include raisins in their diet (Parajas 1999). Additionally, peer-reviewed and popular press articles have specifically identified or listed raisins among the foods to avoid for prevention of cavity formation, due to these past studies and popular perceptions (Lewis 1992; Bosco and Higbee 1993; Faine and Oberg 1995; Lee 1995; Pilar 2002; Healthy Snacks Lead to Healthy Smiles: Some Tips on Kids' Treats for National Nutrition Month 2005; Blevins 2011). Recent evidence has shown that these claims are not substantiated by human clinical trials.

The etiology of the dental caries is multifactorial in nature and diet and nutrition are very important elements (Touger-Decker and van Loveren 2003). Diet and its nutritional consequences can have a profound influence on tooth development, as well as on the development and progression of diseases of the oral cavity (Varoni and others 2012). Three conditions are known to promote the formation of dental caries; low oral pH, adherence of food to teeth, and biofilm (bacterial) behavior. Although in

the past, raisins have been perceived as cariogenic, more recent evidence casts some doubt on the role of raisins with regards to tooth decay. Although the current research provides a small amount of evidence that raisins may provide some protective benefits against dental caries, more research is warranted to validate this claim. The purpose of this review is to discuss recent information on how raisins affect the 3 oral conditions that promote dental caries and provide a case that raisin consumption may not contribute to the development of dental caries as traditionally thought.

Raisins and Oral Acidogenicity

Dental caries disease, a transmissible disease, can lead to damage to the teeth that result in tooth decay or cavities. There are a number of variables that can contribute to the formation of cavities. One of these variables is an acidic oral environment. The tooth surface is covered with a protective pellicle and biofilm layer that is attached to the pellicle. Cavities result when the acid production by cariogenic (acid-producing) bacteria present in the biofilm diffuses into the tooth and dissolves the enamel causing a cavitation (cavity or hole) in the tooth surface. The bacteria that contribute to dental caries are aciduric (able to live in an acid environment) and acidogenic (able to produce acid) (Marsh 2006; Kutsch and Young 2011). These acid producing bacteria consume fermentable carbohydrates, especially sucrose, which is converted to acid (Luke and others 1999). The acid production then supports a drop in oral pH that, in turn, favors the growth of more pathogenic (cavity causing) bacteria and suppresses the beneficial healthy bacteria (Marsh 2006; Kutsch and Young 2011). If the increase in acid production drops the pH below the critical threshold of about 5.5 enamel demineralization will result (Luke and others 1999; Wu 2009). Frequent snacking and foods rich in certain sugars are variables that contribute to more mineral loss and an acidic oral environment that favors the caries pathogens (Marsh 2006; Kutsch and Young 2011).

In order to determine the effects of raisins and oral acidogenicity, Utreja and others (2009) conducted a randomized control study of 20 children between the ages of 7 to 11 y. The researchers examined the effect of 4 test foods; raisins, bran flakes, commercial

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raisin bran cereal (cRB), and experimental raisin bran cereal with no added sugar (eRB), on plaque acidogenicity. Sucrose (10%) and sorbitol solutions were used as positive and negative controls. The researchers ranked the test foods in promoting plaque acidogenicity from highest to lowest as follows: commercial raisin bran cereal > bran flakes > raisins > experimental raisin bran cereal (Figure 1). After the consumption of raisins or experimental raisin bran cereal with no added sugar, the dental plaque pH never dropped below 6.0, and thus never reached the critical pH point of 5.5 that is thought to be necessary to demineralize enamel. Sugar profiles of test foods were also determined. The raisins contained 68% sugar, which was the highest among all the test foods, yet had the least acidogenic effect compared to cRB, bran flakes, and 10% sucrose solution. Additionally, it was noted that raisins aided in clearing bran flakes from the mouth, which was one of the most acidogenic test foods. They concluded that, sweet as they are, raisins do not contribute to an acidogenic effect in the mouth and are rapidly cleared from the mouth (Utreja and others 2009).

One suggested explanation was that although raisins are high in sugar, they may not have reduced oral pH because raisins contain mainly glucose and fructose, but not sucrose that serves as the main substrate for the synthesis of human dental plaque (Cury and others 2000; Rivero-Cruz and others 2008). Although the type of sugar may play a role in the formation of dental caries, Marsh (2006) has proposed different explanation. Marsh (2006) experiment showed that it was the low pH, not the sugar per se, that selects for pathogenic bacterial behavior. Based on this theory, individuals' pH determines the bacterial makeup and behavior (the extent to which they can produce acid) and is a key factor that should be considered when reviewing the literature cited previously. In other words, certain foods may be high in sugar but if the bacteria of the individual are not able to produce acid, then there will not be a drop in pH and no demineralization of teeth will occur.

Because fruit and fruit juices are a large part of the American diet and their intake is suggested for overall health, Issa and others (2011) sought to compare the *in situ* effect of enamel demineralization, caused by their acidogenic potential, from fruits and vegetables consumed in whole and juiced form. Subjects were assigned to a regime of 7 times per day consumption of either one of the test foods/drinks or controls for a period of 10 d. Test foods consisted of apples, oranges, grapes, carrots, and tomatoes consumed whole or as a juice. Raisins were also used since they can be consumed in whole form. Positive and negative controls consisted of 10% sucrose and 10% sorbitol. Subjects wore removable mandibular appliances that contained human enamel slabs

that had artificial lesions already created *in vitro*, and were worn continuously, except when eating or drinking foods other than the test food/drinks. Different enamel slabs were used in each condition and the thickness (demineralization) of the appliance was measured through a technological process using transverse microradiography, which also measures mineral content, mineral changes, and mineral distributions. Results showed demineralization with raisins, tomato, tomato juice, apple, apple juice, orange, orange juice, carrot and carrot juice ($P < 0.01$), grape, grape juice as well as the positive control sucrose ($P < 0.001$). There was no statistically significant demineralization with sorbitol. There were no significant differences between the test products when consumed either whole or in juiced form. Along with the other test foods, raisins resulted in enamel demineralization, but they were found to have the one of the lowest mean mineral losses among the other test foods, and were much lower when compared to 10% sucrose (raisins 1007.88 compared to 10% sucrose 1534.88).

Adherence of Raisins on Teeth

Another variable that may contribute to dental caries is adherence of foods to the surface of the teeth. Cariogenic effects of food are also related to the retention time of the food particles that remain trapped on the teeth (Luke and others 1999). According to Kashket and others (1991), particles that become trapped on the surfaces of teeth are considered to contribute to the development and progression of dental caries. These retained particles serve as reservoirs of fermentable carbohydrates and permit plaque microorganisms to continue to produce acids and prolong the cariogenic environment. Kashket and others (1991) reported that a number of factors contribute to adherence of foods such as adhesiveness, chewiness, viscosity, and moisture content. Additionally, the degree to which a food is retained may depend on factors such as salivary flow rates, tongue movements, chewing and swallowing patterns, and individual tooth anatomy (Kashket and others 1991).

Traditionally, raisins have been thought of as promoting caries disease (tooth decay) due to their suspected "stickiness." However, Kashket and others (1991) suggested that there is little correlation between perceived stickiness and actual retention of food particles on the teeth. In order to determine this, researchers examined the relationship between consumer evaluation of stickiness and the actual amount of food particles retained on the teeth for 21 commercially available foods, raisins were included. To determine stickiness, 315 consumers over 18 y of age were randomly chosen from shopping malls in 8 cities. Subjects were asked to rate each food on a 1 to 9 scale from sticky to not sticky.

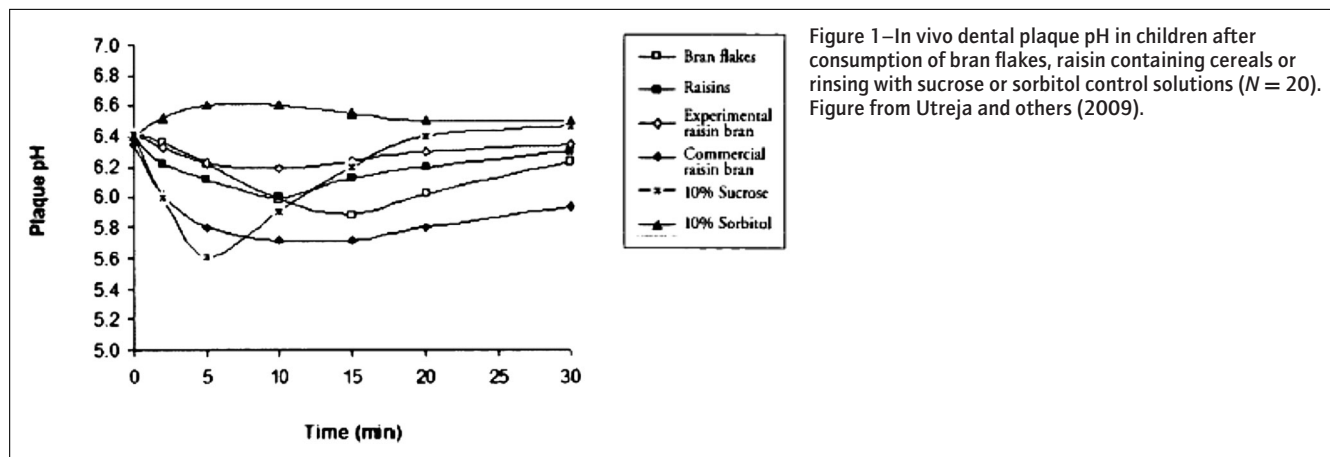


Figure 1—In vivo dental plaque pH in children after consumption of bran flakes, raisin containing cereals or rinsing with sucrose or sorbitol control solutions ($N = 20$). Figure from Utreja and others (2009).

To determine food retention, 5 subjects consumed all 21 foods on a random basis. Researchers assessed retention at 1, 2, and 5 min after swallowing. They found that the rates of clearance of food particles from the teeth were not correlated with ratings of food stickiness. Subjects ranked raisins 9th out of the 21 foods for stickiness and were perceived to be as sticky as granola bars, crème sandwich cookies, and oatmeal cookies, yet they were 14% less retentive than these foods (Kashket and others 1991; Bell 2011). In contrast to these findings, Utreja and others (2009) observed that raisins are rapidly cleared from the mouth, are less retentive on tooth surfaces, and aid in clearing high-acidogenic foods.

Inhibitory Effects of Raisins on Bacteria

Cavities can also result from bacterial imbalance. In the past decade, there has been a significant change in thought on the bacterial etiology of dental caries disease. With regard to human teeth, differing amounts of bacteria are present in the biological makeup of the biofilm that covers the teeth. Bacteria are acquired most likely from the child's caregiver at a very young age and the makeup of organisms is in a constant evolution depending on the local environment in which they live (Marsh 2006). Bacteria in the biofilm can become more pathogenic in response to environmental changes that favor acids (Takahashi and Nyvad 2008).

The multifactorial etiology of dental caries disease includes bacteria such as *Streptococcus mutans* and *Lactobacillus* (LB); however, the current biofilm disease model for caries disease is one of multiple pathogens. *Streptococcus Mutans* has previously been found to play an important role in the development of dental caries, which is a contributing factor in the decline of dental health (Hamada and Slade 1980; Loesche 1986; Wu 2009). As previously mentioned, dental caries is a pH-specific disease. However, Takahashi and Nyvad (2008) also determined that even nonaciduric and nonacidogenic organisms that are usually associated with dental health can evolve to become aciduric and acidogenic if placed in a low pH environment. In other words, given enough time in a low pH environment, even bacteria considered to be "good bacteria" can adapt to live in an acid environment and even create acid themselves. In order to effectively treat caries, it is important to restore the biofilm to health, as well as balance the pH of the oral environment.

Many researchers have conducted studies using functional foods, such as green tea and cloves, as a method to reduce oral pathogens and benefit oral health (Cai and Wu 1996; Li and others 1997). In order to continue the discovery of oral antibacterial compounds from plants, Rivero-Cruz and others (2008) extracted 8 known compounds from Thompson seedless raisins and evaluated them for their antibacterial activity against the oral pathogen, *Streptococcus mutans*. These compounds consisted of oleanolic acid, oleanolic aldehyde, linoleic acid, linolenic acid, betulin, betulinic acid, 5-(hydroxymethyl)-2-furfural, and b-sitosterol. Oleanolic acid, oleanolic aldehyde, 5-(hydroxymethyl)-2-furfural, and b-sitosterol were active against *S. mutans* (0.0078 to 0.0625 mg/mL). Overall results showed that oleanolic aldehyde and 5-(hydroxymethyl)-2-furfural found in raisins may benefit oral health since they possess the antimicrobial property that suppress growth of oral bacteria associated with dental diseases (Rivero-Cruz and others 2008).

Other researchers suggest that raisins contain phenolic acids or flavonoids that may suppress by antioxidant activity oral pathogens associated with caries disease and are thus hypothesized to benefit oral health (van Loveren and others 2012).

Raisins Reduce Adenosine Triphosphate (ATP)

A new methodology has been developed to determine the caries pathogenicity of biofilm by measuring ATP activity. What is common to aciduric organisms is an ATP-driven pump in their cell membranes to actively pump out hydrogen ions from the cytoplasm (Len and others 2004). Without this proton pump, the bacteria's cell will acidify and cause its death.

Recently, the dental industry has developed a device to identify ATP activity of biofilms (Fazilat and others 2010). The concept of ATP bioluminescence testing is to measure light energy emitted from the chemical reaction between bacteria-produced ATP and luciferin (in the reagent) to form luciferyl adenylate that is oxidized by the luciferase and emits photons of light that can be detected in a luminometer. It is important to note that the ATP being measured is a numeric reading measured as relative light units (RLUs). High amounts of RLU (>1500 RLU) are indicative of acid activity of bacteria, but does not identify specific species of bacteria per se (Pellegrini and others 2009).

In dentistry, ATP-testing has been modified to test plaque samples from a patient's teeth. The swab that gathers the sample on a sterile cotton tip housed in a self-contained tube. The cotton tip collects a plaque sample from 6 tooth surfaces. Once the plaque sample is taken, the cotton-tipped swab is placed back in its tube and the reagent luciferin is released and mixed in for 15 s. The tube is then placed in the ATP meter for 15 s and a numeric RLU reading appears. The cutoff number is 1500 RLUs; above this number, it is considered to indicate high biofilm caries activity (Pellegrini and others 2009).

ATP bioluminescence (biofilm caries activity) readings for the use of prediction of dental caries risk were sampled in a pilot study at the 2011 World Games in Athens, Greece with the Special Olympics population (Wu 2011). The clinical data were based on the hypothesis that ATP measurements have a strong statistical association with bacterial number in plaque and saliva specimens, including numbers for oral streptococci, and may be used as a potential assessment tool for oral hygiene and caries risk in children (Fazilat and others 2010.)

The majority of patients with special needs are high to extreme in dental caries risk and periodontal disease risk; therefore, the goal of this project was to lower their risk both through dental advice and nutritional counseling. The purpose of this pilot study was to determine if the risk for dental caries among Special Olympics athletes increases, decreases, or remains the same after eating raisins. A total of 156 athletes elected to be swabbed and completed the study. The 6 tooth surfaces were swabbed for plaque samples, and using the ATP bioluminescence meter, the RLU was determined. Upon having a reading above 1500 RLUs, significant for high biofilm caries activity, the athlete was asked to consume a 1-oz package of California raisins with no water. After raisin consumption, researchers waited 15 min and swabbed 6 different teeth in another area of the mouth to determine the RLU. A control group participated in the same protocol, however, instead of consuming raisins, the subjects waited for 15 min without consuming any food or drink and were then retested for an RLU score (Wu 2011).

Of those in the experimental group, the average RLU score prior to consuming raisins was 5949. After raisin consumption, the RLU score dropped to 3356, which is a 43% reduction. In the control group, the initial RLU score was 6141. After the 15 min waiting period, the RLU score was 6131 RLU, which is less than a 1% difference. Although a reduction was seen between the experimental and control group, the RLU score was still

higher than 1500 RLU value that indicates dental caries risk. Calculations of the average values seem to support the theory that raisins do not increase the caries disease rate because a decrease in RLU was seen among the experimental group (Wu 2011). Future research is needed to determine if there is a rebound effect or sustaining effect with raisin consumption and RLU values.

Conclusion

This review of the literature suggests that raisins may not be cariogenic as once thought, and they may contain antibacterial properties that may reduce oral pathogens that contribute to dental diseases. Although raisins are sweet and are considered “sticky,” some research may imply that they do not adhere to the teeth long enough to promote dental caries formation and may help clear other cariogenic sugars from the tooth surface. Additionally, an individual’s pH and bacteria make up, not the sugar in raisins, may select for the acidogenicity potential of the biofilm, and raisins may not be a food that contributes to this unique acidic oral environment. Furthermore, the recent pilot study assessing a new methodology measuring ATP levels corroborates previous findings that raisins do not contribute to dental caries formation. More studies are needed to validate these theories. Due to limited research on raisins and oral health benefits, their potential to contribute to oral health is just now being realized and should be further investigated. Although the current research provides a small amount of evidence that raisins may provide protective benefits against dental caries, more research is warranted to make this claim. The amount of raisins needed for an effect needs to be evaluated. The study design must account for the individual patient’s biofilm pathogenicity. If raisins are shown to be beneficial to oral health, it could be a snack that deserves further consideration.

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References

- Bell S. 2011. Here come the raisins. *RDH-Regist Dent Hyg* 31(3):56–63.
- Blevins JY. 2011. Oral health care for hospitalized children. *Pediatr Nurs* 37(5):229–235.
- Cai L, Wu CD. 1996. Compounds from *Syzygium aromaticum* possessing growth inhibitory activity against oral pathogens. *J Nat Prod* 59(10):987–90.
- Cury JA, Rebelo MAB, Del Bel Cury AA, Derbyshire MTV, Tabchoury CPM. 2000. Biochemical composition and cariogenicity of dental plaque formed in the presence of sucrose or glucose and fructose. *Caries Res* 34(6):491–7.
- Bosco D, Higbee B. 1993. Raising cavity free kids. *Prevention*. 45(5):113–115.
- Faine MP, Oberg D. 1995. Survey of dental knowledge of WIC nutritionists and public health dental hygienists. *J Am Diet Assoc* 95:190–4.
- Fazilat S, Sauerwein R, McLeod J, Finlayson T, Adam E, Engle J, Gagneja P, Maier T, Machida CA. 2010. Application of adenosine triphosphate-driven bioluminescence for quantification of plaque bacteria and assessment of oral hygiene in children. *Pediatr Dent* 32(3):195–204.
- Hamada S, Slade HD. 1980. Biology, immunology, and cariogenicity of *Streptococcus mutans*. *Microbiol Rev* 44(2):331–84.
- Healthy Snacks Lead to Healthy Smiles: Some Tips on Kids’ Treats for National Nutrition Month. 2005. PR Newswire
- Issa AI, Toumba KJ, Preston AJ, Duggal MS. 2011. Comparison of the effects of whole and juiced fruits and vegetables on enamel demineralisation in situ. *Caries Res* 45(5): 448–52.
- Kashket S, Van Houte J, Lopez LR, Stocks S. 1991. Lack of correlation between food retention on the human dentition and consumer perception of food stickiness. *J Dent Res* 70(10):1314–9.
- Kutsch VK, Young DA. 2011. New directions in the etiology of dental caries disease. *J Calif Dent Assoc* 39(10):716–21.
- Lee Y. 1995. Sneaky snacks. *Prevention* 47(10):52.
- Len AC, Harty DW, Jacques NA. 2004. Stress-responsive proteins are upregulated in *Streptococcus mutans* during acid tolerance. *Microbiology* 150(5):1339–51.
- Lewis R. 1992. The bugs within us. *FDA Consum* 26(7):37–42.
- Li XC, Cai L, Wu CD. 1997. Antimicrobial compounds from *Ceanothus americanus* against oral pathogens. *Phytochemistry* 46(1):97–102.
- Loesche WJ. 1986. Role of *Streptococcus mutans* in human dental decay. *Microbiol Rev* 50(4):353–80.
- Luke G, Gough H, Beeley J, Geddes D. 1999. Human salivary sugar clearance after sugar rinses and intake of foodstuffs. *Caries Res* 33(2):123–9.
- Marsh PD. 2006. Dental plaque as a biofilm and a microbial community—implications for health and disease. *BMC Oral Health* 6(Suppl 1):S14.
- Mundorff SA, Featherstone JDB, Bibby BG, Curzon M EJ, Eisenberg AD, Espeland MA 1990. Cariogenic potential of foods. *Caries Res* 24(5):344–55.
- Parajas IL. 1999. Sugar content of commonly eaten snack foods of school children in relation to their dental health status. *J Philipp Dent Assoc* 51(1):4–21.
- Pellegrini P, Sauerwein R, Finlayson T, McLeod J, Covell Jr DA, Maier T, Machida CA. 2009. Plaque retention by self-ligating vs elastomeric orthodontic brackets: quantitative comparison of oral bacteria and detection with adenosine triphosphate-driven bioluminescence. *Am J Orthod Dentofacial Orthop* 135(4):426.e1–9.
- Pilar N. 2002. Sugar hurts tooth enamel (Medical Mailbox). *Saturday Evening Post* p91.
- Rivero-Cruz JF, Zhu M, Kinghorn AD, Wu CD. 2008. Antimicrobial constituents of Thompson seedless raisins (*Vitis vinifera*) against selected oral pathogens. *Phytochem Lett* 1(3): 151–4.
- Takahashi N, Nyvad B. 2008. Caries ecology revisited: microbial dynamics and the caries process. *Caries Res* 42(6):409–18.
- Touger-Decker R, Van Loveren C. 2003. Sugars and dental caries. *Am J Clin Nutr* 78(4):881S–92S.
- Utreja A, Lingstrom P, Evans CA, Salzmann LB, Wu CD. 2009. The effect of raisin-containing cereals on the pH of dental plaque in young children. *Pediatr Dent* 31(7): 498–503.
- Varoni ME, Lodi G, Sardella A, Carrassi A, Iriti M. 2012. Plant polyphenols and oral health: old phytochemicals for new fields. *Curr Med Chem* 19(11):1706–20.
- van Loveren C, Broukal Z, Oganessian E. 2012. Functional foods/ingredients and dental caries. *Eur J Nutr* 51(2):15–25.
- Wu CD. 2009. Grape products and oral health. *J Nutr* 139(9):1818S–23S.
- Wu CD. 2011. Bioluminescence pilot study. Unpublished data