CONSUMPTION OF CARBONATED DRINKS AS A RISK FACTOR FOR DENTAL CARIES AND DENTAL EROSION

A Review of Current Evidence

Aleksandra Jokovic, Preeti Prakash and David Locker

COMMUNITY DENTAL HEALTH SERVICES RESEARCH UNIT

HEALTH MEASUREMENT AND EPIDEMIOLOGY REPORT NO. 20

August 2004

ABSTRACT

In the last few decades, a significant increase in the intake of carbonated drinks among children and adolescents has occurred. These drinks are characterized by high sugar content and low pH value. Considering the role of fermentable carbohydrates and acids in the pathogenesis of dental caries and dental erosion, there is concern that the consumption of these drinks represents a risk for both these conditions in the population aged 0-18 years. The report reviewed the epidemiological evidence concerning this relationship. A search of the dental and medical literature in Medline and Cinahl databases for the period January 1996-March 2004 was conducted to identify relevant studies. Following the inclusion/exclusion criteria, four and ten papers pertaining to dental caries and dental erosion (respectively) were selected. Quality assessment of the papers retained for the review involved evaluation of the study design, sample characteristics, exposure and outcome measurement, controlling for confounders and data analysis. Due to the differences in the study designs, characteristics of the study populations, research and clinical heterogeneity, as well as flaws in the methodology and/or reporting of the studies, the quality of the evidence is weak and equivocal. Although the studies reviewed do not provide an answer as to whether carbonated drinks are a risk factor for the occurrence and/or progression of either dental caries or dental erosion, they suggest the possibility of the existence of such a link. If the link does exist, longitudinal studies involving the standardized measurements of exposure and controlling for potential confounders are necessary to obtain information for establishing its cause-effect relationship.

TABLE OF CONTENTS

1.0 INTRODUCTION

Dental caries results from the dissolution of tooth enamel caused by acids produced from the action of oral bacteria on dietary fermentable carbohydrates (sugars). If not stopped, this mechanism leads to the demineralization of the dentin followed by the enzymatic decomposition of its organic matrix.

Enamel solubility and the length of exposure to acids play important roles in the carious process. The first depends primarily on pre-eruptive ("systemic") and post-eruptive ("topical") fluoride exposure. The latter is determined by the interplay between oral hygiene habits and the pattern (i.e. frequency, amount and form) of the consumption of sugars. The carious process is also influenced by other factors (Appendix 1). They are numerous and diverse and exert their influence in a complex web of causation underlying dental caries.

Dental erosion is the loss of dental hard tissues induced by acids that do not result from the involvement of oral bacteria but are rather of intrinsic (gastrointestinal) or extrinsic (dietary, medicamentous, environmental) origin.[1] The pathogenesis of this condition is influenced by factors described for the carious process as well as those shown in Appendix 2. It is also co-factored by and occurs in unison with the other types of tooth wear: attrition, abrasion and abfraction (Appendix 3). Their differential diagnosis is difficult in adults. However, in children erosion is the predominant type of tooth wear and often appears in isolation.

Dental erosion has received considerable interest in the U.K. since the Dental Health Component of the National Diet and Nutrition Survey (NDDS) reported prevalences of 51%, 14% and 28% in the age groups 5-6, 7-10 and 11-15, respectively.[2] Subsequent studies conducted in the U.K., Ireland, Iceland and the U.S. found prevalences of dental erosion in the range of 10% to 60% (Appendix 4). The majority of these studies indicated a higher prevalence of dental erosion in males than in females.

In the last few decades, a significant shift in the types of drinks consumed in industrialized countries has occurred. There has been a massive increase in the consumption of carbonated drinks[a] and a large reduction the consumption of milk among children and adolescents.[3,4] It has been reported that over 82% of 13-18-year-olds in the U.S. consume carbonated drinks on a regular basis and that 65% of the carbonated drinks in the U.K. are consumed by children and adolescents.[5,6]

In the U.K. consumption more than doubled between 1985 and 1996, with the average amount rising from 388 to 884 ml/person/week.[7] In 2002, the intake of carbonated drinks amounted to almost 212 litres per person in the U.S. This represented 27% of total intake of drinks for that year.[8] The data indicate that the North American market accounts for 27% of the total world carbonated drinks sales. It is followed by the European market (21%) with the consumption of 50.8 litres/person/year.[9]

Multinational companies control the global carbonated drink industry. It is concentrated mainly within the Coca-Cola Co., the PepsiCo., and the Cadbury Schweppes. The Coca-Cola brand, a product of the Coca Cola Co., holds the largest segment of the market (60%) with its Coca-Cola brand.[9]

[a] This class of products is also known as: soft drinks, sodas, pops, cokes colas and fizzy drinks.

Carbonated drinks are non-alcoholic, water-based, flavored drinks that constitute a homogenous group designated by the following common major ingredients that are present in different proportions in different brands:

a. Water
b. Sweeteners (sacharose/sugar, fructose, glucose, saccharin, aspartame)
c. Carbon dioxide
d. Acids (phosphoric acid, citric acid, malic acid)
e. Aromatic substances (caffeine)
f. Colouring matter (caramel, betakarotin)
g. Antioxidants (ascorbic acid)
h. Preservatives (natrium benzoate, potassium sorbate).

Considering these components and the change in the pattern of drinks intake, there is a growing concern in the medical and dental scientific communities about the harmful health effects associated with the consumption of carbonated beverages. Research in medicine has suggested several implications for general health, such as childhood obesity, diabetes, osteoporosis, bone fractures, neurological disorders and gastronomic distress.[10-13] In dentistry, carbonated drinks have been studied in relation to dental caries and dental erosion because of the role that fermentable carbohydrates and acids have in their pathogenesis.

Carbonated drinks have been reported to be the largest source of non-milk extrinsic sugars (NMES) (Appendix 5) in the U.S., accounting for one-third of the total sugar intake. [14] In the U.K., there has been a significant increase in NEMS contained in processed and manufactured drinks, including those that are carbonated. [14] The average 12-ounce can of carbonated drinks contains about 40 grams of fermentable sugars. They are also available in

20-ounce bottles and as large size fountain drinks. Appendix 6 shows the sugar content and pH levels of the major brands of carbonated drinks.

Based on the research evidence, the consensus view is that NEMS are highly cariogenic, contrary to intrinsic sugars whose cariogenicity is low and to milk that is virtually non-cariogenic.[14] It has been also proven that the amount of NEMS intake is closely correlated with the frequency of the eating/drinking of NEMS, which is itself directly related to the risk of caries development.[14]

All carbonated drinks are very acidic, particularly dark colas such as Coke and Pepsi (Appendix 6). The low pH results from the component acids and the carbonic acid (H_2CO_3) originating from the carbon dioxide dissolved in the water in the manufacturing process. Several *in vitro* studies have examined the erosive potential of carbonated beverages.[15-18] Their findings have suggested that these drinks may be acidic enough to demineralize tooth enamel.

2.0 OBJECTIVES

The purpose of this report is to review the literature concerning the relationship between the consumption of carbonated drinks and the oral health of children and adolescents. The best available epidemiological evidence regarding the following was evaluated:

1. Is carbonated beverage consumption a risk factor for dental caries in the population aged 0-18 years?

2. Is carbonated beverage consumption a risk factor for dental erosion in the population aged 0-18 years?

3.0 METHODS

3.1 Literature search

Studies pertaining to the research questions were identified by means of a two-stage iterative search of the dental and medical literature for the period from January 1966 to March 2004 (Appendix 7 & 8). First, lists of potentially relevant citations were generated from two electronic bibliographic databases: Medline and Cinahl. The following Medical Subject Headings (MeSH) were applied: dental caries/ dental erosion; carbonated beverages; and beverages. These terms and their combinations were also used in the key-word search. Second, subsequent to the exclusion of the citations judged irrelevant based on their titles and abstract, the bibliographies of those that were retrieved were screened for further citations. Two rounds of this process were undertaken. Hand-searching of journals was not involved and no attempt was made to identify unpublished literature.

3.2 Selection criteria

The search was limited to English-language literature to allow for full text evaluation. Based on the objectives of this report, the following papers were not considered: (i) studies involving adult populations; (ii) in-vitro studies; (iii) animal studies; (iv) case reports/series;

(v) editorials; and (vi) traditional narrative reviews. The absence of systemic and developmental disorders among the study participants was also an inclusion criterion. In addition, the retrieval of papers was limited to studies conducted in developed industrialized countries.

Experimental studies (i.e. randomized and non-randomized controlled trials) would have provided the most valid information. However, they were not expected to exist as they would have been unethical given the nature of the scientific question. Consequently, the following hierarchy of research designs with assigned levels of evidence was used:

Research design	Evidence level
Prospective cohort study	I
Retrospective cohort study	II
Case-control study	III
Cross-sectional study	IV

Anticipating that cohort and case-control studies would be scarce, the threshold for study selection was set to low. However, cross-sectional studies were to be included only in case of an insufficient number of preferred studies.

3.3 Quality assessment

An in-depth critical appraisal of the studies retained from the literature search was performed to assess the quality of evidence they provide. This was achieved by evaluating the following: (a) study design; (b) sample characteristics; (c) exposure measurement; (d) outcome measurement; (e) method of data analysis (including control for confounding factors).

4.0 RESULTS

4.1 CARBONATED DRINKS AND DENTAL CARIES

Based on the inclusion/exclusion criteria, four papers were selected for a review of evidence concerning the relationship between the consumption of carbonated drinks and the occurrence of dental caries.[19-22] According to research design, one was a retrospective cohort study[19] and three were cross-sectional studies.[20-22]

Appendices 9, 10 & 11 summarize the information obtained from these studies according to the criteria set for the quality assessment. The strength of the evidence they provide is shown in Appendix 12.

In addressing the secondary objective of the Iowa Fluoride Study, Marshall *et al*[19] evaluated the relationship between the diet and the presence of dental decay in a retrospective study involving a birth cohort of 642 children. A weighted average of the 1- through 5-year carbonated beverage intake was estimated from a minimum of 1 diary/year completed by parents or caregivers. The exposure categories derived from these data were: none (nonconsumers); low (consumption below the median) and high (consumption above the median). Caries experience, defined as a cavitated or filled surface, was determined for participants between the ages 4 and 7 years. This was achieved by means of a visual examination aided by probing when there was need to confirm the diagnosis. Logistic regression and multivariable Tobit regression models were developed for a set of exposure variables (Appendix 11). The former suggested significantly higher risk of caries occurrence in the 'high intake' than in the 'none/low intake' group (OR=2.2; 95% CI=1.4-3.6); the latter found a significant positive association between the *dfs*[b] and the dietary categories (B=0.767; $p<0.00$: 'high intake' vs. 'none/low intake').

Ismail *et al*[21] and Heller *et al*[20] analyzed the National Health and Nutrition Examination Survey 1971-74 (NHANES I) and 1988-94 (NHANES III) data, respectively, to explore the association between the consumption of carbonated drinks and the occurrence of dental caries.[20,21] This survey is based on a multistage cluster sampling of the US non-institutionalized civilian population. The dental examinations conducted in the surveys determined the DMFS[c] scores for the participants aged 9-29 years (NHANES I) and 6+ years (NHANES III), and the *dfs* scores for the participants aged 2-11 years (Appendix 10). The frequency and the amount of carbonated drinks intake were estimated by means of a 24-hour dietary recall. In the NHANES I, both at- and between-meal data were collected. In addition, the NHANES III determined the frequency of consumption in the age group 12+ yrs during the previous month. In the logistic regression models controlling for age, gender, race, family income, parental education and the consumption of table sugars and sugary

---

[b] Number of decayed and filled surfaces of primary teeth
[c] Number of decayed, missing and filled surfaces of permanent teeth

desserts, the frequency of consuming carbonated drinks at- and between-meals was a significant predictor of high (i.e. $\geq 80^{th}$ percentile) DMFS scores in comparison to low (i.e. $\leq 20^{th}$ percentile) DMFS scores among the NHANES I participants aged 12-14 and 14-16 yrs. However, the statistics (OR; p-value) are not reported. The three measures of carbonated drink consumption obtained in the NHANES III were not associated with the caries level examined in participants younger than 25 years. This was determined through a set of multiple linear regressions for each of the exposure measurements and the individual ages that included the covariates presented in Appendix 11.

A school-based survey conducted in the UK in 1994/95 suggested a significant correlation between the average number of cans of carbonated drinks consumed per week and the DMFT[d] scores among 14-year-old children.[22] However, the logistic regression model did not indicate the independent effect of this variable (OR=1.03).

Results of the quality assessment

Exposure measurement - Consumption of carbonated drinks was measured by means of: (a) 3-day diet diary[19]; (b) 1-day diet recall[20,21]; and (c) frequency questionnaires assessing the intake in the previous month[20] and on the most days[22] (Appendix 10). The diet diaries and recalls collected information about the amount consumed and the intake frequency. The frequency of intake was estimated as the number of cans[22] or the number of servings[20,21] consumed per day (Appendix 10). None of the studies inquired about drinking habits (e.g. drinking with/without the straw; drinking at/between meals; holding the drink in the mouth).

---

[d] Number of decayed, missing and filled permanent teeth

Outcome Measurement – Caries experience was measured using different indices: *dfs*[19,20], DMFT[21] and DMFS[20] indices (Appendix 10). Jones *et al*[22], on the other hand, classified children only as caries-free or caries-affected. The diagnostic methods varied across the studies. They included: visual examination[19]; visual-tactile examination[20]; and visual examination aided by probing in case of uncertainty[19] (Appendix 10). Ismail *et al*[21] did not report the diagnostic criteria. The case definition, provided in only two studies was different. As in the study by Marshall *et al*[19] the criterion for diagnosing caries was a cavitated/filled tooth surface, while in the study by Jones *et al*[22] it was dentinal caries.

Statistical analysis - The data were analyzed by means of either multiple linear regression (MLR)[19,20] or logistic regression (LR) [19,21,22] (Appendix 11). Both studies using the MLR explored the effect of the amount of carbonated drinks intake. Heller *et al*[20] also tested the association with the frequency of exposure. The outcome variables in the LRs were different. While Marshall *et al*[19] and Jones *et al*[22] explored the differences in the exposure to carbonated drinks between caries-free and caries-affected children, Ismail *et al* [21] compared the consumption among children whose DMFT scores were below and above the 10th percentile.

Confounding factors – All studies included in the review considered the potential confounders in estimating the effect of the consumption of carbonated drinks on the occurrence of dental caries (Appendix 11). However, they varied between the studies and represented small sub-sets of the factors that may affect this association.

The results of these studies are inconsistent. Two studies showed no association[20,22] and two suggested an association between carbonated drinks consumption and dental caries occurrence[19,21](Appendix 11 & 12). Of the latter two, one did not report the effect size[21] and one found an odds ratio of 2.2 (95% CI = 1.4-3.6). In the MLR model, the regression coefficient for the amount of carbonated drinks consumed was statistically significant, having a value of 0.767.

4.2 CARBONATED DRINKS AND DENTAL EROSION

Following the inclusion/exclusion criteria, ten papers were selected for the review of the evidence concerning the relationship between carbonated drinks consumption and the occurrence of dental erosion.[2,6,23-30] According to their research design, three were case-control studies [6, 23,24] and seven were cross-sectional studies (n=7).[2, 25-30]

Appendices 13, 14 & 15 summarize the information obtained from these studies according to the criteria set for the quality assessment. The strength of the evidence they provide is shown in Appendix 16.

In a matched case-control study, Milosevic *et al*[23] explored the association between the frequency of daily exposure to carbonated drinks and the occurrence of erosion into the dentin in 15-year-old children. The cases were identified in a random sample of school children surveyed one year prior to the initiation of the case-control study and consisted of those deemed to be most severely affected. After excluding these children, the registers of the same schools were used to select the controls by means of a systematic random sample.

The conditional logistic regression involving 37 case-control pairs found that the consumption of carbonated drinks had an effect on the occurrence of dental erosion. However, the association was with borderline statistical significance.

In a school-based case-control study, Arnadóttir *et al*[24] found no association between exposure to carbonated drinks and the presence of dental erosion in 15-year-olds. Here, the control was the first erosion-free child surveyed following the case identification.

In a convenience sample of 3-16-year-olds recruited from the diverse clinical settings, O'Sullivan *et al*[6] found that the mean consumption frequency of carbonated drinks reported for the four days preceding the study was higher among children with dental erosion than children who either did or did not have dental caries. No inferential statistics were reported from this study.

The cross-sectional studies were surveys of different types: (a) national - with complex sampling designs[2]; (b) county/regional - with systematic random sampling of schoolchildren[25]; census in a stratified convenience sample of schools[26]; proportional random samples from schools selected on a convenience basis[27]; and local – with school-based[28,29] and clinically-based[30] samples. The number of children involved ranged from one hundred[30] to over seventeen hundred.[2] In only five of these studies data were analyzed at the multivariate level, i.e. controlling for potential confounders.[2,23-26] While Nunn *et al*[2] and Harding *et al*[26] found no association, the research by Dugmore *et al*[25] suggested a link between the consumption of carbonated drinks and the presence of dental erosion.

Results of the quality assessment

Exposure measurement – The consumption of carbonated drinks was assessed by means of: (a) diet diary – 4-day[2] and 7-day[2] (b) 4-day diet recall[6]; and (c) questionnaires measuring the intake frequency for different reference periods and in the form of either dichotomous[28] or count variables[23-27,29,30] (Appendix 14). Some studies collected information about drinking habits (e.g. drinking time: with/without the straw, at/between meals; holding in the mouth). However, neither this information nor the data on the amount of carbonated drinks consumed were used in the data analysis.

Outcome measurement - These studies used different indices, the majority of which address tooth wear in general and not dental erosion specifically. They are: (a) Smith & Knight Tooth Wear Index (TWI); (b) modified versions of the Smith & Knight TWI; (c) Al-Malik TWI; (d) modified Lussi *et al.* Dental Erosion Index (DEI), and (e) a DEI designed for the purpose of the study (Appendix 14). One study did not report the diagnostic criteria it used.[6] The versions of the Smith & Knight TWI differed in the groups of teeth and tooth surfaces that were examined (Appendix 14). Furthermore, the case definition (when provided) varied between the studies. While Milosevic *et al*[23] and Harding *et al*[26] included only the severe erosion group (i.e erosion into dentin/pulp) in the analysis, the remaining studies considered any level of the erosion severity (Appendix 14).

Confounding factors - Potential confounders for the association between the consumption of carbonated drinks and the occurrence of dental erosion are numerous and diverse (Appendix

2). Although most of the studies included in the review measured some of these factors, they were controlled for in only five studies.[2,23-26] Of these, three indicated the confounders included in the analysis.[2,24,25] As Appendix 2 shows, they represent markedly different sets of variables.

Statistical analysis - One study reported descriptive statistics only.[6] The inferential statistics provided by the remaining studies are derived from either bivariate[27,29,30]or multivariate analyses[2,23-26] (Appendix 15). Therefore, only five studies examined the independent effect of the exposure to carbonated drinks. The data were analyzed by means of multiple logistic regression, with the intake of carbonated drinks being only in the form of dichotomous variables. While Harding *et al*[26] explored the risk associated with <1 vs ≥ 1 exposure per day, the remaining studies compared the occurrence of dental erosion in the groups of 'non-users' and 'users on most days'.

Of the six studies which tested the independent effect of the carbonated drinks consumption on the occurrence of dental erosion, three demonstrated positive associations with the odds ratios 1.6[25,26], 2.2[25] and 2.6[23] (Appendix 15 & 16). The statistical significance was borderline in the study by Milosevic *et al*[23] and Harding *et al.*[26]

5.0 CONCLUSIONS

The research evidence concerning the relationship between the consumption of carbonated drinks and the occurrence of either dental caries or dental erosion is weak and equivocal and, therefore, inconclusive. The reasons are two-fold.

The first reason is that the findings pertaining to this association cannot be compared. This is due to differences in the characteristics of the study populations, methodological heterogeneity of the studies (i.e. differences in the study designs and quality) and clinical heterogeneity (i.e. differences in exposure and outcome measurement) of the studies.

The second reason is that the studies included in the review are subject to methodological and/or reporting flaws that compromise the soundness of the evidence they provide, i.e. their internal and external validity. This pertains primarily to the control for confounders since, if accounted for in the studies, they were represented by a limited and dissimilar set of factors. The questionnaires are a crude way of assessing the diet and, therefore, may produce biased estimates. The significant weakness is also that the majority of the studies are cross-sectional studies (i.e. surveys) and case-control studies of the cross-sectional type. They are inadequate for exploring the cause-effect relationship unless it can be safely assumed that the putative causal factor is stable. Since little is known about the stability of either eating or drinking habits over long periods of time, the validity of this assumption for the intake of carbonated drinks is yet to be proven. The relationship observed in these studies is further attenuated by the fact that the indices of dental caries and dental erosion experience, given the irreversibility of these conditions, represent cumulative measures. Thus, this could also introduce a degree of inaccuracy in estimating the association explored in this review.

The existing epidemiological studies do not provide the answer as to whether carbonated drinks are a risk factor for the occurrence and/or progression of either dental caries or dental erosion. However, they suggest the possibility of this link. Taking this into account and considering the high consumption of carbonated drinks among children and adolescents and the fact that it is biologically plausible for carbonated drinks to have the adverse effect on tooth tissues, there is clearly a need for further research. Longitudinal studies involving standardized measurements of exposure and controlling for potential confounding factors are necessary to obtain information that can be used to establish the cause-effect relationship if it does exist.

6.0 REFERENCES

1. Pindborg JJ. In: Pathology of the dental hard tissues. 1st Edition, pp 312-321. Munksgaard, Copenhagen. 1970.
2. Nunn JH, Gordon PH, Morris AJ, Pine CM, Walker A. Dental erosion –changing prevalence? A review of British national childrens' surveys. Int J Paediatr Dent 2003; 13: 98-105.
3. Borrud LG, Enns CW, Mickle S. What we eat in America: USDA surveys food consumption changes. Food Rev 1996; 19: 14-19. Available at http://www.ers.usda.gov/publications/foodreview/sep1996/sept96.pdf. Accessed March 19, 2004.
4. Cavadini C, Seiga-Riz AM, Popkin BM. US adolescent food intake trends from 1965-1996. Arch Dis Child 2000; 83:18-24.
5. Harnack L, Stang J, Stiry M. Soft drink consumption among US children and adolescents: Nutritional consequences. J Am Dietetic Assoc 1999; 99: 436-41.
6. O'Sullivan EA, Curzon ME. A comparison of acidic dietary factors in children with and with out dental erosion. J Dent Child 2000; May-June: 186-192.
7. Ministry of Agriculture, Fisheries and Food: National Food Survey; London: Her Majesty's Stationery Office, 1996.
8. National Soft Drink association. Available at www.nsda.org. Accessed March 19, 2004.
9. Mathur HB, Johnson S, Kumar A. Analysis of pesticide residues in soft drinks. Centre for science and environment. Available at http://www.cseindia.org/html/cola-indepth/index.htm. Accessed July 8, 2004.

10. Ludwig DS, Peterson KE, Gormarker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational; analysis. Lancet 2001; 351: 505-8.

11. Wyshak G. Mazariegos-Ramos E, Guerrero-Romero F, Rodriguez-Moran M, Lazcano-Burciaga G, Paniagua R, Amato D. Consumption of soft drinks with phosphoric acid as a risk factor for the development of hypocalcaemia in children: a case-control study. J Pediatr 1995; 126:940-942.

12. Teenaged girls, carbonated beverage consumption, and bone fractures. Arch Pediatr Adolesc Med 2000; 154: 610-613.

13. Guerrero-Romero F, Rodriguez-Moran M, Reyes E. Consumption of soft drinks with phosphoric acid as a risk factor for the development of hypocalcaemia in postmenopausal women. J Clin Epidemiol 1999; 52: 1007-1010.

14. Daly B, Watt R, Batchelor P, Treasure E. Sugars and caries prevention. In Essential Dental Public Health. Oxford: Oxford University Press2002, pp 187.

15. Larsen MJ, Nyvad B. Enamel erosion by some soft drinks and orange juices relative to their pH, buffering effect and contents of calcium phosphate. Caries Res 1999; 33: 81-7.

16. Zero DT. Etiology of dental erosion-extrinsic factors. Eur J Oral Sci 1996; 104: 162-177.

17. Lussi A, Kohler N, Zero D, Schaffner M, Megert B. A comparison of the erosive potential of different beverages in primary and permanent teeth using an invitro model. Eur J Oral Sci 2000; 108:110-114.

18. Lussi A, Jaggi T, Scharer S. The influence of different factors on invitro enamel erosion. Caries Res 1993; 27:387-93.

19. Marshall TA, Levy SM, Broffitt B, Warren JJ, Eichenberger-gilmore JM,Burns T et al. Dental caries and beverage consumption in young children. Pediatrics 2003; 112: e 184-89.

20. Heller KE, Burt BA, Eklund SA. Sugared soda consumption and dental caries in the United States. J Dent Res 2001; 80: 1949-53.

21. Ismail AI, Burt BA, Eklund SA. The cariogenicity of soft drinks in the United States. JADA1984; 109:241-45.

22. Jones C, Woods K, Whittle G, Worthington H, Taylor G. Sugar, drinks, deprivation and dental caries in 14-year-old children in the north wear of England in 1995. Community Dent Health 1999; 16: 68-71.

23. Milosevic A, Lennon MA, Fear SC. Risk factors associated with tooth wear in teenagers: a case control study. Community Dent Health 1997; 14: 143-147.

24. Arnadottir IB, Saemundsson SR, Holbrook PW. Dental erosion in Icelandic teenagers in relation to dietary and lifestyle factors. Acta Odontol Scand 2003; 61:25-28.

25. Dugmore CR, Rock WP. The prevalence of tooth erosion in 12-year-old children. Br Dent J 2004; 196: 279-82.

26. Harding MA, Whelton H, O'Mullane DM, Cronin M. Dental erosion in 5-year-old Irish school children and associated factors: a pilot study. Community Dent Health 2003; 20: 165-170.

27. Al-Dlaigan YH, Shaw L, Smith A. Dental erosion in a group of British 14-year-old school children Part II: Influence of dietary intake. Br Dent J 2001; 190: 258-61.

28. Deery C, Wagner ML, Longbottom C, Simon R, Nugent ZJ. The prevalence of dental erosion in a United States and a United Kingdom sample of adolescents. Pediatr Dent 2000; 22: 505-510.

29. Bartlett DW, Coward PY, Nikkah C, Wilson RF. The prevalence of tooth wear in a cluster sample of adolescent school children and its relationship with potential explanatory factors. Br Dent J 1998; 184:125-129.

30. Millward A, ShawL, Smith AJ, Rippin JW, Harrington E. The distribution and severity of tooth wear and the relation ship between dietary constituents in a group of children. Int J Paediatr Dent 1994; 4:151-157.

31. Johnson NW. Risk markers for oral disease v.1. : Dental caries: markers of high and low risk groups. Cambridge: New York: Cambridge University Press, 1991.

32. Milosevic A. Toothwear: aetiology and presentation. Dental Update 1998; 25: 6-11.

33. Milosevic A, Young PJ, Lennon MA. The prevalence of tooth wear in 14-year-old school children in Liverpool. Community Dent Health 1994; 11:83-86.

7.0 APPENDICES

Appendix 1

Risk factors for dental caries[31]

Intrinsic factors	Extrinsic factors	
	Sugars other than from carbonated drinks	**Behavioural, lifestyle & general health factors**

Enamel acid resistance
  Fluoride exposure pre- & post-eruptive
  Genetic make-up
  Pre-natal deprivation & disturbance
Saliva
  Buffering capacity
  Flow rate
  Clearance rate
Tooth
  Anatomy
  Position
  Occlusion
Other
  Soft tissue
  Swallowing pattern
	Dietary
	  Table sugar
	  Confectionary
	  Juices
	Non-dietary
	  Medications
	  Vitamin supplements
		Oral hygiene habits
		  Frequency
		  Time
		  Technique
		Eating & Drinking habits
		  Frequency
		  Amount
		  Time: at/between meals; bed-time
		  Drinking with straw
		  Drinking at once/sipping
		Lifestyle
		  Use of drugs (e.g. "Ecstasy"):
		    Increased consumption of carbonated drinks due to dehydration
		  Physical activity
		    Increased consumption of carbonated drinks due to dehydration
		Medications
		  Xerogenic
		    Decreased salivary flow and clearance
		    Increased consumption of carbonated drinks due to dry mouth symptoms
		  Immunosuppressive

Appendix 2
Risk factors for dental erosion[1]

Intrinsic factors	Extrinsic factors	
	Acids other than from carbonated drinks	Behavioural & lifestyle factors
Enamel acid resistance Fluoride exposure pre- & post-eruptive Genetic make-up Pre-natal deprivation & disturbance Saliva Buffering capacity Flow rate Clearance rate Tooth Anatomy Position Occlusion Other Soft tissue Swallowing pattern Gastric acids Reflux Regurgitation Vomiting	Dietary Fruit juices Acidic foods Sport supplemental drinks Alcopops Non-dietary Environmental Industrial Swimming pool Therapeutic Medications for asthma, renal disease Aspirin Vitamin C tablets	Oral hygiene habits Frequency Time Technique Drinking habits Frequency Amount Time: at/between meals; bed-time Straw use At once/sipping Holding, swishing & frothing around the mouth Drink T° Lifestyle Use of alcohol: Increased gastric reflux & vomiting Use of drugs (e.g. "Ecstasy"): Increased consumption of carbonated drinks due to dehydration Increased gastric reflux & vomiting Eating disorders Bulimia Physical activity Recreational: Increased consumption of carbonated drinks due to dehydration Competitive: Sport supplements Parafunctional habits Bruxism; clenching; grinding

Appendix 3[32]

Tooth wear		
Term	**Definition**	**Clinical appearance**
Erosion	Progressive loss of hard tissue by chemical processes not involving bacterial action	• Broad concavities within smooth surface enamel • Cupping of occlusal surfaces/incisal grooving with dentin exposure • Increased incisal translucency • Wear on non-occluding surfaces • "Raised" amalgam restorations • Clean, non-tarnished appearance of amalgams • Loss of surface characteristics of enamel in young children • Preservation of enamel "cuff" in gingival crevice is common • Hypersensitivity • Pulp exposure in deciduous teeth
Attrition	Loss by wear of surface of tooth or restoration caused by tooth to tooth contact during mastication or parafunction	• Matching wear on occluding surfaces • Shiny facets on amalgam contacts • Enamel and dentin wear at the same rate • Possible fracture of cusps or restorations
Abrasion	Loss by wear of dental tissue caused by abrasion by foreign substance (e.g. toothbrush, dentifrice)	• Usually located at cervical areas of teeth • Lesions are more wide than deep • Premolars and cuspids are commonly affected
Abfraction	Loss of tooth surface at the cervical areas of teeth caused by tensile and compressive forces during tooth flexure *(Studies needed to prove this hypothetical phenomenon)*	• Affects buccal/labial cervical areas of teeth • Deep, narrow V-shaped notch • Commonly affects single teeth with excursive interferences or eccentric occlusal loads

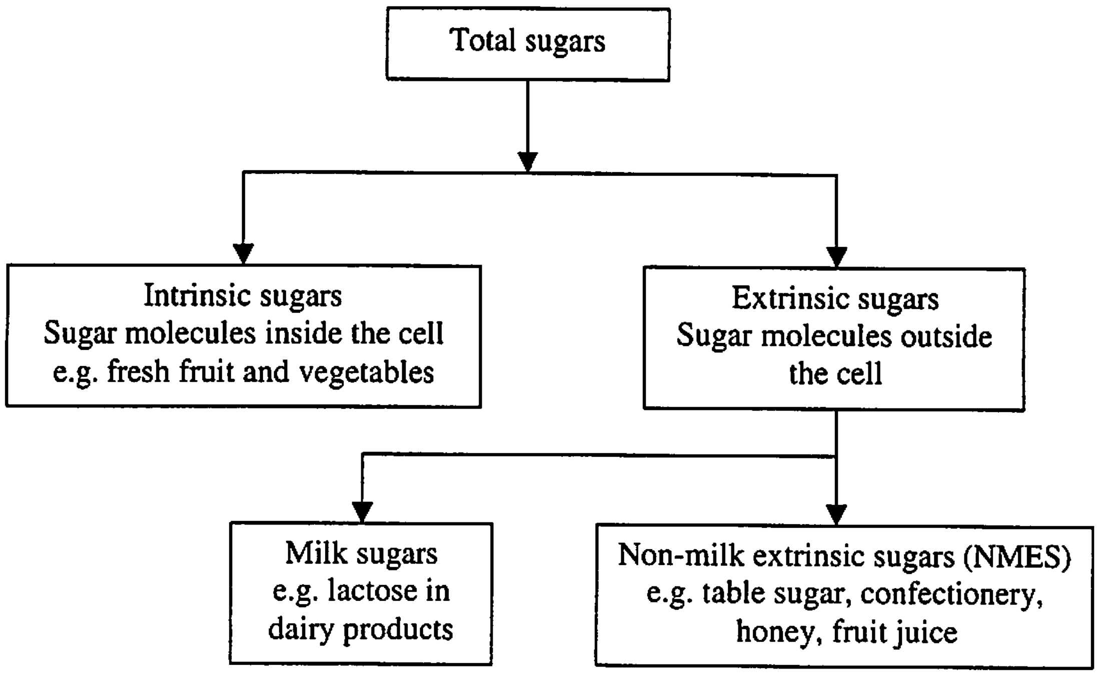
Appendix 4

Prevalence of Dental Erosion in Children

Author/Year	Country	Age (yrs)	Prevalence (%)
UK Child Dental Health Survey 1993[2]	UK	5-6 7-10 11-15	51 14 28
Milosevic et al., 1994[33]	UK	14	30
Millward et al., 1994[30]	UK	4-16.5	35
Bartlett et al., 1998[29]	UK	11-14	57
Deery et al., 2000[28]	UK	11-13	37
	US	11-13	41
Nunn JH et al, 2003[2]	UK	4-6 7-10 11-14 15-18	10 25 42 56
Arnadóttir et al., 2003[24]	Iceland	15	21.6
Harding et al., 2003[26]	Ireland	5	47
Dugmore et al., 2004[25]	UK	12	59.7

Appendix 5

Classification of sugars



Adapted from: Daly et al. Essential Dental Public Health. Oxford University Press, 2002

Appendix 6

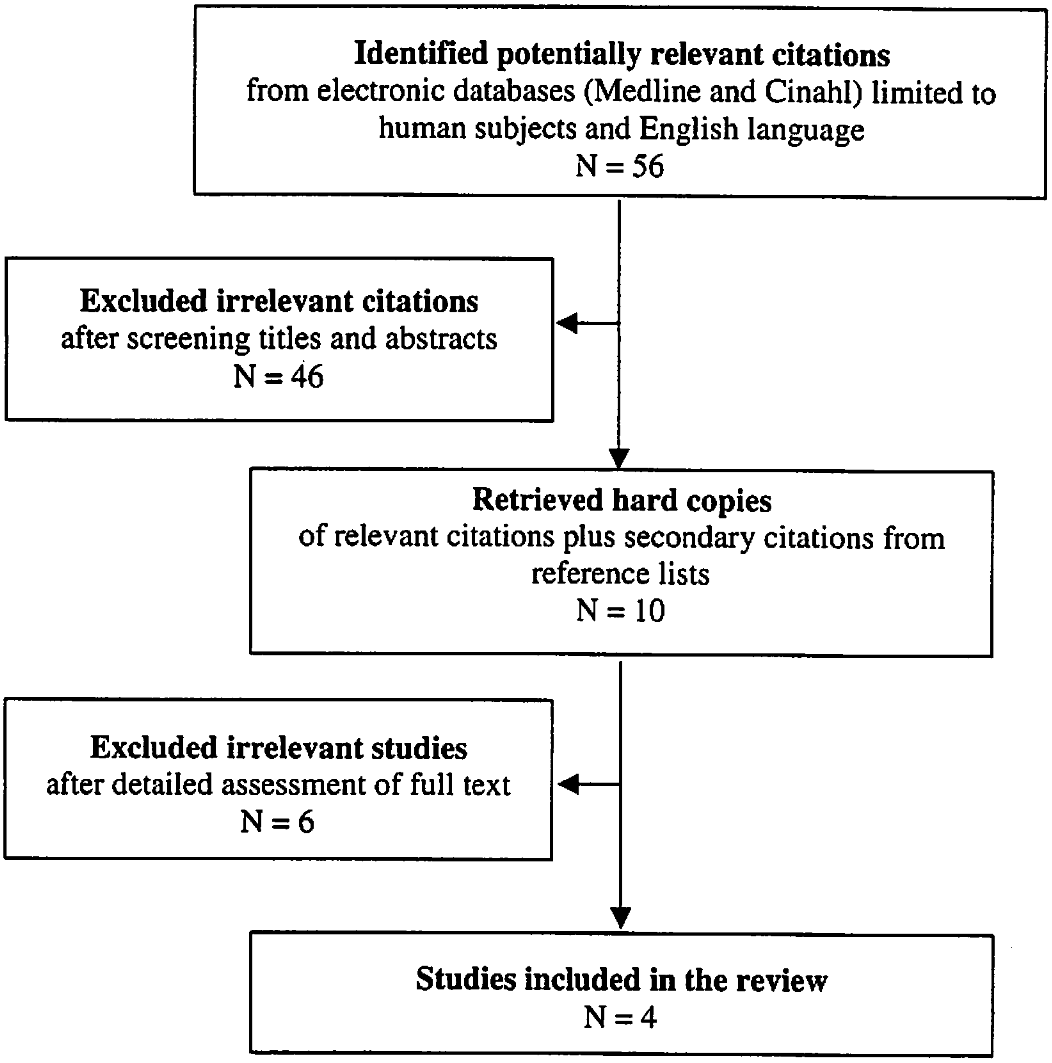
Sugar content and pH levels of carbonated non-diet soft drinks

Beverage	Sugars (grams / 360 ml)	pH
Mountain Dew (PepsiCo)	6	-
Pepsi	41	2.7
Dr. Pepper (Cadbury Schweppes)	40	2.17
Coca-Cola Classic	39	2.7
7 Up (Cadbury Schweppes)	39	3.5
Sprite (Coca- Cola Co.)	38	-

From: http://www.cspinet.org/sodapop/liquid_candy.htm (Accessed: July 23, 2004)

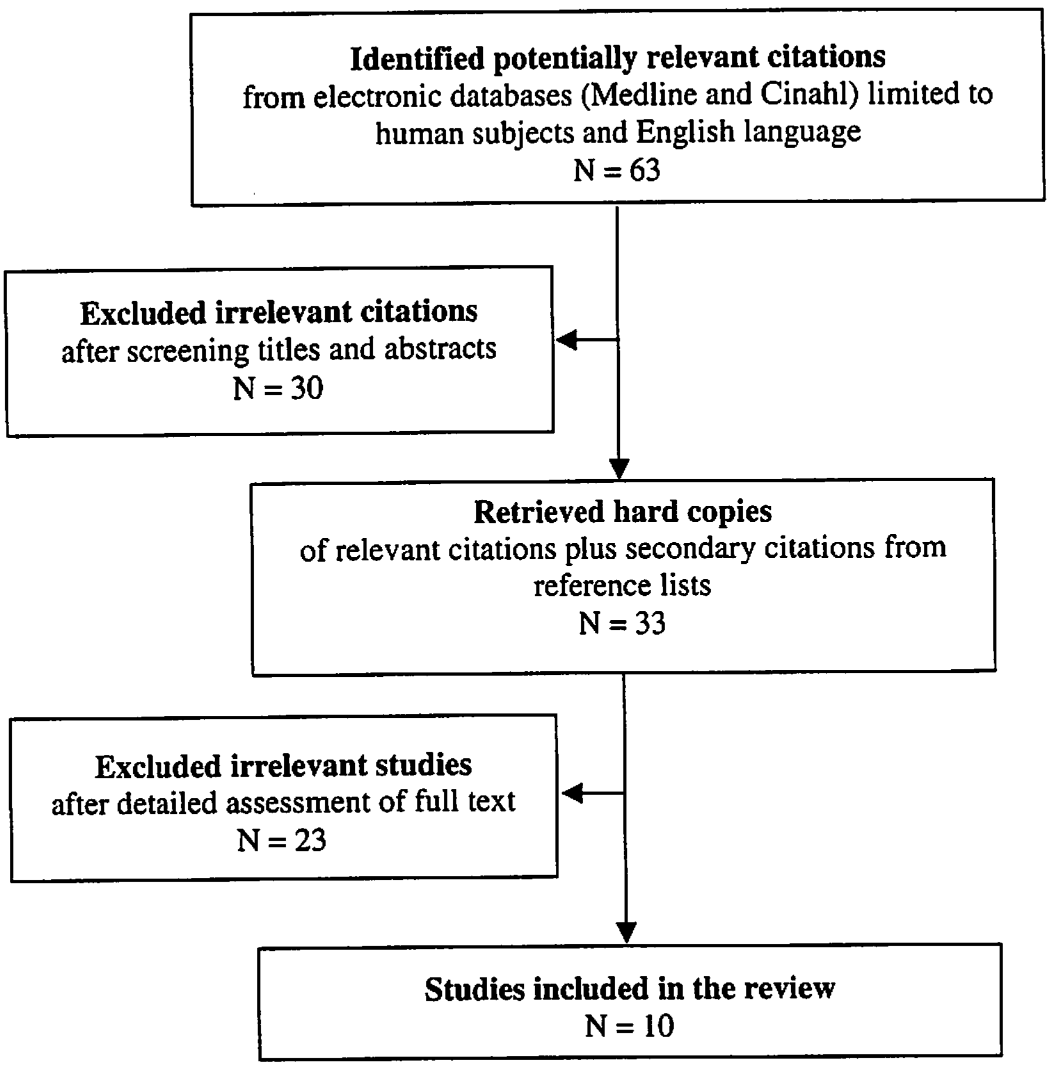
Appendix 7

Process of identifying relevant literature for examining the relationship between the consumption of carbonated beverages and dental caries



Appendix 8

Process of identifying relevant literature for examining the relationship between the consumption of carbonated beverages and dental erosion



Appendix 9

Main characteristics of the studies concerning the relationship between carbonated drinks consumption and dental caries

Author (year) & country	Study type	Age (yrs)	Sample size	Sample design	Sampling unit
Marshall *et al* (2003)[19]; U.S.	Retrospective cohort	4-7	642	Birth cohort (convenience)	Hospital
Heller *et al* (2001)[20]; U.S.	Cross-sectional	2-18	6,079 (for *dfs*) 4,630 (for DMFS)	Multistage cluster probability	Members of civilian non-institutionalized population
Jones *et al* (1999)[22]; U.S	Cross-sectional	14	6,014	1st : Probability cluster 2nd: Simple random	1st: School 2nd: Child
Ismail *et al* (1984)[21]; U.S.	Cross-sectional	9-17	3,194 (9-29 yrs)	Multistage cluster probability	Members of civilian non-institutionalized population

Appendix 10

Main characteristics of the studies concerning the relationship between carbonated drinks consumption and dental caries (cont'd)

Study	Exposure measurement (Carbonated drinks consumption)			Outcome measurement (Dental caries occurrence)
	Measure	Report (self/proxy)	Follow-up (yrs)	
Marshall *et al*[19]	3-day diet diary ≥1/yr between 1-5 yrs of age: amount (gr)	Proxy (parent)	1-4	Criterion: Cavitated/filled tooth surface Index: a.Child affected – yes/no b.*dfs*[b] Method: Visual examination & probing
Heller *et al*[20]	1. 24-hr dietary recall a. Frequency: # servings/day b. Amount (oz) 2. Frequency: past month	Ø[a]	0	Criterion: Ø Index: a.*dfs* (2-11yrs) b.DMFS[c] (6+yrs) Method: Visual-tactile examination
Jones *et al*[22]	Q: Frequency: average #cans/day	Self	0	Criterion: Caries into dentin Index: Child affected – yes/no Method: Visual examination
Ismail *et al*[21]	24-hr dietary recall c. Frequency: # servings/day d. Amount (gr)	Ø	0	Criterion: Ø Index: DMFT[d] Method: Ø

[a] Not reported; [b] # of decayed and filled primary teeth surfaces
[c] # of decayed, missing and filled permanent teeth surfaces; [d] # of decayed, missing and filled permanent teeth surfaces

Appendix 11

Main characteristics of the studies concerning the relationship between carbonated drinks consumption and dental caries (cont'd))

Study	Data analysis	Control for confounding	Association
Marshall *et al*[19]	1. LR[a] Outcome: caries (no=0; yes=1) Exposure (amount: none/<median; >median=1) 2. MLR[b] Outcome: *dfs*[c] Exposure: Amount	Age; gender; fluoride exposure; dietary variables	1. OR[d]=2.2 (95%CI=1.4-3.6) 2. B[e]=0.767 (p<0.001)
Heller *et al*[20]	MLR Outcome: *dfs* Exposure: a. amount b. frequency (0,1,2,3/day)	Age; gender; poverty/income ratio; dietary variables	No
Jones *et al*[22]	LR Outcome: caries (no=0; yes=1) Exposure: frequency/day	Age; gender; consumption of table sugars	No
Ismail *et al*[21]	LR Outcome: caries (DMFT[f]≤10[th] percentile=0; DMFT≥10[th] percentile=1) Exposure: a. amount b. frequency	Age; gender; race; family income; parental education; consumption of table sugars	Yes (OR Ø)

[a] Logistic regression; [b] Multiple linear regression;
[c] # of decayed and filled primary teeth surfaces; [d] Odds ratio; [e] MLR regression coefficient ;
[f] # of decayed and filled permanent teeth surfaces

Appendix 12

The strength of evidence concerning the relationship between carbonated drinks consumption and dental caries

Level of evidence	Quality of evidence	Association	
		Yes	No
II	Good	Marshall *et al* (2003)[19] Positive Multivariate analysis	
	Fair		
	Poor		
IV	Good		Heller *et al* (2001)[20] Jones *et al* (1999)[22]
	Fair	Ismail *et al* (1984)[21] Positive Multivariate analysis	
	Poor		

Appendix 13

Main characteristics of the studies concerning the relationship between carbonated drinks consumption and dental erosion

Author (year) & country	Study type	Age (yrs)	Sample size	Sample design	Sampling unit
Milosevic *et al* (1997)[23]; U.K.	Matched case-control a. Population-based b. Cross-sectional exposure measurement	15	37	Convenience cluster Controls: Systematic random from the same sampling frame	School
Arnadóttir *et al* (2003)[24]; Iceland	Case-control a. Population-based b. Cross-sectional exposure measurement	15	278	Convenience cluster Controls: consecutive to cases	School
O'Sullivan *et al* (2000)[6]; U.K.	Matched case-control a. Population-based b. Cross-sectional exposure measurement	3-16	103	Convenience clinically-based	Child
Nunn *et al* (2003)[2]; U.K.	Cross-sectional	(a) 1.5-4.5 (b) 4-18	(a) 1,451 (b) 1,726	Ø	Ø
Dugmore and Rock (2004)[25]; U.K.	Cross-sectional	(a) 12 (b) 14	(a) 1,149 (b) 1,149	Systematic random	Child
Harding *et al* (2003)[26]; Ireland	Cross-sectional	5	202	Stratified convenience cluster	School
Al-Dlaigan *et al* (2001)[27]; U.K.	Cross-sectional	14	418	Stratified convenience cluster	School & child
Derry *et al* (2000)[28]; U.K. & U.S.	Cross-sectional	11-13	245	Convenience	Child
Bartlett *et al* (1998)[29]; U.K.	Cross-sectional	11-14	210	Convenience cluster	School
Millward *et al* (1994)[30] U.K.	Cross-sectional	9-17	101	Convenience clinically-based	Child

Appendix 14

Main characteristics of the studies concerning the relationship between carbonated drinks consumption and dental erosion (cont'd)

Study	Exposure measurement (Carbonated drinks consumption)		Outcome measurement (Dental erosion occurrence)	
	Measure	Report (self/proxy)	Index	Teeth & surfaces examined
Milosevic *et al*[23]	Q - Frequency: # times/week	Self	Smith & Knight TWI[a]	All teeth: all surfaces
Arnadóttir *et al.*[24]	Q - Frequency: previous day	Self	Modified Lussi et al. DEI[b]	All teeth: all surfaces
O'Sullivan *et al*[6]	4-day dietary recall	Self: younger children Parent: older children	DEI designed for the study	Ø
Nunn *et al*[2]	(a) 4-day diet diary (b) 7-day diet diary	(a) Parent (b) Self: older children Parent: younger children	Modified Smith & Knight TWI	Upper incisors: labial & palatal surfaces 1st molars: occlusal surfaces
Dugmore *et al*[25]	Q - Frequency: #cans/day	Self	Modified Smith & Knight TWI	All incisors: labial & palatal surfaces 1st molars: occlusal & lingual surfaces
Harding *et al*[26]	Q - Frequency: #times/day	Proxy	Al-Malik TWI	Upper incisors: labial & palatal surfaces
Al-Dlaigan *et al*[27]	Q - Frequency: #times/week	Self	Ø[c] In: Part I of the study	Ø[c] In: Part I of the study
Deery *et al*[28]	Q - Yes/No	Self	Modified Smith & Knight TWI	Upper incisors: labial & palatal surfaces
Bartlett *et al*[29]	Q - Frequency: #cans/day	Self	Smith & Knight TWI	All teeth: all surfaces
Millward *et al*[30]	Q - Frequency: #times/week	Self: older children Parent: younger children	Smith & Knight TWI	All teeth: all surfaces

[a] Tooth Wear Index; [b] Dental Erosion Index; [c] Not reported

Appendix 15

Main characteristics of the studies concerning the relationship between carbonated drinks consumption and dental erosion (cont'd)

Study	Data analysis	Control for confounding	Association
Milosevic *et al*[23]	Conditional LR[a] Outcome: erosion to dentin/pulp (no=0; yes=1) Exposure: consumption on most days (no=0; yes=1)	Ø[b]	Yes (borderline) OR[c]=2.6 (95% CI = 0.91-7.45)
Arnadóttir *et al*[25]	LR Outcome: erosion (no=0; yes=1) Exposure: no=0; yes=1	Gender; Gastric reflux; Asthma inhalers; Fruit juice consumption	No
O'Sullivan *et al*[6]	Descriptive: Mean consumption frequency in study groups: erosion: 2.72; caries-active: 1.52; caries free: 1.20	None	Not tested
Nunn *et al*[2]	LR Outcome: erosion (no=0; yes=1) Exposure: consumption on most days (no=0; yes=1)	Age; parental education; household type; other drinks; drinking habits; gastric reflux; asthma	No
Dugmore *et al*[25]	LR Outcome: erosion (no=0; yes=1) Exposure: consumption (no=0; yes =1)	Dental decay; calculus; dental cleanliness; orthodontic anomaly; eating: apples, citrus, chips with tomato sauce/vinegar, chocolate, sweets; drinking fruit juice; asthma	Yes 12 yrs: OR=1.59 (95%CI=1.18-2.13) 14 yrs: OR=2.21 (95%CI=1.87-4.24)
Harding *et al*[26]	LR Outcome: erosion to dentin/pulp (no=0; yes=1) Exposure: frequency/day (<1=0; ≥1=1)	Ø	Yes (borderline) OR=1.59 (95%CI=1.0-5.0)
Al-Dlaigan *et al*[27]	Correlation: Prevalence of erosion (#buccal/labial and palatal/lingual surfaces) & Consumption frequency/week	None	Cola drinks: rho=? (p<0.001) Other fuzzy drinks: rho=? (p<0.0010
Deery *et al*[28]	Ø	None	No
Bartlett *et al*[29]	Correlation: TWI & # cans/day	None	No (R[d]=-0.09)
Millward *et al*[30]	One-way ANOVA: Mean consumption frequency in study groups: no/mild:3.9; moderate:5.8; severe: 13.9	None	<0.001

[a] Logistic regression; [b] Not reported; [c] Odds ratio; [d] Correlation coefficient

Appendix 16

The strength of evidence concerning the relationship between carbonated drinks consumption and dental erosion

Level of evidence	Quality of evidence	Association		
		Yes	No	Not tested
III	Good		Arnadóttir *et al* (2003)[24] Multivariate analysis	
	Fair	Milosevic *et al* (1997)[23] Positive borderline Multivariate analysis		O'Sullivan *et al* (2000)[6]
	Poor			
IV	Good	Dugmore *et al* (2004)[25] Positive Multivariate analysis		Nunn *et al* (2003)[2]
	Fair	Harding *et al* (2003)[26] Positive borderline Multivariate analysis		Deery *et al* (2000)[28] Bartlett *et al* (1998)[29]
	Poor	Al-Dlaigan *et al* (2001)[27] Positive Bivariate analysis Millward *et al* (1994)[30] Positive Bivariate analysis		