Prevalence of Dental Fluorosis in the Primary Dentition

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Abstract

Objectives: *This paper presents data on the prevalence of primary tooth fluorosis among children residing in Iowa, and the relationships between fluorosis prevalence and selected measures of fluoride exposures.* ***Methods:*** *Children in the study cohort were followed prospectively during the first year of life. This study assessed their home water fluoride concentrations and use of fluoride dentifrice or dietary fluoride supplements. A total of 637 children (320 females and 317 males) were examined for fluorosis using a modification of the TSIF index at age 4 1/2 to 5 years, with 90.4 percent having intact primary dentitions.* ***Results:*** *74 children (11.6%) had fluorosis present on one or more of their primary teeth, and 71 children (11.1%) had two or more teeth affected. Nearly all fluorosis was mild, with the primary second molar teeth most commonly affected. Fluorosis was significantly associated with higher water fluoride concentration, but not with the use of dentifrice or fluoride supplements.* ***Conclusions:*** *The results of this study show that primary tooth fluorosis is relatively uncommon, but is most frequently seen on the posterior teeth, particularly the primary second molars, which form at later stages of development. This finding suggests that primary tooth fluorosis is mostly a postnatal phenomenon, and is associated with higher water fluoride levels. [J Public Health Dent 2001;61(2):87-91]*

Key Words: dental fluorosis, primary dentition, fluorides, water fluoridation.

There have been an increasing number of studies on the prevalence of dental fluorosis in the permanent dentition and its risk factors (1-6). One such study found that fluorosis in the permanent dentition was associated with fluorosis in the primary dentition (7). However, many fewer studies have assessed the prevalence of primary tooth fluorosis, particularly in North America.

A number of studies of primary tooth fluorosis have been conducted in isolated areas in Africa and Europe with high levels of water fluoride (8-14). These studies have found nearly universal prevalence of primary tooth fluorosis in these locations, and have found that the primary molars, particularly the primary second molars, are most often and most severely affected (8-10,13).

Studies of primary tooth fluorosis in industrialized nations in Europe, with more moderate levels of water fluoride (2.0 ppm or less), are less common. Forsman (15) found in the 1970s that fluorosis did not occur in the primary dentition among children living in an area in Sweden with water fluoride levels of 0.2 ppm or less; however, in communities with 0.8 ppm F, 43 percent were affected and 76 percent had primary tooth fluorosis in a community with a water fluoride level of 2.75 ppm. The study found that fluorosis of the primary dentition was less severe than that in the permanent dentition, with primary tooth fluorosis more prevalent and severe in the primary molars than the anterior primary teeth. A study of children residing in communities in Greenland and Denmark (16), with water fluoride concentrations from 1.1 to 1.6 ppm, found fluorosis prevalence in the primary dentition to be about 15–30 percent, with prevalence and severity positively associated with consumption of powdered infant formula reconstituted with tap water. This study found all primary fluorosis to be in the mild or very mild categories, with the second primary molars most commonly affected (16).

More recently in the United Kingdom, Booth et al. (17) found primary tooth fluorosis prevalence in a community with 0.3 ppm F (32%) to be similar to that in a community with 1.0 ppm F (34%). That study of 3-year-olds found primary tooth fluorosis to be somewhat more common in the incisors than in the first molars, although fluorosis on the primary first molars was significantly (P=.005) more prevalent in the 1.0 ppm F community than in the nonfluoridated community. In contrast, a similar study also conducted in the United Kingdom found the prevalence of primary tooth fluorosis in an optimally fluoridated community (1.0 ppm) to be 29 percent compared to 14 percent in a low-fluoride community (0.3 ppm) (18). As in most other studies, primary tooth fluorosis was more prevalent on the molars than the canines or incisors.

The only contemporary study of primary tooth dental fluorosis in the United States was conducted by Leverett et al. (19) in nonfluoridated areas (<0.3 ppm) of Maine. The study was designed as a double-blind, placebo-controlled study to investigate the effectiveness of prenatal fluoride supplements in caries prevention, where both the prenatal fluoride and placebo groups received postnatal dietary fluoride supplements. The study found no statistically significant effect of the prenatal supplements in terms of caries experience or fluorosis prevalence. Overall, this study found that about 3 percent of children had primary fluorosis at age 5 years. The highest prevalence was in the primary second molars, which accounted for 70 percent of all affected teeth (19). There

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have been no contemporary US studies of primary tooth dental fluorosis in fluoridated areas, or in locations with combinations of both fluoridated and nonfluoridated water supplies.

This paper presents data on the prevalence of primary tooth fluorosis and associations with water fluoride levels, use of fluoride supplements, and use of fluoride dentifrice among 637 Iowa children participating in a longitudinal cohort study.

Methods

Children included in the present study were part of the Iowa Fluoride Study cohort (20-24), which had been followed prospectively since birth. The children were examined mostly at age 4 1/2 to 5 years (mean age=4.63 years), 98.0 percent were white, and 90.4 percent had intact primary dentitions. A total of 637 children (320 females and 317 males) were examined for dental caries and fluorosis.

Because there are no published criteria specific for primary tooth fluorosis, criteria were based on the Tooth Surface Index of Fluorosis (TSIF) (25). Using descriptions from published literature, photographs, extensive consultation with a developer of the TSIF index, and other sources of information, these criteria were adapted for the primary dentition (26). One adaptation was to score the presence or absence of fluorosis on each of the gingival, middle, and occlusal thirds of the buccal surface when fluorosis was identified on the buccal surface of a specific tooth. Other adaptations were more minor and were due to differences in location and appearance between primary tooth fluorosis and the permanent tooth fluorosis for which TSIF was developed. TSIF codes 4 and 6 were not used because fluorosis is less severe and staining is less common in primary tooth fluorosis than in permanent tooth fluorosis (27), and for TSIF code 1, "snow-capping" was not used as a definitive criterion for diagnosis. As with permanent teeth, the scores of 1, 2, and 3 were based on collapsing the area affected to approximate the amount of the surface affected. Also consistent with the TSIF protocol, teeth were examined using a mouth mirror and examination light, but the teeth were not dried.

Fluorosis was distinguished from other lesions, such as isolated non-fluoride opacities, largely by applying Russell's criteria for differential diagnosis of fluorosis (28). Specifically, fluorosis was differentiated from non-fluoride opacities based on differences in shape, demarcation, color, and detection ability of the lesions. Because fluorosis in the primary dentition is somewhat different from fluorosis in the permanent dentition, Russell's criteria regarding specific teeth affected and areas affected were not applicable. In addition, Russell's observation that fluorosis in the permanent teeth generally occurs bilaterally (28) was not used as a strict criterion for fluorosis in the present study. Fluorosis was distinguished from enamel demineralization ("white spot" lesions) based on color, texture, demarcation, and relationship to the gingival margin, with areas closely adapted to and paralleling the gingival margin generally being consistent with demineralization, while areas more variable in their approximation to the gingival margin generally being consistent with fluorosis.

Examinations were conducted by one of two trained and calibrated examiners using a portable chair and examination light. Interexaminer reliability was assessed by examinations of approximately 10 percent of subjects by both examiners periodically throughout data collection, which took place from August 1997 through March 2000. Percent agreement and kappa statistics were computed at the subject, tooth, and surface levels. At the person level, percent agreement was 86.2 percent and kappa was 0.49; at the tooth level, percent agreement was 97.5 percent and kappa was 0.57; and at the surface level, percent agreement was 98.3 percent and kappa was 0.46.

Fluoride exposure data were obtained using questionnaires completed by parents periodically throughout the child's first year of life. Specifically, fluoride dentifrice use was assessed by questionnaires mailed at 9 months and 12 months of age, which asked whether dentifrice was used during the preceding three-month period. Fluoride supplement use was assessed at 6 months and 9 months of age, and subjects were categorized as having used supplements if supplement use was reported at either of these time points. Home water fluoride concentration was measured at 6 months of age. For the home water fluoride determination, those using municipal water supplies were assigned fluoride values for their community as reported to the Iowa Department of Health. For those consuming water from private wells or filtered municipal water, water samples were obtained and tested by the study team using a fluoride-specific electrode.

Data were entered using SPSS® Data Entry software (29), and statistics were generated using SPSS® (30) and SAS (31). Chi-square analyses were used to assess associations between categorical or ordinal variables, while *t*-tests were used to compare differences between group measured on an interval scale, with $P<.05$ considered statistically significant.

Results

Of the 637 children examined, 74 (11.6%) had fluorosis present on one or more of their primary teeth, 71 (11.1%) had fluorosis on two or more teeth, and 39 (6.1%) had fluorosis on four or more teeth. As shown in Table 1, fluorosis occurred bilaterally in one or both arches most of the time, so that usually an even number of teeth (78%) were affected per person. Based on previous definitions (32-34), those with fluorosis were considered to be those with two or more teeth affected. Using this definition, the mean number of teeth affected for the entire

TABLE 1
Number of Teeth with Fluorosis Present

Number of Teeth with Fluorosis	*N*	% of Children	% of Children with Fluorosis
0	563	88.4	—
1	3	0.5	4.1
2	23	3.6	31.1
3	9	1.4	12.2
4	21	3.3	28.4
5	2	0.3	2.7
6	10	1.6	13.5
7	1	0.2	1.3
8	3	0.5	4.1
9	1	0.2	1.3
12	1	0.2	1.3
Total	637	100	100

sample was 0.44, and among those with fluorosis it was 3.93. The mean numbers of surfaces affected were 0.67 and 5.96, respectively. Nearly all fluorosis was mild (TSIF score=1), with only two individuals having higher TSIF scores.

Figure 1 depicts the percentages of subjects with fluorosis by individual tooth identity. By far, the primary second molar teeth were most commonly affected, with fluorosis prevalence ranging from 8.8 percent to 9.4 percent. First molars were affected in 1.2–2.2 percent of the subjects, and canines and incisors were affected in fewer than 1 percent of the subjects. Among the second molar teeth with fluorosis, the buccal surface was affected in nearly 80 percent of cases, with the occlusal and lingual surfaces each affected in about 25–35 percent of uses. On the buccal surface of the second molars, the most common location was near the gingival margin, with 75 percent to 91 percent of affected teeth exhibiting fluorosis on the gingival third of the buccal surface. The middle and occlusal thirds of the buccal surface of the second molars were each involved in 26 percent to 44 percent of affected teeth and varied among specific teeth.

Use of fluoride dentifrice was reported by 25 percent of the subjects and 25.8 percent reported using fluoride supplements at some time during the first year of life. The mean home water fluoride concentration at six months of age was 0.82 ppm, with a range from <0.01 ppm to 2.40 ppm.

There were significant differences in the prevalence of fluorosis among suboptimal, optimal, and high water fluoride concentrations, with higher fluorosis prevalence in areas with higher water fluoride levels (Table 2). The mean home water fluoride concentration for those with fluorosis (0.90 ppm) was also significantly higher (P=.001, t-test) than for those with no fluorosis (0.76 ppm). In addition, the prevalence of fluorosis was higher among children who had used dietary fluoride supplements than among those with no supplement use; however, this difference was not statistically significant. There was no significant difference in fluorosis prevalence between those reporting dentifrice use during the first year of life and those not using fluoride dentifrice during this period. Fluorosis was slightly more prevalent in males than in females, but this difference also was not statistically significant.

FIGURE 1
Percent of Subjects with Fluorosis by Tooth

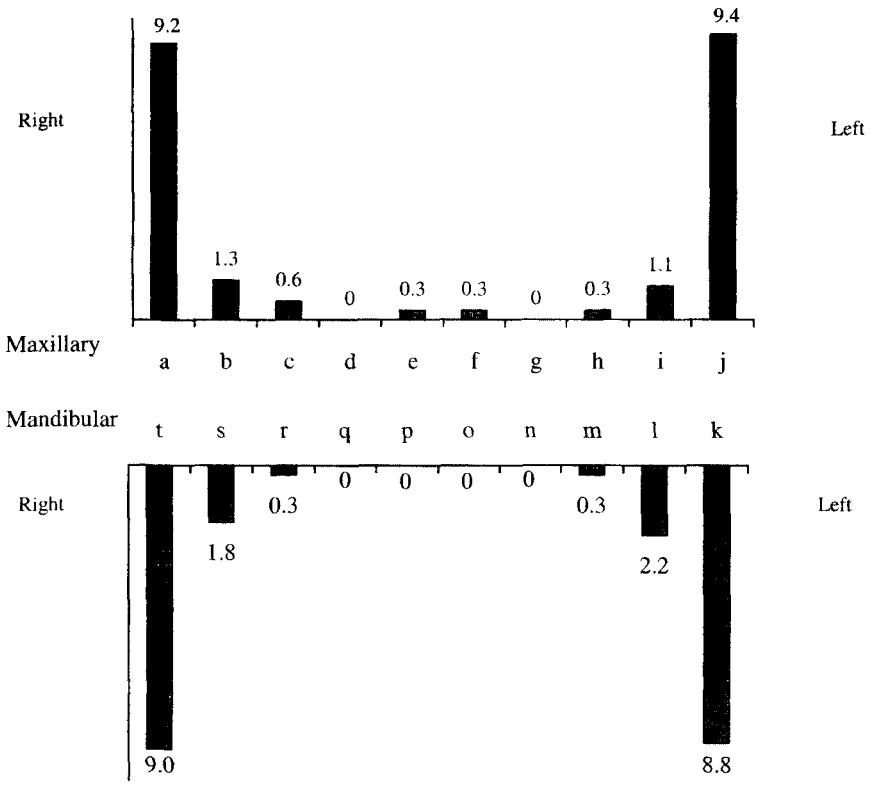

TABLE 2
Prevalence of Dental Fluorosis* by Fluoride Exposure Variables and Sex*

Variable	n†	Fluorosis Prevalence		P-value‡
		n	%	
Home water fluoride concentration				.002
<0.70 ppm	173	10	5.8	
0.70–1.20 ppm	305	38	12.5	
>1.20 ppm	81	17	21.0	
Dietary fluoride supplement use				.612
Yes	131	13	10.0	
No	506	58	11.5	
Fluoride dentifrice use				.428
Yes	159	15	9.4	
No	478	56	11.7	
Sex				.502
Male	317	38	12.0	
Female	320	33	10.3	

*Defined as two or more teeth affected by fluorosis.
†Water fluoride concentration was available for 559 of 637 subjects at 6 months of age.
‡P-values based on chi-square analyses.

Discussion

The data for this descriptive analysis were obtained as part of a longitudinal study of fluoride exposures and dental fluorosis, and the main goal was to relate the presence and severity of fluorosis in the permanent dentition to the longitudinally collected meas-

ures of fluoride exposure. As intermediate steps toward this ultimate study goal, the primary dentition was assessed for the prevalence and severity of fluorosis and those findings were related to measures of fluoride exposures, focusing on the first year of life. Thus, as a longitudinal cohort study, the sample is not representative of any defined population, and comprises children whose parents chose to keep them in the study. Therefore, generalizations and comparisons to other studies should be made with caution. In particular, cohort families were of relatively higher socioeconomic status than the general population as measured by income and education levels.

As with studies of fluorosis in the permanent dentition, several different indices have been used to assess primary tooth fluorosis, making comparisons across studies difficult. For example, in the only other US study of fluorosis in the primary dentition, Leverett et al. (19) used Dean's Index (33,35) and reported primary tooth fluorosis prevalence of 3 percent. However, Dean's Index (33,35) has a "questionable" category and the reported prevalence did not include cases in that category. Although it appears that fluorosis prevalence in the present study is somewhat higher than reported in nonfluoridated areas of Maine, differences in the indices used may account for at least some of the difference in prevalence.

In addition, because few studies have focused on primary tooth fluorosis and no indices have been developed specifically for it, it is possible that some studies misclassified other conditions as primary tooth fluorosis or did not recognize primary tooth fluorosis when it was present. As part of the present study, substantial effort was made to characterize primary tooth fluorosis through photographs obtained from other clinicians and researchers, correspondence with other investigators, and extensive review of the literature. In addition, the present study used Russell's criteria (28) to distinguish fluorosis from nonfluoride opacities based on shape, demarcation, and color of the lesions. Fluorosis was also distinguished from enamel demineralization based on color, texture, demarcation, and approximation to the gingival margin. Thus, while primary tooth fluorosis can be easily overlooked or confused with other conditions (such as "white spot" lesions or isolated opacities), the present study took steps to ensure that the presence or absence of primary tooth fluorosis was correctly assessed.

Consistent with most other studies, we found that primary tooth fluorosis was most prevalent on the primary second molars, particularly on the gingival third of the primary second molars. Because the gingival portions of the second molars are the parts of the primary tooth crowns that form latest in development among all primary teeth, it appears that primary tooth fluorosis, like permanent tooth fluorosis, may be due primarily to postnatal fluoride exposure. Although it appears that fluorosis can occur via prenatal exposure in isolated areas with extremely high water fluoride levels (8,10,13), it is likely that primary tooth fluorosis is related primarily to the level of postnatal fluoride ingestion during infancy.

The findings from the present study support the concept that fluorosis is mostly associated with postnatal fluoride because fluorosis occurred mostly on the later developing teeth and was associated with water fluoride concentration. Although there appeared to be a dose-response relationship between water fluoride concentration and fluorosis prevalence, it was not as strong or as dramatic as the relationship between water fluoride concentration and fluorosis in the permanent dentition as described by Dean (32,33,35). That is, although the prevalence of fluorosis was higher among children living in areas with higher water fluoride concentrations (>1.2 ppm) than among those in areas with lower fluoride concentrations, the prevalence was still relatively low. There may be several reasons for this finding, most notably the limited diets of children in the first year of life and variable consumption of water, such that only those children fed infant formulas mixed with fluoridated water are likely to be exposed to significant amounts of water fluoride early in life. As infants mature, their diets become more varied; nevertheless, water fluoride exposure is still likely limited to infant formula, concentrated fruit juices, and infant cereals mixed with water, with little water by itself (20).

Fluorosis occurred even among children residing in areas with lower fluoride levels (<0.7 ppm). Again, this finding may be due to dietary fluoride sources other than water, such as infant fruit juices, pureed fruits, and vegetables that have been shown to contain significant amounts of fluoride in some cases (22,36). Unlike studies of risk factors for fluorosis in the permanent dentition (37-42), the present study did not find either fluoride supplement use or dentifrice use to be significantly associated with fluorosis, although fluorosis prevalence was slightly higher among supplement users. These findings may be due to the relatively low intensity of supplement use in this sample as described previously (23), and the use of fluoride dentifrice limited to only the later stages of primary tooth formation (21,43,44).

Finally, it is important to note that although primary tooth fluorosis was associated only with water fluoride concentration in the present study, its prevalence, like that of permanent tooth fluorosis, is dependent on total fluoride intake from all sources. Thus, further research, including continued analyses of data from the present study, is needed to better characterize the link between total fluoride intake and dental fluorosis in both the primary and permanent dentitions.

In summary, the results of this study suggest that primary tooth fluorosis is relatively uncommon, but that it is most common on the posterior teeth, particularly the primary second molars, which form at later stages of development. That pattern strongly suggests that primary tooth fluorosis is mostly due to postnatal exposure to fluoride.

References

1. Kumar JV, Green EL, Wallace W, Carnahan T. Trends in dental fluorosis and dental caries prevalence in Newburgh and Kingston, NY. Am J Public Health 1989;79:565-9.
2. Williams JE, Zwemer JD. Community water fluoride levels, preschool dietary patterns, and the occurrence of fluoride enamel opacities. J Public Health Dent 1990;50:276-81.
3. Burt BA, Eklund SA. Dentistry, dental practice and the community. 4th ed. Philadelphia: W. B. Saunders, 1992:147.
4. Clark DC, Hann HJ, Williamson MF, Berkowitz J. Influence of exposure to various fluoride technologies on the prevalence of dental fluorosis. Community Dent Oral Epidemiol 1994;22:461-4.
5. Milsom K, Mitropoulos CM. Enamel defects in 8-year-old children in fluoridated and nonfluoridated parts of Cheshire. Caries Res 1990;24:286-9.

6. Rozier RG. The prevalence and severity of enamel fluorosis in North American children. J Public Health Dent 1999;59: 239-46.
7. Milsom KM, Woodward M, Haran D, Lennon MA. Enamel defects in the deciduous dentition as a potential predictor of defects in the permanent dentition of 8- and 9-year-old children in fluoridated Cheshire, England. J Dent Res 1996;75: 1015-18.
8. Forsman B. Dental fluorosis and caries in high fluoride districts in Sweden. Community Dent Oral Epidemiol 1974;2:132-48.
9. Olsson B. Dental findings in high-fluoride areas in Ethiopia. Community Dent Oral Epidemiol 1979;7:51-6.
10. Thylstrup A. Distribution of dental fluorosis in the primary dentition. Community Dent Oral Epidemiol 1978;6:329-37.
11. McInnes PM, Richardson BD, Cleaton-Jones PE. Comparison of dental fluorosis and caries in primary teeth of preschool-children living in arid high and low fluoride villages. Community Dent Oral Epidemiol 1982;10:182-6.
12. Mann J, Mahmoud W, Ernest M, Sgan-Cohen H, Shoshan N, Gedalia I. Fluorosis and dental caries in 6–8-year-old children in a 5 ppm fluoride area. Community Dent Oral Epidemiol 1990;18:77-9.
13. Opinya GN, Valderhaug J, Birkeland JM, Lokken P. Fluorosis of deciduous teeth and first permanent molars in a rural Kenyan community. Acta Odont Scand 1991;49:197-202.
14. Lewis HA, Chikte UME. Prevalence and severity of fluorosis in the primary and permanent dentition using the TSIF. J Dent Assoc S Afr 1995;50:467-71.
15. Forsman B. Early supply of fluoride and enamel fluorosis. Scand J Dent Res 1977; 85:22-30.
16. Larsen MJ, Senderovitz F, Kirkegaard E, Poulsen S, Fejerskov O. Dental fluorosis in the primary and permanent dentition in fluoridated areas with consumption of either powdered milk or natural cow's milk. J Dent Res 1988;67:822-5.
17. Booth IM, Mitropoulos CM, Worthington HV. A comparison between the dental health of 3-year-old children living in fluoridated Huddersfield and nonfluoridated Dewsbury in 1989. Community Dent Health 1992;9:151-7.
18. Weeks KJ, Milsom KM, Lennon MA. Enamel defects in 4- to 5-year-old children in fluoridated and nonfluoridated parts of Cheshire, UK. Caries Res 1993; 27:317-20.
19. Leverett DH, Adair SM, Vaughan BW, Proskin HM, Moss ME. Randomized clinical trial of the effect of prenatal fluoride supplements in preventing dental caries. Caries Res 1997;31:174-9.
20. Levy BT, Bergus GR, Levy SM, Slager SL, Kiritsy MC. Longitudinal feeding patterns of Iowa infants. Ambulatory Child Health 1996;2:25-34.
21. Levy SM, Kiritsy MC, Slager SL, Warren JJ, Kohout FJ. Patterns of fluoride dentifrice use among infants. Pediatr Dent 1997;19:50-5.
22. Heilman JR, Kiritsy MC, Levy SM, Wefel JS. Fluoride content of infant foods and cereals. J Am Dent Assoc 1997;128:857-63.
23. Levy SM, Kiritsy MC, Slager SL, Warren JJ. Patterns of dietary fluoride supplement use during infancy. J Public Health Dent 1998;58:228-33.
24. Warren JJ, Levy SM, Nowak AJ, Tang S. Nonnutritive sucking behaviors in preschool children: a longitudinal study. Pediatr Dent 2000;22:187-90.
25. Horowitz HS, Driscoll WS, Meyers RJ, Heifetz SB, Kingman A. A new method for assessing the prevalence of dental fluorosis—the Tooth Surface Index of Fluorosis. J Am Dent Assoc 1984;109:37-41.
26. Warren JJ, Kanellis MJ, Levy SM. Fluorosis of the primary dentition: what does it mean for permanent teeth? J Am Dent Assoc 1999;130:347-56.
27. Fejerskov O, Manji F, Baelum V, Møller IJ. Dental fluorosis—a handbook for health workers. Copenhagen: Munksgaard, 1988.
28. Russell AL. The differential diagnosis of fluoride and nonfluoride enamel opacities. J Public Health Dent 1961;21:143-6.
29. SPSS Data Entry Builder™ 1.0 user's guide. Chicago: SPSS, Inc., 1998.
30. SPSS user's guide. Version 7.5. Chicago: SPSS, Inc., 1996.
31. SAS user's guide. Statistics. Version 6. Cary, NC: SAS Institute, Inc., 1995.
32. Dean HT. Epidemiological studies in the United States. In: Moulton FR, ed. Dental caries and fluorine. Washington, DC: American Association for the Advancement of Science, 1946:21.
33. Dean HT, Arnold FA Jr, Elvove E. Domestic water and dental caries. V. Additional studies of the relation of fluoride domestic waters to dental caries experience in 4,425 white children, aged 12–14 years, of 13 cities in 4 states. Public Health Rep 1942;57:1155-79.
34. Pendrys DG. The fluorosis risk index: a method for investigating risk factors. J Public Health Dent 1990;50:291-8.
35. Dean HT Dean HT, Arnold FA Jr, Elvove E. Domestic water and dental caries II. A study of 2,832 white children, aged 12–14 years, of eight suburban Chicago communities, including Lactobacillus acidophilus studies of 1,761 children. Public Health Rep 1941;56:761-92.
36. Kiritsy MC, Levy SM, Warren JJ, Guha-Chowdhury N, Heilman JR, Marshall T. Fluoride concentrations of juices and juice drinks. J Am Dent Assoc 1996;127: 895-902.
37. Pendrys DG. Risk for fluorosis in a fluoridated population. J Am Dent Assoc 1995; 126:1617-24.
38. Pendrys DG, Katz RV. Risk of enamel fluorosis associated with fluoride supplementation, infant formula, and fluoride dentifrice use. Am J Epidemiol 1989; 130:1199-208.
39. Pendrys DG, Katz RV, Morse DE. Risk factors for enamel fluorosis in a fluoridated population. Am J Epidemiol 1994; 140:461-71.
40. Pendrys DG, Katz RV, Morse DE. Risk factors for enamel fluorosis in a nonfluoridated community. Am J Epidemiol 1996;143:808-15.
41. Lalumandier JA, Rozier RG. The prevalence and risk factors of fluorosis among patients in a pediatric dental practice. Pediatr Dent 1995;17:19-25.
42. Osuji OO, Leake JL, Chipman ML, et al. Risk factors for dental fluorosis in a fluoridated community. J Dent Res 1988; 67:1488-92.
43. Logan WHG, Kronfeld R. Development of the human jaws and surrounding structures from birth to the age of 15 years. J Am Dent Assoc 1933;20:379-427.
44. Lunt RC, Law DB. A review of the chronology of eruption of deciduous teeth. J Am Dent Assoc 1974;89:878.