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ABSTRACT

This study used an 11-month break in water fluoridation to identify the time when developing incisors are most sensitive to fluorosis development. The study was based in Durham, NC, where an interruption to water fluoridation occurred between September, 1990, and August, 1991. A total of 1896 children was dentally examined. Fluorosis was measured by the TF index, and parents or guardians completed a questionnaire on demographics and fluoride history. Age cohorts ranged from those born 5 years before the break, to those born 1 year after the resumption of fluoridation. Fluorosis prevalence for seven age cohorts whose birth years ranged from 1985-86 to 1991-92 was 57.1, 62.3, 33.0, 32.3, 39.8, 30.2, and 36.8%, respectively. Children aged from birth to 3 years at the break, and those born 1 year after it, had less fluorosis than those aged 4-5 years at the break.

KEY WORDS: fluoridation, fluorosis, epidemiology, caries, children.

Fluorosis Development in Seven Age Cohorts after an 11-month Break in Water Fluoridation

INTRODUCTION

In the United States, the main policy for ensuring widespread fluoride exposure is through water fluoridation, which has been categorized as one of the major public health achievements of the 20th century (US Public Health Service, 1999). Fluoridation's benefits have been accompanied by an increased prevalence of dental fluorosis (Szpunar and Burt, 1987; Pendrys and Stamm, 1990; Williams and Zwemer, 1990), though fluorosis in the United States is usually seen only in the mild to very-mild categories. Children are thought to be at particular risk of fluorosis if fluoride is ingested during the "critical period", the developmental time around late secretion/early maturation at which the unerupted tooth appears to be especially sensitive to fluoride (Evans and Stamm, 1991b; DenBesten, 1999).

In view of these "critical periods", a more precise definition of the ages at which fluoride most affects both caries and fluorosis will expand the science base for the use of fluoride. An opportunity to study this issue came from Durham, North Carolina, where the water supply has been fluoridated at 1.0 mg/L since the 1960s. Records from the water treatment plant in Durham show that fluoridation ceased between September, 1990, and August, 1991, because of technical problems. The fluoride level in Durham water during September, 1990, dropped sporadically to 0.1 mg/L. In October, the mean level was 0.5 mg/L, then it dropped to zero until August, 1991, when it was 1.1 mg/L. Fluoride remained around that level for the rest of 1991, and fluoridation has not been interrupted since. The stoppage was not publicized at the time, and later discussions with health professionals indicate that dentists and physicians did not try to compensate for it by prescribing fluoride supplements during the no-fluoridation period.

In an earlier report from Phase 1 of this project (Burt *et al.*, 2000), we found no caries effects, but we did find that fluorosis was less prevalent in children aged 1 to 3 years at the break than in those aged 4 or 5 years. We concluded that this was because the maxillary permanent incisors, in which fluorosis was measured, were at a more critical stage of development in the younger children at the time of the break. If that conclusion were correct, we would expect that children born after the break would have a fluorosis prevalence approximating that seen in those aged 4 and 5 when it occurred. In Phase 2 of the project, described in this paper, we tested that hypothesis by adding fluorosis data from children born 1990-92 (the same year as the break and 1 year after it) to the results for the five age cohorts seen in Phase 1.

MATERIALS & METHODS

Details of the study methods were described in the Phase 1 report (Burt *et al.*, 2000). In brief, the study design was a follow-up cohort comparison. The time interval between the age cohorts was one year. Since fluoridation ceased on September 17, 1990, children included in a cohort were those born on September 17 \pm six months (*e.g.*, the 1985-86 cohort was comprised of children born

between March 18, 1985, and March 17, 1986). Although the data were cross-sectional, examinations were spread over 6 years so that children were as close as possible to the same age at the time of examination. Phase 1 (1996-98) assessed children born between 1985 and 1989, and Phase 2 (2000-01) saw the addition of data from children born between 1990 and 1992.

Informed written consent was received from the parents or guardians of all subjects who participated in the study. The research protocol was approved by the University of Michigan Health Sciences Institutional Review Board (IRB), and by the Duke University Medical Center IRB before the study began.

Recruitment of Participants

For Phase 2 of the study, consent forms were distributed by classroom teachers to the 5689 children in Grades 3, 4, and 5 in the 22 participating elementary schools. Completed consent forms were received from 2336 children after one follow-up round. Of these responses, 2000 (85.6%) were positive consents, and 336 were negative. Of those 2000 positive consents, there were 761 children in Phase 2 who met the inclusion criteria of age and continuous residence in the Durham water supply area. This group became the study participants.

Clinical Examinations

There were two examiners, who standardized their clinical criteria during a training period before both phases of the study. All CDC recommendations for infection control in the field setting (Summers *et al.*, 1994) were observed. Fluorosis was scored on the labial surfaces of the permanent upper central and lateral incisors by the TF index (Thylstrup and Fejerskov, 1978). Teeth were dried with gauze, isolated with cotton rolls, and allowed to dry for at least 30 sec before diagnosis. Diagnostic data were recorded directly into laptop computers and later transferred to a desktop for analysis.

Questionnaire Information

A trained interviewer presented the same 11-item questionnaire as used in Phase 1 to the parent or guardian of each participant child. Most interviews were by 'phone, with a personal interview for those who could not be reached by 'phone. The questionnaire covered standard demographic information and the history of the participating child's fluoride exposure.

Statistical Analysis

Statistical methodology was similar to that used in our previous report (Burt *et al.*, 2000). Chi-squared tests were used to determine the association between fluorosis prevalence and each of the predictor variables from the questionnaire. Multivariable analyses were carried out by multiple linear and logistic regression models, with standard methods of model formulation (Hosmer and Lemeshow, 1989). Fluorosis outcomes were prevalence measures, defined as all children with at least one tooth with a TF score of 1 or higher. Independent variables that showed a moderate level of association ($p < 0.25$) with fluorosis in the bivariable analyses were considered in the logistic regression model-building procedure. Epi Info v.6.04d and SAS release 8.1 were used for the data management and statistical analysis.

RESULTS

Examiner Reliability

Each examiner independently re-examined the same group of 23 children during the 2001 clinical examinations. Comparison

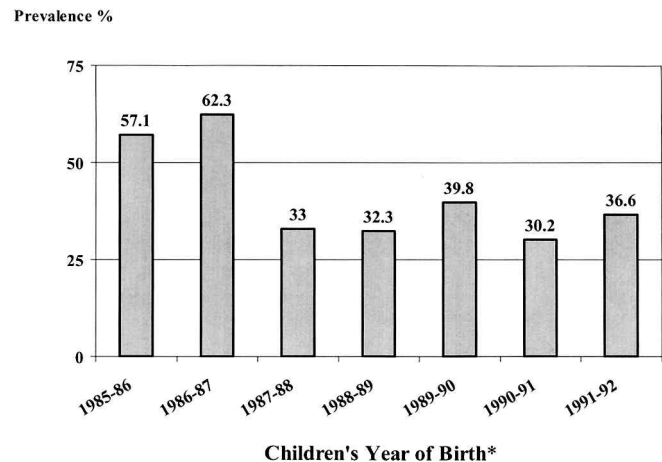


Figure 1. Distribution of fluorosis prevalence by age cohort. *The birth years cover the period from March 18 in the first year to March 17 in the second, e.g., March 18, 1985, to March 17, 1986.

of results showed 79% agreement for fluorosis, with a kappa score of 0.55. The analytic unit in each case was the tooth.

Demographics and Fluoride History

Clinical examination data and completed questionnaires were obtained from 597 of the 761 participants (78.4%) during Phase 2 of the study. When added to the 1299 children for whom fluorosis was scored in Phase 1, the total number of participants after Phase 2 was 1896 children. All were between 7 and 10 years old (mean = 8.8 yrs) at the time of the dental examination. Demographic attributes were little different from those seen in Phase 1 of the study (Burt *et al.*, 2000).

Fluorosis

Fluorosis prevalence, defined as those with at least one tooth with a TF fluorosis score of 1 or higher, was 40.6% for the entire group of 1896. The distribution of fluorosis scores for all age cohorts (Fig. 1) shows that the overall prevalence by age cohort was essentially similar for those children born 1987-92, with higher prevalence for those born 1985-87.

Table 1 shows the bivariate relationships between fluorosis prevalence and the demographic and fluoride exposure variables from the questionnaire. Cohort effects are evident, and toothbrushing frequency and use of fluoride supplements also were significantly associated with fluorosis.

Table 2 gives results from the logistic regression model for the case-control analysis. The cohort effects were confirmed, and reported toothbrushing frequency and use of fluoride supplements in infancy were also significant predictors of fluorosis. Not included in this model, because of weak relationships in Table 1, were gender, formula feeding in infancy, use of fluoride gels and rinses, tap or bottled water, race/ethnicity, and educational level of the female head of household.

DISCUSSION

In Phase 1 of this study (Burt *et al.*, 2000), we examined the impact of the fluoridation break on caries as well as fluorosis. No caries effects could be discerned, a finding we attributed to continuing exposure to fluoride from other sources as well as to generally low levels of caries. We looked for caries effects in

Table 1. Bivariate Relationships between Fluorosis Prevalence (TF = 1) and Questionnaire Responses on Demographics and Self-reported Fluoride Exposure

Variable	N	Fluorosis Prevalence (%)	Standard Error	p
Children born 1991-92 (mean age at exam, 9.4 yrs)	272	36.8	2.90	0
Children born 1990-91 (mean age at exam, 10.1 yrs)	325	30.2	2.50	
Children born 1989-90 (mean age at exam, 8.6 yrs)	256	39.8	3.10	
Children born 1988-89 (mean age at exam, 8.4 yrs)	288	32.3	2.80	
Children born 1987-88 (mean age at exam, 9.3 yrs)	279	33.0	2.80	
Children born 1986-87 (mean age at exam, 9.3 yrs)	257	62.3	3.00	
Children born 1985-86 (mean age at exam, 10.3 yrs)	219	57.1	3.30	
Female	1001	42.0	1.60	0.21
Male	895	39.1	1.60	
Brush 2+/day	1153	43.1	1.40	0.01
Brush 1/day	671	37.3	1.90	
Brush < 1/day	65	30.8	5.80	
Formula-fed only	1135	40.5	1.50	0.83
Breast-fed only	211	39.3	3.40	
Both formula- and breast-fed	528	41.7	2.10	
Fluoride supplements: yes	199	49.9	3.60	0.01
Fluoride supplements: no	1624	40.0	1.20	
Fluoride gel treatment: yes	1013	41.3	1.50	0.43
Fluoride gel treatment: no	771	39.4	1.80	
Fluoride rinse: yes	416	43.8	2.40	0.14
Fluoride rinse: no	1462	39.7	1.30	
Drink tap water only	1030	41.5	1.50	0.75
Drink bottled water only	72	38.9	5.80	
Drink both tap and bottled water	790	39.9	1.70	
Race: African-American	1281	39.8	1.40	0.52
Race: White	532	42.7	2.10	
Race/ethnicity: other	83	39.8	5.40	
Education, female head of household: low	666	39.8	1.90	0.27
Education, female head of household: high	1142	42.5	1.50	

Table 2. Logistic Regression Model for Case-Control^a Analysis for Fluorosis and Three Independent Variables

	Beta	Standard Error	Odds Ratio (95% CI)	p
Intercept	-1.04	0.27		0
Children born 1989-90 (referent)			1	
Children born 1991-92	-0.04	0.18	0.95 (0.67, 1.37)	0.18
Children born 1990-91	-0.33	0.18	0.72 (0.50, 1.02)	0.07
Children born 1988-89	0.31	0.18	0.74 (0.52, 1.05)	0.09
Children born 1987-88	-0.25	0.18	0.79 (0.54, 1.11)	0.17
Children born 1986-87	0.92	0.19	2.51 (1.74, 3.60)	0
Children born 1985-86	0.75	0.19	2.12 (1.46, 3.08)	0
Toothbrushing frequency	0.26	0.09	1.24 (1.04, 1.48)	0.02
Fluoride supplements	0.34	0.16	1.41 (1.04, 1.91)	0.03

^a Case-definition for fluorosis was at least 1 tooth with TF score = 1 (cases N = 753), controls had all teeth TF = 0 (controls N = 1107).

these additional two cohorts and again could find none, so caries data are not presented.

A continuing decrease in caries severity was found following the cessation of fluoridation in Finland (Seppä *et al.*, 1998) and in Germany (Künzel *et al.*, 2000), when children were examined several years after fluoridation ceased permanently. However, in both instances there was continuing fluoride exposure from toothpaste, and Finland has comprehensive school dental services. In the German communities, social change following German re-unification included increased use of dental services and dietary improvements. But in Durham, the 11-month interruption in water fluoridation is most likely not long enough to have adverse caries effects in this generally low-caries population, and, as in the European examples, exposure to other forms of fluoride did not cease during the break.

With fluorosis, the Hong Kong studies found that a permanent reduction in fluoridation level from 1.0 mg/L to 0.7 mg/L led to a reduction in fluorosis prevalence from

64% to 47% (Evans and Stamm, 1991a). Based on this finding, our hypothesis was that the children born in 1990-92 would show the same fluorosis prevalence as those children born in 1985-87 (whose anterior teeth were reasonably well-developed by the time of the fluoridation break in 1990). This hypothesis could not be supported. Fluorosis prevalence in the age cohorts born in 1990-92 was similar to the prevalence in those born in 1987-90, mostly children for whom the fluoride exposure from drinking water was interrupted at an earlier developmental stage.

In trying to explain this unexpected result, the first possibility we checked was whether the children born in 1985-87 were younger at the time of examination than those born in 1990-92.

The significance of age in fluorosis measurement is that newly erupted maxillary incisors can frequently exhibit fluorosis only in the mammelons (the "snowcapping" effect), and these mammelons are worn away a few years later. Table 1 displays the mean age of each cohort at the time of examination, and no age difference between these cohorts is seen. The possibility of an age effect was thus dismissed.

Another possibility was examiner error. This would account for our results if the examiners were using more stringent criteria for fluorosis when they examined those born in 1985-87 than they did for those born in 1990-92. The examinations were conducted over 6 years, and "examiner drift" with respect to criteria is always a possibility in studies running for that period of time. Inter-examiner consistency scores for fluorosis (kappa) were 0.67 in Phase 1 and 0.55 in Phase 2, scores which demonstrate a moderate level of *inter-examiner* agreement, and which are similar to the level of *intra-examiner* agreement reported from the Hong Kong studies (Evans and Stamm, 1991a). However, low precision in *inter-examiner* scores biases toward the null, so that a lower level of *inter-examiner* agreement would tend to make a significant difference between cohort scores less, rather than more, likely. We found in Phase 1 that one examiner scored higher than the other, so we looked at the fluorosis distribution for each examiner separately. These distributions are shown in Fig. 2. What this graphic demonstrates is that one examiner scored consistently higher than the other throughout the study, but the patterns and the inter-cohort relationships were much the same for both examiners. The higher-scoring examiner also saw 57% of the 1896 children and the lower-scoring examiner 43%, so the combined data are weighted in favor of the higher-scoring examiner. It is therefore possible that the extent of the differences between the cohorts was exaggerated, even though the basic pattern was the same for both examiners. While the possibility of "examiner drift" cannot be excluded, we think that our standardization procedures and frequent consultation in the field make it unlikely that the rejection of the hypothesis resulted from examiner error. If "examiner drift" did occur, both examiners "drifted" together, an occurrence which also seems unlikely.

If our results are not due to age bias or examiner error, the observed pattern of high fluorosis in children born in 1985-87 could be real. That could occur if children in those cohorts were exposed to unusually high levels of ingested fluoride during their early years, though there is nothing in our data to support such a happening. Formula feeding in infancy, which has been cited as a risk factor for fluorosis (Pendrys *et al.*, 1994), was not associated with fluorosis in our study (Table 1), and in any event formula feeding was distributed evenly throughout the cohorts. In addition to cohort effects, fluorosis was associated with more-frequent toothbrushing and the use of fluoride supplements in infancy. We checked for the possibility that these behaviors were concentrated in those born in 1985-87, but they were not: Both were distributed evenly across all cohorts. If the high prevalence of fluorosis in those born in 1985-87 is real, the reason would have to be some excess ingestion of fluoride in the early years. However, the actual source remains unexplained.

The fluorosis effects of this 11-month break in fluoridation, following these Phase 2 results, remain

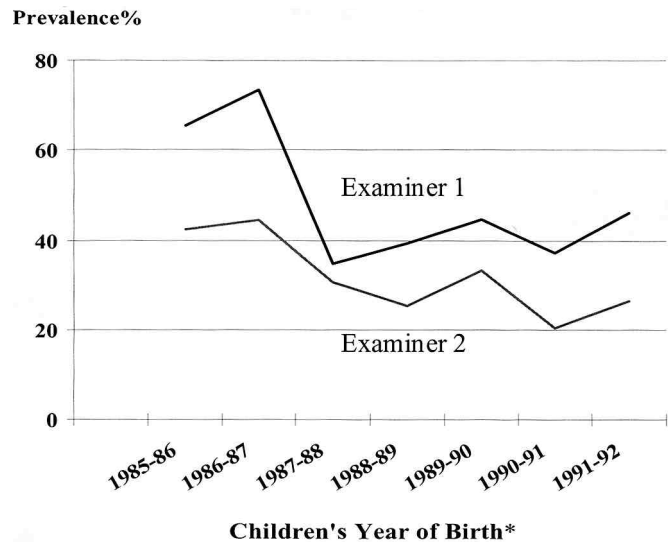


Figure 2. Fluorosis prevalence for each age cohort, scored individually for each examiner. *The birth years cover the period from March 18 in the first year to March 17 in the second, e.g., March 18, 1985, to March 17, 1986.

uncertain. We think it most likely that the break was not long enough to lead to a reduction of fluorosis prevalence, and that the higher prevalence levels seen in children born in 1985-87 are the result of some unexplained exposure to excess fluoride in their infant years. After Phase 1 of this study, we concluded that even the short cessation of exposure to water-borne fluoride seemed to reduce fluorosis prevalence in those cohorts whose teeth were in mid-development. Our results from Phase 2, however, do not permit this conclusion to be supported.

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