CAClinch © 2010

# Delayed Eruption of Teeth & Time at Risk for Cavities

compiled by Carole Clinch BA, BPHE
Research Coordinator
People for Safe Drinking Water

WHY has no one bothered to count the number of teeth in the mouth?

If fluoride delays tooth eruption (70+ studies) isn't it **LOGICAL & INTUITIVE** to assume that if you have **fewer teeth i**n your mouth, then you are likely to have **fewer cavities**?

Water fluoridation studies of the past 2 decades consistently show variations around the "no effect" mark. In other words, there is a similar number of studies showing a small benefit as there are showing no effect or a disbenefit (dental fluorosis, fluoride bombs, increased tooth fractures, increased gingivitis).

The earlier studies showed an apparent benefit for water fluoridation but reanalysis demonstrates that this may be due to a lack of control of many "confounding" factors.

An important confounding variable that requires attention is the issue of delayed eruption of teeth potentially caused by fluoride. According to the York Review (McDonagh et al 2000);

> "no study used an analysis that would control for the frequency of sugar consumption and for the number of erupted teeth"**.**

Second Look Fluoride & delayed eruption

A significant body of research evidence (70+ studies) available since the 1940s (Short et al. 1944) suggests that fluoride delays the eruption of teeth, thereby merely delaying the development of cavities. This delay has been reported to be anywhere from several months (Tseng et al, 1989) to 2 years (Campagna et al, 1995). This delay in the onset of caries due to a delay in eruption of teeth with fluoride use cannot be viewed as a health benefit. Proposed mechanisms for this delayed tooth eruption include**:**

1. Delayed eruption may be due to thickening of the bone around the emerging teeth (Kunzel 1976)

2. Delayed eruption may be due to thyroid hormone suppression by fluoride. Fluoride is well-known to mimic TSH (Thyroid Stimulating Hormone) via activation of G-proteins (proteins located on cell membranes to relay/amplify signals from outside cells to inside cells). (Strunecka et al 2007)

# Delaying the development of cavities is no solution. Preventing cavities is the only solution.

**Selected Quotes**

**NRC 2006, p236**

"A number of authors have reported delayed eruption of teeth, enamel defects, or both, in cases of congenital or juvenile hypothyroidism (Hinrichs 1966; Silverman 1971; Biggerstaff and Rose 1979; Noren and Alm 1983; Loevy et al. 1987; Bhat and Nelson 1989; Mg'ang'a and Chindia 1990; Pirinen 1995; Larsen and Davies 2002; Hirayama et al. 2003; Ionescu et al. 2004). No information was located on enamel defects or effects on eruption of teeth in children with either mild or subclinical hypothyroidism. The possibility that either dental fluorosis (Chapter 4) or the delayed tooth eruption noted with high fluoride intake (Chapter 4; see also Short 1944) may be attributable at least in part to an effect of fluoride on thyroid function has not been studied."

**Dr. Kathleen Thiessen PhD, committee member of NRC Review 2006**

"The small apparent benefits of fluoride exposure seen in some studies are likely due to the effect of delayed tooth eruption (e.g., Komárek et al. 2005)—permanent teeth erupt later in children in fluoridated areas, and thus have been exposed to a cariogenic environment for a shorter time than teeth of children of comparable age in unfluoridated areas. Although delayed tooth eruption as a result of fluoride exposure has been known since the 1940s (Short et al. 1944), this effect has not been considered in most studies reporting caries-reducing effects of fluoride." *Source: Thiessen K 2009 Comments on Prioritization of Chemicals for Carcinogen Identification Committee Review: Proposed Chemicals for Committee Consideration and Consultation March 2009 Proposition 65 Implementation, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency* http://www.oehha.ca.gov/prop65/public_meetings/052909coms/fluoride/SENESFluoride.pdf

"Neither McDonagh et al. (2000) nor the ADA (2005) mention that fluoride exposure appears to delay the eruption of permanent teeth, although this has been known since the 1940s (Short 1944; NRC 2006a). A delay in tooth eruption alters the curve of caries rates with respect to age and complicates the analysis of age-specific caries rates (Psoter et al. 2005; Alvarez 1995; Alvarez and Navia 1989). Komárek et al. (2005) have calculated that the delay in tooth eruption due to fluoride intake may explain the apparent reduction in caries rates observed when comparisons are made at a given age, as is usually done." *Source: Thiessen K 2009 Comments on Fluoride in Drinking Water Document for public comment, September 2009, Prepared by the Federal-Provincial-Territorial Committee on Drinking Water, Health Canada* http://www.newmediaexplorer.org/chris/Kathleen_Thiessen_Nov27_2009_Health_Canada.pdf

**Sutton PRN. The Question of Delayed Onset of Caries. 1996; [in] The Greatest Fraud Fluoridation. ISBN 0949491128**

“There have been suggestions that the "decreases" in dental caries reported from fluoridation trials may be merely a statistical artifact due to a delay in the onset of the caries process.

This question was considered in 1980 in the present author's book Fluoridation: Scientific Criticisms and Fluoride Dangers. It cited the studies by Dr Robert Weaver in 1944 and 1948 in North Shields and the "naturally fluoridated" South Shields. He said in 1948:

"I think that the most important lesson to be learned from the North and South Shields investigation is that the caries-inhibitory property of fluorine seems to be of rather short duration." He concluded: "...there is in fact no very striking difference in the incidence of caries in the two towns."

A professional statistician, K.K. Paluev, reported in 1957 that there had been a similar delay in the rates published after ten years in the reports from the two fluoridation trial cities of Grand Rapids and Newburgh.

In 1978 Professor Arvid Carlsson illustrated the results obtained by Forsman, who in 1974 surveyed the caries rates in two towns in Sweden which had 1.2 and 0.2 ppm fluoride in their water supplies. Professor Carlsson stated:

> "The difference is so small that it corresponds to a retardation of the caries progress by about 1 year. The possibility that the difference is only apparent cannot be excluded, since it is well known that the effect of fluoride is to retard the diagnosis of caries lesions."

The Royal College of Physicians stated in 1976 that in the U.K. studies by the Department of Health, in the age group 8 to 11 years: "...it appears that fluoridation merely postpones caries by about 0.8 cavities a year."

In their paper entitled "The failure of fluoridation in the United Kingdom," when discussing the final report of that U.K. Health Department study, Professor A. Schatz and Dr J.J. Martin stated in 1972:

> "It is thus clear that fluoridation does not prevent or reduce tooth decay. Instead, it merely postpones the appearance of caries by about 1.2 years. Fluoridated children develop the same amount of tooth decay as their non fluoridated counterparts. The only difference is that caries starts developing approximately 1.2 years later in the fluoridated group."

This delay, at least partly, could be due to the teeth of children in fluoridated areas erupting (breaking through the gums) at a slightly older age, and therefore being exposed to decay-producing factors for a shorter period.

Although the number of erupted teeth at each age was not stated in any of the four main trials, in the Newburgh one it was possible to calculate from the published data that the number of erupted teeth was less than expected. The data published from the Evanston study suggested that there had been a progressive decline in the number of erupted first permanent molar teeth in six-year-old children in Evanston between the commencement of the study in 1946 and 1951. Unfortunately, this trend could not be studied because, after that time, the authors ceased publishing the data they obtained for the first

permanent molars.

Drs R. Feltman and G. Kosel (1961) over a period of fourteen years fed fluoride tablets to children

> "through their eighth year of life" and reported that there was: "... a delay in the eruption of the teeth in some cases by as much as a year from the accepted eruption dates."

Dr J.W. Benfield remarked that three years after New York was fluoridated it became apparent that delays of two to three years in eruption time were common, and that there appeared to be an increase in the need for orthodontic treatment. However those clinical impressions were not written up into a formal study. The physiological basis for this delay in eruption was mentioned in discussing the hypothesis, that fluoridation increases orthodontic problems (Sutton, 1988a).

Unfortunately this extremely important question still has not been resolved because the published data are so scarce. It is remarkable that in the British official experiments no count appears to have been made of the number of teeth erupted, or if it was, the data have never been published.
In 1960 Lord Douglas of Barloch referred to the possible delay in the eruption of teeth, and stated: "If this is so, it is a matter of grave concern for it indicates a profound physiological change."

In a dental examination it is standard practice to record, for each person, the teeth which are decayed, filled or missing due to having been extracted or shed, and those which have not yet erupted. Therefore it is a very simple matter to determine, for each sex group, the average number of each type of tooth, and the total number of teeth, which have erupted at each age. However, even as recently as 1979, Whittle and Downer, published a report in the British Dental Journal on a fluoridation study in Birmingham, U.K., which failed to mention this vital subject. Both of these workers, although government employees, had post-graduate degrees in science.

However, the obvious importance of this factor was recognized by a general dental practitioner who made the examinations in a small-scale survey in the town of Bacchus Marsh, Victoria, in the "baseline examination" in 1963 (fluoridation had commenced in 1962). He recorded the number of erupted teeth in the 322 children (aged 5, 8, 11 and 14 years) who drank fluoridated water in the town. (Wood, 1975) The fact that practically no reports on fluoridation trials state these obviously-available data, shows that the authors either did not realize the importance of this factor, despite its mention on numerous occasions in the dental literature, or that they intentionally suppressed the results.

In 1963, Conner and Harwood stated that in their study in Brandon, Canada: "A record was kept of all erupted permanent teeth", but they did not publish their findings.

The suggestion has been made repeatedly that fluoride inhibits thyroid function, which in turn delays the eruption of teeth (e.g. Baume and Becks, 1954) In 1979, Drs L. Krook and G.A. Maylin described a mechanism which could have produced the considerable delay in the eruption of the teeth, of between 1.5 and 3.0 years, which occurred in cattle which were crippled with fluorosis (fluoride damage to bone) due to having been exposed to atmospheric fluoride pollution. They found that exposure to fluoride had produced a great decrease in the number of certain cells in bone (resorbing osteocytes) which play a major role in the resorption of the roots of the deciduous (first) teeth and of bone, both of

which processes are necessary before permanent teeth can erupt normally. They stated:

> "The delay in the eruption of the permanent teeth has also been reported in children in fluoridated communities. The cause of the delay in eruption was shown in the present material. Fluoride arrests resorption of deciduous tooth roots and of the supporting bone. By inducing one disease [fluorosis], delays the manifestations of another [dental caries]."

One more factor which could produce what appears to be a delay in the onset of caries is unconscious bias in favour of fluoridation if the examiners have already formed the opinion that fluoridation reduces caries. Such a bias could affect their assessment when they are determining whether very early caries is present - a matter for their personal judgement and opinion.

That is, there could be delay, not in the development of caries but in its recognition and recording in children in the fluoridated areas (and possibly the exaggeration of the caries score in nonfluoridated ones) Either or both of those processes would produce the illusion that dental caries had been delayed in the fluoridated cities.

This failure to avoid bias is a major deficiency in fluoridation trials and, to the scientist, the fact that observations were not made "blind' must make suspect all the results reported from such a trial, particularly as the dental examination of each child was made by only one examiner and there is no way of checking the results of the examinations.

A similarly biased result is obtained when the examiners are directed, after the commencement of the study to alter their criteria for recording the presence of caries so that the number of carious cavities filled is reduced. This occurred in the fluoridation trial in Hastings, New Zealand. After the initial examination, the dental "nurses" (who examine and fill the teeth of school children) were told by the experimenter who was conducting the trial, T.G. Ludwig, to alter their criteria for determining whether a tooth should be filled.

As a result of this change, in the fluoridated children, they filled only about a quarter of the very small carious cavities which they would have done using their former examination criteria (Colquhoun and Mann, 1986).

This change greatly reduced the number of filled teeth in the fluoridated test area. As the examiner carried out his examination soon after the children's dental treatment was completed, it was not necessary for him to examine for decayed teeth. His task when examining the fluoridated Hastings children was the straightforward one of totalling the small number of missing teeth and those which had been filled which, due to his instructions to the dental "nurses", was much smaller than it would have been if they had filled the teeth judged to be carious using their former criteria, which were still in use everywhere else in New Zealand.

These changes in the criteria for assessing the presence of caries were not mentioned by the author in any of his reports on his trial, they were revealed by Colquhoun and Mann (1986), who found out about these instructions by using the provisions of the freedom of information legislation.”

**Waldbott GL, Burgsthaler AW, McKinney HL 1978 The Great Dilemma. Coronado Press, Lawrence, Texas. P 185-190**

"Another effect associated with increased fluoride intake and with mottling is a delay in the eruption of teeth. This was recognized by a number of early investigators and was later attributed to fluoride-induced suppression of thyroid function [Feltman 1961] which laboratory studies have shown to be a direct cause of retarded tooth eruption. [Garren 1955]

Short 1944-Colorado Springs [2.5ppm] permanent teeth of children exhibited "an appreciably lower eruption rate" than in low-fluoride cities.

Garren 1955-long-term investigation of the administration of fluoride tablets to pregnant mothers and to children up to age 9, resulting a marked "delay in the eruption of the teeth, in some cases by as much as a year from the accepted eruption dates".

In Newburgh, New York, after 10 years of fluoridation, the average number of erupted permanent teeth per 9 to 12 year old children, was 9.35 compared with 9.82 in the non-fluoridated control city of Kingston. [Ast 1956]

In the original report, it was declared that fluoridation was successful in reducing cavities. In **1998 the cumulative results of this fifty-year fluoridation experiment** involving Kingston, New York (un-fluoridated) and Newburg, New York (fluoridated) were published. In summary, there is **no overall significant difference in rates of dental decay** in children in the two cities, but children in the fluoridated city show significantly **higher rates of dental fluorosis** than children in the un-fluoridated city. **SOURCE:** Kumar, J.V. and Green, E.L. 1998 Recommendations for fluoride use in children. New York State Dent. J. 40-47.

In Brazil, "a delay in tooth eruption" was found with fluoridation [Freitas 1971] was found as well.

Weaver 1944 compared decay rates between a naturally fluoridated community [1.4ppm-South Shields] to a lower-fluoridated community [North Shields 0.25ppm]. He commented:

"such a comparison can be most misleading. The question which really needs to be answered is: How many years does it take for the figure 2.4 DMF permanent teeth in South Shields to reach 4.3 in North Shields? The answer is approximately three years… the fact remains that children 15 years of age in South Shields have the same average amount of caries as is found in North Shields at 12 years of age."

Paulev 1955 – a professional statistician and research engineer carried out a similar analysis of the 10 year DMF figures from fluoridated Grand Rapids and Newburgh, New York. He found the same phenomenon – when you take into account the delayed tooth eruption, no difference in caries can be found.

Ziegelbecker 1970 reanalyzed older fluoridation studies and showed that the annual increments in tooth decay among older children in the nonfluoridated control communities decline faster than in the fluoridated ones. This nullifies any apparent, initial benefit of fluoridation.

Kunzel 1976 writes: "The **delayed eruption of all premolars in children of the area with optimally fluoridated water was the only systematic effect which could be detected.** This normalization is explained by a prolonged stay of the deciduous teeth in the dental arch which is due to a lesser caries prevalence."

Ainsworth in Britain 1933, Ast 1956 in USA, Garren 1955, Freitas 1971 in Brazil, Masaki in Japan 1931, Lemmon in USA 1934, Pauley 1955 etc. **- all reported a delay in the eruption of teeth with fluoridation.**

1949 Official report of 11 years of fluoridation in the UK showed that the amount of caries increase from **permanent teeth** from age 8 to 14 was virtually identical [5.1 in fluoridated areas vs 5.2 in unfluoridated areas].

**International Academy of Oral Medicine and Toxicology (IAMOT) Policy Position on Ingested Fluoride and Fluoridation. http://www.iaomt.org/articledetails.cfm?artid=97**

> Fluoride has produced considerable delay in the eruption of children's teeth.[51] Drs. L. Krook and G. A. Maylin 1983 describe a mechanism that produces marked delay in the eruption of teeth (1.5 to 3.0 years) in **cattle** crippled with fluorosis (fluoride damage to bone), due to **atmospheric fluoride pollution.**
>
> There are several studies which have found a **delay in tooth eruption** for children ingesting fluoride from the drinking water ( Sutton[44], Limeback[45], NIDR 1987). The delay in eruption **fully accounts for the transient reduction in tooth decay** seen in the 5 to 8 year old children (Yiamouyiannis [46]).
>
> The delay in eruption is not a health benefit, but is indicative of a **generalized slow-down in the growth of the child** that has enormous implications for the future health of that child.
>
> **Krook et. al.** found that exposure to fluoride had produced a great decrease in the number of certain cells in bone (resorbing osteocytes) which play a major role in the resporption of the roots of the deciduous (first) teeth and of bone; both of which processes are necessary before permanent teeth can erupt normally. They stated:
>
> > "The delay in eruption in the permanent teeth has also been reported in children in fluoridated communities." "The cause of the delay in eruption was shown in the present material. Fluoride arrests resorption of deciduous tooth roots and of the supporting bone. By inducing one disease (fluorosis), fluoride delays the manifestations of another (dental caries)[52]."

**Schuld 2005 Is Dental Fluorosis Caused by Thyroid Hormone Disturbances? Fluoride 2005 35(2): 91-94.** http://www.fluorideresearch.org/382/files/38291-94.pdf

> Although it has been known since 1917 that mottled dental enamel (later recognized as dental

fluorosis—DF) is identical with that observed in thyroid dysfunction, disturbances of thyroid hormone metabolism during crucial periods of tooth development as the primary cause of DF have received very little consideration by dental researchers. New findings indicate that thyroid hormone metabolism is disturbed in peripheral tissue of children with DF, thereby helping to account for timing of events observed in DF and the delayed eruption of teeth in fluoridated areas and further suggesting the use of DF as a marker and diagnostic aid for iodine deficiency disorders.

Perhaps the most obvious indication that DF is a condition caused by disordered thyroid hormone signaling during the time of enamel development is the **long-standing observation of delayed eruption of teeth in fluoridated areas.**9 DF is invariably associated with dental age and eruption of teeth, a process closely controlled by thyroid hormone (TH).

> **TH deficiency leads to delayed tooth eruption**,
> **TH excess leads to the acceleration of tooth eruption**.

The more fluoride ingested, the longer it takes for the tooth to erupt. The later in life maturation of enamel is completed, the greater is the severity of dental fluorosis.10

At the same time, other risk factors known to influence DF are identical to those observed in thyroid dysfunction. Thus, while DF gets more severe at **higher altitudes,** the same is generally true for iodine deficiency.11

Furthermore, while the frequency of DF is **significantly greater among Blacks**, it is now known that they also have a more sensitive thyroid status.12

It is well established that DF can only occur as a result of excessive fluoride exposure during crucial times of development—*in utero to approximately 30 months for deciduous teeth and permanent incisors*— and **is marked by events related to *timing*.** Thus, it is associated with

3. ***delayed*** **tooth eruption,9,10**
4. ***delayed*** **removal of enamel matrix proteins,13**
5. ***delayed*** **enamel maturation,10,13 etc.,**

clearly indicating that a **tissue-specific differentiation program is being disturbed**.

Endocrinology has firmly established **TH to be the crucial regulator of all tissuespecific differentiation programs during development**. Appropriate TH levels at the precise time are critically important for the coordination of developmental processes. This is most clearly demonstrated in amphibia, in which **metamorphosis does not occur in thyroidectomized larvae unless TH is present**. The metamorphic transitions of individual organs in amphibia are all controlled by TH, with
each event occurring at distinct developmental stages, requiring correct spatial and temporal manner.14 (Since the 1930s fluoride has been known to inhibit and delay metamorphosis in amphibia.6 Just a coincidence?)

In the physiology of human development, the importance of TH is especially evident in the **central nervous system** in which TH deficiency during fetal and neonatal periods can lead to morphological and functional abnormalities, the most severe manifestation of which is **cretinism.**

The findings by **AK Susheela** and co-workers, as published in this issue of *Fluoride*, 15 present not only the **first reports on TSH and free TH levels in children and adolescents with DF**, but, in addition, show that **even in children without DF—but with elevated fluoride serum levels—abnormal TH metabolism is present, as previously observed in workers exposed to fluoride**,16 as well as in children and adults with various amounts of fluoride in the water supply.6,17 This new evidence indicates that **iodine metabolism is being disturbed in peripheral tissue through manipulation of the deiodinases, the three enzymes which delicately regulate TH metabolism through external TSH/G-protein activation**. The disturbances in TH levels observed are identical with those observed in **iodine deficiency disorders (IDD).**

**TH deficiency during the secretory stage of amelogenesis results in poorly calcified enamel,18 a hallmark of dental fluorosis.** Since it is during this time that any alterations in TH may also influence the neurological development of the child, **enamel defects in deciduous teeth and permanent incisors, such as dental fluorosis, should be used as a marker and aid in the diagnosis of neurological and iodine deficiency disorders, as has been suggested by others**.19-22

10 Rwenyonyi CM, Birkeland JM, Haugejorden O, Bjorvatn K. Dental variables associated with differences in severity of fluorosis within the permanent dentition. Clin Oral Investig. 2000 Mar;4(1):57-63.

19 Noren JG, Alm J. Congenital hypothyroidism and changes in the enamel of deciduous teeth. Acta Paediatr Scand 1983 Jul;72(4):485-9.

20 Noren JG, Gillberg C. Mineralization disturbances in the deciduous teeth of children with so called minimal brain dysfunction. Swed Dent J 1987;11(1-2):37-43.

21 Phyllis J. Mullenix, Ph.D. Letter to Dr. Irwin Kash. June 17, 1999. Available from: http://www.bruha.com/pfpc/html/mullenix2.htm

22 Bhat M, Nelson KB. Developmental enamel defects in primary teeth in children with cerebral palsy, mental retardation, or hearing defects: a review. Adv Dent Res 1989 Sep;3(2):132-42.

**PFPC-Delayed Eruption ©2001-2002 PFPC** http://bruha.com/science/html/tooth_eruption.html

In 1911 it was first proposed that hormonal factors were involved in the regulation of tooth eruption (Keith, 1911). A few years later Erdheim (1914) reported on dental effects of thyroidectomy in rats, and Biedl (1914) did the same from a study in dogs. Hoskins (1928) and Karnofsky (1939) reported that hyperthyroidism produced accelerated tooth eruption and the rate of incisor tooth eruption was markedly accelerated by the injection of thyroxine, while Ziskin et al. (1940) revealed retardation in eruption time, as well as dentin and root development, in the opposite state - hypothyroidism.

Detailed investigations by Baume, Becks & Evans (1954) confirmed that thyroid hormones controlled tooth eruption.

It is now well established that in hypothyroid children the eruption of primary and permanent teeth is delayed, and the teeth may have hypoplastic enamel - “dental fluorosis”, while hyperthyroid children, on the other hand,  will have accelerated tooth eruption.

The greater dose of fluoride is taken up by the organism in the period of development the longer is the retardation of dental age (i.e. Szelag,1990).

This retardation in dental age is a clear sign that thyroid hormone activity has been disturbed.

The later a tooth erupts, the more severe is the “dental fluorosis” (i.e. Larson et al, 1987;van Palenstein et al, 1997)

==========

If fluoride is an anti-thyroid, delayed tooth eruption should obviously be observable in fluoridated areas or as a result of other fluoride supplementation measures.
To view some tables showing this, see:
Newark Brantford Grand Rapids Evanston

**Newark, Delaware**

| | Decayed-Missing-Filled Teeth | | Percent Caries-Free Children | |
|---|---|---|---|---|
| Age | After Fluoridation | Before Fluoridation | After Fluoridation | Before Fluoridation |
| 6 | 0.2 | 1.1 | 88.8 | 54.8 |
| 7 | 1.1 | 2.3 | 44.9 | 22.7 |
| 8 | 1.7 | 2.9 | 31.5 | 8.6 |
| 9 | 2.8 | 3.7 | 11.3 | 4.8 |
| 10 | 3.4 | 4.9 | 6.4 | 7.5 |

Reference: Journal American Dental Assoc. Vol. 54, June 1957
Note: 1-year DELAY in DMF per child. At age 10 FEWER caries-free children AFTER fluoridation than BEFORE.

Brantford, Ontario
Before and after 10 years fluoridation

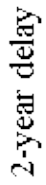
| Age | 1955 | 1945 |
|---|---|---|
| 6 | 0.16 | 0.40 |
| 7 | 0.55 | 1.66 |
| 8 | 1.13 | 2.44 |
| 9 | 1.71 | 3.18 |
| 10 | 2.21 | 3.80 |
| 11 | 2.84 | 4.68 |
| 12 | 3.30 | 6.31 |
| 13 | 4.52 | 7.78 |
| 14 | 5.54 | 8.60 |
| 15 | 6.51 | 9.94 |

2-year delay

1959 Report
Dept. Nat. Health and Welfare, Ottawa

Grand Rapids, Michigan
Decayed - Missing - Filled Teeth per child
Before and after 10 years fluoridation

| Age | 1954 | 1944 |
|---|---|---|
| 6 | 0.19 | 0.78 |
| 7 | 0.69 | 1.89 |
| 8 | 1.27 | 2.95 |
| 9 | 1.97 | 3.90 |
| 10 | 2.34 | 4.92 |
| 11 | 2.98 | 6.41 |
| 12 | 3.87 | 8.07 |
| 13 | 5.05 | 9.73 |
| 14 | 6.78 | 10.95 |
| 15 | 8.07 | 12.48 |
| 16 | 9.95 | 13.50 |




Official Public Health Service Data
Public Health Reports 71:652, 1956

Evanston, Illinois
Decayed - Missing - Filled Teeth per child
Before and after 12 years fluoridation

| Age | 1958 | 1946 |
|---|---|---|
| 6 | 0.04 | 0.46 |
| 7 | 0.53 | 1.53 |
| 8 | 0.93 | 2.49 |
| 9 | 2.00 | 3.60 |
| 10 | 2.40 | 4.80 |
| 11 | 3.00 | 6.00 |
| 12 | 3.56 | 7.63 |
| 13 | 5.11 | 10.09 |
| 14 | 7.25 | 11.65 |


J. R. Blayney, D.D.S.
at Hearings, Schuringa et al versus City of Chicago
Values for 9-10-11 interpolated from graph.

# Bibliography: Fluoride & Delayed Eruption of Teeth

Ainsworth NJ. Mottled teeth. British Dental Journal 1933;55: 233-250.

Armfield JM, Spencer AJ. Consumption of nonpublic water: implications for children's caries experiences. Community Dent Oral Epidemiol 2004;32:283-96. [abstract and comments by Dr Mark Diesendorf in Fluoride 2004;37:316-7].

Ast DB, Smith DJ, Wachs B, Cantwell, KT - "Newburgh-Kingston Caries-Fluorine Study;XIV. Combined Clinical and Roentgenographic Dental Findings After Ten Years of Fluoride Experience" JADA 1956;52:314-325.

Backer Dirks O. The benefits of water fluoridation. Caries Res 1974;8 Suppl 1:2-15.
Baume LJ, Becks H. Hormonal Control of Tooth Eruption. Part I , J Dent Res 1954;33: 80-103.

Baume LJ, Becks H. Hormonal Control of Tooth Eruption. Part III, J Dent Res 1954; 33:104-114.

Bauer P, Binder K, Bukovics E, Daimer I, Keresztesi K, Kleinert W, Scheiber V, Uberhuber CW, Westphal G, Wohlzogen FX. Eruption of permanent teeth in areas of low and high fluoride content in drinking water. Osterr Z Stomatol 1974;71(5):162-74.

Bauer P, Binder K, Husinsky I, Kleinert W, Kunkel W, Scheiber V, Uberhuber CW, Westphal G, Wohlzogen FX - "Determining the average posteruptive age of teeth from the results of cross-sectional studies" Zahn Mund Kieferheilkd Zentralbl 1978;66(3):227-41

> If results of cross-sectional studies must be used as a basis for setting up models of development of caries in permanent teeth, then the average rather than individual posteruptive age of teeth has to be resorted to. Data available for this purpose had been obtained from a total of 54,000 children living in two G.D.R. cities, namely, Plauen and Karl-Marx-Stadt, as well as from two surveys made in 1959 and 1971, respectively. Calculation of the average posteruptive age of teeth assumes good knowledge of the distribution of dates of eruption in a population. Of the three types of distribution considered, namely, normal, lognormal, and log-logistic distribution, the log-logistic model proved to be superior to others because of better adaptation to the data available. The method of calculating the posteruptive age of teeth is described and illustrated by use of a number of examples. Since one of the two surveys (namely that which was made in Karl-Marx-Stadt in 1971) was made after twelve years' drinking water fluoridation, it was possible to study the effect of fluoridation upon the eruptive behavior of permanent teeth. The results of this study confirmed those obtained during previous investigations.

Biedl A. Innere Sekretion. Urban & Schwarzenberg, publs. Berlin (1914)

Bigeard L, Obry-Musset MA. Eruption pattern of permanent teeth in a transversal sample of children from Strasbourg. Faculté de Chirurgie, Strasbourg, France ABSTRACTS from 3nd EADPH Congress September 8-9, 1999, Strasbourg, France. Originally Published in Community Dental Health Volume

16, Number 3
http://www.dundee.ac.uk/dhsru/cdh/ab163.htm

> "The delay in tooth eruption was in line with previous reports from Europe and USA for fluoridated areas, and concerned in particular the canines and premolars. The principle reasons of a general retardation of tooth eruption need to be analysed taking into consideration the fluoride intake, the oral health improvement but also the general growth and maturation of the children."

Campagna L, Tsamtsouris A, Kavadia K. Fluoridated drinking water and maturation of permanent teeth at age 12. J Clin Pediatr Dent 1995;19(3):225-8.

> Department of Pediatric Dentistry, Tufts University School of Dental Medicine, Boston, MA 02111, USA.
> Dental age was evaluated in 88 children aged 12 years + 6 months in both areas of Boston with fluoridated drinking water and nonfluoridated areas surrounding Athens, Greece. Fluoridation of drinking water in the Boston areas was 1.0 ppm, a level considered 'optimal' in the USA. **Girls from the fluoridated Boston area were shown in this study to have a significantly ($p<0.05$) delayed dental age when compared to their chronological age according to the tables of Nolla.** Boys from the Boston area and boys and girls from the Athens area showed no significant difference when comparing dental age to chronological age.

Cowan. Fluorine and dental caries. Med Officer 1952;88:281-2.

Feltman R, Kosel G. Prenatal and postnatal ingestion of fluorides - Fourteen years of investigation - Final report. Journal of Dental Medicine 1961;16: 190-9.

Deatheridge SF. Fluori Domestic Waters and Dental Caries Experience. J Dent Res 1943; 22:129-37.

De Eds - American Journal Med Sciences 1942;203(5):687-92.

De Eds - JADA 1936;April, 568-74.

Erdheim J. Rachitis und Epithelkoerperchen. Akad der Wissensch 1914;90:363.

Feltman R. Prenatal and Postnatal Ingestion of Fluorides. A Progress Report. Dental Digest, August 1956.

Feltman R, Kosel G. Prenatal and Postnatal Ingestion of Fluorides - Fourteen Years of Investigation - Final Report. J Dent Med 1961;16:190-199.

Freitas JA, et al. Influence of fluoridation in the chronology of eruption of permanent teeth. Estomatologia e Cultura 1971;5: 156-165.

Garren L, Greep R. Effects of thyroid hormones and propylthiouracil on eruption rate of upper incisor teeth in rats. Proceedings of the Society for Experimental Biology and Medicine 1955;90:652-655.

Haikel Y, Cahen PM, Turlot JC, Frank RM. Dental caries and fluorosis in children from high and low fluoride areas of Morocco. Assoc J Dent Child 1989;56:5, 378-381.

> The percentage and average number of erupted permanent teeth were higher in Beni Mellal (no-fluoride) than in Khouribga for 11-12 and 13-14-year-old age-groups.

Hoskins MM. The effect of acetyl thyroxin on the teeth of newborn rats. Proc Soc Exper Biol & Med 1927-28;25:55.

Jonas I, Schienle R - "Changes in tooth morphology as affected by fluoridated drinking water" Schweiz Monatsschr Zahnmed 94(5):399-4089 (1984)

Karnofsky D, Cronkite EP. Effect of thyroxine on eruption of teeth in newborn rats. Proc Soc Exper Biol & Med 1939;40:458.

Keith A. An inquiry into the nature of acromegaly. Lancet 1911;1:993.

Kick CH, Bethke RM, Edgington BH, Wilder OHM, Record PR, Wilder W, Hill TJ, Chase SW. Fluorine in Animal Nutrition. Bulletin 558, US Agricultural Experiment Station, Wooster, Ohio (1935).

KIrzIoğlu Z, Sağlam AMS, Simşek S. Occlusal disharmonies of primary dentition in a high and low fluoride area of Turkey. Fluoride 2005;38(1):57–64.

> In the high fluoride area, anterior crossbite was significantly higher ($p<0.001$), whereas anterior openbite and anterior crowding were significantly lower ($p<0.05$) in the high fluoride area compared to the low fluoride area.

Komarek A, Lesaffre E, Harkanen T, Declerck D, Virtanen JI. A Bayesian analysis of multivariate doubly-interval-censored dental data. Biostatistics 2005;6:145-55.

Krook L, Maylin GA, Lillie JH, Wallace RS. Dental fluorosis in cattle. Cornell-Vet 1983; 73(4):340-62. (See abstract)

> Five expressions of dental fluorosis are described in cattle exposed to industrial fluoride pollution:
> 1. Hypercementosis with tooth ankylosis, cementum necrosis and cyst formation;
> 2. Delayed eruption of permanent incisor teeth;
> 3 Necrosis of alveolar bone with recession of bone and gingiva;
> 4. Oblique eruption of permanent teeth, hypoplasia of teeth with diastemata; and
> 5. Rapid progression of dental lesions.
> The five entities are not recognized in the "standard for the classification of dental fluorosis" by the National Academy of Sciences. Since this classification it too limited and superficial,

adherence to this standard has left severe cases of fluoride intoxication in cattle undetected in field surveys.

Krylov SS, Pettsol'd K. [Deciduous tooth eruption and fluorosis in the case of increased fluorine content in the drinking water] Stomatologiia (Mosk). 1982 Jan-Feb;61(1):75-7. Russian. No abstract available.

Kunzel VW. Cross-sectional comparison of the median eruption time for permanent teeth in children from fluoride poor and optimally fluoridated areas. Stomatol DDR 1976;5:310-21.

Nearly 57000 children (aged from 4 years, 4 months to 15 years, 9 months) of Karl-Marx-Stadt (1.0 ppm F) and Plauen (0.2 ppm F) were examined to compare the mean eruption times of permanent teeth before and after 12 years of water fluoridation. Whereas a direct influence of internally administered fluorides is to be excluded, an indirect action on the premolars may be assumed with certainty. The delayed eruption of all premolars in children of the area with optimally fluoridated water was the only systematic effect which could be detected. This normalization is explained by a prolonged stay of the deciduous teeth in the dental arch which is due to a lesser caries prevalence.

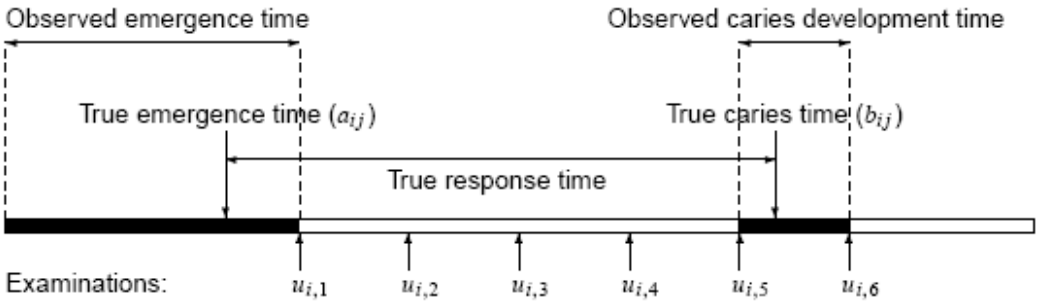



Fig. 1. Possible evolution of a sample tooth and resulting observations. This scheme shows the $j$th tooth of the $i$th child whose emergence time is left-censored at time $u_{i,1}$ and the time until caries is interval-censored in the interval $(u_{i,5}, u_{i,6}]$.

Kunzel W, Bluthner K. Comparson of occlusal conditions in juveniles from residential areas with different drinking water fluoride contents. Zahn Mund Kieferheilkd Zentralbl 1979;67(6):563-71.

The investigations reported in this paper were conducted in Karl-Marx-Stadt after 16 years from the introduction of drinking water fluoridation (1.0 ppm F), with a total of 792 randomly selected juveniles (whose average age was 14.9 years) being examined. Serving as a control group were 780 juveniles of the same age (15.4 years). The control persons were from Leipzig (0.2 ppm F). Included in the comparison with metric occlusal traits, use being made in this connection of the method for measuring occlusal traits, which was recommended in 1973 by an F.D.I. commission (COCSTOC-MOT). Despite the considerable reduction of caries in the deciduous dentition and the change in the eruptive behavior of permanent teeth, which is of great importance of the development, of dentition, no systematic differences could be observed

between the two groups of subjects. An improvement in the occlusal conditions could not, after consumption of drinking water with an optimum fluoride content, be convincingly demonstrated by means of the epidemiological method used in these investigations.

Kunzel W. [Dependence of the dentition behavior of the permanent teeth on the persistence of deciduous teeth] Zahn Mund Kieferheilkd Zentralbl. 1984;72(5):411-9. German. No abstract available.

Kunzel W, Arnold A. [Influence of internal fluoride uptake on the eruption of deciduous teeth] Zahn Mund Kieferheilkd Zentralbl. 1977;65(2):168-75. German

The possibility of influencing the eruptive behavior of deciduous teeth through the intake of fluorine is analyzed by comparing the average dates of eruption determined for **several thousand subjects** living in regions in which the drinking water has low and optimum contents of fluorine.

Kunzel W. Influence of water fluoridation on the eruption of permanent teeth. Caries Res 1976;10(2):96-103.

Larsen MJ, Kirkegaard E, Poulsen S. Patterns of dental fluorosis in a European country in relation to the fluoride concentration of drinking water. J Dent Res 1987;66(1):10-2

"It was found that the later in childhood the tooth was formed, the higher was the prevalence of dental fluorosis. The fluoride concentration in the drinking water affected the prevalence of dental fluorosis in all teeth except the lower incisors, which are formed very early in life... the actual amount of increase in the prevalence and degree of fluorosis was greatest among those teeth that formed later during childhood."

Larsen MJ, Senderovitz F, Kirkegaard E, Poulsen S, Fejerskov O - "Dental fluorosis in the primary and the permanent dentition in fluoridated areas with consumption of either powdered milk or natural cow's milk." J Dent Res 1988;67(5):822-5.

Larsen MJ, Kirkegaard E, Poulsen S, Fejerskov O "Dental fluorosis among participants in a non-supervised fluoride tablet program" Community Dent Oral Epidemiol 1989;17(4):204-6.

Department of Oral Anatomy, Royal Dental College, Aarhus, Denmark.

The aim of the present study was to describe the intraoral pattern of dental fluorosis among fluoride tablet consumers. One hundred and forty-two children, of whom 56 had participated in a fluoride tablet program of 0.5/1.0 mg NaF per day were examined blindly for possible fluoride-induced enamel changes. A low prevalence of dental fluorosis was found among non-participants. The later in life the tooth was formed, the higher was the prevalence. The subjects who had participated in the fluoride tablet program showed a significantly higher prevalence of fluorosis. They could be divided into three groups:

a) Group 1 exhibited a tooth prevalence pattern not statistically different from that of the

non-participants,
b) group 2 showed dental fluorosis in almost all teeth except in those formed before the start of the tablet program.
c) group 3 the early and the late formed teeth showed very little fluorosis while those formed in the few years just after the initiation of the fluoride tablet intake were affected by fluorosis.

Lemmon JR. Mottled enamel of teeth in children. Texas State Journal of Medicine 1934;30: 332-336.

Leroy R, et al. The effect of fluorides and caries in primary teeth on permanent tooth emergence. Community Dentistry and Oral Epidemiology 2003;31(6):463-70.

Catholic University Leuven, School of Dentistry, Oral Pathology and Maxillofacial Surgery, Leuven, Belgium. roos.leroy@med.kuleuven.ac.be

This study addressed two questions:
(i) is there an effect of exposure to fluorides on the timing of emergence of permanent teeth?
(ii) can a difference in timing of tooth emergence be explained by the impact of fluorides on the caries experience of the predecessors?
Data were obtained from a long-term follow-up study of the oral health condition in a sample of 4468 Flemish children. Survival analyses with log-logistic distribution were performed to calculate median emergence ages and 95% confidence intervals; four fluoride exposure parameters
a) fluorosis,
b) use of systemic fluoride supplements,
c) age at which tooth brushing started
d) frequency of tooth brushing
and caries experience were taken as covariates in the model.
The present study indicates that the impact of any of the four fluoride exposure parameters on permanent tooth emergence was relatively minimal. Caries experience in the primary molars had a more pronounced impact on the timing of emergence of the successors than exposure to any of the four fluoride parameters.Jonas I, Schienle R. Changes in tooth morphology as affected by fluoridated drinking water. Schweiz Monatsschr Zahnmed 1984;94(5):399-409.

Karnofsky D, Cronkite EP. Effect of thyroxine on eruption of teeth in newborn rats. Proc Soc Exper Biol & Med 1939;40:458.

Keith A. An inquiry into the nature of acromegaly. Lancet 1911;1:993.

Kick CH, Bethke RM, Edgington BH, Wilder OHM, Record PR, Wilder W, Hill TJ, Chase SW. Fluorine in Animal Nutrition. Bulletin 558, US Agricultural Experiment Station, Wooster, Ohio (1935).

KIrzIoğlu Z, Sağlam AMS, Simşek S. Occlusal disharmonies of primary dentition in a high and low fluoride area of Turkey. Fluoride 2005;38(1):57–64.

In the high fluoride area, anterior crossbite was significantly higher ($p<0.001$), whereas anterior openbite and anterior crowding were significantly lower ($p<0.05$) in the high fluoride area compared to the low fluoride area.

Komarek A, Lesaffre E, Harkanen T, Declerck D, Virtanen JI. A Bayesian analysis of multivariate doubly-interval-censored dental data. Biostatistics 2005;6:145-55.

Krook L, Maylin GA, Lillie JH, Wallace RS. Dental fluorosis in cattle. Cornell-Vet 1983; 73(4):340-62. (See abstract)

Five expressions of dental fluorosis are described in cattle exposed to industrial fluoride pollution:
1. Hypercementosis with tooth ankylosis, cementum necrosis and cyst formation;
2. Delayed eruption of permanent incisor teeth;
3 Necrosis of alveolar bone with recession of bone and gingiva;
4. Oblique eruption of permanent teeth, hypoplasia of teeth with diastemata; and
5. Rapid progression of dental lesions.
The five entities are not recognized in the "standard for the classification of dental fluorosis" by the National Academy of Sciences. Since this classification it too limited and superficial, adherence to this standard has left severe cases of fluoride intoxication in cattle undetected in field surveys.

Krylov SS, Pettsol'd K. [Deciduous tooth eruption and fluorosis in the case of increased fluorine content in the drinking water] Stomatologiia (Mosk). 1982 Jan-Feb;61(1):75-7. Russian. No abstract available.

Kunzel VW. Cross-sectional comparison of the median eruption time for permanent teeth in children from fluoride poor and optimally fluoridated areas. Stomatol DDR 1976;5:310-21.

Nearly 57000 children (aged from 4 years, 4 months to 15 years, 9 months) of Karl-Marx-Stadt (1.0 ppm F) and Plauen (0.2 ppm F) were examined to compare the mean eruption times of permanent teeth before and after 12 years of water fluoridation. Whereas a direct influence of internally administered fluorides is to be excluded, an indirect action on the premolars may be assumed with certainty. The delayed eruption of all premolars in children of the area with optimally fluoridated water was the only systematic effect which could be detected. This normalization is explained by a prolonged stay of the deciduous teeth in the dental arch which is due to a lesser caries prevalence.

Kunzel W, Bluthner K. Comparson of occlusal conditions in juveniles from residential areas with different drinking water fluoride contents. Zahn Mund Kieferheilkd Zentralbl 1979;67(6):563-71.

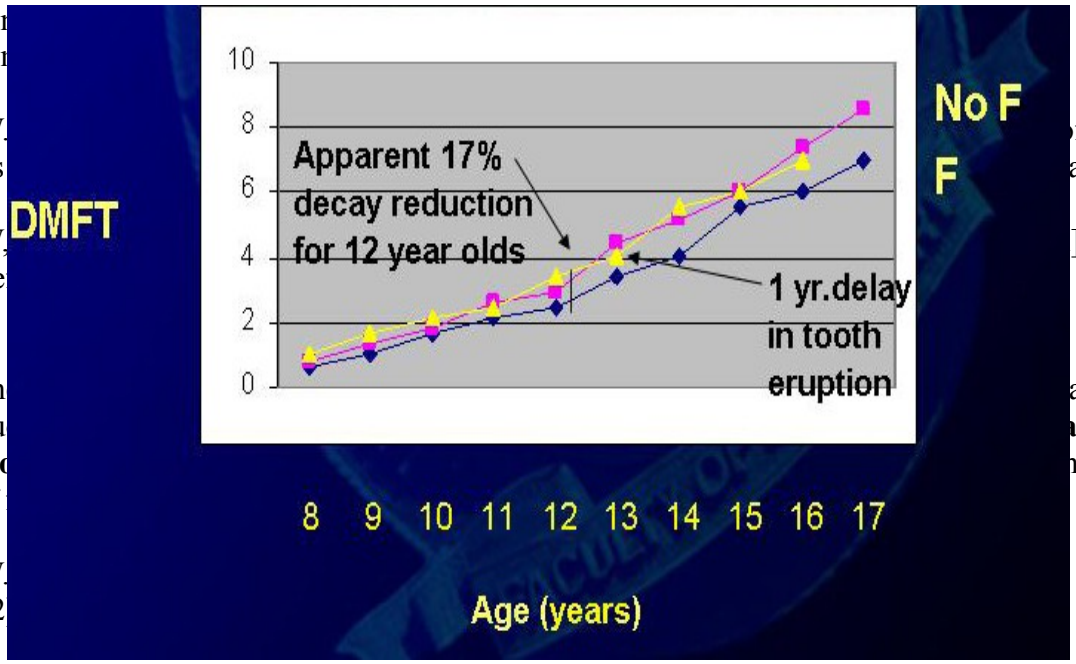
The investigations reported in this paper were conducted in Karl-Marx-Stadt after 16 years from the introduction of drinking water fluoridation (1.0 ppm F), with a total of 792 randomly selected juveniles (whose average age was 14.9 years) being examined. Serving as a control group were 780 juveniles of the same age (15.4 years). The control persons were from Leipzig

**MEAN DMFS OF U.S. CHILDREN WITH PERMANENT TEETH BY AGE AND WATER FLUORIDATION EXPOSURE**

| Age | Life-long Water Fluoridation Exposure Mean DMFS | No Water Fluoridation Exposure Mean DMFS | Percent Difference |
|---|---|---|---|
| 5 | 0.03 | 0.10 | 70 |
| 6 | 0.14 | 0.14 | 0 |
| 7 | 0.36 | 0.53 | 32 |
| 8 | 0.64 | 0.79 | 19 |
| 9 | 1.05 | 1.33 | 21 |
| 10 | 1.64 | 1.85 | 11 |
| 11 | 2.12 | 2.63 | 19 |
| 12 | 2.46 | 2.97 | 17 |
| 13 | 3.43 | 4.41 | 22 |
| 14 | 4.05 | 5.18 | 22 |
| 15 | 5.53 | 6.03 | 8 |
| 16 | 6.02 | 7.41 | 19 |
| 17 | 7.01 | 8.59 | 18 |
| All Ages | 2.79 | 3.39 | 18 |

Brunelle JA, Carlos JP. **Journal of Dental Research** 1990;69 (Spec. Iss.): 723-727.

(0.2 ppm F). Included in the comparison with metric occlusal traits, use being made in this connection of the method for measuring occlusal traits, which was recommended in 1973 by an F.D.I. commission (COCSTOC-MOT). Despite the considerable reduction of caries in the deciduous dentition and the change in the eruptive behavior of permanent teeth, which is of great importance of the development, of dentition, no systematic differences could be observed between the two groups of subjects. An improvement in the occlusal conditions could not, after co
de




Kunzel W. f
deciduous available.

Kunzel W. ] Zahn
Mund Kie

Th ake of
flu **al**
**th** n contents
of

Kunzel W.
1976;10(2

Larsen MJ, Kirkegaard E, Poulsen S. Patterns of dental fluorosis in a European country in relation to

the fluoride concentration of drinking water. J Dent Res 1987;66(1):10-2

> "It was found that the later in childhood the tooth was formed, the higher was the prevalence of dental fluorosis. The fluoride concentration in the drinking water affected the prevalence of dental fluorosis in all teeth except the lower incisors, which are formed very early in life... the actual amount of increase in the prevalence and degree of fluorosis was greatest among those teeth that formed later during childhood."

Larsen MJ, Senderovitz F, Kirkegaard E, Poulsen S, Fejerskov O - "Dental fluorosis in the primary and the permanent dentition in fluoridated areas with consumption of either powdered milk or natural cow's milk." J Dent Res 1988;67(5):822-5.

Larsen MJ, Kirkegaard E, Poulsen S, Fejerskov O "Dental fluorosis among participants in a non-supervised fluoride tablet program" Community Dent Oral Epidemiol 1989;17(4):204-6.

> Department of Oral Anatomy, Royal Dental College, Aarhus, Denmark.
>
> The aim of the present study was to describe the intraoral pattern of dental fluorosis among fluoride tablet consumers. One hundred and forty-two children, of whom 56 had participated in a fluoride tablet program of 0.5/1.0 mg NaF per day were examined blindly for possible fluoride-induced enamel changes. A low prevalence of dental fluorosis was found among non-participants. The later in life the tooth was formed, the higher was the prevalence. The subjects who had participated in the fluoride tablet program showed a significantly higher prevalence of fluorosis. They could be divided into three groups:
>
> d) Group 1 exhibited a tooth prevalence pattern not statistically different from that of the non-participants,
> e) group 2 showed dental fluorosis in almost all teeth except in those formed before the start of the tablet program.
> f) group 3 the early and the late formed teeth showed very little fluorosis while those formed in the few years just after the initiation of the fluoride tablet intake were affected by fluorosis.

Lemmon JR. Mottled enamel of teeth in children. Texas State Journal of Medicine 1934;30: 332-336.

Leroy R, et al. The effect of fluorides and caries in primary teeth on permanent tooth emergence. Community Dentistry and Oral Epidemiology 2003;31(6):463-70.

> Catholic University Leuven, School of Dentistry, Oral Pathology and Maxillofacial Surgery, Leuven, Belgium. roos.leroy@med.kuleuven.ac.be
>
> This study addressed two questions:
> (i) is there an effect of exposure to fluorides on the timing of emergence of permanent teeth?
> (ii) can a difference in timing of tooth emergence be explained by the impact of fluorides on the caries experience of the predecessors?
> Data were obtained from a long-term follow-up study of the oral health condition in a sample of

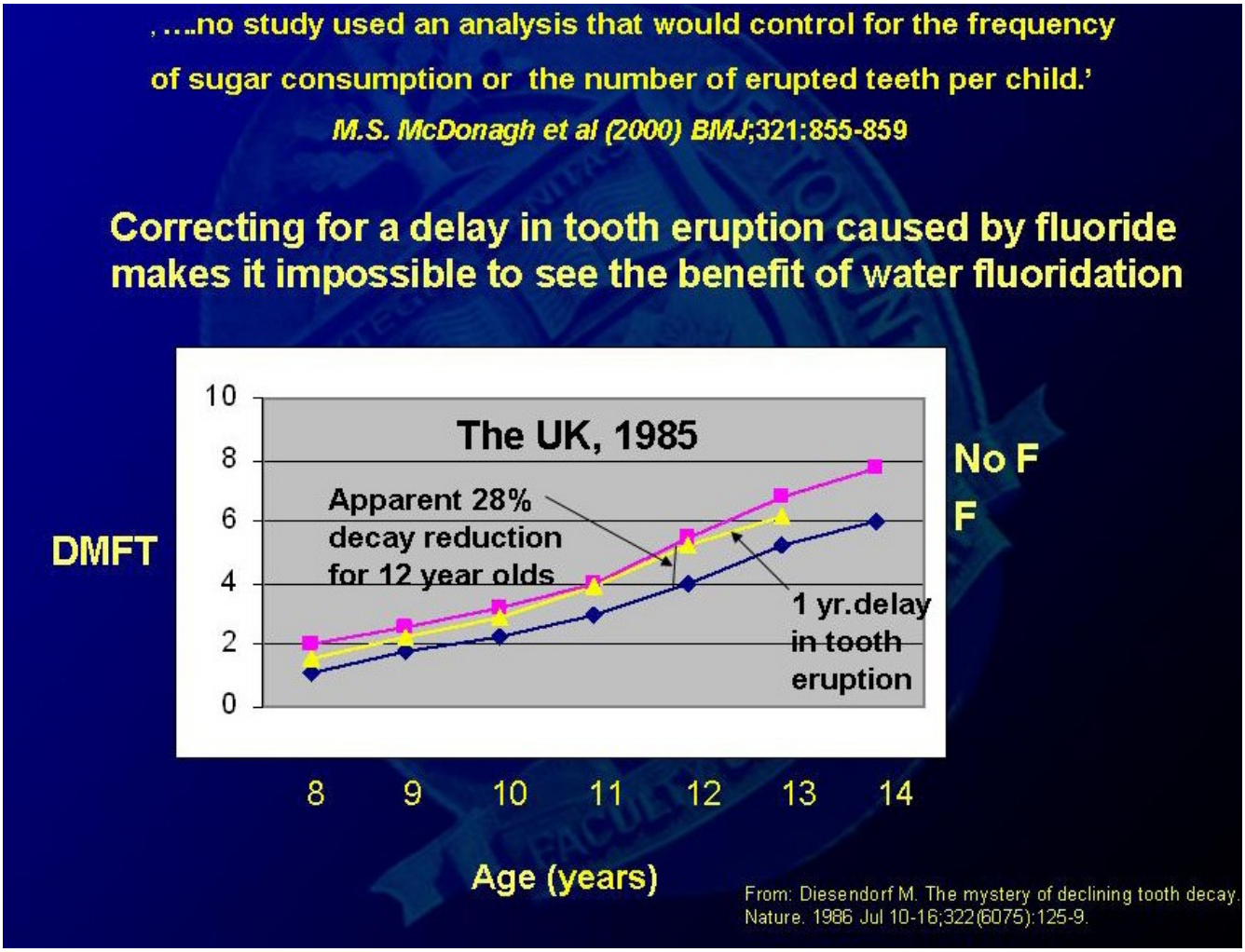
4468 Flemish children. Survival analyses with log-logistic distribution were performed to calculate median emergence ages and 95% confidence intervals; four fluoride exposure parameters

e) fluorosis,
f) use of systemic fluoride supplements,
g) age at which tooth brushing started
h) frequency of tooth brushing

and caries experience were taken as covariates in the model.
The present study indicates that the impact of any of the four fluoride exposure parameters on permanent tooth emergence was relatively minimal. Caries experience in the primary molars had a more pronounced impact on the timing of emergence of the successors than exposure to any of the four fluoride parameters.

Limeback, H. (2002). Systemic Fluoride: Delayed Tooth Eruption and DMFT vs Age Profiles. abstract presented at IADR/AADR/CADR 80th General Session. San Diego, California. March 6-9. University of Toronto, Canada

Recent concerns about the risks vs benefits of water fluoridation have lead to renewed examination of its effectiveness as a public health measure. Furthermore, chronic ingestion of fluoride may have long-term systemic side effects. Several researchers have reported a delay in tooth eruption in children growing up in fluoridated areas. This delay has been reported to be anywhere from 0.7 years (Virtanen et al, 1994) to 2 years (Campagna et al, 1995).
In the recent meta-analysis by McDonagh et al, it was noted that 'no (fluoridation) study used an analysis that would control for…the number of erupted teeth".
Objectives: An attempt was made to determine how much influence various delays in tooth eruption would have on the DMFT vs Age profiles in fluoridated (F) vs non-fluoridated (non-F) areas.
Methods: Modern (1980's) data from the NIDR study (Brunelle and Carlos, 1990), from the UK (Diesendorf, 1986) and older (1950's) data from the original Grand Rapids trial (Arnold et al, 1962) was used for this analysis. The data from the F areas was adjusted by 1 - 2 years. The adjusted DMFT vs Age data was then re-plotted and the profiles compared.
Results: In all cases, a delay in tooth eruption reduced the difference between F and non-F communities. The profiles were indistinguishable when a 1-year delay was used for the modern data and a 2-year delay was used for the older data.
CONCLUSION: The small benefit that remains today from water fluoridation can, in part, be explained by fluoride ingestion retarding tooth eruption, resulting in a delay in dental caries. The effect of the delay in tooth eruption from systemic fluoride is likely less evident in more recent fluoridation studies because of increasing ingestion of other sources of fluoride such as fluoridated dentifrices.




**Legend:**
blue line = DMFT with fluoridated water
yellow line = DMFT with 1 year adjustment for delayed tooth eruption
pink line = DMFT with low fluoride water

Masaki T. Geographical distribution of 'mottled teeth' in Japan. Shikwa Gakuho 1931;36:875.

First molars appear usually at age 5, in fluoridated areas at age 8

Masden, Hodge H. 1946 Investigation of the Nature of Fluoride in Blood

6. There is considerable uncertainty as the base level of blood fluorides
7. An antagonism appears to exist between fluorine compounds and thyroxine
8. Organic fluorine compounds appear to be more toxic than the fluoride ion
9. Fluorine apparently exists in the blood in an organic and an inorganic state somewhat analogous to blood iodine

10. In hemophilia the blood fluorine often rises to very high levels
11. 3-fluorotyrosine has given excellent results in the treatment of Basedow's disease (toxic hyperthyroidism)

These observations have suggested the following broad outline for a research program on blood fluorine.

PROBLEM: Preliminary experiments in this laboratory have tended to confirm the observation that blood fluorides exist in both an "organic" and "inorganic" form. The purpose of this problem is to investigate the nature of the compounds of fluorine existing in the blood, devoting special attention to the so-called "organic" fractiton.

METHOD OF ATTACK: Reference to the literature indicates that the normal range of F. in the blood is rather wide; however, the methods used have always included operations which in our experience have resulted in loss of fluorine. Accordingly, it will first be necessary to determine the range of normal values as measured by our technique. The approach to the main problem might well proceed according to the following lines:

1. Determine the fluorine content for whole blood, cells and plasma
2. Prepare hemoglobin, protein and lipid fractions and determine the fluorine content of each
3. Investigate the complexing of fluorine with hemoglobin and other proteins
4. Investigate the relation between fluorine and non-diffusable (protein bound) blood calcium

In addition to the main problem of elucidating the nature of blood fluorides, the following secondary problems are of considerable interest:

a) an investigation of the possible relations between fluorides, iodide and calcium levels and the thyroid gland.
b) The effect of fluorine upon enzyme systems of the blood, particularly by means of an in vivo experiment
c) The solubililty of fluoride salts and complexes in plasma
d) How high can the blood fluoride level be raised before ill effects are observed in animals.

Significance of the Problem: These experiments are intended to give fundamental information regarding the mode of action and metabolism of fluorine in the system. This information would appear to be of value for the following reasons:

5. Fluorides are used in the treatment of disease. Blood levels appear to show significant variations in certain pathological conditions
6. Exposoure to fluorides is of industrial significance, particularly since the advent of atomic energy programs
7. Exposure to fluoride may occur through consumption of foods heavily contaminated with fluoride containing fertilizers
8. The determination of base levels is of immediate practical value in the impending litigation between DuPont Co. and residents of New Jersey areas.

Nadler GL. Earlier dental maturation: fact or fiction? Angle Orthod 1998; 68(6):535-8.
EGNadler@aol.com

The objective of this investigation was to determine if there is a difference in dental age of maturation between adolescents treated in the 1970s and those treated in the 1990s. Records of

150 Caucasian patients, 8.5 to 14.5 years old and treated in a private orthodontic office between 1972 and 1974, were randomly selected; records of another 150 patients of the same race and age range but treated between 1992 and 1994 were also collected. The percentage of calcification of the mandibular canines was rated according to methods used by Demirjian, who divided tooth development into eight segments, A to H. Using stage G to compare the 1970 and 1990 patient samples, we demonstrated dental age reductions of 1.21 years for males and 1.52 years for females, and a combined reduction of 1.40 years.

Paluev KK. Fluoridation causes a delay in onset or recognition of caries. Australian J Dent 1955;51:13 (1955)

Peiris TS, Roberts GJ, Prabhu N. Dental Age Assessment: a comparison of 4- to 24-year-olds in the United Kingdom and an Australian population. International Journal of Paediatric Dentistry. 2009 Sept;19(5):367-376(10).

Department of Paediatric Dentistry, Westmead Centre for Oral Health, Sydney, NSW 2145 Australia.

BACKGROUND: The physiological age of a person is determined by the degree of maturation of the different tissue systems. Children of the same chronological age (CA) can demonstrate different degrees of maturation. Dental age (DA) is based on the maturation of teeth. Tooth formation is a continuous process, where the developmental stages of the tooth can be sequenced and defined depending on the degree of mineralization. These stages can be visualized on a dental panoramic tomograph (DPT).

AIM: The aim of this study was to use a new method of Dental Age Assessment (DAA) to compare a United Kingdom (UK) and an Australian (AUS) population.

DESIGN: The DPTs used are from the archives of the Westmead Centre for Oral Health (Westmead, Australia) and the King's College London Dental Institute. From the preliminary sample of 89 DPTs from each population, 77 were suitable for use as matched pairs. The radiographic technique used was developed by Demirjian and describes eight stages of tooth development. This was used in combination with numerical data derived from a meta-analysis of a single UK subject.

RESULTS: A significant difference was shown between the CA and DA of the AUS patients. The AUS patients were also shown to have a **significant 0.82 years delay in their DA compared to the UK patients.** The findings indicate a difference in AUS compared to UK patients. These results indicate the need to develop a reference data set for the AUS population for DAA.

CONCLUSIONS: This research is of significance in a number of clinical disciplines and can also be used to assist in age determination of subjects of unknown birth date to assist in forensic dentistry or social deliberations.

Russo MC de, Andrioni JN, Benfatti SV, Adas N, Marchi F. The effect of fluoride on the chronology of

eruption of permanent teeth. Rev Fac Odontol Aracatuba 2(2):197-207.
Rwenyonyi CM, Birkeland JM, Haugejorden O, Bjorvatn K. Dental variables associated with differences in severity of fluorosis within the permanent dentition. Clin Oral Invest 2000;4(1):57-63.

Short EM. Domestic water and dental caries. VI. The relation of fluoride domestic waters to permanent tooth eruption. J Dent Res 1944;23:247-55.

Smiech-Slomkowska G, Cichocka D. Dental age of children with mottled enamel living in an area with excess fluorides in the drinking water. Czas Stomatol 1984;37(4):259-62.

Smith MC, Lantz EN, Smith HV. Further studies in mottled enamel. JADA 1935;May, 817-29.

Souza Freitas JA de, Lopes ES, Alvares LC, Tavano O. Influence of fluoridation on the chronology of eruption of permanent teeth. Estomatol Cult 1971;5(2):156-65.

Szelag J. Evaluation of the effects of various fluoride concentrations in drinking water and atmospheric air on permanent teeth eruption in children aged 12 years. Czas Stomatol 1990;43(3):154-9.

> Dental age was evaluated in 382 children aged 12 +/- 6 years in four Silesian localities differing in the content of fluorine in drinking water and air: in Milicz--without fluoride, in Wroclaw where artificial Fluoridation of drinking water is conducted to levels regarded as optimal--from 0.8 to 1.2 mg/l, in Gryfow where the mean annual pollution of atmospheric air with fluorides was from 0.044 to 0.059 mg/m3 in Nysa where drinking water contains an excess of fluorides 4.0 to 7.0 mg/l. **In the light of these studies fluoride was found to retard teeth age. The greater dose of fluorine is taken up by the organism in the period of development the longer is the retardation of dental age.**

Tseng CC, Chen RS, Guo MK, Hsieh CC, Hong YC. Influence of water fluoridation on the eruption of permanent teeth in Chung-Hsing New Village, Taiwan. Taiwan I Hsueh Hui Tsa Chih 1989;88(3):272-7.

> Along with an evaluation of the effects of 12 years' water fluoridation in the prevention of caries, the present study was conducted to explore the influence of water fluoridation on the eruption of permanent teeth. The survey was carried out in fluoridated Chung-Hsing New Village and the control town of Tsao-Tun from October to December, 1984. Dental examinations were performed on 3,459 children Chung-Hsing New Village and 4,610 in Tsao-Tun, at ages ranging from 3 to 15 years. The results showed that in general, the mean tooth eruption time of girls was earlier than that of boys, and that teeth in the lower jaw emerge sooner than their homologues in the upper jaw, except for premolars. In both fluoridated and control areas the order of tooth eruption was very similar except for the teeth of girls in the upper jaw. The eruption sequence was first premolar, canine then second premolar in the girls of Chung-Hsing New Village, while first premolar, second premolar then canine in those of Tsao-Tun. By comparing the eruption time of permanent teeth in the children of fluoridated and control areas, it was found that they were very alike in general, the only difference being observed in the premolars of the sexes. The **premolars of the children in the fluoridated area**

**emerged in the oral cavity 2.5 to 4.5 months later than did their counterparts in the control area**
***(More girls)***

Valery LP. Fluorine: effect on the loss of deciduous teeth and on the eruption of permanent teeth. Chir Dent Fr 1976;46(300):77.

van Palenstein, Helderman WH, Mabelya L, van't Hof MA, Konig KG. Two types of intraoral distribution of fluorotic enamel. Community Dent Oral Epidemiol 1997;25(3):251-5.

WHO Collaborating Centre for Oral Health Care Planning and Future Scenarios, Faculty of Dentistry, University of Nijmegen, The Netherlands.

> "The similarity of the curves suggests that the later in life enamel is completed, the higher is the severity of dental fluorosis"

Venkata Ranga Rao Kodali, Krishnamachari KAVR, Gowrinathsastry J. Eruption Of Deciduous Teeth:Influence Of Undernutrition and Environmental Fluoride. Fluoride 1994;27(4)236-237 also: Ecology of Food and Nutrition 1993;30:2.

Virtanen JI, et al. Timing of eruption of permanent teeth: standard Finnish patient documents. Community Dentistry and Oral Epidemiology 1994;22(5 Pt 1):286-8.

> Department of Preventive Dentistry and Cariology, University of Oulu, Finland.
>
> Current standards for the eruption of teeth are constructed mostly on the basis of cross-sectional data. The aim here was to analyze the suitability of the standard patient documents created for health center dental care purposes for the collection of longitudinal data on tooth emergence. Copies of the oral health records of the 911 children born in 1970 and 1971 and in 1980 and 1981 living in three rural communities in Finland were re-examined and analyzed using a specially compiled computer program. The means and standard deviations are in line with previous results. The sex difference in emergence timing ranged from 0.1 to 1.0 years for the various teeth. The emergence of the teeth of the second phase of the mixed dentition was later in the children living in an endemic fluoride area, this difference being statistically greater for the boys than for the girls (95% Cl for differences between means was used to evaluate statistical significance). A secular trend in the eruption of permanent teeth was found between 1970 and 1980. Patient documents are shown to be suitable for the collection of longitudinal data on dental emergence.

Wodniecki J, Knychalska-Karwan Z, Dyras M, Gawrzewska B, Michalik B, Szafraniec I. Dynamics of tooth eruption under the influence of air polluted by industrial gases and dust containing fluorine compounds. Folia Med Cracov 1975;17(2):235-43.

Weaver, R. The Inhibition of Dental Caries by Fluorine. Proc Roy Soc Med 1948;(41) 284-90.

Weaver R. Fluorine and dental caries: further investigations on Tyneside and in Sunderland. Br Dent J 1944;(77):185-93.

Whelton H. Overview of the impact of changing global patterns of dental caries experience on caries clinical trials. J Dent Res 2004;83 Spec No C:C29-34

Ziskin D,Applebaum E, Salmon TN. The effect of thyro-parathyroidectomy at birth and at 7 days on dental and skeletal development of rats. J Dent Res 1940;19:93.

Ziegelbecker R. A critical review on the fluorine caries problem. Fluoride 1970;3:71-9.

**Books: 4**

Orban B. Oral Histology and Embryology. St. Louis, p.304 (1949)

Schatz, A -"Low-level fluoridation and low-level radiation --Two case histories of mis-conduct in science" by Albert Schatz, Ph.D,1996, Philadelphia, Pennsylvania
http://www.cadvision.com/fluoride/schatz.htm

Sutton P -"The Greatest Fraud Fluoridation" ISBN 0949491128 (1996)
http://www.fluoridation.com/sutton.htm

Waldbott, G. L., Burgstahler, A. W., and McKinney, H. L. Fluoridation: The Great Dilemma. Lawrence, Kansas: Coronado Press, 1978.

**In Animals: - 7 studies**

Berkovitz BK - "Effect of fluoride on eruption rates of rat incisors" Dent Res 53(2):334-7 (1974)

George B, Kobylanska M - "Effect of fluoridated drinking water on rat's teeth. I. Its effect on erupted teeth" Czas Stomatol 27(6):581-7 (1974)

Kick CH, Bethke RM, Edgington BH, Wilder OHM, Record PR, Wilder W, Hill TJ, Chase SW - "Fluorine in Animal Nutrition" Bulletin 558, US Agricultural Experiment Station, Wooster, Ohio (1935)

Krook L, Maylin GA, Lillie JH, Wallace RS - "Dental fluorosis in cattle" Cornell-Vet 73(4):340-362 (1983)

describe a mechanism that produces marked delay in the eruption of teeth (1.5 to 3.0 years) in **cattle** crippled with fluorosis (fluoride damage to bone), due to **atmospheric fluoride pollution.**

Five expressions of dental fluorosis are described in cattle exposed to industrial fluoride pollution:
1. Hypercementosis with tooth ankylosis, cementum necrosis and cyst formation;
2. Delayed eruption of permanent incisor teeth;

3 Necrosis of alveolar bone with recession of bone and gingiva;
4. Oblique eruption of permanent teeth, hypoplasia of teeth with diastemata; and
5. Rapid progression of dental lesions.

The five entities are not recognized in the "standard for the classification of dental fluorosis" by the **National Academy of Sciences**. Since this classification is **too limited and superficial**, adherence to this standard has left severe cases of fluoride intoxication in cattle undetected in field surveys._

Rozeik, F - "Über den Einfluss toxischer Fluordosen auf Zahnhartsubstanzen und Knochen"; in :Gordonoff, T, ed. - Fluor und die Schilddrüse, Toxikology des Fluors, Basel/Stuttgart, pp.99-105 (1964)

Skobe Z, Heeley JD, Dobeck JM, Prostak KS, Maravelis L, Stern DN - "Comparison of rates of enamel synthesis in impeded and unimpeded rat incisors" Dent Res 72(1):46-50 (1993). Forsyth Dental Center, Boston, Massachusetts 02115.

Periodic intubations of rats with solutions of fluoride (F) lead to the appearance of bands of disru**pted pigmentation in continuously erupting incisors**. Distances between fluorotic bands reflect time intervals between intubations. In this experiment, the periodicity of fluorotic banding was used for estimation of the rate of enamel synthesis in impeded and unimpeded rat incisors. Rats kept on a low-F diet and distilled water were intubated two or four times per week with 2 mg NaF/150 g body weight. In a group of rats, one of the mandibular incisors was cut at the gingival margin after two weeks, and intubations were continued for an additional two weeks. In another group of F-intubated rats, incisors were cut or notched at the gingival margin twice, six days apart. Control rats either received the same periodic F intubations or were maintained on the low-F diet without intubation. Measurements of spacing between fluorotic bands were identical in impeded and unimpeded teeth, even though the latter erupted at a faster rate. In an unimpeded mandibular incisors, there was a **significant elongation of the secretory zone and a shortening of the pigmentation zone, resulting in reduced pigmentation intensity of the erupted portions of the teeth.** The results show that the **rate of enamel** synthesis is independent of the eruption rate.

Smith MC - "Effects of fluorine upon rate of eruption of rat incissors" J Dent Res 14:139-144 (1934)

## No Delay in Eruption Studies (2 papers)

Blayney JR, Hill IN. 1967 Fluorine and Dental Caries. Journal of the American Dental Association 74(2): 233-302.

Stephen KW, Macpherson LM, Gilmour WH, Stuart RA, Merrett MC. A blind caries and fluorosis prevalence study of school-children in naturally fluoridated and nonfluoridated townships of

Morayshire, Scotland. Community Dent Oral Epidemiol. 2002 Feb;30(1):70-9. Erratum in: Community Dent Oral Epidemiol 2002 Oct;30(5):397.

University of Glasgow Dental School, Department of Statistics, University of Glasgow, Morayshire, Scotland. k.stephen@dental.gla.ac.uk

OBJECTIVES: To undertake a blind caries and fluorosis prevalence study of Grade 1 (aged 5/6 yr) and Grade 4-7 (aged 8-12 yr) children from naturally water-fluoridated (1 ppm, since 1985) Burghead, Findhorn & Kinloss (F), and nearby nonfluoridated Buckie & Portessie (N-F), in rural Morayshire, Scotland.

METHODS: A blind clinical (+ 10% repeats) caries study of the above townships' 5/6-yr-old lifetime (15 F; 43 N-F), and 8-12-yr-old lifetime (55 F; 136 N-F)/school-lifetime (31 F; 37 N-F) residents was undertaken following bussing of these children to a common examination site in close-by Elgin Town Hall. Initially, each child was asked about their own perception of the aesthetics of their maxillary front teeth. Fluorosis was assessed clinically using the TF Index, as well as photographically - for later blind scoring (+ 10% repeats for lifetime 8-12-yr-olds) of slides by four dental and two lay 'jurors', alongside a now-established UK 'bench-mark' mildly mottled (TFI = 2), fluorosis comparator slide, judged in previous studies to be aesthetically lay-acceptable. In addition, by parental questionnaire, information was sought concerning their child's fluoride supplement and dentifrice usage histories.

RESULTS: For 5/6-yr-olds, mean primary caries scores were 96.0% less in fluoridated than nonfluoridated subjects ($P < 0.01$). In 8-12-yr-olds, DMFT values favoured water-fluoridated subjects; their caries-free trend was significant ($P < 0.001$ overall). Clinically, 33% of all lifetime F subjects and 18% of all N-F pupils had fluorosed maxillary anterior teeth ($P = 0.045$), but no statistically significant difference was found between the 7% F and 3% N-F subjects with TFI scores > 2 ($P = 0.25$). Photographically, 'jury' mottling assessment (+ 10% repeats) of projected slides resulted in at least 1 : 6 positive scores in 43.6% of F and 30.9% of N-F pupils, albeit they unanimously scored only nine F and five N-F children as having fluorosed teeth ($P < 0.01$). In no case did all members score TFI > 2. Dental and lay scorers rated TFI = (1/2) in only a further 9.1% and 5.5% of F subjects, respectively, compared to 0.7% and 1.5% respectively of N-F pupils. Again, TFI > 2 was scored unanimously in no child. No differences were found regarding the children's own degree of anterior tooth aesthetic nonacceptability between F (11%) and N-F (12%) prevalence ($P = 0.75$). Finally, only one F child had taken F supplements and, while 26 N-F had used F drops, no significant relationship was found between their usage and TFI values in the latter group ($P = 0.49$). Additionally, no relationship was noted between clinical TFI scores and the age at which parents stated fluoridated dentifrice toothbrushing commenced, between 0 and 24 + months of age.

CONCLUSIONS: Considerable caries benefit has accrued to those Morayshire rural children who have received naturally fluoridated water (at 1 ppm) throughout their lives, as compared to their socioeconomically similar, nonfluoridated rural counterparts. Furthermore, in spite of all but two subjects claiming to have brushed regularly with fluoridated dentifrice (and no evidence of the availability of nonfluoridated toothpaste being purchasable in the five townships), only borderline mild fluorosis disadvantages have been noted clinically, and none by the subjects'

own aesthetic perceptions. Finally, **no evidence was found to suggest any delay in permanent tooth eruption patterns of the F subjects**. It would seem appropriate therefore, that adjustment of Scots' drinking waters' natural fluoride levels to 1 ppm should be pursued to extend similar dental advantages to the vast majority of that population (both young and old) which, it is well documented, has the worst dental health of mainland UK.