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Effects of Fluoride Availability on Fluoride Content of Deciduous Teeth of Children and Bone Measurements of the Mothers in Some Utah Communities

Monika Margareta Lowgren
Utah State University

EFFECTS OF FLUORIDE AVAILABILITY ON FLUORIDE CONTENT OF DECIDUOUS TEETH OF CHILDREN AND BONE MEASUREMENTS OF THE MOTHERS IN SOME UTAH COMMUNITIES

by

Monika Margareta Löwgren

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ABSTRACT

Effects of Fluoride Availability on Fluoride Content of

Deciduous Teeth of Children and Bone Measurements

Of the Mothers in Some Utah Communities

by

Monika Margareta Löwgren, Master of Science

Utah State University, 1976

Major Professor: Dr. Arthur Mahoney
Department: Nutrition and Food Sciences

The relationship between the fluoride content of the enamel and dentine of deciduous teeth and the number of tooth lesions/mouth was compared to fluoride treatment in Logan, Brigham City, Helper and Milford, Utah. Bone measurement data was obtained on the second metacarpal bones of the mothers of the participating children from the same communities except Brigham City.

The fluoride treatment in the communities were as follows: Logan has no fluoride added to the domestic watersupply but some children received fluoride treatment through tablets, drops, etc. Milford has water which naturally contains 0.8 ppm fluoride, Helper and Brigham City have 1 ppm fluoride added to their water supplies.

The results showed that significantly less ($P > 0.05$) fluoride existed in the dentine of teeth originating from Logan children without fluoride treatment than the dentine from fluoride treated children in Logan and Brigham City. The fluoride content of the enamel showed a tendency to be increased with increasing amount of fluoride exposure.

The fluoride content of the enamel and dentine from nonfluoride treated Logan children was considerably less than from fluoride treated Logan children and from Brigham City children.

Logan children who had received fluoride treatments had significantly ($P > 0.05$) fewer dental lesions/mouth compared to the nontreated Logan children. The fluoride exposed Milford and Helper children had a greater frequency of dental lesions/mouth than even nonfluoride treated Logan children. This might be explained by the socioeconomic differences existing between Logan in comparison to Helper and Milford.

Midshaft bone measurements were taken on x-rays of the second metacarpal of women (average age 35.7) residing ten or more years in the respective community. When measuring cortical thickness, cortical area, medullary area, and percent cortical area, no significant differences could be shown.

(103 pages)

INTRODUCTION

Fluoride (F) is the seventh in the order of abundance of elements in the earth's crust. Since its abundance is of this magnitude, it is not surprising that important amounts of fluoride are found in sea water, in numerous supplies of drinking water, in mineral deposits of fluorspar, cryolite and fluorapatite, and in surface dusts found close to a few of the mineral deposits. The principal sources of supply of fluoride available to man are (1) water, (2) some species of vegetation, (3) certain edible marine animals, (4) dusts in certain parts of the world, and (5) certain industrial processes (Largent, 1970).

Fluoride is the most exclusive bone seeking element existing, owing to its great affinity for calcium phosphate. It is, therefore, accumulated in every tissue showing calcification, be it physiological or pathological (Ericsson, 1970).

The ability of fluoride in very low doses to reduce the number of dental caries is well over 50 percent. This is a finding which should be elaborated and utilized as extensively as possible in a situation where dental caries is by far the most wide-spread of all diseases. It is, moreover, a problem that is increasing constantly, especially in the developing countries.

In the bones, fluoride increases the size of the apatite crystals and reduces their solubility. Large doses of NaF have been tried as a therapeutic agent in osteoporosis, sometimes but not always with success (Ericsson, 1970).

Fluoride is a natural constituent of water and is considered to be an essential nutrient through the studies of Messer et al. (1972) and Schwarz and Milne (1972). Mice maintained on a low fluoride diet over two generations showed a progressive decline in litter production. Addition of fluoride restored their reproductive capacity. It was also found that fluoride is essential for optimal growth and improved pigmentation of teeth in rats which were kept in trace element controlled isolators.

The aim of this study was to investigate how fluoride in different Utah communities affected the fluoride content in deciduous enamel and dentine as well as the caries frequency.

Milford is a community where the water has a natural fluoride content of 0.8 ppm. Helper and Brigham City have fluoridated their water supplies to 1.00 ppm. The water in Logan does not contain fluoride, but some children have received fluoride treatment through tablets, drops, etc. throughout their lives.

The effect of fluoride on the skeleton was also studied, of women residing in the same areas.

TEETH

Water Fluoridation and Caries--Frequency

The idea of utilizing fluoride in the control of dental caries originated from the observations that mottled teeth were conspicuously resistant to caries. Dean (1938) noted that among nine-year-old children from areas with 0.6 to 1.5 ppm fluoride in the domestic water supply, 4 to 5 percent were caries free, while 22 to 27 percent were caries free from an area with 1.7 to 2.5 ppm fluoride.

Based on clinical examinations in 21 cities, Dean and Arnold (1942) stated:

> Strikingly low dental caries prevalence was found associated with the continuous use of domestic waters whose fluoride content was as low as about 1 ppm, a concentration which under the conditions prevailing in the localities studied, produced only sporadic instances of the mildest forms of dental fluorosis of no practical esthetical significance. (Dean and Arnold, 1942, p. 26-28)

The above results led to the first artificial fluoridation of domestic water supply, to 1 ppm fluoride, on January 25, 1945, in Grand Rapids, Michigan (Arnold et al., 1956, 1962).

During the years, numerous articles have been published concerning the caries inhibiting effects of fluoride. Table 1 is a summary of some publications. Among three- to eight-year-old children, who all their lives have been exposed to fluoridated water, a 34 to 91 percent decrease in def (decayed, extracted, or exfoliated filled deciduous teeth) have been reported (Table 1). The greatest reduction (88 percent) was on smooth surfaces. The proximal surfaces showed 74 percent, and

Table 1. Reductions in decayed, missing, filled permanent teeth after fluoridation of domestic water supplies

Community	Years of fluoridation	Age of children	Percent reduction	Reference
District of Columbia	10	6	59	Dunning, 1965
Evanston, Ill.	10	6-7-8	91-65-63	"
Fort Wayne, Ind.	10	6-10	50	"
Hopkinsville, Ky.	10	children	56	"
Louisville, Ky.	10	6-8	62	"
Hagerstown, Md.	10	7-13	57	"
Grand Rapids, Mich.	10	6-7-8	75-63-57	"
Grand Rapids, Mich.	10	9-10	50-52	"
Grand Rapids, Mich.	10	11	54	Arnold et al., 1956
Newburgh, N. Y.	10	6-9	58	Dunning, 1965
Newburgh, N. Y.	10	10-12	57	Ast et al., 1956
Newburgh, N. Y.	10	13-14	48	Dunning, 1965
Newburgh, N. Y.	10	16	41	"
Charlotte, N. C.	10	6-11	60	"
Chattanooga, Tenn.	10	6-14	71	"
Marshall, Tex.	10	7-15	54	"
Brantford, Ont.	10	6-7-8	60-67-54	"
Brantford, Ont.	10	9-10	46-41	"
Brantford, Ont.	10	11-13	44	"
Brantford, Ont.	10	14-16	35	"
Hastings, New Zealand	10	10	55	Murray, 1970
Grand Rapids, Mich	15	12-14	50-63	Arnold et al., 1962
Grand Rapids, Mich.	15	15-16	48-50	"
Grand Rapids, Mich.	6 1/2	6	67	Arnold et al., 1953
Newburgh, N. Y.	15	13-14	70	"
Danvers, Mass.	8	7	86	"
Danvers, Mass	8	10	49	"
Athol, Mass.	8	6	75	"
Athol, Mass	14	11	53	"
Evanston, Ill.	14	14	49	Blayney and Hill 1967
Evanston, Ill	12	8	35	"
Kilmarnock, U. K.	5	3	66	Murray, 1970
Anglesey, U. K.	5	4	57	"
Watford, U. K.	5	5	50	"
Anglesey, U. K.	11	8	43	"
Watford, U. K.	11	9	36	"

Table 1. Continued

Community	Years of fluoridation	Age of children	Percent reduction	Reference
Tiel, Holland	13 1/2	13	58	"
Brantford, Ont.	17	16-17	55	"
Indianapolis, Ind.	13	5	46[α]	Katz and Muhler, 1968
Indianapolis, Ind.	13	6	34[α]	"
Indianapolis, Ind.	13	5	55[β]	"
Indianapolis, Ind.	13	6	69[β]	"
Muncie, Ind.	7	5	39[α]	"
Muncie, Ind.	7	6	33[α]	"
Muncie, Ind.	7	5	35[β]	"
Muncie, Ind.	7	6	33[β]	

[α]deft
[β]defs

pits and fissures showed a 43 percent caries reduction (Murray, 1970). Among 16- to 17-year-old children, 41 to 64 percent reduction in DMF (decayed, missing, filled permanent teeth) is noted (Table 1). Rampant caries (incidence of caries attack is much higher than that which normally occurs--when one deciduous incisor possesses labial caries) in an area with 1.5 to 2.0 ppm fluoride in domestic water supply among five-year-old children was reduced by 60 percent compared with a low fluoride area (0.2 ppm fluoride)(Jackson, Murray, and Fairpo, 1974).

Topical Fluoridation

The slow progress towards fluoridation of domestic water supplies turns many to other sources of fluoride. Fluoridated mouth rinses, school water fluoridation, tablets, gels and dentifrices are used.

It is postulated that the effectiveness of a topical agent is proportional to its ability to deposit fluoride as fluoroapatite in the enamel (Brudevold et al., 1967). All studies referred to, in this section, are summarized in Table 2.

School water fluoridation

Children consume only a part of their daily intake of water at school, hence higher levels than are optimal for municipal water supplies were used for schoolwater fluoridation in areas where the natural level of fluoride was deficient. A 30 percent decrease in DMFS (decayed, missing, filled permanent surface) was noted among six- to ten-year-old children who had been exposed to 6.3 ppm fluoride in schoolwater supplies. The same conditions but 4.1 ppm fluoride in the school water showed a decrease in DMFS with 22 percent (Heifetz and Horowitz, 1974).

Table 2. Topical fluoride treatments and their effect on caries reduction

Type	Amt. of fluoride concen.	DMF red.	Time treated	Age	Comment	Reference
				yrs		
Schoolwater	6.3 ppm	30%	4 yr	6-10		Heifetz and Horowitz, 1974
	4.1 ppm	22%	4 yr	6-10		
Mouthrinse	0.05% NaF	36%[a] 32%[b]	34 mo	12	Every schoolday	Rugg-Gunn, Holloway, and Davies, 1973
Tin						
Dentifrice	0.4% SnF_2	23%[a] 25%[b]	1 yr	6-15		Muhler, 1958
Dentifrice	0.4% SnF_2	61%[a]	2 yr	adult	Daily	Scola and Ostom, 1968
Paste	17.5% SnF_2				Annual	
Aqueous sol.	10% SnF_2	77%[b]			Annual	
Mouth rinse	0.1% SnF_2	33-43%[a]	20 mo	8-13	250 ppm, once each schoolday	Radike et al., 1973
Cavity	30.0% SnF_2	66%	24 mo	6-9	Incidence of recurrent caries which had been fluoride treated before restored with amalgam	Alexander, McDonald, and Stookey, 1973
APF-tablet	1 mg	6%[a]	30 mo	5-6	Teeth present at start	Driscoll, Heifetz, and Kortz, 1974
	2 mg	27%[a]	30 mo	5-6	Teeth present at start	"

Table 2. Continued

Type	Amount of fluoride concen.	DMF red.	Time treated	Age	Comment	Reference
				yrs		
APF-tablet	1 mg	37%[a]	30 mo	5-6	Teeth erupting during study	Driscoll, Heifetz, and Korts, 1974
	2 mg	27%[a]	30 mo	5-6	Teeth erupting during study Chewed every schoolday	"
APF tablet	1 mg	20-23%[a]	20 mo	8 1/2	Chewable APF-tablet	dePaola and Lax, 1968
	1 mg	53%[e]	20 mo	8 1/2	Newly erupted teeth	"
F-tablet with and without vitamins	1 mg	deft[f] defs[g]	24 mo	18-39 (mo)	Chew daily	Hennon, Stookey, and Muhler, 1972
		68 66			F + vitamins	
		55 63			F	
Application						
APF	1.23% F	44%	1 yr	8-12	Exposed for 4 min, once	Wellock, Maitland, and Brudevold, 1965
SnF_2	8.0% SnF_2	-9%	1 yr	8-12	Exposed for 4 min, once	"

Table 2. Continued

Type	Amount of fluoride concen.	DMF red.	Time treated	Age	Comment	Reference
APF			3 yr	yr 10-12	Solution appered for 5 min., pH 4.6, 0.1% H_3PO_4, 4% NaF	Vrbic, Kosmelj, and Ravnik, 1974

DFS		DFT		
c	d	c	d	
23	31	16	29	All teeth
29	29	24	26	Teeth erupted initially
8	34	7	33	Teeth erupted during study

[a]DMFS = decayed, missing, filled permanent surface.
[b]DMFT = decayed, missing, filled permanent teeth.
[c]Annually.
[d]Semi-annually.
[e]DFS = decayed, filled permanent surface.
[f]deft = decayed, extracted or exfoliated, filled deciduous teeth.
[g]defs = decayed, extracted or exfoliated, filled deciduous surface.

Mouthrinse

School children who received supervised mouth rinses containing 0.05 percent NaF, from their twelfth year every schoolday during three years, showed a 36 percent reduction in DMFS (Rugg-Gunn, Holloway, and Davies, 1973).

Tin

A dentifrice containing 0.4 percent SnF_2 caused reductions in DMFT (decayed missing, filled permanent teeth) and DMFS of 25 and 23 percent, respectively, after one year of use among six- to fifteen-year-old children (Muhler, 1958). When the dentifrice was combined with an annual application of a 17.5 percent SnF_2 prophylactic paste in 90 percent SnF_2 aqueous solution, a caries reduction of 61 to 77 percent was seen after 2 years among adult Naval personnel (Scola and Ostrom, 1968). A mouth rinse containing 0.1 percent SnF_2 (250 ppm F), used every school day by children, decreased caries frequency by 33 to 43 percent after two years use (Radike et al., 1973).

Recurrent caries among children were significantly reduced by treating cavities with a SnF_2 solution before they were restored with silver amalgam (Alexander, McDonald, and Stookey, 1973).

Acidulated phosphate fluoride solutions--APF

Brudevold and co-workers are critical of the effect of stannous ion. They reported an identifiable staining of teeth among SnF_2 treated children (Wellock, Maitland, and Brudevold, 1965), and a depression of fluoride deposition in intact enamel (Brudevold et al., 1967). Also the uptake of fluoride by intact enamel was much greater from phosphate

solutions than from stannous fluoride solutions, having the same fluoride concentrations and similar pHs. In search for acid fluoride solutions which provided maximal fluoride uptake while causing minimal demineralization, Brudevold et al. (1963) presented the solution of choice--acidulated phosphate fluoride solution. The fluoride acquired by the outer enamel from these topical treatments appeared to be permanently bound and progressively increased with the number of treatments completed (Mellberg et al., 1970).

At pH 3, more than 50 percent of the fluoride occurs as undissociated hydrogen fluoride which is likely to diffuse into intercrystalline spaces and pathways in the enamel with greater ease than the charged species, F^- or HF_2^-. The fluoride deposited in the intact enamel from APF solutions appears to be present primarily as fluoroapatite (Brudevold et al., 1963). CaF_2, if present at all, is only a minor reaction product even in highly acid solutions.

The APF agent was used semiannually during three years by school children and resulted in a 34 percent reduction in caries in newly erupted teeth (Vrbric, Kosmelj, and Ravnik, 1974). Children who chewed, rinsed, and swallowed an APF tablet once or twice a day in school, showed a decrease, especially in newly erupted teeth, during two to three years of treatment (Driscoll, Heifertz, and Korts, 1974; and de Paola and Lax, 1968).

Vitamins

Administration of vitamins together with fluoride did not enhance caries reduction (Hennon, Stookey, and Muhler, 1972).

Dentifrices

The abrasive agents in dentifrices may abrade the fluoride taken up in the surface enamel and thus lower the caries resistance (Friberger and Lindquist, 1974). When a mild silicon dioxide base was used as the polishing agent and pH lowered from 7.0 to 5.6 in a dentifrice the effectiveness to donor fluoride ion was increased (Friberger, 1974). Gerdin (1974) found that nonabrasive potassium fluoride manganese dentifrice with 250 ppm fluoride and pH 5.5 had a better inhibitory effect on caries than a 1000 ppm fluoride toothpaste at pH 6.5 and containing a nonabrasive agent.

It was not possible to detect any differences in fluoride content in surface enamel between school children who used fluoride containing dentifrice in the morning before examination and those who did not (Aasenden, 1973).

Sodium fluoride is partly inactivated by commonly used polishing agents such as calcium carbonate. The product formed is CaF_2, but when sodium monofluorophosphate is present CaF_2 dissociates and liberates fluoride ions (Norén and Härse, 1974).

Effect on bacterial action

The action of topical treatment on bacterial action has been studied in vitro. A stannous fluoride mouth rinse had a greater effect in reducing microbial populations than an equivalent NaF mouthrinse (Andres, Shaeffer, and Windeler, 1974). Bibby and van Kesteren (1940) showed that NaF-solutions up to 250 ppm was needed to decrease growth rate of mouth organisms. Fluoride concentrations less than 1 ppm limited acid production.

APF gel has a direct antibacterial effect on Streptococci mutans and to a variable extent on S. sanguis (Loesche, Murray and Mellberg, 1973; and Kronche and Kronche, 1974). Weiss, Schnetzer, and King (1964) reported up to 50 percent decrease in polysaccharide synthesis by cariogenic organisms in the presence of 10 ppm fluoride.

Prenatal Effects of Fluoride

The belief that fluoride ingestion during pregnancy will benefit dental health of the child is based on the knowledge that calcification of deciduous teeth is initiated in utero and that fluoride will pass through the placenta to the fetus. In line with these conclusions are the works of Arnold et al. (1953, 1956), where 5-6 year-old children exposed to fluoride both pre- and postnatally had a greater def reduction than similar children exposed postnatally only. The observations were confirmed by Blayney and Hill (1964). However, the work of Katz and Muhler (1968) and Carlos, Gittelsohn, and Haddon (1962) opposed these results. Relative to these findings, Reiss (1961) demonstrated that mineralization of deciduous teeth is not so advanced at birth as is generally believed. But based on samples from stillborn infants, the mineralization rate might have been influenced by whatever fact caused the fetal death. One must also question whether the plasma fluoride values which result in fluorosis can occur before birth. It is established that the placenta acts as an effective barrier to fluoride during the greater part of pregnancy (Zipkin and Babeaux, 1965; and Sforzoline, Savino, and Pascasio, 1972). However, the fluoride content in urine in pregnant women was lower than in nonpregnant women, and

three months after delivery it was normal again (Gedalia, Brzesinski, and Bercovici, 1959). Knouff et al. (1936) found that extremely high ingestion over a long time resulted in increased fluoride deposition in the fetus.

Stookey, Hennon, and Muhler (1969) used rats to conclude that prenatal $NaSn_2F_5$ in a vitamin supplement reduced caries in the offspring more efficiently than NaF in the same supplement.

Adult Dental Benefits from Fluoride

In an area with one of the lowest dentist/population ratios (1/8700) in Britain, the presence of 1.5 to 2.0 ppm fluoride in drinking water resulted in a lower caries experience, a lower tooth mortality, and a smaller need for partial dentures compared with a non fluoride area with one of the most favorable dentist/population ratios (1/3030) in the country. These benefits were observed for all ages up to 65 years (Murray, 1971). Later studies (Murray, 1973) from the same area showed no effect of fluoride on the pattern of gingival recession in adults.

Dietary Fluoride

Almost every known food contains traces of fluoride. Our richest dietary sources of fluoride are sea food and tea. Sea water may contain approximately 1.2 to 1.4 ppm fluoride and is the source of fluoride in sea food. The amount of remaining bone, particularly in canned fish, determines to a major extent the quantity of fluoride contained in the product. Tea is an unusual plant substance in its fluoride content. More than 75 percent of the fluoride in tea is extracted by boiling water.

About 0.1 mg fluoride may be present in hot water extract from one tea ball (dry tea contains 63-186 ppm fluoride)(McClure, 1949; Cook, 1969). Milk has a negligible concentration of fluoride (about 0.1 ppm) like other body fluids, and is virtually unaffected by the extent of fluoride ingestion by the cow (McClure, 1949).

The evidence regarding soil fluoride and its effect on fluoride content of plants shows generally a negative effect; however, in animal nutrition plants may become contaminated by soil dust before they are consumed (McClure, 1949).

It is estimated that diets from an area with low fluoride availability supplies approximately 1 mg fluoride per day, while an area with optimal fluoride content in the water supply provides 2 to 5 mg fluoride per day (Kramer, Wiatrowski, and Osis, 1974). Diets in great part consist of processed, canned foods which may have been prepared in areas where the fluoride content of the water differed from the fluoride content of the locality from which the diets were obtained (Marier and Rose, 1966).

The amount of fluoride in the diet in St. John's, Newfoundland, averages to 2.74 mg fluoride per day, without any fluoride in the water supply. The diet is based on cod, flour enriched with bone meal and a high tea consumption. This area was compared with Toronto, Ontario, where the diet contains low amounts of fluoride. The content of fluoride in deciduous and young permanent dentin was higher in St. John's than Toronto. Conversely, older permanent dentin from people in these areas contained similar amounts of fluoride perhaps because of earlier poor nutrition (Triers, Elliott, and Smith, 1968).

Comparing the Newfoundland data with Stratford, which has 1.3 ppm fluoride in the drinking water, the content of fluoride in dentin and

enamel were much higher than those produced by ingestion of dietary fluoride alone (Elliott and Smith, 1960).

Cook (1969) was concerned with the additional dietary fluoride caused by tea drinking. In a high tea drinking area, children ingest nearly four times more fluoride without water fluoridation than children that do not consume tea. Jenkins (1969), however, consider Cook's (1969) estimations too high by a factor of two.

It is suggested that ascorbic acid may be a significant factor influencing fluoride deposition in teeth. Guinea pig leucocytes containing high levels of ascorbic acid took up more fluoride than leucocytes with low ascorbic acid levels (Boruch et al., 1968).

Hamsters received grapefruit beverage with and without 1.9 ppm fluoride. The erosion in the molars of the animals given the grapefruit beverage supplemented with fluoride was significantly lower than that in the teeth of hamsters given pure grapefruit beverage, and fed the standard or cariogenic diet (Fuks et al., 1973).

Laurentz and Mitchell (1941) found in experiments with rats that dietary calcium protects the body against dietary fluoride.

The content of fluoride in breast milk is well below 0.05 ppm, about the same as the ionized fluoride content of plasma and of saliva, and does not appear to be influenced by the mother's fluoride ingestion (Ericsson and Ribelius, 1971). However, some milk formulas contain 0.5 to 2 mg fluoride per kg powder. In an area with 1 ppm fluoride in the tap water, the gruel will supply over 40 times more fluoride than the same volume of breast milk (Forsman and Ericsson, 1974). No difference in caries rate was found between breast- and formula-fed children

(Ericsson and Ribelius, 1974). Similar results were obtained from a low fluoride area (0.2 ppm fluoride) (Forsman and Ericsson, 1974).

Fish protein concentrate (FPC) contains 150 to 300 ppm fluoride. Fluoride absorption from FPC is similar to NaF, with 88 percent absorption from FPC compared to 94 percent absorption for NaF (Spencer, et al., 1970). Therefore, the distribution of FPC to malnourished children should be restricted to avoid an unesthetic mottling of the teeth (Hadjimarkos, 1974).

Fluoridation of Vehicles other than Water

Many people do not live in areas served by community water supplies and have to rely on well water. The necessity of finding for these areas an adjunct or alternative was therefore apparent. Table salt and milk have been the most common used vehicles (Table 3).

Milk is the food used most universally by infants and children during tooth formation. For 3.5 years children, 6 to 8 years of age initially, drank milk fortified with 1 mg fluoride per half pint. A 70 percent decrease in caries incidence of erupting teeth was observed (Rusoff et al., 1962). Because dietary calcium decreases the utilization of fluoride (Laurentz and Mitchell, 1941), fluoride in milk is absorbed slower due to formation of either CaF_2 or fluoride complexes with organic matter, principally casein (Ericsson, 1958).

Fluoridated salt (250 mg F/kg salt) was delivered to children aged 2 to 6 years. Over a 5-year period there was a reduction of 40 percent in caries in the experimental group and an increase of 12 percent in the control group with a significant difference (Toth, 1973).

Table 3. Caries reductions among children fed fluoridated milk or salt

Vehicle	Years of use	Age	Percent reduction	Reference
Milk				
	3.5	6-9	50	Rusuff et al. 1962
	3.5	6	59	
1 mg F/1/2 pint	3.5	7	41	
	3.5	8	45	
	3.5	9	49	
Salt				
	4	2-6	40	Tóth, 1973
	4	2	100	
	4	3	58	
250 mg F/kg salt	4	4	52	
	4	5	20	
	4	6	11	

Marthaler and Schenardi (1962) could not show a great difference between children using table salt containing 200 mg NaF/kg and children using unfluoridated salt. They concluded that the fluoride effect is lower from fluoridated salt than the one obtained by water fluoridation. Ericsson (1958) indicated that uptake of ^{18}F by the enamel surface was increased in the presence of chloride ions.

Fluoride Content of Teeth

Fluoride administered orally increases the fluoride content of the enamel and the dentin (Bervenmark and Hamberg, 1974). There are three different techniques to determine the content of fluoride in teeth. The oldest is based on the fluoride content in the whole layer of enamel or dentin, and requires the extracted tooth for the determination (Manly and Hodge, 1939). Newer techniques are possible to apply on teeth *in situ* through an etching process on the teeth. An acid is used to extract a sample of the tooth surface (Brudevold, McCann and Grøn, 1968; Athanassouli, Papastrathopoulos, and Hajioannou, 1973; and Charlton, Blainey, and Schamschula, 1974). The sample is analyzed for its fluoride content with a specific fluoride ion electrode (Frant and Ross, 1966). Another technique to use, though *in vitro*, was designed by Rytömaa, Keinonen, and Anttila (1974). A proton beam is used to determine fluoride content in enamel at a depth of 6 μm.

A high dietary fluoride intake leads to an increased incorporation of fluoride in dentine and enamel, compared to a low fluoride diet. However, the fluoride from water supplies is more effectively incorporated than the dietary fluoride (Elliott and Smith, 1960). See Table 4.

Table 4. Fluoride content in enamel and dentine

Enamel ppm	Dentine ppm	Comment	Reference
Deciduous			
47 ± 3	94 ± 5	Low F-diet	Elliott and Smith, 1960
67 ± 4	164 ± 9	High-F diet, 2.74 mg F/d	
128 ± 16	325 ± 28	1.3 ppm F in water	
47 ± 9	112 ± 10	High F-diet, 2.74 mg F/d	Triers, Elliott, and Smith, 1968
50 ± 5	74 ± 3	Low F-diet	
103 ± 29	155 ± 35	0.5 mg F+vit daily during 7 years	Bervenmark and Hamberg, 1974
58 ± 22	79 ± 21	Vit daily during 7 years	
Permanent			
58 ± 7	235 ± 23	High F-diet	Triers, Elliott, and Smith, 1968
62 ± 6	226 ± 26	Low F-diet	

The fluoride content in the enamel decreases with depth, hence biopsy samples of teeth show higher fluoride concentrations than bulk enamel. It is necessary to standardize these biopsy methods, to be able to compare the different biopsy results. It is necessary to define the kind of tooth, the sample site, the sample weight, the depth, the etching solution and the etching time (Aasenden, Allukian, and Brudevold, 1971: and Poulsen and Larsen, 1975).

By using the formula $[F^-] = \frac{K_f}{D^\alpha}$, it is possible to calculate the fluoride concentration at the depth of 1 μm. D is the depth, $[F^-]$ is the the fluoride concentration in ppm F, and α is the constant which is a measure of fluoride concentration decrease with depth (Athanassouli, Papastrathopoulos, and Hadjioannon, 1973).

The amount of fluoride in surface enamel was related not only to the fluoride content of the domestic water supply but also to the volume of plaque on the tooth surface and hydrogen ion activity in the plaque during fermentation of sucrose. The study suggested that plaque may serve as a means of introducing additional fluoride into enamel after tooth eruption, because fluoride can be released into saliva at pH 5. This pH is frequently reached in plaque during fermentation (Charlton, Blainey, and Schamschula, 1974).

With the proton beam technique (Rytömaa, Keinonen, and Anttila, 1974), fluoride concentration at different spots on the teeth can easily be measured. It was found that, in general, the bottom and walls of the fissures tended to contain more fluoride than the cusps. Teeth from a high fluoride area had a higher fluoride content than teeth from a low fluoride area. The variation of fluoride concentration between individual teeth was wide in both groups.

Mottled Teeth

The earliest sign of abnormality due to excess fluoride in the drinking water is enamel opacity or mottling of the teeth (Dunning, 1965). However, there is a type of enamel defect which could easily be confused with fluoride mottling but nevertheless differed from it in some respects (assymetric distribution) (Zimmerman, 1954).

A terminology and a classification was proposed by Dean (1934) to describe enamel mottling. He classified enamel mottling into six grades, namely: questionable, very mild, mild, moderate, moderately severe, and severe.

Occurrence of dental fluorosis is closely related to fluoride supply during the period of mineralization of the teeth and is considered to be due to a blocking of the enamel forming function of the ameloblasts. Caries frequency (DMFS) in the permanent teeth was higher in a 10 ppm area in comparison with a 1 ppm area. For the primary teeth the deft value decreased with increasing water fluoride content and the caries frequently was not related to the degree of fluorosis (Forsman, 1974).

Incidence of enamel mottling among eight to eleven-year-old children was 34 percent in areas with 1.0 to 2.0 ppm fluoride in water and 31 percent in areas with 1.0 to 1.2 ppm fluoride (Grahnén, Lysell, and Myrberg, 1974).

An assessment of mottling in major teeth was carried out on a random sample of 13- to 16-year-olds in Anglesey (0.9 ppm fluoride) and Leeds (0.1 ppm fluoride). The mouth prevalence of mottling was

found to be 39 percent in Anglesey and 52 percent in Leeds. The tooth prevalence of mottling was 9 percent in Anglesey and 12 percent in Leeds, with statistical significance ($p < 0.01$) (Al-Alousi et al., 1975a,b).

Enamel biopsies of children with mottled teeth from an area with up to 4.05 ppm fluoride in the water did not show that the degree of discoloration of the fluoride hypoplasias had connection with the fluoride content of the teeth (Lex, 1974). When Keene et al. (1975) compared the DMFT between 17- to 23-year-old Marine Corps boys, who either came from a low fluoride area (< 1 ppm) or a high fluoride area (1.1 to 5.5 ppm) no statistical difference was shown.

In temperate zones, 1 ppm fluoride in water is the accepted level at which there is a minimal risk for enamel mottling. Around 2.5 ppm fluoride a few cases of discoloration occur, and the number of cases increases with increasing fluoride concentration in the water.

Mottling of the primary dentition is very rare. This is thought to be due to the fact that the primary teeth are to a significant extent mineralized in utero, and that the placenta acts as a barrier to the transfer of fluoride to the fetus (Forsman, 1974. Formula fed children from Uppsala (1.2 ppm fluoride) and Billesholm (5.5 ppm fluoride) showed only an insignificant trend towards increased enamel mottling compared to breast fed children (Ericsson and Ribelius, 1971).

The etiology of mottling is obscure. Mottled enamel areas were surveyed of the teeth, using polarized light and microradiography. It was found that mottled areas are areas of hypocalcification. It would appear, therefore, that in mottled areas the protein matrix is different from that in "normal" enamel and that this difference impedes mineral

salts from being "seeded" into crystals in the normal way. It is quite possible that certain genetic faults could occur in small groups of ameloblasts and that these faults are manifested as mottled areas (Al-Alousi, 1975b).

It has been suggested that mottling of the teeth was due to a high fluoride intake accompanied by malnutrition, but in both Leeds and Anglesey the children were well nourished.

Jackson (1961) suggested that slight traumatic blows to deciduous teeth might be an etiological factor, because there were four times as many damaged maxillary central incisors as there were damaged mandibular lateral incisors. Purely on an anatomical basis it is reasonable to suppose that any trauma to the anterior part of the mouth in young children, that is, up to four years, would be similarly distributed.

FLUORIDE IN TEETH AND BONE

Fluoride, the most electronegative of all elements, is an unique ion in that it continues to deposit in the calcified structures in the body after the other constituents of bone have already reached a steady state. About 96 percent of the fluoride found in the body is deposited in the hard tissue (Ericsson, 1970).

Tooth material from different geographical areas varies in composition. The concentration of fluoride in surface enamel of similarly aged groups of teeth increased nearly linearly with increased levels of fluoride in the water, whereas the increase in the bulk of the enamel was appreciable only when the water fluoride exceeded 3 ppm. The superficial portions of all dental structures acquired increasing amounts of fluoride with age, while the subsurface material remained virtually unchanged. In enamel, certain constituents, including carbonate, sodium, and magnesium increased in concentrations from the surface inward while others, such as copper and strontium, were evenly distributed. Among the trace elements only fluoride and zinc occur in relatively large concentrations. Other elements including aluminum, strontium, lead, manganese, copper, silica, silver, iron and tin are normally present in concentrations below 10 μm/g and therefore less likely to affect the physical properties of dental structures (Brudevold, Steadman, and Smith, 1960). Crown dentin is exposed to fluids on one side, but unlike enamel, the surface is continuously renewed because of oppositional dentin formation (Sognnaes, Shaw, and Bogoroch, 1955). It is realized that water may

occur in many forms, that it may be bound in the crystal lattice and in the organic matrix in addition to filling free spaces. Deakins (1942) has shown that the process of calcification in the enamel involves the displacement of water by minerals, suggesting an inverse relation between the extent of calcification and the concentration of water. When the spaces between crystals approach atomic dimensions, the movement of ions becomes increasingly restricted by charges on the crystal surfaces (Neuman and Neuman, 1958). The rate of calcification which initially is high will therefore gradually slow down.

Chemical analyses of fluoride rich hard tissues have shown that the incorporation of fluoride slightly alters the chemical composition of bone and teeth. The carbonate and citrate contents are lowered and the magnesium level increased. The calcium to phosphorous ratio, however, remains unchanged. At first the citrate decrease was explained by a substitute of citrate by fluoride. The dissimilarity of fluoride and citrate in valency and size, however, made this explanation somewhat tenuous. Fluoride uptake was similarly reduced by raising the pH of the incubation which suggested an analogous competition between fluoride and hydroxide ions (Weidmann and Weatherell, 1970) for sites in the apatite crystal. Stereochemically, fluoride ion is about the same size and shape as hydroxide ions. It can readily exchange isomorphically with only a small decrease in the axis from 9.42 Å for hydroxyapatite to 9.37 Å for fluoroapatite. The reaction can be expressed by the following equation:

$$Ca_5 (PO_4)_3 OH + F^- \rightleftharpoons Ca_5 (PO_4)_3 (OH)_x (F)_{1-x}$$ (Weidmann and Weatherell, 1970).

The only deviation from the concentration that fluoride ions do not replace phosphate groups of hydroxyapatite is during the topical application of concentrated fluoride solutions (1 percent = 10,000 ppm) to enamel surfaces. CaF_2 will precipitate and phosphate is released:

$$Ca_{10}(PO_4)_6(OH)_2 + 20\ NaF \rightarrow 10\ CaF_2 + 6\ Na_3PO_4 + 2\ NaOH$$

Fluoride may enter the bone mineral by its incorporation during the phase of crystal growth or through a surface reaction. More than twice as much fluoride was deposited in actively growing sites than mature sites (Hac and Freeman, 1969).

The incorporation of fluoride by human enamel from aqueous solutions and dentrifrices involves the process of diffusion (Duckworth and Braden, 1967; Myers, Hamilton, and Becks, 1952; Stearns, 1970). If the fluoride chemical potential (or free energy) is higher in the topical agent phase than in the enamel phase, the fluoride must flow in such a way as to reduce the chemical potential gradient. The direction of flow is from the phase of higher chemical potential to the region of lower chemical potential (Shoemaker and Garland, 1967, cited in Stearns, 1970).

The thermodynamic basis for the reaction of hydroxyapatite to fluoroapatite is that the liquid phase is undersaturated with respect to hydroxyapatite and supersaturated with respect to fluoroapatite which leads to the forming of fluoroapatite. Fluoroapatite is formed at the crystals of the enamel in the form of crystal growth or as a substitution for hydroxide ions by fluoride ions into the existing lattice (Larsen, 1974b; Larsen and Thorsen, 1974; Mir and Higuchi, 1969). The above mechanism follows first order kinetics. It is increased as pH decreases. At high hydroxide ion concentration in the solution, there are few

hydroxide positions in the crystal. Intracrystalline exchange is nearly abolished while at low hydroxide ion concentration the fluoride ion easily diffuses into the crystal interior (Larsen, 1974a).

Stearns (1973) indicated that high fluoride concentrations decrease the second order reaction. The effect of 0.9 ppm fluoride in a saliva sucrose solution was investigated based on an ionic strength of 0.05 M and the ion activity coefficients. Fluoroapatite and CaF_2 are the only solid fluoride compounds known to form in aqueous solution like saliva. The fluoride ion activity must be 1.3×10^{-1} M (about three times that of the present activity) to precipitate CaF_2 if the total calcium of 4×10^{-3} M is ionized at pH 5. When the solubility product of fluoroapatite is $10^{-2.2}$ less than that of hydroxyapatite, fluoridated redeposition of mineral in the enamel occurs. At pH 5, a fluoride ion activity of only 10^{-11} is sufficient to favor precipitation of fluoridated apatite rather than hydroxyapatite provided that their ion activities exceed the solubility products of the solids. Based upon ion activities of saliva and a pH level of 5, fluoroapatite may remain undissolved at about half a pH-unit below that required to dissolve hydroxyapatite (Birkeland, 1975). McCann (1968b) showed that fluoroapatite in saliva was essentially insoluble.

Finely pulverized human enamel was reacted with APF solution. The fluoride content of the products and the amount of CaF_2 formed increased with increased reaction time and temperature, or both (Stearns and Berndt, 1973).

The chief mechanism of ^{18}F uptake is a simple heteroionic exchange between radiofluoride in the solution phase and dissimilar ions in the solid phase. However, iso-ionic exchange, a process by which ions from

the solution phase exchange with similar ions in the surface of the solid phase, may occur (Neuman and Neuman, 1953). Therefore, fluoride uptake measured with ^{18}F is not always equal to uptake determined by chemical analysis (Joyston-Bechal, Duckworth, and Braden, 1967). The diffusion of fluoride into enamel is an exothermic chemical process and the conversion of hydroxyapatite to fluoroapatite is therefore an exothermic chemical reaction (Stearns, 1971). The conversion of hydroxyapatite through fluoroapatite to CaF_2 and $Ca_3(PO_4)_2$ involves an endothermic reaction (Stearns, 1973) and the formation of CaF_2 and $Ca_3(PO_4)_2$ directly from hydroxyapatite is an exothermic reaction

$$Ca_5 (PO_4)_3 OH_{(s)} + 2HF_{(aq)} = CaF_{2(s)} + 3Ca_3 (PO_4)_{2(s)} + 2H_2O_{(1)}$$

The uptake of fluoride from APF solutions was too rapid to be a process of diffusion, therefore, Joyston-Bechal, Duckworth, and Braden (1973) suggested it is more likely surface absorption.

The mineral fraction in the enamel is responsible for the electrochemical properties in that it acts as an ion exchange membrane whose selectivity to ionic transport is a function of the electrokinetics of the crystalline surfaces adjacent to the diffusion pathways. The effective fixed negative charge density associated with the surfaces of hydroxyapatite crystals along the diffusion pathways may be increased by a topical fluoride treatment and may induce a higher cationic selectivity to the structure (Waters, 1971, 1972). In the presence of divalent cations such as Ca^{2+}, enamel possesses exchange properties which could absorb anions at the surfaces of the hydroxyapatite crystallites along the diffusion pathways.

It was shown that carious enamel contains more fluoride than does intact enamel (Little, Posen, and Singer, 1962). These findings can be explained as resulting from the low pH prevailing in the plaque and in the carious cavity itself. Following carbohydrate consumption, the low pH enhances the formation of fluoroapatite considerably (Stephan, 1944). Luoma (1975) showed that such caries preventing factors as fluoride and chlorhexidine may in part exert their protective action by rendering or enhancing the phosphate of plaque bacteria to become employed for the repair of enamel, previously demineralized by acid. The hydroxide ions released from the apatite into the enamel-bacteria interphase may elevate the pH considerably localized around the phosphate transporting mechanism of the bacteria. It can be changed from pH 5.9 towards the optimum pH for phosphate uptake which in this particular strain (Streptococcus mutans, FA-1) is near neutrality. Koulourides, Cueto, and Pigman (1961) implied that topically applied fluoride may prevent caries by enhancing mineralization of hypomineralized or precarious enamel. The results of Feagin (1971) showed that fluoride increases the rate of calcium phosphate deposition during remineralization of acid-softened enamel and itself incorporated into the mineral formed.

The mechanism by which the fluoride would exert its action would be by stabilizing the apatite phase under conditions of demineralization and by favoring its formation during remineralization (Grøn, Brudevold, and Aasenden).

Experimental data on the composition of enamel indicate that the incorporation of fluoride will stabilize the enamel crystals by the apparent diminution of Na^{+} and CO_3^{2-} incorporation. The additional effect

may be a change in the type of the main ternary apatite. The enamel apatite crystals change from carbonatoapatite to hydroxyapatite. This is induced by incorporation of a relatively small amount of fluoride ions. It is estimated that these effects in combination lower the solubility product of enamel apatite by 5 to 10 orders of magnitude (Driessens, 1973).

The mechanism of uptake of fluoride by synthetic hydroxyapatite was investigated in pre-equilibrated solution-hydroxyapatite systems containing initial concentration of 3 to 10 ppm fluoride in solution and trace amounts of ^{45}Ca or ^{32}P. Decreasing pH increased the magnitude of both precipitation and recrystallization with high surface area apatites. Adsorption or exchange was the major mechanism for fluoride uptake at neutral pH although recrystallization was also increased by increased surface area (Spinelli, Brudevold, and Moreno, 1971). Fluoride during mineral dissolution may reduce the release of calcium due to their ability to enhance a redeposition of fluoroapatite (Brudevold and McCann, 1968). Koulourides (1968) showed that 1 ppm fluoride (in vitro) increases the remineralization of enamel 4 to 5 times, and that as little as 0.1 ppm fluoride reduces the solubility of powdered enamel in an acetate buffer (Manly and Harrington, 1959).

In a remineralization study on human carious dentine, it was found that the optimal fluoride concentration for a buffered solution is between 5 to 20 ppm fluoride. If the initial fluoride concentration is raised to 100 ppm, a rapid collapse of the remineralization system was described (Levine and Rowles, 1973). Distribution of 250 ppm fluoride in drinking water showed an increased deposition of calcium in growing rat dentine

while at 25 ppm such effects were not evident (Meffert and Kämmerer, 1973).

Magrill (1975) concluded that CaF_2 plays no part in reducing hydroxyapatite solubility, thus, the solubility of hydroxyapatite was reduced by amounts of fluoride ions insufficient to form any fluoro-apatite but merely adequate either to replace a few hydroxide ions or to occupy vacant hydroxide ion sites on crystallite surfaces.

When teeth pretreated with fluoride were exposed to salad dressing, spagetti sauce, sauerkraut, or applesauce to induce decalcification, microscopic examination of the teeth showed that NaF had no effect. SnF_2 exerted a protective action and an APF-gel seemed to enhance enamel demineralization (Liatukas, 1973).

Mice which had received NaF and complex fluoride showed a considerably lower occurrence of dental fluorosis in treatment with complex fluoride (sodium monofluoro phosphate, sodium difluoro phosphate, and ammonium hexafluor aluminate) compared to NaF (Ruzicka, Mrklas, and Rokytova, 1974). Breuer and Cussler (1975) increased the incorporation of fluoride through the diffusion of monofluorophosphate by using a 70 percent aqueous glycerol or a 32 percent aqueous alcohol instead of water as solution.

The effect of various vitamins on fluoride metabolism have been studied. Earlier works did not note any changes in fluoride retention when fluoride was administered in vitamin preparations (Hennon, Stookey, and Muhler, 1964; Harkins, Longenecker, and Sarett, 1963). It was noted that large doses of thiamin had no effect on skeletal fluoride accretion (Nikitin, 1961; Stookey, 1974), riboflavin and cyanocobalamin

decreased skeletal fluoride deposition and pyridoxine enhanced this phenomenon (Nikitin, 1961).

BONE

The deposition of fluoride in hard tissues depends on the level of fluoride ingested and the age of the individual (Kuo and Stamm, 1974; and Zipkin, 1973). Fluoride deposits in apatite by substituting hydroxide ions to form fluoroapatite. No evidence of formed CaF_2 has been found in bone (Zipkin, 1973).

Osteoporosis is an absolute reduction in bone mass. The osteopororotic bone is histogically normal, with a normal mineral to matrix ratio (Bell et al., 1967). The disease is common, particularly beyond the age of 60, with an increasing incidence as aging ensues. Women are more often affected than men, particularly a few years after menopause.

Epidemiological Findings

One of the early suggestions that fluoride might benefit the skeleton followed the finding of Leone et al. (1960) that eight new cases of osteoporosis developed during a ten-year period in Framingham, Massachusetts (0.04 ppm fluoride) compared to a single new case in Bartlett, Texas (8 ppm fluoride). By x-rays it was found that fluoride increases the bone density, increases thickening of cortical bone and periosteum. Except for dental mottling, ingestion of water containing 8 ppm fluoride produces no deleterious bone changes.

Unusual incidence of bone fractures, arthritis, hypertrophic bone changes or exostoses, or interference with fracture healing, and evidence of associated functional or systemic effects were all absent.

In a radiological study, Bernstein et al. (1966) compared two populations of individuals having 4 to 5.8 ppm or 0.15 to 0.3 ppm fluoride, respectively, in the water supply. The incidences of osteoporosis, reduced bone density and collapsed vertebrae were substantially higher in the low fluoride area, especially in women. Visible calcification of the aorta was significantly higher in the low fluoride area, particularly in men. Alffram, Hernborg, and Nilsson (1969) found a greater skeletal mass in female residents of a high fluoride area (4 to 6.8 ppm fluoride) compared with women from a low fluoride area (0.2 to 0.4 ppm fluoride) (Table 5).

Therapeutic Uses of Fluoride

It has been amply confirmed that osteosclerosis can result from prolonged exposure to fluoride and that subjects with minimal or moderate osteosclerosis are usually asymptomatic and in good general health. This leads to the consideration that salts of fluoride might be used as treatment for patients with osteoporosis to cause a degree of osteosclerosis that would strengthen the skeleton but not lead to other changes (Rich, Ensink, and Ivanovich, 1964).

Bone diseases, as Paget's disease and multiple myeloma, have also been treated with fluoride (Lukert, Bolinger, and Meek, 1972; Cohen, 1966). The discussed studies are summarized in Table 6.

Effect of fluoride on bone

Roentgenologically, large, thick, heavy bone with increased bone density and increased mineralization is noted, after prolonged fluoride administration (Rich and Ivanovich, 1965; Dambacher, 1974; and Gedalia

Table 5. Epidemiological studies concerning the effect of fluoride on bone

ppm fluoride in water	Notations	Reference
8 ppm (Bartlett, Texas) 0.09 ppm (Framingham, Mass.)	Less osteoporosis in Bartlett	Leone et al., 1955
4-5.8 ppm 0.15-0.3 ppm	Osteoporosis, reduced bone density, collapsed vertebrae higher in the low F area, especially in women. Visible calcification of the aorta was sign, higher in low F area, particularly among men.	Bernstein et al., 1966
4.0-6.8 ppm 0.2-0.4 ppm	Bone density greater in high F area. (Low probability obtained.)	Alffram, Hernborg, and Nilsson, 1969

Table 6. Bone diseases treated with fluoride

Disease	Treatment	Time	Results	Reference
Osteoporosis	22 mg F twice daily before meal and Ca-lactate after meal	15 mo	Backpain ceased, fluorosis apparent on x-ray	Cohen and Garden, 1966
Osteoporosis	10-66 mg F/d	> 1 yr	Modest Ca retention, increase in bone density	Bernstein and Cohen, 1967
Osteoporosis	6-88 mg F/d		Iliac crest biopsies were hard to saw	Baylink and Bernstein, 1967
Osteoporosis	60 mg F/d	$\geq$ 14 w	Positive Ca balance	Rich and Ensinck, 1961
Pagets disease	60 mg F/d	$\geq$	Decreased uninary Ca	Rich and Ensinck, 1961
Severe primary osteoporosis	40-50 mg F/d	122 w	Ca retention increased, bone density increased	Rich and Ivanovich, 1965
Osteoporosis	45.4 mg F/d	1 yr	Stimulated bone formation	Dambacker and Haas, 1974
Osteoporosis	50 mg F/d	1 yr	Active bone formation	Cohen, Nichols, and Banks, 1969
Osteoporosis	20-60 mg F/d	12-42 mo	Alleviation of pain, 16% increase in bone density	Franke, Rempel, and Franke, 1974
Osteoporosis	22-91 mg F/d		Positive Ca retention increased bone mineralization	Bernstein et al., 1963

Table 6. Continued

Disease	Treatment	Time	Results	Reference
Pagets disease	Initially 28 mg F/d, followed by 9 mg F/d	2-3 mo	14 patients had lost much of their bone pain after 4-6 w, 6 patients came in more positive Ca balance	Purves, 1962
Otosclerosis	14, 28, or 56 mg F/d	2-3 mo	71% no significant change, 5.5% increase in spongy focus, 19% recalcification of the focus, 4.5% questionable improvement, 28 mg F/d was best treatment	Shambaugh and Petrovic, 1968
Otosclerosis	18-28 mg F/d	1-8 yr	Effective in 80% of cases in stabilizing the sensoneural component of loss of hearing	Shambaugh and Causse, 1974

et al., 1970). Fluoride depresses bone resorption (Petrovic and Shambaugh, 1966; and Faccini, 1967) through an inhibition of osteoclastic activity (Shambaugh and Chausse, 1974) and it also promotes calcium deposition (Shambaugh and Petrovic, 1967). Trabecular thickening was irregular and abnormal in appearance. Bernstein and Cohen (1967) suggested that because the formation rate is greater than the resorption, older areas of bone accumulate more calcium and therefore present a ragged appearance. Indices of bone formation were noted by Cohen, Nichols, and Banks (1969) and Rich and Ensinck (1961) as indicated by modestly increased serum alkaline phosphatase and urinary hydroxyproline excretion. Positive calcium balance was noted (Purves, 1962; and Bernstein et al., 1963).

Relief of bone pain by fluoride administration is reported (Bernstein et al., 1963; Purves, 1962; and Franke, Rempel, and Franke, 1974).

Shambaugh and co-workers (Petrovic and Shambaugh, 1966; Shambaugh and Petrovic, 1967, 1968; and Shambaugh and Causse, 1974) have sucessfully treated otosclerotic patients with fluoride. The disease results in a loss of hearing due to a localized osteoporosis of the labyrinthine capsule in the ear (actually the disease is osteoporotic or osteospongiotic and not a sclerotic condition).

Hodge and Smith (1968) summarized the studies of the effect of fluoride on osteoporosis. They reported that doses of 0.4 mg fluoride/kg bodyweight/day are necessary to observe a decrease in urinary calcium. To get relief from bone pain, 0.5 mg fluoride/kg/day or more for more than two months is required. The same dose is sufficient to get a positive calcium balance or lessened negative calcium balance, if it is

distributed more than 10 months. When 0.6 mg fluoride/kg/day or more is given for 14 to 15 months, osteosclerosis develops.

Compared to the above summary, the negative results from Lukert Bolinger, and Meek (1967), Cohn et al. (1971), and Henrikson et al. (1970) seem to depend on the short time for treatment, the low doses used and the small number of patients treated.

The effect fluoride produced on bone showed similar changes to those associated with rickets, such as low mineral density, many inactive areas covered with osteoid and an increased thickness of osteoid tissue (Milicic and Jowsey, 1968; and Spencer, Cohen and Garner, 1974). Morphologic studies of bone biopsy samples have shown that the predominant effect of fluoride therapy on the skeleton is osteoblastic stimulation (Jowsey, Schenk, and Reutter, 1968). The newly formed osteoid tissue is poorly mineralized resulting in the histologic picture of osteomalacia (Jowsey, Schenk, and Reutter, 1968; and Baylink and Bernstein, 1967). Bone from fluoride-treated patients was difficult to saw. The amount of bone forming surface was increased and it had developed a decreased solubility (Baylink and Bernstein, 1967). Spencer, Cohen, and Garner (1974) presented a hypothesis that fluoride interferes with the availability of calcium to cells. He based this on the following facts (1) a low bone mineralization defect becomes more severe with calcium deficiency, (2) the parathyroid is stimulated by fluoride, (3) an interaction of fluoride with thyrocalcitonin (TCT) in cortical remodeling, (4) leisons of fluoride toxicity are reduced by increased calcium in the ration, (5) normal blood calcium concentration occurs in all animals exposed to chronic doses of fluoride, (6) fluoride inhibits certain

enzymes, such as esterases, and thus might effect membranes or other cell functions (Lukert, Bolinger, and Meek, 1972), through alterations in the synthesis of citric acid inhibitors (Peter, Shorthouse, and Ward, 1969) and effects on ribonuclease (Chang, 1970).

The situation in which fluoride does not function like an anti-mineralization factor is when minimal toxic doses are given with plenty of calcium and phosphorus available for mineralization. Under these conditions, large, thick, heavy bones are formed and increased remodeling surface makes the bones more responsive to deposition to counter stresses. It is suggested that fluoride treatment could be a better preventative than theurapeutic measures for osteoporosis (Spencer, Cohen, and Garner, 1974; and Franke, Rempel, and Franke, 1974).

Fluoride given with a calcium deficient diet resulted in depressed serum calcium, abnormally wide osteoid tissue, and increased formation and resorption of bone (Buckhart and Jowsey, 1968). The subperiosteal activity was greater and the collagen matrix was abnormal (Forsyth et al., 1972). However, the administration of calcium has been reported to prevent the osteomalacic effect (Buckhart and Jowsey, 1968) and induced radiologic fluorosis (Cohen and Gardner, 1966).

Effect of fluoride together with other substances

Several reports show the beneficial effects of combining the fluoride dose with both calcium and vitamin D to receive a new bone which is normal both histologically and microradiologically (Jowsey et al., 1968, 1971, 1972). However, osteoporotic patients that received only a vitamin D supplement to the fluoride-dose also showed an increased bone

density (Reutter and Olah, 1974). Forsyth et al. (1972) included calcium and phosphorus with fluoride to increase bone density. Havivi (1972) reported that vitamin D_2 increases fluoride retention regardless of the calcium content of the diet.

Feeding a low calcium and low vitamin C diet together with fluoride administration, abnormal bone formation was induced. If calcium and vitamin C were adequate in the diet together with fluoride administration, it was found to mitigate the toxic effect of fluoride.

Fluoride in a low protein diet appeared to accelerate the development of rarefaction of bones (Reddy and Srikentia, 1971). Fluoride with adequate diet was found to increase the cumulative retention of labeled calcium (Reddy and Rao, 1971). Ericsson (1972) indicated a parallelism in the absorption and utilization of fluoride and calcium when given simultaneously in a 1:50 ratio as Na_2PO_3F and calcium-gluconate. In spontaneous and traumatic bone fractures, accelerated repair with thickened boney trabeculae was shown, with a combination treatment of NaF, NaCl and calcium (deGubareff and Platt, 1969).

Action of Hormones

A low intake of calcium causes a decrease in the serum calcium level, which stimulates the parathyroids to increase production of parathyroid hormone (PTH); this, in turn, increases bone resorption producing osteoporosis (Jowsey and Raisz, 1968). An administration of fluoride protects against the action of PTH (Levy et al., 1970). Thyrocalcitonin (TCT) acts by supressing the mobilization of calcium from bone (O'Riordan and Aurbach, 1968). The action of TCT is independent of PTH and it blocks

the entire mechanism regardless of the particular agent inducing bone resorption (Aliapoulious, Goldhaber, and Munson, 1966). The effects of PTH on bone have led to the hypothesis that new bone formation in skeletal fluorosis is due to stimulation of the parathyroid glands (Faccini, 1967). Fluoride administration can lead to secondary hyper parathyroidism owing to the failure of bone resorption to maintain a satisfactory serum concentration of ionized calcium at the parathyroid gland surface (Faccini, 1969).

A reduction in bone resorption was produced by both estrogens and androgens but high calcium intakes failed to change either bone formation or resorption (Lafferty, Spencer, and Pearson, 1964).

Mechanical Properties

Compression tests on humerus and femur from rats fed up to 200 ppm fluoride showed that fluoride did not affect the breaking strength (Saville, 1967). Further, feeding 10 ppm dietary fluoride did not affect cortical thinning, breaking load or chemical composition of the rat femur (Rao, Ts'ao, and Draper, 1972). But, rats fed 50 ppm fluoride showed a decreased breaking strain in the tibia (Nordenberg et al., 1971). Japanese quails fed 75 ppm dietary fluoride showed a 30 percent decrease in bone torsional strength (Chan, et al, 1973). Beary (1969) indicated that calcium is needed in the diet in order to give bone strength and that fluoride, at the 45 ppm range, will increase bone flexibility. Bell et al. (1967) used human vertebra and explained with Euler's equation how in osteoporosis the reduction of the diameter of the vertical trabeculae and the loss of transverse ties, causes a loss of strength

proportionately greater than the loss of osseous tissue. The quality of the osseous tissue is not changed. Bone microhardness increased in bone formed during fluoride treatment in rats given 30 ppm fluoride in drinking water (Yamamoto, Wergedal, and Baylink, 1974).

TOXIC EFFECTS OF FLUORIDE

High Levels of Fluoride

The most severe incidences of fluorosis have been presented by Jolly et al. (1969) and Singh et al. (1963). From a high fluoride area in Punjab, India, fluoride content varied from 1.2 to 16.2 ppm in the water. Strenuous work, excessive sweating, copious intake of water, diet deficient in animal protein and green vegetables was observed. When an individual has been exposed to 20 to 80 mg fluoride daily over 10 to 20 years, it results in crippling fluorosis yielding gross changes in the skeleton, with irregular bone deposits that lead to limitations of movement and neurological complications due to compression of the spinal cord.

Among children (0-5 years) the occurrence of mottled enamel in an area with 9.12 to 10.68 ppm fluoride in the drinking water was 82 percent. In the permanent dentition group ($\geq$ 6 years) 98.1 percent were suffering from mottled enamel, half of them were severely mottled, with well established brown stains.

In areas in the United States of America with natural water fluoride content of 8 ppm (Leone et al., 1955) no deleterious skeletal changes were found.

Normal Levels of Fluoride

Despite earlier reports, the prevalence of mongolism (Down's syndrome) was not higher in a high fluoride area compared with a non-fluoride area (Needleman, Pueschel, and Rothman, 1974).

The American Academy of Allergy stated, "There is no evidence of allergy or intolerance to fluoride as used in the fluoridation of community water supplies" (Editorial, 1971).

Parson et al. (1975) observed that renal patients tended to continue to excrete normal loads of fluoride quite well, until renal function was seriously reduced. Three months after renal transplantation fluoride excretion had reached normal levels.

The National Cancer Institute states that the NCI reaffirmed that its epidemiological studies show no relationship between the fluoridation of water and cancer. In fact, the results of the study rather suggest a protective influence from fluoride absorption (U.S. Department of Health, Education, and Welfare, 1975).

MATERIALS AND METHODS

Deciduous teeth were collected from children. Through Tooth Fairy advertisements in local newspapers in Logan, children in Logan sent their teeth to the Tooth Fairy at Utah State University. The collection of teeth from Milford, Helper, and Brigham City were made with help of teachers in the schools. A total collection of about 450 deciduous, permanent and animal teeth were obtained, and purchased at 50 cents each.

Contact was made with each child's parents to obtain data on the child in question. To fulfill the requirement to be used in this study, the child must have, since birth, been residing in each community, or in a community with equal fluoride concentration in the domestic water supply. Only deciduous teeth were used in the study.

Dentists examined the mouths of the children in Logan, Helper, and Milford. The number of decayed, missing, and filled permanent and deciduous teeth were reported.

Milford has a natural fluoride concentration of 0.8 ppm in the water. Helper and Brigham City have artificially fluoridated their water to 1 ppm ever since 1956 and 1966, respectively. The water in Logan contains only minute amounts of fluoride. Some children in Logan have received continuous fluoride treatments since birth through tablets, brushings, mouth rinses, topical treatments, etc.

A radiologist at the nearest community hospitals in Logan, Helper, and Milford, took an x-ray of the hands of the mothers of the selected

children. The women had to be residents for at least 10 years in each community (or a community with a similar level of fluoridation) to fulfill the criteria to participate in the study. On these x-rays, bone measurements as subperiosteal and medullary thickness at the mid-shaft of the second metacarpal was made by caliper. By the methods of Garn (1970), the cortical thickness, cortical and medullary areas, and percent cortical area were calculated.

The teeth were ground in a diamond mortar to a fine mesh powder. The powder was transferred via a funnel to a small glass tube, with the help of a brush.

Separation of dentine and enamel was based on a method of Manley and Hodge (1939). All glassware was rinsed with five percent NaOH and deionized water before use. Polypropylene and polyethylene items were used as much as possible to avoid the silica in glass which can absorb fluoride. Centrifuge tubes of polypropylene (17 x 100 mm) were filled with three to five ml of a bromoform-acetone mixture. The mixture consists of 91 volume percent bromoform and nine volume percent acetone, with a density of 2.6 to 2.7. An innertube of glass was constructed to fit the centrifuge tubes. The innertube had a conical end with an opening at the tip, as a pipet, and a flanged upper end that rested on the edge of the outer tube. The powdered tooth was dumped into the innertube. These tubes were centrifuged for 10 minutes at 2500 rpm. A polyethylene covered wire was used to bring down the sample that accidently was attached to the wall of the inner tube. The surface was also carefully stirred because enamel might be trapped in the floating dentine. Centrifuging was repeated twice with stirring in between.

To separate the layers, the innertube was closed with a finger and lifted out like a pipet and positioned again into a second tube of similar dimensions. Both layers in each tube were washed with acetone three times and centrifuged between each washing. The sample was set to dry after decanting off the acetone.

To measure the fluoride content of the enamel or dentine, a sample size of 1 to 20 mg was weighed in a centrifuge tube, as above. Into these, 1 ml of 0.50 M perchloric acid was added. Occasionally the tubes were agitated by mild shaking so that the sample did not become attached to the wall. With a polyethylene covered wire any material that became attached to the tube wall was pushed down into the solution. After one hour of acid digestion, 4 ml of 0.50 M trisodium citrate buffer was added. The pH was about 6 (McCann, 1968a). Fluoride activity could be measured immediately with an Orion Research Inc. fluoride ion activity electrode, model 94-09. The readings were made on a Corning Model 12 Research pH meter. To reach equilibrium faster, a stirring bar was used. It was made of a section of a paper clip that was covered with a polypropylene tube (size PE 50) and sealed at both ends. Care had to be taken that bubbles did not become adhered to the electrode membrane when the electrode was put down into the sample solution. Readings were made after precisely five minutes. Between the readings, the electrode was rinsed for three minutes in the citrate buffer solution. Standards ranged from 0.095 µg fluoride/5 ml to 95 µg fluoride/5 ml. Millivolt readings were plotted against fluoride concentrations on semilogerithmic paper. The fluoride concentrations in the samples were calculated after computing the equation for standard curve by the method of least squares

where x equalled the millivolt reading and y equalled the fluoride concentration and adjusting the slope of the intercept to account for dilution of the sample.

The data were tested by F-test, analysis of variance and Duncan's test (Steel and Torrie, 1960).

DISCUSSION

The fluoride content in enamel and in dentine showed a tendency and a significant increase, respectively, when originating from children exposed to optimal amounts of fluoride. This observation confirms earlier data. Triers, Elliott, and Smith (1968) showed a content of 67 ppm fluoride in enamel and 164 ppm fluoride in dentine from an area with a high dietary fluoride intake in Newfoundland. Data from a corresponding low fluoride area showed 47 ppm fluoride in the enamel and 94 ppm fluoride in the dentine of permanent teeth among 8 to 16 year old residents. Bervenmark and Hamberg (1974) had treated children daily for seven years with 0.5 mg fluoride. The deciduous incisors contained 103 ppm fluoride in the enamel and 155 ppm fluoride in the dentine. The control group had 58 ppm fluoride in the enamel and 79 ppm fluoride in the dentine (Table 4).

The fluoride content data in this study vary from 22 to 32 ppm fluoride in the enamel and from 53 to 96 ppm fluoride in the dentine. Compared to the above cited studies, these results are lower. During the acid digestion of the sample, not all of the sample went into solution. McCann (1968a), who designed this method, observed the same phenomenon, but did not report whether the total amount of the fluoride in the sample was liberated or not. This could account for the low concentrations of fluoride in the enamel and dentine in this study.

The fluoridation equipment, in Helper, is frequently shut down during the summer months when the water pressure is low. Therefore,

the exposure to fluoride is irregular during the year. The content of fluoride in dentine is lower in Helper than in Brigham City, Milford and Logan, full fluoride, where all children obtained optimal fluoride intake all year around. The partial fluoridated group from Logan contained children who had received fluoride during some years of their life, but were not eligible for the full fluoride-group or the no-fluoride group. This partial fluoride group was therefore very heterogeneous.

The dentine is a more vascular tissue than enamel and it has the ability to incorporate more fluoride than enamel. Elliott and Smith (1960) reported that the fluoride concentration in bulk enamel does not increase after a certain age or the increase is too gradual to be measurable. The fact that little replacement of the constituents of enamel takes place after calcification and eruption of a tooth suggests that the observed deposition of fluoride occurred during calcification, and subsequently either ceased or progressed at a very slow rate. Absorption of dietary fluoride on the enamel surface of the teeth takes place but only in the outermost layer (Charlton, Blainey, and Schamschula, 1974). Aasenden et al. (1971) could not show any differences in fluoride content in enamel surface between 9 and 11 year old children from a 0.1 ppm fluoride and 1.0 ppm fluoride area, respectively. Poulsen and Larsen (1975) failed to correlate low enamel fluoride level with prevalence of dental caries for individuals from low fluoride areas.

A definite relationship between levels of fluoride in dentine and the age of the child was found, when the dietary fluoride intake was high, through a diet based on fish, tea and flour fortified with fluoride

containing bonemeal. The water borne fluoride (1.3 ppm) in the same study showed a higher incorporation in enamel (128 ppm) and dentine (325 ppm) than the dietary fluoride-incorporation (Triers, Elliott, and Smith, 1968). Similar results were obtained by McClure and Likins (1951).

To get a ratio for lesions in each mouth, an average of all decayed, missing, and filled deciduous and permanent teeth were summarized together. The children aged from 8.3 to 10.2 years (no significant difference) among the communities. By this age many of the deciduous teeth are lost, but the number of permanent teeth is not complete, and both def and DMF data would be very low.

The lesion/mouth ratio showed a significant difference between the various groups. The fluoride treated Logan children had 41 percent fewer lesions/mouth compared to non-fluoride treated children in Logan. Both the children in Milford and Helper had a higher incidence of lesions/mouth compared to non-fluoride treated and fluoride-treated children in Logan. The water in these communities contain almost optimal amounts of fluoride. This might be explained by the difference in other variables, as the socioeconomic status that is not comparable to Logan or Brigham City. These two communities are mining and railroad towns, respectively. Development of tooth cavities depends not only on the fluoride content in the teeth, but also on nutritional status and especially on oral hygiene.

Holm et al. (1975) concluded that children of parents with a higher education level had lower caries indexes. That education and income separately and together were very highly correlated with preventive dental care was summarized in a review of Rayner (1970) and by Perlus (1972).

The socioeconomic variables between the no-fluoride treated children and the fluoride-treated children in Logan are more likely to be similar. The fewer incidence of leisons/mouth are, therefore, comparable.

The intake of fluoride in the Logan children has been dependent on the daily consumption of the fluoride tablets or drops. Even if a continuous intake has been claimed, the actual intake might be somewhat less due to occasional failure to dispense the drops or tablets or the failure of the children to ingest them once dispensed.

Caries reduction data from Brigham City are available through a study by Wright (1975). Deciduous decayed, missing and filled teeth data showed a 30 percent reduction and permanent decayed, missing and filled teeth showed a 44 percent reduction after eight years of fluoridation. Previous studies concerning fluoride treated children are summarized in Tables 1 and 2. Results of this study correlates well with the reductions observed earlier.

Prevalence of osteoporosis was lower in a high fluoride area, with 8 ppm fluoride (Bartlett) than in an area with minute amounts of fluoride (Leone et al., 1960). Similar results were obtained by Bernstein et al. (1966) in their North Dakota study.

The outer, subperiosteal surface of the second metacarpal in the hand shows at midshaft a general predilection for apposition, which continues throughout life. This expansion is partially a compensate for the inner, endosteal, bone losses. Until adulthood prepuberally, bone is lost at the endosteal surface. Steroid mediation, especially during pregnancy, results in a gain of endosteal bone. After the fourth decade, resorption starts again, in both sexes, peaking in the sixth

decade and followed by a decline with a greater relative endosteal loss in the female at adult ages (Garn, 1970).

For studies of osteopenia and bone loss, Percent Cortical Area (PCA) is a useful measure, because PCA is very similar in various populations. The cortical thickness has both genetic and nutritional determinants. It is thicker in Blacks and thinner in Mexican-Americans, Japanese and Chinese. In many osteopenias, the cortex is reduced through excessive endosteal resorption, and this is true in malabsorption diseases (protein-calorie-malnutrition, sprue, celiac disease). Increased bone loss is observed in immobilization of bone, due to low calcium diets, chelating agents, cyanosis, leukemia, and castration.

The PCA values at the midshaft of the second metacarpal in this present study were not different between women living in an area with 1 ppm fluoride in the domestic water supply (Helper and Milford) for ten years or more and women living in a non-fluoride area (Logan). Ansell and Lawrence (1965) reported a lower prevalence of osteoporosis among women after five years of water fluoridation (1 ppm). The prevalence was based on x-rays from hand and cervical spine. Only women over 65 years of age were x-rayed. Goggin et al. (1965) could not show any differences in femoral fracture rates among women at 60 and over, when the water had been fluoridated to 1 ppm for five years. Neither could Korns (1969) report any differences in hip or wrist fractures among women 60 years and over, after 22 years of fluoridation (1 ppm).

The prevalence of osteoporosis in high-fluoride areas is lower than in low fluoride areas. The mechanism by which fluoride would

exert its action in bone would be by stabilizing the apatite phase, under conditions of demineralization, and by favoring its formation during remineralization (Grøn, Brudevold, and Aasenden). In a high-fluoride area (4.0 to 6.8 ppm) the bone mass in limbs, among women, was demonstrated to be greater than among women from a low fluoride area (0.2 to 0.4 ppm). They obtained a low significance when they measured the second metacarpal on its combined thickness of the two cortci as a fraction of the total thickness of the metacarpal (Alffram, Hernborg, and Nilsson, 1969). Based on earlier reports and this one, it seems that to show a difference in bone mass, the fluoride content in the water must exceed 1 ppm. The average age of the women in this study is 35.7 years, an age where the onset of the postmenopausal resorption of the endosteal surface has not started. No age differences between the communities were observed.

RESULTS

The fluoride concentration in dentine was significantly higher ($P < 0.05$) in Brigham City, and in Logan, with full fluoride treatment compared to Logan without fluoride treatment. No significance was obtained between Logan, no fluoride, and Milford, Helper, or Logan, partial fluoride (Table 7 and Figure 1).

The fluoride concentration in enamel showed a tendency to be increased among the children who had been exposed to fluoride compared to the non-fluoride treated children in Logan (Table 7 and Figure 1).

All decayed, missing, or filled deciduous and permanent teeth were summarized per mouth in a lesions/mouth ratio. Logan children with fluoride treatment had 41 percent fewer lesions than Logan children without fluoride treatment ($P < 0.05$). Incidence of lesions in Helper and Milford was higher than for children with no fluoride treatment (Table 7).

Bone data, as cortical thickness, cortical and medullary areas and percent cortical area were measured from x-rays of the second metacarpal of women in Logan, Helper and Milford. No significant differences between the different communities were observed. The ages of the women averaged 36.2 years (Logan), 35.9 years (Helper) and 34.9 years (Milford) (Table 8).

The analyses of variance for the enamel, dentine, and lesions/mouth data are presented in the Appendix.

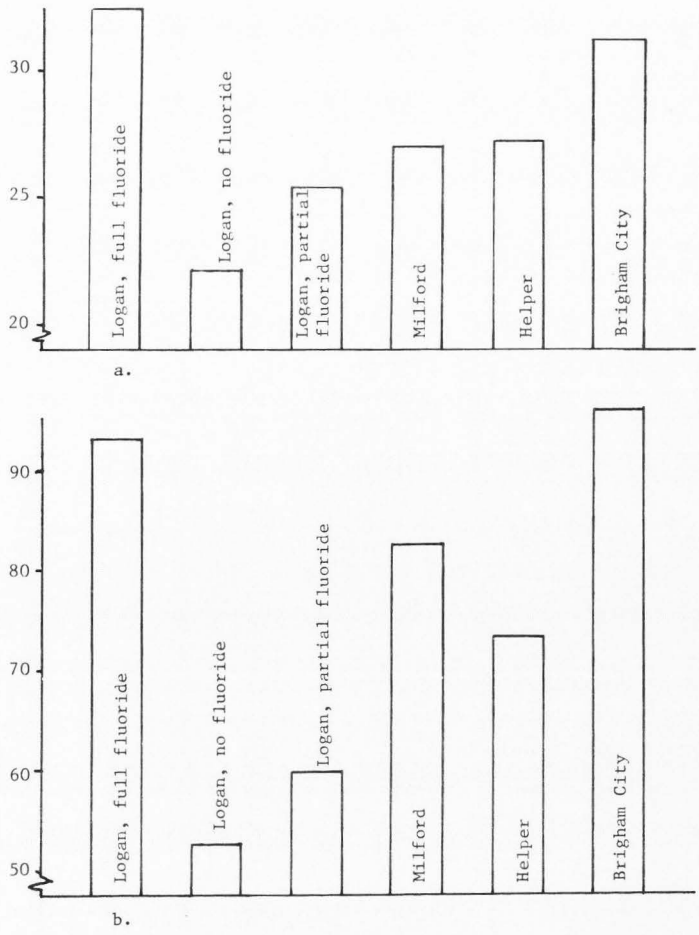

Figure 1.

a. Fluoride content (ppm) in enamel from four Utah communities.

b. Fluoride content (ppm) in dentine from four Utah communities.

Table 7. Average fluoride content in deciduous enamel and dentine and the lesions/mouth from children in four Utah communities

	ppm fluoride in enamel			ppm fluoride in dentine			Lesions/mouth		
	#	$\bar{x}$	$S_{\bar{x}}$	#	$\bar{x}$	$S_{\bar{x}}$	#	$\bar{x}$	$S_{\bar{x}}$
Logan, full fluoride	12	32.45	5.98	14	93.07[a]	15.06	10	3.90[b]	1.34
Logan, no fluoride	14	22.10	4.64	18	52.66	5.85	11	6.64	1.24
Logan, partial fluoride	8	25.43	3.35	10	59.92	8.17	-	-	-
Milford	12	27.03	4.43	15	82.49	9.99	12	7.42	1.72
Helper	12	27.18	3.92	12	73.28	9.20	9	10.00[c]	1.84
Brigham City	14	31.27	4.50	15	95.85[a]	16.58	-	-	-

[a]Significantly higher than Logan, no fluoride ($P < 0.05$).
[b]Significantly lower than Logan, no fluoride ($P < 0.05$).
[c]Significantly higher than Logan, no fluoride ($P < 0.05$).
$\bar{x}$ = mean.
$S_{\bar{x}}$ = standard error of mean.

Table 8. Cortical thickness, cortical area, medullary area and percent cortical area measured at the midshaft at the second metacarpal on women from three Utah communities

	#	Age	CT*		CA*		MA*		PCA*	
			$\bar{x}$	$S_{\bar{x}}$	$\bar{x}$	$S_{\bar{x}}$	$\bar{x}$	$S_{\bar{x}}$	$\bar{x}$	$S_{\bar{x}}$
Logan	19	36.2	5.79	0.18	49.24	2.15	5.07	0.77	94.22	3.84
Helper	17	35.9	5.51	0.17	46.77	2.13	6.36	1.05	86.21	2.77
Milford	16	34.9	5.71	0.22	44.83	1.23	4.17	0.58	91.45	1.20

*CT-cortical thickness.
CA-cortical area.
MA-medullary area.
PCA-percent cortical area.
$\bar{x}$ = mean.
$S_{\bar{x}}$ = standard error of mean.

SUMMARY

Deciduous teeth from Logan, Milford, Helper, and Brigham City were investigated for fluoride content. The amount of fluoride in enamel was not significantly different though there was a tendency to higher concentrations of fluoride in teeth of children that had received fluoride. The water in Logan contains only 0.2 ppm, Milford has a natural fluoride content of 0.8 ppm, the water in Helper is irregularly fluoridated to 1 ppm, since 1955, and Brigham City has fluoridated water with 1 ppm since 1966. The content of fluoride in dentine showed a significant difference between Logan children who had received no fluoride and Logan children who had received fluoride in tablets and topical treatments as well as children from Brigham City.

The lesions/mouth indicated that fluoride treated children in Logan had 41 percent fewer lesions than non-fluoride treated children in Logan. The fluoride containing communities of Helper and Milford showed higher prevalence of lesions than all other groups. This might be explained by the economical status and, therefore, poorer oral hygiene.

Bone measurements on the second metacarpal in the hand of women, averaging 35.7 years of age, did not show any significant differences in cortical thickness, cortical area, medullary area, or percent cortical area. The onset of bone rarefaction commences after the menopause, when there is no longer a hormonal protection in women.

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APPENDIX

Table 9. Analysis of variance for dentine, enamel, and lesions/mouth data

	df	SS	MS	F
Analysis of variance for dentine data				
Total	83	170,722.37		
Treatment	5	23,098.78	4,619.75	2.44*
Error	78	147,623.59	1,892.61	
Analysis of variance for enamel data				
Total	71	18,446.59		
Treatment	5	967.87	193.57	.75
Error	66	17,478.72	264.82	
Analysis of variance for the lesions/mouth data				
Total	42	1,165.16		
Treatment	3	239.11	79.70	3.36*
Error	39	926.05	23.74	

Table 10. Tables for average age, content of fluoride (ppm) in deciduous enamel, dentine and lesions/mouth for Logan, full fluoride; Logan, no fluoride; Logan, partial fluoride; Helper; Milford; Brigham City.

Subject	Age	ppm fluoride		def	DMF	Lesions/mouth = def + DMF
		Enamel	Dentine			
Logan, full F						
1	11	12.6	51.5	7	8	15
1	(11)*	25.5	55.5	-	-	-
2	8	-	107.5	2	0	2
1	(11)	16.6	35.2	-	-	-
1	(11)	11.1	55.7	-	-	-
3	13	14.8	57.3	0	4	4
4	(7)	51.3	127.0	-	-	-
5	11	25.4	71.2	0	1	1
6	10	52.8	201.0	1	3	4
7	(7)	-	107.1	-	-	-
8	10	-	-	0	1	1
9	6	48.2	78.1	0	0	0
10	9	70.6	216.1	0	3	3
11	11	49.3	101.3	0	4	4
12	13	11.2	38.5	0	5	5
n	10	12	14			10
Σx	102.00	389.40	1303.00			39.00
Σx^2	1082.00	17355.04	162526.18			313.00
$\bar{x}$	10.2	32.45	93.07			3.90
S_d	2.15	20.71	56.33			4.23
$S_{\bar{x}}$	.68	5.98	15.06			1.34

*Age in parentheses is not counted into the average age.

Table 10. Continued

Subject	Age	ppm fluoride		def	DMF	Lesions/mouth = def + DMF
		Enamel	Dentine			
Logan, nonfluoride						
1	6	39.5	65.3	7	0	7
2	9	-	27.5	2	3	5
2	(9)	-	44.4	-	-	-
3	10	5.5	21.8	2	5	7
4	10	48.7	95.2	7	4	11
5	13	6.8	17.8	0	0	0
6	9	53.4	104.7	9	7	16
7	12	12.8	45.8	1	5	6
8	6	7.9	39.3	1	6	7
9	11	7.6	49.4	3	0	3
10	7	-	90.6	7	1	8
11	(13)	5.0	41.6	-	-	-
12	(7)	-	72.3	-	-	-
13	(7)	25.3	53.1	-	-	-
13	(7)	38.4	52.8	-	-	-
14	(6)	8.8	26.1	-	-	-
15	(7)	16.0	53.6	-	-	-
16	6	33.7	46.5	3	0	3
n	11	14	18			11
Σx	99	309.40	947.80			73
Σx^2	953	10752.78	60387.48			667
$\bar{x}$	9.00	22.10	52.66			6.64
S_d	2.49	17.35	24.82			4.27
S_x	.75	4.64	5.85			1.29

Table 10. Continued

Subject	Age	ppm fluoride		def	DMF	Lesions/mouth = def + DMF
		Enamel	Dentine			
Logan, partial fluoride						
	5	19.3	95.2			
	10	23.0	37.6			
	5	12.7	19.3			
	7.5	-	32.8			
	6	31.2	85.2			
	6	43.0	67.1			
	12	-	90.4			
	8	23.2	65.1			
	11	19.4	61.9			
	8	31.6	44.6			
n	10	8	10			
Σx	71.5	203.40	599.20			
Σx^2	608.25	5798.38	41917.52			
$\bar{x}$	7.15	25.43	59.92			
S_d	3.28	9.46	25.85			
$S_{\bar{x}}$	1.04	3.35	8.17			

Table 10. Continued

Subject	Age	ppm fluoride		def	DMF	Lesions/mouth = def + DMF
		Enamel	Dentine			
Helper						
1	11	12.6	27.8			5
1	(11)	23.9	-			-
2	(7)	8.6	35.1			-
3	6	42.1	108.7			8
4	(6)	38.2	82.7			-
5	8	43.3	117.8			14
6	10	27.8	87.0			3
7	10	17.1	52.7			6
8	10	50.7	96.8			7
9	(7)	26.3	53.2			-
10	(6)	14.7	75.0			-
11	7	20.8	34.2			14
12	-	-	108.3			-
13	7	-	-			13
14	6	-	-			20
n	9	12	12			9
Σx	75	326.10	879.30			90.00
Σx^2	655	10886.63	75606.97			1144.00
$\bar{x}$	8.33	27.18	73.28			10.00
S_d	1.94	13.57	31.88			5.52
$S_{\bar{x}}$	.65	3.92	9.20			1.84

Table 10. Continued

Subject	Age	ppm fluoride		def	DMF	Lesions/mouth = def + DMF
		Enamel	Dentine			
Milford						
1	7	43.5	121.9	14	4	18
2	(8)	24.1	79.2	-	-	-
3	6	42.6	167.6	0	0	0
4	12	27.1	81.0	0	3	3
5	6	14.7	78.5	12	0	12
6	7	4.3	20.9	5	0	5
7	6	-	85.8	1	4	5
8	8	39.7	122.6	1	0	1
9	11	11.3	32.6	6	7	13
10	(6.5)	-	83.7	-	-	-
11	9	22.5	66.2	14	2	16
12	10	15.9	41.8	3	1	4
13	12	22.7	119.6	2	6	8
14	7	-	84.1	4	0	4
14	(7)	55.9	51.8	4	0	(4)
n	12	12	15			12
$\sum x$	101.00	324.30	1237.30			89.00
$\sum x^2$	909.00	11359.75	123032.81			1049.00
$\bar{x}$	8.42	27.03	82.49			7.42
S_d	2.31	15.36	38.70			5.95
$S_{\bar{x}}$	.67	4.43	9.99			1.72

Table 10. Continued

Subject	Age	ppm fluoride		def	DMF	Lesions/mouth = def + DMF
		Enamel	Dentine			
Brigham City						
1	7	56.0	179.8			
2	7	39.4	92.8			
3	7	43.5	102.5			
4	6	42.1	108.5			
5	5	48.2	161.8			
6	8	53.3	169.4			
7	14	19.4	22.8			
8	8	39.3	132.8			
9	6	-	217.0			
10	11	14.5	34.5			
11	10	4.6	36.0			
12	10.5	15.6	29.9			
13	12	25.1	77.7			
14	10	26.5	45.3			
15	11	9.8	26.9			
n	15	14	15			
Σx	132.5	437.3	1437.7			
Σx^2	1264.25	17341.07	195531.91			
$\bar{x}$	8.83	31.24	95.85			
S_d	2.59	16.83	64.22			
$S_{\bar{x}}$	.67	4.50	16.58			

Table 11. Analysis of variance for the lesions/mouth

Logan full fluoride and Logan no fluoride

	#	$\bar{x}$	Σx	Σx^2	$(\Sigma x)^2/n$
Logan-full fluoride	10	3.90	39	313	152.10
Logan-no fluoride	11	6.64	73	658	484.45
Σ	21		110	971	636.55

	df	SS	MS	F
Total	20	396		
Treatment	1	61.55	61.55	3.52*
Error	19	334.45	12.5	

$$SS_{tot} = \Sigma x^2 - \frac{(\Sigma X)^2}{N}$$

$$SS_{treat} = (\Sigma x)^2/n - \frac{(\Sigma X)^2}{N}$$

$$MS_{treat} = SS_{treat}/df_{treat}$$

$$MS_{err} = SS_{err}/df_{err}$$

$$F = MS_{treat}/MS_{err}$$

Conclusion: A significant difference between fluoride treated and non fluoride treated Logan children ($P \geq 0.05$) in lesions/mouth.

Table 11. Continued

Logan full fluoride and Helper

	#	$\bar{x}$	Σx	Σx^2	$(\Sigma x)^2/n$
Logan-full fluoride	10	3.90	39	313	152.10
Helper, 1 ppm fluoride	9	10.00	90	1144	961.10
Σ	19		129	1452	1113.20

	df	SS	MS	F	
Total	18	581.16			
Treatment	1	237.36	237.36	11.74*	
Error	17	343.80	20.22		

Conclusion: There is a significant difference ($P > 0.01$) between Logan-no fluoride and Helper in lesions/mouth.

Logan full fluoride and Milford

	#	$\bar{x}$	Σx	Σx^2	$(\Sigma x)^2/n$
Logan-full fluoride	10	3.90	39	313	152.10
Milford	12	7.42	89	1049	660
Σ	22		128	1362	810.10

	df	SS	MS	F	
Total	21	617.27			
Treatment	1	65.37	65.37	237	NS
Error	20	551.90	27.60		

Conclusion: There is no significant difference between the lesions/mouth in Logan-full fluoride to Milford.

Table 11. Continued

Logan-no fluoride and Helper

	#	$\bar{x}$	Σx	Σx^2	$(\Sigma x)^2/n$
Logan-no fluoride	11	6.64	73	658	484.45
Helper	9	10.00	90	1144	961.10
Σ	20		163	1802	145.55

	df	SS	MS	F	
Total	19	473.55			
Treatment	1	117.10	117.10	5.91*	
Error	18	356.45	19.80		

Conclusion: Significant difference ($P > 0.05$) between Logan-no fluoride and Helper in lesions/mouth.

Logan-no fluoride and Milford

	#	$\bar{x}$	Σx	Σx^2	$(\Sigma x)^2/n$
Logan-no fluoride	11	6.64	73	658	484.45
Milford	12	7.42	89	1049	660
Σ	23		162	1707	1144.45

	df	SS	MS	F	
Total	22	565.96			
Treatment	1	3.41	3.41	0.13	NS
Error	21	562.55	26.79		

Conclusion: There is no significant difference between the lesions/mouth in Logan-no fluoride and Milford.

Table 12. Average ages and average bone measurements from the midshaft of the left and right second metacarpal in the hands in women from Logan

Subject	Age	T[#]	C[*]	M[+]	CA[†]	MA[α]	PCA[β]
1	39	8.65	5.90	2.75	52.72	6.033	89.76
2	30	8.60	5.35	3.25	49.90	8.294	85.95
3	32	8.50	6.05	2.45	52.12	4.730	91.90
4	31	8.55	6.15	2.40	52.81	4.593	92.03
5	31	7.25	5.90	1.35	39.80	1.480	96.46
6	31	7.70	5.70	2.00	74.10	2.140	159.21
7	41	7.55	4.60	2.85	38.37	6.378	85.75
8	40	9.40	7.55	1.95	66.65	2.846	96.09
9	51	8.35	5.25	3.10	46.97	7.929	85.82
10	29	9.10	6.40	2.70	59.28	5.731	91.19
11	37	7.95	6.05	1.90	46.80	2.834	94.33
12	44	7.90	5.30	2.60	43.67	5.338	89.14
13	34	8.05	6.80	1.25	49.64	1.245	97.58
14	45	7.95	6.70	1.25	48.55	1.229	97.86
15	32	7.55	5.75	1.80	42.15	2.614	94.20
16	29	8.20	4.95	3.25	44.48	8.310	84.27
17	34	6.85	6.00	0.85	36.27	0.585	98.47
18	44	8.20	4.65	3.55	42.95	9.911	81.37
19	34	8.85	4.80	4.05	48.42	13.114	78.75

[#]Total subperiosteal diameter.
[*]Cortical thickness T - M.
[+]Medullary cavity diameter.
[†]Cortical area 0.785 $(T^2 - M^2)$.
[α]Medullary area 0.785 M^2.

[β]Percent cortical area $\frac{0.785\ (T^2 - M^2)}{0.785\ T^2} \times 100$

	Age	C	CA	MA	PCA
n	19	19	19	19	10
Σx	688	109.95	935.65	96.32	1790.11
Σx^2		646.82	47662.77	691.07	173710.78
$\bar{x}$	36.21	5.79	49.24	5.07	94.22
S_d	6.40	.77	9.39	3.36	16.76
S_x		.18	2.15	.77	

Table 13. Average ages and average bone measurements from the midshaft of the left and right second metacarpal in the hand, in women from Helper

Subject	Age	T	C	M	CA	MA	PCA
1	41	8.00	4.95	3.05	42.93	7.320	85.45
2	23	7.75	6.25	1.50	45.39	1.775	96.27
3	45	7.65	4.35	3.30	37.40	8.557	81.41
4	30	7.15	5.10	2.05	36.83	3.301	91.77
5	41	9.80	5.55	4.25	61.20	14.197	81.18
6	31	7.15	5.90	1.25	38.92	1.229	96.98
7	30	7.50	5.40	2.10	40.62	3.533	91.99
8	37	9.10	5.10	4.00	52.46	12.560	80.70
9	40	7.05	5.25	1.80	36.47	2.552	93.47
10	36	8.10	5.45	2.65	45.91	5.609	89.14
11	40	8.65	6.35	2.30	54.85	4.224	93.38
12	28	8.70	4.40	4.30	44.97	14.523	75.69
13	39	8.80	5.20	2.80	44.09	6.186	72.53
14	45	9.20	6.15	3.05	59.05	7.399	88.87
15	30	9.65	6.10	3.55	63.21	9.911	86.47
16	34	8.20	7.05	1.15	52.03	1.134	98.51
17	40	7.4	5.10	2.30	38.83	4.153	90.33

	Age	C	CA	MA	PCA
n	17	17	17	17	17
Σx	610	93.66	795.16	108.16	1465.51
Σx^2		524.11	38430.53	990.62	128424.24
$\bar{x}$	35.88	5.51	46.77	6.36	86.21
S_d	6.32	.71	8.79	4.35	11.43
S_x		.17	2.13	1.05	

Table 14. Average ages and average bone measurements from the midshaft of the left and right second metacarpal in the hand, in women from Milford

Subject	Age	T	C	M	CA	MA	PCA
1	45	8.00	6.35	1.65	48.06	2.187	95.66
2	30	8.30	7.85	0.45	53.93	0.161	99.73
3	27	8.15	4.95	3.20	44.08	8.070	84.54
4	31	8.20	6.90	1.30	51.42	1.398	97.42
5	37	7.80	5.20	2.60	42.46	5.315	88.90
6	30	7.80	5.35	2.45	43.16	4.730	90.37
7	-	6.95	4.85	2.10	34.45	3.470	90.86
8	43	8.00	6.45	1.55	48.50	1.935	96.54
9	37	7.65	5.30	2.35	41.61	4.353	90.57
10	34	7.70	5.15	2.55	41.45	5.107	89.06
11	33	8.00	6.25	1.75	47.84	2.406	95.22
12	46	8.05	5.60	2.45	46.14	4.730	90.70
13	33	7.65	4.55	3.10	38.39	7.552	83.57
14	38	7.85	6.20	1.65	46.21	2.187	95.53
15	31	7.85	4.85	3.00	41.40	7.073	85.58
16	29	8.30	5.55	2.75	48.13	5.986	89.00

	Age	C	CA	MA	PCA
n	15	16	16	16	16
Σx		91.35	717.23	66.66	1463.25
Σx^2		533.35	32516.63	359.12	134162.84
$\bar{x}$	34.93	5.71	44.83	4.17	91.45
S_d	5.93	.89	4.94	2.33	4.79
$S_{\bar{x}}$		.22	1.23	.58	

Table 15. Analysis of variances for bone measurements at the midshaft of the second metacarpal

Cortical thickness

	#	$\bar{x}$	Σx	Σx^2	$(\Sigma x)^2/n$
Logan	19	5.79	109.95	646.82	636.26
Helper	17	5.51	93.65	524.11	515.90
Milford	16	5.71	91.35	533.35	521.55
Σ	52		294.95	1704.28	1673.72

	Df	SS	MS	F	
Total	51	31.12			
Treatment	2	0.73	0.365	0.59	NS
Error	49	30.39	0.62		

$SS_{tot} = \Sigma x^2 - (\Sigma X)^2/N$

$SS_{treat} = (\Sigma x)^2/n - (\Sigma X)^2/N$

$SS_{err} = SS_{tot} - SS_{treat}$

$MS_{treat} = SS_{treat}/df_{treat}$

$MS_{err} = SS_{err}/df_{err}$

$F = MS_{treat}/MS_{err}$

Conclusion: No significance in cortical thickness between Logan, Helper, and Milford.

Table 15. Continued

Cortical area

	#	$\bar{x}$	Σx	Σx^2	$(\Sigma x)^2/n$
Logan	19	49.24	935.65	47662.77	46075.84
Helper	17	46.77	795.16	38430.53	37192.91
Milford	16	44.83	717.23	32516.63	32151.18
Σ	52		2448.04	118609.93	115419.93

	df	SS	MS	F	
Total	51	3361.86			
Treatment	2	171.86	85.93	1.32	NS
Error	49	3190.00	65.10		

Conclusion: No significant difference in cortical area between Logan, Helper, and Milford.

Medullary area

	#	$\bar{x}$	Σx	Σx^2	$(\Sigma x)^2/n$
Logan	19	5.07	96.33	691.07	488.39
Helper	17	6.36	108.16	990.62	688.15
Milford	16	4.17	66.66	359.12	277.72
Σ	52		271.15	2040.92	1454.27

	df	SS	MS	F	
Total	51	627.03			
Treatment	2	40.38	20.19	1.69	NS
Error	49	586.65	11.97		

Conclusion: No significant difference in medullary area between Logan, Helper, and Milford.

Table 15. Continued

Percent cortical area

	#	$\bar{x}$	Σx	Σx^2	$(\Sigma x)^2/n$
Logan	19	94.22	1790.11	173710.78	168656.82
Helper	17	86.21	1465.51	128424.24	126336.44
Milford	16	91.45	1463.25	134162.84	133818.79
Σ	52		4718.87	436297.86	428812.05

	df	SS	MS	F	
Total	51	8072.19			
Treatment	2	586.38	293.19	1.91	NS
Error	49	7485.81	152.77		

Conclusion: No significant difference in percent cortical area between Logan, Helper, and Milford.

VITA

Monika Margareta Löwgren

Candidate for the Degree of

Master of Science

Thesis: Effects of Fluoride Availability on Fluoride Content of Deciduous Teeth of Children and Bone Measurements of the Mothers in Some Utah Communities

Major Field: Nutrition

Biographical Information:

Personal Data: Born in Gävle, Sweden, Sept. 6, 1951, daughter of Gunnar and Ann-Marie Löwgren; two brothers, Claes and Per; one sister, Malin.

Education: Attended elementary school in the Swedish school in Hamburg, Germany, and continued in elementary school in Oxelösund, Sweden; attended "gymnasium" in Nyköping and matriculated 1970; attended the Universities of Upsala and Stockholm in Sweden. Received Filosofie Kandidatexamen in Nutrition at University of Stockholm in 1974. Received the Rotary International Graduate Fellowship for 1974-1975; attended Utah State University and fulfilled the requirements for Master of Science in Nutrition in 1976.

Work Experience: Worked five consecutive summers in the chemical laboratory at Gränges Steel in Oxelösund. Been in charge of the metabolic kitchen at the research hospital "Serafimerlasarettet" in Stockholm. During one semester from August 1975 to December 1975, worked as a research graduate assistant in the Department of Nutrition at Utah State University.