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Editorial

DENTAL FLUOROSIS AND FLUORIDATION

Dental fluorosis, an often unsightly condition of the teeth involving diffuse, symmetrically-arranged opacities of the enamel, sometimes with yellow or brown staining and pitting, has been the focal point of research ever since its apparent connection with drinking water was discovered at the beginning of this century. Even before then, early settlers of the U.S. Southwest had encountered it as a form of dental mottling which they called "Texas teeth." But it was not until the year 1931 that fluoride in water consumed during infancy and early childhood was independently recognized as the specific cause by three different teams of scientists (1-3).

In the 1930s, the U.S. Public Health Service conducted surveys to identify endemic areas and correlated them with the fluoride content of local water supplies. At the same time, data became available suggesting that fluoride in drinking water might also be associated with lower rates of dental caries, especially in children. By 1950, after only five years of trials, health officials agreed to raising the concentration of fluoride in drinking water to 1 mg/L (1 ppm) as a desirable "optimum" for "the point of minimum caries and maximum safety" (4) even though they recognized that some individuals would thereby also develop some degree of dental fluorosis.

Previously, in 1943, a concentration of more than 1 ppm fluoride in water had constituted grounds for rejection of a water source because of potential harm (5). Actually, numerous reports of dental fluorosis from levels well below 1 ppm in natural fluoride water (6-10) and from "optimally" fluoridated drinking water (11,12) have appeared. Moreover, dental fluorosis has been reported from fluoride in food and beverages (13-14), from pharmaceuticals (15), from fluoride administered in tablets (16,17), from fluoride-polluted air (18,19), and from various combinations of the foregoing.

It should also be noted that a 1957 report of the American Medical Association (AMA) viewed dental fluorosis as "the most delicate criterion of harm from fluoride (ingestion)"(20). In 1955, crippling skeletal fluorosis had been recorded from as little as 2 ppm fluoride in the drinking water (21). In that same year, the first report of chronic fluoride poisoning from artificially fluoridated water was published (22). Subsequently, some 400 well-documented cases of preskeletal fluorosis from drinking 1 ppm fluoridated water have appeared (23-27).

Where water is fluoridated, many people are now estimated to be consuming 3-5 mg of fluoride per day (28) instead of the originally anticipated 1.5 mg/day from food and drink (29). In 1975, an AMA report (30) asserting the efficacy and safety of fluoridation acknowledged that the average adult consumption of fluoride in a fluoridated community was as high as 5 mg/day.

What does all this mean with respect to dental fluorosis? A 1982 article on "Fluorides and the Changing Pattern of Dental Caries" disclosed that 28% of children 11 to 13 years of age in fluoridated U.S. communities exhibited dental fluorosis (mostly mild), whereas less than 15% were expected to be so affected (31). The following year, the results of a USPHS
survey in Illinois were published (32). They showed that definitely disfiguring (moderate and severe) dental fluorosis was occurring in school children in a community (Kewanee) where the level of fluoride naturally in the water was "optimal" (1.06 ppm). No other sources of excessive fluoride intake besides the drinking water could be identified. In several other Illinois communities with higher fluoride concentration (3-4 ppm), increased dental fluorosis was found to be associated with decreased resistance to dental caries.

Thus, it should not come as a surprise that the recently retired Principal Dental Health Officer of New Zealand's largest city, Auckland, found it necessary to report the occurrence of disfiguring dental fluorosis from artificially fluoridated drinking water (33). This broadly-based study which involved nearly 2700 lifetime resident children, aged 7 to 12, also included pertinent socio-economic (income) level comparisons. Overall, the survey revealed a dental fluorosis incidence of 24.86% (486 cases among 1955 children) in the fluoridated Auckland area. In the nonfluoridated area, on the other hand, only 29 of 732 children (3.96%) had dental fluorosis. Moreover, the incidence of obviously disfiguring (staining and/or pitting) categories was 3.58% (70 cases) in the fluoridated area compared to only 2 cases (0.27%) in the nonfluoridated area, both of which were caused by fluoride tablets. The incidence of fluorosed incisors, the most visible of all teeth, was ten times greater in the fluoridated area than in the nonfluoridated area (9.62% versus 0.95%).

As would be expected, a larger proportion of cases in the fluoridated area involved more teeth per child than in the nonfluoridated area. The author notes: "In the fluoridated area, 50% of all cases involved only 2 or 4 teeth, and 15% involved more than 10 teeth. In the nonfluoridated area, 83% of the cases involved 2 or 4 teeth, and 7% (two fluoride tablet takers) involved more than 10 teeth."

In discussing his findings, he observes: "Critics who claim that disfiguring tooth defects are uncommon have failed to explain why school staff and the author found so many 'uncommon' cases in the fluoridated area, but not in the nonfluoridated one. When similar socio-economic groups are compared, "the prevalence of decayed, missing and filled teeth of 12 and 13-year-old children is now lower in the nonfluoridated part of the Auckland Health District than in the fluoridated part." Likewise, "the percentage of caries-free children is higher in the nonfluoridated areas when the socio-economic variable is taken into account."

The author concludes: "Current dental health statistics [in other countries as well as in New Zealand] indicate that declines in caries rates are now occurring in nonfluoridated communities to about the same extent as in fluoridated ones. Thus earlier predictions of the dental benefits of fluoridation appear to be considerably exaggerated, and the possible damage, especially from dental fluorosis, greatly underestimated."

References


**********
TRIAL OF MAGNESIUM COMPOUNDS IN THE PREVENTION OF SKELETAL FLUOROSIS - AN EXPERIMENTAL STUDY

by

D. Raja Reddy, K. Lahiri, N.V. Ram Mohan Rao, H.S. Vedanayakam
L.N. Ebenezer, and Suguna Ram Mohan
Hyderabad, India

SUMMARY: Magnesium compounds are beneficial to some extent because they lessen the amount of fluoride retained in bones. However they fail to prevent the development of fluorosis in animals or in humans in endemic regions. For this reason, we are searching for better compounds or methods to prevent and treat cases of fluorosis.

KEY WORDS: Magnesium compounds; Skeletal fluorosis

Introduction

In recent years magnesium compounds, namely serpentine and its active ingredients magnesium oxide and magnesium hydroxide, have been found useful in the treatment of skeletal fluorosis (1-4). Consequently, the current study was intended to determine whether prophylactic use of such compounds would prevent the development of fluorosis in rabbits.

Material and Methods

Twenty-two young rabbits, the mean weight of which was about 1 Kg with 1:1 male-female sex ratio, were grouped in four batches (Fig. 1).

<table>
<thead>
<tr>
<th>Group Designation</th>
<th>Group</th>
<th>No. of Animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Control</td>
<td>4</td>
</tr>
<tr>
<td>B</td>
<td>Fluoride</td>
<td>6</td>
</tr>
<tr>
<td>C</td>
<td>Fluoride + Mg (OH)₂</td>
<td>6</td>
</tr>
<tr>
<td>D</td>
<td>Fluoride + Serpentine</td>
<td>6</td>
</tr>
</tbody>
</table>

All were given standard diet of lucern grass, soaked bengal gram and water ad libitum. The amount of material which was force fed is shown in Figure 2. The amounts of Mg(OH)₂ and serpentine given to the rabbits were calculated weight-wise to correspond to the maximum dosage of these drugs administered to humans suffering from fluorosis. Fluoride intake was 22 mg daily, a large dose for rabbits and regarded as a maximum safe limit for humans. The experiment was terminated at the end of 70 days because in groups B and C, 3 animals died and the surviving animals, especially of group C, were sick owing to diarrhea which was bloody and they failed to take nourishment. Group B animals died due to fluoride toxicity, whereas those of group C died due to gastrointestinal upset, mainly caused by the material

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used for enteric coating of these granules. Group A and D animals were healthy and actually gained weight, which meant that serpentine protected them from fluoride toxicity. Obviously even the Mg(OH)$_2$ group would have survived had they not contracted diarrhea.

**Figure 2**

**Force-Fed Material**

<table>
<thead>
<tr>
<th>Group</th>
<th>Force-Fed</th>
<th>Daily Dose Per Animal</th>
<th>Concentration and Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>NaF</td>
<td>0.5 ml</td>
<td>4.4% NaF solution (Ridell De Hon)</td>
</tr>
<tr>
<td>C</td>
<td>(1) NaF</td>
<td>0.5</td>
<td>4.4% NaF solution granules containing 2.5 g of Mg(OH)$_2$ I.P./ea. 4.0 g. (Enteric-coated with shellac and stearic acid)</td>
</tr>
<tr>
<td></td>
<td>and</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mg(OH)$_2$ Granules</td>
<td>200 mg</td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>(1) NaF</td>
<td>0.5 ml</td>
<td>4.4% solution</td>
</tr>
<tr>
<td></td>
<td>and</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Serpentine</td>
<td>0.5 ml</td>
<td>0.4% Serpentine (mesh size 200 of the mineral containing principally Mg hydroxy metasilicate). Suspension in water.</td>
</tr>
</tbody>
</table>

Since 96-99% of fluoride retained in the body combines with bones, four types of studies were conducted on bones exhumed after sacrificing the animals, namely (1) histology, (2)'x'-ray diffraction, (3) fluoride and (4) strength (Fig. 3). After decalcification, two bones of animals were studied histologically. One powdered bone of each animal was studied by x-ray diffraction which reveals the spatial arrangement of atoms of the crystal lattice and detects the presence of elements which may sometimes be intractable to chemical methods. The fluoride content of bones was estimated on similar bones of each animal because it varies in individual bones of the same animal. Fat free dried bones were powdered and ashed after heating them overnight at 550°; their fluoride content was estimated by Orion ion specific electrode. To study bone strength Instron, a universal testing machine, used for studying compression and tensile strength of alloys, was used (Fig. 4). Two vertebrae of each animal were subjected to compressive stresses and the weight, at which fracture occurred, was recorded (Graph 1). Similarly tensile strength was studied after applying force to the ends of the fixed lengths of shafts of long bones till they cracked. To ensure that the ends of the bones did not crack due to the grip of the instrument, the ends were filled with alloy (Fig. 5). This test, for obvious reasons, was not possible in the case of every specimen studied; the results, however, were similar to those of the compressive group, although they broke at higher pressures (Graph 2).
Results

Histological studies failed to reveal any difference in the morphological features of the bones. X-ray diffraction studies, likewise, failed to reveal any changes in the configuration of the fluoroapatite crystals in any of the groups (Graph III and IV).

According to the compression test (Fig. 6), the strength of bones of groups A, B, and D did not differ materially although in groups B and D they were uniformly strong with little standard deviation and variance.

C group bones were markedly weaker than the others which might have been due to the malnutrition suffered by these animals. The levels of fluoride in bones are shown in Table 1. As expected, the retention of fluoride is high in Mg(OH)₂ treated group owing to their intolerance of the granules. A lower retention of fluoride occurred in the serpentine and Mg(OH)₂ groups. Nevertheless, these levels are higher than those from drugs which are ineffective in preventing the development of fluorosis in rabbits given massive doses of fluoride. Our clinical experience in the treatment of human fluorosis has been similar. These drugs were without value in patients who have suffered from overwhelming fluoride intoxication.

Discussion

Although an experiment of this nature has many limitations, certain
conclusions can be drawn. For instance, these compounds lessen the amount of fluoride retained in bones by increasing excretion of fluoride in urine and feces. Whereas the exact mechanism of the action of magnesium compounds is unknown, the following two observations are pertinent. Magnesium has special affinity and interrelationship with fluoride. It combines with fluoride of the fluoroapatite crystal of bones (5,6) and the resultant compound, which is 65 times more soluble, is probably excreted from the body. Increased fluoride excretion may be due to increased alkalinity of urine which occurs during use of these drugs; increased alkalinity of urine in turn promotes fluoride elimination from the body.

The following additional observations add support to the beneficial role of magnesium compounds in fluorosis. In Prakasam and Nalgonda districts fluoride levels are high; climatic conditions, food habits and occupations of residents are similar with the following two differences: In Prakasam the

### Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>Sample No.</th>
<th>%</th>
<th>Sample No.</th>
<th>%</th>
<th>Sample No.</th>
<th>%</th>
<th>Sample No.</th>
<th>%</th>
<th>Sample No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>1</td>
<td>0.055</td>
<td>B</td>
<td>1</td>
<td>0.64</td>
<td></td>
<td>C</td>
<td>1</td>
<td>0.55</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>2</td>
<td>0.05</td>
<td>B</td>
<td>2</td>
<td>0.55</td>
<td></td>
<td>C</td>
<td>2</td>
<td>0.40</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>3</td>
<td>0.065</td>
<td>B</td>
<td>3</td>
<td>0.66</td>
<td></td>
<td>C</td>
<td>3</td>
<td>0.47</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>4</td>
<td>0.125</td>
<td>B</td>
<td>4</td>
<td>0.50</td>
<td></td>
<td>C</td>
<td>4</td>
<td>0.45</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>5</td>
<td>0.105</td>
<td>B</td>
<td>5</td>
<td>0.60</td>
<td></td>
<td>C</td>
<td>5</td>
<td>0.35</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>6</td>
<td>0.10</td>
<td>B</td>
<td>6</td>
<td>0.58</td>
<td></td>
<td>C</td>
<td>6</td>
<td>0.50</td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>0.076</td>
<td>0.588</td>
<td></td>
<td></td>
<td>0.470</td>
<td></td>
<td></td>
<td></td>
<td>0.441</td>
<td></td>
</tr>
<tr>
<td>Variance</td>
<td>0.0012</td>
<td>0.0035</td>
<td></td>
<td></td>
<td>0.0046</td>
<td></td>
<td></td>
<td></td>
<td>0.0032</td>
<td></td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>0.0346</td>
<td>0.0592</td>
<td></td>
<td></td>
<td>0.0678</td>
<td></td>
<td></td>
<td></td>
<td>0.0566</td>
<td></td>
</tr>
</tbody>
</table>
incidence of crippling skeletal fluorosis is lower (Tables 2 and 3 and personal observations of one of the authors, N.V.R.M. Rao). According to our observations, people living in endemic fluorosis regions seem to need more magnesium compounds than those of nonendemic areas with the result that less fluoride is retained in the skeleton. Magnesium levels play a beneficial role.

<table>
<thead>
<tr>
<th>Village</th>
<th>F(^-) Levels mg/litre</th>
<th>Alkalinity mg/litre</th>
<th>Hardness mg/litre</th>
<th>pH range</th>
<th>Incidence of dental fluorosis</th>
<th>Incidence of skeletal fluorosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Naibai</td>
<td>2.0 - 6.2</td>
<td>100 - 460</td>
<td>200 - 760</td>
<td>7.2-8.1</td>
<td>89.3%</td>
<td>6.8%</td>
</tr>
<tr>
<td>Yedavalli</td>
<td>4.4 - 7.5</td>
<td>350 - 504</td>
<td>270 - 450</td>
<td>7.1-7.9</td>
<td>93.0%</td>
<td>13.5%</td>
</tr>
<tr>
<td>Yellareddyguda</td>
<td>2.6 - 10.0</td>
<td>400 - 820</td>
<td>180 - 400</td>
<td>6.8-7.8</td>
<td>91.2%</td>
<td>14.6%</td>
</tr>
</tbody>
</table>

**Conclusion**

When magnesium is adequate in foodstuffs, its deficiency observed clinically is rarely noticed in nonendemic regions. Thus metabolic studies of magnesium compounds may prove rewarding in elucidating the relationship between magnesium and fluoride elements.

<table>
<thead>
<tr>
<th>Village</th>
<th>pH</th>
<th>Alkalinity mg/l</th>
<th>Hardness mg/l</th>
<th>Fluoride mg/l</th>
<th>Magnesium mg/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kammavari</td>
<td>8.4</td>
<td>604</td>
<td>220</td>
<td>5.0</td>
<td>27.7</td>
</tr>
<tr>
<td>Baswapuram</td>
<td>8.1</td>
<td>432</td>
<td>172</td>
<td>7.0</td>
<td>23.3</td>
</tr>
<tr>
<td>Umameheswara Puram</td>
<td>8.4</td>
<td>900</td>
<td>128</td>
<td>10.0</td>
<td>14.3</td>
</tr>
</tbody>
</table>

**Acknowledgement**

Gratitude is expressed for help in the present study to Dr. Bhale Rao, Mr. Krishna Das of Regional Research Laboratories; Messrs. Bose and Deshpande of Defense Metallurgical Research Laboratories and to Dr. Ramchander Rao of Biological Evans Ltd., Hyderabad.

**References**


**********

EVALUATION OF COMMUNITY FLUOROSIS INDEX FOR THE INDIAN POPULATION IN ENDEMIC FLUOROSIS AREAS

by

M. Rahmatulla, and A. Rajasekar
Madras, India

SUMMARY: Two communities in endemic and nonendemic areas of the Salem district in Tamilnadu State, South India, were studied according to Dean's community fluorosis index (CFI), the upper acceptable limit of which was set at 0.6. When this limit is exceeded in a community, the fluoride in water is considered a public health problem requiring defluoridation measures. Among other communities studied, one with 0.5 ppm F⁻ in local water revealed a CFI of 1.25 which apparently constitutes a public health problem. The authors disagree with the suggestion that the upper CFI limit in Indian communities be raised in the absence of extensive base line data on climatic conditions, dietary habits and nutritional status of Indian communities, particularly in nutritionally poor rural communities which may be more vulnerable to the toxic effects of fluoride.

KEY WORDS: Community fluorosis index; Dental Fluorosis; India

Introduction

Dental fluorosis or mottled enamel, unlike skeletal fluorosis, is considered by some to be mainly an esthetic problem. The magnitude of the disorder in endemic fluorosis areas is determined by Dean's community fluorosis index (CFI) (1). In the present study an attempt has been made to evaluate Dean's CFI in Indian communities in both endemic and nonendemic fluorosis areas.

From Kilpauk Medical College and Stanley Medical College, Madras. Presented at the 13th conference of the International Society for Fluoride Research, Nov. 14-17, 1983, New Delhi, India.
Dental fluorosis or mottled enamel is the clinical manifestation of excessive fluoride ingestion during the first twelve years of life when the permanent, human dentition undergoes calcification. Fejerskov, et al. (2) have reviewed the literature on the possible pathogenic mechanism in the disorder. The minimum threshold value at which perceptible changes appear in the developing enamel of permanent teeth is well below 1.0 ppm for people in the U.S.A. living in a temperature zone (3). Although the degree of mottling may parallel the level of fluoride in water this relation is by no means consistent because the amount of water consumed and thus the quantity of fluoride ingested plays a role. When the average annual temperature is high, more water is consumed and hence more fluoride is ingested (4). In India humans have displayed signs of skeletal fluorosis from about 1 ppm fluoride naturally in water or less (5).

Thystrup (6) observed a progressive increase in severity from anterior to posterior teeth in areas where F⁻ in water is high. Where people are malnourished, dental fluorosis occurred at a lower F⁻ concentration (7). A calcium-deficient diet might enhance dental as well as skeletal fluorosis (8). According to Venkateswarlu et al. (9), 0.9 - 1 ppm in Indian drinking waters was associated with mottled enamel. This is about the level to which fluoride is being added to public water supplies in the USA. They have further reported that the incidence of caries and mottled enamel varies in communities consuming the same level of F⁻ in drinking water. According to their fluorosis index, the F⁻ content of water remaining the same, the incidence of caries varied inversely with the dental fluorosis index of the community. They further observed that nutritional and other factors, which lower the toxicity, increase the chances of dental caries. However, such factors, besides lowering toxicity, may also themselves effectively control caries incidence. Hence they have recognized the need to investigate other factors which contribute to low caries incidence.

Dean's classification of dental fluorosis (10,11), includes the following categories which range from normal to severe, depending upon the fluoride content in the local water supply.

1. Normal - enamel is translucent, smooth and has a glossy appearance.
2. Questionable - seen in endemic areas, borderline between normal and very mild.
3. Very mild - small opaque, paper white areas scattered irregularly over labial and buccal surface of teeth.
4. Mild - opaque area involving at least half of the teeth, brown stain present.
5. Moderate - generally entire tooth surface involved, minute pitting often present on labial and buccal surface, brown stains, frequently disfiguring.
6. Moderately severe - entire tooth surface involved, marked pitting, intense brown stain.
7. Severe - form of the teeth affected, widespread, deep brown or black areas, corrosion type of mottled enamel.

To determine the severity of dental fluorosis as a public health problem, Dean (12) devised a means to calculate the prevalence and degree of severity in a group or a community which he termed community fluoride index (CFI). Numerical statistical values were given to each degree of mottling namely, normal = 0; questionable = 0.5; very mild = 1; mild = 2; moderate = 3 and moderately severe and severe = 4. The CFI or community flu-
Orosis index is determined by the products of frequency and weight and divided by the number of persons examined. Further, when the fluorosis index was below 0.4, Dean (1) considered it of little or no public health concern, indices between 0.4 to 0.6 were borderline; above 0.6 removal of excess fluoride is indicated, hence a CFI of 0.6 is the limit of fluoride concentration. Whereas Dean set the borderline limit for dental fluorosis in midwestern USA in the vicinity of 1 to 1.5 ppm, studies in Japan, Kyoto district, revealed the corresponding border limit as 0.9 to 1.1 ppm, which is lower. The reason for the difference may be due in part to larger intake of fluoride-rich seafood by Japanese (13). Minoguchi (13) who considers optimum F- concentration a function of CFI and annual mean temperature, has computed the permissible 'F' concentration and the limit of 'F' concentration at each annual and mean temperature.

Materials and Methods

Two hundred and nine persons of both sexes ranging between the ages of 12 and 75 were surveyed in four villages of Salem district, State of Tamilnadu, South India. Fluoride in well-water ranged from 0.3 to 6 ppm. Sengatoor and Nirmalakottai were high fluoride villages with 6 ppm and 3.2 ppm F- in water respectively; Reddiampettai and Sengapuram, low fluoride communities, with 0.5 ppm and 0.3 ppm respectively. Only erupted permanent teeth were examined, in daylight with mouth mirror and explorer. Table 1 contains the details of the findings.

<table>
<thead>
<tr>
<th>Village</th>
<th>Between 0-1</th>
<th>Between 1-2</th>
<th>Between 2-3</th>
<th>Between 3 &amp; above</th>
<th>Total cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>F</td>
<td>M</td>
<td>F</td>
<td>M</td>
</tr>
<tr>
<td>Sengatoor 6 ppm</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Nirmalakottai 3.2 ppm</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Reddiampettai 0.5 ppm</td>
<td>23</td>
<td>18</td>
<td>13</td>
<td>8</td>
<td>18</td>
</tr>
<tr>
<td>Sengapuram 0.3 ppm</td>
<td>27</td>
<td>20</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

For assessing the CFI, Dean's classification of dental fluorosis was adopted (10,11). Lesions ranged from normal, questionable, very mild, mild, moderate, moderately severe to severe. To each degree of dental fluorosis numerical values (weight) were assigned as normal, questionable 0.5, very mild, mild 2, moderate 3 and moderately severe and severe 4. The CFI was determined by product of frequency and weight and its division by the number of persons examined.

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Example of CFI calculation: 100 cases, if 50 have a score of 2, 30 have 1.0 and 20 have 3, the CFI would be 50 x 2 + 30 x 1 + 20 x 3 \[ \text{CFI} = \frac{50 \times 2 + 30 \times 1 + 20 \times 3}{100} \].

The CFI of persons in different communities was determined based on Dean's formula. Each individual score was recorded in the particular community separately. The CFI among males and females was also assessed separately.

**Results**

Data collected from high fluoride communities and control (low fluoride) communities reveal that the community fluorosis index (CFI) is higher in study areas compared to control. The CFI is above 2.4 in communities where the local water supply contains 6 ppm and 3.2 ppm; it increased in both sexes in the study communities compared to controls (Table 2).

**Table 2**

<table>
<thead>
<tr>
<th>Village</th>
<th>F⁻ ppm</th>
<th>CFI M</th>
<th>CFI F</th>
<th>Average CFI</th>
</tr>
</thead>
<tbody>
<tr>
<td>First group:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sengatoor (study village)</td>
<td>6.0</td>
<td>2.44</td>
<td>2.48</td>
<td>2.41</td>
</tr>
<tr>
<td>Sengapuram (control village)</td>
<td>0.3</td>
<td>0.107</td>
<td>0</td>
<td>0.053</td>
</tr>
<tr>
<td>Second group:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nirmalaikottai (study village)</td>
<td>3.2</td>
<td>2.21</td>
<td>2.66</td>
<td>2.435</td>
</tr>
<tr>
<td>Reddiampettai (control village)</td>
<td>0.5</td>
<td>1.34</td>
<td>1.15</td>
<td>1.245</td>
</tr>
</tbody>
</table>

**Discussion**

The data obtained in the study generally reveal the CFI of both the study and control communities run parallel with the F⁻ content in the local water supply. Although the higher CFI in endemic communities is understandable, nevertheless a control community with 0.5 ppm F⁻ reveals CFI as high as 1.25. This shows that 0.5 ppm F⁻, although considered a safe upper limit in communal water supplies by western standards, nevertheless it can produce a community fluorosis index of about 0.6 in Indian villages.

Why does even 0.5 ppm produce CFI higher than 0.6 established as the upper limit? Fluoride ingestion during the first 12 years or precisely during the first six months to 7 years influences the degree of dental fluorosis. The safe F⁻ concentration in water for Indian communities is

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therefore not only dependent upon the mean annual temperature on which Galagan (14) based his formula but also the quantity of F\textsuperscript{-} intake during the first seven years of a child's life when most of the esthetically important teeth in human dentition undergo calcification. The determination of the safe F\textsuperscript{-} concentration in Indian communities requires a thorough analysis of the population for F\textsuperscript{-} content. This is mandatory in computing the acceptable level of fluoride in communities with annual mean temperature above 70° F as cautioned by Minoguchi (13) particularly among the rural Indian communities which are predominantly undernourished.

It was suggested (15) that the CFI in Indian subjects should be raised to 1 instead of 0.6 established by Dean. No convincing arguments have been advanced for raising the upper limit. A high fluorosis index is the sign of excessive F\textsuperscript{-} intake. It is also well known that nutritionally poor communities are more vulnerable to the toxic effect of fluoride. Hence, raising the limit would increase fluoride intake which is likely to induce a rise in the incidence of dental fluorosis among the poorer segment in Indian communities. Pending extensive epidemiological studies and dietary analysis in endemic fluorosis areas in India, the CFI limit of 0.6 should be retained for Indian subjects.

The observation that CFI is consistently higher in females in endemic communities may reflect high fluoride ingestion by females possibly because women folk remaining at home partake of 'Poidu' or cold rice (cooked in water with high fluoride sea salt, a popular readily available food item in Indian villages.

The most commonly affected teeth observed were maxillary incisors which is in agreement with the study by Thylstrup et al. (6). Another interesting observation, the reason for which is not clear, is the difference in severity of mottling displayed by members of the same family although they consume water from the same source.

Evaluation of the CFI in the present study is part of a larger study conducted on the dental health status of Indian communities in high and low fluoride areas by the authors (16). The present attempt to evaluate the CFI in endemic fluorosis areas in the Indian population is a preliminary step toward initiating another study along these lines.

**Conclusion**

No valid conclusion can be drawn on the existing CFI upper limit until extensive data from various centers are available taking cognizance of Indian climatic conditions, dietary habits and the nutritional status of the Indian population. It is hoped that additional studies will be undertaken along these lines in other centers in India.

**References**


EFFECT OF FLUORIDE ON TOTAL LIPID, CHOLESTEROL AND TRIGLYCERIDE LEVELS IN LIVER OF ALBINO RABBITS

by

J.P. Singh*, Shashi*, and S.P. Thapar**
Patiala, India

SUMMARY: White albino rabbits received subcutaneous injections containing 0, 5 and 10 mg NaF/kg body weight in distilled water for three and one-half months and were sacrificed. The liver was analyzed for total lipids, cholesterol and triglycerides. The decrease in the concentration of total lipids and cholesterol in the livers of rabbits consuming fluoride in water was proportional to the increase in fluoride intake. The decrease in liver cholesterol and triglyceride content was statistically significant in 10 mg group.

KEY WORDS: Rabbits, lipid levels; $F^-$ effect on liver; Cholesterol; Triglycerides; Rabbit liver

Introduction

A high intake of fluoride results in various biochemical changes including alterations in the lipid metabolism. In experiments with rats, lipid metabolism was altered by fluoride (1). Many investigators have shown that fluoride influences serum-lipid metabolism in rabbits. According to Bacon (2), increased fluoride intake initially increases and then decreases serum cholesterol concentration whereas, according to Leipzig et al. (3), increased fluoride intake decreases the concentration of serum triglycerides but fails to influence serum cholesterol. However, Townsend and Singer (4) report an increase in serum triglycerides in guinea pigs fed fluoride. They also reported that fluoride failed to alter liver cholesterol. Therefore, the effect of administration of varying concentrations of sodium fluoride on various biochemical parameters namely, total lipids, cholesterol and triglycerides was undertaken.

Material and Methods

Fifteen male white albino rabbits, obtained from Kaila Scientific Corporation, Agra, weighing 450-600 gm each, were used in the present studies. All animals were provided the same diet consisting of cabbage, palak, gram seeds soaked in water, and green grass. The animals were divided into three groups of five animals each. Group A, given no sodium fluoride, served as control. Groups B and C were given sodium fluoride (NaF) injections, 5 mg/kg and 10 mg/kg, for three and one-half months respectively. All animals were weighed at the beginning of the experiment and sacrificed under general anesthesia. The liver was taken out and preserved in chloroform:methanol (2:1 v/v) for the extraction of lipids. Parts of the liver weighing 100-300 mg were homogenized in a glass homog-
Enzimer with chloroform:methanol (2:1 v/v) according to the method of Folch et al. (5). Each homogenate was shaken periodically for an hour at room temperature and refrigerated overnight. Total lipids were determined gravimetrically. The estimation of cholesterol was carried out according to Stadtman (6). The triglycerides were estimated by the colorimetric method of VanHandel and Zilversmit (7) using tripalmitin as standard.

All reagents were of analytic grade. Differences between means of treated and control groups were calculated and statistical significance determined by student's 't' test.

**Results**

Table 1 shows the effect of NaF on total lipids of the liver. In groups A, B, and C, total lipids were 72.11, 41.81 and 36.05 mg/g respectively. In groups A, B, and C the cholesterol content in liver was 3.76, 2.50 and 2.17 mg/g respectively; however, differences were statistically significant in Group C at the level of P < .01 (Table 2).

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Effect of F⁻ on Total Lipids in Liver of Male Rabbit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>F⁻ Concentration</td>
</tr>
<tr>
<td>---------</td>
<td>-------------------</td>
</tr>
<tr>
<td>A</td>
<td>Control</td>
</tr>
<tr>
<td>B</td>
<td>5 mg/kg body wt.</td>
</tr>
<tr>
<td>C</td>
<td>10 mg/kg body wt.</td>
</tr>
</tbody>
</table>

The liver triglyceride content, after F⁻ administration, decreased significantly (Table 3). In groups A, B, and C the triglyceride concentrations were 18.73, 17.94 and 10.32 mg/g. The difference in triglyceride concentration in Group C was also significant at the P < 0.001 level.

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Effect of F⁻ on Triglyceride Content in Liver of Male Rabbit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>F⁻ Concentration</td>
</tr>
<tr>
<td>---------</td>
<td>-------------------</td>
</tr>
<tr>
<td>A</td>
<td>Control</td>
</tr>
<tr>
<td>B</td>
<td>5 mg/kg body weight</td>
</tr>
<tr>
<td>C</td>
<td>10 mg/kg body weight</td>
</tr>
</tbody>
</table>

*P < 0.001; P-value calculated applying student's 't' test of significance.

**Discussion**

The level of fluoride intake has a pronounced effect on the total lipids in the liver of rabbits. The amount of total lipids decreased in the livers of experimental rabbits compared to controls, in contrast to the findings of Vatassery et al. (8) who reported that concentrations of total lipids of liver as well as that of serum increases in the high fluoride group. Wolinsky, et al. (9) found no significant differences in the level of total lipids in the liver of control and experimental animals.
The concentration of liver cholesterol was not significantly different in Groups A, B, and C. Townsend and Singer (4) reported that fluoride intake fails to influence the amount of liver cholesterol in guinea pigs.

Fluoride also affects the metabolism of triglycerides. The level of triglycerides decreased significantly in fluoridated rabbits compared to controls. If fluoride inhibits an intestinal lipase, as reported, it may also inhibit a lipase involved in the removal of triglyceride from liver (10). Inhibition of lipase may account for the decrease in liver triglycerides in Groups B and C. Liver assimilates plasma triglycerides without prior hydrolysis (11,12). Townsend and Singer (4) record an increase in serum triglyceride in guinea pigs fed high fluoride.

Conclusion

The decrease, observed in the liver concentrations of the triglycerides in fluoridated animals, may be due to inhibition by fluoride in the uptake of triglycerides from the blood plasma.

Acknowledgement

All authors express thanks to Dr. S.S. Dhillon, Head, Department of Zoology, for providing laboratory facilities and one of the authors to the Indian Council of Medical Research, New Delhi for financial support.

References


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EPIDEMIOLOGICAL STUDY OF ENDEMIC FLUOROSIS IN THE VILLAGE KHERA IN PUNJAB

by


Patiala, India

SUMMARY: The present study is based on epidemiological data on 50 cases (30 males and 20 females) of endemic fluorosis. Epidemiological data, which were collected, pertain to duration of residence in the endemic area which leads to fluorosis, age of onset of pathological condition, literacy, socio-economic and nutritional status and clinical symptomatology. Detailed clinical examinations were undertaken which pertain to the incidence of the various symptoms of skeletal fluorosis namely, dental mottling, joint pains, stiffness of joints and pain in back, difficulty in rising from a squatting position, weakness of limbs, crippling causing the patient to be bedridden and loss of sensory perception.

KEY WORDS: Epidemiological; Fluoride; Pathological; Symptomatology

Introduction

The term, fluoride, was derived from its characteristic property of acting as a flux. Numerous authors have contributed significant information pertaining to the presence of fluoride in various body parts and its toxicity (1-11).

Bartolucci (12) noted a disease resembling osteomalacia in cattle grazing near a superphosphate factory which he attributed to excess intake of fluoride being thrown out by that factory; he named this disease "Fluorosis."

Fluorosis, an endemic disease, has been reported by many workers (13-16). Shortt et al. (17) recorded chronic fluoride intoxication for the

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first time from India. Subsequently many studies have emanated from different parts of India on different aspects of fluorosis, namely, Pandit (10,11) and Pillai (18) from Madras; Daver (19) from Hyderabad; Majumdar (20) from U.P.; Venkateswarlu (21) from Vishakhapatnam; and Kashiiwal & Solomon (22) from Rajasthan.

Singh et al. (23), Jolly et al. (24-25) and Makhni et al. (26-28) reported data from Punjab on the fluoride content (in water) in various endemic areas and its effect on different parts of the human body. The present paper reports epidemiological data pertaining to the duration of residence in a fluorotic area which led to fluorosis, age of onset, literacy, socio-economic and nutritional status, and clinical symptomatology.

Material and Methods

The present study was conducted in Village Khera, Tehsil Mansa Dist. Bhatinda, State of Punjab, which is located in the endemic fluorosis belt. This village is comprised of 164 families with 1122 individuals (611 males and 511 females) who reside in 156 houses. Their main source of drinking water is wells. The fluoride content of the subsoil water is 9.95 ppm. In this area, temperatures range from 46° C in summer to 8° C in winter. The inhabitants of this area consume 3 to 8 litres of water per day during summer depending upon age, sex and nature of their work.

A sample of 50 skeletal fluorosis patients (30 males, 20 females) showed unequivocal clinical evidence of skeletal fluorosis. The data were collected by the random sampling method with special emphasis on economic, social, educational and nutritional status. Detailed information regarding family history, duration of residence and source of drinking water was recorded. During careful clinical examination, particular attention was paid to pigmentation of teeth, joint pains, stiffness and pain in back, difficulty in rising from a squatting position. Dental mottling has been classified according to Kawahara (29).

### Table 1

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-20</td>
<td>2</td>
<td>6.67</td>
</tr>
<tr>
<td>21-30</td>
<td>1</td>
<td>3.33</td>
</tr>
<tr>
<td>31-40</td>
<td>12</td>
<td>4.00</td>
</tr>
<tr>
<td>41-50</td>
<td>5</td>
<td>16.67</td>
</tr>
<tr>
<td>51-60</td>
<td>6</td>
<td>20.00</td>
</tr>
<tr>
<td>61-70</td>
<td>3</td>
<td>10.00</td>
</tr>
<tr>
<td>71-80</td>
<td>1</td>
<td>3.33</td>
</tr>
<tr>
<td>Above 80</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Total</th>
<th>30</th>
<th>100.00</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>20</td>
<td>100.00</td>
</tr>
</tbody>
</table>

Age indicates years of residence in Khera Village

Observations and Discussion

1. Duration of residence in endemic area: Based on 50 cases (30 males, 20 females) of fluorosis, Table 1 shows the distribution of fluorotic cases according to age and sex. In females, age refers to the years of residence in the endemic fluorosis area of the village Khera. The mean, minimum and maximum duration of residence of the diseased cases (Table 2), were 40, 12, and 73 years respectively. The mean number of years of residence was 46 for males and 20 for females. All

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males included in the present study have been residing in this particular village since birth. Duration of residence of females was determined according to the day they migrated into this village. Eighteen years minimum was required for males and 12 for females to develop fluorosis symptoms (9.95 ppm F⁻ in water) (Table 2). Thus females were adversely affected by fluoride earlier than males.

2. Socio-economic status (Table 3):

a) Educational Status: 73.33% of the fluorotic males and 95.00% of the fluorotic females were illiterate. Details of male education status are presented in Table 3-A.

b) Social Classification: Social status grouping (Table 3-B) is based on Census Survey of the Government of India (1962). Respecting the social status of male cases, 80% were in Class 4, 13.33% in Class 3 and 6.67% in Class 2. No one was in Class 1. Whereas the social status of females has not been analyzed, no female was an independent resident.

Table 3
Socio-economic Status in Fluorosis Cases

<table>
<thead>
<tr>
<th>Group</th>
<th>Educational Status (A)</th>
<th>Post-Matric</th>
<th>Social Classification (B)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Illiterate</td>
<td>Primary</td>
<td>Middle</td>
</tr>
<tr>
<td>Males</td>
<td>no.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>22</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>73.33</td>
<td>6.67</td>
<td>6.67</td>
</tr>
<tr>
<td>Females</td>
<td>no.</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Nature of Work: Except for four crippled or partially crippled cases, all males (86.67%) were engaged in manual labor (Table 3-C), most of them in farming (73.33%), a few in technical work (6.67%), 3.33% were masons and 3.33% shopkeepers. All females were housewives. In addition to housework, females also assist their husbands in farming. The economic status of the residents of this village is below that of an average Punjab village.

d) Type of Houses: Table 3-D indicates that most of the diseased subjects are residing in Kacha houses (made of mud) (46.67% of the males and 80% of the females), whereas the percentage of males living in semi-pucca (partially mud and partially brick) or pucca houses (cemented bricks) is higher compared to females.

3. Nutritional Status: According to Table 4; 36.6% males and 60.00% females are vegetarian, the rest occasionally partake of a non-vegetarian...
diet. For the details of eating eggs and mutton (non-vegetarian diet) see Table 4. On the average, a resident of this village consumes 2500-2800 Kilocalories of food per day; 40-50 gms of protein; 300-500 gms of carbohydrates and 30-55 gms of fats. The source of these dietary ingredients is mainly from a vegetarian diet (Table 4).

Table 4

<table>
<thead>
<tr>
<th>Group</th>
<th>Vegetarian</th>
<th>Non-Vegetarian</th>
<th>Eggs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Daily</td>
</tr>
<tr>
<td>Males no. 11</td>
<td>19</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>%</td>
<td>36.67</td>
<td>63.33</td>
<td>10.00</td>
</tr>
<tr>
<td>Females no. 12</td>
<td>8</td>
<td>-</td>
<td>5</td>
</tr>
<tr>
<td>%</td>
<td>60.00</td>
<td>40.00</td>
<td>5.00</td>
</tr>
</tbody>
</table>

4. Symptomatology: More than one symptom has been found in each patient (Table 5). Joint pains, back stiffness and pain, and difficulty in rising from squatting position were found in 92%, 84% 44%, and 30% respectively. Stiffness of back was maximum in the morning.

Table 5

<table>
<thead>
<tr>
<th>No.</th>
<th>Symptoms</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Joint pains</td>
<td>46</td>
<td>92.00</td>
</tr>
<tr>
<td>2.</td>
<td>Pigmentation of teeth</td>
<td>45</td>
<td>90.00</td>
</tr>
<tr>
<td>3.</td>
<td>Stiffness of back</td>
<td>42</td>
<td>84.00</td>
</tr>
<tr>
<td>4.</td>
<td>Back pain</td>
<td>22</td>
<td>44.00</td>
</tr>
<tr>
<td>5.</td>
<td>Difficulty in rising from squatting position</td>
<td>15</td>
<td>30.00</td>
</tr>
<tr>
<td>6.</td>
<td>Weakness of lower limbs</td>
<td>3</td>
<td>6.00</td>
</tr>
<tr>
<td>7.</td>
<td>Weakness of upper limbs</td>
<td>1</td>
<td>2.00</td>
</tr>
<tr>
<td>8.</td>
<td>Foot pains</td>
<td>1</td>
<td>2.00</td>
</tr>
<tr>
<td>9.</td>
<td>Weakness of both upper and lower limbs</td>
<td>1</td>
<td>2.00</td>
</tr>
<tr>
<td>10.</td>
<td>Completely bedridden</td>
<td>3</td>
<td>6.00</td>
</tr>
<tr>
<td>11.</td>
<td>Loss of sensation</td>
<td>1</td>
<td>2.00</td>
</tr>
</tbody>
</table>

Dental mottling was observed in 90% of the cases (Table 8). The average duration of residence in the fluorosis area for the appear-

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Table 6
Incidence of Various Symptoms According to Sex
Related to Duration of Residence in Fluorotic Area

<table>
<thead>
<tr>
<th>No.</th>
<th>Complaints</th>
<th>Sex</th>
<th>Cases with Symptoms</th>
<th>Average years of residence in fluorotic area</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>M</td>
<td>No.</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Joint pains</td>
<td></td>
<td>27</td>
<td>90.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td>F</td>
<td>19</td>
<td>95.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Total: 46</td>
<td>92.00</td>
</tr>
<tr>
<td>2</td>
<td>Stiffness of back</td>
<td>M</td>
<td>24</td>
<td>80.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td>F</td>
<td>18</td>
<td>90.00</td>
</tr>
<tr>
<td></td>
<td></td>
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<td>84.00</td>
</tr>
<tr>
<td>3</td>
<td>Back pain</td>
<td>M</td>
<td>16</td>
<td>53.33</td>
</tr>
<tr>
<td></td>
<td></td>
<td>F</td>
<td>6</td>
<td>30.00</td>
</tr>
<tr>
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<td></td>
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</tr>
<tr>
<td>4</td>
<td>Difficulty in getting up</td>
<td>M</td>
<td>10</td>
<td>33.33</td>
</tr>
<tr>
<td></td>
<td></td>
<td>F</td>
<td>5</td>
<td>25.00</td>
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<tr>
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</tr>
<tr>
<td>5</td>
<td>Crippled (Bedridden)</td>
<td>M</td>
<td>3</td>
<td>10.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td>F</td>
<td></td>
<td></td>
</tr>
<tr>
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<td></td>
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</tr>
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<td>Loss of Sense perception</td>
<td>M</td>
<td>1</td>
<td>3.33</td>
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<tr>
<td></td>
<td></td>
<td>F</td>
<td></td>
<td></td>
</tr>
<tr>
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<td></td>
<td>Total: 1</td>
<td>2.00</td>
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</tbody>
</table>

ance of dental pigmentation was 15 years in males and 12.30 years in females according to the Kawahara (29) classification of dental mottling. Table 9 presents the age distribution at which pigmentation of teeth starts. The distribution of the various degrees of dental mottling and the presence of one or more degree of dental mottling has been presented in Tables 10 and 11.

Tables 7, 8, 9, 10, and 11 are on the following pages.

Conclusion

In this study, of a limited but typical population from Khera village, much the same characteristic pattern with respect to endemic fluorosis is seen as has been observed in numerous other fluorotic regions of India. The onset of skeletal fluorotic symptoms is sex-related: The average exposure time for adverse effects in females is approximately ten years less than that for males. The severity of skeletal fluorotic symptoms ranged from joint pains to total disability (crippled, bedridden); the more severe effects predominated in males.

FLUORIDE
<table>
<thead>
<tr>
<th>S. No.</th>
<th>Complaints</th>
<th>Sex</th>
<th>Age Groups (Years)</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td>0 - 15</td>
</tr>
<tr>
<td>1.</td>
<td>Joint pains</td>
<td>M</td>
<td>- 0.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td>F</td>
<td>7 35.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T</td>
<td>7 14.00</td>
</tr>
<tr>
<td>2.</td>
<td>Stiffness of back</td>
<td>M</td>
<td>- 0.00</td>
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<td></td>
<td></td>
<td>F</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>T</td>
<td>5 10.00</td>
</tr>
<tr>
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<td>Back pain</td>
<td>M</td>
<td>- - -</td>
</tr>
<tr>
<td></td>
<td></td>
<td>F</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>T</td>
<td>2 4.00</td>
</tr>
<tr>
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<td>M</td>
<td>- - -</td>
</tr>
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<td></td>
<td></td>
<td>F</td>
<td>- - -</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T</td>
<td>- - -</td>
</tr>
<tr>
<td>5.</td>
<td>Crippled</td>
<td>M</td>
<td>- - -</td>
</tr>
<tr>
<td></td>
<td></td>
<td>F</td>
<td>- - -</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T</td>
<td>- - -</td>
</tr>
<tr>
<td>6.</td>
<td>Loss of senses</td>
<td>M</td>
<td>- - -</td>
</tr>
<tr>
<td></td>
<td></td>
<td>F</td>
<td>- - -</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T</td>
<td>- - -</td>
</tr>
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</table>
Table 8

Distribution of Dental Mottling Cases

<table>
<thead>
<tr>
<th>Group</th>
<th>Pigmentation of Teeth</th>
<th>Average Age (Yrs.) of onset of dental pigmentation</th>
</tr>
</thead>
<tbody>
<tr>
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<td>%</td>
</tr>
<tr>
<td>Males</td>
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<td>86.67</td>
</tr>
<tr>
<td>Females</td>
<td>19</td>
<td>95.00</td>
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<tr>
<td>Total</td>
<td>45</td>
<td>90.00</td>
</tr>
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</table>

Table 9

Distribution of Dental Pigmentation in Relation to Residence

<table>
<thead>
<tr>
<th>Group</th>
<th>Cases</th>
<th>Duration of Residence (Years)</th>
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</thead>
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<td>no.</td>
<td>0 - 5</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>no. %</td>
</tr>
<tr>
<td>Males</td>
<td>26</td>
<td>86.67</td>
</tr>
<tr>
<td>Females</td>
<td>19</td>
<td>95.00</td>
</tr>
<tr>
<td>Total</td>
<td>45</td>
<td>90.00</td>
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</table>

Table 10

Distribution of Various Degrees of Dental Mottling

<table>
<thead>
<tr>
<th>Group</th>
<th>Streaks</th>
<th>Porcelain</th>
<th>Brown</th>
<th>Defects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S-System</td>
<td>P-System</td>
<td>B-System</td>
<td>D-System</td>
</tr>
<tr>
<td></td>
<td>no.</td>
<td>%</td>
<td>no.</td>
<td>%</td>
</tr>
<tr>
<td>Males</td>
<td>-</td>
<td>-</td>
<td>6</td>
<td>20.00</td>
</tr>
<tr>
<td>Females</td>
<td>3</td>
<td>15.00</td>
<td>6</td>
<td>30.00</td>
</tr>
<tr>
<td>Total</td>
<td>3</td>
<td>6.00</td>
<td>12</td>
<td>24.00</td>
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</table>

Table 11

Additional Degrees of Dental Mottling

<table>
<thead>
<tr>
<th>Group</th>
<th>P &amp; B System</th>
<th>B &amp; D System</th>
<th>S, P &amp; D System</th>
<th>P, B &amp; D System</th>
<th>Fallen teeth</th>
</tr>
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<td></td>
<td>no. %</td>
<td>no. %</td>
<td>no. %</td>
<td>no. %</td>
<td>no. %</td>
</tr>
<tr>
<td>Males</td>
<td>1 3.33</td>
<td>16 53.33</td>
<td>-</td>
<td>5 16.67</td>
<td>4 13.33</td>
</tr>
<tr>
<td>Females</td>
<td>2 10.00</td>
<td>10 50.00</td>
<td>1 5.00</td>
<td>3 15.00</td>
<td>1 5.00</td>
</tr>
<tr>
<td>Total</td>
<td>3 6.00</td>
<td>26 52.00</td>
<td>1 2.00</td>
<td>8 16.00</td>
<td>5 10.00</td>
</tr>
</tbody>
</table>

FLUORIDE
References


***********
EFFECTS OF VENTILATORY CONDITIONS ON DEPOSITION OF
NaF IN ISOLATED RABBIT LUNGS

by

S. Ichinohe, M. Mita, and H. Tsunoda
Morioka, Japan

SUMMARY: The effects of tidal volume and respiratory frequency on the deposition of NaF aerosol particles were studied using lungs isolated from 20 rabbits. Total lung deposition decreased with increases in tidal volume and respiratory flow rate and was in the range of 20-80% when the reciprocal of the respiratory flow rate was 0.05-0.5 sec/ml. Deposition of aerosol particles in various lung lobes had a 1:1 ratio with respect to the weight of each lobe. The deposition was especially high on the right middle lobe and markedly decreased with increases in respiratory flow rate.

KEY WORDS: Lung deposition; NaF aerosol particle; Ventilatory conditions

Introduction

The toxicity of fluoride in the air of certain factories has been a health concern partly because it can cause systematic injury by absorption following inhalation. Whereas the absorption rate of fluoride through the respiratory tract has been studied for gaseous HF (1), information is unavailable concerning the particulate form of this pollutant.

Using rabbits and NaF aerosol, in previous inhalation experiments we estimated, from urinary fluoride excretion, the NaF absorption rate from the respiratory tract (2). Since, in the discussion of particle inhalation, not only its absorption, but also its deposition is of concern, the mechanism by which the particles are deposited becomes important. It is known that particle size affects the total and regional deposition of particles in the respiratory tract (3). Since the influence of tidal volume (TV) and respiratory frequency (f) on the deposition of particles has not been studied extensively, the present study was initiated to clarify this aspect by using generated NaF aerosol and lungs isolated from rabbits.

Material and Methods

Twenty rabbits were used in this study (average body weight 2.75 ± 0.30 g). The weight of the lungs ranged from 7.2 to 12.3 (average 9.9±1.4g).

The apparatus, depicted in Figures 1-a and 1-b, consisted of a NaF aerosol generator, inhalation chamber and artificial thorax. The mist, formed from 0.3 M NaF solution by means of an ultrasonic nebulizer, was

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blown to the mixing chamber by the carrier air, where it was mixed with dilution air heated to 140°C with a mixing ratio of 1.24 (Figure 1a). This process produced dry solid NaF aerosol particles. The NaF aerosol concentration inhaled was maintained at approximately 10 mgF/m³ and averaged 0.3 µm in particle diameter. The concentrations of the aerosol were monitored by a calibrated digital counter (SHIBATA KAGAKU: Type P-5). These levels were kept within 5% coefficient of variation.

The inhalation chamber was made of acrylics with a capacity of 10 liters (Fig. 1b). A constant amount of NaF aerosol, ca. 6.0 l/min was passed into the chamber with laminar flow. The chamber was maintained at 28°C with a humidity of 40%.

**Figure 1-a**
NaF Aerosol Generating System

A: NaF mist generating chamber; B: mixing chamber; C: ultrasonic nebulizer; D: heater; a: thermometer

**Figure 1-b**
NaF Aerosol Inhalation System

A: inhalation chamber; B: artificial thorax; C: numerical-controlled oscillator; D: pressure adjustor; a: particle counter; b: flow transducer; c: pressure transducer; d: hygrothermometer

The artificial thorax was connected with a numerical-controlled oscillator. Made of acrylics with a capacity of 1.5 liters, it was used to hold the lungs. A brass duct was placed in the trachea of the isolated lungs as a tracheal adaptor and was attached to the center of the top of the artificial thorax. The lower part of the artificial thorax was filled with water up to about 2 cm deep. The humidity of the artificial thorax was maintained at approximately 100% and a temperature of 30°C. Lung ventilation was carried out by numerical-controlled oscillator (4). TV was measured by a flow transducer (Shibata Kagaku: Type ISA-8) at the entrance of a tracheal adaptor.

Following the inhalation study, the lungs were divided according to individual lobes and the fluoride deposition determined in each of them. Each of the lobes was weighed and homogenized with distilled water. The extract was centrifuged at 1000 xg for 15 min. To the resulting supernatant was added an equal amount of TISAB, and the fluoride content was determined with an ion selective electrode (Orion Research: Model 96-09). The lung fluoride deposition was calculated by dividing the fluoride content in the lung by the amount of fluoride inhaled. Inhaled fluoride was computed by multiply-
ing the fluoride concentration in the inhalation chamber by the f, TV and inhalation time.

The validity of the method for determination of fluoride in lung tissues was established by a study on recovery of fluoride from spiked samples. For this purpose, 12 lung tissues, each weighing about 1 g, were used. A 2 ml solution, containing 0.5, 1, and 2 ppm F, respectively, was added to the tissue sample; the mixture was homogenized, and the homogenate, centrifuged. The fluoride content of the supernatant was determined by the method described previously. The percentage recovery from these experiments ranged from 92.0 to 106.0%, with an overall average of 98.3±4.2%.

Results

Several mechanical factors, for each of the rabbits used in this work, were studied. The total lung capacity (TLC), static lung compliance (Cst), and pulmonary resistance (Rp) were determined. Average values, for the 20 rabbits, follow: TLC, 95.4 16.5 ml; Cst, 13.0 3.5 ml/cm H2O; Rp, 0.019 ± 0.007 cm H2O/ml/sec.

The TLC and Cst were measured from the volume-pressure curve, and the Rp was obtained by the oscillation method. When TLC was 95.4±16.5 ml and the Cst 13.0±3.5 ml/cm H2O, the Rp was 0.019±0.007 cm H2O/ml/sec. Little variation was observed in these values which confirms earlier reports concerning mechanical factors of pulmonary ventilation (5).

The TV, f, V, reciprocal of respiratory flow rate (1/V), ventilation time (T), fluoride concentration in the air (Fair), amounts of fluoride inhaled (Finh), amounts of fluoride deposited in the lungs (Fdep), and total lung deposition (D) are shown in Table 1.

<table>
<thead>
<tr>
<th>No.</th>
<th>f (Hz)</th>
<th>TV (m³)</th>
<th>T (sec/min)</th>
<th>Fair (µg/mL)</th>
<th>Finh (µg)</th>
<th>Fdep (µg)</th>
<th>D (µg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.25</td>
<td>4.3</td>
<td>2.4</td>
<td>0.62</td>
<td>20</td>
<td>10.1</td>
<td>13.0</td>
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<tr>
<td>2</td>
<td>0.25</td>
<td>6.7</td>
<td>3.9</td>
<td>0.26</td>
<td>30</td>
<td>11.1</td>
<td>33.5</td>
</tr>
<tr>
<td>3</td>
<td>0.25</td>
<td>3.3</td>
<td>2.0</td>
<td>0.50</td>
<td>20</td>
<td>10.8</td>
<td>10.7</td>
</tr>
<tr>
<td>4</td>
<td>0.25</td>
<td>9.9</td>
<td>5.1</td>
<td>0.20</td>
<td>30</td>
<td>11.3</td>
<td>30.3</td>
</tr>
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<td>5</td>
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<td>8.7</td>
<td>5.8</td>
<td>0.17</td>
<td>30</td>
<td>9.9</td>
<td>38.8</td>
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<td>6</td>
<td>0.5</td>
<td>8.0</td>
<td>9.3</td>
<td>0.11</td>
<td>30</td>
<td>12.8</td>
<td>92.2</td>
</tr>
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<td>75.6</td>
</tr>
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<td>10</td>
<td>12.2</td>
<td>16.5</td>
</tr>
<tr>
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<td>0.5</td>
<td>7.9</td>
<td>9.1</td>
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<td>20</td>
<td>11.8</td>
<td>55.9</td>
</tr>
<tr>
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<td>11.8</td>
<td>56.2</td>
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<td>10.7</td>
<td>53.4</td>
</tr>
<tr>
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<td>2.0</td>
<td>2.1</td>
<td>9.7</td>
<td>0.10</td>
<td>12</td>
<td>12.0</td>
<td>36.3</td>
</tr>
<tr>
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<td>4.7</td>
<td>22.2</td>
<td>0.065</td>
<td>15</td>
<td>10.6</td>
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</tr>
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<td>2.0</td>
<td>7.6</td>
<td>34.2</td>
<td>0.029</td>
<td>15</td>
<td>11.3</td>
<td>194.6</td>
</tr>
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</table>

f = respiratory frequency; TV = tidal volume; T = respiratory flow rate; 1/V = reciprocal of respiratory flow rate; T = ventilation time; Fair = fluoride in air; Finh = fluoride inhaled; Fdep = fluoride deposited in lung; D = total lung deposition.
The relationship between total lung deposition (D) and TV to f frequency is shown in Figure 2. D tended to increase with decreases in TV and f. The V is the product of f and TV in sinusoidal ventilation. To examine the relationship between V, and D, I/V and D were used to plot Figure 3. A linear relationship existed between the two variables (r= 0.803 and n=20) and was significant at the 99% level. D was in the range of 20-80% when I/V was 0.05-0.5 sec/ml (Figure 3).

![Figure 2](image1)

**Figure 2**
Relationship Between Tidal Volume and Total Lung Deposition

![Figure 3](image2)

**Figure 3**
Relationship Between Reciprocal of Respiratory Flow Rate and Total Lung Deposition (D)

The distribution of deposited fluoride onto various lung lobes (Df) and distribution of various lung lobe weight (Dw) are shown in Table 2. Deposition of the fluoride in the lung lobes was in a 1:1 ratio with the weight of the lung lobes. However the right middle lobe received a disproportionate deposition. To examine the influence of V on the Fdep in each lobe, an adjusted distribution of fluoride deposited into various lung lobes was computed. This was done by dividing Df by Dw in each category in Table 2. The values obtained were averaged, and the results are

![Table 2](image3)

**Table 2**
Deposited Fluoride and Distribution of Lung Lobes Weight

<table>
<thead>
<tr>
<th>No.</th>
<th>R-u (%)</th>
<th>R-m (%)</th>
<th>R-l (%)</th>
<th>L-u (%)</th>
<th>L-l (%)</th>
<th>R-u (%)</th>
<th>R-m (%)</th>
<th>R-l (%)</th>
<th>L-u (%)</th>
<th>L-l (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AV.</td>
<td>8.9</td>
<td>14.5</td>
<td>37.2</td>
<td>10.5</td>
<td>29.4</td>
<td>8.5</td>
<td>11.4</td>
<td>39.9</td>
<td>10.2</td>
<td>30.0</td>
</tr>
<tr>
<td>S.D.</td>
<td>3.2</td>
<td>3.0</td>
<td>6.4</td>
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<td>3.4</td>
<td>0.8</td>
<td>1.2</td>
<td>2.2</td>
<td>1.3</td>
<td>1.8</td>
</tr>
</tbody>
</table>

R-u = right upper lobe; R-m = right middle lobe; R-l = right lower lobe; L-u = left upper lobe; L-l = left lower lobe; AV. = average; S.D. = standard deviation.
shown in Table 3. The ratio for the right middle lobe tended to decrease as \( \dot{V} \) increased and was more affected by \( \dot{V} \) than the other lobes.

<table>
<thead>
<tr>
<th>( \dot{V} ) (ml/sec)</th>
<th>( n = 4 )</th>
<th>5.0-9.9</th>
<th>10.0-19.9</th>
<th>20.0-30.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>R - u</td>
<td>1.10</td>
<td>0.93</td>
<td>1.27</td>
<td>0.80</td>
</tr>
<tr>
<td>R - m</td>
<td>1.45</td>
<td>1.29</td>
<td>1.25</td>
<td>1.05</td>
</tr>
<tr>
<td>R - l</td>
<td>0.81</td>
<td>1.04</td>
<td>0.99</td>
<td>0.92</td>
</tr>
<tr>
<td>L - u</td>
<td>0.83</td>
<td>1.04</td>
<td>1.03</td>
<td>1.30</td>
</tr>
<tr>
<td>L - l</td>
<td>0.82</td>
<td>0.82</td>
<td>0.86</td>
<td>1.06</td>
</tr>
</tbody>
</table>

These values were obtained as the Df/Dw ratio to compensate the deposited fluoride by each lung lobe weight. Df = distribution of deposited fluoride onto various lung lobes. Dw = distribution of various lung lobes weight. \( \dot{V} \) = respiratory flow rate; R-u = right upper lobe; R-m = right middle lobe; R-l = right lower lobe; L-u = left upper lobe; L-l = left lower lobe.

**Discussion**

The influence of \( f \) and \( TV \) on particle deposition in the respiratory tract has been investigated by several workers. Hatch and Gross (5) pointed out that increases in \( f \) would lower the respiratory tract deposition. Lippmann (6) attributed this to decreased residence time of particles with increasing \( f \). Kimura (7) suggested that increased \( TV \) enhanced the respiratory tract deposition. Lippmann, on the other hand, claimed that when \( TV \) was increased, particles penetrated deeper in the lung, resulting in increased particle residence time.

The results obtained in our study using sinusoidal ventilation demonstrated that increases both in \( TV \) and \( f \) lowered the D. The data on TD are in contrast to the report made by Kimura (7). Presumably this is due to the different experimental conditions used in our study. The physiological ventilatory conditions used by Kimura were different in its ventilation patterns from the sinusoidal ventilation employed in our study (8), and an increase in \( TV \) does not necessarily result in lowered residence time.

Heyder et al. (9) reviewed the regional deposition of aerosol particles in the human respiratory tract and demonstrated that increase in \( \dot{V} \) would lower the deposition in the respiratory tract. As seen in Figure 3, the results that D increased with increases in \( I/V \) agree with observations made by Heyder et al. (9). It is noted from these observations that the ventilatory conditions that influence deposition in the respiratory tract could be examined by using a parameter such as \( \dot{V} \).

As shown previously increases in \( \dot{V} \) caused a greater decrease of deposition in the right middle lobe than in other lobes, suggesting that the right middle lobe is different from other lobes in its response to changes in ventilatory pressure. This may be justified in view of the developmental and anatomical differences between the right lobe and other lobes of the lung.
It is concluded that $V$ is an important ventilatory condition parameter which influences particle deposition in the respiratory tract.

Acknowledgement

The authors are grateful to Dr. Ming-Ho Yu, Huxley College of Environmental Studies, Western Washington University, U.S.A. for helpful comments and suggestions in the preparation of this manuscript.

References


**********
ACCUMULATION OF FLUORIDE IN LEAVES OF SHRUBS IN THE VICINITY OF A FLUORINE INDUSTRY

by

K.S. Pillai, and U.H. Mane
Aurangabad City, India

SUMMARY: Deposition of fluoride on surface and accumulation in tissues of leaves of 4 species of shrubs, namely, Prospolis juliflora, Acacia nilotica, Calotropis procera, and Zizyphus nummularia in the vicinity of a fluorine industry was studied. The amount of fluoride deposited on the surface of C. procera leaves was significantly higher (P < 0.05) than on the leaves of the other 3 species. Deposition of fluoride was not significantly different (P > 0.05) between P. juliflora, A. nilotica and Z. nummularia. The four species varied significantly (P < 0.05) in the level of fluoride accumulation in leaf tissues.

KEY WORDS: Fluoride deposition; Leaf; Meteorological conditions; Shrubs

Introduction

In an area contaminated by industry, fluoride content in vegetation, particularly in leaf tissues, can increase abnormally. In beech leaves 2585 mg F/kg dry weight was observed in an industrial area of Germany (1). Gilbert (2) found high fluoride levels in the vegetation around an aluminium smelter in Scotland; Fischer (3) in leaves of beech (Fagus silvatica) in Austrian urban areas of heavy moderate pollution. Foliage fluoride levels around an aluminum smelter in Montana, USA, are available (4 – 6). Rao and Pal (7) reported high fluoride concentrations in forage and crop in the vicinity of an aluminum industry in Varanasi, India.

The aim of the present study is to determine the extent of fluoride accumulation on the surface as well as in the tissues of 4 commonly occurring species of shrubs in the vicinity of a fluorine industry situated at Surat, India. Fluoride content, measured in monthly samples (October 1978 – September 1979) was correlated with meteorological data namely, temperature, rainfall and wind speed.

Materials and Methods

Six plants of P. juliflora, A. nilotica, C. procera and Z. nummularia in the vicinity of a fluorine industry and South Gujarat University Campus, India (non-polluted area – about 12 km distant from the industry)

From the Department of Zoology, Marathwada University, City Aurangabad, India.
were selected. The leaves, collected in polythene bags were iced and brought to the laboratory. Leaf samples, always from the same plants, were collected monthly. The polluted leaves were separated, pooled species-wise and 3 samples of each species were processed to analyze the fluoride deposition on the surface and in the tissues of leaves. For analysis, only young and healthy leaves were considered. Fluoride deposition on the surface of the leaves was analyzed according to the Intersociety Committee on Methods for Ambient Air Sampling and Analysis (8). For this purpose leaves (100 - 200 g) were placed in a polythene beaker containing 0.05% Na₄ EDTA solution for one minute. The leaves were rewarshed in distilled water. The Na₄ EDTA solution and distilled water were mixed together and the fluoride content in it was measured using a fluoride electrode (Orion Research Inc., USA). The fluoride content in leaf tissue was measured according to Gordon et al. (9).

Fluoride levels of the 4 species were compared using Keuls multiple-range test (10).

**Results**

Data on temperature and rainfall (during September 1978 through August 1979) are presented in Figs. 1 and 2, respectively. Data on wind speed and direction (during September 1978 through August 1979) were compiled, frequencies of calm and wind speed of 1-8 km/h were debiased, and given as 'wind rose' (Fig. 3).

**Figure 1**

Monthly Fluctuation in Temperature at Surat from September 1978 to August 1979

All 4 species showed significantly higher (P < 0.01) fluoride levels on the surface of leaves, than the nonpolluted species (in nonpolluted plants, fluoride on the surface of leaves was below 1 ppm). Deposition of fluoride was significantly higher (P < 0.05) on the surface of leaves of *C. procera* than on the other 3 species. Fluoride deposition did not vary significantly (P > 0.05) among *P. juliflora*, *A. nilotica* and *Z. nummularia*. These 3 species showed a similar pattern of monthly fluctuation in fluoride deposition (Fig. 4). The fluoride content on the surface of these species declined in November after which it increased gradually till May. It again dropped in June and increased till September. In *C. procera*, the fluoride content dropped considerably in November; it increased
(during the study period fluoride content fluctuated in the range 4.2-7.2, 3.7-7.0, 4.2-6.6 and 3.4-7.0 ppm in P. juliflora, A. nilotica, C. procera and Z. nummularia respectively). Fluoride content in leaf tissues of Z. nummularia, A. nilotica and P. juliflora decreased until March and then increased until August. In September it again declined. In C. procera, fluoride content increased in November and dropped considerably until February. It increased in March and remained almost steady until May. During June to September, it again increased considerably.

Discussion

Airborne fluoride pollutants enter the plant primarily through the stomata, they pass into the intercellular spaces, are absorbed by the mesophyll tissues (11) and are retained in a water soluble inorganic form (12). It has been shown that inorganic particulate fluorides, which are soluble in water, can be absorbed directly through the leaves (13,14).

The range of dispersion of fluoride in the atmosphere is determined by meteorological conditions, chemical stability and physical state of fluoride (gas, aerosol, smoke or dust) (15). Deposition of particulate fluorides on the surface of leaves is correlated with meteorological conditions, parti-

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Mean values of 3 replicates; Vertical bars indicate standard deviation of

cularly rainfall. In this study, fluoride deposition on the surface of leaf
can be negatively correlated with rainfall (the correlation coefficient, γ =
-0.50, -0.56, -0.68 and -0.62 for P. juliflora, A. nilotica, C. procera and
Z. nummularia, respectively). In June, when the rainy season started after
long dry spell of summer, the fluoride content on the surface of leaves of a
species declined considerably, which indicates that rain washes away partic-
ulate fluoride deposited on the surface of leaves. Higher fluoride content du-
ing December through May can be explained on the basis of wind data. Durin
this period, the frequency of slow winds (1-8 km/h) and calms was very high.
It has been shown that slow wind concentrates particulate matter near the
source of discharge (16,17).

Deposition of higher levels of fluoride on the surface of leaves of C.
procera, compared to P. juliflora, A. nilotica and Z. nummularia, may be due
to the difference in leaf morphology. The larger leaves of C. procera can
receive and hold more particulate fluoride. Moreover, the leaves of C. pro-
cera, unlike the other 3 species, are heavier. The lighter leaves respond
to wind with a wave motion, thus they do not long retain particulate fluorid
on their surface.

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Fluoride accumulation in leaf tissue of the 4 species was comparatively less during winter which may be a reflection of low radiation during winter resulting in closed stomata. Since the primary entrance of gaseous pollutants is through the stomata (18,19) gaseous fluoride which enters through partially closed stomata during winter will be reduced. Increase in temperature may be the reason for higher levels of fluoride in tissues of leaves of the 4 species in summer. Studies conducted by MacLean and Schneider (20,21) revealed that fluoride accumulation in Helianthus annuus is directly proportional to the temperature. All 4 species under study showed increase in fluoride content in leaf-tissues during rainy months. In a humid atmosphere, stomata remain fully opened (22) which allows more fluoride to pass through the stomata from the atmosphere. Some of the particulate fluorides, deposited on the surface of leaves, can be dissolved in rainwater depending upon the solubility of particles (23). These water soluble fluorides might have penetrated into the leaf tissues of the 4 species, thus increasing their fluoride content during the rainy season. Rainwater itself can absorb fluoride from the polluted atmosphere and can increase the fluoride content in leaf tissues (24). Gordon et al. (9) reported abnormal fluoride levels, due to pollution, in coniferous trees of Montana during the rainy season.

Conclusion

The significant difference in fluoride content in leaf tissues between the species may be due to the variation in leaf morphology and physiology. Abnormal fluoride levels in these species confirm a raised fluoride level (both gaseous and particulate) in air, in the vicinity of industry. The fluoride levels found in and on vegetation would be damaging to animals.

Acknowledgement

We thank the late Dr. K.M. Desai, South Gujarat University for suggestions and Mr. Aniruddh Desai for preparing the figures. Dr. A. Thomson Mathai commented on the manuscript.

References


FLUORIDE


Abstract

EFFECTS OF DIETARY FLUORIDE ON THE HARD TISSUE FLUORIDE CONCENTRATION IN GROWING RAT

by

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Fluoride is deposited mainly in bones and teeth. The object of this investigation was to study the effect of dietary fluoride on hard tissue fluoride levels in growing rats.

Male Donryu rats, weighing 71-90 g each, were divided into 9 groups of 8 each. Five groups were fed diets containing 0, 10, 20, 40, 60 mg F/kg body weight/day. To check body gain, 4 pair groups received 10, 20, 40, 60 mgF/kg body weight/day; 15 g food per day and deionized water ad libitum was provided.

After four consecutive weeks of treatment, fluoride in blood plasma, femur, mandible and molar enamel were elevated in proportion to dietary fluoride. Fluoride levels of femur, mandible, incisor and molar enamel, were significantly related.

The results suggest that the fluoride dose is associated with hard tissue fluoride concentration and that Donryu rats are suitable for fluoride metabolism experiments.

KEY WORDS: Fluoride intoxication; Hard tissue; Rat

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**********

EXPERIMENTAL STUDIES ON ACUTE TOXICITY OF FLUORIDE COMPOUNDS

by

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(Abstracted from The Shikwa Gakuho, 83:1239-1242, 1983)

The acute toxicity of sodium fluoride (NaF), acidulated phosphate fluoride (APF), and/or sodium monofluorophosphate (MFP) - which are used as preventive drugs for dental caries - was investigated. Male (STD-ddy)*

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mice were used for acute toxicity tests after breeding for 7 days where room temperature and humidity were controlled. Animals were given water ad libitum and fed freely until 18 hours prior to the experiment.

NaF or MFP were dissolved in distilled water; APF was made by dissolving NaF in phosphoric acid; all solutions were 2% concentrations. The fluoride compound solutions were administered orally; toxic symptoms or the number of deaths were observed for one week. LD50 values were calculated according to the Van Der Waerden method.

The LD50 value and confidence limit of NaF, APF or MFP were 158 mg/kg (147 – 168 mg/kg), 152 mg/kg (141 – 164 mg/kg) or 555 mg/kg (481 – 640 mg/kg), respectively. Administration of these drugs caused excitement followed by clonic or tonic convulsions and death. Results show that the toxicity of NaF is almost the same as that of APF and that the toxicity of MFP is much lower than that of NaF or APF.

*Abbreviation for breeding laboratory mice.

KEY WORDS: Acidulated phosphate fluoride; Sodium fluoride; Sodium monofluoro phosphate; LD50; Mouse

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**********

FURTHER STUDIES ON THE EFFECTS OF FLUORIDES UPON LACTIC ACID PRODUCTION BY BACTERIA

by

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(Abstracted from Odontology, 71:30-39, 1983)

The inhibitory action of fluoride upon lactic acid production by Streptococcus salivarius ATCC 9758, S. lactis ATCC 7963, and S. faecalis ATCC 10541, was studied. Suspension of bacteria (0.5 ml), buffer solution (2.0 ml), 800 mg glucose/100 ml (0.5 ml) and solution of fluoride (1.0 ml) were mixed and incubated. Lactic acid was measured by the Barker-Summerson method.

When fluoride was not added, after 30 minutes 0.4-1.1 mg/100 ml lactic acid was produced. After 24 hours, 22.7-25.6 mg/100 ml lactic acid was detected. When 45 ppm NaF was added and incubated for 24 hours, inhibition was slight; 135 ppm NaF induced 72.7-51.2% inhibition, 450 ppm NaF caused practically complete inhibition of lactic acid production.

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45 ppm $\text{F}^-$ of acidulated phosphate fluoride (APF) failed to inhibit lactic acid production of Streptococcus salivarius, but that of Streptococcus lactis and Streptococcus faecalis was inhibited 29.8-46.8%. Inhibition was 78.8-96.8% by 135 ppm APF. The inhibitory effect of fluoride, which is affected by time and dosage, was stronger by APF than by NaF.

KEY WORDS: Lactic acid production; NaF and APF effect on enzyme inhibition

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**********

EFFECT OF LONG-TERM ADMINISTRATION OF FLUORIDE ON LEVELS OF EDTA-SOLUBLE PROTEIN AND $\gamma$-CARBOXYGLUTAMIC ACID IN RAT INCISOR TEETH

by

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(Abstracted from J. Dent. Res. 63:1061-1063, 1984)

EDTA-soluble material was extracted from incisor teeth of rats which received 50 ppm fluoride (NaF) in drinking water ad libitum from conception to age nine weeks. It contained significantly higher levels of protein ($P < 0.01$) and $\gamma$-carboxyglutamic acid ($P < 0.01$) than did similar material from control rats. The data indicate that mild fluorosis of rat incisor teeth involves not only characteristic disturbances of pigmentation and mineral structure but also alteration of the matrix protein.

Fluoride treatment significantly increased ($P < 0.001$) the fluoride content of the incisors: The fluoride concentration in the teeth, retained from the fluoride groups, was $854 \pm 123 \mu g/g$ (dry wt.) compared with $168 \pm 9 \mu g/g$ in controls (mean $\pm$ 2SE). As expected, the incisors also showed signs of enamel fluorosis. Instead of normal glossy orange-brown pigmentation of labial surfaces, they had a lackuster faint sandy color. Mean body weights of young rats of fluoride-treated litters ($171 \pm 9$ g, $n=45$) and of control litters ($173 \pm 12$ g, $n=39$) did not differ significantly.

KEY WORDS: Dental fluorosis, experimental; EDTA-soluble protein; Rat incisor teeth; $\gamma$-carboxyglutamic acid

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FLUORIDE
Abstract

CELL-FREE DESENSITIZATION OF CATECHOLAMINE-SENSITIVE
ADENYLATE CYCLASE

by

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(Abstracted from J. Biol. Chem., 259:4629-4633, 1984)

Conditions have been developed for desensitizing the β-adrenergic receptor-coupled adenylyl cyclase of turkey erythrocytes in a cell-free system. Desensitization is observed when cell lysates are incubated with isoproterenol or cAMP analogs for 30 min at 37° C. Maximally effective concentrations of isoproterenol produce a 41.0 ± 1.55% loss of isoproterenol-stimulated and a 15.0 ± 2.35% loss of fluoride-stimulated enzyme activity. cAMP causes a 26.5 ± 1.5% fall in isoproterenol-stimulated and a 21.5 ± 4.4% fall in fluoride-sensitive activity. Desensitization by isoproterenol is dose-dependent stereospecific, and blocked by the β-adrenergic antagonist propranolol. Cell-free desensitization required ATP, Mg2+, and factor(s) present in the soluble fraction of the cell. Nonphosphorylating analogs of ATP did not support desensitization. Desensitization by agonist or cAMP in the cell-free system caused structural alterations in the β-adrenergic receptor peptides apparent as an altered mobility of the photoaffinity labeled receptor peptides on sodium dodecyl sulfate-polyacrylamide gel electrophoresis. As with the desensitization reaction, supernatant factors and ATP were also required for the agonist or cAMP-promoted receptor alterations. These data indicate that β-adrenergic agonists promote a cAMP-mediated process which leads to receptor alterations and desensitization. The reactions involved in this process require ATP and soluble cellular factors. Additional processes must also occur to account for decreases in fluoride-sensitive enzyme activity. The availability of this cell-free system should facilitate elucidation of the molecular mechanisms involved in these processes.

KEY WORDS: β-adrenergic; cAMP desensitization; Fluoride-stimulated enzyme; Turkey erythrocytes

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**********

Authors' Abstract
Abstract

URINARY FLUORIDE CONCENTRATION AS AN ESTIMATOR OF WELDING FUME EXPOSURE FROM BASIC ELECTRODES

by

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The primary purpose of this study was to determine the relationship between the particulate fluoride concentration in air and the postshift urinary fluoride concentration and secondly, between the total concentration of welding fume particles and the postshift concentration of fluoride in the urine of welders using a specific electrode with a known composition of calcium fluoride. Should a strong relation exist, postshift urinary F⁻ could be used for biological monitoring of welding fume exposure.

In one study, in which total welding fume exposure and urinary fluoride levels were measured, 64 railway track welders whose mean age was 48 (28-59) all worked with the same basic electrode, the fumes of which contain 18-20% of fluoride; their welding exposure time ranged from 5 to 41 years (mean 18). Controls, 70 non-welding railway workers (ages 27-59; mean 47) who were residing in the same area as the railway track welders were occupied with electric and signal installation and track construction. Postshift urinary concentrations of fluoride were higher in the welders than in the non-welding controls. Age and urinary fluoride were not related in either welders or controls. The linear relationship between particulate air fluoride and urinary fluoride in welders in this study has not been described previously.

KEY WORDS: Basic welding electrodes; Particulate fluoride fumes; Urinary F⁻ in welders

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***********
Abstract

PREVALENCE OF DENTAL CARIES AND DENTAL FLUOROSIS IN AREAS WITH OPTIMAL AND ABOVE-OPTIMAL WATER FLUORIDE CONCENTRATIONS

by

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(Abstracted from JADA, 107:42-47, 1983)

This study assessed the prevalence and severity of dental fluorosis and the corresponding prevalence of dental caries in communities where the level of fluoride naturally in drinking water is at or above the recommended level. The overall mean age of the study population, 11.48 yrs, was similar in the four categories of communities. Distribution by sex, approximately 46% males and 54% females, also was similar among the four categories.

In almost 98% of the children drinking water at the recommended 0.7-1.2 ppm fluoride level, scores were 2 or less, or the extent of fluorosis did not exceed mild. Eight children, 2.4% of all children examined, in Kewanee (1.06 ppm) had either moderate or severe fluorosis.

The authors were surprised to find any advanced forms of fluorosis in an area where fluoride in water was within the recommended level. To learn why this occurred a detailed questionnaire, which covered such factors as erroneous residence history, prolonged absence from the community, use of water from sources other than the community supply, consumption of high-fluoride infant formulas, use of dietary fluoride supplements, and ingestion of unusual amounts of fluoride dentifrice, was developed. The parents of the eight children were queried in detail by phone. However, their responses to the questionnaire did not explain the clinical findings.

Fluorosis scores for children at 2 to 3 times the recommended fluoride levels were similar, primarily very mild, mild and moderate. Of particular significance is the increase in the proportion of children with severe fluorosis, defects which range from "isolated pits to multiple areas of confluent pitting that may change the shape of a tooth and cause it to appear corroded."

The additional caries protection from levels of fluoride higher than those recommended may diminish if the concentration is too high. According to a study by Ockerse, in South Africa in 1941, the percentage of children with dental caries was higher in an area where the water supply contained 6.8 ppm fluoride than in a comparable area where the fluoride concentration averaged about 2.5 ppm. A later study, by Forrest in England, showed that children residing in an area with 5.8 ppm fluoride in its water, caries experience was similar to that of children living where the fluoride level was near-optimal. Most of these reports explain a decrease in added protection as the result of severe fluorosis. Possibly, by destroying the protective enamel, affected teeth have become more susceptible to caries because food, debris, and plaque are entrap-
ped in the hypoplastic areas.

In several of the studies, individual caries experience was related to severity of fluorosis. Teeth of individuals with severe fluorosis had more dental caries than teeth of individuals that had less fluorosis.

KEY WORDS: Dental caries, Fluorosis, dental

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**********

IN VITRO AND IN VIVO STUDIES OF CELLULAR LYSIS OF ORAL BACTERIA BY A LYSOZYME–PROTEASE–INORGANIC MONOVALENT ANION ANTIBACTERIAL SYSTEM

by

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(Abstracted from Infection and Immunity, 45:610-617, 1984)

Compared with anion-activated cell lysis of oral bacteria damaged with either lysozyme or trypsin, cells which were treated with both of these enzymes showed a far greater degree of lysis, whether or not the turbidometric, DNA release, or electron microscopic assays were used to monitor the lytic process. At an acidic pH of 5.2 and an NaHCO₃ concentration of 100 mM, the kinetics of lysis for two different serotype c strains of Streptococcus mutans were similar. At 0 to 100 mM bicarbonate, however, differences in the lytic susceptibilities of the two strains were evident. At pH 5.2, NaHCO₃ but not NaSCN, NaCl, or NaF, was effective in promoting cell lysis of oral bacteria. At apparent sublytic concentrations of NaHCO₃, lysis was achieved by adding appropriate concentrations of NaSCN, NaCl, or NaF to the lysozyme–protease–damaged cells. In in vivo studies hamsters given a combination of NaHCO₃, NaCl, and NaSCN had significantly reduced levels of S. mutans on their molar teeth compared with that found in controls or animals exposed to any one of the salts alone or to a combination of chloride and thiocyanate only. The results suggest that bicarbonate is an essential anion which, together with the other major salivary inorganic monovalent anions, plays an active role in the lysis and ultimate elimination of cariogenic bacteria.

KEY WORDS: Anticaries role of anions; Bicarbonate and caries; Lysis of oral bacteria; Streptococcus mutans.

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Authors' Abstract
Abstract

CHANGING TRENDS IN DENTAL CARIES

by

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In underdeveloped countries the incidence of dental caries is increasing at a frightening rate whereas in industrialized countries the caries rate has declined by about 40% during the past 10 years. In 1982, for the first time ever, the average 12-year-old in underdeveloped countries had a higher dental caries score (decayed, missing, filled - DMF) than those in industrialized countries. Of about 1500 million children less than 15 years of age in the world today, 80% reside in underdeveloped countries.

The increase in caries is associated with increases in sugar consumption. By 1984, sugar consumption in underdeveloped countries is predicted to exceed that in industrialized countries. Whereas the decline in caries is associated with the widespread availability of fluoride toothpaste, with changes in the pattern and quantity of sugar consumption and, possibly, with the frequent use of antibiotics, no single factor has been found to account for the decline in most Western industrialized countries. Most likely a combination of factors is responsible. Dental decay is a sugar-dependent infective disease. Fluoride reduces enamel solubility in acid; it influences the remineralization of lesions as well as the metabolism of oral bacteria. Some authors believe that the main mechanism whereby fluoride acts in caries prevention is in promoting remineralization. Despite decreases, the prevalence of dental caries is still unacceptably high; in some countries, little or no decline has occurred.

Changes for the better, in the pattern of sucrose consumption reported from a number of countries, are the following: Increase in breast-feeding; reduction in the number of parents adding sugar to bottle feeds and in the use of sweetened comforters; changes in the composition of baby formulas and of processed baby foods; and a wider variety of sugar-free and low sugar foods. Reduced intake of sucrose by infants affects the number of cariogenic bacteria and the incidence of caries.

Evidence is not unequivocal that toothbrushing and good oral cleanliness reduces caries experience. The anti-bacterial action of some antibiotics and the increased caries resistance of teeth affected by tetracycline suggests that antibiotics may play an important unintended role in decreasing dental caries in children who are eating less sucrose and using fluoride toothpaste. The prevalence and severity of caries increased first in the upper income groups, then in the urbanized populations followed by changes in disease prevalence in rural groups. In Ethiopia, children from more affluent families had four times more caries in primary teeth, and twice as many in permanent teeth, as poorer children. In the Sudan, 15-19-year-old urban children had seven times more caries than children in rural areas where sugar consumption was below 5 lbs/person/yr.
Because urbanized populations in underdeveloped countries are more likely to consume refined sugar than those in rural areas, caries rates are higher in the former.

Industrialized countries are decreasing their imports of sugar from underdeveloped countries, many of which are sugar-producers, and are consuming four-fifths of what they produce whereas previously they exported four-fifths of their production. In addition to local production, in some underdeveloped countries sugar is the second largest imported food item.

Numerous studies have shown that, in populations where diet has changed from locally available agricultural products to manufactured and processed foods, particularly those containing sugar, dental caries has increased.

Wilska was first to find a strong positive relationship between per capita sugar consumption and caries rates. Buttner, who studied the relationship based on data from 19 countries, reached a similar conclusion. More recently, Sreebny found a highly significant relationship between availability of sugar and DMF in 47 countries. Narendran, who analyzed the relationship between sugar availability and caries in countries which had experienced increased dental caries, recorded a marked rise in caries in 15 or 20 countries where two surveys had been conducted on 12-year-olds. DMF increased from 2.8 to 6.3 in Jordan, from 1.2 to 3.6 in Lebanon, from 0.6 to 4.4 in Thailand, and from 4.7 to 9.8 in the Philippines.

The average annual per capita sugar consumption level increased in underdeveloped countries from 22.3 kg in 1968 to 27.4 kg in 1981. African countries had the largest increase, namely 39% in annual per capita consumption over that period. In Asia, the increase was 24%, in Latin American countries, 21%.

Takechi showed that where sugar consumption increased from 0.2 to 15 kg/person/year, the annual caries incidence rate was positively correlated with sugar consumption \((r = 0.8)\). The rate of attack increased at sugar levels about 10 kg/person/year. At consumption levels of 15-21 kg/person/year the intensity of the attack further increased. When annual levels of consumption are below 15 kg, most sugar consumed is visible sugar. Above that level, an increasing percentage of sugar consumed is in more cariogenic manufactured products such as soft drinks, sweets and biscuits.

In view of the trends in industrialized and underdeveloped countries, prevention aimed at controlling the availability of refined sugars and sugar-containing foods, drinks, and sweets is needed.

**KEY WORDS:** Dental caries, changing trends; Sugar and dental caries

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FLUORIDE
FLUORIDE IN SALIVA AFTER VARIOUS TOPICAL TREATMENTS

by

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The concentration and clearance of fluoride in saliva were studied in a test panel of four dental students after application of the following products: 2% sodium fluoride solution; Elmex solamine fluoride solution; Elmex gel sodium fluoride and amine fluoride gel; and Duraphat sodium fluoride varnish. Pretreatment control samples were taken by paraffin stimulation of salivary secretion. Subsequent post-treatment samples were taken 2, 4, 8, and 12 hrs. after the application of fluoride, before toothbrushing and breakfast the following morning, and on days 2, 4, 6, and 8 after treatment. Fluoride concentrations were determined using a fluoride electrode.

The Duraphat varnish gave higher peak fluoride values (mean F⁻ concentration 10 ppm, 2 hrs after treatment) and a longer period of clearance from the saliva than the other products. The amine fluorides gave F⁻ means of 3 ppm, 2 hrs. after treatment, whereas the sodium fluoride solution gave F⁻ means a little above 1 ppm, 2 hrs. after application. The amine fluorides and sodium fluoride solutions were cleared to below 1 ppm of F⁻ during the first few hours after treatment.

From a toxicological point of view, the application of 5 ml of gel may be undesirable. Individual trays should be used to minimize the amount of gel needed. Suction apparatus has been recommended during treatment and patients should expectorate after treatment. Three times higher enamel fluoride concentration has been reported after Elmex solution treatment than after sodium fluoride treatment. The enamel fluoride concentration, however, is not always directly comparable with caries susceptibility or resistance.

Unfortunately no long-term clinical evaluation of the caries-reducing potential of the products studied are available.

KEY WORDS: Fluoride; Saliva; Topical application

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THE SIGNIFICANCE OF REMINERALIZATION IN CARIES PREVENTION

by

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Remineralization which occurs naturally during the formation of a carious lesion in human enamel, is responsible for the appearance of two of four histological zones. Exposure of small lesions to a synthetic calcifying fluid in vitro results in a significant increase in remineralization.

The degree of mineralization is influenced by fluoride ions in the calcifying fluid, only low levels of which seem to be necessary to initiate the mechanism. A higher fluoride level fails to induce greater remineralization. The calcium ion concentration of the calcifying fluid, which appears to be critical with respect to which components of the fluid are supersaturated, determines the degree of remineralization. In both the dark and surface zone, crystal diameters are significantly larger than those in sound enamel, direct evidence that crystal growth is a result of remineralization.

Exposure of small artificial caries-like lesions to a calcifying fluid in vitro produced a significant degree of remineralization. After the experiment, lesions showed histological features which indicated that they were at a much earlier stage in development than prior to exposure to the calcifying fluid. Thus, creation of a lesion in human dental enamel is the result of a dynamic series of events, not a process of simple continuing demineralization.

In the present studies, two aspects of the remineralization process were investigated: first, the effect of fluoride ions on remineralization of enamel lesions of the teeth in vitro; second, the effect of remineralization on the submicroscopic crystalline structure of the lesion.

Artificial caries-like lesions were used in the experiments to provide a source of highly reproducible lesions with histological characteristics identical to those of enamel caries. Tooth halves were exposed to synthetic calcifying fluid; one tooth half was exposed to the experimental fluids, the adjacent half of the same tooth was exposed to identical fluid to which fluoride ions were added.

When synthetic calcifying fluid was employed, remineralization occurred; differences were due to different calcium levels of fluoride. In addition, the presence or absence of fluoride ions significantly affected the results.

When the 3 mM calcium calcifying fluid was used, without the addition of fluoride ions, mean reduction for 10 lesions in area of the body
of the lesion was 9%. Addition of fluoride ions caused a 24% mean reduction in porosity. When the calcifying fluid contained only 1 mM calcium, the apatitic phases were supersaturated; remineralization occurred through the entire depth of the lesion.

It is significant that comparable degrees of remineralization occurred when either six-minute or one-hour exposure increments were employed, indicating that six minute exposure is sufficient to initiate the remineralization mechanism. The "number" of exposure increments employed appear to be more important than "time" duration of exposure to calcifying fluid.

This is the only study which has recorded that all crystals within two of the four zones of the enamel lesion are significantly larger than those of sound enamel. In addition, after remineralization of the lesion, in vitro, it has been possible to increase significantly crystal diameters throughout the entire lesion. Therefore, in a "caries-free" patient, many interproximal enamel regions could have small lesions which are maintained at their histological sub-clinical size by continued remineralization.

Recent clinical studies have supported the frequent supply of low concentrations of fluoride rather than the infrequent use of high concentrations. The maximum degree of remineralization occurred with a "low" fluoride concentration of 0.5 mM (1 ppm) available at the enamel surface. A ten-fold increase in the fluoride level failed to affect the degree of remineralization. Since it can take three or four weeks for a smooth surface lesion to invade the dentin, there is adequate time to intercept the carious process.

These studies have important implications in terms of understanding the caries prevention mechanism and the development of new preventive agents.

KEY WORDS: Calcium in remineralization; Caries prevention; Enamel remineralization; fluoride in remineralization

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