CENTO SCIENTIFIC PROGRAMME

# SEMINAR ON

# "PROBLEMS OF HIGH FLUORIDE WATERS"

held in
Atatürk University,
Erzurum - Turkey

6 - 10 ~~September~~, 1977
June

Report No. 28

Technical Editor : A. Sevinç Kışlalı
Typesetting : Carol Çıpa

CONTENTS

# FOREWORD

Pollution or the environment is a subject of great topical interest and is associated in most people's minds with the rise of modern Technology.

Dangerous pollution to the general population can and does occur in quite natural ways. Spring water supplies can contain toxic materials such as fluorides picked up along the flow of the water, both above and below ground. In the neighbourhood of volcanic regions, water supplies with a high fluoride content, having ill-effects to man-kind are known to occur.

Because it was understood that the problem of a dangerously high fluoride-content in water supplies existed in several places in the CENTO region, the Council for Scientific Education and Research of CENTO organised this meeting to study the problem. The meeting was held at Atatürk University, Erzurum, where scientific,medical and dental studies into the occurrence and effects on humans and animals of high fluoride concentrations in natural drinking water supplies, were underway at a particular site, the village of Doğubayazıt.

The programme of the meeting is summarised below.

The Scientific Secretariat of CENTO must express its gratitude to the President of Atatürk University Prof. Dr. Hurşit Ertuğrul for allowing us to use the facilities of the University, and for his generous hospitality, and also to Prof. Dr. Nazmi Oruç, the host organiser and his colleagues for their help in the organisation of the meeting and the subsequent field trips.

Dr. Ray W.H. Wright
CENTO Scientific Secretary
Tehran - Iran

# PROGRAMME

MONDAY ~~16~~ 6-10 June 1977 Opening Ceremony

Morning: Inauguration
by Prof.Dr. Hurşit Ertuğrul
President of Atatürk University

Introduction to Technical Session
by Dr. R.W.H. Wright
CENTO Scientific Secretary

Introductiory Paper
by Prof. Dr. Nazmi Oruç
Host Chairman

Afternoon: General papers

TUESDAY 07 June 1977

Morning: Papers on Medical and Dental Problems

Afternoon: Defluoridation Methods

WEDNESDAY & THURSDAY 08 & 09 June 1977

Field Observation trip to Doğubayazıt

PARTICIPANTS

IRAN

Dr. Gholam Reza Gholamhosseiny, Azarbaydjan Univ. Tabriz

Prof.Dr. K. Simjour, Faculty of Dentistry, Univ. of Tehran

TURKEY

Hasan Akyar, D.S.I. İnşaat Mühendisi, Ankara

Sıtkı Aksoğan, Y.S.E. Plânlama Daire Başkanı, Ankara

Prof. Dr. Ayhan Aksoy, Atatürk Univ. Ziraat Fak. Erzurum

Mustafa Aktürk, Y.S.E. İçme Suları Daire Başkanı Ankara

Diş Hekimi Cemil Arabacıer, Sağlık Bakanlığı Diş Hekimliği Şube Müdürü, Ankara

Erol Babacan, Fırat Univ. Veteriner Fak., Elazığ

A. Tezer Bal, Kimya Mühendisi, Aluminyum Tesisleri Seydişehir, Konya

Doç.Dr. Aysel Bayraktar, Ege Univ. Ziraat Fak. İzmir

Doç.Dr. Nihat Baysu, Veteriner Fak. Ankara Univ. Ankara

Doç.Dr. Ahmet Çakır, Ziraat Fak. Atatürk Univ. Erzurum

Mehmet Çelikbilek, Uzman Veteriner Hekim, Yarbay 16. No. Gıda Kontrol, Erzurum

TURKEY(continued)

Namık Cimilli, Y.S.E. Genel Müdür Başyardımcısı Ankara

Tekin Çoruh, D.S.I VIII. Bölge Müdürlüğü, Erzurum

Prof.Dr. Sevim Devrim, T.B.T.A.K. Tıp. Grubu, Ankara

İsmail Girgin, Fen Fak. Ankara University, Ankara

Türkân Giritlioğlu, Y.Kimya Mühendisi, İller Bankası Ankara

Kerim G. ~~Göknil~~ Gülsün, D.S.I. Drenaj Başmühendisi, Ankara

Prof.Dr. Turgut Gündüz, Fen Fak. Ankara Univ. Ankara

Prof.Dr. Burhan Kaçar, Ziraat Fak. Ankara Univ. Ankara

Adile Nurşen İpekoğlu, I.T.Ü. Kimya Fak. Istanbul

Oya Karabağ, D.S.I. Yüksek Kimya Mühendisi, Ankara

I. Hakkı Karamanderisi, Jeolog M.T.A. Ankara

Prof.Dr. Halit Keskin, Kimya Fak. Istanbul Univ. Istanbul

Erol Kural, Kimya Mühendisi, Azot Sanayii, Samsun

Prof.Dr. Rüknettin Öğütman, Atatürk Univ. Tıp Fak. Erzurum

Prof.Dr. Cengiz Oktay, Atatürk Univ. Diş Hekimliği Fak. Erzurum

Tahir Öngür, Y. Jeoloji Mühendisi, M.T.A. Ankara

Prof. Dr. Nazmi Oruç, Ziraat Fak. Atatürk Univ. Erzurum

TURKEY(continued)

Dr. Esen Özalp, Diş Hekimliği Fak. Istanbul Univ. Istanbul

Doç.Dr. Mustafa Özcan, Fen Fak. Atatürk Univ.Erzurum

Doç.Dr. Mehmet Öztopçular, Tıp Fak. Atatürk Univ. Erzurum

Işıl Salihoğlu, D.S.I. Çevre Yüksek Mühendisi Ankara

Doç.Dr. Çetinkaya Şendil, Veteriner Fak.Ankara Univ. Ankara

İbrahim Siner, D.S.I. Y.Kimya Mühendisi, Ankara

Fikret Tekin, Y. Kimya Mühendisi, Refik Saydam Ankara

Halûk Toprakoğlu, D.S.I. Drenaj Fen Heyeti Müdürü, Ankara

Selma Türkoğlu, Y. Kimya Mühendisi, Refik Saydam, Ankara

Prof.Dr. Mithat Torunoğlu, Tıp Fak. Ankara Univ. Ankara

Prof.Dr. Fahrettin Tosun, Ziraat Fak. Atatürk Univ. Erzurum

Şükran Yalçın, Jeolog, İller Bankası, Ankara

Cemil Yunmuş, Kimya Mühendisi, Azot Sanayii, T.A.Ş. Samsun

UNITED KINGDOM

Dr. John R. Reid, Senior Medical Officer, Department of Health and Social Security, London

UNITED KINGDOM(continued)

Dr. J.B.D. Robinson, Long Ashton Research Station, University of Bristol

UNITED STATES OF AMERICA

Dr. Donald R. Taves, Univ. of Rochester, School of Medicine and Dentistry, Rochester, New York

CENTO

Mr. S. Douglas Martin, Deputy Secretary General CENTO Ankara

Mr. Clyde Adams, Senior Agricultural Advisor, CENTO Ankara

Dr. Ray W. H. Wright, Scientific Secretary, CENTO Tehran

Dr. S.M. Jafar, Deputy Scientific Secretary, CENTO Tehran

# THE EFFECTS OF FLUORIDES ON HUMAN HEALTH
# (A REVIEW)

by Dr. John R. Reid
Senior Medical Officer
Dept. of Health and
Social Security
Hannibal House
Elephant and Castle
London

## THE TOXICITY OF FLUORIDES

### (A) ACUTE

Acute poisoning by both organic and inorganic fluoride compounds has been extensively documented in various countries. Most recorded cases have been the result of accidental poisoning but some have been deliberate - attempts at suicide. The usual occurrence is sodium fluoride being in the wrong place, for example, in the home and being mistaken for other household compounds such as sugar, salt or baking powder. There are instances in institutions where mass poisoning has occurred from similar mistakes of fluoride compounds (Lidbeck et al 1943). Certain rodenticides which used to contain fluoride have also been implicated in outbreaks of acute poisoning. One case recorded in the literature has been the result of the ingestion of laundry powder (Yolken et al 1976). However it is recognised that acute poisoning or intoxication is rare as only 435 cases have been recorded up to 1970 (WHO 1970) and as mentioned the mass institution outbreak accounted for 236 cases of this total in a single episode at the Oregon State Hospital (Lidbeck et al 1943).

It is generally accepted that the fatal dose of sodium fluoride for adults is about 5 gms. As with many other substances there is a wide range on the one hand recovery following the ingestion of large doses as high as 50 - 80 gms (Alexander 1967) and on the other hand dosages as low as 2 gms have been fatal. Indeed some authorities consider a dose as low as 200 mgms is dangerous.

The symptoms of acute poisoning have been well documented from observations on cases of acute poisoning and from animal studies where acute intoxication has been induced by administering fluoride compounds by different routes. From these studies post mortem recording and histology the effects have been classified. In the initial stages of acute poisoning there is congestion and irritation of the mucosal surfaces causing abdominal pain, diarrhoea and vomitting. Depending on treatment, the vehicle of transmission, route of administration and the solubility of the fluoride compound will decide what further symptoms develop both in degree and rapidity. These further symptoms consist of excessive thirst, salivation and excessive lacrymal duct secretion. Cramps or painful spasms of the limbs then develop possibly due to dehydration and lowering of blood calcium levels. There is depression of the vasomotor centre with lowered blood pressure and respiratory paralysis.

The above description of acute poisoning is included for the sake of completeness as it is not a common occurrence. It should be noted that more cases may be seen in the future due to the greater use of topical preparations and fluoride tablets which could possibly get within the reach of children.

(B) CHRONIC

The manifestation of chronic fluoride intoxication will depend on the rate of ingestion, the duration of exposure, and the age of the subject. It is not intended that this paper should deal with dental fluorosis.

The chronic exposure of fluorides can be broadly divided into the effects of small doses of fluoride and into the effects of larger doses of fluoride both over a long duration of exposure. Indeed as in many other fields of study in trace elements is there an intermediate stage between a subclinical effect on the enzyme systems which in some ill-defined manner can lead to vague clinical effects and the level at which overt clinical effects are observed ? This type of problem has emerged in the study of lead poisoning and does subclinical lead poisoning exist. Like lead the fluorides have an affinity to hard tissues such as bone and teeth.

They are also accumulative over a life time of exposure. Unlike lead there is no doubt that fluorine is benefical up to certain levels. The classification of fluorine as an essential or non-essential element depends on the criteria employed in determining essentiality (Underwood 1973). An essential element has been defined as one that must be present in the diet to permit survival. This cannot be truly said of fluorine in our present state of knowledge but care should be exercised in this statement as efforts to produce animals without fluorine in the bone have so far failed. That being so it may well be an essential element although the exact role is yet to be defined.

The effects of chronic exposure to fluorides on the various systems in the body have now to be reviewed.

1. <u>Chronic Toxic Effects On The Skeleton</u>

The effects of fluorides on the skeletal system in humans has been extensively studied in areas of high natural fluoride levels in water supplies and by many different means. There is a wide geographical distribution of these areas involving provinces of India, Pakistan, Sri Lanka, China, Japan, Saudi Arabia, U.S.A., Canada and certain parts of Europe. In the United Kingdom there are several aquifiers that have higher than normal levels mainly in the Hartlepool area and small areas of Essex in the South of England. Most of these supplies are either not used now or are diluted by water from other sources so that levels in the UK are such that there are no dental or skeletal problems.

The level of fluoride intake below which no retention takes place must be very low even if such a situation exists. In metabolic studies with rats using 18F it would appear that there is no level at which complete elimination occurs (Wallace - Durbin 1954). Long term studies in rats indicate that there is a continuous retention of fluoride in bones even at the lowest dietary levels of fluoride that can be employed (Smith et al 1952). The bone fluoride is roughly proportional to the water fluoride due both to (1) exchange at the mineral surface and (2) incorporation of fluoride in newly formed bone (Murray 1976). The critical levels in bone at which fluorosis develops has not been so well estimated in humans as it has in cattle. A daily intake of fluoride at 2 ppm in the diet over 50 years results in chemical fluorosis only; that is with not bone mottling in other abnormalities but even at this level other factors have to be considered such as climate, nutritional status, age and possible individual variations of absorption or sensitivity. From the study of literature on the subject it has been stated that 8 ppm F in the diet for 35 years is necessary before the critical levels of 6000 ppm in bone are attained (McClure et al 1958). Further human studies (Krishnamachari et al 1973) have described the severe ill effects in Andhra Pradesh where fluorosis is endemic with levels of fluoride in the drinking water of 3.5 - 6 ppm. At these levels the authors recorded genu valgum producing a slow ponderous gait, osteoporosis of the skeleton and flexion deformities of the knees. The recorded cases were in males with a range from 8 - 40 years and the fluorosis developed during school age and could be fully developed by 15 years. The article emphasises that there is a low calcium intake in the diet and moreover sorghum was the staple diet of the area containing phytate which depresses calcium absorption from the gut. As males were only affected a hormonal cause is also postulated. This type of picture is in marked contrast to the findings in Bartlett Texas (Leone et al 1954) and this contrast evoked an editorial (Lancet 1973) which concluded that high fluoride intake cannot be the only major factor responsible for the Andhra Pradesh deformities. Perhaps recent animal experiments may resolve this issue of fluoride intake and osteoporosis. These experiments indicate that nutrition may be the key factor. Cattle were used in one experiment (Suttie et al 1973) where heifers fed on 40 ppm F with only 60% of total digestible nutriments showed a terminal increase in skeletal retention of fluoride compared with the control group receiving 100% total digestable nutrient diet. The role that calcium plays in the diet has been confirmed in rats which on low calcium diet had altered bone metabolism and x-rays showed osteoporosis (Srirangareddy et al 1972). Thus although cautions in accepting animal experiments applying to humans these experiments confirm the findings in humans that diet plays an important role in the types of bone fluorosis encountered in different geographical locations. A more recent paper deals with the nutritional and vitamin deficiency problems in different parts of India and Andhra Pradesh where genu valgum only occurs in one area of the province near where a dam was built and similar findings have come from an area of South Africa close to a dam(Krishnamachari 1976). Other factors such as uraemia and disease may also result in atypical bone pictures and some will be discussed under different sections.

The clinical features of skeletal fluorosis may not always be obvious in the early stages and may be misdiagnosed as of rheumatoid origin especially in young adults who complain of vague and transient pains in the smaller joints usually the hands and feet. As the absorption levels and desposition levels increase so do the symptoms becoming more definite with the development of stiffness of the spine and limitation of movements. At still a later stage other joints become stiff and the individual now develops an increasing kyphosis. The picture in advanced stages is not unlike ankylosing spondylitis ( Steinberg et al 1955 ). In extreme cases the rigidity of the thoracic cage and the posture can induce dyspnoea. The crippling deformities that eventually develop are partly due to mechanical factors and partly due to immobilisation resulting from pain and paraplegia (Singh et al 1962).

The radiological changes are characterised by an increase in the density of trabecular bone especially in the lumbar spine and pelvis in that order and then other bones are also involved. An increase in the thickness of long bone cortices with narrowing of the medullary canal is also observed due to endosteal and periosteas apposition. More advanced cases have deposition of bone in ligaments especially the spinal ligaments. This radiological picture was described by Roholm in 1937 when studying the toxicity of chronic exposure to fluorides in cryolite workers. He classified three radiological stages. In stage 1 the spinal column and pelvis show roughening and blurring of the trabeculae. Stage 2 then advances the picture to the merging of trabeculae giving the bone a diffuse structureless appearance. Later in stage 3 the bone appears as marble white shadows and the configurataion is wooly. Although this is a very short condensation of the x-ray finds by Roholm it should be noted that unlike the changes seen in cryolite workes in stage 1 this is not often seen in endemic fluorosis (WHO 1970). In these endemic areas the changes are those of stages 2 and 3 with irregular exostosis throughout the skeleton but more pronounced in the spinal column with encroachment of intervertebral foramina, and deposition of bone along ligaments and tendons due to irregular periosteal bone formation. The osteosclerosis is more pronounced in the spine and then the pelvis.

The histopathological changes in fluoritic bone present a mixed and at times a confusing picture. There is an increase in new bone formation with an increase in the width and number of osteoid borders (Weatherell et al 1959). There is also a resemblance to osteomalacia which has never been completely explained and although the plasma alkaline phosphatase level is raised consistent with this finding the plasma calcium is invariably normal and the plasma phosphate is the same which is not consistent (Murray 1976). The strong chemical bonding of fluoride to calcium and phosphorus accounts for its deposition in hard tissue but a similar bonding is exhibited by fluoride for other cations principally magnesium and manganese (Fluoride 1971). In addition there are resorption spaces with fibrous tissue replacement which is attributed to secondary parathyroidism (Faccini 1966). He postulates that bone which has taken up fluoride becomes resistant to resorption which results in para-thyroid overactivity. However this observation is not uniformly accepted ( Rao et al 1975 ) and this will be further discussed nephrotoxicity.

Although fluoritic bone is sclerotic and in a sense there is more of it than normal it is not as strong as normal bone allowing spontaneous fractures to occur which may be due to this factor combined with altered stress areas due to deformity. It is recorded that these spontaneous fractures are common in this condition(Nordin 1973) and has been likened to a similar tendency in Pagets Disease.

The effects induced in the skeleton by the consumption of different levels of fluoride in drinking waters have been studied by many authors using different methods. These studies have involved histology, chemical, roentogenology and post mortem surveys for levels of up to 4 ppm F in drinking water.

The ribs of individuals living in areas with 0.5, 0.8 and 1.9 ppm F in the drinking water were examined histologically (Weidmann et al 1963). The ribs were examined for the number and thickening of the cortical trabeculae as well as for width of cortex. No differences were seen in these three groups as judged by the rib parameters.

The chemical changes in bone exposed to fluoride have been studied by using post-mortem specimens of the iliac crest, rib and vertebrae of individuals exposed to drinking water containing up to 4 ppm F (Zipkin et al 1960). The findings in bone ash relating to calcium, phosphorus and potassium were unaffected by a mean concentration of bone fluoride as high as 0.8 per cent. The carbohydrate content decreased by 10 per cent and the citrate content was reduced by 30 per cent whereas the magnesium content increased by about 15 per cent when the fluoride in bone showed an eight fold increase. It would appear therefore that fluoride is deposited in mature bone at the expense of other surface ions such as sodium, potassium, citrate and carbonate. The increase in the bone fluoride is associated with an increase in the crystallinity of the bone apatite (Posner et al 1963).

The x-ray surveys some of which were done on children receiving drinking water containing 1.2 ppm F in Newburgh over a period of 10 years (Schlesinger et al 1956). The right hand, both knees and lumber spine were x-rayed and judged for bone density and age. At the end of the ten year period no difference of any significance could be found in the x-rays. Other studies have shown that there are no x-ray changes when fluoride was less than 4 ppm, when urinary fluoride concentration was less than 10 ppm (Sargent et al 1951) or when bone contained less than about 5000 ppm F on a dry fat-free basis (Weidmann et al 1963). However these fiqures can well be not applicable to other geographically significant regions because of other factors.

As the levels of fluoride intake rises to and above 10 ppm F in drinking water such as can occur in parts of the Punjab and Southern India the bone and soft tissue deposition increase to produce the croppling deformities already mentioned under clinical symptoms.

## 2. Chronic Toxic Effects on the Kidney

There is no doubt that in doses of fluoride associated with fluoridation there has been no recorded damage in people with normal kidneys. The role of the human kidney in urinary excretion makes it a subject of importance. Apart from bone the kidney more than any other organ in the body is exposed to ingested fluoride and it has been extensively studied (Fluoridation of Public Water Supplies 1968). There are two facets to this problem (a) does fluoride in itself have a role in producing or aggravating a nephritic state and (b) as a result of impaired or diseased kidneys a person drinking water with certain level of fluoride is subjected to the risk of fluoride retention with toxic effects? As to the first problem fluoride induced renal pathology has never been reported in man from chronic exposures (WHO 1970). Also in the studies of cryolite workers (Roholm 1937) that although there was extensive changes in bones and ligaments there were no changes in the kidneys except in one case where there was recorded 'a slight grade of chronic nephritis' in a patient exposed for 24 years to excessive quantities of cryolite dust. Animal experiments have induced renal changes in very high doses when rats were fed 100 ppm )10 mgm/Kgm) over a period of six months. Hodge has found that 50 ppm F was insufficient and 100 ppm be regarded as 'the borderline water concentration at which some individuals of certain species (but not all) exhibit changes is about 100 ppm (Hodge et al 1964). In adult and elderly cancer patients doses up to 320 mgm of sodium fluoride a day for a period of five to six months and up to 200 mgm daily to leukaemic children had no evidence of acute or chronic effects in the kidneys or in relation to any other vital function (Black et al 1949).

Turning to the second problem there appears to be no increase in renal damage as judged by continued examinations of renal function. Even in the presence of extensive renal damage the ability of the kidney to excrete fluoride is quite remarkable and may be due to the diffusibility of the fluoride ion across tissue membranes. Recent reports would indicate that extensive disease of the kidneys will in time cause increased levels of plasma fluorine with effect upon bone (Cordy et al 1974). In this study patients dialysed with fluoridated water developed osteomalacia but fluorine-induced bone disease can mimic renal osteodystrophy. In uraemia there are complicated physiological pictures due to retained phosphorus, depressed serum calcium concentration, impaired formation of 1.25 (OH) $_2D_3$, (dihydrotachysterol) excess parathyroid concentration, trace metal accumulation and impaired collagen synthesis affect bone integrity and added fluoride is difficult to interpret (Rao et al 1975).

## Toxic Effects on the Thyroid

At the turn of the century fluorine was a compound used in the treatment of thyrotoxicosis. Sodium fluoride in a daily dosage of 50 - 100 mgm has been reported as causing clinical improvement and reduction of the BMR. The medication of fluoride was not without complications and its action was weak and often transitory. As iodine was a proved drug in the treatment and in particular for preoperative preparation of the patient it is not surprising that the use of fluoride was discontinued. Fortunately today other drugs can control the overactive thyroid such as thiouracil and thiourea with surgery being reserved for special cases such as adenoma of the thyroid gland with thyrotoxicosis.

The above presumed action of fluorine raised the questions would the consumption of fluoride induce goitre in areas where fluorine levels were high in the drinking water and would endemic goitre become more prevalent in these areas ? There seem to be two general premises for these questions. The group of elements of halogens includes iodine, bromine, chlorine and fluorine. It is known that iodine is essential for the proper functioning of the thyroid gland which accumulates iodine to synthetise thyroxine. Bromine and chlorine are also taken up by the thyroid in small amounts. It would therefore seem reasonable that fluorine would also be taken up by the thyroid. Also it was assumed that as fluoride acted on the thyroid there could be an iodine-fluorine antagonism. The position has been fully investigated both in humans and in animal experiments which have allowed the following conclusions to be made (a) in normal subjects fluorides have no effect after a study of forty patients given thyroid over several months (Korrodi et al 1956) (b)in the United Kingdom a country-wide survey of thyroid enlargement failed to demonstrate any connection between goitre and fluoride concentrations in water (Murray et al 1948). Neither did studies of chronic fluorosis in the Punjab (Singh et al 1962, Siddiqui 1960) nor did investigations in cryolite workers with the exception of one doubtful case in over sixty subjects examined (Roholm 1937) reveal an association between high fluoride ingestion and endemic goitre. Also in the Fort William Investigation (Agate et al 1949)workers exposed to fluorine fumes in the aluminium smelter industry showed no signs of goitre. Further studies in animal with 18 F have corroborated these findings and these studies are extensively documented (WHO 1970). There was a brief revival of this controversy following a survey of 17 Himalayan villages (Day et al 1972). The article was criticised on both the estimation of fluoride - iodine consumption by people in the region and also the statistical correlation was called into question (Neil Jenkins et al 1972). Perhaps Hodge sums the matter up and puts the problem into perspective when talking about a dog and an enlarged thyroid which has clouded text books for such a long time (Hodge H.L. et al 1974).

## Toxic Effects on the Systemic and Visceral Systems.

### (a) Endocrine System

Particular attention has been paid to the effect of fluorosis on the parathyroid glands. The calcium ion is an essential component of the skeleton as of many other functions such as muscle contraction and membrane permeability. A complicated process is thus developed to keep the balance of calcium in the body within a narrow range to allow for ingestion and excretion. This control is exercised primarily by the interactions of three major hormones involving parathyroid hormone, calcitonin and vitamin D. Other hormones may also be involved under certain conditions such as adrenal corticosteroids, oestrogens, thyroxine somatotropin and glucagon (Capen 1975). The parathyroids react to alterations of blood calcium levels and to a lesser extent the magnesium ion in the serum but not to such a marked extent as that of calcium (Mayer 1974). Among many of the factors influencing calcium are low levels of consumption and blockage of calcium absorption due to certain substances in the diet, disease in the body such as renal failure and fluorosis. In induced fluorosis in sheep there are hyperplasia of the parathyroid glands (Faccini et al 1965) where there was a five fold increase in parathyroid hormone levels in the blood after 1 week and remained elevated for 1 month. These changes were interpreted as an increased demand for parathyroid hormone due to the decreased mobilation of calcium from fluoroapatite crystals which are larger, more stable and less reactive to exchange reactions. Another effect could be lowered magnesium levels as the fluoride ion limits magnesium in the skeletal system. Hyperparathyroidism has been recorded in patients with skeletal fluorosis where the glands showed morphological evidence of hyperactivity (Teotia et al 1973).

(b) Metabolic Effects

There is a considerable review of the literature on the metabolism of fluoride (Hodge 1964) and it seems clear, as one would expect from a small molucule in the same chemical group as chloride, that it is rapidly absorbed from the intestinal tract being maximal in the stomach but can be absorbed from all parts of the intestinal tract. Rate of absorption will depend on the solubility of the fluoride compound, the presence of other trace metals such as aluminium, calcium and magnesium which can form insoluble compounds with fluorine. Calcium and phosphate will react with the more active simple fluoride compounds but will not react with the less soluble covalently bound fluorides and will allow these compounds to be available for absorption. The role of molybdenum is still to be elucidated but mention is made of increased molybdenum in soil and crops in certain parts of Andhra Pradesh (Krishnamachari 1976).

As fluoride is absorbed there is a rise of fluorine in the plasma levels but this is soon corrected with fluorine being deposited in other organs of the body especially those with a good vascular supply thus keeping the plasma fluorine level fairly constant around 0.14 to 0.19 ppm. Sudden fluctuations of the serum plasma are difficult to detect under present analytical methods but no doubt they do rise but of very short duration.

The action on the enzyme system has of late received more attention but its action has adequately been examined with documentation of the literature (Venkateswarlu WHO 1970). It can be true to say that routine laboratory tests fail to demonstrate any distinct biochemical changes resulting from the chronic toxic action of fluorides (Guminska et al 1975). In this paper the authors studied the action of fluoride on red blood cells which for their energy rely on the glycolytic pathway. Aluminium workers exposed to fluorides were examined and it was hoped that the experiments would confirm previous in vitro experiments. The greatest changes involved the magnesium dependent enzymes enolase pyruvate kinase (PK) and adenosine triphosphatase (ATP-ase) and the level of ATP in the cell. It was found that there was a decrease in ATP levels, lowered blood glucose, inhibition of glucose-6-phosphatase due to resynthesis of lactic acid in gluconeogenesis. Lactic acid levels were raised. The authors also found that there was increased cell permeability. There is no doubt that many enzyme systems are affected including glycogen metabolism but to what extent they cause symptoms is open to conjecture.

The method in which fluoride inhibits calcification is not known with certainty. Fluoride is believed to be an essential element in the structure of bone - not attached to calcium as first thought but rather by replacing hydroxyl ions in key situations of the crystal lattice as an essential "defect" to use the notions of the solid state theory.

(c) Haemopoietic System

The main difficulty in assessing the effects of fluoride on haemotological finding is the separation of other factors such as nutrition imbalances and malnutrition. No difference in blood counts or haemoglobin levels has been found in studies of industrial fluorosis (Agate et al 1949). In livestock exposed to high fluorides it was found that anaemia was not a factor. Some mention is made of low levels of fluoride which under experimental conditions will inhibit coagulation of blood but the mechanism is not known. On balance high fluoride intake does not appear to affect the haempopietic system.

(d) Neurological System

Fluorides do affect the nervous system and there is a mass of literature some of which is conflicting and has been dealt with at length (WHO 1970). Neurological complications only appear after prolonged ingestion of high levels of fluoride and where there is advanced skeletal fluorosis. There are two main types radicular and myelopathic: In the radicular type there is muscle wasting usually of the small muscles of the hand, acroparaesthesia and pain along the nerve roots affecting the upper limbs. The picture is similar to that of cervical spondylosis and the mechanics is probably due to the compression of the anterior nerve roots in the cervical region from pressure of excess bone formation with narrowing of foramina. It is a lower motor neurone lesion. The myelopathic type affects the spinal column in advanced fluorosis. The symptoms will depend at which level pressure affects the spinal cord and also the area of the cord subjected to pressure. This is an upper motor neurone lesion. The posterior columns are more commonly involved and next the spinothalamic tracts. The clinical symptoms are those associated with these tracts being affected and can progress to typical bilateral pyramidal lesions in which in advanced cases there are flexor spasms of limbs. There is an ability for fluorosis to mimic several diseases of the cord but x-rays make the diagnosis clear.

Other vague neurological symptoms have been described but in the main have not been corroborated with further observations.

(e) Cardio Vascular System

There is no evidence that fluorides causing toxicological changes in other systems have any effect on the heart or the blood vessels. The Japanese and the American studies were examined and dismissed by several commissions of enquiry. Also ectopic calcification is now dismissed as being due to fluoride but it is possible for fluoride to bind with hydroxyapatite in these calcified areas and is a secondary phenomenon.

It is also dangerous to use retrospective epidemiological studies to substantiate any points in relation to fluorides as this is a very imprecise epidemiological tool as has been shown so often in the past.

## Fluoride and Mutagenicity

From time to time this question has been raised in the past. No direct evidence has ever been demonstrated that fluorides cause mutagenicity but articles have alleged that they can cause uterine bleeding, abortion still births, dry birth and other obstetrical abnormalities. The literature and physiology have been reviewed by many (Gedalia, Hodge et al, WHO 1970). All these claims have been investigated and rejected by Commissioners of Enquiry (Royal Commission Tasmania 1968). The question of chromosomal damage has been revived following a paper at a fairly recent meeting of the American Chemical Society delivered by Dr. Aly H. Mohamed (1976) where test groups of animals were given 200, 100, 50, 5 or 1 ppm sodium fluoride in drinking water. Chromosomal damage is reported at the high dosage range but the report is far from clear at the lower range. Animal studies are always dangerous when applied to human beings as the animal model may not be correct. Further if a mother is on a urinary output of 1 ppm fluoride here soft tissues and body fluids will equiliberate at about 0.01 to 0.02 ppm (Shen et al 1974) and foetal tissues will equiliberate at a little lower level as the placenta seems to act as a slight barrier and in fact has an inverse relationship to levels of maternal fluorides much of the fluoride being deposited in calcification loci. Thus at a time when the foetus would be expected to be most vulnerable to genetic damage none has been found. Also the numerous epidemiological studies into fluorides have never confirmed congenital abnormalities above normal in high fluoride levels. It is hard therefore to understand the report of the Department of Biology, University of Missouri (1976) stating that "a concentration of 1 ppm sodium fluoride in drinking water was able to induce permanent damage to the genetic material of mice".

## Fluorides and Cancer

It has been asserted in the past that fluorides can cause cancer but epidemiological studies have not substantiated any of these claims. More recently there has been a revival of these assertions from studies in America (Yiamouyannis et al 1975) where conclusions showed an 18 per cent high total cancer rate in the ten largest American central cities (11 million population) whose public water supplies had been fluoridated for 15 years compared to ten large non-fluoridated American cities (7 million population). The American National Cancer Institute reported that there was no difference when standardisation was made to the figures. The NCI opinion was supported by Dr. D.B. Taves when he examined the same raw data (Doll 1976).

## BIBLIOGRAPHY


ALEXANDER, A.,(1967) Sodium fluoride. A summary of its toxicity with symptoms and treatment in cases of poisoning. Monthly Index of Medical Specialities 23-24 June.

AGATE, J. N., BELL, G. E., BODDIE, G. F., BOWLER, R.G., BUCKELL, M., CHEESMAN, E. A., DOUGLAS, T.H.J., DRUETT, H. A., GARRARD, Jessie., HUNTER, D., PERRY, K.M. A., RICHARDSON, J.D., and de V. WEIR, J.B. (1949) Industrial Fluorosis. A study of the hazards to man and animals near Fort William Scotland. A report to the Fluorosis Committee Privy Council Medical Research Council. Memo HMSO.

BLACK, M.M., KLEINER, I.S., and BALKER, H. (1949). The toxicity of sodium fluoride in man N.Y. St.J.Med.49, 1187-8.

CAPEN, C.C., (1975) Functional and fine structural relationships of the parathyroid glands. Adv.Vet.Sci.Comp. Med. 249-285.

CORDY, P.E., GAGON, R., TAVES, D.R., and KAYE, M., (1974) Bone disease in haemodialysis patients with particular reference to the effect of fluoride. C.M.A.Journal 110 1349-1353.

DAY, T.K., POWELL-JACKSON, P.R., (1972) Fluoride; water hardness and endemic goitre. Lancet 1135-1138.

DOLL, R., (1974) Fluoridation, New Scientist 174.

FACCINI, J.M., and CORE, A.D.,(1965) The effect of sodium fluoride on the ultrastructure of the parathyroid gland of sheep. Nature 207, 1399.

FACCINI, J.M.,(1969) Fluoride and bone. Calcif. Tissue Res. 3, 1-16.

FLOURIDE (1971)Editorial, Fluoride intoxication in humans 4, 102-108.

GOODMAN, L.S., and GILMAN (1965)The pharmacological basis of therapeutics (3rd Edition MacMillan).

GUMINSKA, Moira., STERKOWICZ, Jan., (1975) Biochemical changes in the blood of humans chronically exposed to fluorides. Acta.Med.Pol. 16, 3, 315-224.

HODGE, H.C., DOWNS, W.L., SMITH, F.A., MAYNARD, E.A., SCOTT, J.K., and GARDNER, D.E.,(1964) J.Dent.Surg.43, No. 5., Suppl., 864-865.

HODGE, H.C., and SMITH, F.A., (1974) Fluorides Chapt.7. Metallic contaminants and human health Editor Lee,D.H.K., Academic Press, 177.

KORRODI, H., et al. (1956) Helv. Med. Acta., 23, 601.

KRISCHNAMACHARI, K.A.V.R., and KRISHNASWAMY, Kamal (1973) Genu valgum and osteoporosis in an area of endemic fluorosis. Lancet ii, 877-879.

KRISCHNAMACHARI, K.A.V.R., (1976) Further observations on the syndrome of endemic genu valgum of South India. Indian J.Med.Res. 64(2) 284-291.

LANCET (1973) Editorial, Fluoride Toxicity ii, 889.

LARGENT, E.J., BOVARD, P.G., and HAYROTH, F.F., (1951). Roent genographic changes and urinary fluoride excretion among workmen engaged in the manufacture of inorganic fluorides Am.J.Roentgenol.Radium,Ther.Nucl.Med.65,42- 48.

LEONE, N.E., SHIMKIN, M.B., ARNOLD, F.A., STEVENSON,C.A., ZIMMERMAN, E.R., GEISER, P.A., and LIEBERMAN, J.E.,(1954) Publ.Hlth.Rep. (Wash). 69, 925-936.

LIDBECK, W.L., HILL, I.B., and BEEMAN, J., (1943) Acute sodium fluoride poisoning. J.A.M.A. 121, 826-827.

MAYER, G.P., 1974. Relative importance of calcium and magnesium in the control of parathyroid secretion. Programme of the 56th annual meeting, the Endocrine Soc. Atlanta, Georgia, Endocrine Soc. p, A-181.

McCLURE, F.J., McCANN, H.G., and LEONE, N.C., (1958) Pub. Hlth. 73, 741.

MOHAMED, A.H., (1976) Reports of a paper to American Chemical Society, Food Chemical News, 30.

MURRAY, M.M., RYLE, J.A., SIMPSON, B.W., and WILSON,D.C., (1948) Endemic goitre in Great Britain in relation to the iodine, calcium, fluoride content of water supply in thyroid enlargement and other changes related to the mineral content of the drinking water. Memor.Med.Res.Coun. 18:3.

MURRAY, J.J., (1976) Fluorides in caries prevention. John Wright Bristol, 164-165.

NEIL JENKINS, G., and COOKE, J.A. (1972) Letter to Lancet 1293.

NORDIN, B.E.C., (1973) Metabolic bone and stone disease Churchill Livingston Edinburgh.

POSNER, A.S., EAMES, E.D., HARPER, R.A., and ZIPKIN, I., (1963) x-ray diffraction analysis of the effects of fluoride on human bone apatite. Arch.Oral Biol. 8, 549-570.

ROA, T.K.S., and FRIEDMAN, E., (1975) Editorial: Fluoride and bone disease in uraemia. Kidney International.Vol. 7, 125-129.

ROHOLM, K., (1937) Fluorine intoxication: A clinical-hygiene study with a review of the literature and some experimental investigations. Lewis London.

SCHLESINGER, E.R., OVERTON, D. E., CHASE, H.C., and CANTWELL, K.T., (1956) Newburgh-Kingston caries-flourine study, xiii Pediatrict findings after 10 years J.Am.Dent. Assoc. 52, 296-306.

SINGH, A., DASS,R., HAYRETH, S.S., and JOLLY, S.S. (1962) Skeletal changes in endemic fluorosis J.Bone. Joint.Serg. 44B, 806-815.

SMITH, F.A., GARDINER, D.E., and HODGE, H.C., (1952) At. Energy Comm. (U.S.) Quart.Tech.Rep.UR-200.

SRIRONGAREDDY, G., and RAO, B.S., 1972 Effect of fluoride on the skeleton of rats maintained on different levels of calcium in the diet. Indian J.Med.Res.80,481.

STEINBERG, C.L., GARDNER, D.E., SMITH, F.A., and HODGE, H.C., Comparison of rheumatoid (ankylosing) spondylitis and crippling fluorosis. Am.Rheum.Dis.14, 378-384.

SUTTIE, J.W., and FALTIN, E.C., 1973. Effects of sodium fluoride on dairy cattle: influence of nutritional state. Am.J.Vet.Res, 34, 479.

SHEN, Y.W., (1964) Fluoride in mineral metabolism. Ed. Comar, C.L., and Bronner, F., Academic Press 11A, 573.

TEOTIA, S.P.S., and TEOTIA, M., (1973) Secondary hyperthyroidism in patients with endemic skeletal fluorosis. Brit.Med.Jour.1, 637.

UNDERWOOD, E.J., (1971) Trace Elements in humans & animal nutrition. Academic Press p.370.

WALLACE-DURBIN, P., (1954) J.Dent.Res. 33, 789.

WEATHERALL, J.A., and WEIDMANN, S.M., (1959) The skeletal changes of chronic experimental fluorosis. J.Path. Bact. 78, 233-241.

WEIDMANN, S.M., WEATHERALL, J.A., and JACKSON, D., (1963) The effect of fluoride on bone.Proc.Nutr.Soc.22, 105-1110.

WORLD HEALTH ORGANIZATION (1970) Fluorides and human health. Monogr.Ser.No.59 Geneva.

YIAMOUYIANNIS, and BURK, D., (1975) U.S.Congressional Record 21 July pH7172 and 16 Dec. pH12731.

YOLKEN,R.,et al(1976)Acute fluoride poisoning Paediatrics Vol. 58, 90.

ZIPKIN, I., McCLURE, F.J., and LEE, W.A., (1960) Relation of the fluoride content of human bone to its chemical composition. Arch.Oral.Biol.2, 190-195.

# A PRELIMINARY STUDY ON THE EFFECT OF WATER-BORNE FLUORIDE ON THE FLUORIDE CONTENT OF SOILS AND PLANTS

by Professor Nazmi Oruç
Atatürk University
Faculty of Agriculture
Soil Science Department
Erzurum - Turkey

## Abstract

The purpose of this study was to determine the effect of naturally occuring high-fluoride waters on the fluoride content of soils and plants in the endemic fluorotic areas in eastern Turkey. Water-soluble fluoride determinations were made on the soil and plant samples collected from the fluoride contaminated areas and the control sites. Determination of fluoride was made using an ion-selective electrode. The fluoride levels of soils taken from contaminated and control sites ranged from 1.64 to 13.75 ppm (median = 12.00) and from 0.05 to 0.54 ppm (median = 0.1500),respectively. Analysis of the plants growing in fluoride-contaminated soil and control sites showed levels of fluoride ranging from 1.85 to 33.60 ppm (median = 4.66)and from 0.51 to 1.13 ppm (median = 0.90), respectively. A significant positive correlation ( r = 0.606, p = 0.05) was found between the water-soluble fluoride level of soil and plant samples. It is concluded that water-borne fluoride contribution to the water - soluble fluoride content of soils has a significant impact on the fluoride uptake of the plants. In addition to the high uptake of fluoride (6.5 - 12.5 ppm) from drinking waters, livestock grazing in these areas, can ingest some fluoride from the soils and plants.

## Abstract in Turkish (Türkçe Özet)


### Doğal Koşullarda Fluorürlü Sularla Bulaşmış Bazı Toprak ve Bitki Örneklerinde Fluorür İçeriği

Bu çalışma Doğubayazıt yöresinde içme ve sulamada kullanılan fluorürce zengin sularla bulaşmış bazı toprak ve bitki örneklerinde fluorür içeriğini belirlemek ve bu yolla hayvanların alabileceği fluorür durumu hakkında bazı ipuçları sağlamak amacıyla yapılmıştır. Bu nedenle fluorürlü sularla bulaşmış sahalardan alınan 9 adet bitki ve 7 adet yüzey toprak örneğinde ve kontrol sahalardan alınmış 7 adet bitki ve 10 adet yüzey toprak örneğinde su ile eksrakte edilebilir fluorür içeriği, fluorüre duyarlı elektrot aracılığı ile tayin edilmiştir. Analiz sonuçlarına göre fluorürlü sularla bulaşmış bitki örneklerinde fluorür içeriği 1.85 ile 33.60 ppm (median = 4.66), toprak örneklerinde ise 1.64 ile 13.75 ppm (median = 12.00) arasında değişmektedir. Kontrol olarak alınan bitki örneklerinde fluorür içeriğinin 0.51 ile 1.13 ppm (median = 0.90), toprak örneklerinde 0.05 ile 0.54 ppm (median = 0.12) arasında değiştiği saptanmıştır. Bazı toprak ve bitki örnekleri (n = 11) arasındaki fluorür içeriği ile ilgili istatistiki hesaplamada korelasyon kat-sayısı ($r = 0.606^x$) önemli bulunmuştur. Elde edilen bulgular fluorürce doğal olarak zengin suların kullanıldığı toprak ve bu topraklarda yetişen bitkilerde fluorür içeriğinin arttığını göstermektedir. Bu durumda bölgedeki çiftlik hayvanlarının içme suyundaki fluorüre ilâveten, toprak ve bitkiler aracılığı ile bir miktar daha fluorür almaları ve diş ve iskelet florozuna yol açan toksik fluorür seviyesinin artması mümkündür.

## Introduction

Fluoride is widely distributed in nature and is a common consituent of most soils and rocks. It occurs chiefly as fluorapatite. ($Ca_{10}F_2(PO_4)_6$, fluorspar ($CaF_2$), cryolite ($Na_3AlF_6$) and in combination with silicates as topaz, tourmaline, the micas etc. In soils fluoride is mainly derived from these minerals and usually has a concentration on the range of 20 - 500 ppm (Mitchell and Edman, 1945., Robinson and Edgington, 1946). Artificially high soil fluoride levels can occur through contamination by polluted air, the addition of phosphate fertilizers, from organic and inorganic pesticides(Cooke et al, 1976), or from water-borne fluoride. However, little is known about the effect of naturally occuring high-fluoride water on soil fluoride and subsequent uptake by vegetation.

Machle and associates (Mitchell and Edman, 1945) found no correlation between the fluoride content of food plants grown in Arizona and the fluoride content of the respective local water supply. MacIntere and coworkers (1952) concluded that soil was not an important fluoride source for forage crops growing in a flouride-contaminated atmosphere in Tennessee. It has been shown conclusively that the amount of fluoride which is taken up from the soil by plants is usually unrelated to the fluoride content of the soil (MacIntere, et al, 1942, Prince et al, 1949, Hurd-Karrer, 1950, Garner, 1963, Manley et al, 1975, McClenahen, 1976). Soil type calcium and phosphorus content, and soil reaction (pH) seem to be the predominant factors controlling fluoride availability to plants. It has been well demonstrated that only under very special conditions, such as very acid, sandy soils low in phosphorus and calcium, plant uptake of fluoride is related to total fluoride content of the soil (Brewer, 1965, 1966). Almost invariably calcium is the dominant exchangeable cation and precipitative ion in the soils of arid and humid areas. Conversion to calcium fluoride, therefore, will be the fate of the other fluoride compounds introduced into the soil by phosphatic fertilizers, slags, spray residues, gaseous and solid emissions from manufacturing operations, rain water (MacIntere et al, 1942, Brewer, 1966) and high-fluoride water.

Brewer (1965) also indicated that presumably the soluble fluoride content should correlate more closely with plant uptake, since solubility is probably the primary factor involved, but he also added that corroborating data are lacking. However, Larsen and Widdowson (1970) stated that, in any event, the uptake of fluoride by plants would not be controlled by the concentration of water-soluble fluoride alone. Although most workers have found no relationship between total fluoride in the soil and that in the plant, recently Israel (1974) found a strong correlation between fluoride levels in surface soils and in forage near an alumina reduction facility in Maryland. He explained the good correlation by pointing out that, because of low availability of soil fluoride a significant correlation could be expected only if the soils studied had large variations in their fluoride content. Vegetation growing in areas free from fluoride pollution generally contains 10 ppm or less fluoride in the foliage, indicating that most plants are poor accumulators of soil fluorides (Hansen et al, 1958, MacIntere et al, 1949). Notable exceptions include tea leaves, which have been found to contain, on the average, 50 to 90 ppm of fluoride. Fluoride which is not considered essential for the normal growth of plant, is essential, for animals. It is concluded that animals obtain their fluoride from the plants which they eat and the water which they drink. However, the animals may be detrimentally affected by eating forage containing 50 ppm or less fluoride, or drinking water containing 10 tO 20 ppm of fluoride (Mitchell and Edmand, 1952, Brewer, 1966). It has been reported by Oruç (1977) that fluoride levels of some spring waters, used for drinking and irrigation purposes, ranged from 6.5 to 12.5 ppm in the eastern Anatolia. The dental and skeletal fluorosis were observed in the human beings and livestock around the Tendürek volcano (Oruç, 1973, 1975, Şendil and Bayşu, 1973, Öztopçular, 1975).

The purpose of this preliminary study was: a) to determine the effect of naturally occuring high-fluoride water on the water-soluble fluoride content of soils and plants, and thus, b) to get some information about the extent of fluoride ingestion by the farm animals grazing in these areas.

## Materials and Methods

High-fluoride spring waters are located around the Tendürek volcano, near the Iranian border, in the eastern Anatolia. Fluoride content of levels of fluoride found in these waters, which have been in use for both drinking and irrigation purposes for many years, are considered to be hazardous to human and livestock health.

Table 1. Fluoride Content in ppm of Some Spring Waters in Doğubayazıt District

| Location | Fluoride | Location | Fluoride |
|---|---|---|---|
| Gökçekaynak | 11.7 | Celâl | 5.0 |
| Girnevik | 12.5 | Kanikork | 9.2 |
| Altıntepe | 12.0 | Topçatan | 8.0 |
| Üçgöze | 6.5 | Çiftlik | 5.0 |

Nine plant and 7 surface soil samples were collected from different localities in fluoride-contaminated areas. Seven plant and 10 surface soil samples obtained from different uncontaminated areas were used as control. Air-dry soil samples were ground to pass a 425 //sieve. In the determination of the water-soluble fluoride content of soils, it is desirable to extract only that fluoride which is likely to be soluble in the soil solution. For this reason 1:1 soil-to-water extract was used to avoid that appreciable amounts of relatively insoluble fluoride compounds like $CaF_2$ are not solubilized (Brewer, 1965). pH (1: 2.5 $H_2O$) values, electrical conductivities (1 :1 extract) and $CaCO_3$ percentages (titration) of the soil samples were also determined. Aerial part of the plants were taken as a whole and oven dried at 65 °C for 12 hours, and ground, but not sieved. In general, plants were at the stage of flowering, when they were collected. One gm oven-dried and milled plant material was placed in a 50 ml centrifuge tube and 20 ml distilled water added. After shaking for three hours in a rotary shaker, samples were centrifuged and then filtered. Determination of fluoride in soil and plant extracts were made by using a specific-ion fluoride electrode (Activion). It works in a manner similar to the glass electrode for hydrogen ion determination, the principal difference being replacement of the glass membrane by a single crystal of lanthanium fluoride (Larsen and Widdowson, 1970). This electrode is coupled with a single junction reference electrode and used with a potentiometer as for pH measurement. The electrode is specifically sensitive to the activity of fluoride ions the pH range 5-8. To dissociate fluoride complexes and stabilize, both standards and samples were diluted 1:1 with a buffer solution consisting of 57 ml glacial acetic acid, 58 gm sodium chloride and 0.3 gm sodium citrate per litre, adjusted to pH 5.0-5.5 with NaOH (Frant and Ross, 1968). The fluoride ion activity electrode was used with the expanding scale pH meter (EIL model 7030) and a mercury-mercurous reference electrode. All solutions were stirred with a magnetic stirrer and the electrode potentials were determined after five minutes.

## Results and Discussion

The data in table 2 show that water-soluble fluoride levels of the soil samples collected from fluoride-contaminated areas are much higher that the soils of control sites. It has been reported by Cooke and coworkers (1976) that water-soluble fluoride levels of the soils collected from fluorspar mine waste ranged from 1.7 to 9.6 ppm. Whilst, water-soluble fluoride content of the control site was reported as being 0.1 ppm. Since the pH values of soils are above 7 and in general they contain rather high amounts of $CaCO_3$, calcium fluoride can be considered as the form to which water-borne fluoride pass after incorporation into the soils (Brewer, 1966). Even though the formation of relatively insoluble calcium fluoride is expected, the results indicate that irrigation of these soils with high-fluoride waters increased the water-soluble fluoride levels upon time. It has been reported by Oruç, et al, (1975). that the high fluoride waters

Table 2. Water-soluble fluoride, pH, electrical conductivity and % $CaCO_3$ values of the soil samples

Fluoride-contaminated

| Location | Fluoride ppm | pH | $Ecx10^3$ | % $CaCO_3$ |
|---|---|---|---|---|
| Gökçekaynak | 7.3 | 8.70 | 0.46 | 10.0 |
| Altıntepe | 16.0 | 8.25 | 0.62 | 11.2 |
| Uçgöze | 12.8 | 8.40 | 0.96 | 8.4 |
| Çiftlik | 1.6 | 8.30 | 6.10 | 2.7 |
| A. Yılanlı | 12.0 | 9.00 | 0.78 | 40.6 |
| Topçatan | 8.2 | 9.85 | 1.60 | 11.2 |
| Atabakan | 13.7 | 9.70 | 14.50 | 12.0 |

Control Sites

| Location | Fluoride ppm | pH | $Ecx10^3$ | % $CaCO_3$ |
|---|---|---|---|---|
| Gökçekaynak | 0.23 | 7.90 | 0.40 | 9.6 |
| Altıntepe | 0.54 | 8.10 | 0.58 | 20.0 |
| Uçgöze | 0.05 | 8.10 | 0.70 | 6.6 |
| Erzurum | 0.06 | 7.25 | 0.30 | Nil |
| Erzurum | 0.17 | 8.20 | 0.38 | Nil |
| Erzurum | 0.07 | 7.00 | 0.32 | Nil |
| Atabakan | 0.32 | 7.95 | 11.50 | 20.3 |
| Altıntepe | 0.12 | 8.10 | 0.44 | Nil |
| Gökçekaynak | 0.11 | 8.10 | 0.50 | 43.7 |
| Erzurum | 0.22 | 7.90 | 0.72 | Nil |

around the Tendürek volcano are $NaHCO_3$ type. The data in table 2 indicate that pH values and electrical conductivities of the fluoride-contaminated soils are much higher than the soils of control sites. This can be related to the presence of high amounts of $NaHCO_3$ in high-fluoride waters.

Table 3, shows the water-soluble fluoride levels of plant samples collected from fluoride-contaminated and control sites. The date in table 3, indicate that the fluoride content of plants grown on contaminated soils are much higher than the plants of control sites.

Table 3. Water-soluble fluoride levels of the plants collected from fluoride-contaminated and control sites.

Fluoride-contaminated

| Location | Species | fluoride ppm |
|---|---|---|
| Gökçekaynak | Medicago sativa | 2.41 |
| Altıntepe | Medicago sativa, Koeleria cristata, Poa bulbosa | 4.66 |
| Oçgöze | Glyceria sp. | 14.25 |
| Çiftlik | Medicago sativa | 2.68 |
| A.Yılanlı | Medicago sativa | 4.84 |
| Topçatan | Medicago sativa | 1.85 |
| Atabakan | Poa bulbosa, Koeleria cristata, Salvia sp. | 3.27 |
| İçtegel | Phragmites sp. | 33.60 |
| A.Yılanlı | Puccinella sp. | 4.70 |

Control Sites

| Location | Species | fluoride ppm |
|---|---|---|
| Erzurum | Festuca ovina, Poa bulbosa, Koeleria cristata | 1.13 |
| Erzurum | Medicago sativa | 0.84 |
| Erzurum | Medicago sativa | 1.08 |
| Erzurum | Medicago sativa | 0.57 |
| Karabulak | Phragmites sp. | 0.90 |
| Erzurum | Poa bulbosa Koeleria cristata | 0.51 |
| Erzurum | Puccinella | 1.10 |

It is expected that water-soluble fluoride levels of the plants are generally lower than the total-or acid extractable fluoride values, due to the incomplete extraction of fluoride from plants with deionized water (Jacopson et,al,1966, Cooke et al 1976). However, water-soluble fluoride levels of the plants can be used as an index by showing the most toxic fluoride form to the farm animals. It has been reported by Cooke and coworkers (1976)that extraction of leaf tissue with water indicated that little of the fluoride was present in a readily ionized form suggesting the presence of calcium magnezium, aluminium or silicate complexes which might be expected to be less toxic than fluoride ions. As toxicity is usually greater when fluoride is in a more soluble (e.g sodium fluoride) rather than in a sparingly soluble (e.g. calcium fluoride) from (Mitchell and Edman, 1952,Eagers, 1969), it is considered that the water-soluble fluoride fraction of plants can be an appropriate indicator in the diagnosis and extent of fluorosis in livestock.

A significant positive correlation ($r = 0.606$, $p = 0.05$) was found between the water-soluble fluoride level of the soil and plant samples ($n = 11$) taken from the same positions. This can be related to the fact that the soils studied had large variations in their water-soluble fluoride levels. Cooke et al, (1976) reported that analysis of plants from fluorspar-contaminated sites showed elevated levels of fluoride in leaves compared with the normal range. They also indicated that as with total soil fluoride, water-soluble fluoride showed no correlation with plant concentrations. However, Israel (1974) found a strong correlation between fluoride levels in surface soils and in forage near an alumina reduction facility in Maryland.

It is concluded that continuous use of high fluoride water (6.5 - 12.5 ppm) in irrigation for many years, has increased the water-soluble fluoride content of soils and subsequent uptake of the fluoride by the vegetation grown on these areas. In addition to the high uptake of fluoride from drinking waters, therefore, livestock grazing in these areas can ingest some fluoride from the soils and plants.

## Acknowledgements

Thanks are due to Professor D.J. Greenland, Head, Dept. of Soil Sci. Reading Univ.England, and his academic and technical staff for providing necessary facilities, Financial support was provided by the Atatürk University for three months.

## BIBLIOGRAPHY

BREWER, R.F.(1965), Fluorine, in methods of soil analysis Part 2 BLACK, C.A. Editor-in-chief American Soc. of Agronomy, Madison, Wisconsin, Agronomy Ser.No.9.

BREWER, R.F. (1966), Fluorine in diagnostic criteria for plants and soils. Edited by H.D. Chapman.Univ. of California, Division of Ag. Sci. U.S.A.

COOKE, J.A., JOHNSON, M.S., DAVISON, A.W. and BRADSHAW, A.D. (1976). Fluoride in plants colonising fluorspar mine wast in the Peak district and Weardale, Environ. Pollut. (11) 9-23.

EAGERS, R.Y. (1969). Toxic properties of inorganic fluorine compounds. Elsevier Publ. Com. LTD. London.

FRANT, M.S. and ROSS, J.W. (1968). Use of a totallionic strength adjustment buffer for electrode determination of fluoride in water supplies. Analytical Chem. Vol.:40, No. 7, p. 1169-1171.

GARNER, R.J. (1963). Fluorine, Veterinary toxicology. Second edition. The Williams and Wilkins Company. Baltimore. p. 83-91.

HANSEN, E.D., WIEBE, H.H. and THORNE, W. (1958). Air pollution with relation to agronomic crops VII. Fluoride uptake from soils. Agronómy Journal. Vol. 50 : 565-568.

HURD-KARRER, A.M. (1950). Comparative fluorine uptake by plants in limed and unlimed soil. Soil Sci. 70 : 153-160.

ISRAEL, G.W. (1974). A field study of the correlation of static lime paper sampler with forage and cattle urine. Atmos. Environ. 8 : 167-181.

JACOBSON,J.S., WEINSTEIN,L.H., McCUNE,D.C. and HITCHCOCK, A.E. (1966). The accumulation of fluorine by plants. J. Air Pollution Cont. Assoc. Vol : 18 Part 8, p. 412-417.

LARSEN, S. and WIDDOWSON, A.E. (1970). Soil fluorine. The J. of Soil Sci. Vol : 22, No. 2, p. 210-221.

MacINTERE, W.H., WINTENBERG, S.H., THOMPSON, J.G. and HATCHER, B.W. (1942). Fluorine content of plants. Ind. Eng. Chem. 34 : 1469-1479.

MacINTERE, W.W. and Associated (1949). Effects of fluorine in Tennessee Soils and Crops. Ind. Eng.Chem. 41 : 2466-2475.

MANLEY, T.R., STEWART, D.J., WHITE, D.A. and Harrison,D.L. (1975). Natural fluorine levels in Bluff area, New Zealand, New Zealand Journal of Science Vol : 18,433-440.

McCLENAHAN, J.R. (1976). Distribution of soil fluorides near an airborne fluoride source J. Environ. Qual.Vol : 5, No. 4.

MITCHELL, H.H., EDMAN, M. (1945). Fluorine in soils, plants and animals. Soil Sci. 60 : 81-90.

MITCHELL, H.H. and EDMAN, M., (1952). The fluorine problem in livestock feeding: The commonwealth bureau of ani-nutrition. Nutrition Abs. and Reviews. 21.4.

ORUÇ, N. (1973). Doğubayazıt Kazası ve bazı köylerinde kullanılan sularda fluorür konsantrasyonu ve önemi. Atatürk Üni.Ziraat Fak.Ziraat Dergisi, Cilt:4, Sayı : 1.

ORUÇ, N., ALPMAN, N., KARAMANDERESİ, İ.H. (1975). Hydrogeology of the spring waters with high F content from the surroundings of Tendürek volcano. Bulletin of the Geological Society of Turkey. Vol: 19, 1-8, Feb.

ORUÇ, N. (1977). Fluoride content of some spring waters and fluorosis in the eastern Anatolia. Presented to the symposium "Problems of high fluoride waters" held on June 6-10, 1977, Erzurum, Turkey.

ÖZTOPÇULAR, M., (1975). Doğubayazıt yöresinde endemik fluorosis. Atatürk Üni. Tıp Fakültesi, Nüroloji Servisi, ~~(Unpublished)~~ In this issue.

PRINCE, A.L., BEAR, F.E., BRENNAN, E.G., LEONE, I.A. and DAINES, R.H. (1949). Fluorine : Its toxicity to plants and its control in soils. Soil Sci. 67 : 269-277.

ROBINSON, W.O. and EDGINGTON, G. (1946). Fluorine in soils. Soil Sci. 61 : 341-353.

ŞENDİL, Ç. ve BAYŞU, N. (1973). İnsan ve hayvanlarda Ağrı ili Doğubayazıt ilçesi köylerinde görülen flor zehirlenmeleri ve bunu Van ili Muradiye ilçesi köylerinde de saptamamızla ilgili ilk tebliğ. Ankara Üni. Veteriner Fak. Dergisi, Cilt : XX, No. 4.

FLUORIDE CONTENT OF SOME SPRING WATERS

AND FLUOROSIS IN THE EASTERN ANATOLIA

by Professor Nazmi Oruç
Atatürk University
Faculty of Agriculture
Soil Science Department
Erzurum - Turkey

Abstract

Some spring water samples used for drinking and irrigation purposes were collected from the Eastern Anatolia, mostly around the Tendürek Volcano. The fluoride concentrations of the water samples were determined by the colorimetric acid zirconium alizarin method. It was found that seven spring water samples taken from Doğubayazıt district(north of Tendürek volcano)containing very high amounts of fluoride, ranging from 6.5 to 12.5 ppm. The fluoride concentrations in five water samples collected from Çaldıran area (south of Tendürek volcano ) ranged from 2.0 to 7.5 ppm. The water samples taken from Sarısu (Süphan volcano) and Tatvan (Nemrut volcano) districts contained fluoride, ranging from 0.2 to 0.7 and from 2.0 to 5.0 ppm, respectively. Except Sarısu samples, the levels of fluoride found in these water samples were considered to be hazardous to the human and stock health. Dental fluorosis was observed in the local population and in cattle and sheep, during the survey of the endemic fluorotic areas, around the Tendürek volcano. The investigations carried out by the medical and veterinary researchers indicated chronic fluoride intoxication in human beings and livestock in the studied areas. It has been estimated that approximately 15 thousand people and 60 thousand livestock are being exposed to high fluoride ingestion around the Tendürek volcano. Dilution of fluoride-rich waters with other suitable waters down to a safe level (1.5 ppm) was recommended to prevent the hazardous effects of the fluoride on the human and stock health and also to avoid the contamination of the soils and the pastures in these areas.

## Abstract in Turkish (Türkçe Özet)


### Doğu Anadolu Bölgesinde Bazı Kaynak Suları, Toprak Ve Bitki Örneklerinde Fluorür İçeriği ve Bununla İlgili Olarak Fluorür Zehirlenmesi

Doğu Anadolu bölgesinde Tendürek volkanının kuzey eteklerindeki Doğubayazıt ovasında ve güney eteklerindeki Çaldıran ovasında içme ve sulamada kullanılan bazı kaynak sularında yapılan fluorür analızı sonucunda Doğubayazıt yöresindeki suların litrede miligram olarak 6.5 ile 12.5 arasında, Çaldıran yöresindeki suların ise litrede miligram olarak 2.0 ile 7.5 arasında fluorür içerdikleri saptanmıştır. Bulunan bu değerler literatürde insan ve hayvanlar için zehirli olarak belirlenen seviyelerin üstündedir. Bölgede tıp ve veteriner uzmanlarınca yapılan çalışmalarda insan ve hayvanlarda diş ve iskelet florozu tespit edilmiştir. İçme suyu veya diğer yollarla, özellikle kemiklerin teşekkülü devresinde insan ve hayvanların bünyelerine yüksek seviyede ve devamlı olarak fluorür alınması halinde ilk plânda kesici dişlerin ön yüzeyinde sarı-kahverengi lekeler meydana gelmektedir. Dişlerdeki aşırı derecede aşınma ve kırılmalar dolayısıyla sığır ve koyunlar normal olarak beslenememekte ve bu nedenle yöre halkı büyük ekonomik kayba uğramaktadır. İnsanlarda ise orta yaşları takiben iskelet sisteminde ortaya çıkan anormallikler nedeniyle omurga ve eklem hareketleri çok güçleşmekte ve büyük acı vermektedir. Bölgede 15 bin dolayında insan ve 60 bin dolayında büyük ve küçükbaş hayvan bu suları kullanmak, içmek veya bu sularla sulanan tarla ve çayırlarda yetişen bitkileri yemek mecburiyetindedir. Fluorürlü suların kullanıldığı tarla ve çayırlardan alınan çeşitli toprak ve bitki örneklerindeki fluorür seviyesinin, kontrol olarak alınan toprak ve bitkilerin fluorür seviyelerinden çok daha fazla olduğu tesbit edilmiştir. Bu durumda bölgedeki çiftlik hayvanlarının içme suyundaki fluorüre ilâveten, toprak ve bitkiler aracılığı ile bir miktar daha fluorür almaları ve diş ve iskelet florozuna yol açan zehirli fluorür seviyesinin artması mümkündür.

Öneriler

1) Bölge halkına ve tek geçim kaynağı olan çiftlik hayvanlarına en kısa zamanda Balık Gölünden veya diğer kaynaklardan fluorürsüz su sağlanmalıdır. 2) Doğubayazıt ilçe merkezine su sağlıyan Topçatan pompasında aktive aluminyum veya iyon değiştirici rezinler aracılığı ile fluorürün uzaklaştırılması mümkündür, 3) Yüksek seviyede fluorür ile bulaşmış tarla ve çayırlar aracılığı ile hayvanların fluorür almalarının önlenmesi için fluorürlü suların temiz sularla karıştırılarak fluorür seviyesinin düşürülmesi gerekmektedir.

## Introduction

Origins of fluoride in waters : Fluoride is widely distributed in a combined form in several naturally occurring minerals including fluorite ($CaF_2$), cryolite, ($AlF_3$, $3NaF$), topaz, tourmaline, the micas, etc., in which it is found in combination with silicates, but particularly in association with phosphorus as fluoroapatite ($Ca_5(PO_4)_3F$). It was estimated that the element fluoride in the form of fluorides constitutes about 0.032% of the earth's crust (Mitchell and Edman, 1945, Garner, 1963, Brewer, 1966). Because of the wide interest in the relationships of fluorides to general health, an extensive literature is available on the distribution of fluoride-containing waters (Fluorides and Human Health, 1970). Except in unusual circumstances, surface waters are generally low in fluoride, the levels being below 1 ppm. On the other hand underground and subsoil waters may have greater opportunity to contact fluoride bearing materials so that these waters may contain appreciable amounts of fluoride depending on geological conditions. These waters containing higher concentrations of fluoride are generally obtained from deep wells, and the fluoride seems to originate, not from leachings from surface zones but from deep-seated rock formations where the gaseous exhaltations have been retained under pressure and are commonly regarded as of pneumatolytic origin (MacIntere, et al 1942, Mitchell and Edman, 1952).

Effects of fluoride on human health : The principal sources of supply of fluoride available to the physiology of man are: (1) Water, (2) Some species of vegetation, (3) Certain edible marine animals, (4) Dusts in certain parts of the world, (5) Certain industrial processes. It has been well established that where fluoride occurs in abnormal amounts in the water or soil, a chronic endemic fluorosis is likely to exist, the most obvious sign of which are characteristic changes in the teeth of local population. On the basis of the clinical appearance of the teeth, the term "mottled enamel" was introduced and mottled teeth are characterized by minute white flecks yellow or brown spot areas scattered irregularly over the tooth surface. The permanent teeth are particularly affected, although occasional mottling of the primary teeth may also be observed. The chronic toxic effects of fluoride on the skeleton system have been described from certain geographical regions of the world where drinking water contains excessive quantities of natural fluoride (Eagers, 1969. Fluorides and Human Health, 1970). Dental fluorosis in the local people of Isparta was first reported by Ata (1966). And it was related to the drinking water containing 4.03 ppm fluoride.

The relationship between the concentrations or doses of fluoride and its biological effects is indicated in the table 1 (Fluorides and Human Health, 1970). p. 225-226

Table 1. Relationship between the concentrations or doses of fluoride and its biological effects

| Concentration or dose of fluoride | Medium | Effect |
|---|---|---|
| 2 parts per 1000 million | Air | Injury to vegetation |
| 1 ppm | Water | Dental caries reduction |
| 2 ppm or more | Water | Mottled enamel |
| 5 ppm | Urine | No osteosclerosis |
| 8 ppm | Water | 10% osteosclerosis |
| 20-80 mg/day or more | Water or air | Crippling fluorosis |
| 50 ppm | Food & water | Thyroid changes |
| 100 ppm | Food & water | Growth retardation |
| More than 125 ppm | Food & water | Kidney changes |
| 2.5 -5.0 gm. | Acute dose | Death |

These levels are based on the USA and European experiences. However, the variations in the climatic, nutritional and environmental conditions greatly influence the effect of fluoride in drinking water. In the USA manifest mottling is reported to be associated with at least 3 to 4 ppm fluoride - a level at which many workers in India have recorded cases of skeletal fluorosis. Each country should, therefore, make its own assessment of the permissible concentration of fluoride in its water supplies.

Effects of fluorides on animals : Various sources may contribute to the total fluoride intake of animals. The most commonly encountered sources of excessive fluorides are : (1) High fluoride water from natural or industrial sources, (2) Forages subjected to airborne contamination in areas near certain industrial operations, (3) Feed supplements and mineral mixtures containing excessive fluoride, (4) Vegetation growing in soils high in fluoride, and (5) A combination of any or all of the preceding four. The most sensitive clinical index of fluoride absorption in the mottling, staining and excessive wearing of permanent teeth that were forming at the time of the fluoride ingestion, Teeth that had matured before the time of fluoride absorption, are not affected. At a more advanced stage of fluorosis, a distinctive bilateral lameness and stiffness of gait may be observed (Hobbs, et al 1954, Garner, 1963, Eager, 1969, Shupe and Olson, 1971, Jones, 1977).

Drinking water may contribute significantly to the fluoride hazard if the fluoride content attains levels of several parts per million. Chronic endemic dental fluorosis has been observed in sheep and cattle who drank waters containing 10 to 20 ppm of fluoride (Mitchell and Edmand, 1952, Bear 1957).

Fluoride content of some spring waters and fluorosis : After receiving a letter from the headman of Gökçekaynak village, one of the most seriously affected villages in the endemic fluorotic area in Doğubayazıt district, at eastern Anatolia, near the Iranian border, the researchers from Atatürk University began to study on this problem in March, 1972. In his letter to the University the headman, Mr. Abdülkadir İlhan indicated the symptoms of dental fluorosis in the local population and livestock drinking water from the stream flowing near the village. Eighteen water samples, mostly from the areas around the Tendürek volcano, were collected during the survey of the endemic fluorotic areas. The fluoride concentrations of the water samples were determined by the colorimetric acid zirconium alizarin method (Yenal,1970). It has been estimated that approximately 15 thousand people and 60 thousand livestock have been drinking these spring waters, around the Tendürek volcano. The results of the fluoride analysis of the water samples are given in table 2. It shows that seven spring water samples taken from Doğubayazıt district (north of Tendürek volcano) are containing excessive amounts of fluoride, ranging from 6.5 to 12.5 ppm. The fluoride concentrations in five water samples collected from Çaldıran district(south of Tendürek volcano) are ranging from 2.0 to 7.5 ppm. Whereas the water samples collected from Sarısu (Süphan volcano) and Tatvan (Nemrut volcano) districts are having fluoride levels, ranging from 0.2 to 0.7 and, from 2.0 to 5.0 ppm, respectively. It has been reported in the study of hydrogeology of the spring waters with high fluoride content from the surroundings of Tendürek volcano, that high fluoride waters are $NaHCO_3$ type, and low fluoride waters are Ca-$MgHCO_3$ type. Fluoride which might be transported by fumeroles or escaped from devitrified lavas, could be held on the surface of some minerals and then exchanged with OH of underground waters with high pH. At the end of this hypothetic process these waters might be discharged as high-fluoride water at the foothills of Tendürek volcano, (Oruç et al, 1975).

The results in table 1. indicate that some of the spring waters used for drinking and irrigation purposes around the Tendürek volcano contain very high amounts of fluoride to be toxic to the human beings and animals. It has been reported that in many parts of the world, chronic fluoride intoxication in human and animals can result from endemic exposure usually to drinking water containing excess of fluoride (Eagers, 1969, Fluoride and Human Health, 1970). In addition to the dental fluorosis in the local population the occurance of mottling, staining and excessive wearling of permanent teeth of the

Table 2. Fluoride content of some spring waters in the Eastern Anatolia

| Location | Fluoride ppm | Location | Fluoride ppm |
|---|---|---|---|
| Doğubayazıt | | Çaldıran District | |
| Gökçekaynak Kaynağı | 11.7 | Aşağımutlu Köyü | 7.5 |
| Girnevik Kaynağı | 12.5 | Alkaya Köyü | 5.0 |
| Celâl Köyü(Bataklık) | 5.0 | Soğuksu Deresi | 2.5 |
| Kanikork(Doğubayazıt İçmesuyu tesisleri) | 9.2 | Alakaya Demircik Mah. | 2.5 |
| Topçatan Kaynağı | 8.0 | Bendimahı Çayı | 3.2 |
| Üçgöze Köyü Kaynağı | 6.5 | Mutlu Köyü | 4.3[x] |
| Altıntepe Kaynağı | 12.0 | Turşık Sazlığı | 1.9[x] |
| Süphan Sarısu District | | Nemrut, Tatvan District | |
| Köseler Köyü | 0.7 | Nemrut crater(10°C) | 5.0 |
| Çaputlu Köyü | 0.5 | Nemrut crater(4°) | 5.0 |
| Sarısu Bucağı | 0.2 | Tatvan Kıyıdüzü | 2.0 |
| | | Üçgöze Kaynağı | 6.5[xx] |

x Oruç et al (1975).
xx Girgin (1975).

cattle and the sheep was observed during the investigation of the endemic areas, around the Tendürek volcano (Oruç, 1976). Aysan and Aksoy (Oruç, 1972) reported the fluorosis in livestock at Doğubayazıt. Leloğlu (1975) also indicated the fluorosis in cattle and sheep in some of the villages in Doğubayazıt. Mottled enamel in the local population from an endemic fluorosis area in Doğubayazıt, has been reported by various investigators (Baydaş, 1972, Gülhan and Mermutlu, 1973, Oruç, 1973, Oktay, 1975). It was associated with the excessive amounts of fluoride in drinking waters used in these areas. Şendil and Bayşu (1973) reported dental stains and abrasions and intermittent lameness in sheep, cattle, water buffaloes and some horses in the villages of Muradiye-Van and Doğubayazıt-Ağrı. They also indicated that "similar disorders including mottling, staining and abrasions, arthrosis of joints and stiffness in walking were observed in human beings from the same areas". They reported that "the measured fluorine concentrations in seven water samples obtained from Muradiye-Van ranged from 5.70 - 15.20 mg/lt., and six water samples from Doğubayazıt-Ağrı, were 10.26 - 12.54 mgçlt.".

The chronic toxic effects of fluoride on the skeletal system have been described from certain geographical regions of the world where drinking water contains excessive quantities of natural fluoride. This form of chronic intoxication was first described in India from the state of Madras as early as 1937, (Fluorides and Human Health). Öztopçular (1975) has been extensively studying on the chronic toxic effects of fluoride on the skeletal system. He indicated that quite distinctive and characteristic bone changes and skeletal abnormalities have developed in some of the local people in Doğubayazıt district.

## Recommendations

1) Defluoridation of the water is possible by adding some chemicals such as $CaCl_2$, $AlCl_3$ etc. which make rather insoluble complexes with fluoride, or by passing the high fluoride containing water through an anion exchanger, such as alumina treated with HCl (Girgin, 1975). But under the prevailing social and economical conditions, it seems quite difficult to establish such a system and espcially to maintain it running properly.

2) A good quality water should be supplied for the human beings and livestock, as soon as possible.

3) If economically feasible, dilution of fluoride-

rich spring waters with other suitable waters down to a safe level (1.5 ppm) is recommended to prevent the hazardous effects of fluoride on human and stock health and also to avoid the contamination of the soils and the pastures.

## BIBLIOGRAPHY


BAYDAŞ, S. (1972). Doğubayazıt köylerinde fluorosis. Dentoral. Cilt IV, Sayı: 18, Kasım-Aralık.

BEAR, F.F. (1975). Toxic elements in soils. The, 1957, Yearbook of agriculture. The USDA p. 167-168.

BREWER, R.F. (1966). Fluorine in Diagnostic criteria for plants and soils. Edited by H. D. Cahpman, Univ. of California. Division of Ag.Sci., USA.

EAGERS, R.Y. (1969). Toxic properties of inorganic fluorine compounds, Elsevier Pub. Comp. Ltd., London.

Fluorides and Human Health (1970). World Health Organization Monograph Series No. 59, Geneva.

GARNER, R. J. (1963). Fluorine, Veterinary toxicology. Second edition. The Williams and Wilkins Company. Baltimore. p. 83-91.

GİRGİN, I. (1975). İçme sularının fluorürden kurtarılması. Ankara Üni. Fen Fakültesi. Yüksek Lisans Tezi. TBTAK Proje No. TBAG-166.

GÜLHAN, A. ve MERMUTLU, B. (1973). Relations entre la fluorose endemique et la frequence de la caria. İstanbul Üni. Dişhekimliği Dergisi, 7, Sayı: 2, S: 130-140.

HOBBS, C.S. and et al (1954). Fluorosis in cattle and sheep. Bulletin No. 235, The University of Tennessee Agricultural Experiment Station.

JONES, W.G. (1977). Fluorosis in dairy cattle. Papers and Articles The Veterinary Record. 100: 84-89.

LELOĞLU, N. (1975). Personal Communication. Atatürk Üniversitesi, Ziraat Fakültesi, Erzurum.

MacINTERE, W. H., WINTERBERG, S. H., THOMPSON, J.G. and HATCHER, B.W. (1942). Fluorine content of plants.Ind. Eng.Chem. 34: 1469-1479.

MITCHELL, H. B. and EDMAN, M. (1945). Fluorine in soils, plants and animals. Soil Sci. 60: 81-90.

MITCHELL, H.H. and EDMAN, M. (1952). The fluorine problem in livestock feeding. The commonwealth Bureau of animal nutrition. Nutrition Abs. and Reviews, 21.4.

OKTAY, C. (1975). Doğubayazıt yöresi doğa sularında fluorür içeriğine bağlı olarak 5-12 yaş grubundaki çocuklarda dental yaş ve kemik yaşı üzerine bir araştırma. Atatürk Üniversitesi,Dişhekimliği Fakültesi~~(Unpublished)~~. In this issue.

ORUÇ, N. (1972). Ağrı ili Doğubayazıt köylerinde görülen Fluorozis vakası ile ilgili ön rapor. Atatürk Üniversitesi Yayım Müdürlüğü. Rapor No. 250.

ORUÇ, N. (1973). Doğubayazıt kazası ve bazı köylerinde kullanılan sularda fluorür konsantrasyonu ve önemi. Atatürk Üniversitesi Zir. Fakültesi, Ziraat Dergisi Cilt: 4, Sayı: 1.

ORUÇ, N., ALPMAN, N., Karamanderesi İ.H. (1975). Hydrogeology of the spring waters with high F content from the surroundings of Tendürek volcano. Bulletin of the Geological Society of Turkey. V. 19, 1-8 Feb.

ORUÇ, N. (1976). Van Gölü çevresindeki bazı doğa sularında fluorür konsantrasyonu ve önemi. Atatürk Üniversitesi, Ziraat Fakültesi, Ziraat Dergisi. Cilt:7, Sayı:1.

ÖZTOPÇULAR, M. (1975). Doğubayazıt yöresinde endemik fluorosis. Atatürk Üniversitesi Tıp Fakültesi, Nöroloji Servisi ~~(Unpublished)~~ In this issue.

ROBINSON, W. O., and ENGINGTON, G. (1946). Fluorine in soils. Soil Sci. 61: 341-353.

SHUPE, J.L. and OLSON, A.E. (1971). Clinical aspects of fluorosis in horses. J.Amer.vet.med.assoc. 158: 167-174.

ŞENDİL, Ç. ve BAYŞU, N. (1973). İnsan ve hayvanlarda Ağrı ili Doğubayazıt ilçesi köylerinde görülen flor zehirlenmeleri ve bunu Van İli Muradiye İlçesi köylerinde de saptanmamızla ilgili ilk tebliğ, Ankara Üniversitesi Veteriner Fakültesi Dergisi, Cilt : XX, No.4.

YENAL, O. (1970). Türkiye maden suları. İstanbul Üniversitesi Tıp Fakültesi Hidroklimatoloji Kürsüsü yayını.

# EFFECT OF HIGH FLUORIDE CONTAINING DRINKING WATER ON SKELETAL AND DENTAL AGE

by Dr. Cengiz Oktay
Associate Professor in Dentistry
Atatürk University
Erzurum - Turkey

## Introduction

Fluorides have benn used in the prevention of dental caries in U.S.A. and in some western countries. Fluorides seem to have positive effect in preventive dentistry, if it is applied systematically in the development stages of teeth. The accepted optimal dosage for systemic application is in between, 1-1.5 ppm., in another word, 1 mgm per liter. If applied in optimal doses, it does not have any undesirable effect either on teeth or on bones. However, if it is exceeded for a long period of time, then one may expect certain changes in dental and skeletal tissues. Skeletal changes may also give rise to concomitant neurological signs. This aspect of the problem will be discussed by Dr. Öztopçular, in detail.

The aim of this study is to find out the difference between dental age and skeletal age, among the children between the ages 3 - 14, if there is any.

The first serious research work related with fluorosis had been, presented in Turkey, by Ata(1). In the province of Isparta, which is situated in the south-west of Turkey, drinking water of a certain area contains fluoride upto 4.03 ppm. Ata(1) is the first research worker who points out the relevant dental problems related with the high fluoride content of drinking water in Turkey.

Another Turkish research worker Usmen(2), in a field study, related with D.M.F. values in the villages of

Ankara among the children in elementary schools, found that the fluoride contents of drinking waters are not higher than 0.6. ppm. As you may well visualize this level is below the optimal.

Gülhan and Mermutlu[3] also in a field study in Doğubayazıt tried to correlate D.M.F. rates with the fluoride content of drinking water and found the level as high as 13 ppm.

Oruç[4], in his evaluations found varying levels of fluorides in drinking waters of Doğubayazıt region, the values vary from 6.5 ppm upto 12.5 ppm. After this brief introduction, I would like to present some relevant data related with this particular problem.

Fluoride is the most electronegative element of whole elements. Since it is the most electronegative element, it is found in the form of compounds on the earth surface. Fluoride compounds are mostly found in inorganic forms, however, organic fluoride compounds are not rare.

There are various ways through which we take fluorides, such as drinking water, eating plants and marine products and inhalation. The detrimental effect of fluorides is in most of the cases through drinking water. In the development stages, fluorides taken in high dosages deteriorate the dental and skeletal structures, forming fluoro-apatites in the mineralization phase of teeth and bones. The effect of fluorides is seen not in primary dentition, but in permanent dentition. So, the result is a typical browny appearence of permanent dentition: particularly in the areas taking ultraviolet rays. The appearance of the teeth seems to be hypoplastic. However, most of the teeth are caries free and D.M.F. values seem to be less than the values of normal population(Mermutlu, Gülhan)[3].

The long term usage of high fluorides through drinking water has multiple effect on human health. In extreme cases, bone and joint deformities and some neuropathological symptoms can be observed.

Fluorides are absorbed through gastro-intestinal tract. 80 percent of the fluorides taken orally are being absorbed through this route, compounds such as calcium fluoride and cryolite are absorbed on the level of 60 percent. It is a common belief that fluorides are absorbed by means of simple diffusion. If elements such as aluminium and magnesium are found together with fluorides in G.I. tract, then the absorbtion of fluorides decrease and accordingly these elements make compounds with fluorides which are less soluble. Fluorides may also be absorbed through respiratory tract. The absorbtion rate through this route seems to be faster than the G.I.tract. This type of inhalation hazards are seen particularly in the industrial areas where the air pollution is high.

It is particularly important to know where the absorbed fluorides are accumulated in the organism. Nickles(5) is the first research worker finding that the animal blood contains fluorides, Tamman(6) in 1888, showed that egg yolk, calf's brain and milk had contained fluorides.

Brandl and Tappeiner(7) applied sodium fluorides to a dog on the level of 0.1-1 gram daily for 21 months, at the end of this period, they found that the blood of the dog contained 0.14 equivalent gram, the musculature 1.84 equivalent gram and the liver of the animal 0.51 equivalent gram sodium fluoride. The accumulated amounts corresponded to 93,690 and 161 ppm fluoride in these tissues respectively.

Today, plasma is being accepted the most reliable tissue fluid in the determination of fluoride content of tissues. The fluoride content of crytrocytes and plasma show an unbalanced distribution. If volume is considered, erytrocytes contain 40-50 percent fluoride, in comparison to the plasma. However, Carlson, Armstrong and Singer(8) have showed that plasma contains 75.percent of fluorides of the whole blood. When hemocryt values change, the fluoride contents also change accordingly. However, in these situations plasma fluoride contents seem to be relatively stable. These three research workers believe that a regulation mechanism functions through the dilution of the resorbed fluorides with a large volume of extracellular fluid, through excretion and through bind- to the osseous tissues forming fluoro-apatites.

Singer and Armstrong[9] in their research work related with plasma content of fluorides have concluded that the plasma content of fluorides ranged in between 0.14 - 0.19 ppm in communities where the drinking waters contain 0.15 - 2.5 ppm fluoride, however,in communities where the drinking water contains 5.4 ppm fluorides, the plasma values has been found 0.26 ppm. These values indicate that the control mechanism described above, is effective to a certain level of fluoride. After a certain point, this mechanism seems to be uneffective. The values given above depend upon total fluoride content of the plasma. Most of the fluorides are bound to the albumins of the plasma. Taves[10] has demonstrated that a small amount was found in the ion-phase and in physiologic reactions these unbounded ions played an important role and were bound to calcium phosphate in the bones to form fluoro-apatities. Various research workers have showed that the 15 - 20 percent of the total fluorides were bound to the calcified tissues of the organism (11, 12).

Fluorides show a specific affinity to the osseous and dental tissues. The retention of fluorides in these tissues depend upon the amount of water taken daily, duration seems also to be an important factor in the retention process of fluorides. On tissue level, fluoride content of tissues varies from one person to another. Also fluoride contents of dental tissues vary, even if the dental tissue looks like a homogen type. This point has been shown by Weidmann and Weatherell[13], as far as we know, certain communities in India use drinking water up to a fluoride level of 20 ppm. In Turkey, in the Isparta region and Doğubayazıt regions, there are communities drinking high fluoride containing water upto 13 ppm. Climate seems also to be an effective factor in these ticular regions. During warm periods, daily water intake also increases, accordingly, fluoride retention of the skeletal and dental tissues vary.

Zipkin[14], has shown that there is a linear relationship between the amount of drinking water containing high fluoride level and the retention of fluoride by skeletal and dental tissues. Foods binding fluorides interfere with the absorbtion of fluorides through the gastro-intestinal tract, particularly, foods containing high level of calcium and magnesium interfere to the absorbtion. Fluorides bound with these elements are eliminated with stool.

Largent[15] has also demonstrated that fluorides taken between the ranges 1 - 20 ppm, are retained on 50 percent level in skeletal and dental tissues. The rest of the fluorides taken are eliminated through urinary excretion and stool. According to Largent, retention of fluorides in blood and in soft tissues doesn't take place for a long period of time. However, it may be possible to detect some retention in ectopic pockets. Exluding these unimportant retention areas, we may conclude that the retention of fluorides occur solely in skeletal and dental tissues.

Binding of fluorides to skeletal and dental tissues.

In invitro studies, it has been shown by Neumann[16] and et al[17] that fluorides in ionic phase interchange with bicarbonate groubs on the crystal surfaces of bones. Schein[18] has found that the binding of fluoride to the crystal surfaces of bones and dental tissues was highly correlated to the PH of the medium. The same research worker claims that there is an analoguous competition between hydroxyl groups of hydroxy-apatite crystals and fluoride ions. Schein in an invitro study has also shown that the binding capacity of enamel depended upon PH, fluoride concentration and the treatment duration in the medium.

Neumann and Neumann[16] have developed a model explaining how fluoride bound to apatite-crystal. According to these research workers, in the first stage, fluoride ions enter the medium enveloping the crystalite, either through an ion exchange or through an exchange with a polarized molecule taking place in the medium. Inn the second stage, a heteroionic exchange takes place between fluoride ions, bicarbonate and hydroxyl groups on the bone and enamel surfaces. Iso-ionic exchange also takes place between the fluoride ions in the medium and fluorides bound previously to the crystal surfaces. Brudewald[19] in 1962, has also shown that on enamel surfaces an isoionic exchange of fluorides penetrate to the empty areas within the crystal; this corresponds to the remineralization stage. The ratio of calcium and phosphate in hydroxy-apatite crystal seems to be not effected by the entrance of fluoride ions exchanging with hydroxyl groups. However, in animal experiments Zipkin[14], Neumann and Neumann[16] have shown that citrate level decreased down to 40 percent by the introduction of fluorides. This decrease seems to be related with the binding of fluorides to the crystal surfaces. It is the opinion of these researchers that the decrease occurs as a result of an inhibition of citrate binding capacity of the crystals in a medium containing high concentration of fluoride ions. This inhibition may be a result of enzyme inactivity occuring through fluoride ions.

The binding potential of skeletal and dental tissues is highest particularly, in the developmental stages of these tissues. After that time, the capacity decreases significantly.

Bauer, Carlson and Linguist[20] with phosphate 32 and calcium 45 radioactive elements have demonstrated that binding capacity of the newforming crystals seems to be higher than the remineralization of crystals which have previously formed up. In the formative stages, the fluoride binding capacity of newly formed apatite crystals and remineralization capacity(ion exchange capacity) seem to be at a highest level. It is for this reason that fluoride retention is at the highest level in the development stages of bones and dental tissues. This point has been shown by Zipkin[14], McClure[21], Philips and Suttie[22] in animal experiments. The same research workers have also shown that the fluoride binding capacity decreases later. Spongiose and compact bones show also different fluoride binding capacities. Parkinson[23] in 1955, in animal experiments with F 18 has shown that fluoride retention capacity of spongiose bones was higher than the compact bones.

The binding of fluoride to dental tissues resembles more or less the osseous tissues. However, there are certain aspects which differ from bones. Remodelling process taking place in bones is lacking in enamel, since this tissue completes its cellular activity after a certain period of time. In addition to this point, mature dentin and enamel show a great resistance to ion exchange which bone tissue does not show. This may be explained through ion exchange dynamics, in experiments with radioactive isotopes, it has been shown that the ion exchange ratio between bone and dental tissues was 6 to 1, for this reason, ion exchange after the maturation in dental tissues is less than the skeletal tissues. This holds true for fluoride exchange in dental tissues. This point is very important in the explanation of our findings in the comparison of dental age and skeletal age.

As I mentioned previously, the fluoride binding capacity is highly correlated with the developmental stages, Wallace and Durbin[24] in an animal experiment have shown that the fluoride retention of the rat molars was less than the incisors which show continuous regeneration. On the other hand, in the formative stages, these teeth do not show difference in terms of fluoride retention. These findings indicate once more that fluoride binding capacity of the dental tissues is related with the developmental stages.

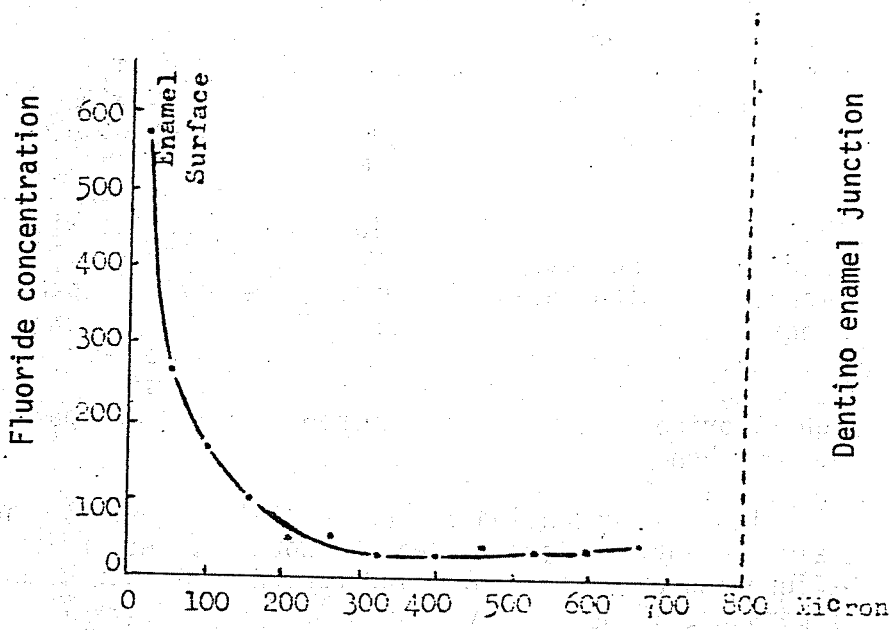
If we think about the timing, in terms of the fluoridation. The best period seems to be the developmental periods of teeth, in terms of the caries prevention.

Fluoride retention possibly takes place in three stages in teeth.

The first stage which corresponds the organic matrix formation. Fluorides are found as ion in the media enveloping the matrix which has already been formed.

The second stage corresponds to the calcification period, at this stage, fluoride ions are bound to newly formed calcified areas. At the third stage, calcification has already been completed, fluoride binding takes place solely on the marginal areas. Fluoride retention on enamel seems to be at a highest point at the surfaces, the inner parts of the enamel holds less fluoride, this may be explained by ionic penetrance of the fluoride which seems to be less capable to penetrate deep into the enamel. According to Weatherell and Hargreaves[25] the fluoride distribution of enamel is as shown in the following graphic:

DISTRIBUTION OF FLUORIDES ON THE LABIAL SURFACE
OF PERMANENT UPPER INCISOR




Thickness of enamel
from Weatherell and Hargreaves

The fluoride retention in dentin is twice that of the enamel and the binding capacity is also higher in developmental period of dentin than the mature dentin. The fluoride binding capacity of dentin is on the highest level at the calcification period, this capacity is particularly higher where the odontoblastic activity is utmost. This is why, fluoride retention capacity is higher in a newly formed secondary dentin than the primary dentin which has already been matured. Cementum shows higher fluoride retention capacity in camparison to dentin. This higher retention capacity may be due to the thickness difference of these two tissues and concominant easiness of ion penetration to the cementum.

Flour retention and the blood supply

There seems to be a high correlation between the blood supply of skeletal tissue and fluoride retention. In a series of research work completed by Volker, Bibby and Sognaes(26), fluoride binding capacity of maxilla has been found higher than that the long bones such as femur. These research workers attribute this difference to the blood supply of the bones. According to them, where the blood supply is higher, there, retention occurs at a high level. Experiments in animals with F 18 have showed that mandibula, epephysis and diaphisis of bones have significantly high level of fluorides, compared with the other bones and metaphysis. Wallace and Durbin(24) attribute this high level of fluoride retention capability directly to the vascularization. This is why, the subperiostal areas of bones show higher fluoride retention. According to Weatherell(25) mandibular exestoses show high fluoride content in comparison to the other parts of the same bone.

Fluoride retention of the dental tissues has been evaluated by various research workers. According to McClure and Likins(21) fluoride content of mature dentin of persons taking 0.1 ppm fluoride, fluoride level is 332 ppm. In persons taking 7.6 ppm this level jumps upto 1968, ppm.

Jackson and Weidmann(27) in series of research work have concluded that persons drinking water with different level of fluoride content showed different level of fluoride retention in the dentin of their teeth. According to their findings, there seems to be a linear relationship between the fluoride content of dentin and the fluoride level of the drinking water. Fluoride retention in enamel is particularly high in the period of amelogenesis, as soon as amelogenesis is completed, a satiation to fluoride becomes apparent. This holds true for the bones, as the maturation takes place. Suttie and Philips(22) have shown that satiation could be reached quickly when high dosage of fluorides was applied to the animals such as 500 ppm. The satiation occurs first in the marginal areas of bones and enamel. One may raise the question if fluoridation ceases when that happens, Savchuck and

Armstrong[28] in animal experiments have showed that after the cessation of fluoridation within 40 days 10-15 percent of fluoride were eliminated. However, after this time, eliminations stopped. McCann and Bullock[29] have also shown that fluoride elimination was minimal even if fluoridation was ceased.

## Fluoride, calcium and phosphorus

Various research workers have shown that there seemed to be close relationships among these elements. The distribution of fluorides shows a close relation to the distribution of calcium. Armstrong, Carlson et al[8] are in the opinion that calcium may have a role in the binding of fluoride with plasma proteins. Calcium interferes with fluoride absorption by establishing calcium fluorides. In this way, the organism is protected from the hazardous effect of fluoride[30,31].

Under normal circumstances or in slight calcium deficiencies, fluoride intake from 7-14 mgm seems to have no deleterious effect on the calcium balance[32].

Fluorides nowadays has been found to be an effective means for the treatment of osteoporosis, it seems to have a positive effect on calcium balance[33], when applied in excessive dosages.

In the last trimester of the pregnancy the serum fluoride level shows a drastic decrease. This can be attributed to the mineralization process occuring in fetus. As we mentioned above where the calcification is high, there, fluoride retention is also found to be high. Gedalia et al[34] if fluorides are taken within physiological limits, they do not have any effect either on serum calcium or phosphate level. This is also true for the salivary secretion, (Knappwost-Tochtermann)[35].

Phosphates seem to have positive effect on the absorption of fluorides, Bixler Muhler[36] are in the opinion that this effect is a reverse effect of phosphates to calcium which inhibits the absorption.

On enzyme level, animals showing intense fluorosis serum alkalen phosphates activity diminishes significantly Riektniece, Myers et al(37). As is Well known alkalen phosphatase is a magnesium dependent enzyme and in bone tissue it is also an indicator of osteoblastic activity. Fluoride may possibly interfere with the absorption of magnesium by establishing insoluble compounds in the intestines and by this way a magnesium deficiency may arise.

It is also quite important to review some aspects of collagen biosynthesis in chronic fluoride intoxication. Zipkin, Whedan and Peck(38) have shown that the collagen biosynthesis in the calvaries of the rats had been significantly depressed in animals drinking 10 ppm fluoride for 10 months. This depression was apparent on tissue level. In vitro bone culture studies with fluorides, application of 10 - 20 ppm fluoride to the media cause an enormous depression in collagen biosynthesis which can be demonstrated through C 14-Proline binding.

Golub, Glimcher, Goldhaber(39) et al have also shown that even if in the media less than 0.2 ppm fluoride was found, collagen break down also occured.

In the tissue fluids fluoride content is 0.1-0.2 ppm and in soft tissues the fluoride concentration is approximately 1 ppm. In fluorosis these figures will definitely be increased and collagen break down will accordingly change.

In fluorotic cases roentgenological studies have also been done. Schlesinger(40) et al have taken X-rays from the children drinking 1.2 ppm fluoride. At this level, they could not find any pahtological change either on bone age or on bone density in comparison to the control groups. McCauley and McClure(41) in terms of bone age also were unable to find any difference between the control group and the group which consisted of the children from 7 years of age upto 14 years of age, drinking water with a fluoride content of 6.2 ppm. According to these research workers, carpal index values show no difference between the control group and the group in question.

In addition to these findings Geever(42), Morris, Stevenson and Watson[44] have found that bone density doesn't change upto 4 ppm of fluoride intake, however, above this level they found changes in bone tissues.

From the dental standpoint Zipkin[14] in his evaluations were unable to find extreme cases of malocclusions and periodontal pathology in comparison to the control groups.

Here I would like to summarize some of the findings of other research workers.

1. High level of fluoride intake for long durations yields to an hypoplastic appearance of the enamel which we call "mottled enamel". Appearance of mottled enamel may also be influenced by other factors which are directly related with nutrition.

2. In severe cases of fluorosis, appearance of the enamel is typically hypoplastic, continuity of the enamel is disturbed.

3. The outer 1/3 of the teeth showing mottled appearance is hypocalcified and shows less radio density than normal teeth. These areas are less acid soluble and show high penetrance to dyes.

4. In animal experiments, it has been shown that the application of high dosage of fluorides with drinking water, resulted in morphological changes in teeth particularly on the fusion lines of the enamel.

5. Histologically in enamel, ameloblasts show some sort of disturbances which are manifested with the retardations both in apposition and mineralization.

6. There seems to be a high correlation between fluoride intake and calcium and phosphorus intakes, cardinal symptoms of fluorosis seem to be allievated with the application of calcium and phosphorus in combination.

7. Chemical analysis of the mottled enamel has shown that even if the organic content of the enamel shows a relative increase,in comparison to the calcified enamel, calcium and phosphate ratio stays unchanged.

In our research it is of prime importance to consider the malnutrition which directly effects the skeletal age.

At present, there are a couple of methods in the determination of malnutrition. Gomez(45) has classified malnutritions in three categories in which the first group corresponds to latent malnutrition and the third group corresponds to severe malnutrition. One of the methods of determination of malnutrition is through the ratio of hydroxyproline and creatinin in urine, the ratio is multiplied with height and weight of the children. This is a highly precise method for the evaluation of malnutrition(46). Another method particularly valid in children upto the age of seven years is, the circumferential measurement of the arm and the head of the children. The ratio of these two measurements, yields to highly precise criteria in the evaluation of malnutrition(47).

## Material and method applied in the research

The research has been completed with 60 girls and 69 boys in Doğubayazıt region where fluoride content of drinking water is upto 13 ppm. The age range is between 3-14 years of age. We were unable to determine the actual chronological ages of these children, because of socio-cultural problems. These people experience routine X ray of the right hand wrist and lateral mandibular radiographic view of each child is taken. For this purpose we used S.K.-150 teleradiographic unit. In order to avoid the superposition of the dental structures in mandible, we applied modified lateral graphy technique. Roentgenologic factors were manipulated as follows for this technique.

| | |
|---|---|
| K.V.P | 65-70 |
| m Amp. | 15 |
| Film - Tube distance | 160 cm |
| Exposure time | 2 sec |
| Film type | Dupont Cronex |

For wrist graphics the roentgenologic factors were manipulated as follows:

| | |
|---|---|
| K.V.P. | 50 |
| m Amp. | 15 |
| Film - Tube distance | 60 cm |
| Exposure time | 0,6 - 0,8 sec |
| Film type | Dupont cronex |

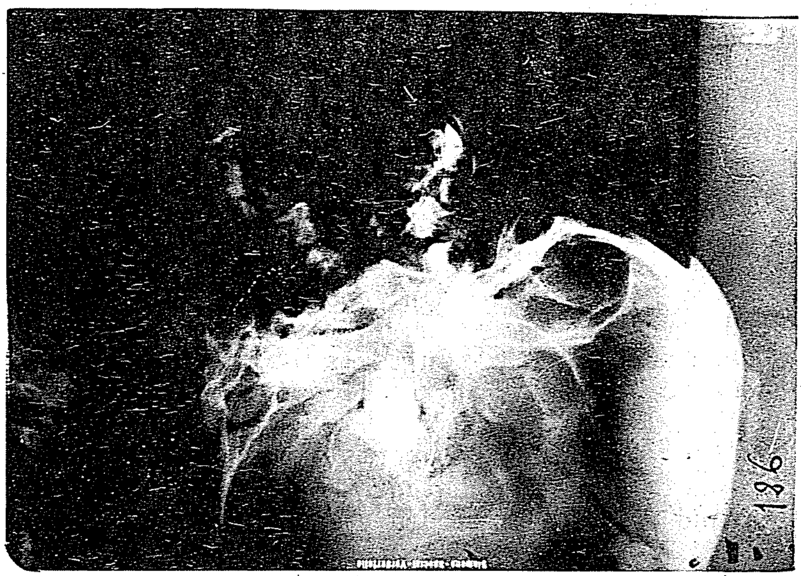
In the plates you see the lateral view of the mandible and the wrist X rays of one of these cases. (Case No. 126, Page 70)

In the evaluation of dental age, we have used norms developed by Moorress, Fanning and Hunt(48), these norms have been developed according to the developmental stages of the permanent teeth. In terms of crown, root formation and apical closure of teeth.

Now you will see the norms at the following pages for the mandibular permanent canines,premolars and molars of males and females developed by Moorress et al.(pp.71-77)

Evaluations of the carpal index values in years and months were completed. For this purpose, we have used the Atlas of Greuclich and Pyle(49), in which all developmental stages of carpal and metacarpal bones are shown. In the following Table 1. you see developmental periods of these bones. (page 78)

In order to determine the nutritional status of the children, we have used the method developed by McLaren(47), this method is also recommended by the World Health Organisation. In this method, the only problem seems to be the age of the children, since the method is valid only for the children upto the age of seven. In 34 children cosisting of 20 boys and 14 girls upto the age of seven, we have used this method with a band having milimetric measurement scale, we have measured the circumferance of the arm and the widest part of the head. For the measurement of the arm, processus acromion of scapula and olecranon of ulna have been found and in between point of these two processes, measurements were made with a band measurement without pressing to the soft tissues underneath. Head measurements have been made covering the supra optical tuberance of os frontalis and the apparent tuberance of os occipitalis. The ratio of



Case No. 126

these two measurements gives a rather precise result for the determination of nutritional status. According to McLaren[47], if the ratio is 0.310 and above, this means that the child's nutritional status is normal, if the ratio is in between 0.310 - 0.280 than child has a mild protein malnutrition, if ratio is below 0.279 and down to 0.25 child has a moderate malnutrition, if the ratio is below 0.249 than the child has a severe malnutrition.

## Findings for malnutrition

In the following Table 2. head and arm measurements and their ratios have been shown. (page 79)

## Findings for dental and osseous ages

Evaluations of the dental and osseous ages of the children drinking high fluoride containing water in Doğubayazıt region have been shown in the Tables 3, 4, 5 and 6 (pages 80-93)

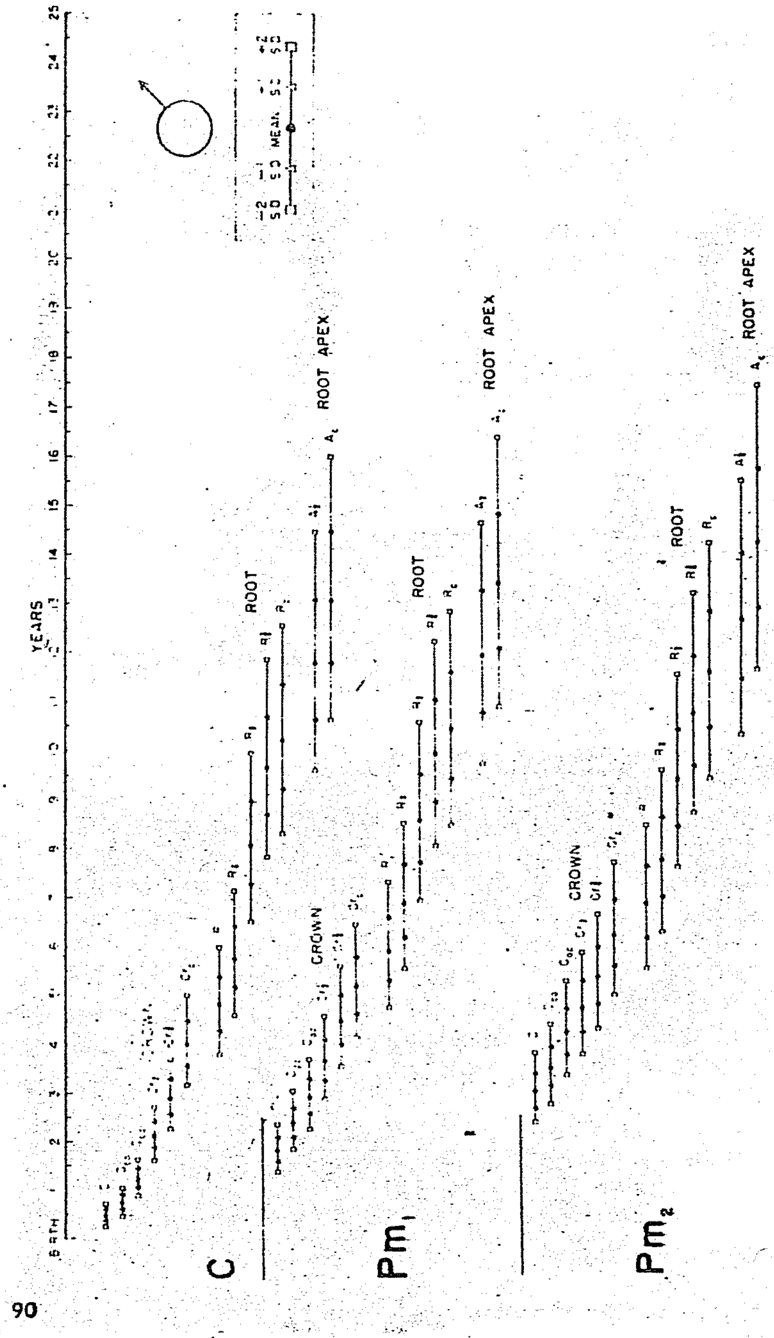
## Statistical analysis of the findings

Chi-square test has been applied in order to determine if any significant difference between dental age and skeletal age, exists of the studies group of boys (60) and girls (69) separately, with =0.0005, the observed t value for boys is 8.81 which highly exceeds t = 3.46 of the table value. This finding indicates that there is a statistically significant difference between skeletal development and dental development of boys. Dental development of boys highly exceeds the carpal and metacarpal developments.

The same procedure has been repeated for females (69).

The t value for females has been found as t = 14.08, with =0.0005 significance level, this value highly exceeds the table value. This indicates that the dental age in girls also exceeds the skeletal age.

Another statistical analysis has been made in order to determine if any difference exists between the dental




Norms for the Mandibular Permanent Canines, Premolars and Molars of Males and Females Developed by Moorrees et al.

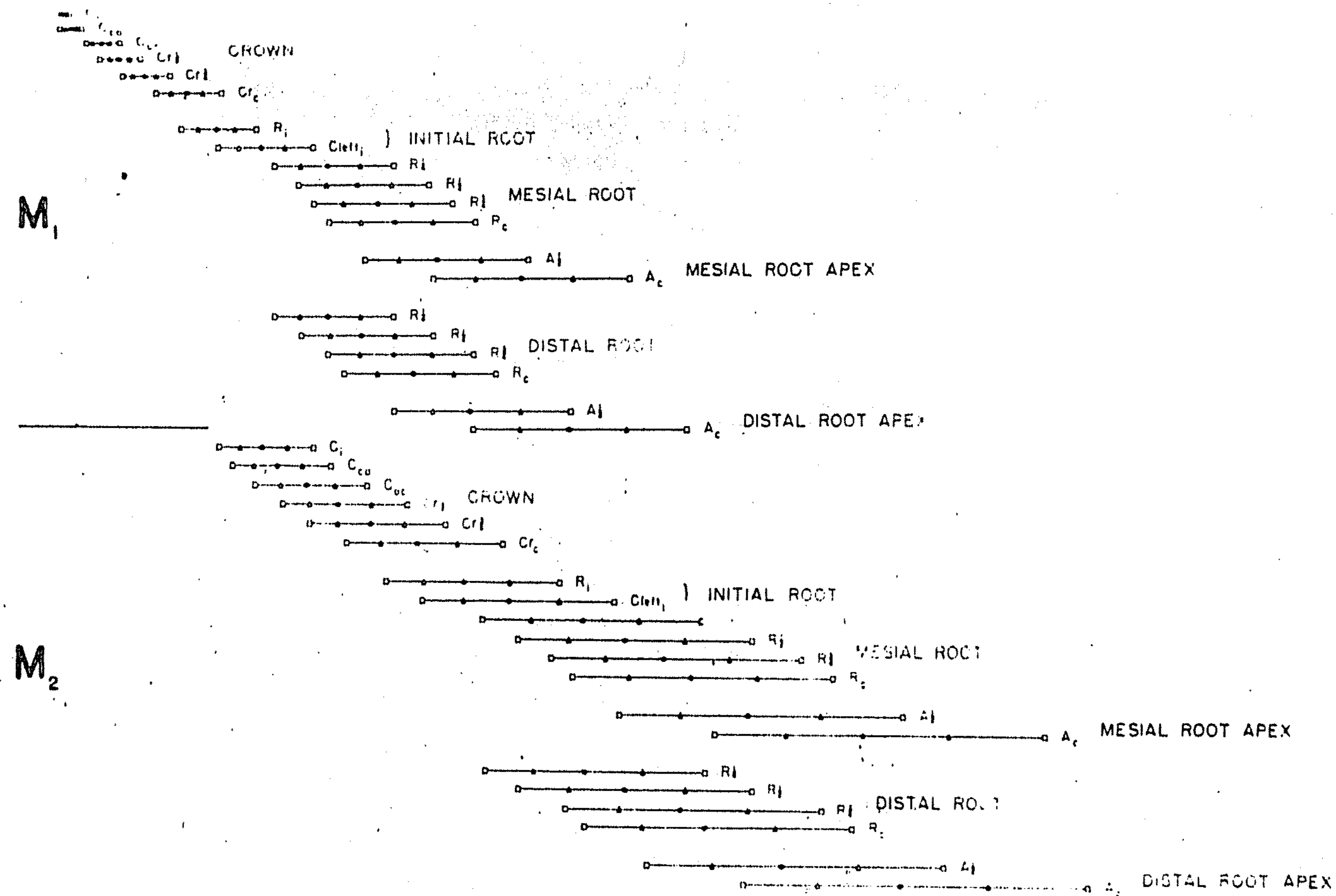



Figure 2-58. Norms of tooth formation of permanent mandibular canines, premolars and molars of males. (From Moorrees, C. F. A., Fanning, E. A., and Hunt, E. E., Jr.: Variation of formation stages for ten permanent teeth. J. Dent. Res., 42:1490–1502, 1963.)

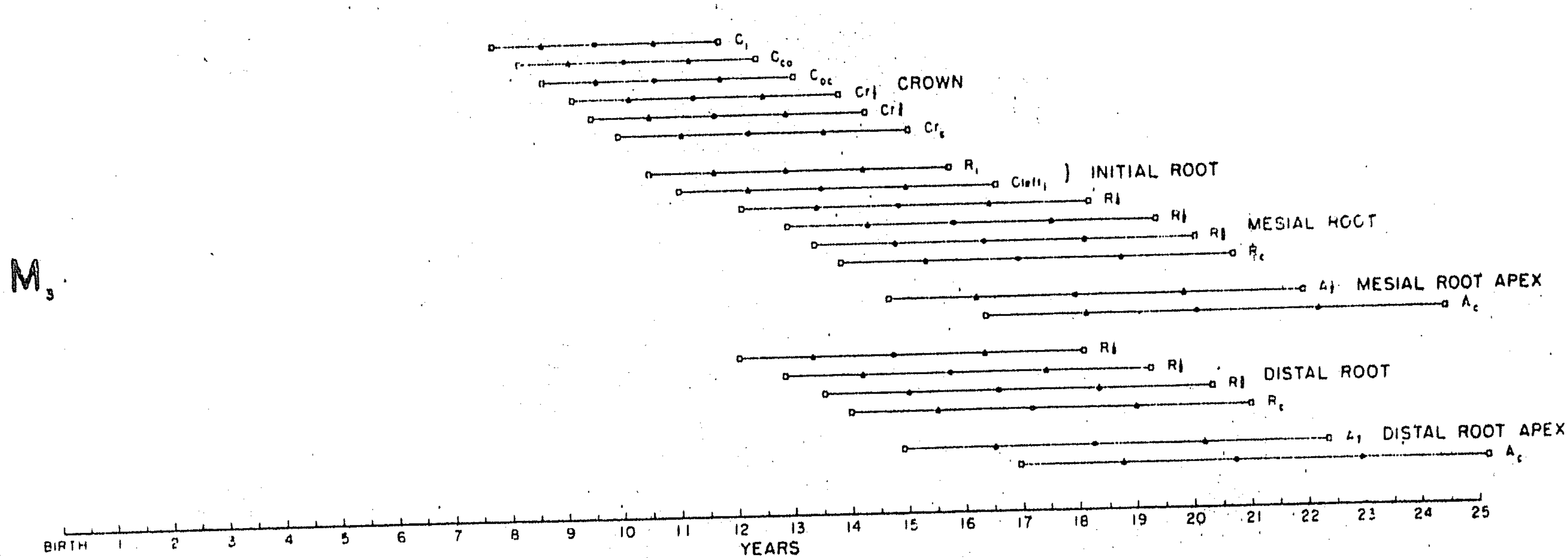
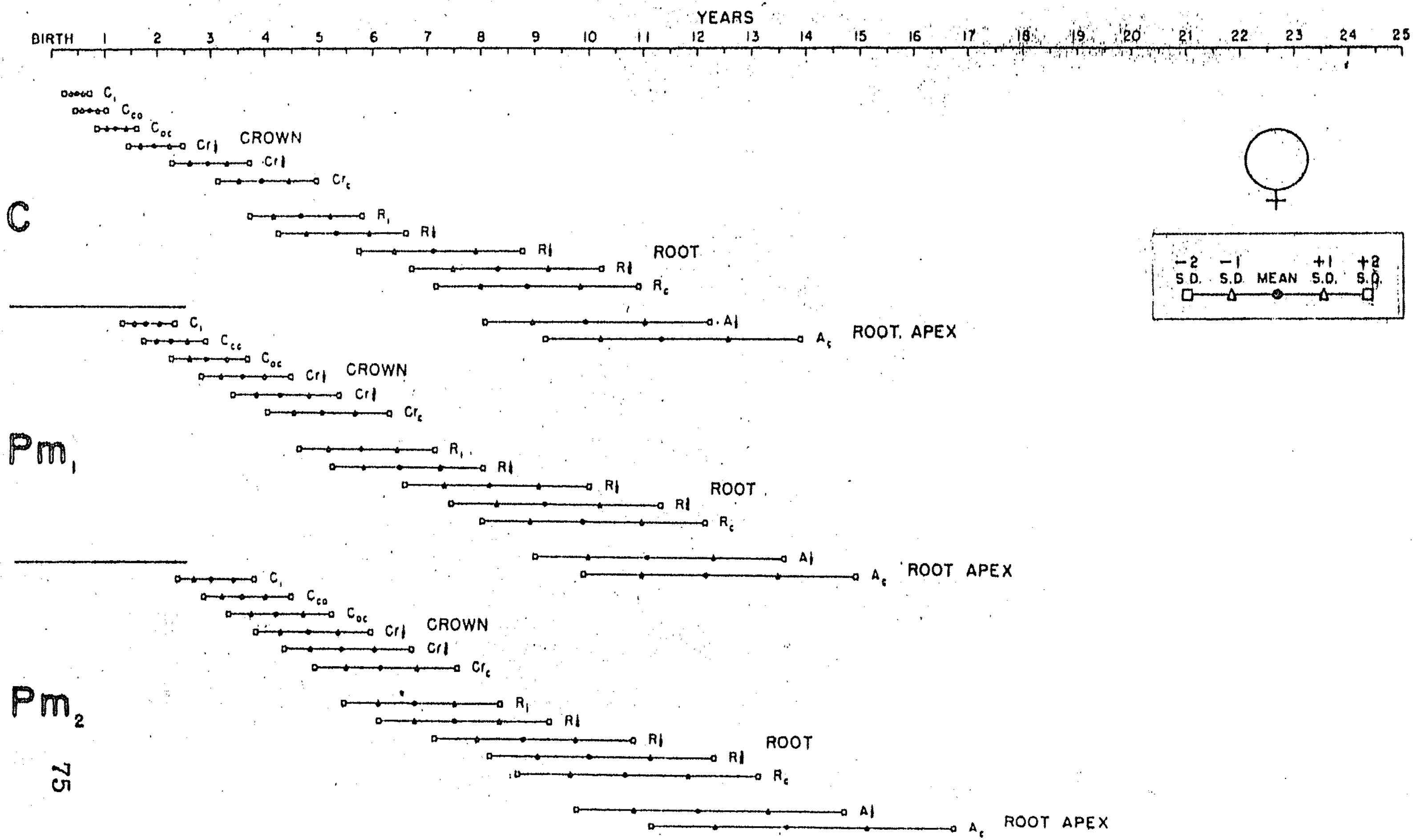



**Figure 2-58.** *Continued.*


YEARS
BIRTH
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
C
CROWN
ROOT
ROOT. APEX
Pm1
CROWN
ROOT
ROOT APEX
Pm2
CROWN
ROOT
ROOT APEX
−2 S.D.
−1 S.D.
MEAN
+1 S.D.
+2 S.D.

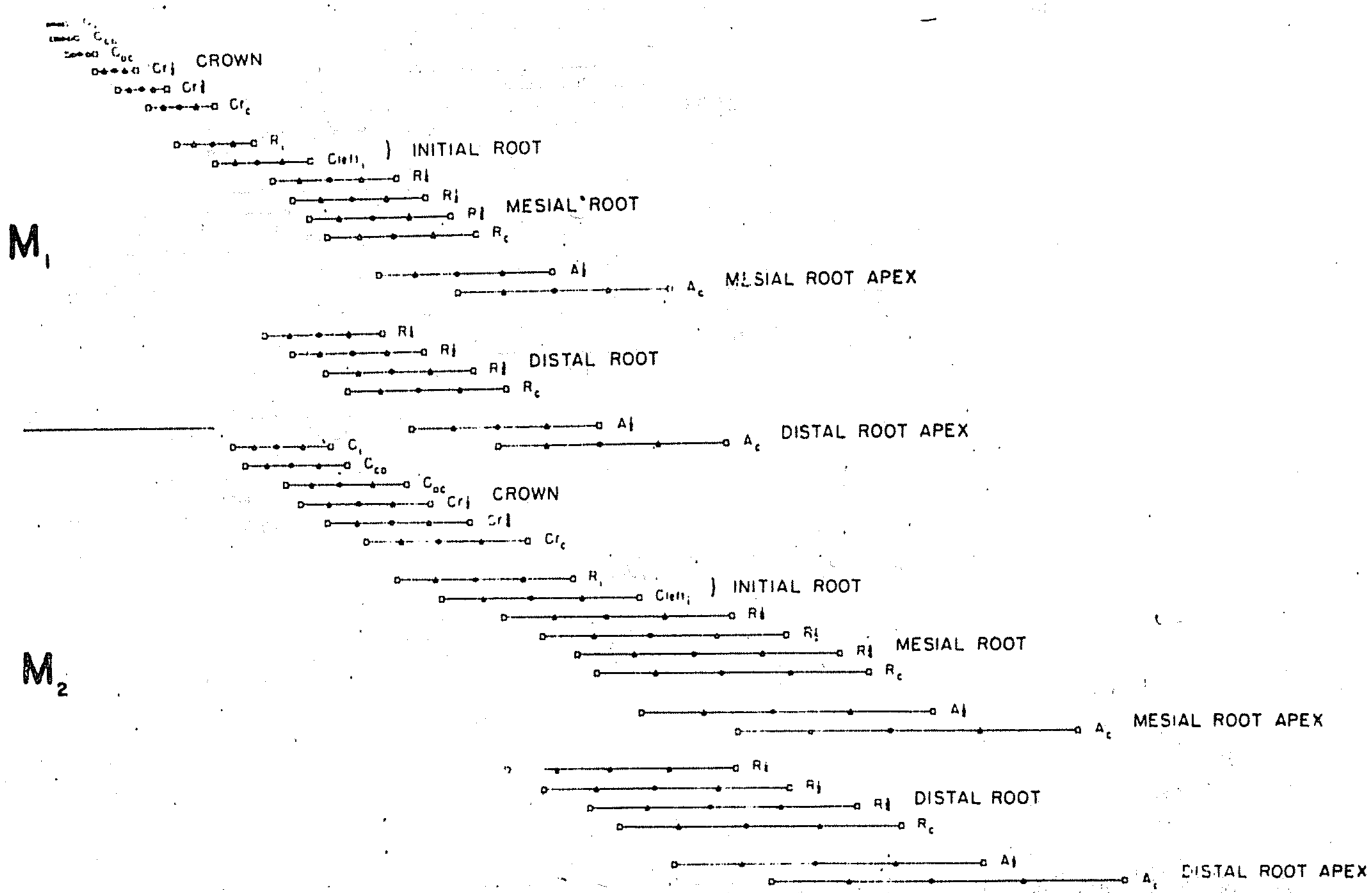



**Figure 2-59.** Norms of tooth formation of permanent mandibular canines, premolars and molars of females. (From Moorees, C. F. A., Fanning, E. A., and Hunt, E. E., Jr.: Age variation of formation stages for ten permanent teeth. J. Dent. Res., 42:1490–1502, 1963.)

M3

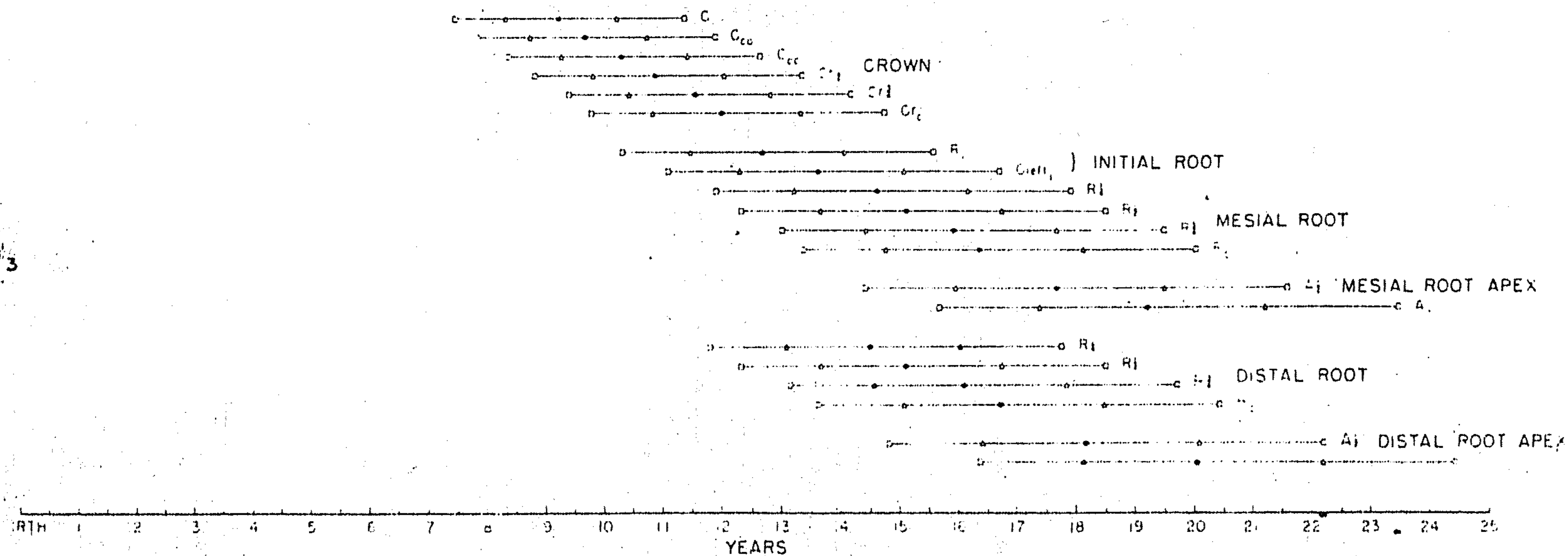



Figure 2-59. *Continued.*

TABLE I

BEGINNING OF AVERAGE OSSIFICATION PERIODS
IN CARPAL AND METACARPAL BONES FOR MALE
AND FEMALE CHILDREN

| BONES | BEGINNING OF OSSIFICATION | |
|---|---|---|
| | Female | Male |
| Capitatum | 2 months | 2 months |
| Hamatum | 2 " | 2 " |
| Radium Distal Epiphysis | 10 " | 1 year 1 " |
| 3-Proximal Epiphysis | 10 " | 1 " 4 " |
| 2-Proximal Epiphysis | 11 " | 1 " 4 " |
| 4-Proximal Epiphysis | 11 " | 1 " 5 " |
| II-Metacarpal Epiphysis | 1 year | 1 " 6 " |
| I-Distal Epiphysis | 1 " | 1 " 7 " |
| III-Metacarpal Epiphysis | 1 " 1 " | 1 " 8 " |
| IV-Metacarpal Epiphysis | 1 " 3 " | 1 " 11 " |
| 5-Proximal Epiphysis | 1 " 2 " | 1 " 9 " |
| 3-Medial Epiphysis | 1 " 3 " | 2 years |
| 4-Medial Epiphysis | 1 " 3 " | 2 " |
| V-Metacarpal Epiphysis | 1 " 4 " | 2 " 2 " |
| 2-Medial Epiphysis | 1 " 4 " | 2 " 2 " |
| Iriquetrium | 1 " 9 " | 2 " 5 " |
| 3-Distal Epiphysis | 1 " 6 " | 2 " 4 " |
| I-Metacarpal Epiphysis | 1 " 6 " | 2 " 8 " |
| 1-Proximal Epiphysis | 1 " 8 " | 2 " 8 " |
| 5-Distal Epipysis | 1 " 11 " | 3 " 1 " |
| 2-Distal Epiphysis | 1 " 11 " | 3 " 1 " |
| 5-Medial Epiphysis | 1 " 10 " | 3 " 3 " |
| Lunatum | 3 years 11 " | 5 " 6 " |
| Gross Multangulare | 3 " 11 " | 5 " 7 " |
| Small Multangulare | 4 " 1 " | 5 " 9 " |
| Navicularis | 4 " 3 " | 5 " 6 " |
| Ulna Distal Epiphysis | 5 " 9 " | 6 " 10 " |
| Adductor Pollicis Sesamoid | 10 " 1 " | 12 " 8 " |
| FROM PYLE | | |

TABLE 2

THE PRESCHOOL CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT REGION MALNUTRITION EVALUATION (MALES)

| No. | Approx. age in months | Villages | Circumferential measurements of head | Circumferential measurements of the right arm | Ratio of the circumferential measurements of arm the head | Deviation from the norms |
|---|---|---|---|---|---|---|
| 1 | 6 | Gökçekaynak (11.40 ppm) | 43.8 | 14 | 0.319 | 0.009 |
| 2 | 14 | " | 47.5 | 16.5 | 0.347 | 0.037 |
| 3 | 30 | " | 50 | 16.5 | 0.330 | 0.020 |
| 4 | 72 | " | 51 | 17 | 0.333 | 0.023 |
| 5 | 13 | " | 48 | 14.5 | 0.302 | 0.008 |
| 6 | 36 | " | 50 | 17 | 0.340 | 0.030 |
| 7 | 72 | " | 52.5 | 17.5 | 0.333 | 0.023 |
| 8 | 42 | " | 51.5 | 17.5 | 0.339 | 0.029 |
| 9 | 72 | " | 54 | 15 | 0.277 | 0.033 |
| 10 | 36 | " | 48 | 13.5 | 0.281 | 0.029 |

TABLE 2 (contd..2)

THE PRESCHOOL CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT REGION MALNUTRITION EVALUATION (MALES)

| No. | Approx. age in months | Villages | Circumferential measurements of head | Circumferential measurements of the right arm | Ratio of the circumferential measurements of arm the head | Deviation from the norms |
|---|---|---|---|---|---|---|
| 11 | 36 | Gökçekaynak (11.40 ppm) | 47.5 | 16 | 0.336 | 0.026 |
| 12 | 42 | " | 51 | 14.5 | 0.281 | 0.029 |
| 13 | 60 | Yeni köy (11.78 ppm) | 50.5 | 15.5 | 0.306 | 0.004 |
| 14 | 60 | " | 50.5 | 15 | 0.296 | 0.016 |
| 15 | 48 | " | 52.5 | 14.5 | 0.276 | 0.034 |
| 16 | 60 | " | 51.5 | 15 | 0.291 | 0.019 |
| 17 | 48 | " | 53 | 15 | 0.283 | 0.027 |
| 18 | 60 | Atabakan (12.16 ppm) | 51.5 | 14 | 0.271 | 0.039 |
| 19 | 30 | " | 49 | 15 | 0.306 | 0.004 |
| 20 | 18 | " | 46 | 14 | 0.304 | 0.006 |

TABLE 2 (contd..3)

THE PRESCHOOL CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT REGION MALNUTRITION EVALUATION (FEMALES)

| No. | Approx. age in months | Villages | Circumferential measurements of head | Circumferential measurements of the right arm | Ratio of the circumferential measurements of arm the head | Deviation from the norms |
|---|---|---|---|---|---|---|
| 1 | 18 | Gökçekaynak (11.40 ppm) | 50 | 14 | 0.280 | 0.039 |
| 2 | 17 | " | 47.8 | 13 | 0.272 | 0.038 |
| 3 | 24 | " | 47 | 17 | 0.360 | 0.050 |
| 4 | 84 | " | 49 | 17.5 | 0.357 | 0.047 |
| 5 | 84 | " | 51 | 17 | 0.333 | 0.023 |
| 6 | 48 | " | 51.5 | 17.5 | 0.339 | 0.029 |
| 7 | 14 | " | 48 | 16 | 0.300 | 0.010 |
| 8 | 36 | " | 47 | 14 | 0.297 | 0.013 |
| 9 | 18 | " | 43 | 15 | 0.348 | 0.038 |

TABLE 2 (contd..4)

THE PRESCHOOL CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT REGION MALNUTRITION EVALUATION (FEMALES)

| No. | Approx. age in months | Villages | Circumferential measurements of head | Circumferential measurements of the right arm | Ratio of the circumferential measurements of arm to head | Deviation from the norms |
|---|---|---|---|---|---|---|
| 10 | 60 | Gökçekaynak (11.40 ppm) | 51 | 15 | 0.294 | 0.016 |
| 11 | 24 | " | 49 | 14 | 0.285 | 0.025 |
| 12 | 42 | Yeni köy (11.78 ppm) | 50 | 15 | 0.300 | 0.010 |
| 13 | 36 | " | 50 | 14 | 0.280 | 0.030 |
| 14 | 18 | Atabakan (12.16 ppm) | 44 | 15 | 0.340 | 0.030 |

Ratio of the arm and head
0.310 < Normal
0.309-0.280 : Mild protein malnutrition
0.279-0.250 : II Degree malnutrition
0.249 : III Degree malnutrition .

TABLE 3

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT (MALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 1 | Gökçekaynak | 11.40 | 126 | 115 | 11 |
| 2 | " | " | 102 | 100 | 2 |
| 3 | " | " | 96 | 80 | 16 |
| 4 | " | " | 120 | 108 | 18 |
| 5 | " | " | 96 | 84 | 12 |
| 6 | " | " | 60 | 42 | 18 |
| 7 | " | " | 156 | 140 | 16 |
| 8 | " | " | 120 | 108 | 12 |
| 9 | Yılanlı | 10.26 | 84 | 60 | 24 |
| 10 | " | " | 96 | 90 | 6 |
| 11 | " | " | 72 | 56 | 16 |

TABLE 3 (contd..2)

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATERS IN DOĞUBEYAZIT (MALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 12 | Yılanlı | 10.26 | 60 | 40 | 20 |
| 13 | " | " | 84 | 66 | 18 |
| 14 | " | " | 96 | 62 | 34 |
| 15 | " | " | 108 | 104 | 4 |
| 16 | Atabakan | 12.54 | 108 | 104 | 4 |
| 17 | " | " | 120 | 108 | 12 |
| 18 | " | " | 108 | 96 | 12 |
| 19 | " | " | 80 | 64 | 24 |
| 20 | " | " | 120 | 120 | 0 |
| 21 | " | " | 108 | 102 | 6 |

TABLE 3 (contd..3)

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT (MALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 22 | Atabakan | 12.54 | 108 | 132 | -24 |
| 23 | " | " | 96 | 72 | 24 |
| 24 | " | " | 120 | 96 | 26 |
| 25 | Yeni köy | 11.78 | 108 | 96 | 12 |
| 26 | " | " | 84 | 100 | -16 |
| 27 | " | " | 72 | 68 | 4 |
| 28 | " | " | 72 | 52 | 20 |
| 29 | " | " | 36 | 32 | 4 |
| 30 | " | " | 48 | 40 | 8 |
| 31 | " | " | 36 | 28 | 8 |

TABLE 3 (contd..4)

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT (MALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 32 | Yeni köy | 11.78 | 36 | 32 | 4 |
| 33 | " | " | 42 | 40 | 2 |
| 34 | " | " | 108 | 100 | 8 |
| 35 | " | " | 72 | 48 | 24 |
| 36 | " | " | 48 | 38 | 10 |
| 37 | " | " | 36 | 34 | 2 |
| 38 | " | " | 30 | 34 | -4 |
| 39 | " | " | 36 | 25 | 11 |
| 40 | Tanıktepe | - | 90 | 84 | 6 |
| 41 | " | - | 108 | 96 | 12 |

TABLE 3 (contd..5)

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT (MALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 42 | Tanıktepe | - | 54 | 36 | 18 |
| 43 | " | - | 60 | 66 | -6 |
| 44 | " | - | 90 | 48 | 42 |
| 45 | " | - | 66 | 48 | 18 |
| 46 | " | - | 42 | 32 | 10 |
| 47 | " | - | 72 | 60 | 12 |
| 48 | " | - | 108 | 96 | 12 |
| 49 | " | - | 54 | 45 | 9 |
| 50 | Atabakan | 12.16 | 36 | 36 | 0 |
| 51 | " | " | 84 | 60 | 24 |

TABLE 3 (contd..6)

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT (MALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 52 | Atabakan | 12.16 | 72 | 66 | 6 |
| 53 | " | " | 60 | 45 | 15 |
| 54 | Pullutarla | - | 108 | 108 | 0 |
| 55 | " | - | 84 | 70 | 14 |
| 56 | " | - | 90 | 80 | 10 |
| 57 | " | - | 96 | 84 | 12 |
| 58 | " | - | 78 | 60 | 18 |
| 59 | " | - | 120 | 108 | 12 |
| 60 | " | - | 156 | 125 | 31 |

TABLE 3 (contd..7)

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT (MALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 61 | Pullutarla | - | 120 | 115 | 5 |
| 62 | Ortadirek | 12.16 | 108 | 84 | 24 |
| 63 | " | " | 168 | 144 | 24 |
| 64 | " | " | 60 | 48 | 12 |
| 65 | " | " | 108 | 96 | 12 |
| 66 | " | " | 132 | 132 | 0 |
| 67 | " | " | 108 | 105 | 3 |
| 68 | Türkmen | - | 132 | 120 | 12 |
| 69 | " | - | 156 | 120 | 36 |

TABLE 4

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT (FEMALES)

| No. | Village | Fluoride content in drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 1 | Gökçekaynak | 11.40 | 120 | 110 | 10 |
| 2 | " | " | 144 | 113 | 31 |
| 3 | " | " | 144 | 110 | 34 |
| 4 | " | " | 102 | 90 | 12 |
| 5 | " | " | 84 | 50 | 34 |
| 6 | " | " | 78 | 55 | 23 |
| 7 | " | " | 60 | 58 | 2 |
| 8 | " | " | 120 | 105 | 15 |
| 9 | " | " | 138 | 105 | 33 |
| 10 | " | " | 132 | 105 | 27 |

TABLE 4 (contd..2)

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT (FEMALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 11 | Gökçekaynak | 11.40 | 102 | 85 | 17 |
| 12 | " | " | 108 | 83 | 25 |
| 13 | " | " | 102 | 83 | 19 |
| 14 | " | " | 114 | 96 | 18 |
| 15 | Yeni köy | 11.78 | 60 | 47 | 13 |
| 16 | " | " | 132 | 108 | 24 |
| 17 | " | " | 72 | 58 | 14 |
| 18 | " | " | 48 | 38 | 10 |
| 19 | " | " | 104 | 90 | 14 |
| 20 | " | " | 84 | 68 | 16 |

TABLE 4 (contd..3)

EVALUATIONS OF THE DENTAL AND ASSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZİT (FEMALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 21 | Yeni köy | 11.78 | 96 | 80 | 16 |
| 22 | " | " | 72 | 50 | 22 |
| 23 | " | " | 72 | 25 | 47 |
| 24 | " | " | 84 | 65 | 19 |
| 25 | " | " | 114 | 105 | 9 |
| 26 | " | " | 120 | 100 | 20 |
| 27 | Tanıktepe | - | 120 | 115 | 5 |
| 28 | " | - | 84 | 50 | 34 |
| 29 | " | - | 96 | 94 | 2 |
| 30 | Atabakan | 12.16 | 168 | 121 | 47 |

TABLE 4.(contd..4)

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT (FEMALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 31 | Atabakan | 12.16 | 114 | 100 | 14 |
| 32 | " | " | 132 | 128 | 4 |
| 33 | " | " | 54 | 45 | 9 |
| 34 | " | " | 60 | 45 | 15 |
| 35 | " | " | 72 | 62 | 10 |
| 36 | " | " | 80 | 71 | 9 |
| 37 | " | " | 96 | 85 | 11 |
| 38 | " | " | 138 | 121 | 17 |
| 39 | " | " | 150 | 121 | 29 |
| 40 | Pullutarla | - | 144 | 110 | 34 |

TABLE 4 (contd..5)

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT (FEMALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 41 | Pullutarla | - | 84 | 76 | 8 |
| 42 | " | - | 108 | 94 | 14 |
| 43 | " | - | 96 | 65 | 31 |
| 44 | " | - | 96 | 83 | 13 |
| 45 | " | - | 60 | 56 | 4 |
| 46 | Ortadirek | 12.16 | 108 | 83 | 25 |
| 47 | " | " | 180 | 140 | 40 |
| 48 | " | " | 150 | 121 | 29 |
| 49 | " | " | 114 | 94 | 20 |
| 50 | " | " | 120 | 100 | 20 |

TABLE 4 (contd..6)

EVALUATIONS OF THE DENTAL AND OSSEOUS AGES OF THE CHILDREN DRINKING HIGH FLUORIDE CONTAINING WATER IN DOĞUBEYAZIT (FEMALES)

| No. | Village | Fluoride content of drinking water p.p.m. | Dental age in months | Osseous age in months | Difference between dental age and osseous age |
|---|---|---|---|---|---|
| 51 | Ortadirek | 12.16 | 96 | 71 | 25 |
| 52 | " | " | 72 | 58 | 14 |
| 53 | " | " | 96 | 83 | 13 |
| 54 | " | " | 84 | 57 | 27 |
| 55 | " | " | 72 | 71 | 1 |
| 56 | " | " | 132 | 110 | 22 |
| 57 | " | " | 72 | 60 | 12 |
| 58 | " | " | 84 | 71 | 13 |
| 59 | Türkmen | - | 108 | 94 | 14 |
| 60 | " | - | 108 | 83 | 25 |

age and skeleton age of boys and girls.

With = 0.0005 type I error and with 127 degree of freedom the observed t value has been found as $t = 3.762$. This value exceeds the table value which is 3.460. This shows that the difference between dental age and skeletal age is extremely higher in females than in boys. The chi-square test's results which have been used to determine if any malnutritiion exists among boys and girls, do not show any malnutritiion among the children either boys or girls.

Discussion of the findings

Even though we were unable to determine the actual chronological age of the children because of socio-cultural reasons, the results of this study are interesting in that roentgenological evaluations of the dental system, carpals and metacarpals of the children have uncovered certain questions in our minds.

First, high flouride ingestion with long duration seems to have a more deleterious effect on the development of the skeleton than on the teeth of the children.

Dental development seems to be more or less within the normal ranges even though we were unable to determine the actual chronological ages. There seem to be various factors playing an important role in the difference of skeletal and dental age. One of these factors seems to be the dynamic difference which exists between bones and dental tissues. In experiments with radio active elements, it has been shown that the calcium turnover from bones to extracellular fluid is six times higher in bones than in teeth[50]. Flouride inhibits the calcium to be dissolved from bones and from teeth by forming flouro-apatites which are less soluable than hydroxy-apatites, but the turnover rate seems to be less than the given ratio above.

Another important factor seems to be related to collagen biosynthesis. Various research workers such as Gollup, Glimcher and Goldhaber[39], Peck, Zipkin, and Whedon[38] have shown that the collagen biosynthesis in the calvarias of the rats was significantly depressed in animals drinking 10 p.p.m. flouride for 10 months. This depression was found to be apparent at tissue level. Gollup, Glimcher and Goldhaber(39) have also shown that even if in the bone culture media of less than 0.2 p.p.m. flouride was found, collagen breakdown also occurred. Flouride concentration in extracellular fluid may possibly increase, this in turn inhibits the collagen biosynthesis which is the prerequisite for the dental and skeletal development. This inhibition seems to be on a lesser extent in dental tissues than in bone tissues, otherwise we are unable to explain the difference which we observed in x-rays.

Another factor which may result in the difference which has been observed between skeletal growth and dental growth is the nutritional status of the children, since we were unable to detect any malnutrition, it is not worthwhile to consider malnutrition here.

If there are no other factors playing role in the difference observed between skeletal development and dental development, than we may conclude that high flouride intake with long duration has a deleterious effect on skeletal development, one additional point may be the thyroid function. It is well known that the thyroid dysfunction affects both dental and skeletal developments, however, the research work done up to now has not demonstrated any deleterious effect of flourides upon this function.

Another point which has been noticed by Tuncel(52) is the band formation on the bones. This finding will be discussed by Dr. Tuncel in detail.

McCauley and McClure(41) in their research work could not find any significant difference between dental age and skeletal age of the children drinking 6.2 p.p.m. flouride containing water. This finding is definitely open to discussion. It is our opinion that flouride intake above 10 p.p.m. seems to cause this deleterious effect which we observed. The control mechanism which I suggested above possibly does not function above a certain level of flouride intake. One may also raise the question that race and inherited factors may play a role in the difference between skeletal age and dental age. Since we do not have the Turkish norms of development of dental and skeletal systems, we are unable to discuss this very important point for the time being.

Conclusions

From the research described here it is clear that:

1. High flouride intake has a deleterious effect on skeletal development, and

2. this deleterious effect occurs also, but to a lesser extent on dental tissue.

## REFERENCES


1. Ata, P.: Diş Hekimliği Tedavisi Ders Notları, 1957.

2. Usmen, E.: Isparta il, ilçe ve köylerinde diş flourosisi (Unpublished data), 1975.

3. Gülhan, A.; Mermutlu, B.: Relations entre la flourose endemique et la frequence de la caria. Istanbul Üniversitesi Diş Hekimliği Fakültesi Dergisi Cilt 7, sayı 2: 130-140, 1973.

4. Oruç, N.: Doğu Beyazıt kazası ve bazı köylerinde kullanılan sularda flourür konsantrasyonu ve önemi, Atatürk Üniversıtesi Ziraat Fakültesi, Ziraat Dergisi Cilt 4, sayı 1, 1973.

5. Nickles, J.: C.R. Acad. Sci. (Paris), 43:885, 1856.

6. Tamman, G.: Hoppe-Seylers Z. Physiol. Chem., 12: 322, 1888.

7. Brandl, J. and Tappeıner, H.: Z. Biol., 28: 518, 1891.

8. Carlson, C.H., Armstrong, W.D., Singer, L.: Amer. J. Physiol., 199: 187, 1960.

9. Singer, L., Armstrong, W.D.: G. app. Physiol., 15: 508-510, 1960.

10. Taves, D.R.: Nature (Lond.), 217: 1050, 1968.

11. Singer, L., Armstrong, W.D.: Arch. Orol Biol., 14, 1969.

12. Armstrong, W.D., Venkates, W. and Singer, L.: Adapted from Flourides and Human Health, W.H.O., 96, 1970.

13. Weidmann, S.M., Weatherell, J.A.: J. Path. Bact., 78: 243, 1959.

14. Zipkin, I. et al: Publ. Health. Rep. (Wash) 73: 732-740, 1958.

15. Largent, E.G.: Fluorosis, the health aspects of fluorine compounds, Columbus, Ohio State University Press, p:22, 1961.

16. Neumann, W.F., Neumann, M.W.: The chemical dynamics of bone meneral Chicago, University of Chicago Press, 1958.

17. Neumann, W.F., Neumann, M.W., et al: J. Biol. Chem., 187: 655-661, 1950.

18. Scheinin, A.: Suom. Hammaslääk Seur, Toîm., 50, Suppl. 2: 53-56, 1954.

19. Brudevald, F.: Chemical composition of teeth in relation to carries, In: Sognnaes, R.F. ed., Chemistry and prevention of dental caries, Springfield, Ill., Thomas, pp. 32-88, 1962.

20. Bauer, G.C.H., Carlson, A., Linguist, B.: Kungl. Fysiogr. Sällsk, Lund Förh., 25: 1, 1955.

21. McClure, F.J. Likins, R.C.: J. Dent. Res., 30: 172, 1951.

22. Suttie, J.W., Philips, P.H.: Arch. Biochem. 83: 355, 1959.

23. Perkinson, J.D. et al: Am. Jour. Physiol., 182: 383-389, 1955.

24. Wallace - Durbin, P.: J. Dent. Res., 33: 789 - 800, 1954.

25. Weatherell, J.A., Hargreaves, J.A. Arch. Oral. Biol. 10: 139, 1965.

26. Volker, J.F., Sognaes, R.F. and Bibby, B.G.: Amer. J. Physiol., 132: 707-712, 1941.

27. Jackson, D., Weidmann, S.M.: Brit. Dent. J., 107: 303, 1959.

28. Savchuck, W.B., Armstrong, W.D.: J. Biol. Chem., 193: 575, 1951.

29. McCann, H.G., Bullock, F.A.: J. Dent. Res., 36: 391, 1957.

30. Lawrenz, M., Mitchell, H.M.: J. Nutr., 22: 91 - 101, 1941.

31. Narayana, R.: The role of calcium inchronic fluorine intoxication (Thesis, Madras University) adapted from fluorides and human health, W.H.O. Monograph Series, No: 39, 1970.

32. Wagner, M.J., Muhler, J.C.: J. Dent. Res., 38: 1078, 1959.

33. Purves, M.J.: Jancet, 1188-1189, 1962.

34. Gedalia, I. et al: Harefuah, 64: 191, 1963.

35. Knappwost, A., Tachtermann, H.G.: Dtsch. Zahnarstl Z. 10: 1231, 1955.

36. Bixler, D., Muhler, J.C.: J. Nutr., 70: 26, 1960.

37. Riektniece, E. et al: Arch. Oral. Biol., 10: 107, 1965.

38. Zipkin, I., Peck, W.A., Whedon, G.D.: Clin. Res. 13: 330, 1965.

39. Golub, L., Glimcher, M.J., Goldhaber, P.: Proc. Soc. Exp. Biol. (N.Y.) 129: 973, 1968.

40. Schlesinger, E.R. et al: J. Am. Dent. Ass., 52: 296, 1956.

41. McCauley, H.B., McClure, F.J.: Pub. Health Rep., (Wash) 69: 671-683, 1954.

42. Geever, E.F. et al: Publ. Health Rep., (Wash) 73: 721-731, 1958.

43. Morris, J.W.: Amer. J. Roentgenol., 94: 608-615, 1965.

44. Stevenson, C.A., Watson, A.R.: Amer. J. Roentgenol., 78: 13-18, 1957.

45. Gomez, F. et al: Advances in Pediatricis, New York, Year Book Publishers, Vol. 7: 131, 1955.

46. Kürkçüoğlu, M.: Erzurumda latent malnütrüsyonların değerlendirilmesinde hidroxyproline ölçütleri, Atatürk Üniversitesi Tıp Bülteni, 3: 1-18, 1970.

47. McLaren, D.S.: The arm and head circumference as a public health index of protein calorie malnutrition of early childhood, J. Trop. Pediat., 15: 233, 1970.

48. Moorress, C., Fanning, F.A., Hunt, E.E.: J. Dent. Res. 42: 1490-1502, 1963.

49. Greulich, W.W., Pyle, S.I.: Radiographic atlas of skeletal development of the hand and wrist, Stanford University Press, 2nd ed., 1959.

50. White, A., Handler, P., Smith, E.L.: Principles of Biochemistry, pp. 887-898, McGraw-Hill. Comp. 'th ed., 1968.

51. Nicholas, G., Flanagan, B.: Fed. Prac. 25: 922, 1966.

52. Tuncel, E.: Personal communication, Dept. of Radiolog., School of Med., Atatürk Üniversitesi, Erzurum, Turkey.

# EVALUATION OF THE CHRONIC FLUORIDE INTOXICATION IN THE DOĞUBEYAZIT REGION FROM THE NEUROLOGICAL STANDPOINT

By Dr. Mehmet Öztopçular
Associate Professor
Department of Neurology
Faculty of Medicine
Atatürk University
Erzurum, Turkey

(Abstract)

This pilot study has been made on 29 males and 11 females from Doğubeyazıt region where the fluoride contents of drinking water are above the optimal level.

Clinical, laboratory, roentgenologic, and E.E.G. evaluations of these cases were made. In the studied group, walking difficulties, skeletal changes and changes in tendon reflexes were observed.

Laboratory findings reveal that chronic fluoride intoxication causes disturbances in serum electrolytes and in some cases collagen breakdown (rheumatoid factor). E.E.G. activities of the cases studied, show significant changes with a tendency towards hypoactive E.E.G. patterns.

Various aspects of fluoride intoxication have been discussed particularly in terms of laboratory findings and E.E.G. patterns.

## INTRODUCTION

This study has been undertaken in order to determine the neurological problems which are caused by chronic fluoride intoxication in the villages of Doğubeyazıt where the fluoride content of the drinking waters is up to 12 p.p.m.

In the article, the pathological symptoms due to fluorosis observed among the villages of Doğubeyazıt are thoroughly evaluated from the medical point of view and possible solutions to the problem are discussed.

## Materials and Method

40 adult cases with an affected area of fluorosis were hospitalized on various occasions between 1975-1977, in the Medical Center of Atatürk University, for roentgenologic and E.E.G. evaluations. Clinical examinations on these people particularly dwelt upon the neurological standpoint. Laboratory evaluations covered blood proteins, serum natrium, potassium, calcium, phosphorus levels and alkaline phosphatase activity. Latex fixation tests were made in order to determine the R.F. factor suspected to be positive among these type of cases.

In addition to these evaluations, skeletal surveys were made, and x-rays were taken particularly of skulls (A-P, lateral and submento vertical), cervix (A-P, lateral), lumbo-sacral regions, pelvises and joints of the cases.

E.E.G. evaluation of the cases was made with 8-channel Siemens-Elema Schönander equipment.

Chi-square tests were given in order to determine the relationship between clinical findings, laboratory findings, E.E.G. pattern, and the factors which effect these.

## Findings

The studied cases consisted of 11 females and 29 males. The age ranged between 20-85. Table 1 shows the villages using drinking water with a high flouride content.

Table 2 gives the age and sex distribution of the studied cases.

Subjective complaints of the studied persons are shown in Table 3.

TABLE 1

FLUORIDE LEVELS OF THE SAMPLES TAKEN FROM AFFECTED AREA

| Name of the village | Previous Fluoride Content (as p.p.m) 1973 | Recent Fluoride Content 1977 |
|---|---|---|
| Gökçekaynak | 11.70 | 0.3 |
| Yılanlı | " | 0.8 |
| Atabakan | - | 3.5 |
| Çiftlik | - | 5.7 |
| Celal | 5 | - |
| Yanoba | - | - |
| Esnemez | - | - |

TABLE 2

DISTRIBUTION OF THE CASES TO AGE GROUPS AND SEXES

| | 20-30 | 31-41 | 41-50 | 51-60 | 61-70 | 71-80 | 81+ | Sum of the cases |
|---|---|---|---|---|---|---|---|---|
| Female | 1 | 3 | 4 | 1 | 1 | 1 | - | 11 |
| Male | 3 | 2 | 6 | 5 | 10 | 2 | 1 | 29 |
| Sum of the cases | 4 | 5 | 10 | 6 | 11 | 3 | 1 | 40 |

TABLE 3

THE COMPLAINTS OF THE CASES

| Case No. | Sex | Age | Extremity and joint pains | Pains in Cervical and dorsal regions | Difficulty in walking | Headaches | Lassitude | Seizures |
|---|---|---|---|---|---|---|---|---|
| 1 | M | 85 | + | − | − | + | − | − |
| 2 | M | 65 | + | + | + | + | − | − |
| 3 | M | 60 | + | − | − | − | − | − |
| 4 | M | 66 | + | + | − | − | − | − |
| 5 | M | 80 | + | + | − | − | − | − |
| 6 | M | 44 | + | + | + | + | − | − |
| 7 | F | 45 | + | + | − | − | − | − |
| 8 | F | 50 | + | − | + | + | − | − |
| 9 | F | 20 | − | + | − | − | − | − |
| 10 | M | 27 | + | − | − | − | − | − |
| 11 | M | 55 | + | − | − | − | − | − |
| 12 | M | 65 | + | + | − | − | − | − |
| 13 | M | 60 | + | − | − | − | − | − |
| 14 | M | 70 | + | − | − | + | − | − |
| 15 | M | 70 | + | − | − | + | − | − |
| 16 | F | 40 | − | − | + | − | − | − |
| 17 | F | 45 | + | − | + | − | − | − |
| 18 | M | 60 | + | − | − | − | − | − |
| 19 | M | 50 | + | − | − | + | − | − |
| 20 | M | 64 | + | + | + | − | − | − |

TABLE 3 (contd..2) THE COMPLAINTS OF THE CASES

| Case No. | Sex | Age | Extremity and joint pains | Pains in Cervical and dorsal regions | Difficulty in walking | Headaches | Lassitude | Seizures |
|---|---|---|---|---|---|---|---|---|
| 21 | M | 45 | + | + | + | – | – | – |
| 22 | M | 43 | + | – | + | – | – | – |
| 23 | M | 47 | + | + | – | – | – | – |
| 24 | M | 20 | + | – | – | – | – | + |
| 25 | F | 40 | + | – | – | – | – | – |
| 26 | M | 70 | + | + | – | + | – | – |
| 27 | M | 40 | + | + | – | + | – | – |
| 28 | M | 35 | + | + | – | – | + | – |
| 29 | M | 55 | + | + | – | – | – | – |
| 30 | F | 40 | + | – | – | – | – | – |
| 31 | M | 64 | + | + | – | – | + | – |
| 32 | M | 70 | + | + | – | – | – | – |
| 33 | M | 68 | + | + | – | + | – | – |
| 34 | M | 30 | + | – | + | – | + | – |
| 35 | F | 60 | + | + | + | + | + | – |
| 36 | M | 48 | + | – | – | – | + | – |
| 37 | F | 50 | + | + | + | – | – | – |
| 38 | F | 65 | + | + | – | + | + | – |
| 39 | M | 80 | + | + | – | + | – | – |
| 40 | F | 75 | + | + | + | + | – | – |
| | Total | | 38 | 22 | 12 | 14 | 6 | 1 |

As shown in Table 3, 38 cases out of 40 have extremity and have joint pains; 22 cases have cervical and dorsal pains; 14 have only headaches; 12 cases have difficulty in walking; 6 cases have lassitude and one case has seizures (clinical findings are shown in Table 4). We were unable to observe any clinical symptoms in 2 of the total cases. These two cases are in the 20-30 year age group, we found reflex changes in 30 of the cases; 15 of these showed significant decrease in their tendon reflexes, and 14 showed increased tendon reflexes. In only one case were we unable to find any tendon reflex. Two cases, however, showed pathological reflexes.

In terms of the atrophies, seen in the masculature, we noticed that 30 of the cases had atrophies of muscalature. 10 out of 30 cases showed a diffuse type of muscle atrophy, 9 cases showed atrophies both on the lower and the upper extremities; 10 cases showed atrophies only on the lower extremities, and 1 case had atrophies on the upper extremities and on the trunk.

In the tonicity evaluation of the muscles, in 17 cases we found muscle stiffness, in 6 cases we noticed hypotony of the muscles, in 19 cases we observed skeletal deformities consisting of 15 kyphosis, 2 pectus excavatum, 3 X-leg, 7 O-leg, and we observed limitations in the joint movements of 34 cases.

In 11 cases we observed difficulties in walking (Table 4), and in 12 cases we noticed walking complaints (Table 3).

In the following pictures, a case with O-leg, kyphosis and diffuse muscular atrophy can be seen (Picture 1-2).

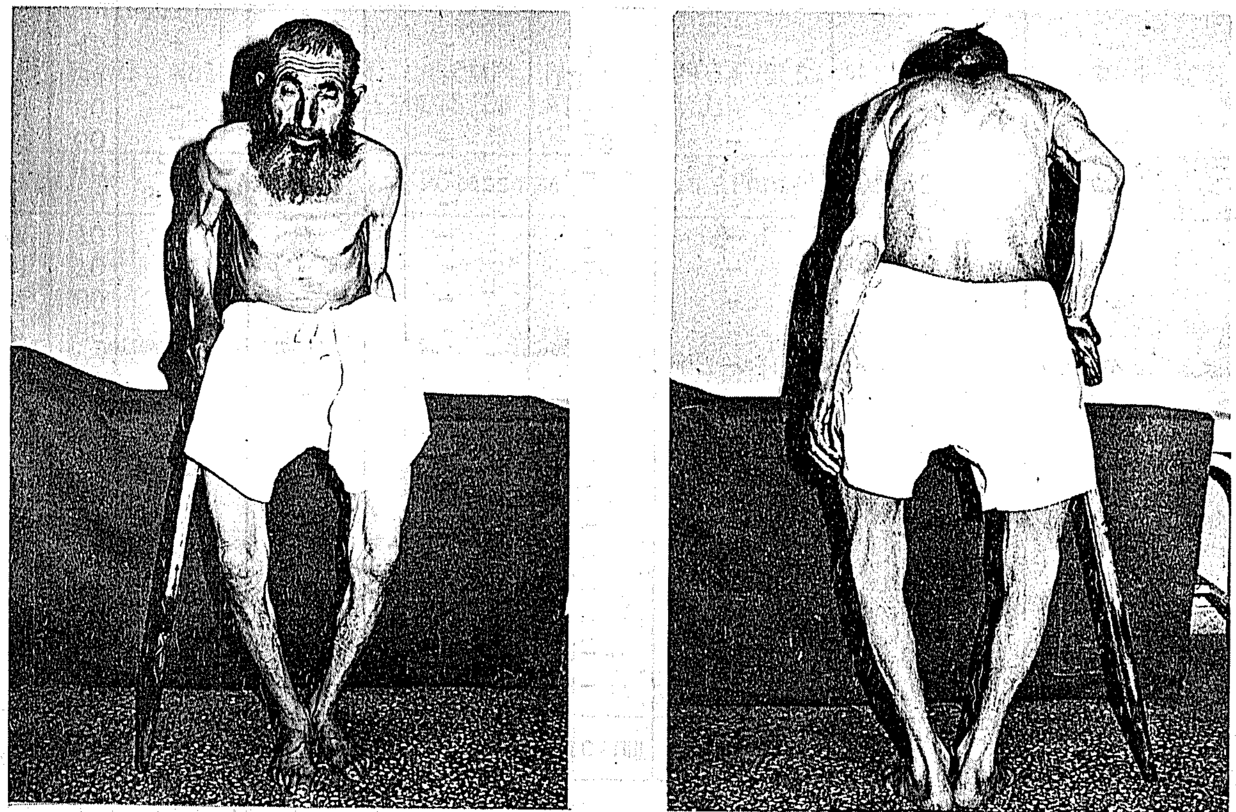
We have made laboratory tests in order to determine the serum natrium, potassium, calcium and phosphorus level of the cases. In addition to these electrolytes, we determined the serum alkaline phosphatase activity and rheumatoid factor for each case. The laboratory findings are shown in Table 5.

As seen in Table 5, serum proteins seem to have de-

TABLE 4 

CLINICAL FINDINGS OBSERVED ON THE CASES

| Case No. | Sex | Age | TENDON REFLEXES: Increased | TENDON REFLEXES: Decreased | TENDON REFLEXES: Absent | TENDON REFLEXES: Normal | Pat. Ref. | MUSCULATURE FINDINGS: Atrophy in the muscles: Lower Extr. | MUSCULATURE FINDINGS: Atrophy in the muscles: Upper Extr. | MUSCULATURE FINDINGS: Atrophy in the muscles: Trunk | MUSCULATURE FINDINGS: Stiffness in the muscles | MUSCULATURE FINDINGS: Hypotony in the muscles | SKELETAL DEFORMITIES: Kyphosis | SKELETAL DEFORMITIES: Pectus Excavatum | SKELETAL DEFORMITIES: X Leg | SKELETAL DEFORMITIES: O Leg | Limitations in the joint movements | Difficulty in walking |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | M | 85 | + | - | - | - | - | + | + | - | + | - | - | - | - | - | + | + |
| 2 | M | 65 | - | + | - | - | - | + | + | + | + | - | + | - | - | + | + | - |
| 3 | M | 60 | - | - | - | + | - | + | + | + | - | - | - | - | - | + | + | - |
| 4 | M | 66 | - | - | - | + | - | + | - | - | + | - | + | - | - | - | + | - |
| 5 | M | 80 | - | + | - | - | - | + | - | - | - | - | + | - | - | + | + | + |
| 6 | M | 44 | + | - | - | - | + | + | - | - | - | + | - | - | + | - | - | - |
| 7 | F | 45 | - | - | - | + | - | + | - | - | - | - | - | - | - | - | - | - |
| 8 | F | 50 | - | + | - | - | - | - | - | - | - | - | - | - | - | - | + | - |
| 9 | F | 20 | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | - |
| 10 | M | 27 | - | - | - | + | - | - | - | - | - | - | - | - | - | - | - | - |
| 11 | M | 55 | - | - | - | + | - | - | - | - | + | - | - | - | - | - | + | - |
| 12 | M | 65 | - | - | - | + | - | + | + | - | - | - | + | - | - | + | + | - |
| 13 | M | 60 | - | - | - | + | - | - | - | - | - | - | - | - | - | - | + | - |
| 14 | M | 70 | - | + | - | - | - | + | - | - | - | - | + | - | - | - | + | + |
| 15 | M | 70 | - | - | + | - | - | + | - | - | - | - | + | - | - | - | + | + |
| 16 | F | 40 | + | - | - | - | - | + | - | - | - | - | - | - | - | - | - | - |
| 17 | F | 45 | + | - | - | - | + | + | - | - | + | + | - | - | + | - | - | - |
| 18 | M | 60 | - | - | - | + | - | + | - | - | - | - | - | - | - | - | + | + |
| 19 | M | 50 | - | + | - | - | - | - | - | - | - | - | - | - | - | - | + | - |
| 20 | M | 64 | + | - | - | - | - | - | - | - | + | - | + | - | - | - | + | - |
| 21 | M | 45 | + | - | - | - | - | + | + | - | + | - | + | - | - | - | + | - |
| 22 | M | 43 | - | + | - | - | - | + | + | + | - | + | + | - | - | - | + | - |
| 23 | M | 47 | + | - | - | - | - | - | + | + | + | - | - | - | - | - | + | - |
| 24 | M | 20 | - | - | - | + | - | + | + | - | - | - | - | - | - | - | + | - |
| 25 | F | 40 | + | - | - | - | - | - | - | - | + | - | - | - | - | - | + | - |
| 26 | M | 70 | - | + | - | - | - | + | + | + | - | - | + | - | - | - | + | + |
| 27 | M | 40 | - | + | - | - | - | + | + | + | - | - | - | - | - | - | + | - |
| 28 | M | 35 | - | + | - | - | - | + | + | - | - | + | - | - | - | - | + | - |
| 29 | M | 55 | + | - | - | - | - | - | - | - | + | - | - | - | - | - | + | - |
| 30 | F | 40 | - | + | - | - | - | + | + | + | - | + | - | - | - | + | + | - |
| 31 | M | 64 | + | - | - | - | - | + | + | + | + | - | + | + | - | + | + | - |
| 32 | M | 70 | + | - | - | - | - | + | + | + | - | - | - | - | - | - | + | + |
| 33 | M | 68 | + | - | - | - | - | + | + | + | + | - | - | + | - | - | + | + |
| 34 | M | 30 | - | + | - | - | - | + | - | - | - | + | - | - | - | - | + | - |
| 35 | F | 60 | + | - | - | - | - | + | + | - | + | - | + | - | - | - | + | - |
| 36 | M | 48 | - | + | - | - | - | + | + | - | - | - | - | - | - | - | + | - |
| 37 | F | 50 | + | - | - | - | - | - | - | - | + | - | - | - | - | - | + | - |
| 38 | M | 65 | - | + | - | - | - | + | + | + | + | - | + | - | + | - | + | + |
| 39 | M | 80 | - | + | - | - | - | + | + | - | + | - | + | - | - | - | + | + |
| 40 | F | 75 | - | + | - | - | - | + | + | - | + | - | + | - | - | + | + | + |
| Total | | | 14 | 15 | 1 | 10 | 2 | 29 | 20 | 11 | 17 | 6 | 15 | 2 | 3 | 7 | 34 | 11 |



Pictures 1 & 2 (Case No. 2)

TABLE 5

SERUM PROTEINS, Na, POTASSIUM, CALCIUM, PHOSPHORUS LEVELS, ALKALINE PHOSPHATASE ACTIVITY AND THE RESULTS OF LATEX FIXATION TESTS (R.F.)

| Case No. | Sex | Age | Serum Proteins | Sodium | Potassium | Calcium | Phosphorus | Alkaline Phosphatase | Latex Fixation (R.F.) |
|---|---|---|---|---|---|---|---|---|---|
| 1 | M | 85 | – | 135 | – | – | 3.4 | 5.6 | – |
| 2 | M | 65 | – | 137 | 3 | – | – | 5.6 | + |
| 3 | M | 60 | – | – | – | – | 3.4 | 4.8 | – |
| 4 | M | 66 | – | – | – | – | 3.2 | – | – |
| 5 | M | 80 | – | – | – | – | 3.4 | 4.8 | – |
| 6 | M | 44 | – | 134 | 3.2 | – | – | 6.4 | – |
| 7 | F | 45 | – | – | – | 8.7 | – | – | – |
| 8 | F | 50 | – | – | – | – | – | – | – |
| 9 | F | 20 | – | – | – | – | – | – | – |
| 10 | M | 27 | – | – | – | – | – | – | – |
| 11 | M | 55 | – | – | – | – | – | – | + |
| 12 | M | 65 | – | 136 | – | 13 | – | – | – |
| 13 | M | 60 | – | – | – | 14 | – | – | + |
| 14 | M | 70 | – | – | – | 14 | – | – | + |
| 15 | M | 70 | – | – | – | 13 | – | – | – |
| 16 | F | 40 | – | – | – | – | – | 4.8 | + |
| 17 | F | 45 | – | 129 | – | – | 3.4 | – | – |
| 18 | M | 60 | – | 134 | – | 13 | – | – | – |
| 19 | M | 50 | – | 137 | – | 13 | – | – | – |
| 20 | M | 64 | – | – | – | 12.7 | – | – | – |
| 21 | M | 45 | – | – | – | 12 | – | 4.8 | – |

TABLE 5 (contd..2)

SERUM PROTEINS, Na, POTASSIUM, CALCIUM, PHOSPHORUS LEVELS, ALKALINE PHOSPHATASE ACTIVITY AND THE RESULTS OF LATEX FIXATION TESTS (R.F.)

| Case No. | Sex | Age | Serum Proteins | Sodium | Potassium | Calcium | Phosphorus | Alkaline Phosphatase | Latex Fixation (R.F.) |
|---|---|---|---|---|---|---|---|---|---|
| 22 | M | 43 | – | – | – | – | – | – | + |
| 23 | M | 47 | – | – | – | – | – | – | + |
| 24 | M | 20 | – | – | – | – | – | – | – |
| 25 | F | 40 | – | – | – | – | – | – | – |
| 26 | M | 70 | – | – | – | – | 3.4 | – | + |
| 27 | M | 40 | – | – | – | – | – | – | – |
| 28 | M | 35 | – | – | – | – | 3.4 | – | – |
| 29 | M | 55 | – | – | – | – | – | – | + |
| 30 | F | 40 | – | – | – | 11.7 | – | – | + |
| 31 | M | 64 | – | – | – | – | – | – | – |
| 32 | M | 70 | – | 137 | – | – | – | 4.8 | + |
| 33 | M | 68 | – | 136 | – | 7.65 | – | 4.8 | + |
| 34 | M | 30 | – | – | – | – | – | – | + |
| 35 | F | 60 | – | – | – | 11.6 | – | – | + |
| 36 | M | 48 | 4.4 | 132 | – | 11.8 | – | 4.8 | – |
| 37 | F | 50 | – | 135 | – | – | – | – | – |
| 38 | F | 65 | – | – | – | – | – | – | – |
| 39 | M | 80 | 4.4 | 147 | – | – | – | – | – |
| 40 | F | 75 | – | 137 | – | – | – | 8 | + |
| | Total | | 2 | 13 | 2 | 13 | 7 | 11 | 15 |

crease in two of the cases, natrium levels have also decreased in 12 of the cases. In one case, we found an increase of the serum natrium level. 2 of the cases showed decreases in the serum potassium level. In 11 of the cases, we determined the calcium level to be increased and in two of the cases to be decreased. Serum phosphorus levels of 7 cases were found to be low.

Alkaline phosphatase activities of 11 cases showed an elevation. We found R.F. to be positive in 15 of the cases.

The results of the skeletal surveys are shown in Table 6. X-ray evaluations of the cases indicate that 5 of these cases are within the normal acceptance level. In 35 cases, we found axial osteosklerosis, 9 of these cases showed severe, 10 moderate, and 16 mild osteosklerosis. In 28 cases we found extremiter osteosklerosis, 5 cases showed ligament calcification, in 15 cases we found periostal reactions, 7 cases showed calcanael spine. There was thickening of the skull in 2 of the cases.

In terms of the E.E.G. evaluations, we found the following results: 14 of the cases were within normal E.E.G. appearance, 13 cases showed marked flat E.E.G. pattern, in 11 cases we found hypoactive E.E.G. 1 case showed diffuse beta activity in repeated E.E.G.'s, and in 1 case we observed pathologic E.E.G. activity with tetha and delta waves indicating a localized pathology. In this particular case, we were unable to accomplish further studies because of a lack of cooperation by the patient. In Table 7, you see the distribution of E.E.G. activity in the observed cases.

We were able to recheck 14 cases within 1 to 2 years. As far as we know, the fluoride content of the drinking water from which these people drink, are almost within optimal range or diminished in comparison to the previous level, and these people have been drinking from these sources for 4 years.

In our controls 10 of the 14 cases showed significant improvement in the E.E.G. activity. In pictures 9 and 10 a comparative picture of two cases has been presented.

TABLE 6

DISTRIBUTION OF THE SKELETAL, LIGAMENT AND PERIOSTAL REACTIONS OF THE CASES DETERMINED BY MEANS OF X-RAYS OF THE CASES

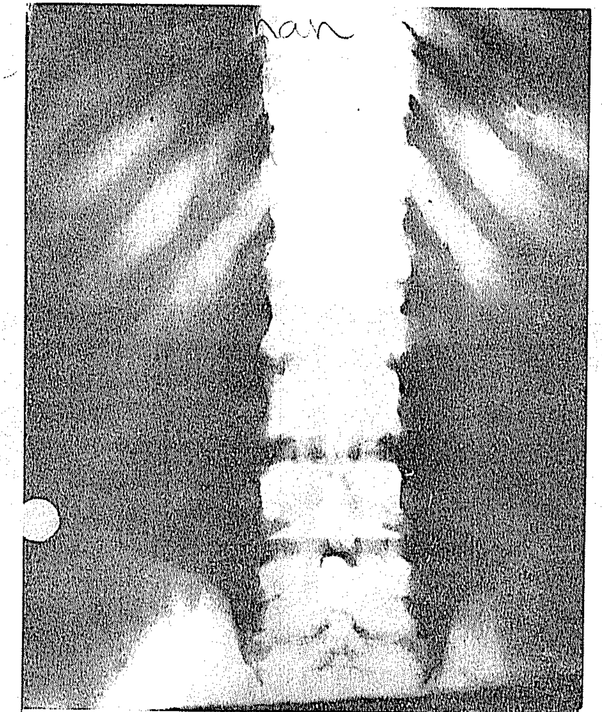
| Case No. | Sex | Age | Osteosklerosis | | Calcifications on the Ligaments | Periostal Reactions | Thickening of the bones of the skull | Calcaneal spine |
|---|---|---|---|---|---|---|---|---|
| | | | Axial | Extremities | | | | |
| 1 | M | 85 | - | - | - | - | - | - |
| 2 | M | 65 | +++ | + | - | (+) Forearm/tibia/femur | - | + |
| 3 | M | 60 | + | - | - | - | - | + |
| 4 | M | 66 | +++ | + | - | (+) Costae/tibia | - | - |
| 5 | M | 80 | ++ | + | - | - | - | - |
| 6 | M | 44 | +++ | + | (+) Sacrospinose, sacrotuberose | (+) Forearm/tibia | - | + |
| 7 | F | 45 | - | - | - | - | - | - |
| 8 | F | 50 | + | - | - | - | - | - |
| 9 | F | 20 | + | - | - | - | - | - |
| 10 | M | 27 | + | - | - | - | - | - |
| 11 | M | 55 | ++ | + | - | - | - | + |
| 12 | M | 65 | +++ | ++ | - | (+) Forearm | - | - |
| 13 | M | 60 | + | + | - | - | - | - |
| 14 | M | 70 | ++ | + | - | (+) Forearm | - | - |
| 15 | M | 70 | - | - | - | - | - | - |
| 16 | F | 40 | + | - | - | - | - | - |
| 17 | F | 45 | +++ | + | - | (+++) Costae | - | + |
| 18 | M | 60 | - | - | - | - | - | - |
| 19 | M | 50 | + | - | - | (+) Ulna | - | - |
| 20 | M | 64 | +++ | + | - | (+) Tibia/humerus/forearm | - | - |
| 21 | M | 45 | ++ | + | (+) Anterior cervical ligament | (+) Fibula/forearm | - | + |
| 22 | M | 43 | ++ | - | - | - | - | - |
| 23 | M | 47 | ++ | + | - | - | - | - |
| 24 | M | 20 | + | + | - | - | - | - |
| 25 | F | 40 | + | + | - | - | - | - |
| 26 | M | 70 | ++ | + | - | - | - | - |
| 27 | M | 40 | ++ | + | - | - | - | - |
| 28 | M | 35 | + | + | - | - | - | - |
| 29 | M | 55 | ++ | + | (+) Calcification on knee ligaments | - | + | + |
| 30 | F | 40 | + | + | - | - | - | - |
| 31 | M | 64 | +++ | ++ | (+) Anterior lumbar vertebrae calification and on pelvis ligaments | (+) Tibia/fibula/forearm/costae | - | - |
| 32 | M | 70 | + | + | - | - | - | - |
| 33 | M | 68 | ++ | + | - | - | - | - |
| 34 | M | 30 | + | + | - | - | - | - |
| 35 | F | 60 | - | - | - | - | - | - |
| 36 | M | 48 | + | + | - | (+) Forearm | - | - |
| 37 | F | 50 | + | + | - | (+) Forearm | - | - |
| 38 | F | 65 | + | + | - | (+) Forearm/humerus | - | - |
| 39 | M | 80 | +++ | ++ | (+) Sacrospinose, vertebrae | (+++) Humerous/forearm/tibia/fibula | - | - |
| 40 | F | 75 | +++ | ++ | - | (+) Forearm/fibula/tibia | + | - |

(+) Mild (++) Moderate (+++) Severe

TABLE 7

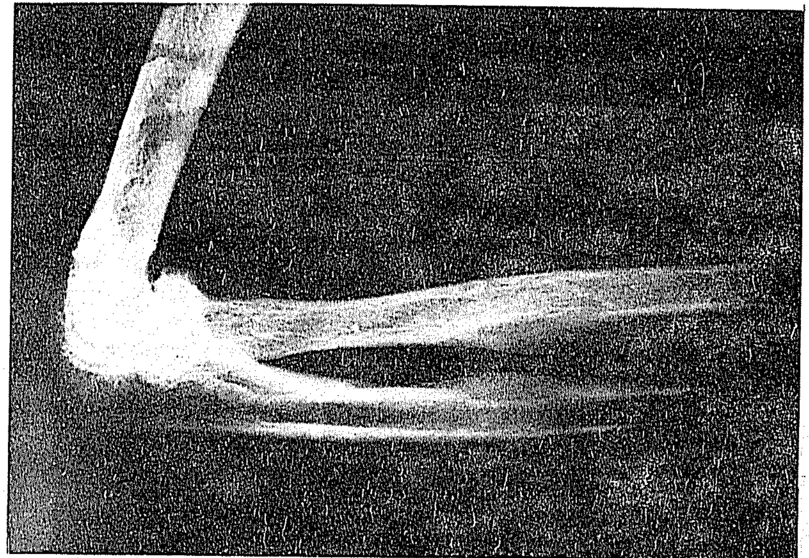
DISTRIBUTION OF THE E.E.G. ACTIVITY AMONG THE CASES

| Case No. | Sex | Age | Flat E.E.G. | Hypoactive E.E.G. | Localized Pathologic E.E.G. | Beta Activity in repeated E.E.G. | Normal |
|---|---|---|---|---|---|---|---|
| 1 | M | 85 | - | - | - | - | + |
| 2 | M | 65 | + | - | - | - | - |
| 3 | M | 60 | + | - | - | - | - |
| 4 | M | 66 | + | - | - | - | - |
| 5 | M | 80 | + | - | - | - | - |
| 6 | M | 44 | - | + | - | - | - |
| 7 | F | 45 | - | + | - | - | - |
| 8 | F | 50 | - | - | - | - | + |
| 9 | F | 20 | - | - | - | - | + |
| 10 | M | 27 | - | - | - | - | + |
| 11 | M | 55 | + | - | - | - | - |
| 12 | M | 65 | - | + | - | - | - |
| 13 | M | 60 | - | + | - | - | - |
| 14 | M | 70 | - | - | - | - | + |
| 15 | M | 70 | - | + | - | - | - |
| 16 | F | 40 | - | - | - | - | + |
| 17 | F | 45 | - | + | - | - | - |
| 18 | M | 60 | - | + | - | - | - |
| 19 | M | 50 | + | - | - | - | - |
| 20 | M | 64 | + | - | - | - | - |
| 21 | M | 45 | + | - | - | - | - |
| 22 | M | 43 | - | + | - | - | - |
| 23 | M | 47 | + | - | - | - | - |
| 24 | M | 20 | - | + | - | - | - |
| 25 | F | 40 | - | - | - | - | + |
| 26 | M | 70 | - | - | - | + | - |
| 27 | M | 40 | - | - | - | - | + |
| 28 | M | 35 | - | - | - | - | + |
| 29 | M | 55 | + | - | - | - | - |
| 30 | F | 40 | - | - | - | - | + |
| 31 | M | 64 | - | + | - | - | - |
| 32 | M | 70 | + | - | - | - | - |
| 33 | M | 68 | + | - | - | - | - |
| 34 | M | 20 | + | - | - | - | - |
| 35 | F | 60 | - | - | + | - | - |
| 36 | M | 48 | - | + | - | - | - |
| 37 | F | 50 | - | - | - | - | + |
| 38 | F | 65 | - | - | - | - | + |
| 39 | M | 80 | - | - | - | - | + |
| 40 | F | 75 | - | - | - | - | + |
| | Total | | 13 | 11 | 1 | 1 | 14 |



Picture 3 (Case No. 17)

Periostal reactions on the costae, chalky appearance on the columa vertebralis.

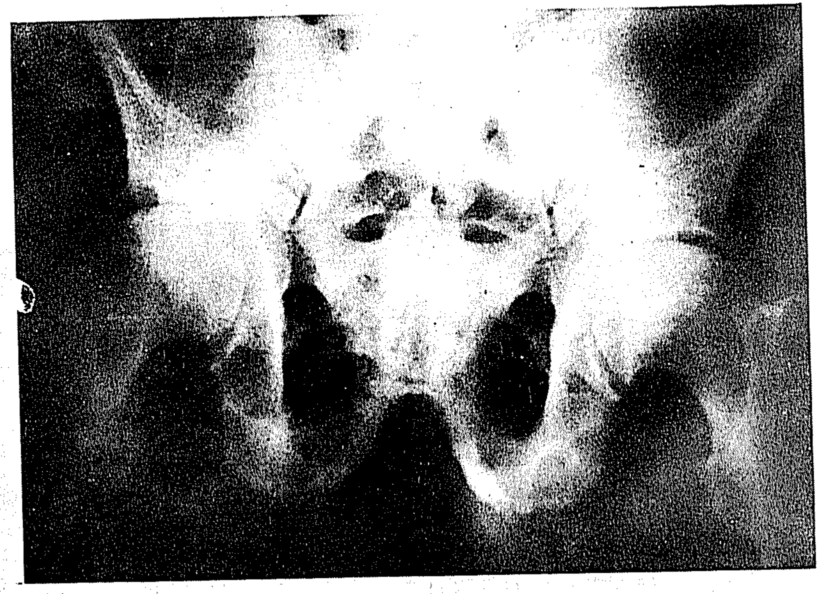


Picture 4 (Case No. 39)

Severe periostal reaction between radius and ulna.

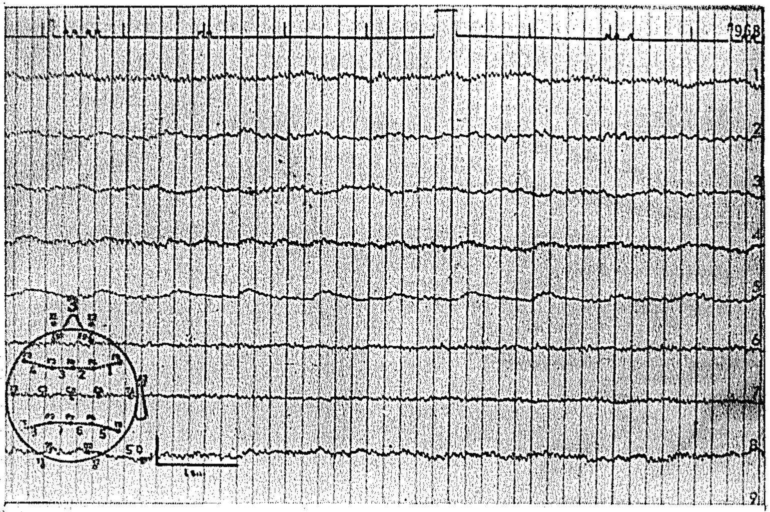
Picture 5 (Case No. 31)

Calcification in the intervertebral areas, severe osteosklerosis on vertebrae (chalky appearance).



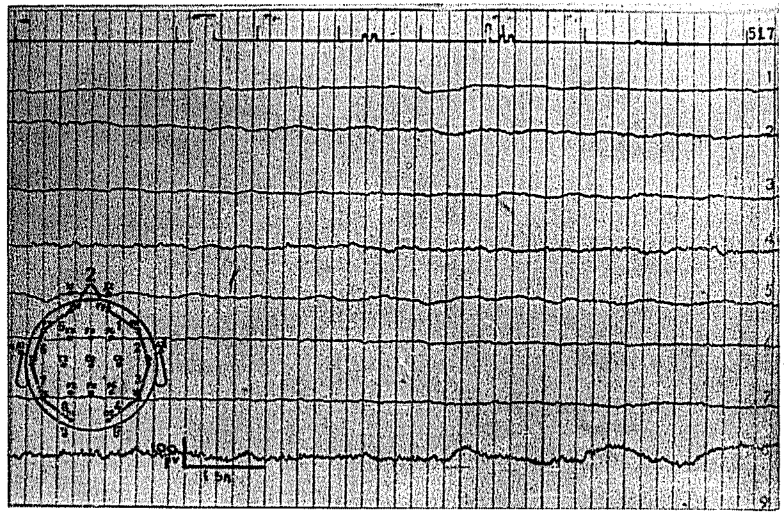
Picture 6 (Case No. 61)

Calcification on sacrospinose and sacrotuberose ligaments.



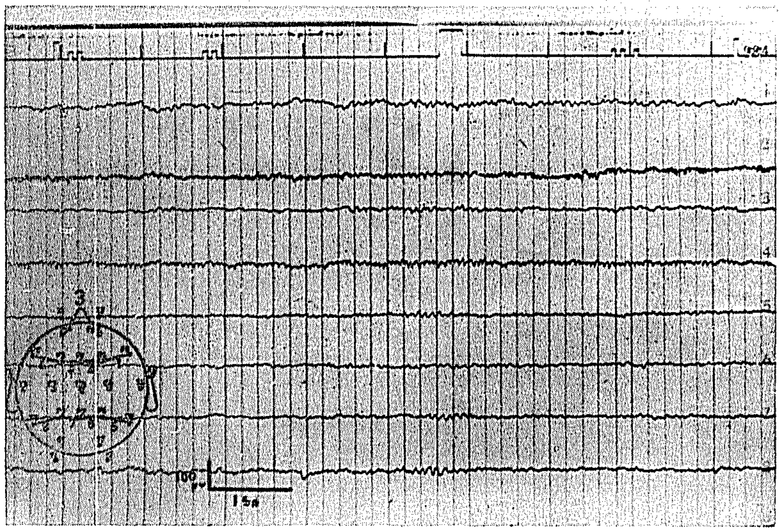
Picture 7 (Case No. 32)

Alfa activity with an amplitude reaching 10-15 m. volt. and beta activities appearing here and there (Flat E.E.G.)



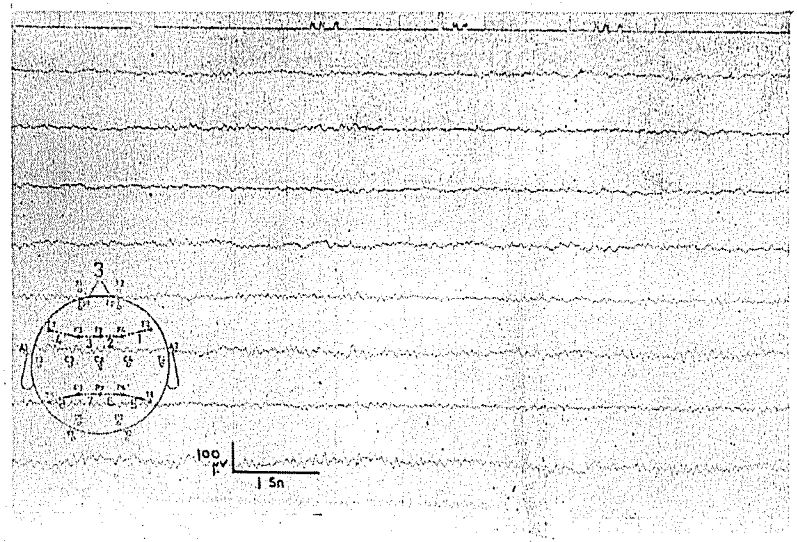
Picture 8 (Case No. 34)

Alfa activity with an amplitude reaching 10-15 m. volt., and Beta activities here and there (Flat E.E.G.)



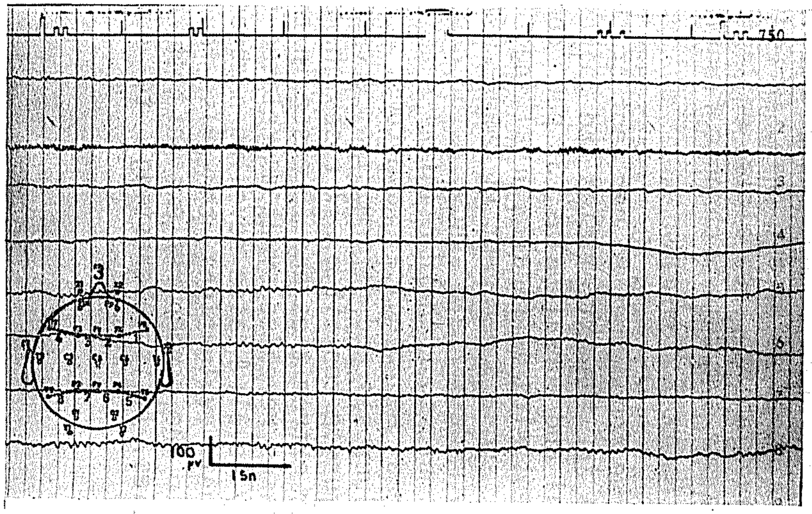

Picture 9 (Case No. 7)

Previous E.E.G. activity, showing a hypo-active pattern of alfa activity reaching to 35-40 m.volt. Here and there, Beta activity is also characteristic.



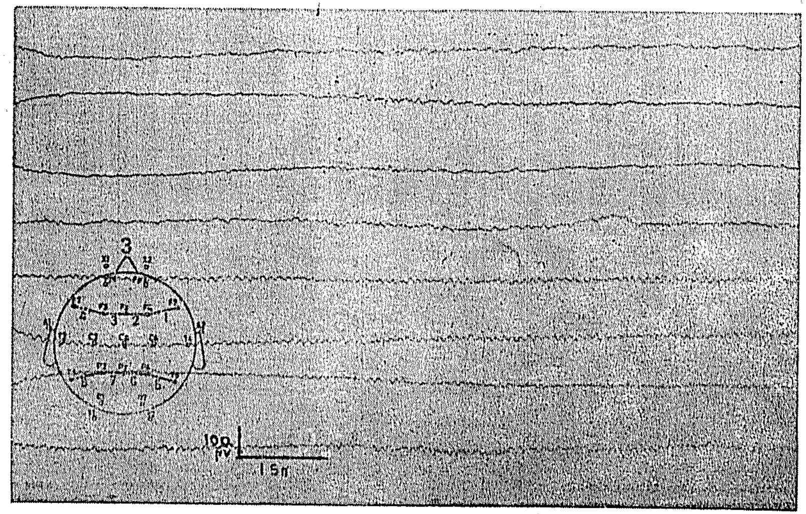

Picture 10 (Case No. 7)

Recent E.E.G. taken two years later, Alfa activity reaching to 60 m. volt. is a chatacteristic. This E.E.G. pattern is within normal acceptance range.



Picture 11 (Case No. 11)

Previous E.E.G. activity; alfa activity reaching to 20 m. volt which is within the acceptance range of flat E.E.G. Here and there, Beta activities are also characteristic.



Picture 12 (Case No. 11)

Recent E.E.G. activity 2 years later. Alfa activity reaching to 40 m. volt. This activity is being accepted hypoactive. Here and there we see Beta activities also.

## Discussion

As shown in Table 1, we did not present the water fluoride content of some villages. As far as we know, and we have observed in the area, the villagers were using Sarısu which contains fluoride up to 12 p.p.m. However, today, except for Gökçekaynak and Yılanlı villages, others are using drinking waters containing fluorides at least on the level of 3.5 p.p.m.(2,3).

In 68 cases showing chronic cryolite intoxication, Waldbott observed 80.9% gastric problems with symptoms of nausea, vomiting and loss of apetite. In this group, he also found 33.8 per cent intestinal problems such as predisposition to diarrhea and constipation. Symptoms related to muscle, joint and bone pains simulating rheumotoid arthritis were present in 35.3 per cent of the cases. According to Waldbott, 22.1 per cent of these cases showed neurological symptoms and 11.8 per cent skin problems(4). Frada and Mentesana also point out the gastrointestinal problems of chronic fluoride intoxications(5). Sıddıqui found that 7 out of 32 of the cases with crippling fluorosis due to chronic fluoride intoxication, showed hearing problems(6). Spira and Waldbott point out that, in addition to the symptoms given above, neuralgia, epileptic seizure and alopesy are common symptoms in chronic fluoride intoxication (7,8,9).

In our study group, we found that 95 per cent of the cases suffered with pains in the extremities and joints; 55 per cent with cervical and dorsal pains; 35 per cent with headaches; 30 per cent with difficulty in walking; 15 per cent with lassitude; and 2.5 per cent (1 case) with seizure. Singh and Jolly made a field study of 60 cases with obvious fluorosis symptoms and their clinical examinations revealed that 27 had neurological symptoms. 25 cases showed crippling fluorosis and deformities; 4 cases had extreme increases in muscle tonicity; 17 cases showed involvements in the lower extremities and 10 cases in the upper extremities. 9 cases revealed symptoms related to compression at a medullary level; 4 cases revealed numbness in certain areas. In their evaluations, they also found an increase or decrease in the tendon reflexes of both the upper and lower extremities. It is interesting to note that 22 cases revealed Babinsky sign. 10 cases had kyphosis, and there were limitations in the movements of columna vertebralis(10).

Jolly in Patiata-India, examined 358 persons with definite fluorosis symptoms. 62 of these cases had myeloradiculopathy and radicular compression was obvious in clinical symptoms(11).

In a joint study, Singh and Jolly found definite paraplegia in 21 of 46 cases with extreme fluorosis(11).

In another study, Singh, Jolly, Bansal and Mathur observed 42 cases with neurological complications in a group of 409 persons showing extreme fluorotic signs such as Babinsky (37 cases), changes in the tendon reflexes, kyphosis, etc. (7).

Jolly, Singh and Singh, in a field study of 6,022 persons with fluorisis, found that 624 persons showed neurological symptoms and out of those cases, 200 revealed paraplegia(12).

Jolly, Singh et al, in 1,065 cases with skeletal fluorosis found 624 crippling fluorosis. The number of cases showing deformities and neurological symptoms reached 855 (13). Contrary to the findings of various research workers, we found neurological complications in 92.5 per cent of our cases. Of the cases we examined, the tendon reflexes were increased, decreased or lost in 75 per cent. In 2 of the cases we also observed pathological reflexes.

Singh, Jolly, Bansal and Mathur, in 42 cases with neurological complications found muscle atrophy in the hand muscles whereas we found diffuse muscular atrophy involving the upper and lower extremities together or alone in 75 per cent of the cases we examined. Singh and Jolly also stated that they found an increase in the muscle tonicity in almost all the cases they examined. Contrary to their findings, we have seen an increase in the muscle tonicity of 17 cases and a decrease in 6.

In connection with the kyphosis, Singh and Jolly found this symptom in 10 cases in a study group of 60 persons.

Singh, Jolly, Bansal and Mathur observed kyphosis in 10 cases of the 42 cases they examined. We found 15 cases with kyphosis out of the 40 cases we evaluated.

In addition to these findings, different from the literature, we have observed pectum excavatum in 2 of our cases, X-leg in 3 cases, and O-leg in 7 of our cases, but we were unable to find any genu valgum as observed by Raddy and Raju[14].

As far as walking difficulties are concerned, various researchers found this problem, probably in the form of paraplegia and crippling. The percentage varied from 25 to 45 per cent in the literature we reviewed[10,15, 7,13, 12]. The cases we observed with walking difficulties were at a level of 27.5 per cent.

In terms of the laboratory test results Shortt, Pandit and Raghavachari found in ten chronic fluorosis cases, which they evaluated, the blood glucose level was within normal limits in 6 cases. 2 cases showed increased glucose levels and in two cases the glucose level was below normal. Calcium levels of the cases were elevated in 5 cases, decreased in 2 cases and normal in 3 cases. Serum phosphorus levels were high in 4 cases, low in 1 case and normal in 5 cases; serum alkaline phosphatase activity was normal in 5 cases and elevated in the rest of the group. Serum natrium levels were elevated in 3, decreased in another 3 and normal in 4 cases. Serum potassium levels were found to be increased in 2 cases, decreased in 6, and normal in 2[16].

Contrary to Shortt, Pandit and Raghavachi's findings Chhuttani, Wahi and Singh found the electrolytes normal in 10 of the cases they studied[17].

When we compare our laboratory findings with Shortt, Pandit and Raghavachi's we are unable to find any change in terms of the blood glucose level. These research workers had found an elevated glucose level in two of the cases they studied. They also found the serum calcium level elevated in 50 per cent of their cases and a lowered calcium level in 20 per cent of the cases. Whereas we found an elevated calcium level in 27.5% and a lowered calcium level in 5% of the cases we studied. Shortt et al, also stated that they found the serum phosphorus level increased in 40 and decreased in 10 per cent of the cases. We found a decreased phosphorus level in 17.5 per cent of our cases and no elevation at all.

In an evaluation of the serum natrium level, Shortt et al found an elevated natrium level of 30%. However they also found a decreased serum natrium level in 30% of the cases which is similar to our findings. We only found 2.5% increase in all the cases we studied.

The serum potassium levels of fluorosis show a marked difference from Shortt's findings. They found an elevated potassium level in 20 per cent of their cases and a lowered level in 60 per cent, we only found a decreased serum potassium level in 5% of our cases and the rest came within normal limits.

According to Leona, Shimkin et al, the hematocryt values in 92 cases showing fluorosis symptoms, the number of neutrophyls were elevated(18) and we were unable to find any change in hemotocryt values in our study group.

In the literature we reviewed, we did not come across work related to the rheumotoid factor. For this reason we are unable to discuss our findings comparatively.

Chronic fluorosis definitely causes some skeletal changes. Latham and Grech, in their roentgenologic evaluations found skeletal changes in 87% of the cases (112 cases). These changes include: Paravertebral ligament calcification (20%); osteophytic appearance on the vertebrae (45%); calcification on the muscle attachments (26%) (9).

In their roentgenologic evaluations, Singh, Jolly, Bansal and Mathur also found moderate to severe skeletal changes in cases with neurological symptoms(7).

Vittori and Pinet had also concluded that chronic fluorosis caused thoroxic and vertebral osteoschlerosis at a level of 30% in the cases they examined(15).

According to Waldbott, osteoschlerotic changes in the skeleton appear in 45% of the cases showing fluorosis.[4].

Our findings related to osteoschlerosis are more or less similar to those in literature. However, our study group also found calcanel spina. In addition to this finding, we also observed a thickening of the skull bones in two of our cases.

In connection with the E.E.G. evaluations of chronic fluorosis. Waldbott assumes that E.E.G. changes may occur due to a decrease in the serum calcium level[4]. Singh, Bansal, Jolly and Mathur stated that they were unable to detect any E.E.G. changes in chronic fluorosis[7]. Contrary to these findings we found 35% of the cases normal. 32.5% flat, 27.5% hypoactive, 2.5% had persistent Beta activity and 2.5% pathologic E.E.G. (with a pattern of delta and tetha waves).

14 cases in our study group were re-evaluated with respect to possible E.E.G. changes. These people have been drinking water from sources containing optimal fluoride or significantly lower fluoride containing water for almost 4 years. 10 of these cases showed a significant improvement in the E.E.G. activities. There was a marked improvement in their locomotions.

Singh, Jolly et al found in their evaluations of persons with chronic fluoride intoxication, that the serum calcium levels were from 9.2 to 13.5 mgm %. On the other hand the same research workers determined the serum phosphorus levels varying from 1.9 to 5.5 mgm%. These findings indicated that elimination of phosphorus by means of urine seemed to increase in cases showing chronic fluoride intoxication. The eliminated phosphorus level reached up to 234mg%, starting from 150 mgm%. As far as calcium elimination through urine is concerned there seems to be no change (7).

It is obvious from the results of this study that some cases with chronic fluoride intoxication may need phosphorus supplementation as after a while a negative phosphorus balance will be established. This point has been shown in animal experiments by Gabovich and his co-workers[19]. In our study we found a decrease of phosphorus in the serum of 7 of our cases (17.5%), we may assume that this type of change in the serum phosphorus level might deteriorate Cax P equilibrium, and thus in return cause an increase of the serum calcium level which simulates hyperparathyroidism. This speculation fits more or less to Cannel's findings (20).

In our evaluation of serum alkaline phosphatase activity we found an increase of 27.5% in our cases. This finding agreed with Weidmann and his co-worker's findings. Contrary to this finding, Singh, Jolly and others found no significant change in the serum alkaline phosphatase activity of chronic fluorosis[7].

In order to determine the rheumotoid factor (R.F.), we used the latex fixation test on the cases we studied. The test resulted in a 37.5% R.F. positive in the whole group. As far as we know R.F. is an indicator of the collagen breakdown and is related to the inhibition in collagen biosynthesis. In-vitro studies on bone cultures reveal that even if fluoride exists at as low as 0.2 p.p.m. the collagen breakdown increases and the collagen biosynthesis diminishes[21].

This finding definitely reinforces our finding related to R.F. in chronic fluorosis. It is very interesting that serum electrolytes have changed in R.F. positive cases; in six of fifteen positive R.F. cases, the serum electrolytes varied from normal values. E.E.G. patterns of most of these varied; we noted 7 flat, 2 hypoactive, 1 persistent beta activity and 1 pathological E.E.G. appearance in these cases.

Even if it is highly speculative, it is relevant to assume that there seems to be a close relationship between bioelectric activity of the brain and collagen breakdown.

According to Weidmann and Weatherell, the serum alkaline phosphatase activity increases in chronic fluoride intoxication[22]. We also found an increase in the alkaline phosphatase activity of 11 cases (27.5%).

In our evaluations of the serum electrolytes some decrease of natrium in serum of 30% of the cases was revealed. E.E.G. activities in the cases which show a decrease in their serum sodium level are markedly different from normal. We found 5 hypoactive and 4 flat E.E.G. activity in these particular cases. As far as we know from classical physiology, an increase or a decrease in the serum natrium level of extracellular fluid did not have any effect on the membrane activity of the neurons. If the situation was such, then for the time being we are not able to discuss further the aspect of chronic intoxication. We also found some decrease in the serum potassium level of 5% of our cases. A serum potassium level may have an effect on the E.E.G. activity. In two of our cases we had 1 flat and 1 hypoactivity. A decreased level of potassium anion in extracellular fluid may cause hyperpolarization on the neuron membrane, this in turn may increase the membrane activity which manifests itself as a lowered bioelectric activity[23].

We found the serum calcium level elevated in 11 cases (27.5%), elevation of calcium also seems to have had a reverse effect on the E.E.G. activity as we noticed 3 flat, 5 hypoactivity, pathologic E.E.G. patterns and only 2 normal E.E.G. activity among the cases. This situation may be seen by the changes taking place on the neuron membranes, it is highly probable that an increased level of calcium may cause membrane polarization which in turn results in the inhibition of impulse generation.

Statistical analyses were also made in order to reveal the relations among various factors. The only statistically significant difference has been found between E.E.G. findings and sex. Females seem to be less affected by chronic fluorosis than males (significance level $\alpha = 0.01$). In terms of their E.E.G. patterns, women have normal or close to abnormal E.E.G. patterns compared to men.

Preventive Measures

It is of prime importance to take preventive measures in order to alleviate the problem to some extent. We may consider home defluoridation units as a possible preventive measure. Bone charcoal seems to be a very effective material in the defluoridation process. The pilot study done

BY Taves in the area, disclosed that bone charcoal was an effective means of reducing fluoride levels in drinking water[25].

Supplementary vitamin C may also alleviate the detrimental effects of fluorosis. This may be helpful particularly in collagen biosynthesis which seems to be inhibited with fluoride intoxication.

The basic solution to the problem is definitely to supply this community with new water resources which contain optimal or close to optimal fluoride.

## Conclusions

29 males, 11 females from the Doğubeyazıt region where the drinking water contains fluoride in toxic levels were evaluated from the neurological standpoint. The following conclusions have been drawn from this study:

A. Clinical and roentgenographic conclusions:

- pain in the extremities and joints (95%)
- cervical and dorsal pain (55%)
- difficulties in walking (30%)
- headache (35%)
- lassitude (15%)
- seizures (2.5%)
- decrease, increase, or absence of tendon reflexes (75%)
- pathologic reflexes (5%)
- various muscular atrophy (75%)
- limitations in the joint movements (85%)
- kyphosis (37.5%)

- pectum excavatum (5%)
- X-leg (7.5%)
- O-leg (17.5%)
- axial osteoschlerosis (87.5%)
- extremiter osteoschlerosis (67.5%)
- calcification of the ligaments (12.5%)
- periostal reactions (37.5%)
- thickening of the skull (5%)
- clacaneal spina (17.5%)

B. Laboratory Findings:

- decrease in the total serum proteins (5%)
- decrease in the serum natrium level (30%)
- increase in the serum natrium level (2.5%)
- decrease in the serum phosphorus level (17.5%)
- decrease in the serum potassium level (5%)
- increase in the serum calcium level (27.5%)
- decrease in the serum calcium level (5%)
- increase in serum alkaline phosphatase activity (27.5%)
- R.F. (Rheumotoid factor) positive (37.5%)

C. E.E.G. Findings:

- 1 Flat E.E.G. (32.5%)
- hypoactive E.E.G. (27.5%)

- normal E.E.G. (35%)

- persistent beta activity (2.5%)

- pathologic E.E.G. (2.5%)

- a great percentage of the cases having positive R.F. and disturbed electrolytes show hypoactive or flat E.E.G. patterns

- reevaluated 14 cases at 1-2 year intervals show some improvements (10 cases) in their E.E.G. patterns. These improvements may be attributed to lowered level of fluoride intake

D. Statistical Results:

- there is a significant statistical difference between the E.E.G. activity and sex ($\alpha$ = 0.01 significance level), women show better E.E.G. patterns than men

- there seems to be no relation between osteoschlerosis and blood values, osteoschlerosis seems to be the same among men and women

- there seems to be no relation between age groups and E.E.G. activity

- median age of the group studied is 52.5; there seems to be a tendency towards hypoactive and flat E.E.G. patterns above this age; there also seems to be a tendency towards normal below this median age

- there seems to be a relationship between E.E.G. findings and axial osteoschlerosis at a level of C = 0.4989; the relationship between E.E.G. activities and extremiter osteoschlerosis is on the level of C = 0.35

## REFERENCES


1. Oktay, C.: Personal Communication, Faculty of Dentistry, Atatürk University, Erzurum, Turkey, 1977.

2. Oruç, N.: Doğubeyazıt kazası ve bazı köylerinde kullanılan sularda fluor konsantrasyonu ve önemi, Atatürk Üniversitesi Ziraat Fakültesi, Ziraat Dergisi, C: 4, S: 1, pp: 45-50, 1973.

3. Oruç, N.: Personal Communication, Faculty of Agriculture, Atat rk University, Erzurum, Turkey.

4. Waldbott, G.L.: Incipient Fluorine Intoxication from Drinking Water, Acta Medica Scandinavica, Vol. 156, fasicul 3, pp: 157-168, 1956.

5. Frada, G., Mentesana, G.: Boll. D.I. Soc. Ital. Biol. Sper, 29: p: 750. 1953 (from Waldbott, G.L.: Incipient Fluorine Intoxication from Drinking Water, Acta, Med, Scand. Vol. 156, Fasc. 3, 1956).

6. Siddiqui, A.H.: Fluorosis in Nalgonda District, Hyderabad-Deccan, Brit. Med. Journ. pp: 1408-1413, 1955.

7. Singh, A., Jolly, S.S., Bansal, B.L., Mathur, C.C.: Endemic Fluorosis, Epidemiological, Clinical and Biochemical Study of Chronic Fluorine Intoxication in Punjap (India). Medicina (Baltimore), 42, pp: 229-246, 1963.

8. *Bal ova, G.: Der lanfristige Einfluss von fluoremissionen auf den kinderorganizmus. Med. Lavora, Vol.: 62, n-4, p: 202, 1971.

9. Latham, M.C., et al.: Excessive fluoride intake, A. J.P.H. Vol: 57, No: 4, Ap. 1967.

10. Singh, A., Jolly, S.S.: Endemic fluorosis, Journal of Medicine, New Series 30, No. 120, pp: 357-372, 1961.


* *Sic*

11. Jolly, S.S., Singh, B.M., Mathur, O.C.: Endemic fluorosis in Punjab (India). American Journal of Medicine Vol: 47, pp: 553-561, Oc. 1969.

12. Jolly, S.S., Singh, I., Singh, A.: A Study of Paraplegia in Punjab, J. of the Association of Physicians of India, pp: 14-19, 1960.

13. Jolly, S.S., Singh, I.D., et al.: An Epidemiological Study of Endemic Fluorosis in Punjab, Ind. Jour. Med. Res., 57. p: 7. July, 1969.

14. Reddy, D.B., Raju, G.C.: Fluorosis, J. Indian Med. Association 63(7): pp: 234-235, 1 Oct. 1974.

15. Vittori, E. et al.: A propos d'un cas D'osteopetrose flucree, Lyon Medical, 223, No. 11 pp: 621-624, 1970.

16. Shortt, H.E., Pandit, C.G., Raghavachari, R.S.: Endemic Fluorosis in the Nellore District of South India the Indian Medical Cazette July, pp: 396-398, 1937.

17. Chhuttani, P.N., Wahi, P.L., Singh, S.J. Neurological Complications of Skeletal fluorosis, Indian Med. Ass., 39, p: 61, 1962.

18. Leone, N.C., Shimkin, M.B., et al.: Medical Aspects of Excessive Fluoride in a Water Supply Public Health Reports, Vol. 69, No. 10, pp: 925, Oct. 1954.

19. Gabovich, R.D., Bukhovets, V.I., Ermakova, N.A. (1960), Vrach, Delo, 6, p: 627 (from Fluorides and Human Health World Health Organization Monograph Series No. 59, 1970).

20. Cannel, W.A.: Medical and Dental Aspects of General Fluoridation, London, H.K. Lewis, and Co. Ltd. 1960.

21. Golbu, L., Glimcher, M.J. Goldhaber, P. (1968), Abstracts, Chicago, A.D.A., p: 97, Abst. No. 240 (from Fluorides and Human Health World Health Organization Monograph Series No. 59, 1970).

22. Weidmann, S.M., Weatherell, J.A.: The Skeletal Changes

of Chronic Experimental Fluorosis, J. Path. Bact., 78, pp: 233-241, 1959.

23. Hodgkin, A.L., Horowicz, P.: The Influence of Potassium and Chloride Ions on the Membrane Potential of Single Muscle Fibre, J. Physiol. (London), 148, pp. 127-160, 1959.

24. Tan, U.: Personal Communication, Faculty of Medicine, Department of Physiology Atatürk University, Erzurum, Turkey, 1977.

25. Taves, D.R.: Personal Communication, Department of Pharmacology and Toxicology, School of Medicine, Rochester University, U.S.A., 1977.

# THE pH VALUES AND FLUORIDE CONTENT

# OF MACO WATER SOURCES

by C. Gholamhosseiny,
Faculty of Pharmacy,
Hydrology Department,
Azarabadegan University,
Tabriz, Iran.

The purpose of this study is to determine the fluoride concentration in Maco water sources, and to find out the relationship between pH value and fluoride content of waters.

## Location

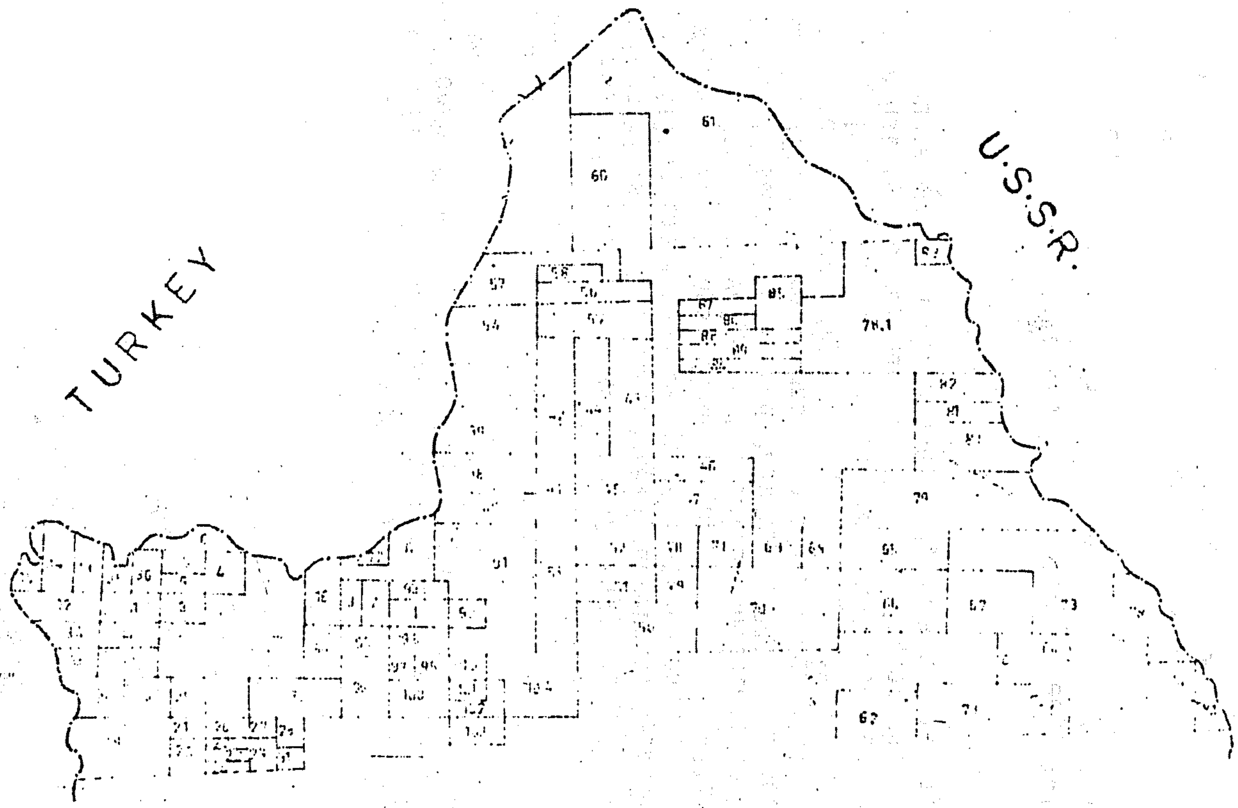
Maco, the northernmost city of Iran, is situated on the foot hills of Ararat Mountain surrounded by 530 villages. All of these villages have their own natural source of water, i.e. springs, rivers, aqueducts and wells (1). Determination of pH values and fluoride concentrations were made on 117 samples obtained from about 350 water sources from the villages around Maco. No other industrial pollution has been observed in the water sources other than that of Aras River (Table 1, code No. 78-1) which suffers badly with industrial contaminants. The location of the Maco water source which has been analysed, is shown in Fig.1.

## Method

Water samples collected in chemically clean, 1-liter polythene-stoppered bottles, were analysed for their pH and fluoride content.

The pH values were determined by Orion Research Ion Analyser - 140 with a glass electrode at 20°C.

Fluoride concentration was determined by the SPADNS method[2], This method: 5,000 mg/l alkalinity as $CaCO_3$, 16 mg/l phosphates, 1 mg/l hexametaphosphate, 200 mg/l sulfates, 7,000 mg/l chlorides, 0.1 mg/l aluminium or 10




Fig$_1$ – The location of Maco water sources.

TABLE 1

THE pH VALUES AND FLUORIDE CONTENT OF THE WATER SOURCE OF VILLAGES AROUND MACO

| No. | *Code | **Location | ***Water Source | pH(20°C) | $F^-$(ppm) |
|---|---|---|---|---|---|
| 1 | 4 | Bedowli | R. Bedlowli | 8.3 | 0.00 |
| 2 | 11 | Beyg Kandi | R. Bedowli | 8.3 | 0.00 |
| 3 | 13 | Pir Ahmad Kandi | S | 6.7 | 0.00 |
| 4 | 15 | Soleyman Aghel | S | 7.9 | 0.00 |
| 5 | 17 | Arab Dizaj | S | 7.0 | 0.00 |
| 6 | 23-1 | Arkhashan | S | 7.7 | 0.00 |
| 7 | 32 | Tahmaseb Kandi | S | 8.4 | 0.00 |
| 8 | 33 | Nadow | S | 8.0 | 0.00 |
| 9 | 78-1 | Poldasht | R. Ras | 7.8 | 0.00 |
| 10 | 31 | Seffow | S | 7.6 | 0.07 |
| 11 | 41 | Guri Shakkak | S | 7.1 | 0.08 |
| 12 | 14 | Jamal Kandi | S | 8.0 | 0.08 |
| 13 | 25 | Halhal-e-Sofla | S. Gara Su | 7.3 | 0.11 |
| 14 | 3 | Bash Kand | A.20m | 7.8 | 0.15 |
| 15 | 12-2 | Giyan Kandi | S. Zay Su | 6.6 | 0.15 |
| 16 | 18 | Qoli Dizaj | S | 7.3 | 0.15 |
| 17 | 20 | Arab Kandi | S | 7.4 | 0.15 |
| 18 | 23-3 | Arkhashan | S. Sayed Sadr | 7.8 | 0.15 |
| 19 | 34 | Sufi Ali | S | 7.4 | 0.15 |
| 20 | 69-1 | Hasoon-e-Bozorg | S | 7.6 | 0.15 |
| 21 | 80 | Pezi | S | 8.4 | 0.15 |
| 22 | 21 | Jang Tappeh | S | 7.1 | 0.17 |
| 23 | 23-2 | Arkhashan | W.5m | 7.4 | 0.17 |
| 24 | 19 | Delak Verdi | S | 8.8 | 0.18 |

| No. | *Code | **Location | ***Water Source | pH(20°C) | $F^-$(ppm) |
|---|---|---|---|---|---|
| 25 | 10 | Kalisa Kandi | S | 6.7 | 0.23 |
| 26 | 49 | Tarah Kameh | S | 7.4 | 0.25 |
| 27 | 100 | Baghchejug | S | 7.8 | 0.25 |
| 28 | 97 | Kusij | S | 7.7 | 0.30 |
| 29 | 30 | Jabbarlou | S | 8.2 | 0.30 |
| 30 | 1 | Keshmish Tappeh | W.20m | 7.8 | 0.33 |
| 31 | 9 | Ali Abad | S | 8.1 | 0.33 |
| 32 | 12-2 | Qiyan Kandi | S | 7.1 | 0.33 |
| 33 | 16 | Mara Kumi | S | 7.8 | 0.33 |
| 34 | 24 | Shegefti | S | 7.8 | 0.33 |
| 35 | 61 | Qush | R. Gara Su | 8.3 | 0.33 |
| 36 | 69-2 | Hasoon-e-Bozorg | A.24m | 8.3 | 0.33 |
| 37 | 81 | Bahlul Kandi | W.10m | 8.9 | 0.35 |
| 38 | 82 | Zakarlu | W.10m | 6.9 | 0.36 |
| 39 | 99 | Mohammad Kandi | S | 8.3 | 0.36 |
| 40 | 103-1 | Qarah Khach Olya | S | 8.1 | 0.36 |
| 41 | 50 | Rend | S | 7.7 | 0.41 |
| 42 | 79 | Qareh Khojehlou | S | 7.8 | 0.41 |
| 43 | 88 | Mowlik-e-Sofla | S | 8.1 | 0.43 |
| 44 | 103-2 | Qareh Khach-Olya | S | 7.8 | 0.43 |
| 45 | 46 | Injeh-ye-Sofla | S | 7.6 | 0.43 |
| 46 | 22 | Orush Kandi | S | 7.6 | 0.48 |
| 47 | 35 | Salman Abad | S | 7.1 | 0.50 |
| 48 | 47-2 | Tatar | A.20m | 7.8 | 0.50 |
| 49 | 2 | Gachut | S | 8.2 | 0.52 |
| 50 | 6 | Jawzar | S | 7.7 | 0.52 |
| 51 | 7 | Alow Jenni | S | 8.2 | 0.52 |
| 52 | 42 | Tikman | S | 7.9 | 0.52 |

| No. | *Code | **Location | ***Water Source | pH(20oC) | F⁻(ppm) |
|---|---|---|---|---|---|
| 53 | 54 | Qeshlag-e-Garik | S | 7.7 | 0.52 |
| 54 | 83 | Zanganeh | W.50m | 7.6 | 0.52 |
| 55 | 102 | Galehjug | S | 7.7 | 0.52 |
| 56 | 93 | Tazeh Kand | S | 8.6 | 0.53 |
| 57 | 40 | Kharman Yeri | S | 6.9 | 0.56 |
| 58 | 39 | Yarim Qayeh-ye-Olya | S | 7.9 | 0.60 |
| 59 | 45-1 | Adaghan | S. Shor Su | 6.1 | 0.60 |
| 60 | 8 | Agh Bolag | S | 7.7 | 0.61 |
| 61 | 48 | Hasoon-e-Kuchek | A.18m | 8.0 | 0.61 |
| 62 | 86 | Gom | S | 7.7 | 0.61 |
| 63 | 98 | Nayeb Kandi | S | 7.9 | 0.61 |
| 64 | 38 | Yarim Qayeh-ye-Sofla | W.8m | 8.1 | 0.65 |
| 65 | 53-1 | Danalo-ye-Bozorg | S.West | 7.3 | 0.65 |
| 66 | 53-2 | Danalo-ye-Bozorg | S.Midle | 7.3 | 0.65 |
| 67 | 5 | Qezil Bolak | S | 7.3 | 0.69 |
| 68 | 36 | Nefto | S | 7.6 | 0.69 |
| 69 | 29 | Shah Bandlu | S | 7.6 | 0.78 |
| 70 | 68 | Makhand | S | 7.7 | 0.78 |
| 71 | 71 | Mahmud Agheli | W.10m | 8.2 | 0.78 |
| 72 | 56 | Qeshlaq-e-Haji | A.10m | 8.2 | 0.87 |
| 73 | 60 | Gadai | S | 8.2 | 0.87 |
| 74 | 84 | Gareh Bolak | W.50m | 7.8 | 0.87 |
| 75 | 101 | Padegan | S | 7.6 | 0.87 |
| 76 | 104-2 | Maco | S. Ag. Su | 7.8 | 0.87 |
| 77 | 44 | Isa Khan | S | 7.7 | 0.88 |
| 78 | 104-1 | Maco | S. Qareh Su | 8.9 | 0.89 |

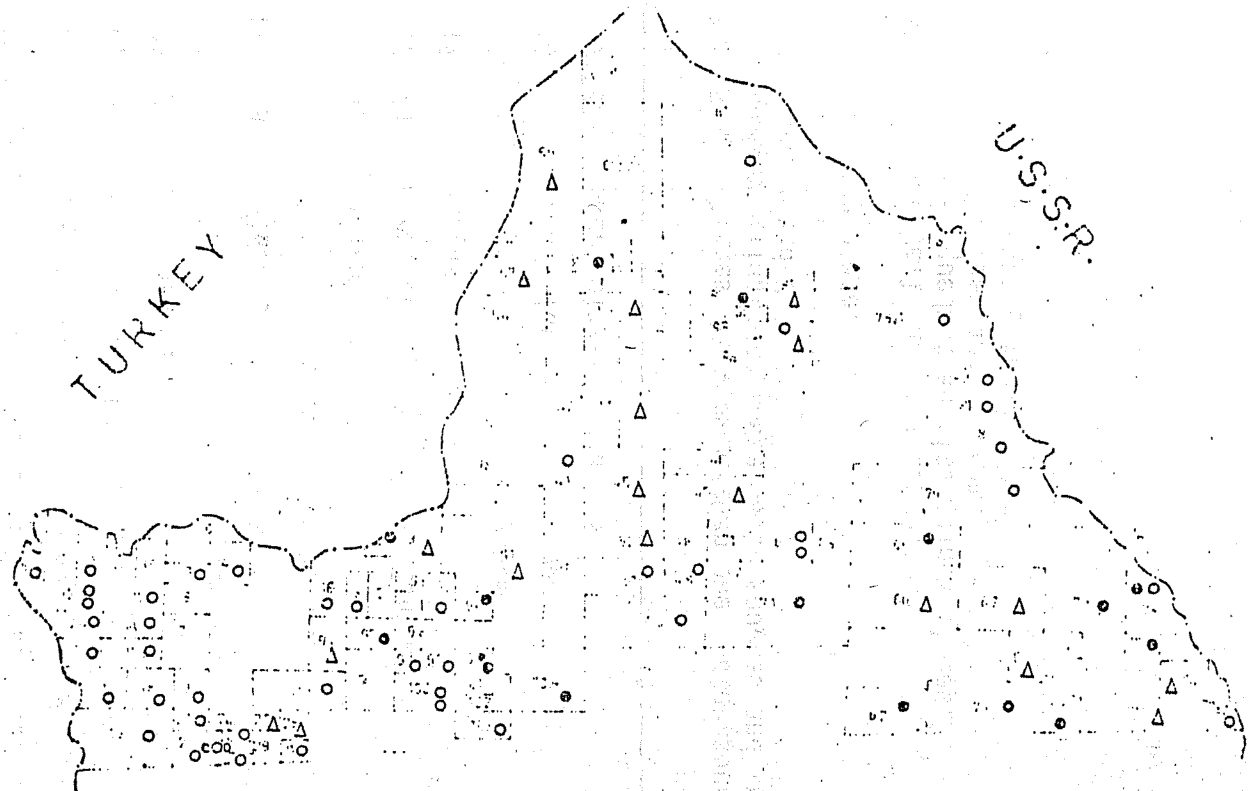
| No. | *Code | **Location | ***Water Source | pH(20°C) | $F^-$(ppm) |
|---|---|---|---|---|---|
| 79 | 26 | Halhal-e-Olya | S | 7.2 | 0.95 |
| 80 | 96 | Pare Khodik | S | 7.9 | 0.95 |
| 81 | 94 | Gory Bolak | S | 7.7 | 1.01 |
| 82 | 74 | Nazar Khan | W.10m | 8.2 | 1.04 |
| 83 | 37-1 | Bazargan | S. Shor Su | 7.6 | 1.05 |
| 84 | 89 | Mowlik-e-Olya | S | 8.3 | 1.05 |
| 85 | 57 | Mus | S | 8.4 | 1.07 |
| 86 | 27 | Shadlu-ye-Sofla | S | 7.7 | 1.13 |
| 87 | 43 | Galleh Zaghesi | S | 8.3 | 1.14 |
| 88 | 66 | Shor Bolak-e-Olya | S | 7.7 | 1.16 |
| 89 | 37-2 | Bazargan | S | 7.6 | 1.19 |
| 90 | 45-2 | Adaghan | S | 7.6 | 1.24 |
| 91 | 28 | Qorban Kandi | S | 8.1 | 1.24 |
| 92 | 75 | Gotan | W.60m | 8.2 | 1.25 |
| 93 | 63 | Eshg Abad | W.11m | 8.9 | 1.25 |
| 94 | 52 | Kahriz | A.20m | 8.8 | 1.31 |
| 95 | 47-1 | Tatar | S | 7.8 | 1.38 |
| 96 | 91 | Shater | R. Sari Su | 8.3 | 1.40 |
| 97 | 55 | Qeshlag-e-Shurik | S | 7.6 | 1.45 |
| 98 | 67 | Gulus | A.7m | 7.8 | 1.45 |
| 99 | 51 | Sheytan Abad | R. Sari Su | 8.5 | 1.45 |
| 100 | 70-2 | Qara Tapeh | R. Sari Su | 8.6 | 1.45 |
| 101 | 85 | Agh Goul | S | 7.8 | 1.48 |
| 102 | 59 | Haji Hasan | S | 8.5 | 1.50 |
| 103 | 58 | Tajdo | A.8m | 8.5 | 1.56 |
| 104 | 70-1 | Qara Tapeh | W.15m | 8.6 | 1.57 |
| 105 | 64 | Sari Su | R. Sari Su | 8.3 | 1.57 |

| No. | *Code | **Location | ***Water Source | pH(20$^{o}$C) | F$^{-}$(ppm) |
|---|---|---|---|---|---|
| 106 | 77 | Djamal Abad | W.38m | 8.3 | 1.59 |
| 107 | 95 | Mostafa Qalesi | S | 7.6 | 1.60 |
| 108 | 72 | Bazargan Marz | S | 8.0 | 1.60 |
| 109 | 104-3 | Maco | R. Zangmar | 8.2 | 1.60 |
| 110 | 78-2 | Poldasht | S | 7.7 | 1.66 |
| 111 | 87 | Shur Aghel-e-Gader | W.8m | 7.9 | 1.66 |
| 112 | 92 | Millan | S | 7.9 | 1.66 |
| 113 | 73 | Moradlou | R. Zangmar | 8.1 | 1.68 |
| 114 | 90 | Sangar | S | 8.1 | 1.68 |
| 115 | 76 | Tapeh Bashi | W.50m | 8.0 | 1.75 |
| 116 | 62 | Yula Galdy | W.80m | 8.4 | 1.77 |
| 117 | 65 | Shur Bolak-e-Sofla | S | 8.0 | 1.79 |

* Some villages have more than one water source and the code numbers illustrate these water sources and their location in Figs. 1 and 2.

** Sampling place.

*** Key: W - Well and its depth.
A - Aqueduct and its main well depth.
R - River and its name.
S - Spring and its name




Fig$_2$-Fluoride content of Maco water sources.

o-Less than 0.5 ppm.

Δ-Between 1–1.5 ppm.

•-More than 1.5 ppm.

mg/l iron can cause interference in the determination of fluoride[2].

To determine the above-mentioned interferences the most sensitive procedures were followed[2,3,4]. Whenever any interfering substance(s) was(were) present in sufficient quantity or whenever the total interfering effect was in doubt, a preliminary distillation step was performed on the samples.

The fluoride content of samples without any interference and those subjected to preliminary distillation were determined by the SPADNS method using a Jonure II spectro photometer at 570 nm. and 20°C. Three standard curves between 0-2, 2-4 and 4-6 ppm $F^-$ were produced. The pH values and fluoride concentration of the Maco villages' water sources are shown in Table 1.

## Results and Discussions

Out of 117 samples from Maco sources, 45 samples contained less than 0.5 ppm, 15 contained 1.5 ppm or more, and the remaining 55 were between 0.5 - 1.5 ppm fluoride content (Table 1). The distribution of these water sources is shown in Fig. 2.

The pH of all the water sources lies between 6.1-8.9. In the samples with the same pH values, the concentration of fluoride is different. Samples with more than 1 ppm fluoride content have a varied pH of between 7.6-8.9, and are thus alkaline (Table 2). This property is possibly due to the presence of major alkaline substances [3,5] and not mainly due to fluoride in spite of the fact that HF($PK_a$ 3.17) acts as a moderately weak acid in aqueous solutions (6) and can affect the pH of alkaline waters.

TABLE 2

THE pH VALUES OF MACO SOURCES
CONTAIN MORE THAN 1 PPM FLUORIDE

| No. | Code | pH(20°C) | $F^-$ (ppm) |
|---|---|---|---|
| 1 | 37-1 | 7.6 | 1.05 |
| 2 | 37-2 | 7.6 | 1.19 |
| 3 | 42-2 | 7.6 | 1.24 |
| 4 | 55 | 7.6 | 1.45 |
| 5 | 95 | 7.6 | 1.60 |
| 6 | 27 | 7.7 | 1.13 |
| 7 | 66 | 7.7 | 1.16 |
| 8 | 78-2 | 7.7 | 1.66 |
| 9 | 94 | 7.7 | 1.01 |
| 10 | 47-1 | 7.8 | 1.38 |
| 11 | 67 | 7.8 | 1.45 |
| 12 | 85 | 7.8 | 1.48 |
| 13 | 87 | 7.9 | 1.66 |
| 14 | 92 | 7.9 | 1.66 |
| 15 | 65 | 8.0 | 1.79 |
| 16 | 72 | 8.0 | 1.60 |
| 17 | 76 | 8.0 | 1.75 |
| 18 | 28 | 8.1 | 1.24 |
| 19 | 90 | 8.1 | 1.68 |
| 20 | 74 | 8.2 | 1.04 |
| 21 | 75 | 8.2 | 1.25 |
| 22 | 104-3 | 8.2 | 1.60 |
| 23 | 43 | 8.3 | 1.14 |
| 24 | 77 | 8.3 | 1.59 |

| No. | Code | pH(20°C) | $F^-$(ppm) |
|---|---|---|---|
| 25 | 89 | 8.3 | 1.05 |
| 26 | 91 | 8.3 | 1.40 |
| 27 | 64 | 8.3 | 1.57 |
| 28 | 57 | 8.4 | 1.07 |
| 29 | 62 | 8.4 | 1.77 |
| 30 | 73 | 8.4 | 1.66 |
| 31 | 58 | 8.5 | 1.56 |
| 32 | 59 | 8.5 | 1.50 |
| 33 | 51 | 8.5 | 1.45 |
| 34 | 70-1 | 8.6 | 1.57 |
| 35 | 70-2 | 8.6 | 1.45 |
| 36 | 52 | 8.8 | 1.31 |
| 37 | 63 | 8.9 | 1.25 |

## REFERENCES


1. Igipo, 1972. Village Gazetteer. West Azarbayjan Ostan. Vol. 2. Iran Statistics Cor. pp: 31-39.

2. Standard Methods for the Examination of Water and Wastewater 1975. 14th ed. APHA. AWWA. WPCF. pp: 387-466.

3. Golterman, H.L. 1972. Methods of Chemical Analysis of Fresh Waters. IBP Handbook No. 8. Blackwell Scientific Pub. Oxford. pp: 28-47-88.

4. Allen, E.S. 1974. Chemical Analysis of Ecological Materials. Blackwell Scientific Pub. Oxford. p:122.

5. Twort, A.C., and Hoather, R.C., and Law, F.M., 1974. Water Supply, 2nd ed. Edward Arnold, p: 184.

6. Shinskey, F.G., 1973. pH and p Ion Control in Process and Waste Streams. John Wiley Pub. p: 66.

FLUORIDE: ITS OCCURRENCE, ANALYSIS, EFFECTS ON PLANTS, DIAGNOSIS AND CONTROL

by Dr. J.B.D. Robinson
Long Ashton Research Station,
University of Bristol,
and Ministry of Overseas Development,
London.

## INTRODUCTION

Fluoride very rarely occurs naturally in the elemental form and when we consider relationships between this element and plant production we are concerned with the fluoride ion in compounds with other elements. The fluoride ion is widely distributed; it is a natural component of rocks and soils and is present in plant and animal tissues. It is not considered to be an essential element for the growth of plants, but is essential for animals. The margin between safe and toxic levels particularly in animals, is a relatively narrow one.

The pathological effects of excess fluoride in domestic herbivores (dental fluorosis) were recognized in Iceland and described nearly 1,000 years ago; they followed feeding on grass contaminated by ash deposition after volcanic eruption. The adverse, toxic effects on plants were first recognized a little less than 100 years ago (1883) followed by reports of damage to vegetation in areas surrounding superphosphate and glass factories in Germany (1893-1898).

Since the 1940's there has been a greater awareness of and concern about, atmospheric fluoride pollution arising from industry. Whilst plants may tolerate high accumulations of fluoride in the aerial parts particularly the leaves, animals grazing on contaminated forage crops may be adversely affected.

Fluoride is the lightest of the four halogens; it is very chemically active, violently oxidative and potentially explosive in contact with inorganic and organic compounds. It is inert towards oxygen and ozone, attacks water vigorously and combines directly with many metals.

I have attempted to summarize present knowledge and information about the occurrence of fluoride, its chemical determination, its reaction with soils and plants, the toxic symptoms in plants and the methods for preventing or controlling excess fluoride when it is detected.

## THE OCCURRENCE OF FLUORIDE

### Natural Occurrence of Fluoride

General values for naturally occurring fluoride are summarized in Table 1 (Bowen, 1966).

TABLE 1

SUMMARY OF FLUORIDE OCCURRENCE IN NATURE

| MATTER | MEDIAN VALUE OR RANGE (Parts $10^6$ dm) |
|---|---|
| Igneous Rocks | 625 |
| Shales | 740 |
| Sandstones | 270 |
| Limestones | 330 |
| Soils | 200 : 30-300 |
| Land Plants* | 0.5 - 40 |
| Marine Plants | 4.5 |
| River Water | 0.09 |
| Sea Water | 1.3 |
| Air | <0.01 g/m$^3$ |
| Land Animals: Bones | 1500 |
| Soft Tissue | 150 - 500 |
| Marine Animals | 2.0 |

Note: *Excluding accumulators

In Minerals: The fluoride content of some common minerals is quoted by Brewer (1966) in percentages, as follows: Biotite, trace - 4.23; Tourmaline, 0.06-1.19; Muscovite, 0.12-1.26; Apatite, 1.31-3.86; Fluospar, 48.9 and Phlogopite, 0.82-5.67.

In Soils: Some examples of the total fluoride content of topsoils are given in Table 2 and Manley et al. (1975) present a summary of total fluoride values in soils of several other parts of the world.

It seems to be generally established that silt and clay loam soils contain greater amounts of total fluoride

TABLE 2

| Location | No. of Samples | Median Value or Range (Parts $10^6$ ads) | Reference |
|---|---|---|---|
| U.S.A. | 37 | 290: 20-590 | Robinson & Edgington (1946) |
| U.S.A. (Eastern States) | 15 | 240 | Brewer (1966) |
| Whole U.S.A. | na | <100->1,000* | Anon. (1971) |
| Various Tropical Areas | 37 | 131: 68-216 | Hall & Cain (1972) |
| Netherlands | 102 | 39-679 | Eysinga (1974) |
| New Zealand (S. Islands) | 25 | 134: 22-230 | Manley et al. (1975) |

Note: * Higher values probably represent heavily phosphated soils or other contamination.

TABLE 3

EXAMPLES OF TOTAL NATURAL FLUORIDE IN PLANT FOLIAGE

| PLANT TYPE | MEDIAN VALUE OR RANGE (Parts $10^6$ dm) | REFERENCE |
|---|---|---|
| A. NON-ACCUMULATORS | | |
| General | Seldom 20, Most 10 | Eysinga (1972) |
| General | 2-20 | Koster (1972) |
| General | 3-30, Most 10-20 | Istas & Alaerts (1974) |
| General | 2-20 | Brewer (1966) |
| General | 1-15 | Webber (1977) |
| General | 10 or less | McClenahen (1976) |
| Pasture | 5 | Manley (1975) |
| Pasture | 6.8 | Oelschläger et al. (1968) |
| B. ACCUMULATORS (INORG.) | | |
| Camellia japonica | | |
| Older leaf samples | 790-3,062 | |
| Young leaf sample | 67 (single value) | |
| Camellia sinensis (Tea) | | Zimmerman et al. (1957) |
| Domestic dry tea - Indian | 72-115 | |
| " " " China | 131-178 | |
| Greenhouse grown tea leaves | 1,530 | |
| C. ACCUMULATORS (TOXIC ORGANIC) | | |
| Dichapetalum cymosum | 111 | |
| D. stuhlmannii | 142 | |
| D. toxicarium | 145 | |
| Gastrolobium bilobum | 175 | Hall (1972) |
| Oxylobium parviflorum | 116,465 | |
| Palicourea marcgravii | 455 | |

than do lighter, sand soils (e.g. Brewer 1966; Manley et al., 1974). Eysinga (1974) found a very close positive correlation for 102 Netherland cultivated soil samples, between total fluoride and the proportion of clay in the samples. It has frequently been observed that under natural, surface-contamination-free and virgin conditions, fluoride generally increases with profile depth (Robinson & Edgington, 1946; Gorski & Stupnicka, 1961; McClenahen, 1976).

*In Plants*: Median values or ranges of values for total fluoride in plant foliage are summarized in Table 3. As far as it is possible to be certain, plants for which these analyses were made had not been exposed to abnormal levels of fluoride in the soil, water or atmosphere.

Other plant species that are recognized as fluoride accumulators (section B, Table 3), are *Carya cordiformis*, *Carya tomentosa*, *Fagopyrum saggitatum* and *Cornus florida* (McClenahen, 1976).

Eysinga (1972) has tabulated fluoride values for different parts of plants grown under a variety of conditions without specific treatment with fluoride; his data refer to nine vegetable species, maize (*Zea mays*) and gladiolus sp. Further healthy leaf data published for tomato are 23 parts $10^6$ (Prince et al. 1949) and $<8.2$ parts $10^6$ (Leone et al., 1948), navel orange $<10$ parts $10^6$ (Prince et al., 1949), peach 6 parts $10^6$ (Leone et al., 1948) and Robinson & Edgington (1946) presented leaf values for 13 non-accumulator plant species grown in a soil containing 137 (surface) and 172 (subsurface) parts $10^6$ fluoride, of median value 7.4, range 1.7 to 28.3 parts $10^6$ fluoride in the dry matter. The broad general range of uncontaminated foliage fluoride values for non-accumulator crops of commercial significance seems to be quite well established. Nevertheless, a group of particularly fluoride sensitive plants is recognized some of which may serve as indicator crops (Istas & Alaerts, 1974).

*In Water*: Rainwater will contain traces only of fluoride unless it falls through a polluted atmosphere, gaseous or particulate, such as may occur after volcanic eruptions or over an industrial or a coal burning city area.

In New Zealand Manley et al. (1975) reported levels of $<0.05$ (fresh water) and 0.15-0.22 (artesian water) parts $10^6$ fluoride; in Germany Kopf et al. (1968) found a median value for 27 well waters of 0.14 parts $10^6$ fluoride with only an occasional value $>0.2$. They noted that higher fluoride content occurred in limestone than in non-limestone areas (0.128 versus 0.08 parts $10^6$ fluoride); land drainage waters from a silt loam (grey-brown podzolic soil) contained 0.096-0.129 parts $10^6$ fluoride. In the Netherlands tap water, much used in greenhouse culture and not fluoridated contains 0.05-0.45 parts $10^6$ fluoride (Koster, 1972) and Eysinga (1972) reports that fresh water in the Netherlands usually contains $<0.5$ and land drainage waters approximately 0.2 parts $10^6$ fluoride respectively.

There are areas, often with a recent volcanic geology where both surface and shallow groundwaters that are either potential or actual sources for irrigation, contain high levels of fluoride. Walker (1955) and Walker, and Milne (1955) recorded fluoride levels in river waters rising on an extinct volcano in the north eastern part of Tanzania of 1.1 to 45.5 (median 13.0) parts $10^6$. In the Bhilwara district of Rajasthan, India, 118 well waters used for crop irrigation contained a mean (weighted) level of 5.8 with a range of 2.1-24.0 parts $10^6$ fluoride (Paliwal et al., 1969), and Somani (1974) examined 166 well water samples in the Udajpur district of Rajasthan and found a median value of 4.5 with a range of 0.15-21.6 parts $10^6$ fluoride. He noted that the fluoride content of the well water increased with the depth of the well in this region.

In Air. Reference to Table 1 shows that uncontaminated air contains little more than a trace of fluoride, but it is difficult to locate published figures for apparently uncontaminated or normal air measured for agricultural purposes. Istas & Alaerts (1974) do quote a range of $10^{-7}$ to $5.10^{-6}$ g/m$^3$ for fluoride compounds in uncontaminated air i.e. country conditions, rising to $10^{-5}$ g/m$^3$ or higher in cities.

## Artificial Occurrence of Fluoride

In Fertilizers. Some fertilizers, natural minerals and artificial materials that are applied to soils to increase plant productivity contain appreciable levels of fluoride as an impurity. Examples of the fluoride contents of such materials are given in Table 4.

It will be noted in Table 4 that some of the range values of Swaine (1962) are much wider than figures quoted later in time for similar materials e.g. compare data for basic slag or rock phosphates (Swaine, 1962 and Eysinga, 1972). This is attributed to the wider range of samples and often much older analytical information given by Swaine.

The fluoride impurity in phosphatic fertilizers and that present naturally in phosphate rocks or as a contaminant in industrial by-products or wastes, is comparatively high and it is from these sources that an appreciable quantity of fluoride may get into regularly fertilized soils, potting mixtures and horticultural growing media, e.g. peat, compost, (Oelschläger, 1968; Kudzin & Pashova, 1970; Larsen & Widdowson, 1971; Eysinga, 1971, 1972, 1974; Stewart, et al., 1974). Commercial phosphate rocks all contain combined phosphorus and fluorine in apatite mineral structures associated with clacium (McClellan & Lehr, 1969; Lehr & McClellan, 1972, 1973). Bovay et al. (1969( reported enhanced crop uptake of fluoride from borinated compound fertilizers attributed to the presence of $KBF_4$.

Data for the fluoride content of fertilizers manufactured from specific phosphate rock are shown in Table 5.

<u>In Air</u>. The damage that can be caused to trees, agricultural and horticultural crops through exposure to air containing fluorides, particularly in gaseous form, is generally recognized by the industrial nations. Atmospheric pollution with fluorides occurs wherever natural materials such as coal, clays, rock minerals or ores are heated or when fluoride fluxes are used and waste products either escape or are vented into the atmosphere. Sources of such pollution have been listed by MacIntire & Associates (1949), Treshow (1971), Istas & Alaerts (1974), as industries concerned with phosphoric acid and superphosphate production, aluminium works, steel foundries, glazing and enamelling works, glass foundries, processing of non-ferrous metals, brick and tile works, fluorinated hydrocarbon and plastics production and the burning of coal.

TABLE 4 EXAMPLES OF THE TOTAL FLUORIDE CONTENT OF FERTILIZERS, RAW MATERIALS AND OTHER SOIL AMENDMENTS

| MATERIAL | NO. OF SAMPLES | MEDIAN VALUE OR RANGE (Parts $10^2$ dm) | REFERENCE |
|---|---|---|---|
| A. NITROGEN FERTILIZERS | | | |
| Ammonium Sulphate | 1 | 0.0003 | Eysinga (1972) |
| Calcium Nitrate | 1 | 0.058 | - " - |
| Calcium Cyanamide | 1 | 0.058 | Swaine (1962) |
| Calcium Ammonium Nitrate | 3 | 0.0010-0.0014 | Eysinga (1972), Swaine (1962) |
| B. COMMERCIAL ROCK PHOSPHATES | | | |
| Various | >350 | trace-6.80 | Swaine (1962) |
| Rock Phosphates (General) | ns | 3.0-4.0 | Eysinga (1972) |
| Apatites (Typical Values) | 6 types | 4.09-4.93 | Lehr & McClellan (1972, 1973) |
| U.S.A. Rock Phosphates | 15 | 3.06-3.95 | |
| Morocco " " | ns | ≃4.24 | Sauchelli (1965) |
| Tunisia " " | ns | ≃3.46 | |
| Nauru " " | ns | ≃2.70 | |
| Ocean Islands Rock Phos. | ns | ≃3.08 | |
| Curacao Rock Phosphates | ns | ≃0.74 | Evans et al. (1971) |
| Christmas Island Rock Phosphates | ns | ≃1.51 | |

TABLE 4 (contd..2)

| MATERIAL | NO. OF SAMPLES | MEDIAN VALUE OR RANGE (Parts $10^2$ dm) | REFERENCE |
|---|---|---|---|
| C. PHOSPHORUS FERTILIZERS | | | |
| Single Superphosphate | 245 | 0.30-2.65 | Swaine (1962) |
| " " | ns | 1.50-2.50 | Eysinga (1972) |
| Double " | 84 | 0.75-3.90 | Swaine (1962) Evans et al. (1971) Anon (1974) |
| " " | ns | ≃2.0-2.29:1.80-2.78 | Eysinga (1972, 1974) |
| " " | 5 | 0.80-3.49 | Sauchelli (1963) |
| Diammonium phosphate | ns | 2.0 | Anon (1974) |
| Mixed Compound fertilizers cont. phosphate | 60 | 0.16-3.38 | Swaine (1962) |
| Bone Meal (various) | ns | 0.027-0.066 | Eysinga (1972, Swaine (1962) |
| Basic Slag | 94 | 0.001-1.70 | Swaine (1962) |
| " " | ns | 0.001-0.016 | Eysinga (1972) |
| D. POTASSIUM FERTILIZERS | | | |
| Potassium Chloride 40 | ns | 0.002-0.003 | Swaine (1962), Eysinga (1972, 1974) |
| Kainite | ns | 0.001-0.002 | Swaine (1962), Eysinga (1972) |
| E. CALCIUM MATERIALS | | | |
| Limestone (mixed) | 194 | <0.0005-0.61 | Swaine (1962) |
| Lime Marl | ns | 0.006-0.03 | Swaine (1962), Eysinga (1972) |
| Dolomite | 5 | 0.011-0.04 | Swaine (1962) |
| Gypsum | ns | 0.087-1.0 | Swaine (1962), Eysinga (1972) |

Note: ns = No. of samples not stated.

TABLE 5

EXAMPLES OF FLUORIDE CONTENT IN RAW PHOSPHATE ROCK MATERIAL AND RELATED FERTILIZER

| ROCK PHOSPHATE (A) | FERTILIZER (B) | VALUE OR RANGE (Parts $10^2$ dm) (A) | (B) | REFERENCE |
|---|---|---|---|---|
| Nauru | Single Superphosphate | 2.73 | 1.58 | Evans et al. (1971) |
| Christmas Is. A. | " " | 2.15 | 1.08 | Evans et al. (1971) |
| Morocco | Triple Superphosphate | 3.83-3.98 | 2.22-2.26 | Smallpage (1977) |
| Morocco | Amm. Phosphate (Gran.) | 3.83-3.98 | 0.27-0.48 | Smallpage (1977) |

Natural fluoride levels around large industrial complexes can be raised as much as one thousand fold, to levels in excess of $10^{-4}$ g/m$^3$ (Istas & Alaerts, 1974). These levels may be generally toxic or plants (and, indirectly to animals following particle settlement on or absorption of gases by forage crops) and particularly toxic to highly susceptible plants such as peach or gladiolus varieties.

The forms in which fluoride occurs in a polluted atmosphere are (a) gases - HF, $SiF_4$, $H_2SiF_6$ and $CF_4$ (b) dust particles of NaF, $CaF_2$ (Fluorspar), $Na_3AIF_6$ (Cryolite), $AIF_3$, $Na_2SiF_6$ and $CaSiF_6$ (c) aerosol form containing NaF, $Na_3AIF_6$ and $AIF_3$ (Bovay, 1969; Istas & Alaerts, 1974). Fluoride toxicity to plants is greatest with gas pollution of the atmosphere particularly HF (Compton, 1970; Istas & Alaerts, 1974) and least with particulate matter the toxicity of which will depend upon particle size and solubility; toxicity of any aerosol will depend upon the chemical form of the liquid(s) or solid(s) held in suspension.

In Water. Official fluoridation of public water supplied (0.8-1.6 parts $10^6$) for dental reasons can have adverse effects upon greenhouse flower crop production and upon the appearance and life of some cut flowers in vases (Koster, 1972; Cruyn & Hulsman, 1972). Flowers like freesias grown in hydroponic cultures may be damaged by such concentrations of fluoride in municipal waters (Koster, 1972).

Rainwater may be enriched by dissolving gaseous fluorides or by removing fluoride containing particles from the atmosphere and conveying them to the vegetation and the soil (Davison et al., 1976). MacIntire & Associates (1949) reported fluoride levels in rainwater increased from 0-0.02 to 0.06-1.23 parts $10^6$ in the vicinity of a soft coal-burning town. Conversely, there will be situations in which crops already polluted with particulate fluoride from the atmosphere will have some of it washed off and even leaf fluorides leached out, by rainfall which will transfer it to the soil. In times of drought and in arid areas irrigation with water containing fluorides will directly increase soil (surface irrigation) or both plant and soil (overhead irrigation) fluorides, but there is little information on this subject.

Zimmerman et al. (1957) reported that an infusion of commercial tea (1 tea bag to approximately 128 ml water) raised the fluoride level in the water by 0.8-1.7 parts $10^6$.

In Other Materials. Fluoride may occur in wood preservatives, herbicides, pesticides and glass cleaners (Eysinga, 1972). They may represent local pollutant potential and an awareness of possible side effects is always necessary when handling such materials; in many cases when handled and applied correctly, the quantity of fluoride involved may be quite small. For example, the application of Norfluorazon selective herbicide ($C_{12}H_yClF_3N_3O$) in cotton at a rate of 2 Kg a.i./ha would add only 375 g per application.

## FLUORIDE ANALYSIS METHODS

The determination of fluoride in agricultural samples has been greatly facilitated by development of the fluoride ion specific electrode e.g. Phillips IS 550F; Beckman Select Ion 39650 and 39600, Orion 94 - 09, which has largely replaced titrimetric or spectrophotometric methods.

### Soil

The value most frequently determined is total fluoride (Brewer, 1965, 1966; Hall, 1968; Hesse, 1971; McClenahen & Schulz, 1976) followed by water soluble fluoride (Brewer, 1965, 1966; Hall, 1968; Hesse, 1971). Since only the soluble soil fluoride is available to the plant it is assumed that total and soluble levels are closely related though there is little published evidence on this point. More recent determinations of acid labile (Hall, 1968; Hall & Cain, 1972) and resin absorbed fluoride (Larsen & Widdowson, 1971; Eysinga, 1974) have been made as more direct measures of plant available soil fluoride.

There is some difference of opinion about the comparative merits of single (McClenahen & Schulz, 1976) versus double acid distillation (Brewer, 1965, 1966; Hesse, 1971) or an ashing or fusion technique (Hall, 1968; Hall & Cain, 1972; Suketa et al., 1975) for solubilizing the fluoride totally, though for large numbers of samples the first method is less troublesome, faster and may be generally satisfactory (a simple comparison on representative soil samples will decide this point).

Titrimetric (Brewer, 1965; Hesse, 1971) and spectrophotometric (Hall, 1968; Yamazoe, 1970) techniques for measuring the fluoride concentration have been described as has the newer fluoride-ion specific electrode technique (Verloo & Cottenie, 1969; McClenahen & Schulz, 1976).

Extraction of soluble soil fluoride is usually made with water at soil (air dry) to water ratios not wider than 1:3 though Larsen & Widdowson (1971) used 0.01 M $CaCl_2$ for this purpose.

## Plants

As for soils there are several alternative methods available for bringing fluorides into solution. Hall (1968) recommends careful ashing followed by KOH fusion to ensure conversion of fluorosilicates into fluorides. The more recently described AOAC method involves a simpler acid/alkali extraction (AOAC, 1975; Jacobson & Heller, 1975), but Van Den Heede et al. (1975) describe a low temperature ashing procedure which gave the best recovery in their samples since it included both organic and insoluble inorganic fluorides which are not included in the AOAC determination.

If a fluoride-ion specific electrode is not used in the analysis, details of a spectrophotometric method is given by Hall (1968) or a standard thorium nitrate backtitration technique may be applied after ashing and distillation from perchloric acid (AOAC, 1970), though this is less accurate at low fluoride levels.

## Phosphate Rock and Phosphate Fertilizer

Evans et al. (1970) compared four fluoride solubilisation methods and determination by either colourimetric or fluoride electrode methods for phosphate rocks. They concluded that dilute perchloric acid digestion and the fluoride-ion specific electrode was comparable with and much faster than, the standard method.

With slight modification they developed a solubilisation method coupled with the use of the fluoride electrode for phosphatic fertilizers (Evans et al., 1971).

Rogers (1973) reported fluoride-ion specific electrode methods for phosphate fertilizer plant effluents (gases and scrubber liquors) as well as for phosphate rocks and fertilizers.

## Water

Water samples may be examined directly for soluble fluoride; suitable treatment of free organic fluoride before either colourimetric or fluoride electrode determination, may be taken if necessary.

## FLUORIDE IN SOILS

The fluoride in soils is commonly present in micaceous clays, common fluoro-minerals as cryolite ($Na_3AlF_6$), fluorospar ($CaF_2$), fluorapatite ($Ca_5F(PO_4)_3$) and silliate ($MgF_2$) and is related to the soil parent material; it is present in soil solution or absorbed as ions, and as undissociated compounds (Brewer, 1965, 1966; Bowen, 1966; Eysinga, 1972).

The fluoride content of the soil is increased locally by fluorides from a polluted atmosphere (MacIntire, Sterges & Shaw, 1955; Compton, 1970; McClenahen, 1976), by water used to irrigate crops and by persistent use of phosphatic fertilizers or of phosphate rock applications (Brewer, 1965, 1966; Oelschläger, 1968; Kudzin & Pashova, 1970; Larsen & Widdowson, 1971; Eysinga, 1971, 1974). When superphosphate fertilizer is applied to forage crops for grazing, physical ingestion of fluoride contaminant for a short period after treatment may be more significant than uptake from soil residues via the forage (Stewart et al., 1974). Most of the added fluoride is retained by heavier textured soils whereas light sand soils retain less (Robinson & Edgington, 1946; Brewer, 1966). In acid soils a lesser proportion of fluoride is retained and a greater proportion of that which remains may be in a soluble form (Treshow, 1971). According to him soluble fluorides present in acid soils derived from atmospheric sources may present more of a threat to plants. Retention in acid soils is primarily as aluminium complexes (Bower & Hatcher, 1967), in neutral to calcareous soils containing ionic calcium, fluoride retention is largely in the form of fluorspar (Brewer, 1966; Eysinga, 1972) and fluorapatite. In contaminated soils the neutral pattern of distribution is changed and the surface layers contain more fluoride than there is lower down in the profile (Larsen & Widdowson, 1971; McClenahen, 1976).

Total soil fluoride is not usually related to fluoride uptake by plants (Brewer, 1966; Treshow, 1971; Hall & Cain, 1972; Cooke et al., 1976; Webber, 1977), which is measured in the leaves or less commonly, in the above-ground parts of the plant. Uptake is influenced by many soil factors about which there is an extensive literature; among the more important are pH, phosphate, calcium, clay and organic matter content (Brewer, 1966; Eysinga, 1972; Cooke et al., 1976; McClenahen, 1976), as well as plant factors which are discussed later. The higher the levels of these factors in a soil the more readily is fluoride rendered unavailable.

It seems reasonable to suppose that the soluble, or the labile, rather than the total fluorides in soils will be related closely to plant uptake. Measurements of water-soluble fluorides have been made and examples of levels found are, Belgian soils: 0.1-8.0 parts $10^6$ (Bertrand & Wolf, 1970); English soils using 0.01 M $CaCl_2$: all less than 0.2 parts $10^6$ and this method showed a measurable increase in soluble fluoride for agricultural soils that had received 38 Kg P/ha of fertilizer annually for seven years (Larsen & Widdowson, 1971). Uptake of fluoride by various plant species growing on fluorspar mine wastes was not related to water-soluble soild fluoride (Cooke et al., 1976) neither were Hall & Cain (1972) able to relate water soluble fluoride but Eysinga (1974) did find a positive relationship.

Measurement of labile soil fluoride by resin absorption (Larsen & Widdowson, 1971) gave a mean value of 20 parts $10^6$ for agricultural soils and these workers found that the resin labile fluoride agreed closely with isotopically exchangeable fluoride ($^{18}F$) in the soils. They also found significantly higher values in arable soils treated regularly with superphosphate fertilizer and Manley et al. (1974) found increased resin labile fluoride residues in a pasture soil after phosphorus fertilizer top dressing treatment. In his study on fluoride uptake by crops Eysinga (1974) found that the resin labile fluoride value was negatively correlated with the percentage of calcium carbonate in his soils, calcareous soils being low in labile fluoride. He also found that it was related to the leaf fluoride level in freesia plants. For comparing active fluoride levels in soils this method would seem to merit further study.

Plants which accumulate fluoride in the roots (Cooke et al., 1976) or which have a selective mechanism that impedes uptake (Koster, 1972) complicate relationships between soil and foliar analyses. Concentrations of fluoride in the substrate and in foliage samples have been correlated under nutrient culture conditions, in greenhouse studies with soluble fluoride compounds e.g. NaF and with acid soils, but information about such relationships is rarer under field conditions.

The presence of fluorides in the soil will lead to uptake by crops in varying degrees for different species, clones or even varieties (Koster, 1972), but it has been generally accepted that plant toxicity (N.B. not toxicity to animal or human following the ingestion of plant material) arising from soil fluoride even on acid soils is unlikely to be a major problem.

## FLUORIDE IN PLANTS

### Type of Fluoride

Hall (1968) broadly grouped the fluorine-containing compounds in plants as:

i. simple, inorganic fluorides that are water and dilute acid extractable, and inorganic compounds that are perchloric acid diffusible, possibly inorganic fluorophosphates,

ii. alkali labile, short and long chain carbon-fluorine compounds soluble in organic solvents.

Plants may be grouped broadly into:

(a) toxic plants which contain fluorocitrate or fluoroacetate, such as Dichapetalum cymosum from South Africa, and other tropical species (Hall, 1972).

(b) fluoride accumulators e.g. Camellia spp, Carya cordiformis (Zimmerman & Hitchcock, 1956; McClenahen, 1976).

(c) non-accumulators which range from fluoride sensitive e.g. Fressia sp. to fluoride tolerant e.g Lycopersicon esculentum (Istas & Alaerts, 1974).

Part only of the fluoride in group (a) plants is converted into organic compounds (Hall 1972) whereas all the fluoride in group (b) and (c) plants examined by Zimmerman et al. (1957) was reported by them to be inorganic. We do not have sufficient information to be sure about the detailed distribution for plants of group (a) and perhaps group (b) and (c) plants as well but there may be a relationship between plant species, the source, the form, and the level of the fluoride compounds to which they are exposed. Lovelace et al. (1968) and Istas & Alaerts (1974) have reported fluoride conversion to, and accumulation as both fluoroacetate and fluorocitrate in otherwise healthy looking plants exposed to high levels of HF in the atmosphere; but Cooke et al. (1976) could not find any organic fluoride in grass or clover species grown on fluorspar waste with high fluoride, even though some of the plants accumulated very high fluoride levels. They concluded that the fluoride which was largely present in a non-ionisable (inactive) form, consisted of calcium, magnesium, aluminium and silicate complexes. Their finding that the fluoride was inorganic supports earlier, pioneer work in this field by Peters & Shorthouse (1964).

Uptake of Fluoride

Fluoride may be taken up through the roots or through the leaves of plants. When taken up via the roots it may accumulate in them, but when translocated from the roots it normally accumulates in the foliage, particularly in the apical regions. Fluoride absorbed by foliage through the stomata, and studies have generally been concerned with gaseous fluorides, also accumulates in the apical leaf tissue and is not as a rule translocated into the stems and roots (Brewer, 1966; Cormis, 1968a, 1968b; Compton, 1970; Treshow 1971; Eysinga, 1972, 1974). Jacobson (1966) stated that fluoride always moved in the direction of the transpiration stream and did not move into non-transpiring areas.

The fluoride content of plant stems and fruit is generally low whereas the leaf content is high, when taken up via the roots (Eysinga, 1972). Comparison of leaf and seed levels for grasses, clover and broad-leaved plants by Cooke et al. (1976) show that in some cases seed levels are higher and in some lower than leaf levels. Data reported for maize (Zea mays) (Hitchcock et al., 1964; Kudzin & Pashova, 1970) wine grapes (Brewer et al., 1957) and navel orange (Brewer et al., 1959, 1967) show that seed or fruits often do have the lowest fluoride concentration in the above-ground portions of the plant or tree. Koster (1972) states that there is no relationship between fluoride in the roots and its concentration in the leaves, but that there is a relationship between the quantity of fluoride in the leaf and visible injury (for a particular plant species or cultivar).

## Fluoride Biochemistry

Treshow (1971) reviewed this subject extensively but there is not a great deal of very detailed information available.

In his categorization of fluoride effects on plants McCune (1969) included changes in physiological processes, metabolic activities and cellular structures in the absence of measurable changes in growth or yield. Treshow (1971) added an extra category covering cytogenetic effects.

Treshow (1971) presumed that fluoride which accumulated in leaf tips and margins passes into the mesophyll cells and Istas & Alaerts (1974) refer to gaseous fluoride entering the leaf via stomata, being absorbed by the mesophyll cells from the intercellular spaces. Ledbetter et al. (1959) reported that the concentration of fluoride in tomato plants derived from HF decreased in tissues from cell walls through chloroplasts, soluble proteins and mitochondria to mitosomes. Chang & Thompson (1966a) found the greatest concentration of fluoride, possible 60 parts $10^2$, in the chloroplasts of navel orange leaves.

It is widely reported that increased fluoride concentrations disturb photosynthesis, carbohydrate metabolism and water relations causing more intensive respiration, growth retardation, chlorosis, wilting, plasmolysis and collapse of cells followed by necrosis. Chloroplast breakdown occurs and synthesis of pigments by them is inhibited so that amounts of chlorophyll, leaf carotenoids and other soluble magnesium constituents decrease whilst the amounts of free amino acids increase (Treshow, 1971; Koster, 1972; Istas & Alaerts, 1974).

Enzyme activity is inhibited, notably ATP which is concerned with carbohydrate breakdown, enolase, phosphorylases and cholinesterase. This effect is probably attributable to the formation of fluormetal complexes. Interference with carbohydrate metabolism results in retarded growth and increased respiratory rate as has been shown by different workers for Kalanchoe spp, maize, Brassica oleracae var acephala and Pisum sativum (Koster, 1972).

Fluoride effects have also been reported for rice under water culture conditions and exposed to HF (Yamazoe & Nakamura, 1960), for photosynthesis and respiration of three pine and six hardwood trees using detached needle or leaf specimens (McLaughlin & Barnes, 1975) and for the seedling growth of Pinus taeda and Acer rubrum (Davis & Barnes, 1973).

The cytogenetic effects were reported for germinating root growth of Zea mays (Chang & Thompson, 1966b), and for fumigated Lycopersicon esculentum and Phaseolus vulgaris plants when exposed to 2-10 g/$m^3$ HF, as discussed by Treshow (1971).

## PLANT SYMPTOMS AND DIAGNOSIS OF FLUORIDE STATUS

### Symptoms of Toxic Fluoride

The symptoms and effects of toxic fluoride levels are caused by accumulation in plants which vary widely in their sensitiveness to injury. Differences in susceptibility occur not only between species, but may be present between varieties e.g. Gladiolus cvs. or individual plants within a clone e.g. Gerbera sp. Susceptibility is regulated by the degree of translocation within the plant; where atmoshperic pollution is present non-susceptible plants may accumulate fluoride on the leaf surfaces from whence it can be washed off.

The common visual symptoms of toxicity are described as follows:

Monocotyledons - on susceptible crops chlorosis followed by necrosis starting from the leaf tips and moving towards the leaf base. On maize and Sorghum spp. which are not particularly sensitive, symptoms generally develop as a chlorosis without marginal necrosis.

Dicotyledons - chlorosis and necrosis of leaf margins. In some plant species necrosis is preceded by grey or light green, water-soaked areas on the leaf most easily seen by transmitted light, which may later desiccate and turn to a tan or red-brown colour e.g. Pisum sativum.

The fruits of some plant species are highly sensitive. The peac fruit develops symptoms known as red blotch of the sutu the skin splits in this region and develops a bright red-coloured halo on each of the cheeks. The apricot develops similar symptoms and necrosis can occur on plum, cherry, pear and apple fruits.

Flowers - flowers are seldom attacked outside, but fluoride in the vase water of cut flowers can cause necrosis of the sepals and petals.

Pine Trees - the young needles develop tip burn.

Reference to symptoms of specific crops are reported by Hurd-Karrer (1950) for Japanese buckwater leaves, by Zimmerman & Hitchcock (1956) for peach, sweetcorn and tomato leaves; by Benson (1959) for peach fruits and by Brewer (1966), Bovay (1969), Spierings (1969), Compton (1970), Treshow (1971), Eysinga (1972), Koster (1972) and Istas & Alaerts (1974) for a number of different crops. MacLean et al. (1973) were able to show that visual tomato symptoms are the same whether the fluoride is accumulated through the roots or through the leaves. Photographic illustrations of plant symptoms are reported by Brewer (1966) for apricot, grapefruit, gladiolus, roses and maize leaves; by Bovay (1969) for apricot leaves and fruits, pear fruit, Berberis vulgaris and Colchicum autumnale leaves; by Eysinga (1971, 1974) for freesia leaves, by Poole & Conover (1973) for Cordyline terminalis and for tea, which is a well-known fluoride accumulator, by Pethiyagoda & Krishnapillai (1971).

Other symptoms of fluoride toxicity of less primary value for diagnosis, but of significance in production, are growth retardation, wilting and in some species death (Koster 1972). Clearly it is necessary to be extremely careful in making a diagnosis on the basis of visual symptoms alone since these may be confused with other nutritional e.g. salt excess, or disease disorders for many crops. Previous reference has been made to other factors that can influence the uptake of fluoride by plants viz. soil pH and rainfall; but toxic effects may also be modified by temperature which increases the likelihood of damage as it rises (Treshow, 1971), but the season (Eysinga 1974), by the stage of plant development when they may be more severe in young stages of growth, e.g. the sensitive needle emergence period for pine trees with atmospheric pollution. The sensitivity of a crop to fluoride damage may also be influenced by its nutritional status.

Atmospheric fluoride damage on commercial apricots and wine grapes was severe when mixed fertilizer applications made in the Spring contained potassium, present as the chloride, but not when it was present as the sulphate. If no fertilizer was applied or if it was applied in the Autumn and not the Spring, no visible necrosis developed (Bovay et al., 1969). The beneficial effect of calcium in reducing fluoride uptake through the roots by maize ($NH_4F$

in nutrient culture) has been demonstrated by Gligny et al. (1973). Deficiencies of Mg, Ca or K in tomatoes (sand/nutrient culture) which were exposed to fluoride either as NaF in solution or HF gas to the leaves, produced different results. Fluoride accumulation in leaves was suppressed by Mg deficiency, enhanced by K deficiency and unaffected by a Ca deficiency. The necrotic symptoms on apical leaves were increased by deficiencies of Ca and K, but not Mg (MacLean et al. 1969).

Fluoride may also adversely affect seed germination and root growth. Morse (1935) reported laboratory, greenhouse and field tests with contaminated single and double superphosphate fertilizers demonstrating this for maize. This damage could be prevented by thoroughly mixing the fertilizer with the soil, correct placement in the soil not too close to or above the seed or by allowing it to react with the soil before sowing the seed. Specht & MacIntire (1961) also found that after allowing a reaction time fluoride retained in acid sandy soils low in iron and aluminium did not reduce the germination of Pasapalum notatum seed. Conversely, seeds that were soaked in soluble fluoride solutions (NaF) had their germination suppressed though at differing concentrations viz. pea and soyabean at 0.5, vetch and cabbage at 1.0, radish, barley, and cole at 1.5 maize, cauliflower, lucerne, mustard, oats, clover and kohlrabi at 3.0 parts $10^6$ of fluoride; carrot, poppy and tomato seed germination was only partly inhibited at the highest concentration (Navarra et al. 1966). The toxicity of this fluoride compound and penetration of the testa could account for this adverse effect since Cooke et al. (1976) found that seeds of Minuartia verna and Rumex obtusifolus harvested from plants containing substantial quantities of soil-derived fluoride, and themselves containing high natural levels of fluoride, i.e. 10,480 and 1,248 µg/Fg respectively, germinated well. Two-day-old germinated seedlings were analysed after removal of the seed coat and were found to contain only 31 and 37 µgF/g respectively; most of the fluoride was associated with the seed coat, presumably in a naturally inactive form.

Damage caused to flower crops by fluoride in irrigation water, in hydroponic cultures and in vase water for cut flowers, has been reviewed by Koster (1972). Irrigation water containing hydrofluosilicic acid ($H_2SiF_6$) providing up to 10 parts $10^6$ fluoride was without observable effect upon a wide range of flower species including *Azalea*, *Calceolaria*, *Cyclamen*, *Freesia* and *Saintpaulia*. A total of 6g of NaF applied in the irrigation water weekly to *gladiolus* cv. Snow Princess caused a reduction in fresh weight, an increase in leaf tip necrosis and a decrease in flower numbers whereas it had no apparent effect on the cv. Valeria.

Fluoride (as KF) in hydroponic solutions at concentrations of 1, 2 and 5 parts $10^6$ caused moderate to severe leaf edge necrosis to Freesia cv. Golden Yellow.

Fluoride in the vase water of cut flowers may cause damage to sensitive species at quite low concentrations. Thus from 1 to 8 parts $10^6$ fluoride as $H_2SiF_6$ or $Na_2SiF_6$ in vase water caused damage to a number of flowers including carnation, gerbera, gladiolus and tulip cultivars; damage occurred to the petals, sepals, and leaf tips usually as a colour change or necrotic spots on flower parts or as necrosis of leaf tips and leaf edges.

## Diagnosis and Detection of Fluoride Status

A firm diagnosis of fluoride damage cannot be confidently made by observing visual plant symptoms alone; supplementary information is necessary.

Soil or substrate analysis will not provide the necessary supplementary information for no universal relationships between soil (total, labile or water soluble) and plant fluoride levels have been established for plant species nor is it likely that such associations will be found. Soil analysis is a valuable tool for site comparison or survey purposes on a local basis and may be of greatest significance and value in studies of horticultural and particularly greenhouse problems. Eysinga (1974) found that both water soluble and resin labile fluoride values were correlated with foliar fluoride in freesias, taken up through the roots, in the Netherlands.

Chemical analysis of leaves or of the whole above ground plant parts is an essential part of fluoride toxicity diagnosis (Raay 1969; Compton, 1970) and when associated with observations on indicator plants, to the monitoring of fluoride contaimination or pollution. In recent years most of the studies reported using these techniques have been concerned with atmospheric pollution, but where the presence of fluoride in this form is either absent or of minor concern as in many developing agricultural areas remote from industrial sites and city complexes, they may be applied to soil and water contamination studies.

Differentiation between plant species and cultivars that are sensitive or insensitive (resistent) to higher than normal fluoride concentrations have been drawn up by many workers and there is only general agreement. Individual lists of relative fluoride susceptibility often show variation in the ranking of the species (Davison et al. 1974) because of the many environmental and physiological factors that can affect the fluoride status of a plant. Furthermore, it may be essential to differentiate quite carefully between species on the basis of uptake either via the roots or through the leaves. Brewer et al. (1959) found that navel orange trees supplied with fluoride in nutrient solution culture accumulated much less in the tops of the trees than was found after exposure to gaseous fluoride for a much shorter time and at a concentration of only 1 part $2.10^3$ as great.

Appendix I lists plants divided between sensitive and insensitive species/cultivars reported by various workers. Such information should assist workers to select alternative plants or to establish related species that will thrive best under local conditions, may be more easily available and will best suit their particular requirements.

Certain plant species and cultivars have been proposed as indicator plants of fluoride contamination/pollution. Some of these are listed in Appendix 2. Raay (1969) observed that sequential sampling and analysis of grasses provides a good indication of fluoride accumulation if the intervals between samplings are short, say 7-14 days, and the same plants are sampled on each occasion.

Davison et al. (1974) have proposed a laboratory test as a measure of susceptibility of plants to gaseous fluoride which consists of placing 10 mm. of the base of mature, non-senescent leaves in a range of fluoride solutions (0-100 μgF/ml as pure reagent grade NaF) in a growth cabinet for 24 hours, followed by 48 hours immersion in deionised water. Tissue damage is assessed over the 48 hour period after which leaves are washed, sectioned, dried and analysed. The percentage of leaf area that developed necrosis for a species or cultivar was positively related to the concentration of fluoride in the solution. The maximum concentration of fluoride in the test solution that produced leaf damage on a number of different species and cultivars is shown in Table 6. The results clearly illustrate differential sensitivity to fluoride under the test conditions.

TABLE 6

COMPARISON OF THE REACTIONS OF SPECIES AND CULTIVARS TO FLUORIDE CONCENTRATION IN THE FLUORIDE SUSCEPTIBILITY TEST (Davison et al. 1974)

| SPECIES | MINIMUM CONCENTRATION FOR PRONOUNCED LEAF DAMAGE (μgF/ml) |
|---|---|
| Gladiolus cv. Snow Princess | 1 |
| Crocus (unknown cv.) | <5 |
| Tulipa (unknown cv.) | <5 |
| Narcissus poeticus cv. | 5 |
| Muscari (unknown cv.) | 10 |
| Chionodoxa (unknown cv.) | 10 |
| Galanthus nivalis | 25 |
| Narcissus cv. King Alfred | 50 |
| Endymion non-scriptus | >100 |

## Critical Fluoride Levels in Plants

Although foliar analysis data may be used to diagnose fluoride-induced symptoms or to monitor accumulation in plants, particularly monocotyledons, they do not indicate the duration, frequency or concentration of contamination/pollution under field conditions.

Use of the foliar analysis technique requires great care in the selection and preparation of standard samples; this is particularly important with fluoride which is toxic, accumulated and not mobile or recycled within the plant. Since fluoride is concentrated in the leaf tips and leaf margins (refer Table 7) the sample taken should be for a standard leaf or leaf portion, of known physiological age.

TABLE 7

EXAMPLES OF FLUORIDE IN DIFFERENT PARTS AND AGES OF MONOCOTYLEDON PLANT LEAVES - ($\mu$gF/g dm)

| SPECIES AND LEAF AGE | SAMPLE AND LEAF PART | | | |
|---|---|---|---|---|
| 1. Avena Sativa | 0-7.5 | 7.5-15 | 15-22.5 | 22.5-30 cm |
| Tip | 180 | 32 | 16 | 12 |
| Middle | 226 | 53 | 25 | - |
| Basal | 251 | 60 | 27 | - |
| 2. | 0-5 cm | Rest | | |
| Holcus lanatus | 174 | 103 | | |
| Desc[illegible]mpsia cespitosa | 106 | 79 | | |
| Dacty[illegible]s glomerata | 615 | 278 | | |

Note: 1 = Compton (1970); 2 = Cooke et al. (1976)

It is necessary to differentiate between the internal and external (surface) leaf fluoride particularly when the source of pollution is airborne, whether gaseous, particulate, suspended or dissolved in water. Surface deposits should be removed without injuring the leaf and without leaching internal fluoride away.

At Long Ashton Research Station for general analysis, we immerse leaves in 0.1 parts $10^2$ v/v Teepol solution for 15 seconds with gentle wiping of the sample (Bould et al. 1974); Compton (1970) lists cleaning treatments with detergent solution alone, detergent plus EDTA, acid-detergent or Alconox-EDTA. More recently Cooke et al. (1976) compared several washing procedures and found that (i) washing with deionised water alone or with 0.05 to 0.1 parts $10^2$ v/v Teepol was unsatisfactory and did not remove surface particulates completely (ii) a 0.3 M hydrochloric acid wash caused leaching of internal leaf fluoride and (iii) satisfactory methods were (a) 0.2 parts $10^2$ v/v Teepol or (b) 0.1 v/v Teepol plus 0.1 w/v EDTA, followed by four rinses in deionised water. Samples were brushed and agitated for one minute in these solutions.

When sampling by comparative analysis to determine the change in supply and the degree of accumulation i.e. an index of supply and an index of accumulation, it is necessary to sample leaves sequentially in time, in terms of (i) comparable physiological leaf age and (ii) comparable actual leaf age. If the plant continues to produce leaves during some or all of the sampling period this could be resolved by taking samples of (say) the first fully mature leaf at each sampling time to give the index of supply together with the first, third and sixth fully mature leaf when the first samples are taken followed at later sampling times, by the same actual aged leaves when they may by that time be in the second, fourth and seventh positions, etc. Very little critical work seems to have been published on this sort of sampling for fluoride toxicity evaluation.

Leaf samples should be taken from the apical parts of monocotyledonous plants and whole leaves may be taken for dicotyledons, selecting the youngest fully mature leaf as the starting point. The highest fluoride levels in Avena sativa were found in the apical 0-7.5 cm; for grasses, in the apical 0-5 cm (refer Table 7). Eysinga (1971) working with freesias sampled the leaf tips when relating leaf fluoride with fertilizer treatment but later (Eysinga 1974) he compared the apical 15 cm with the whole (above-ground) plant and obtained results favouring the latter sample.

Brewer (1966) lists the fluoride content of plant parts with special reference to toxicity; for a total of 158 sets of data covering 52 plant species, only 23 sets of data on 15 species concern crops grown on soils without possible atmospheric contamination nearby. Other and in many cases more recent data about critical levels, threshold values and fluoride concentrations associated with toxic symptoms are summarized in Appendix 3.

Although the values for normal, healthy crops are generally comparable the values for threshold or 'above normal' levels at which no visible symptoms have been detected, are extremely variable. This may, of course, reflect differences between plant species and/or the influence of treatment conditions, fluoride source, environmental differences or interactions among the investigations reported. Whatever the reasons it is difficult to establish menaingful critical levels with any real degree of precision though it is possible with present knowledge to differentiate between normal and abnormal (high) levels.

## THE EFFECTS OF HIGH FLUORIDE LEVELS ON CROP PRODUCTION

Whilst qualitative information about fluoride toxicity effects on crops is available there is relatively little quantitative information published especially under field-problem situations.

Heavy atmospheric pollution has caused the total death of plant and tree species but the condition is generally observed over a relatively small area. It seems likely that a more serious effect may be present at the sub-lethal level causing reduced crop productivity. It is generally agreed that exposure to gaseous fluoride is more damaging than either particulate matter in the atmosphere or to fluoride in the soil (Hanson et al., 1958; Compton, 1970; Treshow, 1971).

Leaf chlorosis/necrosis will reduce photosynthetic efficiency; reduced plant growth, leaf size, "physiological" wilt and root damage may all be expected to contribute to lower end-point yields.

Thus, Robak (1969) reported the death of <u>Pinus sylves-</u>

*tris* through air pollution, Rhoades & Brennan (1975) reported defoliation but not death, of *Pseudotsuga menziesii* and Prunus persica; Brewer et al. (1957) reported yield reductions of 15 and 22 parts $10^2$ in bearing Washington navel oranges after premature leaf drop and reduced leaf size, but no reduction in fruit quality. The trees were treated for 6 years with sprays of dilute NaF solutions (.00125 and .0025N) receiving 15 sprays in all and the yield reduction was observed from year 3.

Koster (1972) summarized data on the reduction of flower yields and flower damage in several susceptible species, attributable to fluoride in the soil or substrate and in the irrigation water. Koster (1972), Bruyn & Hulsman (1972) also noted that the quality of susceptible species of vase flowers, and hence the economic value, was reduced due to damage from fluoride in the vase water.

When soil, water or the atmosphere contain fluoride at levels thought to be harmful to plants the situation should be clearly defined and characterised in the initial phase, after which a critical evaluation of the effects on plant yields, crop quality and economics should be undertaken.

## THE CONTROL OF EXCESS FLUORIDE

The adverse effects of plant exposure to high fluoride pollution/contamination may be overcome by growing resistant species and tolerant varieties provided the end product does not itself contain undesirably high fluoride levels e.g. animal forages or food crops for human consumption. A more satisfactory solution is the prevention or removal of fluoride pollution within the plant environment by control or elimination of the source.

### In Soils

A process that accelerates the removal of active fluoride from the soil to another part of the environment e.g. the ground water, is not necessarily an acceptable solution to soil decontamination.

Under irrigated, dry land farming conditions it is possible to avoid concentration of soluble fluoride in the surface soil which may have occurred through upward water movement, by applying heavy irrigation treatments to leach it out of the profile which may be considered as a natural chromatographic column (Graham-Bryce, 1973). Moving soluble fluoride in this manner may also contribute to its conversion to insoluble forms or to biological inactivation within the soil profile.

Liming acid soils or where liming is not desirable the application of gypsum ($CaSO_4$), both in finely ground form, will render soluble fluoride insoluble. Larsen & Widdowson (1971) found that resin labile soil fluoride was least soluble at about pH 6.0; Brewer (1966) recommends raising the pH of acid soils to pH 6.5. Hurd-Karrer (1950) raised the pH of a sandy loam from 4.9 to 7.5 and demonstrated substantial reductions in fluoride uptake by collard and Japanese buckwheat. Working with light sand and loam soils Prince et al (1949) found reduced accumulation of fluoride by Japanese buckwheat and tomato plants as they raised the pH with lime. Tomato leaves were found to contain 23 or 1,008 at pH 4.5; 15 or 480 at pH 5.5 and 7 or 91 parts $10^6$ fluoride at pH 6.5 in the absence or presence of 360 parts $10^6$ fluoride as NaF to the soil, as the pH value was raised by liming. The value of high calcium levels in reducing the uptake and accumulation of fluoride by plants is discussed by Eysinga (1972) and Koster (1972). Poole and Conover (1973) working with rooting media like peat and perlite, found that liming with $Ca(OH)_2$ and dolomite decreased the uptake of fluoride by Cordyline terminalis from added superphosphate which contained 1.6 parts $10^2$ fluoride. Eysinga (1974) found that lilies and freesias grown on peat were protected against fluoride damage at high pH values of 6.5-7.0, over the tested range of $<4.0$ to 7.0.

The regular application of low fluoride phosphatic or compound fertilizers and the avoidance of soil amendment materials containing high levels of fluoride, are ways to prevent the build-up of fluoride in soil or other substrate. This may be of particular importance in the greenhouse and in horticulture (Eysinga, 1974).

Phosphorus in the soil added as fluoride-free fertilizer reduces uptake of fluoride by Japanese buckwheat, tomato (Prince et al., 1949) and freesia (Eysinga, 1974) compared with P equivalent dressings of a contaminated commercial product.

Bovay et al. (1969) found that the application of boron containing compound fertilizer caused undesirable toxicity in apricot and grape vines, indicating that care is necessary at all times. In this instance the cause was attributed to potassium fluoborate in the fertilizer.

In Plants

The safest way to control fluoride excess in plants is to avoid exposing them to soil, water or atmosphere with abnormal fluoride content. This is not always practicable or possible.

Entry of gaseous and soluble fluorides into the leaf may be reduced by spraying or dusting calcium onto the foliage (Eysinga, 1972). Benson (1959) found that soft suture and splitting of peach due to toxic fluoride was prevented by $CaCl_2$ or $Ca(OH)_2$ sprays; the rates were 100 g.1 later reduced to 50 g.1. In addition to fixing fluoride outside the leaf and so preventing entry, there appeared to be an increase in internal leaf tolerance. Foliar sprays of lime were applied to Satsuma mandarins by Watanabe & Amano (1973) to counteract toxic fluoride in the air. The sprays reduced leaf fall, increased the percentage of fruit set, reduced titratable acidity in the fruits and increased the chlorophyll content of the leaves. Other salts ($MnSO_4$, $ZnSO_4$, $MgCa(NO_3)4$) and water did not produce these effects.

Growth suppression in Valencia orange by gaseous fluoride (HF) was reduced more by drenching showers (simulated) than by light daily sprinkling, though with young leaf growth both water treatments were equally effective (Brewer et al., 1969). Fluoride pollution of pasture from the atmosphere was reduced by rainfall by both washing and leaching from the leaf (Davison et al., 1976). However, in both these situations fluoride was only moved from a more critical (atmosphere) to a less critical (soil and ground vegetation) part of the environment. Eysinga (1972) also refers to the effect of sprinkler irrigation in washing off and leaching out fluoride from plants.

## In Fertilizers

Control of fluoride contaminant in fertilizers rests with the raw materials used (see Table 4 and Swaine, 1962) and the manufacturing processes. Lehr & McClellan (1972) discuss the raw materials used by the phosphate industry which are very variable in composition and represent one of the largest single sources of fluoride contamination. After beneficiation of raw rock phosphate fluoride recovery from it by defluorination varies considerably; this is because the wide compositional range presents problems for the efficient and effective removal and recovery at any one stage and by a single method.

## In Air

Rainfall and overhead irrigation will 'scrub' gaseous and particulate fluorides out of the air to an extent depending upon frequency, intensity and duration relative to emission by the pollution source.

Bernatzky (1969) describes the use of protective tree plantations or windbreaks of resistant, insensitive tree species which need to be both well-sited apropos the prevailing wind(s), and dense. The maximum area over which they will be effective is given at 5 times the height on the windward side and 25 times the height on the leeward side.

The planting of amenity grass areas in and around city, industrial and suburban sites to absorb fluoride has been suggested as a way to fix atmospheric fluoride and remove it to a less dangerous part of the general environment.

Gaseous fluoride in the air used to aerate hydroponic and nutrient culture solutions should be removed or it may accumulate and become toxic to the roots and aerial parts of the plants, particularly if they are sensitive species.

## In Water

Water that contains calcium ions is unlikely to carry soluble fluoride when irrigating crops. Brennan et al. (1950) and Bligny et al. (1973) demonstrated for tomato and maize respectively that fluoride applied to the roots was much less toxic in nutrient culture with calcium ions. Fluoride toxicity decreased as calcium increased in these studies. It is possible to render fluoride dissolved in water insoluble by the addition of chemicals e.g. $CaCl_2$ as recommended by Walker (1955) for cattle drinking purposes, but it is impracticable for large volumes of irrigation water.

The addition of aluminium sulphate $(Al_2(SO_4)3).18H_2O$ to the water culture in which lettuce and freesia were being grown, and to which fluoride was added in the range 0-10 mgF/l, had no effect on damage to lettuce (fluoride insensitive species), but reduced both leaf necrosis and foliar fluoride in the very sensitive freesia (Eysinga, 1974).

Injury to the cut-flowers of susceptible species may be reduced by adding aluminium sulphate to the water. Injury to cut Gerbera sp. flowers in fluoridated vase water (≡ 1 part $10^6$ F) was materially reduced by adding aluminium sulphate (600-700 parts $10^6$) or a proprietary product based on Alum both of which precipitate soluble fluoride out of solution (Bruyn & Hulsman, 1972).

## FLUORIDE STANDARDS

There are no standards for soil, rooting media or fertilizers, but they should contain as little soluble fluoride as possible to avoid adverse plant effects.

### For Plants

Standards or threshold values at which damage occurs vary widely with plant species, but should be maintained at normal, healthy levels (see Apendix 3). Threshold values can vary from as little as zero for susceptible freesia cultivars to as much as 600 parts $10^6$ for spinach though the general range is between 0-40 parts $10^6$ fluoride in dry matter (Appendix 3).

For forage crops in New Zealand exposed to contamination from the atmosphere the following rigorous standards have been established to prevent damage to vegetation and livestock (Farrier & Pullen, 1973):

F content of forage must not exceed:

- 40 mg F/kg dry basis over 1 year
- 60 mg F/kg dry basis over any 2 consecutive months
- 80 mg F/kg dry bassis for more than 1 month

## For Water

For potable waters the general standard is that a fluoride content >1 part $10^6$ is unsuitable for drinking, and the usual range for artificial fluoridation of potable waters is quoted as 0.8-1.6 parts $10^6$. Standards for irrigation water are difficult to define because visible damage varies with so many factors i.e. type of irrigation (surface versus overhead versus trickle type), plant species and age, greenhouse or intensive versus outside, extensive cultural conditions, the form of fluoride in the water, etc. The safest standard would be not to irrigate with water that exceeds the general average for surface waters namely, 0.1 parts $10^6$, but there are many situations where this is exceeded without apparent ill effect. It is tentatively suggested that where water containing more than 5-10 parts $10^6$ fluoride is regularly applied to crops it will be desirable to monitor them for damage from time to time by plant and soil analysis, preferably by comparing commercial practice with a fluoride-free water treatment in the absence of atmospheric pollution. Establishing such conditions of comparison on an acceptable basis will not usually be simple or easy.

## For Air

When establishing critical air quality standards the criteria has been to determine atmospheric fluoride concentrations that injure the most sensitive plant species, but this does not cover fluoride concentrations that cause non-visible damage. The establishment of critical atmospheric levels of fluoride (gaseous or alternative forms) is difficult because of the many factors that can influence the effect on plants and the lack of knowledge in this area. Environmental temperature, rainfall or watering regime, moisture status of the plant, type of plant (variety or clone), stage of growth, nutritional status and the exposure time to concentration relationship can all influence dose-response relationships (Treshow, 1971).

In California, U.S.A., sensitive gladiolus cultivars are grown outdoors and the distal 15 cm of the leaf is sampled for analysis; it should not contain more than 35 parts $10^6$ of fluoride in dry matter (Bovay, 1969; Istas & Alaerta, 1974). Istas & Alaerts (1974) have noted that gladioli exposed to 0.1 parts $10^9$ gaseous fluoride for five weeks, may accumulate up to 150 parts $10^6$ fluoride in this type of leaf sample.

A number of atmospheric fluoride standards have been established and examples are summarized in Table 8.

There is a considerable variation among these figures emphasizing the difficult in defining a single, general, critical value.

The monitoring of forage contamination by fluoride from atmospheric or fertilizer sources is possible by analysing the urine of grazing animals. In the south island of New Zealand which have similar or lower than average natural fluoride levels in soils, waters and forages, normal values for urine fluoride are given as: bovine = 0.3-7.4, mean 2.8 and ovine = 0.4-5.0 (Mean 1.5) mg F/l (Manley et al., 1975). Stewart et al. (1974) report normal ovine values of <5 increasing to 11-15 mg F/l for animals grazing on pasture contaminated with fluoride from top dressed superphosphate fertilizer (application rates of 250-500 Kg/ha fertilizer containing 0.93 parts $10^2$ fluoride). Farrier & Pullen (1973) reported ovine urine levels of between 1.4-11.5 mg F/l for animals on pasture within the potential contamination area of an aluminium smelter, and considered that the higher values indicated a mild contamination level.

Standards of toxicological residues in food crops arising from pesticide use have changed dramatically over the past twenty years. The proposed world-wide standard was 1.0 in 1950, 0.1 in 1960 and 0.01 parts $10^6$ F in 1970. In 1969 the Canada Food and Drugs Directorate proposed a limited of 2 parts $10^6$ F in fruits. Since 1960 in the United Kingdom, the general standard for acidic phosphate food additives has been 30 parts $10^6$F; from 1974 this limit was developed more critically for specific compounds as, for example: $H_3PO_4$ and $NaH_2.H_2)$ - 10 parts $10^6$ F; Ca $(H_2PO_4)_2.2H_2O$ - 30 parts $10^6$F and $CaHPO_4.H_2O$ - 50 parts $10^6$F.


SUMMARY

Presentation has been made with emphasis on sources of fluoride pollution or contamination in crop production other than atmospheric which is the area in which there is currently most interest by developed, industrial countries.

The natural and artificial sources of fluoride have been summarized and chemical analysis methods for determining fluoride in soil, plant, phosphate rock and fertilizer, and in water are discussed briefly. Reference is made to the value of the fluoride - ion selective electrode for faster and less complicated analytical routines.

A survey of fluoride in soils and in plants with a brief summary of fluoride biochemistry is presented. This is followed by descriptions of the visible symptoms of toxicity and also of the less obvious effects; the use of visible symptoms together with plant/soil analysis to diagnose fluoride status is discussed.

More recent information about fluoride levels in plant species is summarized. Definition of threshold levels above which visible toxicity symptoms are noted is complicated by variations between plant species and even between cultivars within a species. Indicator plant families, species and cultivars are noted.

Very little appears to have been recorded about the quantitiative effects of sub-lethal fluoride toxicity on the production of commercial and amenity crops. Adverse effects are assumed and there is a need to define

TABLE 8

EXAMPLES OF ATMOSPHERIC FLUORIDE STANDARDS

| COUNTRY | MAXIMUM PERMISSIBLE ATMOSPHERIC FLUORIDE LEVELS ($\mu gF/m^3$)* | | REFERENCE |
|---|---|---|---|
| 1. U.S.S.R. (1955) | 10 | for permanent exposure | |
| (Inhibited Areas) | 30 | for short periods | |
| 2. U.S.A. (1967) | 5 | Pennsylvania; av. over 24 hours | |
| | 3.3 | New York, Industrial zones | quoted by Istas & Alaerts(1974) |
| | 0.8 | Residential and Rural Areas | |
| 3. Germany (1968) | 1.2 | Tentative to avoid plant damage | |
| 4. New Zealand | 4.5 | for any 12 consecutive h. | |
| | 3.5 | for any 24 consecutive h. | |
| | 2.0 | for any 1 calendar week ) | Farrier & Pullen (1973) |
| | 1.0 | for any 1 calendar month ) | |

Note: * 1 $\mu g/m^3$ of fluoride is equal to 0.874 parts $10^9$ by weight or 1.33 parts 109 by volume of any gas containing one fluoride atom per molecule.

these for particular crops and situations. When excess fluoride is detected in the soil and other solid substrates, the measures that may be taken to detoxicate the soluble fluoride ion are described. Similar information is included about preventing the entry of soluble and gaseous fluoride into the plant through the foliage.

The establishment of reliable and critical, single standard values for toxic fluoride levels in soils, rooting media, and irrigation waters that are universally applicable under field conditions, seems unlikely; the establishment of such values for fertilizer materials seems more probable. Some of the recognized difficulties of achieving this type of definition for fluoride pollution in the air are discussed.

Very brief reference is made to safe toxicological residue limits from pesticides, in fruit produce and in additives for processed foodstuffs.

Vegetation acts as a sink for fluoride which accumulates in it. Absorbed fluoride may be phytotoxic to plants and can adversely affect animals through the grazing and fodder crops, and human beings through the food chain. It is perhaps fortunate that generally speaking, seeds and fruits are not major sites of accumulation.

For plants (and animals) it is much more satisfactory and effective to prevent fluoride toxicity situations developing than it is to have to attempt to cure them.

## REFERENCES


Anon, 1971. Fluoride, Rep. Am. Nat. Acad. Sciences, Comm. on Biological Effects of Air Pollution, Washington D.C., 295 pp.

Anon, 1974. Effects of Fluorides in Animals. Rep. Am. Nat. Acad. Sciences, 8-9.

Association of Official Analytical Chemists, 1970. Official Methods of Analysis. 11th Ed. A.O.A.C., Washington, D.C., sect. 25.029-25.035.

Association of Official Analytical Chemists, 1975. Changes in Methods, Fluorine. J. of the A.O.A.C., 58, 384-385.

Benson, N.R., 1959. Fluoride injury or soft suture and splitting of peaches. Proc. Amer. Soc. Hort. Sci., 74, 184-198.

Bernatzky, A., 1969. Shelterbelts for air purification. Air Pollution. Proc. 1st Eur. Congr. on the influence of air pollution on plants and animals. Wageningen, 1968. Pudoc, Wageningen, 383-395.

Bertrand, D. & Wolf, A. de., 1970. Soluble fluorides in French arable soils. C. r. hebd. Seanc. Acad. Agric. Fr., 56, 867-868.

Bligny, R., Garrec, J.P. & Fourey, A., 1973. Effect of calcium on the migration and accumulation of fluorine in Zea mays. C. r. hebd. Seanc. Acad. Sci., 276, 961-964.

Bolay, A., Bovay, E., Neury, C. & Zuber, R., 1969. Interactions between fertilizers and the toxicity of atmospheric fluoride compounds. Air Pollution. Proc. 1st Eur. Congr. on the influence of air pollution on plants and animals. Wageningen, 1968. Pudoc Wageningen, 143-160.

Bould, C. & Associates, 1974. Long Ashton Manual of Chemical Methods of Plant Analysis. Cyclos. 58 pp.

Bovay, E., 1969. Effects of sulphur dioxide and fluorine compounds on vegetation. Air Pollution. Proc. 1st Eur. Congr. on the influence of air pollution on plants and animals. Wageningen, 1968. Pudoc Wageningen, 111-135.

Bovay, E. & Associates, 1969. Influences of some compound boron fertilizers on the accumulation of fluorine in plants. Rev. Suisse Vitic. Arboric., 1, 30-35.

Bowen, H.J.M., 1966. Trace Elements in Biochemistry. Academic Press London and New York, 241 pp.

Bower, C.A. & Hatcher, J.T., 1967. Absorption of Fluoride by soils and minerals. Soil Sci., 103, 151-154.

Brennan, E.G., Leone, I.A. & Davies, R.H., 1950. Fluorine toxicity in tomato as modified by alteration in the nitrogen, calcium and phosphorus nutrition of the plant. Plant Physiol., 25, 736-747.

Brewer, R.F., 1965. Fluorine. In, "Methods of Soil Analysis", ed. Black, C.A., Am. Soc. Agron., Madison, Wisconsin. Agronomy Ser. No. 9, Vol. 2, 1135-1148.

Brewer, R.F., 1966. Fluorine. In "Diagnostic Criteria for Plants and Soils", ed. Chapman, H.D., Univ. Calif., 793 pp. Chapter 12, 180-196.

Brewer, R.F., McCulloch, R.C. & Sutherland, F.H., 1957. Fluoride accumulation in soliage and fruit of wine grapes growing in the vicinity of heavy industry. Proc. Amer. Soc. Hort. Sci., 70, 183-188.

Brewer, R.F., Chapman, H.D., Sutherland, F.H. & McCulloch, R.B., 1959. Effect of fluoride additions to substrate on navel orange trees grown in solution cultures. Soil Sci., 87, 183-188.

Brewer, R.F., Garber, M.J., Guillemet, F.B. & Sutherland, F.H., 1967. Effects of accumulated fluoride on yield and fruit quality of Washington Navel Oranges. Proc. Am. Soc. Hort. Sci., 91, 150-156.

Brewer, R.F., Sutherland, F.H. & Perez, R.O., 1969. The effects of simulated rain and dew on fluoride accumulation by citrus foliage. J. Amer. Hort. Sci., 94, 284-286.

Bruyn, J.W. De & Hulsman, A.N., 1972. Fluorine injury in cut flowers of gerbera. Bedrijfsontwikkeling, 3, 209-211.

Chang, C.W. & Thompson, C.R., 1966a. Site of fluoride accumulation in navel orange leaves. Pl. Physiol., 41, 211-213.

Chang, C.W. & Thompson, C.R., 1966b. Effect of fluoride on nucleic acids and growth of germinating corn seedling roots. Physiologia Pl., 19, 911-918.

Compton, O.C., 1970. Plant tissue monitoring for fluorides. Hort. Science, 5, 244-246.

Cooke, J.A., Johnson, M.S., Davison, A.W. & Bradshaw, A.D., 1976. Fluoride in plants coloniding fluorspar mine waste in the Peak District and Weardale. Environ. Pollut., 11, 9-23.

Cormis, L. de., 1968a. Uptake of fluorides in solution and the migration of fluorine in tomato plants. Annls. Physiol. veg. Paris, 10, 155-169.

Cormis, L. de., 1968b. Absorption and accumulation of atmospheric fluorine by the leaves of some herbaceous plants. Annls. Physiol. veg. Paris, 10, 251-262.

Davis, J.B. & Barnes, R.L., 1973. Effects of soil applied fluoride and lead on growth of loblolly pine and red maple. Environ. Pollut. 5, 35-44.

Davison, A.W., Marsland, A. & Betts, W.E., 1974. A proposed rapid test for susceptibility to gaseous fluorides. Environ. Pollut., 7, 269-282.

Davison, A.W., Blackemore, J. & Wright, D.A. 1976. A reexamination of published data on the fluoride content of pastures. Environ. Pollut., 10, 209-215.

Evans, L., Hoyle, R.D. & McCaskill, J.B., 1970. An accurate and rapid method of analysis for fluorine in phosphate rocks. New Zealand Jl. Sci., 13, 143-148.

Evans, L., Hoyle, R.D. & MaCaskill, J.B., 1971. Fluoride analysis of phosphatic fertilizers. New Sealand Jl. Sci., 14, 851-855.

Eysinga, J.P.N.L. Roorda van., 1971. Fluorine toxicity in freesia through the use of triple superphosphate: a provisional communication. Bedrijfsontwikkeling, ed. Tuinb. 2, 49-51.

Eysinga, J.P.N.L. Roorda van., 1972. The uptake of plants of fluorine from the soil; a literature review. Inst. Bodemvruchtbaarheid, Rapp. 3. 51 pp.

Eysinga, J.P.N.L. Roorda van., 1974. The uptake of fluoride by the root and its effect on various crops, particularly freesias. Agricultural Res. Repts., Wageningen, No. 821, 83 pp.

Farrier, P.M. & Pullen, D.R., 1973. Survey of fluoride levels in the area around the Bluff smelter. Proc. Poll. Res. Conf. June 1973. New Zealand D.S.I.R. Infor. Ser., No. 97, 341-356.

Gorski, M. & Stupnicka, H., 1961. The effect of superphosphate application on the fluorine content of the soil. Roczn. Glebozn., 10, 453-468.

Graham-Bryce, I.J., 1973. Solutions to problems of soil pollution by agricultural chemicals. In, "Pollution Engineering and Scientific Solutions", ed. Barrekette, E.S. Proc. Lst Int. Mtg. of Soc. Engineering, Tel Aviv, June 1972. Plenúm Press New York and London, 1973, 2, 133-147.

Haas, A.R.C. & Brusca, J.N., 1955. Fluoride toxicity in citrus. Calif. Agric., 9, 15-16.

Hall, R.J., 1968. Observation on the distribution and determination of fluorine compounds in biological materials, including soils. Analyst, 93, 461-468.

Hall, R.J., 1972. The distribution of organic fluorine in some toxic tropical plants. New Phytol., 71, 855-871.

Hall, R.J. & Cain, R.G., 1972. Organic fluorine in tropical soils. New Phytol., 71, 839-853.

Hansen, E.D., Wiebe, H.H. & Thorne, W., 1958. Air pollution with relation to agronomic crops VII. Fluoride uptake from soils. Agron. J., 50, 565-568.

Hesse, P.R., 1971. Fluorine. In "A Textbook of Soil Chemical Analysis". John Murray. 401-404.

Hitchcock, A.E., Weinstein, L.H. & McCune, D.C. et al., 1964. Effects of fluorine compounds on vegetation with special reference to maize. J. Air Pollut. Control Assoc., 14, 503-508.

Horntvedt, R. & Robak, H., 1975. Relative susceptibility of eleven conifer species to fluoride air pollution. Meddelser Norsk Institutt Skogforskning, 32, 185-206.

Hurd-Karrer, A.M., 1950. Comparative fluorine uptake by plants in limed and unlimed soil. Soil Sci., 70, 153-159.

Istas, J.R. & Alaerts, G., 1974. The effect of fluorine and its compounds on plants. Rev. de l'Agriculture, 27, 487-496.

Jacobson, J.S., 1966. Accumulation and translocation of fluoride in plants. Abstr. in Proc. Ann. Meet. Pl. Physiol., Univ. Md., XXXV.

Jacobson, J.S. & Heller, L.I., 1975. Collaborative study of a potentiometric method for the determination of fluoride in vegetation. J. of the A.O.A.C., 58, 1129-1134.

Jeffries, C.D., 1951. Occurrence of fluorine in limestones and dolomites. Soil Sci., 71, 287-289.

Köpf, H., Oelschläger, W. & Bleich, K.E., 1968. Fluorine contents of waters associated with soils and rocks. S. Pfl: Ernähr. Bodenk., 121, 133-141.

Koster, L.M., 1972. The effect of fluorine in the soil and in water on floricultural crops. Landbouwdocumentatie, 28, 436-439.

Kudzin, Yu. K. & Pashova, V.T., 1970. Fluorine content of soils and plants after prolonged application of fertilizers. Pochvovedenie, 2, 74-79. Soils and Ferts., 1970, 33 (3398).

Larsen, S. & Widdowson, A.E., 1971. Soil Fluorine. J. Soil Sci., 22, 210-221.

Leblanc, F., Comeau, G. & Rao, D.N., 1971. Fluoride symptoms in epiphytic lichens and mosses. Canadian J. Bot., 49, 1691-1698.

Ledbetter, M.C., Mavrodineanu, R. & Weiss, A.J., 1959. Distribution studies of radioactive fluorine 18 and stable fluorine 19 in tomato plants. Contribs. Boyce Thompson Inst., 20, 331-348.

Lehr, J.R. & McClellan, G.H., 1972. Fluorine content of commercial phosphate rocks. A.C.S. Symposium on "Fluorine Sources and Technology", Aug. 30th, New York, 11p.

Lehr, J.R. & McClellan, G.H., 1973. Phosphate rocks: important factors in their economic and technical evaluation. CENTO Symposium on the Mining and Beneficiation of Fertilizer Minerals, No. 99, 194-242.

Leone, I.A., Brennan, E.G., Daines, R.H. & Robbins, W.R., 1948. Some effects of fluorine on peach, tomato and buckwheat when absorbed through the roots. Soil Sci., 66, 259-266.

Lovelace, J., Miller, G.W. & Welkie, G.W., 1968. The accumulation of fluoroacetate and fluorocitrate in forage crops collected near a phosphate plant. Atmos. Environ., 2, 187-190.

MacIntire, W.H. & Associates. 1949. Effects of fluorine in Tennessee soils and crops. Ind. Eng. Chem., 41, 2466-2475.

MacIntire, W.H., Sterges, A.J. & Shaw, W.M., 1955. Fate and effects of hydrogen fluoride added to four Tennessee soils in a 4-year lysimeter study. J. Agr. Food Chem., 9, 777-782.

MacLean, D.C., Roark, O.F. & Folkerts, S., 1969. Influence of mineral nutrition on the sensitivity of tomato plants to hydrogen fluoride. Environ. Sci. Technol., 3, 1201-1204.

Manley, T.R., Stewart, D.J., White, D.A. & Harrison, D.L., 1975. Natural fluorine levels in the Bluff Area, New Zealand. 2. Concentrations in pasture, soil, water and urine of sheep and cattle. New Zealand J. of Sci., 18, 433-440.

McCune, D.C., 1969. On the establishment of air quality criteria with reference to the effects of atmospheric fluorine on vegetation. Am. Petroleum Inst. Air Quality Monogr. 69-103.

McLaughlin, S.B. & Barnes, R.L., 1975. Effects of fluorides on photosynthesis and respiration of some south-east American forest trees. Environ. Pollut., 8, 91-96.

McClellan, G.H. & Lehr, J.R., 1969. Crystal chemical investigation of natural apatites. American Mineralogist 54, 1374-1391.

McClenahen, J.R., 1976. Distribution of soil fluorides near an airborne fluoride source. J. Environ. Qual., 5, 472-475.

McClenahen, J.R. & Schulz, E.R., 1976. Total soil fluoride determination by a single distillation-ion selective electrode procedure. Soil Sci., 122, 267-270.

Morse, H.H., 1935. The toxic influence of fluorine in phosphatic fertilizers on the germination of corn. Soil Sci., 39, 177-195.

Navara, J., Holub, Z. & Bedatsova, L., 1966. Effects of fluorine on the germination of some species of seed. Biologia. Bratisl., 21, 89-98.

Oelschläger, W. 1968. Fluorine accumulation in soil due to flue dust and fertilizers. Z. Pflernähr. Bodenk., 121, 142-146.

Oelschläger, W., Wohlbier, W. and Menke, K.H., 1968. The fluorine contents of plant, animal and other matter in regions without and with fluorine emissions iii. Fluorine contents of plant, animal and other matter in regions with fluorine emissions. Landw. Forsch., 21, 82-87.

Official Methods of Analysis, 1970. 11th Ed. A.O.A.C., Washington, D.C., secs. 25.029-25.035.

Paliwal, K.V., Mehta, K.K., & Gandhi, A.P., 1969. Fluorine in irrigation waters pf Bhilwara District of Rajasthan, Indian J. Agric. Sci., 39, 1083-1087.

Peters, R., & Shorthouse, M., 1964. Fluoride metabolism in plants. Nature, 202, 21-22.

Pethiyagoda, U. & Krishnapillai, S., 1971. Studies on the mineral nutrition of tea 3-experimentally induced minor-nutrient deficiency symptoms. Tea Q., 42, 19-29.

Poole, R.T., & Conover, C.A., 1973. Fluoride induced necrosis of Cordyline terminalis kunth. "Baby Doll", as influenced by medium and pH. J. Am. Soc. Hort. Sci., 98, 447-448.

Prince, A.L., Bear, F.E., Brennan, E.G., Leone, I.A. & Davies, R.H., 1949. Fluorine: its toxicity to plants and its control in soils. Soil Sci., 67, 269-277.

Raay, A. van., 1969. The use of indicator plants to estimate air pollution by $SO_2$ and HF. Air Pollution. Proc. 1st Eur. Congr. on the influence of air pollution on plants and animals. Wageningen, 1968. Pudoc Wageningen, 319-328.

Rhoades, A.F., & Brennan, E., 1975. Fluoride damage to woody vegetation in New Jersey in 1974. Plant Disease Reporter, 59, 427-429.

Robak, H., 1969. Aluminium plants and conifers in Norway. Air Pollution. Proc. 1st European Cong. on the Influence of Air Pollution on Plants and Animals, Wageningen, 1968. Pudoc Wageningen, 27-31.

Rogers, J., 1973. The fluoride electrode for determination of fluorine in effluents. Proc. Poll. Res. Conf., June 1973. New Zealand D.S.I.R. Infor. Ser., No. 97, 355 - 340.

Robinson, W.O. & Edgington, G., 1946. Fluorine in soils. Soil Sci., 61, 341-353.

Sauchelli, V., 1963. Manual of Fertilizer Manufacture. Industry Publications Inc., New Jersey, 230.

Sauchelli, V., 1965. Phosphates in Agriculture. Rheinhold Publishing Corp., New York. 37-39.

Smallpage, K.E., 1977. (Pers. Comm).

Somani, L.L., 1974. Toxicity of fluorine in well waters of Udaipur District of Rajasthan. Indian J. Agric. Res., 8, 57-60.

Specht, R.C. & MacIntire, W.H., 1961. Fixation, leaching and uptake from additions of certain fluorides in representive sandy soils. Soil Sci., 92, 172-176.

Spierings, F.H.F.G., 1969. A special type of leaf injury caused by hydrogen fluoride fumigation of narcissus and nerine. Air Pollution. Proc. 1st Eur. Congr. on the influence of air pollution on plants and animals. Wageningen, 1968. Pudoc Wageningen, 87-89.

Stewart, D.J., Manley, T.R., White, D.A. & Harrison, D.L., 1974. Fluorine residues on pasture, in soil and in sheep urine, resulting from top dressing with superphosphate. New Zealand Jl. Exptl. Agric., 2, 129-133.

Suketa, Y., Shimokawa, S. & Yamamoto, T., 1975. Studies on environmental pollution by fluoride ii. Determination of fluoride in soil. Eiseikagaku, 21, 89-92. (Anal. Abs., 30, 296).

Swaine, D.J., 1962. The trace-element content of fertilizers. C.A.B. Tech. Commun. No. 52, Common. Bur. Soils, 306 pp. (Fluorine chapter, 102-131).

Treshow, M., 1971. Fluorides as air pollutants affecting plants. Ann. Rev. Phytopath., 9, 21-44.

Van Den Heede, M.A., Heyndrickx, A.M., Van Peteghem, C.H. & Van Zele, W.A., 1975. Determination of fluoride in vegetation; a comparative study of four sample preparation methods. J. of the A.O.A.C., 58, 1135-1137.

Verloo, M. & Cottenie, A., 1969. The determination of fluorine in soil extracts by means of the fluoride ion selective membrane electrode. Meded. Rijksfac. LandWet., Gent 34, 137-152.

Walker, G.W., 1955. Removal of excess fluorine from drinking water for stock. E. Afr. Agric. J., 20, 220.

Walker, G.W. & Milne, A.H., 1955. Fluorosis in cattle in the Northern Province of Tanganyika. E. Afr. Agric. J., 21, 2-5.

Watanabe, J. & Amano, S., 1973. Effects of foliar spray of mineral solutions on the leaf fall, recovery from foliar chlorosis and fruit set of contaminated Satsuma mandarins grown in the vicinity of an alumina-refining factory. Mem., Coll. Agric., Ehime Univ. 18, 143-155. (Soils and Ferts., 1974, 37, (3162).)

Webber, J., 1977. (Pers. Comm.)

Yamazoe, F. & Nakamura, T., 1960. Metabolic changes in the paddy plant exposed to hydrogen fluoride, J. Sci. Soil, Tokyo, 31, 29-32.

Yamazoe, F., 1970. Spectrophotometric determination of fluoride in fertilizers and plants. J. Sci. Soil Man., Japan, 41, 37-42.

Zimmerman, P.W. & Hitchcock, A.E., 1956. Susceptibility of plants to hydrofluoric acid and sulphur dioxide gases. Contr. Boyce-Thompson Inst., 18, 263-279.

Zimmerman, P.W., Hitchcock, A.E. & Gwirtsman, J., 1957. Flurine in food with special reference to tea. Contr., Boyce-Thompson Inst., 19, 49-53.

APPENDIX 1

PLANTS THAT ARE SENSITIVE OR INSENSITIVE/RESISTANT TO FLUORIDE UPTAKE A. FROM THE SUBSTRATE THROUGH THE ROOTS B. FROM THE ATMOSPHERE (OR WATER) THROUGH THE LEAVES

| PLANT REACTION | SPECIES (CULTIVAR) | REFERENCE |
|---|---|---|
| A. +SENSITIVE | | |
| 1. | Cordyline terminalis | Poole & Conover (1973) |
| 2. | Freesia cv. | Eysinga (1971, 1974) |
| 3. | Tigridia pavonia; Montbretia crocosmiflora- Ixia hydr. (very sensitive). Hippeastrum hybr.; Nerine bowdenii cv. Pink Triumph; Vallota speciosa; Sprekelia formossisima; Galtonia candicans; Lilium longiflorum; L. regale; L. henry; L. speciosum cv. Rubrum; L. Mid-century hybr.cv.Enchantment and Five King; Ornithogalum thyrsoides; Tulipa Hybr.cv. Preludium and Blue Parrot;Alstroemeria hybr.; Acidanthera bicolor; Bladiolus hybr.cvs. Snow Princess & White Friendship; Bletitta Striata. | Eysinga (1974) |
| A. +INSENSITIVE | | |

APPENDIX 1 (contd..2)

| PLANT REACTION | SPECIES (CULTIVAR) | REFERENCE |
|---|---|---|
| 1. | Maize (Zea mays); Asparagus(A. officinalis); Chive (Allium schoenoprasum); Lycopersicon esculentum; Capsicum annum; Solanum capsicastrum; Lactuca sativa; Cucumis sativus; Fahlia hybr.; Ranunculus hybr.; Sinningia hybr.; Anemone coronaria. | Eysinga (1974) |
| + This list does not include information for nutrient solution culture. | | |
| B. SENSITIVE | | |
| 1. | Solanum pseudo-capsicum; Gladiolus cv.: Tulips cv. (very sensitive). Maize; Sorghum sp. (Milo); Ixora sp.; Prunus sp. (Apricot, Prune); Vitis sp. (cvs. Carugnane, Muscat of Alexandria, Pedro Ximenes, Salvador); Hypericum sp; Lilium regale; Prunus sp. (Peach). | Zimmerman & Hitchcock (1956) |
| 2. | Crocus; Freesia; Gladiolus; Ixia; Tulipa cvs. | Spierings (1969) |
| 3a. Germany (Borsdorf) | Vitis vinifera; Carfinus betulus; Iris germanica; Dactylis glomerata; Arrhenatherum elatius (Gramineae and Polygonaceae are among the mpst sensitive families). | quoted by Bovay (1969) |

APPENDIX I (contd..3)

| PLANT REACTION | SPECIES (CULTIVAR) | REFERENCE |
|---|---|---|
| b. France (Bossavy) | Acer monspessulanum; Berberis vulgaris; Lonicera alpigena; Colchicum autumnale; Hypericum perforatum; H. maculatum; Silene inflata. (Most sensitive of 76 herbaceous species and shrubs). | quoted by Bovay (1969) |
| c. U.S.A. (Thomas) | Gladiolus; Pine; Apricot; Azalea; Tulip; Larch (Larix sp.); St. John's Wort (Hypericum sp.). (Particularly sensitive). | quoted by Bovay (1969) |
| d. U.S.A. (Hendriz and Hall) | Gladiolus cvs. Bo Peep, Green Light, Peter Pan, Stormy Weather (screened out of 110 cvs. as most sensitive). | quoted by Bovay (1969) |
| 4. | Pinus sylvestris; Abies alba; Pseudotsuga menziesii; Picea abies; P. engelmannii, P. amorica. | Robak (1969) |
| 5. | Gladiolus cvs.; Apricot; Maize | Eysinga (1972) |
| 6. | Freesia; Tulipa cvs.; Gladiolus cvs.; (leaf symptoms). Petunia hybr.; Cyclamen sp. (Flower symptoms). Maize; Arrhenatherum elatius; vitis vinifera; | Istas & Alaerts (1974) |

APPENDIX 1 (contd..4)

| PLANT REACTION | SPECIES (CULTIVAR) | REFERENCE |
|---|---|---|
| | Prunus persica; P. armeniaca; P. domestica (Prunus symptoms on leaf and fruit). Pinus strobus; P. contorta; P. sylvestris; P. mugho; p. ponderosa; Picea pungens; Larix occidentalis; Pseudotsuga taxifolia; Acer negundo; Castanea sativa. | Istas & Alaerts (1974) |
| 7. | Iris germanica; Maize; Gladiolus cv. Snow Princess; Crocus purpureus hybr.; Narcissus poeticus. | Davison et al. (1974) |
| 8. | Prunus persica; Pseudotsuga taxifolis. | Rhoades & Brennan (1975) |
| 9. | Pseudotsuga menziesii; Abies spp.; Picea spp.; Pinus sylvestris | Horntvedt & Robak (1975) |
| B. INSENSITIVE | | |
| 1. | Piqueria trinervia; Gossypium hirsutum; Nicotiana tabacum; Taxus spp.; Vicia spp.; Cichorium spp.; Taraxacum officinale; Solanum nigrum; S. melongena; Apium graveolens; Lycopersicon esculentum; Curcubita pepo; C. sativus; Amaranthus retroflexus; Medicago sativa; Melilotus spp.; Coleus blumei; Pelargonium; | Zimmerman & Hitchcock (1956) |

APPENDIX I (contd..5)

| PLANT REACTION | SPECIES (CULTIVAR) | REFERENCE |
|---|---|---|
| | Cephalanthus spp.;Galinsoqa parviflora; Potentilla spp.; Hibiscus rose-sinensis;Lactuca sativa;Lilium speciosum var. rubrum; Philadelphus spp.; Plantago spp.; Portulaca oleracea; Fragaria spp.; Weigela spp.; Bidens spp. | Zimmerman & Hitchcock (1956) |
| 2. | Sparaxis; Fritillaria; Chionodoxa;Scilla; Muscari; Galanthus. | Spierings (1969) |
| 3. | Larix decidula; L. leptolepis; Tsuga heterophylla; Abies nordmanniana; A. procera; A. concolor. | Robak (1969) |
| 4. | Tobacco; Cotton; Chrysanthemum; Strawberry. | Eysinga (1972) |
| 5. | Ligustrum vulgare; Triticum aestivum; Hordeum vulgare; Galanthus nivalis; Sambucus nigra. | Davison et al. (1974) |
| 6. | Rosa hybr.; Fragaria spp.;Lycopersicon esculentum; Curcubita pepo;Gossypium hirsutum; Nicotiana tabacum; Asparagus spp.; Triticum aestivum; Agropyron repens; Heracleum sphondylium; Pyrus communis; | Istas & Alaerts (1974) |

APPENDIX I (contd..6)

| PLANT REACTION | SPECIES (CULTIVAR) | REFERENCE |
|---|---|---|
| | Fraxinus velutina; ULmus americana; Tilia americana; T. cordata; Plantanus spp.; Salix spp. | Istas & Alaerta (1974) |
| 7. | Ilex opaca; Betula alba var. populifolia; Platanus acerifolia; Cornus florida; Magnolia spp.; Moru spp.; Malus pumila; Acer platanoides. | Rhoades & Brennan (1975) |
| 8. | Carya cordiformis; Fagopyrum sagittatum; Cornus florida; Carya tormentosa; Camellia spp. | McClenahen (1976) |

APPENDIX 2

SUGGESTED INDICATOR PLANTS FOR DETECTING OR MONITORING FLUORIDE POLLUTION/CONTAMINATION

| PLANT REACTION | SPECIES (CULTIVAR) | REFERENCE |
|---|---|---|
| 1. SENSITIVE (ap) | Gladiolus cvs.; Freesia sp.; Tulipa cvs.; | Raay (1969) |
| RESISTANT (ap) | Medicago sativa; Hordeum spp.; Trifolium spp. | |
| 2. SENSITIVE (a) | Chenopodium murale; Stellaria media; Amaranthus retroflexus; Poa annua; Chenopodium album; Sinapsis arvensis. | Benedict & Breen quoted by Bovay (1969) |
| 3. SENSITIVE (ap) | Families: Monocotyledons (unspecified); Polygonaceae; Chenopodiaceae; Caryophyllaceae; Berberidaciae; Rosaceae; Vitaceae. Species: Lily, Iris, Tulip, Narcissus and Gladiolus cvs.; Colchicum autumnale; Fritillaria imperialis; Muscari comosum; Convallaria majalis; Berberis vulgaris; Silene cucubalus; Orchis militaris; Millefolium perforatum; M. maculatum, Apricot, Wine Grape; Arrhenatherum sp.; Dactylis sp.; Festuca sp.; Anthoxanthum sp. | Bovay (1969) |
| RESISTANT (ap) | Families: Cruciferae and Compositae. | |

APPENDIX 2 (contd..2)

| PLANT REACTION | SPECIES (CULTIVAR) | REFERENCE |
|---|---|---|
| 4. SENSITIVE (ap) | Lichen and Moss-bearing bark discs: Parmelia sulcata; Orthotrichum obtrusifolium. | Leblanc et al, (1971) |
| 5. SENSITIVE (ap) | Tulipa cvs.; Pinus strobus; Begonia; Crocus spp.; Hyacinthus. | Istas & Alaerts (1974) |
| 6. SENSITIVE | Apricot (Chinese variety); Oregon grape; Hypericum spp.; Gladiolus cvs. Snow Princess and Pink Prospector (Hypersensitive species). | Treshow (1971) |
| 7. SENSITIVE (sp) | Cordyline terminalis. | Poole & Conover (1973) |
| 8. SENSITIVE (sp) | Freesia;Tigridia pavonia;Montbretia crocosmiflora; Ixia hybr. | Eysinga (1974) |
| RESISTANT (sp) | Lycopersicon esculentum; Capsicum annuum; Solanum capsicastrum;Lactuca sativa;Cucumis sativus;Maize; Allium Schoenoprasum. | |

Note: ap = indicator plants for atmospheric fluoride (leaf intake)
sp = indicator plants for substrate fluoride (root uptake)

APPENDIX 3

## LEAF ANALYSIS VALUES INDICATING FLUORIDE STATUS OF PLANTS

| PLANT SPECIES | GROWING CONDITIONS | FLUORIDE LEVELS (Parts $10^6$ dry matter) | | | | | REFERENCE |
|---|---|---|---|---|---|---|---|
| | | Normal Healthy | No Visible Symptoms | Limiting/ Threshold Value | Sl.-Mod. Visible Toxic Effects | Mod.-Sev. Visible Toxic Effects | |
| A. GENERAL PLANT DATA | | | | | | | |
| Sensitive spp. | Field or Greenhouse | - | 10-100 | - | - | - | Eysinga (1972) |
| Insensitive/ susceptible spp. | (s, ap) | - | 200-400 | - | - | - | Eysinga (1972) |
| Sensitive spp. (a) | General (ap) | 15 | 15- 30 | 30- 40 | 40- 60 | 60-110 | Istas & Alaerts (1974) |
| Sensitive spp. (b) | General (ap) | - | - | - | - | 50-220 | Istas & Alaerts (1974) |
| Insensitive/ Resistant spp. | General (ap) | - | 500+ | - | - | - | Istas & Alaerts (1974) |
| Most Graminae | Field (ap) | - | up to 500 | - | - | - | Istas & Alaerts (1974) |
| B. PARTICULAR PLANT DATA | | | | | | | |
| Orange (C.sinensis) | Field (sp) | <10 | - | - | - | - | Brewer et al. (1959) |
| " " | Nutrient Culture (sp) | - | - | - | 34- 59 | - | Brewer et al. (1959) |
| Lemon (C. Limon) | Sand Culture(s, ap) | - | - | >50-100 | - | 400 | Haas & Brusca (1955) |
| Peach (P. persica) | Sand/Nut. Culture (sp) | - | - | <6 | - | - | Leone et al. (1948) |
| Apricot (P. armeniaca) | Field (ap) | - | - | 15 | - | >100 | Bovay (1969) |

APPENDIX 3 (contd..2)

| PLANT SPECIES | GROWING CONDITIONS | FLUORIDE LEVELS (Parts $10^6$ dry matter) Normal Healthy | No Visible Symptoms | Limiting/ Threshold Value | Sl.-Mod. Visible Toxic Effects | Mod.-Sev. Visible Toxic Effects | REFERENCE |
|---|---|---|---|---|---|---|---|
| Apricot (P. armeniaca) | Field (ap) | - | 15- 50 | - | - | 200-600 | Bovay et al. (1969) |
| " | Field (sp) | 1- 6 | 15 | 15-105 | 32 to | 640 | quoted by Eysinga(1972) |
| Apple (M.pumila) | Field (ap) | - | - | 70 | - | >160 | Bovay (1969) |
| Hornbeam (Carpinus sp.) | Field (ap) | 10 | - | - | - | 2,585 | Oelschläger et al.(1968) |
| Maize (Zea Mays) | Field (ap) | - | - | - | 93- 99 | - | Hitchcock et al.(1964) |
| Grass spp. | Field (ap) | 6.8 | 15 | - | - | - | Oelschläger et al.(1968) |
| Vine (V.vinifera) | Field (ap) | - | - | 25 | - | >100 | Bovay (1969) |
| Tomato (L. esculentum) | Sand/Nut. Culture (sp) | - | - | <82 | - | - | Leone et al.(1948) |
| " | Field (sp) | 10- 14 | 123-291 | 420 | 207 to | 2,179 | quoted by Eysinga(1972) |
| Spinach (S. oleracae) | Field (sp) | - | - | 200-600 | - | - | quoted by Eysinga(1972) |
| Cordyline terminalis | Greenhouse (sp) | - | - | - | 21-28 | 106-270 | Poole & Conover (1973) |
| Gladiolus cvs. Emilia | Field (ap) | - | 93 | - | - | - | Bovay (1969) |
| " Friendship | Field (ap) | - | - | - | - | 70 | Bovay (1969) |
| " | Field (sp) | - | - | 7- 10 | - | 30- 50 | quoted by Eysinga(1972) |
| Freesia sp. | Greenhouse (sp) | - | - | 0 | - | - | Eysinga (1974) |
| Lilies (L. longiflorum) cv.Long White | Greenhouse (sp) | - | - | 7 | - | - | Eysinga (1974) |

Note: sp = uptake via the roots. ap = uptake via the leaves.

# FLUORIDE AND PLANTS:

## SUPPLEMENTARY REFERENCES (SELECTED)

by Dr. J.B.D. Robinson

### Analytical Method

Buck, M. and Reusmann, G., 1971. A new semi-automatic method for fluoride determination in plant and air samples. Fluoride Q. Rep., 4, 5-15.

Shaw, W.M., 1954. Colourimetric determination of fluorine in water and soil extracts. Anal. Chem., 26, 1212-1214.

Willard, H.H. and Winter, O.B., 1933. Volumetric method for determination of fluorine. Ind. Engng. Chem. Analyt. Edn., 5, 7-10.

### Biochemistry of Plant Fluoride

Chang, C.W., 1970. Effect of fluoride on ribosomes from corn roots. Changes with growth retardation. Physiol. Plant., 23, 536-543.

Chang, C.W. and Thompson, C.R., 1965. Subcellular distribution of fluoride in navel orange leaves. Int. J. Air Water Pollut., 9, 685-691.

Chang, C.W. and Thompson, C.R., 1968. Effect of fluoride on nucleotides and ribonucleic acid in germinating corn seedling roots. Plant Physiol., 43, 669-678.

Cormis, L. de, 1968. Absorption des fluorures en solution et migration de fluor chez la tomate. Ann. Physiol. Veg. 10, 155-169.

McCune, D.C., Weinstein, L.H., Jacobson, J.S. and Hitchcock, A.E., 1964. Some effects of atmospheric fluoride on plant metabolism. J. Air Pollut. Contr. Ass., 14, 465-468.

McCune, D.C., Weinstein, L.H. and Mancini, J.F., 1970. Effects of hydrogen fluoride on the acid-soluble nucleotide metabolism of plants. Contrib. Boyce Thompson Inst. Plant Res., 24, 213-225.

Mohamed, A.H., 1968. Cytogenetic effects of hydrogen fluoride treatment on tomato plants. J. Air Pollut. Contr. Ass., 18, 395-398.

Navara, J., Horvath, I., Holub, Z., 1970. Effect of fluorine on plants in various phases of autogenesis. Biologia, 25. 219-232.

Pilet, P.E., 1970. RNA - metabolism and fluoride action. Fluoride Q. Rep., 3, 153-159.

Poovaiah, B.W. and Wiebe, H.H., 1969. Tylosis formation in response to fluoride fumigation of leaves. Phytopathology, 59, 518-519.

Woltz, S.S., 1964. Translocation and metabolic effect of fluorides in gladiolus leaves. Proc. Fla. St. Hort. Soc., 77, 511-515.

Ye-O Cheng, J., Ming-Ho Yu, Miller, G.W. and Welkie, G.W. 1968. Fluoro-organic acids in soybean exposed to fluoride. Environ. Sci. Technol., 2, 367-370.

Yang, S.F., and Miller, G.W., 1963. Biochemical studies on the effect of fluoride on higher plants. I. Metabolism of carbohydrates, organic acids and amino acids. Biochem. J., 88, 505-509.

Plant Symptoms

Dorst, H.J.M. Van, 1973. Two new disorders in freesias. Neth. J. Pl. Path., 79, 130-137.

Darley, E.F., Nichols, C.W., and Middleton, J.T., 1966. Identification of air pollution damage to agricultural crops. Calif. Dept. Agri. The Bulletin, 55, 10-19.

Eaton, F.M., Olmstead, W.R. and Taylor, O.C., 1971. Salt injury to plants with special reference to cations versus anions and ion activities. Pl. Soil, 35, 533-547.

Hendrix, J.W., and Hall, H.R., 1958. The relationship of certain leaf characteristics and flower colour to atmospheric fluoride - sensitivity in gladiolus. Proc. Amer. Soc. Hort. Sci., 72, 503-510.

Hitchcock, A.E., Zimmerman, P.W. and Coe, R.R., 1963. The effects of fluorides on Milo maize (Sorghum bicolor). Contrib. Boyce Thompson Inst. Plant Res., 22, 175-206.

McCune, D.C., Hitchcock, A.E. and Weinstein, L.H., 1966. Effect of mineral nutrition on the growth and sensitivity of gladiolus to hydrogen fluoride. Contr. Boyce Thompson Inst. Pl. Res., 23, 295-299.

Soil Fluoride

Gemmel, G.D., 1946. Fluorine in New Zealand Soils. New Zealand Journ. Sci. Tech., 27B, 302-306.

MacIntire, W.H., 1952. Air versus soil as channels for fluorine contamination of vegetation in two Tennessee locales. U.S. Tech. Conf. on Air Pollution. Air Pollution Proc., chap. 4, 53-58.

MacIntire, W.H., Shaw, W.M., Robinson, B. and Sterges, A.J., 1948. Disparity in the leachability of fluorine from incorporations in phosphated and slagged soils. Soil Sci., 65, 321-339.

MacIntire, W.H., Shaw, W.M., and Robinson, B., 1955a. Behaviour of incorporations of potassium and calcium fluorides in a six-year lysimeter study. Jour. Agr. Food Chem., 3, 772-777.

MacIntire, W.H., Sterges, A.J. and Shaw, W.M., 1955b. Fate and effects of hydrofluoric acid added to four Tennessee soils in a four-year lysimeter study. Jour. Agr. Food Chem., 3, 777-782.

Mitchell, H.H. and Edman, M., 1945. Fluorine in soils, plants and animals. Soil Sci., 60, 81-90.

Oelschläger, W., 1971. Fluoride uptake in soil and its depletion. Fluoride Q. Rep., 4, 80-84.

Ross, C.W., Wiebe, H.W., Miller, G.W., and Hurst, R.L., 1968. Respiration pathway, flower colour, and leaf area of gladiolus as factors in the resistance to fluoride injury. Bot. Gaz., 129, 49-52.

Spierings, F.H.F.G., 1970. Injury to gladiolus by fluoridated water. Fluoride Q. Rep., 3, 66-71.

Thompson, C.R., Taylor, O.C., Thomas, M.D. and Ivie, J.O., 1967. Effects of air pollutants on apparent photosynthesis and water use by citrus trees. Environ. Sci. Tech., 1, 644-650.

Treshow, M., and Harner, F.M., 1968. Growth response of pinto bean and alfalfa to sub-lethal fluoride concentrations. Can. J. Bot., 46, 1207-1210.

Treshow, M., and Pack, M.R., 1970. Fluoride. Ch. 3. In: "Recognition of air pollution injury to vegetation: A pictorial atlas". Air Pollut. Control Assoc. Inf. Rep. No. 1.

Woltz, S.S., Waters, W.E. and Leonard, D.C. 1971. Effects of fluorides on metabolism and visible injury in cut flower crops and citrus. Fluoride Q. Rep., 4, 30-36.

## Plant Uptake and Fluoride Levels

Jacobson, J.S., Weinstein, L.H., McCune, D.C. and Hitchcock, A.E., 1966. The accumulation of fluorine by plants. J.Air Pollut. Contr. Ass., 16, 412-417.

MacIntire, W.H., Winterburg, S.H., Clements, L.B., Hardin, L.J., and Jones, L.S., 1951. Crop and soil reactions to application of hydrofluoric acid. Ind. Eng. Chem., 43, 1800-1803.

MacLean, D.C., Schneider, R.E. and Weinstein, L.H., 1969. Accumulation of fluoride by forage crops. Contrib. Boyce Thompson Inst., 24, 165-166.

Matuura, S., Kokuba, N., Wakimoto, S. and Koga, A., 1954a. Fluorine content in plants. Mem. Faculty Sci., Kyushu Univ., Ser. C-2, 1-35.

Matuura, S., Kokuba, N., Wakimoto, S. and Tokimasa, M., 1954b. The fluorine in tea. Mem. Faculty Sci., Kyushu Univ., Ser. C-2, 36-47.

Miller, V.L., Johnson, F., and Allmendinger, D.F., 1948. Fluorine analysis of Italian prune foliage affected by marginal scorch. Phytopathology, 38, 30-37.

Nikolic, S., 1956. The action of nickel, cobalt and fluorine in the nutrition and fertilization of plants. Centre Intern. Engrais. Chim., 5eme Assemblee Gen. (Belgrade), Rappt. 1, 195-218.

Pourchet - Campos, M.A., 1951. Content of fluorine in cultivated tea in Sao Paulo. Univ. Sao Paulo Anais Faculdade Farm. e Odontol, 9, 57-59.

Romney, E.M., Wood, R.A. and Weiland, P.A.T., 1969. Radioactive fluorine 18 in soil and plants. Soil Sci., 108, 419-423.

Ventakeswarlu, P., Armstrong, W.D., and Singer, L., 1965. Absorption of fluoride and chloride by barley roots. Plant Physiol., 40, 255-261.

Air Pollution and Standards

Adams, D.F., 1956. The effects of air pollution on plant life. A.M.A. Arch. Ind. Health, 14, 229-245.

Adams, D.F. Hendrix, J.W. and Applegate, H.G., 1957. Relationship among exposure periods, foliar burn and fluoride content of plants exposed to hydrogen fluoride. Jour. Agr. Food Chem., 5, 108-116.

Hill, A.C., 1969. Air quality standards for fluoride vegetation effects. J. Air Pollut. Control Assoc., 19, 331-336.

Leone, I.A., Brennan, E., and Daines, R.H., 1962. Daily fluctuations of phytotoxic air pollutants in three New Jersey communities as influenced by certain meteorological parameters. Plant Dis. Reptr., 46, 140-143.

MacLean, D.C., and Schneider, R.E., 1970. Fluoride phytotoxicity: its alteration by temperature. 2nd Int. Clean Air Congr., Washington, D.C.

Others

Quinche, J.P., Zuber, R., and Bovay, E., 1971. A method of detecting fluorborate in roots. Rev. Suisse Viticult. Arboricult., 3, 71-74.

Quinche, J.P., Bolay, A. and Zuber, R., 1971. Root absorption of fluorborate by the apricot. Rev. Suisse Viticult. Aboricult., 3, 62-70.

Nömmik, H., 1952. Fluorine in Swedish agricultural products, soil and drinking water. Acta polytech., Ser. a, 7, 1-121.

Garber, K., 1970. Fluoride in rainwater and vegetation. J. Int. Soc. Fluoride Res., 3, 22-26.

Allmendinger, D.F., Miller, V.L., and Johnson, F., 1950. The control of fluorine scorch of gladiolus with foliar dusts and sprays. Proc. Amer. Soc. Hort. Sci., 56, 427-432.

# ALLEVIATION OF FLUORINE TOXICITY IN ANIMALS - A REVIEW

by Dr. Ahmet Çakır,
Atatürk University,
Faculty of Agriculture,
Animal Sciences Dept.,
Erzurum,
Turkey.

(Abstract)

About the turn of the century, phosphates such as rock phosphates, acid or superphosphate and phosphatic limestone were being added to animal diets to provide supplemental quantities of phosphorus. However, these sources of phosphorus were soon recognized to cause fluorine toxicity in animals.

Rock phosphate was potentially the most plentiful and, of course, the cheapest source of phosphorus. Therefore methods were developed to reduce the fluorine content of this source to a harmless level. However, the removal of excess fluorine in rock phosphate is very expensive, because it requires considerable amounts of heat or energy and other plant expenses. In order to by-pass this expensive process, the phosphorus product would need to be fed to animals either with less processing or with chemical or non-chemical substances which could prevent or at least decrease the toxic effect of fluorine.

Fluorine toxicity may also occur among farm animals in association with their prolonged exposure to unusual quantities of fluorine, in the peculiar environment of certain geographic areas which naturally have high fluorine soils and water or which are contaminated by some industrial plants.

Certain organic compounds have been extensively studied for alleviation of fluorine toxicity in laboratory animals and ruminants since the early 1930's. Calcium and aluminium compounds were found especially effective in

eliminating the toxic effect of fluorine. However, the use of fluorine alleviators has been studied very recently in poultry.

The mode of action of alleviators has been related to reduction of fluorine absorption.

## INTRODUCTION

Although there are indications that F is beneficial in preventing dental caries (McClure, 1970); essential in fertility of mice (Messer *et al.*, 1972); an effective drug in metabolic bone diseases such as osteoporosis and Paget's disease (Bernstein *et al.*, 1963); the main point of concern in animal nutrition is not its beneficial effects, but its toxic effect. Because from a practical standpoint a fluorine deficiency is not possible in normal geed grain diets.

There is an enormous number of publications on fluoride toxicity. Excessive F ingestion can induce either acute or chronic toxicity. Acute F toxicity has not been studied as extensively as chronic fluorosis because it occurs very rarely. Chronic F toxicity is the type of fluoride poisoning most often observed in livestock. It is difficult to define the precise point at which fluoride ingestion becomes harmful to the animal. It can vary from case to case and may be influenced by the following factors: Amount of F ingested; duration of ingestion; fluctuation in fluoride intake time; solubility of fluoride ingested; species of animal involved; age at time of ingestion; general level of nutrition; stress factors; and individual biological response (N.R.C., 1974).

The National Research Council (1974) compiled recommended levels of dietary fluorine that can be ingested safely. The following values are the current N.R.C. guidelines.

Chronic fluoride toxicity occurs among farm animals in association with their prolonged exposure to unusual quantities of fluoride, in the peculiar environment of certain geographic areas and as the result of components of feed (raw rock phosphate, acidic limestone and fertilizer

| ANIMALS | p.p.m. F* |
|---|---|
| Beef or dairy heifers | 40 |
| Finishing cattle | 100 |
| Breeding ewes | 60 |
| Horses | 60 |
| Breeding sows | 150 |
| Finishing pigs | 150 |
| Broiler chicks | 300 |
| Layers | 400 |
| Turkeys | 400 |

*When the fluoride in the ration is from a defluorinated rock phosphate tolerances may be increased by 50%.

phosphates) otherwise well suited to the profitable production of animals.

Fluoride reduces feed intake and growth rate of chickens (Çakır, 1976), of cattle (Hobbs and Merriman, 1962), and of sheep (Hobbs et al.,1954). Several mechanisms have been proposed to account for the reduced growth rate. Phillips et al., (1935) demonstrated that the growth of chicks was inhibited by feeding 70 mg. of fluoride per kg. of body weight per day. The authors postulated that this level of fluoride caused growth inhibition by restriction of feed consumption. In addition, intraperitoneal injections of fluoride also restricted feed consumption. The restriction of feed intake by both methods indicated that the action of fluoride was systematic in nature and independent of any action in the digestive tracts. This was supported by the data of Shearer and Suttie (1967). These authors reported a decrease in feed consumption in rats receiving intravenously 2.4 or 6 mg fluorine per day.

There are some indications that fluorine is effective in reducing M.E. values of feeds (Gardiner et al., 1968) and in vitro digestibility of cellulose (Chamberlain and Burroughs, 1960). It may be related to some enzymes in glycolytic and citric acid cycle. However, the real mechanism of fluorine toxicity is not known.

As pointed out in previous paragraphs, fluorosis is a big problem in some geographic areas. Furthermore, high-fluorine content of some sheep, and potentially more plentiful phosphorus sources, might bring fluorosis with them when they are fed as phosphorus sources. Therefore, alleviation of fluorine toxicity might have an economic importance and must be known in detail.

## Alleviation of Fluorine Toxicity

The effect of other dietary components on the toxicity of fluorine has been studied largely with the rate. Especially the mitigating effect of calcium salts demonstrated in this species by several groups of workers.

Hauck et al. (1933a), fed rats a rachitogenic ration containing 0.22, 1.22, and 0.6% calcium with or without 0.15% NaF. They found that 0.15% NaF severely reduced body weight in the 0.22% calcium group, but addition of F to diets containing 1.22 and 0.6% Ca did not produce any further reduction in body weight. In another experiment, Hauck et al. (1933b), observed the same effect, "that toxicity of F increased with decrease in dietary calcium level". Sharpless (1936) described rat experiments in which diets containing 1,000 p.p.m. of NaF retarded growth, but if 2% $CaCo_3$ was added, growth was normal.

Peters et al., (1948) were among the pioneers of alleviation studies. They gave 1.0 ml. of a 5% solution NaF in water by stomach tube. Without treatment death occurred in 24 hours or less in 75 to 95% of animals, but the introduction of 3 ml. of a 4% solution of $CaCl_2$ in water, just prior to or within 5 minutes after administration of F by stomach tube, the mortality was reduced to less than 10%. The authors pointed out that 10 minutes delay in the administration of $CaCl_2$ to fluoride-poisoned rats reduced significantly the efficacy of treatment. $CaCO_3$ as 5 per cent of feeding chalk in the diet depressed the storage of fluoride compounds in rat skeletons. In the group of rats receiving 250 ppm of NaF and 5% chalk, the increase in F-content compared to the basal group was less 50.5%, than that of the group receiving 250 ppm of NaF alone (Boddie, 1957).

Calcium was also studied as an alleviator in ruminants. Suttie et al., (1957) studied $CaCO_3$ as an alleviator in cattle. These authors furnished 20, 30, 40 and 50 ppm fluorine and 50 ppm fluorine plus 200 gm. of $CaCO_3$ daily in a 5½ year experimental period. Inclusion of added calcium in the ration increased the tolerance of cows to fluorine.

Additional calcium requires an increased amount of phosphorus in rations. Since most of the natural phosphorus sources contain remarkable amounts of F, this also leads to extra high-dietary-fluorine levels. There is not such a problem in using aluminium compounds. Therefore, aluminium compounds are the most frequently used alleviators against fluorine toxicosis.

Kempt et al., (1937) reported several mottled teeth in rats fed 0.025% NaF, whereas those rats fed 0.025% NaF plus 0.396% $Al_2(SO_4)_3.18H_2O$ showed no motting. The authors thus concluded that the ingestion of aluminium sulphate, simultaneously with fluorides, prevented or at least markedly reduced the effect of fluorine on teeth.

Venkataramanan and Krishnaswamy (1949) carried out experiments with rats extending over 15 weeks. There were three groups, one received a normal diet, the second group received 1 mg. of fluorine and the third group received 1 mg. of fluorine and 200 mg. of aluminium sulphate. F levels of bones were 1,140, 3,810 and 3,210 ppm respectively for control, fluorine and fluorine plus aluminium sulphate groups. Using the same criteria, F level of bones, Becker et al., (1950) reported that aluminium sulphate, fed as 0.53 per cent of the diet, caused a 45 per cent decrease in the fluorine content of the bones. Aluminium chloride was stated to be equal value in alleviating chronic fluorosis. $Al_2O_3$ was not considered to be so effective as the sulphate or chloride.

The administration by stomach tube of certain amounts of F (as NaF), in the presence of $CaCl_2$, $Ca_2P_2O_7$, $MgCl_2$, $AlCl_3$ or $Al_2O_3$ has shown that (Table 1) not only the concentration, but also solubility are of importance when considering their effects on the storage of F in the skeleton of the rat (Weedle and Muhler, 1954). In their experiment, the inhibitory effects of aluminium (as $AlCl_3$) were evident at the 1.0 and 0.1% of cation concentration, but $Al_2O_3$ did not decrease the amount of fluorine stored (Table 1).

Griffith et al., (1955) stated that the addition of aluminium sulphate or aluminium chloride the ration decreased the amount of fluorine stored in the bones by 30 to 40 for cattle ingesting 0-50 ppm fluorine or sheep ingesting 0.-200 ppm added to their rations. In another study Griffith et al., (1956) reached the same conclusion, that the addition of aluminium sulphate or $AlCl_3$ to the rations of animals receiving F decreases the amount of F stored in the skeleton. Boddie (1960) concluded that the alleviators retarded, but did not prevent absorption of fluoride. He worked with two groups of cattle-grazing pastures contaminated with about 40 ppm F for three years. One of the groups received 2 oz. alleviator of equal parts $CaCO_3$ mixed with the concentrates. The group given alleviator seemed to be in a slightly better body condition, and to have a less severe dental lesion.

Hobbs and Merriman (1959), fed 28, 38, 48 and 58 ppm and 28, 38, 48, and 58 ppm fluorine plus aluminium su phate at the rate of 0.5% of the total ration. The expe iment lasted 8 years. Fifty-eight ppm F caused a slight decrease in feed consumption. All the other groups and the 58 ppm F plus 0.5% aluminium sulphate group had a normal feed consumption. In all lots, the addition of aluminium su hate decreased the amount of dental fluorosis. The addition of aluminium sulphate also decreased bone F storage by 31% to more than 40%.

Cakır et al., (1976a) evaluated Al compounds as F toxicity alleviators in starting broiler chicks and turkeys. Added F levels from NaF ranged from 0 to 1,000 ppm, where-

TABLE 1

PERCENTAGE OF THE TOTAL FLUORIDE INGESTED WHICH IS STORED IN THE FEMUR AND WHOLE CARCASS OF THE RAT WHEN DIFFERENT INORGANIC SALTS ARE ADMINISTRATED WITH THE FLUORINE SOLUTION*
(Weedle and Muhler, 1954. J. Nutr., 54:437-444)

| SALTS | Cation Con. % | Total F in femur mg. | Total F in whole carcass mg. | Total F stored in carcass in percentage of total ingested |
|---|---|---|---|---|
| $CaCl_2$ | 1.0 | 0.140 | 3.13 | 11 |
| | 0.1 | 0.220 | 5.92 | 21 |
| | 0.01 | 0.443 | 10.95 | 39 |
| $Ca_2P_2O_7$ | 0.1 | 0.509 | 13.91 | 50 |
| | 0.01 | 0.505 | 13.59 | 49 |
| $MgCl_2$ | 1.0 | 0.261 | 5.88 | 21 |
| | 0.1 | 0.329 | 7.77 | 28 |
| | 0.01 | 0.521 | 11.43 | 41 |
| $AlCl_3$ | 1.0 | 0.206 | 4.22 | 15 |
| | 0.1 | 0.422 | 8.49 | 30 |
| | 0.01 | 0.509 | 12.21 | 44 |
| $Al_2O_3$ | 0.1 | 0.510 | 13.22 | 47 |
| | 0.01 | 0.502 | 12.44 | 44 |
| None | - | 0.544 | 11.98 | 43 |

*A total of 24 mg. of fluorine was given to all animals by stomach tube.

as Al levels varied from 0 to 800 ppm. Al was fed either as $Al_2O_3$ or $Al_2(SO_4)_3.18H_2O$. When fed as the sukphate salt, 800 ppm of Al completely prevented the toxic effect of at least 1,000 ppm of fluorine. $Al_2O_3$ was not effective as an alleviator of fluorine toxicity (Tables 2,3 and 4). In another study Cak r et al., (1976b) evaluated some high-fluorine fertilizer phosphates such as diammonium phosphate (DAP), concentrated superphosphate (CSP) and partially defluorinated phosphate and monocalcium phosphate (MCP), as sources of supplemental phosphorus with large white turkeys in a long-term experiment, 20 weeks. Al was used as an alleviator of fluorine toxicity in these starting and gorwing-finishing turkeys. Al:F ratio was 1:1. CSP and DAP were found somewhat toxic, significantly depressing body weight and feed efficiency when fed to supply all supplemental phosphorus from day-old to 20 weeks. Only CSP and DAP significantly increased bone strength, bone ash and fluorine level of bone ash. Fluorine content of muscle was significantly increased by DAP. Al partially eliminated the toxic effect of DAP and CSP, but was not effective in preventing all symptoms of fluorine toxicity produced by the fertilizer phosphates (Table 5).

## The Mode of Action of Alleviators

The mode of action of alleviators is believed to be inhibition of fluorine absorption from gastrointestinal tract. Indirect evidence, such as fluoride deposition in carcass or in bones, better growth and feed consumption and better teeth quality supports this hypothesis. There is also some direct evidence.

The average absorption rate of fluorine was reduced from 52.8% to 36.2% with intact rats by adding $CaCl_2$ (Stookey et al., 1964a). Using in vitro studies with gastrointestinal segments from albino rats, Stookey et al., (1964b) attempted to elucidate factors involved in the mechanism of absorption, and reported decreased diffusion of fluoride through the intestinal wall with the addition of Ca and Mg to the lumen of the intestine.

Hobbs et al., (1954) furnished a 106 ppm fluorine diet to sheep and added either 0.1 or 0.5% aluminium sulphate or 0.1% aluminium chloride. Fluorine levels in feces

TABLE 2

EFFECT OF FLUORINE AND ALUMINIUM SULPHATE ON 4-WEEK BODY WEIGHT, FEED/GAIN RATIO AND FEED CONSUMPTION OF STARTING TURKEYS*

| $Al_2(SO_4)_3.18H_2O$ | FLUORINE LEVELS, p.p.m. | | | | | AVERAGE[2] |
|---|---|---|---|---|---|---|
| | 000 | 200 | 400 | 600 | 800 | |
| | Body weight, gm.[1] | | | | | |
| % | | | | | | |
| 0.0 | 864efg | 688efg | 676defg | 641bcde | 533a | 644AB |
| 0.2 | 671efg | 682efg | 649bcde | 600bc | 549ab | 630A |
| 0.4 | 662cdef | 740g | 666cdef | 698efg | 606bcd | 674BC |
| 0.6 | 723fg | 725fg | 661cdef | 651cde | 669cdef | 686C |
| Average[2] | 685BC | 709C | 663B | 647B | 589A | |
| | 0-4 week feed/gain[1] | | | | | |
| 0.0 | 1.61abc | 1.61abc | 1.63abc | 1.58ab | 1.74c | 1.63AB |
| 0.2 | 1.59ab | 1.60ab | 1.70bc | 1.67abc | 1.66abc | 1.65B |
| 0.4 | 1.59ab | 1.57ab | 1.60ab | 1.57ab | 1.61abc | 1.59A |
| 0.6 | 1.55a | 1.58ab | 1.64abc | 1.61abc | 1.57ab | 1.59A |
| Average[2] | 1.59A | 1.59A | 1.64AB | 1.61AB | 1.65B | |

TABLE 2 (contd..2)

| $Al_2(SO_4)_3.18H_2O$ | FLUORINE LEVELS p.p.m. | | | | | AVERAGE[2] |
|---|---|---|---|---|---|---|
| | 000 | 200 | 400 | 600 | 800 | |
| | 0-4 week feed consumption/bird, gm.[1] | | | | | |
| 0.0 | 997cde | 1014cde | 1006cde | 916abc | 823a | 951A |
| 0.2 | 975bcde | 992cde | 971bcde | 910abc | 812a | 932A |
| 0.4 | 958bcde | 1075e | 976bcde | 1006cde | 876ab | 978AB |
| 0.6 | 1037e | 1055de | 1001cde | 961bcde | 964bcde | 1003B |
| AVERAGE[2] | 992BC | 1034C | 989BC | 948B | 868A | |

CAKIR et al., (1976a)

* Five groups of 3 male and 3 female poults were randomly assigned to each treatment at day-old.

[1] Means for either body weight, feed/gain ratio or feed consumption in each column and row which are not followed by the same letter differ significantly ($P < 0.05$).

[2] Composite means for either fluorine or aluminium sulphate levels within each response parameter which are not followed by the same letter differ significantly($P < 0.05$).

TABLE 3

EFFECT OF FLUORINE AND ALUMINIUM SULPHATE ON 4-WEEK BODY WEIGHT, FEED/GAIN AND FEED CONSUMPTION OF STARTING BROILER CHICKS*

| $Al_2(SO_4)_3.18H_2O$ | FLUORINE LEVELS p.p.m. | | | | | AVERAGE[2] |
|---|---|---|---|---|---|---|
| | 000 | 200 | 400 | 600 | 800 | |
| % | Body weight, gm.[1] | | | | | |
| 0.0 | 592abc | 618abc | 614abc | 590abc | 558a | 594A |
| 0.2 | 581abc | 608abc | 573ab | 590abc | 568ab | 584A |
| 0.4 | 639c | 622bc | 614abc | 615abc | 615abc | 622B |
| 0.6 | 572ab | 594abc | 614abc | 601abc | 600abc | 596A |
| Average[2] | 596A | 610A | 604A | 599A | 585A | |
| | Feed/gain[1] | | | | | |
| 0.0 | 1.81bc | 1.81bc | 1.74abc | 1.75abc | 1.71ab | 1.76A |
| 0.2 | 1.76abc | 1.73ab | 1.70ab | 1.78abc | 1.80abc | 1.76A |
| 0.4 | 1.86a | 1.72ab | 1.75abc | 1.75abc | 1.73ab | 1.76A |
| 0.6 | 1.75abc | 1.76abc | 1.68a | 1.76abc | 1.76abc | 1.74A |
| Average[2] | 1.79A | 1.76A | 1.72A | 1.76A | 1.75A | |

TABLE 3 (contd..2)

| $Al_2(SO_4)_3 \cdot 18H_2O$ | FLUORINE LEVELS p.p.m. | | | | | AVERAGE[2] |
|---|---|---|---|---|---|---|
| | 000 | 200 | 400 | 600 | 800 | |
| | Feed consumption/bird, gm.[1] | | | | | |
| 0.0 | 988b | 1035b | 990b | 956ab | 865a | 967A |
| 0.2 | 940ab | 974ab | 933ab | 973ab | 926ab | 949A |
| 0.4 | 1009b | 976ab | 986b | 999b | 967ab | 988A |
| 0.6 | 928ab | 959ab | 958ab | 966ab | 973ab | 955A |
| AVERAGE[2] | 966A | 985A | 967A | 974A | 933A | |

CAKIR et al., (1976a)

* Five groups of 3 female and 3 male broiler chicks were randomly assigned to each treatment at day-old.

1 Means for either body weight, feed/gain ratio or feed consumption in each column and row which are not followed by the same letter differ significantly ($P < 0.05$).

2 Composite means for either fluorine or aluminium sulphate levels within each response parameter which are not followed by the same letter differ significantly ($P < 0.05$).

TABLE 4

EFFECT OF FLUORINE, ALUMINIUM SOURCE AND Al:F RATIO ON 4-WEEK BODY WEIGHT, FEED/GAIN AND FEED CONSUMPTION OF STARTING TURKEYS*

| Al SOURCE[1] | FLUORINE LEVELS p.p.m. | | | | | | | | AVERAGE[2] |
|---|---|---|---|---|---|---|---|---|---|
| | 400 | | 600 | | 800 | | 1000 | | |
| | $SO_4$ | $O_3$ | $SO_4$ | $O_3$ | $SO_4$ | $O_3$ | $SO_4$ | $O_3$ | |
| Al:F Ratio | Body weight, gm. | | | | | | | | |
| 0:1 | 689 | 696 | 685 | 658 | 625 | 634 | 456 | 534 | 622A |
| 0.4:1 | 682 | 717 | 688 | 683 | 676 | 638 | 664 | 485 | 654B |
| 0.6:1 | 684 | 688 | 707 | 698 | 735 | 643 | 671 | 503 | 666CB |
| 0.8:1 | 751 | 703 | 730 | 671 | 718 | 625 | 702 | 505 | 678C |
| Average[2] | 727D | | 701C | | 671B | | 604A | | |
| Average[2] | $SO_4$[1] - 679B | | $Al_2O_3$ - 630A | | | | | | |
| | Feed/gain | | | | | | | | |
| 0:1 | 1.56 | 1.55 | 1.56 | 1.58 | 1.63 | 1.57 | 1.85 | 1.72 | 1.63A |
| 0.4:1 | 1.55 | 1.50 | 1.56 | 1.53 | 1.58 | 1.58 | 1.56 | 1.77 | 1.58A |
| 0.6:1 | 1.55 | 1.53 | 1.56 | 1.53 | 1.52 | 1.60 | 1.57 | 1.77 | 1.58A |
| 0.8:1 | 1.54 | 1.50 | 1.52 | 1.60 | 1.51 | 1.58 | 1.56 | 1.72 | 1.57A |
| Average[2] | 1.53A | | 1.55A | | 1.57A | | 1.69B | | |
| Average[2] | $SO_4$[1] - 1.57A | | $Al_2O_3$ - 1.60B | | | | | | |

TABLE 4 (contd..2)

| Al SOURCE[1] | FLUORINE LEVELS p.p.m. | | | | | | | | AVERAGE[2] |
|---|---|---|---|---|---|---|---|---|---|
| | 400 | | 600 | | 800 | | 1000 | | |
| | $SO_4$ | $O_3$ | $SO_4$ | $O_3$ | $SO_4$ | $O_3$ | $SO_4$ | $O_3$ | |
| | Feed consumption/bird, gm. | | | | | | | | |
| 0:1 | 933 | 997 | 994 | 936 | 892 | 906 | 710 | 774 | 900A |
| 0.4:1 | 997 | 980 | 992 | 946 | 975 | 924 | 932 | 747 | 934B |
| 0.6:1 | 940 | 972 | 1020 | 984 | 1015 | 936 | 971 | 795 | 954B |
| 0.8:1 | 1058 | 976 | 1035 | 987 | 1004 | 888 | 1016 | 756 | 964B |
| Average[2] | 986C | | 987C | | 942B | | 838A | | |
| Average[2] | $SO_4^1$ - 970B | | $Al_2O_3$ - 906B | | | | | | |

CAKIR *et al.*, (1976a)

* Five groups of 3 females and 3 male poults were randomly assigned to each treatment at day-old.

[1] $Al_2(SO_4)_3.18H_2O$ and $Al_2O_3$.

[2] Composite means for either Al:F ratios, fluorine levels or aluminium sources within each response parameter which are not followed by the same letter differ significantly ($P < 0.05$).

TABLE 5

EFFECT OF FERTILIZER PHOSPHATE WITH AND WITHOUT $Al_2(SO_4).18H_2O$ ON BREAKING STRENGTH, BONE ASH AND PER CENT F IN BONE ASH AND MUSCLE TISSUE

| $Al_2(SO_4).18H_2O$ | P-SOURCES[1] | | | | | AVERAGE[4] |
|---|---|---|---|---|---|---|
| | CSP | DAP | PDP | MCP | MCP+NaF | |
| | Tibia breaking strength, kg.[3] | | | | | |
| Without | 85b | 89b | 46a | 47a | 46a | 63B |
| With[2] | 54a | 67ab | 46a | 47a | 43a | 51A |
| Average[4] | 69B | 78B | 46A | 47A | 45A | |
| | F content of bone ash, p.m.p.[3] | | | | | |
| Without | 10375d | 14625e | 900a | 772a | 1908a | 5715B |
| With[2] | 5541b | 7833c | 525a | 525a | 1825a | 3250A |
| Average[4] | 7958B | 11229C | 712A | 649A | 1865A | |
| | F content of muscle tissue, p.p.m.[3] | | | | | |
| Without | 1.65abc | 2.16c | 0.61a | 0.45a | 0.79a | 1.13A |
| With[2] | 2.06bc | 1.43abc | 0.48a | 0.38a | 0.60a | 0.99A |
| Average[4] | 1.86B | 1.80B | 0.54A | 0.41A | 0.70A | |

TABLE 5 (contd..2)

| $Al_2(SO_4).18H_2O$ | P-SOURCES[1] | | | | | AVERAGE[4] |
|---|---|---|---|---|---|---|
| | CSP | DAP | PDP | MCP | MCP+NaF | |
| | Bone Ash, %[3] | | | | | |
| Without | 58.3cde | 59.4e | 55.4a | 55.5a | 55.1a | 56.7A |
| With[2] | 58.2bcde | 58.5de | 55.9a | 56.6abcd | 56.9abcd | 57.2A |
| Average[4] | 58.2B | 59.0B | 55.6A | 56.1A | 56.0A | |

ĊAKIR <u>et al</u> (1976b)

[1] Dietary F levels from CSP and DAP decreased from about 670 to 280 ppm as the trial progressed; PDP furnished only about 1/10 as much F as CSP and DAP. NaF wasted with MCP to provide F levels equal to those from DPD.

[2] Al:F ratio was maintained at about 1:1 during the entire feeding trial.

[3] Means within each column and row which are not followed by the same letter differ significantly (P 0.05).

[4] Composite means for either phosphate sources or aluminium sulphate within each response parameter which are not followed by the same letter differ significantly (P 0.05).

were 7.6, 20.7, 27.5, and 33.3 mg./day, respectively, for 106 ppm dietary fluorine only, 106 ppm F plus 0.1% aluminium sulphate, 0.5% aluminium sulphate and 0.1% $AlCl_3$. The authors concluded that aluminium tied up ingested fluorine in the gastrointestinal tract with the $AlCl_3$ showing some advantage over the aluminium sulphate. Peters, (1971) fed 0.7, 24 and 35 ppm of F with 0, 50, 100, 200 and 400 ppm of Al as aluminium phosphate. Urine and feces were collected for 5-7 days prior to sacrifice. He stated that supplementation to diets with aluminium phosphate reduced the gastrointestinal absorption of fluorine from the diets. This was shown by the increased concentration of fluorine in the feces.

When the mode of action of Al against F toxicity in colostomized turkeys was studied, Al significantly reduced F absorption (Cakır, et al., 1976a). Urinary F levels were about 2.4 ppm in the control groups, 17.8 ppm in group receiving 1,000 ppm fluoride and 6.70 ppm in group receiving 1,000 ppm fluorine plus 800 ppm Al, respectively. Fluorine levels of feces were 33.6, 406 and 666 ppm for control, control plus 1,000 ppm F, and control plus 1,000 ppm F plus 800 ppm Al, respectively. (Table 6).

## DISCUSSION

Studies which were conducted on alleviation of fluorine toxicity are not enough in quantity. This makes quite difficult to reach an exact conclusion on alleviation. However, it can be said that several compounds such as Ca, Mg, and Al can be used as alleviators in farm animals. Calcium and Magnesium might bring some problems together. Therefore, the use of these two minerals may be avoided. Aluminium does not have such problems. It can be used in the form of $AlCl_3$, aluminium phosphate, aluminium sulphate and $Al_2O_3$ in ruminants. However, $Al_2O_3$ is not effective in pultry and rats.

TABLE 6

EFFECT OF ALUMINIUM ON FLUORIDE CONTENT OF FECES AND URINE IN COLOSTOMIZED TURKEYS, 12 WEEKS OF AGE.

| DIETARY TREATMENTS | | |
|---|---|---|
| Control[1] | Control + 1,000 ppm F[1] | Control + 1,000 ppm F + 800 ppm Al[2] |
| | Fluorine, ppm in fresh feces | |
| 24.7 | 387 | 468 |
| 21.7 | 425 | 713 |
| 43.7 | 354 | 714 |
| 37.6 | 450 | 706 |
| 40.3 | 427 | 731 |
| Average[3]: | | |
| 33.6a | 409b | 666c |
| | Fluorine, ppm in urine | |
| 2.02 | 5.92 | 7.48 |
| 3.28 | 6.92 | 4.11 |
| 1.66 | 26.9 | 6.35 |
| 2.84 | 25.7 | 9.34 |
| 2.22 | 23.9 | 6.23 |
| Average[3]: | | |
| 2.40a | 17.8b | 6.70a |

CAKIR *et al.*, (1976a)

[1] Each value is an average of five daily composite samples from three Turkeys.

[2] Each value is an average of five daily composite samples from four Turkeys.

[3] Composite means for each parameter which are not followed by the same letter differ significantly (P 0.05).

## REFERENCES


Becker, D.E.,J.M. Griffith, C.S. Hobbs and W.H. McIntire, 1950. The alleviation of fluorine toxicosis by means of certain aluminium compounds, J. Anim. Sci., 9:647.

Bernstein, D.C., C. Guri, P. Cohen, J.J. Collins and S. Tamvakopoulos, 1963. The use of sodium fluoride in metabolic bone disease. J. of Clin. Investigation, 42:916.

Boddie, C.E., 1957. Fluorine alleviators. II. Trials involving rats. Vet. Rec., 69:483-488.

Boddie, C.F., 1960. Fluorine alleviation. III. Field trials involving cattle. Vet. Rec., 72:441:445.

Cakır, A., T.W. Sullivan and F.B. Mather, 1976a. Aluminium and fluorine toxicity in chicks and poults. Nebraska Agric. Exp. Sta., Journal Series No. 5168.

Cakır, A., T.W. Sullivan and F.J. Struwe, 1976b. Al and fertilizer phosphates. Nebraska Agric. Exp. Sta., Jour. Series No. 5165.

Cakır, A., 1976. Alleviation of fluorine toxicity in starting chicks and turkeys with aluminium. University of Nebraska, Lincoln-Nebraska, U.S.A.

Chamberlain, C.C. and W. Burroughs, 1960. Effects of fluoride, magnesium and manganese upon cellulose digestion in vitro. J. Anim. Sci., 19:1253.

Gardiner, E.E., K.S. Winchell and R. Hironaka, 1968. The influence of dietary sodium fluoride on the utilization and metabolizable energy value of a poultry diet. Poultry Sci., 47:1241-1244.

Griffith, J.M., C.S. Hobbs, R.P. Moorman, R.D. Gregory, G.M. Merriman, W.A. McIntire and L.J. Hardin, 1955. Uptake, depletion and alleviation of fluorine in bones of cattle and sheep. J. Anim. Sci., 14:1204.

Griffith, J.M., C.S. Hobbs, G.M. Merriman and W.H. McIntire, 1956. Uptake and depletion of fluoride in bones of cows subjected to increased levels of fluorine for variable periods. J. Anim. Sci., 15:1261.

Hauck, H.M., H. Steenbock and H.T. Parson, 1933a. The effect of the level of calcium intake on the calcification of bones and teeth during fluorine toxicosis. Am. J. Physiol., 103:489-493.

Hauck, H.M., H. Steenbock and H.T. Parson, 1933b. Is the effect of fluorine in teeth produced through the parathyroid glands? Am. J. Physiol., 103:480-488.

Hobbs, C.S., R.P. Moorman, J.M. Griffith, J.L. West, G.M. Merriman, S.L. Hansard and C.C. Chamberlain, 1954. Fluorosis in cattle and sheep. Tenn. Univ. Agric. Sta. Bull. No. 235.

Hobbs, C.S. and G.M. Merriman, 1959. The effect of eight years continuous feeding of different levels of fluorine and alleviator on feed consumption, teeth bones, and production of cows. J. Anim. Sci., 18:1526.

Hobbs, C.S. and G.M. Merriman, 1962. Fluorosis in beef cattle. Tenn. Univ. Agric. Sta. Bull. No. 351.

Kempf, C.A., D.A. Greenwood and V.E. Nelson, 1937. Studies relating to the toxicity of fluorine compounds. J. Lab. Clin. Med., 22:1133-1137.

McClure, F.J., 1970. Water fluoridation. The Search and the Victory. U.S. Department of Health, Education and Welfare. National Institute of Health, Washington, D.C.

Messer, H.H., W.D. Armstrong and L. Singer, 1972. Fertility impairment in mice on a low fluorine intake. Science, 177:893-894.

National Research Council, 1974. Effects of fluorides in animals. National Academy of Sciences. Washington, D.C.

Peters, J.H., L. Greenman and T.S. Danowski, 1948. Beneficial effects of calcium chloride in fluoride poisoning. Fed. Proc., 7:93.

Peters, L.H., 1971. The effect of aluminium phosphate on the gastrointestinal absorption of fluoride from diets containing fish protein concentrate or casein. Desertation Abstracts International. Series B, 31 (7) : 3844-3845.

Phillips, P.H., H. English, E.B. Hart, 1935. The augmentation of the toxicity of fluorosis in the chick by feeding deccicated thyroid. J. Nutr., 10:399-407.

Sharpless, G.R., 1936. Limitation of fluorine toxicosis in the rat with aluminium chloride. Proc. Soc. Exp. Biol. Med., 34:562-564.

Shearer, T.R. and J.W. Suttie, 1967. Effects of fluorine administration of plasma fluoride and food intake in the rat. Am. J. Physiol. 212:1165-1168.

Stookey, G.K., D.B. Crane and J.C. Muhler, 1964a. Further studies on fluoride absorption. Proc. Soc. Exp. Biol. Med., 115:295-298.

Stookey, G.K., E.L. Dellinger and J.C. Muhler, 1964b. In vitro studies concerning fluoride absorption. Proc. Soc. Biol. Med., 115:299-301.

Suttie, J.W., R.F. Miller and P.H. Phillips, 1957. Studies of the effect of dietary NaF on dairy cows. 1. The physiological effects and the developmental symptoms of fluorosis. J. Nutr., 63:211-224.

Venkataraman, K. and N. Krishnaswamy, 1949. The amelioration of symptoms of fluorosis by aluminium salts. Indian J. Med. Res., 37:277-282.

Weedle, D.A. and J.C. Muhler, 1954. Effects of inorganic salts on fluoride storage in the rat. J. Nutr., 54:437-444.

# DEFLUORIDATION METHODS FOR SMALL COMMUNITIES

by Prof. Sorab Arceivala
Project Manager, (TUR/SES/005),
World Health Organization,
Ankara, Turkey.

*(Presented by Dr. Halit Göknil
Istanbul Technical University*

Several methods have been suggested from time to time for removing excessive fluorides. These may be divided into two basic types - those based upon an exchange process or absorption, and those based upon the addition of chemicals to water during treatment. The materials reported to have been used in the contact beds include processed bone, natural or synthetic tricalcium phosphate, hydroxyapatite, magnesia, activated alumina, activated carbons, and ion exchangers. Chemical treatment methods include the use of lime either alone or with magnesium salts and aluminium salts either alone or in combination with a coagulant aid. Other methods include the addition to fluoride water of materials like magnesia, calcium phosphate, bentonite, fuller's earth, bentonite and diatomaceous earth, mixing and their separation from water by settling and or filtration. All these methods suffer from one or other drawbacks: high initial cost, lack of selectivity for fluorides, poor fluoride removal capacity, separation problem, complicated or expensive regeneration. Some of these materials are dealt with briefly below.

Phosphatic Compounds: These include several bone formulations, synthetic tricalcium phosphate, and a mixture of calcium phosphate and hydroxyapatite.(1)(2)

Processed Bone: The bone is degreased, dried and pulverised to 40-60 mesh size. The powder is carbonised in a closed retort at 1,380-1,740°F. The product contains tricalcium phosphate, and has a capacity to remove 1,000-1,500 mg F per litre of medium. After saturation with fluorides, it can be recalcined at around 750°F, under restricted air supply to restore the absorbing capacity of the char. Alternatively, the bed may be regenerated by sodium hydroxide solution.

Bone Charcoal: The bone is processed by burning in air and pulverising to 60-100 mesh. The fluoride removal capacity of the product is about 1,000 mg F per litre of media.

Synthetic Tricalcium Phosphate: The product is prepared by reacting phosphoric acid with lime. It has a capacity of 700 mg F per litre. The medium is regenerated with 1 per cent sodium hydroxide solution, followed by a mild acid rinse.

Florex: It is a trade name for a mixture of tricalcium phosphate and hydroxyapatite. The fluoride removal capacity of the medium is 600 mg F per litre and is regenerated with 1.5 per cent sodium hydroxide solution. Florex was tried in Pilot Plant at Climax, Colo., U.S.A. in 1937 and Scobba, Miss., U.S.A. in 1940, but without much success owing to high attritional losses and the plants were abandoned(3).

Activated Alumina: The use of this material for the removal of fluoride ion from drinking water appears to have been first suggested by Boruff in 1934(1). The bed was regenerated with a 2 per cent solution of sodium hydroxide, followed by neutralisation of the excess alkali with dilute hydrochloride acid. The capacity of the medium was found to be about 800 mg F per litre of alumina. Many modifications of the process were suggested by subsequent workers. Several patents based on the use of aluminium-oxide for fluoride removal were issued to Heinzel and Churchill in 1936, Goetz in 1938, and Urbain and Stemen in 1940. Savinelli and Black(4) have used filter alum to regenerate activated alumina bed. The capacity of aluminium to remove fluorides was reported to be proportional to the amount of filter alum used for regeneration upto a level of about 0.2 kg alum per litre (12 lbs alum per cft) of alumina. At this level, the fluoride removal capacity was approximately 5,500 mg F per litre of alumina. The most important single factor affecting the fluoride exchange capacity of alumina was the alkalinity of the influent water.

Lime: It has been observed that while giving lime treatment to waters containing magnesium salts, fluorides are absorbed on magnesium hydroxide flocs, and it results in fluoride removal(1). Empirically, the amount of fluoride removed is equal to 0.07 Mg, where F represents μmg fluoride initially present and Mg the mg of magnesium removed in the form of flocs. In this case, the water must be treated to a caustic alkalinity of 30 mg/l, a pH of 10.5 or above and as such recarbonation is later necessary(5). Magnesia and calcined magnesite have also been used for fluoride removal from water and the fluoride removal capacity was reported to be better at high temperatures(6).

Activated Carbons: Most of the carbons prepared from different carbonaceous sources showed fluoride removal capacity after alum impregnation. McKee and Johnston (7) have reported good fluoride removing capacity of various types of activated carbons. Venkataraman(8) prepared a carbon from rice paddy husk by digestion in 1 per cent KOH and soaking it overnight in 2 per cent alum solution. The material removed about 320 mg F per kg (150 mg F per l) and showed a maximum removal efficiency at pH 7. The carbon was regenerated by soaking the spent material in 2 per cent alum solution for 12-14 hours. A pilot plant with this material was installed at Guntakal, India., to treat water containing 2.8 mg F/l, which was since abandoned. Activated carbons prepared by other workers from cotton waste, coffee waste, coconut waste, etc. were tried for defluoridation, but all these materials were of academic interest only.

Ion Exchange Resins: Strong base anion exchange resins remove fluorides either on hydroxyl cycle or chloride cycle along with anions(9). Since the proportional quantity of fluoride as compared to other anions is very small, the effective capacity of such resins works out quite low. There are no known commercial anion exchange resins which are selective for fluoride only. Some inorganic ion exchangers, e.g. complex metal chloride silicate, formed from barium or ferric chloride with silicic acid, also exchange fluoride for chloride.

Cation exchange resins impregnated with alum solution have been found to act as defluoridating agents. Various workers have used cation exchange resins after treatment with alum solution for defluoridation. Venkataraman, et al., (10) reported that "Avaram bark" based cation exchange resins works effectively in removing fluorides from water.

## Defluoridation Work at the National Environmental Engineering Research Institute (NEERI), Nagpur, India.

Work on defluoridation was taken up by NEERI in 1961 on a reference from some State Governments afflicted with the problem(11). A review of the materials developed by different workers on defluoridation revealed that most of the methods involved acid and alkalis either during pre-treatment or regeneration and were not suitable for rural conditions. The phosphate compounds and ion-exchange material are not practicable on large scale. Paddy husk carbon demonstrated by Venkataraman has a low capacity and poor attritional quality.

Several materials like clays, minerals, ion exchange resins, activated carbons, activated alumina, sulphonated coals and serpentine were tried at NEERI for the removal of excess fluorides from water. Chemical treatment, in situ, with lime, magnesium salts, iron and aluminium salts were also studied. Only those that showed an encouraging trend on bench-scale were studied in detail, and are presented in this paper. These include, carbion, defluoron-2 and the Nalgonda technique. Ion exchange resins, saw dust carbon, defluoron-1, magnesia and serpentine did not prove useful beyond bench-scale.

Pilot plant studies were carried out at Gangapur, (Rajasthan) using carbion. Full size plants were installed, using defluoron-2, in two other regions, while the Nalgonda technique has been used successfully in over 200 villages in India.

The following results are reported from the works of Nawlakhe, Kulkarni and Pathak under the leadership of Bulusu (12)(13)(14).

## Carbion

Carbion is a cation exchange resin of good durability and can be used both on sodium and hydrogen cycles. It has a bulk density of 680 g/l with a grading of 16-30 mesh.

Laboratory experiments using 50 ml aliquots 4.3 mg F/l trst water, and regeneration using 200 ml of 1 per cent alum solution indicated an average fluoride removal capacity of 320 mg F/l or 470 mg F/kg carbion. A pilot plant was installed using a mixture of this medium with defluoron-1 in the proportion of 8:1 at Gangapur (Rajasthan). The plant was charged with 224 l of carbion and 28 l of defluoron-1. The total quantity of water treated was only 659.3 $m^3$.

The fluoride content of the raw water was 4.8 mg F/l. In all, 29 cycles of operation were studied, using filter alum as a regenerant. During the first few cycles of operation, most of the defluoron-1 was washed out during backwash operation due to its lightweight and what eventually remained in the plant was essentially carbion. The results of the study showed that 1.08 kg of filter alum were needed per $m^3$ of raw water fluoride concentration after treatment ranged between 0.88 and 1.6 mg F/l (11)(11a).

<u>Defluoron-2</u>

To overcome the problems faced defluoron-1, a medium "defluoron-2" was developed in 1968 for the removal of fluorides from drinking water(14). Extensive trials with the medium, both in the laboratory and in the field have shown that, it does not suffer from most of the handicaps which some of the indigenous materials developed earlier suffered from.

Defluoron-2 is a sulphonated coal and works on the aluminium cycle. It was found to give the best results with one bed volume of 4 per cent (w/v) alum solution as a regenerant. A bed volume is equal to the volume of media in the unit. The life of the medium was found to be 2-4 years.

To study the effect of prolonged use of this medium on fluoride removal capacity, work was carried out to an aggregate of 55 cycles of continuous operation of 100 mm diameter column charged with a total of 6 litres of medium.

The test water containing 5-6 mg F/l and 140-168 mg/l alkalinity was used throughout (50,51). The average fluoride removal capacity of the medium was 484 mg F per litre with 1.15 mg F/l as a mean value of the leakage.

Two plants with a capacity to treat 91 $m^3$ (20,000 gal) per regeneration were installed at the Municipal Corporation, Nalgonda and Central Training Institute, Hyderabad, India. The plant includes a pressure shell, regeneration tank and a storage reservoir of 4-6 hours capacity. The process in its simplest form consists of passing water through a bed of defluoron-2 medium contained in a cylindrical steel shell to which are attached the necessary pipework and control valves. Immediately adjacent to shell the regeneration tank is located so that the whole installation is compact and is easily maintained. The plants at Nalgaonda and CTI were operated through 55 and 40 cycles respectively, by NEERI. While the plant at CTI is operating, the plant at Nalgonda is kept idle by the Municipality for want of funds.

<u>Characteristics of Defluoron-2</u>

| | | |
|---|---|---|
| Bulk density | .. | 810 kg per $m^3$ |
| Size of grains | .. | 0.6-2.0 mm |
| Voids (approximate) | .. | 40 per cent |
| Attritional losses | .. | Negligible |

<u>Maximum operating service flow</u> rates:

(a) 5.0 $m^3$ per sq. meter of bed area per hour
(b) 5.5 $m^3$ per hour per $m^3$ of medium

Wash water treatment:

0.15-0.22 $m^3$ per sq. meter per minute for a maximum duration of 10 minutes.

Regenerant:

One bed volume of 4 per cent (w/v) alum solution. A bed volume is equal to the volume of the medium in the unit. The alum solution has a pH between 2.6 and 2.8.

Regenerant contact period with medium:

30-40 minutes.

Pressure drop through the medium of depth 0.6-1.0 m:

1.2-1.5 meter per meter depth of medium at 5 $m^3$ flow-rate per sq. meter bed area per hour.

## Alkalinity Tolerance

Bicarbonate alkalinity:

With raw water fluorides ranging between 8 and 12 mg/l the average defluoridation capacity is 650 mg fluoride per litre of medium. However, because of greater difficulty in fluoride removal at lower concentrations, the capacity of the medium reduces to 480 mg/l of medium, when using a raw water containing between 5 and 7 mg/l of fluoride. These capacities correspond to a raw water alkalinity of 160 mg/l as $CaCO_3$. The capacity of the medium at other alkalinity levels is given below:

FLUORIDE REMOVAL CAPACITY mg FLUORIDE PER l OF MEDIUM

| Bicarbonate alkalinity of the raw water mg $CaCO_3$/l | Raw water fluoride, mg per litre | | | |
|---|---|---|---|---|
| | 3-5 | 5-7 | 7-8 | 8-10 |
| 160 | 340 | 480 | 560 | 650 |
| 200 | 300 | 400 | 460 | 530 |
| 240 | 230 | 300 | 350 | 400 |
| 300 | 190 | 250 | 270 | 300 |
| 400 | 140 | 170 | 210 | 250 |
| 600 | 100 | 120 | 180 | 200 |
| 900 | 80 | 100 | 120 | 150 |

Based on an average fluoride concentration of 0.6-0.8 mg/l in the treated water.

Hydroxyl alkalinity:

The medium can tolerate hydroxyl alkalinity up to about 5 mg/l as $CaCO_3$. The fluoride concentration in the treated water increases with the increase in hydroxyl alkalinity. There is a 30 per cent reduction in the capacity of the medium when hydroxyl alkalinity is 25 mg/l as $CaCO_3$.

Continuous operation of the plant at Central Training Institute for about four years had resulted in the formation of white deposits over the medium and a consequent fall in the defluoridating capacity by about 60 per cent. The volume of the medium had swollen considerably (40 per cent). The following were the major constituents of the deposits:

| | | | |
|---|---|---|---|
| Carbonates and hydroxides | .. | 4,80,000 | ppm |
| Fluorides | .. | 6,100 | " |
| Sulphates | .. | 6,000 | " |
| Aluminium | .. | 1,73,000 | " |
| Calcium | .. | 24,000 | " |
| Magnesium | .. | 34,000 | " |
| Iron | .. | 11,000 | " |
| Acid insoluble (1:1 HCl) | .. | 1,69,700 | " |

The deposits were probably due to inadequate wash after regeneration with alum solution. The medium was taken out, acid washed and recharged. The plant has since been working. A typical installation is shown in Fig. 1.

Nalgonda Technique

Recently, NEERI has developed another method for fluoride removal which has been named the Nalgonda Technique after the region in which it was used (13). The method has been very successful and has been used in over 200 villages. Though the Defluoron-2 process was also successful in removing fluorides, the periodic regeneration and maintenance of the plant required a certain degree of skilled operation which was not available everywhere. The process also required the manufacture of the medium Defluoron-2 which though not difficult in itself, required establishing special facilities.

The Nalgonda Technique, on the other hand, only involves the addition of two readily available chemicals -

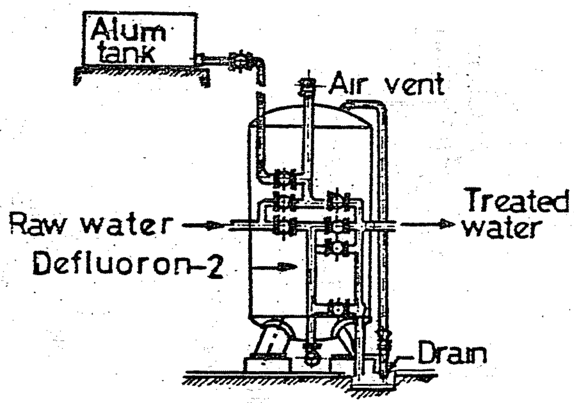



FIGURE 1

Defluoridation Using
DEFLUORON-2 Process

lime and alum - to the raw water followed by stirring and settling. The technique could be adapted to any size of population, large or small, and could even be used in the home by a family. For home use, the required chemicals could be supplied in the form of tablets with simple instructions to add the required number of tablets, first of lime and then of alum, to a container of water, hand-stir it for a while and allow it to settle. The supernatant could be decanted off and used. Bleaching powder for disinfection could be added along with lime and alum, if required.

With domestic treatment, no capital investment is required, and the cost of treatment is limited to the cost of chemicals only.

On a community-scale, the process could be used equally easily, on intermittent or continuous basis, using the following general flowsheet which is typical of a water treatment plant (Figure 2).

The dose of lime and alum required depends on the

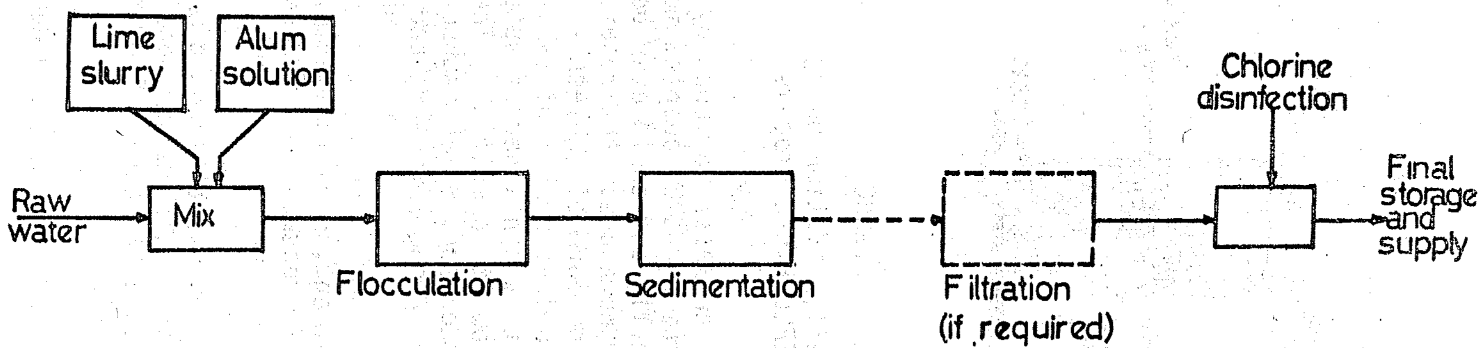



FIGURE 2

NALGONDA TECHNIQUE FOR DEFLUORIDATION

fluoride concentration and alkalinity present in the raw water. Table 1 gives some idea of the alum requirements in order to produce a final water with fluoride concentration of 1 mg/l or less to meet drinking water standards. Lesser doses of alum are required if fluoride concentrations upto 2 mg/l can be allowed in the final waters. The lime dose required is 1/20 of the alum dose in all cases. Conditions comprising high fluorides and low alkalinity are not usual in India. When such conditions prevail, the alkalinity of the raw water can be increased as desired by lime.

Some consideration may need to be given to the total dissolved solids in the final water in case of raw waters with excessive dissolved solid, concentration.

TABLE 1

RELATION BETWEEN ALUM DOSE, AND THE RAW WATER CONCENTRATION OF FLUORIDE AND ALKALINITY, TO GIVE FINAL WATER WITH 1 mg F/l OR LESS (LIME DOSE = 1/20 OF THE ALUM DOSE)

| Raw Water Fluorides mg F/l | Approximate Alum*Dose at Different Raw Water Alkalinities as mg $CaCO_3$/l | | | | | | |
|---|---|---|---|---|---|---|---|
| | 125 | 200 | 300 | 400 | 500 | 600 | 820 |
| 2 | 143 | 221 | 273 | 312 | 351 | 403 | 468 |
| 4 | - | 403 | 416 | 468 | 559 | 598 | 689 |
| 6 | - | - | 611 | 715 | 780 | 936 | 1066 |
| 8 | - | - | - | - | 988 | 1118 | 1300 |
| 10 | - | - | - | - | - | - | 1508 |

* Filter alum (16-17% alumina)

The mixing, flocculation and sedimentation stops are identical to those usually employed in conventional water treatment. Sand filtration may be required for better clarification, but could be omitted in case of certain ground waters. Disinfection may be provided as shown in Fig.2. The treated water is then stored and supplied as required. Any pH adjustment is generally not necessary.

## CONCLUSION

Engineers are familiar with the design and construction of conventional water treatment plants and should be capable of installing and operating a plant of the type just described. As stated earlier, the Nalgonda Technique is capable of being adapted to both community-wide service and simple home use, with a simplicity well within the competence of any villager. The Defluoron-2 process could also be used if desired.

It is hoped that a pilot installation will soon materialize in Turkey so as to gain actual operating experience with the method or methods considered most suitable under Turkish conditions.

# REFERENCES


1. Boruff, C.S., "Removal of Flüoride from Drinking Water" Ind. Eng. Chem. Vol. 27, p. 69 (1934).

2. Smith, H.V., Smith, M.C., "Bone Contact Removes Fluorides" Water Works Vol. 90, p. 1600 (1937).

3. Swope, G.H. and Hess, R.H. "Removal of Fluoride from Natural Water", Ind. Eng. Chem. Vol. 29, p.424(1937)

4. Savinelli and Black, "Defluoridation with Activated Alumina" J. Amer. Wat. Work Assoc., Vol. 50, p. 33, (1958).

5. Maier F, "Defluoridation of Municipal Water Supply" J. A.W.W.A., Vol. 45, (1953).

6. Venkateswarlu, P., and Rao, N., "Fluoride Removal by Magnesia" Indian. Med. Lab., Vol. 41, p. 475, (1953).

7. McKee, R. Johnston "Removal of Fluorides", Ind. Eng. Chem. Vol. 26, p. 848, (1934).

8. Srinivasan T., "Alkali-Alum Impregnated Paddy Husk Carbon", CPHERI Bulletin Vo. 1, p. 30 (1959).

9. Kunin and McGarve, "Fluoride Removal by Strong Base Anion Exch. Resin", Ind. Eng. Chem. Vol. 41, p. 1265, (1948).

10. Venkataraman et al., "Indian Jour. Medical Research, Vol. 39, (1951).

11. Bhakuni, T. "Studies on Fluoride Removal by Indigenously Developed Ion-Exchange Material", Ph.D Thesis, Nagpur University, (1970).

11a. Bhakuni and Sastry, "Defluoridation Using Cation Exchangers", Indian Jour. Environ. Health, Vol. 6, p. 246, (1964).

12. Bulusu, K.R., Pathak B.N., "Defluoridation Methods and their Limitations", Report prepared for NEERI, Nagpur, India, (1976).

13. Nawlakke, Kulkarni, Pathak and Bulusu "Defluoridation of Water by the Nalgonda Technique", Indian Journal of Environmental Health, Vol. 17, p. 26 (1975).

14. Thergaonkar et al, "Defluoron-2 for Fluoride Reduction", Ind. Jour. Env. Health, Vol. 11, p. 108, (1969)

# TEA DRINKING AS AN ALTERNATIVE
# TO FLUORIDATION OF WATER SUPPLIES

by Prof. Dr. K. Simjour,
Faculty of Denistry,
University of Tehran.

(Abstract)

The beneficial dental effects of fluoridation of water supplies to a whole population are well known.

The concentration of fluoride in different varieties of tea has been measured. It is shown that the concentration ranges to values well above 200 parts per million in the dry tea leaves. An experimental study shows that in the normal process of making tea if the tea is allowed to steep for 10 minutes the majority of the fluoride are dissolved in the liquid.

It is pointed out that most of the advantages obtained by fluoridation of the water supplies for the whole population may be obtained on an individual basis by the appreciable consumption of tea.

## CONCLUSIONS AND RECOMMENDATIONS

1. The meeting noted the several effects on human health experienced by persons who, over long periods, drink water containing excessive levels of fluoride. The ill-effects of high fluoride waters on animal health were also noted.

2(a) The meeting noted the apparent differences in the results of measurements of the fluoride content of waters from similar sources. This suggests the need for comparisons of the different methods of measurement in use.

2(b) The meeting recommended that measurements should be continued on a comparative-survey basis of the fluoride content of the groundwaters in the Mount Ararat Region in both Turkey and Iran, and that similar surveys should be carried out in the high fluoride areas in Pakistan wherever necessary.

3. The meeting noted some interesting hypotheses and problems of the clinical effects of high fluoride waters which have been observed from preliminary observations and which were presented and discussed. The importance of these exploratory studies and investigations was recognised. It is recommended that a well-defined epidemiological study be undertaken to test such hypotheses in areas where these conditions occur. In particular:

(a) The environmental objectives should be clarified.

(b) It is necessary to carry out plant and animal monitoring.

(c) There should be regular monitoring of drinking-water sources.

(d) There should be biological testing of the population at risk.

(e) Dental health studies should be included in these studies.

4. The meeting recommended that a workshop/study group

be called together for the implementation of these recommendations.

5. The meeting noted that there seems to be appreciable knowledge of methods of defluoridation already available. It is recommended that a further study of these methods and the introduction of a pilot scheme for defluoridation of drinking water should be undertaken simultaneously.

6. The meeting noted the contribution that air pollution made to the levels of fluoride in certain areas. It is recommended that indicator plant monitoring be introduced around air pollution sources.

7. The meeting also drew attention to the importance of other possible harmful chemical pollutants in water supplies.

CENTO SCIENTIFIC PROGRAMME PUBLICATIONS

*1. High Yielding Wheat: Panel held in Tehran, December 1970.

*2. Environmental Pollution: Panel held in Tehran, March 1971.

*3. Food Processing: Travelling Workshop held in Izmir, Tehran, and Lahore, September 1971.

*4. Scientific Laboratories, Organization and Administration: Travelling Workshop held in Lahore, Tehran and Istanbul, April 1972.

*5. Cereals: Programme Meeting held in Lahore, December 1972.

*6. Natural Drugs, and their Commercial Utilization: Panel Meeting held in Peshawar, December 1972.

*7. Pests and Diseases of Wheat: Panel Meeting held in Karaj, Iran, February 1973.

8. Rural Housing: Symposium held in Ankara, May 1973.

*9. Village Water: Programme Meeting held in Izmir, November 1973.

*10. Village Water: Project Report, 1971-1973.

11. Parasitic Diseases: Symposium held in Shiraz, December 1973.

12. Scientific Documentation: Conference held in Tehran, April 1974.

13. Economics of Nuclear Power Plants: Symposium held in Ankara, June 1974.

*14. Cereals: Programme Meeting held in Izmir, November 1974.

*15. University Services for Industry: Panel Meeting held in Lahore, December 1974.

16. Directory of Scientific Research Institutes in the CENTO Region (1976).

*17. Optimum Use of Water in Agriculture: Panel Meeting held in Lyallpur, Pakistan, March 1975.

*18. Earthquake Engineering: Symposium held in Ankara, November 1974.

*19. Regional Problems of Oils and Fats: Panel Meeting held in Lahore, December 1975.

20. Environmental Problems of Liquid Effluents: Symposium held in Karachi, February 1976.

*21. Plant Production under Saline Conditions: Symposium held in Adana, May 1976.

22. Wheat Pests: Symposium held in Ankara, June 1976.

23. Ten Years of CENTO Science: A review of the CENTO Scientific Programme, 1967-1976.

24. Cereal Technology: Programme Meeting held in Shiraz, July 1976.

25. 1977 Directory of Scientific Research Institutions in the CENTO Region.

*26. Solar Energy

27. Earthquake Hazard Minimization, Tehran, November 1976.

* *Out of print*

*These reports are available free of charge from:*

*CENTO Scientific Secretary,*
*P.O. Box 1828,*
*Tehran, Iran.*

*or*

*Mr. Nezih Biket,*
*Public Relations Division,*
*Central Treaty Organization,*
*Eski Buyuk Millet Meclisi Binasi,*
*Ulus, Ankara,*
*Turkey.*

*NB. The Technical Editor regrets that becuase of a staff shortage she is unable to send replies to those individual requests for CENTO publications which are out of stock and thus no longer available.*