Chapter - 1

INTRODUCTION AND REVIEW OF LITERATURE

Water is essential to sustain life and has long been suspected of being the source of many illnesses in human beings. It was not until a little over 100 years ago that definite proof of disease transmission through water was established. Although the provision of clean water and sanitation is often omitted from the list of priority environmental challenges, in many parts of the developing countries it ranks at the top. Still, access to safe water remains an urgent human need in many parts of the world. The problem is compounded in some places by growing water scarcity which makes it difficult to meet increasing demand except at escalating cost.

As population increases, the demand for water grows accordingly and at a much more rapid rate if the population growth is accompanied by improved living standards. Official WHO figures also suggest that between 1980 and 1990 more than 1.6 billion additional people were provided with access to water of reasonable quality. At least, 170 million people in urban areas still lack a source of potable water near their homes, and in rural areas, although access has increased rapidly in the past decade, more than 855 million are still without safe drinking water (World Bank, 1992).
The use of polluted water for drinking and bathing is one of the principle pathways for infection or causing disease which kill millions and sicken more than a billion people each year.

**FLUOROSIS - A CRIPPLING SCOURGE:**

Excess of fluoride intake in our body may result in a slow progressive, crippling scourge known as fluorosis. It was first detected in India, among cattle by the farmers of Andhra Pradesh during early 1930s (RGNDWM, 1993). Shortt et al. (1937) had published the first report on endemic fluorosis from India. The disease was then known to be prevalent in 4 states in India i.e. Andhra Pradesh, Tamil Nadu, Punjab and Uttar Pradesh. However, a recent report from Rajiv Gandhi National Drinking Water Mission elucidates that fluorosis is prevalent in various proportions in a total of 15 states. It is estimated that about 25-30 million people in about 150 districts in India are suffering from varying grades of fluorosis. With the available information on the prevalence of the disease, the states have been categorized as follows:

<table>
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<tr>
<th>Category and Percentage of affliction</th>
<th>Name of the States</th>
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| Category - I  
(Less than 30% of districts affected) | Jammu & Kashmir, Delhi, Kerala and Orissa |
| Category - II  
(30 to 50% of districts affected) | Punjab, Haryana, Madhya Pradesh, Maharashtra, Karnataka and Bihar |
| Category - III  
(50 to 100% of districts affected) | Uttar Pradesh, Rajasthan, Gujarat, Andhra Pradesh and Tamil Nadu |
Globally, it has been reported that apart from India, there are about 20 other developed and developing nations which have come under the threat of fluorosis. These are Argentina, United States of America, Morocco, Algeria, Libya, Egypt, Jordan, Syria, Turkey, Iraq, Iran, Pakistan, Kenya, Tanzania, South Africa, China, Australia, New Zealand, Japan and Thailand.

**SOURCES AND OCCURRENCE OF FLUORIDE:**

Fluoride is a geochemical contaminant and it enters into the body through water, air, medicines and cosmetics. Karunakaran (1974) has reported the most common occurrence of fluoride minerals. Due to the rich mineral content, fluoride leaches out and contaminates the water, earth or soil.

**CHEMICAL PROPERTIES OF FLUORINE:**

Fluorine is the most reactive element known among all halogens with a strong affinity to combine with other elements to produce fluorides. It is estimated to be the 13th most abundant element in the earth’s crust. Due to the most electronegative of all the elements, fluorine is the strongest oxidising agent known.

**HALF-LIFE:**

The detailed studies on toxicokinetics revealed that the absorbed fluoride is distributed between blood and soft tissues and the skeleton. The half-life of fluoride in blood and soft tissues has been reported to be few hours, while in skeleton, it has a longer half-life of about 8 years (WHO, 1970; Philippee, 1985).

**FLUORIDE TOXICITY AND BODY METABOLISM:**

In fluorosis the dental and skeletal structures are affected severely besides other organs or systems.
DENTAL FLUOROSIS

Dental fluorosis is seen in children who are born and brought up in an endemic region. This condition can occur in milk teeth and permanent teeth. The teeth become discoloured and mottled due to excess fluoride intake.

SKELETAL FLUOROSIS

Numerous structural changes occur in a fluorosed bone viz. increased bone mass and density, bony outgrowth, increased trabecular bone volume, cortical porosity, increased osteon diameter and their mottling, formation of unmineralized cartilaginous loci within the trabeculae of cancellous bone but not the cortical bone, accumulation of glycosaminoglycans, etc.

Extensive work has been done on skeletal and dental fluorosis, which is well-documented all over the world. But, there is a paucity of data on effects of fluoride toxicity and its metabolism on soft tissues.

Fluoride induced toxicity also affects general body and tissue metabolism. Therefore, the role of fluoride in soft tissue metabolism is presented here.

FLUORIDE AND PROTEIN METABOLISM

Fluoride inhibits protein synthesis mainly due to impairment of polypeptide chain initiation (Hoerz and McCarty, 1971; Vesco and Colombo, 1970; Godchau and Atwood, 1976; Holland, 1979). The reduction in protein is also due to weak incorporation of amino acids into proteins as a result of their abnormal accumulation in these organs (Helgeland, 1976). The protein levels in stomach, duodenum and ileum of fluoride treated rabbits were also decreased (Zhavronkov and Strochkova, 1981; Shashi et al., 1987). Recently, it has been reported that protein levels were
significantly reduced in various reproductive tissues, liver, muscle, kidney of fluoride intoxicated laboratory animal models (Chinoy and Sequeira, 1989a; Chinoy, 1991a,b,1992; Chinoy et al., 1991a,b; 1992a; 1993a; 1994a,b). It was also reported that fluoride treatment induces formation of some stress proteins in testis and epididymis of rats. This is the first report of its kind (Chinoy et al., 1994b,e). Susheela and Sharma (1980) reported very low levels of glycoproteins in rabbit blood plasma by low dose (10 mg/kg body weight) of NaF, while, a higher dose (50 mg NaF/kg of body weight) increased their levels, which is attributed to inhibition of certain lysosomai enzymes reducing glycoprotein catabolism and thereby causing its accumulation.

**FLUORIDE AND CARBOHYDRATE METABOLISM:**

Fluoride causes severe changes in carbohydrate metabolism. Rabbits treated with fluoride showed a decrease in glycogen concentration in various organs like spleen, lens, liver and skeletal muscle (Shashi et al., 1988). On the contrary, glycogen accumulation occurred in fluoride treated fishes (Shaikh and Hiradhar, 1985; Chinoy et al, 1994c) and in liver, muscle, vas deferens and uterus of rats and mice (Chinoy and Sequeira, 1989a; Chinoy et al, 1991a; 1993b; 1994d), concomitant with a reduction in the activity of phosphorylase (Chinoy et al, 1993b).

Cell structure studies demonstrated inhibition of enolase levels suggesting that fluoride reduces cell growth through an enolase mediated inhibition of glycolysis (Suttie et al., 1974). They further determined the levels of 3-phosphoglyceric acid (3 PGA), 2-phosphoglyceric acid (2 PGA), phosphoenol pyruvate (PEP) and pyruvate. The results revealed an accumulation of 3 PGA and 2 PGA relative to PEP and a 20-
25% curtailment in glycolytic flux during this period. A decrease in isocitrate dehydrogenase and accumulation of citrate which is known to be a negative effector for phosphofructokinase was reported by Dousset et al. (1987). Inhibition of enzymes involved in glycolysis could also affect carbohydrate levels. An increase in serum citrate levels was observed in fluoride treated rats (Hac and Freeman, 1967) and in horses grazing on fluoride contaminated forage (Lovelace et al., 1968). Similarly, the liver citrate concentrations were augmented in rats fed fluoride in the diet (Shearer and Suttie, 1970). The activity of liver aconitase, isocitrate dehydrogenase, malate dehydrogenase, citrate condensing enzyme and citrate cleavage enzyme (all of which could influence the state of citrate levels) were not affected by 600 ppm fluoride fed to rats. Therefore, fluoride was not manifesting any direct effects on the activities of TCA cycle enzymes (Shearer et al., 1971). However, higher concentrations of fluoride inhibit some glycolytic enzymes. Hence, fluoride affects carbohydrate metabolism mainly through inhibition of glycolytic pathway, rather than affecting citrate metabolism/tricarboxylic pathway.

FLUORIDE AND LIPID METABOLISM:

Fluoride has been implicated in atherosclerosis, hence its relationship with lipid metabolism assumes considerable importance (Exner and Waldbott, 1957). In rats fed 100 ppm of fluoride a marked reduction in plasma free fatty acid occurred either due to partial inhibition of lypolysis or to the lesser availability of depot fat in the extra-hepatic tissues. The liver and serum lipid fractions were also affected (Saralakumari et al., 1988). The treatment brought about an increase in total lipid and triglycerides in the liver of fluoride treated rats (Townsend and Singer, 1971) which
suggests the formation of fatty liver. However, in rabbits treated with NaF, liver triglycerides decreased with a concomitant decline in lipase activity (Singh et al., 1985). Similarly, phospholipids, cholesterol in several tissues of rats and mice and sudanophilic lipids of rat gastrocnemius muscle were also reduced after NaF treatment (Chinoy, 1992; Chinoy et al., 1993b; Narayana and Chinoy, 1994a; Chinoy et al., 1994d).

In guinea pigs exposed to hydrogen fluoride (HF), a significant enhancement in plasma cholesterol and glucose-6-phosphate dehydrogenase activity occurred. The enzyme is highly specific for NADP+ and will result in increased levels of H+. The increased cholesterol biosynthesis was co-related with acceleration of pentose phosphate pathway due to the enhanced production of NADPH by HF (Dousset et al., 1987). According to Leipzig et al. (1967), excess fluoride intake decreased the triglycerides but failed to influence serum cholesterol. Work from our laboratory (Chinoy and Sequeira 1989a; Chinoy, 1991a,b; Chinoy et al., 1992a; 1994a) have also elucidated that cholesterol levels in testis and serum were within the normal range in short-term fluoride treated laboratory rodents and in endemic human population of North Gujarat. However, later studies revealed that cholesterol levels, activities of 3β and 17β hydroxysteroid dehydrogenase, Leydig cell functions of rat testis and circulating testosterone were affected causing reduction in steroidogenesis (Narayana and Chinoy, 1994a).

**FLUORIDE AND OTHER INORGANIC CONSTITUENTS:**

Certain elements are known to influence fluoride toxicity; as they have affinity to form complexes with fluoride e.g., Ca²⁺, Mg²⁺ and phosphorus etc., and therefore
reduce fluoride action.

In the laboratory study, calcium kinetics showed that the cumulative retention of radioactive calcium was enhanced in animals fed with fluoride, but higher retention was observed in those receiving low calcium diets (Krishnamachari, 1978). Urinary calcium excretion, its deposition into bone and mass of exchangeable calcium were not affected by fluoride. However, fecal excretion of fluoride was very low indicating that this was an indirect effect (Ramberg et al., 1970). That $\text{Ca}^{2+}$ and $\text{P}$ have little effect on fluoride metabolism in man was reported by Spencer et al. (1975), whereas, Wanger and Muhler (1960) found a reduction in absorption of fluoride by $\text{Ca}^{2+}$ intake. The results of Rogler and Parker (1972) elucidate the beneficial effects of excess $\text{Ca}^{2+}$ on the $\text{F-Mg}^{2+}$ interrelationship mediated primarily through reduced absorption of fluoride. Recently, Kharb and Susheela (1994) have reported that total inorganic content in ligament and the aorta was increased significantly due to the deposition of calcium in these tissues of NaF treated rabbits. Calcium was increased possibly because of the formation of octa-calcium phosphate, dicalcium phosphate dehydrate or amorphous calcium phosphate.

Apart from calcium, magnesium and phosphorous, fluoride is also known to alter the levels of copper, manganese and zinc in femur bone and manganese and zinc in kidney due to their affinity (Singh and Kanwar, 1981). Copper depletion in bone is known to impair erythropoiesis hence cause anemia.

**EFFECTS OF FLUORIDE ON SOME TISSUES AND SYSTEMS:**

**Muscle:**

Fluoride altered the structure and metabolism of muscle cells by chronic
treatment to rats (Kaul and Susheela, 1974). The muscles revealed reduction of fibers, vacuolization and necrosis (Shashi, 1989). Higher doses of NaF in rabbits increased the serum phosphocreatine kinase levels, which is an index of degeneration of muscle fibres and high permeability of plasma membrane (Kaul et al., 1974). Similarly, ingestion of lower doses of NaF (i.e. 5 and 10 mg/kg body weight) in prepubertal and adult rats for 30 days caused a decrease in muscle SDH, lipid, ATPase and protein levels but an increase in muscle cholinesterase (Chinoy et al., 1993b). These data reveal that the oxidative metabolism and contractile mechanism of the muscle fibres would be affected in fluorotic animals.

Excess of fluoride intake can disturb the collagen formation, but interfere with normal hydroxylation of proline causing intermediates of tropocollagen molecules and decrease the amount of soluble and insoluble collagen (Chinoy, 1991b).

Skin:

Waldbott and Steingger (1973) described a sign of chronic toxicity of fluoride poisoning which occurred in children and women, mainly pinkish to bluish-brown skin lesions called "Chizzola Maculae" i.e. inflammation around capillary blood vessels. These signs were not produced experimentally. This data suggests that other factors besides fluoride may be involved in these lesions.

Liver:

Zonal necrosis is the most common symptom in liver of NaF treated rats, mice and mudskippers. The hepatic cells were hyalinized, loss and cells and cytoplasmic vacuolization was observed. The arrangement of hepatic cord was also disturbed (Kour and Koul, 1981; Chinoy, 1991a,b; Chinoy et al., 1991a). Therefore, the structural
alterations would affect the liver metabolism. The significant increase in the activities of serum transaminases (SGOT and SGPT) in animal models and human endemic population indicate alterations in liver function as these enzymes are specific markers (Chinoy, 1991a,b; 1992; Chinoy and Narayana, 1992; Chinoy et al., 1992b; 1994f). Similar results were also reported by Tsunoda et al. (1985) in goats exposed to airborne-fluoride. Experimental evidence showed that ingestion of fluoride in rats and mice resulted in an increase in vitamin C in liver and adrenal which might help to overcome the stress due to treatment (Chinoy, 1991a,b; 1992; Chinoy et al., 1993b).

A significant decrease in serum protein correlated with the liver damage was observed in rats given a dose of 10 mg NaF/kg body weight (Chinoy, 1991a,b; 1992). Kaur et al. (1978) also reported a fall in total serum proteins in fluoride ingested rabbits. Similar findings were also reported in fluorotic human population of north Gujarat (Chinoy et al., 1992b; 1994f). An accumulation of triglycerides in the liver of fluoride treated rats led to the formation of fatty liver (Saralakumari et al., 1988). Hence, it is evident that fluoride affects liver structure and its metabolism.

Kidney:

Kidney is the main organ through which maximum concentration of fluoride is being excreted. Any alteration in the structure of kidney would affect its function. In mice, cloudy swelling of the kidney tubular cells, marked necrosis and atrophy of the glomeruli occurred which affected kidney function (Kour and Singh, 1980a). The total lipids, cholesterol, triglycerides and phospholipids were decreased in the kidney of fluorotic rats. The renal and serum Na⁺, K⁺ levels were altered in rats which would affect the electrolyte balance and kidney function (Chinoy, 1991a, b; 1992).
Blood:

Haematological studies in patients with fluorosis has yielded controversial data. Industrial exposure to fluoride in England revealed normal blood count and haemoglobin concentration. On the other hand, Macuch et al. (1963) reported decreased haemoglobin, but increased erythrocyte and abnormal lymphocyte count in children living near aluminium plants. Morphological abnormalities in cell structure and mitotic figure formation in immature leukocytes of mice given NaF in drinking water were also observed (Greenberg, 1982).

The coagulation time of blood was prolonged in goats fed with fluoride for 20 months. This is attributed to precipitation of calcium fluoride.

DIGESTIVE SYSTEM:

The fluoride gains access into our body by ingestion. The site of fluoride absorption is the gastrointestinal tract. Symptoms of vomiting, abdominal pain and diarrhoea due to the formation of hydrofluoric acid in the gut were noticed.

An experimental study on rabbits by Shashi and Singh (1987) revealed that fluoride affected cellular protein synthesis in gastrointestinal organs. The erosion and necrosis of mucosal and submucosal layers, diffused absorption and disintegration of gastric glands occurred which produced systematic effects such as depilation and spastic paralysis (Shashi and Singh, 1987). Rajiv Gandhi National Drinking Water Mission (1993) has further elaborated that acute abdominal pain, constipation, blood in stools, bloated feeling (gas), tenderness in stomach, nausea (flu like symptoms) and mouth sores, loss of appetite are common complaints due to fluoride toxicity. Fluoride is know to combine with hydrochloric acid of the stomach and is converted
to hydro fluoric acid (F\(^-\) + HCl \(\rightarrow\) HF + Cl\(^-\)) which is highly corrosive. The stomach and intestinal mucosa is destroyed with loss of microvilli, damage to intestinal tract is affected by a variety of factors such as the chemical nature of ingested fluoride, solubility, pH, gastric acidity, nature and extent of other constituents of ingesta, the simultaneous presence of strongly fluoride-binding ions etc. (Burke et al., 1973; Ekstrand and Ehrnebo, 1979). Thus, the highest amount of fluoride consumed by man is chiefly absorbed in intestine.

**RESPIRATORY SYSTEM:**

Respiratory system is yet another route of entry of fluoride into the human body especially in the vicinity of industrial zones. Industrial fluorosis is associated with exposure to fluoride containing chemicals or fumes in aluminium smelters, fluorine manufacturing and processing industries, fertilizer, ceramic, glass, brick-works and insecticide industries. Air-borne fluoride is readily absorbed through the respiratory tract and immediately ionises in contact with blood, and is carried by it to different parts of the body. Exposure to gaseous fluoride causes respiratory irritation. In mouse, rat and guinea pigs exposed to different concentration of hydrogen fluoride, irritation of mucous membranes of the nose and eyes, acute inflammation, focal necrosis of the nasal mucosa and tracheobronchitis, depressed respiration, bronchial asthma, necrosis and congestion in lungs has been reported in acute toxicity (Rybicki, 1970; Rigaud et al., 1976; Dipasquale and Davis, 1971; Wohlschlagel et al., 1976).

**EXCRETORY SYSTEM:**

No sex difference in fluoride excretion has been found (Toth and Sugar, 1978;
Vandeputte et al., 1977). Ingestion of fluoride in higher doses induced necrosis of the convoluted tubules and inflammation of glomeruli, which led to impaired kidney function such as polyurea and increased non-protein nitrogen (Jankauskas, 1974). Nephropathy is the principle manifestation of fluoride toxicity in the early stage of exposure.

As fluoride ion in larger doses undergoes filtration in the kidney, a reduction in its efficiency to eliminate fluoride would lead to elevated serum fluoride levels with a tendency for accumulation in bone (Parsons et al., 1975). In normal persons, kidney performs the function of fluoride homeostasis along with calcified tissues. In residents of fluoride afflicted regions, renal tubular and glomerular dysfunctions were investigated (Reggabi et al., 1984). A significant decline in glomerular functions including area clearance, creatinine and fluoride excretion in fluoride affected human subjects were observed (Jolly et al., 1980). Fluoride has been found to be implicated in renal calculi. In a different study, conducted in Germany, fluoride content in the incidence of renal calculi was twice or as high as that found in the residents of non-fluoridated area (Hesse et al., 1978).

CENTRAL NERVOUS SYSTEM (CNS):

Lu et al. (1961) have reported the stimulation of CNS by intraperitoneal injection of NaF to rats. A diet of 70 ppm of NaF, caused an increase in sensitivity to the paralytic effects of succinylcholine in rats, which was attributed to the inhibition of cholinesterase activity, an effect which was reversible. Latency and/or disruption of some of the learned responses were observed by hydrogen fluoride administration to rats. Hence, the mechanism of fluoride toxicity on central nervous
system is not yet clear.

**CARDIOVASCULAR SYSTEM:**

There is a paucity of data on the effects of fluoride toxicity on the cardiovascular system. Hypertrophy of ventricles and increased capillary permeability in workers of aluminium factory have been reported. Intravenous dose of fluoride caused a depression of blood pressure, heart and respiratory rate. Caruso et al. (1970) observed a direct vasodilatory effect of fluoride. It was reported by Susheela and Kharb (1990) and Kharb and Susheela (1994) that administration of fluoride to rabbits caused ectopic calcification in the aorta.

**IMPACT ON ENDOCRINE SYSTEM AND HORMONAL ALTERATIONS:**

Environmental fluoride also causes alterations in hormonal profile and endocrine function. In one of the investigations, estimations of circulating immunoreactive growth hormone values in adolescent patients with severe bone deformities due to skeletal fluorosis showed increasing levels (Sivakumar, 1977). Abnormality in vitamin D metabolism is attributed to fluorosis (Krishnamachari, 1986). However, the mechanism of these changes is not understood.

**THYROID GLAND:**

The actual relationship between goiter and fluoride toxicity is very limited. Jolly et al. (1976) found no significant differences in basal metabolic rate, protein bound iodine (PBI) and serum cholesterol values between the two study group of control and skeletal fluorosis. Tiagi et al. (1972) also reported normal PBI levels in four persons who had skeletal fluorosis. McLaren (1976) reviewed the effect of fluoride on thyroid gland and concluded that an increased accumulation of fluoride
occurred in thyroid as compared to the other organs, when expressed on wet weight basis. In experimental animals, the morphological changes included thyroid hypoplasia, colloid goitre, degeneration of follicular epithelium and true granular hypertrophy (Wadhwani and Ramaswamy, 1953). Chongwan and Daijei (1988) demonstrated swelling of mitochondria with disintegrated cristae in follicular epithelial cells of thyroid gland in fluoride intoxicated rabbits. In addition to morphological changes reported by many workers, functional alterations in thyroid gland metabolism were reported in experimental animals subjected to excess feeding of fluoride.

PARATHYROID GLAND:

A great deal of attention has been paid to the role of fluoride on parathyroid gland functions, since the gland plays a key role in calcium metabolism. Faccini and Care (1965) demonstrated augmented serum immuno reactive parathyroid hormone levels in young sheep reared on water containing 100 ppm fluoride. Similar data is available for human fluorotic subjects but the levels of calcitonin were not affected (Sivakumar and Krishnamachary, 1976). However, Teotia et al. (1978) obtained simultaneous elevation of circulating parathyroid hormone and thyrocalcitonin levels which were substantiated by Makhni et al. (1980).

REPRODUCTIVE SYSTEM:

Sodium fluoride in drinking water improved normal reproduction over an extended period for two generations in mouse. Higher intake of fluoride, after previous exposure to a low fluoride intake restored fertility (Messer et al., 1972). Fluoride is required in the diet of mice to support normal reproduction. But low
concentration of fluoride have failed to show an impairment of reproduction in rats or mice (Tao and Suttie, 1976).

Later experiments on testis of mouse showed that the treatment of 500 and 1000 ppm fluoride for a month caused necrosis in the seminiferous tubules which lacked differentiation and maturation of spermatocytes (Kour and Singh, 1980b). Chinoy and Sequeira (1989b; 1992) reported that mice fed with 10 and 20 mg per kg body weight of sodium fluoride (NaF) for 30 days caused alterations in testis, epididymis, vas deferens and fertility impairment.

In rats, when given a single microdose (50μg/50μl) injection of NaF directly into the vas deferens, similar histological changes were observed in testis (Chinoy et al., 1991c). This data revealed that fluoride intoxication has a significant role in male reproductive system.

In mice, rats, and guinea pigs withdrawal of NaF treatment and/or administration of ascorbic and/calcium alone or in combination for 30 to 70 days brought about significant recovery in all NaF induced effects. This finally contributed towards restoration of normal structure and metabolism of the target organs. As a result, a significant recovery in sperm motility was observed (Chinoy and Sequeira, 1992; Narayana and Chinoy, 1994b). But, on the whole, a faster recovery was observed by ascorbic acid or calcium feeding to NaF treated animals. A synergistic effect was noticed by combined administration of ascorbic acid and calcium (Chinoy, 1991a,b; 1992; Chinoy et al., 1991 b; 1993b; 1994a,b,d,e; Narayana and Chinoy 1994b).
In Vitro studies in human spermatozoa indicated decrease in the lysosomal enzyme activity after 20 minute treatment which could have been due to the gradual increase in fluoride accumulation by spermatozoa leading to membrane damage. Significant elevated values were also reported in case of ACPase and hyaluronidase activities by the 250 mM dose (Chinoy and Narayana, 1994).

DEFLUORIDATION

Since, Fluorosis is prevalent all over the world, it is essential to lower fluoride levels in our natural waters.

Fluoride content of natural waters could be brought down to safe limits by any one of the following ways:

1. By diluting the high fluoride containing water with low fluoride containing waters.

2. By chemical methods of treatment of the water such as formation of insoluble compounds containing fluoride or using ion exchange methods or by absorption.

Various methods are available for the defluoridation of water. Viswanadham et al. (1974) have tested about 20 readily available and relatively cheap materials for their capacity to remove fluoride from waters containing fluoride upto 10 mg/L. Magnesium oxide, calcium phosphate and aluminium oxide were the most effective defluoridating agents among all the substances tested. National Environmental Engineering Research Institute (NEERI) Nagpur, India had taken up a project in 1961. Materials like anion exchange resins, carbion of defluoron-1,defluoron-2, activated
Magnesia, activated alumina, serpentine minerals and Nalgonda technique were used for defluoridation. Depending upon the treatment cost and capital cost per m³ of water defluoridated, Nalgonda technique has been adopted all over the country for the removal of excess fluoride from drinking water (Bulusu, 1983).

**NALGONDA TECHNIQUE**

This technique involves addition of aluminium salts, lime and bleaching power. It is a combination of several unit operations and processes incorporating rapid mixing, chemical interaction, flocculation, sedimentation, filtration, disinfection and sludge concentration to recover water and aluminium salts such as aluminium sulphate and aluminium chloride or in combination since they are responsible for removal of fluoride from water. The selection of either aluminium sulphate or aluminium chloride also depends on sulphate and chloride contents of the raw water to avoid exceeding their permissible limits. The dose of lime is empirically 1/20th that of the dose of aluminium salts. Lime facilitates forming denser flocs for rapid settling. Bleaching powder is added to the raw water at the rate of 3mg/L for disinfection. Approximate doses of alum required to obtain permissive limit (1mgF⁻/L) in water at various fluoride and alkalinity levels are given in the following table (NEFRI, 1992).
APPROXIMATE ALUM DOSE (Mg/L) REQUIRED TO OBTAIN PERMISSIBLE LEVEL (1mg F/L) OF FLUORIDE IN WATER AT VARIOUS ALKALINITY AND FLUORIDE LEVELS

<table>
<thead>
<tr>
<th>Test water fluorides mg F⁻/L</th>
<th>Test Water Alkalinity, mg CaCO₃/L</th>
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<tr>
<td></td>
<td>125</td>
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<td>2</td>
<td>145</td>
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<td>3</td>
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* To be treated after increasing the alkalinity with lime or Sodium Carbonate.

Rapid Mixing:

It is a unit operation which provides uniform mixing of alkali, aluminium salts and bleaching powder with water. The chemicals are added just when the water enters the system.

Flocculation:

Flocculators are provided for subsequent gentle agitation before entry to the

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sedimentation tank. The flocculation period permits close contact between fluoride and polyalumernic species formed in the system. The interaction between fluoride and aluminium species attains equilibrium.

The chemical reaction involving fluorides and aluminium salts is complex. It is a combination of polyhydroxy aluminium complex formation with fluorides and their adsorption on polymeric alumina hydroxides (Floc). Besides fluorides, turbidity, colour, odour, pesticides and organics are also removed. The bacterial load is also reduced significantly. All these phenomena take place by adsorption on the flocs.

Sodium carbonate and sodium bicarbonate ensures adequate alkalinity for effective hydrolysis of aluminium salts, so that aluminium residuals do not remain in treated water. The reactions are depending upon the nature of alkalinity. Chemical reactions involved in this process are as follows:

1. \[ 3\text{Al}_2(\text{SO}_4)_3\cdot 18\text{H}_2\text{O} + \text{NaF} + 17\text{NaHCO}_3 \rightarrow [5\text{Al(OH)}_3\cdot \text{Al(OH)}_2\text{F}] + 9\text{Na}_2\text{SO}_4 + 17\text{CO}_2 + 18\text{H}_2\text{O} \]

2. \[ 3\text{Al}_2(\text{SO}_4)_3\cdot 18\text{H}_2\text{O} + \text{NaF} + 9\text{NaHCO}_3 \rightarrow [5\text{Al(OH)}_3\cdot \text{Al(OH)}_2\text{F}] + 9\text{Na}_2\text{SO}_4 + \text{NaHCO}_3 + 8\text{CO}_2 + 45\text{H}_2\text{O} \]

Sedimentation:

It permits settleable floc loaded with fluorides, turbidity, bacteria and other impurities to be deposited and thus reduces concentration of suspended solids that must be removed by filters. The floc is not uniform and hence its basic sedimentation properties cannot be given quantitative values and because the influence of eddy currents cannot be predicted. Hence, various factors which influence sedimentation in relation to design and operation rely largely on experience.
Filtration:

Rapid gravity sand filters are suggested to receive coagulated and settled water in those filters and unsettled gelatinous floc is retained. Residual fluorides and bacteria are absorbed on the gelatinous floc retained on filter bed.

Disinfection and distribution:

The filtered water collected in the storage tank is rechlorinated with bleaching powder before distribution.

MASS-BALANCE RELATION AND NALGONDA TECHNIQUE

Mass-balance is the fundamental approach used to delineate the changes that take place, when reaction is occurring in a container or in some definable portion of the container, it will be assumed that

1. Volumetric flow rate into and out of the container is constant.
2. The liquid within the reactor is not subject to evaporation (Isothermal conditions).
3. The liquid within the container is mixed completely.
4. A chemical reaction involving the reactant must be occurring within the reactor only.

A mass-balance of any process is an exact accounting of all the materials that enter, leave, accumulate or are depleted in the course of a given time interval of operation.

Nawlakhe (1978) has carried out an experimental study on mass-balance relationship in Nalgonda technique of defluoridation of water. The results revealed that fluoride is removed along with the sludge agglomerated and settled at the

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bottom. When this removed fluoride is added to fluoride concentration in treated water, the sum is almost equal to the fluoride content in test water. Hence, a proper balance was established in the treated water and sludge settled at the bottom. The percentage deviation of the sum from the test water fluoride content ranges from +2.9 to -6.9 indicating that the fluoride balance was established. These experiments were done in the test water prepared by adjusting the composition of stock tap water with distilled water, sodium fluoride and sodium bicarbonate in a plastic beaker. But, material balance studies in a working defluoridation plant has hitherto not been carried out. Hence, these studies were undertaken further to develop a proper defluoridation technique which could help in providing safe drinking water.

The role of fluoride on some soft tissue functions in fluorotic individuals of fluoride endemic regions of North Gujarat are also limited. Therefore, to fill up these lacunae, the present investigations have been undertaken. The work incorporated investigates the effects of fluoride on haematology, some specific parameters in liver, muscle and kidney as well as electrolyte balance in experimental mice. The parameters studied in fluorotic human population have a direct or indirect bearing on the soft tissue functions.