A BRIEF REVIEW ON FLUORINE CONCENTRATION AND ITS ADVERSE EFFECT

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ABSTRACT

Fluorine is a univalent poisonous gaseous halogen that can be present in trace amounts in air, water, livestock, and plants. Fluorine is needed for bone solidification and maintenance, as well as the prevention of dental deterioration. When fluoride is found in low concentrations in drinking water, it benefits bones and teeth; however, prolonged contact to fluoride in drinking water, alone or in conjunction with fluoride from different sources, may cause osteoporosis, teeth loss, and damage to the bones, muscles, kidney, nerves, and reproductive organs. F has a higher toxicity in diabetic patients as an endocrine disruptor. As a result, in order to determine if citizens need fluoride supplementation or not, the government can take steps to avoid health issues caused by fluoride loss or over consumption. In order to enlighten better practises in the usage of fluoride-containing products for the protection and welfare of public health, further rigorous analysis centered on empirical findings is needed.

Keywords: Fluorine, Fluoride, Fluorosis, Oral health, Water contamination.

I. INTRODUCTION

Fluorine is a poisonous gaseous halogen with a pale yellow-green colour and is the strongest chemical reactive element as compared to other elements [1]. Fluorine is typically present as fluoride in aqueous solutions (F). Fluorine is the lightest component of the halogen group and the world's 13th most plentiful substance, accounting for 0.08 percent of the earth's crust. Fluorine levels in the soil are about 330 ppm. Fluorine is found naturally in air, water, livestock, and plants in small amounts. As a consequence, fluorine is used in food, water for drinking, and in air. Fluorine is crucial for the conservation and protection of bones, as well as the prevention of dental caries. If it is ingested very much, though, it can have the opposite effect, triggering osteoporosis, teeth loss, and damage to the bone, kidney, muscle, and nerve system. The optimum fluoride level in drinking water, as per the WHO Guidelines for the quality of Drinking Water, is 1.5 mg/L. Fluorine, as the most electronegative component, has a strong propensity to develop a -ve charge and forms FTM ions in solution. the charge on fluoride ion is same as that of hydroxide ions and have about the same radius, but they may override each other in mineral systems [2].

Fluoride is one of the few chemicals which has been shown to have major impacts on the population in a variety of forms, including: climate, drinking water, meats, oral products, salts, and drinks are all examples of natural resources. When fluoride is found in low concentrations in drinking water, it benefits teeth and bones, however prolonged contact to F in drinking water, either or in conjunction with different sources, may cause a variety of problems.

Fluorosis is caused by a combination of factors like fluoride content in drinking water, exposure, normal consumption, and climatic conditions. Fluorosis affects an estimated 62 million people in India, including six million infants, as a result of drinking fluoride-contaminated water. If the degree and duration of exposure raises, the negative consequences vary from moderate oral diseases to crippling skeletal fluorosis. In certain parts of the country, crippling skeletal fluorosis is a major cause of morbidity. The estimated regular dietary consumption of fluoride by children in fluoridated (1 ppm) populations is 0.05 mg/kg daily, according to many studies; estimated intakes for children in communities lacking optimally fluoridated water was around 50percent smaller[3]. Fluorosis impacts about one out of every four Americans between the ages of 6 and 49. It is most common among children
between the ages of 12 and 15. The improper usage of fluoride-containing oral materials like mouth wash and toothpastes [4] is a significant source of fluorosis.

II. SOURCES OF FLUORINE

Fluorine is found in variable amounts in drinking water, rocks, sea water, mineral deposits of villiaumite (NF), sallaité (MgF2), cryolite (Na3 Al F6), fluorspar (CaF2), fluoroapatite [Ca5(P04)3 F], bastnaesite (CeLaY) (CO3)F, etc., with concentrations (percent) of 55, 61, 45, 49, 3.5, and 9, respectively and dust. In unpolluted environments, fluoride concentrations in the atmosphere usually range between 0.02 to 2.0 prg/m3 (USEPA 1980). Fluoride in the atmosphere may be particulate or gaseous. Sulphur hexafluoride (SF6), hydrogen fluoride (HF), hexafluorosilicic acid (H2SiF6), silicon tetrafluoride (SiF4), and CF4 are the most popular gaseous types. Particulate sources of calcium phosphate fluoride (CaF04P), sodium aluminium fluoride (NaAlF3), aluminium fluoride (AlF3), sodium hexafluorosilicate (F6Na2Si), lead fluoride include sodium hexafluorosilicate (PbF2), calcium phosphate fluoride (CaF2), sodium aluminium fluoride (NaAlF3), and calcium fluoride (CaF2). Inorganic fluorides such as inorganic and hydrogen fluoride particulates (calcium and sodium fluoride) make up the majority of inorganic fluoride in the environment, accounting for about 75% and 25%, respectively [5].

Drinking water, plants raised on fluorotic soils and water, many edible aquatic organisms, mineral mixture, fluoride heavy phosphorus feed supplements, cosmetics, medications, dust in the environment, and certain agricultural activities are the main sources of fluoride for humans and animals [6,7]. In India, however, recurrent fluoride contamination or toxicity in humans and domestic animals is mostly caused by consuming groundwater. Bore well water has a significantly higher fluoride concentration than surface water in certain places [8].

Chronic fluoride toxicity is widespread in many Indian states due to high fluoride levels in drinking water supplies, particularly in rural regions where this health issue is more prominent [9,10]. Fluoride poisoning affects 50–100 percent of districts in Gujarat, Andhra Pradesh, Uttar Pradesh, and Rajasthan, among other states in India. Fluoride poisoning is endemic in 30–50 percent of districts in Haryana, Bihar, Madhya Pradesh, Tamil Nadu, Karnataka, Punjab, and Maharashtra [11].

Industrialization is crucial for the development of the economy and long-term stability. However, India’s rapid industrial development is causing a bunch of health issues, not just for humans but also for domestic animals residing in vicinity of many fluoride emission factories. Welding processes, coal-burning power plants, and the manufacture or development of iron! steel, zinc, aluminium, chemical fertilizers, hydrofluoric acid, arsenic, glass, bricks, cement, and rubber are examples of actions which emit fluoride into the atmosphere of all particulate and gaseous types [5,6]. The released synthetic fluoride eventually settles on the land and in the vegetation/herbage. Animal fluorosis is the main risk of ingesting fluoride-contaminated herbage over an extended period of time. Skeletal, dental, and non-skeletal defects or modifications are caused by prolonged inhalation or reaction to fluoride from such a toxic or infected atmosphere in factory workers/employees, as well as people residing around such factories [5].

It presents naturally in the rocks, especially in the presence of phosphate. Surface water leaching from rocks and soils extracted from rocks can contain rich amounts of fluoride [12]. Chronic fluoride toxicity in animals has been linked to the usage of rock phosphate and nutrient quality phosphorous supplements (mono- and di-ammoniumphosphate) [13]. And in the absence of abnormal fluoride toxicity, adult human and animal bones had higher fluoride levels. As a result, bone meals may be a major reservoir of fluoride for livestock [8]

III. BENEFICIAL EFFECT OF DENTAL HEALTH FLUORIDE UPON –

Tooth decay is also the world’s most prevalent chronic childhood condition. Fluoride has been shown to be beneficial in preventing caries and offering optimum defence against dental caries thus reducing the risk of fluorosis in the enamel. Regular fluoride penetration during tooth growth provides long-term defence towards dental decay and enamel fluorosis (Table 1). Enamel is a carbonate-richhydroxyapatite, calcium-deficient. There is enough PO43−, Ca2+, F− ion, and OH in the immediate vicinity of crystals to preserve equilibrium with ambient fluid in its steady state. Plaque bacteria produce organic acids from carbohydrate during cariogenic acid assault, which dissociate and release H+ ions, lowering the pH around the tooth. The H+ ions in plaque fluid protonate phosphate ions (PO43−) present in HPO42−, especially H2PO4−. This mechanism still preserves neutrality and eventually contributes to calcium release from the hard tooth material [14]. Free fluoride ions in solution around enamel crystals and tooth are thought to perform an effective role in preventing caries than fluoride presented in the enamel, according to the theory. This results in a state of super saturation or equilibrium in relation to fluoro-hydroxyapatite,
and thus mineral reprecipitation. Furthermore, fluoride adsorption on the crystal provides defense against de-mineralization. If fluoride is not present for a long period, enamel crystal may be destroyed during an acid attack. Since the F level of saliva substantially rises for around 30 min after eating foods comprising fluoridated table salt, these low fluoride levels may also be achieved [15]. Because the development of CaF2 at these low concentrations is impossible, it can be assumed that table salt and fluoridated drinking water work in the same way. Fluoride has antimicrobial properties as well. Fluoride has been shown to impair the carbohydrate synthesis of oral lactobacilli and streptococci in the laboratory [16]. The process of glucose transfer through cell may be hampered by excessive cytoplasmic acidification [17].

1. Industrial and neighborhood fluorosis

Fluoride exposure by water, food, dirt, and air over an extended period of time produces a variety of negative health or harmful effects in mammals, especially in domestic animals and humans, in the form of fluorosis. If these symptoms are caused by consuming fluoridated water, they are attributed to as hydro-fluorosis, this is a normal occurrence that is more widely spread in nature. Industrial and neighborhood fluorosis, on the other hand, are less common, anthropogenic, limited to a certain place, and exacerbated by long-term exposure to fluoride released from various industrial activities. The word industrial fluorosis is commonly used and limited to domestic animals and industrial workers, while neighborhood fluorosis is used to describe domestic animals and humans who live near fluoride emission factories. On the other hand, Hydro-fluorosis is prevalent in almost every state in the world [10].

Bartolucci (1912) [23] identified fluorosis in cattle on a farm near a super-phosphate factory in Italy for the first time in the world. He first diagnosed it as osteitis, blaming it on fluorine from the factory. In a 1927 paper [24], he first used the word fluorosis as a synonym for cachexiefluorique, as Christiani&Gautier (1925) [25] had done earlier. Rao and Pal (1978) [26] in India were the first to consider industrial fluorosis in cattle residing near an aluminium factory in the state of Orissa. The issue of lameness in farm animals was verified by pathological and chemical analysis of the infected animals' bones. Fluorine levels were observed to be very high, ranging from 1.0 to 2.7 percent on the bone ash, relative to typical levels of 0.05 to 0.08 percent. The amount of fluoride in urine mirrored existing fluoride ingestion and was heavily correlated with the duration of exposure or gross fluoride ingested. The infected cattle were found to have high levels of fluorine in their urine, up to 68 ppm, relative to less than 5 ppm in the urine of cattle from different regions. Fluoride levels in the urine were large, indicating industrial fluorosis [27].
Moller and Gudjonsson (1932) [28] were the first to identify persistent industrial fluoride intoxication in humans in Danish cryolite staff. Since Roholm (1937)[29] identified and thoroughly investigated many cases of osteo-dental fluorosis caused by inhaling fluoride dusts, industrial fluorosis was documented in humans [30] and domestic animals [31] from various countries. However, research on occupational fluoride toxicity or community fluorosis of people residing around different factories is still scarce [32,33]. However, on the west coast of India, north of Bombay, a survey report on neighbourhood fluorosis. His survey included 7059 individuals of 3 urban and 15 villages areas within 3.5km area from the fluoride-processing company (productionindustry of Freon gas ‘a refrigeration gas’) [34].

2. Fluoride inflicted a variety of health problems

Chronic fluoride exposure, whether from fluoride emitted from industry or fluoridated drinking water, causes accumulation of fluoride in hard tissues like bones and teeth, resulting in a variety of adverse effects in man, including dental mottling (dental fluorosis) and bone deformities (skeletal fluorosis)[10]. Non-skeletal fluorosis or harmful effects of prolonged fluoride penetration in organs or soft tissues, such as neurological abnormalities, gastrointestinal discomforts, compromised reproductive and endocrine functions, renal effects, excitotoxicity,

<table>
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<tr>
<th>Fluoride concentration</th>
<th>Exposure</th>
<th>Physiological effects</th>
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<tbody>
<tr>
<td>Greater than 1 ppm</td>
<td>Chronic</td>
<td>Impaired glucose metabolism in both animals and humans [40].</td>
</tr>
<tr>
<td>Greater than 4 ppm</td>
<td>Chronic</td>
<td>Skeletal fluorosis, dental fluorosis hypomineralization of tooth enamel [35].</td>
</tr>
<tr>
<td>Sub-acute exposure to fluoride at a dose of 20 ppm/day (orally to rat)</td>
<td>Chronic</td>
<td>Induces thyroidal dysfunction including suppressed synthetic machinery of the thyroid gland to produce thyroid hormones, mainly T3 and T4. Alteration of Na^+\cdotK^+\cdotATPase, thyroid peroxide, and 5,5'-deiodinase [18].</td>
</tr>
<tr>
<td>100-200 ppm</td>
<td>Chronic</td>
<td>Changes on the thyroid hormone status, the histopathology of discrete memory abilities in multi-generation rates. [19].</td>
</tr>
<tr>
<td>High fluoride</td>
<td>Chronic</td>
<td>Increases risk of Alzheimer’s disease [20].</td>
</tr>
<tr>
<td>High fluoride</td>
<td>Chronic</td>
<td>Increased testosterone level of FSH and LH . Decreased estrogen levels. Decreased testosterone levels and changes in its potent metabolites. Reduced thyroid hormones.[21]</td>
</tr>
<tr>
<td>1,000-1,500 ppm (in toothpastes)</td>
<td>Chronic</td>
<td>Prevent caries in permanent teeth [22]</td>
</tr>
<tr>
<td>Greater than 1,000-1,500 ppm (toothpastes)</td>
<td>Chronic</td>
<td>Enamel fluorosis of the front permanent incisors [35].</td>
</tr>
<tr>
<td>Regular exposure during teeth development</td>
<td>Chronic</td>
<td>Protection against enamel fluorosis in adult age also [14].</td>
</tr>
<tr>
<td>1.5mg/L or above (through water)</td>
<td>Daily intake</td>
<td>Produces insulin resistance.</td>
</tr>
<tr>
<td>0.05 0.07 mg/kg body weight (should not exceed from all sources)</td>
<td>Daily</td>
<td>In order to minimize the risk of dental association 2012).</td>
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</table>
teratogenic effects, apoptosis, and genotoxic effects, and so on, have all been identified in humans and laboratory and domestic animals. These results have recently been clinically checked [11].

3. Fluoride Impact on Dental & Skeletal Health –

Fluorosis is a long-term disease induced by an accumulation of fluorine compounds in the diet, characterised by tooth mottling and, in extreme cases, ligament calcification. There are two forms of fluorosis: dental and skeletal fluorosis. Fluoride poisoning has been linked to bone deterioration and an uptick in hip and wrist fractures. Fractures are often correlated with fluoride amounts of 1-4 ppm, according to the National Research Council of the United States. Skeletal fluorosis is caused by long-term use of fluoride at amounts higher than this in fluoridated water. Skeletal fluorosis is common in some places, especially the Asian subcontinent. Early stages are also misdiagnosed as rheumatoid arthritis or ankylosing spondylitis since they are not clinically evident (National Research Council 2006). Too much fluoride during enamel development causes dental fluorosis [35], which is characterized by hypomineralization of tooth enamel. It manifests itself as a variety of visual improvements in enamel as a function of varying degrees of inherent tooth discoloration. The magnitude of the disorder is determined by the individual's dosage, size, and age [36]. There are slight white lines or specks in the mildest (most common) type. White mottled patches occur in somewhat more extreme circumstances, while severe fluorosis is marked by brown discolouration and rusty, pitted, and rugged enamel.

Tooth enamel hypomineralization is exacerbated by the absorption of so much fluoride during enamel creation. An accumulation of fluoride ions in the extracellular atmosphere of maturing enamel alters the pace at which amelogenin are chemically segmented and the resulting breakdown products are extracted. Fluoride can also affect protease activity indirectly by reducing the supply of free calcium ions in mineralization situation [37,38]. Other side effects have been documented, including nephrosis, increased hepatic cell size, testis seminiferous tubule degeneration, and myocardial mineralization [39]. Since the bulk of fluoride is excreted by the kidneys [40].

4. Non-skeletal fluorosis or a variety of undesirable changes in soft tissues –

In fluoride-endemic regions, the most prevalent health problems include polyuria/itching in the region, gastrointestinal discomforts (gas formation or bloating, nausea/loss of appetite, constipation, pain in the stomach/colic, headache, intermittent diarrhoea, and so on), extreme weakness/muscle weakness, excessive thirst (polydipsia), bronchitis, asthma, and allergic reactions[41]. In a survey of 462 workers of the aluminium industry in the northeastern part of U.P., many of them showed non-skeletal symptoms, indicating that they are suffering from industrial fluorosis [42]. Following the termination of fluoride exposure, the above-mentioned health complaints typically vanish within 7–10 days. These complaints are important since they could be the first symptom of fluoride toxicity [42]. However, these health issues may be caused by a variety of factors. However, the appearance of skeletal and dental modifications, in addition to the estimate of fluoride concentration in blood serum and urine, are more relevant and extremely essential for the evidence of industrial fluoride toxicity.

5. Toxicity in Aquatic Plants & Algae –

Depending on fluoride levels, algal organisms, and exposure length, fluoride may either impede or stimulate algae population development (Table 2). Some algae can withstand inorganic fluoride concentration of up to 200 mg F/l. Fluoride's hazardous effect on algal development may be explained by the concept that fluoride ions may alter nucleic acid and nucleotide metabolism, which governs algal cell division processes[43]. Development stimulus of some algal organisms, on the other hand, may be attributed to a fluorine prerequisite for optimum growth [44]. Under field and laboratory settings, aquatic organisms seem to be good at extracting fluoride from tainted water. With rising fluoride accumulation and exposure period, the fluoride content of aquatic macrophytes rises [45].

In laboratory studies, [46] discovered that low concentration of sodium fluoride induced oxygen intake & overall phosphorylated nucleotides in the respiration of *Chlorella pyrenoidosa*. In comparison, Holzer (1954) [47] found that 570 mg F/l decreased the respiration rate in this algal genus by 50 percent, and in another survey 1900 mg F/l inhibited respiration in *C. pyrenoidosa* by 26 percent. Following laboratory experimentation with this freshwater algal group, Smith and Woodson (1965) [48] discovered that a fluoride level of 190 mg F/l suppressed the rate of growth of *C. pyrenoidosa* by 58–82 percent after 72 hours of treatment.

Dunaliellatertiolecta, Prasinocladosmarimus, Rhodomonas lens, Chroomonas salina, Chaetocerosgracilis, Bellerochea polymorpha, and Thalassiosiraweissflogii) to fluoride levels for 25 days. The levels ranged from 0 to 100 mg F/l.

They discovered that while fluoride levels of 25, 50, and 100 mg F/l increased population of the cryptomonad R. lens by 20–30 percent, fluoride conc. of 100 mg F/l inhibited the rates of growth of the diatom N. angularis var and two others by 25 to 30 percent. This inhibition of algal development was not followed by any fatal cell lysis signals. According to a survey [44], the growth stimulus in R. lens may be attributed to a fluorine necessity for optimum microalgae growth. The development of different 8 test algal species was normal, with no evidence of major (inhibition or enhancement) results.

<table>
<thead>
<tr>
<th>Table 2 Effect of fluoride on population growth of freshwater and marine algae [43,44,52]</th>
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<tbody>
<tr>
<td>Species</td>
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<tr>
<td>--------------------------------------</td>
</tr>
<tr>
<td>Chlorella pyrenoidosa</td>
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<tr>
<td>Selenasirum capricornumum</td>
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<tr>
<td>Ankistrodesmus brunnii</td>
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<tr>
<td>Cyclotella meneghiniana</td>
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<tr>
<td>Oscillatoria limnetica</td>
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<tr>
<td>Scenedesmus quadricula</td>
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<tr>
<td>Stephanodiscus minutus</td>
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<tr>
<td>Synecococcus leopoliensis</td>
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<tr>
<td>Nitzschia palea</td>
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<tr>
<td>Chlorella vulgaris</td>
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<tr>
<td>Amphora coffeaeformis</td>
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<tr>
<td>Agmenellum quadruplicatum</td>
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<tr>
<td>Amphidinium carteri</td>
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<tr>
<td>Dunaliella tertiolecta</td>
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<tr>
<td>Nannocharis oculata</td>
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<tr>
<td>Pavlova lutheri</td>
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<tr>
<td>Prasinoclados marimus</td>
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<tr>
<td>Chroomonas salina</td>
</tr>
<tr>
<td>Rhodomonas lens</td>
</tr>
<tr>
<td>Bellerochea polymorpha</td>
</tr>
<tr>
<td>Chaetoceros gareilis</td>
</tr>
<tr>
<td>Nitzschia angularis var. affinis</td>
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<tr>
<td>Thalassiosira weissflogii</td>
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6. Fluoride Toxicity in Fishes –

Fluoride toxicity in fish rises as the concentration of fluoride in aquatic medium, water temperature, and exposure period rise [49]. Fluoride toxicity declines with rising chloride and calcium and content and intraspecific fish size in the water [50]. Until death, lethal concentration of fluoride trigger fluorosis symptoms in fish [49]; initial lethargy and apathy accompanied by reduced respiratory rates, anorexia hypoeicitation, dorsal side (a darkening of the skin), increased mucus secretion, and increased fluoride level in blood. Furthermore, high fluoride levels may trigger a lag in the hatching of fertilised freshwater fish eggs [51].

IV. CONCLUSION

Fluoride is among the most useful micronutrients for us, but too much of it will affect in a variety of ways. Complete daily fluoride intake will be approximately 0.6 mg/day for adults in an area where no fluoride is applied to the drinking water and 2 mg/day for adults in a fluoridated setting, according to WHO recommendations. People must be cautious when drinking fluoridated water, meats, salt, or utilising heavily fluoridated tooth paste in order to reap the benefits of fluoride. Before the latest guideline is introduced, low fluoride (less than 600 ppm) comprising paediatric toothpastes should only be used by children rather than fluoride toothpaste comprising 1,000 to 1,500 ppm fluoride. Fluoride concentrations in various drinking water sources and ground water should be monitored by the government, and a comprehensive fluoride chart should be made accessible to the public. To determine if citizens need fluoride supplementation, the government can publish the WHO recommendations in a circular form to avoid health complications caused by fluoride deficiency or over exposure. The recommendations shall provide an evidence-based review of existing findings and information in order to illuminate best practises through the usage of fluoride-containing products for public health care and security.

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