Susheela, J Oral Hyg Health 2021, 9:7

Journal of Oral Hygiene & Health

Short Communication Open Access

Why Fluoride Afflicts Tooth and Bone Preferentially Resulting in Dental and Skeletal Fluorosis Respectively?

Andezhath Kumaran Susheela*

Department of Medical Science, All India Institute of Medical Sciences, New Delhi, India

***Corresponding author:** Dr A.K. Susheela, Former Professor of All India Institute of Medical Sciences New Delhi India, E-mail: FRnRDF.aks@gmail.com

Received date: August 03, 2021; **Accepted date:** August 17, 2021; **Published date:** August 24, 2021

Citation: Susheela AK (2021) Why Fluoride Afflicts Tooth and Bone Preferentially Resulting in Dental and Skeletal Fluorosis Respectively? J Oral Hyg Health 9: 293

Introduction

Dental and Skeletal Fluorosis are known to occur since many decades in India and other nations. The two disorders are described in detail in publications since early 1930s. But the reasons for the tooth and bone to be attracted by Fluoride are not dealt with. The immediate response is that both tissues are rich in positively charged cations. It may be due to the anion-cation reaction. However, there are other cation rich tissues in the body, which does not get afflicted with Fluoride toxicity along with tooth and bone. Therefore that possibility is ruled out. What could be the other reason(s)?

In an adult human body, there are 32 teeth in the oral cavity and 200 bones irrespective of the gender. The disease nomenclature denotes F^{-} preferentially afflicted tooth and bone. Those who address the disease(s) should have clarity of thought and reason(s) for the affliction.

As there are commonalities in the matrices of the 2 tissues, studies with focus on GAGs and glycoprotein (sialic acid) have been investigated in greater detail to understand their role in mineralization, hypo-mineralization or demineralization. The status of sulphated isomers of GAGs in human fluorosed teeth was probed into, which provided a wealth of information. The enamel has negligible GAGs; and the GAG extracted was mainly from dentin. Control from endemic and non-endemic areas were introduced to evaluate the results of the fluorosed victims.

Matrices of tooth and bone

To understand the primary reason for the affliction, the chemical configuration of the matrices of the 2 mineralized tissues were examined. The tooth and bone matrices are predominantly constituted of collagen protein. Two other constituents of the matrix which play a role in the pathogenesis of the two tissues are:

- Glycosaminoglycans and
- Glycoproteins (sialic acid)

Glycosaminoglycans occur in the matrices as sulphated and non-sulphated isomers. Sulphated isomers of GAGs play a prominent role in mineralization as well as hypo mineralization in pathogenesis.

It was the observation in human tooth samples, total GAG disaccharides are constituted of chondroitin-4-sulphate, Dermatan sulphate and chondroitin-6-sulphate. The Dermatan Sulphate content unlike chontroitin-4-sulphate and chondroitin-6-sulphates were significantly enhanced in fluorosed teeth compared to controls collected from endemic and non-endemic areas [1,2]. The presence of Dermatan Sulphate in fluorosed rat tooth and rabbit bones has been reported earlier by [3-5]. Our studies have clearly shown that overtly visible Dental Fluorosis was evident only in those subjects with high Dermatan Sulphate content. There was a significant increase in Dermatan Sulphate content and that may be the detrimental factor in the development of Dental Fluorosis [6].

In Dental Fluorosis, the disease may occur as:

- The teeth in pairs based on development, had white opacities and faint yellow horizontally aligned lines.
- The teeth in pairs had brown stains, spot or lines.
- The teeth may get pitted, perforated and chipped off edges were signs of hypo mineralization/demineralization.
- In Skeletal Fluorosis, the disease may occur as:
- The victims appear asymptomatic but radiographs revealed increase in bone density.
- The victims are symptomatic with aches and pain in major joints, vertebral column and difficulty in walking.

Severe form: The victims are symptomatic with involvement of restricted movements of spine, neck and joints with rigidity, crippling deformity and fracture of bones.

In Skeletal Fluorosis in human and toxicity induced animal models were investigated with a different approach. One of the earliest manifestations of fluoride toxicity is in and around the Osteoblast or an Osteocyte and can be detected from the accumulation of GAG [7]. As GAGs formation is in and around Osteoblasts and Osteocytes, it is unlikely a needle biopsy from patients may not be helpful. Therefore, blood samples were investigated for GAGs and the Glycoprotein (sialic acid). It was found that GAGs enhanced significantly in blood samples both in human and animal models upon fluoride ingestion/ fluorosed victims. However, sialic acid was reduced [2].

Characterization of GAGs in cancellous bone from iliac crest region of the animal model was carried out. The GAGs extracted from fluoride treated animals were double the quantum compared to controls. Chontroitin-4-sulphate and Chontroitin-6-sulphate isomers were present in sulphated GAGs in sample and controls. However, Dermatan sulphate (Chondroitin sulphate B) appeared in sample animals but not detected in control animals.

The observations in bone and tooth in human and animal models exposed to Fluoride are similar in the changes taking place in the configuration of the matices. Dermatan sulphate in cancellous bone has also been localized with use of Alcian Blue/Ruthenium red dyes revealing chondrocytes in the cartilaginous lesions developed in cancellous bone (tralecular bone) which is “neo-bone” formation.

The observation was that Dermatan sulphate when present, the bone and tooth were demineralized/unmineralized [8]. Bio-chemical characterization of GAGs in Fluoride exposed leads to Dermatan

sulphate formation which under normal circumstances do not occur in tooth or bone. This primarily may be the impetus for Fluorosis development, preferentially compared to other soft tissues, viz. ligament, skin, muscles and the aorta.

This is also to report the soft tissues which are not destined to be mineralized have high concentration of Dermatan sulphate content. Upon exposure to fluoride Dermatan sulphate begin to disappear and the tissue get mineralized (ectopic calcification) [9]. In Fluorosis the ligaments are calcified and seen in radiographs is a classic example.

Conclusion

The dermatan sulphate content, its absence in normal mineralized tissues and presence after exposure to fluoride, results in demineralization. The reverse events occur in soft tissues. The high dermatan sulphate content in soft tissues tends to disappear upon exposure to fluoride and mineralization sets in. Dermatan sulphate, therefore appear to be the crux of the problem of Fluorosis/Fluoride toxicity leading to demineralization in bone and tooth; and mineralization in soft tissues.

References

1. Susheela AK (2007) A treatise on fluorosis. Fluorosis Research and Rural Development Foundation. 34: 181-183.
2. Jha M, Susheela AK, Krishna N, Rajyalakshmi K, Venkiah K, et al. (1982) Excessive ingestion of fluoride and the significance of sialic acid: Glycosaminoglycans in the serum of rabbit and human subjects. J Toxicol Clin Toxicol 19: 1023-1030.
3. Embry G, Stanbury J, Smalloy JW (1979) The metabolism of proteoglycans and glycosaminoglycans in inflamed human gingiva. J Periodontal Res 14: 165-167.
4. Jha M, Susheela AK (1982) Characterization of glycosaminoglycans from normal and fluoride treated rabbit iliac crest. Biochem Biophys Res Commun 105: 711-6.
5. Susheela AK, Jha M (1983) Cellular and histochemical characteristics of osteoid formed in experimental fluoride poisoning Toxicology letters 16: 35-40.
6. Susheela AK, Sharma K, Rajan BP, Gnanasundaram N (1988) The status of sulphated isomers of glycosaminoglycans in fluorosed human teeth. Arch Oral Biol 33: 765-767.
7. Susheela AK, GS Toteja (2018) Prevention & control of fluorosis & linked disorders: Developments in the 21st Century-Reaching out to patients in the community & hospital settings for recovery. Ind J Med Res 148: 539-547.
8. Susheela AK, Sharma K (1988) Fluoride-induced changes in the tooth glycosaminoglycans: An in vivo study in the rabbit. Arch Toxicol 62: 328-330.
9. Susheela AK (1994) Studies on some aspects of Fluorosis. Recent Trends in Nutrition 143-157.