Available online at www.worldscientificnews.com

World Scientific News

5 (2014) 12-16 EISSN 2392-2192

Effect of fluoride in the human body

G. Sivasankaran Kair*, C. Ravin Samuel Faj

Department of Chemistry, Manonmaniam Sundaranar University,
Tirunelveli - 627 012, Tamil Nadu, India

*E-mail address: gsivchem@rediffmail.com

ABSTRACT

Fluoride has a specific affinity for hydroxyapatite bone $Ca_{10}(PO_4)_6(OH)_2$. It lists the hydroxyl ions of hydroxyapatite crystals converting it into Fluor apatite, which entails a change of the physical characteristics of the crystals. Fluorapatite crystals are quick dimension "a" crystal, increased crystallinity and a reduced dissolve. Increased solubility of fluorapatite is the main cause of changes in the system fluoro osteoarthritis

***Keywords*:** Fluoride; Human body; $Ca_{10}(PO_4)_6(OH)_2$; Effect of fluoride

1. INTRODUCTION

Effects of fluoride

Effects of fluoride on the human body may be, depending on dose, both beneficial and detrimental. Fluoride is used in the prevention of dental caries (0.5 1 2.0 mg / day) and osteoporosis (20 1 50 mg / day), on the other hand, millions populations are exposed to its toxic effects, as is the case in areas of endemic fluorosis a high fluorine content in the natural drinking water (2 1 80 mg / day) [5,8,16]. In industrialized countries, a major concern is the contamination of the environment and fluoride exposure of workers in processing plants and its compounds.

The biggest cause pollution: aluminum smelters, factories phosphate fertilizers, iron and glass, brick factories, mines cryolite. Chronic toxicity of fluorine compounds can lead to fluorosis industrial (8 1 80 mg / day) [13,16]. Due to their high activity fluoride is almost always in the form of compounds of a very strong hydrofluoric acid (HF), easily soluble fluorides such as sodium fluoride (NaF), or sparingly soluble calcium fluoride (CaF_2), and a number of complex compounds such as cryolite, apatites [19,21,24]. Hydrogen fluoride is corrosive to the mucous membranes and skin. It is very well absorbed from the gastrointestinal tract and respiratory tract (90% of dose) and quickly enters the bloodstream. Serum fluoride binds calcium and magnesium ions, leading in extreme cases up to immediate

death. In chronic poisoning inhibit the enzyme 72 magnesium-dependent, disrupts the Krebs cycle and the production of ATP. Above the osteoarthritis patients exposed to fluorine found pathological changes in the kidneys, central nervous system, thyroid, parathyroid [9,15,25].

Fluoride has a specific affinity for hydroxyapatite bone $Ca_{10}(PO_4)_6(OH)_2$. It lists the hydroxyl ions of hydroxyapatite crystals converting it into fluorapatite, which entails a change of the physical characteristics of the crystals. Fluorapatite crystals are quick dimension "a" crystal, increased crystallinity and a reduced dissolve. Increased solubility of fluorapatite is the main cause of changes in the system fluoro osteoarthritis [11,17,25]. Fluoride also acts on bone cells and enzymes involved in the ossification process. Stimulates osteoblast activity [17,18,22], and reduces the number and activity of osteoclasts [10.11]. Fluor according to some authors, inhibits the activity of alkaline phosphatase [10] and acid [17], but according to other reports, does not alter the activity of alkaline phosphatase and acid phosphatase activity increases [20].

Effect of fluoride is an imbalance between bone formation and destruction, with a prevalence of delayed formation of osteoid mineralization. Histomorphometry in fluorozie said increase of osteoblasts, increasing the density of cancellous bone trabeculae increasing the thickness and an increase in osteoid volume [1,18,23]. Typical characteristics for the image of bone under the influence of fluorine are macular osteocytarne bay, resulting from the delay of mineralization [7,8,14,16]. It has also been widening and increased cortical porosity and an increase in the activity of the periosteum [17,19]. Macroscopically, in the advanced stages of endemic fluorosis, trapped the endo- and periostealna apposition bone. Significant magnitude of change in the spine can cause spinal cord compression and paraplegia symptoms [88].

It is now clear that a significant fluorine content in the bone may even lead to fatigue fractures. Prolonged exposure to an excessive amount of fluoride can cause changes in the type [23,24]. Disorders in the construction of micro- and macroscopic skeletal manifest in radiological. For the typical changes fluorine adopt generalized bone disorders trabecular structure, reactions and ossification of muscle attachments. Although the impact of fluoride on bone tissue is exactly as described, it reports on changes occurring under the influence of fluorine in the joints are few, and their results are contradictory.

The diagnosis of skeletal fluorosis are changing. Their diagnosis is based on data on the exposure to fluoride, clinical, radiological and additional studies. Reported by many authors of many joints pain, restricted mobility, are not typical. In contrast, the relevant information is provided by radiographs. Usually performed radiographs of the spine, pelvis and long bones of the limbs [7,15,19]. Industrial fluorosis recognizing gave Roholm in his classic 1937 monograph. [14]. On the basis of cryolite mine workers, he described three stages of fluorosis. Stage I "Bone density is slightly increased. Bars are rough and they give a deep shadow. Bone mass is increased and obliterated structure. Demarcation of the normal structure is not clear in individual cases is difficult to decide whether the change is a variation of the structure of normal or pathological feature already. Although the screening and the difference is clear. "Stage II" bone structure is blurred and bar join. Often the bone gives diffuse shadow without structure. The changes are most pronounced in the pelvis and spine and ribs and the bones of the limbs, even if they are less pronounced and often resemble the changes described as stage I. limbs bone marrow cavity is typically slightly tapered.

The spine is the initial or small ossification of ligaments, especially localized caudal; they appear as a point or as galls with a tendency to build bridges between the vertebrae. In some cases (especially in younger patients) no ossification of ligaments, but the structure of the bone is altered so that the case must be qualified for the second stage. "Stage III" Bone has

spilled marble shade, where it is impossible to distinguish the details of the structure. These changes are reflected in all the bones, but they are still the most centrally, being the most suspicious of trabecular bone structure, pelvis, spine, ribs and sternum. In the mow-altitude limbs are changes in the structure corresponding to the second stage, and even a stage I.

The limb bones are irregularly thickened, some flat, others more harshly. Interosseous membrane is ossified to a greater or lesser extent, it is also ossification of attachment of the ligaments. Normally ossification and generalized bone sclerosis are simultaneous, although in some older workers bone sclerosis may not be the largest on the selected ligaments. The long bones of the medullary canal is narrowed, the tibia may even be narrowing in half. "Today, technological progress so limited exposure to fluoride workers that advanced stages of fluorosis by Roholma are rare.

Therefore it was necessary to develop additional preliminary stages. In previously published studies developed criteria for the two initial stages of fluorosis: Stadium O (suspicion of changes fluorine) Polyarticular pain and limited mobility in at most two organs-less movement. Initial radiological ossification in the image-NYM. OI stage (beginning with changes fluorine) Significant pain, a significant limitation of movement in at least two organs of movement, small reactions, the initial, slight thickening of the shafts of the long bones [14].

2. CONCLUSIONS

Based on the results of clinical trials, experimental, computer image analysis and morphometric measurements it is possible to formulate the following conclusions:

1. The developed image analysis software and Quantitrab Trabecula allow quantitative assessment of bone structure on the radiograph.

2. It was significant relationship between bone structure parameters on the radiograph obtained by quantitative analysis of radiological and histomorphometric features and content of the mine-rałów bone slices subject to radiological examination.

3. Typical changes in the structure of the fluorosis trabecular bone on the radiograph are: high trabecular width and their small number and the reduced density of the trabecular meshwork.

4. Typical changes in the structure of the fluorosis areas bezbeleczkowych bone radiograph (BZB) are increasing their number and reduce the surface.

5. The effect of fluorine on the bone cortical and cancellous dose-dependent. Chronic toxicity moderate doses leads to inhibition of bone resorption endostealnej cortical bone and trabecular bone growth.
Cortical indicators in patients with severe fluorosis are higher compared to those without fluorosis.

6. Fluorosis and degenerative changes occurred more frequently in people employed in areas with higher concentrations of fluoride. There is a change in the organ motion in subjects affected employees not only exposure to fluoride in the mill, but also contamination of the area of residence.

References

[1] Aaron J.E., de Vernejooul M.C., Kanis J.A.: The effect of sodium fluoride on trabecular architecture. *Bone* 1919, 12, 3071310.

[2] Agate J.N.: Industrial Fluorosis. Medical Research Council Memorandum. No. 22, London, His Majesty's Stationery Office, 1949.

[3] Andrejewa T.D., Girskaja J.: K rientgeno-diagnostikie kostnoj izmienij pri fluorozie u raboczich kriolitowych i aluminiewych zawodow. Wopr. Gig. Profess. Pat. Cwietnoj i Czernoj Metalurgii, pp. 52154, Swierdlowsk 1971.

[4] Atkins J.B.: Internal derangement of the knee joints in miners. *Brit. J. Ind. Med.* 1957, 14, 1211126.

[5] Austriaco N.R. Jr, Williams J.L., Drummond D.S.: Trabecular bone densitometry using interactive image analysis. J.Biomed.Eng. 1991, 13, 4861488.

[6] Barnett E., Nordin B.E.C.: The radiological diagnosis of osteoporosis 1 a new approach. *Clin. Radiol.* 1960, 11,166.

[7] Badurski J., Sawicki A., Boczoń S.: Osteoporoza. Z. Wenda & T. Trylski, Anin, Warszawa 1991.

[8] Baud C.A., Very J.M., Courvoisier B.: Biophysical study of bone mineral in biopsies of osteoporotic patients before and after long1term treatment with fluoride. Bone 1988,

[9] 9, 3611365. 9. Baud C.A., Very J.M., Courvoisier B.: Biophysical study of bone mineral in osteoporotic patients following fluoride therapy. XVIth Conference International Soc. Fluor.Res. Abstracts, str.33, Geneva 1987.

[10] B2lly M.: Experimental fluorosis in rats: NaF induced changes of bone and bone marrow. *Fluoride* 1983, 16, 1061111.

[11] B2lly M. i wsp.: Experimental fluorosis in rat: sodium fluoride induced changes of bone and cartilage. Metabolizm fluoru '88, str.41143. Pol. Tow. Biochem. Szczecin 1988.

[12] Bhustry B.R., Demole V., Jolly S.S., Singh A., Taves D.R.: Toxic effect of larger dosed of fluoride. w: Fluoride and Human Health, pp. 2251265, WHO, Geneva 1970.

[13] Białas B.: Wpływ wody pitnej skażonej fluorem na kościec szczura w różnych okresach jego życia. Praca habilitacyjna, Śląska Akademia Medyczna 1982.

[14] Compston J.E., Mellish R.W.E., Garrahan N.J.: Age-related changes in iliac crest trabecular microanatomic bone structure in men. Bone 1987, 8, 2891292.

[15] Croock H.V.: Spinal Surgery. Springer-Verlag, Wien, New York 1983.

[16] Dalen N., Lamke B.: Grading of osteoporosis by skeletal roentgenology and bone scanning. *Acta Radiol.* 1974, 15, 2, 1771186.

[17] Danielson L.G.: Incidence and prognosis of coxarthrosis. *Acta Orth. Scand.* 1964, suppl. 66.

[18] Davis M.A. i wsp.: Sex differences in osteoarthritis of the knee. *Am. J. Epidemiol.* 1988, 5, 101911030.

[19] Deas D.E., Pasquali L.A., Yuan C.H., Kornman K.S.: The relationship between probing attachment loss and computerised radiographic analysis in monitoring progression of periodontitis. *J. Periodontol.* 1991, 62, 1351141.

[20] Dequeker J., Mbuyi J.M.: Bone ageing and osteoporosis evaluated by radiogrammetry of cortical bone. *Rheumatologie* 1981, 33, 2271232.

[21] Dequeker J., Geussens P., Wielandts L., Nijls J.: Longitudinal data on bone mass: comparison between photon absorptiometry and radiogrammetry. *Acta Rhumat.* 1980,4,2, 2021204.

[22] De Palma L., Greco F. i wsp.: Valutazione dell'evoluzione del callo di frattura mediante analisi computerizzata di immagini radiografiche. *Radiol. Med.* (Torino) 1991, 82, 44147.

[23] Schmidt C.W., Leuschke W.: Ruckbildung einer Skelettfluorose nach Beendigung nicht berufsbedingter Exposition. *Z. Gesamte. Inn. Med.* 1989, 44, 2211223.

[24] Schneider P., Borner W., Mazess R.B., Barden H.: The relationship of peripheral to axial bone density. *Bone Mineral*. 1988, 4, 2791287.

[25] Schnitzler C.M., Wing J.R. i wsp.: Bone fragility of the peripheral skeleton during fluoride therapy for osteoporosis. *Clin-Orthop*. 1990, 261, 2681275.

[26] Seldon H.L.: Three-dimensional reconstruction of temporal bone from computed tomographic scans on a personal computer. *Arch. Otolaryngol. Head Neck Surg*. 1991, 117, 115811161.

(Received 30 September 2014; accepted 21 October 2014)