Research Article

ANTHROPOGENIC FLUORIDE CONTAMINATION AND OSTEO-FLUOROSIS IN BOVINES INHABITING UMARDA, JHAMARKOTRA AND LAKKADWAS VILLAGES OF UDAIPUR, RAJASTHAN, INDIA

***Z. Sheikh and L. Panchal**
Department of Zoology, Govt. Meera Girls College,
Udaipur-313001(Raj.)
Author for Correspondence: zulfiyasheikh@gmail.com

ABSTRACT
A study was conducted to assess osteo-fluorosis in bovines in Umarda, Lakkadwas and Jhamarkotra villages of Girwa tehsil, Udaipur, Rajasthan. For the evaluation of osteo-fluorosis 1528 cattle (*Bos taurus*) and buffaloes (*Bubalus bubalis*) of different age groups of both sexes were examined. Various symptoms of osteo-fluorosis like stiff joints, poor body condition, muscle wasting, bony exostoses, diffidence to move, foot dragging, and snapping sound during walking were looked for in the animals. Chronic signs of skeletal fluorosis were shown by 52.92% of cattle & 49.47% of buffaloes while 39.40% of cattle & 36.64% of buffaloes showed mild to moderate signs of fluorosis. It was observed that cattle were more sensitive to fluoride as compared to buffaloes. Results suggested that fluoride contaminated hard ground water, fodder grown on F contaminated soil, gaseous emission from surrounding factories and rock phosphate mining were the main sources of fluoride in these villages. Apart from the natural fluoride sources these anthropogenic sources were mainly responsible for fluoride toxicosis in animals.

Key words: *Bovines; Fertilizer Factories; Osteo-fluorosis; Rock Phosphate Mining.*

INTRODUCTION
Fluoride toxicosis is a dreaded disease caused by excessive ingestion of fluoride. Prolonged exposure with high amount of fluoride exerts deleterious effects on human beings, animals and plants too (Connett *et al.*, 2010; Sauerheber, 2013; Choubisa, 2018). Fluoride can enter into the body through contaminated water, food and by inhalation of contaminated gases. Another source of fluoride uptake in grazing animals can be contaminated soil ingestion. Fluoride is an inevitable industrial toxicant and pollutant of geological origin which is frequently found in water. Anthropogenic sources of fluoride like phosphate fertilizer factories, phosphoric acid forming factories, brick kilns, coal burning and mining of rock phosphate release fluoride into the environment and effluents & waste products of these factories contaminate the soil and groundwater when discharged untreated (Mishra *et al.*, 2010). These fluoride emitting industries are serious issue of concern not only for man but also for animals (Choubisa and Choubisa, 2015; Choubisa and Choubisa, 2016). Fluoride enters in to water, from natural as well as anthropogenic sources like mining process and industries, where fluoride mineral is used as crude material for making fertilizers and other products (Mohammadi *et al.*, 2017). Water and fodder are the main sources of fluoride intake in animals. According to WHO (2004) permissible limit of fluoride in drinking water is 1.5ppm. Main observable symptoms of fluoride ingestion are dental and skeletal fluorosis (Darchen *et al.*, 2016). Fluoride generally affects hard tissues of the body like teeth, bones and cartilaginous tissues. Bones are important site for fluoride storage in animals; therefore bones give evidence of potential exposure to toxic level of fluoride (Ouko and Peter, 2016). Fluoride can alter a bone's internal environment, structure and growth. It can induce many deformities like hyperostosis, exostoses, osteosclerosis and osteomalacia (Shupe *et al.*, 1992). Fluoride contaminated groundwater and air cause skeletal fluorosis in animals. Skeletal fluorosis is a disorder related to prolonged accumulation of fluoride in bones resulting into fragile bones having low elasticity in tendons, fused bones and immoderate calcification (Kadu *et al.*, 2012). Common symptoms of skeletal fluorosis are poor body condition, muscle wasting, locked up joints, diffidence to move, painful and rigid joints, bony outgrowths and osseous lesions in animals.

Research Article

Therefore the present study was focused on assessment of osteo-fluorosis in bovines reared in the proximity of rock phosphate mining sites and processing units in three villages of Udaipur, Rajasthan.

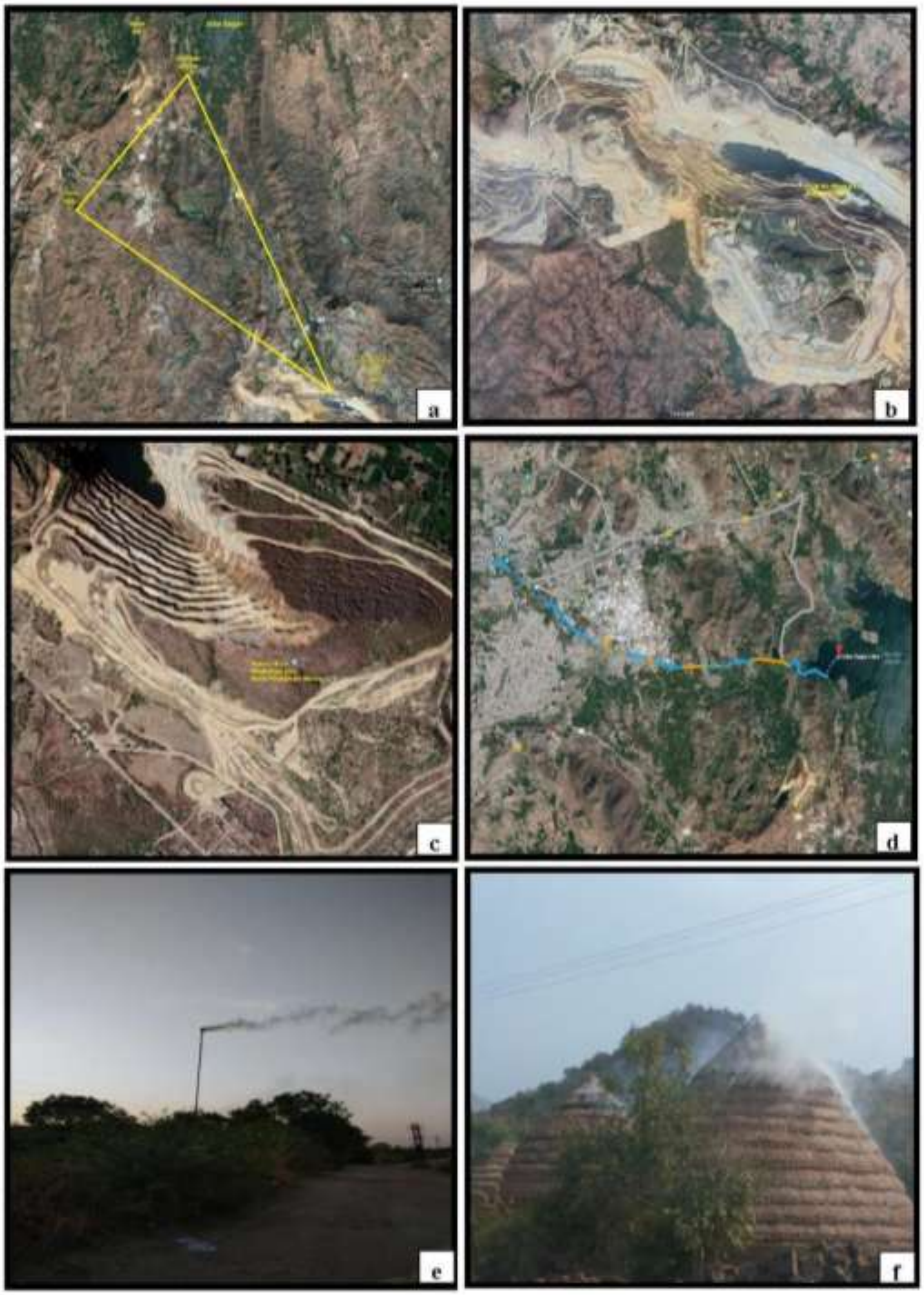

Figure 1: (a) Satellite image of the study area (Umarda, Lakkadwas and Jhamarkotra) (b) Jhamarkotra mines of rock phosphate (c) Matoon rock phosphate mines near Lakkadwas village (d) Ayad river traversing the city and emptying into Udaisagar lake (Source google.co.in) (e) Gaseous emission from phosphate fertilizer factories and (f) Brick kilns

MATERIALS AND METHODS

The study area included three villages- Jhamarkotra, Lakkadwas and Umarda. Umarda village is divided into 12 regions, Lakkadwas into 15 regions and Jhamarkotra is divided into 9 regions. These villages are a part of Girwa tehsil and surrounded by many phosphate fertilizer plants, brick kilns and rock phosphate mining sites. Umarda is surrounded by several processing units of phosphate fertilizers and brick kilns whereas Lakkadwas (Matoon mines) and Jhamarkotra (RSMML mines, largest reserve of phosphates in India) are mining areas of rock phosphate (Fig.1 a, b, c).

For the evidence of skeletal fluorosis, a survey was done in all the three villages. A total of 1528 domestic animals including cattle (*Bos taurus*) and buffaloes (*Bubalus bubalis*) were observed for signs of osteo fluorosis. Both Indian, as well as exotic breeds as Kathiyawadi, Nagauri, Tharparkar, Holstein-friesian, and Jersey of cattle while Murrah and Surati breeds of buffaloes were reared in the study area. Symptoms of osteo fluorosis like stiff joints, poor body condition, hoof deformities, muscle wasting, bony outgrowths or exostoses, reluctance to move or restricted movement, osseous lesions, arched back bone etc. were looked for in the animals. On the basis of the severity of these symptoms of skeletal fluorosis the animals were divided into mild, moderate or severe categories. Other common complaints like lack of appetite, bloating, poor lactation, low birth rate etc. were also taken into consideration. At the same time animal's water and food supply were also investigated.

RESULTS AND DISCUSSION

The survey for assessment of skeletal fluorosis in bovines revealed that animals in all the three villages were affected by natural as well as anthropogenic fluoride contamination. The study area has large deposits of rock phosphates and is regularly mined for related industries. The mining and processing units in this area release fluoride in the surroundings. The ground water and Udaisagar lake are main sources of drinking water; both of these are contaminated with fluoride. Of the three villages, Lakkadwas village was found to be the most afflicted. Lakkadwas is receiving highly contaminated water supply from Udaisagar lake and at the same Matoon mines of rock phosphate is also adding fluoride content to it. Ayad river which is laden with domestic, industrial effluents from Madri industrial area and sewage discharge of the city, supplies water to Udaisagar lake and in turn dumps all the polluted waste (Fig. 1 d). The fluoride concentration of Udaisagar lake (surface water) ranged from 2.1 to 3.6ppm suggesting airborne F contamination of surface water as well. Rock phosphate leaching from Matoon mines is making the situation more worse (Das, 1999). Variability of fluoride in water depends on the soil and rocks of the area. Earlier studies have suggested that water-rock interaction is responsible for fluoride enrichment in ground water (Dehbandi *et al.*, 2017).

Table 1: Prevalence of skeletal fluorosis in cattle

Age group	No. of animals	Mild to moderate	Severe
<1 year	208	41(19.71%)	65(31.25%)
1-4 years	267	118(44.19%)	149 (55.80%)
>4 years	398	150(37.69%)	248 (62.31%)
Total	**873**	**309 (39.40%)**	**462(52.92%)**

*Numbers in parenthesis indicate percentage

Table 1 depicts skeletal fluorosis in cattle of different age group. In the age group of less than one year, 208 calves were examined for the evidence of skeletal fluorosis and 19.71% of calves showed mild to moderate signs of osteo fluorosis, while 31.25% of calves showed severe signs of skeletal fluorosis. Poor body condition, muscle exhaustion and stunted growth were common symptoms in the fluorotic calves (Fig. 2a). Earlier studies also reported similar symptoms in calves (Choubisa *et al.*, 2011). Sources of fluoride in this young stage might be fluoridated water and milk. Calves generally depend on mother for their food supply and there are chances of fluoride transmission through milk (Hobbs *et al.,* 1954). Calves are exposed to fluoride during development in uterus also because mothers are also exposed to high fluoride and reports suggest that fluoride could pass through placenta (Krook and Maylin, 1979). Maylin *et al*., (1987) also reported that calves born from fluoride intoxicated cows had severe symptoms of osteo dental fluorosis such as mottling, discolouration of teeth, enamel hypoplasia, severe malfunctioning in cartilage cell differentiation, osteoblasts bone marrow atrophies and stunted growth. Immature cattle have high sensitivity and susceptibility and less tolerance to fluoride (Kumar *et al.,* 2015), hence at very young stage of life calves reveal the signs of fluoride toxicity. Calves of the study areas were reared in the vicinity of rock phosphate fertilizer factories and brick kilns, and received fluoride through gaseous emission from these factories and fluoride contaminated fodder that resulted in dental and skeletal fluorosis.

In the age group of 1-4 years, 44.19% of the cattle showed mild to moderate signs of skeletal fluorosis and 55.80% of cattle showed severe signs of osteo fluorosis. Apart from extreme muscle atrophy especially in pelvic region, shoulder muscles and frail body, the cattle had stiff joints & legs, bony exostoses, long bones alterations, arched backbone, reluctance to move, painful gait, and lameness (Fig. 2 b). Vertebral column and ribs of the animals were the most affected especially thoracic and lumbar regions. Similar symptoms of industrial fluorosis had been observed in cattle population of Orissa by Mishra and Pradhan, 2007. Choubisa *et al.,*(2011) also observed neighbourhood fluorosis while Kumar *et al.,*(2015) reported similar symptoms of skeletal fluorosis in cattle of Bikaner and Naguar district of Rajasthan. Chronic dental fluorosis in live stock in these areas has also been reported by Panchal and Sheikh in the year 2017. Water is the major medium of fluoride intake by animals and humans. Animals may consume naturally occurring fluoride as well as ingestion of contaminated forage increases chances of fluoride toxicity (Narwariya and Saksena, 2012). The topsoil, forage and grasses grown in industrial and rock phosphate mines areas are generally contaminated by industrial fluoride emission and the accumulation of fluoride on foliage depends on the atmospheric loading. Rock phosphate contains about 25% fluorine which is converted into vapor and around 25000 tons of fluorine is added in to the environment annually. The environmental chronic fluoride intoxication leads to dental and skeletal deformities (Begum *et al.,* 2008). Excess sources of fluoride intake in animals of the study area were fluoridated drinking water, contaminated soil, fodder and gaseous emissions (Fig. 1 e & f). Pandey and Pandey (2011) reported that airborne fluoride was a major factor for fluorosis in the vicinity of phosphate fertilizer factories in the rural areas of Udaipur.

Most industrial fluoride emissions are airborne and may contaminate soil, water, and vegetation not only in the industrial vicinity, but also up to a substantial distance from the emission source (Ranjan and Ranjan, 2015). Airborne fluoride generally settles over plant leaves and is consumed by grazing animals. Thus herbivorous are expected to consume more fluoride than grainivorous (Swarup and Dwivedi, 2002). In the older age cattle belonging to more than 4 years age group, 62.31% of cattle exhibited chronic signs of fluoride toxicity. Animal of this age group were severely affected due to prolong and constant exposure to high fluoride. Restricted movement, painful and stiff joints, lameness, fragile bone, visibly enlarged bones, bony exostoses, osteosclerosis, and snapping sound during walking were observed as severe signs of skeletal fluorosis in cattle (Fig. 2 c). Few animals in this age group were totally immovable and posed a burden on the owner who had no other option than to abandon them or leave to die.

Highest prevalence of osteo-fluorosis in this age group is alarming as these animals when consumed by human or other carnivores would result in an increased intake of fluoride. Extreme muscle atrophy was observed in hip regions of these animals. Stiff painful joints & lameness restricted the movement of animals which resulted into low food intake and poor body condition. Industrial fluorosis and its effects on cattle have also been reported by Kumar and Arvindaksham (2015). Hyperostosis, exostoses and osteosclerosis were observed in cattle by Shupe (1992). Jena *et al*., (2016) also speculated these chronic signs of fluoride toxicosis in cattle reared in the vicinity of aluminium smelter in Odisha. Bony exostoses in metacarpal, metatarsal, ribs and frontal bones and lameness in cattle are also reported by Gupta *et al*.,

(2013) as signs of skeletal fluorosis. Fluoride accumulated fodder is a threat to feeding cattle (Mondal *et al.*, 2017). Vegetation used for fodder grown in the close proximity of the factories is polluted by gaseous emission as well as by F in water & soil.

Figure 2: (a) Exposed vertebral column and ribs with muscle deterioration in an eight months calf (b) Feeble body with pelvic muscle loss (c) Extreme muscle atrophy and stiffened bones leading to immobilization

Figure 3: (a) Feeble body with pelvic muscle regression in six months calf (b) Sinking pelvic muscles with bony lesions and lameness (c) Arched backbone, extreme muscle wasting and exposed pelvic bones

Table 2 depicts the status of skeletal fluorosis in buffaloes of different age group. In the age group of less than 1 year, 17.79% of buffalo calves showed mild to moderate symptoms of skeletal fluorosis and 26.38% of buffalo calves were found afflicted with severe skeletal fluorosis. Calves were feeble and showed muscle regression especially in the pelvic region (Fig. 3 a). Leg bones as metatarsus and tibia fibula appeared delicate and rib cage was also exposed. Choubisa (2014) stated that bovine calves are less tolerant and highly susceptible to fluoride.

Table 2: Prevalence of skeletal fluorosis in buffaloes

Age group	No. of animals	Mild to Moderate	Severe
<1 year	163	29(17.79%)	43(26.38%)
1-4 years	229	97(42.35%)	130 (56.76%)
>4 years	263	114(43.34%)	151 (57.41%)
Total	**655**	**240(36.64%)**	**324(49.47%)**

**Numbers in parenthesis indicate percentage*

In the age group of 1-4 years 56.76% of buffaloes showed severe signs of skeletal fluorosis while 42.35% of buffaloes were found to be mild to moderately afflicted with skeletal fluorosis. Frail body, bony exostoses, locked up joints, sinking and flattened hind muscles, exposed skeleton were common in buffaloes of this age group (Fig. 3 b). Lameness and immobilization were also noticed in buffaloes. Painful gaits due to periosteal exostoses at ligament & tendons, osteomalacia and osteoporosis have been observed in fluorotic animals by Samel *et al.*, (2016).

In buffaloes belonging to more than 4 years age group, 57.41% showed chronic signs of osteo fluorosis while 43.34% showed mild to moderate symptoms of skeletal fluorosis. Extreme muscle atrophy, arched backbone, weak and delicate metatarsus and tibia fibula were observed in older age buffaloes. Hoof deformities, bony exostoses, feeble body and deteriorating muscles were quite noticeable in buffaloes of this age (Fig. 3 c). Yanhu (1991) reported industrial fluorosis in buffaloes in China and 30% of buffaloes were found affected with skeletal fluorosis. With increasing age the amount of fluoride intake, duration of exposure make these animals reluctant to move or graze.

Fluoride concentrations in plasma are not controlled homeostatically and thus chronic doses lead to accumulation in bone and plasma. Most of the fluoride in the body is contained in bones. The crystallites of developing bone are small sized, more in number and heavily hydrated, providing an enormous surface area for reactions involving fluoride as compared to mature bone. Fluoride is readily incorporated into calcified tissues, as bone and teeth, substituting for hydroxyls in hydroxyapatite crystals. Fluoride exchanges between bone and body fluids in a short term process at surface layer of bone and long-term process in remodeling bones. High concentrations of calcium in gastrointestinal tract are known to cause net excretion of fluoride (Whitford, 1996). So absorption of fluoride from water manifests the adverse effects on skeleton in long term. Owners should be made aware of the issue and supplements of calcium, vitamin C and Boron should be given to animals as a remedial measure (Sheikh, 2011; Bharti *et al.*, 2007).

CONCLUSION

In the present study it was perceived that cattle were more susceptible than buffaloes in the age group of more than 4 years. Among the three villages surveyed, highest prevalence of chronic fluoride toxicity was shown by cattle of Lakkadwas village. It is probably due to the leaching of contaminated water from Udaisagar lake, located in Lakkadwas village which is filled by highly polluted Ayad river and rock phosphate mines in Matoon near the village, added fluoride to the ground water as well. Gaseous emission from the factories made the situation worst. Results of our study stipulated that anthropogenic sources were mainly responsible for fluoride toxicosis in animals of the study area. Major anthropogenic sources of excess fluoride were industrial emission, mining process of rock phosphate, contaminated soil, water and vegetation. Animals are the pivot of the Indian village economy and it's mandatory to provide a good health to animals. Also, the industries must develop proper waste treatment strategies to combat environmental pollution. If the drinking water sources contain fluoride more than the permissible limit, defluoridation of water using various methods is suggested. The villagers can be educated to store rainwater for domestic use and irrigation. Locally available plants and their products rich in calcium can be fed to the animals to lower the risk of fluoride toxicity.

REFERENCES

Begum A, Harikrishna S and Khan I (2008). Environmental fluoride level in Anekal Taluk of Banglore district. *Rasayan Journal of Chemistry* **1**(4)774-781.

Bharti VK, Gupta M, Lall D and Kapoor V (2007). Effects of boron on haemogram and biochemical profile of urine in buffalo calves fed a high fluoride ration**.** *Fluoride* **40** (4)238-243.

Choubisa SL (2014). Bovine calves as ideal bio-indicators for fluoridated drinking water and endemic osteo-dental fluorosis. *Environmental Monitoring and Assessment* **186** (7) 4493-4498.

Choubisa SL (2018). A brief and critical review on hydrofluorosis in diverse species of domestic animals in India. *Environmental Geochemistry and Health* **40**(1) 99-114.

Choubisa SL and Choubisa D (2015). Neighbourhood fluorosis in people residing in the vicinity of superphosphate fertilizer plants near Udaipur city of Rajasthan (India). *Environmental Monitoring and Assessment* 187:497.

Choubisa SL and Choubisa D (2016). Status of industrial fluoride pollution and its diverse adverse health effects in man and domestic animals in India. *Environmental Science and Pollution Research* **23**(8)7244-7254.

Choubisa SL, Mishra GV, Sheikh Z, Bhardwaj B, Mali P and Jaroli VJ (2011). Toxic effects of fluoride in domestic animals. *Advances in Pharmacology and Toxicology* **12**(2)29-37.

Connett P, Beck J and Micklem HS (2010). The case against fluoride- how hazardous waste ended up in our drinking water and the bad science and politics that keeps it there. *Fluoride* **43**(3)170–173.

Darchen A, Sivasankar V, Prabhakaran M and Bharathi CB (2016). Health effects of direct or indirect fluoride ingestion. In: Sivasankar V. (eds) Surface modified carbons as scavengers for fluoride from water. Springer, Cham 33-62.

Das BK (1999). Environmental pollution of Udaisagar lake and impact of phosphate mine, Udaipur, Rajasthan, India. *Environmental Geology* **38**(3)244-248.

Dehbandi R, Moore F, Keshavarzi B and Abbasnejad A (2017). Fluoride hydrogeochemistry and bioavailability in groundwater and soil of an endemic fluorosis belt, central Iran. *Environmental Earth Sciences* 76:177.

Google.co.in[**2017**]. https://www.google.co.in/maps/place/Udaipur,Rajasthan/@24.4701319, 73.8022377, 11546m/data *[Accessed on 18 Dec. 2017]*.

Gupta AR, Dey S, Swarup D, Saini M, Saxena A and Dan A (2013). Ameliorative effect of *Tamarindus indica L*. on biochemical parameters of serum and urine in cattle from fluoride endemic area. *Veterinarski Arhciv* **83**(5)487-496.

Hobbs CS, Moorman RP, Griffith JM. West JL, Merriman GM, Hansard SL, Chamberlain CC (1954). Fluorosis in cattle and sheep. *University of Tennessee Agriculture Experiment Station, Bulletin no.* 235.

Jena CK, Gupta AR and Patra RC (2016). Osteo-dental fluorosis in cattle reared in villages on the periphery of the aluminium smelter in Odisha, India. *Fluoride* **49**(4)503-508.

Kadu AS, Nampalliwar AR and Gothecha VK (2012). Skeletal fluorosis due to chronic fluoride intoxication- An over review. *International Journal of Ayurvedic and Herbal Medicine* **2**(3)561-568.

Krook L and Maylin GA (1979). Industrial fluoride pollution. Chronic fluoride poisoning in Cornwall Island cattle. *The Cornell Veterinarian* **69**(8)1-70.

Kumar M, Yadav M and Choudhary A (2015). Effect of fluoride concentration on cattle of Nagaur district (Rajasthan). *International Journal of Scientific and Research Publications* **5**(9)1-2.

Kumar PS and Aravindakshan CM (2015). Industrial fluorosis and its effects on serum biochemistry and haemogram in cattle of Kerala, India. *Proceedings of the National Academy of Sciences, India section B: Biological Sciences* **85**(3)867–872.

Maylin GA, Eckerlin RH and Krook L (1987). Fluoride intoxication in dairy calves. *The Cornell Veterinarian* **77**(1)84-98.

Mishra CSK, Nayak S, Guru BC and Rath M (2010). Environmental impact and management of wastes from phosphate fertilizer plants. *Journal of Industrial Pollution Control* **26** (1)57-60.

Mishra PC and Pradhan K (2007). Prevalence of fluorosis among school children and cattle population of Hirakud town in Orissa. *The Bioscan* **2**(1)31-36.

Mohammadi AA, Yousefi M, Yaseri M, Jalilzadeh M and Mahvi AH (2017). Skeletal fluorosis in relation to drinking water in rural areas of West Azerbaijan, Iran. *Scientific Reports* 7: 17300 doi: 10.1038/s41598-017-17328-8.

Mondal, NK (2017). Effect of fluoride on photosynthesis, growth and accumulation of four widely cultivated rice (*Oryza sativa L*.) varieties in India in. *Ecotoxicology and Environmental Safety* 144:36-44.

Narwariya YS and Saksena DN (2012). Incidence of dental fluorosis in domestic animals of Shivpuri, Madhya Pradesh, India. *Journal of Environmental Research and Development* **7**(1A) 426-30.

Ouko OC and Peter AW (2016). Importance of contaminated soils in supplying bioaccesssible fluoride to grazing animals from the historic metalliferous mining areas of the UK. *Journal of Health and Environmental Research* **2**(5)27-33.

Panchal L and Sheikh Z (2017). Dental fluorosis in domesticated animals in and around Umarda village of Udaipur, Rajasthan, India. *Haya:Saudi Journal of Life Sciences* **2**(7)248-254.

Pandey J and Pandey U (2011). Fluoride contamination and fluorosis in rural community in the vicinity of a phosphate fertilizer factory in India. *Bulletin of Environmental Contamination and Toxicology* **87**(3)245-9.

Ranjan R and Ranjan A (2015). Sources of fluoride toxicity. Springer Briefs in Animal Sciences. doi 10.1007/978-3-319-17512-6_1. 2015.

Samal P, Jena D, Mahapatra A, Sahoo D and Behera S (2016). Fluorosis: An epidemic hazard and its consequences on livestock population. *Indian Farmer* **3**(3):180-184.

Sauerheber R (2013). Physiologic conditions affect toxicity of ingested industrial fluoride. *Journal of Environmental and Public Health.* http://dx.doi.org/10.1155/2013/439490.

Sheikh Z (2011). Prevention of fluorosis in domestic animals. *Current Science* **101**(9)1124-25.

Shupe JL, Bruner R H, Seyhmor JL and Alden CL (1992). The pathology of chronic bovine fluorosis: A review. *Toxicologic Pathology* **20**(2)274-282.

Swarup D and Dwidedi SK (2002). Environmental pollution and effects of lead and fluoride on animal health. Directorate of Information and Publications of Agriculture, Indian Council of Agricultural Research, Krishi Anusandhan Bhavan, Pusa, New Delhi.

Whitford GM (1996). The Metabolism and Toxicity of Fluoride. 2nd Rev. Ed. Monographs in Oral Science, Vol.16, New York: Karger.

WHO (2004). Fluoride in drinking-water. Background document for preparation of WHO Guidelines for Drinking-water Quality. Geneva, World Health Organization (WHO/SDE/WSH/03.04/96.2004).http://www.who.int/water_sanitation_health/dwq/chemicals/fluoride.pdf). [accessed on 18 Dec. 2017].

Yanhu Y (1991). The influence of industrial fluorine pollution on livestock ecological environment and its health. *Journal of Agro Environmental Science* 04.