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A BRIEF REVIEW OF FLUORIDE TOXICITY AND ITS ADVERSE EFFECTS ON DOMESTIC ANIMALS

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ABSTRACT

Fluoride (F) toxicity is the major challenging health issue of the 47 nations of the world including India. Fluoride is toxic to all living things. Living organisms are received excess fluoride along with their food, water and air. Exposure of excess F for a long period causes fluorosis in animals and human. South Asia is considered as the epicentre of F contamination in groundwater. Groundwater of all South Asian countries like India, Bangladesh, Pakistan and Sri Lanka are highly polluted with fluoride. Almost all Indian states have excess F in groundwater. Goundwater contaminated with F, uses for irrigation and drinking purposes are considered to be harmful not only the human beings but also to the animals and plants.

Plants and other vegetations received fluoride from soil and water by their roots and through stomata by air, and accumulate it. Animals received excess F from drinking water, household products, chemical used in agriculture (pesticides and fertilizers), use of industrial ndustrial fluoride contaminated or volcanic ash deposited fodder and grasses, and sometimes via medicine and feed supplements. Soil rich in fluoride bearing minerals and rocks are chief source of soluble fluoride. In grazing animals soluble fluoride is prime source of fluorosis. Although oral ingestion is the principal route of fluoride intake in animals but airborne also contribute it. Some reports revealed that the pasture contaminated with volcanic ash containing fluoride causes mortality in grazing animals. Several workers have been earlier reported detrimental effects of fluoride in domestic animals viz. osteo-dental fluorosis, gastrointestinal disorders, hepatic, renal and neurological malfunctioning, endocrine and reproductive manifestations. These detrimental effects of fluoride reported in domestic animals are found to be similar in human and other livestock.

Keywords: Fluoride toxicity, sources, metabolism, adverse effects, domestic animals

INTRODUCTION

Fluorine (F) is the 13th most abundance element found in the Earth crust and considered most important natural pollutant. Last few decades, it receiving great attention due to its high toxicity and reactivity. Fluorine is member of halogen family, placed in 7th group of periodic table. Ionic form of Fluorine is called Fluoride (F). Fluoride is most electronegative and highly reactive ion cannot exist in free state in nature and always found to associate with another element or compounds. Fluoride is potentially harmful to our environment and living organisms (Schlesinger *et al*., 2020; Fuge, 2019). Fluorine released in the environment due to natural and anthropogenic activities. The major geogenic or natural sources of fluoride are fluorine bearing minerals such as cryolite, apatite, topaz fluorite, micas, amphiboles and sellaite, all are found in earth crust throughout world (Hem,1985; Pickering, 1985). Fluoride reached in soil water by the process of weathering and leaching of mineral rocks, industrial emission and volcanic eruption, (Yadav *et al*., 2021). Hem (1985) suggested, weathering and leaching of fluoride-bearing minerals are associated with low calcium and high bicarbonate ions. Churchill *et al.* (1948) stated that coal contains about 229 ppm of fluorine. Dutta *et al*. (1996) and Ali *et al.* (2016) reported main anthropogenic sources of soil water system include mining activities, brick kilns, thermal power plants, pesticides and fertilizers, cement, tiles, ceramics, steel, glass and aluminum manufacturing industries.

According to Millar (2000) excessive fluoride exists in natural environment (air, soil and water) may affected every living organism. Choubisa *et al* (2022) suggested that fluoride is toxic to human, plants and animals. World Health Organization (WHO, 2004) and Bureau of Indian Standards (BIS, 2012) recommended maximum permissible limit of fluoride in drinking water is 1.5 mg/L.

Elevated fluoride level in water, food and air not only threaten to human but also to animals. Several workers have been reported prevalence of fluorosis in domestic animals in different states of India such as Rajasthan (Choubisa *et al*., 1996; Choubisa, 1999, 2000, 2013, 2014 & 2021), Andhra Pradesh and Uttar Pradesh (Dwivedi *et al*.,1997), Punjab (Sharma *et al.*,1997), Karnataka (Muralidhara *et al.,* 2000) and Orissa (Maiti *et al.,* 2004).

Yu *et al*. (2011) reported acute and chronic effects of fluoride toxicity in animals including nausea, vomiting, diarrhoea, gastroenteritis, muscular weakness, pulmonary congestion, respiratory and cardiac failure and eventually death as under acute effects of fluoride whereas dental and skeletal fluorosis as under chronic effects of fluoride. All these acute and chronic effects of fluoride was found similar in both human and animals. They also performed various scientific studies on animals and suggested that fluoride has adverse effects on feeding efficiency, blood composition, abilities of learning and memory, reproductive system, growth and developmental.

Alonso & Camargo (2011) in a laboratory experiment observed, reduce number of shelled embryos and abnormal behavioural activities in aquatic snail, *Potamopyrgus antipodarum* under fluoride toxicity and suggested that fluoride is toxic to snails and it can cause mortality, impairment of reproduction and behaviour. In an experiment Gupta *et al.* (2007) reported high intake fluoride in drinking water is associated with decreased birth rates in rats. They were also

reported testicular disorders, impaired reproductive system and fertility in male rats after administration of sodium fluoride in drinking water for 6 months.

Basha *et al*. (2011) observed significant decline in serum-free thyroxin, free triiodothyronine levels, acetylcholine esterase activity in fluoride treated rats and suggested that decreased levels of thyroid hormone might be due to inhibition of iodine absorption by fluoride, resulting impairment of learning and memory in rats. Madan *et al*. (2009), studied fluoride toxicity in animals and suggested that the biological responses of animals are directly related to the fluoride concentration and many other factors that influence the physiological and anatomical responses in animals.

Choubisa (2012), Choubisa & Mishra (2013) and Jena *et al.* (2016) were reported that the excess amount of fluoride in groundwater causes dental and skeletal fluorosis in animals. They reported common signs of dental fluorosis are staining, irregular enamel pits, patches, receding gums and exposed teeth roots in animals. Findings of several other workers (Choubisa, 2017; Narwariya & Saksena, 2012; Tiwari & Kaur, 2008) revealed that severely affected animals shows abrasion of enamel, loose tooth, fragile and damaged teeth, and total loss of teeth. In these severe conditions animals feel difficulty in mastication and show lack of interest in food intake results dullness and weakness. In India, most survey and review work related to fluoride toxicity in different species of cattle and domestic animals were carried out by Choubisa and co-workers. Choubisa (2012) published a comphenshive review article entitled "Status of fluorosis in animals". In successive years (Choubisa, 2017, 2018, 2021, 2022 and 2023) he also published review articles related to fluoride toxicity in animals. Although the toxic effects of fluoride in human beings are well studied and documented time to time whereas adverse effects of fluoride on animals are least concern, scanty and can be need to compilation. Keeping this view in mind the present manuscript has been prepared.

FLUORIDE SOURCES FOR DOMESTIC ANIMALS:

Domestic animals intake excess fluoride from a variety of geogenic, natural and anthropogenic sources. The chief sources of fluoride for animals include forage or vegetations grown in fluoride rich soil or contaminated by industrial effluents or windblown, fluoride contaminated drinking water, and feed supplements containing excess fluoride. (Shupe 1980; Swarup and Dwivedi, 2002). Shupe (1980) stated, although the fluoride absorbed in animals by several routes but oral ingestion is considered the major route of fluoride absorption. According to Shupe and Olson (1971) due to natural processes such as erosion, leaching, mining and volcanic eruption, soluble fluoride reach on the earth's surface and atmosphere and animals are ingested it directly when grazing over the fluoride rich soil. The important sources of fluoride toxicity to animals are described briefly in Figure -1.

Mineral sources of Fluoride:

Fluorine containing rocks and minerals occur in earth's crust are the main sources of fluoride. Soluble fluoride mix in water and atmosphere by degradation of rocks (Jacks *et al*., 2005). Some important F containing minerals are fluorite (CaF_2), cryolite (Na_3AlF_6) and fluroapatite ($Ca_5(PO_4)_3F$ (Edmunds & Smedley, 2005; Tavener & Clark, 2006).

Anthropogenic sources of Fluoride:

The main anthropogenic sources of fluoride are industrial effluents and emissions, motorization, use of fluoridation of drinking water, use of fluoride containing agrochemicals such as pesticides & phosphate fertilizers, , dental & cosmetic products, drugs & food supplements, fire extinguishers and refrigerators (WHO, 2002; Paul *et al*., 2011). Fuge & Andrews (1988) reported, rainfall contaminated with industrial emission rich in fluoride are also responsible for fluorosis in farm animals, situated nearby the industries.

Fluoride rich forage and grasses:

Forages and grasses, present near the industrial area are highly contaminated with fluoride rich dust, industrial effluents, ash, splashing on soil particles on fodder by rain. When these fluoride rich food is ingested by animals causes fluorosis. Some studies revealed that the contamination of vegetation with fluoride depends on various factors like distance from the F source, amount of F in emitted in effluents and emissions, types of vegetation, atmospheric conditions, seasons and height of the plants etc. (NRC 1960; Mascola *et al*., 1974).

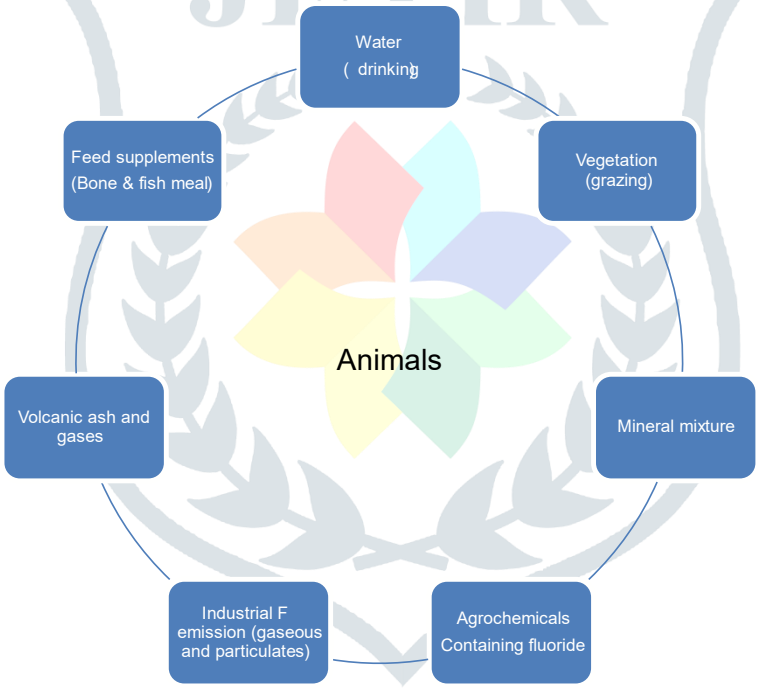

Fig. 1 Fluoride sources for domestic animals

Volcanic sources of Fluoride:

During volcanic eruption huge amount of fluoride released in atmosphere in the form of HF. Erupted F spread upto long distance and cover a large area. Emitted F may exists for long time on vegetations or on due to decaying and leaching activities, it mixed in soil water system and caused severe effects on domestic and wild animals (Araya *et al*., 1993; Bellomo *et al*., 2007).

FLUORIDE METABOLISM IN DOMESTIC ANIMALS

According to Whitford (1996) fluoride mostly enters the animal body via gastrointestinal tract and is absorbed quickly in the stomach and mostly in intestine without need of any specialized enzyme. In aqueous medium of gastrointestinal tract fluoride form HF, which crosses the gastric epithelium and finally reached in blood stream.The rate of fluoride absorption from the stomach is directly related to the acidity of its contents (Whitford & Pashley, 1984). However, several other factors also influence the rate of absorption.

Once absorbed F reach in plasma, it rapidly deposited in both hard and soft tissues of the body. Excess amount of F remain in the plasma after metabolism in animal body is excreted in urine via kidneys, faeces, milk, sweat and saliva . The outline of fluoride metabolism in animals well described in Figure -2.

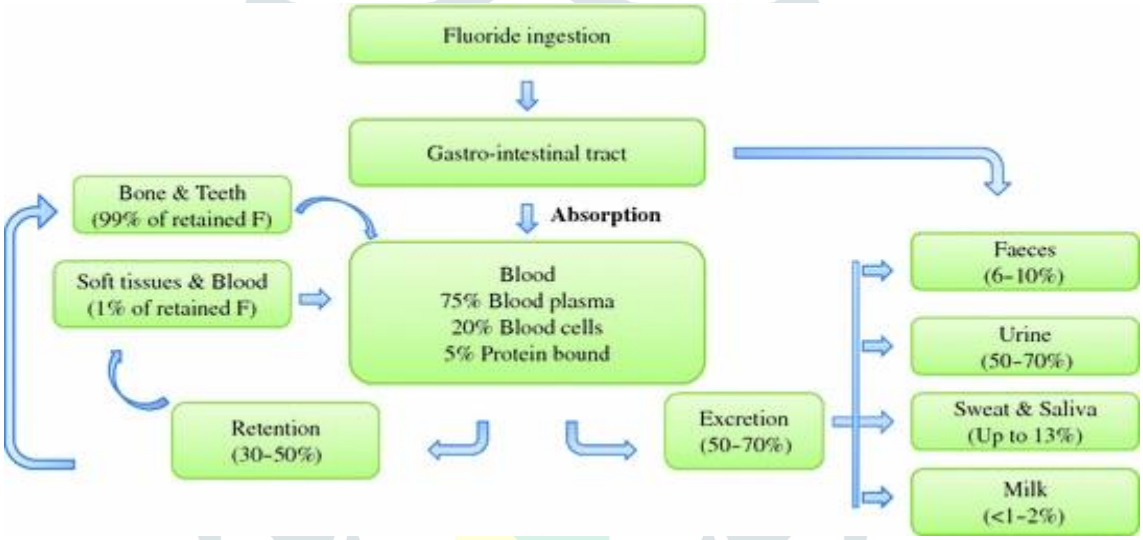

Fig. 2 Flow chart of fluoride metabolism in animals (Adapted from Ranjan & Ranjan, 2015)

ADVERSE EFFECTS OF FLUORIDE ON DOMESTIC ANIMALS

Intake of excess fluoride for a long period causes osteo-dental fluorosis in animals. Excess fluoride accumulated in hard body tissues specially in teeth and bones. Besides these it also causes adverse effects on other body organs by altering its architecture and physiology. In recent decades several workers have been established by their findings and observations that the excess fluoride in food and drinking water causes dental, skeletal and non-skeletal fluorosis in different species of domestic animals.

Anonymous (1973) reported F is one of the 14th essential element and performing various physiological functions in the animal's body such as calcification of teeth and bones, activation of enzymes, fertility and haematopoiesis. Wheeler and Fell (1983) stated necessity of fluoride in biological functions is still doubtful. Swarup and Dwivedi (2002) were observed intake of high fluoride causes fluorotoxicity in animals and reported various fluorotic symptoms in teeth, bones and soft tissues of body are commonly referred as dental, skeletal and non-skeletal fluorosis.

Many other studies suggested that the young animals are more prone to fluoride deposition in its body tissues and deposition of it dependent on various factors such as food, nutrients, age, exposure duration and frequency (Choubisa, 1999; Choubisa, 2007). Kaminsky *et al*. (2011) were reported fluoremia in animals which increases along with the age of animal. Some important adverse effects of fluoride toxicity reported by several workers are described briefly-

Dental fluorosis:

Swarup and Dwivedi (2002) reported excessive intake of fluoride via water and food causes dental fluorosis and enamel defects in animal. They were suggested that the F interferes in the process of amelogenesis and dentinogenesis resulting in the formation of defective enamel and dentine in animals. Mascarenhas (2000) suggested, dental fluorosis was result of excessive intake of fluoride during teeth development. Shearer *et al.* (1978) observed dental lesions in animals characterised by hypomineralization of outer enamel, hyperplasia, hypoplastic pits, disrupted pigment band, puckered incremental lines, and decreased microhardness of the outer enamel. Shupe (1980) reported pitches and fine dots on the enamel surface of teeth, results loss of natural shine and early loss of the teeth. Wang (1987) and Choubisa (2015) were observed excessive abrasion, wearing of enamel and loss of teeth in goats living in F polluted industrial area. Wang (1992 and 2003) and Singh *et al.* (2002) were observed clinico-pathological signs of fluorosis in dogs with chronic fluorosis. Kilicalp *et al*., (2004) were 0 reported abnormal shape, size, colour, orientation and structure of dog teeth. They were also reported pitted incisors and abraded molars in fluorotic dog. Choubisa (1997) and Choubisa (2000) mentioned that the prevalence of dental fluorosis is relatively higher in calves than the adult bovines when both are reared in same F endemic areas. Choubisa (1998) reported 100% dental mottling in bovine calves and no sign in adult bovines when exposed in 4.7 ppm of F concentration in drinking water.

Choubisa, (2010) conducted a study in Bikaner district of Rajasthan and reported 33.3% of dental fluorosis in calves and 40% in cows. Similarly, Pati (2014) observed 79.5% prevalence of dental fluorosis in calves.

Choubisa (2008 and 2010) were reported mild to severe dental fluorosis in camels (*Camelus dromedarius*) living in fluoride affected areas.

Ray *et al.* (1993) reported, 91.02% of dental fluorosis in animals reared within 0.5 km of the aluminium smelter Odisha and suggested that the percentage of animals with severe dental fluorosis decreased with increasing distance from the smelter plant. Ray *et al.* (1993), Patra *et al*. (2000) and Maiti *et al.* (2004) were observed rough body coat, stunted growth, emaciation, decrease in milk production, unthriftiness, and chronic wasting in the fluorotic cattle. Choubisa *et al.* (2010) were reported dental fluorosis in horses and donkeys in Rajasthan.

Shupe (1980) conducted a survey study among domestic ruminants in Dungarpur district of Rajasthan and reported 48% cattle (*Bos taurus*), 55.9% buffaloes (*Bubalus bubalis*), 5.3% camels (*Camelus dromedarius*), 7.3% sheep (*Ovis aries*) and 10.7 % goats (*Capra hircus*) showed signs of dental fluorosis. Similarly, Choubisa *et al*. (2011) and Choubisa (2013) were observed varying degrees of dental fluorosis among cows and buffaloes of F endemic areas.

Skeletal fluorosis:

Farley *et al*. (1983) mentioned after their study that the excess fluoride accumulate in bones and stimulates proliferation of osteoblasts which gradually builds up the bones by increasing the uptake of calcium. Krook & Maylin (1979) reported common symptoms of skeletal fluorosis in animals are poor body condition, difficulty in movement, muscle wasting, painful, rigid and locked up joints, bony outgrowths and osseous lesions. They also reported skeletal fluorosis in developing foetus of cow when mother cow exposed to excess F during gestational period and advocated that fluoride could pass through placental barrier during gestational period. Similar observations were reported by Maylin *et al.* (1987) in calves which born from fluoride intoxicated cows showed severe symptoms of osteo-fluorosis such as abnormal differentiation in chondroblast and osteoblasts, bone marrow atrophy, stunted growth, lameness, snapping sound in feet during walking and bony lesions in mandibles, ribs, metacarpus and metatarsus regions. Choubisa (1999) observed similar changes in other domestic animals and concluded these changes are progressive, unrepairable and become more severe with advancing of age. Swarup and Dwivedi (2002) reported skeletal fluorosis sign in camels and in cattles found near the aluminium smelter. They were observed bony lesions in ribs, mandible, metacarpal, metatarsal and pelvic vertebrae, swelling of joints, lameness, bending of limbs, and deformed over growth of hooves in fluorotic cattle. Similar signs of skeletal fluorosis were also reported by Ray *et al.* (1993), Patra *et al*. (2000) and Maiti *et al.* (2004) in different animals or cattle under industrial F intoxication.

Kilicalp *et al*. (2004) and Kant *et al*. (2009) reported skeletal fluorosis after fluoride administration in dogs and goats respectively. Besides these several other workers earlier reported skeletal deformities caused by fluoride intoxication in different domestic animals such as in horses (Choubisa, 2010), donkeys (Choubisa, 2010), camels (Choubisa, 2010; Choubisa, 2011), goats (Swarup & Dwivedi, 2002; Choubisa, 2011; Choubisa, 2013; Modasiya *et al.* 2014), sheep (Choubisa, 2011), buffaloes (Choubisa, 2011).

Recently, Samel *et al.* (2016) reported painful walking due to periosteal exostoses at ligament & tendons, osteomalacia and osteoporosis in fluorotic animals. Sheikh and Panchal (2018) reported skeletal fluorosis in cows, goats and buffaloes of Udaipur, Rajasthan.

Non-skeletal fluorosis:

Toxic effects of fluoride on various organ systems are referred as non-skeletal fluorosis. Non skeletal fluorosis symptoms develops in animals before the onset of osteodental deformities . Singh and Swarup (1999) observed biochemical changes in serum and urine in fluorotic cows and buffaloes and recorded elevated levels of fluoride in urine and serum.

Patra *et al*. (2000) reported NaF toxicity produce deleterious effects in soft tissues such as gastrointestinal tract, lungs, heart, kidneys and liver of domestic animals. Shinde and Shinde (2006) stated that the ingestion of excess fluoride for a long period causes fluorosis and it affect multiple organ systems of the body which leads several clinical deformities. Kumar and Choudhary, (2015) reported general systems of chronic fluoride toxicity as colic, intermittent diarrhoea, polydypsia and polyuria in sheep and cattles.

Gastrointestinal toxicity of fluoride:

Generally fluoride compounds are easily soluble and quickly absorbed by digestive tract of animals. Swarup and Dwivedi (2002) were reported that due to highly electronegative property F show strong affinity with Ca ion in gastrointestinal tract it strongly bind with calcium ion and reduces its absorption results a significant decrease in serum calcium level in fluorotic animals. Similar changes in fluorotic cattles and buffaloes were also recorded by Maiti *et al.,* (2004) and Bharti *et al*. (2007). Choubisa (2010) reported discomforts, constipation, excess gas production, decreased appetite, abdominal pain and watery loose motion in calves

Hepatotoxicity of fluoride:

Araya *et al.* (1990) reported an elevated activities of serum transaminases, sorbitol dehydrogenase and decreased albumin and serum protein reflecting abnormal functioning of liver function in fluorotic animals. Similar observations in fluorotic goats were also observed by Tsunoda *et al.* (1985). According to WHO (2002) report liver play important role in metabolism and detoxification of foreign substances. These alterations in liver functioning correlated with toxicity of fluoride in domestic animals.

Renal toxicity of fluoride:

Kidneys play important role in removal of excess fluoride out from the body and in chronic fluorosis retention of fluoride in kidneys take place which causes severe damage in renal tubules and also form stones. Kessabi *et al.* (1985) observed kidney degeneration, glomular inflammation and tubular necrosis in fluorotic sheep. Shupe *et al*. (1992) and Kapoor *et al*. (1993) were reported similar changes in fluorotic cows in their experiment. Singh and Swarup (1994) reported elevated level of urea and creatinine in serum of fluorotic cows, buffaloes and goats. Singh & Swarup (1999) observed many degenerative changes in kidneys of fluorotic animals and reported elevated level of urea, nitrogen and creatinine in serum of fluorotic cows and buffaloes. Zhang *et al.* (2015) reported increased fluoride in serum of camels are due to fluoride exposure. Recently, Dharmaratne (2019) published a review study and stated that excess fluoride causes chronic kidney diseases (CKD) in human and animals.

Cardiovascular toxicity of fluoride:

Cummings and McIvor (1988) reported prolonged ingestion of high fluoride causes cardiac arrest which occur due to hypocalcemia or hyperkalemia that develop following exposure to high doses of fluoride because F is highly electronegative and bind strongly with calcium ions results hypocalcemia. Electrocardiogram changes along with a decrease in heartbeat were reported in dogs (Kilicalp *et al.*, 2004) and sheep (Donmez and Cinar, 2003). Deposition of F in vascular wall also increases the risk of cardiovascular changes in the affected animals.

Neurotoxicity of fluoride:

Choubisa (1999) and Choubisa (2007) reported that the cattle, buffaloes and other domestic animals living in fluoride affected areas for long time may developed para and quadriplegia, polyurea and neurological disorders. Hence the toxicity of fluoride gives rise to various neurological disorders in the animals.

Endocrine toxicity of fluoride:

According to Wheeler (1983) and Swarup & Dwivedi (2002) excess fluoride interfere normal functioning of thyroid, parathyroid and adrenal gland and its harmonal profile. Fluoride induced effects on reproductive organs, gametogenesis, embryogenesis and brain are also not well studied in domestic animals. Thus, it is observed that excessive intake of fluoride leads to malfunctioning of the endocrine organs in the animals, which further causes changes in their humoral profile.

Reproductive toxicity of fluoride:

Schoff & Lardy (1987) and Tanyildizi & Bozkurt (2002) were recorded adverse effects of F on sperm motility, sperm morphology, and semen hyaluronidase activity in an invitro studies on bovine semen. Ulemale *et al* (2010) and Choubisa (2012) studied toxic effects of F on reproduction and they have been reported significant increase in post calving anestrous and decreased fertility rate in cows receiving 8-12 ppm fluorine in their diet for a year. Choubisa (2011) recorded placental transfer of fluoride in cows and buffaloes during gestational period from fluorosed cattle to their faetus (calves) in fluoride endemic areas. Recently, Choubisa (2015) reported repeated abortions, still births and irregular estrous cycles in goats due to industrial fluorosis in Rajasthan.

CONCLUSION

On the basis of above findings and observations, it can be concluded that F is highly toxicity to domestic animals. Domestic animals received excess fluoride via fluoridated groundwater, fodder, soil, emission from various factories and mining of rock phosphate. This excess F reach in its body mostly by oral ingestion, absorbed by gastrointestinal tract passively and accumulated in all body parts mainly in hard tissues causes osteo-dental fluorosis, gastrointestinal disorders, impaired hepatic, renal and reproductive activities in livestock.

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