Environmental pollutants and livestock health: a review

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Received: 07/06/2013
Revised: 09/07/2013
Accepted: 11/07/2013

Abstract

Environmental pollutants always have hazardous impact on living organisms. Of all the pollutants, environmental pollutants stand first in affecting life-style of living entities throughout the globe. Environmental pollutants that adversely affect the health of livestock as well as human beings have been broadly categorized as heavy metals, pesticides, mycotoxins etc. Heavy metals like Lead (Pb), Cadmium (Cd), Mercury (Hg), Arsenic (As), Chromium (Cr) get deposited in the vital organs through the food chain. Their high concentration may have deleterious effect causing disorders such as minamata and itai itai by heavy metals Hg and Cd, respectively. Excessive use of pesticides in agriculture to increase crop productivity also contributes quantum of such substances in stable food materials. Moreover, the ubiquitous nature of mycotoxins producing fungi may act as potential contaminants of the environment poses adverse effects including immunosuppression. Necessary action should have to be taken to avoid hazardous situation which may arise in future for betterment of next generation.

Key words: Environmental pollutants, heavy metal, livestock health, mycotoxicosis, pesticide, toxicity

Introduction

More pollution is the direct result of more industrialization. Advent of industrial and for that matter agriculture revolution in India has added many pollutants in the environment which are potentially hazardous, some may be toxic, inflammable, explosive and even corrosive (Patra et al., 2005; Swarup et al., 2005). In last five decades there is many fold increase in environmental gases which are not the normal constituents of biosphere but were reported to be toxic to the all living organisms. Out of various pollutants present in the hydrosphere and lithosphere, heavy metals and pesticides for example are the most toxic to livestock as well as human beings (WHO, 2001). Due to pesticides, river water was found to be more polluted than lakes (Konstantinou et al., 2006) which are source of drinking water for animals and human also.

Heavy metals are categorized under environmental pollutants owing to their toxic effects on plants, animals and ultimately on health of mankind through food chain. Phyto-remediation is the process by which plant uptake heavy metal (Tangahu et al., 2011). Therefore, such plants are major source of heavy metals and causes toxicity in animals who consume these plants. Anthropogenic activities have been augmented due to industrialization and urbanization which contribute to the entry of toxic metals, gases etc. in food chain of animal and human (Kumar et al., 2007; Kumar et al., 2008). Chronic exposure of sulfhydryl reactive metals results in higher accum-
ulture in tissues, bones, hair and blood. It appears to be highly imperative to monitor these toxic metals in biological materials which indicate the status of environmental degradation (Patra et al., 2007). Some of the heavy metals Pb, Cd, Hg, As, Cr are cumulative poison; ores accumulate in food chain through the primary producer. These metals are also potent carcinogenic and mutagenic. The high accumulation of Hg and Cd caused minamata and itai itai diseases, respectively. Pesticide in general used to remove weeds, insect, molds, rodents (Aspelin, 1994). Synthetic pesticides have been popular among the farmers because of simple application, highly effective and economic returns in terms of high yield of healthy crops. They are hardly to be degraded as they are artificially synthesized. Continuous use of pesticides results in deposition of harmful chemicals in environment as residue. The amount of residues left depends on the nature of chemical, dose of application and prevailing weather condition (Raikwar et al., 2007). These types of residue are acts as a potent source of various diseases in livestock as well as human beings.

Mycotoxins are a relatively large, diverse group of naturally occurring secondary metabolites secreted by toxigenic fungi of genera Aspergillus, Penicillium and Fusarium. They are produced in cereal grains as well as forages before, during and after harvest, in favourable environmental conditions such as hot and humid climate. It is estimated that 25% of the world’s grain supply is contaminated by mycotoxins, annually (CAST, 1989). Inhibition of growth of mycotoxin producing fungi in animal feed/feedstuff or the amelioration or the detoxification strategy suggested as an important role for the prevention of mycotoxicosis in animals or human beings (Katole et al., 2011). There is plethora of literature available on individual environmental pollutants and their effect on animal health. It is desirable to compile major environment pollutants at one place. Therefore, present review highlights some environmental pollutants/toxicants and their adverse effects on animal health.

**Heavy metal toxicity**

Natural and anthropogenic activities such as industrial wastes discharge, mining, agriculture, household waste disposal and fuel combustion are the origin of heavy metals pollution. In animal physiological system, metals entered through feeds, green fodder, drinking water and contaminated air (fields alongside industries are major source). Other sources are accidental access to lime field, mineral supplements with high content of trace metal and licking of painted surfaced containing metallic pigments, empty containers of pesticides and synthetic colours (Patra et al., 2005; Burger, 2008). Animals had higher bioaccumulation rate from surrounding waters than from sediments or soils (Vrhovnik et al., 2013). The toxic limit and permissive level or safe intake of heavy metal for human health is given in Table 1.

Moreover, livestock as well as human beings are exposed concurrently or sequentially to multiple metals via several routes (through air, diet or drinking water) from different occupational and environmental settings and pose potential health hazard by their interaction (antagonistic or synergistic) which could be the critical modifiers for exerting the toxic effects (Jadhav et al., 2007).

**Arsenic (As):** The organo-arsenicals in food is one of the most common poisoning in livestock now days because of the displacement of arsenic from almost all phases of farming activities other than the factories which augment the present condition. The common source of arsenic is the chemical suspension used for spraying on animal to control ecto-parasites. Clinical signs of arsenic toxicity in cattle vary form gastrointestinal troubles to deadly nervous signs (Krajcovicová and Eschenroeder, 2007). Chronic arsenic toxicity is mostly manifested in weight loss, capricious appetite, conjunctivitis, mucosal and erythematic lesion including mouth ulceration, anemia, liver and kidney damage, hyper pigmentation, skin damage (keratosis) and black foot disease (Bahri and Romdane, 1991; Wu et al., 1989; ATSDR, 2000). Acute toxic effects include abdominal cramping, hyperesthesia in extremities, abdominal patellar reflexes and abdominal electrocardiogram (Franzblau and Lilis, 1989). Such effects generally occur at the levels of exposure equal to 50µg/kg weight/day. Recently, a study was undertaken in
India to evaluate an alternative source of arsenic in human food chain through livestock. This study concluded that consumption of egg, agricultural produces grown in arsenic contaminated soil and milk might causes arsenic toxicity (Datta et al., 2012).

**Lead (Pb):** The source of lead includes natural and anthropogenic processes such as combustion of coal and mineral oil, smelters, mining, alloy processing units, paint industries etc. Newly born animals are particularly susceptible to lead exposure due to high gastrointestinal uptake and the permeable blood brain barrier leading to neurotoxic effects even at low exposure (Allcroft and Blaxter, 1950). It is the most common industrial metal that has become widespread in air, water, soil and food and easily accumulates in the different vital organs especially liver, kidney, bones and brain of the body. Besides, direct ingestion of lead which causes increased blood lead levels, accumulated lead in the body also acts as a significant source of blood lead burden (Swarup et al., 2005). Higher levels of lead ingestion produce mainly acute or chronic poisoning. It causes decreased hemoglobin synthesis, impairment of neurobehavioral and psychological function, peripheral neuropathy and reproductive effects. Lead toxicity has been recorded in fishes, birds, dogs, goats, sheep, buffalo, cow etc. (Swarup et al., 1990; Sallam et al., 1999; Mousa et al., 2002; De Francisco et al., 2003; Liu, 2003; Phillips et al., 2004; Swarup et al., 2005; Alves et al., 2006; Balagangatharathilagar et al., 2006; Patra et al., 2007; Vellinger et al., 2012). There is a potential risk to human consumers of beef from the Pb polluted industrial areas where such livestock are raised (Rodríguez-Estival et al., 2012).

Although soils can contain appreciable concentration of lead in ethylene diamine tetra-acetic acid (EDTA) extractable form, lead is poorly taken up by plants and concentrations in pastures and crops rarely exceed 5 mg/kg dry matter (DM). The principal threat to livestock therefore comes from the soil and the vulnerable animals are those consuming soil while grazing or foraging on contaminated land.

**Mercury (Hg):** Mercury is considered as a highly toxic metal in the environments. The sources of mercury are plastics (vinyl chloride), chlor-alkali (in the manufacture of chlorine and caustic soda), electrical (electrical switches, batteries and fluorescent light) and electronic batteries. The effluents from these industries served as a source of mercurial contamination of water (Nicholson et al., 1983). Soil contains less mercury i.e. maximum 2.12 and avg. 0.09 mg kg DM. Pastures and crops also contain mercury in minute amount, fish meal is important source. Mercurous chloride intended for treatment of club routes in Brassica species has recently caused death of dairy heifers and topical administration of mercury containing drugs (Neathery and Miller, 1975). Soil erosion contributes to the Hg contamination (Tania et al., 2013).

The toxicity of mercury depends on its chemical form e.g. methyl mercury being the most hazardous metal and stable form of mercury which has been attributed to the suffering of most avian and mammalian predators at the top of contaminated tropics.

Table 1: Toxic level and permissive/safe intake of heavy metal (Oliver, 1997)

<table>
<thead>
<tr>
<th>Heavy Metal</th>
<th>Toxic Limit</th>
<th>Recommended intake / Safe intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic</td>
<td>3 mg/day for 2-3 weeks</td>
<td>15 – 25 µg/day (adults)</td>
</tr>
<tr>
<td>Cadmium</td>
<td>200 µg/kg of fresh weight</td>
<td>15 -50 µg/day adults</td>
</tr>
<tr>
<td></td>
<td>≥ 500 µg/L (Blood)</td>
<td>2 -25 µg/day children</td>
</tr>
<tr>
<td>Lead</td>
<td>20 - 280 µg/day adults</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10 – 275 µg/day children</td>
<td></td>
</tr>
<tr>
<td>Mercury</td>
<td>&gt;80 µg/day</td>
<td>43 µg/day</td>
</tr>
<tr>
<td>Zinc</td>
<td>150 µg/day</td>
<td>15 µg/day</td>
</tr>
<tr>
<td>Chromium</td>
<td>-</td>
<td>50-200 µg/day</td>
</tr>
</tbody>
</table>
Mercury passes through placental barrier and may cause intra-uterine death of foetus, foetal resorption and stillbirth. Fish containing more than 0.4 ppm Hg are unfit human consumption; the critical urinary concentration of Hg has been suggested as 1 to 2 µg/ml. Minamata disease is characterized by symptoms of fatigue, loss of memory concentration, tremors, constriction of visual field and cortical blindness (Jarup, 2003). The animal consumed high mercury containing vegetation will be affected and will suffer from alopecia, neuropathy visual and gastrointestinal tract disorder. Selenium and mercury is mutual antagonist and addition of selenium in the diet provides substantial protection against inorganic and organic mercury toxicity in poultry (Jagadeesan et al., 2007; Ekino et al., 2007).

**Cadmium (Cd):** It is a heavy metal grouped with Zn and Hg in the periodic table. It is very reactive, toxic and is a cumulative poison for animal and equally for human beings. Pure cadmium is a soft, silver-white metal. It is usually found as a mineral combined with other elements in the form of cadmium oxide, cadmium chloride, cadmium sulphate etc. Refined foods, water foods, water pipes, coffee, tea, coal burning and cigarette are all the most important source of cadmium (Patra, 2005; Kumar, 2007; Kumar et al., 2007; Kumar et al., 2008). Cadmium is sparsely distributed in agricultural ecosystem including the soil. Uptake by plants is poor and it is true particularly for clay soils. Occasionally, soils become enriched with cadmium from cadmium rich fertilizers or the dispersal of wastes from mining and smelting factories. Super phosphate fertilizers contain <5 to 134 mg Cd kg depending on country and region of origin. Sewage sludges contain variable and occasionally excessive cadmium concentrations (upto 20 mg/kg DM) (ASTDR, 1999).

Cadmium accumulated in the kidney and liver over long time. It is interacted with numbers of minerals mainly Zn, Fe, Cu and Se due to chemical similarities and competition for binding stage. It is also reported that Cd causes renal tubules damage, cardiovascular disease, cancer, osteomalacia and also affect calcium, phosphorus and bone metabolism. Sheep developed cadmium toxicity with Cd >40 mg kg DM and shows signs similar to zinc deficiency e.g. loss of appetite, poor growth, retarded testicular development, parakeratosis, paradoxically high Cd increases Zn requirements in sheep. Later, with less severe Cd containing feed and copper content barely adequate, sheep developed signs similar to deficiency of copper viz. anaemia, impaired bone mineralization, loss of wool crimp, low birth weight, abortion and still birth (Nicholson et al., 1983; Radostits, 2000; Wright and Baccarelli, 2007). A study from Ethiopia indicated that the levels of toxic metals such as Cd and Pb were beyond the acceptable limit in cow milk which can be a potential health concern for public health which consumes livestock products (Dawd et al., 2012).

**Chromium:** Factory wastes of stainless steel, leather tanning, explosives, ceramics, paint pigments, photography and wood preservatives are the main sources of chromium. Chromium has potent carcinogenic effects on livestock as well as human. Trivalent form of chromium is essential to livestock and human because it plays an important role in insulin metabolism. Chromium toxicity includes liver necrosis, nephrites, gastrointestinal irritation, ulcers (coetaneous, nasal and mucus membrane). Trace amount of chromium is important in glucose metabolism (Edwards and Gregory, 1991; ATSDR, 2000).

**Pesticide toxicity**

Pesticides are toxic xenobiotics which can be classified on the basis of different criteria such to use, chemical classes, mode of action and toxicity etc. Pesticides are of many types on the basis of their mode of action i.e. insecticides, fungicides, herbicides, algacides, disinfectants, rodenticides, antimicrobes, nematocides pheromones, repellents etc. Chlorinated hydrocarbons are neurotoxicants and damage the nervous system of the pest organisms; the organophosphorous insecticides and carbamates exert their toxicity by inactivating the vital acetyl cholinesterase enzymes (Gallo and Lawryk, 1991). There are three possible ways by which pesticides can enter into the animal body. The possibility of
contamination of milk and milk product with Dichloro Diphenyl Trichloroethane (DDT) and Benzene Hexa Chloride (BHC) are mainly through feed and fodder (Battu et al., 1996; Mukherjee and Gopal, 1993). Pesticide also used in large scale to kill ectoparasites like ticks, mites, insect which normally present on the body of animal. Some time drinking water may also be a source of contamination (WHO, 1984).

Pesticides have an affinity for lipid material and are not biodegradable, accumulate in animal systems, such pesticides are the main cause of contamination of animal products like milk, meat, egg, plants (feeds and fodders), fruits etc. (Gupta et al., 1997; Raikwar et al., 2007) and as well as human milk (Gupta et al., 2001). The residues of these pesticides in animal products are decline at very slow rate, even after sources of contamination are eliminated (Martínez-Haro et al., 2008). Dhaliwal and Kalra (1977) and Karla et al. (1978) was the first who reported contamination of bovine milk with organochlorine pesticides particularly with DDT and hexachlorocyclohexane (HCH) in 1960’s and 1970’s in India from area of Punjab and Delhi. Maximum residues limit (mg/kg or mg/l) of organochlorine pesticides for milk and milk products recommended by IDF 9701 (1997) are lindane (0.01 and 0.5), DDT (0.05 and 1.25), aldrin (0.006 and 0.15), endosulfan (0.004 and 0.1), dialdrin (0.006 and 0.15), endrin (0.0008 and 0.02), hexachlorobenzene (0.02 and 0.5), heptachlor (0.006 and 0.15), methoxychlor (0.02 and 0.5) and chlordane (0.002 and 0.05). Some important pesticide and their ill effects are described below.

**Acute effects:** The short-term toxicity of a chemical, manifested over a period of hours or days, is referred to as its acute toxicity. Acute toxicity typically is expressed as the concentration required for killing 50 per cent of a population of test animals such as laboratory rats either through ingestion or through contact with the skin. These lethal concentrations can vary greatly from one pesticide to another and this also depends on class of pesticide used.

**Chronic effects:** Pesticides are more likely to cause chronic health effects that occur long after repeated exposure to small amounts of pesticides in feed items. Examples of chronic health effects include cancer, liver and kidney damage, disorders of the nervous system, damage to the immune system, and birth defects. Evidence relating to chronic health effects to specific drinking water contaminants is limited. In the absence of exact scientific information, on the basis of their chronic toxic effects, organic chemicals are grouped into the three major classes viz. carcinogens, mutagens and teratogens.

**Dichloro Diphenyl Trichloroethane (DDT):** DDT has been one of the best known, cheapest and probably one of the most effective of the synthetic insecticides. DDT was used extensively during World War II in control of lice and other insects by direct application to soldiers. DDT affects the permeability of potassium ions, reducing its transport across the membrane. DDT alters the porous channels through which sodium ions pass - these channels activate (open) normally but once open are inactivated (closed) slowly, thereby interfering with the active transport of sodium out of the nerve axon during repolarization. The functional injury produced by high doses of DDT is usually related to its effects on the central nervous system. Major symptoms are instability, dizziness, disturbed equilibrium, tremor and convulsions. There is little pathogenic change in cells and the tissues of the central nervous system in acute case of poisonings. The locus of primary toxic action of DDT is believed to be sensory and motor nerve fibers and motor cortex. Humans and animals exposed to high oral doses of DDT show symptoms of perithecia (burning, pricking, tickling sensation) of the tongue, lips, and face. Apprehension, hyper-susceptibility to external stimuli, such as light, touch, sound; irritability; dizziness; vertigo; tremor and tonic and chronic convulsions (Joachim et al., 2001; Eskenazi et al., 2008). Liver and reproductive organs (testes) are commonly injured organs by DDT.

**Endosulfan:** Endosulfan is an organochlorine insecticide and acaricide, and acts as a contact poison in a wide variety of insects and mites. Endosulfan is effective against a wide range of insects and certain mites found on cereals, coffee,
cotton, fruit, oilseeds, potato, tea, vegetable and other crops. Short-term toxicity is high, and influenced by the solvents and emulsifiers used to dissolve it. The stomach easily absorbs endosulfan, by the lungs and through the skin, means all routes of exposure can pose a hazard. Exposure to endosulfan may result from, for example breathing of air near spraying on crops, drinking water which is contaminated, eating contaminated food, touching contaminated soil, smoking cigarettes made from tobacco with endosulfan residues or working in an industry where endosulfan is used. It enhances the effect of estrogens and acts as an endocrine disruptor, causing reproductive and developmental damage in animals and human beings; and it also reported to cause cancer and immunosuppressive during experimental conditions (Gupta and Gupta, 1979; Sutherland et al., 2004; Singh et al., 2007a, b; Kumar et al., 2011; Singh et al., 2011).

**Permethrin:** It is a synthetic pyrethroid insecticide and is neurotoxin. It causes acute toxicity in animals and human. The US Environmental Protection Agency has classified it as a human carcinogen and it has been shown to cause immune system damage as well as birth defects (Macan et al., 2006). Pyrethroids are highly toxic to fish, crustaceans and bees.

**Mycotoxicosis**

More than 350 mycotoxins identified in nature, the important mycotoxins that pose the greatest potential risk to human and animal health as food and feed contaminants are aflatoxin B₁ (AFB₁), ochratoxin A (OTA), citrinin (CTN), T-2 toxin and fumonisin B₁ (FB₁) (Asrani et al., 2013). Aflatoxin B₁ is the most common and more toxic than others (AFB₂, AFG₁, AFG₂); and mainly produced by *Aspergillus flavus* and *A. parasiticus*. AFB₁ is primarily hepatotoxic, also affects kidneys and other organs including lymphoid organs which led to immunosuppressive effects in animals and human beings. OTA mainly produced by *Aspergillus ochraceus*, occurs more abundantly, often with AFB₁ in feed and is the most toxic. OTA is primarily nephrotoxic (nephropathy), secondarily hepatotoxic and also affects other systems in animals and human beings (Gowda and Ledoux, 2008; Patial et al., 2013). Inhibition of mycotoxin in animal feeds is the art of prevention of various factors which encourages the growth of this fungus (Katole et al., 2011).

CTN is a metabolite of toxic strains of *Penicillium citrinum* and is often encountered together with OTA (Pittet, 1998). Kidney appears to be the primary site of action (Kumar et al., 2012). T-2 toxins, an important member of trichotheccene mycotoxins and mainly produced by *Fusarium sporotrichioides*. It causes human alimentary toxic aleukia (Beardall and Miller, 1994) and affects liver, kidney, heart, lung, gastrointestinal tract and skin in animals. Fumonisins are produced by *Fusarium verticillioides*. Six types of fumonisins have been reported and FB₁ is the most toxic to animals and birds (Khan et al., 2012). It causes a fatal disease of horses i.e. equine leukoencephalomalacia (ELEM) and porcine pulmonary oedema (PPE) in pigs (Galvano et al., 2001).

**Acute toxicity:** Ruminants are relatively resistant to mycotoxicosis, due to active metabolism by the ruminal microflora. In general, young animals are more sensitive than adults. Clinically, acute toxicity include initial anorexia, weight loss and retching followed by tenesmus, elevated rectal temperature, bilateral purulent conjunctivitis, tonsillitis, polydypsia, polyuria, passage of clots of blood-stained mucus from the rectum, dehydration, prostration and finally death (Moon et al., 2007).

**Chronic toxicity:** In animals, chronic toxicity occurs due to consumption of mycotoxin contaminated feed for a long duration and affects target organs i.e. liver and kidneys as well as nervous system, gastrointestinal tract, lymphoid organs, skeletal system, haematopoietic tissues and reproductive organs (Atroshi et al., 2000; Ozcelik et al., 2004). Clinically, reduced growth rate, impaired coagulation of blood resulting in congestion and wide spread haemorrhages, hypoproteinaemia, loss of carotenoid pigments, glycogen accumulation/depletion in liver, decreased skeletal integrity, anaemia, leucocytopenia, altered glycogenesis, altered intestinal fragility and impaired phagocytosis are observed during chronic exposure to various
mycotoxins (Cavin et al., 2007; Gowda and Ledoux, 2008; Asrani et al., 2013).

**Immunosuppression:** Mycotoxins are potent immunosuppressive agents. Several mycotoxins are cytotoxic to lymphocytes *in vitro*, perhaps because of their effects on membranes (including those involving lymphocytic receptors) or interference with macromolecular synthesis and function (Minervini et al., 2005). OTA causes immunosuppression following prenatal, postnatal and adult life exposure. Degeneration and necrosis of B and T lymphocytes and plasma cells in primary (thymus, bursa) and secondary (spleen, lymph nodes, Peyer’s patches, caecal tonsils, hardener gland) lymphoid organs, cause depression in humoral as well as cell mediated immune responses (Dwiwedi and Burns, 1985; Minervini et al., 2005; Oswald et al., 2005; Satheesh et al., 2005; Bryden, 2007; Kumar et al., 2008), leading to susceptibility to secondary infections and vaccination failure.

**Carcinogenicity:** AFB1 causes primary hepatocellular carcinomas of liver in various experimental animals as well as in human being (Ellis et al., 1991; Fujimoto et al., 1994). Oral administration of OTA produced tubular cell carcinoma of the kidneys in males and female rats and fibroadenomas of the mammary gland in the females. It has been suggested that OTA can cause testicular cancer in human (Schwartz, 2002; Hof, 2008).

**Genotoxicity:** Various *in vitro* studies indicate that several mycotoxins such as AFB1, OTA, FB1 and T-2 toxin have genotoxic effects. These include chromosomal aberration, micronucleus formation, sister chromatid exchange, gene mutation, abnormal DNA damage and repair, unscheduled DNA synthesis and DNA adducts formation in various *in vivo* and *in vitro* test systems in the laboratory animals (Zeljezić, 2006; Domijan et al., 2007; Patil, 2007).

**Teratogenicity:** In rodents, AFB1 caused gross anomalies like enlarged and abnormal head, external anophthalmia, open eyelids, protruded tongue, micromelia and ear abnormality. Skeletal defects found are incomplete ossification of skull bones, and agenesis of sacral and caudal vertebrae, sternaebrae, metacarpals and metatarsals bones. The visceral anomalies included internal hydrocephalus, microphthalmia, cardiac defects and multilobulation of liver (Wangikar, 2003).

OTA having high affinity towards the developing nervous system mainly affects central nervous system, the eye and the axial skeleton system in the offspring’s of OTA treated females. In rodents, OTA causes gross anomalies like exencephaly, incomplete closure of skull, gastroschisis, syndactyly, micromelia, hypognathia, haematomata, scoliosis, curled tail, protruded tongue and external anophthalmia. Skeletal anomalies, such as, incomplete ossification of skull bones, wavy, fused and branched ribs, agenesis of sacral and caudal vertebrae, sternaebrae, metacarpals, metatarsals and ischium bones and visceral anomalies such as internal hydrocephalus, microphthalmia, hydrenephrosis, cryptorchid testis and dilated renal pelvis (Wangikar, 2003; Patil et al., 2006; Patil, 2007; Pfohl-Leszkowicz and Manderville, 2007).

In rats, CTN causes decreased foetal body weights and crown to rump lengths. Moreover, the foetal malformations such as internal hydrocephalus, cerebellar hypoplasia, microphthalmia, contracted and notched kidneys, multilobulated liver, dilated renal pelvis, incomplete ossification of skull bones, rib anomalies and sacral and caudal vertebrae agenesis were also observed (Singh et al., 2007a)

**Dioxin toxicity**

Dioxins, most toxic chemicals known, are a serious public health hazards. Dioxin and polychlorinated biphenyls (PCBs) are toxic pollutants released by industries that present everywhere and persistent in the environment and may contaminate feeds, particularly herbage which finally graze by livestock and enter the body tissue of animals. Animal products are the primary source of dioxins to human beings (ATSDR, 1989; Schecter et al., 2006). Recently, Wright et al. (2013) described the factors that most likely to influence the physical impacts of microplastics such as accumulation and translocation and trophic transfer of microplastics in aquaculture.
The major source of dioxin for the human population is the food of animal origin which feeds on forage near industrial areas. Since dioxin is fat-soluble, it bio-accumulates by climbing up the food chain and mainly (97.5%) through animal products viz. dairy products, milk, chicken, pork, fish and eggs etc. Major food includes fish oil, fish meal, recovered vegetable oil, grease and many byproducts from food industry, bleaching earths, kaolinitic clays and milk products (Delistraty and Stone, 2007; Kulkarni et al., 2008).

Most dioxins are byproducts of chemical reactions used to make the herbicides 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T); the wood preservative pentachlorophenol; and the antibacterial agent hexachlorophene. Dioxins have been found in the fly ash of municipal incinerators, formed in the burning of wastes containing chlorine (ATSDR, 1989; Mandal, 2005). While all dioxins have been found to be toxic in animal studies, one in particular, 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin (TCDD) is referred to even in the scientific literature - as “one of the most toxic man-made compounds known”. TCDD is fat soluble and resistant to metabolic breakdown and biodegradation.

The most common health effect in people exposed to large amounts of dioxin is chloracne. Chloracne is a severe skin disease with acne-like lesions that occur mainly on the face and upper body. Other effects of exposure to large amounts of dioxin include skin rashes, skin discoloration, excessive body hair, and possibly mild liver damage. One of the main concerns over health effects for dioxins is the risk of cancer in adults. Several studies suggest that workers exposed to high levels of dioxins at their workplace over many years have an increased risk of cancer. Animal studies have also shown an increased risk of cancer from long-term exposure to dioxins. Research on animals indicates that chronic exposure to dioxins might results in reproductive and/or developmental defects (ATSDR, 1999; Wiberg et al., 2007, Kulkarni et al., 2008).

**Fluorine toxicity**

Water, soil and plants with naturally high fluorine (F) content are usually the main cause of fluorosis. The most important effects are seen in the wild and domestic animals that are exposed for long periods to excess fluorine due the industrial pollution (WHO, 1984; Gu, et al., 1990). In a number of small villages in Sicily, Turkey and India there is naturally occurring fluoride in the water ranging from 0.7 to 5.4 ppm. The villagers and their livestock are chronically ill, while neighboring villages with no fluoride have no such illnesses. Lower milk production has been described amongst cattle. Cows which were exposed to inorganic fluoride in drinking water at concentrations of 5, 10 or 12 mg F/kg produced significantly fewer calves than the controls. This effect preceded the development of clinical symptoms of fluorosis in domestic as well as wild animals (Shupe et al., 1972).

In case of animals, dental fluorosis is generally characterized by the presence of various enamel defects and lesions such as mottling, hypoplasia, hypocalcification and increased wear. Mottled and defective enamel is believed to be solely an indication of inorganic fluoride exposure during the development of the teeth. Other symptoms of the disease are lameness, hyperostosis and exostosis of the long bones, tendon calcification, and a systemic effect on ration intake, usually have not been seen until the ration contains in excess of 40 ppm F (Suttie, 1980).

Chronic toxic effects of fluoride (F) as osteo-dental and non-skeletal fluorosis were reported in 99 domesticated cattle (Bos taurus) from Chani village, Bikaner district of Rajasthan which is a part of Thar Desert in India. Fluoride in drinking water sources e.g. bore wells of this village varies between 1.5 and 2.5 ppm. Out of 24 calves (<2 years age) and 75 cows (>3 years age), 10 (41.7%) and 28 (37.3%), respectively, exhibited mild to severe dental mottling (Choubisa et al., 2012).

**Conclusion**

Based on above findings by the various researchers across the globe, it is concluded that environmental pollutants whatever form may be, have an adverse impact on health and well being of livestock as well as human beings and it is a matter of serious concern. Concerted efforts
should be diverted to address this burning issue and should be solved by negotiations between policy makers, researchers and non-governmental organizations. This will in long-term secure the safe future for livestock as well as human health.

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