EFFECT OF SOME CHEMICAL POLLUTANTS ON ANIMAL HEALTH.

Thesis Presented
By

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DEDICATED TO

MY MOTHER,

MY FATHER

AND

MY BROTHERS AND SISTER

CERTIFICATE OF APPROVAL

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Abbreviations

Abbreviation	Item
ALP	= Serum alkaline phosphatase.
ALT	= Serum alanine aminotransferase.
AST	= Serum aspartate aminotransferase.
ATP	= Adenosine tri-phosphate.
Ca 10(Po$_4$) F$_2$	= Fluoro-apatite.
CaF$_2$	= Fluor-spar.
Cd	= Cadmium.
CK	= Creatine kinase.
EPA	= Environmental Protection Agency
F	= Fluoride.
H$_2$S	= Hydrogen sulfide.
H$_2$SO$_4$	= Sulfuric acid.
MT	= Metallothionein.
Na$_2$S$_2$O$_3$	= Sodium metabisulphite.
NRC	= National Research Council
PEM	= Polioencephalomalcia.
S	= Sulfur.
(-SH)	= Sulfhydryl group.
SO$_2$	= Sulfur dioxide.
SO$_3$	= Sulfur trioxide.
TISAB	= Total Ionic Strength Adjustment Buffer.
WHO	= World Health Organization

1. INTRODUCTION

Toxic metals are natural components of the environment, but human activities, notably industrial and agricultural development, have been largely responsible for wider diffusion of elements. Toxic metals are accumulated in soils, plants and animals fed with these plants will tend to accumulate toxic metals themselves. In terms of potential adverse effects on animal; fluorine, sulfur and cadmium, are amongst the elements that have caused most concern. This is because they are readily transferred through food-chains. Sub-lethal exposure to these elements can result in adverse effects on a variety of physiological and biochemical processes.

Cattle fluorosis has been observed in several parts of the world as a consequence of high fluoride concentration in rock phosphates, water resources and brick manufacture ***(Clarke and Clarke, 1975).*** One of the most aggressive forms of fluoride intoxication is related to the intake of dust containing fluoride.

Sulfur and sulfur compounds- sulfates and sulfur containing amino acids are nontoxic to ruminant, but these are converted to more toxic ions, such as sulfides, by rumen microflora. Inhalation of hydrogen sulfide from diets high in sulfate has been implicated as a potential cause of sulfur toxicosis in ruminants ***(Pandher, 2000).***

Interest in cadmium poisoning has grown because of the increased contamination levels of cadmium in some feeds and foods of plant or animal origin ***(Blanusa and Juresa, 2001***). Cadmium emissions have increased

dramatically during the 20th century, one reason being that cadmium-containing products are rarely re-cycled, but often dumped together with household waste. Recent data indicate that adverse health effects of cadmium exposure may occur at lower exposure levels than previously anticipated, primarily in the form of kidney damage but possibly also bone effects and fractures ***(Jarup, 2003).*** Therefore, measures should be taken to reduce cadmium exposure in the general population and animal farms in order to minimize the risk of adverse health effects.

The need to reduce toxic metal contamination in animal feed in fact posses a significant problem for agricultural regions located in more or less industrialized areas in which animals are reared on locally produced feed. There are many such regions worldwide, including the regions of *Kafr El-Zayiat* and *Kom Hamada* in *Gharbia* and *Behera* provinces, respectively Egypt, where animals graze year-round on pastures very close to industrial emissions.

Knowledge levels of toxic metals in livestock are important for assessing the potential effects of pollutants on domestic animals themselves and in quantifying contaminant intake in humans. Although contamination of animals feed by toxic metals cannot be entirely avoided given the prevalence of these pollutants in the environment, these a clear need for such contamination to be minimized, with the aim of reducing both direct effects on animal health and indirect effects on human health.

Due to the persistence complain of population, our study aimed to:

- Evaluate the contribution of industrial pollution on Kom Hamada and Kafr El-Zayiat localities.
- Asses the toxic metal levels in the different biological fluids and tissues of cattle and buffalo.
- Explain the biochemical changes and possible clinical toxic manifestations of fluoride, sulfur and cadmium in farm animals.
- Clarify the secondary effects on the metabolites of related essential elements (calcium and inorganic phosphorous) in the animal body.
- Elucidate their possible toxic effects on the hepatic and renal function tests.

2. REVIEW OF LITERATURE.

1. Fluoride (F) Toxicosis

Fluoride intoxication has been observed in most countries, usually in association with natural or industrial hazards. Chronic fluorosis in man, livestock and wildlife was identified as an important toxicologic problem in several parts of the world because of spreading with the increase and expansion of certain industries into agricultural areas ***(Choubisa, 1999).***

1.1. Sources of Fluoride Pollution:

Fluoride is the most electro-negative element, found in both igneous and sedimentary rocks and constitutes about 0.06-0.09 % of the upper layer of earth crust. Fluoride rarely occurs free in the nature, because of its chemical reactivity, but combines chemically to form fluorides, fluorite, fluor-spar (CaF_2) and fluoro-apatite [Ca $10(Po_4)$ F_2] that widely, but variably distributed in soils, water, atmosphere, vegetation and animal tissues ***(NRC, 1980)***. Because of this variation, the sources and their relative importance for animals also vary. The most commonly encountered sources of excessive fluoride are:

1.1.1. Water-borne Fluoride:

Chronic fluoride intoxication, or fluorosis, is a worldwide health problem and is endemic in those areas where the fluoride content is high in drinking water. Contamination of the drinking water with 2 ppm of fluoride resulted in a significant increase in the quantity of fluoride in the enamel and dentine of the teeth. An increase in the fluoride content of the bones was found when fluoride was present in the drinking water ***(NRC, 1974).***

Griffith-Jones (1977) reported that a high incidence of arthritis in dairy cows disclosed elevated fluoride levels in bone samples because of water contamination by industrial fall-out. As well, ***Abd El-Aal (1981)*** and ***Ibrahim (1983)*** documented that the water pollution by the sewage discharge of the super-phosphate factory is considered the main source of fluorosis where the fluoride concentration of the drinking water at the Manqabad village, Assiut was more than 2 ppm.

Recently, the consumption of water contaminated by the fumes and dusts emitting from the industrial sources resulted in the development of chronic fluorotic lesions in the different animal species where the fluoride concentration in the drinking water is varied between 1.5-4 ppm ***(Dwivedi et al., 1997; Choubisa, 1999*** and ***Patra et al., 2000).*** Generally, the upper safe limit of fluoride in the drinking water for livestock is about 2 mg/L, but it may not protect animal health from fluorosis as reported by the ***NRC (1980).***

1.1.2. Feed-stuff Pollution:

The interest expressed in the past, in dairy and beef cattle producers, for fluorides as toxic agents has been directed toward the possible pollution of forages by industrial effluents containing fluoride. In addition, toxicant could appear from the supplementation of grain rations by fluoride bearing phosphate ingredients ***(Phillips et al., 1960).*** The effect of ingestion of soluble fluorides by dairy cattle as one of the important sources of fluoride pollution have been studied by ***Harris et al. (1964)***

Most reviews on the fluoride problem in livestock production indicated that the normal forages contain less than 10 ppm fluoride and a publication

by ***Merriman and Hobbs (1962)*** indicated that normal fluoride values are probably 5-10 ppm for legumes and about half of this for grasses. Also, ***Gordon and Tourangeau (1977)*** suggested a single standard of 20 ppm fluoride, air-dried basis, for all fodder. However, it must be noted that soils and fertilizers also contribute to the fluoride content of fodder. ***Suttie (1969b)*** reported that "some rather high fluoride forages (112 ppm) can be found in areas with no known source of industrial fluorides..." Thus, regulations that attempt to control the level of fluoride in fodder by restricting airborne industrial emissions may prove inadequate.

Standards controlling the fluoride content of fodders also fail to provide protection against high fluoride levels in mineral supplements and other types of feed ***(Obel, 1971; Griffith-Jones, 1977*** and ***Hillman, 1977).*** Furthermore, ***Eckerlin et al. (1986)*** reported that the fluoride is often introduced into livestock through the feed with mineral supplements.

NRC (1980) recorded that the maximum tolerable levels for young, mature dairy, mature beef and finishing cattle are set at 40, 40, 50 and 100 ppm, respectively. The prolonged intakes of dry diet fluoride concentrations above these maximum tolerable levels may result in reduced performance, in spite of the small intake of fluoride may be beneficial, or even essential.

One of the greatest difficulties is that practically every natural water supply and foodstuff contains traces of fluoride and it is almost impossible to prepare fluoride-free (e.g. < 0.005 ppm) control diets adequate in other respects ***(NRC, 1974).***

1.2. Toxicokinetics of Fluoride:

The primary mode of entry of fluoride into the animals' body is through the digestive tract; as cattle develop fluorosis by feeding on pasture or drinking water contaminated with fluorides, via inhalation of fluoride vapor and particulate but the direct inhalation of fluoride emissions does not contribute significantly to fluoride accumulation in animal's body ***(Shupe, 1971*** and ***NRC, 1980),*** and in extreme cases of acute exposure, through the skin. Dermal absorption of fluoride has only been reported in case of burns resulting from exposure to hydrofluoric acid ***(Burke et al., 1973).***

In the industrial environment, the respiratory tract is the major route of absorption of both gaseous and particulate fluoride. Hydrogen fluoride being highly soluble in water is rapidly taken up in the upper respiratory tract ***(Dinman et al., 1976).***

Fluoride is a general protoplasmic poison; its exact mechanism of action is not clearly examined. Soluble inorganic fluorides that was ingested through water and foods are almost completely absorbed and also those from the respiratory tract. But absorption of less soluble inorganic and organic fluorides varies from 60-80 % ***(NRC, 1980).*** Fluorides are absorbed from the GIT probably from the rumen in cattle and sheep ***(Perkinson et al., 1955)*** by a process of simple diffusion without any mechanism of active transport being involved ***(NRC, 1980).*** Observations show that after absorption from the gut fluorides enters the circulation. About 75% of the fluoride in the blood is in the plasma ***(Carlson et al., 1960)*** with 15-70% (0.01-0.04 ppm) in ionic form ***(Singer and Armstrong, 1964)*** and nearly 5% of plasma fluoride is bound to protein.

Plasma fluoride concentrations tend to increase slowly over the years. It is seen that plasma levels of fluoride do not fluctuate widely and maintained within narrow limits despite a wide variation of fluoride levels in drinking water presumably because of the action of some regulatory mechanisms involving skeletal and renal tissues ***(Smith et al., 1950; Singer and Armstrong, 1964*** and ***NRC, 1980)***. The sequestration of fluoride into the skeleton, urinary excretion and loss sustained through sweat help in regulation of plasma fluoride. The normal cattle have blood levels of up to 0.2 mg fluoride per 100 ml of blood ***(Radostits et al., 1994***). Approximately 99% of the fluoride in the body is localized in the skeleton. The rest is distributed between the blood and soft tissues.

Fluoride has a remarkable affinity for bone and teeth even at low levels of fluoride intake which incorporates it into hydroxyapatite to form fluoroapataite. The amount of fluoride stored is more readily in the active, growing and cancellous bones within limits over a period, without inducing any pathological evidences. However, in some cases of high fluoride intake structural bone changes occur ***(Shupe et al., 1963b)***. The osteofluorotic lesions may be porosis, sclerosis, hyperostosis, osteophytosis, and malacia, depending on the interacting factors influencing the degree of fluorosis ***(Shupe and Olson, 1971)***.

Most soft tissues do not accumulate much fluoride, even during high intakes, although tendon ***(Armstrong and Singer, 1970)***, aorta ***(Ericsson and Ullberg, 1958)***, and placenta have higher fluoride concentrations than other soft tissues, possibly associated with their relatively high levels of calcium and magnesium. Kidneys usually exhibit a high fluoride

concentration during high-fluoride ingestion due to urine retained in the tubules and collecting ducts ***(NRC, 1980).***

Not all of the fluoride that is ingested or inhaled is absorbed, and a proportion is excreted by various means ***(WHO, 1984).*** Fluoride present in feces results from tow sources; the ingested non-absorbed fluoride and the absorbed fluoride that is excreted into GIT. About 10-25% of daily intake of fluoride is excreted in the feces. The elimination of absorbed fluoride occurs almost exclusively via the kidney. Alternatively, ***Phillips et al. (1955)*** reported that the normal excretion of fluoride in cattle via urine is normally less than 5 ppm. Milk fluoride concentrations are affected only minimally by dietary fluoride, and ***Dirks et al. (1974)*** found that normal cow's milk contains 0.087 to 0.132 ppm reached to 0.287 ppm in exposed farms.

Krook and Maylin (1979), Crissman et al. (1980) and ***Maylin et al. (1987)*** found a clearly transplacental transmission of fluoride from the cow to the fetus (congenital fluorosis) with a slight, but gradual, increase in fetal bone fluoride with greater fluoride intake by the mother. While, ***Radostits et al. (1994)*** stated that fluoride does not pass the placental barrier nor does it occur in the colostrum of milk in appreciable amounts so that the newborn infants are not exposed to danger of intoxication until they begin to drink water. Recently, ***Shupe and Bagley (1992)*** concluded that, in cows, a partial placental barrier to fluoride limits concentrations in fetal circulation and tissues.

1.3. Toxicological Features and Diagnosis of Fluorosis:

Chronic fluoride toxicosis which most detected in livestock is most commonly refereed to as fluorosis; "Gaddur" in Iceland ***(Roholm, 1937)***, "Slobbers" in Australia ***(Hard and Atkinson, 1967)***, and "El-Darmous" in North African Coast and Morocco ***(Kessabi et al., 1983)***, a general term that includes osteofluorosis and dental fluorosis.

The diagnosis of fluoride toxicosis is difficult, because there are an extended interval between the ingestion of elevated levels and the appearance of toxic signs ***(Shupe, 1970)***. Dietary history, clinical evidence, radiography, chemical analysis, necropsy and biopsy findings, and histopathology were all important. The degree of dental fluorosis, osteofluorosis, evidence of intermittent lameness, and the concentration of fluoride in diet, urine, serum, and bone were of particular importance ***(NRC, 1980)***.

1.3.1. Clinical Toxic Signs:

1.3.1.1. Dental Fluorosis:

Dental fluorosis is usually diagnosed in animals by clinical examination of the incisor teeth. Cheek teeth are also important in evaluating the effects of fluorosis, but difficult to be examine in live animals ***(Shupe and Olson, 1971*** and ***Griffith-Jones, 1977)***. The fluorosis score of the incisor teeth in cattle is one of the most frequently used criteria for the diagnosis of excess exposure to environmental fluoride ***(Suttie, 1969a)***.

Dental fluorosis affected only the developing permanent teeth during the formative stage, and the subsequent abnormalities in the permanent

dentition are the most obvious sign of the ingestion of increased concentration of fluorides in the ration, while the permanent teeth that fully formed and erupted before ingestion of excess fluoride show neither lesions nor deleterious effects ***(Shupe et al., 1963b*** and ***Shearer et al., 1978).*** Because the developing enamel is sensitive to excess concentration of fluorides, the enamel changes serve as a constant and permanent index, reflecting levels over normal ***(Garlick, 1955).***

The effects of excess fluorides on the teeth are permanent brown or even black staining and mottled in the form of chalky-white like spots, patches, striations or more diffusely due to incomplete enamel calcification and/or poorly developed interprismatic substance ***(Griffith-Jones, 1977, Krook and Maylin, 1979*** and ***Maiti et al., 2003).*** There's also attrition or uneven and excessive wearing of the fluorotic deciduous teeth with pitting or hypoplasia of enamel ***(Araya et al., 1993; Radostits et al., 1994; Jones et al., 1997*** and ***Maiti et al., 2003),*** bulging of the gingiva and delaying or oblique eruption of permanent teeth ***(Krook and Maylin, 1979 and Krook et al., 1983).***

1.3.1.2. Skeletal Fluorosis:

Lameness and unthriftness were the first symptoms that to be noticed by farmers ***(Kerur, 1971*** and ***Radostits et al., 1994)*** and the chief clinical signs but inconclusive measures of fluorosis in the affected animals ***(Shupe and Olson, 1971*** and ***Araya et al., 1990).***

Clinically, the lameness is transitory, shifting from leg to leg and intermittent showing a marked seasonal incidence recurring in summer

months ***(Suttie et al., 1961, Shupe and Olson, 1971*** and ***NRC, 1980).*** The lamed animal suffer from joint stiffness, painful gait, movement with short steps, dragging of fore or hind limbs while walking, arching back, and raising tail while the head and neck carried well down. The affected animal may recumbence or standing on the carpases for long period and reluctance to eat or graze causing secondary emaciation and drop in milk yield and forcing the culling of large numbers of cows ***(Suttie et al., 1957*** and ***1961; Kerur, 1971; Griffith-Jones, 1977; Araya et al., 1990; Jubb et al., 1993; Singh and Swarup, 1994; Dwivedi et al., 1997; Choubisa, 1999*** and ***Patra et al., 2000).*** The pervious signs occur because of arthritis due to exostosis formation under tendon insertions and periarticular structures ***(Shupe and Olson, 1971*** and ***Griffith-Jones, 1977)*** or pedal bone fractures in sever cases of lameness ***(Jubb et al., 1993).*** All of these occur in animals of any age especially older ones.

The lameness and stiffness were affecting fore- and hind-limbs equally but most marked in the loins, hip joints and hind legs ***(Griffith-Jones, 1977*** and ***Choubisa, 1999).*** Inconsequence, toe and claws of sound legs were severely misshapen, showing deformities and overgrowth because of bearing weight for long period of time ***(Griffith-Jones, 1977; Eckerlin et al., 1986*** and ***Dwivedi et al., 1997).***

The disease is mainly characterized by clinical signs of a marked skeletal abnormalities; osteofluorosis can eventually develop if excessive fluoride is ingested over a prolonged period of time and include distorted skull giving a "Roman nose" effect ***(Suttie et al., 1961*** and ***Shupe and Olson, 1971),*** visible and palpable painful (bilateral) bony exostosis and

callus formation usually develop first on the medial surface of metatarsals, subsequently were seen on metacarpals, ribs, forehead, mandibles, sternum and phalanges ***(NRC, 1980; Araya et al., 1990; Raghib et al., 1993; Singh and Swarup 1994; Dwivedi et al., 1997*** and ***Patra et al., 2000).*** Subsequently, spontaneous fractures can occurred easily, notably of the ribs.

1.3.1.3. General Health Condition or Non-skeletal Signs:

The general health of the animal is not necessarily affected when mild dental changes are the only sign of fluorosis, but in sever cases the signs appear indirectly by the reduced food intake, including unthriftness, anorexia, emaciation, stunted growth, loss of body weight, inappetence, excessive salivation with shedding of poorly masticated food, dry hair, rough coat, reduced milk production and fat content, increment in the post-calving anoestrus and inability of animals to bear weight of the bull during mating with subsequent decline in fertility on further exposure ***(Stoddard et al., 1963; Ammerman et al., 1980*** and ***Radostits et al., 1994).*** Digestive and respiratory troubles were reported in some animals with clear dental fluorosis ***(Raghib et al., 1993).***

A mild degree of anemia and a decrease in erythrocytic activity of bone marrow is found in fluorosis. On the other hand, a sever anemia may occur, although this is not a constant sign, as a result of associated nutritional factors ***(Hobbs et al., 1954*** and ***Hoogstratten et al., 1965).***

1.3.2. Laboratory diagnosis:

1.3.2.1. Fluoride Estimation:

The abnormally high levels of fluoride in the serum, urine and bone provide strong additional support for an osteofluorosis diagnosis ***(Araya et al., 1990).*** Many methods are available for determination of fluoride and the most widely used involve colorimetery or the fluoride specific ion electrode.

1.3.2.1.1. Serum Fluoride Estimation:

Blood is known to be a very labile pool of fluoride as stated by ***Suttie et al. (1972)*** whose data revealed a close positive relationship between the concentrations of fluoride in plasma and fluoride intake. The plasma fluoride concentrations of cattle receiving 3 mg/kg for 4 months raised to near 1 ppm, followed by sever dental fluorosis in at least one pair of incisors. ***Suttie and Faltin (1973), Griffith-Jones (1977), Shehata et al. (1989), Karram and Ibrahim (1992), Botha et al. (1993)*** and ***Dwivedi et al. (1997)*** supported these results in different animal species.

Radostits et al. (1994) reported that the higher levels of fluoride in the storage capacity of organs reflected on plasma fluoride values and also reported that the normal cattle have blood levels of up to 0.2 mg fluoride per 100 ml of blood. Cattle on fluoride intakes sufficient to cause intoxication may have blood levels of 0.6 mg/100 ml, although the blood levels are often normal.

Fluoride levels were increased in the plasma, urine and bone in affected dairy cows as a result of rock phosphate addition to the ration ***(Shlosberg***

et al., 1980), in sheep *(Abd El-Aal, 1981)* and buffaloes *(Ibrahim, 1983)* raised near to the super-phosphate factory, in sheep *(Samal and Naik, 1992)* and Friesian cows *(Raghib et al., 1993)* in the vicinity of an aluminum factory depending upon the distance from the source of pollution and the period of exposure.

1.3.2.1.2. Urinary Fluoride Estimation:

Shupe et al. (1963a) discussed the use of urinary fluoride as an estimator of fluoride intake in cattle. In addition, *Burns (1970)* suggested that the fluoride content of urine considered as an index of fluoride ingestion by cattle. This index might be advantageous because of the ease of sample collection, but the relation between fluoride intake and urinary fluoride is not well established. Measurements of the fluoride concentrations of urine are important practical criteria for the dental and skeletal fluorosis *(McDowell, 1985; Ergun et al., 1987* and *Walton, 1988).* Also, *Shehata et al. (1989)* concluded that the urine is more indicative in case of fluoride toxicity and the results of *Patra et al. (2000)* studies on cattle and buffalo indicating that the body burden of fluoride and the urinary elimination pattern were similar irrespective of external lesions.

Moreover, ruminants are more sensitive to the fluoride toxicosis than the other animals *(Ammerman, 1980* and *McDowell 1985).* Normal cattle have a urine fluoride concentration of about 2 to 6 ppm. Cattle exhibiting moderate fluorosis have urine fluoride concentration of about 15 to 20 ppm. Cattle with urinary fluoride concentration 40 ppm or greater could be suspected of ingesting a diet with fluoride concentration of 60 ppm or greater *(Shupe et al., 1963ab).*

Furthermore, ***Radostits et al. (1994)*** concluded that the fluoride content in urine must be above 10 ppm to indicate current consumption of fluoride in damaging amounts and cattle on fluoride intakes sufficient to cause intoxication may have urine levels of 16-68 ppm, although the blood levels are often normal. Urinary fluoride excretion may remain sometimes or for some months elevated, following a change of diet from high to low fluoride ration, because of fluoride mobilization from skeletal tissues ***(Suttie et al., 1972).*** Therefore, such high levels may not are an indication of high intakes immediately preceding the examination ***(Radostits et al., 1994).***

In addition, ***NRC (1980)*** reported that the urinary fluoride levels are roughly correlated with dietary intake, although the duration of ingestion, sampling time, and total urinary output will introduce variation. Nevertheless, ***Vandersmissen et al. (1993)*** stated that there is no obvious difference in the fluoride concentration of urine samples taken from cattle at different times of year, and they concluded that the measured urinary fluoride levels were indicative of industrial pollution.

Recently, ***Füdanci and Sel (2001)*** found that the higher fluoride ion concentration levels in sheep urine samples (7.86 ± 0.77 ppm) collected from the surroundings of Coal-Burning power station were indicative of presence of chronic industrial fluorosis and reported significant seasonal effects on the urine fluoride ion concentrations.

1.3.2.1.3. Soft Tissues Fluoride Estimation:

Because of the small increase caused by fluoride ingestion (1 to 2 mg F/kg body weight /day), ***Suttie et al. (1961)*** stated that the soft tissues fluoride concentration was unreliable as a measure of the degree of fluorosis. In addition, ***Shupe and Olson (1971)*** reported that only small amounts (< 2.5 ppm) of fluoride were retained in soft tissues. Therefore, hair, skin, hooves and soft tissues do not evidence any significant pathognomonic changes. Unlike fluoride in bone, the concentration does not increase with age or duration of exposure, because of the great sensitivity of bone to fluoride and to efficiency with which they protected by bone deposition and urinary excretion, the determination of fluoride levels in the blood or soft tissues are of limited diagnostic value ***(Underwood, 1971).*** General sings of toxicity and metabolic disturbances thus appears in tissues at the time once bone is saturated and as bone lesions develop ***(Radostits et al., 1994).***

Furthermore, ***U.S. EPA (1980)*** recorded no increase in fluoride concentrations in soft tissues could be found in cattle with a high fluoride intake, severe dental fluorosis, and a very high level of bone fluoride. Whereas, the data obtained by ***Ibrahim (1983)*** revealed a highly significant elevation of fluoride levels in buffaloes' muscle and kidneys in the polluted areas. In ***1989, Shehata et al.*** observed absence of any significant changes in fluoride level in the meat and organs of camels.

1.3.2.1.4. Hard Tissues Fluoride Estimation:

Measurement of bone fluoride content is quite helpful and considered one of the most important aids in diagnosing cases of endemic fluorosis as a

quantitative index of exposure to fluoride, even under condition where the level of exposure has varied considerably ***(NRC, 1971*** and ***Suttie et al., 1972)***, which allows the determination of the extent of bone fluoride retention and could be used for the management of fluoride treatment of osteoporosis.

For monitoring of live animals, this would require inconvenient biopsies of ribs or coccygeal vertebrae; but in farm herds, post-mortem samples from slaughtered animals are often available ***(Burns and Allcroft, 1962; Suttie, 1967*** and ***NRC, 1971).*** Mandibles were found to contain the highest levels of fluoride than metatarsal bones, while the lowest concentration was observed in the incisory teeth ***(Mortenson, et al., 1964)***. Fluoride content in cancellous bone such as ribs, vertebrae and pelvis is greater than in cortical compact metacarpals and metatarsals, even after 6 to 10 years of exposure. In addition, fluoride concentration may vary in different areas of the same bone. The metatarsus and metacarpus are commonly analyzed for fluoride content. For all practical and clinical purposes, fluoride concentration is equal in either of these bones from the same patient ***(Shupe et al., 1963b; NRC, 1974; Griffith-Jones, 1977*** and ***Krook and Maylin 1979),*** but the fluoride concentration of the fourteenth coccygeal vertebrae (ash basis) is approximately twice that of the metatarsus (dry, fat-free basis) ***(Suttie, 1967).***

Schmidt et al. (1954) and ***Hillman et al. (1979)*** recorded that the fluoride deposition in bone and cartilage was found to be the most reliable indicator of the levels of the ingested fluoride. Analysis of metacarpal bones of newborn calves demonstrated a slight placental transfer of fluoride. In

addition, ***Krook and Maylin (1979)*** found fluoride in bone ash of a 7-month old fetus exceeded 500 ppm; fluoride thus was passed transplacentally. Analysis of fluoride in ash of bones obtained at necropsy of cattle from 4 months to 4-5 years of age showed increased amounts with age. Fluoride accumulations were severing in the bone ash and the anatomical changes were advanced even in young and mature animals. Cancellous bone retained far higher amounts than cortical bone. Concentrations exceeding 10000 ppm fluoride were recorded in cancellous bone of a 4-5 year-old cow. Furthermore, ***Crissman et al. (1980)*** reported that the ash fluoride in a stillbirth calf was 280 ppm and in oldest cattle 2800; the fluoride is transmitted transplacentally and that the increase was significantly correlated to age.

Suttie et al. (1961) and ***NRC (1971)*** stated that bone fluoride levels in "the range of 4500 to 5500 ppm (dry, fat-free basis in long bones such as metacarpal or metatarsal) might be considered as the marginal zone of fluoride toxicosis and those levels below this were not harmful". While, ***Mortenson et al. (1964)*** observed that normal levels were up to 1200 ppm and but may be increased to 3000 ppm in animals exposed to fluoride and showing only mottling of the teeth. Animals showing sever clinical signs have levels greater than 4000 ppm of bone.

Obel and Erne (1971) observed serious fluorosis in calves with 500 to 2400 ppm, and in cows with 900 to 2800 ppm fluoride in metacarpal bone ash (assuming 60% ash, these figures correspond to 300, 1440, 540 and 1680 ppm, dry fat-free basis, respectively). Records of ***Shupe et al. (1992)*** indicated that over 170 cattle were exposed to dietary fluorides levels in

excess of 25 ppm (dry wt), for most of their life span, and these animals exhibited bone fluoride concentrations ranging between 2000 and 12500 ppm (dry wt). Chemically bones of appreciably poisoned animals contain fluoride to the extent of 4000-15000 ppm (1.5%) ***(Jones et al., 1997).***

1.3.2.2. General Blood Biochemistry:

The effects of chronic fluorosis on serum biochemical parameters were discussed in overwhelming papers. ***Poey et al. (1976)*** have reported that the early stages of chronic fluoride intoxication are associated with changes in blood and urine components, and that these precede radiologically detectable bone abnormalities. In the early phase, there was an increase in blood urea and acid phosphatase, with a concomitant increase in urinary output of phosphorus and urea. As the fluoride intoxication progressed, there was a gradual impairment of urinary creatinine clearance, leading to renal insufficiency.

Fluorides are known to activate and inhibit enzyme system. At low levels, fluorides are known to stabilize and activate several enzymes and at higher levels, fluorides inhibit enzymes such as adenyl cyclase and pyrophosphatase ***(Hodge et al., 1970).*** The alkaline phosphatase (ALP) is often found to elevate in fluorotic cases, which may be due to increased turnover rather than any specified effect of fluoride on the enzyme ***(Rosenquist, 1974).*** The serum and bone (ALP) activity was significantly increased directly proportional to the fluoride concentration of the bone as observed by ***Nielsen et al. (1973)*** and ***Frada et al. (1974)***. There is a significant correlation between the amount of fluoride fed and the concentration of serum (ALP) which is probably related to the abnormal formation of bone as reported by ***Radostits et al. (1994)***.

The effects of chronic fluorosis on serum biochemical parameters of cattle and goats in the Darmous area, an intensive phosphate mining area, of Morocco were studied. Cattle showed increases in potassium, urea, gamma-globulins, lactate dehydrogenase, alkaline phosphatase and aspartate aminotransferase, whereas calcium, total proteins and albumin were lowered ***(Kessabi et al., 1983)***. Furthermore, in Turkish sheep ***Sel and Ergun (1992)*** reported an increase in the serum aspartate and alanine aminotransferases and decreases in lactate dehydrogenase, while those of alkaline phosphatase showed little change.

The studies of ***Araya et al. (1990)*** and ***Botha et al. (1993)*** on cattle revealed an inversion in the albumin/globulin ratio and the alkaline phosphatase; aspartate aminotransferase and alanine aminotransferase activities were elevated. In addition, ***Wang Jundong et al. (1992)*** found that the serum alkaline phosphatase was increased in goats due to industrial fluoride pollution. ***Abd El-Hamid and Dorra (1993)*** and ***Seddek et al. (1997)*** recorded hepatic dysfunction (higher lipoprotein content, triglycerides and lower enzyme activity) and increased alkaline phosphatase activity in fluorotic chickens.

An increased serum alkaline phosphatase activity at higher levels of fluoride intake or intoxication in different animal species had been reported earlier by ***Gregory (1996), Jagadish et al. (1998), Liangx Feng (1999)*** and ***Kapoor et al. (2001)***. Alkaline phosphatase, aspartate aminotransferase and alanine aminotransferase enzyme activities were found to be significantly higher in sheep serum samples collected from the surroundings of Coal-Burning power station ***(Füdanci and Sel, 2001)***.

1.3.2.3. Correlation between Serum Fluoride and Other Minerals:

Benetato et al. (1970) and ***Suketa and Mikami (1977)*** found that urinary excretion of inorganic phosphate, calcium, magnesium, potassium, and sodium increased significantly with increasing fluoride in the diet of rats. ***Henrikson et al. (1970)*** found that, in osteoporotic bones of dogs, fluoride caused a slight decrease in calcium content, and an increase in phosphorus content of bone ash. ***Miller et al. (1977)*** reported a higher calcium and somewhat lower phosphorus content in bones of cows suffering from osteoporosis due to environmental exposure to fluoride. ***Shearer et al. (1978)*** showed hypermineralized calcium content in fluorotic bovine teeth.

Griffith-Jones (1977) reported that bovine herd affected by industrial fluorosis had normal serum levels of calcium. These later finding was supported by ***Radostits et al. (1994)*** who recorded the serum calcium and phosphorus were usually normal.

Abd El-Aal (1981) during his study on the effect of industrial fluorosis on sheep found a decreased serum calcium levels, while serum inorganic phosphorous levels was increased, these result only confined to one area, while other areas showed normal values. Nevertheless, ***Ibrahim (1983)*** recorded a significant decrease in calcium, phosphorous and copper in serum samples of all investigated buffaloes raised in the vicinity of super-phosphate factory. Similarly, urinary calcium and phosphorous had showed significant elevation. In soft tissues phosphorous was lowered in muscle, liver, lungs, and heart. ***Wang Jundong et al. (1992)*** recorded a decrease in serum calcium level of goats pastured in an area severely polluted by industrial fluoride.

Sulfur (S) Poisoning

Sulfur is considered one of the most important pollutants emitted from some industrial factories and induces harmful effects to livestock.

2.1. Sources of Sulfur Toxicosis:

Sulfur oxides attracted the major attentions due to their common occurrence and their harmful effects at higher concentrations. According to ***Clarke and Clarke, (1975)*** hydrogen sulfide gas is often present in the gases emitted from oil and natural gas wells in cesspools and in wells functioning imperfectly for disposal of manure from a slatted floor system. The gas may be formed from sulfur within the gut or from sulfate in the rumen, although in the latter case there is no evidence that it is produced in amounts sufficient to be toxic ***(Lewis, 1954).*** While, ***Olkowski, (1997)*** recorded that the ruminal H_2S gas can cause an acute systemic response, if it is in sufficient amounts. However, in situations of moderate excess dietary sulfur, the gas accumulated in the rumen and low levels of eructated gas is inhaled and can contribute to chronic aspects of sulfur toxicosis pathogenesis.

Parker, 1977 stated that the major sulfur containing compounds in the atmosphere are sulfur dioxide (SO_2), sulfur trioxide (SO_3), hydrogen sulfide (H_2S), sulfuric acid (H_2SO_4) and sulfate (SO_4) salts. He added that SO_2 is derived from the combustion of fuel that contain sulfur, and from the smelting of non-ferrous metals in industrial countries. Emission of SO_2 pollutes air and reacts with some substances to form harmful compounds that precipitated in the soil affecting plants, animals and human being.

Ibrahim (1983) and ***Sayed (1987)*** recorded that S, SO_2 and SO_3 that emitted from the super-phosphate factory during the process of H_2SO_4 production, pollute and affect areas around the factory at Manqabad district, Assiut governorate, Egypt. ***Kandylis (1984)*** discussed poisoning of animals with sulfur from industrial emissions (SO_2) and proposed that the effect of excess sulfur intake might be secondary to the effect of sulfide on ruminal and intestinal motility.

In some instances the sulfate source is water ***(Harris, 1987),*** and in others the sulfate or other sulfur forms is a dietary component ***(Raisbeck, 1982; Gibson et al., 1988; Jeffrey et al., 1994*** and ***Hill and Ebbett, 1997).***

Gooneratne et al. (1989) mentioned that the excessive intake of sulfur was primarily related to the concentration of sulfur in the feeds and/or waters with particular involvement of later. There after, ***Bulgin et al. (1996)*** reported that high dietary levels of either elemental sulfur or sulfur in other forms can be toxic to ruminants. ***Olkowski (1997)*** concluded that the excessive dietary sulfur may be originated from industrial sources as power practices and fertilizer waste products of animal industry. In areas where sulfur content of soil and water is higher, the uptake of sulfur by plant may be increase and cause accumulation of sulfur in these plants. So, grazed animals in these areas receive excessive sulfur which is transferred through the food chain to human beings.

2.2. Toxicokinetics of Sulfur:

Sulfur is an essential nutrient for ruminant, and the microbial population in the rumen. At the recommended dietary level (0.2 – 0.3%),

sulfur is constantly recycled between free and combined forms ***(Andersen, 1978).*** The recommended and maximum tolerable dietary concentration of sulfur is 0.15% and 0.4% on a dry matter basis, respectively ***(NRC, 2000)*** and a level greater than 0.4% sulfur is toxic ***(Beke and Hironaka, 1991).***

After ingestion, sulfur compounds - sulfates and sulfur containing amino acids - are non-toxic to ruminant, but these are converted to more toxic ions, such as hydrogen sulfide which is the highly toxic form of sulfur, by the rumen microflora ***(Beauchamp et al., 1984).***

Sulfide is readily adsorbed through the rumen wall and more efficiently in the small intestine into the blood stream ***(Bray, 1969).*** Once absorbed, it is converted into sulfate in liver and is re-circulated into the rumen through saliva and blood. Sulfide ions inhibit the functions of cytochrome oxidase system, adversely affecting oxidative metabolism and production of ATP ***(Short and Edwards, 1989).*** Neurons are extremely sensitive to ATP depletion and rapidly undergo necrosis. In ruminant, as much as 60% of eructed gases are inhaled and enters the respiratory tract. Thus, inhalation of H2S from diets high in sulfate has been implicated as a potential cause of cerebrocortical necrosis or what's called polioencephalomalacia, PEM ***(Bulgin et al., 1996).*** In addition, they reported that sulfides also bind to hemoglobin creating sulfhaemogolbin, reducing the ability of the blood to carry oxygen to tissues. Furthermore, excess dietary sulfur can interfere with the absorption of other elements, particularly copper and selenium ***(NRC, 2001).*** The urinary excretion is the main route of sulfur excretion from the body. About 49% of sulfur intake is excreted in urine of sheep ***(Kulwich et al., 1957).***

2.3. Toxicological Features and Diagnosis of Sulfurosis:

The toxicity of sulfur is dependent upon its form and route of exposure. Whereas elemental sulfur is considered one of the least toxic elements, H_2S rivals cyanide in toxicity ***(NRC, 1993).***

2.3.1. Clinical Toxic Signs:

The acute sulfur poisoning in cattle and sheep is manifested by depression of animals, colicy pain, unwilling to stand, fast and shallow breathing beside smell of H_2S gas and increased body temperature up to 40.5 °C. Apparent diarrhea was characterized by blackish coloration ***(McFarlene, 1952*** and ***White, 1964).***

Anemias, achromotrichia, neonatal ataxia, loss of wool crimp and bone fractures were described by ***Underwood (1962)*** in sheep suffering from sulfate poisoning.

Weeth and Capps (1972) reported that heifers consumed 2814 ppm sulfate were anemic, emaciated and showed reduction in weight gain with increased level in blood methaemogolobin and sulfhaemogolbin. In addition, ***Twort et al. (1974)*** recorded diarrhea in animals consumed polluted water by sulfate or as a result of atmospheric pollution in the industrial areas.

In horses exposed to high level of SO_2, ***Spiegelman et al. (1968)*** recorded a decreased rate of lower lung clearance and a slowing mucous transport in the upper bronchi and trachea. ***Clarke and Clarke (1975)*** reported sever respiratory and circulatory disturbances. They added that the clinical signs in horse and cows are purgation, dullness, pale and dirty

mucous membranes and weak rapid pulse. ***Corke (1981)*** recorded an outbreak of sulfur toxicosis in horses due to error in feeding of 0.2 – 0.4 kg per animal.

Ibrahim (1980 and ***1983)*** mentioned that the toxic signs of chronic sulfurosis in sheep, goats and buffaloes reared near in the vicinity of super-phosphate factory were in the form of emaciation, rough easily detached hair and wool, dehydrated skin, paleness of nasal and conjunctival mucous membranes, often enteritis and some of animals were suffered from respiratory distress.

Kandylis (1984), Gunn et al. (1987) and ***Zinn et al. (1997)*** stated that toxicology of sulfur in ruminant includes toxicity, neurotoxic effect and mechanism of toxic action of H_2S. They reported the effect of excessive intake of sulfur by ruminant on feed intake, animal performance, ruminal digestion and motility, rumination and other physiological functions. The manifestations of high concentrations of sulfates in drinking water include decreased feed and water consumption ***(Weeth and Hunter, 1971*** and ***Weeth and Capps, 1972)*** and dietary sulfur intake includes diarrhea, reduced feed intake and growth rate, and death ***(Bulgin et al., 1996*** and ***Loneragan, et al., 2001).***

Naturally occurring and experimentally induced sulfur intoxications with hydrogen sulfide, sulfates or sulfides are a cause of "Polioencephalomalacia" ***(Raisbeck, 1982; Gooneratne et al., 1989,*** and ***Rousseaux et al., 1991).*** A nervous condition affecting ruminants, especially young (sheep, weaning to 18 months and cattle, three months to

a year) and characterized clinically by a brief period of disorientation or ataxia, aimless wandering with central blindness and head pressing progressing to intermittent hyperaesthesia and opisthotonos. The condition is a major cause of animal mortalities under feedlot management system ***(Olkowski, 1997; Gould, 1998; Niles et al., 2000; Pandher, 2000*** and ***Haydock, 2003).***

Selim et al. (2000) examined farm animal grazed in Ezbet Shokry, Kaliopia province near Abo Zabal super-phosphate factory as a source of sulfur and fluoride pollution, at distance 100 meters approximately. The affected animals showed signs of reduced appetite, decrease in weight gain, lower milk yield, severe emaciation, loss of general health condition with dental and skeletal lesions. Biochemical parameters revealed highly significant increase of sulfur in goat and sheep.

2.3.2. Laboratory Diagnosis:

2.3.2.1. Sulfur Estimation:

2.3.2.1.1. Serum Sulfur Estimation:

Serum sulfate values are considered a thermometer of sulfur intake through sheep ration ***(Starks et al., 1954).*** The level of inorganic sulfate in sheep serum varies according to the animal intake of sulfur. The amount of serum sulfate is increased by addition of methionine to the ration ***(Weir and Rending, 1954).*** The serum sulfur level in dairy cows was also elevated by increased uptake of inorganic sulfate ***(Bouchard and Gonard, 1973).***

Ryssen and Stielau (1980) reported that the increase of sulfur in ration from 2.9 to 4.0 gm per sheep per day resulted in significant increase of blood sulfur level.

The level of serum sulfur in the sulfurosed sheep, goat, cattle and camel is inversely related to the distance from the super-phosphate factory ***(Ibrahim, 1980, 1983*** and ***Sayed, 1987).***

2.3.2.1.2. Urinary Sulfur Estimation:

Correlation of the urinary sulfur and inorganic sulfate intake was proved by ***Starks et al. (1954)*** that sulfide could not be detected in the blood while a large portion of the dose was excreted rapidly by the urinary pathway. The urinary excretion is the main route of sulfur discard from the body as confirmed by ***Kulwich et al. (1957)***, where about 49% of sulfur intake is excreted in urine of sheep. Similarly, in dairy cows the urinary sulfur was elevated by increased uptake of inorganic sulfate ***(Bouchard and Gonard, 1973).***

Urinary sulfur level was considered as a diagnostic tool of industrial pollution by sulfur toxicity in animals reared adjacent to super-phosphate factory ***(Ibrahim, 1983*** and ***Sayed, 1987).***

2.3.2.2. General Blood Biochemistry:

Gooneratne et al. (1989) studied the effect of high dietary sulfur supplementation on blood chemistry in sheep. They found that there was increase in plasma creatine kinase (CK) and aspartate aminotransferase (AST) in all animals. Also, ***Jeffrey et al. (1994)*** found a higher serum

creatine phosphokinase activity in sheep ingested ammonium sulfate. Wherever, ***Madej et al. (1994)*** stated that excess sulfur intake to cows did not cause any significant changes in blood metabolic indicators. Alkaline phosphatase and serum transaminases levels were very high due to certain hepatic troubles of a toxic nature ***(Statov et al., 1994).***

In addition, ***Olkowski (1997)*** studied the biochemical changes associated with excess sulfur intake in ruminants. He found that serum creatinine and urea levels were moderately elevated in some animals. Aspartate aminotransferase and creatinine phosphokinase activities were increased substantially in severely affected animals. Serum gamma glutamyltransferase level did not appear changed.

In the study of ***Selim and Amany (2000)***, the donkeys grazed near to super-phosphate factory suffered from chronic sulfur and fluoride poisoning showed a significant increase in the ALP activity.

Recently, ***Sayed (2001)*** revealed an increase in serum enzymatic activity of ALT and urea values more than normal levels in goats reared near the super-phosphate factory. While serum AST and ALP activities and creatinine values were within the normal physiological limits.

2.3.2.3. Correlation between Serum Sulfur, Calcium and Inorganic Phosphorous:

Ewes suffering from sulfur poisoning have magnesium and phosphorous levels higher than normal with some hypocalcaemia ***(White, 1964).*** In addition, feeding sulfate ration contained 100 ppm of copper

elevates urinary calcium level that leads to lowered body calcium and increased phosphorous retention ***(Goodrich and Tillman, 1966).***

Ibrahim (1980) reported that there was a highly significant decrease in serum phosphorous level in sheep with high concentration of serum sulfur. Also he observed a significant decrease in calcium level in animals in the vicinity of super-phosphate factory. ***In 1983,*** his study on the effect of super-phosphate factory by-products on Egyptian buffaloes revealed a significant decrease in serum phosphorous and calcium concentrations.

Madej et al. (1994) observed only moderate decrease of phosphorous in cows exposed to excess sulfur intake. Furthermore, ***Olkowski (1997)*** added that the retention of calcium and phosphorous was reduced by addition of sulfate to the diet of ruminants.

Industrial chronic sulfur and fluoride poisoning in donkeys ***(Selim and Amany, 2000)*** and in goats ***(Sayed, 2001)*** was associated with a significant decrease in serum calcium in both species while serum phosphorous levels increased in donkeys and within the normal physiological levels in goats.

3. Cadmium (Cd) Poisoning

Cadmium is a modern non-essential toxic metal to which humans are exposed through a variety of foods, particularly leafy vegetables, grains and cereals ***(Sapunar-Postruznik et al., 1996*** and ***Blanusa and Juresa, 2001)*** or through tobacco ***(Goyer, 1995).*** By the same ways, ***Srebocan et al. (1991)*** reported that animals are exposed to cadmium.

3.1. Sources of Cadmium Toxicosis:

The industrial uses of cadmium are the largest sources of environmentally hazardous amounts of cadmium. The points at which pollution is most apt to occur begin with mining and smelting, followed by manufacturing, loss from manufactured products during use and when discarded, and the reclamation and use of waste products contaminated with cadmium. The air, water and soil provide pathways by which cadmium may be dissipated and enters animal and human either directly or via the food chain. Numerous aspects of these problems have been reviewed by ***Fleischer et al. (1974)*** and ***Friberg et al. (1979).***

Urban sewage sludge contains significant amounts of cadmium. Use of high cadmium sludges and mine drainage for fertilizing animal croplands has been shown to increase substantially the cadmium content of animal feeds ***(Council for Agriculture Science and Technology, 1976*** and ***Mason, 1991).*** Some phosphate fertilizers can also contain significant amount of cadmium ***(NRC, 1980).*** Most forages and plant materials feed to animals contain levels of cadmium will below 0.5 ppm on a dry weight basis ***(Underwood, 1977 and Baker et al., 1979)*** so that cadmium toxicosis is not a concern.

Underwood (1977) reviewed the effects of pollution on cadmium content of animal feed. Pollution has been shown to increase the cadmium of mixed pasture herbage by more than 40-fold. ***Johnson and Eaton (1980)*** stated that non-ferrous mines represent a major source of cadmium release to the environment. In addition, ***Davis (1984)*** reported that the main source of cadmium pollution are the combustion of gasoline, utilization of cadmium containing pesticides, phosphate fertilizers and industrial wastes.

Also, ***Oronsaye and Brafield (1984)*** stated that heavy metals including cadmium are released into aquatic environment by industrial concerns such as mining and plating processes. In ***1989, Abd El-Massih*** noticed that 20% of El-Max gandoufly samples contain higher levels of cadmium than the standard limits attributing that to the area overcrowding with the Petro-chemical industries and oil pipes.

Deposition of cadmium on forage from industrial and automobile emissions is the source most likely to intoxicate farm animals and horses ***(Swarup et al., 1997; Farmer and Farmer, 2000*** and ***Casteel, 2001)***.

3.2. Toxicokinetics of Cadmium:

Cadmium is not known to have any beneficial effects, but can cause a broad spectrum of toxicological and biochemical dysfunctions ***(Funakoshi et al., 1995)***. According to ***Khan-Dawood and Satyaswaroop (1995)***, cadmium is a ubiquitous environmental pollutant entering the body via food, water and air.

Less than 1% of dietary cadmium is absorbed by ruminants ***(Neathery et al., 1974).*** In human, gastrointestinal absorption of cadmium is about 5 to 8 %. Absorption is enhanced by dietary deficiencies of calcium and iron and by diets low in protein. Low dietary calcium stimulates synthesis of calcium-binding protein, which enhances cadmium absorption. Respiratory absorption of cadmium is greater than gastrointestinal absorption and independent on solubility of cadmium compound, but it ranges from about 15 to 30 per cent ***(Klaassen, 2001).***

There's no haemostatic control mechanism to limit cadmium absorption and retention below a non-toxic threshold levels. In cows, the cadmium retained by the gastrointestinal tract appears to represent primarily the fraction that's most rapidly cleared from the body. This phase takes about 4 to 12 days in cows ***(Miller et al., 1968).*** In addition, there was little transfer of cadmium across the placenta ***(Neathery et al., 1974),*** markedly limited transfer via the mammary gland into the milk and intestinal metallothionein (MT) binds cadmium tightly limiting its absorption ***(Sharma et al., 1979).*** Cadmium is transported in blood by binding to red blood cells and high-molecular weight proteins in plasma, particularly albumin ***(Klaassen, 2001),*** then left the blood rapidly and accumulated in the liver, kidney, gastrointestinal mucosa, salivary glands, pancreas, adrenal, thyroid and spleen ***(NRC, 1980).***

Neathery et al. (1974) stated that concentration of cadmium in tissues was in decreasing order as follow; kidneys, liver and small intestine. About 50 to 75 per cent of the body burden of cadmium is in the liver and kidneys. Also, ***Zasadowski et al. (1999)*** cited that cadmium is a

cumulative toxicant in the continental ecological cycling; it accumulates mostly in the liver and kidneys.

Cadmium is very similar in structure to essential nutritional metal ions, but it is not an essential nutrient, even in minute amounts. Chronic exposure to this very toxic heavy metal will produce an indirect form of toxicosis as it has a negative influence on hundreds of enzymatic systems of cells as it will substitute for essential metal ions, primarily zinc, copper and iron in metallothionein. Cadmium binds to metallothionein very tightly, which competitively decrease the absorption of copper, and to a lesser extend absorption of zinc. Liver and kidneys contains metallothionein that accumulate cadmium through out the life of animals ***(Kägi et al., 1974)***.

Also, cadmium has a strong affinity for biological structures containing (-SH) groups. As a result, the indirect effect on multiple cellular and enzymatic systems will produce many negative effects on the health of plants and animals ***(Swerczek, 2001).*** An induced metallothionein synthesis has also been observed in various rat tissues after exposure to cadmium ***(Onosaka and Cherian, 1981*** and ***Brzoska et al., 2000).***

Only a small proportion of absorbed cadmium (less than 10% in animal experiments) is eliminated, mainly in the urine and feces. Negligible amounts are eliminated through hair, nails and sweat ***(Friberg et al., 1974).*** Following oral administration of cadmium, ***Miller et al. (1967)*** reported that only very little amount of cadmium is secreted into cow's milk. When cadmium chloride was given orally (3 g/day) for two weeks to Holstein cows, the milk content of cadmium was below the detectable limits of

0.1µg/g. On this basis, less than 0.002 % of the administered amount appeared in the milk ***(Dorn, 1979).***

Doyle et al. (1974) found that growing sheep fed 60 ppm cadmium excreted 95 % of their cadmium intake in feces. ***Neathery et al. (1974)*** estimated that the intestinal tract still retained a significant amount of cadmium in dairy cows. Absorbed cadmium is excreted in urine, especially when cadmium concentration in renal cortex reached to 200 ppm ***(NRC, 1980).*** While gastrointestinal excretion is possible, particularly in bile as a glutathione complex, Cadmium excretion in urine increases proportionally with body burden ***(Friberg et al., 1986).***

3.3. Toxicological Features and Diagnosis of Cadmium Toxicosis:

3.3.1. Clinical Toxic Signs:

The maximum tolerable dietary level for cadmium had been set at 0.5 mg/kg for domestic animals. In the young animals, high levels of cadmium cause reduced growth rate and anemia. Sheep fed cadmium had lost the crimp of their wool. Reproductive problems related to ingested cadmium had been produced in cattle, sheep and goat. Those include abortion, deformed young, ovarian atrophy, testicular degeneration, and infertility. At very high levels cadmium can cause death ***(NRC, 1980*** and ***2001).*** Also, it reported that cadmium concentrations as low as 1 ppm in the drinking water or diet produced adverse effects in monogastric animals. These changes include hypertension, decreased kidney manganese and acute degenerative damage to the intestinal villi.

Gunson et al. (1982) investigated the following observations of lameness, swollen joints, and unthriftness; particularly in foals were due to chronic zinc/cadmium toxicosis in horses near a zinc smelter. In human, ***Jin et al. (1998)*** indicated that cadmium exposure can cause a variety of adverse health effects, among which kidney dysfunction, lung diseases, disturbed calcium metabolism and bone effects were most prominent.

Casteel (2001) reported that subchronic to chronic cadmium intoxication problem in horses was manifested as disease of the musculoskeletal system and kidneys.

Signs of chronic wasting, elevated back, emaciation, rough and rusty color hair coat that failed to shed, thin narrow neck, submandibular edema, and staining of the tail from fecal matter due to watery diarrhea in Simmental heifer and cows were believed to be related to cadmium toxicity ***(Swerczek, 2001),*** resulting in secondary Cu and Zn deficiency.

Liu (2003) suggested that the obvious signs of emaciation, anemia, anorexia and weakness in sheep and emaciation, inspiratory dyspnea in horses in the vicinity of non-ferrous metal smelters in Baiyin of Gansu province in China were caused by lead poisoning combined with cadmium.

Experimentally, ***Stoev et al. (2003)*** found that sheep treated with 4.5 mg cadmium sulfate/ kg b.w. given as a 1% water solution of cadmium sulfate per os for 8 subsequent days having clinical signs of scarce that not observed up to the day 4 of the experiment. After that day there was a loss of appetite and increased thirst in cadmium dosed animals. Also, atony of

the rumen, tachycardia, hurried and/or hard breathing were seen at that time. In addition, there was painfulness after deep palpation in the region of kidneys. The feces were soft and fetid. These signs were persistent up to the day 30 of the experiment, although they were less pronounced during the last several days.

3.3.2. Laboratory Diagnosis:

3.3.2.1. Cadmium Estimation:

3.3.2.1.1. Serum Cadmium Estimation:

Blood cadmium levels generally reflect recent exposure rather than accumulated body burden ***(Lauwerys et al., 1994).*** Also, ***Puls (1994)*** recorded that normal levels of blood cadmium in cattle do not exceed 0.001-0.04 ppm and any level above that is considered to be toxic. However, the blood cadmium is not diagnostically elevated in toxicity situations.

Moreover, ***López Alonso et al., (2000)*** reported that cadmium levels in blood were generally low and its concentrations were below the limits of detection in over 50% of the animals analyzed for assessing the effects of metal pollutants on domestic animals. On the other hand, ***Miranda et al. (2005)*** found that cattle from the industrialized area showed significantly higher cadmium levels than cattle from the rural area in all tissues analyzed except blood.

3.3.2.1.2. Urinary Cadmium Estimation:

With excessive exposure to cadmium, an increase in urinary cadmium may not occur until all of the available cadmium binding sites are saturated. However, when the available binding sites (e.g., metallothionein)

are saturated, increased urinary cadmium reflects recent exposure, body burden, and renal cadmium concentration, so that urinary cadmium measurement does provide a good index of excessive cadmium exposure ***(Shaikh et al., 1989).*** The most important measure of excessive cadmium exposure is increased cadmium excretion in urine ***(Klaassen, 2001).***

3.3.2.1.3. Tissues Cadmium Estimation:

The kidney, and to a lesser extent the liver, were the critical organs for cadmium accumulation. ***Neathery et al. (1974)*** and ***Friberg et al. (1979)*** stated that cadmium tissues concentration were in decreasing order as follow; kidneys, liver and small intestine. About 50 to 75 per cent of the body burden of cadmium is in the liver and kidneys. Concentrations of cadmium in muscle are lower than kidneys and liver ***(Sharma et al., 1979).*** However, significant accumulation of cadmium in muscle was reported after prolonged feeding diets high in cadmium to cattle ***(Smith, 1986).***

NRC (1980) stated that as cadmium exposure level and/or time of exposure increased, the concentration of cadmium in the liver, kidney, and muscle increased. Although concentrations of cadmium are higher in the liver and kidney than in muscle, increases of cadmium may be of greater importance in muscle because of the greater consumption of muscle meat by human. ***Elinder (1992)*** reported that in mammals and birds, cadmium accumulates in livers and kidneys at concentrations of 0.1-2 mg/kg and 1-10 mg/kg wet weight, respectively. Animals with a long life span such as horses have very high concentrations of cadmium in their organs: in renal cortex samples obtained from old horses, concentrations of nearly 200

mg/kg have been found. It has been suggested that the lowest critical concentration in the liver is 20 mg/kg wet weight ***(Krajnc et al., 1983).*** The most typical feature of chronic cadmium intoxication is kidney damage, which is generally assumed to occur at cadmium concentrations of 80-200 mg/kg wet weight ***(Shore and Douben, 1994).***

Also, ***Han et al. (1994)*** cited that cadmium is a cumulative toxicant in the continental ecological cycling; it accumulates mostly in the liver and kidneys because its rate of elimination from these organs is relatively low. This is partly due to the binding of cadmium to metallothioneins in these tissues ***(García-Fernández et al., 1996).*** They also suggested that the kidney is the main cadmium storage organ in animals subject to chronic low-level cadmium exposure.

Estimation of cadmium in the parenchymatous organs of goats was often a reliable indicator of environmental pollution as stated by ***Antoniou et al. (1995).*** The cadmium is gradually and progressively accumulated in animal tissues especially kidneys and higher cadmium concentrations were accompanied by lower copper and/or Zinc levels ***(Zasadowski et al., 1999).*** Recently, it is generally believed that high skeletal cadmium levels are characteristic of chronic exposure to cadmium ***(Berglund et al., 2000).***

3.3.2.2. General Blood Biochemistry:

Smith et al. (1991) found that calves from dams consuming 5 ppm cadmium had increased blood urea nitrogen by 63% with great reduction in serum copper and zinc. Histochemical examinations of ***Ibrahim (1993)*** revealed that dogs treated with cadmium chloride (248.8 and 2500 ppm/

liter of drinking water daily for 2 months) was significantly decreased alkaline phosphatase and increased acid phosphatase activities in the liver, kidneys and testes and reported there is a significant increase in serum urea, creatinine and glucose levels.

In human exposed to environmental cadmium and lead, ***Staessen et al. (1996)*** cited that serum alkaline phosphatase activity and urinary excretion of calcium were significantly and positively correlated with urinary cadmium.

Haneef et al. (1998) studied the effect of cadmium exposure on renal function of goats. They found that there was significant increase in serum urea nitrogen and creatinine levels.

Yamano et al. (1998) studies' revealed an increase in serum alanine aminotransferase activity 24 hrs after S/C administration of 3 to 6 mg cadmium /kg body weight of male Wibstar rats.

Because of incorporation of activated sludge into cattle and poultry rationale, ***Bag et al. (1999)*** study was undertaken to determine the toxic effects of cadmium contaminated domestic sewage sludge on Wibstar rats. The sludge was found to be contaminated with 0.005 mg cadmium per gram of dry sludge. They found that the levels of serum alanine aminotransferase and succinate dehydrogenase were significantly low in all rats. On the other hand, the serum aspartate aminotransferase and alkaline phosphatase activities were significantly higher in all animals.

3.3.2.3. Correlation between Serum Cadmium, Calcium and Inorganic Phosphorous:

Gunson et al. (1982) found that the zinc and cadmium concentrations were markedly increased in the pancreas, liver, and kidney of two foals born and raised near the zinc smelter which suffered from lameness and joint swelling. In serum of one foal, zinc and potassium concentrations were high, whereas calcium and magnesium concentrations were low.

Cadmium has the same ionic charge and radius as calcium and has been found to alter calcium haemostasis in vivo ***(Staessen et al. 1991).*** While, ***Kosla et al. (1993)*** found that the concentration of cadmium had no effect on the serum levels of calcium, magnesium, copper, zinc and inorganic phosphorous of cows in an environment polluted with cadmium.

Ibrahim (1993) encountered a significant increase in serum potassium and inorganic phosphate levels beside a significant decrease in serum sodium and calcium levels in dogs treated with cadmium chloride.

3. MATERIALS AND METHODS

3.1. Materials:

3.1.1. Areas of the study:

Our study involved 3 areas, the 1st area is *Kom-Hamadah* city (*Behera* governorate) containing 3 localities that are highly intensive with more than 50 brick kilns, and the 2nd area is *Kafr El-Zayiat* city (*Gharbia* governorate); containing super-phosphate factory in addition to other factories and several brick kilns as showed in the demonstrative map (Fig. 1). The water of the River Nile considered the main source of land cultivation as well as human and animals uses. The 3rd one is *Ez-Zéferani* area, belonging to *Kom Hamadah* city and about 20 km away from the studied localities and source of pollution and kept as a control area.

A. Kom Hamadah Localities:

3. Shabour:

It was Located 2.5 km south-western to the super-phosphate factory and 2 km southern to 50 brick kilns on western strand of the River Nile.

2. Mansheit Amin Esmaiel:

This village facing the super-phosphate factory on the western strand of the River Nile and under the effect of multiple brick kilns within 0.5 to 2 km distance.

1. Kafr Al-Aes:

This village is 3 km south-western to the super-phosphate factory and 0.5 km northern to some brick kilns.

B. Kafr El-Zayiat Localities:

1. Kafr Hashad:

This village located before the Tala canal drainage on the River Nile and about 3 km south-eastern to the super-phosphate factory facing *Shabour* village on the opposite strand.

2. Kafr El-Naseria:

This village located 1.5 km south-eastern to the super-phosphate factory on the eastern bank of the River Nile and under the effect of brick kilns.

3. Binûfar:

This village located 1.5 km south-western to super-phosphate factory drainage on the River Nile.

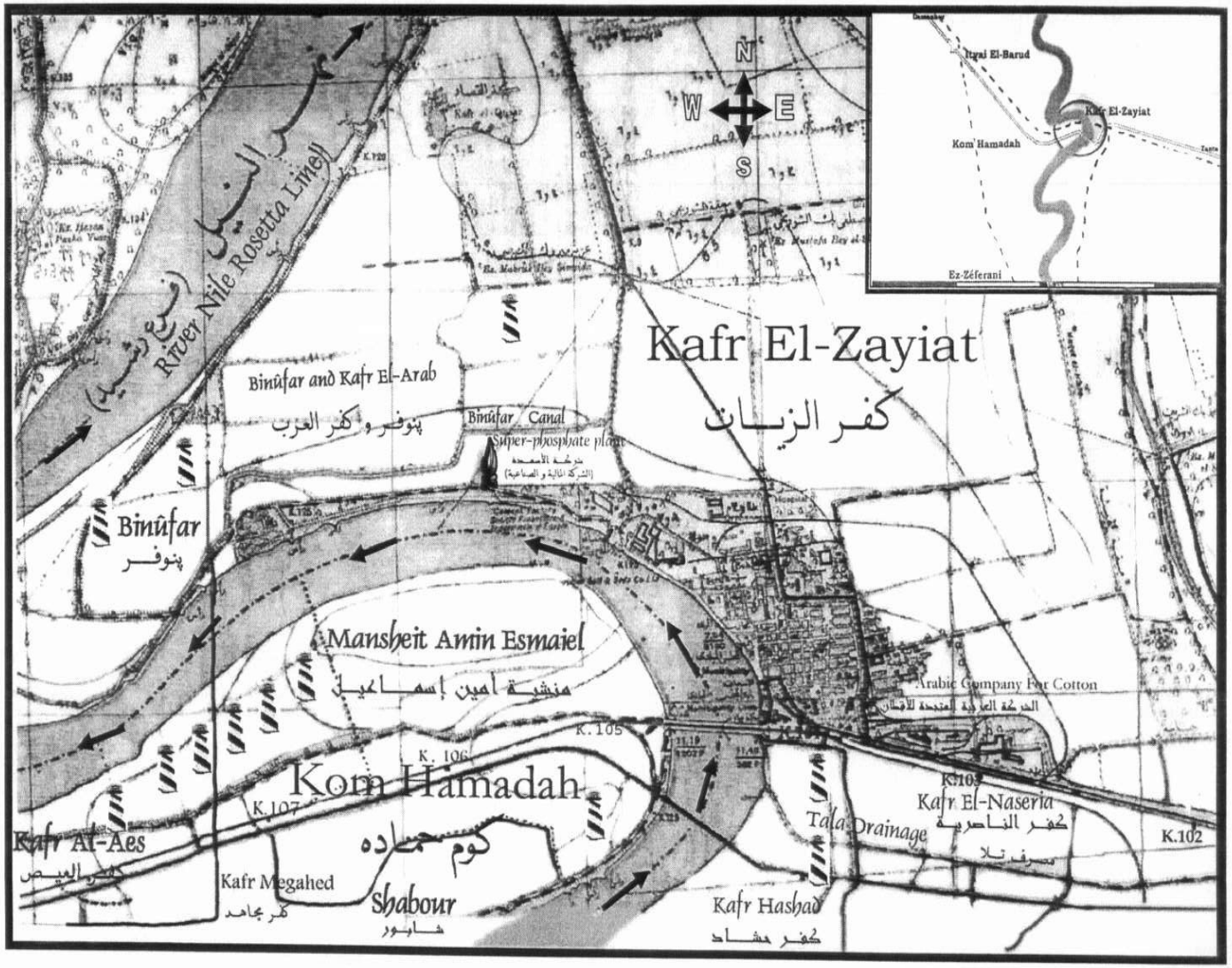


Fig. (1): -Geographical distribution of the studied areas around the super-phosphate factory.

= Brick kilns

3.1.2. Animals:

A random number of available cattle and buffaloes as recorded in table (1), belonging to the aforementioned villages were examined in this work. Animals of control group were chosen from *Ez-Zéferani* area.

Table (1): Number of examined animals and obtained samples from the areas of the study and control one.

Area	Type of Sample					
	Serum		Urine		Soft tissues	Hard tissues
	Cattle	Buffalo	Cattle	Buffalo	Buffalo	Buffalo
Kom Hamadah	45	40	30	30	20	20
Kafr El-Zayiat	45	50	30	30	22	22
Control (Ez-Zéferani)	10	10	10	10	10	10

3.1.3. Samples:

A. Macro- environmental samples:

1. Water samples:

About 8-10 surface water samples were collected from the River Nile every 0.5 km for about 2.5 km distance with and against water stream from the super-phosphate factory; the zero point of collection according to the standard method of ***USDA (1960).*** In addition water samples were collected from the control village and drainage system of super-phosphate factory before finding its way to River Nile water.

Water samples were collected from the surface water in volumetric flasks, 250 ml capacity and kept at room temperature till quantitative determination of samples contents of fluoride, sulfur and cadmium had been carried out.

2. Feed stuff samples:

8 samples of each feed stuff that consumed by studied animals were taken and analyzed. Cattle and buffaloes were generally fed on drees (barseem hay) and tibn (wheat straw). The quantities of fluoride, sulfur and cadmium levels were determined in the two forms of consumed feed stuff.

B. Micro-environmental samples:

Serum, urine, soft tissues (liver, kidney, lung, heart and muscle) and hard tissues (Incisors, premolars, molars, mandibles, maxilla and rib bones) were collected from the cattle and/or buffaloes of the studied areas.

Blood collection:- In clean, dry, sterile centrifuge tubes without anticoagulant, a sample of whole blood was allowed to flow freely and gently from the jugular vein on the inner wall of the tubes, then the non-haemolysed serum was obtained by centrifugation at 3000 rpm for 20 min., and kept in clean sterilized glass vials at –70°C till used ***(Coles, 1986)***. The prepared sera were used for determination of some biochemical parameters (AST, ALT and ALP enzyme activities, and total protein, albumin, globulin, urea, and creatinine content) and for quantitative determination of their fluoride, sulfur, cadmium, calcium and inorganic phosphorous levels.

Urine collection:- voided urine samples were obtained simultaneously in McCartney bottles and preserved in ice bags at -70°C till analysis of fluoride, sulfur, cadmium, calcium and inorganic phosphorous content.

3.1.4. Chemicals and Instruments:

Chemicals:- Diethyl ether (E.P. 34-46°C), N/10 HCl (metal-free analytical quality), petroleum ether (B.P. 40-60°C), anhydrous sodium fluoride, sodium citrate and sodium nitrite were purchased from Sigma Chemical Co. (St. Louis, MO, USA), Aldrich Chemical Company, LTD and Fluka Co. Kits for determination of (AST), (ALT) and (ALP) enzyme activities, and total protein, albumin, urea, and creatinine content were supplied by Isotech Co. and Biocon Co., respectively. Calcium and inorganic phosphorous kits were supplied by Biodiagnostic Co. (29 Tahreer St., Dokki, Giza, Egypt). All other chemicals and reagents were of the highest purity grade and commercially available.

Instruments: - Expandable ion analyzer (Orion Research) spectrophotometer (Pharmacia, LKB, Novaspec II, England), cooling centrifuge (4°C) (Heraeus, Christ), muffle furnace, electric oven, boiling water bath, and surgical instruments (scalpel, forceps, and scissors).

3.2. Methods:

3.2.1. Clinical examination of animals:

Animals were initially subjected to clinical examination to detect any apparent toxic symptoms.

3.2.2. Analytical Examination:

3.2.2.1. Sample Preparation:

a- Feed Stuff:

1 gm of each feed sample was placed in clean, dry (100 ml) Kjeldahl flask and 20 ml purified nitric acid was added. The samples were gently heated till appearance of brownish fumes with continued heating until the volume reach 2-3 ml in 90-120 min. Complete digestion is indicated by the disappearance of the liquid colour. The digested sample was diluted with bi-distilled water up to 50 ml in a volumetric flask ***(Koirtyohann et al., 1982).***

b- Soft Tissues:

Random samples of liver, kidneys, lungs, heart and muscles were collected from slaughtered animals at villages of the study and digested according to the method described by ***Koirtyohann et al. (1982).*** 0.5 gm of each tissue was placed in clean and dry (100 ml) Kjeldahl flask and 10 ml purified nitric acid was added. The acid was allowed to react at room temperature overnight. Then the digestion mixture was divided into two parts; one part for determination of sulfur, and other part were prepared for the analysis of fluoride, cadmium, calcium and inorganic phosphorous levels, which was heated gently on hot plates at 50-70 ° C until the appearance of brownish fumes. Complete digestion is indicated by the disappearance of nitric oxide and ashing of samples. The digested sample was diluted with bi-distilled water up to 25 ml in a volumetric flask.

c- Hard Tissues:

Incisors, premolars, molars, mandibles, maxilla and rib bones were collected from the slaughtered buffaloes and extracted with petroleum ether (B.P. 40-60°C). The dry, fat free samples were burned in muffle furnace at 200°C to facilitate crushing and grinding. The grounded bony tissues were then ashed at 600°C for 12 hrs in the muffle furnace. The ashed bone was used for estimation of fluoride, calcium and inorganic phosphorous.

3.2.2.2. Analytical Methods:-

1. Determination of Fluoride:

Fluoride ions were determined in water, feed stuff, serum, urine and tissues according to the method of ***Fry and Taves (1970),*** by means of expandable ion analyzer EA 920, Orion Research using single fluoride electrode (Model 94 – 09 – 00).

Stock Standard Fluoride Solution:

It was prepared by dissolving 221 mg of anhydrous sodium fluoride (NaF) in distilled water and diluted to 1000 ml (1.0 ml equal to 100 µg F).

Working Standard Solution:

It was prepared by diluting 100 ml from the stock standard solution to 1000 ml distilled water (1 ml equal to 10 µg F). Two standard solutions were prepared for the adjustment of standard I and II of the apparatus. The first standard was equal to 1/10 of the second one.

Total Ionic Strength Adjustment Buffer (TISAB):

To approximately 500 ml distilled water, 57 ml of analytical reagent

grade glacial acetic acid, 58 gm of analytical reagent grade NaCl, and 0.3 gm of sodium citrate were added. The solution was titrated to PH 5.0 – 5.5 using analytical reagent grade 5N, NaOH. The solution was cooled and diluted to make 1 liter.

Procedures:

Equal amounts of sample and (TISAB) were added. The solution was stirred and the electrodes immersed in it. Reading was recorded after three minutes ***(Frant and Ross, 1968).***

2. Determination of Sulfur:

Sulfur was determined in serum, urine, soft tissues, water and feed stuff according to the method described by ***Stockholm and Roch (1957),*** which based on precipitation of sulfur by using barium chloride. The barium sulphate thus formed, can be weighted and the sulfur content was calculated by the following formula:

$S = B \times 0.1374$

Where; S= sulfur concentration. B= weight of barium sulphate.

3. Determination of Cadmium:

Cadmium concentrations of prepared samples were estimated according to the method of ***Gardiner (1974),*** using specific ion electrode model (94-84) attached to expandable ion analyzer EA 920 Orion Research with Orion model 90 – 02 double junction reference electrodes, Orion American Company. The PH of each sample had been adjusted just before analysis.

4. Determination of Calcium:

Calcium was determined in serum, urine and tissues by *O*-cresolphthalein complexone method that described by ***Gosling (1986)*** and ***Farrel (1987)*** using kits supplied by Biocon Co.

5. Determination of Inorganic Phosphorous:

Determination of Inorganic phosphorous colorimetrically in serum, urine and tissues was carried out by the use of test kits supplied by Biodiagnostic Co. and after the method described by ***El-Merzabani et al. (1977).***

6. Biochemical analysis of some serum parameters:

1) Determination of serum total protein level (g/dl):

Serum total protein was determined colorimetrically according to the method described by ***Henry (1964)*** using kits supplied by Diamond Diagnostic Co.

2) Determination of serum albumin level (g/dl):

Serum albumin level was determined photometrically according to the method described by ***Doumas et al. (1971)*** using albumin kits supplied by Diamond Diagnostic Co.

3) Determination of serum globulin level (g/dl):

Serum globulin level was determined by subtracting the albumin value from the total protein value of the same sample according to ***Coles (1986).***

4) Determination of serum urea level (mg %):

Serum urea level was determined spectrophotometrically after the method described by ***Fawcett and Scott (1960)*** using kits supplied by Quimica Clinica Aplicada S.A. Company.

5) Determination of serum creatinine level (mg %):

Serum creatinine level was determined colorimetrically according to the method described by ***Husdan and Rapapost (1968)*** using kits supplied by Biocon Co.

6) Determination of serum AST and ALT enzyme activities (IU/L):

Serum AST and ALT activities were determined photometrically according to the method described by ***Reitman and Frankel (1957)*** using kits supplied by Diamond Diagnostic Co.

7) Determination of serum ALP enzyme activity (IU/L):

Serum alkaline phosphatase activity was estimated colorimetrically according to the method described by ***Belfield and Goldberg (1971)*** using kits supplied by Biodiagnostic Co.

3.2.3. Statistical Analysis:

The data obtained were statistically analyzed using the GLM procedure of the Statistical Analysis System computer package ***(SAS, 1987)***. The statistical model included the effects of locality and species. Means were separated using the Least Square Means (LSM) of the same program.

4. RESULTS

1) Water Sources and Feed Stuff Analysis:

Analytical findings of fluoride, sulfur and cadmium levels in the River Nile water samples collected every 0.5 km distance around *Kafr El-Zayiat* super-phosphate factory for a distance of about 5 km are shown in Table (2) and Fig. (2). As regard to the area of the study, it's generally evident that there was a significant elevation of fluoride levels at *Mansheit Amin Esmaeil, Shabour* and *Binūfar*; and sulfur levels at the same localities as well as *Kafr El-Naseria* locality, while cadmium concentration was significantly elevated only at *Mansheit Amin Esmaeil and Binūfar*. In relation to the super-phosphate factory, the three elements were significantly increased within 0.5 km pre-factory (except cadmium) and extended to about 1.5 to 2 km post-factory towards the direction of water. Predictably, the highest values of fluoride, sulfur and cadmium were recorded in samples taken from the drainage system of the super-phosphate factory.

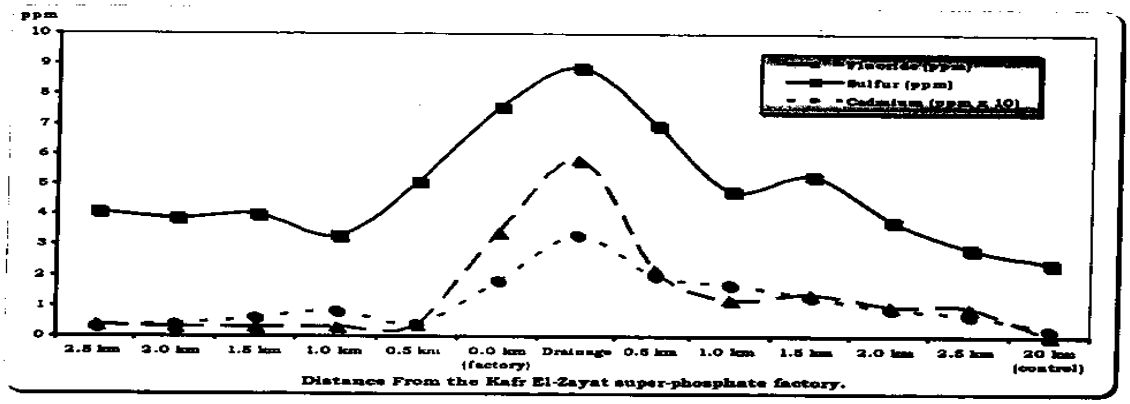
Regarding to the feed stuff analytical findings, the fluoride concentration was significantly increased in tibn and drees samples at *Mansheit Amin Esmaeil, Shabour, Binūfar* and *Kafr Al-Aes* localities. Whereas sulfur concentrations were significantly elevated in tibn samples collected from all localities except *Kafr Hashad* and in drees samples from *Mansheit Amin Esmaeil* and *Shabour* only. On the other hand, the significant concentrations of cadmium were noticed in samples collected from *Mansheit Amin Esmaeil* only. The results were recorded in Table (3) and Fig. (3).

Table (2): Fluoride, sulfur and cadmium concentrations in River Nile water with and against water stream around Kafr El-Zayiat super-phosphate factory:

Distance	Studied Locality	Fluoride (ppm)	Sulfur (ppm)	Cadmium (ppm)
2.5 km (against water stream)	Shabour	0.4± 0.02 ef	4.1± 0.18 d	0.03± 0.007 c
2.0 km (against water stream)	Kafr El-Naseria	0.3±0.16 fg	3.9±0.26 de	0.04±0.01 c
1.5 km (against water stream)		0.3±0.13 efg	4.0±0.10 d	0.06±0.02 c
1.0 km (against water stream)		0.3±0.16 gf	3.3±0.13 ef	0.08±0.04bc
0.5 km (against water stream)		0.4±0.02 ef	5.1±0.16 c	0.04±0.01 c
0.0 km (Factory)	Mansheit Amin Esmaeil	3.4±0.58 b	7.6±0.45 b	0.18±0.06 b
Drainage (Factory)		5.8±0.29 a	8.9±0.36 a	0.33±0.11 a
0.5 km (with water stream)		2.2±0.92 c	7.0±0.81 b	0.20±0.09 ab
1.0 km (with water stream)		1.2±0.06 d	4.8±0.56 c	0.17±0.06 b
1.5 km (with water stream)	Binûfar	1.4±0.60 d	5.3±0.44 c	0.13±0.09 b
2.0 km (with water stream)		1.0±0.19 ed	3.8±0.18 ed	0.09±0.02 bc
2.5 km (with water stream)	Kafr Al-Aes	0.93±0.12 def	2.9±0.30 gf	0.07±0.02 c
20 km (control)	Ez-Zéferani	0.02±0.009 g	2.4±0.34 g	0.02±0.007 c

-Means in the same column with similar letter do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means + SE).



← against water stream with water stream →

Fig (2): Fluoride, sulfur and cadmium concentrations in River Nile water in relation to Kafr El-Zayiat super-phosphate factory.

Table (3): Fluoride, sulfur and cadmium contents in the feedstuff (tibn and drees) at the studied areas:

Area Locality	Fluoride (ppm)		Sulfur (ppm)		Cadmium (ppm)	
	Tibn	Drees	Tibn	Drees	Tibn	Drees
Kom Hamadah:-						
Shabour	2.7 ± 0.61 b	3.7± 1.04 a	5.8± 0.68 b	4.3± 0.16 ab	0.027± 0.013 b	0.037± 0.003 b
Mansheit Amin Esmaeil	5.4± 0.52 a	4.3± 0.64 a	7.1± 0.05 a	5.4± 0.29 a	0.063± 0.01 a	0.11± 0.024 a
Kafr Al-Aes	1.81± 0.35 bc	2.6± 0.75 ab	2.5± 0.33 cd	2.8± 0.27 bc	0.024± 0.006 b	0.031± 0.001b
Kafr El-Zayiat:-						
Kafr Hashad	0.19± 0.03 d	0.8± 0.32 c	2.1± 0.02 de	2.2± 0.2 c	0.024± 0.001 b	0.054± 0.02 b
Kafr El-Naseria	0.75± 0.36 cd	1.4± 0.29 bc	3.3± 0.35 c	3.1± 0.86 bc	0.028± 0.018 b	0.044± 0.03 b
Binûfar	1.9 ± 0.08 bc	2.7± 0.21 ab	2.6± 0.28 cd	2.5± 0.27 bc	0.035± 0.013 ab	0.066± 0.02 b
Control (Ez-Zéferani)	0.006± 0.003 d	0.18± 0.04 c	1.6± 0.19 e	1.4± 0.18 c	0.0014± 0.0001 b	0.044± 0.01 b

-Means in the same column with similar letter (a-e) do not differ significantly at P ≤ 0.05.

-Values are represented as (Means ± SE).

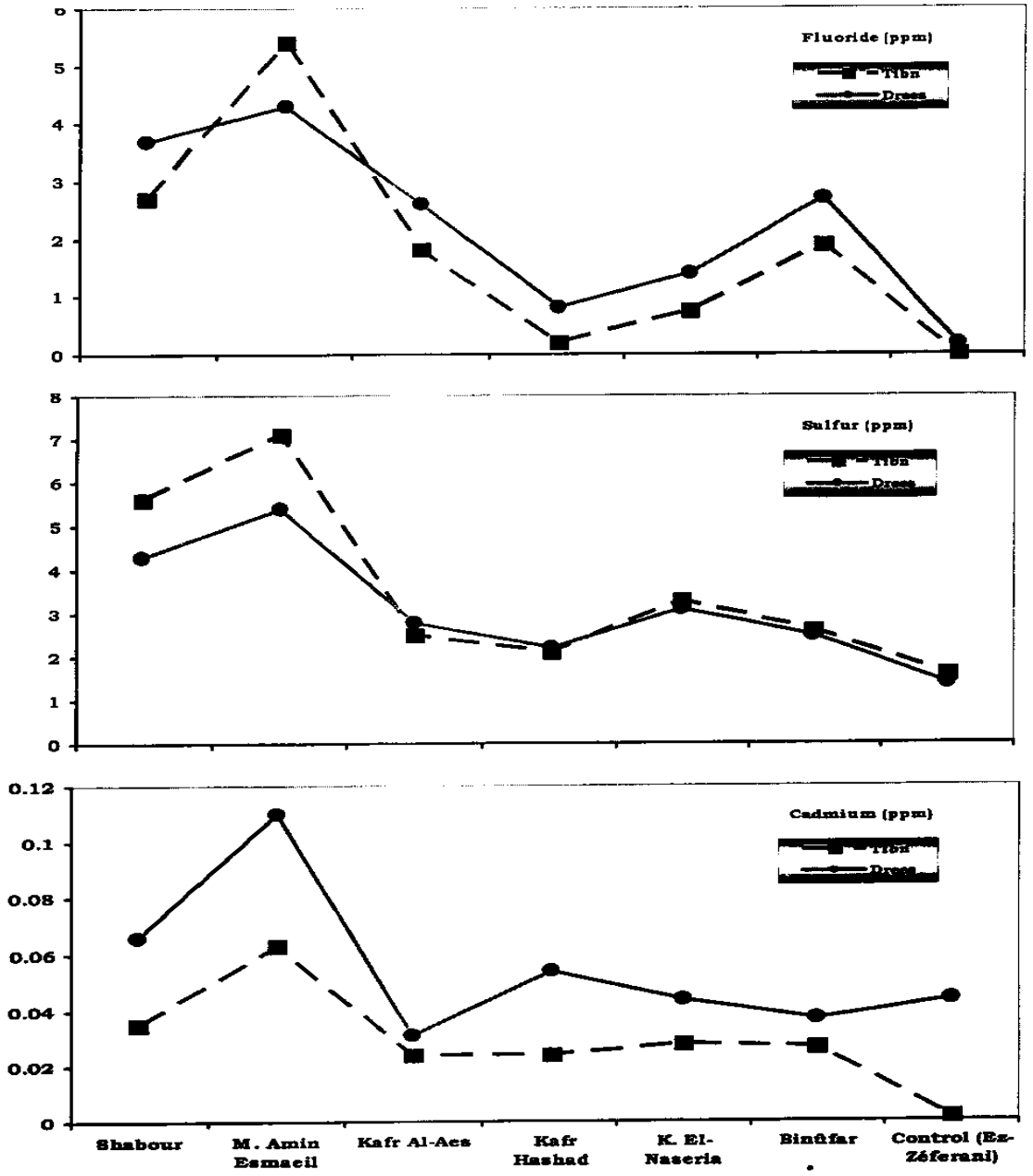


Fig (3): Fluoride, sulfur and cadmium concentrations in the feed stuff (tibn and drees) from the studied localities.

2) Toxicological Feature and Diagnosis:

I. Clinical toxic signs:

Cattle and buffaloes in the different localities in the studied areas around the super-phosphate factory and brick kilns examined clinically and the clinical toxic signs were recorded. Animals at *Kom Hamadah* localities were so severely affected more than those at *Kafr El-Zayiat*, and ranged in a descending order of *Mansheit Amin Esmaeil, Shabour,* and *Kafr Al-Aes,* followed by *Binûfar and Kafr El-Naseria,* then *Kafr Hashad*, respectively.

Examined cattle showed cachexia, emaciation, pale mucous membrane, rough dry coat, and drop in milk yield with an incidence of reproductive failure as a general signs. Dental changes were observed as brown to blackish discoloration, pitting and attrition of incisors and irregular enamel surface (Figs. 4 and 5). There were no any observed signs of skeletal changes or lameness.

While in buffaloes, the signs were more pronounced including severe emaciation and weakness, unthriftness, rough dry hair losing its luster appearance and easily detached, depressed appetite, paleness of mucous membranes, poor general health condition (Figs.6 and 7).

Most of the affected buffaloes suffered from a pronounced lameness that was intermittent and recurrent annually in summer months, shifting from leg to leg, sometimes affecting the four limbs according to the history of the animal (Fig. 6). During movement, the animal was showing abducted elbow and arched back with pronounced bony extremities and weak musculature of lamed legs (Fig. 7).

The severely lamed animal was unwilling to stand, cannot bear weight, and became recumbent for long period ended by its slaughtering (Fig.8).

The examined buffaloes showed the most obvious signs of dental changes in variable degrees ranged from brown to dark brown or even blackish discoloration in severely affected cases with mottling of all permanent teeth especially pre-molar and molar teeth compared to incisors (Figs. 9, 10, 11 and 12). However, attrition was so severe with oblique eruption observed in most clinically affected incisive teeth.

There were no any signs of bony exostosis; only peri-odentosis was observed in severely intoxicated animals (Fig. 12).

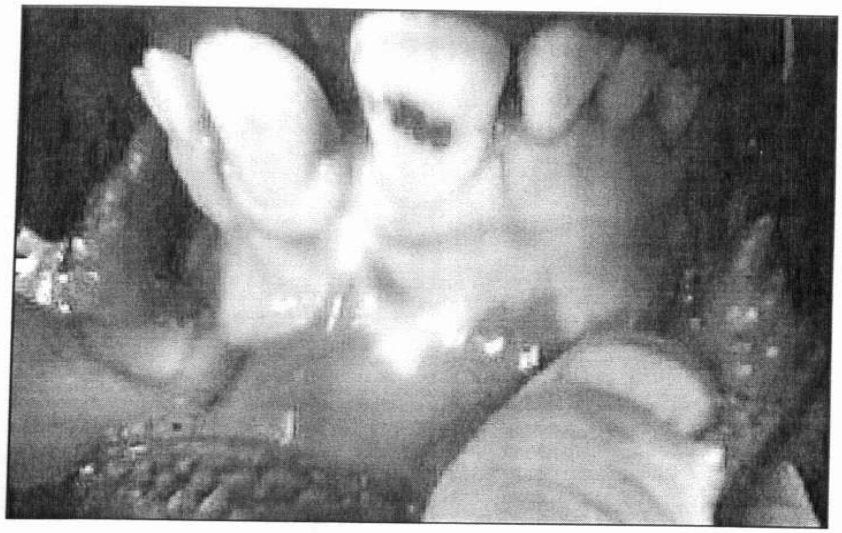


Fig. (4): Dental fluorosis in cattle (2 yrs old) showing patched brown discoloration of one central incisor.

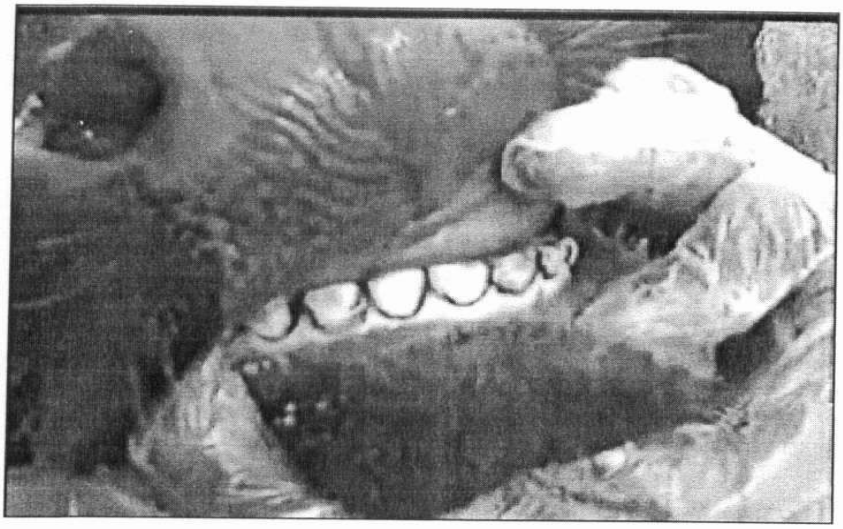


Fig. (5): Cattle (5 years) showing dental fluorosis, attrition, pitting with chalky appearance of enema in all incisors teeth.

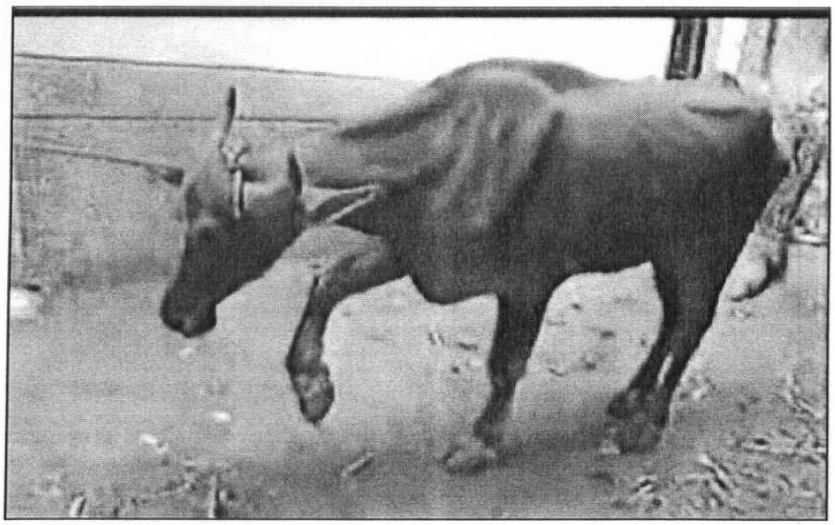


Fig. (6): Buffalo (5.5 years) showing pronounced intermittent lameness that annually recurrent in summer months and shifting from leg to leg.

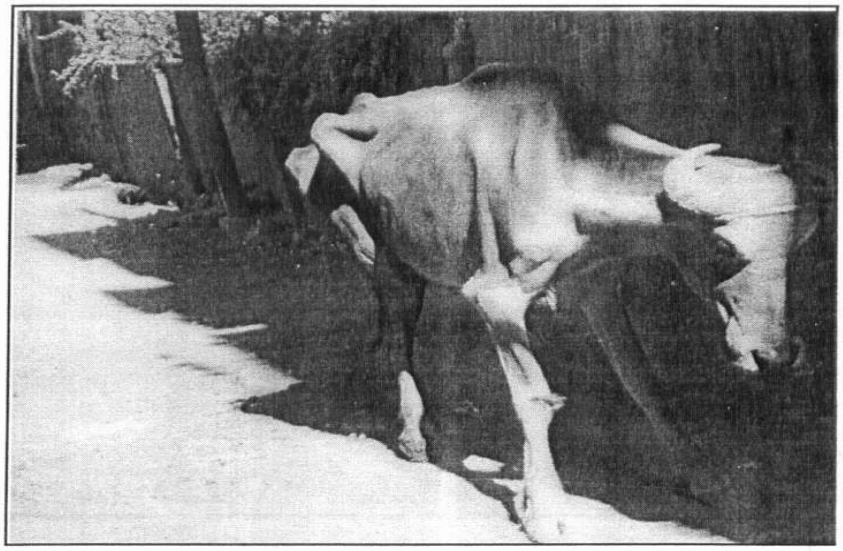


Fig. (7): Lamed buffalo (5 years) showing abducted elbow and arched back with pronounced bony extremities and weak musculature, also showing severe emaciation, unthriftness and poor general health condition.

Fig. (8): Recumbent buffalo for long period due to incurable lameness.

Fig. (9): Dental fluorosis in buffalo (4 yrs old) showing diffused brown discoloration, pitting and chalky-appearance of enema of incisive teeth.

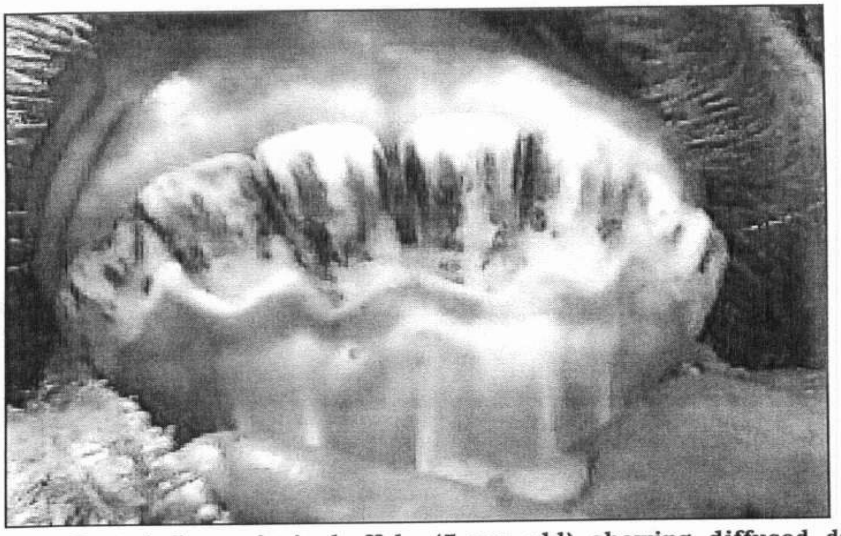


Fig. (10): Dental fluorosis in buffalo (5 yrs old) showing diffused dark brown to blackish discoloration, pitting and chalky-appearance of enema of the incisive teeth.

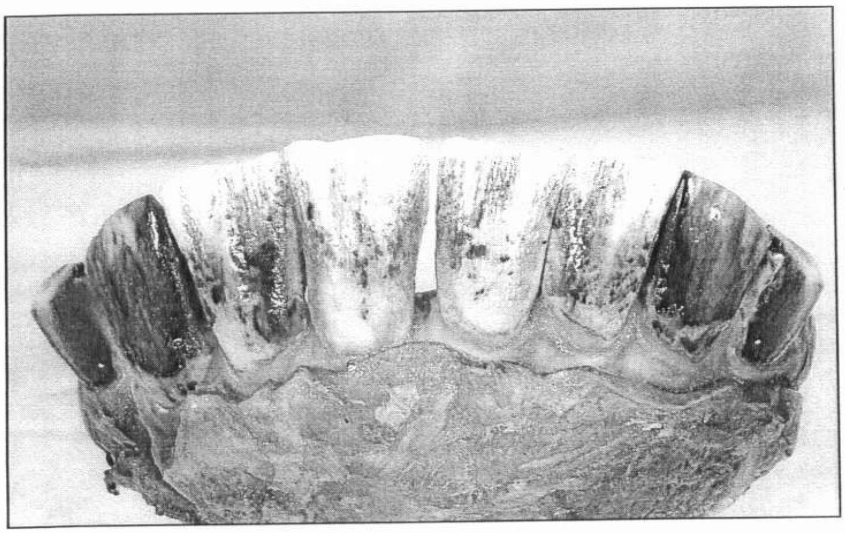


Fig. (11): Dental fluorosis in buffalo (5.5 yrs old) showing pitting and diffused brown to blackish discoloration of incisive teeth (PM sample).

Fig. (12): Dental fluorosis in severely intoxicated buffalo showing diffused dark blackish discoloration of cheek teeth with peri-odentosis (the arrow), PM sample.

II. Analytical findings:

1. Fluoride Concentrations:

1.1. Serum fluoride:

Analytical findings of fluoride concentrations in serum of cattle in the studied areas were illustrated in Table (4) and Fig. (13), where the higher serum fluoride concentrations are recorded in *Mansheit Amin Esmaeil* (1.27 ± 0.07 ppm) that facing the super-phosphate factory, followed by *Shabour* (0.99 ± 0.12 ppm) which are significantly higher than those of other localities and control one, while in buffaloes, Table (5) and Fig. (13) showed that the higher levels of serum fluoride are noticed in *Mansheit Amin Esmaeil* (1.34 ± 0.14 ppm) and *Shabour* (1.18 ± 0.10 ppm), followed by *Kafr El-Naseria* (0.58 ± 0.08 ppm) than other localities and control area.

1.2. Urinary fluoride:

The urinary fluoride levels of cattle show great significant differences between the studied localities when compared with the control area, where the highest values are observed in *Mansheit Amin Esmaeil* (43.3 ± 2.7 ppm), followed by *Shabour* (34.3 ± 1.8 ppm) and *Kafr El-Naseria* (29.7 ± 1.6 ppm) localities (Table, 4 and Fig. 14). In buffaloes, urine analysis revealed a highly significant elevation of fluoride in the various localities when compared with control area, with highest observed values in *Kom Hamadah* localities and *Kafr El-Naseria,* followed by *Binūfar* as shown in the Table (4) and Fig. (14).

1.4. Soft tissues fluoride:

Table (5) and Fig. (15) summarize the analytical finding of fluoride content in soft tissues of buffaloes (liver, kidneys, lungs, heart and muscle). The significant concentration of fluoride was detected only in the renal tissues in *Kom Hamadah* (1.38 ± 0.01 ppm) and *Kafr El-Zayiat* (1.26 ± 0.12 ppm) as compared to *Ez-Zéferani* area (0.67 ± 0.14 ppm).

1.5. Hard tissues fluoride:

Analytical finding of fluoride concentrations in the bony tissues in the studied areas was illustrated in Table (6) and Fig. (16), in which a significant rise in the fluoride level are detected in all samples of bony tissues samples when compared with control ones. In addition, the maximum fluoride concentrations are observed in the cheek teeth and mandible followed by incisive teeth and maxilla, while the ribs contains the lowest values of fluoride. In relation to areas, *Kom Hamadah* recorded a significant elevation in the fluoride levels in the incisive (3800 ± 544.7 ppm) and pre-molar (5300 ± 418.3 ppm) teeth in comparison with those of *Kafr El-Zayiat*, which are (2163 ± 313.2 ppm) and (4300 ± 460.1 ppm), respectively.

Table (4): Fluoride concentrations (ppm) in serum and urine samples of cattle and buffaloes in the studied areas:

Area Locality	Serum		Urine	
	Cattle	Buffaloes	Cattle	Buffaloes
Kom Hamadah:				
Shabour	0.99 ± 0.12 b	1.18 ± 0.10 a	34.3 ± 1.8 b	50.3 ± 1.88 a
Mansheit Amin Esmaeil	1.27 ± 0.07 a	1.34 ± 0.14 a	43.3 ± 2.7 a	48 ± 1.47 a
Kafr Al-Aes	0.19 ± 0.01 c	0.19 ± 0.02 c	27.6 + 1.3 c	44 + 2.04 a
Kafr El-Zayiat:				
Kafr Hashad	0.20 ± 0.02 c	0.19 ± 0.02 c	10.0 ± 0.8 e	19.2 ± 3.5 c
Kafr El-Naseria	0.35 ± 0.09 c	0.58 ± 0.08 b	29.7 ± 1.6 bc	47.8 ± 4.3 a
Binûfar	0.25 ± 0.07 c	0.26 ± 0.02 c	15.8 ± 1.8 d	29.2 ± 1.18 b
Control (Ez-Zéferani)	0.19 ± 0.008 c	0.19 ± 0.02 c	3.88 ± 0.3 f	3.14 ± 0.22 d

-Means in the same column with similar letter (a-f) do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

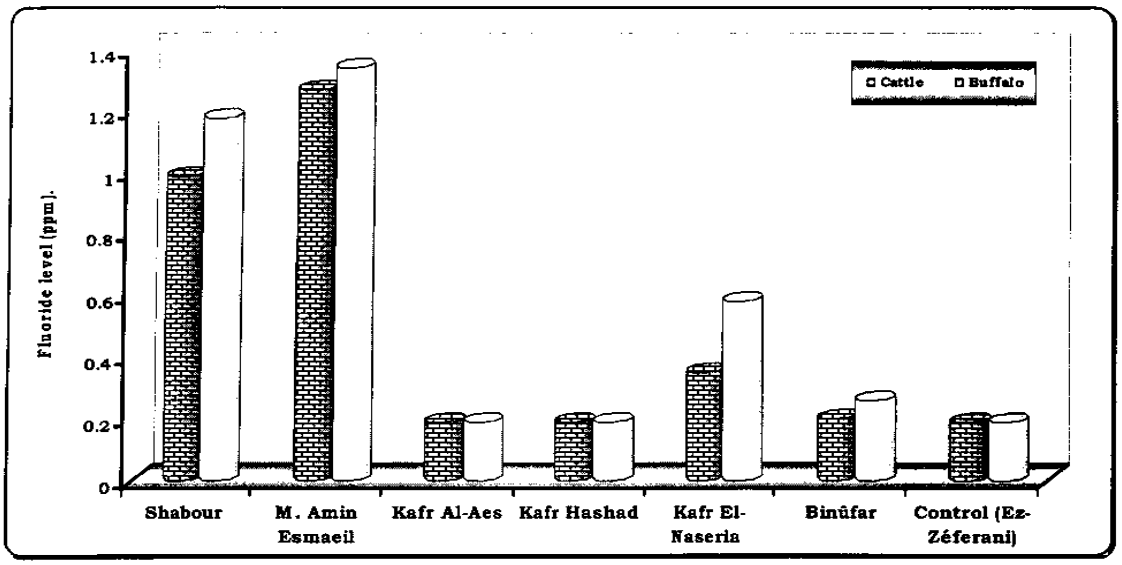


Fig. (13): Serum fluoride content (ppm) of cattle and buffaloes.

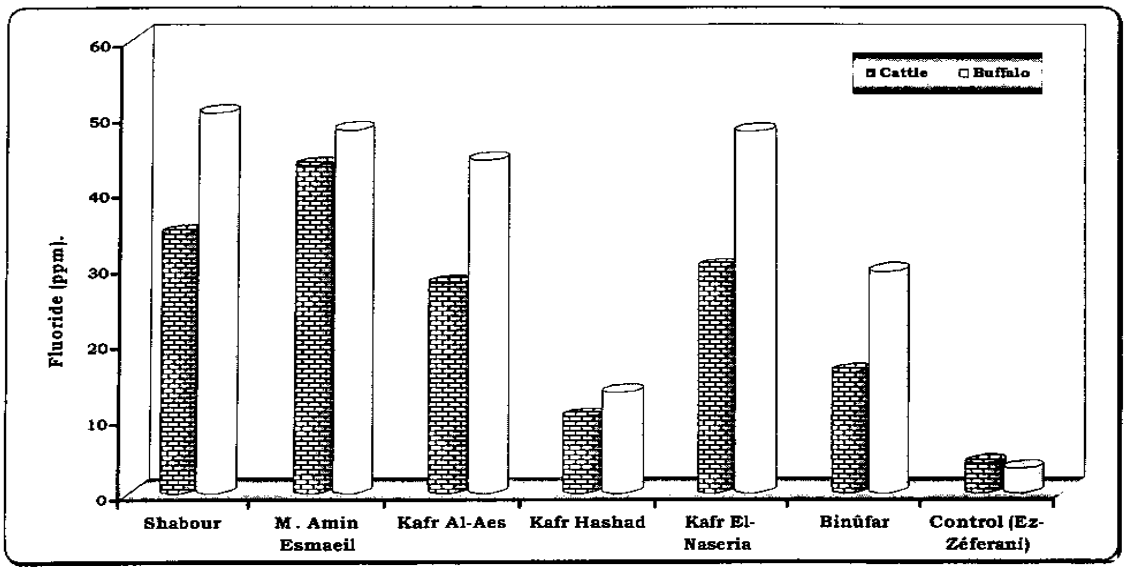


Fig. (14): Urinary fluoride content (ppm) of cattle and buffaloes.

Table (5): Fluoride concentrations (ppm) in the soft tissues of buffaloes in the studied areas:

Organ / Area	Liver	Kidneys	Lung	Heart	Muscle
Kom Hamadah	0.86 ± 0.06 a	1.38 ± 0.01 a	0.25 ± 0.09 a	0.57 ± 0.06 a	0.44 ± 0.08 a
Kafr El-Zayiat	1.03 ± 0.39 a	1.26 ± 0.12 a	0.27 ± 0.09 a	0.63 ± 0.03 a	0.37 ± 0.09 a
Control (Ez-Zéferani)	0.56 ± 0.04 a	0.67 ± 0.14 b	0.23 ± 0.04 a	0.28 ± 0.007 a	0.29 ± 0.005 a

-Means in the same column with similar letter do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means + SE).

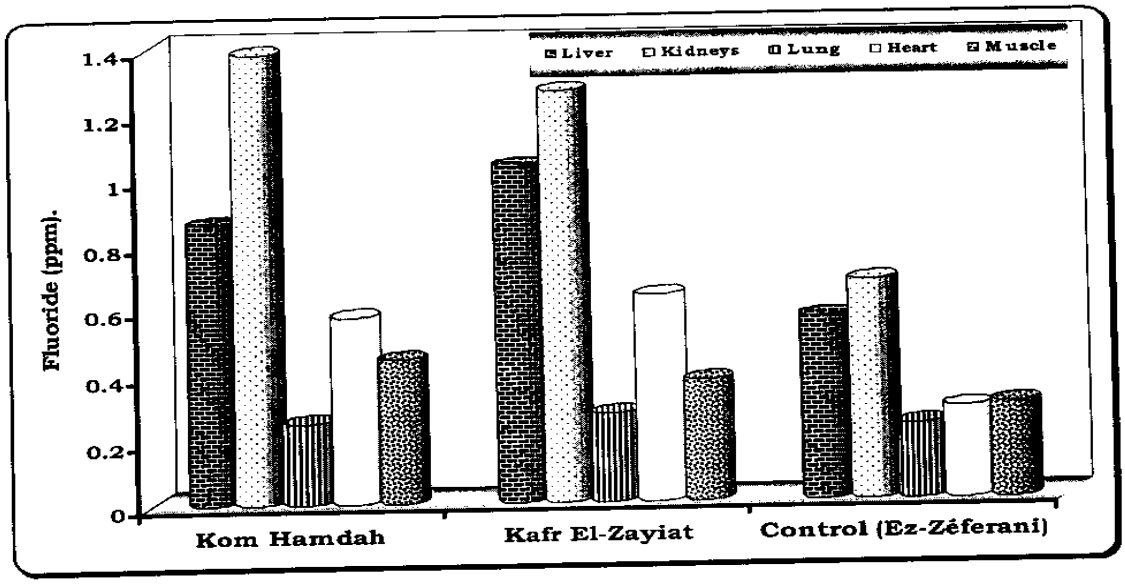


Fig. (15): Fluoride content (ppm) in the soft tissues of buffaloes.

Table (6): Fluoride concentrations (ppm) in the hard tissues of buffaloes in the studied areas:

Tissue / Area	Teeth			Mandible	Maxilla	Ribs
	Incisor	Premolar	Molar			
Kom Hamadah	3800 ± 544.7 a	5300 ± 618.3 a	4187 ± 214.5 a	4475 ± 931.1 a	3287 ± 409 a	2500 ± 247 a
Kafr El-Zayiat	2163 ± 313.2 b	4300 ± 460.1 b	4850 ± 756.9 a	3138 ± 474.1 ab	2763 ± 55.4 a	2300 ± 81.6 a
Control (Ez-Zéferani)	348 ± 90.4 b	1165 ± 488.9 c	1188 ± 303.0 b	1188 ± 296.8 b	200 ± 55.0 b	110 ± 14.7 b

-Means in the same column with similar letter do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

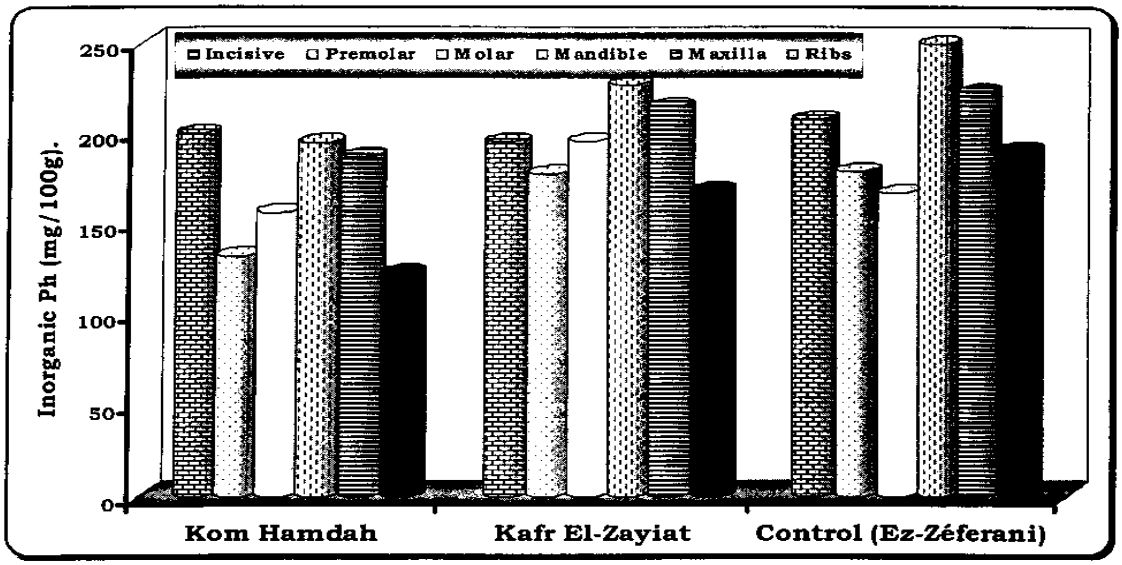


Fig. (16): Fluoride content (ppm) in the hard tissues of buffaloes.

2. Sulfur Concentrations

Sulfur levels of cattle and buffaloes were determined in their serum, urine and different organs.

2.1. Serum and Urinary sulfur:

Serum and urinary sulfur levels of cattle and buffaloes in the studied areas are shown in the Table (7) and Figs. (17 & 18). It is clear that cattle at all localities except *Kafr Hashad* showed significant levels of serum and urinary sulfur when compared with the control area, where the highest values were observed in *Mansheit Amin Esmaeil and Shabour*. However, in buffaloes, the significant concentrations were noticed only in *Mansheit Amin Esmaeil and Shabour*, but in lower values than that of cattle.

2.3. Soft tissues sulfur:

Estimation of sulfur content in renal, hepatic and pulmonary samples collected from buffaloes revealed a significant increase in the studied areas, without any significant variations in between, as compared with those of control area, while the cardiac and skeletal muscles showed a non-significant rise in their sulfur content in either *Kom Hamadah* or *Kafr El-Zayiat* areas when compared with their respective control. The highest value is observed in both renal and hepatic tissues as presented in Table (8), and Fig. (19).

Table (7): Sulfur concentrations (ppm) in serum and urine samples of cattle and buffaloes in the studied areas:

Area Locality	Serum		Urine	
	Cattle	Buffaloes	Cattle	Buffaloes
Kom Hamadah:				
Shabour	6.94 ± 0.16 a	4.32 ± 0.05 a	25.8 ± 0.13 b	9.6 ± 0.43 a
Mansheit Amin Esmaeil	8.94 ± 0.25 a	4.38 ± 0.07 a	32.3 ± 0.19 a	10.8 ± 0.11 a
Kafr Al-Aes	2.16 ± 0.09 b	0.96 ± 0.02 b	16.4 ± 0.08 c	5.04 ± 0.14 b
Kafr El-Zayiat:				
Kafr Hashad	1.50 ± 0.04 c	0.99 ± 0.07 b	11.9 ± 0.11d	6.6 ± 0.28 b
Kafr El-Naseria	1.68 ± 0.03 b	0.84 ± 0.01 b	16.6 ± 0.19 c	5.02 ± 1.09 b
Binûfar	2.22 ± 0.09 b	0.96 ± 0.02 b	19.6 ± 0.12 c	5.03 ± 0.24 b
Control (Ez-Zéferani)	0.72 ± 0.08 c	0.78 ± 0.004 b	5.1 ± 0.08 d	6.2 ± 0.76 b

-Means in the same column with similar letter (a-c) do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

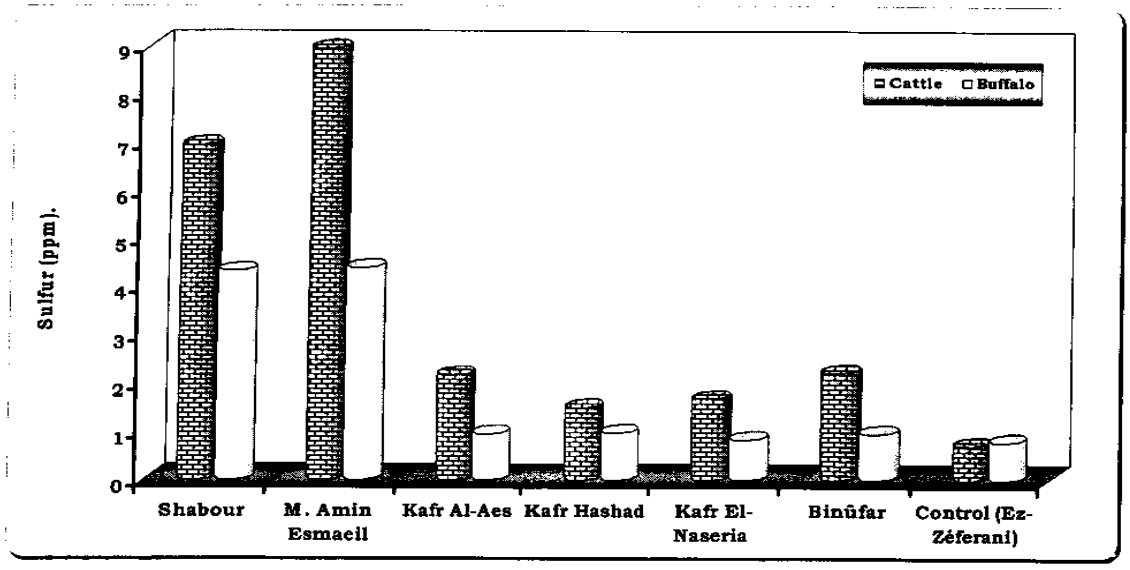


Fig. (17): Serum sulfur content (ppm) in cattle and buffaloes.

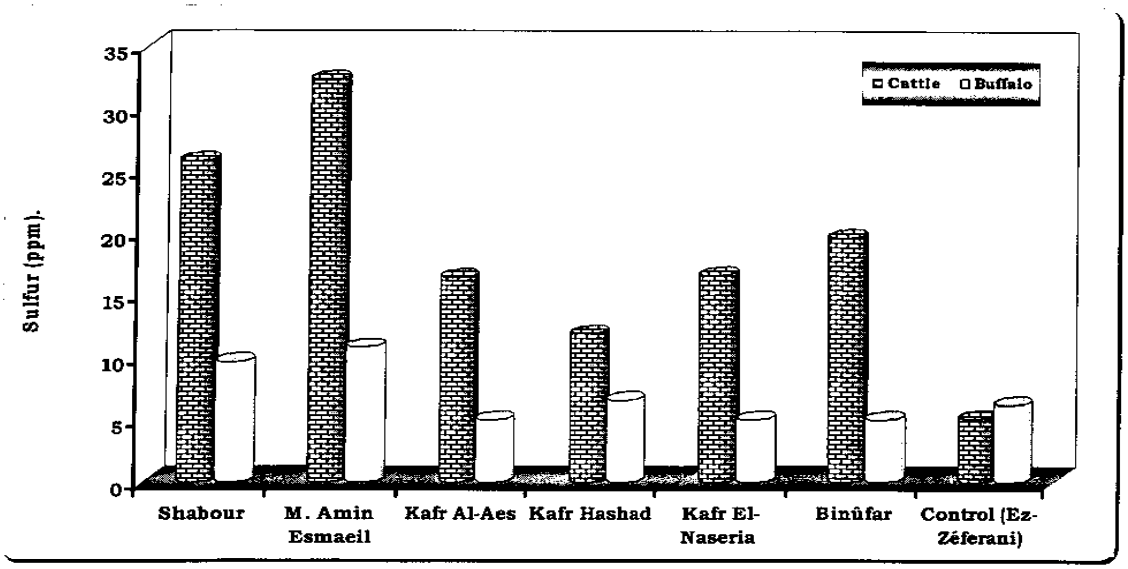


Fig. (18): Urinary sulfur content (ppm) in cattle and buffaloes.

Table (8): Sulfur concentrations (ppm) in the soft tissues of buffaloes in the studied areas:

Organ / Area	Liver	Kidneys	Lung	Heart	Muscle
Kom Hamadah	8.63 ± 1.84 a	8.86 ± 1.28 a	1.10 ± 0.29 a	0.33 ± 0.06 a	0.80 ± 0.07 a
Kafr El-Zayiat	5.11 ± 0.82 ab	5.28 ± 0.37 a	1.24 ± 0.32 a	0.49 ± 0.19 a	0.73 ± 0.09 a
Control (Ez-Zéferani)	0.67 ± 0.15 b	1.15 ± 0.27b	0.16 ± 0..05 b	0.31 ± 0.08 a	0.56 ± 0.09 a

-Means in the same column with similar letter do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

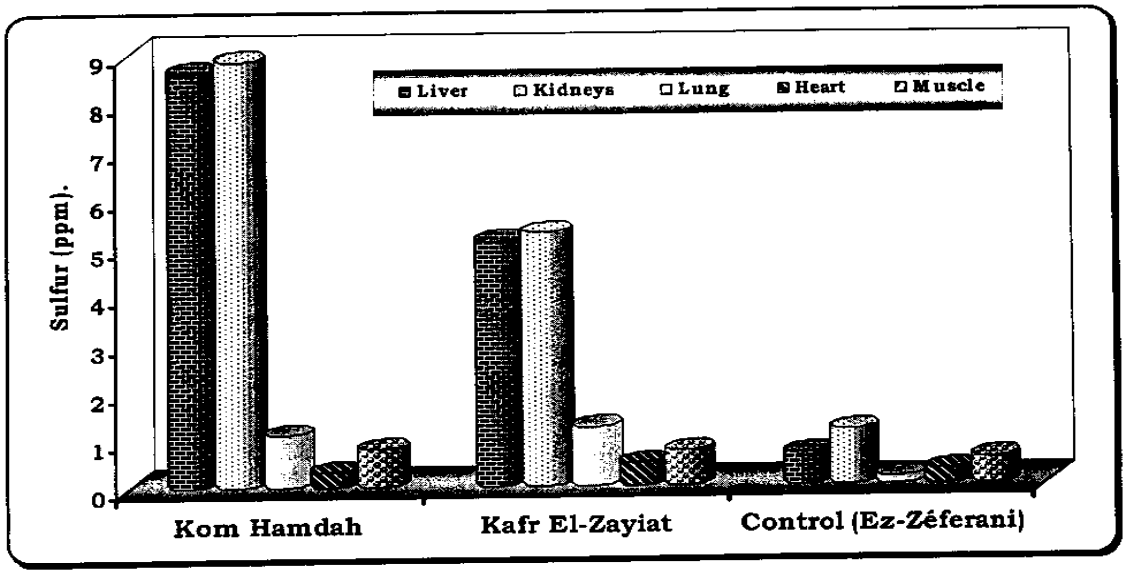


Fig. (19): Sulfur content (ppm) of soft tissues of buffaloes.

3. Cadmium concentrations
3.1. Serum and urinary cadmium:

Estimation of serum and urinary cadmium concentrations in cattle at various localities (Table, 9 and Figs., 20 & 21) showed significant levels only in *Mansheit Amin Esmaeil* (0.99 ± 0.13 ppm and 1.09 ± 0.12 ppm), and *Binûfar* (0.19 ± 0.06 ppm and 0.37 ± 0.09 ppm) localities, respectively. However, other studied localities recorded higher values but non-significantly differ from control area.

Similarly, tested sera and urine samples of buffaloes at the various localities revealed highly significant levels in *Mansheit Amin Esmaeil* (1.03 ± 0.12 ppm and 1.86 ± 0.14 ppm) followed by *Binûfar* (0.45 ± 0.06 ppm and 1.30 ± 0.10 ppm) and *Kafr Al-Aes* (0.27 ± 0.07 ppm and 0.97 ± 0.08 ppm) when compared with control values (0.12 ± 0.01 ppm and 0.15 ± 0.03 ppm), respectively as shown in Table (9) and Figs. (20 & 21).

3.2. Soft tissues Cadmium:

Data presented in Table (10) and Fig. (22) showed a significant increase in cadmium concentrations in kidney, liver and heart tissues in the studied areas when compared with their respective control. Concerning to lung and muscle, their cadmium contents do not differ significantly in all areas, but slightly higher in the studied areas than their respective controls.

Table (9): Cadmium concentrations (ppm) in serum and urine samples of cattle and buffaloes in the studied areas:

Area Locality	Serum		Urine	
	Cattle	Buffaloes	Cattle	Buffaloes
Kom Hamadah:				
Shabour	0.11 ± 0.06 bc	0.17 ± 0.04 d	0.24 ± 0.03 bc	0.22 ± 0.10 e
Mansheit Amin Esmaeil	0.99 ± 0.13 a	1.03 ± 0.12 a	1.09 ± 0.12 a	1.86 ± 0.14 a
Kafr Al-Aes	0.16 ± 0.07 bc	0.27 ± 0.07 c	0.25 ± 0.06 bc	0.97 ± 0.08 c
Kafr El-Zayiat:				
Kafr Hashad	0.10 ± 0.04 c	0.18 ± 0.03 d	0.21 ± 0.09 c	0.16 ± 0.09 e
Kafr El-Naseria	0.13 ± 0.04 bc	0.15 ± 0.04 d	0.16 ± 0.08 c	0.28 ± 0.05 de
Binûfar	0.19 ± 0.06 b	0.45 ± 0.06 b	0.37 ± 0.09 b	1.30 ± 0.10 b
Control (Ez-Zéferani)	0.09 ± 0.03 c	0.12 ± 0.05 d	0.14 ± 0.04 c	0.15 ± 0.03 e

-Means in the same column with similar letter (a-e) do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

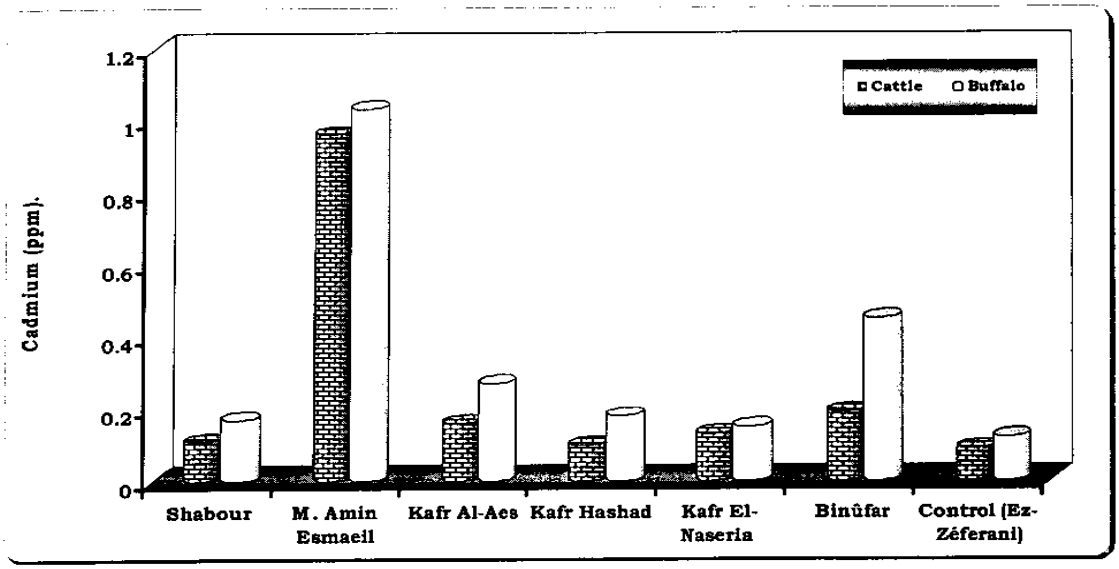


Fig. (20): Serum cadmium content (ppm) in cattle and buffaloes.

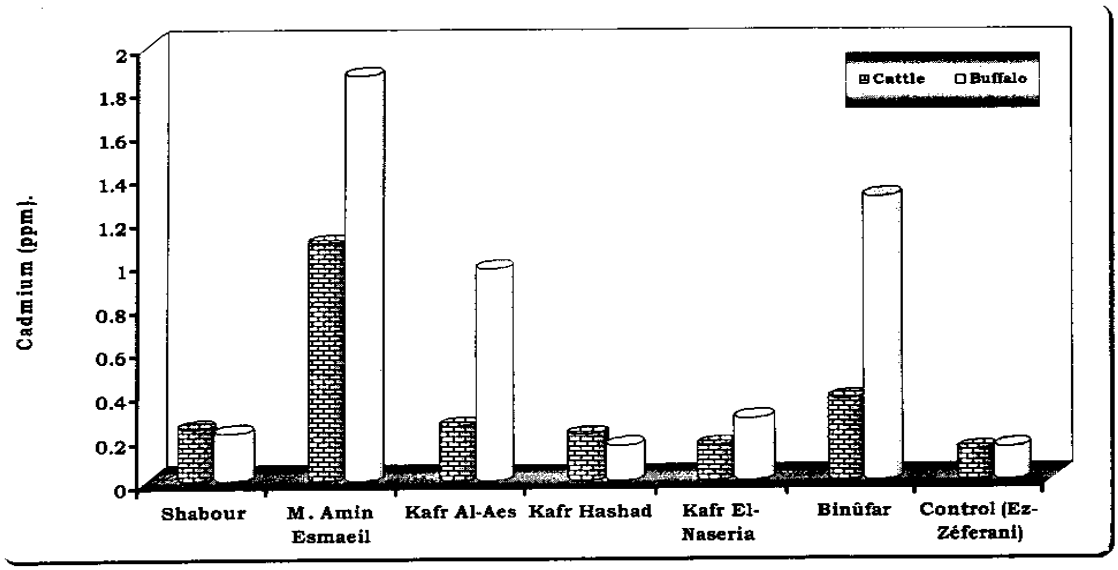


Fig. (21): Urinary cadmium content (ppm) in cattle and buffaloes.

Table (10): Cadmium concentrations (ppm) in the soft tissues of buffaloes at the studied areas:

Organ / Area	Liver	Kidneys	Lung	Heart	Muscle
Kom Hamadah	0.51 ± 0.12 a	1.26 ± 0.23 a	0.07 ± 0.001 a	0.03 ± 0.002 a	0.05 ± 0.007 a
Kafr El-Zayiat	0.53 ± 0.12 a	1.03 ± 0.39 a	0.07 ± 0.009 a	0.03 ± 0.003 ab	0.07 ± 0.009 a
Control (Ez-Zéferani)	0.08 ± 0.05 b	0.08 ± 0.02 b	0.04 ± 0.001 a	0.01 ± 0.001 b	0.03 ± 0.007 a

-Means in the same column with similar letter do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

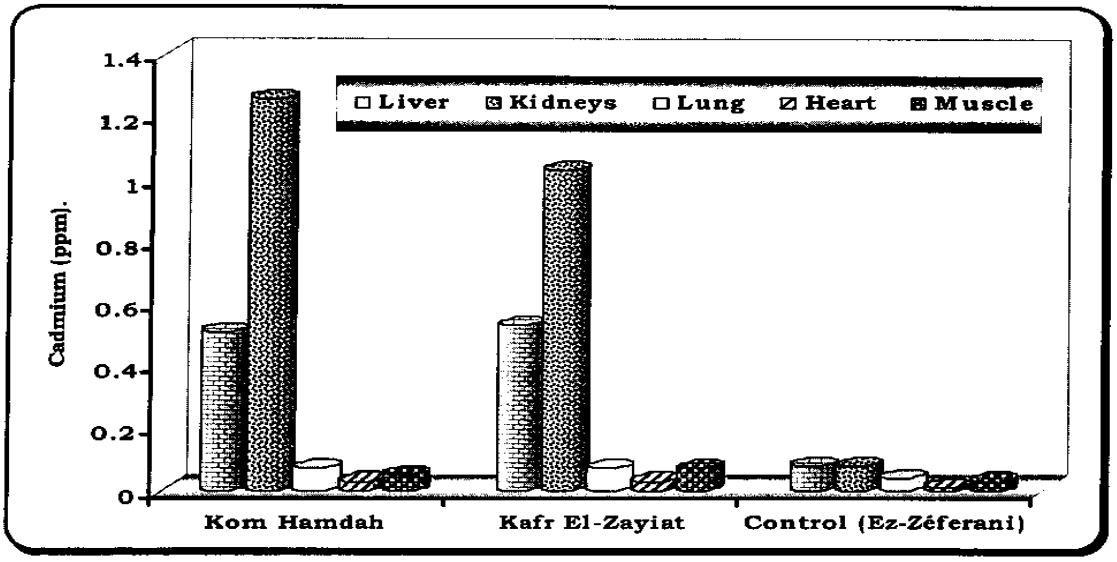


Fig. (22): Cadmium content (ppm) in soft tissues of buffaloes.

4. Calcium concentrations

4.1. Serum and urinary calcium:

Tested animals either cattle or buffaloes showed a significant lowered serum calcium content in *Mansheit Amin Esmaeil* and *Shabour* localities in comparison with control ones. Furthermore, the urinary excretion of calcium in cattle was significantly increased in all localities, while in buffaloes showed only a significant elevation at *Mansheit Amin Esmaeil* and *Shabour* localities when compared with their respective control animals (Table 11 and Figs. 23 & 24).

4.2. Soft and hard tissues calcium:

Data presented in Table (12) and Fig. (25) revealed a significant decrease in calcium concentrations of the renal and skeletal muscle samples collected from *Kom Hamadah* when compared to their respective control ones.

Regarding to the hard tissues calcium concentrations Table (13) and Fig. (26) showed non- significant changes between the studied areas and control ones, but their calcium values are higher than those of control and without any differences between *Kom Hamadah* and *Kafr El-Zayiat* values.

Table (11): Calcium concentrations (mg/dl) in serum and urine samples of cattle and buffaloes in the studied areas:

Area / Locality	Serum		Urine	
	Cattle	Buffaloes	Cattle	Buffaloes
Kom Hamadah:				
Shabour	10.3 ± 0.14 b	10.5 ± 0.69 b	363.7 ± 42.1 a	367.3 ± 19.1 a
Mansheit Amin Esmaeil	9.9 ± 0.72 b	10.1 ± 0.87 b	374.2 ± 52.9 a	359.2 ± 26.5 a
Kafr Al-Aes	11.8 ± 0.69 a	12.6 ± 0.9 a	351.2 ± 58.9 a	250.7 ± 41.8 c
Kafr El-Zayiat:				
Kafr Hashad	12.1 ± 0.44 a	11.2 ± 0.85 ab	336.8 ± 98.8 a	337.0 ± 34.5 ab
Kafr El-Naseria	11.1 ± 0.67 ab	11.0 ± 0.44 ab	324.7 ± 65.8 a	343.8 ± 36.5 ab
Binûfar	10.6 ± 0.97 ab	11.2 ± 0.38 ab	338.8 ± 35.6 a	324.8 ± 32.4 ab
Control (Ez-Zéferani)	13.1 ± 1.06 a	12.5 ± 1.02 a	250.4 ± 36.8 b	262.0 ± 19.1 bc

-Means in the same column with similar letter (a-c) do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

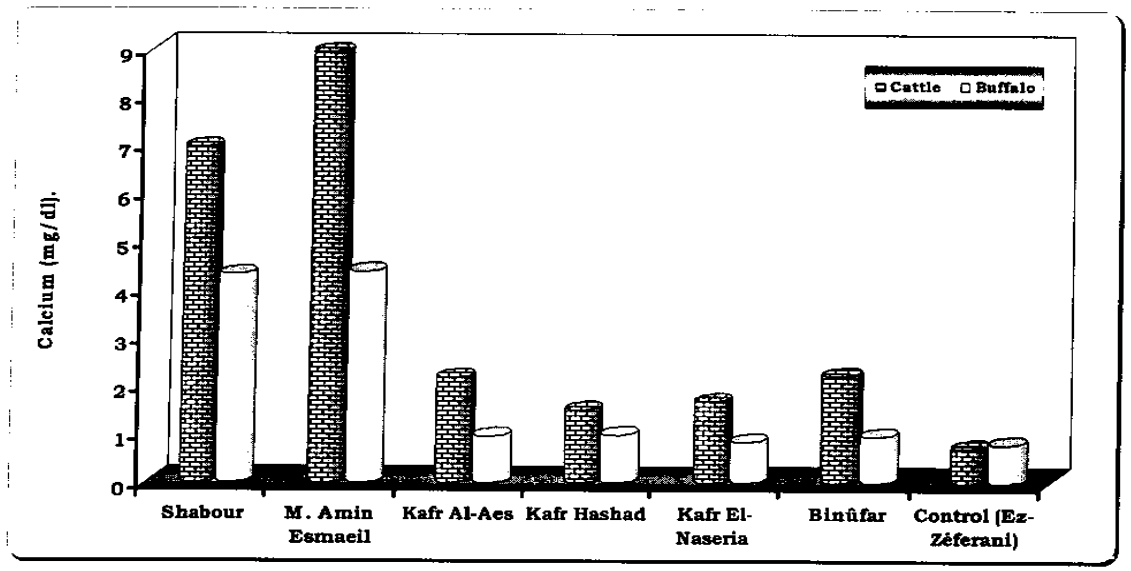


Fig. (23): Serum calcium content of cattle and buffaloes.

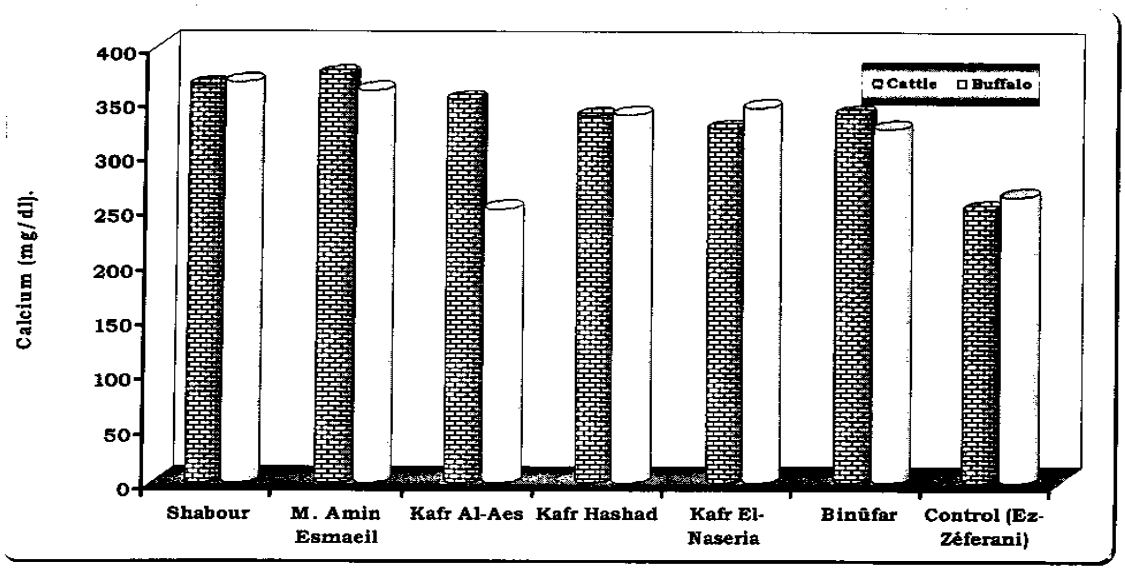


Fig. (24): Urinary calcium content of cattle and buffaloes.

Table (12): Calcium concentrations (mg/100 g, dry weight tissue) in the soft tissues of buffaloes in the studied areas:

Area \ Organ	Liver	Kidneys	Lung	Heart	Muscle
Kom Hamadah	10.53 ± 0.26 a	10.33 ± 0.33 b	13.33 ± 1.76 a	12.93 ± 1.42 a	10.93 ± 1.30 b
Kafr El-Zayiat	10.75 ± 0.83 a	12.08 ± 0.68 ab	12.48 ± 0.51 a	13.9 ± 0.40 a	13.53 ± 0.34 ab
Control (Ez-Zéferani)	10.95 ± 1.16 a	12.50 ± 0.65 a	15.30 ± 1.00 a	13.45 ± 1.31 a	14.18 ± 0.74 a

-Means in the same column with similar letter do not differ significantly at $P \leq 0.05$.
-Values are represented as (Means ± SE).

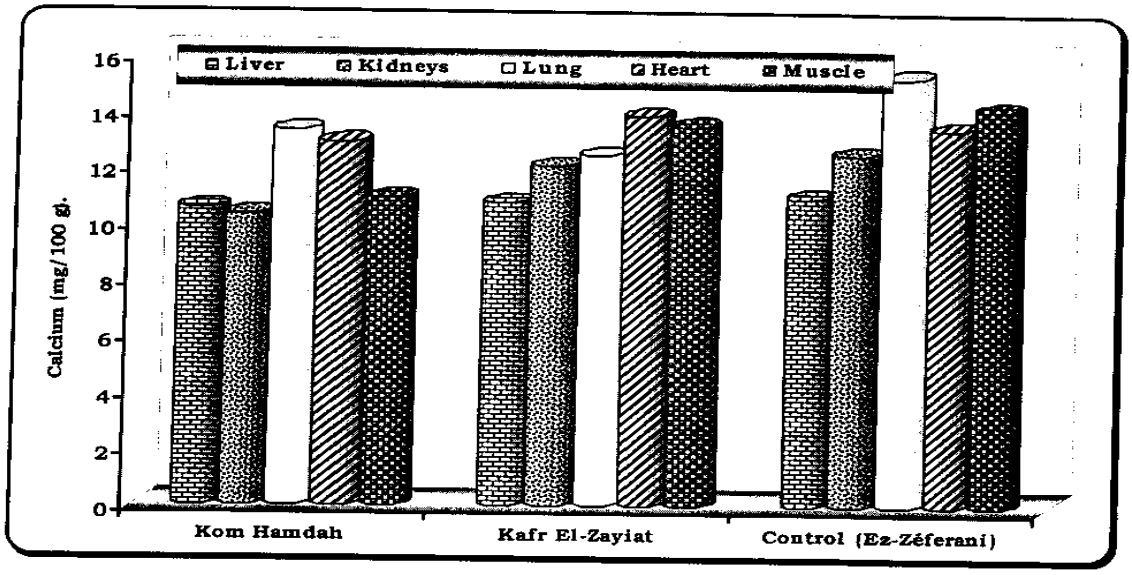


Fig. (25): Calcium content of soft tissues of buffaloes.

Table (13): Calcium concentrations (g/kg, dry weight tissue) in the hard tissues of buffaloes in the studied areas:

Area \ Tissue	Teeth			Mandible	Maxilla	Ribs
	Incisor	Premolar	Molar			
Kom Hamadah	200.5 ± 15.4 a	132.5 ± 27.7 a	156.5 ± 23.6a	195.3 ± 16.7 a	187.5 ± 45.9 a	123.4 ± 41.9a
Kafr El-Zayiat	195.5 ± 12.9 a	177.8 ± 11.8 a	195.7 ± 10.4a	226.8 ± 19.7 a	214.6 ± 22.7 a	168.5 ± 13.0a
Control (Ez-Zéferani)	207.4 ± 10.8 a	178.9 ± 16.1 a	166.9 ± 25.6a	249.2 ± 30.4 a	222.8 ± 21.7 a	191.1 ± 12.2a

-Means in the same column with similar letter do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

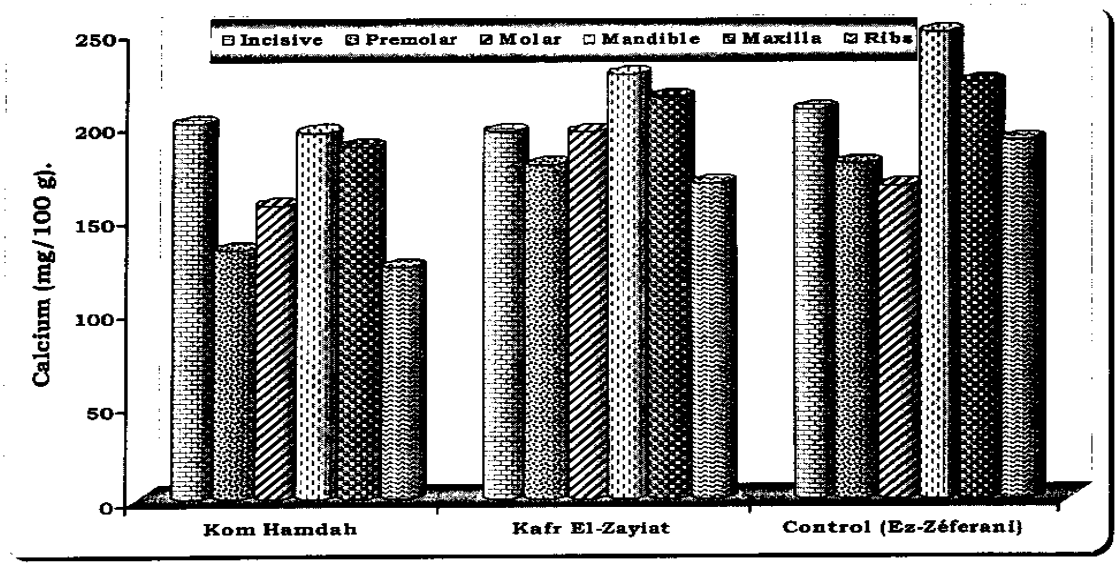


Fig. (26): Calcium content of hard tissues of buffaloes.

5. Inorganic phosphorous concentrations

5.1. Serum and urinary inorganic phosphorous:

Estimation of serum inorganic phosphorous levels revealed a highly significant decrease in cattle and buffaloes at *Kom Hamadah* localities except buffalo *Kafr Al-Aes* when compared with their respective control values (Table, 14 and Fig., 27).

The urinary inorganic phosphorous content in cattle and buffaloes as presented in the Table (14) and Fig. (28) revealed significant excretion of higher amounts in most of studied localities with highest concentrations noticed in *Kom Hamadah* localities when compared with other localities and control area.

5.2. Soft and hard tissues inorganic phosphorous:

Our results revealed a non-significant decrease in the inorganic phosphorous content either in the soft and hard tissues in the studied areas when compared with control as noticed in Tables (15 & 16) and Figs. (29 & 30).

Table (14): Inorganic phosphorous concentrations (mg/dl) in serum and urine samples of cattle and buffaloes in the studied areas:

Area / Locality	Serum		Urine	
	Cattle	Buffaloes	Cattle	Buffaloes
Kom Hamadah:				
Shabour	3.7 ± 0.42 b	3.8 ± 0.94 c	12.0 ± 4.5 abc	10.3 ± 0.82 ab
Mansheit Amin Esmaeil	3.5 ± 0.33 b	3.6 ± 0.33 c	15.1 ± 1.7 a	10.8 ± 1.76 ab
Kafr Al-Aes	4.0 ± 0.30 b	5.3 ± 2.4 b	10.3 ± 2.5 bc	12.4 ± 2.08a
Kafr El-Zaylat:				
Kafr Hashad	6.6 ± 0.45 a	6.7 ± 0.58 a	5.7 ± 3.7 de	7.5 ± 1.02 bc
Kafr El-Naseria	6.0 ± 0.48 a	6.1± 0.46 ab	9.9 ± 4.3 cd	9.3 ± 1.92 abc
Binûfar	5.6 ± 0.07 a	5.9 ± 0.33 ab	13.2 ± 4.0 abc	10.6 ± 1.55 ab
Control (Ez-Zéferani)	6.0 ± 1.1 a	6.6 ± 0.45 ab	5.3 ± 2.6 e	5.7 ± 0.82 c

-Means in the same column with similar letter (a-e) do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

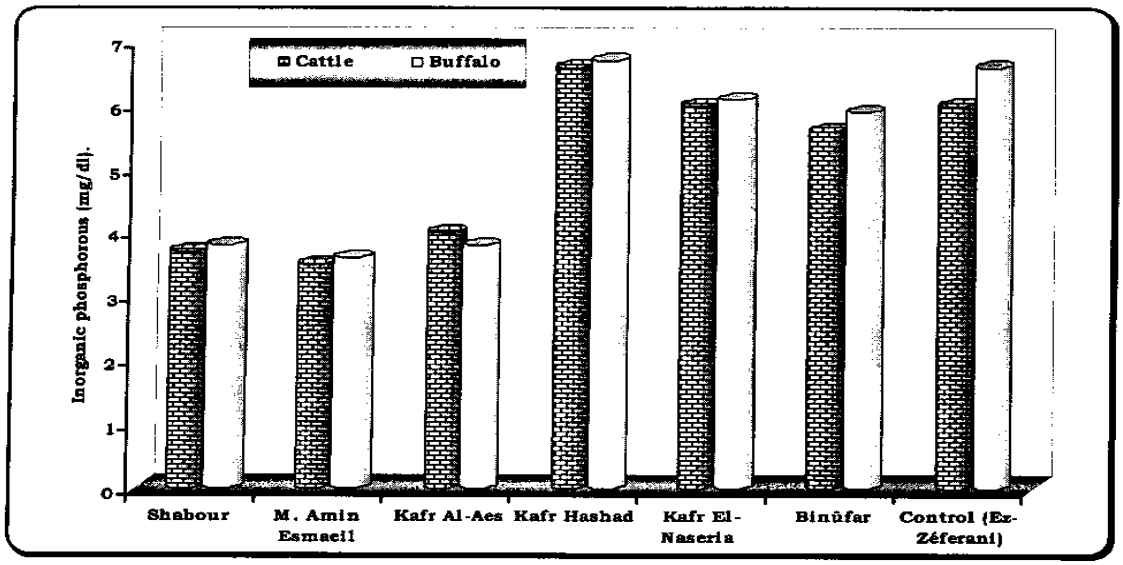


Fig. (27): Serum inorganic phosphorous content of cattle and buffaloes.

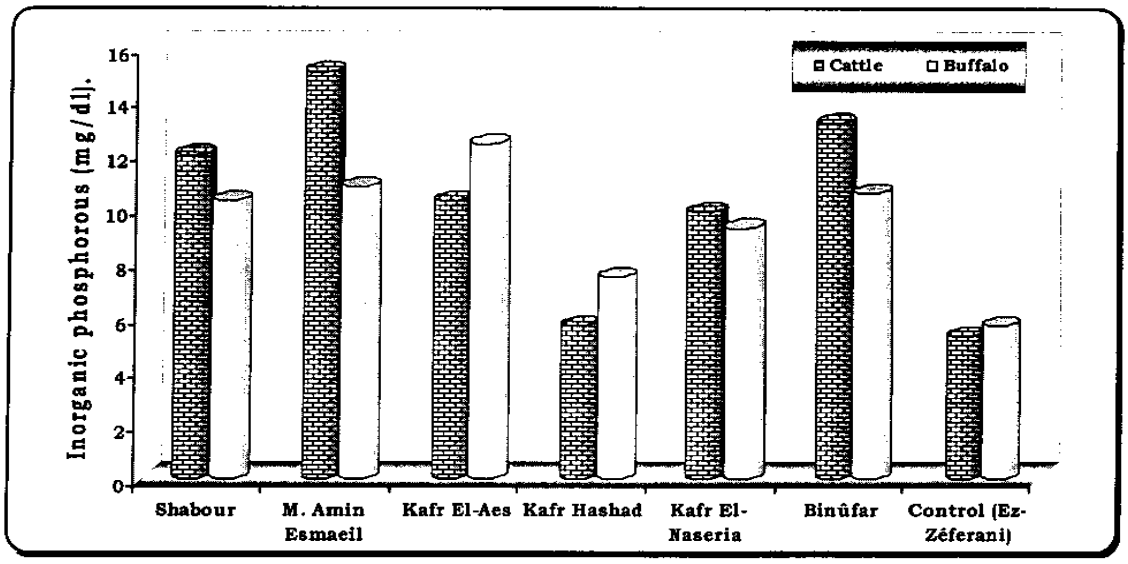


Fig. (28): Urinary inorganic phosphorous content of cattle and buffaloes.

Table (15): Inorganic phosphorous concentrations (mg/100 g on dry weight basis) in the soft tissues of buffaloes in the studied areas:

Area \ Organ	Liver	Kidneys	Lung	Heart	Muscle
Kom Hamadah	8.3 ± 0.92 a	8.6 ± 0.47 a	8.6 ± 0.79 a	8.7 ± 0.21 a	7.9 ± 1.09 a
Kafr El-Zayiat	8.5 ± 0.23 a	7.7 ± 0.92 a	9.3 ± 0.72 a	8.5 ± 0.74 a	9.0 ± 0.66 a
Control (Ez-Zéferani)	9.5 ± 1.10 a	7.9 ± 0.69 a	10.3 ± 0.24 a	10.0 ± 0.81 a	9.6 ± 0.55 a

-Means in the same column with similar letter do not differ significantly at $P \leq 0.05$.
-Values are represented as (Means ± SE).

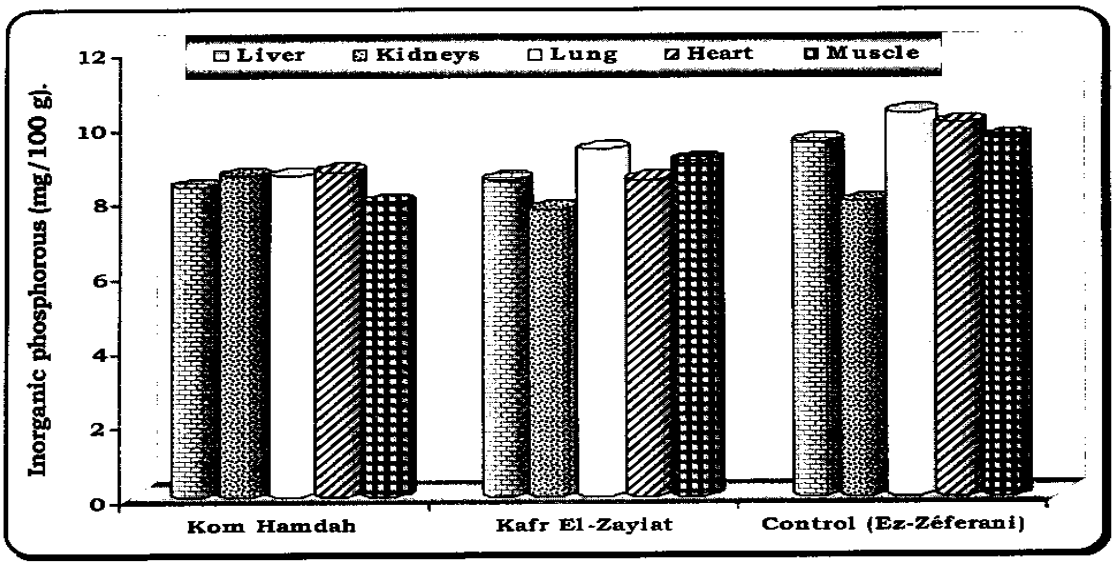


Fig. (29): Inorganic phosphorous content of soft tissues of buffaloes.

Table (16): Inorganic phosphorous concentrations (g/kg, dry weight basis) in the hard tissues of buffaloes in the studied areas:

Tissue / Area	Teeth			Mandible	Maxilla	Ribs
	Incisor	Premolar	Molar			
Kom Hamadah	163.5 ± 10.38a	177.4 ± 21.21a	178.2 ± 15.32a	165.5 ± 4.31a	168.5 ± 14.59a	164.5 ± 12.05a
Kafr El-Zayiat	133.4 ± 21.54a	192.7 ± 22.13a	209.9 ± 10.67a	174.0 ± 12.82a	155.2 ± 25.26a	173.1 ± 9.24a
Control (Ez-Zéferani)	149.6 ± 19.40a	207.7 ± 16.82a	188.9 ± 24.83a	172.0 ± 7.45a	178.8 ± 10.09a	181.3 ± 14.43a

-Means in the same column with similar letter do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

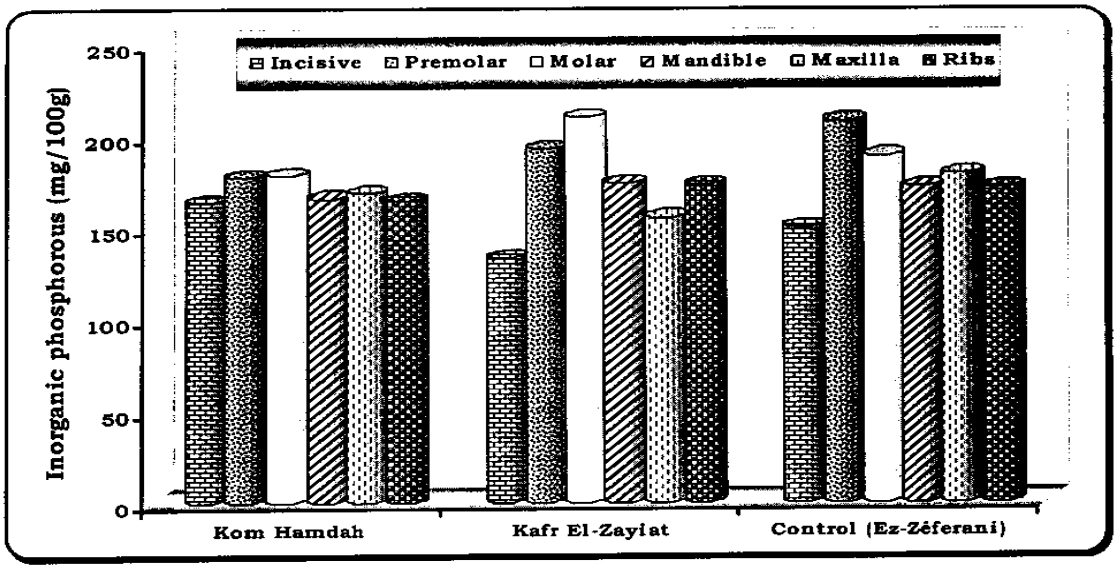


Fig. (30): Inorganic phosphorous content of hard tissues of buffaloes.

6. Biochemical Blood Parameters:

6.1. Liver function tests:

Analytical finding of serum proteins in cattle and buffaloes as shown in Tables (17 & 18) revealed significant decrease in serum total proteins in *Kom Hamadah* locality, and serum albumin in all studied localities, whereas the serum globulin content recorded a significant increment only in buffaloes nearly at all areas when compared with control one.

Activities of liver transaminases (ALT and AST) are rarely affected and within normal physiological limits as observed in Tables (17 & 18), where ALT activity was increased significantly in cattle of *Mansheit Amin Esmaeil* locality. The ALP enzyme activity in cattle and buffaloes was significantly increased especially in *Mansheit Amin Esmaeil, Shabour and Binūfar* localities when compared with their respective control.

6.2. Kidney function tests:

The levels of blood urea and serum creatinine in cattle and buffaloes were significantly increased in *Mansheit Amin Esmaeil* when compared with control values (Tables, 17 & 18).

Table (17): Some serum biochemical parameters in cattle in the studied areas:

Area	Locality	Serum proteins			Hepatic enzymes			Renal tests	
		TP (g/dl)	AL (g/dl)	GL (g/dl)	ALT (IU/L)	AST (IU/L)	ALP (IU/L)	Urea (mg %)	Creatinine (mg %)
Kom Hamadah:-									
	Shabour	5.2±0.2 c	4.0±0.1 d	1.4±0.2 abc	11.9±1.1 c	37.2±3.3 bc	164.1±11.1 a	25.9±2.3 bcd	0.47±0.04 bc
	Mansheit Amin Esmaeil	5.2±0.1 c	4.1±0.1 cd	1.1±0.2 c	17.5±1.4 a	42.3±3.8 ab	175.9±11.8 a	45.2±4.1 a	0.70±0.05 a
	Kafr Al-Aes	5.9±0.2 bc	4.5±0.2 bcd	1.5±0.2 abc	13.6±1.4 bc	37.1±4.2 bc	138.5±14.6 ab	28.3±3.3bc	0.51±0.05 b
Kafr El-Zayiat:-									
	Kafr Hashad	6.6±0.2 a	4.8±0.2 bc	1.8±0.2 ab	10.5±0.99 cd	36.7±2.95 c	66.8±18.2 b	15.6±2.2 d	0.33±0.02 d
	Kafr El-Naseria	6.2±0.3 ab	4.3±0.2 bcd	1.9±0.3 a	12.0±1.2 ab	36.3±4.1 c	104.4±14.9 ab	23.6±3.4 cd	0.37±0.04 cd
	Binûfar	6.1±0.2 ab	4.9±0.2 b	1.2±0.1 bc	11.9±1.2 c	32.2±2.83 cd	164.0±25.4 a	34.3±4.8 b	0.45±0.03 bcd
Control (Ez-Zéferani)		6.8±0.5 a	5.8±0.4 a	1.0±0.2 bc	10.7±1.7 cd	38.0±3.2 bc	90.5±9.9 b	25.5±8.2 bcd	0.45±0.08 bcd

-Means in the same column with similar letter (a-d) do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

Table (18): Some serum biochemical parameters in buffaloes in the studied areas:

Area / Locality	Serum proteins			Hepatic enzymes			Renal tests	
	TP (g/dl)	AL (g/dl)	GL (g/dl)	ALT (IU/L)	AST (IU/L)	ALP (IU/L)	Urea (mg %)	Creatinine (mg %)
Kom Hamadah:-								
Shabour	5.6±0.17 c	4.4±0.14 c	1.2±0.12 bc	14.0±1.19 ab	43.1±3.50 a	184.4±30.3 a	22.3±2.7 b	0.42±0.05 c
Mansheit Amin Esmaeil	5.5±0.19 c	4.0±0.08 cd	1.5±0.20 ab	14.8±1.38 a	44.2±3.20 a	186±19.2 a	35.1±5.8 a	0.67±0.07 a
Kafr Al-Aes	5.8±0.24 bc	4.2±0.18 c	1.6±0.24 ab	13.3±1.22 ab	40.3±2.77 a	145.8±14.7 ab	28.7±3.4 ab	0.51±0.05 abc
Kafr El-Zayiat:-								
Kafr Hashad	6.8±0.21 a	5.1±0.15 b	1.7±0.16 ab	11.6±1.01 ab	37.8±3.70 ab	93.4±15.3 b	22.4±2.4 b	0.49±0.06 bc
Kafr El-Naseria	6.3±0.29 ab	4.2±0.16 c	2.1±0.36 a	13.1±1.56 ab	36.8±3.01 ab	102±24.2 ab	25.7±4.1 ab	0.40±0.04 c
Binûfar	5.9±0.27 bc	4.2±0.12 c	1.8±0.18 ab	10.9±0.90 b	35.0±4.75 b	177±6.5 ab	31.4±4.4 ab	0.59±0.05 ab
Control (Ez-Zéferani)	6.9±0.29 a	6.0±0.34 a	0.9±0.13 c	10.2±1.37 ab	32.2±4.02 ab	95.0±17.6 b	20.7±4.7 b	0.37±0.04 bc

-Means in the same column with similar letter (a-d) do not differ significantly at $P \leq 0.05$.

-Values are represented as (Means ± SE).

5. DISCUSSION

Industrial evolutions, the intense use of raw materials and agriculture technology have all somehow improved our lifestyle while simultaneously polluting the natural environment. Air pollution of local or distant origin may contribute significantly to the load metals in natural terrestrial ecosystems. For many years environmental pollution has been considered to be a hazard to health and the industrial pollution caused a serious and world widespread damage to the domestic animals. The release of waste products from the super-phosphate fertilizer factory and brick kilns at *Kom Hamadah* (Behera province) and *Kafr El-Zayiat* (Gharbeia province) may become toxic by continuous inhalation and/or ingestion of water and feed contaminated with fluoride, sulfur and cadmium compounds. In this study, we get an attempt to evaluate the contribution of these toxic metals on the health of the livestock.

I. Analytical findings of macro-environmental samples:

The study of the macro-environment (air, water and feed) is the initial basic step in evaluating toxic waste hazards on animals and human beings.

1. Water:

One of our critical aim in this study was to evaluate the problem of River Nile pollution at different locations representing different distances (every 0.5 km) and directions (with and against water way) from the source of pollution. Since measurement of the pollution hazards depending upon the first instance of area considered as control. For that, *Ez-Zéferani* area was chosen for this purpose as it is located 20 km western to the source of

pollution, in which the toxicological study revealed no clinical signs pointing to exposure to any hazardous substances.

The mean fluoride, sulfur and cadmium concentrations had been reached 5.8 ± 0.29, 8.9 ± 0.36 and 0.33 ± 0.11 ppm, respectively at the drainage system of the *Kafr El-Zayiat* super-phosphate factory that fall out directly into the River Nile water indicating the main source of pollution by these elements at neighboring areas.

The fluoride concentration in River Nile water was significantly elevated at super-phosphate factory (*Mansheit Amin Esmaeil*), and then gradually decreased up to 2.5 km with water stream after the super-phosphate factory (*Binûfar* and *Kafr Al-Aes*) and 0.5 km before the factory. On the other hand, the significant increase of fluoride at *Shabour* locality that located at about 2.5 km south-western to the super-phosphate factory and against water stream was attributed mainly to the smog of more than 50 brick kilns.

In addition, the sulfur and cadmium concentrations in water behave similarly as fluoride with water flow up to 1.5 km after the super-phosphate factory. However, sulfur recorded significant elevation against water stream, and this was attributed to the fumes and dusts of either super-phosphate factory and/or brick kilns. In this respect, it seems that water stream has a considerable role in the distribution of these toxic pollutants.

The results in Table (2) and Fig (2) indicated that the highest concentrations of fluoride, sulfur and cadmium in River Nile water were recorded at *Mansheit Amin Esmaeil* that was closely facing the super-

phosphate factory and under the effect of multiple brick kilns, followed by *Binūfar* (1.5 km south-western to super-phosphate factory), then *Shabour* in respect to fluoride and sulfur (2.5 km south-western to the super-phosphate factory and southern to about 50 brick kilns on the River Nile).

The results of fluoride concentrations in River Nile water in the studied areas are parallel with those obtained by ***Ibrahim (1983)*** and ***Sayed (2001)*** who estimated the fluoride in other localities in Assiut province, Egypt which located under the effect of Assiut super-phosphate factory. ***NRC (1974)*** stated that the fluoride concentration in water is considered in the harmful limits when reaches more than 1 ppm. It is also observed that the consumption of water contaminated by the fumes and dusts emitting from the industrial sources resulted in the development of chronic fluorotic lesions in the different animal species where the fluoride concentration in the drinking water is varied between 1.5-4 ppm ***(Dwivedi et al., 1997; Choubisa, 1999*** and ***Patra et al., 2000)***.

Also, estimation of sulfur in water at the investigated areas is nearly corresponding to that recorded in *El-Twabiya* locality (1 km north-eastern to Assiut super-phosphate factory on the River Nile) by ***Sayed (1995).*** Sulfur concentrations in the water samples are much lower than the maximum contaminant levels for sulfates in drinking water standards; 250 mg/L ***(Csuros, 1997).***

Estimation of cadmium in the River Nile water at the investigated localities revealed significant elevation at *Mansheit Amin Esmaeil* due to their close location to super-phosphate factory. Such results are in

agreement with those of ***Sayed (1995)*** who concluded that cadmium concentrations were attributed to several factories a long the course of the River Nile at the Manqabad city, Assiut province, Egypt. These results are higher than that recommended in the international standards of ***U.S. EPA (1986)*** quality criteria for water, which is 0.01 mg/L. In natural fresh water, cadmium sometimes occurs at concentrations as low as 0.01 µp/L, but in environments impacted by man activities, concentrations may be several micrograms per liter or greater.

2. Feed stuff:

Regarding to fluoride concentration in the feed stuff, it was significantly increased in the selected feed stuff; tibn and drees samples at *Mansheit Amin Esmaeil* (5.4 ± 0.52 and 4.3 ± 0.64 ppm), *Shabour* (1.7 ± 0.61 and 3.7 ± 1.04 ppm), *Binūfar* (1.9 ± 0.08 and 2.7 ± 0.21 ppm) and *Kafr Al-Aes* (1.81 ± 0.35 and 2.6 ± 0.75 ppm), respectively in comparison with control area (0.006 ± 0.0003 and 0.18 ± 0.04 ppm), respectively. Our results were in agreement with those obtained by ***Sayed (1995*** and ***2001).*** However, these levels were lesser than those recorded by ***Ibrahim (1983)*** and ***Seddek (1988)***, which ranged from (7.33 ± 1.33 ppm) to (32.0 ± 2.31 ppm) in tibn and drees at Assiut province.

From our results, it was found that the significantly increased levels of fluoride in feed stuff were inversely correlated to the distance from the super-phosphate factory as the highest recorded levels were observed in *Mansheit Amin Esmaeil* (facing the super-phosphate factory on the River Nile), followed by *Binūfar* (1.5 km south-western to super-phosphate factory) and *Kafr Al-Aes* (2.5 km south-western to the super-phosphate factory and

southern to some brick kilns) localities. On the other hand, the significant fluoride concentrations in feed stuff at *Shabour* locality was attributed to both effects of super-phosphate factory emissions and smog of brick kilns. ***Suttie and Faltin (1971)*** stated that short term exposure of calves to fluoride (2-5 mg/kg ration daily) was sufficient to induce dental fluorosis in one pairs of incisive teeth. For that the feedstuff in the areas surrounding either the super-phosphate or brick factories contain fluoride be able to cause fluorosis problem in livestock.

The analytical finding of sulfur in the feed stuff revealed significant elevation in tibn samples collected from all localities except *Kafr Hashad* when compared with their respective control values. Again, the highest estimated concentrations of sulfur in both tibn and drees samples were observed in *Mansheit Amin Esmaeil* (7.1 ± 0.05 and 5.4 ± 0.29 ppm) and *Shabour* (5.8 ± 0.68 and 4.3 ± 0.16 ppm) localities, respectively. These results were in the range of sulfur concentrations recorded by ***Ibrahim (1983)*** and ***Seddek (1988).***

Beke and Hironaka (1991) and ***NRC (2000)*** considered that the recommended and maximum tolerable dietary concentrations of sulfur are 0.15% and 0.4% on a dry matter basis, respectively and any level greater than 0.4% sulfur is toxic. Therefore, the sulfur content recorded in feed stuff in *Mansheit Amin Esmaeil* and *Shabour* localities may play a role in the general body changes noticed in animals reared at such locations, as those reported by ***Ibrahim (1983)*** and ***Selim et al., (2000).***

On the other hand, the highest cadmium content in feed stuff samples was noticed in *Mansheit Amin Esmaeil* and *Binūfar* when compared with their respective control values. This may be explained on the basis of the cadmium content of feed stuff was nearly correlated with that of water in the same localities and inversely related to distance from highway automobile emissions, confirming our suggestion for the source of cadmium pollution. These recorded data are higher than the recorded normal cadmium levels in feed stuff at USA; less than 0.05 ppm ***(Takayuki and Leonard, 1993)***. The obtained results were nearly in concord with ***Sayed (2001)*** study's that revealed a significant increase in cadmium content in wheat straw samples grown near to the super-phosphate factory at Assiut province which ranged from 0.03 to 0.06 mg/kg.

Estimation of total fluoride, sulfur and cadmium intake from the water and feed indicated their ingestion by exposed animals similar to those causing ailments in experiments using added toxic compounds in the water, concluding that the tolerance of cattle and buffaloes to the fluoride, sulfur and cadmium content in the water was dependent upon their content of the feed.

II. Clinical Toxic Signs:-

The average age of the studied animals was ranged from 2 to 5.5 years old. There were a general complains of farmers in the areas of the study with a history of seasonally recurrent and therapeutically unresponsive lameness, with unthriftness, anorexia, general weakness and emaciation in some animals especially those lived for long period in the affected areas.

The signs indicating fluoride intoxication in cattle was mainly confined to dental changes as brown to blackish discoloration, pitting and attrition of incisors and irregular enamel surface (Figs. 4 and 5). These signs were nearly in accordance with those of ***Krook and Maylin (1979)*** and ***Maiti et al. (2003)***. These signs become worth in buffaloes, where they were severe and represented in brown to dark brown or even blackish discoloration in severely affected buffaloes with mottling of all permanent teeth especially pre-molar and molar teeth when compared to incisors (Figs. 9, 10, 11 and 12). However, attrition was so severe with oblique eruption observed in most clinically affected incisive teeth. Similar signs were documented by ***Griffith-Jones (1977), Ibrahim (1983)*** and ***Singh and Swarup (1994).*** This may be due to that the permanent teeth erupt without exfoliation of the deciduous predecessors, the retained deciduous teeth deviates the advance of permanent teeth from the normal either labially or linguinally to the retained deciduous teeth ***(Krook et al., 1983).***

In addition, most of the affected buffaloes suffered from a pronounced lameness that was intermittent and recurrent annually in summer months, shifting from leg to leg (Fig. 6), these symptoms are in accordance with the description of fluorosed lamed animals by ***Shupe and Olson (1971)*** and ***NRC (1980).*** During movement, the animal showing abducted elbow and arched back with prominent bony extremities and weak musculature of lamed legs (Fig. 7). The severely lamed animal was unwilling to stand, cannot bear weight, and became recumbent for long period ended by its slaughtering (Fig. 8). Similar toxic signs of fluoride were demonstrated nearly in all animal species by ***Griffith-Jones (1977), Araya et al. (1990),***

Jubb et al. (1993), Singh and Swarup (1994), Dwivedi et al. (1997), Choubisa (1999) and ***Patra et al. (2000).***

There was no any signs of bony exostosis observed in affected animals and this in agreement with the results of ***Suttie et al. (1985)*** and ***Karram et al. (1989)*** in their studies in deer and camel intoxication by fluoride, but not coincide with the results obtained by ***Araya et al. (1990), Raghib et al. (1993), Singh and Swarup (1994), Dwivedi et al. (1997)*** and ***Patra et al. (2000).*** However, there was only peri-odentosis had been observed in severely intoxicated buffaloes (Fig. 12). These may be attributed to the seasonal changes in the feeding diets as a nutritional factor and the affected animals received low doses of fluoride over a prolonged period not allowed to cause osteoporosis and/or periosteal hyperostosis. Besides, ***Ibrahim (1983)*** stated that differences in clinical toxic signs did not only exist between different species but also within the same species and the same host due to many factors.

The specific clinical signs of sulfur and/or cadmium toxicosis as cough, respiratory distress or nervous symptoms had not been encountered in our cases. The results did not match with those noticed by ***Ibrahim (1983), Gunn et al. (1987), Zinn et al. (1997), Liu (2003)*** and ***Stoev et al. (2003).*** Furthermore, examination of affected animals revealed that they suffered from cachexia, poor general health condition, emaciation, paleness of mucous membrane, rough dry coat losing its luster appearance and easily detached, and drop in milk yield with an incidence of reproductive failure as general toxic signs (Figs. 6 and 7). These results were noticed in cases of chronic fluoride, sulfur and/or cadmium toxicosis by ***Weeth and***

Capps (1972), Ammerman et al. (1980), Ibrahim (1983), Radostits et al. (1994), Selim et al. (2000), NRC (2001), Swerczek (2001) and ***Liu (2003).*** Consequently, our results suggested that the disease of affected animals might be as a multifaceted interactions between fluoride, sulfur and cadmium, primarily due to environmental pollution by industrial activities and their greatest degree of disruptions in elements homeostasis such as calcium, phosphorous, copper, molybdenum, zinc, iron and selenium as discussed by ***Smith et al. (1991), Ivancic and Weiss (2001), Liu (2003), Sedki et al.*** *(2003)* and ***Miranda et al. (2005).***

III. Analytical findings of micro-environmental samples:-

1. Fluoride:

Analytical findings of serum fluoride concentrations as shown in Table (4) and Figs. (13 & 14) revealed a highly significant elevation in both cattle and buffaloes at *Mansheit Amin Esmaeil* locality that facing the *Kafr El-Zayiat* super-phosphate factory and *Shabour* locality that sited underneath the wind direction of the smog emitted from brick kilns zone. On the other hand, the urinary fluoride revealed the maximum concentrations that reached about 15-fold than control, whereas the value of serum not exceed 7-folds the normal ones. In compliance, the study of ***Ibrahim (1983)*** revealed that the urinary and serum fluoride values was reached about 5- and 2-folds, respectively as their respective control values in buffaloes, and with ***Radostits et al. (1994)*** who stated that the normal cattle have urinary and blood fluoride levels up to 6 and 2 ppm, respectively. These data revealed a close positive relationship between serum and urinary fluoride levels ***(Singh and Swarp, 1994*** and ***Dwivedi et al. 1997).***

In spite of the normal serum fluoride levels noticed in the animals of *Kafr Al-Aes* and *Binûfar* localities, the urinary fluoride levels were higher and significant when compared with their respective control values (Table, 4 and Figs., 13 & 14). This in synchronization, with those of ***Radostits et al. (1994)*** who concluded that cattle on fluoride intakes sufficient to cause intoxication may have urine levels of 16-68 ppm, even though the blood levels are often normal.

Consequently, it could be concluded that urinary fluoride is more indicative however in the initial stages of harmful exposure to fluoride. This is of great significance in the early detection of fluorosis due to the truth that the major part of fluoride intake was excreted via urine, and even after diminution of signs, the urinary fluoride excretion may remain sometimes elevated as reported earlier by ***Ibrahim (1983), Shehata et al. (1989), Vandersmissen et al. (1993)*** and ***Patra et al (2000).***

Also, these results indicated that the levels of serum and urinary fluorides in the affected areas were inversely proportional to the distance from the source of pollution and the direction of wind. These results were in concurrence with those obtained by ***Griffith-Jones (1977), Krook and Maylin (1979), Ibrahim (1983), Raghib et al. (1993)*** in the different animal species.

Concerning the variations between cattle and buffaloes in serum and urinary fluoride levels, our data indicated that buffaloes recorded the higher values more than cattle either in serum (up to 7-Vs 6-folds) or urine (up to 15- Vs 11-fold) respectively, in relation to control fluoride concentrations in

each variety. This may be attributed to the higher intake of feed and water by buffaloes more than cattle in relation to their body weight, thus they exposed to more fluoride ***(Dwivedi et al., 1997)***.

Analysis of fluoride in the parenchymatus organs revealed a significant elevation only in renal tissue about 2- folds the control levels in *Kom Hamadah* and *Kafr El-Zayiat* areas, while other soft tissues revealed non-significant elevation in their fluoride contents not more than 1.03 ppm (Table, 5 and Fig., 1 5). Similar data were reported by ***NRC (1980), US EPA (1980), Milhaud et al. (1983)*** and ***Kessabi et al. (1983)*** where kidneys usually exhibit a high fluoride concentration during high-fluoride ingestion due to urine retention in the tubules and collecting ducts. These data supports our previous suggestion that the urine is the major route of fluoride excretion.

Formerly, ***Ibrahim (1983)*** revealed a highly significant elevation of fluoride levels not only in buffaloes' kidneys but also in muscle rendering them playing a significant role in the interrelationship between animal-human toxicity with fluoride. While it is worthy to state here the safe use of most organs of intoxicated animals by the human being and their fluoride concentrations are undependable indicators for the degree of fluorosis.

Unlike soft tissues, Table (8) and Fig (16) shown that there is a highly significant increase in fluoride level in all samples of bony tissues ranged from 1300 to 5300 ppm which caused dental fluorosis without osteoflourosis as clinically observed when compared with their respective controls values. The fluoride deposition in the bony tissues is one of most

reliable indicators of ingested fluoride, as recorded by many authors ***(Obel and Erne, 1971; Shupe et al., 1992*** and ***Jones et al., 1997).*** In addition, the maximum fluoride levels were observed in cheek teeth and mandible followed by incisive teeth and maxilla, while the ribs contained the lowest values of fluoride. Our results are in harmony with those obtained by ***Mortenson, et al. (1964).*** Anywhere, ***Puls (1994)*** recorded that the leg bones generally contain half as much as ribs, and mandibles and ribs are the best bones for determining fluoride status. However, ***Ibrahim (1983)*** reported an evident distribution of fluoride in hard tissues especially in ribs followed by mandibles and incisive teeth. These results are in agreement with those of ***Mortenson et al. (1964)*** and ***Obel and Erne (1971).***

2. Sulfur:

Analysis of serum and urinary sulfur revealed a significant elevation in cattle at all localities except *Kafr Hashad* when compared with *Ez-Zéferani* area, attributing that to the air pollution by sulfur resulting from gasoline combustion used in brick kilns in affected areas, where the highest levels were observed in *Mansheit Amin Esmaeil and Shabour*, and the extra effect of water sources and feed stuff contamination by sulfur and sulfur–compounds (SO_2 and SO_3) emissions that liberated from super-phosphate factory which use sulfuric acid as reported by ***Ibrahim (1983), Kandylis (1984)*** and ***Sayed (1987).*** While in buffaloes, the highly significant elevation of serum and urinary sulfur was observed only at *Mansheit Amin Esmaeil* and *Shabour* when compared with other localities and control area as recorded in Table (7) and Fig. (17), similar to the trend of fluoride. It was observed that cattle were more affected than buffaloes besides the sulfur values are higher, this may be related the higher metabolic rate and

activities of former species. These results are nearly supported by those of water and feed stuff in the same localities.

The data presented in Tables (7) and Figs. (17 & 18) indicated that the sulfur is excreted in the urine of cattle and buffaloes in significant amounts indicating that the urine is the main route of sulfur excretion. These results are in agreement with those obtained by ***Ibrahim (1983)*** and ***Sayed (1987).*** Therefore the urine is critical sample in the detection of sulfur content in the body even in small amount rather than serum which may reveal negative analytical findings in such cases.

Estimation of sulfur content in renal, hepatic and pulmonary samples collected from buffaloes revealed a highly significant increase in the studied areas, without any significant variations in between, as compared with those of control area (Table, 8 and Fig., 19). This is indicated that sulfur is distributed and stored in these organs. The highest values are observed in both renal and hepatic tissues. This support our finding that urine is the main route of sulfur excretion and the highest liver sulfur content may be due to presence of many (-SH) containing enzymes in the hepatocytes, while the higher sulfur values of lung may be attributed to inhalation of sulfur fumes emitted from brick kilns and/or super-phosphate factory in affected areas. These results are in concord with those obtained by ***Seddek et al (1991)*** and ***Sayed (2001)*** in goat who found that the highest sulfur content in the liver and kidney followed by lung and muscle of goat reared in the vicinity of super-phosphate factory. However, the analytical findings of ***Ibrahim (1983)*** revealed negative sulfur content in the muscle and parenchymatus organs of buffaloes exposed to industrial sulfur emissions from super-phosphate factory.

3. Cadmium:

Analytical findings of serum cadmium at various localities of the study have been showed in the Table (9) and Fig. (20). The significant levels of cadmium are recorded in *Mansheit Amin Esmaeil* followed by *Binūfar* in both cattle and buffaloes, in addition to buffaloes at *Kafr Al-Aes*, while other localities showed slight elevations but non-significantly different from their respective control values. It was observed that cadmium levels increased only in animals which located under the effect of super-phosphate factory. This indicated that the manufacture of phosphate fertilizer is the main contributor source of cadmium ***(NRC, 1980*** and ***Davis 1984).*** Besides, deposition of cadmium on forage from automobile emissions from the Cairo-Alexandria agriculture highway is also significant source causing intoxication of farm animals ***(Swarup et al., 1997; Farmer and Farmer, 2000*** and ***Casteel, 2001).***

The species of animal is important factor for bio-accumulation of metals such as cadmium, where the data showed in Table (9) and Figs. (20 & 21) declared that the amount of serum and urinary cadmium were higher in buffaloes than cattle. This may be due to the buffaloes are heavy graze animals consuming more feed than cattle so it exposed to more cadmium via forages.

The blood cadmium levels obtained in the studied areas were in the range of those reported for cattle suffering from cadmium poisoning from automobile emission, phosphate fertilizer and/or mine drainage polluting plants and water as possible sources of cadmium at *Beni-Suif* province, Egypt (0.04 ± 0.01 to 1.01 ± 0.1 ppm) ***(Abdou et al. 2004)*** and were

significantly higher than those in the control animals. Also, these results are in agreement with the study of ***Sharkawy et al. (2002)*** that revealed elevation of blood cadmium concentrations compared with normal values (0.48 ± 0.05 Vs 0.23 ± 0.03 ppm) in male camel as a remarkable for environmental pollution. However, ***Penumarthy et al. (1980)*** and ***López Alonso et al., (2000)*** reported the blood cadmium levels near or below the detection limits of 0.005 ppm in over 50% of the animals analyzed for assessing the effects of metal pollutants in all species. Generally, according to the declaration of ***Puls (1994),*** the obtained blood cadmium values are considered to be toxic and indicating long term exposure to environmental cadmium pollution.

Estimation of urinary cadmium in cattle and buffaloes revealed the same tendency of their respective serum samples but recorded in higher concentrations as shown in Table (9) and Fig. (21), where the increased urinary cadmium reflects the recent exposure. Therefore, urinary cadmium measurement will provide a good index of excessive cadmium exposure ***(Klaassen, 2001)***.

The cadmium concentrations significantly increased in the examined buffaloes kidney (1.26 ± 0.13 and 1.03 ± 0.39 ppm), liver (0.51 ± 0.06 and 0.53 ± 0.12 ppm) and heart (0.03 ± 0.002 and 0.03 ± 0.003 ppm) tissues in both *Kom Hamadah* and *Kafr El-Zayiat* areas, respectively when compared with their respective control values. In addition, concerning to lung and muscle samples, their cadmium contents do not differ significantly in all areas, but somewhat higher in the studied areas than their respective controls. These results are consistent with those in livestock obtained by

Falandysz (1993), Huang and Liu (2001) and ***Liu (2003)*** who indicate the studied tissues are the critical organs for cadmium accumulation and in the order of the liver, kidney and muscle ***(Elinder, 1992).***

However, the cadmium levels in soft tissues that obtained in the studied areas in the present study are in order of magnitude lower than in the earlier studies on cattle reared on pasture receiving wastewaters (liver; 5.1 ppm and kidney; 10.3 ppm) ***(Sedki et al., 2003).*** Our results nearly similar to those of ***Abou-Arab (2001)*** who found that the cadmium concentrations for buffaloes has been proposed as 0.58 ppm for kidney, correspondingly 0.34 ppm for liver and 0.011 ppm for muscle samples collected from industrial areas (employing intense manufacturing processes such as cares, fertilizers, ceramics, refrigerators, incineration of refuse and ferrous scrap) in Egypt.

Our results showed that the cadmium concentrated primarily in the kidneys and liver tissues. This may be related to differences in the specific physiological functions of this heavy metal and depends on its relative abundance in the intracellular ligands that able to bind metal, such as sulfhydryl groups in the protein metallothionein ***(Kāji and Kojima, 1987*** and ***Husain et al., 1996)***, and because of the rate of cadmium elimination from these organs is relatively low ***(Han et al., 1994)***. These confirm, in the presence of such marked renal values, that urinary cadmium is a marker of total cadmium body burden, and thus, cumulative exposure to cadmium.

The most typical feature of chronic cadmium intoxication is kidney damage, which is generally assumed to occur at cadmium concentrations of

80 – 200 mg/kg wet wt. ***(Shore and Douben, 1994).*** It has been suggested that the lowest critical concentration in the liver is 20 mg/ kg wet wt. ***(Krajnc et al., 1983).*** The mean cadmium concentration in the kidneys and liver of studied animals were of superior magnitude below these critical concentrations.

As in many trace elements, the concentrations of metals in animals' tissues are largely dependent on the metal content of diet. So that in the present study the concentrations of cadmium in meat and tissues may be attributed to the contamination of water and feed stuff on which the animal were fed confirming the potential toxicity of the studied areas.

4. Calcium:

Tested animals either cattle or buffaloes showed significant lowered serum calcium contents in *Mansheit Amin Esmaeil* and *Shabour* localities when compared with their control values (Tables, 11 and Fig., 23). Similar results were obtained by ***Ibrahim (1983)*** who observed a significant decrease in calcium levels (ranged from 9.68 to 10.48 mg/dl) in buffaloes reared around super-phosphate factory. However, our obtained serum calcium levels in cattle and buffaloes were within the adequate limits (8.0 – 11.0 mg/dl) that recorded by ***Puls (1994).*** The degree of hypocalcaemia appeared to be related to the severity of the disease ***(Sayed, 1995).*** Therefore, the toxic signs of hypocalcaemia were not encountered in animals indicating that the decrease in blood calcium is not enough to be clinically manifested.

Jagadish et al. (1998) found that serum calcium level of cows in areas polluted by industrial effluents was significantly lower (6.20 ± 0.23

mg/dl). Also, industrial chronic sulfur and fluoride poisoning in donkeys and goats ***(Selim and Amany, 2000*** and ***Sayed, 2001)*** were associated with a significant decrease in serum calcium in both species. On the other hand, non-significant changes in serum calcium content in camels and goats around super-phosphate factory were recorded by ***Sayed (1987)*** and ***Seddek (1988).***

Regarding to the urinary excretion of calcium in cattle, it was significantly increased in all localities, while in buffaloes showed only a significant elevation at *Mansheit Amin Esmaeil* and *Shabour* localities when compared to their respective control animals (Table 11 and Figs. 23 & 24). Similar results were recorded by ***Ibrahim (1983)*** who attributing that to the fluoride-calcium interrelationship that makes it possible to trap calcium in urine even in the presence of severs hypocalcaemia.

A positive association had been seen between urinary excretion of fluoride and calcium (calciuria) suggests that increased exposure to fluoride may be associated with increased renal wasting of calcium. The calcium concentration in serum was negatively associated with urinary fluoride excretion.

Soft tissues calcium values do not differ significantly in the examined samples only showed slight decrease from control. In accordance ***Ibrahim (1983)*** concluded that the fluoride distribution in toxicity and consequently calcium are insignificant in soft tissues. In addition, ***Goodrich and Tillman (1966)*** found feeding sulfate ration elevates urinary calcium level that leads to lowered body calcium. Also, it reasonable to conclude that the

tissue calcium levels are not good guide to nutritional status of poisoned animals. However, the calcium concentrations in the kidneys and skeletal muscle at the studied areas are significantly lower than their respective control values.

Regarding to the hard tissues calcium level, there was no significant changes between the areas of the study and control ones, however their calcium values are higher. These data are in agreement with that of ***Miller et al. (1977)*** and ***Shearer et al. (1978)*** in cows suffering from osteoporosis due to environmental exposure to fluoride. In addition, ***Kawai et al. (1976)*** observed that the effects of oral administration of cadmium on bone and calcium metabolism caused decreased calcium content in bone and increased urinary excretion of cadmium. Consequently, it can be concluded that the disturbances in calcium metabolism and its tissues content is considered to be a multi-factorial and complex interrelationship between fluoride, sulfur and cadmium environmental exposure.

5. Inorganic phosphorous:

Estimation of serum inorganic phosphorous levels revealed a highly significant decrease while urinary inorganic phosphorous content exhibited significant excretion of higher amounts in the majority of studied localities especially in *Kom Hamadah* area that was highly affected by industrial pollution when compared with control area (Table, 14 & Figs., 27 & 28). These findings were in concord with the results of ***Ibrahim (1983), Madej et al. (1994), Metwalli et al. (1995)*** and ***Olkowski (1997)*** in ruminant suffer from fluorosis and/or sulfurosis, and in inconsistent with those obtained by ***Abd El-Aal (1981)*** and ***Sayed (2001)***. Moreover, the decrease

in serum phosphorous indicates that the disturbance not only confined to calcium, but also extends to include phosphorus attributing that to adverse effect of fluoride on the parathyroid gland which my disturbed calcium and phosphorus metabolism and subsequent changes in their serum and urinary levels as documented by ***Singh and Swarup (1994)***.

Furthermore, the *Kafr El-Zayiat* area had normal serum and urinary values for inorganic phosphorous concomitantly with the results of ***Kosla et al. (1993)*** who found that the concentration of cadmium had no effect on the serum levels of inorganic phosphorous of cows in an environment polluted with cadmium. Conversely, ***Ibrahim (1993)*** recorded a significant increase in serum inorganic phosphate levels in dogs treated with cadmium chloride.

The results revealed a non-significant decrease in inorganic phosphorous content either in the soft or hard tissues in the studied areas when compared with their respective controls. The same results observed by ***Ibrahim (1983)*** in buffaloes reared around super-phosphate factory. In accordance, ***Olkowski (1997)*** found that the retention of phosphorous was reduced by addition of sulfate to the diet of ruminants. All of these indicate the direct relationship between fluoride, sulfur and/or cadmium and elemental status of animal.

Generally, biochemical changes in calcium and phosphorous concentrations of serum and urine of affected animals especially at *Mansheit Amin Esmaeil* and *Shabour* localities (Tables, 11 & 14 and Figs., 23, 24, 27 & 28) were attributed mainly to the fluoride toxicosis, due to

metabolic conjunctions in-between which increase the urinary excretion of calcium and phosphorous as a compensatory mechanism decreasing their serum and tissues content and to contribute intoxication in the biological system, while recorded sulfur and cadmium concentrations had no effect on the calcium and phosphorous metabolism. Many investigations have documented this concept ***(Ibrahim, 1983; Kosla et al., 1993; Radostits et al., 1994; Singh and Swarup, 1994; Metwalli et al. 1995*** and ***Olkowski, 1997).***

6. Biochemical blood parameters:-

6.1. Liver function tests:

Analytical finding of plasma proteins in cattle and buffaloes as shown in Tables (17 & 18) revealed a significant decrease in plasma total proteins in *Kom Hamadah* localities, and serum albumin in all studied areas, whereas the serum globulin exhibited a significant increment only in buffaloes nearly at all areas when compared with control one.

Our results were concomitant with ***Kessabi et al. (1983), Araya et al. (1990)*** and ***Botha et al. (1993)*** who found that the total proteins and albumin were lowered and inversion in albumin/globulin ratio in fluorotic cattle and sheep, while ***Metwalli et al. (1995)*** not only observed a significant decrease in serum total protein and albumin, but also in serum globulin levels in cattle raised nearer to a heavy intensive area with brick kilns in Kafr Al-Sheikh province, Egypt.

Generally, the activities of liver enzymes are rarely affected and within normal limits of their control values however the AST and ALT activities

were increased at all areas but non-significantly changed, with exception ALT activity was increased significantly in cattle of *Mansheit Amin Esmaeil* that was the most affected locality with chronic industrial toxicosis. This increment in serum AST and ALT enzymes activities are within the normal physiological values of adult cattle recorded by ***Puls (1994)*** which are 31 – 70 IU/L for AST and 5 – 15 IU/L for ALT enzyme activities. This indicates a slight hepatic dysfunction confirming our suggestion that the fluoride is a non hepatotoxic element especially at low levels, while the disturbances in plasma proteins and activities of liver enzymes at *Mansheit Amin Esmaeil* may be attributed to the prolonged exposure of animals to fluoride, sulfur and/or cadmium, the joint interaction in-between or exposure of affected animals to higher levels of these toxicants as revealed earlier by ***Araya et al. (1990)***, ***Sel and Ergun (1992)***, ***Botha et al. (1993)***, ***Statov et al. (1994)***, ***Olkowski (1997)***, ***Jagadish et al. (1998)***, ***Bag et al. (1999)***, ***Liangx Feng (1999)*** and ***Kapoor et al. (2001).***

While the ALP enzyme activity in cattle and buffaloes was significantly increased in *Mansheit Amin Esmaeil, Shabour and Binūfar* localities when compared with their respective control. These results were in harmony with ***Sayed (2001)***. The increased level of ALP activity could be referred to the alterations induced by the effect of higher levels of fluoride, sulfur and/or cadmium in bones of intoxicated animals at the affected areas ***(Wang Jundong et al., 1992; Statov et al., 1994; Bag et al., 1999; Selim and Amany, 2000*** and ***Kapoor et al., 2001).***

6.2. Kidney function tests:

Regarding to the renal function tests, the levels of blood urea and serum creatinine in cattle and buffaloes were significantly increased especially in *Mansheit Amin Esmaeil* when compared with control values (Tables, 17 & 18). The marked elevation in serum urea and creatinine was observed in locality under the effect of super-phosphate factory and brick kilns, suggesting that alterations revised to renal toxic effect of both cadmium and /or fluoride. In agreement with these results, ***Metwalli et al. (1995)*** recorded an increased blood urea and creatinine concentrations in cows in the vicinity of brick kilns, attributing that to renal dysfunction associated with fluorosis.

Recently, ***Sayed (2001)*** found that the urea and creatinine values in goats' serum reared nearby the super-phosphate factory were more than normal limits, but higher than control values attributing that to the cellular degenerative changes in the parenchymatous organs caused by fluoride and cadmium toxicosis. The elevation of serum urea and creatinine content was also observed in ruminant and canines due to renal insufficiency induced by fluoride ***(Poey et al., 1976*** and ***Kessabi et al., 1983),*** sulfur ***(Olkowski, 1997)*** and cadmium exposure ***(Ibrahim, 1993*** and ***Haneef et al., 1998)***.

However, the other localities were within the limits of their control values. These results in concord with those of ***Jagadish et al., (1998)*** who observed that blood urea and creatinine were within normal values in cows exposed to excessive amount of fluoride.

In conclusions:-

1) It appears from the study the dangerous extension of the industrial pollution that arise from the super-phosphate factory, scattered brick kilns and automobile emissions as possible sources of fluoride, sulfur and/or cadmium that pollute the environment in *Kom Hamadah* and *Kafr El-Zayiat* areas.
2) The highest degree of the studied environmental pollutants recorded mainly in *Mansheit Amin Esmaeil*, followed by *Shabour* locality, whereas the lowest pollution occurred at *Kafr El-Naseria* locality.
3) The present study revealed intoxication of buffaloes and cattle mainly by fluoride with minor effect of sulfur and cadmium compounds emitted from heavy industrial activities. Therefore, it is important to prevent or even minimize such environmental pollution to protect animal health, and indirectly human being.
4) Our results suggested that the disease of affected animals might be complicated interactions between fluoride, sulfur and cadmium, primarily due to environmental pollution by industrial activities and their greatest degree of disruptions in elements homeostasis such as calcium, phosphorous, copper, molybdenum, zinc, iron and selenium.
5) It was declared that the amounts of serum and urinary fluoride and cadmium were higher in buffaloes than cattle, and vice versa in sulfur concentrations. Hence, the species of animal is important factor for bio-accumulation of different metals.
6) The River Nile water plays a significant role in the distribution of these toxic hazardous into the surrounding areas, together with feed stuff. Therefore, yearly water evaluations of all sources are a crucial monitor of animal health.
7) It must be compulsion the vinery companies and obligate them to accommodate their environmental situations, prohibiting the

permissions for building up any recent industrial activities in the concurrent time on the River Nile banks, and working to augment the hygienic disposal systems till coverage all public areas to prevent the direct disposal and throw out the industrial waste by-products into the River Nile water.

8) Urine analysis of fluoride, sulfur and cadmium toxicants is of great value in the assessment of poisoning even if in the early stages, compared with the troublesome of serum analytical measures, but cannot neglect its important role in toxicity diagnosis.

9) The veterinarians who worked at the studied areas must be awareness of the industrial toxicants affecting animal health and learn how to deal with them.

10) It should be advice the farmers and breeders to:-

   a. Use water and feed stuff free from these toxicants and away from the source of pollution.

   b. Add feed stuff supplements and feed additive with mineral mixtures especially those containing calcium and phosphorous to prevent or even minimize the occurrence of their animal intoxication.

   c. Rare the animal of short life span like chickens, sheep and goats, and finishing cattle in the affected areas.

11) Attendance of these toxicants in the parenchymatous organs and meat of studied animal was often a reliable indicator of environmental pollution and as an index for human intoxication even at undetectable concentrations if consumed for long period of time.

12) There is an urgent need to initiate extensive epidemiological and monitoring studies of people inhabiting the studied areas to assure health safety.

6. SUMMARY

For many years environmental pollution has been considered to be a hazard to health and the industrial pollution caused a serious and world widespread damage to the domestic animals. Grazing animals are often a reliable indicator of environmental pollution, either by feed, water or air exposure; they receive contaminants which are transferred through the food chain to human being.

The release of waste products from the super-phosphate fertilizer factory and brick kilns at *Kom Hamadah* (Behera province) and *Kafr El-Zayiat* (Gharbia province) may become toxic by continuous inhalation and/or ingestion of water and feed contaminated with toxic hazardous compounds. Therefore, the present study has been carried out to evaluate the contribution of fluoride, sulfur and cadmium compounds to the health condition of livestock and to reveal the extent of this pollution danger in the surrounding areas.

Our study involved 3 localities in *Kom-Hamadah* (Kafr Al-Aes, Mansheit Amin Esmaeil and Shabour) which are highly intensive areas with multiple brick kilns, and another 3 localities in *Kafr El-Zayiat* (Binûfar, Kafr El-Naseria and Kafr Hashad); containing super-phosphate plant in addition to other factories and several brick kilns, the areas represented different distances and directions from the sources of pollution.

During this investigation a random number of available cattle and buffaloes were used for clinical and laboratory assessment.

The macro-environmental sampling represented in surface water collected from the River Nile (Rosetta line) and feed stuff at the various localities in the order of 8 samples at least for each analysis for determination of their fluoride, sulfur and cadmium concentrations, whereas the micro-environmental samples (serum, urine, soft tissues and hard tissues) were collected from the cattle and/or buffaloes at the studied localities for determination of fluoride, sulfur, cadmium, calcium, inorganic phosphorous, serum proteins and hepatic and renal function tests for monitoring the health status of animals.

The study revealed the following results:

I. Water and feed stuff analysis:

The mean fluoride, sulfur and cadmium concentrations had been reached 5.8 ± 0.29, 8.9 ± 0.36 and 0.33 ± 0.11 ppm, respectively at the drainage system of the *Kafr El-Zayiat* super-phosphate factory.

There was a significant elevation of fluoride levels in water samples collected from the River Nile at *Mansheit Amin Esmaeil, Shabour* and *Binūfar*, and sulfur levels at the same localities as well as *Kafr El-Naseria* locality, while cadmium concentration was significantly elevated only at *Mansheit Amin Esmaeil and Binūfar* localities. The three elements were significantly increased within 0.5 km pre-factory (except cadmium) and extended to about 1.5 to 2 km post-factory with the direction of water.

The fluoride concentration was significantly increased in tibn and drees samples at *Mansheit Amin Esmaeil, Shabour, Binūfar* and *Kafr Al-Aes* localities, whereas sulfur concentrations were significantly elevated in tibn

samples collected from all localities except *Kafr Hashad* and in drees samples from *Mansheit Amin Esmaeil* and *Shabour* only. On the other hand, the significant concentrations of cadmium were noticed in samples collected from *Mansheit Amin Esmaeil* only.

II. Clinical examinations:

Cattle and buffaloes in the different localities around the super-phosphate factory and brick kilns were examined. Animals at *Kom Hamadah* were so severely affected more than those at *Kafr El-Zayiat* area, in a descending order of *Mansheit Amin Esmaeil, Shabour,* and *Kafr Al-Aes,* followed by *Binūfar and Kafr El-Naseria,* then *Kafr Hashad,* respectively.

The signs indicating fluoride intoxication in cattle and buffaloes were mainly confined to dental changes as severe brown to dark brown or even blackish discoloration, pitting and attrition of incisors and irregular enamel surface, with mottling of all permanent teeth, attrition and oblique eruption of most clinically affected incisive teeth. In addition, most of the affected buffaloes suffered from a pronounced lameness that was intermittent and recurrent annually in summer months and shifting from leg to leg. During movement, the animal was showing abducted elbow and arched back with pronounced bony extremities and weak musculature of lamed legs. On the contrary, there were no any signs of bony exostosis observed in affected animals

The specific clinical signs of sulfur and/or cadmium toxicosis as cough, respiratory distress or nervous symptoms had not been encountered in our cases. Furthermore, examination of affected animals revealed that they

suffered from cachexia, poor general health condition, emaciation, paleness of mucous membrane, rough dry coat losing its luster appearance and easily detached, and drop in milk yield with an incidence of reproductive failure as general toxic signs.

III. Analytical Findings of Biological Samples:

1. Fluoride:

Analytical findings of serum fluorine concentrations revealed a significant elevation in both cattle and buffaloes at *Mansheit Amin Esmaeil* locality and *Shabour* locality. Analysis of fluoride in the parenchymatous organs of buffaloes revealed a significant elevation only in renal tissue about 2- folds the control levels in *Kom Hamadah* and *Kafr El-Zayiat* areas. Unlike soft tissues, there is a significant increase in fluoride level in all samples of bony tissues ranged from 1300 to 5300 ppm. In addition, the maximum fluoride level is observed in cheek teeth and mandible followed by incisive teeth and maxilla, while the ribs contains the lowest values of fluoride.

2. Sulfur:

It is clear that cattle at all localities showed significant levels of serum and urinary sulfur except *Kafr Hashad* when compared with the control area, where the highest levels observed in the cattle and buffaloes at *Mansheit Amin Esmaeil* and *Shabour* localities. Estimation of sulfur content in renal, hepatic and pulmonary samples collected from buffaloes revealed a significant increase in the studied areas, without any significant variations in between, as compared with those of control area.

3. Cadmium:

The significant levels of cadmium in serum are recorded in *Mansheit Amin Esmaeil* followed by *Binūfar* in cattle and buffaloes, in addition to buffaloes at *Kafr Al-Aes*, while other localities showed slight elevations but non-significantly different from their respective control values. Estimation of urinary cadmium in cattle and buffaloes revealed the same tendency of their respective serum samples but recorded in higher concentrations. The cadmium concentrations increased significantly in the kidney, liver and heart tissues of buffaloes in *Kom Hamadah* and *Kafr El-Zayiat* areas, when compared with their respective controls.

4. Calcium:

Tested animals either cattle and buffaloes at *Mansheit Amin Esmaeil* and *Shabour* localities showed a significant decrease in their serum calcium content in comparison with control ones. In relation to urinary calcium content, it was significantly increased in all animals of tested villages except in buffaloes at *Kafr El-Zayiat* localities which showed non-significant elevation in relation to their respective control animals. Soft tissues calcium values did not differ significantly in the examined samples only documented slight decrease from control. Also, the hard tissues calcium level revealed non-significant changes between the areas of the study and control ones.

5. Inorganic Phosphorous:

Estimation of serum inorganic phosphorous levels revealed a significant depletion while urinary inorganic phosphorous content represented a significant excretion of higher amounts in the majority of

studied localities with highest concentrations in *Kom Hamadah* localities when compared with control animals. Our results revealed a non-significant decrease in inorganic phosphorous content either in the soft or hard tissues in the studied areas in relation to the control one.

6. Biochemical blood parameters:

6.1. Liver function tests:

There were a significant decrease in serum total proteins in *Kom Hamadah* locality, and serum albumin in all studied localities, whereas the serum globulin content recorded a significant increment only in buffaloes nearly at all areas when compared with control one.

Activities of liver transaminases (ALT and AST) are rarely affected and within normal physiological limits, where ALT activity was increased significantly in cattle of *Mansheit Amin Esmaeil* locality. The ALP enzyme activity in cattle and buffaloes was significantly increased especially in *Mansheit Amin Esmaeil, Shabour and Binûfar* localities when compared with their respective control.

6.2. Kidney function tests:

The levels of blood urea and serum creatinine in cattle and buffaloes were significantly increased in *Mansheit Amin Esmaeil* locality when compared with control values.

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الملخص العربي

(دِراسة تَأثيرٌ بَعْضُ المُلوِثاتِ الكِيميَائيةِ عَلى صِحْةِ الحَيْوَانِ)

أعتبرَ التلوث البيئي واحداً من أهم المخاطر التي تهدد الصحة العامة و تؤثر سلبياً علي الحياة الطبيعية، بالإضافة إلي أن التلوثِ الصناعي وحاجة الإنسان إلي الرفاهية، سبّبا ضرراً بيئياً خطيراً واسع الانتشار علي الحيوانات الأليفةِ، ومنها الأبقار والجاموس التي اعتبرت مؤشر موثوق به مِنْ مؤشرات التلوثِ البيئي، إمّا بالغذاء، أو بالتعرّض للهواء والماء؛ الذي ينتقل بدورة من خلال السلسلة الغذائية إلى الإنسان.

ومما لا شك فيه أن انبعاث المخلفات مِنْ مصنع السوبر فوسفات (الشركة الماليةِ) وأفران الطوب الأحمر بقُرى مدينتي كوم حماده (محافظة البحيرة) وكفر الزيات (محافظة الغربية) ووصول هذه الملوثات بشتي الطرق إلي الحياة البيلوجية يعد خطراً حقيقياً علي حياة الإنسان و الحيوان و النبات بها. لذا، فقد تم إجراء هذه الدراسة بهدف تقييم مدي تأثير مُركبات الفلورين والكبريتِ والكادميوم علي الصحة العامة للماشيةِ ولكشْف مدى خطر هذا التلوثِ علي المناطق المحيطةِ.

تضمّنتْ دراستُنا ٣ مناطق تابعة لمدينة كوم حماده وهي (شابور ، منشية أمين إسماعيل، كفر العيص)، وهي مناطقَ تحتوي علي أكثر مِنْ ٥٠ فرن من أفران الطوب الأحمر، و٣ مناطق أخرى تابعة لمدينة كفر الزيات و هي (كفر حشاد، كفر الناصرية، بنوفر)؛ لإحتواءها علي مصنع السوبر فوسفات، بالإضافة إلى مصانعَ أخرى وعِدّة أفران لإنتاج الطوب الأحمر، و قد كانتْ هذه المناطقَ علي مسافاتَ واتجاهات مختلفة مِنْ مصدر التلوث. واختيرت قرية الزعفراني التابعة لمدينة كوم حماده و التي تبعد حوالي ٢٠ كيلو متر عن مصدر التلوث والقرى الأخرى كمنطقة ضابطة لتقييم مدي التلوث بالقرى الأخرى.

ولإجراء هذا البحث يم إختيار عدداً عشوائياً مِنْ الأبقار المتوفرةِ والجاموس (تراوحت أعمارها من ٢-٥,٥ سنة) والتي ترعي بمناطق الدراسة، و تم فحصهم إكلينيكيا لتسجيل أعراض التسمم، وتقيمهم معملياً بشكل دقيق. أُخذتْ العينات البيئية كالماء السطحي مِنْ مجري نهر النيل (فرع رشيد) والغذاء الحيواني من مناطق الدراسة المُختلِفةِ بحد أدني (٨) عيناتِ على الأقل لكلّ تحليل لتحديد كميةِ الفلورين، والكادميوم، والكبريت. بالإيضافة إلي المصل الدموي، والبول، والأنسجة الداخلية الرخوة (الكبد، الكلي، الرئتين، القلب، العضلات)، والأنسجة العظمية كعظام الضلوع، الأسنان بأنواعها (القواطع، وضروس المقدمة و المؤخرة)، عظام الفكين السفلي و العلوي، و كلها جُمِعت مِنْ الأبقار و/أو الجاموس من المناطق المدروسةِ لتقييم المستوى الكمي لكل من الفلورين، الكبريت، الكادميوم، الكالسيوم، الفوسفور الغير عضوي، بالإضافة إلي تحديد وظيفةِ كل من الكبد والكلي و ذلك لمراقبة الحالة الصحيّةِ للحيوانات.

<u>ويمكن تلخيص نتائج ما اشتملت عليه هذه الدراسة في الآتي:-</u>

أولاً: النتائج التحليلية لمصدري الماء و الغذاء:

إن متوسطات الفلورين والكبريتَ والكادميوم قدْ سجلت القيم التالية(٥,٨ ± ٠,٢٩ و٨,٩ ± ٠,٣٦ و٠,٣٣ ± ٠,١١ جزء في المليون، علي التوالي) في شبكةِ صرفَ الشركة المالية الذي يصب مباشرةً في ماء نهر النيل والذي يُشكلُ المصدرَ الرئيسيَ لارتفاع هذه المواد الخطرة في ماء نهر النيل و المناطق المحيطة.

و لوحظ أن تلوث ماء نهر النيل بالفلورين قد ارْتفعَ بشكل ملحوظ، ولكنه قل بشكل تدريجي بالابتعاد عن المصنع حتى مسافة ٢ كم في اتجاه مجرى الماء بعد مصنع السوبر فوسفات ، و٠,٥ كم فقط ضدّ المجري المائي قبل المصنع. وكانت مستويات الكادميوم والكبريتَ تتمشي مع اتجاه الفلورين مع تدفق الماء بحدود ١,٥ كم بعد مصنع السوبر الفوسفات، و سُجّلَ الكبريتَ كميات معنوية ضدّ مجرى الماء.

أما تركيز الفلورين في الغذاءَ الحيواني، فقد ارتفع بشكل ملحوظ في العينات المنتقاة من التبن والدريس في منشية أمين إسماعيل (٥,٤ ± ٠,٥٢، ٤,٣ ± ٠,٦٤ جزء في المليون)، شابور (١,٧ ± ٠,٦١، ٣,٧ ± ١,٠٤ جزء في المليون)، بنوفر (١,٩ ± ٠,٠٨، ٢,٧ ± ٠,٢١ جزء في المليون)، كفر العيص (١,٨١ ± ٠,٣٥، ٢,٦ ± ٠,٧٥ جزء في المليون)، مقارنة بالمنطقةِ الضابطة (٠,٠٠٦ ± ٠,٠٠٠٣ و٠,١٨ ± ٠,٠٤ جزء في المليون)، على التوالي. النتائج التحليلية للكبريتِ في الغذاء الحيواني أظهرتْ ارتفاع هامً في عيناتِ التبن في كلّ المناطق باستثناء كفر حشاد. و كانت أعلى القيم المسجّلة في كل من التبن والدريس في منشية أمين إسماعيل (٧,١ ± ٠,٠٥، ٥,٤ ± ٠,٢٩ جزء في المليون) و شابور (٥,٨ ± ٠,٦٨، ٤,٣ ± ٠,١٦ جزء في المليون)، على التوالي.

من الناحية الأخرى، فإن الزيادة المعنوية للكادميوم في عيناتِ الغذاء سُجّلتْ في منشية أمين إسماعيل كالتالي (٠,٠٦٣ ± ٠,٠١ جزء في المليون) في التبن و (٠,١١ ± ٠,٠٢٤ جزء في المليون) في الدريس، ثم في قريةِ بنوفر، مُضَاهاةً بنفس العينات المنطقة الضابطة.

ثانياً: الفحصُ الإكلينيكي:-

تم فحص الماشية في مناطق البحث المختلفة حول مصنع السوبر فوسفات وأفران الطوب الأحمر فحصا سريرياً وسُجّلتْ العلاماتَ السامّة الإكلينيكية. ولوحظ أن الحيوانات في قري كوم حماده تأثرتْ أكثر وبشدّة مِنْ تلك الموجودة بنواحي كفر الزيات، وكان ترتيب القرى محل الدراسة تنازلياً حسب نسبة الإصابة كالتالي:- منشية أمين إسماعيل، شابور، كفر العيص، تليا بقرى بنوفر و كفر الناصرية، ثمّ كفر حشاد، على التوالي.

بالاستناد إلي التاريخ المرضي و الشكوى العامة مِنْ المربين في مناطق الدراسةِ من أن حيواناتهم تعاني من الضعفِ و العرج الموسمي المتكرّر الغير مستجيب للعلاج، مَع فقدان شَهية، ضعف ونحول عامّ في بَعْض الحيواناتِ خصوصاً تلك المرباة لفترةٍ طويلةٍ في المناطق المتأثرةِ. أظهر الفحص الإكلينيكي أن الأبقار تعاني من وجود ضعف البنية و الهزال، جفاف الجلد، خشونة الشعر، بهتان الأغشية المخاطية، تلون الأسنان بشكل رئيسي باللون البني الذي يميل إلي الإسوداد في بعض الحالات مع تآكلها، وتَحريض القواطع وسطح المينا الشاذ.

في الجاموس، أكثر الأعراض وضوحاً تلون الأسنان بمختلف درجات اللون البني الذي يميل إلي الأسود الداكن، وبتَبْقيع كُلّ الأسنان الدائمة خصوصاً الأضراس مقارناً بالقواطع. على أية حال، التآكل في سطح الأسنان كان حاداً، مع نمو الأسنان بشكل مائل خاصة. إضافة إلي ذلك، أن هذه الحيوانات كانت غير راغبَة في الوقوف، ولا تستطيع تحمل ثقل وزن الجسم، مع تقوس العمود الفقري ووضوح البروزات العظمية الدالة علي الضعف و الهزال، وأن أغلب الجاموس المتأثر عَانَ مِنْ عرج حاد يَنتقلُ من ساق إلى ساق ومتكرّر سنوياً خاصة في شهور الصيف، وهذه أكثر الأعراض المرضية ترجع أساساً إلي التسمم بالفلورين.

بينما الأعراض الإكلينيكية المعنية للتسمم بالكبريت أو الكادميوم كالسعال، الضيق التنفسي أو الأعراض العصبية فلم تلاحظ في الحيوانات التي تم فحصها. و عند فحص الحيواناتِ المتأثرةِ وجد أنهم يُعَانوا مِنْ حالة ضعف صحي عام، نحول، وانخفاض في عائد الحليب وحدوثِ العجز التناسلي كعلامات سامّة عامّة بالإضافة إلي الأعراض التي تمثلت في شكوى المربين. ولذلك، نستخلص بأنّ مرضَ الحيواناتِ المتأثرةِ قدْ يَكُونُ نتيجة العلاقة التداخلية بين الفلورين والكبريتِ والكادميوم، بسبب التلوثِ البيئي بالنشاطاتِ الصناعيةِ وتأثيرهم الأكبر في عرقلة توازن العناصر المعدنية داخل جسم الحيوان مثل الكالسيوم، الفوسفور، النحاس، الموليبدنوم، الزنك، و السيلينيوم.

ثالثاً: النتائج التحليلية للعينات البيولوجية:-

١- الفلورين:

أوضحت النتائج التحليلية لكمية الفلورين وجود ارتفاع معنوي في مصل كل من الأبقار والجاموس في منطقة منشية أمين إسماعيل (١,٢٧ ± ٠,٠٧ ، ١,٣٤ ± ٠,١٤ جزء في مليون، علي التوالي) وقرية شابور (٠,٩٩ ± ٠,١٢، ١,١٨ ± ٠,١٠ جزء في المليون، علي التوالي). من الناحية الأخرى، سجلَ الفلورين البوليَ أعلي تركيز له ١٥ ضعف ما كانت علية المجموعة الضابطة، بينما لم تتجاوز كمية الفلورين المصلي ٧ أضعاف القيمة الطبيعية.

تحاليل الفلورين في الأعضاء الرخوة المأخوذة من ذبائح الجاموس أوضحَت وجود ارتفاع معنوي في كلي الجاموس (٢ضعف مستوي المجموعة الضابطة) في كل مناطق الدراسة، بينما كان الفلورين في معدلاته الطبيعية في باقي الأنسجة.

على خلاف الأنسجةِ الرخوةِ، أظهرتْ النتائج بأنّ هناك زيادة معنوية في مستوى الفلورين في كُلّ عيناتِ الأنسجةِ العظميةِ يتراوح بين ١٣٠٠ إلي ٥٣٠٠ جزء في المليون، عمقارنة بالمجموعة الضابطة، بالإضافة، إلي أن أقصى مستوى للفلوريد لوحظ في عينات الأضراس(٥٣٠٠ ± ٦١٨,٣ جزء في المليون) والفكّ السفلي (٤٤٧٥ ± ٩٣١,١ جزء في المليون)، تليا بالقواطع (٣٨٠٠ ± ٥٤٤,٧ جزء في المليون) والفك الأعلى(٣٢٨٧ ± ٤٠٩ جزء في المليون)، بينما تَحتوي عينات الضلوعَ القِيَمَ الأدنى للفلورين وهو ٢٥٠٠ ± ٢٤٧ جزء في المليون.

٢-الكبريت:

أظهرت نتائج تحليل مصل وبول الأبقار وجود ارتفاع معنوي للكبريتِ في كل المناطق باستثناء كفر حشّاد مقارنة بالمنطقةِ الضابطة. ولُوحظ الحدَ الأعلى في الأبقار و الجاموس في منطقتي منشية أمين إسماعيل، و شابور. علي عكس الفلورين، لوحظ أنّ الأبقار كانتْ أكثر تأثُراً مِنْ الجاموس إضافة إلى احتواءها علي القِيَم الأعلى للكبريتَ، يعزي ذلك إلي ارتفاع نسبة التمثيل الغذائي والنشاط الأيضى في الأبقار عن الجاموس.

أوضح تقدير محتوى الكبريتِ في عيناتِ الأعضاء الداخلية والعضلات لذبائح الجاموسَ عن زيادة معنوية في أنسجة الكُليَ والكبدَ والرئتينَ، في قري كوم حماده وكفر الزيات، مع عدم وجود أيّ اختلافات معنوية بينهما، و كان أعلي تركيز للكبريت في أنسجة الكبد و الكلي.

٣-الكادميوم:

ابلغت أعلي مستويات الكادميوم في أمصال الأبقار و الجاموس بمنطقتي منشية أمين إسماعيل و بنوفر، بالإيضافة إلي جاموس كفر العيص، بينما القرى الأخرى كان بها ارتفاع طفيف غير معنوي عند المقارنة بقرية الزعفراني. وقد لوحظ بأنّ مستويات الكادميوم ازدادت في الحيواناتِ التي ترعي في الأماكن التي تقع تحت تأثير مصنع السوبر فوسفاتِ.

ارتفعت تركيزات الكادميوم بشكل ملحوظ في أنسجة الجاموس المُختَبَرةِ و كانت كالتالي: في الكُلي (١,٢٦ ± ٠,١٣ و١,٠٣ ± ٠,٣٩ جزء في المليون)، الكبد (٠,٥١ ± ٠,٠٦ و٠,٥٣ ± ٠,١٢ جزء في المليون) والقلب (٠,٠٣ ± ٠,٠٠٢ و٠,٠٣ ± ٠,٠٠٣ جزء في المليون) في كل من كوم حماده وكفر الزيات، على التوالي عندمّا ضُوهيتْ بالمجموعة الضابطة. كما أظهرتْ النّتائجُ بأن الكادميوم يتَركيز أولياً في الكُلي والكبد. بالإضافة، إلي أن محتويات الكادميوم بعيناتِ العضلاتَ والرئتين، لا تختلفُ بشكل ملحوظ في كلّ المناطق، لكن أعلى بكثير في المناطق المدروسةِ عن المنطقة الضابطة.

٤-الكالسيوم:

إنخفض مستوي الكالسيوم معنوياً في أمصال الأبقار أو الجاموس بقُرى منشية أمين إسماعيل وشابور مقارنة بمثليهم في القرية الضابطة. فيما يتعلق بمحتوى الكالسيوم البولي، فإنه ارتفع بشكل ملحوظ في كُلّ حيواناتِ القُرى المختبرة ماعدا في جاموس قري كفر الزيات، والتي أظهرتْ ارتفاع غيرَ معنوي بالنسبة لحيواناتِ المجموعة الضابطة.

أما قِيَم الكالسيوم في الأنسجةِ الرخوةِ فلا تَختلفُ بشكل ملحوظ في العيناتِ المَفْحُوصةِ، حيث أظهرَت انخفاض طفيفَ فقط منْ المجموعة الضابطة. أما بالنسبة لمستوى الكالسيوم في الأنسجةِ العظمية، لم تكنْ هناك أيّ تغييرات معنوية، لكن قِيَمَ الكالسيوم كانتِ أعلى بكثير بين مناطق الدراسة والضابطة.

٥-الفوسفور الغير عضوي:

انخفضت مستويات الفوسفور الغير عضوي المصلي، بينما شكّلَ المحتوى الفوسفور الغير عضوي البوليّ إخراج كمياتِ معنوية في أغلبيةِ المناطق المدروسة. وكانت التركيزات الأعلى في كوم حماده التي تأثرتَ إلى حدٍ كبير بالتلوثِ الصناعي بالمقارنة بقرية التحكيم البعيدة عن مصدر التلوث البيئي.

أظهرتْ النتائج انخفاض غير معنوي في محتوى الفوسفور الغير عضوي في الأنسجةِ الرخوةِ والصلبةِ المأخوذة من الذبائح في المناطق الملوّثةِ مِنْ تلك البعيدة عن مصدر التلوث. كُلّ هذا يَدُلُّ على العلاقةِ المباشرةِ بين الفلورين والكبريت والكادميوم ووضع كل من الكالسيوم والفوسفور الغير عضوي في الحيوان.

٦-نتائج وظائف الكبد و الكلي:

سُجلَ انخفاض هامّ في كل من البروتين الكلي المصليّ في قُرى كوم حماده، والزلال المصلي في كُلّ المناطق المختبرة، وكان أعلى انخفاض في قُري منشية أمين إسماعيل وشابور. على العكس من ذلك، كان تقدير محتوى الجلوبيولين المصلي ذو زيادة معنوية في كل المناطق المدروسة عند مضاهاتها بالمنطقة الضابطة.

إنّ نشاطات الإنزيمات الكبدية كانت ذات تأثير بسيط وضمن الحدود الطبيعيةِ مِنْ قِيَم المجموعة الضابطة وضمن المستوي القياسي الفسيولوجي للماشية البالغةِ. لكن معدل نشاط إنزيم الآلنين أمينوترانسفيريز في دم الأبقار كانت مرتفع معنوياً في منشية أمين إسماعيل. كما تبين أن نشاط الإنزيم الفوسفاتي القلوي ذو زيادة معنوية في قُري منشية أمين إسماعيل و شَابور و بنوفر مقارنة بالزعفراني. أظهرت نتائج الاختبارات الوظيفيةِ للكُلي بأنّ مستويات كل من البولينا والكرياتينين قد ارتفعا بشكل ملحوظ في قريةِ منشية أمين إسماعيل، مقارنة بالمنطقة الضابطة.

رابعاً: الخلاصة و توصيات الدراسة:-

يظهر من الرسالة الامتداد الخطر للتلوث الصناعي الصادر من مصنع كفر الزيات للسوبر فوسفات و أفران الطوب الأحمر بالإضافة إلي إنبعثات الطريق الزراعي، علي مدينتي كوم حماده و كفر الزيات، وكانت قرية منشية أمين إسماعيل من أكثر المناطق تضرراً، ثم قرية شابور.

أتضح من هذه الدراسة أن الأبقار والجاموس قد تأثرا تقريبا علي حد السواء ولكنْ الجاموس كان أكثر تضرراً من الأبقار بالمواد السمية المتطايرة مثل الفلورين والكبريت والكادميوم، وظهرت عليهما أعراض التسمم، الأمر الذي يؤكد التأثير الضار لمخلفات المصانع المنتشرة بطول نهر النيل علي صورة الحياة الحيوانية.

و قد اتضح أيضاً الدور الخطير الذي يلعبة نهر النيل في نقل السموم من مصدر انبعاثها ليصيب دائرة كبري تمثلت في الإصابات المذكورة في القرى محل البحث و النباتات الغذائية التي يتغذى عليها كل من الإنسان و الحيوان، و تلي ذلك الأهمية الكبرى للنباتات في نقلها للسموم حيث أنه من المستبعد إيجاد أو تجهيز غذاء خالي من هذه السميات. لذلك يجب إلزام الشركات المعنية بضرورة الالتزام بتوفيق أوضاعها البيئية، وعدم التصريح بإنشاء أي أنشطة صناعية أخرى بمدينتي كوم حماده وكفر الزيات في الوقت الحالي، والعمل علي زيادة تغطية خدمة الصرف الصحي للمناطق السكانية علي طول المصارف التي تصب علي فرع رشيد ووقف الصرف بكافة أنواعه علي نهر النيل.

مما لا شك فيه أن تحليل البول لهذه السموم البيئية يعد من أهم الطرق لتقييم التَسَمّم حتى إذا كان في المراحل المبكرةِ، إذا ما قُورنَ بإجراءاتِ المصل التحليليةِ، لكن لا نَستطيعُ إهْمال دورُ المصل الجليٌ في تشخيص التسمم. و وجود هذه السموم في المنتجات الحيوانية في مختلف صورها مؤشراً لحدوث التسمّم للعنصر البشري حتى عند المستويات الغير قابلة للكشفِ عنها إذا ما أستهلكتْ هذه المنتجات لفترة زمنيةٍ طويلة من العمر. مما يدعو إلي ضرورة اتخاذ إجراءات فورية و حاسمة لوضع حد لمثل هذا التلوث، ووقاية لصحة الحيوان، وحماية لصحة الإنسان الذي يعتمد علي منتجات هذه الحيوانات في طعامه.

كما ننصح السادة الأطباء البيطريين الذين يعملون في المناطق الصناعية أن يكونوا علي وعي كاف بتلك الملوثات الصناعية و مخاطرها. و ننصح المربين باستعمال الأغذية الحيوانية من مناطق بعيدة عن التلوث والاعتماد علي المياة الخالية من الملوثات الصرف الصناعي، واستعمال الأملاح المعدنية وإضافات الأعلاف التي تحتوي علي الكالسيوم و الفسفور في تغذية حيواناتهم للتقليل والحد من نسبة التسمم....كما نقترح أيضاً عليهم تربية الحيواناتِ ذات فترة الإنتاج القصيرةِ نسبياً في المناطق المتأثرةِ، ومثال على ذلك:- الدواجن، أو ماشيةِ التسمين والخرافِ.

قـرار لجنـة الحكم والمُناقشة

قـــررت لجـــنة الحكـــم والمنـــاقشـة و المجتمعـه بمقـر كليـة الطـب البيطـري – جامعـة الإسكندرية، منـــح الســـيد **ط.ب/ ياسـر سعيـد السيد سالـم** المـدرس المـساعد بقـسم الطـب الـشرعي و الـسموم و الإجـراءات البيطريـة درجـة دكتور الفلسفة فـــي العلـــوم الطبيـة البيطـرية تخصص **(الطب الشرعي والسموم والإجراءات البيطرية)**.

لجنة الحكم والمناقشة:-

١- أ.د / ثـابت عـبدالمنعـم إبراهيـم

أستـاذ و رئيـس مجلس قسم الطـب الشـرعي و السموم- كلية الطـب البيطري - جـامعة أسيـوط.

(عضـــواً)

٢- أ.د/ حـامـد عبـدالتـواب سمـاحـه

أستاذ صحـة الحيـوان والأمـراض المشتركـة وعميد كلية الطـب البيطري - جامعة الإسكندرية.

(عضـــواً)

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أستاذ أمراض الحيوان المعدية - كلية الطب البيطري - جامعة الإسكندريـة.

(مشـرفـا و عضـواً)

٤- د / خـالـد محمـد السيد عشـري

أستاذ مساعد الطب الشرعي والسموم - كلية الطـب البيطري- جامعة الإسكندرية.

(مشـرفـا و عضـواً)

تحريراً في: ٢٠٠٥/١٠/١ م

تحـــت إشـــراف

الأستـاذ الدكتـور
عبـدالكـريم عبـدالتـواب محمـود
أستـاذ الأمـراض المعـديـة
قسم طب الحيـوان
كليـــة الطـــب البيطـــري
جامعـة الإسكنـدريـة

الدكتـور
خالـد محمـد السيـد عشـرى
أستـــاذ الطب الشـرعي و السمـوم المسـاعـد
قسـم الطـب الشـرعي و السمـوم و الإجـراءات البيطـريـة
كليـــة الطـــب البيطـــري
جامعـة الإسكنـدريـة

دِراســـة تَأثيرُ بَعْضُ المـــلوِثات الكِيميَـــائيـــة عَلى صِحْـــة الحَيْـــوَان.

رسالة مقدمة من

ط.ب./ ياسر سعيد السيد سالم

بكالوريوس العلوم الطبية البيطرية- كلية الطب البيطري - جامعة الإسكندرية(١٩٩٨م)

ماجستير العلوم الطبية البيطرية- كلية الطب البيطري - جامعة الإسكندرية (٢٠٠٢م)

للحصول على

درجة دكتور الفلسفة في العلوم الطبية البيطرية

في الطب الشرعي و السموم

مقدمة إلى

قسم الطب الشرعي و السموم و الإجراءات البيطرية

كلية الطب البيطري

جامعة الإسكندرية

(٢٠٠٥)