ASSESSMENT OF CADMIUM TOXICITY IN CATTLE OF ELOOR INDUSTRIAL AREA

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THESIS

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KERALA, INDIA

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DECLARATION

I hereby declare that this thesis entitled **"ASSESSMENT OF CADMIUM TOXICITY IN CATTLE OF ELOOR INDUSTRIAL AREA"** is a bonafide record of research work done by me during the course of research and that this thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title of any other University or Society.

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CERTIFICATE

Certified that this thesis, entitled **"ASSESSMENT OF CADMIUM TOXICITY IN CATTLE OF ELOOR INDUSTRIAL AREA"** is a record of research work done independently by **Ms.A.R.Nisha** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to her.

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Dedicated to

the almighty

in gratitude

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Introduction

1. INTRODUCTION

Increased industrialisation and congestion of industrial complexes have accrued in high emission of pollutants to environment causing health hazards to human and animal population living around. In recent years increasing concentration of heavy metals especially cadmium in the environment have precipitated concern over toxicologic potential.

The element cadmium was discovered in 1817 by Strohmezer and Herman in Germany (Nriagu, 1980). It was seldom used for industrial purposes until recently. A higher resistance to corrosion and valuable electrochemical properties enhanced the use of cadmium in various industries.

The concentration of cadmium in earth's crust is less than any other heavy metal. The earth's crust averages only 0.15 ppb cadmium, marine water 0.15 ppb, fresh water less than 1 ppb and in air about 0.002-0.02 ppb (Fasset, 1975). Hence pollution with cadmium occurs mainly in industrial areas where it is handled. Atmospheric wash out, surface run off and acid rains resulting from increased acid sulfate and nitrate in the air lead to increased soil leaching and all this account for large amount of cadmium in sewage sludge from industries (Fasset, 1975). Contamination of environment with cadmium occurs in the vicinity of industries using zinc, because cadmium found as a constituent of zinc ores (Clark and Clark, 1981). Cadmium levels found to build up near fertilizer industries because phosphate fertilizer contain high cadmium (Mennear, 1979). It is widely used in electroplating, galvanisation, paint pigments and nickel cadmium batteries, access to these agents cause accidental cadmium poisoning (Hardman and Limbird, 1996).

The assimilation of cadmium from ground and the absorption of atmospheric cadmium by the aerial part of vegetables has been proved (Isane and Rincon, 1982). Studies showed that fodder irrigated with waste water had higher concentration of cadmium than those irrigated with tube well water (Krishnamurty, 1987). Continuous exposure of animals to industrial effluents containing cadmium level either through polluted atmosphere or through the food chain viz., vegetation, forage, water etc. increases body cadmium level, which is eventually excreted in milk, urine and bile (Sharma and Street, 1980).

Little is known about the status of cadmium in field and biological samples of cattle reared in industrial localities of Kerala. Eloor is one of the most industralized areas of Kerala. The major industrial units like Fertilizers and Chemicals Travancore (FACT), Travancore Cochin Chemicals (TCC), Hindustan Insecticide Limited (HIL), Indian Aluminium Company (INDAL), Indian Rare Earth (IRE), Travancore Chemical and Manufacturing Company (TCM) and Binani Zinc are situated here. These industries liberaly contribute to the pollution of that area.

The Fertilizers and Chemicals, Travancore is manufacturing nitrogenous as well as phosphatic chemicals. High level of cadmium in rock phosphate fertilizers may leads to liberation of cadmium to air and effluents (Vyas, 1996). In Binani Zinc electrolytic zinc is being manufactured in the smelter and effluents of this factory are likely to carry cadmium and zinc, since zinc is found always in association with cadmium. The other industrial units are not likely to cause any pollution with cadmium. Hence Eloor industrial area was selected for impact assessment studies of effluents on cattle health. The aim of the study was to assess the extent of industrial pollution with cadmium, to find out impact of pollution on cattle health and to suggest remedial measures.

Review of Literature

2. REVIEW OF LITERATURE

The element cadmium was discovered in 1817. Since then many studies have been carried out on its various toxicological aspects in different species of animals. The earlier works on cadmium is explained under four headings for convenience.

2.1. Occurrence and sources of contamination

2.2. Mechanisms of toxicity

2.3. Tissue localization and effects produced

2.4. The remedial and preventive measures to cadmium toxicity

2.1 OCCURRENCE AND SOURCES OF CONTAMINATION

Cadmium is a relatively rare and toxic element. It is present in traces in sea water, broad species of plants and animals (Vallee and Ulmer, 1972).

On an average the earth's crust contain 0.15 ppb and in marine water 0.15 ppb of cadmium. In fresh water cadmium content is less than 1 ppb and in air only 0.002-0.02 ppm (Fasset, 1975).

Industrial use of cadmium is mainly responsible for its pollution and subsequent toxicity. Fasset (1975) proved that atmospheric wash out, surface run off and acid rains resulting from increased acid sulfate and nitrate in the air lead to increased soil leaching and all this account for large amount of cadmium in sewage sludge.

Subacute cadmium intoxication was reported by Baker *et al.* (1979) in jewellery workers. They noticed symptoms like dyspnoea, chest pain, dysuria and dizziness in majority of workers.

Carruthers and Smith (1979) noticed cadmium toxicity with clinical and biochemical evidence in a population living near Zinc mining area. They observed that cadmium toxicity frequently occurs in combination with Zinc.

Mennear (1979) observed that the sintering process intended primarily to remove metal impurities from roasted Zinc ores, largely account for contamination of pastures with cadmium. He also noted a high level of cadmium in rock phosphate fertilizer which accumulate in crop lands and plants. The cadmium level in sewage sludge in non industrialized area and in phosphate fertilizer industrailized area were 8 ppm and 883 ppm respectively.

Occasional cases of poisoning have been recorded in man and animals following the use of cadmium plated vessels (Clark and Clark, 1981).

Albader *et al.* (1999) noticed sperm defect in cadmium toxicity due to heavy cigarette smoking.

Underwood and Suttle (1999) observed that cadmium uptake by the plants which grown on normal soil will be generally poor and they usually contain less than 1 ppm cadmium (on dry matter basis). They also noted that occasionally soils become enriched with cadmium from cadmium rich fertilizers or the disposal of wastes from the mining of metals such as Zinc and lead. According to them the cadmium content in the diet of ruminants should be in the range of 0.1-0.2 ppm(on dry matter basis).

2.2 MECHANISMS OF TOXICITY

The mechanisms of cadmium toxicity is explained depending on target sites of action like enzymes, glucose, mineral absorption, immune system and other actions.

2.2.1 Action on enzymes

Vallee and Ulmer (1972) reported that cadmium replace Zinc from enzyme like carboxypeptidase and alkaline phosphatase and impair the action of these enzymes.

Cousins *et al.* (1973) studied cadmium toxicity by adding 50, 150, 450, 1350 ppm cadmium in basal diet of 8 week old pigs. These animals showed reduced growth rate, haematocrit values, serum phosphorus and leucine amino peptidase activity.

Physiological response of calves to cadmium was studied by Lynch *et al.* (1976). They observed a reduced feed intake and body weights when calves were given cadmium for 6 week period. They also noticed an intermediate inhibition of δ amino levulinic acid dehydrase and reduced haemoglobin concentration.

An elevation of alanine amino transferase and glutaryl transpeptidase and a decrease in carbonic anhydrase level was noted by Faeder *et al.* (1977) when male wistar rats were given 0.25, 0.5, 0.75 mg cadmium per kg body weight subcutaneously for 8 weeks.

Clark and Clark (1981) observed that cadmium generally exert its toxic effect by inhibiting the sulphydril group of enzymes. It also inhibits the serum protein metabolism.

Cadmium chloride was injected subcutaneously into male and female rats of wistar strain at doses of 1.0, 1.5, 2.0 mg kg^{-1} body weight by Uehara *et al.* (1985). A decrease in the serum cholinesterase activity and an increase in glutamic pyruvic transaminase were observed.

Reddy and Bhagyalakshmi (1994) observed an increase in protease and alanine aminotransferase in hepatic and muscle tissues of crab in response to sublethal concentration (2.5 ppm) of cadmium chloride. They interpreted that these changes are suggestive of proteolysis and transamination of amino acids.

Novelli *et al.* (1998) utilized bioassay for the identification of toxic effects of cadmium intake. They demonstrated increased total urinary protein and increased kidney weight in rats exposed to cadmium chloride for 7 days in drinking water (100 mg/l). An increase in serum creatinine, total and direct bilirubin concentration and alanine amino transferase activity were also noticed indicating renal and hepatic toxicity.

2.2.2 Effects of glucose metabolism

Taghi and John (1973) noticed that administration of a single dose of cadmium acetate (2.6 mg/kg i/p) produced hyperglycemia and glucose intolerance in intact mice. Glucose intolerance was associated with a decreased pancreatic activity as evidenced by decreased insulinogenic indices in cadmium treated mice. A reduction in circulating serum insulin was also detected after subacute cadmium exposure.

Rats which are given cadmium chloride intraperitonialy at a dose rate of 1 mg/kg daily for 45 days showed a drop in hepatic glycogen and a rise in blood glucose and urea concentrations (Fasset, 1975).

Mennear (1979) studied the effect of cadmium on carbohydrate metabolism and inhibitory effect on pancreatic secretions. He observed that cadmium produced hyperglycemia and glycosuria in intoxicated rats.

Hyperglycemia with an increase in liver, kidney and ovary cholesterol concentration during an acute cadmium poisoning was observed by Gill and Pant (1983) in fishes. While hypoglycemia and diminished tissue cholesterol were manifested in chronically intoxicated fish.

Reddy and Bhagyalakshmi (1994) observed changes in oxidative metabolism in tissues of crab in response to sublethal concentration (2.5 ppm) of cadmium chloride. A reduction in glycogen, total carbohydrate and pyruvate and an increase in lactate levels in hepatic and muscle tissue were observed.

2.2.3 Action on mineral absorption

Vallee and Ulmer (1972) reported that cadmium compete with Zinc for 3-sulphydril groups of metallothionein which was identified in kidney of horse, rabbit and rat and liver of rabbits.

Fasset (1975) reported that when rats were given cadmium chloride intraperitonially at the dose rate of 1 mg/kg daily for 45 days showed hypochromic anaemia. He stated that anemia showed by rats may be related to effect of cadmium in iron metabolism.

Mennear (1979) found that cadmium antagonizes the essential minerals like zinc, selenium, iron and calcium. It also binds with RNA thereby interfering with process of transcription and translation.

The antagonism between selenium and cadmium in fresh water mollusc was reported by Paymbroeck *et al.* (1982). They observed that in the presence of sublethal amounts of selenium, sensitivity to cadmium was nearly halved.

Nath *et al.* (1984) reported that cadmium interferes with the utilization of essential metals like calcium, zinc, selenium, chromium and iron. Deficiencies of these essential metals, proteins and vitamins exaggerate cadmium toxicity due to increased absorption and greater retention. It also inhibit protein synthesis carbohydrate metabolism and drug metabolizing enzymes in liver of animals.

Tandon *et al.* (1994) showed that cadmium toxicity is reflected by induction of hepatic, renal and intestinal metallothionein in rats. Cadmium deposition in liver was enhanced by iron deficiency in the diet because iron deficiency enhanced hepatic zinc in response to cadmium.

2.2.4 Effect on immune body reaction

Immunosuppressant action of cadmium was reported by Koller (1973). Cadmium chloride was given to rabbits (300 ppm in drinking water) for 70 days and then inoculated with pseudorabies virus as antigen. In this situation, cadmium lowered the neutralizing antibody titre than did controls.

Lynch *et al.* (1976) reported a reduced haemoglobin concentration in cadmium ingested calves. They also noted an increase in lymphocytes and a decrease in neutrophils and monocytes.

Blakley (1984) reported that humoral immune response against sheep RBC was suppressed by cadmium in a dose dependent fashion. The maximum suppression of 28.2 per cent was observed in the highest exposure group.

2.2.5 Other effects

Nakada *et al.* (1989) reported a case of Itai-Itai disease in human with impaired renal concentrating ability, reduced urinary kallikrein and slightly enhanced renin-angiotensin-aldosterone system.

Tolerance to cadmium toxicity is often due to induction of metallothionein which sequesters cadmium and lowers its concentration at intracellular sites (Cherian *et al.*, 1994). Metallothionein plays an important role in carcinogenic process and induction of metallothionein is an important mechanism of resistance of tumour cells to chemotherapeutic agents.

Skoezynska and Smolik (1994) carried out experiments in male rats given intragastrially 20 mg cadmium per kilogram body weight and 70 mg lead/kg (as lead acetate) body weight. They noticed a lowered total, free and HDL cholesterol concentrations in serum. Rats poisoned with cadmium displayed a decrease of serum lipid peroxides level and increased blood dismutase activity.

Misra *et al.* (1996) reported that nitric oxide enhanced the cadmium toxicity by displacing the metal from metallothionein. Nitric oxide mediates cadmium release from metallothionein *in vivo*. They opined that intracellular generation of the cadmium induces DNA damage and force cells into a period of growth arrest.

Chisolam and Handorf (1996) explained that cadmium metallothionein mobilized from the liver might be the toxic serum factor associated with pre-eclampsia. They also observed that metallothionein bound cadmium may be mobilized from the liver into the serum during pregnancy as it follows the mobilization of metallothionein bound zinc.

Lyons-Alcantara *et al.* (1996) studied the differential cytotoxic effects of cadmium on fish and mammalian epithelial cells. The experiments using immortalized cell line showed that mammalian cells were more sensitive than fish cells to cadmium.

Effect of *in vivo* administration of 0.5, 1 or 2 mg cadmium/kg body weight on peripheral blood polymorphonuclear leucocyte and splenic lymphocyte functional state and viability was studied in rats by Kataramovski *et al.* (1999). Increase in some aspects of granulocyte function including spontaneous adhesion and activation was seen and was accompanied by increase in granulocyte survival.

2.3 TISSUE LOCALIZATION AND EFFECTS

Gunn *et al.* (1963) reported the selective injuries of testicular and epididymal blood vessels to cadmium. They gave a single subcutaneous injection of 0.03 mM per kg of cadmium chloride to study the specific effects of cadmium on testes. They found that testis and proximal end of the caput epididymis were specifically damaged by cadmium by virtue of injury to their vascular supply, internal spermatic artery and pampiniform plexus complex.

Cadmium metabolism was investigated by Miller *et al.* (1970) following a single oral dose of Cd^{109} in six months old male goats. Highest concentrations were found in the kidney followed by liver, duodenum and abomasum. Total body retention was 0.3 to 0.4 per cent of the dose with one half of this in the liver one fourth in the kidneys and a large part of remainder in the gastrointestinal tissue and contents. Bone, muscle and keratinized tissues contained only small amounts.

Miller *et al.* (1971) evaluated cadmium metabolism in 2.5 month old goats following single oral or intravenous tracer dose of cadmium. More than 90 per cent of the oral dose was excreted in the faeces within five days after dosing. Only 5.6 per cent of the intravenous dose was excreted via. faeces during the time. After intravenous dosing highest concentration were observed in the liver, kidney and spleen in descending order in contrast highest concentration were found in the wall of small intestine, kidney and liver after oral administration.

Fasset (1975) described the renal effects of cadmium from two sources. Low molecular weight type protein appeared in the urine after long exposure to excessive concentrations of cadmium by inhalation. It was also noted that cadmium is stored in the kidney in the form of cadmium binding proteins known as metallothionein. The proteinuria is considered to be first sign of tubular dysfunction and said to occur when renal cortical levels of cadmium reach above 200 ppm.

Biochemical and ultra structural changes in liver of cadmium treated rats was described by Faeder *et al.* (1977). Liver tissues of cadmium treated rats indicated dilatation of rough endoplasmic reticulam and proliferation of prominent connective tissue fibre bundles.

Mennear (1979) observed that cadmium entering into the body accumulated and cleared rapidly from blood. The cadmium content in blood is partitioned between fluid and erythrocyte parts. When erythrocyte level declines fluid level increases finally reaching a steady level when all binding sites get saturated.

Dalgarno (1980) studied the effect of low level exposure to cadmium on copper and zinc contents of affected tissue in pregnant ewes. Increased dietry cadmium was found to decrease the liver zinc and copper content at slaughter.

Grote and Speck (1980) carried out experiments to find accumulation of cadmium in broiler chicken. The broiler chicken were given 10 mg cadmium per day in drinking water for 15 days, concentration of cadmium was highest in kidney (18 ppm) and liver (11 ppm) whereas concentration in muscle and bones were about 10 times less than that of kidney levels .

Johnson *et al.* (1981) evaluated heavy metal retention in tissues of cattle fed high cadmium sewage sludge. Retention of cadmium in animal from sludge ingestion averaged 0.09 per cent and this retention increased the liver and kidney cadmium concentration by 5.20 fold.

Oral administration of 100 and 200 ppm of cadmium in drinking water induced diffuse membraneous glomerulo-nephritis in rats after 30 weeks of exposure (Joshi *et al.*, 1981). They also described that it was the immune complex formed that induced glomerular nephropathy.

Peterson and Vemmer (1981) described influence of increased cadmium doses in fattening pigs. It was found that above 3.8 mg/kg cadmium chloride in ration increased the volume of kidney and heart and above 123 mg/kg increased volume of liver.

In human Lauwerys *et al.* (1982) found that kidney is the critical organ first affected by cadmium. In kidney, it interferes not only with the tubular reabsorption process for low molecular weight proteins but also with the glomerular or tubular mechanism determining the excretion of high molecular weight proteins.

Induction of hepatic metallothionein in the rabbit foetus following maternal cadmium exposure was studied by Michael *et al.* (1982) after single subcutaneous injection of cadmium chloride to pregnant rabbits. A dose level of 0.5 mg/kg produced a reduction in foetal body weight, liver weight and kidney weight while having no effect on placental weight and an increase in foetal hepatic metallothionein concentration.

The effects of acute and chronic cadmium poisoning on blood and tissue metabolite levels of fish was described by Gill and Pant (1983). An hyperglycemia with increased tissue cholesterol content occurred in acute cadmium poisoning, conversely hypoglycemia and reduced tissue cholesterol were manifested in chronically intoxicated fish. In both cases, hypocholesterolemia glycogenolysis and a rise in myocardium glycogen concentration were noticed.

Liver, kidney, muscle and hair samples from new born and dead calves from 3 areas of Czechoslovakia were analysed for cadmium by Cibulka *et al.* (1989). The cadmium content was 0.007-0.03 ppm in muscle, 0.008-0.075 in liver, 0.007-0.062 in kidney and 0.009-0.074 ppm in hair.

Dwivedi *et al.* (1997) analysed milk samples from cows and buffaloes reared around the localities of textile, metallurgical and petrochemical industries. The mean cadmium concentration were 0.608, 0.012 and 0.008 μg/ml respectively in these areas, which were significantly higher than industry free rural localities which was noted as 0.004 μg/ml.

Underwood and Suttle (1999) reported that major excretory pathways of body cadmium was renal and faecal routes. The other alternate excretory pathways were milk, bile and saliva. They also noted that normal blood concentration of cadmium in ruminants was 0.01 ppm.

2.4 REMEDIAL MEASURES

Pond *et al.* (1973) observed that cadmium induced anemia in pigs was protected either by the daily administration of iron with feed or by a single intramuscular injection of 500-1000 mg iron as iron dextran.

Petering (1978) reported the preventive role of zinc, copper and iron in reducing, eliminating or combating the toxic effects of wide spread exposure to lead and cadmium in humans.

Effects of chelating agents on oral uptake and renal deposition and excretion of cadmium was reported by Engstrom (1984). He could reduce the acute toxicity by enhancing the elimination of cadmium after combining with chelating agents. These chelating had no effect on the metabolism of cadmium after long term low dose oral exposure.

Blalock and Hill (1988) noted an increased toxicity with cadmium in iron deficient birds than in iron supplemented ones.

Hardman and Limbird (1996) recommended chelature therapy with calcium disodium EDTA for cadmium toxicity. The dosage of chelating agent used was 75 mg/kg per day in 3 to 6 divided doses for 5 days.

Brzoska and Moniuszko (1998) reported that cadmium interfere with biological functions of Ca^{2+} ions. Large intake of calcium protect against absorption, cumulation and toxicity of cadmium. They also noted that interaction between calcium and cadmium take place at different stages of their metabolism.

A Japanese drug containing glycine has been reported to protect against chronic cadmium toxicity (Shaikh and Tang, 1999). The mechanism of protection against cadmium toxicity by glycine was by reducing oxidative stress.

Protection against chronic cadmium toxicity by caloric restriction was reported by Shaikh *et al.* (1999). They showed that caloric restriction in rats prevent cadmium induced nephrotoxicity and also appear to control the osteotoxicity of cadmium. Protection against chronic cadmium toxicity by caloric restriction was reported by Shaikh *et al.* (1999). They prevent cadmium induced nephrotoxicity and the osteotoxicity by caloric restriction in rats.

A study to investigate the role of zinc therapy in male cigarette smokers with infertility was done by Albader *et al.* (1999). They reported that a zinc deficient diet led to high cadmium accumulation in testis comparable with those supplemented with zinc.

Materials and Methods

3. MATERIALS AND METHODS

The study was carried out in different steps as follows:

3.1. Selection of representative areas in Eloor industrial belt for the study

3.2. Visits to veterinary hospitals in and around the chosen industrial area and survey of incidence of diseases by retrospective analysis of case sheets

3.3. Collection of field and biological samples

3.4. Analysis of samples

3.5. Analysis of blood and serum

3.6. Evaluation of data

3.1 Selection of representative areas in Eloor industrial belt

The area selected for impact assessment studies of effluents of cadmium on cattle include industrial units like Fertilizers and Chemicals Travancore (FACT), Travancore Cochin Chemicals (TCC), Hindustan Insecticides Limited (HIL), Indian Aluminium Company (INDAL), Indian Rare Earth (IRE) and Binani Zinc. Eloor industrial belt (Fig. 1 & 2) is classified into 4 areas for convenience of study.

(a) Eloor South (around Fertilizers and Chemicals, Travancore)

(b) Eloor East (around aluminium processing industry)

(c) Eloor North (around insecticide industries)

(d) Binanipuram (around zinc processing industries)

The samples were collected from areas around the industries.

3.2 Survey of incidence of diseases

The case sheets and case register in the veterinary hospitals of Eloor industrial area (i.e., Eloor and Muppathadam veterinary hospitals) were screened to find out common diseases affecting cattle in that area.

3.3 Collection of field and biological samples

3.3.1 Field samples

(a) Water samples were collected observing sampling rules (Lorgue *et al.*, 1987) from wells, paddy fields and marshy area of Eloor industrial area and water bodies to which effluents are released.

(b) Forage samples which include fodder, tree leaves and grasses were also collected.

Samples were collected from areas around University Livestock Farm, Mannuthy as controls.

3.3.2 Biological samples

Biological samples like urine, faeces, milk and blood (heparinised and whole blood) were collected from minimum of 10 cattle maintained in that area at least for preceeding three years. Samples were also collected from 10 animals above 3 years of age from ULF, Mannuthy as controls.

3.4 Analysis of samples

The representative samples were sun dried, pulverised and weighed (except blood ,serum ,urine, milk and water). The samples were digested(except water and serum) using special digestion apparatus (AOAC, 1973) (Fig. 3).

PROCEDURE

One gram / one millilitre was placed in a digestion flask with glass beads. Concentrated nitric acid (20 ml) was added to the sample, swirl and then connected the flask to the assembly. The sample was allowed to stand for 30 minutes for predigestion. A rapid stream of water is passed through the condenser and the stopcock is adjusted to reflux position. Ten ml of water is added through dropping funnel and heated cautiously until initial vigorous reaction was over. After completing the vigorous reaction heated gently just to reflux the digest. The refluxing was continued for about 30 minutes and then cooled. Five ml of cold nitric acid, sulphuric acid mixture (1:1) was slowly added through and heated the digest to dark brown colour. Trapped water is poured back by opening the stopcock. Continued heating until the digest become transparent.. Added 5 ml of 40percent (w/v) urea to cooled digest and refluxed for 15 minutes. The condenser is washed for 3-5 times with little water. The reaction flask is removed and cooled under tap water. The insoluble matter was filtered through a small pledget of glass wool. The filtrate is transferred to 100 ml volumetric flask and made upto 100 ml with distilled water.

The digested samples were analysed for cadmium by an Atomic absorption spectrophotometer (Perkin-Elmer) at 228.8 nm. The cadmium content in the water and serum was estimated directly (after appropriate dilution) .

3.5 Analysis of blood and serum

3.5.1 Haematology

3.5.1.1 Total erythrocyte count (TEC)

Total erythrocyte count was determined using Hayem's fluid as per method described by Schalm (1975).

3.5.1.2 Total leucocyte count (TLC)

TLC was determined using Thomas fluid as mentioned by Schalm (1975).

3.5.1.3 Differential leucocyte count (DC)

Differential count was done using Wright's stain as per method described by Schalm (1975).

3.5.1.4 Haemoglobin (Hb)

Haemoglobin was estimated by acid haematin method using Sahl's haemoglobinometer as described by Benjamin (1985).

3.5.1.5 Packed cell volume (PCV)

PCV was determined using Wintrobe haematocrit tube as mentioned by Wintrobe (1981).

3.5.1.6 Mean corpuscular volume (MCV), Mean corpuscular haemoglobin concentration (MCHC) and Mean corpuscular haemoglobin (MCH)

MCV, MCHC and MCH were determined as mentioned by Benjamin (1985).

3.5.2 Serum Biochemistry

3.5.2.1 Total protein and albumin

Total protein and albumin in the serum was estimated using total protein and albumin kit[1] in the MERCK Microlab 200 blood analyser.

3.5.2.2 Serum urea and creatinine

Serum urea and creatinine was determined using urea and creatinine kit[2] in the MERCK Microlab 200 blood analyser.

3.5.2.3 Serum enzymes

Serum alkaline phosphatase, serum alanine amino transferase, serum aspartate amino transferase were estimated using the enzyme kits[3] in the MERCK microlab 200 blood analyzer.

3.6 Evaluation of data

Results were statistically analysed using (CRD) complete randomized design (Snedecor and Cochran, 1967). The mean were compared using least significant difference test.

1, 2 and 3 - Kits - E-Merck (India) Ltd., M.I.D.C., Taloja-410 208

Fig. 1. MAP OF KERALA SHOWING STUDY AREA

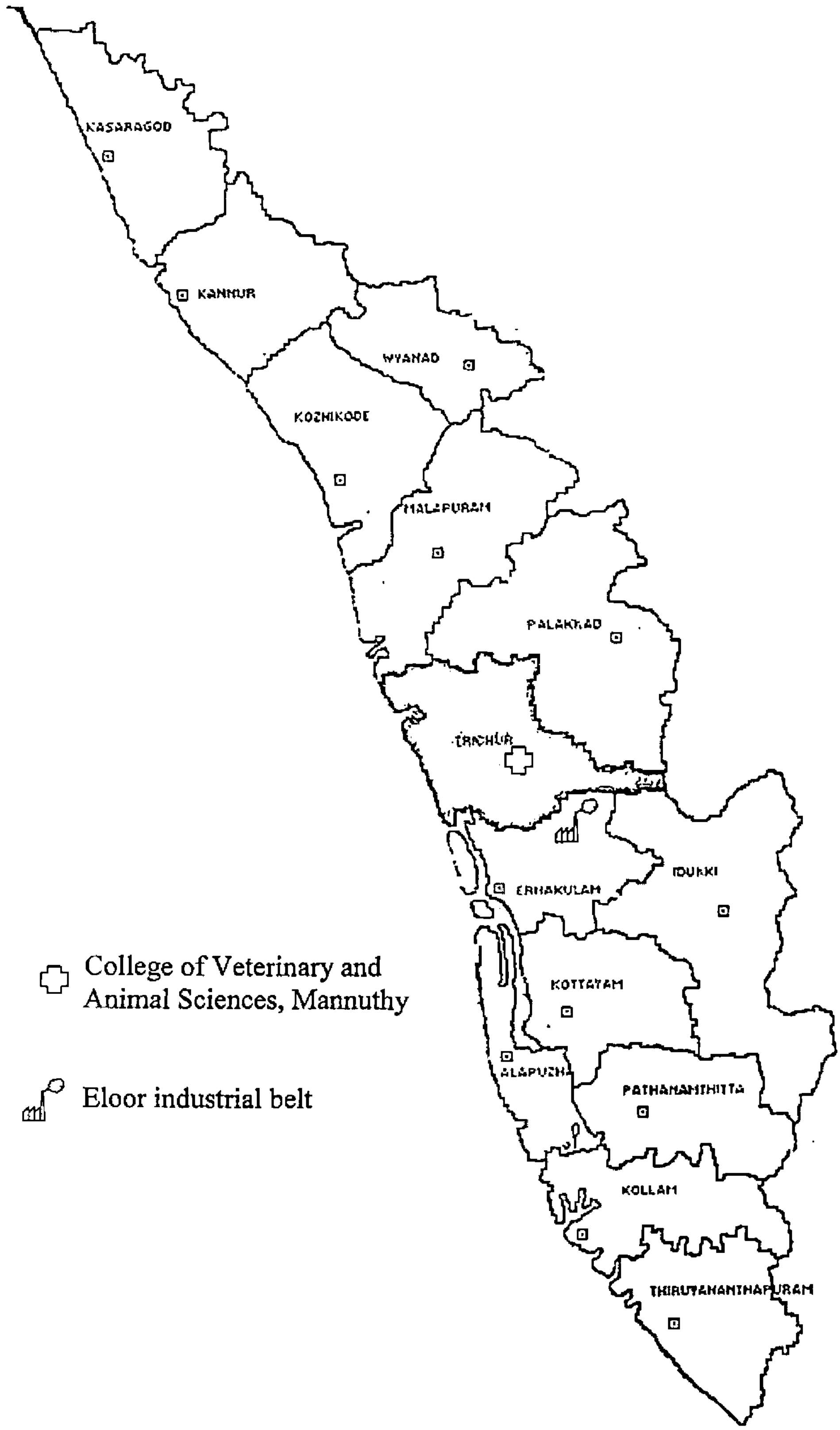
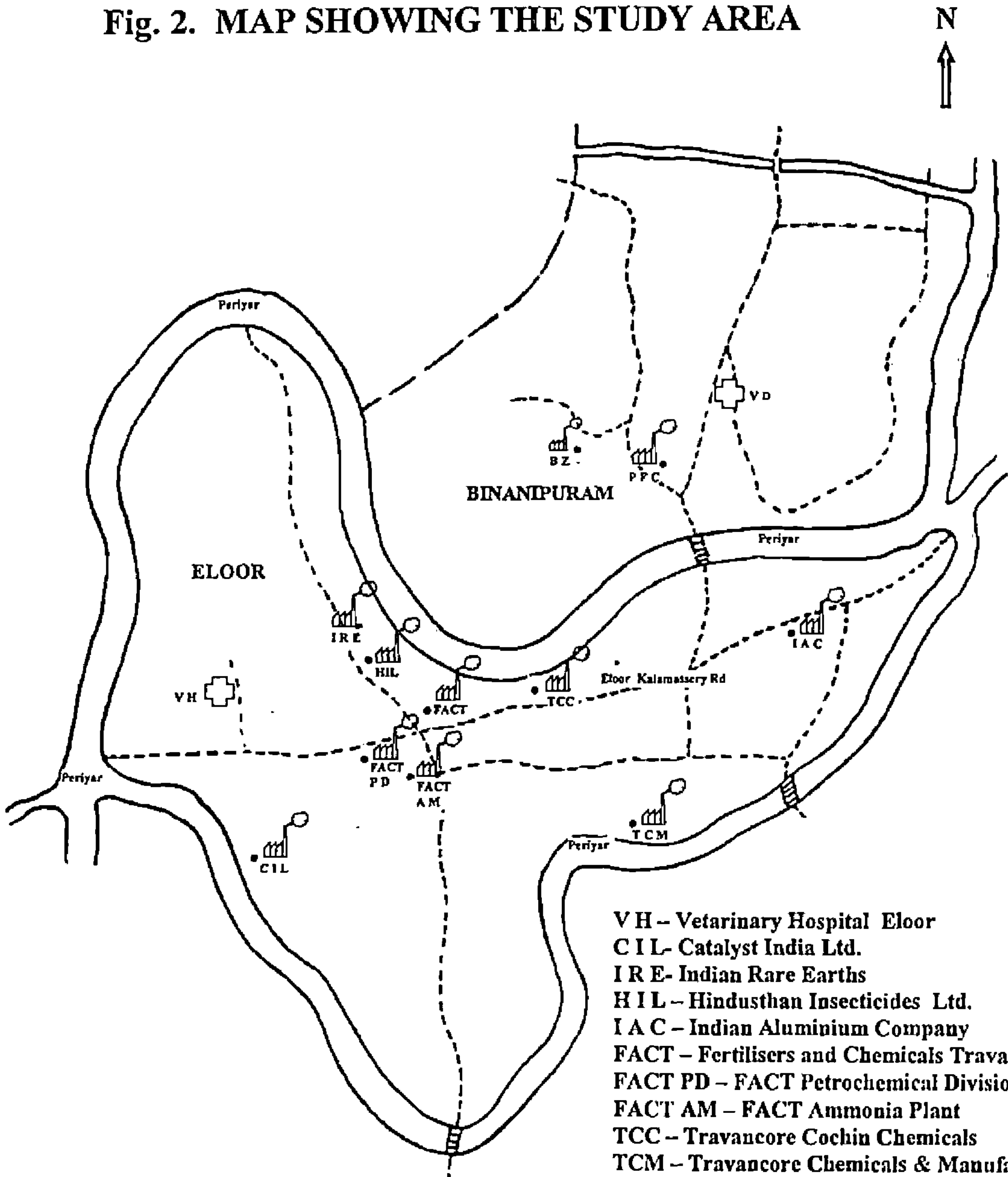
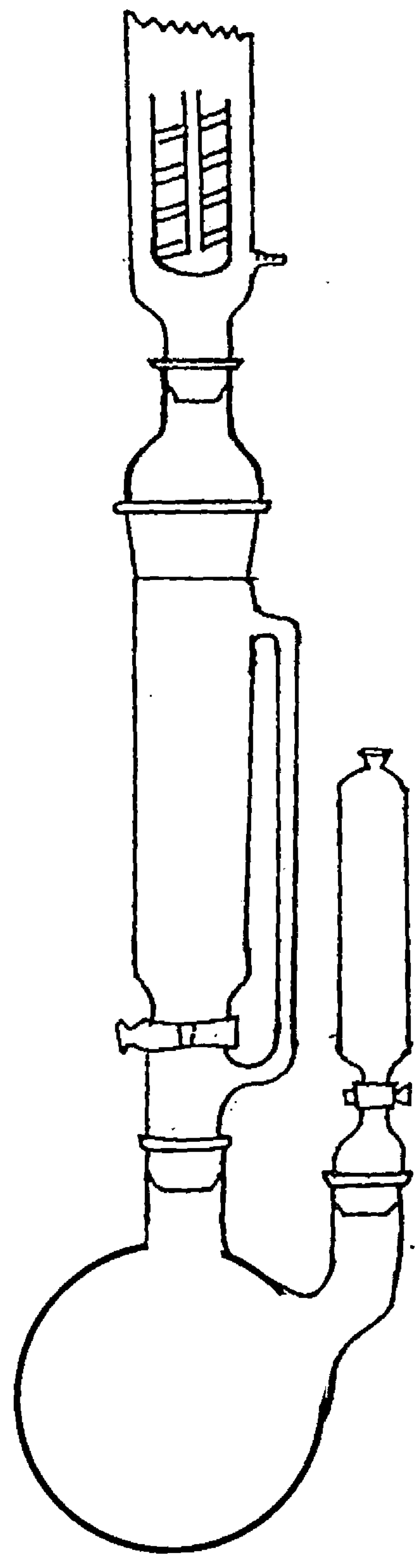


Fig. 2. MAP SHOWING THE STUDY AREA



V H – Vetarinary Hospital Eloor
C I L- Catalyst India Ltd.
I R E- Indian Rare Earths
H I L – Hindusthan Insecticides Ltd.
I A C – Indian Aluminium Company
FACT – Fertilisers and Chemicals Travancore
FACT PD – FACT Petrochemical Division
FACT AM – FACT Ammonia Plant
TCC – Travancore Cochin Chemicals
TCM – Travancore Chemicals & Manufacturing Co.
B Z – Binani Zinc Ltd.
P F C – Prima Feed Co.
V D – Veterinary Dispensary, Binanipuram

Fig. 3. SPECIAL DIGESTION APPARATUS



Results

4. RESULTS

4.1 Retrospective analysis of case sheets

Retrospective analysis of 7423 case sheets for the period from January 1994 to February 2000 was carried out in Eloor and Muppathadam Veterinary Hospitals. The results are presented in Table 1 and Fig. 4 and 5. In Eloor Veterinary hospital out of the 1503 case sheets examined 851 digestive disorders, 126 repeat breeders, 75 respiratory diseases and 41 deficiency diseases were observed. In Muppathadam Veterinary hospital out of the 5920 case sheets examined 3348 digestive disorders, 1284 repeat breeders, 398 respiratory diseases and 433 deficiency diseases were observed.

Table 1. Type of cases recorded in Veterinary hospitals

Disorders	Eloor	Muppathadam
Digestive problems	851 (56%)	3348 (56.57%)
Repeat breeders	126 (8.54%)	1284 (21.7%)
Respiratory disorders	75 (5.08%)	398 (6.73%)
Deficiency diseases	41 (2.77%)	433 (7.33%)
Miscellaneous	410 (27.61%)	457 (7.71%)

Fig. 4. Types of disorders treated in the Eloor Veterinary Hospital

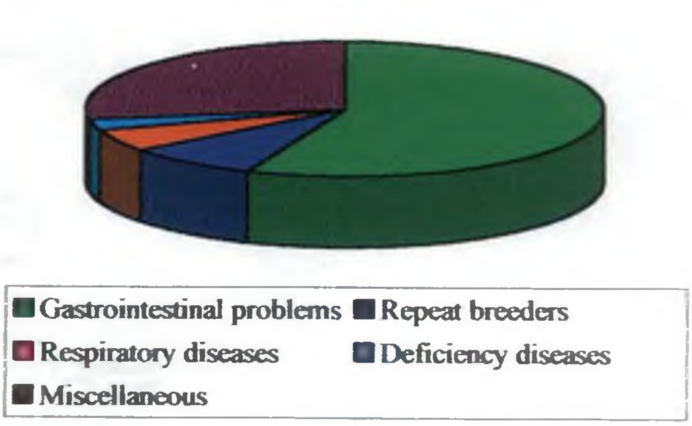
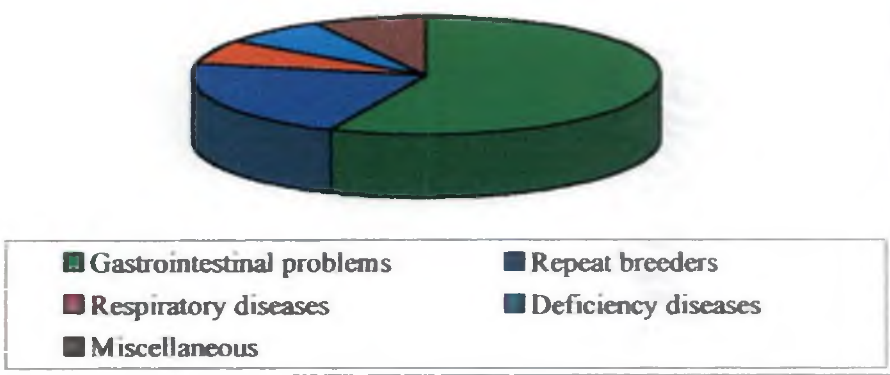


Fig. 5. Types of disorders treated in the Muppathadam Veterinary Hospital



4.2 Cadmium content in field samples

4.2.1 Water

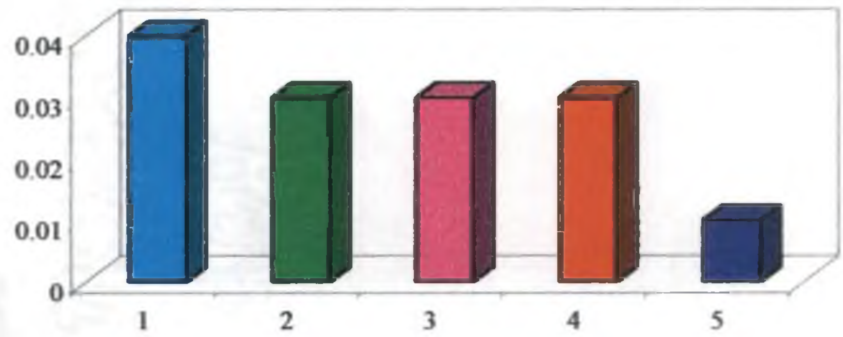
The mean cadmium (ppm) level in areas of Binanipuram, Alupuram, Eloor South, Eloor North were 0.04±0.01, 0.03±0.01, 0.03±0.01 and 0.03±0.01 respectively as compared to 0.01±0.01 in the area of control study. The results are presented in Table 2 and Fig. 6.

Table 2. Cadmium content in water (ppm) from study areas

Sl. No.	Binanipuram	Alupuram	Eloor south	Eloor north	Control
1	0.04	0.04	0.04	0.02	0.01
2	0.03	0.03	0.03	0.02	0.02
3	0.03	0.02	0.04	0.01	0.01
4	0.06	0.01	0.05	0.02	0.02
5	0.01	0.03	0.04	0.04	0.01
6	0.05	0.04	0.02	0.03	0.01
7	0.04	0.02	0.02	0.02	0.02
8	0.01	0.03	0.04	0.05	0.10
9	0.04	0.01	0.03	0.02	0.02
10	0.04	0.03	0.02	0.02	0.01
11	0.04	0.04	0.04	0.03	-
12	0.05	0.04	0.04	0.02	-
13	0.04	0.04	0.04	0.03	-
14	0.05	0.02	0.03	0.03	-
15	0.04	0.03	0.03	0.03	-
Mean ± SE	0.04±^{a} 0.01	0.03±^{b} 0.01	0.03±^{ab} 0.01	0.03±^{b} 0.01	0.01±^{c} 0.01

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

Fig. 6. Cadmium content in water from study areas



1 - Binanipuram, 2 - Alupuram, 3 - Eloor South
4 - Eloor North, 5 - Control

4.2.2 Forage samples

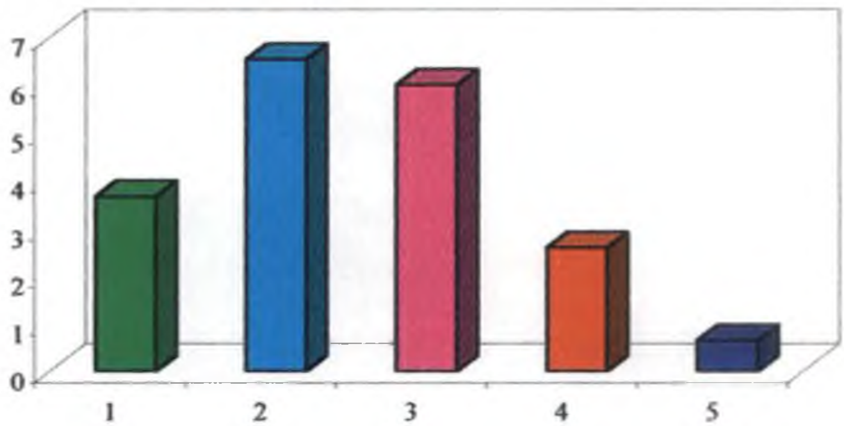
The mean cadmium content (ppm) of forage samples in the areas of Alupuram, Binanipuram, Eloor south, Eloor north and control areas were 3.67±0.64, 6.53±1.28, 6.00±4.85, 2.60±0.70 and 0.65±0.29 respectively. The values are presented in Table 3 and Fig. 7.

Table 3. Cadmium content of forage samples (ppm) on dry matter basis

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	3.00	10.0	3.0	2.0	1.0
2	3.50	8.0	3.0	3.0	1.0
3	4.50	8.0	3.0	3.0	0.5
4	5.50	7.0	3.0	3.0	0.5
5	4.00	7.0	5.0	3.0	0.0
6	4.50	6.0	6.0	4.0	0.0
7	3.00	4.0	10.0	3.0	0.0
8	2.50	4.0	7.0	3.0	1.0
9	4.50	5.0	4.0	1.0	1.0
10	4.50	5.0	9.0	4.0	1.0
11	3.50	4.0	10.0	3.0	-
12	2.50	8.0	6.0	1.0	-
13	3.50	8.0	7.0	2.0	-
14	2.50	7.0	8.0	1.0	-
15	3.50	7.0	6.0	3.0	-
Mean + SE	$3.67\pm^{b}$ 0.64	$6.53\pm^{ab}$ 1.28	$6.00\pm^{a}$ 4.85	$2.60\pm^{b}$ 0.70	$0.65\pm^{c}$ 0.29

a, b, c - Means with different superscripts within same row differs significantly (P<0.01)

Fig. 7. Cadmium content of plants in study areas



1 - Alupuram, 2 - Binanipuram, 3 - Eloor South, 4 - Eloor North,
5 - Control

4.3 Cadmium content in the biological samples

The cadmium content in blood, serum, dung, urine and milk were estimated and the results are presented in Table 4, 5, 6, 7 and 8 respectively.

4.3.1 Cadmium concentration in blood

Cadmium concentration in blood of cattle in Alupuram, Binanipuram, Eloor south, Eloor north and control were 1.23±0.35, 1.72±0.55, 3.33±0.84, 0.41±0.15 and 0.70±0.21 respectively. The highest value of blood cadmium was found in Eloor south followed by Binanipuram, Alupuram and Eloor north which were significantly higher ($P<0.01$) than controls.

Table 4. Cadmium content in blood of cattle from study areas

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	2.0	1.5	2.5	0.8	0.8
2	0.2	1.5	4.0	0.7	0.4
3	0.4	2.5	2.5	0.2	0.2
4	1.6	2.0	1.0	0.4	0.8
5	1.8	3.5	3.5	0.2	0.6
6	1.2	0.2	2.5	0.6	1.2
7	1.6	0.6	4.0	0.2	0.8
8	1.0	1.5	5.0	0.4	1.2
9	0.6	1.5	4.0	0.2	0.8
10	1.4	1.5	2.0	0.6	0.6
11	1.4	2.0	1.0	0.4	-
12	1.2	1.5	4.0	0.6	-
13	1.4	2.5	4.0	0.2	-
14	1.2	1.5	5.0	0.4	-
15	1.4	2.0	4.0	0.2	-
Mean ± SE	$1.23\pm^{b}$ 0.35	$1.72\pm^{b}$ 0.55	$3.33\pm^{a}$ 0.84	$0.41\pm^{c}$ 0.15	$0.70\pm^{c}$ 0.21

a, b, c - Means with different superscripts within same row differs significantly (P<0.01)

4.3.2 Cadmium concentration in serum

Cadmium concentration in serum of cattle from Alupuram, Binanipuram, Eloor south, Eloor north and control were 0.37±0.15, 0.23±0.07, 0.20±0.08, 0.29±0.06 and 0.11±0.06 respectively. The values are presented in Table 5.

The values were significantly higher (P<0.01) for all samples from test areas than controls which differ significantly from each other in areas of Alupuram, Binanipuram and Eloor north.

Table 5. Cadmium content in serum (ppm) of cattle from study areas

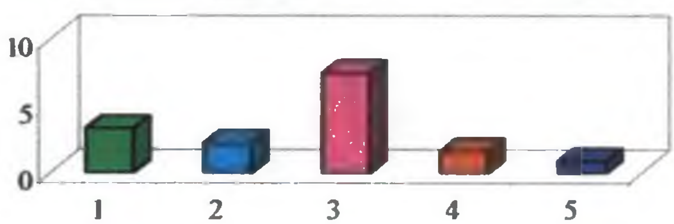
Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	0.5	0.4	0.2	0.3	0.1
2	0.4	0.1	0.3	0.2	0.2
3	0.5	0.3	0.1	0.2	0.2
4	1.0	0.2	0.1	0.1	0.2
5	0.5	0.3	0.1	0.3	0.0
6	0.4	0.2	0.3	0.3	0.0
7	0.1	0.3	0.1	0.3	0.1
8	0.2	0.4	0.45	0.2	0.2
9	0.4	0.3	0.4	0.4	0.1
10	0.3	0.2	0.4	0.2	0.0
11	0.3	0.2	0.1	0.4	-
12	0.3	0.1	0.15	0.4	-
13	0.3	0.13	0.1	0.4	-
14	0.2	0.2	0.1	0.3	-
15	0.4	0.3	0.2	0.4	-
Mean + SE	$0.37\pm^{a}$ 0.15	$0.23\pm^{bc}$ 0.07	$0.20\pm^{c}$ 0.08	$0.29\pm^{b}$ 0.06	$0.11\pm^{c}$ 0.06

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

4.3.3 Cadmium concentration in dung

Cadmium concentration in dung of cattle from Alupuram, Binanipuram, Eloor south, Eloor north and control were 3.33±0.90, 2.23±0.57, 7.40±1.58, 1.60±1.58 and 0.90±0.40 respectively. The values are presented in Table 6 and Fig. 8. All values from test areas were significantly higher than controls.

Fig. 8. Cadmium content of dung in study areas



1 - Alupuram, 2 - Binanipuram, 3 - Eloor South,
4 - Eloor North, 5 - Control

Table 6. Cadmium concentration in dung (ppm) from cattle of study areas

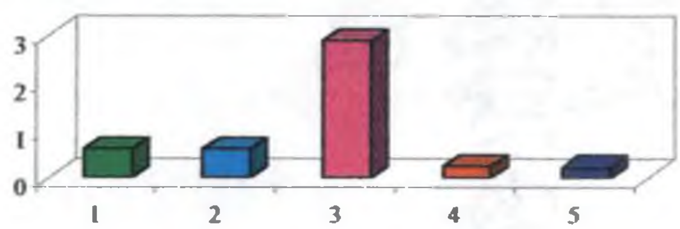
Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	3.0	3.0	9.0	2.0	0.5
2	3.0	0.5	10.0	3.0	2.0
3	7.5	2.5	8.5	4.0	1.0
4	3.5	2.0	2.0	4.0	0.5
5	3.5	3.0	8.0	1.5	1.5
6	3.5	3.0	3.5	1.5	0.5
7	2.5	1.0	7.5	1.0	0.5
8	2.5	2.0	7.5	1.0	0.5
9	3.2	3.0	6.0	1.0	0.5
10	3.1	2.5	10.0	1.0	1.5.
11	1.5	2.5	8.0	1.0	-
12	3.1	2.5	6.0	1.0	-
13	3.5	2.0	9.0	1.0	-
14	3.5	1.5	8.5	1.0	-
15	3.1	2.5	8.0	1.0	-
Mean + SE	$3.33\pm^{b}$ 0.90	$2.23\pm^{bc}$ 0.54	$7.40\pm^{a}$ 1.58	$1.60\pm^{bc}$ 1.58	$0.90\pm^{c}$ 0.40

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

4.3.4 Cadmium concentration in urine

Cadmium concentration in urine of cattle from Alupuram, Binanipuram, Eloor south, Eloor north and control were 0.63±0.15, 0.60±1.67, 2.90±1.12, 0.24±0.08 and 0.23±0.14 respectively (Table 7 and Fig. 9). The highest value was found in samples from Eloor south.

Fig. 9. Cadmium content in urine of cattle from study areas



1 - Alupuram, 2 - Binanipuram, 3 - Eloor South
4 - Eloor North, 5 - Control

Table 7. Cadmium concentration in urine (ppm) from cattle of study areas

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	0.3	0.8	5.00	0.2	0.1
2	0.3	0.4	1.00	0.3	0.1
3	0.6	0.8	2.50	0.2	0.0
4	0.9	0.6	2.00	0.3	0.2
5	0.7	0.4	2.00	0.4	0.1
6	0.9	0.6	5.02	0.4	0.1
7	0.6	0.4	2.50	0.2	0.6
8	0.7	0.6	1.00	0.1	0.4
9	0.7	0.4	5.00	0.2	0.5
10	0.6	0.1	3.00	0.1	0.2
Mean + SE	0.63± 0.15b	0.60± 1.67^{b}	2.90± 1.12^{a}	0.24± 0.08^{c}	0.23± 0.14^{c}

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

4.3.5 Cadmium concentration in milk

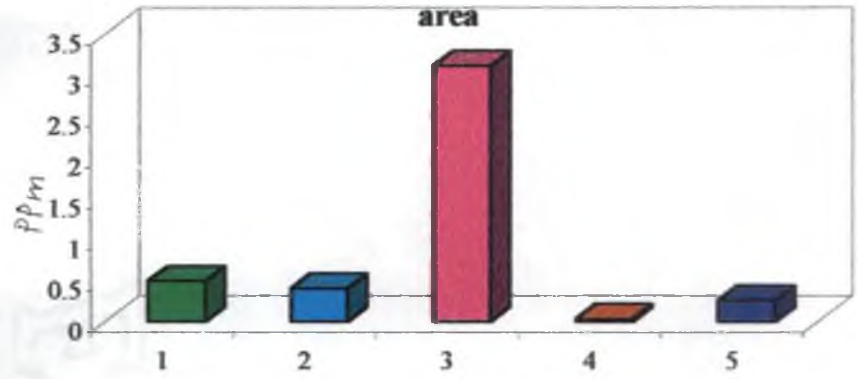
The cadmium level in milk from Alupuram, Binanipuram, Eloor south, Eloor north and control were 0.49±0.10, 0.40±0.11, 3.11±1.32, 0.03±0.02 and 0.26±0.13 respectively (Table 8 and Fig. 10). The levels were significantly higher than control ($P<0.01$) in Alupuram, Binanipuram and Eloor south. The highest value of cadmium was found in samples from Eloor south.

Table 8. Cadmium concentration in milk (ppm) from cattle of study areas

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	0.4	0.2	0.12	0.1	0.1
2	0.4	0.6	2.4	0.01	0.1
3	0.4	0.6	5.6	0.03	0.1
4	0.5	0.2	4.0	0.02	0.6
5	0.6	0.4	6.5	0.02	0.6
6	0.5	0.6	2.5	0.03	0.2
7	0.6	0.2	2.0	0.04	0.2
8	0.3	0.4	3.5	0.05	0.3
9	0.8	0.4	2.5	0.02	0.2
10	0.4	0.4	2.0	0.1	0.2
Mean + SE	0.49± 0.10^{b}	0.40± 0.11^{b}	3.11± 1.32^{a}	0.03± 0.02^{c}	0.26± 0.13^{c}

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

Fig. 10. Cadmium content in milk of cattle from study area



1 - Alupuram, 2 - Binanipuram, 3 - Eloor South
4 - Eloor North, 5 – Control

4.4 Haemogram

Total leucocyte count (Table 9). Total erythrocyte count (Table 10), Haemoglobin (Table 11), Packed cell volume (Table 12), mean corpuscular volume, mean corpuscular haemoglobin, mean corpuscular haemoglobin concentration (Table 13), differential count (Table 14) were estimated and the observations are presented in Tables 9 to 14 respectively.

4.4.1 Total leucocyte count

The mean total leucocyte count ($x10^3/nm^3$) in cattle of Alupuram, Binanipuram, Eloor South, Eloor North and control were 10.69±0.48, 10.57±0.16, 10.20±0.40, 10.05±0.47 and 10.04±0.06 respectively. There was no significant difference observed between the samples.

Table 9. Total leucocyte count of blood samples (thousand/mm^3)

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	10.5	10.65	9.20	10.30	10.35
2	9.20	10.20	9.60	10.20	9.30
3	11.45	9.20	9.20	9.35	9.40
4	9.80	11.60	9.45	9.20	10.15
5	11.20	9.60	10.20	9.40	9.60
6	10.65	10.65	10.45	10.15	9.80
7	10.20	10.20	10.60	9.10	10.15
8	10.85	9.25	10.55	9.60	10.20
9	9.60	10.20	9.85	8.65	9.80
10	10.25	10.80	10.60	9.65	9.60
11	10.60	11.65	10.45	9.25	-
12	9.25	9.20	10.20	9.40	-
13	10.20	10.60	9.80	10.65	-
14	10.85	11.60	10.30	10.25	-
15	11.60	9.45	10.10	9.80	-
Mean + SE	10.69±0.48	10.57±0.16	10.20±0.40	10.05±0.47	10.04±0.06

4.4.2 Total erythrocyte count (TEC)

The total erythrocyte count (x10^6/mm^3) from Eloor south (6.40±0.81), Alupuram (5.74±0.16), Binanipuram (6.40±1.39) and Eloor north (5.59±0.17) were significantly lower than control values (7.74±0.31).

Table 10. Total erythrocyte count of blood samples (million/mm^3)

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	6.02	6.02	6.21	5.30	7.20
2	5.92	5.91	6.30	5.81	6.81
3	7.32	5.22	7.28	6.20	7.90
4	5.34	5.43	5.82	6.84	8.22
5	6.81	6.82	6.40	5.20	6.60
6	5.92	5.31	6.21	5.83	6.26
7	6.39	5.24	7.84	5.20	8.29
8	6.02	7.22	8.20	6.82	7.81
9	6.30	7.81	5.23	6.20	6.72
10	7.28	5.21	6.42	5.20	7.72
11	5.86	5.62	6.20	6.26	-
12	7.34	5.33	7.21	6.88	-
13	6.22	5.21	5.20	6.45	-
14	6.01	5.02	6.26	6.26	-
15	5.21	5.71	6.80	5.20	-
Mean +SE	6.40±0.81^b	5.74±0.16^c	6.40±1.39^c	5.59±0.17^b	7.74±0.31^a

a, b, c - Means with different superscripts within same row differs significantly (P<0.01)

4.4.3 Packed cell volume

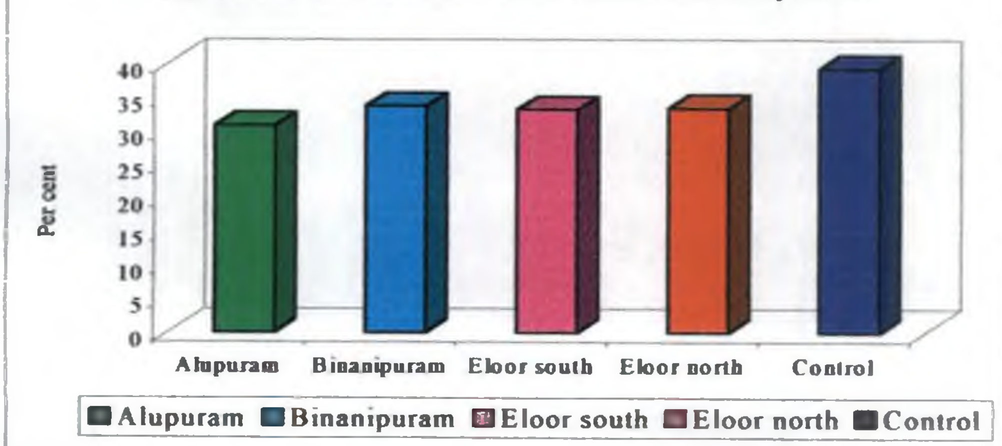
The packed cell volume per cent in cattle blood samples from Alupuram (30.80±1.51), Binanipuram (33.73±5.62), Eloor south (33.15±2.20), Eloor north (33.40±1.76) were significantly lower than control (39.20±1.19) (Table 11, Fig. 11).

Table 11. Packed cell volume of cattle from test areas (Per cent)

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	25.0	34.0	28.0	36.0	46.0
2	32.0	32.0	36.0	32.0	42.0
3	30.0	28.0	32.0	34.0	38.0
4	32.0	26.0	34.0	26.0	36.0
5	34.0	34.0	30.0	28.0	32.0
6	28.0	32.0	28.0	27.0	33.0
7	26.0	30.0	26.0	34.0	40.0
8	30.0	32.0	34.0	32.0	42.0
9	32.0	33.0	32.0	28.0	38.0
10	28.0	38.0	34.0	34.0	40.0
11	32.0	28.0	34.0	32.0	-
12	30.0	26.0	32.0	28.0	-
13	30.0	30.0	29.0	26.0	-
14	28.0	36.0	33.0	32.0	-
15	30.0	32.0	34.0	38.0	-
Mean + SE	30.80 ± 1.51^{a}	33.73 ± 5.62^{b}	33.15 ± 2.20^{b}	33.40 ± 1.76^{b}	39.20 ± 1.19^{c}

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

Fig. 11. Packed cell volume of cattle from study areas



4.4.4 Haemoglobin (Hb) concentration

The mean haemoglobin (g/dl) concentration in cattle of Alupuram, Binanipuram, Eloor south, Eloor north and control areas were 7.91±0.29, 7.40±0.42, 8.40±0.17, 8.42±0.41 and 11.26±0.71 respectively. The values were significantly lower ($P<0.05$) in all test areas from control (Table 12).

Table 12. Haemoglobin concentration in blood samples (g/dl) of cattle from study areas

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	8.2	7.5	8.2	8.4	10.8
2	8.1	7.8	8.8	8.8	12.0
3	7.4	8.2	7.8	8.5	11.6
4	7.8	7.2	8.2	7.8	12.0
5	7.9	7.0	8.5	8.9	10.2
6	8.2	7.6	8.4	8.2	13.2
7	7.6	7.8	7.8	8.4	11.2
8	7.8	7.2	8.9	8.2	10.2
9	7.9	7.3	8.2	7.2	11.8
10	8.3	7.4	8.4	8.2	11.1
11	7.2	7.8	8.5	8.4	-
12	7.8	7.2	8.4	8.5	-
13	8.4	7.3	8.2	8.2	-
14	8.2	7.4	8.4	8.7	-
15	7.8	7.5	8.5	8.5	-
Mean + SE	7.91 ± 0.29^{b}	7.40 ± 0.42^{b}	8.40 ± 0.17^{c}	8.42 ± 0.41^{c}	11.26 ± 7.08^{a}

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

4.4.5 Differential leucocyte count

The mean neutrophil content in blood samples from Alupuram, Binanipuram, Eloor south, Eloor north and control were 34.50±2.17, 32.60±1.30, 31.20±1.40, 31.80±1.78 and 27.70±1.39 per cent respectively. There was no significant difference among test areas.

The mean lymphocyte count in cattle blood samples from Alupuram, Binanipuram, Eloor south, Eloor north and control were 54.80±2.27, 57.40±1.38, 58.80±1.49, 57.50±1.61 and 62.10±1.47 per cent respectively. There was no significant difference among sample areas.

The mean eosinophil count were 7.70±0.82, 7.00±1.23, 7.00±0.60, 6.80±0.64 and 5.90±0.31 per cent in Alupuram, Binanipuram, Eloor south, Eloor north and control samples. There was no significant difference observed among sample areas.

The mean monocyte percentage in Alupuram, Binanipuram, Eloor south, Eloor north and control were 2.00±0.33, 2.10±0.84, 2.00±0.29, 2.20±0.29 and 3.00±0.35 per cent respectively. No significant difference existed among sample areas.

The mean basophil count were 1.00±0.71, 1.90±1.03, 2.00±0.31, 1.70±0.31, 2.00±0.73 per cent respectively. No significant difference existed among sample areas (Table 13).

Table 13. Differential leucocyte count in blood samples of Eloor industrial area and controls (per cent)

		Alupuram	Binanipuram	Eloor south	Eloor north	Control
Differential leucocyte count (per cent)	Neutrophil	34.50±2.17	32.60±1.30	31.20±1.40	31.80±1.78	27.70±1.39
	Lympocyte	54.80±2.27	57.40±1.38	58.80±1.49	57.50±1.61	62.00±1.47
	Eosinophil	7.70±0.82	7.00±1.23	7.00±0.56	6.80±0.64	5.90±0.31
	Monocyte	2.00±0.33	2.10±0.84	2.00±0.29	2.20±0.29	3.00±0.35
	Basophil	1.00±0.71	1.90±1.03	2.00±0.31	1.70±0.31	2.00±0.73

4.4.6 Mean corpuscular volume, mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration in cattle of test areas

The mean corpuscular volume (MCV) values of cattle from Alupuram, Binanipuram, Eloor south, Eloor north and control areas were 54.10±2.44, 58.76±2.90, 55.43±3.94, 51.05±3.70, 51.02±2.70 femtolitre (fl) respectively. There was no significant difference observed among sample areas.

The mean corpuscular haemoglobin concentration in cattle of Alupuram, Binanipuram, Eloor south, Eloor north and control areas were 13.88±0.41, 12.89±0.79, 14.11±0.64, 12.87±0.82 and 14.68±1.25 picogram(pg) respectively. There was no significant difference observed between the samples.

The mean corpuscular haemoglobin concentration values of Alupuram, Binanipuram, Eloor south, Eloor north and control were 25.69±0.69, 21.90±0.80, 26.41±1.50, 25.63±2.25 and 28.62±0.71 (g/dl). There was no significant difference observed between the samples (Table 14).

Table 14. Mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) in cattle of test areas

	Alupuram	Binanipuram	Eloor south	Eloor north	Control
MCV (fl)	54.10±2.44	58.76±2.96	55.43±3.94	51.05±3.70	51.02±2.70
MCH (pg)	13.88±0.41	12.89±0.79	14.11±0.64	12.87±0.82	14.68±1.25
MCHC (g/dl)	25.69±0.67	21.90±0.80	26.41±1.50	25.63±2.25	28.62±0.71

4.5 Serum biochemistry

Total protein, albumin, aspartate amino transferase, alanine aminotransferase and alkaline phosphatase were estimated.

4.5.1 Total serum protein

The mean total serum protein (gm/dl) in cattle of Binanipuram, Alupuram, Eloor south, Eloor north and control were 6.01±0.39, 6.86±0.83, 6.69±0.54, 6.82±0.68 and 8.14±0.40 respectively (Table 15, Fig. 12).

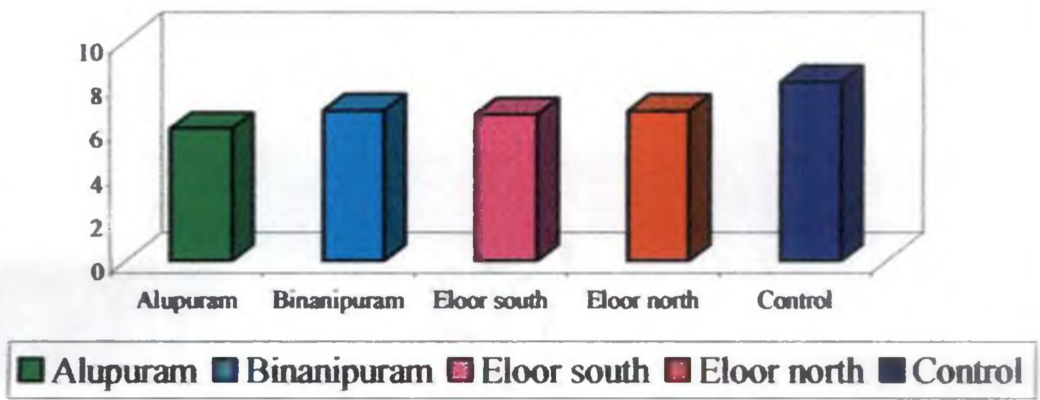
All the values of test areas were significantly lower than control.

Table 15. Total serum protein in cattle of Eloor industrial area and control (gm/dl)

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	6.36	6.21	6.83	7.21	8.32
2	5.84	7.82	7.42	6.62	8.51
3	8.23	7.26	6.68	7.81	7.52
4	5.04	6.42	6.99	5.32	7.73
5	5.03	6.26	7.35	6.92	8.92
6	6.06	6.93	7.66	7.10	8.21
7	4.83	7.62	6.12	7.25	8.03
8	6.34	7.29	6.24	6.63	7.92
9	7.32	6.62	7.36	6.30	8.13
10	8.12	6.84	6.10	8.21	8.05
11	5.12	6.32	7.02	6.62	-
12	6.32	7.36	6.92	6.95	-
13	5.92	6.63	6.43	7.01	-
14	6.32	7.25	6.34	6.62	-
15	5.51	6.81	6.63	6.92	-
Mean + SE	6.01±0.35^{b}	6.86±0.83^{b}	6.69±054^{b}	6.82±0.68^{b}	8.14±3.40^{a}

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

Fig. 12. Total serum protein of cattle from study areas



4.5.2 Serum albumin

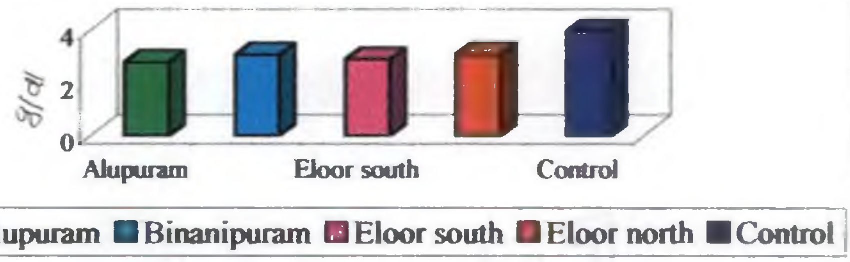
The mean serum albumin (g/dl) concentration in cattle of Alupuram, Binanipuram, Eloor south, Eloor north and control were 2.77±0.38, 3.06±0.27, 2.89±0.35, 3.08±0.27, 3.95±0.40 respectively. All the serum albumin values were significantly lower than controls (Table 16, Fig. 13).

Table 16. Serum albumin values of cattle in Eloor industrial area and control (gm/dl)

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	2.73	3.43	3.12	3.02	4.31
2	2.54	2.62	2.91	3.10	3.63
3	2.98	3.23	2.62	3.82	3.96
4	2.65	2.82	2.83	2.23	3.72
5	2.84	3.05	2.89	3.04	4.12
6	2.16	2.83	2.62	3.12	3.95
7	3.32	3.23	3.02	3.04	3.63
8	2.34	3.51	3.43	3.10	4.23
9	2.42	2.51	2.26	3.04	3.98
10	3.02	3.29	3.02	3.12	3.92
11	3.12	2.73	2.92	3.20	-
12	2.56	3.43	2.64	2.82	-
13	2.93	2.62	2.73	2.92	-
14	2.43	3.26	2.83	3.12	-
15	3.05	2.83	2.92	3.09	-
Mean + SE	2.77±0.38[b]	3.06±0.27[b]	2.89±0.35[b]	3.08±0.27[b]	3.95±0.40[a]

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

Fig. 13. Serum albumin of cattle from study areas



4.5.3 Serum aspartate amino transferase (ASAT)

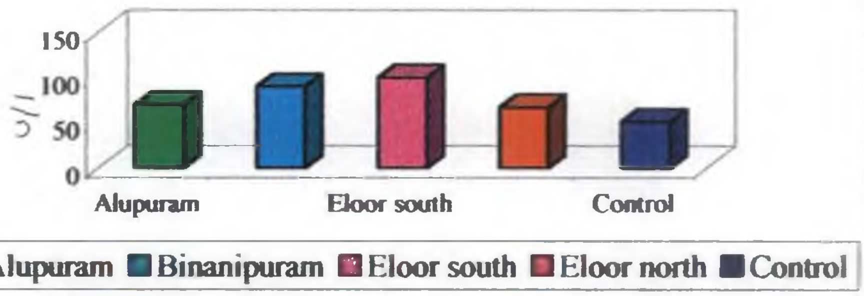
The serum aspartate amino transferase level in Alupuram, Binanipuram, Eloor south, Eloor north and control were 69.67±5.81, 90.27±21.46, 100.33±13.55, 67.20±11.28, 51.40±7.46 respectively. The samples from Binanipuram and Eloor south shows higher values than rest of the samples (Table 17, Fig. 14).

Table 17. Serum arpartate amino transferase in cattle of Eloor industrial area and controls (U/L)

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	78.66	100.83	120.38	87.20	58.20
2	61.82	82.74	98.36	62.12	52.13
3	73.30	90.27	80.36	57.12	44.82
4	65.23	98.38	102.83	78.12	53.81
5	61.82	100.38	110.31	72.83	54.20
6	78.37	82.37	80.13	58.36	48.32
7	69.88	80.36	90.31	62.31	43.85
8	69.10	120.84	106.31	71.23	61.23
9	68.36	98.46	126.12	63.32	48.50
10	71.25	90.50	94.28	59.31	51.31
11	63.71	82.54	108.31	65.32	
12	75.42	110.26	103.12	69.38	
13	68.36	97.31	92.62	68.38	
14	70.39	70.83	97.78	87.39	
15	69.92	83.12	108.21	58.12	
Mean + SE	69.67±5.81^{b}	90.27±21.46^{c}	100.33±13.55^{c}	67.20±11.28^{b}	51.40±7.46^{a}

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

Fig. 14. Serum aspartate amino transferase of cattle from study areas



5.5.4 Serum alanine amino transferase (ALAT)

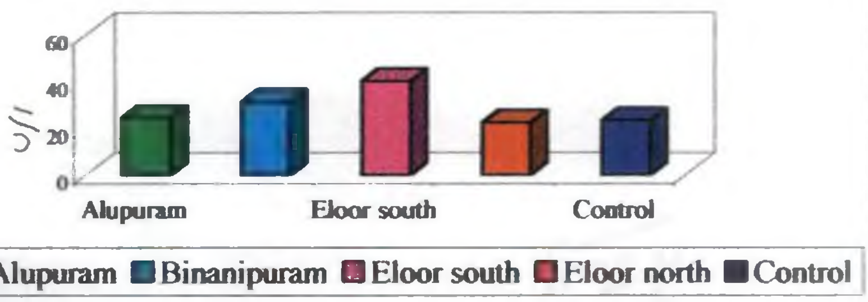
The mean serum alamine amino transferase levels in cattle of Alupuram, Binanipuram, Eloor south, Eloor north and control were 24.53±0.09, 31.33±12.88, 40.07±17.16, 22.47±7.52, 23.30±6.29 respectively. The values around Binanipuram and Eloor south were significantly higher than the control (Table 18, Fig. 15).

Table 18. Serum alanine aminotransferase levels in cattle of Eloor industrial area and controls(Units/litre)

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	22.84	33.42	48.42	24.26	21.42
2	18.32	33.43	40.12	26.23	24.31
3	26.32	29.42	32.56	20.18	23.80
4	30.63	28.17	60.13	18.31	22.12
5	28.32	36.31	40.06	21.13	25.42
6	24.38	32.13	30.23	22.16	24.32
7	20.43	26.11	35.63	23.43	21.68
8	24.54	31.42	42.85	26.12	25.61
9	30.83	38.14	38.23	28.12	22.16
10	32.83	24.21	44.56	18.43	24.28
11	28.53	32.43	36.51	16.12	-
12	18.58	33.12	48.13	22.43	-
13	28.42	31.43	32.63	23.42	-
14	23.12	33.42	42.12	21.12	-
15	26.42	29.42	38.42	23.42	-
Mean +SE	24.53 ± 6.09^{b}	31.33 ± 12.88^{a}	40.07 ± 17.16^{a}	22.47 ± 7.52^{b}	23.30 ± 6.29^{b}

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

Fig. 15. Serum alanine aminotransferase levels of cattle from study areas



4.5.5 Serum alkaline phosphatase

The serum alkaline phosphatase level around Alupuram, Binanipuram, Eloor south, Eloor north and control samples were 272.80±36.71, 252.10±32.13, 211.90±34.97, 187.70±25.71 and 82.40±12.50 respectively. The values around all the test areas were significantly higher than controls (Table 19, Fig. 16).

Table 19. Serum alkaline phosphatase levels in cattle of Eloor industrial area and controls (Units/Litre)

Sl. No.	Alupuram	Binanipuram	Eloor south	Eloor north	Control
1	278.30	256.10	202.80	182.80	98.40
2	256.10	304.40	228.50	193.40	73.80
3	301.80	204.80	245.30	130.60	87.10
4	243.60	272.80	184.80	235.60	68.30
5	260.70	311.50	230.50	207.10	85.30
6	320.10	234.00	180.60	160.80	79.80
7	210.60	196.80	190.80	198.70	83.80
8	240.60	211.80	285.20	207.80	78.90
9	330.80	298.40	130.80	176.80	78.30
10	262.80	238.50	230.80	168.30	92.70
11	243.60	252.60	190.10	162.80	-
12	310.70	278.90	222.80	173.80	-
13	210.80	298.10	203.50	211.20	-
14	330.60	212.80	210.20	198.10	-
15	282.30	254.10	211.10	188.10	-
Mean + SE	272.80±36.71[b]	252.10±32.13[b]	211.90±34.97[b]	187.70±25.74[b]	82.40±12.50[a]

a, b, c - Means with different superscripts within same row differs significantly ($P<0.01$)

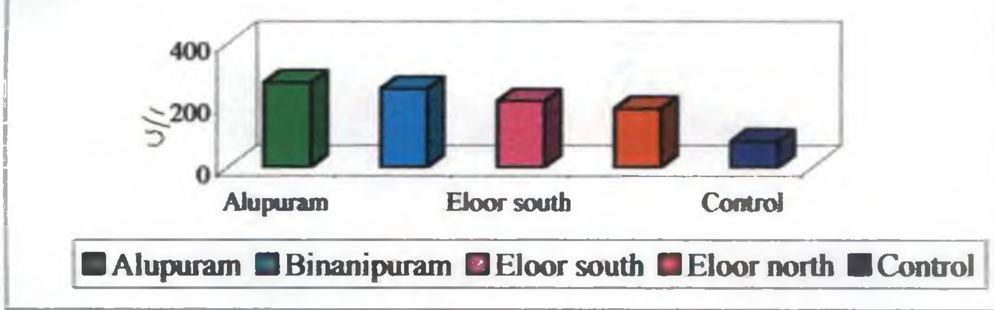


Fig. 16. Serum alkaline phosphatase levels of cattle from study areas

Discussion

5. DISCUSSION

5.1 Retrospective analysis of case sheets

The screening of case sheets from Eloor and Muppathadam veterinary hospitals revealed that, about 56 percent of the total cases recorded were gastrointestinal disorders. The gastrointestinal disorders include mainly anorexia, enteritis and indigestion. These symptoms are suggestive of cadmium toxicity. After oral ingestion, cadmium locates in the GI tract and produce subsequent effects (Miller *et al.*, 1970, 1971). Lynch *et al.* (1976) also recorded reduced feed intake and loss in body weight resulted from cadmium ingestion for prolonged periods. Mennear (1979) opioned that the indigestion caused by cadmium is due to the inhibition of pancreatic secretions. A reduction in growth rate also was reported in cadmium toxicity (Cousins *et al.*, 1973, Lynch *et al.*, 1976).

A very high incidence of reproductive disorders were also evidenced by the case sheet analysis of these hospitals. The incidence of repeat breeders comes about 8.45 per cent in Eloor veterinary hospital and it is comparitively high in Muppathadam veterinary hospital (21.7%). This correlates with the higher content of cadmium in and around Muppathadam veterinary hospital. Cadmium indirectly affect the general body condition of the animals and thereby causing infertility conditions.

According to Roberts(1971) and Arthur *et al.* (1980), low plane of nutrition and disturbed carbohydrate and protein metabolism cause reproductive disorders and infertility. Cadmium is known to disturb carbohydrate metabolism and produce gluclose intolerance and thereby reproductive disorders (Taghi and John, 1973; Fasset, 1975; Mennear, 1979; Gill and Pant, 1983; Reddy and Bhagyalakshmi, 1994). Cadmium is also known to inhibit serum protein metabolism and cause proteolysis (Clark and Clark, 1981, Reddy and Bhagyalakshmi, 1994). This also adversely affect fertility.

In the present study deficiency diseases like hypocalcemia may also contribute to total disease cases which come around 2.77 per cent in Eloor and 7.33 per cent in Binanipuram. Cadmium antagonizes the essential minerals like zinc, selenium, iron and calcium because of divalent nature (Fasset, 1975; Mennear, 1979; Paymbroeck *et al.*, 1982 and Nath *et al.*, 1984). So higher cadmium content in the blood may be one of the cause for hypocalcemia and reduced milk yield.

5.2 Cadmium content in field samples

Cadmium is a rare element in the earth's crust and accumulation of cadmium in water and plants mainly occur from man made sources (Vallee and Ulmer, 1972). In the present study the highest cadmium concentration is found in water samples from Binanipuram (0.04±0.01), followed by area around fertilizer industries (0.03±0.01). The high concentration of cadmium around Binanipuram is due to zinc processing industries as cadmium always found in association with zinc (Carruthers and Smith, 1979, Mennear, 1979). The concentration of cadmium in water from Eloor south is high (0.03±0.01) due to fertilizer industries because of high level of cadmium in rock phosphate fertilizer (Mennear, 1979).

The normal fresh water concentration of cadmium is less than 1 ppb (Fasset, 1975). Maximum permissible level for drinking water is 0.01 ppm (Camrin,A.M, 1973). The permissible limits prescribed for industrial effluents by the State Pollution Control Board is about 2 ppm. The samples analysed in the present study were mainly from the wells. The water samples contain high concentration of cadmium than permissible level in Alupuram (0.03±0.01), Eloor south (0.03±0.01) and Binanipuram (0.04±0.01).

Uptake of cadmium by plants is generally poor particularly from clay soil. Forages and crops grown on normal soil usually contain less than 1 ppm. Occasionally soils became enriched with cadmium from cadmium rich fertilizers or the dispersal of wastes from the mining and smelting of metals like zinc and lead (Underwood and Suttle, 1999). In the present study cadmium concentration in plants were highest in samples around Binanipuram (6.53±1.28) followed by Eloor south (6.00±4.85) and Alupuram (3.67±0.64). The high level of cadmium in plant samples around Eloor south may be due to contamination of pastures by sewage sludge from phosphate fertilizer industries. The high concentration of cadmium around Alupuram is due to increased soil leaching of cadmium because of high acid sulfate and nitrate in the air (Fasset, 1975). The plants around Binanipuram contain high concentration of cadmium due to zinc processing industries. This is in agreement with the findings of Carruthers and Smith (1979) and Mennear (1979) that plants in the vicinity of zinc processing industries contain high concentration of cadmium.

According to Underwood and Suttle (1999) the normal cadmium content in the diet of ruminants should not exceed the range of 0.1-0.2 mg kg^{-1} dry matter (DM). In the present study all the plant samples collected contain more than 0.5 ppm of cadmium which is higher than permissible level causing health hazard to cattle population.

5.3 Cadmium content in biological samples

The results of field samples analysed were supported by results of analysis of biological samples like blood, serum, faeces, urine and milk.

The blood cadmium concentration is highest around Eloor south (3.33±0.84) followed by Alupuram (1.72±0.55). All the samples showed cadmium concentration above control (0.70±0.21).

The serum cadmium values were highest around Binanipuram (0.37±0.15) followed by Eloor north (0.29±0.06). The levels of cadmium in blood and serum is not always proportionate as cadmium in blood are partitioned between fluid part and erythrocyte part, because of mobilization and redistribution of cadmium (Mennear, 1979). In the present study also serum concentration is not proportionate with blood concentration (Table 4).

The cadmium concentration in dung is highest in samples around Eloor south (7.43±1.58) and Binanipuram (3.33±0.90). The excretion of cadmium in dung was found to be proportional with the intakes. About 90 per cent of total oral intake is excreted in the faeces (Miller *et al.*, 1970, Miller *et al.*, 1971).

The urine cadmium concentration was highest in samples around Eloor south (2.90±1.12). This is also found to be proportionate with intake and blood cadmium concentration. A major part of cadmium entering into animal body is concentrated in the kidney (Miller *et al.*,· 1970, 1971, Johnson *et al.*, 1981). Cadmium is stored in the kidney in a bound form with cadmium binding protein known as metallothionein, finally causing tubular dysfunction (Fasset, 1975). Cadmium concentration in milk was highest in samples around Eloor south (3.11±1.32). It is 12 times more than that of control values (0.26±0.13). All the test samples show increased cadmium concentration than those recommended by Dwivedi *et al.* (1997) that is (0.004 μg ml^{-1}).

5.4 Haemogram

The cadmium content in the blood and serum collected from the animals in the test areas are higher than control.. The levels of blood and serum cadmium are not proportionate. These observations are similar to a report by Mannear (1979). He observed that cadmium was cleared rapidly from blood during parenteral and accidental intakes. But in chronic cases, blood cadmium continued to rise and plateau after 50 days, then the cadmium binding site in the blood became saturated leading to a steady state exchange between the accumulated cadmium and newly entered cadmium. During the time of its existence in blood, the cadmium might induce changes in blood components.

In the present study a significant reduction in packed cell volume and haemoglobin were noticed in Binanipuram and Eloor south area samples. This may be due to increased cadmium levels in blood of cattle in these areas because cadmium is known to reduce haemoglobin concentration (Lynch *et al.*, 1976). Cadmium being an element of bivalent nature it will replace all the cations of same valency like zinc, selenium, iron and calcium thereby interfering their utilization (Mennear, 1979, Nath *et al.*, 1984). Fasset (1975) reported hypochromic anemia in cadmium ingested rats as cadmium replaces iron from haemoglobin. Thus, reduced levels of haemoglobin and packed cell volume in cattle of Binanipuram and Eloor south area correlate to their increased cadmium concentration.

The other haemotological values like total erythrocyte count, total leucocyte count, differential lencocyte count are not showing any significant difference between the test area samples and control. All these haematological values are in the normal range for cattle (Swenson and Reece, 1976).

5.5 Serum values

The serum total protein and albumen levels were reduced in all test areas than control.. Cadmium is known to exert its toxic effect by inhibiting sulphydril group of enzymes. It also inhibit serum protein metabolism (Clark and Clark, 1981).

An increase in protease activity by cadmium causing proteolysis and transamination of amino acids are reported by Reddy and Bhagyalakshmi (1994). It is found that cadmium induces increased protein excretion by causing tubular dysformation (Fasset, 1975 and Novelli *et al.*, 1998). Lauwerys *et al.* (1982) also reported an increase in excretion of high molecular weight proteins. So these findings support the present observation of reduced serum total protein and albumin in animals in the test areas with increased cadmium levels.

The serum enzyme values like aspartate amino transferase, alanine amino transferase and alkaline phosphatase levels in cattle from all test areas are higher than controls. Cadmium induces increased alanine amino transferase activity as it causes renal and hepato- toxicity (Faeder *et al.*, 1977, Novelli *et al.*, 1998). An increase in alanine amino transferase activity due to cadmium toxicity was reported by Reddy and Bhagyalakshmi (1994). Usually damage to heart, liver, skeletal muscles, kidney and brain tissues, haemolysis and stress cause an increase in the serum aspartate levels. In tissue injury these will be 3 to 8 fold increase in enzyme levels in the serum (Coles, 1986). Since the alanine amino transferase level in the present study is not increased that much, cadmium may not be cause discernible tissue damage to these organs. Membrane damage to vital organs like heart, liver, kidney and lung cause 2 to 4 fold increase in aspartate amino transferase (Coles, 1986). In cattle serum aspartate amino transferase concentration test areas showed an increasing tendency in test areas, but these difference seen is not clinically significant. It indicates that existing cadmium level do not cause any appreciable damage to these organs. Four fold increase in alkaline phosphatase level is suggestive of some involvement of bone tissues (Coles, 1986). Since the increase in alkaline phosphatase levels in test areas is less than this indicating that bone tissue is unaffected by this level of cadmium.

So the enzyme values higher than normal indicate some degree of tissue or cellular disruption, which may not be always indicative of disease (Shihabudeen, 2000).

The present study illustrated the cadmium induced toxicity in cattle of Alupuram, Binanipuram, Eloor South and Eloor North areas and awareness needs to be created in farmers and veterinarians in these areas.

Summary

6. SUMMARY

A study was undertaken in Eloor industrial area of Kerala state to find out the extent of environmental pollution with cadmium and its impact on cattle health. Eloor industrial area consists of seven major industrial units viz., Fertilizers and Chemicals Travancore (FACT), Indian Aluminium Company (INDAL), Travancore Cochin Chemicals (TCC), Travancore Chemicals and Manufacturing Company (TCM), Hindustan Insecticide Limited (HIL) and Binanizinc. Of these FACT, INDAL and Comincobinani are the major industrial units concerned with cadmium pollution. So these areas are selected for the conduct of the study.

To investigate the type of disease conditions prominent in the cattle population of Eloor, retrospective analysis of case sheets for the period from 1994 to February 2000 was carried out in nearby veterinary hospitals (Eloor and Muppathadam). The diseases related to cadmium toxicity were noted in detail.

Field samples like water and plants and biological samples like blood, dung, urine and milk from cattle were collected and analysed for cadmium content . Blood and serum samples were also collected to study haematology and serum biochemistry.

The collected field and biological samples were digested by special tissue digestion apparatus and analysed the cadmium content by Atomic absorption spectrometry. Blood samples were tested for total erythrocyte count, total leucocyte count, differential leucocyte count, packed cell volume, haemoglobin, mean corpuscular haemoglobin, mean corpuscular haemoglobin concentration and mean corpuscular volume. Serum samples were also analysed for total proteins, albumin, serum enzymes like aspartate aminotransferase, alanine aminotransferase and alkaline phosphatase.

Ten cattle above 3 years of age were selected from University Livestock Farm, Mannuthy as control. Biological samples like blood, serum, milk, urine and dung were collected from these animals for the analysis of cadmium. Field samples like water and fodder were also collected from University Livestock Farm, Mannuthy. The data obtained were statistically analysed by Completely Randomized Design (CRD) as per method described by Snedecor and Cochran (1967).

The retrospective analysis of 1503 case sheets from Eloor veterinary hospital showed 851 digestive disorders, 126 repeat breeders, 75 respiratory diseases and 41 deficiency diseases. Out of 5920 case sheets analysed in Muppathadam veterinary hospital 3348 digestive disorders, 1284 repeat breeders, 398 respiratory diseases and 433 deficiency diseases were observed. The major disease conditions like digestive disorders, deficiency diseases and repeat breeding are suggestive of cadmium toxicity.

Water and forage samples from study area showed significantly higher cadmium content than controls. The cadmium content (ppm) in water and forage from Alupuram (0.03±0.14, 3.67±0.64), Binanipuram (0.04±0.01, 6.53±1.28), Eloor south (0.03±0.01, 6.00±4.85) and Eloor north (0.02±0.06, 2.60±0.70) were higher than control samples (0.01±0.01, 0.65±0.29).

The cadmium content (ppm) in biological samples like blood, serum, dung, urine and milk from the cattle of Alupuram (1.23±0.35, 0.37±0.15, 3.33±0.90, 0.63±0.48, 0.26±0.13), Binanipuram (1.72±0.55, 0.23±0.07, 2.23±0.54, 0.60±1.67, 0.40±0.11), Eloor south (3.33±0.84, 0.20±0.08, 7.40±1.58, 2.90±1.12, 3.11±1.32) and Eloor north (0.41±0.15, 0.29±0.06, 1.60±1.58, 0.24±0.08, 0.03±0.02 respectively). All the values were significantly higher than control values.

A significant reduction was noticed in total erythrocyte count, haemoglobin and packed cell volume of the animals in the affected area. Total leucocyte count, differential leucocyte count, mean corpuscular volume, mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration did not show any significant difference.

Total serum protein and albumin values from cattle of Alupuram, Binanipuram, Eloor south and Eloor north were significantly lower than control values. Significant increase in serum aspartate aminotransferase, serum alanine amino transferase and serum alkaline phosphatase were noticed in cattle from all test areas than control . The present investigation has lead to the following salient observations.

1. Cadmium content in field and biological samples of all zones of Eloor industrial area were higher than control.
2. The highest pollution level of cadmium was found around fertilizer and zinc processing industries.

Since treatment is ineffective in long term effects produced by chronic cadmium toxicity, it is better to take preventive measures like supplementation of feed with iron ,selenium ,magnesium,calcium,zinc and chromium to protect cattle health. The findings of the study will help the industrial authority and veterinary professionals to take preventive measures, thereby making cattle rearing more economic in Eloor.

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ASSESSMENT OF CADMIUM TOXICITY IN CATTLE OF ELOOR INDUSTRIAL AREA

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ABSTRACT OF THE THESIS

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ABSTRACT

A study was undertaken to assess impact of environmental pollution with cadmium in cattle of Eloor industrial belt.

Areas around Fertilizers and Chemicals Travancore (FACT), Indian Aluminium Company (INDAL), Binani Zinc and Hindustan Insecticides Limited (HIL) were selected for the study.

As the first step, retrospective analysis of case sheets was done in Eloor and Muppathadam Veterinary hospitals. In Eloor veterinary hospital out of the 1503 case sheets examined 851 digestive disorders, 126 repeat breeders, 75 respiratory diseases and 41 deficiency diseases were observed. Out of 5920 case sheets analysed in Muppathadam veterinary hospital 3348 digestive disorders, 1284 repeat breeders 398 respiratory diseases and 433 deficiency diseases were observed. Disease conditions like digestive disorders, deficiency diseases and repeat breeders are suggestive of cadmium toxicity.

The field samples like water and forages, biological samples like blood, serum, urine, dung and milk were collected from cattle in the industrial field localities. The cadmium content in this field and biological sample were estimated by Atomic Absorption Spectrophotometer. Animals kept in the University Livestock Farm, Mannuthy were taken as controls. Field samples like water and fodder were also collected from area around University Livestock Farm, Mannuthy.

The cadmium content of water from Alupuram, Binanipuram, Eloor south, Eloor north ranges from 0.03-0.04 ppm. These were significantly higher than controls (0.01 ppm). The cadmium content of fodder from Alupuram, Binanipuram, Eloor south, Eloor north ranges from 2.60-6.53. These were also significantly higher than controls (0.65 ppm).

The cadmium level of blood, serum, milk, urine and dung from cattle of Alupuram, Binanipuram, Eloor south and Eloor north were significantly higher than controls.

Haematological values like total erythrocyte count, haemoglobin and packed cell volume showed significant decrease in cattle of test areas than controls. Other haematologic values like differential leucocyte count, total leucocyte count, mean corpuscular volume, mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration did not show any significant changes. Total serum protein and albumin values from cattle of Alupuram, Binanipuram, Eloor south and Eloor north were significantly lower than control values. Serum enzymes like aspartate amino transferase, alamine amino transferase and alkaline phosphatase levels were higher in cattle from test areas than controls. It can be concluded that field and biological samples collected from the vicinities around Fertilizers and Chemicals, Travancore (FACT), Binani Zinc and Indian Aluminium Company (INDAL) are contaminated with cadmium.