ASSESSMENT OF MERCURY TOXICITY IN CATTLE OF ELOOR INDUSTRIAL AREA

By

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THESIS

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DEPARTMENT OF PHARMACOLOGY AND TOXICOLOGY
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
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DECLARATION

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

Mannuthy
1.11.2000.

A. T. _____

A.THIRUNAVUKKARASU

To

My beloved

Parents & sister

CERTIFICATE I

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

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A. THIRUNAVUKKARASU

CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

INTRODUCTION

India is endowed with the largest livestock population in the world. It accounts for 57 per cent of world's buffalo population and 15 per cent of cattle population. India is the largest producer of milk in the world (India, 2000). With about 48 per cent of milk supply from cow (India livestock sector Review, 1999), animal husbandry and dairy sector play a prominent role in rural economy in supplementing the income of rural households, particularly the landless, small and marginal farmers. These facts imply the growing concern over the factors affecting the cattle health and thereby production.

Other than the disease conditions affecting cattle, the insight into pollutants especially industrial pollutants is gaining momentum day by day. Though the chemical pollutants are studied commonly, the catastrophic outbreaks like "Minamata disease" in Japan, (Cassidy and Furr, 1979) and mercury poisoning in Iraq (Bakir *et al.*, 1980) have turned toxicologists' and environmentalists' view point towards the possible heavy metal especially mercury poisoning from industries.

Mercury (Hg) is "the hottest, the coldest, a true healer, a wicked murderer, a precious medicine, and a deadly poison, a friend that can flatter and lie" (Woodall, 1639). Mercury, a naturally occurring highly toxic heavy metal moves through water, air and soil as a result of natural and human activities. It enters the environment from sources like chloralkali plants, electrical equipment industry, coal-fired power plants, mining and smelting of various ores, gold and silver mining, paint industry, textile and dye industry, thermometer industry and dental amalgamation units. Because it is a Persistent, Bioaccumulative Toxicant (PBT), the amount of mercury in the biosphere has been increasing since the beginning of the industrial age. Further, mercury is also cited to be a global pollutant (Rudd, 1998). About 10,000 tons of mercury are mined each year, half of which is lost in the environment.

Mercury toxicity can result from three different forms of it, elemental, inorganic or organic mercury compounds. It can be absorbed through respiratory tract, skin or digestive tract and give rise to poisoning. The toxicity is related to its affinity to form tight co-ordinate bonds with sulphydryl groups, diffusely disrupting enzyme systems of brain, kidney, liver and lungs.

Mercury in the environment can be converted to compounds, which can be carried through food chain to human and animals. Methyl mercury may be formed in water and soil, accumulated in fish and biomagnified through food chain.

In Kerala, as per records of State Industrial Department, a total of about 197 medium and large scale industries exist. Under small scale industrial sector, a total of about 1721 industries are present (Cheeran *et al.*, 1987). These industries cause pollution ranging from marginal to intense in the surrounding environment.

The Central Pollution Control Board, in consultation with State Pollution Control Boards has identified critically polluted areas in the country which need special attention for control of pollution. In Kerala, Udyogamandal (Eloor) is identified to be such an area (India, 2000). There are about seven major industries comprising Fertilizers and Chemicals Travancore Ltd., Travancore Cochin Chemicals, Indian Aluminium Company, Catalyst India Limited, Indian Rare Earths, Hindustan Insecticides Limited, Travancore Chemicals and Manufacturing Company, FACT Petrochemical division and Ammonia plant. Cheeran *et al.* (1987) in their preliminary investigation. reported,increased mercury levels in field samples and bio-samples collected from Eloor area.

In the light of above observations, Eloor industrial area is selected for the present study to assess the extent of mercury pollution by industries at Eloor.

The objective of the study is to,

1) Assess the extent of environmental pollution with mercury by industrial effluents in Eloor.
2) Evaluation of its impact on health of cattle population.

This study will give an indication on level of mercury in environment and biomaterials of cattle. Further it will help veterinarians to take appropriate remedial measures. Also it will make awareness among industries concerned and in public.

REVIEW OF LITERATURE

2. REVIEW OF LITERATURE

2.1 Mercury Toxicity

2.1.1 Humans

The "Minamata bay disaster" of Japan is a historically well known methylmercury poisoning. From 1930's to 1960's a chemical company dumped tonnes of mercury in the Minamata bay in Japan. Thousands of people living around the bay developed methylmercury poisoning through the consumption of contaminated fish and is known as "Minamata disease". Animals and birds living around the bay were also affected (Cassidy and Furr, 1979).

The most catastrophic outbreak occurred in 1972 at Iraq. Consumption of wheat and barley seeds treated with methyl mercury resulted in 6530 victims and 500 deaths (Bakir *et al.*, 1980).

2.1.2 Ruminants

2.1.2.1 Cattle

Irving and Butler (1975) presented a case report of ammoniated mercury toxicity in cattle treated topically with an inorganic mercurial ointment.

Boyd (1985) stated organomercuric poisoning in fat cattle fed with phenyl – mercuric acetate treated barley.

Clinico – biochemical changes induced with mercuric chloride as intravenous injections (1 mg/kg and 2 mg/kg body weight) were studied by Parai and Pandey (1992).

Simpson *et al.* (1997) diagnosed poisoning of dairy heifers with mercurous chloride. The animals were poisoned by ingestion of soil contaminated with mercurous chloride.

2.1.2.2 Sheep

Robinson and Trafford (1977) conducted a study on early changes of urinary enzymes like alkaline phosphatase, leucine amino peptidase, γ-glutamyl transpeptidase, β-glucuronidase, aspartate aminotransferase and lactate dehydrogenase by inducing nephropathy in sheep using mercuric chloride. Six adult cross-bred ewes were given a single i/v dose of mercuric chloride solution at three different doses (0.1, 0.25 and 0.5 mg/kg body weight), two sheep being injected with each dose. The results showed increased alkaline phosphatase and γ-glutamyl transpeptidase activity in urine.

2.1.2.3 Goat

Kumar and Pandey (1993) studied clinico-haematological profile in mercuric chloride induced nephrosis in goats. Nephrosis was induced by intravenous injection of mercuric chloride @ 0.2 mg/kg body weight in 3ml of normal saline on days 0,7 and 14 followed by @ 0.4 and 0.8 mg/kg body weight in 3 ml of normal saline on days 21 and 28 respectively. Clinical observations showed dullness, anorexia, kyphosis, muscular weakness, diarrhoea, dehydration, generalized alopecia and scabby lesions around the anus. Haematological observations showed significant leucocytosis, neutrophilia, monocytosis and lymphopenia.

Biochemical changes in blood and urine of the same goats were also reported by Kumar and Pandey (1994). There was marked increase in the levels of serum urea nitrogen, creatinine and marginal increase in the alkaline phosphatase. Biochemical changes in urine included marked decrease in the levels of urea nitrogen, creatinine and inorganic phophorus with a marked elevation of alkaline phosphatase.

Chronic toxicity of inorganic mercury in goats was induced by giving mercuric chloride solution (100 μg/ml) orally *ad lib* for 90 days. The study was

conducted by Pathak and Bhowmik (1998) to evaluate the effect of chronic toxicity on humoral immune response in goats exposed to an antigen. The results clearly demonstrated that mercury given to goats interfere with their ability to form normal circulating levels of antibodies resulting in immunosuppression.

Kumar and Pandey (1998) conducted an experiment on acute mercuric chloride intoxication in goats in order to study the biochemical alterations in blood and urine. Acute toxicity was induced by single intravenous injection of mercuric chloride @ 1.5 mg/kg body weight in 3 ml of normal saline solution. The results showed increased serum urea nitrogen and creatinine but serum total proteins and alkaline phosphatase values remained unaffected. Urine showed marked decrease in urea nitrogen, creatinine and inorganic phosphorus but marked elevation in urine alkaline phosphatase level.

2.1.3 Non-ruminants

2.1.3.1 Horse

Short and Edwards (1988) reported a case of mercury poisoning in a thorough bred race horse after it was liberally treated with a blistering agent containing mercury.

2.1.3.2 Pigs

Raszyk *et al.* (1997) conducted immunological studies on porcine and bovine to assess the effects of mercury. They found decreased blood level of lysozyme (0.92 mg/l) and lymphocytopenia leading to immunosuppression.

2.1.4 Birds

The toxic effects of feeding ethyl mercury chloride on chicken were studied by Soudi *et al.* (1976). The chicken were fed with wheat containing 40 per cent ethyl mercuric chloride for 88 days. The amount of mercury residues in egg

white was almost 3 times as much as that in yolk. The liver and kidneys accumulated highest amounts of mercury.

Pribilincova *et al.* (1996) studied the effects of phenyl mercury on reproductive performance in laying hens. Mercury was administered in feed at dosages of 5 and 30 mg/kg. Mercury exposure did not affect egg shape, egg albumin height, and yolk colour but egg shell thickness and egg weight were decreased.

The toxicity of mercury and methyl mercury to birds, terrestrial and aquatic life was reviewed by Wolfe *et al.* (1998).

The immunopathological effect of mercuric chloride on humoral immune response in chickens was recorded by Kumar *et al.*(1999). It was suggested that mercury @ 125 ppm may be immunotoxic and affect the immune system of poultry adversely which may lead to vaccination failures.

2.1.5 Lab animals

2.1.5.1 Rabbits

The immunosuppressive effects produced by lead, cadmium and mercury in rabbits were compared by Koller (1973). Mercuric chloride (10 ppm) was given through water for 70 days. The results showed marked immunosuppression in rabbits treated with mercuric chloride.

2.1.5.2 Rats and mice

Morcillo and Santamaria (1996) observed mercury distribution and renal metallothionin induction after subchronic oral exposure of mercuric chloride (5, 50 and 500 mg via drinking water for 8 weeks) in rats. The greatest concentration of mercury was found in kidneys.

The kinetics of methyl mercury and inorganic mercury in lactating and non-lactating mice were investigated by Sundberg *et al.* (1998). The lactating and non-lactating mice were given an intravenous injection of 0.5 mg Hg/kg body weight as mercuric chloride or as methyl mercuric chloride. The plasma clearance and volume of distribution at steady state for methyl mercury were higher in lactating than in non-lactating mice. There were no differences in pharamacokinetic parameters of lactating and non-lactating mice treated with inorganic mercury.

Newland and Reile (1999) measured blood and brain mercury levels after chronic gestational exposure to methyl mercury (0.05 and 6ppm Hg as methyl mercuric chloride in drinking water). The quantity of mercury in brain of off-spring decreased between birth and weaning. The level of mercury in blood and brain of dams were closely related to oral consumption during gestation.

2.1.5.3 Guinea pigs

Iverson *et al.* (1973) displayed acute toxicity, tissue distribution and decay profiles of methyl mercury in guinea pigs. Kidney and liver consistently contained the highest levels of mercury and plasma had the lowest level.

2.2 Sources of mercury toxicity

There are many sources of contamination of mercury (Lofroth, 1970) from paper and pulp factories to organic and inorganic compounds used for plant protection measures. The major source of mercury in the environment is the natural degassing of earth's crust. Metallic mercury and its inorganic salts are used extensively in medicine as diuretics, laxatives, skin ointments, antiseptics and vesicants. Irving and Butler (1975) reported ammoniated mercury toxicity in cattle when it was applied topically. Mercury was also used as a fungicide which acts as a source of toxicity (Neathery and Miller, 1975). Short and Edwards (1988) reported a case of mercury poisoning in a race horse after it was treated with a

blistering agent containing mercury. Fishes also act as a source of mercury poisoning when consumed because of bioaccumulation in them (Holsbeek *et al.*, 1997).

Mercury in soil - plant - animal system further act as a source of toxicity to human (Kralovec and Slavik, 1997). Experiment conducted by Vimy *et al.* (1997) showed mercury from maternal "silver" tooth fillings in sheep and human milk as a source of neonatal exposure. Atmospheric deposition of mercury in the sandy soils surrounding the chloralkali plant act as a main source of contamination (Inacio *et al.*, 1998).

Lacerda *et al.* (1998) have reviewed the current information on mercury from gold mining, its cycling in environment and its long term ecological impact. Morel *et al.* (1998) stated that elemental mercury pollution originates from industrial sources like power plants. There is also possibility of dangers to babies and young children of exposure to methyl mercury from fish eaten during pregnancy and while nursing (Grawe, 1999).

2.3 Mercury levels

Iverson *et al.* (1973) induced acute toxicity with methyl mercuric chloride (1 mg Hg/kg body weight, orally) in guinea pigs and studied tissue distribution. Kidney and liver consistently contained the highest (7.63 ± 0.2 ppm and 4.50 ± 0.22 ppm respectively) levels of mercury and plasma the lowest (0.03 ± 0.001 ppm). In Central Nervous System (CNS), the concentration of mercury decreased in the order cerebrum > cerebellum > spinal cord.

Five per cent ammoniated mercury ointment when applied externally for ring worm infection caused toxicity. Irving and Butler (1975) found that kidney contained 5.5 ppm of mercury while urine and blood contained less than 0.01 ppm of mercury.

Boyd (1985) described organomercuric poisoning in fat cattle which were fed phenyl mercuric acetate-treated barley seeds. The tissue mercury levels of liver and kidney were analysed to be 11.7 ppm and 43.5 ppm respectively.

Cheeran *et al.* (1987) analysed mercury levels in different samples collected from Eloor industrial area of Kerala. The mean mercury levels found in samples were blood - 0.027 ppm, Effluents - 1.74 ppm, milk - 0.07 ppm, urine - 0.014 ppm, Dung - 0.118 ppm, feed - 0.024 ppm, fodders - 0.09 ppm and solid wastes - 0.14 ppm.

Mercury content of Ultra High Temperature (UHT) milk, pasteurised milk, evaporated milk, natural and fruit yoghurts were estimated by Gajewska *et al.* (1994). Values for mercury in milk ranged from 0.4 to 29.7 µg/100 ml and those in yoghurts ranged from 1.4 to 22.9 µg/kg. The highest values were found in UHT skim milk and in fruit yoghurt.

Total mercury concentration in the muscle of common fresh water and anadromous species from Bangladesh were low, varying from 2 to 430 mg/g fresh weight (Holsbeek *et al.*, 1997).

Estimation of mercury levels in various feed samples revealed that feeds of pig generally contained more mercury than those for cattle and horses. Poultry feeds contained intermediate amounts of mercury. Processed fish was a significant source of mercury to the feeds (Sager *et al.*, 1997).

Rule and Iwashchenko (1998) estimated the mean soil mercury concentration as 1.06 mg/kg for samples collected within 2 km of a former chloralkali plant in Virginia, USA.

2.4 Pathological changes of mercury toxicity

There was relative increase in accumulation of mercury in organs with respect to increase in body weight of fattening bulls following the intake of mercury contaminated feed stuff (Jonas and Kretzschmar, 1979).

Mercury contents were lower in meat than in kidney and liver samples collected from sheep in agricultural and industrial areas of Bavaria (Knoppler *et al.*, 1979).

Continuous exposure of experimental animals to heavy metal environmental pollutants like mercury, cadmium and lead were reported to cause impairment of both humoral and cell mediated immunity responses (Koller, 1980).

Short and Edwards (1988) found kidney having the highest concentration of mercury followed by liver, intestine, esophagus and stomach of horse poisoned with mercury.

Kumar *et al.* (1992) in their study of mercuric chloride induced nephrotoxicity (0.2 mg/kg body weight in 3 ml normal saline on days 0, 7 and 14 followed by @ 0.4 and 0.8 mg/kg body weight on days 21 and 28 respectively) in goats revealed that adverse effects of mercury were dose-dependant with cumulative effect on renal tissue.

Gajewska *et al.* (1994) identified mercury levels in milk and milk products.

Significantly elevated mercury levels were found in wool of sheep exposed to mercury (Gebel *et al.*, 1996).

MATERIALS & METHODS

3. MATERIALS AND METHODS

In Kerala, Eloor is an industrial area where several industries are contributing towards the environmental pollution (Fig. 1). The whole Eloor industrial area was considered for study and carried out in five phases.

3.1 Layout of the study

3.1.1 Phase One

Ward-wise survey and interview of the farmers to appraise the total cattle population and problems faced by farmers in raising cattle at Eloor industrial belt.

3.1.2 Phase Two

Visit to veterinary hospital in Eloor and survey of incidences of diseases in the area.

3.1.3 Phase Three

Based on the results of the study conducted by Cheeran *et al.* (1987), Eloor industrial area was divided into Eloor East, West, North and South (Fig.2). The details of the industrial units in this areas are furnished below:

3.1.3.1 Eloor East

Eloor East comprises of Travancore Cochin Chemicals (TCC), Fertilizers and Chemicals Travancore (FACT) and Indian Aluminium Company (INDALCO or IAC).

3.1.3.1(i) Fertilizers and Chemicals Travancore (FACT)

FACT is manufacturing nitrogenous as well as phosphatic chemicals. The chemical wastes are released in to the surroundings and in river Chaliyar as

Fig.1. Map of Kerala showing the proposed area of study

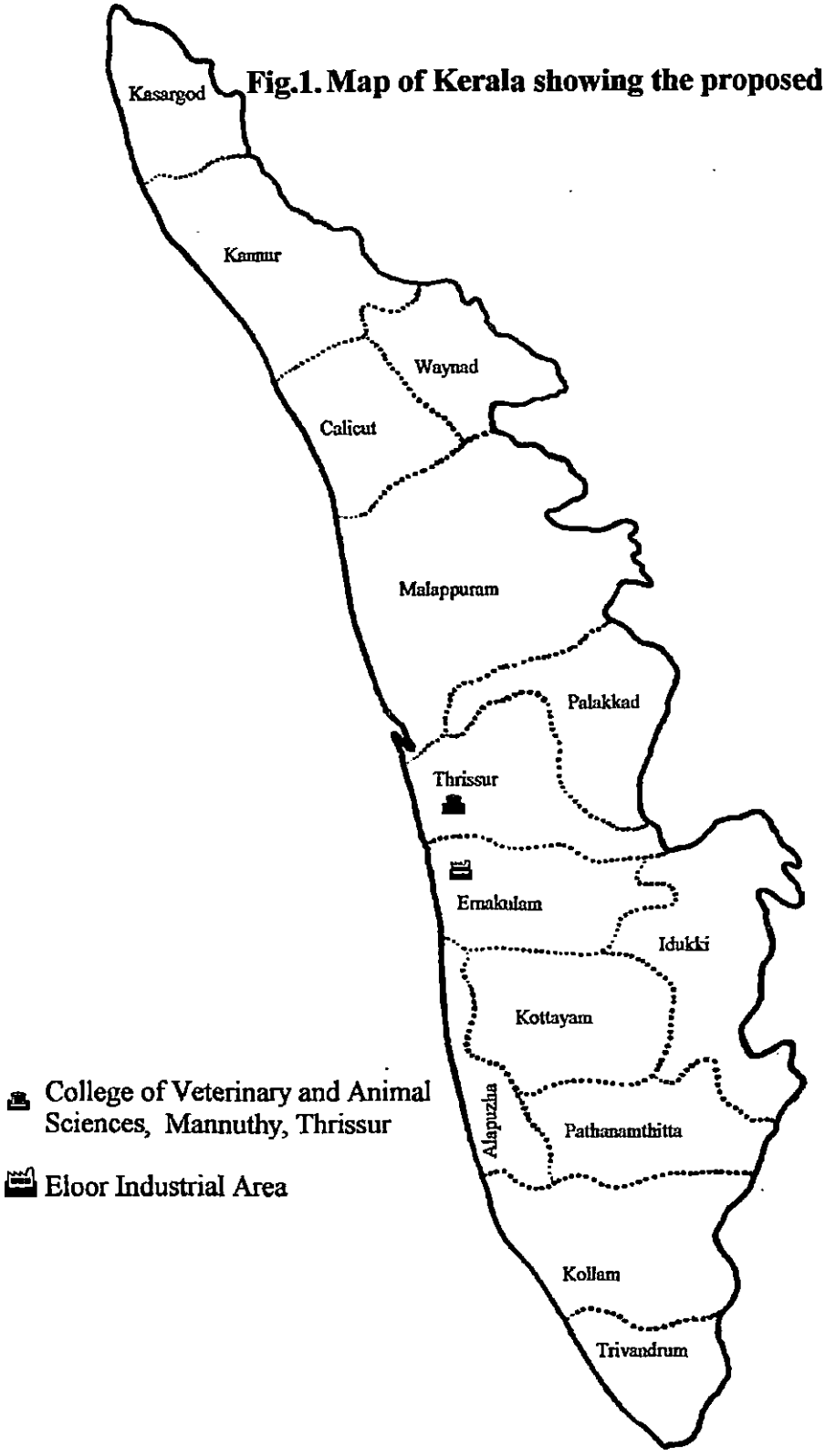
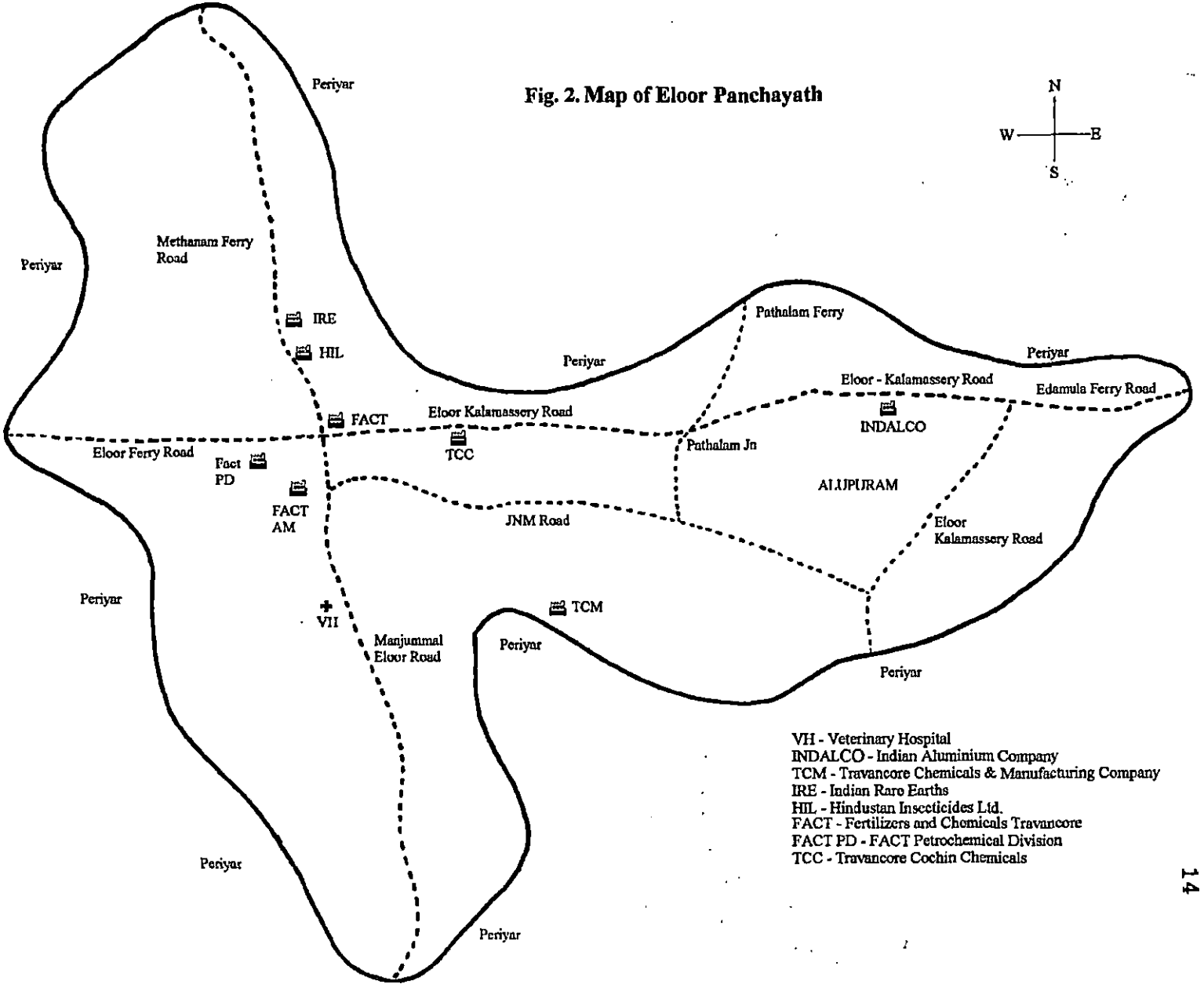


Fig. 2. Map of Eloor Panchayath



VH - Veterinary Hospital
INDALCO - Indian Aluminium Company
TCM - Travancore Chemicals & Manufacturing Company
IRE - Indian Rare Earths
HIL - Hindustan Insecticides Ltd.
FACT - Fertilizers and Chemicals Travancore
FACT PD - FACT Petrochemical Division
TCC - Travancore Cochin Chemicals

effluents. Other than this FACT possesses separate Ammonia plant and petrochemical division.

3.1.3.1(ii) Travancore cochin chemicals (TCC)

TCC manufactures caustic soda, liquid chlorine, hydrochloric acid and sodium sulphide. Caustic soda is manufactured by electrolysis of brine using graphite anode and steel cathode with which sodium liberated from brine forms amalgam and liberates itself in the presence of water. The cathode usually contains mercury and as a result of this, traces of mercury and its compounds can be released in to the effluents.

3.1.3.1(iii) Indian Aluminium Company (IAC)

IAC produces aluminium ingots by electrolysis of bauxite and cryolite. This process liberates large quantities of fluorine which is discharged into surroundings through effluents.

3.1.3.2 Eloor West

Eloor west area has no major industries contributing to the heavy metal pollution. So this area was not considered for the study.

3.1.3.3 Eloor North

Indian Rare Earths (IRE) and Hindustan Insecticides Limited (HIL) are the two industries in Eloor North.

3.1.3.3(i) Indian Rare Earths (IRE)

Radio active mesothorium is manufactured and stored in this factory. Radio active barium sulphate and lead sulphide are discharged through effluents in traces. The effluents are also reported to carry phosphates, sulphates and chlorides.

3.1.3.3(ii) Hindustan Insecticides Limited (HIL)

HIL produces DDT, BHC and Endosulfan. A number of hazardous chlorinated organic compounds are utilized in the factory either as basic chemicals or as intermediaries, for the production of plant protection chemicals. Mercury can possibly be a pollutant discharged from this industry.

3.1.3.4 Eloor South

Travancore Chemicals and Manufacturing Company (TCM) is the only major industry in Eloor south.

3.1.3.4(i) Travancore Chemicals and Manufacturing Company (TCM)

TCM produces copper sulphate, copper oxychloride and potassium chlorate. Traces of these chemicals, mineral acids, salts and mercury may be discharged into the surroundings from this industry.

Phase three involved collection of field samples and bio-samples from cattle of the above said areas.

* Field samples include

a) Water samples collected from wells, marshy areas and water bodies to which effluents were released.
b) Surface sludge where solid wastes were disposed.
c) Samples of forage and feeds at sampling sites.

* Biological samples like urine, dung, milk and blood (heparinised and EDTA added) were collected from adult cattle at sample sites.

3.1.4 Phase Four

The collected samples were analysed in the laboratory for the following:

(a) Levels of mercury in field and biological samples.

(b) Hematological parameters - Total Erythrocyte Count, Total Leucoyte Count, Packed Cell Volume (PCV). Differential Leucocyte Count (DLC), Haemoglobin per cent (Hb%), Mean Corpuscular Volume (MCV), Mean Corpuscular Haemoglobin (MCH) and Mean Corpuscular Haemoglobin Concentration (MCHC).

(c) Total serum proteins (albumin and globulin)

(d) Serum enzymes level - Alkaline Phosphatase (ALP), Alanine Amino Transferase (ALAT), Aspartate Amino Transferase (ASAT), creatinine and Blood Urea Nitrogen (BUN).

(e) Urine enzyme level - Alkaline Phosphatase (ALP)

(f) Urine analysis for urine casts and crystals

3.1.5 Phase Five

The data obtained by analysing the field samples and biological samples from cattle of Eloor industrial area were compared with that of cattle reared under ideal management conditions at University Livestock Farm (ULF), Mannuthy.

3.2 Details of analysis

3.2.1 Analysis of mercury

The collected field samples and biological samples were digested using a special digestion apparatus (Fig.3) as specified in "Official methods of analysis of Association of Official Analytical Chemists" (AOAC), (1980).

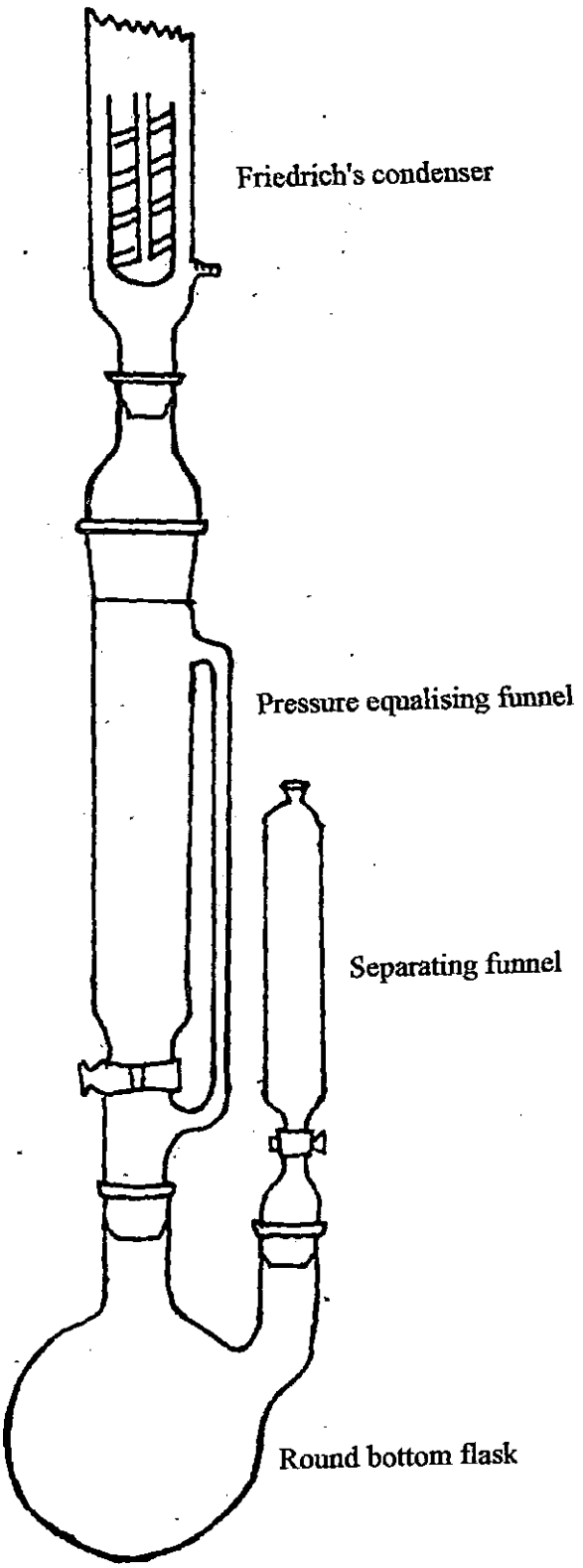
Digestion process was carried out in following steps

a) One gram of dried sample was placed in digestion flask with five or six glass beads.

b) Five ml of concentrated Nitric acid was added through dropping funnel.

c) The stop cock of soxhlet unit was adjusted to reflux position and flask was heated. Refluxing was allowed for 30 minutes.

Fig. 3. Special digestion apparatus for Mercury estimation



d) Five ml of cold Nitric acid - sulphuric acid (1:1) mixture was added and heated.

e) Stop cock was adjusted to trapping position to trap acid and water. Trapped acid and water were drained to the digest.

f) Five ml of 40 per cent w/v urea solution was added and further refluxed for 15 minutes.

The reaction flask was cooled and the digest was filtered. The filtrate was made up to 100 ml with distilled water.

Mercury level in the digested samples were analysed using mercury analyser MA 5800B (Fig. 4), a cold vapour atomic absorption spectrophotometer supplied by Electronic Corporation of India Limited (ECIL), Hyderabad. The procedures as per the instruction manual of MA 5800 B were carried out to assess mercury levels in samples.

3.2.2 Haematological parameters

Haematological parameters were studied for blood samples collected using disodium EDTA as anticoagulant.

3.2.2.1 Total erythrocyte (RBC) count

Total RBC were counted by using haemocytometer (Benjamin, 1978).

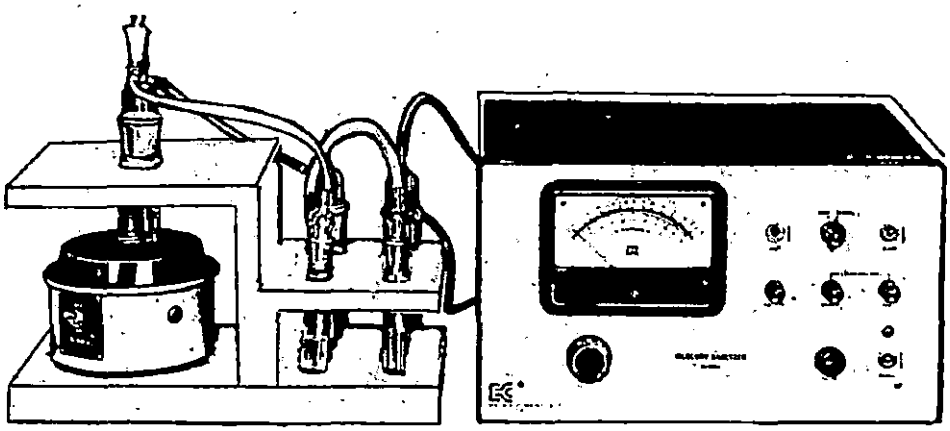
3.2.2.2 Packed Cell Volume (PCV)

Packed cell volume was estimated by filling Wintrobe haematocrit tubes using spinal needle. The tubes were centrifuged at a speed of 6000 rpm for 15 minutes (Wintrobe *et al.*, 1981).

3.2.2.3 Haemoglobin per cent

Haemoglobin concentration was estimated by acid haematin method (Benjamin, 1985).

Fig. 4. Mercury Analyser



3.2.2.4 Total Leucocyte (WBC) Count

The total WBC were counted by standard dilution technique using Thomas fluid and haemocytometer (Benjamin, 1978).

3.2.2.5 Differential Leucocyte Count (DLC)

Blood smears were prepared from freshly drawn blood (without anticoagulant) by using slide method. After staining with Wright's stain, differential leucocyte count was done by counting and classifying 200 leucocytes under oil immersion (Benjamin, 1978).

3.2.2.6 Haemoglobin indices

Mean Corpuscular Volume (MCV), Mean Corpuscular Haemoglobin (MCH), Mean Corpuscular Haemoglobin Concentration (MCHC) were calculated using the following formulas.

$$\text{MCV in } \mu m^3 = \frac{\text{PCV} \times 10}{\text{No. of erythrocytes per ml of blood} \times 10^{-6}}$$

$$\text{MCH in } \mu \text{ mg or pg} = \frac{\text{Hb in (g/dl)} \times 10}{\text{No. of erythrocytes per ml of blood} \times 10^{-6}}$$

$$\text{MCHC in g \%} = \frac{\text{Hb in (g/dl)} \times 100}{\text{PCV in (ml/dl)}}$$

3.2.3 Other parameter analysis

The evaluation of serum total proteins (albumin and globulin), serum creatinine, Blood Urea Nitrogen (BUN), serum alkaline phosphatase, serum ALAT, ASAT and urine alkaline phosphatase were carried out using a semi automatic blood analyser ("Microlab 200"). Ecoline® kits manufactued by E. Merck (India) Limited, MIDL, Taloja were used for estimation of respective items.

3.2.4 Urinalysis for Urinary casts and crystals

Urinalysis for casts and crystals were carried out as per the method cited by Benjamin (1985).

3.2.5 Statistical analysis

The data were analysed statistically by Completely Randomised Design (CRD) method (Snedecor and Cochran, 1980).

RESULTS

4. RESULTS

The data of the study conducted are presented in the Tables 1 to 6.

4.1 Cattle population and Disease incidence

Ward-wise survey of cattle population showed the presence of about 216 number of cattles in Eloor panchayat area (Table 1). Out of these, 121 adult animals above 3 years of age were considered for the study.

The interview conducted with farmers of Eloor panchayat revealed that almost all farmers in the area are facing problems in maintaining the livestock. The occurrence of gastrointestinal disorders was the most frequently reported problem in cattle fed with the grass or green fodders from the area and also due to the drinking water from certain localities in the study area. Infertility problem was also widely noticed in the local animals. In some localities (INDALCO Premises) dental caries and frequent bone related problems were also reported.

The case sheets of the Eloor panchayat Veterinary hospital for the past five years (1994-1999), were subjected to a thorough screening. A total of 455 case sheets were screened and the percentage incidence of diseases are presented in Table 2.

The occurrence of gastrointestinal disorders were 53.4 per cent followed by reproductive disorders including udder related problems, lameness and joint related problems, skin disorders, respiratory problems, and other diseases in the order of 18.7%, 10.9%, 10.5%, 4.8% and 1.7% respectively.

4.2 Mercury levels in field and biological samples

Mean mercury levels in the field and biological samples collected from Eloor East, North and South areas are presented along with that of control samples collected at University Livestock Farm (ULF), Mannuthy in Table 3.

Table 1. Cattle population in Eloor panchayat

Ward number	Number of cattle
1	31
2	27
3	33
4, 5, 6, 7 and 8	53
9, 13	39
10, 11, 12	33
Total	216

Table 2. Incidence of diseases in cattle of Eloor panchayat

Disease	Per cent of incidence
Gastro intestinal disorders	53.4
Reproductive disorders (including udder related problems)	18.7
Lameness and joint related problems	10.9
Skin disorders	10.5
Respiratory problems	4.8
Other diseases	1.7

Table 3. Mercury levels in field and biological samples of Eloor industrial area and University Livestock Farm (control), Mannuthy (Mean ± SE)

Sl. No.	Samples	Mercury levels in ppm			
		Control (n =10)	Eloor East	Eloor North	Eloor South
1	Sludges	-	0.24	0.34	0.26
2	Fodders	0.016±0.006[a]	0.113±0.024[b]	0.168±0.015[c]	0.094±0.019[b]
3	Water	0.00	0.00	0.00	0.00
4	Blood	0.010±0.003	0.026±0.009	0.027±0.003	0.023±0.009
5	Dung	0.014±0.006[a]	0.086±0.024[b]	0.081±0.018[c]	0.124±0.021[b]
6	Milk	0.006±0.003	0.020±0.004	0.014±0.004	0.027±0.011
7	Urine	0.012±0.000[a]	0.007±0.004[b]	0.035±0.000[c]	0.025±0.012[d]

Mean bearing different superscripts in a row differ significantly

4.2.1 Biological samples

4.2.1.1 Blood

The mean mercury levels in blood of cattle maintained at Eloor East, North and South were 0.026 ± 0.009 ppm, 0.027 ± 0.003 ppm and 0.023 ± 0.009 ppm respectively. These levels were comparatively higher than that of control samples (0.010 ± 0.003 ppm). But statistical analysis proved that there was no significant difference between the control and other groups.

4.2.1.2 Dung

The mean mercury level in dung samples from Eloor East, North and South were 0.086±0.024 ppm, 0.081 ± 0.018 ppm and 0.124 ± 0.021 ppm respectively. These levels were significantly ($P<0.01$) higher than that of control samples (0.014 ± 0.006 ppm). However, there was no significant difference between the mercury levels of samples collected from the three areas of Eloor.

4.2.1.3 Milk

The mean mercury levels of milk samples collected from Eloor East, North and South were 0.020 ± 0.004 ppm, 0.014 ± 0.004 ppm and 0.027 ± 0.011 ppm respectively. These values were comparatively higher than that of controls (0.006 ± 0.003 ppm). But statistical analysis showed no significant difference between and within these values.

4.2.1.4 Urine

The mean mercury levels of urine samples collected from Eloor East, North and South were 0.007 ± 0.004 ppm, 0.035 ± 0.0 ppm and 0.025 ± 0.012 ppm respectively. These values were higher than that of control values (0.012 ± 0.0 ppm). Statistical analysis showed that there was significant difference ($P<0.01$) between control and samples from three Eloor areas and also within samples.

4.2.2 Field samples

4.2.2.1 Fodders

The mean mercury level of fodder samples collected from Eloor North (0.168 ± 0.015 ppm) was the highest, followed by Eloor East (0.113 ± 0.024 ppm) and Eloor South (0.094 ± 0.019 ppm). These values were very high when compared with that of controls (0.016 ± 0.006 ppm). Statistical analysis showed that there was significant difference ($P<0.01$) between control and samples from industrial areas. Significant difference ($P<0.01$) was also noticed between Eloor East and Eloor North samples.

4.2.2.2 Water

The water samples collected from the ULF, Mannuthy and Eloor areas showed no detectable levels of mercury.

4.2.2.3 Sludges

There was no sludge in the ULF, Mannuthy, so no control sample was collected. The sludge collected from Eloor East, North and South areas showed mean mercury levels of 0.24 ppm, 0.34 ppm and 0.26 ppm respectively.

4.3 Haematological parameters

The results of the haematological parameters are presented in the Table 4.

4.3.1 Red Blood cell (RBC) count

Mean RBC count (millions/mm^3) of the control, Eloor East, North and South samples were 9.44 ± 0.43, 5.897 ± 0.35, 6.45 ± 0.27 and 5.70 ± 0.06 respectively. The RBC counts of field samples were significantly lower ($P<0.01$) than that of controls, without significant difference in values among field samples.

Table 4. Haematological parameters of cattle in Eloor industrial area and University Livestock Farm (control), Mannuthy (Mean ± SE)

Sl. No.	Parameters	Control (n=10)	Eloor East	Eloor North	Eloor South
1	RBC (millions/mm^3)	9.44±0.43^{a}	5.89±0.35^{b}	6.45±0.27^{b}	5.70±0.06^{b}
2	WBC (numbers/mm^3)	10540±706.7	9403±401.4	9391±173.3	9434±155.8
3	PCV (%)	38.7±1.08^{a}	35.31±2.14^{ab}	43.46±1.63^{ac}	32.77±2.01^{ab}
4	Hb (g %)	9.6±0.25^{a}	8.6±0.31^{a}	11.1±0.41^{b}	8.9±0.22^{a}
5	Differential Leucocyte count (%) Neutrophil	36±0.99^{a}	30.1±1.77^{b}	32.3±1.08^{b}	29.2±0.48^{b}
6	Lymphocytes	60.1±0.94^{a}	66.5±2.26^{b}	65.9±1.32^{b}	68.8±0.43^{b}
7	Eosinophils	2.2±0.25^{a}	2.9±0.80^{ab}	1.3±0.29^{ac}	1.1±0.28^{ac}
8	Monocytes	0.7±0.15	0.5±0.18	0.5±0.14	0.6±0.18
9	Basophils	0.00	0.2±0.1	0.00	0.2±0.1
10	MCV (μm^3)	39.5±2.45^{a}	59.9±1.31^{b}	68.9±2.83^{c}	57.7±3.69^{b}
11	MCH (pg)	10.4±0.54^{a}	14.9±0.54^{b}	17.5±0.68^{c}	16.0±0.37^{bc}
12	MCHC (g %)	24.9±0.29	24.9±0.98	25.9±1.05	28.3±2.12

Mean bearing different superscripts in a row differ significantly

4.3.2 White Blood Cell (WBC) count

The mean WBC count (numbers/cu.mm) of the blood samples collected from Eloor East, North and South were 9403 ± 401.4, 9391 ± 173.3 and 9434 ± 155.8 respectively. They were comparatively less than that of control samples (10,540 ± 706.7). But there was no significant difference between WBC counts of control and field samples.

4.3.3 Packed cell volume (PCV)

The mean PCV (%) of the control samples was 38.7 ± 1.08 which showed no statistical difference when compared with that of Eloor East (35.31 ± 2.14), North (43.46 ± 1.63) and South (32.77 ± 2.01). There was significant difference ($P<0.01$) between mean PCV values of Eloor East and North samples.

4.3.4 Haemoglobin (Hb) concentration

The average Hb concentration (g%) of control, Eloor East, North and South samples were 9.6 ± 0.25, 8.6 ± 0.31, 11.1 ± 0.41 and 8.9 ± 0.22 respectively. The mean Hb value of samples from Eloor North was significantly ($P<0.01$) higher than that of the controls as well as those of Eloor East and South.

4.3.5 Differential Leucocyte Count (DLC)

The mean neutrophil count of blood from control animals (36 ± 0.99%) was significantly ($P<0.01$) higher than those of Eloor East (30.1 ± 1.77%), North (32.3 ± 1.08%) and South (29.2 ± 0.48%). There was no significant difference among the three areas of study.

The mean lymphocytic count of Eloor East (66.5 ± 2.26%), North (65.9 ± 1.32%) and South (68.8 ± 0.43%) were significantly ($P<0.01$) higher than those of controls (60.1 ± 0.94%). But among the three areas of Eloor, there was no significant difference.

The mean Eosinophil count of Eloor East, North and South were 2.9 ± 0.80%, 1.3 ± 0.29% and 1.1 ± 0.28% respectively. There was no significant difference of these values with that of controls (2.2 ± 0.25%). But Eloor East differ significantly ($P<0.05$) with those of Eloor North and South.

The mean monocyte count values of control, Eloor East, North and South were 0.7 ± 0.15%, 0.5 ± 0.18%, 0.5 ± 0.14% and 0.6 ± 0.18% respectively. There was no significant difference among these values.

In the control samples and Eloor North samples, no basophils could be seen but Eloor East and South samples had 0.2 ± 0.1% and 0.2 ± 0.1% respectively as their mean basophil count.

4.3.6 Erythrocyte indices

The Mean Corpuscular Volume (MCV) values (μm^3) of samples from Eloor East (59.9 ± 1.31), North (68.9 ± 2.83) and South (57.7 ± 3.69) were significantly ($P<0.01$) higher than that of control samples (39.5 ± 2.45). There was significant ($P<0.01$) increase in mean MCV of Eloor North samples when compared with Eloor East and South samples.

The Mean Corpuscular Haemoglobin (MCH) values (pg) of control, Eloor East, North and South samples were 10.4 ± 0.54, 14.9 ± 0.54, 17.5 ± 0.68 and 16.0 ± 0.37 respectively. Statistical comparison revealed that MCH values of Eloor samples were significantly ($P<0.01$) higher than that of control samples. Significant ($P<0.01$) difference was also noticed between samples of Eloor East and North areas.

The Mean Corpuscular Haemoglobin Concentration (MCHC) values (g%) of control samples (24.9 ± 0.29), Eloor East (24.9 ± 0.98), North (25.9 ± 1.05) and South (28.3 ± 2.12) had no significant difference among them.

4.4 Serum biochemical parameters

The estimated values of serum total proteins, albumin, globulin, creatinine and BUN are presented in Table 5.

4.4.1 Protein levels

The mean total protein values (g%) of serum samples collected from Eloor East, North and South areas were 8.7 ± 0.94, 10.9 ± 0.79 and 12.4 ± 1.01 respectively. There was no significant difference with that of control samples (11.1 ± 0.67). However, significant ($P<0.05$) difference was noticed between Eloor East and South samples.

The mean albumin values (g%) of control, Eloor East, North and South samples were 4.4 ± 0.37, 4.9 ± 0.35, 4.2 ± 0.31 and 4.9 ± 0.33 respectively. The Eloor samples had neither significant difference with control samples nor within themselves.

The mean globulin values (g%) of serum from Eloor East, North and South were 3.8 ± 0.78, 6.6 ± 0.51 and 7.5 ± 0.90 respectively. Among these, Eloor East samples showed significant ($P<0.01$) lower values than that of control samples (6.7 ± 0.72). Within the field samples, Eloor East samples showed significant ($P<0.01$) decrease than the other two areas.

4.4.2 Creatinine level

The mean creatinine values (mg%) of the control, Eloor East, North and South samples were 0.97 ± 0.16, 1.23 ± 0.23, 1.33 ± 0.10 and 1.1 ± 0.14 respectively. There was no significant difference among these values.

4.4.3 Blood Urea Nitrogen (BUN) level

The mean BUN value (mg%) of control samples (29.9 ± 1.94) was significantly ($P<0.01$) higher than that of Eloor East (17.5 ± 1.80) and South (16.0

± 2.69). There was no significant difference between the control and Eloor North (22.7 ± 2.89) samples. No significant difference was seen among samples from three Eloor areas.

4.5 Serum and urine enzyme levels

The mean serum Alkaline Phosphatase (ALP), serum Alanine Amino Transferase (ALAT), serum Aspartate Amino Transferase (ASAT) and urine Alkaline Phosphatase (ALP) levels (u/l) are presented in Table 6.

4.5.1 Serum Alkaline Phosphatase (ALP) levels

The mean serum Alkaline Phosphatase (ALP) levels (u/l) of control, Eloor East, North and South samples were 121.4 ± 17.70, 163.7 ± 45.6, 170.5 ± 37.45 and 208.5 ± 35.98 respectively. Though the values of field samples were comparatively higher than that of controls, there was no significant difference between and among the samples.

4.5.2 Serum Alanine Amino Transferase (ALAT) levels

The mean serum Alanine Amino Transferase (ALAT) values (u/l) of control, Eloor East, North and South samples were 16.1 ± 1.13, 28.3 ± 4.43, 23.9 ± 4.88 and 25.2 ± 2.44 respectively. There was no significant difference between control and field samples or within the field samples.

4.5.3 Serum Aspartate Amino Transferase (ASAT) levels

The mean Aspartate Amino Transferase (ASAT) values (u/l) of control, Eloor East, North and South samples were 21.5 ± 1.49, 91.5 ± 21.83, 75.6 ± 8.04, and 71.5 ± 5.88 respectively. There was significant ($P<0.01$) increase in the values of Eloor samples when compared to that of controls. There was no significant difference among the samples from the three Eloor areas.

Table 5. Biochemical composition of serum of cattle in Eloor industrial area and University Livestock Farm (control), Mannuthy (Mean ± SE)

Sl. No.	Parameters	Control (n=10)	Eloor East	Eloor North	Eloor South
1	Total protein (g %)	11.11±0.67 [a]	8.70±0.94 [ab]	10.90±0.79 [a]	12.40±1.01 [ac]
2	Albumin (g %)	4.4±0.37	4.9±0.35	4.2±0.31	4.9±0.33
3	Globulin (g %)	6.7±0.72 [a]	3.8±0.78 [b]	6.6±0.51 [a]	7.5±0.90 [a]
4	Creatinine (mg %)	0.97±0.16	1.23±0.23	1.33±0.10	1.1±0.14
5	BUN (mg %)	29.9±1.94 [a]	17.5±1.80 [b]	22.7±2.89 [ab]	16.0±2.69 [b]

Mean bearing different superscripts in a row differ significantly

Table 6. Enzyme levels in blood serum and urine of cattle in Eloor industrial area and University Livestock Farm (control), Mannuthy (Mean ± SE)

Sl. No.	Enzymes (u/l)	Control (n=10)	Eloor East	Eloor North	Eloor South
1	Serum alkaline phosphatase (ALP)	121.4±17.70	163.7±45.6	170.5±37.45	208.5±35.98
2	Serum Alanine Amino Transferase (ALAT)	16.1±1.13	28.3±4.43	23.9±4.88	25.2±2.44
3	Serum Aspartate Amino Transferase (ASAT)	21.5±1.49 [a]	91.5±21.83 [b]	75.6±8.04 [ba]	71.5±5.88 [b]
4	Urine alkaline Phosphatase (ALP)	8.3±0.2	14±0.0	11.7±5.14	16±3.0

Mean bearing different superscripts in a row differ significantly

4.5.4 Urine Alkaline Phosphatase (ALP) levels

The mean urine Alkaline Phosphatase (ALP) values (u/l) of control, Eloor East, North and South samples were 8.3 ± 0.2, 14 ± 0.0, 11.7 ± 5.14 and 16 ± 3.0 respectively. There was a comparative increase in the values of Eloor samples than that of control but the increase was non-significant.

4.6 Urinalysis

Urinalysis showed no casts or crystals in samples collected from controls and animals from Eloor areas.

DISCUSSION

5. DISCUSSION

5.1 Cattle population and Disease Incidence

Ward-wise survey of cattle population was done and that accounted to about 216 cattle in Eloor panchayat area. The following were the main plight of farmers in maintaining livestock.

1. The most frequent problem bothering the farmers was the gastrointestinal disorders. In certain localities of the study area (TCC and HIL premises) cattle, fed with grass or green fodders, were reported to suffer from diarrhoea frequently.
2. Infertility problem was also widely noticed in local animals.
3. Around INDALCO premises, cattle were having dental caries and were also reported to suffer from bone and joint associated problems.

The above reports were confirmed when the case sheets of the Eloor panchayat area from 1994 to 1999 (last five years) were screened. It was revealed that incidence of the gastrointestinal disorders was 53.4% and that of the reproductive disorders was 18.7% in cattle of Eloor area.

Short and Edwards (1988) reported the occurrence of gastrointestinal mucosal ulcers, gastroenteritis, diarrhoea or steatorrhea in acute inorganic mercury poisoning in horse. The chronic signs of mercury poisoning observed by them were anorexia, stomatitis, gastroenteritis, weight loss, dullness, progressive renal disease (nephritis) and alopecia. On experimental mercuric chloride intoxication in goats, Kumar and Pandey (1993) noticed diarrhoea as a symptom of mercury toxicity. They opined that it could be due to the entry of mercury through the mucosa of the gastrointestinal tract causing coagulative necrosis of epithelium.

Depression, diarrhoea, weakness and loss of body weight were the symptoms exhibited in cattle with induced nephrosis using mercuric chloride (Parai *et al.*, 1993).

The common form of mercury toxicity was chronic mercurialism where small amounts of mercury were ingested over long periods. There was depression, anorexia, emaciation and a stiff, stilted gait, alopecia, scabby lesions around the anus and vulva, pruritus, petechiation, tenderness of gums and shedding of teeth which were accompanied by chronic diarrhoea, weakness, incoordination and convulsions (Radostits *et al.*, 1994).

In the light of above observations, increased incidence of gastrointestinal disorders, bone and teeth problems, infertility problems and presence of seven major industries suspected to cause mercury contamination were suggestive of mercury as one of the possible etiological factors causing diseases in cattle of Eloor area. To explore this in detail, mercury level in the field and biological samples were analysed.

5.2 Mercury levels in field and biological samples

Mercury levels in all the samples collected from the study area as field samples (sludges, fodders and water samples) and biological samples (dung, blood, milk and urine) were very high when compared to the control samples collected from University Livestock Farm (ULF), Mannuthy. This indicated that mercury eliminated from the industries in trace amounts will be accumulated in water, soil, sediments or plants and animals.

5.2.1 Sludges

The highest mercury level was found in sludges collected from Eloor North (0.34 ppm) followed by South (0.26 ppm) and then East (0.24 ppm). These values were higher than the mercury level observed by Cheeran *et al.* (1987) in sludges of Eloor i.e. 0.14 ppm. The general standards for discharge of effluents permit only 0.01 ppm of mercury at a maximum (Trivedy, 1996). In Eloor North area, Indian Rare Earths (IRE) and Hindustan Insecticides Limited (HIL) were the two main industries present. Out of these two, HIL was considered to be the main

source of mercury contamination, since the company uses chlorinated organic compounds as a basic chemical for producing insecticides. Mercury is a pollutant of chlorinated compounds.

In Eloor South area, Travancore Chemicals and Manufacturing Company (TCM) was the only company present and since it produces copper oxychloride and potassium chlorate, mercury is considered to be discharged in traces from this company.

In Eloor East, Fertilizer and Chemicals Travancore (FACT), Travancore Cochin Chemicals (TCC) and Indian Aluminium Company (INDALCO) were the three industries present. Among these, FACT and TCC were the possible sources of mercury contamination in this area. TCC manufactured caustic soda, liquid chlorine and hydrochloric acid. The cathode used in electrolysis process of manufacturing caustic soda contained mercury and as a result mercury in traces may be released as effluents. FACT deals with fertilizers where mercury is a pollutant.

5.2.2 Fodders

In fodders too, samples from Eloor North possessed highest mercury level (0.168 ± 0.015 ppm) followed by Eloor East (0.113 ± 0.024 ppm) and Eloor South (0.094 ± 0.019 ppm), when compared to the mercury level of controls (0.016 ± 0.006 ppm). The contribution of mercury to the fodders by the industrial effluents can be clearly understood by the significantly increased mercury level in Eloor samples when compared to the controls.

The mercury levels of fodders agree with the observation of Cheeran *et al.*, 1987 (0.02 - 0.19 ppm).

Toxicosis may occur in cattle on an average daily intake of mercury @ 10 mg/kg per day (Radostits *et al.*, 1994). Boyd (1985) observed that the estimated

ingestion of 0.275 mg/kg in cattle was below that required to induce renal failure and also found certain animals succumbed while others, on the same diet, remained apparently unaffected. The mean mercury levels found in fodder samples from Eloor industrial area were too less to induce renal toxicity in cattle and would explain why there was no distinct specific symptoms of mercury toxicity. So this level of toxicity could be equated to sub chronic type of toxicity. But since mercury is a bioaccumulative heavy metal, it may pose dangerous effects in the same animals later or even cause toxicity in human or carnivorous animals eating the meat of these cattle.

5.2.3 Water samples

Samples of water collected from Eloor areas and control area had no detectable level of mercury in them. This corroborates the finding of Cheeran *et al.* (1987). The reasons for this non-detectable level of mercury may be

1) Most of the industries emit only inorganic salts of mercury as effluents.
2) Inorganic mercury is highly unstable in sediments and could be transformed by anaerobic microorganism in the water column to methyl mercury, which gets bioaccumulated at successive trophic levels in food chain (Wolfe *et al.*, 1998).
3) Methyl mercury, highly lipid-soluble compound, would be identified only in sediments, plants or fishes and not in clean, well water. This proves that fodders were the main source of mercury to cattle and is further confirmed by increased mercury levels in dung samples. It was this high amount of mercury in fodders that contribute to higher mercury levels in dung, blood, milk and urine of cattle.

5.2.4 Dung samples

In Dung samples examined, Eloor South had the highest amount of mercury (0.124 ± 0.021 ppm) followed by Eloor East (0.086 ± 0.024 ppm) and then Eloor North (0.081 ± 0.018 ppm). These values were in accordance with the

findings of Cheeran *et al.* (1987) who found that mercury level of dung samples was in the range of 0.020 to 0.190 ppm.

High level of mercury in dung samples of Eloor industrial area substantiated that only a small amount of mercury is absorbed through the gastrointestinal tract of cattle. Further, the dung samples may bioaccumulate mercury in soil and plants showing the persistant nature of mercury in the environment.

5.2.5 Blood samples

Eloor North area had the highest blood mercury level of 0.027 ± 0.003 ppm followed by Eloor East (0.026 ± 0.009 ppm) and then by Eloor South (0.023 ± 0.009 ppm). These values were highly in consonance with the mean mercury level of blood samples (0.027 ppm) recorded by Cheeran *et al.* (1987). The concentration of mercury in blood had been used as a biologic indicator of exposure (Hammond and Beliles, 1980). Klaassen (1996) reported that the upper limit of a nontoxic concentration of mercury in blood of human was generally considered to be 3 to 4 μg/dl (0.03-0.04 ppm).

The mean mercury levels of blood samples collected from Eloor area were less to produce any prominent toxic symptoms but these trace levels were liable to cause disease along with other etiologic factors.

5.2.6 Milk samples

Milk samples of Eloor South area contained the highest mercury level of 0.027 ± 0.011 ppm when compared to Eloor East (0.020 ± 0.004 ppm) and Eloor North (0.014 ± 0.004 ppm) areas. These values were in high concordance with value range of 0 to 0.042 ppm found by Cheeran *et al.* (1987) and a value range of 0.004 to 0.30 ppm recorded by Gajewska *et al.* (1994). Limited data regarding ruminants indicated that only traces of mercury either in the organic or inorganic form was secreted into the milk.

5.2.7 Urine samples

Eloor north area urine samples contained the highest mercury level of 0.035 ± 0.000 ppm followed by Eloor South (0.025 ± 0.012 ppm) and then Eloor East (0.007 ± 0.004 ppm). These values were slightly higher than that (0.014 ppm) observed by Cheeran *et al.* (1987). The concentration of mercury in urine had been used as a measure of the body burden of the metal. The upper limit for excretion of mercury into the urine in the normal population (Human) is 5 µg/liter (Klaassen, 1996). In the present study, the mercury levels of the urine samples were higher than that specified by Klaassen (1996) which proved that a subchronic toxicity is existing among the cattle of Eloor industrial area.

By assessing the mean mercury levels in field and biological samples of Eloor industrial area, the following were the conclusions arrived at:

(1) No difference among the three areas of Eloor could be established based on the level of exposure to Hg. HIL in Eloor North area, TCM in Eloor South area, FACT and TCC in Eloor East area were the companies suspected to be the probable source of mercury to sludges collected from the respective areas.

(2) Fodders were the main source of mercury to adult cattle at Eloor, since water contains no detectable level of mercury.

(3) Mercury could be detected at higher levels than normal in the blood, dung, urine and milk of Eloor cattle, but these higher levels were not enough to cause toxicity symptoms in cattle. They could only produce additive toxic effect with other factors. So this could be considered as a subchronic mercury toxicity.

(4) The non-toxic levels of mercury recorded in the present study does not rule out toxicity. Since mercury is a Persistent, Bioaccumulative and Toxic (PBT) heavy metal, its biomagnification property possess an enormous risk of toxicity in future.

5.3 Haematological parameters

5.3.1 Erthrocytes (RBC) count

In the present study the number of RBC in the cattle of Eloor industrial area was significantly less than that of the controls, but the values lie close to the normal range of 6-9 millions/mm^3 (Swenson, 1996).

This was not in agreement with the findings of Parai and Pandey (1992) who found that there was a significant increase in total erythrocyte count in cattle experimentally induced nephrotoxicity using mercuric chloride.

5.3.2 White Blood Cells (WBC) count

There was no significant difference in the WBC count among control and samples from Eloor area. The Eloor samples showed leucopenia but the values were within the normal range of 7,000-10,000 numbers/mm^3 (Swenson, 1996). This was not in accordance with observations of Kumar and Pandey (1993) who observed significant leucocytosis in mercuric chloride induced nephrosis in goats.

5.3.3 Packed Cell Volume (PCV)

Packed cell volume of Eloor cattle were less than that of controls, except Eloor North samples but the values were within the normal range of 33-47 per cent (Benjamin, 1985). This finding was in discordance with Parai and Pandey (1992), who found significantly increased PCV values in cattle having induced nephrotoxicity with mercuric chloride.

5.3.4 Haemoglobin (Hb) concentration

Haemoglobin values of Eloor cattle showed no significant difference between them and control samples except Eloor North which showed significant increase than the control samples. This coincides with the finding of Kumar and Pandey (1993), that Hb remained largely unaltered except for a mild elevation.

5.3.5 Differential Leucocyte Count (DLC)

In the present study, there was significant neutropenia and lymphocytosis in the cattle of Eloor industrial area, when compared with controls. However, the values remained within the normal range specified by Swenson (1996). These finding were not in agreement with Kumar and Pandey (1993), who observed neutrophilia, monocytosis and corresponding lymphopenia in nephrotoxic goats intoxicated experimentally with mercuric chloride.

5.3.6 Erythrocyte indices

In the present study, the values for Mean Corpuscular Volume (MCV), Mean Corpuscular Haemoglobin (MCH) and Mean Corpuscular Haemoglobin Concentration (MCHC) were significantly higher in the Eloor samples than the control blood samples but the values of Eloor sample were close to the normal value range specified by Swenson (1996).

The following may be attributed to be the probable explanation for the dissidence in haematological values of the present study with that of previous works related to it.

(1) The very fact that the present study was a field study involving many factors may be the reason for the discrepancy in values, when compared with other studies which were experimental studies specifically done with mercuric chloride alone.

(2) The prevailing condition in the present study may be equated to a subchronic mercury toxicity unlike other studies which dealt with acute or chronic mercury toxicities.

(3) In the present study, mercury alone cannot be considered to be a pollutant, since so many chemicals were also expected to be released from industries into the effluents. The other chemicals might have also influenced the haematological values.

It could be inferred from the present study that these haematological parameters were not indicative of mercury toxicity alone, but a complex of chemicals are involved in it.

5.4 Serum biochemical parameters

In the present study similar total protein values were observed among Eloor and control samples which corroborates well with Kumar and Pandey (1998), who experimentally induced mercuric chloride toxicity in goats and found that serum total protein values were not influenced by mercury toxicity. Albumin and globulin levels too showed no significant difference between Eloor and control samples. Creatinine levels were very well within the normal range of 1-2 mg/dl (Swenson, 1996). The BUN values were also within the normal range of 10-30 mg/dl observed by Swenson (1996). But the observations made in an experimental mercuric chloride intoxication in goats by Kumar and Pandey (1998) were inconsistent to the present finding. They found marked increase of BUN and creatinine values.

The reasons for the normal values of the proteins, creatinine and BUN in the study samples may be explained as:

(1) Even though, the mercury level in blood samples of Eloor area were more than that of control samples, the levels were not to the extent of causing symptomatic toxicity and marked biochemical changes (subchronic toxicity).

(2) In case of experimental acute toxicity study conducted by Kumar and Pandey (1998), severe renal insufficiency was noticed which was indicated by increased serum creatinine and BUN levels. But in the present study there was no increase in creatinine and BUN values indicating the absence of renal insufficiency in cattle of Eloor industrial area.

5.5 Serum and urine enzymes level

In the present study, no significant change in serum alkaline phosphatase activity was observed although there was an increasing trend and the levels were within the normal range (2 to 809 u/l) in accordance with Kaneko *et al.* (1999).

This was in accordance with Kumar and Pandey (1994) who also observed no change in serum Alkaline Phosphatase (ALP) activity though, an increasing trend was apparent beyond 21 days in goats induced with mercuric chloride nephropathy. Parai and Pandey (1992) also noticed increasing trend till 14^{th} day followed by maintenance of already increased values on 17^{th} to 20^{th} day in cattle with induced nephrotoxicity using mercuric chloride.

The urinary alkaline phosphatase levels in the present study showed no significant difference between the Eloor samples and control samples though, a slight increase in values were noted. This finding was not in agreement with the findings of Parai and Pandey (1992) and Kumar and Pandey (1994). They found markedly elevated activity of urinary alkaline phosphatase which could be attributed to excess release of enzyme in urine by damaged renal tubules. However, Parai and Pandey (1992) opined that urinary alkaline phosphatase could be a good indicator of renal insufficiencies in ruminants. From the present observations of urine alkaline phosphatase levels it could be inferred that there may be no marked renal tubular damage in the Eloor cattle. The serum Alanine Amino Transferase (ALAT) and Aspartate Amino Transferase (ASAT) levels of the Eloor samples, though had higher values than control samples, were within the normal range (ALAT - 11 to 40 u/l, ASAT - 78 to 132 u/l) observed by Kaneko *et al.* (1999).

From the present study, it could be inferred that serum and urine enzyme levels were well within normal range, even though the values were more

towards the upper limits of normal values, which may be due to mild renal tubular damage.

5.6 Urinalysis

Analysis of urine showed no casts and crystals in Eloor and control samples. In case of extensive tubular damage, presence of granular and epithelial casts in urine were reported by Robinson and Hesketh (1976). This again confirmed the observation that there may be only mild renal tubular damage in Eloor cattle.

From the present study following were the conclusions that could be arrived at:

(1) Mercury was detected at higher levels than normal in blood, dung, urine and milk of Eloor cattle. No difference in level of mercury exposure among the three areas of Eloor could be established. Fodders may be the main source of mercury to adult cattle at Eloor industrial area.
HIL in Eloor North, TCM in South, FACT and TCC in East were suspected to be probable industries eliminating mercury through effluents. The higher mercury levels noticed in Eloor area were not enough to cause toxic symptoms in cattle.

(2) The haematological and serum biochemical parameters, serum and urine enzyme levels and urinalysis revealed no definite change in the cattle of control and Eloor area as that of experimental mercuric chloride toxicity.

(3) The present condition may be regarded as subchronic mercury toxicity. But this could not be considered safe because the mercury levels in all samples were found to be above the maximum limit specified by standard i.e. 0.01 ppm (Trivedy, 1996). Mercury is also a persistent, bioaccumulative toxic heavy metal. If left unattended, this may lead to a major outbreak in future. So this study may be considered to be a caution to the industries concerned, the public and veterinarians to take appropriate remedial measures to prevent further environmental contamination with mercury.

SUMMARY

6. SUMMARY

A study was conducted to assess the extent of mercury pollution by industries at Eloor Industrial area, Kerala.

Initially ward-wise survey of cattle population and interview of the farmers to appraise the common problems faced by them to raise cattle, were carried out. There were about 121 adult cattle in Eloor industrial area which were considered for the study. Gastrointestinal disorders, Reproductive disorders, Lameness and joint related problems were reported to be the most frequent problems in cattle of Eloor area.

The incidencesof diseases of cattle in Eloor panchayat were analysed by thorough screening of the case sheets for the last five years duration (1994-1999). A total of about 455 case sheets were analysed and found that Gastrointestinal disorders were the major problem seen in 53.4% cases followed by reproductive disorders (18.7%) and then, the bone and joint related problems (10.9%).

Eloor industrial area was divided into Eloor East, North and South for the purpose of study. Field samples and Biological samples of cattle were collected from the study areas. Field samples included sludge, water and fodder samples. Biological samples included Blood, urine, dung and milk collected from adult cattle at sampling sites.

In the laboratory, the collected samples were analysed for the mercury level, haematological and biochemical (Total protein level, creatinine and BUN level) parameters of serum, enzymes in serum (ALP, ASAT, ALAT) and urine (ALP) and then urinary casts. The data obtained by analysing the field and biological samples from cattle of Eloor area were compared with that of cattle reared under ideal managemental conditions at University Livestock Farm (ULF), Mannuthy.

A higher level of mercury could be detected in the blood, dung, urine and milk of Eloor cattle than control animals. Higher mercury levels in sludges and fodders, and below detectable level of mercury in water samples putforth fodders to be a probable mercury source to cattle of Eloor area.

The haematological parameters like RBC count, WBC count, PCV, Hb, differential leucocyte count, erythrocyte indices (MCV, MCH and MCHC) showed no similarity to that of experimental mercuric chloride toxicity. The role of other chemicals also were suspected in the changes of haematological parameters.

The total protein level, albumin, globulin, creatinine and BUN level were found to be within normal limits showing little changes. Even the serum and urine enzyme analysis showed only slight increase, that too within normal range. So it could be inferred from the present observation that mercury toxicity may be only in subchronic level. Urinalysis showed no casts or crystals.

From the study HIL, TCM, FACT and TCC were the industries suspected to be probable ones eliminating mercury through effluents. The mercury toxicity though suspected to be at a subchronic level may pose serious threats to human and live stock in future. The study revealed a note of caution to the industries concerned, public and veterinarians to take appropriate remedial measures, to curb further environmental pollution and to safeguard human and livestock health.

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ASSESSMENT OF MERCURY TOXICITY IN CATTLE OF ELOOR INDUSTRIAL AREA

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ABSTRACT OF THE THESIS

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ABSTRACT

A detailed study was conducted to assess the extent of mercury toxicity in cattle of Eloor industrial area. An initial ward-wise survey of cattle population and interview with farmers to hear the problems faced by them in raising cattle at Eloor industrial belt were carried out. It was learnt that a total of about 216 cattle were present and they experienced frequent gastrointestinal disorders and reproductive disorders along with bone and joint related problems. A detailed case sheet study for the past five years duration confirmed the same.

Eloor industrial belt was divided into Eloor East, North and South for the study purpose. Field samples like sludges, water and fodders and Biological samples like blood, dung, urine and milk of cattle in the study area were collected. The collected samples were analysed for mercury level in the laboratory, and compared statistically with those of the control samples collected from University Livestock Farm (ULF), Mannuthy.

Higher mercury levels were found in all the field and biological samples. Fodders were suspected to be the main source of mercury to cattle. The higher but nontoxic level of mercury in the samples could only produce subchronic level of toxicity with no definite toxicity symptoms. Mercury levels within the Eloor East, North and South showed no significant difference.

Further haematological parameters, serum protein levels, creatinine and BUN levels remained within normal limits with slight changes. The serum enzyme levels of Alkaline Phosphatase (ALP), Aspartate Amino Transferase (ASAT), Alanine Amino Transferase (ALAT) and urine Alkaline Phosphatase (ALP) levels showed increased levels but the levels were within normal range. These observations ruled out the absence of severe nephrotoxicity with the present levels of mercury.

Urine analysis showed no casts or crystals in them, again confirming the absence of any severe nephrosis in cattle of Eloor area. But higher than normal mercury levels in all samples may be looked with caution because of its persistent, bioaccumulative and toxic nature.