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STUDIES ON DDT AND ITS METABOLITES IN ENVIRONMENT, HUMAN TISSUES AND THEIR HEALTH EFFECTS

THESIS

Submitted to the University of Roorkee
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in

CENTRE OF BIOSCIENCES

By
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MAY 1985

CANDIDATE'S DECLARATION

I hereby certify that the work which is being presented in this thesis entitled "Studies on DDT and its metabolites in environment, human tissues and their health effects" in fulfilment of the requirement for the award of the Degree of Doctor of Philosophy in Biosciences, submitted in the Centre of Biosciences, University of Roorkee, Roorkee is an authentic record of my own work carried out during the period March, 1983 to May, 1985, under the supervision of Dr.C.B. Sharma and Dr. Jagdish Chandra.

The matter embodied in this thesis has not been submitted by me for the award of any other degree.

Charu
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This is to certify that the above statement made by the candidate is correct to the best of our knowledge.

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Dedicated to my parents

ABSTRACT

DDT residue in soil from 50 sites surrounding a DDT factory in Delhi were monitored. DDT was detected in all the samples. Total DDT ranged from 0.498 to 7.270 ppm with a mean value of 1.670 ppm. The highest concentration of total DDT, 7.270 ppm, was detected in the soil from Durga Nagar in the vicinity of a DDT factory. Other areas surrounding DDT factory contained moderate to high levels of DDT and its metabolites in soil. West Zone soils were heavily contaminated with DDT residues as compared to other zones of Delhi.

Blood samples from 50 occupationally unexposed volunteers of Delhi area were examined for DDT residues1 The total DDT concentration in the whole blood ranged from 0.112 to 0.663 ppm in males and 0.053 to 0.560 ppm in females. The DDT metabolites detected were : p,p'-DDE, o,p'-DDT, p,p'-DDT and very low quantities of p,p'-DDD. DDE accounted for most of the total DDT. Age was shown to have no effect on blood DDT levels but dietary habits influenced the accumulation of DDT in the blood; non-vegetarians had higher total DDT in the blood as compared to vegetarians ($p<0.05$).

Transfer of organochlorine pesticides from mother to fetus was studied in 50 women. The concentrations of DDT and its metabolites were examined in maternal blood, placental tissue and umbilical cord blood of the same mother/child pair. Residue levels of DDT and its metabolites were detected in all the samples analyzed indicating their transfer from mother to fetus via placenta. No effect of age and dietetic habits was found on accumulation of DDT and its metabolites in pregnant women.

The excretion of DDT and its metabolites in human milk was studied. The circulating blood and breast milk of nursing Indian mothers were examined for the presence of DDT and its metabolites. The mean total DDT in milk (2.030 ppm) was significantly higher than that of blood (0.253 ppm; $p<0.001$). The daily intake of total DDT residues by an Indian child weighing 5 kgs and consuming 1 kg of milk per day was calculated to be 0.406 mg/kg body weight per day.

Premature labour and toxemia of pregnancy due to the higher concentrations of DDT and its metabolites were studied. Considerably higher amounts of DDT residues were reported in the maternal blood, placental tissue and umbilical cord blood of women undergoing premature labour and toxemia of pregnancy as compared with women with full term pregnancy. The following trend was observed:

Premature > Toxemia > Full term

Storage of DDT residues in relation to disease was investgated. High DDT levels were reported in the blood and uterine tissues of women undergoing hysterectomy as compared to blood and uterine tissues collected at the time of autopsy. The total DDT concentrations in blood and uterine tissues showed no correlation with the age.

The health effects posed by DDT residues in general population were studied to explore the possible health hazards. No consistent pattern of abnormalities was present either in medical histories or physical examination, nor were any clinical symptoms found to be associated with the concentration of DDT in the blood.

There was also a paucity of abnormal results in the biochemical studies. No correlation was observed between DDT concentration in the blood and altered biochemical test results, if any. The Erythrocyte Sedimentation Rate (ESR) and hemoglobin levels were within the normal limit.

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ABBREVIATIONS

DBP	4,4'-Dichlorobenzophenone
DBH	2,2-Bis(p-chlorophenyl)methanol
DDA	2,2-Bis(p-chlorophenyl)acetic acid
DDCN	2,2-Bis(p-chlorophenyl)acetonitrite
DDM	2,2-Bis(p-chlorophenyl)methane
DDMS	1-chloro-2,2-bis(p-chlorophenyl)ethane
DDMU	1-chloro-2,2-bis(p-chlorophenyl)ethylene
DDNU	1,1-Bis(p-chlorophenyl)ethylene
DDOH	2,2-Bis(p-chlorophenyl)ethanol
Dieldrin	1,2,3,4,10,10-Hexachloro-6,7-epoxy-1,4,4a, 5,6,7,8,8a-octahydro-1,4-endo-exo-5,8-dimethano-naphthalene
Kelthane (Dicofol)	1,1-Bis(p-chlorophenyl)2,2,2-trichloro-ethanol
o,p'-DDD	1,1-Dichloro-2-(O-chlorophenyl)-2-(p-chlorophenyl)ethane
o,p'-DDE	1,1-Dichloro-2-(o-chlorophenyl)-2-(p-chlorophenyl)ethylene
o,p'-DDT	1,1,1-trichloro-2-(o-chlorophenyl)-2-(p-chlorophenyl)ethane
p,p'-DDD, DDD or TDE	1,1-Dichloro-2,2-bis(p-chlorophenyl)ethane
p,p'-DDE or DDE	1,1-Dichloro-2,2-bis(p-chlorophenyl)ethylene
p,p'-DDT or DDT	1,1,1-Trichloro-2,2-bis(p-chlorophenyl)ethane
PCB	Polychlorinated biphenyls
ppb	Parts per billion (ug/litre or ng/gm)
ppm	Parts per million (ug/ml or ug/gm)
SGOT	Serum glutamic oxaloacetic transaminase
SGPT	Serum glutamic pyruvic transaminase
TLC	Thin layer chromatography

CONTENTS

1

INTRODUCTION

Today, the greatest threat posed to humanity, and to a great extent to the whole biosphere, is environmental contamination, a byproduct of man's advancement in industralization, urbanization and modern agricultural practices. Emphasis has always been in the extensive use of pesticides to curb parasitic infections and other vector borne diseases. Among the pesticides, organochlorine compounds are well known for their long persistence in the environment and in living organisms (280); thus posing a challenge to the ecologists and toxicologists. For this reason, some advance countries which can afford even costlier chemicals have introduced safer and easily degradable pesticides as a measure to check unwarranted environmental pollution. Despite this the consumption of organochlorine pesticides particularly DDT and endrin have been continued in India and may be intensified during the course of years (224). It is still a matter of concern that no suitable substitute for DDT/BHC has so far been found. The quality of persistence makes DDT and BHC ideal insecticide under our geographical and economic conditions for public health and agriculture. The organochlorine insecticides are the most stable pesticide residues in the environment with the half life ranging upwards upto 20 years and perhaps longer in the case of DDT (87,194). This is a result of two characteristics of these compounds: they are nonpolar and they are relatively resistant to degradation by either chemical or biological means in the environment. The volatility of the chemical (263, 284) and its liposolubility (189) allows its incorporation and transportation within the food chain (98, 227). The concentration of pesticide residues in non target organisms may cause, sooner or later, disturbance in the ecosystem (150).

The human exposure to small amount of organochlorine insecticide is widespread as a result of environmental contamination and high stability of these compounds. High exposure to organochlorine compounds may occur among workers manufacturing or using them. In addition to their occupational exposure, workers may be exposed to these compounds carried into their homes from the work place, from general contamination of the ambient air and water and to their residues in diets. The major source of population exposure appears to be the ingestion of food contaminated as a result of general environmental contamination (53, 65, 113, 145, 192, 253); direct exposure to contaminated air, water and dust have also contributed to body burden (15, 28, 138, 212).

The pharmacological and toxicological manifestations exerted by acute and chronic doses of organochlorine compounds in animals, especially the sublethal chronic effects resulting from the extensive bioaccumulation in fatty tissue are of great significance. High oral doses of DDT can cause genetic alteration (96), carcinogenicity (74, 234), and abnormalities in the reproductive functioning in a wide variety of animals (30, 125, 210, 257).

Organochlorine compounds have long been shown to produce toxic effects in prolonged industrial exposure. Conditions representative of general debility include dermatitis, subtle blood changes, general weakness, palpitations, functional angiospasm, headache, dizziness, diminished appetite, vomiting, lower abdominal pain, chronic gastritis, benign chronic hepatitis, isomnia, a sympathetic vascular/asthenic syndrome, vegetative dystonia, and confusion (27, 163). Organs, systems, or functions that have been studied with the exclusion of other organs, systems,or function of the same workers include: the respiratory system (32), liver (26), stomach (165), kidneys (164), adrenals (18), skin (148, 149) and labour and

the puerperium (198).

The groups at high risk in the general human population are the young who are exposed to relatively high organochlorine pesticide levels during intrauterine life, infancy and childhood. Relatively high organochlorine pesticide residues are received by the fetus due to placental transfer, by the infant during nursing, due to the excretion in the milk, and by the child, due to higher intake per kilogram body weight in comparison with the adults.

Organochlorine pesticides have been shown to have various effects on the reproduction. DDT have been shown to be estrogenically active (52). Other effects on reproduction attributed to DDT include inhibition of testicular growth and secondary sexual characteristics in Cockrels (41); decrease in the reproductive success in birds due to diminished egge production (257), egg shell thinning (156, 158) and increased rate of embryonic mortality (30, 125); reduced fertility in mallards (125), and teratogenicity in birds (210). In rats, o,p'-DDT significantly advances puberty and induces persistent vaginal estrous after a normal estrous cycle(126). Studies on women with reproductive pathology has revealed an association between the higher plasma levels of organochlorine compounds and toxemia of pregnancy (290), missed abortions (243, 246), undersized fetuses for state of development (291) and premature labour (243, 246, 291).

Due to dearth of information on effects of organochlorine pesticides on humans, it is not possible to assign with any certainity a level of risk or to identify a critical threshold of risk which may be associated with human exposure to these chemicals. For this reason long-range public health significance of organochlorine pesticide contamination in humans still remain unknown.

In the present study an attempt has been made to assess the extent of environmental contamination and seriousness of the potential health hazards posed by DDT and its metabolites in occupationally unexposed population of Delhi area.

Objectives

I. *Studies on soil samples*

(a) To estimate the levels of DDT and its metabolites in soil samples collected from 50 sites surrounding a DDT manufacturing factory in Delhi.

(b) To relate the levels of DDT in soil with the location of DDT factory.

II. *Studies on occupationally unexposed population of Delhi area*

(a) To estimate the levels of DDT and its metabolites in the blood of occupationally unexposed population of Delhi area.

(b) To study the adverse health effects, if any, of high DDT levels in occupationally unexposed population.

III. *Studies on pregnant and lactating women*

(a) To indicate the transfer of DDT to prenates via placenta and neonates via milk through studies on maternal blood, umbilical cord blood, milk and placental tissue of pregnant and lactating women.

(b) To calculate the daily intake of DDT by the neonates.

(c) To compare the levels of DDT and its metabolites in women undergoing full term pregnancy, premature labour and in cases of toxemia of pregnancy.

(d) To study the adverse health effects, if any, of high DDT levels in pregnant women.

IV. ***Studies on blood and uterine tissue***

(a) To compare the levels of DDT and its metabolites in blood and uterine tissue of women undergoing autopsy and hysterectomy.

(b) To study the adverse health effects, if any, of high DDT levels in women undergoing hysterectomy.

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REVIEW OF LITERATURE

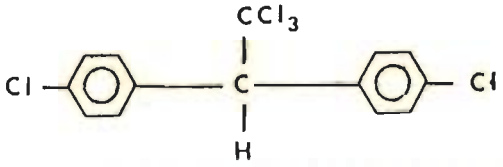
2.1 Introduction

Chlorinated hydrocarbons are a group of synthetic chemical compounds which have gained immense importance due to their persistent pesticidal activity. These compounds have been extensively used for controlling the vectors of typhoid and malaria and against insect pests which causes greater havoc and danger to agricultural crops. During the last two decades the use of some of these compounds as pesticides has been gradually minimize as these compounds were found to be the major pollutants of the environment. In recent years, there have been many reports of organochlorine pesticides in air, rain, water, dust, rivers, and the sea, and the body of aquatic and terrestrial vertebrates, fish, birds, mammals and man. These discoveries began to cause concern about possible long-term effects of the residues present in the environment and human tissues and thus helped in increased awareness of the need for research in this field.

DDT does not occur naturally. It was first synthesized by Zeidler as reported in 1874. However, it was not put to any use until its insecticidal properties were discovered by Paul Muller in 1939. The term DDT is a common name approved by International Standard Organization for the technical product of which 1,1,1-trichloro-2,2-di(4-chlorophenyl)ethane (p,p'-DDT) is the predominant component. The commercial product contain: p,p'-DDT, 77.1 %; o,p'-DDT, 14.9 %; p,p'-DDD, 0.3 %; o,p'-DDD, 0.1 %; p,p'-DDE, 4.0 %; o,p'-DDE, 0.1 %; and unidentified compounds, 3.5 %.

2.1.1 *Chemical structure*

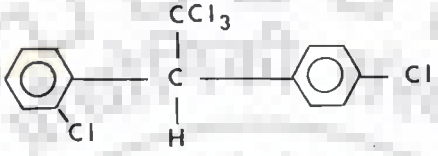
p,p'-DDT



$C_{14}H_9Cl_5$

Mol. wt: 354.5

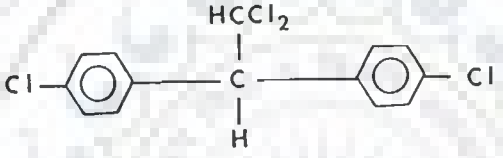
o,p'-DDT



$C_{14}H_9Cl_5$

Mol. wt: 354.5

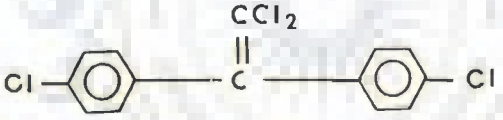
p,p'-TDE (DDD)



$C_{14}H_{10}Cl_4$

Mol. wt: 320.0

p,p'-DDE



$C_{14}H_{10}Cl_4$

Mol. wt: 318.0

2.1.2 *Synonyms and trade names*

Arkotine	Cesarex	Dinocid
Gesarol	Guesapon	Gyron
Guesarol	Ixodex	Neocid
Estonate	Dicophane	Neocidal

2.1.3 *Production*

Technical DDT is made by condensing chloral hydrate with chlorobenzene. It is known that DDT has been manufactured in many parts of the world including the developing countries. However, at present there is only one factory in USA, one

in France, and one in India. At the height of DDT production, about 1964, 400,000 metric tonnes was used annually in worldwide agriculture, forestry, public health and other pest control programmes. Since 1964, global production of DDT has decreased progressively. In India, large scale use of DDT in public health started in 1963-54 with the Malarial Control Programme. This later was changed into National Malarial Eradication Programme. Since then there had been a gradual increase in the consumption of organochlorine pesticides. The figures for DDT was mere 375 tonnes per year preceding the launching of Malaria Control Programme in 1952 but reached the peak figure of 25,000 tonnes in recent years. In 1967, the agricultural application of DDT totaled 34,000 tonnes from which it appears that the agricultural and vector control uses are of similar order (224).

2.1.4 *Properties*

All isomers of the compound DDT are white, crystalline, tasteless, almost odourless solids with the empirical formula $C_{14}H_9Cl_5$ and a relative molecular mass of 354.5. The melting range of p,p'-DDT is 108.5 - 109.0 °C and its vapour pressure is 2.53×10^{-5} Pa (1.9×10^{-7} mmHg) at 20°C. DDT is insoluble in organic solvents as follows (g/100 ml): benzene, 106; cyclohexane, 100; chloroform, 96; petroleum solvents, 4-10; ethanol, 1.5. It is highly insoluble in water.

2.1.5 *Uses*

The organochlorine compounds are used on a large scale in the fields of agriculture and public health. In agriculture,

they are used as insecticides, acaricides and fumigants to control pests in orchards, vegetable, grain, cotton and tobacco fields, vineyards and forests. They are also used for seed dressing, and in certain cases as rodenticides. In the field of public health, they have played a decisive role in eradicating certain parasitic diseases such as malaria, chag's disease, plague, typhus, yellow fever, dengue/haemorrhagic fever, encephalitis, filariasis, African trypanosomiasis, onchocerciases and leishmaniasis.

2.1.6 *Sources and pathways in the environment*

Organochlorine insecticides, like DDT, has been sprayed on people, domestic animals, buildings, agricultural crops, and forests. This has resulted in the ubiquitous distribution of residues of DDT in the environment (39, 91, 305). These insecticides after their application are not confined to the target organisms, because they are not taken up and degraded completely by these organisms, certain non-target organisms also accumulate their residues. Some DDT in soil enters the air by evaporation or on wind-blown dust. Even if water courses are avoided initially, some insecticides will be washed into them by rains, mainly in conjugation with soil particles (295). The problem of contamination of pesticides in food grains, dairy products, vegetables, fruits and in living environment as a whole can be visualized in the schematic diagram shown in Fig.A.

The residues of DDT and other insecticides, although not immediately toxic to a variety of animal life at environmentally occurring concentrations, have been reported to cause delayed disturbances in the ecosystem (86, 239, 132).

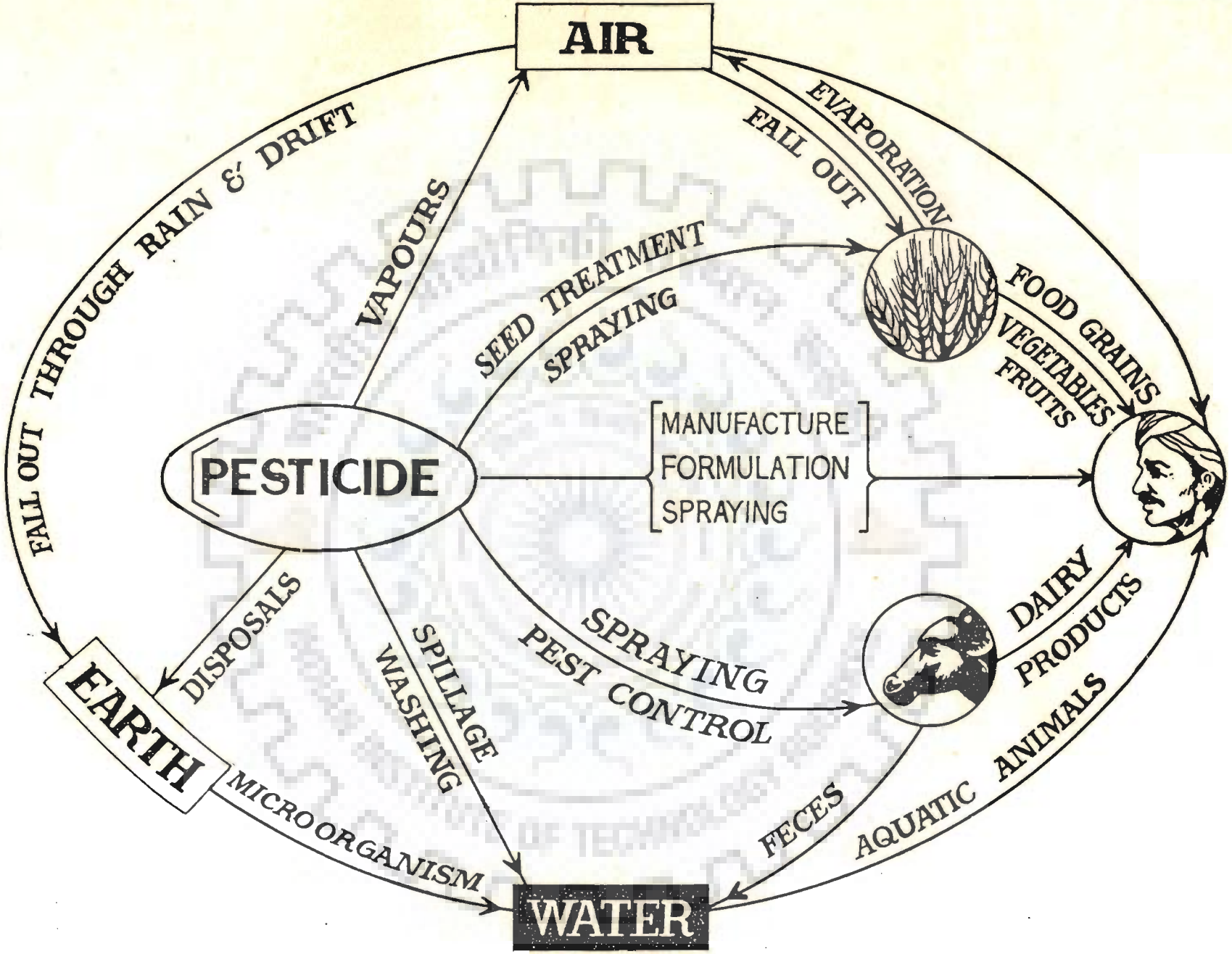

AIR
EVAPORATION
FALL OUT
FALL OUT THROUGH RAIN & DRIFT
VAPOURS
SEED TREATMENT
SPRAYING
FOOD GRAINS
VEGETABLES
FRUITS
PESTICIDE
MANUFACTURE
FORMULATION
SPRAYING
DISPOSALS
SPILLAGE
WASHING
SPRAYING
PEST CONTROL
DAIRY
PRODUCTS
EARTH
MICROORGANISM
FECES
AQUATIC ANIMALS
WATER

Fig. A

2.2 Residues in soil

Widespread application of pesticides in the environment has resulted in the global contamination of the ecosystem (1, 39, 91, 237, 309). Residues of DDT and other organochlorine insecticides have been detected from all the components of the environment viz. soil, water, air and biota. Large amount of pesticides reached the soil, either as direct applications, from fall out from an aerial spraying, in rain or dust or from plant and animal remains which become incorporated with the soil. Thus, the soil is an environmental reservoir for the residues of pesticides from where they move into the atmosphere, water or living organisms (91). The most common residues in soil are of DDT and related compounds in almost all the soil surveyed for pesticide residues. The most likely explanation for the larger residues in soil seem to be the longer period of usage of DDT and its persistence. The organophosphate and carbamate insecticides decompose in the soil more rapidly whereas chlorinated hydrocarbons are highly stable and persistent (87, 194, 285). The average time for 95 % disappearance of DDT would be 10 years (range, 4-30 years) with an average of about 50 % (26-78 %) remaining after three years (87). It was estimated that more than 50 % of applied DDT would remain in soils for at least 15 years (55). In a study conducted at the Indian Agricultural Research Institute in India, it was found that in 180 days, 94.8 % of p,p'-DDT mixed as 5 % dusts into the top 15 cms layer of soil were lost. The initial levels of these compounds ranged from 2.4 to 3.35 ppm (9).

Soil acts as reservoir for the pesticide residues in

the environment from where they either move into aquatic system by leaching or run off (88), or may be accumulated in plants and animals (91, 213) or may be volatilized into atmosphere (235, 263, 284), depending upon various factors such as temperature, soil type, moisture content, solubility of pesticide, degree of absorption and vapour pressure of pesticides (111, 116, 118). Chlorinated hydrocarbon insecticides being hydrophobic have been shown to be readily absorbed on particulate matter (56, 119). The organic matter accelerate pesticide degradation in soil (51).

Generally residues of DDT in soil are mainly composed of p,p'-DDT, p,p'-DDD and p,p'-DDE and sometimes o,p'-DDT (10, 35, 39, 42, 45, 187, 285). In addition to the usually reported metabolites of DDT, Kelthane and DDMU were also detected (78). In the surface soil of five west Alabama Counties the presence of DDT was reported (10). In orchard soil, presence of an oxidation product of DDT, DBP, was reported (155). Soil concentration of DDT vary according to the land used. In general, orchards and vegetable growing areas showed high DDT concentrations than did corn and hay-growing area (271). In desert and prairie soils, a mean of 1.6 ppm of DDT was reported (170). In another study a mean of 0.48 ppm DDT in a desert soil was calculated (283). A mean of 0.09 ppm of DDT was reported from Canadian Tundra(37).

In a survey of 1483 sites in 37 states of USA,dieldrin, DDT, aldrin, chlordane, heptachlor epoxide were reported(48). DDT residues consisted of o,p'-DDT, o,p'-DDD, o,p'-DDE, p,p'-DDT, p,p'-DDE, and p,p'-DDD. In another survey (49) soil samples from five metropolitan areas of USA were surveyed

for organochlorine pesticide residues. All areas exhibited heavy concentrations of total DDT. Within the metropolitan areas, samples from the urban or core city locations generally had higher pesticide concentrations than did samples from suburban locations. Pesticide residue concentrations were generally higher in soils of metropolitan areas than in nearby agricultural soils. The organochlorine pesticides in the major agricultural areas of the US were monitored annually from 1968-73 (46). The agricultural soils were widely contaminated with low levels of organochlorine pesticide residues (DDT, DDE, dieldrin etc.). The residue concentration decreased as the application of the compound decreased. In another survey (233) soil samples collected from the Eastern Everglades National Park, Florida, U.S.A., and adjacent farm lands were analyzed. Agricultural sites showed high concentrations of DDT whereas little contamination of park lands was reported. Kuyumdzhieva (169) monitored levels of DDT in soils of 14 districts of Bulgaria. DDT residues consisted of p,p'-DDE, o,p'-DDT, p,p'-DDD and p,p'-DDT; the values ranged from thousands to several mg/kg soil. In India, DDT residues were monitored from 50 sites in Delhi (307). Maximum total DDT concentration of 2.6 ppm was detected from Durga Nagar in the vicinity of a DDT factory. In Canada, the maximum total DDT was found to be 30.38 ppm and residues of p,p'-DDE, p,p'-DDD and p,p'-DDT were detected (39). In another survey of soil in Ontario, Canada (187), the presence of a maximum of 28.6 ppm total DDT which included p,p'-DDT, o,p'-DDT, p,p'-DDD and p,p'-DDE was reported.

2.3 Residues in human

The human pesticide residue is a biological index of pesticide exposure. There are three types of human exposure to pesticides. Acute exposure which is usually the result of accidental contamination by excessive amounts of pesticides, chronic exposure which most frequently occurs in pesticide workers by virtue of their occupation, and incidental exposure which is the consequence of ubiquity of pesticides and their presence in trace amounts in air, water, food and dust. The route of absorption of pesticides in human body is mainly through gastrointestinal tract (278). When present in the air in the form of a small particle sized aerosol or dust, it is likely to enter the alveoli of the lungs, from which it is readily absorbed. Skin absorption of DDT depends on the solvent or vehicle employed: crystalline or powdered DDT is not usually absorbed through the skin, but emulsions of DDT are absorbed to some degree. It has been shown that persistent chemicals are taken up to a greater extent than non-persistent substances by cultured human cells and increased uptake of chemicals by the cells accorded with decreasing water solubility in general. The water solubility of DDT is approximately 0.04 mg/litre(295). Because of this property DDT is taken up easily by tissues and accumulated in large quantities as compared to other pesticides where water solubility is higher than DDT. It has been assumed that these chemicals are inextricably deposited in adipose tissue; this assumption is based mainly on evidence of their limited biotransformation (with minimal urinary excretion) and high lipid solubility (relatively low

blood-fat partition). The distribution of DDT may depend on the specific type of lipid present in a given tissue (e.g. phospholipid vs. triglyceride) or possibly by the presence of specific binding proteins both membrane bound and intercellular (114).

2.3.1 *Residues in occupationally exposed workers*

The DDT and its metabolites were monitored in tissues of workers exposed to DDT in various occupations. The highest storage of DDT was reported in a healthy worker whose fat contained DDT and DDE (as DDT) at concentrations of 648 and 483 mg/kg, respectively (121). In contrast to this study, considerably lower storage values were reported among the most exposed persons in a DDT manufacturing plant (172); total DDT-related material in the serum of a worker was 2.7 mg/litre. In another study (102) value as high as 38.4 mg/litre was reported in the blood of a pilot and 195 mg/litre in the blood of warehouseman. This concentration was about 20 times higher than the values found in control group.

In Sudan (92), blood of people occupationally exposed to DDT contained DDE and p,p'-DDT in all the samples. The concentration ranged from 0.01 - 0.12 µg/ml for DDE and 0.02 - 1.01 µg/ml for p,p'-DDT. DDD was found in only two cases.

In a study of 182 subjects occupationally exposed in agriculture and 27 samples exposed in the chemical industry (57), was found that DDT residues increased with the age.

In India (252) blood serum of 20 workers occupationally exposed to DDT for an average duration of 14 years was analuzed. p,p'-DDT, p,p'-DDD, and p,p'-DDE were detected. A

higher incidence of total DDT equivalent, about 10 times, was observed in DDT exposed workers when compared with unexposed persons from the general populations. The levels of DDT in serum of occupationally exposed workers were more than the permitted level of 200 ppb. The daily intake was computed to be about 10 mg/man/day as against 0.25 mg - the acceptable daily intake. Poland et al (221) reported an average level of 573 ppb DDT in the blood of exposed workers with an average daily intake of about 18 mg/man/day.

A survey of 144 spraymen associated with malaria control in India by the World Health Organization revealed that blood levels of DDT were 7.5 - 15 times those in controls and were at least as high as those reported for workers who manufactured or formulated DDT elsewhere in the world(295).

The first evidence that a part of DDT absorbed by man is metabolized to DDE was obtained from the analysis of fat from a DDT plant worker (179). DDA is the main urinary metabolite of DDT. In occupationally exposed workers whose DDT intake was about 35 mg/man/day, the concentration of DDA in urine ranged from 0.12 to 7.56 mg/litre and averaged 1.71 mg/litre (206). In workers whose exposure was about half as high, DDA in urine ranged from 0.01 to 2.67 mg/litre with a mean of 0.97 mg/litre. Continuous sampling of a DDT formulating workers' urine showed that excretion of DDA increased promptly, when exposure began on each of five consecutive work days and often continued to increase after exposure, sometimes reaching a peak about midnight before decreasing rapidly. The highest concentration of DDA reported in this study was 0.68 mg/litre (301).

2.3.2 *Residues in general population*

BLOOD:

The DDT and related compounds were reported in blood of general population of different countries. No significant difference was reported in the concentrations of chlorinated hydrocarbon insecticides in plasma and serum (67). However, the compounds are not always evenly distributed between the red cells and the plasma. Some compounds e.g. DDD, are more concentrated in the red cells otherwise most of the compounds are more concentrated in plasma. The p,p'-DDT, o,p'-DDT, p,p'-DDE, o,p'-DDE, p,p'-DDD and o,p'-DDD were reported in the blood in different surveys all over the world (295). The persons without special exposure to DDT had relatively constant serum levels of DDT and DDE, the DDE value differed more than the DDT values from persons to persons (14). The concentrations of DDT in the sera of blacks was reported to be 2 to 3 times greater than those in whites (242). There was a small but statistically significant decrease in the concentrations of DDT-related compounds in the plasma of women 1-6 day postpartum compared with the same women early in pregnancy. Most of the decrease seems to occur about the last 10 days before delivery (62). The concentration of DDT-related compounds in various tissues of women at the time of caesarian section or normal delivery is less than in non-pregnant women in the same community (222). The concentration of p,p'-DDE was remarkably constant throughout the day, but minor increase in p,p'-DDE and p,p'-DDT occurred after a meal (230). The postmortem blood was reported to contain more DDD (109).

In Japan (82), 27 samples of human blood were analysed. All the samples were found to contain substantial quantities of organochlorine pesticides. DDE ranged from 3.2 ppb - 196 ppb with a mean value of 11.2 ppb on whole blood basis.

In Canada (38), mean value of total derived DDT in blood was found to be 0.032 ppm. The residents of the area where DDT was recently used showed higher levels of total derived DDT. The hemoglobin level for men and women of the two areas were within normal limits. Therefore, absorption of DDT seemed to have no effect on erythropoisis of red cells.

In India (7), blood samples from 182 people were analysed. All the samples except 8 contained DDT and its metabolites. The DDT-metabolites detected were p,p'-DDE, p,p'-DDD, p,p'-DDT and o,p'-DDT. The average total DDT concentration in the whole blood ranged from 0.177 to 0.683 mg/litre in males and from 0.166 to 0.329 mg/litre in females. DDE accounted for most of the total DDT.

In U.S.A. (22), survey of organochlorine pesticide in the blood revealed that p,p'-DDE and p,p'-DDT were present in 59 blood samples but no o,p'-DDT was detected.

The serum samples of 193 people living in four different parts of Yugoslavia and in two groups of occupationally exposed workers were analysed (151). All the samples contained p,p'-DDE. Workers engaged in the production, formulation and packing of pesticides had a higher incidence and higher contents of the residues than the general population.

The geometric mean level of total DDT in serum samples (76.2 ng/ml) from 499 persons living downstream from a DDT-manufacturing plant was found to be several times the national

geometric mean (15.0 ng/ml) level (167). DDE isomers, metabolites of DDT accounted for an average of 87.6 % of total DDT. Total DDT levels increased with age even when controlled for other independent variables significantly associated with DDT: race, sex, fish consumption, years of residence, socio-economic status, alcohol consumption and serum triglyceride levels. Total DDT levels were not associated with specific illness or ill health. Total DDT levels were positively associated with levels of serum cholesterol, triglyceride, and glutamyl transpeptidase. The findings that serum DDE levels increased with age suggested that no equilibrium in body has reached or that pharmokinetics or serum/adipose partition varied with age (167).

MILK:

The DDT and related compounds are reported to be excreted in milk of women from general population (124). Table A shows the mean pesticide residues in human milk from different countries. The contamination of human milk by DDT has a special significance since newborn infants are involved and milk constitute for them for a certain time, almost the sole source of nourishment. The concentration of DDT and DDE in milk were reported from different countries (295). The highest concentration of p,p'-DDT in a single sample of human milk (5.94 ppm) and the highest average concentration in samples collected in one locality (1.78 ppm) were reported from Guatemala (205). The babies in that town had a maximal intake of 1.06 mg/kg/day and an average intake of 0.32 mg/kg/day. In other studies more numerous samples taken only four years later in the same and other communities in Guatemala revealed

TABLE-A: MEAN PESTICIDE RESIDUES IN HUMAN MILK FROM DIFFERENT COUNTRIES.

Country	Year	DDT	DDE	Total DDT
Western Australia	1970-71	0.010	0.063	0.078
United Kingdom	1963-64	0.045	0.073	0.127
United States	1960-61	0.08	0.04	0.12
United States	1967-68	0.014	0.038	0.056
Western Australia	1969-70	0.04	0.12	0.17
Netherlands	1971	0.016	0.030	0.049
Belgium	1969	0.048	0.072	0.128
Sweden	1970	0.039	0.067	0.114
Germany	1970	0.031	0.081	0.121
Guatemala	1976	-	-	0.378
Guatemala	1979			
(a) Guatemala City		-	-	0.480
(b) Morales,Izabel		-	-	2.55
(c) Escuintla		-	-	3.54
El Salvador	1979	-	-	0.695

Data from Conway et al (60), de Campos and Olszyna-Marzys (73), and Winter et al (300).

entirely different results. The highest single value of observed total DDT in 1974 was 5.69 mg/litre. The range of average values for different locations was 0.04 - 0.86 mg/litre. The means for total DDT for three communities studied earlier were: La Bomba, 0.59 mg/litre; El Rosario, 0.28 mg/litre; and Cerro Colorado, 0.47 mg/litre (300). The authors recognised the importance of the agricultural uses of DDT as a potential source of the compound in human milk. However, they attributed the change between 1970 and 1974, almost exclusively, to the substitution of propoxur for DDT in residential spraying to combat malaria.

A negative DDT balance during lactation was reported in women (226). Many authors studied the ingestion of DDT in food and secretion of DDT in milk in the same human. The negative balance was confirmed (3, 4, 59). The slightly greater secretion of DDT by urban mothers (compared to rural mothers) was reported in Japan and was attributed to greater intake of cows milk by urban mothers (267). Significantly lower levels of DDT (mean of 0.008 ppm) and of DDE (mean of 0.035 ppm) were reported (139) as compared to earlier reports of DDT in the milk of city dwellers. In some rural black people, levels quite high as 0.05 to 1.90 ppm were reported (304).

The milk samples collected from rural localities were reported to contain extremely high concentrations of DDT(204). The milk in Guatemala contained between 7 and 244 times the maximum DDT concentration legally tolerated in USA and in number of other countries. The amount of mothers' milk consumed daily by Guatemalan child was between 6 and 207 times the amount of DDT considered by WHO as acceptable.

The organochlorine residues in human milk residents of Alberta, Canada were analysed during 1966-70 and 1977-78 (64). The average residue levels were generally lower in 1977-78 samples whereas the incidence of residues was generally lower in 1966-70 samples. Average levels of p,p'-DDT and its metabolites were substantially lower in second survey than during the first. No correlation could be inferred between the donors' age groups and average pesticide residue level.

Human milk samples obtained from 34 women living in the Mississippi Delta, a high pesticide usage area, and from six women living in Starkville, Mississippi, a low pesticide usage area were analysed (21). Nine samples were collected from women before and after their babies had nursed so that fat levels and total DDT levels could be compared on whole milk and milk fat bases. Total DDT values were independent of collection time if calculated on a milk fat bases, but not if calculated on a whole milk basis. Residue level for p,p'-DDE, p,p'-DDD and total DDT were significantly different ($p<0.01$) in samples from two areas. A mean value of 10.17ppm total DDT, found in the milk fat of samples from the high pesticide usage area, was the highest ever reported. Samples from the low pesticide usage area contained a mean level of 2.36 ppm total DDT.

In Canada (185), 100 human milk samples were reported to contain p,p'-DDE ranging upto 144 ppb with a mean value of 35 ppb; o,p'-DDT ranging upto 6 ppb with a mean value of 3 ppb and p,p'-DDT ranginf upto 21 ppb with a mean value of 6 ppb.

The human milk samples (34) obtained 3-5 days after delivery and 37 samples obtained at later time of lactation

(upto 55 weeks) were analysed by Kauthacker et al (151). All samples contained 31 µg/litre in the beginning of lactation and 53 µg/litre at later time interval. No definite trend was found in the p,p'-DDE levels in the breast milk of five women upto 45 weeks, although initially a decrease in p,p'-DDE level was observed.

The DDT and DDE levels in human milk samples were compared with respect to race, rural vs. urban home, previous nursing number and body weight (180). The average level of total DDT in human milk was 1.8 times higher than the maximum allowed in cow milk, but lower than values found in some regions of industralized countries.

The organochlorine residues in human milk samples from an agricultural area of Slavonia, Yugoslavia were determined (161). The most abundant contaminant p,p'-DDE ranged from 42.0 - 418.5 ppb.

In Sweden (128), individual samples of human milk collected in Uppsala at three month post-partum (18 samples) or six months post-partum (23 samples) were analysed. Mean levels of organochlorine compounds in three and six month groups were similar. Mean content of DDE, the major metabolite of DDT in all samples was 54 ug/kg fresh weight (1.3 mg/kg on a fat basis) with the range of 8.4 - 220 ug/kg (0.49 - 3.0 mg/kg fat). The calculated intake of DDT complex (DDT + DDE + DDD) by some suckling infants exceeded acceptable daily intake proposed by FAO/WHO expert group.

In Canada (79), 154 human milk samples collected 3-6 days after parturition from four hospitals in Quebec, were analysed. The USA Codex Alimentarius Commission maximum

residue level limits were exceeded in 30 % of the samples with regard to total DDT (mean ± S.D. 1.087 ± 0.880 mg/kg milk fat). A significant positive correlation was observed between cigarette smoking and DDE content of human milk fat.

In U.S.A. (268) human milk samples from Oaher residents contained p,p'-DDT ranging from 0.32 to 520 ppb with a mean value of 170 ppb and p,p'-DDE ranging from 260 to 570 ppb with a mean value of 2000 ppb. The human milk samples of residents of neighbouring islands contained p,p'-DDT ranging from 32 to 290 ppb with a mean value of 150 ppb and p,p'-DDE ranging from 540 to 3700 ppb with a mean value ranging from 1800 ppb. The levels of pesticides were not associated with age of the mother. The weight of the mother did not appear to affect the excretion of pesticides. No significant correlation was observed between the intake of meat and dairy products and residues in milk but individuals who reported higher frequency of meat consumption tended to have higher levels than those who ate meat products less frequently. No significant difference was found in residue level of milk of women whose spouse were occupationally exposed to pesticides compared to those whose spouse were not exposed. The frequency of household use of non-persistent pesticides did not effect the concentration of pesticides in milk.

In Norway (254), 133 human milk samples collected from 7 cities with various degree of industrialization were reported to contain organochlorine pesticide residues. The average DDT and DDE levels in human milk were 29.8 ± 23.5 and 25.8 ± 20.7 ppb, respectively. No dramatic geographical differences were observed.

In India (251), 25 breast milk samples were analysed. The mean total DDT in milk was reported to be 0.127 ± 0.019 ppm. The daily intake of about 108.628 ppm of total DDT by neonates was suggested. In another study (144) conducted in Punjab, India, samples of human milk showed the presence of DDT residues in amounts greater than those reported for most of the other countries.

In Spain (20), 20 samples of human milk were analysed for organochlorine pesticide residues. p,p'-DDE ranged from 0.052 to 0.250 ppm with a mean value of 0.017 ppm; p,p'-DDD ranged from 0.000 to 0.008 ppm with a mean value of 0.003 ppm and p,p'-DDT ranged from 0.027 to 0.150 ppm with a mean value of 0.083 ppm.

In Finland (296), total DDT in 50 human milk samples ranged from 0.005 to 0.084 ppm with a mean value of 0.031 ppm (on whole milk basis). The average daily intake of total DDT of a Finnish child weighing 5 kg and consuming 1 kg of milk a day was calculated to be 0.006 mg/kg, which exceeded ADI value of 0.005 mg/kg proposed by WHO.

In Sweden (199), 745 human milk samples were reported to contain total DDT ranging from 0.97 to 2.43 ppm with a mean value of 1.54 ppm in 1978, 0.94 to 2.01 ppm with a mean value of 1.24 ppm in 1979 and 0.86 to 2.05 ppm with a mean value of 1.22 ppm in 1980.

Variation in the organochlorine pesticide residues during breast feeding and its diurnal pattern was studied(184). Total DDT increased during each feeding and reached their maximum at the very end of the mid-day feeding, coinciding with maximum fat output (184). Mes et al (186) reported significant decrease in the level of p,p'-DDE and p,p'-DDT during lactation.

2.4 Degradation of DDT residues

Pesticide residues in the environment are degraded under the influence of several physical and biological factors depending upon the stability, persistence and chemical nature of the pesticide itself. A number of physical and chemical factors such as light, air, surface, moisture, pH, metal etc. are known to degrade DDT and other pesticide residues (177, 118, 303, 311). Conversion of DDT to DDE was reported to enhance significantly at higher temperature and soil water (111). The photooxidation products of DDT include benzoic acid, aromatic ketones and chlorinated phenols (220).

Chemical transformation of the pesticides are usually mediated through water and may involve hydrolysis or oxidation. In soil, these reactions may be catalyzed by clay surface, metal oxides, metal ions, organic surface etc.(108). The metal centres may be involved in the degradation of DDT (261) and thus zinc mixed with DDT, acetone and acetic acid led to the formation of DDD and DDMS, which are environmentally less objectionable than DDT or DDE. The chemical structure also affects the biodegradability of DDT (262).

The role of micro organisms being involved in the degradation of DDT has been thoroughly reviewed (12, 36, 43, 178, 274). The metabolites of DDT, DDD and DDE, were detected in surface water sediment model ecosystem receiving organic amendments (142), however, none of the compounds generated microbiologically were observed in the model ecosystem. Later it was observed that 43 % of the bacteria isolated from sea water and sediments converted between 5 and 10 % of the DDT supplied *in vitro* to water soluble products (142). About 35 %

of bacteria, however, converted 5 % or less DDT to water soluble products (143). The major metabolites of DDT in a water sediment ecosystem were DDD, DBP and DDE (216).

Algae as a whole are not very active in degrading pesticidal chemicals. No metabolism of DDT by an alga (Anacystis nidulans) was reported (34). Conversion of DDT to DDE has been reported in marine diatoms and in fresh water diatoms (188).

The role of higher animals in the degradation of pesticides has been discussed in context of survival mechanisms. Both invertebrates and vertebrates including man are known to metabolize DDT to less toxic metabolites. Sternberg et al (260) first reported that DDT resistant house-flies had the ability to detoxify DDT to non-toxic metabolite, DDE. After that large number of reports demonstrating the metabolic pathways of DDT in different insects and other animals have been published. Among insects DDT is metabolized to DDE, in mosquito larvae (157, 218), in housefly (260) and in German cockroach (182) and to DDA by the homogenate of body louse (214).

Metabolism of DDT in soil invertebrates has been reported (270). Terrestrial snails and slugs have been found to metabolize DDT to DDD and DDE with DDD being the predominant metabolite (80, 105). DDE was reported to be the major metabolite of DDT in earthworms (90). DDMU was reported in the earthworms but not in the soil (70). In later studies(71) DDMU was not detected in the earthworm which were exposed to soil containing DDT. The conversion of ^{14}C-DDT to DDE and DDD by Pheretima posthuma was reported (8). After 24 hours about 35 % of the DDT was converted to metabolites DDE and DDD.

Pretreatment with DDT increased the metabolism of DDT and thus about 42 % of total DDT was converted to DDE + DDD after 24 hours. Similarly, pretreatment with lindane and dieldrin also increased the rate of metabolism by the earthworms (8).

DDD and DDE, the degradation product of DDT are common among aquatic invertebrates. A fresh water planarian, Phagocata velata was reported to convert DDT to DDE and DDD (217). DDD was the major degradation product, while only a small portion was converted to DDE. In vivo metabolism of DDT to DDE has been reported in snails, Physa (146), vivipara species (306) and in crustaceans (197). No metabolite of DDT was detected in marine copepod, Calanus sp. (68). In Marine invertebrates DDE was the major metabolite but DDD was detected only in Daphnia magna and Palaemonetes sp.(136). Both of the major metabolites of DDT i.e. DDE and DDD have also been reported in the squid, Loligo forbesi (93), mussels, Mytilus sp. (107, 120), and in oysters, Crassostrea commercialis (58).

Fish are shown to metabolize DDT to mainly DDE and occasionally DDD in some species. In cat fish, Hetropneustes fossilis, DDE was the only metabolite of DDT (6). Metabolism of DDT to DDE and DDD has been shown in brook trout, Salvelinus fontinalis (312); goldfish, Carassius auratus(110); mosquito fish, Gambusia affinis (219). No effect of pretreatment of p,p'-DDT, DDD, DDE and DDMU on the rate of dehydrochlorination of ^{14}C-DDT in brook trout, Salvelinus fontinalis was observed (5). About 35 % of DDT was reported to be degraded to DDE and DDD in the eggs of brook trout, Salvelinus fontinalis (16) by the time of yolk depletion in fry.

Birds, predominantly metabolize DDT to DDE. The p,p'-DDE as the only metabolite of DDT has been reported in purple heron, Ardea purpurea and spoon ducks, Anas clupeata (19); sparrow hawks (33); water fowl (293) and in marine birds, cormrants and gulls (309). Another metabolite DDD has also been reported in addition to DDE in small migratory birds (137); Rudy ducks (294), and in Opsrey eggs (297). In pigeons, p,p'-DDT was mainly converted to p,p'-DDE and also some DDD which was further dehydrochlorinated to DDMU and DDMS (17). In domestic chicks very little DDE was converted to DBD but DDD was further metabolized to DBP through DDMS, DDNU, DDOH, DDA and DDM (2). In vitro studies have shown that about 35 % of the p,p'-DDT was converted to DDD under anaerobic condition in pigeon by liver microsomes (281).

Metabolism of DDT in mammals has been reviewed by Hayes (123). DDA was the major metabolite found in the rat faeces (141). In mouse, DDE, DDD, and DDA along with some unknown compounds were reported (147); DDE was the major degradation product in urine whereas DDD comprised the bulk of metabolites in faeces. The rats fed with p,p'-DDT and o,p'-DDT in their diet could dechlorinate p,p'-DDT to DDD in the liver but not in the fat whereas o,p'-DDT was converted to p,p'-DDT in the fat stores (159). In another study (215) it was found that DDT was finally converted to DDA through DDD, DDMU, DDMS, DDNU and DDOH. In addition to these metabolites DDE was formed which did not metabolise further. In mice DDE was the major metabolite of DDT in almost all the tissues whereas DDD was mainly produced in the liver (112). The metabolism of DDT in mice and hamsters was studied(104).

The presence of phenotic metabolites in the faeces of rats, wild seals and guilmots after they were fed with DDD were reported (264). The metabolism of p,p'-DDT and p,p'-DDE in the pig was studied (265). DDA was the major metabolite in pigs fed with DDT and it was detected in both conjugated and free forms.

The fact that DDE is stored in tissue was first demonstrated by (211) in connection with human fat. The authors pointed out that they did not know whether the compound resulted from partial degradation of DDT residues on plants or whether the DDE was formed during the process of digestion or after absorption. It is known that some food contains DDE but that man is capable of forming the product from DDT. The exact mechanism of the biotransformation of DDT to DDE remains in doubt. The identity of DDE was established by comparing the colorimetric and column chromatographic behaviour of the residue with those of a chemical standard (211). The identity of DDE was confirmed by infrared spectroscopy and by gas chromatography (179).

The metabolic pathway of DDT to form DDA was demonstrated (215). Organ perfusion studies have indicated that the liver is capable of biotransformation of DDT, DDE, DDD, DDMU, and DDMS, while the kidney transforms DDMS, DDNU, and DDOH (69). Cultures of embryonic lung cells are capable of metabolizing DDT to DDA via DDD (200).

Some aspects of non-mammalian, as well as, mammalian metabolism have been reviewed (83, 96, 160, 162, 249). The metabolism of microorganisms and plants, as well as of domestic animals,may influence the composition of DDT-residues in human food.

2.5 Effects of DDT on man

2.5.1 *Studies on occupationally exposed workers*

The exposure of DDT is greatest among manufacturers and formulators, moderate, among those applying it for agricultural purposes, less among the general population, and least among special groups whose location or practices minimize their exposure. However, for brief intervals the exposure during agricultural application may exceed anything that good industrial practice permits. The occupational exposure through areas of skin that are frequently unclosed (face, hands, forearms, neck and "V" of chest) is far greater than total respiratory exposure. Dermal and respiratory exposures were reported in workers spraying the interior of hundreds of millions of homes in tropical and subtropical countries (302). Dermatitis was commonly observed among men who used DDT solution (122). Some persons suffered temporary irritability, fatigue, and other ill defined symptoms after exposure to the dusty atmosphere of a delousing station, but the relation to these atopical findings to DDT were not clear (103). With these exceptions due largely to solvents, no illness clearly attributable to the formulations, much less to DDT, were revealed by the earlier studies.

Mild to moderate poisoning by DDT itself may have occurred among the group of active workers exposed to air concentrations of 500 to 42,000 mg/m^3. The men complained of parasthesia of the extremities, headache, dizziness and some other difficulties less clearly linked to DDT (11). Even higher concentrations in air have been associated with tremors of the tongue and hands as well as the numerous subjective

findings (40).

Clinical and laboratory examinations were carried out on 40 workers, all of whom were exposed to DDT and some of whom were exposed to number of pesticides (206). The men had been employed at this work with heavy exposure for 0.4 to 6.5 years and with slightly less exposure for as much as 8 years. Exposure was so intense that during working hours, many of the men reported with a heavy layer of concentrated DDT dust. By comparing their excretion of DDA with that of a volunteer given known doses of DDT, it was possible to estimate that the average dosages of three groups of the workers with different degree of occupational exposure were 14, 30 and 42 mg/per day, respectively. With the exception of the excretion of DDA and the occurrence of a few cases of minor irritation of the skin and eyes, no correlation was found between any abnormality and exposure to the insecticide. Since very large doses of DDT injure the nervous system and liver of experimental animals, special attention was given to a complete neurological examination and to laboratory test for liver function. Although few abnormalities were revealed, none related to DDT were detected (206).

In another study (172), 35 men employed from 11 to 19 years in a plant that had produced DDT exclusively since 1947 were studied. Finding from medical history of physical examinations, routine clinical laboratory tests, and chest x-ray films did not reveal any ill effects attributable to exposure to DDT. No case of cancer or blood dyscracis was found among the 35 heavily exposed workers in a DDT factory nor did the medical record of 63 men who had worked there for more than 5 years revealed these diseases. Two men were

employed who had a history of successfully treated cancer before they came to work, but no employee had contracted cancer during the 19 years the plant had operated; during this period, the work force varied from 110 to 135. Measurement of storage offered direct evidence of the men's heavy exposure. The overall range of storage of the sum of isomers and metabolites of DDT in the men's fat was 38-647 mg/kg compared with an average of 8 mg/kg, for the general population. Based on their storage of DDT in fat and excretion of DDA in urine, it was estimated that the average daily intake of DDT by the men with high occupational exposure was 17.5 - 18 mg/man/day compared with an average of 0.028 mg/man/day for members of the general population. There was significant correlation (r = +0.64) between the concentration of total DDT related material in the fat and the serum of workers. The average concentration in fat was 338 times higher than that in serum - a factor about 3 times greater than that for people with occupational exposure.

Compared to members of general population, the workers were found to store smaller proportions of DDT-related material in the form of DDE; the difference was shown to be related chiefly to intensity rather than to duration of exposure. DDE is relatively much less important and DDA much more important as excretory products in occupationally exposed men than in men of the general population (172).

The 36 most heavily exposed workers involved had fathered 58 children before they began working at the DDT factory and 98 children afterwards (299).

The largest number of heavily exposed workers whose health had been investigated were those associated with

malaria control in Brazil and India (295). In Brazil, periodic clinical examinations were made of 202 spraymen exposed to DDT for 6 or more years, 77 spraymen exposed for 13 years ending in 1959, and 406 controls. In the first examination carried out in 1971, minor differences between exposed and unexposed groups were observed in some neurological tests, but this result was not confirmed by the second examination in the same year nor in subsequent examinations. During a three year period, a survey of illness requiring medical care during the six month preceeding each periodic medical examination failed to demonstrate any differences between exposed and control groups. A relatively small number of analyses indicated that the concentration of DDT in the blood of spraymen was about 3 times higher than that of control.

In India (295) the blood levels of 144 spraymen were 7.5 - 15 times higher than those of the controls and were atleast as high as those workers who make and formulate DDT elsewhere. When the spraymen were examined, the only differences from the controls were that knee reflexes were brisker, slight tremor was more often present, and a time Romberg test was more poorly performed by the spraymen. The positive results lead to the selection of 20 men for reexamination by a neurologist who concluded that the differences found initially were not real or that the tests had returned to normal within the few months between the two examinations. In any event, the signs were not dosage-related, since they showed no correlation with serum level of DDT.

A detailed study of the liver function was performed on 31 men who had made and formulated DDT (173). Judging

from their excretion and storage, the men's exposure was equivalent to an oral intake of DDT at rates ranging from 3.6 to 18 mg/man/day for periods ranging from 16 to 25 years and averaging 21 years. All tests were normal such as total protein, albumin, total bilirubin, thymol turbidity, and retention of sulfobromophthalein sodium (BSP). One man had mild elevation of alkaline phosphatase (16 units) and SGPT (42 units). Another man had an alkaline phosphatase concentration of 14 units, while a third man had an SGPT level of 49 units. The alpha feto protein test was negative for all 20 of the men for whom the test was performed.

The induction of DDT or microsomal enzymes of human liver was demonstrated first in workers (124). DDT may be more important than DDE in this regard as indicated by the fact that Poland et al (221) observed induction in men with average serum levels of 0.573 and 0.506 ppm for DDT and DDE, respectively, while Morgan and Roan (191) found no induction in men with corresponding values of 0.052 and 0.222 ppm. The DDT has been used successfully to induce microsomal enzymes in order to promote metabolism of bilirubin in a case of congenital defect and to promote metabolism of phenobarbital in a case of overdose.

In addition to above studies, large number of workers with lesser exposure were also studied (124, 190, 209, 273). These studies failed to reveal effects of clinical significance among workers with prolonged, moderate exposure to a wide variety of pesticides. In a review of results for 2620 persons exposed to pesticides and of 1049 persons not occupationally exposed (190), it was found that apart from serum pesticide concentrations,the only significant and consistent change

associated with occupational exposure was a depression of serum bilirubin. This presumably was a reflection of a slight induction of liver microsomal enzymes. In addition, there was a tendency for serum alkaline phosphatase, SGOT, SGPT, and LDH to increase with increasing concentrations of DDT plus DDE in the serum, but the differences were small in all instances and statistically significant for SGOT and LDH only.

A positive linear correlation has been reported for the concentrations of vitamin A and of DDT-related compounds in the serum of men with at least 5 years of occupational exposure to DDT. However, the workers' DDT levels were little higher than those of persons in the general population, and their vitamin A levels were within normal limits (153).

Evidences regarding mutagenic activity of DDT and its significance in man is uncertain partly because the chromosomal changes that are examined are sensitive to viral infections and chemotherapy, which may not be recognized at the time of sampling and, in any event, have not been shown to injure health through a mutagenic mechanism. Comparing samples collected in winter and during the peak season of pesticide application, a slight increase in chromatid breaks were reported in the cultured lymphocytes of workers exposed to a wide variety of insecticides said to include DDT. A somewhat larger increase was reported for men exposed mainly to herbicides (308). In another study lymphocytes cultured from workers with an average DDT plasma level of 0.999 ppm show significantly more chromosomal and chromatid aberrations than did cells cultured from controls with an average plasma level of 0.275 ppm. The difference was not significant in

other comparisons in which the average plasma levels were 1.030 vs. 0.380 ppm and 0.240 vs. 0.030 ppm, respectively (227).

Recently increased risk of lung cancer was reported in pesticide exposed male agricultural workers (23).

2.5.2 *Studies on volunteers*

ORAL EXPOSURE:

Several studies were performed on volunteers to see the effects of one or a few carefully measured oral doses of DDT (131). A single dose at the rate of 10 mg/kg produces illness in some but not all subjects even though no vomiting occurs. Smaller doses generally produce no illness although a dosage of 6 mg/kg produced perspiration, headache, and nausea in a man who was sickly and hungry at the time of eating. Persons who were made sick by 10 mg/kg have not shown convulsions but convulsions had occurred in accidents when the dosage level was 16 mg/kg or greater (131). Rarely a dosage as high as 20 mg/kg may be taken without apparent effect (175). Dosages as high as 285 mg/kg have been taken accidently without any fatal results (101). However, large doses lead to prompt vomiting, so the amount actually retained cannot be determined accurately.

In acute poisoning a slight decrease in hemoglobin and a moderate leukocytosis without any constant deviation in the differential white count have been observed in volunteers (279). These findings are considered secondary to the neurological effects.

The possible clinical effects of many repeated doses of DDT were first reported by Fennah (94). He expressed the

exposures in terms of environmental levels rather than in dosage units. The exposures were clearly higher than those ordinarily encountered. In one test, lasting a total of 11.5 months, Fennah daily inhaled 100 mg of pure DDT and drank water dusted at the rate of 3240 mg/m^2. Much of the inhaled dust must have been deposited in the upper respiratory tract and swallowed. Later, for one month Fennah ate food all of which had been sprayed at the rate of 2160 mg/m^2 after it had been served. No ill effect of any kind was observed.

DERMAL EXPOSURE:

The oral dosage necessary to produce any clinical effect was almost always 10 mg/kg or more. In two studies involving only three subjects in all, experimental dermal exposure to DDT was followed by fatigue, aching of the limbs, axiety or irritability, and other subjective complaints. Recovery was delayed a month or more (50, 298). In another study no toxic or irritant effect was reported in six volunteers (67).

With the exceptions mentioned above, dermal exposure of DDT has been associated with no illness and usually no irritation (44, 54, 67, 84, 85, 94, 115, 286). The subcutaneous injection of colloidal suspensions of DDT in saline in concentrations upto 30 ppm caused no irritation (127, 310). The DDT impregnated clothing causes slight, transient dermatitis (310). Other more thorouth studies of DDT impregnated clothings have found it non-irritating (44, 83).

Small pads impregnated with different formulations of DDT to the inner surface of the forearm of 32 volunteers whose cutaneous sensation has previously been measured for

a period of five weeks. Pads impregnated with all the elements of the formulation except DDT were applied to the corresponding position on the other arm as a control. Powdered DDT and 5 % solutions of DDT showed little effect (54).

RESPIRATORY EXPOSURE:

The DDT deposited on the nasal vibrissae of the volunteers had produced moderate irritation of the nose, throat and eyes (195). A slight odour and some dryness of the throat was reported in volunteers exposed to DDT dispersed into the air either by volatilizing units or by aerosol dispensers(258).

2.5.3 *Studies on general population: Accidents and suicides*

Several studies were performed on cases of accidental and suicidal poisoning caused by DDT. The earliest symptom of poisoning by DDT was hyperesthesia of the mouth and lower part of the face. This was followed by the paraesthesia of the same area and of the tongue and then by dizziness, an objective disturbance of equlibrium, parasthesis and tremors of the extremities, confusion, malaise, headache, fatigue and delayed vomiting. The vomiting was probably of central origin and not due to local irritation. Convulsions occurred only in severe poisoning cases. Onset may be as soon as 30 minutes after ingestion of large dose or as late as six hours after smaller but still toxic doses. Recovery from mild poisoning completed in 24 hours, but recovery from severe poisoning required several days (295).

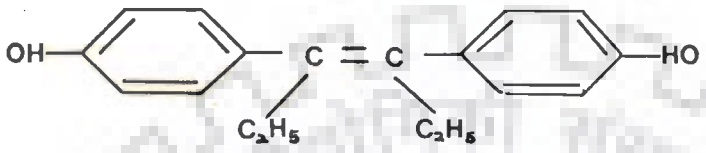
The effect of DDT on human heart such as palpitations, tachycardia and irregular heart actions had been noticed in some but not all cases of acute poisoning (176, 131, 193).

2.6 Effects on reproduction

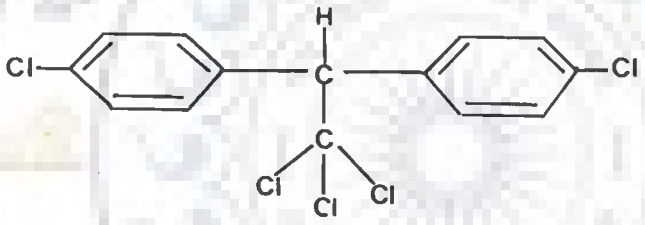
The uncontrolled usage of organochlorine insecticides in agriculture, forestry and public health has resulted in considerable residues of these materials and their metabolites in wildlife and humans. The lower doses can produce many sublethal effects. The DDT related compounds are reported to be capable of causing reproductive abnormalities at levels ranging as low as part per billion. The menstrual cycles become irregular and in most instances prolonged. There is prolonged and excessive haemorrhage during menstruation. These changes are associated with reduced conception rates as well as high instance of early abortion usually following extremely heavy post conceptional bleeding. All these alterations are thought to be related to the changes in the sex hormones. The serum progesterone peak is flattened and lengthened in most cases. Animals showing no alterations in serum estradiol and progesterone experienced none of the reproductive problems with organochlorine pesticides. However, in the animals with the most dramatic changes in the sex hormones, the reproductive alterations were most severe and in some instances progressed to an ovulatory stage. Alterations in levels of enzymes involved in the breakdown of these hormones may, at least in part, be involved (31).

DDT have been shown to stimulate the activity of enzyme responsible for metabolising steroids such as estradiol and endosterone (201). DDT have been shown to be estrogenic. Burlington and Lindeman (41) were the first to suggest that DDT might produce estrogenic effects. Their hypothesis was

that since the molecular configuration of DDT molecule was similar to that of synthetic estrogen diethylstilboestrol it might elicit the same response in the animal.



Diethylstilboesterol



Ortho-para'-DDT

The o,p'-DDT and p,p'-DDT isomers of DDT were tested separately on ovariectomized rats (174). o,p'-DDT exerted an estrogen like effect by increasing the wet weight of the uterus, while p,p'-DDT was only weakly active. These results were confirmed in another study (29) which also indicated increase in weight, water content, glycogen and RNA in the rat uterus after administration of o,p'-DDT; p,p'-DDT exhibited only slight activity. The pure o,p'-DDT or p,p'-DDT as well as technical DDT significantly increased the uterine weight in rats when injected as a single dose (269). The estrogenic activity of o,p'-DDT approaches 10^{-4} of that of estradiol (52). Administration of o,p'-DDT to immature female

rats produce a maximum increase in uterine weight and DNA synthesis at a dose of 250-1000 mg/kg. These responses were specific for o,p'-DDT since p,p'-DDT was much less potent(133).

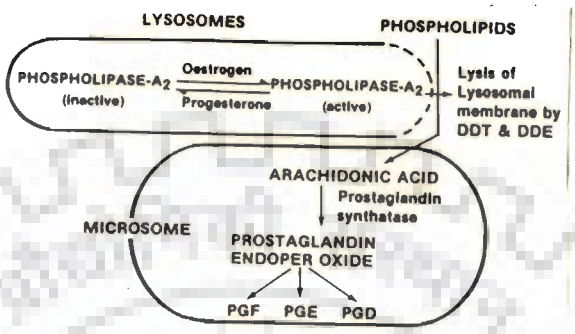
17-β-estradiol and o,p'-DDT produced increase in total uterine DNA content and total uterine protein content. The uterine responses to maximum doses of 17-β-estradiol and o,p'-DDT were not additive, suggesting that both interact with the same receptor. Thus, DDT is thought to produce estrogenic response as a result of its interaction with specific estrogen receptors in target tissues, since o,p'-DDT competitively inhibits the binding of estradiol to the uterine receptor itself; and DDT isomers e.g. p,p'-DDT, which are poor inhibitors of receptor hormone bindings are only weakly estrogenic (168). In another study (259), it was reported that o,p'-DDT stimulates the production of a specific uterine protein, the so called induced protein (IP),normally associated with an estrogenic response of the uterus. In vivo, stimulation of IP production was observed one hour after administration of 250 mg/kg of o,p'-DDT to immature rats. In vitro,stimulation of IP production was observed after one hour incubation of uteri with 100 um o,p'-DDT. This in vitro, response was blocked by actinomycin D. In contrast to o,p'-DDT, which binds to the cytoplasmic estrogen receptor and stimulates IP production, p,p'-DDT which does not bind well to the estrogen receptor does not stimulate IP production in vitro.

The estrogens are thought to produce their effects by combining with specific receptors present in the cytoplasm of target cell. The resultant hormone receptor complex then

migrates to the nucleus where they interact with nuclear acceptor site to initiate changes in transcription which eventually lead to estrogenic effects in the target tissue. It is the quantity of the estrogen receptor translocated to the nucleus that thus correlates with the biological response of the hormone. It was reported that administration of o,p'-DDT to female rats causes a translocation of estrogen receptors to uterine nuclei which is maximum 3 hours after pesticide treatment and a subsequent increase in uterine weight at 24 hours (236). A high degree of correlation (r = 0.98) exists between the level of nuclear estrogen receptor and increase in uterine weight measured 3 and 24 hours, respectively, after administration of o,p'-DDT, in the dose range of 10-1000 mg/kg. This high dose correlation between these two parameters lands strong support to the hypothesis that the estrogenic effects observed following in vivo administration of o,p'-DDT results from interaction of the pesticide with the classical estrogenic receptor system (236).

The presence of prostaglandins in the decidusa was reported to be closely associated with the inhibition of labour, and they are known to be released at the time of labour (275). The biosynthesis of prostaglandin is governed by phospholipid A_2 present in lysosomes through the release of arachidonic acid - a precursor of prostaglandin (241). DDT and DDE are reported to cause labilization of the lysosomes (238). It was presumed that higher levels of DDT and DDE may cause the lysis of sysosomal membrane, thereby releasing the phospholipase A_2, a rate limiting factor in the bio-synthesis of prostaglandin and thus may help affect the onset

of labour (246). The proposed hypothesis is shown below:



Dinerman and Rozhdestvenskaya (81) reported extensive haemorrhages and necrosis and absence of glycogen and RNA in the placenta of rats given DDT, 170 ng/kg body weight per day on day 9 and 10 of pregnancy. These placental lesions may interfere with the normal activity of this organ and upset hormonal synthesis, nutrient supply, and/or other processes indispensible to the maintenance of pregnancy homeostasis. Several reports on effects of DDT on terrestrial mammals are available. No effect on fecundity (i.e. total number of young per producing pair was reported in rats, but rats fed diets containing 50, 100 or 600 ppm diet, DDT showed a progressive decline in the percentage of young successfully weaned, as compared with rats fed diets containing 0 or 100 ppm DDT(97). It was suggested that this mortality of suckling rats or dams fed 50 or 100 ppm, although greater than mortality in controls, was no greater than the normal mortality in this

age group in many laboratories. Also, 600 ppm in a toxic level for female rats and DDT would be secreted in milk at a high level, the reported results at this dose-rate may indicate solely a general systemic toxicity rather than an effect on reproduction (122).

In a long term study (207) the conclusion that DDT has little effect on the success of breeding rats was supported. Dose rates of 0, 20 and 200 ppm technical grade DDT in the diet were begun with a weaning parent generation and terminated with the weaning of the third litters by the F_1 generation. DDT had no apparent effect on the fecundity of the dams, litter size, weight at birth or viability of the young. Comparison of total numbers of pregnancies indicated that DDT had no effect on fertility either. Growth rates were similar at all dose rates and DDT may actually increase reproductive performance in 52 week old rats by exerting some protective effect against the normal age of decrement in reproduction.

Balb/c and C_3H strains of male and female mice were fed 100 ppm DDT for two years. The interstial cell carcinomas of testis was reported in males and varied from well differentiated to poorly differentiated and undifferentiated and were capable of metastasis (234).

The action of o,p'-DDT was studied on plasma steroids and steroidogenesis in adrenal and brain tissues using 26 sprague dawley adult male rats. A decrease in testosterone and an increase in estradiol were found in plasma of rats treated with 20 mg of o,p'-DDT in 0.5 ml cesame oil. Apart from progesterone and in unchanged progesterone in the adrenal

glands, an increase in dihydrotestosterone and a decrease in androstenediol formation from testosterone in the brain were reported. The decrease in androgen biosynthesis was evident from the lower level of plasma testosterone. The decreased plasma estradiol level indicated binding of o,p'-DDT to estradiol receptor sites and decreased 11^{B}-hydroxylase activity was evident from the lower amount of corticosterone formed from progesterone in adrenals (106).

Significant elevation in the metabolism of testosterone and aminopyrine by liver microsomes was reported from 2-10 days old male rats having 10 days prenatal and variable post-natal exposure to DDT via maternal milk secretions. There were no compensatory changes in either serum androgens or testicular synthesis of testosterone (72).

The mouse appears to be more sensitive than the rats and show some detrimental effects on reproductive success. Lower dose rate, 7 ppm was used on two separate strains(282). The first strain showed some reduction in fertility (55.4 % compared to 67 % in control) but no effect on fecundity or litter size. However, a repeat of this test did not confirm this reduction in fertility. Another test, with another strain, showed little difference in fertility, fecundity or litter size of controls and dosed animals. Examination of the number of days to produce the first litter showed a delay in both tests with the first strain but with the second(282). Even lower dose rate (2.8-3.0 ppm DDT) corresponding to a daily intake of 0.4-0.7 mg/kg body weight showed no difference fertility, fecundity, litter size or numbers of surviving weanlings in a five generation study (269).

The female rats were found to reproduce normally when fed DDT for two generations at dietary levels of as high as 200 ppm (207). At a dietary level of 20 ppm the dams had a significantly longer reproductive life span (14.55 months) than did their littermate controls (8.91 months); the number of females becoming pregnant after the age of 17 months and the number of successful pregnancies after that age were significantly different in the two groups (207).

It was reported that o,p'-DDT significantly advances puberty, induces persistent vaginal estrus after a period of normal estrus cycle, and causes other reproductive abnormalities in female rats. The abnormal effects were obtained initially by injecting 1 mg of the o,p'-DDT subcutaneously on the 2nd, 3rd and 4th days of life (counting the days of birth as zero). Because rat pups on the 3rd day weigh about 12 gm or less each, it follows that the subcutaneous dosage was about 83.3 mg/kg/day or more, that is, about 40 times greater than the highest oral dosage of o,p'-isomer fed to breeding rats and about 10^5 times greater than what ordinary people get in their food (126).

In a six-generation test of reproduction in mice (152) was reported that there was no effect of DDT at a dietary level of 25 ppm on fertility, gestation, viability, lactation, and survival. A level of 100 ppm produced a slight reduction in lactation and survival in some generations but not all, and the effect was not progressive. A level of 250 ppm was distinctly injurious to reproduction. The dietary concentrations were used to determine dosage of 3.33, 13,3, and 33.2 mg/kg/day in nonpregnant, nonlactating, adult female mice.

The intake was much higher in both young and lactating mice.

The p,p'-DDT administered to pregnant mice at a rate of 1 mg/kg on days 10, 12 and 17 of gestation was not teratogenic but did alter the gonads and decreased the fertility of the young especially the females (181). A single dose of DDT at the rate of 25 mg/kg or repeated doses of 2.5 mg/kg/day given during pregnancy may be embryotoxic but not teratogenic to mice (248).

The effect of DDT on the length of the estrus cycle was studied. It was found that injection of 40 mg/kg DDT significantly prolonged the cycle by about a day. This implies less frequent period of sexual receptivity in the female mice and caused a decline in reproductive capacity.

The reproductive functions following prenatal exposure to pesticides were studied (255). Rats were either injected prenatally (15-21 days of gestation) or neonatally (1-6 days of age) with o,p'-DDT (100 mg/kg body weight in corn oil) to study effects upon estradiol 17B (E_2) cytosol receptor binding in uterus and testis as adults. Prenatal administration of DDT produced a constant estrus cycle in some animals. Pre and postnatal exposure significantly decreased E_2 binding to uterine cytosolic proteins by 18 % over controls. Results suggested that perinatal exposure to o,p'-DDT may lead to permanent estrogenic effects by interfering with estrogen binding in the reproductive organ. o,p'-DDT may have either altered the neuroendocrine axis early in life or the pesticide and (or metabolites) persisted and competed for binding in the adult reproductive organs.

The effects of DDT on reproduction was studied in dogs.

Four female dogs of unstated age that previously had received DDT at the rate of 12 mg/kg/day, 5 days a week, for 14 months were bred when they went into heat. The males involved had been fed DDT (60 mg/kg/day) plus aldrin (0.15 mg/kg/day) for 14 months prior to breeding but not during breeding. Two of the females went into heat but failed to become pregnant, and one failed to come into heat during 12 months after feeding. Four of six pups born to the fourth female died within 1 week of birth; the other two were weaned successfully even though only two posterior mammal of the mother were functional (76).

In the three-generation study male and female dogs were fed technical DDT from weaning at rates of 0, 1, 5 and 10 mg/kg/day. Observations were made on 135 adult females, 63 adult males, and 650 pups. There were no statistically significant differences among controls and DDT-treated dogs in length of gestation, fertility, success of pregnancy, litter size, or lactation ability of the dams or in viability at birth, survival to weaning, sex distribution, and growth of pups or in morbidity, mortality, organ/body weight ratios, or gross histological abnormalities in all the animals studied. The only clear differences was that DDT-treated females had their first estrous 2 or 3 months earlier than the control dogs. There was a slight increase in liver/body weight ratio in some DDT-treated animals but the difference was not statistically significant, not dosage related, and not associated with any histological change (208).

Organochlorine pesticides have been reported to influence human reproduction. The passage of organochlorine pesticides

through the placenta was established by the detection of these compounds in the tissues of stillborns and cord blood obtained during normal deliveries. Organochlorine compounds were demonstrated in the tissues of stillborn (63, 287), the cord blood of newborn babies (22, 232) and in human placenta (232, 222). In man, there is no indication of that DDT effect reproduction; no impairment of fertility was observed in study of man occupationally exposed for more than 10 years to a measured average daily in the region of 10 mg per man per day (equivalent to 0.25 ng/kg/day). It was also found that 36 most heavily exposed workers involved had fathered 58 children before they began working at the DDT factory and 98 children afterwards (299).

In females, accumulation of organochlorine residues in blood and then their subsequent transfer to the fetus through placenta during pregnancy, and their excretion through breast milk and its consumption by the neonates posed varies problems of management of neonatal nutrition and health.

In U.S.A. (250), 53 paired samples of placenta and maternal blood collected from general population were analysed for DDT and its metabolites. The placental and maternal blood samples were reported to contain DDT and its metabolites. No marked difference were observed among residence, age or race. The levels were higher in the placental specimens than in blood.

The DDT and its metabolites were analysed in adipose tissue, liver, adrenals, lungs, heart, brain, kidneys, and spleen of 10 human infants, 8 of whom were stillborn and 2 who died after birth. Samples of spinal cord and pancreas

from 3 of the stillborn fetuses were also analyzed. The DDT and its metabolites were reported in various tissues analyzed and the ranges fell within the range of concentrations that have been found in various tissues and blood of the general adult population. In a separate study cord blood of 30 normal term babies were analyzed. The mean total DDT was found to be 0.0144 ppm and the metabolites reported were p,p'-DDT, o,p'-DDT, p,p'-DDE, o,p'-DDE and p,p'-DDD (63).

The concentration of DDT-related compounds in various tissues of women at the time of caesarean section or normal delivery was reported to be less in nonpregnant women in the same community (222).

The lower concentration of several organochlorine insecticides and some of their metabolites found in adipose tissue of pregnant women suggests a more active metabolism of these compounds during pregnancy. All the metabolites of DDT found in the adipose tissue of pregnant women were present in both maternal as well as in umbilical cord blood in all the cases studied. Thus, the crossing of the placental barrier by organochlorine insecticides can be concluded. The concentration of organochlorine insecticides was generally lower in fetal blood than in maternal blood. It was reported that the women who aborted contained high levels of DDT and no relationship was established between age or parity(202).

Storage of organochlorine compounds in mother and fetus during labour was investigated (223). DDT and its metabolites were assessed in maternal adipose tissue, maternal blood, fetal blood, maternal uterine muscle, placenta and amniotic fluid. The concentration of DDT was greater in the

extracted lipid of fetal blood than in the maternal blood and still higher in the uterine muscles. The metabolites of DDT were found in different ratios in mother and fetus. The percentage of p,p'-DDE related to total DDT was 51.51 for fetal plasma, 62.20 for maternal plasma, 68.81 for adipose tissue, 58.98 for the placenta and 78.79 for the uterus. The metabolization of p,p'-DDT to p,p'-DDE in the uterus resembled that in the adipose tissue, whereas placental values were nearer the figures obtained for fetal blood. The same was observed when metabolization of o,p'-DDT to o,p'-DDE was compared in uterus and placenta i.e. metabolization of o,p'-DDT was more acute and/or storage of o,p'-DDE was higher in the uterus than in the placenta.

Serum and cord blood samples of 35 mothers were reported to contain p,p'-DDE at a concentration of 18 ug/litre and 6.8 µg/litre respectively (151). Serum samples of 24 non-pregnant women contained same amount of p,p'-DDE (20 µg/litre) as mother's serum.

The levels of DDT and its metabolites in toxemia of pregnancy were investigated (290). The mean total DDT serum level was highest in the high PCB levels toxemia group (28.6 ppb) in comparison with the low PCB levels toxemia group (22.7 ppb) and the controls (26.2 ppb). These differences were statistically significant. The p,p'-DDT/p,p'-DDE ratio was lowest in the high PCB levels toxemia group, 1.59, in comparison with the low PCB levels toxemia group, 2.47, and the controls, 3.46. The same relationship was found for serum o,p'-DDT/o,p'-DDE ratio, which was 1.21, 3.83 and 3.90 for the high and low PCB levels toxemic groups and controls respectively. A lower serum DDT/DDE ratio, that is higher

p,p'- and o,p'-DDE values might mean an increase degradation of p,p'- and o,p'-DDT to p,p'-and o,p'-DDT to p,p'-and o,p'-DDE, respectively, and for a decrease excretion of p,p'-and o,p'-DDE.

An association between high DDT cord blood levels and prematurity was investigated (225). No difference in DDT levels was observed between pregnant women of full term and premature deliveries. There was a significant difference in DDT cord blood levels between term and preterm infants and between prematurity and high cord blood levels in both groups. DDT cord blood levels correlated negatively with infants birth weights. The authors concluded that a cause effect association between high DDT cord blood level and prematurity could not be ruled out.

In another study levels of DDT were assessed in 17 cases of premature deliveries (291). 5 out of 17 cases of premature deliveries (PD) were associated with high DDT serum levels (119.6 ppb versus 26.5 ppb in control group). The percentage of total o,p'-DDT levels was usually high (50 % of the total DDT in the study group versus 30 % in the control group).

In women with recent missed abortions (25) increased level of o,p'-DDT was reported. The percentage of total o,p'-DDT serum levels as related to total DDT serum levels, showed an increase in the missed abortion groups, 33.9 and 36.2, respectively, and in the former missed abortion group, 40.0 versus 25.6 in the controls.

In India, DDT residues in pregnant women were studied to indicate storage and transfer of DDT to prenates via placenta and to neonates via milk. DDT residues were detected in 50

specimens of placenta and accompanying fluid but no correlation could be found between the compound and age, race and residence of women (244). The umbilical cord blood collected during labour from 100 women was reported to contain significant levels of p,p'-DDT and its metabolites (245). The residues in umbilical cord blood (neonatal blood) were related to age, dietetic habits and area of residence of mothers(245).

In another study, nursing Indian women (251), mean residue levels of total DDT in milk were 0.127 ± 0.019; in maternal blood 0.020 ± 0.002 ppm and in umbilical cord blood 0.015 ± 0.001 ppm. The site of preferential accumulation of DDT in the body was milk > maternal blood > cord blood. No effect of social status, age, dietetic habit or area of residence on the susceptibility of subjects examined to accumulate organochlorines was noticed. The presence of DDT and its metabolites in the cord blood indicated their passage across the placenta to prenates, whereas contamination of breast milk light on a far greater daily intake of toxic organochemicals.

Considerably higher amounts of organochlorine pesticides residues were reported in the circulating blood and placental tissue of Indian women undergoing abortion and premature labour as compared to pregnant women in full term labour(243). The mean concentration of DDT in maternal blood of cases of premature labour and abortions was 250.6 and 25.3 ppb, respectively. The mean residue level of DDT in placental tissue and for controls were 117.2 and 22.2 ppb.

The DDT and its metabolites were analyzed in blood,

placenta and fetus of Indian women experiencing spontaneous abortions, preterm labour or full term labour (246). The ratio for p,p'-DDE and total DDT was found to be highest in the abortive subjects, followed by preterm and the lowest in full term cases. It was suggested that organochlorine insecticides act as antagonists to pregnancy.

Recently the quantification of DDT and its metabolites in specimens of maternal blood, placenta and umbilical cord blood from Indian women experiencing still birth and life birth was done (247). The specimens of still birth cases had higher total DDT residues as compared to matched controls A significant correlation was noted between maternal blood and placenta and maternal blood and umbilical cord blood for total DDT.

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C. Laboratory tests

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3

MATERIALS AND METHODS

A. Determination of DDT residues

3.1 *Cleaning of laboratory glassware*

Glassware was cleaned by the method described in EPA manual (EPA, 1980). The basic cleaning steps were:

a) Removal of surface residuals immediately after use.

b) Hot soak to loosen and floatate most of the soil.

c) Hot water rinse to flush away floatated soil.

d) Soak with deep penetrated or oxidizing agent to destroy traces of organic soil.

e) Hot water rinse to flush away materials loosened by deep penetrant soak.

f) Distilled water rinse to remove metallic deposit from the tap water.

g) Acetone rinse to flush off any final traces of organic material.

h) A preliminary flush of the glassware just before use with the same solvent to be used in the analysis.

3.2 *Sample collection*

3.2.1 *Soil:*

The soil samples were collected from 50 selected sites (Fig.4) in Delhi during October to November, 1983. Soil samples were collected with a core soil sampler which is a stainless steel tube 30 mm in diameter. The sampler was pushed into the soil to take a sample about 15 cms in depth. On removal the core was separated into two portions and only the upper 5 cm sample was taken. Normally 10 such cores were taken from each site covering long distances between each other. The samples were mixed thoroughly and transported to the laboratory in pre-

washed glass bottles. Physical and chemical properties of soil were not studied.

3.2.2 *Human tissues and secretions*

Selection of subjects:

The subjects were drawn from rural and urban population of Delhi and its nearby areas. Subject from general population had no history of accidental or occupational exposure to pesticides. However, they were asked to fill out a detailed questionnaire relating to age, dietetic habits, ethnicity (rural/urban), exposure to chemicals, reproductive history and other clinical symptoms to explore DDT intoxication. To avoid malnutrition, which is one of the factors for premature labour, women with hemoglobin level within the normal range, 9-12 g % were included.

3.2.2.1 *Blood of subjects from general population:*

Fifty healthy volunteers were studied from 5 zones of Delhi and nearby areas. They were between the ages of 13 to 48 years; 31 were males and 19 were females.

Circulating blood (10 ml) was collected in heparinized vials (20 ml glass tube containing 200 USP units of heparin in 0.2 ml solution) by venepuncture and transported to laboratory in ice buckets.

3.2.2.2 *Tissues and secretions of subjects with full term pregnancy:*

Fifty pregnant women, admitted to Obstetrics and Gynaecology Department of All-India Institute of Medical Sciences, New Delhi were studied. Women were between the ages of 18 to 30 years, and had full term labour, caesarian or normal.

Circulating maternal blood (10 ml) was collected 8 to 12 hrs before parturition by venepuncture in heparinized vials.

Placental tissue (10 gm) was collected in acetone washed aluminium foil at the time of labour. Umbilical cord blood (10 ml) was collected by milking the cord into heparinized vials at the time of labour. Milk (5 ml) was collected by manual expression, 48 hrs after parturition.

3.2.2.3 *Tissue of subjects with premature labour and toxemia:*

Fifty pregnant women, admitted to Obstretrics and Gynaecology Department of All-India Institute of Medical Sciences, New Delhi were studied. Ten women were admitted in preterm labour and 40 cases of toxemia of pregnancy were studied.

Circulating maternal blood (10 ml) was collected by venepuncture in heparinized vials at the time of labour. Placental tissue (10 gm) was collected in acetone washed aluminium foil. Umbilical cord blood (10 ml) was collected by milking the cord into heparinized vials at the time of labour.

3.2.2.4 *Blood and uterine tissue of cadavers:*

Twenty-five unembalmed cadavers killed by traumatic injury during May to October, 1983 were studied. Women were between the ages of 11 to 40 years. Blood (10 ml) was collected from the heart in a clean glass vial at the time of autopsy. Uterine tissue (10 gm) was also collected at the same time in acetone washed aluminium foil.

3.2.2.5 *Blood and uterine tissues of subjects undergoing hysterectomy:*

Twenty-five women undergoing hysterectomy in All-India Institute of Medical Sciences, New Delhi were studied. Women were between the ages of 26 to 55 years.

Blood (10 ml) was collected by venepuncture in heparinized

vials. Uterine tissue (10 gm) was collected at the time of hysterectomy in acetone washed aluminium foil.

3.3 *Sample storage*

All the collected samples were immediately kept in ice buckets and transported to laboratory as soon as possible to store in a deep freezer at -4°C until analyzed, generally within 48 hrs. No fixative or preservative were used. Blood samples collected for laboratory analyses in unheparinized vials were stored after separating the serum.

3.4 *Extraction procedures*

3.4.1 *Soil:*

Soil samples (5 to 10 gm) were moistened and mixed with equal weight of anhydrous sodium sulphate and ground in a mortar and pestle. This was mixed with acetone:hexane (59:41 v/v) and stirred on a magnetic stirrer in a 100 ml conical flask for 1.5 hrs and filtered. The extraction was repeated two more times with fresh volumes of the solvent mixture. The total volume of the solvent used was 100 ml. The pooled extract from the soil was washed with 100 ml of 2 % sodium sulphate in a 500 ml separatory funnel and the hexane layer formed after the separation was filtered through anhydrous sodium sulphate. The hexane extract was concentrated on a rotary evaporator. Normally three replicates were run simultaneously.

3.4.2 *Human tissues and secretions:*

Human tissues (uterus and placenta) were cut into small pieces and accurately weighed 2 gm tissue was ground in a mortar and pestle with four times their weight of anhydrous sodium sulphate. Blood and milk (5 ml) were taken into a 100 ml

conical flask containing acetone washed glass wool. Samples were mixed with acetone:hexane (1:1 v/v). The mixture was stirred for one hour on a magnetic stirrer and filtered. The extraction was repeated two more times with fresh volumes of the solvent mixture and a total of 100 ml of the solvent was used for the samples weighing upto 2 gms. The extract was washed with 100 ml of 2 % sodium sulphate in a 500 ml separatory funnel and the hexane layer formed after the separation was filtered through anhydrous sodium sulphate. The hexane extract was concentrated on a rotary evaporator. Normally three replicates were run simultaneously.

3.5 *Clean up procedures*

The crude extract obtained from the above sample contains lipids which may contaminate glc column and detector. The removal of lipids from the extracts is referred here as clean-up. The clean up was done using only one of the following methods.

3.5.1 *Sulphuric acid clean up:*

The crude extract (5 to 10 ml) was shaken with 1 ml of concentrated sulphuric acid for 30 seconds in a 100 ml conical flask. The hexane phase was separated and further washed with fresh concentrated sulphuric acid, two times, to remove the fat content. The cleaned hexane was collected and concentrated for gas liquid chromatography.

3.5.2 *Chromatography on alumina:*

The alumina column chromatography was done using method described by Holden and Marsden (1969). Aluminium oxide standardized for chromatography was partially deactivated by

adding 5 % distilled water, shaking for about 20 minutes and storing in a closed vessel. The chromatography was carried out in a 200 mm X 15 mm (Length X Internal diameter) glass column. A small wad of glass wool pre-extracted with petroleum ether or hexane was placed at the bottom of the column to retain the alumina. The column was filled with hexane and then packed with alumina (6 gms) through a narrow funnel with constant tapping of the column. This was done to avoid trapping of air bubbles. At the top, alumina was covered with about 3 cms of anhydrous sodium sulphate. The column was prewashed with 100 ml of hexane and prevented from drying by allowing the solvent to remain 1 to 2 cms above the sodium sulphate layer. About 1 ml concentrated extract was introduced on the top of the column using Mohr or long disposable pipettes and allowed to sink into the column. The evaporator tube containing sample was rinsed with 2 ml of hexane and rinsings were added to the column successively as soon as the previous one was absorbed. This was followed by the elution using 50 ml of hexane at approximately 5 ml/min. The effluent was collected in a 100 ml evaporator assembly and concentrated. The elutions were carried out at room temperature.

3.6 *Detection and quantitation of residues*

This was carried out by:

a) Gas liquid chromatography, and

b) Thin layer chromatography.

3.6.1 *Gas liquid chromatography:*

Qualitative and quantitative analyses of residues were carried out by a Packard A 7400 series Gas chromatograph equipped with an electron capture detector. The details of the columns and

Table-1.

COLUMN SPECIFICATIONS AND OPERATING CONDITIONS OF PACKARD A-7300 GAS LIQUID CHROMATOGRAPH

	COLUMNS		
	I	II	III
TYPE OF DETECTOR	Electron capture	Electron capture	Electron capture
COLUMN LENGTH	6 feet	2 meters	2.5 meters
INTERNAL DIAMETER	4 mm	2 mm	2 mm
COATING MATERIAL	3 % SE_{30}	1.5 % OV_{17} & 1.95 % QF_1	5 % DEGS
SOLID SUPPORT	GAS CHROM Q	GAS CHROM Q	GAS CHROM Q
MESH SIZE	100 - 120	100 - 120	100 - 120
COLUMN TEMPERATURE	220°C	190°C	180°C
DETECTOR TEMPERATURE	230°C	210°C	190°C
INLET TEMPERATURE	230°C	210°C	190°C
CARRIER GAS	NITROGEN	NITROGEN	NITROGEN
FLOW RATE	60 ml/min	60 ml/min	75 ml/min
SAMPLE SIZE	3-5 ul	3-5 ul	3-5 ul

operating conditions are given in Table 1.

Aliquots were injected into a gas chromatograph with the working parameters described in Table 1 with the help of a 10 ul Hamilton syringe. The peaks in the samples were identified by comparing their retention time with standards (Fig.1, 2 and 3). Quantitation was made on the basis of peak heights. In order to keep track of the correct retention time and detector response, standards were run before and after all analyses. Blanks were also run after every tenth sample. Every tenth sample was a duplicate and was spiked to ensure valid recovery.

The retention time of standard in three columns is given in Table 2. Column II was used for routine work while other columns were used for confirmation of DDT residues.

Table-2.

RETENTION TIME OF ORGANOCHLORINE INSECTICIDES (in minutes) IN A STANDARD MIXTURE USING THREE DIFFERENT COLUMNS

PESTICIDE/METABOLITES	COLUMNS		
	I	II	III
p,p'-DDE	3.70	8.25	6.75
o,p'-DDT	4.70	12.00	9.75
p,p'-DDD	4.70	13.50	24.75
p,p'-DDT	6.00	16.35	19.35

3.6.2 ***Thin layer chromatography:***

Clean glass plates (200 X 200 mm) rinsed with acetone were coated with a 250 μ thick layer of silica gel G with 50 ml of distilled water and shaking for 55 seconds in a closed

Fig.1 Gas chromatogram of a mixture of standard DDT and its metabolites and lindane on the column containing 1.5 % OV_{17} and 1.95 % QF_1 on Gas Chrom Q 100-120 mesh. Further details of operating conditions are given in Table 1.

Peak No.	1	Lindane
	2	Kelthane
	3	DDMU/o,p'-DDE
	4	p,p'-DDE
	5	o,p'-DDT
	6	p,p'-DDD
	7	p,p'-DDT

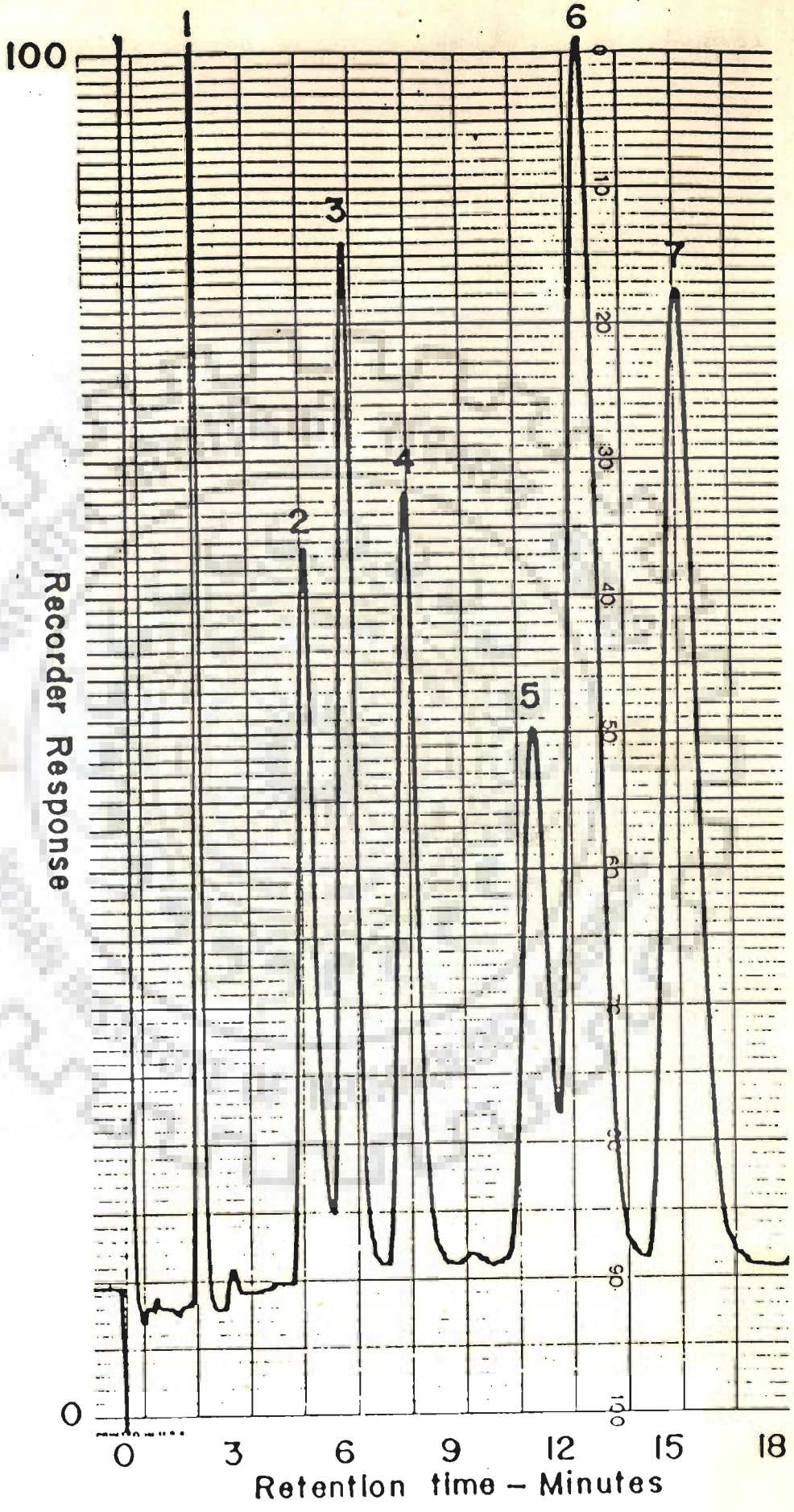
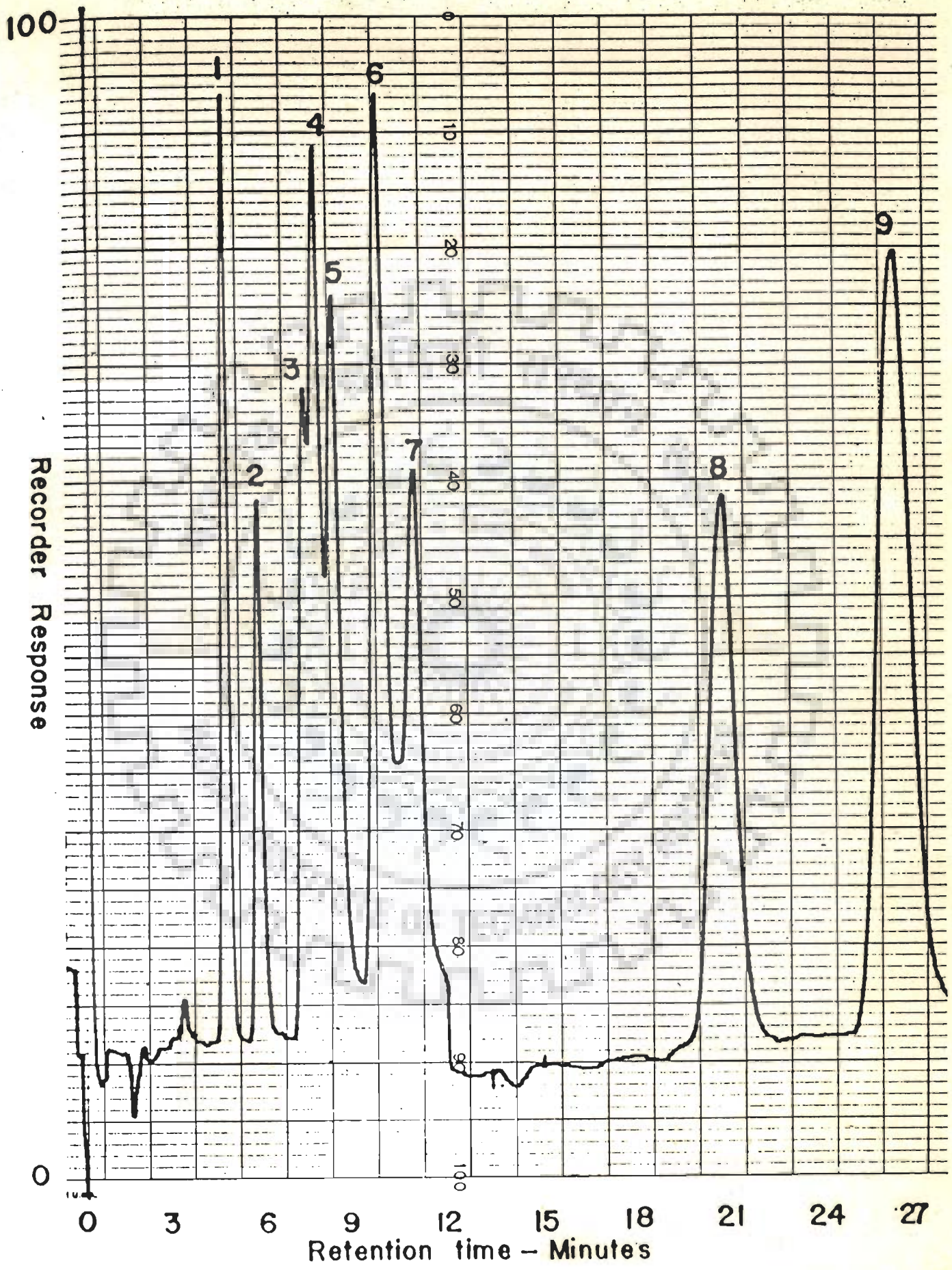

100
0
1
2
3
4
5
6
7
Recorder Response
0
3
6
9
12
15
18
Retention time – Minutes

Fig.2 Gas chromatogram of a mixture of standard DDT and its metabolites, lindane and dieldrin on the column containing 5 % DEGS on Gas Chrom Q 100-120 mesh. Further details of operating conditions are given in Table 1.

Peak No.	1	Lindane
	2	o,p'-DDE
	3	DDMU
	4	p,p'-DDE
	5	Kelthane
	6	Dieldrin
	7	o,p'-DDT
	8	p,p'-DDT
	9	p,p'-DDD


100
0
Recorder Response
1
2
3
4
5
6
7
8
9
0
10
20
30
40
50
60
70
80
90
100
0
3
6
9
12
15
18
21
24
27
Retention time – Minutes


100
2
1
3
Recorder Response
0
5
10
15
Retention Time min.

Fig.3 Gas chromatogram of a mixture of standard DDT and its metabolites on the column containing 3 % SE_{30} on Gas Chrom Q 100-120 mesh. Further details of operating conditions are given in Table 1.

Peak No. 1 p,p'-DDE

2 p,p'-DDD/o,p'-DDT

3 p,p'-DDT

vessel. A Baird and Tatlock (London) Ltd., motorised TLC coater was used for coating the plates. Subsequently, the plates were dried at ambient temperature and stored in a dessicator for further use. Immediately before use, the plates were activated by heating at 100°C for 90 min. The TLC plates were divided into a number of lanes required for each sample, to prevent their lateral movements. A line was marked at the top of the plate upto which the solvent front was to rise. Aliquots of the cleaned up sample extract were applied on the plate 2 cm from the base of the plate, in the middle of the lanes which were 2 cm wide. The spots on the plate were allowed to dry completely and the plates were transferred to a developing chamber for the vertical development of the chromoplates upto a height of 15 cm from the point of application of the spot. The developing chamber lined with filter paper was loaded with 120 ml of the solvent system sufficiently in advance to ensure complete saturation of the chamber. The plates were developed at the room temperature.

The developed TLC plates were air dried and sprayed with 0.05 per cent rhodamine in ethanol (w/v) and dried. The spots were located under U.V. light, which appeared purple against a pink background. Alternatively, the plates were sprayed with Morley's spray and allowed to dry for 10 to 20 minutes, until dark spots of pesticides appeared on a light grey background.

Spots on the chromatoplates were identified by comparing their Rf values with those of the standard compounds. The Rf values of the organochlorine insecticide residues were calculated as the observed movement of the spot after several runs divided by the distance of solvent front (15 cm). In two

solvent systems (Hexane:Acetone and Hexane:Chloroform) the organochlorine insecticide residues show different Rf values as shown in Table 3.

Table-3.

Rf VALUES OF ORGANOCHLORINE INSECTICIDES

COMPOUND	HEXANE:ACETONE 98:2	HEXANE:CHLOROFORM 90:10
p,p'-DDT	0.79	0.83
p,p'-DDD	0.39	0.69
p,p'-DDE	0.87	0.89
o,p'-DDT	0.82	0.86

3.7 *Confirmation of residues*

3.7.1 *TLC-GLC PAIRING:*

For extraction of insecticides and their metabolites from the thin layer plates, the lanes for the standard compounds were sprayed with the detecting reagent to reveal their location. The corresponding spots in the unsprayed lanes were marked with reference to the standards and silica gel G from those areas was scraped into a centrifuge tube. Hexane was used for the extraction of insecticide residues from silica gel. The sample was centrifuged and the supernatant transferred to another tube and this process was repeated two more times with fresh volumes of hexane. The pooled hexane extract was concentrated and injected into the gas chromatograph and compared with the standards.

3.7.2 *CHEMICAL CONFIRMATION:*

The identification of p,p'-DDT, p,p'-DDD and o,p'-DDT were

confirmed by dehydrochlorination. It has long been known that p,p'-DDT can be easily dehydrochlorinated with alkali to the corresponding olefin, p,p'-DDE. Similarly, o,p'-DDT can be converted to o,p'-DDE by alkali, however this reaction is considerably slower than the dehydrochlorination of p,p'-DDT. p,p'-DDD can be dehydrochlorinated to DDMU by alkali.

To a concentrated extract (0.1 to 0.2 ml) of the sample in the test tube, was added 1 ml of 0.1 N sodium methoxide/ methanol solution and incubated for 1 hour at 60°C. To this 5 to 6 ml hexane was then added and the mixture was shaken for a few seconds. The hexane layer was removed, concentrated and injected into the glc whereby due to dehydrochlorination, the peaks for DDT appeared as the corresponding DDE and DDD as DDMU respectively.

3.8 *Recovery studies*

The recovery values for DDT and its metabolites from the spiked samples of soil and tissues were determined. The recovery standards were carried through extraction and clean up procedures along with the unspiked samples. The recovery values thus obtained are given in Table 4.

Table-4.

PERCENT RECOVERY OF DDT AND ITS METABOLITES FROM SPIKED SAMPLES

MATERIAL EXTRACTED	SOLVENT USED	p,p'-DDE	o,p'-DDT	p,p'-DDD	p,p'-DDT
SOIL	HEXANE:ACETONE *49:51	105.75 ±2.98	97.00 ±5.51	85.89 ±4.74	98.89 ±7.15
TISSUE	HEXANE:ACETONE *1:1	87.22 ±1.96	96.20 ±10.91	80.92 ±1.56	76.00 ±1.98

* Proportions (v/v) used in extraction procedure.

B. Clinical studies

All subjects underwent routine physical examination and a detailed questionnaire was filled as described in Table 5.

Table-5.

SURVEY PROTOCOL

ITEMS	INFORMATION
Occupational history/ environmental exposure	Past and present occupational history and environmental exposure
Demographic data	Age, sex, weight etc.
Ethnicity	Rural/urban
Dietary habits	Non vegetarian/vegetarian
Past medical history	Past and present medical difficulties, medications, hospitalizations, alcohol use etc.
Reproductive history	Past reproductive history and neonatal health/abnormalities.
Symptomatology	Past and present symptoms grouped by organ systems.
(a) Ear,Nose,Throat	Eye irritation Nasal irritation Sore throat
(b) Respiratory	Cough Wheezing Tightness in chest
(c) Skin	Skin rash Acne Sensitivity to sun Burning sensation of skin Thickening of skin Discoloration or deformation of nails.
(d) Gastrointestinal	Weight loss Appetite loss Nausea Vomiting Abdominal pain Pain related to meals Crampy abdominal pain Constipation, diarrhea

(e) Urinary	Red urine Brown urine
(f) Neurological	Headache Blurred vision Dizziness Depression Tiredness Perceptional changes Nervousness Sleeplessness Muscle weakness Sleepiness Difficulty in walking Parasthesia Loss of balance
(g) Musculoskeletal	Swelling of joints Pain in lower back and legs
(h) Cardiovascular	Blood pressure(high/low) Pain in chest Palpitation of heart
Physical examination(adult)	Complete
Physical examination(neonate)	Complete

C. Laboratory tests

(a) Serum biochemistry:

A broad spectrum of laboratory tests were performed on serum collected from the subjects. The biochemical constituents were analysed using kits supplied by J. Mitra,India (Manufactured in technical collaboration with M/s.Diagnostic Reagents International, Philipines).

Table-6.
NORMAL VALUES FOR THE LABORATORY TESTS

BIOCHEMICAL PARAMETERS	NORMAL VALUES
Glucose	70 - 110 mg/dl (Fasting)
Urea	10 - 46 mg/dl
Total protein	5.5 - 8.0 gm/dl
Albumin	3.5 - 5.0 gm/dl
Cholesterol	150 - 250 mg/dl
Bilirubin (Total)	0 - 1.2 mg/dl
Creatinine	0.5 - 1.2 mg/dl(Females) 0.7 - 1.5 mg/dl(Males)
Alkaline Phosphatase	6 - 14 Units
SGOT	10 - 40 UNITS
SGPT	10 - 53 UNITS

(b) *Haematology:*

Hemoglobin and Erythrocyte Sedimentation Rate (ESR) were done by routinely used tests. Hemoglobin was estimated by the cyanmethemoglobin method and ESR was estimated by Wintrobe's method.

(c) *Urine analyses:*

Complete urine analysis was performed on morning urine samples by standard methods.

D. Statistical methods

Comparisons of DDT levels and of other variables within and between different groups were made using the 't' test and one way analysis of variance followed by multiple range tests after adjusting unequal variances as appropriate. The relationship between residue levels and other parameters were investigated using correlation analysis. All statistical analyses were performed on HCL System II computer.

RESULTS

A. DDT levels

4.1 Residues in soil

4.2 Residues in blood of occupationally unexposed population

4.3 Residues in pregnant and lactating women

4.4 Residues in blood and uterine tissue

B. Clinical data

4.5 Clinical findings among occupationally unexposed population

4.6 Clinical findings among pregnant women

4.7 Clinical findings among women who underwent hysterectomy

C. Laboratory data

4.8 Blood tests

4.9 Hematology

4.10 Urine tests

4

RESULTS

A. DDT levels

The residues of DDT and its metabolites in soil and human tissues were analyzed on a gas liquid chromatograph equipped with an electron capture detector. Confirmation was done using different glc columns, by thin layer chromatography, TLC-GLC pairing and chemical methods.

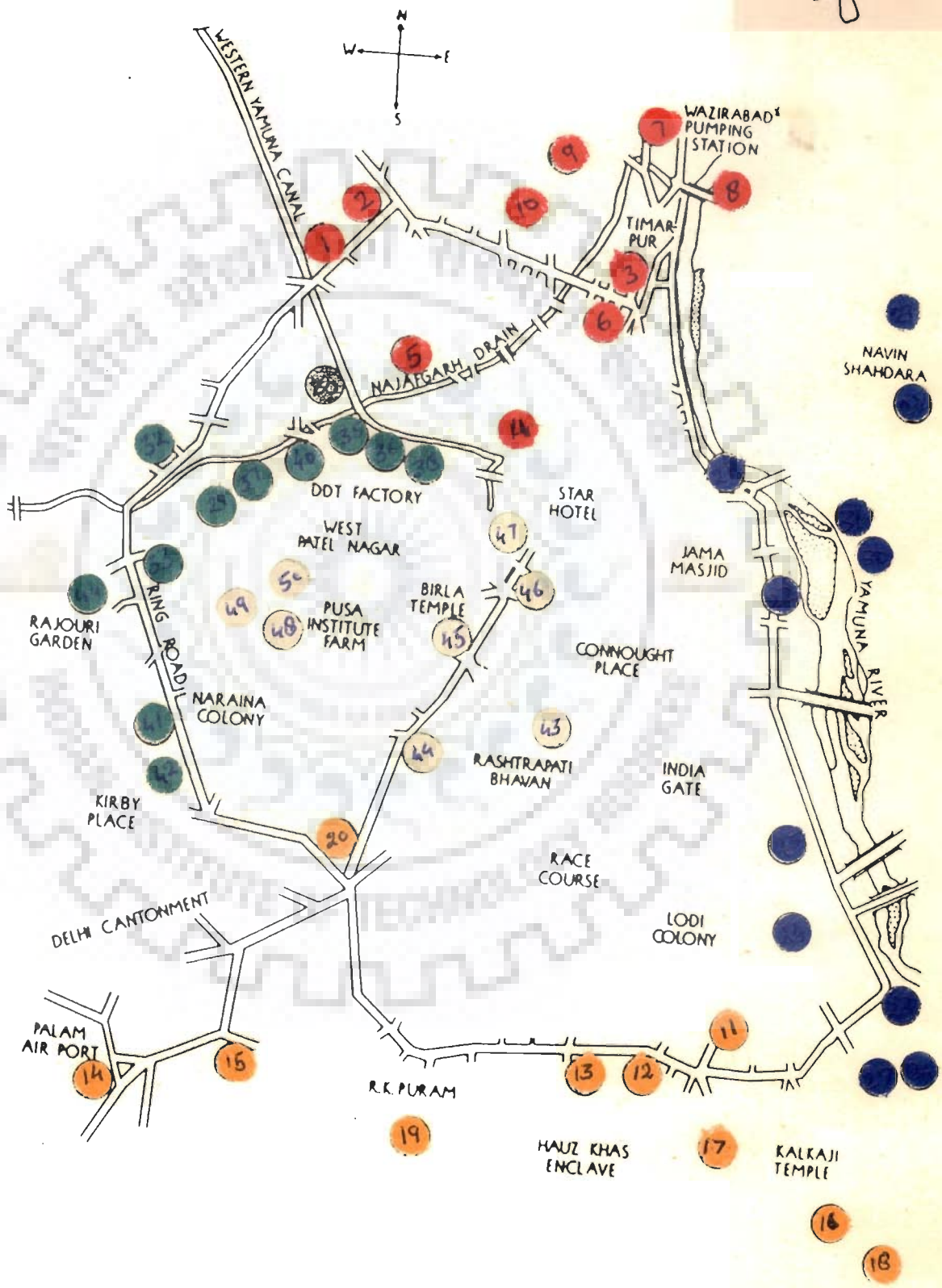
4.1 *Residues in soil*

Monitoring of pesticide residues in soil was conducted during October to November, 1983 from the areas shown in Fig. 4, covering five zones of Delhi. The areas included in this study were near and away from the DDT manufacturing factory situated in West Zone (Site No.40). The East Zone sites were far away from the DDT factory and also included agricultural lands.

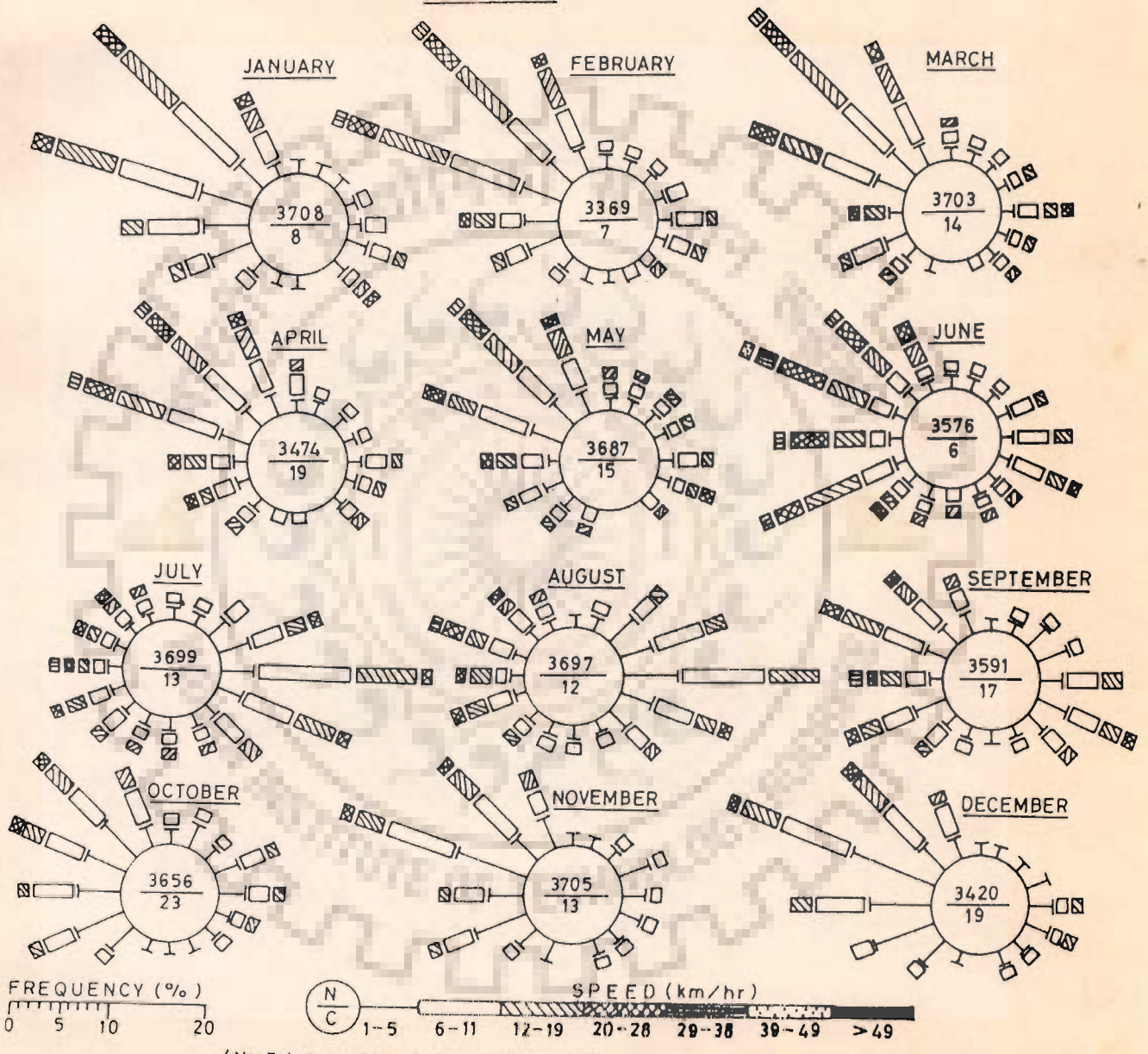
The soil samples subjected to gas chromatographic analysis, resolved into four peaks having retention times identical to p,p'-DDE, p,p'-DDD, o,p'-DDT and p,p'-DDT (Fig.5). The concentration of DDT and its metabolites is given in Table 7. The statistical outcome of the data representing range of concentrations and mean values with standard deviations is summarized in Table 8a. All the 50 soil samples analyzed in this study contained DDT and its metabolites and total DDT ranged from 0.498 to 7.270 ppm with a mean value of 1.670 ppm. The median DDT value was calculated to be 1.223 ppm with a quartile deviation of 1.027 ppm.

It is clear from Table 7 that Durga Nagar, Tilak Nagar, Moti Nagar, IARI fields, Wazirabad water pumping station and Delhi Air Port contained high levels of DDT residues, maximum

Fig.4 Areas sampled for organochlorine insecticide residues in the soil of Delhi.


Delhi
N
W
E
S
WESTERN YAMUNA CANAL
WAZIRABAD PUMPING STATION
TIMAR-PUR
NAJAFGARH DRAIN
NAVIN SHAHDARA
DDT FACTORY
STAR HOTEL
WEST PATEL NAGAR
JAMA MASJID
RAJOURI GARDEN
RING ROAD
PUSA INSTITUTE FARM
BIRLA TEMPLE
YAMUNA RIVER
CONNOUGHT PLACE
NARAINA COLONY
RASHTRAPATI BHAVAN
INDIA GATE
KIRBY PLACE
RACE COURSE
DELHI CANTONMENT
LODI COLONY
PALAM AIR PORT
R.K. PURAM
HAUZ KHAS ENCLAVE
KALKAJI TEMPLE

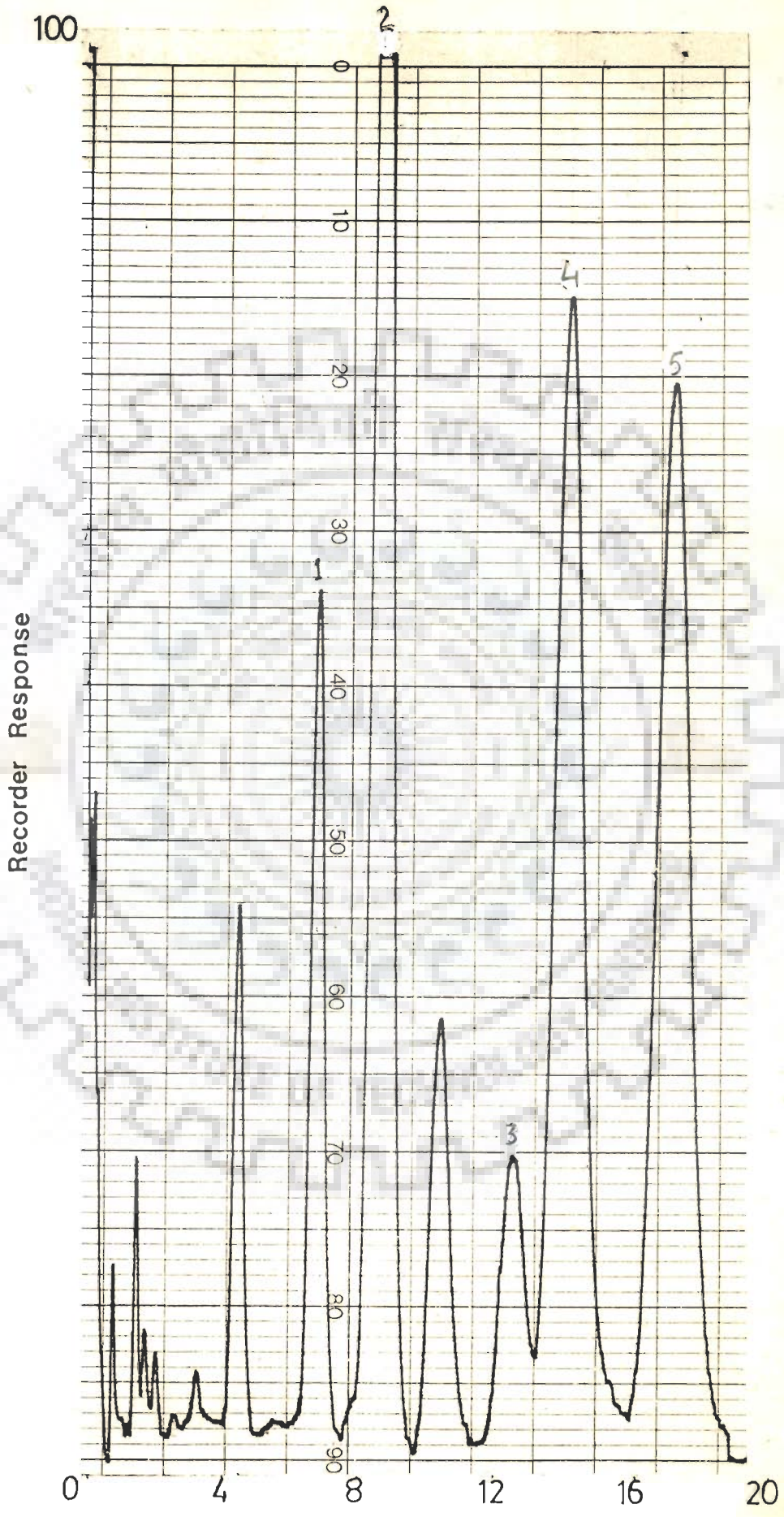
WIND DIRECTION
NEW DELHI



(N = Total number of observations, C = Number of calms as percentage of the total)

Fig.5 Gas chromatographic analysis of DDT and its metabolites in soil on column containing 1.5 % OV_{17} and 1.95 % QF_1 on Gas Chrom Q 100-120 mesh. Further details of operating conditions are given in Table 1.

Peak No.	1	DDMU/o,p'-DDE (not included)
	2	p,p'-DDE
	3	o,p'-DDT
	4	p,p'-DDD
	5	p,p'-DDT


100
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20
30
40
50
60
70
80
90
1
2
3
4
5
0
4
8
12
16
20
Recorder Response
Retention Time min.

TABLE-7: CONCENTRATIONS OF DDT AND ITS METABOLITES IN SOIL OF DELHI AREA COLLECTED DURING OCTOBER TO NOVEMBER, 1983(ppm).

Locality	Zone	p,p'-DDE	o,p'-DDT	p,p'-DDD	p,p'-DDT	Total DDT
Wazirpur Ind.Area(1)	N	0.276	0.000	0.290	0.417	0.983
Azadpur (2)	N	0.328	0.162	0.528	1.012	2.030
Timarpur (3)	N	0.467	0.117	0.262	0.510	1.356
Roshanara Garden (4)	N	0.204	0.115	0.202	0.293	0.814
Daya Basti (5)	N	0.653	0.325	0.600	0.971	2.549
Delhi University (6)	N	0.332	0.147	0.273	0.545	1.297
Wazirabad (7)	N	0.270	0.072	0.204	0.393	0.939
Wazirabad Water Pumping Station (8)	N	0.736	0.275	0.694	1.299	3.004
Nirankari Colony (9)	N	0.221	0.085	0.192	0.312	0.800
Radio Colony (10)	N	0.268	0.115	0.261	0.519	1.163
Lajpat Nagar (11)	S	0.456	0.132	0.317	0.621	1.526
South Extension (12)	S	0.281	0.155	0.267	0.437	1.140
Safdarjang Enclave (13)	S	0.404	0.216	0.543	0.823	1.986
Delhi Airport (14)	S	1.993	0.201	0.546	0.873	3.613
Palam Road (15)	S	0.804	0.256	0.793	1.663	3.516
Kalkani (16)	S	0.380	0.132	0.420	0.796	1.728
Greater Kailash (17)	S	0.142	0.042	0.115	0.231	0.530
Gobind Puri (18)	S	0.451	0.098	0.214	0.443	1.206
R.K. Puram (19)	S	0.235	0.077	0.162	0.369	0.845
Dhaula Kuan (20)	S	0.281	0.100	0.274	0.538	1.193

Cont'd..

Continued..

Old Jamuna Bridge (21)	E	0.512	0.171	0.542	1.100	2.325
Raj Ghat (22)	E	0.144	0.062	0.124	0.255	0.585
Kalandi Colony (23)	E	0.212	0.079	0.212	0.393	0.896
Kataria Nursery (24)	E	1.063	0.166	0.357	0.528	2.114
Delhi Zoo (25)	E	0.271	0.125	0.258	0.500	1.154
Jamia Nagar (26)	E	0.228	0.080	0.234	0.437	0.979
Okhla (27)	E	0.348	0.128	0.258	0.492	1.226
Navin Shahadra (28)	E	0.290	0.095	0.280	0.393	1.058
Vivek Vihar (29)	E	0.269	0.035	0.304	0.501	1.109
Geeta Colony (30)	E	0.469	0.102	0.325	0.558	1.454
Jheel (31)	E	0.182	0.053	0.124	0.244	0.603
Punjabi Bagh (32)	W	0.327	0.129	0.188	0.572	1.216
Tilak Nagar (33)	W	1.235	0.237	0.653	1.461	3.586
Moti Nagar (34)	W	1.000	0.000	0.333	1.293	2.626
Inderlak (35)	W	1.110	0.096	0.495	1.060	2.761
Sarai Rohilla (36)	W	0.514	0.132	0.109	0.839	1.594
Karam Pura (37)	W	0.661	0.211	0.434	0.949	2.255
Bharat Nagar (38)	W	0.138	0.079	0.114	0.229	0.560
Raja Garden (39)	W	0.429	0.079	0.440	0.980	1.928
Durga Nagar (40)	W	1.618	1.520	1.373	2.759	7.270
Naraina Ind.Area (41)	W	0.396	0.000	0.152	0.446	0.994
Naraina (42)	W	0.298	0.000	0.166	0.560	1.024

Cont'd..

Continued..

Central Secretariat (43)	C	0.419	0.147	0.198	0.456	1.220
Budha Memorial Park (44)	C	0.191	0.770	0.245	0.441	1.647
Karol Bagh (45)	C	0.344	0.095	0.258	0.582	1.279
Pahar Ganj (46)	C	0.313	0.084	0.254	0.569	1.220
Ajmal Khan Park (47)	C	0.149	0.060	0.133	0.329	0.671
IARI Fields-I (48)	C	0.690	0.183	0.740	1.543	3.156
IARI Fields-II (49)	C	0.235	0.000	0.073	0.190	0.498
IARI Campus (50)	C	0.626	0.238	0.512	0.934	2.310

Figures in parentheses indicate site number as shown in Fig.4.

N = North,
S = South,
E = East,
C = Central,
W = West

being 7.270 ppm in Durga Nagar. The lowest total DDT level of 0.498 ppm was found at IARI campus. Some areas such as Azadpur, Daya Basti, Palam Road, Old Jamuna Bridge, Kataria Nursery and Karam Pura contained moderate levels of DDT residues; total DDT ranged from 2.030 to 3.516 ppm. Other areas such as Jheel, R.K. Puram, Raj Ghat, Kalindi Colony, Jamia Nagar, Roshanara Garden, Bharat Nagar, Ajmal Khan Park, Nirankari Colony, Greater Kailash and Naraina Industrial Area had total DDT residues less than 1.00 ppm.

The results presented in Table 8a show that the main constituent of DDT residues was p,p'-DDT in most of the soil samples. p,p'-DDT was more than p,p'-DDE in 45 out of 50 soil samples analyzed. The proportion of p,p'-DDT to p,p'-DDE was 2:1. Other predominant residue was p,p'-DDD in most of the soil samples. In addition to p,p'-DDT, detectable amounts of o,p'-DDT were obtained in 45 out of 50 soil samples. The mean values of four DDT metabolites were compared using one way analysis of variance on log transformed values followed by multiple range test (Table 8b). The mean p,p'-DDE value (0.477 ppm) was higher than that of p,p'-DDD (0.340 ppm; $p < 0.05$) and o,p'-DDT (0.160 ppm; $p < 0.001$). The mean p,p'-DDT value (0.693 ppm) was higher than that of o,p'-DDT (0.160 ppm; $p < 0.001$), p,p'-DDD (0.340 ppm; $p < 0.001$) and p,p'-DDE (0.477 ppm; $p < 0.05$). The mean p,p'-DDD value (0.340 ppm) was higher than that of o,p'-DDT (0.160 ppm; $p < 0.001$).

Zonal distribution of DDT and its metabolites is presented in Table 9 and Fig.6. The mean total DDT values of 5 zones were compared using one way analysis of variance. No statistically significant difference was observed in mean

TABLE-8a: LEVELS OF DDT AND ITS METABOLITES IN SOIL OF DELHI AREA (ppm).

Compound	No. of samples	Range Mean ± S.D.	Geometric mean	Median	Quartile deviation
p,p'-DDE	50	0.138 - 1.993 0.477 ± 0.378 (50)	0.385[a]	0.338	0.239
o,p'-DDT	50	0.000 - 1.520 0.160 ± 0.228 (45)	0.139[b]	0.116	0.086
p,p'-DDD	50	0.073 - 1.373 0.340 ± 0.232 (50)	0.284[c]	0.264	0.232
p,p'-DDT	50	0.190 - 2.759 0.693 ± 0.467 (50)	0.583[d]	0.533	0.451
Total DDT	50	0.498 - 7.270 1.670 ± 1.163 (50)		1.223	1.027

Figures in parentheses indicate the number of positive samples.

TABLE-8b: RESULTS OF MULTIPLE RANGE TEST.

	b	c	a
c	$p < 0.001$	-	
a	$p < 0.001$	$p < 0.05$	-
d	$p < 0.001$	$p < 0.001$	$p < 0.05$

TABLE-9: ZONAL DISTRIBUTION OF DDT AND ITS METABOLITES IN SOIL OF DELHI AREA (ppm).

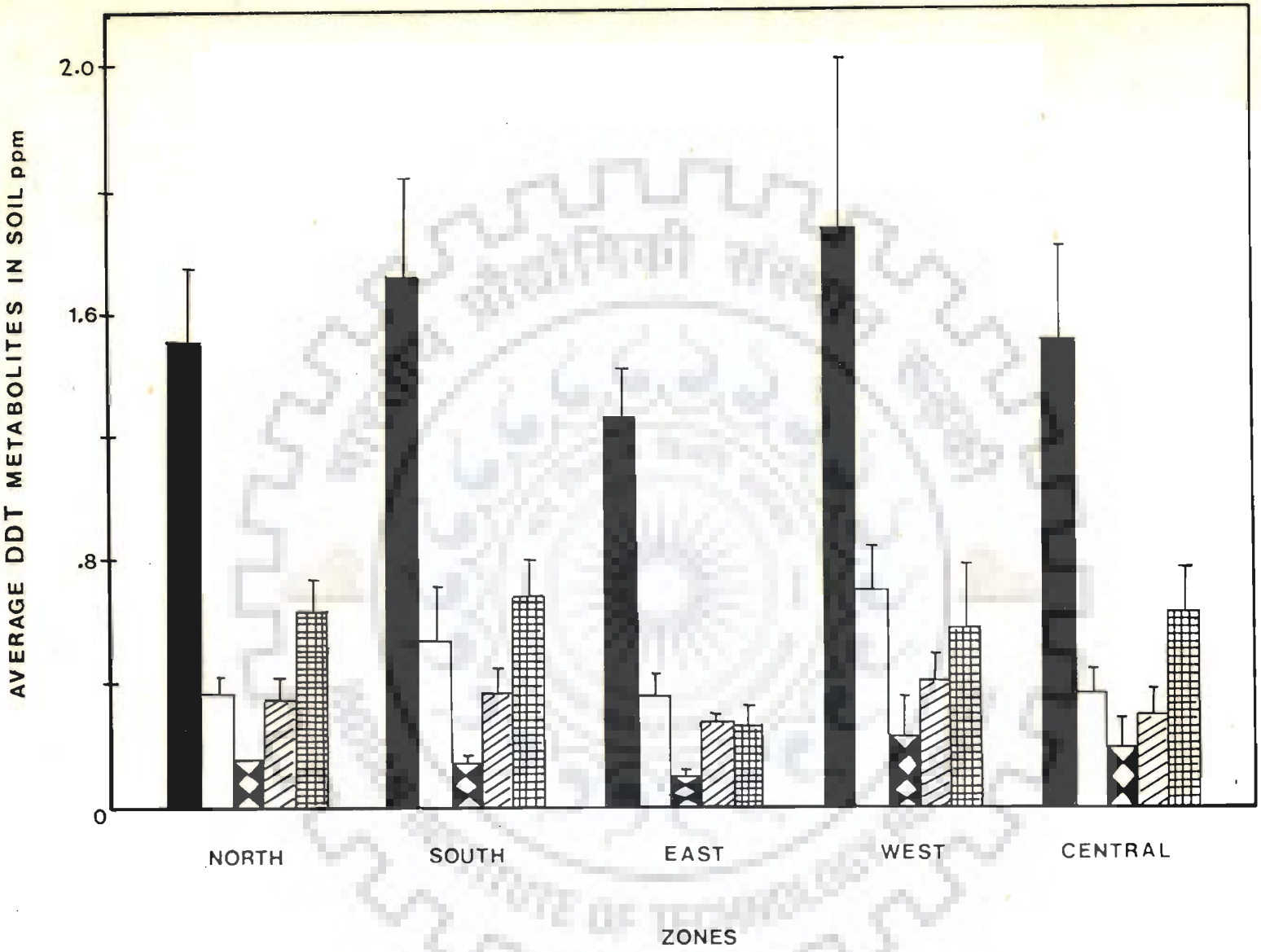
Compound	RANGE OF CONCENTRATIONS AND MEAN ± S.D.				
	Zones				
	North N = 10	South N = 10	East N = 11	West N = 11	Central N = 8
p,p'-DDE	0.204 - 0.736 0.374 ± 0.185 (10)	0.142 - 1.993 0.542 ± 0.540 (10)	0.144 - 1.063 0.362 ± 0.258 (11)	1.380 - 1.618 0.702 ± 0.469 (11)	0.149 - 0.690 0.370 ± 0.197 (8)
o,p'-DDT	0.000 - 0.325 0.147 ± 0.095 (9)	0.042 - 0.256 0.140 ± 0.066 (10)	0.035 - 0.171 0.099 ± 0.044 (11)	0.000 - 1.520 0.225 ± 0.436 (8)	0.000 - 0.740 0.197 ± 0.243 (7)
p,p'-DDD	0.192 - 0.694 0.350 ± 0.184 (10)	0.115 - 0.793 0.365 ± 0.210 (10)	0.124 - 0.542 0.274 ± 0.115 (11)	0.109 - 1.373 0.405 ± 0.368 (11)	0.073 - 0.740 0.301 ± 0.218 (8)
p,p'-DDT	0.293 - 1.299 0.627 ± 0.343 (10)	0.073 - 1.663 0.679 ± 0.404 (10)	0.244 - 1.110 0.491 ± 0.227 (11)	0.229 - 2.759 1.013 ± 0.685 (11)	0.190 - 1.543 0.630 ± 0.427 (8)
Total DDT	0.800 - 3.004 1.493[a] ± 0.771 (10)	0.530 - 3.613 1.728[b] ± 1.052 (10)	0.585 - 2.325 1.227[c] ± 0.553 (11)	0.560 - 7.270 2.346[d] ± 1.866 (11)	0.498 - 3.156 1.500[e] ± 0.871 (8)

N = Number of samples.

Figures in parentheses indicate the number of positive samples.

a, b, c, d, e = Statistically not significant.

Fig.6 Bar diagram showing zonal distribution of DDT and its metabolites in the soil of Delhi area.


AVERAGE DDT METABOLITES IN SOIL ppm
2.0
1.6
.8
0
NORTH
SOUTH
EAST
WEST
CENTRAL
ZONES

total DDT levels of soil of 5 zones of Delhi. The maximum mean total DDT value, 2.346 ppm, was found in West Zone soils and minimum, 1.227 ppm, in East Zone soils.

4.2 *Residues in blood of occupationally unexposed population*

The DDT and its metabolites were analyzed in 50 blood samples of occupationally unexposed population of Delhi. The gas chromatographic pattern of DDT residues in the blood of occupationally unexposed population of Delhi is presented in Fig. 7. The results presented in Table 10 show the occurrence of DDT residues in whole blood of occupationally unexposed population of Delhi area. The statistical outcome of the data representing the range of concentrations and mean values with standard deviations is presented in Table 11. The total DDT concentration in blood ranged from 0.053 to 0.663 ppm with a mean value of 0.301 ± 0.169 ppm. The metabolites detected were p,p'-DDE, o,p'-DDT, p,p'-DDD and p,p'-DDT.

The sex wise distribution of DDT residues in whole blood of occupationally unexposed population is presented in Table 11 and Fig.8. The mean total DDT values of two sexes were compared using 't' test. The results indicate that DDT level in males (0.344 ppm) was higher than that of females (0.229 ppm; $p < 0.05$).

The accumulation of DDT residues in artificially grouped ages is presented in Table 12 and Fig.9. The mean DDT levels of different age groups were compared using one way analysis of variance. No statistically significant difference in blood DDT levels was observed between the age groups. No positive correlation was observed between DDT concentrations in blood and the age (Fig.10).

Fig.7 Gas chromatographic analysis of DDT and its metabolites in blood of occupationally unexposed population on the column containing 1.5 % OV_{17} and 1.95 % QF_1 on Gas Chrom Q 100-120 mesh. Further details of operating conditions are given in Table.1.

Peak No. 1 p,p'-DDE
2 o,p'-DDT
3 p,p'-DDD
4 p,p'-DDT

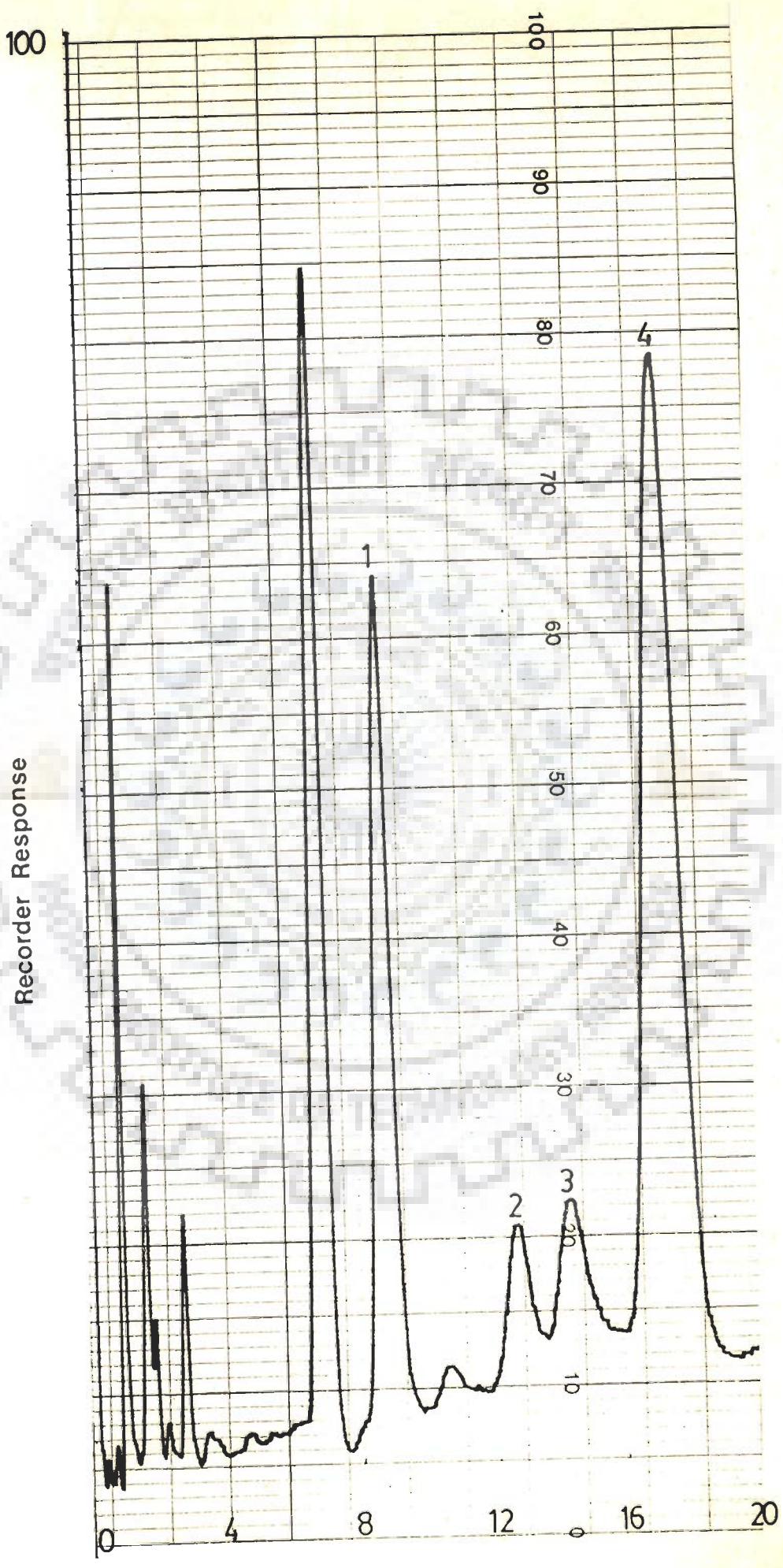

100
Recorder Response
100
90
80
70
60
50
40
30
20
10
0
1
2
3
4
0
4
8
12
16
20
Retention Time min.

TABLE-10: CONCENTRATION OF DDT AND ITS METABOLITES IN WHOLE BLOOD OF OCCUPATIONALLY UNEXPOSED POPULATION OF DELHI(ppm).

S. No.	Age/ Sex	Dietetic habit	Zone	p,'-DDE	o,p'-DDT	p,p'-DDD	p,p'-DDT	Total DDT
1.	23F +	V*	South	0.057	0.036	0.000	0.112	0.205
2.	25M ++	NV**	South	0.090	0.039	0.003	0.106	0.238
3.	25M	NV	South	0.125	0.079	0.003	0.180	0.387
4.	24M	NV	South	0.123	0.111	0.000	0.295	0.529
5.	24F	NV	South	0.057	0.050	0.002	0.081	0.190
6.	26M	NV	South	0.160	0.118	0.004	0.176	0.458
7.	26M	NV	South	0.210	0.154	0.000	0.240	0.604
8.	30M	V	South	0.224	0.154	0.000	0.285	0.663
9.	25M	NV	South	0.185	0.090	0.004	0.172	0.451
10.	25F	NV	South	0.079	0.045	0.002	0.073	0.199
11.	27M	NV	West	0.206	0.181	0.000	0.263	0.650
12.	27F	NV	West	0.114	0.090	0.000	0.131	0.335
13.	30F	NV	West	0.202	0.117	0.000	0.194	0.513
14.	22M	V	West	0.156	0.093	0.000	0.164	0.413
15.	20M	NV	West	0.208	0.117	0.000	0.172	0.497
16.	48F	NV	West	0.124	0.054	0.000	0.086	0.264
17.	28M	V	West	0.184	0.117	0.000	0.116	0.417
18.	20M	NV	West	0.119	0.119	0.000	0.094	0.332
19.	28M	NV	West	0.164	0.039	0.000	0.116	0.319
20.	42F	V	West	0.088	0.015	0.001	0.047	0.151
21.	41F	NV	North	0.096	0.062	0.001	0.082	0.241
22.	45F	V	North	0.115	0.060	0.000	0.090	0.265
23.	25M	NV	North	0.241	0.148	0.003	0.212	0.604
24.	46F	V	North	0.118	0.084	0.001	0.106	0.309

Cont'd..

Continued..

25. 39M	NV	North	0.239	0.135	0.000	0.208	0.582
26. 22M	V	North	0.128	0.074	0.000	0.100	0.302
27. 24M	NV	North	0.200	0.067	0.000	0.116	0.383
28. 19F	NV	North	0.110	0.064	0.000	0.071	0.245
29. 24M	V	North	0.118	0.068	0.000	0.077	0.263
30. 31M	NV	North	0.142	0.054	0.000	0.084	0.280
31. 37M	V	East	0.080	0.034	0.006	0.032	0.152
32. 24M	V	East	0.080	0.020	0.000	0.012	0.112
33. 26F	V	East	0.057	0.012	0.000	0.004	0.073
34. 13F	NV	East	0.042	0.010	0.001	0.000	0.053
35. 25M	V	East	0.114	0.025	0.000	0.000	0.139
36. 27M	V	East	0.116	0.034	0.000	0.040	0.190
37. 21M	NV	East	0.110	0.032	0.002	0.006	0.150
38. 19F	NV	East	0.088	0.016	0.002	0.000	0.106
39. 22F	V	East	0.060	0.017	0.000	0.008	0.085
40. 24F	V	East	0.106	0.032	0.000	0.008	0.146
41. 24M	NV	Central	0.110	0.022	0.000	0.023	0.155
42. 35M	V	Central	0.095	0.022	0.000	0.000	0.117
43. 24M	V	Central	0.026	0.000	0.000	0.000	0.026
44. 40M	NV	Central	0.193	0.030	0.000	0.020	0.243
45. 18M	NV	Central	0.239	0.072	0.000	0.142	0.453
46. 30F	NV	Central	0.074	0.045	0.001	0.073	0.193
47. 23M	NV	Central	0.119	0.054	0.000	0.106	0.279
48. 34F	NV	Central	0.088	0.050	0.000	0.085	0.223
49. 33F	NV	Central	0.269	0.115	0.000	0.176	0.560
50. 35M	NV	Central	0.149	0.054	0.000	0.139	0.342

\+ Female
++ Male

*Vegetarian
**Non-vegetarian

TABLE-11: LEVELS OF DDT AND ITS METABOLITES IN WHOLE BLOOD OF OCCUPATIONALLY UNEXPOSED POPULATION OF DELHI (ppm).

	Range of concentrations and means ± S.D.				
	p,p'-DDE	o,p'-DDT	p,p'-DDD	p,p'-DDT	Total DDT
Male N=31	0.026-0.239	0.000-0.180	0.000-0.006	0.000-0.295	0.112-0.663
	0.147±0.059	0.076±0.048	0.0008±0.001	0.119±0.088	0.344±0.175
	(31)	(30)	(7)	(28)	(31)
					t=2.63
					p<0.05
Female N=19	0.042-0.269	0.010-0.117	0.000-0.002	0.000-0.194	0.053-0.560
	0.102±0.053	0.051±0.032	0.0005±0.0007	0.075±0.055	0.229±0.133
	(19)	(19)	(8)	(17)	(19)
Total N=50	0.026-0.269	0.000-0.180	0.000-0.006	0.000-0.295	0.053-0.663
	0.129±0.061	0.066±0.044	0.0005±0.0007	0.102±0.079	0.301±0.169
	(50)	(49)	(15)	(45)	(50)

N = Number of samples.

Figures in parentheses indicate the number of positive samples.

TABLE-12: AGE WISE DISTRIBUTION OF DDT AND ITS METABOLITES IN WHOLE BLOOD OF OCCUPATIONALLY UNEXPOSED POPULATION OF DELHI (ppm).

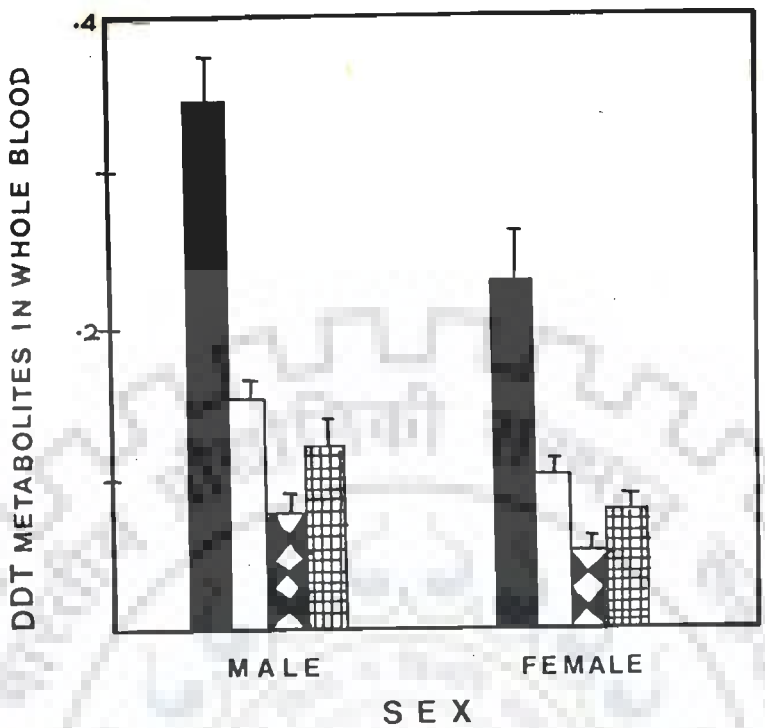
Compound	Age groups			
	10-20 yrs N = 6	21-30 yrs N = 31	31-40 yrs N = 8	41-50 yrs N = 5
p,p'-DDE	0.042 - 0.239 0.134 ± 0.074 (6)	0.026 - 1.241 0.129 ± 0.056 (31)	0.080 - 0.269 0.156 ± 0.071 (8)	0.088 - 0.124 0.108 ± 0.015 (5)
o,p'-DDT	0.010 - 0.119 0.066 ± 0.047 (6)	0.000 - 0.181 0.069 ± 0.048 (30)	0.022 - 0.135 0.061 ± 0.041 (8)	0.015 - 0.084 0.055 ± 0.025 (5)
p,p'-DDD	0.000 - 0.002 0.0005± 0.0008 (2)	0.000 - 0.004 0.0007± 0.001 (9)	0.000 - 0.006 0.0007± 0.002 (1)	0.000 - 0.001 0.0006± 0.0005 (5)
p,p'-DDT	0.000 - 0.172 0.079 ± 0.071 (4)	0.000 - 0.295 0.112 ± 0.088 (30)	0.000 - 0.208 0.093 ± 0.075 (7)	0.047 - 0.106 0.082 ± 0.021 (5)
Total DDT	0.053 - 0.497 0.281 ± 0.180 (6)	0.026 - 0.663 0.310 ± 0.182 (31)	0.152 - 0.582 0.312 ± 0.174 (8)	0.151 - 0.309 0.246 ± 0.058 (5)

N = Number of samples.

Figures in parentheses indicate the number of positive samples.

Fig.8 Bar diagram showing sex wise distribution of DDT residues in the blood of occupationally exposed population of Delhi.

Fig.9 Bar diagram showing age wise distribution of DDT residues in the blood of occupationally unexposed population of Delhi.


.4
.2
DDT METABOLITES IN WHOLE BLOOD
MALE
FEMALE
SEX

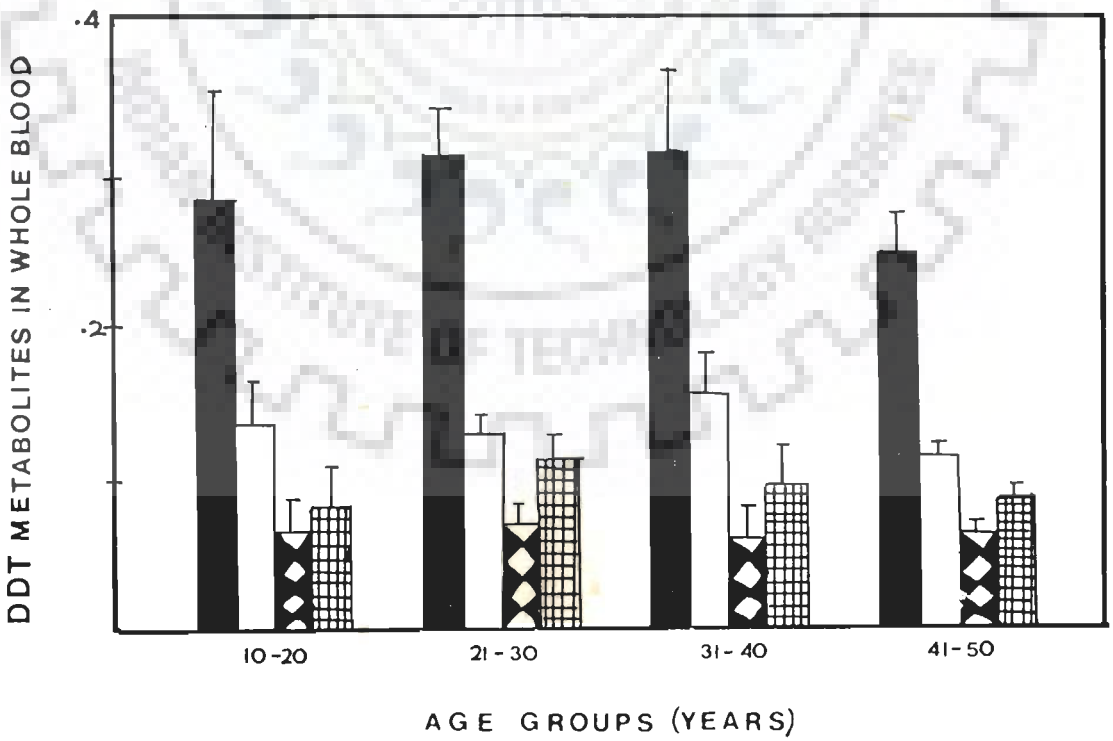

.4
.2
DDT METABOLITES IN WHOLE BLOOD
10-20
21-30
31-40
41-50
AGE GROUPS (YEARS)

Fig.10 Scatter diagram showing correlation between total DDT concentrations in the blood and age.

r = 0.057

n = 50

NS

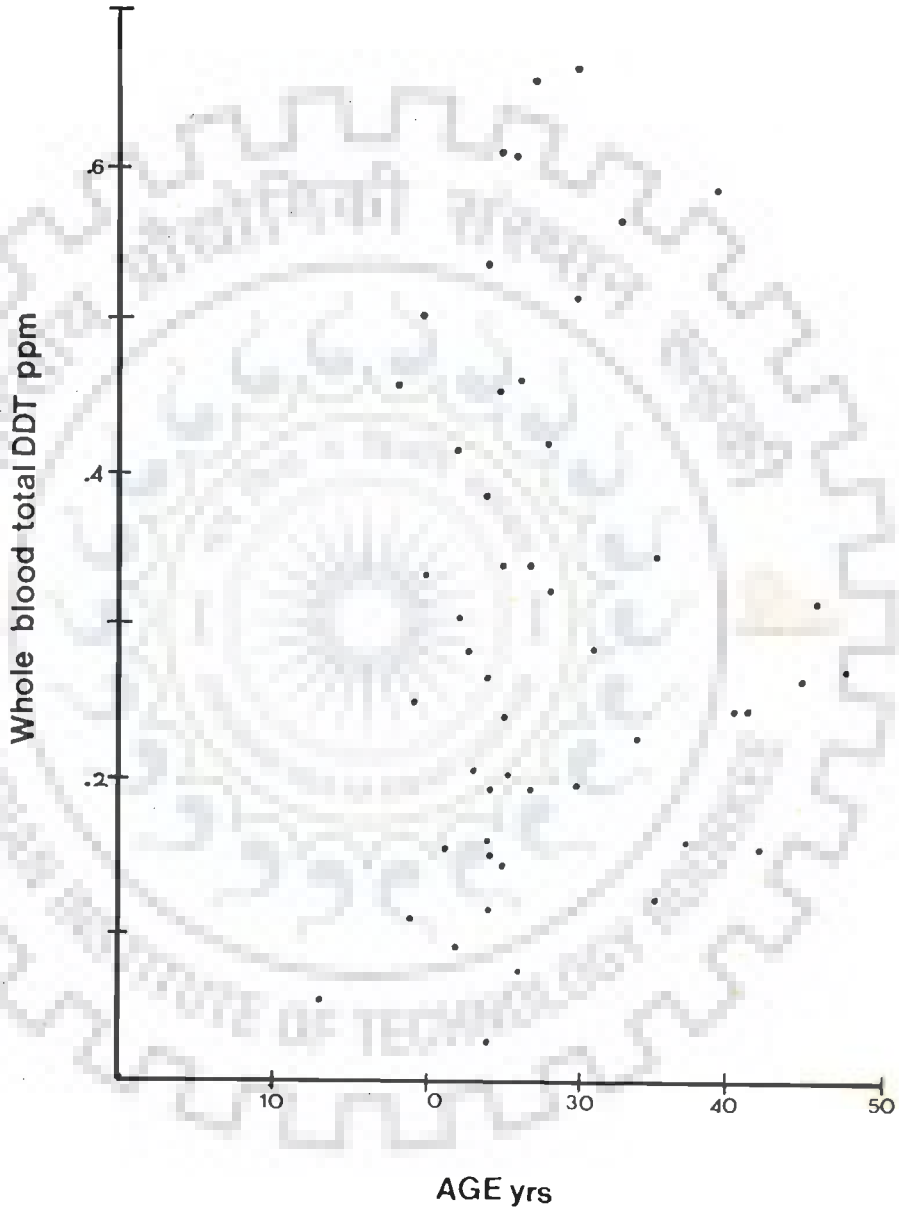

Whole blood total DDT ppm
.6
.4
.2
10
0
30
40
50
AGE yrs

TABLE-13: DDT AND ITS METABOLITES IN WHOLE BLOOD OF OCCUPATIONALLY UNEXPOSED POPULATION OF DELHI AS RELATED TO DIETETIC HABITS (ppm).

Compound	Range of concentrations and mean ± S.D.		
	Dietetic habits		
	Non-vegetarians N = 32		Vegetarians N = 18
p,p'-DDE	0.042 - 0.269 0.146 ± 0.060 (32)		0.026 - 0.224 0.106 ± 0.047 (18)
o,p'-DDT	0.010 - 0.154 0.076 ± 0.043 (32)		0.012 - 0.154 0.049 ± 0.041 (18)
p,p'-DDD	0.000 - 0.004 0.0008± 0.001 (12)		0.000 - 0.006 0.0004± 0.001 (3)
p,p'-DDT	0.000 - 0.295 0.122 ± 0.075 (30)		0.000 - 0.285 0.066 ± 0.074 (15)
Total DDT	0.053 - 0.650 0.345 ± 0.161 (32)	$t = 2.67$ $p < 0.05$	0.026 - 0.663 0.221 ± 0.156 (18)

N = Number of samples.

Figures in parentheses indicate the number of positive samples.

TABLE-14a: ZONAL DISTRIBUTION OF DDT AND ITS METABOLITES IN WHOLE BLOOD OF OCCUPATIONALLY UNEXPOSED POPULATION OF DELHI (ppm).

Compound	Range of concentrations and means ± S.D.				
	Zones				
	North N = 10	South N = 10	East N = 10	West N = 10	Central N = 10
p,p'-DDE	0.096 - 0.241 0.150 ± 0.054 (10)	0.057 - 0.224 0.131 ± 0.061 (10)	0.042 - 0.116 0.085 ± 0.026 (10)	0.088 - 0.208 0.156 ± 0.043 (10)	0.026 - 0.269 0.136 ± 0.076 (10)
o,p'-DDT	0.054 - 0.148 0.081 ± 0.032 (10)	0.036 - 0.154 0.087 ± 0.045 (10)	0.010 - 0.034 0.023 ± 0.009 (10)	0.015 - 0.181 0.094 ± 0.047 (10)	0.000 - 0.115 0.046 ± 0.031 (9)
p,p'-DDD	0.000 - 0.003 0.0005 ± 0.0009 (3)	0.000 - 0.004 0.002 ± 0.001 (6)	0.000 - 0.006 0.001 ± 0.002 (4)	0.000 - 0.001 0.0001± 0.0003 (1)	0.000 - 0.001 0.0001± 0.0003 (1)
p,p'-DDT	0.071 - 0.212 0.114 ± 0.052 (10)	0.073 - 0.295 0.172 ± 0.080 (10)	0.000 - 0.040 0.011 ± 0.014 (7)	0.047 - 0.263 0.138 ± 0.061 (10)	0.000 - 0.176 0.076 ± 0.063 (8)
Total DDT	0.241 - 0.604 0.347 ± 0.135 (10) $\underline{0.328}^{a}$	0.190 - 0.663 0.392 ± 0.176 (10) $\underline{0.354}^{b}$	0.053 - 0.190 0.120 ± 0.036 (10) $\underline{0.113}^{c}$	0.151 - 0.650 0.389 ± 0.141 (10) $\underline{0.364}^{d}$	0.026 - 0.560 0.259 ± 0.158 (10) $\underline{0.203}^{e}$

N = Number of samples.
Figures in parentheses indicate the number of positive samples.
Figures underlined are geometric mean values.

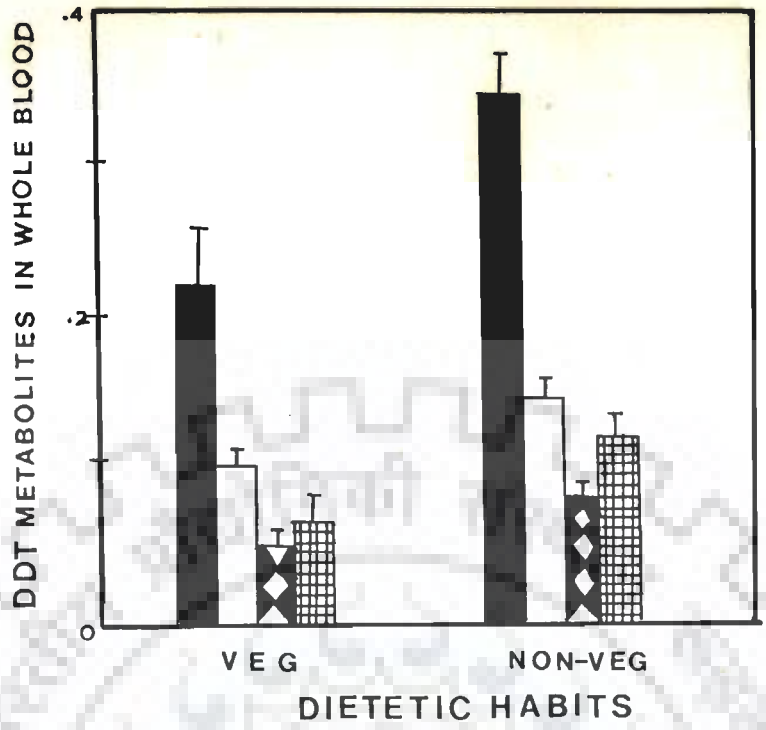
TABLE-14b: RESULTS OF MULTIPLE RANGE TEST PERFORMED ON LOG TRANSFORMED VALUES.

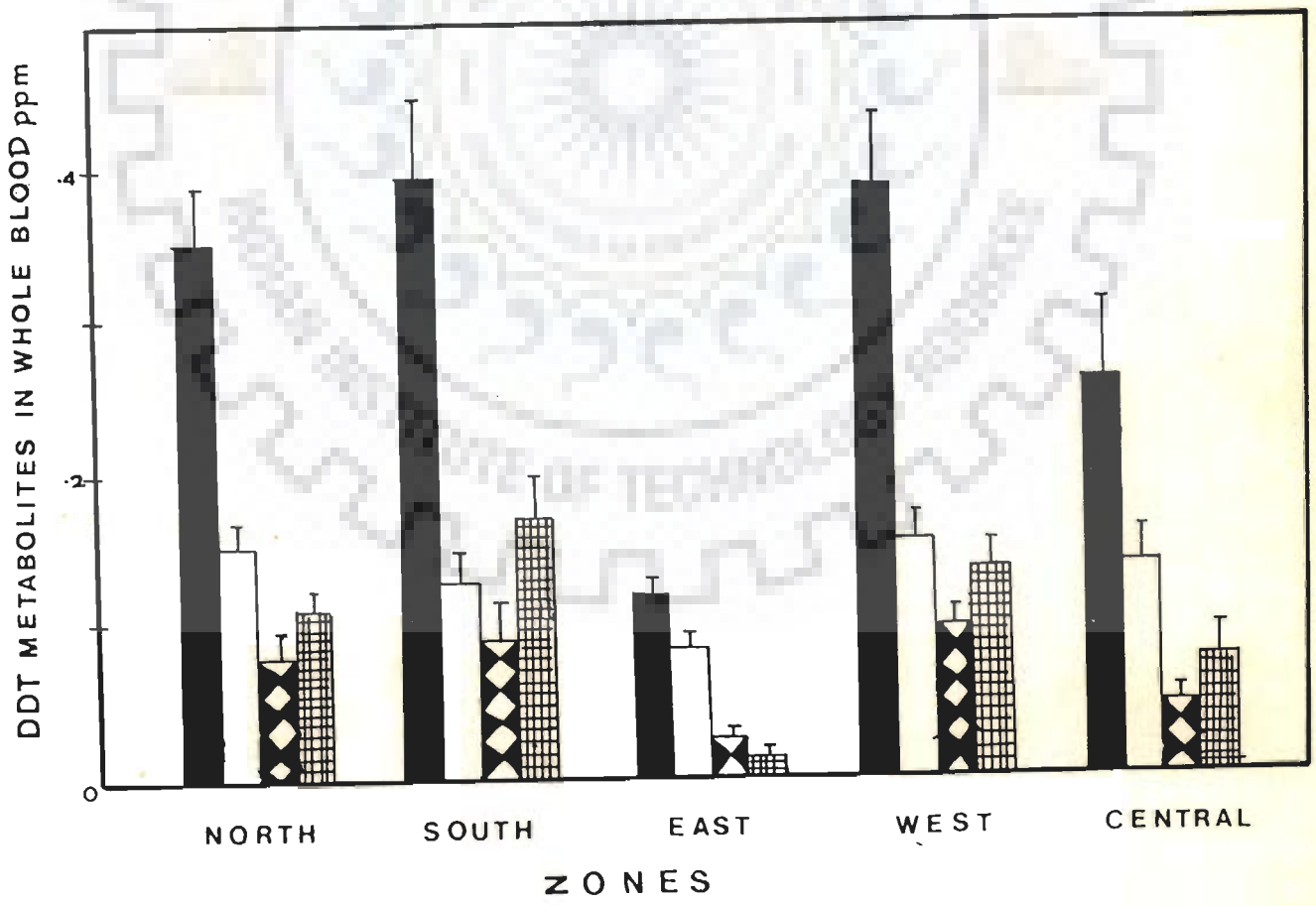
	c	e	a	b
e	p<0.05	-		
a	p<0.001	NS		
b	p<0.001	NS	NS	-
d	p<0.001	NS	NS	NS

NS = Statistically not significant.

Fig.11 Bar diagram showing distribution of DDT residues in the blood of occupationally unexposed population of Delhi as related to dietetic habits.

Fig.12 Bar diagram showing zonal distribution of DDT residues in the blood of occupationally unexposed population of Delhi.


DDT METABOLITES IN WHOLE BLOOD
.4
.2
0
VEG
NON-VEG
DIETETIC HABITS


DDT METABOLITES IN WHOLE BLOOD ppm
.4
.2
0
NORTH
SOUTH
EAST
WEST
CENTRAL
ZONES

The results presented in Table 13 and Fig.11 show that the mean total DDT value in non-vegetarians (0.345 ppm) was higher than that of vegetarians (0.221 ppm; $p < 0.05$).

Zonal distribution of DDT residues in whole blood of occupationally unexposed population of Delhi is presented in Table 14a. The mean total DDT values of 5 zones were compared using one way analysis of variance on log transformed values followed by multiple range test (Table 14b). The DDT residues in the blood of East Zone residents (0.120 ppm) differed significantly from residents of Central Zone (0.259 ppm; $p < 0.05$), North Zone (0.347 ppm; $p < 0.001$), South Zone (0.392 ppm; $p < 0.001$) and West Zone (0.389 ppm; $p < 0.001$). No significant difference in DDT levels was observed in the blood of residents from four zones viz. North, South, East and West.

4.3 *Residues in pregnant and lactating women*

The DDT and its metabolites in maternal blood, umbilical cord blood and placental tissue were estimated in women who underwent full term pregnancy, premature labour and toxemia of pregnancy. The gas chromatographic pattern of DDT residues in maternal blood, umbilical cord blood and placental tissue is presented in Fig. 13, 14 and 15. Case wise findings are summarized in Table 15 and 16. The statistical outcome of the data representing the range of concentrations and mean values with standard deviations is summarized in Table 17a,b and 18a,b.

The mean total DDT levels in maternal blood, umbilical cord blood and placental tissue of women with full term pregnancy were compared using one way analysis of variance on

Fig.13 Gas chromatographic analysis of DDT residues in maternal blood of pregnant women on the column containing 1.5 % OV_{17} and 1.95 % QF_1 on Gas Chrom Q 100-120 mesh. Further details of operating conditions are given in Table 1.

Peak No.		
	1	p,p'-DDE
	2	o,p'-DDT
	3	p,p'-DDD
	4	p,p'-DDT

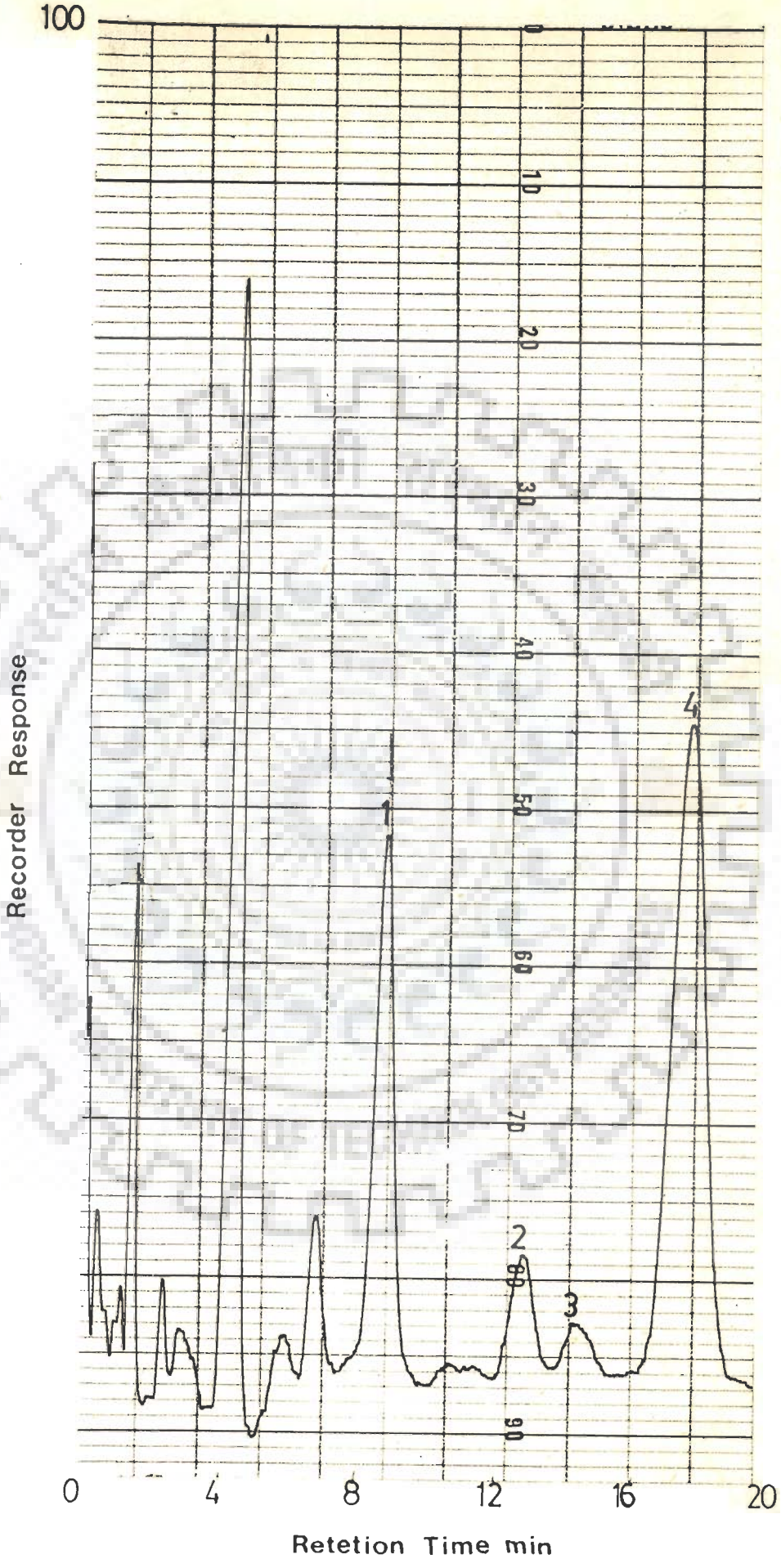

100
Recorder Response
0
10
20
30
40
50
60
70
80
90
1
2
3
4
0
4
8
12
16
20
Retetion Time min

Fig.14 Gas chromatographic analysis of DDT residues in umbilical cord blood on the column containing 1.5 % OV_{17} and 1.95 % QF_1 on Gas Chrom Q 100-120 mesh. Further details of operating conditions are given in Table 1.

Peak No. 1 p,p'-DDE
2 o,p'-DDT
3 p,p'-DDD
4 p,p'-DDT

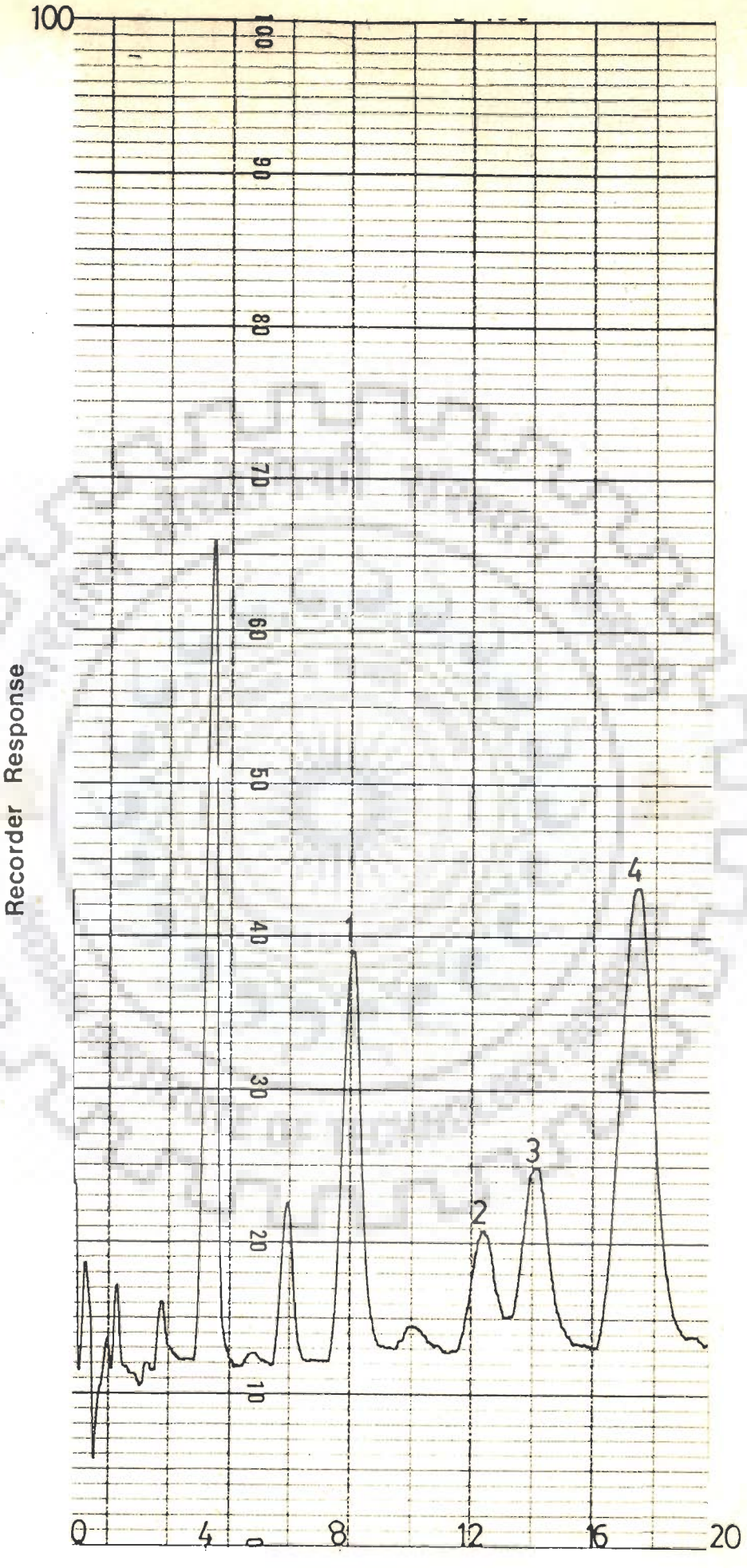

100
90
80
70
60
50
40
30
20
10
0
Recorder Response
1
2
3
4
0
4
8
12
16
20
Retention Time min.

Fig.15 Gas chromatographic analysis of DDT residues in placental tissue of pregnant women on the column containing 1.5 % OV_{17} and 1.95 % QF_1 on Gas Chrom Q 100-120 mesh. Further details of operating conditions are presented in Table 1.

Peak No. 1 p,p'-DDE
2 o,p'-DDT
3 p,p'-DDD
4 p,p'-DDT

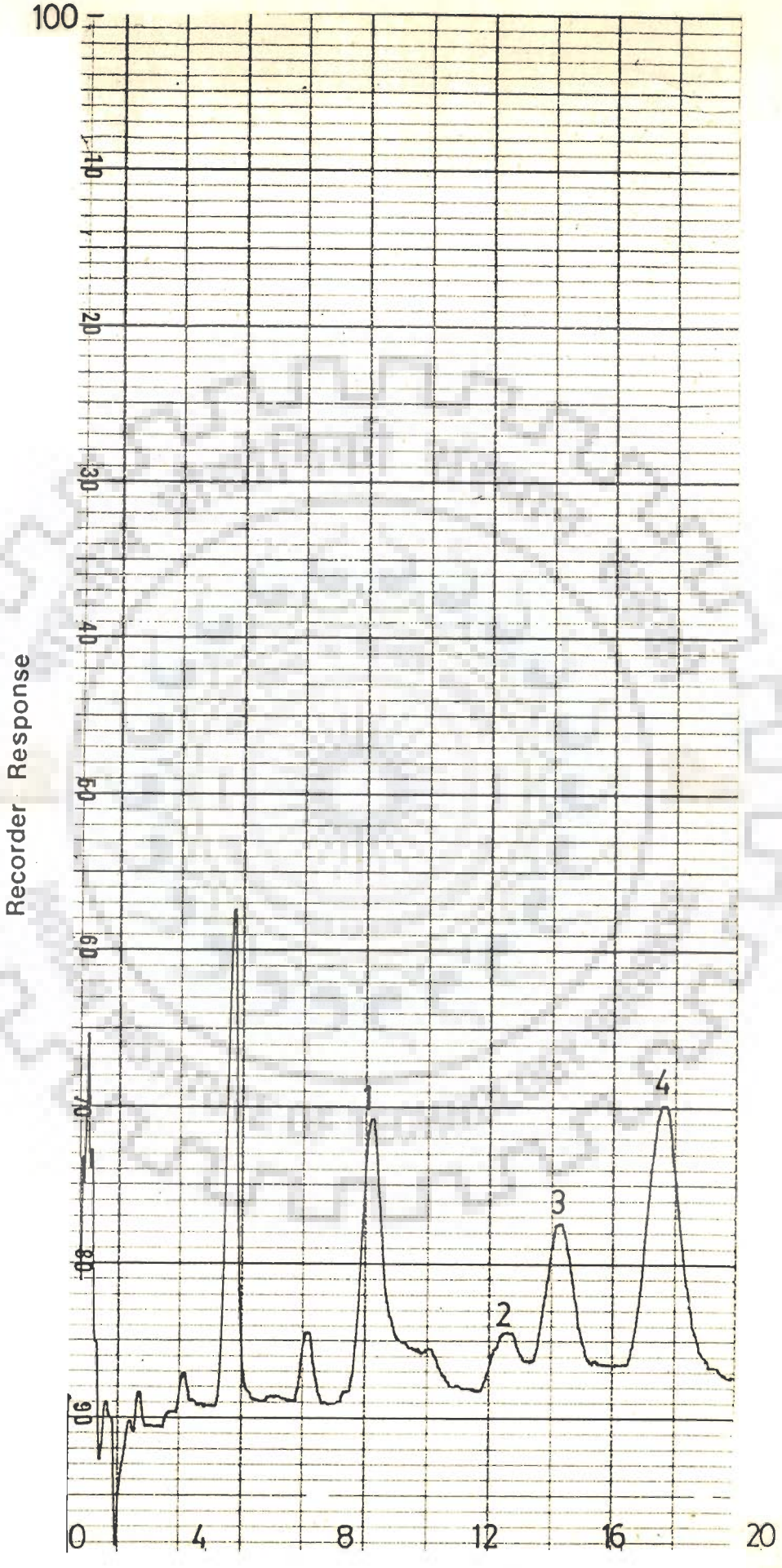

100
10
20
30
40
50
60
70
80
90
Recorder Response
1
2
3
4
0
4
8
12
16
20
Retention Time min.

TABLE-15: CONCENTRATION OF DDT AND ITS METABOLITES IN MATERNAL BLOOD, UMBILICAL CORD BLOOD AND PLACENTAL TISSUE OF WOMEN WITH FULL TERM PREGNANCY(ppm).

S. No.	Age/ Dietetic habit	Tissue	p,p'-DDE	o,p'-DDT	p,p'-DDD	p,p'-DDT	Total DDT
		*Maternal blood	0.113	0.122	0.003	0.224	0.462
1.	20	**Cord blood	0.011	0.012	0.0001	0.024	0.047
	NV	***Placenta	0.139	0.077	0.000	0.214	0.430
		Maternal blood	0.063	0.068	0.002	0.224	0.357
2.	18	Cord blood	0.119	0.097	0.000	0.053	0.269
	NV	Placenta	0.039	0.046	0.000	0.204	0.289
		Maternal blood	0.074	0.079	0.001	0.174	0.328
3.	22	Cord blood	0.015	0.018	0.0001	0.109	0.142
	V	Placenta	0.023	0.016	0.0006	0.065	0.104
		Maternal blood	0.084	0.088	0.001	0.291	0.464
4.	22	Cord blood	0.016	0.019	0.0003	0.103	0.138
	V	Placenta	0.016	0.018	0.0003	0.057	0.091
		Maternal blood	0.026	0.029	0.0002	0.086	0.141
5.	25	Cord blood	0.017	0.021	0.0005	0.030	0.068
	V	Placenta	0.018	0.019	0.000	0.053	0.090
		Maternal blood	0.028	0.018	0.0007	0.075	0.121
6.	24	Cord blood	0.029	0.019	0.0001	0.040	0.088
	V	Placenta	0.049	0.051	0.001	0.056	0.157
		Maternal blood	0.076	0.083	0.001	0.187	0.347
7.	27	Cord blood	0.025	0.027	0.0006	0.041	0.093
	NV	Placenta	0.072	0.170	0.006	0.056	0.304
		Maternal blood	0.008	0.012	0.000	0.046	0.066
8.	23	Cord blood	0.163	0.139	0.000	0.044	0.346
	NV	Placenta	0.031	0.033	0.000	0.051	0.115
		Maternal blood	0.030	0.030	0.0005	0.048	0.108
9.	20	Cord blood	0.038	0.038	0.0006	0.063	0.139
	NV	Placenta	0.035	0.038	0.0006	0.055	0.128

Cont'd..

Continued..

	Maternal blood	0.274	0.198	0.000	0.197	0.669
10. 26	Cord blood	0.012	0.013	0.0003	0.086	0.111
V	Placenta	0.029	0.032	0.0006	0.113	0.174
	Maternal blood	0.050	0.054	0.0007	0.392	0.496
11. 23	Cord blood	0.132	0.132	0.002	0.138	0.404
NV	Placenta	0.021	0.022	0.000	0.136	0.179
	Maternal blood	0.064	0.065	0.0005	0.099	0.228
12. 28	Cord blood	0.013	0.016	0.0003	0.069	0.098
V	Placenta	0.201	0.202	0.002	0.149	0.554
	Maternal blood	0.014	0.015	0.0005	0.161	0.190
13. 30	Cord blood	0.016	0.017	0.003	0.100	0.136
V	Placenta	0.184	0.155	0.007	0.195	0.541
	Maternal blood	0.090	0.126	0.003	0.197	0.416
14. 22	Cord blood	0.032	0.039	0.0008	0.083	0.154
V	Placenta	0.189	0.155	0.002	0.082	0.428
	Maternal blood	0.018	0.019	0.000	0.085	0.122
15. 22	Cord blood	0.026	0.023	0.000	0.043	0.092
NV	Placenta	0.011	0.039	0.000	0.064	0.114
	Maternal blood	0.049	0.054	0.001	0.123	0.227
16. 25	Cord blood	0.123	0.095	0.005	0.033	0.256
V	Placenta	0.019	0.024	0.0005	0.064	0.107
	Maternal blood	0.026	0.023	0.001	0.054	0.104
17. 28	Cord blood	0.018	0.019	0.001	0.042	0.080
V	Placenta	0.066	0.061	0.001	0.049	0.177
	Maternal blood	0.025	0.029	0.000	0.314	0.368
18. 22	Cord blood	0.022	0.023	0.000	0.039	0.084
V	Placenta	0.187	0.125	0.004	0.062	0.378
	Maternal blood	0.027	0.017	0.000	0.068	0.112
19. 24	Cord blood	0.044	0.041	0.000	0.036	0.121
V	Placenta	0.048	0.031	0.000	0.063	0.142
	Maternal blood	0.022	0.018	0.0005	0.061	0.101
20. 27	Cord blood	0.026	0.016	0.0006	0.041	0.083
NV	Placenta	0.145	0.124	0.009	0.088	0.366

Cont'd..

Continued..

	Maternal blood	0.038	0.041	0.0005	0.069	0.148
21. 20	Cord blood	0.053	0.041	0.0006	0.031	0.125
NV	Placenta	0.073	0.073	0.001	0.051	0.198
	Maternal blood	0.084	0.079	0.0008	0.058	0.221
22. 23	Cord blood	0.029	0.029	0.0006	0.102	0.160
NV	Placenta	0.058	0.061	0.000	0.051	0.170
	Maternal blood	0.189	0.063	0.004	0.087	0.343
23. 20	Cord blood	0.039	0.043	0.0003	0.045	0.127
V	Placenta	0.169	0.131	0.002	0.096	0.398
	Maternal blood	0.030	0.030	0.001	0.058	0.119
24. 24	Cord blood	0.035	0.036	0.001	0.046	0.118
NV	Placenta	0.035	0.037	0.001	0.073	0.146
	Maternal blood	0.007	0.005	0.000	0.057	0.069
25. 25	Cord blood[2]	0.011.	0.007	0.000	0.047	0.065
V	Placenta	0.095	0.083	0.003	0.067	0.248
	Maternal blood	0.166	0.188	0.002	0.307	0.663
26. 26	Cord blood	0.029	0.028	0.000	0.059	0.116
NV	Placenta	0.023	0.021	0.000	0.042	0.086
	Maternal blood	0.042	0.047	0.001	0.103	0.192
27. 25	Cord blood	0.041	0.041	0.0006	0.078	0.160
V	Placenta	0.177	0.133	0.000	0.196	0.506
	Maternal blood	0.063	0.067	0.001	0.134	0.265
28. 24	Cord blood	0.019	0.018	0.0003	0.051	0.088
NV	Placenta	0.051	0.057	0.003	0.121	0.232
	Maternal blood	0.038	0.048	0.002	0.135	0.223
29. 22	Cord blood	0.012	0.016	0.000	0.035	0.063
NV	Placenta	0.235	0.239	0.0007	0.347	0.821
	Maternal blood	0.016	0.021	0.001	0.058	0.096
30. 25	Cord blood	0.041	0.050	0.0003	0.079	0.170
V	Placenta	0.035	0.047	0.002	0.098	0.182
	Maternal blood	0.211	0.160	0.000	0.267	0.638
31. 28	Cord blood	0.032	0.034	0.0008	0.056	0.122
V	Placenta	0.076	0.069	0.000	0.098	0.243

Cont'd..

Continued..

	Maternal blood	0.039	0.040	0.0005	0.100	0.179
32. 26	Cord blood	0.011	0.014	0.0003	0.043	0.068
NV	Placenta	0.237	0.169	0.000	0.279	0.681
	Maternal blood	0.091	0.099	0.001	0.212	0.403
33. 22	Cord blood	0.023	0.027	0.0003	0.059	0.109
V	Placenta	0.180	0.189	0.000	0.285	0.654
	Maternal blood	0.123	0.098	0.003	0.238	0.462
34. 22	Cord blood	0.065	0.027	0.0003	0.050	0.142
V	Placenta	0.039	0.036	0.0005	0.088	0.163
	Maternal blood	0.143	0.152	0.001	0.282	0.578
35. 26	Cord blood	0.113	0.091	0.008	0.159	0.371
V	Placenta	0.132	0.107	0.000	0.145	0.384
	Maternal blood	0.094	0.108	0.001	0.244	0.447
36. 23	Cord blood	0.013	0.016	0.0003	0.041	0.070
V	Placenta	0.107	0.070	0.002	0.168	0.347
	Maternal blood	0.117	0.108	0.002	0.231	0.458
37. 23	Cord blood	0.039	0.039	0.0003	0.070	0.148
V	Placenta	0.217	0.189	0.001	0.253	0.660
	Maternal blood	0.105	0.117	0.002	0.251	0.475
38. 24	Cord blood	0.014	0.016	0.0001	0.041	0.071
V	Placenta	0.236	0.194	0.000	0.273	0.703
	Maternal blood	0.095	0.133	0.003	0.209	0.440
39. 21	Cord blood	0.015	0.019	0.0001	0.036	0.070
V	Placenta	0.046	0.028	0.0004	0.075	0.149
	Maternal blood	0.037	0.046	0.0008	0.018	0.101
40. 30	Cord blood	0.012	0.015	0.0005	0.079	0.106
V	Placenta	0.042	0.113	0.000	0.119	0.274
	Maternal blood	0.040	0.041	0.000	0.068	0.149
41. 25	Cord blood	0.017	0.019	0.000	0.047	0.083
V	Placenta	0.193	0.022	0.0003	0.069	0.284
	Maternal blood	0.087	0.080	0.002	0.205	0.374
42. 28	Cord blood	0.050	0.049	0.0001	0.059	0.158
NV	Placenta	0.009	0.009	0.0001	0.079	0.097

Cont'd..

Continued..

	Maternal blood	0.093	0.058	0.003	0.178	0.323
43. 27	Cord blood	0.041	0.042	0.002	0.037	0.122
V	Placenta	0.031	0.031	0.0007	0.055	0.117
	Maternal blood	0.026	0.032	0.001	0.074	0.133
44. 29	Cord blood	0.012	0.016	0.0002	0.038	0.067
NV	Placenta	0.029	0.031	0.0007	0.050	0.110
	Maternal blood	0.025	0.028	0.0002	0.098	0.151
45. 25	Cord blood	0.014	0.018	0.0005	0.044	0.076
NV	Placenta	0.034	0.039	0.001	0.085	0.159
	Maternal blood	0.046	0.058	0.002	0.064	0.170
46. 25	Cord blood	0.022	0.022	0.000	0.054	0.098
NV	Placenta	0.024	0.025	0.0007	0.069	0.118
	Maternal blood	0.028	0.036	0.001	0.082	0.147
47. 28	Cord blood	0.015	0.018	0.0002	0.039	0.072
NV	Placenta	0.016	0.018	0.0003	0.056	0.090
	Maternal blood	0.037	0.039	0.0005	0.078	0.154
48. 25	Cord blood	0.028	0.031	0.0003	0.052	0.111
V	Placenta	0.017	0.019	0.0003	0.049	0.085
	Maternal blood	0.025	0.028	0.0005	0.069	0.122
49. 26	Cord blood	0.019	0.021	0.0002	0.048	0.088
V	Placenta	0.011	0.014	0.0003	0.038	0.063
	Maternal blood	0.0211	0.096	0.000	0.029	0.336
50. 26.	Cord blood	0.139	0.094	0.000	0.161	0.394
V	Placenta	0.065	0.048	0.000	0.084	0.197

NV = Non-vegetarian

V = Vegetarian

* (Whole basis)

** (Whole basis)

*** (Wet basis)

TABLE-16: CONCENTRATION OF DDT AND ITS METABOLITES IN MATERNAL BLOOD, UMBILICAL CORD BLOOD AND PLACENTAL TISSUE OF WOMEN WITH PREMATURE LABOUR AND TOXEMIA OF PREGNANCY(ppm).

S. No.	Age/ Dietetic habit/ Pregnancy	Tissue	p,p'-DDE	o,p'-DDT	p,p'-DDD	p,p'-DDT	Total DDT
	38	*Maternal blood	0.073	0.052	0.001	0.130	0.256
1.	V	**Cord blood	0.021	0.017	0.000	0.036	0.074
	P	***Placenta	0.063	0.045	0.000	0.090	0.198
	35	Maternal blood	0.114	0.077	0.000	0.219	0.410
2.	V	Cord blood	0.061	0.016	0.000	0.047	0.124
	P	Placenta	0.054	0.066	0.000	0.123	0.243
	27	Maternal blood	0.064	0.036	0.000	0.091	0.191
3.	V	Cord blood	0.038	0.024	0.000	0.007	0.069
	T	Placenta	0.051	0.033	0.000	0.081	0.165
	28	Maternal blood	0.121	0.067	0.000	0.219	0.407
4.	NV	Cord blood	0.046	0.029	0.000	0.057	0.132
	T	Placenta	0.066	0.042	0.002	0.087	0.197
	22	Maternal blood	0.105	0.061	0.000	0.156	0.322
5.	V	Cord blood	0.046	0.031	0.000	0.057	0.134
	T	Placenta	0.075	0.042	0.000	0.096	0.213
	29	Maternal blood	0.064	0.037	0.000	0.091	0.192
6.	V	Cord blood	0.038	0.023	0.0004	0.067	0.128
	T	Placenta	0.054	0.330	0.003	0.627	1.014
	20	Maternal blood	0.075	0.085	0.000	0.188	0.348
7.	V	Cord blood	0.036	0.038	0.000	0.072	0.146
	T	Placenta	0.063	0.060	0.000	0.117	0.240
	29	Maternal blood	0.090	0.094	0.000	0.157	0.341
8.	NV	Cord blood	0.042	0.038	0.000	0.064	0.144
	T	Placenta	0.066	0.054	0.000	0.120	0.240
	25	Maternal blood	0.113	0.072	0.000	0.174	0.359
9.	NV	Cord blood	0.060	0.036	0.000	0.088	0.184
	T	Placenta	0.066	0.042	0.000	0.153	0.261

Cont'd..

Continued..

	21	Maternal blood	0.113	0.072	0.000	0.175	0.360
10.	NV	Cord blood	0.049	0.029	0.000	0.070	0.148
	T	Placenta	0.078	0.045	0.000	0.123	0.246
	21	Maternal blood	0.123	0.063	0.000	0.155	0.341
11.	V	Cord blood	0.052	0.030	0.000	0.068	0.150
	P	Placenta	0.081	0.045	0.000	0.048	0.174
	20	Maternal blood	0.064	0.045	0.000	0.095	0.204
12.	NV	Cord blood	0.051	0.034	0.0004	0.065	0.150
	T	Placenta	0.075	0.045	0.000	0.099	0.219
	35	Maternal blood	0.141	0.088	0.000	0.064	0.293
13.	NV	Cord blood	0.037	0.024	0.000	0.046	0.107
	T	Placenta	0.084	0.042	0.000	0.090	0.216
	24	Maternal blood	0.217	0.129	0.004	0.292	0.642
14.	NV	Cord blood	0.076	0.052	0.0008	0.119	0.248
	T	Placenta	0.069	0.042	0.000	0.096	0.207
	25	Maternal blood	0.192	0.129	0.000	0.292	0.613
15.	NV	Cord blood	0.055	0.032	0.000	0.071	0.158
	T	Placenta	0.114	0.069	0.000	0.288	0.471
	27	Maternal blood	0.217	0.708	0.004	0.280	1.209
16.	NV	Cord blood	0.085	0.050	0.0008	0.124	0.260
	T	Placenta	0.069	0.057	0.000	0.168	0.294
	26	Maternal blood	0.190	0.092	0.000	0.198	0.480
17.	NV	Cord blood	0.051	0.029	0.000	0.068	0.148
	T	Placenta	0.078	0.057	0.000	0.174	0.309
	21	Maternal blood	0.158	0.091	0.002	0.219	0.470
18.	V	Cord blood	0.060	0.039	0.000	0.088	0.187
	P	Placenta	0.087	0.054	0.000	0.117	0.258
	30	Maternal blood	0.108	0.051	0.000	0.131	0.290
19.	V	Cord blood	0.067	0.037	0.000	0.100	0.204
	T	Placenta	0.165	0.096	0.000	0.225	0.486
	22	Maternal blood	0.183	0.147	0.000	0.274	0.604
20.	NV	Cord blood	0.043	0.041	0.0004	0.158	0.242
	T	Placenta	0.123	0.084	0.000	0.210	0.417

Cont'd..

Continued..

	19	Maternal blood	0.005	0.099	0.000	0.151	0.255
21.	NV	Cord blood	0.069	0.041	0.000	0.093	0.203
	T	Placenta	0.102	0.072	0.000	0.147	0.321
	20	Maternal blood	0.139	0.092	0.000	0.195	0.426
22.	V	Cord blood	0.074	0.059	0.000	0.120	0.253
	T	Placenta	0.096	0.054	0.000	0.129	0.279
	21	Maternal blood	0.164	0.108	0.001	0.233	0.506
23.	V	Cord blood	0.068	0.042	0.000	0.094	0.204
	P	Placenta	0.033	0.027	0.000	0.060	0.120
	21	Maternal blood	0.271	0.193	0.001	0.408	0.873
24.	NV	Cord blood	0.062	0.042	0.001	0.103	0.208
	T	Placenta	0.105	0.078	0.000	0.240	0.423
	22	Maternal blood	0.177	0.126	0.000	0.286	0.589
25.	V	Cord blood	0.057	0.042	0.000	0.105	0.204
	T	Placenta	0.036	0.030	0.000	0.072	0.138
	17	Maternal blood	0.178	0.133	0.000	0.298	0.609
26.	NV	Cord blood	0.062	0.048	0.0004	0.084	0.194
	T	Placenta	0.072	0.048	0.000	0.129	0.249
	22	Maternal blood	0.146	0.116	0.001	0.248	0.511
27.	V	Cord blood	0.058	0.049	0.000	0.111	0.218
	T	Placenta	0.066	0.063	0.000	0.135	0.264
	27	Maternal blood	0.179	0.123	0.001	0.283	0.586
28.	NV	Cord blood	0.063	0.034	0.000	0.082	0.179
	T	Placenta	0.093	0.063	0.000	0.138	0.294
	20	Maternal blood	0.243	0.210	0.001	0.613	1.067
29.	NV	Cord blood	0.036	0.028	0.000	0.063	0.127
	P	Placenta	0.117	0.084	0.000	0.174	0.375
	20	Maternal blood	0.151	0.098	0.000	0.258	0.507
30.	V	Cord blood	0.102	0.277	0.002	0.180	0.561
	T	Placenta	0.132	0.099	0.000	0.150	0.381
	30	Maternal blood	0.034	0.089	0.000	0.042	0.165
31.	NV	Cord blood	0.062	0.043	0.000	0.100	0.205
	T	Placenta	0.111	0.114	0.001	0.108	0.334

Cont'd..

Continued..

	27	Maternal blood	0.166	0.123	0.000	0.237	0.526
32.	V	Cord blood	0.044	0.032	0.000	0.063	0.139
	T	Placenta	0.027	0.027	0.000	0.024	0.078
	35	Maternal blood	0.161	0.123	0.000	0.229	0.513
33.	V	Cord blood	0.036	0.039	0.000	0.068	0.143
	T	Placenta	0.078	0.057	0.000	0.114	0.249
	25	Maternal blood	0.180	0.128	0.000	0.280	0.588
34.	V	Cord blood	0.102	0.089	0.0008	0.173	0.365
	T	Placenta	0.129	0.099	0.000	0.219	0.447
	26	Maternal blood	0.157	0.125	0.000	0.298	0.580
35.	V	Cord blood	0.046	0.030	0.000	0.069	0.145
	T	Placenta	0.057	0.045	0.000	0.108	0.210
	25	Maternal blood	0.039	0.106	0.000	0.097	0.242
36.	V	Cord blood	0.028	0.019	0.000	0.017	0.064
	T	Placenta	0.015	0.060	0.000	0.054	0.129
	25	Maternal blood	0.061	0.141	0.001	0.128	0.331
37.	NV	Cord blood	0.040	0.063	0.000	0.057	0.160
	T	Placenta	0.054	0.105	0.000	0.096	0.255
	23	Maternal blood	0.184	0.251	0.001	0.229	0.665
38.	V	Cord blood	0.074	0.100	0.001	0.092	0.267
	T	Placenta	0.057	0.111	0.000	0.102	0.270
	29	Maternal blood	0.201	0.907	0.000	0.227	1.335
39.	V	Cord blood	0.049	0.300	0.000	0.075	0.424
	T	Placenta	0.081	0.405	0.000	0.114	0.600
	30	Maternal blood	0.112	0.442	0.000	0.111	0.665
40.	V	Cord blood	0.038	0.158	0.000	0.036	0.232
	P	Placenta	0.066	0.330	0.000	0.081	0.477
	30	Maternal blood	0.165	0.315	0.001	0.188	0.669
41.	V	Cord blood	0.052	0.094	0.000	0.036	0.182
	P	Placenta	0.078	0.150	0.000	0.054	0.282
	18	Maternal blood	0.201	1.965	0.003	0.164	2.333
42.	V	Cord blood	0.102	0.650	0.001	0.059	0.812
	T	Placenta	0.183	0.900	0.000	0.123	1.206

Cont'd..

Continued..

	29	Maternal blood	0.255	1.500	0.003	0.297	2.055
43.	V	Cord blood	0.111	0.568	0.001	0.182	0.862
	T	Placenta	0.183	0.900	0.000	0.123	1.206
	25	Maternal blood	0.224	1.342	0.009	0.218	1.793
44.	V	Cord blood	0.096	0.379	0.000	0.095	0.570
	P	Placenta	0.129	0.804	0.002	0.087	1.022
	33	Maternal blood	0.247	1.547	0.001	0.227	2.022
45.	NV	Cord blood	0.089	0.559	0.003	0.067	0.718
	P	Placenta	0.093	0.804	0.002	0.087	0.986
	22	Maternal blood	0.067	0.077	0.000	0.039	0.183
46.	NV	Cord blood	0.042	0.063	0.000	0.016	0.121
	T	Placenta	0.066	0.141	0.002	0.066	0.275
	30	Maternal blood	0.069	0.159	0.001	0.109	0.338
47.	V	Cord blood	0.049	0.094	0.001	0.061	0.205
	T	Placenta	0.042	0.108	0.000	0.075	0.225
	25	Maternal blood	0.123	0.236	0.000	0.158	0.517
48.	V	Cord blood	0.032	0.063	0.000	0.044	0.139
	T	Placenta	0.042	0.093	0.000	0.057	0.192
	20	Maternal blood	0.119	0.276	0.000	0.178	0.573
49.	NV	Cord blood	0.048	0.117	0.002	0.075	0.242
	T	Placenta	0.078	0.216	0.000	0.135	0.429
	26	Maternal blood	0.185	0.434	0.003	0.281	0.903
50.	V	Cord blood	0.043	0.094	0.000	0.063	0.200
	T	Placenta	0.060	0.123	0.000	0.099	0.282

NV = Non-vegetarian
V = Vegetarian
P = Premature delivery
T = Toxemia of pregnancy.
* (Whole basis)
** (Whole basis)
*** (Wet basis)

log transformed values followed by multiple range test (Table 17b). The results indicate that mean total DDT in maternal blood (0.280 ppm) was higher than that of umbilical cord blood (0.133 ppm; $p < 0.001$) and placental tissue (0.268 ppm; $p < 0.001$).

The mean total DDT levels in maternal blood, umbilical cord blood and placental tissue of women with premature labour and toxemia of pregnancy were compared using one way analysis of variance on log transformed values followed by multiple range test (Table 18b). In women with premature labour, mean total DDT in maternal blood (0.820 ppm) was higher than that of cord blood (0.256 ppm; $p < 0.001$) and placental tissue (0.413 ppm; $p < 0.05$). In women with toxemia of pregnancy, mean total DDT in maternal blood (0.577 ppm) was higher than that of cord blood (0.227 ppm; $p < 0.001$) and placental tissue (0.348 ppm; $p < 0.01$).

The trend is:

Maternal blood > Placental tissue > Umbilical cord blood

The mean total DDT levels in maternal blood, umbilical cord blood and placental tissue of women with full term pregnancy, premature labour and toxemia of pregnancy were compared using one way analysis of variance on log transformed values followed by multiple range test (Table 19b). The results indicate that maternal blood total DDT levels in women with premature labour (0.819 ppm) and toxemia of pregnancy (0.573 ppm) were significantly higher than that of women with full term pregnancy (0.280 ppm; $p < 0.001$). No significant difference was observed in maternal blood total DDT levels of women with premature labour and toxemia of pregnancy. Similar trend was observed when mean total DDT

TABLE-17A: LEVELS OF DDT AND ITS METABOLITES IN MATERNAL BLOOD, UMBILICAL CORD BLOOD AND PLACENTAL TISSUE OF WOMEN WITH FULL TERM PREGNANCY (ppm).

Tissue	Range of concentrations and means ± S.D.				
	p,p'-DDE	o,p'-DDT	p,p'-DDD	p,p'-DDT	Total DDT
Maternal blood	0.007 - 0.274 0.070 ± 0.058 (50)	0.005 - 0.198 0.065 ± 0.046 (50)	0.000 - 0.004 0.001 ± 0.0009 (41)	0.018 - 0.392 0.143 ± 0.090 (50)	0.066 - 0.669 0.280 ± 0.169 (50) 0.231[a]
Umbilical cord blood	0.011 - 0.163 0.038 ± 0.037 (50)	0.007 - 0.139 0.035 ± 0.029 (50)	0.000 - 0.008 0.0008± 0.001 (39)	0.024 - 0.161 0.059 ± 0.031 (50)	0.047 - 0.404 0.133 ± 0.085 (50) 0.116[b]
Placental tissue	0.009 - 0.236 0.084 ± 0.073 (50)	0.009 - 0.239 0.074 ± 0.061 (50)	0.000 - 0.009 0.001 ± 0.002 (33)	0.038 - 0.347 0.108 ± 0.075 (50)	0.063 - 0.821 0.268 ± 0.194 (50) 0.213[c]

Figures in parentheses indicate the number of positive samples
Figures underlined indicate geometric mean values

TABLE-17b: RESULTS OF MULTIPLE RANGE TEST ON LOG TRANSFORMED VALUES

	b	c
c	p <0.001	-
a	p < 0.001	NS

NS = Statistically not significant

Fig.16 Scatter diagram showing correlation between total DDT concentrations in maternal blood and umbilical cord blood of women with full term pregnancy.

r = 0.208

n = 50

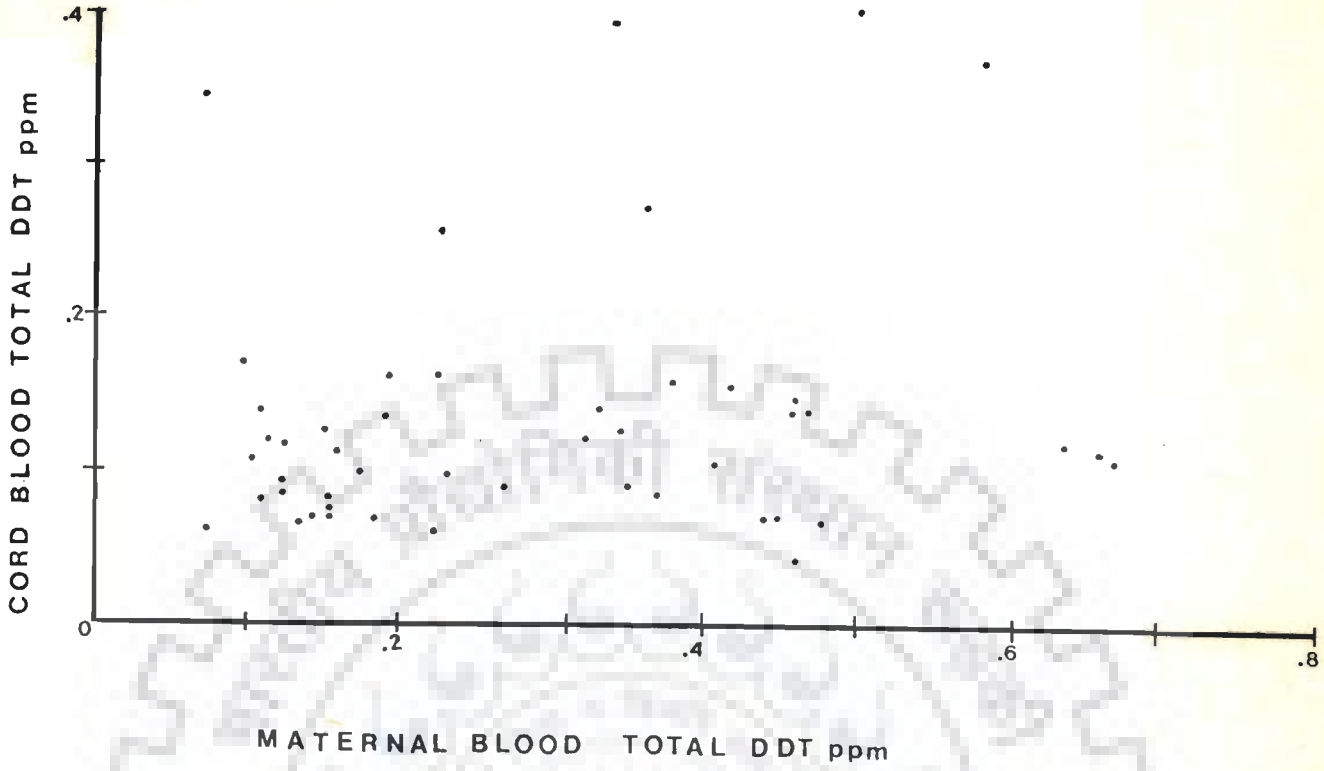
NS

Fig.17 Scatter diagram showing correlation between total DDT concentrations in maternal blood and placental tissue of women with full term pregnancy.

r = 0.177

n = 50

NS


CORD BLOOD TOTAL DDT ppm
.4
.2
0
.2
.4
.6
.8
MATERNAL BLOOD TOTAL DDT ppm

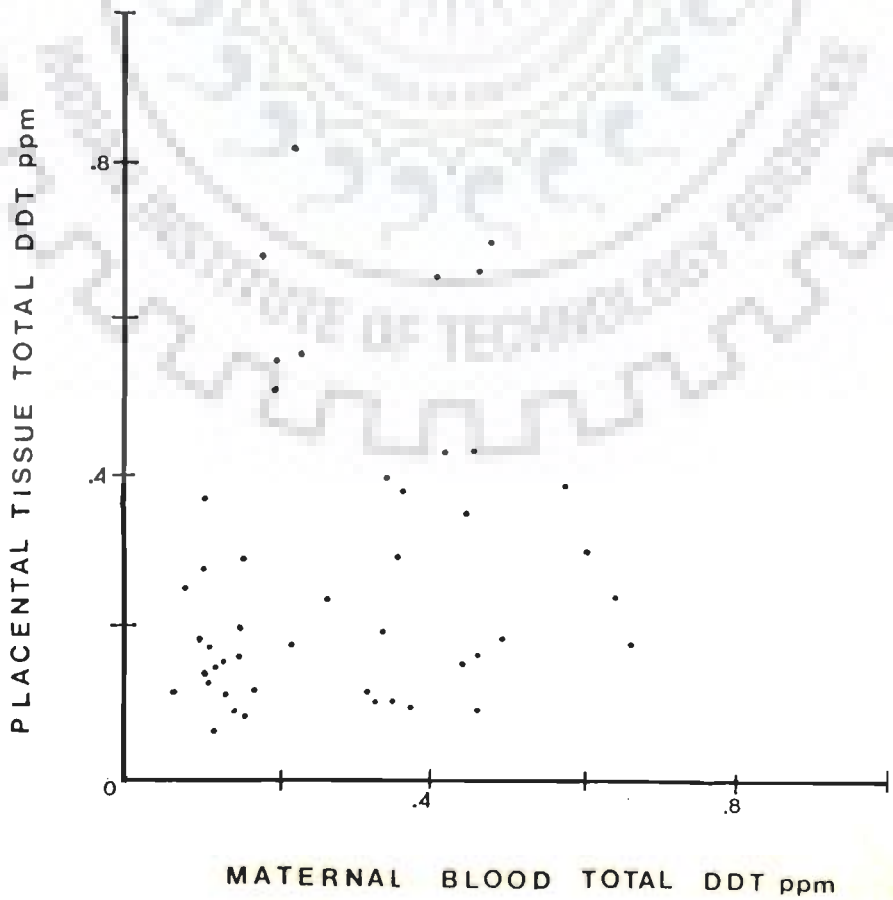

PLACENTAL TISSUE TOTAL DDT ppm
.8
.4
0
.4
.8
MATERNAL BLOOD TOTAL DDT ppm

TABLE-18a: LEVELS OF DDT AND ITS METABOLITES IN MATERNAL BLOOD, UMBILICAL CORD BLOOD AND PLACENTAL TISSUE OF WOMEN WITH PREMATURE LABOUR AND TOXEMIA OF PREGNANCY(ppm).

Compound	Range of concentrations and means ± S.D.					
	Premature labour N = 10			Toxemia of pregnancy N = 40		
	Maternal blood	Cord blood	Placenta	Maternal blood	Cord blood	Placenta
p,p'-DDE	0.073 - 0.247 0.162 ± 0.059 (10)	0.021 - 0.096 0.057 ± 0.023 (10)	0.033 - 0.129 0.080 ± 0.028 (10)	0.005 - 0.217 0.127 ± 0.061 (40)	0.028 - 0.111 0.057 ± 0.020 (40)	0.015 - 0.183 0.081 ± 0.037 (40)
o,p'-DDT	0.052 - 1.547 0.425 ± 0.554 (10)	0.016 - 0.559 0.136 ± 0.185 (10)	0.027 - 0.804 0.240 ± 0.309 (10)	0.036 - 1.965 0.239 ± 0.389 (40)	0.019 - 0.650 0.086 ± 0.134 (40)	0.027 - 0.900 0.129 ± 0.194 (40)
p,p'-DDD	0.000 - 0.009 0.002 ± 0.002 (7)	0.000 - 0.003 0.0004± 0.0009 (1)	0.000 - 0.004 0.0004± 0.0008 (2)	0.000 - 0.003 0.0005± 0.001 (11)	0.000 - 0.002 0.0003± 0.0005 (14)	0.000 - 0.003 0.0002± 0.0006 (4)
p,p'-DDT	0.111 - 0.613 0.231 ± 0.140 (10)	0.036 - 0.095 0.063 ± 0.023 (10)	0.048 - 0.174 0.092 ± 0.037 (10)	0.039 - 0.408 0.200 ± 0.085 (40)	0.007 - 0.182 0.083 ± 0.040 (40)	0.024 - 0.288 0.138 ± 0.095 (40)
Total DDT	0.256 - 2.022 0.819 ± 0.618 (10) 0.656[a]	0.074 - 0.718 0.257 ± 0.212 (10) 0.202[b]	0.120 - 1.022 0.414 ± 0.327 (10) 0.325[c]	0.165 - 2.333 0.573 ± 0.458 (40) 0.467[d]	0.064 - 0.862 0.228 ± 0.168 (40) 0.175[e]	0.078 - 1.206 0.348 ± 0.253 (40) 0.294[f]

N = Number of samples Figures in parentheses indicate the number of positive samples
Figures underlined indicate geometric mean values.

TABLE-18b: RESULTS OF MULTIPLE RANGE TEST ON LOG TRANSFORMED VALUES

	b	c	a
c	NS	-	
a	p<0.001	p<o.05	-

	e	f	d
f	p<0.001	-	
d	p<0.001	p<o.001	-

levels in umbilical cord blood and placental tissue of women with full term pregnancy, premature labour and toxemia of pregnancy were compared (Table 19c,d).

In women with full term pregnancy, no correlation was observed between total DDT concentrations in maternal blood and placental tissue (Fig. 17) and umbilical cord blood (Fig. 16). In case of toxemia of pregnancy, total DDT concentrations in maternal blood were positively correlated with cord blood ($r = 0.846$; $p < 0.001$; Fig.18) and placental tissue ($r = 0.698$; $p < 0.001$; Fig.19). Similarly, in women with premature labour, a positive correlation was observed between total DDT concentrations in maternal blood and cord blood ($r = 0.927$; $p < 0.001$; Fig.20) and placental tissue($r = 0.952$; $p < 0.001$; Fig.21).

The age wise distribution of DDT residues in pregnant women is presented in Table 20 and 21. The mean total DDT levels in maternal blood umbilical cord blood and placental tissue of two age groups were compared using 't' test. No statistically significant difference was observed between the two age groups. The total DDT concentrations in maternal blood and placental tissue of women with full term pregnancy showed no correlation with the age (Fig. 22 and 23). Similarly, total DDT concentrations in maternal blood and placental tissue of women with premature labour and toxemia of pregnancy showed no correlation with the age (Fig. 24 and 25).

The accumulation of DDT residues in the tissues of pregnant women in relation to dietetic habits is presented in Table 22 and 23. The mean total DDT levels in maternal blood, umbilical cord blood and placental tissue of vegetarians and

Fig.18 Scatter diagram showing correlation between total DDT concentrations in maternal blood and umbilical cord blood of women with toxemia of pregnancy.

$r = 0.846$

$n = 40$

$p < 0.001$

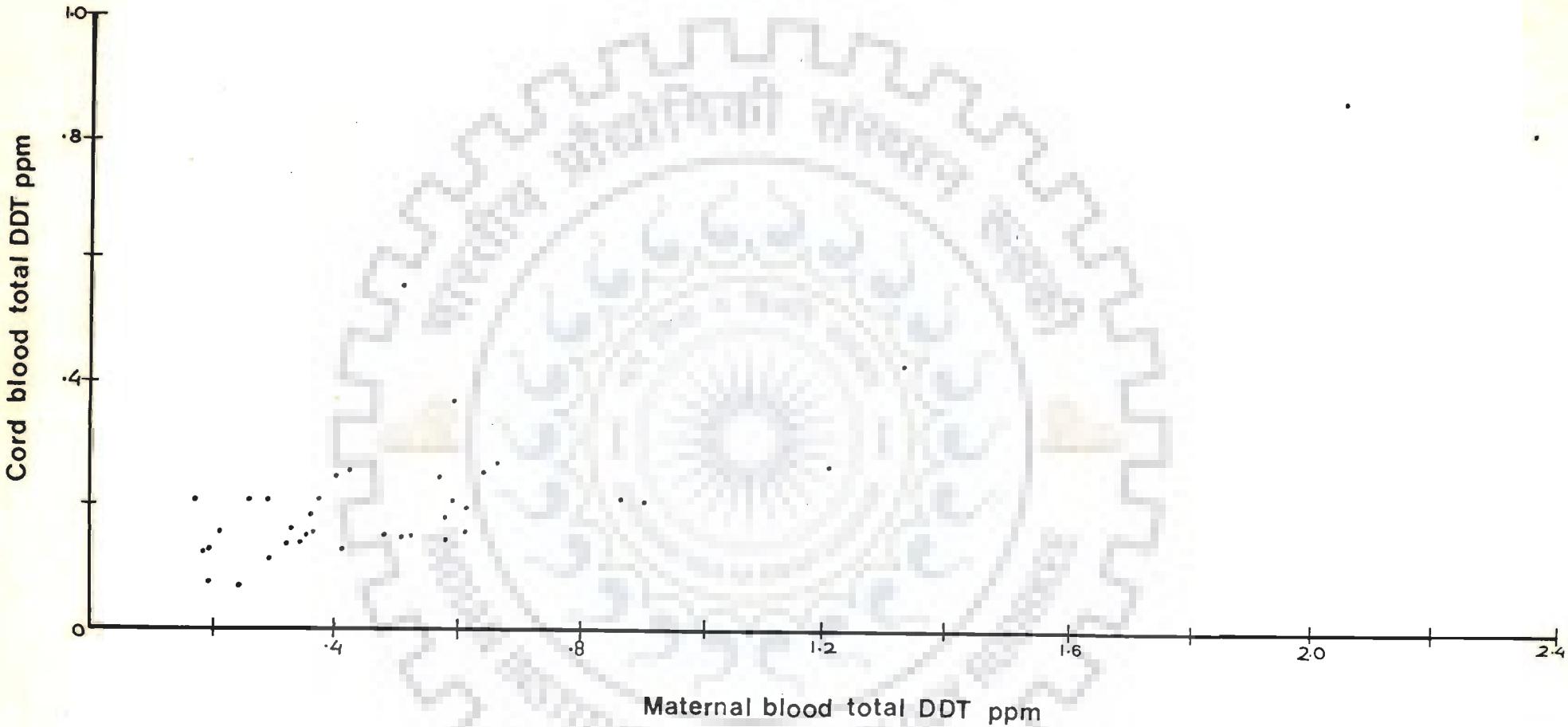

Cord blood total DDT ppm
1·0
·8
·4
0
·4
·8
1·2
1·6
2·0
2·4
Maternal blood total DDT ppm

Fig.19 Scatter diagram showing correlation between total DDT concentrations in maternal blood and placental tissue of women with toxemia of pregnancy.

$r = 0.698$

$n = 40$

$p < 0.001$

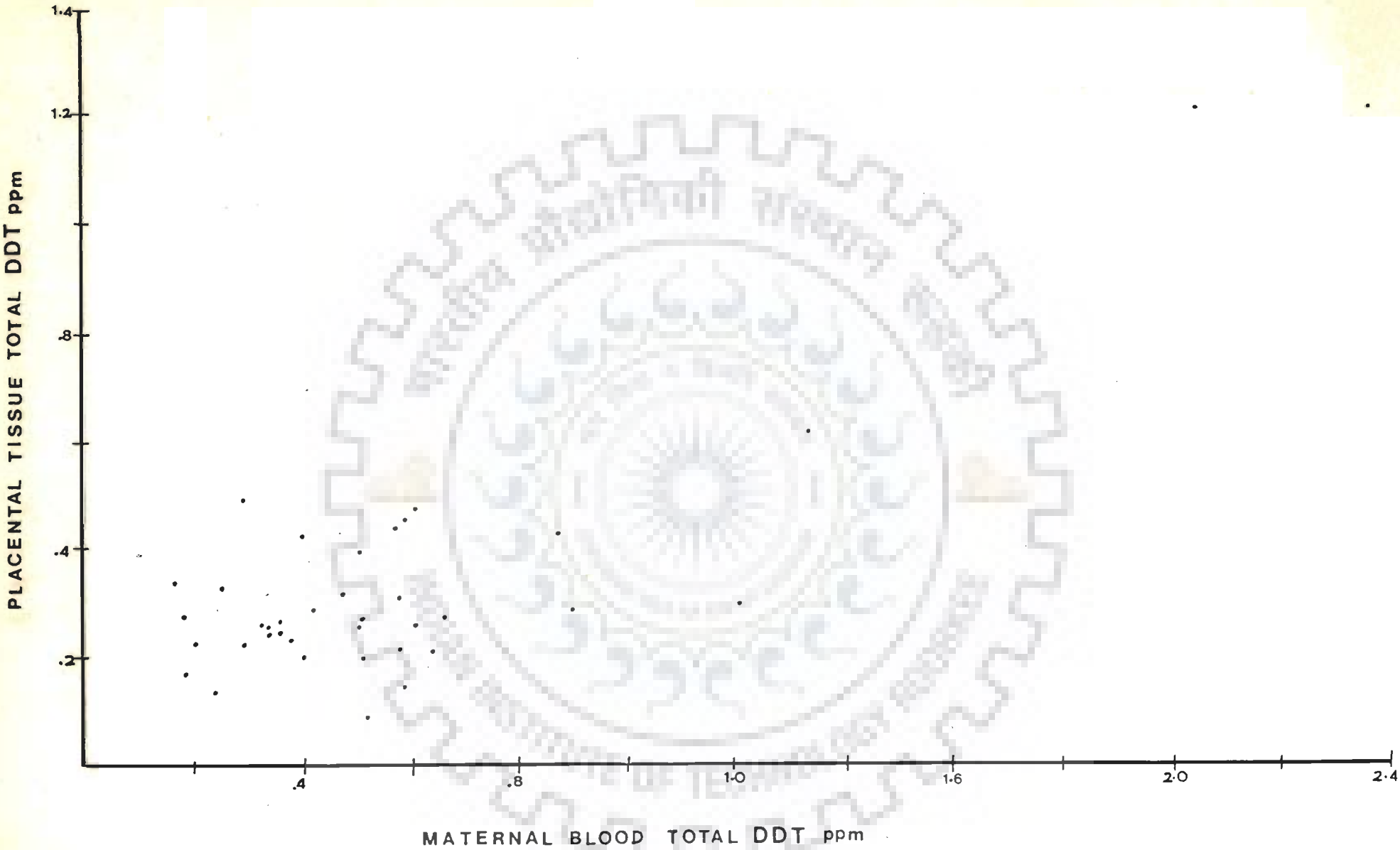

PLACENTAL TISSUE TOTAL DDT ppm
1.4
1.2
.8
.4
.2
.4
.8
1.0
1.6
2.0
2.4
MATERNAL BLOOD TOTAL DDT ppm

Fig.20 Scatter diagram showing correlation between total DDT concentrations in maternal blood and umbilical cord blood of women with premature labour.

$r = 0.927$

$n = 10$

$p < 0.001$

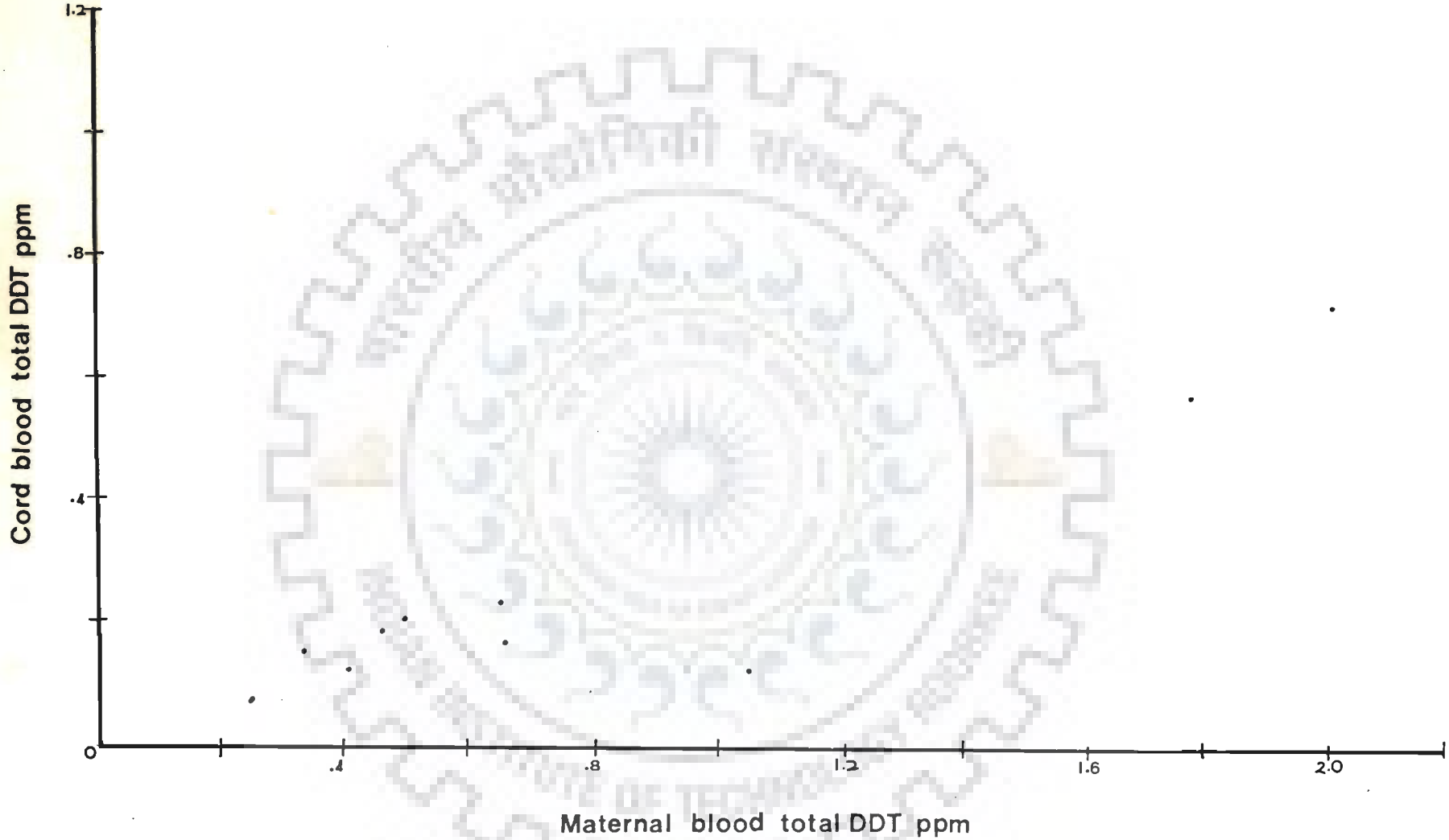

Cord blood total DDT ppm
1.2
.8
.4
0
.4
.8
1.2
1.6
2.0
Maternal blood total DDT ppm

Fig.21 Scatter diagram showing correlation between total DDT concentrations in maternal blood and placental tissue of women with premature labour.

$r = 0.952$

$n = 10$

$p < 0.001$

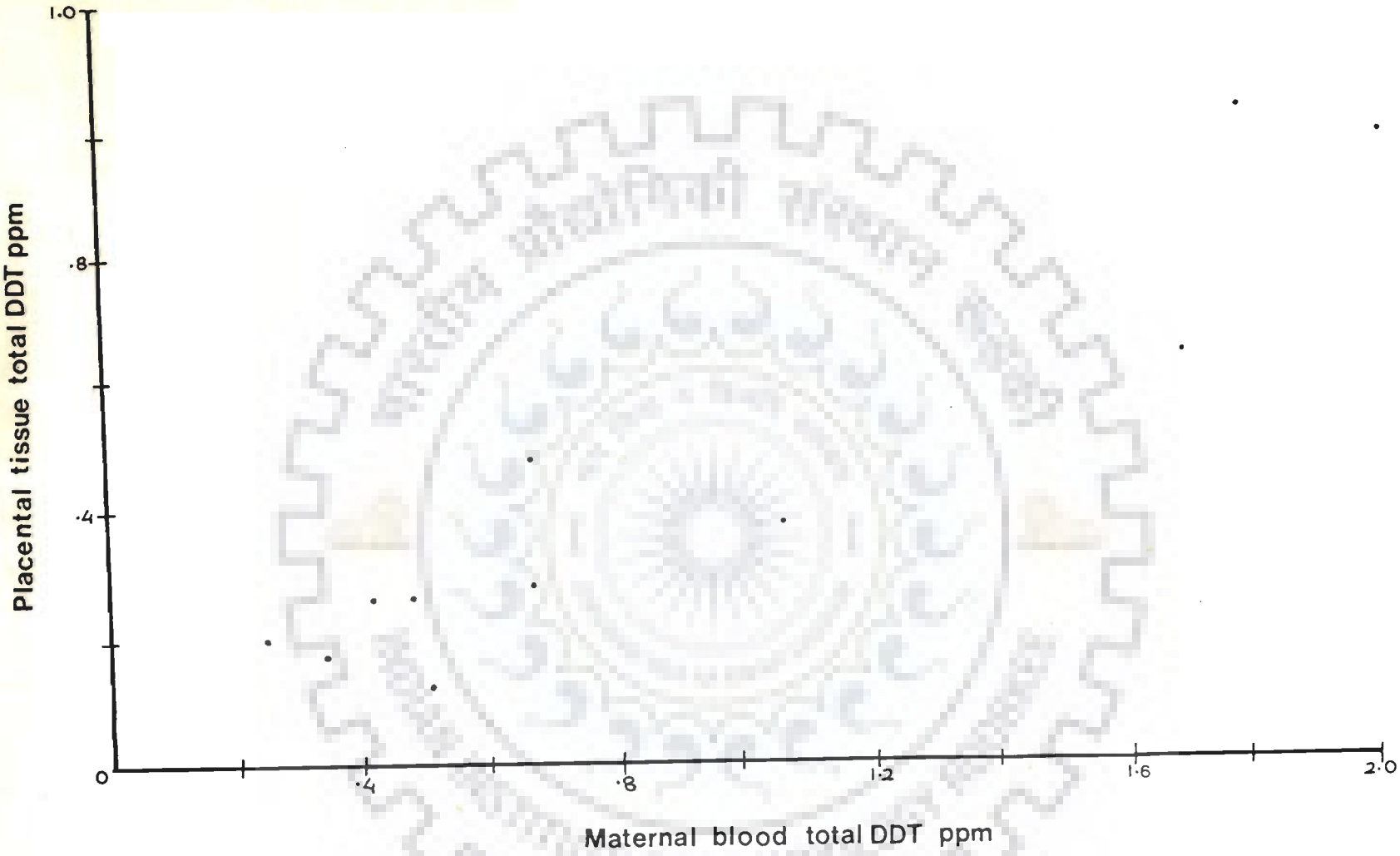

1.0
.8
.4
0
Placental tissue total DDT ppm
.4
.8
1.2
1.6
2.0
Maternal blood total DDT ppm

TABLE-19a: LEVELS OF TOTAL DDT IN MATERNAL BLOOD, UMBILICAL CORD BLOOD AND PLACENTAL TISSUE OF WOMEN WITH FULL TERM PREGNANCY, PREMATURE LABOUR AND TOXEMIA OF PREGNANCY (ppm).

Total DDT	Range of concentration and means ± S.D.		
	Full term N = 50	Premature N = 10	Toxemia N = 40
Maternal blood	0.066 - 0.669 0.280 ± 0.169(50) 0.231a	0.256 - 2.022 0.819 ± 0.618(10) 0.656b	0.165 - 2.373 0.573 ± 0.458(40) 0.467c
Umbilical cord blood	0.047 - 0.404 0.133 ± 0.085(50) 0.116d	0.074 - 0.718 0.257 ± 0.212(10) 0.202e	0.064 - 0.862 0.228 ± 0.168(40) 0.175f
Placental tissue	0.063 - 0.821 0.268 ± 0.194(50) 0.213g	0.120 - 1.022 0.414 ± 0.327(10) 0.235h	0.078 - 1.206 0.348 ± 0.253(40) 0.294i

N = Number of samples
Figures in parentheses indicate the number of positive samples.
Figures underlined indicate geometric mean values

TABLE-19b,c,d: RESULTS OF MULTIPLE RANGE TEST ON LOG TRANSFORMED VALUES

19b

a		c
c	p <0.001	-
b	p <0.001	NS

19c

d		f
f	p <0.001	-
e	p <0.001	NS

19d

g		i
i	p<0.05	-
h	NS	NS

NS = Statistically not significant

TABLE-20 : AGE WISE DISTRIBUTION OF DDT AND ITS METABOLITES IN MATERNAL UMBILICAL CORD BLOOD AND PLACENTAL TISSUE OF WOMEN WITH FULL TERM PREGNANCY (ppm).

Compound	Range of concentrations, means and ± S.D.					
	Age groups (yrs)					
	15 - 24 yrs N=24			25 - 30 yrs N=26		
	Maternal blood	Cord blood	Placenta	Maternal blood	Cord blood	Placenta
p,p'-DDE	0.008 - 0.189 0.069 ± 0.043 (24)	0.013 - 0.163 0.042 ± 0.040 (24)	0.011 - 0.236 0.093 ± 0.077 (24)	0.007 - 0.274 0.070 ± 0.071 (26)	0.011 - 0.139 0.034 ± 0.035 (26)	0.009 - 0.23 0.076 ± 0.07 (26)
o,p'-DDT	0.012 - 0.133 0.068 ± 0.039 (24)	0.012 - 0.139 0.038 ± 0.034 (24)	0.016 - 0.239 0.081 ± 0.066 (24)	0.005 - 0.198 0.063 ± 0.053 (26)	0.007 - 0.095 0.032 ± 0.025 (26)	0.009 - 0.20 0.068 ± 0.05 (26)
p,p'-DDD	0.000 - 0.004 0.002 ± 0.001 (20)	0.000 - 0.002 0.0003± 0.0004 (18)	0.000 - 0.004 0.0004± 0.001 (15)	0.000 - 0.003 0.0008± 0.0007 (21)	0.000 - 0.008 0.0006± 0.0001 (21)	0.000 - 0.00 0.001 ± 0.00 (18)
p,p'-DDT	0.046 - 0.392 0.169 ± 0.097 (24)	0.024 - 0.138 0.057 ± 0.028 (24)	0.051 - 0.347 0.125 ± 0.089 (24)	0.018 - 0.307 0.119 ± 0.079 (26)	0.030 - 0.161 0.062 ± 0.033 (26)	0.038 - 0.27 0.094 ± 0.05 (26)
TOTAL DDT	0.066 - 0.496 0.309a± 0.148 (24)	0.047 - 0.404 0.138b± 0.086 (24)	0.091 - 0.821 0.300c± 0.216 (24)	0.069 - 0.669 0.253d± 0.186 (26)	0.065 - 0.394 0.129e± 0.085 (26)	0.063 - 0.68 0.239f± 0.17 (26)

N = Number of samples.
Figures in parentheses indicate the number of positive samples.
a,d; b,e; c,f = Statistically not significant.

Fig.22 Scatter diagram showing correlation between total DDT concentrations in maternal blood and age in women with full term pregnancy.

r = -0.176

n = 50

NS

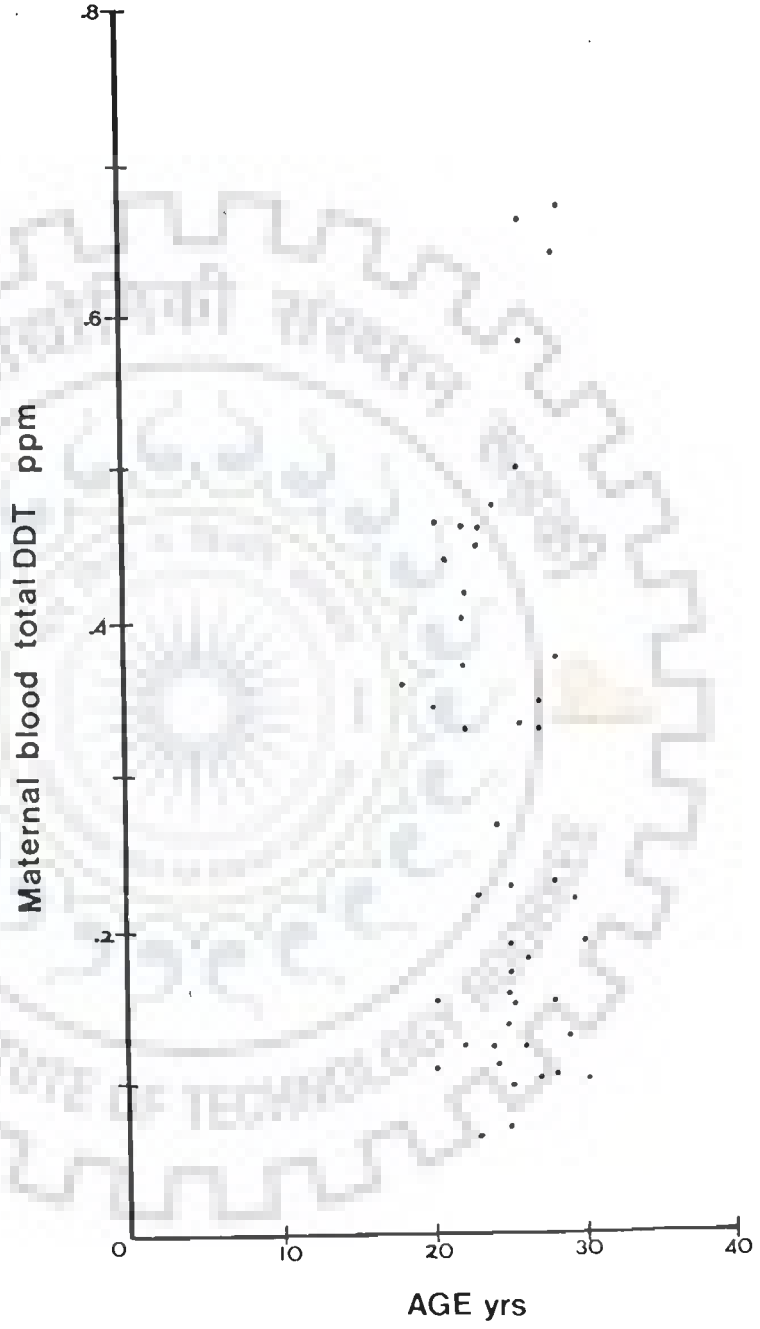

.8
.6
.4
.2
0
10
20
30
40
Maternal blood total DDT ppm
AGE yrs

Fig.23 Scatter diagram showing correlation between total DDT concentrations in placental tissue and age in women with full term pregnancy.

r = 0.069

n = 50

NS

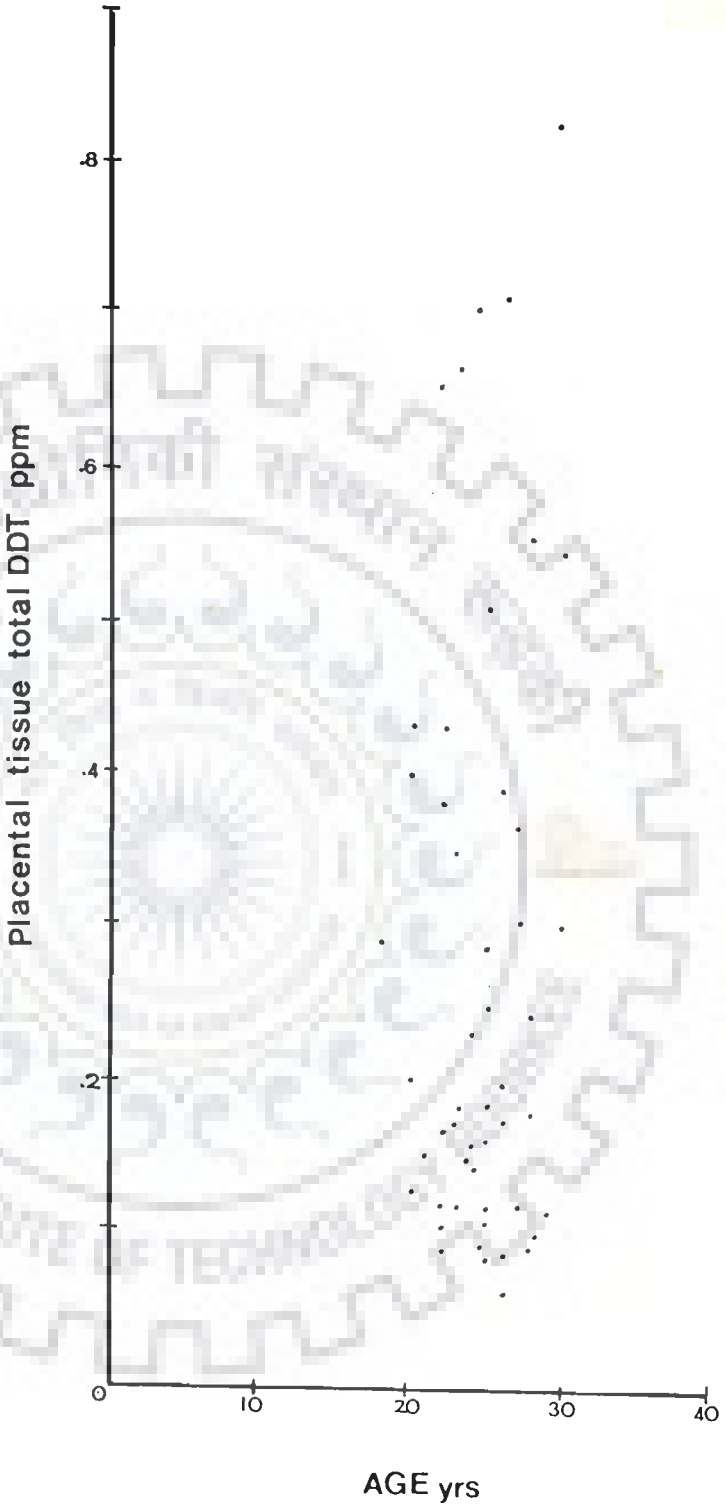

Placental tissue total DDT ppm
.8
.6
.4
.2
0
10
20
30
40
AGE yrs

Fig.24 Scatter diagram showing correlation between total DDT concentrations in maternal blood and age in women with premature labour and toxemia of pregnancy.

r = -0.009

n = 50

NS

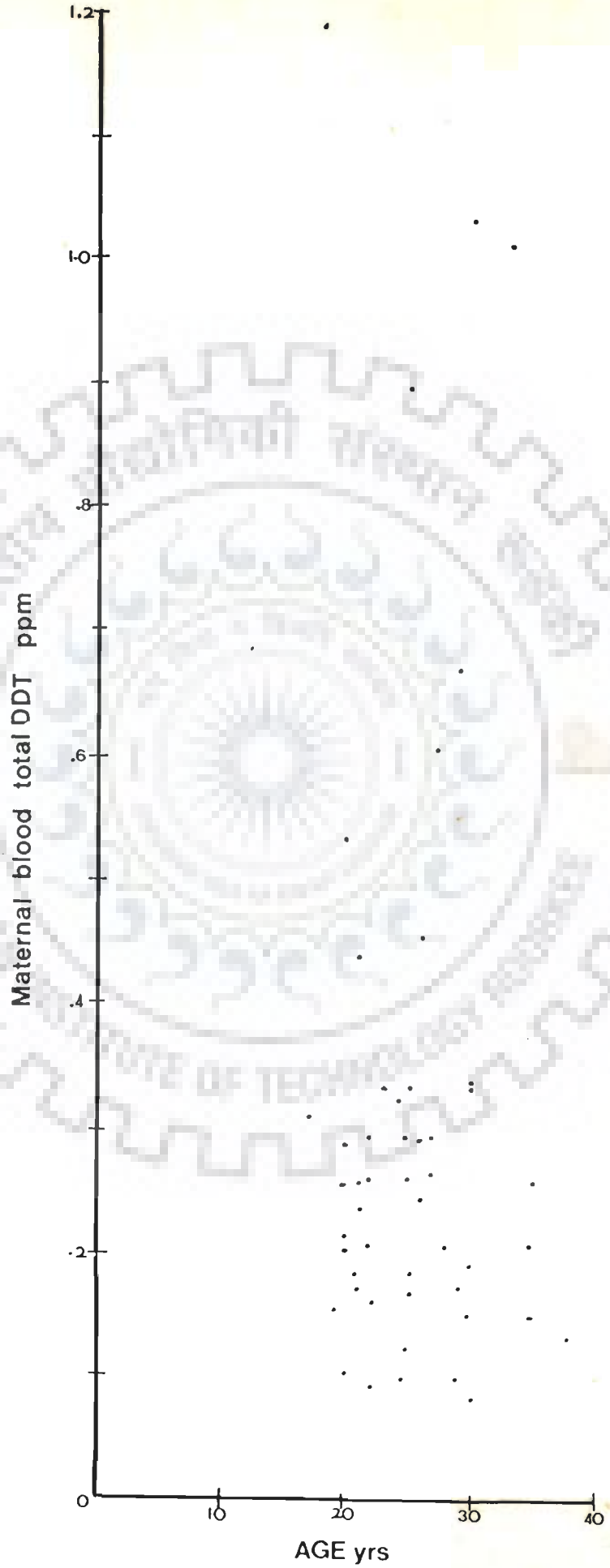

1.2
1.0
.8
.6
.4
.2
0
Maternal blood total DDT ppm
10
20
30
40
AGE yrs

Fig.25 Scatter diagram showing correlation between total DDT concentrations in placental tissue and age in women with premature labour and toxemia of pregnancy.

$r = 0.065$

$n = 50$

NS


2.0
1.0
.8
.6
.4
.2
0
10
20
30
40
PLACENTAL TISSUE TOTAL DDT ppm
AGE (YEARS)

TABLE-21: AGE WISE DISTRIBUTION OF DDT AND ITS METABOLITES IN MATERNAL BLOOD, UMBILICAL CORD BLOOD AND PLACENTAL TISSUE OF WOMEN WITH PREMATURE LABOUR AND TOXEMIA OF PREGNANCY(ppm).

	Range of concentrations, means and ± S.D.					
	Age groups (yrs)					
	15 - 25 yrs N=28			26 - 40 yrs N=22		
	Maternal blood	Cord blood	Placenta	Maternal blood	Cord blood	Placenta
p,p'-DDE	0.039 - 0.271 0.143 ± 0.063 (28)	0.032 - 0.102 0.060 ± 0.021 (28)	0.033 - 0.183 0.083 ± 0.036 (28)	0.034 - 0.255 0.142 ± 0.061 (22)	0.021 - 0.111 0.053 ± 0.020 (22)	0.042 - 0.183 0.078 ± 0.036 (22)
o,p'-DDT	0.045 - 1.965 0.235 ± 0.413 (28)	0.019 - 0.650 0.090 ± 0.134 (28)	0.027 - 0.900 0.129 ± 0.208 (28)	0.036 - 1.547 0.327 ± 0.449 (22)	0.016 - 0.568 0.106 ± 0.161 (22)	0.033 - 0.900 0.179 ± 0.242 (22)
p,p'-DDD	0.000 - 0.009 0.001 ± 0.002 (10)	0.000 - 0.002 0.0003± 0.0005 (10)	0.000 - 0.002 0.002 ± 0.000 (2)	0.000 - 0.004 0.0006± 0.0001 (8)	0.000 - 0.003 0.0002± 0.0006 (5)	0.000 - 0.003 0.0003± 0.0008 (4)
p,p'-DDT	0.039 - 0.613 0.221 ± 0.108 (28)	0.016 - 0.180 0.087 ± 0.039 (28)	0.048 - 0.288 0.126 ± 0.058 (28)	0.042 - 0.298 0.186 ± 0.080 (22)	0.007 - 0.182 0.069 ± 0.035 (22)	0.054 - 0.627 0.132 ± 0.118 (22)
Total DDT	0.183 - 2.333 0.601a± 0.462 (28)	0.064 - 0.812 0.237b± 0.160 (28)	0.120 - 1.206 0.338c± 0.240 (28)	0.165 - 2.055 0.655d± 0.541 (22)	0.069 - 0.862 0.228e± 0.197 (22)	0.078 - 1.206 0.390f± 0.299 (22)

N = Number of samples
Figures in parentheses indicate the number of positive samples
a,d; b,e; c,f = Statistically not significant.

TABLE-22: LEVELS OF DDT AND ITS METABOLITES IN MATERNAL BLOOD, UMBILICAL CORD BLOOD AND PLACENTAL TISSUE OF WOMEN WITH FULL TERM PREGNANCY AS RELATED TO DIETETIC HABITS (ppm).

Compound	Range of concentrations and means ± S.D.					
	Dietetic habits					
	Vegetarians (N=30)			Non-vegetarians (N=20)		
	Maternal blood	Cord blood	Placenta	Maternal blood	Cord blood	Placenta
p,p'-DDE	0.007 - 0.274 0.080 ± 0.068 (30)	0.011 - 0.139 0.035 ± 0.033 (30)	0.111 - 0.236 0.096 ± 0.075 (30)	0.008 - 0.166 0.048 ± 0.038 (20)	0.111 - 0.163 0.042 ± 0.043 (20)	0.009-0.237 0.066±0.068 (20)
o,p'-DDT	0.015 - 0.198 0.072 ± 0.050 (30)	0.007 - 0.095 0.032 ± 0.023 (30)	0.014 - 0.202 0.080 ± 0.062 (30)	0.009 - 0.188 0.053 ± 0.042 (20)	0.012 - 0.139 0.039 ± 0.037 (20)	0.009-0.239 0.066±0.061 (20)
p,p'-DDD	0.000 - 0.004 0.001 ± 0.001 (23)	0.000 - 0.008 0.0008± 0.0001 (25)	0.000 - 0.004 0.001 ± 0.001 (21)	0.000 - 0.103 0.0009± 0.0008 (18)	0.000 - 0.002 0.0003± 0.0004 (14)	0.000-0.009 0.0003±0.002 (12)
p,p'-DDT	0.029 - 0.314 0.146 ± 0.090 (30)	0.030 - 0.161 0.065 ± 0.034 (30)	0.038 - 0.285 0.108 ± 0.069 (30)	0.046 - 0.392 0.126 ± 0.094 (20)	0.024 - 0.138 0.052 ± 0.025 (20)	0.050-0.347 0.108±0.085 (20)
Total DDT	0.069 - 0.669 0.299a± 0.179 (30)	0.065 - 0.394 0.133b± 0.078 (30)	0.063 - 0.703 0.286c± 0.191 (30)	0.066 - 0.663 0.228d± 0.158 (20)	0.047 - 0.346 0.134e± 0.096 (20)	0.086-0.821 0.241f±0.199 (20)

N = Number of samples

Figures in parentheses indicate the number of positive samples.

a,d; b,e and c,f = Statistically not significant.

TABLE-23: LEVELS OF DDT AND ITS METABOLITES IN MATERNAL BLOOD, UMBILICAL CORD BLOOD AND PLACENTAL TISSUE OF WOMEN WITH PREMATURE LABOUR AND TOXEMIA OF PREGNANCY AS RELATED TO DIETETIC HABITS (ppm).

Compound	Range of concentrations and means ± S.D.					
	Dietetic habits					
	Vegetarians(N=29)			Non-vegetarians(N=21)		
	Maternal blood	Cord blood	Placenta	Maternal blood	Cord blood	Placenta
p,p'-DDE	0.039 - 0.255 0.140 ± 0.052 (29)	0.021 - 0.111 0.057 ± 0.024 (29)	0.015 - 0.183 0.078 ± 0.044 (29)	0.005 - 0.271 0.145 ± 0.074 (21)	0.036 - 0.089 0.055 ± 0.015 (21)	0.066 - 0.123 0.085 ± 0.020 (21)
o,p'-DDT	0.036 - 1.965 0.319 ± 0.484 (29)	0.016 - 0.650 0.118 ± 0.162 (29)	0.027 - 0.900 0.182 ± 0.255 (29)	0.045 - 1.547 0.215 ± 0.335 (21)	0.028 - 0.559 0.068 ± 0.114 (21)	0.042 - 0.216 0.109 ± 0.164 (21)
p,p'-DDD	0.000 - 0.009 0.0009± 0.001 (11)	0.000 - 0.002 0.0002± 0.0004 (7)	0.000 - 0.003 0.0002± 0.0006 (2)	0.000 - 0.004 0.0006± 0.001 (7)	0.000 - 0.003 0.0004± 0.0007 (8)	0.000 - 0.002 0.0003±0.0007 (4)
p,p'-DDT	0.091 - 0.298 0.197 ± 0.065 (29)	0.007 - 0.182 0.079 ± 0.043 (29)	0.024 - 0.627 0.120 ± 0.107 (29)	0.039 - 0.613 0.218 ± 0.131 (21)	0.016 - 0.158 0.079 ± 0.030 (21)	0.066 - 0.288 0.139 ± 0.055 (21)
Total DDT	0.191 - 2.333 0.657a± 0.540 (29)	0.064 - 0.862 0.255b± 0.203 (29)	0.078 - 1.206 0.380c± 0.321 (29)	0.165 - 1.209 0.579d± 0.431 (21)	0.107 - 0.718 0.203e± 0.126 (21)	0.197 - 0.986 0.334f± 0.169 (21)

N = Number of samples
Figures in parentheses indicate the number of positive samples
a,d; b,e and c,f = Statistically not significant.

Fig.26 Gas chromatographic analysis of DDT residues in milk on the column containing 1.5 % OV_{17} and 1.95 % QF_1 on Gas Chrom Q 100-120 mesh. Further details of operating conditions are given in Table 1.

Peak No.		
	1	p,p'-DDE
	2	o,p'-DDT
	3	p,p'-DDD
	4	p,p'-DDT

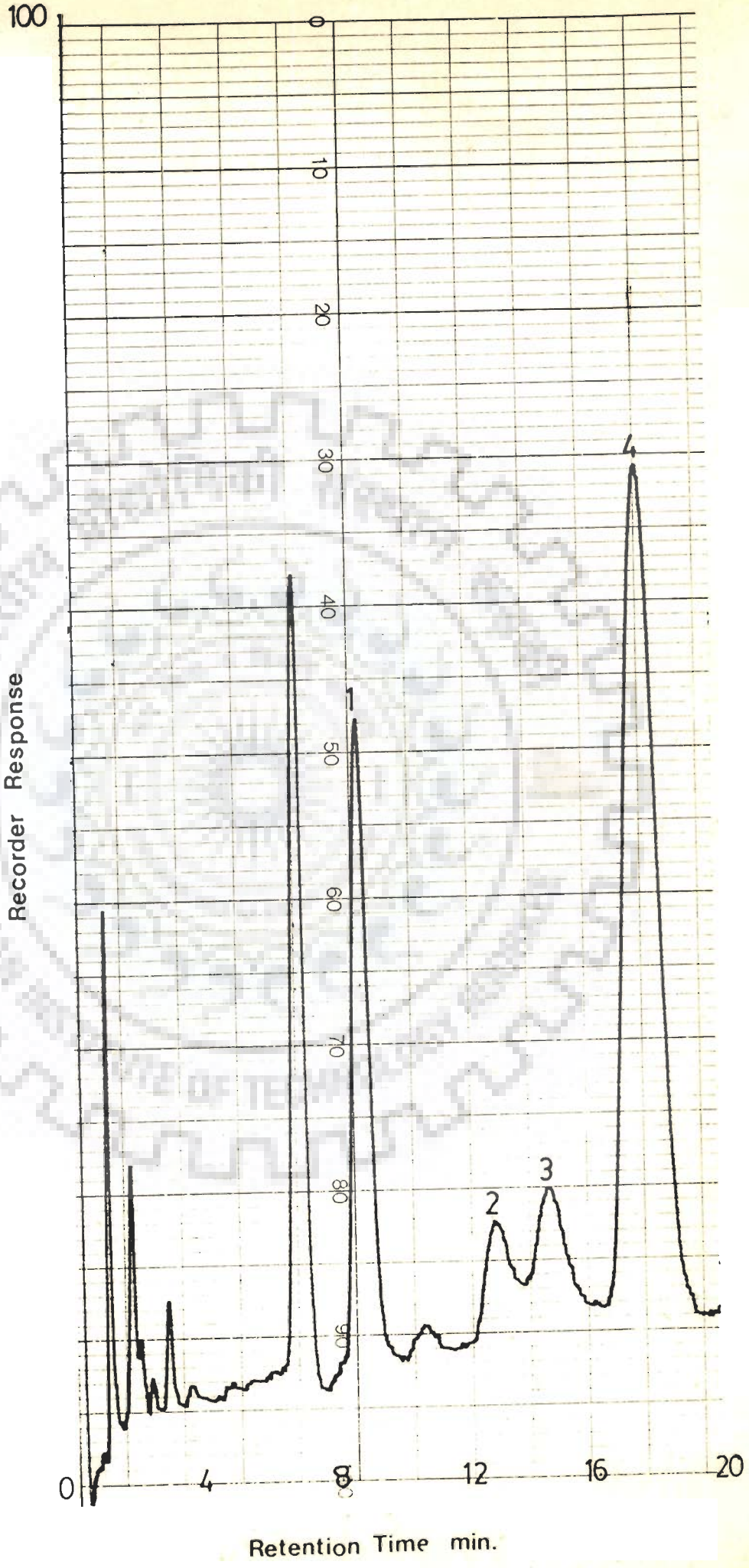

100
0
10
20
30
40
50
60
70
80
90
1
2
3
4
0
4
8
12
16
20
Recorder Response
Retention Time min.

TABLE-24a : CONCENTRATION OF DDT AND ITS METABOLITES IN BLOOD AND MILK (ppm).

S.No.	Tissue	p,p'-DDE	o,p'-DDT	p,p'-DDD	p,p'-DDT	Total DDT
1.	Blood*	0.113	0.122	0.003	0.224	0.462
	Milk**	1.234	0.709	0.004	0.856	2.803
2.	Blood	0.063	0.068	0.002	0.224	0.357
	Milk	1.364	0.598	0.003	1.577	3.542
3.	Blood	0.074	0.079	0.001	0.174	0.328
	Milk	1.344	0.708	0.013	1.999	4.064
4.	Blood	0.084	0.088	0.001	0.291	0.464
	Milk	4.244	3.415	0.000	3.380	11.039
5.	Blood	0.026	0.029	0.0002	0.086	0.141
	Milk	0.788	0.367	0.009	0.789	1.953
6.	Blood	0.028	0.018	0.0007	0.075	0.121
	Milk	0.250	0.127	0.000	0.356	0.733
7.	Blood	0.076	0.083	0.001	0.187	0.347
	Milk	1.610	0.183	0.004	0.907	2.704
8.	Blood	0.008	0.012	0.000	0.046	0.066
	Milk	0.814	0.306	0.007	0.842	1.969
9.	Blood	0.030	0.030	0.0005	0.048	0.108
	Milk	0.516	0.255	0.000	0.480	1.251
10.	Blood	0.274	0.198	0.000	0.197	0.669
	Milk	1.776	0.852	0.024	1.352	4.004
11.	Blood	0.050	0.054	0.0007	0.392	0.496
	Milk	0.206	0.084	0.002	0.281	0.573
12.	Blood	0.064	0.065	0.0005	0.099	0.228
	Milk	0.410	0.175	0.002	0.406	0.993
13.	Blood	0.014	0.015	0.0005	0.161	0.190
	Milk	0.588	0.295	0.002	0.324	1.209
14.	Blood	0.090	0.126	0.003	0.197	0.416
	Milk	0.528	0.258	0.002	0.497	1.285

Cont'd..

Continued..

15.	Blood	0.018	0.019	0.000	0.085	0.122
	Milk	0.698	0.215	0.000	0.557	1.470
16.	Blood	0.049	0.054	0.001	0.123	0.227
	Milk	0.522	0.257	0.003	0.620	1.402
17.	Blood	0.026	0.023	0.001	0.054	0.104
	Milk	0.562	0.312	0.005	0.613	1.492
18.	Blood	0.025	0.029	0.000	0.314	0.368
	Milk	0.280	0.117	0.000	0.255	0.652
19.	Blood	0.027	0.017	0.000	0.068	0.112
	Milk	0.260	0.117	0.005	0.409	0.791
20.	Blood[2]	0.022	0.018	0.0005	0.061	0.101
	Milk	0.454	0.252	0.002	0.522	1.230
21.	Blood	0.038	0.041	0.0005	0.069	0.148
	Milk	0.256	0.145	0.004	0.394	0.799
22.	Blood	0.084	0.079	0.0008	0.058	0.221
	Milk	0.462	0.297	0.000	0.679	1.438
23.	Blood	0.189	0.063	0.004	0.087	0.343
	Milk	0.526	0.271	0.002	0.496	1.295
24.	Blood	0.030	0.030	0.001	0.058	0.119
	Milk	0.478	0.300	0.004	0.644	1.426
25.	Blood	0.007	0.005	0.000	0.057	0.069
	Milk	0.266	0.118	0.005	0.246	0.635

* (Whole basis)

** (Whole basis)

TABLE-2B&b: LEVELS OF DDT AND ITS METABOLITES IN BLOOD AND MILK OF LACTATING WOMEN (ppm).

Compound	RANGE OF CONCENTRATIONS & MEANS ± S.D.		
	Milk N=25		Blood N=25
p,p'-DDE	0.206 - 4.244 0.817 ±0.840 (25)		0.007 - 0.274 0.060 ±0.060 (25)
o,p'-DDT	0.084 - 3.415 0.430 ±0.654 (25)		0.005 - 0.198 0.055 ±0.044 (25)
p,p'-DDD	0.000 - 0.024 0.004 ±0.005 (19)		0.000 - 0.003 0.0009 ±0.001 (19)
p,p'-DDT	0.246 - 1.099 0.779 ±0.686 (25)		0.046 - 0.392 0.137 ±0.095 (25)
Total DDT	0.573 - 11.039 2.030 ±2.131 (25)	t=4.16 p<0.001	0.066 - 0.669 0.253 ±0.162 (25)

N = Number of samples

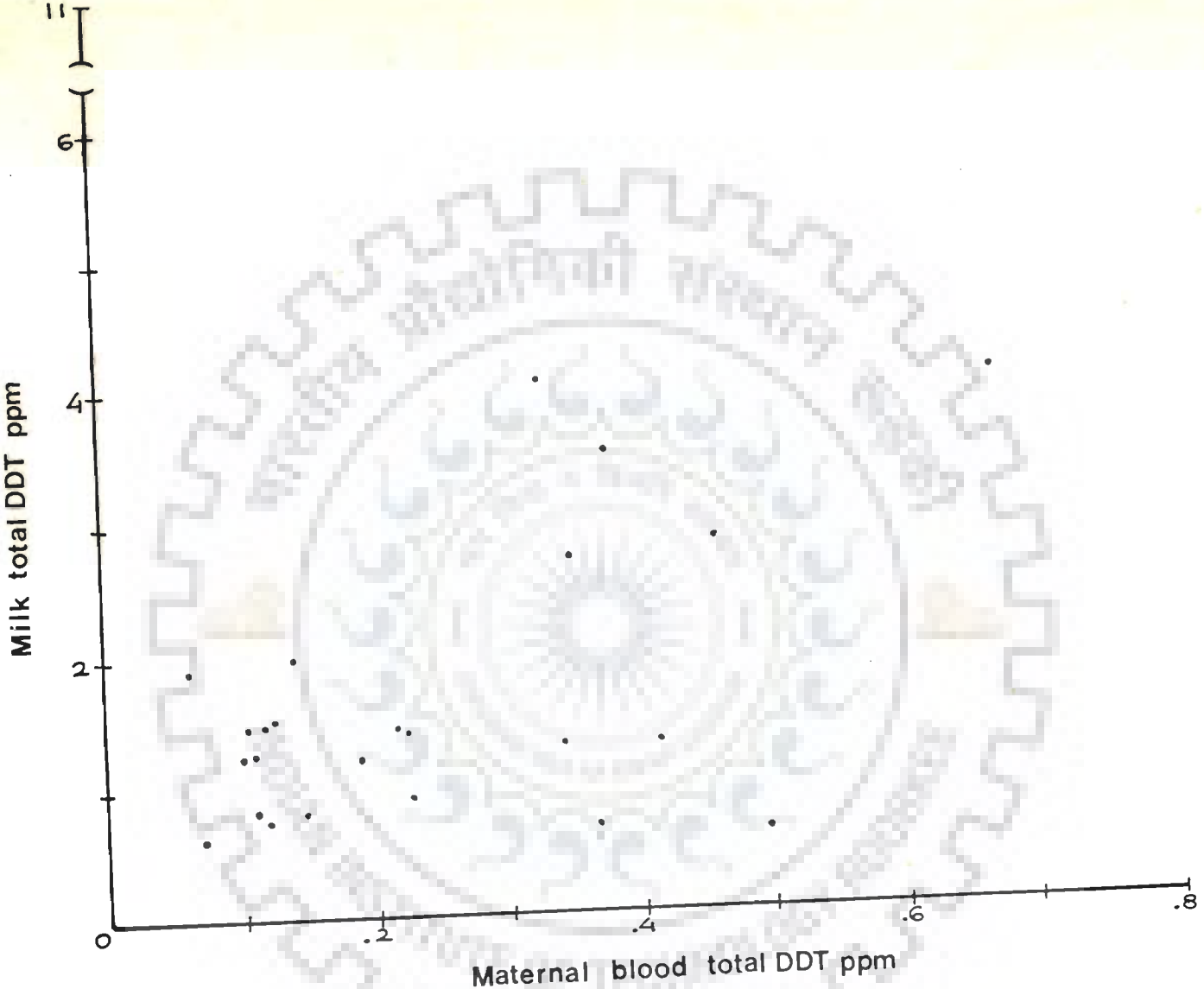
Figures in parentheses indicate the number of positive samples.

Fig.27 Scatter diagram showing correlation between total DDT concentrations in blood and milk of lactating women.

$r = 0.469$

$n = 25$

$p < 0.05$


11
6
4
2
0
Milk total DDT ppm
.2
.4
.6
.8
Maternal blood total DDT ppm

non-vegetarians were compared using 't' test. No significant difference in total DDT level was observed in vegetarians and non-vegetarians.

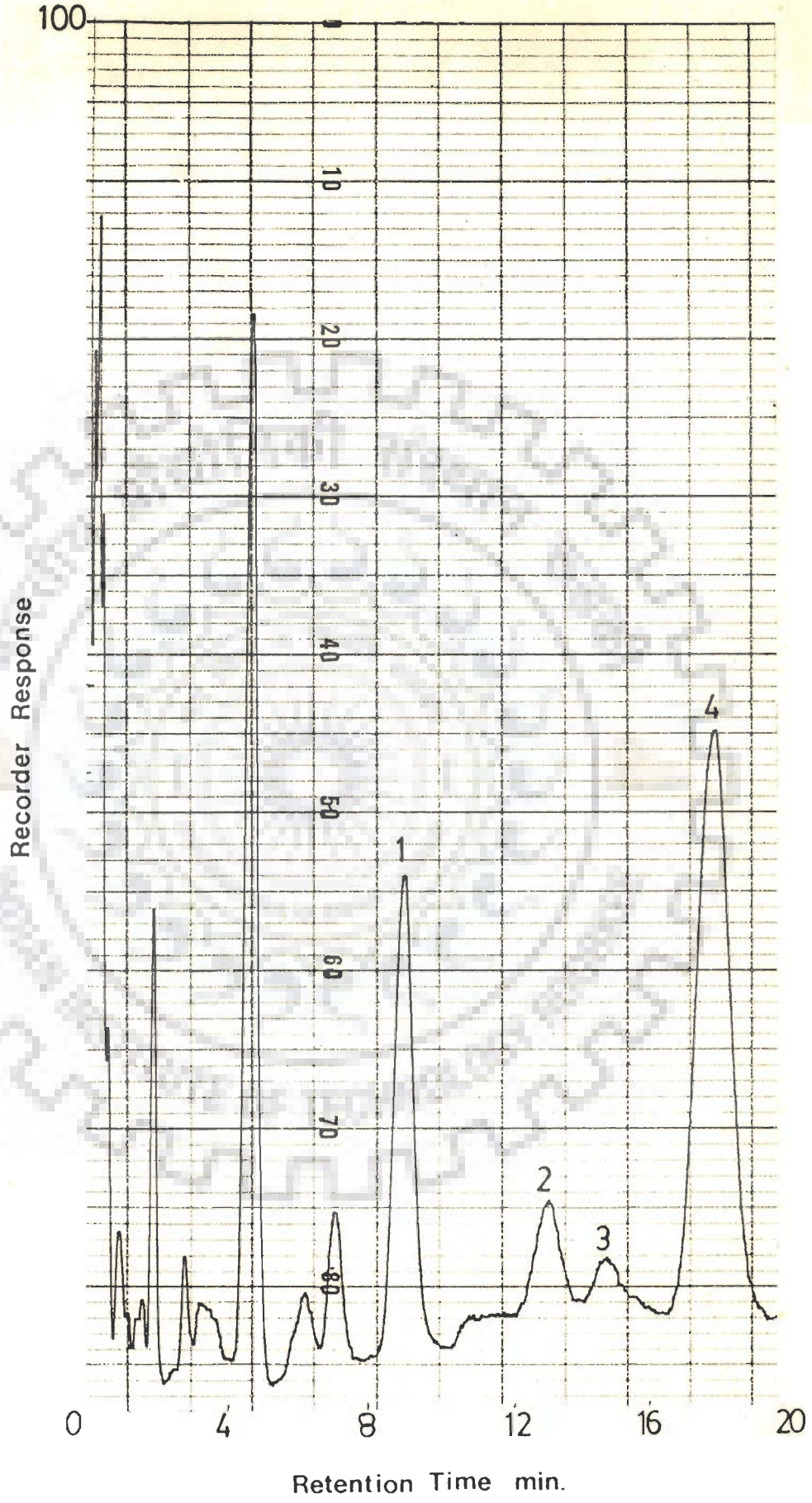
The DDT residues in blood, milk and umbilical cord blood of lactating women were estimated. The gas chromatographic pattern of DDT residues in milk is presented in Fig.26. Case wise findings are summarized in Table 24a. The statistical outcome of the data representing the range of concentrations and mean values with standard deviations is presented in Table 24b. The mean total DDT level in milk (2.030 ppm) was significantly higher than that of blood (0.253 ppm; $p < 0.001$). A positive correlation was observed between the total DDT concentrations in blood and milk ($r = 0.469$; $p < 0.05$; Fig.27).

The daily intake of DDT by an Indian child weighing 5 kg and consuming 1 kg of milk per day was calculated to be 0.406 mg/kg body weight/day.

4.4 *Residues in blood and uterine tissue*

The DDT residues in blood and uterine tissue of women undergoing autopsy and hysterectomy were determined. The gas chromatographic pattern of DDT residue in blood and uterine tissue of women undergoing autopsy and hysterectomy is presented in Fig. 28a,b and 29a,b. The case wise findings are summarized in Table 25 and 26. The statistical outcome of the data representing the range of concentrations and mean values with standard deviations is presented in Table 27.

The mean total DDT level in blood and uterine tissue of autopsy cases was found to be 0.219 ± 0.186 and 0.013 ± 0.055 ppm, respectively (Table 27 and Fig.30). When 't' test


100
10
20
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40
50
60
70
80
Recorder Response
1
2
3
4
0
4
8
12
16
20
Retention Time min.

Fig.28b Gas chromatographic analysis of DDT residues in uterine tissue of women undergoing autopsy on the column containing 1.5 % OV_{17} and 1.95 % QF_1 on Gas Chrom Q 100-120 mesh. Further details of operating conditions are given in Table 1.

Peak No.1	p,p'-DDE
2	o,p'-DDT
3	p,p'-DDD
4	p,p'-DDT

TABLE-25: CONCENTRATION OF DDT AND ITS METABOLITES IN BLOOD AND UTERINE TISSUE OF WOMEN WHO UNDERWENT AUTOPSY(ppm).

S. No.	Age	Tissue	p,p'-DDE	o,p'-DDT	p,p'-DDD	p,p'-DDT	Total DDT
1.	22	Blood*	0.041	0.586	0.001	0.139	0.767
		Uterus**	0.014	0.022	0.0007	0.064	0.100
2.	27	Blood	0.021	0.027	0.0007	0.067	0.116
		Uterus	0.008	0.009	0.0004	0.029	0.046
3.	17	Blood	0.048	0.062	0.000	0.111	0.221
		Uterus	0.013	0.016	0.000	0.042	0.071
4.	23	Blood	0.016	0.015	0.001	0.063	0.095
		Uterus	0.023	0.029	0.000	0.065	0.117
5.	27	Blood	0.027	0.037	0.001	0.103	0.168
		Uterus	0.036	0.043	0.000	0.088	0.167
6.	22	Blood	0.091	0.106	0.002	0.206	0.405
		Uterus	0.023	0.026	0.0005	0.061	0.110
7.	11	Blood	0.039	0.039	0.000	0.124	0.202
		Uterus	0.008	0.009	0.000	0.029	0.046
8.	30.	Blood	0.017	0.021	0.0007	0.069	0.108
		Uterus	0.032	0.039	0.001	0.033	0.105
9.	25	Blood	0.021	0.024	0.001	0.079	0.125
		Uterus	0.025	0.033	0.0002	0.056	0.114
10.	36	Blood	0.024	0.032	0.001	0.095	0.152
		Uterus	0.017	0.019	0.000	0.041	0.077
11.	25	Blood	0.036	0.049	0.001	0.103	0.189
		Uterus	0.028	0.031	0.000	0.053	0.112
12.	19	Blood	0.030	0.035	0.001	0.088	0.154
		Uterus	0.005	0.008	0.0007	0.022	0.036
13.	21	Blood	0.081	0.148	0.000	0.165	0.394
		Uterus	0.027	0.036	0.000	0.069	0.132
14.	16	Blood	0.043	0.049	0.001	0.105	0.198
		Uterus	0.026	0.031	0.0004	0.056	0.113

Cont'd..

Continued..

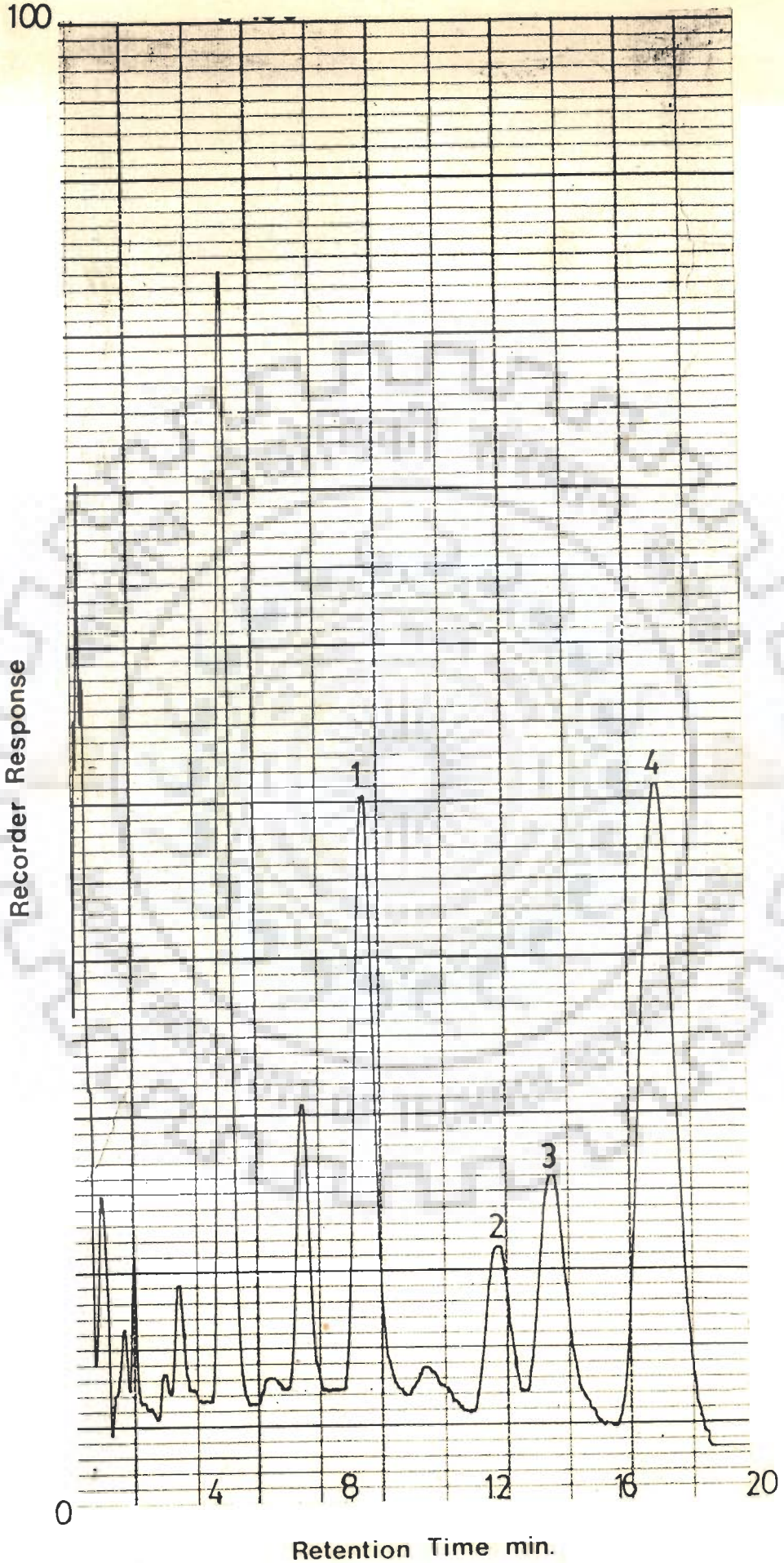
15.	22	Blood	0.063	0.069	0.000	0.128	0.260
		Uterus	0.033	0.035	0.003	0.059	0.127
16.	22	Blood	0.063	0.046	0.000	0.089	0.198
		Uterus	0.029	0.024	0.000	0.047	0.100
17.	20	Blood	0.034	0.037	0.000	0.105	0.176
		Uterus	0.018	0.012	0.003	0.030	0.063
18.	22	Blood	0.048	0.043	0.000	0.095	0.186
		Uterus	0.019	0.014	0.000	0.032	0.065
19.	35	Blood	0.012	0.014	0.001	0.056	0.083
		Uterus	0.017	0.014	0.0005	0.041	0.073
20.	28	Blood	0.037	0.050	0.002	0.101	0.190
		Uterus	0.033	0.042	0.006	0.062	0.143
21.	20	Blood	0.051	0.025	0.000	0.031	0.107
		Uterus	0.084	0.083	0.000	0.115	0.282
22.	40	Blood	0.251	0.225	0.002	0.291	0.769
		Uterus	0.052	0.047	0.0004	0.073	0.172
23.	27	Blood	0.017	0.012	0.000	0.023	0.052
		Uterus	0.034	0.036	0.0004	0.067	0.137
24.	18	Blood	0.031	0.023	0.0004	0.028	0.086
		Uterus	0.015	0.011	0.000	0.016	0.042
25.	25	Blood	0.037	0.015	0.000	0.021	0.073
		Uterus	0.011	0.008	0.000	0.011	0.030

* Blood (Whole basis)

** Uterus (Wet basis)

Fig.29a Gas chromatographic analysis of DDT residues in blood of women undergoing hysterectomy on the column containing 1.5 % OV_{17} and 1.95 % QF_1 on Gas Chrom Q 100-120 mesh. Further details of operating conditions are given in Table 1.

Peak No. 1 p,p'-DDE
2 o,p'-DDT
3 p,p'-DDD
4 p,p'-DDT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Recorder Response
1
2
3
4
0
4
8
12
16
20
Retention Time min.

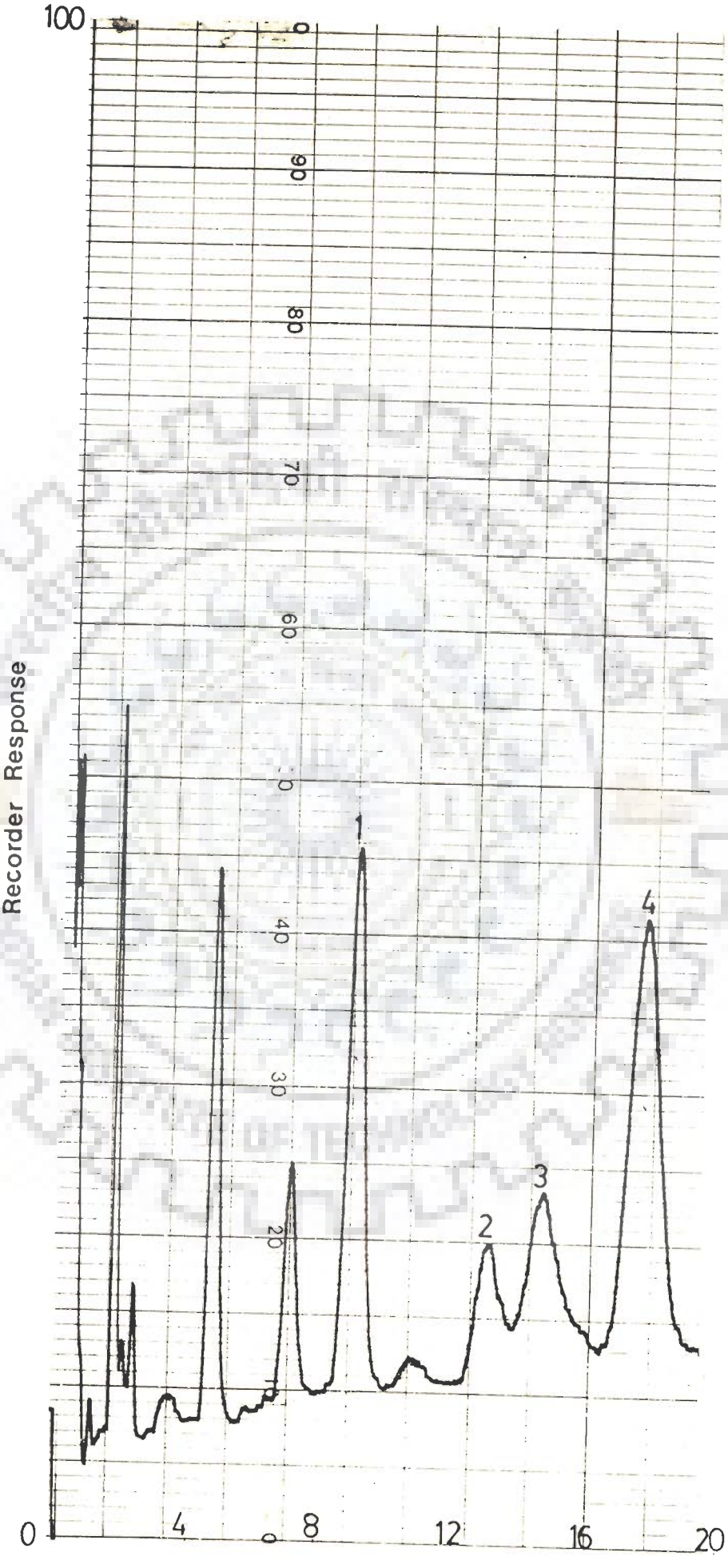

100
90
80
70
60
50
40
30
20
10
0
Recorder Response
1
2
3
4
0
4
8
12
16
20
Retention Time min.

Fig.29b Gas chromatographic analysis of DDT residues in uterine tissue of women undergoing hysterectomy on the column containing 1.5 % OV_{17} and 1.95% QF_1 on Gas Chrom Q 100-120 mesh. Further details of operating conditions are given in Table 1.

Peak No.	1	p,p'-DDE
	2	o,p'-DDT
	3	p,p'-DDD
	4	p,p'-DDT

TABLE-26: CONCENTRATION OF DDT AND ITS METABOLITES IN BLOOD AND UTERINE TISSUE OF WOMEN WHO UNDERWENT HYSTERECTOMY(ppm).

S. No.	Age	Tissue	p,p'-DDE	o,p'-DDT	p,p'-DDD	p,p'-DDT	Total DDT
1.	38	Blood*	0.143	0.874	0.042	0.044	1.103
		Uterus**	0.154	0.161	0.055	0.196	0.566
2.	38	Blood	0.181	0.161	0.016	0.179	0.537
		Uterus	0.302	0.080	0.021	0.247	0.650
3.	33	Blood	0.063	0.058	0.008	0.071	0.200
		Uterus	0.100	0.032	0.003	0.125	0.260
4.	45	Blood	0.096	0.064	0.014	0.301	0.474
		Uterus	0.061	0.032	0.013	0.140	0.246
5.	50	Blood	0.260	0.259	0.000	0.442	0.961
		Uterus	0.152	0.129	0.000	0.371	0.652
6.	44	Blood	0.236	0.051	0.006	0.610	0.903
		Uterus	0.200	0.155	0.006	0.592	0.953
7.	42	Blood	0.185-	0.071	0.013	0.524	0.793
		Uterus	0.180	0.064	0.016	0.553	0.813
8.	55	Blood	0.180	0.007	0.002	0.445	0.634
		Uterus	0.208	0.051	0.016	0.416	0.691
9.	50	Blood	0.247	0.216	0.000	0.268	0.723
		Uterus	0.327	0.324	0.000	0.491	1.142
10.	45	Blood	0.285	0.064	0.006	0.334	0.689
		Uterus	0.260	0.778	0.022	0.285	1.345
11.	38	Blood	0.223	0.642	0.011	0.254	1.130
		Uterus	0.279	1.174	0.019	0.510	1.982
12.	45	Blood	0.258	0.865	0.013	0.357	1.493
		Uterus	0.320	0.951	0.010	0.535	1.816
13.	48	Blood	0.239	0.832	0.020	0.267	1.358
		Uterus	0.298	0.854	0.017	0.526	1.695
14.	45	Blood	0.339	0.324	0.000	0.669	1.332
		Uterus	0.339	0.378	0.000	0.625	1.342

Cont'd..

15.	46	Blood	0.260	0.216	0.000	0.464	0.940
		Uterus	0.239	0.683	0.016	0.421	1.359
16.	45	Blood	0.100	0.103	0.000	0.250	0.453
		Uterus	0.139	0.129	0.000	0.264	0.532
17.	36	Blood	0.201	0.148	0.000	0.357	0.706
		Uterus	0.201	0.247	0.000	0.459	0.907
18.	48	Blood	0.193	0.138	0.000	0.393	0.723
		Uterus	0.136	0.121	0.000	0.285	0.542
19.	40	Blood	0.206	0.262	0.010	0.186	0.664
		Uterus	0.123	0.131	0.000	0.167	0.421
20.	33	Blood	0.180	0.220	0.011	0.156	0.567
		Uterus	0.086	0.091	0.000	0.117	0.294
21.	46	Blood	0.103	0.229	0.000	0.076	0.408
		Uterus	0.168	0.114	0.000	0.195	0.477
22.	26	Blood	0.107	0.146	0.000	0.173	0.426
		Uterus	0.134	0.091	0.000	0.156	0.381
23.	30	Blood	0.140	0.174	0.000	0.195	0.509
		Uterus	0.163	0.229	0.000	0.217	0.609
24.	50	Blood	0.144	0.172	0.000	0.217	0.533
		Uterus	0.233	0.286	0.000	0.271	0.790
25.	30	Blood	0.226	0.257	0.003	0.334	0.820
		Uterus	0.192	0.229	0.003	0.239	0.663

* Blood (Whole basis)

** Uterus (Wet basis).

was applied on mean total DDT levels, it was found that the mean total DDT in blood (0.219 ppm) was higher than that of uterine tissue (0.103 ppm; $p < 0.01$). No correlation was observed between total DDT concentrations in blood and uterine tissue in women who underwent autopsy (Fig.31).

The mean total DDT levels in blood and uterine tissue of women undergoing hysterectomy were found to be 0.763 ± 0.325 and 0.845 ± 0.402 ppm, respectively (Table 27 and Fig. 30). The mean total DDT values were compared using 't' test. No statistically significant difference was observed in mean total DDT values of blood and uterine tissue. A positive correlation was observed between total DDT concentrations in blood and uterine tissue in women who underwent hysterectomy ($r = 0.783$; $p < 0.001$; Fig.32).

The mean total DDT levels in blood and uterine tissue of women who underwent autopsy and hysterectomy were compared. The mean total DDT in the blood of hysterectomy cases (0.763 ppm) was about three times higher than that of autopsy cases (0.219 ppm; $p < 0.001$). Similarly, mean total DDT level in uterine tissue of hysterectomy cases (0.845 ppm) was about seven times higher than that of autopsy cases (0.103 ppm; $p < 0.001$).

The accumulation of DDT residues in artificially grouped ages is presented in Table 28 and 29. The mean total DDT levels were compared using 't' test. The results indicate that no statistically significant difference in total DDT was observed in two age groups in women who underwent autopsy and hysterectomy (Fig.33). The total DDT concentrations in blood and uterine tissue of women undergoing autopsy and hysterectomy showed no correlation with the age (Fig.34,35,36 and 37).

TABLE-27: LEVELS OF DDT AND ITS METABOLITES IN BLOOD AND UTERINE TISSUE OF WOMEN UNDERGOING AUTOPSY AND HYSTERECTOMY (ppm).

Compound	Range of concentrations and means ± S.D.			
	Autopsy		Hysterectomy	
	Blood N = 25	Uterus N = 25	Blood N = 25	Uterus N = 25
p,p'-DDE	0.012 - 0.251 0.047 ± 0.046 (25)	0.005 - 0.084 0.025 ± 0.016 (25)	0.063 - 0.339 0.191 ± 0.067 (25)	0.061 - 0.339 0.199 ± 0.079 (25)
o,p'-DDT	0.012 - 0.225 0.071 ± 0.117 (25)	0.008 - 0.083 0.026 ± 0.017 (25)	0.007 - 0.874 0.262 ± 0.256 (25)	0.032 - 1.174 0.300 ± 0.321 (25)
p,p'-DDD	0.002 - 0.004 0.0008± 0.0009 (15)	0.000 - 0.006 0.0005± 0.001 (14)	0.000 - 0.042 0.006 ± 0.009 (14)	0.000 - 0.055 0.008 ± 0.012 (12)
p,p'-DDT	0.021 - 0.291 0.099 ± 0.058 (25)	0.011 - 0.115 0.049 ± 0.023 (25)	0.044 - 0.669 0.303 ± 0.161 (25)	0.117 - 0.625 0.337 ± 0.162 (25)
Total DDT	0.052 - 0.769 0.219 ± 0.186 (25)	0.030 - 0.282 0.103 ± 0.055 (25)	0.200 - 1.493 0.763 ± 0.325 (25)	0.246 - 1.982 0.845 ± 0.492 (25)
	t=2.99 p<0.01		t=-0.70 NS	

N = Number of samples.
Figures in parentheses indicate the number of positive samples.

Fig.30 Bar diagram showing levels of DDT and its metabolites in blood and uterine tissue of women undergoing autopsy and hysterectomy.

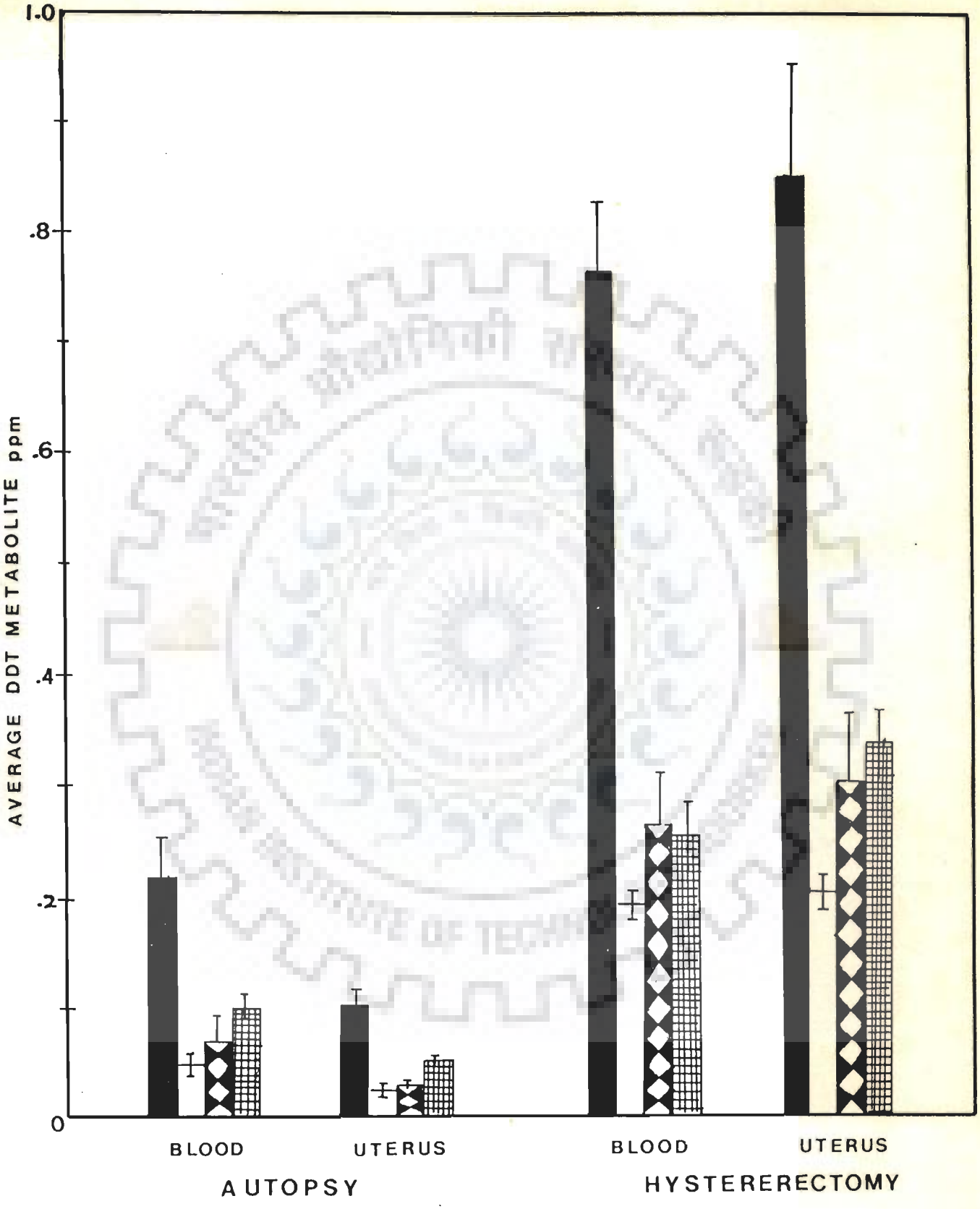

1.0
.8
.6
.4
.2
0
AVERAGE DDT METABOLITE ppm
BLOOD
UTERUS
BLOOD
UTERUS
AUTOPSY
HYSTERERECTOMY

Fig.31 Scatter diagram showing correlation between total DDT concentrations in blood and uterine tissue of women undergoing autopsy.

$r = 0.200$

$n = 25$

NS

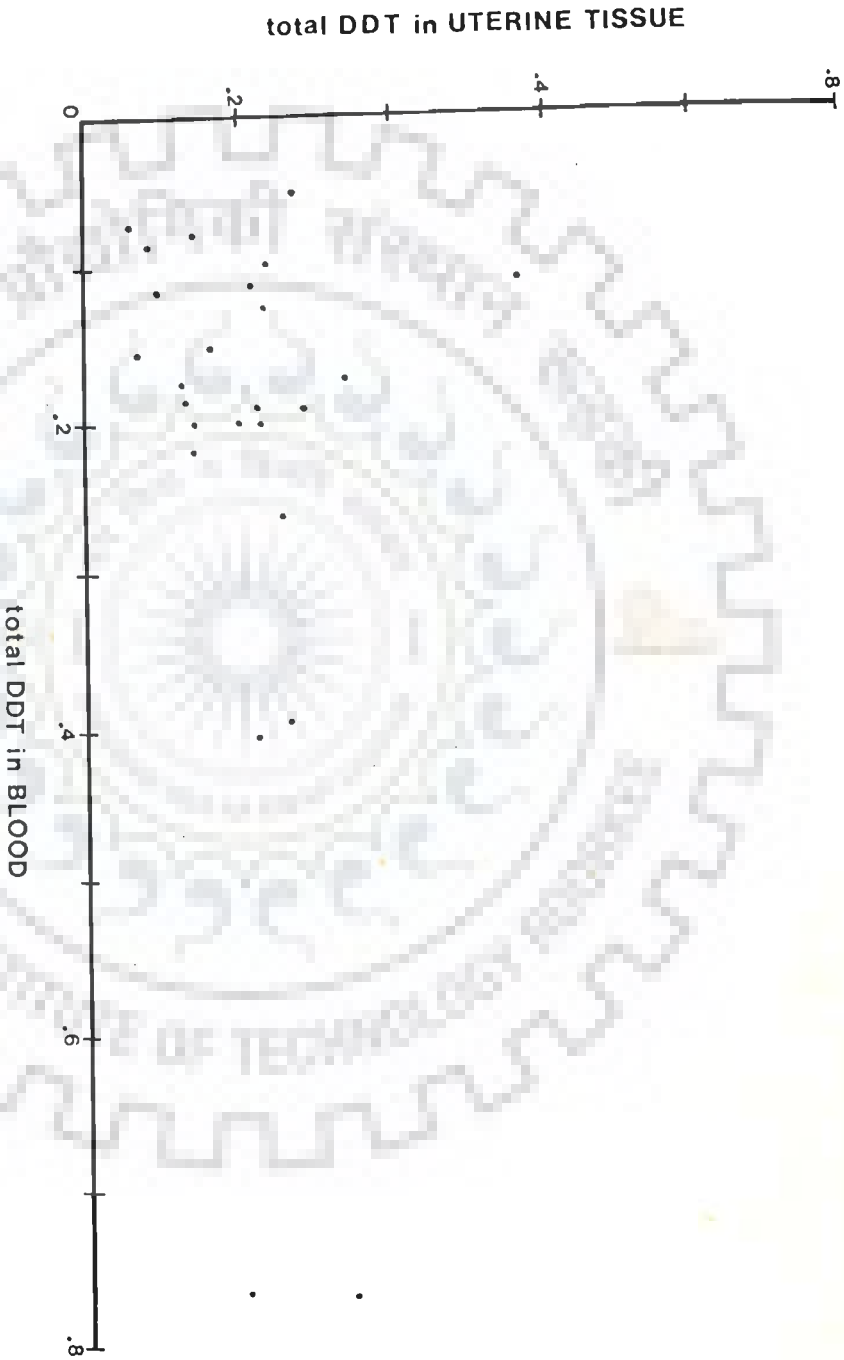

total DDT in UTERINE TISSUE
0
.2
.4
.8
total DDT in BLOOD
.2
.4
.6
.8

Fig.32 Scatter diagram showing correlation between total DDT concentrations in blood and uterine tissue of women undergoing hysterectomy.

$r = 0.783$

$n = 25$

$p < 0.001$

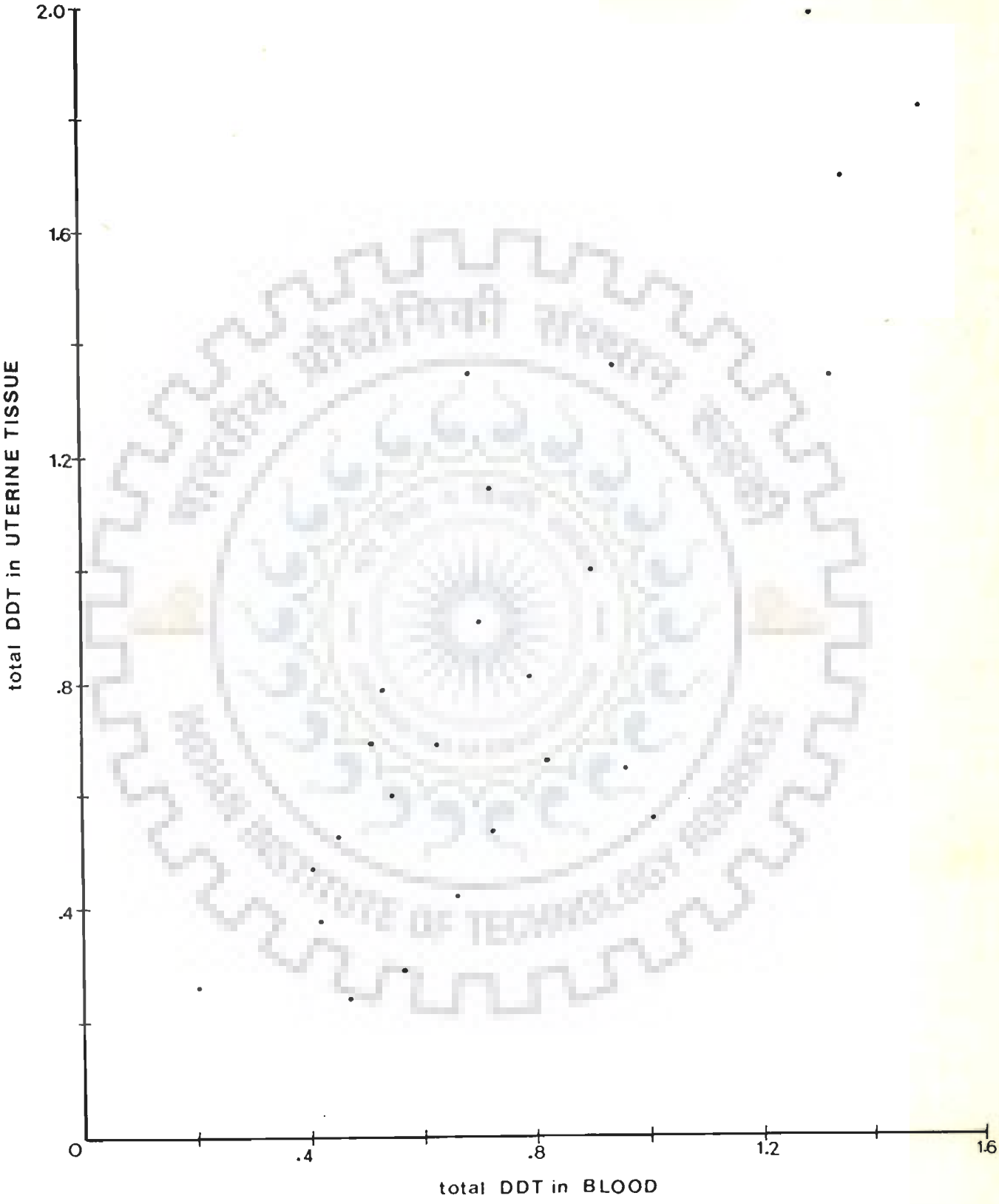

2.0
1.6
1.2
.8
.4
0
.4
.8
1.2
1.6
total DDT in UTERINE TISSUE
total DDT in BLOOD

TABLE-28: AGE WISE DISTRIBUTION OF DDT AND ITS METABOLITES IN BLOOD AND UTERINE TISSUE OF WOMEN UNDERGOING AUTOPSY (ppm).

Compound	Range of concentrations and means ± S.D.			
	Age groups			
	Blood		Uterus	
	10-25 yrs N = 17	26-40 yrs N = 8	10-25 yrs N = 17	26-40 yrs N = 8
p,p'-DDE	0.016 - 0.091 0.046 ± 0.019 (17)	0.012 - 0.251 0.050 ± 0.081 (8)	0.005 - 0.084 0.023 ± 0.017 (17)	0.008 - 0.052 0.028 ± 0.013 (8)
o,p'-DDT	o.015 - 0.586 0.080 ± 0.124 (17)	0.012 - 0.225 0.053 ± 0.070 (8)	0.008 - 0.083 0.024 ± 0.018 (17)	0.009 - 0.047 0.032 ± 0.014 (8)
p,p'-DDD	0.000 - 0.004 0.0007± 0.001 (8)	0.000 - 0.002 0.001 ± 0.0006 (7)	0.000 - 0.003 0.0003± 0.0007 (8)	0.000 - 0.006 0.001 ± 0.002 (6)
p,p'-DDT	0.021 - 0.206 0.098 ± 0.047 (17)	0.023 - 0.291 0.100 ± 0.081 (8)	0.011 - 0.115 0.047 ± 0.025 (17)	0.029 - 0.088 0.054 ± 0.021 (8)
Total DDT	0.073 - 0.767 0.225^{a}± 0.167 (17)	0.052 - 0.769 0.204^{b}± 0.232 (8)	0.030 - 0.282 0.096^{c}± 0.059 (17)	0.046 - 0.172 0.115^{d}± 0.046 (8)
	t=0.23 NS		t=0.88 NS	

N = Number of samples.
Figures in parentheses indicate the number of positive samples.
a, c (t = 3.00; p < 0.01)
b, d (t = 1.06 Statistically not significant)

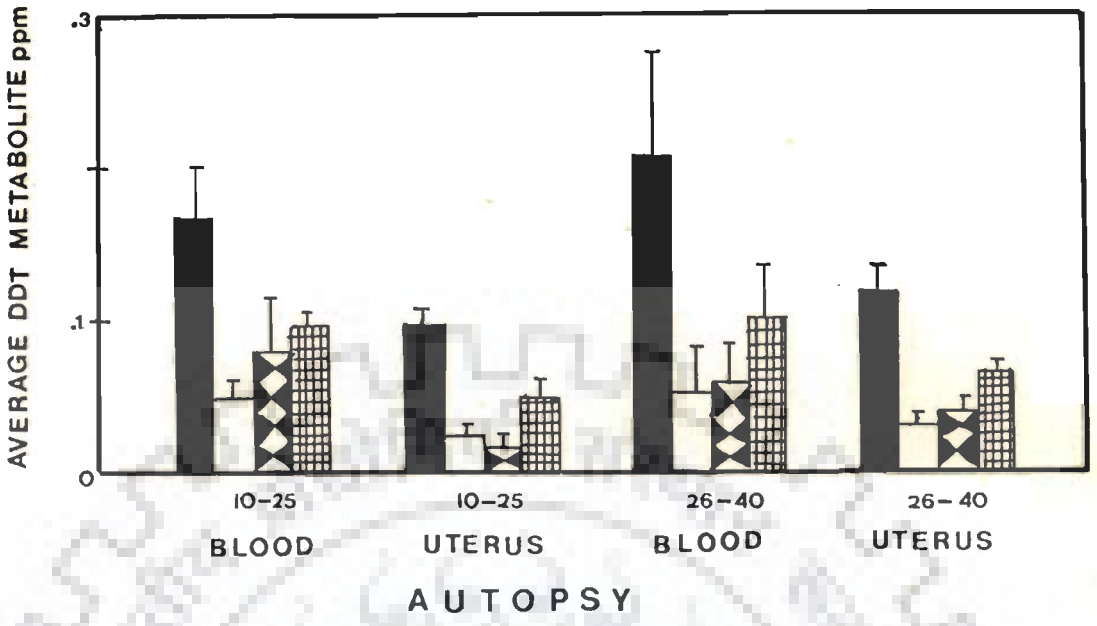
TABLE-29: AGE WISE DISTRIBUTION OF DDT AND ITS METABOLITES IN BLOOD AND UTERINE TISSUE OF WOMEN UNDERGOING HYSTERECTOMY (ppm).

Compound	Range of concentrations and means ± S.D.			
	Blood		Uterus	
	Age groups			
	25-40 yrs N = 10	41-60 yrs N = 15	25-40 yrs N = 10	41-60 yrs N = 15
p,p'-DDE	0.063 - 0.226 0.167 ± 0.053 (10)	0.096 - 0.339 0.208 ± 0.073 (15)	0.086 - 0.302 0.173 ± 0.071 (10)	0.061 - 0.339 0.217 ± 0.081 (15)
o,p'-DDT	0.058 - 0.874 0.294 ± 0.257 (10)	0.007 - 0.865 0.240 ± 0.262 (15)	0.032 - 1.174 0.246 ± 0.333 (10)	0.032 - 0.951 0.336 ± 0.319 (15)
p,p'-DDD	0.000 - 0.042 0.010 ± 0.012 (7)	0.000 - 0.020 0.004 ± 0.006 (7)	0.000 - 0.055 0.010 ± 0.017 (5)	0.000 - 0.022 0.007 ± 0.008 (8)
p,p'-DDT	0.044 - 0.357 0.194 ± 0.099 (10)	0.076 - 0.669 0.374 ± 0.156 (15)	0.117 - 0.510 0.243 ± 0.134 (10)	0.140 - 0.625 0.398 ± 0.152 (15)
Total DDT	0.200 - 1.130 0.666[a] ± 0.290 (10)	0.408 - 1.493 0.827[b] ± 0.340 (15)	0.260 - 1.982 0.673[c] ± 0.499 (10)	0.246 - 1.816 0.959[d] ± 0.468 (15)
	t=1.27 NS		t=1.44 NS	

N = Number of samples.
Figures in parentheses indicate the number of positive samples.
a,c and b,d = Statistically not significant.

Fig.33a Bar diagram showing age wise distribution of DDT residues in blood and uterine tissues of women undergoing autopsy.

Fig.33b Bar diagram showing age wise distribution of DDT residues in blood and uterine tissue of women undergoing hysterectomy.


AVERAGE DDT METABOLITE ppm
.3
.1
0
10–25
BLOOD
10–25
UTERUS
26–40
BLOOD
26–40
UTERUS
AUTOPSY

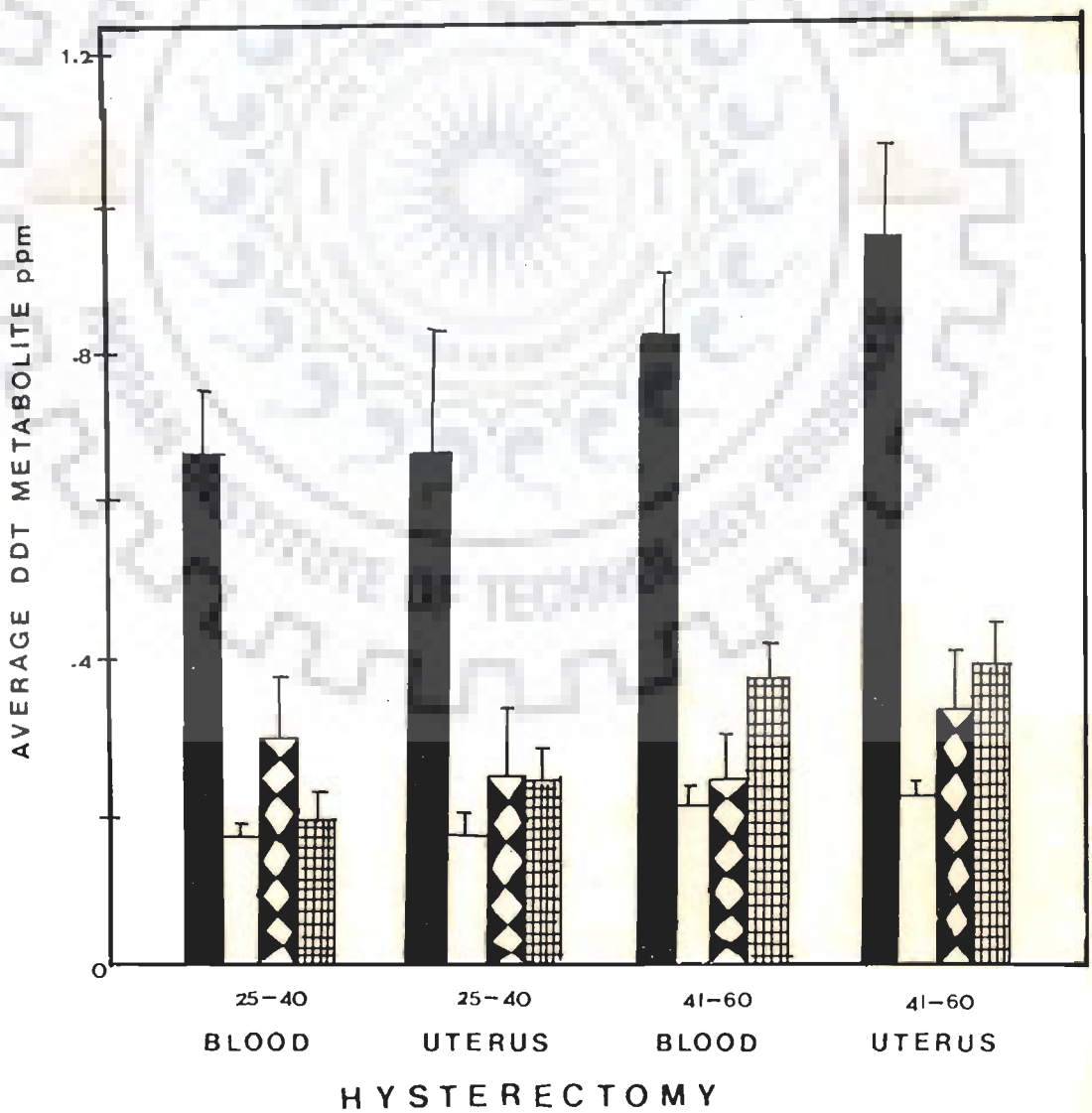

AVERAGE DDT METABOLITE ppm
1.2
.8
.4
0
25–40
BLOOD
25–40
UTERUS
41–60
BLOOD
41–60
UTERUS
HYSTERECTOMY

Fig.34 Scatter diagram showing correlation between total DDT concentrations in blood and age in women undergoing autopsy.

r = 0.164
n = 25
NS

Fig.35 Scatter diagram showing correlation between total DDT concentrations in uterine tissue and age in women undergoing autopsy.

r = 0.195
n = 25
NS

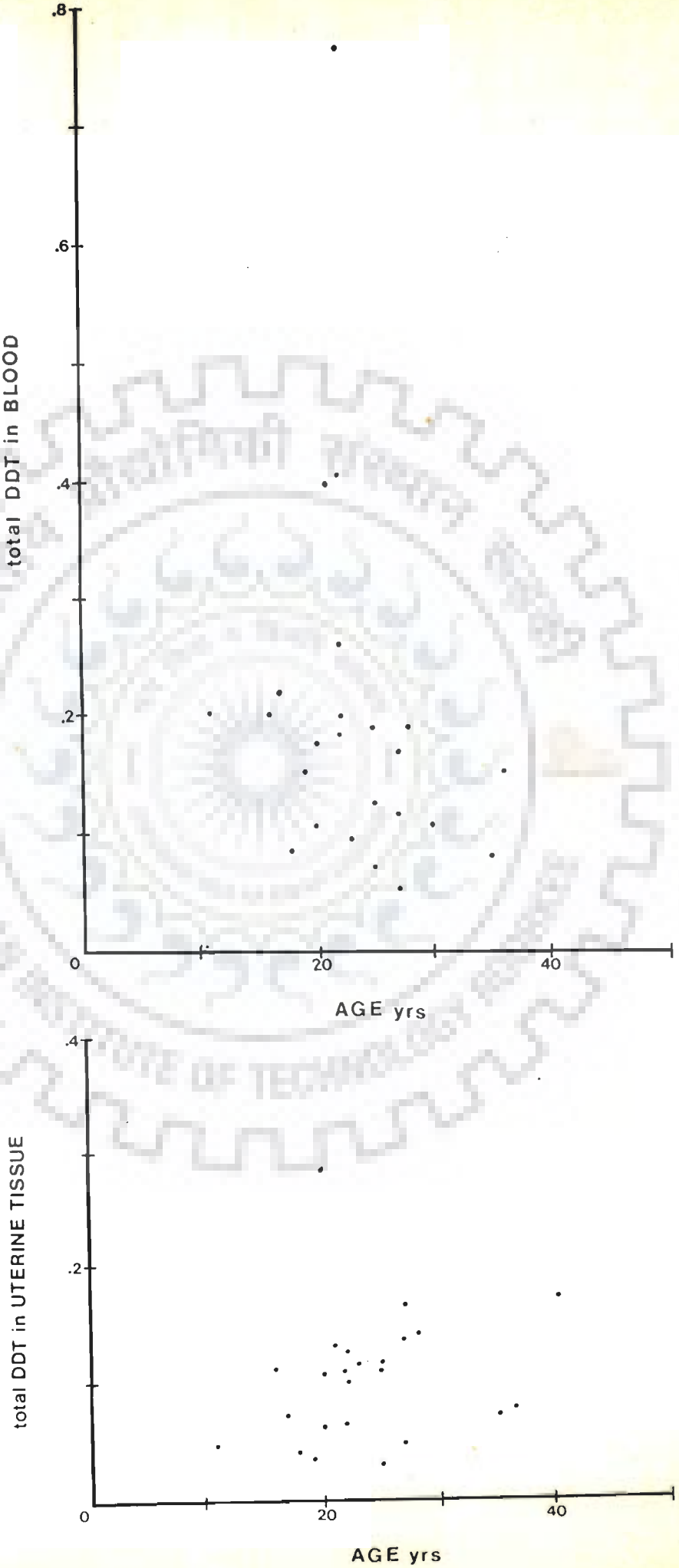

.8
.6
.4
.2
0
20
40
total DDT in BLOOD
AGE yrs
.4
.2
0
20
40
total DDT in UTERINE TISSUE
AGE yrs

Fig.36 Scatter diagram showing correlation between total DDT concentrations in blood and age in women undergoing hysterectomy.

r = 0.300

n = 25

NS

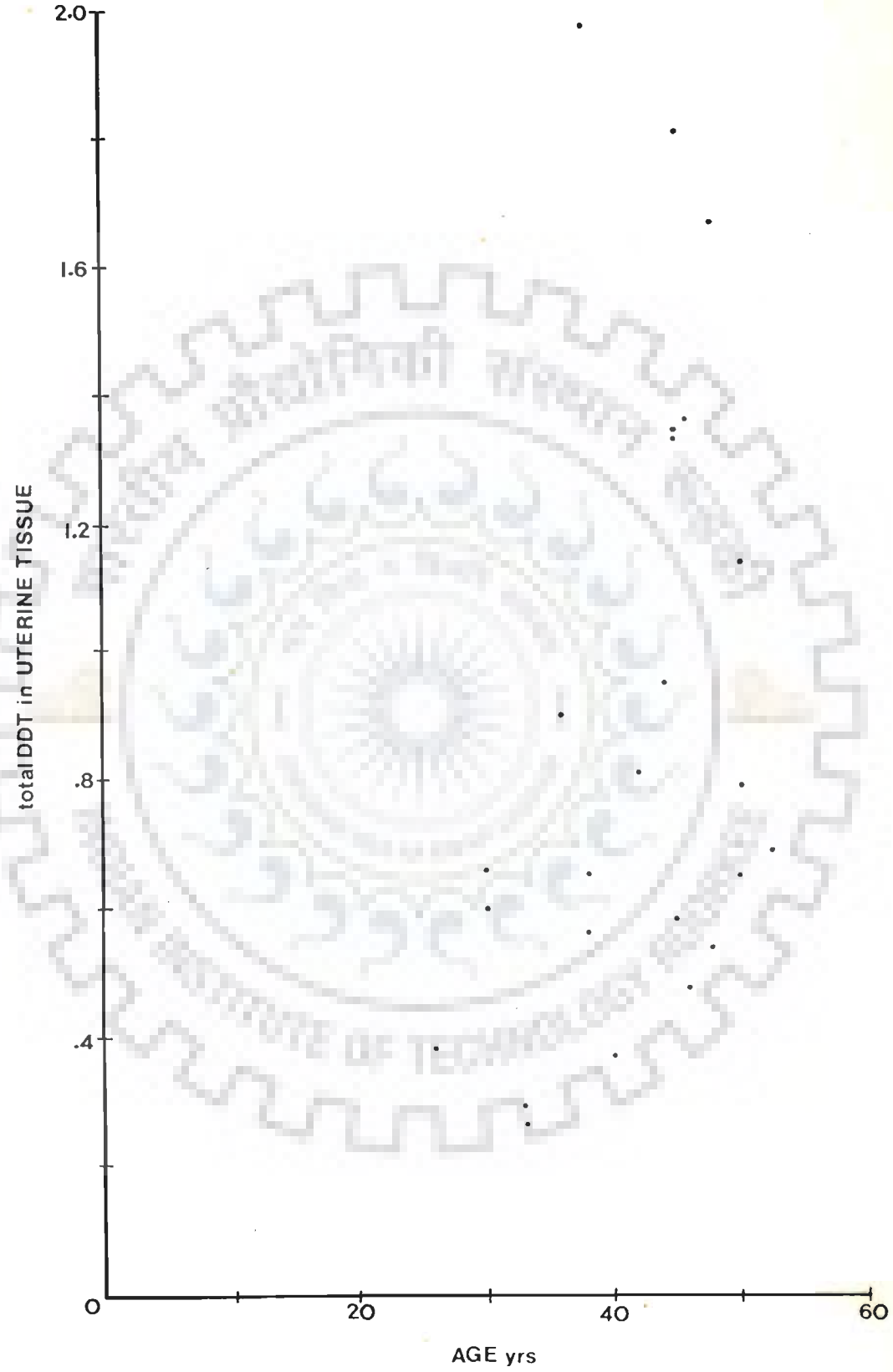
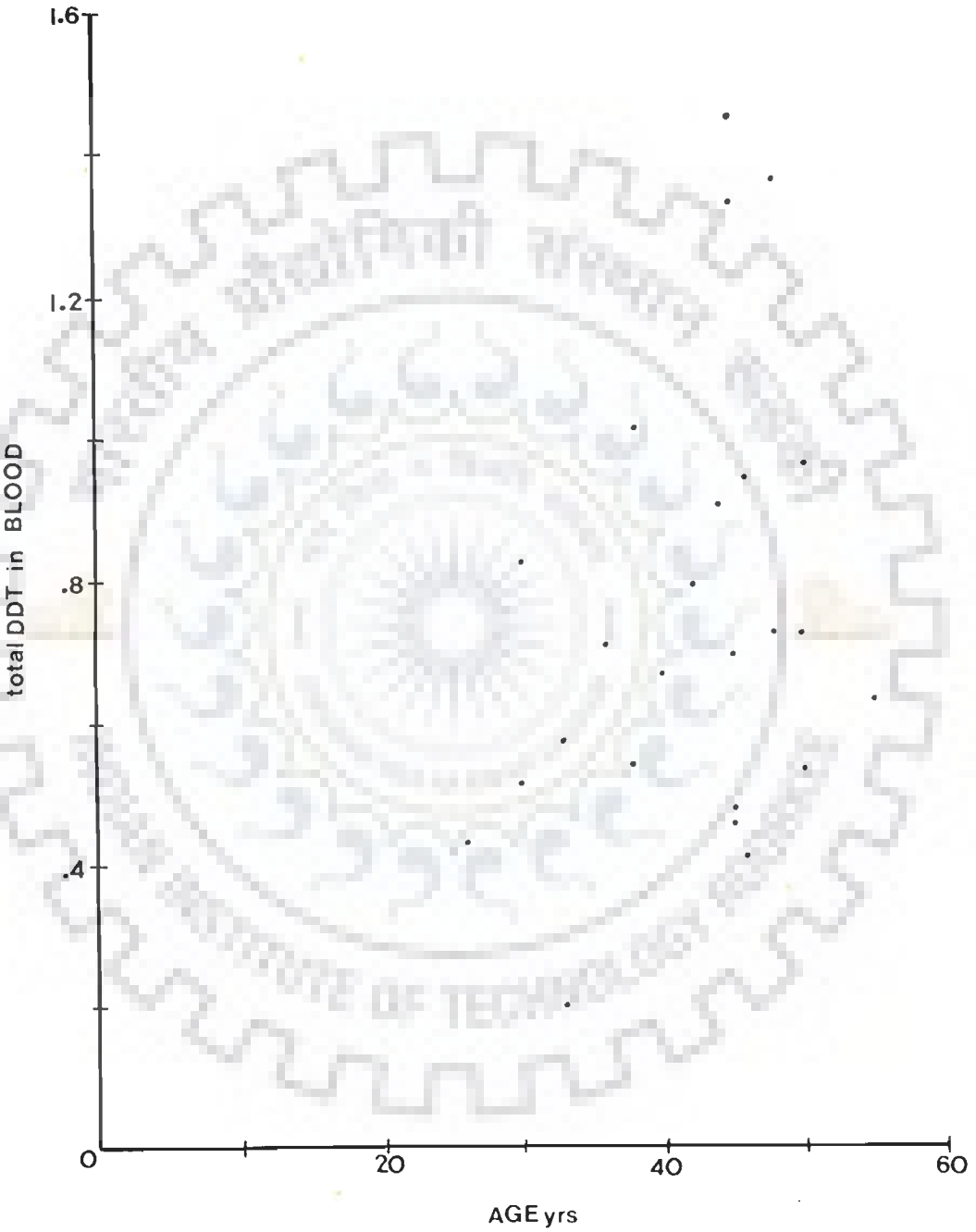

2.0
1.6
1.2
.8
.4
0
20
40
60
totalDDT in UTERINE TISSUE
AGE yrs

Fig.37 Scatter diagram showing correlation between total DDT concentrations in uterine tissue and age in women undergoing hysterectomy.

r = 0.273

n = 25

NS


1.6
1.2
.8
.4
0
20
40
60
total DDT in BLOOD
AGE yrs

B. Clinical data

The adverse health effects of DDT in occupationally unexposed population, pregnant women and women undergoing hysterectomy were studied. No consistent patterns of abnormality were found either in medical histories or physical examination, nor were any clinical symptoms found to be associated with DDT concentrations in the blood.

4.5 *Clinical findings among occupationally unexposed population*

Results in Table 30 summarize the clinical findings among occupationally unexposed population of Delhi area.

Symptoms

Respiratory:

The respiratory symptoms were: cough in 2 (6.4 %) males and 7 (36.8 %) females; wheezing in 1 (3.2 %) male and 1 (5.2 %) female; and eye and nose irritation in 1 (5.2 %) female.

Dermatologic:

Few dermatologic findings were reported in occupationally unexposed population. Skin rash in 2 (6.4 %) males and 7 (36.8 %) females; acne in 8 (25.8 %) males and 12 (63.1 %) females.

Gastrointestinal:

In occupationally unexposed population, appetite loss was reported in 2 (5 %) males and nausea in 2 (10.5 %) females. The vomiting sensation was reported in 1 (5.2 %) female.

Neurologic:

The neurologic symptoms were: headache in 2 (6.4 %) males and 5 (26.3 %) females, and sleeplessness in 1 (3.2 %) male and

TABLE-30: CLINICAL SYMPTOMS AMONG OCCUPATIONALLY UNEXPOSED POPULATION OF DELHI.

Symptoms	Occupationally unexposed population			
	Male (31)		Female(19)	
	No.	%	No.	%
Respiratory				
Cough	2	6.4	7	36.8
Wheezing	1	3.2	1	5.2
Tightness in chest	-	-	-	-
Eye and nose irritation	-	-	1	5.2
Dermatological				
Rash	2	6.4	7	36.8
Acne	8	25.8	12	63.1
Burning sensation	-	-	-	-
Darkening of skin	-	-	-	-
Thickening of skin	-	-	-	-
Discolouration of finger nails	-	-	-	-
Gastrointestinal				
Weight loss	-	-	-	-
Appetite loss	2	5.0	-	-
Nausea	-	-	2	10.5
Vomiting	-	-	1	5.2
Abdominal pain	-	-	-	-
Constipation	-	-	-	-
Diarrhea	-	-	-	-
Neurological				
Headache	2	6.4	5	26.3
Blurred vision	-	-	-	-
Dizziness	-	-	-	-
Depression	-	-	-	-
Tiredness	-	-	-	-
Perceptional changes	-	-	-	-
Nervousness	-	-	-	-

Cont'd..

Continued..

Sleeplessness	1	3.2	4	21.0
Muscle weakness	-	-	-	-
Difficulty in walking	-	-	-	-
Parasthesia	-	-	-	-
Loss of balance	-	-	-	-
Musculo-skeletal				
Muscle pain	-	-	1	5.2
Joint pain	2	6.4	3	15.7
Cardiovascular				
High blood pressure	-	-	-	-
Low blood pressure	-	-	-	-
Pain in chest	1	3.2	-	-
Palpitation of heart	-	-	-	-

Figures in parentheses indicate the number of subjects.

4 (21 %) females.

Musculo-skeletal:

In occupationally unexposed population, muscle pain was reported in 1 (5.2 %) female and joint pain in 2 (6.4 %) males and 3 (15.7 %) females.

Cardiovascular:

No cardiovascular symptoms were reported in occupationally unexposed population except pain in chest in one male subject.

4.6 *Clinical findings among pregnant women*

The clinical findings among women with full term pregnancy, premature labour and toxemia of pregnancy are summarized in Table 31.

Symptoms

Respiratory:

The main respiratory complaints were: cough in 6 (12 %) women with full term pregnancy, 1 (10 %) woman with premature labour, and 10 (25 %) women with toxemia of pregnancy; wheezing in 1 (10 %) woman with premature labour and 1(2.5 %) woman with toxemia of pregnancy. No other respiratory symptoms were reported in pregnant women.

Dermatologic:

The pregnant women reported few dermatologic symptoms. Skin rash was reported in 4 (8 %) women with full term pregnancy, 2 (20 %) women with premature labour and 3 (7.5 %) women with toxemia of pregnancy. Acne was reported in 10 (20 %) women with full term pregnancy, 2 (20 %) women with premature labour, and 7 (17.5 %) women with toxemia of pregnancy.

TABLE-31: CLINICAL SYMPTOMS AMONG PREGNANT WOMEN

Symptoms	Type of pregnancy					
	Full term(50)		Premature(10)		Toxemia(40)	
	No.	%	No.	%	No.	%
Respiratory						
Cough	6	12.0	1	10.0	10	25.0
Wheezing	2	4.0	-	-	4	10.0
Tightness in chest	-	-	1	10.0	1	2.5
Dermatological						
Rash	4	8.0	2	20.0	3	7.5
Acne	10	20.0	2	20.0	7	17.5
Burning sensation	-	-	-	-	-	-
Darkening of skin	-	-	-	-	-	-
Thickening of skin	-	-	-	-	-	-
Discolouration of finger nails	-	-	-	-	-	-
Gastrointestinal						
Weight loss	1	2.0	2	20.0	7	17.5
Appetite loss	4	8.0	1	10.0	4	10.0
Nausea	2	4.0	1	10.0	5	12.5
Vomiting	1	2.0	1	10.0	3	7.5
Abdominal pain	-	-	-	-	-	-

Cont'd..

Continued..

Constipation	-	-	-	-	-	-
Diarrhea	-	-	-	-	-	-
Neurological						
Headache	4	8.0	2	20.0	10	25.0
Blurred vision	-	-	-	-	-	-
Dizziness	-	-	-	-	-	-
Depression	1	2.0	-	-	4	10.0
Tiredness	2	4.0	5	50.0	7	17.5
Perceptional changes	-	-	-	-	-	-
Nervousness	-	-	-	-	1	2.5
Sleeplessness	-	-	-	-	-	-
Sleepiness	-	-	-	-	-	-
Muscle weakness	-	-	-	-	-	-
Difficulty in walking	-	-	1	10.0	-	-
Parasthesia	-	-	-	-	-	-
Loss of balance	-	-	-	-	-	-
Musculo-skeletal						
Muscle pain	-	-	1	10.0	-	-
Joint pain	2	4.0	4	40.0	7	17.5
Cardiovascular						
High blood pressure	2	4.0	3	30.0	40	100.0
Low blood pressure	7	14.0	-	-	-	-
Pain in chest	-	-	-	-	-	-
Palpitation of heart	-	-	-	-	-	-

Figures in parentheses indicate the number of subjects.

Gastrointestinal:

Few gastrointestinal symptoms were reported in pregnant women. The main symptoms were: weight loss in 1 (2.%) woman with full term pregnancy, 2 (20 %) women with premature labour and 7 (17.5 %) women with toxemia of pregnancy; appetite loss in 4 (8 %) women with full term pregnancy, 1 (10 %) woman with premature labour and 4 (10 %) women with toxemia of pregnancy; nausea in 2 (4 %) women with full term pregnancy, 1 (10 %) woman with premature labour and 5(12.5 %) women with toxemia of pregnancy; vomiting in 1 (2 %) woman with full term pregnancy, 1 (10 %) woman with premature labour and 3 (7.5 %) women with toxemia of pregnancy. Abdominal pain, constipation and diarrhoea were not reported in pregnant women.

Neurologic:

The neurologic symptoms reported in pregnant women were: headache in 4 (8 %) women with full term pregnancy, 2 (20 %) women with premature labour, and 10 (25 %) women with toxemia of pregnancy; depression in 1 (2 %) woman with full term pregnancy and 4 (10 %) women with toxemia of pregnancy; tiredness in 2 (4 %) women with full term pregnancy, 5 (50 %) women with premature labour, and 7 (17.5 %) women with toxemia of pregnancy. Difficulty in walking was reported in one woman with premature labour.

Musculo-skeletal:

Muscle pain was reported in only 1 (10 %) woman with premature labour. Joint pain was reported in 2 (4 %) women with full term pregnancy, 4 (40 %) women with premature labour and 7 (17.5 %) women with toxemia of pregnancy.

Cardiovascular:

Few cardiovascular symptoms were reported in pregnant women. High blood pressure was reported in 2 (4 %) women with full term pregnancy, 3 (30 %) women with premature labour,and 40 (100 %) women with toxemia of pregnancy. Low blood pressure was reported in 7 (14 %) women with full term pregnancy.

4.7 *Clinical findings among women who underwent hysterectomy*

A summary of clinical findings in women undergoing hysterectomy is presented in Table 32.

Symptoms

Respiratory:

The main respiratory symptoms reported in women who underwent hysterectomy were: cough in 3 (12 %), wheezing in 1 (4 %) and eye and nose irritation in 1 (4 %).

Dermatologic:

Dermatologic symptoms were: skin rash in 2 (8 %) and acne in 5 (20 %).

Gastrointestinal:

The gastrointestinal symptoms reported in women who underwent hysterectomy were: weight loss in 5 (20 %), loss of appetite in 1 (4 %), nausea in 3 (12 %), vomiting in 1 (4 %), and abdominal pain in 2 (8 %).

Neurologic:

The main neurologic symptoms were: headache in 3 (12 %), tiredness in 2 (8 %) and muscle weakness in 1 (4 %).

TABLE-32: CLINICAL SYMPTOMS AMONG 25 WOMEN WHO UNDERWENT HYSTERECTOMY.

Symptoms	Number	Percent
Respiratory		
Cough	3	12.0
Wheezing	1	4.0
Tightness in chest	-	-
Dermatological		
Rash	2	8.0
Acne	5	20.0
Burning sensation	-	-
Darkening of skin	-	-
Thickening of skin	-	-
Discolouration of finger nails	-	-
Gastrointestinal		
Weight loss	5	20.0
Appetite loss	5	20.0
Nausea	3	12.0
Vomiting	1	4.0
Abdominal pain	2	8.0
Constipation	-	-
Diarrhea	-	-
Neurological		
Headache	3	12.0
Blurred vision	-	-

Cont'd..

Continued..

Dizziness	-	-
Depression	2	8.0
Tiredness	-	-
Perceptional changes	-	-
Nervousness	-	-
Sleeplessness	-	-
Sleepiness	-	-
Muscle weakness	1	4.0
Difficulty in walking	-	-
Parasthesia	-	-
Loss of balance	-	-
Musculo-skeletal		
Muscle pain	1	4.0
Joint pain	2	8.0
Cardiovascular		
High blood pressure	3	12.0
Low blood pressure	1	4.0
Pain in chest	-	-
Palpitation of heart	-	-

Musculo-skeletal:

One (4 %) woman complained of muscle pain and 2 (8 %) women reported joint pain.

Cardiovascular:

High blood pressure was reported in 3 (12 %) women and low blood pressure in only 1 (4 %) woman.

C. Laboratory data

4.8 *Blood tests*

The pertinent blood test findings are summarized in Table 33-37. The results indicate that there is a paucity of abnormal results in the biochemical studies. In all the subjects no correlation was observed between DDT concentrations in the blood and altered biochemical findings.

No altered biochemical finding was reported in the occupationally unexposed population except low blood glucose values in 2 (4 %) and high cholesterol levels in 3 (6 %) subjects (Table 33).

In pregnant women with full term pregnancy, no altered biochemical finding was reported except low blood glucose values in 3 (6 %) subjects (Table 34). In women with premature labour (Table 35), the most frequently altered laboratory test results were albumin in 5 (50 %), total proteins in 4 (40 %), glucose in 3 (30 %), and urea in 2 (20 %). These biochemical parameters also altered in women with toxemia of pregnancy (Table 36).

Table 37 shows that few biochemical parameters altered in women undergoing hysterectomy. These include: albumin in 4 (16 %), cholesterol in 3 (12 %), and total proteins in 1 (4 %) subject.

TABLE-33: BIOCHEMICAL FINDINGS IN 50 SUBJECTS FROM OCCUPATIONALLY UNEXPOSED POPULATION OF DELHI.

Biochemical parameters	Normal		Low		High	
	No.	%	No.	%	No.	%
Glucose	48	96.0	2	4.0	-	-
Urea	50	100.0	-	-	-	-
Total Proteins	50	100.0	-	-	-	-
Albumin	50	100.0	-	-	-	-
Cholesterol	47	94.0	-	-	3	6.0
Total bilirubin	50	100.0	-	-	-	-
Creatinine	50	100.0	-	-	-	-
Alkaline Phosphatase	50	100.0	-	-	-	-
SGOT	50	100.0	-	-	-	-
SGPT	50	100.0	-	-	-	-

TABLE-34: BIOCHEMICAL FINDINGS IN 50 WOMEN WITH FULL TERM PREGNANCY.

Biochemical parameters	Normal		Low		High	
	No.	%	No.	%	No.	%
Glucose	47	94.0	3	6.0	-	-
Urea	50	100.0	-	-	-	-
Total proteins	50	100.0	-	-	-	-
Albumin	50	100.0	-	-	-	-
Cholesterol	50	100.0	-	-	-	-
Total bilirubin	50	100.0	-	-	-	-
Creatinine	50	100.0	-	-	-	-
Alkaline Phosphatase	50	100.0	-	-	-	-
SGOT	50	100.0	-	-	-	-
SGPT	50	100.0	-	-	-	-

TABLE-35: BIOCHEMICAL FINDINGS IN 10 WOMEN WITH PREMATURE LABOUR

Biochemical parameters	Normal		Low		High	
	No.	%	No.	%	No.	%
Glucose	7	70.0	-	-	3	30.0
Urea	8	80.0	-	-	2	20.0
Total proteins	6	60.0	-	-	4	40.0
Albumin	5	50.0	-	-	5	50.0
Cholesterol	10	100.0	-	-	-	-
Total bilirubin	10	100.0	-	-	-	-
Creatinine	10	100.0	-	-	-	-
Alkaline Phosphatase	10	100.0	-	-	-	-
SGOT	10	100.0	-	-	-	-
SGPT	10	100.0	-	-	-	-

TABLE-36: BIOCHEMICAL FINDINGS IN 40 CASES OF TOXEMIA OF PREGNANCY.

Biochemical parameters	Normal		Low		High	
	No.	%	No.	%	No.	%
Glucose	32	80.0	-	-	8	20.0
Urea	32	80.0	-	-	8	20.0
Total proteins	22	55.0	-	-	18	45.0
Albumin	16	40.0	-	-	24	60.0
Cholesterol	40	100.0	-	-	-	-
Total bilirubin	40	100.0	-	-	-	-
Creatinine	40	100.0	-	-	-	-
Alkaline Phosphatase	40	100.0	-	-	-	-
SGOT	40	100.0	-	-	-	-
SGPT	40	100.0	-	-	-	-

4.9 *Hematology:*

Table 38-40 show the hemoglobin levels of subjects investigated in this study. No significant abnormalities were reported in Erythrocyte Sedimentation Rate (ESR) and hemoglobin levels.

In occupationally unexposed population, hemoglobin level 9-10 gm/100 ml was observed in 6 (19.4 %) male and 14 (73.7 %) female subjects; 10-12 gm/100 ml was observed in 15 (48.4 %) male and 4 (21.1 %) female subjects; >12 gm/100 ml was observed in 10 (32.2 %) male and 1 (5.2 %) female subject (Table 38).

In pregnant women with full term pregnancy (Table 39), hemoglobin level 9-10 gm/100 ml was observed in 32 (64 %); 10-12 gm/100 ml in 16 (32 %), and >12 gm/100 ml in 2 (4 %). In women with premature labour, hemoglobin level 9-10 gm/100ml was observed in 7 (70 %); 10-12 gm/100 ml in 2 (20 %), and >12 gm/100 ml in 1 (10 %). In women with toxemia of pregnancy, hemoglobin level 9-10 gm/100 ml was observed in 29 (72.5 %), and 10-12 gm/100 ml in 11 (27.5 %).

In women who underwent hysterectomy (Table 40), hemoglobin level 9-10 gm/100 ml was observed in 2 (8 %); 10-12 gm/100 ml in 16 (64 %), and >12 gm/100 ml in 7 (28 %).

4.10 *Urine tests*

No altered urine test results were observed in the subjects investigated in this study.

TABLE-37: BIOCHEMICAL FINDINGS IN 25 WOMEN WHO UNDERWENT HYSTERECTOMY.

Biochemical parameters	Normal		Low		High	
	No.	%	No.	%	No.	%
Glucose	24	96.0	1	4.0	-	-
Urea	25	100.00	-	-	-	-
Total proteins	24	96.0	-	-	1	4.0
Albumin	21	84.0	-	-	4	16.0
Cholesterol	22	88.0	-	-	3	12.0
Total bilirubin	25	100.0	-	-	-	-
Creatinine	25	100.00	-	-	-	-
Alkaline Phosphatase	25	100.0	-	-	-	-
SGOT	25	100.0	-	-	-	-
SGPT	25	100.0	-	-	-	-

TABLE-38: HEMOGLOBIN LEVELS OF OCCUPATIONALLY UNEXPOSED POPULATION OF DELHI.

Hemoglobin g/100 ml	Occupationally unexposed population			
	Male (31)		Female(19)	
	No.	%	No.	%
9-10	6	19.4	14	73.7
10-12	15	48.4	4	21.1
>12	10	32.2	1	5.2

Figures in parentheses indicate the number of subjects.

TABLE-39: HEMOGLOBIN LEVELS OF WOMEN WHO UNDERWENT FULL TERM PREGNANCY AND IN CASES OF PREMATURE LABOUR AND TOXEMIA OF PREGNANCY.

Hemoglobin g/100 ml	Full term (50)		Premature (10)		Toxemia (40)	
	No.	%	No.	%	No.	%
9 - 10	32	64.0	7	70.0	29	72.5
10 - 12	16	32.0	2	20.0	11	27.5
> 12	2	4.0	1	10.0	-	-

Figures in parentheses indicate the number of subjects.

TABLE-40: HEMOGLOBIN LEVELS OF 25 WOMEN WHO UNDERWENT HYSTERECTOMY.

Hemoglobin g/100 ml	Number	Percent
9 - 10	2	8.0
10 - 12	16	64.0
> 12	7	28.0

DISCUSSION

DDT residues

5.1 Soil

5.2 Blood of occupationally unexposed population

5.3 Pregnant and lactating women

5.4 Blood and uterine tissue

Health effects

DISCUSSION

The accumulation of organochlorine pesticides in the environment and tissues of human beings and other life forms is an established fact. The residue levels of these insecticides, especially DDT, have evoked interest of scientists all over the world in regard to their long term toxicity. The problem is of even greater concern in India where DDT is still being used extensively in agriculture and malaria control programmes. Moreover, a DDT manufacturing unit is also located in the heart of the city and contributing to environmental pollution load. The present study was conducted in areas surrounding DDT manufacturing unit in Delhi to assess the extent and magnitude of environmental contamination and to explore the health hazards posed by DDT residues in occupationally unexposed population of Delhi.

The analysis of DDT residues in environmental samples and human tissues was done on a gas liquid chromatograph equipped with an electron capture detector after proper extraction and clean up procedures as described in EPA manual (277). Four metabolites of DDT viz. p,p'-DDE, o,p'-DDT, p,p'-DDD and p,p'-DDT were confirmed by methods described in Chapter 3. The routine analysis of DDT residues was done on column containing 1.5 % OV_{17} and 1.95 % QF_1 on Gas Chrom Q 100-120 mesh.

DDT residues

5.1 *Soil*

The soil is an environmental reservoir for the residues of pesticides from where they move into the atmosphere, water and living organisms (91). It is evident from the present data that soils of Delhi area contain high levels of DDT

residues; total DDT residues ranged from 0.498 to 7.270 ppm with a mean value of 1.670 ppm (Table 8a). DDT residues in soil were also reported in soil surveys conducted in U.S.A. (46, 48, 49), Canada (39, 187), Bulgaria (169) and India (272, 307). Tripathi (272) reported DDT in 120 of 138 samples analysed from Tarai area in Uttar Pradesh, India. In the present study highest concentration of DDT, 7.270 ppm, was found at Durga Nagar, where a DDT factory is located. Other areas contained moderate to high levels of DDT in soils depending on the distance from the factory (Table 7). The East Zone sites which were far away from the DDT factory and also included agricultural land contained minimum, 1.227 ppm, total DDT residues as compared to other 4 zones of Delhi (Table9). In West Zone where DDT factory is located, soils contained maximum, 2.346 ppm, total DDT residues. Similar trend was observed in a survey conducted by Yadav et al (307) in Delhi area. Total DDT as high as 29.45 ppm in agricultural soil (48) and 388.16 ppm in urban soils (46) has been reported in the United States. Lang et al (171) found a maximum of 13.93 ppm total DDT from a survey of six U.S. Air Force Bases. The occurrence of DDT residues in Delhi soils might be predominantly attributed to volatilization and subsequent dispersal of DDT in the vicinity of the factory; DDT has been shown to volatilize into the atmosphere (263, 284), from where it ultimately reaches the surface soils. In addition to the dispersal from the DDT factory, large scale use of DDT in the control of malaria might have resulted in widespread contamination of soils in Delhi area. DDT residues in soil are highly stable and persists for a long time (87, 193, 285). It was estimated that more than 50 % of applied DDT would

remain in soils for at least 15 years (55). The persistence of DDT residues in soil also contribute to accumulation of high concentrations of these residues.

In the present study, DDT in soil was comprised mainly of p,p'-DDT and its metabolites p,p'-DDE and p,p'-DDD. In addition to p,p'-DDT and its metabolites o,p'-DDT was also detected. These are commonly reported DDT component in soil (39, 187, 266). Carey et al (47, 48) reported o,p'-DDE and o,p'-DDD residues in addition to these commonly occurring metabolites. DDT in soil undergoes transformation in the presence of various physical, chemical and biological factors and degrade to DDE, the terminal residue of DDT (177). DDD is also formed in the soil mainly as a result of microbial degradation (274, 249).

In the present study, soil samples had higher concentration of p,p'-DDT as compared to its main metabolite p,p'-DDE; p,p'-DDT was more than p,p'-DDE in 45 out of 50 soil samples analysed (Table 7). The concentration of p,p'-DDD was less than p,p'-DDE. A similar trend of less DDD as compared to DDE have been reported (99). Carey et al (48) detected p,p'-DDE and p,p'-DDT in most of the soil samples and traces of p,p'-DDD and o,p'-DDT in cropland soil samples. In the present study, o,p'-DDT was detected in 45 out of 50 samples in low quantities. However, unlike in the soil surveys in United States (49) o,p'-DDE could not be detected in any soil samples in Delhi area. It is clear from the data of Carey et al (49) that they found o,p'-DDE only in 0.7 % soil samples. The high concentration of p,p'-DDT in Delhi area may be contributed to continuous addition of p,p'-DDT residues in the soil from the DDT factory and malaria control programmes.

5.2 *Blood of occupationally unexposed population of Delhi*

DDT is a bioaccumulative poison which is only slowly metabolized thus, it is possible that the continued general exposure to DDT may result in biologically significant accumulation of DDT residues in the general population. The human pesticide residue is a biological index of pesticide exposure. Residues of organochlorine pesticides have been detected in both human fat and blood from different parts of the world (295). By comparison with levels found in other parts of the world, very high levels of DDT and its metabolites have been reported in the body fat of people of India (66, 231). Since there is a definite relationship between the amount of DDT and its metabolites in blood and those present in human depot fat, blood can be used for assessing the total body burden of DDT in various populations (38).

In the present study, the whole blood samples of occupationally unexposed population from areas surrounding DDT factory in Delhi contained DDT and its metabolites; total DDT ranged from 0.053 to 0.663 ppm with a mean value of 0.301 ppm (Table 11) In a similar survey conducted in Delhi (7), 174 out of 182 blood samples were reported to contain DDT and its metabolites and total DDT ranged from 0.166 to 0.683 mg/litre. A higher concentration of DDT in the blood indicate a higher total body burden of DDT in the population (38). The present data agree closely with those reported by Ramachandran et al(231) and Dale et al (66) who found about 10 times more DDT in the body fat of people in India than in that of people from other countries showing that data from blood sample give an indication of the total DDT content of the body.

The main DDT metabolites detected in the present study were p,p'-DDE, p,p'-DDT and o,p'-DDT. Traces of p,p'-DDD were also reported in few blood samples. These metabolites were also detected in surveys conducted elsewhere (295). In an earlier study conducted in Delhi (7) blood samples were reported to contain p,p'-DDE, p,p'-DDD, p,p'-DDT and o,p'-DDT. In contrast to these findings (22), p,p'-DDE and p,p'-DDT were reported in 59 blood samples collected in U.S.A. while no o,p'-DDT and p,p'-DDD were detected in these samples. The predominant metabolite of DDT was DDE and was generally present in larger quantities than DDT (Table 10) indicating that DDE is stored in the human body in preference to DDT or its other metabolites. A similar relationship is found in human adipose tissue and in many animals (295).

The mean total DDT value in male (0.344 ppm) was higher than that of female (0.229 ppm; $p<0.05$). In other surveys also males were reported to contain higher DDT residues than females (7, 109). This sexual difference in residue levels may be due to excretion of these chemicals in mother's milk and during menses in females. Moreover, females have more fat than males.

In the present study, dietetic habits were shown to effect the accumulation of DDT residues in the blood. The mean total DDT in non-vegetarian (0.345 ppm) was higher than that of vegetarians (0.221 ppm; $p<0.05$). This difference may be due to high DDT values in meat products.(145) which are common in non-vegetarian meals.

The blood DDT concentrations in the present study were not related to the age of the subject (Table 12). In contrast

to this finding, Kreiss et al (167) reported that total DDT residues increased with the age of the donor.

The presence of high levels of DDT and its metabolites in the blood of residents from areas surrounding the DDT factory in Delhi may be due to the ingestion of DDT through food contaminated as a result of general environmental contamination; food is responsible for more than 80 % intake of these pesticides by general population (166). The direct exposure to contaminated air, water and dust may also contribute to body burden. In the present study, it was observed that the blood of residents of West Zone, where the DDT factory is situated, contained higher DDT levels as compared to residents from other zones of Delhi (Table 14a). Kreiss et al (167) reported that the geometric mean level of total DDT in 499 persons living downstream from a DDT manufacturing plant was several times the national geometric mean level.

5.3 *Pregnant and lactating women*

A number of organochlorine pesticides used in different national programmes are accessible to the human organism. Thus, the organochlorine pesticides circulating in the blood of human get an opportunity to reach the growing fetus via placenta and to neonates via milk. In the present study, a detailed and systematic study of the transfer of DDT residues from mother to the fetus was investigated in 50 pregnant women of different age and dietetic habits. The residues of DDT and its metabolites detected in the circulating maternal blood, umbilical cord blood and placental tissue of women who underwent full term pregnancy represents the total body burden of mothers and reflect the transfer of DDT and its metabolites

to growing fetus via placenta (Table 15). The study has also revealed that maternal blood DDT levels were significantly higher than the levels in umbilical cord blood at delivery (Table 17a), and the trend was:

Maternal blood > Placental tissue > Umbilical cord blood

These findings were in agreement with several authors (151, 222, 223, 232, 244, 245, 246, 250). The low levels of DDT and its metabolites in the umbilical cord blood may be due to lower serum lipid concentrations; serum lipids in cord blood at delivery was about one third than that of maternal blood at delivery (186).

The mother's age and dietetic habits were not shown to influence the accumulation of DDT in circulating blood and its subsequent transfer to fetus via umbilical cord blood (Table-20-23). In contrast to these findings, age and dietetic habits were shown to influence the accumulation of DDT in pregnant women and its subsequent transfer to fetus (245).

It is evident from the data on women with full term pregnancy that DDT and its metabolites are transported from the mother to fetus crossing the placental barrier. The placental transfer of DDT was also reported in earlier studies (223, 244, 245, 246). The prenatal growth is more or less directly related to the transport of various compounds across the placental barrier. Evidence has been brought forth that the uptake of amino acids is retarded by the presence of metabolic poisons (140). Pesticides like DDT may presumably be one of them, so there is every reason to believe that their presence in the maternal blood or fetal blood may hamper the transfer of amino acids - the building-block units of proteins. Since

all the enzymes are proteins and are required to direct metabolic pathways, logic can dictate to presume an unfortunate situation leading to congenital abnormalities and other deformities in prenates and neonates.

The excretion of DDT in milk represent an efficient means for their removal from the female, but these compounds are also transferred with milk to neonates. In the present study on lactating Indian mothers, it was found that milk contained considerably higher levels of total DDT as compared to maternal blood. The results of this study clearly indicate that the DDT levels present in the milk of Indian women were far greater than the values reported in surveys elsewhere (295). In Indian lactating mothers, the mean total DDT in milk (2.030 ppm) was about 10 times higher than that reported in the maternal blood (0.253 ppm). A positive correlation was observed between the concentrations of total DDT in milk and maternal blood ($r = 0.469$; $p<0.05$; Fig.27). Similar correlation was also observed in a study conducted on women living in Lucknow, India (251).

On the basis of total DDT levels in milk, daily intake of total DDT by the Indian child weighing 5 kg and consuming 1 kg of milk a day was calculated to be 0.406 mg/kg body weight/day. A daily intake of about 108.628 ppm of total DDT by the neonates was reported in Lucknow, India (251). The calculated daily intake in the present study was far greater than 0.01 mg/kg/day, the maximum safe intake of DDT, recommended by WHO(13).

The neonatal health hazards are evident from such a perplexing situation due to pesticide pollution in view of their role in carcinogenesis, mutagenesis and teratogenesis (13, 95).

It would also be significant to note that DDT represses the formation of antibodies, an act of immunosuppressor (228). The consequences are obvious, the infants would be more vulnerable to infections.

Keeping in view the aforesaid facts and the report revealing the skin disorders and deaths occurred in children of nursing mothers who had eaten hexachlorobenzene-treated seeds in Turkey in 1956 (201), it may be inferred that higher the pesticide pollution of food and environment, the greater would be the body burden of pesticides in the breast feeding mothers, and consequently the risk for health hazards to prenates and neonates would be greater.

The placental tissue of pregnant women also contained high total DDT concentrations (Table 17a). The presence of DDT residues in placental tissue of pregnant women was also reported in other studies (223, 244, 245, 246). The presence of DDT and its metabolites in placental tissue of pregnant women is of great significance in the light of existing knowledge on placental lesions following ingestion of DDT (170 mg/kg body weight/day) in rats on days 9 and 10 of the pregnancy (81). Extensive hemmorhages, necrosis, absence of glycogen and RNA were observed in the placenta. Placental lesions may interfere with the normal activity of this organ and may upset hormonal synthesis, nutrient supply and/or other processes indispensible for the maintenance of pregnancy homeostasis.

Organochlorine pesticides have been shown to have various effects on reproductive system. In the present study, high levels of DDT and its metabolites were detected in the maternal blood, umbilical cord blood and placental tissue of women

undergoing premature labour and in cases of toxemia of pregnancy as compared to those with full term pregnancy (Table 19a). The trend was:

Premature > Toxemia > Full term

The samples of this study were collected from women in child bearing age group. No effect of age and dietetic habits on accumulation of DDT residues in pregnant women was observed (Table 20-23). This fact shows that the higher DDT levels in women with premature labour and toxemia of pregnancy were not related to age and dietetic habit of the subjects.

Many authors have reported an association between high plasma levels of DDT and toxemia of pregnancy (290), missed abortions (24, 243, 246), premature labour (225, 243, 246, 291) and recent and missed abortions (25). The mere association between the presence of a relatively high amount of xenobiotic and a pathological condition is not enough to support a cause-effect relationship between this xenobiotic and the pathological finding. The identification of a xenobiotic does not exclude the presence of other agent(s) with a role in the etiopathology of the pathological condition. Female California sea lions who had premature pups had not only higher organochlorine compounds but also higher concentrations of several metals in comparison with females that delivered mature pups (203).

Premature labour is defined as the delivery of a fetus of more than 500 gm and less than 2500 gm after the 20th and before 37th week of gestation. The occurrence of premature labour was attributed to a variety of anomalies related to uterus, placenta or fetus, to diseases of mother, and about two-third of cases of unknown circumstances (291). The role of

xenobiotics and among them organochlorine pesticides, like DDT, in the occurrence of premature labour was suggested by the higher premature labour rate in humans exposed to these xenobiotics (291, 243, 246). In heavy smokers and narcotic users, premature labour is one of the complications of pregnancy. Also, DDT residues were higher in California sea lions which gave birth prematurely than in those with full term pups (77). Eight out of 14 rabbits which received p,p-DDT, 50 mg/kg body weight, on days 7, 8, and 9 of pregnancy delivered prematurely (129).

The mechanism of action of these organochlorine pesticides in possible initiation of premature labour is still unknown. DDT may disturb the hormonal balance of pregnancy and perhaps so precipitate labour. A high level of progesterone and relatively low levels of estrogens have been reported during pregnancy. The peripheral levels of estradiol 17-β, probably the estrogen most relevant to parturition in terms of biological activity, rises rapidly in the last month of the pregnancy suggesting a facilitatory role of estrogen in labour. Transient contractility or reduction of oxytocic threshold of the uterus may be the function of estrogen. Estrogens are also concerned with the synthesis of contractile proteins, actin and myosin, deposition of glycogen and the enzymes concerned with deriving chemical energy from glucose.

o,p'-DDT was shown to be the most effective estrogenic compounds among organochlorine compounds in general, and among DDT analogs in particular. The estrogenic activity of o,p'-DDT approaches 10^{-4} of that of estradiol (52). However, DDT with estrogenic activity are anti-estrogenic, since in

competing with estrogen for binding sites, they actually reduce the estrogenic effects in target organs. Other mechanism of reduction of estrogenic activity by DDT may be the decrease of estrogen synthesis in estrogen-secreting organs and/or the increased degradation of estrogen in the liver; DDT have been shown to stimulate the activity of enzymes responsible for metabolizing steroids such as estradiol and andosterone (202).

Malnutrition is also one of the factors for premature labour but in the present study only healthy patients with normal hemoglobin levels were included. Thus, the probability that nutritional status of women played a role in premature labour in this study is very low.

The presence of prostaglandins in the decidua was reported to be closely associated with the initiation of labour, and they are known to be released at the time of labour (275). Higher levels of DDT and DDE reported in the present study may cause the lysis of the lysosomal membrane, thereby releasing the phospholipase A2, a rate limiting factor in the biosynthesis of prostaglandins and thus may help affect the onset of labour (246).

The etiology of toxemia of pregnancy, which occur generally in primiparae, is not yet clearly understood. The clinical features of hypertension and edema, serious complications like disseminated intravascular coagulation and seizures and laboratory tests showing renal lesions (proteinuria and morphological changes), placental lesions, the immunological response, the hormonal and enzymatic activity etc., may represent a chain reaction to a unique etiological factor or to several factors

entering in the maternal organism from the placental blood.

It is believed that normally such factors foreign to the maternal organism are inactivated by the appearance of an adequate amount of antibodies. In organisms which are not capable of a normal immunological defence response during the materno-fetal interaction, the excess of fetal antigen may initiate the physiological phenomena of toxemia of pregnancy. Some women who developed symptoms of toxemia of pregnancy proved to be immunologically hyporesponsive to phytohemagglutinine, to their husbands' leukocytes and cord leukocytes, and had a lower tendency to form HL-A antibodies (196). The fact that immunological reactions are usually weak after a first stimulation and are stronger after a second one, was related to the occurrence of toxemia especially in primiparae (i.e. a weak response to a first exposure to fetoplacental antigen in immunologically hyporesponsive women).

Non immunological factors such as malnutrition (vitamin deficiency, starvation) may adversely affect the immunologic reactivity (130).

DDT appears to have a depressant effect on the immune system. Rats and rabbits receiving DDT in aqueous suspension at a concentration of 200 mg/litre showed a depression in antibody formation and decrease in at least one globulin fraction of the blood (288, 289). In the guinea pig dosage of 1-20 mg/kg did not have any effect on antitoxin production but produces a reduction in tissue histamine level (100).

It may be concluded that relatively higher levels of DDT and its metabolites in pregnant women of present study may be a direct or an indirect cause of premature labour and toxemia of pregnancy. The DDT residues might have led to the retention

of another toxic agent, the direct cause of the biological effects observed.

5.4 *Blood and uterine tissue*

The storage of DDT residues in relation to disease was investigated in the present study. High levels of DDT and its metabolites were reported in the blood and uterine tissue of women who underwent hysterectomy because of some pathological condition of uterus. The uterus collected during autopsy was considered as normal. The mean total DDT in blood and uterine tissue of autopsy cases was found to be 0.219 ± 0.186 and 0.103 ± 0.055 ppm, respectively.(Table 27). The mean total DDT levels in blood and uterine tissue of women undergoing hysterectomy was found to be 0.76 ± 0.325 and 0.845 ± 0.402 ppm,respectively (Table 27). The age of the subjects had no effect on the residue levels of DDT in blood and uterine tissue (Table 28,29). Polishuk et al (223) reported that maternal blood and uterine tissue of pregnant women who underwent cesarian section for obstetric indication contained mean total DDT level of 4.577 ± 4.033 and 11.782 ± 3.05 ppm, respectively. The authors also suggested the role of uterus in protection against environmental hazards during pregnancy. Some investigators (75, 229) have reported that DDT storage was 1.7 - 7.6 times greater in persons dying of cirrhosis, atherosclerosis, hypertension, idiopathic amyloidosis, and certain forms of cancer. Recently (240) p,p'-DDT, p,p'-DDD and p,p'-DDE in the lungs of patients dying from neoplasm were reported to be 3 times as high as in the lungs of patients dying from other causes. Unger and Olsen (276) also reported higher concentrations of DDE in extracted lipids of adipose tissue samples from terminal

Cancer patients (malignant lymphoma, retroperitoneal carcinoma, adenocarcinoma, cancer of the breast, mesothelioma, carcinoma of the cervix, pulmonary carcinoma, cancer of the rectum, cancer of the colon, lymphosarcoma etc.) than in adipose tissue of patients who died of other diseases. This difference was statistically significant.

In the present study the role of high levels of DDT residues in blood and uterine tissue of women undergoing hysterectomy still remains unknown.

Health effects

The concern with potential human health effects is augmented by the fact that human exposure to DDT seems to be widespread as a result of environmental contamination. The effects on human health of prolonged exposure to DDT are not well delineated. This gap is particularly evident for chronic low dose effects such as those occurring in environmental exposures. In the present study adverse health effects of DDT were evaluated in occupationally unexposed population, pregnant women and in women who underwent hysterectomy. The results indicate that in occupationally unexposed population, pregnant women and women undergoing hysterectomy showed no consistent pattern of abnormalities either in medical histories or physical examination, nor were any clinical symptoms found to be associated with DDT levels in the blood (Table 30, 31, 32).

In the present study, commonly reported respiratory symptoms were: cough, wheezing, eye and nose irritation. Minor irritation of the skin and eyes were reported (206) in workers occupationally exposed to DDT. Recently increased risk of lung cancer was reported in DDT exposed male agricultural workers

(23). In workers, it was reported that much of the inhaled dust has deposited in the upper respiratory tract and swallowed (94). In general population, DDT residues entering through respiration may cause respiratory symptoms as evident from the present study.

The dermatological symptoms reported in the present study were: skin rash and acne. In a study on occupationally exposed workers (122), skin rashes were very frequently observed. The acne reported in the present study is the most characteristic dermatological response resulting from exposure to halogenated aromatic hydrocarbons. The mechanism by which chloroaryl hydrocarbons induce acne is not known: it was suggested that the sebaceous gland damage might be due to the local contact of these substances. Recently, it has been reported that skin lesions' DDT is transported by the blood to the site of lesion after both gastrointestinal and cutaneous absorption.

The main neurological symptoms reported were: nausea, vomiting, headache, dizziness, depression, tiredness and sleeplessness. In workers exposed to DDT, parasthesia of the extremities, headache and dizziness were reported (11). In another study (103), irritability and fatigue was reported in workers exposed to DDT. The volunteers taking dosage as high as 285 mg/kg of DDT reported vomiting, headache and nausea (101).

Few cardiovascular symptoms were reported in the present study. These include: change in the blood pressure and palpitation of heart. The effect of DDT on human heart such as palpitations, tachycardia and irregular heart actions had been noticed in some but not all cases of acute poisoning (176, 131, 193).

There was also a paucity of abnormal results in the

biochemical studies and altered biochemical findings, if any, were not correlated with blood DDT concentrations (Table 33-37). The commonly altered biochemical test results were: glucose, total proteins, albumin, and cholesterol. Laws et al (173) reported that total protein and albumin were within the normal limits in occupationally exposed workers but elevated alkaline phosphatase and SGPT levels were observed in workers showing involvement of liver. Morgan and Lin (190) reported increase in the levels of SGOT, SGPT and LDH in occupationally exposed workers. In the present study no effect on serum levels of SGOT, SGPT and alkaline phosphatase was observed indicating no effect of DDT residues on liver.

It is evident from the studies conducted in the Delhi area where a DDT manufacturing factory is located, that high levels of DDT and its metabolites are present in the environment and human tissues. It is therefore important to monitor the amount of DDT in human tissues from time to time to discover any change in environmental pollution load. Findings of DDT residues in environment and human tissues will provide a major element in DDT regulatory decisions.

SUMMARY

DDT levels

Residues in soil

Residues in blood of general population

Residues in pregnant and lactating women

Residues in blood and uterine tissue

Health effects

SUMMARY

1. Organochlorine compounds are globally distributed, persistent environmental contaminants, and pollutants of human tissues and milk. The extent and seriousness of the potential hazards due to these chemicals still remain to be fully defined. The population of Delhi is exposed to high levels of DDT due to the presence of a DDT manufacturing factory and extensive use of DDT in malarial eradication programme. The present study was aimed to assess the extent of environmental contamination and explore the possible health hazards posed by DDT in general population of Delhi area.

2. The DDT and its metabolites in environment and human tissues were analyzed on a gas liquid chromatograph equipped with an electron capture detector. Confirmation was done by using different GLC columns, thin layer chromatography, TLC-GLC pairing and chemical methods.

DDT LEVELS

Residues in soil

3. The soil samples collected from 50 sites, near and away from the DDT factory were analyzed for DDT and its metabolites. DDT was detected in all the soil samples. The total DDT ranged from 0.498 to 7.270 ppm with a mean value of 1.670 ± 1.163 ppm. The median DDT level was calculated to be 1.223 ppm.

4. The highest DDT concentration, 7.270 ppm, was detected in the soil from Durga Nagar in the vicinity of the DDT factory. The lowest DDT concentration, 0.498 ppm, was found in the soil from IARI campus.

5. The most common DDT metabolites in soil were: p,p'-DDT,p,p'-

DDE and p,p'-DDD. o,p'-DDT was found in 45 out of 50 samples. The proportion of p,p'-DDT to p,p'-DDE was 2:1.

6. Zonal distribution of DDT residues in soil was studied. The mean total DDT values of five zones of Delhi were compared using one way analysis of variance. No statistically significant difference in DDT levels was observed in soil samples of the five zones of Delhi. The West Zone of Delhi where DDT factory is located contained higher DDT concentrations in soil as compared to other zones.

7. It is apparent from the data that areas close to the DDT factory contained higher DDT levels in soil as compared to areas away from the factory.

Residues in blood of general population

8. Blood samples of 50 volunteers from occupationally unexposed population of Delhi were examined for DDT residues. All samples contained DDT and its metabolites. The total DDT concentration ranged from 0.112 to 0.663 ppm in males and 0.053 to 0.560 ppm in females.

9. The DDT metabolites detected in blood were: p,p'-DDE, p,p'-DDD, o,p'-DDT and p,p'-DDT. p,p'-DDE and p,p'-DDT accounted for most of the total DDT.

10. The average total DDT concentration in males (0.344 ppm) was significantly higher than that of females (0.229 ppm;$p<0.05$).

11. The mean total DDT values of different age groups were compared using one way analysis of variance. No statistically significant difference was observed in blood DDT levels of 4 age groups. The total DDT concentrations in blood showed no correlation with the age.

12. The dietetic habits were shown to effect accumulation of DDT in the blood. The average total DDT concentration in non-vegetarians (0.345 ppm) was significantly higher than that of vegetarians (0.221 ppm; $p<0.05$).

13. Zonal distribution of DDT residues in the blood of general population of Delhi was studied. The mean blood total DDT values of 5 zones of Delhi were compared using one way analysis of variance on log transformed values followed by multiple range test. The results indicate that residents of West and South zones contained higher concentrations of DDT in the blood as compared to residents from other zones.

Residues in pregnant and lactating women

14. The transfer of DDT to prenates via placenta and to neonates via breast milk was investigated. DDT levels were detected in maternal blood, umbilical cord blood, milk, and placental tissue of 50 women with full term pregnancy.

15. The DDT and its metabolites were detected in all the samples analyzed indicating their transfer through placenta.

16. The mean total DDT levels in maternal blood, umbilical cord blood and placental tissue of women with full term pregnancy were compared using one way analysis of variance followed by multiple range test. The results indicate that concentration of DDT was higher in maternal blood (0.280 ppm) as compared to umbilical cord blood (0.133 ppm) and placental tissue (0.268 ppm). The trend was:

Maternal blood >Placental tissue >Umbilical cord blood

17. No correlation was found to exist between the total DDT

concentrations and age and dietetic habits of women with full term pregnancy.

18. The study has also revealed that human placenta stores considerably high amounts of DDT and its metabolites as evident from high mean total DDT concentration, i.e. 0.268 ± 0.194 ppm.

19. The excretion of DDT and its metabolites in milk was evident from high levels of DDT in milk of lactating Indian mothers. The average total DDT concentration in milk (2.030 ppm) was significantly higher than that of maternal blood (0.253 ppm; $p < 0.001$).

20. A correlation was found to exist between DDT concentrations in milk and maternal blood ($r = 0.469$; $p < 0.05$).

21. The daily intake of DDT by an Indian infant weighing 5 kg and consuming 1 kg of milk a day, was calculated to be 0.406 mg/kg body weight/day.

22. The DDT residues were compared in women with full term pregnancy and women with premature labour and toxemia of pregnancy.

23. Considerably higher levels ($p < 0.001$) of DDT and its metabolites were present in the maternal blood, umbilical cord blood and placental tissue of women with premature labour and toxemia of pregnancy as compared to women with full term pregnancy. The trend was:

Premature > Toxemia > Full term pregnancy

24. No correlation was found to exist between total DDT concentrations and age and dietetic habits of women with premature labour and toxemia of pregnancy.

Residues in blood and uterine tissue

25. The DDT and its metabolites were investigated in relation to disease. DDT residues were estimated in blood and uterine tissue collected from women who underwent autopsy and hysterectomy.

26. The DDT levels in blood and uterine tissue of women who underwent autopsy averaged 0.219 ± 0.189 and 0.103 ± 0.055 ppm, respectively.

27. The DDT levels in blood and uterine tissue of women who underwent hysterectomy averaged 0.763 ± 0.325 and 0.845 ± 0.402 ppm, respectively.

28. The results indicate that considerably higher levels of DDT were present in the blood and uterine tissue of women who underwent hysterectomy as compared to women who underwent autopsy ($p < 0.001$).

29. The total DDT concentrations in blood and uterine tissue of women who underwent autopsy and hysterectomy showed no correlation with the age.

HEALTH EFFECTS

30. The effects on human health of prolonged exposure to DDT are yet not well delineated. In the present study, occupationally unexposed population, pregnant women and women who underwent hysterectomy were investigated to explore the adverse health effects, if any, of high DDT concentrations in the body.

31. The clinical data obtained from occupationally unexposed population, pregnant women and women who underwent hysterectomy indicated that no consistent pattern of abnormalities was present either in medical histories or physical examination,

nor were any clinical symptoms found to be associated with concentrations of DDT in the blood.

32. The laboratory data obtained from occupationally unexposed population, pregnant women and women who underwent hysterectomy was indicative of paucity of abnormal results in the biochemical studies. No correlation was observed between DDT concentrations in the blood and altered biochemical findings, if any.

33. The Erythrocyte Sedimentation Rate (ESR) and hemoglobin levels were within the normal limits.

34. The urine test results showed no abnormal findings.

It is evident from the studies conducted in Delhi area, where a DDT factory is located, that high levels of DDT and its metabolites are present in the environment and human tissues. It is therefore important to monitor the amount of DDT in human tissues from time to time to discover any change in environmental pollution load. Findings of DDT residues in environment and human tissues will provide a major element in DDT regulatory decisions.

REFERENCES

1. Abbott, D.C.; Harrison, R.B.; Tatton, J.O.G., and Thompson,J. Organochlorine pesticides in the atmosphere. Nature 211: 259 (1966).

2. Abou-Donia, M.B. and Menzel, D.B. The metabolism in vivo of 1,1,1-trichloro-2,2-bis(p-chlorophenyl) ethylene (DDE) in the chick by embryonic injection and dietary ingestion. Biochem. Pharmacol. 17: 2143(1968).

*3. Adamovic, V.M. and Sokic, B. Lower level phenomenon of DDT cumulation in female abdominal fatty tissue. Arch. Hig. Rada Toksikol. 24: 303(1973).

4. Adamovic, V.M.; Sokic, B., and Jovanovic-Smilganski, M. Some observations concerning the ratio of the intake of organochlorine insecticides through food and amounts excreted in the milk of breast feeding mothers. Bull. Environ. Contam. Toxicol. 20: 280(1978).

5. Addison, R.F. and Zinck, M.E. Rate of conversion of ^{14}C-p,p'-DDT to p,p'-DDE by brook trout (Salvelinus fontanalis): Absence of effect of pretreatment of fish with compounds related to p,p'-DDT. J. Fish Res. Board Can. 34: 119(1977).

6. Agarwal, H.C. and Gupta, B.R. Distribution and metabolism of DDT in the cat fish (Heteropneustes fossilis) in relation to the signs of poisoning. Toxicol.Appl. Pharmacol. 29: 204(1974).

7. Agarwal, H.C.; Pillai, M.K.K.; Yadav, D.V.; Menon, K.B., and Gupta, R.K. Residues of DDT and its metabolites in human blood samples in Delhi, India. Bull.W.H.O. 54: 349(1976).

8. Agarwal, H.C.; Yadav, D.V., and Pillai, M.K.K. Metabolism of ^{14}C-DDT in Pheretima posthuma and effect of pre-treatment with DDT, lindane and dieldrin. Bull. Environ. Contam. Toxicol. 19: 295(1978).

9. Agnihotri, N.P.; Pandey, S.Y.; Jain, H.K., and Srivastava,K.P. Persistence of aldrin, dieldrin, lindane, heptachlor and p,p'-DDT in soil. J. Entomol. Res. (New Delhi). 1: 89(1977).

10. Albright, R.N.; Johnson, N.; Sanderson, T.W.; Farbs, R.M.; Melton, R.; Fisher, L.; Wells, G.R.; Parsons,W.R.; Scott, V.C.; Speake, J.L.; Stallworth, J.R.;Moore, B.G., and Hayer, A.W. Pesticide residues in the top soil of five West Alabama Counties. Bull. Environ. Contam. Toxicol. 12: 378(1974).

*11. Aleksieva, T.; Vasilev, G., and Spasovoki, M. Study of the toxic effects of DDT. Hyg. Epidemiol. Microbiol.6:8(1959).

12. Alexander, M. Microbial degradation of pesticides. In: Environmental Toxicology of pesticides. F. Matsumura, G.M. Boush and T. Misato (Eds.) Academic Press, New York and London pp.365(1972).

13. Anonymous. Comments on alleged carcinogenicity of DDT as documented by EPA, review prepared for House Committee on Appropriation, July, 1975. Clin. Toxicol. 9: 827(1976).

14. Apple, G.; Morgan, D.P., and Roan, C.C. Determinants of serum DDT and DDE concentrations. Bull. Environ.Contam. Toxicol. 5: 16(1970).

15. Arthur, R.D.; Cain, J.D., and Barrentine, B.F. DDT residues in air in the Mississippi Delta. Pestic. Monit. J. 10: 168(1977).

*16. Atchison, G.J. and Johnson, H.E. The degradation of DDT in brook trout eggs and fry. Tr. Am. Fish Soc. 104: 782(1975).

17. Bailey, S.; Bunyan, P.J.; Rennison, B.D., and Taylor,A. The metabolism of DDT, DDD, DDE and DDMU in the pigeon. Toxicol. Appl. Pharmacol. 14: 13(1969).

*18. Bakseyev, N.S. Prophylaxis of pathology of the fetus and newborn child (experience of the Ukrainian SSR). Vestn. Akad. Med. Nauk (SSSR), 28: 69(1973).

19. Baluja, G.; Murado, M.A., and Hernandez, L.M. Organochlorine pesticides and PCBs distribution in tissues of purple heron and spoon ducks from the biological reserve of Donana (Spain). Bull. Environ. Contam. Toxicol. 17: 603(1977).

20. Baluja, G.; Hernandez, L.M.; Gonzalez, M.J., and Rico,M.C. Presence of organochlorine pesticides and polychlorinated biphenyls and mercury in Spanish human milk samples. Bull. Environ. Contam.Toxicol. 28: 573(1982).

21. Barnett, R.W.; D'erole, A.J.; Cain, J.D., and Arthur,R.D. Organochlorine pesticide residues in human milk samples from women living in north-west and north east Mississipi, USA, 1973-1975. Pestic. Monit.J. 13: 47(1979).

22. Barquet, A.; Morgade, C., and Pfaffenberger, C.D. Determination of organochlorine pesticides and metabolites in drinking water, human blood serum and adipose tissue. J. Toxicol. Environ. Health 7: 469(1981).

23. Barthel, E. Increased risk of lung cancer in pesticide-exposed male agricultural workers. J. Toxicol. Environ. Health 8: 1027(1981).

24. Bercovici, B.; Wassermann, M.; Wassermann, D.; Cucos,S., and Ron, M. Missed abortions and some organochlorine compounds: Organochlorine insecticides(OCl) and polychlorinated biphenyls(PCBs). Acta Med. Leg. Socialis 30: 177(1980).

25. Bercovici, B.; Wassermann, M.; Cucos, S.; Ron, M.; Wassermann,D., and Pines, A. Serum levels of polychlorinated biphenyls and some organochlorine insecticides in women with recent and former missed abortions. Environ. Res. 30: 169(1983).

*26. Bezuglyi, V.P. and Kaskevich, L.M. Some indices of the functional state of the liver in flying personnel engaged in aerial chemical operations. Gig. Tr. Prof. Zabol. 13: 52(1969).

*27. Bezuglyi, V.P.; Odintsova, I.L.R., and Gorskaya, N.Z. Morphological composition of the blood in persons working with a complex of organochlorine and organophosphorus pesticides. Vrach. Delox. 11: 150(1973).

28. Bidleman, T.F.; Christensen, E.J.; Billings, W.N., and Leonard, R. Atmospheric transport of organochlorines in the North Atlantic gyre. J. Mar. Res.39:443(1981).

29. Bitman, J.; Cecil, H.C.; Harris, S.J., and Fries, G.H. Estrogenic activity of p,p'-DDT in the mammalian uterus and avian oviduct. Science 162: 371(1968).

30. Bitman, J.; Cecil, H.C.; Harris, S.J., and Fries,G.H. DDT induces a decrease in eggshell calcium. Nature 224: 44 (1969).

31. Bitman, J.; Cecil, H.C.; Harris, S.J., and Feil, V.J. Estrogenic activity of o,p'-DDT metabolites and related compounds. J. Agric. Food Chem. 26: 149(1978).

*32. Boiko, V.G. and Krasnyuk, E.P. Clinical characteristics of the pathology of respiratory tracts of persons working with DDT. Z. Ushn. nos. gorl. Bolezn.29:35(1969).

33. Bogan, J.A. and Newton, I. Redistribution of DDE in sparrow hawks during starvation. Bull. Environ. Contam. Toxicol. 18: 317(1977).

34. Bouch, G.M. and Matsumura,F. Pesticide degradation by marine algae. Natl. Tech. Inform. Serv. AD-A008, 275: pp.25(1975).

35. Bowman, M.C.; Schechter, M.S., and Carter,R.L. Behaviour of chlorinated insecticides in broad spectrum of soil types. J. Agric. Food Chem. 13: 360(1965).

36. Brown, A.W.A. Ecology of pesticides. Wiley-Interscience Publication. pp.280(1978).

37. Brown, N.J. and Brown, A.W.A. Biological fate of DDT in a subarctic environment. J. Wildl. Manage. 34: 929(1970).

38. Brown, J.R. and Chow, L.Y. Comparative study of DDT and its derivatives in human blood samples in Norfolk

county and Holland Marsh, Ontario. Bull. Environ. Contam. Toxicol. 13: 483(1975).

39. Brown, J.R.; Chow, L.Y., and Chai, F.C. Distribution of organochlorine pesticides in an agricultural environment Holland Marsh, Ontario, 1970-72. Pestic. Monit. J. 9: 30(1975).

*40. Burkatzkaya, E.N.; Voitenko, G.A.,and Krasniuk, E.P. Working condition and health status of workers at DDT production plants. Gig. Sanit. 26: 24(1961).

41. Burlington, H. and Lindeman, V.F. Effect of DDT on testes and secondary sex characteristics of White Leghorn cockerels. Proc. Soc. Exp. Biol. 74: 48(1950).

42. Burns, R.G. Factors affecting pesticide loss from soil. In: Soil Biochemistry Vol. 4, E.A. Paul and A.D.McLaren (Eds.) Marcel Dekker Inc., pp.103 (1975).

43. Butler, G.L. Algae and Pesticides. Residue, Rev. 66: 19(1977).

44. Cameron, G.R. and Burgess, F. The toxicity of 2,2-bis-(p-chlorophenyl)-1,1,1-trichloroethane (DDT). Br. Med. J. 1: 865(1945).

45. Canonne, P. and Marabachi, G. Organochlorine insecticide residues in sediments of the upper estuary of the St. Lawrence River. Bull. Environ. Contam. Toxicol. 14: 83(1975).

46. Carey, A.E. Monitoring pesticides in agricultural and urban soils of the USA. Pestic. Monit. J. 13: 23(1979).

47. Carey, A.E.; Wiersma, G.B., and Tai, H. Pesticide residues in urban soils from 14 United States cities, 1970. Pestic. Monit. J. 10: 54(1976).

48. Carey, A.E.; Gowen, J.A.; Tai, H.; Mitchell, W.G., and Wiersma, G.B. Pesticide residue levels in soils and crops from 37 States, 1972-National Soil Monitoring Programme (IV). Pestic. Monit. J. 12:209(1979a).

49. Carey, A.E.; Douglas, P.; Tai, H.; Mitchell,W.G.,and Wiersma, G.B. Pesticide residue concentrations in soil of 5 USA cities, 1971: Urban Soil Monitoring Programme. Pestic. Monit. J. 13: 17(1979b).

50. Case, R.A.M. Toxic effects of 2,2-bis(p-chlorophenyl)-1,1,1-trichloroethane (DDT) in man. Bri Med. J. 2: 842(1945).

*51. Castro,T.F. and Yoshida, T. Effects of organochlorine matter on the biodegradation of some organochlorine insecticides in submerged soils. Soil Sci. Plant Nutr. 20: 363(1974).

52. Cecil, H.C.; Bitman, J. and Harris, S.J. Estrogenicity of o,p'-DDT in rats. J. Agric. Food Chem. 19:61(1971).

53. Chauhan, R.; Singh, Z.,and, Singh, S. Determination of organochlorine insecticide residues in vegetable samples at Hissar (Haryana) India. Indian J. Ecol. 10: 16(1983).

54. Chin, Y. and T'ant, C. The effect of DDT on cutaneous sensation in man. Science 103: 654(1946).

55. Chisholm, D. and McPhee, A.W. Persistence and effects of some pesticides in soil. J. Econ. Entomol. 65: 1010(1972).

56. Choi, W..W. and Chen, K.Y. Association of chlorinated hydrocarbon with fine particles and humic substances near shore superficial sediments. Environ. Sci. Technol. 10: 782(1976).

*57. Ciupe, R.; Gabor, S.; Fabian, F., and Olteam,L. Contribution to the evaluation of occupational exposure to DDT and BHC. Rev. Ig. Bacteriol. Virusol. Parazitol. Epidemio. Pneumofliziol. Ser. Ig. 28:357(1979).

58. Clegg, D.E. Chlorinated hydrocarbon pesticide residues in oysters (Crassostrea commercialis) in Moreton Bay, Queensland, Australia, 1970-72. Pestic. Monit.J. 8: 162(1974).

*59. Cocisium, M.; Aizicovici, H.; Nistor,C; Unterman,H.W.; Barbuts, R., and Gugles, E. Variation of the DDT and HCH contents of maternal milk in connection with food intake. Igiena 25: 105(1976).

60. Conway, I.S. and Brain, W.T. Organochlorine pesticide residues in human milk, Western Australia, 1970-1971. Pestic. Monit. J. 9: 64(1975).

61. Cooke, B.K. and Stringer, A. Distribution and breakdown of DDT in orchard soil. Pestic. Sci. 13:545(1982).

62. Curley, A. and Kimbrough, R. Chlorinated hydrocarbon insecticides in plasma and milk of pregnant and lactating women. Arch. Environ. Health 18: 156(1969).

63. Curley, A.; Copeland, M.F., and Kimbrough, R.D. Chlorinated hydrocarbon insecticides in organs of stillborn and blood of newborn babies. Arch. Environ. Health 19: 628(1969).

64. Currie, R.A.; Kadis, V.W.; Breitkreitz, W.E.; Cunningham,G.B., and Bruns, G.W. Pesticide residues in human milk, Alberta, Canada: 1966, 1970, 1977-1978. Pestic. Monit. J. 13: 52(1979).

65. Dahya, B. and Chauhan, R. Organochlorine insecticide residues in vegetable samples from Hissar (India),market. Indian J. Agric. Sci. 52:533(1982).

66. Dale, W.E.; Copeland, M.F., and Hayes, W.J.,Jr. Chlorinated insecticides in the body fat of people of India. Bull. W.H.O. 33: 471(1965).

67. Dale, W.E.; Curley, A.,and Cueto, C.,Jr. Hexane extractable chlorinated pesticides in human blood. Life Sci. 5:47(1966).

68. Darrow, G.C. and Harding, G.C.H. Accumulation and apparent absence of DDT metabolism by marine copepod, Calanus Sp. in culture. J. Fish. Res. Board Can. 32: 1845(1975).

69. Datta, P.R. and Nelson, M.F. p,p'-DDT detoxication by isolated perfused rat liver and kidney. Ind. Med. Surg. 39: 195(1970).

70. Davis, B.N.K. The soil macrofauna and organochlorine residues at twelve agricultural sites near Huntingdon. Ann. Appl. Biol. 61: 29(1968).

71. Davis, B.N.K. Laboratory studies on the uptake of dieldrin and DDT by earthworms. Soil Biol. Biochem. 3: 221(1971).

72. Dean, M.E.; Smeaton, T.C., and Stock, B.H. The influence of fetal and neonatal exposure to DDT on the testosterone status of neonatal male rat. Toxicol. Appl. Pharmacol. 53: 315(1980).

73. deCampos, M. and Olszyna-Marzys, A.E. Contamination of human milk with chlorinated pesticides in Guatemals and in El Salvador. Arch. Environ. Contam. Toxicol. 8: 43(1979).

74. Deichmann, W.B. and MacDonald, W.E. Organochlorine pesticides and liver cancer deaths in the United States, 1930-1972. Ecotoxicol. Environ. Saf. 1:89(1977).

75. Deichmann, W.B. and Radomski, J.L. Retention of pesticides in human adipose tissue-Preliminary report. Ind. Med. Surg. 37: 218(1968).

76. Deichmann, W.B.; MacDonald, W.E.; Beasley, A.G.,and Cubit,D. Subnormal reproduction in beagle dogs induced by DDT and aldrin. Ind. Med. Surg. 40:10(1971).

77. DeLong, R.L., Gilmartin, W.G., and Simpson, J. Premature birth in California sea lions: Association with high organochlorine pollutant residue levels. Science 181: 1168(1973).

78. Dharam Vir. Studies on the levels of DDT-residues in soil and human blood in Delhi and its metabolism in certain animals. Ph.D. Thesis, University of Delhi, India (1976).

79. Dillion, J.C.; Martin, G.B., and O'brien, H.T. Pesticide residue in human milk. Food Cosmet. Toxicol. 19: 437(1981).

80. Dindal, D.L. and Wurzinger, K.H. Accumulation and excretion of DDT by the terrestrial snail, Cepaca hortensis. Bull. Environ. Contam. Toxicol. 6: 362(1971).

*81. Dinerman, A.A. and Rozhdestvenskaya, N.A. An experimental study of remote consequences of the migration of pesticides. Gig. Sanit. 39: 22(1974).

82. Doguchi, M. and Fukano, S. Residue levels of polychlorinated

terphenyls, polychlorinated biphenyls and DDT in human blood. Bull. Environ. Contam. Toxicol. 13: 57(1975).

*83. Domenjoz, R. Experimental findings with a new inescticide (Neocid, Geigy), a contribution to the theory contact-toxicity action. Schweiz. Med. Wochenschr. 74: 952(1944).

84. Domenjoz, R. On the biological action of a DDT-derivative. Arch. Int. Pharmacodyn. Ther. 73:128(1946).

85. Draize, J.H.; Nelson, A.A., and Calvery, H.O. The percutaneous absorption of DDT (2,2-bis-(p-chlorophenyl)-1,1,1-trichloroethane) in laboratory animals. J. Pharmacol. Exp. Ther. 82: 159(1944).

86. Dustman, E.H. and Stickel, L.F. The occurrence and significance of pesticide residues in wild animals, Conference on Biological Effects of Pesticides in Mammalian Systems. Ann. N.Y. Acad. Sci. 16: 162(1969).

87. Edwards, C.A. Insecticide residues in soil. Res.Rev.13:83 (1966).

88. Edwards, C.A. Nature and origins of pollution of aquatic systems by pesticides. In: Pesticide in aquatic environments. M.A.Q Khan (Ed.) Plenum Press. pp.11(1977).

89. Edwards, C.A. Environmental problems caused by pesticides. Proceedings of "All India Symposium of Pesticide Residues in the Environment" - UNDP/ICAR/UAS, Bangalore, India (1978).

90. Edwards, C.A. and Jeffs, K. Rate of uptake of DDT from soil by earthworms. Nature 247:157(1974).

91. Edwards, C.A. Persistent pesticides in the environment, C.R.C. Press, Cleveland, Ohio. pp.1-170(1973).

92. Elzorgani, G.A. and Musa, A. Organochlorine insecticides in the blood of occupationally exposed people in Sudan. Bull. Environ. Contam. Toxicol. 16: 15(1976).

93. Ernst, W.; Goerke, H.; Eder, G., and Schaefer, R.G. Residues of chlorinated hydrocarbons in marine organisms in relation to size and ecological parameters.1 PCBs, DDT, DDE and DDD in fishes and mollusks from the English Channel. Bull. Environ. Contam. Toxicol. 15: 55(1976).

94. Fennah, R.G. Preliminary tests with DDT against insect pests of food crops in the Lesser Antilles. Trop. Agric. 22: 222(1945).

95. Fishbein, L. Teratogenic, mutagenic and carcinogenic effects of insecticides. In: Proceedings of International Symposium on Recent Advances in the Assessment of the Health Effects of Environmental Pollution, Commission of the European Communities, Luxembourg.

96. Fishbein, L.F. Chromatographic and biological aspects of DDT and its metabolites. J. Chromatogr. 98:177(1974).

*97. Fitzhugh, O.G. Use of DDT insecticides on food products. Ind. Eng. Chem. 40: 704(1948).

98. Fowler, S.W. and Elder, D.L. PCB and DDT residues in a Mediterranean pelagic food chain. Bull. Environ. Contam. Toxicol. 19: 244(1978).

99. Frank, R.; Armstrong, A.E.; Boelams, R.G.; Braun, H.E.,and Douglas, C.W. Organochlorine insecticide residues in sediment and fish tissues. Ontario, Canada. Pestic. Monit. J. 7: 165(1974).

100. Gablick, J.; Al-Zubaidy, T., and Askari, E. DDT and immunological responses.III. Reduced anaphylaxis and mast cell population in rats fed DDT. Arch. Environ. Health 30: 81(1975).

*101. Garrett, R.M. Toxicity of DDT for man. J. Med. Assoc. Alabama 17: 74(1947).

102. Genina,S.A.; Svetlaja, E.N., and Komarova,L.J. Blood levels of organochlorine pesticides and some haematological indicators in people employed in applying pesticides from the air. In: The hygiene of applica-

tion and the toxicology of pesticides and the clinical features of pesticide poisoning. A collection of papers, Issue No.7, Medved,M.E. (Ed.), Kiew, Vniigintoks, pp.492(1969).

*103. Gil, G.P. and Miron, B.F. Investigations on toxicity of DDT man. Med. Colon. 14: 459(1949).

104. Gingell, R. Metabolism of ^{14}C-DDT in the mouse and hamsters. Xenobiotica 6: 15(1976).

105. Gish, C.D. Organochlorine insecticide residues in soils and soil invertebrates from agricultural lands. Pestic. Monit. J. 3: 241(1970).

106. Gobbetti, A. Endocrine modifications produced by o,p'-DDT in the male adult rat. Pestic. Biochem. Physiol. 14: 139(1980).

107. Goldberg, E.D.; Bowen, V.T.; Farrington, J.W.; Harvey, G.; Martin, J.H.; Parker, P,O.; Risebrough, R.N.; Robertson, W.; Schneider, E., and Gamble, E. The mussel watch. Environ. Conserv. 5:101(1978).

108. Goring, C.A.I.; Laskowski, D.A.; Hamaker, and Meikle, R.W. Principles of pesticide degradation in soil.In: Environmental dynamics of pesticides. R. Haque and V.H. Freed (Eds.), Plenum Press. pp.135(1975).

109. Griffith, F.D.,Jr. and Blanke, R.V. Pesticides in people - Blood organochlorine pesticide levels in Virginia residents. Pestic. Monit. J. 8: 219(1975).

110. Grzenda, A.R.; Parris, D.F., and Taylor, W.J. The uptake, metabolism and elimination of chlorinated residues by Gold fish (Carassius auratus) fed on 14 ^{14}C-DDT contaminated diet. Trans. Am. Fish Soc. 99: 185(1970).

111. Guenzi, W.D. and Beard, W.D. The effect of temperature and soil.water on conversion of DDT to DDE in soil. J. Environ. Quality 5: 243(1976).

112. Gupta, B. and Agarwal, H.C. Metabolism and distribution of DDT in mouse in relation to poisoning symptoms. Ind. J. Exp. Biol. 12: 402(1974).

113. Gupta, S.C. and Kalra, R.L. Insecticide residues in market and farm-gate samples of vegetables at Ludhiana, Punjab, India. Ind. J. Ecol. 10: 20(1983).

114. Guzelian, P.S. and Wolff, M.S. Body clearance of halogenated hydrocarbons: Workshop Summary. Ann. N.Y. Acad. Sci. 320: 271(1979).

115. Haag, H.B.; Einnegan, J.K.; Larson, P.S.; Dreyfuss, M.L.; Main, R.J., and Riese, W. Comparative chronic toxicity for warm-blooded animals of 2,2-bis-(p-chlorophenyl)-1,1,1-trichloroethane(DDD). Ind. Med. Surg. 17: 477(1948).

*116. Hamakar, J.W. and Goring, C.A.I. Turnover of pesticide residues in soil. ACS Symp. Ser. 29:239(1976).

117. Haque, R. Role of adsorption in studying the dynamics of pesticides in a soil environment. In: Environmental dynamics of pesticides. R. Haque and V.H. Freed (Eds.) Plenum Press, New York and London, pp.97-114(1975).

118. Haque, R. and Ash, N. Factors affecting the behaviour of chemicals in the environment. In: Survival in toxic environment. M.A.Q.Khan and J.P.Bederka, Jr.(Eds.) Academic Press, New York,pp.357-371 (1975).

*119. Hargrave,B.T. and Phillips, G.A. Adsorption of ^{14}C-DDT to particle surfaces. Proc. Int. Cong. Transp. Persistent Chem. Aquat. Ecosyst. 2:13(1974).

120. Hargrave, B.T. and Phillips, G.A. DDT residues in benthic invertebrates and demersal fish in St.Margret's Bay, Nova Scotia. J. Fish Res. Board Can. 33: 1698(1976).

121. Hayes, W.J.,Jr.; Durham, W.F., and Cueto, C.,Jr. The effect of known repeated oral doses of chlorophenothane (DDT) in man. J. Am. Med. Assoc. 162: 890(1956).

122. Hayes, W.J.,Jr. Pharmacology and toxicology of DDT. In: DDT, the insecticide dichlorodiphenyl trichlorothane and its significance. Vol.2 P.Muller(Ed.)

Birkhauser Verlag, Basel. pp.9-247(1959).

123. Hayes, W.J.,Jr. Review of the metabolism of chlorinated hydrocarbon insecticides especially in mammals. Ann. Rev. Pharmacol. 5: 27(1965).

124. Hayes, W.J., Jr. Toxicology of pesticides, Baltimore, Williams & Wilkins Co., pp.580(1975).

125. Heath, R.G.; Spann, J.W., and Kreitzer, J.F. Marked DDT impairment of mallard reproduction in controlled studies. Nature 224: 47(1969).

126. Heinricks, W.J.; Gellert, R.J.; Bakke, J.L., and Lawrence,N.L. DDT administered to neonatal rats induces persistent estrous syndrome. Science 173: 642(1971).

*127. Hoffman, I. and Lendle, L. On the action of new insecticide compounds. Arch. Exp. Path. Pharmakol. 205: 223(1948).

128. Hofvander, Y.; Hagman, U.; Linder, C.E.; Vaz, R., and Slorach, S.A. WHO Collaborative Breast Feeding Study: 1. Organochlorine contaminants in individual samples of Swedish human milk, 1978-79. Acta Paediatr. Scan. 70: 3(1981).

129. Hort, M.M.; Andson, R.H., and Fabros, S. Prematurity and intrauterine growth retardation induced by DDT in rabbit. Arch. Int. Pharmacodyn. Ther. 192: 286(1971).

130. Howie, P.W.; Purdie, D.W.; Begg, C.B., and Prentice,C.R.M. Use of coagulation tests to predict the clinical progress of pre-eclampsia. Lancet 2:323(1976).

131. Hsieh, H.C. DDT intoxication in a family in Southern Taiwan. Am. Med. Assoc. Arch. Ind. Hyg. 10:334(1954).

132. Hurlbert, S.H. Secondary effects of pesticides on aquatic ecosystem. Res. Rev. 57:81(1975).

133. Ireland, J.S.; Mukku, V.R.; Robison, A.K., and Stancel,G.M. Stimulation of uterine DNA synthesis by o,p'-DDT. Biochem. Pharmacol. 29:1469(1980).

134. Gager, K.W. Aldrin, Dieldrin, Endrin, and Telodrin: An epidemiological and toxicological study of long-term occupational exposure, New York, Elsevier, pp.239(1970).

*135. Jafferies, D.J. Some sublethal effects of p,p'-DDT and its metabolites p,p'-DDE on breeding passerine birds. Meded Fakult Land bouw wetenschappen Gent. 36: 34(1971).

136. Johnson, B.T.; Sanders, C.R.; Sanders, H.O., and Campbell,R.S. Biological magnification and degradation of DDT and aldrin by fresh water invertebrates. J.Fish Res. Board Can. 28: 709(1971).

137. Johnston, D.W. Organochlorine pesticide residues in small migratory birds, 1964-73. Pestic. Monit. J. 9: 79(1975).

138. Jonas, R.B. and Pfaender, F.K. Chlorinated hydrocarbon pesticides in Western North Atlantic Ocean. Environ. Sci. Technol. 10: 770(1976.

139. Jonsson, V.; Liu, G.J.K.; Armbruster, J.; Kettlehut, L.L.,and Drucker, B. Chlorohydrocarbon pesticide residues in human milk in Greater St.Louis Missouri, 1977. Am. J. Clin. Nutr.30:1106(1977).

140. Joseph, D.; William, L.; Money, D.S., and Mortimer,L. Transport of aminoacids by placenta. Am. J. Obst. Gynaecol. 101: 820(1968).

141. Judah, J.D. Studies on the metabolism and mode of action of DDT. Br. J. Pharmacol. 4: 120(1949).

142. Juengst, F.W.,Jr. and Alexander,M. Effect of environmental conditions on the degradation of DDT in model marine ecosystems. Natl. Tech. Inform. Serv. AD-A020, 570. pp.6(1975).

143. Juengst, F.W., Jr. and Alexander, M. Conversion of DDT to water soluble products by micro-organisms. J. Agric. Food. Chem. 24: 111(1976).

144. Kalra, R.L. and Chawla, R.P. Occurrence of DDT and BHC residues in human milk in India. Experientia 37:404(1981).

145. Kaphalia, B.S. and Seth, T.D. DDT and BHC residues in some body tissues of goat, buffalo and chickens, Lucknow, India. Pestic. Monit. J.15:103(1981).

146. Kapoor, I.P.; Metcalf, R.L.; Nystrom, R.F., and Sangha, G.K. DDT in mouse, insect and in a model ecosystem. J. Agric. Food Chem. 18: 1145(1970).

147. Kapoor, I.P.; Metcalf, R.L.; Hirwe, A.S.; Po-Yung Lu; Coats, J.R., and Nystrom, R.M. Comparative metabolism of DDT, methoxychlor and ethoxychlor in mouse, insects and in a model ecosystem. J. Agric.Food Chem. 20: 1(1972).

*148. Karimov, A.M. Skin sensitivity to organochlorine insecticides. Med. Zh. Uzbekistant. 9: 58(1969).

*149. Karimov, A.M. Occupational skin diseases in cotton growers caused by chemical poisons and measures for their prevention. Gig. Tr. Prof. Zabol.14:35(1970).

150. Kashyap, S.K.; Nigam, S.K.; Karnik, A.B.; Gupta, R.C., and Chatterjee, S.K. Carcinogenicity of DDT in pure inbred Swiss mice. Int. J. Cancer 19: 725(1977).

151. Kauthacer, B.; Alebic-Kolbah,T.; Buntic, A.; Tkalcevic,B.,and Reiner, E. DDT residues in samples of human milk, and mother's and cord blood serum in a continental town in Crotia (Yugoslavia). Int.Arch. Occup. Environ. Health 46: 267(1980).

152. Keplinger, M.L.; Deichmann, W.B., and Sala, F. Effect of combinations of pesticides on reproduction in mice. In: Pesticide Symposia. W.B. Deichman(Ed.) Halos and Associate, Miami, pp.125(1970).

153. Keil, J.E.; Sandifer, S.H.; Finklea, J.H., and Priester, L.E. Serum vitamin A elevation in DDT exposed volunteers. Bull. Environ. Contam. Toxicol. 8: 317(1972).

154. Khandekar, S.S.; Noronha, A.B.C., and Banerji,S.A. Organochlorine pesticide residues in vegetables from Bombay (India) markets. A 3 year assessment. Environ. Pollut. Ser. B Chem. Phys. 4:127(1982).

155. Kiigemagi, V. and Terriere, L.C. Persistence of DDT in orchard soils. Bull. Environ. Contam. Toxicol. 7: 348(1972).

*156. Kilf, L.F.; Peakall, D.D., and Wilbur, S.R. Recent changes in California condor (Gymnogyps californianus) eggshell. Condor. 81: 166(1979).

157. Kimura, T. and Brown, A.W.A. DDT dehydrochlorinase in Aedes aegypti. J. Econ. Entomol. 57: 710(1964).

158. King-Kirke, A.; Meeker, D.L., and Swineford, D.M. White-faced ibis (Plegadis chihi) populations and pollutants in Texas, U.S.A., 1969-1976. Southwest Nat. 25: 225(1980).

159. Klein, A.K.; Laug, E.P.; Datta, P.R.; Watts, J.O., and Chen, J.T. Metabolites:Reductive dechlorination of DDT to DDD and isomeric transformation of o,p'-DDT to p,p'-DDT in vivo. J. Assoc. Agric. Chem. 47: 1129(1964).

*160. Klein, W. and Korte, F. Metabolism of chlorinated hydrocarbons. In: Chemie der Pflanzenschutz-und Schadlingsbekampfungsmittel. R. Wegler(Ed.) Band I,Berlin, Springer Verlag, pp.199-218(1970).

161. Kodric-Smit, M.; Smit, Z., and Olie, K. Organochlorine contaminants in human milk from slavonia Province, Yugoslavia, 1978. Pestic. Monit. J. 14:1(1980).

162. Korte, F. Transformation of p,p'-DDT in the environment. In: Environmental Health Criteria. 9. DDT and its metabolites. United Nations Environmental Program and the World Health Organization, Geneva (1979).

*163. Kostiuk, O.T. and Mukhtarova, N.D. Catecholamines and the functional state of the hypothalamus under the action of a complex of organochlorine and organophosphorus pesticides. Gig. Tr. Prof. Zabol. 14: 35(1970).

*164. Krasniuk, E.P.; Loganovskii, N.G.; Makovskaya,E.I., and Rapport, M.B. Functional and morphological changes in the kidneys during the effects of DDT on the body. Sov. Med. 31: 38(1968).

*165. Krasniuk, E.P. and Platonova, V.I. Functional disorders of the stomach under prolonged effects of organochlorine chemical poisons. Urach. Delo. 9: 99(1969).

166. Kraybill, H.F. Significance of pesticide residues in foods in relation to total environmental stress. Can. Med. Assoc. J. 100: 204(1969).

167. Kreiss, K.; Zack, M.W.; Kimbrough, R.D.; Needham, L.L.; Smerk, A.L., and Jones, B.T. Cross-sectional study of a community with exceptional exposure to DDT. J. Am. Med. Assoc. 245: 1926(1981).

168. Kuper, D. and Bulgar, W.H. In: Estrogens in the environment. J.A. McLachlan (Ed.) Elsevier/North Holland, New York, pp.239(1980).

*169. Kuyumdzhieva, T. Organochlorine pesticide level in soil of Bulgaria. Khig. Zdraveopaz. 22: 163(1979).

170. Lahser, C. and Applegate, H.G. Pesticides at Presidio. III. Soil and water. Texas J. Sci. 18: 12(1966).

171. Lang, J.T.; Rodriguez, L.L.,and Livingston, J.M. Organochlorine pesticide residues in soils from six U.S. Airforce Bases, 1975-76. Pestic. Monit. J. 12: 230(1979).

172. Laws, E.R., Jr.; Curley, A., and Biros, F.J. Men with intensive occupational exposure to DDT. A clinical and chemical study. Arch. Environ. Health 15:766(1967).

173. Laws, E.R., Jr.; Maddrey, W.C.; Curley, A., and Burse, V.W. Long-term occupational exposure to DDT. Arch. Environ. Health 27: 318(1973).

*174. Levin, W.; Welch, R.M., and Conney, A.H. Estrogenic action of DDT and its analogs. Fedn. Proc. Fedn. Am. Soc. Exp. Biol. 27: 649(1968).

*175. MacCormack, J.D. Infestation and DDT. Irish J. Med. Sci. 6: 627(1945).

*176. Mackerras, I.M. and West, R.F.K. 'DDT' poisoning in man. Med. J. Aust. 1: 400(1946).

177. Matsumura, F. Degradation of pesticide residues in the environment. In: Environmental pollution by pesticides. C.A. Edwards (Ed.) Plenum Press. pp.494-513(1973).

178. Matsumura, F. Microbial degradation of pesticides. In: Survival in toxic environments. M.A.Q. Khan and J.P. Bederka Jr.(Eds.). pp.129-154(1974).

179. Mattson, A.M.; Spillane, J.T.; Baker, C., and Pearce,G.W. Determination of DDT and related substances in human fat. Annal. Chem. 25: 1065(1953).

*180. Matuo, Y.K.; Lopes, J.N.C., and Lopes, J.L.C. DDT levels in human milk from Ribeirao Preto (Brazil). Rev. Bras. Biol. 40: 293(1980).

181. McLachlan, J.A. and Dixon, R.L. Gonadal function in mice exposed prenatally to p,p'-DDT. Toxicol. Appl. Pharmacol. 22: 327(1972).

182. Menzel, D.B.; Smith, S.M.; Miskus, P.,and Hoskins, W.M. The metabolism of ^{14}C-labelled DDT in the larvae and adult of Drosophila melanogaster. J. Econ. Entom. 54: 9(1961).

183. Menzie, C.M. Metabolism of pesticides, U.S. Government Printing Office, Special Scientific Report, Wildlife No.127, Washington,D.C.(1969).

184. Mes, J. Variation in the polychlorinated biphenyl and organochlorine pesticide residues during breast feeding and its diural pattern. Chemosphere 7:699(1978).

185. Mes, J. and Davies, D.J. Presence of polychlorinated biphenyls and organochlorine pesticide residues and the absence of polychlorinated terphenyls in Canadian human milk samples. Bull. Environ. Contam. Toxicol. 21: 381(1979).

186. Mes, J.; Doyle, J.A.; Adams, B.R.; Davies, D.J., and Turton,D. Polychlorinated biphenyls and organochlorine pesticides in milk and blood of Canadian women during lactation. Arch. Environ. Contam. Toxicol. 13: 217(1984).

187. Miles, J.R.W. and Harris, C.R. Insecticide residues in organic soils of six vegetable growing areas in South Western Ontario, 1976. J. Environ. Sci. Health 13: 199(1978).

188. Miyazaki, S. and Thorsteinson, A.J. Metabolism of DDT by fresh water diatoms. Bull. Environ. Contam. Toxicol. 8: 81(1972).

189. Moakes, D.N. and Benfield, C.A. Tissue accumulation of DDT and its metabolites in the domestic fowl. J. Sci. Food Agric. 16: 693(1965).

190. Morgan, D.P. and Lin, I.L. Blood organochlorine pesticide concentrations, clinical hematology and biochemistry in workers occupationally exposed to pesticides. Arch. Environ. Contam. Toxicol. 7: 423(1978).

191. Morgan, D.P. and Roan, C.C. Liver function in workers having high tissue stores of chlorinated hydrocarbon pesticides. Arch. Environ. Health 29:14(1974).

192. Mukerjee, D.; Roy, B.R.; Chakraborty, J.,and Ghosh, B.N. Pesticide residues in human foods in Calcutta, India. Indian J. Med. Res. 72: 577(1980).

*193. Naevested, R. Poisoning with DDT powder and some other incidences of poisoning. Tidsskr. Norske Laegeforen. 67: 261(1947).

194. Nash, R.G. and Woolson, E.A. Persistence of chlorinated hydrocarbon insecticides in soils. Science 157: 924(1967).

195. Neal, P.A.; Von Oettingen, W.F.; Smith, W.W.; Malino, R.B.; Dunn, R.C.; Moran, H.E.; Sweeney, T.R.; Armstrong, D.W., and White, W.C. Toxicity and potential dangers of aerosols, mists, and dusting powders containing DDT. Public Health Rep. 177:1(1944).

*196. Need, J.A. Some immunological aspects of pre-eclampsia. Perspect. Nephrol. Hypertension 5:169(1976).

197. Neudorf, S. and Khan, M.A.Q. Pick up and metabolism of DDT, dieldrin and photodieldrin by a fresh water alga (Ankistro desmus ammalloides) and a micro

crustacean (Daphnia pulex). Bull. Environ. Contam. Toxicol. 13: 433(1975).

198. Nikitina, Y.I. Course of labor and puerperium in vineyard workers and milk maids in the crimea. Gig. Tr. Prof. Zabol. 18: 17(1974).

199. Noren, K. Organochlorine contaminants in Swedish human milk from the Stockholm region. Acta Paediatr. Scan. 72: 259(1983).

200. North, H.H. and Menzer, R.E. Metabolism of DDT in human embryonic lung cell cultures. J. Agric. Food Chem. 21: 509(1973).

201. O'Leary, J.A.; Davies, J.E.; Edmundson, W.F., and Reich, G.A. Transplacental passage of pesticides. Am. J. Obst. Gynaecol. 107: 65(1970a).

202. O'Leary, J.A.; Davies, J.E., and Feldman, M. Spontaneous abortion and human pesticide residues of DDT and DDE. Am. J. Obst. Gynaecol. 108:1291(1970b).

203. Olsson, M.; Kihlstrom, J.E.; Jensen, S., and Orberg, J. Cadmium and mercury concentrations in mink (Mustela vison) after exposure to PCBs. Ambio 8: 25(1979).

204. Olszyna-Marzys, A.E. Contaminants in human milk. Acta Paediatr. Scan. 67: 571(1978).

*205. Olszyna-Marzys, A.E.; de Campos, M.; Farvar, M.T., and Thomas, M.R. Residues of chlorinated pesticides in human milk in Guatemala. Bol. of Sanit. Panam 74: 93(1973).

206. Ortelee, M.H. Study of men with prolonged intensive occupational exposure to DDT. Am. Med. Assoc. Arch. Ind. Health 18: 433(1958).

207. Ottoboni, A. Effects of DDT on reproduction in the rat. Toxicol. Appl. Pharmacol. 14: 74(1969).

208. Ottoboni, A.; Bissell, G.D., and Mexter, A.C. Effect of DDT on reproduction in multiple generations of beagle dogs. Arch. Environ. Contam. Toxicol. 6: 83(1977).

*209. Ouw, K.H. and Shander, A.G. A health survey of Wee Waa residents during 1973 aerial spraying season. Med. J. Aust. 2: 871(1974).

*210. Paz de La Vega, Y.; Cerda, M.; Bergquist, E.; Fuenzalinda, M., and Lemus, D. Teratogenic effects of DDT and polychlorinated biphenyls in chicken embryos. Arch. Med. Vet. 12: 107(1980).

211. Pearce, G.W.; Mattson, A.M., and Hayes, W.J.,Jr. Examination of human fat for presence of DDT. Science 116: 254(1952).

212. Pearce, P.A.; Reynold, L.M., and Peakall, D.B. DDT residues in rain water in New Brunswick and Lawrence, 1967-68. Pestic. Monit. J. 11: 199(1978).

213. Perfect, T.J.; Cook, A.G.; Critchley, B.R.; Critchley, V.; Moore, R.L.; Russel-Smith, A.; Swift, M.J.,and Yendon, R. The effects of DDT on the population of soil organisms and the processes of decomposition in cultivated soil in Nigeria. Ecol. Bull. 25: 565(1977).

214. Perry, A.S.; Miller, S., and Buckner, A.J. The enzymatic *in vitro* degradation of DDT by susceptible and DDT resistant body lice. J. Agric. Food Chem. 11: 457(1963).

215. Peterson, J.E. and Robinson, W.H. Metabolic products of p,p'-DDT in the rat. Toxicol. Appl. Pharmacol. 6: 321(1964).

216. Pfaender, F.K. and Alexander, M. Extensive microbial degradation of DDT *in vitro* and DDT metabolism by natural communities. J. Agric. Food Chem. 20: 842(1972).

217. Phillips, J.; Wells, M., and Chandler, C. Metabolism of DDT by the fresh water Plannarian, *Phagocata velata*. Bull. Environ. Contam. Toxicol. 12: 355(1974).

218. Pillai, M.K.K. and Brown, A.W.A. Physiological and genetical studies on resistance to DDT substitutes in

Aedes aegypti. J. Econ. Entomol. 58:255(1965).

219. Pillai, M.K.K.; Agarwal, H.C., and Yadav, D.V. Tolerance, uptake and metabolism of DDT in Gambusia affinis. Indian J. Exp. Biol.15:40(1977).

220. Plimmer, J.R.; Klingebiel, U.I., and Hummer, B.E. Photo-oxidation of DDT and DDE. Science 167:67(1970).

221. Poland, A.; Smith, D.; Kuntzman, R.; Jacobson, M., and Conney, A.H. Effect of intensive occupational exposure to DDT on phenylbutazone and cortisol metabolism in human subjects. Clin. Pharmacol. Ther. 11: 724(1970).

222. Polishuk, Z.W.; Wassermann, M.; Wassermann, P.; Grover, Y.; Lazarovici, S., and Tomatis, L. Effects of pregnancy on storage of organochlorine insecticides. Arch. Environ. Health 20: 215(1970).

223. Polishuk, Z.W.; Wassermann, D.; Wassermann, M.; Cucos, S., and Ron, M. Organochlorine compounds in mother and fetus during labour. Environ. Res. 13: 278(1977).

224. Proceedings Indo-US Workshop on biodegradable pesticides held at ITRC, Lucknow, India, April, 16-19, (1979).

225. Procianoy, R.S. and Schvartsman, S. Blood pesticide concentration in mothers and their newborn infants: relation to prematurity. Acta Pediatr. Scan. 70: 925(1981).

226. Quinby, G.E.; Armstrong, J.F., and Durham, W.F. DDT in human milk. Nature 207: 726(1965).

*227. Rabello, M.N.; Becak, W.; Almeida, W.F.; Pigati, P; Ungaro,M.T.; Murata, T., and Pereira, C.A.B. Cytogenic study on individuals occupationally exposed to DDT. Mutat. Res. 28: 449(1975).

228. Radhakrishnan, C.V.; Thompson, N.P., and Forrestor, D.J. Susceptibility of chickens fed p,p'-DDT to histomoniasis. Bull. Environ. Contam. Toxicol. 8: 147(1972).

229. Radomski, J.L.; Deichmann, W.B., and Clizer, E.E. Pesticides concentrations in the liver, brain, and adipose tissue of terminal hospital patients. Food Cosmet Toxicol. Appl. Pharmacol. 20: 175(1968).

230. Radomski, J.L.; Deichmann, W.B.; Rey, A.A., and Merkin, T. Human pesticide blood levels as a measure of body burden and pesticide exposure. Toxicol. Appl. Pharmacol. 20: 175(1971).

231. Ramachandran, M.; Sharma, M.I.D.; Sharma, S.C.; Mathur,P.S.; Aravindkshan, A., and Edwards, G.J. DDT and its metabolites in human body fat in India. Bull. W.H.O. 49: 637(1973).

232. Rappolt, R.T. and Hall, W.E. p,p'-DDE and p,p'-DDT residues in human placentas, cords and adipose tissues. Clin. Toxicol. 1: 57(1968).

233. Requejo, A.G.; West, R.H.; Hatcher, P.G., and Mcgillivary, P.A. Polychlorinated biphenyls and chlorinated pesticides in soils of the Everglades National Park, Florida, USA, and adjacent agricultural areas. Environ. Sci. Technol. 13: 931(1979).

234. Reuber, M.D. Interstitial cell carcinomas of the testis in Balb/c male mice ingesting methoxychlor. J. Cancer Res. Clin. Oncol. 93: 173(1979).

235. Risebrough, R.W.; Huggett, R.J.; Griffin, J.J., and Goldberg, E.D. Pesticides: Transatlantic movements in the northeast trades. Science 159: 1233(1968).

236. Robinson, A.K. and Stancel, G.M. The estrogenic activity of DDT: correlation of estrogenic effect with nuclear level of estrogen receptor. Life Sci. 31: 2479(1982).

237. Robinson, J.; Richardson,A.; Carbtree, A.N.; Caulson, J.C., and Potts, G.R. Organochlorine residues in marine organisms. Nature 214: 1307(1967).

238. Rogers, A.; Mellors, A. and Safe, S. Lysosomal membrane labilization by DDT, DDE and PCB. Res.

Commun. Chem. Pathol. Pharmacol.13:341(1976).

239. Rudd, R.L. and Herman, S.G. Ecosystemic transfer of pesticide residues in an aquatic environment. In: Environmental Toxicology of Pesticides. F. Matsumura, G.M. Boush and T. Misato (Eds.) Academic Press, New York and London. pp.471 (1971).

*240. Sadowski, S. Chlorinated hydrocarbon pesticides in the lungs of dead humans. Rocz. Panstw. Zakl. Hig. 31: 313(1980).

*241. Samuelson, B. Biosynthesis and metabolism of prostaglandins. In: Proceedings of the Fourth International Congress on Pharmacology Vol.IV. Schwabe,Basel. pp.12-13(1970).

242. Sandifer, S.H. Industrial and agricultural chemicals. Pediatrics 53: 843(1974).

243. Saxena, M.C.; Siddiqui, M.K.J.; Bhargava, A.K.; Seth, T.D.; Krishnamurti, C.R. and Kutty, D. Role of organochlorine hydrocarbon pesticides in abortion and premature labour. Toxicology 17: 323(1980a).

244. Saxena, M.C.; Seth, T.D. and Mahajan, P.L. Organochlorine pesticides in human placenta and accompanying fluid. Int. J. Environ. Anal. Chem. 7:245(1980b).

245. Saxena, M.C.; Siddiqui, M.K.J.; Bhargava, A.K.; Krishnamurti, C.R. and Kutty, D. Placental transfer of pesticide in humans. Arch. Toxicol. 48:127(1981a).

246. Saxena, M.C.; Siddiqui, M.K.J.; Seth, T.D.; Krishnamurti,C.R.; Bhargava, A.K., and Kutty, D. Organochlorine pesticides in specimens from women undergoing spontaneous abortion, premature or full-term delivery. J. Anal. Toxicol. 5: 6(1981b).

247. Saxena, M.C.; Siddiqui, M.K.J.; Agarwal, V., and Kutty,D. A comparison of organochlorine insecticide contents in specimens of maternal blood, placenta and umbilical cord blood from stillborn and live-born cases. J. Toxicol. Environ.Health

11: 71(1983).

*248. Schmidt, R. Effect of 1,1,1-trichloro-2,2-bis(p-chlorophenyl) ethane (DDT) on the prenatal development of the mouse (under consideration of distribution of tritium-labelled and carbon - 14 - labelled DDT in pregnant mice). Biol. Rundsch 11: 316(1973).

*249. Schroeder, G. and Dorozalska, A. Degradation of DDT and its analogues. Wiad. Chem. 29: 553(1975).

250. Selby, L.A.; Newell, K.W.; Hauser, G.A., and Junker, G. Comparison of chlorinated hydrocarbon pesticides in maternal blood and placental tissue. Environ. Res. 2: 247(1969).

251. Siddiqui, M.K.J.; Saxena, M.C.; Bhargava, A.K.; Seth, T.D.; Krishnamurti, C.R., and Kutty, D. Agrochemicals in the maternal blood, milk and cord blood: a source of toxicants for prenates and neonates. Environ. Res. 24: 24(1981a).

252. Siddiqui, M.K.J.; Saxena, M.C.; Mishra, U.K.; Krishnamurti, C.R., and Nag, D. Long-term occupational exposure to DDT. Int. Arch. Occup. Environ. Health 48: 301(1981b).

253. Siddiqui, M.K.J. and Saxena, M.C. Biological monitoring of environmental contaminants: chlorinated hydrocarbons in eggs of hens. Sci. Total Environ. 32: 29(1983).

254. Skaare, J.U. Persistent organochlorinated compounds in Norwegian human milk in 1979. Acta Pharmacol. Toxicol. 49: 384(1981).

255. Sonawane, B.R.; Muccitelli, R.M.; Wolfe-Lottes, S., and Yaffe, S.J. Long-term effects from prenatal exposure to o,p'-DDT on estradiol receptor binding in reproductive organs. Pediatr. Res. 14: 473(1980).

256. Sowbhagya, H.B.; Usha Devi, A., and Visweswariah,K. Chlorinated insecticide residues in certain food

samples. Indian J. Med. Res. 78: 403(1983).

*257. Springer, M.A. Pesticide levels, eggs and eggshell parameters of great horned owls (Bubo virginianus). Ohio J. Sci. 80: 184(1980).

258. Stammers, F.M.G. and Whitfield, F.G.S. Toxicity of DDT to man and animals. Bull. Entomol. Res. 38:1(1947).

259. Stancel, G.M.; Ireland, J.S.; Mukku, V.R., and Robinson,A.K. The estrogenic activity of DDT: In vivo and in vitro induction of a specific estrogen inducible uterine protein by o,p'-DDT. Life Sci. 27: 1111(1980).

260. Sternberg, J.; Kearns, C.W., and Moorefield, H. DDT dehydrochlorinase, an enzyme found in DDT-resistant flies. J. Agric. Food Chem. 2: 1125(1954).

261. Stotter, D.A. Metal centres and DDT. J. Inorg. Nucl. Chem. 39: 721(1977).

262. Subba Rao, R.V. and Alexander, M. Products formed from analogues of DDT metabolites by Pseudomonas putida. Appl. Environ. Microbiol. 33:101(1977).

*263. Sundaram, K.M.S. DDT residues may be lost from soil by indirect volitilization. Bi-Monthly Res. Notes Can. For. Serv. 30: 14(1974).

264. Sundstrom, G.; Jansson, B.,and Jensen, S. Structure of phenolic metabolites of p,p'-DDE in rats, wild seals and guillemots. Nature 255:627(1975).

265. Sundstrom, G.; Hutzinger, O.; Safe, S., and Platonov, N. The metabolism of p,p'-DDE in the pig. In: Fate of Pesticides in Large animals. G.W. Ivie, and H.W. Dorough (Eds.) Academic Press, NewYork. pp.175(1977).

266. Suzuki, M.; Yamato, Y., and Watanabe, T. Organochlorine insecticide residues in field soils of the Kita Kyushu District-Japan, 1970-74. Pestic. Monit. J. 11: 88(1977).

*267. Takano, H. Results of inspection of pesticide residues in mother's milk and market milk. Annu. Rep. Iwate

Inst. of Public Health. 15: 88(1972).

268. Takashi, W.; Saidin, D.; Takei, G., and Wong, L. Organochlorine pesticide residues in human milk in Hawaii, USA, 1979-1980. Bull. Environ. Contam. Toxicol. 27: 506(1981).

269. Tarjan, R. and Kemeny, T. Multigeneration studies on DDT in mice. Food Cosmet Toxicol. 7:215(1964).

270. Thompson, A.R. Pesticide residues in soil invertebrates. In: Environmental Pollution by Pesticides. C.A. Edwards (Ed.) Plenum Press, London. pp.87-133 (1973).

271. Trautmann, W.L.; Chesters, G.,and Pionke, H.B. Organochlorine insecticide composition of randomly selected soils from nine states - 1967. Pestic. Monit. J. 2: 93(1968).

272. Tripathi, H.C. Organochlorine insecticide residues in agricultural and animal products in Tarai area. M.Sc. Thesis, U.P. Agric. Univ., Pant Nagar, India (1966).

273. Tsutsui, J.; Kato, T., and Nishikawa, T. Results of health survey on persons engaging in pesticide application in Suzuka area, Mie Prefecture. J.Jpn. Assoc. Rural Med. 23: 518(1974).

274. Tu, C.M. and Miles, J.R.W. Interaction between insecticides and soil microbes. Res. Rev. 64: 17(1976).

275. Turnbull, A.C. and Alexander, A.B.M. Uterine function in human pregnancy and labor. In: Scientific Basis of Obstetrics and Gynaecology. R.R.Mac Donald (Ed.) Churchill Livingstone Publisher, Edinburgh. pp.92(1978).

276. Unger, M. and Olssen, J. Organochlorine compounds in the adipose tissue of diseased people with and without cancer. Environ. Res.23:257(1980).

277. U.S. Environmental Protection Agency - Manual of analytical methods for the analysis of pesticides in human and environmental samples. R.R. Watts (Ed.)

EPA-600/8-80-038, Health Effects Research Laboratory, Research Triangle Park, NC 27111 (1980).

278. Vasuki, K. and Ghai, O.P. A prelude to pesticide residues in different diagnostic situations. J. Food Sci. Technol.(Mysore) 12: 203(1975).

279. Velbinger, H.H. Contribution to the toxicity of DDT - agent, dichlorodiphenyl trichloroethane. Pharmazie 2: 268(1947).

280. Vettorazzi, G. State of the art of the toxicological evaluation carried out by the Joint FAO/WHO expert committee on pesticide residues. I. Organohalogenated pesticides used in public health and agriculture. Res. Rev. 56: 107(1975).

281. Walker, C.H. Reductive dechlorination of p,p'-DDT by pigeon liver microsomes. Life Sci. 8:111(1969).

282. Ware, G.W. and Good, E.E. Effects of insecticides on reproduction in the laboratory mouse II Mirex, telodrin and DDT. Toxicol. Appl. Pharmacol. 10:54(1967).

283. Ware, G.W.; Estesen, B.J., and Cahill, W.P. An ecological study of DDT residues in Arizona soils and alfalfa. Pestic. Monit. J. 2: 129(1968).

284. Ware, G.W.; Cahill, W.P., and Estensen, B.J. Volatilization of DDT and related material from dry and irrigated soils. Bull. Environ. Contam. Toxicol. 14: 88(1975).

285. Ware, G.W.; Estensen, B.J.; Buck, N.A., and Cahill, W.P. DDT moratorium in Arizona - Agricultural residues, After seven years. Pestic. Monit. J. 12:1(1978).

*286. Wasicky, R. and Unti, O. Dichloro-diphenyl-trichloroethanol (DDT) does not have any effect on the larvae of "culicides". Arch. Hig. S. Paulo. 9: 87 (1944).

287. Wassermann, M.; Wassermann, D., and Zellermayer, L. Pesticides in people, storage of DDT in the people of Israel. Pestic. Monit. J.1:15(1967).

288. Wassermann, M.; Wassermann, D.; Gershon, Z., and Zellermayer, L. Effects of organochlorine insecticides on body defence systems. Ann.N.Y. Acad. Sci. 160: 393(1969).

289. Wassermann, M.; Wassermann, D.; Kedar, E., and Djavaherian, M. Immunological and detoxication interactions in p,p'-DDT fed rabbits. Bull. Environ. Contam. Toxicol. 6: 426(1971).

290. Wassermann, M.; Bercovici, B.; Cucos, S., Wassermann,D., and Ron,M. Storage of some organochlorine compounds in toxemia of pregnancy. Environ.Res. 22: 404(1980).

291. Wassermann, M; Bercovici, B.; Cucos, S., and Pines, A. Premature delivery and organochlorine compounds: Polychlorinated biphenyls and some organochlorine insecticides. Environ. Res. 28: 106(1982).

292. Welch, R.M.; Levin, W., and Conney, A.H. Estrogenic action of DDT and its analogs. Toxicol. Appl. Pharmacol. 14: 358(1969).

293. White, D.H. Residues of DDT and DDE in livers of water fowl, North-eastern Louisiana, 1970-71. Pestic. Monit. J. 10: 2(1976).

294. White, D.H. and Kaiser, T.E. Residues of organochlorine and heavy metal in ruddy ducks from the Delaware river, 1973. Pestic. Monit. J. 9: 155(1976).

295. WHO. DDT and its metabolites. In: Environmental Health Criteria 9. United Nations Environmental Programme and the World Health Organization, Geneva (1979).

296. Wickstrom, K.; Pyysalo, H., and Siimes, M.A. Levels of chlordane, hexachlorobenzene, PCB and DDT

compounds in Finnish human milk in 1982. Bull. Environ. Contam. Toxicol. 31: 251(1983).

297. Wiemeyer, S.N.; Swineford, D.M.; Spitzer, P.R., and McLain, P.D. Organochlorine residues in New Jersey Osprey eggs. Bull. Environ. Contam. Toxicol. 19: 56(1978).

298. Wigglesworth, V.B. A case of DDT poisoning in man. Br. Med. J. 1: 517(1945).

299. Wilcox, A.R. USPH investigation. DDT health effects. Inter-office correspondence to M.V. Anthony, Stauffer Chemical Co. (1967).

300. Winter, M.; Thomas, M.; Wernick, S.; Levin, S., and Farvar, M.T. Analysis of pesticide residues in 290 samples of Guatemalan mother's milk. Bull. Environ. Contam. Toxicol. 16: 652(1976).

301. Wolfe, H.R. and Armstrong, J.F. Exposure of formulating plant workers to DDT. Arch. Environ. Health 23: 169(1971).

302. Wolfe, H.R.; Walker, K.C.; Elliot, J.W., and Durham, W.F. Evaluation of the health hazards involved in house spraying with DDT. Bull. W.H.O. 20: 1(1959).

303. Wolfe, N.L.; Zepp, R.G.; Paris, D.F.; Baughman, G.L.,and Hollis, R.C. Methoxychlor and DDT degradation in water: rates and products. Environ. Sci. Technol. 11: 1077(1977).

304. Woodard, B.T.; Ferguson, B.B., and Wilson, D.J. DDT levels in milk of rural indigent blacks, Research Triangle Park, NC, Health Effect Research Laboratory, US Environmental Protection Agency, EPA 600/1-76-032 (1976).

305. Woodwell, G.M.; Craig, P.P., and Johnson, H.A. DDT in the biosphere: where does it go? Science 174: 1101 (1971).

306. Yadav, D.V.; Agarwal, H.C., and Pillai, M.K.K. Uptake, metabolism and excretion of DDT by the fresh water snail, Vivipara heliciformis. Bull.

Environ. Contam. Toxicol. 21: 300 (1978).

307. Yadav, D.V.; Mittal, P.K.; Agarwal, H.C., and Pillai, M.K.K. Organochlorine insecticide residues in soil and earth worms in the Delhi area, India. Aug-Oct.,1974. Pestic. Monit. J. 15:80(1981).

*308. Yoder, J.M.; Watson, M., and Benson, W.W. Lymphocyte chromosome analysis of agricultural workers during extensive occupational exposure to pesticides. Mutat. Res. 21: 335(1973).

*309. Young, D.R. and Heesen, T.C. Marine bird deaths at Los Angeles Zoo Coast. Water Res. Proj. Annu. Rep. pp.193(1977).

*310. Zeine-El-Dine, K. The insecticide DDT. J. Egypt Med. Assoc. 29:38(1946).

311. Zepp, R.G.; Wolff, N.L.; Azarge, L.V.; Cox, R.H., and Pape, C.W. Photochemical transformation of the DDT and methoxychlor degradation products, DDE and DMDE by sunlight. Arch. Environ. Contam. Toxicol. 6: 305(1977).

312. Zinck, M.E. and Addison, R.F. The effect of temperature on the rate of conversion of p,p'-DDT in brook trout, Salvelinus fontanalis. Can. J. Biochem. 53: 636(1975).

EXPLANATIONS

Table 4 & page 65

Since tissue samples contain high amount of lipids, the removal of lipids before analysis of DDT was essential. This step was omitted in soil samples as the lipid contents of soil were virtually negligible. Because of this additional step it is very likely that some DDT was lost during the cleaning procedure. Thus the recovery of DDT residues and its metabolites from tissues were lower than those observed in the case of soil samples.

Fig 8 & page 76

Higher DDT levels in males than females have been reported by Griffith & Blanke (109) in resdidents of Virginia. In India also, Agarwal et al (7) reported higher blood DDT residues in males as compared to females in Delhi area. The sexual difference in residue levels may be due to excretion of these chemicals in mother's milk and loss during menses in females. Morevoer, females have more fat than males.

In the present study residues were compared in male and female subjects and while comparing sexual differences dietetic habits were not taken into consideration. Similarly when DDT storage in relation to dietetic habits was studied, sexual differences were not taken into account.

Body weight and height were omitted because of the large variations in population under study such as different age groups, dietetic habits etc. Although body weight and height of these subjects are important parameters, our emphasis was more on the interorgan and intercellular distribution of pesticide residues rather than that on the whole body system. The quantum of the data was so large that it would have been rather difficult to correlate the findings properly.

Obesity determination was not our objective, therefore the need for the same was not considered essential.