

A STUDY OF THE IMPACT OF LEAD IN THE ENVIRONMENT
ON SCHOOL CHILDREN IN THE CAPE TOWN AREA

by

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ABSTRACT

The present study was designed to provide information on lead absorption amongst schoolchildren in Cape Town. The primary aim of the study was to determine the degree of lead absorption in part of the community. Further secondary and tertiary aims were to briefly investigate possible sources of lead exposure in children identified with increased lead levels and to determine whether any evidence of behaviour disorder was apparent.

The evidence presented in the study suggests that a significant proportion of the population, primarily children living in urban-industrial areas, may have increased lead levels and may be suffering certain metabolic and behavioural impairments due to lead.

It is suggested that prudent precautions be taken to limit exposure to lead in the environment.

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INTRODUCTION

1.1 HUMAN EXPOSURE TO LEAD

1.1.1 Introduction

Lead has been used since antiquity (1) and its effects were recognized before the Christian era (2,3). There is nevertheless still controversy as to how serious a health hazard lead is. There is no uncertainty as to the health effects of lead at high levels of exposure to the metal, but there is considerable debate as to the health effects of relatively low levels of lead exposure.

It is evident that people living in industrialized countries, exposed to low levels of lead in the environment, may have significantly higher blood lead concentrations than people living in remote areas of the world. For instance, it has been shown that children living in remote areas have blood lead concentrations between three and five ug/dl (4), whereas blood lead levels of adults and children living in industrialized countries are generally in the range 10-25 ug/dl (5). Furthermore, it has been estimated that lead levels of Americans are about 500 times greater than those of prehistoric peoples (6).

This is of particular concern as there is considerable

uncertainty as to what constitutes a safe blood lead level (7). Children are more susceptible to lead toxicity than adults and even slightly increased exposure may have a serious impact on health (8). For instance, it has been demonstrated that adverse metabolic effects and intellectual and behavioural impairments may occur in children at lead concentrations below 30-40 ugPb/dl blood (9,10,11,12). This has become a subject of much controversy in the scientific literature (13,14).

It is also a matter of much debate as to which source(s) of lead in the environment is the most important source of human exposure (15,16). Among the many sources of lead, particular concern has been expressed about the extent to which petrol-derived lead, the major contributor to lead in the atmosphere (17), is potentially hazardous to health (18,19,20).

The current controversy about lead thus centres mainly on two issues:

1. Can lead levels in the environment be considered safe?
2. Which source(s) of lead in the environment is the most important source of human exposure?

1.1.2 Lead Production and Usage

Lead is produced from ores and occurs in a variety of minerals, the most important being Galena (PbS) (21). It

is found mostly in deposits associated with other minerals, particularly those containing zinc, but the proportion of different metals such as copper, silver and zinc may differ in the ores of various countries (5).

Evidence of the production and usage of lead pre-dates recorded history and the use of primitive furnaces to extract lead from its ores may have been the first such smelting process used by humans (22). One of the earliest references to lead is found in Egyptian hieroglyphics of about 1500 BC (23) but it is thought that it was used for glazing pottery by the Egyptians as long ago as 7000-5000 BC (22). In the old Testament there are references to lead as an item of trade with the Phoenicians and it was mined extensively by the Greeks and Romans. Hadrian's Wall is thought to have been built partly to protect the mines in Northumberland and Cumberland, which were used by the Romans at that time (23).

Roman lead technology was impressive and it was used largely to line aqueducts, water mains and water reservoirs (used to collect water from lead lined roofs) (23) and earthenware containers (for storing olive oil, grape syrup and preserved fruits (2)).

The quest for silver was the principal stimulus for lead production in early times and it was only during the last century that lead mined and smelted for itself constituted

a significant portion of total lead production (6). Changes in world lead production over time are shown in Figure 1.

Lead has a considerable number of uses in present technological society. It has been estimated that 35% of total world supply comes from secondary sources, of which lead-acid batteries account for 70-80% (5). Appendix 1 lists the principal uses of lead today. The main properties of lead, which account for its versatility, are listed in Appendix 2.

1.1.3 Sources of Exposure

1.1.3.1 GENERAL SOURCES

The mining, smelting and refining of lead, as well as the production and use of lead-based products, gives rise to the release of lead into the environment (24). Lead is present in the air, soil, dust, food and water (15), and most people, to a greater or lesser degree, are exposed to lead.

Air

Lead is routinely emitted into the atmosphere from the burning of lead alkyls in petrol and, to a much lesser

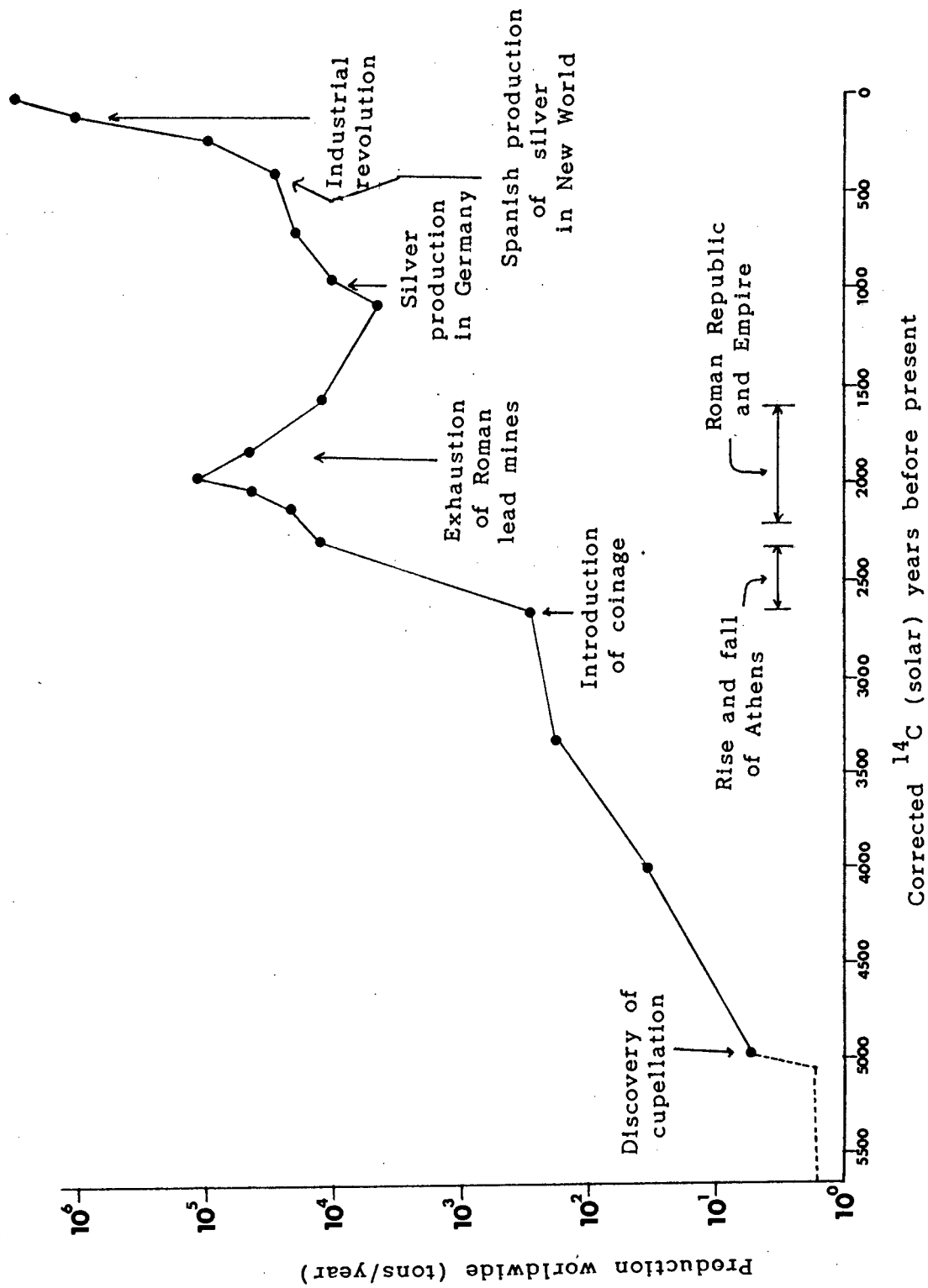


FIG 1. WORLD LEAD PRODUCTION DURING PAST 5500 YEARS
(after Settle and Patterson, 1980)

extent, from certain industrial processes such as coal combustion, lead smelting and iron and steel production (25). The relative contributions of various sources to the global atmospheric lead balance are given in Appendix 3.

Due to the fact that lead aerosols are globally distributed, it is difficult to define natural concentrations of lead in the atmosphere (6). Lead levels in areas most remote from civilization have been found to be in the order of $.0001 - .001 \text{ ug/m}^3$ (5), whereas it has been estimated from geochemical data that the concentration of lead in air of natural origin is about $.0006 \text{ ug/m}^3$ (6). Atmospheric lead concentrations in the "cleanest" regions of North America are thought to be about 100 - 100 000 times higher than ambient concentrations believe to have existed 10 000 years ago (26).

Atmospheric lead concentrations have generally been found to be highest in urban and industrial areas and the highest lead concentrations have been found in densely populated urban areas, particularly over streets with heavy traffic densities (5). High air lead concentrations have also been found in the vicinity of certain industrial sources, such as lead smelters (8). Atmospheric lead concentrations measured in various different parts of the world are listed in Appendix 4.

Soil and Dust

Lead aerosols accumulate in soil and dust (22). The concentration of lead in soil and dust in urban and industrial areas has been found to be higher than in rural areas (22,27,28) and correlations between lead levels in dust, soil and traffic densities have been demonstrated (27,29). The lead content of various soils and street dusts are given in Appendices 5 and 6.

Food

There is considerable controversy over the extent to which airborne lead can contaminate food. According to the Conservation Society in the United Kingdom (15), airborne lead is the chief source of lead in food, both through direct contamination of crops and through entry into the food chain. Isotopic studies have shown that elevated levels of petrol-derived lead may be present in soils up to 50 kilometres from a source, in a form readily available for uptake by plants (30).

The Lawther report (31) maintains that even with heavily contaminated produce from the vicinity of heavily travelled roads or factories, most of the lead is removed in food preparation.

1.1.3.2 LOCALISED SOURCES

Exposure to lead may also be more localised - for instance, lead plumbing may contaminate domestic water supplies (32) or lead paint may contaminate dust and soil or be directly ingested by children (33,34). Workers may also be exposed to lead in certain industries and occupations (35).

Certain food products may be contaminated by the use of lead solder in tins (6,36), or lead glazes in pottery (37). Car batteries used as fuel (38), brightly coloured magazine pages, painted food wrappers (2) and certain hair products and cosmetics (31) may all be potential sources of exposure in particular circumstances.

1.1.4 Uptake of Lead Into the Body

Lead is absorbed into the body via ingestion and inhalation and a very small amount is absorbed through the skin (this is of importance in the case of organic lead compounds only) (39).

1.1.4.1 INHALATION

The uptake of particulate lead is a function of the particle size and solubility of the aerosol inhaled (39). The fractional deposition of lead in the lungs depends mainly on the particle size of the aerosols inhaled, whilst the amount absorbed into the bloodstream depends on the chemical

composition and solubility of the aerosols, as well as the site of deposition (40). The solubility of lead aerosols increases as particle size decreases (39).

The majority of lead aerosols to which the general population is exposed are in sub-micrometer particles and are therefore easily absorbed into the bloodstream (25). Metabolic balance studies have indicated that approximately 30-40% of ambient air inhaled is absorbed (23,40).

These estimates are very approximate and there are many difficulties in predicting the percentage uptake of inhaled lead in the general population. Air lead concentrations vary in time and place and most people are exposed to a range of different concentrations. Also, the lead aerosols inhaled by the general population are not well enough characterised to accurately predict absorption (41).

1.1.4.2 INGESTION

Metabolic balance studies have indicated that about 8-10 percent of ingested lead is absorbed from the gut in adults (23,40) and a much higher percentage, approximately 40-50%, is absorbed in children (42).

Many factors can affect the rate of absorption, and the influence of certain dietary components has been extensively studied. Elements such as calcium, iron, zinc, copper

have been shown to diminish lead absorption (13) but absorption is increased with a high vitamin D concentration, a high fat diet (43) and in the absence of food (44).

1.1.5 Distribution of Lead in the Body

After absorption, lead is transported by the blood to the soft tissues. The highest concentrations are found in the liver and kidneys, but some goes to other organs such as the brain, lungs, spleen and heart (23).

Deposition of lead takes place in the bone, where more than 90% of the body lead is found as relatively insoluble phosphates (43). In certain circumstances, lead may be remobilized from the bone after initial absorption (20). In children, considerably less lead is deposited in bone and a greater proportion is deposited in a mobile form in the soft tissues (38). Lead can cross the placental barrier and concentrations in the foetus may increase during pregnancy (13).

1.1.6 Effects of Lead

There is no known biological function for lead and any lead absorbed by humans is potentially toxic (3).

Lead poisoning is essentially chronic, as the accumulation of a significant body burden occurs over a period of time.

At low exposure levels it may take months or even years before clinical symptoms appear and certain biochemical effects and neuropsychological effects may be present long before clinical symptoms occur. At higher levels of exposure, accumulation of toxic levels may occur within weeks and damage to tissues is caused (8). Effects at high and low levels of exposure are commonly referred to as clinical and sub-clinical effects.

1.1.6.1 CLINICAL EFFECTS

At high levels of lead absorption (between 40 and 80 ugPb/dl blood) lead causes damage to the blood system, resulting in anaemia. It also affects the kidney, liver and the central nervous system (23). At very high blood lead concentrations (above 80 ugPb/dl blood) it can cause acute brain damage, leading to encephalopathy and may eventually result in death (2).

1.1.6.2 SUB-CLINICAL EFFECTS

Effects at low levels of lead absorption (below 40 ugPb/dl blood) include intellectual impairment, behaviour disorders and certain biochemical disorders (9,10,11,12). Biochemical effects form the basis upon which various diagnostic tests used in screening have developed (45). These will be briefly discussed.

At levels of lead absorption between 15 and 20 ugPb/dl blood lead inhibits heme synthesis (8,24,45). The two most important points of interference in heme synthesis are the inhibition of two enzymes, Ferrochelatase and ALA-D (Delta Aminolaevulinic Acid Dehydratase). The inhibition of Ferrochelatase, the enzyme responsible for the insertion of iron into Zinc Protoporphyrin, results in an increase of Zinc Protoporphyrin in the red blood cells (46). Inhibition of the enzyme ALA-D results in an increase of ALA in the blood and urine. Measurement of the inhibition of ALA-D, the accumulation of ALA in the blood and urine, and the accumulation of Zinc Protoporphyrin in the red blood cells, have all been used as screening tools (45).

1.1.7 Contribution of Sources to the Body Burden

1.1.7.1 SOURCES OF HIGH LEVEL EXPOSURE

There is a long history of human exposure to high levels of lead normally associated with lead poisoning. Some of the more common sources of lead poisoning will be briefly discussed.

Food and Drink: The most common cause of lead poisoning has been attributed to high levels of lead in food and drink, due to cooking and storing food in lead-lined or

lead-glazed earthenware pots and the supply of water through lead pipes (2,8). Some historians believe that one of the factors in the fall of the Roman Empire may have been a decrease in reproductive capacity and "madness" of the ruling class due to poisoning from lead contaminated food and water (1,3).

Still today, in some parts of the world, many houses have lead plumbing. A national survey in Great Britain revealed that in Glasgow, Scotland, where a large percentage of houses have lead piping, unacceptably high levels of lead were found in household water samples and in people (32,47).

Occupational Exposure: Occupational lead poisoning was first reported among lead workers by Pliny in the first century AD (3). With increasing industrialization and use of lead in the 18th and 19th centuries, lead poisoning became an established occupational disease. Improvements in industrial methods and in industrial health standards have helped reduce the occurrence of occupational lead poisoning today, but in many work environments lead poisoning still occurs. In Great Britain, lead poisoning has been cited as the most common notifiable industrial disease (35,38).

Paint: In children, the major cause of lead poisoning over the last century, and still today, has been attributed to

the ingestion of flaking lead-based paint chips (38). This is a common occurrence amongst young children of pre-school age, with a tendency to pica (an appetite for non-food items) who live in old, deteriorating houses with peeling lead-based paint. Daily ingestion of a few small paint chips can cause lead poisoning within a few months (37).

Flaking paint may also contaminate dust and soil, which may be ingested by children (34).

In recent years, mass screening programs initiated in countries such as America, have helped reduce the incidence of lead poisoning in children (38). Of more concern today are the sources of low level lead exposure to which people are routinely submitted.

1.1.7.2 SOURCES OF LOW LEVEL EXPOSURE

Low level sources of lead to which people are exposed and which may add to the total body lead burden, include lead concentrations in the air, water, soil, dust and food (15). In particular circumstances (such as ingestion of lead contaminated soil and dust by children) exposure to these sources can lead to lead poisoning (33).

It is generally accepted that blood lead levels of adults and children living in urban-industrial areas are higher than in people living in rural areas and that people

exposed to high traffic densities or to certain industrial sources of lead have higher blood lead levels than people not so exposed. These associations have been demonstrated in numerous epidemiological studies (5,48,49,50,51,52,53) and indicate that airborne lead may be an important source of exposure in these circumstances.

These data, however, must be regarded as qualitative indications of source contributions and cannot be accepted as quantitatively reliable. Recent epidemiological data which have examined multiple sources and mechanisms of lead exposure (such as lead aerosols and lead contaminated soil and dust) have indicated that the relationship between blood lead levels and various sources is likely to be multifactorial; the exact relationship depending on many difference factors and circumstances (54,55,56,57).

Attempts which have been made to quantify sources must be treated with caution. The Lawther report has estimated that in adults, not especially exposed to lead (with blood lead levels between 10 and 20 ug/dl), 45-90% of the body burden is derived from food, 0-45% from water, and 10-20% from air (31). The United Kingdom Conservation Society, on the other hand, has estimated that 32-69% of the body burden is derived from air (15).

At present, very little is known of the dynamics of multiple exposure and further study on the sources and mechanisms

of exposure is needed, in order that the relative importance of each factor may be accurately assessed.

1.1.8 Susceptibility of Children

Children are generally regarded as being particularly susceptible to clinical and sub-clinical lead poisoning (58). There is evidence that children are more exposed to lead, that they absorb a greater proportion of lead and that they are particularly susceptible to its potential toxic effects.

Several sources of lead exposure may be present in the child's environment. There may be flaking chips of lead-based paint or lead-contaminated soil and dust. These sources may be of particular importance in young children, between the ages of 1 and 3 years, when normal hand-to-mouth activities are most common and when pica is most likely to occur (59). In addition, children may be exposed to lead from dust brought into the house by lead workers (60,61). Children may also be exposed to higher levels of lead in the air, as the lead content in ambient air is greater at the height at which young children breathe (38).

Relative to their body size, infants and young children have a higher alimentary intake and respiratory ventilation than adults (8) and they absorb a much higher proportion

of lead from their diet. Certain nutritional deficiencies which are known to potentiate lead toxicity are also more likely to occur in children (59).

Not only do children have a greater intake and uptake of lead from the environment than adults, they are also more susceptible to the toxic effects of lead. There is a larger proportion of mobile lead in the soft tissues of children (which is potentially more toxic), their metabolic pathways are incompletely developed and the developing brain of a young child is particularly vulnerable to lead (59).

Childhood lead poisoning constitutes a serious environmental health problem. In the United States of America, for instance, each year tens of thousands of children are found to have elevated blood lead levels (62,63). According to the United States Center for Disease Control, no childhood disease approaches lead poisoning in the breadth of its impact (62).

1.1.8.1 SOCIO-ECONOMIC STATUS

It is generally accepted that lead poisoning and elevated blood lead levels are more prevalent among children of low socio-economic status (38,62,63,64,65).

Poor children are more likely to reside in badly maintained

old houses with peeling lead-based paint, or with lead plumbing. They may also be exposed to greater amounts of dust and dirt in and around their houses and may be more exposed to sources of lead from traffic and industry. Also, nutritional deficiencies known to potentiate lead toxicity are more likely to occur in poor children (13). Children of low socio-economic status who are most likely to be affected by lead are also the least equipped to deal with the problem (62).

1.1.9 Screening for Lead

Lead poisoning is a preventable disease and is one of the few preventable diseases which have been neglected for such a long time (38). In the United States of America, over the past decade, mass screening programs have been initiated. These are aimed at detecting children at risk, before clinical symptoms occur, and have helped to reduce the incidence of lead poisoning among children (63).

At the screening level, diagnosis of lead poisoning and elevated blood lead levels is usually based on metabolic indices of lead exposure (45). Measurement of the concentration of lead in blood is the traditional indicator of current lead absorption, but it gives no indication of a metabolic effect due to lead (5). Blood lead measurements are also expensive, require sophisticated equipment and are subject to considerable inter-laboratory variation

(66). For these reasons, metabolic indices of lead exposure are widely used in screening.

Diagnostic tests indicative of metabolic effects of lead are generally based on derangements in heme metabolism.

1.1.9.1 THE ZINC PROTOPORPHYRIN (ZPP) SCREENING TEST

The accumulation of ZPP is regarded as being the first sign that physiological impairment due to lead has occurred (24). Consequently, the United States Centre for Disease Control has recommended that the measurement of ZPP be used as a primary screening test for lead absorption (68).

The test requires only a drop of capillary blood, and is rapid and inexpensive, requiring no laboratory facilities.

Use of the hematofluorometer, a portable field instrument for measuring ZPP (69), enables workers to obtain results instantaneously in the field. Measurement of ZPP has thus become an important tool in the screening of occupationally exposed populations and asymptomatic children (68).

The ZPP concentration is representative of exposure to lead over the preceding 3-4 months. The elevation of ZPP, however, is not specific for lead poisoning, and may also reflect other abnormalities of heme synthesis such as iron deficiency. For this reason, elevated ZPP levels require subsequent verification of blood lead measurements (46).

1.2 INTRODUCTION TO THE STUDY

1.2.1 Motivation

There is little information on lead absorption amongst the general public, or amongst children, in South Africa (70, 71). It is evident, however, that people may be exposed to significant levels of lead in the environment. Concern has recently been expressed regarding the impact of lead on sections of the South African population (70,71,72, 73,74,75,76,77,78).

Air pollution monitoring has revealed that South African cities, and Cape Town in particular, have lead levels comparable to those found in large urban and industrial centres of the world. This has given rise to concern relative to international safety standards (76).

Concern has also been expressed regarding the potential hazard of lead-based paints to young children. Such paints were in general use until recently and are still used to a lesser extent today. A recent country-wide survey revealed that a large percentage of houses had paint-work containing significant amounts of lead (70). There are no regulations or standards restricting the use of lead-based paints in South Africa (79).

Up until the early part of this century, lead plumbing was

in general use in South Africa, and many cases of lead poisoning were reported. Houses with lead plumbing are still found today; this was revealed by the recent demolition of District Six in Cape Town (73).

Lead poisoning is a notifiable disease in South Africa, yet over the past ten years only one case amongst children was reported (80). In Cape Town there were no reported cases during this period amongst children or adults (81). A few cases of lead poisoning in children (from burning battery casings for fuel) have been reported in the literature (72).

South Africa may be in the same position as New York in 1950, when, due to lack of awareness of the problem, only one case of lead poisoning was reported (70). Lead poisoning is now recognised as a major childhood health problem in America (62).

The present study is designed to provide information on lead absorption amongst children in Cape Town.

1.2.2 Approach

The study is designed to determine the impact of lead in the environment on schoolchildren in greater Cape Town. It is limited to 'coloured' children for reasons relating to socio-economic status.

The **primary** aim of the study is:

- to determine the degree of lead absorption amongst schoolchildren in part of the Cape Town community.

Further **secondary** and **tertiary** aims of the study are:

- to investigate possible contributory sources of lead exposure in children identified with increased blood lead levels;
- to briefly investigate whether any evidence of behaviour disorder is apparent in children identified with increased blood lead levels.

LEAD ABSORPTION IN THE STUDY POPULATION

2.1 INTRODUCTION

The primary aim of this study was to determine the degree of lead absorption in part of the Cape Town community.

The study population was defined as "coloured" sub A pupils attending schools believed to be exposed to a source of lead, in the Wynberg, Cape Town and Bellville Magisterial districts.

2.2 SAMPLE DESCRIPTION

2.2.1 Selection of Schools

In order to ensure exposure to lead and to standardise sampling over a large area of Cape Town, it was decided to sample schools situated on major through roads in Cape Town.

Multistage sampling was used to select a sample from the study population. From a list of primary schools obtained from the Cape Provincial Department of Education, schools situated on through roads (as indicated on a map of Cape Town) were identified. Out of a total of 262 schools listed in the Wynberg, Cape Town and Bellville magisterial

districts, 26 schools situated on through roads were identified. One school in each suburb was selected and 13 schools were chosen for study. In order to improve the distribution of data points in the immediate Cape Town urban/industrial complex, an additional five schools (not situated on through roads) were selected, as well as one school in a semi-rural area. In all, 19 schools were finally selected for study. The situation of schools is given in Table 1 and Figure 2.

2.2.2 Selection of Pupils

Permission to undertake the study during school hours was obtained from all school principals. Informed, written consent was obtained from parents of all children sampled. The parental response rate was 97%.

With the exception of one school (school number 15), all sub A pupils with parental consent, present at school on the day of the study, were sampled. In all, 917 sub A pupils were selected for study. In addition to these children, at schools with fewer than 40 sub A pupils, it was decided to sample children from other classes. These children, however, were excluded from the formal statistical analysis of results (i.e. significance testing), as were children from a correctional school for boys of all ages (school number 10) which was also included in the sample.

TABLE 1: SITUATION OF SCHOOLS

School No.	Suburb	Recruitment Area	Prox. to through road
1	Elsies River	U/I	+
2	Woodstock	U/I	+
3	Claremont	R	+
4	Constantia	R	+
5	Salt River	U/I	+
6	Bellville South	U/I	+
7	Cape Town	U/I	-
8	Malay Quarter	U/I	-
9	Central Cape Town	U/I	-
10	Wynberg	R	+
11	Hanover Park	R	+
12	Athlone	R	+
13	Retreat	R	+
14	Steenberg	R	+
15	Lotus River	R	+
16	Salt River	U/I	-
17	Maitland	U/I	-
18	Mitchells Plain	R	+
19	Hout Bay	R	+

U/I : Mixed residential/urban-industrial area

R : Residential area

+ : On through road

- : Away from through road

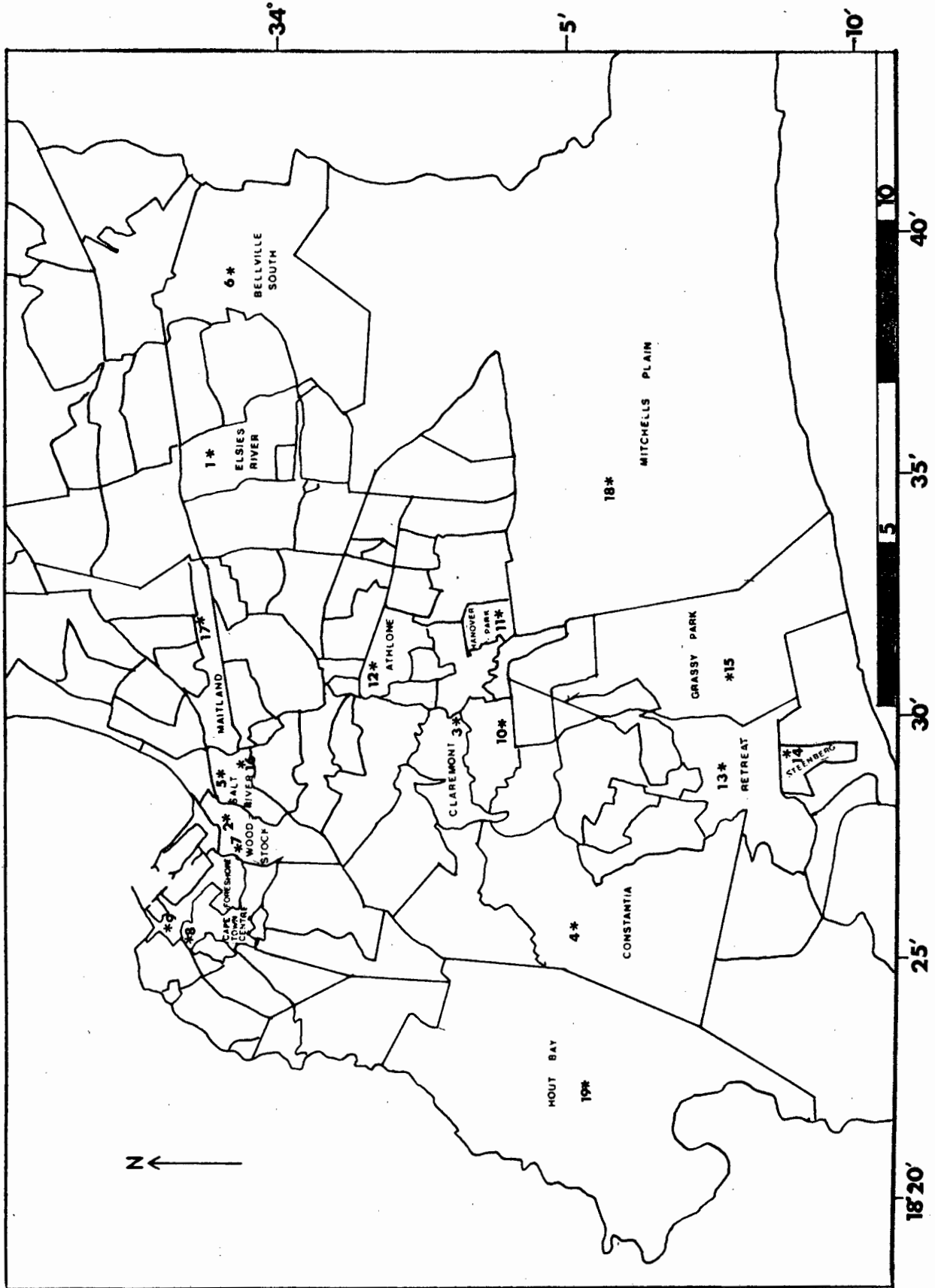


FIG 2. SITUATION OF SCHOOLS

Altogether 1 234 pupils were selected for study, of whom 74% were sub A pupils. The distribution of children by school, class and sex is given in Table 2.

2.3 METHODS AND TECHNIQUES

The Zinc Protoporphyrin (ZPP) screening test was administered to all children. Those children identified with elevated ZPP levels received confirmatory blood lead and other hematological tests. All children were tested between the months of September and December.

2.3.1 ZPP Tests

A drop of blood obtained from each child by finger puncture was analysed for ZPP concentration.

Apparatus: An hematofluorometer (AVIV model) was used to perform ZPP analyses. The blood sample is deposited in the centre of a glass cover slip which is then inserted into the apparatus. As it is inserted, an exciting light focuses first on a blank, then on a calibrating sample and finally on the blood sample, where it is completely absorbed within a few microns. The fluorescent intensity is independent of the thickness of the blood sample (82). The emitted light is then filtered and detected by a photo-multiplier. The result is expressed in ug ZPP/g Hb

TABLE 2. DISTRIBUTION OF CHILDREN BY SCHOOL,
CLASS AND SEX

School	Class	Males	Females	Total
1	Sub A	24	21	45
2	Sub A	28	18	46
3	Sub A	24	31	55
4	Sub A	7	10	17
	Sub B	8	10	18
	Std 1	9	6	15
	Correctional Class	3	5	8
	Total	27	31	58
5	Sub A	30	32	62
6	Sub A	24	28	52
7	Sub A	22	15	37
	Sub B	14	9	23
	Total	36	24	60
8	Sub A	54	45	99
9	Sub A	10	15	25
	Sub B	10	12	22
	Std 1	16	10	26
	Std 2	12	12	24
	Total	48	49	97
10	Total	30	-	30

(continued)

TABLE 2

School	Class	Males	Females	Total
11	Sub A	33	27	60
	Sub B	20	28	48
	Total	53	55	108
12	Sub A	59	38	97
13	Sub A	36	30	66
14	Sub A	17	15	32
	Sub B	16	24	40
	Total	33	39	72
15	Sub A	7	1	8
	Sub B	24	19	43
	Total	31	20	51
16	Sub A	16	24	40
17	Sub A	26	23	49
18	Sub A	51	44	95
19	Sub A	18	15	33
	Sub B	9	10	19
	Total	27	25	52
GRAND TOTAL		657	577	1234

and the accuracy is within 5-10%.

2.3.2 Blood Lead Tests

Approximately 5 mls of venous blood from each child with an elevated ZPP level was obtained by venipuncture into a lead-free heparinised tube. The samples were frozen for 3-4 weeks at a temperature of -20°C until such time as they could be delivered to a laboratory for analysis. The analysis of lead concentration in blood samples was performed using atomic absorption spectrophotometry.

Preparation of Samples: Lead in blood samples and in standards of 10, 20, 50 and 100 ug/100 ml was concentrated by chelation with a solution of ammonium pyrrolidene dithiocarbamate. The lead complex formed was then extracted into approximately 4 mls of a methyl isobutyl ketone (MIBK) solution.

Apparatus: A flame atomic absorption spectrophotometer (Varian Techtron Model 1200) was used to perform blood lead analyses.

The previously prepared MIBK solution was aspirated into an acetylene air flame and the absorbance read at 217 nms.

2.3.3 Full Blood Counts

Full blood counts were performed on venous blood samples obtained from children with elevated ZPP levels. Measurements including haemoglobin and hematocrit levels were determined.

Apparatus: A coulter counter, Model S Plus, was used to perform analyses.

2.4 RESULTS AND DISCUSSION

PART I

2.4.1 ZPP Frequency Distributions

From an examination of the frequency distributions of ZPP within each school, it was evident that they were not normally distributed, but generally positively skewed. For the purpose of comparison, description statistics including the mean, median, range, standard deviation and various percentiles, are used to describe distributions. These results form the basis of the discussion of the ZPP concentrations. Table 3 gives a breakdown of ZPP distributions according to sex for school entrants, non entrants and all pupils, Table 4 gives a breakdown by school and sex for entrants only and Appendix 7 gives a breakdown by

TABLE 3. ZPP DISTRIBUTIONS (ug/gHb)
Breakdown by sex for school
entrants, non-entrants and
all pupils

	School entrants			Non School entrants			Total		
	Freq.	Mean	S.d. Median ≥ 5	Freq.	Mean	S.d. Median ≥ 5	Freq.	Mean	S.d. Median ≥ 5
Males	485	3,3	1,7 8,0	172	3,3	1,0 3 7,1	657	3,3	1,6 3 7,7
Females	430	3,1	1,1 5,4	147	3,0	0,7 2,9 2,1	577	3,1	1,0 2,8 4,4
TOTAL	915	3,2	1,5 6,9	319	3,2	0,9 3 4,7	1234	3,2	1,3 2,9 6,3

TABLE 4 (continued)

School	Females				Males				Total						
	Freq.	Mean	S.d.	Median % \geq 5	Freq.	Mean	S.d.	Median % \geq 5	Freq.	Mean	S.d.	Median	% \geq 5		
11	27	2,7	0,4	2,7	0	33	2,9	0,5	3	0	60	2,8	0,5	2,7	0
12	38	2,9	0,5	2,8	0	59	3,5	3,2	3	5,1	97	3,2	2,6	2,9	3,1
13	30	3,0	1,2	2,85	6,7	36	3,2	1,7	2,7	5,5	66	3,1	1,5	2,75	6,1
14	15	3,2	2,7	2,4	6,7	17	2,7	0,6	2,6	0	32	3,0	1,9	2,5	3,0
15	1	3,7	-	3,7	0	7	2,9	0,4	3,0	0	8	3,0	0,4	3,05	0
16	24	2,7	0,4	2,5	0	16	2,7	0,5	2,65	0	40	2,7	0,5	2,6	0
17	23	2,4	0,5	2,3	0	26	3,0	1,6	2,5	7,7	49	2,8	1,2	2,4	4,1
18	43	2,5	0,5	2,5	0	50	2,6	0,7	2,45	2	93	2,6	0,6	2,5	1,1
19	14	2,5	0,5	2,35	0	18	2,9	1,4	2,4	11,1	32	2,7	1,1	2,4	6,3

sex, school and class for all children tested. Further details of ZPP distributions are given in Figures 3 and 4.

Discussion:

It is instructive to compare the results obtained in this study with those reported by other workers.

The range of average ZPP levels reported in the literature for non-occupationally exposed populations varies extensively. One of the reasons for this may be due to the fact that methods for detecting increases in protoporphyrins are not standardised. For instance, erythrocyte protoporphyrin may be measured by fluorometry after extraction from the red cells, or by direct measurement of its fluorescence in intact red cells, with a hematofluorometer. The hematofluorometer measures only Zinc Protoporphyrin, while the acid extraction techniques measure Zinc Protoporphyrin plus Protoporphyrin Free Base (Free Erythrocyte Protoporphyrin (45,67,68). There are also different ways of expressing ZPP based on different calibration criteria and expressed in different units (83). The lack of standardisation of methods and units used makes it difficult to compare reports by different workers. This is further complicated by the fact that reports published generally refer to occupationally exposed populations and very little data exists for children. Where such data are available, they are usually from studies of selected "high-risk" populations.

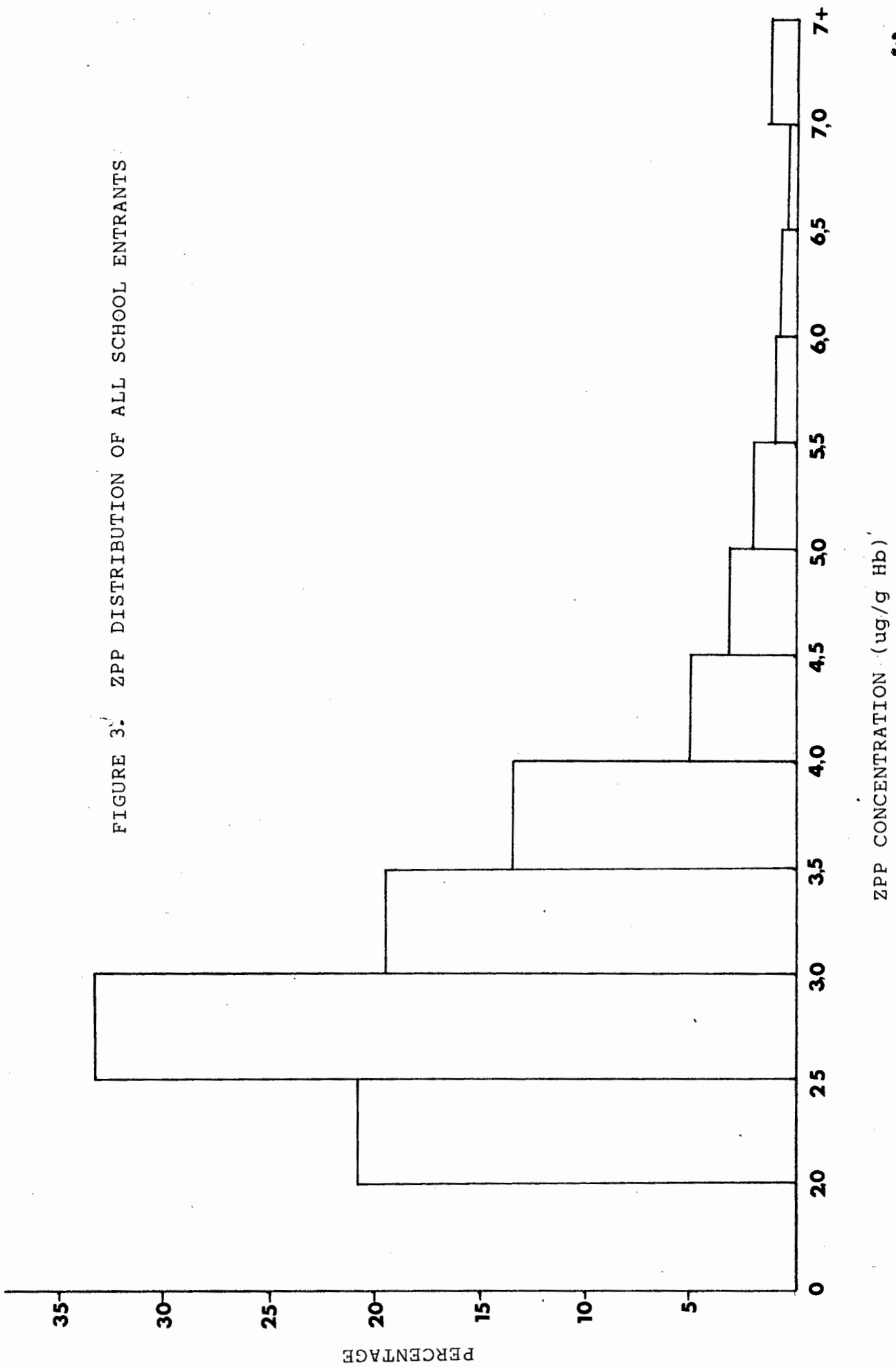
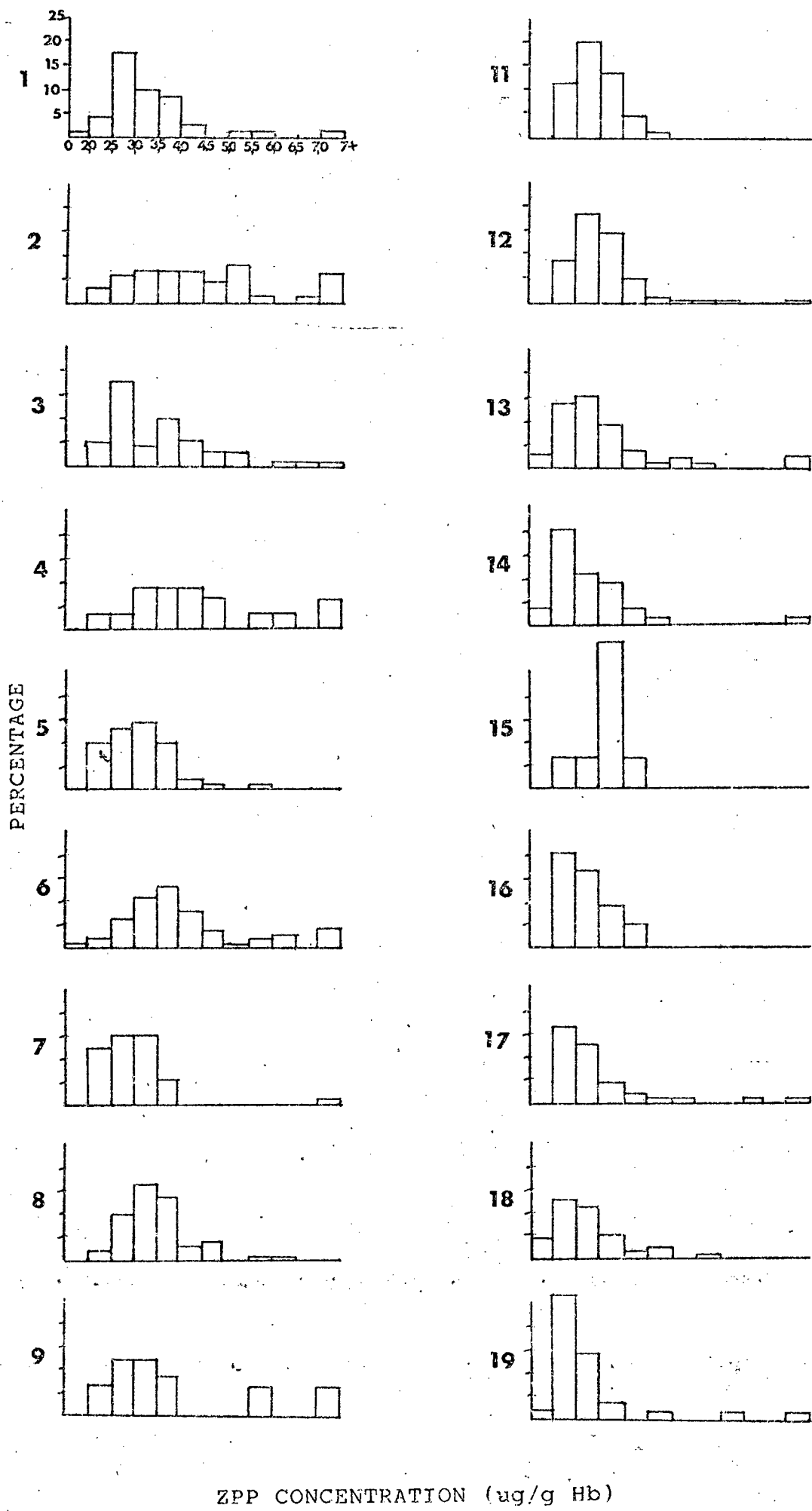


FIGURE 3. ZPP DISTRIBUTION OF ALL SCHOOL ENTRANTS

FIG 4. ZPP DISTRIBUTIONS (ug/gHb) OF CHILDREN BY SCHOOL.



ZPP CONCENTRATION (ug/g Hb)

Nevertheless, there are a few recently published studies which are of interest. In a study in Yugoslavia (84) it was found that the mean ZPP level for children aged 5-10 years was 1.5 ug ZPP/gm Hb. This is about half the mean ZPP level for this study population, which was 3.2 ug ZPP/g Hb. The Yugoslavian study, however, was based on a very small sample, involving only 20 individuals. In a study by Grandjean (85) of 1 295 non-occupationally exposed adult workers in Denmark, the median ZPP value was found to be 2.8 ug/g Hb with 97% of the population below 4.4 ug ZPP/g Hb. These results are comparable to the results obtained for children in the present study - the median for this population (males and females together) was 2.9, with 90,4% of the populaiton below 4.4 ug ZPP/g Hb.

Little is known regarding the health significance of ZPP levels in the range reported in these studies. Attempts to correlate ZPP values with blood lead concentrations have not been able to demonstrate consistent dose-response relationships and appear to differ for various population groups studied. In a study by Zielhuis and Wibowo (83) these workers stated that a ZPP reading of 2.5 - 3 ug ZPP/g Hb (in occupationally exposed workers) is indicative of increased lead exposure and recommended that occupational physicians keep levels below 2.5 ug ZPP/g Hb. This view is not shared by others (85).

For the purpose of this study, in accordance with the revised

1978 standards set for ZPP levels by the United States Center for Disease Control (45,68), levels below 5.0 ug ZPP/g Hb are regarded as being within normal limits. It must be stressed that in light of the considerable ambiguity which exists regarding acceptable standards for safety, although APP levels below 5 ug ZPP Hb may be typical for this study population (corresponding to approximately the 94th percentile) they may not necessarily be safe.

2.4.1.1 SIGNIFICANCE TESTS

There was a small difference in mean ZPP levels between various schools and between the sexes. It was therefore decided to determine whether such differences were statistically significant. As frequency distributions were positively skewed, a log transformation was used to try and achieve normalisation. This was not entirely successful, as was indicated by the skewness and kurtosis indices and semi-interquartile ranges. It was therefore not possible to use parametric tests of significance. As the shape of the distributions were rendered acceptably similar to one another, however, it was decided to use non-parametric tests of significance.

(a) Sex Difference

A median test was performed for sex differences among school entrants within each school. Significant differences

were observed between the sexes for schools 9 and 11 at the 5% level. Differences between the sexes were also found to be significant for all school entrants analysed together (chi.sq. 7.150, d.o.f.1, p.0075).

Although the difference between the median ZPP levels of males and females was found to be statistically significant, the significance in terms of health, at such levels, is uncertain. It is interesting to note that the distribution of ZPP levels was such that 8% of males had ZPP levels equal to or above 5 ug ZPP/g Hb, whereas only 5% of females had ZPP values equal to or above this level. Whether or not this difference reflects a difference in exposure to lead, or a difference in biological response to lead, is not known.

There is some evidence that amongst adults, at comparable degrees of exposure, women exhibit a larger increase in erythrocyte protoporphyrin than do men (86,87). Few studies have tested for differences between the sexes amongst children. Because of the significant difference observed between ZPP values of males and females, it was decided to analyse the sexes separately for differences between the schools.

(b) Variability Among Schools

A median test was performed which indicated highly significant variation among schools for males and females.

(females: chi sq. 105.335 d.o.f. 17, p .0001, males: chi sq. 95.785, d.o.f. 17, p .0001). It was therefore decided to look for differences between schools on and away from through roads.

- Difference between schools on and away from through roads

A median test was performed which did not indicate any significant difference in ZPP between the two groups.

- Difference between schools in residential and mixed residential/urban-industrial areas

Schools were grouped into two broad categories: those in residential areas and those in mixed residential/urban-industrial areas. A median test was performed which indicated highly significant variation in ZPP between the two groups. This held for males and for females respectively. (males: chi sq. 24.038, d.o.f.1, p. 0001. females: chi sq. 14.973, d.o.f.1, p. .0001). The results are given in Table 5.

It was subsequently decided to determine whether there was a difference between schools on and away from through roads within the two categories.

- Difference between schools in residential and mixed residential/urban-industrial areas, according to position of school with respect to through road

In this analysis (a median test was performed) significant

TABLE 5. ZPP DISTRIBUTIONS BY RECRUITMENT AREA

Site	Males					Females				
	Freq.	Mean	S.d.	Median	% ≥ 5	Freq.	Mean	S.d.	Median	% ≥ 5
U/R	234	3,6	1,5	3,25	10,3	221	3,3	1,1	3	7,2
R	251	3,1	1,9	2,8	5,2	209	3,0	1,2	2,7	3,8

TABLE 6. ZPP DISTRIBUTIONS BY RECRUITMENT AREA
W.R.T. THROUGH ROAD

Site	Males				Females			
	Freq.	Mean	S.d.	Median	Freq.	Mean	S.d.	Median
U/R+	106	3,9	1,6	3,45	99	3,5	1,2	3,2
U/R-	128	3,3	1,2	3,1	122	3,1	1,0	2,85
R+	233	3,1	1,9	2,9	195	3,0	1,2	2,7
R-	18	2,9	1,4	2,4	14	2,5	0,5	2,35

U/R+ - Mixed residential/urban-industrial area, on through road

U/R- - Mixed residential/urban industrial area, away from through road

R+ - Residential area on through road

R- - Residential area away from through road.

differences in ZPP among the four groups were found (females: chi sq. 27.961, d.o.f.3, p .0001, males: chi sq. 24.504, d.o.f.2, p .0001) and the average values formed a gradient, as shown in Table 6.

Discussion

It is of great interest that the ZPP levels of children attending schools in mixed residential/urban-industrial areas were significantly higher than children attending schools in residential areas, and that children attending schools on through roads in these areas had significantly higher ZPP levels than children attending other schools.

These differences were found to hold for males and females respectively, with males having slightly higher averages than females. There were almost twice as many children with raised ZPP levels (equal to or above 5 ug/g Hb) in the mixed residential/urban-industrial group as there were in the residential group. Of the males in the respective groups, 9,4% and 5,2% had raised ZPP levels. Of the females in the two groups, 7,2% and 3,8% had raised ZPP levels.

It is also of interest that a significant difference in ZPP levels between schools on and away from through roads was only found when the type of recruitment area was taken into account.

These data suggest that ZPP may be a sensitive indicator of lead exposure. The results indicate that exposure to traffic may be an important source of lead absorption in children, although the nature of the recruitment area in which the children live and attend school may be of more significance in determining exposure than traffic on the street facing the school.

It must be stressed that whilst these results may be of considerable significance, the implications of the results should be evaluated by a controlled study and confirmed by blood lead and other hematological tests. This was not in the scope of the present study.

2.4.2 Detection of Children at Risk

It is important to note that the prime purpose of the screening test was to detect those children potentially at risk - i.e. children identified with elevated ZPP levels who required blood lead tests to confirm increased lead absorption.

It was therefore necessary to designate some ZPP value as a cut-off point - a limiting value below which children were not considered at risk of developing clinical symptoms of lead poisoning.

As has been indicated in Section 2.4.1, there is much uncertainty as to the health significance of ZPP levels which

are not markedly raised. Despite the fact that no base line for normality has been established, it was decided to use a ZPP value approximating the upper limit of normal established for children in the United States of America (45,68), as a cut-off level for the study population. It was felt that the screening test should enable children to achieve the maximum possible benefit from the test, and that the cut-off level should be set at a conservative value which would result in the detection of children in need of medical attention for reasons other than lead intoxication (for instance, iron deficiency). It was therefore decided to accept the occurrence of a large number of false positives against the risk of missing children in need of medical attention. A cut-off level of 5 ug ZPP/g Hb was thus set.

Of a total of 1 234 children who had ZPP tests performed, 76 (6%) had ZPP levels greater than or equal to 5 ug/g Hb. Of these children, 71 had blood lead tests and 62 had full blood counts performed. The results of blood analyses of individual children by sex, class and school are given in Appendix 8 whilst descriptive statistics of the distributions are given in Table 7. Further details of the blood lead distribution are given in Figure 5.

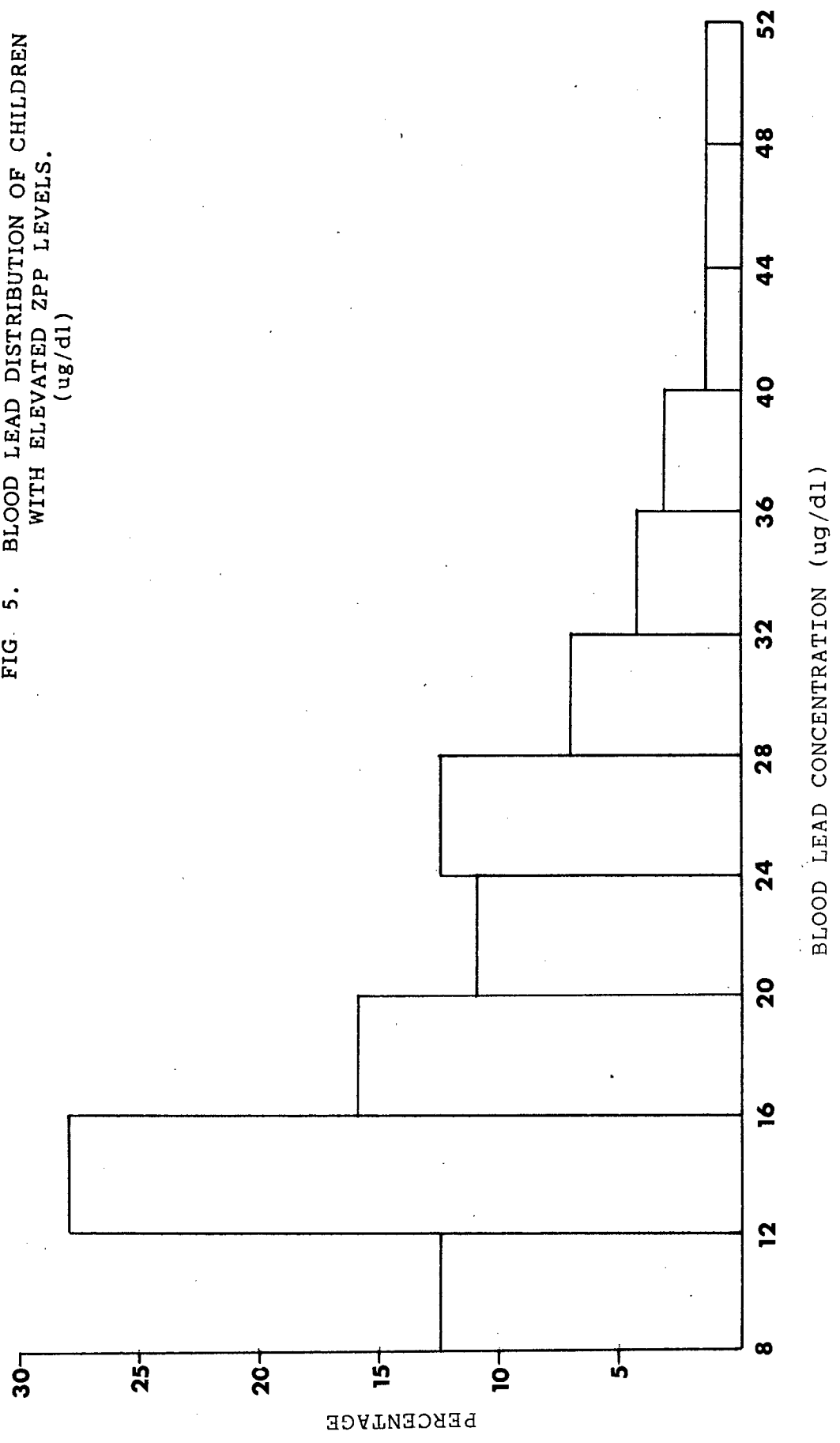
BLOOD LEAD DISTRIBUTIONS

In order to establish a base line for normality (for Cape

TABLE 7. ZPP, BLOOD LEAD AND HAEMOGLOBIN DISTRIBUTIONS (by sex)

	Males			Females			Total		
	Freq.	Mean	S.d. Median	Freq.	Mean	S.d. Median	Freq.	Mean	S.d. Median
ZPP (ug/gHb)	53	7,1	3,3 6,0	23	6,4	1,7 5,9	76	6,9	2,9 5,6
Blood lead (ug/dL)	49	21,1	9,7 18	22	18,5	6,5 17,5	71	20,3	8,9 18
Hb (g/dl)	43	12,3	1,6 12,6	19	12,4	1,9 12,9	62	12,3	1,7 12,7

FIG. 5. BLOOD LEAD DISTRIBUTION OF CHILDREN WITH ELEVATED ZPP LEVELS. (ug/dl)



Town children) with which to compare the blood lead results obtained in this study, it was decided to perform blood lead analyses on a sample of children not thought to be significantly exposed to lead. It was decided that children attending the school in Hout Bay, with a median ZPP level of 2.4 ug/g Hb (see Table 4) would serve as a suitable control group for this purpose.

2.4.2.1 HOUT BAY CASE STUDY

Sample Description: All sub A and sub B pupils, present at school on the day of the study, were sampled. Informed, written consent was obtained from parents of all children. The parental response rate was 100%. Altogether 40 children were sampled, of whom 33 (82%) had previously had ZPP tests performed.

Methods and Techniques: Approximately 5 mls of venous blood were obtained from each child by venipuncture into lead-free heparinised tubes. All samples were obtained during the second week of February and were analysed directly after collection. Blood lead tests and full blood counts were performed on all samples.

Results and Discussion

Descriptive statistics of the haemoglobin and blood lead distributions are given in Table 8. Details of the blood

TABLE 8. HOUT BAY SCHOOLCHILDREN:
DISTRIBUTION OF BLOOD LEAD AND HAEMOGLOBIN
(Breakdown by sex)

	Males			Females			Total		
	Freq.	Mean	S.d. Median	Freq.	Mean	S.d. Median	Freq.	Mean	S.d. Median
Blood lead (ug/dl)	16	10,9	2,8 12	24	11,1	2,8 11	41	11,1	2,7 11
Hb (g/dl)	16	12,4	0,9 12,2	23	12,3	1,1 12,0	39	12,3	1,0 12,1

FIG 6.

BLOOD LEAD DISTRIBUTION OF
HOUT BAY SCHOOL CHILDREN

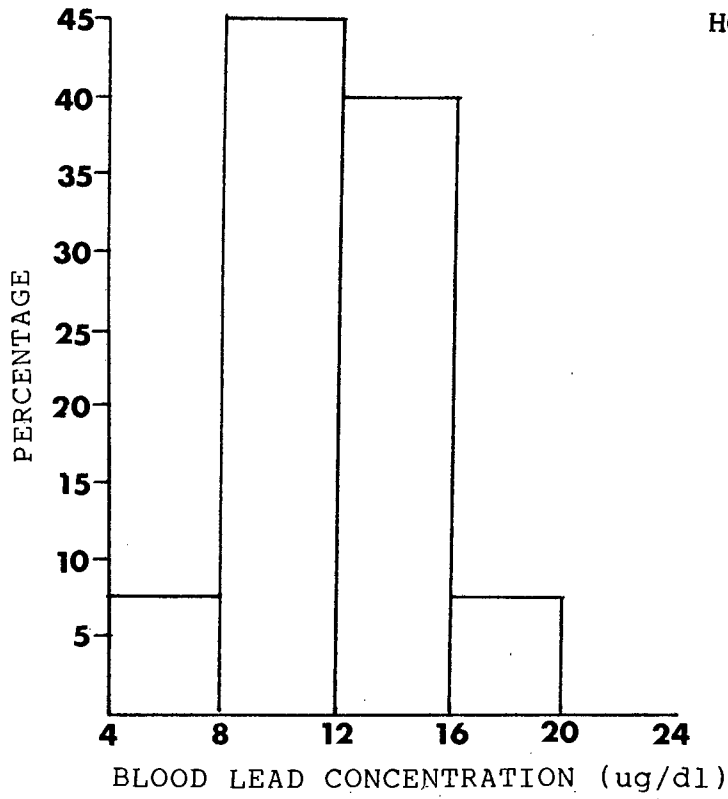
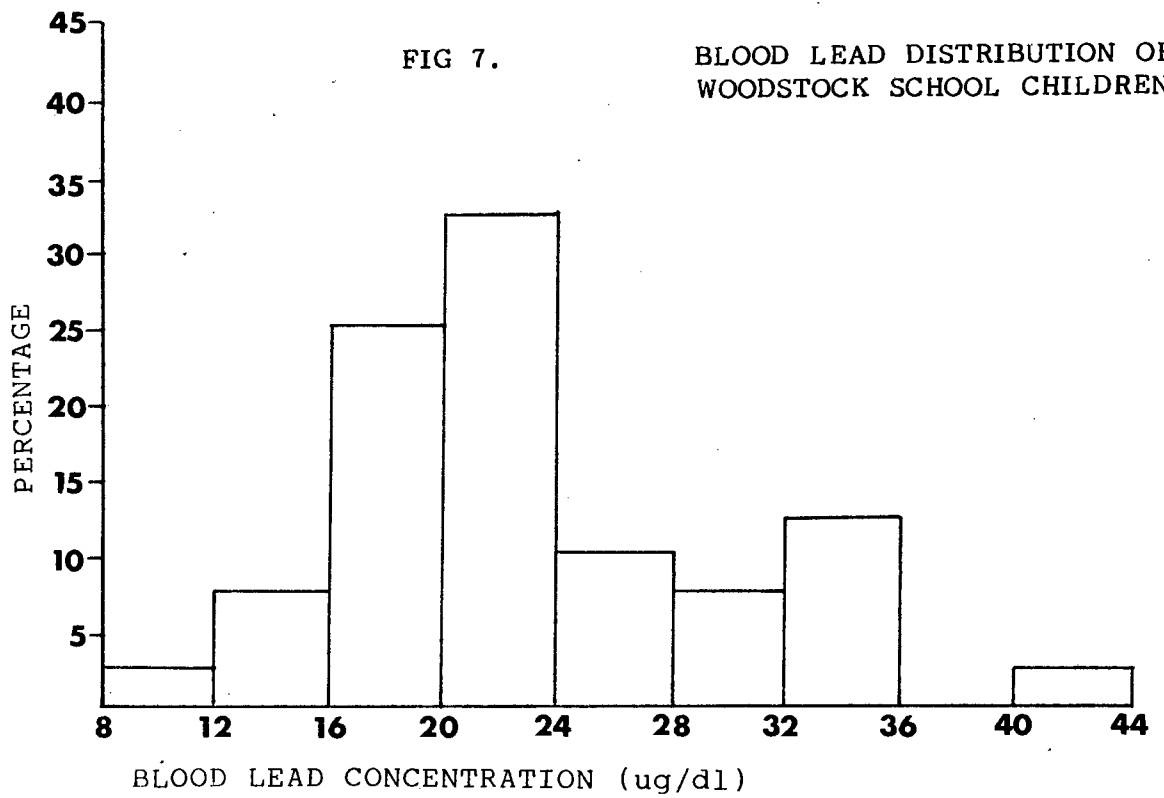


FIG 7.

BLOOD LEAD DISTRIBUTION OF
WOODSTOCK SCHOOL CHILDREN



lead distribution are given in Figure 6. From Tables 7 and 8, it is evident that the blood lead distribution of the children living in Hout Bay is markedly different from that of the children with elevated ZPP levels. On average, children identified with raised ZPP levels by the screening test had blood lead concentrations nearly twice as high as children in the control group. Approximately 50% of the children in Hout Bay had blood lead concentrations below 11 ug/dl, whereas only 10% of the children identified by the screening test had levels below this value. Twenty-five percent of these children had blood lead concentrations over or above 25 ug/dl. It was evident, therefore, that the children identified by the screening test had significantly increased blood concentrations when compared with a control group.

Whether or not such blood lead levels can be considered acceptable from a health point of view, is uncertain. There is at present considerable debate as to what a 'safe' blood lead level is, and the threshold level for blood lead concentrations, above which children are considered to be at risk, has been continually downgraded in recent years. The United States Center for Disease Control (88) has set a limit of 29 ug/dl above which children are considered in potential danger of developing clinical lead poisoning (a revision of the previous 1970 United States recommendation that 40 ug/dl be considered a cut-off level) (2).

There is now evidence that adverse health effects may occur at levels below 29 ug/dl (9,10,11,12) and many workers believe that the current safety threshold level should be lowered further (9,10,89).

Although there is at present little consensus as to what constitutes an acceptable blood lead concentration in children, depending on the standard one wishes to adhere to, 15% of the children with elevated ZPP levels would be regarded at risk in terms of the United States standard of 29 ug/dl and almost twice as many would be regarded at risk if a safety threshold of 25 ug/dl were adopted.

Looking at the distribution of blood lead concentrations by school (Appendix 8), it is evident that a larger percentage of children living in urban-industrial areas had blood lead levels greater than or equal to 25 ug/dl than children living in residential areas. 36% in the former group as compared to 6% in the latter group had blood lead concentrations greater than or equal to 25 ug/dl. Of the children in the urban-industrial group a large percentage (56%) of children with 'high' blood lead concentrations attended one school in particular - Woodstock.

It was subsequently decided to perform blood lead analyses on all sub A children at the school in Woodstock to determine the blood lead distribution for the whole class.

2.4.2.2 WOODSTOCK CASE STUDY

Sample Description: All sub A pupils, with parental permission, present at school on the day of the study, were sampled. The parental response rate was 97,5%. Altogether 40 children were sampled, of whom 39 had previously had ZPP tests performed.

Methods and Techniques: Approximately 5 mls of venous blood were taken from each child by venipuncture into lead-free heparinised tubes. Samples were taken during the last week of February. Blood lead tests and full blood counts were performed on all samples.

Results and Discussion

Descriptive statistics of the blood lead and haemoglobin distributions are given in Table 9. Details of the blood lead distribution are given in Figure 7.

(a) Confirmation of Blood Lead Results of Children with Elevated ZPP Levels

Although blood lead analyses on children with elevated ZPP levels had previously been performed, it was decided to repeat these tests in order to confirm the results. The results of the two analyses are given in Table 10.

TABLE 9: WOODSTOCK SCHOOLCHILDREN:
DISTRIBUTION OF BLOOD LEAD AND HAEMOGLOBIN (Breakdown by Sex)

	Males			Females			Total		
	Freq.	Mean	S.d. Median	Freq.	Mean	S.d. Median	Freq.	Mean	S.d. Median
Blood lead (ug/dL)	22	23,9	7,8 23	18	21,3	5,0 20	40	22,8	6,7 21
Hb (g/dl)	10	13,1	0,9 12,9	5	13,3	0,5 13,4	15	13,1	0,8 13,4

TABLE 10. RESULTS OF FIRST AND SECOND BLOOD LEAD ANALYSES
(ug/dl)

Blood Lead (2)	Blood Lead (1)
19	17
30	30
23	23
34	30
23	22
26	28
18	18
30	27
33	20
29	26
20	20
41	37
33	32
20	24
22	19
34	30

It is evident from these results that there was a close correlation between the blood lead concentrations in the two series of samples taken, with the blood lead levels in the first series being on average slightly lower than the second. Two factors may account for this:

Firstly, there was a difference of four months between the times when the samples were taken. The initial samples were obtained in September, whereas the subsequent ones were obtained in February. It is possible that during this period there was a change in lead levels to which children were exposed.

Secondly, there may have been a slight change in blood lead concentration due to the freezing of samples prior to analysis. (The initial samples were frozen for a period of approximately one month prior to analysis whereas the second samples were analysed directly after collection.) It has been documented, however, that blood lead concentrations are not affected by temperature (66,90). This has been verified by quality control tests carried out at Red Cross Hospital (Mr. Dempster, pers. comm.).

What is of significance for this study is the close correlation between the two series of blood lead analyses, which serves to indicate that the results obtained are reliable; and, do not merely reflect a temporary rise in blood lead concentration.

(b) Difference Between Classes

A considerable difference was evident in the mean blood lead levels obtained for children in the two sub A classes at Woodstock School. The blood lead distributions for the two classes are given in Table (11) and Figures 8 and 9.

The children in the A2 class had higher blood lead levels on average than the children in the A1 class. Seven children in the A2 class (five males and two females) had blood lead concentrations greater than or equal to 30 ugPb/dl blood whereas only one child in the A1 class had a blood lead concentration of this magnitude.

It was decided to determine whether the difference in mean blood lead concentration between the two classes was statistically significant. A median test was performed and the difference between the blood lead levels in the two classes was found to be significant (chi sq. 5.777, d.o.f.1, p .0162).

Further investigation is needed to explain the difference in blood lead concentration observed between the two classes. There was no obvious source of lead in either sub A classroom and all children lived in the same residential area of Woodstock.

TABLE 11. BLOOD LEAD DISTRIBUTION OF WOODSTOCK SCHOOL CHILDREN
 (ug/dl)
 Breakdown by class.

Class	Frequency	Mean	s.d.	Median
A1	18	20,1	5,06	20
A2	21	25,3	7,16	26

FIG 8. BLOOD LEAD DISTRIBUTION (ug/dl) OF WOODSTOCK SCHOOL CHILDREN, CLASS A.1

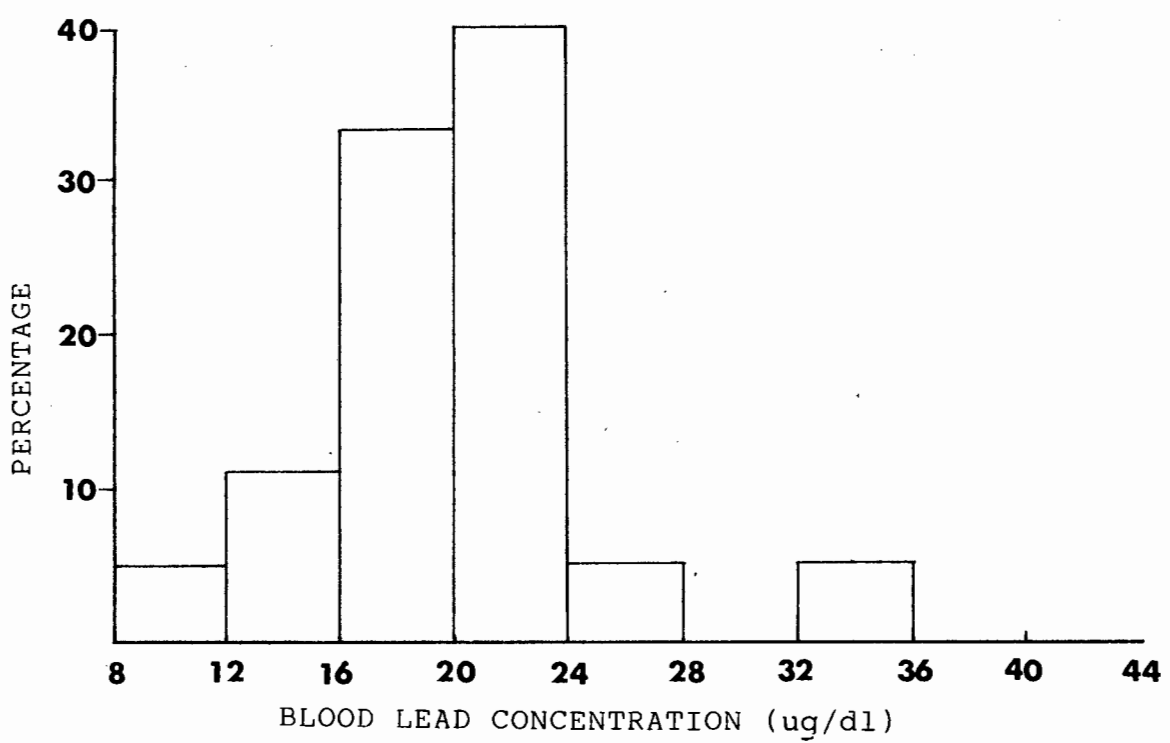
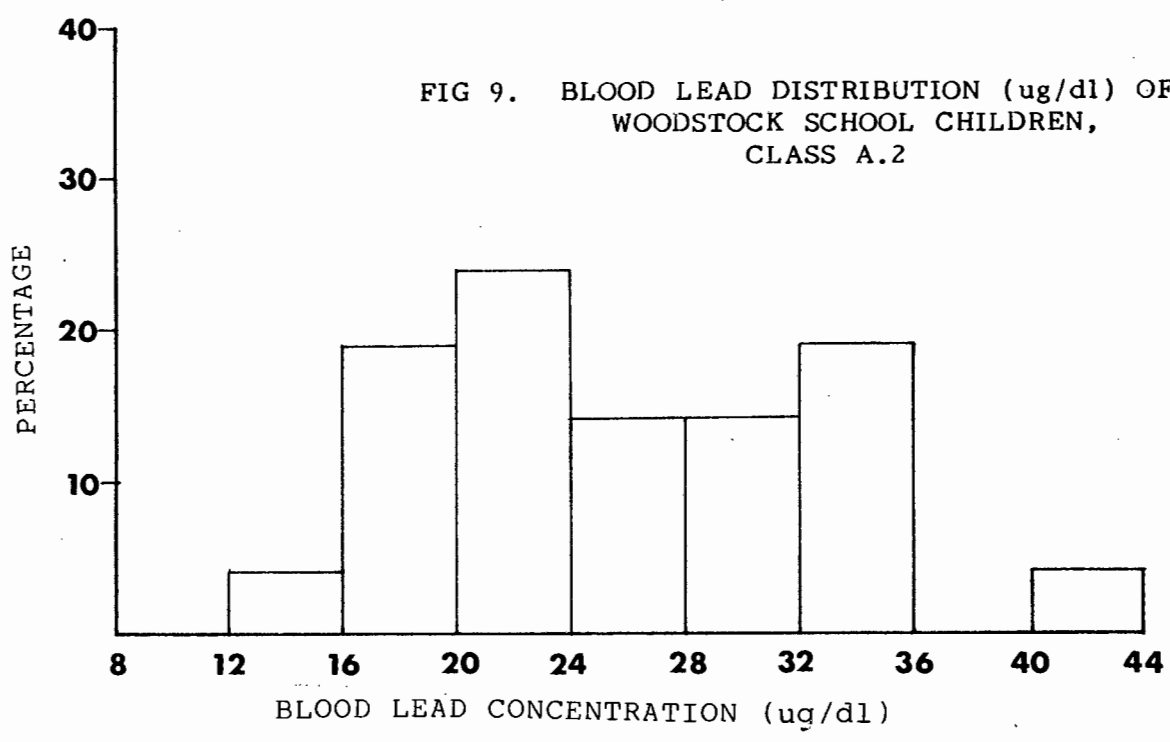


FIG 9. BLOOD LEAD DISTRIBUTION (ug/dl) OF WOODSTOCK SCHOOL CHILDREN, CLASS A.2



(c) Difference Between Schools: Hout Bay and Woodstock

From Tables 8 and 9, and Figures 6 and 7, it is evident that children at the Woodstock school had considerably higher blood lead levels than children at the Hout Bay school. The mean blood lead concentration of the Woodstock children (22.8 ug/dl) was more than twice that of the Hout Bay children, which was 11 ug/dl. At the Woodstock school the mean blood lead concentration of females was slightly less than that of males, but no difference was observed between the sexes at the Hout Bay school.

The distribution of blood lead concentrations was such that there was almost a three-fold difference between the highest blood lead concentrations at the two schools. At Woodstock the highest blood lead level was 41 ug/dl, whilst at Hout Bay the highest was 15 ug/dl. Thirty-two-and-a-half percent of the Woodstock children had blood lead concentrations greater than or equal to 25 ug/dl.

A median test was performed to determine whether the observed difference in blood lead concentrations between the schools was statistically significant. The difference between the two schools was found to be highly significant (chi sq. 68.014, d.o.f. 1, p .0001).

From these results it is evident that the blood lead concentrations of children from a school in an urban environment are significantly higher than those of children from a school in a semi-rural environment. The difference

in blood lead concentrations may relate to a difference in exposure to lead aerosols in the two areas. For instance, the children from Woodstock were more exposed to traffic than children from Hout Bay (the hourly traffic flow measured on the street in which the Woodstock school is situated, is given in Appendix 9). Many studies have shown that blood lead concentrations of urban populations and of people heavily exposed to automobile exhaust are generally higher than those of rural populations and of people living in areas with less traffic (5,48,49,50,51).

Although traffic may be an important source of exposure in Woodstock children, it would be necessary to investigate other possible sources of exposure in order to determine the relative importance of various sources.

Possible contributory sources of lead exposure in children identified with increased blood lead levels in the screening study (Woodstock children and others) are discussed in Chapter 3.

2.4.3 Summary and Conclusions

ZPP concentrations were measured in 1 234 schoolchildren from various schools in greater Cape Town. Approximately 7% of the study population had raised ZPP levels.

Highly significant differences in ZPP concentration were

found between the sexes and between groups of schools:

- * Males had higher ZPP levels than females. Eight percent of males as opposed to 5% of females had raised ZPP levels.
- * Children from schools in urban-industrial areas had higher ZPP levels than children from other schools. Almost twice as many children in the former group had raised ZPP levels.
- * Children from schools situated on through roads had higher ZPP levels than children from schools on other roads, when the nature of the recruitment area was taken into account.

Confirmatory blood lead and other hematalogical tests were performed on children with raised ZPP levels. Twenty-five percent of children had blood lead concentrations close to, or exceeding, the currently accepted safety level of 29 ug/dl. Blood lead concentrations were highest amongst children from Woodstock and central Cape Town.

Further blood lead and other hematological tests were performed on two groups of children: all sub A pupils at the Wodstock school and sub A and sub B pupils at the school in Hout Bay. A highly significant difference in blood lead concentration was found between children at the two schools.

- * Blood lead concentrations of Woodstock children were twice as high as those of Hout Bay children. Seventeen percent of Woodstock children had blood lead levels exceeding the safety level of 29 ug/dl.

A significant difference in blood lead concentration was also found between the two sub A classes at Woodstock.

It is concluded that a significant proportion (7%) of the study population, mainly children living in urban-industrial areas, may have increased lead burdens and may be undergoing certain biochemical changes in heme synthesis.

Children living in areas such as Woodstock may be at particular risk. It is suggested that detailed environmental investigations in these areas be undertaken, in order to determine the major source(s) of exposure.

PART II

2.4.4 The Relationship Between ZPP and Blood Lead Concentration

In order to determine whether a correlation existed between ZPP and blood lead concentration, Spearman rank correlation coefficients were estimated on two samples: children with elevated ZPP levels and children from Hout Bay and Woodstock schools. Regression analyses were performed on the log transformed values of ZPP for both samples.

Results

2.4.4.1 CHILDREN WITH ELEVATED ZPP LEVELS

Correlations: Correlation coefficients were estimated for males, females, and for both sexes together. The results are given in Table 12.

It is evident that correlation coefficients were weak, particularly in females, in which the coefficient was not statistically significant. In males, it was significantly different from zero, although the correlation explained only 13,7% of the observed variation in the two variables. With males and females analysed together, the correlation coefficient was significant, but explained only 9% of the variation.

Regression Analysis: Regression analysis was performed for males only, as no significant correlation was found in females.

Using blood lead as the response variable, the regression equation was:

$$\text{blood lead} = -10.37 + (16.55 \times \log \text{ of ZPP})$$

With r square = .267, the regression model explained only 26,7% of the observed variation. A scatter diagram showing

TABLE 12: CORRELATIONS

	Hout Bay/Woodstock			ZPP > 5		
	Males	Females	Total	Males	Females	Total
ZPP vs Blood lead	r 0,67052	0,58882	0,63545	r 0,37464	0,11670	0,30076
	p 0,0001	0,0001	0,0001	p 0,0080	0,6050	0,0108
	d.o.f. 36	37	73	d.o.f. 49	22	71
ZPP vs BPb	r 0,32189	0,29751	0,31599	r 0,05368	0,04495	0,05088
	p 0,1166	0,1580	0,270	p 0,7325	0,8550	0,6947
	d.o.f. 25	24	49	d.o.f. 43	19	62
BPb vs Hb	r 0,33733	0,30611	0,34125	r 0,10577	0,08087	0,00588
	p 0,6853	0,1131	0,0108	p 0,5160	0,7497	0,9651
	d.o.f. 27	28	55	d.o.f. 40	18	58

observed and predicted values was plotted. Similarly, residuals were plotted against the independent variable. This plot of residuals showed no particular pattern. (See Figures 10 and 11.)

Using ZPP as the response variable, the regression equation was:

$$\log \text{ of ZPP} = 1.56 + (0.02 \times \text{blood lead})$$

A scatter diagram showing observed and predicted values was plotted as well as a plot of the residuals; this showed no particular pattern (see Figures 12 and 13).

2.4.4.2 CHILDREN FROM HOUT BAY AND WOODSTOCK SCHOOLS

Correlations: Correlation coefficients were estimated for males, females and for both sexes together. Results are given in Table 12. In these schools the correlation coefficients were stronger and statistically significant in both sexes, although the correlation was stronger in males than in females. It explained 45% and 35% of the variation in males and females respectively. For all children analysed together, the correlation coefficient explained 40% of the variation.

Regression Analysis: Regression analysis was performed on males and females together, as a significant correlation

FIGURE 10. SCATTER DIAGRAM WITH REGRESSION LINE

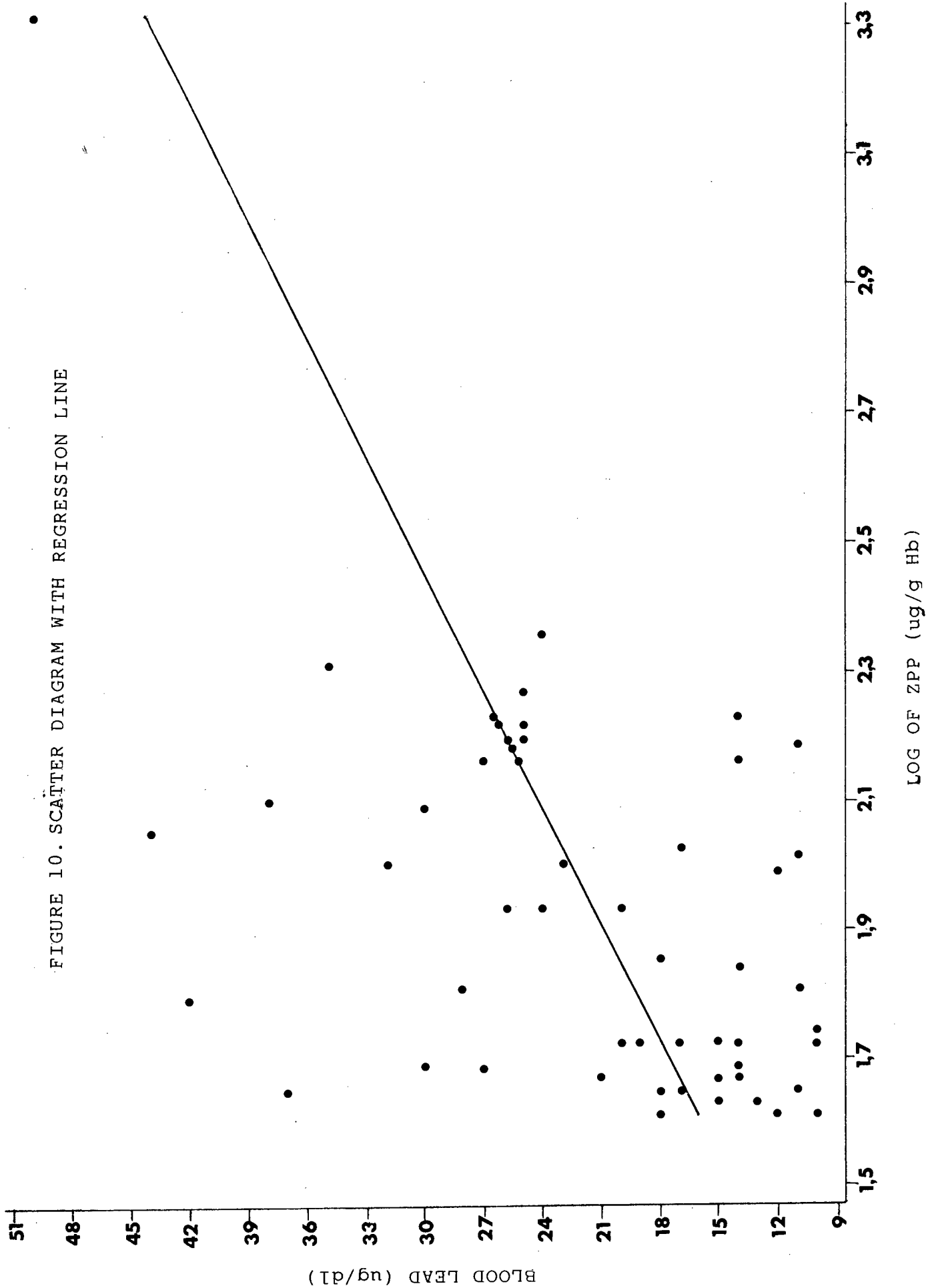
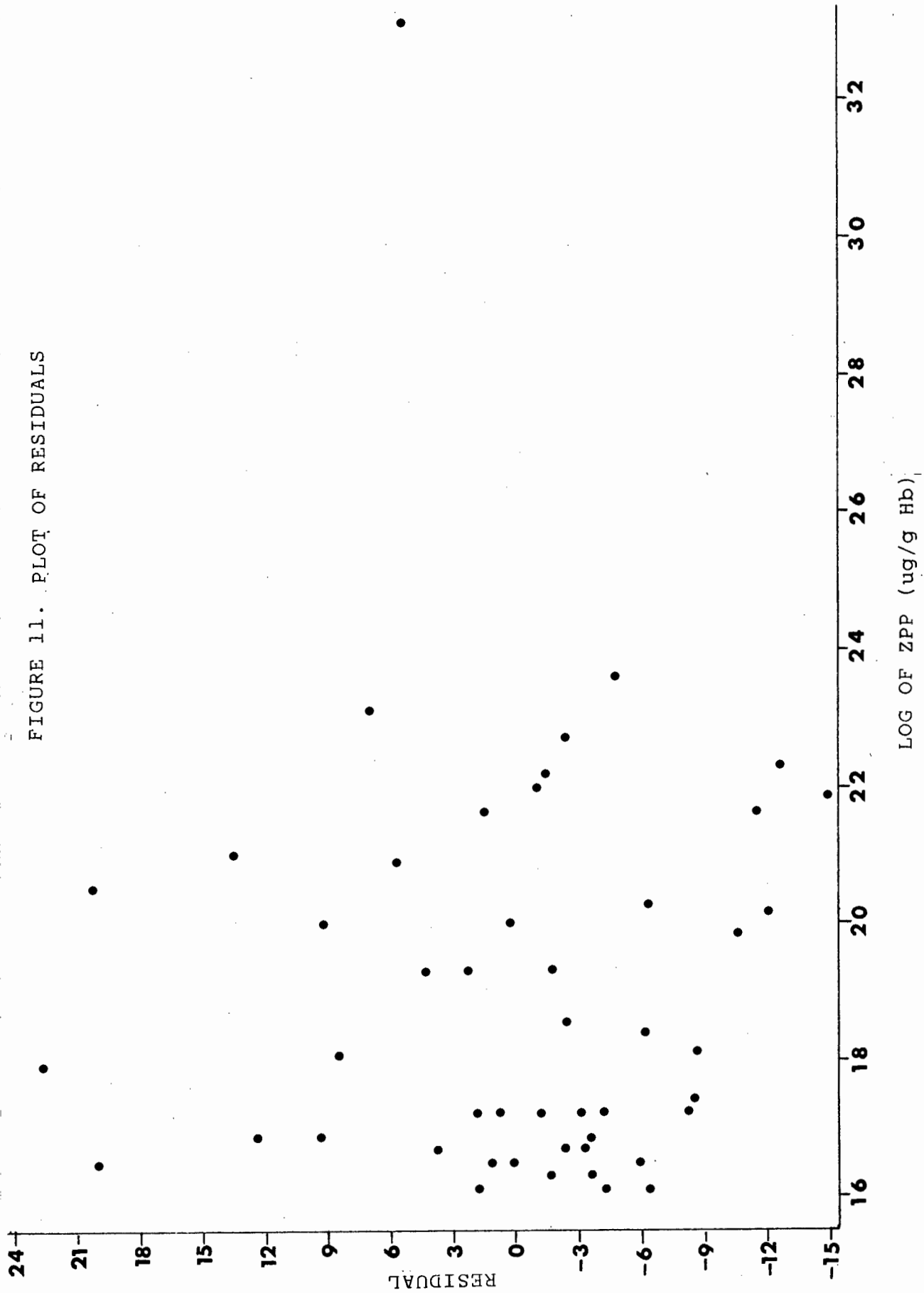


FIGURE 11. PLOT OF RESIDUALS



LOG OF ZPP (ug/g Hb)

FIGURE 12. SCATTER DIAGRAM WITH REGRESSION LINE

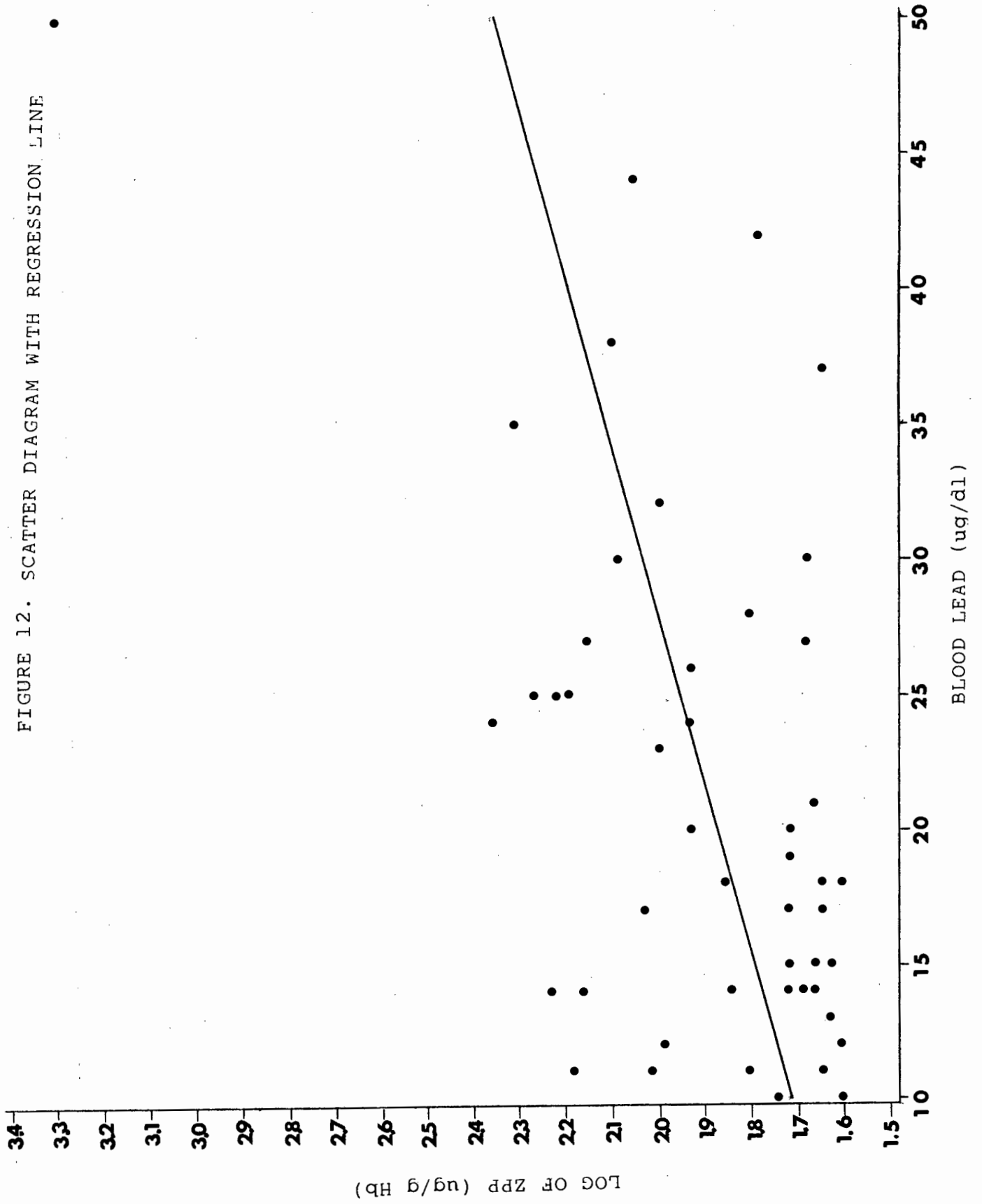
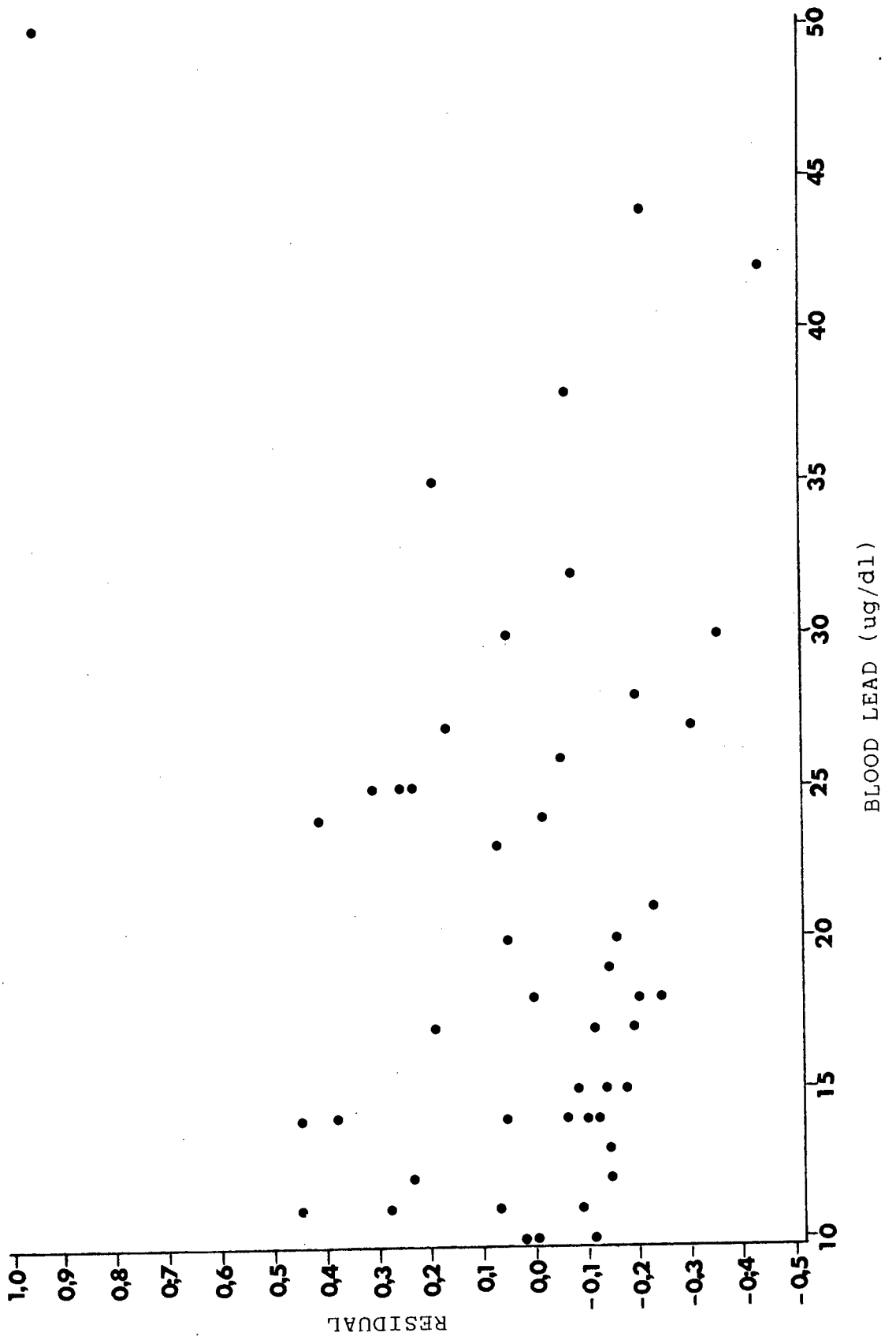


FIGURE 13. PLOT OF RESIDUALS



was found for both sexes. Using blood lead as the response variable, the regression equation was:

$$\text{blood lead} = 1.83 + (12.26 \times \log \text{ of ZPP})$$

With r square = .406, the regression equation explained 40,6% of the variation.

Using log of ZPP as the response variable, the regression equation was:

$$\log \text{ of ZPP} = .70 + (.03 \times \text{blood lead})$$

A scatter diagram of observed and predicted values was plotted for both regression analyses as well as for the residuals - these showed no particular pattern (see Figures 14, 15, 16, 17).

Discussion

The correlation coefficients determined on the two samples, whilst significant, are weaker than those that have been reported in the literature. These range from .72 to .91 (91), but refer mainly to occupationally exposed populations.

Correlation coefficients determined on children have been reported to be somewhat lower. In a study by Sassa et al. (92) on children with blood lead concentrations between 20 and 80 ug/dl, a correlation coefficient of .72 was determined. In a study by Lamola et al. (93) on a group

FIGURE 14. SCATTER DIAGRAM WITH REGRESSION/LINE

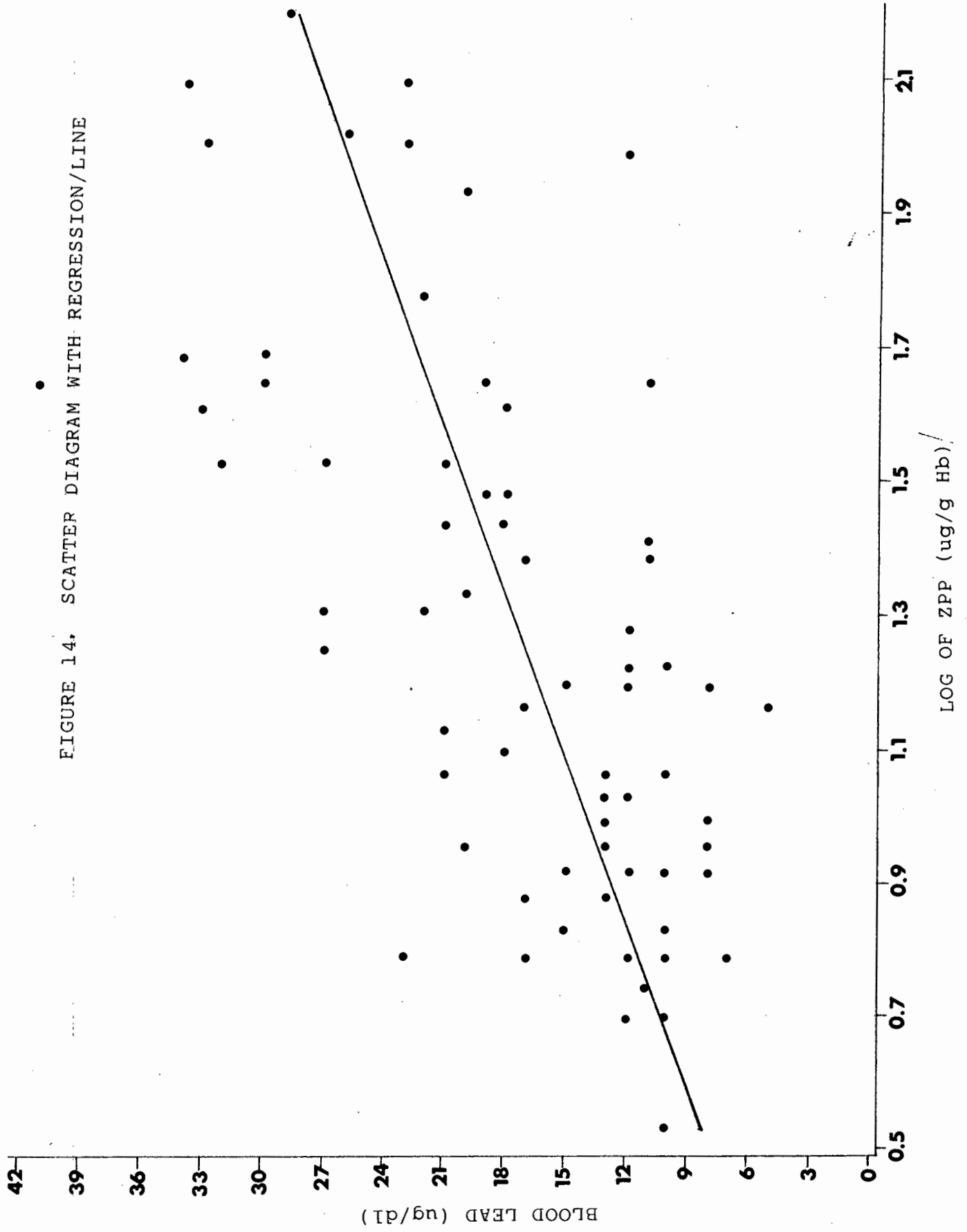


FIGURE 15. PLOT OF RESIDUALS

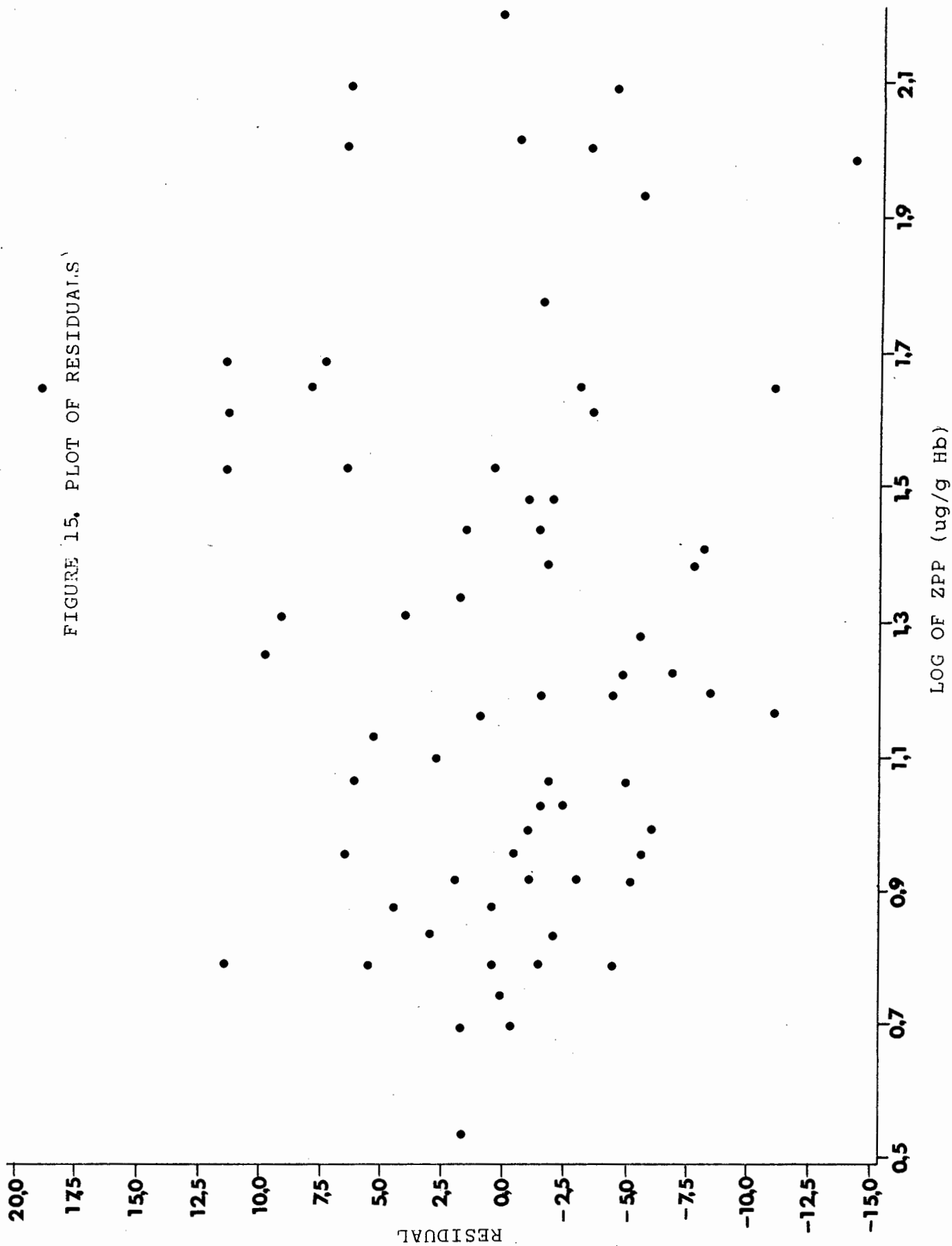


FIGURE 16. SCATTER DIAGRAM WITH REGRESSION/LINE

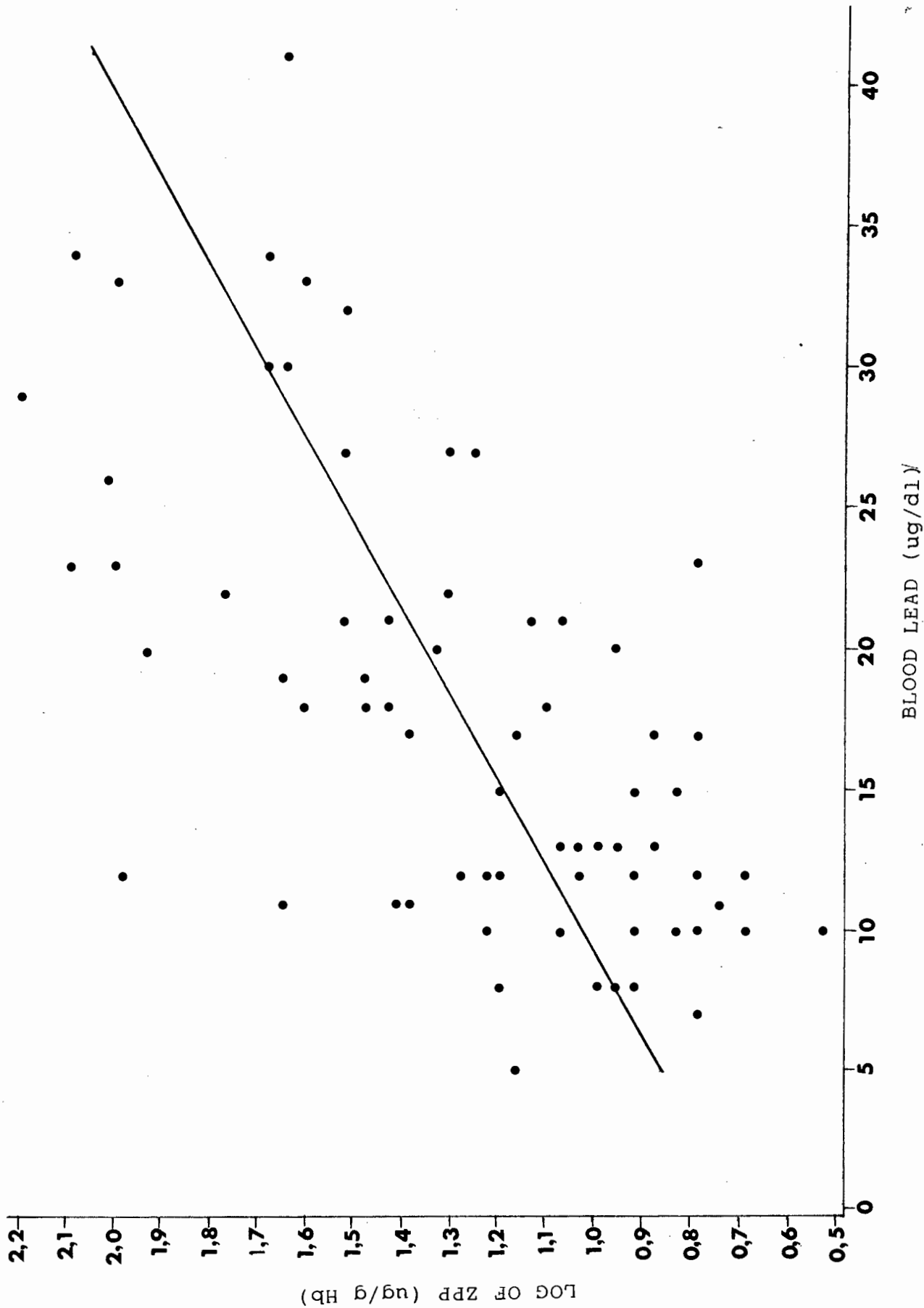
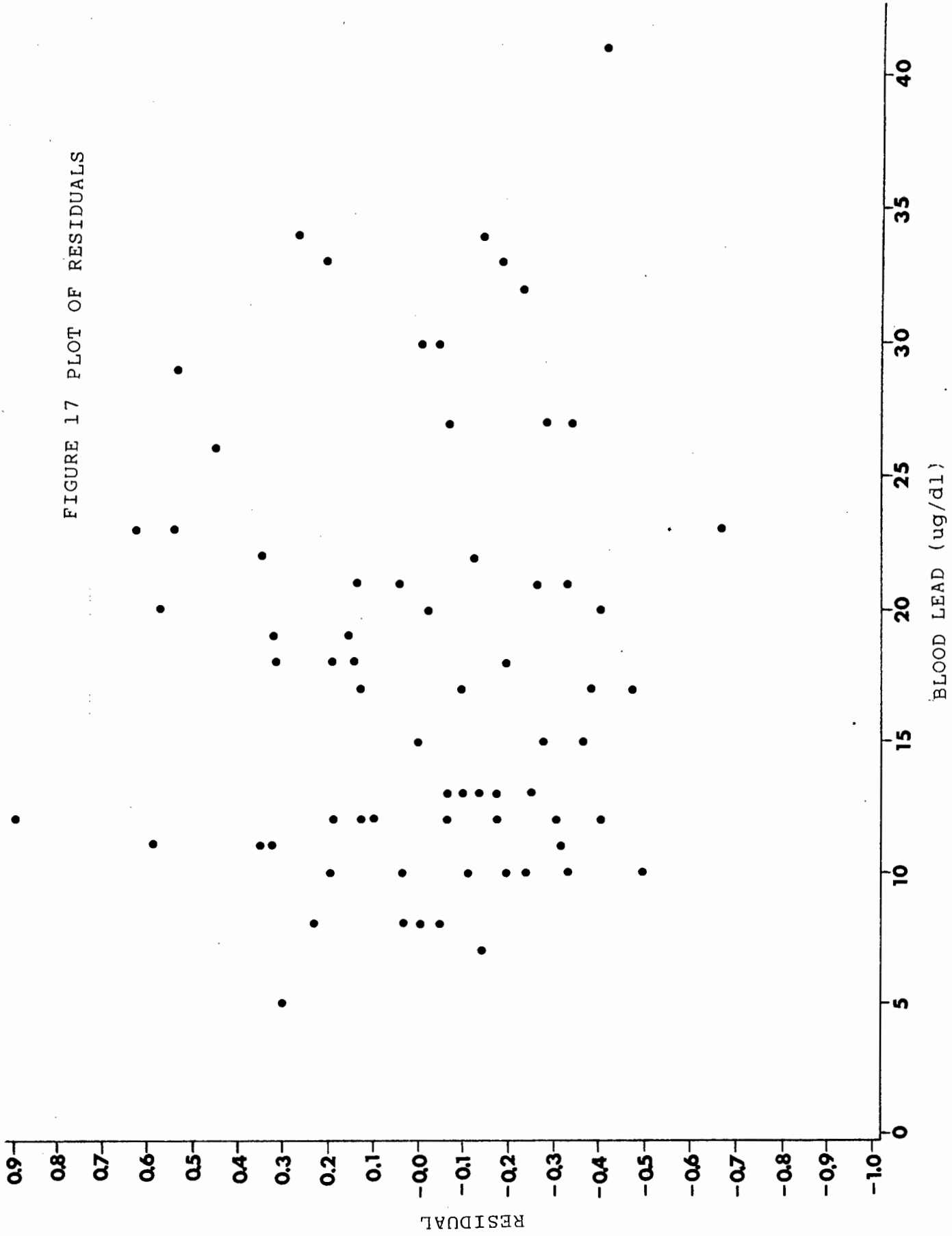


FIGURE 17 PLOT OF RESIDUALS



of 250 young children, a correlation coefficient of .77 was determined. It was not stated in what way children tested were chosen for study - for instance, whether they were from selected "high risk" populations or from the general population.

There is at present much uncertainty regarding the minimum blood lead concentrations necessary for correlations between ZPP and blood lead to be significant.

According to Hesley (91), in studies where no correlation between ZPP and blood lead concentrations have been shown, the blood lead concentration may not have been stable for a long enough time to allow ZPP to accurately reflect the body burden. For instance, there is a time lag in the appearance of ZPP following exposure to lead, and a similar time lag following removal from the exposure.

Previously, workers suggested that ZPP levels only increase significantly at blood lead levels of 50 ug/dl. Others have stated that increases occur in children at blood lead levels of 25-30 ug/dl (89), whilst a recent study has shown that increases occur in children at blood lead levels as low as 15 ug/dl (9).

Data collected in the present study are not sufficient to determine accurately the nature of the relationship between ZPP and blood lead. This would require larger samples

and an even distribution of data points - the data in this study were not collected for this purpose.

Nevertheless, from the correlations demonstrated in the present study, it is evident that ZPP may be a sensitive indicator of lead absorption, but not necessarily specific for lead absorption. The weak correlation coefficient determined in the sample of children with elevated ZPP levels (which was significant only in males) may indicate that the ZPP test is not specific for lead absorption; whilst the much stronger, statistically significant correlation determined in the sample of Woodstock-Hout Bay children may indicate that the test is sensitive to lead absorption, even at considerably low blood lead concentrations.

2.4.5 The Relationship Between ZPP, Haemoglobin and Blood Lead Concentration

It has been well documented that an accumulation of ZPP may occur in conditions other than increased lead absorption, for instance, iron-deficiency and significant correlations between ZPP and haemoglobin concentration have been demonstrated (94).

On the other hand, increased lead absorption may cause a decrease in haemoglobin concentration and significant correlations between haemoglobin and blood lead concentration have been demonstrated (95).

Despite the fact that the majority of children in both samples had haemoglobin levels in the normal range, it was decided to determine whether a correlation existed between haemoglobin and ZPP or blood lead concentrations.

2.4.5.1 HAEMOGLOBIN AND ZPP CONCENTRATION

Correlation coefficients were estimated for males and females in both samples, but were not found to be statistically significant in either group. Results are given in Table 12.

2.4.5.2 HAEMOGLOBIN AND BLOOD LEAD CONCENTRATION

Similarly, correlation coefficients were estimated for males and females in both samples, but were not found to be statistically significant in either group. Results are given in Table 12.

It is suggested that haemoglobin concentration is only significantly decreased with relatively high blood lead concentrations, above those in the range reported here.

Conclusions

It is concluded that at low levels of lead exposure, ZPP is sensitive, but not specific to lead. As children with

raised ZPP levels were not iron-deficient, and there was no significant correlation between ZPP and haemoglobin concentration, it is suggested that conditions other than iron-deficiency may cause an accumulation of ZPP. Further investigation is needed to determine the factors affecting blood protoporphyrin synthesis.

LEAD EXPOSURE IN SELECTED CASE STUDIES3.1 INTRODUCTION

A secondary aim of the present study was to determine possible sources of lead to which children identified with increased blood lead levels were exposed. It was decided to investigate the home environments of children, to determine whether there were any obvious localised sources of exposure, such as lead piping, or flaking lead-based paint.

3.2 SELECTION OF CHILDREN

Children with a blood lead level greater than or equal to 25 ug/dl were judged as having increased blood lead levels. There were twenty such children who were selected for study.

3.3 HOME INVESTIGATIONS

At each house visited, a questionnaire was administered to the parent or guardian of the child, and a water sample was obtained for lead analysis. In certain homes, (those built prior to 1950) paint, soil and dust samples were also obtained for lead analysis.

3.3.1 Questionnaire

Personal interviews with parents or guardians were granted in all homes visited. Information was obtained on the family's socio-economic circumstances, the physical condition of the house (with particular reference to factors such as condition of painted surfaces, piping) and the physical environment (with reference to factors such as position of the house with respect to traffic or industry).

In addition, information was obtained on the occupational histories of residents in the house, medical and behavioural histories of children (with reference to factors which might affect exposure, such as pica, primary play-site, amount played outdoors) and other individual items of importance. A checklist of items contained in the questionnaire is given in Table 13.

Results

Personal Data

Personal details of children are given in Table 14.

Socio-Economic Data

(See Table 15). The majority of parents or guardians of children had completed (or had partially completed) primary school, and were employed as unskilled or semi-skilled

TABLE 13. CHECKLIST OF ITEMS IN QUESTIONNAIRE.

<u>Personal Data:</u>	Name, School, Class, Address, Sex, Date of Birth, Religion, Home Language.
<u>Socio-Economic Data:</u>	Father's/Mother's Occupation, Education, Income; Rent, Number of Children, Number of People Living in Dwelling, Number of Rooms in Dwelling.
<u>Housing and Environmental Data:</u>	Type of Dwelling, Ownership, Age of Dwelling, Storey, Length of Residence, General Condition, Piping, Energy Source, Condition of Painted Surfaces, Time since Last Painted, Position of Dwelling with Respect to Major Roads, Industry.

Table 13 Continued

Behavioural and Other Data: Amount Played Outdoors, Primary Play Site, Route Travelled to School, Behavioural History (Evidence of Pica), Medical History (Number of Hospital Admissions), Behavioural and Medical History of Siblings, Nutrition Status, Amount of Tinned Food Consumed, Cooking Utensils, Occupational Histories of Residents in House.

TABLE 14: PERSONAL DATA

Child No.	Results of Blood Analyses			Sex	Age (years)	Home Language	Religion	Address	School
	ZPP (ug/g Hb)	BPb (ug/dl)	Hb (g/dl)						
1	5.2	41	11.9	M	6	Afrikaans	Christian	Woodstock	2
2	5.4	30	13.9	M	6	Afrikaans	Christian	Woodstock	2
3	5.0	33	12.8	F	7	Afrikaans	Christian	Woodstock	2
4	5.4	34	13.3	M	6	Afrikaans	Christian	Woodstock	2
5	9.0	29	13.0	M	7	Afrikaans	Muslim	Woodstock	2
6		30		F	7	Afrikaans	Christian	Hanover Pk (prev. Woodstock)	2
7	8.1	34	13.4	M	6	Afrikaans	Muslim	Cape Town	2
8	7.4	33	13.6	M	8	English	Christian	Woodstock	2
9	7.5	28	13.9	F	6	Afrikaans	Christian	Woodstock	2
10	9.7	25	13.4	M	7	Afrikaans	Christian	Cape Town	9

Table 14 Continued

Child No.	Results of Blood Analyses		Sex	Age (years)	Home Language	Religion	Address	School	
	ZPP (ug/g Hb)	BPb (ug/dl)							Hb (g/dl)
11	10.1	35	-	F	8	Afrikaans	Christian	Cape Town	9
12	7.8	44	13.7	M	6	English	Muslim	Cape Town	9
13	6.1	28	13.6	M	9	Afrikaans	Christian	Cape Town	9
14	27.5	50	9.9	M	7	Afrikaans	Christian	Athlone	12
15	8.7	27	12.6	M	6	Afrikaans	Christian	Bellville South	6
16	6.0	42	11.5	M	9	Afrikaans	Christian	Maitland	17
17	8.2	38	7.8	M	8	Afrikaans	Christian	Retreat	13
18	6.9	26	12.6	M	7	Afrikaans	Christian	Claremont	3
19	7.3	35	11.5	F	7	Afrikaans	Muslim	Claremont	3
20	9.2	25	9.0	M	8	Afrikaans	Muslim	Elsies River	1

TABLE 15. SOCIO-ECONOMIC DATA

Father's Occupation	Mother's Occupation	Father's Educatn.	Mother's Educatn.	Family Income (per month)	Rent (per month)	Number of people per dwelling	Number of rooms per dwelling
1 Caretaker/ handyman	Home	Sub B.	Std. 2	R140, 00	-	4	3
2 Dock worker	Sweet factory	Std. 5	Std. 5	R312, 00	-	5	4
3 Cleaner	Home	std. 8	Std. 5	R236, 00	R7.50	13	1
4 Sea Patrol	Home	Std. 3	Std. 7	R270, 00	-	-	-
5 Prison	Home	Std. 6	Std. 2	R130, 00	-	11	2
6 Cleaner	Home	Std. 3	Std. 4	R189, 00	R33-00	5	3
7 Printing works	Clerical	Matric	Std. 8	-	R50-00	7	4
8 Forklift driver	Home	Std 5	Std. 5	R324, 00	R28-00	13	3
9 Labourer	Clothing factory	Std. 6	Std. 5	R240, 00	R30-00	7	3
10 Labourer (plumbing)	Home	Std. 4	Std. 3	R180, 00	R24-00	6	3

(Table 15 continued)

Father's Occupation	Mother's Occupation	Father's Educatn.	Mother's Educatn.	Family Income (per month)	Rent (per month)	Number of people per dwelling	Number of rooms per dwelling
11 Plumber	Home:	Std. 6	Std. 4	R360 -00	R13-00	11	3
12 Painter	Home	Std. 1	Std. 5	R260 -00		6	4
13 Driver	Home	Std. 7	Std. 6	R360 -00	R40-00	6	2
14 Plasterer	Domestic Serv.	Std. 4	Std. 5	R200 -00	R62-00	7	3
15 Bricklayer	Domestic Serv.	Std. 7	Std. 4	R330 -00	R45-00	10	3
16 Caretaker	Clothing factory	None	Std. 4	R140 -00	R30-00	11	3
17 Painter	Home	Std. 5	Std. 6	-	-	5	2
18 -	Home	-	Std. 6	R171 -00	-	5	3
19. Gardener	Clothing factory	None	Std. 4	R385 -00	R9-00	5	3
20. Labourer	Domestic Serv.	Std. 4	Std. 3	R215 -00	R6,40	13	2

workers. Women were employed in the clothing industry or domestic service, whilst men were mainly employed in the building industry as artisans.

The average family income was R246,00 per month, ranging from R130 - R385 per month. On average about five to six people were supported on this income. The majority of families lived in rented flats or houses, whilst one family lived in a tin shanty. The average rent paid was R29,00 per month, ranging from R6,40 to R62,00 per month. Many of the children lived in overcrowded conditions, with an average of seven to eight people living in three-roomed flats or houses. A third of the homes had ten or more inhabitants. In one particular case, 13 people lived in a one-room flat.

Physical Characteristics of Houses

(See Table 16). The majority of houses (particularly those in Woodstock and central Cape Town) were over fifty years old and were poorly maintained and dilapidated. Most had no electricity supply and used paraffin or gas for fuel. Many of the houses were dark, poorly ventilated and dusty.

All houses had internal copper piping, with the exception of one house which had galvanised iron pipes.

TABLE 16: HOUSING AND ENVIRONMENTAL DATA

No.	Condition of Dwelling (with ref. to painted surfaces)	Age of Dwelling	Piping	Energy Source	Position of Dwelling with respect to major road(s) and industry.
1	Well maintained	> 50 years	Cu	Electricity	< 50 metres from railway line, < 50 m from major road, < 20 m from automobile body works.
2	Interior well maintained, exterior deteriorating, flaking.	> 50 years	Cu	Oil, paraffin	50-200 m from major road.
3	Very badly maintained, dilapidated. Ceiling flaking.	> 50 years	Cu	Gas, paraffin	50-200 m from major road. < 50 m from automobile body works.
4	Badly maintained, dilapidated, exterior flaking	> 50 years	Cu	Gas	On major road. < 20 m from sheet metal works.
5	Badly maintained, dilapidated. Isolated flaking surfaces	> 50 years	Cu	Gas, paraffin	> 200 m from major road.
6	Well maintained	< 20 years	Cu	Paraffin	> 200 m from major road.
7	Interior very well maintained, exterior deteriorating, flaking	> 50 years	Cu	Electricity	> 200 m from major road.
8	Badly maintained, dilapidated. Windowsill flaking	> 50 years	Cu	Wood, paraffin	50-200 m from major road, < 50 m from railway line < 20 m from sheet metal works.
9	Quite well maintained	> 50 years	Cu	Electricity	50-200 m from major road, < 50 m from railway line. < 20 m from sheet metal works.
10	Interior quite well maintained, isolated flaking surfaces. Exterior dilapidated.	> 50 years	Cu	Gas	> 200 m from major road. < 20 m from garage.

Table 16 Continued

No.	Condition of Dwelling (with ref. to painted surfaces)	Age of Dwelling	Piping	Energy Source	Position of Dwelling with respect to major road(s) and industry.
11	Interior quite well maintained, exterior deteriorating. Isolated flaking surfaces	> 50 years	Cu	Gas	> 200 m from major road.
12	Very well maintained	> 50 years	Cu	Electricity	On major road, < 20 m from spray painting and body works.
13.	Interior quite well maintained. Exterior deteriorating.	> 50 years	Cu	Gas	< 50 m from major road.
14.	Well maintained	> 50 years	Cu	Electricity	< 50 m from major road.
15	Well maintained	> 50 years	Cu	Electricity	> 200 m from major road. 50-200 m from shooting range.
16.	Well maintained.	< 40 years	Galv. iron	Electricity	> 200 m from major road.
17	Well maintained	< 20 years	Cu	Electricity	> 200 m from major road.
18	Badly maintained, dilapidated. Interior and Exterior surfaces flaking.	> 50 years	Cu	Wood	50-200 m from major road.
19	Interior quite well maintained. Exterior dilapidated, flaking	> 50 years	Cu	Wood	50-200 m from major road.
20	Very badly maintained, dilapidated shanty. Isolated flaking surfaces	< 20 years	-	Paraffin	> 200 m from major road.

In many of the houses, the exterior walls had a significant degree of flaking paint, but the interior surfaces were mostly intact. Usually only one or two flaking surfaces could be found. One house, however, had nearly all its interior walls in a flaking condition.

Physical Environments

(See Table 16). Most of the children lived in mixed residential/urban-industrial areas such as Woodstock and central Cape Town, whilst others lived in various residential suburbs throughout greater Cape Town. The majority of children lived in the near vicinity (approximately 500 metres) of the school they attended.

Most children did not live directly on major roads but lived between 50 and 200 metres from such roads. Many of the children from Woodstock and central Cape Town lived in close proximity to small factories, including spray-painting and automobile body works and sheet metal works. Some of the Woodstock children lived within 50 metres of the railway line.

Behavioural and Medical Factors

(See Table 17). The majority of children played primarily outdoors, in the immediate vicinity of their homes; many in busy roads. Most children walked to school, often along busy roads in peak hour traffic.

TABLE 17: BEHAVIOURAL AND OTHER DATA

No.	Amount Played Outdoors	Primary Play Site	Route Travelled to School	Evidence of Pica	Nutrition Status	Amount of Tinned Food Eaten	Workers in Lead Related Indust.
1	Majority of free time	Backyard	> 500 m along major road	-	Satisfactory	Insignificant	-
2	Majority of free time	Backyard	> 500 m along major road	-	Satisfactory	Insignificant	-
3	Approx. half of free time	Side street, 50m from home	> 500 m along major road	-	Satisfactory(?)	Insignificant	-
4	Majority of free time	Major road 100-200 m from home	> 500 m along major road	-	Satisfactory(?)	Insignificant	-
5	Majority of free time	Backyard	> 500 m along major road	-	Satisfactory(?)	Insignificant	-
6	Majority of free time	Yard	200-500 m along major road	-	Satisfactory	Tinned food daily	-
7.	Majority of free time	Yard	200-500 m along major road	-	Satisfactory	Insignificant	-
8	Majority of free time	Backyard, side streets, 100-200 m from home	100-200 m along major road	-	Eats no meat or vegetables	Insignificant	-
9	Majority of free time	Backyard	100-200 m along major road	-	Eats no cooked meat or fresh vegetables	Tinned food daily	Spray painter/ panel beater
10	Approx. half of free time	Backyard, side streets	100-200 m along major road	Eats pa- per, sticks, dirt	Satisfactory	Insignificant	-

Table 17 Continued

No.	Amount Played Outdoors	Primary Play Site	Route Travelled to School	Evidence of Pica	Nutrition Status	Amount of Tinned Food Eaten	Workers in Lead Related Indust
11	Majority of free time	Side streets approx 50 m from home	100-200 m along major road	Mouths objects such as buttons, beads	Satisfactory	Insignificant	-
12	Less than half of free time	Inside	> 500 m along major road	Eats paper, sticks	Satisfactory	Insignificant	-
13	Majority of free time	Side streets 100-200 m from home	< 50 m along major road	-	Satisfactory	Insignificant	-
14	Approx. half of free time	Yard	200-500 m along major road	Eats matches	Satisfactory	Insignificant	-
15	Majority of free time	Grounds of shooting range, 200-500 m from home	-	-	Satisfactory	Insignificant	-
16	Majority of free time	Garden	-	-	Satisfactory	Insignificant	-
17	Majority of free time	Yard	100-200 m along major road	-	Unsatisfactory	Insignificant	-
18	Less than half of free time	Inside	< 50 m along major road	-	Satisfactory	Insignificant	-
19	Majority of free time outdoors	Side streets, 100-200 m from home	< 50 m along major road	-	Satisfactory	Insignificant	-
20	Majority of free time	Yard	100-200 m along major road	-	Satisfactory(?)	Insignificant	-

Three children exhibited evidence of pica and one child showed a marked tendency to mouth objects. Children showed evidence of pica for paper, sticks, earth and matchsticks. No children showed any evidence of pica for paint.

Fifty percent of the children had been admitted to hospital at some stage of their lives, mainly for chest problems, such as bronchitis. No children had been investigated for lead poisoning.

Other Factors:

(See Table 17).

Nutrition Status

From superficial questioning, it appeared that most children received adequate nourishment. Two children, however, had abnormal eating habits, eating no meat or vegetables and one child was extremely malnourished, eating primarily mealie-meal. (This child was very iron-deficient).

The majority of children ate little or no tinned food, apart from two children who received tinned food daily to supplement their diet.

Cooking Utensils

All families used aluminium pots for cooking and no pottery or ceramic-ware was used for storing or cooking food in.

Occupational Histories

With the exception of one resident who was employed as a spray-painter/panel beater, employees were not occupationally exposed to lead.

Discussion

From the home investigations, it is evident that most children lived in extremely poor socio-economic conditions. It has been estimated that the minimum subsistence level for a 'Coloured' family of five living in Cape Town is R231,00 per month (96). Most of the families visited were living close to, or below, this level.

It has been widely documented that lead poisoning and elevated blood lead levels are more common among people of low socio-economic status (58,62,63,64,65). This association was recently confirmed in a nation-wide health survey undertaken in America (63). It was shown that children from low income groups had a significantly higher prevalence of elevated blood lead levels than those from moderate or high income groups. For instance, elevated lead levels were found in 1.2% of children from families with high incomes, but in 10.8% of those from families

with low incomes.

Poor environments hold many risks of excessive lead exposure. Children may be more exposed to lead from sources such as traffic, industry, dust, dirt, flaking paint in old badly maintained houses. Poor children also are more likely to suffer from nutritional deficiencies known to potentiate lead toxicity.

Among the social and familial factors reported to be associated with increased lead absorption are factors such as absence of parents, lack of day-care facilities or a stimulating environment and inability to cope with a stressful city life (58).

The overall poor social and physical environmental circumstances in which the children investigated in the present study live, are likely to be predisposing factors to their increased lead intake from the environment.

3.2.2 Environmental Lead Analyses

From the preliminary home investigations, it did not seem likely that paint or water represented significant sources of exposure to lead in the children. The majority of houses had few interior flaking surfaces and no children exhibited evidence of pica for paint. Although lead pipes have been found in some old houses in Cape Town (73),

the houses investigated in the present study did not have lead pipes.

To discount the possibility that old lead pipes beneath the ground may be present, water samples were obtained for lead analysis. In addition, paint, soil and dust samples from houses built prior to 1950 were obtained for lead analysis. Paint films used prior to this period are liable to contain large amounts of lead, although certain modern decorating paints may still contain a significant amount. (These are usually used in industrial applications (97).) As old layers of paint are rarely stripped off when redecorating, lead based paints can remain potentially harmful for many years.

Results and Discussion

3.3.2.1 WATER

Collection of Samples: One litre daytime samples of hot water were obtained from the kitchen tap at each house. For control purposes, a sample of distilled water and a sample of tap water from a laboratory were also obtained for analysis.

Samples were collected and stored in glass bottles which had been washed in a solution of nitric acid and rinsed

thoroughly. Approximately 10 mls of nitric acid were added to each sample immediately after collection.

Analysis: All samples were analysed within two weeks of collection, by flame atomic absorption spectrophotometry. Samples were heated in platinum containers, evaporated to a level of 10 mls and aspirated directly into an air acetylene flame. The absorbance was read at 217 nms.

Results and Discussion

Results of analyses performed are given in Table 18. The total variation in lead content of samples was between 3 and 12 ug/l. The sample of distilled water had a lead content between 5 and 15 ug/l, whilst the sample of tap water from the laboratory had a lead content between 7 and 11 ug/l.

The lead content measured in treated waters leaving Cape Town's filtration plants is less than 1 ug/l (98). The lead levels in all samples analysed in the present study were higher than this, but within the South African Bureau of Standards recommended limit for lead in domestic water supplies, which is 50 ug/l. The maximum allowable limit is 100 ug/l (99).

TABLE 18. RESULTS OF WATER LEAD ANALYSES

Child No	Water Lead Concentration (ug/l)
1	6 - 10
2	6 - 12
3	3 - 6
4	3 - 7
5	3 - 8
6	6 - 8
7	3 - 8
8	4 - 8
9	3 - 7
10	3 - 8
11	4 - 9
12	3 - 7
13	4 - 8
14	6 - 11
15	5 - 9
16	6 - 10
17	4 - 7
18	5 - 8
19	5 - 8
20	3 - 5

3.3.2.2 PAINT

Collection of Samples: Two or more full-thickness (multi-layer) paint samples were obtained from 13 houses built prior to 1950. Samples were obtained from surfaces likely to have been coated with lead-based paints and thought to be accessible to children (for example indoor and outdoor surface coverings of windows, doors and walls). Due to the difficulty in many cases of obtaining representative samples, it was not possible to sample a precise and reproducible volume of paint.

Altogether 35 samples (of at least two square centimetres in area) were collected and stored in cleaned glass vials with tight-fitting plastic stoppers.

Analysis: All samples were analysed within two weeks of collection, by flame atomic absorption spectrophotometry. Samples were digested in boiling nitric acid, condensed, filtered or centrifuged (if necessary) and aspirated into an air acetylene flame. If lead levels were very low, they were further condensed and complexed with an ammonium pyrrolidene dicarbamate solution with subsequent solvent extraction.

Results

Results of analyses performed are given in Table 19. The lead content of samples varied within homes and between

TABLE 19. RESULTS OF PAINT LEAD ANALYSES

Child No.	Sample Description	Paint lead concentration (%)
1	Bedroom (cream)	.07
2	Kitchen (green)	.3
	Outside door (green)	1.6
3	Ceiling (brown)	4.9
	Outside (blue)	1.6
	Lounge (cream)	.06
4	Kitchen (brown)	1
	Bedroom (green)	1
5	Kitchen (grey)	.43
	Bedroom (blue)	.18
6	-	
7	Outside (green)	2
	Outer windowsill (blue)	11.4
	Lounge (cream)	.6
	Outside (blue)	.23
8	Lounge (blue)	.03
	Lounge windowsill (blue)	3.5
9	Bedroom windowsill (green)	5.0
	Outside (white)	4.8
10	Outside (pink)	3.8
	Bedroom (cream)	.08
	Hallway (pink)	1
11	Lounge/bedroom (cream)	5.9
	Kitchen (cream)	.4
	Outside door (silver)	3.8

Table 19 Continued

Child No.	Sample Description	Paint lead concentration (%)
12	-	-
13	Hallway (red)	1.2
	Kitchen (blue)	.08
	Bedroom (blue)	.46
14	-	-
15	-	-
16	-	-
17	-	-
18	Kitchen (grey)	.3
	Outside (brown)	1
	Bedroom (brown)	.02
	Lounge (grey)	.02
19	Kitchen (cream)	1.8
	Outside (white)	.02
	Lounge (cream)	1
	Bedroom (cream)	.2
20	-	-

homes. The average lead content of samples was 1.7%, ranging from .02 - 11.4%. Lead content was highest in paint samples from wooden windowsills, doors and ceilings; the average lead content in these samples was 5%. Paint on exterior walls generally had a higher lead content than paint on interior walls; the respective average lead content in these samples was 1.44% and 0.7%. Forty percent of the paint samples had a lead content exceeding 1%; regarded in the United Kingdom as the safety level for paints applied to surfaces liable to be chewed by children (97).

Discussion

Although paint samples were obtained from old houses, built prior to 1950, it is thought that the lead content of samples analysed reflects primarily the lead content in modern decorating paints. The majority of houses were painted frequently (every 2-3 years) and few houses had multi-layer flaking surfaces.

Although the lead content in many paint samples was unacceptably high, it is not thought that paint represents a significant source of exposure in these children. As has been discussed, the majority of houses had few interior flaking surfaces and no children exhibited evidence of pica for paint. One house had nearly all its interior surfaces in a flaking condition, but acceptable lead

concentrations were found in paint samples analysed.

Nevertheless, such paints may represent a significant source of exposure for young children, between the ages of 1-3 years when pica is most prevalent. Painted wooden surfaces had the highest lead content and were probably coated in high-lead primers. South Africa has no standards for lead in paint and cheaper low grade paints may have lead pigments containing up to 25% lead. In certain primers, lead pigments may contain 60% lead (79). These are still used in residential housing in Cape Town (100).

3.3.3.3 DUST AND SOIL

Collection of Samples: Surface soil samples of approximately 5 gms were collected from 9 houses. Samples were collected at a distance of approximately 1 metre from either the front or the back of the house.

Dust samples of approximately 250 mgs were collected from the interior rooms of 12 houses.

Soil and dust samples were collected from the same houses as the paint samples, but in some cases it was not possible to obtain a sufficient amount for analysis.

Analysis: Samples were prepared and analysed for lead according to the same procedure used for paint analyses.

Results and Discussion

Results of analyses performed are given in Table 20. The average lead content in soil samples was 1300 ppm ranging from 23-5000 ppm. Three samples had lead levels of 1000, 2000 and 5000 ppm. The lead content in all but one of the soil samples were above natural soil lead concentrations, which are estimated to be in the range 2-220 ppm (in South African soils, the natural concentration level is estimated to be 12 ppm) (22). The average lead content in dust samples was 1000 ppm, ranging from 100-2600 ppm. In fifty percent of the samples, the lead content exceeded 500 ppm, the United States safety level for lead in dust (101).

The close correlation in lead concentrations between some soil and dust samples (see Table 20) may indicate that they are derived from the same source. Although the lead content in certain samples may reflect some contribution from flaking paint, it is thought that the contribution from aerosols, particularly in samples from Woodstock and Cape Town, is likely to be substantial. From Appendices 5 and 6 it is evident that the lead content in these samples is comparable to that found in urban soils and street dusts in some parts of the world. As petrol-derived lead constitutes the major proportion of airborne lead (see Appendix 3) and many children lived in areas with heavy traffic densities, the contribution from this source is likely to be significant.

TABLE 20. RESULTS OF SOIL AND DUST LEAD ANALYSES

Child No	Soil Lead Concentration (ppm)	Dust Lead Concentration (ppm)
1	-	2600
2	460	400
3	390	-
4	-	1300
5	-	100
6	-	-
7	2100	1500
8	23	-
9	1000	1000
10	910	200
11	710	1500
12	-	300
13	-	900
14	-	-
15	-	-
16	-	-
17	-	-
18	380	800
19	5000	2000
20	-	-

General Discussion

It is concluded that there is no obvious local source of exposure such as a significant degree of flaking lead-based paint, or drinking water with a high lead content, in the homes of the children investigated. Further investigation is needed in order to determine the major source(s) and site(s) of exposure in many of the children identified with increased lead levels. Nevertheless, a brief attempt will be made to give an indication of various sources, which might be of importance in determining children's lead burdens.

It is suggested that lead aerosols, (possibly petrol-derived aerosols), may be an important source of exposure in children from Woodstock and Cape Town, through direct inhalation or indirectly through soil and dust.

Children were exposed to significant traffic densities in the home environment, walking to and from school and at school. For example, the Woodstock school was situated on a major road (see Appendix 9 for the hourly traffic flow measured in this road) and the Cape Town school, although not situated directly on a major road, was bounded within 50 metres by three major roads.

In addition, children may have been exposed to industrial sources of lead, such as emissions from spray painting and automobile body works, sheet metal works, and a battery

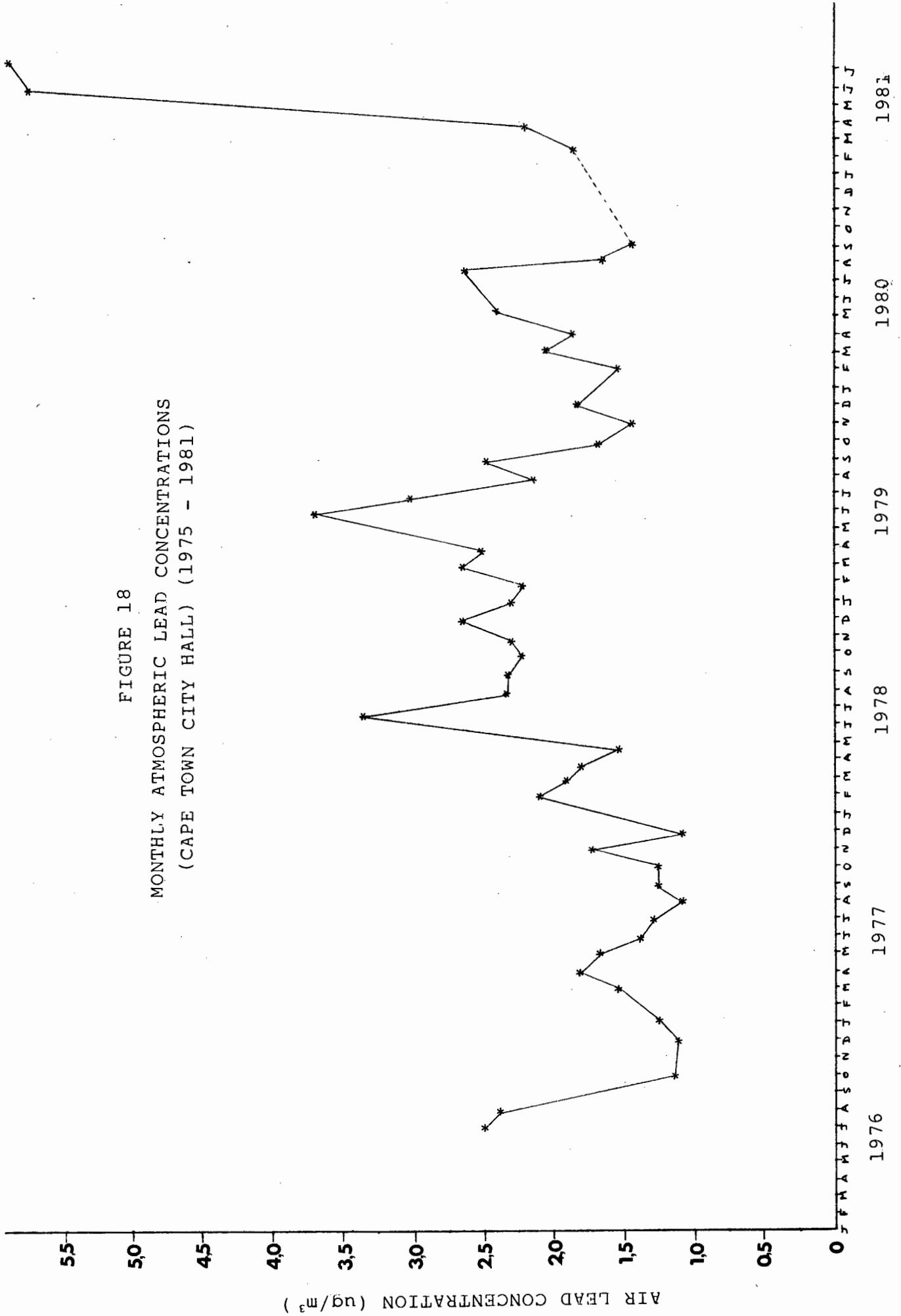
factory, situated in the same road as the Woodstock school. Situated directly adjacent to the school in Woodstock was a garage; children may have been exposed to inorganic lead particulates and organic vapours from this source. A garage as well as a railways depot, was situated directly oppoiste the Cape Town school.

High concentrations of airborne lead have been measured in the city of Cape Town (76) (these are given in Figure 18) which are comparable to those measured in major urban and industrial centres of the world. (See Appendix 4). Caution must be used in interpreting these air lead measurements, however, as airborne lead concentrations fall off rapidly from the source (17) and they may therefore only reflect the air lead concentration at a particular site. Nevertheless, the high air concentrations measured in th city suggest that children may be exposed to a significant amount of airborne lead.

Pica may also be a source of increased lead intake in Cape Town children. Two children exhibited evidence of pica for soil, paper and sticks, which may be potential sources of lead (2).

Pica is probably the major cause of the extremely high lead

FIGURE 18
MONTHLY ATMOSPHERIC LEAD CONCENTRATIONS
(CAPE TOWN CITY HALL) (1975 - 1981)



intake in the child from Athlone. This child exhibited pica for matchsticks. Further investigation is needed to determine the major source of intake.

Lead aerosols from fallout in the shooting range may be a source of exposure in the child from Bellville (86). Exposure may have occurred through direct inhalation of aerosols, or indirectly through soil, dust or contamination of vegetables grown in the vicinity of the shooting range.

The source of lead intake in the child from Retreat needs further investigation, but severe iron-deficiency and the overall poor nutrition status of this child may have played a role in causing increased lead absorption.

Exposure in most children is likely to be multifactorial, depending on individual environmental and social circumstances. Further detailed environmental investigations are needed in order to determine the relative importance of various possible sources of exposure in the children investigated.

BEHAVIOURAL EFFECTS IN
SELECTED CASE STUDIES

4.1 INTRODUCTION

In recent years, concern has been expressed about the adverse effects of relatively low levels of lead on children's behaviour and intelligence. Various studies have attempted to show a relationship between increased body lead burdens and intelligence and behavioural abnormalities in children (10,12,103,104). Many of these, however, suffer from various methodological weaknesses, such as lack of controls for social and other factors, or inadequate estimates of lead exposure.

The most sophisticated study to date, and one which has received most attention in both the scientific literature and the news media, is that by Needleman (11). This study will be briefly discussed.

On the basis of dentine lead concentrations, a 'high lead' and a 'low lead' group of children were chosen for study. The 'high lead' group had lead concentrations corresponding to approximately 35.5 ugPb/dl blood, whilst the 'low lead' group had lead concentrations approximating 23.8 ugPb/dl blood.

Children in both groups underwent detailed neuropsychological testing and it was found that the high lead children differed from the low lead children on a number of measures, such as intelligence and verbal performance. Non-lead co-variates which could also affect development were controlled for in the statistical analysis.

Needleman also tested for differences in the classroom behaviour of children. Teachers were asked to score children on items such as distractability, ability to follow simple directions, overall functioning compared to peers, and other items. High lead children were found to differ significantly from low lead children on most behaviour characteristics tested.

It was evident, thus, that children in the top extreme of the dentine lead distribution differed from those in the bottom extreme in both intelligence and behaviour. Although the study has been criticised on a number of points (106, 107,108), in a recent literature review it was described as the best study to date, the results of which demand serious consideration (105).

In a more recent study (12) it was found that a group of children with a mean blood lead concentration of 16,6 ug/dl had significantly lower IQ levels than a group of children with blood lead concentrations of 10.39 ug/dl. The authors urged caution in drawing firm conclusions from

their results but there is nevertheless substantial evidence to cause concern about the effects of low level lead absorption on children's intellect and behaviour.

In the present study it was decided to investigate whether there was any difference in the classroom behaviour of children identified in the screening test with increased blood lead concentrations, and children with low blood lead concentrations attending the school in Hout Bay.

4.2 SELECTION OF CHILDREN

It was decided to select 30 children (15 children from each group) with the highest and lowest blood lead concentrations in the respective groups. The high lead group consisted of 11 boys and 4 girls; this sex ratio was matched in the low lead group. Descriptive statistics of the blood lead distributions of the two groups are given in Table 21.

All children selected for study were sub A pupils, with the exception of one child in the high lead group who was in sub B.

4.3 METHODS

Behavioural evaluations were performed within a 2 month period of the blood lead tests. The teachers, all of

TABLE 21. BLOOD LEAD DISTRIBUTION (ug/dl)
OF HIGH AND LOW LEAD GROUPS.

	Mean	Standard deviation	Median
High blood lead group	35,67	6,29	35
Low blood lead group	11,67	3,2	12

TABLE 22:
DISTRIBUTION OF UNFAVOURABLE BEHAVIOURAL
RATINGS IN HIGH AND LOW BLOOD LEAD GROUPS

Question Number	High blood lead group	Low blood lead group
1	87	47
2	87	47
3	80	40
4	67	40
5	27	20
6	27	33
7	33	13
8	80	33
9	67	20
10	80	40
11	73	47
Overall	64	35

whom had had at least 6 months teaching experience with the children, and who had no knowledge of the blood lead results, were given Needleman's rating scale and asked to rate the children on various behavioural characteristics. They were asked to score the children, answering yes and no, to the following questions:

1. Is this child easily distracted during his or her work?
2. Can he/she persist with a task for a reasonable amount of time?
3. Can this child work independently and complete assigned tasks with minimal assistance?
4. Is his/her approach to tasks disorganised (constantly misplacing books, pencils, etc.)?
5. Do you consider this child hyperactive?
6. Is she/he over impulsive and excitable?
7. Is he/she easily frustrated by difficulties?
8. Is he/she a daydreamer?
9. Can she/he follow simple directions?
10. Can she/he follow a sequence of directions?
11. In general, is this child functioning as well in the classroom as other children his/her age?

4.4 RESULTS AND DISCUSSION

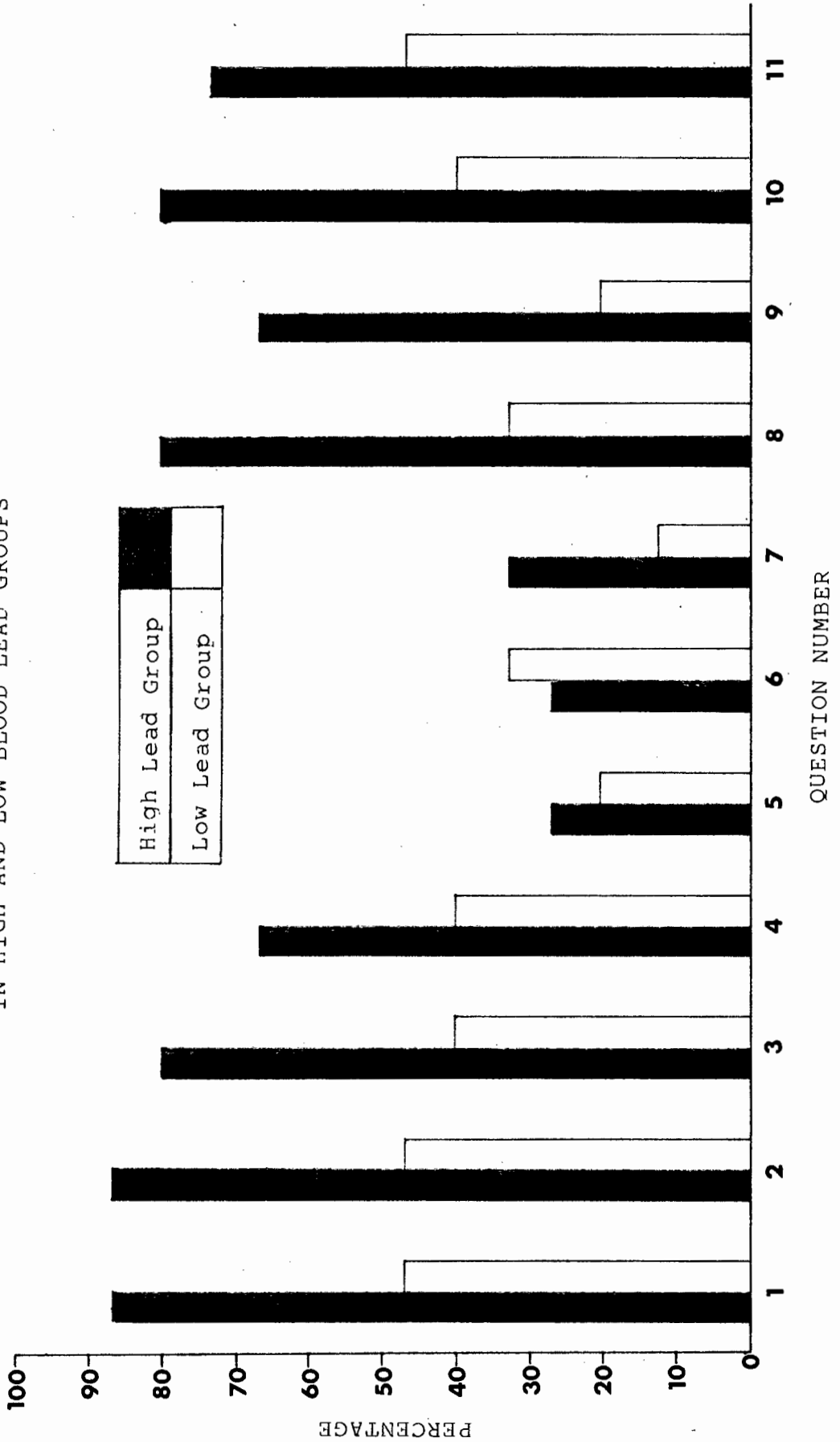
The responses to these questions were analysed in two ways. Firstly, the percentage of unfavourable responses for the test as a whole was compared for both groups, and secondly, the percentage of unfavourable responses for each item were compared for the two groups. The results of the test are given in Table 22 and Figure 19.

It is evident from these results that the children in the high lead group received approximately twice as many unfavourable responses as did the children in the low lead group. The low lead group also scored more favourably than the high lead group on all items except item number six.

In Needleman's study, children in the high lead group received a significantly higher percentage of unfavourable responses (over twice that of the low lead children) on 9 of the 11 items. The two items on which the difference between the low lead and the high lead group did not achieve significance, were items numbers 5 and 6.

An important difference between the two studies is that in the present study, blood lead levels were used as an estimate of lead exposure. The blood lead concentration is representative of short-term exposure (as opposed to dentine lead, which is an indicator of long-term exposure) (105) and provides no estimate of the duration of exposure. There

FIGURE 19
DISTRIBUTION OF UNFAVOURABLE BEHAVIOURAL RATINGS
IN HIGH AND LOW BLOOD LEAD GROUPS



was the possibility, therefore, that exposure in the high lead group may not have occurred over a long enough time to cause significant changes in behaviour.

There was also the possibility that socio-economic status may have been a confounding variable in the study. Low socio-economic status and lead are independently associated with behavioural and intellectual deficiencies in children. It is therefore often difficult to differentiate between the two factors as being causally related to behaviour.

In the present study, although children from both groups came from families of low socio-economic status, the children in the low lead group came from a restricted farming community and lived in extreme conditions of poverty. (In South Africa, farm workers are the lowest paid group of workers (109).) It was possible that the difference in socio-economic status between the groups may have masked any behavioural differences apparent in the respective groups.

Despite these factors, the difference in classroom behaviour between the two groups was striking and suggests that children with moderately increased blood levels may be undergoing certain behavioural changes. It should be noted, however, that the study was based on very small samples and there were no controls on possible confounding variables such as pica. Also, children in the high lead group came

from one particular school; there may therefore have been an element of teacher bias in the rating of children.

Although the results of this study must be interpreted with caution, they are highly suggestive and may have serious implications regarding the health status of certain sections of the community.

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

The evidence presented in this study suggests that a significant proportion of the population, primarily children living in urban-industrial areas, may have increased lead levels and may be suffering certain metabolic and behavioural impairments due to lead.

For instance, 7% of the study population was found to have increased Zinc Protoporphyrin levels (greater than or equal to 5 ug/g Hb) which is an early indicator that physiological impairment due to lead may have occurred. Twice as many children living in predominantly urban-industrial areas had raised ZPP levels compared to children living in other areas. This difference is likely to be due to some extent, to a difference in exposure in traffic. For instance, within these areas, children attending schools situated on major roads had slightly higher ZPP levels than children attending other schools.

Blood lead concentrations of children with raised ZPP levels were found to be considerably higher than those of children in a control group. Twenty-five percent of these children had blood lead levels close to, or exceeding, the currently accepted safety level of 29 ug/dl.

A large percentage of children with raised ZPP and blood lead concentrations attended two schools in particular, Woodstock and Central Cape Town. At the school in Woodstock, average blood lead concentrations were found to be twice as high as those of a control group of children in Hout Bay. The average blood lead concentrations at the two schools were 22.8 ug/dl and 11 ug/dl respectively. Seventeen percent of the children from Woodstock had blood lead concentrations exceeding the safety level.

No local source of exposure was found in the home environments of the children investigated. No houses had lead piping and negligible amounts of lead were found in water samples analysed. Houses investigated did not have significant amounts of flaking lead-based paint and children exhibited no evidence of pica for paint.

Nevertheless, unacceptably high concentrations of lead were found in some paint samples, which may be a potential source of exposure in younger children, when pica and normal hand-to-mouth activities are most common. It is therefore recommended that legislation be introduced (along the lines of that in the United Kingdom and the United States of America) limiting the lead content of certain paints used on surfaces likely to be chewed by children.

Significant levels of lead were found in interior dust and soil samples and it is suggested that lead aerosols,

possibly petrol-derived aerosols, may be an important source of exposure in many of the inner city-Woodstock children, who lived and attended schools in areas of high traffic densities. A further detailed environmental study is necessary in order to determine the relative importance of various potential sources of exposure, such as lead emissions from traffic and industry.

Of particular concern is the fact that children with moderately increased blood lead concentrations (35.5 ug/dl) were found to exhibit striking differences in classroom behaviour compared to children with low blood lead levels. For instance, there was a two fold increase in the prevalence of deviant behavioural traits in children with increased lead levels.

The evidence presented, although tentative, suggests that there is cause for concern regarding the impact of lead in the environment on children in parts of Cape Town.

It is suggested that a continuous screening programme be initiated, to monitor the lead levels of children who may be at particular risk, for instance, children in Woodstock and central Cape Town. It is also recommended that continuous air pollution monitoring be carried out in these areas and that a detailed inventory of all industrial sources of exposure be taken.

Furthermore, it is suggested that legislation limiting the lead content of petrol, the major contributor to lead in the atmosphere, be introduced. South Africa has a standard of .836 gms/l for lead in petrol, which is many times higher than the permissible level for lead in petrol in most countries. In view of the evidence of the toxicity of lead additives in petrol, many countries have reduced, have plans to reduce, or have banned the use of lead in petrol (110). Although the evidence presented in this study is not sufficient to prove a causal link between petrol derived aerosols and lead levels in children, the evidence is suggestive. In view of the serious toxic effects even slightly increased lead concentrations may have on children, it is recommended that prudent precautions be taken to limit lead exposure from this source.

Finally, it is suggested that a major educational effort be directed at the community and to the political structures that relate to the community, to make people aware of the hazards and sources of lead exposure in Cape Town, and to equip people to deal with the problem.

BIBLIOGRAPHY

1. Gilfillan, S.C. 1965. Lead Poisoning and the Fall of Rome. Journal of Occupational Medicine, 7: 53.
2. Wessel, M. 1977. Our Children's Daily Lead. American Scientist, 65(3): 294.
3. Ember, L.R. 1980. Environmental Lead: Insidious Health Problem. Chemical and Engineering News, 58(25): 28.
4. Grandjean, P. 1981. Blood Lead Concentrations Reconsidered. Nature, 291(21 May): 188.
5. World Health Organization 1977. Environmental Health Criteria 3 Lead. Who, Geneva.
6. Settle, D. and Patterson, C. 1980. Lead in Albacore: Guide to Lead Pollution in Americans. Science, 207 (14 March): 1167.
7. Waldron, H.A. 1974. The Blood Lead Threshold, Archives of Environmental Health, 29(Nov): 271.
8. Grandjean, P. and Fischbein, A. 1980. Public Health and Preventive Medicine. Chapter 14. Ed.J. Last
Apelton - Century - Crafts. U.S.A.

9. Piomelli, S. et.al., 1978. The Threshold of Lead Toxicity in Children. Pediatric Research 12: 426.
10. David, O. et.al. 1972. Lead and Hyperactivity, The Lancet, (Oct. 28): 900.
11. Needleman, H.L. et.al., 1979. Deficits in Psychologic and Classroom Performance of Children with Elevated Dentine Lead Levels. New England Journal of Medicine, 300(13): 689.
12. Yule, W. et.al 1981. The Relationship between Blood Lead Concentrations, Intelligence and Attainment in a School Population: a Pilot Study. Developmental Medicine and Child Neurology, 23: 567.
13. Gloag, D. 1980. Is Low Level Lead Pollution Dangerous? British Medical Journal, 281(13 Dec.): 1622.
14. Jones, R.R. 1981. Is Low Level Lead Pollution Dangerous? (Letter), British Medical Journal, 282 (Jan): 147.
15. Gloag, D. 1981. Sources of Lead Pollution, British Medical Journal. 282(Jan): 41.
16. Jones, R.R. 1981. Sources of Lead Pollution (Letter). British Medical Journal. 477 (Feb.): 477.

17. Nriagu J.O. 1978. Lead in the Atmosphere In: The Biogeochemistry of Lead the Environment. Chapter 6 Ed.J.O. Nriagu, Elsevier, Holland.
18. Jones, R.R. 1981. Lead in Petrol (Letter) The Lancet. (May 23): 1160.
19. Grandjean, P. and Andersen, O. 1982. Toxicity of Lead Additives. (Letter) The Lancet. (Aug. 7): 333.
20. Bryce - Smith, D. 1971. Lead Pollution and Mental Health. Biologist, (18).
21. Kesler, S. 1978, Economic Lead Deposits. In: The Biogeochemistry of Lead in the Environment. Chapter 3, Ed.J.O. Nriagu, Elsevier, Holland.
22. Nriagu, J.O. 1978, Properties and the Biogeochemistry of Lead. In: The Biogeochemistry of Lead in the Environment. Chapter 1, Ed. J.O. Nriagu, Elsevier, Holland.
23. Moore, M.R., et al., 1977. The Environment and Man. Vol.6. The Chemical Environment. Ed's: Lenithan, J. and Fletcher, W. Blackie, Glasgow.
24. Harrison R.M. and Laxen, D.P.H., 1981. Lead Pollution Causes and Control. University Press, Cambridge.

25. Corrin, M.L. and Natusch, D.F. 1977. Physical and Environmental Characteristics of Environmental Lead. In: Lead in the Environment. Chapter 1 Ed: W.R. Boggess.
26. United States Environmental Protection Agency 1980 Annual Report.
27. Antopol, W. et.al. 1972. Lead Exposure: Focal Concentrations from Atmospheric Fallout. Miscellaneous Report. Mount Sinai School of Medicine, University New York, USA.
28. Chow, T.J. 1972. Our Daily Lead. Chemistry in Britain. 9 :258.
29. Agrawal, Y.K. et.al. 1980. Effect of Lead from Motor-Vehicle Exhausts on Plant and Soil Along a Major Thoroughfare in Baroda City. International Journal of Environmental Studies. 14(4) :313.
30. Beel, A. 1981. Lead in Petrol. Where does it Go? ECOS. CSIRO Environmental Research 29. (Aug): 3.
31. Lead and Health 1980. Report by DHSS Working Party on Lead in the Environment. London.
32. Mathew, G.K. 1980. Lead in Drinking Water and Health. Studies in Environmental Science, 12. Eds: van Lelyveld, H. and Loekman, B.C.J. Amsterdam.

33. Lin-Fu, J.S. 1973. Vulnerability of Children to Lead Exposure and Toxicity. Part One. New England Journal of Medicine. (Dec. 6): 1229.
34. Sayre, J.W. et.al. 1974. House and Hand Dust as a Potential source of Childhood Lead Exposure. American Journal of Diseases of Children, 127: 167.
35. Catton, M.J. et.al. 1970. Subclinical Neuropathy in Lead Workers. British Medical Journal. 2: 80.
36. Shaffner, R.M. 1981. Lead in Canned Foods. Food Technology, (Dec.): 60.
37. Public Safety: Lead. 1973. Which , (April): 104.
38. Lin-Fu, J.S. 1975. Undue Lead Absorption and Lead Piosoning in Children: An Overview. In: Proceedings of the International Conference on Heavy Metals in the Environment. Toronto, Canada.
39. Waldron, H.A. and Stöfen, D. 1974. Sub-Clinical Poisoning. Academic Press. London.
40. Hammond, P.B. 1977. Exposure of Humans to Lead. Annual Review of Pharmacology and Toxicology. 17: 197.

41. Sterling, T. 1964. Epidemiology of Disease Associated with Lead. Archives of Environmental Health. 8: 145.
42. Ziegler, E.E. 1978. Absorption and Retention of Lead by Infants. Pediatric Research. 21: 29.
43. Moore, M.R. 1979. Diet and Lead Toxicity. Proceedings of the Nutritional Society. 38: 243.
44. Rabinowitz, M.B. et.al. 1980. Effect of Food Intake and Fasting on Gastrointestinal Lead Absorption in Humans. The American Journal of Clinical Nutrition. 33 (Aug): 1784.
45. Piomelli, S. and Graziana, J. 1980. Laboratory Diagnosis of Lead Poisoning. Pediatrics Clinics of North America 27(4): 843.
46. Zinc Protoporphyrin (ZPP): A Simple, Sensitive, Fluorometric Screening Test for Lead Poisoning. Clinical Chemistry, 21(1): 93.
47. Pocock, S.J. 1980. Factors Influencing Household Water Lead: A British National Survey. Archives of Environmental Health. 35(1): 45.
48. Thomas, H.V. et.al. 1976. Blood Lead of Persons Living near Freeways. Archives of Environmental Health. 15:695.

49. Daines, R.H. et.al. 1972. Air Levels of Lead Inside and Outside of Homes. Industrial Medicine. 41: 26.
50. Cohen, C.J. et.al. 1973. Epidemiology of Lead Poisoning. - A Comparison Between Urban and Rural Children. Journal of the American Medical Association. 226: 1430.
51. Caprio, R.J. et.al. 1974. Lead Absorption in Children and it's Relationship to Urban Traffic Densities. Archives of Environmental Health. 28: 195.
52. Landrigan, P.J. and Baker, E.L. 1981. Exposure of Children to Heavy Metals from Smelters: Epidemiology and Toxic Consequences. Environmental Research, 25: 204.
53. McIntire, M.S. and Angle, C.R. 1972. Air Lead: Relation to Lead in Blood of Black School Children Deficient in Glucose - 6- Phosphate - Dehydrogenase. Science. 177: 520.
54. Angle, C.R. et.al 1975. High Urban Lead and Decreased Red Blood Cell Survival. In: Proceeding of the International Conference on Heavy Metals in the Environment. Toronto, Canada.
55. Galke, W.A. et.al. 1975. Environmental Determinants of Lead Burdens in Children. In: Proceedings of the Intenational Conference on Heavry Metals in the Environ-ment Toronto, Canada.

56. Charney, E. et.al. 1980. Increased Lead Absorption in Inner City Children: Where Does the Lead Come From? Pediatrics, 65(2): 226.
57. Ter Haar, G. and Chadzynski, L. 1979. An Investigation of Elevated Blood Lead Levels in Detroit Children. Archives of Environmental Health. 34(3): 145.
58. Lin-Fu, J. 1982. Editorial, Children and Lead The New England Journal of Medicine. 307(10): 615.
59. Lin-Fu, J.S. 1973. Vulnerability of Children to Lead Exposure and Toxicity. Part 2. The New England Journal of Medicine. (Dec. 13): 1289.
60. Watson, W.N. et.al. 1978. Increased Lead Absorption in Children of Workers in a Lead Storage Battery Plant. Journal of Occupational Medicine. 20(11): 759.
61. Morton, D.E. et.al. 1982. Lead Absorption in Children of Employees in a Lead - Related Industry. American Journal of Epidemiology. 115(4): 549.
62. Marshall, E. 1982. E.P.A. May Allow More Lead in Gasoline. Science, 215(12 March): 1375.

63. Mahaffey, K.R. et.al. 1982. National Estimates of Blood Lead Levels: United States, 1976-1980. The New England Journal of Medicine. 307(10): 573.
64. Egbuonu, L. et.al. 1982. Child Health and Social Status. Pediatrics, 69(5): 550.
65. Starfield, B.H. 1982, Editorial Child Health and Socio-Economic Status. American Journal of Public Health. 72(6): 532.
66. Lerner, S. Blood Lead Analysis. Precision and Stability. Journal of Occupational Medicine. 17(3): 153.
67. Baloh, R.W. 1974. Laboratory Diagnosis of Increased Lead Absorption. Archives of Environmental Health. 28 (April): 198.
68. AVIV Hematofluorometer Model ZPP, Manual, U.S.A.
69. Blumberg, W.E. et.al., 1977, Zinc Protoporphyrin Level in Blood Determined by a Portable Hematofluorometer: A Screening Device for Lead Poisoning. Journal of the Laboratories of Clinical Medicine, 89: 712.
70. Lead Poisoning in Children, 1979. Department of Health, South Africa.

71. Kibel, M.A. 1980. Health Screening in Childhood: The Present State of the Art. Inaugural Lecture no.63. University of Cape Town.
72. Harris, I. 1976. Lead Encephalopathy Case Reports. South African Medical Journal. (Aug.): 1371.
73. Retief- Steyn, F. 1976. Loodas Bedryfs en Openbare Gesondheidsgevaar in Suid Afrika. Unpublished M.D. Thesis, University of Pretoria.
74. Editorial, 1977. Vergiftiging en Dokters se Speurvernuf. South African Medical Journal, (March): 328.
75. Editorial, 1979. Kinders en Blootstelling aan Lood. South African Medical Journal, 56(16): 621.
76. Vleggaar, C.M. et.al. 1980. Trace Elements in Airborne Particulates in South Africa. Atomic Energy Board, Pelindaba 274, (Oct.).
77. Dutkewitz R.K. et.al. 1981. Air Pollution Survey of Greater Cape Town. Vol.V, Cape Town City Council.
78. Editorial 1981. Clear the Air, The Star April 15th.

79. Representative, South African Bureau of Standards.
80. Health Statistics, 1981. Department of Health, Welfare and Pensions, South Africa.
81. Annual Health Report, 1979. Cape Town City Council.
82. Fishbein, A. et.al., 1976. Zinc Protoporphyrin Determination: A Rapid Screening Test for the Detection of Lead Poisoning. The Mount Sinai Journal of Medicine. 43(3): 294.
83. Mets, J.T. 1981. Biological Monitoring of Occupational Exposure to Lead with a Zinc Protoporphyrin (ZPP) Meter. South African Medical Journal. 60(5 Dec): 891.
84. Popovac, D. et.al. 1982. Elevated Blood Lead in a Population Near a Lead Smelter in Kosovo, Yugoslavia. Archives of Environmental Health. 37(1): 19.
85. Grandjean, P. 1979. Occupational Lead Exposure in Denmark: Screening with the Hematofluorometer. British Journal of Industrial Medicine. 36: 52.
86. Richter, E.D. et.al. Lead Exposure: Effects in Israel. Israeli Journal of Medical Sciences. 16(2):

87. Roels, H.A. 1975. Response in Some Heme Biosynthetic Pathway Parameters in Men, Women and Children Moderately Exposed to Lead. In: Proceedings of the International Conference on Heavy Metals in the Environment, Toronto, Canada.
88. Centers for Disease Control. Preventing Lead Poisoning in Young Children. Journal of Pediatrics, 93: 704.
89. Roels, H. et.al. 1976. Impact of Air Pollution by Lead on the Heme Biosynthetic Pathway in School Age Children. Archives of Environmental Health, (Nov./Dec.): 310.
90. Moore, M.R. and Meredith, P.A. 1977. The Storage of Samples for Blood and Water Lead Analysis. Clinica Chimica Acta, 75: 167.
91. Hesley, K.L. and Wimbish, G.H. 1981. Blood Lead and Zinc Protoporphyrin in Lead Industry Workers. Journal of the American Industrial Hygiene Association. 42 (Jan): 42.
92. Sassa, S. et.al. 1973. Studies in Lead Poisoning. Biochemistry and Medicine, 8: 135.

93. Lamola, A.A. et al. 1975. Zinc Protoporphyrin (ZPP): A Simple, Sensitive, Fluorometric Test for Lead Poisoning. Clinical Chemistry 21(93):
94. Meredith, P.A. et al 1979. Erythrocyte Delta-Aminolaevulinic Acid Dehydratase Activity and Blood Protoporphyrin Concentrations as Indices of Lead Exposure and Altered Haem Biosynthesis. Clinical Science 56:61.
95. Grandjean, P. and Lintrup, J. 1978. Erythrocyte-Zn-Protoporphyrin as an indicator of Lead Exposure. Scandinavian Journal of Clinical Laboratory Investigations 38:669.
96. Horrell, M. 1981. Survey of Race Relations in South Africa. South African Institute of Race Relations Johannesburg.
97. Lead in Paint 1981. Notes to Industry Number 11. Paint Research Association, United Kingdom.
98. Office of the City Engineer, Cape Town City Council.
99. South African Bureau of Standards Specification No SABS 241 - 1971.
100. Local Paint Company. (Name withheld).
101. Rogers, R. 1980. Lead Pollution. The Danger that Stalks London's Playgrounds. New Statesman (5 December)
102. Davidson, C.I. et al. 1981. Airborne Lead and Other

Elements Derived from Local Fires in the Himalayas.

Science 214 (18 Dec): 1344.

103. de la Burd , B. and Choate, M. Early Asymptomatic Lead Exposure and Development at School Age. Journal of Pediatrics 87(4) : 638.
104. Landrigan, P.J. et al. 1975. Neuropsychological Dysfunction in Children with Chronic Low Level Lead Absorption. The Lancet (March 29) : 708.
105. Rutter, M. 1980. Raised Lead Levels and Impaired Cognitive/Behavioural Functioning: A Review of the Evidence. Supplement to Developmental Medicine and Child Neurology 22(1).
106. Needleman, H.L. 1979. Research into Lead Pollution (letter) The Lancet (May 12) : 1024.
107. Graham, P. 1979. Research into Lead Pollution (Letter) The Lancet (May 12) : 1024.
108. Needleman, H.L. 1979. Lead and Neurobehavioural Deficit in Children (Letter) The Lancet (July 14) : 104.
109. Work and Health South African Labour Bulletin. 1981. Fact Sheet Number 3. Nusas
110. Dover, C. 1980. Deadly Emissions. Pretoria News, April 16.

PRINCIPAL USES OF LEAD
(after Moore, 1977)

Grid structure and compounds in batteries.

Coverings for power and telephone cables.

Sheet and pipe in plumbing.

Sheet and castings for shielding against gamma-rays and X-rays.

Foil for packaging and damp protection.

Caulking.

Solders.

Shot and bullets.

A principal or secondary ingredient in bearing metals.

Type metals.

For plating, dipping or spraying on steel and other metals.

For improving machinability of steel.

Lead tetra alkyls in petrol.

Litharge and red lead in glass and glazes.

Pigments for paints and inks.

Frits for enamelling aluminium.

Sodium plumbite in oil refining.

In accelerators in the rubber, artificial leather and linoleum industries.

Lead arsenate in insecticides.

Compounds in plastics, matches, explosives.

Sound and vibration attenuation.

Lead naphthenate as a dryer in paints and linseed-oil products

THE PROPERTIES OF LEAD
(after Moore, 1977)

Atomic number	82
Atomic arrangement	Face-centred cubic
Interatomic distance	0.349 nm (3.49 Å)
Atomic weight	207.2 (lead derived from radioactive sources may have atomic weights varying from this figure)
Density at 20°C	11343 kg m ⁻³ 11.34 g/cm ³ (708 lb/ft ³)
Melting point	327.502°C (600.66 K)
Latent heat of fusion	26.2 kJ/kg (6.26 cal/g) (11.27 B.Th.U/lb)
Thermal conductivity 0°C	36 W/(m K) (0,083 cal/(cm s K)
100°C	34 W/(m K) (0,081 cal/(cm s K)
Specific heat (0°C to 100°C)	(127 J/(kg K) 0.031 cal/g/°C (average)
Coefficient of linear expansion	0.000029 °C

GLOBAL LEAD EMISSIONS FROM ANTHROPOGENIC SOURCES
(after Nriagu, 1978)

Source	Global emission (10 ⁶ kg/yr)
Anti-knock additives	267
Soldering	0,22
Pigments	1,31
Metal fabrication	3,82
Lead mining	3,36
Lead milling	4,88
Lead smelting and refining	26,7
Secondary lead	0,77
Coal combustion	14,9
Oil (fuel + waste) combustion	6,14
Wood combustion	1,15
Sewage sludge incineration	0,91
Refuse incineration	3,57
Zinc production	15,7
Nickel production	2,47
Copper production	26,6
Iron and steel production	49,7
Ferroalloys	2,05
Iron foundries	3,90
Cement manufacture	1,41
Crushed stone	1,15
Clay production	0,66
Fertilizer and phosphate rock	0,05
TOTAL	438

ATMOSPHERIC LEAD IN URBAN AND RURAL AREAS
(after Nriagu, 1978)

Location		Lead Conc. (ug/m ³)
Frankfurt	residential area	0,8
	heavy traffic	2,7
Vienna	winter	2,6 (0,2- 14.0)
	summer	2,9 (0,8-6,6)
Belgium	background level	0,23
The Hague	downtown	1,2
	residential	0,4
Amsterdam		0,9
Tula	before ban on leaded gasoline	1,4
	after ban on leaded gasoline	0,24
London	downtown	5,1
UK (rural locations)	1973	0,13
New York	metro sites	1,7 (0,3-2,1)
Boston	downtown	4,5 (2,3-18,9)
Los Angeles	downtown (1968-69)	3,6
Rio de Janeiro	city centre	0,8
Ottawa	downtown	1,3
Montreal		2,0
Toronto	downtown commercial	1,6
Guayaquil Ecuador		0,29 (0,15-0,35)

LEAD CONTENT OF VARIOUS SOILS
(After Chow, 1972)

Location		Pb(ppm)
Austria	Vienna	85
Belgium	Brussels	859
Denmark	Copenhagen	105
France	Paris	220
Germany	Munchen	158
	Hanover	757
Holland	Amsterdam	893
USSR	Moscow	19
England	Cambridgeshire	300
	Sussex	100
Chile	Santiago	90
Peru	Lima	223
Mexico	Mexico City	179
USA	New York City	834
	Los Angeles	442
Canada	British Columbia	500
Japan	Tokyo	195
China	Taipei	80
Hongkong	Victoria	107
Thailand	Bangkok	1175

LEAD IN STREET DUSTS
(After Nriagu, 1978)

Location	Lead Concentration* (ppm)
Manchester	970 (90 - 10 200)
Zurich	2000
Rio de Janeiro	700
Toronto, industrial	67800
New York, heavy traffic area	20000
77 Cities in USA	
Commercial sites	2413
London	
Main roads gutter	1530 (<u>±</u> 600)
Side roads gutter	1030 (<u>±</u> 450)
Birmingham	
Major arterial roads gutter	2350 (160-10 000)
Residential roads gutter	1050 (220-4 300)
Dustfall at highway interchange	1093 (160-8 640)
Ohio (12 cities)	
residential areas	206-2639
commercial areas	352-2933

* Ranges in reported concentrations are shown
in parentheses.

ZPP DISTRIBUTIONS (ug/gHb)
Breakdown by school, class and sex for all pupils.

School	Class	FEMALES					MALES					ALL		
		No.	Mean	S.d.	Median	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median	
1	A1	5	2,7	,3	2,8	7	3,2	0,4	3,3	12	3,0	,4	2,95	
	A2	6	3,9	1,3	3,6	6	3,5	1,1	3,2	12	3,7	1,2	3,4	
	A3	4	3,7	1,2	3,6	7	3,9	2,4	2,9	11	3,8	2,0	2,9	
	A4	6	3,0	0,8	3,95	4	2,6	0,6	2,5	10	2,8	,7	2,8	
	T	21	3,3	1,1	3,0	24	3,4	1,4	3,2	45	3,4	1,2	3,1	
2	A1	10	3,4	1,1	2,95	13	4,2	1,7	3,7	23	3,8	1,5	3,4	
	A2	8	5,2	1,8	5,0	15	5,2	1,7	4,6	23	5,2	1,7	5,	
	T	31	3,4	1,0	3,3	24	3,7	1,3	3,35	55	3,5	1,1	3,3	
3	A1	16	3,2	0,8	2,85	10	4,2	1,1	4,05	26	3,6	1,1	3,15	
	A2	15	3,7	1,1	3,6	14	3,4	1,4	2,9	29	3,5	1,2	3,0	
	T	31	3,4	1,0	3,3	24	3,7	1,3	3,35	55	3,5	1,1	3,3	
4	A1	10	4,6	1,5	4,45	7	4,3	1,2	3,7	17	4,5	1,7	4,1	
	B1	10	3,0	0,8	2,9	8	3,9	0,9	3,95	18	3,4	0,9	3,3	
	C1	6	3,0	0,4	3,15	9	3,8	1,0	3,5	15	3,5	0,8	3,3	
	X1	5	3,2	0,7	2,9	3	4,1	1,7	4,4	8	3,5	1,1	3,05	
	T	31	3,6	1,2	3,2	27	4,0	1,3	3,7	58	3,8	1,3	3,35	

School Class	FEMALES				MALES				ALL				
	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median	
5	A1	14	3,0	0,6	3,1	13	3,0	0,4	2,9	27	3,0	0,5	2,9
	A2	18	3,2	0,8	3,0	17	3,0	0,7	3,0	35	3,1	0,7	3,0
	T	32	3,1	0,7	3,0	30	3,0	0,6	3,0	62	3,1	0,6	3
6	A1	17	3,8	1,2	3,6	12	4,6	2,1	3,85	29	4,2	1,6	3,7
	A2	9	3,8	1,0	3,5	9	4,6	2,0	4,1	18	4,2	1,6	3,75
	A3	2	2,8	0,2	2,85	3	4,1	1,4	3,7	5	3,6	1,2	3,0
	T	28	3,8	1,1	3,55	24	4,6	1,9	3,85	52	4,1	1,6	3,65
7	A1	11	3,2	1,4	2,7	9	2,8	0,5	3,0	20	3,0	1,1	2,75
	A2	4	3,0	0,2	3,0	13	2,8	0,5	2,8	17	2,8	0,4	2,8
	B1	4	3,2	0,6	3,1	5	3,9	0,6	3,9	9	3,6	0,7	3,4
	B2	5	3,2	0,5	3,2	9	3,0	0,4	3,1	14	3,0	0,4	3,15
	T	24	3,2	1,0	2,95	36	3,0	0,6	3	60	3,1	0,8	3
8	A1	20	3,4	,4	3,4	18	3,4	0,5	3,4	38	3,4	0,5	3,4
	A2	14	3,6	,8	3,45	17	3,5	0,9	3,4	31	3,5	0,9	3,4
	A3	11	3,3	,7	3,1	19	3,4	0,7	3,3	30	3,4	0,7	3,3
	T	45	3,4	,6	3,4	54	3,4	0,7	3,4	99	3,4	0,7	3,4

School Class	FEMALES				MALES				ALL				
	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median	
9	A1	15	3,5	2,0	2,9	10	4,9	2,3	3,95	25	4,0	2,2	3
	B1	12	3,6	0,8	3,85	10	3,3	1,1	2,95	22	3,5	0,9	3,25
	C1	10	3,1	1,0	3,05	16	3,4	1,6	2,85	26	3,3	1,4	3
	D1	12	3,2	0,7	3,15	12	3,3	0,9	3,0	24	3,2	0,8	3
	T	49	3,4	1,3	3,0	48	3,7	1,6	3,05	97	3,5	1,5	3
10						29	3,4	1,0	3,2	29	3,4	1,0	3,2
11	A1	3	2,6	0,2	2,6	6	2,9	,6	3,1	9	2,8	0,5	2,8
	A2	2	2,7	0,1	2,75	6	3,2	,6	3,05	8	3,1	0,6	2,9
	A3	3	2,6	0,2	2,7	7	3,1	,5	3,2	10	3,0	0,5	2,75
	A4	5	2,6	0,4	2,7	3	2,8	,6	3,1	8	2,7	0,5	2,8
	A5	5	2,6	,4	2,7	4	2,7	,4	2,75	9	2,7	0,4	2,7
	A6	6	2,8	,4	2,55	6	2,7	,5	2,45	12	2,7	0,4	2,5
	A7/8	3	2,7	,5	2,4	1	3,0	-	3	3,1	2,7	,5	2,4
	B1	5	2,6	,3	2,7	3	2,8	,5	2,8	8	2,7	,3	2,7
	B2	2	2,5	,1	2,5	4	3,0	,5	3,05	8	2,8	,5	2,75
	B3	3	2,6	,2	2,6	6	3,0	,7	2,65	6	2,9	,6	2,6

School	FEMALES					MALES					ALL		
	Class	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median
11 cont	B4	11	3,4	,8	3,8	4	3,0	,3	3,05	15	3,3	,7	3,2
	B5	6	3,2	,4	3,15	3	2,5	,1	2,5	9	2,9	,4	3
	B7	1	3,4	-	3,4	-	-	-	-	1	3,4	-	3,4
	T	55	2,9	,5	2,7	53	2,9	,5	2,9	108	2,9	,5	2,75
12	A1	13	2,6	,4	2,7	19	3,1	,6	3,1	32	2,9	,6	2,85
	A2	17	2,9	,5	2,8	18	4,6	5,8	3,2	35	3,8	4,2	3
	A3	8	3,2	,6	2,95	22	2,8	,4	2,7	30	2,9	,5	2,8
	T	38	2,9	,5	2,8	59	3,5	3,2	3,0	97	3,2	2,6	2,9
13	A1	8	2,8	0,4	2,8	4	4,1	2,8	3,15	12	3,2	1,6	2,8
	A2	4	3,6	1,2	3,5	8	3,1	,8	3,0	12	3,3	,9	3,05
	A3	7	3,6	2,3	2,8	7	3,6	,8	2,7	14	3,3	1,7	2,75
	A4	7	2,6	,6	2,4	10	3,4	2,6	2,5	17	3,1	2,0	2,5
	A5	4	2,7	,7	2,8	7	2,5	,4	2,5	11	2,6	0,5	2,6
T	30	3,0	1,2	2,85	36	3,2	1,7	2,7	66	3,1	1,5	2,7	

		FEMALES						MALES						ALL			
School	Class	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median
14	A1	15	3,2	2,7	2,4	17	2,7	,6	2,6	32	3,0	1,9	2,5				
	B1	11	2,7	,9	2,5	11	2,4	,4	2,25	22	2,5	,7	2,4				
	B2	13	2,6	,5	2,4	5	3,4	1,4	2,8	18	2,8	,9	2,6				
	T	39	2,9	1,8	2,4	33	2,7	,8	2,6	72	2,8	1,4	2,5				
15	A1	1	3,7	-	3,7	1	3,2	-	3,2	2	3,4	,3	4,5				
	B1	5	2,7	,2	2,6	8	3,5	1,3	3,0	13	3,2	1,1	2,8				
	B2	2	4,2	1,3	4,2	8	3,0	,9	2,75	10	3,2	1,0	2,95				
	B3	4	2,9	,6	3,05	3	3,8	,9	3,4	7	3,3	0,8	3,3				
	B7	1	3,0	-	3					1	3,0	-	3,0				
	?	7	2,9	,3	3	5	3,5	,8	3,4	12	3,1	,6	3,05				
	T	20	3,1	,7	3	31	3,2	1,0	3	51	3,2	,9	3,0				
	A2	-	-	-	-	3	2,6	,4	2,6	3	2,6	,4	2,6				
	A3	-	-	-	--	3	3,1	,1	3,1	3	3,0	,1	3,1				

		FEMALES						MALES						ALL			
School	Class	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median	No.	Mean	S.d.	Median
16	A1	12	2,9	,5	2,75	9	2,6	,5	2,6	21	2,7	,5	2,6	21	2,7	,5	2,6
	A2	12	2,5	,2	2,45	7	3,0	,6	3,2	19	2,6	,4	2,5	19	2,6	,4	2,5
	T	24	2,7	,4	2,5	16	2,7	,5	2,65	40	2,7	,5	2,6	40	2,7	,5	2,6
17	A1	10	2,5	,5	2,3	11	3,3	2,1	2,6	21	2,9	1,6	2,4	21	2,9	1,6	2,4
	A2	13	2,4	,6	2,3	15	2,9	1,1	2,5	28	2,6	,9	2,4	28	2,6	,9	2,4
	T	23	2,4	,5	2,3	26	3,0	1,6	2,5	49	2,8	1,2	2,4	49	2,8	1,2	2,4
18	A1	21	2,5	,5	2,5	24	2,6	,7	2,4	45	2,5	,6	2,45	45	2,5	,6	2,45
	A2	23	2,4	,6	2,45	27	2,7	,7	2,6	50	2,6	,7	2,5	50	2,6	,7	2,5
	T	44	2,5	,5	2,5	51	2,6	,7	2,45	95	2,6	,6	2,5	95	2,6	,6	2,5
19	A1	15	2,5	,5	2,35	18	2,9	1,4	2,4	33	2,7	1,1	2,4	33	2,7	1,1	2,4
	B1	10	2,9	,5	2,95	9	3,1	1,1	2,9	19	3,0	,8	2,9	19	3,0	,8	2,9
	T	25	2,7	,5	2,4	27	2,9	1,3	2,95	52	2,8	1,0	2,5	52	2,8	1,0	2,5

BLOOD ANALYSES OF CHILDREN
BY SCHOOL, CLASS AND SEX

School	Class	Sex	ZPP (ug/g Hb)	Blood lead (ug/dl)	Hb (g/dl)
1	A2	M	5,6	17	
	A2	F	6,5	14	
	A3	F	5,2	22	
	A3	M	9,2	25	9,0
2	A1	M	5,2	17	14,6
	A1	M	7,4	23	12,8
	A1	M	8,1	30	13,4
	A1	F	5,9	19	12,7
	A2	F	5,2	30	
	A2	F	8,1	22	13,7
	A2	F	7,5	28	13,9
	A2	F	5,0	18	13,4
	A2	M	5,4	27	13,9
	A2	F	5,0	20	12,8
	A2	M	9,0	26	13,0
	A2	M	6,9	20	11,8
	A2	M	5,2	37	11,9
	A2	M	7,4	32	13,6
	A2	M	6,9	24	12,3
	A2	M	5,4	30	13,3
3	A1	F	5,3		12,7
	A1	M	6,4	18	12,3
	A1	M	5,0	12	12,6

School	Class	Sex	ZPP (ug/g Hb)	Blood lead (ug/dL)	Hb (g/dl)
3 cont.	A2	F	7,3	35	11,5
	A2	M	6,9	26	12,6
	A2	M	5,3	21	12,1
4	A1	F	5,6	11	13,4
	A1	F	6,3	17	14,0
	A1	F	7,4	9	13,8
	A1	M	8,7	14	13,0
	B1	M	5,1	15	12,8
	C1	M	5,4	14	
	Remedial	M	5,6	15	
5	A2	F	5,6	12	12,3
6	A1	F	6,1	15	12,9
	A1	F	5,5	14	13,4
	A1	F	5,2	12	
	A1	M	8,7	27	12,6
	A1	M	7,6	17	
	A1	M	7,5	11	
	A2	F	6,0	17	12,6
	A2	M	9,3	14	
	A2	M	6,1	11	12,4
	A3	M	5,6	10	
7	A1	F	7,0	20	14,4

School	Class	Sex	ZPP (ug/g Hb)	Blood lead (ug/dL)	Hb (g/dl)
8	A2	M	6,3	14	12,8
	A3	M	5,7	10	1,
9	A1	M	5,6		12,5
	A1	M	9,7	25	13,4
	A1	M	5,6	14	12,5
	A1	F	10,1	35	
	A1	M	7,8	44	13,7
	A1	F	5,5	14	13,0
	B1	M	6,1	28	13,6
	C1	M	8,9	11	
	C1	M	5,0	18	12,9
	C1	M	5,3	14	13,1
12	A1	M	5,0	10	13,0
	A2	M	5,6	19	12,7
	A2	M	27,5	50	9,9
13	A1	M	8,2	38	7,8
	A2	F	5,0	19	10,3
	A3	F	8,5	24	7,8
	A4	M	10,6	24	12,8
14	A1	F	12,9	15	7,4
	B1	M	5,3	15	11,4
	B2	M	5,6	20	9,8

School	Class	Sex	ZPP (ug/g Hb)	Blood lead (ug/dL)	Hb (g/dl)
15	B1	M	5,9		15,4
	B1	M	5,2	18	11,5
	B2	M	5,1	13	12,4
17	A1	M	9,4		6,5
	A2	M	6,0	42	11,5
18	A1	M	5,3	15	13,4
19	A1	M	5,2	11	11,2
	A1	M	5,6		
	A1	M	7,3	12	12,7

WOODSTOCK SCHOOL: HOURLY TRAFFIC FLOW IN STREET.

TIME	HOURLY FLOW NORTHBOUND	HOURLY FLOW SOUTHBOUND	TOTAL HOURLY FLOW	% OF 24 HOUR FLOW
0.00 - 1.00	62	66	128	0.7
1.00 - 2.00	40	41	87	0.5
2.00 - 3.00	15	21	36	0.2
3.00 - 4.00	26	22	48	0.3
4.00 - 5.00	36	34	70	0.4
5.00 - 6.00	88	120	208	1.1
6.00 - 7.00	196	173	369	2.0
7.00 - 8.00	728	345	1073	5.9
8.00 - 9.00	646	453	1099	6.0
9.00 - 10.00	565	568	1133	6.2
10.00 - 11.00	588	643	1231	6.8
11.00 - 12.00	680	779	1459	8.0
12.00 - 13.00	723	814	1537	8.4
13.00 - 14.00	581	634	1215	6.7
14.00 - 15.00	630	686	1316	7.2
15.00 - 16.00	557	746	1303	7.2
16.00 - 17.00	590	777	1367	7.5
17.00 - 18.00	522	891	1413	7.8
18.00 - 19.00	362	432	794	4.4
19.00 - 20.00	323	360	689	3.8
20.00 - 21.00	225	275	500	2.7
21.00 - 22.00	170	175	345	1.9
22.00 - 23.00	177	269	446	2.5
23.00 - 24.00	160	169	329	1.8
TOTAL 24HRS :	8696	9499	18195	

PEAK HOUR FLOWS :	TIME	FLOW	% 24HR
MORNING	11.00 - 12.00	1459	8.0
EVENING	11.45 - 12.45	1539	8.5